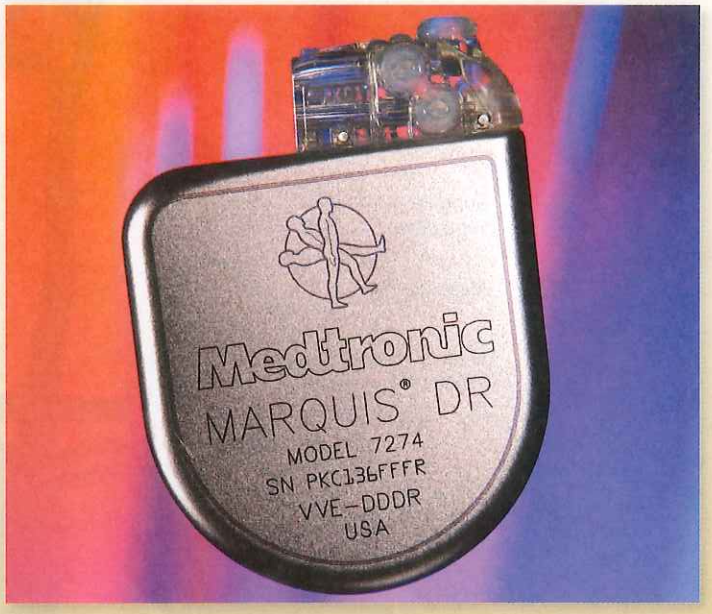
Defibrillators Save Lives

A man rushing to catch a flight at a busy airport stops suddenly, looks about in confusion, and collapses. People gather around him, as a woman runs to a device mounted on a nearby wall. It is an automated external defibrillator (AED) and looks like a laptop computer. The woman learned how to use it in a cardiopulmonary resuscitation class. She brings it over to the man, opens it, and places electrode pads over the man's chest, as indicated in a drawing on the inner cover of the defibrillator. Then the device speaks. "Analyzing heart rhythm," it declares as a computer assesses the heart rhythm. After a short pause, the device says, "charging, stand clear," and then "push button." The woman does so, and the device delivers a shock to the man's chest. It assesses the heart rhythm again, and instructs the woman to deliver a second shock. Soon, the man recovers, just as emergency technicians arrive.

An AED in a public place can save the life of a person suffering sudden cardiac arrest. One study of AED use at airports found that they saved 64% of the people on whom they were used. Without defibrillation, only 5% to 7% of people survive sudden cardiac arrest. Each minute, the odds of survival shrink by 10%, and after six minutes, brain damage is irreversible.

Sudden cardiac arrest can result from an abnormally accelerated heartbeat or a chaotic and irregular contraction of the heart muscle (ventricular fibrillation). The bioelectrical malfunction that usually causes these conditions may result from an artery blocked with plaque or from build up of scar tissue from a previous myocardial infarction (heart attack).

For people who know that they have an inherited disorder that causes sudden cardiac arrest (by having suffered an event and having had genetic tests), a device called an implantable cardioverter defibrillator (ICD) can be placed under the skin of the chest in a one-hour procedure. Like the AED, the ICD monitors heart rhythm. When the telltale deviations of ventricular tachycardia or ventricular fibrillation begin, it delivers a shock, preventing cardiac arrest.



An implantable cardioverter defibrillator delivers a shock to a heart whose ventricles are contracting chaotically, restoring a normal heartbeat.

ICDs have been so successful in preventing subsequent cardiac arrests that they may soon be offered to people at high risk for the condition. The two major risk factors are having had a previous myocardial infarction and a low ejection fraction, which is the volume of blood pumped with each heartbeat. Normal ejection fraction is 50% to 60%; low is below 30% to 40%. Scarring lowers the ejection fraction. An echocardiogram, which is an ultrasound scan of the heart, can reveal the ejection fraction. ■

15.1 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, called arterioles. Arterioles branch into the tiniest tubes, the capillaries, which 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 blood vessels form two circuits. The **pulmonary** (pul'mo-ner"e) **circuit** sends oxygen-poor blood to the lungs to pick up oxygen and unload carbon dioxide. The **systemic** (sis-tem'ik) **circuit** sends oxygen-rich blood and nutrients to all body cells and removes wastes. Without circulation, tissues would lack a supply of oxygen and nutrients, and wastes would accumulate. Such deprived cells soon begin irreversible change, which quickly leads to their death. Figure 15.1 shows blood flow through the heart and the general pattern of blood transport through the blood vessels of the cardiovascular system.

PRACTICE

1 Name the parts of the cardiovascular system.

2 Distinguish between the pulmonary circuit and the systemic circuit of the cardiovascular system.

15.2 STRUCTURE OF THE HEART

The heart is a hollow, cone-shaped, muscular pump. It is in the mediastinum of the thorax and rests on the diaphragm.

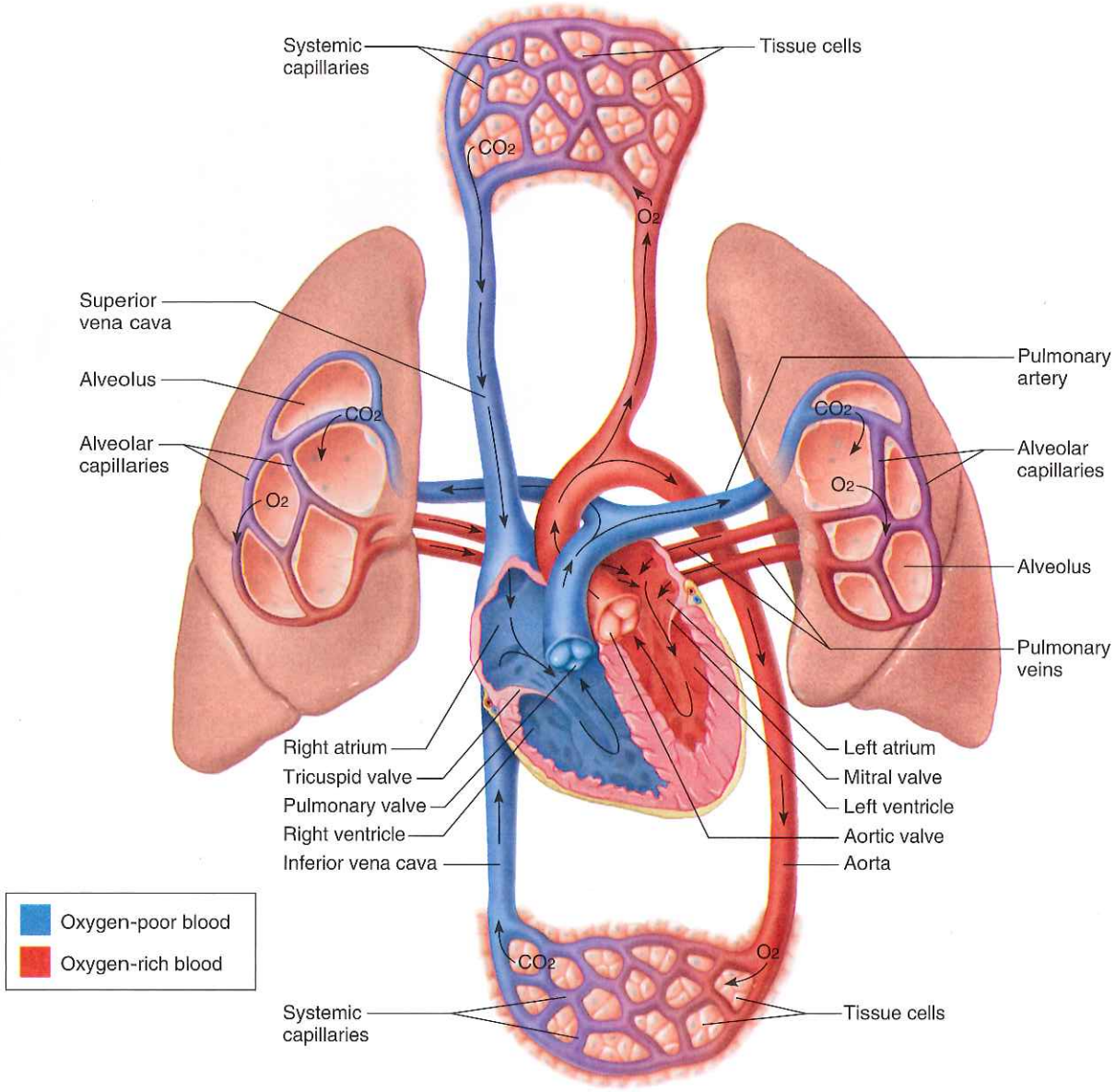


FIGURE 15.1 AP|R The cardiovascular system transports blood between the body cells and organs such as the lungs, intestines, and kidneys that communicate with the external environment. Vessels in the pulmonary circuit carry blood from the heart to the lungs and back to the heart, replenishing oxygen (O_2) and releasing the metabolic waste carbon dioxide (CO_2). Vessels of the systemic circuit supply all of the other cells. (Structures are not drawn to scale.)

Size and Location of the Heart

Heart size varies with body size. An average adult's heart is generally about 14 centimeters long and 9 centimeters wide (fig. 15.2).

The heart is bordered laterally by the lungs, posteriorly by the vertebral column, and anteriorly by the sternum (fig. 15.3 and reference plates 10, 16, 21, and 22, pp. 48, 51, and 54). 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. For this reason, it is possible to sense the *apical heartbeat* by feeling or listening to the chest wall between the fifth and sixth ribs, about 7.5 centimeters to the left of the midline.

Coverings of the Heart

The **pericardium** (per"i-kar'de-um), or pericardial sac, is a covering that encloses the heart and the proximal ends of the large blood vessels to which it attaches. The pericardium consists of an outer fibrous bag, the *fibrous pericardium,* that surrounds a more delicate, double-layered serous membrane. The innermost layer of this serous membrane, the *visceral pericardium* (epicardium), covers the heart. At the base of the heart, the visceral pericardium turns back upon itself to become the *parietal pericardium,* which forms the inner lining of the fibrous pericardium.

The fibrous pericardium is a tough, protective sac composed of dense connective tissue. It is attached to the central portion of the diaphragm, the posterior of the sternum, the

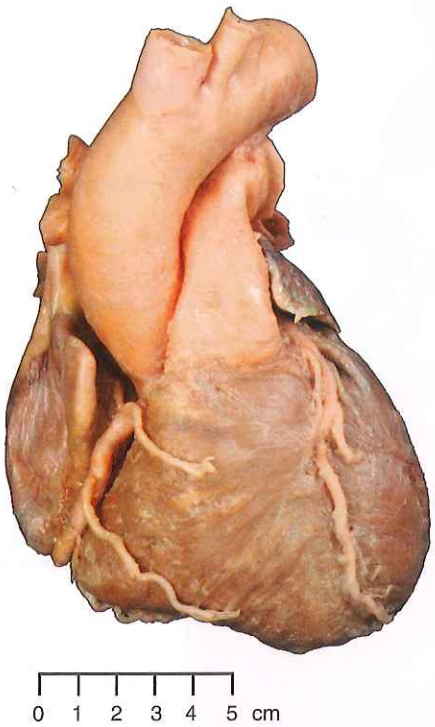


FIGURE 15.2 Anterior view of a human heart. This photo is not life-size, so a proportionately reduced ruler has been included to help the student grasp the true size of the organ.

vertebral column, and the large blood vessels emerging from the heart (see fig. 1.9*b*, p. 21 and reference plates 16 and 17, pp. 51–52 and fig. 15.4). Between the parietal and visceral layers of the pericardium is a space, the *pericardial cavity*, that contains a small volume of serous fluid that the pericardial membranes secrete. 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 can produce adhesions that attach the layers of the pericardium to each other. This painful condition interferes with heart movements.

PRACTICE

3 Where is the heart located?

4 Where would you listen to hear the apical heartbeat?

5 Distinguish between the visceral pericardium and the parietal pericardium.

6 What is the function of the fluid in the pericardial cavity?

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. 15.5).

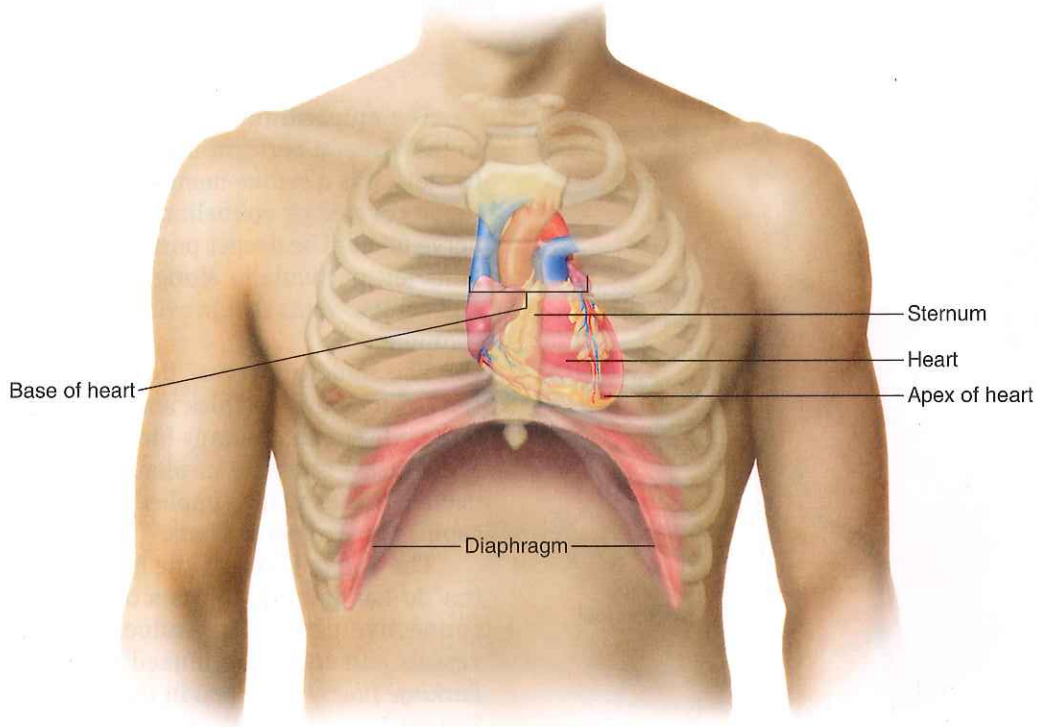


FIGURE 15.3 The heart is posterior to the sternum, where it rests on the diaphragm.

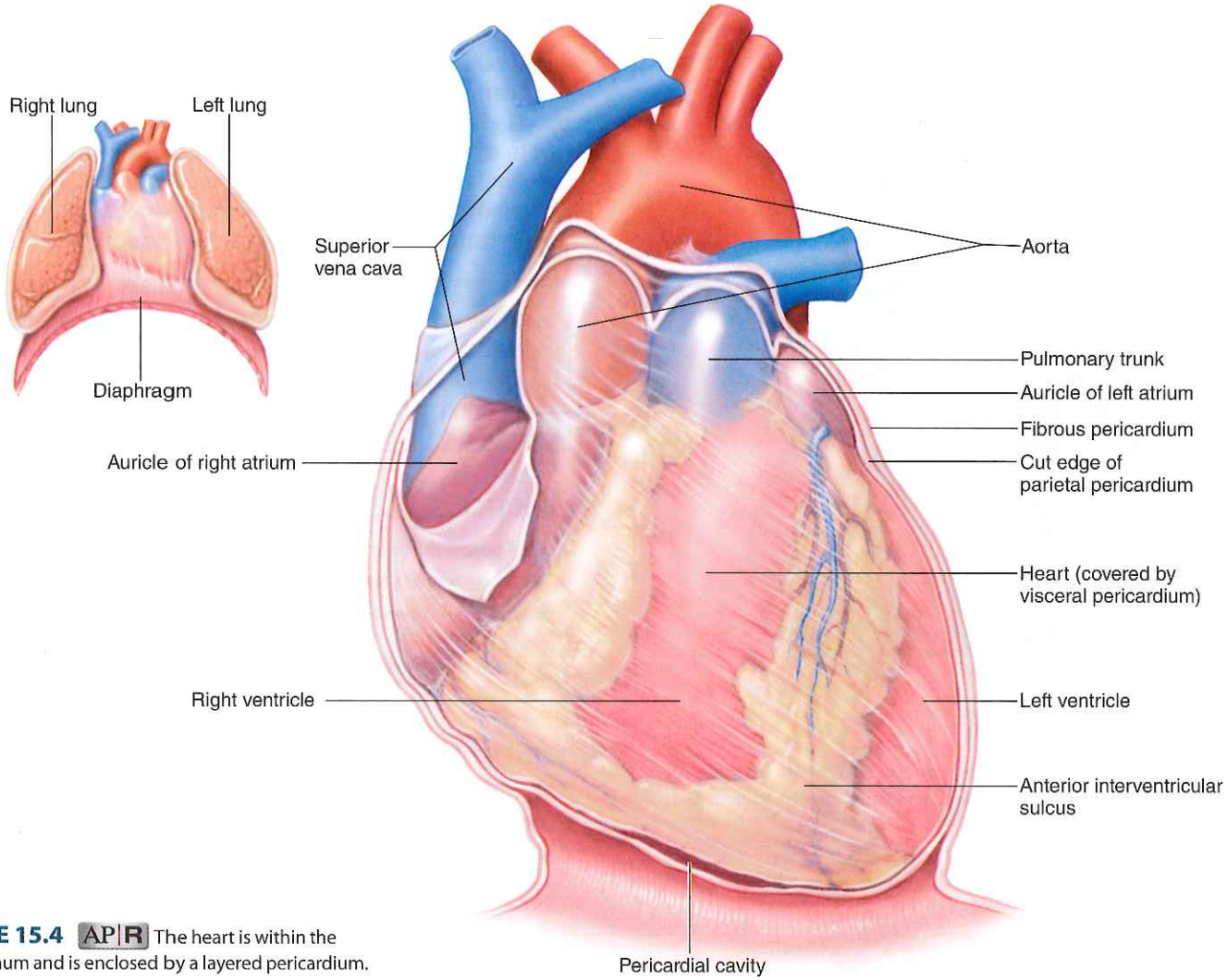


FIGURE 15.4 AP|R The heart is within the mediastinum and is enclosed by a layered pericardium.

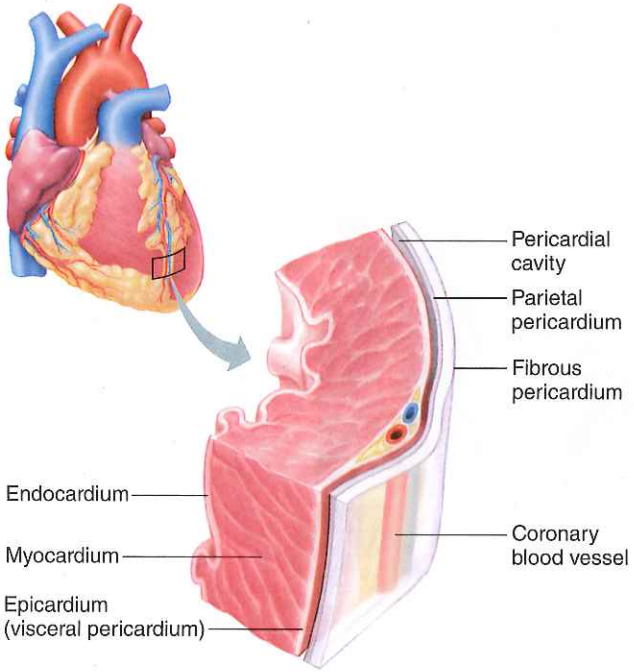


FIGURE 15.5 The heart wall has three layers: an endocardium, a myocardium, and an epicardium.

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 covered by epithelium, and it includes capillaries and nerve fibers. The deeper portion of the epicardium often contains fat, particularly along the paths of coronary arteries and cardiac veins that provide blood flow through the myocardium.

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

The inner layer of the heart wall, or **endocardium** (en″do-kar′de-um), consists of epithelium and underlying connective tissue. The endocardium also contains blood vessels and some specialized cardiac muscle fibers called *Purkinje fibers,* described in the section "Cardiac Conduction System."

The endocardium lines all of the heart chambers and covers the structures, such as the heart valves, that project

TABLE 15.1 | Wall of the Heart

Layer	Composition	Function
Epicardium (visceral pericardium)	Serous membrane of connective tissue covered with epithelium and including blood capillaries, lymph capillaries, and nerve fibers	Forms a protective outer covering; secretes serous fluid
Myocardium	Cardiac muscle tissue separated by connective tissue and including blood capillaries, lymph capillaries, and nerve fibers	Contracts to pump blood from the heart chambers
Endocardium	Membrane of epithelium and underlying connective tissue, including blood vessels and specialized muscle fibers	Forms a protective inner lining of the chambers and valves

into them. This inner lining is also continuous with the inner linings (endothelium) of the blood vessels attached to the heart and throughout the cardiovascular system. Table 15.1 summarizes the characteristics of the three layers of the heart wall.

Heart Chambers and Valves

Internally the heart is divided into four hollow chambers, two on the left and two on the right. The upper chambers, called **atria** (a′tre-ah) (sing., *atrium*), have thin walls and receive blood returning to the heart. Small, earlike projections called **auricles** (aw′ri-klz) extend anteriorly from the atria, slightly increasing atrial volume (see fig. 15.4). The lower chambers, the **ventricles** (ven′tri-klz), force the blood out of the heart into arteries.

A structure called the *interatrial septum* separates the right from the left atrium. An *interventricular septum* separates the two ventricles. The atrium on each side communicates with its corresponding ventricle through an opening called the **atrioventricular orifice** (a″tre-o-ven-trik′u-lar ori-fis), which is guarded by an *atrioventricular valve* (AV valve).

Grooves on the surface of the heart mark the divisions between its chambers, and they also contain major blood vessels that supply the heart tissues. The deepest of these grooves is the **atrioventricular** (coronary) **sulcus** (a″tre-o-ven-trik′u-lar sul′kus), which encircles the heart between the atria and ventricles. Two **interventricular** (anterior and posterior) **sulci** mark the septum that separates the right and left ventricles (see fig. 15.4).

PRACTICE

7 Describe the layers of the heart wall.

8 Name and locate the four chambers of the heart.

The right atrium receives blood from two large veins: the *superior vena cava* and the *inferior vena cava.* These veins return blood, low in oxygen, from tissues. A smaller vein, the *coronary sinus,* also drains venous blood into the right atrium from the myocardium of the heart.

A large **tricuspid valve** (right atrioventricular valve) guards the atrioventricular orifice between the right atrium and the right ventricle. It is composed of three leaflets, or cusps, as its name implies. This valve permits the blood to move from the right atrium into the right ventricle and prevents it from moving in the opposite direction. The cusps fold passively out of the way against the ventricular wall when the blood pressure is greater on the atrial side, and they close passively when the pressure is greater on the ventricular side (figs. 15.6, 15.7, 15.8, and 15.9).

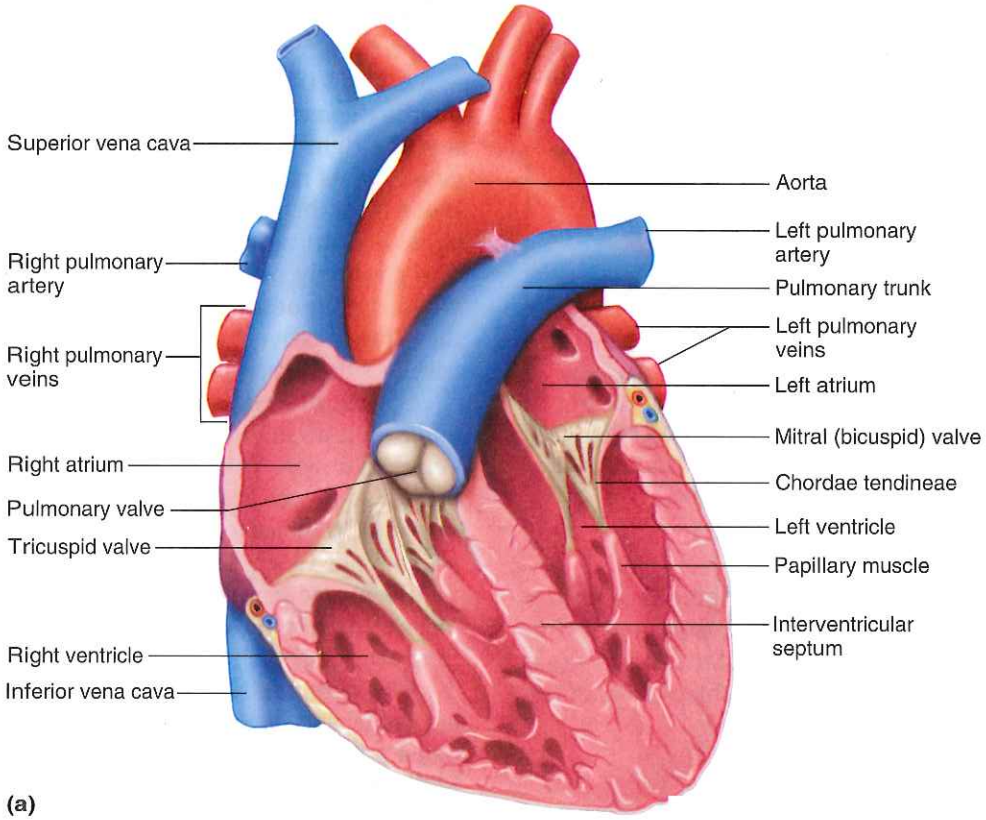
Strong, fibrous strings, called *chordae tendineae* (kor′de ten′dĭ-ne), attach to the cusps of the tricuspid valve on the ventricular side. These strings originate from small mounds of cardiac muscle tissue, the **papillary muscles** (pap′ĭ-ler″e mus′elz), that project inward from the walls of the ventricle (see fig. 15.7). The papillary muscles contract when the right ventricle contracts. As the tricuspid valve closes, these muscles pull on the chordae tendineae and prevent the cusps from swinging back (everting) into the right atrium.

The right ventricle has a thinner muscular wall than the left ventricle (see fig. 15.6). This right chamber pumps the blood a fairly short distance to the lungs against a relatively low resistance to blood flow. The left ventricle, on the other hand, must force the blood to all the other parts of the body against a much greater resistance to flow.

