chapter 13

Cardiovascular System

A BURST BLOOD VESSEL. David Cone was one of the all-time great baseball pitchers when, in the spring of his eleventh season, he began to feel a nagging numbness in his right hand, the one he uses to pitch. He and his doctors were relieved when dye injected into his cardiovascular system showed no blood clot, but his symptoms persisted, even though he was taking blood-thinning drugs. Further investigation revealed an aneurysm, the sudden ballooning of a weakened area, in the subclavian artery in his right shoulder. Years of pitching had built up the muscle in the area, which began to press on the artery, eventually injuring it. Repeated trauma caused this aneurysm. Cone was lucky—the aneurysm did not burst and was surgically repaired.

The rock band R.E.M. was about halfway through a show on their European tour when drummer Bill Berry suddenly developed an excruciating headache in the middle of a song. Holding his head, he stopped playing. Berry's bandmates quickly ended the show and rushed him to a hospital. Berry had an aneurysm in his head. The weakened area had probably been present since birth. Thanks to quick surgery, Berry recovered, and the band was touring the United States two months later.

Photo:
A famous pitcher developed an injury in his pitching arm from repetitive motions, which pressed muscle against an artery, eventually causing an aneurysm.

Chapter Objectives

After studying this chapter, you should be able to do the following:

13.1 Introduction

1. Name the organs of the cardiovascular system, and discuss their functions. (p. 329)

13.2 Structure of the Heart

2. Name and describe the locations and functions of the major parts of the heart. (p. 331)
3. Trace the pathway of the blood through the heart and the vessels of the coronary circulation. (p. 333)

13.3 Heart Actions

4. Discuss the cardiac cycle, and explain how it is controlled. (p. 336)
5. Identify the parts of a normal ECG pattern, and discuss the significance of this pattern. (p. 339)

13.4 Blood Vessels

6. Compare the structures and functions of the major types of blood vessels. (p. 342)
7. Describe how substances are exchanged between blood in capillaries and the tissue fluid surrounding body cells. (p. 345)
8. Describe the mechanisms that aid in returning venous blood to the heart. (p. 346)

13.5 Blood Pressure

9. Explain how blood pressure is produced and controlled. (p. 347)

13.6 Paths of Circulation

10. Compare the pulmonary and systemic circuits of the cardiovascular system. (p. 351)

13.7–13.8 Arterial System—Venous System

11. Identify and locate the major arteries and veins of the pulmonary and systemic circuits. (p. 351)

Aids to Understanding Words

brady- [slow] *brady*cardia: Abnormally slow heartbeat.

diastol- [dilation] *diastol*ic pressure: Blood pressure when the ventricle of the heart is relaxed.

-gram [something written] electrocardio*gram:* Recording of the electrical changes in the heart muscle during a cardiac cycle.

papill- [nipple] *papill*ary muscle: Small mound of muscle within a ventricle of the heart.

syn- [together] *syn*cytium: Mass of merging cells that function together.

systol- [contraction] *systol*ic pressure: Blood pressure resulting from a ventricular contraction.

tachy- [rapid] *tachy*cardia: Abnormally fast heartbeat.

Key Terms

arteriole (ar-te´re-ōl)
atrium (a´tre-um)
capillary (kap´ĭ-lar´´e)
cardiac conduction system (kar´de-ak kon-duk´shun sis´tem)
cardiac cycle (kar´de-ak si´kl)
cardiac output (kar´de-ak owt´poot)
diastole (di-as´to-le)
electrocardiogram (ECG) (e-lek´´tro-kar´de-o-gram´´)
endocardium (en´´do-kar´de-um)
epicardium (ep´´ĭ-kar´de-um)
functional syncytium (funk´-shun-al sin-sish´e-um)
myocardium (mi´´o-kar´de-um)
pericardium (per´´ĭ-kar´de-um´)
peripheral resistance (pĕ-rif´er-al re-zis´tans)
pulmonary circuit (pul´mo-ner´´e sur´kit)
systemic circuit (sis-tem´ik sur´kit)
systole (sis´to-le)
vasoconstriction (vas´´o-kon-strik´shun)
vasodilation (vas´´o-di-la´shun)
ventricle (ven´tri-kl)
venule (ven´ūl)
viscosity (vis-kos´ĭ-te)

13.1 Introduction

The heart pumps 7,000 liters of blood through the body each day, contracting some 2.5 billion times in an average lifetime. This muscular pump forces blood through arteries, which connect to smaller-diameter vessels. The tiniest tubes, the capillaries, are the sites of nutrient, electrolyte, gas, and waste exchange. Capillaries converge into venules, which in turn converge into veins that return blood to the heart, completing the closed system of blood circulation. These structures—the pump and its vessels—form the cardiovascular system.

The cardiovascular system brings oxygen and nutrients to all body cells and removes wastes. A functional cardiovascular system is vital for survival because, without circulation, tissues lack a supply of oxygen and nutrients, and wastes accumulate. Under such conditions, the cells soon begin irreversible change, which quickly leads to death. Figure 13.1 shows the general pattern of blood transport in the cardiovascular system.

13.2 Structure of the Heart

The heart is a hollow, cone-shaped, muscular pump located within the thoracic cavity and resting on the diaphragm (fig. 13.2).

Size and Location of the Heart

Heart size varies with body size. An average adult's heart is about 14 centimeters long and 9 centimeters wide.

The heart is within the mediastinum, bordered laterally by the lungs, posteriorly by the vertebral column, and anteriorly by the sternum. The *base* of the heart, which attaches to several large blood vessels, lies beneath the second rib. The heart's distal end extends downward and to the left, terminating as a bluntly pointed *apex* at the level of the fifth intercostal space.

Coverings of the Heart

The **pericardium** (per´´ĭ-kar´de-um) encloses the heart and the proximal ends of the large blood vessels to which it attaches. The pericardium consists of an outer bag, the fibrous pericardium, that surrounds a more delicate, double-layered sac. The innermost layer of this sac, the *visceral pericardium* (epicardium), covers the heart. At the base of the heart, the visceral pericardium turns back on itself to become the *parietal pericardium,* which forms the inner lining of the fibrous pericardium (figs. 13.2 and 13.3; reference plate 3, p. 24).

The fibrous pericardium is dense connective tissue. It is attached to the central portion of the diaphragm, the posterior of the sternum, the vertebral column, and the large blood vessels emerging from the heart.

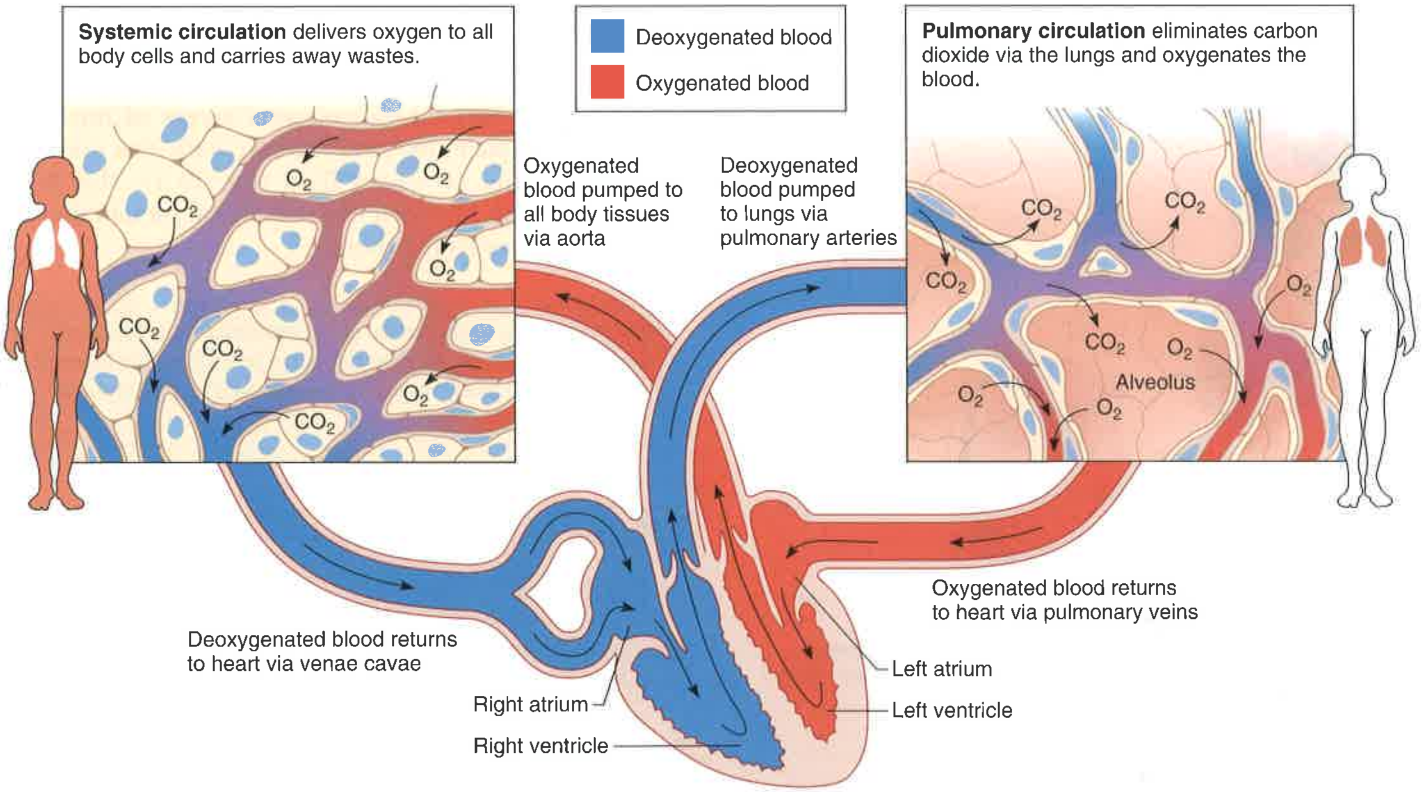


Figure 13.1
The cardiovascular system transports blood between the body cells and organs such as the lungs, intestines, and kidneys that communicate with the external environment.

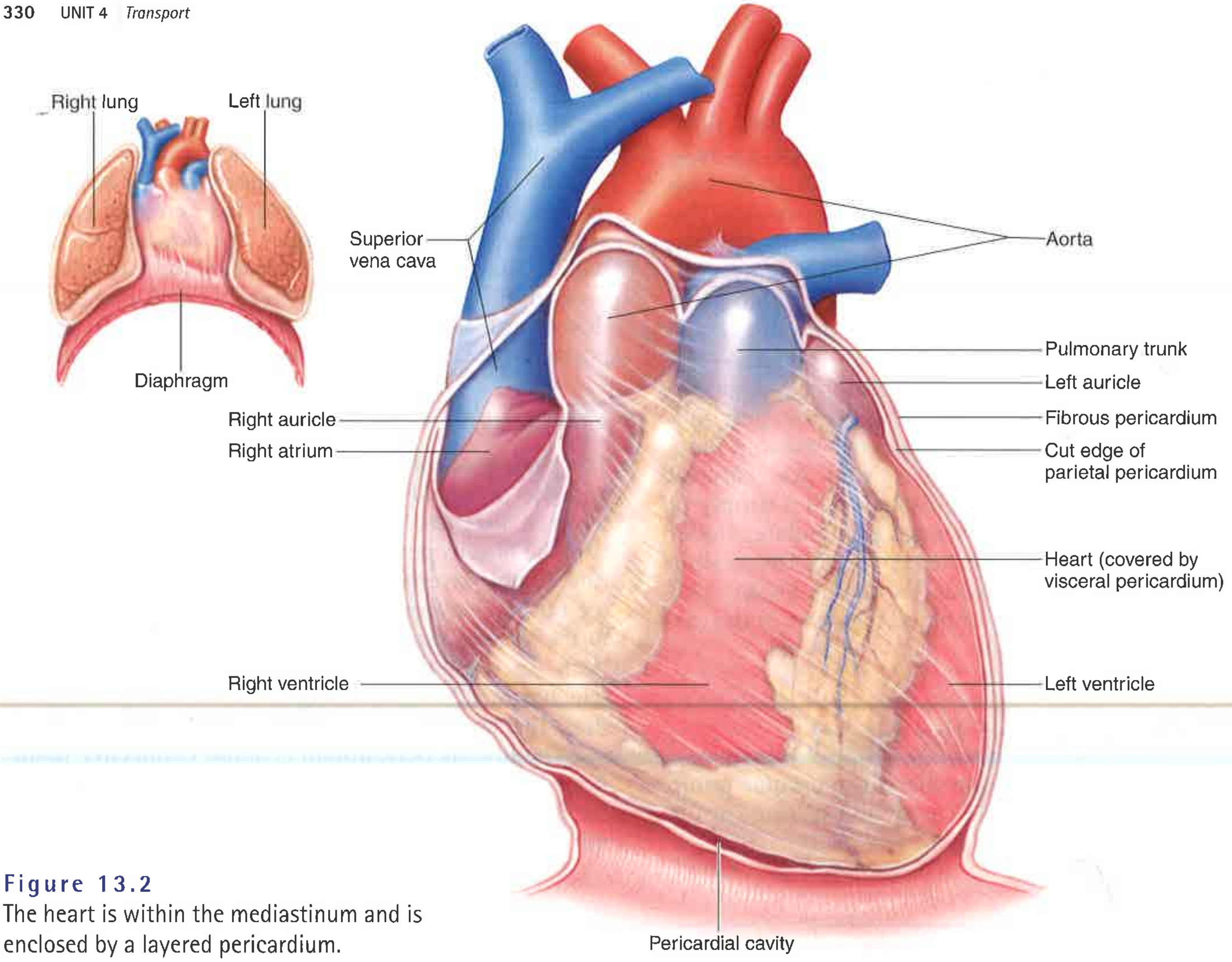


Figure 13.2
The heart is within the mediastinum and is enclosed by a layered pericardium.

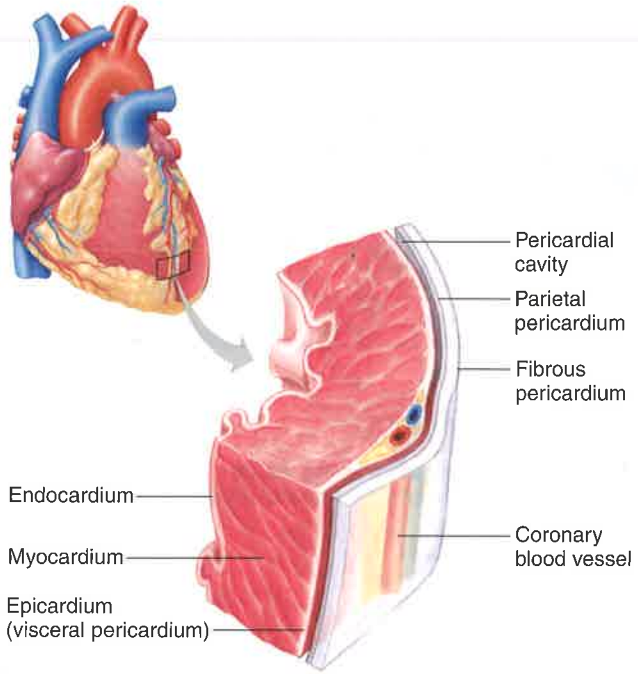


Figure 13.3
The heart wall has three layers: an endocardium, a myocardium, and an epicardium.

Between the parietal and visceral layers of the pericardium is a space, the *pericardial cavity,* that contains a small volume of serous fluid (fig. 13.3). This fluid reduces friction between the pericardial membranes as the heart moves within them.

In *pericarditis,* inflammation of the pericardium due to viral or bacterial infection produces adhesions that attach the layers of the pericardium to each other. This condition is very painful and interferes with heart movements.

CHECK YOUR RECALL

1. Where is the heart located?
2. Distinguish between the visceral pericardium and the parietal pericardium.

Wall of the Heart

The wall of the heart is composed of three distinct layers—an outer epicardium, a middle myocardium, and an inner endocardium (fig. 13.3). The **epicardium** (ep´´ĭ-kar´de-um), which corresponds to the visceral pericardium, protects the heart by reducing friction. It is a serous membrane that consists of connective tissue beneath epithelium. Its deeper portion often contains adipose tissue, particularly along the paths of coronary arteries and cardiac veins that carry blood through the myocardium.

The thick middle layer, or **myocardium** (mi´´o-kar´de-um), consists mostly of cardiac muscle tissue that pumps blood out of the heart chambers. The muscle fibers are organized in planes, separated by connective tissue richly supplied with blood capillaries, lymph capillaries, and nerve fibers.

The inner layer, or **endocardium** (en´´do-kar´de-um), consists of epithelium and connective tissue that contains many elastic and collagenous fibers. The endocardium also contains blood vessels and some specialized cardiac muscle fibers, called *Purkinje fibers,* described later in this chapter. The endocardium is continuous with the inner linings of blood vessels attached to the heart.

Heart Chambers and Valves

Internally, the heart is divided into four hollow chambers—two on the left and two on the right (fig. 13.4). The upper chambers, called **atria** (a´tre-ah; singular, *atrium*), have thin walls and receive blood returning to the heart. Small, earlike projections called *auricles* extend anteriorly from the atria. The lower chambers, the **ventricles** (ven´tri-klz), receive blood from the atria and contract to force blood out of the heart into arteries.

A solid, wall-like **septum** separates the atrium and ventricle on the right side from their counterparts on the left. As a result, blood from one side of the heart never mixes with blood from the other side (except in the fetus, see chapter 20, p. 540). An *atrioventricular valve* (A-V valve), the tricuspid on the right and the bicuspid on the left, ensures one-way blood flow between the atria and ventricles.

The right atrium receives blood from two large veins—the *superior vena cava* and the *inferior vena cava.* A smaller vein, the *coronary sinus,* also drains blood into the right atrium from the myocardium of the heart itself.

A large **tricuspid valve,** which has three *cusps* as its name implies, lies between the right atrium and the right ventricle (fig. 13.4). The valve permits blood to move from the right atrium into the right ventricle and prevents backflow.

Strong, fibrous strings called *chordae tendineae* attach to the cusps of the tricuspid valve on the ventricular side. These strings originate from small mounds of cardiac muscle tissue, the **papillary muscles,** that project inward from the walls of the ventricle. The papillary

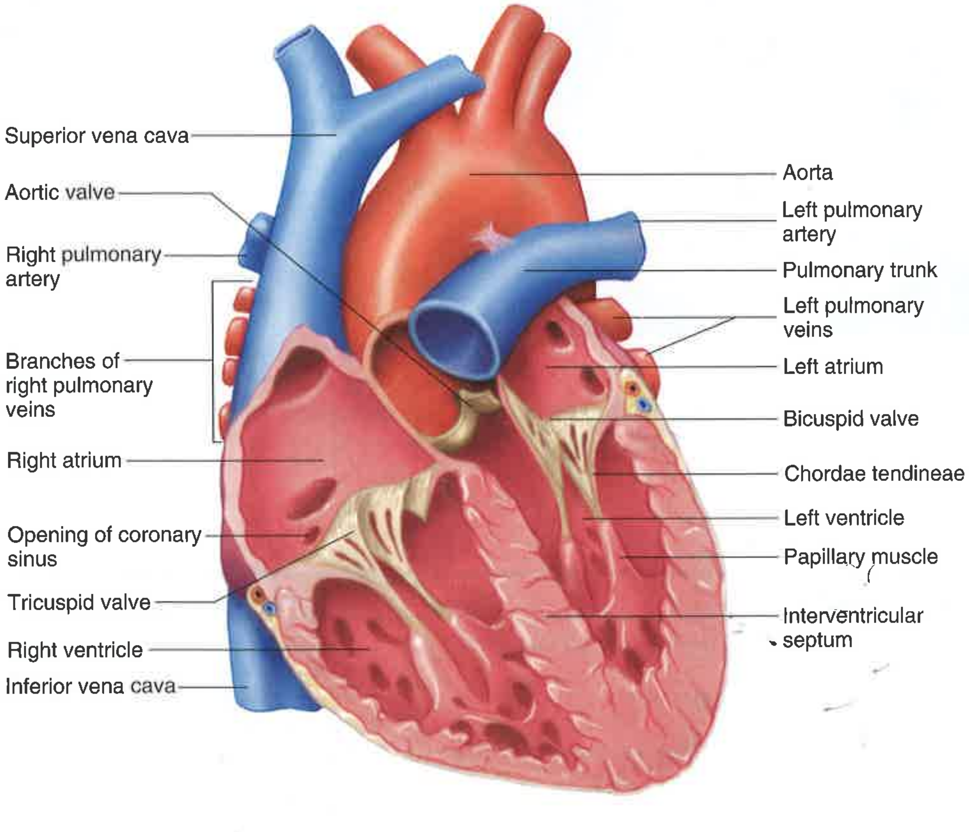


Figure 13.4
Coronal section of the heart showing the connection between the left ventricle and the aorta as well as the four hollow chambers.

muscles contract when the ventricle contracts. As the tricuspid valve closes, these muscles pull on the chordae tendineae and prevent the cusps from swinging back into the atrium.

The right ventricle has a thinner muscular wall than the left ventricle (fig. 13.4). This right chamber pumps blood a short distance to the lungs against a relatively low resistance to blood flow. The left ventricle, on the other hand, must force blood to all the other parts of the body against a much greater resistance to flow.

When the muscular wall of the right ventricle contracts, the blood inside its chamber is put under increasing pressure, and the tricuspid valve closes passively. As a result, the only exit for the blood is through the *pulmonary trunk,* which divides to form the left and right *pulmonary arteries* that lead to the lungs. At the base of this trunk is a **pulmonary valve** with three cusps. This valve allows blood to leave the right ventricle and prevents backflow into the ventricular chamber (fig. 13.5).

