ADDITIONAL RESOURCES

Kumar V, Abbas AK, Fausto M: *Robbins and Cotran Pathologic Basis of Disease*, ed 8, Philadelphia, 2007, Saunders.
Patton KT, Thibodeau GA: *Anatomy and Physiology*, ed 7, St. Louis, 2010, Mosby.

Web Sites

http://www.mayoclinic.com *Mayo Clinic*
http://www.lls.org/ *The Leukemia & Lymphoma Society*
http://www.emedicinehealth.com *e medicine health*
www.cancer.org *American Cancer Society*

CHAPTER 12

Cardiovascular System Disorders

CHAPTER OUTLINE

LEARNING OBJECTIVES

After studying this chapter, the student is expected to:

1. Describe the common diagnostic tests for cardiovascular function.
2. Describe the dietary and lifestyle changes, and the common drug groups used, in the treatment of cardiovascular disease.
3. Explain the role of cholesterol and lipoproteins in the development of atheromas.
4. Explain the significance of metabolic syndrome in the development of cardiovascular disease.
5. State the factors predisposing to atherosclerosis.
6. Compare angina and myocardial infarction.
7. Describe the common arrhythmias and cardiac arrest.
8. Discuss the causes of congestive heart failure and the effects of left-sided and right-sided failure.
9. Explain the changes in blood flow and their effects in common congenital heart defects.
10. Discuss the development of rheumatic fever and rheumatic heart disease.
11. Describe the etiology and pathophysiology of infectious endocarditis and pericarditis.
12. Explain the development and possible effects of essential hypertension.
13. Compare the arterial peripheral vascular diseases atherosclerosis and aneurysms.
14. Describe the development and effects of the venous disorders varicose veins, phlebothrombosis, and thrombophlebitis.
15. Discuss the types of shock and the initial and progressive effects of shock on the body.

KEY TERMS

adrenergic
anastomoses
angioplasty
auscultation
autoregulation
baroreceptors
bradycardia
cardiomegaly
depolarization
ectopic
electrodes
endarterectomy
hemoptysis
microcirculation
murmurs
orthopnea
sulcus
syncope
synergistic
tachycardia

Review of the Cardiovascular System

Heart

Anatomy

The heart functions as the pump for the circulating blood in both the pulmonary and systemic circulations. The path of a specific component of the blood, such as a red blood cell, through the heart and circulation is illustrated in Figure 12-1.

The heart is located in the *mediastinum* between the lungs and is enclosed in the double-walled *pericardial sac* (see Fig. 12-29). The outer fibrous pericardium anchors the heart to the diaphragm. The visceral pericardium, also called the epicardium, consists of a serous membrane that provides a small amount of lubricating fluid within the pericardial cavity between the two pericardial membranes to facilitate heart movements. The middle layer of the heart is the myocardium, composed of specialized cardiac muscle cells that contract rhythmically and forcefully to pump blood throughout the organs. The left ventricular wall is thicker because it must eject blood into the extensive systemic circulation. The inner layer of the heart is the *endocardium*, which also forms the four heart valves that separate the chambers of the heart and ensure *one-way flow* of blood.

The atrioventricular (AV) valves separate the atria from the ventricles; they comprise, on the right side, the tricuspid valve with three leaflets or cusps, and on the left side, the mitral or bicuspid valve with two leaflets. The semilunar valves, each with three cusps, include the aortic and pulmonary valves located at the exits to the large arteries from the ventricles. The *septum* separates the left and right sides of the heart.

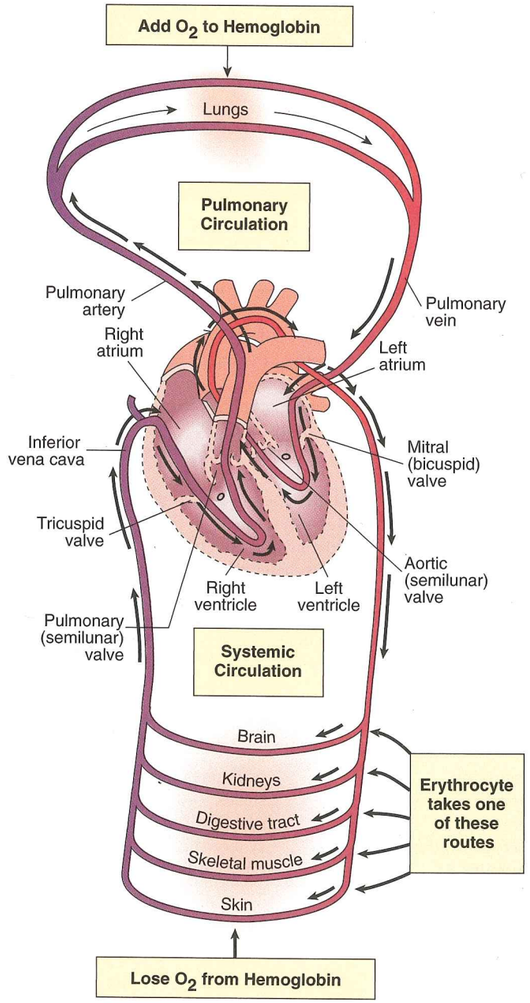

FIGURE 12-1 Path of erythrocyte in the circulation.

Conduction System

Impulses to initiate cardiac contractions are conducted along specialized myocardial (cardiac muscle) fibers. No nerves are present within the cardiac muscle. The unique characteristics of cardiac muscle include the presence of intercalated disks at the junctions between fibers. These disks contain desmosomes, connections to prevent muscle cells from separating during contraction; and gap junctions, which permit ions to pass from cell to cell, facilitating rapid transmission of impulses. These specialized structures ensure that all muscle fibers of the two atria normally contract together, followed shortly by the two ventricles. This coordinated effort results in a rhythmic and efficient filling and emptying of the atria and ventricles that has sufficient force to sustain the flow of blood through the body.

