Color enhanced scanning electron micrograph of an artery.

Cardiovascular System

Peripheral Circulation and Regulation

CHAPTER 21

Complex urban water systems seem rather simple when compared to the intricacy and coordinated functions of blood vessels. The heart is the pump that provides the major force causing blood to circulate, and the blood vessels are the pipes that carry blood to tissues of the body and back to the heart. In addition, the blood vessels participate in the regulation of blood pressure and help to direct blood flow to tissues that are most active.

The peripheral circulatory system comprises two sets of blood vessels: systemic and pulmonary vessels. **Systemic vessels** transport blood through essentially all parts of the body from the left ventricle and back to the right atrium. **Pulmonary vessels** transport blood from the right ventricle through the lungs and back to the left atrium (see figure 20.1). Both the blood vessels and the heart are regulated to ensure that the blood pressure is high enough to cause blood flow in sufficient quantities to meet the metabolic needs of the tissues. The cardiovascular system ensures the survival of the tissues in the body by supplying nutrients to and removing waste products from them.

This chapter explains the *general features of blood vessel structure* (712), *pulmonary circulation* (717), *systemic circulation: arteries* (717), *systemic circulation: veins* (728), *dynamics of blood circulation* (740), *physiology of systemic circulation* (744), *control of blood flow in tissues* (749), and *regulation of mean arterial pressure* (753).

General Features of Blood Vessel Structure

Objectives

- ***Describe the structure and function of the capillaries, arteries, and veins.***
- ***Describe the structural and functional changes that occur in arteries as they age.***

The ventricles pump blood from the heart into large elastic arteries that branch repeatedly to form many progressively smaller arteries. As they become smaller, the arteries undergo a gradual transition from having walls that contain a large amount of elastic tissue and a smaller amount of smooth muscle to having walls with a smaller amount of elastic tissue and a relatively large amount of smooth muscle. Although the arteries form a continuum from the largest to the smallest branches, they normally are classified as (1) elastic arteries, (2) muscular arteries, or (3) arterioles.

Blood flows from arterioles into capillaries. Most of the exchange that occurs between the blood and interstitial spaces occurs across the walls of capillaries. Their walls are the thinnest of all the blood vessels, blood flows through them slowly, and a greater number of them exist than any other blood vessel type.

From the capillaries, blood flows into the venous system. When compared to arteries, the walls of the veins are thinner and contain less elastic tissue and fewer smooth muscle cells. The veins increase in diameter and decrease in number, and their walls increase in thickness as they project toward the heart. They are classified as (1) venules, (2) small veins, or (3) medium or large veins.

Capillaries

All blood vessels have an internal lining of simple squamous epithelial cells called the **endothelium** (en-dō-thē′lē-ŭm), which is continuous with the endocardium of the heart.

The capillary wall consists primarily of endothelial cells (figure 21.1), which rest on a basement membrane. Outside the basement membrane is a delicate layer of loose connective tissue that merges with the connective tissue surrounding the capillary.

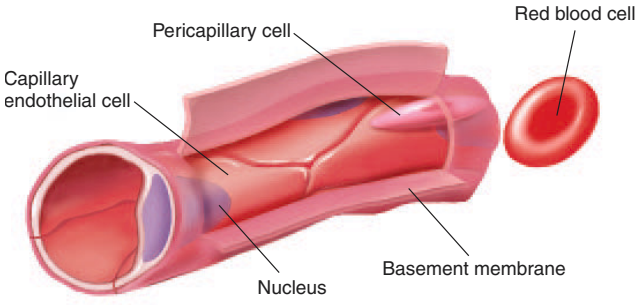


Figure 21.1 Capillary

Section of a capillary showing that it is composed of flattened endothelial cells.

Along the length of the capillary are some scattered cells that are closely associated with the endothelial cells. These scattered cells lie between the basement membrane and the endothelial cells and are called **pericapillary cells.** They are apparently fibroblasts, macrophages, or undifferentiated smooth muscle cells.

Most capillaries range from 7–9 μm in diameter, and they branch without a change in their diameter. Capillaries are variable in length, but in general, they are approximately 1 mm long. Red blood cells flow through most capillaries in a single file and frequently are folded as they pass through the smaller-diameter capillaries.

Types of Capillaries

Capillaries are classified as continuous, fenestrated, or sinusoidal, depending on their diameter and permeability characteristics.

Continuous capillaries are approximately 7–9 μm in diameter, and their walls exhibit no gaps between the endothelial cells. Continuous capillaries are less permeable to large molecules than are other capillary types and occur in muscle, nervous tissue, and many other locations.

In **fenestrated** (fen′es-trā′ted) **capillaries,** endothelial cells have numerous fenestrae. The **fenestrae** (fe-nes′trē; windows) are areas approximately 70–100 μm in diameter in which the cytoplasm is absent and the plasma membrane consists of a porous diaphragm that's thinner than the normal plasma membrane. Fenestrated capillaries are in tissues where capillaries are highly permeable, such as in the intestinal villi, ciliary process of the eye, choroid plexuses of the central nervous system, and glomeruli of the kidney.

Sinusoidal (sī-nŭ-soy′dăl) **capillaries** are larger in diameter than either continuous or fenestrated capillaries, and their basement membrane is less prominent. Their fenestrae are larger than those in fenestrated capillaries. The sinusoidal capillaries occur in such places as endocrine glands, where large molecules cross their walls.

Sinusoids are large-diameter sinusoidal capillaries. Their basement membrane is sparse and often missing, and their structure suggests that large molecules and sometimes cells can move readily across their walls between the endothelial cells. Sinusoids are common in the liver and the bone marrow. Macrophages are closely associated with the endothelial cells of the liver sinusoids. **Venous sinuses** are similar in structure to the sinusoidal capillaries but are even larger in diameter. They occur primarily in the spleen, and they have large gaps between the endothelial cells that make up their walls.

Substances cross capillary walls by diffusing through the endothelial cells, through fenestrae, or between the endothelial cells. Lipid-soluble substances, such as oxygen and carbon dioxide, and small water-soluble molecules readily diffuse through the plasma membrane. Larger water-soluble substances must pass through the fenestrae or gaps between the endothelial cells. In addition, transport by pinocytosis occurs, but little is known about its role in the capillaries. The walls of the capillaries are effective permeability barriers because red blood cells and large water-soluble molecules like proteins cannot readily pass through them.

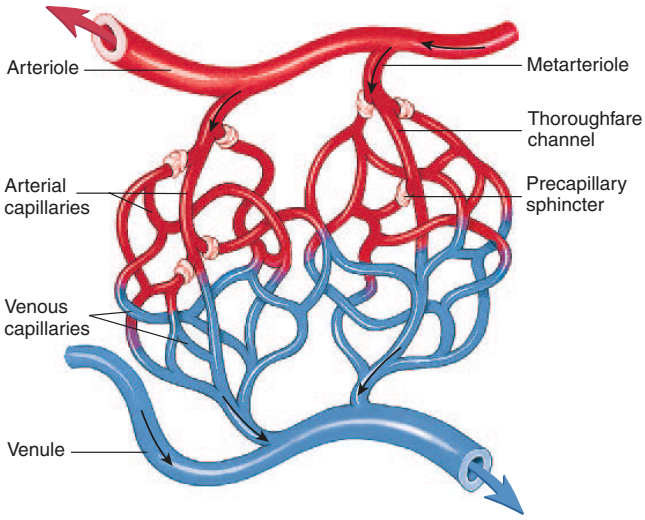


Figure 21.2 Capillary Network

The metarteriole giving rise to the network feeds directly from an arteriole into the thoroughfare channel, which feeds into the venule. The network forms numerous branches that transport blood from the thoroughfare channel and can return to the channel. Smooth muscle cells, called precapillary sphincters, regulate blood flow through the capillaries. Blood flow decreases when the precapillary sphincters constrict and increases when they dilate.

Capillary Network

Arterioles supply blood to each capillary network (figure 21.2). Blood then flows through the capillary network and into the venules. The ends of capillaries closest to the arterioles are **arterial capillaries,** and the ends closest to venules are **venous capillaries.**

Blood flows from arterioles through **metarterioles** (met′ar-tēr′ē-ōlz), which have isolated smooth muscle cells along their walls. Blood flows from a metarteriole into a **thoroughfare channel,** which extends in a relatively direct fashion from a metarteriole to a venule. Blood flow through thoroughfare channels is relatively continuous. Several capillaries branch from the thoroughfare channels, and in these branches blood flow is intermittent. Smooth muscle cells called **precapillary sphincters,** which are located at the origin of the branches (see figure 21.2), regulate flow in these capillaries.

Capillary networks are more numerous and more extensive in highly metabolic tissues, such as the lung, liver, kidney, skeletal muscle, and cardiac muscle. Capillary networks in the skin have many more thoroughfare channels than capillary networks in cardiac or skeletal muscle. Capillaries in the skin function in thermoregulation, and heat loss results from the flow of a large volume of blood through them. In muscle, however, nutrient and waste product exchange is the major function of the capillaries.

1. *Name, in order, all the types of blood vessels, starting at the heart, going into the tissues, and returning to the heart.*
2. *Describe the three types of capillaries. Explain the ways that materials pass through the capillary wall.*
3. *Describe a capillary network. Where is the smooth muscle that regulates blood flow into and through the capillary network located? What is the function of a thoroughfare channel?*
4. *Contrast the function of capillaries in the skin with the function of capillaries in muscle tissue.*

Structure of Arteries and Veins

General Features

Except for the capillaries and the venules, the blood vessel walls consist of three relatively distinct layers, which are most apparent in the muscular arteries and least apparent in the veins. From the lumen to the outer wall of the blood vessels, the layers, or **tunics** (too′niks), are (1) the tunica intima, (2) the tunica media, (3) and the tunica adventitia, or tunica externa (figure 21.3).

The **tunica intima** consists of endothelium, a delicate connective tissue basement membrane, a thin layer of connective tissue called the lamina propria, and a fenestrated layer of elastic fibers called the **internal elastic membrane.** The internal elastic membrane separates the tunica intima from the next layer, the tunica media.

The **tunica media,** or middle layer, consists of smooth muscle cells arranged circularly around the blood vessel. The amount of blood flowing through a blood vessel can be regulated by contraction or relaxation of the smooth muscle in the tunica media. A decrease in blood flow results from **vasoconstriction** (vā′sō-kon-strik′shŭn, vas′ō-kon-strik′shŭn), a decrease in blood vessel diameter caused by smooth muscle contraction, whereas an increase in blood flow is produced by **vasodilation** (vā′sō-dī-lā′shŭn,vas-ō-dī-lā′shŭn), an increase in blood vessel diameter because of smooth muscle relaxation. The tunica media also contains variable amounts of elastic and collagen fibers, depending on the size of the vessel. An external elastic membrane, which separates the tunica media from the tunica adventitia, can be identified at the outer border of the tunica media in some arteries. A few longitudinally oriented smooth muscle cells occur in some arteries near the tunica intima.

The **tunica adventitia** (too′ni-kă ad-ven-tish′ă) is composed of connective tissue, which varies from dense connective tissue near the tunica media to loose connective tissue that merges with the connective tissue surrounding the blood vessels.

The relative thickness and composition of each layer varies with the diameter of the blood vessel and its type. The transition from one artery type or from one vein type to another is gradual, as are the structural changes.

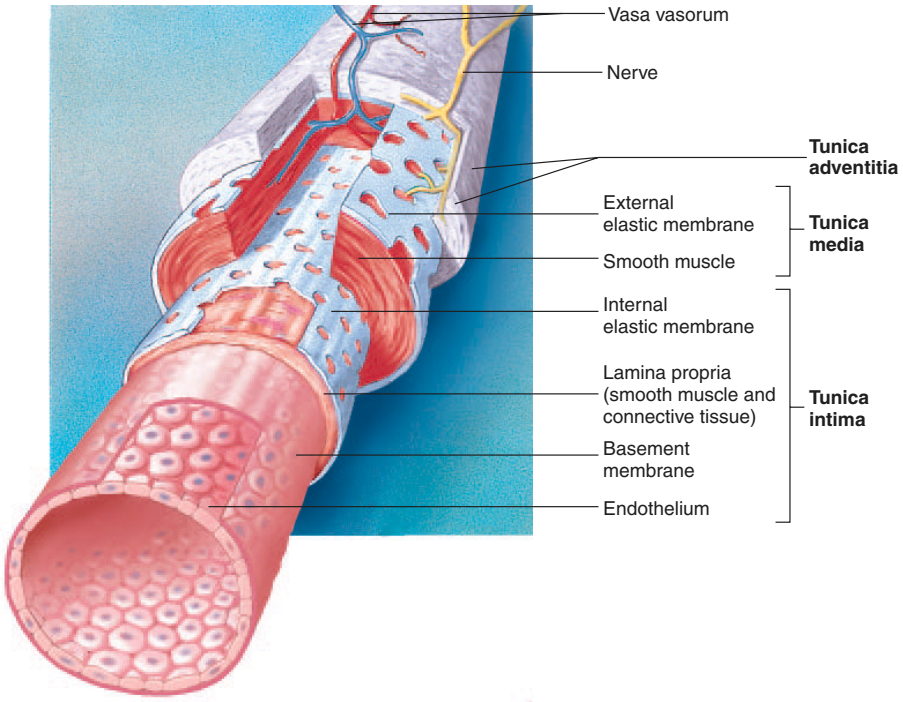


Figure 21.3 Histology of a Blood Vessel

The layers, or tunics, of the blood vessel wall include the intima, media, and adventitia. A vasa vasorum is a blood vessel that supplies blood to the wall of the blood vessel.

Large Elastic Arteries

Elastic arteries have the largest diameters (figure 21.4*a*) and often are called **conducting arteries.** Pressure is relatively high in these vessels, and it fluctuates between systolic and diastolic values. A greater amount of elastic tissue and a smaller amount of smooth muscle occur in their walls compared to the elastic tissue and smooth muscle of other arteries. The elastic fibers are responsible for the elastic characteristics of the blood vessel wall, but collagenous connective tissue determines the degree to which the arterial wall can be stretched.

The tunica intima is relatively thick. The elastic fibers of the internal and external elastic membranes merge and are not recognizable as distinct layers. The tunica media consists of a meshwork of elastic fibers with interspersed circular smooth muscle cells and some collagen fibers. The tunica adventitia is relatively thin.

Muscular Arteries

The larger **muscular arteries,** often called **medium arteries,** can be observed in a gross dissection. They include most of the smaller *unnamed* arteries. Their walls are relatively thick compared to their diameter, mainly because the tunica media contains 25–40 layers of smooth muscle (figure 21.4*b*). The tunica intima of the medium arteries has a well-developed internal elastic membrane. The tunica adventitia is composed of a relatively thick layer of collagenous connective tissue that blends with the surrounding connective tissue. Medium arteries frequently are called **distributing arteries** because the smooth muscle cells allow these vessels to partially regulate blood supply to different regions of the body by either constricting or dilating.

Smaller muscular arteries range from 40–300 μm in diameter, and those that are 40 μm in diameter have approximately three or four layers of smooth muscle in their tunica media, whereas arteries that are 300 μm across have essentially the same structure as the larger muscular arteries. The small muscular arteries are adapted for vasodilation and vasoconstriction.

Arterioles

The **arterioles** (ar-tēr′ē-ōlz) transport blood from small arteries to capillaries and are the smallest arteries in which the three tunics can be identified. They range from approximately 40 μm to as small as 9 μm in diameter. The tunica intima has no observable internal elastic membrane, and the tunica media consists of one or two layers of circular smooth muscle cells. The arterioles, like the small arteries, are capable of vasodilation and vasoconstriction.

Venules and Small Veins

Venules (ven′-oolz, vē′noolz), with a diameter of up to 40–50 μm, are tubes composed of endothelium resting on a delicate basement membrane. Their structure, except for their diameter, is very similar to that of capillaries. A few isolated smooth muscle cells exist

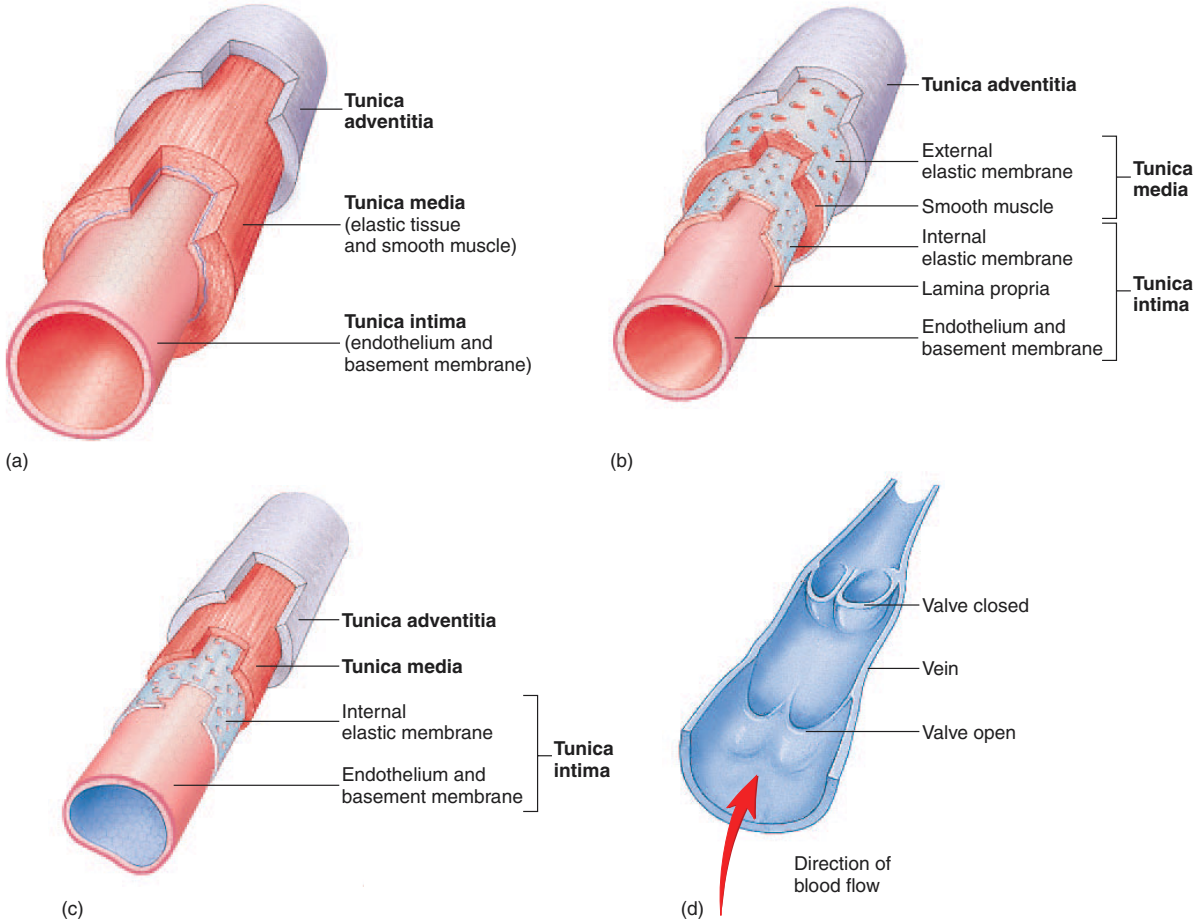


Figure 21.4 Structural Comparison of Blood Vessel Types

(a) Elastic arteries are large-diameter arteries with thick walls that contain a large amount of elastic connective tissue in the tunica media. *(b)* Muscular arteries have a distinctive layer of smooth muscle cells in the tunica media, and they are capable of constriction and dilation. *(c)* Medium veins have thinner walls. The tunica media is thinner than the tunica media in arteries and contains fewer smooth muscle cells. The dominant layer in the veins is the tunica adventitia. *(d)* The valves in veins are folds in the endothelium that allow blood to flow toward the heart but not in the opposite direction.

outside the endothelial cells, especially in the larger venules. As the vessels increase to 0.2–0.3 mm in diameter, the smooth muscle cells form a continuous layer; the vessels then are called **small veins.** The small veins also have a tunica adventitia composed of collagenous connective tissue.

The venules collect blood from the capillaries and transport it to small veins, which in turn transport it to medium-sized veins. Nutrient exchange occurs across the venule walls, but, as the walls of the small veins increase in thickness, the degree of nutrient exchange decreases.

Medium and Large Veins

Most of the veins observed in gross anatomic dissections, except for the large veins, are **medium veins.** They collect blood from small veins and deliver it to large veins. The **large veins** transport blood from the medium veins to the heart. Their tunica intima is thin and consists of endothelial cells, a relatively thin layer of collagenous connective tissue, and a few scattered elastic fibers. The tunica media is also thin and is composed of a thin layer of circularly arranged smooth muscle cells, collagen fibers, and a few sparsely distributed elastic fibers. The tunica adventitia, which is composed of collagenous connective tissue, is the predominant layer (figure 21.4*c*).

Valves

Veins having diameters greater than 2 mm contain **valves** that allow blood to flow toward the heart but not in the opposite direction (figure 21.4*d*). The valves consist of folds in the tunica intima

that form two flaps that are shaped and function like the semilunar valves of the heart. The two folds overlap in the middle of the vein so that, when blood attempts to flow in a reverse direction, the valves occlude the vessel. Many valves are present in the medium veins, and the number is greater in veins of the lower extremities than in veins of the upper extremities.

Varicose Veins, Phlebitis, and Gangrene

Stretching of the vein walls in the lower limbs causes valves to become incompetent, which results in **varicose veins.** The veins become so dilated that the flaps of the venous valves no longer overlap and prevent the backflow of blood. As a consequence, the venous pressure is greater than normal in the veins of the lower limbs, resulting in edema. Blood flow in the veins can become sufficiently stagnant that the blood clots. The condition can result in **phlebitis** (fle-bī′tis), which is inflammation of the veins. If the inflammation is severe and blood flow becomes stagnant in a large area, it can lead to **gangrene** (gang′grēn), which is tissue death caused by a reduction or loss of blood supply. Some people have a genetic propensity for the development of varicose veins. For women with that genetic propensity, some medical conditions increase the pressure in veins, causing them to stretch, and varicose veins can develop. One such condition is pregnancy, in which the venous pressure in the veins that drain the lower limbs increases because of compression of the veins by the expanded uterus.

Vasa Vasorum

For arteries and veins greater than 1 mm in diameter, nutrients cannot diffuse from the lumen of the vessel to all of the layers of the wall. Nutrients are, therefore, supplied to their walls by way of small blood vessels called **vasa vasorum** (vā′să vā′sor-ŭm), which penetrate from the exterior of the vessel to form a capillary network in the tunica adventitia and the tunica media (see figure 21.3).

Arteriovenous Anastomoses

Arteriovenous anastomoses (ă-nas′tō-mō′sez) allow blood to flow from arterioles to small veins without passing through capillaries. A **glomus** (glō′mŭs) is an arteriovenous anastomosis that consists of arterioles arranged in a convoluted fashion surrounded by collagenous connective tissue.

Naturally occurring arteriovenous anastomoses are present in large numbers in the sole of the foot, the palm of the hand, the terminal phalanges, and the nail beds. They function in temperature regulation. **Pathologic arteriovenous anastomoses** can result from injury or tumors. They cause the direct flow of blood from arteries to veins and can, if they are sufficiently large, lead to heart failure because of the tremendous increase in venous return to the heart.

Nerves

Unmyelinated sympathetic nerve fibers (see figure 21.3) richly innervate the walls of most blood vessels. Some blood vessels, such as those in the penis or clitoris, are innervated by parasympathetic fibers. The nerve fibers form plexuses in the tunica adventitia, and nerve terminals project among the smooth muscle cells of the tunica media. Synapses consist of enlargements of the nerve fibers. Small arteries and arterioles are innervated to a greater extent than other blood vessel types. The response of the blood vessels to sympathetic stimulation is vasoconstriction. Parasympathetic stimulation of blood vessels in the penis or clitoris results in vasodilation.

The smooth muscle cells of blood vessels act to some extent as a functional unit. Frequent gap junctions occur between adjacent smooth muscle cells, and as a consequence, stimulation of a few smooth muscle cells in the vessel wall results in constriction of a relatively large segment of the blood vessel.

A few myelinated sensory neurons innervate some blood vessels and function as baroreceptors. They monitor stretch in the blood vessel wall and detect changes in blood pressure.

Aging of the Arteries

The walls of all arteries undergo changes as they age, although some arteries change more rapidly than others and some individuals are more susceptible to change than others. The most significant changes occur in the large elastic arteries like the aorta, large arteries that carry blood to the brain, and the coronary arteries. The age-related changes described here refer to these blood vessel types. Changes in muscular arteries do occur, but they are less dramatic and often do not result in disruption of normal blood vessel function.

Degenerative changes in arteries that make them less elastic are referred to collectively as **arteriosclerosis** (ar-tēr′ē-ō-skler-ō′sis; hardening of the arteries). These changes occur in many individuals and become more severe with advancing age. A related term, **atherosclerosis** (ath′er-ō-skler-ō′sis), refers to the deposition of material in the walls of arteries to form plaques. The material is a fatlike substance containing cholesterol (figure 21.5). The fatty material can be replaced later with dense connective tissue and calcium deposits. The initial signs of arteriosclerosis have

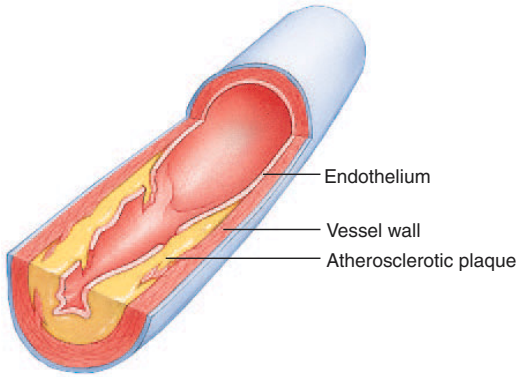


Figure 21.5 Atherosclerotic Plaque in an Artery
Atherosclerotic plaques develop within the tissue of the artery wall.

been identified in the arteries of people in their teens, and it develops earlier and progresses more rapidly in some individuals than in others.

In arteriosclerosis, the tunica intima thickens and the tunica media becomes less elastic, because of a chemical change that takes place in the elastic fibers. Fat gradually accumulates between the elastic and collagen fibers to produce a lesion that protrudes into the lumen of the vessel, which can eventually hamper normal blood flow. In advanced forms of arteriosclerosis, calcium deposits, primarily in the form of calcium carbonate, accumulate in the walls of the blood vessels.

Arteriosclerosis greatly increases resistance to blood flow. Advanced arteriosclerosis, as a consequence, adversely affects the normal circulation of blood and greatly increases the work performed by the heart.

Some investigators think that arteriosclerosis may not be a pathologic process. Instead, it may be simply an aging or wearing-out process. Evidence also suggests that arteriosclerosis may result from inflammation, which, in some cases, may be the result of an autoimmune disease. In either case, several factors increase the rate at which it develops. Obesity, high dietary cholesterol and other fat consumption, and smoking are some of the factors correlated with the premature development of arteriosclerosis.

5. *Name the three layers of a blood vessel. What kinds of tissue are in each layer?*
6. *Compare the amount of elastic fibers and smooth muscle found in each different type of artery and vein.*
7. *What is the function of valves in blood vessels? In which blood vessels are they found?*
8. *Define the terms vasa vasorum and arteriovenous anastomosis, and give their function.*
9. *Describe the innervation of the walls of blood vessels. Which types of vessels have the greatest innervation?*
10. *Describe the changes that occur in arteries due to aging. In which vessels do the most significant changes occur? Name the factors associated with premature arteriosclerosis.*

Pulmonary Circulation

Objective

- *List the blood vessels of the pulmonary circulation, and describe their function.*

The heart pumps blood from the right ventricle into the **pulmonary** (pŭl′mō-nār-ē; relating to the lungs) **trunk** (figure 21.6). This short vessel, 5 cm long, branches into the right and left **pulmonary arteries,** one transporting blood to each lung. Within the lungs, gas exchange occurs between air in the lungs and the blood. Two **pulmonary veins** exit each lung and enter the left atrium (see figure 20.10).

11. *For the vessels of the pulmonary circulation, give their starting point, ending point, and function.*

Systemic Circulation: Arteries

Objective

- *List the major arteries that supply each of the major body areas.*

Oxygenated blood entering the heart from the pulmonary veins passes through the left atrium into the left ventricle and from the left ventricle into the aorta. Blood flows from the aorta to all parts of the body (see figure 21.6).

Aorta

All **arteries** of the systemic circulation are derived either directly or indirectly from the **aorta** (ā-ōr′tă), which usually is divided into three general parts: the ascending aorta, the aortic arch, and the descending aorta. The descending aorta is divided further into a thoracic aorta and an abdominal aorta (see figure 21.12).

At its origin from the left ventricle, the aorta is approximately 2.8 cm in diameter. Because it passes superiorly from the heart, this part is called the **ascending aorta.** It's approximately 5 cm long and has only two arteries branching from it: the right and left **coronary arteries,** which supply blood to the cardiac muscle (see figure 20.6*a*).

The aorta then arches posteriorly and to the left as the **aortic arch.** Three major branches, which carry blood to the head and upper limbs, originate from the aortic arch: the brachiocephalic artery, the left common carotid artery, and the left subclavian artery.

Trauma and the Aorta

Trauma that ruptures the aorta is almost immediately fatal. Trauma can also lead to an **aneurysm** (an′ū-rizm), however, which is a bulge caused by a weakened spot in the aortic wall. If the weakened aortic wall leaks blood slowly into the thorax, the aneurysm must be corrected surgically. The majority of traumatic aortic arch ruptures occur during automobile accidents and result from the great force with which the body is thrown into the steering wheel, dashboard, or other objects. Waist-type safety belts alone do not prevent this type of injury as effectively as shoulder-type safety belts and air bags.

The next part of the aorta is the longest part, the **descending aorta.** It extends through the thorax in the left side of the mediastinum and through the abdomen to the superior margin of the pelvis. The **thoracic aorta** is that portion of the descending aorta located in the thorax. It has several branches that supply various structures between the aortic arch and the diaphragm. The **abdominal aorta** is that part of the descending aorta between the diaphragm and the point at which the aorta ends by dividing into the two **common iliac** (il′ē-ak; relating to the flank area) **arteries.** The abdominal aorta has several branches that supply the abdominal wall and organs. Its terminal branches, the common iliac arteries, supply blood to the pelvis and lower limbs.

Coronary Arteries

The **coronary** (kōr′o-năr-ē; encircling the heart like a crown) **arteries,** which are the only branches of the ascending aorta, are described in chapter 20.