The left atrium receives blood from the lungs through four *pulmonary veins*—two from the right lung and two from the left lung. Blood passes from the left atrium into the left ventricle through the **bicuspid (mitral) valve,** which prevents blood from flowing back into the left atrium from the ventricle. As with the tricuspid valve, the papillary muscles and the chordae tendineae prevent the cusps of the bicuspid valve from swinging back into the left atrium during ventricular contraction.

When the left ventricle contracts, the bicuspid valve closes passively, and the only exit is through a large artery, the **aorta.** At the base of the aorta is the **aortic valve,** which has three cusps. The aortic valve opens and allows blood to leave the left ventricle as it contracts. When the ventricular muscles relax, this valve closes and prevents blood from backing up into the ventricle (fig. 13.5).

The bicuspid and tricuspid valves are called atrioventricular valves because they are between atria and ventricles. The pulmonary and aortic valves are called "semilunar" because of the half-moon shapes of their cusps. Table 13.1 summarizes the locations and functions of the heart valves.

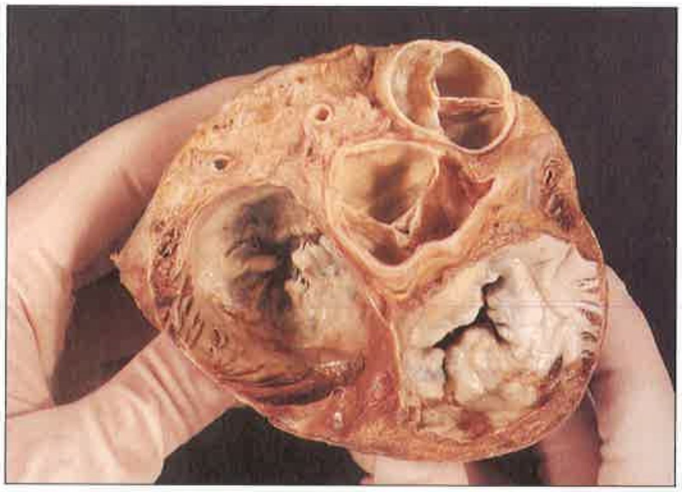


Figure 13.5
Photograph of a transverse section through the heart showing the four valves (superior view, see also fig. 13.6).

M*itral valve prolapse (MVP)* affects up to 6% of the U.S. population. In this condition, one (or both) of the cusps of the mitral valve stretches and bulges into the left atrium during ventricular contraction. The valve usually continues to function adequately, but sometimes blood regurgitates into the left atrium. Through a stethoscope, a regurgitating MVP sounds like a click at the end of ventricular contraction, then a murmur as blood goes back through the valve into the left atrium. Symptoms of MVP include chest pain, palpitations, fatigue, and anxiety.

The mitral valve can be damaged by certain species of *Streptococcus* bacteria. Endocarditis, an inflammation of the endocardium due to an infection, appears as a plantlike growth on the valve. People with MVP are particularly susceptible to endocarditis. Individuals with MVP must take antibiotics before undergoing dental work to prevent infection by *Streptococcus* bacteria in the mouth.

✓ CHECK YOUR RECALL

1. Describe the layers of the heart wall.
2. Name and locate the four chambers and valves of the heart.
3. Describe the function of each heart valve.

TABLE 13.1 HEART VALVES

VALVE	LOCATION	FUNCTION
Tricuspid valve	Opening between right atrium and right ventricle	Prevents blood from moving from right ventricle into right atrium during ventricular contraction
Pulmonary valve	Entrance to pulmonary trunk	Prevents blood from moving from pulmonary trunk into right ventricle during ventricular relaxation
Bicuspid (mitral) valve	Opening between left atrium and left ventricle	Prevents blood from moving from left ventricle into left atrium during ventricular contraction
Aortic valve	Entrance to aorta	Prevents blood from moving from aorta into left ventricle during ventricular relaxation

Skeleton of the Heart

Rings of dense connective tissue surround the pulmonary trunk and aorta at their proximal ends (fig. 13.6). These rings provide firm attachments for the heart valves and for muscle fibers; they also prevent the outlets of the atria and ventricles from dilating during contraction. The fibrous rings, together with other masses of dense connective tissue in the portion of the septum between the ventricles (interventricular septum), constitute the *skeleton of the heart.*

Path of Blood Through the Heart

Blood that is low in oxygen and high in carbon dioxide enters the right atrium through the venae cavae and coronary sinus. As the right atrial wall contracts, the blood passes through the tricuspid valve and enters the chamber of the right ventricle (fig. 13.7). When the right ventricular wall contracts, the tricuspid valve closes, and blood moves through the pulmonary valve and into the pulmonary trunk and its branches (pulmonary arteries).

From the pulmonary arteries, blood enters the capillaries associated with the alveoli of the lungs. Gas exchanges occur between blood in the capillaries and air in the alveoli. The freshly oxygenated blood, which is now relatively low in carbon dioxide, returns to the heart through the pulmonary veins that lead to the left atrium.

The left atrial wall contracts, and blood moves through the bicuspid valve and into the chamber of the left ventricle. When the left ventricular wall contracts, the bicuspid valve closes, and blood moves through the aortic valve and into the aorta and its branches.

Blood Supply to the Heart

The first two branches of the aorta, called the right and left **coronary arteries,** supply blood to the tissues of the heart. Their openings lie just beyond the aortic valve (fig. 13.8).

A thrombus or embolus that blocks or narrows a coronary artery branch deprives myocardial cells of oxygen, producing ischemia and a painful condition called *angina pectoris.* The pain usually occurs during physical activity, when oxygen requirements exceed oxygen supply. Pain lessens with rest. Emotional disturbance may also trigger angina pectoris.

Angina pectoris may cause a sensation of heavy pressure, tightening, or squeezing in the chest. The pain is usually felt in the region behind the sternum or in the anterior portion of the upper thoracic cavity, but may radiate to the neck, jaw, throat, upper limb, shoulder, elbow, back, or upper abdomen. Other symptoms include profuse perspiration (diaphoresis), difficulty breathing (dyspnea), nausea, or vomiting.

A blood clot completely obstructing a coronary artery or one of its branches (coronary thrombosis) kills part of the heart. This is a *myocardial infarction (MI),* more commonly known as a heart attack.

The heart must beat continually to supply blood to body tissues. Therefore, myocardial cells require a constant supply of freshly oxygenated blood. Branches of

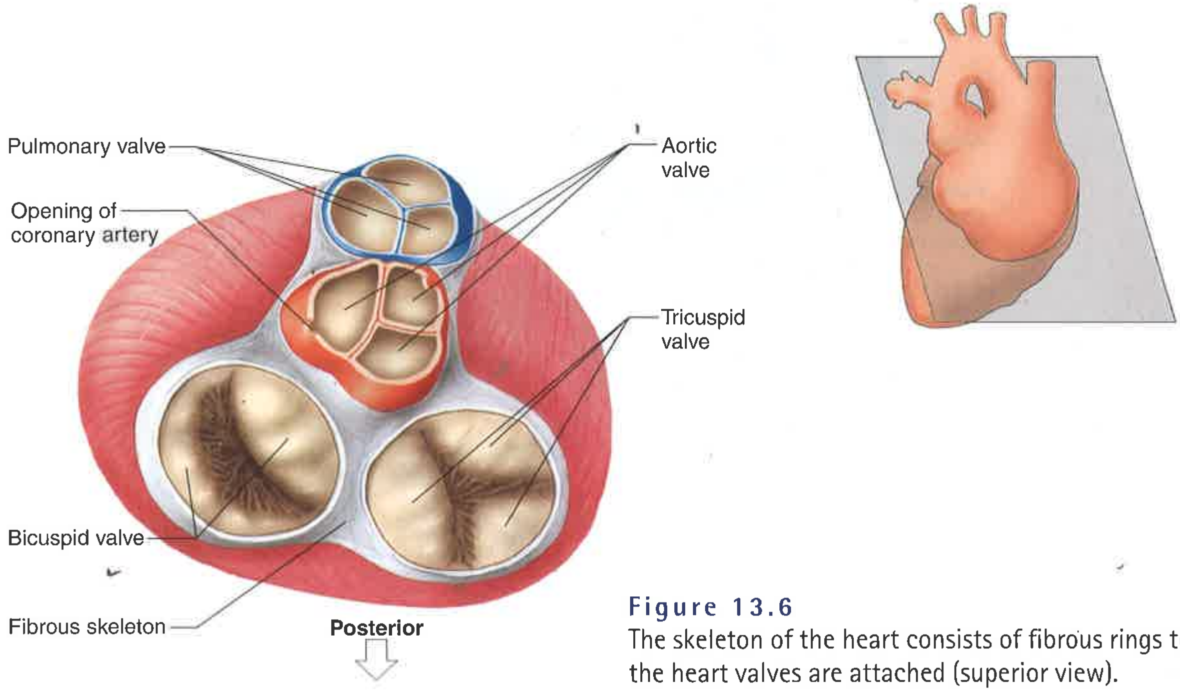


Figure 13.6
The skeleton of the heart consists of fibrous rings to which the heart valves are attached (superior view).

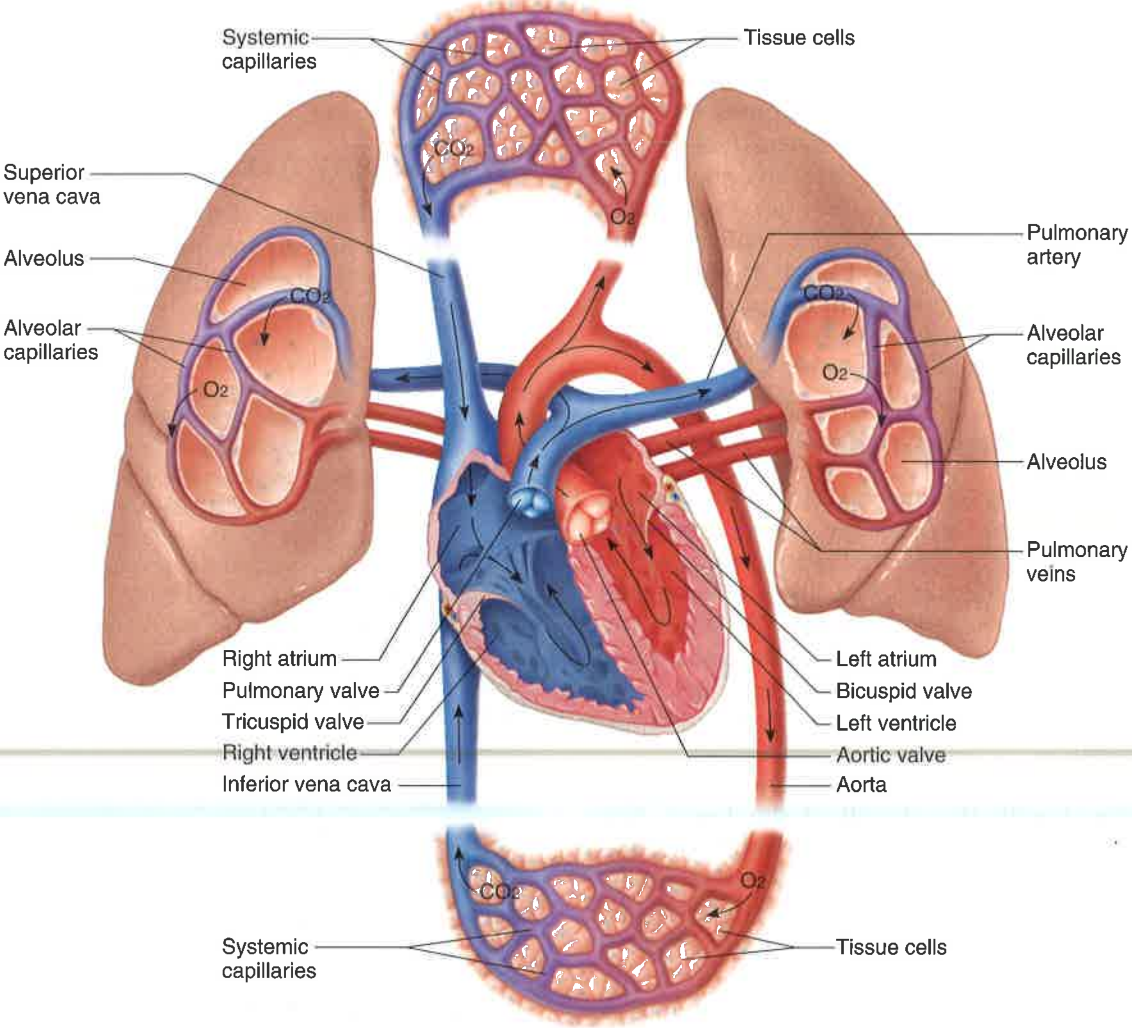


Figure 13.7
The right ventricle forces blood to the lungs, whereas the left ventricle forces blood to all other body parts. (Structures are not drawn to scale.)

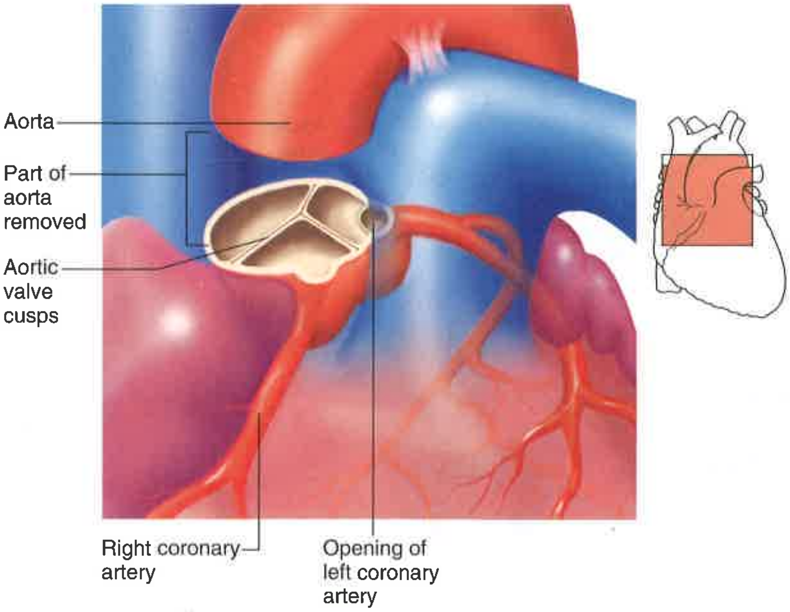


Figure 13.8
The openings of the coronary arteries lie just beyond the aortic valve.

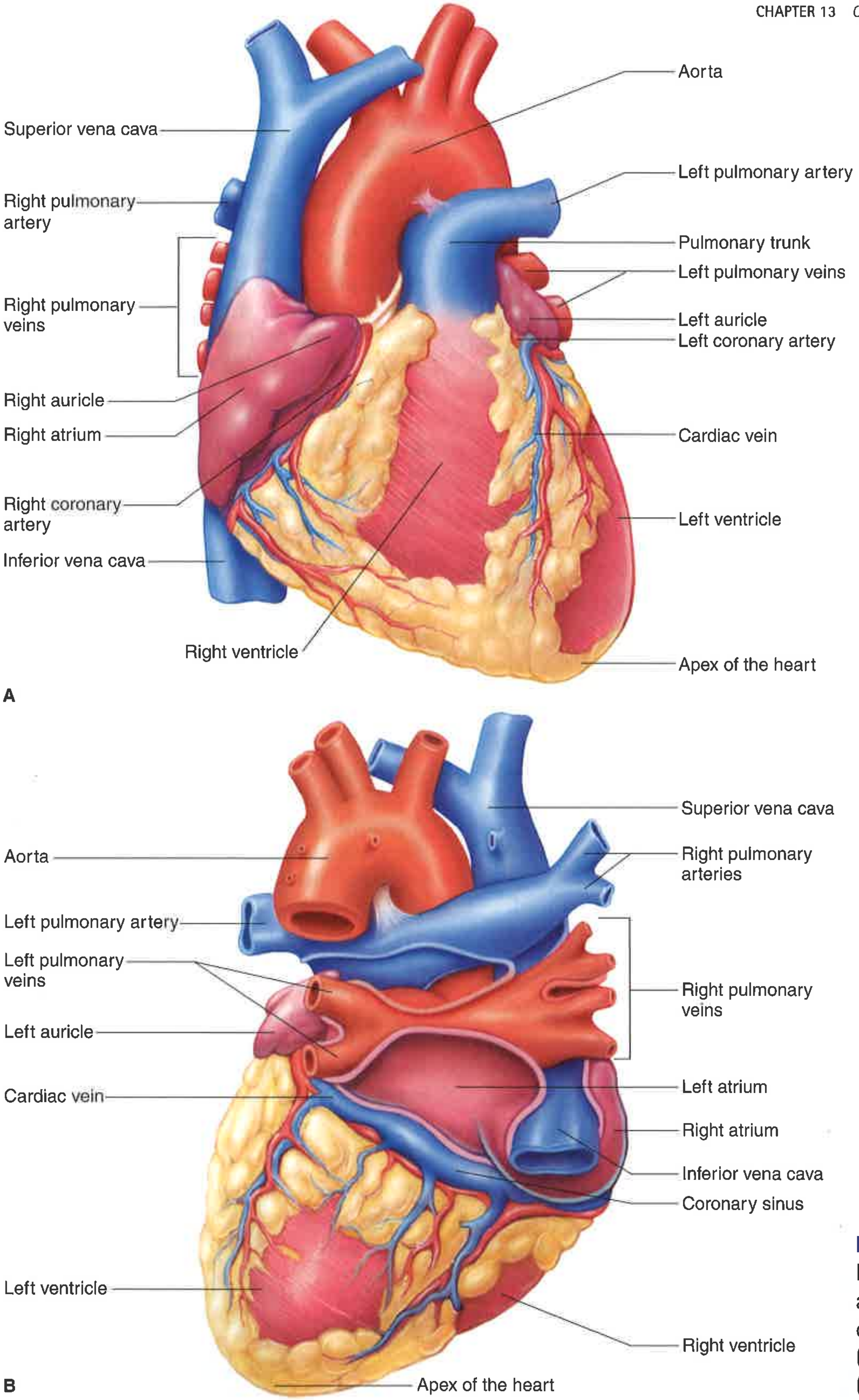


Figure 13.9 Blood vessels associated with the surface of the heart. (*A*) Anterior view. (*B*) Posterior view.

the coronary arteries feed the many capillaries of the myocardium (fig. 13.9). The smaller branches of these arteries usually have connections (anastomoses) between vessels that provide alternate pathways for blood, called collateral circulation. These detours in circulation may supply sufficient oxygen and nutrients to the myocardium when a coronary artery is blocked.

Branches of the **cardiac veins,** whose paths roughly parallel those of the coronary arteries, drain blood that has passed through myocardial capillaries.

As figure 13.9*B* shows, these veins join an enlarged vein on the heart's posterior surface—the **coronary sinus**—which empties into the right atrium (see fig. 13.4).

In *heart transplantation,* the recipient's failing heart is removed, except for the posterior walls of the right and left atria and their connections to the venae cavae and pulmonary veins. The donor heart is prepared similarly and is attached to the atrial cuffs remaining in the recipient's thoracic cavity. Finally, the recipient's aorta and pulmonary arteries are connected to those of the donor heart. The Clinical Connection at the end of this chapter describes a promising alternative to a heart transplant.

CHECK YOUR RECALL

1. Which structures make up the skeleton of the heart?
2. Review the path of blood through the heart.
3. Which vessels supply blood to the myocardium?
4. How does blood return from the cardiac tissues to the right atrium?

13.3 Heart Actions

The heart chambers function in coordinated fashion. Their actions are regulated so that atria contract, called atrial **systole** (sis´to-le), while ventricles relax, called ventricular **diastole** (di-as´to-le); then ventricles contract (ventricular systole) while atria relax (atrial diastole). Then the atria and ventricles both relax for a brief interval. This series of events constitutes a complete heartbeat, or **cardiac cycle** (kar´de-ak si´kl).

Cardiac Cycle

During a cardiac cycle, pressure within the heart chambers rises and falls. Pressure in the ventricles is low early in diastole, and the pressure difference between the atria and ventricles causes the A-V valves to open and the ventricles to fill. About 70% of the returning blood enters the ventricles prior to contraction, and ventricular pressure gradually increases. When the atria contract, the remaining 30% of returning blood is pushed into the ventricles, and ventricular pressure increases a bit more. Then, as the ventricles contract, ventricular pressure rises sharply, and as soon as the ventricular pressure exceeds the atrial pressure, the A-V valves close. At the same time, the papillary muscles contract, and by pulling on the chordae tendineae, they prevent the cusps of the A-V valves from bulging too far into the atria.

During ventricular contraction, the A-V valves remain closed. The atria are now relaxed, and pressure in the atria is quite low, even lower than venous pressure. As a result, blood flows into the atria from the large, attached veins. That is, as the ventricles are contracting, the atria are filling, already preparing for the next cardiac cycle (fig. 13.10).

As ventricular systole progresses, ventricular pressure continues to increase until it exceeds the pressure in the pulmonary trunk (right side) and aorta (left side). At this point, the pressure difference across the semilunar valves causes the pulmonary and aortic valves to open, and blood is ejected from each valve's respective ventricle into these arteries.

As blood flows out of the ventricles, ventricular pressure begins to drop, and it drops even further as the ventricles begin to relax. When ventricular pressure is lower than blood pressure in the aorta and pulmonary trunk, the pressure difference is reversed, and the semilunar valves close. The ventricles continue to relax, and as soon as ventricular pressure is less than atrial pressure, the A-V valves open, and the ventricles begin to fill once more. Atria and ventricles are both relaxed for a brief interval. The graph in figure 13.11 summarizes some of the changes that occur during a cardiac cycle.

