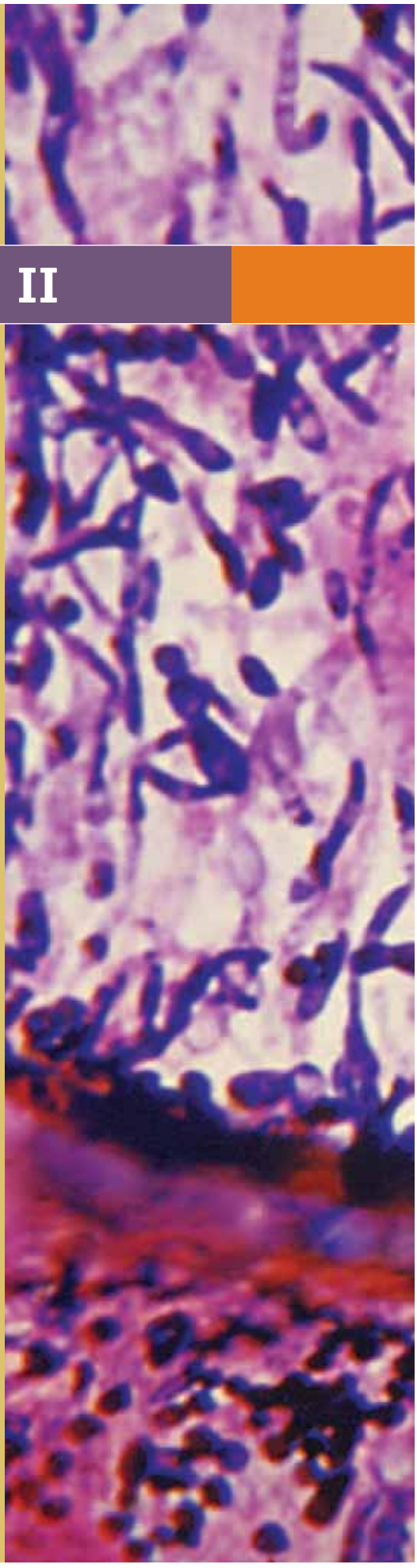

Part II

Diseases of the Systems

Part II presents diseases of the body's systems. Each chapter reviews the normal structure and function of a body system, and then discusses diseases associated with that system. Signs, symptoms, etiology, diagnosis, treatment, and prevention are described for each disease.

Chapters

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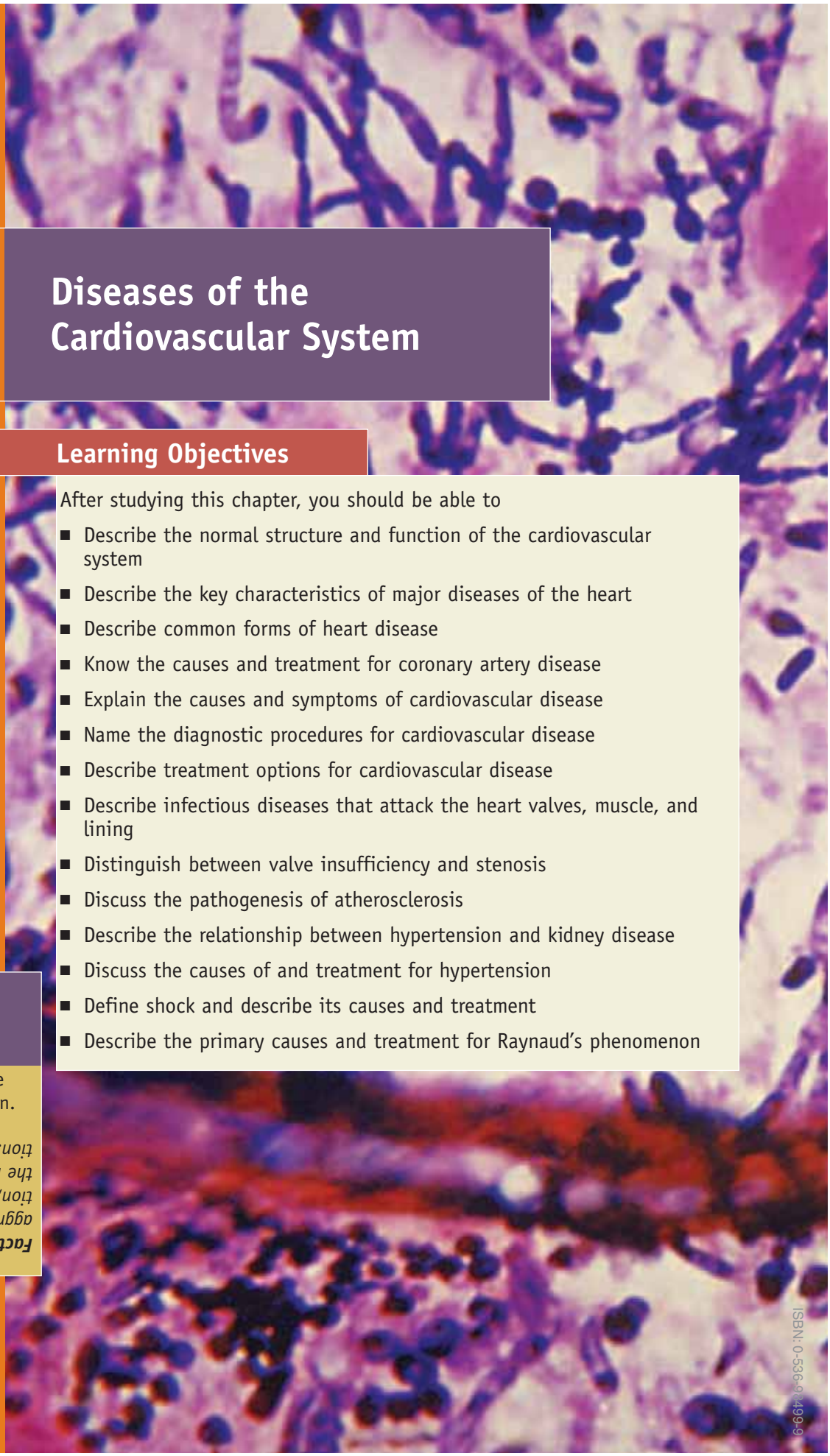

This photomicrograph reveals histopathologic changes indicative of endocarditis caused by the fungus *Candida albicans*. (Courtesy of the CDC/Sherry Brinkman, 1963.)

CHAPTER

7

Diseases of the Cardiovascular System

Learning Objectives

After studying this chapter, you should be able to

- Describe the normal structure and function of the cardiovascular system
- Describe the key characteristics of major diseases of the heart
- Describe common forms of heart disease
- Know the causes and treatment for coronary artery disease
- Explain the causes and symptoms of cardiovascular disease
- Name the diagnostic procedures for cardiovascular disease
- Describe treatment options for cardiovascular disease
- Describe infectious diseases that attack the heart valves, muscle, and lining
- Distinguish between valve insufficiency and stenosis
- Discuss the pathogenesis of atherosclerosis
- Describe the relationship between hypertension and kidney disease
- Discuss the causes of and treatment for hypertension
- Define shock and describe its causes and treatment
- Describe the primary causes and treatment for Raynaud's phenomenon

Fact or Fiction?

Aspirin therapy reduces the risk of myocardial infarction.

Fact: *By minimizing platelet aggregation and clot formation, aspirin therapy reduces the risk of a myocardial infarction.*

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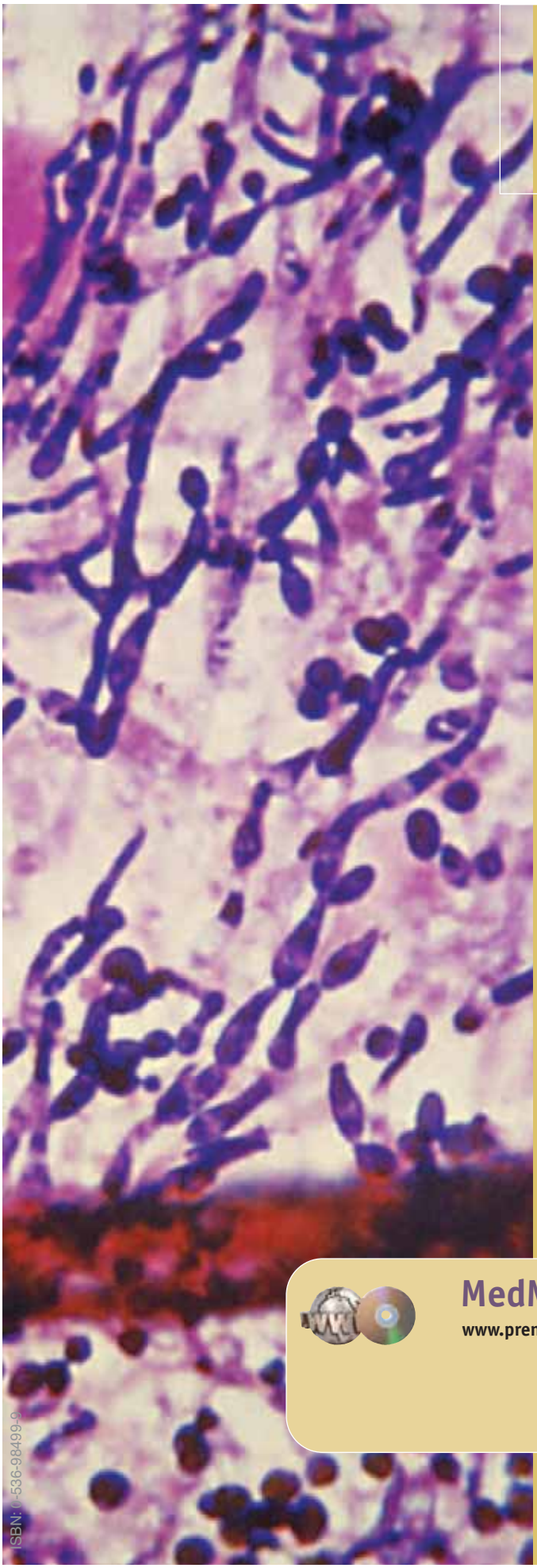

Disease Chronicle

Dr. Christiaan Barnard

Dr. Christiaan Barnard performed the first human heart transplant in 1967. In the Union of South Africa, Dr. Barnard performed this famous surgery on a 53-year-old dentist named Louis Washkansky. The dentist received the donated heart of a 25-year-old auto accident victim named Denise Davall. Although the surgery was a technical triumph and a beacon of hope for many with terminal heart disease, Washkansky died 18 days later from infection. Still risky today, heart transplants owe their successes to the generosity of Denise Davall, the courage of Louis Washkansky, and the brilliance of Dr. Barnard, who died of an apparent heart attack in 2001.

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Use the web address to the left to access the free, interactive Companion Website created for this textbook. It features chapter-specific exercises, Internet links, news links, and an audio glossary. Additionally, explore the CD-ROM that accompanies this book to discover Disease Focus videos and a rich array of activities that accompany this chapter.

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Introduction to the Cardiovascular System

The cardiovascular system supplies blood to the body's tissues, ensuring a continual flow of oxygen and nutrients to every cell. The cardiovascular system consists of the heart, a muscular pump that propels the blood, and blood vessels that convey the blood throughout the body. This chapter briefly reviews the normal structure and function of the cardiovascular system, then discusses the diseases of the system. Diseases involving the cardiovascular system may be classified into one of five categories:

1. congenital heart disease
2. inflammatory heart disease
3. ischemic vascular disease
4. hypertensive disease
5. metabolic disease

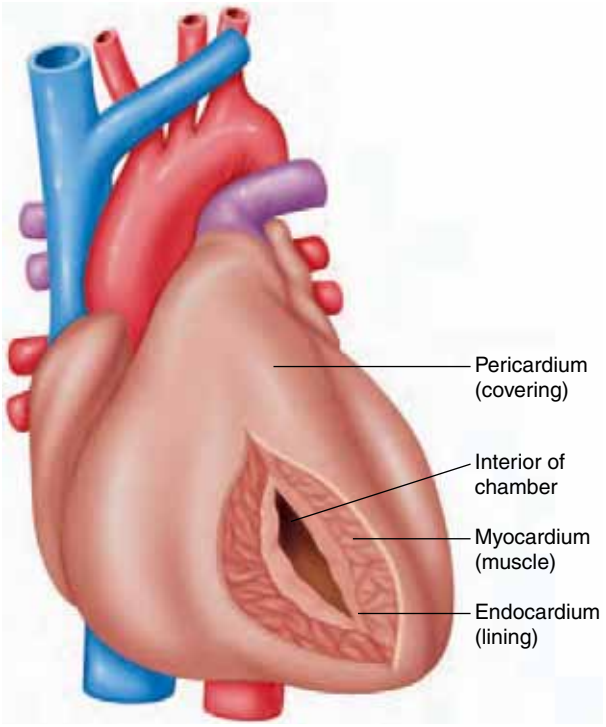

Figure 7–1 Heart covering and layers of the heart.

Structure and Function of the Heart

The heart is a hollow muscular organ located in the center of the chest. The heart consists of four chambers: a right and left atrium and a right and left ventricle. The chamber walls consist of cardiac muscle, known as **myocardium**, and their internal lining consists of a smooth, delicate membrane called the **endocardium**, which is continuous with the lining of the blood vessels. The **pericardium**, a double-layered membrane, encloses the heart. Figure 7–1 shows the tissues of the heart.

The right and left sides of the heart have an upper atrium that collects blood from the body and the lungs and a lower ventricle that ejects blood throughout the body and the lungs.