The pathway for impulses in the cardiac conduction system is as follows:

- All cardiac muscle cells can initiate impulses, but normally the conduction pathway originates at the sinoatrial (SA) node, often called the *pacemaker*, located in the wall of the right atrium.
- The SA node automatically generates impulses at the basic rate, called the *sinus rhythm* (approximately 70 beats per minute), but this can be altered by autonomic nervous system fibers that innervate the SA node and by circulating hormones such as epinephrine.
- From the SA node, impulses then spread through the atrial conduction pathways, resulting in contraction of both atria.
- The impulses then arrive at the AV node, located in the floor of the right atrium near the septum. This is the only anatomical connection between the atrial and ventricular portions of the conduction system.
- There is a slight delay in conduction at the AV node to allow for complete ventricular filling; then the impulses continue into the ventricle through the AV bundle (Bundle of His), the right and left bundle branches, and the terminal Purkinje network of fibers, stimulating the simultaneous contraction of the two ventricles.

Conduction of impulses produces a change in electrical activity that can be picked up by **electrodes** attached to the skin at various points on the body surface, producing the *electrocardiogram (ECG)* (see Fig. 12-2). The atrial contraction is represented by the **depolarization** in the P wave, and the ventricular contraction is shown by the large wave of depolarization in the ventricles (QRS). This wave masks the effect of atrial repolarization, but the third wave (T wave) represents the repolarization of the ventricles, or recovery phase. Abnormal variations in the ECG known as *arrhythmias* or *dysrhythmias* may indicate acute problems, such as an infarction, or systemic problems, such as electrolyte imbalances (for example, potassium deficiency [see Fig. 2-8]).

Control of the Heart

Heart rate and force of contraction are controlled by the *cardiac control center* in the medulla of the brain. The **baroreceptors** in the walls of the aorta and internal carotid arteries detect changes in blood pressure and the cardiac center then responds through stimulation of the sympathetic nervous system (SNS) or the parasympathetic nervous system to alter the rate and force of cardiac contractions as required. Sympathetic innervation increases heart rate (**tachycardia**) and contractility, whereas parasympathetic stimulation by the vagus nerve slows the heart rate (**bradycardia**). The sympathetic or $beta_1$-**adrenergic** receptors in the heart (see Chapter 14) are an important site of action for some drugs, such as beta-blockers. Because beta-blockers fit the receptors and prevent normal SNS stimulation, they are used to block any increases in rate and force of contractions after the heart has been damaged.

Factors that increase heart rate include:

- Elevated body temperature, such as in fever
- Increased environmental temperatures, especially if humidity is high
- Exertion or exercise, notably when beginning, followed by a leveling off
- Smoking even one cigarette
- Stress response
- Pregnancy
- Pain

Any stimulation of the SNS, as with stress, increases the secretion of epinephrine, which in turn stimulates beta-receptors and increases both the heart rate and contractility.

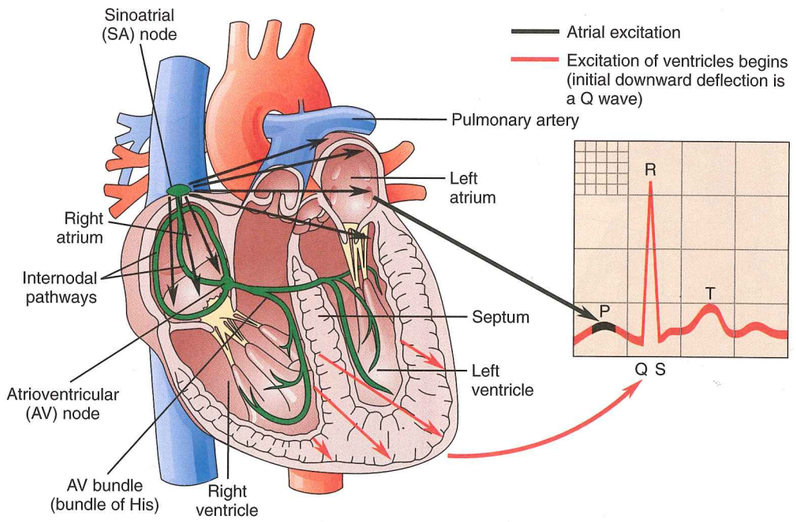

FIGURE 12-2 Schematic drawing of the conducting system of the heart. An impulse normally is generated in the sinus node and travels through the atria to the AV node, down the bundle of His and Purkinje fibers, and to the ventricular myocardium. Recording of the depolarizing and repolarizing currents in the heart with electrodes on the surface of the body produces characteristic waveforms. *(From Copstead-Kirkhorn LE, Banasik JL:* Pathophysiology, *ed 4, Philadelphia, 2009, Saunders.)*

THINK ABOUT 12-1

a. Where is the mitral valve located? Describe the direction and type of blood (oxygenated or nonoxygenated) that flows through this valve.
b. List two functions of the AV node.
c. Describe the control of heart rate during and after exercise.

Coronary Circulation

Cardiac muscle requires a constant supply of oxygen and nutrients to conduct impulses and contract efficiently, but it has very little storage capacity for oxygen.

The distribution of the major blood vessels in the coronary circulation is:

1. Two major arteries, the right and left coronary arteries, branch off the aorta immediately above the aortic valve (Fig. 12-3).
2. The left coronary artery soon divides into the *left anterior descending* or *interventricular artery,* which follows the anterior interventricular **sulcus** or groove downward over the surface of the heart, and the *left circumflex artery,* which circles the exterior of the heart in the left atrioventricular sulcus.
3. Similarly, the *right coronary artery* follows the right atrioventricular sulcus on the posterior surface of the heart and branches into the right marginal artery and the posterior interventricular artery, and then descends in the posterior interventricular groove toward the apex of the heart, where it comes close to the terminal point of the left anterior descending artery.