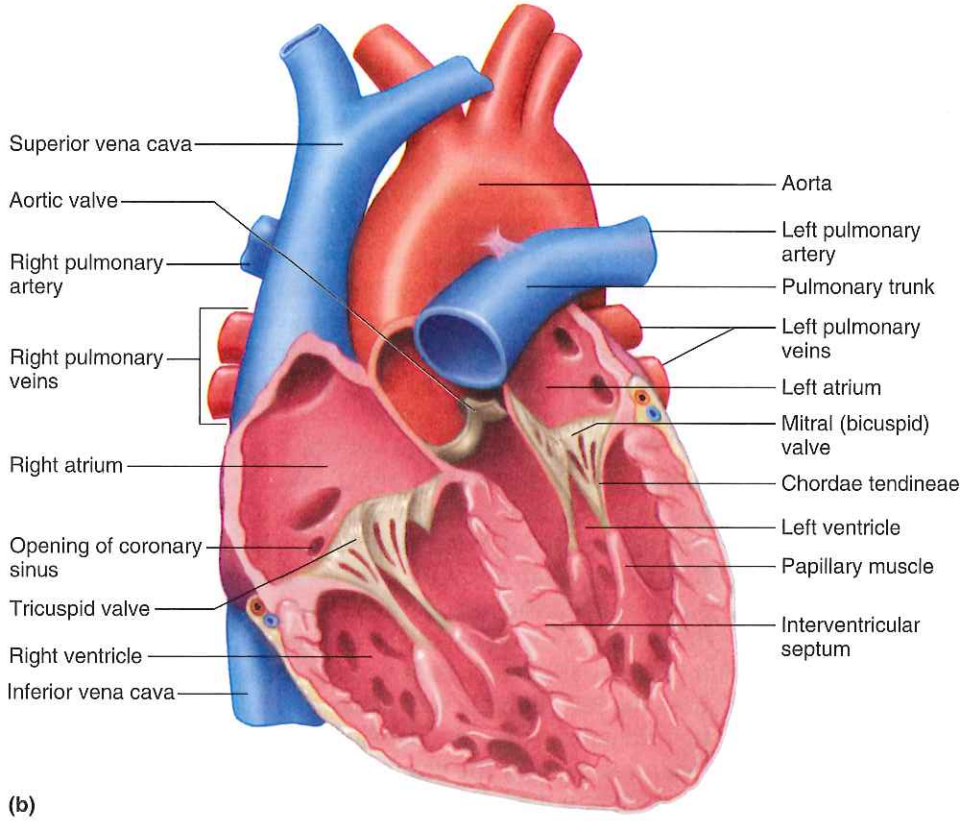
When the muscular wall of the right ventricle contracts, the blood inside its chamber is put under increasing pressure, and the tricuspid valve closes passively. As a result, the only exit for the blood is through the *pulmonary trunk,* which divides to form the left and right *pulmonary arteries* that lead to the lungs. At the base of this trunk is a **pulmonary valve** (pulmonary semilunar valve), which consists of three cusps (see figs. 15.8 and 15.9). This valve opens as the right ventricle contracts. However, when the ventricular muscles relax, the blood begins to back up in the pulmonary trunk. This closes the pulmonary valve, preventing backflow into the right ventricle. Unlike the tricuspid valve, the pulmonary valve does not have chordae tendineae or papillary muscles attached to its cusps.

The left atrium receives the blood from the lungs through four *pulmonary veins*—two from the right lung and two from the left lung. The blood passes from the left atrium into the left ventricle through the atrioventricular orifice, which a valve guards. This valve consists of two leaflets and is named the **mitral valve** (shaped like a mitre, a type of headpiece) or bicuspid valve or left atrioventricular valve. It prevents the blood from flowing back into the left atrium from the left ventricle when the ventricle contracts. As with the tricuspid valve, the papillary muscles and the chordae tendineae prevent the cusps of the mitral valve from swinging back into the left atrium.

When the left ventricle contracts, the mitral valve closes passively, and the only exit is through a large artery called the *aorta.* Its branches distribute blood to all parts of the body.



(a)



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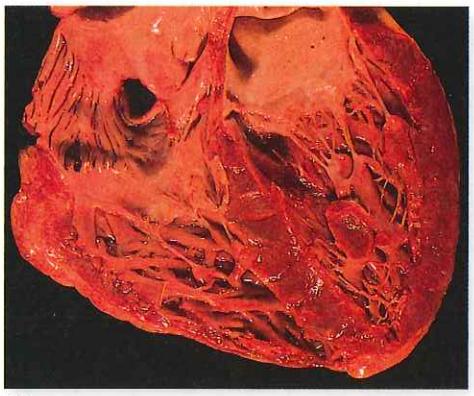


(c)

FIGURE 15.6 AP|R Frontal sections of the heart. (*a*) Drawings show the connection between the right ventricle and the pulmonary trunk and (*b*) the connection between the left ventricle and the aorta, as well as the four hollow chambers. (*c*) A cadaver heart.

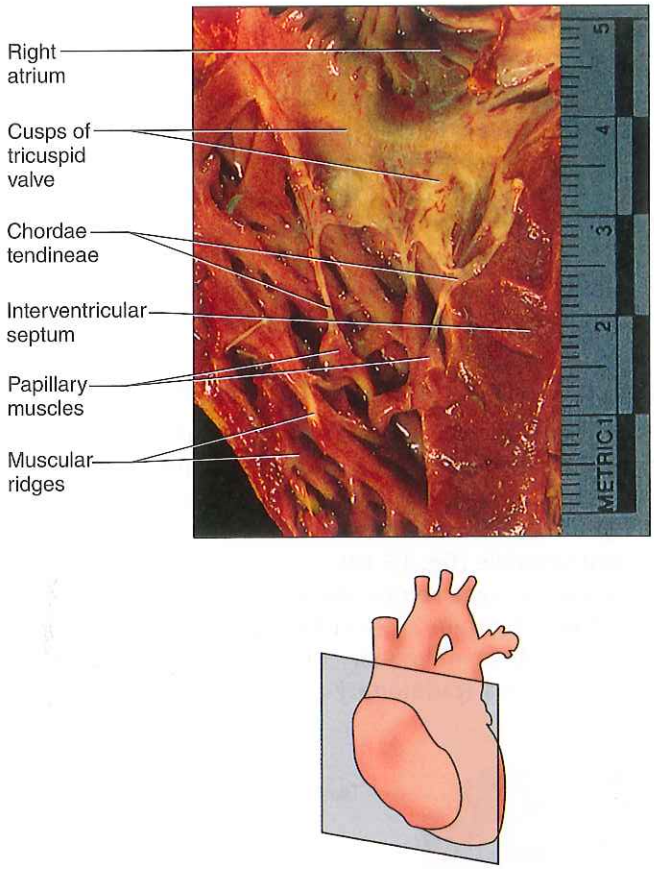


FIGURE 15.7 Photograph of a human tricuspid valve.

FIGURE 15.8 Photograph of the heart valves (superior view). Figure 15.9 labels the valves as seen in this photograph.

At the base of the aorta is an **aortic valve** (aortic semilunar valve) that consists of three cusps (see figs. 15.8 and 15.9). It 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 left ventricle.

The mitral and tricuspid valves are also called atrioventricular valves because they are between atria and ventricles. The pulmonary and aortic valves are also called semilunar because of the half-moon shapes of their cusps. Table 15.2 summarizes the locations and functions of the heart valves.

TABLE 15.2 | Valves of the Heart

Valve	Location	Function
Tricuspid valve	Right atrioventricular orifice	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
Mitral valve	Left atrioventricular orifice	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

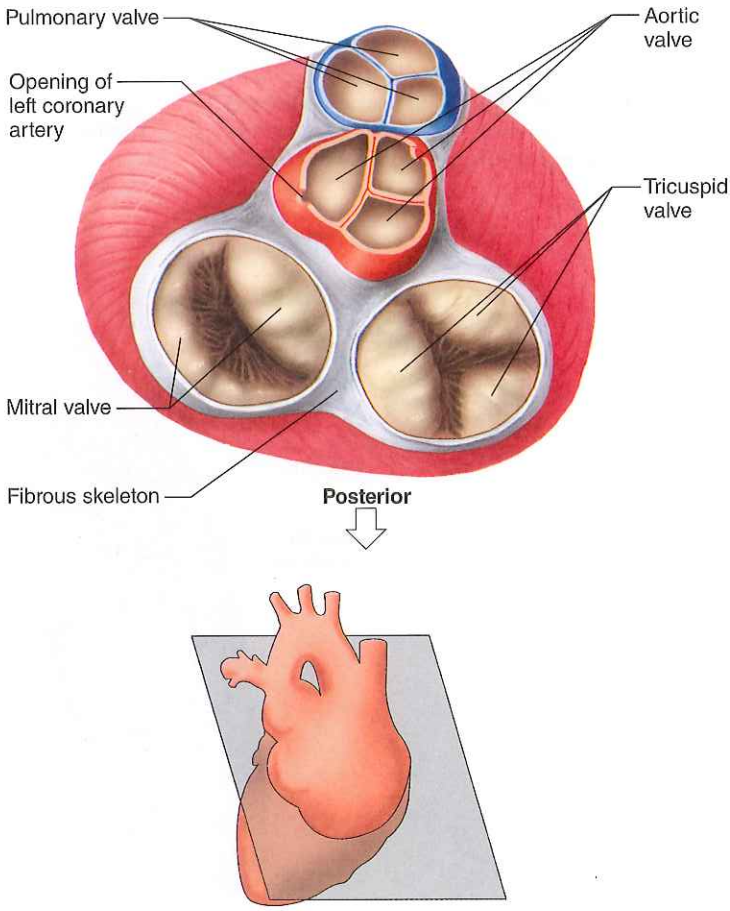


FIGURE 15.9 The skeleton of the heart consists of fibrous rings to which the heart valves are attached (superior view).

Q: *What type of section has been made through the heart to expose the valves?*

Answer can be found in Appendix G on page 938.

Mitral 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 (flows back) into the left atrium. Symptoms of MVP include chest pain, palpitations, fatigue, and anxiety.

People with MVP are particularly susceptible to endocarditis. This inflammation of the endocardium due to an infection appears as a plantlike growth on the valve. People with MVP take antibiotics before undergoing dental work, to prevent *Streptococcus* bacteria in the mouth from migrating through the blood to the heart and spreading this infection.

PRACTICE

9 Which blood vessels carry blood into the right atrium?

10 Where does blood go after it leaves the right ventricle?

11 Which blood vessels carry blood into the left atrium?

12 What prevents blood from flowing back into the ventricles when they relax?

Skeleton of the Heart

Rings of dense connective tissue surround the pulmonary trunk and aorta at their proximal ends. These rings are continuous with others that encircle the atrioventricular orifices. They provide firm attachments for the heart valves and for muscle fibers and 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 part of the septum between the ventricles (interventricular septum), constitute the *skeleton of the heart* (fig. 15.9).

Path of Blood Through the Heart

Blood low in oxygen and high in carbon dioxide enters the right atrium through the venae cavae and the coronary sinus. As the right atrial wall contracts, the blood passes through the right atrioventricular orifice and enters the chamber of the right ventricle (fig. 15.10).

When the right ventricular wall contracts, the tricuspid valve closes the right atrioventricular orifice, and the blood moves through the pulmonary valve into the pulmonary trunk and its branches (pulmonary arteries). From these vessels,

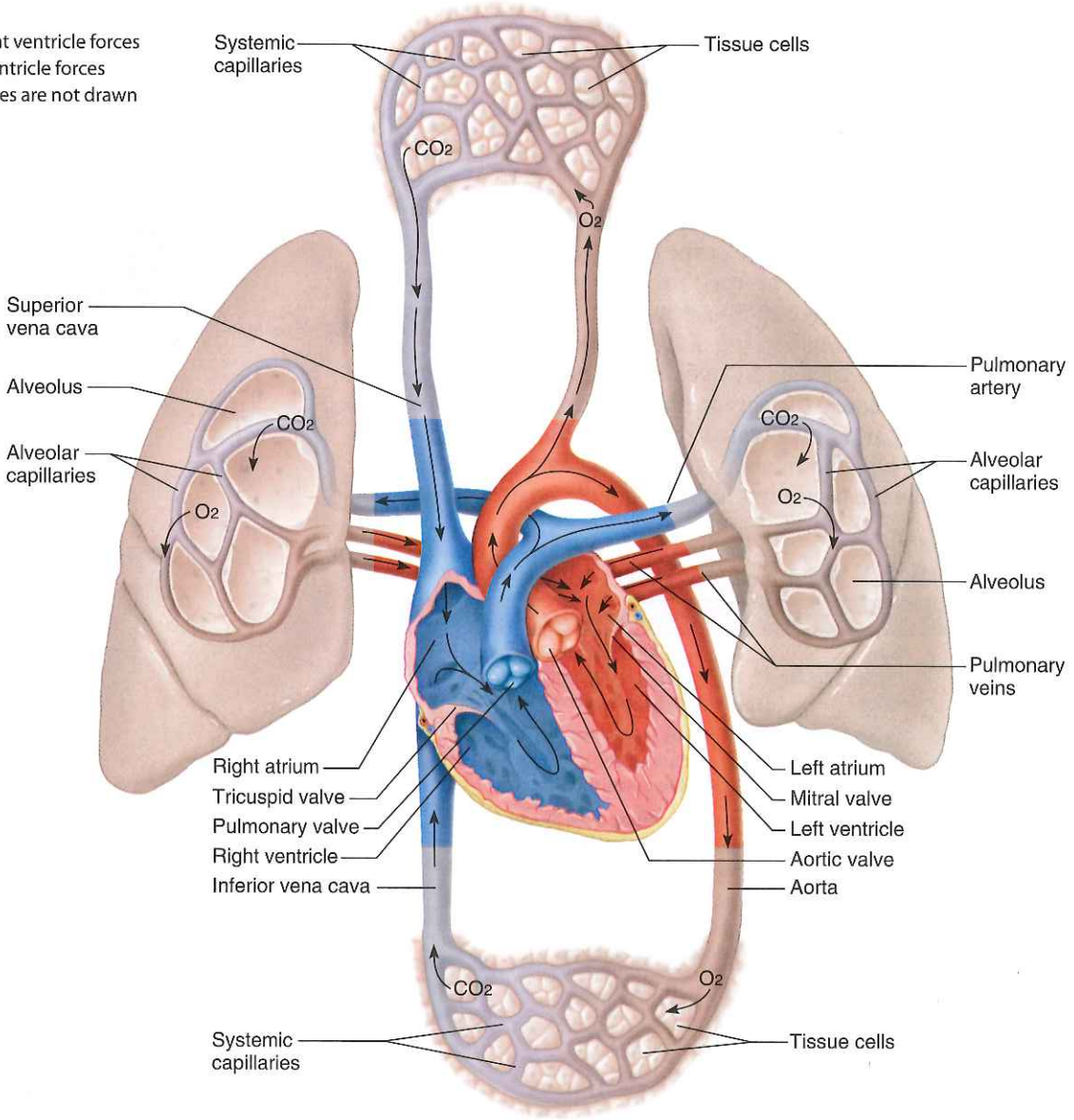


FIGURE 15.10 AP|R The right ventricle forces blood to the lungs, whereas the left ventricle forces blood to all other body parts. (Structures are not drawn to scale.)

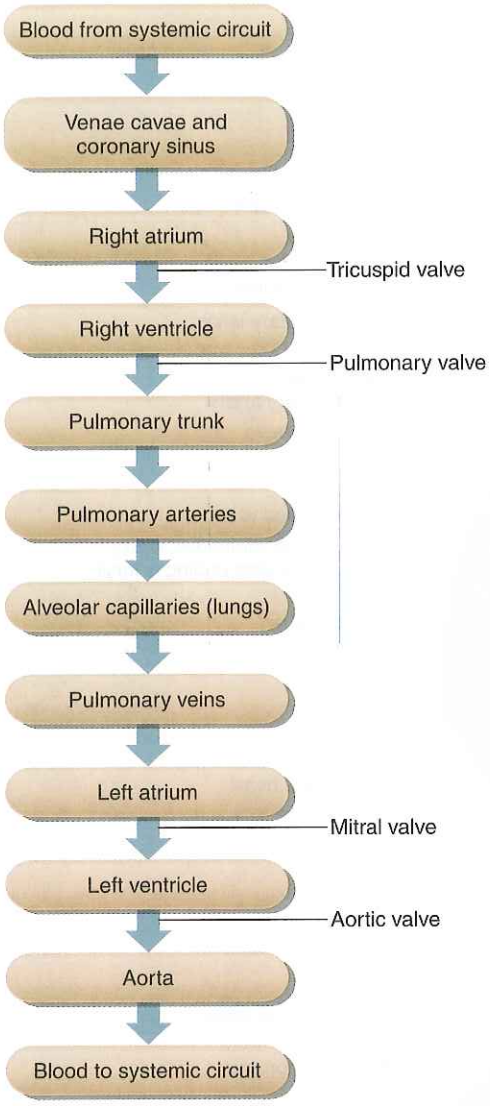


FIGURE 15.11 AP|R Path of blood through the heart and pulmonary circuit.

blood enters the capillaries associated with the alveoli (microscopic air sacs) of the lungs. Gas exchange occurs between the blood in the capillaries and the air in the alveoli. The oxygen-rich blood, 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 the blood moves through the left atrioventricular orifice and into the chamber of the left ventricle. When the left ventricular wall contracts, the mitral valve closes the left atrioventricular orifice, and the blood passes through the aortic valve into the aorta and its branches. Figure 15.11 summarizes the path the blood takes as it moves through the heart to the alveolar capillaries and systemic capillaries, then back to the heart.

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 superior to the aortic valve (fig. 15.12).

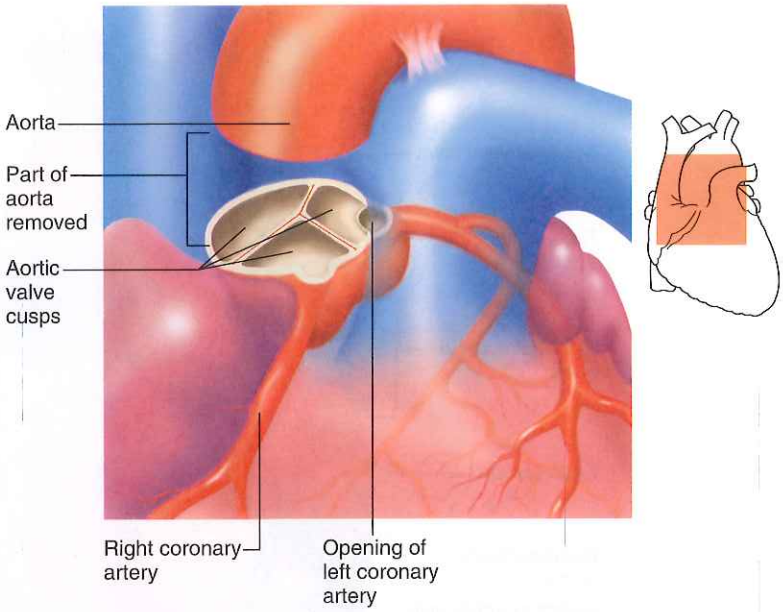


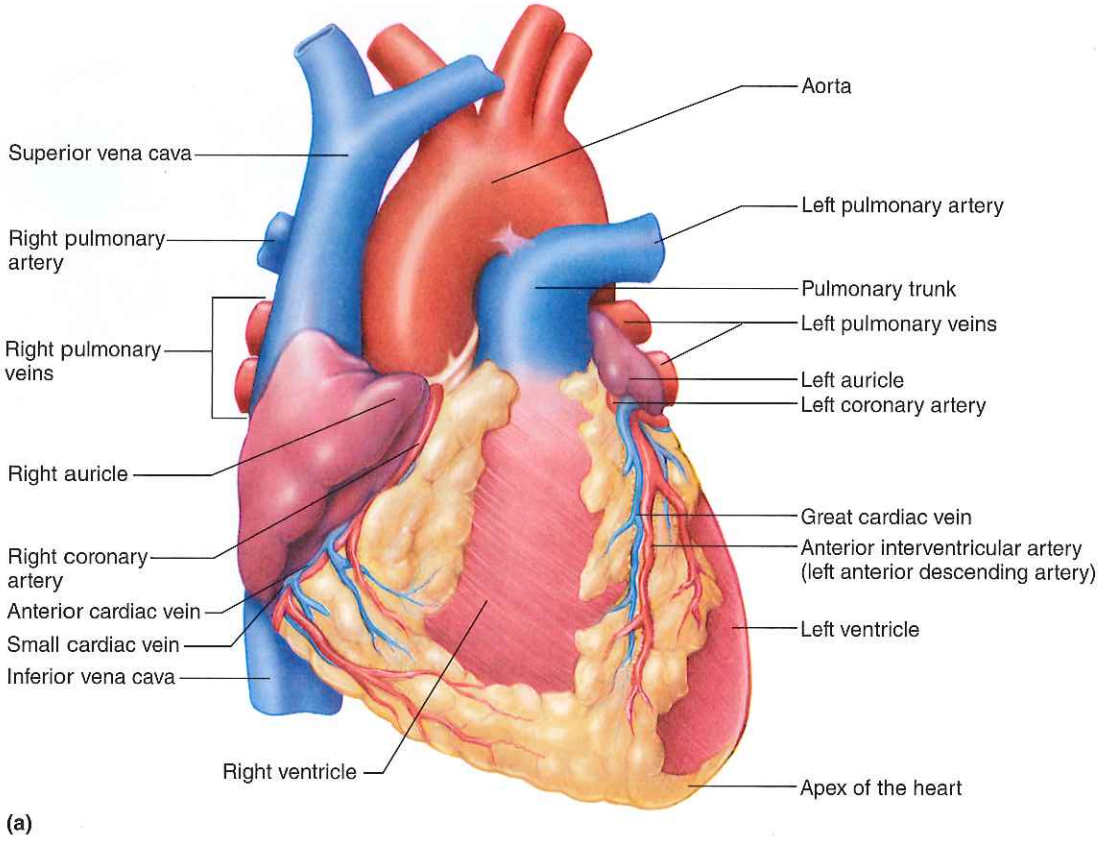
FIGURE 15.12 The openings of the coronary arteries lie just superior to the aortic valve.

The right coronary artery passes along the atrioventricular sulcus between the right atrium and the right ventricle. It gives off two major branches—a *posterior interventricular artery,* which travels along the posterior interventricular sulcus and supplies the walls of both ventricles, and a *marginal artery,* which passes along the lower border of the heart. Branches of the marginal artery supply the walls of the right atrium and the right ventricle (figs. 15.13 and 15.14).

Magnetic resonance imaging (MRI) can image coronary arteries. Blood flow appears as a bright signal, and areas of diminished or absent blood flow, or blood turbulence, appear as blank areas. This approach is less invasive than *coronary angiography,* in which a catheter is snaked through a blood vessel into the heart and a contrast agent is used to show heart structure.

One branch of the left coronary artery, the *circumflex artery,* follows the atrioventricular sulcus between the left atrium and the left ventricle. Its branches supply blood to the walls of the left atrium and the left ventricle. Another branch of the left coronary artery, the *anterior interventricular artery* (or *left anterior descending artery*), lies in the anterior interventricular sulcus. Its branches supply the walls of both ventricles (figs. 15.13 and 15.14).

The heart must beat continually to supply blood to the tissues. To do this, myocardial cells require a constant supply of oxygen-rich blood. The myocardium contains many capillaries fed by branches of the coronary arteries. 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 oxygen and nutrients to the myocardium when a coronary artery is blocked.



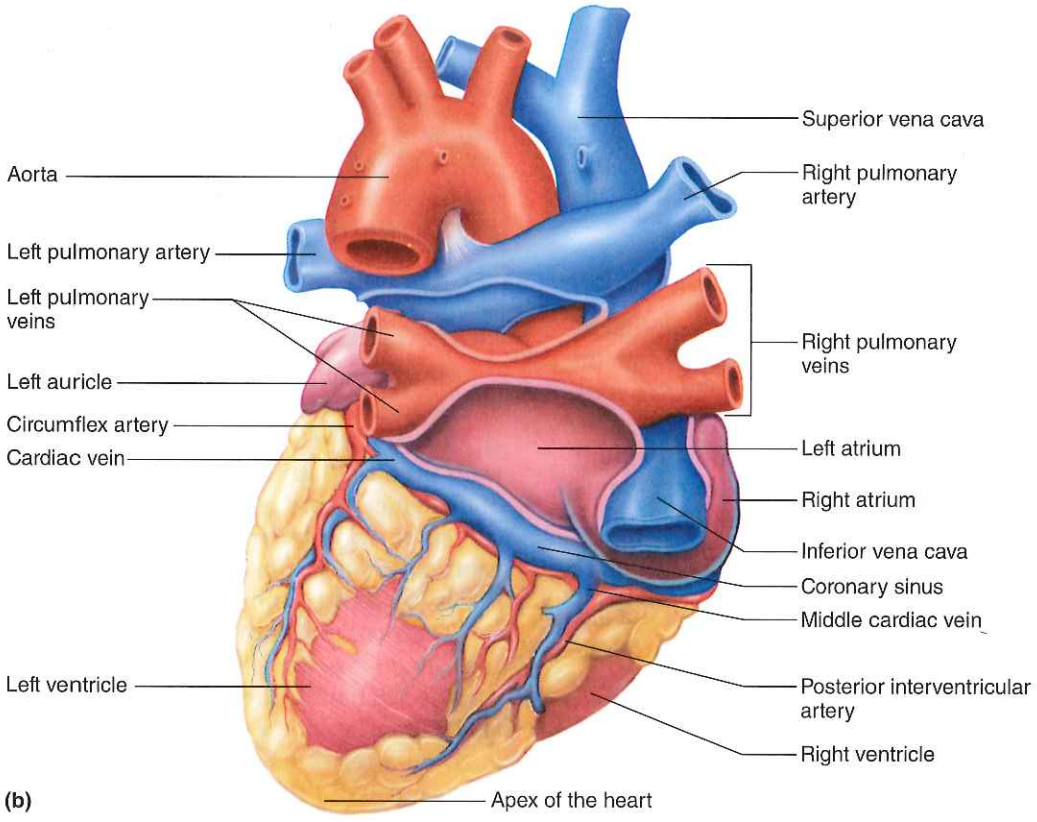


FIGURE 15.13 AP|R Blood vessels associated with the surface of the heart. (*a*) Anterior view. (*b*) Posterior view.

A thrombus or embolus that partially blocks or narrows a coronary artery branch causes a decrease in blood flow called *ischemia*. This deprives myocardial cells of oxygen, producing a painful condition called *angina pectoris*. The pain usually happens during physical activity, when oxygen demand exceeds supply. Pain lessens with rest. Emotional disturbance may also trigger angina pectoris.

Angina pectoris feels like heavy pressure, tightening, or squeezing in the chest, usually behind the sternum or in the anterior upper thorax. The pain may radiate to the neck, jaw, throat, left shoulder, left upper limb, back, or upper abdomen. Profuse perspiration (diaphoresis), difficulty breathing (dyspnea), nausea, or vomiting may occur.

A blood clot may completely obstruct a coronary artery (coronary thrombosis), killing tissue in that part of the heart. This is a *myocardial infarction* (MI) or heart attack.

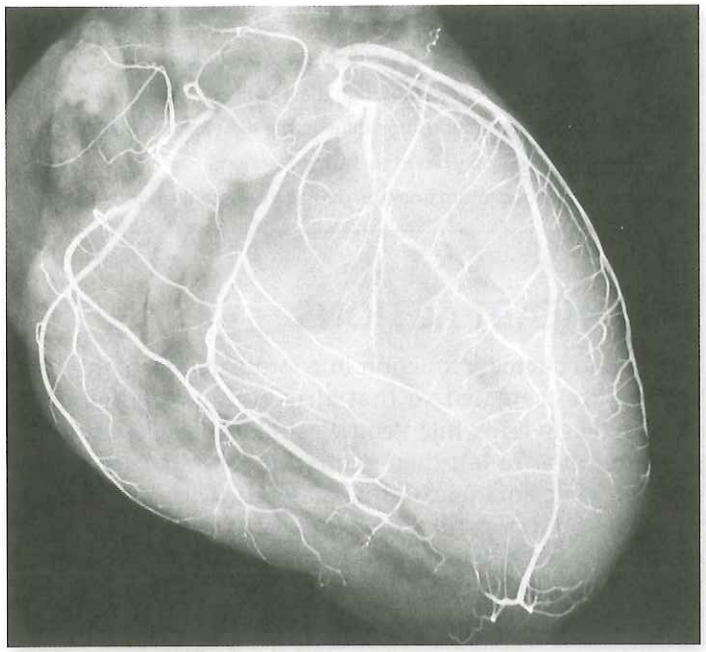


FIGURE 15.14 An angiogram (radiograph) of the coronary arteries is a diagnostic procedure used to examine specific blood vessels.

In most body parts, blood flow in arteries peaks during ventricular contraction. However, blood flow in the vessels of the myocardium is poorest during ventricular contraction. This is because the muscle fibers of the myocardium compress nearby vessels as they contract, interfering with blood flow. Also, the openings into the coronary arteries are partially blocked as the flaps of the aortic valve open. Conversely, during ventricular relaxation, the myocardial vessels are no longer compressed, and the aortic valve does not block the orifices of the coronary arteries. This increases blood flow into the myocardium.