Valves between the atria and the ventricles, the atrioventricular (AV) valves, permit one-way bloodflow from atria to ventricles. The **mitral valve** between the left atrium and left ventricle has two flaps called cusps that meet when the valve is closed. The **tricuspid valve** between the right atrium and right ventricle is named for its

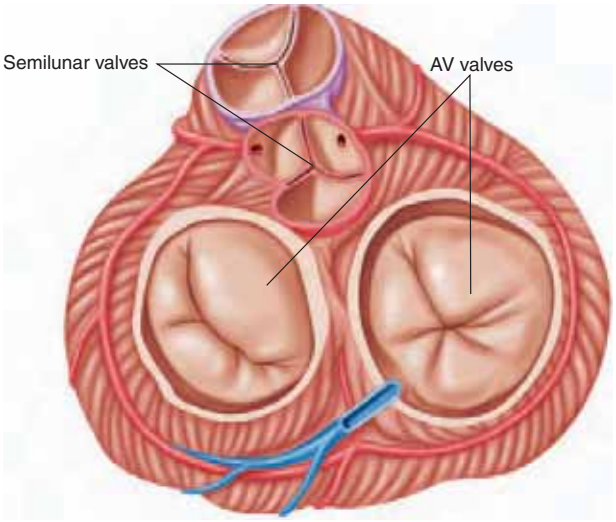

Figure 7–2 Heart valves in closed position viewed from the top.

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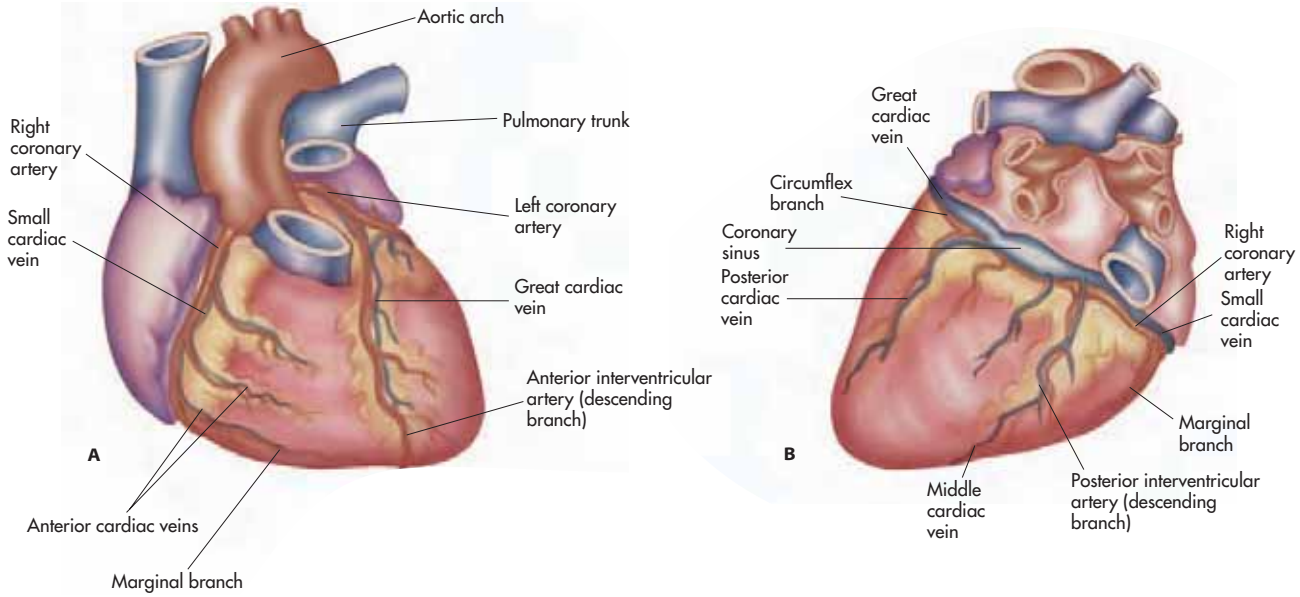

Figure 7–3 Coronary arteries and major blood vessels.

three cusps. Figure 7–2 shows these valves in the closed position.

The pulmonary semilunar valve permits one-way bloodflow from the right ventricle to the pulmonary artery, while the aortic semilunar valve controls bloodflow from the left ventricle to the aorta.

During every heart cycle, each heart chamber relaxes as it fills, and then contracts as it pumps blood. This filling period is the **diastole**, or the diastolic phase, while the contracting phase of each chamber is the **systole**, or systolic phase. The alternate contraction and relaxation of atria and ventricles comprises the **cardiac** cycle, which takes about 0.8 of a second. The flow of blood through the heart chambers, vessels, and lungs is reviewed in Figures 7–3 and 7–4.

Coronary arteries provide the heart muscle with a reliable blood supply. The left coronary artery begins at the aorta on the front of the heart and divides within an inch into the anterior interventricular coronary artery and the circumflex artery, which continues left around the back of the heart. The right coronary artery also branches from the front of the aorta and sends divisions to the right side and back of the heart (Figure 7–5).

Unlike skeletal muscle, cardiac muscle contracts continuously and rhythmically without conscious effort. A small patch of tissue, the **sinoatrial node** (SA node), acts as the pacemaker of the heart. The impulse for contraction initiates at the SA node, spreads over the atria, and passes to the ventricles via conductive tissue called the atrioventricular (AV) node. The impulse continues along left and right bundle branches, and terminates in the **Purkinje fibers**, which further branch throughout the ventricle walls. This conduction system is illustrated in Figure 7–6.

Heart muscle does not depend on nerve stimulation for contraction, but it is influenced by the autonomic nervous system and hormones such as epinephrine. Two sets of nerves work antagonistically, one slowing the heart and the other accelerating it. The vagus nerve slows heart rate during rest and sleep by means of a chemical it secretes, acetylcholine. The excitatory portion of the autonomic nervous system increases heart rate during periods of stress, strenuous physical activity, and

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excitement. This excitation is brought about by the release of epinephrine and its cousin norepinephrine, which stimulate the heart's pacemaker.

Blood flows through two circulatory routes: the systemic circulation and the pulmonary circulation. The systemic circulation distributes oxygenated blood from the left ventricle, beginning at the **aorta** and continuing through arteries to all parts of the body, and returns deoxygenated blood by veins to the right atrium. The pulmonary circulation carries deoxygenated blood from the right ventricle, beginning at the pulmonary trunk and continuing through smaller arteries to the lungs to be oxygenated, and returns the blood through pulmonary veins to the left atrium (Figure 7–7). Partitions called the interatrial septum and interventricular septum separate oxygenated from deoxygenated blood in the atria and ventricles respectively.

Branches of the aorta carry blood to the head, upper extremities, chest, abdomen, pelvis, and lower extremities. These arteries continue to divide into smaller and smaller arteries, and eventually into vessels called arterioles, the smallest arteries. Arterioles lead into capillaries, the connecting links between arteries and veins. Capillaries deliver oxygen and nutrients to tissues. Blood continues into venules, the smallest veins, and then into larger veins. Veins from the

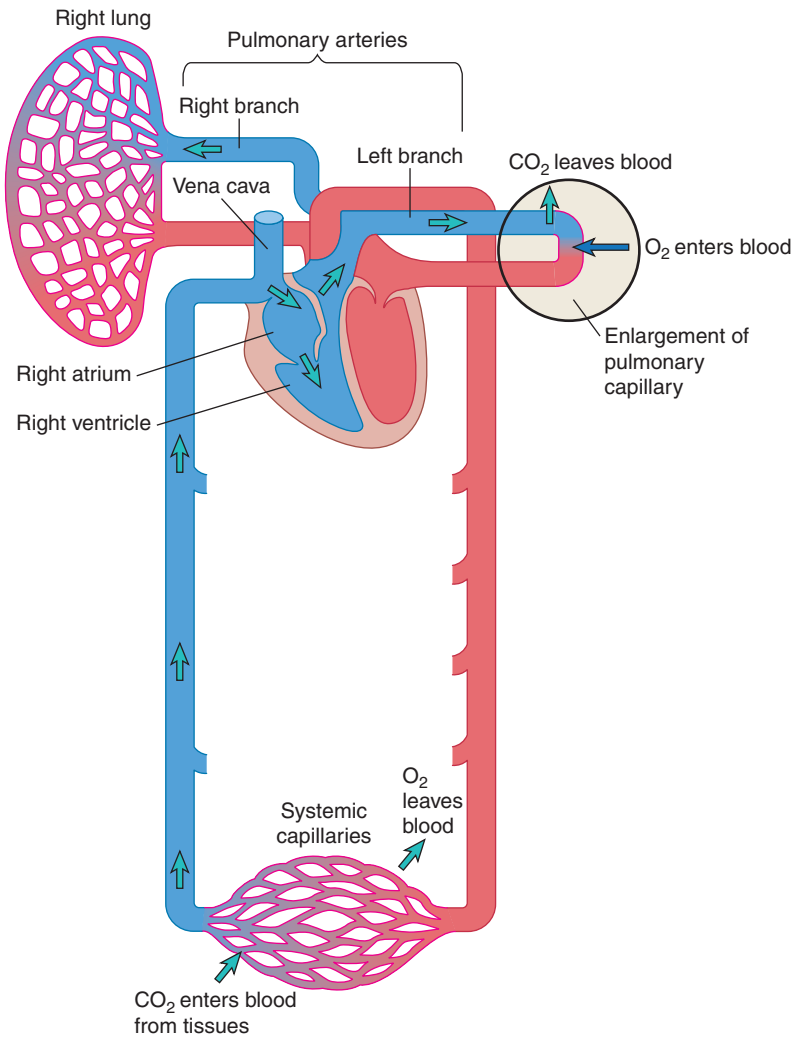

Figure 7–4
Venous return to the heart and bloodflow to the lungs.

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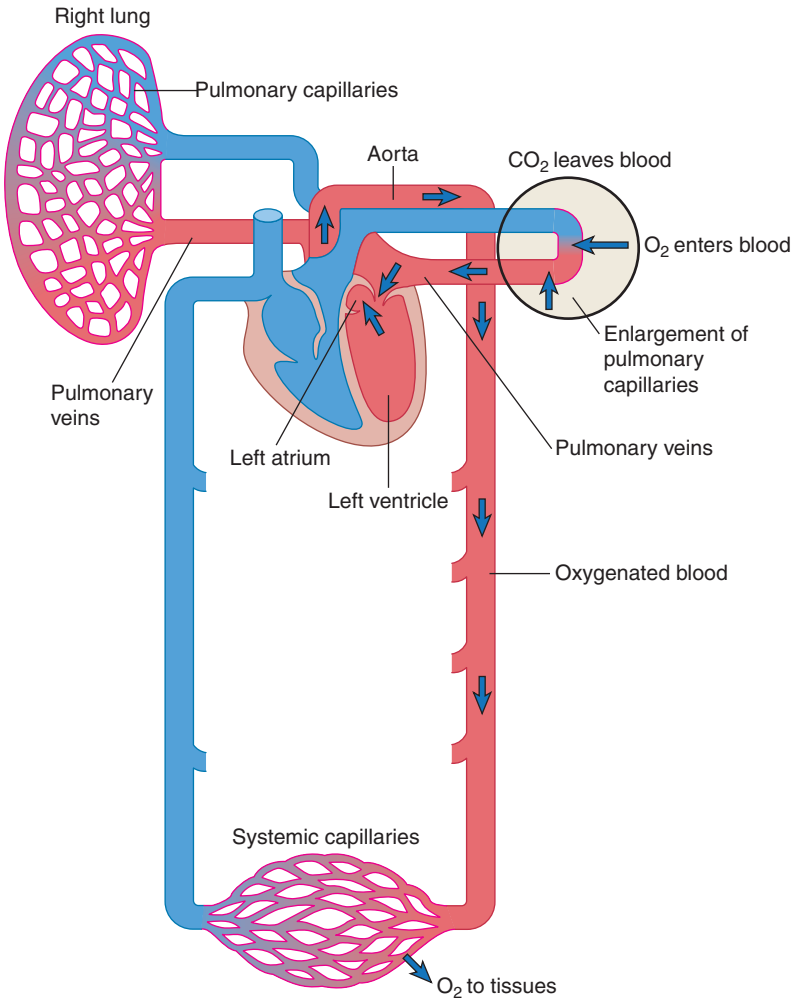

Figure 7–5
Return of oxygenated blood to heart and entry into aorta (red = oxygenated blood, blue = deoxygenated blood).

upper body empty blood into the superior vena cava, and veins of the lower body carry blood to the inferior vena cava. The superior and inferior **venae cavae** deliver systemic blood to the right atrium.

Structure and Function of the Blood Vessels

The walls of arteries are muscular, thick, and strong, with considerable elastic tissue, and are lined with endothelium. Arterioles have a smaller diameter than arteries, with thinner walls consisting mostly of smooth muscle fibers arranged circularly, and a lining consisting of endothelium. Arterioles can change their diameter by constricting or dilating, which alters bloodflow to the tissues. Capillaries are minute vessels about 1/2 to 1 mm long with a lumen as wide as a red blood cell. Their wall consists only of a layer of endothelium. Vein walls are much thinner than companion arteries, but their lumens are considerably larger. With less muscle and elasticity in their walls, veins tend to collapse when empty. Veins, particularly those of the legs, contain valves that help return blood upward to the heart against gravity.

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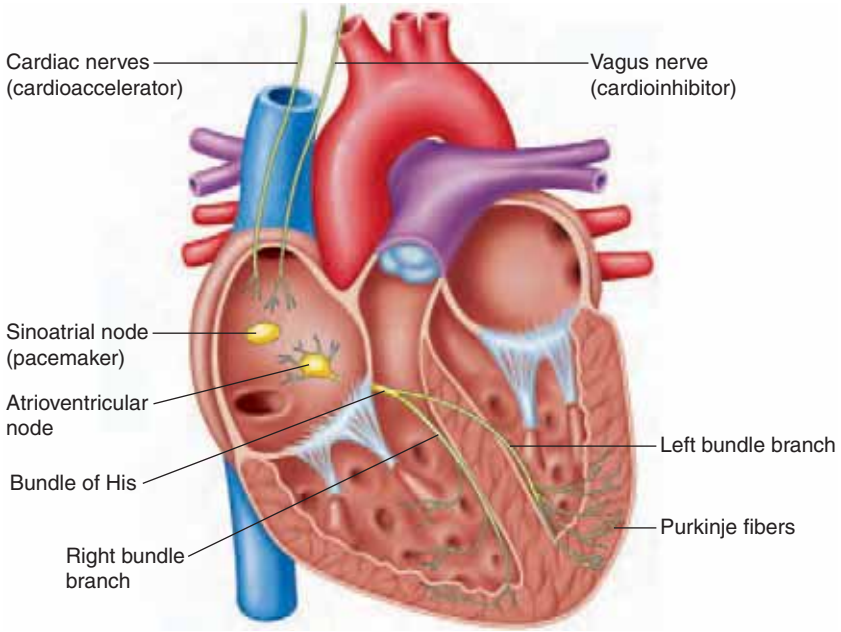

Figure 7–6
Conducting system of the heart.