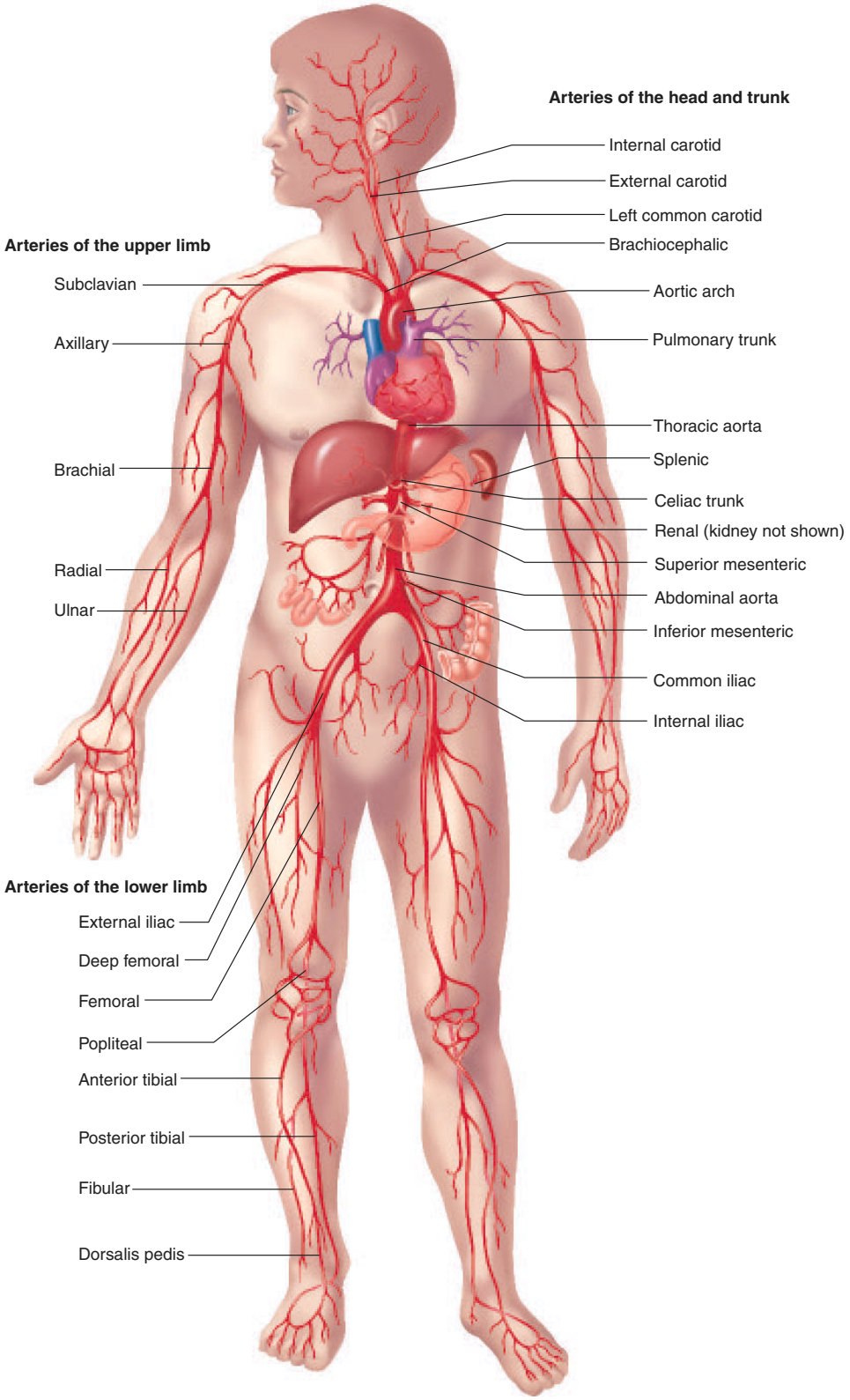


Figure 21.6 The Major Arteries

The arteries blood from the heart to the tissues of the body.

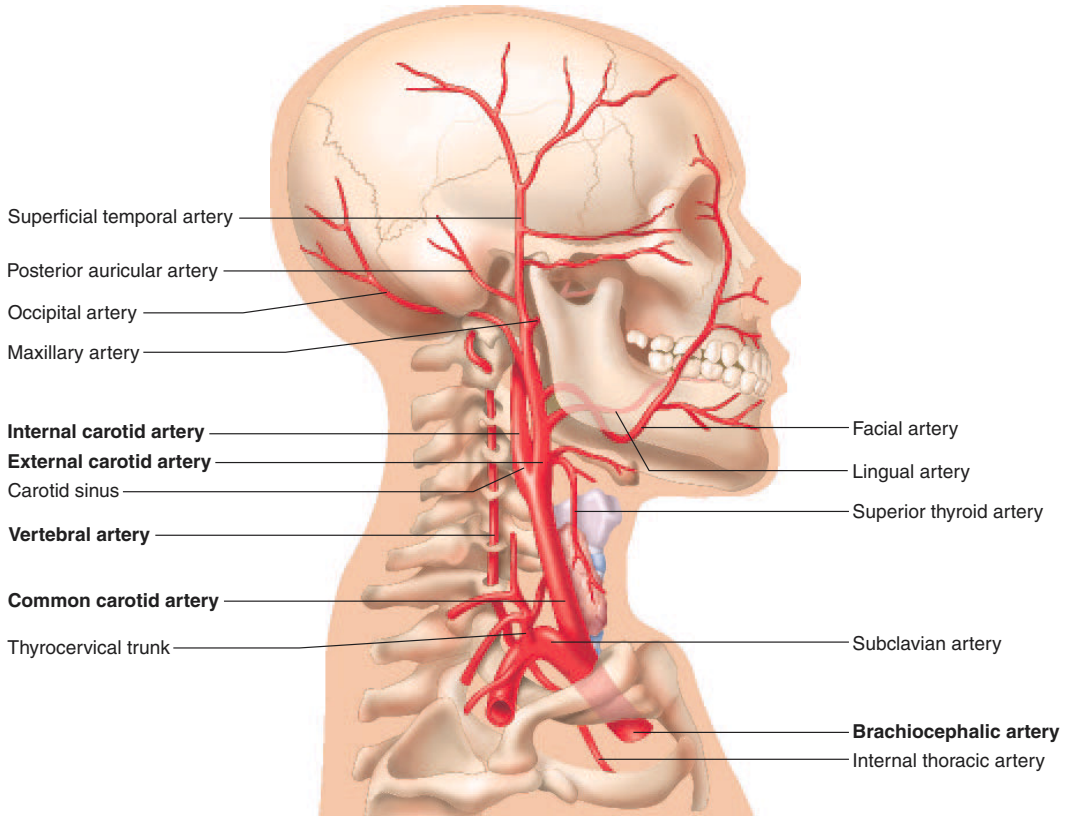


Figure 21.7 Arteries of the Head and Neck

The brachiocephalic artery, the right common carotid artery, the right subclavian artery, and their branches. The major arteries to the head are the common carotid and vertebral arteries.

Arteries to the Head and the Neck

The first vessel to branch from the aortic arch is the **brachiocephalic** (brā′kē-ō-se-fal′ik; arm and head) **artery** (figure 21.7). It is a very short artery, and it branches at the level of the clavicle to form the **right common carotid** (ka-rot′id) **artery,** which transports blood to the right side of the head and neck, and the **right subclavian** (sŭb-klā′vē-an; below the clavicle) **artery,** which transports blood to the right upper limb (see figures 21.6, 21.7, 21.9, 21.10, and 21.11).

The second and third branches of the aortic arch are the **left common carotid artery,** which transports blood to the left side of the head and neck, and the **left subclavian artery,** which transports blood to the left upper limb.

The common carotid arteries extend superiorly, without branching, along either side of the neck from their base to the inferior angle of the mandible, where each common carotid artery branches into **internal** and **external carotid arteries** (see figures 21.7 and 21.9). At the point of bifurcation on each side of the neck, the common carotid artery and the base of the internal carotid artery are dilated slightly to form the **carotid sinus,** which is important in monitoring blood pressure (baroreceptor reflex). The external carotid arteries have several branches that supply the structures of the neck and face (table 21.1; see figures 21.7 and 21.9). The internal carotid arteries, together with the vertebral arteries, which are branches of the subclavian arteries, supply the brain (see table 2.1 and figures 21.7, 21.8, and 21.9).

P R E D I C T 1

The term *carotid* means to put to sleep, implying that if the carotid arteries are occluded for even a short time, the patient could lose consciousness (go to sleep). The blood supply to the brain is extremely important to its function. Elimination of this supply for even a relatively short time can result in permanent brain damage because the brain is dependent on oxidative metabolism and quickly malfunctions in the absence of oxygen. What is the physiologic significance of arteriosclerosis, which slowly reduces blood flow through the carotid arteries?

Branches of the subclavian arteries, the **left** and **right vertebral arteries,** enter the cranial cavity through the foramen magnum, give off arteries to the cerebellum, and then unite to form a single, midline **basilar** (bas′i-lăr) **artery** (see figures 21.8 and 21.9; see table 21.1). The basilar artery gives off branches to the pons and the

Table 21.1 Arteries of the Head and Neck (see figures 21.7, 21.8, and 21.9)

Arteries	Tissues Supplied
Common Carotid Arteries	Head and neck by branches listed below
External Carotid	
Superior thyroid	Neck, larynx, and thyroid gland
Lingual	Tongue, mouth, and submandibular and sublingual glands
Facial	Mouth, pharynx, and face
Occipital	Posterior head and neck and meninges around posterior brain
Posterior auricular	Middle and inner ear, head, and neck
Ascending pharyngeal	Deep neck muscles, middle ear, pharynx, soft palate, and meninges around posterior brain
Superficial temporal	Temple, face, and anterior ear
Maxillary	Middle and inner ears, meninges, lower jaw and teeth, upper jaw and teeth, temple, external eye structures, face, palate, and nose
Internal Carotid	
Posterior communicating	Joins the posterior cerebral artery
Anterior cerebral	Anterior portions of the cerebrum and forms the anterior communicating arteries
Middle cerebral	Most of the lateral surface of the cerebrum
Vertebral Arteries	
(branches of the subclavian arteries)	
Anterior spinal	Anterior spinal cord
Posterior inferior cerebellar	Cerebellum and fourth ventricle
Basilar Artery	
(formed by junction of vertebral arteries)	
Anterior inferior cerebellar	Cerebellum
Superior cerebellar	Cerebellum and midbrain
Posterior cerebral	Posterior portions of the cerebrum

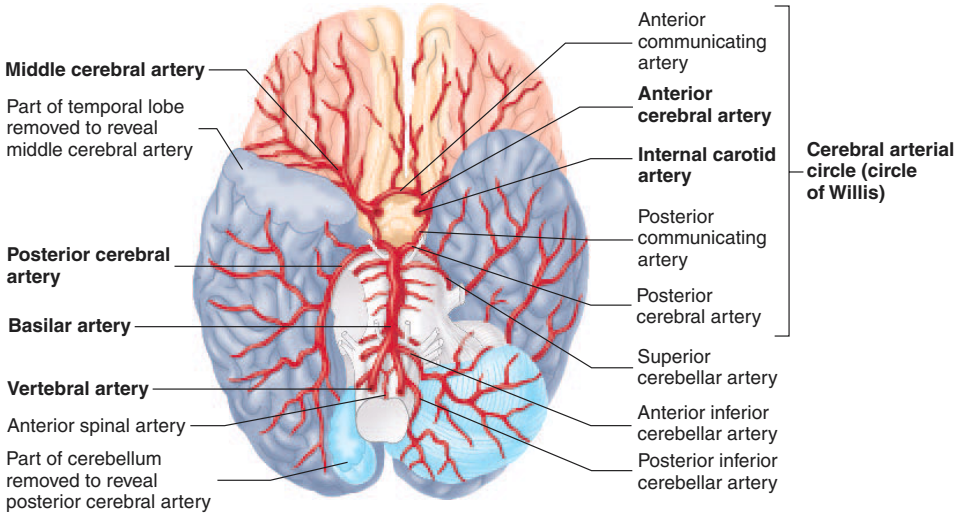


Figure 21.8 Arteries of the Brain

Inferior view of the brain showing the vertebral, basilar, and internal carotid arteries and their branches. (Colors indicate brain regions supplied by various arteries: *yellow,* anterior cerebral; *pink,* middle cerebral; *purple,* posterior cerebral; *blue,* cerebellar arteries; *white,* arteries to brainstem.)

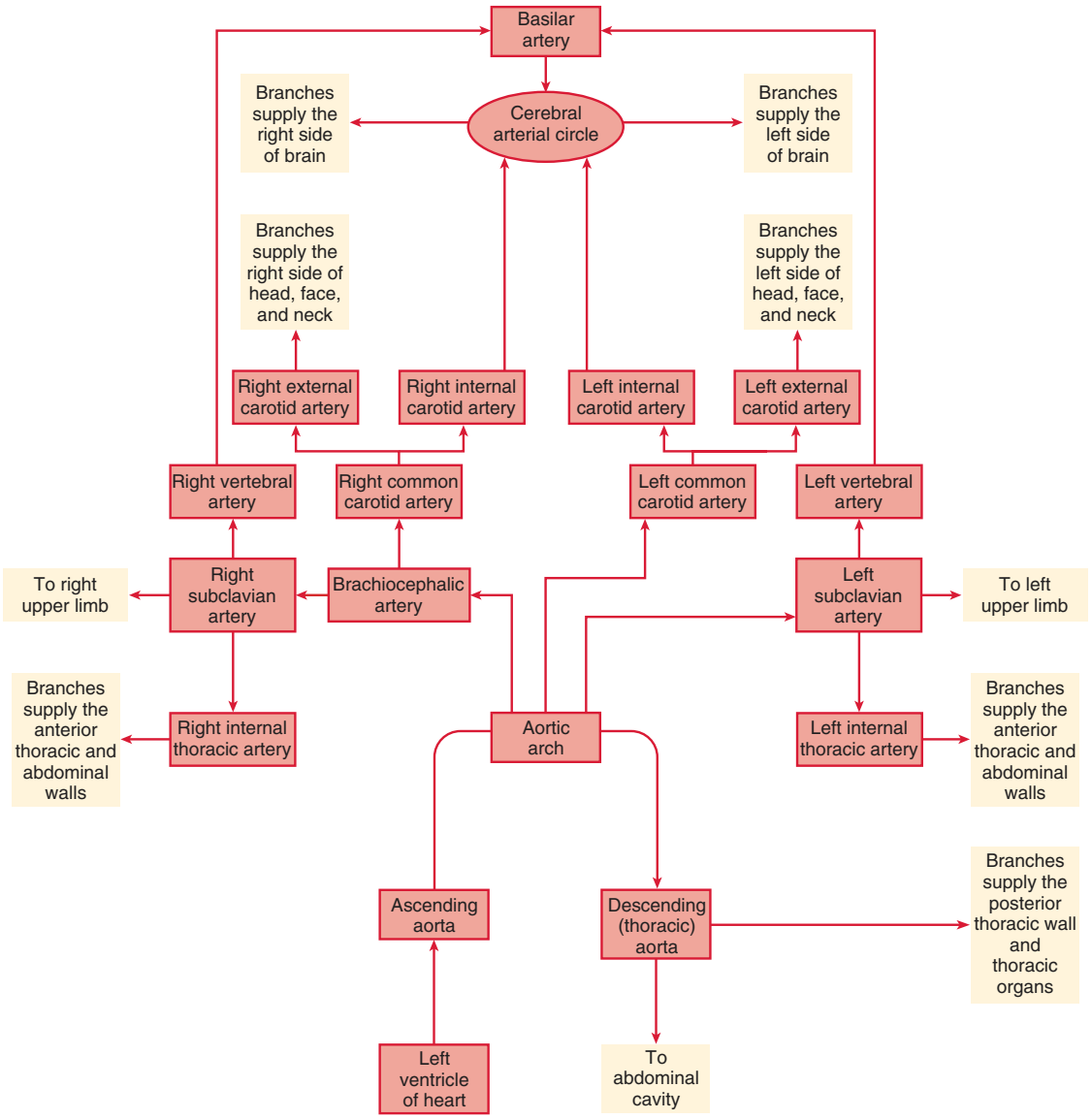


Figure 21.9 Major Arteries of the Head and Thorax

cerebellum and then branches to form the **posterior cerebral arteries,** which supply the posterior part of the cerebrum (see figure 21.8).

The internal carotid arteries enter the cranial vault through the carotid canals and terminate by forming the **middle cerebral arteries,** which supply large parts of the lateral cerebral cortex (see figure 21.8). Posterior branches of these arteries, the **posterior communicating arteries,** unite with the posterior cerebral arteries; and anterior branches, the **anterior cerebral arteries,** supply blood to the frontal lobes of the brain. The anterior cerebral arteries are in turn connected by an **anterior communicating artery,** which completes a circle around the pituitary gland and the base of the brain called the **cerebral arterial circle** (circle of Willis) (see figures 21.8 and 21.9).

Stroke

A **stroke** is a sudden neurologic disorder often caused by a decreased blood supply to a part of the brain. It can occur as a result of a **thrombosis** (throm-bō′sis; a stationary clot), an **embolism** (em′bō-lizm; a floating clot that becomes lodged in smaller vessels), or a **hemorrhage** (hem′ō-rij; rupture or leaking of blood from vessels). Any one of these conditions can result in a loss of blood supply or in trauma to a part of the brain. As a result, the tissue normally supplied by the arteries becomes **necrotic** (nĕ-krot′ik; dead). The affected area is called an **infarct** (in′farkt; to stuff into, an area of cell death). The neurologic results of a stroke are described in chapter 14.

Arteries of the Upper Limb

The three major arteries of the upper limb, called the **subclavian, axillary,** and **brachial arteries,** are a continuum rather than a branching system. The axillary artery is the continuation of the subclavian artery, and the brachial artery is the continuation of the axillary artery. The subclavian artery is located deep to the clavicle, the axillary artery is within the axilla, and the brachial artery lies within the arm itself (table 21.2 and figures 21.10 and 21.11).

The brachial artery divides at the elbow into **ulnar** and **radial arteries,** which form two arches within the palm of the hand, referred to as the superficial and deep palmar arches. The **superficial palmar arch** is formed by the ulnar artery and is completed by anastomosing with the radial artery. The **deep palmar arch** is formed by the radial artery and is completed by anastomosing with the ulnar artery. This arch is not only deep to the superficial arch but is proximal as well.

Digital (dij′i-tăl; relating to the digits—the fingers and the thumb) **arteries** branch from each of the two palmar arches and unite to form single arteries on the medial and lateral sides of each digit.

Thoracic Aorta and Its Branches

The branches of the thoracic aorta are divided into two groups: the **visceral branches** supplying the thoracic organs, and the

Table 21.2 Arteries of the Upper Limbs (see figure 21.10)

Arteries	Tissues Supplied
Subclavian Arteries	
(right subclavian originates from the brachiocephalic artery, and left subclavian originates directly from the aorta)	
Vertebral	Spinal cord and cerebellum form the basilar artery (see table 21.1)
Internal thoracic	Diaphragm, mediastinum, pericardium, anterior thoracic wall, and anterior abdominal wall
Thyrocervical trunk	Inferior neck and shoulder
Axillary Arteries	
(continuation of subclavian)	
Thoracoacromial	Pectoral region and shoulder
Lateral thoracic	Pectoral muscles, mammary gland, and axilla
Subscapular	Scapular muscles
Brachial Arteries	
(continuation of axillary arteries)	
Deep brachial	Arm and humerus
Radial	Forearm
Deep palmar arch	Hand and fingers
Digital arteries	Fingers
Ulnar	Forearm
Superficial palmar arch	Hand and fingers
Digital arteries	Fingers

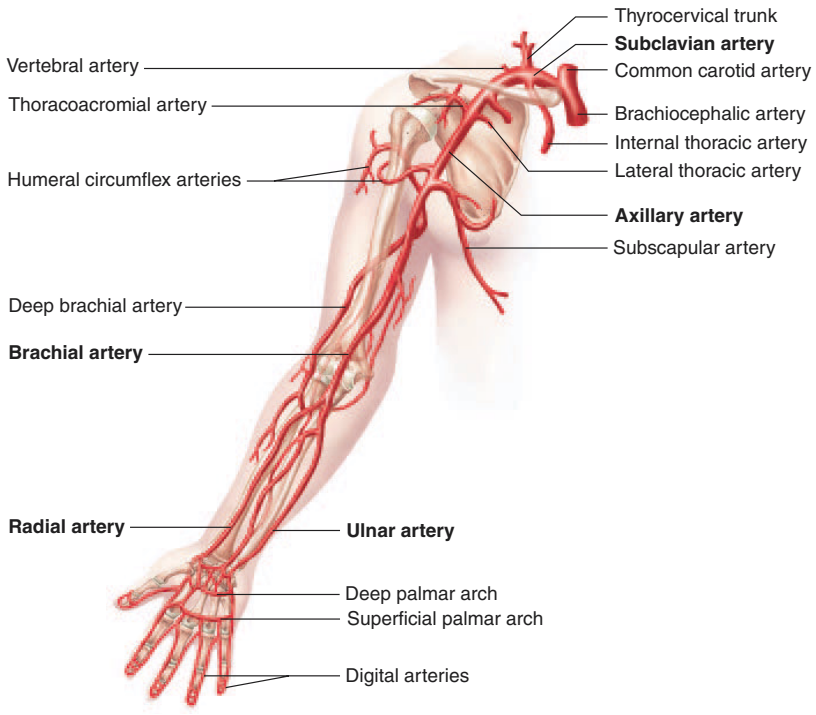


Figure 21.10 Arteries of the Upper Limb

The right brachiocephalic, subclavian, axillary, and brachial arteries and their branches.

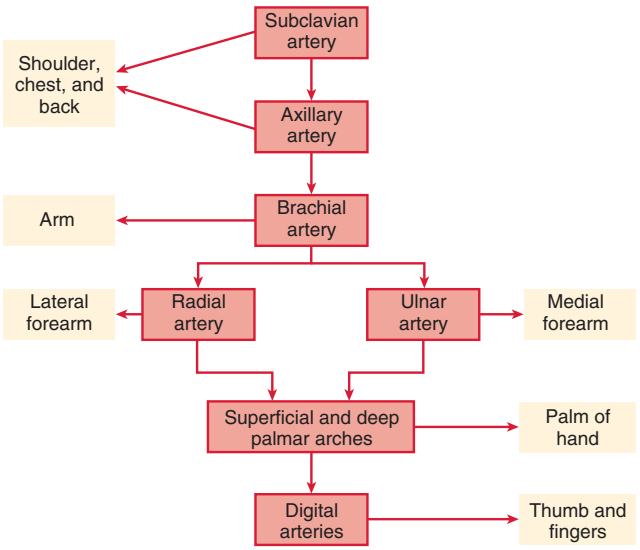


Figure 21.11 Major Arteries of the Shoulder and Upper Limb

Table 21.3 Thoracic and Abdominal Aorta (see figures 21.12 and 21.13)

Arteries	Tissues Supplied
Thoracic Aorta	
Visceral Branches	
Bronchial	Lung tissue
Esophageal	Esophagus
Parietal Branches	
Intercostal	Thoracic wall
Superior phrenic	Superior surface of diaphragm
Abdominal Aorta	
Visceral Branches	
Unpaired	
Celiac trunk	
Left gastric	Stomach and esophagus
Common hepatic	
Gastroduodenal	Stomach and duodenum
Right gastric	Stomach
Hepatic	Liver
Splenic	Spleen and pancreas
Left gastroepiploic	Stomach
Superior mesenteric	Pancreas, small intestine, and colon
Inferior mesenteric	Descending colon and rectum
Paired	
Suprarenal	Adrenal gland
Renal	Kidney
Gonadal	
Testicular (male)	Testis and ureter
Ovarian (female)	Ovary, ureter, and uterine tube
Parietal Branches	
Inferior phrenic	Adrenal gland and inferior surface of diaphragm
Lumbar	Lumbar vertebrae and back muscles
Median sacral	Inferior vertebrae
Common iliac	
External iliac	Lower limb (see table 21.5)
Internal iliac	Lower back, hip, pelvis, urinary bladder, vagina, uterus, rectum, and external genitalia (see table 21.4)

parietal branches supplying the thoracic wall (table 21.3 and figure 21.12*a* and *b*). The visceral branches supply the lungs, esophagus, and pericardium. Even though a large quantity of blood flows through the lungs, the lung tissue requires a separate oxygenated blood supply from the left ventricle through small bronchial branches from the thoracic aorta.

The walls of the thorax are supplied with blood by the **intercostal** (in-ter-kos′tăl; between the ribs) **arteries,** which consist of two sets: the anterior intercostals and the posterior intercostals. The **anterior intercostals** are derived from the **internal thoracic arteries,** which are branches of the subclavian arteries and lie on the inner surface of the anterior thoracic wall (see table 21.3 and figure 21.12*a* and *b*). The **posterior intercostals** are derived as bilateral branches directly from the descending aorta. The anterior and posterior intercostal arteries lie along the inferior margin of each rib and anastomose with each other approximately midway between the ends of the ribs. **Superior phrenic** (fren′ik; to the diaphragm) **arteries** supply blood to the diaphragm.

Abdominal Aorta and Its Branches

The branches of the abdominal aorta, like those of the thoracic aorta, are divided into visceral and parietal parts (table 21.3 and figures 21.12*a* and *c* and 21.13). The visceral arteries are in turn divided into paired and unpaired branches. Three major unpaired branches exist: the **celiac** (sē′lē-ak; belly) **trunk,** the **superior mesenteric artery** (mez-en-ter′ik; relating to the mesenteries), and the **inferior mesenteric artery** (see figure 21.12*a* and *c*). Each has several major branches supplying the abdominal organs.

The paired visceral branches of the abdominal aorta supply the kidneys, adrenal glands, and gonads (testes or ovaries). The parietal arteries of the abdominal aorta supply the diaphragm and abdominal wall. The arteries of the abdomen and the areas they supply are shown schematically in figure 21.13.

Arteries of the Pelvis

The abdominal aorta divides at the level of the fifth lumbar vertebra into two **common iliac arteries.** They divide to form the **external iliac arteries,** which enter the lower limbs, and the **internal iliac arteries,** which supply the pelvic area. Visceral branches supply the pelvic organs, such as the urinary bladder, rectum, uterus, and vagina; and parietal branches supply blood to the walls and floor of the pelvis; the lumbar, gluteal, and proximal thigh muscles; and the external genitalia (table 21.4 and figures 21.13 and 21.14).

Table 21.4 Arteries of the Pelvis (see figures 21.13 and 21.14)

Arteries	Tissues Supplied
Internal Iliac	Pelvis through the branches listed below
Visceral Branches	
Middle rectal	Rectum
Vaginal	Vagina and uterus
Uterine	Uterus, vagina, uterine tube, and ovary
Parietal Branches	
Lateral sacral	Sacrum
Superior gluteal	Muscles of the gluteal region
Obturator	Pubic region, deep groin muscles, and hip joint
Internal pudendal	Rectum, external genitalia, and floor of pelvis
Inferior gluteal	Inferior gluteal region, coccyx, and proximal thigh

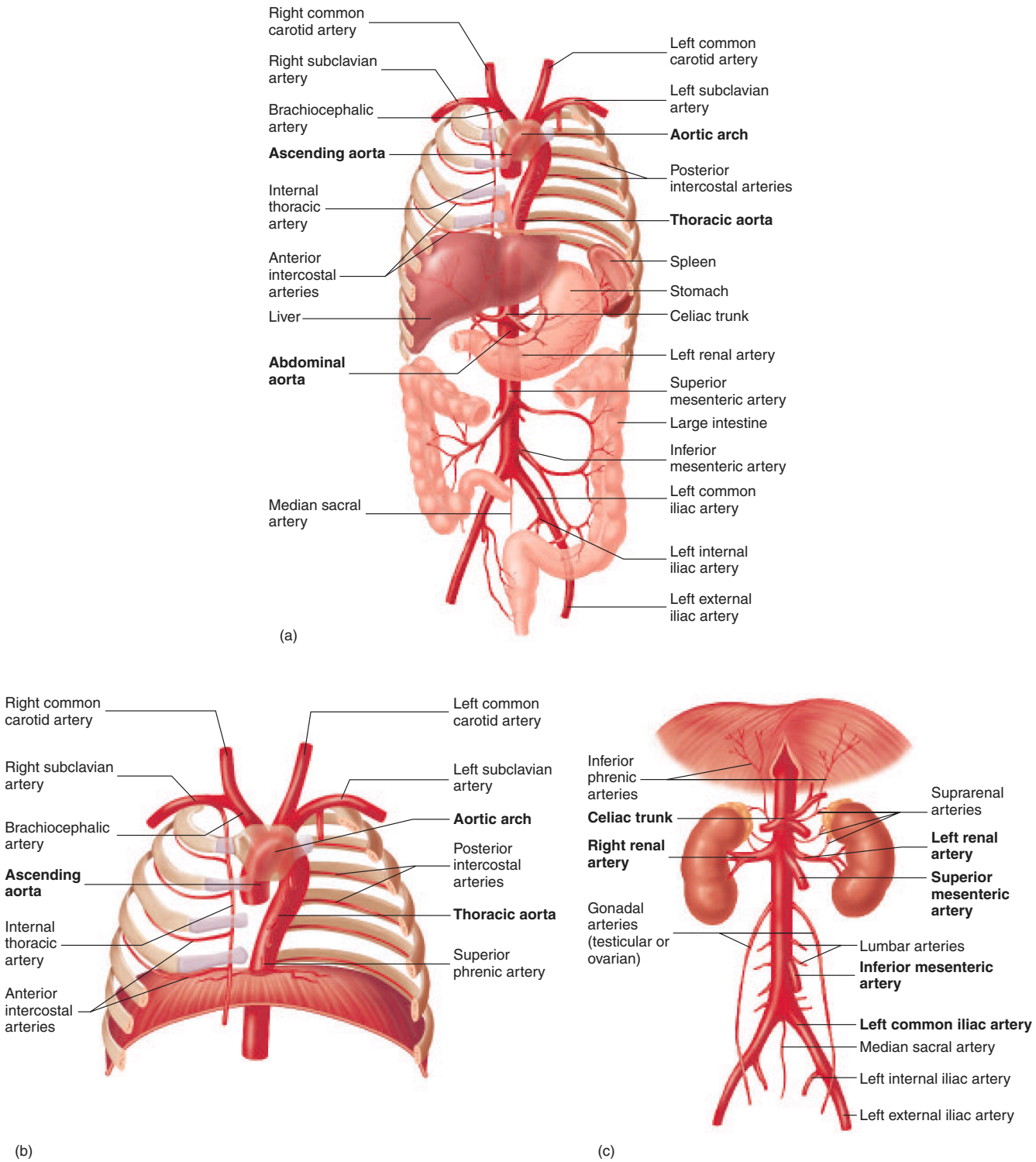


Figure 21.12 Branches of the Aorta

(a) The aorta is considered in three portions: the ascending aorta, the aortic arch, and the descending aorta. The descending aorta consists of the thoracic and abdominal aorta. *(b)* The thoracic. *(c)* The abdominal aorta.