   The passage of arteries over the surface of the heart in these grooves is helpful because it permits surgical replacement of obstructed arteries with "bypasses"—using sections of other veins or arteries (see Fig. 12-13 for a diagram of a bypass).
4. Many small branches extend inward from these large arteries to supply the myocardium and endocardium. Blood flow through the myocardium is greatest during diastole or relaxation and is reduced during systole or contraction as the contracting muscle compresses the arteries. Thus, very rapid or prolonged contractions can reduce the blood supply to the cardiac muscle cells.

Anastomoses, or direct connections, exist between small branches of the left and right coronary arteries near the apex, as well as in other areas in which branches are nearby (see Fig. 12-3). These junctions have the potential to open up and provide another source of blood to an area. *Collateral* circulation (alternative source of blood and nutrients) is important if an artery becomes obstructed. When obstruction develops gradually, more capillaries from nearby arteries tend to enlarge or extend into adjacent tissues to meet the metabolic needs of the cells. Regular aerobic exercise contributes to cardiovascular fitness by stimulating the development of collateral channels.

Any interference with blood flow will affect heart function, depending on the specific area supplied by that artery. Generally, the right coronary artery supplies the right side of the heart and the inferior portion of the left ventricle, as well as the posterior interventricular septum. The left anterior descending artery brings blood to the anterior wall of the ventricles, the anterior septum, and the bundle branches, and the circumflex artery nourishes the left atrium and the lateral and posterior walls of the left ventricle. The source of blood for the SA node depends on the specific position of the arteries, which varies in individuals. The SA node is supplied by the right coronary artery in slightly more than half the population and by the left circumflex artery in the remainder. The AV node is nourished primarily by the right coronary artery. This information implies that blockage of the right coronary artery is more likely to result in conduction disturbances of the AV node (resulting in dysrhythmias), whereas interference with the blood supply to the left coronary artery will most likely impair the pumping capability of the left ventricle (potentially leading to congestive heart failure).

The course of the coronary or cardiac veins generally parallels that of the arteries, with the majority of the blood returning to the coronary sinus and emptying directly into the right atrium.

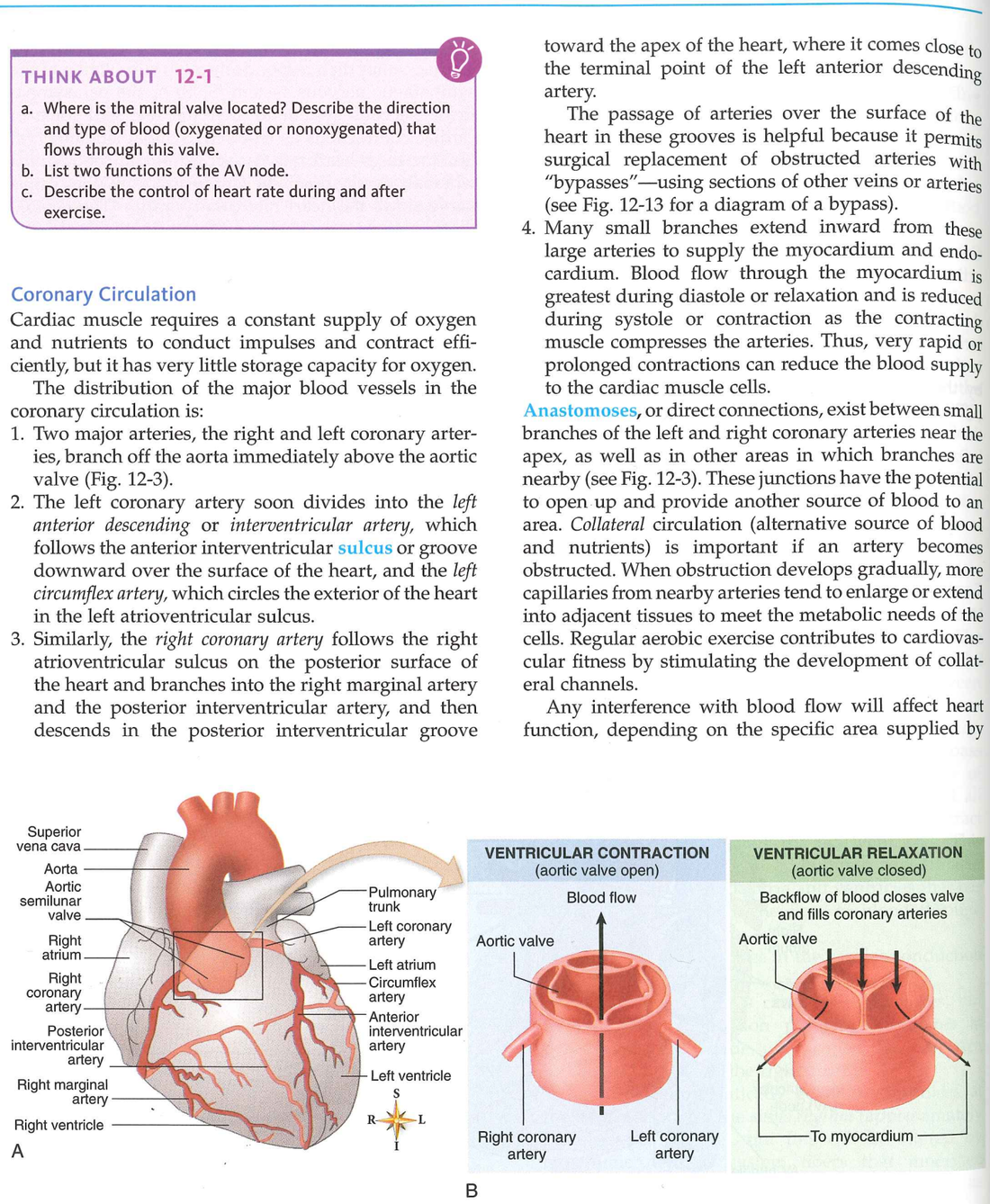

FIGURE 12-3 **Coronary arteries. A,** Diagram showing the major coronary arteries (anterior view). Clinicians often refer to the interventricular arteries as *descending* arteries. Thus a cardiologist would refer to the *anterior descending artery* and an anatomist would refer to the same vessel as the *anterior interventricular artery.* **B,** The unusual placement of the coronary artery opening behind the leaflets of the aortic valve allows the coronary arteries to fill during ventricular relaxation. *(From Patton KT, Thibodeau GA:* Anatomy & Physiology, *ed 8, St. Louis, 2013, Mosby.)*

