

Polymer cast of blood vessels of the eye (SEM)

CHAPTER

20

The Circulatory System: Blood Vessels and Circulation

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Brushing Up

To understand this chapter, it is important that you understand or brush up on the following concepts:

- Set point and dynamic equilibrium in homeostasis (p. 17)
- Diffusion (p. 106)
- Equilibrium between filtration and osmosis (p. 107)
- Transcytosis (p. 113)
- Viscosity and osmolarity of blood (pp. 680–681)
- Principles of pressure and flow (p. 733)
- Autonomic effects on the heart (p. 738)

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The route taken by the blood after it leaves the heart was a point of much confusion until the seventeenth century. Chinese emperor Huang Ti (2697–2597 B.C.E.) correctly believed that it flowed in a complete circuit around the body and back to the heart. But in the second century, Roman physician Claudius Galen (129–c. 199) argued that it flowed back and forth in the veins, like air in the bronchial tubes. He believed that the liver received food from the small intestine and converted it to blood, the heart pumped the blood through the veins to all other organs, and those organs consumed the blood.

Huang Ti was right, but the first experimental demonstration of this did not come until the seventeenth century. English physician William Harvey (1578–1657) studied the filling and emptying of the heart in snakes, tied off the vessels above and below the heart to observe the effects on cardiac filling and output, and measured cardiac output in a variety of living animals. He concluded that (1) the heart pumps more blood in half an hour than there is in the entire body, (2) not enough food is consumed to account for the continual production of so much blood, and (3) since the planets orbit the sun and (as he believed) the human body is modeled after the solar system, it follows that the blood orbits the body. So for a peculiar combination of experimental and superstitious reasons, Harvey argued that the blood returns to the heart rather than being consumed by the peripheral organs. He could not explain how, since the microscope had yet to be developed and he did not know of capillaries—later discovered by Antony van Leeuwenhoek and Marcello Malpighi.

Harvey published his findings in 1628 in a short but elegant book entitled *Exercitio Anatomica de Motu Cordis et Sanguinis in Animalibus* (*Anatomical Studies on the Motion of the Heart and Blood in Animals*). This landmark in the history of biology and medicine was the first experimental study of animal physiology. But so entrenched were the ideas of Aristotle and Galen in the medical community, and so strange was the idea of doing experiments on living animals, that Harvey's contemporaries rejected his ideas. Indeed, some of them regarded him as a crackpot because his conclusion flew in the face of common sense—if the blood was continually recirculated and not consumed by the tissues, they reasoned, then what purpose could it possibly serve?

Harvey lived to a ripe old age, served as physician to the kings of England, and later did important work in embryology. His case is one of the most interesting in biomedical history, for it shows how empirical science overthrows old theories and spawns better ones, and how common sense and blind allegiance to authority can interfere with acceptance of the truth. But most importantly, Harvey's contributions represent the birth of experimental physiology—the method that generated most of the information in this book.

General Anatomy of the Blood Vessels

Objectives

When you have completed this section, you should be able to

- trace the route usually taken by the blood from the heart and back again;

- describe some variations on this route;
- describe the structure of a blood vessel; and
- describe the different types of arteries, capillaries, and veins.

Circulatory Routes

At its simplest, the usual route of blood flow is heart → arteries → arterioles → capillaries → venules → veins → heart. Blood usually passes through only one network of capillaries from the time it leaves the heart until the time it returns (fig. 20.1a). But there are exceptions, notably portal systems and anastomoses. In a **portal system** (fig. 20.1b), blood flows through two consecutive capillary networks before returning to the heart. One portal system connects the hypothalamus and anterior pituitary (see chapter 17). Others are found in the kidneys and between the intestines and liver; the latter system is detailed in table 20.13.

An **anastomosis** is a point where two blood vessels merge. In an **arteriovenous anastomosis (shunt)**, blood flows from an artery directly into a vein and bypasses the capillaries (fig. 20.1c). Shunts occur in the fingers, palms, toes, and ears, where they reduce heat loss in cold weather by allowing warm blood to bypass these exposed surfaces. Unfortunately, this makes these poorly perfused areas more susceptible to frostbite. In an **arterial anastomosis**,

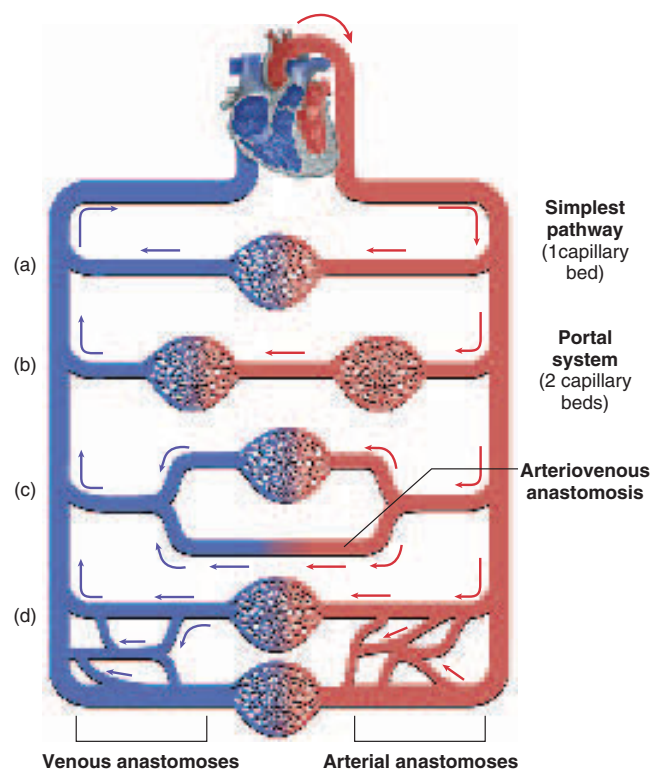


Figure 20.1 Variations in Circulatory Pathways.

two arteries merge and provide *collateral* (alternative) routes of blood supply to a tissue (fig. 20.1d). Those of the coronary circulation were mentioned in chapter 19. They are also common around joints where movement may temporarily obstruct one pathway. **Venous anastomoses** are more common. They provide several alternative routes of drainage from an organ, so blockage of a vein is rarely as life-threatening as blockage of an artery. Several arterial and venous anastomoses are described later in this chapter.

The Vessel Wall

The walls of the arteries and veins have three layers called *tunics* (figs. 20.2 and 20.3):

1. The **tunica externa (tunica adventitia¹)** is the outermost layer. It consists of loose connective

¹ *advent* = added to

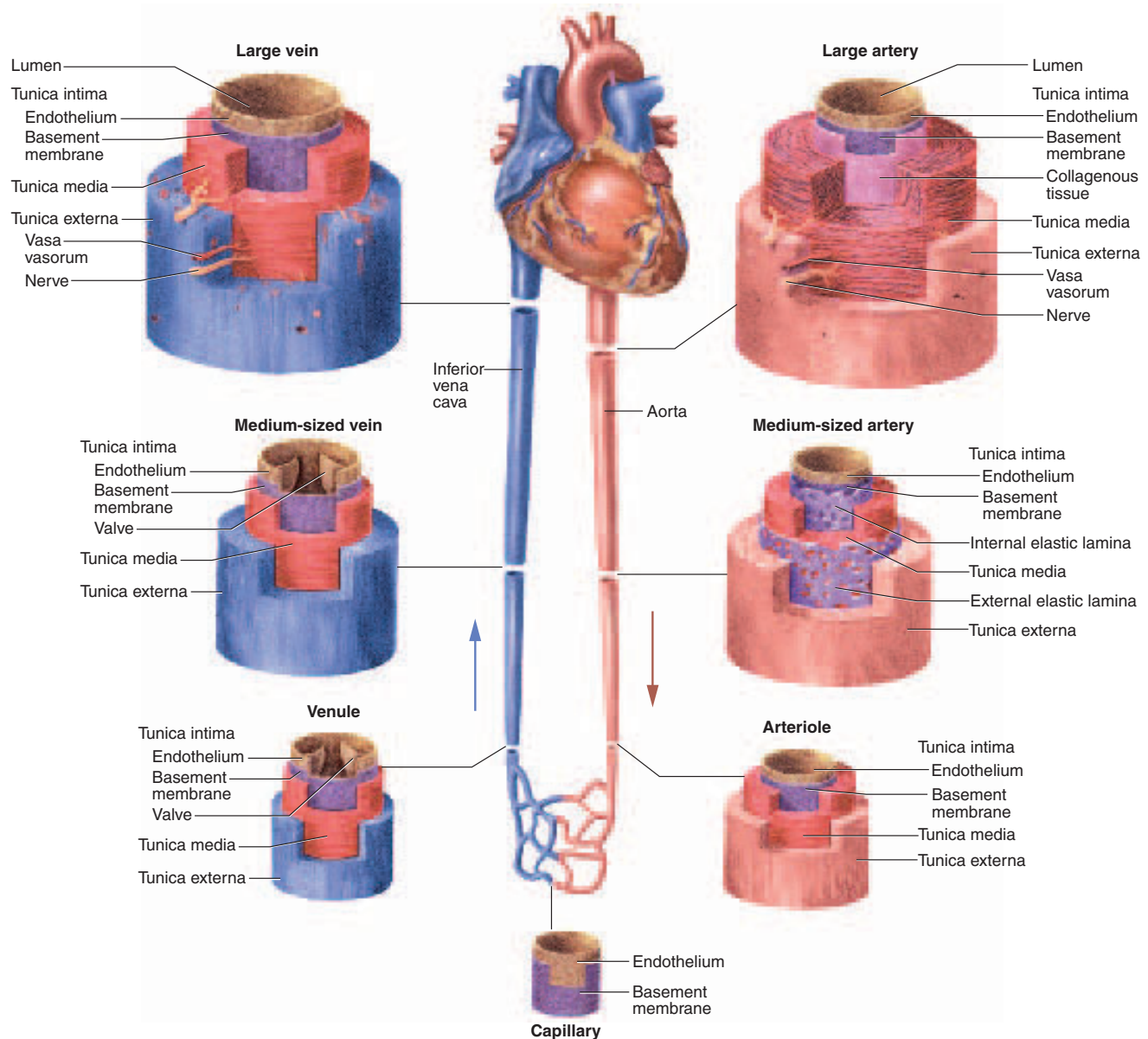


Figure 20.2 The Structure of Arteries and Veins.
Why are elastic laminae found in arteries but not in veins?

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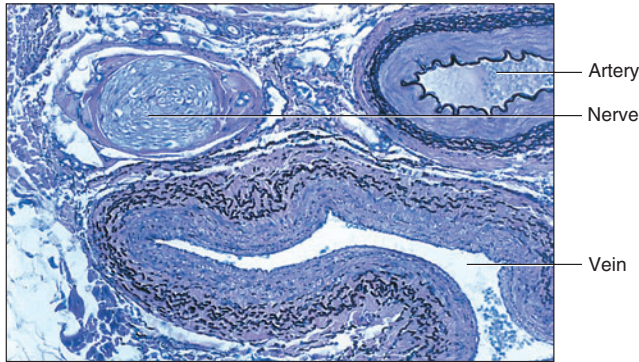


Figure 20.3 A Neurovascular Bundle. A small artery, small vein, and nerve traveling together in a common sheath of connective tissue.

tissue that often merges with that of neighboring blood vessels, nerves, or other organs. It anchors the vessel and provides passage for small nerves, lymphatic vessels, and smaller blood vessels. Small vessels called the **vasa vasorum**² (VAY-za vay-SO-rum) supply blood to at least the outer half of the wall of a larger vessel. Tissues of the inner half of the wall are thought to be nourished by diffusion from blood in the lumen.

2. The **tunica media**, the middle layer, is usually the thickest. It consists of smooth muscle, collagen, and sometimes elastic tissue. The smooth muscle is responsible for the vasoconstriction and vasodilation of blood vessels.
3. The **tunica intima (tunica interna)**, the inner layer, is exposed to the blood. It consists of a simple squamous **endothelium** overlying a basement membrane and a sparse layer of fibrous tissue. The endothelium acts as a selectively permeable barrier to blood solutes, and it secretes vasoconstrictors and vasodilators to be considered later. It also provides a smooth inner lining that normally repels blood cells and platelets. However, platelets may adhere to a damaged endothelium. During inflammation, leukocytes also adhere loosely to it by means of *cell-adhesion molecules* produced by the endothelial cells (see chapter 21).

Arteries and Metarterioles

Arteries are constructed to withstand the surges of blood pressure generated by ventricular systole. They are more muscular than veins and appear relatively round in tissue sections. They are divided into three categories by size,

but of course there is a smooth gradation from one category to the next:

1. **Conducting (elastic) arteries** are the largest. Some examples are the pulmonary arteries, aorta, and common carotid arteries. Their tunica media consists of numerous sheets of elastic tissue, perforated like slices of Swiss cheese, alternating with thin layers of smooth muscle, collagen, and elastic fibers. Conducting arteries expand when the ventricles pump blood into them during systole, and recoil during diastole. This lessens the fluctuations in blood pressure exerted on smaller arteries downstream.
2. **Distributing (muscular) arteries** are smaller branches farther away from the heart that distribute blood to specific organs. You could compare a conducting artery to an interstate highway and distributing arteries to the exit ramps and state highways that serve specific towns. Distributing arteries typically have 25 to 40 layers of smooth muscle cells constituting about three-quarters of the wall thickness. Most arteries to which we give names are in these first two size classes. The brachial, femoral, and splenic arteries are examples of distributing arteries.
3. **Resistance (small) arteries** are usually too variable in number and location to be given names. They exhibit up to 25 layers of smooth muscle cells and relatively little elastic tissue. Their tunica media is thicker in proportion to the lumen than that of larger arteries. The smallest of these arteries, about 40 to 200 μm in diameter and with only one to three layers of smooth muscle, are the **arterioles**. For reasons discussed later, they are the primary points at which the body controls the relative amounts of blood directed to various organs.

Metarterioles³ are short vessels that link arterioles and capillaries. Instead of a continuous tunica media, they have individual muscle cells spaced a short distance apart, each forming a **precapillary sphincter** that encircles the entrance to a capillary.

Capillaries

Capillaries (fig. 20.4) are the “business end” of the circulatory system. All the rest of the system exists to serve them, because capillaries are almost the only point in the circulatory system where materials are exchanged between the blood and tissue fluid. Capillaries are ideally suited to their role. They consist only of endothelium and a basement membrane. Capillaries have walls as thin as 0.2 to 0.4 μm . They average about 5 μm in diameter at the prox-

²*vasa* = vessels + *vasorum* = of the vessels

³*meta* = beyond, next in a series

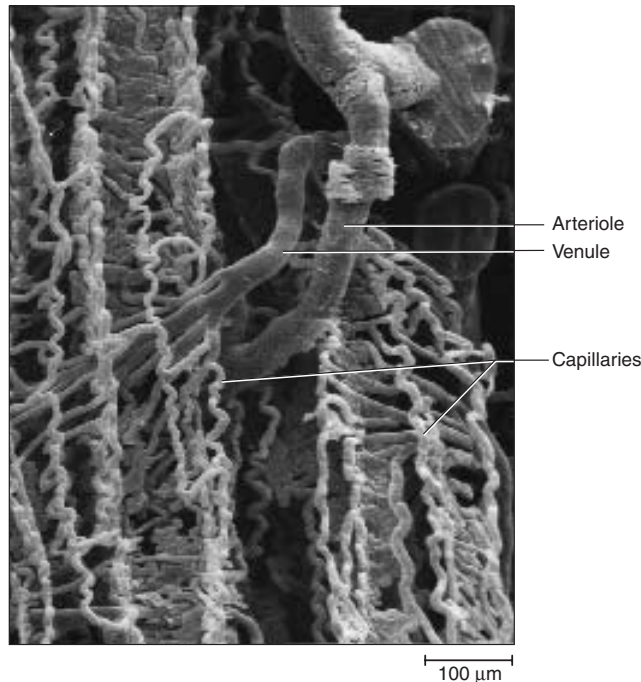


Figure 20.4 Vascular Cast of Blood Vessels in Human Skeletal Muscle. This was prepared by injecting the vessels with a polymer, digesting away all tissue to leave a replica of the vessels, and photographing the cast through the SEM. From R. G. Kessel and R. H. Kardon, *Tissues and Organs: A Text-Atlas of Scanning Electron Microscopy* (W. H. Freeman & Co., 1979).

imal (arterial) end, widen to about $9\ \mu\text{m}$ in diameter at the distal (venous) end, and often branch along the way. (Recall that an erythrocyte is about $7\ \mu\text{m}$ in diameter.) The number of capillaries has been estimated at a billion and their total surface area at $6,300\ \text{m}^2$. But a more important point is that scarcely any cell in the body is more than 60 to $80\ \mu\text{m}$ away from the nearest capillary. There are a few exceptions. Capillaries are scarce in tendons and ligaments and absent from cartilage, epithelia, and the cornea and lens of the eye.

Capillary Beds

Capillaries are organized in groups called **capillary beds**—usually 10 to 100 capillaries supplied by a single metarteriole (fig. 20.5). The metarteriole continues through the bed as a **thoroughfare channel** leading directly to a venule. Capillaries arise from the proximal end of the metarteriole and lead into its distal end or directly into the venule.

There is a precapillary sphincter at the entrance to each capillary. When the sphincters are open, the capillaries are well perfused with blood and they engage in exchanges

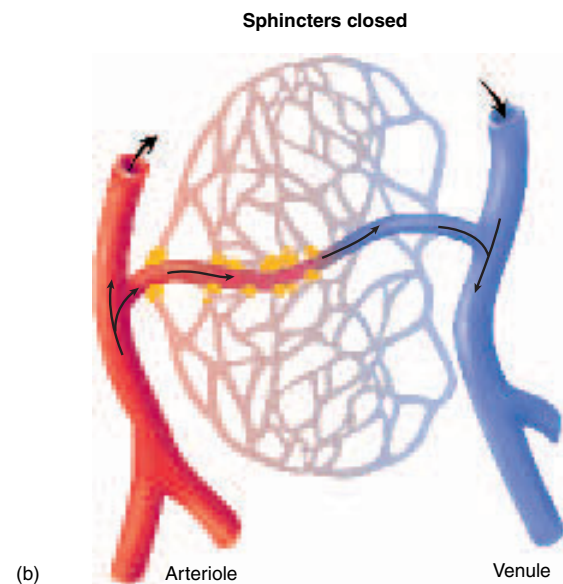
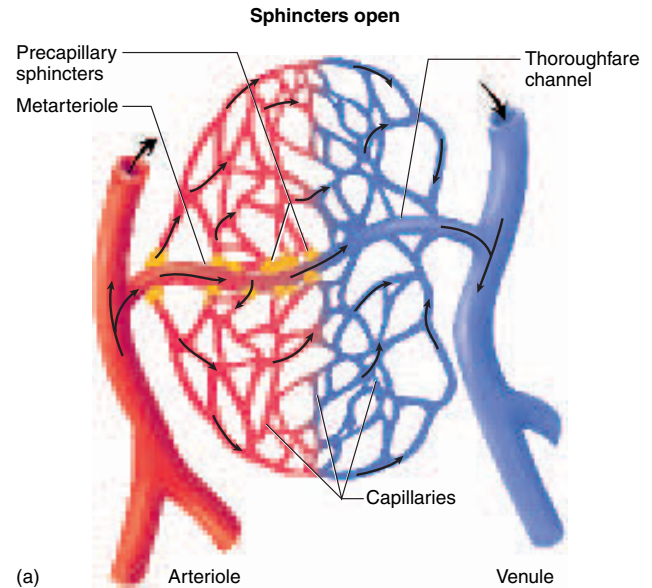


Figure 20.5 Control of Perfusion of a Capillary Bed. (a) Precapillary sphincters dilated and capillaries well perfused. (b) Precapillary sphincters closed, with blood bypassing the capillaries.

with the tissue fluid. When the sphincters are closed, blood bypasses the capillaries, flows through the thoroughfare channel to a venule, and does not engage in significant fluid exchange. There is not enough blood in the body to fill the entire vascular system at once; consequently, about three-quarters of the body's capillaries are closed at any given time. The shifting of blood flow from one capillary bed to another is discussed later in the chapter.

Types of Capillaries

Two types of capillaries are distinguished by the ease with which they allow substances to pass through their walls and by structural differences that account for their greater or lesser permeability:

1. **Continuous capillaries** occur in most tissues, such as skeletal muscle. Their endothelial cells, held together by tight junctions, form an uninterrupted tube. The cells usually have narrow **intercellular clefts** about 4 nm wide between them. Small solutes, such as glucose, can pass through these clefts, but plasma proteins, other large molecules, and formed elements are held back. The continuous capillaries of the brain lack intercellular clefts and have more complete tight junctions that form the blood-brain barrier discussed in chapter 14.
2. **Fenestrated capillaries** have endothelial cells that are riddled with holes called **fenestrations**⁴ (**filtration pores**) (fig. 20.6). Fenestrations are about 20 to 100 nm in diameter and are usually covered by a thin mucoprotein diaphragm. They allow for the rapid passage of small molecules but still retain proteins and larger particles in the bloodstream. Fenestrated capillaries are important in organs that engage in rapid absorption or filtration—the kidneys, endocrine glands, small intestine, and choroid plexuses of the brain, for example.

Sinusoids are irregular blood-filled spaces in the liver, bone marrow, spleen, and some other organs. They are twisted, tortuous passageways that conform to the shape of the surrounding tissue. Some of them are continuous capillaries with very thin walls; others are fenestrated capillaries with extraordinarily large pores that allow the blood plasma to come into direct contact with the perivascular cells. Even proteins and blood cells can pass through these pores; this is how albumin, clotting factors, and other proteins synthesized by the liver enter the blood and how newly formed blood cells enter the circulation from the bone marrow and lymphatic organs.

Veins

After flowing through the capillaries, blood collects in the distal end of the thoroughfare channel and flows into a venule. In the venous circulation, blood flows from smaller vessels into progressively larger ones; hence, instead of giving off *branches* as arteries do, veins receive smaller *tributaries*, just as a river receives water from the many streams that form its tributaries.