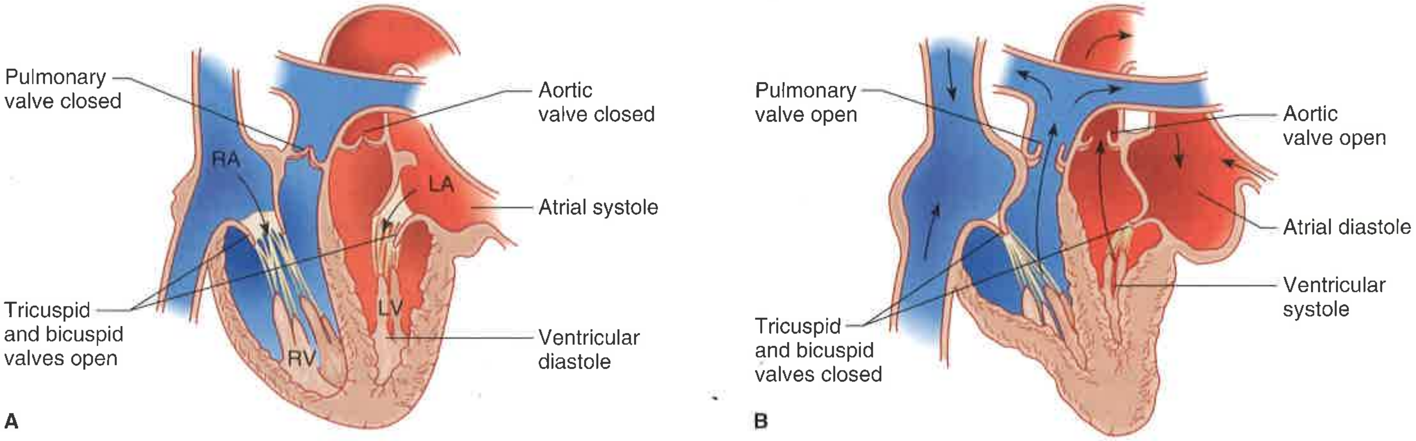


Figure 13.10
The atria (*A*) empty during atrial systole and (*B*) fill with blood during atrial diastole.

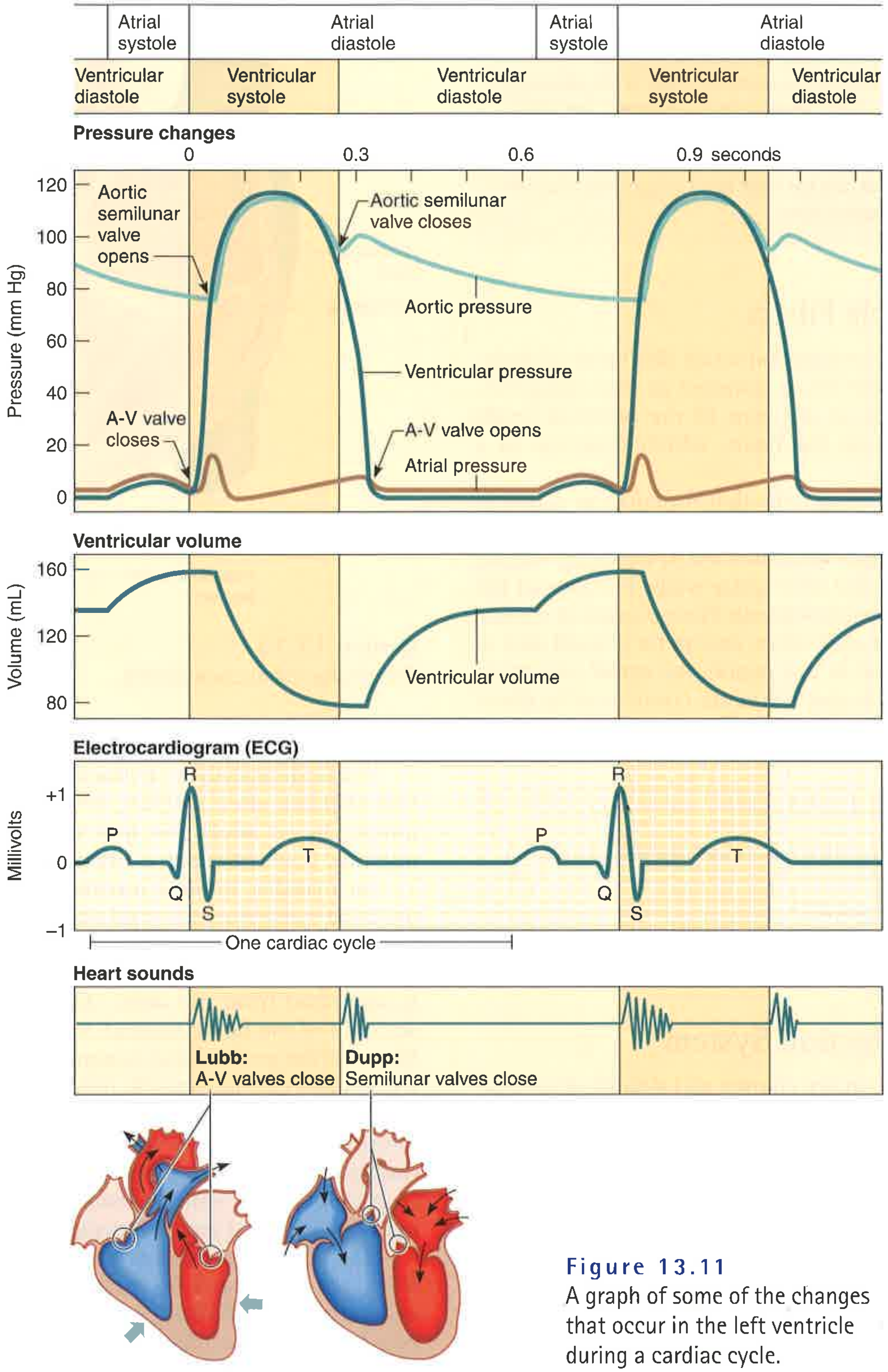


Figure 13.11
A graph of some of the changes that occur in the left ventricle during a cardiac cycle.

Heart Sounds

A heartbeat heard through a stethoscope sounds like *lubb-dupp*. These sounds are due to vibrations in the heart tissues associated with the closing of the valves.

The first part of a heart sound (*lubb*) occurs during ventricular contraction, when the A-V valves are closing. The second part (*dupp*) occurs during ventricular relaxation, when the pulmonary and aortic valves are closing (fig. 13.11).

Heart sounds provide information concerning the condition of the heart valves. For example, inflammation of the endocardium (endocarditis) may erode the edges of the valvular cusps. As a result, the cusps may not close completely, and some blood may leak back through the valve, producing an abnormal sound called a *murmur.* The seriousness of a murmur depends on the amount of valvular damage. Open heart surgery may repair or replace seriously damaged valves.

Cardiac Muscle Fibers

Cardiac muscle fibers function much like those of skeletal muscles, but the fibers connect in branching networks. Stimulation to any part of the network sends impulses throughout the heart, which contracts as a unit.

A mass of merging cells that function as a unit is called a **functional syncytium** (funk´shun-al sin-sish´e-um). Two such structures are in the heart—in the atrial walls and in the ventricular walls. Portions of the heart's fibrous skeleton separate these masses of cardiac muscle fibers from each other, except for a small area in the right atrial floor. In this region, the *atrial syncytium* and the *ventricular syncytium* are connected by fibers of the cardiac conduction system.

CHECK YOUR RECALL

1. Describe the pressure changes in the atria and ventricles during a cardiac cycle.
2. What causes heart sounds?
3. What is a functional syncytium?

Cardiac Conduction System

Throughout the heart are clumps and strands of specialized cardiac muscle tissue whose fibers contain only a few myofibrils. Instead of contracting, these areas initiate and distribute impulses throughout the myocardium. They comprise the **cardiac conduction system** (kar´de-ak kon-duk´shun sis´tem), which coordinates events of the cardiac cycle (fig. 13.12).

A key portion of this conduction system is the **sinoatrial node** (S-A node), a small, elongated mass of specialized cardiac muscle tissue just beneath the epicardium. It is located in the right atrium near the opening of the superior vena cava, and its fibers are continuous with those of the atrial syncytium.

The cells of the S-A node can reach threshold on their own, and their membranes contact one another. Without stimulation from nerve fibers or any other outside agents, the nodal cells initiate impulses that spread into the surrounding myocardium and stimulate cardiac muscle fibers to contract.

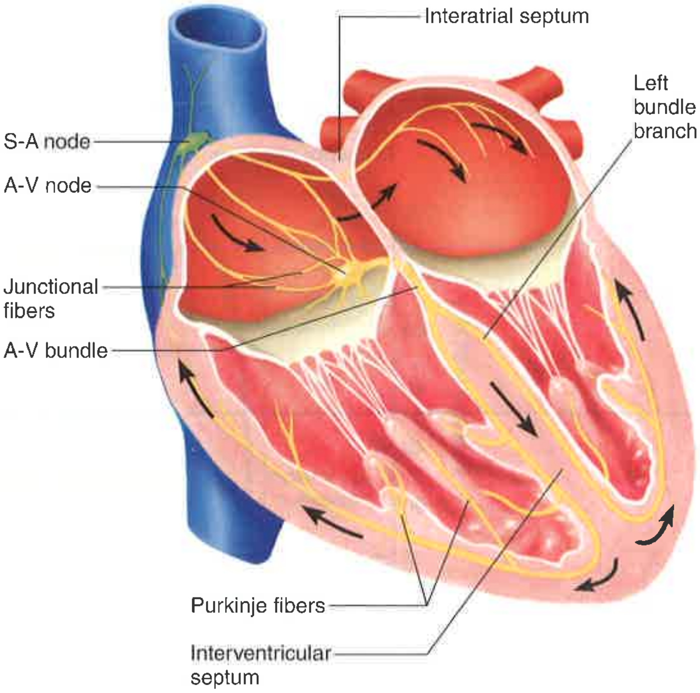


Figure 13.12
The cardiac conduction system.

S-A node activity is rhythmic. The S-A node initiates one impulse after another, seventy to eighty times a minute in an adult. Because it generates the heart's rhythmic contractions, it is often called the **pacemaker.**

As a cardiac impulse travels from the S-A node into the atrial syncytium, the right and left atria begin to contract almost simultaneously. The cardiac impulse does not pass directly into the ventricular syncytium, which is separated from the atrial syncytium by the fibrous skeleton of the heart. Instead, the impulse passes along fibers of the conduction system that lead to a mass of specialized cardiac muscle tissue called the **atrioventricular node** (A-V node). This node is located in the inferior portion of the septum that separates the atria (interatrial septum) and just beneath the endocardium. It provides the only normal conduction pathway between the atrial and ventricular syncytia.

The fibers that conduct the cardiac impulse into the A-V node (junctional fibers) have very small diameters, and because small fibers conduct impulses slowly, they delay impulse transmission. The impulse is delayed further as it moves through the A-V node, allowing more time for the atria to contract completely so they empty all their blood into the ventricles prior to ventricular contraction.

Once the cardiac impulse reaches the distal side of the A-V node, it passes into a group of large fibers that make up the **A-V bundle** (bundle of His). The A-V bundle enters the upper part of the interventricular septum and divides into right and left bundle branches that lie just beneath the endocardium. About halfway down the septum, the branches give rise to enlarged **Purkinje fibers** (pur-kin´je fi´berz).

Purkinje fibers spread from the interventricular septum into the papillary muscles, which project inward from ventricular walls and then continue downward to the apex of the heart. There they curve around the tips of the ventricles and pass upward over the lateral walls of these chambers. Along the way, the Purkinje fibers give off many small branches, which become continuous with cardiac muscle fibers (fig. 13.13).

The muscle fibers in ventricular walls form irregular whorls. When impulses on the Purkinje fibers stimulate these muscle fibers, the ventricular walls contract with a twisting motion (fig. 13.14). This action squeezes blood out of ventricular chambers and forces it into the aorta and pulmonary trunk.

CHECK YOUR RECALL

1. What kinds of tissues make up the cardiac conduction system?
2. How is a cardiac impulse initiated?
3. How is a cardiac impulse transmitted from the right atrium to the other heart chambers?

Electrocardiogram

An **electrocardiogram** (e-lek´´tor-kar´de-o-gram´´), or **ECG,** is a recording of the electrical changes that occur in the myocardium during a cardiac cycle. (This pattern occurs as action potentials stimulate cardiac muscle fibers to contract, but it is not the same as individual action potentials.) Because body fluids can conduct electrical currents, such changes can be detected on the surface of the body.

To record an ECG, electrodes are placed on the skin and connected by wires to an instrument that responds to very weak electrical changes by moving a pen or stylus on a moving strip of paper. Up-and-down movements of the pen correspond to electrical changes in the myocardium. Because the paper moves past the pen at a known rate, the distance between pen deflections indicates the time between phases of the cardiac cycle.

As figure 13.15*A* illustrates, a normal ECG pattern includes several deflections, or *waves,* during each cardiac cycle. Between cycles, the muscle fibers remain polarized, with no detectable electrical changes, and the pen does not move but simply marks along the baseline. When the S-A node triggers a cardiac impulse, atrial fibers depolarize, producing an electrical change. The pen moves, and at the end of the electrical change, returns to the base position. This first pen movement produces a *P wave,* corresponding to depolarization of the atrial fibers that will lead to contraction of the atria (fig. 13.15*B*).

When the cardiac impulse reaches ventricular fibers, they rapidly depolarize. Because ventricular walls are thicker than those of the atria, the electrical change is greater, and the pen deflects more. When the electrical change ends, the pen returns to the baseline, leaving a mark called the *QRS complex.* This mark consists of a *Q wave,* an *R wave,* and an *S wave,* and corresponds to depolarization of ventricular fibers just prior to the contraction of the ventricular walls.

The electrical changes occurring as the ventricular muscle fibers repolarize slowly produce a *T wave* as the

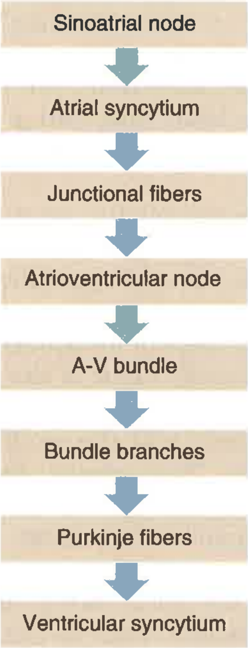


Figure 13.13
Components of the cardiac conduction system.

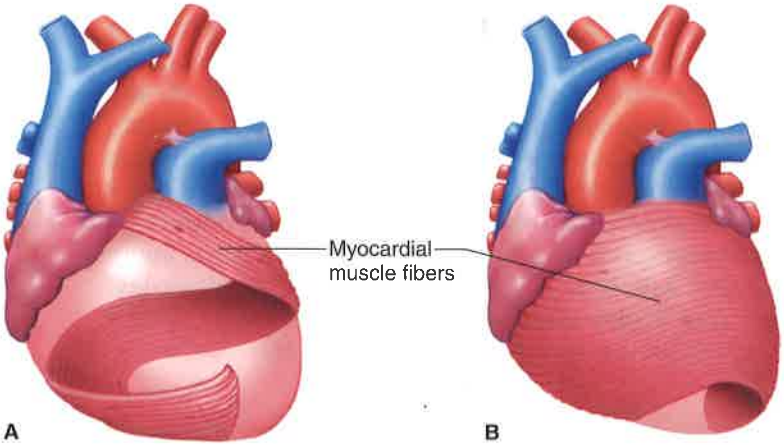


Figure 13.14
The muscle fibers within the ventricular walls are arranged in patterns of whorls. The fibers of groups (*A*) and (*B*) surround both ventricles in these anterior views of the heart.

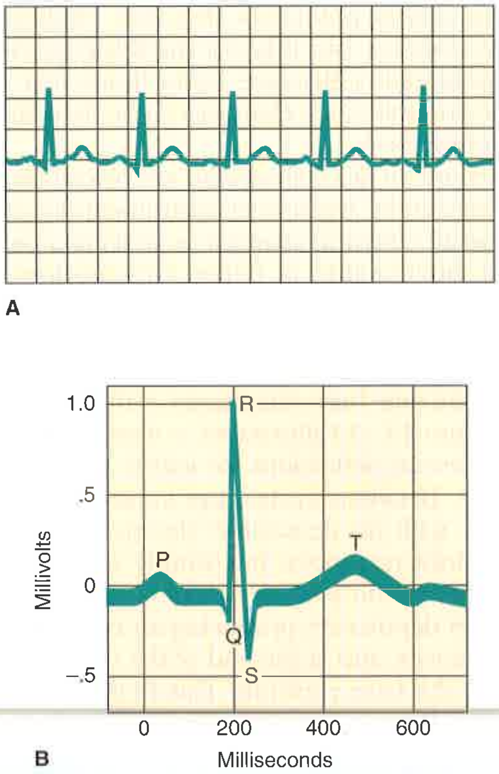


Figure 13.15
Electrocardiogram. (*A*) A normal ECG. (*B*) In an ECG pattern, the P wave results from a depolarization of the atria, the QRS complex results from a depolarization of the ventricles, and the T wave results from a repolarization of the ventricles.

pen deflects again, ending the ECG pattern. The record of atrial repolarization seems to be missing from the pattern because atrial fibers repolarize at the same time that ventricular fibers depolarize. Thus, the QRS complex obscures the recording of atrial repolarization.

Physicians use ECG patterns to assess the heart's ability to conduct impulses. For example, the time period between the beginning of a P wave and the beginning of a QRS complex, *P-Q interval* (or if the initial portion of the QRS complex is upright, P-R interval), indicates the time for the cardiac impulse to travel from the S-A node through the A-V node. Ischemia or other problems affecting the fibers of the A-V conduction pathways can increase this P-Q interval. Similarly, injury to the A-V bundle can extend the QRS complex, because it may take longer for an impulse to spread throughout the ventricular walls (fig. 13.16).

CHECK YOUR RECALL

1. What is an electrocardiogram?
2. Which cardiac events do the P wave, QRS complex, and T wave represent?

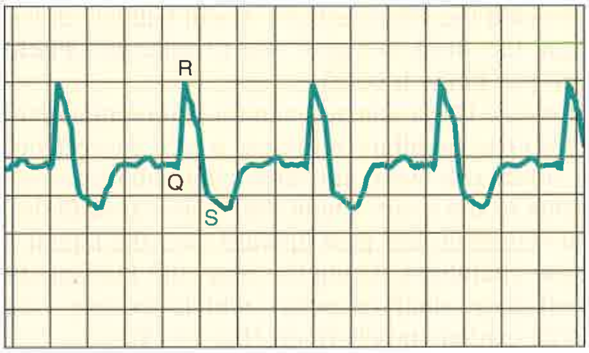


Figure 13.16
A prolonged QRS complex may result from damage to the A-V bundle fibers.

Regulation of the Cardiac Cycle

The volume of blood pumped changes to accommodate cellular requirements. For example, during strenuous exercise, skeletal muscles require more blood, and heart rate increases in response. Since the S-A node normally controls heart rate, changes in this rate are often a response to factors that affect the S-A node, such as the motor impulses carried on the parasympathetic and sympathetic nerve fibers (see chapter 9, p. 245).

The parasympathetic fibers that innervate the heart arise from neurons in the medulla oblongata (fig. 13.17). Most of these fibers branch to the S-A and A-V nodes. When the nerve impulses reach nerve fiber endings, they secrete acetylcholine, which decreases S-A and A-V nodal activity. As a result, heart rate decreases.

Parasympathetic fibers carry impulses continually to the S-A and A-V nodes, "braking" heart action. Consequently, parasympathetic activity can change heart rate in either direction. An increase in the impulses slows the heart rate, and a decrease in the impulses releases the parasympathetic "brake" and increases heart rate.

Sympathetic fibers reach the heart and join the S-A and A-V nodes as well as other areas of the atrial and ventricular myocardium. The endings of these fibers secrete norepinephrine in response to nerve impulses, which increases the rate and force of myocardial contractions.