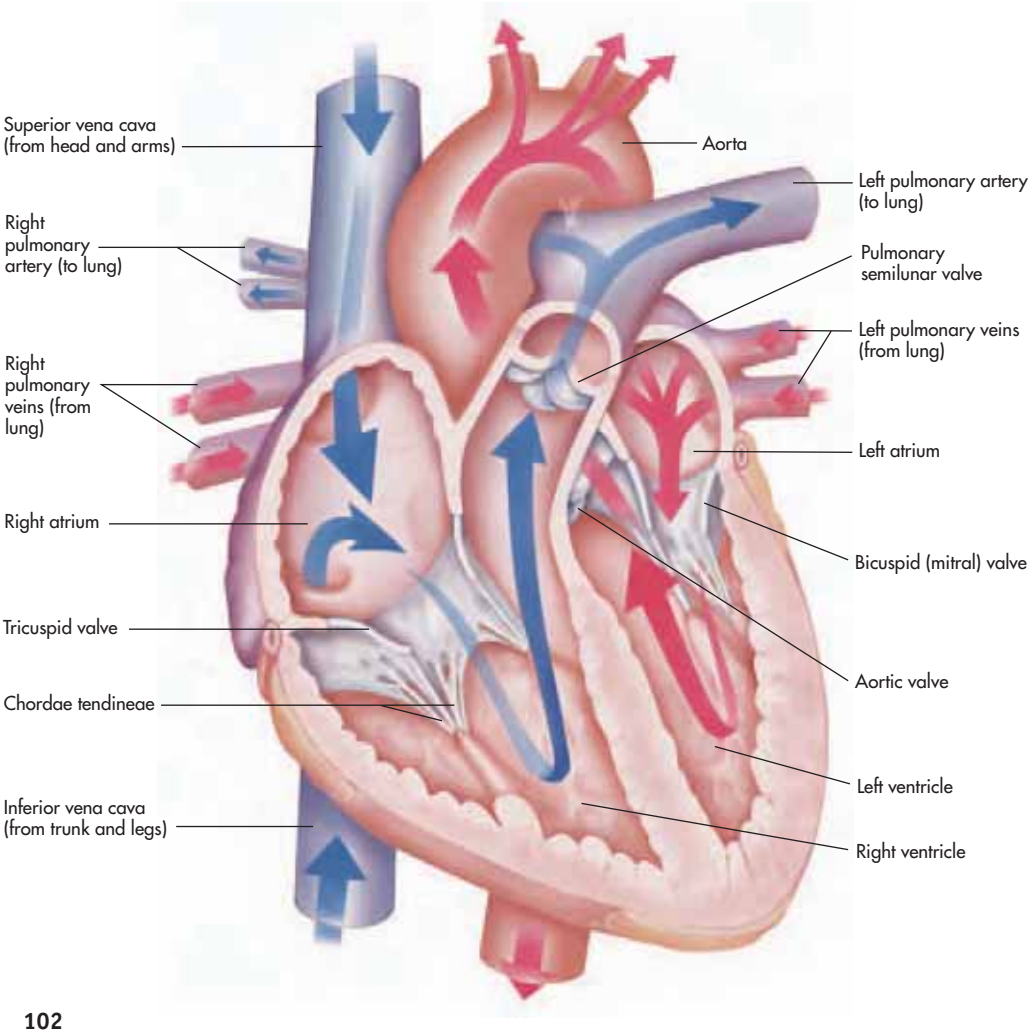

Figure 7–7
The bloodflow through the heart.

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Arteriosclerosis

Arteriosclerosis and atherosclerosis are diseases of the arteries. Because these diseases significantly contribute to the development of many other diseases in the cardiovascular system, most notably heart disease, they are discussed first.

In **arteriosclerosis**, artery walls thicken and become hard and inflexible, partly due to calcium deposition. "Hardening of the arteries" aptly describes this condition, because affected arteries are unable to stretch and rebound in response to the pressure of blood as it is forced through them by contraction of the heart. As a result, arteriosclerosis leads to hypertension. The most common cause of arteriosclerosis is atherosclerosis (discussed next) in which fatty material accumulates within the walls of the artery (Figure 7–8).

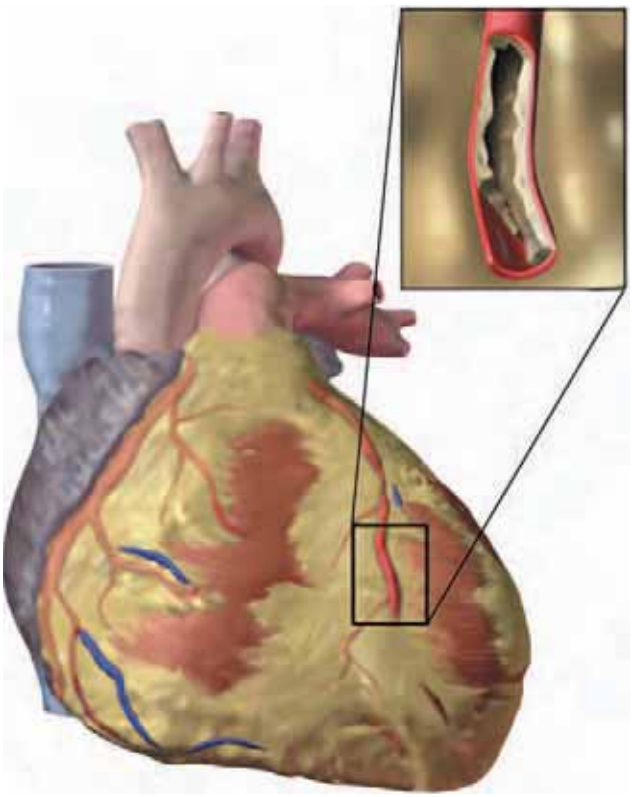

Figure 7–8 An atherosclerotic artery.

Atherosclerosis

Atherosclerosis is characterized by deposition of fatty material within artery walls. The artery walls thicken, narrowing the lumen and reducing bloodflow. In some instances, the lumen becomes completely blocked (occluded).

Atherosclerosis begins with inflammation in the artery wall, which might be triggered by a tear, infection, or chronic damage from hypertension. In response, monocytes accumulate under the inner lining of the arterial wall, forming an atheroma (Figure 7–8). The cells begin accumulating fat and form fatty deposits called **plaques**. This fatty material consists mostly of cholesterol and may also contain complex carbohydrates, blood clots, fibrous tissue, and calcium deposits. Although all arteries may be affected, typically atherosclerosis develops in the aorta and its branches as well as smaller coronary and cerebral arteries (Figure 7–9). If occluded, bloodflow to an organ is interrupted, a condition called **ischemia**, which is especially dangerous for heart and brain tissue. Ischemia is the basis for heart attacks and strokes, among other diseases.

Heredity plays a role in atherosclerosis, and it is a common complication of diabetes. Atherosclerosis is also associated with a sedentary lifestyle, a diet high in fat and cholesterol, and obesity. A low-cholesterol diet and regular exercise reduces the risk of developing atherosclerosis. The role of atherosclerosis and arteriosclerosis in heart disease is discussed later in this chapter. Table 7–1 summarizes the interplay between artery disease, various risk factors, and heart disease.

Diseases of the Heart

Coronary Artery Disease

Cardiac muscle receives a fraction (about 5%) of the blood flowing through the atria and the ventricles. Coronary arteries arising from the aorta supply the heart with oxygen-rich blood, and cardiac veins return oxygen-depleted blood to the right atrium. Unfortunately, these small

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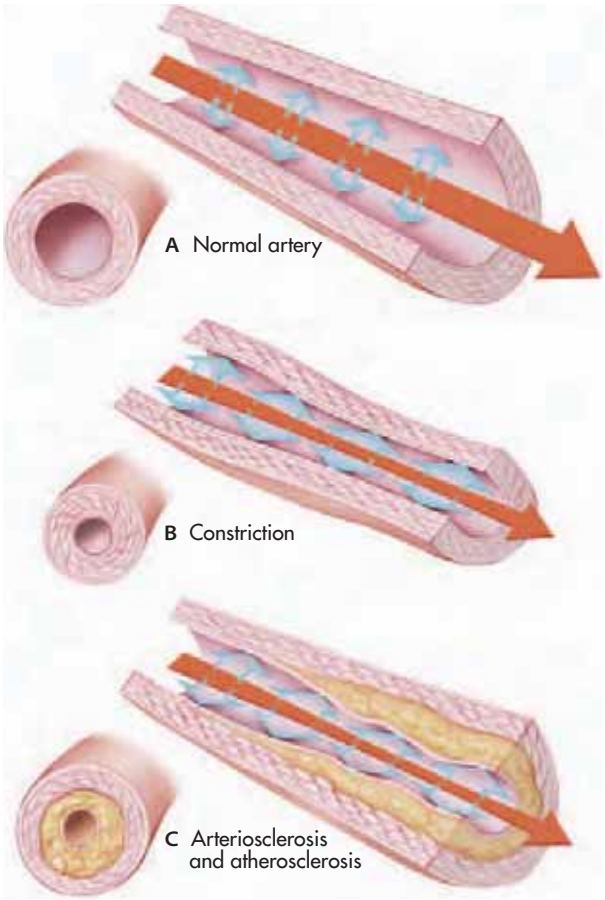

Figure 7–9 Blood vessels: (A) normal artery; (B) constriction; (C) arteriosclerosis and atherosclerosis.

vessels can become occluded by blood clots or by the narrowing of the lumen caused by atherosclerosis. Figure 7–10 illustrates the sequence of events leading to artery occlusion.

Ischemia to cardiac muscle can result in a heart attack. Lack of oxygen and nutrients quickly kills cardiac muscle, and because this dead tissue is called an **infarct**, a heart attack is known as a **myocardial infarction**. Heart attacks and other heart diseases are the leading cause of death in the United States. Although treatment for acute myocardial infarction has improved, heart attacks continue to take their toll. Survival rates for MI are shown in Figure 7–11.

Angina pectoris is temporary chest pain caused by transient oxygen insufficiency. Angina is characterized by pain and a sensation of pressure below the breastbone, which may radiate to the neck, jaw, and arms, and a feeling of tightness and suffocation. Angina is triggered by physical activity, lasts no more than a few minutes, and subsides with rest. Angina may also be triggered by heavy meals, exposure to cold, or emotional stress. Recurrent angina is described as chronic unstable angina. Treatment includes **nitroglycerin** administered in tablet form under the tongue, which dilates coronary arteries, restoring adequate blood flow. Physical activity and stress should be monitored closely to prevent angina attacks. Transdermal nitroglycerin patches may reduce the frequency of episodes.

Angina is a sign that cardiac muscle is not receiving sufficient blood, but it should not be confused with a heart attack. Although severe chest pains generally accompany a heart attack, the pain may be sensed in the neck or left arm and may be accompanied by nausea, restlessness, cold sweats, vomiting, lightheadedness, and clammy skin. Like angina, heartburn may be confused with a heart attack, so it is important to recognize the signs and symptoms associated with these conditions (Table 7–2).

The prognosis for a myocardial infarction depends on many factors, including the speed with which medical attention is provided. **Cardiopulmonary resuscitation** (CPR) can maintain bloodflow and the oxygen supply to the brain and heart until emergency care is available. Among those who survive a heart attack, the prognosis varies, depending on the amount of cardiac muscle damage. Prognosis and survival rates are presented in Figure 7–11.

The extent of cardiac muscle damage in a heart attack depends on the number and size of the coronary vessels that are occluded. This can be determined by a diagnostic angiogram (discussed at end of this chapter). Because the dead cardiac cells release their contents into the blood, the severity of a heart attack can be determined by identifying the type and level of certain enzymes found in the blood (Figure 7–12).