APPLY YOUR KNOWLEDGE 12-1

Predict three basic ways that cardiac function could be impaired.

Cardiac Cycle

The cardiac cycle refers to the alternating sequence of *diastole,* the relaxation phase of cardiac activity, and *systole,* or cardiac contraction, which is coordinated by the conduction system for maximum efficiency (see Fig. 12-4).

1. The cycle begins with the two atria relaxed and filling with blood (from the inferior and superior venae cavae into the right atrium, and from the pulmonary veins into the left atrium).
2. The AV valves open as the pressure of blood in the atria increases and the ventricles are relaxed.

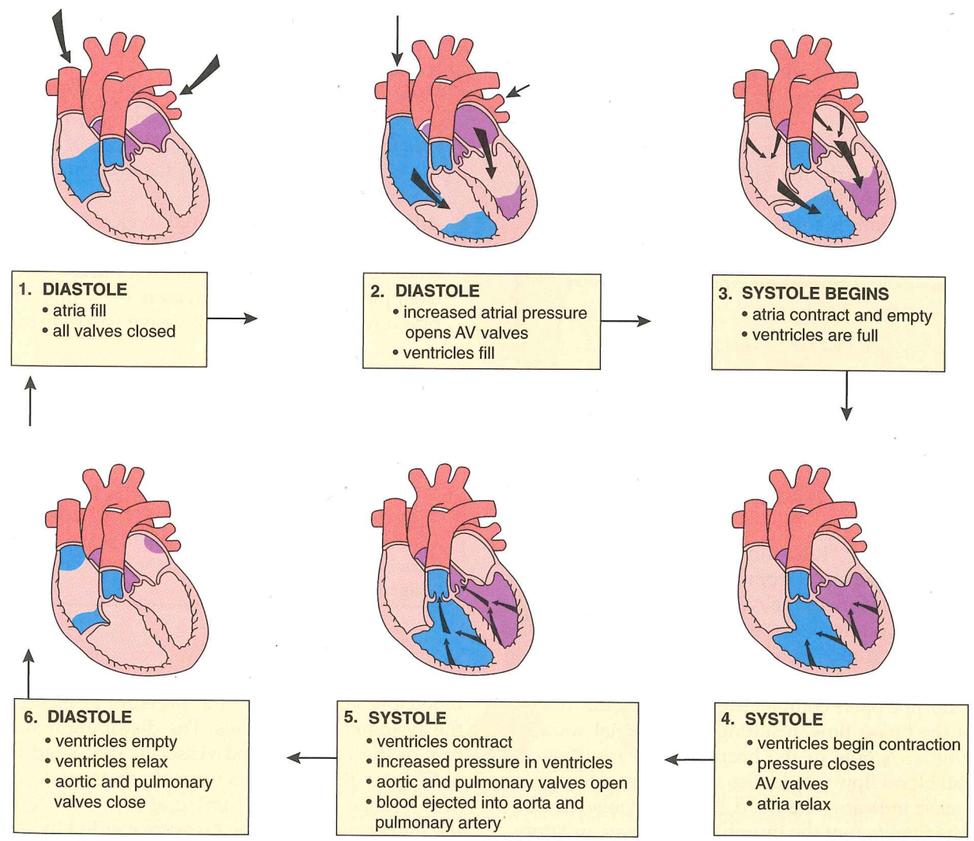

FIGURE 12-4 Cardiac cycle.

3. Blood flows into the ventricles, almost emptying the atria.
4. The conduction system stimulates the atrial muscle to contract, forcing any remaining blood into the ventricles.
5. The atria relax.
6. The two ventricles begin to contract, and pressure increases in the ventricles.
7. The AV valves close.
8. For a brief moment, all valves are closed, the ventricular myocardium continues to contract, building up pressure in this isovolumetric phase (no change in blood volume in the ventricles).
9. Then the increasing pressure opens the semilunar valves; blood is forced into the pulmonary artery and aorta. Note that the muscle contraction must be strong enough to overcome the opposing pressure in the artery to force the valve open. This is significant, particularly in the left ventricle, in which the pressure must be greater than the diastolic pressure in the aorta. Because the pulmonary circulation is a low-pressure system, the right ventricle does not have to exert as much pressure to pump blood into the pulmonary circulation.
10. At the end of the cycle, the atria have begun to fill again, the ventricles relax, the aortic and pulmonary valves close to prevent backflow of blood, and the cycle repeats.

The same volume of blood is pumped from the right and left sides of the heart during each cycle. This is important to ensure that blood flow through the systemic and pulmonary circulations is consistently balanced.

THINK ABOUT 12-2

a. Discuss the importance of collateral circulation and explain how collateral circulation can be maximized.
b. Why is there a pause after the atrial contraction and before the ventricular contraction?
c. Predict the outcome if more blood is pumped into the pulmonary circulation than into the systemic circulation during each cardiac cycle.