The *cardiac control center* of the medulla oblongata maintains balance between the inhibitory effects of parasympathetic fibers and the excitatory effects of sympathetic fibers. This center receives sensory impulses from throughout the cardiovascular system and relays motor impulses to the heart in response. For example, receptors sensitive to stretch are located in certain regions of the aorta (aortic arch) and in the carotid arteries (carotid sinuses) (fig. 13.17). These receptors, called *baroreceptors* (pressoreceptors), can detect changes in blood pressure. Rising pressure stretches the receptors, and they signal the cardioin-

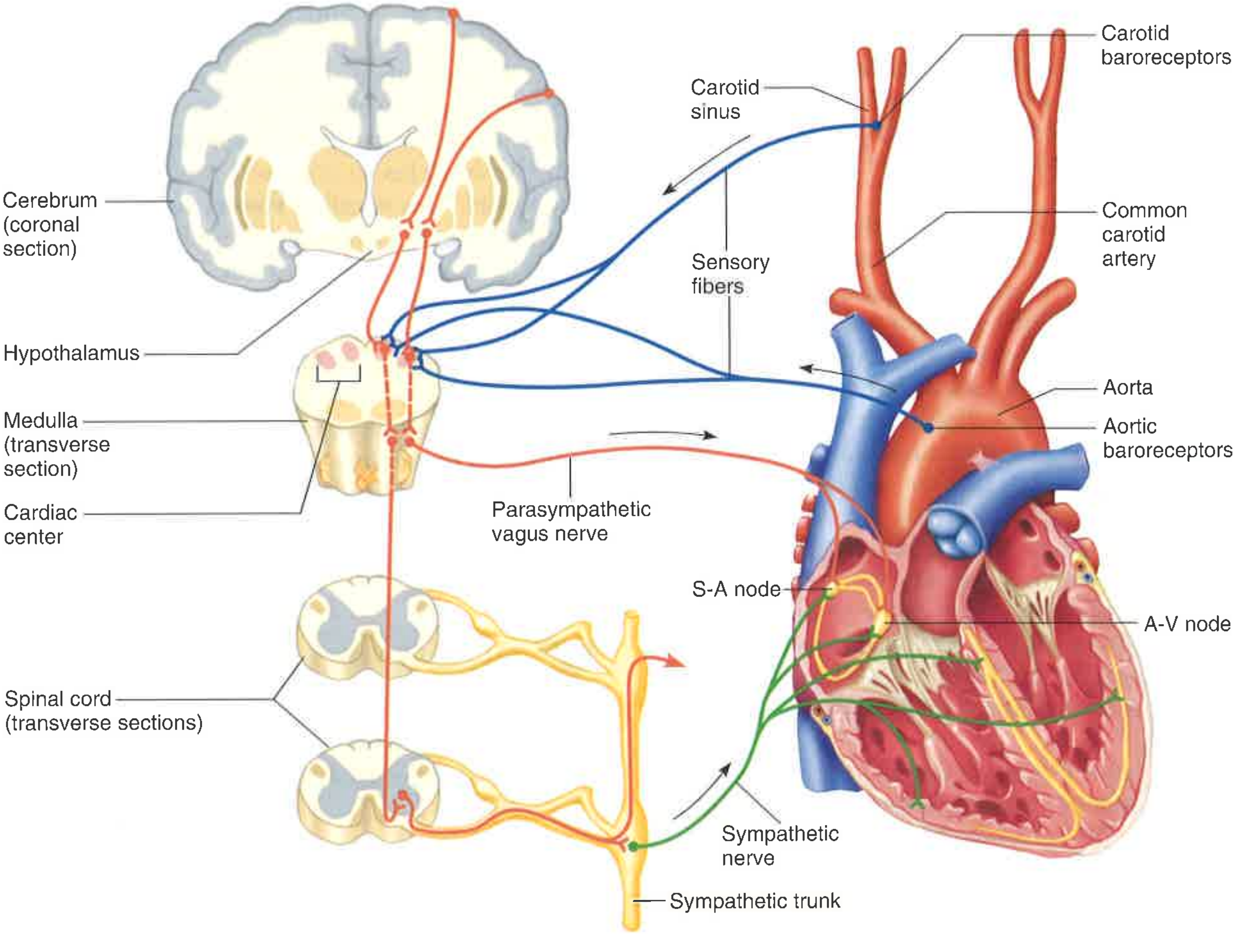


Figure 13.17
Autonomic nerve impulses alter the activities of the S-A and A-V nodes.

hibitor center in the medulla oblongata. In response, the medulla oblongata sends parasympathetic impulses to the heart via the vagus nerve, decreasing heart rate. This action helps lower blood pressure toward normal.

Impulses from the cerebrum or hypothalamus also influence the cardiac control center. Such impulses may decrease heart rate, as occurs when a person faints following an emotional upset, or they may increase heart rate during a period of anxiety.

Two other factors that influence heart rate are temperature change and certain ions. Rising body temperature increases heart action, which is why heart rate usually increases during fever. On the other hand, abnormally low body temperature decreases heart action.

Of the ions that influence heart action, the most important are potassium (K^+) and calcium (Ca^{+2}) ions. Excess potassium ions (hyperkalemia) decrease the rate and force of contractions. If potassium concentration drops below normal (hypokalemia), the heart may develop a potentially life-threatening abnormal rhythm (arrhythmia).

Excess extracellular calcium ions (hypercalcemia) increase heart actions, posing the danger that the heart will contract for a prolonged time. Conversely, low calcium concentration (hypocalcemia) depresses heart action.

✓ **CHECK YOUR RECALL**

1. How do parasympathetic and sympathetic impulses help control heart rate?
2. How do changes in body temperature affect heart rate?
3. Describe the effects on the heart of abnormal concentrations of potassium and calcium ions.

13.4 Blood Vessels

The blood vessels form a closed circuit of tubes that carries blood from the heart to cells and back again. These vessels include arteries, arterioles, capillaries, venules, and veins.

There are about 62,000 miles of blood vessels in the body—2 1/2 times around the world.

Arteries and Arterioles

Arteries are strong, elastic vessels that are adapted for carrying blood away from the heart under high pressure. These vessels subdivide into progressively thinner tubes and eventually give rise to finer, branched **arterioles** (ar-te´re-olz).

The wall of an artery consists of three distinct layers (fig. 13.18*A*). The innermost layer (*tunica interna*) is composed of a layer of simple squamous epithelium, called *endothelium,* which rests on a connective tissue membrane that is rich in elastic and collagenous fibers. Endothelium helps prevent blood clotting by providing a smooth surface that allows blood cells and platelets to flow through without being damaged and by secreting biochemicals that inhibit platelet aggregation. Endothelium also may help regulate local blood flow by secreting substances that dilate or constrict blood vessels. For example, endothelium releases the gas nitric oxide, which causes the smooth muscle of the vessel to relax.

The middle layer (*tunica media*) makes up the bulk of the arterial wall. It includes smooth muscle fibers, which encircle the tube, and a thick layer of elastic connective tissue.

The outer layer (*tunica externa*) is relatively thin and chiefly consists of connective tissue with irregularly organized elastic and collagenous fibers. This layer attaches the artery to the surrounding tissues.

The sympathetic branches of the autonomic nervous system innervate smooth muscle in artery and arteriole walls. Impulses on these *vasomotor fibers* stimulate the smooth muscles to contract, reducing the diameter of the vessel. This is called **vasoconstriction** (vas´´o-kon-strik´shun). If vasomotor impulses are inhibited, the muscle fibers relax, and the diameter of the vessel increases. This is called **vasodilation** (vas´´o-di-la´shun). Changes in the diameters of arteries and arterioles greatly influence blood flow and blood pressure.

The walls of the larger arterioles have three layers similar to those of arteries. These walls thin as arterioles approach capillaries. The wall of a very small arteriole consists only of an endothelial lining and some smooth muscle fibers, surrounded by a small amount of connective tissue (fig. 13.19).

CHECK YOUR RECALL

1. Describe the wall of an artery.
2. What is the function of smooth muscle in the arterial wall?
3. How is the structure of an arteriole different from that of an artery?

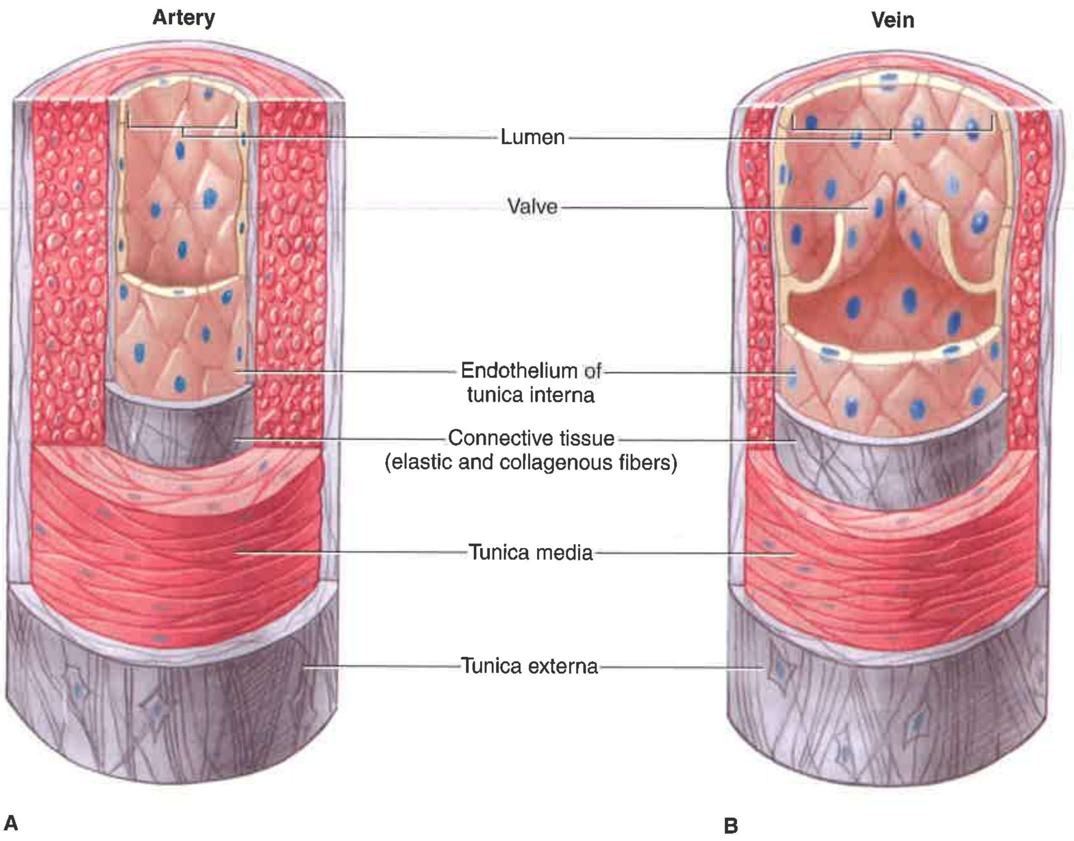


Figure 13.18
Blood vessels. (*A*) The wall of an artery. (*B*) The wall of a vein.

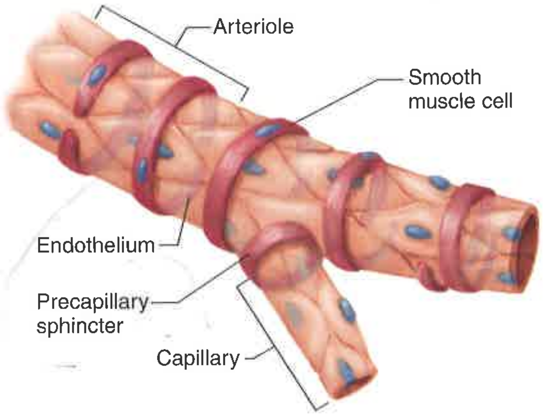


Figure 13.19
The smallest arterioles have only a few smooth muscle fibers in their walls. Capillaries lack these fibers.

Capillaries

Capillaries (kap′i-lar″ez), the smallest-diameter blood vessels, connect the smallest arterioles and the smallest venules. Capillaries are extensions of the inner linings of arterioles in that their walls are endothelium (fig. 13.19). These thin walls form the semipermeable layer through which substances in the blood are exchanged for substances in the tissue fluid surrounding body cells.

The openings in capillary walls are thin slits where endothelial cells overlap (fig. 13.20). The sizes of these openings and, consequently, the permeability of the capillary wall vary from tissue to tissue. For example, the openings are relatively small in capillaries of smooth, skeletal, and cardiac muscle, whereas those in capillaries associated with endocrine glands, the kidneys, and the lining of the small intestine are larger.

Capillary density within tissues varies directly with tissues' rates of metabolism. Muscle and nerve tissues, which use large quantities of oxygen and nutrients, are richly supplied with capillaries. Tissues with slow metabolic rates, such as cartilaginous tissues, the epidermis, and the cornea, lack capillaries.

Patterns of capillary arrangement also differ in various body parts. For example, some capillaries pass directly from arterioles to venules, but others lead to highly branched networks (fig. 13.21).

Smooth muscles that encircle capillary entrances regulate blood distribution in capillary pathways. These muscles form *precapillary sphincters,* which may close a capillary by contracting or open it by relaxing (see fig. 13.19). A precapillary sphincter responds to the demands of the cells supplied by the capillary. When these cells have low concentrations of oxygen and nutrients, the sphincter relaxes; when cellular requirements have been met, the sphincter may contract again. Thus, blood can follow different pathways through a tissue to meet the changing requirements of cells.

Routing of blood flow to different parts of the body is due to vasoconstriction and vasodilation of arterioles and precapillary sphincters. During exercise, for example, blood enters the capillary networks of the skeletal muscles, where the cells have increased oxygen and nutrient requirements. At the same time, blood can bypass some of the capillary networks in the digestive tract tissues, where demand for blood is less immediate.

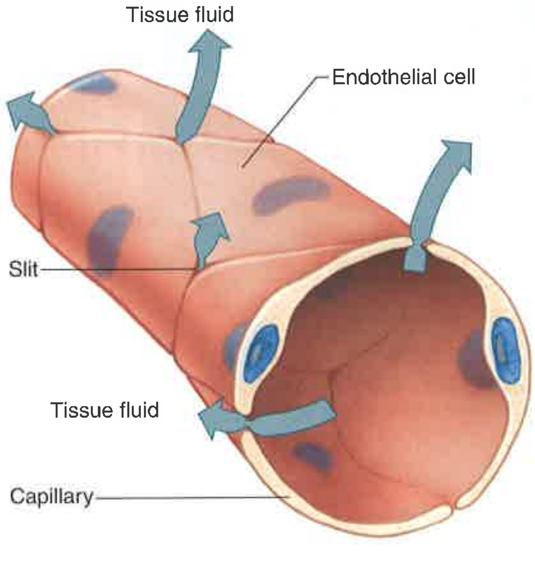


Figure 13.20
In capillaries, substances are exchanged between the blood and tissue fluid through openings (slits) separating endothelial cells.

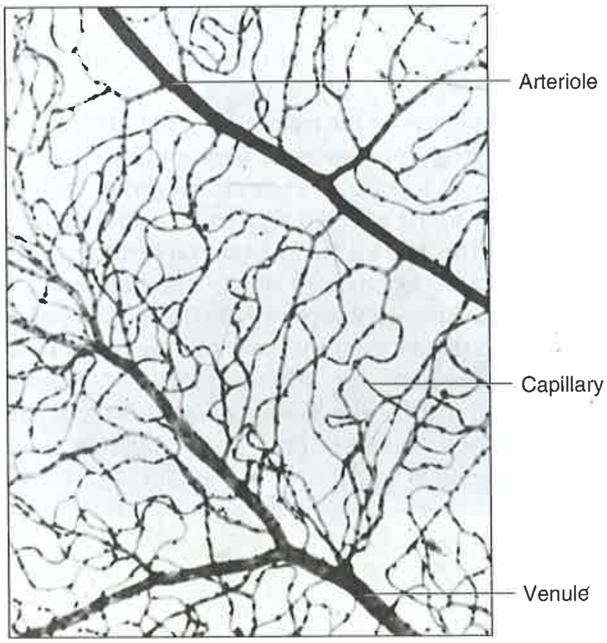


Figure 13.21
Light micrograph of a capillary network (100×).

Topic of Interest ATHEROSCLEROSIS

In the arterial disease *atherosclerosis,* deposits of fatty materials, particularly cholesterol, are formed within and on the inner lining of the arterial walls. Such deposits, called *plaque,* protrude into the lumens of vessels and interfere with blood flow (fig. 13A). Furthermore, plaque often forms a surface texture that can initiate formation of a blood clot, increasing the risk of developing thrombi or emboli that cause blood deficiency (ischemia) or tissue death (necrosis) downstream from the obstruction.

The walls of affected arteries may degenerate, losing their elasticity and becoming hardened, or *sclerotic.* In this stage of the disease, called *arteriosclerosis,* a sclerotic vessel may rupture under the force of blood pressure.

Risk factors for developing atherosclerosis include a fatty diet, elevated blood pressure, tobacco smoking, obesity, and lack of physical exercise. Emotional and genetic factors may also increase susceptibility to atherosclerosis.

Several treatments attempt to clear clogged arteries. In *percutaneous transluminal angioplasty,* a thin, plastic catheter is passed through a tiny incision in the skin and into the lumen of the affected blood vessel. The catheter, with a tiny deflated balloon at its tip, is pushed along the vessel and into the blocked region. Once in position, the balloon is inflated for several minutes, with a pressure high enough to compress the atherosclerotic plaque against the arterial wall, widening the arterial lumen and restoring blood flow. However, blockage can recur if the underlying cause is not addressed.

Laser energy is also used to destroy atherosclerotic plaque and to channel through arterial obstructions to increase blood flow. In *laser angioplasty,* the light energy of a laser is transmitted through a bundle of optical fibers passed through a small incision in the skin and into the lumen of an obstructed artery.

Another procedure for treating arterial obstruction is *bypass graft surgery.* A surgeon uses a portion of a vein from the patient's lower limb or elsewhere to connect a healthy artery to the affected artery at a point beyond the obstruction. This allows blood from the healthy artery to bypass the narrowed region of the affected artery and supply the tissues downstream. The vein is connected backward, so that its valves do not impede blood flow.

A new treatment for atherosclerosis is *fibroblast growth factor,* a body chemical given as a drug. It stimulates new blood vessels to grow in the heart, a process called angiogenesis.

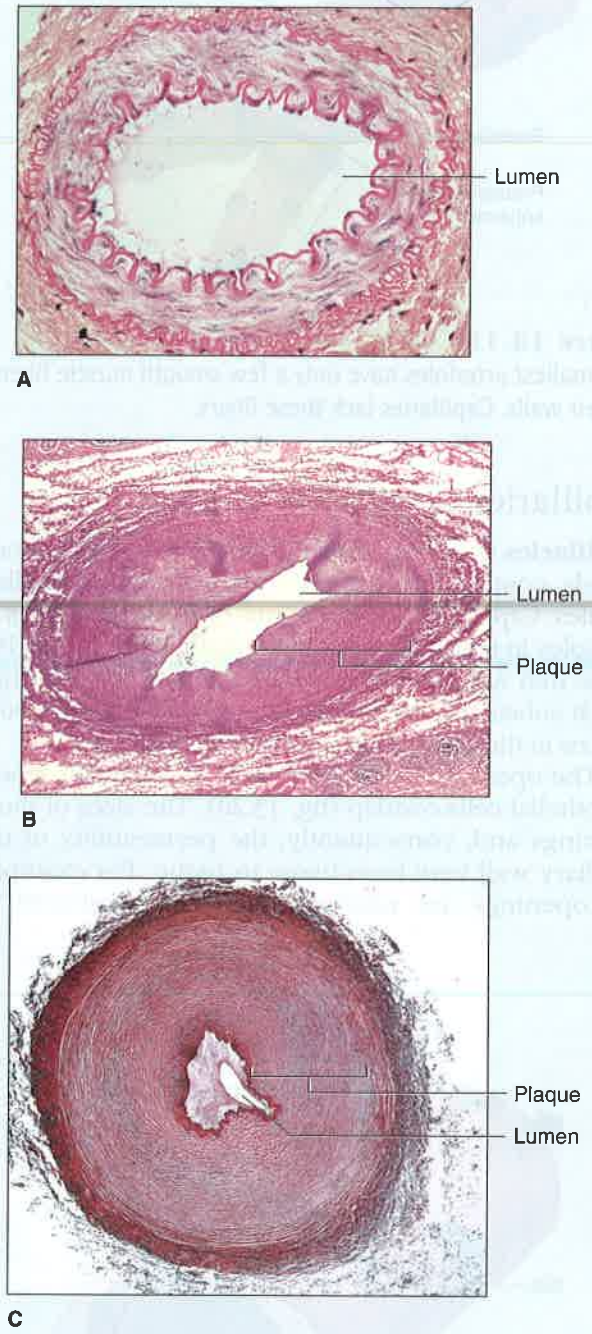


Figure 13A
Development of atherosclerosis. (A) Normal arteriole. (B, C) Accumulation of plaque on the inner wall of an arteriole.

CHECK YOUR RECALL

1. Describe a capillary wall.
2. What is the function of a capillary?
3. What controls blood flow into capillaries?

Exchanges in Capillaries

Gases, nutrients, and metabolic by-products are exchanged between the blood in capillaries and the tissue fluid surrounding body cells. The substances exchanged move through capillary walls by diffusion, filtration, and osmosis (see chapter 3, pp. 58–62).

Because blood entering systemic capillaries carries high concentrations of oxygen and nutrients, these substances diffuse through the capillary walls and enter the tissue fluid. Conversely, the concentrations of carbon dioxide and other wastes are generally greater in the tissues, and such wastes tend to diffuse into the capillary blood.

In the brain, the endothelial cells of capillary walls are more tightly fused than in other body regions. This arrangement forms a blood-brain barrier that protects the brain by keeping toxins out and preventing great biochemical fluctuations. Neuroglial cells also help form the blood-brain barrier. Unfortunately, the barrier keeps out many useful drugs as well. Researchers are developing ways to attach certain drugs to molecules that can cross the blood-brain barrier.

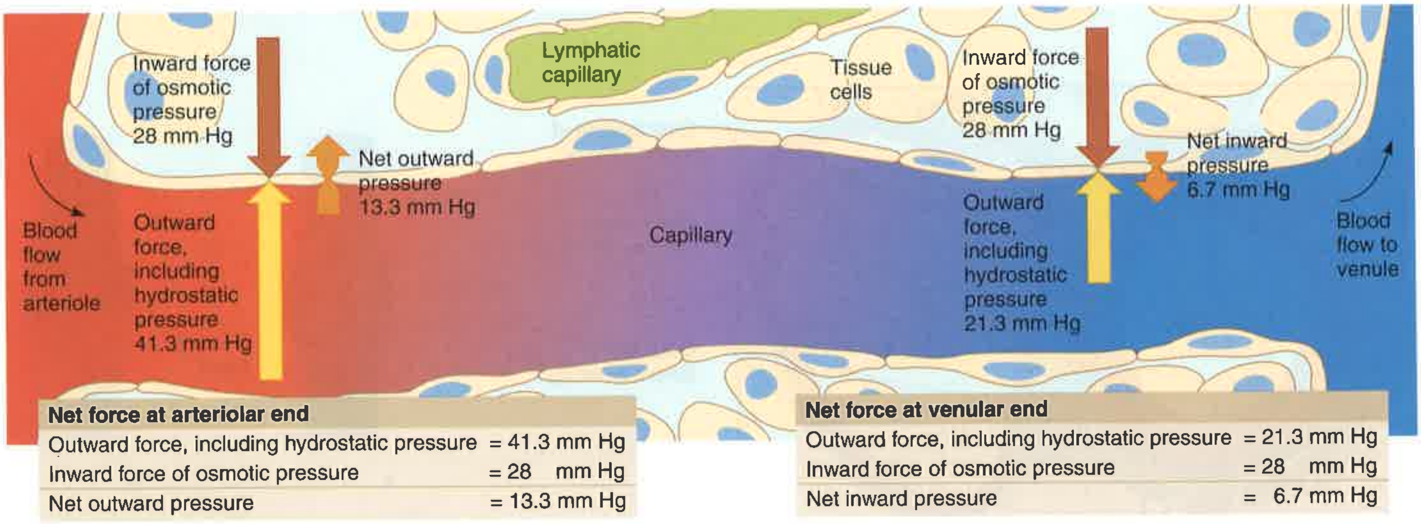
Plasma proteins generally remain in the blood because they are too large to diffuse through the membrane pores or slitlike openings between the endothelial cells of most capillaries. Also, these bulky proteins are not soluble in the lipid portions of capillary cell membranes.

