21

Blood Vessels and Circulation

Learning Outcomes

These Learning Outcomes correspond by number to this chapter's sections and indicate what you should be able to do after completing the chapter.

21-1 Distinguish among the types of **blood vessels** based on their structure and function, and describe how and where **fluid and dissolved materials** enter and leave the cardiovascular system.

21-2 Explain the mechanisms that regulate **blood flow through vessels**, describe the factors that influence **blood pressure**, and discuss the mechanisms that regulate **movement of fluids** between capillaries and interstitial spaces.

21-3 Describe the control mechanisms that regulate **blood flow and pressure in tissues**, and explain how the activities of the **cardiac, vasomotor, and respiratory centers** are coordinated to control blood flow through the tissues.

21-4 Explain the cardiovascular system's **homeostatic response** to exercise and hemorrhaging, and identify the principal blood vessels and functional characteristics of the special **circulation to the brain, heart, and lungs**.

21-5 Describe the three **general functional patterns** seen in the **pulmonary and systemic circuits** of the cardiovascular system.

21-6 Identify the major arteries and veins of the **pulmonary circuit**.

21-7 Identify the major arteries and veins of the **systemic circuit**.

21-8 Identify the differences between **fetal and adult circulation patterns**, and describe the changes in the patterns of blood flow that occur at birth.

21-9 Discuss the **effects of aging** on the cardiovascular system, and give examples of interactions between the cardiovascular system and other organ systems.

Clinical Notes

Spotlight

An Introduction to Blood Vessels and Circulation

Blood circulates throughout the body, moving from the heart through the tissues and back to the heart, in blood vessels. In this chapter we examine the organization of blood vessels and consider the integrated functions of the cardiovascular system as a whole. We begin with a description of the histological organization of arteries, capillaries, and veins. Then we explore the functions of these vessels, the basic principles of cardiovascular regulation, and the distribution of major blood vessels in the body. We will then be ready to consider the organization and function of the lymphatic system, the focus of Chapter 22.

21-1 Arteries, arterioles, capillaries, venules, and veins differ in size, structure, and functional properties

The cardiovascular system has five general classes of blood vessels. **Arteries** carry blood away from the heart. As they enter peripheral tissues, arteries branch repeatedly, and the branches decrease in diameter. The smallest arterial branches are called **arterioles** (ar-TĒR-ē-ōls). From the arterioles, blood moves into **capillaries,** where diffusion takes place between blood and interstitial fluid. From the capillaries, blood enters small **venules** (VEN-ūls), which unite to form larger **veins** that return blood to the heart.

Blood leaves the heart through the pulmonary trunk, which originates at the right ventricle, and the aorta, which originates at the left ventricle. Each of these arterial trunks has an internal diameter of about 2.5 cm (1 in.). The pulmonary arteries that branch from the pulmonary trunk carry blood to the lungs. The systemic arteries that branch from the aorta distribute blood to all other organs. Within these organs, the vessels branch into several hundred million tiny arterioles that provide blood to more than 10 billion capillaries within their own branching networks. These capillaries are barely the diameter of a single red blood cell. If all the capillaries in your body were placed end to end, their combined length would be more than 25,000 miles, enough to circle the planet.

The vital functions of the cardiovascular system depend entirely on events at the capillary level: All chemical and gaseous exchange between blood and interstitial fluid takes place across capillary walls. Cells rely on capillary diffusion to obtain nutrients and oxygen and to remove metabolic wastes, such as carbon dioxide and urea. Diffusion takes place very rapidly, because the distances involved are very short. Few cells lie farther than 125 μm (0.005 in.) from a capillary. As we will see, homeostatic mechanisms operating at the local, regional, and systemic levels adjust blood flow through the capillaries to meet the demands of peripheral tissues.

Blood vessels must be resilient enough to withstand changes in pressure, and flexible enough to move with underlying tissues and organs. The pressures inside vessels vary with distance from the heart, and the structures of different vessels reflect this fact. The arteries, veins, and capillaries also differ in function, and these functional differences are associated with distinctive anatomical features.

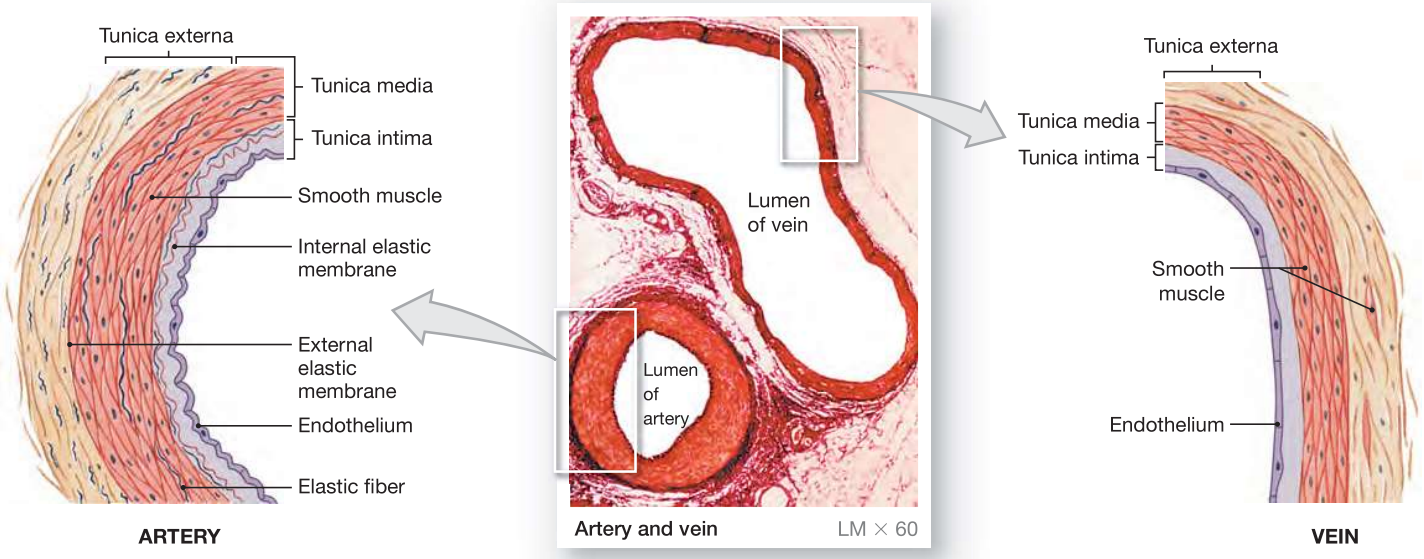
The Structure of Vessel Walls

The walls of arteries and veins have three distinct layers—the tunica intima, tunica media, and tunica externa (**Figure 21–1**):

1. The **tunica intima** (IN-ti-muh), or *tunica interna*, is the inner layer of a blood vessel. It includes the endothelial lining and a surrounding layer of connective tissue with a variable number of elastic fibers. In arteries, the outer margin of the tunica intima contains a thick layer of elastic fibers called the **internal elastic membrane.**
2. The **tunica media** is the middle layer of a blood vessel. It contains concentric sheets of smooth muscle tissue in a framework of loose connective tissue. The collagen fibers bind the tunica media to the tunica intima and tunica externa. The tunica media is commonly the thickest layer in a small artery. It is separated from the surrounding tunica externa by a thin band of elastic fibers called the **external elastic membrane.** The smooth muscle cells of the tunica media encircle the endothelium that lines the lumen of the blood vessel. When these smooth muscles contract, the vessel decreases in diameter, and when they relax, the diameter increases. Large arteries also contain layers of longitudinally arranged smooth muscle cells.
3. The **tunica externa** (eks-TER-nuh) or *tunica adventitia* (adven-TISH-a) is the outer layer of a blood vessel. It is a connective tissue sheath. In arteries, it contains collagen fibers with scattered bands of elastic fibers. In veins, it is generally thicker than the tunica media and contains networks of elastic fibers and bundles of smooth muscle cells. The connective tissue fibers of the tunica externa typically blend into those of adjacent tissues, stabilizing and anchoring the blood vessel.

Their layered walls give arteries and veins considerable strength. The muscular and elastic components also permit controlled changes in diameter as blood pressure or blood volume changes. However, the walls of arteries and veins are too thick to allow diffusion between the bloodstream and surrounding tissues, or even between the blood and the tissues of the vessel itself. For this reason, the walls of large vessels contain small arteries and veins that supply the smooth muscle cells and fibroblasts of the tunica media and tunica externa.

Figure 21–1 Comparisons of a Typical Artery and a Typical Vein.



Feature	Typical Artery	Typical Vein
GENERAL APPEARANCE IN SECTIONAL VIEW	Usually round, with relatively thick wall	Usually flattened or collapsed, with relatively thin wall
TUNICA INTIMA		
Endothelium	Usually rippled, due to vessel constriction	Often smooth
Internal elastic membrane	Present	Absent
TUNICA MEDIA	Thick, dominated by smooth muscle cells and elastic fibers	Thin, dominated by smooth muscle cells and collagen fibers
External elastic membrane	Present	Absent
TUNICA EXTERNA	Collagen and elastic fibers	Collagen and elastic fibers and smooth muscle cells

These blood vessels are called the *vasa vasorum* ("vessels of vessels").

Differences between Arteries and Veins

Arteries and veins supplying the same region lie side by side (**Figure 21–1**). In sectional view, you can distinguish arteries and veins by the following features:

- *Vessel walls* In general, the walls of arteries are thicker than those of veins. The tunica media of an artery contains more smooth muscle and elastic fibers than does that of a vein. These components help resist the arterial pressure generated by the heart as it pumps blood into the pulmonary trunk and aorta.
- *Vessel lumen* When not opposed by blood pressure, the elastic fibers in the arterial walls recoil, constricting the lumen. Thus, seen on dissection or in sectional view, the lumen of an artery often looks smaller than that of the corresponding vein. Because the walls of arteries are relatively thick and strong, they keep their circular shape in section. In contrast, cut veins tend to collapse. In section, these veins often look flattened or grossly distorted.
- *Vessel lining* The endothelial lining of an artery cannot contract, so when an artery constricts, its endothelium is thrown into folds that give sectioned arteries a pleated appearance. The lining of a vein lacks these folds.

In gross dissection, arteries and veins can generally be distinguished because:

- The thicker walls of arteries can be felt if the vessels are compressed.
- Arteries usually keep their cylindrical shape, but veins often collapse.
- Arteries are more resilient: When stretched, they keep their shape and elongate. When released, they snap back. A

small vein cannot tolerate as much distortion without collapsing or tearing.

- Veins typically contain *valves*—internal structures that prevent the backflow of blood toward the capillaries. In an intact vein, the location of each valve is marked by a slight distension of the vessel wall. (We consider valve structure in a later section.)

Arteries

Their relatively thick, muscular walls make arteries elastic and contractile. Elasticity permits the vessel diameter to change passively in response to changes in blood pressure. For example, it allows arteries to absorb the surging pressure waves that accompany the contractions of the ventricles.

The contractility of the arterial walls enables them to actively change diameter. This change takes place primarily under the control of the sympathetic division of the autonomic nervous system. When stimulated, arterial smooth muscles contract, constricting the artery—a process called **vasoconstriction.** When these smooth muscles relax, the diameter of the lumen increases—a process called **vasodilation.** Vasoconstriction and vasodilation affect (1) the afterload on the heart, (2) peripheral blood pressure, and (3) capillary blood flow. We explore these effects in a later section. Contractility is also important during the vascular phase of hemostasis, when the contraction of a damaged vessel wall helps reduce bleeding. ↩ p. 661

In traveling from the heart to peripheral capillaries, blood passes through *elastic arteries*, *muscular arteries*, and *arterioles* (**Figure 21–2**).

Elastic Arteries

Elastic arteries are also known as *conducting arteries* because they carry large volumes of blood away from the heart. They are large vessels with diameters up to 2.5 cm (1 in.) (**Figure 21–2**). The pulmonary trunk and aorta, as well as their major branches (the *pulmonary*, *common carotid*, *subclavian*, and *common iliac arteries*), are elastic arteries.

The walls of elastic arteries are extremely resilient because the tunica media contains a high density of elastic fibers and relatively few smooth muscle cells. As a result, elastic arteries can tolerate the pressure changes of the cardiac cycle. We have already seen that elastic rebound in the aorta helps to maintain blood flow in the coronary arteries. ↩ p. 680 Elastic rebound also occurs to some degree in all elastic arteries. During ventricular systole, pressures rise rapidly and the elastic arteries expand as the stroke volume is ejected. During ventricular diastole, blood pressure within the arterial system falls and the elastic fibers recoil to their original dimensions. Their expansion cushions the sudden rise in pressure during ventricular systole, and their recoil slows the drop in pressure during ventricular diastole. In this way, elastic arteries help to make blood flow continuous.

This function is important because blood pressure is the driving force behind blood flow: The greater the pressure oscillations, the greater the changes in blood flow. The elasticity of the arterial system dampens the pressure peaks and valleys that accompany the heartbeat. By the time blood reaches the arterioles, the pressure oscillations have disappeared, and blood flow is continuous.

Muscular Arteries

Muscular arteries, or *medium-sized arteries,* are also known as *distribution arteries* because they distribute blood to the body's skeletal muscles and internal organs. Most of the vessels of the arterial system are muscular arteries. They are characterized by a thick tunica media. It contains more smooth muscle cells than does the tunica media of elastic arteries (**Figures 21–1** and **21–2**). A typical muscular artery has a lumen diameter of approximately 4.0 mm (0.16 in.), but some have diameters as small as 0.5 mm. The *external carotid arteries* of the neck, the *brachial arteries* of the arms, the *mesenteric arteries* of the abdomen, and the *femoral arteries* of the thighs are examples of muscular arteries. Superficial muscular arteries are important as *pressure points*—places in the body where muscular arteries can be pressed against deeper bones to reduce blood flow and control severe bleeding. Major arterial pressure points are the common carotid, radial, brachial, femoral, popliteal, posterior tibial, and dorsal pedal.

Arterioles

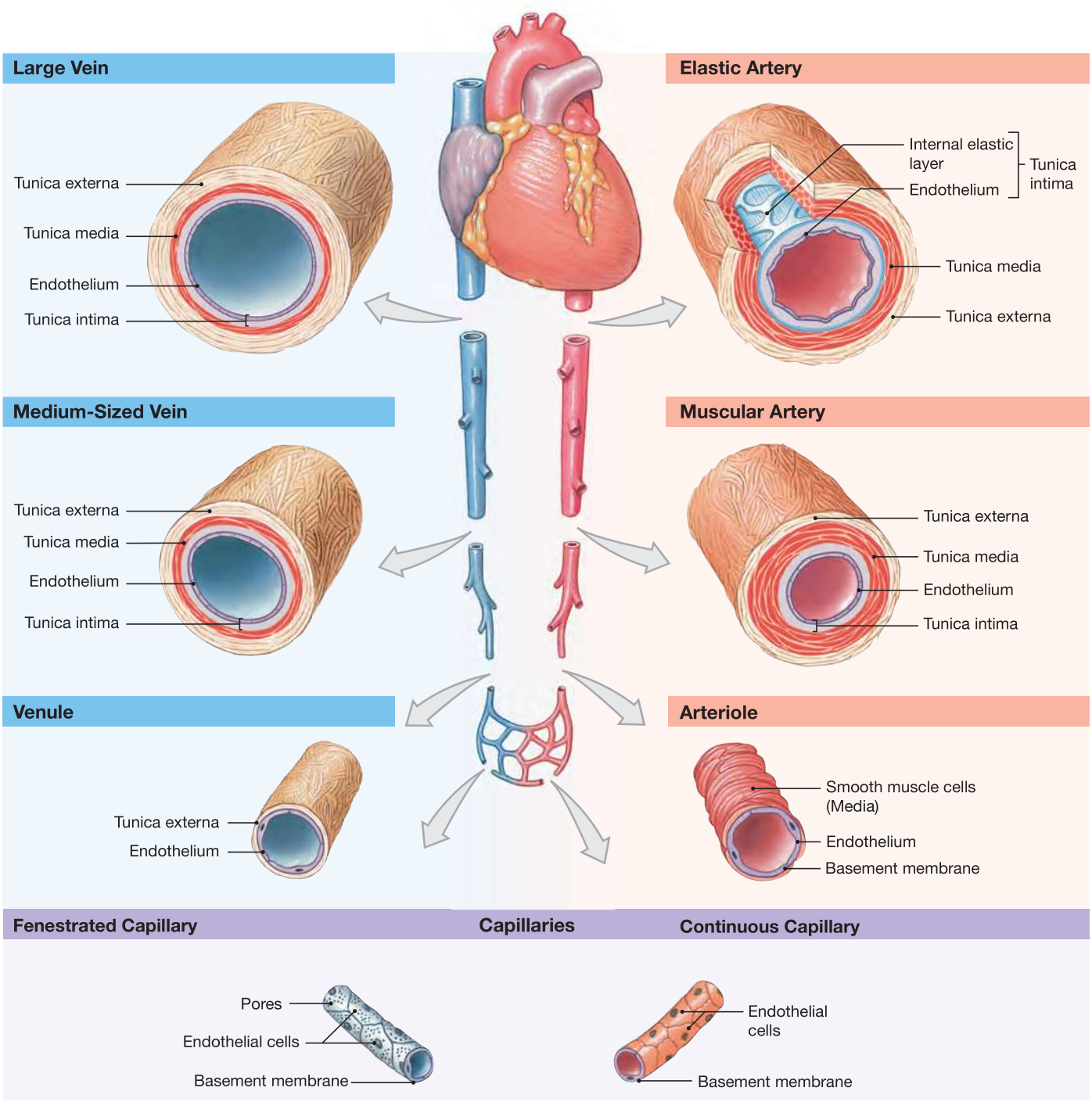
Arterioles, with an internal diameter of 30 μm or less, are considerably smaller than muscular arteries. Arterioles have a poorly defined tunica externa. In the larger arterioles, the tunica media consists of one or two layers of smooth muscle cells (**Figure 21–2**). In the smallest arterioles, the tunica media contains scattered smooth muscle cells that do not form a complete layer.

The diameters of smaller muscular arteries and arterioles change in response to local conditions or to sympathetic or endocrine stimulation. For example, arterioles in most tissues vasodilate when oxygen levels are low. Also, as we saw in Chapter 16, arterioles vasoconstrict under sympathetic stimulation. ↩ p. 525 Changes in their diameter affect the amount of force required to push blood around the cardiovascular system: More pressure is required to push blood through a constricted vessel than through a dilated one. The force opposing blood flow is called **resistance (R),** so arterioles are also called **resistance vessels.**

Vessel characteristics change gradually with distance from the heart. Each type of vessel described here actually represents the midpoint in a portion of a continuum. Thus, the largest muscular arteries contain a considerable amount of elastic tissue, and the smallest resemble heavily muscled arterioles.

Arteries carry blood under great pressure, and their walls are adapted to handle that pressure. Occasionally, local arterial pressure exceeds the capacity of the elastic components of the tunics.

Figure 21–2 Histological Structure of Blood Vessels. Representative diagrammatic cross-sectional views of the walls of arteries, capillaries, and veins. Notice the relative sizes of the layers in these vessels.



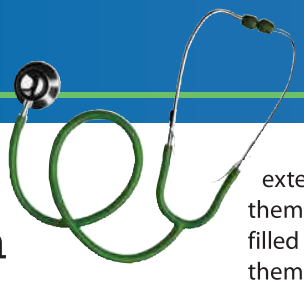
The result is an **aneurysm** (AN-ū-rizm), or bulge in the weakened wall of an artery. The bulge is like a bubble in the wall of a tire—and like a bad tire, the artery can suffer a catastrophic blowout. The most dangerous aneurysms occur in arteries of the brain (where they cause strokes) or in the aorta (where a rupture will cause fatal bleeding in a matter of minutes).

Capillaries

When we think of the cardiovascular system, we think first of the heart or the great blood vessels connected to it. But the microscopic capillaries that permeate most tissues do the real work of the cardiovascular system. These delicate vessels weave

Clinical Note

Arteriosclerosis



These aren't **plaques** you hang on the wall

Arteriosclerosis (ar-tēr-ē-ō-skler-Ō-sis; *arterio-*, artery + *sklerosis*, hardness) is a thickening and toughening of arterial walls. This condition may not sound life-threatening, but complications related to arteriosclerosis account for about half of all deaths in the United States. The effects of arteriosclerosis are varied. For example, arteriosclerosis of coronary vessels is responsible for *coronary artery disease (CAD)*, and arteriosclerosis of arteries supplying the brain can lead to strokes. ↩ p. 682 Arteriosclerosis takes two major forms:

1. **Focal calcification** is the deposition of calcium salts following the gradual degeneration of smooth muscle in the tunica media. Some focal calcification is a part of the aging process, and it may develop in association with atherosclerosis (described next). Rapid and severe calcification may take place as a complication of diabetes mellitus, an endocrine disorder. ↩ p. 622
2. **Atherosclerosis** (ath-er-ō-skler-Ō-sis; *athero-*, fatty degeneration) is the formation of lipid deposits in the tunica media associated with damage to the endothelial lining. It is the most common form of arteriosclerosis.

Many factors may be involved in the development of atherosclerosis. One major factor is lipid levels in the blood. Atherosclerosis tends to develop in people whose blood contains elevated levels of plasma lipids—specifically, cholesterol. Circulating cholesterol is transported to peripheral tissues in *lipoproteins*, which are protein–lipid complexes. (We will discuss the various types of lipoproteins in Chapter 25.)

When plasma cholesterol levels are chronically elevated, cholesterol-rich lipoproteins remain in circulation for an extended period. Circulating monocytes then begin removing them from the bloodstream. Eventually, the monocytes become filled with lipid droplets. Now called *foam cells*, they attach themselves to the endothelial walls of blood vessels, where they release cytokines. These growth factors stimulate smooth muscle cells near the tunica intima to divide, thickening the vessel wall.

Other monocytes then invade the area, migrating between the endothelial cells. As these changes take place, the monocytes, smooth muscle cells, and endothelial cells begin phagocytizing lipids as well. The result is an atherosclerotic **plaque,** a fatty mass of tissue that projects into the lumen of the vessel. At this point, the plaque has a relatively simple structure, and evidence suggests that the process can be reversed with appropriate dietary adjustments.

If the conditions persist, the endothelial cells become swollen with lipids, and gaps appear in the endothelial lining. Platelets now begin sticking to the exposed collagen fibers. The combination of platelet adhesion and aggregation leads to the formation of a localized blood clot, which further restricts blood flow through the artery. The structure of the plaque is now relatively complex.

A typical plaque is shown in **Figure 21–3**. Elderly individuals—especially elderly men—are most likely to develop atherosclerotic plaques. Estrogens may slow plaque formation, which may account for the lower incidence of CAD, myocardial infarctions (MIs), and strokes in women. After menopause, when estrogen production declines, the risks of CAD, MIs, and strokes in women increase markedly.

In addition to advanced age and male gender, other important risk factors for atherosclerosis include high blood cholesterol levels, high blood pressure, and cigarette smoking. Roughly 20 percent of middle-aged men have all three of these risk factors. These individuals are four times more likely to experience an MI or a cardiac arrest than other men in their age group. Fewer women develop atherosclerotic plaques, but

throughout active tissues, forming intricate networks that surround muscle fibers. Capillaries radiate through connective tissues, and branch beneath the basement membrane of epithelia.

Capillaries are the *only* blood vessels whose walls permit exchange between the blood and the surrounding interstitial fluids. Exchange can take place quickly because capillary walls are thin and diffusion distances are short. In addition, blood flows through capillaries relatively slowly, allowing sufficient time for the diffusion or active transport of materials across the capillary walls. In this way, the histological structure of capillaries permits a two-way exchange of substances between blood and interstitial fluid.

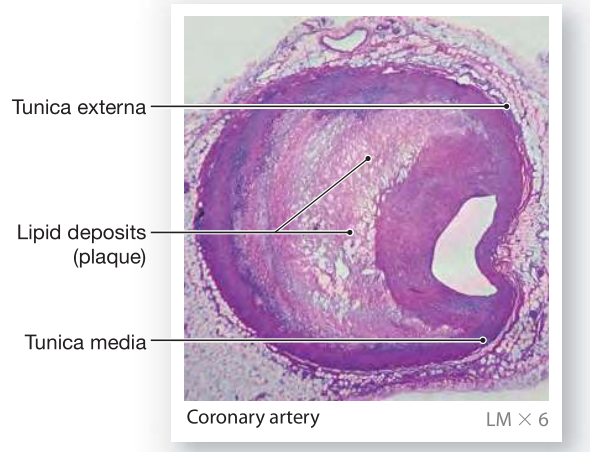
A typical capillary consists of an endothelial tube inside a thin basement membrane. Neither a tunica media nor a tunica externa is present (**Figure 21–2**). The average diameter of a capillary is a mere 8 μm, very close to that of a single red blood cell. The two major types of capillaries are *continuous capillaries* and *fenestrated capillaries*.

Continuous Capillaries

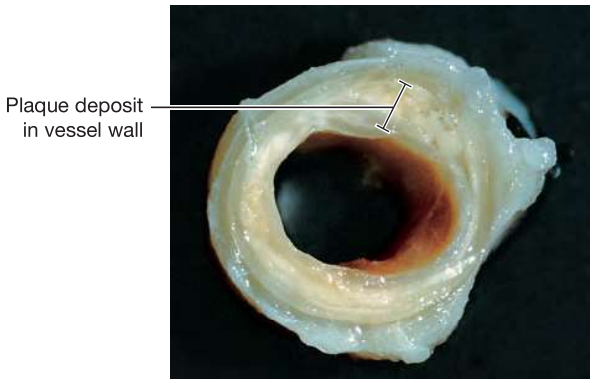
Most regions of the body are supplied by continuous capillaries. In a **continuous capillary,** the endothelium is a complete lining. A cross section through a large continuous capillary cuts across several endothelial cells (**Figure 21–4a**). In a small continuous capillary, a single endothelial cell may completely encircle the lumen.

Continuous capillaries are located in all tissues except epithelia and cartilage. Continuous capillaries permit the water, small solutes, and lipid-soluble materials to diffuse into the interstitial fluid. At the same time, they prevent the loss of blood

Figure 21–3 A Plaque within an Artery.



a A cross-sectional view of a large plaque



b A section of a coronary artery narrowed by plaque formation

elderly female smokers with high blood cholesterol and high blood pressure are at much greater risk than other women. Diabetes mellitus, obesity, and stress can promote the development of atherosclerosis in both men and women. Evidence also indicates that at least some forms of atherosclerosis may be linked to chronic infection with *Chlamydia pneumoniae*, a bacterium responsible for several types of respiratory infections, including some forms of pneumonia.

We discussed potential treatments for atherosclerotic plaques, such as catheterization with balloon angioplasty and stenting, and bypass surgery, in Chapter 20. ↩ p. 683 In the many cases where changes in diet do not lower circulating LDL levels sufficiently, drug therapies can bring them under control. Genetic engineering techniques have been used to treat an inherited form of *hypercholesterolemia* (high blood cholesterol) linked to extensive plaque formation. (Individuals with this condition are unable to absorb and recycle cholesterol in the liver.) In this experimental procedure, circulating cholesterol levels declined after copies of appropriate genes were inserted into some of the individual's liver cells.

Without question, the best approach to atherosclerosis is to avoid it by eliminating or reducing associated risk factors. Suggestions include (1) reducing your intake of dietary cholesterol, saturated fats, and trans fatty acids by restricting consumption of fatty meats (such as beef, lamb, and pork), egg yolks, and cream; (2) not smoking; (3) checking your blood pressure and taking steps to lower it if necessary; (4) having your blood cholesterol levels checked annually; (5) controlling your weight; and (6) exercising regularly.

cells and plasma proteins. In addition, some exchange may occur between blood and interstitial fluid by *bulk transport*—the movement of materials by endocytosis (via endosomes) or exocytosis at the inner endothelial surface. ↩ p. 92

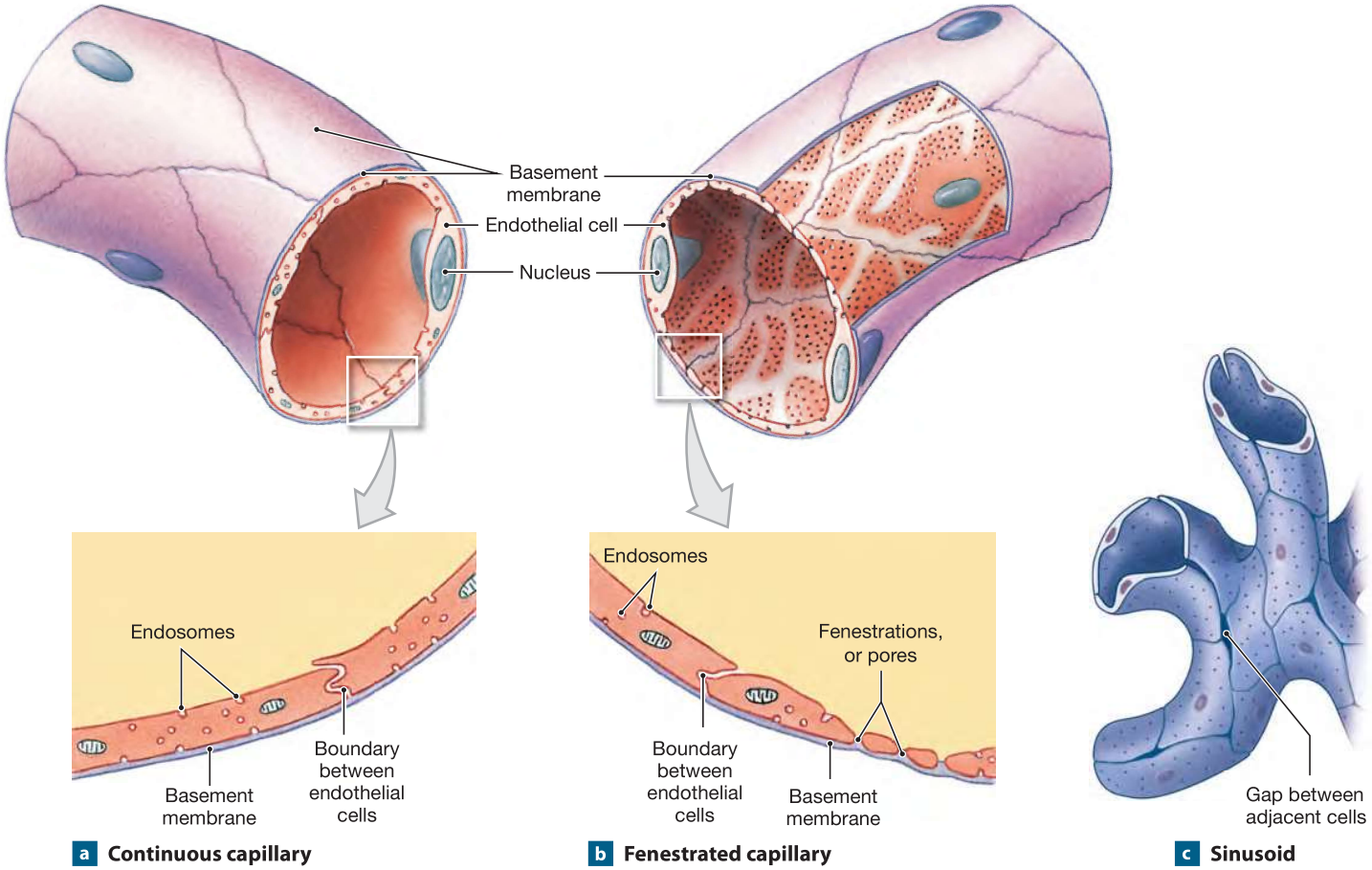
In specialized continuous capillaries in most of the central nervous system and in the thymus, the endothelial cells are bound together by tight junctions. These capillaries have very restricted permeability. We discussed one example—the capillaries responsible for the *blood–brain barrier*—in Chapters 12 and 14. ↩ pp. 381, 455

Fenestrated Capillaries

Fenestrated (FEN-es-trā-ted; *fenestra*, window) **capillaries** contain "windows," or pores, that penetrate the endothelial lining (**Figure 21–4b**). The pores allow rapid exchange of water and solutes between plasma and interstitial fluid. Examples of fenestrated capillaries include the *choroid plexus* of the brain and the blood vessels in a variety of endocrine organs, such as the hypothalamus and the pituitary, pineal, and thyroid glands. Fenestrated capillaries are also found along absorptive areas of the intestinal tract and at filtration sites in the kidneys. Both the number of pores and their permeability characteristics may vary from one region of the capillary to another.

Sinusoids (SĪ-nuh-soydz), also called **sinusoidal capillaries,** resemble fenestrated capillaries that are flattened and irregularly shaped (**Figure 21–4c**). In addition to being fenestrated, sinusoids commonly have gaps between adjacent endothelial cells, and the basement membrane is either thinner or absent. As a result, sinusoids permit the free exchange of water and solutes as large as plasma proteins between blood and interstitial fluid.

Figure 21–4 Capillary Structure.



Blood moves through sinusoids relatively slowly, maximizing the time available for exchange across the sinusoidal walls. Sinusoids occur in the liver, bone marrow, spleen, and many endocrine organs, including the pituitary and adrenal glands. At liver sinusoids, plasma proteins secreted by liver cells enter the bloodstream. Along sinusoids of the liver, spleen, and bone marrow, phagocytic cells monitor the passing blood, engulfing damaged red blood cells, pathogens, and cellular debris.

Capillary Beds

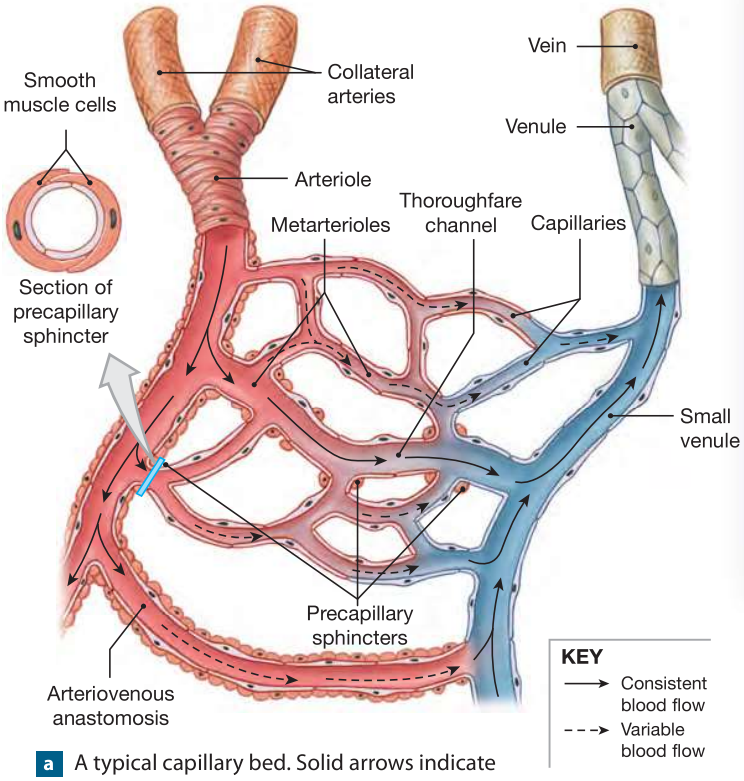
Capillaries function not as individual units, but rather, as part of an interconnected network called a **capillary bed,** or **capillary plexus** (Figure 21–5). A single arteriole generally gives rise to dozens of capillaries. They empty into several *venules*, the smallest vessels of the venous system. The entrance to each capillary is guarded by a **precapillary sphincter.** Contraction of the smooth muscle cells of this sphincter narrows the capillary entrance, reducing or stopping the flow of blood. When one precapillary sphincter constricts, blood is diverted into other branches of the network. When a precapillary sphincter relaxes, the entrance dilates, and blood flows into the capillary.