Branches of the **cardiac veins** drain blood that has passed through the capillaries of the myocardium. Their paths roughly parallel those of the coronary arteries. As figure 15.13*b* shows, these veins join the **coronary sinus,** which is an enlarged vein on the posterior surface of the heart in the atrioventricular sulcus. The coronary sinus empties into the right atrium. Figure 15.15 summarizes the path of blood that supplies the tissues of the heart.

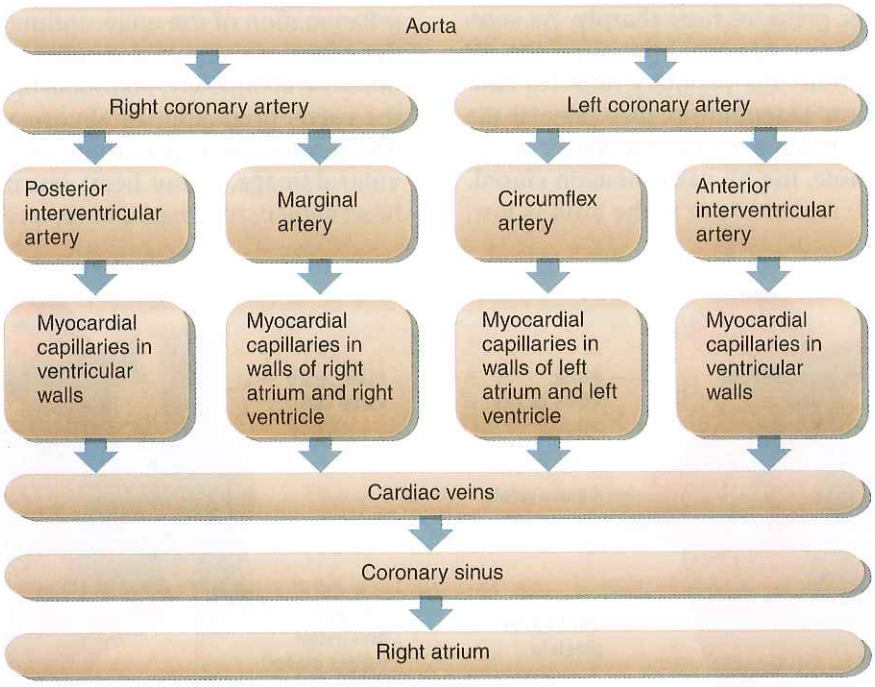


FIGURE 15.15 Path of blood through the coronary circulation.

PRACTICE

13 Which structures make up the skeleton of the heart?
14 Review the path of blood through the heart.
15 How does blood composition differ in the right and left ventricle?
16 Which vessels supply blood to the myocardium?
17 How does blood return from the cardiac tissues to the right atrium?

15.3 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). From Science to Technology 15.1 describes treatments for a failing heart.

Cardiac Cycle

During a cardiac cycle, the pressure in the heart chambers rises and falls. These changes open and close the valves, much like a door being blown open or closed by the wind. Pressure in the ventricles is low early in diastole, and the pressure difference between atria and ventricles opens the AV valves. The ventricles fill. About 70% of the returning blood enters the ventricles prior to contraction, and ventricular pressure gradually increases. During atrial systole, the remaining 30% of returning blood is pushed into the ventricles, and ventricular pressure increases. Then, as the ventricles contract, ventricular pressure rises sharply. As soon as the ventricular pressure exceeds atrial pressure, the AV valves close. At the same time, the papillary muscles contract. By pulling on the chordae tendineae, they prevent the cusps of the AV valves from bulging too far into the atria.

During ventricular systole, the AV valves remain closed. The atria are now relaxed, and pressure in the atria is 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. 15.16).

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 differences across the semilunar valves open the pulmonary and aortic valves, and blood is ejected from each valve's ventricle into these arteries.

As blood flows out of the ventricles, ventricular pressure begins to drop, and it falls even farther as the ventricles relax. When ventricular pressure is lower than the 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 AV valves open, and the ventricles begin to fill once more. Atria and ventricles relax for a brief interval.

Heart Sounds

A heartbeat heard through a stethoscope sounds like "lubb-dupp." These sounds are due to vibrations in the heart tissues produced as the blood flow is suddenly slowed with the contraction and relaxation of the heart chambers and with the closing of the valves.

The first part of a heart sound *(lubb)* occurs during ventricular systole, when the AV valves are closing. The second part (*dupp*) occurs during ventricular diastole, when the pulmonary and aortic valves are closing.

Heart sounds are of particular interest because they can indicate 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 degree of valvular damage. Many heart murmurs are harmless. Open-heart surgery may repair or replace severely damaged valves.

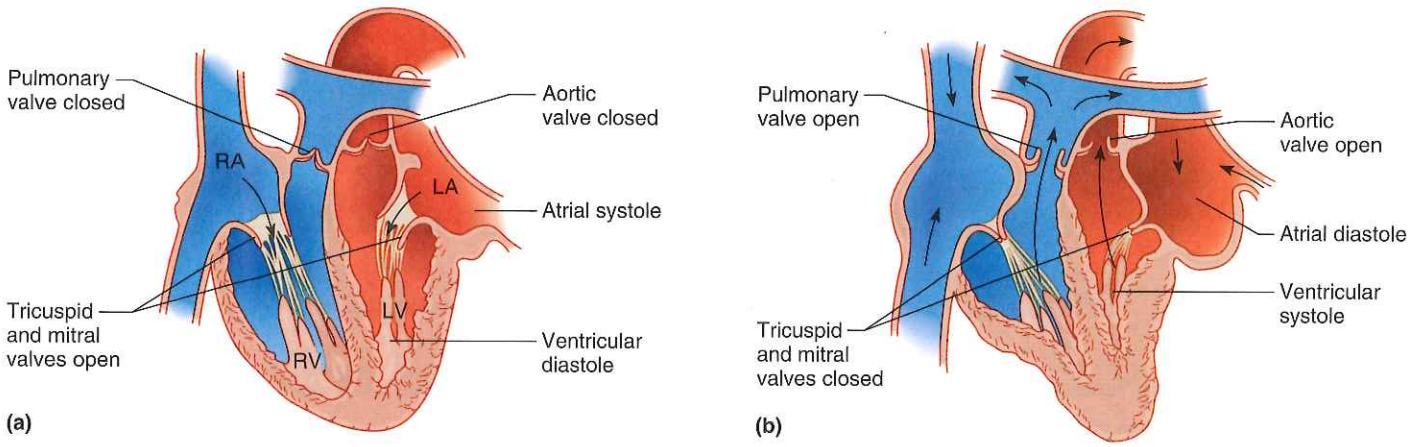


FIGURE 15.16 AP|R The atria (*a*) empty during atrial systole and (*b*) fill with blood during atrial diastole.

15.1 FROM SCIENCE TO TECHNOLOGY

Replacing the Heart—From Transplants to Stem Cell Implants

Medical science offers several ways to aid or even replace a failing heart. In a *heart transplant,* 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 similarly prepared and is attached to the atrial cuffs remaining in the recipient's thorax. Finally, the recipient's aorta and pulmonary arteries are connected to those of the donor heart (fig. 15A).

Donor hearts are scarce. A mechanical half-heart, called a *left ventricular assist device* (LVAD), can often maintain cardiac function and counter deterioration long enough for a heart to become available. An LVAD allows a patient to resume some activities and to exercise, which can increase the chance of success of an eventual heart transplant. Some patients too ill to receive transplants are surviving with permanently implanted LVADs. Developed as a way to keep a patient alive for a short time until a donor heart became available, the LVAD is now considered a long-term therapy.

An implantable replacement heart became available in 2006 for people who are not candidates for heart transplantation and have less than a month to live. The two-pound, titanium and plastic cardiac stand-in consists of an internal motor-driven hydraulic pump, a battery and electronics 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. Newer implantable replacement hearts are smaller and can provide up to five years of life.

Stem cell technology may allow researchers to patch failing hearts with new cardiac muscle. Human cardiac muscle tissue can be cultured from reprogrammed somatic cells (see From Science to Technology 3.1, p. 116) or from stem cells. In laboratory dishes, the tissue contracts, as cardiac muscle would in the body. These cells are combined with a synthetic, compatible biomaterial elastic enough to stretch as the cells divide and degrade in a controlled way. The idea is that "stem cell heart patches" would consist of scaffolding made of such a biomaterial that would support the cells as they nestle into a damaged heart. As the cells contract, the synthetic portion degrades, leaving the pulsating patch. If the cells originate from the patient, the patch would not be rejected. ■

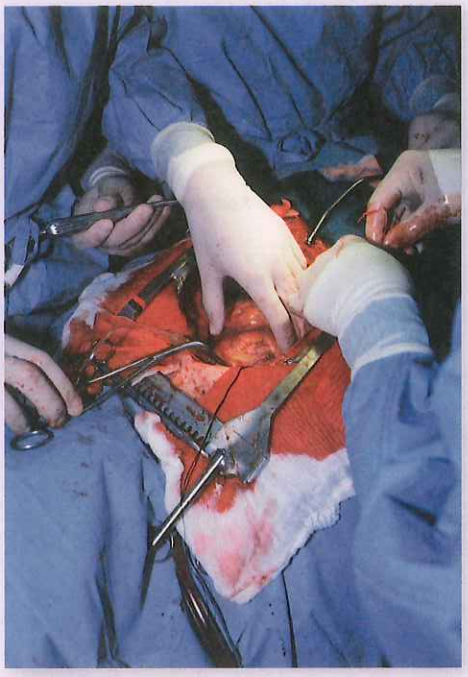


FIGURE 15A A heart transplant can save a life. A heart that might have died with its donor may provide a new lease on life for a recipient, thanks to our understanding of the immune system—and a well-trained medical team.

Using a stethoscope, it is possible to hear sounds associated with the aortic and pulmonary valves by listening from the second intercostal space on either side of the sternum. The *aortic sound* comes from the right, and the *pulmonic sound* from the left. The sound associated with the mitral valve can be heard from the fifth intercostal space at the nipple line on the left. The sound of the tricuspid valve can be heard at the fifth intercostal space just to the right of the sternum (fig. 15.17).

Cardiac Muscle Fibers

Recall that cardiac muscle fibers function like those of skeletal muscles, but the fibers connect in branching networks (chapter 9, pp. 309–310). The intercalated discs, which include gap junctions, join cardiac muscle cells, allowing action potentials to spread throughout a network of cells that contracts as a unit (chapter 5, p. 172).

A mass of merging cells that act 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, except for a small area in the right atrial floor. In this region, fibers of the cardiac conduction system connect the *atrial syncytium* and the *ventricular syncytium.*

PRACTICE

18 Describe the pressure changes in the atria and ventricles during a cardiac cycle.

19 What causes heart sounds?

20 What is a functional syncytium?

21 Where are the functional syncytia of the heart?

Cardiac Conduction System

The heart is *autorhythmic,* able to initiate contraction itself without external nervous stimulation. 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 (cardiac impulses) throughout the myocardium. They comprise the **cardiac conduction system,** which coordinates the events of the cardiac cycle.

A key portion of the conduction system is the **SA node** (**sinoatrial node** or **sinuatrial node**), which is a small, elongated mass of specialized cardiac muscle tissue just beneath

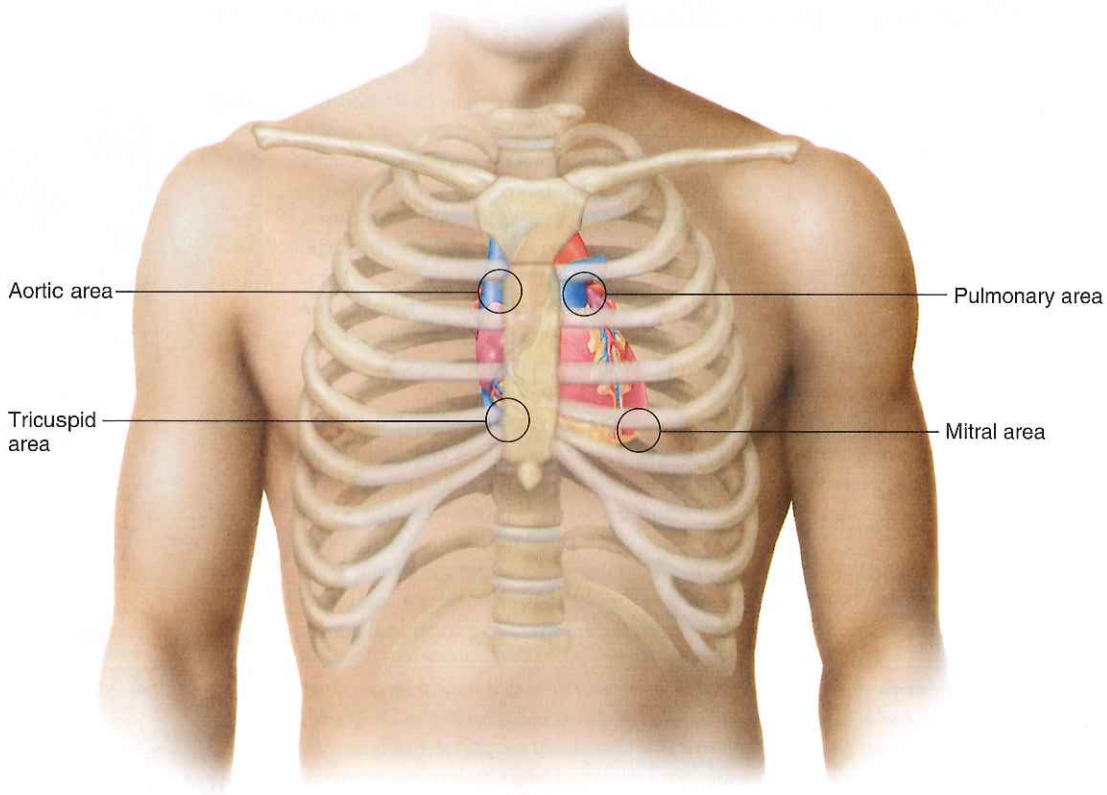


FIGURE 15.17 Thoracic regions where the sounds of each heart valve are most easily heard.

the epicardium. It is 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 SA node reach threshold spontaneously. Recall from chapter 10 (pp. 375–376) that an action potential in a neuron is triggered by a depolarizing input from presynaptic neurons. In contrast, an SA node reaches threshold and triggers an action potential on its own. A number of changing conditions appear to play a role in stimulating the SA node to reach threshold. These include a progressive increase in permeability to calcium ions and sodium ions and decreasing permeability to potassium ions.

SA node activity is rhythmic. The SA node initiates one impulse after another, more than 80 times a minute in an adult (resting heart rate is usually closer to 70 beats per minute due to inhibition by the parasympathetic nervous system). The SA node is also called the **pacemaker** because it initiates the heart's rhythmic contractions. From the SA node, bundles of atrial muscle, called *internodal atrial muscle,* preferentially conduct impulses to more distant regions of the atria. Then, because gap junctions connect cardiac muscle cells, the resulting impulse spreads into the surrounding atrial myocardium and stimulates the muscle fibers to contract.

The right and left atria 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 cardiac impulse passes along fibers (junctional fibers) of the cardiac conduction system that lead to a mass of specialized cardiac muscle tissue called the **AV node** (atrioventricular node). This node is in the inferior part of the interatrial septum and just beneath the endocardium. It provides the only normal conduction pathway between the atrial and ventricular syncytia, because the fibrous skeleton does not conduct the impulse.

The junctional fibers that conduct the cardiac impulse into the AV node have small diameters. Small fibers conduct impulses slowly, and thus they delay transmission of the impulse. The impulse is delayed further as it moves through the AV node. This allows time for the atria to contract completely so they empty all their blood into the ventricles prior to ventricular systole. Once the cardiac impulse reaches the distal side of the AV node, it passes into a group of large fibers that make up the **AV bundle** (atrioventricular bundle or bundle of His), and the impulse moves rapidly through them. The AV 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). These larger fibers carry the impulse to distant regions of the ventricular myocardium much faster than cell-to-cell conduction could. As a result, the massive ventricular myocardium contracts as a functioning unit.

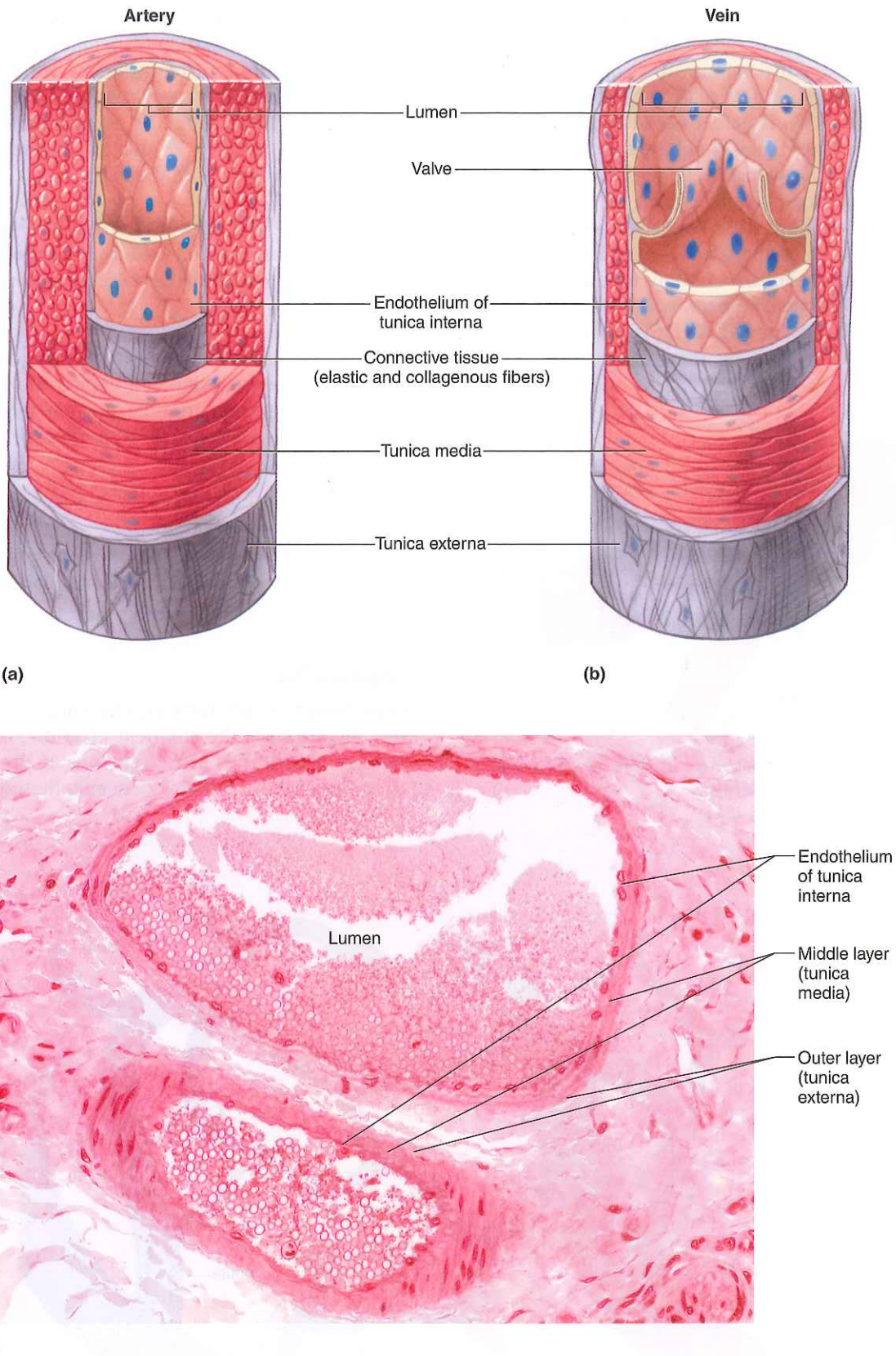


FIGURE 15.25 AP|R Blood vessels. (*a*) The wall of an artery. (*b*) The wall of a vein. (*c*) Note the structural differences in these cross sections of an arteriole (bottom) and a venule (top) (200×).

Vasomotor fibers stimulate the smooth muscle cells 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-dila′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 (fig. 15.25*c*), but the middle and outer layers thin as the arterioles approach the 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. 15.26). Arterioles, which are microscopic continuations of arteries, give off branches called *metarterioles* that, in turn, join capillaries.

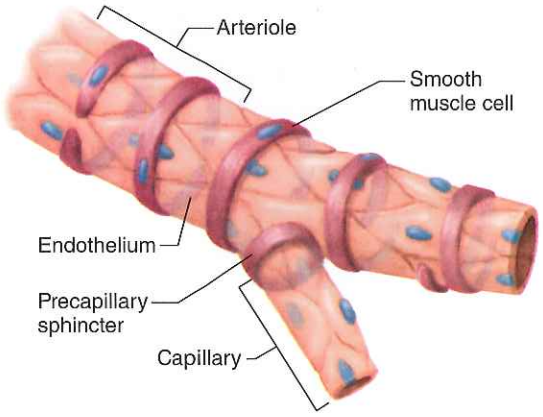


FIGURE 15.26 The smallest arterioles have only a few smooth muscle fibers in their walls. Capillaries lack these fibers.

The arteriole and metarteriole walls are adapted for vasoconstriction and vasodilation in that their muscle fibers respond to impulses from the autonomic nervous system by contracting or relaxing. In this way, these vessels help control the flow of blood into the capillaries.

A GLIMPSE AHEAD | **To Chapter 22**

Local vasodilation, increasing blood flow to the arteries associated with the genitalia, stiffens erectile tissue in both sexes.

In some places metarterioles connect directly to venules, and blood entering them can bypass the capillaries. These connections between arteriole and venous pathways, shown in figure 15.27, are called *arteriovenous shunts*.

PRACTICE

31 Describe the wall of an artery.

32 What is the function of the smooth muscle in the arterial wall?

33 How is the structure of an arteriole different from that of an artery?

Capillaries

Capillaries (kap′ĭ-ler″ēz), 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—a single layer

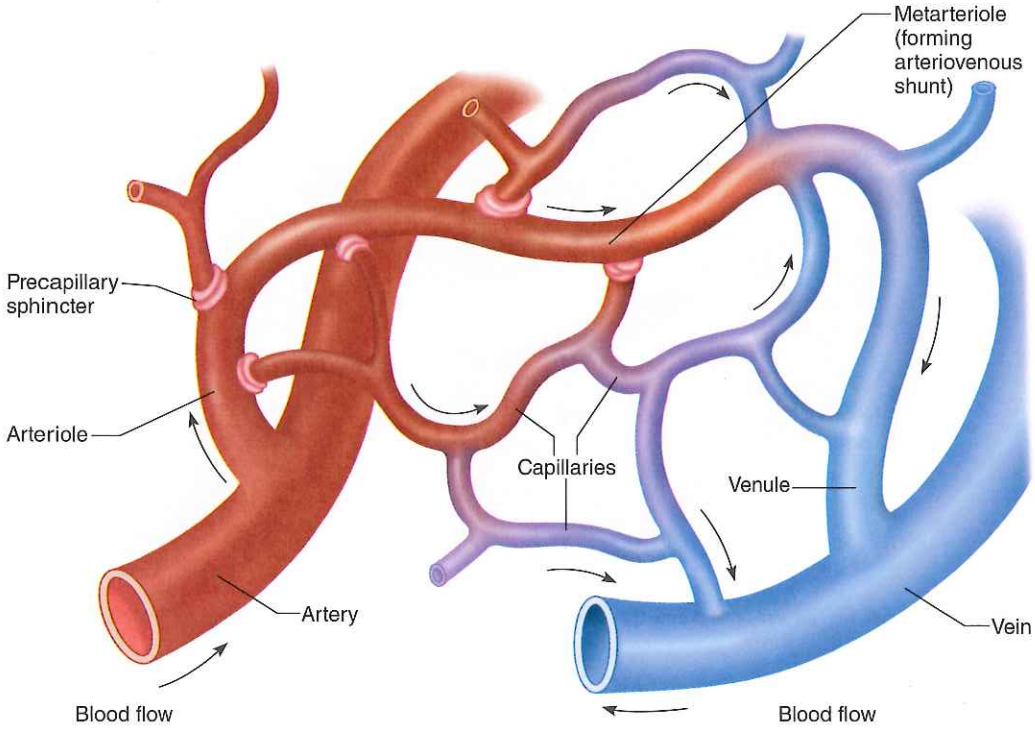
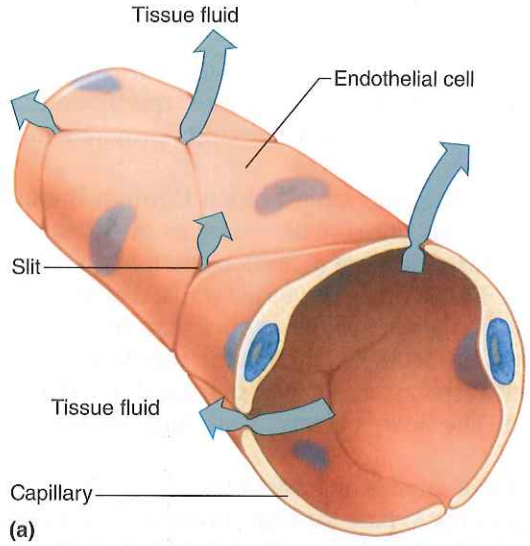


FIGURE 15.27 Some metarterioles provide arteriovenous shunts by connecting arterioles directly to venules.

of squamous epithelial cells (fig. 15.28*a*). These thin walls form the semipermeable layer through which substances in the blood are exchanged for substances in the tissue fluid surrounding body cells.



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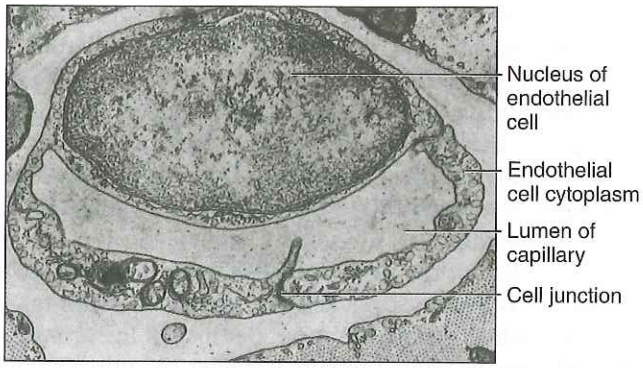

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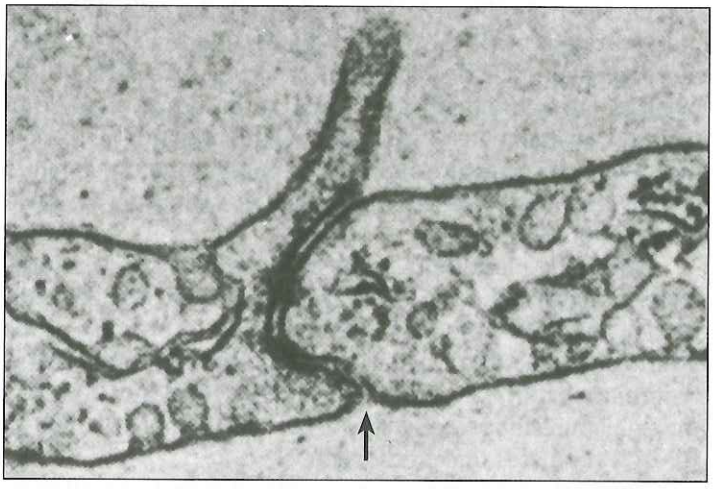


(c)

FIGURE 15.28 Capillary structure. (*a*) Substances are exchanged between the blood and tissue fluid through openings (slits) separating endothelial cells. (*b*) Transmission electron micrograph of a capillary cross section (11,500×). (*c*) Note the narrow slitlike openings at the cell junctions (arrow) (micrograph *b* enlarged to 62,500×).