Venules range from about 15 to 100 μm in diameter. The proximal part of a venule has only a few fibroblasts

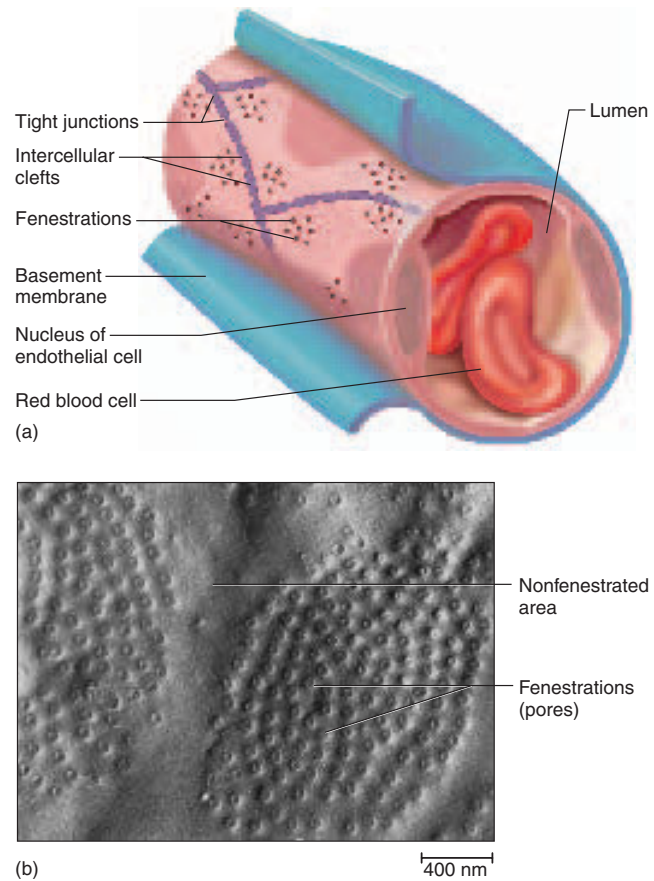


Figure 20.6 A Fenestrated Capillary. (a) Structure of the capillary wall. (b) Surface view of a fenestrated endothelial cell. The cell has patches of fenestrations separated by nonfenestrated areas.

around it and is quite porous; therefore venules, like capillaries, exchange fluid with the surrounding tissues. Farther along, a venule acquires a tunica media of smooth muscle. Even the largest veins, however, have relatively sparse muscular and elastic tissue compared to arteries.

Venous sinuses are veins with especially thin walls, large lumens, and no smooth muscle. Examples include the coronary sinus of the heart and the dural sinuses of the brain.

Because they are farther away from the heart, veins have much lower blood pressure than arteries. In large arteries, it averages 90 to 100 mmHg and surges to 120 mmHg during systole, whereas in veins it averages about 10 mmHg and fluctuates very little with the heartbeat. This has significant implications for the form and function of veins:

- Since they need not withstand high pressure, veins have thinner walls than arteries, with less muscular and elastic tissue. They collapse when empty and look

⁴*Fenestra* = window

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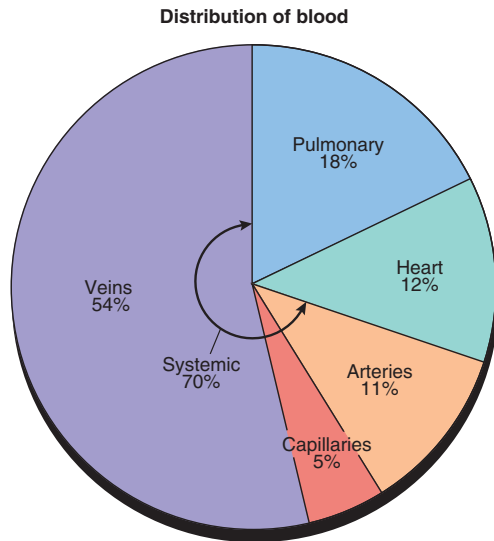


Figure 20.7 Average Distribution of Blood in a Resting Adult.

relatively flattened or irregular in histological sections (see fig. 20.3).

- Since their walls are so thin, veins expand more easily and accommodate more blood than arteries do. About 54% of the blood is found in the systemic veins at rest (fig. 20.7); veins are therefore called *capacitance vessels*.
- The pressure in the veins is not high enough to push blood upward against the pull of gravity to the heart. The upward flow of blood depends in part on the massaging action of skeletal muscles and the presence of one-way **venous valves** that keep the blood from dropping down again when the muscles relax (see fig. 20.2). These valves, similar to the semilunar valves of the heart, occur especially in medium veins of the arms and legs; they are absent from very small and very large veins, veins of the ventral body cavity, and veins of the brain. Varicose veins result in part from the failure of these valves (see insight 20.1).

Insight 20.1 Clinical Application

Varicose Veins

In people who stand for long periods, such as dentists and hair-dressers, blood tends to pool in the lower limbs and stretch the veins. This is especially true of superficial veins, which are not surrounded by supportive tissue. Stretching pulls the cusps of the venous valves farther apart until the valves become incompetent to prevent the backflow of blood. As the veins become further distended, their walls

grow weak and they develop into *varicose veins* with irregular dilations and twisted pathways. Obesity and pregnancy also promote development of varicose veins by putting pressure on large veins of the pelvic region and obstructing drainage from the legs. Varicose veins sometimes develop because of hereditary weakness of the valves. With less drainage of blood, tissues of the leg and foot may become edematous and painful. *Hemorrhoids* are varicose veins of the anal canal.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

1. Explain how an anastomosis and a portal system differ from the simple artery → capillary → vein scheme of circulation.
2. Name the three tunics of a typical blood vessel and explain how they differ from each other.
3. Describe the route of blood flow through a capillary bed.
4. Contrast the two types of capillaries.
5. Explain why many veins have valves but arteries do not.

Blood Pressure, Resistance, and Flow

Objectives

When you have completed this section, you should be able to

- explain the relationship between blood pressure, resistance, and flow;
- describe how blood pressure is expressed and how pulse pressure and mean arterial pressure are calculated;
- describe three factors that determine resistance to blood flow;
- explain how vasomotion influences blood flow; and
- describe some local, neural, and hormonal influences on vasomotion.

Blood flow is the amount of blood flowing through an organ, tissue, or blood vessel in a given time (such as mL/min). **Perfusion** is the flow per given volume or mass of tissue (such as mL/min/g). Perfusion governs the speed of oxygen and nutrient delivery to a tissue and the speed of waste removal. If flow does not keep pace with the metabolic rate of a tissue, the likely result is tissue necrosis and possibly death of the individual. In a resting individual, *total* flow is quite constant and is equal to cardiac output (typically 5.25 L/min). Flow through individual organs, however, varies from minute to minute as blood is redirected from one organ to another. Great variations in regional flow can occur with little or no change in total flow.

Hemodynamics, the physical principles of blood flow, are based mainly on pressure and resistance. The greater the pressure difference (ΔP) between two points, the greater the flow; the greater the resistance (R), the less the flow—in summary, $F \propto \Delta P/R$. Therefore, to understand

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the flow of blood, we must consider the factors that affect pressure and resistance.

Blood Pressure

Blood pressure (BP) is the force that the blood exerts against a vessel wall. It can be measured within a blood vessel or heart chamber by inserting a catheter or needle connected to an external manometer (pressure-measuring device). For routine clinical purposes, however, the measurement of greatest interest is the systemic arterial BP at a point close to the heart. As mentioned in chapter 19, we customarily measure it with a sphygmomanometer at the brachial artery of the arm. It is easy to encircle the arm with a pressure cuff, and this artery is sufficiently close to the heart to reflect the maximum arterial BP found anywhere in the systemic circuit.

Two pressures are recorded: **systolic pressure** is the peak arterial BP attained during ventricular systole, and **diastolic pressure** is the minimum arterial BP between heartbeats. For a healthy person aged 20 to 30, these pressures are typically about 120 and 75 mmHg, respectively. Arterial BP is written as a ratio of systolic over diastolic pressure: 120/75.

The difference between systolic and diastolic pressure is called **pulse pressure** (not to be confused with pulse rate). For the preceding BP, pulse pressure would be $120 - 75 = 45$ mmHg. This is an important measure of the stress exerted on small arteries by the pressure surges generated by the heart. Another measure of stress on the blood vessels is the **mean arterial pressure (MAP)**—the mean pressure you would obtain if you took measurements at several intervals (say every 0.1 sec) throughout the cardiac cycle. Since diastole lasts longer than systole, MAP is not simply the average of systolic and diastolic pressures. The best estimate of MAP is the sum of diastolic pressure and one-third of the pulse pressure. For a blood pressure of 120/75, $MAP \approx 75 + 45/3 = 90$ mmHg, a typical value for vessels at the level of the heart. MAP varies, however, with the influence of gravity. In a standing adult, it is about 62 mmHg in the major arteries of the head and 180 mmHg in major arteries of the ankle.

Hypertension (high BP) is commonly considered to be a chronic resting blood pressure higher than 140/90 (see insight 20.4, p. 792). (*Transient* high BP resulting from emotion or exercise is not hypertension.) Among other effects, it can weaken the small arteries and cause **aneurysms**⁵ (AN-you-rizms) (see insight 20.2). **Hypotension** is chronic low resting BP. It may be a consequence of blood loss, dehydration, anemia, or other factors and is normal in people approaching death.

Insight 20.2 Clinical Application

Aneurysm

An aneurysm is a weak point in a blood vessel or in the heart wall. It forms a thin-walled, bulging sac that pulsates with each beat of the heart and may eventually rupture. In a *dissecting aneurysm*, blood pools between the tunics of a vessel and separates them, usually because of degeneration of the tunica media. The most common sites of aneurysms are the abdominal aorta, the renal arteries, and the arterial circle at the base of the brain. Even without hemorrhaging, aneurysms can cause pain or death by putting pressure on brain tissue, nerves, adjacent veins, pulmonary air passages, or the esophagus. Other consequences include neurological disorders, difficulty in breathing or swallowing, chronic cough, or congestion of the tissues with blood. Aneurysms sometimes result from congenital weakness of the blood vessels and sometimes from trauma or bacterial infections such as syphilis. The most common cause, however, is the combination of atherosclerosis and hypertension.

The ability of the arteries to distend and recoil during the cardiac cycle is important in modulating arterial BP. If the arteries were rigid tubes, pressure would rise much higher in systole and drop to nearly zero in diastole. Blood throughout the circulatory system would flow and stop, flow and stop, thus putting great stress on the small vessels. But when the conducting arteries are healthy, they expand with each systole and absorb some of the force of the ejected blood. Then, when the heart is in diastole, their elastic recoil exerts pressure on the blood and prevents the BP from dropping to zero. The combination of expansion and recoil in the arteries maintains a steady flow of blood downstream, in the capillaries, throughout the cardiac cycle. Thus, the elastic arteries “smooth out” the pressure fluctuations and reduce stress on the smaller arteries.

Nevertheless, blood flow in the arteries is *pulsatile*. Blood in the aorta rushes forward at 120 cm/sec during systole and an average speed of 40 cm/sec over the cardiac cycle. When measured at points farther away from the heart, systolic and diastolic pressures are lower and there is less difference between them (fig. 20.8). In capillaries and veins, the blood flows at a steady speed without pulsation because the pressure surges have been damped out by the distance traveled and the elasticity of the arteries. This is why an injured vein exhibits relatively slow, steady bleeding, whereas blood spurts intermittently from a severed artery. In the inferior vena cava near the heart, however, venous flow fluctuates with the respiratory cycle for reasons explained later, and there is some fluctuation in the jugular veins of the neck.

Think About It

Explain how the histological structure of large arteries relates to their ability to stretch during systole and recoil during diastole.

⁵aneurysm = widening

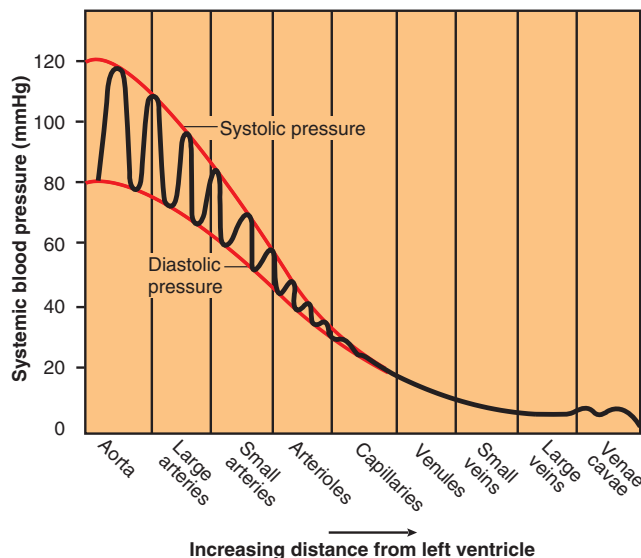


Figure 20.8 Changes in Blood Pressure Related to Distance from the Heart. Because of arterial elasticity and the effect of friction against the vessel wall, all measures of blood pressure decline with distance—systolic pressure, diastolic pressure, pulse pressure, and mean arterial pressure. There is no pulse pressure beyond the arterioles, but there are slight pressure oscillations in the venae cavae caused by the respiratory pump.

Blood pressure rises with age (table 20.1) as the arteries become less distensible and absorb less systolic force. Atherosclerosis also stiffens the arteries and leads to a rise in BP.

Blood pressure is determined mainly by cardiac output, blood volume, and peripheral resistance. The regulation of cardiac output and blood volume are discussed in chapters 19 and 24, respectively. Here, we turn our attention to peripheral resistance.

Resistance

A moving fluid has no pressure unless it encounters at least some resistance. Thus, pressure and resistance are not independent factors in blood flow—rather, pressure is affected by resistance, and flow is affected by both. **Peripheral resistance** is the resistance that the blood encounters in the vessels as it travels away from the heart. It results from the friction of blood against the walls of the vessels and is proportional to three variables: *blood viscosity*, *vessel length*, and *vessel radius*.

Blood Viscosity

Blood viscosity (“thickness” of the blood) is due mainly to erythrocytes and albumin. A deficiency of erythrocytes

Table 20.1 Normal Arterial Blood Pressure at Various Ages *

Age (years)	Male	Female
1	96/66	95/65
5	92/62	92/62
10	103/69	103/70
15	112/75	112/76
20	123/76	116/72
25	125/78	117/74
30	126/79	120/75
40	129/81	127/80
50	135/83	137/84
60	142/85	144/85
70	145/82	159/85
80	145/82	157/83

*Average for healthy individuals

(anemia) or albumin (hypoproteinemia) decreases peripheral resistance and speeds up blood flow. If viscosity increases (as a result of polycythemia or dehydration, for example), resistance increases and flow declines.

Vessel Length

The farther a liquid travels through a tube, the more cumulative friction it encounters; thus, pressure and flow decline with distance. Partly for this reason, if you were to measure MAP in a reclining person, you would obtain a higher value in the arm, for example, than in the ankle. (This would not be true in a standing person because of the influence of gravity, explained earlier.) A strong pulse in the dorsal pedal artery of the foot is a good sign of adequate cardiac output. If perfusion is good at that distance from the heart, it is likely to be good elsewhere in the systemic circulation.

Vessel Radius

In a healthy individual, blood viscosity is quite stable, and of course vessel lengths do not change in the short term. Therefore, the only significant way of controlling peripheral resistance from moment to moment is by adjusting the radius of the blood vessels. A change in vessel radius is called **vasomotion**. This includes **vasoconstriction**, the narrowing of a vessel, and **vasodilation**, the widening of a vessel. Vasoconstriction occurs when the smooth muscle of the tunica media contracts. Vasodilation occurs when this muscle relaxes and allows the blood pressure within the vessel to push its walls outward.

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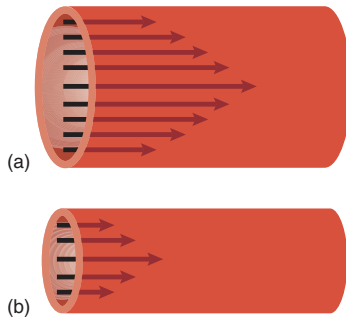


Figure 20.9 Laminar Flow and the Effect of Vessel Radius. Blood flows more slowly near the vessel wall, as indicated by shorter arrows, than it does near the center of the vessel. (a) When vessel radius is large, the average velocity of flow is high. (b) When the radius is less, the average velocity is lower because a larger portion of the blood is slowed down by friction against the vessel wall.

The effect of vessel radius on blood flow is related to the friction of the moving blood against the walls of the vessel. Blood normally exhibits smooth, silent **laminar**⁶ **flow**. That is, it flows in “layers”—faster near the center of a vessel, where it encounters less friction, and slower near the walls, where it drags against the vessel. You can observe a similar effect from the vantage point of a riverbank. The current may be very swift in the middle of a river but quite sluggish near shore, where the water encounters more friction against the riverbank and bottom. When a blood vessel dilates, a greater portion of the blood is in the middle of the stream and the average flow may be quite swift. When the vessel constricts, more of the blood is close to the wall and the average flow is slower (fig. 20.9).

Thus the radius of a vessel markedly affects blood velocity. Indeed, blood flow is proportional not merely to vessel radius but to the *fourth power* of radius—that is, $F \propto r^4$. This makes vessel radius a very potent factor in the control of flow. Arterioles can constrict to as little as one-third of their fully relaxed radius (fig. 20.10). For the sake of simplicity, consider a hypothetical blood vessel with a 1 mm radius when maximally constricted and a 3 mm radius when completely relaxed. At a 1 mm radius, suppose the blood travels 1 mm/sec. By the formula $F \propto r^4$, consider how the velocity would change as radius changed:

$r = 1 \text{ mm}$	$r^4 = 1^4 = 1$	$F = 1 \text{ mm/sec}$ (given)
$r = 2 \text{ mm}$	$r^4 = 2^4 = 16$	$F = 16 \text{ mm/sec}$
$r = 3 \text{ mm}$	$r^4 = 3^4 = 81$	$F = 81 \text{ mm/sec}$

These actual numbers do not matter; what matters is that a mere 3-fold increase in radius has produced an 81-fold increase in velocity—a demonstration that vessel

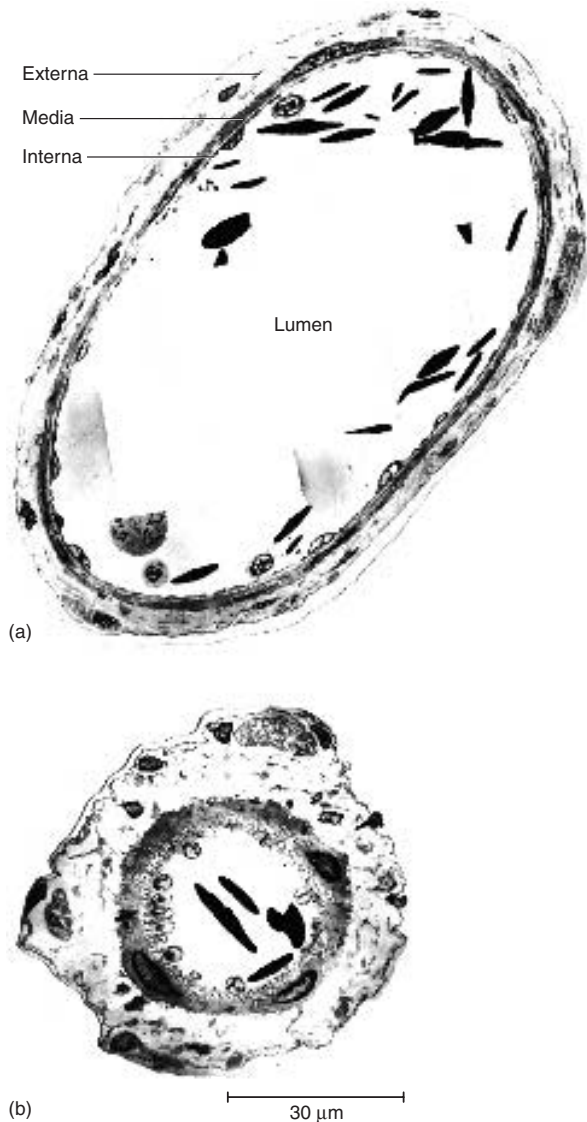


Figure 20.10 The Capacity for Vasoconstriction in an Arteriole. (a) A dilated arteriole. (b) The same arteriole, at a point just 1 mm from the area photographed in a, stimulated by a single drop of epinephrine. The diameter of the dilated region is about three times that of the constricted region.

radius exerts a very powerful influence over flow; moreover, it is the most adjustable of all variables that govern peripheral resistance.

Think About It

Suppose a vessel with a radius of 1 mm had a flow of 5 mm/sec, and then the vessel dilated to a radius of 5 mm. What would be the new flow rate?

⁶lamina = layer

Table 20.2 Blood Velocity in the Systemic Circuit

Vessel	Typical Lumen Diameter	Velocity*
Aorta	2.5 cm	1,200 mm/sec
Arterioles	20–50 μm	15 mm/sec
Capillaries	5–9 μm	0.4 mm/sec
Venules	20 μm	5 mm/sec
Inferior vena cava	3 cm	80 mm/sec

*Peak systolic velocity in the aorta; mean or steady velocity in other vessels

To integrate this information, consider how the velocity of blood flow differs from one part of the systemic circuit to another (table 20.2). Flow is fastest in the aorta because it is a large vessel close to the pressure source, the left ventricle. From aorta to capillaries, velocity diminishes for three reasons: (1) The blood has traveled a greater distance, so friction has slowed it down. (2) The arterioles and capillaries have smaller radii and therefore put up more resistance. (3) Even though the radii of individual vessels become smaller as we progress farther from the heart, the number of vessels and their *total* cross-sectional area becomes greater and greater. The aorta has a cross-sectional area of 3 to 5 cm^2 , while the total cross-sectional area of all the capillaries is about 4,500 to 6,000 cm^2 . Thus, a given volume of aortic blood is distributed over a greater total area in the capillaries, which *collectively* form a wider path in the bloodstream. Just as water slows down when a narrow mountain stream flows into a lake, blood slows down as it enters pathways with a greater total width.

From capillaries to vena cava, velocity rises again. One reason for this is that the veins have larger diameters than the capillaries, so they create less resistance. Furthermore, since many capillaries converge on one venule, and many venules on a larger vein, a large amount of blood is being forced into a progressively smaller channel—like water flowing from a lake into an outlet stream and thus flowing faster again. Note, however, that blood in the veins never regains the velocity it had in the large arteries. This is because the veins are farther from the heart and the pressure is much lower.