A capillary bed contains several direct connections between arterioles and venules. The wall in the first part of such a passageway contains smooth muscle that can change its diameter. This segment is called a **metarteriole** (met-ar-TĒR-ē-ōl) or **precapillary arteriole.** The rest of the passageway resembles a typical capillary in structure and is called a **thoroughfare channel.**

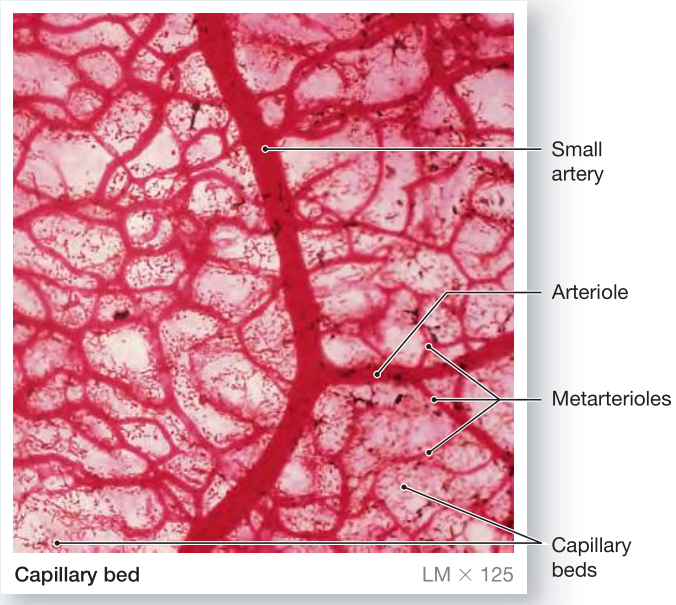
More than one artery may supply blood to a capillary bed. The multiple arteries are called **collaterals.** They fuse before giving rise to arterioles. The fusion of two collateral arteries that supply a capillary bed is an example of an **arterial anastomosis.** (An *anastomosis* is the joining of blood vessels.) The interconnections between the *anterior* and *posterior interventricular arteries* of the heart are arterial anastomoses. ↩ p. 680 An arterial anastomosis acts like an insurance policy: If one artery is compressed or blocked, capillary circulation will continue.

Arteriovenous (ar-tēr-ē-ō-VĒ-nus) **anastomoses** are direct connections between arterioles and venules. When an arteriovenous anastomosis is dilated, blood bypasses the capillary bed and flows directly into the venous circulation. The pattern of blood flow through an arteriovenous anastomosis is regu-

Figure 21–5 The Organization of a Capillary Bed.



a A typical capillary bed. Solid arrows indicate consistant blood flow; dashed arrows indicate variable or pulsating blood flow.



b A micrograph of a number of capillary beds.

lated primarily by sympathetic innervation under the control of the cardiovascular centers of the medulla oblongata.

Angiogenesis (an-jē-ō-JEN-e-sis; *angio-*, blood vessel + *genesis*, production) is the formation of new blood vessels and occurs under the direction of **vascular endothelial growth factor (VEGF).** Angiogenesis occurs in the embryo as tissues and organs develop. It may also occur at other times in any body tissue in response to factors released by cells that are *hypoxic*, or oxygen-starved. Clinically, angiogenesis is probably most important in cardiac muscle, where it takes place in response to a chronically constricted or occluded vessel.

Vasomotion

Although blood normally flows from arterioles to venules at a constant rate, the flow within each capillary varies. Each precapillary sphincter contracts and relaxes, perhaps a dozen times per minute. As a result, the blood flow within any capillary occurs in pulses rather than as a steady and constant stream. The net effect is that blood may reach the venules by one route now and by a different route later. The cycling of contraction and relaxation of smooth muscles that changes blood flow through capillary beds is called **vasomotion.**

Vasomotion is controlled locally by changes in the concentrations of chemicals and dissolved gases in the interstitial fluids. For example, when dissolved oxygen concentrations decline within a tissue, the capillary sphincters relax, so blood flow to the area increases. This process is an example of capillary *autoregulation*. We focus on it in a later section.

When you are at rest, blood flows through about 25 percent of the vessels within a typical capillary bed in your body. Your cardiovascular system does not contain enough blood to maintain adequate blood flow to all the capillaries in all the capillary beds in your body at the same time. As a result, when many tissues become active, the blood flow through capillary beds must be coordinated. We describe the mechanisms by which the cardiovascular centers perform this coordination later in the chapter.

Veins

Veins collect blood from all tissues and organs and return it to the heart. The walls of veins can be thinner than those of corresponding arteries because the blood pressure in veins is lower than that in arteries. We classify veins on the basis of their size.

Even though their walls are thinner, in general veins are larger in diameter than their corresponding arteries. (Review **Figure 21–2** to compare typical arteries and veins.)

Venules

Venules are the smallest venous vessels. They collect blood from capillary beds. They vary widely in size and structure. An average venule has an internal diameter of roughly 20 μm. Venules smaller than 50 μm lack a tunica media, and the smallest venules resemble expanded capillaries.

Medium-Sized Veins

Medium-sized veins are comparable in size to muscular arteries. They range from 2 to 9 mm in internal diameter. Their tunica media is thin and contains relatively few smooth muscle cells. The thickest layer of a medium-sized vein is the tunica externa, which contains longitudinal bundles of elastic and collagen fibers.

Large Veins

Large veins include the superior and inferior venae cavae and their tributaries within the abdominopelvic and thoracic cavities. All large veins have all three layers. The slender tunica media is surrounded by a thick tunica externa composed of a mixture of elastic and collagen fibers.

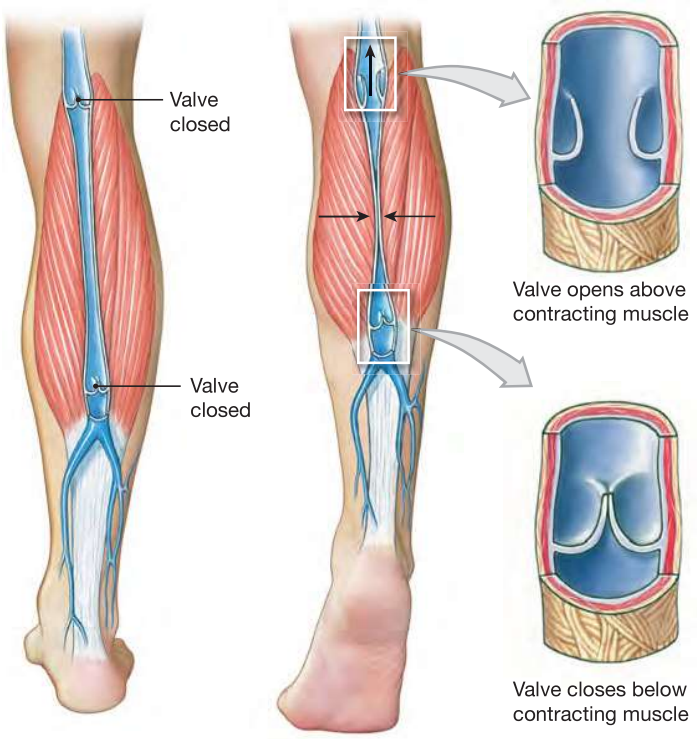
Venous Valves

The arterial system is a high-pressure system: Almost all the force developed by the heart is required to push blood along the network of arteries and through miles of capillaries. Blood pressure in a peripheral venule is only about 10 percent of that in the ascending aorta, and pressures continue to fall along the venous system.

The blood pressure in venules and medium-sized veins is so low that it cannot overcome the force of gravity. In the limbs, veins of this size contain **valves,** folds of the tunica intima that project from the vessel wall and point in the direction of blood flow. These valves, like those in the heart, permit blood flow in one direction only. Venous valves prevent blood from moving back toward the capillaries (**Figure 21–6**).

As long as the valves function normally, any movement that distorts or compresses a vein pushes blood toward the heart. This effect improves *venous return*, the rate of blood flow to the heart. ↩ p. 699 The mechanism is particularly important when you are standing, because blood returning from your feet must overcome gravity to ascend to the heart. Valves compartmentalize the blood within the veins, dividing the weight of the blood among the compartments. Any contraction of the surrounding skeletal muscles squeezes the blood toward the heart. Although you are probably not aware of it, when you stand, rapid cycles of contraction and relaxation occur within your leg muscles, helping to push blood toward the trunk. When you lie down, venous valves play a smaller part in venous return, because your heart and major vessels are at the same level.

Figure 21–6 The Function of Valves in the Venous System.



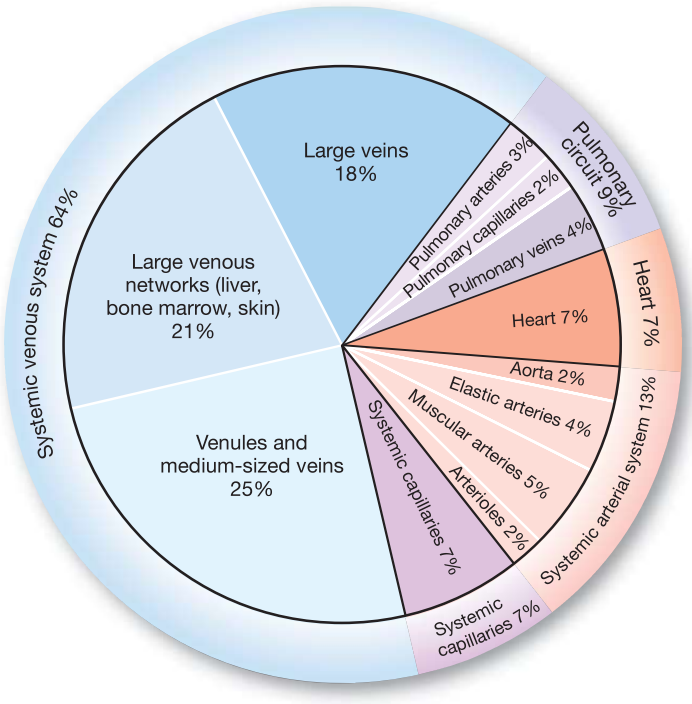
If the walls of the veins near the valves weaken or become stretched and distorted, the valves may not work properly. Blood then pools in the veins, and the vessels become grossly distended. The effects range from mild discomfort and a cosmetic problem, as in superficial **varicose veins** in the thighs and legs, to painful distortion of adjacent tissues, as in **hemorrhoids.**

The Distribution of Blood

Our total blood volume is unevenly distributed among arteries, veins, and capillaries (**Figure 21–7**). The heart, arteries, and capillaries in the pulmonary and systemic circuits normally contain 30–35 percent of the blood volume (roughly 1.5 liters of whole blood). The venous system contains the rest (65–70 percent, or about 3.5 liters). About one-third of the blood in the venous system (about a liter) is circulating within the liver, bone marrow, and skin. These organs have extensive venous networks that at any moment contain large volumes of blood.

Veins are much more *distensible,* or expandable, than arteries because their walls are thinner, with less smooth muscle. For a given rise in blood pressure, a typical vein stretches about eight times as much as a corresponding artery. The *capacitance* of a blood vessel is the relationship between the volume of blood it contains and the blood pressure. If a vessel behaves like

Figure 21–7 The Distribution of Blood in the Cardiovascular System.



a child's balloon, expanding easily at low pressures, it has high capacitance. If it behaves more like a truck tire, expanding only at high pressures, it has low capacitance. Veins, which expand easily, are called **capacitance vessels.** Because veins have high capacitance, they can accommodate large changes in blood volume. If the blood volume rises or falls, the elastic walls stretch or recoil, changing the volume of blood in the venous system.

If serious hemorrhaging occurs, the *vasomotor center* of the medulla oblongata stimulates sympathetic nerves that innervate smooth muscle cells in the walls of medium-sized veins. This activity has two major effects:

1. *Systemic veins constrict.* This process, called **venoconstriction** (vē-nō-kon-STRIK-shun), reduces the amount of blood within the venous system, increasing the volume within the arterial system and capillaries. Venoconstriction can keep the blood volume within the arteries and capillaries at near-normal levels despite a significant blood loss.
2. *The constriction of veins in the liver, skin, and lungs redistributes a significant proportion of the total blood volume.* As a result, blood flow to delicate organs (such as the brain) and to active skeletal muscles can be increased or maintained after blood loss. The amount of blood that can be shifted from veins in the liver, skin, and lungs to the general circulation is called the **venous reserve.** It is normally about 20 percent of total blood volume.

Checkpoint

1. List the five general classes of blood vessels.
2. A cross section of tissue shows several small, thin-walled vessels with very little smooth muscle tissue in the tunica media. Which type of vessel are these?
3. Why are valves located in veins, but not in arteries?
4. Where in the body would you find fenestrated capillaries?

See the blue Answers tab at the back of the book.

21-2 Pressure and resistance determine blood flow and affect rates of capillary exchange

Figure 21–8 provides an overview of the discussion of cardiovascular physiology that follows. The purpose of cardiovascular regulation is the maintenance of adequate blood flow through the capillaries in peripheral tissues and organs. Under normal circumstances, blood flow is equal to cardiac output. When cardiac output goes up, so does the blood flow through capillary beds, and when cardiac output declines, capillary blood flow is reduced.

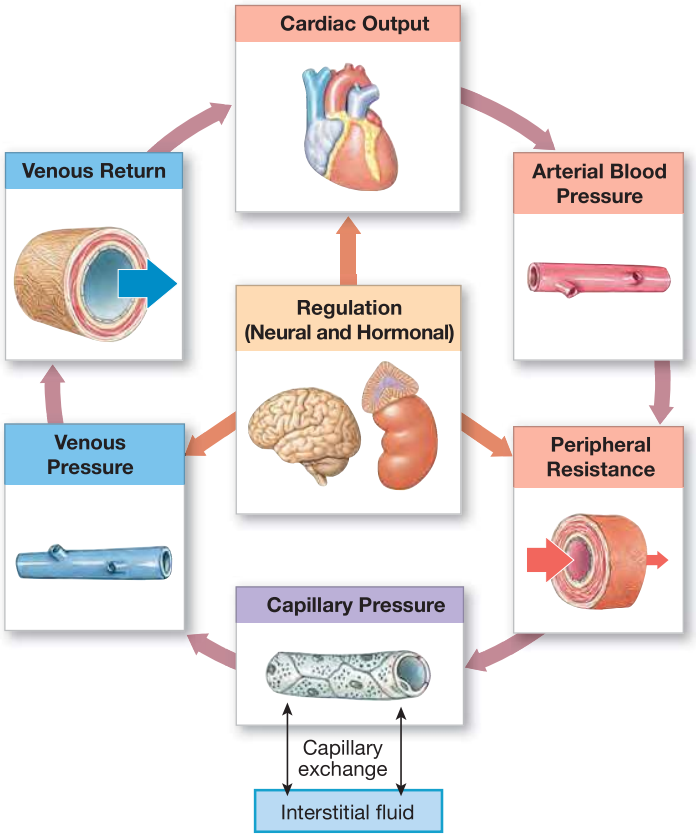
Capillary blood flow is determined by the interplay between *pressure (P)* and *resistance (R)* in the cardiovascular network. To keep blood moving, the heart must generate enough pressure to overcome the resistance to blood flow in the pulmonary and systemic circuits. In general terms, flow (*F*) is directly proportional to the pressure (increased pressure → increased flow), and inversely proportional to resistance (increased resistance → decreased flow). However, the absolute pressure is less important than the pressure *gradient*—the difference in pressure from one end of the vessel to the other. This relationship can be summarized as

$$F \propto \frac{\Delta P}{R}$$

where the symbol ∝ means "is proportional to" and Δ means "the difference in." The largest pressure gradient is found between the base of the aorta and the proximal ends of peripheral capillary beds. Cardiovascular control centers can alter this pressure gradient, and change the rate of capillary blood flow, by adjusting cardiac output and peripheral resistance.

Blood leaving the peripheral capillaries enters the venous system. The pressure gradient across the venous system is relatively small, but venous resistance is very low. The low venous blood pressure—aided by valves, skeletal muscle contraction, gravity, and other factors—is enough to return the blood to the heart. When necessary, cardiovascular control centers can raise venous pressure (through venoconstriction) to improve venous return and maintain adequate cardiac output.

Figure 21–8 An Overview of Cardiovascular Physiology. Neural and hormonal activities influence cardiac output, peripheral resistance, and venous pressure (through venoconstriction). Capillary pressure is the primary drive for exchange between blood and interstitial fluid.



We will begin this section by examining blood pressure and resistance more closely. We will then consider the mechanisms of *capillary exchange*, the transfer of liquid and solutes between the blood and interstitial fluid. Capillary exchange provides tissues with oxygen and nutrients and removes the carbon dioxide and waste products generated by active cells.

Active tissues need more blood flow than inactive ones. Even something as simple as a change in position—going from sitting to standing, for instance—triggers a number of cardiovascular changes. We will end this section with a discussion of what those changes are and how they are coordinated.

Pressure

When talking about cardiovascular pressures, three values are usually reported:

1. *Blood Pressure.* The term **blood pressure (BP)** refers to arterial pressure, usually reported in millimeters of mercury (mm Hg). Average systemic arterial pressures range from an average of 100 mm Hg at the entrance to the aorta to roughly 35 mm Hg at the start of a capillary network.
2. *Capillary Hydrostatic Pressure.* Hydrostatic pressure is the force exerted by a fluid pressing against a wall. **Capillary hydrostatic pressure (CHP),** or *capillary pressure*, is the pressure within capillary walls. Along the length of a typical capillary, pressures decline from roughly 35 mm Hg to about 18 mm Hg.
3. *Venous Pressure.* **Venous pressure** is the pressure within the venous system. Venous pressure is quite low: The pressure gradient from the venules to the right atrium is only about 18 mm Hg.

The difference in pressure (ΔP) across the entire systemic circuit, sometimes called the *circulatory pressure*, averages about 100 mm Hg. For circulation to occur, the circulatory pressure must overcome the **total peripheral resistance**—the resistance of the entire cardiovascular system. The arterial network has by far the largest pressure gradient (65 mm Hg), and this primarily reflects the relatively high resistance of the arterioles.

Total Peripheral Resistance

The total peripheral resistance of the cardiovascular system reflects a combination of factors: *vascular resistance*, *blood viscosity*, and *turbulence*.

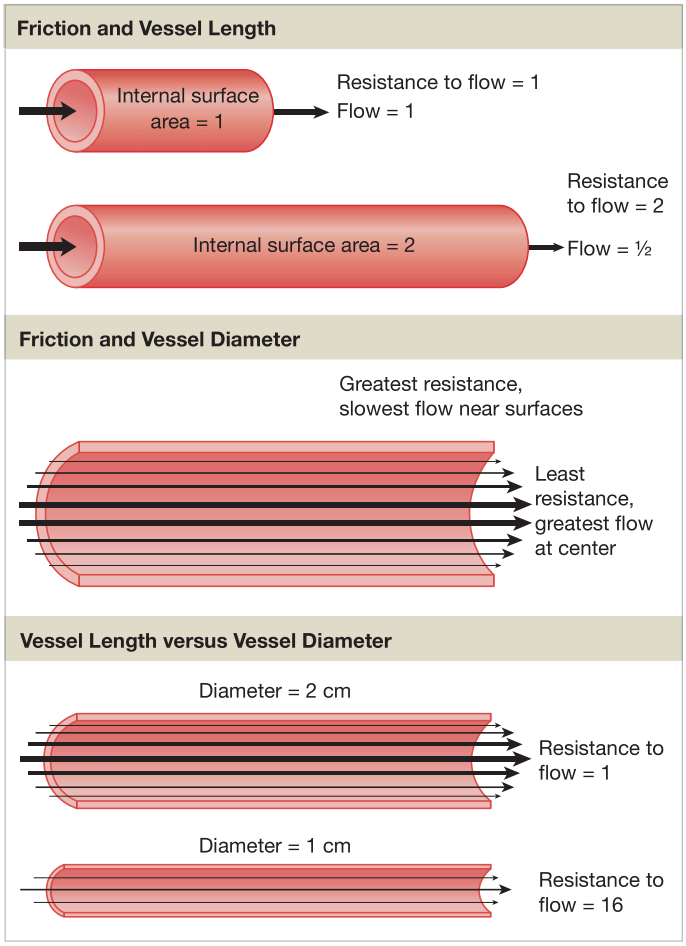
Vascular Resistance

Vascular resistance, the forces that oppose blood flow in the blood vessels, is the largest component. The most important factor in vascular resistance is friction between blood and the vessel walls. The amount of friction depends on two factors: vessel length and vessel diameter. **Figure 21–9** shows the factors affecting friction and vascular resistance.

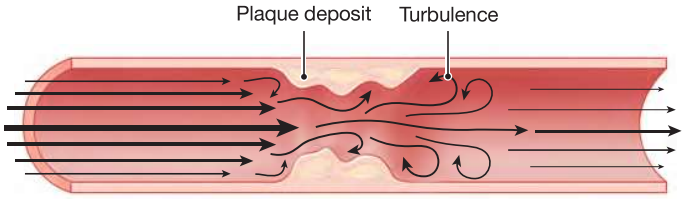
Vessel Length. Increasing the length of a blood vessel increases friction: The longer the vessel, the larger the surface area in contact with blood. You can easily blow the water out of a snorkel that is 2.5 cm (1 in.) in diameter and 25 cm (10 in.) long, but you cannot blow the water out of a 15 m-(16 yard) long garden hose, because the total friction is too great. The most dramatic changes in blood vessel length occur between birth and maturity, as individuals grow to adult size. In adults, vessel length can increase or decrease gradually when individuals gain or lose weight, but on a day-to-day basis this component of vascular resistance can be considered constant.

Vessel Diameter. The effects of friction on blood act in a narrow zone closest to the vessel wall. In a small-diameter vessel, friction with the walls slows nearly all the blood. Resistance is therefore relatively high. Blood near the center of a large-diameter vessel does not encounter friction with the walls, so the resistance in large vessels is fairly low.

Figure 21–9 Factors Affecting Friction and Vascular Resistance.



Factors Affecting Vascular Resistance



Turbulence

Differences in diameter have much more significant effects on resistance than do differences in length. If two vessels are equal in diameter but one is twice as long as the other, the longer vessel offers twice as much resistance to blood flow. But for two vessels of equal length, one twice the diameter of the other, the narrower one offers 16 times as much resistance to blood flow. This relationship, expressed in terms of the vessel radius r and resistance R, can be summarized as $R \propto 1/r^4$.

More significantly, there is no way to control vessel length, but vessel diameter can change quickly through vasoconstriction or vasodilation. Most of the peripheral resistance occurs in arterioles, the smallest vessels of the arterial system. As noted earlier in the chapter, arterioles are extremely muscular: The wall of an arteriole with an inner diameter of 30 μm can have a 20-µm-thick layer of smooth muscle. When these smooth muscles contract or relax, peripheral resistance increases or decreases. Because a small change in diameter produces a large change in resistance, mechanisms that alter the diameters of arterioles provide control over peripheral resistance and blood flow.

Blood Viscosity

Viscosity is the resistance to flow caused by interactions among molecules and suspended materials in a liquid. Liquids of low viscosity, such as water (viscosity 1.0), flow at low pressures. Thick, syrupy fluids, such as molasses (viscosity 300), flow only under higher pressures. Whole blood has a viscosity about five times that of water, due to its plasma proteins and blood cells. Under normal conditions, the viscosity of blood remains stable. Anemia, polycythemia, and other disorders that affect the hematocrit also change blood viscosity, and thus peripheral resistance.

Turbulence

High flow rates, irregular surfaces, and sudden changes in vessel diameter upset the smooth flow of blood, creating eddies and swirls. This phenomenon, called **turbulence,** increases resistance and slows blood flow.

Turbulence normally occurs when blood flows between the atria and the ventricles, and between the ventricles and the aortic and pulmonary trunks. It also develops in large arteries, such as the aorta, when cardiac output and arterial flow rates are very high. However, turbulence seldom occurs in smaller vessels unless their walls are damaged. For example, an atherosclerotic plaque creates abnormal turbulence and restricts blood flow. Because turbulence makes a distinctive sound, or *bruit* (broo-Ē), plaques in large blood vessels can often be detected with a stethoscope.

Table 21–1 provides a quick review of the terms and relationships discussed in this section.

An Overview of Cardiovascular Pressures

Look at the graphs in Figure 21–10 for an overview of the vessel diameters, cross-sectional areas, pressures, and velocity of blood flow in the systemic circuit.

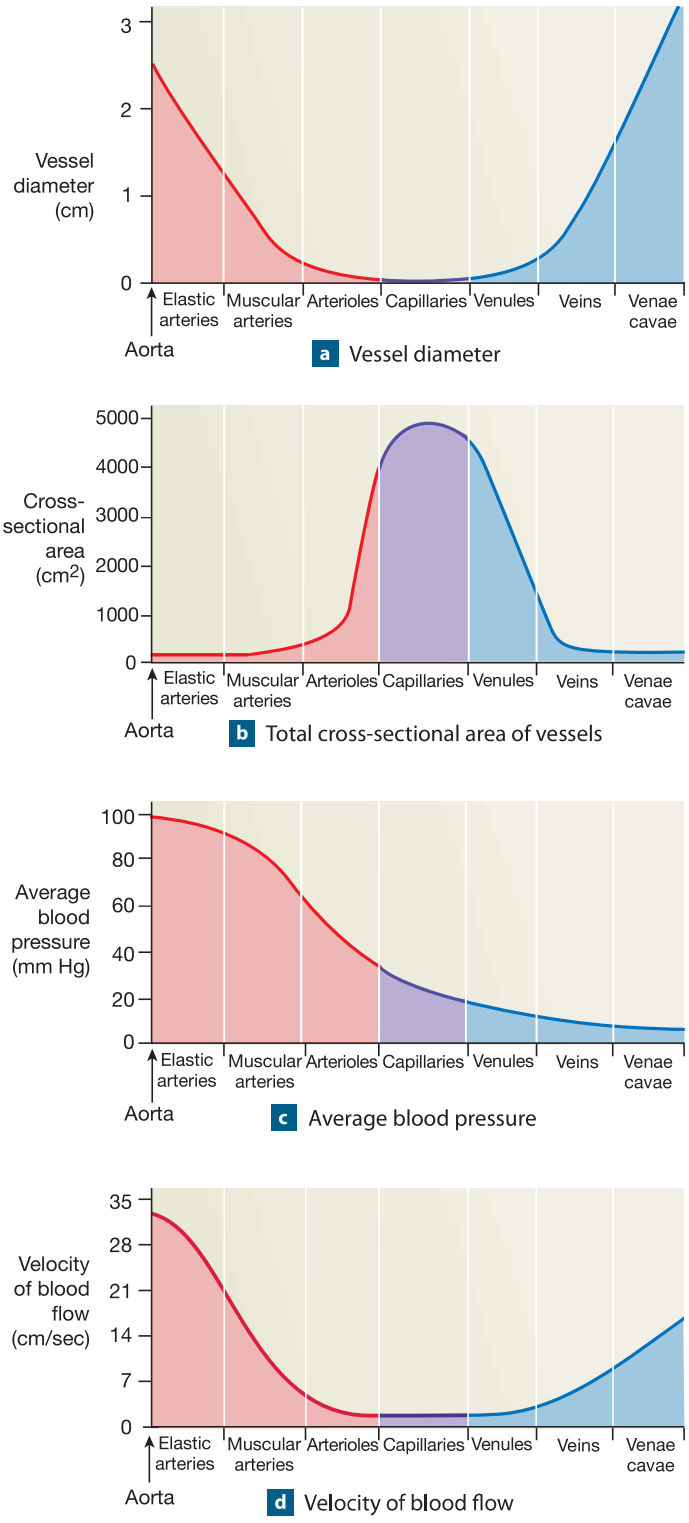
- *Vessel Diameters.* As blood proceeds from the aorta toward the capillaries, vessels diverge. The arteries branch repeatedly, and each branch is smaller in diameter than the preceding one (Figure 21–10a). As blood proceeds from the capillaries toward the venae cavae, vessels converge. Vessel diameters increase as venules combine to form small and medium-sized veins.

Table 21–1 Key Terms and Relationships Pertaining to Blood Circulation

Blood Flow *(F)*:	The volume of blood flowing per unit of time through a vessel or a group of vessels; may refer to circulation through a capillary, a tissue, an organ, or the entire vascular network. Total blood flow is equal to cardiac output.
Blood Pressure *(BP)*:	The hydrostatic pressure in the arterial system that pushes blood through capillary beds.
Circulatory Pressure:	The pressure difference between the base of the ascending aorta and the entrance to the right atrium.
Hydrostatic Pressure:	A pressure exerted by a liquid in response to an applied force.
Peripheral Resistance *(PR)*:	The resistance of the arterial system; affected by such factors as vascular resistance, viscosity, and turbulence.
Resistance *(R)*:	A force that opposes movement (in this case, blood flow).
Total Peripheral Resistance:	The resistance of the entire cardiovascular system.
Turbulence:	A resistance due to the irregular, swirling movement of blood at high flow rates or exposure to irregular surfaces.
Vascular Resistance:	A resistance due to friction within a blood vessel, primarily between the blood and the vessel walls. Increases with increasing length or decreasing diameter; vessel length is constant, but vessel diameter can change.
Venous Pressure:	The hydrostatic pressure in the venous system.
Viscosity:	A resistance to flow due to interactions among molecules within a liquid.
RELATIONSHIPS AMONG THE PRECEDING TERMS	
$F \propto P$	Flow is proportional to the pressure gradient.
$F \propto 1/R$	Flow is inversely proportional to resistance.
$F \propto P/R$	Flow is directly proportional to the pressure gradient, and inversely proportional to resistance.
$F \propto BP/PR$	Flow is directly proportional to blood pressure, and inversely proportional to peripheral resistance.
$R \propto 1/r^4$	Resistance is inversely proportional to the fourth power of the vessel radius.

- *Total Cross-Sectional Areas.* Although the arterioles, capillaries, and venules are small in diameter, the body has large numbers of them. All the blood flowing through the aorta also flows through peripheral capillaries. Blood pressure and the speed of blood flow are proportional to the cross-sectional area of the vessels involved. What is important is not the cross-sectional area of each individual vessel, but the *combined* cross-sectional area of *all* the vessels (**Figure 21–10b**). In effect, your blood moves from one big pipe (the aorta, with a cross-sectional area of 4.5 cm^2) into countless tiny ones (the peripheral capillaries, with a total cross-sectional area of 5000 cm^2), and then blood travels back to the heart through two large pipes (the venae cavae).

Figure 21–10 Relationships among Vessel Diameter, Cross-Sectional Area, Blood Pressure, and Blood Velocity within the Systemic Circuit.



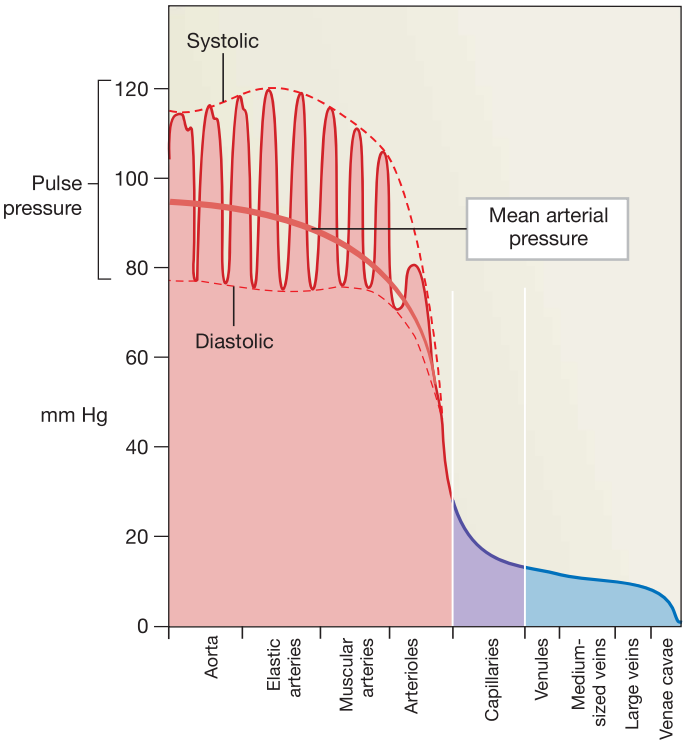
- *Pressures.* As arteries branch, their total cross-sectional area increases, and blood pressure falls rapidly (**Figure 21–10c**). Most of the decline takes place in the small arteries and arterioles. Venous pressures are relatively low.
- *Velocity of Blood Flow.* As the total cross-sectional area of the vessels increases from the aorta toward the capillaries, the velocity of blood flow decreases (**Figure 21–10d**). Blood flow velocity then increases as the total cross-sectional area drops from the capillaries toward the venae cavae.

Figure 21–11 graphs the blood pressure throughout the cardiovascular system. Systemic pressures are highest in the aorta, peaking at about 120 mm Hg. Pressures reach a minimum of 2 mm Hg at the entrance to the right atrium. Pressures in the pulmonary circuit are much lower than those in the systemic circuit. The right ventricle does not ordinarily develop high pressures because the pulmonary vessels are much shorter and more distensible than the systemic vessels, thus providing less resistance to blood flow.

Arterial Blood Pressure

Arterial pressure is important because it maintains blood flow through capillary beds. To do this, it must always be high enough to overcome the peripheral resistance. Arterial pressure is not constant. Rather, it rises during ventricular systole and falls during ventricular diastole. The peak blood pressure measured during ventricular systole is called **systolic pressure,** and the minimum blood pressure at the end of ventricular diastole is called **diastolic pressure.** In recording blood pressure, we separate systolic and diastolic pressures by a slash, as in "120/80" ("one-twenty over eighty") or "110/75."

Figure 21–11 **Pressures within the Systemic Circuit.** Notice the general reduction in circulatory pressure within the systemic circuit and the elimination of the pulse pressure within the arterioles.