Capillary Permeability

The openings or intercellular channels in the capillary walls are thin slits where endothelial cells overlap (fig. 15.28*b* and *c*). The sizes of these openings, and consequently the permeability of the capillary wall, vary from tissue to tissue. For example, the openings are relatively smaller in the capillaries of smooth, skeletal, and cardiac muscle than they are in capillaries associated with endocrine glands, the kidneys, and the lining of the small intestine.

Capillaries with the largest openings include those of the liver, spleen, and red bone marrow. These capillaries are discontinuous, and the distances between their cells appear as small cavities (sinusoids) in the organ. Discontinuous capillaries allow large proteins and even intact cells to pass through as they enter or leave the circulation. From Science to Technology 5.1 (p. 153) discusses the blood-brain barrier, which is formed by the protective tight capillaries in the brain. The blood-brain barrier is not present in the pituitary and pineal glands and parts of the hypothalamus.

Capillary Arrangement

The higher a tissue's rate of metabolism, the denser its capillary networks. Muscle and nerve tissues, which use abundant oxygen and nutrients, are richly supplied with capillaries. Tissues with slower metabolic rates have fewer capillaries, as in cartilage, or lack them entirely, as in the cornea.

> If the capillaries of an adult were unwound and lined up end to end, they would be between 25,000 and 60,000 miles long.

The spatial patterns of capillaries also differ in various body parts. For example, some capillaries pass directly from arterioles to venules, but others lead to highly branched networks (fig. 15.29). Such physical organization makes it

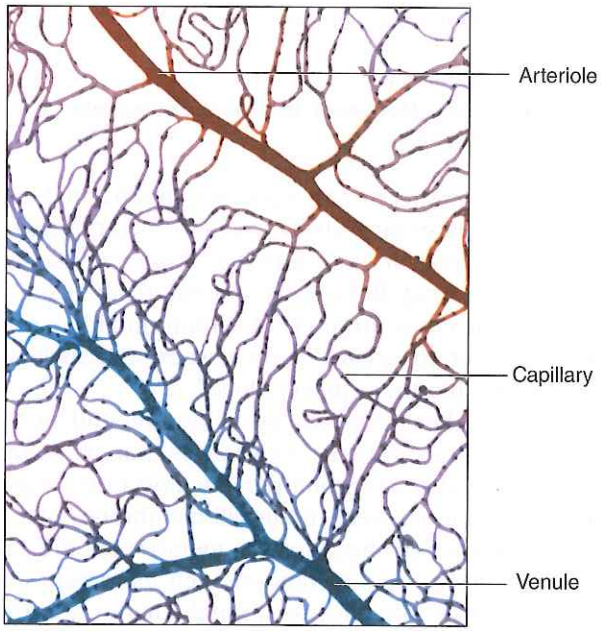


FIGURE 15.29 Light micrograph of a capillary network (100×).

possible for the blood to follow different pathways through a tissue attuned to cellular requirements.

Blood flow can vary among tissues as well. During exercise, for example, blood is directed into the capillary networks of the skeletal muscles, where the cells require more oxygen and nutrients. At the same time, the blood bypasses some of the capillary networks in the tissues of the digestive tract, where demand for blood is less critical. Conversely, when a person is relaxing after a meal, blood can be shunted from the inactive skeletal muscles into the capillary networks of the digestive organs.

Regulation of Capillary Blood Flow

The distribution of blood in the various capillary pathways is mainly regulated by the smooth muscles that encircle the capillary entrances. As figures 15.26 and 15.27 show, these muscles form *precapillary sphincters,* which may close a capillary by contracting or open it by relaxing. A precapillary sphincter responds to the demands of the cells the capillary supplies. When these cells have low concentrations of oxygen and nutrients, the precapillary sphincter relaxes, and blood flow increases; when cellular requirements have been met, the precapillary sphincter may contract again.

PRACTICE

34 Describe a capillary wall.

35 What is the function of a capillary?

36 What controls blood flow into capillaries?

Exchanges in the Capillaries

The vital function of exchanging gases, nutrients, and metabolic by-products between the blood and the tissue fluid surrounding the cells takes place in the capillaries. The biochemicals exchanged move through the capillary walls by diffusion, filtration, and osmosis.

RECONNECT

To Chapter 3, Movements Into and Out of the Cell, pages 100–103.

Diffusion is the most important means of transfer. 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.

The paths these substances follow depend primarily on their solubilities in lipids. Substances that are soluble in lipid, such as oxygen, carbon dioxide, and fatty acids, can diffuse through most areas of the cell membranes that make up the capillary wall because the membranes are largely lipid. Lipid-insoluble substances, such as water, sodium ions, and chloride ions, diffuse through channels in the cell membranes and through the slitlike openings between the endothelial cells that form the capillary wall (see fig. 15.28). Plasma proteins generally remain in the blood because they are not soluble in the lipid of the endothelial cell membranes, and they are too large to diffuse through the membrane channels or slitlike openings between the endothelial cells of most capillaries.

In *filtration, hydrostatic pressure* forces molecules through a membrane. In the 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. It is similarly greater at the arteriolar end of a capillary than at the venular end. The flow is always from high pressure to low pressure.

The walls of arteries and arterioles are too thick to allow blood components to pass through. However, the hydrostatic pressure of the blood pushes small molecules through capillary walls by filtration primarily at the arteriolar ends of capillaries, whereas diffusion takes place along their entire lengths.

The presence of an impermeant solute on one side of a cell membrane creates an osmotic pressure. Plasma proteins trapped in the capillaries create an osmotic pressure that draws water into the capillaries. The term *colloid osmotic pressure* describes this osmotic effect due solely to the plasma proteins.

The effect of the plasma colloid osmotic pressure, which favors reabsorption, opposes the action of capillary blood pressure, which favors filtration. Because at the arteriolar end of capillaries the blood pressure is higher (35 mm Hg outward) than the colloid osmotic pressure (24 mm Hg inward), filtration predominates here. At the venular end, the colloid osmotic pressure is essentially unchanged (24 mm Hg inward), but the blood pressure has decreased due to resistance through the capillary (16 mm Hg outward). Thus, at the venular end, reabsorption predominates (fig. 15.30). (The interstitial fluid also has hydrostatic pressure and osmotic pressure, but the values are low and tend to cancel each other; as such, they are omitted from this discussion.)

Normally, more fluid leaves the capillaries than returns to them because the net inward pressure at the venular ends of the capillaries is less than the net outward pressure at the arteriolar ends of the capillaries. Closed-ended vessels called lymphatic capillaries collect the excess fluid and return it through lymphatic vessels to the venous circulation. Chapter 16 (p. 620) discusses this mechanism.

Unusual events may increase blood flow to capillaries, causing excess fluid to enter the spaces between tissue cells (interstitial spaces). This may occur in response to certain chemicals, such as *histamine,* that vasodilate the metarterioles and increase capillary permeability. Enough fluid may

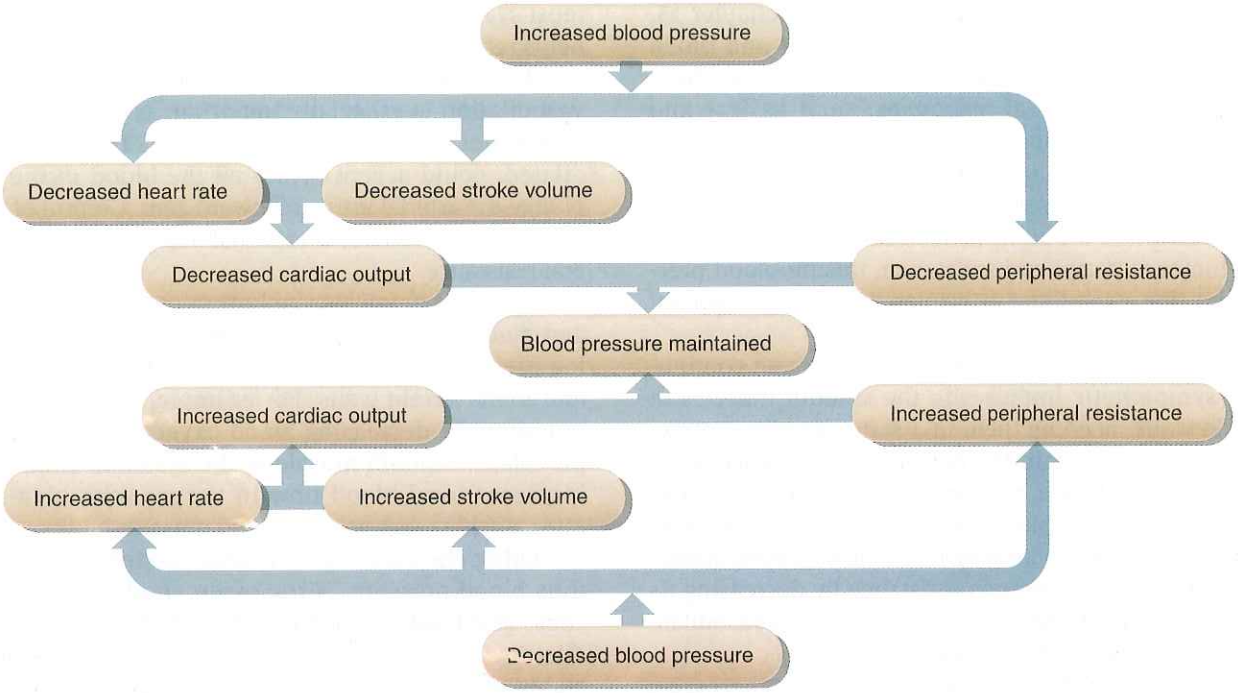


FIGURE 15.36 Controlling cardiac output and peripheral resistance regulates blood pressure.

the heart. The law becomes important, for example, during exercise, when venous return increases. The more blood that enters the heart from the veins, the greater the ventricular distension, the stronger the contraction, the greater the stroke volume, and the greater the cardiac output.

Conversely, the less blood that returns from the veins, the less the ventricle distends, the weaker the ventricular contraction, and the lesser the stroke volume and cardiac output. This mechanism helps to ensure that the volume of blood discharged from the heart is equal to the volume entering its chambers.

Some blood remains in the ventricles after contraction and stroke volume ejection. This ESV is influenced by preload, contractility of the ventricle, and afterload. **Contractility,** the amount of force produced during a contraction at a given preload (EDV), is influenced by autonomic innervation and hormones (epinephrine, norepinephrine, thyroid hormones). Sympathetic stimulation contracts the ventricles more forcefully, increasing the volume ejected and decreasing the ESV. Decreased sympathetic stimulation produces the opposite effect. The amount of force that the ventricles must produce to open the semilunar valves to eject blood is the **afterload.** Increased arterial pressure (hypertension), especially the diastolic pressure, increases afterload. As the afterload increases, the heart must work harder to eject blood.

Recall from chapter 12 (p. 445) that baroreceptors in the walls of the aortic arch and carotid sinuses sense changes in blood pressure. If arterial pressure increases, impulses travel from the receptors to the *cardiac center* of the medulla oblongata. This center relays parasympathetic impulses to the SA node in the heart, and heart rate decreases in response. As a result of this *cardioinhibitor reflex,* cardiac output falls and blood pressure decreases toward the normal level. Figure 15.37 summarizes this mechanism.

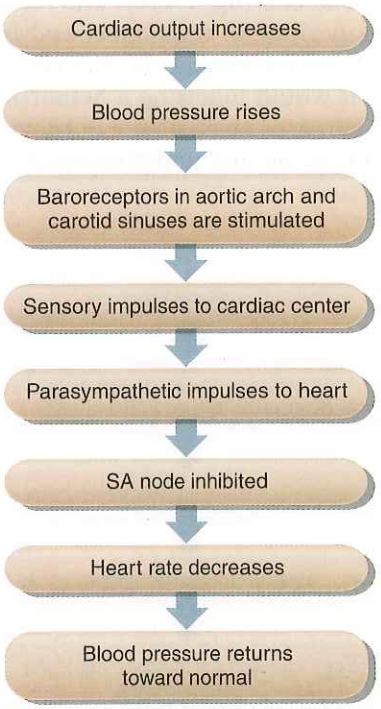


FIGURE 15.37 If blood pressure rises, baroreceptors initiate the cardioinhibitor reflex, which lowers the blood pressure.

Conversely, decreasing arterial blood pressure initiates the *cardioaccelerator reflex,* which sends sympathetic impulses to the SA node. As a result, the heart beats faster, increasing cardiac output and arterial pressure.

Recall that epinephrine increases heart rate (chapter 13, p. 510) and consequently alters cardiac output and blood pressure. Other factors that increase heart rate and blood pressure include emotional responses, such as fear and anger; physical exercise; and a rise in body temperature.

Changes in arteriole diameters regulate peripheral resistance. Because blood vessels with smaller diameters offer a greater resistance to blood flow, factors that vasoconstrict arterioles increase peripheral resistance, raising blood pressure; factors that vasodilate arterioles decrease peripheral resistance, lowering blood pressure.

The *vasomotor center* of the medulla oblongata continually sends sympathetic impulses to the 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. The vasomotor center responds to changes in blood pressure, so 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 decreases.

Whenever arterial blood pressure suddenly increases, baroreceptors in the aortic arch and carotid sinuses signal the vasomotor center, and the sympathetic outflow to the arteriole walls falls (fig. 15.38). The resulting vasodilation decreases peripheral resistance, and blood pressure decreases toward the normal level. Similarly, if blood pressure drops, as it would following a hemorrhage, the vasomotor center increases sympathetic outflow. The resulting release of epinephrine and norepinephrine vasoconstricts most systemic vessels, increasing peripheral resistance. This helps return blood pressure toward normal.

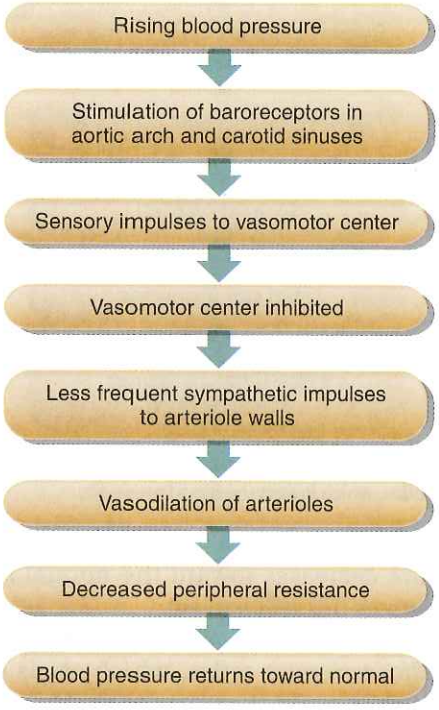


FIGURE 15.38 Dilating arterioles helps regulate blood pressure.

The vasomotor center's control of vasoconstriction and vasodilation is especially important in the arterioles of the *abdominal viscera* (splanchnic region). These vessels, if fully dilated, could accept nearly all the blood of the body and plunge the arterial pressure toward zero. Control of the diameters of these arterioles is essential in regulating normal peripheral resistance.

Certain chemicals, including carbon dioxide, oxygen, and hydrogen ions, influence peripheral resistance by affecting precapillary sphincters and smooth muscles in arteriole and metarteriole 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.

Other chemicals also influence peripheral resistance and thus blood pressure. Nitric oxide, produced by endothelial cells, and bradykinin, formed in the blood, are both vasodilators. Angiotensin II plays a role in vasoconstriction; and endothelin, released by cells of the endothelium, is a powerful vasoconstrictor. Clinical Application 15.4 discusses high blood pressure.

PRACTICE

50 What factors affect cardiac output?

51 Explain the Frank-Starling law of the heart.

52 What is the function of the baroreceptors in the walls of the aortic arch and carotid sinuses?

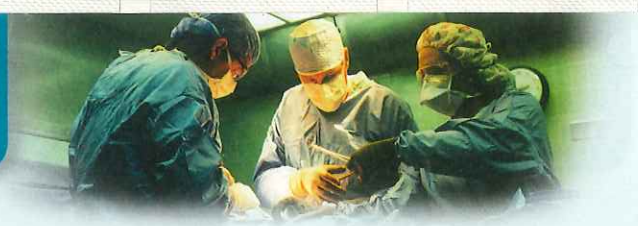
53 How does the vasomotor center control peripheral resistance?

Venous Blood Flow

Blood pressure decreases as blood moves through the arterial system and into the capillary networks, so little pressure remains at the venular ends of capillaries (see fig. 15H). 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. For example, contracting skeletal muscles press on veins, moving blood from one valve section to another. This contraction of skeletal muscles helps push the blood through the venous system toward the heart (fig. 15.39).

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

15.4 CLINICAL APPLICATION



Hypertension

Hypertension, or high blood pressure, is persistently elevated arterial pressure. It is a prevalent disease of the cardiovascular system in industrialized nations.

High blood pressure with unknown cause is called *essential* (also primary or idiopathic) *hypertension.* Elevated blood pressure that is a consequence of another problem, such as arteriosclerosis or kidney disease, is called *secondary hypertension.*

Arteriosclerosis is accompanied by decreased elasticity of the arterial walls and narrowed vessel lumens, which raise blood pressure. Kidney diseases often produce changes that interfere with blood flow to kidney cells. In response, the affected tissues may release an enzyme called *renin* that leads to the production of *angiotensin II,* a powerful vasoconstrictor that increases peripheral resistance in the arterial system, raising arterial pressure (fig. 15I). Angiotensin II also stimulates the adrenal cortex to release *aldosterone,* which stimulates the kidneys to retain sodium ions and water. The resulting increase in blood volume contributes to increased blood pressure. Normally, this mechanism ensures that a decrease in blood flow to the kidneys is followed by an increase in arterial pressure, which, in turn, restores blood flow to the kidneys. In some individuals, high sodium intake leads to vasoconstriction, raising blood pressure. Obesity also is a risk factor for hypertension because it increases peripheral resistance. Psychological stress, which activates sympathetic nerve impulses that cause generalized vasoconstriction, may also lead to hypertension. Yet another cause of hypertension may be an inability of endothelium to respond to a relaxing factor, leading to vasoconstriction.

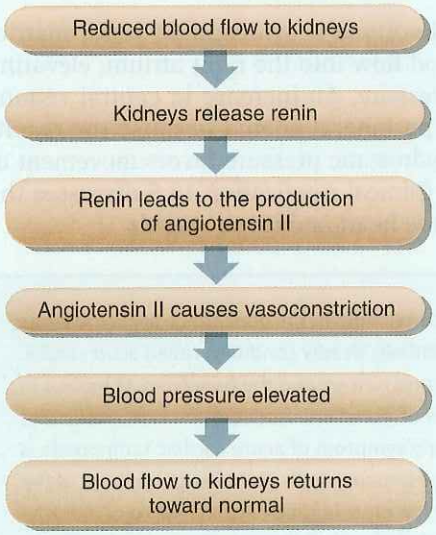


FIGURE 15I Renin stimulates production of angiotensin II, which elevates blood pressure.

Hypertension is called a "silent killer" because it may not have direct symptoms, yet can raise the risk for serious cardiovascular complications. For example, as the left ventricle works harder to pump blood at a higher pressure, the myocardium thickens, enlarging the heart. If the coronary blood vessels cannot support this overgrowth, parts of the heart muscle die and fibrous tissue replaces them. Eventually, the enlarged and weakened heart fails to maintain adequate output for survival.

Hypertension also contributes to the development of atherosclerosis. As arteries accumulate plaque, a *coronary thrombosis* or a *coronary embolism* may occur. Similar changes in the arteries of the brain increase the chances of a *cerebral vascular accident* (CVA), or stroke, due to a cerebral thrombosis, embolism, or hemorrhage. When an embolus or hemorrhage causes a stroke, paralysis and other functional losses suddenly appear. A thrombus-caused stroke is slower. It may begin with clumsiness, progress to partial visual loss, then affect speech. One arm becomes paralyzed, then a day later, perhaps an entire side of the body is affected. Table 15A lists risk factors for a stroke.

A *transient ischemic attack* (TIA, or "ministroke") is a temporary block in a small artery. Symptoms include difficulty in speaking or understanding speech; numbness or weakness in the face, upper limb, lower limb, or one side; dizziness; falling; an unsteady gait; blurred vision; or blindness. These symptoms typically resolve within twenty-four hours with no lasting effects, but may be a warning of an impending, more serious stroke.

Treatment of hypertension varies because the cause varies. Treatment usually includes exercising regularly, controlling weight, reducing stress, and limiting dietary sodium. Drugs, such as diuretics and/or inhibitors of sympathetic nerve activity, may help control blood pressure. Diuretics increase urinary excretion of sodium and water, reducing the volume of body fluids. Sympathetic inhibitors block the synthesis of neurotransmitters, such as norepinephrine, or block receptor sites of effector cells. Table 15B describes how drugs that treat hypertension work. ■

TABLE 15A | Risk Factors for Stroke

Alcohol consumption
Diabetes
Elevated serum cholesterol
Family history of cardiovascular disease
Hypertension
Smoking
Transient ischemic attacks

TABLE 15B | Drugs to Treat Hypertension

Type of Drug	Mechanism of Action
Angiotensin-converting enzyme (ACE) inhibitors	Block formation of angiotensin II, preventing vasoconstriction
Beta blockers	Lower heart rate, reduce contractility
Calcium channel blockers	Dilate blood vessels by keeping calcium ions out of smooth muscle cells in vessel walls
Diuretics	Increase urine output, lowering blood volume

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 (see fig. 15.32). If some blood is lost and blood pressure falls, venoconstriction can force blood out of this reservoir, returning venous blood to the heart. By maintaining venous return, venoconstriction helps to maintain blood pressure.

Central Venous Pressure

All veins, except those returning to the heart from the lungs, drain into the right atrium. Therefore, the pressure in this heart chamber is called *central venous pressure.* It affects the pressure in the peripheral veins. For example, if the heart is beating weakly, the central venous pressure increases and blood backs up in the venous network, raising its pressure too. However, if the heart is beating forcefully, the central venous pressure and the pressure in the venous network decrease.

15.5 CLINICAL APPLICATION

Exercise and the Cardiovascular System

We know that exercise is good for the heart. Yet each year, a few individuals die of sudden cardiac arrest while shoveling snow, running, or engaging in some other strenuous activity. The explanation for this apparent paradox is that exercise is good for the heart—but only if it is a regular part of life.

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. These adaptations improve oxygen delivery to, and use by, muscle tissue.

An athlete's heart typically changes in response to these increased demands and may enlarge 40% or more. Myocardial mass increases, the ventricular cavities expand, and the ventricle walls thicken. At rest, stroke volume increases, and heart rate and blood pressure decrease. To a physician unfamiliar with a conditioned cardiovascular system, a trained athlete may appear to be 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 when a person who never exercises suddenly shovels snow or runs 3 miles. Sedentary people have 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% to 85% of its "theoretical maximum" for 30 to 60 minutes at least three to four times a week, according to the American Heart Association. 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. A rate of 141–172 beats per minute is 70% to 85% of this value. Examples of activities that raise 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 the age of thirty are advised to have a stress test, which is an electrocardiogram taken while exercising. (The standard electrocardiogram is taken at rest.) An arrhythmia that appears only during exercise may indicate heart disease that has not yet produced symptoms.

The American Heart Association suggests that after a physical exam, a sedentary person wishing to start an exercise program begin with 30 minutes of activity (perhaps broken into two 15-minute sessions at first) at least five times per week. ■

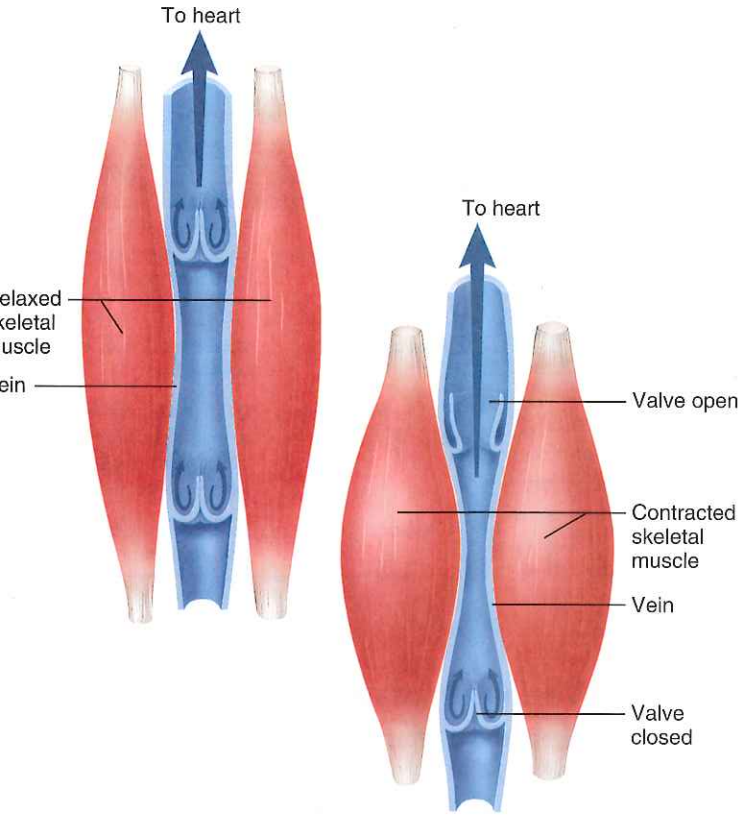


FIGURE 15.39 The massaging action of skeletal muscles helps move blood through the venous system toward the heart.

Increase in blood volume or widespread venoconstriction also increase blood flow into the right atrium, elevating the central venous pressure. An increase in central venous pressure can lead to peripheral edema because the resulting higher capillary hydrostatic pressure favors movement of fluid into the tissues. Clinical Application 15.5 discusses the effects of exercise on the heart and blood vessels.

Blood or tissue fluid accumulating in the pericardial cavity increases pressure, causing a potentially deadly condition called *acute cardiac tamponade*. Increasing pressure around the heart may compress it, interfering with the flow of blood into its chambers and preventing pumping action. An early symptom of acute cardiac tamponade is increased central venous pressure, with visible engorgement of the veins in the neck. Other symptoms include anxiety, rapid or difficulty breathing, light-headedness, palpitations, pallor, and chest pain. Acute cardiac tamponade has several causes, including bacterial or viral infection, injury, acute myocardial infarction, advanced lung cancer, and dissecting aortic aneurysm.

PRACTICE

54 What is the function of the venous valves?

55 How do skeletal muscles affect venous blood flow?

56 How do respiratory movements affect venous blood flow?

57 What factors stimulate venoconstriction?

15.6 PATHS OF CIRCULATION

Recall from figure 15.1 that the blood vessels can be divided into two major pathways. The *pulmonary circuit,* or pulmonary circulation, consists of vessels that carry blood from the heart to the lungs and back to the heart. The *systemic circuit,* or systemic circulation, carries blood from the heart to all other parts of the body and back again. The systemic circuit includes the coronary circulation.

The pathways described in the following sections are those of an adult. Chapter 23 (pp. 887–889) describes the somewhat different fetal pathways.

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, the pulmonary trunk divides into the right and left pulmonary arteries, which penetrate the right and left lungs, respectively. In the lungs, they diverge into *lobar branches* (three on the right side and two on the left) that accompany the main divisions of the bronchi (airways) into the lobes of the lungs. After repeated divisions, the lobar branches give rise to arterioles that continue into the capillary networks associated with the walls of the alveoli (air sacs) (fig. 15.40).