Regulation of Blood Pressure and Flow

Blood pressure is subject to local, neural, and hormonal controls over vasomotion. We now consider each of these three influences in turn.

Local Control

Autoregulation is the ability of tissues to regulate their own blood supply. According to the *metabolic theory of autoregulation*, if a tissue is inadequately perfused, it becomes hypoxic and its metabolites (waste products) accumulate— CO_2 , H^+ , K^+ , lactic acid, and adenosine, for example. These factors stimulate vasodilation, which increases perfusion. As the bloodstream delivers oxygen and carries away the metabolites, the vessels constrict. Thus, a homeostatic dynamic equilibrium is established that adjusts perfusion to the tissue's metabolic needs.

Blood platelets, endothelial cells, and the perivascular tissues secrete a variety of **vasoactive chemicals**—substances that stimulate vasomotion. Histamine, bradykinin, and prostaglandins stimulate vasodilation under such conditions as trauma, inflammation, and exercise. Endothelial cells secrete prostacyclin and nitric oxide, which are vasodilators, and polypeptides called *endothelins*, which are vasoconstrictors.

If a tissue's blood supply is cut off for a time and then restored, it often exhibits **reactive hyperemia**—an increase above the normal level of flow. This may be due to the accumulation of metabolites during the period of ischemia. Reactive hyperemia can be seen when the skin flushes after a person comes in from the cold. It also occurs in the forearm if a blood pressure cuff is inflated for too long and then loosened.

In the long run, a hypoxic tissue can increase its own perfusion by **angiogenesis**⁷—the growth of new blood vessels. (This term also refers to embryonic development of blood vessels.) Three situations in which this is important are the regrowth of the uterine lining after each menstrual period, the development of a higher density of blood capillaries in the muscles of well-conditioned athletes, and the growth of arterial bypasses around obstructions in the coronary circulation. Several growth factors and inhibitors control angiogenesis, but physiologists are not yet sure how it is regulated. Malignant tumors secrete growth factors that stimulate a dense network of blood vessels to grow into them and provide nourishment to the cancer cells. Oncologists are interested in finding a way to block tumor angiogenesis, which would choke off a tumor's blood supply and perhaps shrink or kill it.

Neural Control

In addition to local control, the blood vessels are under remote control by hormones and the autonomic nervous system. The **vasomotor center** of the medulla oblongata exerts sympathetic control over blood vessels throughout the body. (Precapillary sphincters have no innervation, however, and respond only to local and hormonal stimuli.)

⁷ *angio* = vessels + *genesis* = production of

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Sympathetic nerve fibers stimulate most blood vessels to constrict, but they dilate the vessels of skeletal and cardiac muscle in order to meet the metabolic demands of exercise. The role of sympathetic tone and vasomotor tone in controlling vessel diameter is explained in chapter 15.

The vasomotor center is an integrating center for three autonomic reflexes—*baroreflexes*, *chemoreflexes*, and the *medullary ischemic reflex*. A **baroreflex** is an autonomic, negative feedback response to changes in blood pressure. The changes are detected by stretch receptors called **baroreceptors**.⁸ These occur in all of the large arteries above the heart but are especially concentrated in the aortic arch, the *aortic sinuses* behind the aortic valve cusps, and the *carotid sinus* at the base of each internal carotid artery near the angle of the mandible (fig. 20.11). They are branched, knobby nerve fibers somewhat resembling Golgi tendon

⁸ *baro* = pressure

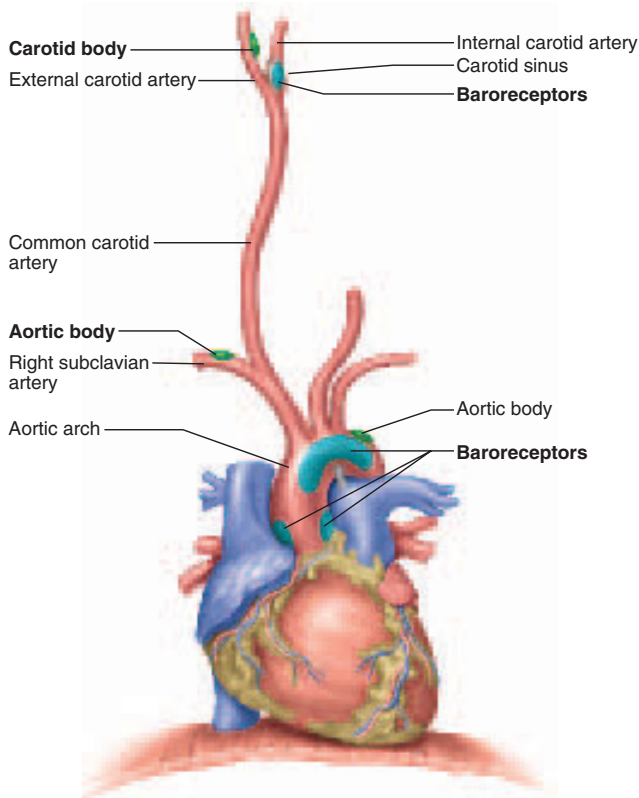


Figure 20.11 Locations of Arterial Baroreceptors and Chemoreceptors. Chemoreceptors are located in the carotid bodies and aortic bodies. Baroreceptors are located in the ascending aorta, aortic arch, and carotid sinus. The structures shown here in the *right* carotid arteries are repeated in the *left* carotids.

organs (see p. 508). The baroreceptors transmit signals continually to the brainstem. When the blood pressure rises, their signaling rate rises. This input inhibits the sympathetic cardiac and vasomotor neurons and reduces sympathetic tone, and it *excites* the vagal fibers to the heart. Thus, it reduces the heart rate and cardiac output, dilates the arteries and veins, and reduces the blood pressure (fig. 20.12). When BP drops below normal, on the other hand, the opposite reactions occur and BP rises back to normal.

Baroreflexes are important chiefly in short-term regulation of BP, for example in adapting to changes in posture. Perhaps you have jumped quickly out of bed and felt a little dizzy for a moment. This occurs because gravity draws the blood into the large veins of the abdomen and lower limbs when you stand, which reduces venous return to the heart and cardiac output to the brain. Normally, the baroreceptors respond quickly to this drop in pressure and restore cerebral perfusion. Baroreflexes are not effective in correcting chronic hypertension, however. Apparently they adjust their set point to the higher BP and maintain dynamic equilibrium at this new level.

A **chemoreflex** is an autonomic response to changes in blood chemistry, especially its pH and concentrations of O_2 and CO_2 . It is initiated by chemoreceptors within small organs called **aortic bodies** and **carotid bodies**, located in the aortic arch, subclavian arteries, and external carotid arteries. The primary role of chemoreflexes is to adjust respiration to changes in blood chemistry, but they have a secondary role in stimulating vasomotion. Hypoxemia (O_2 deficiency), hypercapnia (CO_2 excess), and aci-

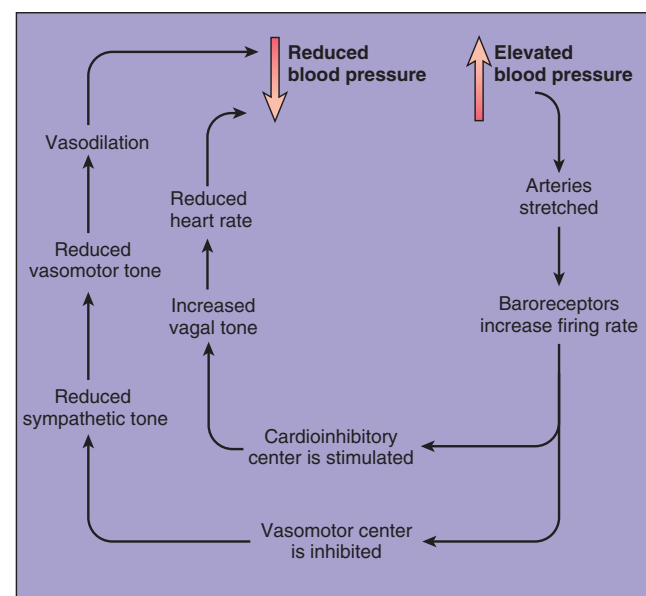


Figure 20.12 Negative Feedback Control of Blood Pressure. The reactions here occur in response to a rise in blood pressure.

dosis (low blood pH) stimulate the chemoreceptors and act through the vasomotor center to cause widespread vasoconstriction. This increases overall BP, thus increasing perfusion of the lungs and the rate of gas exchange. Chemoreceptors also stimulate one's breathing, so increased ventilation of the lungs matches their increased perfusion. Increasing one without the other would be of little use.

The **medullary ischemic (iss-KEE-mic) reflex** is an autonomic response to a drop in perfusion of the brain. Within seconds, the cardiac and vasomotor centers of the medulla oblongata send sympathetic signals to the heart and blood vessels that induce (1) an increase in heart rate and contraction force and (2) widespread vasoconstriction. These actions raise the blood pressure and, ideally, restore normal perfusion of the brain. The cardiac and vasomotor centers also receive input from other brain centers. Thus stress, anger, and arousal can also raise the blood pressure. The hypothalamus acts through the vasomotor center to redirect blood flow in response to exercise or changes in body temperature.

Hormonal Control

All of the following hormones influence blood pressure:

- **Angiotensin II.** This is a potent vasoconstrictor that raises the blood pressure. Its synthesis and action are detailed in chapter 23 (see fig. 23.13). One of the enzymes required for its synthesis is *angiotensin-converting enzyme (ACE)*. Hypertension is often

treated with drugs called *ACE inhibitors*, which block the action of this enzyme, thus lowering angiotensin II levels and blood pressure.

- **Aldosterone.** This “salt-retaining hormone” primarily promotes Na^+ retention by the kidneys. Since water follows sodium osmotically, Na^+ retention promotes water retention, thus promoting a higher blood volume and pressure.
- **Atrial natriuretic peptide.** ANP, secreted by the heart, antagonizes aldosterone. It increases Na^+ excretion by the kidneys, thus reducing blood volume and pressure. It also has a generalized vasodilator effect that contributes to lowering the blood pressure.
- **Antidiuretic hormone.** ADH primarily promotes water retention, but at pathologically high concentrations it is also a vasoconstrictor—hence its alternate name, *vasopressin*. Both of these effects raise blood pressure.
- **Epinephrine and norepinephrine.** These adrenal and sympathetic catecholamines bind to α -adrenergic receptors on the smooth muscle of most blood vessels. This stimulates the muscle to contract, thus producing vasoconstriction and raising the blood pressure. In the coronary blood vessels and blood vessels of the skeletal muscles, however, these chemicals bind to β -adrenergic receptors and cause vasodilation, thus increasing blood flow to the myocardium and muscular system during exercise.

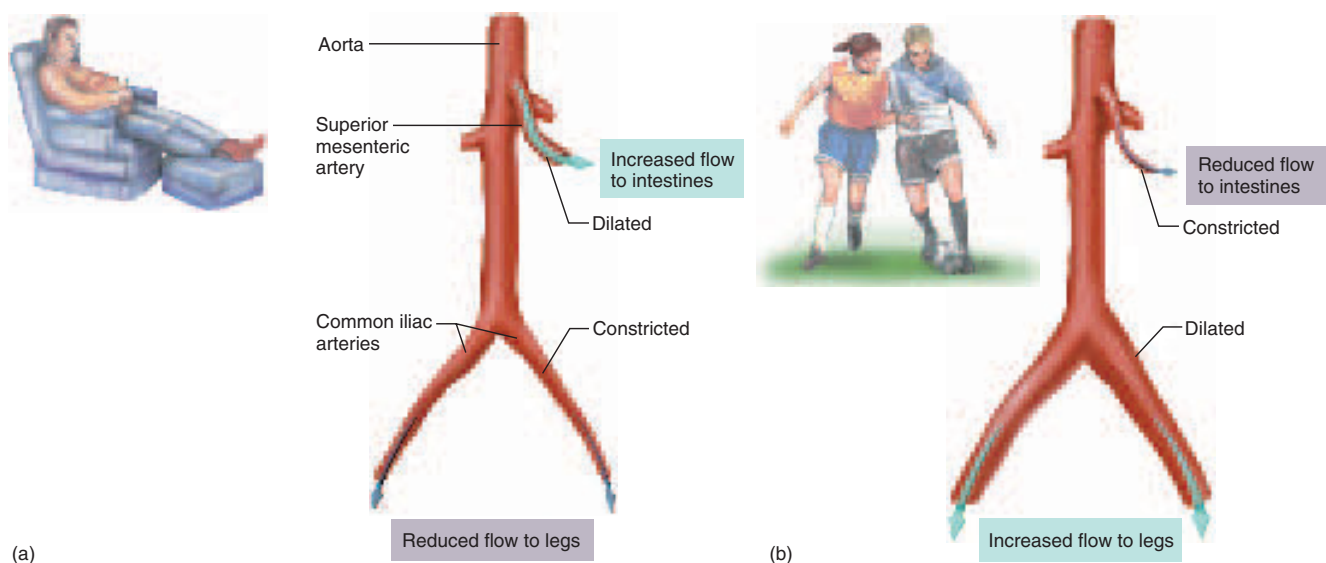


Figure 20.13 Redirection of Blood Flow in Response to Changing Metabolic Needs. (a) After a meal, the intestines receive priority and the skeletal muscles receive relatively little flow. (b) During exercise, the muscles receive higher priority. Although vasoconstriction and vasodilation are shown here in major arteries for illustration purposes, most control occurs at a microscopic level in the arterioles.

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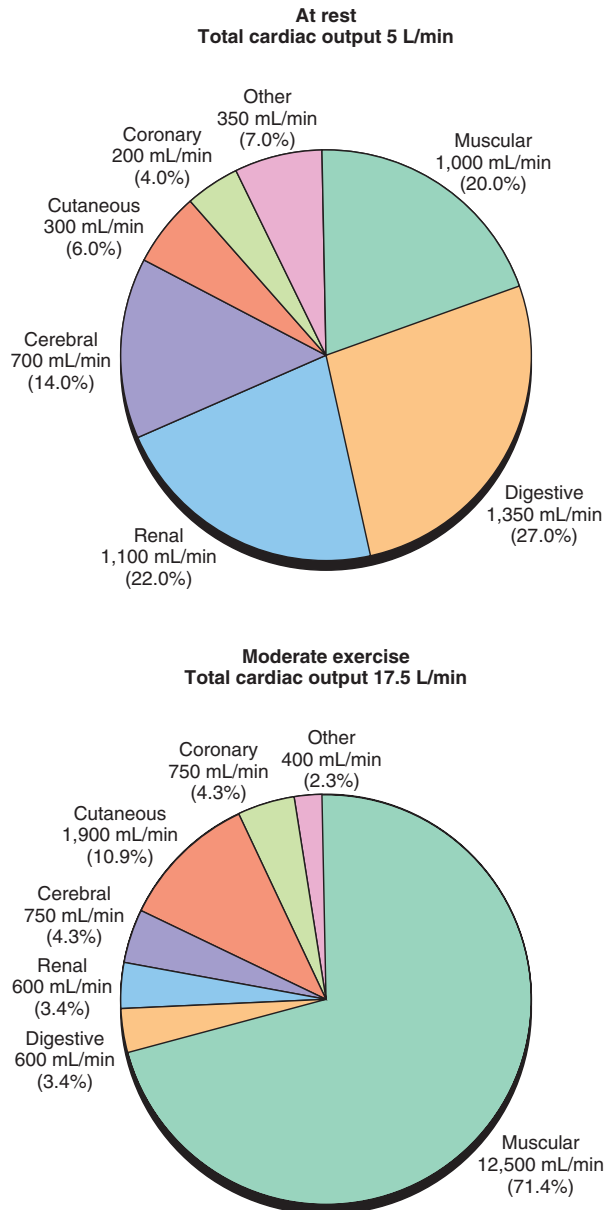


Figure 20.14 Changes in Systemic Blood Flow During Rest and Exercise.

Vasomotion and Routing of Blood Flow

If a chemical such as epinephrine causes widespread vasoconstriction, or if it causes vasoconstriction in a large system such as the integumentary or digestive system, it can produce an overall rise in blood pressure. Localized vasoconstriction, however, has a very different effect. If a par-

ticular artery constricts, pressure downstream from the constriction drops and pressure upstream from it rises. If blood can travel by either of two routes and one route puts up more resistance than the other, most blood follows the path of least resistance. This mechanism enables the body to redirect blood from one organ to another.

For example, if you are dozing in an armchair after a big meal, vasoconstriction shuts down blood flow to 90% or more of the capillaries in the muscles of your lower limbs. This raises the BP above the limbs, where the aorta gives off a branch, the superior mesenteric artery, supplying the small intestine. High resistance in the circulation of the legs and low resistance in the superior mesenteric artery routes blood to the small intestine, where it is needed to absorb digested nutrients (fig. 20.13a).

On the other hand, during vigorous exercise, the arteries in your muscles dilate. To make blood available to the muscles, flow must be reduced elsewhere—notably in the skin, kidneys, and digestive tract (fig. 20.13b). Thus, changes in peripheral resistance can shift blood flow from one organ system to another to meet the changing metabolic priorities of the body. Physical exertion increases perfusion of the lungs, myocardium, and skeletal muscles while reducing perfusion of the kidneys and digestive tract (fig. 20.14).

The arterioles are the most significant point of control over peripheral resistance and blood flow because (1) they are on the proximal sides of the capillary beds, so they are best positioned to regulate flow into the capillaries; (2) they greatly outnumber any other class of arteries and thus provide the most numerous control points; and (3) they are more muscular in proportion to their diameters than any other class of blood vessels and are highly capable of vasomotion. Arterioles alone account for about half of the total peripheral resistance of the circulatory system. However, larger arteries and veins are also capable of considerable vasomotion and control of peripheral resistance.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

- For a healthy 15-year-old girl at rest, what would be typical readings for systolic pressure, diastolic pressure, pulse pressure, and mean arterial pressure?
- Explain why arterial blood flow is pulsatile and venous flow is not.
- What three variables affect peripheral resistance to blood flow? Which of these is most able to change from one minute to the next?
- What are the three primary mechanisms for controlling vessel radius? Briefly explain each.
- Explain how the baroreflex serves as an example of homeostasis and negative feedback.
- Explain how the body can shift the flow of blood from one organ system to another.

Capillary Exchange

Objectives

When you have completed this section, you should be able to

- describe how materials get from the blood to the surrounding tissues;
- describe and calculate the forces that enable capillaries to give off and reabsorb fluid; and
- describe the causes and effects of edema.

Only 250 to 300 mL of blood is in the capillaries at any given time. This is the most important blood in the body, however, for it is mainly across capillary walls that exchanges occur between the blood and surrounding tissues. **Capillary exchange** refers to this two-way movement of fluid.

Substances pass between the blood and tissue fluid by three routes: (1) through the intercellular clefts between endothelial cells, (2) through the fenestrations (pores) of fenestrated capillaries, and (3) through the endothelial cell cytoplasm (fig. 20.15). The mechanisms involved are *diffusion*, *transcytosis*, *filtration*, and *reabsorption*, which we examine in that order.

Diffusion

The most important mechanism of exchange is diffusion. Glucose and oxygen, being more concentrated in the systemic blood than in the tissue fluid, diffuse out of the blood. Carbon dioxide and other wastes, being more concentrated in the tissue fluid, diffuse into the blood. (Oxygen and carbon dioxide diffuse in the opposite directions in the pulmonary circuit.) Such diffusion is only possible if the solute can either permeate the plasma membranes of the endothelial cells or find passages large enough to pass through—namely, the fenestrations and intercellular clefts. Such lipid-soluble substances as steroid hormones, O_2 , and CO_2 diffuse easily through the plasma membranes. Substances insoluble in lipids, such as glucose and electrolytes, must pass through membrane channels,

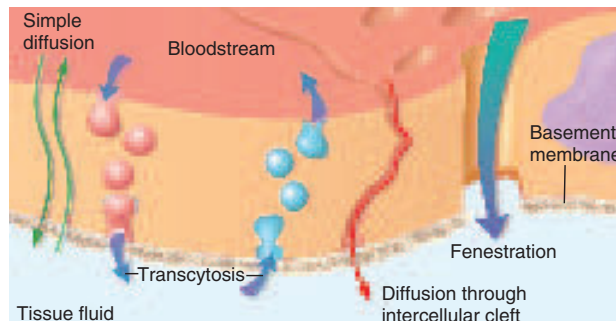


Figure 20.15 Pathways of Capillary Fluid Exchange.

fenestrations, or intercellular clefts. Large molecules such as proteins are usually held back by the small size of these passages.

Transcytosis

Transcytosis is a process in which endothelial cells pick up droplets of fluid on one side of the plasma membrane by pinocytosis, transport the vesicles across the cell, and discharge the fluid on the other side by exocytosis (see fig. 3.23, p. 114). This probably accounts for only a small fraction of solute exchange across the capillary wall, but fatty acids, albumin, and some hormones such as insulin move across the endothelium by this mechanism.

Filtration and Reabsorption

The equilibrium between filtration and osmosis discussed in chapter 3 becomes particularly relevant when we consider capillary fluid exchange. Typically, fluid filters out of the arterial end of a capillary and osmotically reenters it at the venous end (fig. 20.16). This fluid delivers materials to the cells and removes their metabolic wastes.