Whereas diffusion depends on concentration gradients, filtration forces molecules through a membrane with hydrostatic pressure. In capillaries, the blood pressure generated when ventricle walls contract provides the force for filtration.

Blood pressure also moves blood through the arteries and arterioles. This pressure decreases as the distance from the heart increases, because of friction (peripheral resistance) between the blood and the vessel walls. For this reason, blood pressure is greater in the arteries than in the arterioles, and greater in the arterioles than in the capillaries. Blood pressure is similarly greater at the arteriolar end of a capillary than at the venular end. Therefore, the filtration effect occurs primarily at the arteriolar ends of capillaries.

The presence of a solute on one side of a cell membrane that cannot cross it creates an osmotic pressure. Because plasma proteins are trapped within the capillaries, they create an osmotic pressure that draws water into the capillaries. The term *colloid osmotic pressure* is often used to describe this osmotic effect due solely to the plasma proteins.

The effect of capillary blood pressure, favoring filtration, and the plasma colloid osmotic pressure, favoring reabsorption, have opposite actions. At the arteriolar end of capillaries, the blood pressure is higher than the colloid osmotic pressure, so at the arteriolar end of the capillary, filtration predominates. At the venular end, the colloid osmotic pressure is essentially unchanged, but the blood pressure has decreased due to resistance through the capillary. Thus, at the venular end, reabsorption predominates (fig. 13.22).



Net force at arteriolar end	
Outward force, including hydrostatic pressure	= 41.3 mm Hg
Inward force of osmotic pressure	= 28 mm Hg
Net outward pressure	= 13.3 mm Hg

Net force at venular end	
Outward force, including hydrostatic pressure	= 21.3 mm Hg
Inward force of osmotic pressure	= 28 mm Hg
Net inward pressure	= 6.7 mm Hg

Figure 13.22
Water and other substances leave capillaries because of a net outward filtration pressure at the capillaries' arteriolar ends. Water enters at the capillaries' venular ends because of a net inward force of osmotic pressure. Substances move in and out along the length of the capillaries according to their respective concentration gradients.

Normally, more fluid leaves the capillaries than returns to them. Lymphatic vessels collect the excess fluid and return it to the venous circulation. Chapter 14 (p. 369) discusses this mechanism.

Sometimes unusual events may increase blood flow to capillaries, and excess fluid enters spaces between tissue cells. This may occur in response to certain chemicals, such as histamine, that vasodilate the arterioles near capillaries and increase capillary permeability. Enough fluid may leak out of the capillaries to overwhelm lymphatic drainage. Affected tissues become swollen (edematous) and painful.

CHECK YOUR RECALL

1. What forces are responsible for the exchange of substances between blood and tissue fluid?
2. Why is the fluid movement out of a capillary greater at its arteriolar end than at its venular end?

Venules and Veins

Venules (ven´ūlz) are the microscopic vessels that continue from the capillaries and merge to form **veins.** The veins, which carry blood back to the atria, follow pathways that roughly parallel those of the arteries.

The walls of veins are similar to those of arteries in that they are composed of three distinct layers (see fig. 13.18*B*). However, the middle layer of the venous wall is poorly developed. Consequently, veins have thinner walls that contain less smooth muscle and less elastic tissue than those of comparable arteries, but their lumens have greater diameters (fig. 13.23).

Many veins, particularly those in the upper and lower limbs, contain flaplike *valves,* which project inward from their linings. Valves are usually composed of two leaflets that close if blood begins to back up in a vein (fig. 13.24). These valves aid in returning blood to the heart because they open if blood flow is toward the heart, but close if it is in the opposite direction.

Veins also function as blood reservoirs. For example, in hemorrhage accompanied by a drop in arterial blood pressure, sympathetic nerve impulses reflexly stimulate the muscular walls of the veins. The resulting venous constrictions help maintain blood pressure by returning more blood to the heart. This mechanism ensures a nearly normal blood flow even when as much as 25% of blood volume is lost. Table 13.2 summarizes the characteristics of blood vessels.

CHECK YOUR RECALL

1. How does the structure of a vein differ from that of an artery?
2. How does venous circulation help maintain blood pressure when hemorrhaging causes blood loss?

13.5 Blood Pressure

Blood pressure is the force blood exerts against the inner walls of blood vessels. Although this force occurs throughout the vascular system, the term *blood pressure* most commonly refers to pressure in arteries supplied by branches of the aorta.

The human heart creates enough pressure to squirt blood 30 feet.

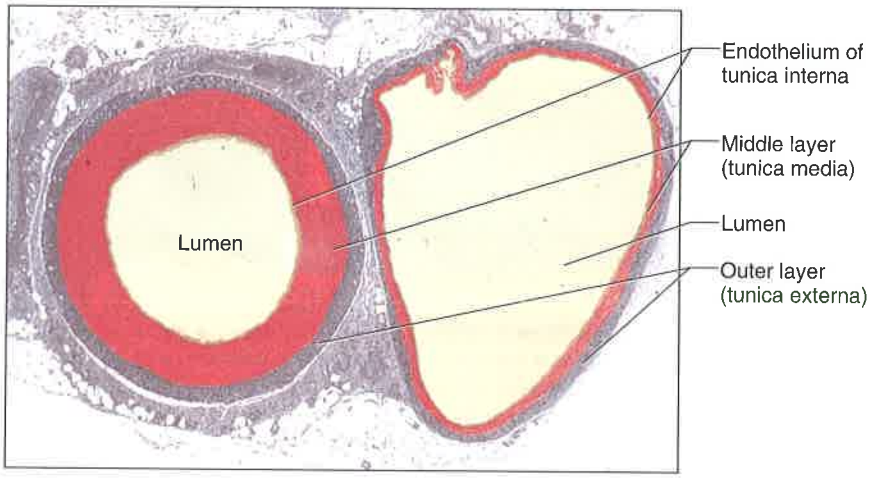


Figure 13.23
Note the structural differences in these cross sections of an artery and a vein (90×).

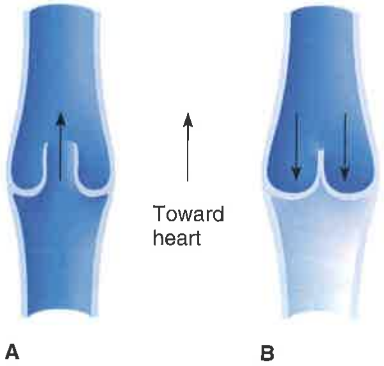


Figure 13.24
Venous valves (*A*) allow blood to move toward the heart, but (*B*) prevent blood from moving backward away from the heart.

TABLE 13.2 CHARACTERISTICS OF BLOOD VESSELS

VESSEL	TYPE OF WALL	FUNCTION
Artery	Thick, strong wall with three layers—an endothelial lining, a middle layer of smooth muscle and elastic tissue, and an outer layer of connective tissue	Carries blood under relatively high pressure from heart to arterioles
Arteriole	Thinner wall than an artery but with three layers; smaller arterioles have an endothelial lining, some smooth muscle tissue, and a small amount of connective tissue	Connects an artery to a capillary; helps control blood flow into a capillary by vasoconstricting or vasodilating
Capillary	Single layer of squamous epithelium	Provides a membrane through which nutrients, gases, and wastes are exchanged between the blood and tissue fluid; connects an arteriole to a venule
Venule	Thinner wall, less smooth muscle and elastic tissue than in an arteriole	Connects a capillary to a vein
Vein	Thinner wall than an artery but with similar layers; the middle layer is more poorly developed; some have flaplike valves	Carries blood under relatively low pressure from a venule to the heart; serves as blood reservoir; valves prevent a backflow of blood

Arterial Blood Pressure

Arterial blood pressure rises and falls in a pattern corresponding to the phases of the cardiac cycle. That is, contracting ventricles (ventricular systole) squeeze blood out and into the pulmonary trunk and aorta, which sharply increases the pressures in these arteries. The maximum pressure during ventricular contraction is called the **systolic pressure.** When the ventricles relax (ventricular diastole), the arterial pressure drops, and the lowest pressure that remains in the arteries before the next ventricular contraction is termed the **diastolic pressure** (fig. 13.25).

The surge of blood entering the arterial system during a ventricular contraction distends the elastic arterial walls, but the pressure drops almost immediately as the contraction ends, and the arterial walls recoil. This alternate expanding and recoiling of the arterial wall can be felt as a *pulse* in an artery that runs close to the surface.

✓ CHECK YOUR RECALL

1. What is *blood pressure?*
2. Distinguish between systolic and diastolic blood pressure.
3. What causes a pulse in an artery?

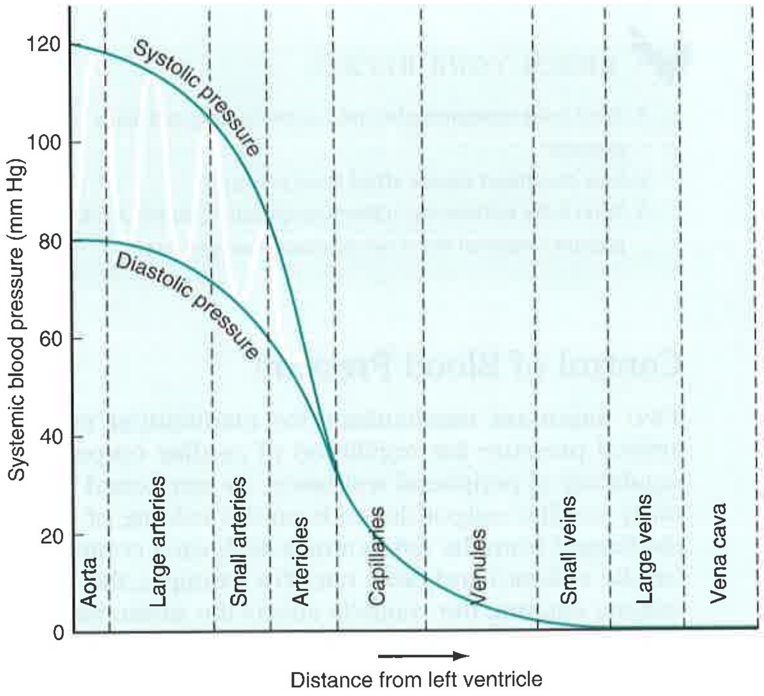


Figure 13.25
Blood pressure decreases as the distance from the left ventricle increases. Systolic pressure occurs during maximal ventricular contraction. Diastolic pressure occurs when the ventricles relax.

Factors That Influence Arterial Blood Pressure

Arterial blood pressure depends on a variety of factors. These include heart action, blood volume, peripheral resistance, and blood viscosity (fig. 13.26).

Heart Action

In addition to producing blood pressure by forcing blood into the arteries, heart action determines how much blood enters the arterial system with each ventricular contraction. The volume of blood discharged from the left ventricle with each contraction is called the **stroke volume** and equals about 70 milliliters in an average-weight male at rest. The volume discharged from the left ventricle per minute is called the **cardiac output,** calculated by multiplying the stroke volume by the heart rate in beats per minute (cardiac output = stroke volume × heart rate). Thus, if the stroke volume is 70 milliliters and the heart rate is 72 beats per minute, the cardiac output is 5,040 milliliters per minute.

Blood pressure varies with cardiac output. If either stroke volume or heart rate increases, so does cardiac output, and as a result, blood pressure initially rises. Conversely, if stroke volume or heart rate decreases, cardiac output decreases, and blood pressure also initially decreases.

Blood Volume

Blood volume equals the sum of the formed elements and plasma volumes in the vascular system. Although the blood volume varies somewhat with age, body size, and sex, it is usually about 5 liters for adults, or 8% of body weight in kilograms.

Blood pressure is normally directly proportional to blood volume within the cardiovascular system. Thus, any changes in blood volume can initially alter blood pressure. For example, if a hemorrhage reduces blood volume, blood pressure initially drops. If a transfusion restores normal blood volume, normal blood pressure may be reestablished. Blood volume can also fall if the fluid balance is upset, as happens in dehydration. Fluid replacement can reestablish normal blood volume and pressure.

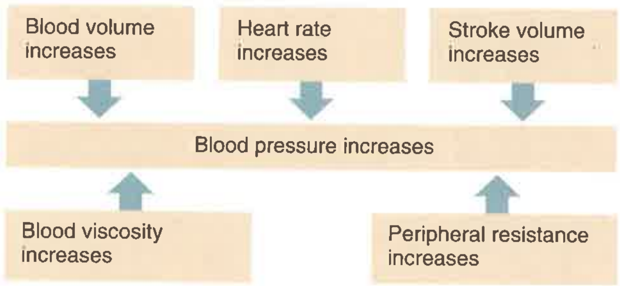


Figure 13.26
Some of the factors that influence arterial blood pressure.

Licorice can raise your blood pressure.

Peripheral Resistance

Friction between the blood and the walls of blood vessels produces a force called **peripheral resistance** (per-rif´er-al re-zis´tans), which hinders blood flow. Blood pressure must overcome this force if the blood is to continue flowing. Therefore, factors that alter the peripheral resistance change blood pressure. For example, contracting smooth muscles in arteriolar walls increase the peripheral resistance by constricting these vessels. Blood tends to back up into the arteries supplying the arterioles, and the arterial pressure rises. Dilation of arterioles has the opposite effect—peripheral resistance lessens, and arterial blood pressure drops in response.

Blood Viscosity

Viscosity (vis-kos´ĭ-te) is the ease with which a fluid's molecules flow past one another. The greater the viscosity, the greater the resistance to flowing.

Blood cells and plasma proteins increase blood viscosity. Since the greater the blood's resistance to flowing, the greater the force needed to move it through the vascular system, it is not surprising that blood pressure rises as blood viscosity increases and drops as viscosity decreases.

CHECK YOUR RECALL

1. What is the relationship between cardiac output and blood pressure?
2. How does blood volume affect blood pressure?
3. What is the relationship between peripheral resistance and blood pressure? Between blood viscosity and blood pressure?

Control of Blood Pressure

Two important mechanisms for maintaining normal arterial pressure are regulation of cardiac output and regulation of peripheral resistance. As mentioned previously, cardiac output depends on the volume of blood discharged from the left ventricle with each contraction (stroke volume) and heart rate. For example, the blood volume entering the ventricle affects the stroke volume. Entering blood mechanically stretches myocardial fibers in the ventricular wall. Within limits, the longer these fibers, the greater the force with which they contract. This relationship between fiber length (due to stretching of the cardiac muscle cell just before contraction) and force of contraction is called *Starling's law of the heart*. Because of it, the heart can respond to the immediate demands placed on it by the varying volumes of blood that return from the venous system. In other words, the more blood that enters the heart from the veins, the more the ventricular distends, the stronger it contracts, the greater the stroke volume, and the greater the cardiac output. The less blood that returns from the veins, the less the ventricle distends, and the lesser the stroke volume, and cardiac output, and the weaker the ventricular contraction. This mechanism ensures that the volume of blood discharged from the heart is equal to the volume entering its chambers.

Hypertension, or high blood pressure, is persistently elevated arterial pressure. It is one of the more common diseases of the cardiovascular system.

High blood pressure with unknown cause is called *essential* (also primary or idiopathic) *hypertension*. Elevated pressure can be caused by another problem, such as kidney disease, high sodium intake, obesity, psychological stress, and arteriosclerosis. In arteriosclerosis, decreased elasticity of arterial walls and narrowed arterial lumens increase blood pressure.

The consequences of prolonged, uncontrolled hypertension can be very serious. As the left ventricle works overtime to pump sufficient blood, the myocardium thickens, enlarging the heart. If coronary blood vessels cannot support this overgrowth, parts of the heart muscle die and are replaced with fibrous tissue. Eventually, the enlarged and weakened heart dies.

Hypertension also contributes to the development of atherosclerosis. Plaque accumulation in arteries may cause a *coronary thrombosis* or *coronary embolism*. Similar changes in brain arteries increase the chances of a *cerebral vascular accident (CVA)*, or *stroke*, due to a cerebral thrombosis, embolism, or hemorrhage.

Treatment of hypertension varies among patients and may include exercising regularly, controlling body weight, reducing stress, and limiting sodium in the diet. Drug treatment includes diuretics and/or inhibitors of sympathetic nerve activity.

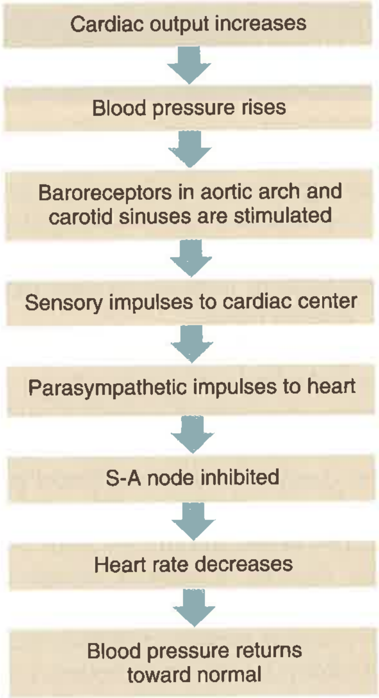


Figure 13.27
If blood pressure rises, baroreceptors initiate the cardioinhibitor reflex to lower the blood pressure.

Baroreceptors in the walls of the aorta and carotid arteries sense changes in blood pressure. If arterial pressure increases, nerve impulses travel from the baroreceptors to the cardiac center of the medulla oblongata. This center relays parasympathetic impulses to the S-A node in the heart, and the heart rate decreases in response. As a result of this *cardioinhibitor reflex*, cardiac output falls, and blood pressure decreases toward the normal level (fig. 13.27). Conversely, decreasing arterial blood pressure initiates the *cardioaccelerator reflex*, which sends sympathetic impulses to the S-A node. As a result, the heart beats faster, increasing cardiac output and arterial pressure. Other factors that increase heart rate and blood pressure include physical exercise, a rise in body temperature, and emotional responses, such as fear and anger.

Peripheral resistance also controls blood pressure. Changes in arteriole diameters regulate peripheral resistance. Because blood vessels with smaller diameters offer a greater resistance to blood flow, factors that cause arteriole vasoconstriction increase peripheral resistance, and factors causing vasodilation decrease resistance.

The *vasomotor center* of the medulla oblongata continually sends sympathetic impulses to smooth muscles in the arteriole walls, keeping them in a state of tonic contraction, which helps maintain the peripheral resistance associated with normal blood pressure. Because the vasomotor center responds to changes in blood pressure, it can increase peripheral resistance by increasing its outflow of sympathetic impulses, or it can decrease such resistance by decreasing its sympathetic outflow. In the latter case, the vessels vasodilate as sympathetic stimulation falls.

Whenever arterial blood pressure suddenly increases, baroreceptors in the aorta and carotid arteries signal the vasomotor center, and the sympathetic outflow to the arterioles falls. The resulting vasodilation decreases peripheral resistance, and blood pressure decreases toward the normal level.

Certain chemicals, including carbon dioxide, oxygen, and hydrogen ions, also influence peripheral resistance

Topic of Interest

EXERCISE AND THE CARDIOVASCULAR SYSTEM

The cardiovascular system adapts to exercise. The conditioned athlete experiences increases in heart pumping efficiency, blood volume, blood hemoglobin concentration, and the number of mitochondria in muscle fibers. All of these adaptations improve oxygen delivery to, and utilization by, muscle tissue.

An athlete's heart typically changes in response to these increased demands, and may enlarge 40% or more. Myocardial mass increases, ventricular cavities expand, and the ventricle walls thicken. Stroke volume increases, and heart rate decreases, as does blood pressure. The lowest heart rate recorded in an athlete was 25 beats per minute! To a physician unfamiliar with a conditioned cardiovascular system, a trained athlete may appear abnormal!

The cardiovascular system responds beautifully to a slow, steady buildup in exercise frequency and intensity. It does not react well to sudden demands—such as a person who never exercises suddenly shoveling snow or running 3 miles.

A recent study confirmed age-old anecdotal reports of unaccustomed exercise causing heart failure. Researchers in the United States and Germany asked about 1,000 patients hospitalized for heart attacks about their exercise habits and what they were doing in the hour before the attack. They also questioned the same number of people who had not had heart attacks about their activities during the same hours as the ill people. The people with heart attacks were much more likely to have been engaging in unaccustomed strenuous exercise. But the study also turned up good news for those who exercise regularly: Although sedentary people have a two- to sixfold increased risk of cardiac arrest while exercising than when not, people in shape have little or no excess risk while exercising.

For exercise to benefit the cardiovascular system, the heart rate must be elevated to 70–85% of its "theoretical maximum" for at least half an hour three times a week. You can calculate your theoretical maximum by subtracting your age from 220. If you are eighteen years old, your theoretical maximum is 202 beats per minute. Then, 70–85% of this value is 141–172 beats per minute. Some good activities for raising the heart rate are tennis, skating, skiing, handball, vigorous dancing, hockey, basketball, biking, and fast walking.