A *pulse* is a rhythmic fluctuation in pressure that accompanies each heartbeat. The difference between the systolic and diastolic pressures is the **pulse pressure** (**Figure 21–11**). To report a single blood pressure value, we use the **mean arterial pressure (MAP).** It is calculated by adding one-third of the pulse pressure to the diastolic pressure:

$$\text{MAP} = \text{diastolic pressure} + \frac{\text{pulse pressure}}{3}$$

For a systolic pressure of 120 mm Hg and a diastolic pressure of 90 mm Hg, we calculate MAP as follows:

$$\text{MAP} = 90 + \frac{(120 - 90)}{3} = 90 + 10 = 100 \text{ mm Hg}$$

A normal range of systolic and diastolic pressures occurs in healthy individuals. When pressures shift outside of the normal range, clinical problems develop. Abnormally high blood pressure is termed **hypertension.** Abnormally low blood pressure is **hypotension.** Hypertension is much more common. In fact, many cases of hypotension result from overly aggressive drug treatment for hypertension.

The usual criterion established by the American Heart Association for hypertension in adults is a blood pressure greater than 140/90. Blood pressure at or below 120/80 is normal, and values between 121/81 and 139/89 indicate *pre-hypertension*. Cardiologists often recommend some combination of diet modification and drug therapy for people whose blood pressures are consistently pre-hypertensive.

Hypertension significantly increases the workload on the heart, and the left ventricle gradually enlarges. More muscle mass means a greater demand for oxygen. When the coronary circulation cannot keep pace, signs and symptoms of coronary ischemia appear. ↩ p. 682 Increased arterial pressures also place a physical stress on the walls of blood vessels throughout the body. This stress promotes or accelerates the development of arteriosclerosis. It also increases the risk of aneurysms, heart attacks, and strokes.

Elastic Rebound

As systolic pressure climbs, the arterial walls stretch, just as an extra puff of air expands a partially inflated balloon. This expansion allows the arterial system to accommodate some of the blood provided by ventricular systole. When diastole begins and blood pressures fall, the arteries recoil to their original dimensions. This phenomenon is called **elastic rebound.**

Some blood is forced back toward the left ventricle, closing the aortic valve and helping to drive additional blood into the coronary arteries. However, most of the push from elastic rebound forces blood toward the capillaries. This maintains blood flow along the arterial network while the left ventricle is in diastole.

Pressures in Small Arteries and Arterioles

The mean arterial pressure and the pulse pressure become smaller as the distance from the heart increases (**Figure 21–11**):

- The mean arterial pressure declines as the arterial branches become smaller and more numerous. In essence, blood pressure decreases as it overcomes friction and produces blood flow.
- The pulse pressure lessens due to the cumulative effects of elastic rebound along the arterial system. The effect can be likened to a series of ever-softer echoes following a loud shout. Each time an echo is produced, the reflecting surface absorbs some of the sound energy. Eventually, the echo disappears. The pressure surge accompanying ventricular ejection is like the shout, and it is reflected by the wall of the aorta, echoing down the arterial system until it finally disappears at the level of the small arterioles. By the time blood reaches a precapillary sphincter, no pressure fluctuations remain, and the blood pressure is steady at approximately 35 mm Hg.

Venous Pressure and Venous Return

Venous pressure, although low, determines venous return—the amount of blood arriving at the right atrium each minute. Venous return has a direct impact on cardiac output. ↩ p. 699 Blood pressure at the start of the venous system is only about one-tenth that at the start of the arterial system, but the blood must still travel through a vascular network as complex as the arterial system before returning to the heart.

Pressures at the entrance to the right atrium fluctuate, but they average about 2 mm Hg. Thus, the effective pressure in the venous system is roughly 16 mm Hg (from 18 mm Hg in the venules to 2 mm Hg in the venae cavae). This pressure compares with 65 mm Hg in the arterial system (from 100 mm Hg at the aorta to 35 mm Hg at the capillaries). Yet, although venous pressures are low, veins offer comparatively little resistance, so pressure declines very slowly as blood moves through the venous system. As blood moves toward the heart, the veins become larger, resistance drops, and the velocity of blood flow increases (**Figure 21–10**).

When you stand, the venous blood returning from your body inferior to the heart must overcome gravity as it travels up the inferior vena cava. Two factors assist the low venous pressures in propelling blood toward your heart: *muscular compression* of peripheral veins and the *respiratory pump*.

Muscular Compression. The contractions of skeletal muscles near a vein compress it, helping to push blood toward the heart. The valves in small and medium-sized veins ensure that blood flows in one direction only (**Figure 21–6**). When you are standing and walking, the cycles of contraction and relaxation that accompany your normal movements assist venous return. If you stand at attention, with knees locked and leg muscles immobilized, that assistance is lost. The reduction in venous return then leads to a fall in cardiac output, which reduces the blood supply to the brain. This decline is sometimes enough to cause **fainting,** a temporary loss of consciousness. You would then collapse, but while you were in the horizontal position, both venous return and cardiac output would return to normal.

The Respiratory Pump. As you inhale, your thoracic cavity expands, reducing the pressure within the pleural cavities. This drop in pressure pulls air into your lungs. At the same time, it also pulls blood into the inferior vena cava and right atrium from the smaller veins of your abdominal cavity and lower body. The effect on venous return through the superior vena cava is less pronounced, because blood in that vessel is normally assisted by gravity. As you exhale, your thoracic cavity decreases in size. Internal pressure then rises, forcing air out of your lungs and pushing venous blood into the right atrium. This mechanism is called the **respiratory pump.** Such pumping action becomes more important during heavy exercise, when respirations are deep and frequent.

Capillary Pressures and Capillary Exchange

Capillary exchange plays a key role in homeostasis. The most important processes that move materials across typical capillary walls are *diffusion*, *filtration*, and *reabsorption*.

Diffusion

As we saw in Chapter 3, *diffusion* is the net movement of ions or molecules from an area where their concentration is higher to an area where their concentration is lower. ↩ p. 86 The difference between the high and low concentrations represents a *concentration gradient*. Diffusion tends to eliminate that gradient. Diffusion occurs most rapidly when (1) the distances involved are short, (2) the concentration gradient is large, and (3) the ions or molecules involved are small.

Different substances diffuse across capillary walls by different routes:

1. *Water, ions, and small organic molecules, such as glucose, amino acids, and urea*, can usually enter or leave the bloodstream by diffusion between adjacent endothelial cells or through the pores of fenestrated capillaries.
2. *Many ions, including sodium, potassium, calcium, and chloride,* can diffuse across endothelial cells by passing through channels in plasma membranes.

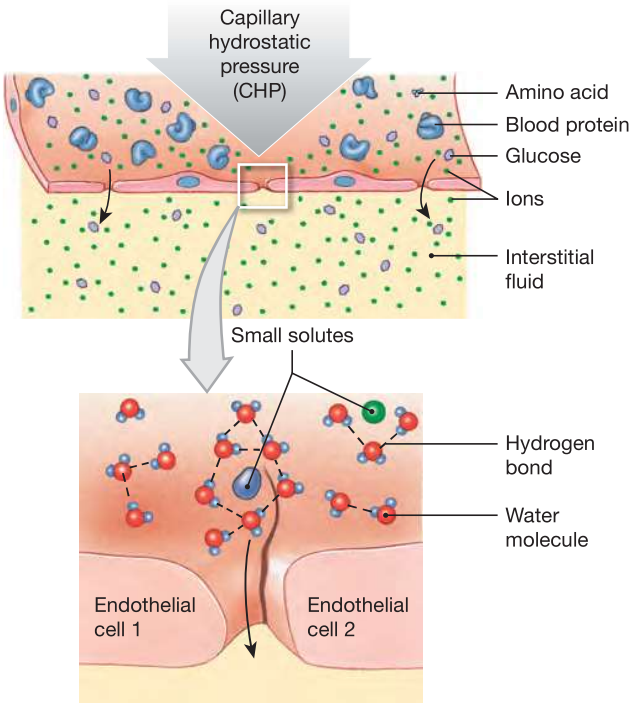
3. *Large water-soluble compounds* are unable to enter or leave the bloodstream except at fenestrated capillaries, such as those of the hypothalamus, the kidneys, many endocrine organs, and the intestinal tract.
4. *Lipids, such as fatty acids and steroids, and lipid-soluble materials, including soluble gases such as oxygen and carbon dioxide,* can cross capillary walls by diffusion through the endothelial plasma membranes.
5. *Plasma proteins* are normally unable to cross the endothelial lining anywhere except in sinusoids, such as those of the liver, where plasma proteins enter the bloodstream.

Filtration

Filtration is the removal of solutes as a solution flows across a porous membrane. Solutes too large to pass through the pores are filtered out of the solution. The driving force for filtration is hydrostatic pressure. As we saw earlier, it pushes water from an area of higher pressure to an area of lower pressure.

In *capillary filtration*, water and small solutes are forced across a capillary wall, leaving larger solutes and suspended proteins in the bloodstream (**Figure 21–12**). The solute molecules that leave the bloodstream are small enough to pass between adjacent endothelial cells or through the pores in a fenestrated capillary. Filtration takes place primarily at the arterial end of a capillary, where capillary hydrostatic pressure (CHP) is highest.

Figure 21–12 Capillary Filtration. Capillary hydrostatic pressure (CHP) forces water and solutes through the gaps between adjacent endothelial cells in continuous capillaries. The sizes of solutes that move across the capillary wall are determined primarily by the dimensions of the gaps.



Reabsorption

Reabsorption occurs as the result of osmosis. *Osmosis* is a special term for the diffusion of water across a selectively permeable membrane that separates two solutions of differing solute concentrations. Water molecules tend to diffuse across a membrane *toward* the solution containing the higher solute concentration (**Figure 3–16**, p. 86).

The **osmotic pressure (OP)** of a solution is an indication of the force of osmotic water movement. In other words, it represents the pressure that must be applied to prevent osmotic movement across a membrane. The higher the solute concentration of a solution, the greater is the solution's osmotic pressure. The presence of suspended proteins that cannot cross capillary walls creates an osmotic pressure called *blood colloid osmotic pressure (BCOP)*. Clinicians often use the term *oncotic pressure* (*onkos*, a swelling) when referring to the colloid osmotic pressure of body fluids. The two terms are equivalent. Osmotic water movement continues until either the solute concentrations are equalized or an opposing hydrostatic pressure prevents the movement.

Now let's look at the interplay between filtration and reabsorption along the length of a typical capillary. In this discussion, remember that hydrostatic pressure forces water *out of* a solution, and osmotic pressure draws water *into* a solution.

The Interplay between Filtration and Reabsorption

The continuous movement of water out of the capillaries, through peripheral tissues, and then back to the bloodstream by way of the lymphatic system has four important functions:

1. It ensures that plasma and interstitial fluid, two major components of extracellular fluid, are in constant communication and mutual exchange.
2. It accelerates the distribution of nutrients, hormones, and dissolved gases throughout tissues.
3. It assists in the transport of insoluble lipids and tissue proteins that cannot enter the bloodstream by crossing the capillary walls.
4. It has a flushing action that carries bacterial toxins and other chemical stimuli to lymphatic tissues and organs responsible for providing immunity to disease.

Capillary blood pressure declines as blood flows from the arterial end to the venous end of a capillary. As a result, the rates of filtration and reabsorption gradually change as blood passes along the length of a capillary. The factors involved are diagrammed in **Figure 21–13**.

Net hydrostatic pressure is the difference between the pressure inside the capillary wall and the hydrostatic pressure outside

the capillary. The *net capillary hydrostatic pressure* tends to push water and solutes out of capillaries and into the interstitial fluid. Factors that contribute to the net hydrostatic pressure include:

1. the *capillary hydrostatic pressure (CHP)*, which ranges from 35 mm Hg at the arterial end of a capillary to 18 mm Hg at the venous end, and
2. the *interstitial fluid hydrostatic pressure (IHP)*. Measurements of IHP have yielded very small values that differ from tissue to tissue—from +6 mm Hg in the brain to −6 mm Hg in subcutaneous tissues. A positive IHP opposes CHP, and the tissue hydrostatic pressure must be overcome before fluid can move out of a capillary. A negative IHP assists CHP, and additional fluid will be pulled out of the capillary. However, under normal circumstances the average IHP is 0 mm Hg, and we can assume that the net hydrostatic pressure is equal to CHP. (For this reason, IHP is not included in **Figure 21–13**.)

Plasma proteins in capillary blood create capillary colloid osmotic pressure. The *net capillary colloid osmotic pressure* tends to pull water and solutes into a capillary from the interstitial fluid. The net colloid osmotic pressure is the difference between

1. the *blood colloid osmotic pressure (BCOP)*, which is roughly 25 mm Hg, and
2. the *interstitial fluid colloid osmotic pressure (ICOP)*. The ICOP is as variable and low as the IHP, because the interstitial fluid in most tissues contains negligible quantities of suspended proteins. Reported values of ICOP are from 0 to 5 mm Hg, within the range of pressures recorded for the IHP. It is thus safe to assume that under normal circumstances the net colloid osmotic pressure is equal to the BCOP. (For this reason, ICOP is not included in **Figure 21–13**.)

The **net filtration pressure (NFP)** is the difference between the net hydrostatic pressure and the net osmotic pressure. In terms of the factors just listed, this means that

$$\begin{array}{ccccc} \text{net filtration} & = & \text{net hydrostatic} & - & \text{net colloid} \\ \text{pressure} & & \text{pressure} & & \text{osmotic pressure} \\ \text{NFP} & = & (\text{CHP} - \text{IHP}) & - & (\text{BCOP} - \text{ICOP}) \end{array}$$

At the arterial end of a capillary, the net filtration pressure can be calculated as follows:

$$\text{NFP} = (35 - 0) - (25 - 0) = 35 - 25 = 10 \text{ mm Hg}$$

Because this value is positive, it indicates that fluid will tend to move *out of* the capillary and into the interstitial fluid. At the venous end of the capillary, the net filtration pressure will be

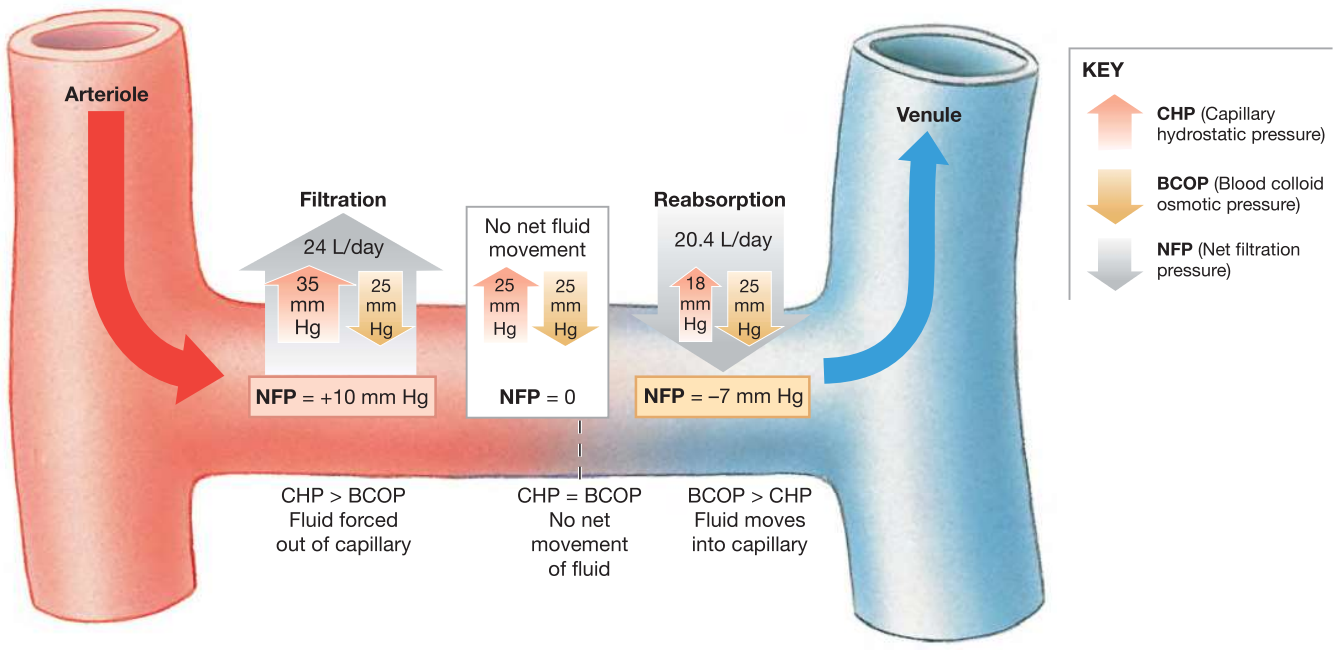
$$\text{NFP} = (18 - 0) - (25 - 0) = 18 - 25 = -7 \text{ mm Hg}$$

The minus sign indicates that fluid tends to move *into* the capillary; that is, reabsorption is occurring.

The transition between filtration and reabsorption occurs where the CHP is 25 mm Hg, because at that point the hydro-

21

Figure 21–13 Forces Acting across Capillary Walls. At the arterial end of the capillary, capillary hydrostatic pressure (CHP) is greater than blood colloid osmotic pressure (BCOP), so fluid moves out of the capillary (filtration). Near the venule, CHP is lower than BCOP, so fluid moves into the capillary (reabsorption). In this model, interstitial fluid colloid osmotic pressure (ICOP) and interstitial fluid hydrostatic pressure (IHP) are assumed to be 0 mm Hg and so are not shown.



Clinical Note Edema

It's not a good time but a swell time

Edema (e-DĒ-muh) is an abnormal accumulation of interstitial fluid. Edema has many causes, and we will encounter specific examples in later chapters. The underlying problem in all types of edema is a disturbance in the normal balance between hydrostatic and osmotic forces at the capillary level. For instance:

- When a capillary is damaged, plasma proteins can cross the capillary wall and enter the interstitial fluid. The resulting rise in the interstitial fluid colloid osmotic pressure (ICOP) reduces the rate of capillary reabsorption and produces a localized edema. This is why you usually have swelling at a bruise.
- In starvation, the liver cannot synthesize enough plasma proteins to maintain normal concentrations in the blood. Blood colloid osmotic pressure (BCOP) declines, and fluids begin moving from the blood into peripheral tissues. In children, fluid builds up in the abdominopelvic cavity, producing the swollen bellies typical of starvation victims. A reduction in BCOP also takes place after severe burns and in several types of liver and kidney diseases.
- In the U.S. population, most serious cases of edema result from increases in arterial blood pressure, venous pressure, or total circulatory pressure. The increase may result from heart problems such as heart failure, venous blood clots that elevate venous pressures, or other cardiovascular abnormalities. The net result is an increase in capillary hydrostatic pressure (CHP) that accelerates fluid movement into the tissues.

static and osmotic forces are equal—that is, the NFP is 0 mm Hg. If the maximum filtration pressure at the arterial end of the capillary were equal to the maximum reabsorption pressure at the venous end, this transition point would lie midway along the length of the capillary. Under these circumstances, filtration would occur along the first half of the capillary, and an identical amount of reabsorption would occur along the second half. However, the maximum filtration pressure is higher than the maximum reabsorption pressure, so the transition point between filtration and reabsorption normally lies closer to the venous end of the capillary than to the arterial end. As a result, more filtration than reabsorption occurs along the capillary. Of the roughly 24 liters of fluid that move out of the plasma and into the interstitial fluid each day, 20.4 liters (85 percent) are reabsorbed. The remainder (3.6 liters) flows through the tissues and into lymphatic vessels, for eventual return to the venous system.

Any condition that affects hydrostatic or osmotic pressures in the blood or tissues will shift the balance between hydrostatic and osmotic forces. We can then predict the effects on the basis of an understanding of capillary dynamics. For example,

- If hemorrhaging occurs, both blood volume and blood pressure decline. This reduction in CHP lowers the NFP and increases the amount of reabsorption. The result is a reduction in the volume of interstitial fluid and an increase in the circulating plasma volume. This process is known as a *recall of fluids*.
- If dehydration occurs, the plasma volume decreases due to water loss, and the concentration of plasma proteins increases. The increase in BCOP accelerates reabsorption and a recall of fluids that delays the onset and severity of clinical signs and symptoms.
- If the CHP rises or the BCOP declines, fluid moves out of the blood and builds up in peripheral tissues, a condition called *edema*.

Checkpoint

5. **Identify the factors that contribute to total peripheral resistance.**
6. **In a healthy individual, where is blood pressure greater: at the aorta or at the inferior vena cava? Explain.**
7. **While standing in the hot sun, Sally begins to feel light-headed and faints. Explain what happened.**
8. **Mike's blood pressure is 125/70. What is his mean arterial pressure?**

See the blue Answers tab at the back of the book.

21-3 Cardiovascular regulatory mechanisms involve autoregulation, neural mechanisms, and endocrine responses

Homeostatic mechanisms regulate cardiovascular activity to ensure that blood flow through tissues, called **tissue perfusion,** meets the demand for oxygen and nutrients. The factors

that affect tissue perfusion are (1) cardiac output, (2) peripheral resistance, and (3) blood pressure. We discussed cardiac output in Chapter 20 (p. 697). We considered peripheral resistance and blood pressure earlier in this chapter.

Most cells are relatively close to capillaries. When a group of cells becomes active, the circulation to that region must increase to bring the necessary oxygen and nutrients, and to carry away the waste products and carbon dioxide they generate. The purpose of cardiovascular regulation is to ensure that these blood flow changes occur (1) at an appropriate time, (2) in the right area, and (3) without drastically changing blood pressure and blood flow to vital organs.

The regulatory mechanisms focus on controlling cardiac output and blood pressure to restore adequate blood flow after blood pressure drops. We can group these mechanisms as follows:

- *Autoregulation.* Local factors change the pattern of blood flow within capillary beds as precapillary sphincters open and close in response to chemical changes in interstitial fluids. This is an example of autoregulation at the tissue level. Autoregulation causes immediate, localized homeostatic adjustments. If autoregulation fails to normalize conditions at the tissue level, neural mechanisms and endocrine factors are activated.
- *Neural Mechanisms.* Neural mechanisms respond to changes in arterial pressure or blood gas levels sensed at specific sites. When those changes occur, the cardiovascular centers of the autonomic nervous system adjust cardiac output and peripheral resistance to maintain blood pressure and ensure adequate blood flow.
- *Endocrine Mechanisms.* The endocrine system releases hormones that enhance short-term adjustments and that direct long-term changes in cardiovascular performance.

Now let's see how each of these regulatory mechanisms responds to inadequate perfusion of skeletal muscles. The regulatory relationships are diagrammed in **Figure 21–14**.

Autoregulation of Blood Flow within Tissues

Under normal resting conditions, cardiac output remains stable, and peripheral resistance within individual tissues is adjusted to control local blood flow.

Factors that promote the dilation of precapillary sphincters are called **vasodilators. Local vasodilators** act at the tissue level to accelerate blood flow through their tissue of origin. Examples of local vasodilators include the following:

- Decreased tissue oxygen levels or increased CO_2 levels.
- Lactic acid or other acids generated by tissue cells.
- Nitric oxide (NO) released from endothelial cells.
- Rising concentrations of potassium ions or hydrogen ions in the interstitial fluid.
- Chemicals released during local inflammation, including histamine and NO. ↩ p. 138
- Elevated local temperature.

These factors work by relaxing the smooth muscle cells of the precapillary sphincters. All of them indicate that tissue conditions are in some way abnormal. An increase in blood flow, which brings oxygen, nutrients, and buffers, may be sufficient to restore homeostasis.

As noted in Chapter 19, aggregating platelets and damaged tissues produce compounds that stimulate precapillary sphincters to constrict. These compounds are **local vasoconstrictors.** Examples include prostaglandins and thromboxanes released by activated platelets and white blood cells, and the endothelins released by damaged endothelial cells.

Local vasodilators and vasoconstrictors control blood flow within a single capillary bed (**Figure 21–5**). In high concentrations, these factors also affect arterioles, increasing or decreasing blood flow to all the capillary beds in a given area.

Neural Mechanisms

The nervous system adjusts cardiac output and peripheral resistance in order to maintain adequate blood flow to vital tissues and organs. Centers responsible for these regulatory activities include the *cardiac centers* and the *vasomotor center* of the medulla oblongata. ↩ p. 458

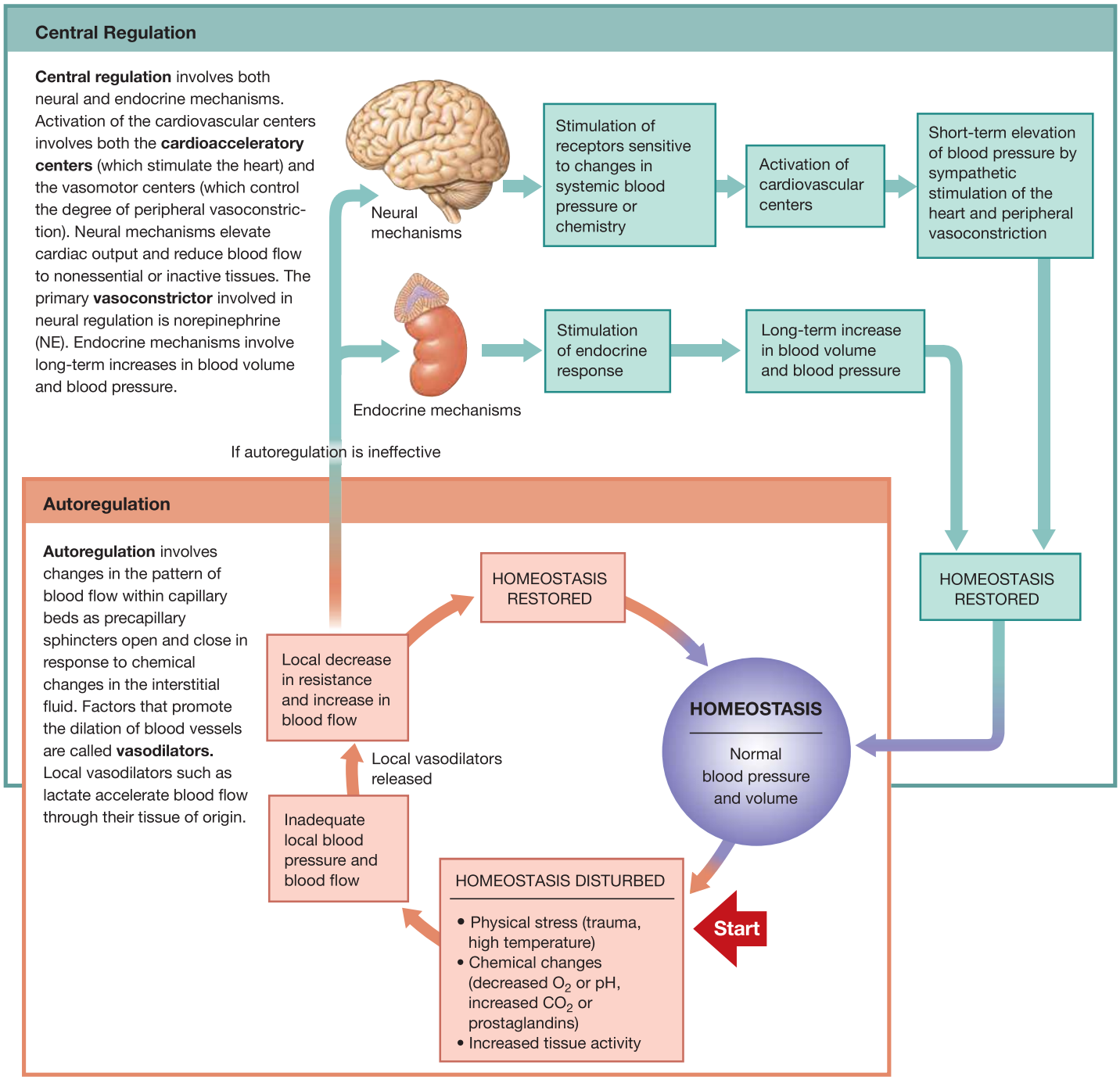
It is difficult to distinguish the cardiac and vasomotor centers anatomically. They are often considered to form complex **cardiovascular (CV) centers.** In functional terms, however, the cardiac and vasomotor centers often act independently.

As noted in Chapter 20, each cardiac center has a *cardioacceleratory center*, which increases cardiac output through sympathetic innervation. Each cardiac center also has a *cardioinhibitory center*, which reduces cardiac output through parasympathetic innervation. ↩ p. 698

The vasomotor center contains two populations of neurons: (1) a very large group responsible for widespread vasoconstriction and (2) a smaller group responsible for the vasodilation of arterioles in skeletal muscles and the brain. The vasomotor center controls the activity of sympathetic motor neurons:

1. *Control of Vasoconstriction.* The neurons innervating peripheral blood vessels in most tissues are *adrenergic*; that is, they release the neurotransmitter norepinephrine (NE). NE stimulates smooth muscles in the walls of arterioles, producing vasoconstriction.
2. *Control of Vasodilation.* Vasodilator neurons innervate blood vessels in skeletal muscles and in the brain. The stimulation of these neurons relaxes smooth muscle cells in the walls of arterioles, producing vasodilation. This relaxation is triggered by the appearance of NO in the surroundings. The vasomotor center may control NO release indirectly or

Figure 21–14 Short-Term and Long-Term Cardiovascular Responses. This diagram indicates general mechanisms that compensate for a reduction in blood pressure and blood flow.



directly. The most common vasodilator synapses are *cholinergic*—their synaptic terminals release ACh. In turn, ACh stimulates endothelial cells in the area to release NO, causing local vasodilation. Other vasodilator synapses are *nitroxidergic*—their synaptic terminals release NO as a neurotransmitter. Nitric oxide has an immediate and direct relaxing effect on the vascular smooth muscle cells in the area.

Vasomotor Tone

In Chapter 16, we saw how autonomic tone sets a background level of neural activity that can increase or decrease on demand. ↩ p. 531 The sympathetic vasoconstrictor nerves are always active, producing a significant **vasomotor tone.** This vasoconstrictor activity normally keeps the arterioles partially

constricted. Under maximal stimulation, arterioles constrict to about half their resting diameter. To dilate fully, an arteriole increases its resting diameter by about 1.5 times.

Constriction has a large effect on resistance, because, as we saw earlier, resistance increases sharply as luminal diameter decreases. The resistance of a maximally constricted arteriole is roughly 80 *times* that of a fully dilated arteriole. Because blood pressure varies directly with peripheral resistance, the vasomotor center can control arterial blood pressure very effectively by making modest adjustments in vessel diameters. Extreme stimulation of the vasomotor centers also produces venoconstriction and mobilizes the venous reserve.

Reflex Control of Cardiovascular Function

The cardiovascular centers detect changes in tissue demand by monitoring arterial blood, especially its blood pressure, pH, and concentrations of dissolved gases. The *baroreceptor reflexes* (*baro-*, pressure) respond to changes in blood pressure, and the *chemoreceptor reflexes* monitor changes in the chemical composition of arterial blood. These reflexes are regulated through a negative feedback loop: The stimulation of a receptor by an abnormal condition leads to a response that counteracts the stimulus and restores normal conditions.

Baroreceptor Reflexes. Baroreceptors are specialized receptors that monitor the degree of stretch in the walls of expandable organs. ↩ p. 501 The baroreceptors involved in cardiovascular regulation are found in the walls of (1) the **carotid sinuses,** expanded chambers near the bases of the *internal carotid arteries* of the neck (**Figure 21–23**); (2) the **aortic sinuses,** pockets in the walls of the ascending aorta adjacent to the heart (**Figure 20–8b**, p. 679); and (3) the wall of the right atrium. These receptors are part of the **baroreceptor reflexes,** which adjust cardiac output and peripheral resistance to maintain normal arterial pressures.

Aortic baroreceptors monitor blood pressure within the ascending aorta. Any changes trigger the **aortic reflex,** which adjusts blood pressure to maintain adequate blood pressure and blood flow through the systemic circuit. Carotid sinus baroreceptors trigger reflexes that maintain adequate blood flow to the brain. The carotid sinus receptors are extremely sensitive because blood flow to the brain must remain constant. **Figure 21–15** presents the basic organization of the baroreceptor reflexes triggered by changes in blood pressure at the carotid and aortic sinuses.

When blood pressure climbs, the increased output from the baroreceptors alters activity in the CV centers and produces two major effects (**Figure 21–15**):

1. *A decrease in cardiac output*, due to parasympathetic stimulation and the inhibition of sympathetic activity.
2. *Widespread peripheral vasodilation*, due to the inhibition of excitatory neurons in the vasomotor center.

The decrease in cardiac output reflects primarily a reduction in heart rate due to the release of acetylcholine at the sinoatrial (SA) node. ↩ p. 698 The widespread vasodilation lowers peripheral resistance, and this effect, combined with a reduction in cardiac output, leads to a decline in blood pressure to normal levels.

When blood pressure falls below normal, baroreceptor output is reduced accordingly (**Figure 21–15**). This change has two major effects working together to raise blood pressure:

1. *An increase in cardiac output*, through the stimulation of sympathetic innervation to the heart. This results from the stimulation of the cardioacceleratory center and is accompanied by an inhibition of the cardioinhibitory center.
2. *Widespread peripheral vasoconstriction*, caused by the stimulation of sympathetic vasoconstrictor neurons by the vasomotor center.

The effects on the heart result from the release of NE by sympathetic neurons innervating the SA node, the atrioventricular (AV) node, and the general myocardium. In a crisis, sympathetic activation occurs, and its effects are enhanced by the release of both NE and epinephrine (E) from the adrenal medullae. The net effect is an immediate increase in heart rate and stroke volume, and a corresponding rise in cardiac output.