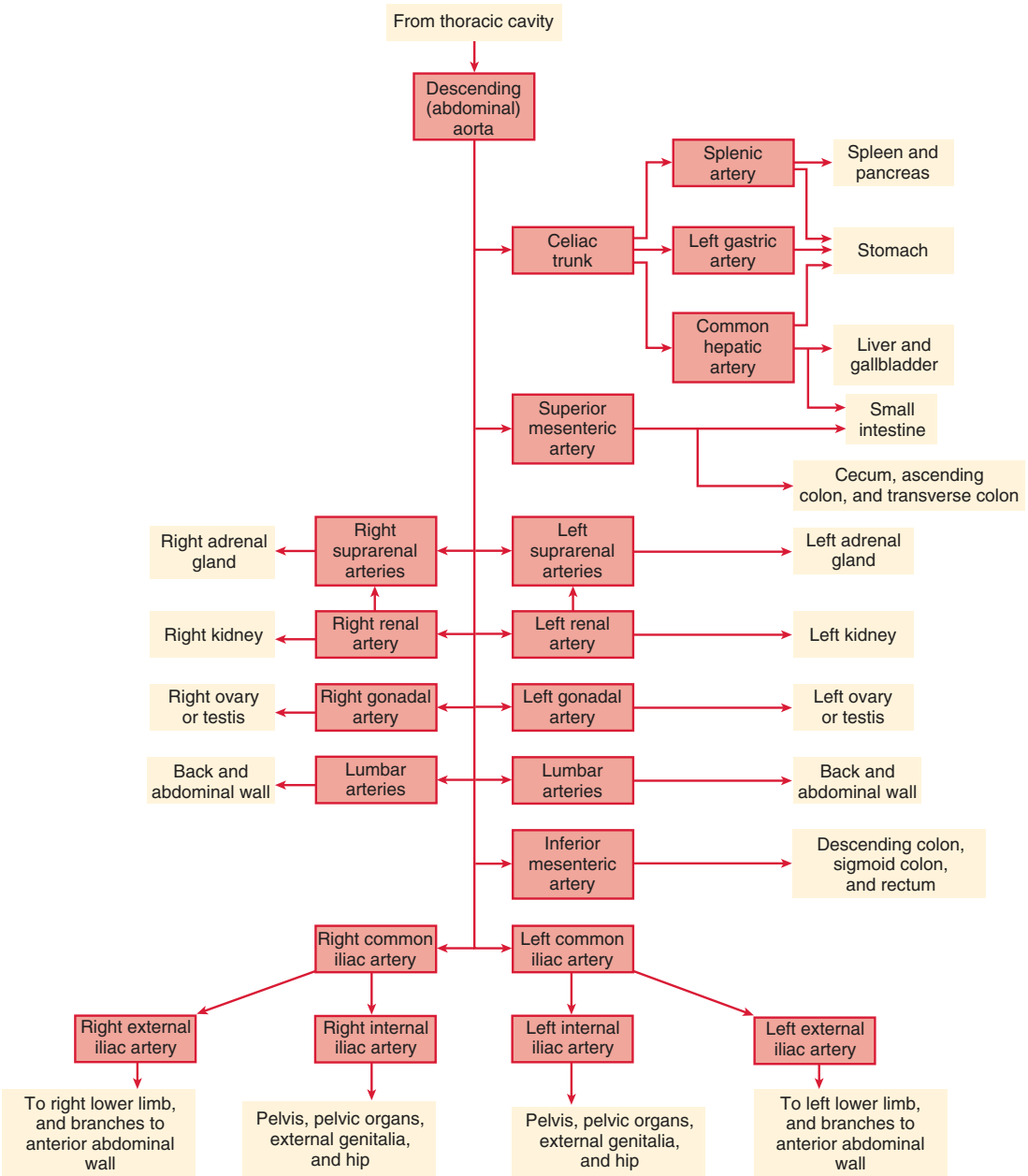


Figure 21.13 Major Arteries of the Abdomen and Pelvis

Arteries of the Lower Limb

The arteries of the lower limb form a continuum similar to that of the arteries of the upper limb. The **external iliac artery** becomes the **femoral** (fem′ŏ-răl; relating to the thigh) **artery** in the thigh, which becomes the **popliteal** (pop-lit′ē-ăl, pop-li-tē′ăl; ham, the hamstring area posterior to the knee) **artery** in the popliteal space. The popliteal artery gives off the **anterior tibial artery** just inferior to the knee and then continues as the **posterior tibial artery.** The anterior tibial artery becomes the **dorsalis pedis artery** at the foot. The posterior tibial artery gives off the **fibular,** or **peroneal, artery** and then gives rise to **medial** and **lateral plantar** (plan′tăr; the sole of the foot) **arteries,** which in turn give off **digital branches** to the toes. The arteries of the lower limb are listed in table 21.5 and illustrated in figures 21.14 and 21.15.

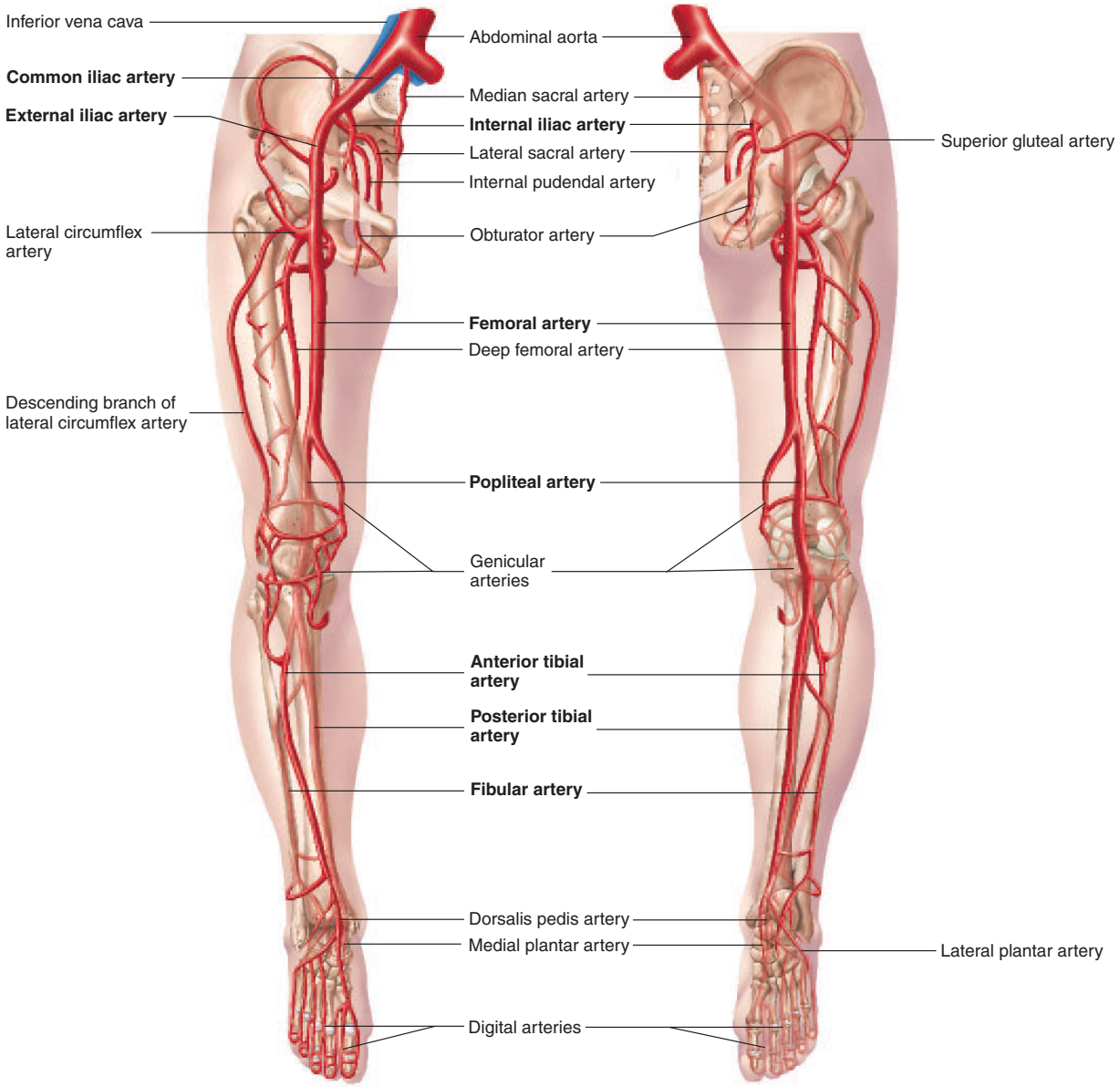


Figure 21.14 Arteries of the Pelvis and Lower Limb

The internal and external iliac arteries and their branches are shown. The internal iliac artery supplies the pelvis and hip, and the external iliac artery supplies the lower limb through the femoral artery.

12. *Name the different parts of the aorta. Name the major arteries that branch from the aorta to supply the heart, the head and upper limbs, and the lower limbs.*
13. *List the arteries that are part of the cerebral arterial circle.*
14. *List, in order, the arteries that travel from the aorta to the digits of the upper limbs.*
15. *Name the two types of branches arising from the thoracic aorta. What structures are supplied by each group?*
16. *What areas of the body are supplied by the paired arteries that branch from the abdominal aorta? The unpaired arteries? Name the three major unpaired branches.*
17. *List, in order, the arteries that travel from the aorta to the digits of the lower limbs.*

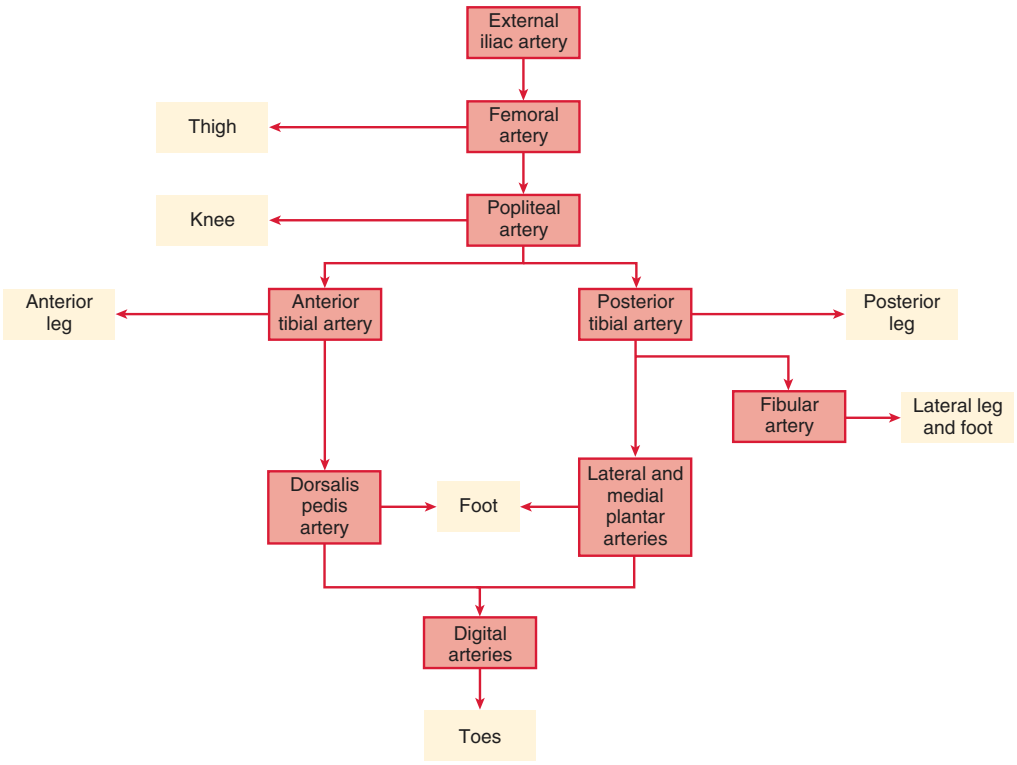


Figure 21.15 Major Arteries of the Lower Limb

Table 21.5 Arteries of the Lower Limb (see figures 21.14 and 21.15)

Arteries	Tissues Supplied
Femoral	Thigh, external genitalia, and anterior abdominal wall
Deep femoral	Thigh, knee, and femur
Popliteal	
(continuation of the femoral artery)	
Posterior tibial	Knee and leg
Fibular (peroneal)	Calf and peroneal muscles and ankle
Medial plantar	Plantar region of foot
Digital arteries	Digits of foot
Lateral plantar	Plantar region of foot
Digital arteries	Digits of foot
Anterior tibial	Knee and leg
Dorsalis pedis	Dorsum of foot
Digital arteries	Digits of foot

Systemic Circulation: Veins

Objective

- *List the major veins that carry blood from each of the major body areas.*

Three major veins return blood from the body to the right atrium: the **coronary sinus,** returning blood from the walls of the heart (see figures 20.6*b* and 20.7); the **superior vena cava** (vē′nă kă′vă, kā′vă; venous cave), returning blood from the head, neck, thorax, and upper limbs; and the **inferior vena cava,** returning blood from the abdomen, pelvis, and lower limbs (figure 21.16).

In a very general way, the smaller veins follow the same course as the arteries and often are given the same names. The veins, however, are more numerous and more variable. The larger veins often follow a very different course and have names different from the arteries.

Three major types of veins exist: superficial veins, deep veins, and sinuses. The superficial veins of the limbs are, in general, larger than the deep veins, whereas in the head and trunk the opposite is the case. Venous sinuses occur primarily in the cranial cavity and the heart.

Veins Draining the Heart

The **cardiac veins,** which transport blood from the walls of the heart and return it through the coronary sinus to the right atrium, are described in chapter 20.

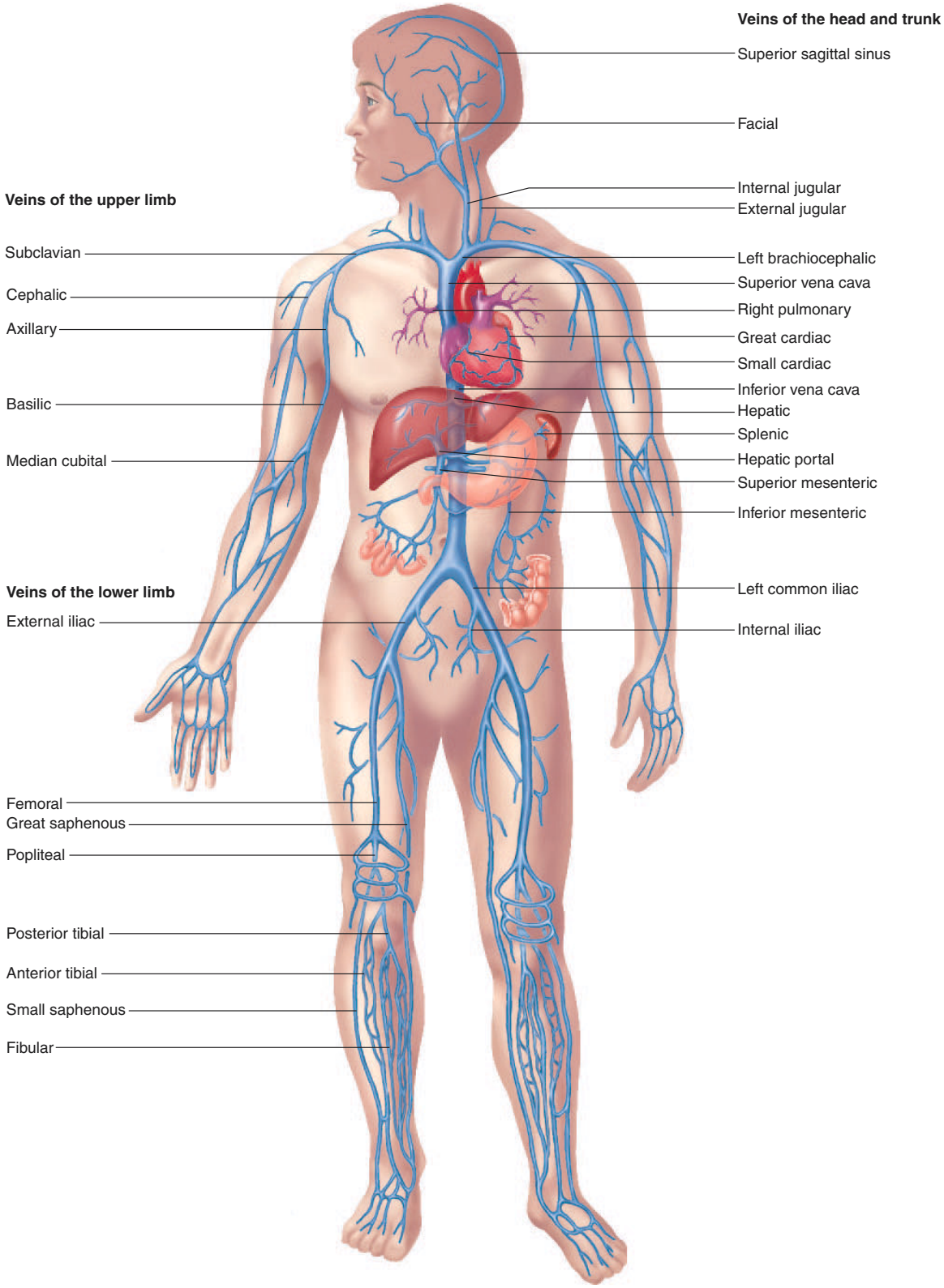


Figure 21.16 The Major Veins

The veins carry blood to the heart from the tissues of the body.

Veins of the Head and Neck

The two pairs of major veins that drain blood from the head and neck are the **external** and **internal jugular** (jŭg′ū-lar; neck) **veins.** The external jugular veins are the more superficial of the two sets, and they drain blood primarily from the posterior head and neck. The external jugular vein usually drains into the subclavian vein. The internal jugular veins are much larger and deeper than the external jugular veins. They drain blood from the cranial cavity and the anterior head, face, and neck.

The internal jugular vein is formed primarily as the continuation of the **venous sinuses** of the cranial cavity. The venous sinuses are actually spaces within the dura mater surrounding the brain (see chapter 13). They are depicted in figure 21.17 and are listed in table 21.6.

Table 21.6 Venous Sinuses of the Cranial Cavity (see figure 21.17)

Veins	Tissues Drained
Internal Jugular Vein	
Sigmoid sinus	
Superior and inferior petrosal sinuses	Anterior portion of cranial cavity
Cavernous sinus	
Ophthalmic veins	Orbit
Transverse sinus	
Occipital sinus	Central floor of posterior fossa of skull
Superior sagittal sinus	Superior portion of cranial cavity and brain
Straight sinus	
Inferior sagittal sinus	Deep portion of longitudinal fissure

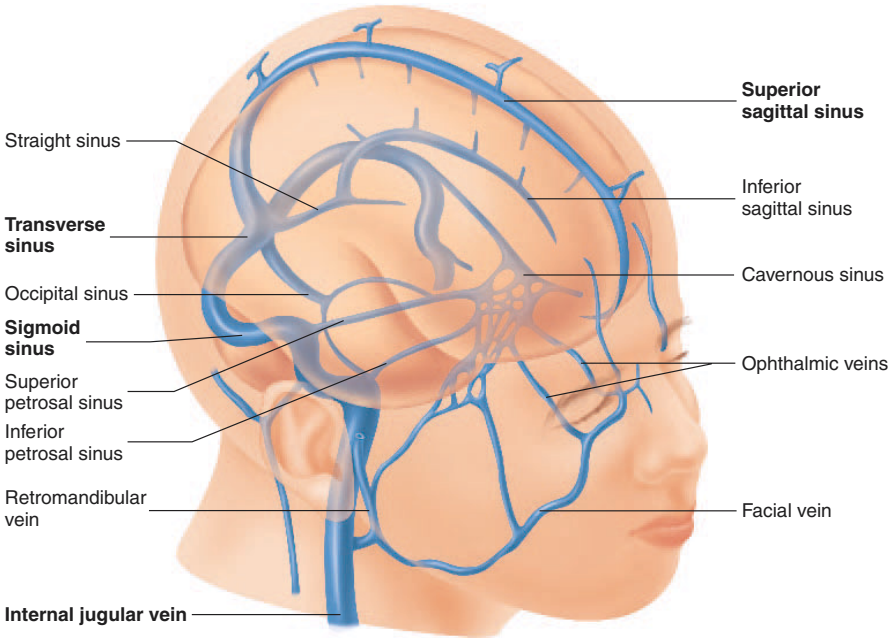


Figure 21.17 Venous Sinuses Associated with the Brain

Facial Pimples and Meningitis

Because venous communication exists between the facial veins and venous sinuses through the ophthalmic veins, infections can potentially be introduced into the cranial cavity through this route. A superficial infection of the face on either side of the nose can enter the facial vein. The infection can then pass through the ophthalmic veins to the venous sinuses and result in meningitis. For this reason people are warned not to aggravate pimples or boils on the face on either side of the nose.

Once the internal jugular veins exit the cranial cavity, they receive several venous tributaries that drain the external head and face (table 21.7 and figures 21.18 and 21.19). The **internal jugular veins** join the **subclavian veins** on each side of the body to form the **brachiocephalic veins.**

Table 21.7 Veins Draining the Head and Neck (see figures 21.18 and 21.19)

Veins	Tissues Drained
Brachiocephalic	
Internal jugular	Brain
Lingual	Tongue and mouth
Superior thyroid	Thyroid and deep posterior facial structures (also empties into external jugular)
Facial	Superficial and anterior facial structures
External jugular	Superficial surface of posterior head and neck

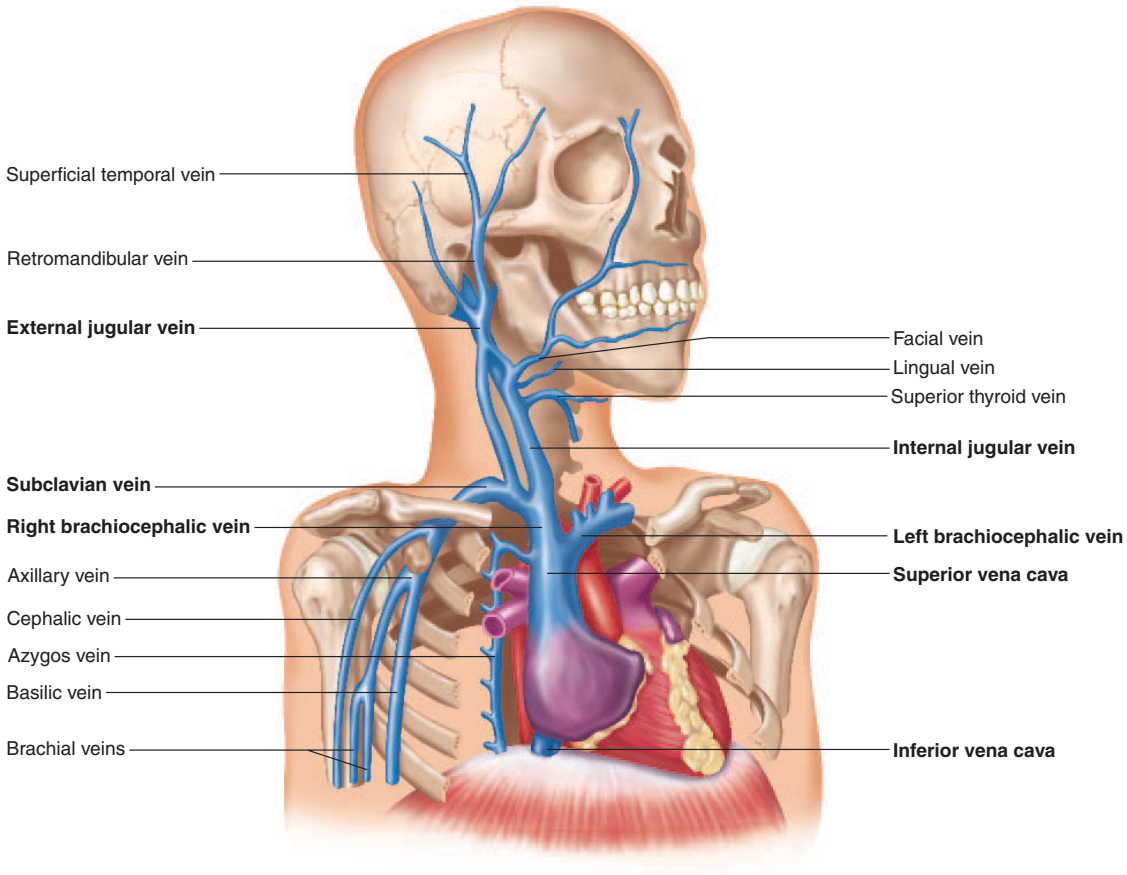


Figure 21.18 Veins of the Head and Neck

The right brachiocephalic vein and its tributaries. The major veins draining the head and neck are the internal and external jugular veins.

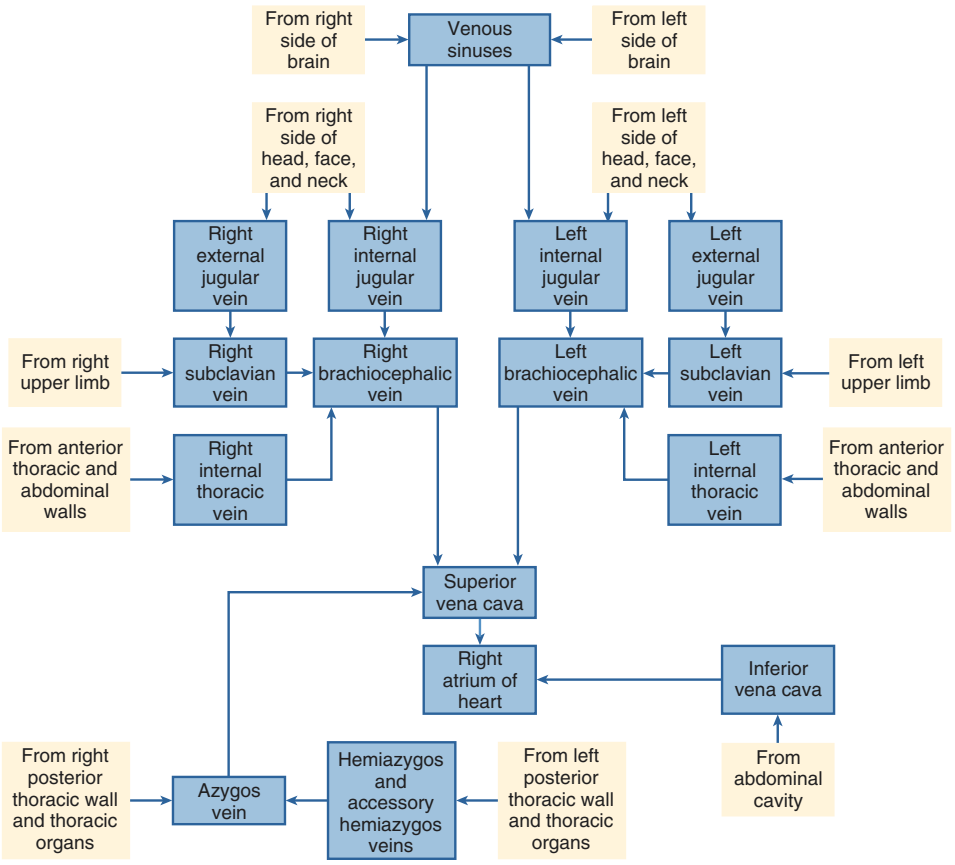


Figure 21.19 Major Veins of the Head and Thorax

Veins of the Upper Limb

The **cephalic** (se-fal′ik; toward the head), **basilic** (ba-sil′ik), and **brachial veins** are responsible for draining most of the blood from the upper limbs (table 21.8 and figures 21.20 and 21.21). Many of the tributaries of the cephalic and basilic veins in the forearm and hand can be seen through the skin. Because of the considerable variation in the tributary veins of the forearm and hand, they often are left unnamed. The basilic vein of the arm becomes the **axillary vein** as it courses through the axillary region. The axillary vein then becomes the **subclavian vein** at the margin of the first rib. The cephalic vein enters the axillary vein.

The **median cubital** (kū′bi-tăl; pertaining to the elbow) **vein** is a variable vein that usually connects the cephalic vein or its tributaries with the basilic vein. In many people, this vein is quite prominent on the anterior surface of the upper limb at the level of the elbow (cubital fossa) and is, therefore, often used as a site for drawing blood from a patient.

The deep veins draining the upper limb follow the same course as the arteries. The **radial** and **ulnar veins,** therefore, are named for the arteries they attend. They usually are paired, with one small vein lying on each side of the artery, and they have numerous connections with one another and with the superficial

Table 21.8 Veins of the Upper Limb (see figures 21.20 and 21.21)

Veins	Tissues Drained
Subclavian	
(continuation of the axillary vein)	
Axillary	
(continuation of the basilic vein)	
Cephalic	Lateral arm, forearm, and hand (superficial veins of the forearm and hand are variable)
Brachial (paired, deep veins)	Deep structures of the arm
Radial vein	Deep forearm
Ulnar vein	Deep forearm
Basilic	Medial arm, forearm, and hand (superficial veins of the forearm and hand are variable)
Median cubital	Connects basilic and cephalic veins
Deep and superficial palmar venous arches	Drain into superficial and deep veins of the forearm
Digital veins	Fingers

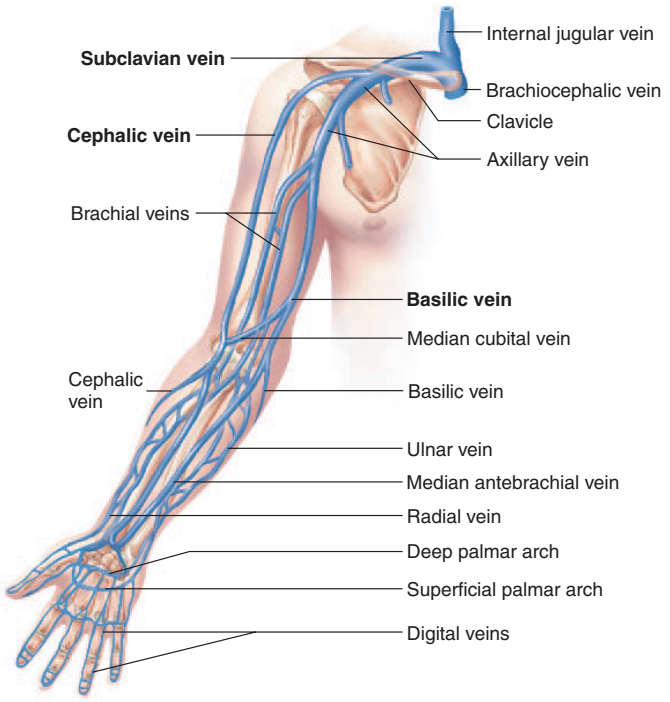


Figure 21.20 Veins of the Upper Limb

The subclavian vein and its tributaries. The major veins draining the superficial structures of the limb are the cephalic and basilic veins. The brachial veins drain the deep structures.

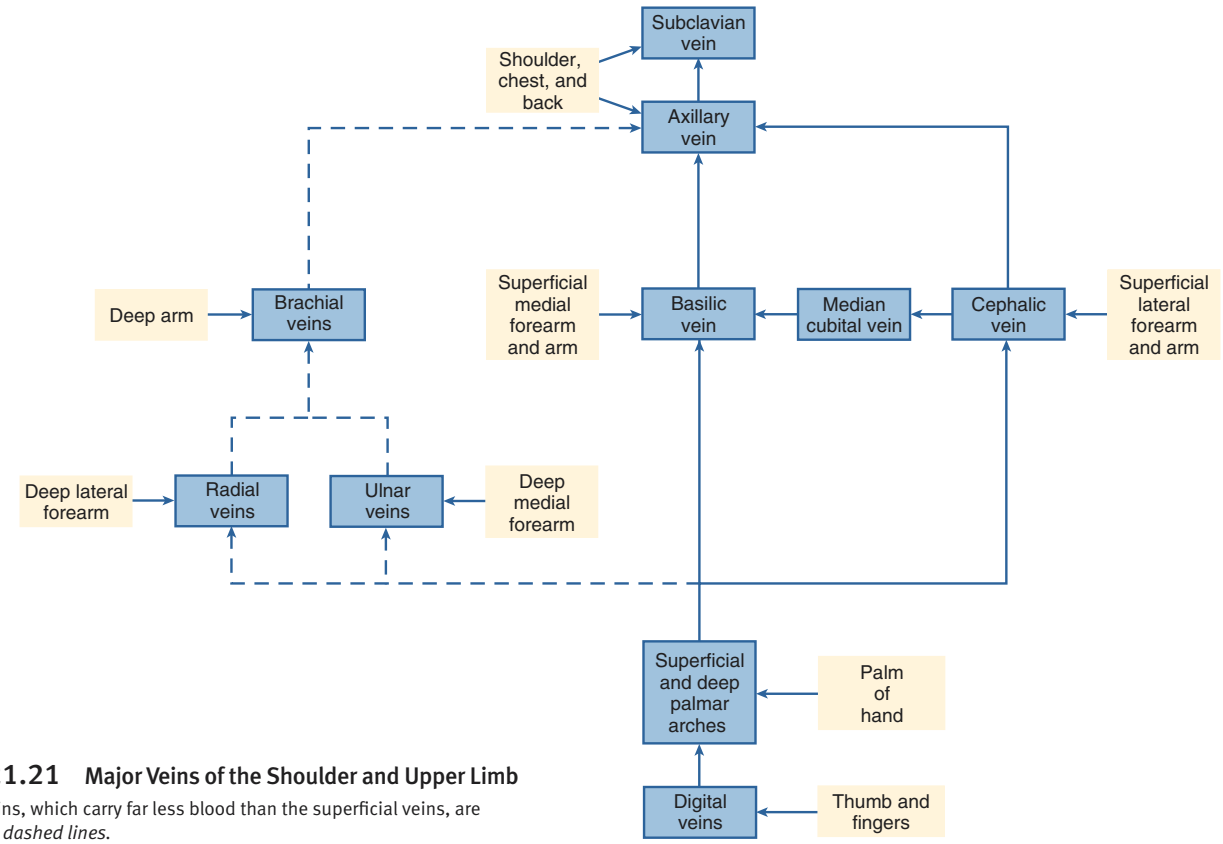


Figure 21.21 Major Veins of the Shoulder and Upper Limb

The deep veins, which carry far less blood than the superficial veins, are indicated by *dashed lines*.

veins. The radial and ulnar veins empty into the **brachial veins,** which accompany the brachial artery and empty into the axillary vein (see figures 21.20 and 21.21).