It is wise to consult a physician before starting an exercise program. People over age thirty are advised to have a stress test, which is an electrocardiogram taken during exercise. (The standard electrocardiogram is taken at rest.) An arrhythmia that appears only during exercise may indicate heart disease that has not yet produced symptoms.

by affecting precapillary sphincters and smooth muscle in arteriole walls. For example, increasing blood carbon dioxide, decreasing blood oxygen, and lowering blood pH relaxes smooth muscle in the systemic circulation. This increases local blood flow to tissues with high metabolic rates, such as exercising skeletal muscles. In addition, epinephrine and norepinephrine vasoconstrict many systemic vessels, increasing peripheral resistance.

✓ **CHECK YOUR RECALL**

1. What factors affect cardiac output?
2. What is the function of baroreceptors in the walls of the aorta and carotid arteries?
3. How does the vasomotor center control peripheral resistance?

Venous Blood Flow

Blood pressure decreases as blood moves through the arterial system and into the capillary networks, so that little pressure remains at the venular ends of capillaries (see fig. 13.25). Instead, blood flow through the venous system is only partly the direct result of heart action and depends on other factors, such as skeletal muscle contraction, breathing movements, and vasoconstriction of veins (*venoconstriction*).

Contracting skeletal muscles press on nearby vessels, squeezing the blood inside. As skeletal muscles press on veins with valves, some blood moves from one valve section to another (see fig. 13.24). This massaging action of contracting skeletal muscles helps push blood through the venous system toward the heart.

Respiratory movements also move venous blood. During inspiration, the pressure within the thoracic cavity is reduced as the diaphragm contracts and the rib cage moves upward and outward. At the same time, the pressure within the abdominal cavity is increased as the diaphragm presses downward on the abdominal viscera. Consequently, blood is squeezed out of abdominal veins and forced into thoracic veins. During exercise, these respiratory movements act with skeletal muscle contractions to increase return of venous blood to the heart.

Venoconstriction also returns venous blood to the heart. When venous pressure is low, sympathetic reflexes stimulate smooth muscles in the walls of veins to contract. The veins also provide a blood reservoir that can adapt its capacity to changes in blood volume. If some blood is lost and blood pressure falls, venoconstriction can force blood out of this reservoir. In both of these examples, venoconstriction helps maintain blood pressure by forcing more blood toward the heart.

CHECK YOUR RECALL

1. What is the function of venous valves?
2. How do skeletal muscles and respiratory movements affect venous blood flow?
3. What factors stimulate venoconstriction?

13.6 Paths of Circulation

The blood vessels can be divided into two major pathways. The **pulmonary circuit** (pul´mo-ner´´e sur´kit) consists of vessels that carry blood from the heart to the lungs and back to the heart. The **systemic circuit** (sis-tem´ik sur´kit) carries blood from the heart to all other parts of the body and back again (see fig. 13.1). The systemic circuit includes the coronary circulation, which supplies the heart itself and has already been described.

The circulatory pathways described in the following sections are those of an adult. Chapter 20 (p. 538) describes the fetal pathways, which are somewhat different.

Pulmonary Circuit

Blood enters the pulmonary circuit as it leaves the right ventricle through the pulmonary trunk. The pulmonary trunk extends upward and posteriorly from the heart. About 5 centimeters above its origin, it divides into the right and left pulmonary arteries (see fig. 13.4), which penetrate the right and left lungs, respectively. After repeated divisions, the pulmonary arteries give rise to arterioles that continue into the capillary networks associated with the walls of the alveoli (air sacs), where gas is exchanged between the blood and the air (see chapter 16, p. 450).

From the pulmonary capillaries, blood enters the venules, which merge to form small veins, and these veins in turn converge to form still larger veins. Four pulmonary veins, two from each lung, return blood to the left atrium. This completes the vascular loop of the pulmonary circuit.

Systemic Circuit

Freshly oxygenated blood moves from the left atrium into the left ventricle. Contraction of the left ventricle forces this blood into the systemic circuit. This circuit includes the aorta and its branches that lead to all the body tissues, as well as the companion system of veins that returns blood to the right atrium.

CHECK YOUR RECALL

1. Distinguish between the pulmonary and systemic circuits of the cardiovascular system.
2. Trace the path of blood through the pulmonary circuit from the right ventricle.

13.7 Arterial System

The **aorta** is the largest-diameter artery in the body. It extends upward from the left ventricle, arches over the heart to the left, and descends just anterior and to the left of the vertebral column. Figure 13.28 shows the aorta and its main branches.

Principal Branches of the Aorta

The first portion of the aorta is called the *ascending aorta*. Located at its base are the three cusps of the aortic valve, and opposite each cusp is a swelling in the aortic wall called an **aortic sinus.** The right and left coronary arteries arise from two of these sinuses (see fig. 13.8).

Three major arteries originate from the *arch of the aorta* (aortic arch): the **brachiocephalic** (brăk´´e-o-sĕ-fal´ik) **artery,** the left **common carotid** (kah-rot´id) **artery,** and the left **subclavian** (sub-kla´ve-an) **artery.**

The upper part of the *descending aorta* is positioned to the left of the midline. It gradually moves medially and finally lies directly in front of the vertebral column at the level of the twelfth thoracic vertebra. The portion of the descending aorta above the diaphragm is the **thoracic aorta.** It gives off many small branches to the thoracic wall and thoracic visceral organs.

Below the diaphragm, the descending aorta becomes the **abdominal aorta,** and it gives off branches to the abdominal wall and various abdominal organs. Branches to abdominal organs include: the **celiac** (se´le-ak) **artery,** which gives rise to the *gastric, splenic,* and *hepatic arteries;* the **superior** (supplies small intestine and superior portion of large intestine) and **inferior** (supplies inferior portion of large intestine) **mesenteric** (mes´´en-ter´ ik) **arteries;** and the **suprarenal** (soo´´prah-re´nal) **arteries, renal** (re´nal) **arteries,** and **gonadal** (go´nad-al) **arteries,** which supply blood to the adrenal glands, kidneys, and ovaries or testes, respectively. The abdominal aorta ends near the brim of the pelvis, where it divides into right and left **common iliac** (il´e-ak) **arteries.** These vessels supply blood to lower regions of the abdominal wall, the pelvic organs, and the lower extremities. Table 13.3 summarizes the main branches of the aorta.

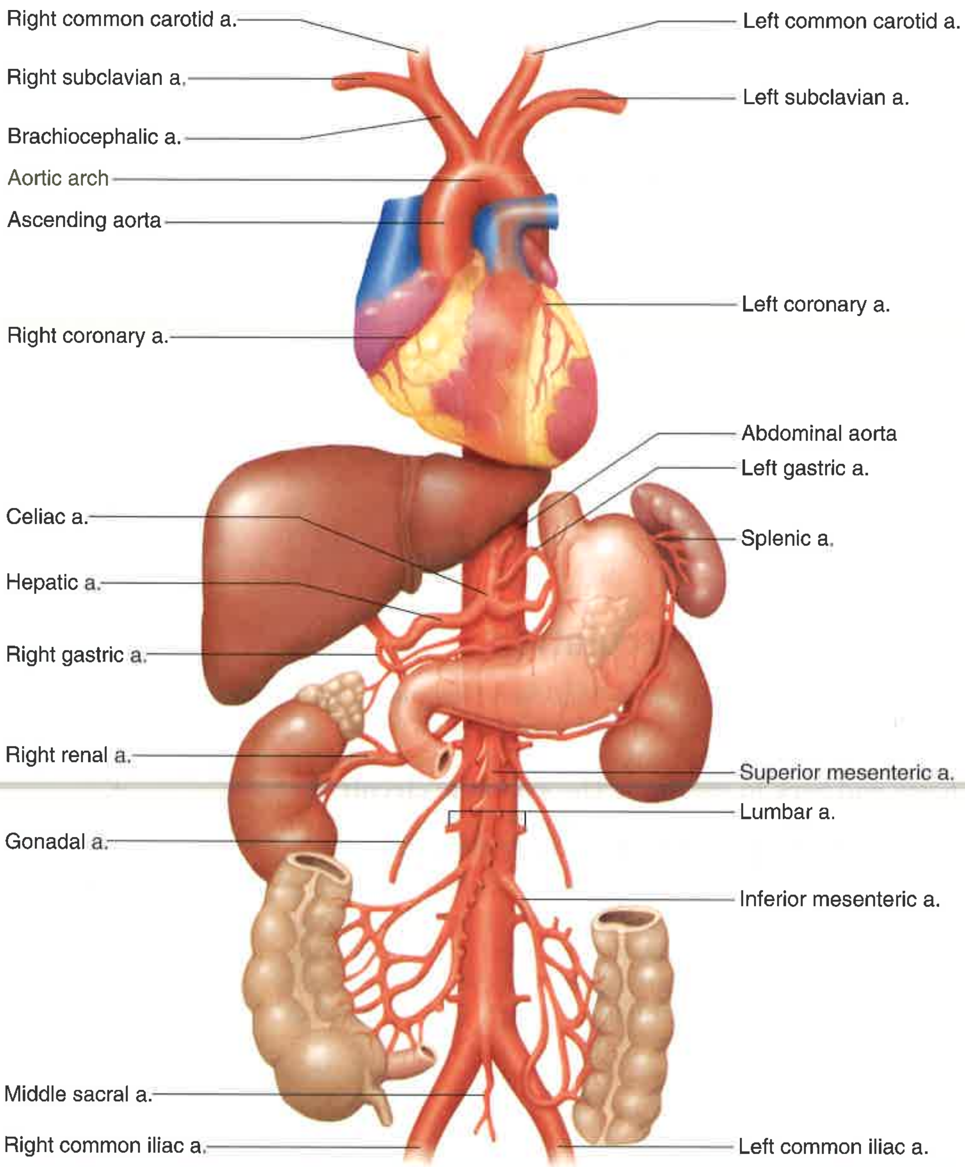


Figure 13.28
Principal branches of the aorta. (*a.* stands for *artery.*)

TABLE 13.3 PRINCIPAL BRANCHES OF THE AORTA

PORTION OF AORTA	MAJOR BRANCH	GENERAL REGIONS OR ORGANS SUPPLIED
Ascending aorta	Right and left coronary arteries	Heart
Arch of the aorta	Brachiocephalic artery	Right upper limb, right side of head
	Left common carotid artery	Left side of head
	Left subclavian artery	Left upper limb
Descending aorta	Bronchial artery	Bronchi
Thoracic aorta	Pericardial artery	Pericardium
	Esophageal artery	Esophagus
	Mediastinal artery	Mediastinum
	Posterior intercostal artery	Thoracic wall
Abdominal aorta	Celiac artery	Organs of upper digestive tract
	Phrenic artery	Diaphragm
	Superior mesenteric artery	Portions of small and large intestines
	Suprarenal artery	Adrenal gland
	Renal artery	Kidney
	Gonadal artery	Ovary or testis
	Inferior mesenteric artery	Lower portions of large intestine
	Lumbar artery	Posterior abdominal wall
	Middle sacral artery	Sacrum and coccyx
	Common iliac artery	Lower abdominal wall, pelvic organs, and lower limb

Arteries to the Neck, Head, and Brain

Branches of the subclavian and common carotid arteries supply blood to structures within the neck, head, and brain (fig. 13.29). The main divisions of the subclavian artery to these regions include the vertebral and thyrocervical arteries. The common carotid artery communicates with these regions by means of the internal and external carotid arteries.

The **vertebral arteries** pass upward through the foramina of the transverse processes of the cervical vertebrae and enter the skull through the foramen magnum. These vessels supply blood to the vertebrae and to their associated ligaments and muscles.

Within the cranial cavity, the vertebral arteries unite to form a single *basilar artery.* This vessel passes along the ventral brain stem and gives rise to branches leading to the pons, midbrain, and cerebellum. The basilar artery ends by dividing into two *posterior cerebral arteries* that supply portions of the occipital and temporal lobes of the cerebrum. The posterior cerebral arteries also help form the **cerebral arterial circle** (circle of Willis) at the base of the brain, which connects the vertebral artery and internal carotid artery systems (fig. 13.30). The union of these systems provides alternate pathways through which blood can reach brain tissues in the event of an arterial occlusion. It also equalizes blood pressure in the brain's blood supply.

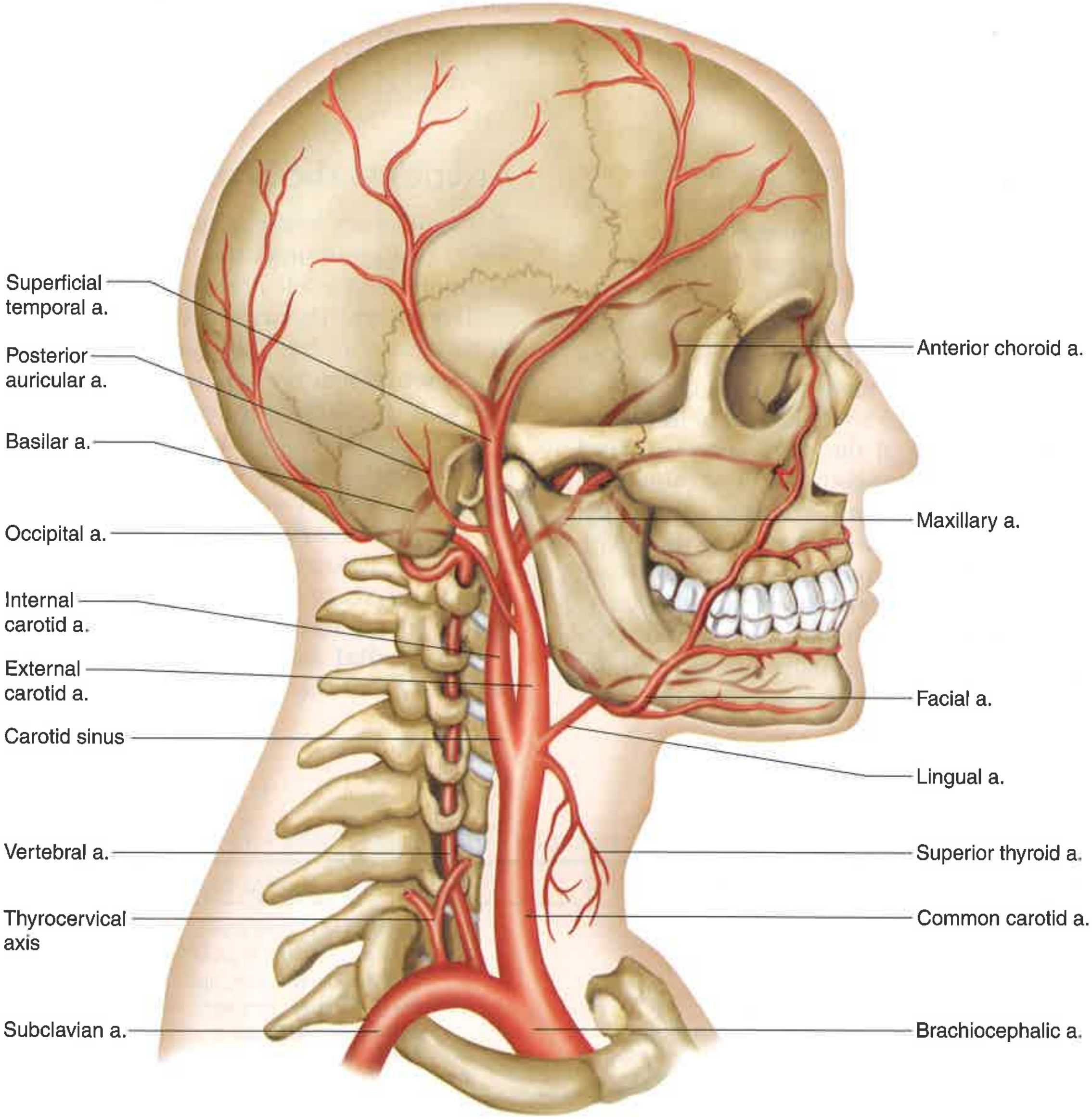


Figure 13.29
The main arteries of the head and neck. Note that the clavicle has been removed. (*a.* stands for *artery.*)

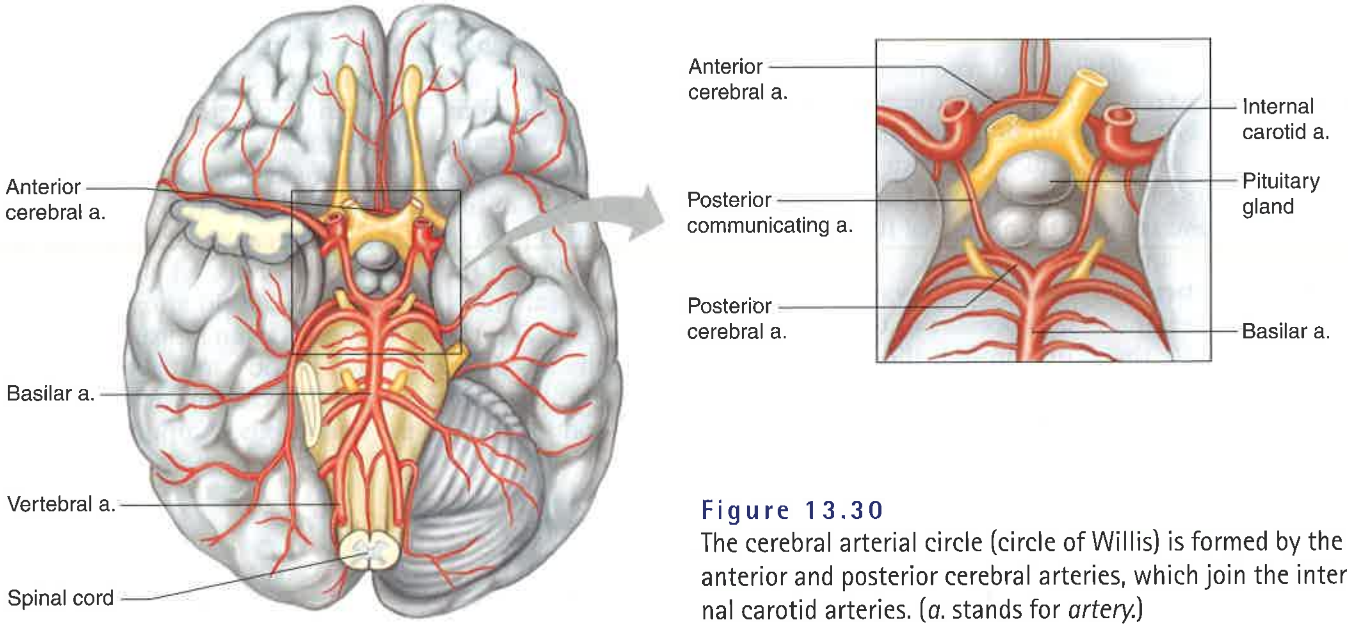


Figure 13.30
The cerebral arterial circle (circle of Willis) is formed by the anterior and posterior cerebral arteries, which join the internal carotid arteries. (*a.* stands for *artery.*)

The **thyrocervical** (thi´´ro-ser´vĭ-kal) **arteries** are short vessels. At the thyrocervical axis, these vessels give off branches to the thyroid gland, parathyroid glands, larynx, trachea, esophagus, and pharynx, as well as to muscles in the neck, shoulder, and back.

The left and right *common carotid arteries* diverge into the internal and external carotid arteries. The **external carotid artery** courses upward on the side of the head, giving off branches to structures in the neck, face, jaw, scalp, and base of the skull. The **internal carotid artery** follows a deep course upward along the pharynx to the base of the skull. Entering the cranial cavity, it provides the major blood supply to the brain. Near the base of the internal carotid arteries are enlargements called **carotid sinuses** that, like aortic sinuses, contain baroreceptors controlling blood pressure. Table 13.4 summarizes the major branches of the external and internal carotid arteries.

Arteries to the Shoulder and Upper Limb

The subclavian artery, after giving off branches to the neck, continues into the arm (fig. 13.31). It passes between the clavicle and the first rib, and becomes the axillary artery. The **axillary artery** supplies branches to structures in the axilla and chest wall and becomes the **brachial artery,** which follows the humerus to the elbow. It gives rise to a *deep brachial artery* that curves posteriorly around the humerus and supplies the triceps brachii. Within the elbow, the brachial artery divides into an ulnar artery and a radial artery.

The **ulnar artery** leads downward on the ulnar side of the forearm to the wrist. Some of its branches supply the elbow joint, whereas others supply blood to muscles in the forearm.