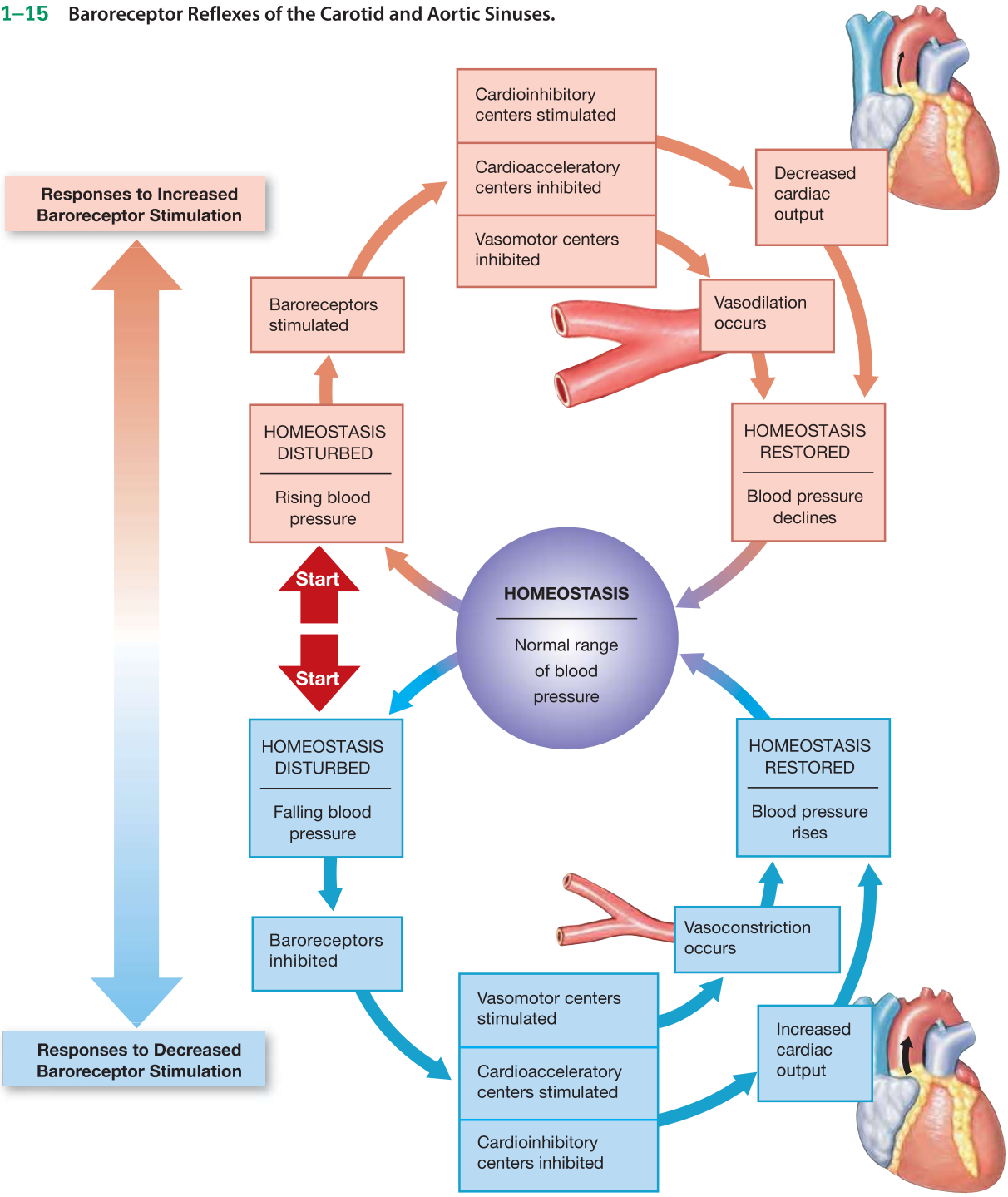
The vasoconstriction, which also results from the release of NE by sympathetic neurons, increases peripheral resistance. These adjustments—increased cardiac output and increased peripheral resistance—work together to elevate blood pressure.

Atrial baroreceptors monitor blood pressure at the end of the systemic circuit—at the venae cavae and the right atrium. Recall from Chapter 20 that the **atrial reflex** responds to a stretching of the wall of the right atrium. ↩ p. 699

Under normal circumstances, the heart pumps blood into the aorta at the same rate at which blood arrives at the right atrium. When blood pressure rises at the right atrium, blood is arriving at the heart faster than it is being pumped out. The atrial baroreceptors correct the situation by stimulating the CV centers to increase cardiac output until the backlog of venous blood is removed. Atrial pressure then returns to normal.

A procedure known as the Valsalva maneuver is a simple way to check for normal cardiovascular responses to changes in arterial pressure and venous return. The *Valsalva maneuver* involves trying to exhale forcefully with closed lips and nostrils so that no air can leave the lungs and pressure in the thoracic cavity rises sharply. This action causes reflexive changes in blood pressure and cardiac output due to increased intrathoracic pressure, which impedes venous return to the right atrium. When internal pressures rise, the venae cavae collapse, and the venous return decreases. The resulting drop in cardiac output and blood pressure stimulates the aortic and carotid baroreceptors, causing a reflexive increase in heart rate and peripheral vasoconstriction. When the glottis opens and pressures return to normal, venous

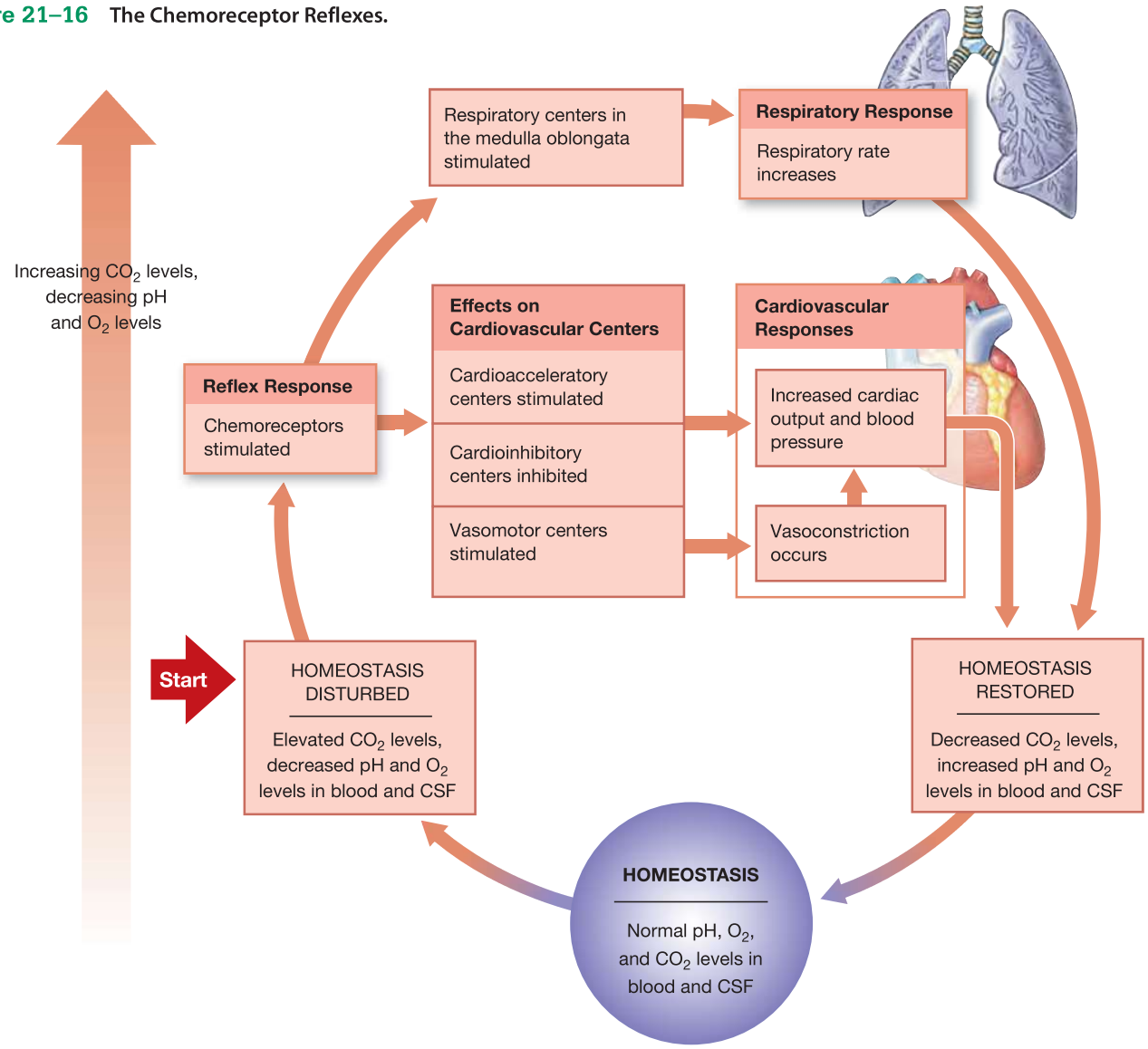
Figure 21–15 Baroreceptor Reflexes of the Carotid and Aortic Sinuses.



return increases suddenly and so does cardiac output. Because vasoconstriction has occurred, blood pressure rises sharply, and this inhibits the baroreceptors. As a result, cardiac output, heart rate, and blood pressure quickly return to normal levels.

Chemoreceptor Reflexes. The **chemoreceptor reflexes** respond to changes in carbon dioxide, oxygen, or pH levels in blood and cerebrospinal fluid (CSF) (**Figure 21–16**). The chemoreceptors involved are sensory neurons. They are located in the **carotid bodies,** situated in the neck near the carotid sinus, and the **aortic bodies,** near the arch of the aorta. ↩ p. 502 These receptors monitor the composition of arterial blood. Additional chemoreceptors located on the ventrolateral surfaces of the medulla oblongata monitor the composition of CSF.

Figure 21–16 The Chemoreceptor Reflexes.



When chemoreceptors in the carotid bodies or aortic bodies detect either a rise in the carbon dioxide content or a fall in the pH of the arterial blood, the cardioacceleratory and vasomotor centers are stimulated. At the same time, the cardioinhibitory center is inhibited. This dual effect causes an increase in cardiac output, peripheral vasoconstriction, and a rise in blood pressure. A drop in the oxygen level at the aortic bodies has the same effects. Strong stimulation of the carotid or aortic chemoreceptors causes widespread sympathetic activation, with more dramatic increases in heart rate and cardiac output.

The chemoreceptors of the medulla oblongata are involved primarily with the control of respiratory function, and secondarily with regulating blood flow to the brain. For example, a steep rise in CSF carbon dioxide levels triggers the vasodilation of cerebral vessels, but produces vasoconstriction in most other organs. The result is increased blood flow—and increased oxygen delivery—to the brain.

Coordination of cardiovascular and respiratory activities is vital, because accelerating blood flow in the tissues is useful only if the circulating blood contains an adequate amount of oxygen. Arterial CO_2 levels can be reduced and O_2 levels increased most effectively by coordinating cardiovascular and respiratory activities. Chemoreceptor stimulation also stimulates the respiratory centers, and the rise in cardiac output and blood pressure is associated with an increased respiratory rate. In addition, a rise in the respiratory rate accelerates venous return through the action of the respiratory pump. (We consider other aspects of chemoreceptor activity and respiratory control in Chapter 23.)

CNS Activities and the Cardiovascular Centers

The output of the cardiovascular centers can also be influenced by activities in other areas of the brain. For example, the activation of either division of the autonomic nervous system affects output from the cardiovascular centers. A general sympathetic activation stimulates the cardioacceleratory and vasomotor centers. As a result, cardiac output and blood pressure increase. In contrast, when the parasympathetic division is activated, the cardioinhibitory center is stimulated. The result is a reduction in cardiac output. Parasympathetic activity does not directly affect the vasomotor center, but vasodilation takes place as sympathetic activity declines.

The higher brain centers can also affect blood pressure. Our thought processes and emotional states can produce significant changes in blood pressure by influencing cardiac output and vasomotor tone. For example, strong emotions of anxiety, fear, and rage are accompanied by a rise in blood pressure, due to cardiac stimulation and vasoconstriction.

Hormones and Cardiovascular Regulation

The endocrine system regulates cardiovascular performance in both the short term and the long term. As we have seen, E and NE from the adrenal medullae stimulate cardiac output and peripheral vasoconstriction. Other hormones important in regulating cardiovascular function include (1) antidiuretic hormone (ADH), (2) angiotensin II, (3) erythropoietin (EPO), and (4) the natriuretic peptides (ANP and BNP). ↺ p. 626 All four are concerned primarily with the long-term regulation of blood volume (**Figure 21–17**). ADH and angiotensin II also affect blood pressure.

Antidiuretic Hormone

Antidiuretic hormone (ADH) is released at the posterior lobe of the pituitary gland in response to a decrease in blood volume, to an increase in the osmotic concentration of the plasma, or (secondarily) to circulating angiotensin II. It brings about a peripheral vasoconstriction that elevates blood pressure. This hormone also stimulates the conservation of water at the kidneys, thus preventing a reduction in blood volume that would further reduce blood pressure (**Figure 21–17a**).

Angiotensin II

Angiotensin II appears in the blood when specialized kidney cells, called *juxtaglomerular cells*, release the enzyme *renin* in response to a fall in renal blood pressure (**Figure 21–17a**). Once in the bloodstream, renin starts an enzymatic chain reaction. In the first step, renin converts *angiotensinogen*, a plasma protein produced by the liver, to *angiotensin I*. In the capillaries of the lungs, *angiotensin-converting enzyme (ACE)* then modifies angiotensin I to angiotensin II, an active hormone with diverse effects.

Angiotensin II has four important functions: (1) It stimulates the adrenal production of aldosterone, causing Na^+ retention and K^+ loss by the kidneys; (2) it stimulates the secretion of ADH, in turn stimulating water reabsorption by the kidneys and complementing the effects of aldosterone; (3) it stimulates thirst, resulting in increased fluid consumption (the presence of ADH and aldosterone ensures that the additional water consumed will be retained, elevating blood volume); and (4) it stimulates cardiac output and triggers the constriction of arterioles, in turn elevating the systemic blood pressure. The effect of angiotensin II on blood pressure is four to eight times greater than the effect of norepinephrine.

Erythropoietin

The kidneys release *erythropoietin (EPO)* if blood pressure falls or if the oxygen content of the blood becomes abnormally low (**Figure 21–17a**). EPO acts directly on blood vessels, causing vasoconstriction, thereby increasing blood pressure. EPO also stimulates the production and maturation of red blood cells. These cells increase the volume and viscosity of the blood and improve its oxygen-carrying capacity.

Natriuretic Peptides

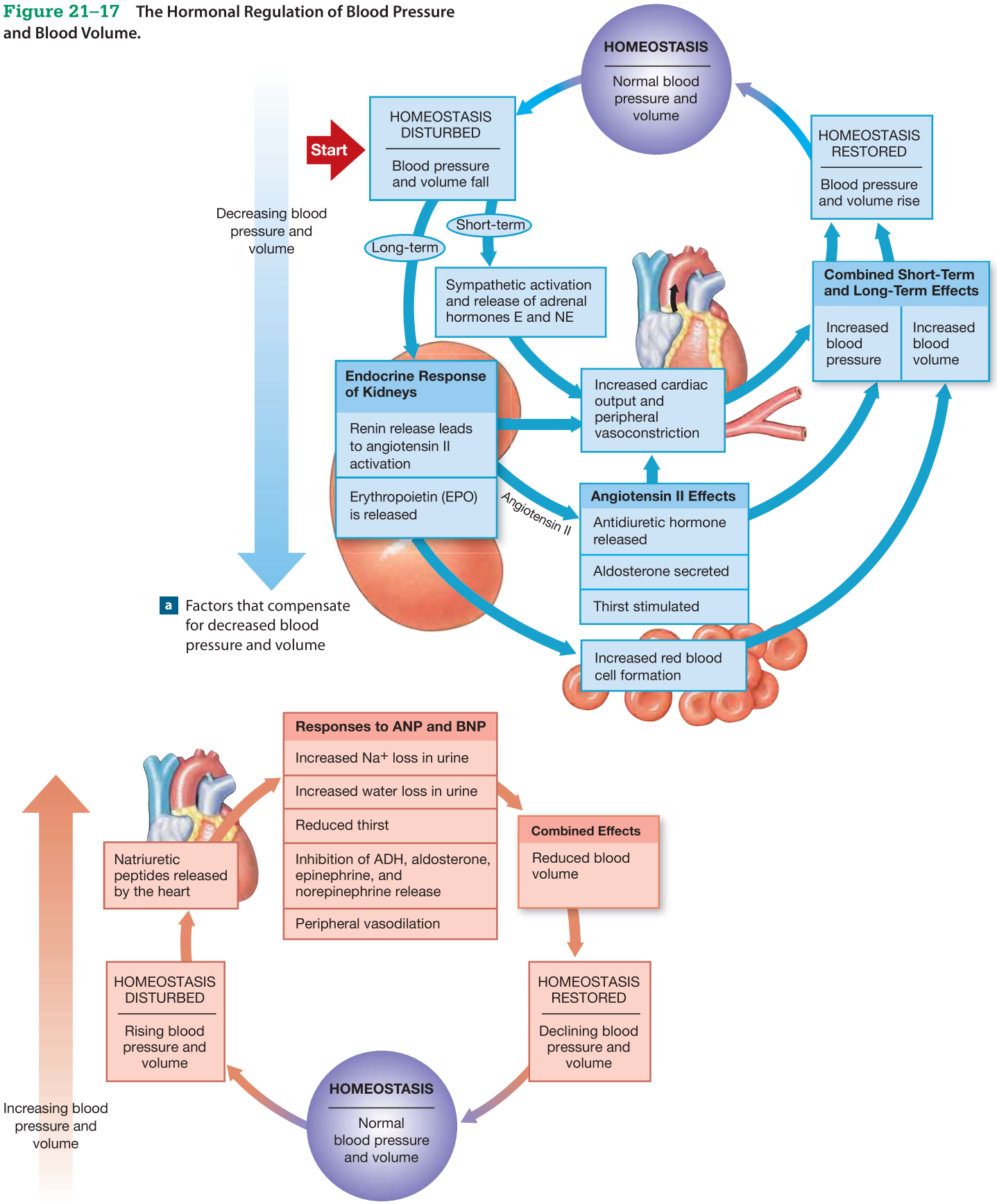
Cardiac muscle cells in the wall of the right atrium of the heart produce *atrial natriuretic peptide* (nā-trē-ū-RET-ik; *natrium*, sodium + *ouresis*, making water), or *ANP*, in response to excessive stretching during diastole. Ventricular muscle cells exposed to comparable stimuli produce a related hormone called *brain natriuretic peptide*, or *BNP*. These peptide hormones reduce blood volume and blood pressure. They do so by (1) increasing sodium ion excretion by the kidneys; (2) promoting water losses by increasing the volume of urine produced; (3) reducing thirst; (4) blocking the release of ADH, aldosterone, epinephrine, and norepinephrine; and (5) stimulating peripheral vasodilation (**Figure 21–17b**). As blood volume and blood pressure decline, the stresses on the walls of the heart are removed, and natriuretic peptide production ceases.

Checkpoint

9. **Describe the actions of vasodilators and local vasodilators.**
10. **How would applying slight pressure to the common carotid artery affect your heart rate?**
11. **What effect would the vasoconstriction of the renal artery have on blood pressure and blood volume?**

See the blue Answers tab at the back of the book.

Figure 21–17 The Hormonal Regulation of Blood Pressure and Blood Volume.



21-4 The cardiovascular system adapts to physiological stress and maintains a special vascular supply to the brain, heart, and lungs

In this chapter and the previous two, we have considered the blood, the heart, and the cardiovascular system as individual entities. Yet in our day-to-day lives, the cardiovascular system operates as an integrated complex. The interactions are fascinating and very important when physical or physiological conditions are changing rapidly.

In this section we begin by looking at the patterns of cardiovascular responses to exercise and blood loss. These two common stresses provide examples of the cardiovascular system's adaptability in maintaining homeostasis. The homeostatic responses involve interplay among the cardiovascular system, the endocrine system, and other systems. These responses are aided by autoregulation at the tissue level. Then we consider the patterns of blood supply to the brain, heart, and lungs, in which blood flow is controlled by separate mechanisms.

The Cardiovascular Response to Exercise

At rest, cardiac output averages about 5.8 liters per minute. That amount changes dramatically during exercise. In addition, the pattern of blood distribution changes markedly, as detailed in Table 21–2.

Light Exercise

Before you begin to exercise, your heart rate increases slightly due to a general rise in sympathetic activity as you think about the workout ahead. As you begin light exercise, three interrelated changes take place:

- *Extensive vasodilation occurs* as skeletal muscles consume oxygen more quickly. Peripheral resistance drops, blood flow through the capillaries increases, and blood enters the venous system at a faster rate.
- *The venous return increases* as skeletal muscle contractions squeeze blood along the peripheral veins and faster breathing pulls blood into the venae cavae via the respiratory pump.
- *Cardiac output rises*, primarily in response to (1) the rise in venous return (the Frank–Starling principle ↩ p. 700) and (2) atrial stretching (the atrial reflex). Some sympathetic stimulation occurs, leading to increases in heart rate and contractility, but there is no massive sympathetic activation. The increased cardiac output keeps pace with the elevated demand, and arterial pressures are maintained despite the drop in peripheral resistance.

This regulation by venous feedback produces a gradual increase in cardiac output to about double resting levels. The increase supports accelerated blood flow to skeletal muscles, cardiac muscle, and the skin. The flow to skeletal and cardiac muscles increases as arterioles and precapillary sphincters dilate in response to local factors. The flow to the skin increases in response to the rise in body temperature.

Table 21–2 Changes in Blood Distribution during Exercise

	Tissue Blood Flow (mL/min)		
Organ	**Rest**	**Light Exercise**	**Strenuous Exercise**
Skeletal muscles	1200	4500	12,500
Heart	250	350	750
Brain	750	750	750
Skin	500	1500	1900
Kidney	1100	900	600
Abdominal viscera	1400	1100	600
Miscellaneous	600	400	400
Total cardiac output	5800	9500	17,500

Heavy Exercise

At higher levels of exertion, other physiological adjustments take place as the cardiac and vasomotor centers activate the sympathetic nervous system. Cardiac output increases toward maximal levels. Major changes in the peripheral distribution of blood improve blood flow to active skeletal muscles.

Under massive sympathetic stimulation, the cardioaccelerator center can increase cardiac output to levels as high as 20–25 liters per minute. But that is still not enough to meet the demands of active skeletal muscles unless the vasomotor center severely restricts the blood flow to "nonessential" organs, such as those of the digestive system. During exercise at maximal levels, your blood essentially races between the skeletal muscles and the lungs and heart. Although blood flow to most tissues is diminished, skin perfusion increases further, because body temperature continues to climb. Only the blood supply to the brain remains unaffected.

Exercise, Cardiovascular Fitness, and Health

Cardiovascular performance improves significantly with training. Table 21–3 compares the cardiac performance of athletes with that of nonathletes. Trained athletes have bigger hearts and larger stroke volumes than do nonathletes, and these are important functional differences.

Recall that cardiac output is equal to the stroke volume times the heart rate. For the same cardiac output, the person with a larger stroke volume has a slower heart rate. An athlete at rest can maintain normal blood flow to peripheral tissues at a heart rate as low as 32 bpm (beats per minute). When necessary,

Table 21–3 Effects of Training on Cardiovascular Performance

Subject	Heart Weight (g)	Stroke Volume (mL)	Heart Rate (bpm)	Cardiac Output (L/min)	Blood Pressure (systolic/diastolic)
Nonathlete (rest)	300	60	83	5.0	120/80
Nonathlete (maximum)		104	192	19.9	187/75
Trained athlete (rest)	500	100	53	5.3	120/80
Trained athlete (maximum)		167	182	30.4	200/90*

* Diastolic pressures in athletes during maximal activity have not been accurately measured.

the athlete's cardiac output can increase to levels 50 percent higher than those of nonathletes. Thus, a trained athlete can tolerate sustained levels of activity that are well beyond the capabilities of nonathletes.

Exercise and Cardiovascular Disease

Regular exercise has several beneficial effects. Even a moderate exercise routine (jogging 5 miles a week, for example) can lower total blood cholesterol levels. Exercise lowers cholesterol by stimulating enzymes that help move low-density lipoproteins (LDLs, or so-called "bad cholesterol") from the blood to the liver. In the liver, the cholesterol is converted to bile and excreted from the body. Exercise also increases the size of the lipoprotein particles that carry cholesterol, making it harder for small proteins to lodge in the vessel walls. A high cholesterol level is one of the major risk factors for atherosclerosis, which leads to cardiovascular disease and strokes. In addition, a healthy lifestyle that includes regular exercise, a balanced diet, weight control, and not smoking, reduces stress, lowers blood pressure, and slows the formation of plaques.

Regular moderate exercise (30 minutes most days of the week) may cut the incidence of heart attacks almost in half. However, only an estimated 8 percent of adults in the United States currently exercise at recommended levels. Exercise also speeds recovery after a heart attack. Regular light-to-moderate exercise (such as walking, jogging, or bicycling), coupled with a low-fat diet and a low-stress lifestyle, reduces symptoms of coronary artery disease (such as angina). Such exercise also improves a person's mood and overall quality of life. However, exercise does not remove underlying medical problems. For example, atherosclerotic plaques, described on p. 712, do not disappear and seldom grow smaller with exercise.

There is no evidence that *intense* athletic training lowers the incidence of cardiovascular disease. On the contrary, the strains placed on all body systems—including the cardiovascular system—during an ultramarathon, iron-man triathlon, or other extreme athletic event can be severe. Individuals with congenital aneurysms, cardiomyopathy, or cardiovascular disease risk fatal cardiovascular problems, such as an arrhythmia or heart attack, during severe exercise. Even healthy individuals can develop acute physiological disorders, such as kidney failure, after extreme exercise. We discuss the effects of exercise on other systems in later chapters.

The Cardiovascular Response to Hemorrhaging

In Chapter 19, we considered the local cardiovascular reaction to a break in the wall of a blood vessel. ↩ p. 661 When hemostasis fails to prevent significant blood loss, the entire cardiovascular system makes adjustments (**Figure 21–18**). The immediate problem is to maintain adequate blood pressure and peripheral blood flow. The long-term problem is to restore normal blood volume.

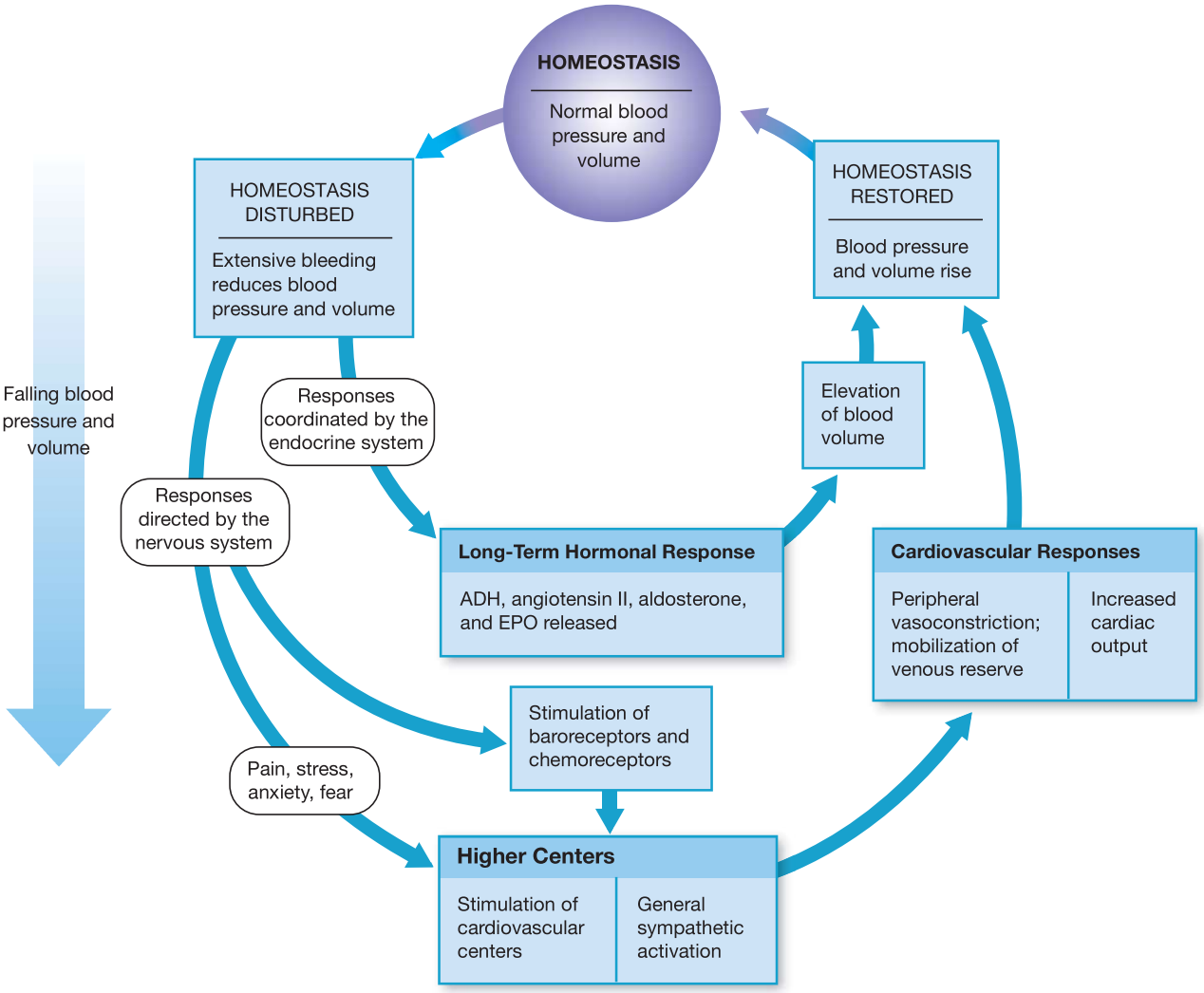
Short-Term Elevation of Blood Pressure

Almost as soon as the pressures start to decline, several short-term responses appear:

- In the initial neural response, carotid and aortic reflexes increase cardiac output and cause peripheral vasoconstriction (pp. 728–730). With blood volume reduced, an increase in heart rate, typically up to 180–200 bpm, maintains cardiac output.
- The combination of stress and anxiety stimulates the sympathetic nervous system headquarters in the hypothalamus, which in turn triggers a further increase in vasomotor tone, constricting the arterioles and raising blood pressure. At the same time, venoconstriction mobilizes the venous reserve and quickly improves venous return (p. 717).
- Short-term hormonal effects also occur. For instance, sympathetic activation causes the adrenal medullae to secrete E and NE. These hormones increase cardiac output and extend peripheral vasoconstriction. In addition, the release of ADH by the posterior lobe of the pituitary gland and the production of angiotensin II enhance vasoconstriction as part of the long-term response.

This combination of short-term responses elevates blood pressure and improves peripheral blood flow. It often restores normal arterial pressures and peripheral circulation after blood losses of up to 20 percent of total blood volume. Such adjustments are more than enough to compensate for the blood loss when you donate blood. (Most blood banks collect 500 mL of whole blood, roughly 10 percent of your total blood volume.) If compensatory mechanisms fail, the individual develops signs of *shock*.

Figure 21–18 Cardiovascular Responses to Hemorrhaging and Blood Loss. These mechanisms can cope with blood losses equivalent to approximately 30 percent of total blood volume.



Long-Term Restoration of Blood Volume

Short-term responses temporarily compensate for a reduction in blood volume. Long-term responses are geared to restoring normal blood volume. This process can take several days after a serious hemorrhage. The steps include the following:

- The decline in capillary blood pressure triggers a recall of fluids from the interstitial spaces (p. 725).
- Aldosterone and ADH promote fluid retention and reabsorption at the kidneys, preventing further reductions in blood volume.
- Thirst increases, and the digestive tract absorbs additional water. This intake of fluid increases the plasma volume and ultimately replaces the interstitial fluids "borrowed" at the capillaries.
- Erythropoietin targets the bone marrow. It stimulates the maturation of red blood cells, which increases blood volume and improves oxygen delivery to peripheral tissues.

Vascular Supply to Special Regions

The vasoconstriction that takes place in response to a drop in blood pressure or a rise in CO_2 levels affects many tissues and organs at the same time. The term *special circulation* refers to the vascular supply through organs in which blood flow is controlled by separate mechanisms. Let's consider three important examples: the blood flow to the brain, the heart, and the lungs.

Blood Flow to the Brain

In Chapter 14, we noted that the blood–brain barrier isolates most CNS tissue from the general circulation. ↩ p. 455 The brain has a very high demand for oxygen and receives a substantial supply of blood. Under a variety of conditions, blood flow to the brain remains steady at about 750 mL/min. That means that roughly 12 percent of the cardiac output is delivered to an organ that is less than 2 percent of body weight.

Neurons do not have significant energy reserves, and in functional terms the cardiovascular system treats blood flow to the brain as the top priority. Even during a cardiovascular crisis, blood flow through the brain remains as near normal as possible: While the cardiovascular centers are calling for widespread peripheral vasoconstriction, the cerebral vessels are instructed to dilate.

Total blood flow to the brain remains relatively constant, but blood flow to specific regions of the brain changes from moment to moment. These changes occur in response to local changes in the composition of interstitial fluid that accompany neural activity. When you read, write, speak, or walk, specific regions of your brain become active. Blood flow to those regions increases almost instantaneously. These changes ensure that the active neurons will receive the oxygen and nutrients they require.

The brain receives arterial blood through four arteries. An interruption of flow in any one of these large vessels does not significantly reduce blood flow to the brain as a whole because these arteries form anastomoses inside the cranium. However, a plaque or a blood clot may still block a small artery, and weakened arteries may rupture. Such incidents temporarily or permanently shut off blood flow to a localized area of the brain, damaging or killing the dependent neurons. Signs and symptoms of a *stroke*, or *cerebrovascular accident (CVA)*, then appear.

Blood Flow to the Heart

We described the anatomy of the coronary circulation in Chapter 20. ↩ p. 680 The coronary arteries arise at the base of the ascending aorta, where systemic pressures are highest. Each time the heart contracts, it squeezes the coronary vessels, so blood flow is reduced. In the left ventricle, systolic pressures are high enough that blood can flow into the myocardium only during diastole. Over this period, elastic rebound helps drive blood along the coronary vessels. Normal cardiac muscle cells can tolerate these brief circulatory interruptions because they have substantial oxygen reserves.

When you are at rest, coronary blood flow is about 250 mL/min. When the workload on your heart increases, local factors, such as reduced O_2 levels and increased lactic acid, dilate the coronary vessels and increase blood flow. Epinephrine released during sympathetic stimulation promotes the vasodilation of coronary vessels. It also increases heart rate and the strength of cardiac contractions. As a result, coronary blood flow increases while vasoconstriction occurs in other tissues.

For reasons that are not clear, some individuals have *coronary spasms*. These spasms can temporarily restrict coronary circulation and produce symptoms of angina. The heart's ability to increase its output, even under maximal stimulation, can be limited by certain conditions. These conditions include a permanent restriction or blockage of coronary vessels (as in coronary artery disease) and tissue damage (as caused by a myocardial infarction). When the cardiac workload increases much above resting levels, individuals with these conditions experience signs and symptoms of heart failure.

Blood Flow to the Lungs

The lungs have roughly 300 million *alveoli* (al-VĒ-ō-lī; *alveolus*, sac), delicate epithelial pockets where gas exchange takes place. An extensive capillary network surrounds each alveolus. Local responses to oxygen levels within individual alveoli regulate blood flow through the lungs. How does this local regulation work? When an alveolus contains plenty of oxygen, its blood vessels dilate. Blood flow then increases, promoting the absorption of oxygen from air inside the alveolus. When the oxygen content of the air is very low, the vessels constrict. They shunt blood to other alveoli that contain more oxygen. This local mechanism makes the respiratory system very efficient. There is no benefit in circulating blood through the capillaries of an alveolus that contains little oxygen.