Veins of the Thorax

Three major veins return blood from the thorax to the superior vena cava: the right and left brachiocephalic veins and the **azygos** (az′ī-gos; unpaired) **vein.** The thoracic drainage to the brachiocephalic veins is through the anterior thoracic wall by way of the **internal thoracic veins.** They receive blood from the **anterior intercostal veins.** Blood from the posterior thoracic wall is collected by **posterior intercostal veins** that drain into the azygos vein on the right and the **hemiazygos** (hem′ē-az′ī-gos) or **accessory hemiazygos vein** on the left. The hemiazygos and accessory hemiazygos veins empty into the azygos vein, which drains into the superior vena cava. The thoracic veins are listed in table 21.9 and illustrated in figure 21.22 (see also figure 21.19).

Table 21.9 Veins of the Thorax (see figure 21.22)

Veins	Tissues Drained
Superior Vena Cava	
Brachiocephalic	
Azygos vein	Right side, posterior thoracic wall and posterior abdominal wall; esophagus, bronchi, pericardium, and mediastinum
Hemiazygos	Left side, inferior posterior thoracic wall and posterior abdominal wall; esophagus and mediastinum
Accessory hemiazygos	Left side, superior posterior thoracic wall

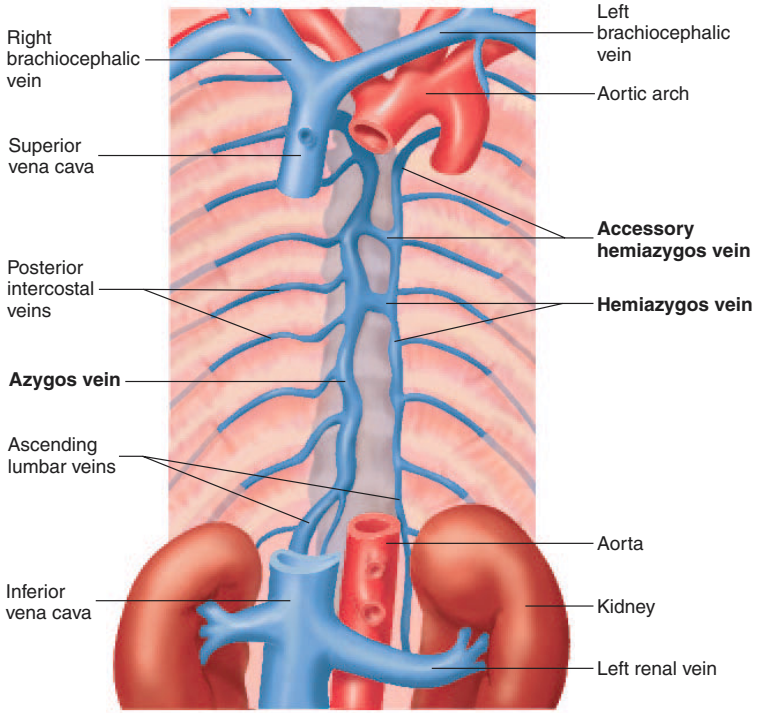


Figure 21.22 Veins of the Thorax

The azygos and hemiazygos veins and their tributaries.

Veins of the Abdomen and Pelvis

Blood from the posterior abdominal wall drains into the **ascending lumbar veins.** These veins are continuous superiorly with the hemiazygos on the left and the azygos on the right. Blood from the rest of the abdomen, pelvis, and lower limbs returns to the heart through the inferior vena cava. The gonads (testes or ovaries), kidneys, and adrenal glands are the only abdominal organs outside the pelvis that drain directly into the inferior vena cava. The **internal iliac veins** drain the pelvis and join the **external iliac veins** from the lower limbs to form the **common iliac veins,** which unite to form the inferior vena cava. The major abdominal and pelvic veins are listed in table 21.10 and illustrated in figures 21.23 and 21.25.

Hepatic Portal System

Blood from the capillaries within most of the abdominal viscera, such as the stomach, intestines, and spleen, drains through a specialized system of blood vessels to the liver. Within the liver, the blood flows through a series of dilated capillaries called **sinusoids.** A **portal** (pōr′tăl; door) **system** is a vascular system

Table 21.10 Veins Draining the Abdomen and Pelvis (see figures 21.23 and 21.25)

Veins	Tissues Drained
Inferior Vena Cava	
Hepatic veins	Liver (see hepatic portal system)
Common iliac	
External iliac	Lower limb (see table 21.12)
Internal iliac	Pelvis and its viscera
Ascending lumbar	Posterior abdominal wall (empties into common iliac, azygos, and hemiazygos veins)
Renal	Kidney
Suprarenal	Adrenal gland
Gonadal	
Testicular (male)	Testis
Ovarian (female)	Ovary
Phrenic	Diaphragm

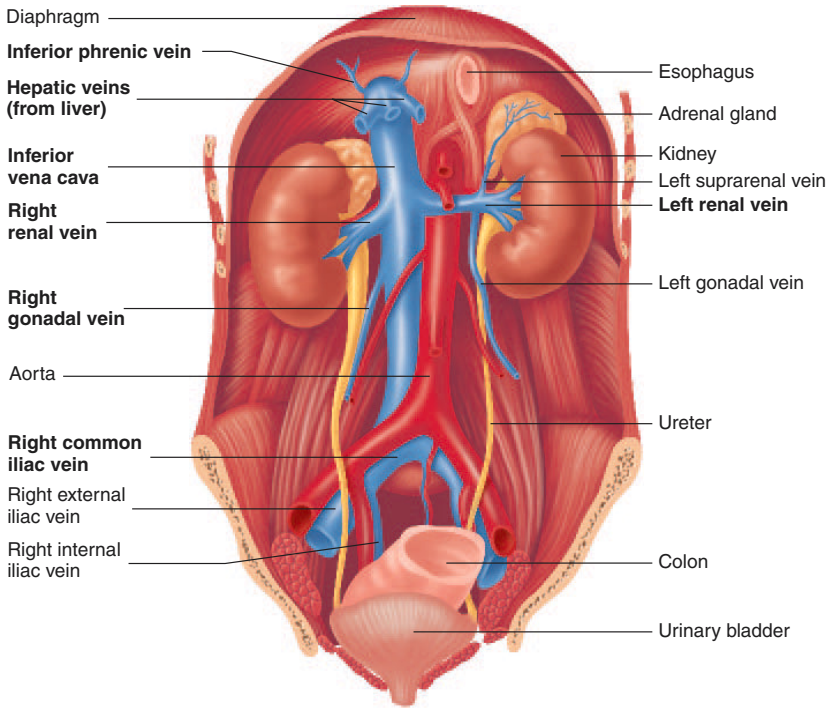


Figure 21.23 Inferior Vena Cava and Its Tributaries

The hepatic veins transport blood to the inferior vena cava from the hepatic portal system, which ends as a series of blood sinusoids in the liver (see figure 21.24).

Table 21.11 Hepatic Portal System (see figures 21.24 and 21.25)

Veins	Tissues Drained
Hepatic Portal	
Superior mesenteric	Small intestine and most of the colon
Splenic	Spleen
Inferior mesenteric	Descending colon and rectum
Pancreatic	Pancreas
Left gastroepiploic	Stomach
Gastric	Stomach
Cystic	Gallbladder

that begins and ends with capillary beds and has no pumping mechanism like the heart between the capillary beds. The portal system that begins with capillaries in the viscera and ends with the sinusoidal capillaries in the liver is the **hepatic** (he-pat′ik; relating to the liver) **portal system** (table 21.11 and figures 21.24 and 21.25). The **hepatic portal vein,** the largest vein of the system, is formed by the union of the **superior mesenteric vein,** which drains the small intestine, and the **splenic vein,** which drains the spleen. The splenic vein receives the **inferior mesenteric vein,** which drains part of the large intestine, and the **pancreatic veins,** which drain the pancreas. The hepatic portal vein also receives gastric veins before entering the liver.

Blood from the liver sinusoids is collected into **central veins,** which empty into **hepatic veins.** Blood from the cystic veins, which drain the gallbladder, also enters the hepatic veins. The hepatic veins join the inferior vena cava. Blood entering the liver through the hepatic portal vein is rich with nutrients collected from the intestines, but it also can contain a number of toxic substances harmful to the tissues of the body. Within the liver, the nutrients are either taken up and stored or are modified chemically and used by other cells of the body (see chapter 24). The cells of the liver also help remove toxic substances by altering their structure or making them water-soluble, a process called **biotransformation.** The water-soluble substances can then be transported in the blood to the kidneys, from which they are excreted in the urine (see chapter 26).

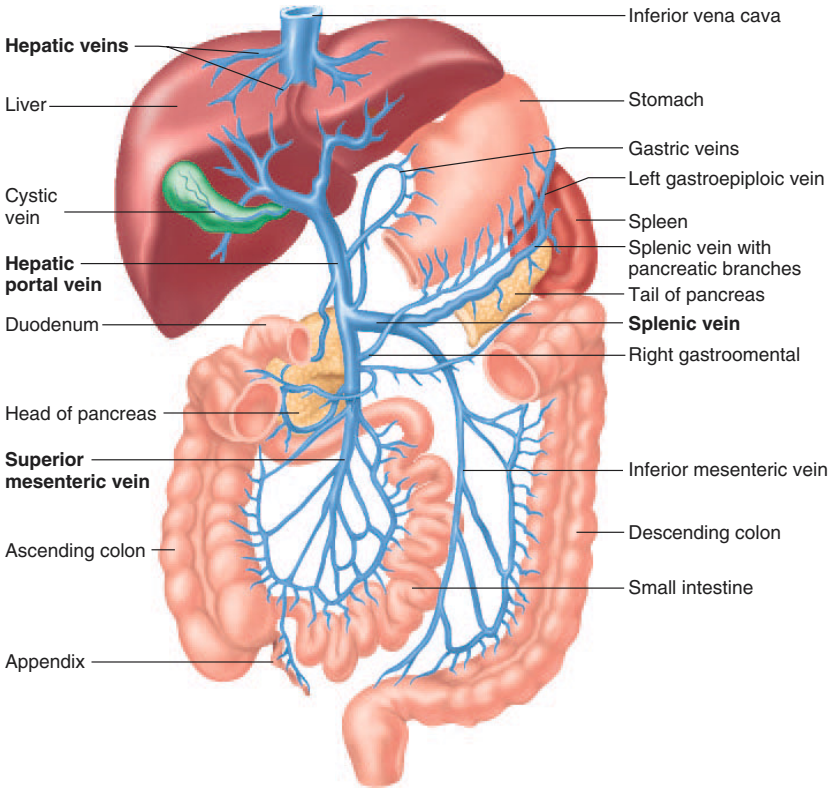


Figure 21.24 Hepatic Portal System

The hepatic portal vein transports blood from most of the abdominal organs to the liver. Hepatic veins drain the liver and enter the inferior vena cava (see figure 21.23).

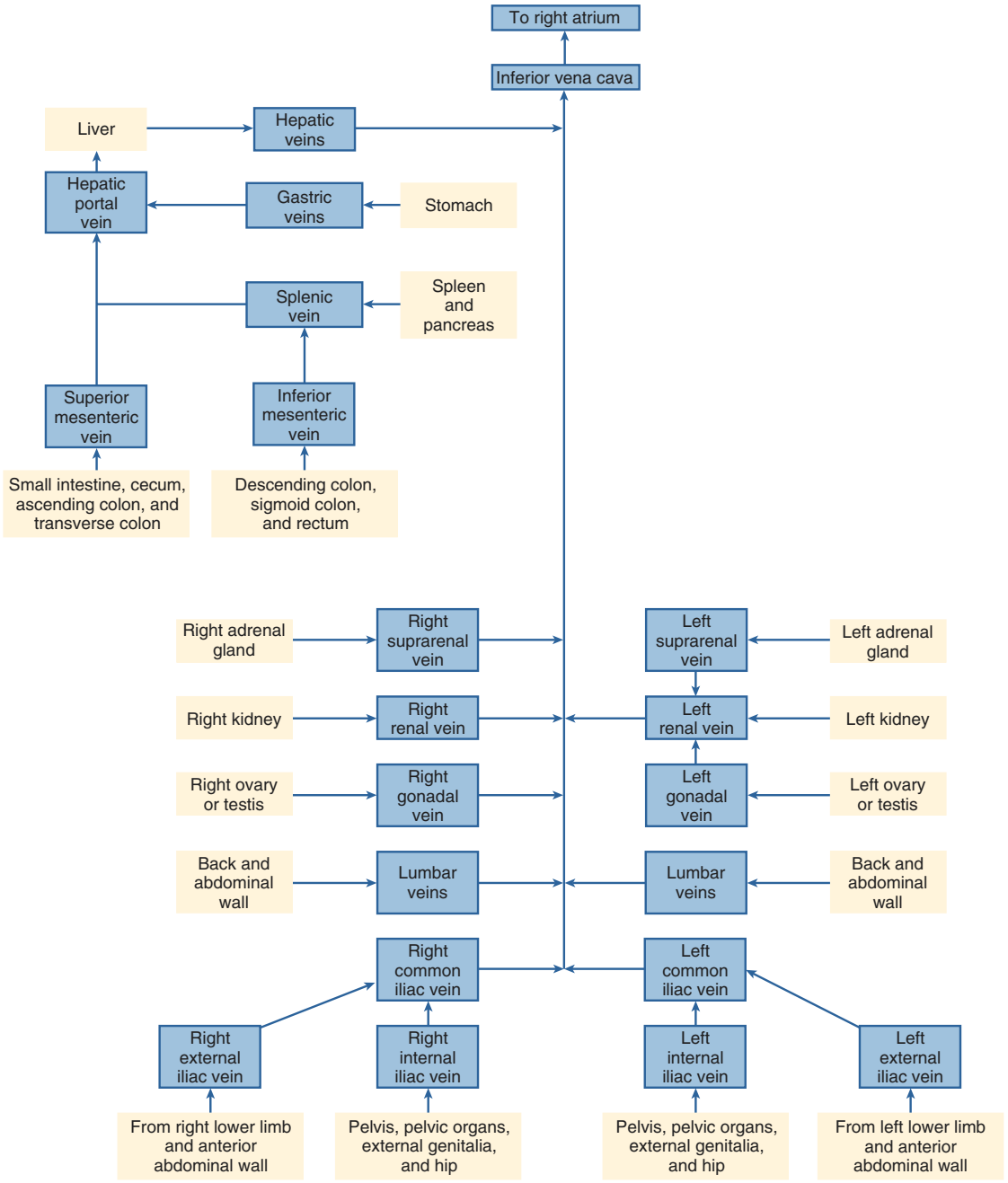


Figure 21.25 Major Veins of the Abdomen and Pelvis

Veins of the Lower Limb

The veins of the lower limb, like those of the upper limb, consist of superficial and deep groups. The distal deep veins of each limb are paired and follow the same path as the arteries, whereas the proximal deep veins are unpaired. The **anterior** and **posterior tibial veins** are paired and accompany the anterior and posterior tibial arteries. They unite just inferior to the knee to form the single **popliteal vein,** which ascends through the thigh and becomes the **femoral vein.** The femoral vein becomes the external iliac vein. **Fibular,** or **peroneal** (per-ō-nē′ăl), **veins** also are paired in each leg and accompany the fibular arteries. They empty into the posterior tibial veins just before those veins contribute to the popliteal vein.

The superficial veins consist of the great and small saphenous veins. The **great saphenous** (să-fē′nŭs; visible) **vein,** the longest vein of the body, originates over the dorsal and medial side of the foot and ascends along the medial side of the leg and thigh to empty into the femoral vein. The **small saphenous vein** begins over the lateral side of the foot and ascends along the posterior leg to the popliteal space, where it empties into the popliteal vein. The veins of the lower limb are illustrated in figures 21.26 and 21.27 and are listed in table 21.12.

18. *Name the three major vessels that return blood to the heart. What areas of the body do they drain?*
19. *List the two pairs of major veins that drain blood from the head and neck. Describe the venous sinuses. To what large vein do the venous sinuses connect?*
20. *List the three major veins that return blood from the thorax to the superior vena cava.*
21. *Explain the three ways that blood from the abdomen returns to the heart.*

Table 21.12 Veins of the Lower Limb (see figures 21.26 and 21.27)

Veins	Tissues Drained
External Iliac Vein	
(continuation of the femoral vein)	
Femoral	Thigh
(continuation of the popliteal vein)	
Popliteal	
Anterior tibial	Deep anterior leg
Dorsal vein of foot	Dorsum of foot
Posterior tibial	Deep posterior leg
Plantar veins	Plantar region of foot
Fibular (peroneal)	Deep lateral leg and foot
Small saphenous	Superficial posterior leg and lateral side of foot
Great saphenous	Superficial anterior and medial leg, thigh, and dorsum of foot
Dorsal vein of foot	Dorsum of foot
Dorsal venous arch	Foot
Digital veins	Toes

22. *List the vessels that carry blood from abdominal organs to the hepatic portal vein.*
23. *List the major deep and superficial veins of the upper and lower limbs.*

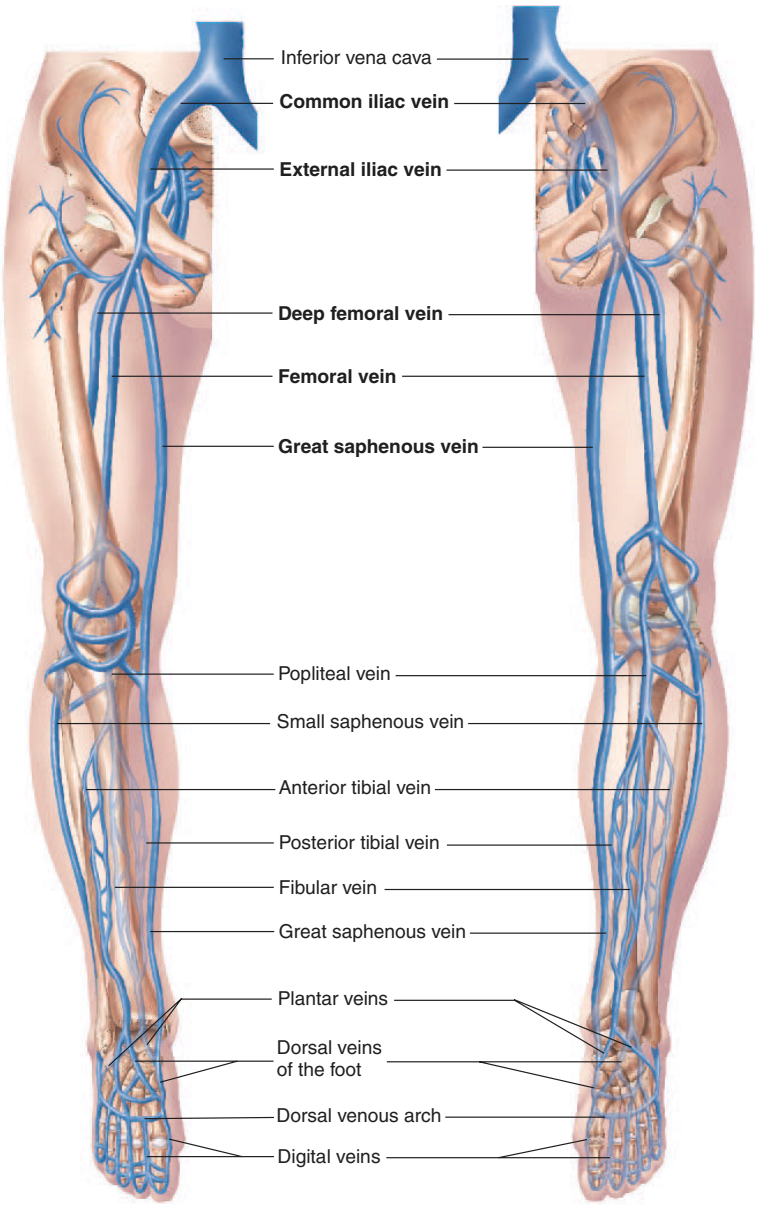


Figure 21.26 Veins of the Pelvis and Lower Limb

The right common iliac vein and its tributaries.

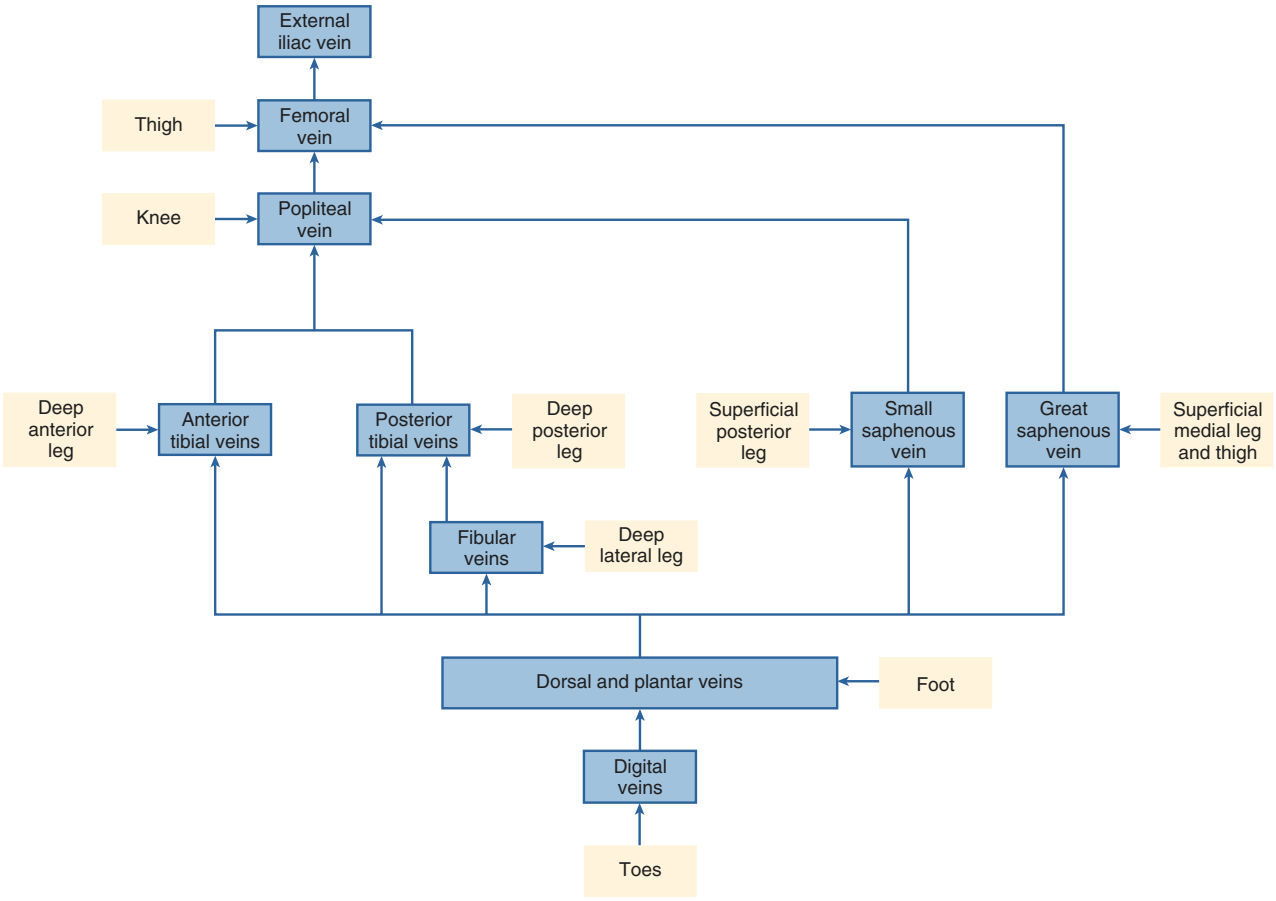


Figure 21.27 Major Veins of the Lower Limb

Dynamics of Blood Circulation

Objectives

- ***Describe the significance of each of the following for the circulation of blood: viscosity, laminar and turbulent flow, blood pressure, rate of blood flow, Poiseuille's law, critical closing pressure, Laplace's law, and vascular compliance.***
- ***Explain how blood pressure can be measured.***

Dynamics of blood circulation through blood vessels are the same as those affecting the flow of water or liquids through pipes. The interrelationships between pressure, flow, resistance, and the control mechanisms that regulate blood pressure and blood flow through vessels play a critical role in the function of the circulatory system.

Laminar and Turbulent Flow in Vessels

Fluid, including blood, tends to flow through long, smooth-walled tubes in a streamlined fashion called **laminar flow** (figure 21.28*a*). Fluid behaves as if it were composed of a large number of concentric layers. The layer nearest the wall of the tube experiences the greatest resistance to flow because it moves against the stationary wall. The innermost layers slip over the surface of the outermost layers and experience less resistance to movement. Thus, flow in a vessel consists of movement of concentric layers, with the outermost layer moving slowest and the layer at the center moving fastest.

Laminar flow is interrupted and becomes **turbulent flow** when the rate of flow exceeds a critical velocity or when the fluid passes a constriction, a sharp turn, or a rough surface (figure 21.28*b*). Vibrations of the liquid and blood vessel walls during turbulent flow cause the sounds produced when blood pressure is measured using a blood pressure cuff. Turbulent flow is also common as blood flows past the valves in the heart and is partially responsible for the heart sounds (see chapter 20).

Turbulent flow of blood through vessels occurs primarily in the heart and to a lesser extent where arteries branch. Sounds caused by turbulent blood flow in arteries are not normal and usually indicate that the blood vessel is constricted abnormally. In addition, turbulent flow in abnormally constricted arteries increases the probability that thromboses will develop in the area of turbulent flow.

Blood Pressure

Blood pressure is a measure of the force blood exerts against blood vessel walls. The standard instrument for measuring blood pressure is the **mercury (Hg) manometer,** which measures pressure in

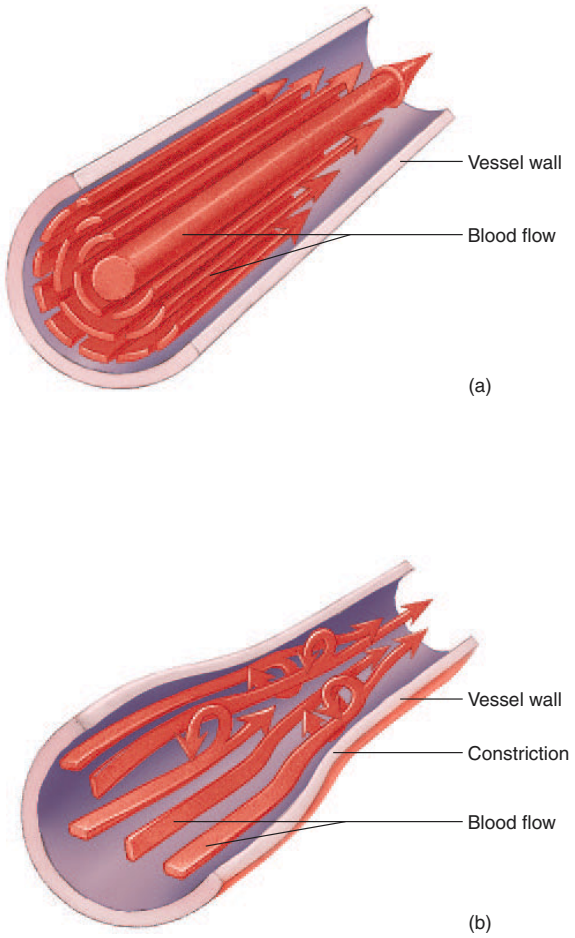


Figure 21.28 Laminar and Turbulent Flow

(a) In laminar flow, fluid flows in long smooth-walled tubes as if it is composed of a large number of concentric layers. *(b)* Turbulent flow is caused by numerous small currents flowing crosswise or obliquely to the long axis of the vessel, resulting in flowing whorls and eddy currents.

millimeters of mercury (mm Hg). If the blood pressure is 100 mm Hg, the pressure is great enough to lift a column of mercury 100 mm.

Blood pressure is measured directly by inserting a **cannula** (or tube) into a blood vessel and connecting a manometer or an electronic pressure transducer to it. Electronic transducers are very sensitive to changes in pressure and can precisely detect rapid fluctuation in pressure.

Placing catheters in blood vessels or in chambers of the heart to monitor pressure changes is possible, but these procedures are not appropriate for routine clinical determinations of systemic blood pressure. The **auscultatory** (aws-kŭl′tă-tō′rē) **method** can be used to measure blood pressure without surgical procedures or causing discomfort, so it's used under most clinical conditions. A blood pressure cuff connected to a **sphygmomanometer** (sfig′mō-mă-nom′ĕ-ter) is placed around the patient's arm just above the elbow, and a stethoscope is placed over the brachial artery (figure 21.29). Some sphygmomanometers have mercury manometers, and others have digital manometers, but they all measure pressure in terms of millimeters of mercury. The blood pressure cuff is inflated until the brachial artery is completely collapsed. Because no blood flows through the constricted area, no sounds can be heard. The pressure in the cuff is gradually lowered. As soon as it declines below the systolic pressure, blood flows through the constricted area during systole. The blood flow is turbulent and produces vibrations in the blood and surrounding tissues that can be heard through the stethoscope. These sounds are called **Korotkoff** (kō-rot′kof) **sounds,** and the pressure at which a Korotkoff sound is first heard represents the **systolic pressure.**

As the pressure in the blood pressure cuff is lowered still more, the Korotkoff sounds change tone and loudness. When the pressure has dropped until continuous laminar blood flow is reestablished, the sound disappears completely. The pressure at which continuous laminar flow is reestablished is the **diastolic pressure.** This method for determining systolic and diastolic pressures is not entirely accurate, but its results are within 10% of methods that are more direct.

Blood Flow

The **rate** at which blood or any other liquid flows through a tube can be expressed as the volume that passes a specific point per unit of time. Blood flow usually is reported in either milliliters (mL) per minute or liters (L) per minute. For example, when a person is resting, the **cardiac output** of the heart is approximately 5 L/min; thus blood flow through the aorta is approximately 5 L/min.

Blood flow in a vessel is proportional to the pressure difference in that vessel. For example, if the pressures at point 1 (P_1) and point 2 (P_2) in a vessel are the same, no flow occurs. If, however, the pressure at P_1 is greater than that at P_2, flow proceeds from P_1 toward P_2, and the greater the pressure difference, the greater is the rate of flow. If P_2 is greater than P_1, flow proceeds from P_2 toward P_1. Flow always occurs from a higher to a lower pressure.

The flow of blood resulting from a pressure difference in a vessel is opposed by a **resistance (*R*)** to blood flow. As the resistance increases, blood flow decreases, and as the resistance decreases, blood flow increases.

The effect of pressure differences and resistance to blood flow can be expressed mathematically.

$$\text{Flow} = \frac{P_1 - P_2}{R}$$

Poiseuille's Law

Several factors affect resistance to blood flow and are expressed individually in **Poiseuille's** (pwah-zuh′yez) **law.**

Poiseuille's law is expressed by the following formula:

$$\text{Flow} = \pi(P_1 - P_2)/8vl/r^4 \quad \text{or} \quad \text{Flow} = \pi(P_1 - P_2)r^4/8vl$$

where v is viscosity of blood, l is length of the vessel, P is pressure, and r is blood vessel radius. The value $\pi/8$ is a constant and does not change in value.