The damaged cardiac muscle repairs itself with scar tissue. The damaged myocardium is susceptible to rupture, which is a dangerous complication, so rest is required for adequate

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Table 7–1 Risk Factors for Heart Disease

Risk Factor	Indicators of High Risk	Role in Heart Disease
Blood pressure	>140/90 mm Hg	Stresses heart muscle and damages arteries
Cholesterol	0.240 mg/100mls blood	Forms plaque in coronary artery walls
Diabetes	Insulin insensitivity	Hypertension damages vessels
Smoking	Current or past heavy cigarette use	Inflames, narrows arteries
Calcium deposits	Deposits detected by CT scans	Hardens coronary arteries
C-reactive protein(CRP)	>3.0mg/L blood	Indicator of inflammation
Fibrinogen	Elevated concentration in blood	Promotes abnormal clotting
Homocysteine	Elevated concentration in blood	Linked to artery damage
Lipoprotein(a)	Increased blood clots	Induces atherosclerosis

Source: Modified from *Science News 31,* January 2003, Heart-Health Indicators

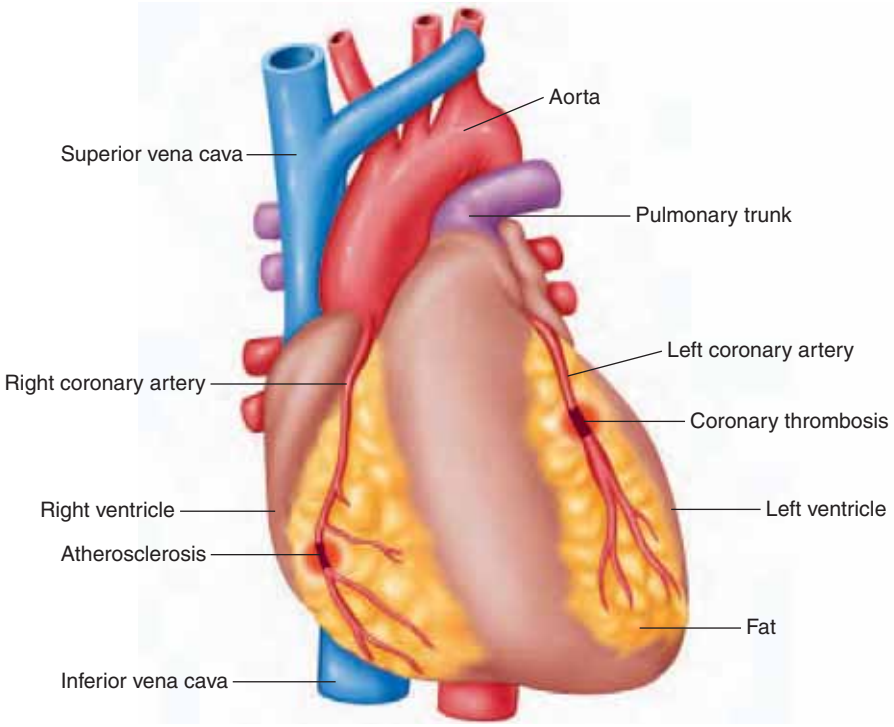

Figure 7–10
Blockage of coronary arteries.

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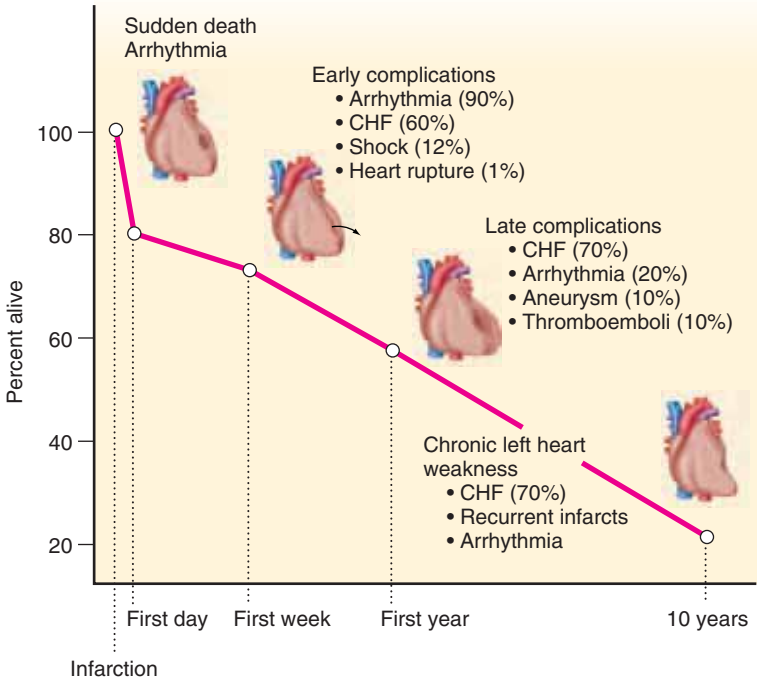

Figure 7–11
Outcome of myocardial infarction. Forty percent of one-week survivors have late complications resulting in death. Ten-year survival is about 25%.

healing. After healing, controlled exercise is advised to maintain circulation.

Advances in the treatment of heart attacks have increased survival and recovery rates. Severe damage to heart muscle has been reduced by early administration of **thrombolytic** (blood clot-dissolving) drugs, including TPA (tissue plasminogen activator) and streptokinase. Anticoagulants such as aspirin and coumadin may reduce the risk of a second heart attack.

Angioplasty can also open a partly occluded artery. The procedure involves inserting a balloon-tipped catheter into the femoral artery, guiding it to the heart and into the narrowed

Table 7–2 Signs and Symptoms of Heartburn and Heart Attack

Heartburn	Heart Attack
Burning, irritation below breastbone	Crushing pressure and pain on chest in men, but not common in women
Central chest pain	Pain sensed in shoulders, neck, arm, jaw, especially in women
Usually occurs after meals	Irregular heartbeat
Gets worse when lying down	Shortness of breath, fatigue
Antacids help reduce or stop pain	Cold sweats
Rarely causes dizziness or shortness of breath	Nausea and paleness, especially in women; vomiting, lightheadedness, weakness, dizziness

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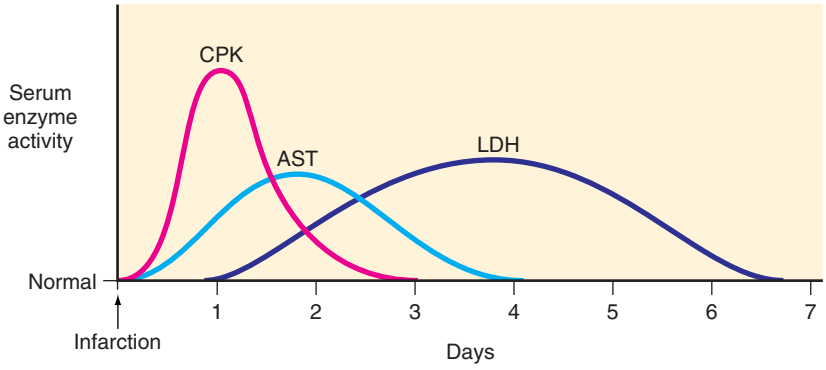

Figure 7–12
Serum enzyme level patterns used to diagnose myocardial infarction. AST = aspartate aminotransferase, CPK = creatine phosphokinase, and LDH = lactate dehydrogenase.

coronary artery. The balloon is expanded to press against the vessel walls and open the lumen. A stent, which is a cylindrical wire mesh of stainless steel or other alloy, surrounds the balloon. Expansion of the balloon forces the mesh into the lining of the vessel, which physically holds the lumen open. Because the vessels commonly become occluded again (restenosis) within months or a year, stents are coated with drugs that prevent restenosis. Treatment of a heart attack may require coronary bypass surgery in which a portion of the saphenous vein of the leg is removed and surgically attached to route blood around the occluded section of a coronary artery.

Cor Pulmonale

Cor pulmonale is a life-threatening condition characterized by right-side heart failure. Cor pulmonale follows chronic lung disease and respiratory failure and results from increased pressure in the blood vessels, a condition called pulmonary hypertension. As hypertension stresses the right ventricle, the ventricle dilates, enlarges, and eventually fails. Treatment is aimed at the underlying lung disease.

Congestive Heart Disease

Congestive heart disease is a progressive decrease in the ability of the heart to contract. Congestive heart disease can be caused by many diseases that damage the heart and interfere with circulation, including coronary heart disease, infection, heart valve disorders, and hypertension.

Congestive heart disease may involve the right or left side of the heart. Right-sided heart disease results in a build-up of blood flowing into the right side of the heart, causing edema of the ankles, distention of the neck veins, and enlargement of the spleen. Left-sided heart failure leads to a build-up of fluid in the lungs, a serious condition called **pulmonary edema**, which causes shortness of breath. Figure 7–13 shows the effect of each type of congestive heart failure.

Congenital Heart Disease

Most congenital heart abnormalities occur in the septum that separates the right and left sides of the heart. An opening in this septum allows a mixing of deoxygenated and oxygenated

Prevention PLUS!

Coronary Heart Disease

Women can reduce their risk of coronary heart disease by 30 to 40% by walking briskly for three or more hours each week.

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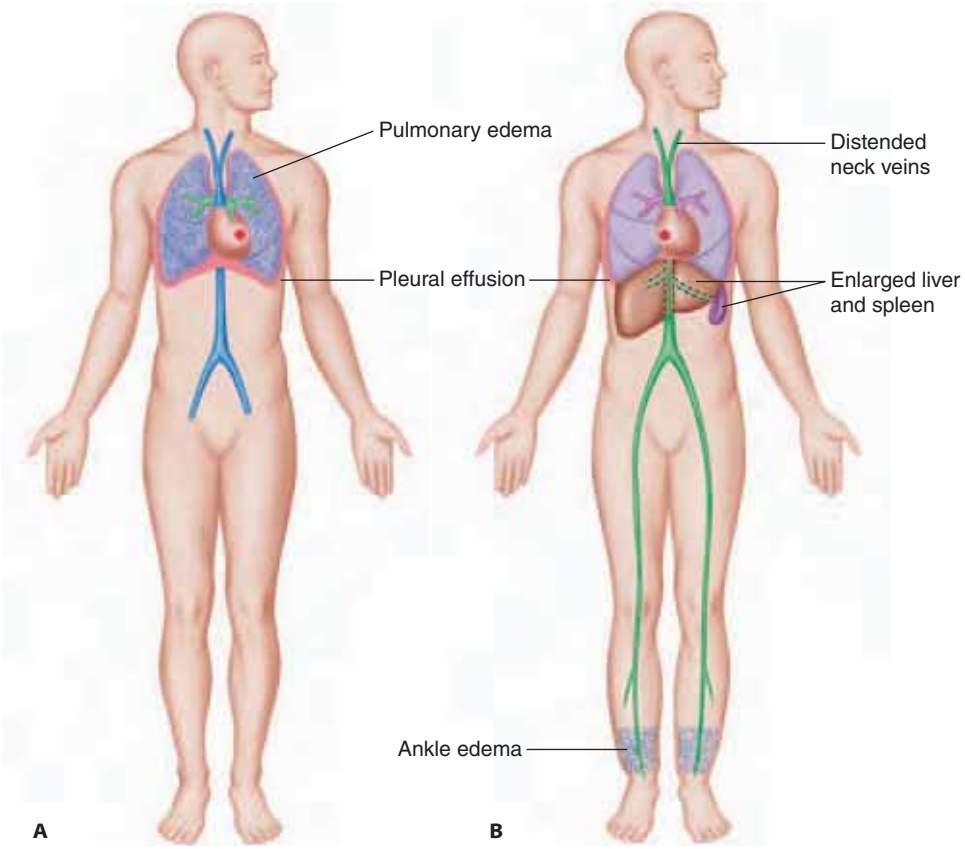

Figure 7–13 (A) Left-sided congestive heart failure. (B) Right-sided congestive heart failure.

blood, which stresses the heart as it attempts to compensate for lower oxygen levels.

Septal defects may be large or small, with the smaller defects causing virtually no problem. An example of a small septal defect is failure of the foramen ovale to close after birth. The **foramen ovale** is a small, natural opening that allows blood from the right side of the heart to enter the left directly, bypassing the nonfunctional fetal lungs. Failure of this opening to close at birth is the most common but least serious septal defect.

Septal defects may occur between the two atria, an atrial septal defect (ASD), or between the ventricles, a ventricular septal defect (VSD), which may be large. Because blood pressure is higher on the left side than on the right side of the heart, blood is generally shunted through the opening in a left-to-right direction. This shift increases the workload of the right ventricle,

Prevention PLUS!

Dietary Salt and Congestive Heart Disease

A low-salt diet can decrease blood volume and fluid accumulation in congestive heart failure. Dietary sodium can easily be reduced to 2 to 4 g per day by eliminating cooking salt.

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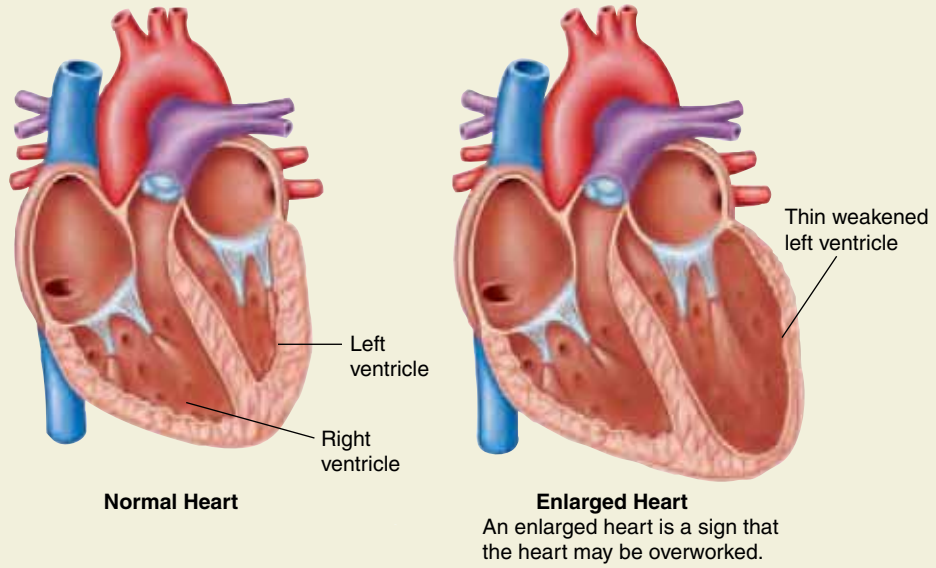
SIDE by SIDE ■ Hypertrophy



Normal Heart

▲ Normal ventricular wall.

Enlarged Heart
An enlarged heart is a sign that the heart may be overworked.

▲ Hypertrophied ventricular wall.

which already receives blood from the venae cavae. To accommodate the increased blood volume, the right ventricle enlarges (hypertrophies).

Cyanosis, a blue color in the tissues, does not occur if the shunt of blood through the septal defect remains left to right. If the pressure becomes greater in the right ventricle than in the left, the shunt reverses and cyanosis occurs. The deoxygenated blood from the right side of the heart then enters the general circulation, causing cyanosis (Figure 7–14).