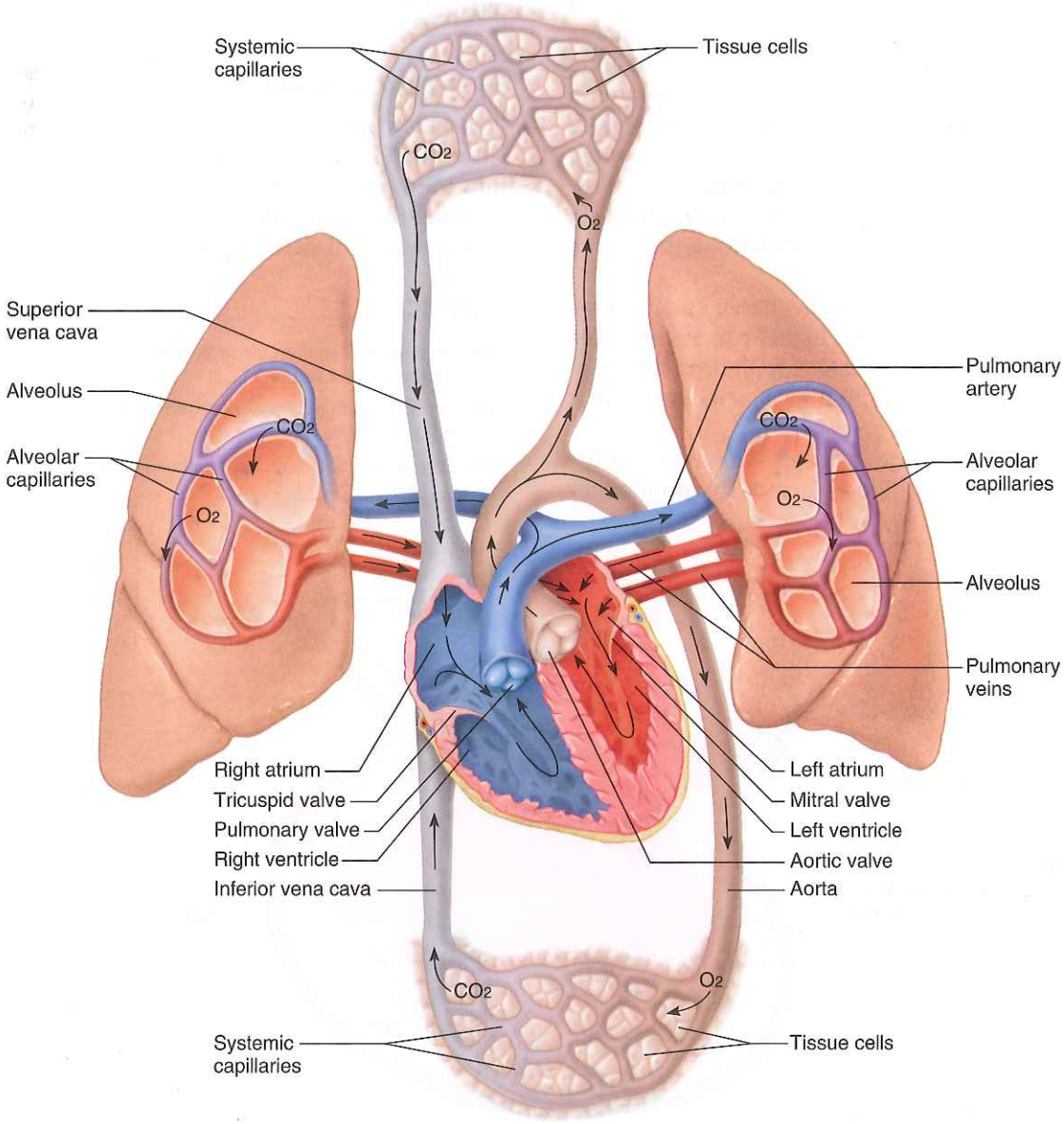


FIGURE 15.40 AP|R Blood reaches the lungs through branches of the pulmonary arteries, and it returns to the heart through pulmonary veins. (Structures are not drawn to scale.)

The blood in the arteries and arterioles of the pulmonary circuit is low in oxygen and high in carbon dioxide. Gases are exchanged between the blood and the air as the blood moves through the *alveolar capillaries*, discussed in chapter 19 (pp. 755–756).

The right ventricle contracts with less force than the left ventricle. Therefore, the arterial pressure in the pulmonary circuit is less than that in the systemic circuit. As a result, the alveolar capillary pressure is low.

The force that moves fluid out of an alveolar capillary is 23 mm Hg; the force pulling fluid into it is 22 mm Hg. Thus, such a capillary has a net filtration pressure of 1 mm Hg. This pressure propels a slight, continuous flow of fluid into the narrow interstitial space between the alveolar capillary and the alveolus.

The epithelial cells of the alveoli are so tightly joined that sodium, chloride, and potassium ions, as well as glucose and urea, enter the interstitial space but usually do not enter the alveoli. This helps maintain a high osmotic pressure in the interstitial fluid. Consequently, osmosis rapidly moves any water that gets into the alveoli back into the interstitial space. Although the alveolar surface must be moist to allow diffusion of oxygen and carbon dioxide, this mechanism keeps excess water out of the alveoli, preventing them from filling with fluid (fig. 15.41).

Fluid in the interstitial space may be drawn back into the alveolar capillaries by the somewhat higher osmotic pressure of the blood. Alternatively, lymphatic capillaries (see chapter 16, pp. 617–618) may return fluid to the circulation (fig. 15.41).

As a result of gas exchange between the blood and the alveolar air, blood entering the venules of the pulmonary circuit is rich in oxygen and low in carbon dioxide. These venules merge to form small veins, and these veins in turn converge to form larger veins. Four *pulmonary veins*, two from each lung, return blood to the left atrium. This completes the vascular loop of the pulmonary circuit.

> *Pulmonary edema*, in which lungs fill with fluid, can accompany a failing left ventricle or a damaged mitral valve. A weak left ventricle may be unable to move the normal volume of blood into the systemic circuit. Blood backing up into the pulmonary circuit increases pressure in the alveolar capillaries, flooding the interstitial spaces with fluid. Increasing pressure in the interstitial fluid may rupture the alveolar membranes, and fluid may enter the alveoli more rapidly than it can be removed. This reduces the alveolar surface available for gas exchange, and the person may suffocate.

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, which includes the aorta and its branches that lead to all of the body tissues, as well as the companion system of veins that returns blood to the right atrium.

PRACTICE

58 Distinguish between the pulmonary and systemic circuits of the cardiovascular system.

59 Trace the path of blood through the pulmonary circuit from the right ventricle.

60 Explain why the alveoli normally do not fill with fluid.

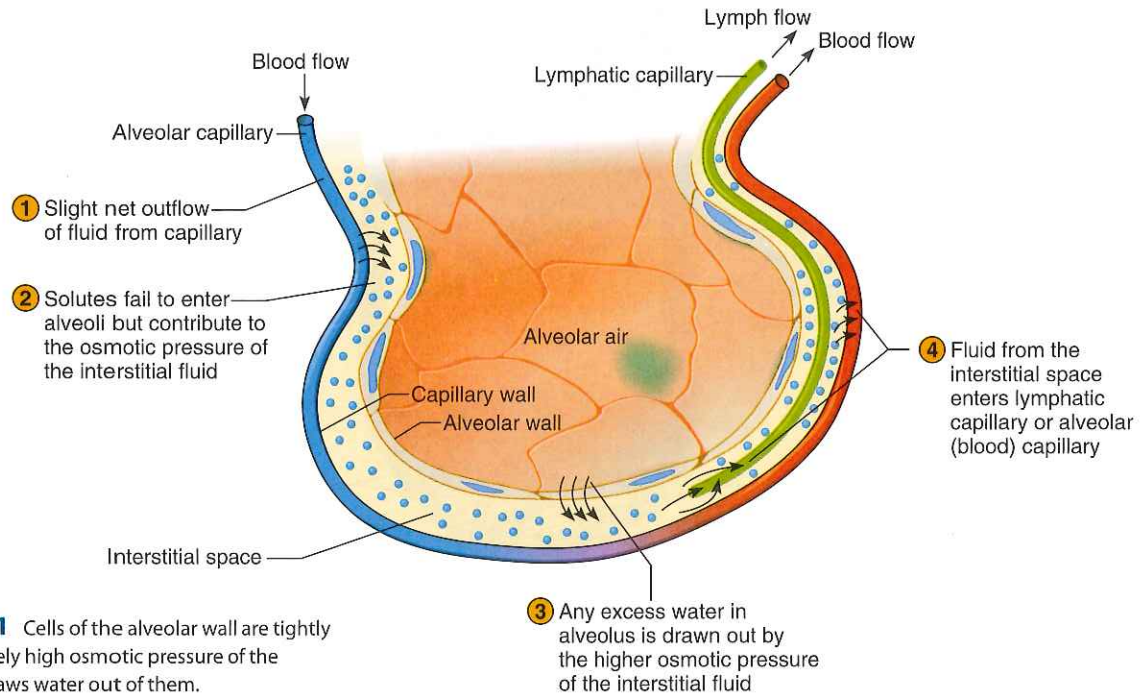


FIGURE 15.41 Cells of the alveolar wall are tightly joined. The relatively high osmotic pressure of the interstitial fluid draws water out of them.

15.7 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.

Principal Branches of the Aorta

The part of the aorta attached to the heart is called the aortic root. From there, the first part of the aorta is called the *ascending aorta.* At the root 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. The elastic recoil of the aortic wall following contraction of the left ventricle helps drive blood flow into these arteries.

Three major arteries originate from the *arch of the aorta* (aortic arch). They are the brachiocephalic artery, the left common carotid artery, and the left subclavian artery. The aortic arch has baroreceptors that detect changes in blood pressure. Several small structures called **aortic bodies** lie in the epithelial lining of the aortic arch. These bodies house chemoreceptors that sense blood levels of oxygen and carbon dioxide.

The **brachiocephalic** (brak″e-o-sĕ-fal′ik) **artery** supplies blood to the tissues of the upper limb and head, as its name suggests. It is the first branch from the aortic arch and rises through the mediastinum to a point near the junction of the sternum and the right clavicle. There it divides, giving rise to the right **common carotid** (kah-rot′id) **artery,** which carries blood to the right side of the neck and head, and the right **subclavian** (sub-kla′ve-an) **artery,** which leads into the right arm. Branches of the subclavian artery also supply blood to parts of the shoulder, neck, and head.

The left *common carotid artery* and the left *subclavian artery* are respectively the second and third branches of the aortic arch. They supply blood to regions on the left side of the body corresponding to those supplied by their counterparts on the right (fig. 15.42 and reference plates 21, 22, and 23, pp. 54–55).

The upper part of the *descending aorta* is left of the midline, but it gradually moves medially and lies directly anterior to the vertebral column at the level of the twelfth thoracic vertebra. The part of the descending aorta above the diaphragm is the **thoracic aorta** (tho-ras′ik a-or′tah). It gives off many small branches to the thoracic wall and the thoracic viscera. These branches, the *bronchial, pericardial,* and *esophageal arteries,* supply blood to the structures for which they were named. Other branches become *mediastinal arteries,*

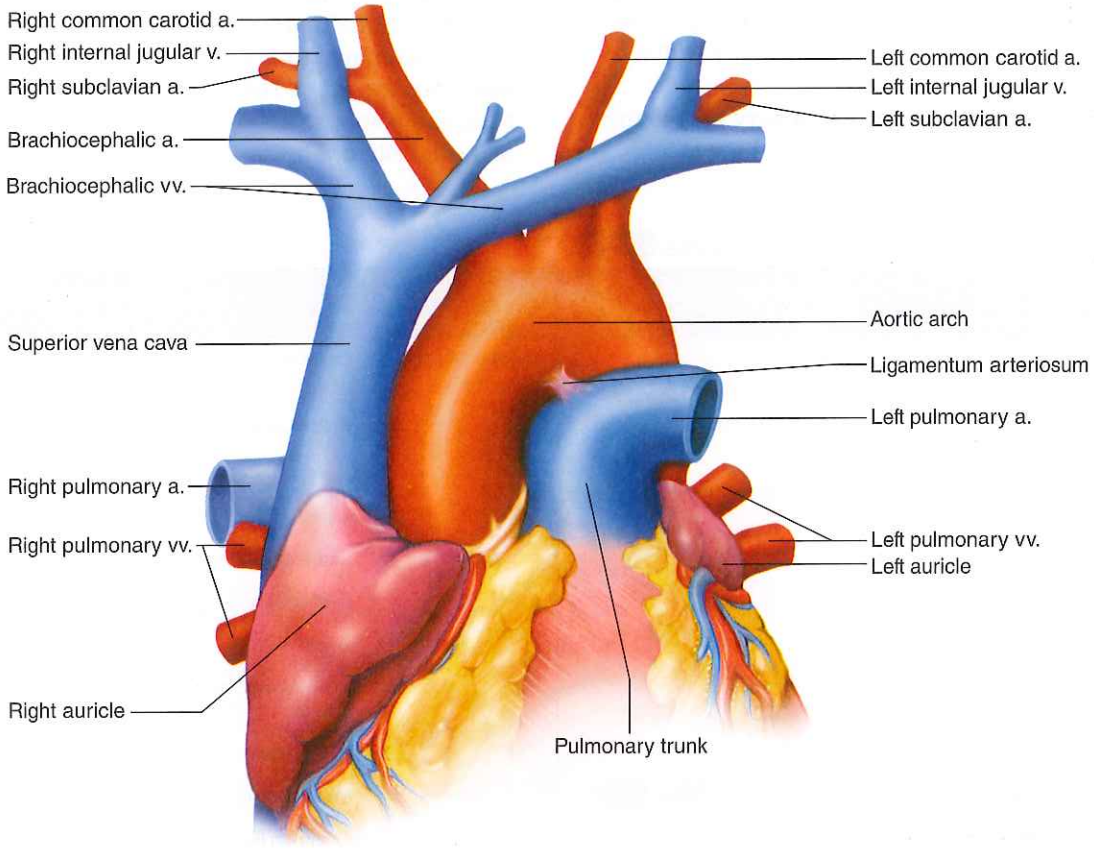


FIGURE 15.42 AP|R The major blood vessels associated with the heart. (*a.* stands for artery, *v.* stands for vein, *vv.* stands for veins.)

supplying various tissues in the mediastinum, and *posterior intercostal arteries,* which pass into the thoracic wall.

Below the diaphragm, the descending aorta becomes the **abdominal aorta,** and it branches to the abdominal wall and several abdominal organs. These branches include the following:

1. The **celiac** (se'le-ak) **artery** gives rise to the left *gastric, splenic,* and *hepatic arteries,* which supply upper portions of the digestive tract, the spleen, and the liver, respectively. (*Note:* The hepatic artery supplies the liver with about one-third of its blood flow, and this blood is oxygen-rich. The remaining two-thirds of the liver's blood flow arrives by means of the hepatic portal vein and is oxygen-poor.)
2. The **phrenic** (fren'ik) **arteries** are paired arteries that supply blood to the diaphragm.
3. The **superior mesenteric** (mes"en-ter'ik) **artery** is a large, unpaired vessel that branches to many parts of the intestinal tract, including the jejunum, ileum, cecum, ascending colon, and transverse colon.
4. The pair of **suprarenal** (soo"prah-re'nal) **arteries** supplies blood to the adrenal glands.
5. The **renal** (re'nal) **arteries** pass laterally from the aorta into the kidneys. Each artery then divides into several lobar branches in the kidney tissues.
6. **Gonadal** (go'nad-al) **arteries** are in the female and male. In a female, paired *ovarian arteries* arise from the aorta and pass into the pelvis to supply the ovaries. In a male, *spermatic arteries* originate in similar locations. They course downward and pass through the body wall by way of the *inguinal canal* to supply the testes.
7. Branches of the **inferior mesenteric artery** lead to the descending colon, the sigmoid colon, and the rectum.
8. Three or four pairs of **lumbar arteries** arise from the posterior surface of the aorta in the region of the lumbar vertebrae. These arteries supply muscles of the skin and the posterior abdominal wall.
9. The **middle sacral artery,** a small vessel, descends medially from the aorta along the anterior surfaces of the lower lumbar vertebrae. It carries blood to the sacrum and coccyx.

The abdominal aorta terminates near the brim of the pelvis, where it divides into right and left *common iliac arteries.* These vessels supply blood to lower regions of the abdominal wall, the pelvic organs, and the lower extremities (fig. 15.43). Table 15.4 summarizes the major branches of the aorta.

Arteries to the Brain, Head, and Neck

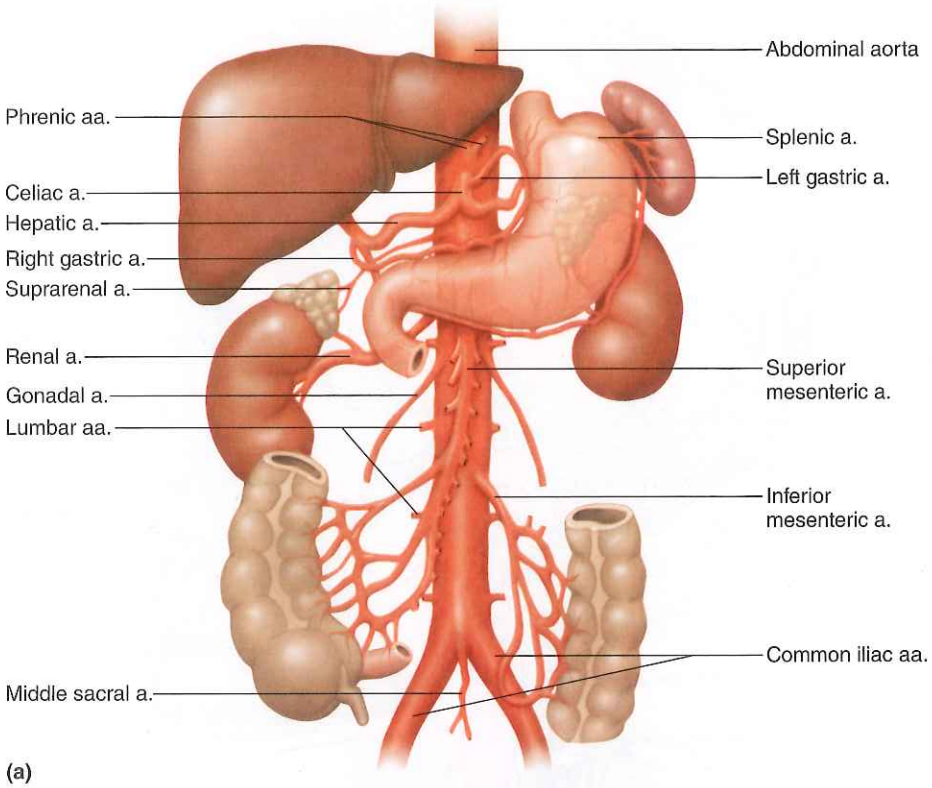
Branches of the subclavian and common carotid arteries supply blood to structures in the brain, head, and neck (figs. 15.44 and 15.45). The main divisions of the subclavian artery to these regions are the vertebral, thyrocervical, and costocervical arteries. The common carotid artery communicates with these regions by means of the internal and external carotid arteries.

The **vertebral arteries** arise from the subclavian arteries in the base of the neck near the tips of the lungs. They pass upward through the foramina of the transverse processes of the cervical vertebrae and enter the skull by way of the foramen magnum. Along their paths, these vessels supply blood to vertebrae and to their associated ligaments and muscles.

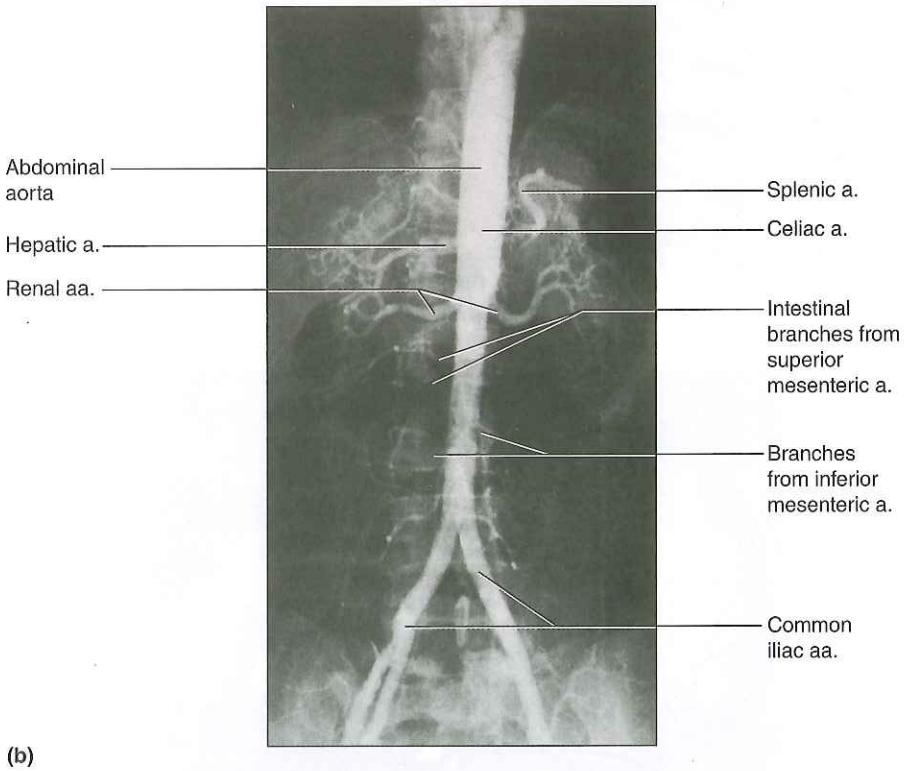
In the cranial cavity, the vertebral arteries unite to form a single *basilar artery.* This vessel passes along the ventral brainstem and gives rise to branches leading to the pons, midbrain, and cerebellum. The basilar artery terminates by dividing into

TABLE 15.4 | Major Branches of the Aorta AP|R

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



(a)



(b)

FIGURE 15.43 AP|R Abdominal aorta. (*a*) Its major branches. (*b*) Angiogram (radiograph). (*a.* stands for artery, *aa.* stands for arteries.)

FIGURE 15.44 AP|R The major arteries of the head and neck. The clavicle has been removed. (*a.* stands for artery.)

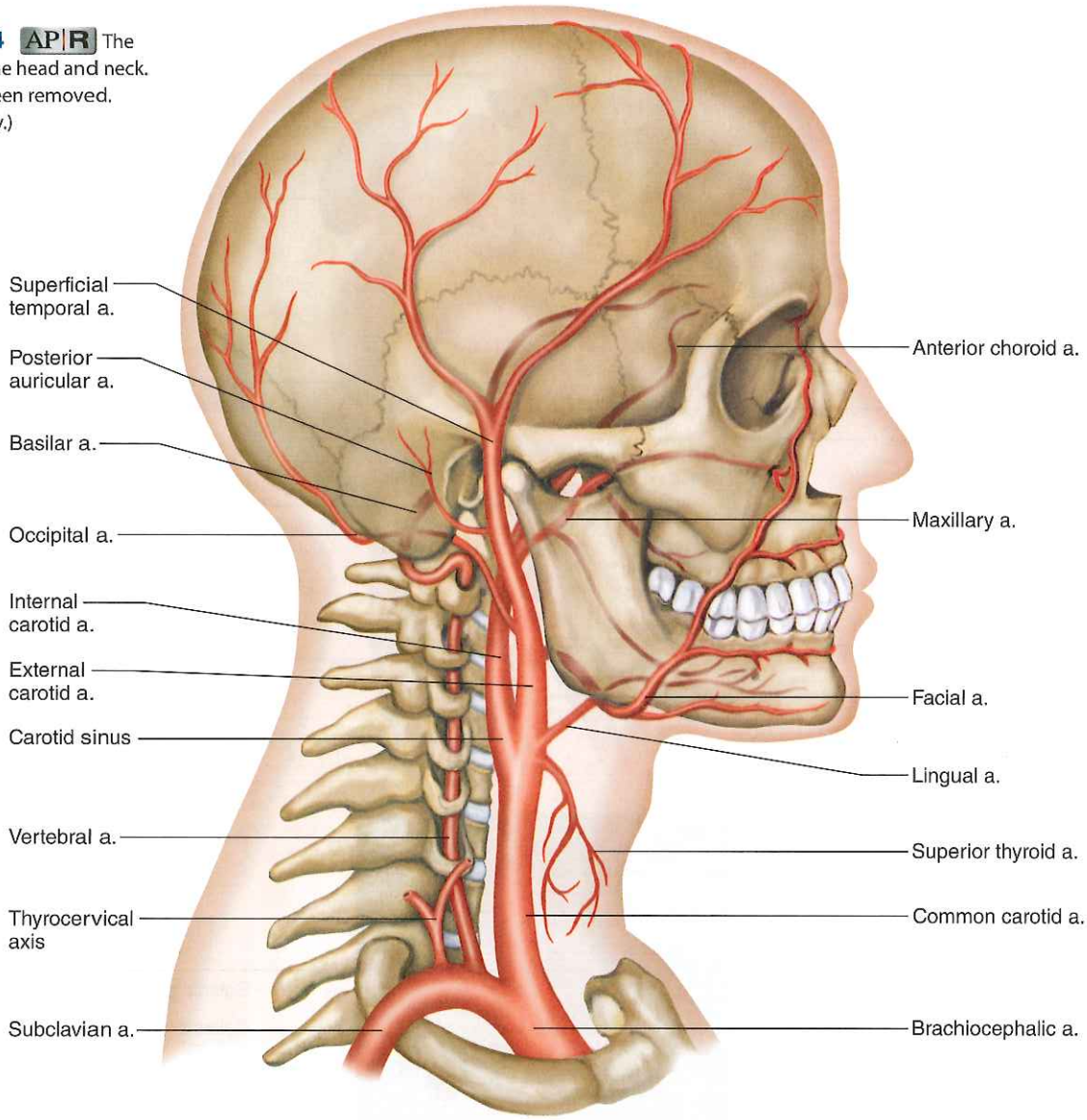
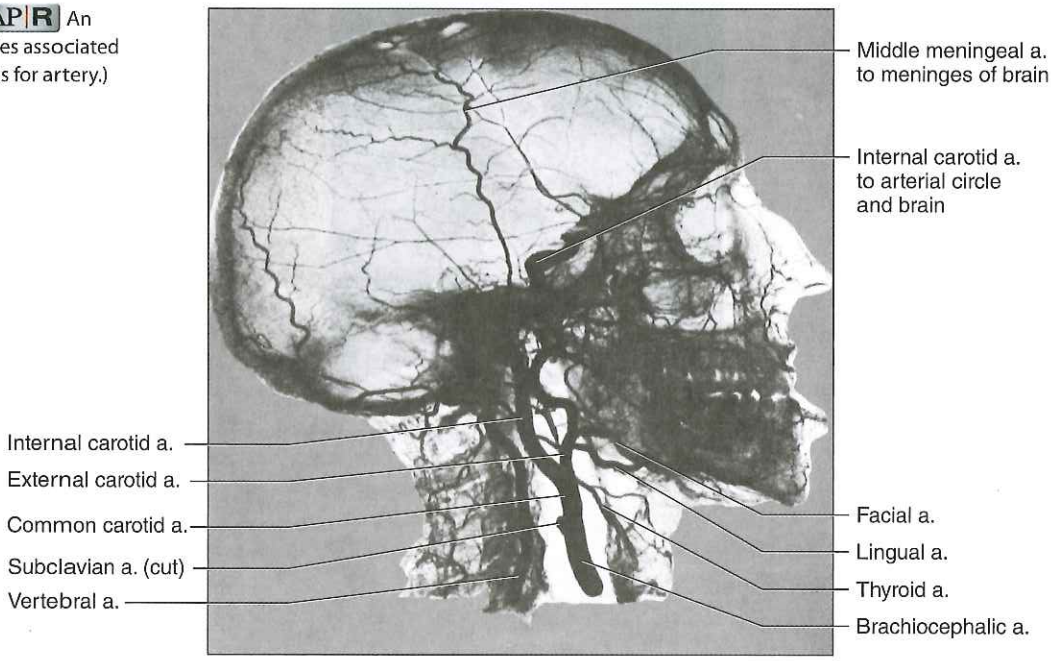


FIGURE 15.45 AP|R An angiogram of the arteries associated with the head. (*a.* stands for artery.)



two *posterior cerebral arteries* that supply parts 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. 15.46). The union of these systems provides alternate pathways for blood to circumvent blockages and reach brain tissues. It also equalizes blood pressure in the brain's blood supply. The circle is complete in only 20% to 30% of the population.