This mechanism is precisely the opposite of that in other tissues, where a decline in oxygen levels causes local vasodilation rather than vasoconstriction. The difference makes functional sense, but its physiological basis remains a mystery.

Blood pressure in pulmonary capillaries (average: 10 mm Hg) is lower than that in systemic capillaries. The BCOP (25 mm Hg) is the same as elsewhere in the bloodstream. As a result, reabsorption exceeds filtration in pulmonary capillaries. Fluid moves continuously into the pulmonary capillaries across the alveolar surfaces. This flow prevents fluid from building up in the alveoli and interfering with the diffusion of respiratory gases. If the blood pressure in pulmonary capillaries rises above 25 mm Hg, fluid enters the alveoli, causing *pulmonary edema*.

Checkpoint

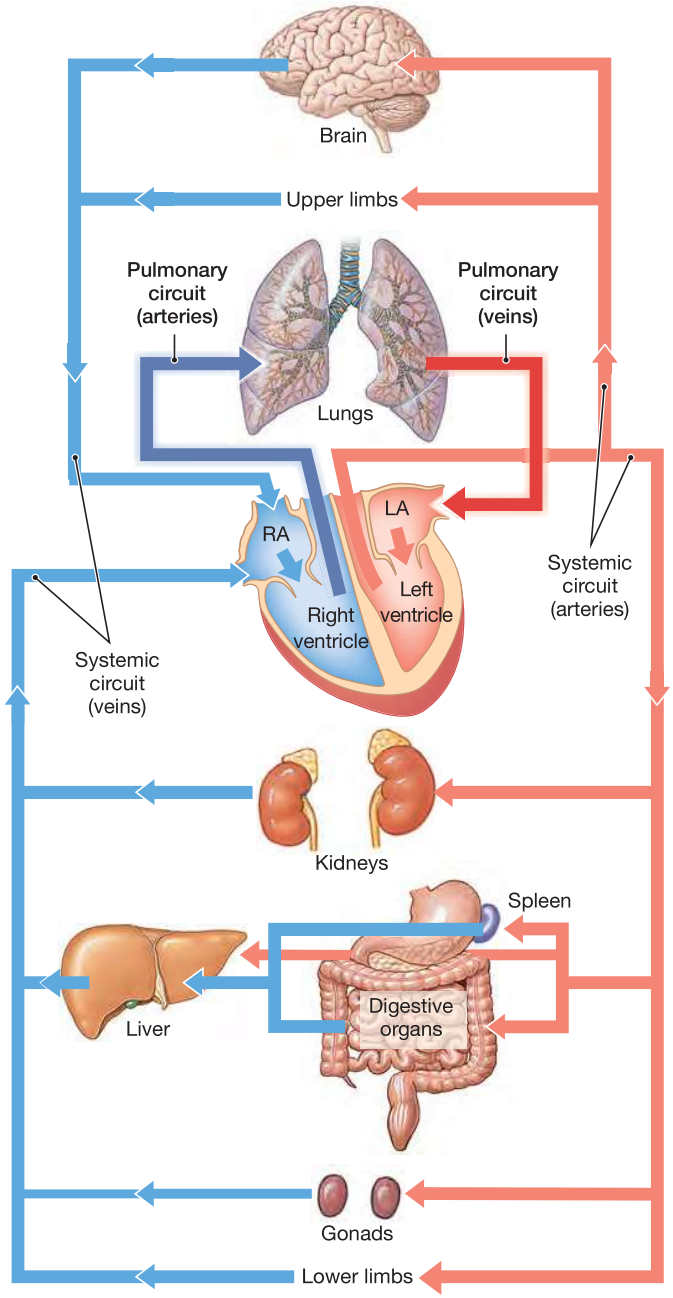
12. **Why does blood pressure increase during exercise?**
13. **Name the immediate and long-term problems related to the cardiovascular response to hemorrhaging.**
14. **Explain the role of aldosterone and ADH in long-term restoration of blood volume.**

See the blue Answers tab at the back of the book.

21-5 The pulmonary and systemic circuits of the cardiovascular system exhibit three general functional patterns

You already know that the cardiovascular system consists of the *pulmonary circuit* and the *systemic circuit*. The pulmonary circuit consists of arteries and veins that transport blood between the heart and the lungs. This circuit begins at the right ventricle and ends at the left atrium. From the left ventricle, the arteries of the systemic circuit transport oxygenated blood and nutrients to all organs and tissues. Veins of the systemic circuit ultimately return deoxygenated blood to the right atrium. **Figure 21–19** summarizes the main distribution routes within the pulmonary and systemic circuits.

Figure 21–19 A Schematic Overview of the Pattern of Circulation. *RA* stands for right atrium, *LA* for left atrium.



In the pages that follow, we examine the vessels of the pulmonary and systemic circuits further. Three major patterns of blood vessel organization are worth noting:

1. The peripheral distributions of arteries and veins on the body's left and right sides are generally identical, except near the heart, where the largest vessels connect to the atria or ventricles. Corresponding arteries and veins usually follow the same path. For example, the left and right subclavian *arteries* parallel the left and right subclavian *veins*.
2. A single vessel may have several names as it crosses specific anatomical boundaries. These names make accurate anatomical descriptions possible when the vessel extends far into the periphery. For example, the *external iliac artery* becomes the *femoral artery* as it leaves the trunk and enters the lower limb.
3. Several arteries and veins usually service tissues and organs. Often, anastomoses between adjacent arteries or veins reduce the impact of a temporary or even permanent blockage, or *occlusion*, in a single blood vessel.

Checkpoint

15. **Identify the two circuits of the cardiovascular system.**
16. **Identify the three major patterns of blood vessel organization seen in the pulmonary and systemic circuits of the cardiovascular system.**

See the blue Answers tab at the back of the book.

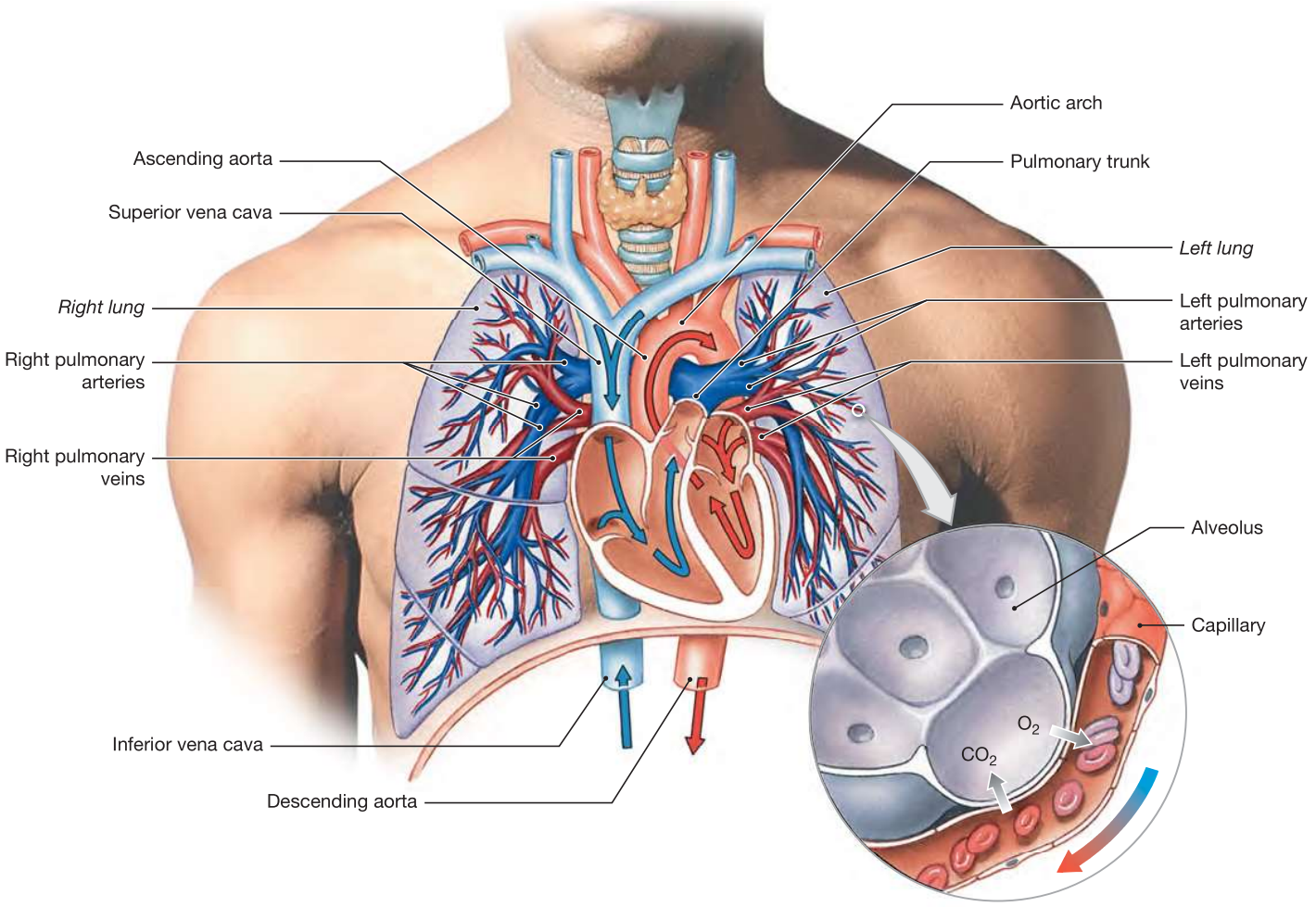
21-6 In the pulmonary circuit, deoxygenated blood enters the lungs in arteries, and oxygenated blood leaves the lungs via veins

Blood entering the right atrium has just returned from the peripheral capillary beds, where it released oxygen and absorbed carbon dioxide. After traveling through the right atrium and ventricle, this deoxygenated blood enters the pulmonary trunk, the start of the pulmonary circuit (**Figure 21–20**). At the lungs, oxygen is replenished, and carbon dioxide is released. The oxygenated blood returns to the heart for distribution via the systemic circuit.

Compared with the systemic circuit, the pulmonary circuit is short: The base of the pulmonary trunk and the lungs are only about 15 cm (6 in.) apart.

The arteries of the pulmonary circuit differ from those of the systemic circuit in that they carry deoxygenated blood. (For this reason, most color-coded diagrams show the pulmonary arteries in blue, the same color as systemic veins.) As the pulmonary trunk curves over the superior border of the heart, it gives rise to the **left** and **right pulmonary arteries.** These large arteries enter the lungs before branching repeatedly, giving rise to smaller and smaller arteries. The smallest branches, the *pulmonary arterioles*, provide blood to capillary networks that surround *alveoli*. The walls of these small air pockets are thin enough for gas to be exchanged between the capillary blood and inspired air. As oxygenated blood leaves the alveolar capillaries, it enters venules that in turn unite to form larger vessels carrying blood toward the **pulmonary veins.** These four veins, two from each lung, empty into the left atrium, completing the pulmonary circuit.

Figure 21–20 The Pulmonary Circuit. The pulmonary circuit consists of pulmonary arteries, which deliver deoxygenated blood from the right ventricle to the lungs; pulmonary capillaries, where gas exchange occurs; and pulmonary veins, which deliver oxygenated blood to the left atrium. As the enlarged view shows, diffusion across the capillary walls at alveoli removes carbon dioxide and provides oxygen to the blood.
ATLAS: Plates 42a; 44c; 47b



Tips & Tricks

Arteries and veins are defined by the direction of blood flow relative to the heart, not by the oxygen content of the blood they carry. So if you remember that ***a***rteries carry blood ***a***way from the heart, and veins carry blood toward the heart, you can remember that the pulmonary ***a***rteries carry oxygen-poor blood ***a***way from the heart to the lungs, and the pulmonary veins deliver oxygen-rich blood to the heart.

Checkpoint

17. Name the blood vessels that enter and exit the lungs, and indicate the relative oxygen content of the blood in each.
18. Trace the path of a drop of blood through the lungs, beginning at the right ventricle and ending at the left atrium.

See the blue Answers tab at the back of the book.

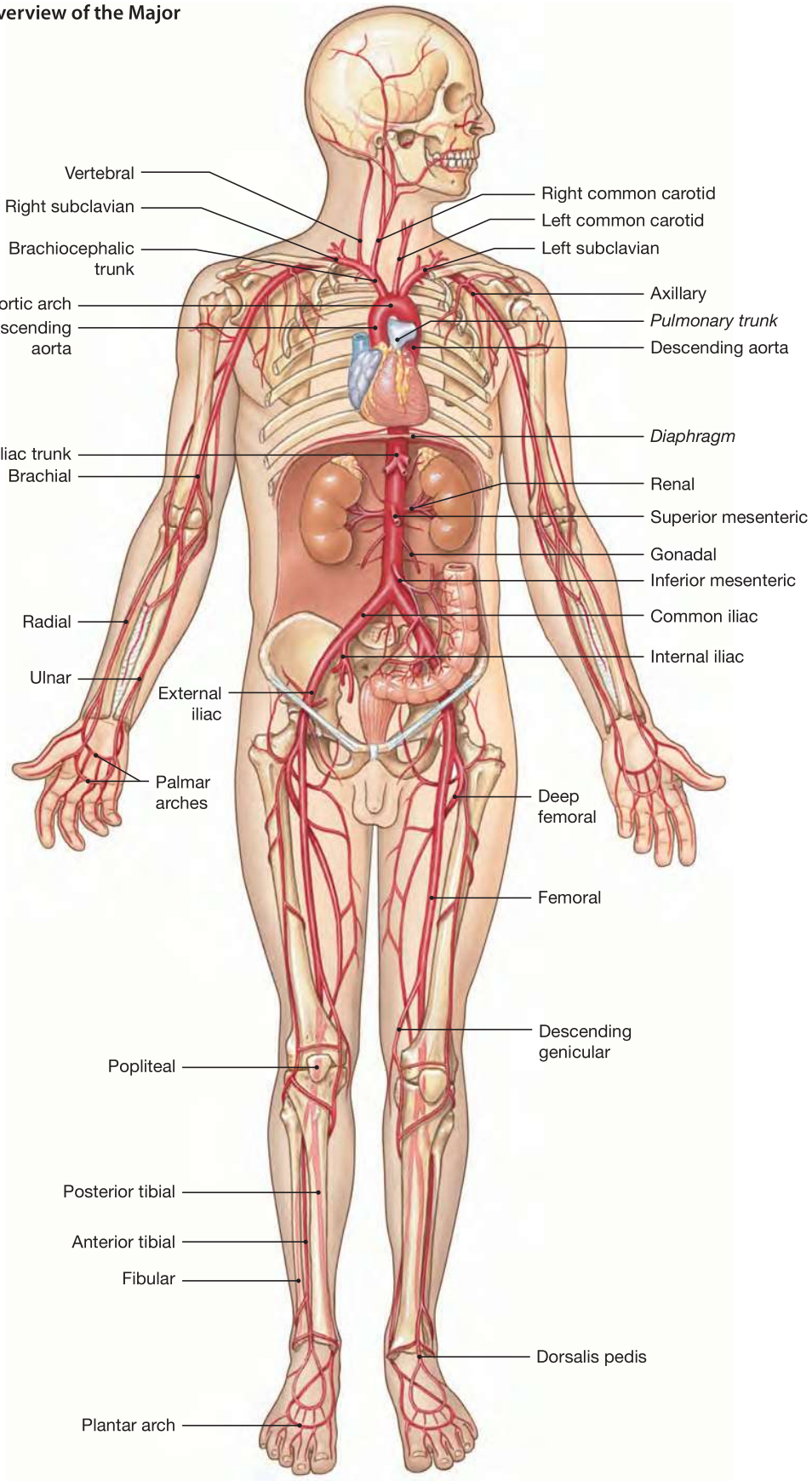
21-7 The systemic circuit carries oxygenated blood from the left ventricle to tissues and organs other than the pulmonary exchange surfaces, and returns deoxygenated blood to the right atrium

The systemic circuit supplies the capillary beds in all parts of the body not serviced by the pulmonary circuit. This circuit begins at the left ventricle and ends at the right atrium. At any moment the systemic circuit contains about 84 percent of total blood volume.

Systemic Arteries

Figure 21–21 provides an overview of the systemic arterial system and shows the relative locations of major systemic arteries.

Figure 21–21 An Overview of the Major Systemic Arteries.



Figures 21–22 to 21–27 show the detailed distribution of these vessels and their branches. By convention, several large arteries are called *trunks*. Examples are the *pulmonary*, *brachiocephalic*, *thyrocervical*, and *celiac trunks*. Most of the major arteries are paired, with one artery of each pair on either side of the body. For this reason, the terms *right* and *left* appear in figures only when the arteries on both sides are labeled.

The Ascending Aorta

The **ascending aorta** (Figure 21–22) begins at the aortic valve of the left ventricle. The left and right coronary arteries originate in the aortic sinus at the base of the ascending aorta, just superior to the aortic valve. The distribution of coronary vessels was described in Chapter 20 and illustrated in Figure 20–9, p. 681.

The Aortic Arch

The **aortic arch** curves like the handle of a cane across the superior surface of the heart, connecting the ascending aorta with the *descending aorta* (Figure 21–21). Three elastic arteries originate along the aortic arch and deliver blood to the head, neck, shoulders, and upper limbs: (1) the **brachiocephalic** (brā-kē-ō-se-FAL-ik) **trunk**, (2) the **left common carotid artery,** and (3) the **left subclavian artery** (Figures 21–22 and 21–23). The brachiocephalic trunk, also called the *innominate artery* (i-NOM-i-nat; unnamed), ascends for a short distance before branching to form the **right subclavian artery** and the **right common carotid artery.**

We have only one brachiocephalic trunk, with the left common carotid and left subclavian arteries arising separately from the aortic arch. However, in terms of their peripheral distribution, the vessels on the left side are mirror images of those on the right side. Figures 21–22 and 21–23 illustrate the major branches of these arteries.

The Subclavian Arteries. The subclavian arteries supply blood to the arms, chest wall, shoulders, back, and CNS (Figures 21–21 and 21–22). Three major branches arise before a subclavian artery leaves the thoracic cavity: (1) the **internal thoracic artery,** supplying the pericardium and anterior wall of the chest; (2) the **vertebral artery,** which provides blood to the brain and spinal cord; and (3) the **thyrocervical trunk,** which provides blood to muscles and other tissues of the neck, shoulder, and upper back.

After leaving the thoracic cavity and passing across the superior border of the first rib, the subclavian is called the **axillary artery.** This artery crosses the axilla to enter the arm, where it gives rise to *humeral circumflex arteries*, which supply structures near the head of the humerus. Distally, it becomes the **brachial artery,** which supplies blood to the rest of the upper limb. The brachial artery gives rise to the *deep brachial artery*, which supplies deep structures on the posterior aspect of the arm, and the *ulnar collateral arteries*, which supply the area around the elbow.

As it approaches the coronoid fossa of the humerus, the brachial artery divides into the **radial artery,** which follows the radius, and the **ulnar artery,** which follows the ulna to the wrist. These arteries supply blood to the forearm and, through the *ulnar recurrent arteries*, the region around the elbow. At the wrist, the radial and ulnar arteries fuse to form the **superficial** and **deep palmar arches,** which supply blood to the hand and to the **digital arteries** of the thumb and fingers.

The Carotid Artery and the Blood Supply to the Brain. The common carotid arteries ascend deep in the tissues of the neck. You can usually locate the carotid artery by pressing gently along either side of the windpipe (trachea) until you feel a strong pulse.

Each common carotid artery divides into an **external carotid artery** and an **internal carotid artery** (Figure 21–23). The **carotid sinus,** located at the base of the internal carotid artery, may extend along a portion of the common carotid. The external carotid arteries supply blood to the structures of the neck, esophagus, pharynx, larynx, lower jaw, and face. The internal carotid arteries enter the skull through the carotid canals of the temporal bones, delivering blood to the brain (Figure 7–3 and 7–4, pp. 201–203.)

Tips & Tricks

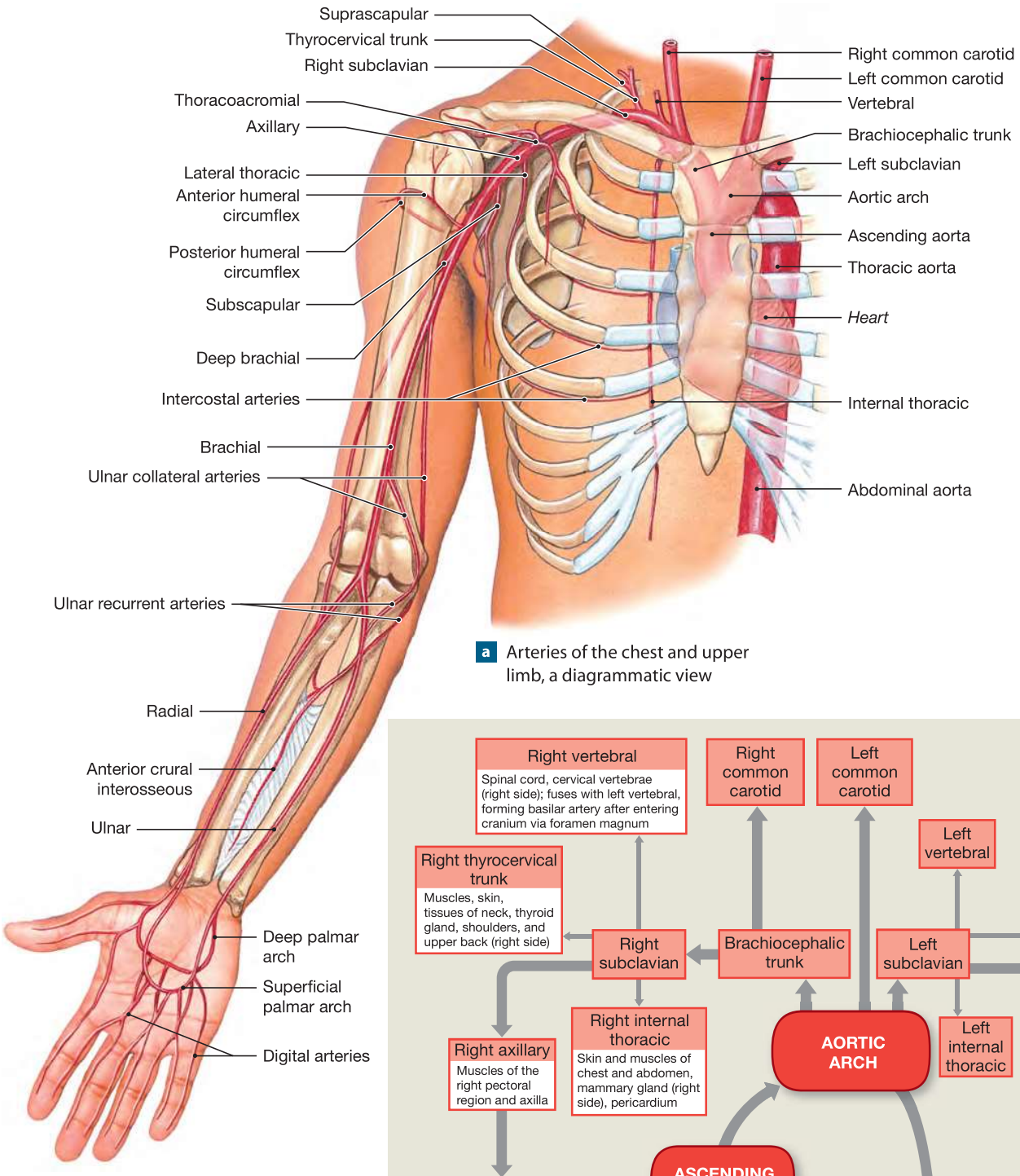
Remember that the *external* carotid artery supplies the face (which is external), and that the *internal* carotid supplies the brain (which is internal).

The internal carotid arteries ascend to the level of the optic nerves, where each artery divides into three branches: (1) an **ophthalmic artery,** which supplies the eyes; (2) an **anterior cerebral artery,** which supplies the frontal and parietal lobes of the brain; and (3) a **middle cerebral artery,** which supplies the midbrain and lateral surfaces of the cerebral hemispheres (Figures 21–23 and 21–24).

The brain is extremely sensitive to changes in blood supply. An interruption of blood flow for several seconds produces unconsciousness. After four minutes some permanent neural damage can occur. Such crises are rare, because blood reaches the brain through the vertebral arteries as well as by way of the internal carotid arteries. The left and right vertebral arteries arise from the subclavian arteries and ascend within the transverse foramina of the cervical vertebrae (Figure 7–19b,c, p. 221). The vertebral arteries enter the cranium at the foramen magnum, where they fuse along the ventral surface of the medulla oblongata to form the **basilar artery.** The vertebral arteries and the basilar artery supply blood to the spinal cord, medulla oblongata, pons, and cerebellum. They then divide into the **posterior cerebral arteries,** which in turn branch off into the **posterior communicating arteries** (Figure 21–24).

The internal carotid arteries normally supply the arteries of the anterior half of the cerebrum, and the rest of the brain receives

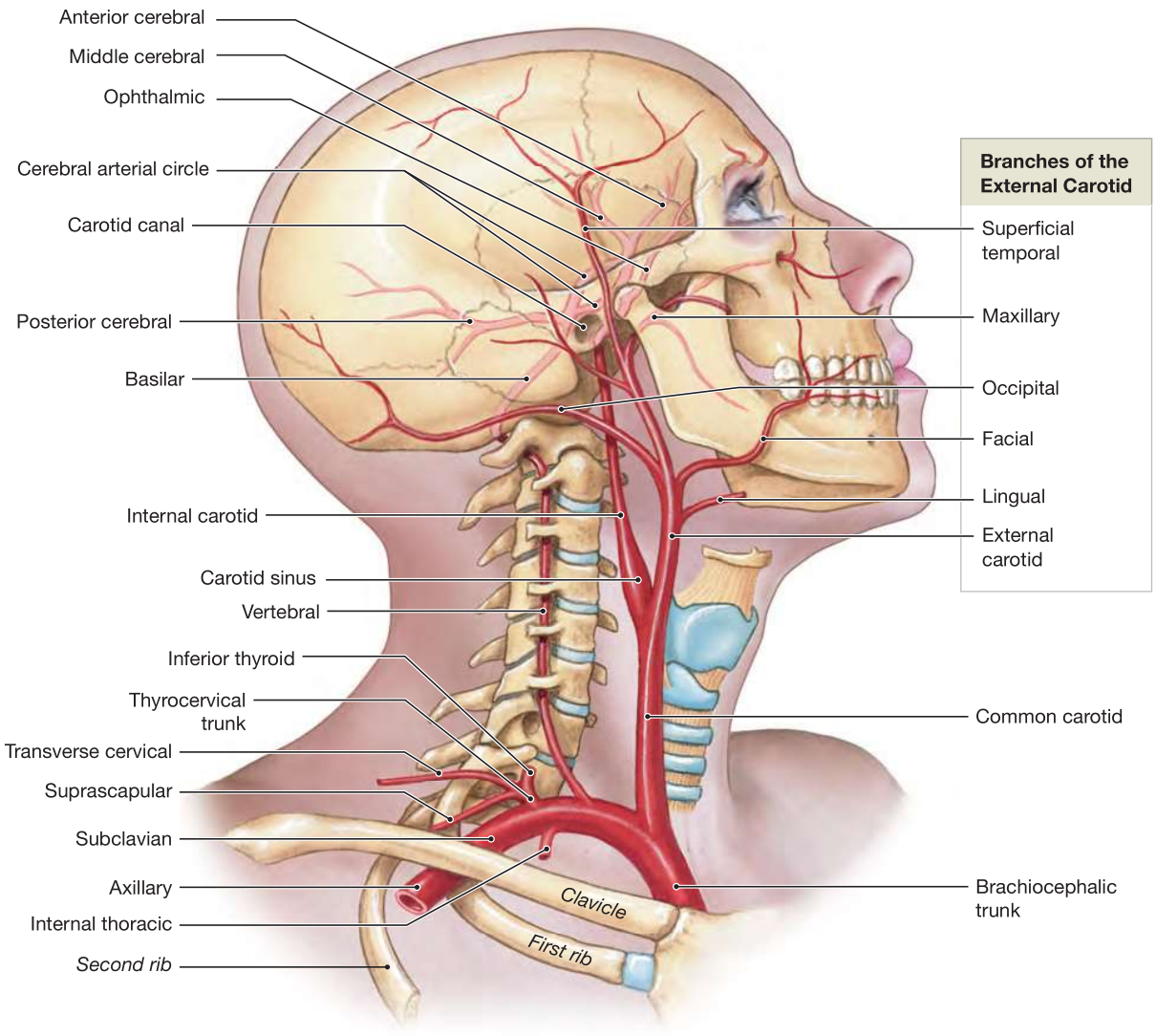
Figure 21–22 **Arteries of the Chest and Upper Limb.** ATLAS: Plates 27a–c; 29c; 30; 45a



a Arteries of the chest and upper limb, a diagrammatic view

b A flowchart of the arteries of the chest and upper limb

Figure 21–23 Arteries of the Neck and Head. Shown as seen from the right side. ATLAS: Plates 3c,d; 15b; 18a–c; 45a



blood from the vertebral arteries. But this pattern of blood flow can easily change, because the internal carotid arteries and the basilar artery are interconnected. They form a ring-shaped anastomosis called the **cerebral arterial circle,** or *circle of Willis*, which encircles the infundibulum of the pituitary gland (**Figure 21–24**). With this arrangement, the brain can receive blood from either the carotid or the vertebral arteries, reducing the likelihood of a serious interruption of circulation.

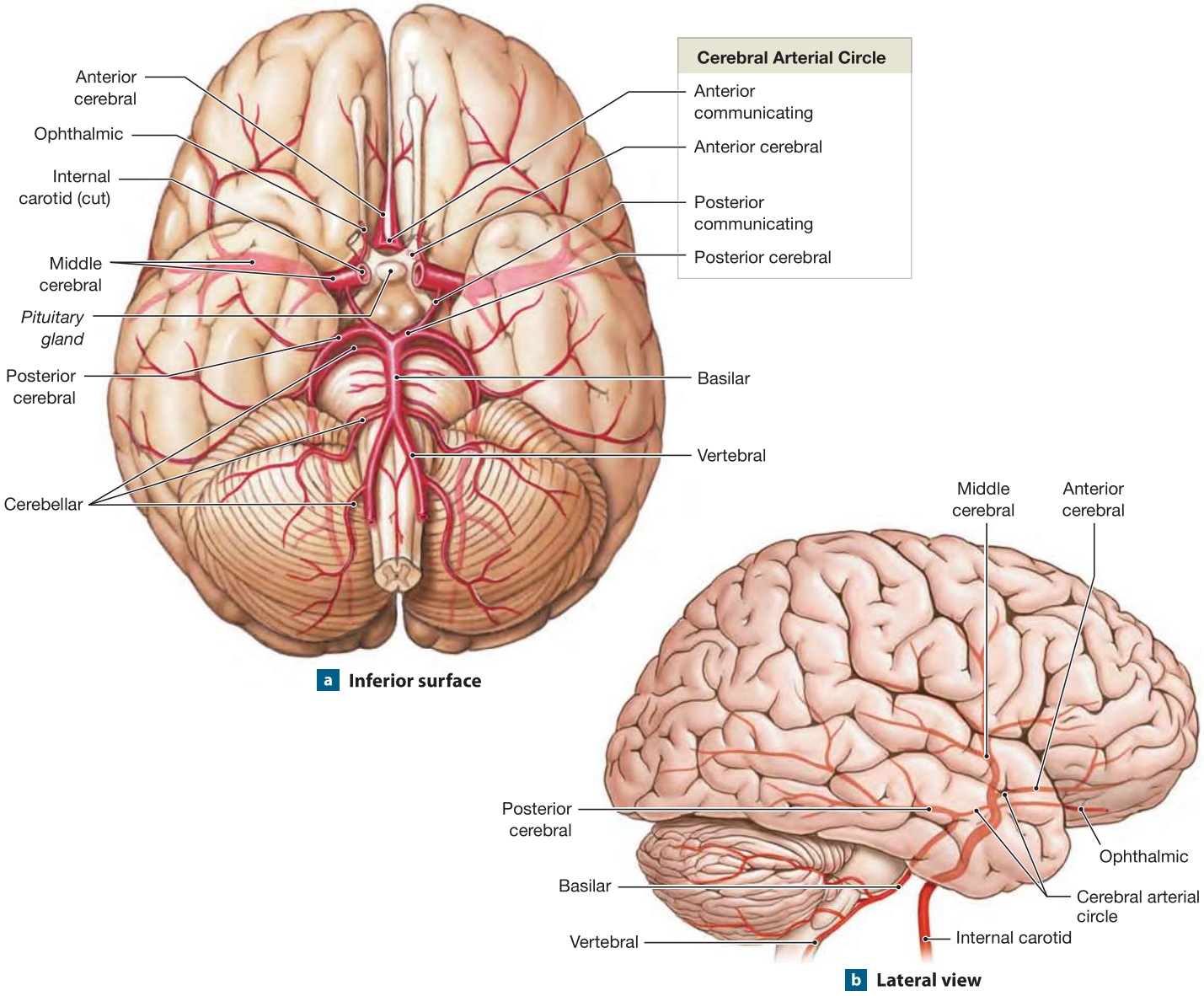
Strokes, or *cerebrovascular accidents (CVAs)*, are interruptions of the vascular supply to a portion of the brain. The *middle cerebral artery*, a major branch of the cerebral arterial circle, is the most common site of a stroke. Superficial branches deliver blood to the temporal lobe and large portions of the frontal and parietal lobes. Deep branches supply the basal nuclei and portions of the thalamus. If a stroke blocks the middle cerebral artery on the left side of the brain, aphasia and a sensory and motor paralysis of the right side of the body result. In a stroke affecting the middle cerebral artery on the right side, the individual experiences a loss of sensation and motor control over the left side of the body and has difficulty drawing or interpreting spatial relationships. Strokes affecting vessels that supply the brain stem also produce distinctive symptoms. Strokes affecting the lower brain stem are commonly fatal.

The Descending Aorta

The **descending aorta** is continuous with the aortic arch. The diaphragm divides the descending aorta into a superior **thoracic aorta** and an inferior **abdominal aorta** (**Figures 21–25** and **21–26**).

The Thoracic Aorta. The thoracic aorta begins at the level of vertebra T_5 and penetrates the diaphragm at the level of vertebra T_{12}.