The *heart sounds*, "lubb-dupp," which can be heard with a stethoscope, result from vibrations due to closure of the valves. Closure of the AV valves at the beginning of ventricular systole causes a long, low "lubb" sound, followed by a "dupp" sound as the semilunar valves close with ventricular diastole. Defective valves that leak or do not open completely cause unusual turbulence in the blood flow, resulting in abnormal sounds, or **murmurs**. A hole in the heart septum resulting in abnormal blood flow would also cause a heart murmur.

The *pulse* indicates the heart rate. The pulse can be felt by the fingers (not the thumb) placed over an artery that passes over bone or firm tissue, most commonly at the wrist (Fig. 12-5). During ventricular systole, the surge of blood expands the arteries. The characteristics of the pulse, such as weakness or irregularity in a peripheral pulse (e.g., the radial pulse in the wrist), often indicate a problem. The *apical* pulse refers to the rate measured at the heart itself. A *pulse deficit* is a difference in rate between the apical pulse and the radial pulse.

Cardiac function can be measured in a number of ways.

- *Cardiac output* is the volume of blood ejected by a ventricle in one minute and depends on heart rate and *stroke volume*, the volume pumped from one ventricle in one contraction) (Fig. 12-6). This means that at rest, the heart pumps into the system an amount equal to the total blood volume in the body every minute, which is a remarkable feat. When necessary, the normal heart can increase its usual output by four or five times the minimum volume.
- *Stroke volume* varies with sympathetic stimulation and venous return. When an increased amount of blood returns to the heart, as during exercise, the heart is stretched more and the force of the contraction normally increases proportionately. During exercise, stress, or infection, cardiac output increases considerably.
- *Cardiac reserve* refers to the ability of the heart to increase output in response to increased demand.
- *Preload* refers to the mechanical state of the heart at the end of diastole with the ventricles at their maximum volume.
- *Afterload* is the force required to eject blood from the ventricles and is determined by the *peripheral resistance* to the opening of the semilunar valves. For example, afterload is increased by a high diastolic pressure resulting from excessive vasoconstriction.

THINK ABOUT 12-3

a. What information does the ECG provide about heart function?
b. Describe the function of the areas of the heart usually supplied by the left coronary artery.
c. Describe the effect if the atria were to contract at the same time as the ventricles, or if the ventricles contracted slightly before the atria.

Blood Pressure

Blood pressure refers to the pressure of blood against the systemic arterial walls. The distribution and structure of the various blood vessels is discussed in detail in Chapter 10. In adults a normal pressure is commonly in the range of 120/70 mmHg at rest. *Systolic pressure*, the higher number, is the pressure exerted by the blood when ejected from the left ventricle. *Diastolic pressure*,

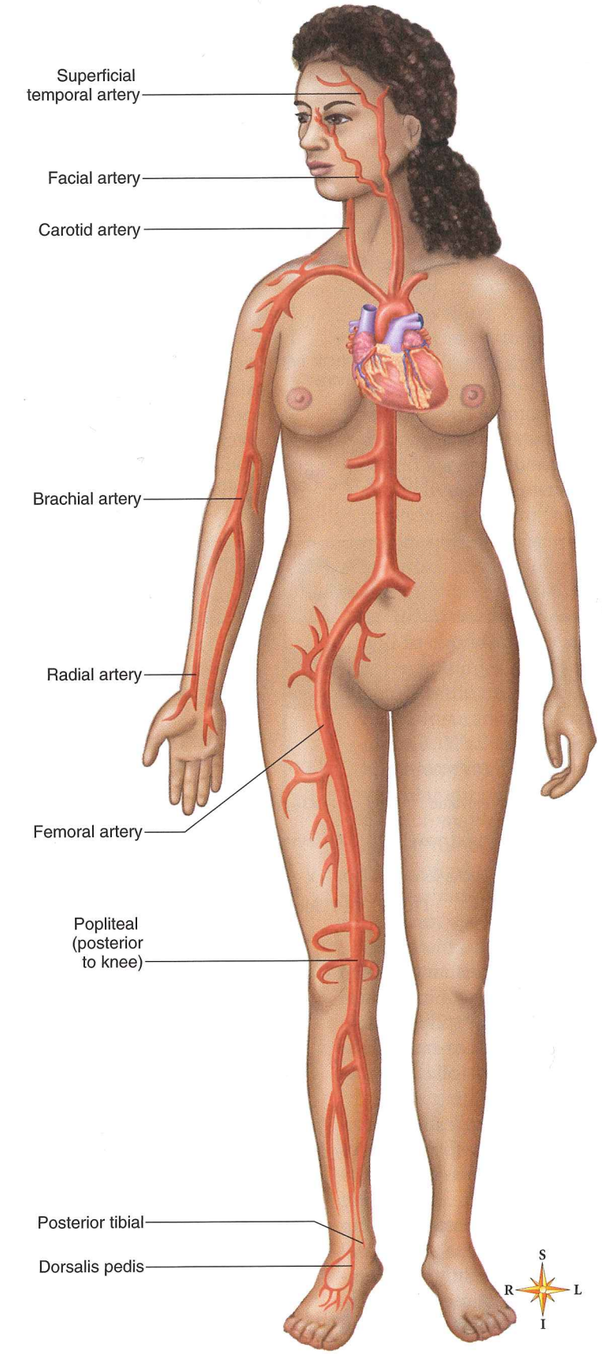

FIGURE 12-5 Pulse points. Each pulse point is named after the artery with which it is associated. (Some arteries in the figure have been enlarged to clarify the location of pulse points.) *(From Patton KT, Thibodeau GA:* Anatomy & Physiology, *ed 8, St. Louis, 2013, Mosby.)*