Tetralogy of Fallot is one of the most serious of the congenital defects and consists of four abnormalities. The baby with this condition is born cyanotic, with all the tissues a definite blue. The first abnormality is **pulmonary stenosis**, a narrowed pulmonary artery, which prevents blood from reaching the lungs to be oxygenated. The second abnormality is a large ventricular septal defect. The third defect is right ventricle hypertrophy that results from the pulmonary stenosis. The fourth abnormality is a misplaced aorta that crosses the interventricular septum. Normally, only oxygenated blood from the left ventricle enters the aorta, but in this case, the right ventricle also feeds into the aorta, permitting the mixing of oxygenated and deoxygenated blood.

Other abnormalities develop as a result of these heart defects, including secondary polycythemia, a disease described in Chapter 8, which occurs to compensate for the low oxygen level. Clubbed fingers and curled fingernails develop because of poor oxygen supply to tissues at the fingertips. As a result, a child may experience **dyspnea**, difficulty breathing, after any exertion, even crying.

Surgical repair consists of patching the ventricular septal defect, opening the narrowed pulmonary valve, and closing any abnormal connection made between the aorta and the pulmonary artery. Figure 7–15 shows the four abnormalities in the tetralogy of Fallot.

Patent ductus arteriosus (PDA) is a common congenital disease in which a fetal blood vessel that connects the pulmonary artery and the aorta persists after birth. In a fetus, this vessel

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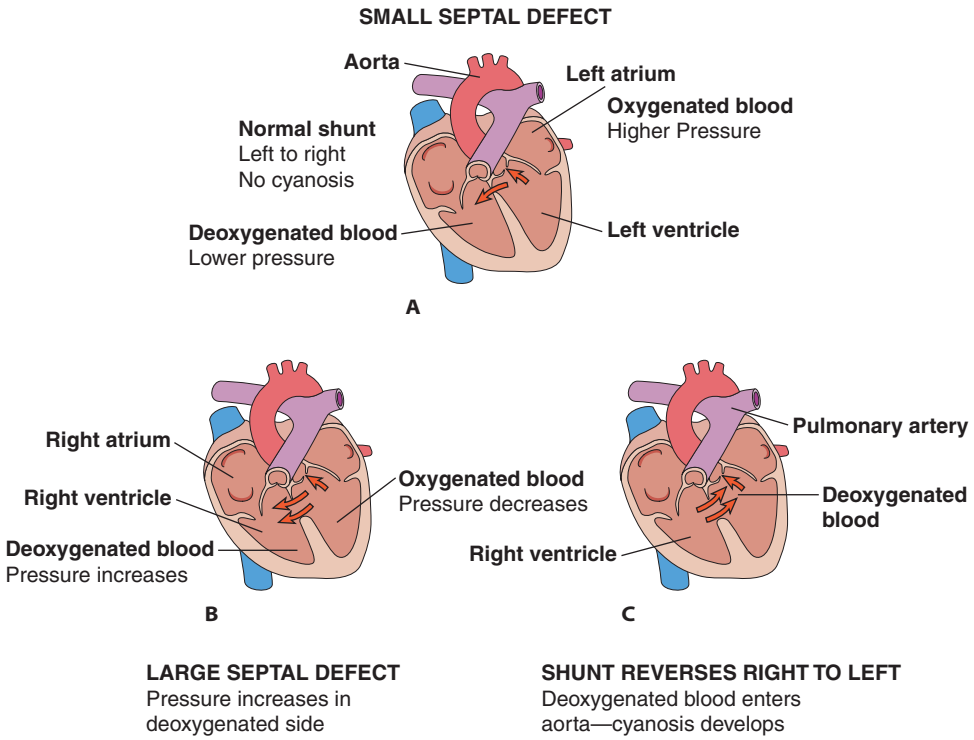

Figure 7–14 Effect of septal defects. (A) Normal shunt: no cyanosis. (B) Increased pressure in right ventricle. (C) Shunt reversal: cyanosis develops.

shunts blood away from the nonfunctional fetal lungs (Figure 7–16), but after birth, it normally closes. If it remains open, blood intended for the body flows from the aorta to the lungs, overloading the pulmonary artery. Because this blood is oxygenated, there is no cyanosis. PDA is associated with a risk of heart failure and infection at the site of the lesion. The PDA may be closed surgically.

Coarctation of the aorta is a narrowing, or stricture, of the artery that provides blood to the entire body. The stricture occurs beyond the arterial branches to the head and arms, so the blood supply to the upper part of the body is adequate. Bloodflow is reduced, however, to the abdomen and legs, resulting in significantly lower blood pressure in the legs. Blood pressure remains high in the arms. Many collateral blood vessels develop to compensate for the poor blood supply to the legs. The coarctation can be corrected surgically by cutting out the narrowed segment of the aorta and rejoining the healthy ends.

Valve Disorders

Valves maintain unidirectional flow of blood through the heart. Valve disorders include **stenosis** and **valvular insufficiency**. Because valve disorders affect bloodflow, valve defects cause **heart murmurs** with characteristic sounds that indicate the nature of the defect.

In **mitral stenosis**, the mitral valve opening is narrow, and the cusps that form the valve, normally flexible flaps, become rigid and fuse together. A deep funnel shape develops, increasing resistance to bloodflow from the left atrium to the left ventricle. As back-pressure develops in the left atrium, it becomes hypertrophied. The right side of the heart is also affected (Figure 7–17). Pressure within the heart makes it difficult for the pulmonary veins to deliver blood to the right atrium, leading to increased pressure within veins. As the congestion builds in the veins, fluid from the blood leaks out into the tissue spaces, causing edema. Poor circula-

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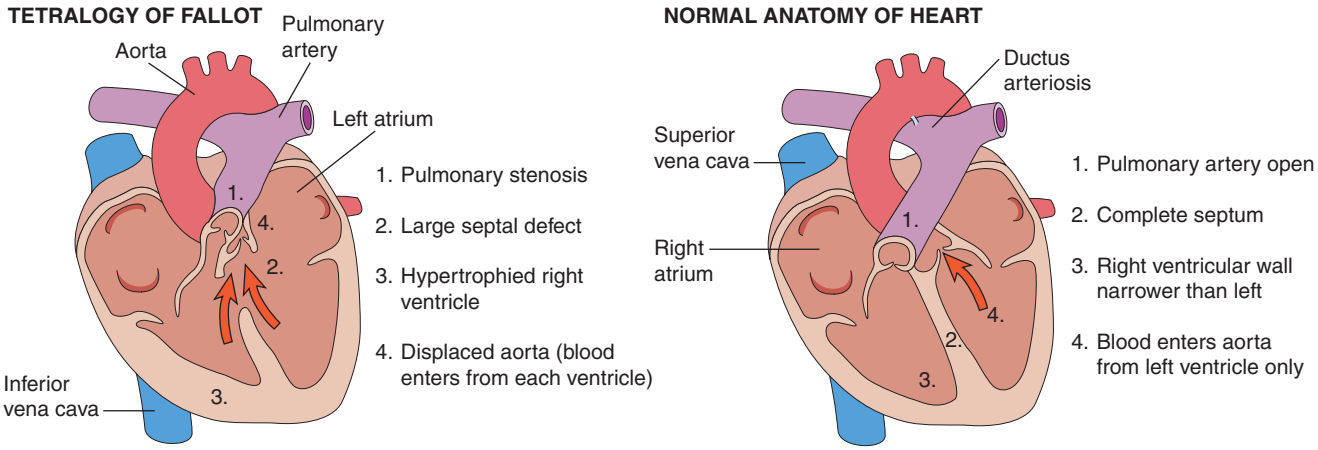

Figure 7–15 Tetrology of Fallot (left) compared to normal anatomy (right).

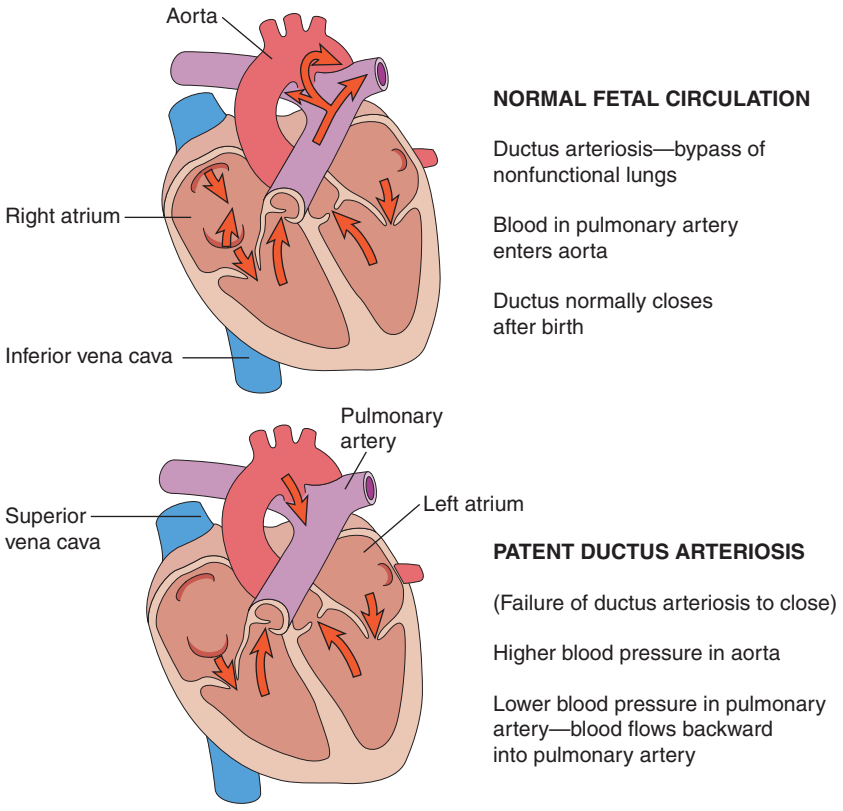

Figure 7–16
Patent ductus arteriosus.

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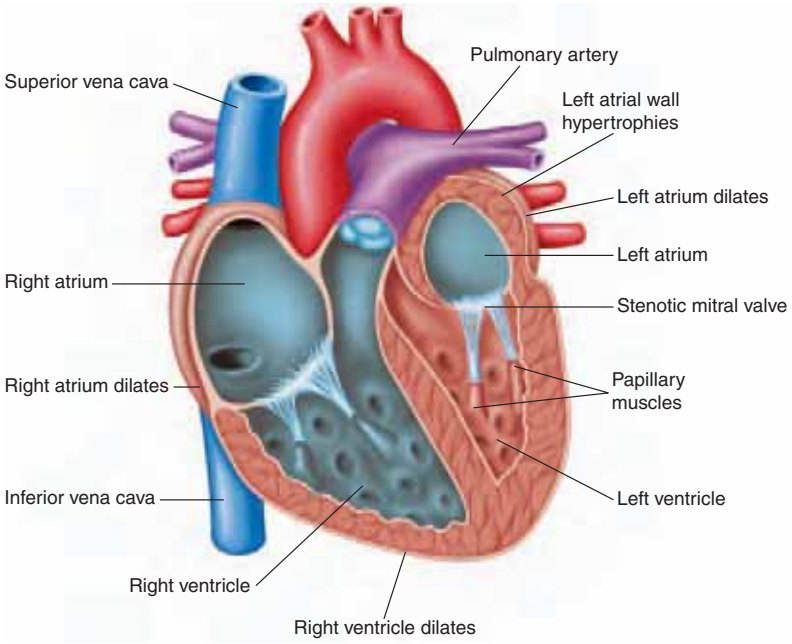

Figure 7–17
Effect of mitral valve stenosis on the heart.

tion causes cyanosis because an inadequate amount of oxygen is reaching the tissues. The backup of blood and congestion cause the heart to become exhausted and may lead to congestive heart failure. Another complication of a valve defect is the increased risk for a thrombus (blood clot) to form on the valve. If the thrombus becomes detached, it travels as an embolism and may occlude a blood vessel supplying the brain, kidney, or other vital organ. Mitral stenosis often follows rheumatic fever and is more common in women than in men.

Stenotic valves may be widened to restore bloodflow with valvuloplasty, a surgical technique similar to angioplasty. A complication of the surgery may be a leaky valve, in which case the valve may be surgically replaced with metal alloy or pig valve.

In mitral insufficiency, also called mitral incompetence, the valve is unable to close completely, which allows blood to leak into the atrium each time the ventricle contracts. As the volume of blood and pressure in the left atrium increases, blood pressure increases in other vessels, including the vessels leading from the lungs to the heart, resulting in lung congestion. The insufficiency is exacerbated by sclerosis and retraction of the valve cusps.

Another cause of insufficiency is the failure of specialized valve muscles in the ventricle, called papillary muscles. These muscles attach to the underside of the cusps by means of small cords (chordae tendinae) that normally prevent the cusps from flipping up into the atria when the ventricles contract. If the papillary muscles fail to contract, the cusps open upward toward the atria under the force of expelled ventricular blood. This failure is commonly called mitral valve prolapse (MVP).

Most individuals with MVP are asymptomatic and lead normal lives. Those who have moderate or more severe cases of MVP take antibiotics like amoxicillin to prevent bacteria from colonizing in the defective valves. If the prolapse becomes severe, it may be corrected with surgical reconstruction or replacement.

Aortic stenosis, the narrowing of the valve leading into the aorta, occurs more often in men than in women and most frequently in men over 50 years old. It may result from rheumatic fever, a congenital defect, or with arteriosclerosis. Aortic stenosis is characterized by rigid cusps that adhere together and deposits of hard, calcified material, giving a warty appearance to the valve. Because the left ventricle pumps blood through this narrowed valve into the aorta, this

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chamber hypertrophies. Even with enlarged ventricles inadequate bloodflow to the brain persists and can cause **syncope** (fainting). This valve defect, like others, can be corrected surgically.

In aortic insufficiency, the valve does not close properly. With each relaxation of the left ventricle, blood flows back in from the aorta. Backflow of blood causes the ventricle to dilate, become exhausted, and eventually fail. This condition can result from inflammation within the heart, endocarditis, or a dilated aorta.