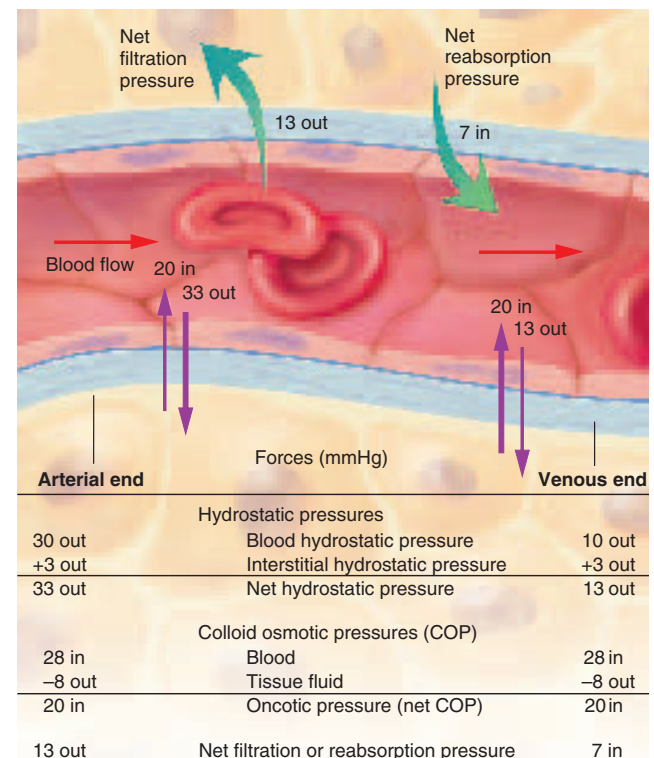


Figure 20.16 The Forces of Capillary Filtration and Reabsorption. Note the shift from net filtration at the arterial (left) end to net reabsorption at the venous (right) end.

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It may seem odd that a capillary could give off fluid at one point and reabsorb it at another. This comes about as the result of a shifting balance between hydrostatic and osmotic forces. A typical capillary has a blood (hydrostatic) pressure of about 30 mmHg at the arterial end. The hydrostatic pressure of the interstitial space has been difficult to measure and remains a point of controversy, but a typical value accepted by many authorities is -3 mmHg. The negative value indicates that this is a slight suction, which helps draw fluid out of the capillary. (This force will be represented hereafter as 3_{out} .) In this case, the positive hydrostatic pressure within the capillary and the negative interstitial pressure work in the same direction, creating a total outward force of about 33 mmHg.

These forces are opposed by **colloid osmotic pressure (COP)**, the portion of the blood's osmotic pressure due to its plasma proteins. The blood has a COP of about 28 mmHg, due mainly to albumin. Tissue fluid has less than one-third the protein concentration of blood plasma and has a COP of about 8 mmHg. The difference between the COP of blood and COP of tissue fluid is called **oncotic pressure**: $28_{\text{in}} - 8_{\text{out}} = 20_{\text{in}}$. Oncotic pressure tends to draw water into the capillary by osmosis, opposing hydrostatic pressure. These opposing forces produce a **net filtration pressure (NFP)** of 13 mmHg out, as follows:

Hydrostatic pressure

Blood pressure	30_{out}
Interstitial pressure	$+ 3_{\text{out}}$
Net hydrostatic pressure	33_{out}

Colloid osmotic pressure

Blood COP	28_{in}
Tissue fluid COP	$- 8_{\text{out}}$
Oncotic pressure	20_{in}

Net filtration pressure

Net hydrostatic pressure	33_{out}
Oncotic pressure	$- 20_{\text{in}}$
Net filtration pressure	13_{out}

The NFP of 13 mmHg causes about 0.5% of the blood plasma to leave the capillaries at the arterial end.

At the venous end, however, capillary blood pressure is lower—about 10 mmHg. All the other pressures are unchanged. Thus, we get:

Hydrostatic pressure

Blood pressure	10_{out}
Interstitial pressure	$+ 3_{\text{out}}$
Net hydrostatic pressure	13_{out}

Net reabsorption pressure

Oncotic pressure	20_{in}
Net hydrostatic pressure	$- 13_{\text{out}}$
Net reabsorption pressure	7_{in}

The prevailing force is inward at the venous end because osmotic pressure overrides filtration pressure. The **net reabsorption pressure** of 7 mmHg inward causes the capillary to reabsorb fluid at this end.

Now you can see why a capillary gives off fluid at one end and reabsorbs it at the other. The only pressure that changes from the arterial end to the venous end is the capillary blood pressure, and this change is responsible for the shift from filtration to reabsorption. With a reabsorption pressure of 7 mmHg and a net filtration pressure of 13 mmHg, it might appear that far more fluid would leave the capillaries than reenter them. However, since capillaries branch along their length, there are more of them at the venous end than at the arterial end, which partially compensates for the difference between filtration and reabsorption pressures. They also typically have nearly twice the diameter at the venous end that they have at the arterial end, so there is more capillary surface area available to reabsorb fluid than to give it off. Consequently, capillaries reabsorb about 85% of the fluid they filter. The other 15% is absorbed and returned to the blood by way of the lymphatic system, as described in chapter 21.

Of course, water is not the only substance that crosses the capillary wall by filtration and reabsorption. It carries along many of the solutes dissolved in it. This process is called **solvent drag**.

Variations in Capillary Filtration and Reabsorption

The figures used in the preceding discussion serve only as examples; circumstances differ from place to place in the body and from time to time in the same capillaries. Capillaries usually reabsorb most of the fluid they filter, but this is not always the case. The kidneys have capillary networks called *glomeruli* in which there is little or no reabsorption; they are entirely devoted to filtration. Alveolar capillaries of the lungs, by contrast, are almost entirely dedicated to absorption so that fluid does not fill the air spaces.

Capillary activity also varies from moment to moment. In a resting tissue, most precapillary sphincters are constricted and the capillaries are collapsed. Capillary BP is very low (if there is any flow at all), and reabsorption predominates. When a tissue becomes metabolically active, its capillary flow increases. In active muscles, capillary pressure rises to the point that it overrides reabsorption along the entire length of the capillary. Fluid accumulates in the muscle, and exercising muscles increase in size by as much as 25%. Capillary permeability is also subject to chemical influences. Traumatized tissue releases such chemicals as substance P, bradykinin, and histamine, which increase permeability and filtration.

Edema

Edema is the accumulation of excess fluid in a tissue. It often shows as swelling of the face, fingers, abdomen, or

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ankles but also affects internal organs, where its effects are hidden from view. Edema occurs when fluid filters into a tissue faster than it is reabsorbed. It has three fundamental causes:

1. **Increased capillary filtration.** This results from increases in capillary BP or permeability. Poor venous return, for example, causes pressure to back up into the capillaries. Congestive heart failure and incompetent heart valves can impede venous return from the lungs and cause pulmonary edema. Systemic edema is a common problem when a person is confined to a bed or wheelchair, with insufficient muscular activity to promote venous return. Kidney failure leads to edema by causing water retention and hypertension. Histamine causes edema by dilating the arterioles and making the capillaries more permeable. Capillary permeability also increases with age, which puts older people at risk of edema.
2. **Reduced capillary reabsorption.** Capillary reabsorption depends on oncotic pressure, which is proportional to the concentration of blood albumin. A deficiency of blood albumin (hypoproteinemia) produces edema because the capillaries osmotically reabsorb even less of the fluid that they give off. Since blood albumin is produced by the liver, liver diseases such as cirrhosis tend to lead to hypoproteinemia and edema. Edema is commonly seen in regions of famine due to dietary protein deficiency. Hypoproteinemia also commonly results from severe burns, radiation sickness, and kidney diseases that allow protein to escape in the urine.
3. **Obstructed lymphatic drainage.** The lymphatic system, described in detail in chapter 21, is a system of one-way vessels that collect fluid from the tissues and return it to the bloodstream. Obstruction of these vessels or the surgical removal of lymph nodes can interfere with fluid drainage and lead to the accumulation of tissue fluid distal to the obstruction.

In severe edema, so much fluid may transfer from the blood vessels to the tissue spaces that blood volume and pressure drop so low as to cause circulatory shock (described later in this chapter). Furthermore, as the tissues become swollen with fluid, oxygen delivery and waste removal are impaired and tissue necrosis may occur. Pulmonary edema presents a threat of suffocation, and cerebral edema can produce headaches, nausea, and sometimes seizures and coma.

13. What forces favor capillary filtration? What forces favor reabsorption?
14. How can a capillary shift from a predominantly filtering role at one time to a predominantly reabsorbing role at another?
15. State the three fundamental causes of edema and explain why edema can be dangerous.

Venous Return and Circulatory Shock

Objectives

When you have completed this section, you should be able to

- explain how blood in the veins is returned to the heart;
- discuss the importance of physical activity in venous return;
- discuss several causes of circulatory shock; and
- name and describe the stages of shock.

Hieronymus Fabricius (1537–1619) discovered the valves of the veins and argued that they would allow blood to flow in only one direction, not back and forth as Galen had thought. One of his medical students was William Harvey, who performed simple experiments on the valves that you can easily reproduce. In figure 20.17, from Harvey's book, the experimenter has pressed on a vein at point *H* to block flow from the wrist toward the elbow. With another finger, he has milked the blood out of it up to point *O*, the first valve proximal to *H*. When he tries to force blood downward, it stops at that valve. It can go no farther, and it causes the vein to swell at that point. Blood can flow from right to left through that valve but not from left to right.

You can easily demonstrate the action of these valves in your own hand. Hold your hand still, below waist level, until veins stand up on the back of it. (Do not apply a tourniquet!) Press on a vein close to your knuckles, and while holding it down, use another finger to milk that vein toward the wrist. It collapses as you force the blood out of it, and if you remove the second finger, it will not refill.



Figure 20.17 An Illustration from William Harvey's *De Motu Cordis* (1628). These experiments demonstrate the existence of one-way valves in veins of the arms. See text for explanation. **In the space between *O* and *H*, what (if anything) would happen if the experimenter lifted his finger from point *O*? What if he lifted his finger from point *H*? Why?**

Before You Go On

Answer the following questions to test your understanding of the preceding section:

12. List the three mechanisms of capillary exchange and relate each one to the structure of capillary walls.

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The valves prevent blood from flowing back into it from above. When you remove the first finger, however, the vein fills from below.

Mechanisms of Venous Return

The flow of blood back to the heart, called **venous return**, is achieved by five mechanisms:

1. **The pressure gradient.** Pressure generated by the heart is the most important force in venous flow, even though it is substantially weaker in the veins than in the arteries. Pressure in the venules ranges from 12 to 18 mmHg, and pressure at the point where the venae cavae enter the heart, called **central venous pressure**, averages 4.6 mmHg. Thus, there is a venous pressure gradient (ΔP) of about 7 to 13 mmHg favoring the flow of blood toward the heart. The pressure gradient and venous return increase when blood volume increases. Venous return decreases when the veins constrict (*venoconstriction*) and oppose flow, and it increases when they dilate and offer less resistance. However, it increases if *all* the body's blood vessels constrict, because this reduces the "storage capacity" of the circulatory system and raises blood pressure and flow.
2. **Gravity.** When you are sitting or standing, blood from your head and neck returns to the heart simply by "flowing downhill" by way of the large veins above the heart. Thus the large veins of the neck are normally collapsed or nearly so, and their venous pressure is close to zero. The dural sinuses, however, have more rigid walls and cannot collapse. Their pressure is as low as -10 mmHg, creating a risk of *air embolism* if they are punctured (see insight 20.3).
3. **The skeletal muscle pump.** In the limbs, the veins are surrounded and massaged by the muscles. They squeeze the blood out of the compressed part of a vein, and the valves ensure that this blood can go in only one direction—toward the heart (fig. 20.18).
4. **The thoracic (respiratory) pump.** This mechanism aids the flow of venous blood from the abdominal to the thoracic cavity. When you inhale, your thoracic cavity expands and its internal pressure drops, while downward movement of the diaphragm raises the pressure in your abdominal cavity. The *inferior vena cava (IVC)*, your largest vein, is a flexible tube passing through both of these cavities. If abdominal pressure on the IVC rises while thoracic pressure on it drops, then blood is squeezed upward toward the heart. It is not forced back into the lower limbs because the venous valves there prevent this. Because of the thoracic pump, central venous pressure fluctuates from 2 mmHg when you inhale to 6 mmHg when you exhale, and blood flows faster when you inhale.

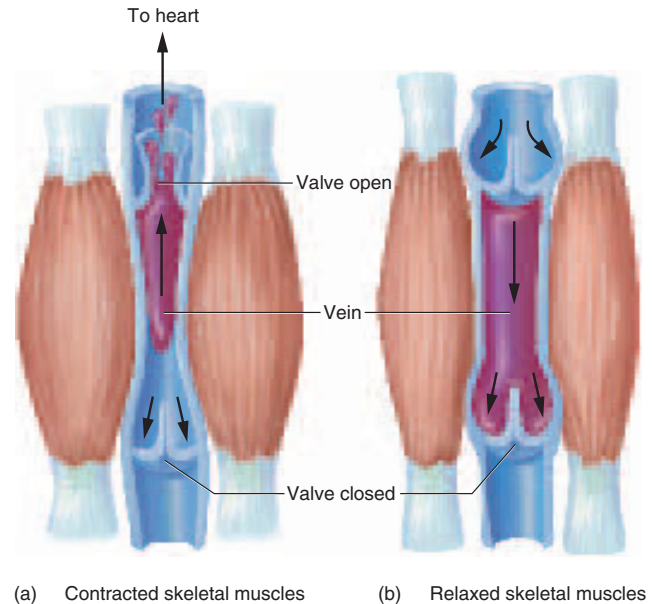


Figure 20.18 The Skeletal Muscle Pump. (a) When the muscles contract and compress a vein, blood is squeezed out of it and flows upward toward the heart; valves below the point of compression prevent backflow of the blood. (b) When the muscles relax, blood flows back downward under the pull of gravity but can only flow as far as the nearest valve.

5. **Cardiac suction.** During ventricular systole, the chordae tendineae pull the AV valve cusps downward, slightly expanding the atrial space. This creates a slight suction that draws blood into the atria from the venae cavae and pulmonary veins.

Insight 20.3 Clinical Application

Air Embolism

Injury to the dural sinuses or jugular veins presents less danger from loss of blood than from air sucked into the circulatory system. The presence of air in the bloodstream is called *air embolism*. This is an important concern to neurosurgeons, who sometimes operate with the patient in a sitting position. If a dural sinus is punctured, air can be sucked into the sinus and accumulate in the heart chambers, which blocks cardiac output and causes sudden death. Smaller air bubbles in the systemic circulation can cut off blood flow to the brain, lungs, myocardium, and other vital tissues.

Venous Return and Physical Activity

Exercise increases venous return for many reasons. The heart beats faster and harder, increasing cardiac output and

blood pressure. Blood vessels of the skeletal muscles, lungs, and heart dilate, increasing flow. The increase in respiratory rate and depth enhances the action of the thoracic pump. Muscle contractions increase venous return by the skeletal muscle pump mechanism. Increased venous return increases cardiac output, which is important in perfusion of the muscles just when they need it most.

Conversely, when a person is still, blood accumulates in the limbs because venous pressure is not high enough to override the weight of the blood and drive it upward. Such accumulation of blood is called **venous pooling**. To demonstrate this effect, hold one hand above your head and the other below your waist for about a minute. Then, quickly bring your two hands together and compare the palms. The hand held above your head usually appears pale because its blood has drained out of it; the hand held below the waist appears redder than normal because of venous pooling in its veins and capillaries. Venous pooling is troublesome to people who must stand for prolonged periods. If enough blood accumulates in the limbs, cardiac output may become so low that the brain is inadequately perfused and a person may experience dizziness or **syncope** (SIN-co-pee) (fainting). This can usually be prevented by periodically tensing the calf and other muscles to keep the skeletal muscle pump active. Military jet pilots often perform maneuvers that could cause the blood to pool in the abdomen and lower limbs, causing partial loss of vision or loss of consciousness. To prevent this, they wear pressure suits that inflate and tighten on the lower limbs during these maneuvers; in addition, they sometimes must tense their abdominal muscles to prevent venous pooling and blackout.

Think About It

Why is venous pooling not a problem when you are sleeping and the skeletal muscle pump is inactive?

Circulatory Shock

Circulatory shock (not to be confused with electrical or spinal shock) is any state in which cardiac output is insufficient to meet the body's metabolic needs. All forms of circulatory shock fall into two categories: (1) **cardiogenic shock**, caused by inadequate pumping by the heart usually as a result of myocardial infarction, and (2) **low venous return (LVR) shock**, in which cardiac output is low because too little blood is returning to the heart.

There are three principal forms of LVR shock:

1. **Hypovolemic shock**, the most common form, is produced by a loss of blood volume as a result of hemorrhage, trauma, bleeding ulcers, burns, or dehydration. Dehydration is a major cause of death

from heat exposure. In hot weather, the body produces as much as 1.5 L of sweat per hour. Water transfers from the bloodstream to replace lost tissue fluid, and blood volume may drop too low to maintain adequate circulation.

2. **Obstructed venous return shock** occurs when a growing tumor or aneurysm, for example, compresses a nearby vein and impedes its blood flow.
3. **Venous pooling (vascular) shock** occurs when the body has a normal total blood volume, but too much of it accumulates in the limbs. This can result from long periods of standing or sitting or from widespread vasodilation. **Neurogenic shock** is a form of venous pooling shock that occurs when there is a sudden loss of vasomotor tone, allowing the vessels to dilate. This can result from causes as severe as brainstem trauma or as slight as an emotional shock.

Elements of both venous pooling and hypovolemic shock are present in certain cases, such as septic shock and anaphylactic shock, which involve both vasodilation and a loss of fluid through abnormally permeable capillaries. **Septic shock** occurs when bacterial toxins trigger vasodilation and increased capillary permeability. **Anaphylactic shock**, discussed more fully in chapter 21, results from exposure to an antigen to which a person is allergic, such as bee venom. Antigen-antibody complexes trigger the release of histamine, which causes generalized vasodilation and increased capillary permeability.

Responses to Circulatory Shock

In **compensated shock**, several homeostatic mechanisms act to bring about spontaneous recovery. The hypotension resulting from low cardiac output triggers the baroreflex and the production of angiotensin II, both of which counteract shock by stimulating vasoconstriction. Furthermore, if a person faints and falls to a horizontal position, gravity restores blood flow to the brain. Even quicker recovery is achieved if the person's feet are elevated to promote drainage of blood from the legs.

If these mechanisms prove inadequate, **decompensated shock** ensues and several life-threatening positive feedback loops occur. Poor cardiac output results in myocardial ischemia and infarction, which further weakens the heart and reduces output. Slow circulation of the blood can lead to disseminated intravascular coagulation (DIC) (see chapter 18). As the vessels become congested with clotted blood, venous return grows even worse. Ischemia and acidosis of the brainstem depress the vasomotor and cardiac centers, causing loss of vasomotor tone, further vasodilation, and further drop in BP and cardiac output. Before long, damage to the cardiac and brain tissues may be too great to be undone.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

16. Explain how respiration aids venous return.
17. Explain how muscular activity and venous valves aid venous return.
18. Define *circulatory shock*. What are some of the causes of low venous return shock?

Special Circulatory Routes

Objectives

When you have completed this section, you should be able to

- explain how the brain maintains stable perfusion;
- discuss the causes and effects of strokes and transient ischemic attacks;
- explain the mechanisms that increase muscular perfusion during exercise; and
- contrast the blood pressure of the pulmonary circuit with that of the systemic circuit, and explain why the difference is important in pulmonary function.

Certain circulatory pathways have special physiological properties adapted to the functions of their organs. Two of these are described in other chapters: the coronary circulation in chapter 19 and fetal and placental circulation in chapter 29. Here we take a closer look at the circulation to the brain, skeletal muscles, and lungs.

Brain

Total blood flow to the brain fluctuates less than that of any other organ (about 700 mL/min at rest). Such constancy is important because even a few seconds of oxygen deprivation causes loss of consciousness, and 4 or 5 minutes of anoxia is time enough to cause irreversible brain damage. While total cerebral perfusion is fairly stable, blood flow can be shifted from one part of the brain to another in a matter of seconds as different parts engage in motor, sensory, or cognitive functions.

The brain regulates its own blood flow in response to changes in BP and chemistry. The cerebral arteries dilate when the systemic BP drops and constrict when BP rises, thus minimizing fluctuations in cerebral BP. Cerebral blood flow thus remains quite stable even when mean arterial pressure (MAP) fluctuates from 60 to 140 mmHg. A MAP below 60 mmHg produces syncope and a MAP above 160 mmHg causes cerebral edema.

The main chemical stimulus for cerebral autoregulation is pH. Poor cerebral perfusion allows CO₂ to accumulate in the brain tissue. This lowers the pH of the tissue fluid and triggers local vasodilation, which improves perfusion. Extreme hypercapnia, however, depresses neural

activity. The opposite condition, hypocapnia, raises the pH and stimulates vasoconstriction, thus reducing perfusion and giving CO₂ a chance to rise to a normal level. Hyperventilation (exhaling CO₂ faster than the body produces it) induces hypocapnia, which leads to cerebral vasoconstriction, ischemia, dizziness, and sometimes syncope.

Brief episodes of cerebral ischemia produce **transient ischemic attacks (TIAs)**, characterized by temporary dizziness, light-headedness, loss of vision or other senses, weakness, paralysis, headache, or aphasia. A TIA may result from spasms of diseased cerebral arteries. It lasts from just a moment to a few hours and is often an early warning of an impending stroke.

A stroke, or **cerebrovascular accident (CVA)**, is the sudden death (infarction) of brain tissue caused by ischemia. Cerebral ischemia can be produced by atherosclerosis, thrombosis, or a ruptured aneurysm. The effects of a CVA range from unnoticeable to fatal, depending on the extent of tissue damage and the function of the affected tissue. Blindness, paralysis, loss of sensation, and loss of speech are common. Recovery depends on the ability of neighboring neurons to take over the lost functions and on the extent of collateral circulation to regions surrounding the cerebral infarction.

Skeletal Muscles

In contrast to the brain, the skeletal muscles receive a highly variable blood flow depending on their state of exertion. At rest, the arterioles are constricted, most of the capillary beds are shut down, and total flow through the muscular system is about 1 L/min. During exercise, the arterioles dilate in response to epinephrine and norepinephrine from the adrenal medulla and sympathetic nerves. Precapillary sphincters, which lack innervation, dilate in response to muscle metabolites such as lactic acid, CO₂, and adenosine. Blood flow can increase more than 20-fold during strenuous exercise, which requires that blood be diverted from other organs such as the digestive tract and kidneys to meet the needs of the working muscles.

Muscular contraction compresses the blood vessels and impedes flow. For this reason, isometric contraction causes fatigue more quickly than intermittent isotonic contraction. If you squeeze a rubber ball as hard as you can without relaxing your grip, you feel the muscles fatigue more quickly than if you intermittently squeeze and relax.