The **thyrocervical** (thi″ro-ser′vĭ-kal) **arteries** are short vessels that give off branches at the thyrocervical axis to the thyroid gland, parathyroid glands, larynx, trachea, esophagus, and pharynx, as well as to various muscles in the neck, shoulder, and back. The **costocervical** (kos″to-ser′vĭ-kal) **arteries,** the third vessels to branch from the subclavians, carry blood to muscles in the neck, back, and thoracic wall.

The left and right *common carotid arteries* ascend deeply in the neck on either side. At the level of the upper laryngeal border, they divide to form 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 main vessels that originate from this artery include the following:

1. *superior thyroid artery* to the hyoid bone, larynx, and thyroid gland
2. *lingual artery* to the tongue, muscles of the tongue, and salivary glands beneath the tongue
3. *facial artery* to the pharynx, palate, chin, lips, and nose
4. *occipital artery* to the scalp on the back of the skull, the meninges, the mastoid process, and various muscles in the neck
5. *posterior auricular artery* to the ear and the scalp over the ear

The external carotid artery terminates by dividing into *maxillary* and *superficial temporal arteries.* The maxillary artery supplies blood to the teeth, gums, jaws, cheek, nasal cavity, eyelids, and meninges. The superficial temporal artery extends to the parotid salivary gland and to various surface regions of the face and scalp.

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. The major branches of the internal carotid artery include the following:

1. *ophthalmic artery* to the eyeball and to various muscles and accessory organs within the orbit
2. *posterior communicating artery* that forms part of the cerebral arterial circle
3. *anterior choroid artery* to the choroid plexus within the lateral ventricle of the brain and to nerve structures in the brain

The internal carotid artery terminates by dividing into *anterior* and *middle cerebral arteries.* The middle cerebral artery passes through the lateral tissue and supplies the lateral surface of the cerebrum, including the primary motor and sensory areas of the face and upper limbs, the optic radiations, and the speech area (see chapter 11, pp. 407–410). The anterior cerebral artery extends anteriorly between the cerebral hemispheres and supplies the medial surface of the brain.

Near the base of each internal carotid artery is an enlargement called a **carotid sinus.** Like the aortic sinuses, these structures contain baroreceptors that control blood pressure. A number of small epithelial masses, called **carotid bodies,** are also in the wall of the carotid sinus. These bodies are vascular and have chemoreceptors that act with those of the aortic bodies to regulate circulation and respiration.

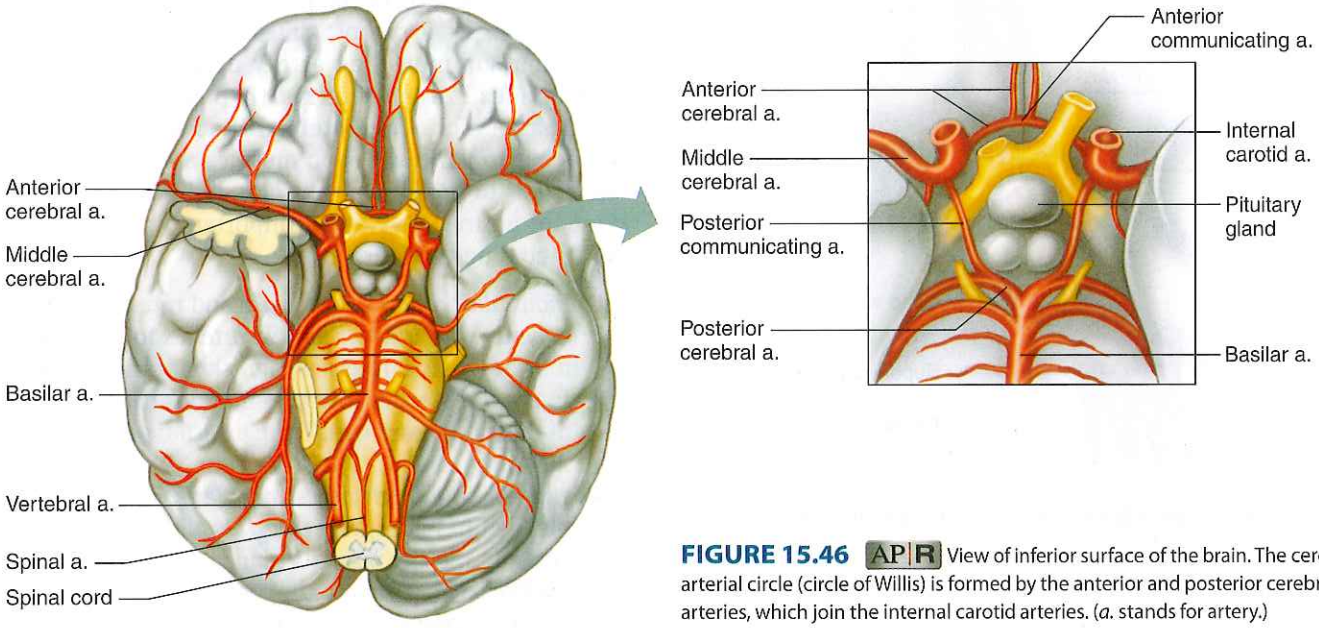


FIGURE 15.46 AP|R View of inferior surface of the brain. The cerebral arterial circle (circle of Willis) is formed by the anterior and posterior cerebral arteries, which join the internal carotid arteries. (*a.* stands for artery.)

Arteries to the Shoulder and Upper Limb

The subclavian artery, after giving off branches to the neck, continues into the arm (fig. 15.47). 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 the chest wall, including the skin of the shoulder; part of the mammary gland; the upper end of the humerus; the shoulder joint; and muscles in the back, shoulder, and chest. As this vessel leaves the axilla, it becomes the brachial artery.

The **brachial artery** courses along the humerus to the elbow. It gives rise to a *deep brachial artery* that curves posteriorly around the humerus and supplies the triceps brachii muscle. Shorter branches pass into the muscles on the anterior side of the arm, whereas others descend on each side to the elbow and connect with arteries in the forearm. The resulting arterial network allows blood to reach the forearm even if a portion of the distal brachial artery becomes obstructed.

In 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 join the anastomosis around the elbow joint, whereas others supply blood to flexor and extensor muscles in the forearm.

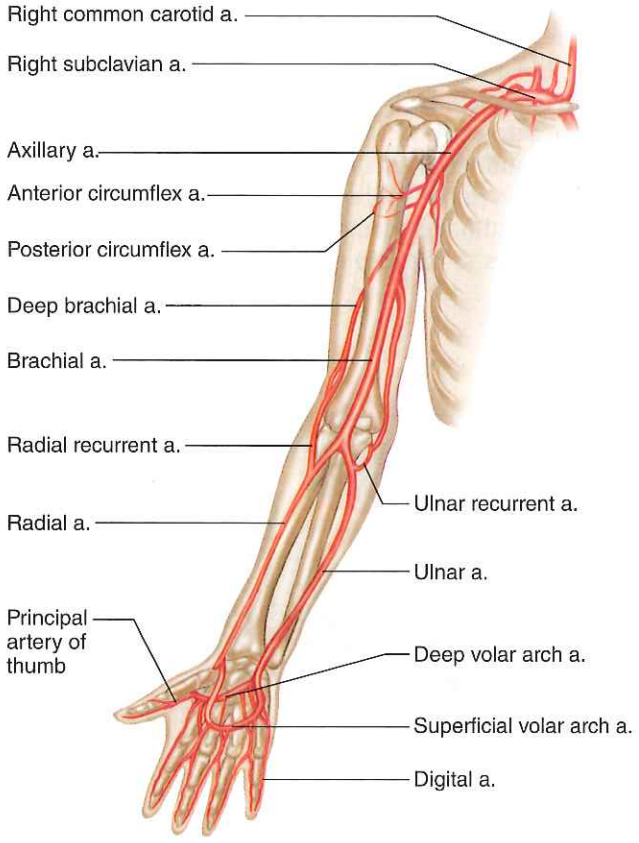


FIGURE 15.47 AP|R The major arteries to the shoulder and upper limb. (*a.* stands for artery.)

Q: *Blood from the brachial artery flows into which artery (arteries)?*

Answer can be found in Appendix G on page 938.

The **radial artery,** which is a continuation of the brachial artery, extends along the radial side of the forearm to the wrist. As it nears the wrist, it comes close to the surface and provides a convenient vessel for taking the pulse (radial pulse). Branches of the radial artery join the anastomosis of the elbow and supply the lateral muscles of the forearm.

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 structures in the hand.

Arteries to the Thoracic and Abdominal Walls

Blood reaches the thoracic wall through several vessels. These include branches from the subclavian artery and the thoracic aorta (fig. 15.48).

The subclavian artery contributes to this supply through a branch called the **internal thoracic artery.** This vessel originates in the base of the neck and passes downward on the pleura and behind the cartilages of the upper six ribs. It gives off two *anterior intercostal arteries* to each of the upper six intercostal spaces; these two arteries supply the intercostal muscles, other intercostal tissues, and the mammary glands.

The *posterior intercostal arteries* arise from the thoracic aorta and enter the intercostal spaces between the third through the eleventh ribs. These arteries branch to supply the intercostal muscles, the vertebrae, the spinal cord, and 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 lateral and posterior 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. These vessels provide blood to the pelvic organs, gluteal region, and lower limbs.

Each common iliac artery descends a short distance and divides into an internal (hypogastric) branch and an external branch. The **internal iliac artery** gives off many branches to various pelvic muscles and visceral structures, as well as to the gluteal muscles and the external genitalia. Parts of figure 15.49 show important branches of this vessel, including the following:

1. *iliolumbar artery* to the ilium and muscles of the back
2. *superior and inferior gluteal arteries* to the gluteal muscles, pelvic muscles, and skin of the buttocks
3. *internal pudendal artery* to muscles in the distal portion of the alimentary canal, the external genitalia, and the hip joint
4. *superior* and *inferior vesical arteries* to the urinary bladder. In males, these vessels also supply the seminal vesicles and the prostate gland
5. *middle rectal artery* to the rectum
6. *uterine artery* to the uterus and vagina

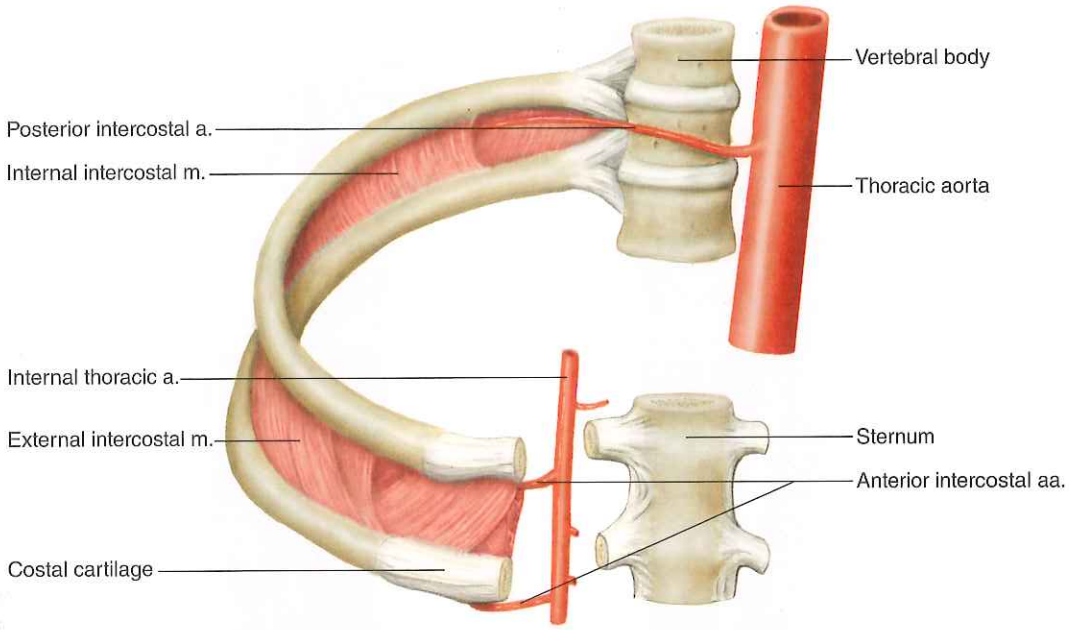


FIGURE 15.48 AP|R Arteries that supply the thoracic wall. (*a.* stands for artery, *aa.* stands for arteries , *m.* stands for muscle.)

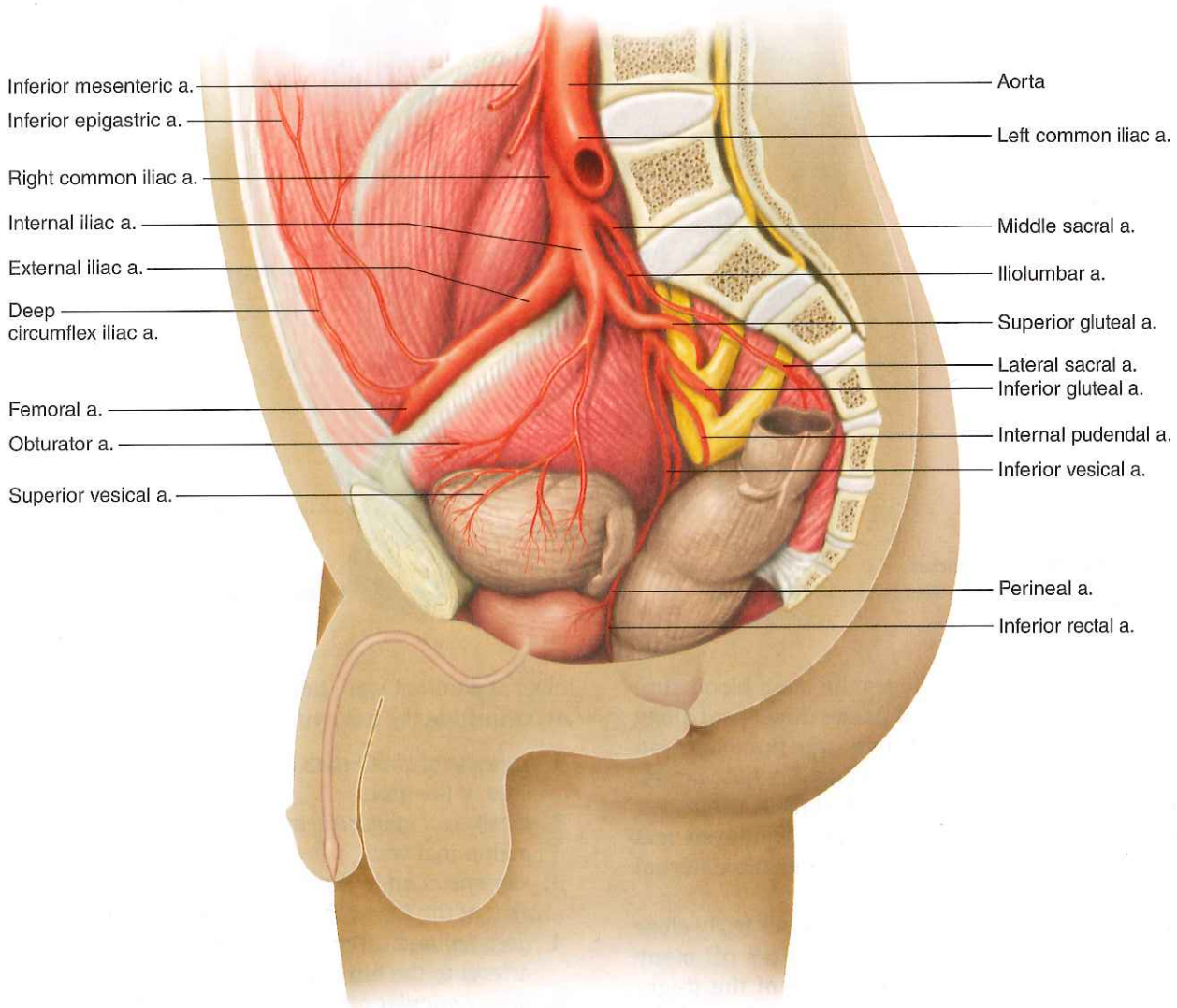


FIGURE 15.49 AP|R Arteries that supply the pelvic region. (*a.* stands for artery.)

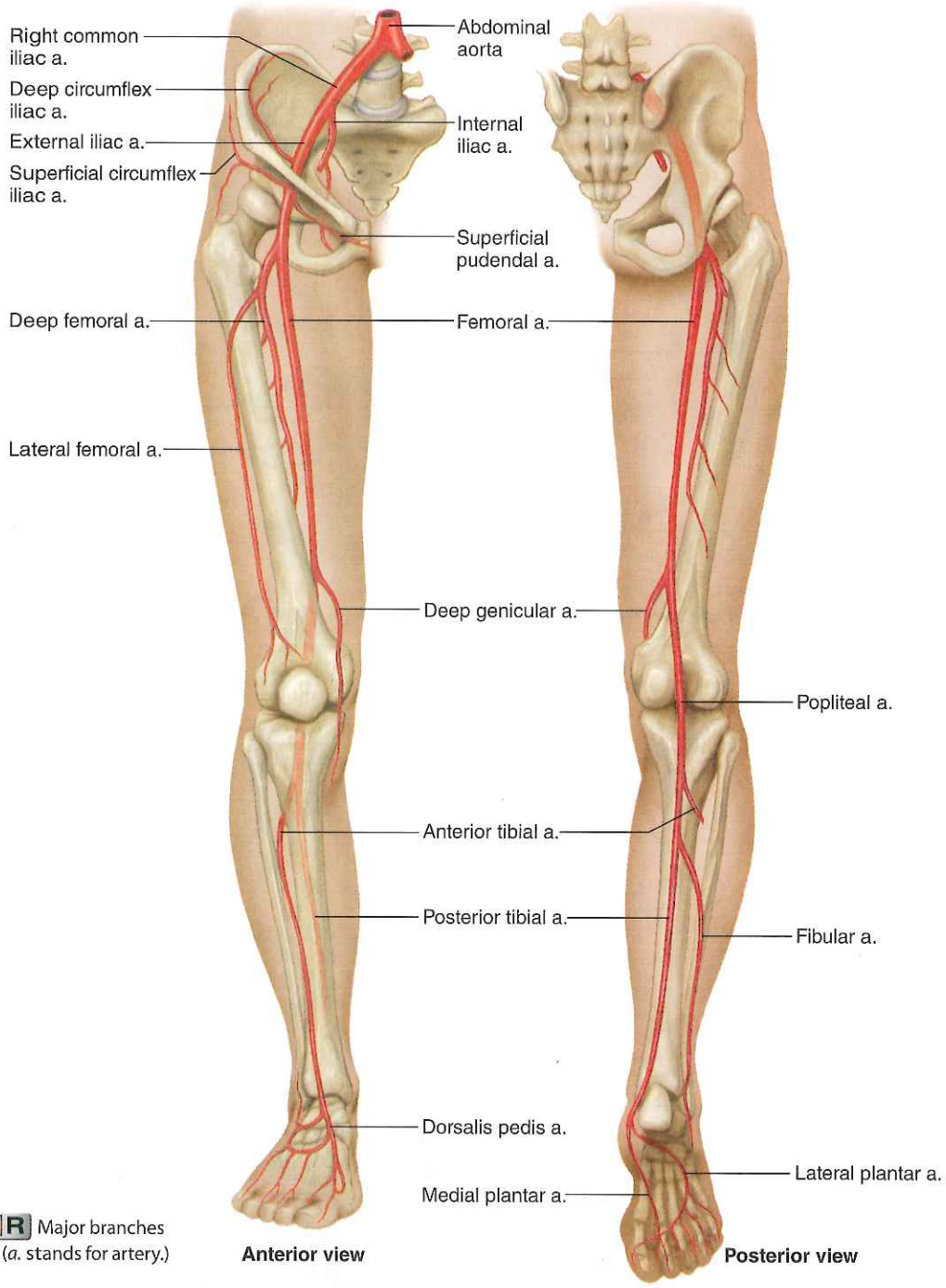


FIGURE 15.50 AP|R Major branches of the external iliac artery. (*a.* stands for artery.)

The **external iliac artery** provides the main blood supply to the lower limbs (fig. 15.50). It passes downward along the brim of the pelvis and gives off two large branches—an *inferior epigastric artery* and a *deep circumflex iliac artery.* These vessels supply the muscles and skin in the lower abdominal wall. Midway between the pubic symphysis and the anterior superior iliac spine of the ilium, the external iliac artery becomes the femoral artery.

The **femoral** (fem′or-al) **artery,** which passes fairly close to the anterior surface of the upper thigh, gives off many branches to muscles and superficial tissues of the thigh. These branches also supply the skin of the groin and the lower abdominal wall. Important subdivisions of the femoral artery include the following:

1. *superficial circumflex iliac artery* to the lymph nodes and skin of the groin
2. *superficial epigastric artery* to the skin of the lower abdominal wall
3. *superficial* and *deep external pudendal arteries* to the skin of the lower abdomen and external genitalia
4. *deep femoral artery* (the largest branch of the femoral artery) to the hip joint and muscles of the thigh
5. *deep genicular artery* to distal ends of thigh muscles and to an anastomosis around the knee joint

As the femoral artery reaches the proximal border of the space behind the knee (popliteal fossa), it becomes the **popliteal** (pop″lĭ-te′al) **artery.** Branches of this artery supply blood to the knee joint and to certain muscles in the thigh and calf. Also, many of its branches join the anastomosis of the knee and help provide alternate pathways for blood in the case of arterial obstructions. At the lower border of the popliteal fossa, the popliteal artery divides into the anterior and posterior tibial arteries.

The **anterior tibial** (tib′e-al) **artery** passes downward between the tibia and the fibula, giving off branches to the skin and muscles in the anterior and lateral regions of the leg. It also communicates with the anastomosis of the knee and with a network of arteries around the ankle. This vessel continues into the foot as the *dorsalis pedis artery,* which supplies blood to the instep and toes.

The **posterior tibial artery,** which is the larger of the two popliteal branches, descends beneath the calf muscles, giving off branches to the skin, muscles, and other tissues of the leg along the way. Some of these vessels join the anastomoses of the knee and ankle. As it passes between the medial malleolus and the heel, the posterior tibial artery divides into the *medial* and *lateral plantar arteries.* Branches from these arteries supply blood to tissues of the heel, instep, and toes.

The largest branch of the posterior tibial artery is the *fibular artery,* which extends downward along the fibula and contributes to the anastomosis of the ankle. Figure 15.51 shows the major vessels of the arterial system.

PRACTICE

61 Name the parts of the aorta.

62 Name the vessels that arise from the aortic arch.

63 Name the branches of the thoracic and abdominal aorta.

64 Which vessels supply blood to the head? To the upper limb? To the abdominal wall? To the lower limb?

15.8 VENOUS SYSTEM

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

Characteristics of Venous Pathways

The vessels of the venous system originate with the merging of capillaries into venules, venules into small veins, and small veins meet to form larger ones. Unlike the arterial pathways, those of the venous system are difficult to follow because the smaller vessels commonly connect in irregular networks. Many unnamed tributaries may join to form a large vein. The pathways of larger veins are easier to follow. These veins typically parallel the courses of named arteries, and many bear 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 the 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, passing over the sternocleidomastoid muscles and beneath the platysma. They empty into the *right* and *left subclavian veins* in the base of the neck (fig. 15.52).

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

A lung cancer, enlarged lymph node, or an aortic aneurysm can compress the superior vena cava, interfering with return of blood from the upper body to the heart. This produces pain; shortness of breath; distension of veins draining into the superior vena cava; and swelling of tissues in the face, head, and lower limbs.

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. Deep venous drainage of the upper limbs begins in the digital veins that drain into pairs of **radial veins** and **ulnar veins,** which merge to form a pair of **brachial veins.** 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 the blood can leave the tissues (fig. 15.53).

The major vessels of the superficial network are the basilic and cephalic veins. They arise from anastomoses in the palm and wrist on the ulnar and radial sides, respectively.

The **basilic** (bah-sil′ik) **vein** passes along the back of the forearm on the ulnar side for a distance and then curves forward to the anterior surface below the elbow. It continues ascending on the medial side until it reaches the middle of the arm. There it deeply penetrates the tissues and joins the *brachial vein.* As the basilic and brachial veins merge, they form the *axillary vein.*

The **cephalic** (sĕ-fal′ik) **vein** courses upward on the lateral side of the upper limb from the hand to the shoulder. In

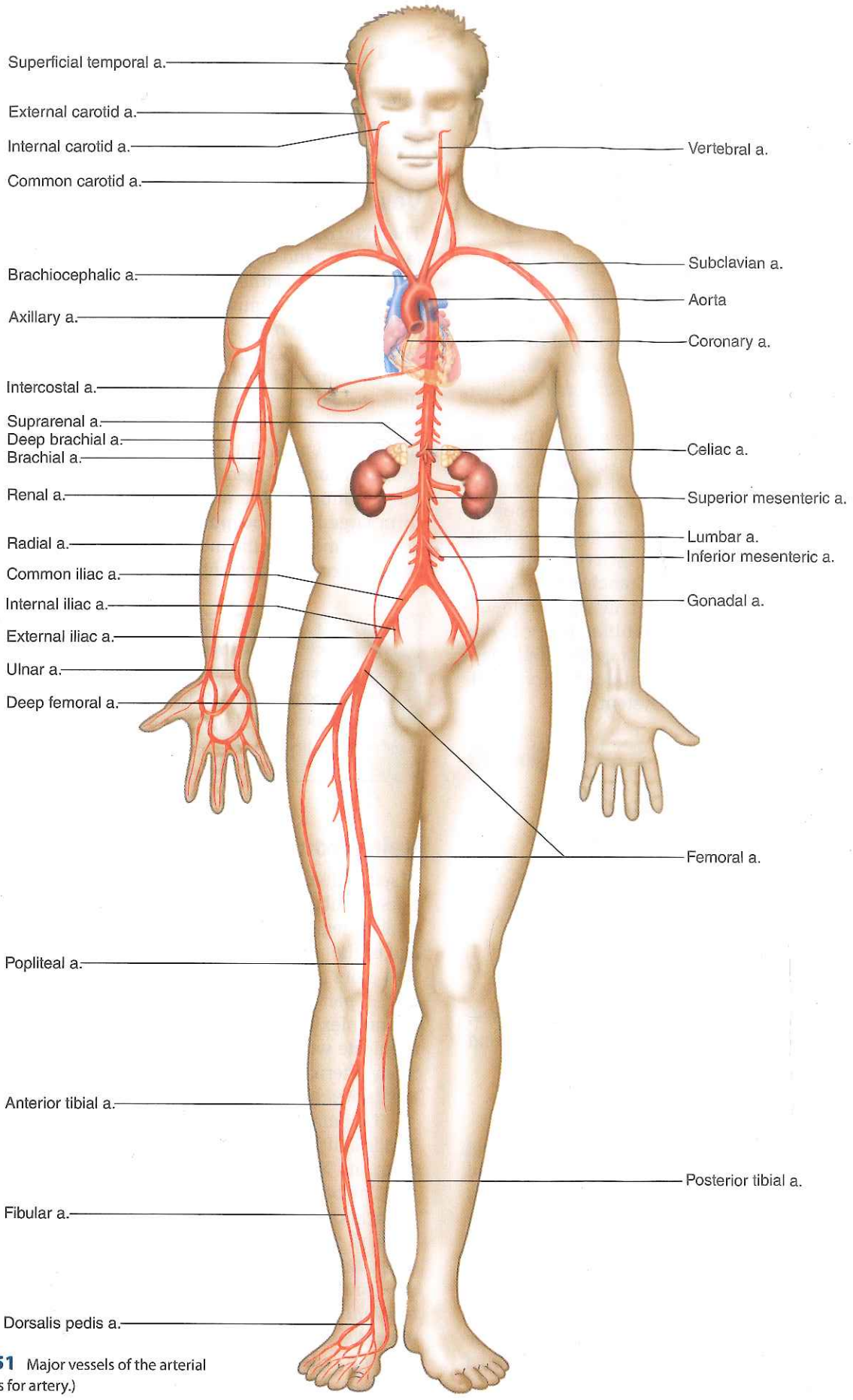


FIGURE 15.51 Major vessels of the arterial system. (*a.* stands for artery.)