The **radial artery** travels along the radial side of the forearm to the wrist, supplying the lateral muscles

TABLE 13.4 MAJOR BRANCHES OF THE EXTERNAL AND INTERNAL CAROTID ARTERIES

ARTERY	MAJOR BRANCH	GENERAL REGION OR ORGANS SUPPLIED
External carotid artery	Superior thyroid artery	Larynx and thyroid gland
	Lingual artery	Tongue and salivary glands
	Facial artery	Pharynx, palate, chin, lips, and nose
	Occipital artery	Posterior scalp, meninges, and neck muscles
	Posterior auricular artery	Ear and lateral scalp
	Maxillary artery	Teeth, jaw, cheek, and eyelids
	Superficial temporal artery	Parotid salivary gland and surface of the face and scalp
Internal carotid artery	Ophthalmic artery	Eye and eye muscles
	Anterior choroid artery	Choroid plexus and brain
	Anterior cerebral artery	Frontal and parietal lobes of the brain

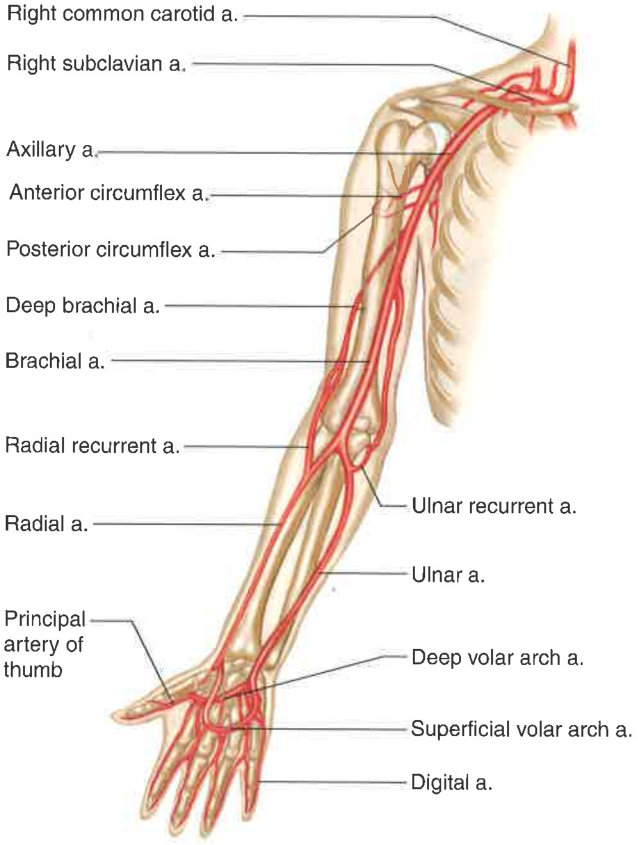


Figure 13.31
The main arteries to the shoulder and upper limb. (*a.* stands for *artery.*)

of the forearm. As the radial artery nears the wrist, it approaches the surface and provides a convenient vessel for taking the pulse (radial pulse).

At the wrist, the branches of the ulnar and radial arteries join to form a network of vessels. Arteries arising from this network supply blood to the wrist, hand, and fingers.

Arteries to the Thoracic and Abdominal Walls

Blood reaches the thoracic wall through several vessels. The **internal thoracic artery,** a branch of the subclavian artery, gives off two *anterior intercostal* (in″ter-kos′tal) *arteries* that supply the intercostal muscles and mammary glands. The *posterior intercostal arteries* arise from the thoracic aorta and enter the intercostal spaces. They supply the intercostal muscles, the vertebrae, the spinal cord, and the deep muscles of the back.

Branches of the *internal thoracic* and *external iliac arteries* provide blood to the anterior abdominal wall. Paired vessels originating from the abdominal aorta, including the *phrenic* and *lumbar arteries,* supply blood to structures in the posterior and lateral abdominal wall.

Arteries to the Pelvis and Lower Limb

The abdominal aorta divides to form the **common iliac** (il′e-ak) **arteries** at the level of the pelvic brim, and these vessels provide blood to the pelvic organs, gluteal region, and lower limbs (fig. 13.32). Each common iliac artery divides into an internal and an external branch. The **internal iliac artery** gives off many branches to pelvic muscles and visceral structures, as well as to the gluteal muscles and the external reproductive organs. The **external iliac artery** provides the main blood supply to the lower limbs. It passes downward along the brim of the pelvis and branches to supply the muscles and skin in the lower abdominal wall. Midway between the symphysis pubis and the anterior superior iliac spine of the ilium, the external iliac artery becomes the femoral artery.

The **femoral** (fem′or-al) **artery,** which approaches the anterior surface of the upper thigh, branches to muscles and superficial tissues of the thigh. These branches also supply the skin of the groin and the lower abdominal wall.

As the femoral artery reaches the proximal border of the space behind the knee, it becomes the **popliteal** (pop-lit′e-al) **artery.** Branches of this artery supply blood to the knee joint and to certain muscles in the thigh and calf. The popliteal artery diverges into the anterior and posterior tibial arteries.

The **anterior tibial artery** passes downward between the tibia and fibula, giving off branches to the skin and muscles in anterior and lateral regions of the leg. This vessel continues into the foot as the *dorsalis pedis artery* (dorsal pedis artery), which supplies blood to the foot and toes. The **posterior tibial artery,** the larger of the two popliteal branches, descends beneath the calf muscles, and branches to the skin, muscles, and other tissues of the leg along the way.

CHECK YOUR RECALL

1. Name the portions of the aorta.
2. Name the vessels that arise from the aortic arch.
3. Name the branches of the thoracic and abdominal aorta.
4. Which vessels supply blood to the head? To the upper limb? To the abdominal wall? To the lower limb?

13.8 Venous System

Venous circulation returns blood to the heart after blood and body cells exchange gases, nutrients, and wastes.

Characteristics of Venous Pathways

Venous vessels begin as capillaries merge into venules, venules merge into small veins, and small veins meet to form larger ones. Unlike the arterial pathways, however,

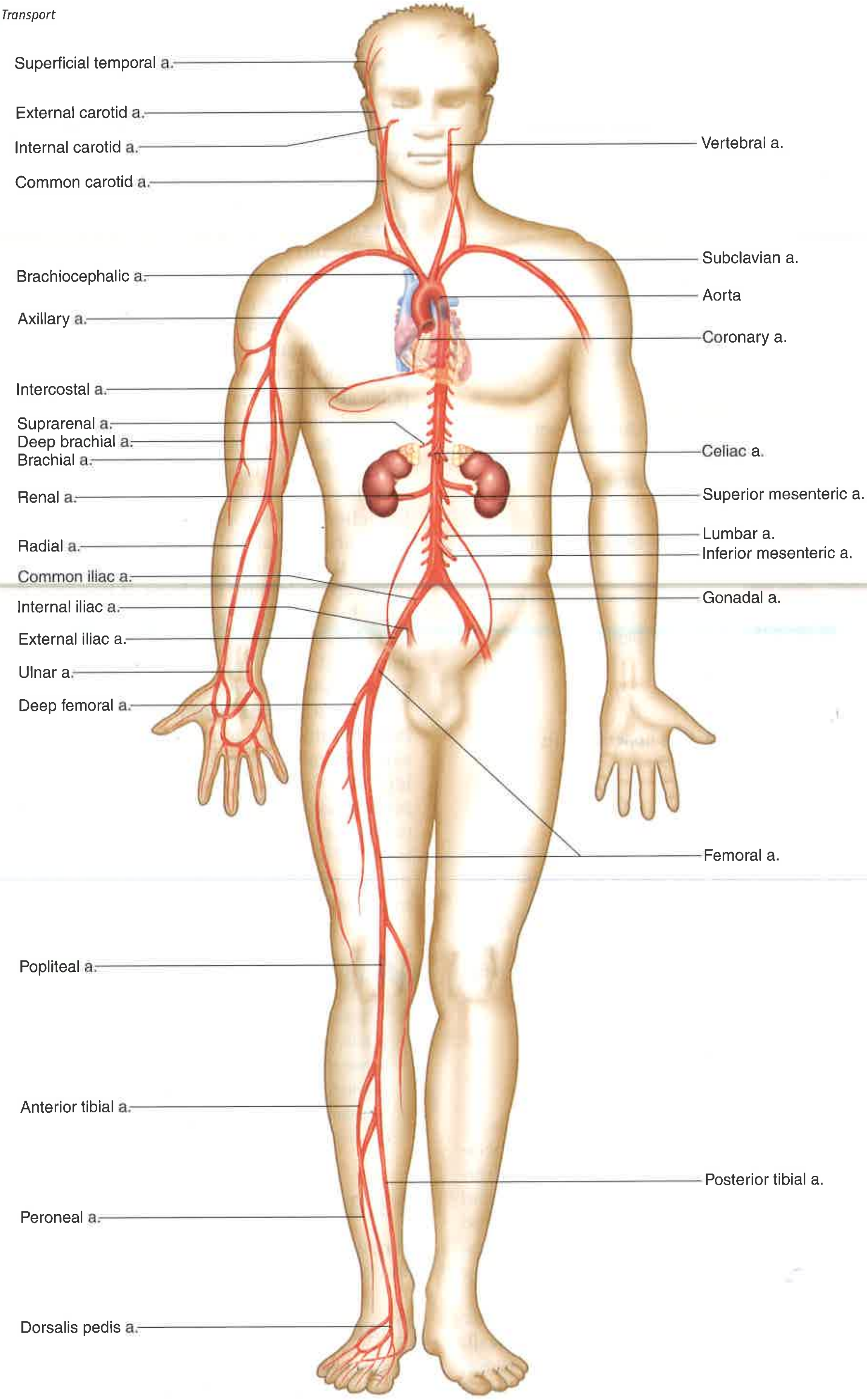


Figure 13.32
Major vessels of the arterial system. (*a.* stands for *artery.*)

the vessels of the venous system are difficult to follow. This is because they connect in irregular networks, so many unnamed tributaries may join to form a large vein.

On the other hand, the larger veins typically parallel the courses of named arteries, and these veins often have the same names as their arterial counterparts. For example, the renal vein parallels the renal artery, and the common iliac vein accompanies the common iliac artery.

The veins that carry blood from the lungs and myocardium back to the heart have already been described. The veins from all the other parts of the body converge into two major pathways, the superior and inferior venae cavae, which lead to the right atrium.

Veins from the Brain, Head, and Neck

The **external jugular** (jug´u-lar) **veins** drain blood from the face, scalp, and superficial regions of the neck. These vessels descend on either side of the neck and empty into the *right* and *left subclavian veins* (fig. 13.33).

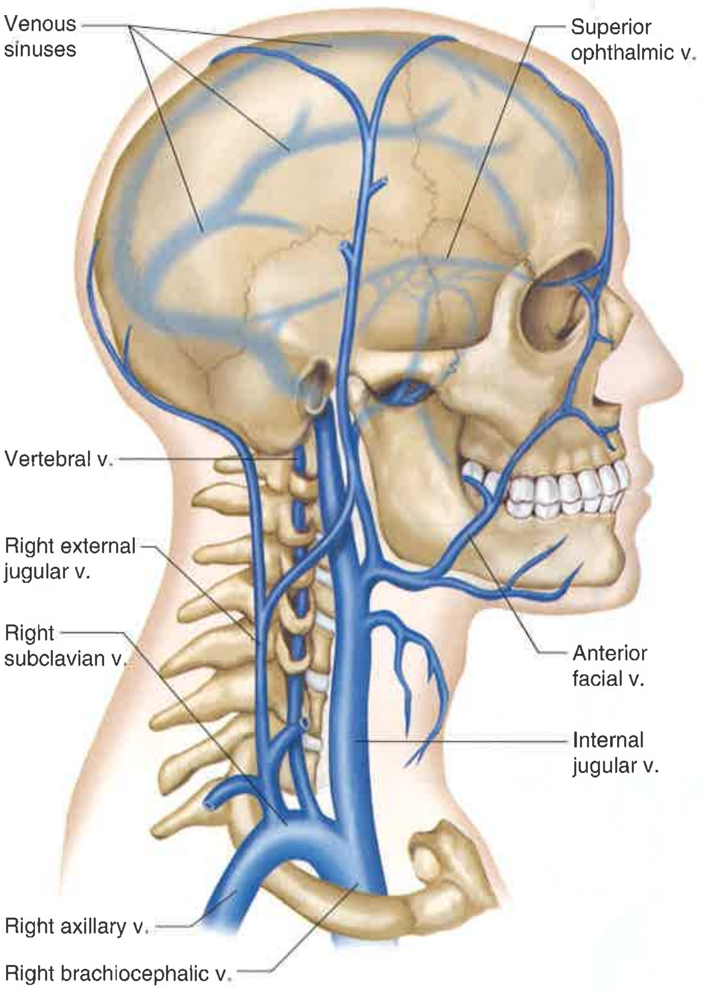


Figure 13.33
The major veins of the brain, head, and neck. Note that the clavicle has been removed. (*v.* stands for *vein.*)

The **internal jugular veins,** which are somewhat larger than the external jugular veins, arise from numerous veins and venous sinuses of the brain and from deep veins in parts of the face and neck. They descend through the neck and join the subclavian veins. These unions of the internal jugular and subclavian veins form large **brachiocephalic veins** on each side. The vessels then merge and give rise to the superior vena cava, which enters the right atrium.

Veins from the Upper Limb and Shoulder

A set of deep veins and a set of superficial ones drain the upper limb. The deep veins generally parallel the arteries in each region and have similar names, such as the *radial vein, ulnar vein, brachial vein,* and *axillary vein.* The superficial veins connect in complex networks just beneath the skin. They also communicate with the deep vessels of the upper limb, providing many alternate pathways through which blood can leave the tissues (fig. 13.34). The main vessels of the superficial network are the basilic and cephalic veins.

The **basilic** (bah-sil´ik) **vein** ascends from the forearm to the middle of the arm, where it penetrates

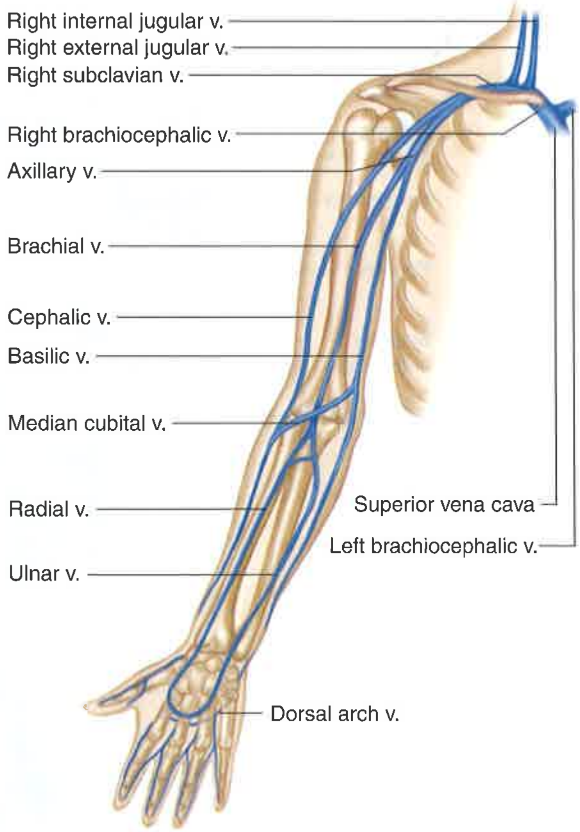


Figure 13.34
The main veins of the upper limb and shoulder. (*v.* stands for *vein.*)

deeply and joins the *brachial vein*. The basilic and brachial veins merge, forming the *axillary vein*.

The **cephalic** (sĕ-fal´ik) **vein** courses upward from the hand to the shoulder. In the shoulder, it pierces the tissues and empties into the axillary vein. Beyond the axilla, the axillary vein becomes the subclavian vein.

In the bend of the elbow, a *median cubital vein* ascends from the cephalic vein on the lateral side of the forearm to the basilic vein on the medial side. This large vein is usually visible. It is often used as a site for *venipuncture*, when it is necessary to remove a blood sample for examination or to add fluids to blood.

Veins from the Abdominal and Thoracic Walls

Tributaries of the brachiocephalic and azygos veins drain the abdominal and thoracic walls. For example, the *brachiocephalic vein* receives blood from the *internal thoracic vein*, which generally drains the tissues the internal thoracic artery supplies. Some *intercostal veins* also empty into the brachiocephalic vein.

The **azygos** (az´ĭ-gos) **vein** originates in the dorsal abdominal wall and ascends through the mediastinum on the right side of the vertebral column to join the superior vena cava. It drains most of the muscular tissue in the abdominal and thoracic walls.

Tributaries of the azygos vein include the *posterior intercostal veins* on the right side, which drain the intercostal spaces, and the *superior* and *inferior hemiazygos veins*, which receive blood from the posterior intercostal veins on the left. The right and left *ascending lumbar veins*, with tributaries that include vessels from the lumbar and sacral regions, also connect to the azygos system.

Veins from the Abdominal Viscera

Veins usually carry the blood directly to the atria of the heart. However, those that drain the abdominal viscera are exceptions (fig. 13.35). They originate in the capillary networks of the stomach, intestines, pancreas, and spleen and carry blood from these organs through a

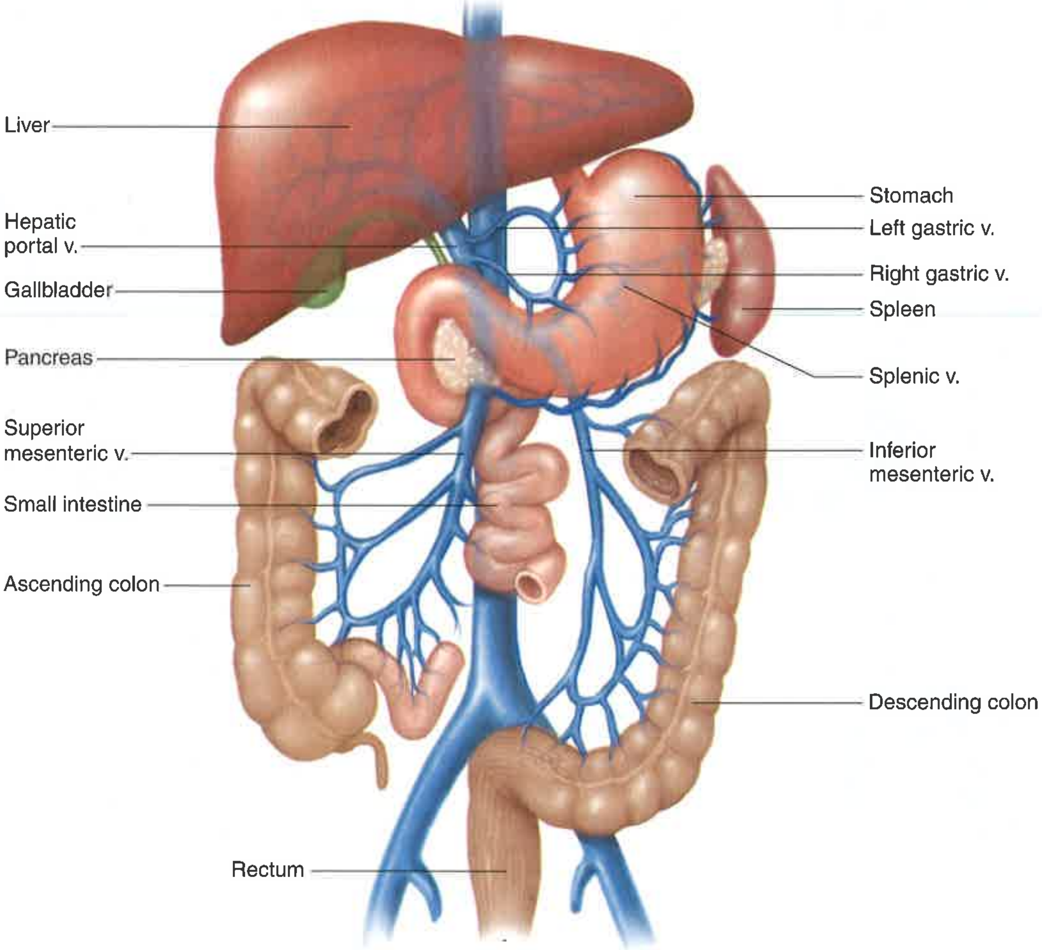


Figure 13.35
Veins that drain the abdominal viscera. (*v.* stands for *vein*.)

hepatic portal (por´tal) **vein** to the liver. This unique venous pathway is called the **hepatic portal system.**

Tributaries of the hepatic portal vein include:

1. Right and left *gastric veins* from the stomach.
2. *Superior mesenteric vein* from the small intestine, ascending colon, and transverse colon.
3. *Splenic vein* from a convergence of several veins draining the spleen, the pancreas, and a portion of the stomach. Its largest tributary, the *inferior mesenteric vein,* brings blood upward from the descending colon, sigmoid colon, and rectum.

About 80% of the blood flowing to the liver in the hepatic portal system comes from capillaries in the stomach and intestines, and is oxygen-poor but rich in nutrients. As discussed in chapter 15 (p. 408), the liver handles these nutrients in a variety of ways. It regulates blood glucose concentration by polymerizing excess glucose into glycogen for storage or by breaking down glycogen into glucose when blood glucose concentration drops below normal. The liver helps regulate blood concentrations of recently absorbed amino acids and lipids by modifying their molecules into forms cells can use, by oxidizing them, or by changing them into storage forms. The liver also stores certain vitamins and detoxifies harmful substances. Blood in the hepatic portal vein nearly always contains bacteria that have entered through intestinal capillaries. Large *Kupffer cells* lining small vessels in the liver called hepatic sinusoids phagocytize these microorganisms, removing them from portal blood before it leaves the liver.

After passing through the hepatic sinusoids of the liver, blood in the hepatic portal system travels through a series of merging vessels into **hepatic veins.** These veins empty into the inferior vena cava, returning the blood to the general circulation.

Veins from the Lower Limb and Pelvis

As in the upper limb, veins that drain blood from the lower limb are divided into deep and superficial groups (fig. 13.36). The deep veins of the leg, such as the *anterior* and *posterior tibial veins,* are named for the arteries they accompany. At the level of the knee, these vessels form a single trunk, the **popliteal vein.** This vein continues upward through the thigh as the **femoral vein,** which in turn becomes the **external iliac vein.**

The superficial veins of the foot, leg, and thigh connect to form a complex network beneath the skin. These vessels drain into two major trunks—the small and great saphenous veins. The **small saphenous** (sah-fe´nus) **vein** ascends along the back of the calf, enters the popliteal fossa, and joins the popliteal vein. The **great saphenous vein,** which is the longest vein in the body, ascends in front of the medial malleolus and extends upward along the medial side of the leg and thigh. In the thigh, it penetrates deeply and joins the femoral vein. Near its termination, the great saphenous vein receives tributaries from a number of vessels that drain the upper thigh, groin, and lower abdominal wall.

In addition to communicating freely with each other, the saphenous veins communicate extensively with the deep veins of the leg and thigh. Blood can thus return to the heart from the lower extremities by several routes.