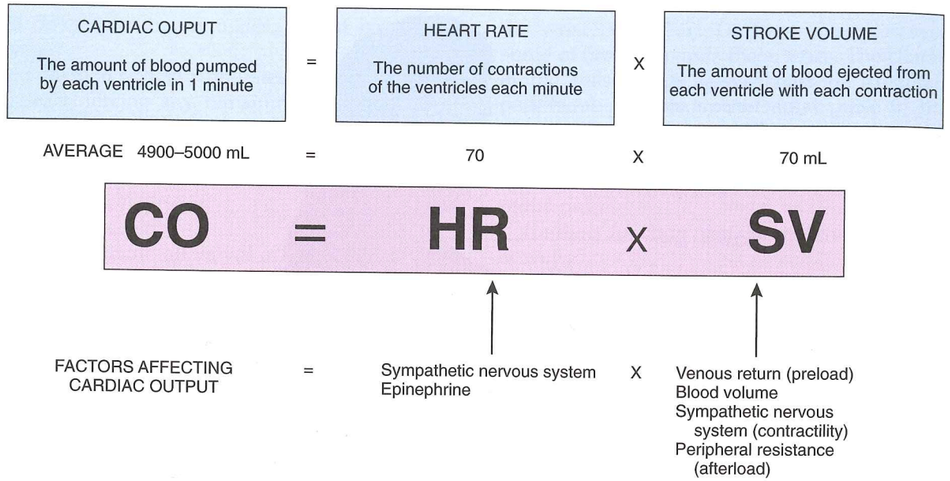

FIGURE 12-6 Cardiac output.

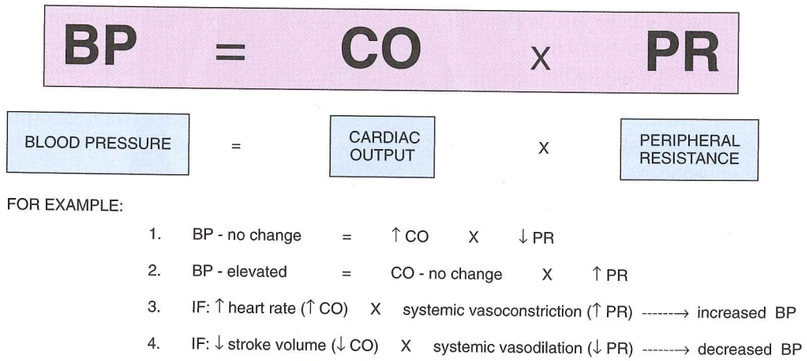

FIGURE 12-7 Blood pressure.

the lower value, is the pressure that is sustained when the ventricles are relaxed. The brachial artery in the arm is used to measure blood pressure with a sphygmomanometer and an inflatable blood pressure cuff. *Pulse pressure* is the difference between the systolic and diastolic pressures.

Blood pressure depends on cardiac output and peripheral resistance (Fig. 12-7). Specific variables include blood volume and viscosity, venous return, the rate and force of heart contractions, and the elasticity of the arteries. *Peripheral resistance* is the force opposing blood flow, or the amount of friction with the vessel walls encountered by the blood. Decreasing the diameter (or lumen) of the blood vessel increases the resistance to blood flow. Normally peripheral resistance can be altered by the systemic constriction or dilation of the arterioles. Systemic or widespread vasoconstriction occurs in response to sympathetic stimulation and increases blood pressure. Systemic or general vasodilation that leads to decreased blood pressure results from reduced SNS stimulation. (There is *no parasympathetic* nervous system innervation in the blood vessels.) Any obstruction in the blood vessel also increases resistance. Local vasoconstriction or dilation does *not* affect the overall systemic blood pressure.

Changes in blood pressure are detected by the baroreceptors and relayed to the vasomotor control center in the medulla, which adjusts the distribution of blood to maintain normal blood pressure. For example, when one rises from a supine position, blood pressure drops momentarily owing to gravitational forces until the reflex vasoconstriction mechanism in the body ensures that more blood flows to the brain.

Blood pressure is elevated by increased SNS stimulation in two ways:

1. SNS and epinephrine act at the beta$_1$-adrenergic receptors in the heart to increase both the rate and force of contraction.
2. SNS, epinephrine, and norepinephrine increase vasoconstriction by stimulating the alpha$_1$-receptors in the arterioles of the skin and viscera. This reduces the capacity of the system and increases venous return.

Other hormones also contribute to the control of blood pressure:

- Antidiuretic hormone (ADH) increases water reabsorption through the kidney, thus increasing blood volume. Antidiuretic hormone, also known as vasopressin, also causes vasoconstriction.
- Aldosterone increases blood volume by increasing reabsorption of sodium ions and water.
- The renin-angiotensin-aldosterone system in the kidneys is an important control and compensation mechanism that is initiated when there is any decrease in renal blood flow. This stimulates the release of renin, which in turn activates angiotensin (vasoconstrictor) and stimulates aldosterone secretion (see Chapter 18). Angiotensin II is a powerful vasoconstrictor.

THINK ABOUT 12-4

a. Explain four factors that can increase blood pressure.
b. List the compensatory mechanisms (in the correct sequence) that can help return the blood pressure to normal levels following a slight drop such as standing up too rapidly.
c. List three ways that systemic circulation could be impaired.
d. Describe the effect of a hot compress on the tissues to which it is applied.
e. How does vasoconstriction in the skin and viscera result in increased venous return to the heart?

Heart Disorders

Heart disease is ranked as a major cause of morbidity and mortality in North America. Common heart diseases include congenital heart defects, hypertensive heart disease, angina and heart attacks, cardiac arrhythmias, and congestive heart failure. There is increasing emphasis on routine preventive measures for all individuals, with a focus on factors such as a healthy diet, regular exercise, moderation in alcohol intake, cessation of smoking, safe sexual practices, immunizations, monitoring body weight and blood pressure, and basic screening tests for cholesterol levels and the presence of cancer.

Diagnostic Tests for Cardiovascular Function

Because many of the same tests are used in the diagnosis and monitoring of a variety of cardiovascular disorders, a few of the basic tests are summarized here.