Lungs

After birth, the pulmonary circuit is the only route in which the arterial blood contains less oxygen than the venous blood. The pulmonary arteries have thin distensible walls with less elastic tissue than the systemic arteries. Thus, they have a BP of only 25/10. Capillary hydrostatic

pressure is about 10 mmHg in the pulmonary circuit as compared with an average of 17 mmHg in systemic capillaries. This lower pressure has two implications for pulmonary circulation: (1) blood flows more slowly through the pulmonary capillaries, and therefore it has more time for gas exchange; and (2) oncotic pressure overrides hydrostatic pressure, so these capillaries are engaged almost entirely in absorption. This prevents fluid accumulation in the alveolar walls and lumens, which would interfere with gas exchange. In a condition such as mitral valve stenosis, however, BP may back up into the pulmonary circuit, raising the capillary hydrostatic pressure and causing pulmonary edema, congestion, and hypoxemia.

Think About It

What abnormal skin coloration would result from pulmonary edema?

Another unique characteristic of the pulmonary arteries is their response to hypoxia. Systemic arteries dilate in response to local hypoxia and improve tissue perfusion. By contrast, pulmonary arteries constrict. Pulmonary hypoxia indicates that part of the lung is not being ventilated well, perhaps because of mucous congestion of the airway or a degenerative lung disease. Vasoconstriction in poorly ventilated regions of the lung redirects blood flow to better ventilated regions.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

19. In what conspicuous way does perfusion of the brain differ from perfusion of the skeletal muscles?
20. How does a stroke differ from a transient ischemic attack? Which of these bears closer resemblance to a myocardial infarction?
21. How does the low hydrostatic blood pressure in the pulmonary circuit affect the fluid dynamics of the capillaries there?
22. Contrast the vasomotor responses of the lungs versus skeletal muscles to hypoxia.

Anatomy of the Pulmonary Circuit

Objective

When you have completed this section, you should be able to

- trace the route of blood through the pulmonary circuit.

The remainder of this chapter centers on the names and pathways of the principal arteries and veins. The pul-

monary circuit is described here, and the systemic arteries and veins are described in the two sections that follow.

The pulmonary circuit (fig. 20.19) begins with the **pulmonary trunk**, a large vessel that ascends diagonally from the right ventricle and branches into the right and left **pulmonary arteries**. Each pulmonary artery enters a medial indentation of the lung called the *hilum* and branches into one **lobar artery** for each lobe of the lung: three on the right and two on the left. These arteries lead ultimately to small basketlike capillary beds that surround the pulmonary alveoli. This is where the blood unloads CO₂ and loads O₂. After leaving the alveolar capillaries, the pulmonary blood flows into venules and veins, ultimately leading to the **pulmonary veins**, which exit the lung at the hilum. The left atrium of the heart receives two pulmonary veins on each side.

The purpose of the pulmonary circuit is to exchange CO₂ for O₂. It does not serve the metabolic needs of the lung tissue itself; there is a separate systemic supply to the lungs for that purpose, the *bronchial arteries*, discussed later.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

23. Trace the flow of an RBC from right ventricle to left atrium and name the vessels along the way.
24. The lungs have two separate arterial supplies. Explain their functions.

Anatomy of the Systemic Arteries

Objectives

When you have completed this section, you should be able to

- identify the principal arteries of the systemic circuit; and
- trace the flow of blood from the heart to any major organ.

The systemic circuit supplies oxygen and nutrients to all the organs and removes their metabolic wastes. Part of it, the coronary circulation, was described in chapter 19. The other systemic arteries are described in tables 20.3 through 20.8 (figs. 20.20–20.30). The names of the blood vessels often describe their location by indicating the body region traversed (as in the *axillary* artery or *femoral* artery); an adjacent bone (as in *radial* artery or *temporal* artery); or the organ supplied or drained by the vessel (as in *hepatic* artery or *renal* vein). There is a great deal of anatomical variation in the circulatory system from one person to another. The remainder of this chapter describes the most common pathways.

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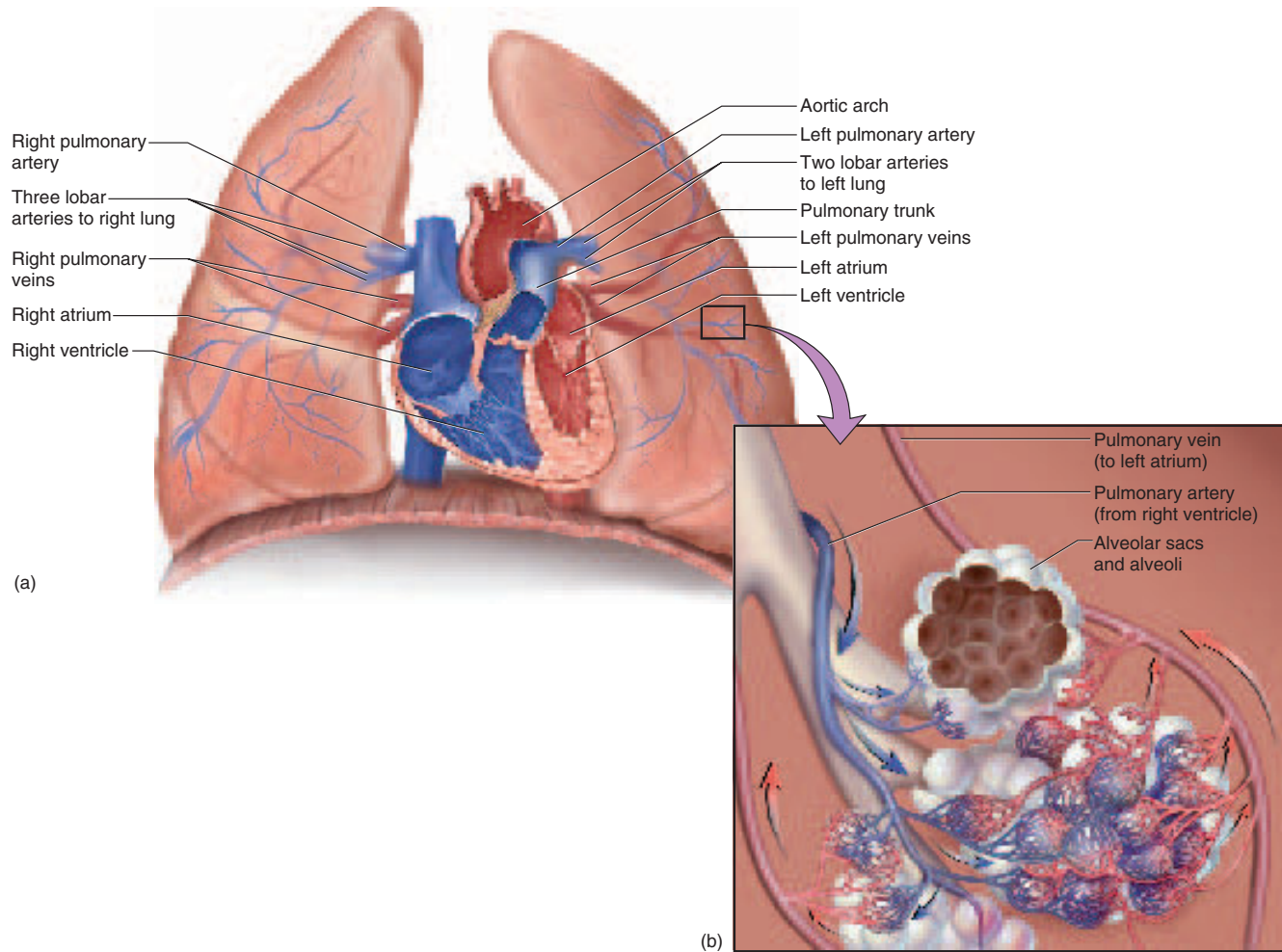


Figure 20.19 The Pulmonary Circulation. (a) Gross anatomy. (b) Microscopic anatomy of the blood vessels that supply the pulmonary alveoli.

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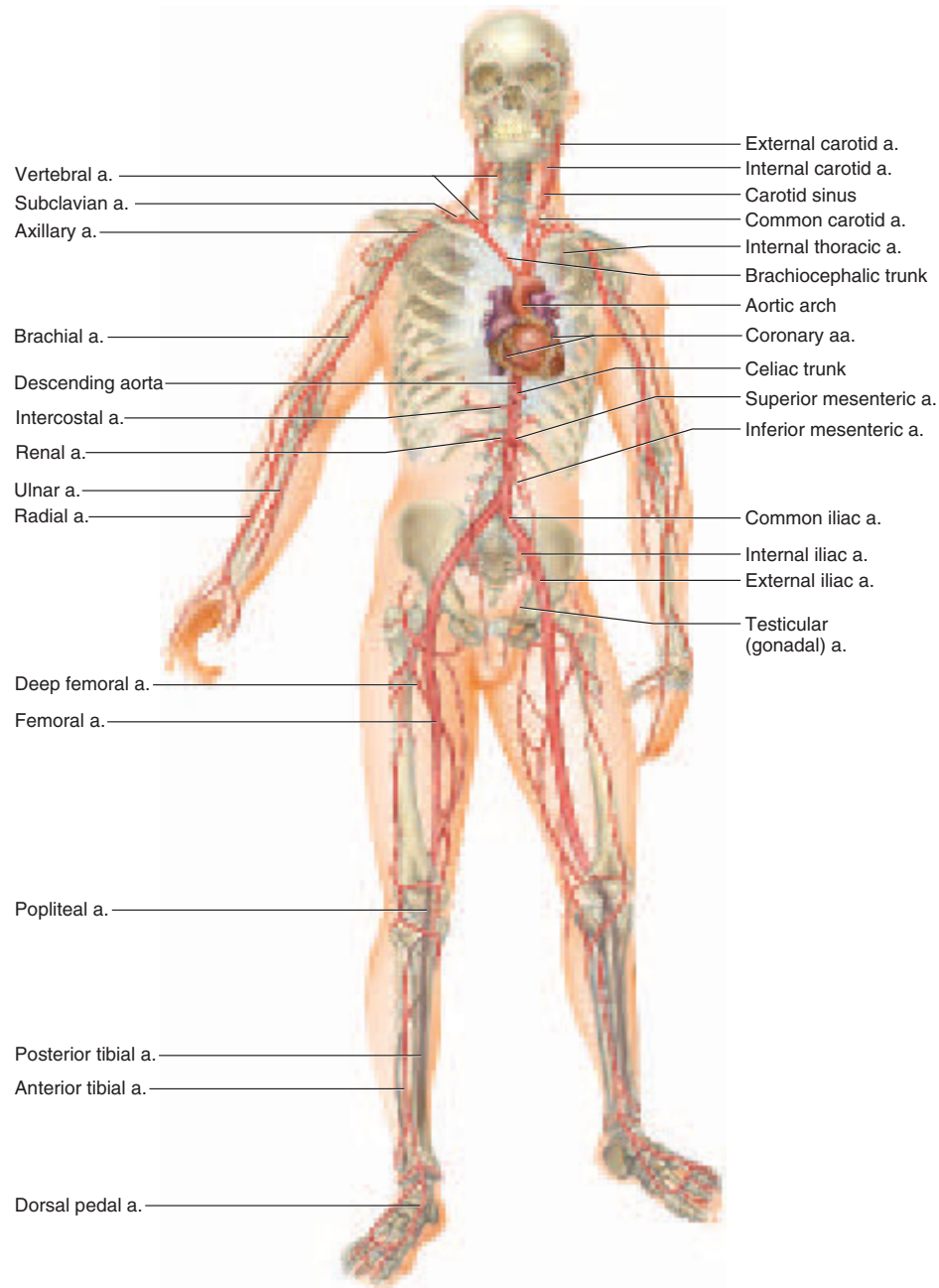


Figure 20.20 The Major Systemic Arteries. (*a.* = artery; *aa.* = arteries)

Table 20.3 The Aorta and Its Major Branches

All systemic arteries arise from the aorta, which has three principal regions (fig. 20.21):

1. The **ascending aorta** rises about 5 cm above the left ventricle. Its only branches are the coronary arteries, which arise behind two cusps of the aortic valve. Opposite each semilunar valve cusp is an **aortic sinus** containing baroreceptors.
2. The **aortic arch** curves to the left like an inverted U superior to the heart. It gives off three major arteries in this order: the **brachiocephalic**⁹ (BRAY-kee-oh-seh-FAL-ic) **trunk**, **left common carotid** (cah-ROT-id) **artery**, and **left subclavian**¹⁰ (sub-CLAY-vee-un) **artery**, which are further traced in tables 20.4 and 20.5.
3. The **descending aorta** passes downward dorsal to the heart, at first to the left of the vertebral column and then anterior to it, through the thoracic and abdominal cavities. It is called the **thoracic aorta** above the diaphragm and the **abdominal aorta** below. It ends in the lower abdominal cavity by forking into the **right and left common iliac arteries**, which are further traced in table 20.8.

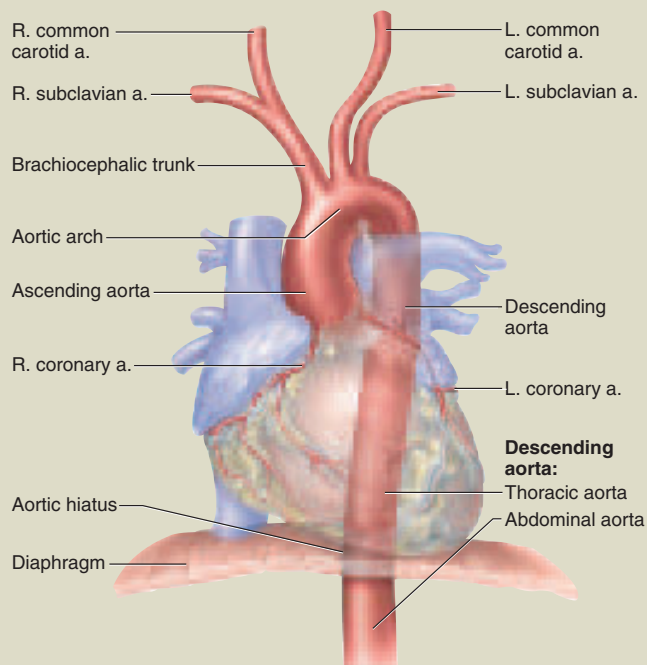


Figure 20.21 Beginning of the Aorta. (R. = right; L. = left; a. = artery)

⁹brachio = arm + cephal = head

¹⁰sub = below + clavi = clavicle, collarbone

Table 20.4 Arterial Supply to the Head and Neck

Origins of the Head-Neck Arteries

The head and neck receive blood from four pairs of arteries (fig. 20.22):

1. The **common carotid arteries**. The brachiocephalic trunk divides shortly after leaving the aortic arch and gives rise to the *right subclavian* and *right common carotid arteries*. The *left common carotid artery* arises directly from the aortic arch. The common carotids pass up the anterolateral aspect of the neck, alongside the trachea.
2. The **vertebral arteries** arise from the right and left subclavian arteries. Each travels up the neck through the transverse foramina of the cervical vertebrae and enters the cranial cavity through the foramen magnum.
3. The **thyrocervical¹¹ trunks** are tiny arteries that arise from the subclavian arteries lateral to the vertebral arteries; they supply the thyroid gland and some scapular muscles.
4. The **costocervical¹² trunks** (also illustrated in table 20.6) arise from the subclavian arteries a little farther laterally. They perfuse the deep neck muscles and some of the intercostal muscles of the superior rib cage.

Continuation of the Common Carotid Arteries

The common carotid arteries have the most extensive distribution of all the head-neck arteries. Near the laryngeal prominence (Adam's apple), each common carotid branches into an *external carotid artery* and an *internal carotid artery*:

1. The **external carotid artery** ascends the side of the head external to the cranium and supplies most external head structures except the orbits. The external carotid gives rise to the following arteries, in ascending order:
 - a. the **superior thyroid artery** to the thyroid gland and larynx,
 - b. the **lingual artery** to the tongue,

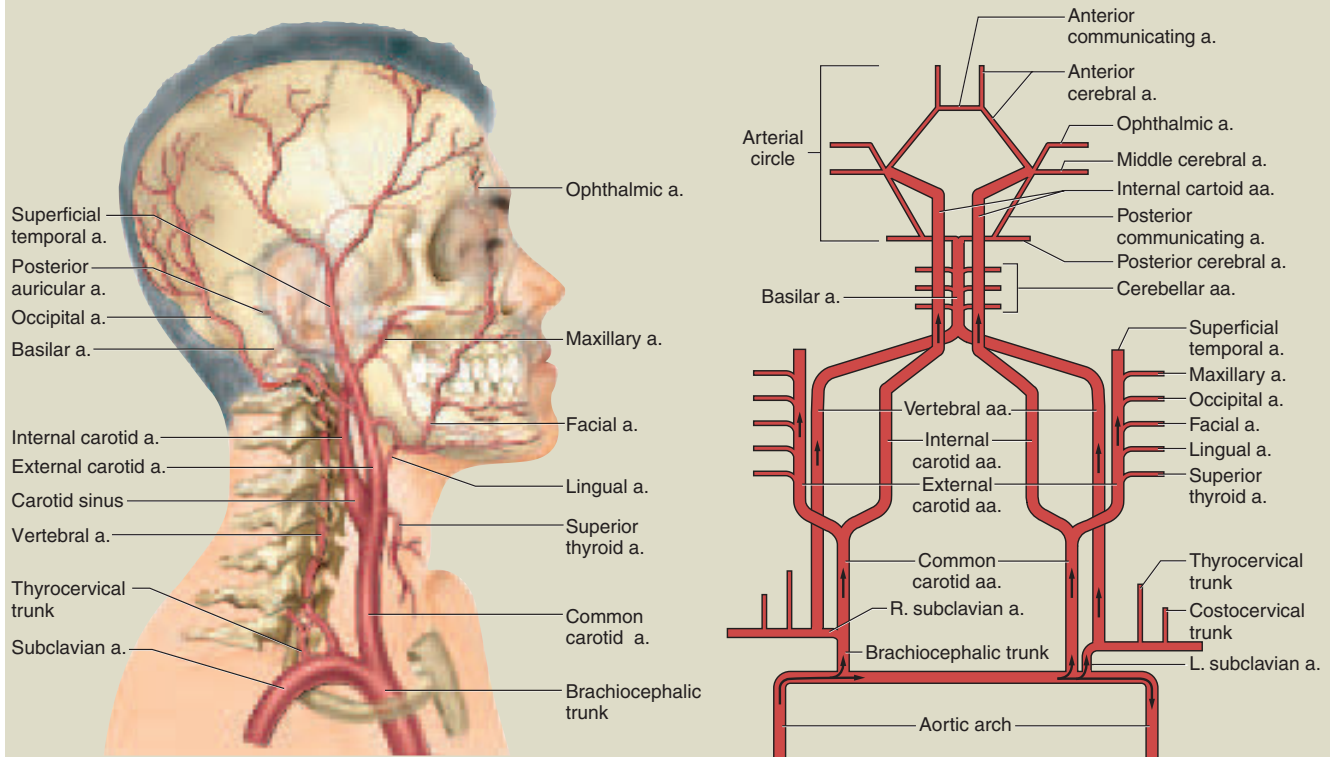


Figure 20.22 Arteries Supplying the Head and Neck.

List the arteries, in order, that an erythrocyte must travel to get from the left ventricle to the skin of the forehead.

¹¹thyro = thyroid gland + cerv = neck

¹²costo = rib

(continued)

Table 20.4 Arterial Supply to the Head and Neck (continued)

- c. the **facial artery** to the skin and muscles of the face,
 - d. the **occipital artery** to the posterior scalp,
 - e. the **maxillary artery** to the teeth, maxilla, buccal cavity, and external ear, and
 - f. the **superficial temporal artery** to the chewing muscles, nasal cavity, lateral aspect of the face, most of the scalp, and the dura mater surrounding the brain.
2. The **internal carotid artery** passes medial to the angle of the mandible and enters the cranial cavity through the carotid canal of the temporal bone. It supplies the orbits and about 80% of the cerebrum. Compressing the internal carotids near the mandible can therefore cause loss of consciousness.¹³ The carotid sinus is located in the internal carotid just above the branch point; the carotid body is nearby. After entering the cranial cavity, each internal carotid artery gives rise to the following branches:
- a. the **ophthalmic artery** to the orbits, nose, and forehead;
 - b. the **anterior cerebral artery** to the medial aspect of the cerebral hemisphere (see *arterial circle*); and
 - c. the **middle cerebral artery**, which travels in the lateral sulcus of the cerebrum and supplies the lateral aspect of the temporal and parietal lobes.

Continuation of the Vertebral Arteries

The vertebral arteries give rise to small branches in the neck that supply the spinal cord and other neck structures, then enter the foramen magnum and merge to form a single **basilar artery** along the anterior aspect of the brainstem. Branches of the basilar artery supply the cerebellum, pons, and inner ear. At the pons-midbrain junction, the basilar artery divides and gives rise to the *arterial circle*.

The Arterial Circle

Blood supply to the brain is so critical that it is furnished by several arterial anastomoses, especially an array of arteries called the **arterial circle (circle of Willis¹⁴)**, which surrounds the pituitary gland and optic chiasm. The arterial circle receives blood from the internal carotid and basilar arteries (fig. 20.23). Only 20% of people have a complete arterial circle. It consists of

1. two **posterior cerebral arteries**,
2. two **posterior communicating arteries**,
3. two **anterior cerebral arteries**, and
4. a single **anterior communicating artery**.