Figure 21–24 **Arteries of the Brain.** ATLAS: Plate 15a–c



It travels within the mediastinum, on the posterior thoracic wall, slightly to the left of the vertebral column. This vessel supplies blood to branches that service the tissues and organs of the mediastinum, the muscles of the chest and the diaphragm, and the thoracic spinal cord.

We group the branches of the thoracic aorta anatomically as either visceral or parietal (**Figure 21–25**):

- *Visceral branches* supply the organs of the chest. The **bronchial arteries** supply the tissues of the lungs not involved in gas exchange. The **pericardial arteries** supply the pericardium. The **esophageal arteries** supply the esophagus, and the **mediastinal arteries** supply the tissues of the mediastinum.
- *Parietal branches* supply the chest wall. The **intercostal arteries** supply the chest muscles and the vertebral column area. The **superior phrenic** (FREN-ik) **arteries** deliver blood to the superior surface of the diaphragm, which separates the thoracic and abdominopelvic cavities.

The Abdominal Aorta. The abdominal aorta is a continuation of the thoracic aorta (**Figure 21–25**). It begins immediately inferior to the diaphragm and descends slightly to the left of the vertebral column but posterior to the peritoneal

cavity. A cushion of adipose tissue commonly surrounds the abdominal aorta. At the level of vertebra L_4, the abdominal aorta splits into two major arteries—the *left* and *right common iliac arteries*—that supply deep pelvic structures and the lower limbs. The region where the abdominal aorta splits is called the *terminal segment of the aorta*.

The abdominal aorta delivers blood to all the abdominopelvic organs and structures. The major branches to visceral organs are unpaired. They arise on the anterior surface of the abdominal aorta and extend into the mesenteries. By contrast, branches to the body wall, the kidneys, the urinary bladder, and other structures outside the peritoneal cavity are paired. They originate along the lateral surfaces of the abdominal aorta. **Figure 21–25** shows the major arteries of the trunk after most thoracic and abdominal organs have been removed. **Figure 21–26** shows the distribution of those arteries to abdominopelvic organs.

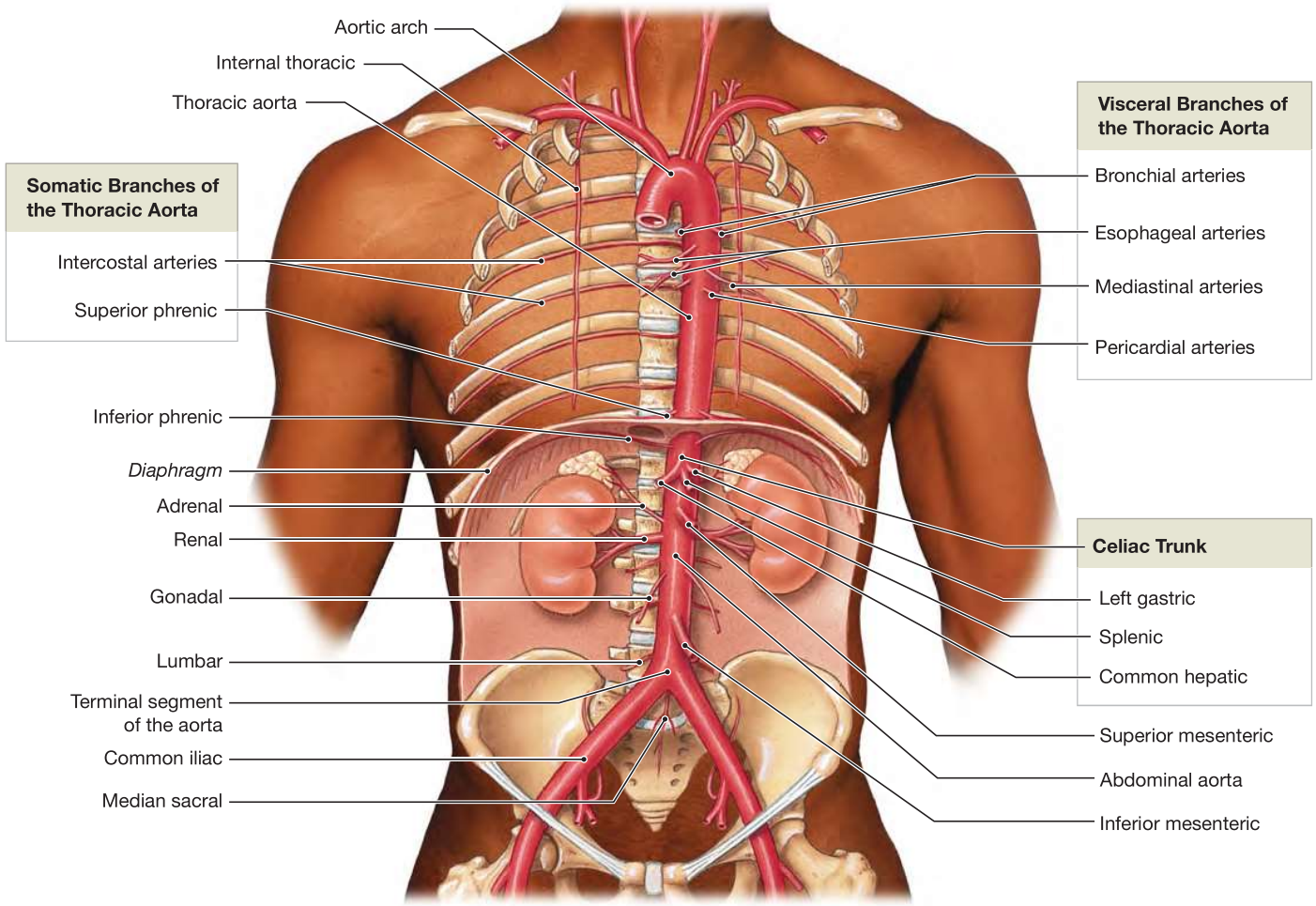
Tips & Tricks

The aorta resembles a walking cane: the ascending aorta, aortic arch, and the start of the descending aorta form the cane's handle, while the thoracic and abdominal segments of the descending aorta form the cane's shaft.

The abdominal aorta gives rise to three unpaired arteries (**Figures 21–25** and **21–26**).

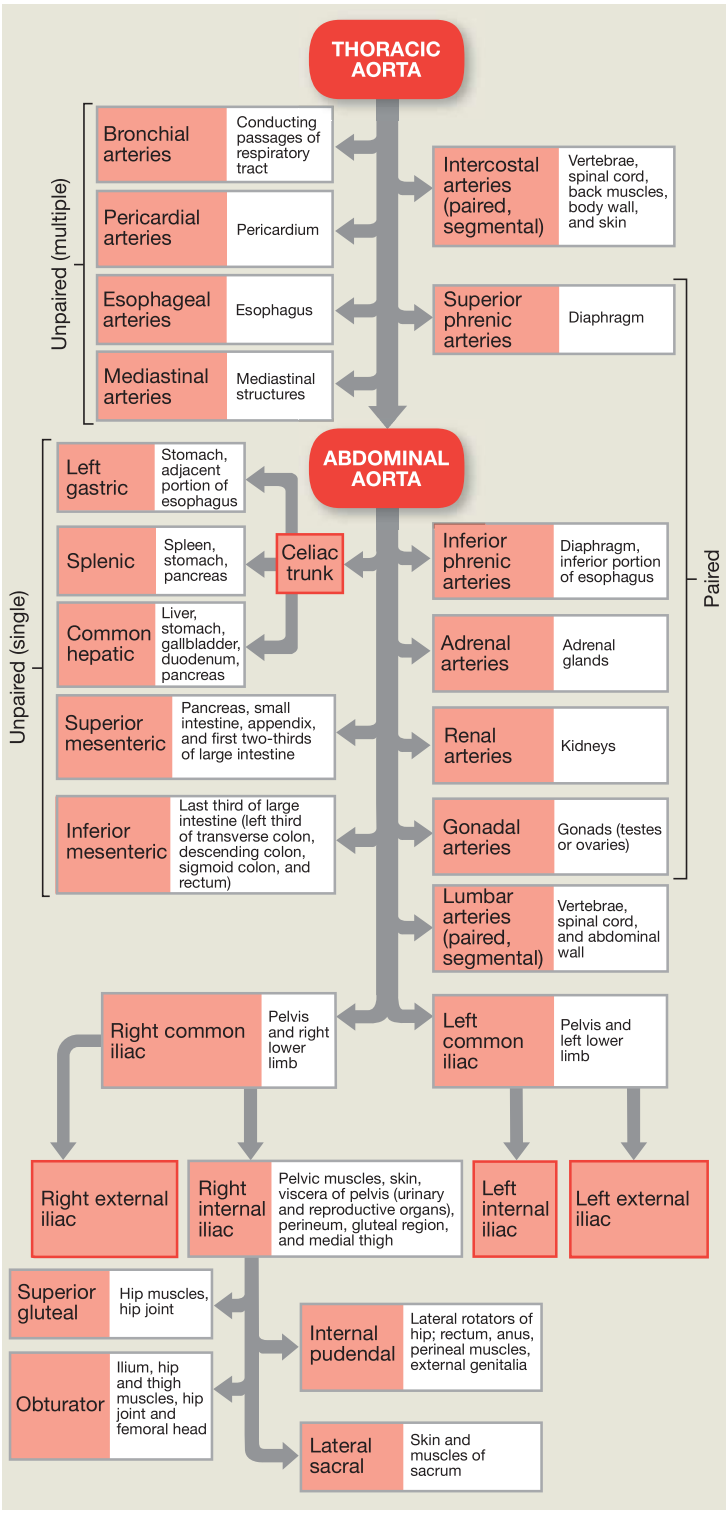
1. The **celiac** (SĒ-lē-ak) **trunk** delivers blood to the liver, stomach, and spleen. The celiac trunk divides into three branches: (a) The **left gastric artery** supplies the stomach

Figure 21–25 **Major Arteries of the Trunk.** ATLAS: Plates 47d; 53c,e; 62a,b.



a A diagrammatic view, with most of the thoracic and abdominal organs removed

Figure 21–25 Major Arteries of the Trunk *(continued)*.



b A flowchart showing major arteries of the trunk

and the inferior portion of the esophagus. (b) The **splenic artery** supplies the spleen and arteries to the stomach *(left gastroepiploic artery)* and pancreas *(pancreatic arteries)*. (c) The **common hepatic artery** supplies arteries to the liver *(hepatic artery proper)*, stomach *(right gastric artery)*, gallbladder *(cystic artery)*, and duodenal area *(gastroduodenal, right gastroepiploic,* and *superior pancreaticoduodenal arteries)*.

2. The **superior mesenteric** (mez-en-TER-ik) **artery** arises about 2.5 cm (1 in.) inferior to the celiac trunk. It supplies arteries to the pancreas and duodenum *(inferior pancreaticoduodenal artery)*, small intestine *(intestinal arteries)*, and most of the large intestine *(right* and *middle colic* and the *ileocolic arteries)*.
3. The **inferior mesenteric artery** arises about 5 cm (2 in.) superior to the terminal aorta. It delivers blood to the terminal portions of the colon *(left colic* and *sigmoid arteries)* and the rectum *(rectal arteries)*.

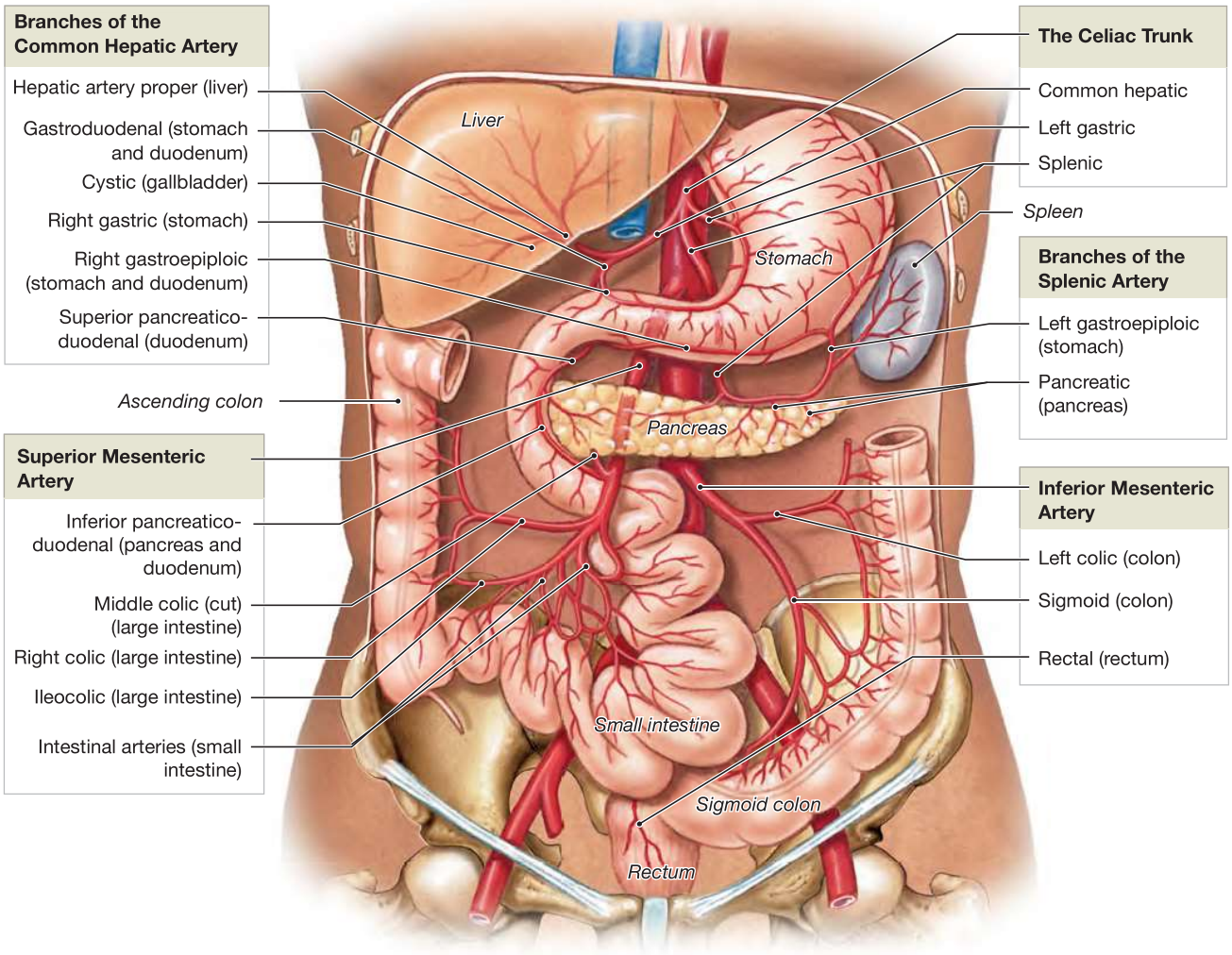
The abdominal aorta also gives rise to five paired arteries (**Figure 21–25**):

1. The **inferior phrenic arteries** supply the inferior surface of the diaphragm and the inferior portion of the esophagus.
2. The **adrenal arteries** originate on either side of the aorta near the base of the superior mesenteric artery. Each adrenal artery supplies one adrenal gland, which caps the superior part of a kidney.
3. The short (about 7.5 cm) **renal arteries** arise along the posterolateral surface of the abdominal aorta, about 2.5 cm (1 in.) inferior to the superior mesenteric artery. They travel posterior to the peritoneal lining to reach the adrenal glands and kidneys. We consider the branches of the renal arteries in Chapter 26.
4. The **gonadal** (gō-NAD-al) **arteries** originate between the superior and inferior mesenteric arteries. In males, they are called *testicular arteries* and are long, thin arteries that supply blood to the testes and scrotum. In females, they are termed *ovarian arteries* and supply blood to the ovaries, uterine tubes, and uterus. The distribution of gonadal vessels (both arteries and veins) differs by gender. We describe the differences in Chapter 28.
5. Small **lumbar arteries** arise on the posterior surface of the aorta. They supply the vertebrae, spinal cord, and abdominal wall.

Arteries of the Pelvis and Lower Limbs

Near the level of vertebra L_4, the terminal segment of the abdominal aorta divides to form a pair of elastic arteries—the

Figure 21–26 **Arteries Supplying the Abdominopelvic Organs.** *(See also Figure 24–24, p. 899.)* ATLAS: Plates 53a–e; 54c; 55

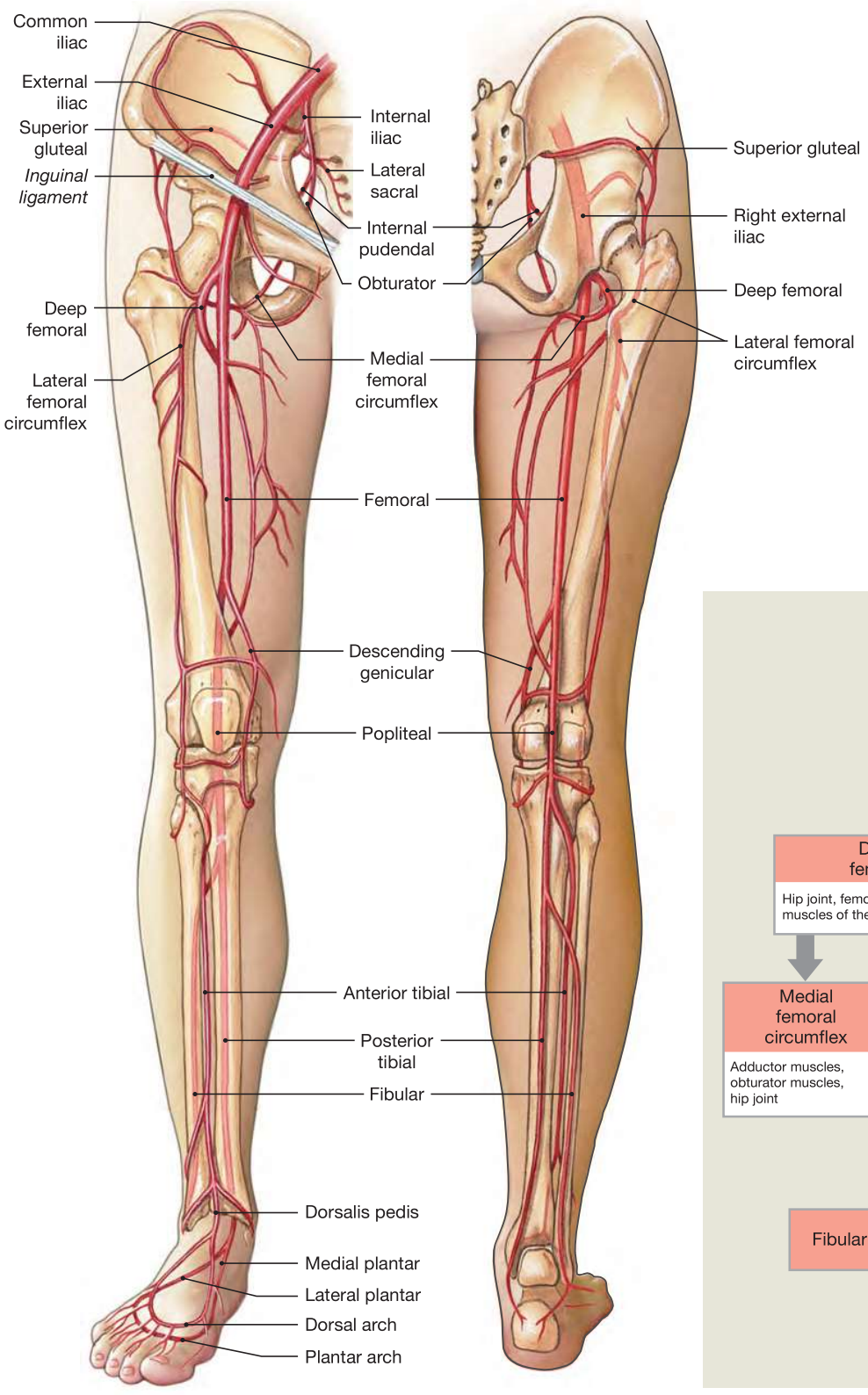


right and **left common iliac** (IL-ē-ak) **arteries**—plus the small **median sacral artery** (Figure 21–25). The common iliac arteries carry blood to the pelvis and lower limbs. They descend posterior to the cecum and sigmoid colon along the inner surface of the ilium. At the level of the lumbosacral joint, each common iliac divides to form an **internal iliac artery** and an **external iliac artery** (Figure 21–26). The internal iliac arteries enter the pelvic cavity to supply the urinary bladder, the internal and external walls of the pelvis, the external genitalia, the medial side of the thigh, and, in females, the uterus and vagina. The major branches of the internal iliac artery are the *gluteal*, *internal pudendal*, *obturator*, and *lateral sacral arteries*. The external iliac arteries supply blood to the lower limbs. They are much larger in diameter than the internal iliac arteries.

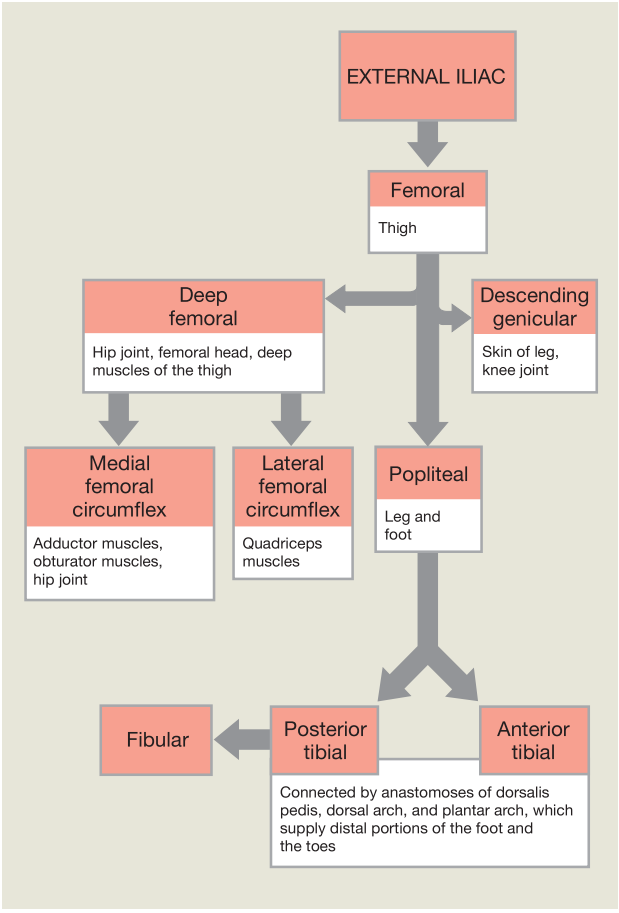
Arteries of the Thigh and Leg. Each external iliac artery crosses the surface of an iliopsoas muscle and penetrates the abdominal wall midway between the anterior superior iliac spine and the pubic symphysis on that side. It emerges on the anterior, medial surface of the thigh as the **femoral artery** (Figure 21–27a,b). Roughly 5 cm (2 in.) distal to the emergence of the femoral artery, the **deep femoral artery** branches off its lateral surface. The deep femoral artery supplies blood to the ventral and lateral regions of the skin and deep muscles of the thigh. It gives rise to the *femoral circumflex arteries.*

The femoral artery continues inferiorly and posterior to the femur. As it approaches the knee, it gives rise to the *descending genicular artery*, which supplies the area around the knee. At the popliteal fossa, posterior to the knee joint, the femoral artery becomes the **popliteal** (pop-LIT-ē-al) **artery,** which then branches to form the **posterior** and **anterior tibial arteries.** The posterior tibial artery gives rise to the **fibular artery,** or *peroneal* (*perone,* fibula) *artery*, and then continues inferiorly along the posterior surface of the tibia. The anterior tibial artery passes between the tibia and fibula. It emerges on the anterior surface of the tibia. As

Figure 21–27 Arteries of the Lower Limb. ATLAS: Plates 68c; 70b; 78b–g



a Anterior view **b** Posterior view



c A flowchart of blood flow to a lower limb

it descends toward the foot, the anterior tibial artery provides blood to the skin and muscles of the anterior portion of the leg.

Arteries of the Foot. At the ankle, the anterior tibial artery becomes the **dorsalis pedis artery.** It then branches repeatedly, supplying the ankle and dorsal portion of the foot (**Figure 21–27a,b**). **Figure 21–27c** charts the flow of blood from the external iliac artery to the lower limbs.

At the ankle, the posterior tibial artery divides to form the **medial** and **lateral plantar arteries.** They supply blood to the plantar surface of the foot. These arteries are connected to the dorsalis pedis artery through a pair of anastomoses. The arrangement produces a **dorsal arch** *(arcuate arch)* and a **plantar arch.** Small arteries branching off these arches supply the distal portions of the foot and the toes.

Systemic Veins

Veins collect blood from the tissues and organs of the body by means of an elaborate venous network that drains into the right atrium of the heart via the superior and inferior venae cavae (**Figure 21–28**). The branching pattern of peripheral veins is much more variable than is the branching pattern of arteries. We base the discussion that follows on the most common arrangement of veins. Complementary arteries and veins commonly run side by side. In many cases they have comparable names.

One significant difference between the arterial and venous systems involves the distribution of major veins in the neck and limbs. Arteries in these areas are deep beneath the skin, protected by bones and surrounding soft tissues. In contrast, the neck and limbs generally have two sets of peripheral veins, one superficial and the other deep. This dual venous drainage is important for controlling body temperature. In hot weather, venous blood flows through superficial veins, where heat can easily be lost. In cold weather, blood is routed to the deep veins to minimize heat loss.

21

The Superior Vena Cava

All the body's systemic veins (except the cardiac veins) ultimately drain into either the superior vena cava or the inferior vena cava. The **superior vena cava (SVC)** receives blood from the tissues and organs of the head, neck, chest, shoulders, and upper limbs.

Venous Return from the Cranium. Numerous veins drain the cerebral hemispheres. The *superficial cerebral veins* and small veins of the brain stem empty into a network of dural sinuses (**Figure 21–29a,b**). These sinuses include the *superior* and *inferior sagittal sinuses*, the *petrosal sinuses*, the *occipital sinus*, the *left* and *right transverse sinuses*, and the *straight sinus* (**Figure 21–29c**). The largest, the **superior sagittal sinus,** is in the falx cerebri (**Figure 14–4**, p. 455). Most of the *inferior cerebral veins* converge within the brain to form the **great cerebral vein.** It delivers blood from the interior of the cerebral hemispheres and the choroid plexus to the **straight sinus.** Other cerebral veins drain into the **cavernous sinus** with numerous small veins from the orbit. Blood from the cavernous sinus reaches the internal jugular vein through the petrosal sinuses.

The venous sinuses converge within the dura mater in the region of the lambdoid suture. The left and right transverse sinuses begin at the confluence of the occipital, sagittal, and straight sinuses. Each transverse sinus drains into a **sigmoid sinus,** which penetrates the jugular foramen and leaves the skull as the **internal jugular vein.** It descends parallel to the common carotid artery in the neck (p. 741).

Vertebral veins drain the cervical spinal cord and the posterior surface of the skull. These vessels descend within the transverse foramina of the cervical vertebrae, along with the vertebral arteries. The vertebral veins empty into the *brachiocephalic veins* of the chest (discussed later in the chapter).

Superficial Veins of the Head and Neck. The superficial veins of the head converge to form the **temporal, facial,** and **maxillary veins** (**Figure 21–29c**). The temporal vein and the maxillary vein drain into the **external jugular vein.** The facial vein drains into the internal jugular vein. A broad anastomosis between the external and internal jugular veins at the angle of the mandible provides dual venous drainage of the face, scalp, and cranium. The external jugular vein descends toward the chest just deep to the skin on the anterior surface of the sternocleidomastoid muscle. Posterior to the clavicle, the external jugular vein empties into the *subclavian vein*. In healthy individuals, the external jugular vein is easily palpable. A *jugular venous pulse (JVP)* is sometimes detectable at the base of the neck.

Venous Return from the Upper Limbs. The **digital veins** empty into the **superficial** and **deep palmar veins** of the hand, which interconnect to form the **palmar venous arches** (**Figure 21–30**). The superficial arch empties into the **cephalic vein,** which ascends along the radial side of the forearm; the **median antebrachial vein;** and the **basilic vein,** which ascends on the ulnar side. Anterior to the elbow is the superficial **median cubital vein,** which passes from the cephalic vein, medially and at an oblique angle, to connect to the basilic vein. (Venous blood samples are typically collected from the median cubital.) From the elbow, the basilic vein passes superiorly along the medial surface of the biceps brachii muscle.

The deep palmar veins drain into the **radial vein** and the **ulnar vein.** These veins fuse to form the **brachial vein,** running parallel to the brachial artery. As the brachial vein continues toward the trunk, it merges with the basilic vein and becomes the **axillary vein,** which enters the axilla.

Formation of the Superior Vena Cava. The cephalic vein joins the axillary vein on the lateral surface of the first rib, forming the **subclavian vein,** which continues into the chest.

Figure 21–28 An Overview of the Major Systemic Veins.

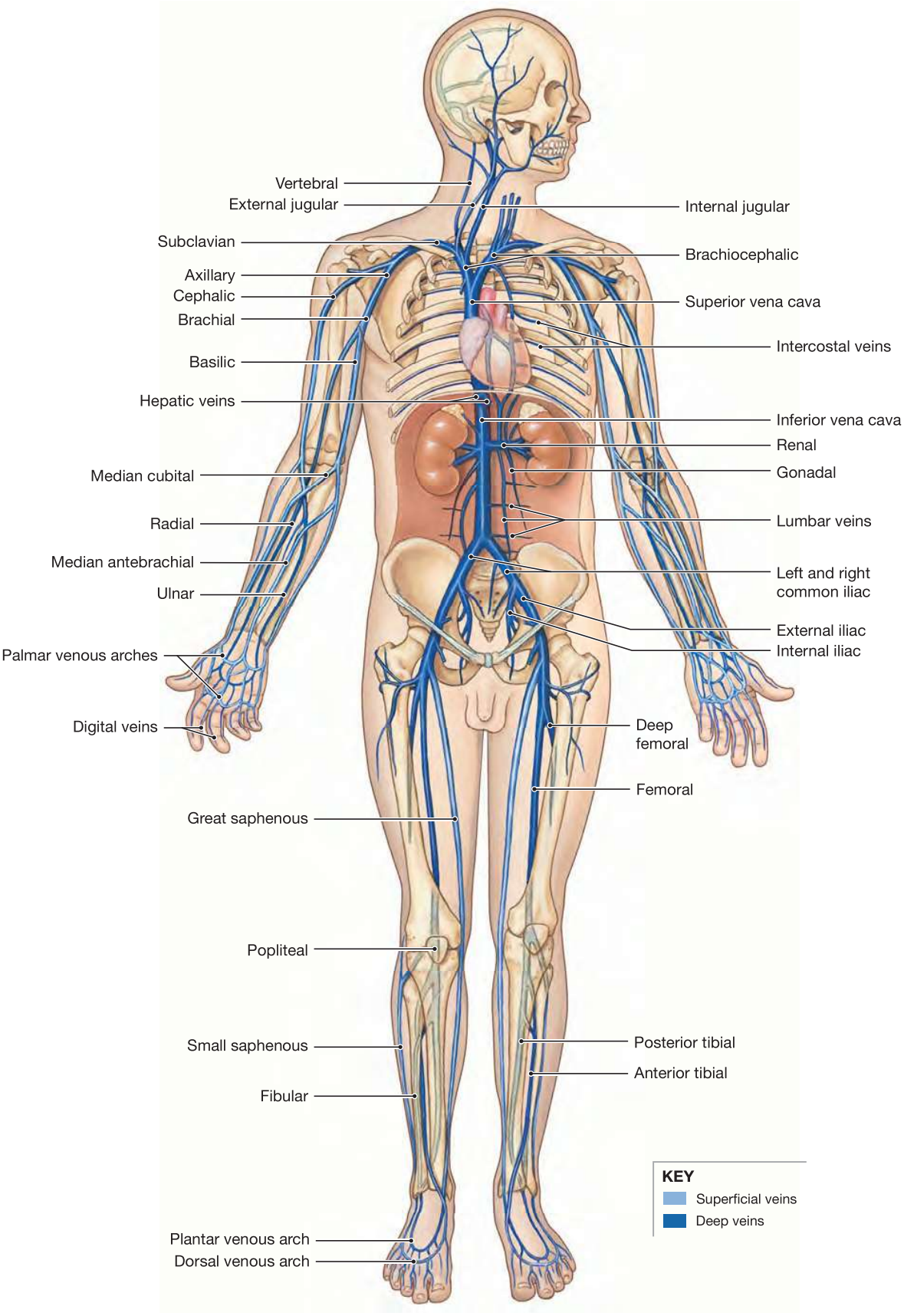
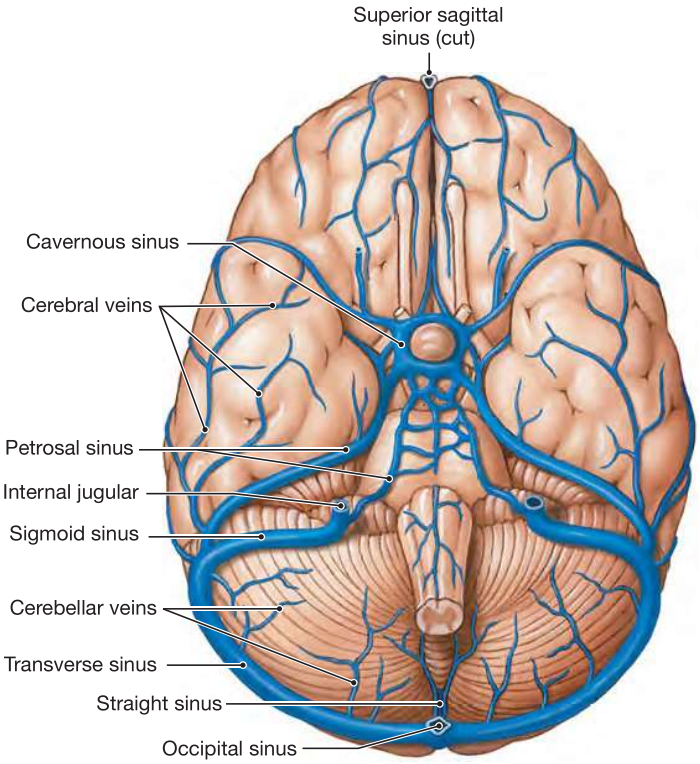
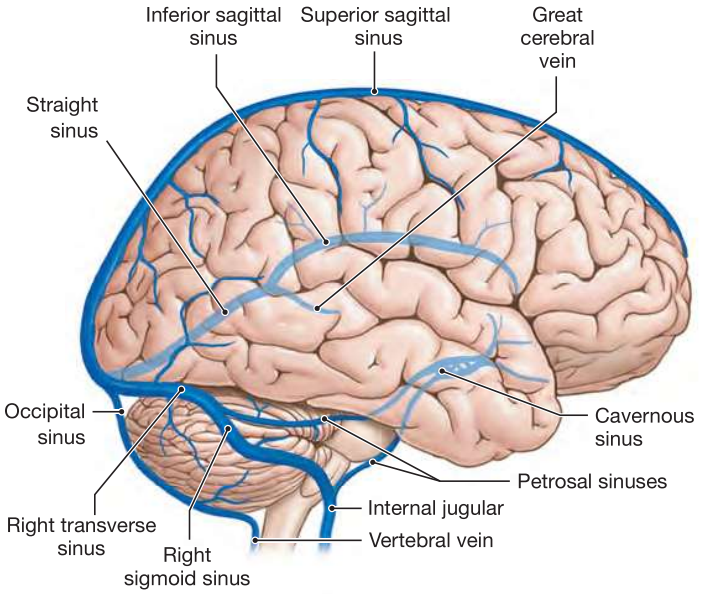


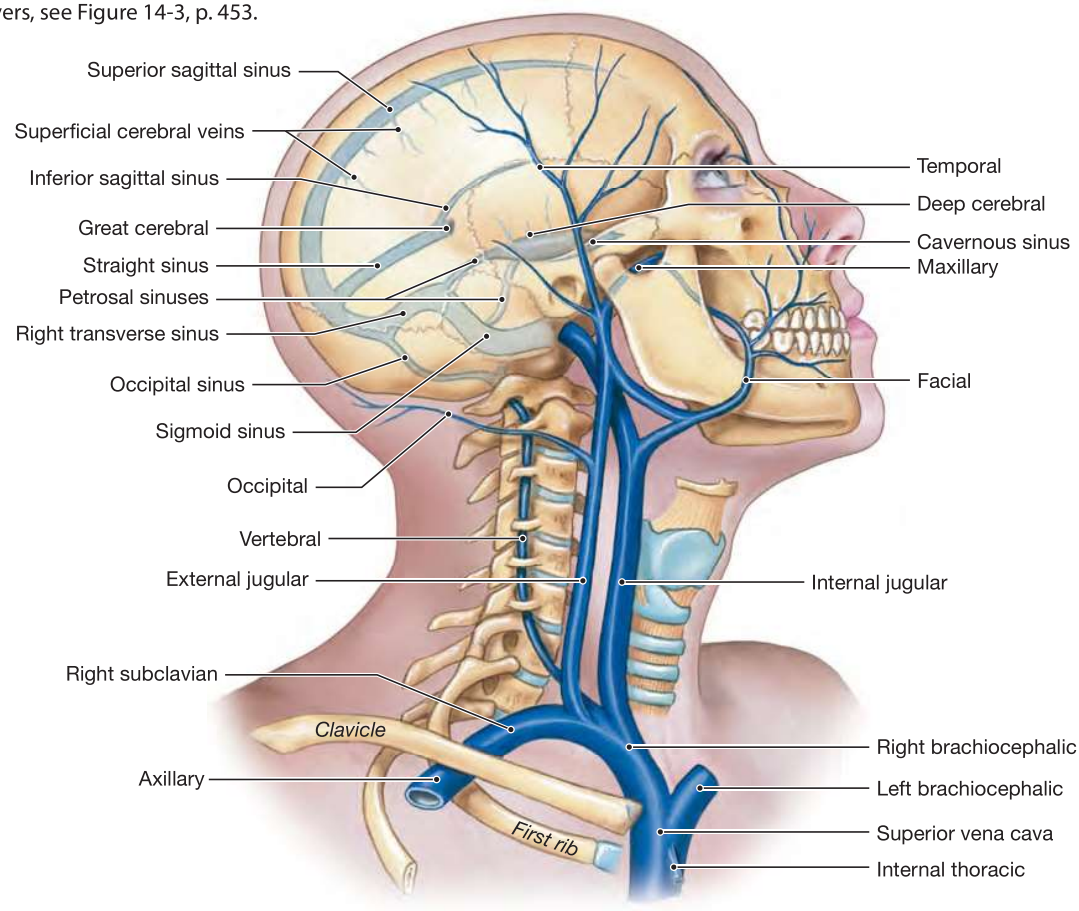
Figure 21–29 Major Veins of the Head, Neck, and Brain. ATLAS: Plates 3c,d; 18a–c



a An inferior view of the brain, showing the venous distribution. For the relationship of these veins to meningeal layers, see Figure 14-3, p. 453.