- An ECG is useful in the initial diagnosis and monitoring of arrhythmias, myocardial infarction, infection, and pericarditis (see Fig. 12-17). It is a noninvasive procedure and can illustrate the conduction activity of the heart as well as the effects of systemic abnormalities such as serum electrolyte imbalance. A portable *Holter monitor* may be worn by an individual to record ECG changes while he or she pursues daily activities. A log of activities is usually maintained to match with the changes in ECG. A normal baseline ECG recording is recommended for everyone; it can be used for comparison if cardiovascular disease ever develops.
- Valvular abnormalities or abnormal shunts of blood cause *murmurs* that may be detected by **auscultation** of heart sounds by means of a stethoscope. A recording of heart sounds may be made with a phonocardiograph. In *echocardiography*, ultrasound, or reflected sound waves, is used to record the image of the heart and valve movements (see Fig. 12-25). These tests provide useful information regarding valvular abnormalities, congenital defects, and changes in heart structure or function.
- *Exercise stress tests* (bicycle, step, or treadmill) are useful in assessing general cardiovascular function and in checking for exercise-induced problems such as arrhythmias. They may be used in fitness clubs before setting up an individualized exercise program or by insurance companies in the evaluation of an individual's health risks, as well as in cardiac rehabilitation programs following heart attacks or cardiovascular surgery.
- *Chest x-ray films* can be used to show the shape and size of the heart, as well as any evidence of pulmonary congestion associated with heart failure.
- *Nuclear imaging* with radioactive substances such as thallium assesses the size of an infarct in the heart, the extent of myocardial perfusion, and the function of the ventricles. *Tomographic studies*, which illustrate various levels of a tissue mass may be used when available. *Nuclear medicine studies* can identify dead or damaged areas of myocardial tissues and may be used to assess the extent of myocardial damage after a myocardial infarction.
- *SPECT* is a specialized CAT scan that accurately assesses cardiac ischemia at rest. Therapeutic intervention is not possible during this procedure. (Compare with coronary angiography below.)
- *Cardiac catheterization*, passing a catheter through an appropriate blood vessel, usually a large vein in the leg, into the ventricle, may also be utilized to visualize the inside of the heart, measure pressures, and assess valve and heart function. Determination of *central venous pressure* and *pulmonary capillary wedge pressure*, which indicate blood flow to and from the heart, can be made with a catheter. After contrast dye is injected into the ventricle, fluoroscopy can

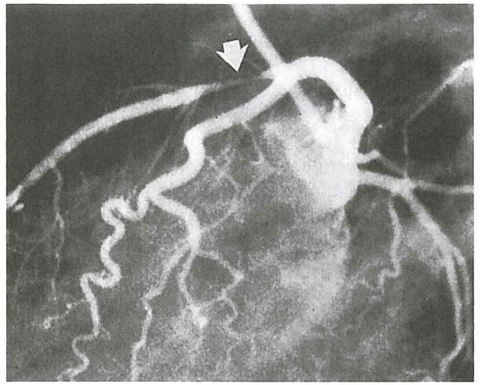

FIGURE 12-8 Coronary angiography shows stenosis (*arrow*) of left anterior descending coronary artery. *(From Braunwald E:* Heart Disease: A Textbook of Cardiovascular Medicine, *ed 4, Philadelphia, 1992, Saunders.)*

monitor blood movement continuously and check for abnormalities. There is some risk with this procedure, but it has proved beneficial in many instances.

- Blood flow in the coronary arteries can be visualized with coronary *angiography* (Fig. 12-8). Current research using very tiny ultrasound instruments within the vessels has proved more effective in diagnosing obstructions. Obstructions can be assessed and then treated with the basic catheterization procedure, with injected thrombolytic agents or laser therapy to break down clots, or balloon angioplasty to open a narrow coronary artery mechanically.
- *Troponin Blood test* is used to measure the levels of blood proteins called troponins. These proteins are released when cardiac muscle has been damaged. The more damage to the heart, the higher the levels of the troponins. Very high levels of the proteins are an indication that a heart attack has occurred.
- Blood flow in the peripheral vessels can be assessed with *Doppler* studies, in which essentially a microphone that records the sounds of blood flow or obstruction is placed over the blood vessel.
- *Blood tests* are used to assess serum triglyceride and cholesterol levels and the levels of sodium, potassium, calcium, and other electrolytes. Hemoglobin, hematocrit, blood cell counts, and the differential count for white cells are also routine aspects of blood tests.
- *Arterial blood gas determination* is essential in patients with shock or myocardial infarction, to check the current oxygen level and acid-base balance.

Other specific tests are mentioned under the appropriate topic and in Ready Reference 5 at the back of the book. More specialized tests may be necessary.

General Treatment Measures for Cardiac Disorders

Because some treatment measures apply to many disorders, a number of common therapies are covered here. Additional specific treatment modalities are mentioned with the disorder to which they apply.