According to Poiseuille's law, flow decreases when resistance increases. Resistance to flow dramatically decreases when blood vessel diameter increases because flow is proportional to the fourth power of the blood vessel's radius. On the other hand, an increase in resistance caused by a small decrease in the blood vessel's radius

1. No sound is heard because there is no blood flow when the cuff pressure is high enough to keep the brachial artery closed.
2. **Systolic pressure** is the pressure at which a Korotkoff sound is first heard. When cuff pressure decreases and is no longer able to keep the brachial artery closed during systole, blood is pushed through the partially opened brachial artery to produce turbulent blood flow and a sound. The brachial artery remains closed during diastole.
3. As cuff pressure continues to decrease, the brachial artery opens even more during systole. At first, the artery is closed during diastole, but as cuff pressure continues to decrease, the brachial artery partially opens during diastole. Turbulent blood flow during systole produces Korotkoff sounds, although the pitch of the sounds change as the artery becomes more open.
4. **Diastolic pressure** is the pressure at which the sound disappears. Eventually cuff pressure decreases below the pressure in the brachial artery and it remains open during systole and diastole. Nonturbulent flow is reestablished and no sounds are heard.

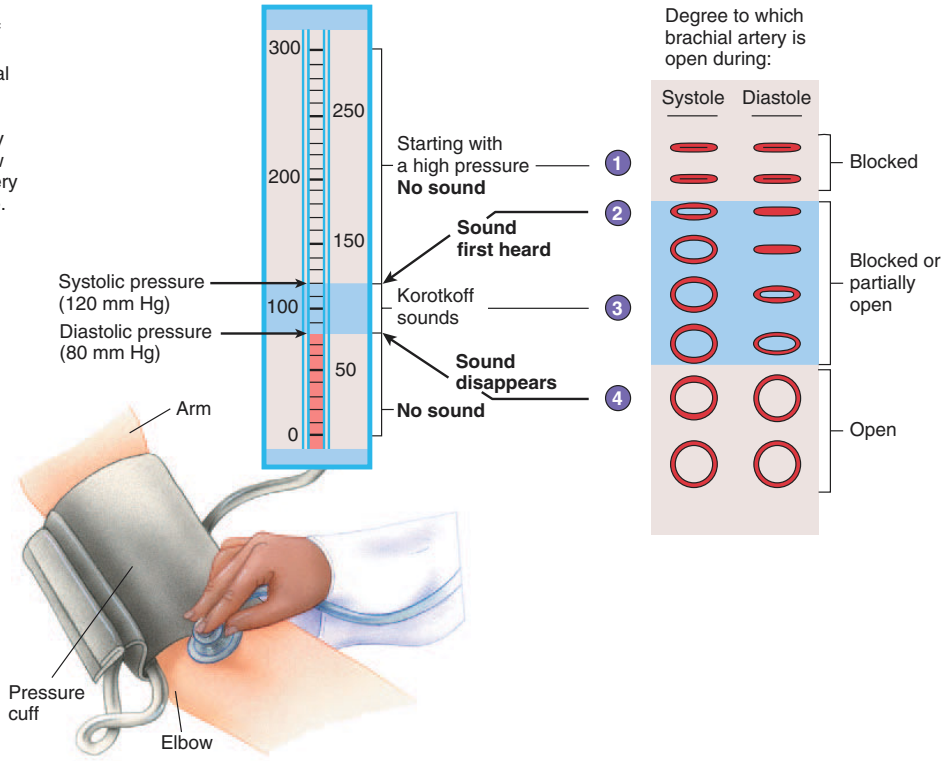


Figure 21.29 Blood Pressure Measurement

results in a dramatic decrease in flow. In addition, either an increase in blood viscosity (see the following section on "Viscosity") or an increase in blood vessel length reduces flow.

During exercise, the heart contracts with greater force, and blood pressure increases in the aorta. In addition, blood vessels in skeletal muscles dilate, thereby making their radii larger and the resistance to blood flow smaller. As a consequence, the rate of flow increases from 5 L/min in the aorta to several times that value.

Viscosity

Viscosity (vis-kos′i-tē) is a measure of the resistance of a liquid to flow. As the viscosity of a liquid increases, the pressure required to force it to flow increases. A common means for reporting the viscosity of liquids is to consider the viscosity of distilled water as 1 and to compare the viscosity of other liquids to it. Using this procedure, whole blood has a viscosity of 3.0–4.5, which means that about three times as much pressure is required to force whole blood to flow through a given tube at the same rate as water.

The viscosity of blood is influenced largely by **hematocrit** (hē′mă-tō-krit, hem′ă-tō-krit), which is the percentage of the total blood volume composed of red blood cells (see chapter 19). As the hematocrit increases, the viscosity of blood increases logarithmically. Blood with a hematocrit of 45% has a viscosity about three times that of water, whereas blood with a very high hematocrit of 65% has a viscosity about seven to eight times that of water. The plasma proteins have only a minor effect on the viscosity of blood. Dehydration or uncontrolled production of erythrocytes can

increase hematocrit and the viscosity of blood substantially. Viscosity above its normal range of values increases the workload on the heart, and, if this workload is great enough, heart failure can result.

PREDICT 2

Predict the effect of each of the following conditions on blood flow: (a) vasoconstriction of blood vessels in the skin in response to cold exposure, (b) vasodilation of the blood vessels in the skin in response to an elevated body temperature, (c) polycythemia vera, which results in a greatly increased hematocrit.

Critical Closing Pressure and Laplace's Law

Each blood vessel exhibits a **critical closing pressure,** the pressure below which the vessel collapses and blood flow through the vessel stops. Under conditions of shock, blood pressure can decrease below the critical closing pressure in vessels (see the Clinical Focus on "Shock," p. 760). As a consequence, the blood vessels collapse, and flow ceases. Tissues supplied by those vessels can become necrotic because of the lack of blood supply.

Laplace's (la-plas′ez) **law** states that the force that stretches the vascular wall is proportional to the diameter of the vessel times the blood pressure. Laplace's law helps explain the critical closing pressure. As the pressure in a vessel decreases, the force that stretches the vessel wall also decreases. Some minimum force is required to keep the vessel open. If the pressure decreases so that the force is below that minimum requirement, the vessel will close. As the pressure in a vessel increases, the force that stretches the vessel wall also increases.

Laplace's law is expressed by the following formula:

$$F = D \times P$$

where F is force, D is vessel diameter, and P is pressure.

According to Laplace's law, as the diameter of a vessel increases, the force applied to the vessel wall increases, even if the pressure remains constant. If a part of an arterial wall becomes weakened so that a bulge forms in it, the force applied to the weakened part is greater than at other points along the blood vessel because its diameter is greater. The greater force causes the weakened vessel wall to bulge even more, further increasing the force applied to it. This series of events can proceed until the vessel finally ruptures. As the bulges in weakened blood vessel walls, called **aneurysms,** enlarge, the danger of their rupturing increases. Ruptured aneurysms in the blood vessels of the brain or in the aorta often result in death.

Vascular Compliance

Compliance (kom-pli′ans) is the tendency for blood vessel volume to increase as the blood pressure increases. The more easily the vessel wall stretches, the greater is its compliance. The less easily the vessel wall stretches, the smaller is its compliance.

Table 21.13 Distribution of Blood Volume in Blood Vessels

Vessels		Total Blood Volume (%)
Systemic		
Veins		64
Large veins	(39%)	
Small veins	(25%)	
Arteries		15
Large arteries	(8%)	
Small arteries	(5%)	
Arterioles	(2%)	
Capillaries		5
	TOTAL IN SYSTEMIC VESSELS	84
Pulmonary vessels		9
Heart		7
	TOTAL BLOOD VOLUME	100

Compliance is expressed by the following formula:

$$\text{Compliance} = \frac{\text{Increase in volume (mL)}}{\text{Increase in pressure (mm Hg)}}$$

Vessels with a large compliance exhibit a large increase in volume when the pressure increases a small amount. Vessels with a small compliance do not show a large increase in volume when the pressure increases.

Venous compliance is approximately 24 times greater than the compliance of arteries. As venous pressure increases, the volume of the veins increases greatly. Consequently, veins act as storage areas, or reservoirs, for blood because their large compliance allows them to hold much more blood than other areas of the vascular system (table 21.13).

24. *Describe laminar flow and turbulent flow through a tube. What conditions cause turbulent flow of blood?*
25. *Define the terms blood pressure, blood flow, and resistance. How can each be determined?*
26. *According to Poiseuille's law, what effect do viscosity, blood vessel diameter, and blood vessel length have on resistance? On blood flow?*
27. *Define the term viscosity, and state the effect of hematocrit on viscosity.*
28. *State Laplace's law. How does it explain critical closing pressure and aneurysms?*
29. *Define the term vascular compliance. Do veins or arteries have greater compliance?*

Physiology of Systemic Circulation

Objectives

- ***Describe the changes in cross-sectional area, blood pressure, and resistance to flow, starting in the aorta, moving through the vascular system, and returning to the right atrium.***
- ***Define pulse pressure, and list the factors that influence it.***
- ***Describe how the exchange of materials across the capillary occurs, and explain the factors that can cause edema.***
- ***Describe the functional characteristics of veins.***

The anatomy of the circulatory system, the dynamics of blood flow, and the regulatory mechanisms that control the heart and blood vessels determine the physiologic characteristics of the circulatory system. The entire circulatory system functions to maintain adequate blood flow to all tissues.

Approximately 84% of the total blood volume is contained in the systemic circulatory system. Most of the blood volume is in the veins, which are the vessels with the greatest compliance. Smaller volumes of blood are in the arteries and capillaries (see table 21.13).

Cross-Sectional Area of Blood Vessels

If the cross-sectional area of each blood vessel type is determined and multiplied by the number of each type of blood vessel, the result is the total cross-sectional area for each blood vessel type. For example, only one aorta exists, and it has a cross-sectional area of 5 square centimeters (cm^2). On the other hand, millions of capillaries exist, and each has a very small cross-sectional area. The total cross-sectional area of all capillaries, however, is 2500 cm^2, which is much greater than the cross-sectional area of the aorta (figure 21.30).

The velocity of blood flow is greatest in the aorta, but the total cross-sectional area is small. In contrast, the total cross-sectional area for the capillaries is large, but the velocity of blood flow is low. As the veins become larger in diameter, their total cross-sectional area decreases, and the velocity of blood flow increases. The relationship between blood vessel diameter and velocity of blood flow is much like a stream that flows rapidly through a narrow gorge but flows slowly through a broad plane (see figure 21.30).

Pressure and Resistance

The left ventricle of the heart forcefully ejects blood from the heart into the aorta. Because the pumping action of the heart is pulsatile, the aortic pressure fluctuates between a systolic pressure of 120 mm Hg and a diastolic pressure of 80 mm Hg (table 21.14 and figure 21.31). As blood flows from arteries through the capillaries and the veins, the pressure falls progressively to approximately 0 mm Hg or even slightly lower by the time it returns to the right atrium.

The decrease in arterial pressure in each part of the systemic circulation is directly proportional to the resistance to blood flow.

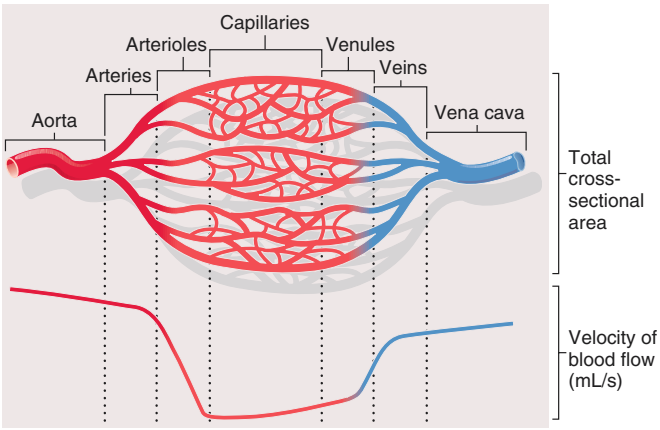


Figure 21.30 **Blood Vessel Types and Velocity of Blood Flow**

Total cross-sectional area for each of the major blood vessel types is illustrated. The cross-sectional area of each blood vessel is the space through which blood flows, measured in square centimeters. The cross-sectional area of the aorta is about 5 cm^2. The cross-sectional area of each capillary is much smaller, but there are so many that the total cross-sectional area of all capillaries is much greater (2500 cm^2) than the cross-sectional area of the aorta. The line at the bottom of the graph shows that blood velocity drops dramatically in arterioles, capillaries, and venules. As the total cross-sectional area increases the velocity of blood flow decreases.

Resistance is small in the aorta, so the average pressure at the end of the aorta is nearly the same as at the beginning of the aorta; about 100 mm Hg. The resistance in medium arteries, which are as small as 3 mm in diameter, is also small, so that their average pressure is only decreased to 95 mm Hg. In the smaller arteries, however, the resistance to blood flow is greater; by the time blood reaches the arterioles, the average pressure is approximately 85 mm Hg. Within the arterioles, the resistance to flow is higher than in any other part of the systemic circulation, and at their ends, the average pressure is only approximately 30 mm Hg. The resistance is also fairly high in the capillaries. The blood pressure at the arterial end of the capillaries is approximately 30 mm Hg, and it decreases to approximately 10 mm Hg at the venous end. Resistance to blood flow in the veins is low because of their relatively large diameter; by the time the blood reaches the right atrium in the venous system, the average pressure has decreased from 10 mm Hg to approximately 0 mm Hg.

The muscular arteries and arterioles are capable of constricting or dilating in response to autonomic and hormonal stimulation. If constriction occurs, the resistance to blood flow increases, less blood flows through the constricted blood vessels, and blood is shunted to other, nonconstricted areas of the body. Muscular arteries help control the amount of blood flowing to each region of the body, and arterioles regulate blood flow through specific tissues. Constriction of an arteriole decreases blood flow through the local area it supplies, and vasodilation increases the blood flow.

Table 21.14 Blood Pressure Classifications

Average Diastolic Blood Pressure (mm Hg)	Average Systolic Blood Pressure (mm Hg)						
	< 120	120–129	130–139	140–159	160–179	180–209	210 or >
< 80	Optimal						
80–84		Normal					
85–89			High Normal				
90–99				Stage 1			
100–109					Stage 2		
110–119						Stage 3	
120 or >							Stage 4

This blood pressure classification system uses the systolic pressure as well as the diastolic pressure in assessing the severity of hypertension. The guidelines also emphasize the assumption that no precise distinction exists between normal and abnormal. The risk of death and disability from heart attack and stroke increases progressively with higher levels of pressure. Even people whose pressure is in the high normal range (systolic between 130 and 139 and diastolic between 85 and 89) are at risk of developing definite high blood pressure and, therefore, should attempt lifestyle modifications.

Normal Pressure: Optimal; Normal; High Normal

Hypertension: Stage 1; Stage 2; Stage 3; Stage 4

Source: National High Blood Pressure Education Program, National Institutes of Health, Bethesda, MD.

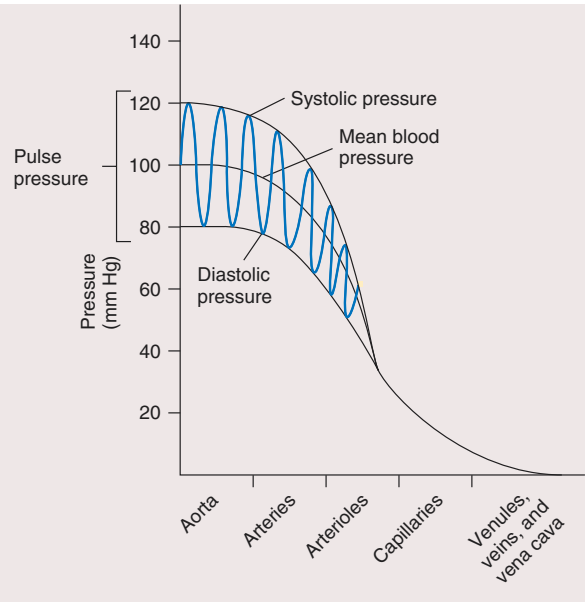


Figure 21.31 Blood Pressure in Major Blood Vessel Types

Blood pressure fluctuations between systole and diastole are damped in small arteries and arterioles. There are no large fluctuations in blood pressure in capillaries and veins.

Clinical Focus Pulse

The pulse is important clinically because one can determine heart rate, rhythmicity, and other characteristics by feeling it. A pulse can be felt at 10 major locations on each side of the body where large arteries are close to the surface.

On the head and neck, a pulse can be felt in three arteries: the common carotid artery in the neck, the superficial temporal artery immediately anterior to the ear, and the facial artery at the point where it crosses the inferior border of the mandible approximately midway between the angle and the genu (figure A).

On the upper limb, a pulse can also be felt in three arteries: the axillary artery in the axilla, the brachial artery on the medial side of the arm slightly proximal to the elbow, and the radial artery on the lateral side of the anterior forearm just proximal to the wrist. The radial artery is traditionally the most common site for taking a pulse, because it is the most easily accessible artery in the body.

In the lower part of the body, a pulse can be felt in four locations: the femoral artery in the groin, the popliteal artery just proximal to the knee, and the dorsalis pedis artery and posterior tibial artery at the ankle.

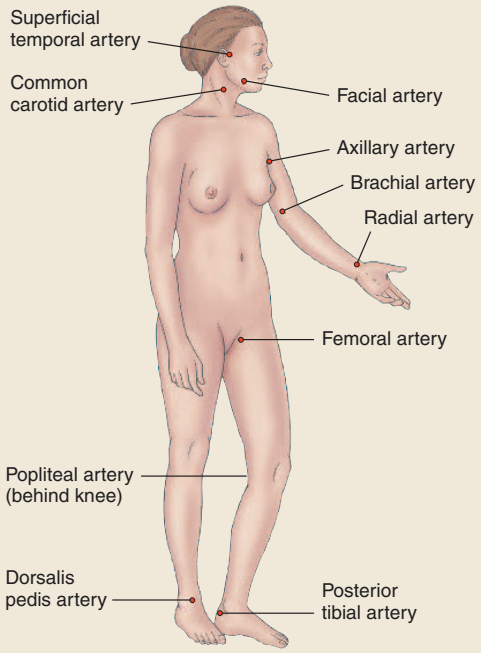


Figure A Location of Major Points at Which the Pulse Can Be Monitored
Each pulse point is named after the artery on which it occurs.

Pulse Pressure

The difference between systolic and diastolic pressures is called **pulse pressure** (see figure 21.31). In a healthy young adult at rest, systolic pressure is approximately 120 mm Hg, and diastolic pressure is approximately 80 mm Hg; thus, the pulse pressure is approximately 40 mm Hg. Two major factors influence pulse pressure: stroke volume of the heart and vascular compliance. When stroke volume decreases, pulse pressure also decreases; and when stroke volume increases, pulse pressure increases. The compliance of blood vessels decreases as arteries age. Arteries in older people become less elastic, or arteriosclerotic, and the resulting decrease in compliance causes the pressure in the aorta to rise more rapidly and to a greater degree during systole and to fall more rapidly to its diastolic value. Thus, for a given stroke volume, systolic pressure and pulse pressure are higher as vascular compliance decreases.

P R E D I C T 3
Explain the consequences of arteriosclerosis that is getting progressively more severe on a large aortic aneurysm.

The pulse pressure caused by the ejection of blood from the left ventricle into the aorta produces a pressure wave, or pulse, that travels rapidly along the arteries. Its rate of transmission is approximately 15 times greater in the aorta (7–10 m/s) and 100 times greater (15–35 m/s) in the distal arteries than the velocity of blood flow. The pulse is monitored frequently, especially in the radial artery, where it's called the **radial pulse,** to determine heart rate and rhythm. Also, weak pulses usually indicate a decreased stroke volume or increased constriction of the arteries as a result of intense sympathetic stimulation of the arteries.

As the pulse passes through the smallest arteries and arterioles, it's gradually damped so that there is a smaller fluctuation between the systolic and diastolic pressure. This difference is almost absent at the end of the arterioles (see figure 21.31). At the beginning of the capillary there is a steady pressure of close to 30 mm Hg.

P R E D I C T 4
Explain each of the following: weak pulses in response to ectopic and premature beats of the heart, strong bounding pulses in a person who received too much saline solution intravenously, weak pulses in a person who is suffering from hemorrhagic shock.

Capillary Exchange and Regulation of Interstitial Fluid Volume

Approximately 10 billion capillaries exist in the body. The heart and blood vessels all function to maintain blood flow through those capillaries and to support **capillary exchange,** which is the movement of substances into and out of capillaries. Capillary exchange is the process by which cells receive everything they need to survive and to eliminate metabolic waste products. If blood flow through capillaries is not maintained, cells cannot survive.

By far, the most important means by which capillary exchange occurs is **diffusion.** Nutrients, such as glucose and amino acids, O_2, and hormones diffuse from a higher concentration in capillaries to a lower concentration in the interstitial spaces. Waste products, including CO_2, diffuse from a higher concentration in the interstitial fluid to a lower concentration in the capillaries. Lipid-soluble molecules cross capillary walls by diffusing though the plasma membranes of the endothelial cells of the capillaries. Examples include O_2, CO_2, steroid hormones, and fatty acids. Water-soluble substances, such as glucose and amino acids, diffuse through intercellular spaces or through fenestrations of capillaries. In a few areas of the body, such as the spleen and liver, the spaces between the endothelial cells are large enough to allow proteins to pass through them. In other areas, the connections between endothelial cells are extensive and few molecules pass between the endothelial cells, such as in the capillaries of the brain that form the blood–brain barrier. In these capillaries mediated transport processes move water-soluble substances across the capillary walls (see chapter 13 for a description of the blood–brain barrier).

Endothelial cells of capillaries appear to take up small pinocytotic vesicles and transport them across the capillary wall. The pinocytotic vesicles, however, don't appear to be a major means by which molecules move across the wall of the capillary.

A small amount of fluid moves out of capillaries at their arterial ends, and most, but not all, of that fluid reenters capillaries at their venous ends (figure 21.32). The remaining fluid enters lymphatic vessels, which eventually return it to the venous circulation (see chapter 22). Alterations in the forces affecting fluid movement across capillary walls are responsible for edema.

Net filtration pressure (NFP) is the force responsible for moving fluid across capillary walls. It is the difference between net hydrostatic pressure and net osmotic pressure.

NFP = Net hydrostatic pressure − Net osmotic pressure

Net hydrostatic pressure is the difference in pressure between the blood and interstitial fluid. **Blood pressure (BP)** at the arterial end of a capillary is about 30 mm Hg. It results mainly from the force of contraction of the heart, but it can be modified by the effect of gravity on fluids within the body (see Blood Pressure and the Effect of Gravity on p. 749).

Interstitial fluid pressure (IFP) is the pressure of interstitial fluid within the tissue spaces. It is –3 mm Hg. IFP is a negative number because of the suction effect produced by the lymphatic

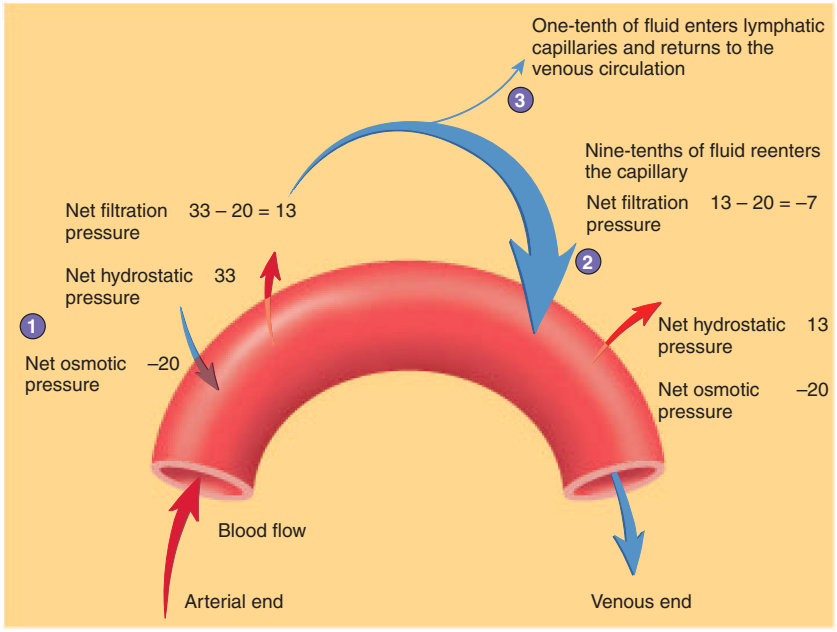
1. At the arterial end of the capillary the net filtration pressure that causes fluid to move from the capillary into the interstitial fluid is 13 mm Hg.

Net hydrostatic pressure	=	33 mm Hg
Net osmotic pressure		–20 mm Hg
Net filtration pressure		13 mm Hg

2. At the venous end of the capillary the net filtration pressure that causes fluid to move from the interstitial fluid into the capillary is –7 mm Hg

Net hydrostatic pressure	=	13 mm Hg
Net osmotic pressure		–20 mm Hg
Net filtration pressure		–7 mm Hg

3. Approximately nine-tenths of the fluid that leaves the capillary at its arterial end reenters the capillary at its venous end. About one-tenth of the fluid passes into the lymphatic capillaries.



Process Figure 21.32 Fluid Exchange Across the Walls of Capillaries

The total pressure differences between the inside and the outside of the capillary at its arterial and venous ends are illustrated.

vessels as they pump excess fluid from the tissue spaces. The lymphatic system is described in chapter 22. Here, it is only necessary to understand that excess interstitial fluid enters lymphatic capillaries and is eventually returned to the blood.

At the arterial end of capillaries, the net hydrostatic pressure that moves fluid across capillary walls into the tissue spaces is the difference between BP and IFP.

Net hydrostatic pressure = BP − IFP
= 30 − (−3)
= 33 mm Hg

Net osmotic pressure is the difference in osmotic pressure between the blood and the interstitial fluid. The osmotic pressure caused by the plasma proteins is called the **blood colloid osmotic pressure (BCOP),** and the osmotic pressure caused by proteins in the interstitial fluid is called the **interstitial colloid osmotic pressure (ICOP).** Large proteins do not freely pass through the capillary walls, and the difference in protein concentrations between the blood and interstitial fluid is responsible for osmosis. Ions and small molecules do not make a significant contribution to osmosis across the capillary wall because they freely pass through it and their concentrations are approximately the same in the blood as in the interstitial fluid.

The BCOP (28 mm Hg) is several times larger than the ICOP (8 mm Hg) because of the presence of albumin and other proteins in the plasma (see chapter 19). Therefore, the net osmotic pressure is equal to BCOP–ICOP.

Net osmotic pressure = BCOP − ICOP
= 28 − 8
= 20 mm Hg

The greater the osmotic pressure of a fluid, the greater is the tendency for water to move into that fluid (see chapter 3). The net osmotic pressure results in the osmosis of water into the capillary because there is a greater tendency for water to move into the blood than into the interstitial fluid.

The net filtration pressure at the arterial end of the capillary is equal to the net hydrostatic pressure, which moves fluid out of the capillary, minus the net osmotic pressure, which moves fluid into the capillary.

NFP = Net hydrostatic pressure − Net osmotic pressure
= 33 − 20
= 13 mm Hg

Between the arterial ends of capillaries and their venous ends, the blood pressure decreases from about 30 mm Hg to 10 mm Hg, which reduces the net hydrostatic pressure moving fluid out of the venous end of the capillary.

Net hydrostatic pressure = BP − IFP
= 10 − (−3)
= 13 mm Hg

The concentration of proteins within capillaries and the concentration of proteins within interstitial fluid do not change significantly because only a small amount of fluid passes from the capillaries into the tissue spaces. Therefore, the net osmotic pressure moving fluid into capillaries by osmosis is still approximately 20 mm Hg. At the venous end of capillaries the NFP now causes fluid to reenter the capillary:

NFP = Net hydrostatic pressure − Net osmotic pressure
= 13 − 20
= −7 mm Hg

Exchange of fluid across the capillary wall and movement of fluid into lymphatic capillaries keep the volume of the interstitial fluid within a narrow range of values. Disruptions in the movement of fluid across the wall of the capillary can result in edema, or swelling, as a result of an increase in interstitial fluid volume.

Edema and Capillary Exchange

Increases in the permeability of capillaries allow plasma proteins to move from capillaries into the interstitial fluid. This causes an increase in the colloid osmotic pressure of the interstitial fluid. An increase in the interstitial colloid osmotic pressure causes a net increase in the amount of fluid moving from capillaries into interstitial spaces. The result is edema. Chemical mediators of inflammation increase the permeability of the capillary walls and can cause edema. Decreases in plasma protein concentration reduce the blood colloid osmotic pressure, which results in more fluid moving out of the capillary at its arterial end and less fluid moving into the capillary at its venous end. The result once again is edema. Severe liver infections that reduce plasma protein synthesis, loss of protein molecules in urine through the kidneys, and protein starvation all result in edema. Blockage of veins, such as in venous thrombosis, increases blood pressure in capillaries and can result in edema. Either blockage or removal of lymphatic vessels causes fluid to accumulate in the interstitial spaces and results in edema. Removal of lymphatic vessels occurs when lymph nodes that are suspected to be cancerous are removed.

P R E D I C T 5

Edema often results from a disruption in the normal inwardly and outwardly directed pressures across the capillary wall. On the basis of what you know about fluid movement across the wall of the capillary and the regulation of capillary blood pressure, explain why large fluctuations in arterial blood pressure occur without causing significant edema and why small increases in venous pressure can lead to edema.

Functional Characteristics of Veins

Cardiac output depends on the preload, which is determined by the volume of blood that enters the heart from the veins (see chapter 20). The factors that affect flow in the veins are, therefore, of great importance to the overall function of the cardiovascular system. If the volume of blood is increased because of a rapid transfusion, the amount of blood flow to the heart through the veins increases. This increases the preload, which causes the cardiac output to increase because of Starling's law of the heart. On the other hand, a rapid loss of a large volume of blood decreases venous return to the heart, which decreases the preload and cardiac output.

Venous tone is a continual state of partial contraction of the veins as a result of sympathetic stimulation. Increased sympathetic stimulation increases venous tone by causing constriction of the veins, which forces the large venous volume to flow toward the heart. Consequently, venous return and preload increase, causing an increase in cardiac output. Conversely, decreased sympathetic

stimulation decreases venous tone, allowing veins to relax and dilate. As the veins fill with blood, venous return to the heart, preload, and cardiac output decrease.

The periodic muscular compression of veins forces blood to flow more rapidly through them toward the heart. The valves in the veins prevent flow away from the heart so that when veins are compressed, blood is forced to flow toward the heart. The combination of arterial dilation and compression of the veins by muscular movements during exercise causes blood to return to the heart more rapidly than under conditions of rest.

Blood Pressure and the Effect of Gravity

Blood pressure is approximately 0 mm Hg in the right atrium, and it averages approximately 100 mm Hg in the aorta. The pressure in vessels above and below the heart, however, is affected by gravity. While a person is standing, the pressure in the venules of the feet can be as much as 90 mm Hg, instead of its usual 10 mm Hg pressure. Arterial pressure is influenced by gravity to the same degree; thus the arterial ends of the capillaries can have a pressure of 110 mm Hg rather than 30 mm Hg. The normal pressure difference between the arterial and the venous ends of capillaries still remains the same, so that flow continues through the capillaries. The major effect of the high pressure in the feet and legs when a person stands for a prolonged time without moving is edema. Without muscular movement, the pressure at the venous end of the capillaries increases. Up to 15%–20% of the total blood volume can pass through the walls of the capillaries into the interstitial spaces of the lower limbs during 15 minutes of standing still.