In the pelvic region, vessels leading to the **internal iliac vein** carry blood away from the organs of the reproductive, urinary, and digestive systems. The internal iliac veins unite with the right and left external iliac veins to form the **common iliac veins.** These vessels, in turn, merge to produce the inferior vena cava.

V*aricose veins* have abnormal dilations. They result from increased blood pressure in the saphenous veins due to gravity, as occurs when a person stands for a prolonged period.

CHECK YOUR RECALL

1. Name the veins that return blood to the right atrium.
2. Which major veins drain blood from the head? From the upper limb? From the abdominal viscera? From the lower limb?

Clinical Terms Related to the Cardiovascular System

anastomosis (ah-nas´´to-mo´sis) Connection between two blood vessels, sometimes produced surgically.

angiospasm (an´je-o-spazm´´) Muscular spasm in the wall of a blood vessel.

arteriography (ar´´te-re-og´rah-fe) Injection of radiopaque solution into the vascular system for X-ray examination of arteries.

asystole (a-sis´to-le) Condition in which the myocardium fails to contract.

cardiac tamponade (kar´de-ak tam´´po-nād´) Compression of the heart by fluid accumulating within the pericardial cavity.

congestive heart failure (kon-jes´tiv hart fāl´yer) Inability of the left ventricle to pump adequate blood to cells.

cor pulmonale (kor pul-mo-na´le) Heart-lung disorder characterized by pulmonary hypertension and hypertrophy of the right ventricle.

embolectomy (em´´bo-lek´to-me) Removal of an embolus through an incision in a blood vessel.

endarterectomy (en´´dar-ter-ek´to-me) Removal of the inner wall of an artery to reduce an arterial occlusion.

palpitation (pal´´pĭ-ta´shun) Awareness of a heartbeat that is unusually rapid, strong, or irregular.

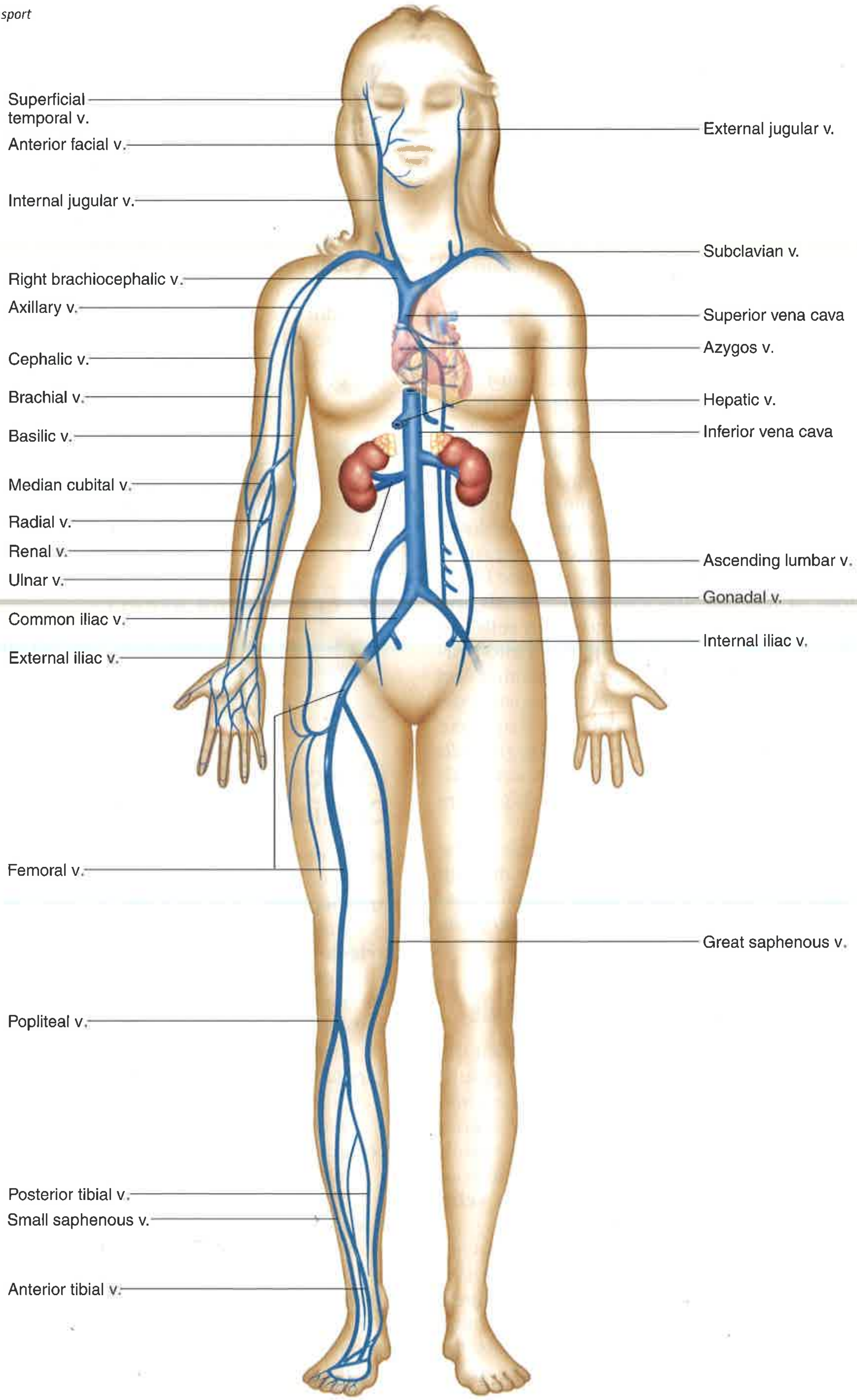
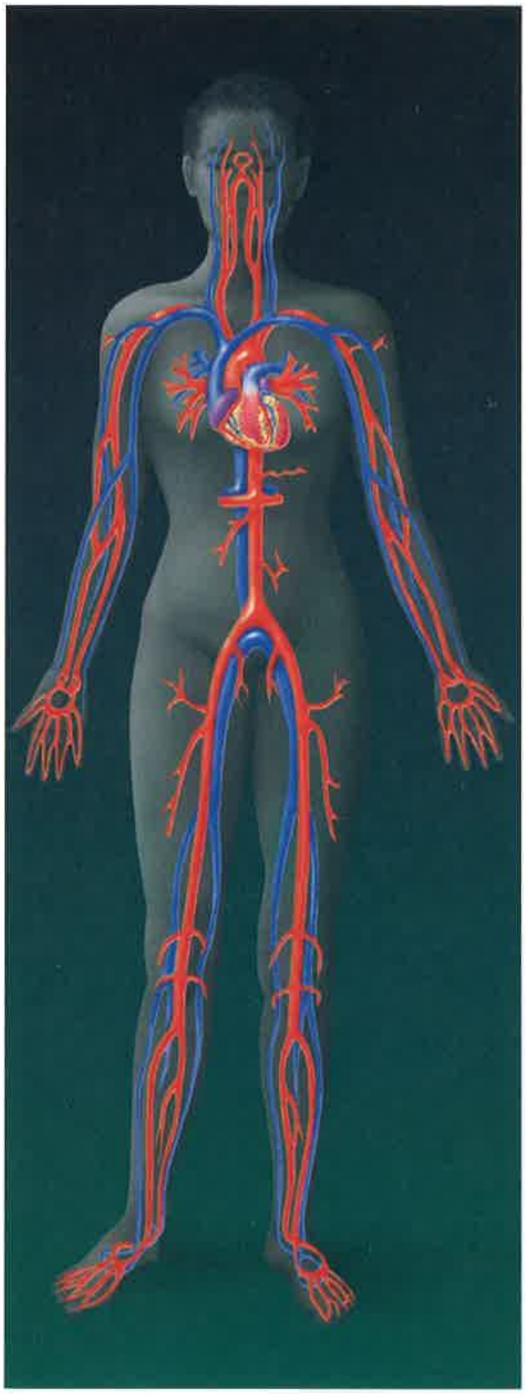


Figure 13.36
Major vessels of the venous system. (*v.* stands for *vein.*)

Organization



Cardiovascular System

The heart pumps blood through as many as 60,000 miles of blood vessels delivering nutrients to, and removing wastes from, all body cells.

Integumentary System

Changes in skin blood flow are important in temperature control.

Skeletal System

Bones help control plasma calcium levels.

Muscular System

Blood flow increases to exercising skeletal muscle, delivering oxygen and nutrients and removing wastes. Muscle actions help the blood circulate.

Nervous System

The brain depends on blood flow for survival. The nervous system helps control blood flow and blood pressure.

Endocrine System

Hormones are carried in the bloodstream. Some hormones directly affect the heart and blood vessels.

Lymphatic System

The lymphatic system returns tissue fluids to the bloodstream.

Digestive System

The digestive system breaks down nutrients into forms readily absorbed by the bloodstream.

Respiratory System

The respiratory system oxygenates the blood and removes carbon dioxide. Respiratory movements help the blood circulate.

Urinary System

The kidneys clear the blood of wastes and substances present in the body. The kidneys help control blood pressure and blood volume.

Reproductive System

Blood pressure is important in normal function of the sex organs.

pericardiectomy (per´´ĭ-kar´´de-ek´to-me) Excision of the pericardium.

phlebitis (flĕ-bi´tis) Inflammation of a vein, usually in the lower limbs.

phlebotomy (flĕ-bot´o-me) Incision or puncture of a vein to withdraw blood.

sinus rhythm (si´nus rithm) The normal cardiac rhythm regulated by the S-A node.

thrombophlebitis (throm´´bo-flĕ-bi´tis) Formation of a blood clot in a vein in response to inflammation of the venous wall.

valvotomy (val-vot´o-me) Incision of a valve.

venography (ve-nog´rah-fe) Injection of radiopaque solution into the vascular system for X-ray examination of veins.

Clinical Connection

On July 2, 2001, 50 year old Bob Tools became the first person to receive an implantable artificial heart, developed at the University of Louisville. Weeks from death due to congestive heart failure at the time of the seven hour surgery, Tools enjoyed five more months of life, thanks to the device that replaced the functioning of his ventricles. The two-pound, titanium and plastic cardiac stand-in consists of an internal motor-driven hydraulic pump, battery and electronic package, and an external battery pack. The electronics component manages the rate and force of the pump's actions, tailoring them to the patient's condition. Several other patients have survived an average of two months with the implantable artificial heart.

Farther in the future, treatment for heart failure may consist of implants of stem cells that divide to produce new cardiac tissue. Evidence that this happens naturally comes from heart transplants from women to men. In one study, at varying times after the transplant, and after the recipients had died (of a variety of causes), researchers detected cells in the donor hearts that had the telltale Y chromosome of males. This meant that the recipient's cells had migrated to and mingled with donor heart cells. Some of the cells had the markings of stem cells, and some had already specialized into connective tissue, cardiac muscle tissue, and epithelium—precisely what was required to accept the new part. These experiments showed that a recipient's cells infiltrate a transplanted organ and eventually provide new specialized cells. The stem cells may come from the bit of recipient tissue to which the new organ is stitched, or migrate from the bone marrow and then differentiate into exactly what is needed to heal.

SUMMARY OUTLINE

13.1 Introduction (p. 329)

The cardiovascular system provides oxygen and nutrients to tissues and removes wastes.

13.2 Structure of the Heart (p. 329)

1. Size and location of the heart
   a. The heart is about 14 centimeters long and 9 centimeters wide.
   b. It is located within the mediastinum and rests on the diaphragm.
2. Coverings of the heart
   a. A layered pericardium encloses the heart.
   b. The pericardial cavity is a space between the parietal and visceral layers of the pericardium.
3. Wall of the heart
   The wall of the heart has three layers—an epicardium, a myocardium, and an endocardium.
4. Heart chambers and valves
   a. The heart is divided into two atria and two ventricles.
   b. Right chambers and valves
      (1) The right atrium receives blood from the venae cavae and coronary sinus.
      (2) The tricuspid valve separates the right atrium from the right ventricle.
      (3) A pulmonary valve guards the base of the pulmonary trunk.
   c. Left chambers and valves
      (1) The left atrium receives blood from the pulmonary veins.
      (2) The bicuspid valve separates the left atrium from the left ventricle.
      (3) An aortic valve guards the base of the aorta.
5. Skeleton of the heart
   The skeleton of the heart consists of fibrous rings that enclose the bases of the pulmonary artery and aorta.
6. Path of blood through the heart
   a. Blood low in oxygen and high in carbon dioxide enters the right side of the heart and is pumped into the pulmonary circulation.
   b. After blood is oxygenated in the lungs and some carbon dioxide is removed, it returns to the left side of the heart.
7. Blood supply to the heart
   a. The coronary arteries supply blood to the myocardium.
   b. Blood returns to the right atrium through the cardiac veins and coronary sinus.

13.3 Heart Actions (p. 336)

1. Cardiac cycle
   a. The atria contract while the ventricles relax. The ventricles contract while the atria relax.
   b. Pressure within the chambers rises and falls in repeated cycles.
2. Heart sounds
   Heart sounds are due to the vibrations the valve movements produce.
3. Cardiac muscle fibers
   a. Cardiac muscle fibers connect to form a functional syncytium.
   b. If any part of the syncytium is stimulated, the whole structure contracts as a unit.
4. Cardiac conduction system
   a. This system initiates and conducts impulses throughout the myocardium.
   b. Impulses from the S-A node pass slowly to the A-V node. Impulses travel rapidly along the A-V bundle and Purkinje fibers.

5. Electrocardiogram (ECG)
    a. An ECG records electrical changes in the myocardium during a cardiac cycle.
    b. The pattern contains several waves.
        (1) The P wave represents atrial depolarization.
        (2) The QRS complex represents ventricular depolarization.
        (3) The T wave represents ventricular repolarization.
6. Regulation of the cardiac cycle
    a. Physical exercise, body temperature, and the concentration of various ions affect heartbeat.
    b. Branches of sympathetic and parasympathetic nerve fibers innervate the S-A and A-V nodes.
    c. The cardiac center in the medulla oblongata regulates autonomic impulses to the heart.

13.4 Blood Vessels (p. 341)

Blood vessels form a closed circuit of tubes that carry blood from the heart to body cells and back again.

1. Arteries and arterioles
    a. Arteries are adapted to carry blood under high pressure away from the heart.
    b. The walls of arteries and arterioles consist of layers of endothelium, smooth muscle, and connective tissue.
    c. Autonomic fibers that can stimulate vasoconstriction or vasodilation innervate smooth muscle in vessel walls.
2. Capillaries
    a. Capillaries connect arterioles and venules.
    b. The capillary wall is a single layer of cells that forms a semipermeable membrane.
    c. Openings in capillary walls, where endothelial cells overlap, vary in size from tissue to tissue.
    d. Precapillary sphincters regulate capillary blood flow.
3. Exchanges in capillaries
    a. Capillary blood and tissue fluid exchange gases, nutrients, and metabolic by-products.
    b. Diffusion provides the most important means of transport.
    c. Filtration, which is due to the hydrostatic pressure of blood, causes a net outward movement of fluid at the arteriolar end of a capillary.
    d. Osmosis due to colloid osmotic pressure causes a net inward movement of fluid at the venular end of a capillary.
4. Venules and veins
    a. Venules continue from capillaries and merge to form veins.
    b. Veins carry blood to the heart.
    c. Venous walls are similar to arterial walls, but are thinner and contain less smooth muscle and elastic tissue.

13.5 Blood Pressure (p. 346)

Blood pressure is the force blood exerts against the insides of blood vessels.

1. Arterial blood pressure
    a. Arterial blood pressure rises and falls with the phases of the cardiac cycle.
    b. Systolic pressure is produced when the ventricle contracts. Diastolic pressure is the pressure in the arteries when the ventricle relaxes.
2. Factors that influence arterial blood pressure
    Arterial blood pressure increases as cardiac output, blood volume, peripheral resistance, or blood viscosity increases.
3. Control of blood pressure
    a. Blood pressure is controlled in part by the mechanisms that regulate cardiac output and peripheral resistance.
    b. The more blood that enters the heart, the stronger the ventricular contraction, the greater the stroke volume, and the greater the cardiac output.
    c. The cardiac center of the medulla oblongata regulates heart rate.
4. Venous blood flow
    a. Venous blood flow depends on skeletal muscle contraction, breathing movements, and venoconstriction.
    b. Many veins contain flaplike valves that prevent blood from backing up.
    c. Venoconstriction can increase venous pressure and blood flow.

13.6 Paths of Circulation (p. 351)

1. Pulmonary circuit
    The pulmonary circuit consists of vessels that carry blood from the right ventricle to the lungs and back to the left atrium.
2. Systemic circuit
    a. The systemic circuit consists of vessels that lead from the heart to the body cells (including those of the heart itself) and back to the heart.
    b. It includes the aorta and its branches.

13.7 Arterial System (p. 351)

1. Principal branches of the aorta
    a. The aorta is the largest artery with respect to diameter.
    b. Its major branches include the coronary, brachiocephalic, left common carotid, and left subclavian arteries.
    c. The branches of the descending aorta include the thoracic and abdominal groups.
    d. The abdominal aorta diverges into the right and left common iliac arteries.
2. Arteries to the neck, head, and brain
    These include branches of the subclavian and common carotid arteries.
3. Arteries to the shoulder and upper limb
    a. The subclavian artery passes into the upper limb, and in various regions is called the axillary and brachial artery.
    b. Branches of the brachial artery include the ulnar and radial arteries.
4. Arteries to the thoracic and abdominal walls
    a. Branches of the subclavian artery and thoracic aorta supply the thoracic wall.
    b. Branches of the abdominal aorta and other arteries supply the abdominal wall.
5. Arteries to the pelvis and lower limb
    The common iliac arteries supply the pelvic organs, gluteal region, and lower limbs.

13.8 Venous System (p. 355)

1. Characteristics of venous pathways
    a. Veins return blood to the heart.
    b. Larger veins usually parallel the paths of major arteries.
2. Veins from the brain, head, and neck
    a. Jugular veins drain these regions.
    b. Jugular veins unite with subclavian veins to form the brachiocephalic veins.
3. Veins from the upper limb and shoulder
    a. Sets of superficial and deep veins drain these regions.
    b. Deep veins parallel arteries with similar names.
4. Veins from the abdominal and thoracic walls
    Tributaries of the brachiocephalic and azygos veins drain these walls.
5. Veins from the abdominal viscera
    a. Blood from the abdominal viscera enters the hepatic portal system and is carried to the liver.
    b. From the liver, hepatic veins carry blood to the inferior vena cava.
6. Veins from the lower limb and pelvis
    a. Sets of deep and superficial veins drain these regions.
    b. The deep veins include the tibial veins, and the superficial veins include the saphenous veins.

REVIEW EXERCISES

1. Describe the general structure, function, and location of the heart. (p. 329)
2. Describe the pericardium. (p. 329)
3. Compare the layers of the cardiac wall. (p. 330)
4. Identify and describe the locations of the chambers and the valves of the heart. (p. 331)
5. Describe the skeleton of the heart, and explain its function. (p. 333)
6. Trace the path of blood through the heart. (p. 333)
7. Trace the path of blood through the coronary circulation. (p. 333)
8. Describe a cardiac cycle. (p. 336)
9. Describe the pressure changes in the atria and ventricles during a cardiac cycle. (p. 336)
10. Explain the origin of heart sounds. (p. 337)
11. Distinguish between the roles of the S-A node and the A-V node. (p. 338)
12. Explain how the cardiac conduction system controls the cardiac cycle. (p. 338)
13. Describe and explain the normal ECG pattern. (p. 339)
14. Discuss how the nervous system regulates the cardiac cycle. (p. 340)
15. Distinguish between an artery and an arteriole. (p. 342)
16. Explain control of vasoconstriction and vasodilation. (p. 342)
17. Describe the structure and function of a capillary. (p. 343)
18. Explain control of blood flow through a capillary. (p. 343)
19. Explain how diffusion functions in the exchange of substances between blood plasma and tissue fluid. (p. 345)
20. Explain why water and dissolved substances leave the arteriolar end of a capillary and enter the venular end. (p. 345)
21. Distinguish between a venule and a vein. (p. 346)
22. Explain how veins function as blood reservoirs. (p. 346)
23. Distinguish between systolic and diastolic blood pressures. (p. 347)
24. Name several factors that influence blood pressure, and explain how each produces its effect. (p. 348)
25. Describe the control of blood pressure. (p. 348)
26. List the major factors that promote the flow of venous blood. (p. 350)
27. Distinguish between the pulmonary and systemic circuits of the cardiovascular system. (p. 351)
28. Trace the path of blood through the pulmonary circuit. (p. 351)
29. Describe the aorta, and name its principal branches. (p. 351)
30. Describe the relationship between the major venous pathways and the major arterial pathways. (p. 355)

CRITICAL THINKING

1. How might the results of a cardiovascular exam differ for an athlete in top condition and a sedentary, overweight individual?
2. If you were asked to invent a blood vessel substitute, what materials might you use to build it? Include synthetic as well as natural materials.
3. What structures and properties should an artificial heart have?
4. Cigarette smoke contains thousands of chemicals, including nicotine and carbon monoxide. Nicotine constricts blood vessels. Carbon monoxide prevents oxygen from binding to hemoglobin. How do these two components of smoke affect the cardiovascular system?
5. Given the way capillary blood flow is regulated, do you think it is wiser to rest or to exercise following a heavy meal? Explain.
6. If a patient develops a blood clot in the femoral vein of the left lower limb and a portion of the clot breaks loose, where is the blood flow likely to carry the embolus? What symptoms are likely?
7. Cirrhosis of the liver, a disease commonly associated with alcoholism, obstructs blood flow through hepatic blood vessels. As a result, blood backs up, and capillary pressure greatly increases in organs the hepatic portal system drains. What effects might this increasing capillary pressure produce, and which organs would it affect?
8. If a cardiologist inserts a catheter into a patient's right femoral artery, which arteries would the tube have to pass through in order to reach the entrance to the left coronary artery?

WEB CONNECTIONS

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