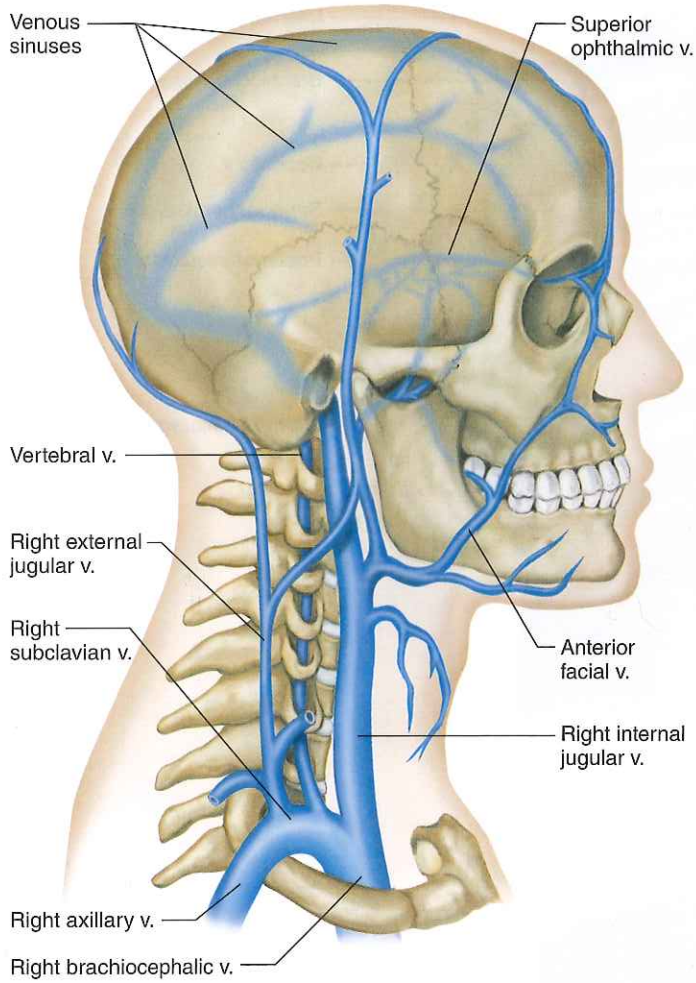


FIGURE 15.52 AP|R The major veins of the brain, head, and neck. The clavicle has been removed. (*v.* stands for vein.)

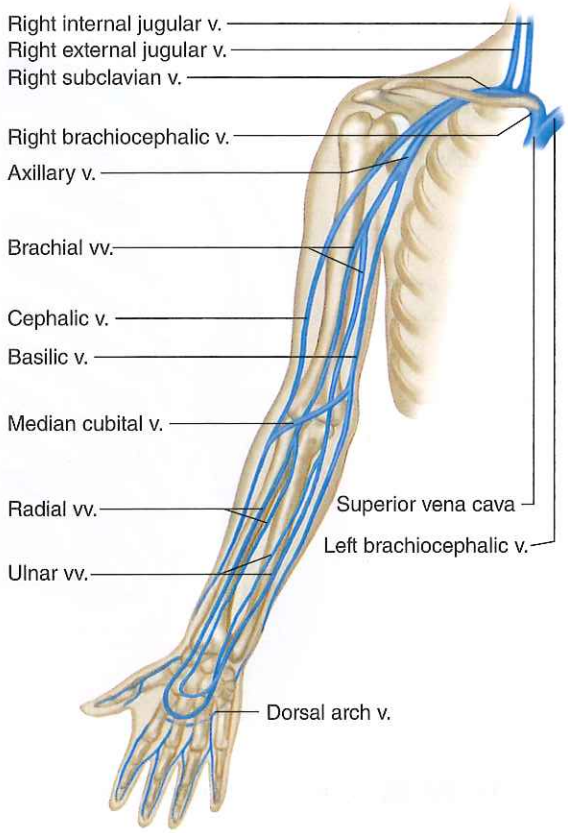


FIGURE 15.53 AP|R The major veins of the upper limb and shoulder. (*v.* stands for vein, *vv.* stands for veins.)

Q: *Blood from the brachial and basilic veins drains into which vein(s)?*

Answer can be found in Appendix G on page 938.

the shoulder, it pierces the tissues and joins the axillary vein, which beyond the axilla 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 sample of blood for examination or to add fluids to the 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 (fig. 15.54).

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 carry blood directly to the atria of the heart, except those that drain the abdominal viscera (fig. 15.55). They originate in the capillary networks of the stomach, intestines, pancreas, and spleen and carry blood from these organs through a **hepatic portal** (por′tal) **vein** to the liver (fig. 15.56). There the blood enters capillary-like **hepatic sinusoids** (hĕ-pat′ik si′nŭ-soidz). This unique venous pathway is called the **hepatic portal system.**

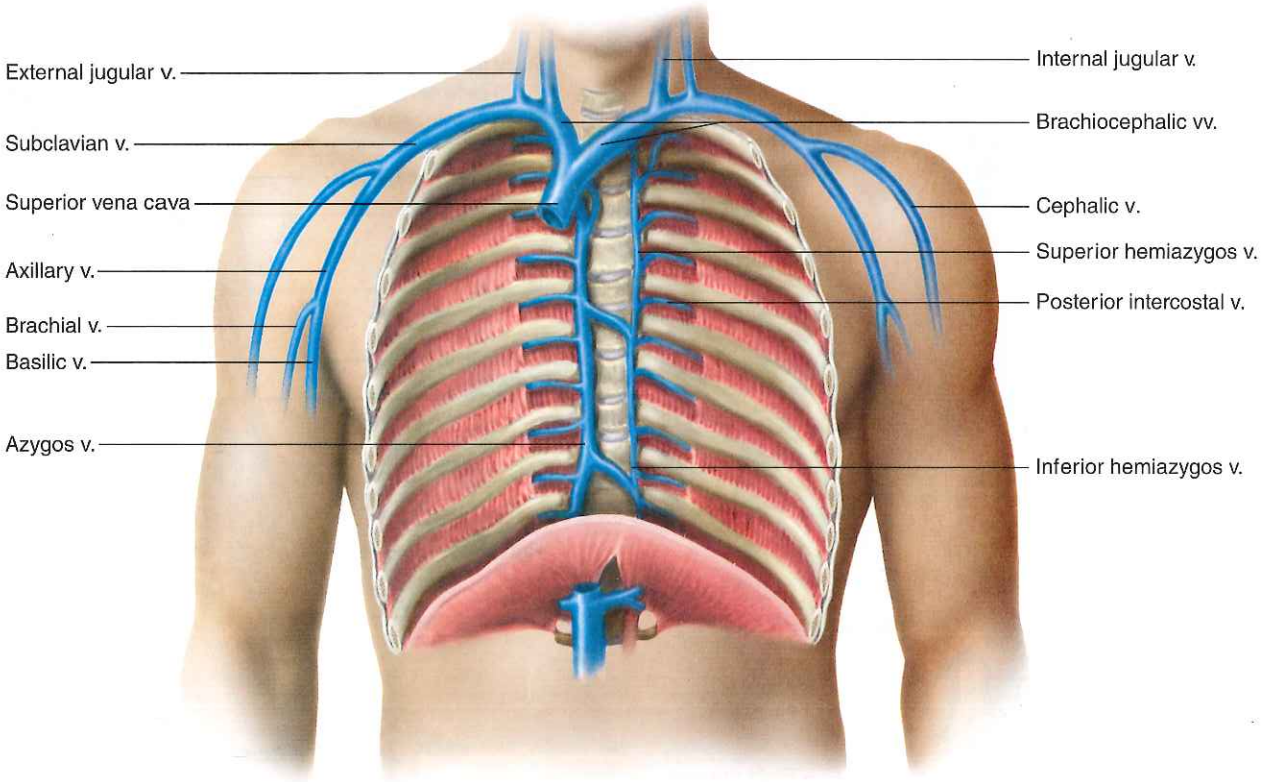


FIGURE 15.54 AP|R Veins that drain the thoracic wall. (*v.* stands for vein, *vv.* stands for veins.)

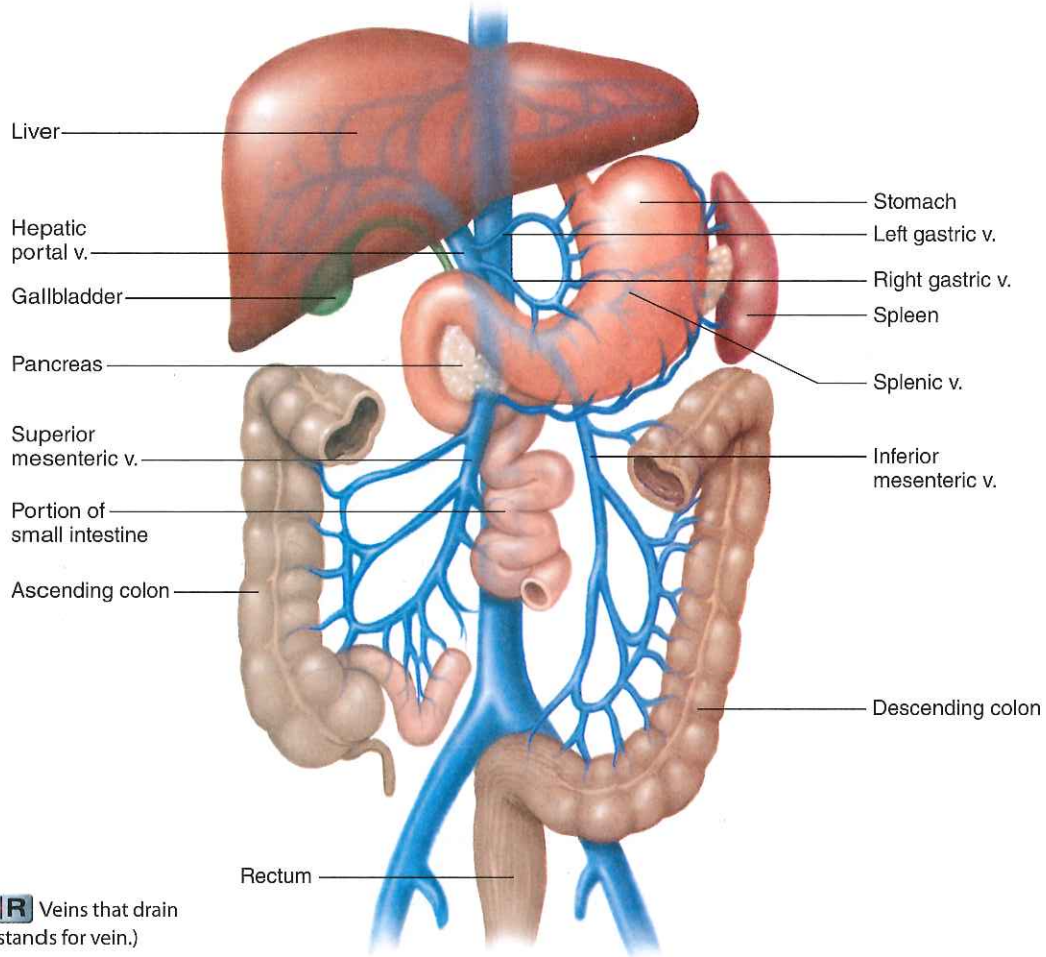


FIGURE 15.55 AP|R Veins that drain the abdominal viscera. (*v.* stands for vein.)

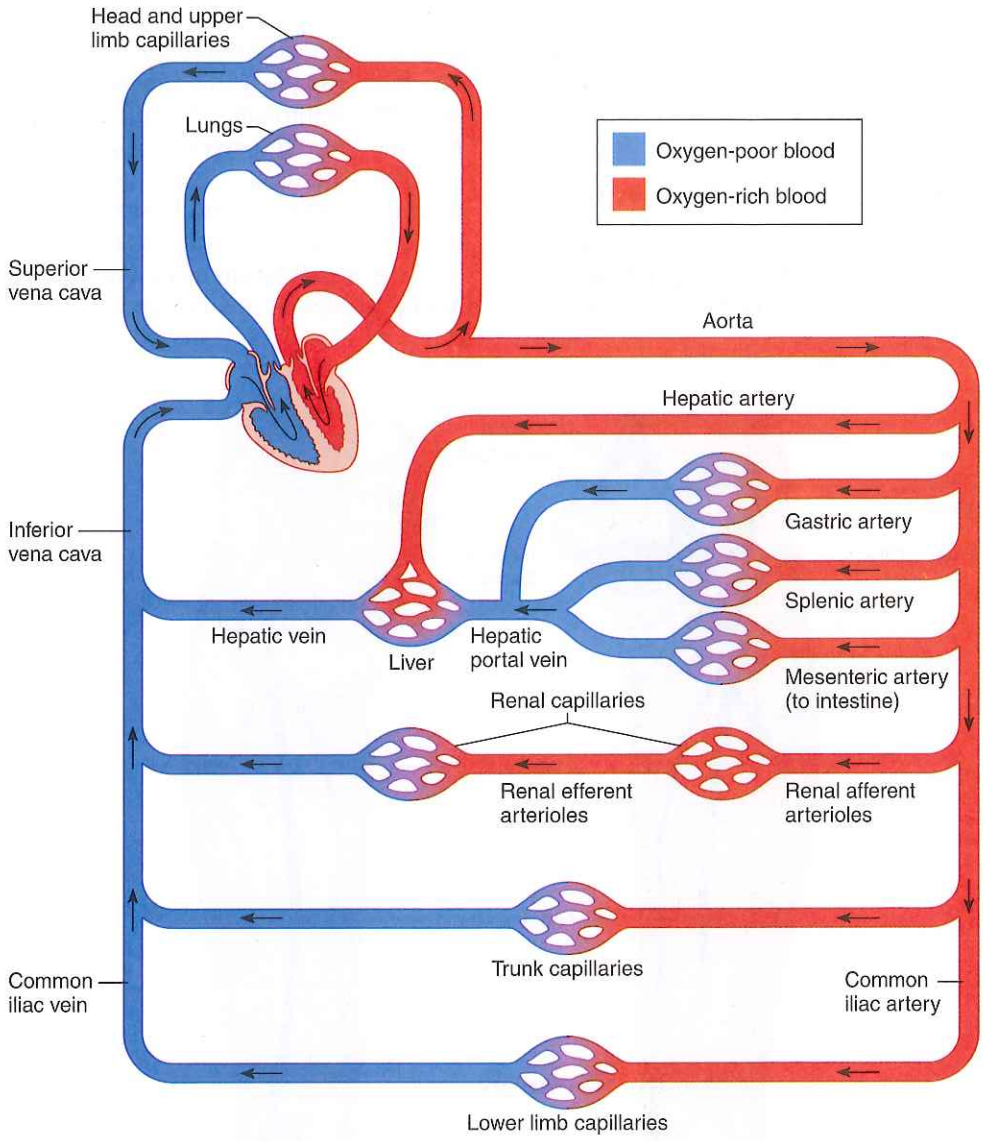


FIGURE 15.56 In this schematic drawing of the cardiovascular system, note how the hepatic portal vein drains one set of capillaries and leads to another set. A similar relationship exists in the kidneys.

The tributaries of the hepatic portal vein include the following vessels:

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; as well as its largest tributary, the *inferior mesenteric vein,* from the descending colon, sigmoid colon, and rectum

About 80% of the blood flowing to the liver in the hepatic portal system comes from the capillaries in the stomach and intestines and is oxygen-poor, but nutrient-rich (see chapter 17, p. 672). 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 them 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 the hepatic sinusoids phagocytize these microorganisms, removing them from the portal blood before it leaves the liver.

After passing through the hepatic sinusoids of the liver, the 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.

Other veins empty into the inferior vena cava as it ascends through the abdomen. They include the *lumbar, gonadal, renal, suprarenal,* and *phrenic veins.* These vessels drain regions that arteries with corresponding names supply.

Veins from the Lower Limb and Pelvis

Veins that drain the blood from the lower limb can be divided into deep and superficial groups, as in the upper limb (fig. 15.57). The deep veins of the leg, such as the paired *anterior* and *posterior tibial veins,* have names that correspond to 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** begins in the lateral portion of the foot and passes upward behind the lat-

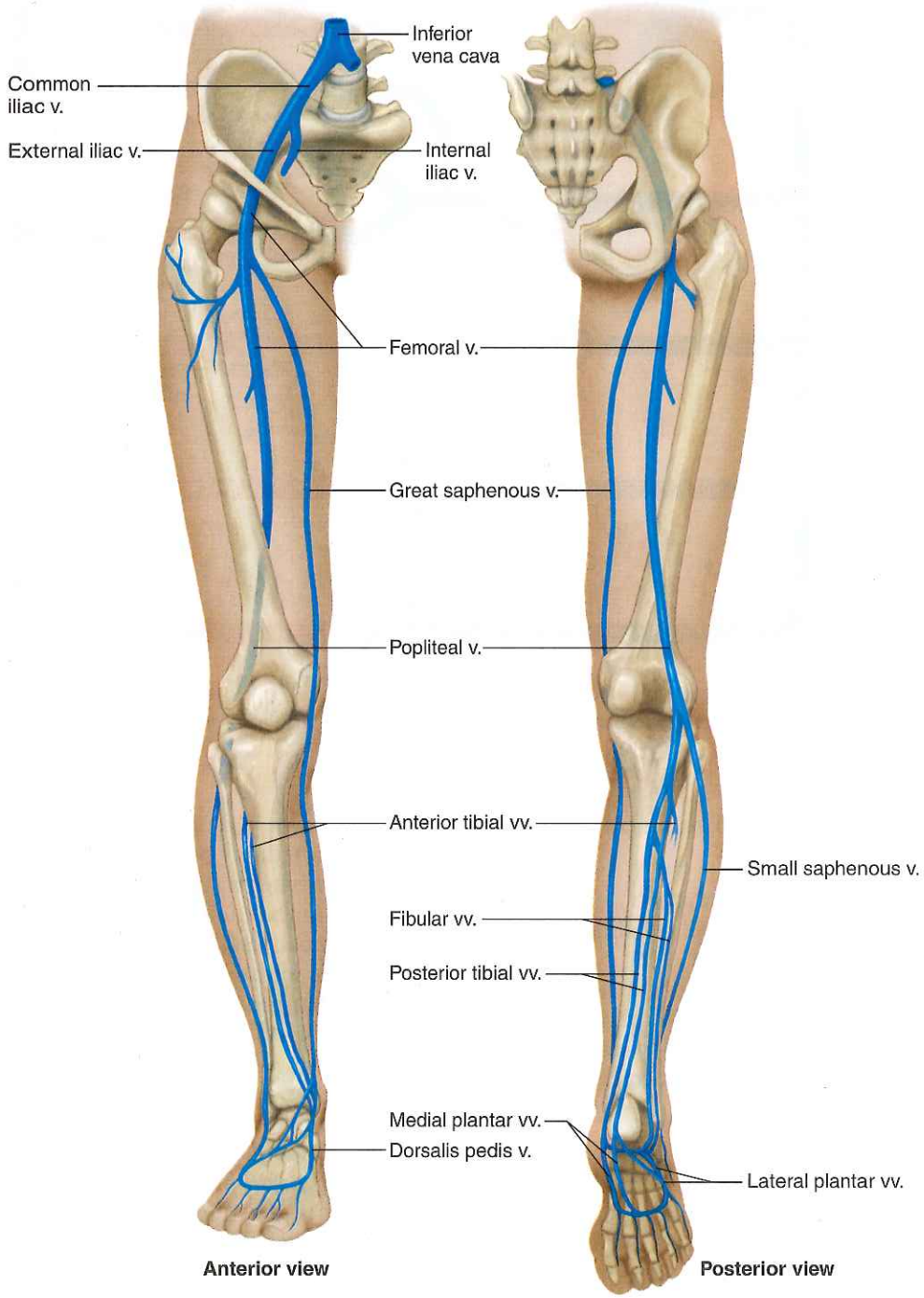


FIGURE 15.57 AP|R The major veins of the lower limb and pelvis. (*v.* stands for vein, *vv.* stands for veins.)

eral malleolus. It ascends along the back of the calf, enters the popliteal fossa, and joins the popliteal vein.

The **great saphenous vein,** the longest vein in the body, originates on the medial side of the foot. It ascends in front of the medial malleolus and extends upward along the medial side of the leg and thigh. In the thigh just below the inguinal ligament, it deeply penetrates 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 organs of the reproductive, urinary, and digestive systems. This vein is formed by tributaries corresponding to the branches of the internal iliac artery, such as the *gluteal, pudendal, vesical, rectal, uterine,* and *vaginal veins.* Typically, these veins have many connections and form complex networks (plexuses) in the regions of the rectum, urinary bladder, and prostate gland (in the male) or uterus and vagina (in the female).

The internal iliac veins originate deep within the pelvis and ascend to the pelvic brim. There they 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* at the level of the fifth lumbar vertebra. Figure 15.58 shows the major vessels of the venous system.

Clinical Application 15.6 looks at molecular explanations of certain cardiovascular disorders. Clinical Application 15.7 discusses coronary artery disease.

PRACTICE

65 Name the veins that return blood to the right atrium.

66 Which major veins drain blood from the head? From the upper limbs? From the abdominal viscera? From the lower limbs?

15.9 LIFE-SPAN CHANGES

The years take a toll on the cardiovascular system. Signs of cardiovascular disease may appear long before symptoms arise. Autopsies of soldiers killed in the Korean and Vietnam Wars, for example, revealed significant plaque buildup in the arterial walls of otherwise healthy young men. Incidence of disease of the heart and blood vessels increases exponentially with age. About 60% of men over age sixty have at least one narrowed coronary artery; the same is true for women over age eighty.

Assessing cardiac output over a lifetime vividly illustrates how cardiovascular disease prevalence can interfere with studying the changes associated with normal aging. Recall that cardiac output is the ability of the heart to meet the body's oxygen requirements and is calculated as the heart rate in beats per minute multiplied by the stroke volume in milliliters per beat. For many years, studies indicated that cardiac output declines with age, but when researchers began to screen participants for hidden heart disease with treadmill stress tests, then evaluated only individuals with completely healthy cardiovascular systems, they discovered that cardiac output at rest is maintained as a person ages. It does decline during exercise for some people, however.

The heart may normally shrink slightly with age, but disease may enlarge it. The proportion of the heart that is cardiac muscle declines with age, even in a healthy person, because cardiac muscle cells do not divide. Lipofuscin pigments become especially prominent in these cells. Fibrous connective tissue and adipose tissue fill in the spaces left by the waning population of cardiac muscle cells, thickening the endocardium. Adipose cells may also accumulate in the ventricle walls and the septum between them. The left ventricular wall may be up to 25% thicker at age eighty than it was at age thirty.

The heart slows slightly with age, the cardiac cycle lengthening by 2% to 5% per year. The aging heart pumps about 8 milliliters less blood per year.

The heart valves thicken and become more rigid after age sixty, although these changes may begin as early as the third decade. The valves may calcify.

The cardiac conduction system may remain functional despite changes to the heart. The sinoatrial and atrioventricular nodes and the atrioventricular bundle become more elastic. These changes may alter the ECG pattern.

Systolic blood pressure increases with age; a blood pressure reading of 140/90 is not abnormal in an older person. In about 40% of the elderly, the systolic pressure exceeds 160. The increase may be due to the decreasing diameters and elasticity of arteries. Regular exercise can slow these changes. Resting heart rate declines from 145 or more beats per minute in a fetus to 140 beats per minute in a newborn, then levels out in an adult to about 70 (range of 60–99) beats per minute.

In the vascular system, age-related changes are most apparent in the arteries. The tunica interna thickens. Dividing smooth muscle cells in the tunica media may push up the endothelium in places, and over time, the lumens of the larger arteries narrow. Rigidity increases as collagen, calcium, and fat are deposited as elastin production declines. Arterial elasticity at age seventy is only about half of what it was at age twenty. The arterioles have diminished ability to contract in response to cold temperatures and to dilate in response to heat, contributing to the loss of temperature control common among the elderly. The extent of change in arteries may reflect stress—that is, not all arteries "age" at the same rate.

Veins may accumulate collagen and calcify but, in general, do not change as much with age as do arteries. Thickened patches may appear in the inner layer, and fibers in the valves, but venous diameters are large enough that these changes have little impact on function. The venous supply to many areas is so redundant that alternate vessels can often take over for damaged ones.

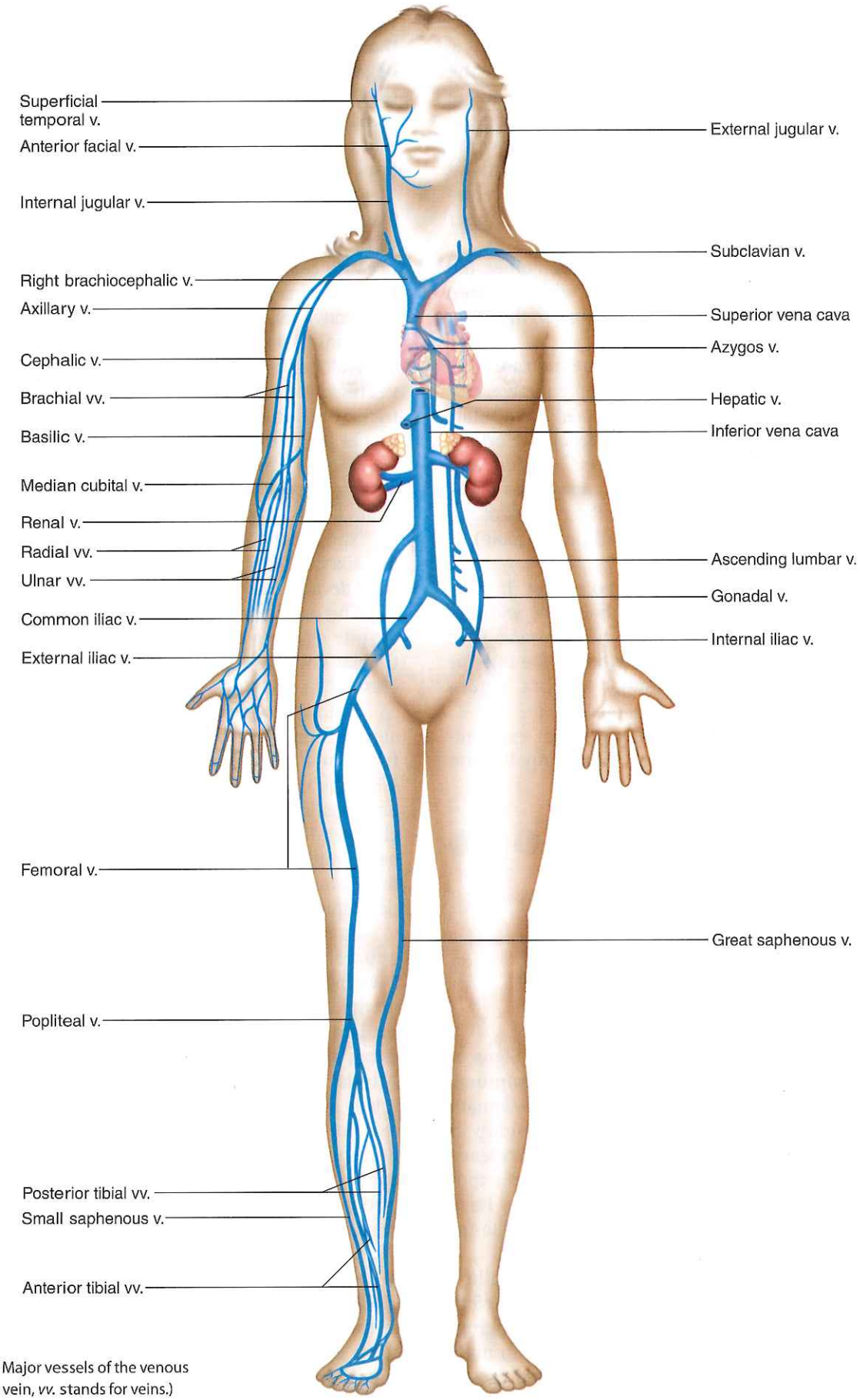
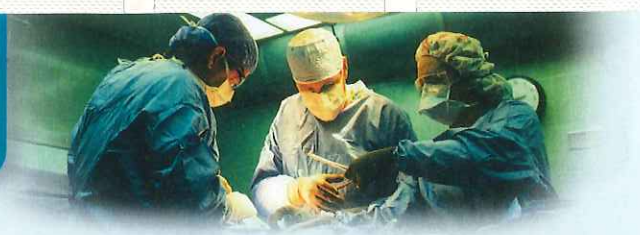


FIGURE 15.58 Major vessels of the venous system. (*v.* stands for vein, *vv.* stands for veins.)