30. *Explain how the total cross-sectional area of blood vessels, blood pressure, and resistance to flow change as blood flows through the aorta, small arteries, arterioles, capillaries, venules, small veins, and venae cavae.*
31. *What is pulse pressure? How do stroke volume and vascular compliance affect pulse pressure?*
32. *What is the most important means by which capillary exchange occurs?*
33. *Describe the factors that influence the movement of fluid from capillaries into the tissues. What happens to the fluid in the tissues? What is edema?*
34. *How do blood volume and venous tone affect cardiac output?*
35. *What effect does standing have on blood pressure in the feet and the head? Explain why this effect occurs.*

PREDICT 6

Explain why people who are suffering from edema in the legs are told to keep them elevated.

Control of Blood Flow in Tissues

Objectives

- *Describe the mechanisms responsible for the local control of blood flow through tissues.*
- *List the characteristics of short and long-term regulation of blood flow through tissues.*

Blood flow provided to the tissues by the cardiovascular system is highly controlled and matched closely to the metabolic needs of tissues. Mechanisms that control blood flow through tissues are classified as (1) local control and (2) nervous and hormonal control.

Local Control of Blood Flow by the Tissues

Blood flow is much greater in some organs than in others. For example, blood flow through the brain, kidneys, and liver is relatively high. The muscle mass of the body is large so that flow through resting skeletal muscles, although not high, is greater than that through other tissue types because skeletal muscle constitutes 35%–40% of the total body mass. Flow through exercising skeletal muscles can increase up to 20-fold, however, and the blood flow through the viscera, including the kidneys and liver, either remains the same or decreases.

In most tissues, blood flow is proportional to the metabolic needs of the tissue; therefore, as the activity of skeletal muscle increases, blood flow increases to supply the increased need for oxygen and other nutrients. Blood flow also increases in response to a buildup of metabolic end products.

In some tissues, however, blood flow serves purposes other than the delivery of nutrients and the removal of waste products. In the skin, blood flow also dissipates heat from the body. In the kidneys, it eliminates metabolic waste products, regulates water balance, and controls the pH of body fluids. Among other functions, blood flow through the liver delivers nutrients that have entered the blood from the small intestine in route to the liver for processing.

Functional Characteristics of the Capillary Bed

The innervation of the metarterioles and the precapillary sphincters in capillary beds is sparse (table 21.15). Local factors regulate these structures primarily. As the rate of metabolism increases in a tissue, blood flow through its capillaries increases. The precapillary sphincters relax, allowing blood to flow into the local capillary bed. Blood flow can increase sevenfold to eightfold as a result of vasodilation of the metarterioles and the precapillary sphincters in response to an increased rate of metabolism.

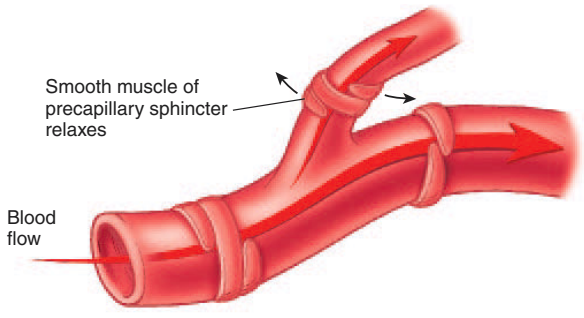
Vasodilator substances are produced as the rate of metabolism increases. The vasodilator substances then diffuse from the tissues supplied by the capillary to the area of the precapillary sphincter, the metarterioles, and the arterioles, to cause vasodilation (figure 21.33*a*). Several chemicals, including carbon dioxide, lactic acid, adenosine, adenosine monophosphate, adenosine diphosphate, endothelium-derived relaxation factor (EDRF), potassium ions, and hydrogen ions, cause vasodilation, and they increase in concentration in the extracellular fluid as the rate of metabolism in tissues increases.

Lack of nutrients can also be important in regulating local blood flow. For example, oxygen and other nutrients are required to maintain vascular smooth muscle contraction. An increased rate of metabolism decreases the amount of oxygen and other nutrients in the tissues. Smooth muscle cells of the precapillary sphincter relax in response to a lack of oxygen and other nutrients, resulting in vasodilation (see figure 21.33*a*).

Blood flow through capillaries is not continuous but cyclic. The cyclic fluctuation is the result of periodic contraction and relaxation of the precapillary sphincters called **vasomotion** (vā-sō-mō′shŭn, vas-ō-mō′shŭn). Blood flows through the capillaries until

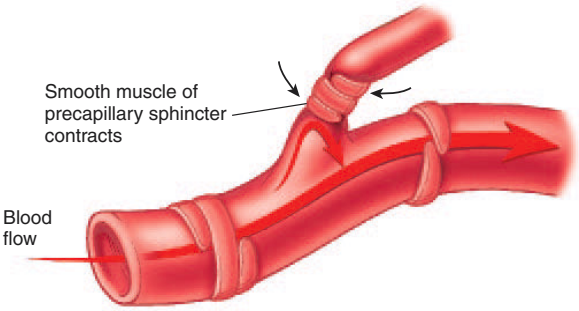
Table 21.15 Homeostasis: Local Control of Blood Flow

Stimulus	Response
Regulation by Metabolic Need of Tissues	
Increased vasodilator substances (e.g., CO_2, lactic acid, adenosine, adenosine monophosphate, adenosine diphosphate, endothelium-derived relaxation factor, K^+, decreased pH) or decreased nutrients (e.g., O_2, glucose, amino acids, fatty acids, and other nutrients) as a result of increased metabolism	Relaxation of precapillary sphincters and subsequent increase in blood flow through capillaries
Decreased vasodilator substances and a reduced need for O_2 and other nutrients	Contraction of precapillary sphincters and subsequent decrease in blood flow through capillaries
Regulation by Nervous Mechanisms	
Increased physical activity or increased sympathetic activity	Constriction of blood vessels in skin and viscera
Increased body temperature detected by neurons of the hypothalamus	Dilation of blood vessels in skin (see chapter 5)
Decreased body temperature detected by neurons of the hypothalamus	Constriction of blood vessels in skin (see chapter 5)
Decrease in skin temperature below a critical value	Dilation of blood vessels in skin (protects skin from extreme cold)
Anger or embarrassment	Dilation of blood vessels in skin of face and upper thorax
Regulation by Hormonal Mechanisms	
(reinforces increased activity of the sympathetic nervous system)	
Increased physical activity and increased sympathetic activity causing release of epinephrine and small amounts of norepinephrine from the adrenal medulla	Constriction of blood vessels in skin and viscera; dilation of blood vessels in skeletal and cardiac muscle
Autoregulation	
Increased blood pressure	Contraction of precapillary sphincters to maintain constant capillary blood flow
Decreased blood pressure	Relaxation of precapillary sphincters to maintain constant capillary blood flow
Long-Term Local Blood Flow	
Increased metabolic activity of tissues over a long period	Increased diameter and number of capillaries
Decreased metabolic activity of tissues over a long period	Decreased diameter and number of capillaries



(a) Precapillary sphincters relax due to an increase in vasodilator substances such as CO_2, lactic acid, adenosine, adenosine monophosphate, adenosine diphosphate, nitric oxide, K^+ and H^+.

The need for O_2, glucose, amino acids, fatty acids, and other nutrients cause precapillary sphincters to relax.



(b) Removal of vasodilator substances and a reduced need for O_2 and other nutrients cause precapillary sphincters to contract.

Figure 21.33 Control of Local Blood Flow Through Capillary Beds

(a) Dilation of precapillary sphincters. *(b)* Constriction of precapillary sphincters.

Clinical Focus Hypertension

Hypertension, or high blood pressure, affects approximately 20% of the human population at sometime in their lives. Generally, a person is considered hypertensive if the systolic blood pressure is greater then 140 mm Hg and the diastolic pressure is greater than 90 mm Hg. Current methods of evaluation, however, take into consideration combinations of diastolic and systolic blood pressure in determining whether a person is suffering from hypertension (see table 21.14). In addition, normal blood pressure is age-dependent, so classification of an individual as hypertensive depends on the person's age.

Chronic hypertension has an adverse effect on the function of both the heart and blood vessels. Hypertension requires the heart to work harder than normal. This extra work leads to hypertrophy of the cardiac muscle, especially in the left ventricle, and can lead to heart failure. Hypertension also increases the rate at which arteriosclerosis develops. Arteriosclerosis, in turn, increases the probability that blood clots, or thromboemboli (throm′bō-em′bō-lī), may form and that blood vessels will rupture. Common medical problems associated with hypertension are cerebral hemorrhage, coronary infarction, hemorrhage of renal blood vessels, and poor vision caused by burst blood vessels in the retina.

Some conditions leading to hypertension include a decrease in functional kidney mass, excess aldosterone or angiotensin production, and increased resistance to blood flow in the renal arteries. All of these conditions cause an increase in total blood volume, which causes cardiac output to increase. Increased cardiac output forces blood to flow through tissue capillaries, causing the precapillary sphincters to constrict. Thus increased blood volume increases cardiac output and peripheral resistance, both of which result in greater blood pressure.

Although these conditions result in hypertension, roughly 90% of the diagnosed cases of hypertension are called **idiopathic,** or **essential, hypertension,** which means the cause of the condition is unknown. Drugs that dilate blood vessels (called vasodilators), drugs that increase the rate of urine production (called diuretics), or drugs that decrease cardiac output normally are used to treat essential hypertension. The vasodilator drugs increase the rate of blood flow through the kidneys and thus increase urine production, and the diuretics also increase urine production. Increased urine production reduces blood volume, which reduces blood pressure. Substances that decrease cardiac output, such as β-adrenergic-blocking agents, decrease the heart rate and force of contraction. In addition to these treatments, low-salt diets normally are recommended to reduce the amount of sodium chloride and water absorbed from the intestine into the bloodstream.

the by-products of metabolism are reduced in concentration and until nutrient supplies to precapillary smooth muscles are replenished. Then the precapillary sphincters constrict and remain constricted until the by-products of metabolism increase and nutrients decrease (figure 21.33*b*).

Autoregulation of Blood Flow

Arterial pressure can change over a wide range, whereas blood flow through tissues remains relatively constant. The maintenance of blood flow by tissues is called **autoregulation** (aw′tō-reg-ū-lā′shŭn). Between arterial pressures of approximately 75 mm Hg and 175 mm Hg, blood flow through tissues remains within 10%–15% of its normal value. The mechanisms responsible for autoregulation are the same as those for vasomotion. The need for nutrients and the buildup of metabolic by-products cause precapillary sphincters to dilate, and blood flow through tissues increases if a minimum blood pressure exists. On the other hand, once the supply of nutrients and oxygen to tissues is adequate, the precapillary sphincters constrict, and blood flow through the tissues decreases, even if blood pressure is very high.

P R E D I C T 7

When blood flow to a tissue has been blocked for a short time, the blood flow through that tissue increases to as much as five times its normal value after the removal of the blockade. The response is called reactive hyperemia. Create a reasonable explanation for this phenomenon on the basis of what you know about the local control of blood flow.

Long-Term Local Blood Flow

The long-term regulation of blood flow through tissues is matched closely to the metabolic requirements of the tissue. If the metabolic activity of a tissue increases and remains elevated for an extended period, the diameter and the number of capillaries in the tissue increase, and local blood flow increases. The increased density of capillaries in the well-trained skeletal muscles of athletes compared to that in poorly trained skeletal muscles is an example.

The availability of oxygen to a tissue can be a major factor in determining the adjustment of the vascularity of a tissue to its long-term metabolic needs. If oxygen is scarce, capillaries increase in diameter and in number, and if the oxygen levels remain elevated in a tissue, the vascularity decreases.

Occlusion of Blood Vessels and Collateral Circulation

Blockage, or occlusion, of a blood vessel leads to an increase in the diameter of smaller blood vessels that bypass the occluded vessel. In many cases, the development of these collateral vessels is marked. For example, if a vessel such as the femoral artery becomes occluded, the small vessels that bypass the occluded vessel become greatly enlarged. An adequate blood supply to the lower limb is often reestablished over a period of weeks. If the occlusion is sudden and so complete that tissues supplied by a blood vessel suffer from ischemia (lack of blood flow), cell death (necrosis) can occur. In this instance, collateral circulation doesn't have a chance to develop before necrosis occurs.

Nervous and Hormonal Regulation of Local Circulation

Nervous control of arterial blood pressure is important in minute-to-minute regulation of local circulation. The blood pressure must be adequate to cause blood flow through capillaries while at rest, during exercise, or in response to circulatory shock, during which blood pressure decreases to a very low value. For example, during exercise, increased arterial blood pressure is needed to cause blood to flow through the capillaries of skeletal muscles at a rate great enough to supply their oxygen need.

Nervous regulation also provides a means by which blood can be shunted from one large area of the peripheral circulatory system to another. For example, in response to blood loss, blood flow to the viscera and the skin is reduced dramatically. This helps maintain the arterial blood pressure within a range sufficient to allow adequate blood flow through the capillaries of the brain and cardiac muscle.

Nervous regulation, by the autonomic nervous system, can function rapidly (within 1–30 seconds). The most important part of the autonomic nervous system for this regulation is the sympathetic division (figure 21.34). Sympathetic vasomotor fibers innervate all blood vessels of the body except the capillaries, precapillary sphincters, and most metarterioles. The innervation of the small arteries and arterioles allows the sympathetic nervous system to increase or decrease resistance to blood flow.

PREDICT 8

A strong athlete just finished a 1-mile run and sat down to have a drink with her friends. Her blood pressure was not dramatically elevated during the run, but her cardiac output was greatly increased. After the run, her cardiac output decreased dramatically, but her blood pressure only decreased to its resting level. Predict how sympathetic stimulation of her large veins, arteries in her digestive system, and arteries in her skeletal muscles change while she is relaxing. Explain why this is consistent with the decrease in her cardiac output.

Sympathetic vasoconstrictor fibers extend to most parts of the circulatory system, but they are less prominent in skeletal muscle, cardiac muscle, and the brain and more prominent in the kidneys, gut, spleen, and skin.

An area of the lower pons and upper medulla oblongata, called the **vasomotor** (vā-sō-mō′ter, vas-ō-mō′ter) **center** (see figure 21.34), is tonically active. A low frequency of action potentials is transmitted continually through the sympathetic vasoconstrictor fibers. As a consequence, the peripheral blood vessels are partially constricted, a condition called **vasomotor tone.**

Part of the vasomotor center inhibits vasomotor tone. Thus, the vasomotor center consists of an excitatory part, which is tonically active, and an inhibitory part, which can induce vasodilation. Vasoconstriction results from an increase and vasodilation from a decrease in vasomotor tone.

Areas throughout the pons, midbrain, and diencephalon can either stimulate or inhibit the vasomotor center. For example, the hypothalamus can exert either strong excitatory or inhibitory effects on the vasomotor center. Increased body temperature detected by temperature receptors in the hypothalamus causes vasodilation of blood vessels in the skin (see chapter 5). The cerebral cortex also can either excite or inhibit the vasomotor center. For example, action potentials that originate in the cerebral cortex during periods of emotional excitement activate hypothalamic centers, which in turn increase vasomotor tone (see table 21.15).

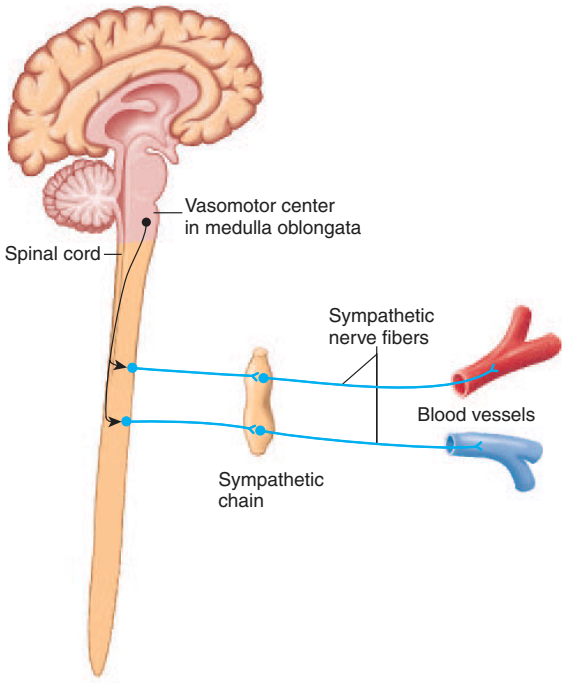


Figure 21.34 Nervous Regulation of Blood Vessels

Most blood vessels are innervated by sympathetic nerve fibers. The vasomotor center within the medulla oblongata plays a major role in regulating the frequency of action potentials in nerve fibers that innervate blood vessels.

The neurotransmitter for the vasoconstrictor fibers is norepinephrine, which binds to α-adrenergic receptors on vascular smooth muscle cells to cause vasoconstriction. Sympathetic action potentials also cause the release of epinephrine and norepinephrine into the blood from the adrenal medulla. These hormones are transported in the blood to all parts of the body. In most vessels, they cause vasoconstriction, but in some vessels, especially those in skeletal muscle, epinephrine binds to β-adrenergic receptors, which are present in larger numbers, and can cause the skeletal muscle blood vessels to dilate.

36. *Explain how vasodilator substances and nutrients are involved with local control of blood flow. What is vasomotion? What is autoregulation of local blood flow?*
37. *How is long-term regulation of blood flow through tissues accomplished?*
38. *Describe nervous and hormonal control of blood flow. Under what conditions is nervous control of blood flow important? Define the term vasomotor tone.*

Regulation of Mean Arterial Pressure

Objectives

- ***Define mean arterial pressure, and explain the factors that determine it.***
- ***Describe the short-term and long-term mechanisms that regulate mean arterial pressure.***
- ***Define hypertension, and explain its effect on the circulatory system.***
- ***Describe how the circulatory system responds to exercise and shock.***

Blood flow to all areas of the body depends on the maintenance of an adequate pressure in the arteries. As long as arterial blood pressure is adequate, local control of blood flow through tissues is appropriately matched to their metabolic needs. Blood flow through tissues cannot be adequate if arterial blood pressure is too low, and damage, including heart and blood vessel damage, can result if arterial blood pressure is too high. This section describes the mechanisms that operate to maintain arterial blood pressure within a normal range of values.

Mean arterial pressure (MAP) is slightly less than the average of systolic and diastolic pressures because diastole lasts longer than systole. MAP is approximately 70 mm Hg at birth, is approximately 100 mm Hg from adolescence to middle age, and reaches 110 mm Hg in the healthy older person, but it can be as high as 130 mm Hg. The range of normal systolic and diastolic blood pressures for adults is presented in table 21.14.

Cardiac output (CO) is the volume of blood pumped by the heart each minute. It is equal to the **heart rate (HR)** times the **stroke volume (SV). Peripheral resistance (PR)** is the resistance to blood flow in all the blood vessels. MAP in the body is proportional to the cardiac output times the peripheral resistance: Blood flow through the entire circulatory system is determined by the cardiac output *(CO),* which is equal to the heart rate *(HR)* times the stroke volume *(SV)* and peripheral resistance *(PR),* which is the resistance to blood flow in all the blood vessels.

$$MAP = CO \times PR \quad \text{or} \quad MAP = HR \times SV \times PR$$

This equation expresses the effect of heart rate, stroke volume, and peripheral resistance on blood pressure. An increase in any one of them results in an increase in blood pressure. Conversely, a decrease in any one of them produces a decrease in blood pressure. The mechanisms that control blood pressure do so by changing peripheral resistance, heart rate, or stroke volume. Because stroke volume depends on the amount of blood entering the heart, regulatory mechanisms that control blood volume also affect blood pressure. For example, an increase in blood volume increases venous return, which increases preload, and the increased preload increases stroke volume.

When blood pressure suddenly drops because of hemorrhage or some other cause, the control systems respond by increasing blood pressure to a value consistent with life and by increasing blood volume to its normal value. Two major types of control systems operate to achieve these responses: (1) those that respond in the short term and (2) those that respond in the long term.

The regulatory mechanisms that control pressure on a short-term basis respond quickly but begin to lose their capacity to regulate blood pressure a few hours to a few days after blood pressure is maintained at higher or lower values. This occurs because sensory receptors adapt to the altered pressures. Long-term regulation of blood pressure is controlled primarily by mechanisms that influence kidney function, and those mechanisms don't adapt rapidly to altered blood pressures.

Short-Term Regulation of Blood Pressure

The short-term, rapidly acting mechanisms controlling blood pressure include the baroreceptor reflexes, the adrenal medullary mechanism, chemoreceptor reflexes, and the central nervous system ischemic response. Some of these reflex mechanisms operate on a minute-to-minute basis and help regulate blood pressure within a narrow range of values. Other mechanisms respond primarily to emergency situations. The mechanisms responsible for the short-term regulation of the blood pressure are summarized in figures 21.38 and 21.39.

Baroreceptor Reflexes

Baroreceptor reflexes are very important in regulating blood pressure on a minute-to-minute basis. They detect even small changes in blood pressure and respond quickly. However, they are not as important as other mechanisms in regulating blood pressure over long periods of time.

Baroreceptors, or **pressoreceptors,** are sensory receptors sensitive to stretch. They are scattered along the walls of most of the large arteries of the neck and thorax, and are most numerous in the area of the carotid sinus at the base of the internal carotid artery and in the walls of the aortic arch. Action potentials are transmitted from the carotid sinus baroreceptors through the glossopharyngeal nerves to the cardioregulatory and vasomotor centers in the medulla oblongata and from the aortic arch through the vagus nerves to the medulla oblongata (figure 21.35). Stimulation of baroreceptors in the carotid sinus activates the **carotid sinus reflex,** and stimulation of baroreceptors in the aortic arch activates the **aortic arch reflex.** Both of these reflexes, are baroreceptor reflexes, and they both function to control blood pressure within a narrow range of values.

In the carotid sinus and the aortic arch, normal blood pressure partially stretches the arterial wall so that the baroreceptors produce a constant, but low, frequency of action potentials. Increased pressure in the blood vessels stretches the vessel walls and causes the baroreceptors to increase the frequency of action potentials. Conversely, a decrease in blood pressure reduces the stretch of the arterial wall and causes the baroreceptors to decrease the frequency of action potentials.

A sudden increase in blood pressure increases the frequency of action potentials produced in the baroreceptors. The increase in action potentials influences the vasomotor and cardioregulatory centers of the medulla oblongata. The vasomotor center responds by

Clinical Focus Blood Flow Through Tissues During Exercise

During exercise, blood flow through tissues is changed dramatically. Its rate of flow through exercising skeletal muscles can be 15–20 times greater than through resting muscles. Increased blood flow is the product of local, nervous, and hormonal regulatory mechanisms. When skeletal muscle is resting, only 20%–25% of the capillaries in the skeletal muscle are open, whereas during exercise 100% of the capillaries are open.

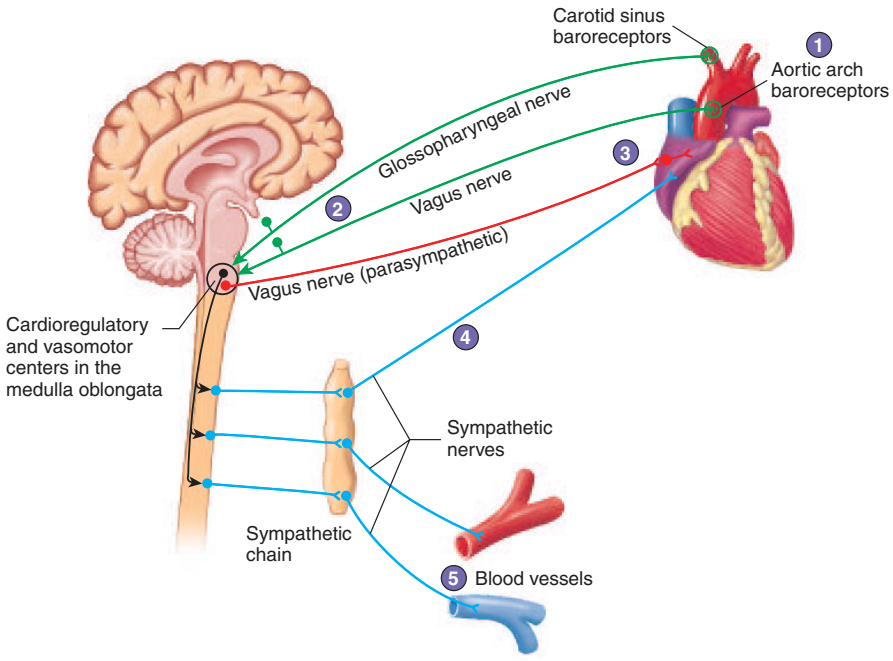
Low oxygen tensions resulting from greatly increased muscular activity or the release of vasodilator substances, such as lactic acid, carbon dioxide, and potassium ions, cause dilation of precapillary sphincters. Increased sympathetic stimulation and epinephrine released from the adrenal medulla cause vasoconstriction in the blood vessels of the skin and viscera and some vasoconstriction in the blood vessels of skeletal muscles. The resistance to blood flow in skeletal muscle decreases even though some vasoconstriction in skeletal muscle blood vessels occurs because the capillaries are all open. Resistance to blood flow in the skin and viscera increases. Blood is therefore shunted from the viscera and the skin to the vessels in skeletal muscles.

The movement of skeletal muscles that compresses veins in a cyclic fashion and the constriction of veins greatly increase the venous return to the heart. The resulting increase in the preload and increased sympathetic stimulation of the heart result in elevated heart rate and stroke volume, which increases cardiac output. As a consequence, blood pressure usually increases by 20–60 mm Hg, which helps sustain the increased blood flow through skeletal muscle blood vessels.

In response to sympathetic stimulation, some decrease in blood flow through the skin can occur at the beginning of exercise. As body temperature increases in response to the increased muscular activity, however, temperature receptors in the hypothalamus are stimulated. As a result, action potentials in sympathetic nerve fibers causing vasoconstriction decrease, resulting in vasodilation of blood vessels in the skin. As a consequence, the skin turns a red or pinkish color, and a great deal of excess heat is lost as blood flows through the dilated blood vessels.

The overall effect of exercise on circulation is to greatly increase blood flow through exercising muscles and to keep blood flow through other organs at a value just adequate to supply their metabolic needs.

1. Baroreceptors in the carotid sinus and aortic arch monitor blood pressure.
2. Action potentials are conducted by the glossopharyngeal and vagus nerves to the cardioregulatory and vasomotor centers in the medulla oblongata.
3. Increased parasympathetic stimulation of the heart decreases the heart rate.
4. Increased sympathetic stimulation of the heart increases the heart rate and stroke volume.
5. Increased sympathetic stimulation of blood vessels increases vasoconstriction.



Process Figure 21.35 Baroreceptor Reflex Control of Blood Pressure

An increase in blood pressure increases parasympathetic stimulation of the heart and decreases sympathetic stimulation of the heart and blood vessels, resulting in a decrease in blood pressure. A decrease in blood pressure decreases parasympathetic stimulation of the heart and increases sympathetic stimulation of the heart and blood vessels, resulting in an increase in blood pressure.

decreasing sympathetic stimulation of blood vessels, and the cardioregulatory center responds by increasing parasympathetic stimulation of the heart. As a result, peripheral blood vessels dilate, the heart rate decreases, and the blood pressure decreases (see figure 21.38).

A sudden decrease in blood pressure results in a decreased frequency of action potentials produced by the baroreceptors. The decreased action potentials influence the vasomotor center and cardioregulatory centers of the medulla oblongata. The vasomotor center responds by increasing sympathetic stimulation of blood vessels, and the cardioregulatory center responds by increasing sympathetic stimulation of the heart. As a result peripheral blood vessels constrict, the heart rate and stroke volume increase, and the blood pressure increases. This increase is accompanied by a decrease in parasympathetic stimulation of the heart (see figures 21.35 and 21.37).

The carotid sinus and aortic arch baroreceptor reflexes are important in regulating blood pressure moment to moment. When a person rises rapidly from a sitting or lying position to a standing position, a dramatic drop in blood pressure in the neck and thoracic regions occurs because of the pull of gravity on the blood. This reduction can be so great that blood flow to the brain becomes sufficiently sluggish to cause dizziness or loss of consciousness. The falling blood pressure activates the baroreceptor reflexes, which reestablish normal blood pressure within a few seconds. A healthy person may experience only a temporary sensation of dizziness.

P R E D I C T 9

Explain how the baroreceptor reflex responds when a person does a headstand.

The baroreceptor reflexes are short term and rapid-acting. They don't change the average blood pressure in the long run. The baroreceptors adapt within 1–3 days to any new sustained blood pressure to which they are exposed. If the blood pressure is elevated for more than a few days, the baroreceptors adapt to the elevated pressure and the baroreceptor reflex does not reduce blood pressure to its original value. This adaptation is common in people who have hypertension.

The Carotid Sinus Syndrome

Occasionally, the application of pressure to the carotid arteries in the upper neck results in a dramatic decrease in blood pressure. This condition, called the **carotid sinus syndrome,** is most common in patients in whom arteriosclerosis of the carotid artery is advanced. In such patients a tight collar can apply enough pressure to the region of the carotid sinuses to stimulate the baroreceptors. The increased action potentials from the baroreceptors initiate reflexes that result in a decrease in vasomotor tone and an increase in parasympathetic action potentials to the heart. As a result of the decreased peripheral resistance and heart rate, blood pressure decreases dramatically. As a consequence, blood flow to the brain decreases to such a low level that the person becomes dizzy or may even faint. People suffering from this condition must avoid applying external pressure to the neck region. If the carotid sinus becomes too sensitive, a treatment for this condition is surgical destruction of the innervation to the carotid sinuses.

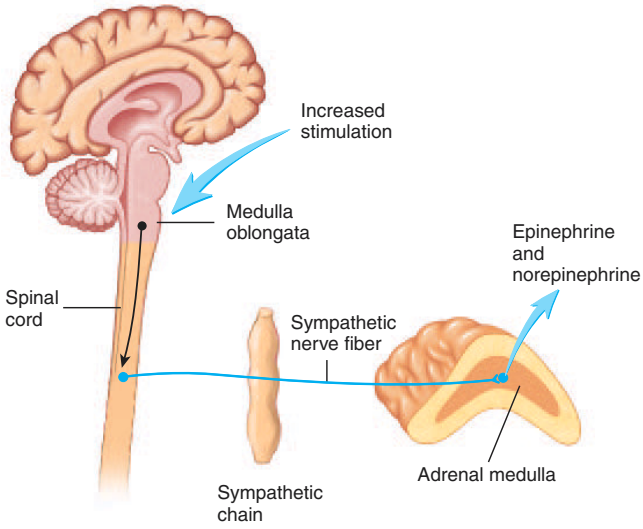


Figure 21.36 The Adrenal Medullary Mechanism

Stimuli that increase sympathetic stimulation of the heart and blood vessels also result in increased sympathetic stimulation of the adrenal medulla and result in epinephrine and some norepinephrine secretion.