Rheumatic Heart Disease

Rheumatic heart disease is a sequela of infection by group A **hemolytic streptococci** of skin, throat, or ear, although the organisms are no longer present when the disease presents itself. Approximately 2 weeks following the streptococcal infection, rheumatic fever develops, characterized by fever, inflamed and painful joints, and sometimes a rash.

Rheumatic fever is an **autoimmune disease** that results from a reaction between streptococcal antigens and the patient's own antibodies against them. All parts of the heart may be affected, frequently including the mitral valve. Blood clots deposit on the inflamed valves, forming nodular structures called **vegetations** along the edge of the cusps. The normally flexible cusps thicken and adhere to each other. Later, fibrous tissue develops, which has a tendency to contract.

If the adhesions of the cusps seriously narrow the valve opening, the mitral valve becomes stenotic. If sufficiently damaged, the cusps may not be able to meet properly, resulting in mitral valve insufficiency.

The incidence of rheumatic fever is highest among children and young adults. Prompt treatment of the streptococcal infection with antibiotics can prevent rheumatic fever and its complications.

Infectious Endocarditis

Infectious endocarditis was once considered fatal but responds well to antibiotics if treated early. Endocarditis is an inflammation of the internal lining of the heart commonly caused by a streptococcus. These organisms can enter the bloodstream from infections at sites such as a tooth, the skin, or the urinary tract. Various routes of bacterial invasion are illustrated in Figure 7–18. This inflammation occurs on previously damaged valves or congenital heart defects.

The nodules or vegetations that form in endocarditis are larger than those of rheumatic fever. They are also **friable**, tending to break apart easily and enter the bloodstream. These vegetations are filled with bacteria, unlike rheumatic fever vegetations. Typical lesions of endocarditis are shown in Figure 7–19. As fragments of the vegetations break apart, they enter

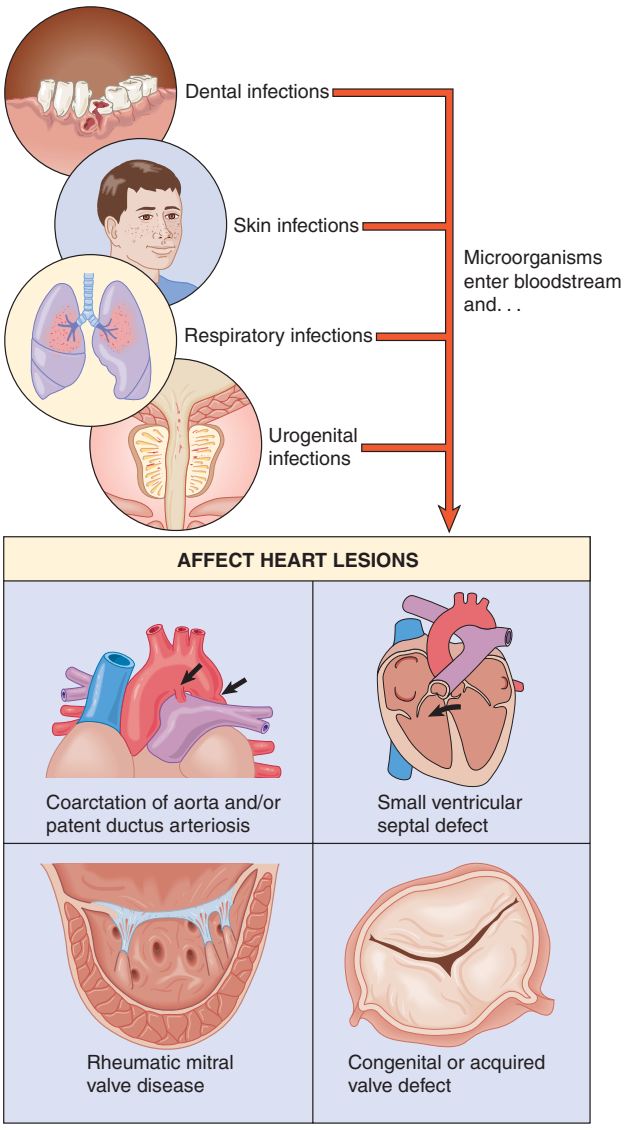

Figure 7–18 Infections resulting in bacterial endocarditis.

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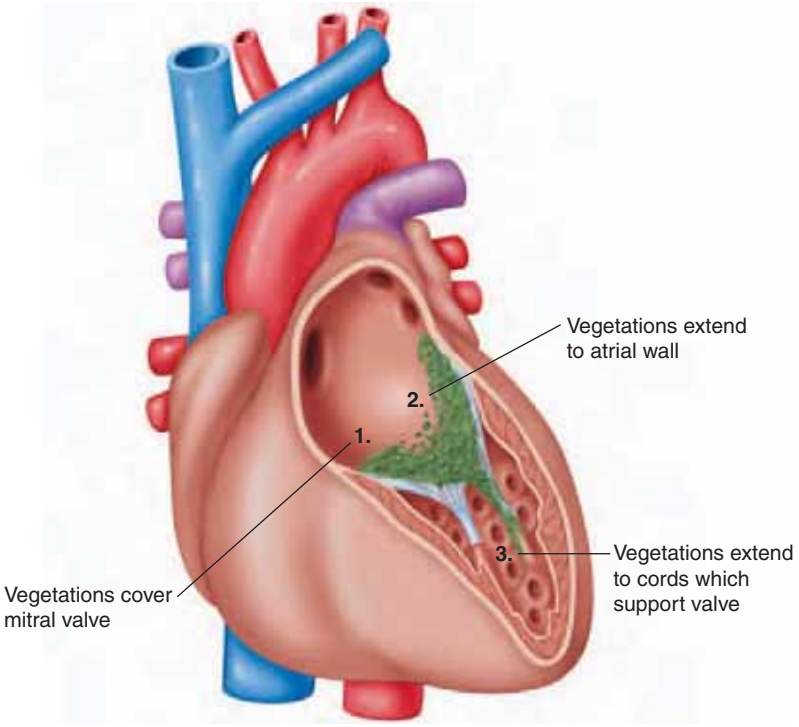

Figure 7–19
Bacterial endocarditis.

the bloodstream to form emboli, which can travel to the brain, kidney, lung, or other vital organs, causing a variety of symptoms. The emboli can lodge in small blood vessels of the skin or other organs and cause the blood vessels to rupture. These small hemorrhages produce tiny red spots called **petechiae**.

Vascular Diseases

Both arteriosclerosis and atherosclerosis were discussed at the beginning of this chapter. This section focuses on other diseases of the blood vessels.

Thrombosis and Embolism

Thrombosis, the formation of blood clots on blood vessel walls, is caused by slow bloodflow, and because blood flows more slowly in veins than in arteries, veins are common sites of thrombus formation. **Thrombi** are also likely to form where there is turbulence in the bloodflow, such as near heart valves, and a diseased valve is a likely site for a clot formation. Increased viscosity of the blood also leads to thrombosis. Dehydration, polycythemia (extra red blood cells), or high platelet levels increase viscosity and the tendency for thrombosis. Figure 7–20 illustrates thrombus formation.

If the thrombus breaks free, it becomes an embolus. These traveling clots typically lodge into the coronary arteries or lung or brain vessels. An embolism containing pyogenic bacteria is called a **septic embolism**. Should such an embolism lodge in a small vessel of the foot or leg, the tissue dies and becomes **necrotic**, leading to gangrene.

Anticoagulants prevent or reduce clot formation and promote bloodflow. Sepsis (blood infection) is a serious complication that occurs in 200,000 individuals annually in the United States.

Aneurysms

A weakening in the wall of a blood vessel can cause local dilation known as an **aneurysm**. Aneurysms most commonly occur in the abdom-

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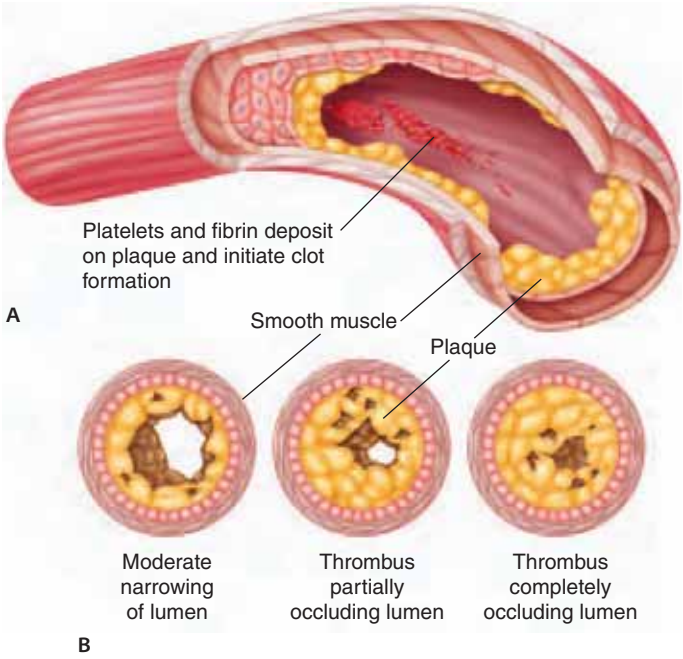

Figure 7–20
Thrombus formation in an atherosclerotic vessel. Depicted are the initial clot formation (A) and the varying degrees of occlusion (B).

inal aorta or brain and result primarily from arteriosclerosis. The danger of an aneurysm is the tendency to increase in size and rupture, resulting in hemorrhage, possibly in a vital organ such as the heart, brain, or abdomen.

Aneurysms usually produce no symptoms and are detected by an x-ray or routine physical exam. Ultrasound techniques can diagnose and measure aneurysms. A **computed tomography** or CT scan is accurate in determining the shape and size of an aneurysm. Early detection prevents rupture.

Surgical procedures have been very successful in repairing blood vessels affected by aneurysm. The diseased area of the vessel is removed and replaced with an artificial graft or segment of another blood vessel. This procedure reduces the risk of hemorrhage and thrombus formation.

Raynaud's Disease

Raynaud's disease is a condition in which small arteries or arterioles in the fingers and toes constrict. Symptoms are spasms including numbness, discoloration of the local skin of the fingers and toes, and pain (see Figure 7–21). Spasms are intermittent and are commonly triggered by cold. As vessels constrict, bloodflow temporarily decreases, causing the fingers and toes to turn white. As the episode resolves, the affected areas may turn pink or blue.

Raynaud's disease can be controlled by protection from cold. Smoking should be avoided, as it constricts blood vessels regardless of environmental conditions. Relaxation techniques can help reduce stress, which may bring about an attack.

Phlebitis

Phlebitis is inflammation of a vein. Any vein may be affected, but phlebitis usually occurs in deep veins of the leg. The greatest danger in the deep veins is thrombosis, a condition called **thrombophlebitis** (Figure 7–22). Edema develops once a vein becomes occluded because increased internal pressure causes fluid to leak out of the vessel. A major complication of thrombophlebitis is an embolism.

Causes of phlebitis include injury, infection, poor circulation, and obesity. Phlebitis is

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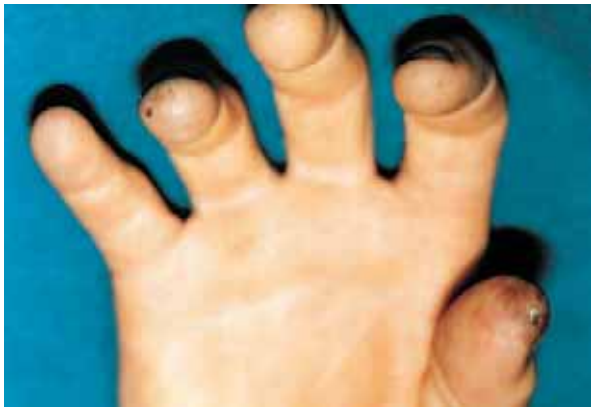

Figure 7–21 Raynaud's disease. (Courtesy of Jason L. Smith, MD)

treated with anticoagulants, including aspirin, and antibiotics. Surgery may be required to remove the thrombus.

Varicose Veins

Varicose veins are dilated, distorted veins that usually develop in the superficial veins of the leg, such as the greater saphenous vein. The veins become swollen and painful, and appear knotty under the skin. Varicose veins are caused by blood pooling within the veins because of decreased, stagnated bloodflow. Varicose veins can be an occupational hazard related to long periods of sitting or standing. Normally, the leg muscle movement moves blood up within the vein from one valve to the next. In the absence of this "milking action" of the muscles, the blood exerts pressure on the closed valves and thin walls of the veins. The veins dilate to the extent that the valves are no longer competent. The blood collects and becomes stagnant, and the veins become more swollen and painful.

Pregnancy or a tumor in the uterus can also cause varicose veins because pressure on veins causes resistance to bloodflow. Heredity and obesity are also associated with varicose veins. Figure 7–23 illustrates the flow of blood through normal veins and varicose veins.

Complications of varicose veins include ulcers and infection, due to poor circulation, and hemorrhage, caused by weakened vein walls.

Treatment depends on the severity of the symptoms. An elastic bandage or support hose may increase circulation and provide relief from discomfort. Symptoms can be relieved by walking, elevating the legs when seated, and weight reduction. A surgical procedure called "stripping the veins" is very successful. The surgery involves removing the veins and tying off the remaining open ends. Collateral circulation tends to develop to compensate for the loss of the vein segment.