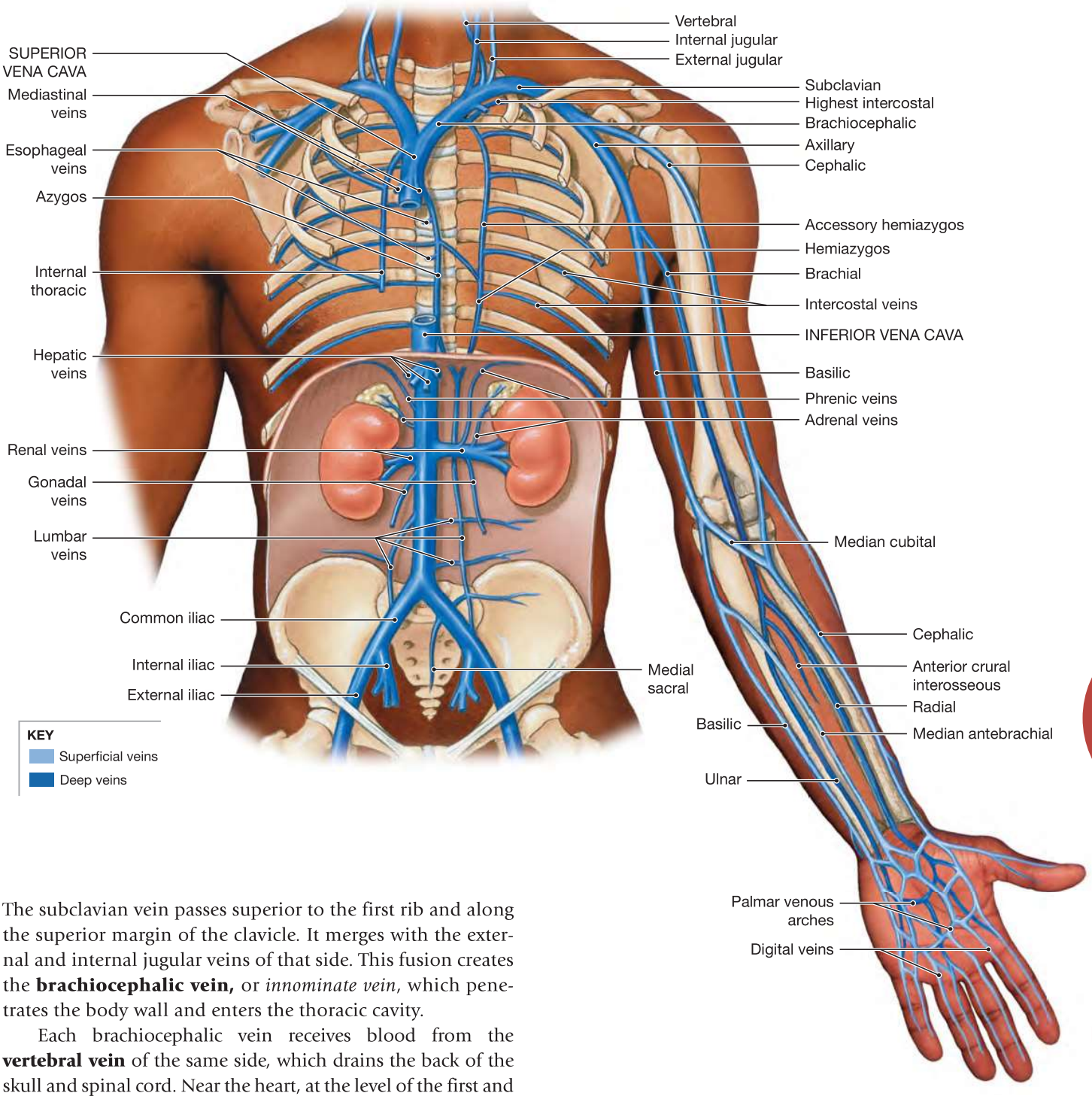


b A lateral view of the brain showing the venous distribution.



c Veins draining the brain and the superficial and deep portions of the head and neck.

Figure 21–30 The Venous Drainage of the Abdomen and Chest. ATLAS: Plates 27c; 29c; 47b,d; 61a; 62a,b



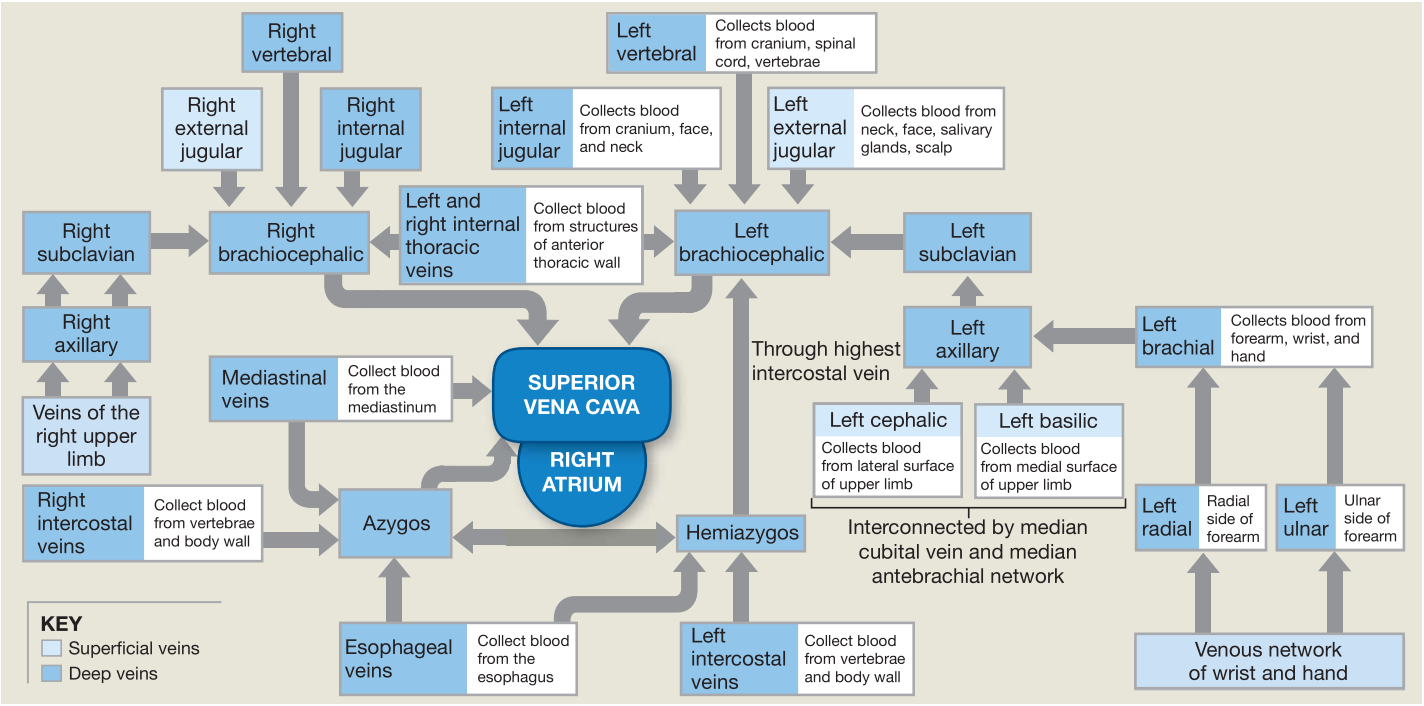
The subclavian vein passes superior to the first rib and along the superior margin of the clavicle. It merges with the external and internal jugular veins of that side. This fusion creates the **brachiocephalic vein,** or *innominate vein*, which penetrates the body wall and enters the thoracic cavity.

Each brachiocephalic vein receives blood from the **vertebral vein** of the same side, which drains the back of the skull and spinal cord. Near the heart, at the level of the first and second ribs, the left and right brachiocephalic veins join, creating the superior vena cava. Close to the point of fusion, the **internal thoracic vein** empties into the brachiocephalic vein.

The **azygos** (AZ-ī-gos) **vein** is the major tributary of the superior vena cava. This vein ascends from the lumbar region over the right side of the vertebral column to enter the thoracic cavity through the diaphragm. The azygos vein joins the superior vena cava at the level of vertebra T_2. On the left side, the azygos receives blood from the smaller **hemiazygos vein,** which in many people also drains into the left brachiocephalic vein through the *highest intercostal vein*.

The azygos and hemiazygos veins are the chief collecting vessels of the thorax. They receive blood from (1) **intercostal**

Figure 21–31 Flowcharts of Circulation to the Superior and Inferior Venae Cavae.



a Tributaries of the superior vena cava

veins, which in turn receive blood from the chest muscles; (2) **esophageal veins,** which drain blood from the inferior portion of the esophagus; and (3) smaller veins draining other mediastinal structures.

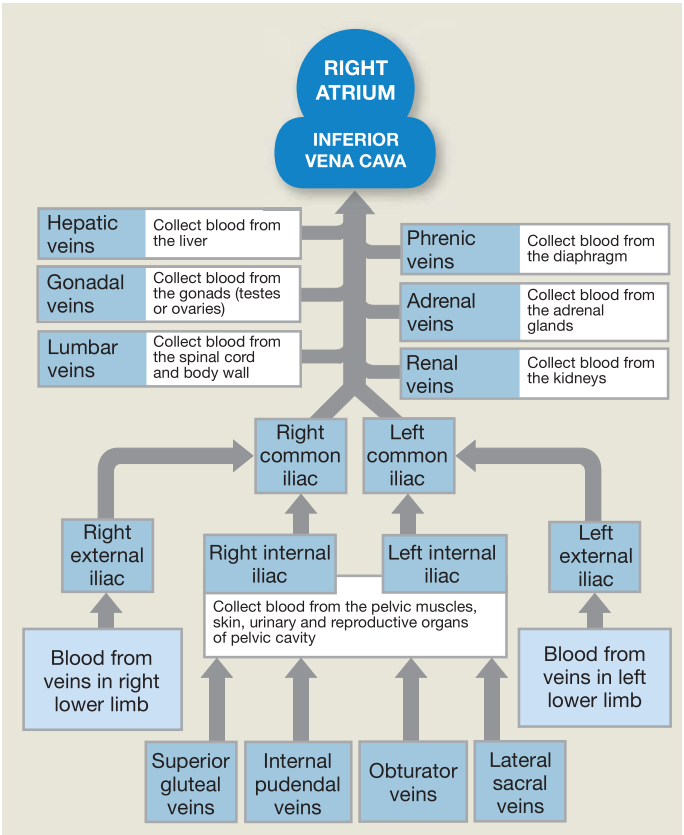
Figure 21–31a diagrams the venous tributaries of the superior vena cava.

The Inferior Vena Cava

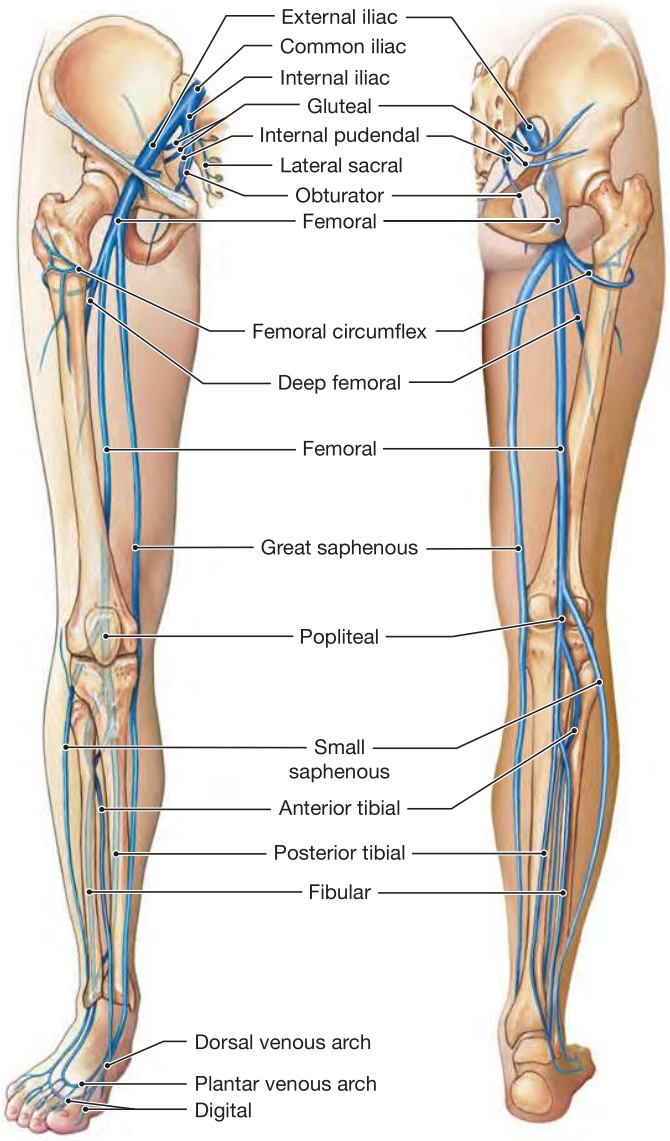
The **inferior vena cava (IVC)** collects most of the venous blood from organs inferior to the diaphragm. (A small amount reaches the superior vena cava via the azygos and hemiazygos veins.)

Veins Draining the Lower Limbs. Blood leaving capillaries in the sole of each foot collects into a network of **plantar veins,** which supply the **plantar venous arch** (**Figure 21–32a**). The plantar network sends blood to the deep veins of the leg: the **anterior tibial vein,** the **posterior tibial vein,** and the **fibular vein.** The **dorsal venous arch** collects blood from capillaries on the superior surface of the foot and the **digital veins** of the toes. The plantar arch and the dorsal arch are extensively interconnected, and the path of blood flow can easily shift from superficial to deep veins.

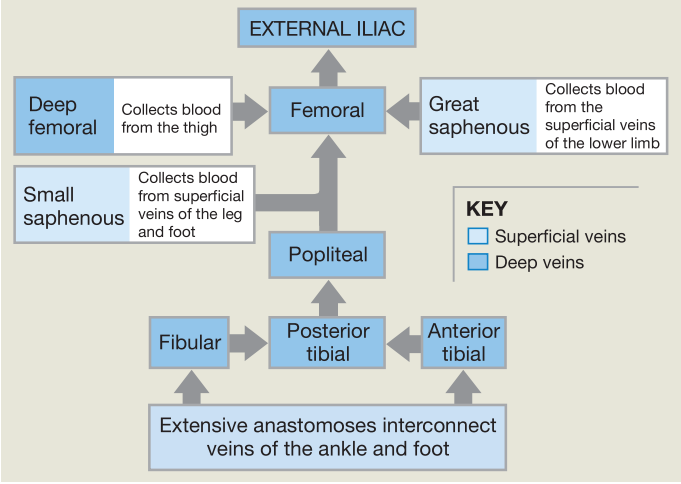
The dorsal venous arch is drained by two superficial veins: the **great saphenous** (sa-FĒ-nus; *saphenes,* prominent) **vein**



b Tributaries of the inferior vena cava



a An anterior view **b** A posterior view



c A flowchart of venous circulation from a lower limb

Figure 21–32 Venous Drainage from the Lower Limb. ATLAS: Plates 70b; 74; 78a–g

and the **small saphenous vein.** The great saphenous vein ascends along the medial aspect of the leg and thigh, draining into the *femoral vein* near the hip joint. The small saphenous vein arises from the dorsal venous arch and ascends along the posterior and lateral aspect of the calf. This vein then enters the popliteal fossa, where it meets the **popliteal vein,** formed by the union of the fibular and both tibial veins (**Figure 21–32b**). The popliteal vein is easily palpated in the popliteal fossa adjacent to the adductor magnus muscle. At the femur, the popliteal vein becomes the **femoral vein,** which ascends along the thigh, next to the femoral artery. Immediately before penetrating the abdominal wall, the femoral vein receives blood from (1) the great saphenous vein; (2) the **deep femoral vein,** which collects blood from deeper structures in the thigh; and (3) the **femoral circumflex vein,** which drains the region around the neck and head of the femur. The femoral vein penetrates the body wall and emerges in the pelvic cavity as the **external iliac vein.** **Figure 21–32c** charts the flow of venous blood in the lower limb.

Veins Draining the Pelvis. The external iliac veins receive blood from the lower limbs, the pelvis, and the lower abdomen. As the left and right external iliac veins cross the inner surface of the ilium, they are joined by the **internal iliac veins,** which drain the pelvic organs (**Figure 21–31**). The internal iliac veins are formed by the fusion of the *gluteal, internal pudendal, obturator,* and *lateral sacral veins* (**Figure 21–32a**).

The union of external and internal iliac veins forms the **common iliac vein.** Its right and left branches ascend at an oblique angle. The left common iliac vein receives blood from the *median sacral vein,* which drains the area supplied by the middle sacral artery (**Figure 21–30**). Anterior to vertebra L_5, the common iliac veins unite to form the inferior vena cava.

Veins Draining the Abdomen. The inferior vena cava ascends posterior to the peritoneal cavity, parallel to the aorta. The abdominal portion of the inferior vena cava collects blood from six major veins (**Figures 21–30** and **21–31b**):

1. **Lumbar veins** drain the lumbar portion of the abdomen, including the spinal cord and body wall muscles. Superior branches of these veins are connected to the azygos vein (right side) and hemiazygos vein (left side), which empty into the superior vena cava.
2. **Gonadal** (*ovarian* or *testicular*) **veins** drain the ovaries or testes. The right gonadal vein empties into the inferior vena cava. The left gonadal vein generally drains into the left renal vein.

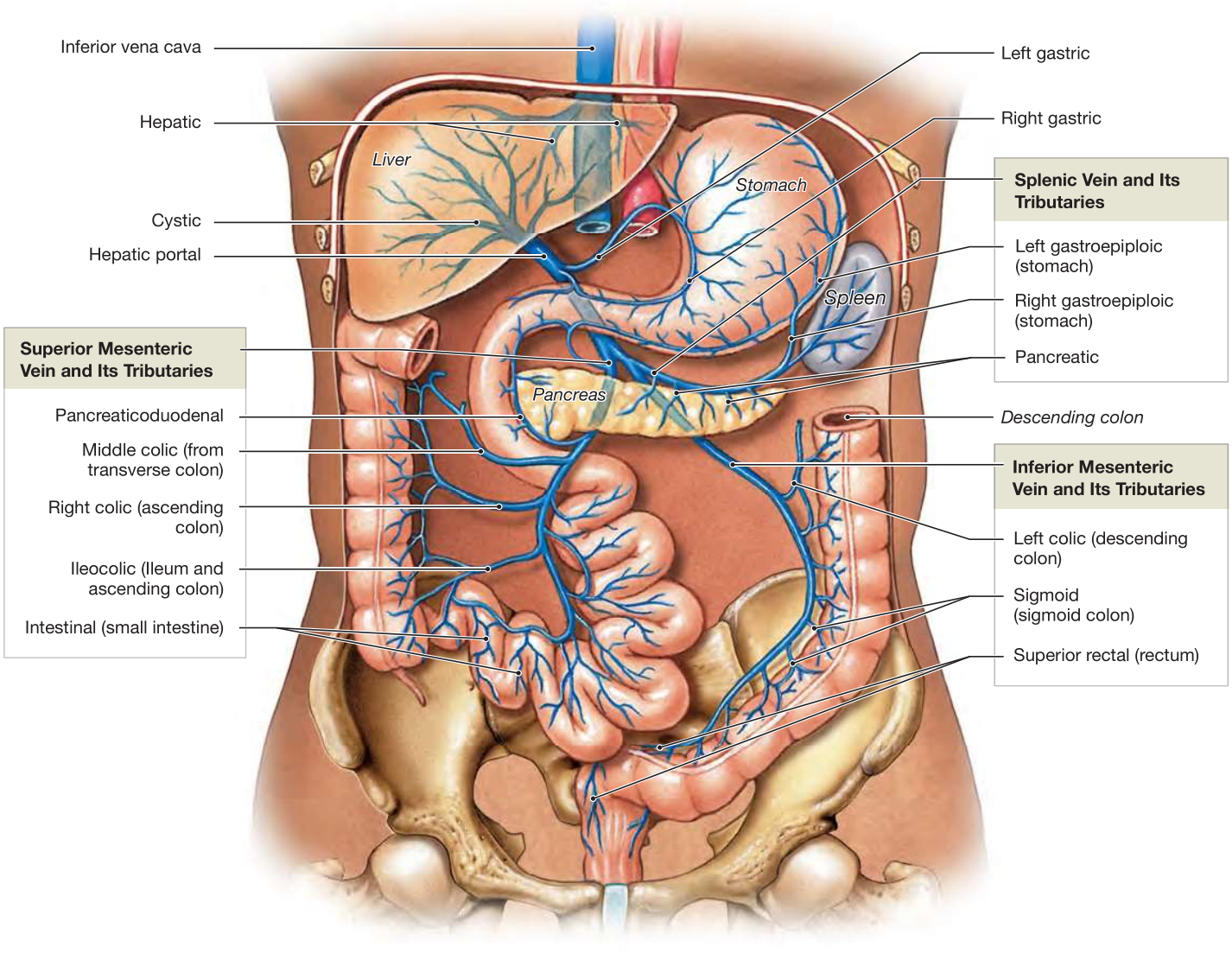
3. **Hepatic veins** from the liver empty into the inferior vena cava at the level of vertebra T_{10}.
4. **Renal veins,** the largest tributaries of the inferior vena cava, collect blood from the kidneys.
5. **Adrenal veins** drain the adrenal glands. In most people, only the right adrenal vein drains into the inferior vena cava. The left adrenal vein drains into the left renal vein.
6. **Phrenic veins** drain the diaphragm. Only the right phrenic vein drains into the inferior vena cava. The left drains into the left renal vein.

Figure 21–31b diagrams the tributaries of the inferior vena cava.

The Hepatic Portal System

The **hepatic portal system** begins in the capillaries of the digestive organs and ends in the liver sinusoids (**Figure 21–33**). (As you may recall from Chapter 18, a blood vessel connecting two capillary beds is called a *portal vessel*. The network is a *portal system*.) Blood flowing in the hepatic portal system is quite different from blood in other systemic veins, because it contains substances absorbed from the stomach and intestines. For example, levels of blood glucose and amino acids in the hepatic portal vein often exceed those found anywhere else in the cardiovascular system. The hepatic portal system delivers these and other absorbed compounds directly to the liver for storage, metabolic conversion, or excretion.

Figure 21–33 The Hepatic Portal System. *(See also Figure 24–24, p. 899.)* ATLAS: Plates 53b; 54a–c; 55; 57a,b



The largest vessel of the hepatic portal system is the **hepatic portal vein** (Figure 21–33). It delivers venous blood to the liver. It receives blood from three large veins draining organs within the peritoneal cavity:

- The **inferior mesenteric vein** collects blood from capillaries along the inferior portion of the large intestine. Its branches include the *left colic vein* and the *superior rectal veins*. They drain the descending colon, sigmoid colon, and rectum.
- The **splenic vein** is formed by the union of the inferior mesenteric vein and veins from the spleen, the lateral border of the stomach *(left gastroepiploic vein)*, and the pancreas *(pancreatic veins)*.
- The **superior mesenteric vein** collects blood from veins draining the stomach *(right gastroepiploic vein)*, the small intestine *(intestinal* and *pancreaticoduodenal veins)*, and two-thirds of the large intestine *(ileocolic, right colic,* and *middle colic veins)*.

The hepatic portal vein forms through the fusion of the superior mesenteric, inferior mesenteric, and splenic veins. The superior mesenteric vein normally contributes the greater volume of blood and most of the nutrients. As it proceeds, the hepatic portal vein receives blood from the left and right **gastric veins,** which drain the medial border of the stomach, and from the **cystic vein** of the gallbladder.

After passing through liver sinusoids, blood collects in the hepatic veins, which empty into the inferior vena cava.

The composition of the blood in the systemic circuit is relatively stable despite changes in diet and digestive activity. The reason for this stability is that blood from the intestines goes to the liver first, and the liver regulates the nutrient content of the blood before it enters the inferior vena cava.

Checkpoint

19. **A blockage of which branch from the aortic arch would interfere with blood flow to the left arm?**
20. **Why would compression of the common carotid arteries cause a person to lose consciousness?**
21. **Grace is in an automobile accident, and her celiac trunk is ruptured. Which organs will be affected most directly by this injury?**
22. **Whenever Tim gets angry, a large vein bulges in the lateral region of his neck. Which vein is this?**
23. **A thrombus that blocks the popliteal vein would interfere with blood flow in which other veins?**

See the blue Answers tab at the back of the book.

21-8 Modifications of fetal and maternal cardiovascular systems promote the exchange of materials, and independence is achieved at birth

The fetal cardiovascular system differs from the adult cardiovascular system because the fetus and the adult have different sources of respiratory and nutritional support. Most strikingly, the fetal lungs are collapsed and nonfunctional, and the digestive tract has nothing to digest. Instead, diffusion across the placenta provides for the respiratory and nutritional needs of the fetus.

Placental Blood Supply

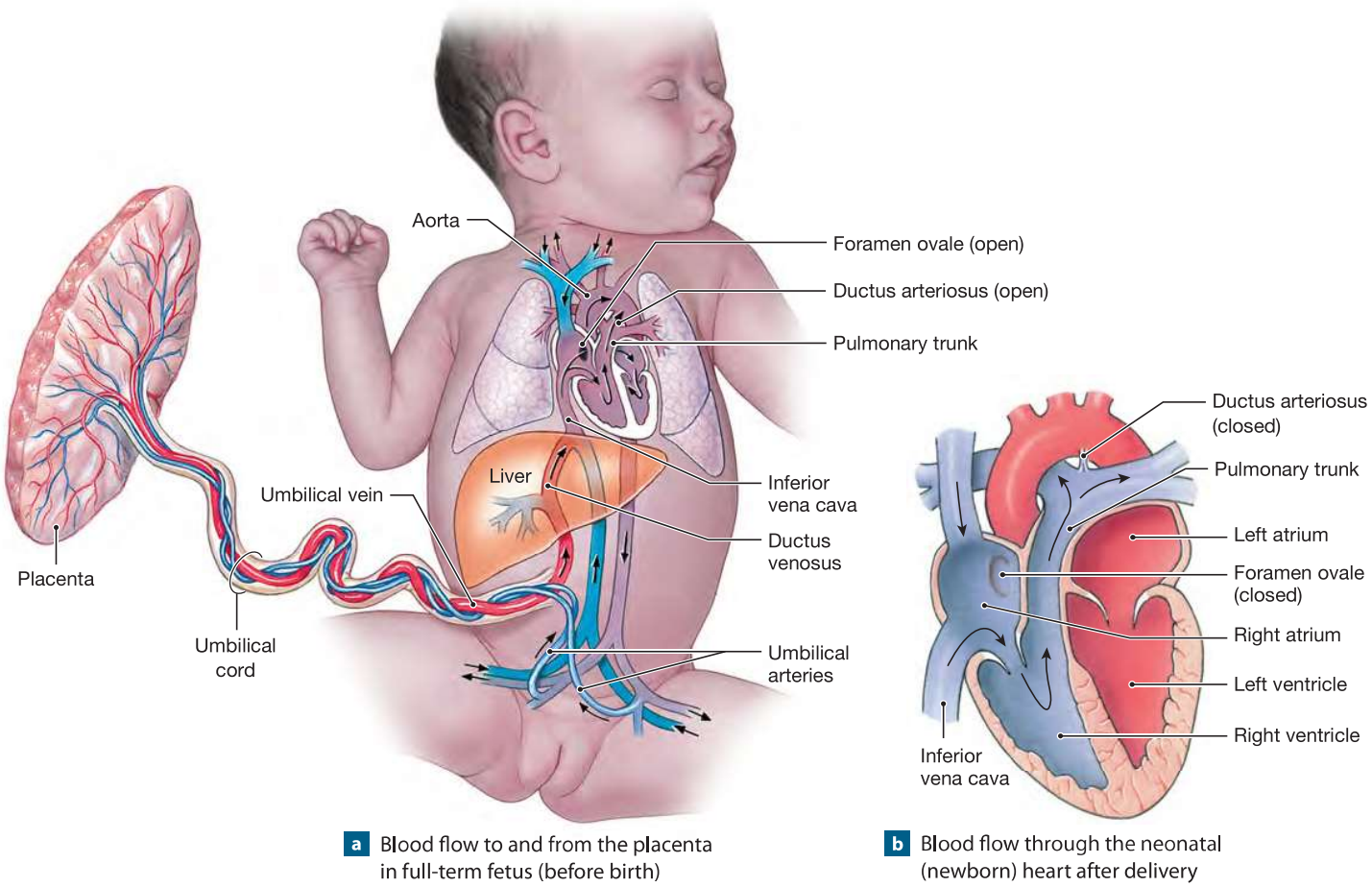
Fetal patterns of blood flow are diagrammed in Figure 21–34a. Blood flows to the placenta through a pair of **umbilical arteries.** They arise from the internal iliac arteries and enter the umbilical cord. Blood returns from the placenta in the single **umbilical vein,** bringing oxygen and nutrients to the developing fetus. The umbilical vein drains into the **ductus venosus,** a vascular connection to an intricate network of veins within the developing liver. The ductus venosus collects blood from the veins of the liver and from the umbilical vein, and empties into the inferior vena cava. When the placental connection is broken at birth, blood stops flowing in the umbilical vessels, and they soon degenerate. Remnants of these vessels persist throughout life as fibrous cords.

Fetal Circulation in the Heart and Great Vessels

One of the most interesting aspects of cardiovascular development reflects the differences between the life of an embryo or fetus and that of an infant. Throughout embryonic and fetal life, the lungs are collapsed. Yet after delivery, the newborn infant must extract oxygen from inspired air rather than across the placenta. ATLAS: Embryology Summary 16: The Development of the Cardiovascular System

The interatrial and interventricular septa develop early in fetal life, but the interatrial partition remains functionally incomplete until birth. The **foramen ovale,** or *interatrial opening,* is associated with a long flap that acts as a valve. Blood can flow freely from the right atrium to the left atrium, but any backflow closes the valve and isolates the two chambers from one another. Thus, blood entering the heart at the right atrium can bypass the pulmonary circuit. A second short-circuit exists

Figure 21–34 Fetal Circulation.



a Blood flow to and from the placenta in full-term fetus (before birth)

b Blood flow through the neonatal (newborn) heart after delivery

between the pulmonary and aortic trunks. This connection, the **ductus arteriosus,** consists of a short, muscular vessel.