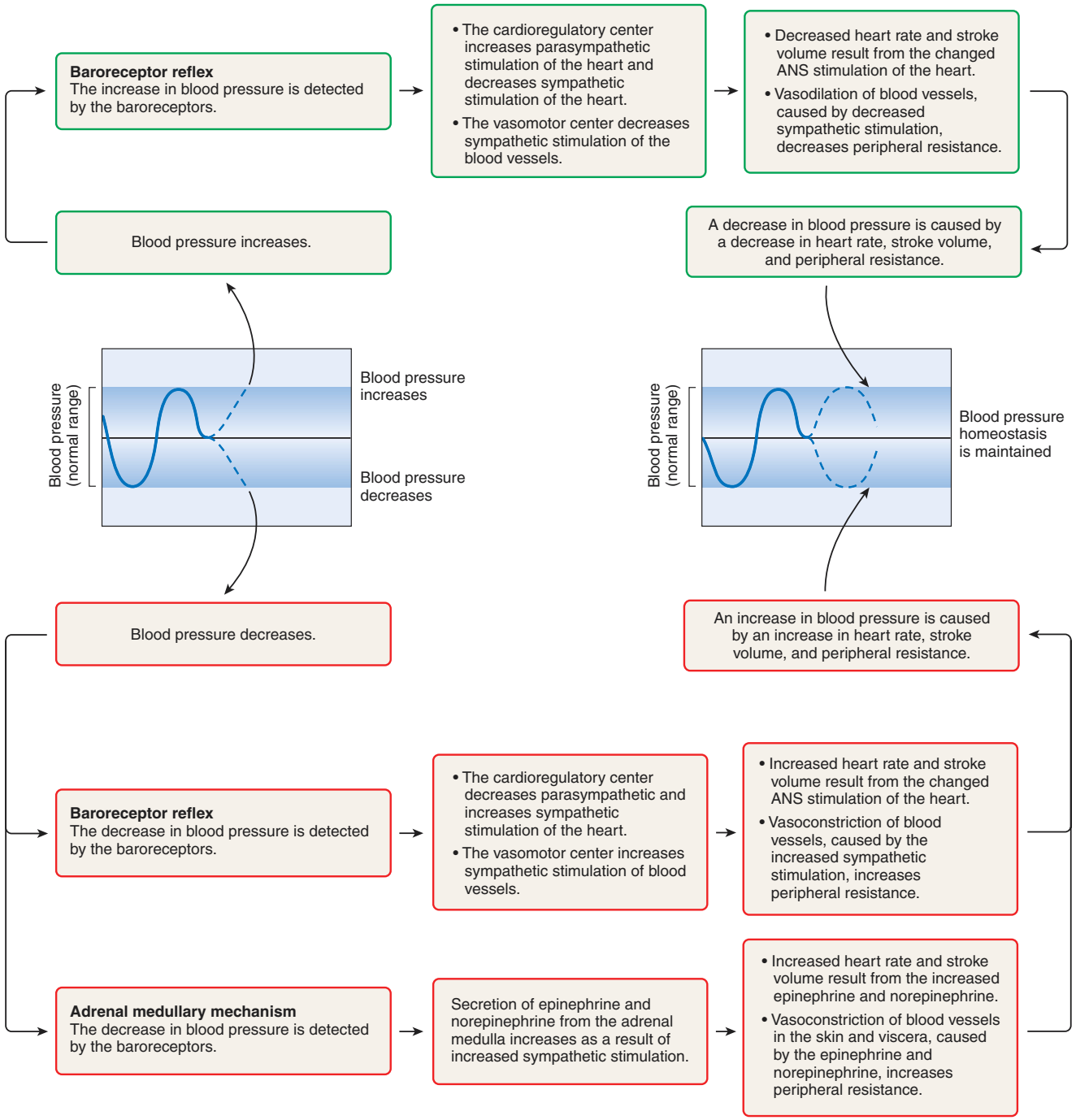
Adrenal Medullary Mechanism

The adrenal medullary mechanism is activated when stimuli result in a substantial increase in sympathetic stimulation of the heart and blood vessels (figure 21.36 and figure 21.37). Large decreases in blood pressure, sudden and substantial increases in physical activity, and other stressful conditions are examples. The adrenal medullary mechanism results from stimulation of the adrenal medulla by the sympathetic nerve fibers. The adrenal medulla releases epinephrine and smaller amounts of norepinephrine into the circulatory system (see figure 21.36 and figure 26.37). These hormones affect the cardiovascular system in a fashion similar to direct sympathetic stimulation, causing increased heart rate, increased stroke volume, and vasoconstriction in blood vessels to the skin and viscera. Epinephrine can indirectly cause vasodilation in blood vessels to the heart because of the increased rate of cardiac muscle metabolism (see chapter 20). The adrenal medullary mechanism is short term and rapid-acting, whereas other hormonal mechanisms are long term and slow-acting (see following sections).

Chemoreceptor Reflexes

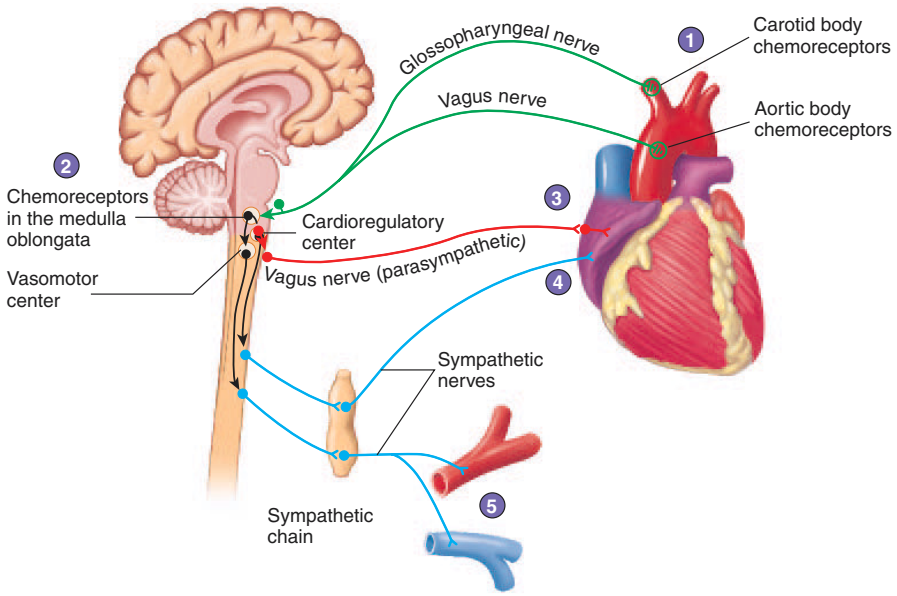
The **chemoreceptor** (kē′mō-rē-sep′tor) **reflexes** help maintain homeostasis when oxygen tension in the blood decreases or when carbon dioxide and hydrogen ion concentrations increase (figure 21.38 and figure 21.39).

Carotid bodies, small organs approximately 1–2 mm in diameter, lie near the carotid sinuses, and several **aortic bodies** lie adjacent to the aorta. Chemoreceptors are located in the



Homeostasis Figure 21.37 Baroreceptor Effects on Blood Pressure

1. Chemoreceptors in the carotid and aortic bodies monitor blood O_2, CO_2, and pH.
2. Chemoreceptors in the medulla oblongata monitor blood CO_2 and pH.
3. Decreased blood O_2, increased CO_2, and decreased pH decrease parasympathetic stimulation of the heart, which increases the heart rate.
4. Decrease blood O_2, increased CO_2, and decreased pH increase sympathetic stimulation of the heart, which increases the heart rate and stroke volume.
5. Increased sympathetic stimulation of blood vessels increases vasoconstriction.



Process Figure 21.38 Chemoreceptor Reflex Control of Blood Pressure

An increase in blood CO_2 and a decrease in pH and blood O_2 result in an increased heart rate and vasoconstriction. A decrease in blood CO_2 and an increase blood pH result in a decreased heart rate and vasodilation.

carotid and aortic bodies. Afferent nerve fibers pass to the medulla oblongata through the glossopharyngeal nerve (IX) from the carotid bodies and through the vagus nerve (X) from the aortic bodies.

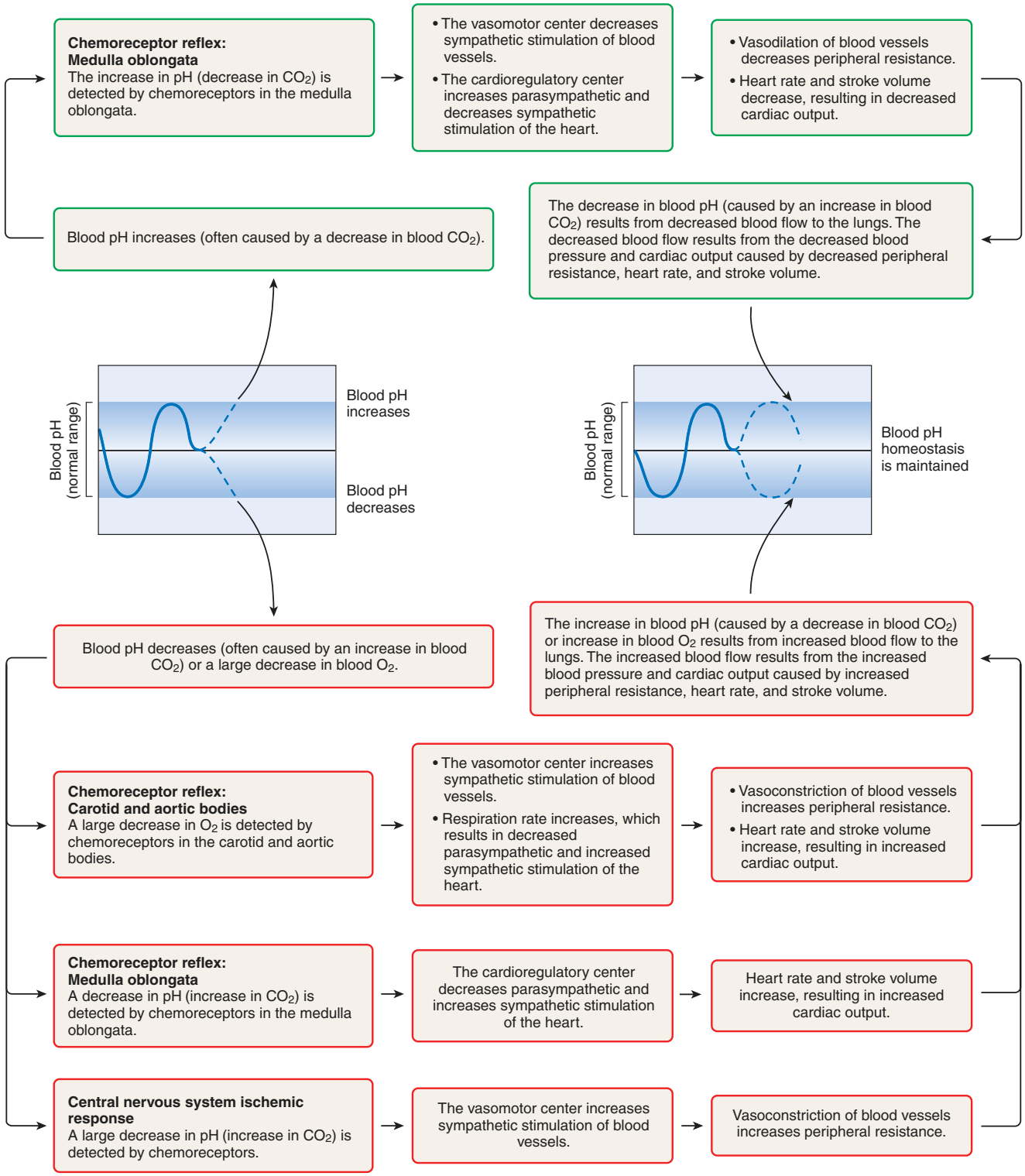
The chemoreceptors receive an abundant blood supply. When oxygen availability decreases in the chemoreceptor cells, the frequency of action potentials increases and stimulates the vasomotor center, resulting in increased vasomotor tone. The chemoreceptors act under emergency conditions and don't regulate the cardiovascular system under resting conditions. They normally don't respond strongly unless oxygen tension in the blood decreases markedly. The chemoreceptor cells are also stimulated by increased carbon dioxide and hydrogen ion concentrations to increase vasomotor tone. The increased vasomotor tone increases the mean arterial pressure. The increased mean arterial pressure increases blood flow through tissues in which blood vessels do not constrict. Blood vessels that are not constricted by the chemoreceptor reflex are blood vessels that deliver blood to the brain and cardiac muscle. Thus, the reflex helps provide an adequate oxygen supply to the brain and the heart when oxygen levels in the blood decrease.

Central Nervous System Ischemic Response

Elevation in blood pressure in response to a lack of blood flow to the medulla oblongata of the brain is called the **central nervous system (CNS) ischemic response.** The CNS ischemic response doesn't play an important role in regulating blood pressure under normal conditions. It functions primarily in response to emergency situations in which blood flow to the brain is severely restricted or when blood pressure falls below approximately 50 mm Hg.

Reduced blood flow results in reduced oxygen, increased carbon dioxide, and reduced pH within the medulla oblongata. Neurons of the vasomotor center are strongly stimulated. As a result, vasoconstriction is stimulated by the vasomotor center, and the systemic blood pressure rises dramatically.

The increase in blood pressure that occurs in response to CNS ischemia increases blood flow to the CNS, provided the blood vessels are intact. However, if severe ischemia lasts longer than a few minutes, metabolism in the brain fails because of the lack of oxygen. The vasomotor center becomes inactive, and extensive vasodilation occurs in the periphery as vasomotor tone decreases. Prolonged ischemia of the medulla oblongata leads to a massive decline in blood pressure and ultimately death.



Homeostasis Figure 21.39 Effects of pH and Gases on Blood Pressure

Long-Term Regulation of Blood Pressure

Regulation of the concentration and volume of blood by the kidneys, the movement of fluid across the wall of blood vessels, and alterations in the volume of the blood vessels all play a central role in the long-term regulation of blood pressure. Some of the long-term regulatory mechanisms begin to respond in minutes, but they continue to function for hours, days, or longer. They adjust the blood pressure precisely and keep it within a narrow range of values for years. Major regulatory mechanisms include the renin-angiotensin-aldosterone mechanism, vasopressin mechanism, atrial natriuretic mechanism, fluid shift mechanism, and stress–relaxation response.

Renin-Angiotensin-Aldosterone Mechanism

The **renin-angiotensin-aldosterone mechanism** helps regulate kidney functions. This mechanism can also influence peripheral resistance by causing vasoconstriction. The kidneys increase urine output as the blood volume and arterial pressure increase, and they decrease urine output as the blood volume and arterial pressure decrease. Increased urine output reduces the blood volume and blood pressure, and decreased urine output resists a further decrease in the blood volume and blood pressure. The control of urine output is not only an important means by which blood pressure is regulated, it continues to operate until the blood pressure is precisely within its normal range of values.

The kidneys release an enzyme called **renin** (rē′nin) into the circulatory system (see chapter 26) from specialized structures called the **juxtaglomerular** (jŭks′tă-glŏ-mer′ū-lăr) **apparatuses** (figure 21.40). Renin acts on plasma proteins, synthesized by the liver, called **angiotensinogen** (an′jē-ō-ten-sin′ō-jen) to split a fragment off one end. The fragment, called **angiotensin** (an-jē-ō-ten′sin) **I,** contains 10 amino acids. Another enzyme, called **angiotensin-converting enzyme,** found primarily in small blood

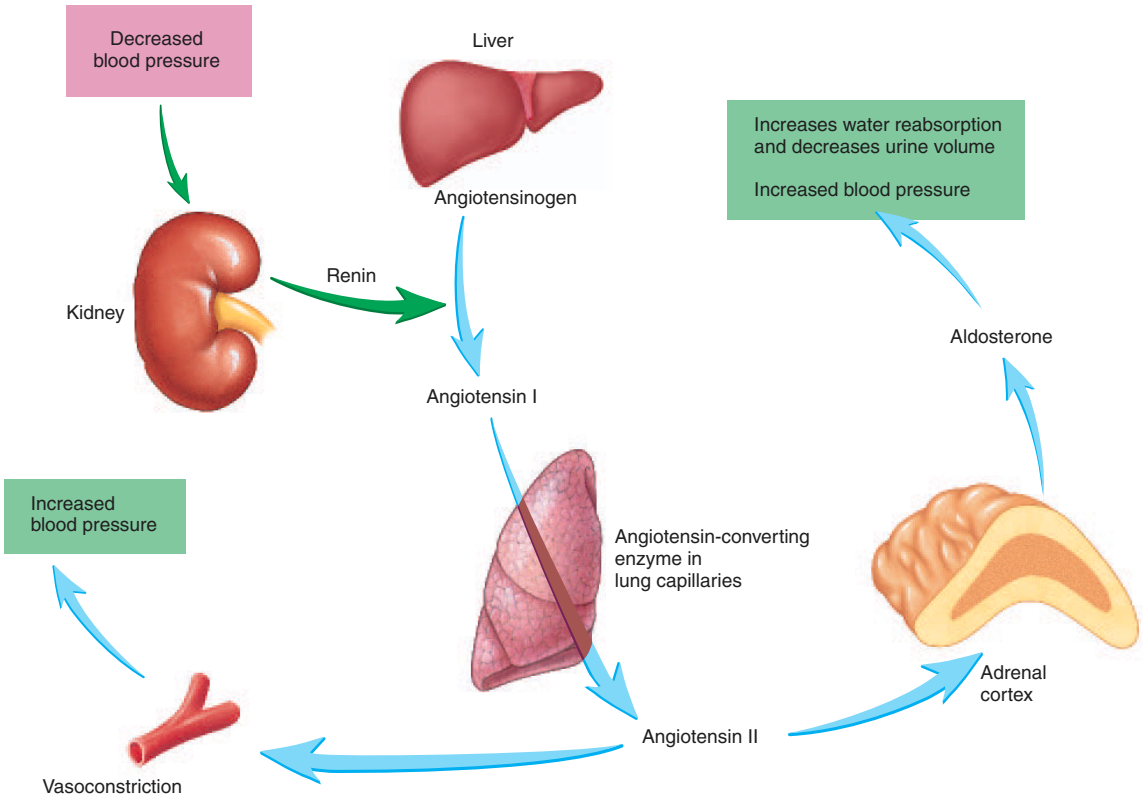


Figure 21.40 The Renin-Angiotensin-Aldosterone Mechanism

Decreased blood pressure is detected by the kidney, resulting in increased renin secretion. The result is vasoconstriction, increased water reabsorption, and decreased urine volume. These changes function to maintain blood pressure.

Clinical Focus Shock

Circulatory shock is inadequate blood flow throughout the body. Failure of mechanisms that function to maintain blood pressure within a normal range of values results in dramatic decreases in blood pressure. As a consequence, tissues suffer damage as a result of too little delivery of oxygen to cells. Severe circulatory shock can damage vital body tissues to the extent that the individual dies.

Depending on its severity, circulatory shock can be divided into three separate stages: (1) nonprogressive or compensated shock, (2) progressive shock, and (3) irreversible shock. All types of circulatory shock exhibit one or more of these stages, regardless of their cause. Several causes of circulatory shock exist, but hemorrhagic, or hypovolemic, shock is used to illustrate the characteristics of each stage.

In **compensated shock,** blood pressure decreases only a moderate amount, and the mechanisms that regulate blood pressure function successfully to reestablish normal blood pressure and blood flow. The baroreceptor reflexes, chemoreceptor reflexes, and ischemia within the medulla oblongata initiate strong sympathetic responses that result in intense vasoconstriction and increased heart rate. As blood volume decreases, the stress–relaxation response of blood vessels causes the blood vessels to contract and helps sustain blood pressure. In response to reduced blood flow through the kidneys, thereby increased amounts of renin are released. The elevated renin release results in a greater rate of angiotensin II formation, causing vasoconstriction and increased aldosterone release from the adrenal cortex. Aldosterone, in turn, promotes water and salt retention by the kidneys, thereby conserving water. In addition, ADH is released from the posterior pituitary gland and enhances the retention of water by the kidneys. Because of the fluid shift mechanism, water also moves from the interstitial spaces and the intestinal lumen to restore normal blood volume. An intense sensation of thirst increases water intake, also helping to elevate normal blood volume.

In mild cases of compensated shock, baroreceptor reflexes can be adequate to compensate for blood loss until blood volume is restored, but in more severe cases, all of the mechanisms described are required to compensate for the blood loss.

In **progressive shock,** the compensatory mechanisms are inadequate to compensate for the loss of blood volume. As a consequence, a positive-feedback cycle develops in which the blood pressure regulatory mechanisms are unable to compensate for circulatory shock. As circulatory shock worsens, regulatory mechanisms become even less able to compensate for the increasing severity of the circulatory shock. The cycle proceeds until the next stage of shock is reached or until medical treatment is applied that assists the regulatory mechanisms in reestablishing adequate blood flow to tissues.

During progressive shock, blood pressure declines to a very low level that is inadequate to maintain blood flow to cardiac muscle; thus, the heart begins to deteriorate. Substances that are toxic to the heart are released from tissues that suffer from severe ischemia. When blood pressure declines to a very low level, blood begins to clot in the small vessels. Eventually blood vessel dilation begins as a result of decreased sympathetic activity and because of the lack of oxygen in capillary beds. Capillary permeability increases under ischemic conditions, allowing fluid to leave the blood vessels and enter the interstitial spaces, and finally intense tissue deterioration begins in response to inadequate blood flow.

vessels of the lung, cleaves two additional amino acids from angiotensin I to produce a fragment consisting of eight amino acids called **angiotensin II,** or **active angiotensin.**

Angiotensin II causes vasoconstriction in arterioles and to some degree in veins. As a result, it increases peripheral resistance and venous return to the heart, both of which function to raise blood pressure. Angiotensin II also stimulates aldosterone secretion from the adrenal cortex. **Aldosterone** (al-dos′ter-ōn) acts on the kidneys to increase the reabsorption of sodium and chloride ions from the filtrate into the extracellular fluid. If antidiuretic hormone (ADH; see chapter 18) is present, water moves by osmosis with the sodium and chloride ions. Consequently, aldosterone causes the kidney to retain solutes such as sodium and chloride ions and water. The results are to decrease the production of urine and to conserve water to prevent further reduction in blood volume caused by the formation of urine. If water intake is adequate, the effect of aldosterone is to increase blood volume (see chapter 26). Angiotensin II also increases the salt appetite, thirst, and ADH secretion.

Decreased blood pressure stimulates renin secretion and increased blood pressure decreases renin secretion. The renin-angiotensin-aldosterone mechanism is important in maintaining blood pressure on a daily basis. It also reacts strongly under conditions of circulatory shock, but it requires many hours to become maximally effective. Its onset of action is not as fast as nervous reflexes or the adrenal medullary response, but its duration of action is longer. Once renin is secreted, it remains active for approximately 1 hour and the effect of aldosterone is much longer (many hours).

Some stimuli can directly stimulate aldosterone secretion. For example, an increased plasma ion concentration of K^+ and a reduced plasma concentration of Na^+ directly stimulate aldosterone secretion from the adrenal cortex (see chapters 18 and 27). The action of aldosterone is to regulate the concentration of these ions in the plasma. A decreased blood pressure and elevated K^+ concentration occur during plasma loss, dehydration, and in response to tissue damage, such as burns and crushing injuries.

Without medical intervention, progressive shock leads to irreversible shock. **Irreversible shock** leads to death, regardless of the amount or type of medical treatment applied. In this stage of shock, the damage to tissues, including cardiac muscle, is so extensive that the patient is destined to die, even if adequate blood volume is reestablished and blood pressure is elevated to its normal value. Irreversible shock is characterized by decreasing heart function and progressive dilation of and increased permeability of peripheral blood vessels.

Patients suffering from shock are normally placed in a horizontal plane, usually with the head slightly lower than the feet, and oxygen is often supplied. **Replacement therapy** consists of transfusions of whole blood, plasma, artificial solutions called plasma substitutes, and physiologic saline solutions administered to increase blood volume. In some circumstances, drugs that enhance vasoconstriction are also administered. Occasionally, such as in patients in anaphylactic (an′ă-fi-lak′tik; allergic) shock, anti-inflammatory substances like glucocorticoids and antihistamines are administered. The basic objective in treating shock is to reverse the condition so that progressive shock is arrested, to prevent it from progressing to the irreversible stage, and to reverse the condition so that normal blood flow through tissues is reestablished.

Several types of shock are classified here by cause:

- **Hemorrhagic shock** is external or internal bleeding that causes a reduction in blood volume.
- **Plasma loss shock** is reduced blood volume that results from a loss of plasma into the interstitial spaces and greatly increased blood viscosity. Plasma loss shock includes **intestinal obstruction,** resulting in the movement of a large amount of plasma from the blood into the intestine, and **severe burns,** resulting in the loss of large amounts of plasma from the burned surface.
- **Dehydration** results from a severe and prolonged shortage of fluid intake.
- **Severe diarrhea** or **vomiting** cause a loss of plasma through the intestinal wall.
- **Neurogenic shock** is a rapid loss of vasomotor tone leading to vasodilation so extensive that a severe decrease in blood pressure results.
- **Anesthesia** includes deep general anesthesia or spinal anesthesia that decreases the activity of the medullary vasomotor center or the sympathetic nerve fibers.
- **Brain damage** leads to an ineffective medullary vasomotor function.
- **Emotional shock (vasovagal syncope)** results from emotions that cause strong parasympathetic stimulation of the heart and results in vasodilation in skeletal muscles and in the viscera.
- **Anaphylactic shock** results from an allergic response that causes the release of inflammatory substances that increase vasodilation and capillary permeability.
- **Septic shock,** or **"blood poisoning,"** results from peritoneal, systemic, and gangrenous infections that cause the release of toxic substances into the circulatory system, depressing the activity of the heart, leading to vasodilation, and increasing capillary permeability.
- **Cardiogenic shock** occurs when the heart stops pumping in response to conditions such as heart attack or electrocution.

ACE Inhibitors and Hypertension

Angiotensin-converting enzyme (ACE) inhibitors are a class of drugs that inhibit angiotensin-converting enzyme, which converts angiotensin I to angiotensin II. These drugs were first identified as components of the venoms of pit vipers. Subsequently, several ACE inhibitors were synthesized. ACE inhibitors are effective in lowering blood pressure in many people who suffer from hypertension and have become one of the drugs commonly administered to people to combat hypertension.

Vasopressin (ADH) Mechanism

The **vasopressin mechanism** works in harmony with the renin-angiotensin-aldosterone mechanism in response to changes in blood pressure (figure 21.41). Baroreceptors are sensitive to changes in blood pressure, and decreases in blood pressure detected by the baroreceptors result in the release of vasopressin (vā-sō-pres′in, vas-ō-pres′in), or ADH, from the posterior pituitary, although the blood pressure must decrease substantially before the mechanism is activated.

ADH acts directly on blood vessels to cause vasoconstriction, although it's not as potent as other vasoconstrictor agents. Within minutes after a rapid and substantial decline in blood pressure, ADH is released in sufficient quantities to help reestablish normal blood pressure. ADH also decreases the rate of urine production by the kidneys, thereby helping to maintain blood volume and blood pressure.

Neurons of the hypothalamus are sensitive to changes in the solute concentration of the plasma. Even small increases in the plasma concentration of solutes directly stimulate hypothalamic neurons that increase ADH secretion (see figure 21.41 and chapter 26). Increases in the concentration of the plasma and decreases in blood pressure stimulate ADH secretion. Dehydration and injuries involving plasma loss, such as extensive burns or crushing injuries, are examples.

Atrial Natriuretic Mechanism

A polypeptide called **atrial natriuretic** (ā′trē-ăl nā′trē-ū-ret′ik) **hormone** is released from cells in the atria of the heart. A major stimulus for its release is increased venous return, which stretches atrial

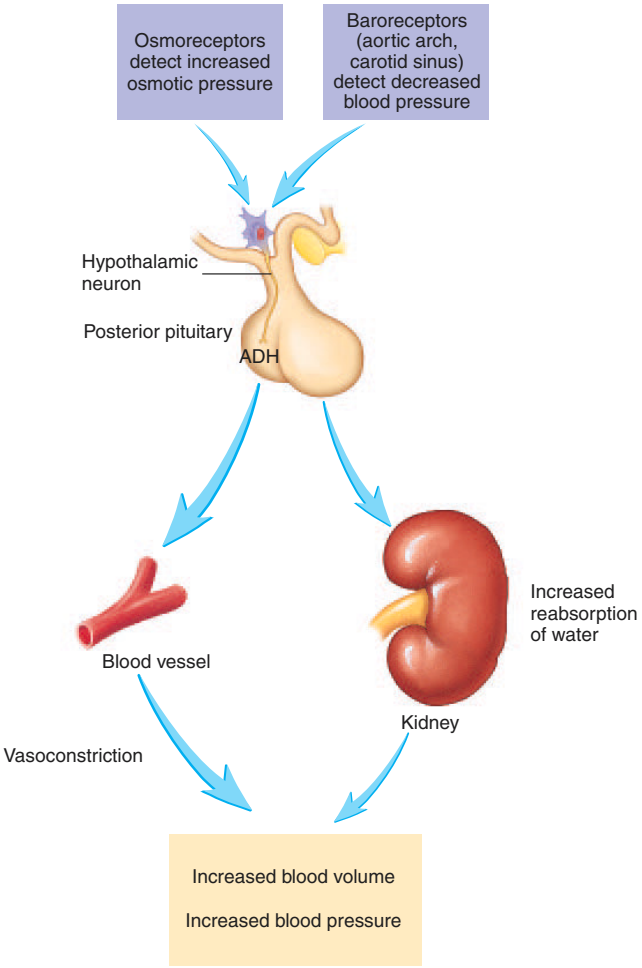


Figure 21.41 The Vasopressin (ADH) Mechanism

Increases in osmolality of blood or decreases in blood pressure result in ADH secretion. ADH increases water reabsorption by the kidney, and large amounts of ADH result in vasoconstriction. These changes function to maintain blood pressure.

cardiac muscle cells. Atrial natriuretic hormone acts on the kidneys to increase the rate of urine production and Na^+ loss in the urine. It also dilates arteries and veins. Loss of water and Na^+ in the urine causes the blood volume to decrease, which decreases venous return, and vasodilation results in a decrease in peripheral resistance. These effects function to cause a decrease in blood pressure.

The renin-angiotensin-aldosterone, vasopressin (ADH), and atrial natriuretic mechanisms work simultaneously to help regulate blood pressure by controlling urine production by the kidneys. If blood pressure drops below 50 mm Hg, the volume of urine produced by the kidneys is reduced to nearly zero. If blood pressure is increased to 200 mm Hg, the urine volume produced is approximately six to eight times greater than normal. The mechanisms that regulate blood pressure in the long term are summarized in figure 21.42.

Fluid Shift Mechanism

The **fluid shift mechanism** begins to act within a few minutes but requires hours to achieve its full functional capacity. It plays a very important role when dehydration develops over several hours, or when a large volume of saline is administered over several hours. The fluid shift mechanism occurs in response to small changes in pressures across capillary walls. As blood pressure increases, some fluid is forced from the capillaries into the interstitial spaces. The movement of fluid into the interstitial spaces helps prevent the development of high blood pressure. As blood pressure falls, interstitial fluid moves into capillaries, which resists a further decline in blood pressure.

The fluid shift mechanism is a powerful method through which blood pressure is maintained because the interstitial volume acts as a reservoir, and it is in equilibrium with the large volume of intercellular fluid.