1. Dietary modifications usually include reducing total fat intake and intake of saturated (hydrogenated or animal) fat as well as "trans" fats, which are commercially hydrogenated plant oils used to stabilize convenience foods. General weight reduction may be recommended for some persons. Salt (sodium) intake is decreased as well in order to reduce blood pressure. The American Heart Association has current dietary guidelines.
2. A regular exercise program is suggested to improve overall cardiovascular function and circulation to all areas of the body. Exercise assists in lowering serum lipid levels, increasing high-density lipoprotein (HDL) levels, and reducing stress levels, which in turn lessen peripheral resistance and blood pressure.
3. Cessation of cigarette smoking decreases the risk of coronary disease. Smoking appears to increase vasoconstriction and the heart rate, thus increasing the workload on the heart. Smoking increases platelet adhesion and the risk of thrombus (clot) formation, as well as increasing serum lipid levels. Also, carbon monoxide, a product of smoking, displaces oxygen from hemoglobin. In a compromised patient, this decrease in oxygen can be dangerous.
4. Drug therapy is an important component in the maintenance of cardiac patients. Many individuals take several drugs. Common medications include:
   - *Vasodilators,* such as nitroglycerin or long-acting isosorbide, reduce peripheral resistance systemically and therefore the workload for the heart and also act as coronary vasodilators. These actions provide a better balance of oxygen supply and demand in the heart muscle. Vasodilators may cause a decrease in blood pressure, resulting in dizziness or syncope and a flushed face. A person should sit quietly for a few minutes after taking nitroglycerin sublingually.
   - *Beta-blockers* such as metoprolol or atenolol are used to treat hypertension and dysrhythmias, as well as to reduce the number of angina attacks. These drugs block the $beta_1$-adrenergic receptors in the heart and prevent the SNS from increasing heart activity.
   - *Calcium channel blockers,* which block the movement of calcium ions into the cardiac and smooth muscle fiber, make up another group of effective cardiovascular drugs. Members of the group may be used as agents to decrease cardiac contractility, as an antidysrhythmic particularly for excessive atrial activity, or as an antihypertensive and vasodilator. They also serve a prophylactic purpose for angina. Some drugs such as diltiazem are more selective for the myocardium and reduce both conduction and contractility. Verapamil slows the heart rate by depressing the action of the SA and AV nodes, preventing tachycardia and fibrillation. Others, like nifedipine, are more effective as peripheral vasodilators. Amlodipine (Norvasc) has been useful in lowering blood pressure. Note that these drugs do not affect skeletal muscle contraction because more calcium is stored in skeletal muscle cells.
   - *Digoxin,* a cardiac glycoside, has been used for many years as a treatment for heart failure and as an antiarrhythmic drug for atrial dysrhythmias. It slows conduction of impulses and heart rate. Digoxin improves the efficiency of the heart because it also is inotropic, increasing the contractility of the heart. The contractions are less frequent but stronger. Because the effective dose is close to the toxic dose, patients must be observed for signs of toxicity, and blood levels of the drug must be checked periodically.
   - *Antihypertensive drugs* may be used to lower blood pressure to more normal levels. There are a number of groups in this category, including the adrenergic or sympathetic-blocking agents, the calcium blockers, the diuretics, the angiotensin-converting enzyme (ACE) inhibitors, and the angiotensin II receptor blocking agents. Combinations of drugs from various classifications are frequently prescribed to effectively lower blood pressure. Some of these drugs do cause orthostatic hypotension, a drop in blood pressure accompanied by dizziness, when arising from a recumbent position. These drugs may be used for treatment of essential hypertension or congestive heart failure or after myocardial infarction. Calcium blockers and beta-adrenergic blockers were discussed previously.
   - *Adrenergic-blocking drugs* may act on the SNS centrally (brain), may block peripheral (arteriolar) $alpha_1$-adrenergic receptors, or may act as direct vasodilators.
   - *Angiotensin-converting enzyme inhibitors* (ACE inhibitors) are currently preferred in the treatment of many patients with hypertension and congestive heart failure (CHF). They act by blocking the conversion of angiotensin I to angiotensin II (stimulated by the release of renin from the kidney). These drugs, such as enalapril (Vasotec), ramipril (Altace), captopril (Capoten) and perindopril (Coversyl), reduce both peripheral resistance (vasoconstriction) and aldosterone secretion (thus decreasing sodium and water retention). The result is a decrease in preload and afterload. Angiotensin II receptor blocking agents such as losartan (Cozaar) and irbesartan (Avapro) prevent angiotensin acting on blood vessels, and thus lower blood pressure. They do not appear to have side effects.
   - *Diuretics* remove excess sodium and water from the body through the kidneys by blocking the reabsorption of sodium or water (see Chapter 18). Patients often refer to them as "water pills." They are useful drugs in the treatment of high blood pressure and congestive heart failure because they increase urine output, reducing blood volume and edema. Examples are hydrochlorothiazide, a mild diuretic, and furosemide, a more potent drug. These diuretics may also remove excessive potassium from the body, requiring supplements to prevent hypokalemia. Spironolactone is an example of a "potassium-sparing" diuretic.
   - *Anticoagulants* or "blood thinners" may be used to reduce the risk of blood clot formation in coronary or systemic arteries or on damaged or prosthetic heart valves. In many cases, a small daily dose of aspirin (ASA) is recommended to decrease platelet adhesion. Oral anticoagulants such as warfarin (Coumadin) may be taken by individuals in high-risk groups. These drugs block the coagulation process (see Fig. 10-9). Individuals must be cautious about taking other medication, including nonprescription drugs, drinking alcohol, and making dietary changes, and avoid potentially traumatic activities. It is essential to monitor clotting ability, measuring prothrombin time or activated partial thromboplastin time closely in these patients to prevent hemorrhage and to observe patients for increased bleeding tendencies (see blood clotting in Chapter 10).
   - *Cholesterol or lipid-lowering* drugs are prescribed when diet and exercise are ineffective in reducing blood levels. These drugs, referred to as the "statins" include simvastatin (Zocor) and atorvastatin (Lipitor). They reduce low-density lipoprotein (LDL) and cholesterol content of the blood by blocking synthesis in the liver. Current investigations are assessing their ability to lower C-reactive protein levels, which has a role in the inflammation associated with atheroma formation.

Table 12-1 provides a summary of common cardiovascular drugs. A drug index may be found in Ready Reference 8 at the back of the book.

Coronary Artery Disease or Ischemic Heart Disease or Acute Coronary Syndrome

Sometimes called coronary heart disease, coronary artery disease includes angina pectoris or temporary cardiac ischemia and myocardial infarction or heart attack. Myocardial infarction results in damage to part of the heart muscle because of obstruction in a coronary