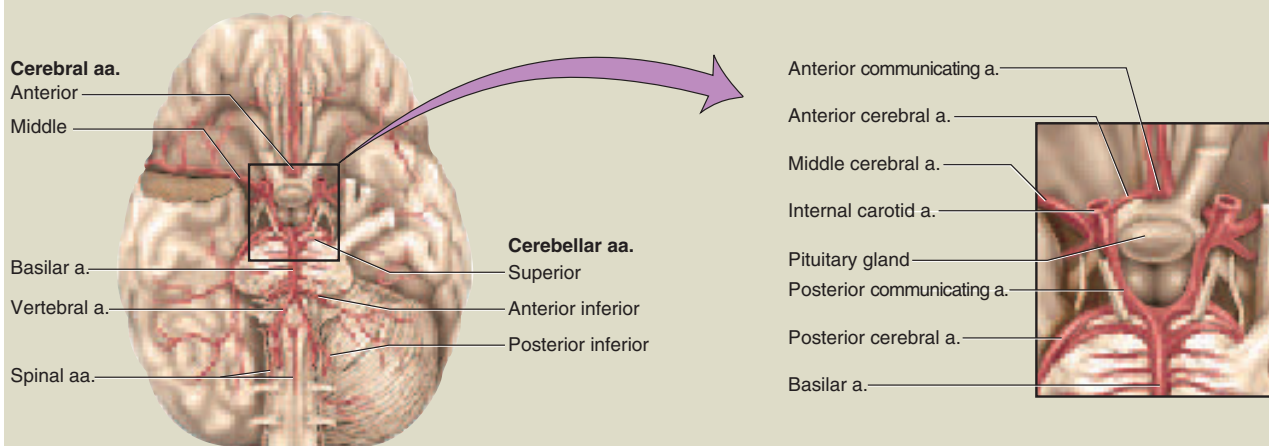


Figure 20.23 The Arterial Circle that Supplies the Brain.

¹³carot = stupor

¹⁴Thomas Willis (1621–75), English anatomist

Table 20.5 Arterial Supply to the Upper Limb

The Shoulder and Arm (brachium)

The origins of the subclavian arteries were described and illustrated in table 20.3. We now trace these further to examine the blood supply to the upper limb (fig. 20.24). This begins with a large artery that changes name from *subclavian* to *axillary* to *brachial* along its course:

1. The **subclavian¹⁵ artery** travels between the clavicle and first rib. It gives off several small branches to the thoracic wall and viscera, considered later.
2. The **axillary artery** is the continuation of the subclavian artery through the axillary region. It also gives off small thoracic branches, discussed later, and then ends at the neck of the humerus. Here, it gives off the **circumflex humeral artery**, which encircles the humerus. This loop supplies blood to the shoulder joint and deltoid muscle.
3. The **brachial (BRAY-kee-ul) artery** is the continuation of the axillary artery beyond the circumflex. It travels down the medial side of the humerus and ends just distal to the elbow, supplying the anterior flexor muscles of the brachium along the way. It exhibits several anastomoses near the elbow, two of which are noted next. This is the most commonly used artery for routine BP measurements.
4. The **deep brachial artery** arises from the proximal end of the brachial artery and supplies the triceps brachii muscle.
5. The **ulnar recurrent artery** arises about midway along the brachial artery and anastomoses distally with the ulnar artery. It supplies the elbow joint and the triceps brachii.
6. The **radial recurrent artery** leads from the deep brachial artery to the radial artery and supplies the elbow joint and forearm muscles.

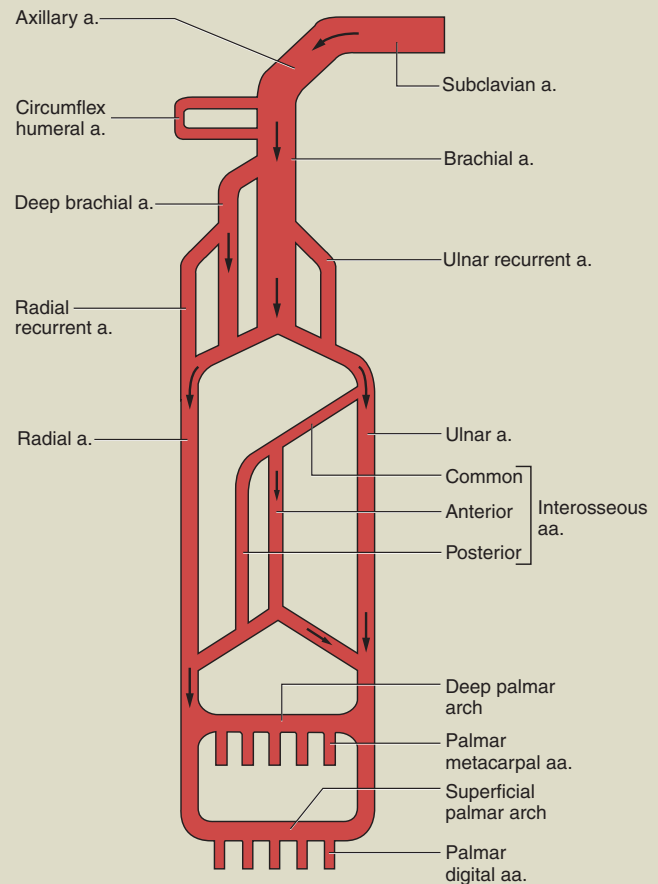
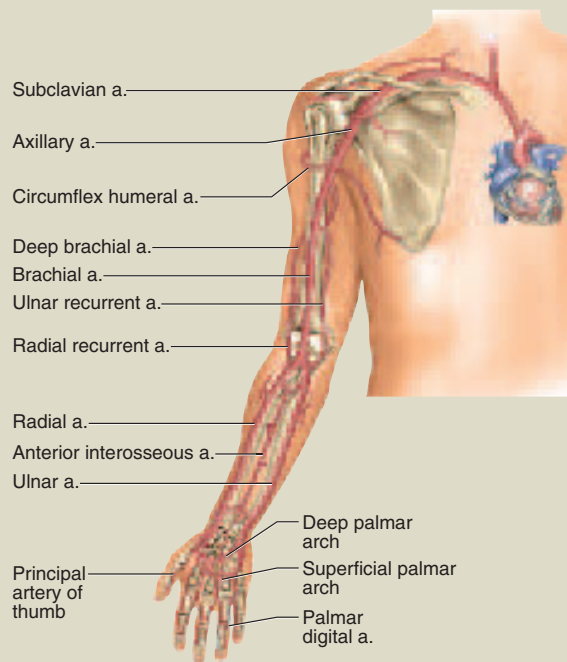


Figure 20.24 Arteries Supplying the Upper Limb.

¹⁵sub = below + clavi = clavicle

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Table 20.5 Arterial Supply to the Upper Limb (continued)**The Forearm (antebrachium)**

Just distal to the elbow, the brachial artery divides into the **radial artery** and **ulnar artery**, which travel alongside the radius and ulna, respectively. The most common place to take a pulse is at the radial artery, just proximal to the thumb. Near its origin, the radial artery receives the deep brachial artery. The ulnar artery gives rise, near its origin, to the **anterior and posterior interosseous¹⁶ arteries**, which travel between the radius and ulna. Structures supplied by these arteries are as follows:

1. Radial artery: lateral forearm muscles, wrist, thumb, and index finger.
2. Ulnar artery: medial forearm muscles, digits 3 to 5, and medial aspect of index finger.
3. Interosseous arteries: deep flexors and extensors.

The Hand

At the wrist, the radial and ulnar arteries anastomose to form two *palmar arches*:

1. The **deep palmar arch** gives rise to the **palmar metacarpal arteries** of the hand.
2. The **superficial palmar arch** gives rise to the **palmar digital arteries** of the fingers.

¹⁶*inter* = between + *osse* = bones

Table 20.6 Arterial Supply to the Thorax

The thoracic aorta begins distal to the aortic arch and ends at the **aortic hiatus** (hy-AY-tus), a passage through the diaphragm. Along the way, it sends off numerous small branches to viscera and structures of the body wall (fig. 20.25).

Visceral Branches

These supply the viscera of the thoracic cavity:

1. **Bronchial arteries.** Two of these on the left and one on the right supply the visceral pleura, esophagus, and bronchi of the lungs. They are the systemic blood supply to the lungs mentioned earlier.
2. **Esophageal arteries.** Four or five of these supply the esophagus.
3. **Mediastinal arteries.** Many small mediastinal arteries (not illustrated) supply structures of the posterior mediastinum.

Parietal Branches

The following branches supply chiefly the muscles, bones, and skin of the chest wall; only the first is illustrated:

1. **Posterior intercostal arteries.** Nine pairs of these course around the posterior aspect of the rib cage between the ribs and then anastomose with the anterior intercostal arteries (see following). They supply the skin and subcutaneous tissue, breasts, spinal cord and meninges, and the pectoralis, intercostal, and some abdominal muscles.
2. **Subcostal arteries.** A pair of these arise from the aorta, inferior to the twelfth rib, and supply the posterior intercostal tissues, vertebrae, spinal cord, and deep muscles of the back.
3. **Superior phrenic¹⁷ (FREN-ic) arteries.** These supply the posterior and superior aspects of the diaphragm.

¹⁷*phren* = diaphragm

(continued)

Table 20.6 Arterial Supply to the Thorax (continued)

The thoracic wall is also supplied by the following arteries. The first of these arises from the subclavian artery and the other three from the axillary artery:

1. The **internal thoracic (mammary) artery** supplies the breast and anterior thoracic wall and issues finer branches to the diaphragm and abdominal wall. Near its origin, it gives rise to the **pericardiophrenic artery**, which supplies the pericardium and diaphragm. As the internal thoracic artery descends alongside the sternum, it gives rise to **anterior intercostal arteries** that travel between the ribs and supply the ribs and intercostal muscles.
2. The **thoracoacromial**¹⁸ (THOR-uh-co-uh-CRO-me-ul) trunk supplies the superior shoulder and pectoral regions.
3. The **lateral thoracic artery** supplies the lateral thoracic wall.
4. The **subscapular artery** supplies the scapula, latissimus dorsi, and posterior wall of the thorax.

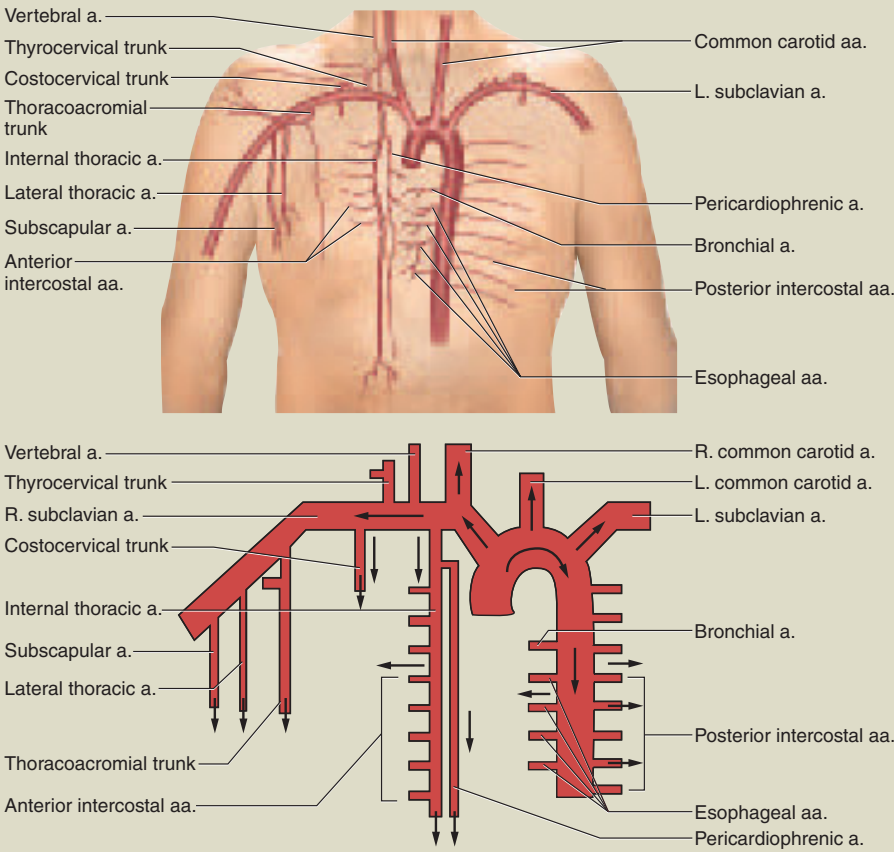


Figure 20.25 Arteries Supplying the Thorax.

¹⁸thoraco = chest + acr = tip + om = shoulder

Table 20.7 Arterial Supply to the Abdomen

Major Branches of Abdominal Aorta

After passing through the aortic hiatus, the aorta descends through the abdominal cavity. The abdominal aorta is retroperitoneal. It gives off arteries in the order listed here (fig. 20.26). Those indicated in the plural are paired right and left, and those indicated in the singular are single median arteries:

1. The **inferior phrenic arteries** supply the inferior surface of diaphragm and issue a small **superior suprarenal artery** to each adrenal (suprarenal) gland.
2. The **celiac**¹⁹ (SEE-lee-ac) **trunk** issues several branches to the upper abdominal viscera, further traced later in this table.
3. The **superior mesenteric artery** supplies the intestines (see mesenteric circulation later in this table).
4. The **middle suprarenal arteries** arise on either side of the superior mesenteric artery and supply the adrenal glands.
5. The **renal arteries** supply the kidneys and issue a small **inferior suprarenal artery** to each adrenal gland.
6. The **gonadal arteries** are long, narrow, winding arteries that descend from the midabdominal region to the female pelvic cavity or male scrotum. They are called the **ovarian arteries** in females and **testicular arteries** in males. The gonads begin their embryonic development near the kidneys. These arteries acquire their peculiar length and course by growing to follow the gonads as they descend to the pelvic cavity during fetal development.
7. The **inferior mesenteric artery** supplies the distal end of the large intestine (see mesenteric circulation).
8. The **lumbar arteries** arise from the lower aorta in four pairs and supply the posterior abdominal wall.
9. The **median sacral artery**, a tiny medial artery at the inferior end of the aorta, supplies the sacrum and coccyx.
10. The **common iliac arteries** arise as the aorta forks at its inferior end. They supply the lower abdominal wall, pelvic viscera (chiefly the urinary and reproductive organs), and lower limbs. They are further traced in table 20.8.

Branches of the Celiac Trunk

The celiac circulation to the upper abdominal viscera is perhaps the most complex route off the abdominal aorta. Because it has numerous anastomoses, the bloodstream does not follow a simple linear path but divides and rejoins itself at several points (fig. 20.27). As you study the following description,

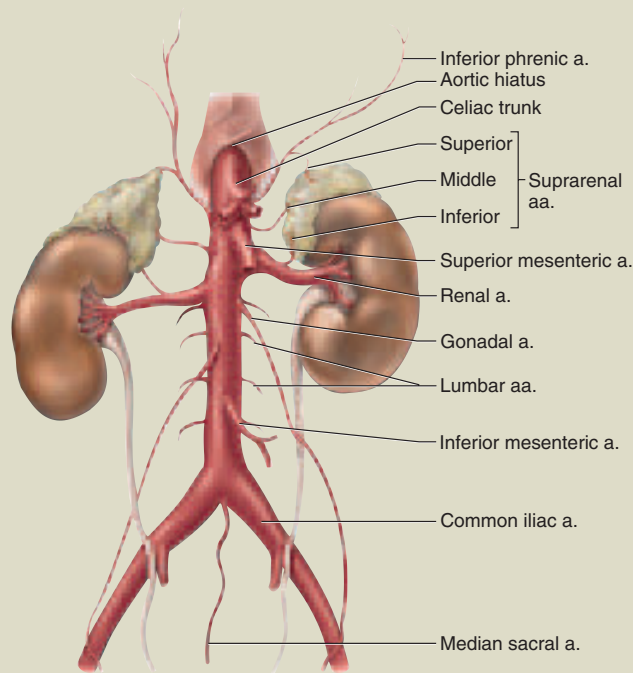


Figure 20.26 The Abdominal Aorta and Its Major Branches.

¹⁹*celi* = belly, abdomen

(continued)

Table 20.7 Arterial Supply to the Abdomen (continued)

locate these branches in the figure and identify the points of anastomosis. The short, stubby celiac trunk is a median branch of the aorta. It immediately gives rise to three principal subdivisions—the *common hepatic*, *left gastric*, and *splenic arteries*:

1. The **common hepatic artery** issues two main branches:

- the **gastroduodenal artery**, which supplies the stomach, anastomoses with the right gastroepiploic artery (see following), and then continues as the **inferior pancreaticoduodenal (PAN-cree-AT-ih-co-dew-ODD-eh-nul) artery**, which supplies the duodenum and pancreas before anastomosing with the superior mesenteric artery; and
- the **proper hepatic artery**, which is the continuation of the common hepatic artery after it gives off the gastroduodenal artery. It enters the inferior surface of the liver and supplies the liver and gallbladder.

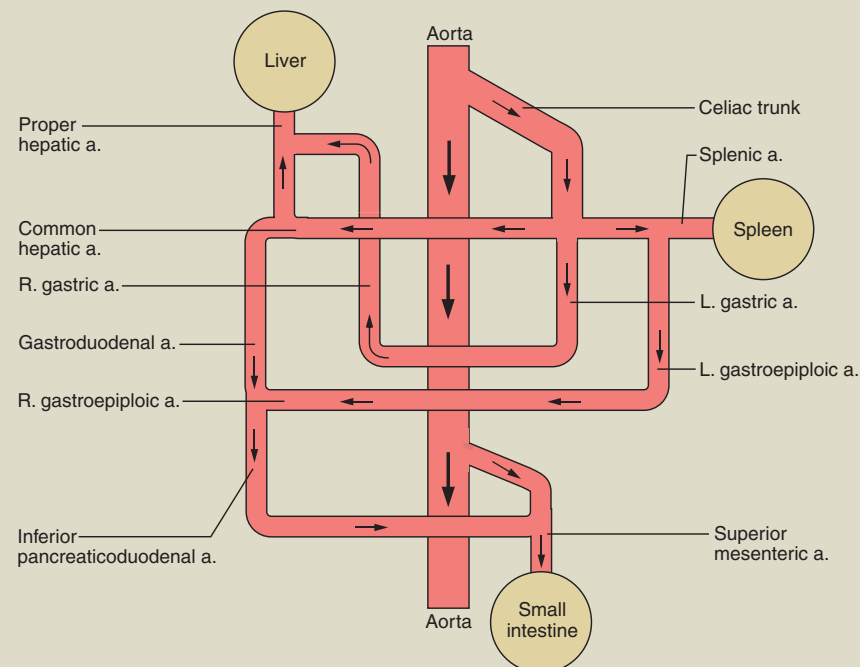
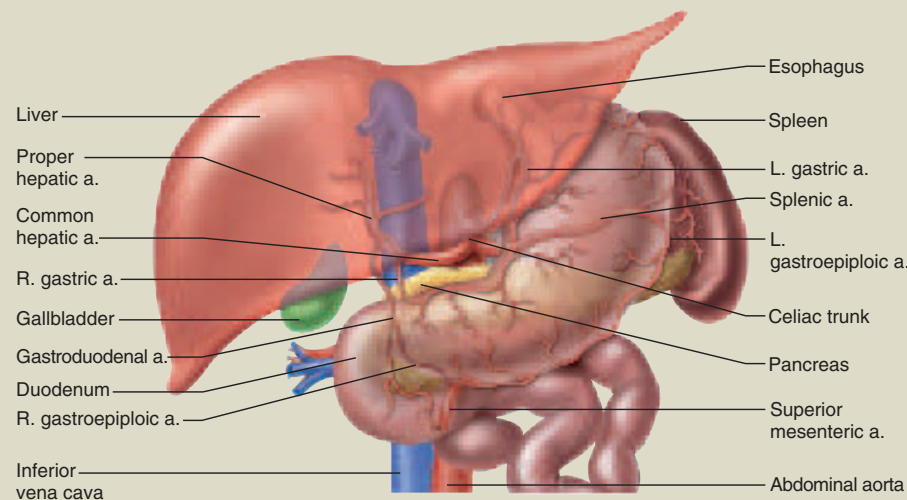


Figure 20.27 Branches of the Celiac Trunk.

Table 20.7 Arterial Supply to the Abdomen (continued)

2. The **left gastric artery** supplies the stomach and lower esophagus, arcs around the *lesser curvature* of the stomach, becomes the **right gastric artery** (which supplies the stomach and duodenum), and then anastomoses with the proper hepatic artery.
3. The **splenic artery** supplies blood to the spleen, but gives off the following branches on its way there:
 - a. the **pancreatic arteries** (not illustrated), which supply the pancreas; and
 - b. the **left gastroepiploic**²⁰ (GAS-tro-EP-ih-PLO-ic) **artery**, which arcs around the *greater curvature* of the stomach, becomes the **right gastroepiploic artery**, and then anastomoses with the gastroduodenal artery. Along the way, it supplies blood to the stomach and *greater omentum* (a fatty membrane suspended from the greater curvature).

Mesenteric Circulation

The mesentery (see atlas A, p. 38) contains numerous mesenteric arteries, veins, and lymphatic vessels that perfuse and drain the intestines. The arterial supply issues from the *superior* and *inferior mesenteric arteries* (fig. 20.28); numerous anastomoses between these ensure collateral circulation and adequate perfusion of the intestinal tract even if one route becomes obstructed. The following branches of the **superior mesenteric artery** serve the small intestine and most of the large intestine, among other organs:

1. The **inferior pancreaticoduodenal artery**, already mentioned, is an anastomosis from the gastroduodenal to the superior mesenteric artery; it supplies the pancreas and duodenum.
2. The **intestinal arteries** supply nearly all of the small intestine (jejunum and ileum).
3. The **ileocolic** (ILL-ee-oh-CO-lic) **artery** supplies the ileum of the small intestine and the appendix, cecum, and ascending colon.
4. The **right colic artery** supplies the ascending colon.
5. The **middle colic artery** supplies the transverse colon.

Branches of the *inferior mesenteric artery* serve the distal part of the large intestine:

1. The **left colic artery** supplies the transverse and descending colon.
2. The **sigmoid arteries** supply the descending and sigmoid colon.
3. The **superior rectal artery** supplies the rectum.