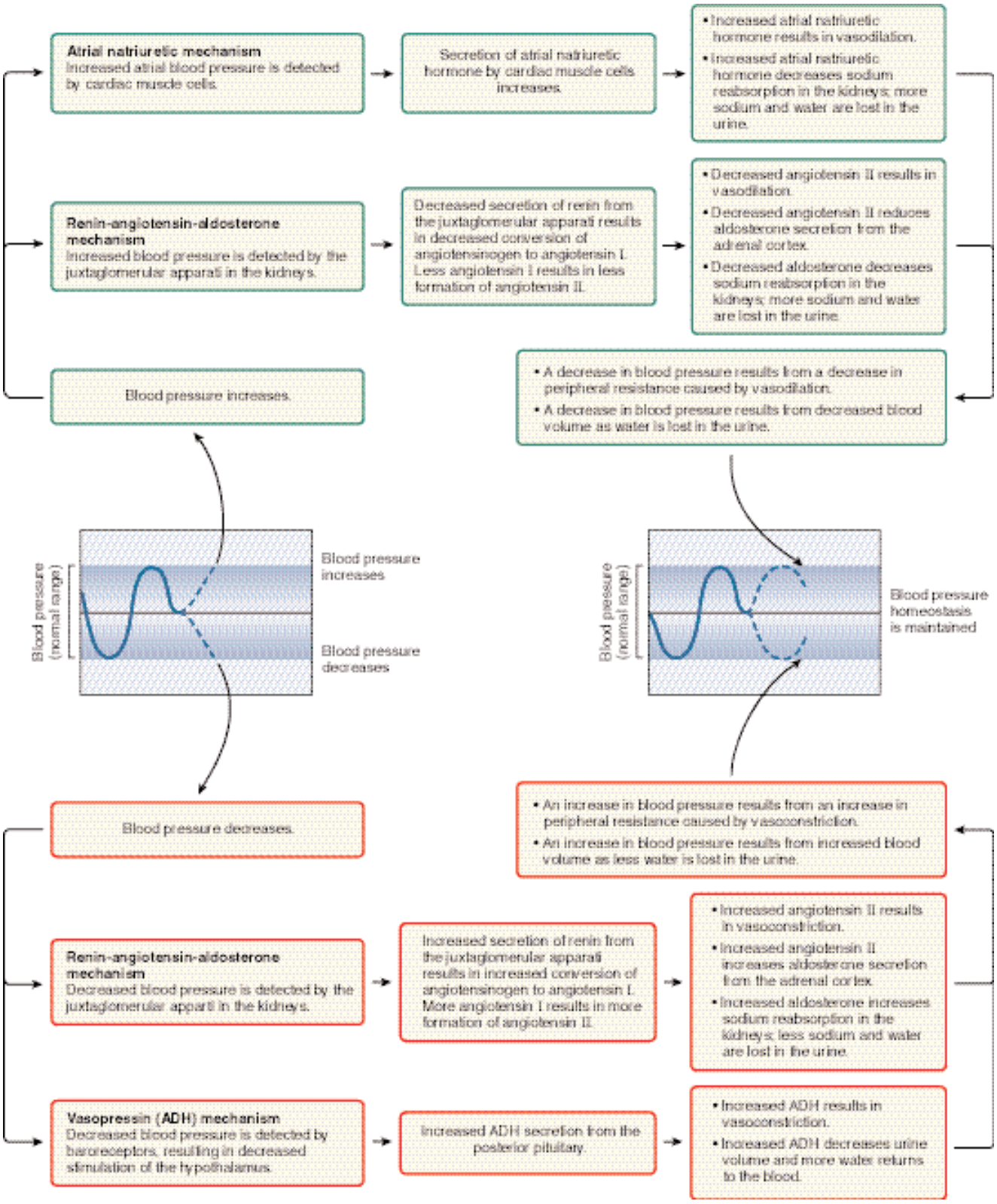
Stress–Relaxation Response

A **stress–relaxation response** is characteristic of smooth muscle cells (see chapter 9). When blood volume suddenly declines, blood pressure also decreases, causing a reduction in the force applied to smooth muscle cells in blood vessel walls. As a result, during the next few minutes to an hour, the smooth muscle cells contract, reducing the volume of the blood vessels and thus resisting a further decline in blood pressure. Conversely, when blood volume increases rapidly, such as during a transfusion, blood pressure increases and smooth muscle cells of the blood vessel walls relax, resulting in a more gradual increase in blood pressure. The stress–relaxation mechanism is most effective when changes in blood pressure occur over a period of many minutes.

P R E D I C T 10

Explain the differences in mechanisms that regulate blood pressure in response to hemorrhage that results in the rapid loss of a large volume of blood compared to hemorrhage that results in the loss of the same volume of blood but over a period of several hours.

39. *Where are baroreceptors located? Describe the response of the baroreceptor reflex when blood pressure increases and decreases.*
40. *Where are the chemoreceptors for carbon dioxide, pH changes, and oxygen located? Describe what happens when oxygen levels in the blood decrease.*
41. *Describe the CNS ischemic response. Under what conditions does this mechanism operate?*
42. *What mechanism is most important for short-term regulation of blood pressure under resting conditions?*
43. *For each of these hormones—epinephrine, norepinephrine, renin, angiotensin, aldosterone, antidiuretic hormone, and atrial natriuretic hormone—state where the hormone is produced and what effects it has on the circulatory system.*
44. *What is fluid shift, and what does it accomplish? Describe the stress–relaxation response of a blood vessel.*
45. *Discuss two ways that the kidneys are involved in long-term regulation of blood pressure.*



Homeostasis Figure 21.42 Control of Blood Pressure Long-Term (Slow-Acting) Mechanisms

S U M M A R Y

General Features of Blood Vessel Structure (p. 712)

1. Blood flows from the heart through elastic arteries, muscular arteries, and arterioles to the capillaries.
2. Blood returns to the heart from the capillaries through venules, small veins, and large veins.

Capillaries

1. The entire circulatory system is lined with simple squamous epithelium called endothelium. Capillaries consist only of endothelium.
2. Capillaries are surrounded by loose connective tissue, the adventitia, that contains pericapillary cells.
3. Three types of capillaries exist.
   - Fenestrated capillaries have pores called fenestrae that extend completely through the cell.
   - Sinusoidal capillaries are large-diameter capillaries with large fenestrae.
   - Continuous capillaries do not have fenestrae.
4. Materials pass through the capillaries in several ways: between the endothelial cells, through the fenestrae, and through the plasma membrane.
5. Blood flows from arterioles through metarterioles and then through the capillary network. Venules drain the capillary network.
   - Smooth muscle in the arterioles, metarterioles, and precapillary sphincters regulates blood flow into the capillaries.
   - Blood can pass rapidly through the thoroughfare channel.

Structure of Arteries and Veins

1. Except for capillaries and venules, blood vessels have three layers. The inner tunica intima consists of endothelium, basement membrane, and internal elastic lamina.
   - The tunica media, the middle layer, contains circular smooth muscle and elastic fibers.
   - The outer tunica adventitia is connective tissue.
2. The thickness and the composition of the layers vary with blood vessel type and diameter.
   - Large elastic arteries are thin-walled with large diameters. The tunica media has many elastic fibers and little smooth muscle.
   - Muscular arteries are thick-walled with small diameters. The tunica media has abundant smooth muscle and some elastic fibers.
   - Arterioles are the smallest arteries. The tunica media consists of smooth muscle cells and a few elastic fibers.
   - Venules are composed of endothelium surrounded by a few smooth muscle cells.
   - Small veins are venules covered with a layer of smooth muscle.
   - Medium-sized veins and large veins contain less smooth muscle and fewer elastic fibers than arteries of the same size.
3. Valves prevent the backflow of blood in the veins.

Vasa Vasorum

1. Vasa vasorum are blood vessels that supply the tunica adventitia and tunica media.
2. Arteriovenous anastomoses allow blood to flow from arteries to veins without passing through the capillaries. They function in temperature regulation.

Nerves

Sympathetic nerve fibers supply the smooth muscle of the tunica media.

Aging of the Arteries

Arteriosclerosis results from a loss of elasticity in the aorta, large arteries, and coronary arteries.

Pulmonary Circulation (p. 715)

The pulmonary circulation moves blood to and from the lungs. The pulmonary trunk arises from the right ventricle and divides to form the pulmonary arteries, which project to the lungs. From the lungs, the pulmonary veins return to the left atrium.

Systemic Circulation: Arteries (p. 715)

Aorta

The aorta leaves the left ventricle to form the ascending aorta, aortic arch, and descending aorta (consisting of the thoracic and abdominal aortae).

Coronary Arteries

Coronary arteries supply the heart.

Arteries to the Head and the Neck

1. The brachiocephalic, left common carotid, and left subclavian arteries branch from the aortic arch to supply the head and the upper limbs. The brachiocephalic artery divides to form the right common carotid and the right subclavian arteries. The vertebral arteries branch from the subclavian arteries.
2. The common carotid arteries and the vertebral arteries supply the head.
   - The common carotid arteries divide to form the external carotids, which supply the face and mouth, and the internal carotids, which supply the brain.
   - The vertebral arteries join within the cranial cavity to form the basilar artery, which supplies the brain.

Arteries of the Upper Limb

1. The subclavian artery continues (without branching) as the axillary artery and then as the brachial artery. The brachial artery divides into the radial and ulnar arteries.
2. The radial artery supplies the deep palmar arch, and the ulnar artery supplies the superficial palmar arch. Both arches give rise to the digital arteries.

Thoracic Aorta and Its Branches

The thoracic aorta has visceral branches that supply the thoracic organs and parietal branches that supply the thoracic wall.

Abdominal Aorta and Its Branches

1. The abdominal aorta has visceral branches that supply the abdominal organs and parietal branches that supply the abdominal wall.
2. The visceral branches are paired and unpaired. The paired arteries supply the kidneys, adrenal glands, and gonads. The unpaired arteries supply the stomach, spleen, and liver (celiac trunk); the small intestine and upper part of the large intestine (superior mesenteric); and the lower part of the large intestine (inferior mesenteric).

Arteries of the Pelvis

1. The common iliac arteries arise from the abdominal aorta, and the internal iliac arteries branch from the common iliac arteries.
2. The visceral branches of the internal iliac arteries supply pelvic organs, and the parietal branches supply the pelvic wall and floor and the external genitalia.

Arteries of the Lower Limb

1. The external iliac arteries branch from the common iliac arteries.
2. The external iliac artery continues (without branching) as the femoral artery and then as the popliteal artery. The popliteal artery divides to form the anterior and posterior tibial arteries.

3. The posterior tibial artery gives rise to the fibular (peroneal) and plantar arteries. The plantar arteries form the plantar arch from which the digital arteries arise.

Systemic Circulation: Veins (p. 728)

1. The three major veins returning blood to the heart are the superior vena cava (head, neck, thorax, and upper limbs), the inferior vena cava (abdomen, pelvis, and lower limbs), and the coronary sinus (heart).
2. Veins are of three types: superficial, deep, and sinuses.

Veins Draining the Heart

Coronary veins enter the coronary sinus or the right atrium.

Veins of the Head and Neck

1. The internal jugular veins drain the venous sinuses of the anterior head and neck.
2. The external jugular veins and the vertebral veins drain the posterior head and neck.

Veins of the Upper Limb

1. The deep veins are the small ulnar and radial veins of the forearm, which join the brachial veins of the arm. The brachial veins drain into the axillary vein.
2. The superficial veins are the basilic, cephalic, and median cubital. The basilic vein becomes the axillary vein, which then becomes the subclavian vein. The cephalic vein drains into the axillary vein.

Veins of the Thorax

The left and right brachiocephalic veins and the azygos veins return blood to the superior vena cava.

Veins of the Abdomen and Pelvis

1. Ascending lumbar veins from the abdomen join the azygos and hemiazygos veins.
2. Vessels from the kidneys, adrenal gland, and gonads directly enter the inferior vena cava.
3. Vessels from the stomach, intestines, spleen, and pancreas connect with the hepatic portal vein. The hepatic portal vein transports blood to the liver for processing. Hepatic veins from the liver join the inferior vena cava.

Veins of the Lower Limb

1. The deep veins are the fibular (peroneal), anterior and posterior tibials, popliteal, femoral, and external iliac.
2. The superficial veins are the small and great saphenous veins.

Dynamics of Blood Circulation (p. 740)

Laminar and Turbulent Flow in Vessels

Blood flow through vessels normally is streamlined, or laminar. Turbulent flow is disruption of laminar flow.

Blood Pressure

1. Blood pressure is a measure of the force exerted by blood against the blood vessel wall. Blood moves through vessels because of blood pressure.
2. Blood pressure can be measured by listening for Korotkoff sounds produced by turbulent flow in arteries as pressure is released from a blood pressure cuff.

Blood Flow

Blood flow is the amount of blood that moves through a vessel in a given period. Blood flow is directly proportional to pressure differences and is inversely proportional to resistance.

Poiseuille's Law

Resistance is the sum of all the factors that inhibit blood flow. Resistance increases when viscosity increases and when blood vessels become smaller in diameter or increase in length.

Viscosity

1. Viscosity is the resistance of a liquid to flow. Most of the viscosity of blood results from red blood cells.
2. The viscosity of blood increases when the hematocrit increases.

Critical Closing Pressure and Laplace's Law

1. As pressure in a vessel decreases, the force holding it open decreases, and the vessel tends to collapse. The critical closing pressure is the pressure at which a blood vessel closes.
2. Laplace's law states that the force acting on the wall of a blood vessel is proportional to the diameter of the vessel times blood pressure.

Vascular Compliance

Vascular compliance is a measure of the change in volume of blood vessels produced by a change in pressure. The venous system has a large compliance and acts as a blood reservoir.

Physiology of Systemic Circulation (p. 744)

The greatest volume of blood is contained in the veins. The smallest volume is in the arterioles.

Cross-Sectional Area of Blood Vessels

As the diameter of vessels decreases, their total cross-sectional area increases, and the velocity of blood flow through them decreases.

Pressure and Resistance

Blood pressure averages 100 mm Hg in the aorta and drops to 0 mm Hg in the right atrium. The greatest drop occurs in the arterioles, which regulate blood flow through tissues.

Pulse Pressure

1. Pulse pressure is the difference between systolic and diastolic pressures. Pulse pressure increases when stroke volume increases or vascular compliance decreases.
2. Pulse pressure waves travel through the vascular system faster than the blood flows. Pulse pressure can be used to take the pulse.

Capillary Exchange and Regulation of Interstitial Fluid Volume

1. Blood pressure, capillary permeability, and osmosis affect movement of fluid from the capillaries.
2. A net movement of fluid occurs from the blood into the tissues. The fluid gained by the tissues is removed by the lymphatic system.

Functional Characteristics of Veins

Venous return to the heart increases because of an increase in blood volume, venous tone, and arteriole dilation.

Blood Pressure and the Effect of Gravity

In a standing person, hydrostatic pressure caused by gravity increases blood pressure below the heart and decreases pressure above the heart.

Control of Blood Flow in Tissues (p. 749)

Local Control of Blood Flow by the Tissues

1. Blood flow through a tissue is usually proportional to the metabolic needs of the tissue. Exceptions are tissues that perform functions that require additional blood.
2. Control of blood flow by the metarterioles and precapillary sphincters can be regulated by vasodilator substances or by lack of nutrients.

3. Only large changes in blood pressure have an effect on blood flow through tissues.
4. If the metabolic activity of a tissue increases, the number and the diameter of capillaries in the tissue increases over time.

Nervous and Hormonal Regulation of Local Circulation

1. The sympathetic nervous system (vasomotor center in the medulla) controls blood vessel diameter. Other brain areas can excite or inhibit the vasomotor center.
2. Vasomotor tone is a state of partial contraction of blood vessels.
3. The nervous system is responsible for routing the flow of blood and maintaining blood pressure.
4. Sympathetic action potentials stimulate epinephrine and norepinephrine release from the adrenal medulla, and these hormones cause vasoconstriction in most blood vessels.

Regulation of Mean Arterial Pressure (p. 753)

Mean arterial pressure (MAP) is proportional to cardiac output times peripheral resistance.

Short-Term Regulation of Blood Pressure

1. Baroreceptors are sensory receptors sensitive to stretch.
   - Baroreceptors are located in the carotid sinuses and the aortic arch.
   - The baroreceptor reflex changes peripheral resistance, heart rate, and stroke volume in response to changes in blood pressure.
2. Chemoreceptors are sensory receptors sensitive to oxygen, carbon dioxide, and pH levels in the blood.
3. Epinephrine and norepinephrine are released from the adrenal medulla as a result of sympathetic stimulation. They increase heart rate, stroke volume, and vasoconstriction.
4. The CNS ischemic response results from high carbon dioxide or low pH levels in the medulla and increases peripheral resistance.

Long-Term Regulation of Blood Pressure

1. Renin-angiotensin-aldosterone mechanism. Renin is released by the kidneys in response to low blood pressure. Renin promotes the production of angiotensin II, which causes vasoconstriction and an increase in aldosterone secretion.
2. Vasopressin (ADH) mechanism. ADH released from the posterior pituitary in response to a substantial decrease in blood pressure causes vasoconstriction.
3. Atrial natriuretic mechanism. Atrial natriuretic hormone is released from the cardiac muscle cells when atrial blood pressure increases. It stimulates an increase in urinary production, causing a decrease in blood volume and blood pressure.
4. Fluid shift mechanism. Fluid shift is a movement of fluid from the interstitial spaces into capillaries in response to a decrease in blood pressure to maintain blood volume.
5. Stress–relaxation response. The stress–relaxation response is an adjustment of the smooth muscles of blood vessels in response to a change in blood volume.

REVIEW AND COMPREHENSION

1. Given these blood vessels:
   1. arteriole
   2. capillary
   3. elastic artery
   4. muscular artery
   5. vein
   6. venule

   Choose the arrangement that lists the blood vessels in the order a red blood cell passes through them as it leaves the heart, travels to a tissue, and returns to the heart.
   a. 3,4,2,1,5,6
   b. 3,4,1,2,6,5
   c. 4,3,1,2,5,6
   d. 4,3,2,1,6,5
   e. 4,2,3,5,1,6
2. Given these structures:
   1. metarteriole
   2. precapillary sphincter
   3. thoroughfare channel

   Choose the arrangement that lists the structures in the order a red blood cell encounters them as it passes through a tissue.
   a. 1,3,2
   b. 2,1,3
   c. 2,3,1
   d. 3,1,2
   e. 3,2,1
3. In which of these blood vessels are elastic fibers present in the largest amounts?
   a. large arteries
   b. medium arteries
   c. arterioles
   d. venules
   e. large veins
4. Comparing and contrasting arteries and veins, veins have
   a. thicker walls.
   b. a greater amount of smooth muscle than arteries.
   c. a tunica media and arteries do not.
   d. valves and arteries do not.
   e. all of the above.
5. The structure that supplies the walls of blood vessels with blood is the
   a. venous shunt.
   b. tunic channel.
   c. arteriovenous anastomosis.
   d. vasa vasorum.
   e. coronary artery.
6. Given these blood vessels:
   1. aorta
   2. inferior vena cava
   3. pulmonary arteries
   4. pulmonary veins

   Which vessels carry oxygen-rich blood?
   a. 1,3
   b. 1,4
   c. 2,3
   d. 2,4
   e. 3,4
7. Given these arteries:
   1. basilar
   2. common carotid
   3. internal carotid
   4. vertebral

   Which of these arteries have *direct* connections with the cerebral arterial circle (circle of Willis)?
   a. 1,2
   b. 2,4
   c. 1,3
   d. 3,4
   e. 2,3

8. Given these blood vessels:
   1. axillary artery
   2. brachial artery
   3. brachiocephalic artery
   4. radial artery
   5. subclavian artery

   Choose the arrangement that lists the vessels in order, going from the aorta to the right hand.
   a. 2,5,4,1
   b. 5,2,1,4
   c. 5,3,1,4,2
   d. 3,5,1,2,4
   e. 4,5,1,2,3
9. A branch of the aorta that supplies the liver, stomach, and spleen is the
   a. celiac trunk.
   b. common iliac.
   c. inferior mesenteric.
   d. superior mesenteric.
   e. renal.
10. Given these arteries:
    1. common iliac
    2. external iliac
    3. femoral
    4. popliteal

    Choose the arrangement that lists the arteries in order going from the aorta to the knee.
    a. 1,2,3,4
    b. 1,2,4,3
    c. 2,1,3,4
    d. 2,1,4,3
    e. 3,1,2,4
11. Given these veins:
    1. brachiocephalic
    2. internal jugular
    3. superior vena cava
    4. venous sinus

    Choose the arrangement that lists the veins in order going from the brain to the heart.
    a. 1,2,4,3
    b. 2,4,1,3
    c. 2,4,3,1
    d. 4,2,1,3
    e. 4,2,3,1
12. Blood returning from the arm to the subclavian vein passes through which of these veins?
    a. cephalic
    b. basilic
    c. brachial
    d. both a and b
    e. all of the above
13. Given these blood vessels:
    1. inferior mesenteric vein
    2. superior mesenteric vein
    3. hepatic portal vein
    4. hepatic vein

    Choose the arrangement that lists the vessels in order going from the small intestine to the inferior vena cava.
    a. 1,3,4
    b. 1,4,3
    c. 2,3,4
    d. 2,4,3
    e. 3,1,4
14. Given this list of veins:
    1. small saphenous
    2. great saphenous
    3. fibular (peroneal)
    4. posterior tibial

    Which are superficial veins?
    a. 1,2
    b. 1,3
    c. 2,3
    d. 2,4
    e. 3,4
15. If you could increase any of these factors that affect blood flow by twofold, which one would cause the greatest increase in blood flow?
    a. blood viscosity
    b. the pressure gradient
    c. vessel radius
    d. vessel length
16. Vascular compliance is
    a. greater in arteries than in veins.
    b. the increase in vessel volume divided by the increase in vessel pressure.
    c. the pressure at which blood vessels collapse.
    d. proportional to the diameter of the blood vessel times pressure.
    e. all of the above.
17. The resistance to blood flow is greatest in the
    a. aorta.
    b. arterioles.
    c. capillaries.
    d. venules.
    e. veins.
18. Pulse pressure
    a. is the difference between systolic and diastolic pressure.
    b. increases when stroke volume increases.
    c. increases as vascular compliance decreases.
    d. all of the above.
19. Veins
    a. increase their volume because of their large compliance.
    b. increase venous return to the heart when they vasodilate.
    c. vasodilate because of increased sympathetic stimulation.
    d. all of the above.
20. Local direct control of blood flow through a tissue
    a. maintains an adequate rate of flow despite large changes in arterial blood pressure.
    b. results from relaxation and contraction of precapillary sphincters.
    c. occurs in response to a buildup in carbon dioxide in the tissues.
    d. occurs in response to a decrease in oxygen in the tissues.
    e. all of the above.
21. An increase in mean arterial pressure can result from
    a. an increase in peripheral resistance.
    b. an increase in heart rate.
    c. an increase in stroke volume.
    d. all of the above.
22. In response to an increase in mean arterial pressure, the baroreceptor reflex causes
    a. an increase in sympathetic nervous system activity.
    b. a decrease in peripheral resistance.
    c. stimulation of the vasomotor center.
    d. vasoconstriction.
    e. an increase in cardiac output.

23. When blood oxygen levels markedly decrease, the chemoreceptor reflex causes
    a. peripheral resistance to decrease.
    b. mean arterial blood pressure to increase.
    c. vasomotor tone to decrease.
    d. vasodilation.
    e. all of the above.
24. When blood pressure is suddenly decreased a small amount (10 mm Hg), which of these mechanisms are activated to restore blood pressure to normal levels?
    a. chemoreceptor reflexes
    b. baroreceptor reflexes
    c. CNS ischemic response
    d. all of the above
25. A sudden release of epinephrine from the adrenal medulla
    a. increases heart rate.
    b. increases stroke volume.
    c. causes vasoconstriction in visceral blood vessels.
    d. all of the above.
26. When blood pressure decreases,
    a. renin secretion increases.
    b. angiotensin II formation decreases.
    c. aldosterone secretion decreases.
    d. all of the above.
27. In response to a decrease in blood pressure,
    a. ADH secretion increases.
    b. the kidneys decrease urine production.
    c. blood volume increases.
    d. all of the above.
28. In response to a decrease in blood pressure,
    a. more fluid than normal enters the tissues (fluid shift mechanism).
    b. smooth muscles in blood vessels relax (stress–relaxation response).
    c. the kidneys retain more salts and water than normal.
    d. all of the above.
29. A patient is found to have severe arteriosclerosis of his renal arteries, which reduced renal blood pressure. Which of these is consistent with that condition?
    a. hypotension
    b. hypertension
    c. decreased vasomotor tone
    d. exaggerated sympathetic stimulation of the heart
    e. both a and c
30. During exercise the blood flow through skeletal muscle may increase up to 20-fold. However, the cardiac output does not increase that much. This occurs because of
    a. vasoconstriction in the viscera.
    b. vasoconstriction in the skin (at least temporarily).
    c. vasodilation of skeletal muscle blood vessels.
    d. both a and b.
    e. all of the above.

Answers in Appendix F

CRITICAL THINKING

1. For each of the following destinations, name all the arteries that a red blood cell would encounter if it started its journey in the left ventricle.
    a. Posterior interventricular groove of the heart
    b. Anterior neck to the brain (give two ways)
    c. Posterior neck to the brain (give two ways)
    d. External skull
    e. Tip of the fingers of the left hand (What other blood vessel would be encountered if the trip were through the right upper limb?)
    f. Anterior compartment of the leg
    g. Liver
    h. Small intestine
    i. Urinary bladder
2. For each of the following starting places, name all the veins that a red blood cell would encounter on its way back to the right atrium.
    a. Anterior interventricular groove of the heart (give two ways)
    b. Venous sinus near the brain
    c. External posterior of skull
    d. Hand (return deep and superficial)
    e. Foot (return deep and superficial)
    f. Stomach
    g. Kidney
    h. Left inferior wall of the thorax
3. In a study of heart valve functions, it's necessary to inject a dye into the right atrium of the heart by inserting a catheter into a blood vessel and moving the catheter into the right atrium. What route would you suggest? If you wanted to do this procedure into the left atrium, what would you do differently?
4. In endurance-trained athletes, the hematocrit can be lower than normal because plasma volume increases more than red blood cell numbers increase. Explain why this condition would be beneficial.
5. All the blood that passes through the aorta, except the blood that flows into the coronary vessels, returns to the heart through the venae cavae. (*Hint:* The diameter of the aorta is 26 mm, and the diameter of a vena cava is 32 mm.) Explain why the resistance to blood flow in the aorta is greater than the resistance to blood flow in the venae cavae. Because the resistances are different, explain why blood flow can be the same.
6. As blood vessels increase in diameter, the amount of smooth muscle decreases and the amount of connective tissue increases. Explain why. (*Hint:* Remember Laplace's law.)
7. A patient is suffering from edema in the lower right limb. Explain why massage helps to remove the excess fluid.
8. A very short nursing student is asked to measure the blood pressure of a very tall person. She decides to measure the blood pressure at the level of the tall person's foot while he is standing. What artery does she use? After taking the blood pressure, she decides that the tall person is suffering from hypertension because the systolic pressure is 200 mm Hg. Is her diagnosis correct? Why or why not?
9. Mr. D. was suffering from severe cirrhosis of the liver and hepatitis. He develop over a period of time severe edema. Explain how decreased liver function can result in edema.
10. During hyperventilation, carbon dioxide is "blown off," and carbon dioxide levels in the blood decrease. What effect does this decrease have on blood pressure? Explain. What symptoms do you expect to see as a result?
11. Epinephrine causes vasodilation of blood vessels in cardiac muscle but vasoconstriction of blood vessels in the skin. Explain why this is a beneficial arrangement.
12. One cool evening, Skinny Dip jumps into a hot Jacuzzi. Predict what happens to Skinny's heart rate.

Answers in Appendix G

ANSWERS TO PREDICT QUESTIONS

1. Arteriosclerosis slowly reduces blood flow through the carotid arteries and therefore the amount of blood that flows to the brain. As the resistance to flow increases in the carotid arteries during the late stages of arteriosclerosis, the blood flow to the brain is compromised, resulting in reduced oxygen delivery. Confusion, loss of memory, and loss of the ability to perform other normal brain functions occur.
2. a. Vasoconstriction of blood vessels in the skin in response to exposure to cold results in a decreased flow of blood through the skin and in a dramatic increase in resistance (see Poiseuille's law). Vasoconstriction makes the skin appear pale.
   b. Vasodilation of blood vessels in the skin results in increased blood flow through the skin. Vasodilation makes the skin appear flushed or red in color.
   c. In a patient with polycythemia vera, the hematocrit increases dramatically. As a result, the viscosity of the blood increases, which increases resistance to flow. Consequently, flow decreases or a greater pressure is needed to maintain the same flow.
3. An aneurysm in the aorta is a major problem because the tension applied to the aneurysm becomes greater as its size increases (see Laplace's law). The aneurysm usually develops because of a weakness in the wall of the aorta. Arteriosclerosis complicates the matter by making the wall of the artery less elastic and by increasing the systolic blood pressure. The decreased elasticity and the increased blood pressure increase the probability that the aneurysm will rupture.
4. Premature beats of the heart and ectopic beats result in contraction of the heart muscle before the heart has had time to fill to its normal capacity. Consequently, a reduced stroke volume occurs, which results in a weak pulse in response to that premature contraction. Other contractions and the resulting pulses are normal. Strong bounding pulses in a person who received too much saline solution in an intravenous transfusion result from an increase in venous return to the heart because of the increased volume of fluid in the circulatory system. Because of the increased venous return (increased preload), the heart contracts with greater force and produces a larger stroke volume. The strong bounding pulse results from the increased stroke volume. Weak pulses occur in response to hemorrhagic shock because of a decreased venous return. The heart does not fill with blood between contractions (decreased preload); the stroke volume is therefore reduced, and the pulse is weak.
5. Arterial blood pressure can increase substantially without resulting in edema. As arterial blood pressure increases the precapillary sphincters constrict to match capillary blood flow with the metabolic needs of tissues. Thus, the capillary blood pressure doesn't change substantially even though the blood pressure may increase to high levels. The blood pressure must increase above approximately 175 mm Hg before edema results. In contrast, a small increase in venous pressure leads to edema because there is no sphincter muscle that protects the capillary from an increase in pressure. Thus, small increases in venous pressure can lead to edema. Blockage of veins by blood clots or increases in venous pressure due to heart failure result in edema.
6. Keeping the legs elevated reduces the blood pressure in the capillaries of the legs because of the effect of gravity on blood flow. A major effect is that the force that moves fluid out of the capillary is decreased. As a result, the net movement of fluid out of the arterial ends decreases and the net movement into the venous ends of the capillaries increases. Therefore excess interstitial fluid is carried away from the legs. In addition, the effect of gravity increases lymph flow into the lymphatic capillaries, which also increases the rate that interstitial fluid is drained from the legs.
7. Reactive hyperemia can be explained on the basis of any of the theories for the local control of blood pressure. When a blood vessel is occluded, nutrients are depleted, and waste products accumulate in tissue that is suffering from a lack of adequate blood supply. Both of these effects cause vasodilation and a greatly increased blood flow through the area after the occlusion has been removed.
8. While this athlete is relaxing, the sympathetic stimulation of arteries in her skeletal muscles, arteries in her digestive system, and large veins decrease. As a result, vasoconstriction increases in the arteries of her muscles, and vasodilation occurs in blood vessels of her digestive system and in the large veins. Blood flow decreased to her skeletal muscles, and blood flow increased to her digestive system. In addition, more blood accumulated in the large veins. Consequently, venous return to the heart decreased, which is consistent with the reduced cardiac output.
9. During a headstand, gravity acting on the blood causes the blood pressure in the area of the aortic arch and carotid sinus baroreceptors to increase. The increased pressure activates the baroreceptor reflexes, increasing parasympathetic stimulation of the heart and decreasing sympathetic stimulation. Thus the heart rate decreases. Because standing on one's head also causes blood from the periphery to run downhill to the heart, the venous return increases, causing the stroke volume to increase because of Starling's law of the heart. Some peripheral vasodilation also can occur because the elevated baroreceptor pressure causes a decrease in vasomotor tone.
10. The baroreceptor reflex, ADH, and renin-angiotensin-aldosterone mechanisms function similarly in both cases. The fluid shift mechanism, however, is important when the loss of blood occurs over several hours, but it doesn't operate within a short period. The fluid shift mechanism plays a very important role in the maintenance of blood volume when blood loss or dehydration develops over several hours. When the blood pressure decreases, interstitial fluids pass into the capillaries, which prevents a further decline in blood pressure. The fluid shift mechanism is a powerful method through which blood pressure is maintained because the interstitial fluid acts as a fluid reservoir. The stress–relaxation mechanism responds to changes in blood pressure, but it is most responsive to sudden changes in blood volume and it responds within minutes to hours.