With the lungs collapsed, the capillaries are compressed and little blood flows through the lungs. During diastole, blood enters the right atrium and flows into the right ventricle, but it also passes into the left atrium through the foramen ovale. About 25 percent of the blood arriving at the right atrium bypasses the pulmonary circuit in this way. In addition, more than 90 percent of the blood leaving the right ventricle passes through the ductus arteriosus and enters the systemic circuit rather than continuing to the lungs.

Cardiovascular Changes at Birth

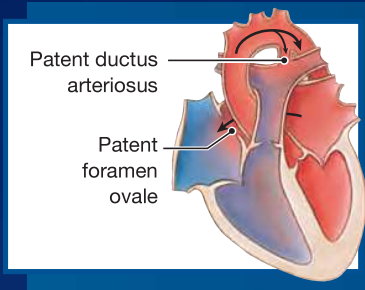
At birth, dramatic changes take place. When an infant takes the first breath, the lungs expand, and so do the pulmonary vessels. The resistance in the pulmonary circuit declines suddenly, and blood rushes into the pulmonary vessels. Within a few seconds, rising O_2 levels stimulate the constriction of the ductus arteriosus, isolating the pulmonary and aortic trunks from one another. As pressures rise in the left atrium, the valvular flap closes the foramen ovale. In adults, the interatrial septum bears the *fossa ovalis*, a shallow depression that marks the site of the foramen ovale (**Figure 20–6a,c**, p. 676). The remnants of the ductus arteriosus persist throughout life as the *ligamentum arteriosum*, a fibrous cord.

If the proper cardiovascular changes do not take place at birth or shortly after, problems eventually develop. Their severity depends on which connection remains open and on the size of the opening. Treatment may involve surgery to close the foramen ovale, the ductus arteriosus, or both. Other congenital heart defects result from abnormal cardiac development or inappropriate connections between the heart and major arteries and veins. **Spotlight Figure 21–35** focuses on congenital heart problems.

Spotlight

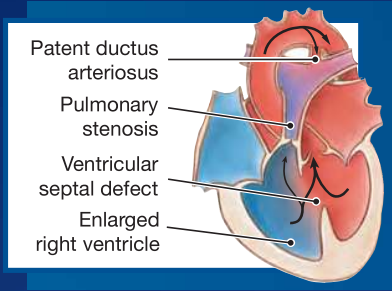
Figure 21–35
Congenital Heart Problems

Although minor individual variations in the vascular network are quite common, congenital heart problems serious enough to threaten homeostasis are relatively rare.



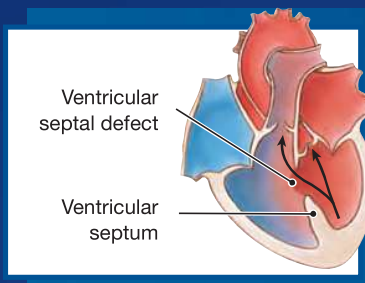
Patent Foramen Ovale and Patent Ductus Arteriosus

If the foramen ovale remains open, or *patent*, blood recirculates through the pulmonary circuit instead of entering the left ventricle. The movement, driven by the relatively high systemic pressure, is called a "left-to-right shunt." Arterial oxygen content is normal, but the left ventricle must work much harder than usual to provide adequate blood flow through the systemic circuit. Hence, pressures rise in the pulmonary circuit. If the pulmonary pressures rise enough, they may force blood into the systemic circuit through the ductus arteriosus. This condition—a patent ductus arteriosus—creates a "right-to-left shunt." Because the circulating blood is not adequately oxygenated, it develops a deep red color. The skin then develops the blue tones typical of cyanosis and the infant is known as a "blue baby."



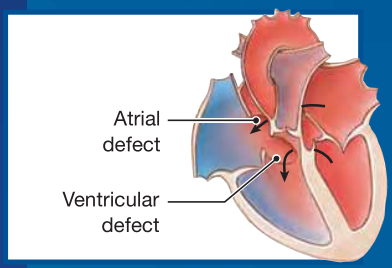
Tetralogy of Fallot

The tetralogy of Fallot (fa-LŌ) is a complex group of heart and circulatory defects that affect 0.10 percent of newborn infants. In this condition, (1) the pulmonary trunk is abnormally narrow (pulmonary stenosis), (2) the interventricular septum is incomplete, (3) the aorta originates where the interventricular septum normally ends, and (4) the right ventricle is enlarged and both ventricles thicken in response to the increased workload.



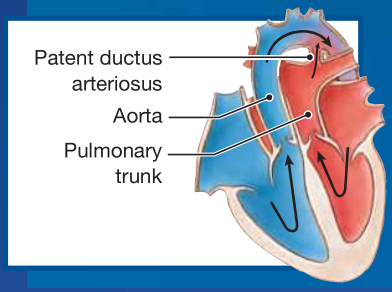
Ventricular Septal Defect

Ventricular septal defects are openings in the interventricular septum that separate the right and left ventricles. These defects are the most common congenital heart problems, affecting 0.12 percent of newborns. The opening between the two ventricles has an effect similar to a connection between the atria: When the more powerful left ventricle beats, it ejects blood into the right ventricle and pulmonary circuit.



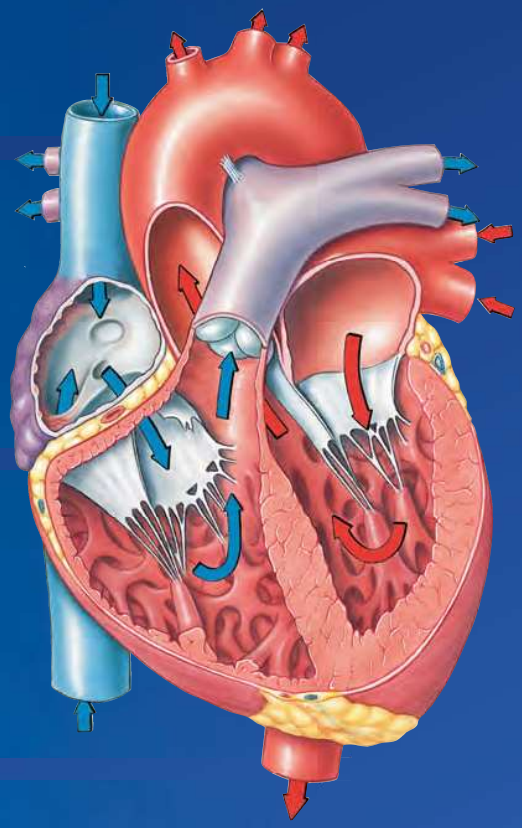
Atrioventricular Septal Defect

In an atrioventricular septal defect, both the atria and ventricles are incompletely separated. The results are quite variable, depending on the extent of the defect and the effects on the atrioventricular valves. This type of defect most commonly affects infants with Down's syndrome, a disorder caused by the presence of an extra copy of chromosome 21.



Transposition of the Great Vessels

In the transposition of great vessels, the aorta is connected to the right ventricle instead of to the left ventricle, and the pulmonary artery is connected to the left ventricle instead of the right ventricle. This malformation affects 0.05 percent of newborn infants.



Normal Heart Structure
Most heart problems reflect deviations from the normal formation of the heart and its connections to the great vessels.

Checkpoint

24. **Name the three vessels that carry blood to and from the placenta.**
25. **A blood sample taken from the umbilical cord contains high levels of oxygen and nutrients, and low levels of carbon dioxide and waste products. Is this sample from an umbilical artery or from the umbilical vein? Explain.**
26. **Name the structures in the fetal circulation that cease to function at birth. What becomes of these structures?**

See the blue Answers tab at the back of the book.

21-9 Aging affects the blood, heart, and blood vessels

The capabilities of the cardiovascular system gradually decline. As you age, your cardiovascular system undergoes the following major changes:

- **Age-related changes in blood** may include (1) a decreased hematocrit; (2) constriction or blockage of peripheral veins by a stationary blood clot called a *thrombus*, which can become detached, pass through the heart, and become wedged in a small artery (commonly in the lungs), causing *pulmonary embolism*; and (3) pooling of blood in the veins of the legs because valves are not working effectively.
- **Age-related changes in the heart** include (1) a reduction in maximum cardiac output, (2) changes in the activities of nodal and conducting cells, (3) a reduction in the elasticity of the cardiac (fibrous) skeleton, (4) progressive atherosclerosis that can restrict coronary circulation, and (5) replacement of damaged cardiac muscle cells by scar tissue.
- **Age-related changes in blood vessels** may be linked to arteriosclerosis: (1) The inelastic walls of arteries become less tolerant of sudden pressure increases, which can lead to an *aneurysm*, whose rupture may (depending on the vessel) cause a stroke, myocardial infarction, or massive blood loss; (2) calcium salts can be deposited on weakened vascular walls, increasing the risk of a stroke or myocardial infarction; and (3) thrombi can form at atherosclerotic plaques.

The cardiovascular system is both anatomically and functional linked to all other systems. **Figure 21–36** shows the relationships between the cardiovascular system and the other body systems we have studied so far.

Checkpoint

27. **Identify components of the cardiovascular system that are affected by age.**
28. **Define thrombus.**
29. **Define aneurysm.**
30. **Describe what the cardiovascular system provides for all other body systems.**
31. **What is the relationship between the skeletal system and the cardiovascular system?**

See the blue Answers tab at the back of the book.

Related Clinical Terms

angiogram: An x-ray of a blood vessel that becomes visible due to a prior injection of dye into the subject's bloodstream.
carotid sinus massage: A procedure that involves rubbing the large part of the arterial wall at the point where the common carotid artery divides into its two main branches.
deep vein thrombosis (DVT): A blood clot in a major vein, usually in the legs. They often occur after extended periods of inactivity, such as long airplane flights. The clot can break free and travel as an embolus to the lungs, where it can cause respiratory distress or failure.
intermittent claudication: A limp that results from cramping leg pain that is typically caused by obstruction of the arteries.
normotensive: Having normal blood pressure.
orthostatic hypotension: A form of low blood pressure that occurs when you stand up from sitting or lying down. It can cause dizziness or a light-headed feeling.
phlebitis: Inflammation of a vein.
Raynaud's phenomenon: A condition resulting in the discoloration of the fingers and/or the toes when a person is subjected to changes in temperature or to emotional stress.
sclerotherapy: The treatment of varicose veins in which an irritant is injected to cause inflammation, coagulation of blood, and a narrowing of the blood vessel wall.
sounds of Korotkoff: Distinctive sounds, caused by turbulent arterial blood flow, heard through the stethoscope while measuring blood pressure.
sphygmomanometer: A device that measures blood pressure using an inflatable cuff placed around a limb.
syncope: A temporary loss of consciousness due to a sudden drop in blood pressure.
thrill: A vibration felt in a blood vessel that usually occurs due to abnormal blood flow. It is also often noticed at the fistula of a hemodialysis patient.
thrombophlebitis: An inflammation in a vein associated with the formation of a thrombus (clot).
vascular murmur: Periodic abnormal sounds heard upon auscultation that are produced as a result of turbulent blood flow.
white coat hypertension: A short-term increase in blood pressure triggered by the sight of medical personnel in white coats or other medical attire.

SYSTEM INTEGRATOR

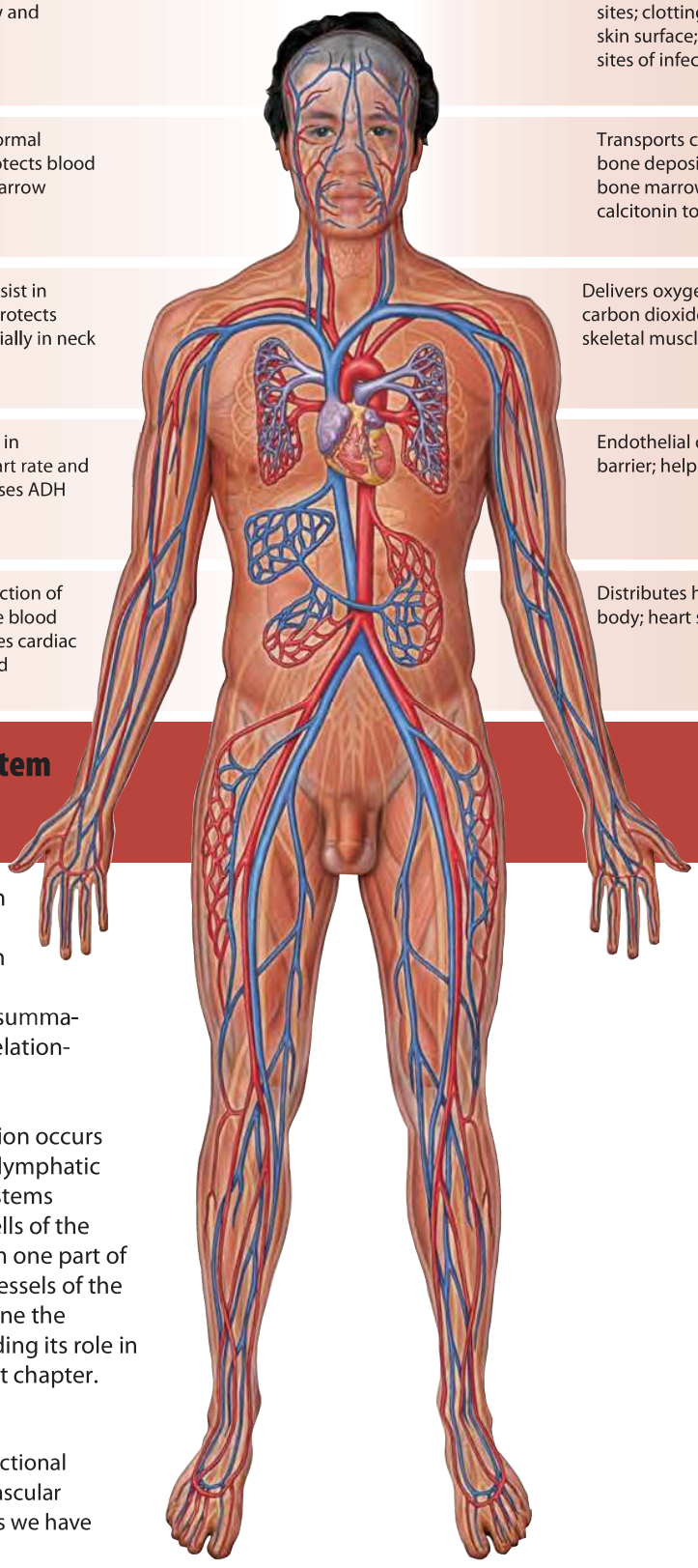
Body System → Cardiovascular System

Integumentary	Stimulation of mast cells produces localized changes in blood flow and capillary permeability
Skeletal	Provides calcium needed for normal cardiac muscle contraction; protects blood cells developing in red bone marrow
Muscular	Skeletal muscle contractions assist in moving blood through veins; protects superficial blood vessels, especially in neck and limbs
Nervous	Controls patterns of circulation in peripheral tissues; modifies heart rate and regulates blood pressure; releases ADH
Endocrine	Erythropoietin regulates production of RBCs; several hormones elevate blood pressure; epinephrine stimulates cardiac muscle, elevating heart rate and contractile force

Cardiovascular System → Body System

Integumentary Page 165	Delivers immune system cells to injury sites; clotting response seals breaks in skin surface; carries away toxins from sites of infection; provides heat
Skeletal Page 275	Transports calcium and phosphate for bone deposition; delivers EPO to red bone marrow, parathyroid hormone, and calcitonin to osteoblasts and osteoclasts
Muscular Page 369	Delivers oxygen and nutrients, removes carbon dioxide, lactic acid, and heat during skeletal muscle activity
Nervous Page 543	Endothelial cells maintain blood–brain barrier; helps generate CSF
Endocrine Page 632	Distributes hormones throughout the body; heart secretes ANP and BNP

The CARDIOVASCULAR System



The section on vessel distribution demonstrated the extent of the anatomical connections between the cardiovascular system and other organ systems. This figure summarizes some of the physiological relationships involved.

The most extensive communication occurs between the cardiovascular and lymphatic systems. Not only are the two systems physically interconnected, but cells of the lymphatic system also move from one part of the body to another within the vessels of the cardiovascular system. We examine the lymphatic system in detail, including its role in the immune response, in the next chapter.

Figure 21–36 diagrams the functional relationships between the cardiovascular system and the other body systems we have studied so far.

Lymphatic Page 807
Respiratory Page 857
Digestive Page 910
Urinary Page 992
Reproductive Page 1072

Chapter Review

Study Outline

21-1 Arteries, arterioles, capillaries, venules, and veins differ in size, structure, and functional properties p. 708

1. Blood flows through a network of arteries, veins, and capillaries. All chemical and gaseous exchange between blood and interstitial fluid takes place across capillary walls.
2. **Arteries** and **veins** form an internal distribution system through which the heart propels blood. Arteries branch repeatedly, decreasing in size until they become **arterioles.** From the arterioles, blood enters **capillary** networks. Blood flowing from the capillaries enters small **venules** before entering larger veins.
3. The walls of arteries and veins contain three layers: the innermost **tunica intima,** the **tunica media,** and the outermost **tunica externa.** *(Figure 21–1)*
4. In general, the walls of arteries are thicker than those of veins. Arteries constrict when blood pressure does not distend them, but veins constrict very little. The endothelial lining cannot contract, so when constriction occurs, the lining of an artery is thrown into folds. *(Figure 21–1)*
5. The arterial system includes the large **elastic arteries,** medium-sized **muscular arteries,** and smaller arterioles. As blood proceeds toward the capillaries, the number of vessels increases, but the diameters of the individual vessels decrease and the walls become thinner. *(Figure 21–2)*
6. **Atherosclerosis,** a type of **arteriosclerosis,** is associated with changes in the endothelial lining of arteries. Fatty masses of tissue called **plaques** typically develop during atherosclerosis. *(Figure 21–3)*
7. Capillaries are the only blood vessels whose walls are thin enough to permit an exchange between blood and interstitial fluid. Capillaries are **continuous** or **fenestrated. Sinusoids** have fenestrated walls and form elaborate networks that allow very slow blood flow. Sinusoids are located in the liver and in various endocrine organs. *(Figure 21–4)*
8. Capillaries form interconnected networks called **capillary beds (capillary plexuses).** A band of smooth muscle, the **precapillary sphincter,** adjusts the blood flow into each capillary. Blood flow in a capillary changes as **vasomotion** occurs. The entire capillary bed may be bypassed by blood flow through **arteriovenous anastomoses.** *(Figure 21–5)*
9. Venules collect blood from the capillaries and merge into **medium-sized veins** and then **large veins.** The arterial system is a high-pressure system; blood pressure in veins is much lower. **Valves** in veins prevent the backflow of blood. *(Figures 21–1, 21–2, 21–6)*
10. Peripheral **venoconstriction** helps maintain adequate blood volume in the arterial system after a hemorrhage. The **venous reserve** normally accounts for about 20 percent of total blood volume. *(Figure 21–7)*

21-2 Pressure and resistance determine blood flow and affect rates of capillary exchange p. 717

11. Cardiovascular regulation involves the manipulation of blood pressure and resistance to control the rates of blood flow and capillary exchange. *(Figure 21–8)*
12. Blood flows from an area of higher pressure to one of lower pressure, and blood flow is proportional to the pressure gradient. The *circulatory pressure* is the pressure gradient across the systemic circuit. It is reported as three values: arterial **blood pressure (BP), capillary hydrostatic pressure (CHP),** and **venous pressure.**
13. The **resistance (R)** determines the rate of blood flow through the systemic circuit. The major determinant of blood flow rate is the **peripheral resistance**—the resistance of the arterial system. Neural and hormonal control mechanisms regulate blood pressure and peripheral resistance.
14. **Vascular resistance** is the resistance of blood vessels. It is the largest component of peripheral resistance and depends on vessel length and vessel diameter. *(Figure 21–9)*
15. **Viscosity** and **turbulence** also contribute to peripheral resistance. *(Table 21–1)*
16. The high arterial pressures overcome peripheral resistance and maintain blood flow through peripheral tissues. Capillary pressures are normally low, and small changes in capillary pressure determine the rate of movement of fluid into or out of the bloodstream. Venous pressure, normally low, determines *venous return* and affects cardiac output and peripheral blood flow. *(Figures 21–10, 21–11; Table 21–1)*
17. Arterial blood pressure rises during ventricular systole and falls during ventricular diastole. The difference between these two blood pressures is the pulse pressure. Blood pressure is measured at the brachial artery with the use of a sphygmomanometer. *(Figures 21–10, 21–11)*
18. Valves, muscular compression, and the **respiratory pump** *(thoracoabdominal pump)* help the relatively low venous pressures propel blood toward the heart. *(Figures 21–6, 21–10)*
19. At the capillaries, blood pressure forces water and solutes out of the plasma, across capillary walls. Water moves out of the capillaries, through the peripheral tissues, and back to the bloodstream by way of the lymphatic system. Water movement across capillary walls is determined by the interplay between osmotic pressures and hydrostatic pressures. *(Figure 21–12)*
20. **Osmotic pressure (OP)** is a measure of the pressure that must be applied to prevent osmotic movement across a membrane. Osmotic water movement continues until either solute concentrations are equalized or the movement is prevented by an opposing hydrostatic pressure.
21. The rates of filtration and reabsorption gradually change as blood passes along the length of a capillary, as determined by the **net filtration pressure** (the difference between the net hydrostatic pressure and the net osmotic pressure). *(Figure 21–13)*

21-3 Cardiovascular regulatory mechanisms involve autoregulation, neural mechanisms, and endocrine responses p. 725

22. Homeostatic mechanisms ensure that **tissue perfusion** (blood flow) delivers adequate oxygen and nutrients.
23. Autoregulation, neural mechanisms, and endocrine mechanisms influence the coordinated regulation of cardiovascular function. Autoregulation involves local factors changing the pattern of blood flow within capillary beds in response to chemical changes in interstitial fluids. Neural mechanisms respond to changes in arterial pressure or blood

gas levels. Hormones can assist in short-term adjustments (changes in cardiac output and peripheral resistance) and long-term adjustments (changes in blood volume that affect cardiac output and gas transport). *(Figure 21–14)*

24. Peripheral resistance is adjusted at the tissues by local factors that result in the dilation or constriction of precapillary sphincters. *(Figure 21–5)*
25. **Cardiovascular (CV) centers** of the medulla oblongata are responsible for adjusting cardiac output and peripheral resistance to maintain adequate blood flow. The vasomotor center contains one group of neurons responsible for controlling vasoconstriction, and another group responsible for controlling vasodilation.
26. **Baroreceptor reflexes** monitor the degree of stretch within expandable organs. Baroreceptors are located in the **carotid sinuses,** the **aortic sinuses,** and the right atrium. *(Figure 21–15)*
27. **Chemoreceptor reflexes** respond to changes in the oxygen or CO_2 levels in the blood. They are triggered by sensory neurons located in the **carotid bodies** and the **aortic bodies.** *(Figure 21–16)*
28. The endocrine system provides short-term regulation of cardiac output and peripheral resistance with epinephrine and norepinephrine from the adrenal medullae. Hormones involved in the long-term regulation of blood pressure and volume are *antidiuretic hormone (ADH)*, *angiotensin II*, *erythropoietin (EPO)*, and *natriuretic peptides* (*ANP* and *BNP*). *(Figure 21–17)*

21-4 The cardiovascular system adapts to physiological stress and maintains a special vascular supply to the brain, heart, and lungs p. 733

29. During exercise, blood flow to skeletal muscles increases at the expense of blood flow to nonessential organs, and cardiac output rises. Cardiovascular performance improves with training. Athletes have larger stroke volumes, slower resting heart rates, and larger cardiac reserves than do nonathletes. *(Tables 21–2, 21–3)*
30. Blood loss lowers blood volume and venous return and decreases cardiac output. Compensatory mechanisms include an increase in cardiac output, mobilization of venous reserves, peripheral vasoconstriction, and the release of hormones that promote the retention of fluids and the manufacture of erythrocytes. *(Figure 21–18)*
31. The blood–brain barrier, the coronary circulation, and the circulation to alveolar capillaries in the lungs are examples of special circulations, in which cardiovascular dynamics and regulatory mechanisms differ from those in other tissues.

21-5 The pulmonary and systemic circuits of the cardiovascular system exhibit three general functional patterns p. 736

32. The peripheral distributions of arteries and veins are generally identical on both sides of the body, except near the heart. *(Figure 21–19)*

21-6 In the pulmonary circuit, deoxygenated blood enters the lungs in arteries, and oxygenated blood leaves the lungs via veins p. 737

33. The pulmonary circuit includes the pulmonary trunk, the **left** and **right pulmonary arteries,** and the **pulmonary veins,** which empty into the left atrium. *(Figure 21–20)*

21-7 The systemic circuit carries oxygenated blood from the left ventricle to tissues and organs other than the pulmonary exchange surfaces, and returns deoxygenated blood to the right atrium p. 738

34. The **ascending aorta** gives rise to the coronary circulation. The **aortic arch** communicates with the **descending aorta.** *(Figures 21–21 to 21–27)*
35. Three elastic arteries originate along the aortic arch: the **left common carotid artery,** the **left subclavian artery,** and the **brachiocephalic trunk.** *(Figures 21–22, 21–23, 21–24)*
36. The remaining major arteries of the body originate from the **descending aorta.** *(Figures 21–25, 21–26, 21–27)*
37. Arteries in the neck and limbs are deep beneath the skin; in contrast, there are generally two sets of peripheral veins, one superficial and one deep. This dual venous drainage is important for controlling body temperature. *(Figure 21–28)*
38. The **superior vena cava** receives blood from the head, neck, chest, shoulders, and arms. *(Figures 21–28 to 21–31)*
39. The **inferior vena cava** collects most of the venous blood from organs inferior to the diaphragm. *(Figures 21–30 to 21–32)*
40. The **hepatic portal system** directs blood from the other digestive organs to the liver before the blood returns to the heart. *(Figure 21–33)*

21-8 Modifications of fetal and maternal cardiovascular systems promote the exchange of materials, and independence is achieved at birth p. 755

41. Blood flows to the placenta in a pair of **umbilical arteries** and is drained by a single **umbilical vein.** *(Figure 21–34)*
42. The interatrial partition remains functionally incomplete until birth. The **foramen ovale** allows blood to flow freely from the right to the left atrium, and the **ductus arteriosus** short-circuits the pulmonary trunk.
43. The foramen ovale closes, leaving the fossa ovalis. The ductus arteriosus constricts, leaving the ligamentum arteriosum. *(Figure 21–34)*
44. Congenital cardiovascular problems generally reflect abnormalities of the heart or of interconnections between the heart and great vessels. *(Spotlight Figure 21–35)*

21-9 Aging affects the blood, heart, and blood vessels p. 758

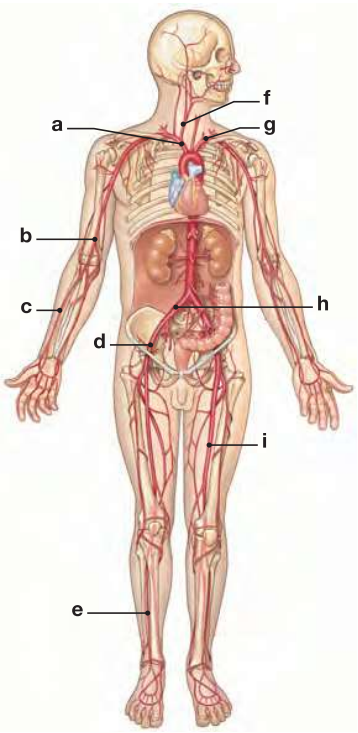
45. Age-related changes in the blood include (1) a decreased hematocrit, (2) constriction or blockage of peripheral veins by a *thrombus* (stationary blood clot), and (3) pooling of blood in the veins of the legs because valves are not working effectively.
46. Age-related changes in the heart include (1) a reduction in the maximum cardiac output, (2) changes in the activities of nodal and conducting cells, (3) a reduction in the elasticity of the fibrous skeleton, (4) progressive atherosclerosis that can restrict coronary circulation, and (5) the replacement of damaged cardiac muscle cells by scar tissue.
47. Age-related changes in blood vessels, commonly related to arteriosclerosis, include (1) a weakening in the walls of arteries, potentially leading to the formation of an *aneurysm;* (2) deposition of calcium salts on weakened vascular walls, increasing the risk of a stroke or myocardial infarction; and (3) the formation of a thrombus at atherosclerotic plaques.
48. The cardiovascular system is anatomically and functionally connected to all other body systems. *(Figure 21–36)*

Review Questions

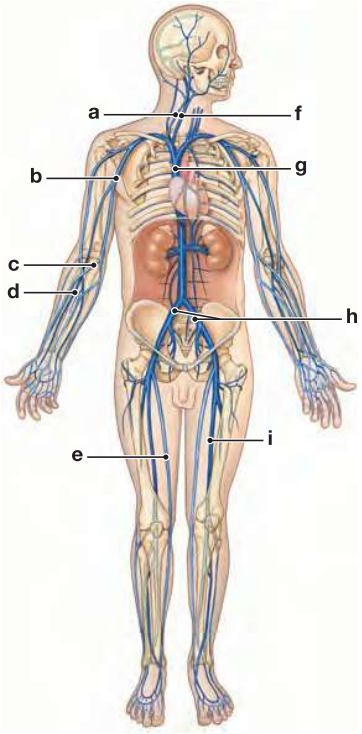
See the blue Answers tab at the back of the book.

LEVEL 1 Reviewing Facts and Terms

1. Identify the major arteries in the following diagram.
   (a) _____
   (b) _____
   (c) _____
   (d) _____
   (e) _____
   (f) _____
   (g) _____
   (h) _____
   (i) _____



2. The blood vessels that play the most important role in regulating blood pressure and blood flow to a tissue are the
   (a) arteries.
   (b) arterioles.
   (c) veins.
   (d) venules.
   (e) capillaries.
3. Cardiovascular function is regulated by all of the following *except*
   (a) local factors.
   (b) neural factors.
   (c) endocrine factors.
   (d) venous return.
   (e) conscious control.
4. Baroreceptors that function in the regulation of blood pressure are located in the
   (a) left ventricle.
   (b) brain stem.
   (c) carotid sinus.
   (d) common iliac artery.
   (e) pulmonary trunk.
5. The two-way exchange of substances between blood and body cells occurs only through
   (a) arterioles.
   (b) capillaries.
   (c) venules.
   (d) a, b, and c.
6. Large molecules such as peptides and proteins move into and out of the bloodstream by way of
   (a) continuous capillaries.
   (b) fenestrated capillaries.
   (c) thoroughfare channels.
   (d) metarterioles.
7. The local control of blood flow due to the action of precapillary sphincters is
   (a) vasomotion.
   (b) autoregulation.
   (c) selective resistance.
   (d) turbulence.
8. Blood is transported through the venous system by means of
   (a) muscular contractions.
   (b) increasing blood pressure.
   (c) the respiratory pump.
   (d) a and c.
9. Identify the major veins in the following diagram.
   (a) _____
   (b) _____
   (c) _____
   (d) _____
   (e) _____
   (f) _____
   (g) _____
   (h) _____
   (i) _____



10. The most important factor in vascular resistance is
    (a) the viscosity of the blood.
    (b) the diameter of the lumen of blood vessels.
    (c) turbulence due to irregular surfaces of blood vessels.
    (d) the length of the blood vessels.
11. Net hydrostatic pressure forces water _____ a capillary; net osmotic pressure forces water _____ a capillary.
    (a) into, out of
    (b) out of, into
    (c) out of, out of
    (d) into, into

12. The two arteries formed by the division of the brachiocephalic trunk are the
    (a) aorta and internal carotid.
    (b) axillary and brachial.
    (c) external and internal carotid.
    (d) common carotid and subclavian.
13. The unpaired arteries supplying blood to the visceral organs include
    (a) the adrenal, renal, and lumbar arteries.
    (b) the iliac, gonadal, and femoral arteries.
    (c) the celiac and superior and inferior mesenteric arteries.
    (d) a, b, and c.
14. The paired arteries supplying blood to the body wall and other structures outside the abdominopelvic cavity include the
    (a) left gastric, hepatic, splenic, and phrenic arteries.
    (b) adrenal, colic, lumbar, and gonadal arteries.
    (c) iliac, femoral, and lumbar arteries.
    (d) celiac, left gastric, and superior and inferior mesenteric arteries.
15. The vein that drains the dural sinuses of the brain is the
    (a) cephalic vein.
    (b) great saphenous vein.
    (c) internal jugular vein.
    (d) superior vena cava.
16. The vein that collects most of the venous blood inferior to the diaphragm is the
    (a) superior vena cava.
    (b) great saphenous vein.
    (c) inferior vena cava.
    (d) azygos vein.
17. What are the primary forces that cause fluid to move
    (a) out of a capillary at its arterial end and into the interstitial fluid?
    (b) into a capillary at its venous end from the interstitial fluid?
18. What cardiovascular changes occur at birth?

LEVEL 2 Reviewing Concepts

19. A major difference between the arterial and venous systems is that
    (a) arteries are usually more superficial than veins.
    (b) in the limbs there is dual venous drainage.
    (c) veins are usually less branched compared to arteries.
    (d) veins exhibit a much more orderly pattern of branching in the limbs.
    (e) veins are not found in the abdominal cavity.
20. Which of the following conditions would have the *greatest* effect on peripheral resistance?
    (a) doubling the length of a vessel
    (b) doubling the diameter of a vessel
    (c) doubling the viscosity of the blood
    (d) doubling the turbulence of the blood
    (e) doubling the number of white cells in the blood
21. Which of the following is *greater*?
    (a) the osmotic pressure of the interstitial fluid during inflammation
    (b) the osmotic pressure of the interstitial fluid during normal conditions
    (c) neither is greater
22. Relate the anatomical differences between arteries and veins to their functions.
23. Why do capillaries permit the diffusion of materials, whereas arteries and veins do not?
24. How is blood pressure maintained in veins to counter the force of gravity?
25. How do pressure and resistance affect cardiac output and peripheral blood flow?
26. Why is blood flow to the brain relatively continuous and constant?
27. Compare the effects of the cardioacceleratory and cardioinhibitory centers on cardiac output and blood pressure.

LEVEL 3 Critical Thinking and Clinical Applications

28. Bob is sitting outside on a warm day and is sweating profusely. Mary wants to practice taking blood pressures, and he agrees to play the patient. Mary finds that Bob's blood pressure is elevated, even though he is resting and has lost fluid from sweating. (She reasons that fluid loss should lower blood volume and, thus, blood pressure.) Why is Bob's blood pressure high instead of low?
29. People with allergies commonly take antihistamines with decongestants to relieve their symptoms. The container warns that individuals who are being treated for high blood pressure should not take the medication. Why not?
30. Jolene awakens suddenly to the sound of her alarm clock. Realizing that she is late for class, she jumps to her feet, feels light-headed, and falls back on her bed. What probably caused this reaction? Why doesn't this happen all the time?