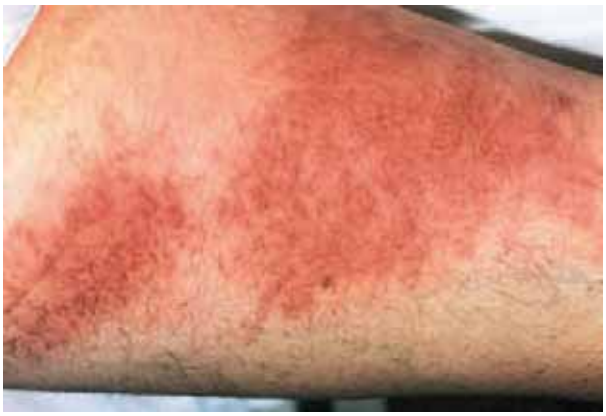

Figure 7–22 Thrombophlebitis. (Courtesy of Jason L. Smith, MD)

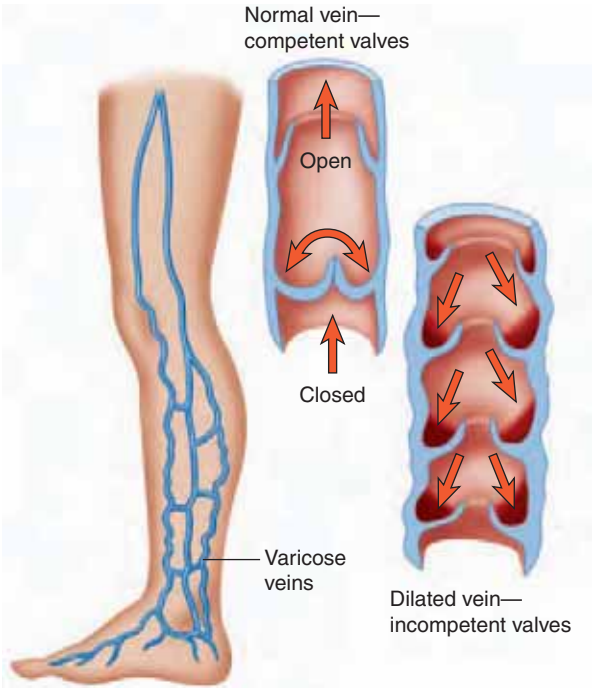

Figure 7–23 Development of varicose veins.

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Small, dense, red networks of veins, called **spider veins**, can be treated with laser. The light heats and scars the tiny superficial veins, which closes them off to blood flow (Figure 7–24). Another treatment is **compression sclerotherapy** in which a strong saline solution is injected into specific sites of the varicose veins. The irritation causes scarring of the inner lining and fuses the veins shut. The procedure is followed by uninterrupted compression for several weeks to prevent reentry of blood. A daily walking program during the recovery period is required to activate leg muscle venous pumps.

Hemorrhoids are varicose veins in the rectum or anus that cause pain, itching, and bleeding. Like varicose veins in the leg, hemorrhoids can develop from pressure on the veins. Straining due to constipation, pressure on the veins from a pregnant uterus, or a tumor may promote their development.

Esophageal varices, or varicose veins of the esophagus, frequently accompany cirrhosis of the liver. They result from pressure that develops within the veins as they try to empty. Because of blocked blood vessels within the damaged liver, there is a backup of blood and general congestion. The major complication from these varices is a fatal hemorrhage.

A relatively new procedure for treating esophageal varices is called **endoscopic sclerotherapy**. In this procedure, a retractable needle is guided into the esophagus by means of a fiberoptic endoscope. The gastroenterologist punctures the varicosities and injects a caustic sclerosis (hardening) solution to occlude the swollen veins. This prevents engorgement, rupture, and hemorrhage, or stops a hemorrhage that has already begun.

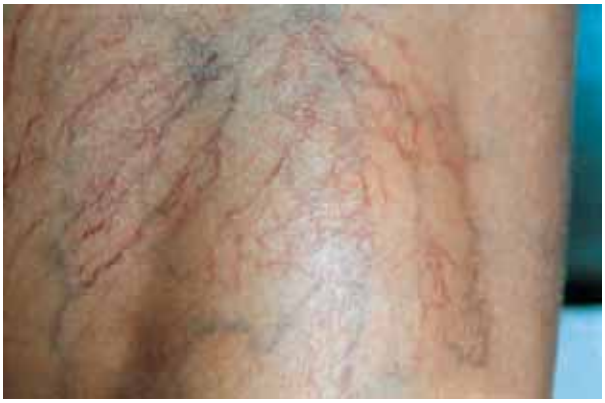

Figure 7–24 Spider veins. (Courtesy of Jason L. Smith, MD)

Hypertension

Hypertension is abnormally high blood pressure in the arteries. Called the "silent killer," hypertension is often asymptomatic and usually not diagnosed until complications arise. The causes of primary hypertension, also called essential hypertension, are not known, although the disease appears to be inherited. Primary hypertension is exacerbated by obesity, lack of exercise, and excessive alcohol and salt intake. Secondary hypertension results from other diseases, such as brain tumors, kidney disease, and endocrine disorders. Hypertension may have a gradual onset and continue for a long time, or it may be malignant, with sudden onset and rapid progression resulting in death if not treated immediately.

A blood pressure measurement consists of two values that correspond to the two phases of heart activity: the systolic and diastolic pressure. The systolic pressure is the highest pressure in the arteries caused by the force of contraction of the ventricles. Diastolic pressure corresponds to the pressure in the arteries when the ventricles are relaxing and refilling. The blood pressure of an average normal adult is 120 mm Hg (systolic) and 80 mm Hg (diastolic), often expressed as 120/80 mm Hg.

Blood pressure normally varies throughout the day, increasing with activity and decreasing with rest. Because of these fluctuations, high blood pressure is not considered abnormal unless there are three sequential elevated readings, each recorded at different times under similar conditions. Generally, pressures that are consistently greater than 140/90 mm Hg are considered high. Tables 7–3 and 7–4 show recent blood pressure guidelines.

The kidneys and the nervous system regulate blood pressure. Blood pressure increases are brought about by increased force of heart contraction and by constriction of arteries and arte-

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Table 7–3 Blood Pressure Guidelines

Healthy (normal)	Below 120/80 mm Hg
Prehypertension	120/80 to 139/89 mm Hg
Stage 1 hypertension	140/90 to 159/99 mm Hg
Stage 2 hypertension	160/100 mm Hg

rioles. As more blood enters the vascular system, the blood pressure increases. The kidney also increases blood pressure by secreting a substance called **renin**, which activates angiotensin, a hormone that causes the walls of the arteries to constrict and increases blood pressure. Angiotensin also triggers the release of another hormone, aldosterone, which causes the kidneys to retain salt (sodium) and water, expanding blood volume and increasing blood pressure.

When blood volume is high, the pressure in the arteries forces fluid through the walls of capillaries into tissue spaces, reducing blood volume and blood pressure. Dilation of arteries and excretion of excess fluid by kidneys reduce blood pressure.

Hypertensive Heart Disease

This condition is caused by chronic hypertension. Over time, the heart adapts to hypertension by enlarging. Enlargement fails to compensate, however, and the left ventricle eventually becomes so weak that it fails to pump blood adequately.

Table 7–4 Risk of Stroke and Heart Disease Increase with Increasing Blood Pressure

Blood Pressure	Risk
115/75	Normal
135/85	2 times normal
155/95	4 times normal
175/105	8 times normal

Hypertension and Kidney Disease

Hypertension contributes to kidney disease, and kidney disease contributes to hypertension. Decreased function of the kidneys leads to water and salt retention, causing increased blood volume and elevated blood pressure levels. Long-standing hypertension causes arteriosclerosis of the renal artery, which reduces blood flow to the kidneys and damages them.

Primary hypertension cannot be cured, but it can be treated to prevent complications. A combination of medication, diet changes, and exercise is the ideal method for controlling high blood pressure. Because there are usually no symptoms of high blood pressure, treatments that make people feel bad or interfere with lifestyle are avoided.

Overweight individuals are advised to reduce their weight. Changes in diet for those who have diabetes and high cholesterol levels are important for overall cardiovascular health. Cutting down on salt and alcohol intake may make drug therapy for high blood pressure unnecessary. Moderate exercise can help control weight and improve circulation.

Drug therapy can help reduce blood pressure. In general, drug therapy is used if lifestyle modification is not effective or produces an inadequate response. One or more drugs with different actions may be used to control blood pressure. For example, a **diuretic** like Lasix that controls blood volume in combination with a drug that blocks sympathetic stimulation may be prescribed. A drug that blocks the renin-angiotensin-aldosterone cascade is the ACE inhibitor (angiotensin-converting enzyme inhibitor). The best combination of drug therapy generally is one that fits the patient's lifestyle with minimal side effects.

Treatment of secondary hypertension depends on the underlying cause. For example, treatment of kidney disease, when recognized, can help normalize and lower blood pressure. If angiography reveals partially occluded vessels, then angioplasty may be used.

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Prevention PLUS!

Take Action to Reduce Your Blood Pressure and Save Your Life

Lose weight	Weight loss is the single most effective nondrug method to reduce blood pressure.
Exercise	Thirty to 35 minutes of exercise three times per week can decrease blood pressure, especially when combined with weight loss.
Limit alcohol	Alcohol raises blood pressure even without hypertensive disease.
Eat a low-fat, high-fruit and vegetable diet	A diet high in vitamins and low in fats is associated lower blood pressure.
Reduce dietary salt	Keep salt intake below 2400 mg per day, or less than 1 tsp.

Abnormalities of Heart Conduction

The conduction system of the heart can fail, a condition known as **heart block**. Heart block can result from scar tissue interfering with the conduction tissue, and it may be necessary to implant an electric pacemaker if the block is complete.

Heart block is graded as first, second, or third degree. First-degree heart block is characterized by slightly delayed conduction to the ventricles and usually produces no symptoms. In second-degree heart block, not every impulse from the atria reaches the ventricles. Some forms of second-degree heart block progress to third-degree heart block in which impulses from the atria are completely blocked. The ventricle beats slowly and less efficiently. Eventually, heart failure ensues.

At times, the impulse for contraction spreads over the atria and the ventricle in an uncoordinated fashion. Atrial **fibrillation** and atrial flutter are very fast, uncoordinated impulses. These fast impulses produce rapid and incompetent contraction of the ventricles. Medications can be administered to slow the conduction through the AV node to the ventricles. This allows the ventricles to fill properly before contraction.

Ventricular fibrillation is far more serious than atrial fibrillation, and it is potentially fatal. A series of uncoordinated impulses spread over the ventricles, causing them to twitch or quiver rather than contract. The ventricle does not carry out effective coordinated contractions. Because no blood is pumped from the heart, ventricular fibrillation is a form of cardiac arrest. Immediate attempts at resuscitation must be made, or death will result. Permanent damage to other organs, particularly the brain, results when blood supply to them is compromised. A machine called an automated external defibrillator (AED) delivers electrical shocks and is used to re-establish normal heart rhythm. Defibrillators implanted under the skin of the shoulder resynchronize the heart on a daily basis, similarly to a pacemaker device. There are also combination devices with built-in pacemakers. In addition, there are multilead pacemakers with biphasic current that give a more complete signal to the heart chambers to initiate heart timing and contraction.

Irregular heartbeat rhythm is known as cardiac **arrhythmia** or **dysrhythmia**. Abnormal rhythms include skipped beats, extra beats, and premature beats; the latter are called premature ventricular contractions (PVCs). Additional irregularities include significant increases in heart rate, **tachycardia**, or abnormally slow hearts rate, **bradycardia**. Medications typically control the irregularities.

Shock

Shock is a life-threatening condition in which blood pressure drops too low to sustain life. Any condition that reduces the heart's ability to pump effectively or decreases venous return can cause shock. This low blood pressure causes an inadequate blood supply to the cells of the body.

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The cells can be quickly and irreversibly damaged and die. **Hypovolemic shock** (hemorrhagic) results from fluid volume loss after severe hemorrhage or loss of plasma in burn patients. Treatment includes administration of plasma or whole blood. **Neurogenic shock** is due to generalized vasodilation, resulting from decreased vasomotor tone. The reduced blood pressure causes poor venous return to the heart and, hence, poor cardiac output. The decreased vasomotor tone may be due to spinal anesthesia, spinal cord injury, or certain drugs. **Anaphylactic shock** accompanies a severe antigen-antibody reaction. **Cardiogenic shock** is the result of extensive myocardial infarction. It is often fatal, but drugs to combat it are sometimes effective. The types of shock are summarized in Figure 7–25.

Age-Related Diseases

Congenital diseases may be first detected in newborns, but may not be revealed until much later. Common congenital defects include holes in the heart septum, patent ductus arteriosus, and coarctation of the aorta. The incidence of rheumatic fever is highest among children. With aging, the lifetime effects of diet, family history, and behavior influence the risk for heart attacks and strokes.

With age, the heart, coronary arteries, and peripheral vessels lose strength and elasticity. The incidence of atherosclerosis, arteriosclerosis, and aneurysms increases with age, and about half of the elderly population have signs of heart disease.

The incidence of hypertension also increases with age. As a result, the average elderly person's heart exhibits approximately 30% thickening in the left ventricle walls.

Except for blood pressure, other cardiac functions show few age-related changes. An age-related decrease in maximum attainable heart rate during exercise or stress is a sign of decreased heart function. The cumulative effects of hypertension, atherosclerosis, and arteriosclerosis lead to an increased incidence of congestive heart disease in elderly persons.

Diagnostic Procedures

Many techniques for diagnosing and treating heart problems exist. **Auscultation**, listening through a stethoscope for abnormal sounds, and the electrocardiogram (ECG) provide valuable information regarding heart condition. The **electrocardiogram** is an electrical recording of heart action and aids in the diagnosis of coronary artery disease, myocardial infarction, valve disorders, and some congenital heart diseases. It is also useful in diagnosing arrhythmias and heart block. **Echocardiography** (ultrasound cardiography) is also a noninvasive procedure that utilizes high-frequency sound waves to examine the size, shape, and motion of heart structures. It gives a time-motion study of the heart, which permits direct recordings of heart valve movement, measurements of the heart chambers, and changes that occur in the heart chambers during the cardiac cycle. Color **Doppler echocardiography** explores bloodflow patterns and changes in velocity of bloodflow within the heart and great vessels. It enables

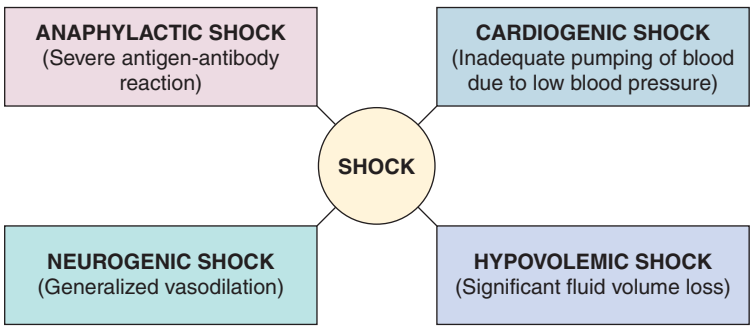

Figure 7–25
Various types of shock.