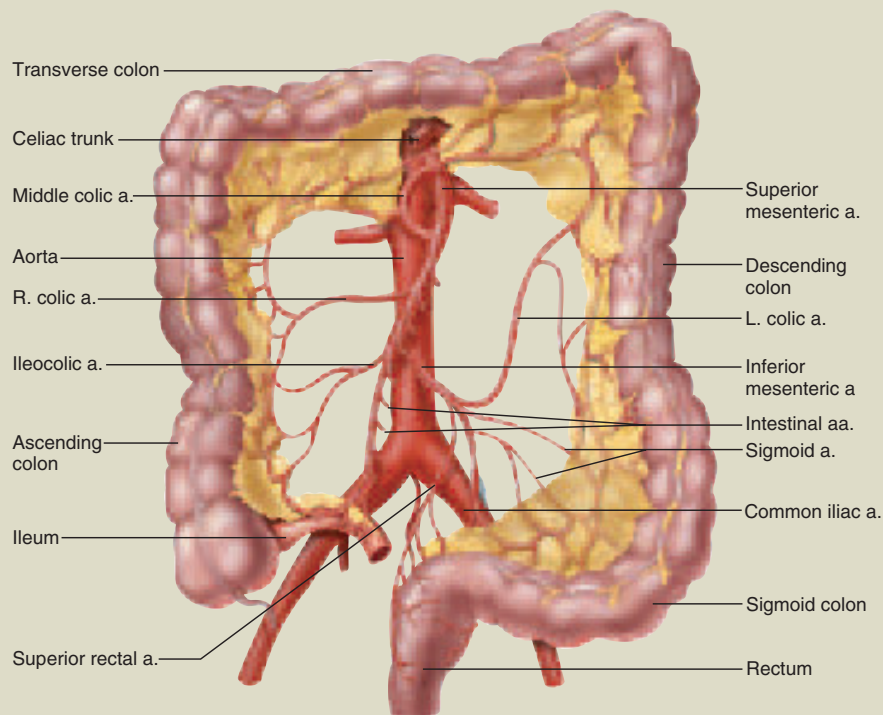


Figure 20.28 The Mesenteric Arteries.

²⁰gastro = stomach + *epi* = upon, above + *ploic* = pertaining to the greater omentum

Table 20.8 Arterial Supply to the Pelvic Region and Lower Limb

The common iliac arteries arise from the aorta at the level of vertebra L4 and continue for about 5 cm. At the level of the sacroiliac joint, each divides into an internal and external iliac artery. The **internal iliac artery** supplies mainly the pelvic wall and viscera, and the **external iliac artery** supplies mainly the lower limb (figs. 20.29 and 20.30).

Branches of the Internal Iliac Artery

1. The **iliolumbar** and **lateral sacral arteries** supply the wall of the pelvic region.
2. The **middle rectal artery** supplies the rectum.
3. The **superior and inferior vesical²¹ arteries** supply the urinary bladder.
4. The **uterine and vaginal arteries** supply the uterus and vagina.
5. The **superior and inferior gluteal arteries** supply the gluteal muscles.
6. The **obturator artery** supplies the adductor muscles of the medial thigh.
7. The **internal pudendal²² (pyu-DEN-dul) artery** serves the perineum and external genitals; it supplies the blood for vascular engorgement during sexual arousal.

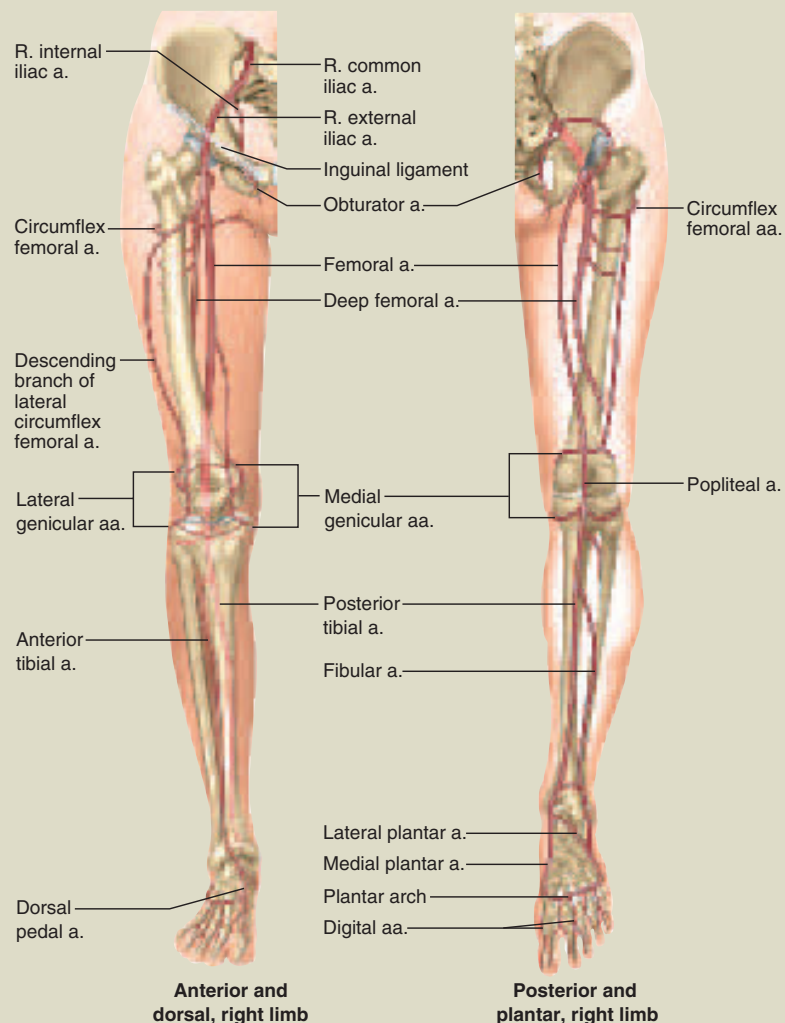


Figure 20.29 Arteries Supplying the Lower Limb.

²¹vesic = bladder

²²pudend = literally "shameful parts"; the external genitals

(continued)

Table 20.8 Arterial Supply to the Pelvic Region and Lower Limb (continued)

Branches of the External Iliac Artery

The external iliac artery sends branches to the skin and muscles of the abdominal wall and pelvic girdle. It then passes deep to the inguinal ligament and gives rise to branches that serve mainly the lower limbs:

1. The **femoral artery** passes through the femoral triangle of the upper medial thigh, where its pulse can be palpated. It gives off the following branches to supply the thigh region:
 - a. The **deep femoral artery**, which supplies the hamstring muscles; and
 - b. The **circumflex femoral arteries**, which encircle the neck of the femur and supply the femur and hamstring muscles.
2. The **popliteal artery** is a continuation of the femoral artery in the popliteal fossa at the rear of the knee. It produces anastomoses (**genicular arteries**) that supply the knee and then divides into the anterior and posterior tibial arteries.
3. The **anterior tibial artery** travels lateral to the tibia in the anterior compartment of the leg, where it supplies the extensor muscles. It gives rise to
 - a. the **dorsal pedal artery**, which traverses the ankle and dorsum of the foot; and
 - b. the **arcuate artery**, a continuation of the dorsal pedal artery that gives off the **metatarsal arteries** of the foot.
4. The **posterior tibial artery** travels through the posteromedial part of the leg and supplies the flexor muscles. It gives rise to
 - a. the **fibular (peroneal) artery**, which arises from the proximal end of the posterior tibial artery and supplies the lateral peroneal muscles;
 - b. the **lateral and medial plantar arteries**, which arise by bifurcation of the posterior tibial artery at the ankle and supply the plantar surface of the foot; and
 - c. the **plantar arch**, an anastomosis from the lateral plantar artery to the dorsal pedal artery that gives rise to the **digital arteries** of the toes.

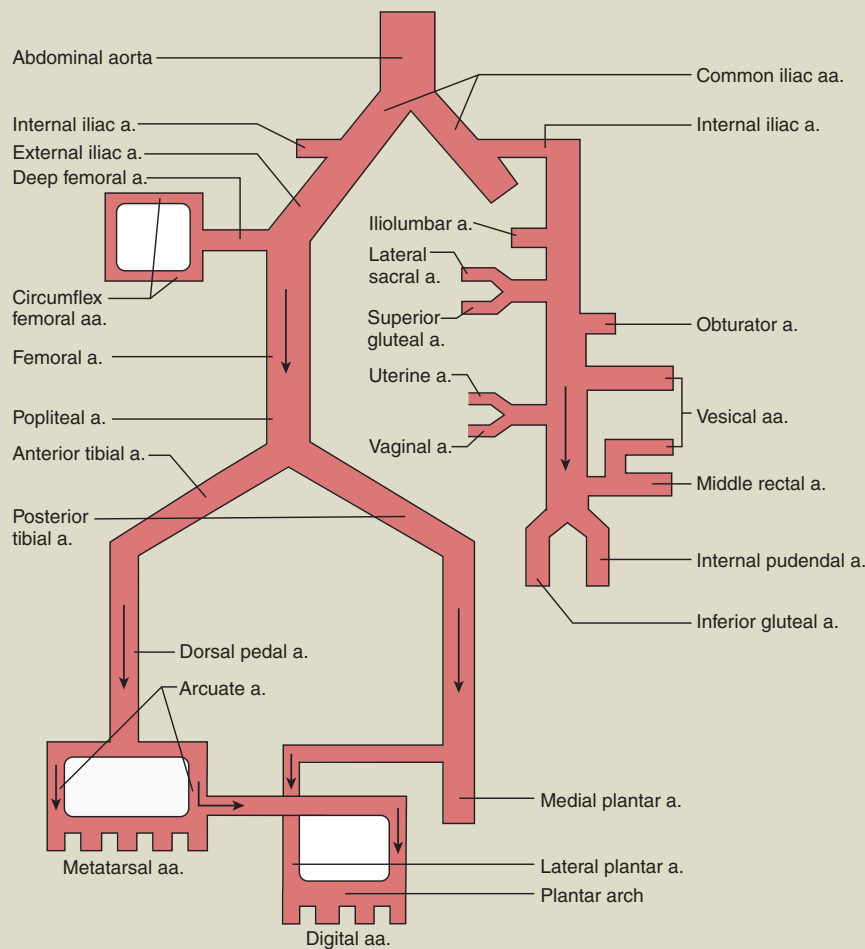


Figure 20.30 Arterial Flowchart of the Lower Limb.

What arteries of the wrist and hand are most comparable to the arcuate artery and plantar arch of the foot?

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In some places, major arteries come close enough to the body surface to be palpated. These places can be used to take a pulse, and they can serve as emergency **pressure points** where firm pressure can be applied to temporarily reduce arterial bleeding (fig. 20.31a). One of these points is the **femoral triangle** of the upper medial thigh (fig. 20.31b, c). This is an important landmark for arterial supply, venous drainage, and innervation of the lower limb. Its boundaries are the sartorius muscle laterally, the inguinal ligament superiorly, and the adductor longus muscle medially. The femoral artery, vein, and nerve run close to the surface at this point.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

25. Concisely contrast the destinations of the external and internal carotid arteries.

26. Briefly state the tissues that are supplied with blood by (a) the arterial circle, (b) the celiac trunk, (c) the superior mesenteric artery, and (d) the external iliac artery.
27. Trace the path of an RBC from the left ventricle to the metatarsal arteries. State two places along this path where you can palpate the arterial pulse.

Anatomy of the Systemic Veins

Objectives

When you have completed this section, you should be able to

- identify the principal veins of the systemic circuit; and
- trace the flow of blood from any major organ to the heart.

The principal veins of the systemic circuit (fig. 20.32) are detailed in tables 20.9 through 20.14. While arteries are usually deep and well protected, veins occur in both

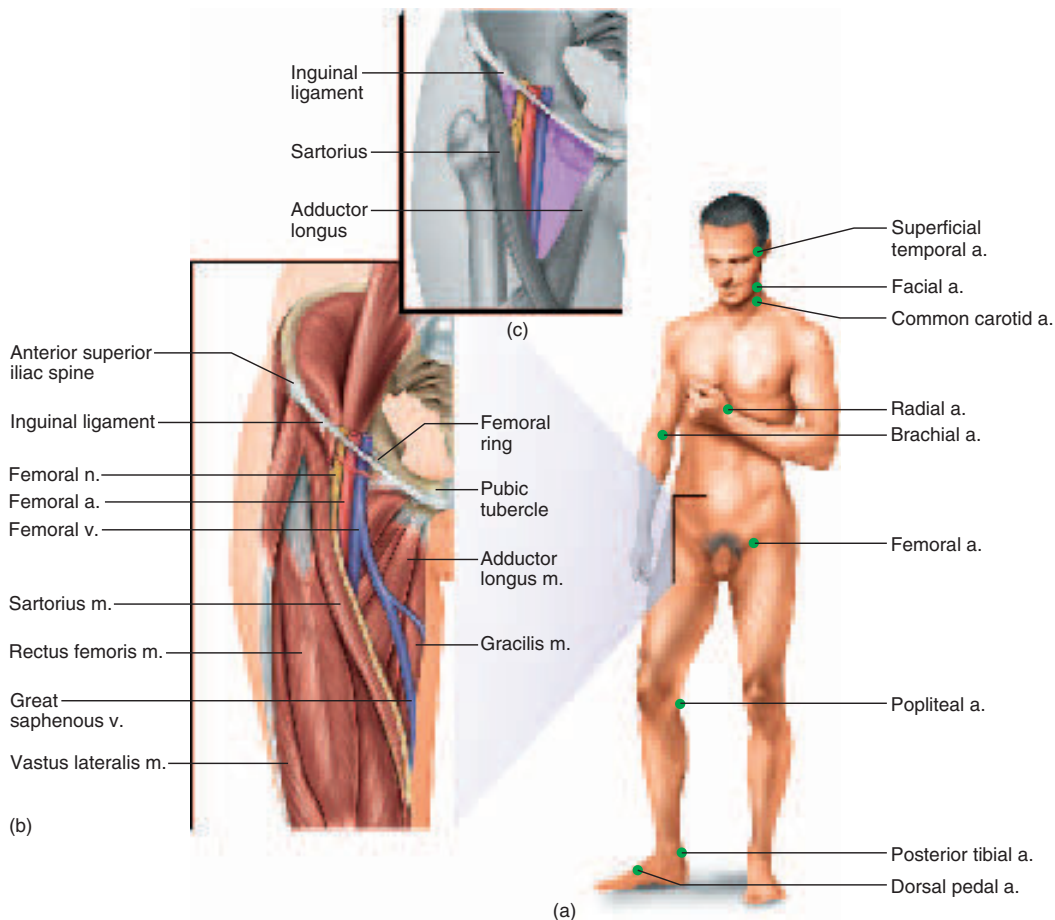


Figure 20.31 Arterial Pressure Points. (a) Areas where arteries lie close enough to the surface that a pulse can be palpated or pressure can be applied to reduce arterial bleeding. (b) Structures in the femoral triangle. (c) Boundaries of the femoral triangle.

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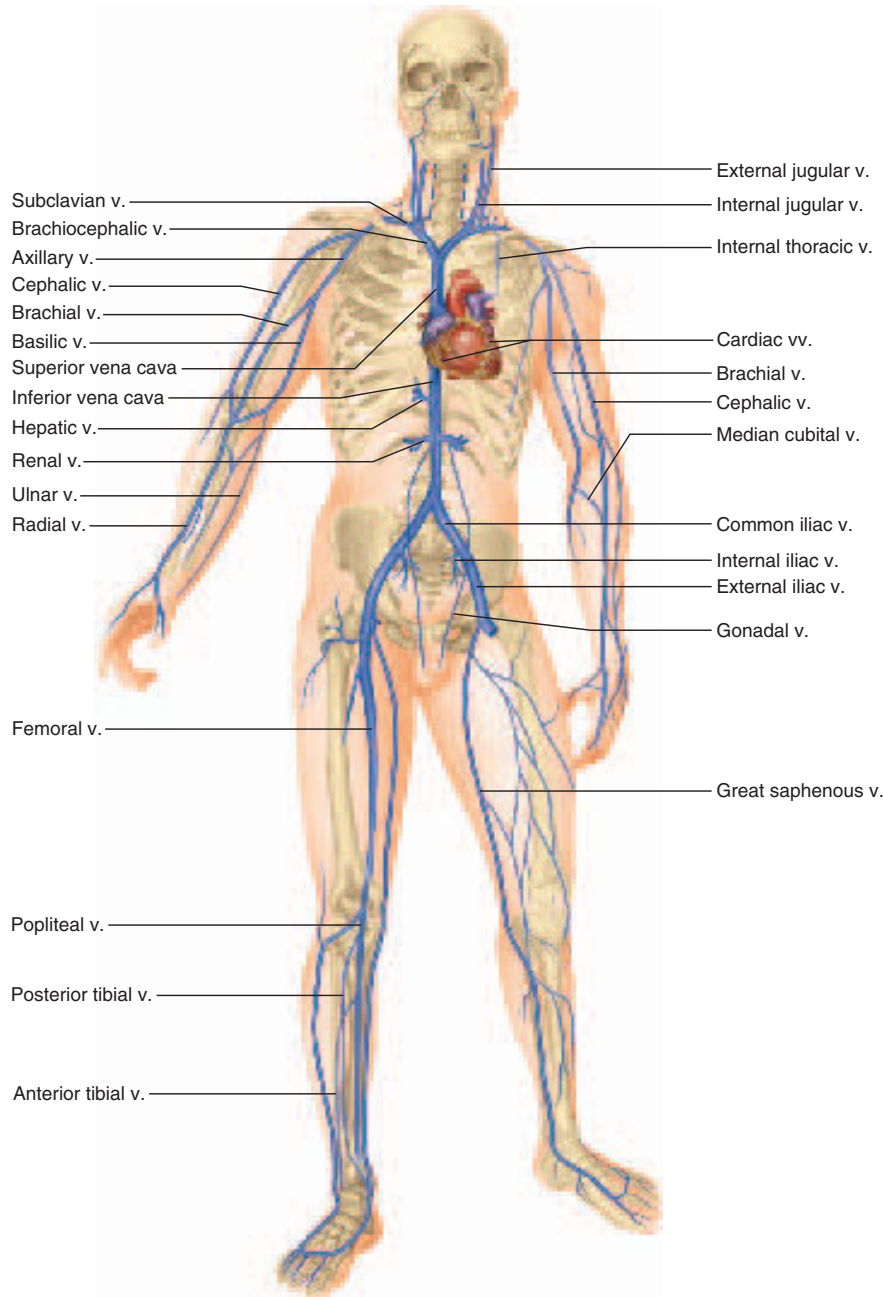


Figure 20.32 The Major Systemic Veins. (v. = vein; vv. = veins)

deep and superficial groups; you may be able to see quite a few of them in your arms and hands. Deep veins run parallel to the arteries and often have similar names (*femoral artery* and *femoral vein*, for example); this is not true of the superficial veins, however. The deep veins are not described in as much detail as the arteries were, since it can usually be assumed that they drain the same structures as the corresponding arteries supply.

In general, we began the study of arteries with those lying close to the heart and progressed away. In the venous system, by contrast, we begin with those that are remote from the heart and follow the flow of blood as they join each other and approach the heart. Venous pathways have more anastomoses than arterial pathways, so the route of blood flow is often not as clear. Many anastomoses are omitted from the following figures for clarity.

Table 20.9 Venous Drainage of the Head and Neck

Most blood of the head and neck is drained by three pairs of veins—the *internal jugular*, *external jugular*, and *vertebral veins*. This table traces their origins and drainage and follows them to the formation of the *brachiocephalic veins* and *superior vena cava* (fig. 20.33).

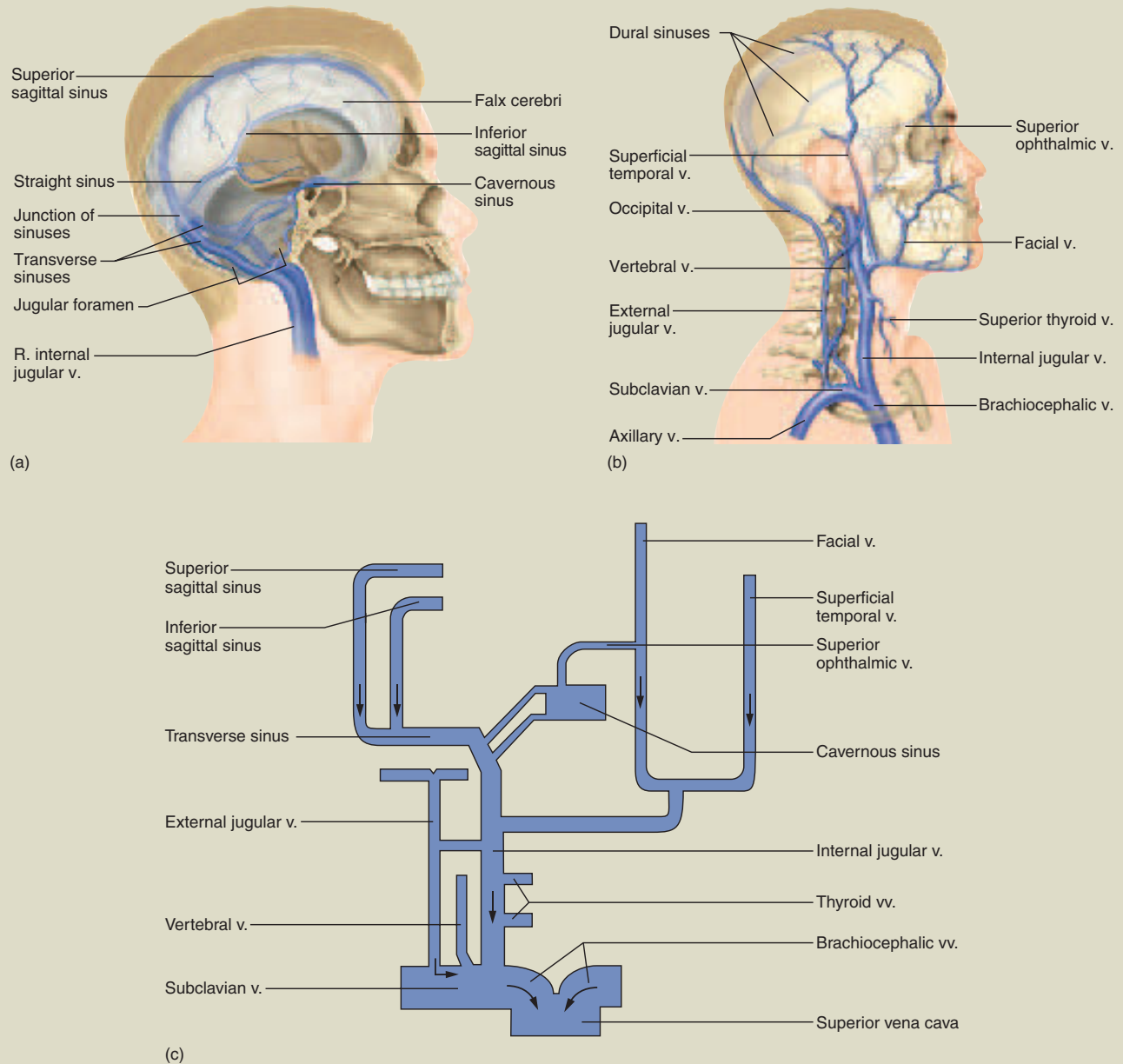


Figure 20.33 Veins Draining the Head and Neck. (a) Deep venous drainage. (b) Superficial venous drainage. (c) Flowchart of venous drainage.