15.6 CLINICAL APPLICATION



Molecular Causes of Cardiovascular Disease

A variety of environmental and inherited factors contribute to cardiovascular disease. These factors include poor diet, sedentary lifestyle, and genetic predisposition. Disorders of the heart and blood vessels caused by single genes are rare, but understanding how they arise can provide insights useful in developing treatments for more prevalent forms of disease. For example, widely used cholesterol-lowering drugs called statins were developed based on understanding an inherited condition, familial hypercholesterolemia, that affects one in a million children.

A Connective Tissue Defect

Just after midnight, the college basketball player lay in her bed watching TV. At about 2 A.M., her roommate heard sounds of disturbed breathing and could not rouse the young player. The nineteen-year-old athlete died within an hour of reaching the hospital. Her aorta had burst. She had *Marfan syndrome,* an inherited condition that also caused the characteristics that led her to excel in her sport—her great height and long fingers.

In Marfan syndrome, an abnormal form of a connective tissue protein called fibrillin weakens the aorta wall, dilating the aortic root (fig. 15J). Identifying the mutation can make it possible to locate and surgically repair a weakened aorta, or to take a drug that can slow the effect of the disease on the aorta. This can prevent a tragic first symptom—sudden death.

A Myosin Defect

Each year, one or two seemingly healthy young people die suddenly during a sports event, usually basketball. The cause of death is often *familial hypertrophic cardiomyopathy,* an inherited overgrowth of the heart muscle. The defect in this disorder is different from that behind Marfan syndrome. It is an abnormality in one of the myosin chains that comprise cardiac muscle. Again, detecting the responsible gene can alert affected individuals to their increased risk of sudden death. They can adjust the type of exercise they do to avoid stressing the cardiovascular system.

A Metabolic Block

Inherited heart disease can strike early in life. Jim D. died at four days of age, two days after suffering cardiac arrest. Two years later, his parents had another son. Like Jim, Kerry seemed normal at birth, but when he was thirty-six hours old, his heart rate plummeted, he had a seizure, and he stopped breathing. He was resuscitated. A blood test revealed excess long-chain fatty acids, indicating inability to use fatty acids. Hunger triggered the symptoms because the boys could not use fatty acids for energy, as healthy people do. Kerry survived for three years by following a diet low in fatty acids and eating frequently. Once he became comatose because he missed a meal. Eventually, he died of respiratory failure.

Jim and Kerry had inherited a deficiency of a mitochondrial enzyme that processes long-chain fatty acids. This is a primary energy source for cardiac muscle, so their hearts failed.

Controlling Cholesterol

Low-density lipoprotein (LDL) receptors on liver cells admit cholesterol into the cells, keeping the lipid from building up in the bloodstream and occluding arteries. When LDL receptors bind cholesterol, they activate a negative feedback system that temporarily halts the cell's production of cholesterol. In the severe form of *familial hypercholesterolemia,* a person inherits two defective copies of the gene encoding the LDL receptors. Yellowish lumps of cholesterol are deposited behind the knees and elbows, and heart failure usually causes death in childhood. People who inherit one defective gene have a milder form of the illness. They develop coronary artery disease in young adulthood, but can delay symptoms by following a heart-healthy diet and regularly exercising. These people have half the normal number of LDL receptors.

In Niemann-Pick type C disease, a defective protein disturbs the fate of cholesterol inside cells. Normally, the protein escorts cholesterol out of a cell's lysosomes, which triggers the negative feedback mechanism that shuts off cholesterol synthesis. When the protein is absent or malfunctions, the cell keeps producing cholesterol and LDL receptors. Coronary artery disease develops, and is typically fatal in childhood. ■

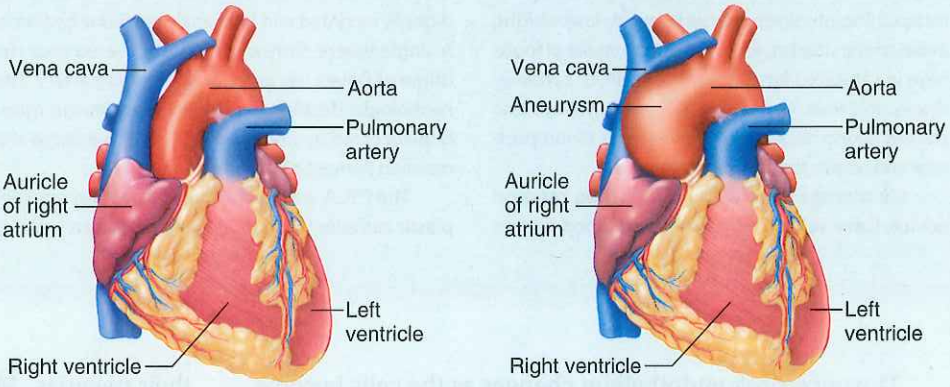


FIGURE 15J The symptoms of Marfan syndrome, including a progressive dilation of the aortic root, arise from an abnormal form of the connective tissue protein fibrillin that in turn increases signaling by a protein called transforming growth factor B (TGF-B). A drug usually used to treat hypertension, losartan, lowers levels of TGF-B and has been shown in preliminary clinical trials to slow the life-threatening ballooning out of the ascending aorta. Surgery can replace the affected part of the aorta with a synthetic graft.

15.7 CLINICAL APPLICATION

Coronary Artery Disease

Dave R., a fifty-two-year-old overweight accountant, had been having occasional chest pains for several months. The mild pain occurred during his usual weekend tennis match, and he attributed it to indigestion. The discomfort almost always diminished after the game, but recently, the pain seemed more severe and prolonged. Dave asked his physician about the problem. The physician explained that Dave was probably experiencing *angina pectoris,* a symptom of *coronary artery disease* (CAD), and suggested that he undergo an *exercise stress test.* Dave walked on a treadmill, increasing speed and incline while he exercised. An ECG was recorded and his blood pressure monitored. Near the end of the test, when Dave's heart reached the desired rate, a small amount of radioactive thallium-201 was injected into a vein. A *scintillation counter* scanned Dave's heart to determine if branches of his coronary arteries carried the blood marked with the thallium uniformly throughout the myocardium.

The test revealed that Dave was developing CAD. He also had hypertension and high serum cholesterol. The physician advised Dave to lose weight; avoid stress; stop smoking; reduce his intake of foods high in saturated fats, cholesterol, refined carbohydrates, and sodium; and exercise regularly. Dave also started taking medications to lower his blood pressure and relieve the pain of angina.

Six months later, despite following medical advice, Dave suffered a heart attack. Blood flow to part of his myocardium was obstructed, producing oxygen deficiency. The attack began as severe, crushing chest pain, shortness of breath, and sweating. Paramedics stabilized Dave's condition and transported him to a hospital. There, a cardiologist concluded from an ECG that Dave's heart attack was caused by a blood clot blocking a coronary artery (occlusive coronary thrombosis). The cardiologist intravenously administered a thrombolytic ("clot-busting") drug.

A repeat ECG a few hours later showed that the blood vessel remained partially obstructed, so the cardiologist ordered a *coronary angiogram* (see fig. 15.14). In this X-ray procedure, conducted in a cardiac catheterization laboratory, a thin plastic catheter was passed through a guiding sheath inserted into the femoral artery of Dave's right inguinal area. From there, the catheter was pushed into the aorta until it reached the region of the openings to the coronary arteries.

X-ray fluoroscopy monitored the progress of the catheter. Each time the catheter was in proper position, a radiopaque dye (contrast medium) was released from its distal end into the blood. X-ray images that revealed the path of the dye as it entered a coronary artery and its branches were digitally recorded and later analyzed frame by frame. A single severe narrowing was discovered near the origin of Dave's left anterior descending artery. The cardiologist decided to perform *percutaneous transluminal coronary angioplasty* (PTCA) to enlarge the opening (lumen) of that vessel.

The PTCA was performed by passing another plastic catheter through the guiding sheath used for the angiogram. This second tube had a tiny deflated balloon at its tip. The balloon was placed in the region of the arterial narrowing and inflated for a short time under high pressure. The inflating balloon compressed the atherosclerotic plaque (atheroma) obstructing the arterial wall and stretched the blood vessel wall, widening its lumen (recanalization). Blood flow to the myocardial tissue downstream from the obstruction immediately improved.

About 50% of the time, a vessel opened with PTCA becomes occluded again, because the underlying disease persists. To prevent this restenosis, the doctor inserted a *coronary stent,* which is an expandable tube or coil that holds the vessel wall open. The cardiologist had two other options that have a slightly higher risk of causing damage. She might have vaporized the plaque obstructing the vessel with an excimer laser pulse delivered along optical fibers threaded through the catheter. Or, she could have performed atherectomy, in which a cutting device attached to the balloon inserted into the catheter spins, removing plaque by withdrawing it on the catheter tip.

Should the coronary stent fail, or an obstruction block another heart vessel, Dave might benefit from *coronary bypass surgery.* A portion of his internal mammary artery inside his chest wall would be removed and sutured between the aorta and the blocked coronary artery at a point beyond the obstruction, restoring circulation through the myocardium. ■

The once-sleek endothelium changes as the cells become less uniform in size and shape. The endothelial inner linings of blood vessels are important to health because these cells release nitric oxide, which signals the vessels to dilate to increase blood flow, which counters atherosclerosis and thrombosis. In addition to the changes in arteries and veins, the number of capillaries declines with age.

Exercise can help maintain a "young" vascular system. One study compared the vascular endothelial linings of athletic and sedentary individuals of various ages and found that the status of the vessels of the exercising elderly were very similar to those of either athletic or sedentary people in their twenties. Many studies have correlated regular exercise to lowered heart disease risk in older people.

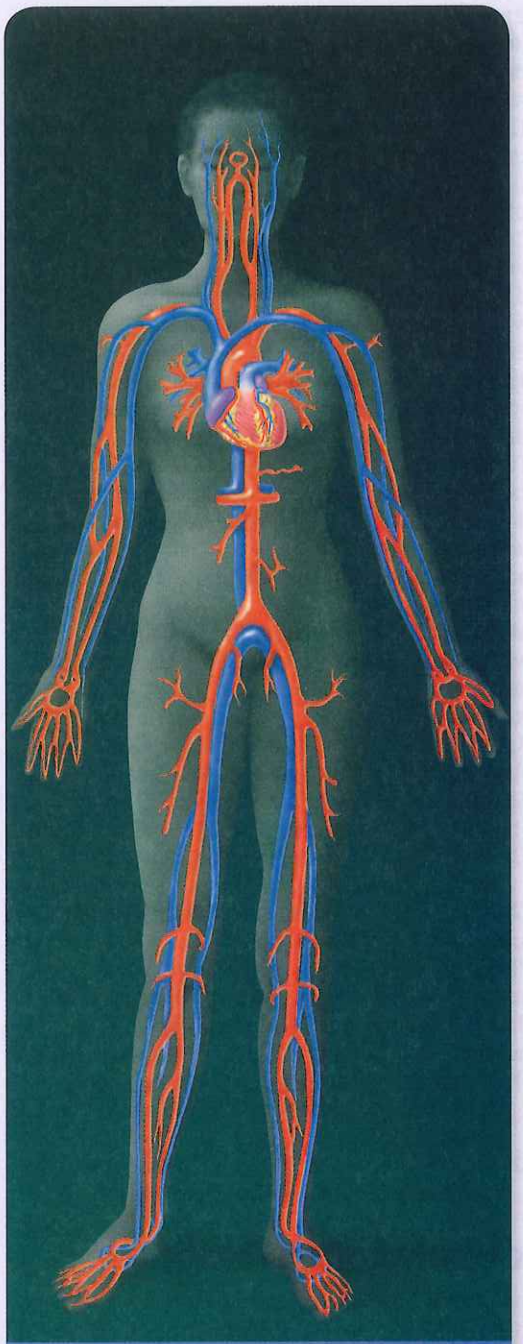
Overall, aging-related changes affect many components of the cardiovascular system. But in the absence of disease, the system is so fine-tuned and redundant that effective oxygen delivery can continue well into the later decades of life.

PRACTICE

67 Explain why the heart may enlarge with age.

68 Describe what happens to resting heart rate with age.

INNERCONNECTIONS ●●● Cardiovascular System



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.

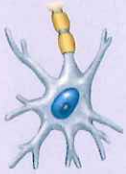
Skeletal System

Bones help control plasma levels of calcium ions, which influence heart action.

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.

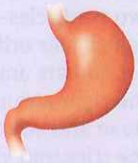
Endocrine System

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.

CHAPTER SUMMARY

15.1 INTRODUCTION (PAGE 555)

The cardiovascular system is composed of the heart and blood vessels, which circulate blood to supply oxygen to, and remove wastes from, body cells.

15.2 STRUCTURE OF THE HEART (PAGE 555)

1. Size and location of the heart
   a. The heart is about 14 centimeters long and 9 centimeters wide.
   b. It is located in 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 visceral and parietal layers of the pericardium.
3. Wall of the heart
   a. The wall of the heart has three layers.
   b. These layers include an epicardium, a myocardium, and an endocardium.
4. Heart chambers and valves
   a. The heart is divided into four chambers—two atria and two ventricles—that communicate through atrioventricular orifices on each side.
   b. Right chambers and valves
      (1) The right atrium receives blood from the venae cavae and coronary sinus.
      (2) The tricuspid valve guards the right atrioventricular orifice.
      (3) The right ventricle pumps blood into the pulmonary trunk.
      (4) 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 mitral valve guards the left atrioventricular orifice.
      (3) The left ventricle pumps blood into the aorta.
      (4) An aortic valve guards the base of the aorta.
5. Skeleton of the heart
   a. The skeleton of the heart consists of fibrous rings that enclose the bases of the pulmonary artery, aorta, and atrioventricular orifices.
   b. The fibrous rings provide attachments for valves and muscle fibers and prevent the orifices from excessively dilating during ventricular contractions.
6. Path of blood through the heart
   a. Blood low in oxygen and high in carbon dioxide enters the right side of the heart from the venae cavae and coronary sinus and then is pumped into the pulmonary circulation.
   b. After the blood is oxygenated in the lungs and some of its carbon dioxide is removed, it returns to the left side of the heart through the pulmonary veins.
   c. From the left ventricle, it moves into the aorta.
7. Blood supply to the heart
   a. The coronary arteries supply blood to the myocardium.
   b. It is returned to the right atrium through the cardiac veins and coronary sinus.

15.3 HEART ACTIONS (PAGE 566)

1. Cardiac cycle
   a. The atria contract (atrial systole) while the ventricles relax (ventricular diastole); the ventricles contract (ventricular systole) while the atria relax (atrial diastole).
   b. Pressure in the chambers rises and falls in cycles.
2. Heart sounds
   a. Heart sounds can be described as *lubb-dupp*.
   b. Heart sounds are due to the vibrations that the valve movements produce.
   c. The first part of the sound occurs as AV valves close, and the second part is associated with the closing of pulmonary and aortic valves.
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.
   c. Except for a small region in the floor of the right atrium, the fibrous skeleton separates the atrial syncytium from the ventricular syncytium.
4. Cardiac conduction system
   a. This system, composed of specialized cardiac muscle tissue, initiates and conducts depolarization waves through the myocardium.
   b. Impulses from the SA node pass slowly to the AV node; impulses travel rapidly along the AV bundle and Purkinje fibers.
   c. Muscle fibers in the ventricular walls form whorls that squeeze blood out of the contracting ventricles.
5. Electrocardiogram
   a. An electrocardiogram (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 concentration of various ions affect heartbeat.
   b. Branches of parasympathetic and sympathetic nerve fibers innervate the SA and AV nodes.
      (1) Parasympathetic impulses decrease heart action; sympathetic impulses increase heart action.
      (2) The cardiac center in the medulla oblongata regulates autonomic impulses to the heart.

15.4 BLOOD VESSELS (PAGE 575)

The blood vessels form a closed circuit of tubes that transports blood between the heart and body cells. The tubes include arteries, arterioles, capillaries, venules, and veins.

1. Arteries and arterioles
   a. The arteries are adapted to carry blood under relatively high pressure away from the heart.
   b. The arterioles are branches of arteries.

c. The walls of arteries and arterioles consist of layers of endothelium, smooth muscle, and connective tissue.
d. Autonomic fibers innervate smooth muscles in vessel walls, causing vasoconstriction or vasodilation.

2. Capillaries

Capillaries connect arterioles and venules. The capillary wall is a single layer of squamous epithelial cells that forms a semipermeable membrane.

a. Capillary permeability
   (1) Openings in the capillary walls are thin slits between endothelial cells.
   (2) The sizes of the openings vary from tissue to tissue.
   (3) Endothelial cells of brain capillaries are tightly fused, forming a blood-brain barrier through which substances move by facilitated diffusion.
b. Capillary arrangement
   Capillary density varies directly with tissue metabolic rates.
c. Regulation of capillary blood flow
   (1) Precapillary sphincters regulate capillary blood flow.
   (2) Precapillary sphincters open when cells are low in oxygen and nutrients and close when cellular needs are met.
d. Exchanges in the capillaries
   (1) Gases, nutrients, and metabolic by-products are exchanged between the capillary blood and the tissue fluid.
   (2) Diffusion provides the most important means of transport.
   (3) Diffusion pathways depend on lipid solubilities.
   (4) Plasma proteins generally remain in the blood.
   (5) Filtration, due to the hydrostatic pressure of blood, causes a net outward movement of fluid at the arteriolar end of a capillary.
   (6) Osmosis due to colloid osmotic pressure causes a net inward movement of fluid at the venular end of a capillary.
   (7) Some factors cause fluids to accumulate in the tissues.

3. 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 muscle and elastic tissue.
   d. Many veins contain flaplike valves that open, allowing blood to flow to the heart, but close to prevent flow in the opposite direction.

15.5 BLOOD PRESSURE (PAGE 582)

Blood pressure is the force blood exerts against the inner walls of blood vessels.

1. Arterial blood pressure
   a. The arterial blood pressure is produced primarily by heart action; it rises and falls with phases of the cardiac cycle.
   b. Systolic pressure occurs when the ventricle contracts; diastolic pressure occurs when the ventricle relaxes.
2. Factors that influence arterial blood pressure
   a. Cardiac output, blood volume, peripheral resistance, and blood viscosity influence arterial blood pressure.
   b. Arterial 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. Cardiac output depends on the volume of blood discharged from the ventricle with each beat (stroke volume) and on the heart rate.
      (1) The more blood that enters the heart, the stronger the ventricular contraction, the greater the stroke volume, and the greater the cardiac output.
      (2) The cardiac center of the medulla oblongata regulates heart rate.
   c. Changes in the diameter of arterioles, controlled by the vasomotor center of the medulla oblongata, regulate peripheral resistance.
4. Venous blood flow
   a. Venous blood flow is not a direct result of heart action; it depends on skeletal muscle contraction, breathing movements, and venoconstriction.
   b. Venoconstriction can increase venous pressure and blood flow.
5. Central venous pressure
   a. Central venous pressure is the pressure in the right atrium.
   b. Factors that influence it alter the flow of blood into the right atrium.
   c. It affects pressure in the peripheral veins.

15.6 PATHS OF CIRCULATION (PAGE 591)

1. Pulmonary circuit
   a. The pulmonary circuit consists of vessels that carry blood from the right ventricle to the alveolar capillaries in the lungs, and vessels that lead back to the left atrium.
   b. Alveolar capillaries exert less pressure than those of the systemic circuit.
   c. Tightly joined epithelial cells of alveoli walls prevent most substances from entering the alveoli.
   d. Osmotic pressure rapidly draws water out of alveoli into the interstitial fluid, so alveoli do not fill with fluid.
2. Systemic circuit
   a. The systemic circuit is composed of vessels that lead from the left ventricle to all body parts (including vessels supplying the heart itself) and back to the heart.
   b. It includes the aorta and its branches as well as the system of veins that return blood to the right atrium.

15.7 ARTERIAL SYSTEM (PAGE 593)

1. Principal branches of the aorta
   a. The branches of the ascending aorta include the right and left coronary arteries.
   b. The branches of the aortic arch include the 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 terminates by dividing into right and left common iliac arteries.

2. Arteries to the brain, head, and neck include branches of the subclavian and common carotid arteries.
3. Arteries to the shoulder and upper limb
   a. The subclavian artery passes into the arm, and in various regions, it 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
   a. The common iliac artery supplies the pelvic organs, gluteal region, and lower limb.
   b. The femoral artery of the lower limb becomes the popliteal artery that branches into the anterior and posterior tibial arteries.

15.8 VENOUS SYSTEM (PAGE 601)

1. Characteristics of venous pathways
   a. The 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. The 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 the upper limb.
   b. Digital veins drain into pairs of radial veins and ulnar veins, which merge to form a pair of brachial veins.
   c. The major superficial veins are the basilic and cephalic veins.
   d. Basilic and brachial veins merge to form the axillary vein.
   e. The median cubital vein in the bend of the elbow is often used as a site for venipuncture.
4. Tributaries of the brachiocephalic and azygos veins drain the abdominal and thoracic walls.
5. Veins from the abdominal viscera
   a. The blood from the abdominal viscera generally enters the hepatic portal system and is carried to the liver.
   b. The blood in the hepatic portal system is rich in nutrients.
   c. The liver helps regulate the blood concentrations of glucose, amino acids, and lipids.
   d. Phagocytic cells in the liver remove bacteria from the portal blood.
   e. 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.

15.9 LIFE-SPAN CHANGES (PAGE 607)

1. Plaque build-up may begin early.
2. Fibrous connective tissue and adipose tissue enlarge the heart by filling in when the number and size of cardiac muscle cells fall.
3. Heart rate and output decline slightly with age.
4. Blood pressure increases with age, while resting heart rate decreases with age.
5. Moderate exercise correlates to lowered risk of heart disease in older people.

CHAPTER ASSESSMENTS

15.1 Introduction

1 Match the structure and its function. (p. 555)

(1) arteries	A. sites of nutrient, electrolyte, gas, and waste exchange
(2) arterioles	B. muscular pump that forces blood through arteries
(3) capillaries	C. branch into capillaries
(4) venules	D. capillaries converge into these
(5) veins	E. transport blood away from the heart
(6) heart	F. transport blood to the heart

15.2 Structure of the Heart

2 Describe the pericardium. (p. 556)
3 Compare the layers of the heart wall. (p. 558)
4 Draw a heart and label the chambers and valves. (p. 559)
5 Blood flows through the vena cavae and coronary sinus into the right atrium, through the _____ to the right ventricle, through the pulmonary valve to the pulmonary trunk into the right and left _____ to the lungs, then leaves the lungs through the pulmonary veins and flows into the _____, through the mitral valve to the _____, and through the _____ to the aorta. (p. 562)
6 List the vessels through which blood flows from the aorta to the myocardium and back to the right atrium. (p. 563)

15.3 Heart Actions

7 Describe the pressure changes in the atria and ventricles during a cardiac cycle. (p. 566)
8 Explain the origins of heart sounds. (p. 566)
9 Describe the arrangement of cardiac muscle fibers. (p. 567)
10 Distinguish between the roles of the SA node and AV node. (p. 567)
11 Explain how the cardiac conduction system controls the cardiac cycle. (p. 568)
12 Describe and explain the normal ECG pattern. (p. 570)
13 Discuss how the nervous system regulates the cardiac cycle. (p. 571)
14 Describe two factors other than the nervous system that affect the cardiac cycle. (p. 574)

15.4 Blood Vessels

15 Distinguish between an artery and an arteriole. (p. 576)
16 Explain control of vasoconstriction and vasodilation. (p. 578)
17 Describe the structure and function of a capillary. (p. 578)
18 Describe the function of the blood-brain barrier. (p. 579)
19 Explain control of blood flow through a capillary. (p. 580)
20 Relate how diffusion functions in the exchange of substances between blood plasma and tissue fluid. (p. 580)
21 Explain why water and dissolved substances leave the arteriolar end of a capillary and enter the venular end. (p. 580)
22 Describe the effect of histamine on a capillary. (p. 580)
23 Distinguish between a venule and a vein. (p. 581)
24 Explain how veins function as blood reservoirs. (p. 581)

15.5 Blood Pressure

25 Arterial blood pressure peaks when the ventricles contract. This maximum pressure achieved is called the _____. (p. 582)
26 Name several factors that influence blood pressure, and explain how each produces its effect. (p. 584)
27 Describe the control of blood pressure. (p. 586)
28 List the major factors that promote venous blood flow. (p. 588)
29 Define *central venous pressure.* (p. 589)

15.6 Paths of Circulation

30 Distinguish between the pulmonary and systemic circuits of the cardiovascular system. (p. 591)
31 Trace the path of blood through the pulmonary circuit. (p. 591)
32 Explain why the alveoli normally do not fill with fluid. (p. 592)

15.7–15.8 Arterial System–Venous System

33 Describe the aorta, and name its principal branches. (pp. 593-598)
34 Discuss the relationship between the major venous pathways and the major arterial pathways to the head, upper limbs, abdominal viscera, and lower limbs. (pp. 601-607)

15.9 Life-Span Changes

35 List and discuss changes in the aging cardiovascular system. (p. 607)

INTEGRATIVE ASSESSMENTS/CRITICAL THINKING

OUTCOMES 5.5, 9.6, 15.2, 15.3

1. What structures and properties should an artificial heart have?

OUTCOMES 15.2, 15.3

2. Why is ventricular fibrillation more likely to be life-threatening than atrial fibrillation?

OUTCOMES 15.2, 15.4, 15.6, 15.7

3. If a cardiologist inserts a catheter into a patient's right femoral artery, which arteries will the tube have to pass through to reach the entrance of the left coronary artery?

OUTCOMES 15.3, 15.4, 15.5, 15.6

4. How might the results of a cardiovascular exam differ for an athlete in top condition and a sedentary, overweight individual?

5. Cigarette smoke contains thousands of chemicals, including nicotine and carbon monoxide. Nicotine constricts blood vessels. Carbon monoxide prevents oxygen binding to hemoglobin. How do these two components of smoke affect the cardiovascular system?

OUTCOME 15.4

6. Given the way capillary blood flow is regulated, do you think it is wiser to rest or to exercise following a heavy meal? Cite a reason for your answer.

OUTCOMES 15.4, 15.5, 15.6, 15.8

7. Cirrhosis of the liver, a disease commonly associated with alcoholism, obstructs blood flow through the hepatic blood vessels. As a result the blood backs up, and the capillary pressure greatly increases in the organs drained by the hepatic portal system. What effects might this increasing capillary pressure produce, and which organs would it affect?

OUTCOMES 15.6, 15.7, 15.8

8. 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?