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the cardiologist to evaluate valve stenosis or insufficiency.

An exercise tolerance test is used to diagnose coronary artery disease and other heart disorders. This test monitors the ECG and blood pressure during exercise. Problems that normally do not occur at rest are revealed.

Cardiac catheterization is a procedure in which a catheter is passed into the heart through blood vessels to sample the blood in each chamber for oxygen content and pressure. The findings can indicate valve disorders or abnormal shunting of blood, and aids in determining cardiac output.

X-rays of the heart and great vessels, the aorta, and the pulmonary artery, in conjunction with **angiocardiography,** in which a contrast indicator (dye) is injected into the cardiovascular system, can detect blockage in vessels. **Coronary arteriography** employs a selective injection of contrast material into coronary arteries for a film recording of blood vessel action.

CHAPTER SUMMARY

After reviewing the normal structure and function of the heart, heart diseases such as coronary artery disease, myocardial infarction, and angina pectoris were discussed. Myocardial infarction and angina pectoris cause severe chest pain or referred pain in the arm or neck. Dyspnea is a common symptom of many heart diseases; the lack of oxygen in the tissues stimulates the respiratory center, causing the person to experience difficulty in breathing or shortness of breath. Fainting or loss of consciousness occurs when the brain is deprived of an adequate blood supply. All of the tissues and organs are affected by poor circulation. Cyanosis occurs when blood is not properly oxygenated and fluid accumulates in the tissues, causing edema when veins become congested.

Hypertensive heart disease develops from long-standing hypertension, and cor pulmonale results from chronic lung disease. Congestive heart failure is characterized inadequate pumping of blood to meet the needs of the body.

Congenital heart diseases, tetralogy of Fallot, patent ductus arteriosis, and coarctation of the aorta were explained. It was noted that a congenital defect is frequently the site of a bacterial infection.

Valve disorders such as stenosis and insufficiency cause heart murmurs. Rheumatic heart disease is a common cause of valve disease. Surgical techniques may be required to correct diseased valves.

Antibiotics have reduced the danger of endocarditis and the frequency of rheumatic heart disease.

Abnormalities of heart action, heart block, fibrillation, and arrhythmia were described. Diagnostic procedures include auscultation, electrocardiography, ultrasound cardiography (echocardiography), cardiac catheterization, and exercise tolerance testing. The condition of coronary arteries and the great vessels can be evaluated through angiocardiography and coronary arteriography. Surgical procedures are available to correct congenital heart defects, replace valves, and implant electric pacemakers or defibrillators. Coronary bypass surgery and angioplasty with stent implants reduce heart damage by increasing coronary circulation. Diet and exercise in moderation continue as major benefits to a healthy cardiovascular system.

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DISEASES AT A GLANCE

Cardiovascular System

DISEASE	ETIOLOGY	SIGNS AND SYMPTOMS
Endocarditis	Streptococcus infection, drugs	Fever, anorexia, weight loss, back pain, night sweats, petechiae, heart murmur
Rheumatic disease	Streptococcus pyogenes, autoimmune to heart	Follows infection by Group A hemolytic streptococci; latent period followed by fever, inflamed and painful joints, rash, heart murmur
Coronary artery disease	Plaque, arteriosclerosis	Angina pectoris following exercise, stress, heavy meal
Myocardial infarct (MI)	Plaque, arteriosclerosis	Chest pain radiating to jaw, neck, left arm, back; feeling of tightness; suffocation; nausea and vomiting may occur; may be "silent" (no symptoms)
Hypertensive heart disease	Essential hypertension, kidney disease	Long-standing High blood pressure, enlargement of heart, later pulmonary edema with dyspnea, dilation and weakness of ventricles
Congestive heart failure	Rheumatic disease, hypertension, myocardial infarction	Fatigue, difficulty breathing; left-side failure leads to pulmonary edema, right-side failure leads to systemic edema; both may occur
Cor pulmonale	Chronic hypertensive lung	Chronic lung disease, right-side heart failure with systemic edema, fatigue, rapid heart rate
Mitral stenosis	Infection, congenital	Heart murmur, dyspnea, fatigue, lower oxygen in arteries; pulmonary edema may develop
Mitral insufficiency (mitral prolapse)	Idiopathic, infection, weak papillary muscles	Heart murmur, may be asymptomatic; palpitations, fatigue, dyspnea may develop
Aortic stenosis	Congenital, infection	Lower output of blood with syncope, heart murmur
Aortic insufficiency	Congenital, idiopathic	Heart murmur, backflow into ventricle, enlarged ventricle

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DIAGNOSIS	TREATMENT
Echocardiography	Antibiotics
Patient history of strep infection; physical exam, strep antibody test	Anti-inflammatory drugs, steroids, diuretics, bed rest
Angiocardiography, coronary arteriography	Drug therapy, nitroglycerin, angioplasty, bypass surgery
ECG, blood test (serum enzymes), physical exam	Drug therapy, oxygen, reduced activities, rest
enlarged heart, x-ray, physical exam	Drug therapy, oxygen
Physical exam, x-ray, scans show enlarged heart	Drug therapy, diuretics, oxygen
Patient history, physical exam	Bronchodilators, ventilator
Echocardiography, auscultation	Valve replacement surgery
Echocardiography, auscultation	Valve replacement surgery only if severe
Echocardiography, auscultation	Valve replacement surgery
Echocardiography, auscultation	Valve replacement surgery

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DISEASES AT A GLANCE (*continued*)

Cardiovascular System

DISEASE	ETIOLOGY	SIGNS AND SYMPTOMS
Heart block	Atherosclerois, scar tissue in conducting pathway	Reduced pulse, palpitations or irregular beat
Atrial fibrillation	Drugs, electrolytes imbalance, hypertrophy	Palpitations, as ventricle contraction is affected
Ventricular fibrillation	Electrical shock, myocardial infarction	Collapse, since bloodflow stops; cardiac arrest
Cardiac arrthymia	Myocardial infarction, drugs, electrolyte imbalance	Palpitations, as beat is uncoordinated
Tachycardia	Congenital, idiopathic	Rapid heart rate, not due to exercise
Bradycardia	Congenital, idiopathic	Slow heart rate, not increasing with exercise
Atrial septal defect (ASD)	Congenital	Small ASDs without symptoms; large ASDs may lead to pulmonary hypertension
Ventricular septic defect (VSD)	Congenital	Loud murmur during contraction of ventricles; right-side heart failure may occur with large defect; cyanosis if blood shunts right to left
Tetralogy of Fallot	Congenital	Cyanotic "blue baby," murmurs, excess number of red cells, dyspnea, fingers and nails clubbed
Patent ductus arteriosus (PDA)	Congenital	Murmur develops
Coarctation of aorta	Congenital	Blood Pressure low in legs, high in arms; congestive heart failure can result
Hypertension	Idiopathic, atherosclerosis	None obvious: "silent killer"

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DIAGNOSIS	TREATMENT
ECG	Artificial pacemaker
ECG	Medication such as lanoxin
ECG	Cardiopulmonary resuscitation, defibrillation
ECG	Medication
ECG	Medication
ECG	Pacemaker may be needed
Chest x-ray, ECG	Large defects surgically repaired
Chest x-ray, ECG, cardiac catheterization, angiography, or echocardiography	Large defects surgically repaired; small ones may spontaneously close
Chest x-ray, ECG, cardiac catheterization, angiography	Surgical repair
Chest x-ray, cardiac catheterization, angiography	Medication, ligation (tying shut) of defect
Chest x-ray, cardiac catheterization	Surgery
Blood pressure measures consistently high	Weight loss, reduced salt and fat in diet, exercise, smoking cessation, medication

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DISEASES AT A GLANCE (*continued*)

Cardiovascular System

DISEASE	ETIOLOGY	SIGNS AND SYMPTOMS
Hypovolemic	Blood loss due to hemorrhage	Severe hemorrhage, severe and extensive burns
Neurogenic shock	CVA, loss of vascular tension	Falling blood pressure, slow heart rate
Anaphylactic shock	Allergic reaction	Hives, itching, wheezing, increased heart rate, increased respiratory rate
Cardiogenic shock	Myocardial infarction, electrical shock	Myocardial infarction; fast heart rate; cool, clammy skin
Raynaud's disease	Idiopathic, smoking	Numbness, discoloration, pain, cold digits
Arteriosclerosis, Atherosclerosis	Genetic, diet, idiopathic	Varies with arteries affected and severity; thrombus (clot) may block artery; embolus (traveling clot) may be released and block artery elsewhere
Aneurysm	Congenital, hypertension	No symptoms until rupture
Thrombophlebitis	Stagnant blood, CHF	Pain, edema, inflammation, thrombus (clot) formation
Varicose veins	Excess weight, continuous standing	Swelling of veins, stasis, rupture may occur; spider veins, hemorrhoids, esophageal varices

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DIAGNOSIS	TREATMENT
Physical exam	Fluid replacement, transfusion
Physical exam, cardiovascular monitoring	Elevation of legs, compressive stockings, drugs, fluids
Physical exam	Epinephrine, steroids, fluids
Physical exam, cardiovascular monitoring	Treatment of heart failure, drugs
Physical exam	Protection from cold, smoking cessation
Ultrasound arteriography, Doppler imaging	Low cholesterol diet, exercise, weight loss, smoking cessation; balloon angiography, bypass surgery
X-ray, MRI, ultrasound	Surgical repair
Physical exam	Anticoagulants, antibiotics, surgery
Physical exam	Compression sclerotherapy, support hose, surgery

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Interactive Activities

Cases for Critical Thinking

1. A 59-year-old male calls paramedics after experiencing an episode of chest pain while shoveling snow. He describes his pain as a crushing, tight feeling that radiates to his left arm and jaw. What type(s) of heart disease is this patient experiencing?
2. A 60-year-old male has been experiencing an increase in shortness of breath, a productive cough, and tiredness over the past few months. On examination, the doctor hears congestion in the lungs. What type of heart failure is this patient currently experiencing?
3. A 12-year-old child experiences high fever and chills. He also says that his heart feels like it's pounding. Two weeks before these symptoms, the child fell off his bike and skinned his knee. This child also has a history of a heart murmur. What disease should we consider?

Multiple Choice

1. Congestive heart failure and shortness of breath due to pulmonary edema result from failure of which heart structure?
   a. right ventricle
   b. left ventricle
   c. septum
   d. right auricle
2. Which four defects in the list below are associated with the tetralogy of Fallot?
   (1) mitral insufficiency
   (2) hypertrophy of right ventricle
   (3) atrial septal defect
   (4) pulmonary stenosis
   (5) aortic stenosis
   (6) ventricular septal defect
   (7) misplaced aorta
   (8) hypertrophy of left ventricle
   a. (1), (2), (4), (8)
   b. (3), (5), (7), (8)
   c. (2), (3), (5), (7)
   d. (2), (4), (6), (7)
3. What causes cyanosis?
   a. failure of the foramen ovale to close
   b. failure of the ductus arteriosus to close
   c. pulmonary stenosis
   d. large septal hole
4. Which of the following requires immediate attempts at resuscitation to prevent cardiac arrest?
   a. atrial fibrillation
   b. ventricular fibrillation
   c. bradycardia
   d. tachycardia
5. Hypertension in a patient's arms but no femoral pulse indicates_____.
   a. tetralogy of Fallot
   b. coarctatin of the aorta
   c. patent ductus arteriosis
   d. failure of the foramen ovale to close
6. The depositing of fatty plaques within the arteries is called _____.
   a. arteriosclerosis
   b. atherosclerosis
   c. petechiae
   d. stasis
7. Phlebitis is an inflammation of the _____.
   a. veins
   b. arteries
   c. aorta
   d. pulmonary valve
8. A bubble-like protrusion of an arterial wall is called _____.
   a. hematoma
   b. petechia
   c. aneurysm
   d. angioplasty
9. An embolus traveling from the leg to the heart would be more likely to block the _____.
   a. aorta
   b. pulmonary artery
   c. mitral valve
   d. kidney artery
10. What is the action of renin on arterioles?
    a. constriction
    b. dilation
    c. no change
    d. blockage

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True or False

_____ 1. The left atrium hypertrophies and dilates when the mitral valve becomes stenotic.
_____ 2. Pain of a myocardial infarction is relieved by nitroglycerin.
_____ 3. Tachycardia refers to a decreased heart rate.
_____ 4. Aortic insufficiency causes backflow of blood from aorta to the left ventricle.
_____ 5. Long-standing hypertension causes the left ventricle to hypertrophy.
_____ 6. Embolisms to the brain can result from a thrombus on the mitral valve.
_____ 7. Neurogenic shock results from severe fluid-volume loss.
_____ 8. After a severe hemorrhage, blood pressure increases.
_____ 9. Women have less chance of myocardial infarction after age 60.
_____ 10. Angioplasty is less invasive than a bypass surgery.

Fill-Ins

1. _____ uses high-frequency sound waves to examine the heart.
2. Enlargement of the walls of the heart is _____.
3. Oxygen-poor blood flows from the body through the _____ artery and to the _____.
4. The left ventricle pumps blood to the body via the _____.
5. Raynaud's disease results in _____ of the arterioles.
6. The highest pressure in a blood pressure reading reflects _____.
7. Anticoagulant medication is administered to prevent a thrombus from becoming an _____.
8. In primary or essential hypertension, the causes are _____.
9. A common drug for anticoagulant use is _____.
10. An implanted device or external instrument that corrects heart rhythm is the _____.

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