(continued)

Table 20.9 Venous Drainage of the Head and Neck (continued)

Dural Sinuses

Large thin-walled veins called **dural sinuses** occur within the cranial cavity between layers of dura mater. They receive blood from the brain and face and empty into the internal jugular veins:

1. The **superior and inferior sagittal sinuses** are found in the falx cerebri between the cerebral hemispheres; they receive blood that has circulated through the brain.
2. The **cavernous sinuses** occur on each side of the body of the sphenoid bone; they receive blood from the **superior ophthalmic vein** draining the orbit and the **facial vein** draining the nose and upper lip.
3. The **transverse (lateral) sinuses** encircle the inside of the occipital bone and lead to the jugular foramen on each side. They receive blood from the previously mentioned sinuses and empty into the internal jugular veins.

Major Veins of the Neck

Blood flows down the neck mainly through three veins on each side, all of which empty into the subclavian vein:

1. The **internal jugular**²³ (JUG-you-lur) **vein** courses down the neck, alongside the internal carotid artery, deep to the sternocleidomastoid muscle. It receives most of the blood from the brain, picks up blood from the **facial vein** and **superficial temporal vein** along the way, passes deep to the clavicle, and joins the subclavian vein. (Note that the facial vein empties into both the cavernous sinus and the internal jugular vein.)
2. The **external jugular vein** drains tributaries from the parotid gland, facial muscles, scalp, and other superficial structures. Some of this blood also follows venous anastomoses to the internal jugular vein. The external jugular vein courses down the side of the neck superficial to the sternocleidomastoid muscle and empties into the subclavian vein.
3. The **vertebral vein** travels with the vertebral artery in the transverse foramina of the cervical vertebrae. Although the companion artery leads to the brain, the vertebral vein does not come from there. It drains the cervical vertebrae, spinal cord, and some of the small deep muscles of the neck.

Drainage from Shoulder to Heart

From the shoulder region, blood takes the following path to the heart:

1. The **subclavian vein** drains the arm and travels inferior to the clavicle; receives the external jugular, vertebral, and internal jugular veins in that order; and ends where it receives the internal jugular.
2. The **brachiocephalic vein** is formed by union of the subclavian and internal jugular veins. It continues medially and receives tributaries draining the upper thoracic wall and breast.
3. The **superior vena cava** is formed by the union of the right and left brachiocephalic veins. It travels inferiorly for about 7.5 cm and empties into the right atrium. It drains all structures superior to the diaphragm except the pulmonary circuit and coronary circulation. It also receives considerable drainage from the abdominal cavity by way of the azygos system (see table 20.11).

²³jugul = neck, throat

Table 20.10 Venous Drainage of the Upper Limb

Table 20.9 briefly noted the subclavian veins that drain each arm. This table begins distally in the forearm and traces venous drainage to the subclavian vein (fig. 20.34).

Deep Veins

1. The **palmar digital veins** drain each finger into the **superficial venous palmar arch**.
2. The **metacarpal veins** parallel the metacarpal bones and drain blood from the hand into the **deep venous palmar arch**. Both the superficial and deep venous palmar arches are anastomoses between the next two veins, which are the major deep veins of the forearm.
3. The **radial vein** receives blood from the lateral side of both palmar arches and courses up the forearm alongside the radius.
4. The **ulnar vein** receives blood from the medial side of both palmar arches and courses up the forearm alongside the ulna.
5. The **brachial vein** is formed by the union of the radial and ulnar veins at the elbow; it courses up the brachium.
6. The **axillary vein** is formed at the axilla by the union of the brachial and basilic veins (the basilic vein is described in the next section).
7. The **subclavian vein** is a continuation of the axillary vein into the shoulder inferior to the clavicle. The further course of the subclavian is explained in the previous table.

(continued)

Table 20.10 Venous Drainage of the Upper Limb (continued)

Superficial Veins

These are easily seen through the skin of most people and are larger in diameter than the deep veins:

1. The **dorsal venous network** is a plexus of veins visible on the back of the hand; it empties into the major superficial veins of the forearm, the cephalic and basilic.
2. The **cephalic vein** arises from the lateral side of the dorsal venous arch, winds around the radius as it travels up the forearm, continues up the lateral aspect of the brachium to the shoulder, and joins the axillary vein there. Intravenous fluids are often administered through the distal end of this vein.
3. The **basilic²⁴** (bah-SIL-ic) **vein** arises from the medial side of the dorsal venous arch, travels up the posterior aspect of the forearm, and continues into the brachium. About midway up the brachium it turns deeper and runs beside the brachial artery. At the axilla it joins the brachial vein, and the union of these two gives rise to the axillary vein.
4. The **median cubital vein** is a short anastomosis between the cephalic and basilic veins that obliquely crosses the cubital fossa (anterior bend of the elbow). It is clearly visible through the skin and is the most common site for drawing blood.
5. The **median antebrachial vein** originates near the base of the thumb, travels up the forearm between the radial and ulnar veins, and terminates at the elbow; it empties into the cephalic vein in some people and into the basilic vein in others.

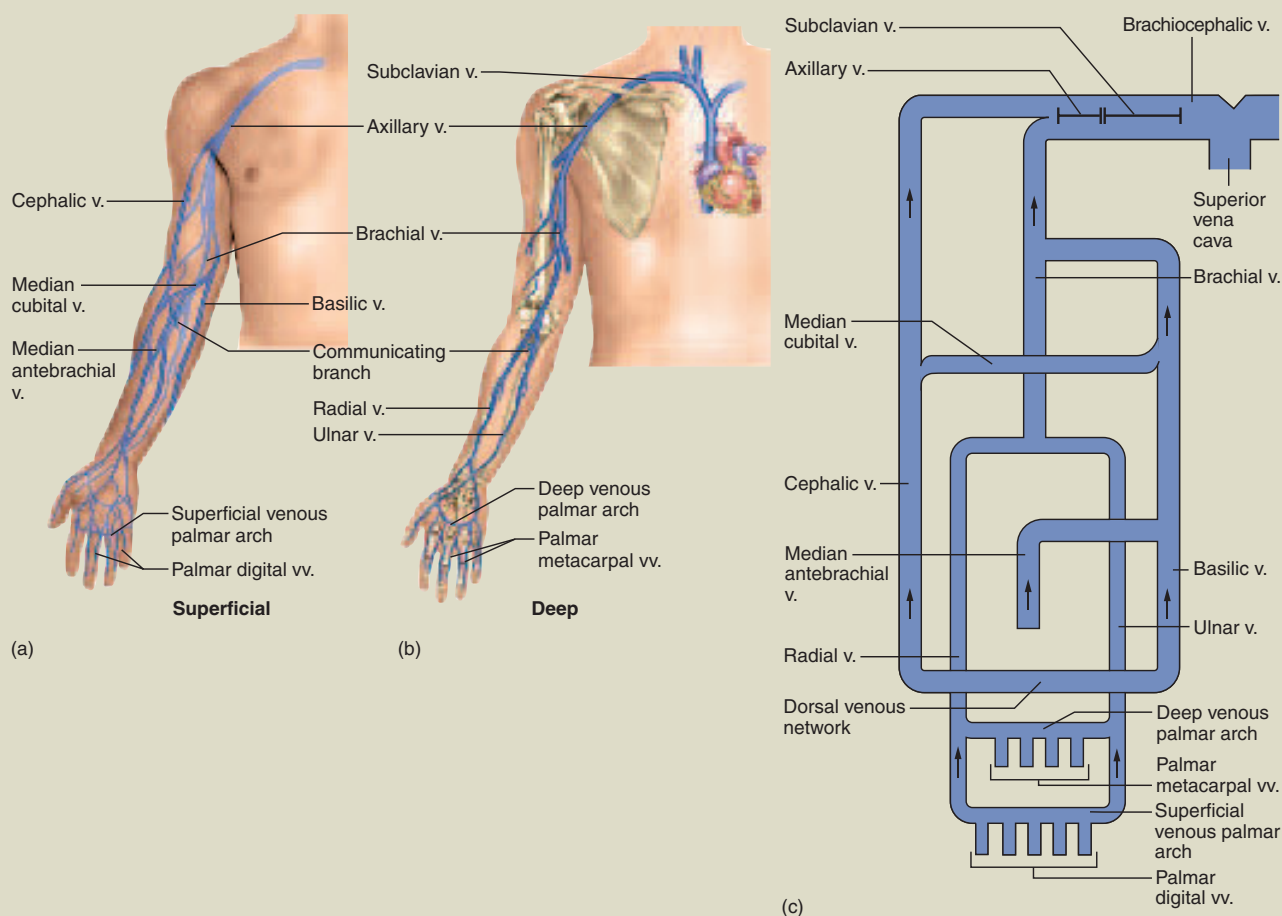


Figure 20.34 Veins Draining the Upper Limb. (a) Superficial venous drainage. (b) Deep venous drainage. (c) Flowchart of venous drainage. Name three veins that are often visible through the skin of the upper limb.

²⁴basilic = royal, prominent, important

Table 20.11 The Azygos System

The superior vena cava receives extensive drainage from the thoracic and abdominal walls by way of the **azygos (AZ-ih-goss) system** (fig. 20.35).

Drainage of the Abdominal Wall

A pair of **ascending lumbar veins** receive blood from the common iliac veins below and a series of short horizontal **lumbar veins** that drain the abdominal wall. The ascending lumbar veins anastomose with the inferior vena cava beside them and ascend through the diaphragm into the thoracic cavity.

Drainage of the Thorax

Right side. After penetrating the diaphragm, the right ascending lumbar vein becomes the **azygos²⁵ vein** of the thorax. The azygos receives blood from the right **posterior intercostal veins**, which drain the chest muscles, and from the **esophageal, mediastinal, pericardial, and right bronchial veins**. It then empties into the superior vena cava at the level of vertebra T4.

Left side. The left ascending lumbar vein continues into the thorax as the **hemiazygos²⁶ vein**. The hemiazygos drains the ninth through eleventh posterior intercostal veins and some esophageal and mediastinal veins on the left. At midthorax, it crosses over to the right side and empties into the azygos vein.

The **accessory hemiazygos vein** is a superior extension of the hemiazygos. It drains the fourth through eighth posterior intercostal veins and the left bronchial vein. It also crosses to the right side and empties into the azygos vein.

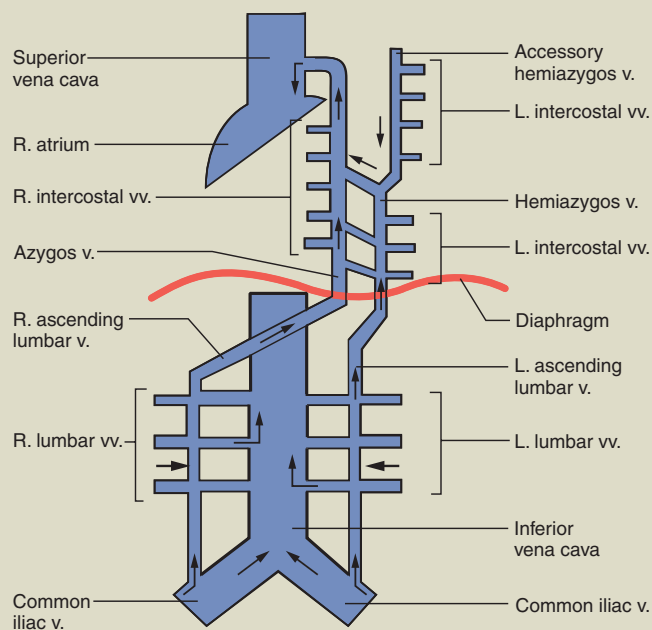


Figure 20.35 Veins of the Azygos System.

²⁵unpaired; from *a* = without + *zygo* = union, mate

²⁶*hemi* = half

Table 20.12 Major Tributaries of the Inferior Vena Cava

The **inferior vena cava (IVC)** is formed by the union of the right and left common iliac veins at the level of vertebra L5. It is retroperitoneal and lies immediately to the right of the aorta. Its diameter of 3.5 cm is the largest of any vessel in the body. As it ascends the abdominal cavity, the IVC picks up blood from numerous tributaries in the order listed here (fig. 20.36):

1. Some **lumbar veins** empty into the IVC as well as into the ascending lumbar veins described in table 20.11.
2. The **gonadal veins** (**ovarian veins** in the female and **testicular veins** in the male) drain the gonads. The right gonadal vein empties directly into the IVC, whereas the **left gonadal vein** empties into the left renal vein.
3. The **renal veins** drain the kidneys into the IVC. The left renal vein also receives blood from the left gonadal and left suprarenal veins.
4. The **suprarenal veins** drain the adrenal (suprarenal) glands. The right suprarenal empties directly into the IVC, and the left suprarenal empties into the renal vein.
5. The **hepatic veins** drain the liver; they extend a short distance from its superior surface to the IVC.
6. The **inferior phrenic veins** drain the inferior aspect of the diaphragm.

After receiving these inputs, the IVC penetrates the diaphragm and enters the right atrium from below. It does not receive any thoracic drainage.

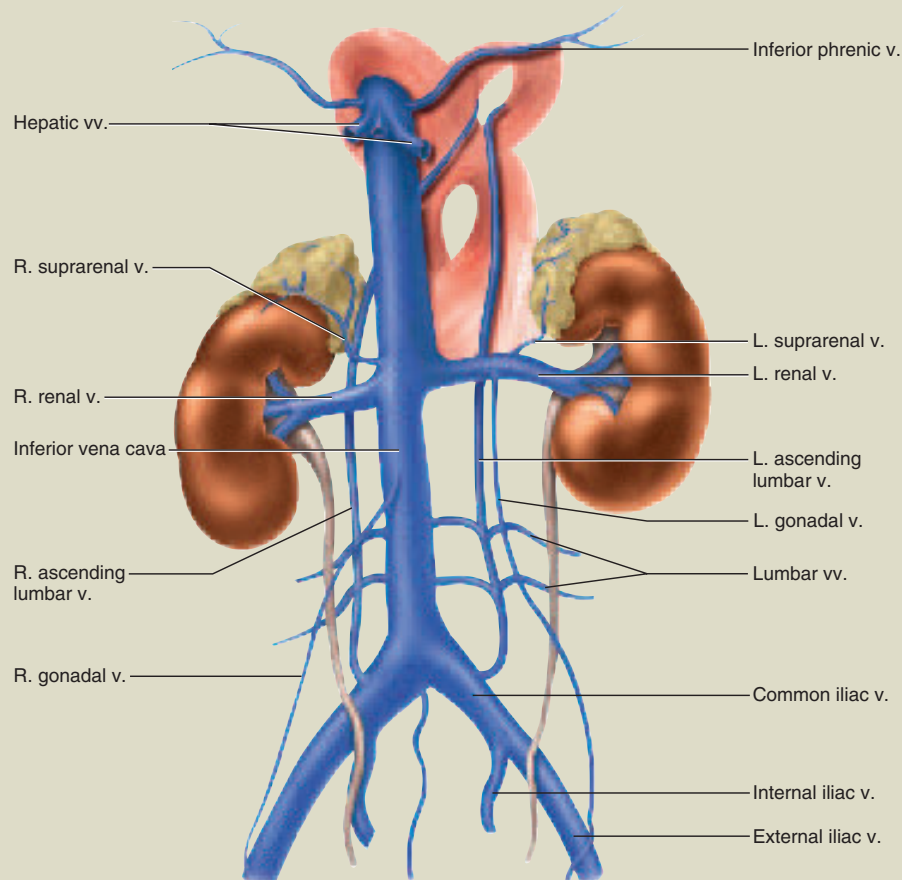


Figure 20.36 The Inferior Vena Cava and Its Tributaries.

Table 20.13 The Hepatic Portal System

The **hepatic portal system** connects capillaries of the intestines and other digestive organs to the **hepatic sinusoids** of the liver. The intestinal blood is richly laden with nutrients for a few hours following a meal. The hepatic portal system gives the liver “first claim” to these nutrients before the blood is distributed to the rest of the body. It also allows the blood to be cleansed of bacteria and toxins picked up from the intestines, an important function of the liver. The route from the intestines to the inferior vena cava follows (fig. 20.37):

1. The **inferior mesenteric vein** receives blood from the rectum and distal part of the large intestine. It converges in a fanlike array in the mesentery and empties into the splenic vein.
2. The **superior mesenteric vein** receives blood from the entire small intestine, ascending colon, transverse colon, and stomach. It, too, exhibits a fanlike arrangement in the mesentery and then joins the splenic vein to form the hepatic portal vein.
3. The **splenic vein** drains the spleen and travels across the abdominal cavity toward the liver. Along the way, it picks up the **pancreatic veins** from the pancreas and the inferior mesenteric vein. It changes name when it joins the superior mesenteric vein, as explained next.

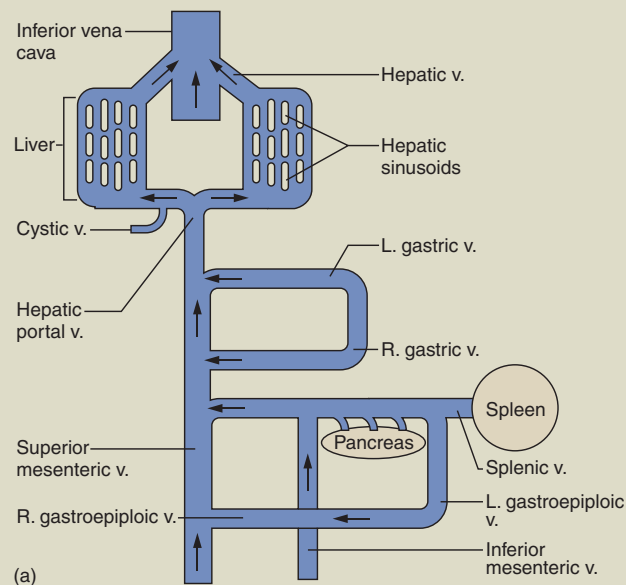


Figure 20.37 Veins of the Hepatic Portal System and Its Tributaries. (a) Flowchart. (continued)

Table 20.13 The Hepatic Portal System (continued)

- The **hepatic portal vein** is formed by convergence of the splenic and superior mesenteric veins. It travels about 8 cm up and to the right and then enters the inferior surface of the liver. Near this point it receives the **cystic vein** from the gallbladder. In the liver, the hepatic portal vein ultimately leads to the innumerable microscopic hepatic sinusoids. Blood from the sinusoids empties into the hepatic veins described earlier. Circulation within the liver is described in more detail in chapter 25.
- The left and right **gastric veins** form an arch along the lesser curvature of the stomach and empty into the hepatic portal vein.

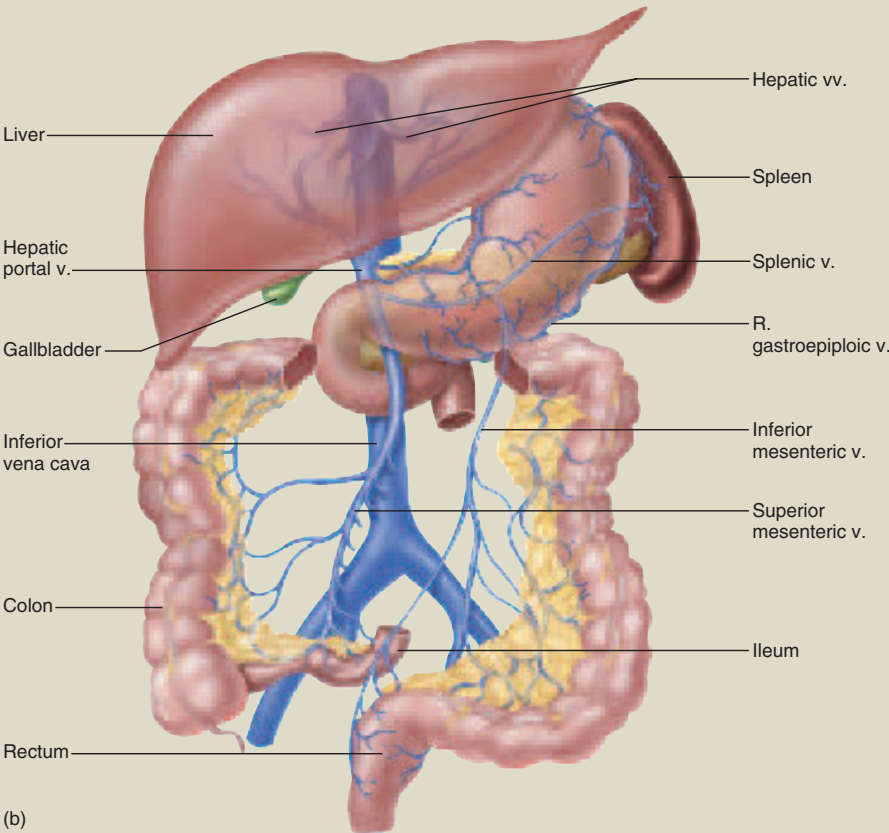


Figure 20.37 Veins of the Hepatic Portal System and Its Tributaries (continued). (b) Anatomy.

Table 20.14 Venous Drainage of the Lower Limb and Pelvic Organs

Drainage of the lower limb is described starting at the toes and following the flow of blood to the inferior vena cava (fig. 20.38). As in the upper limb, there are deep and superficial veins with anastomoses between them.

Deep Veins

1. The **plantar venous arch** drains the plantar aspect of the foot, receives blood from the **plantar digital veins** of the toes, and gives rise to the next vein.
2. The **posterior tibial vein** drains the plantar arch and passes up the leg embedded deep in the calf muscles; it receives drainage along the way from the **fibular (peroneal) vein**.
3. The **dorsal pedal vein** drains the dorsum of the foot.
4. The **anterior tibial vein** is a continuation of the dorsal pedal vein. It travels up the anterior compartment of the leg between the tibia and fibula.

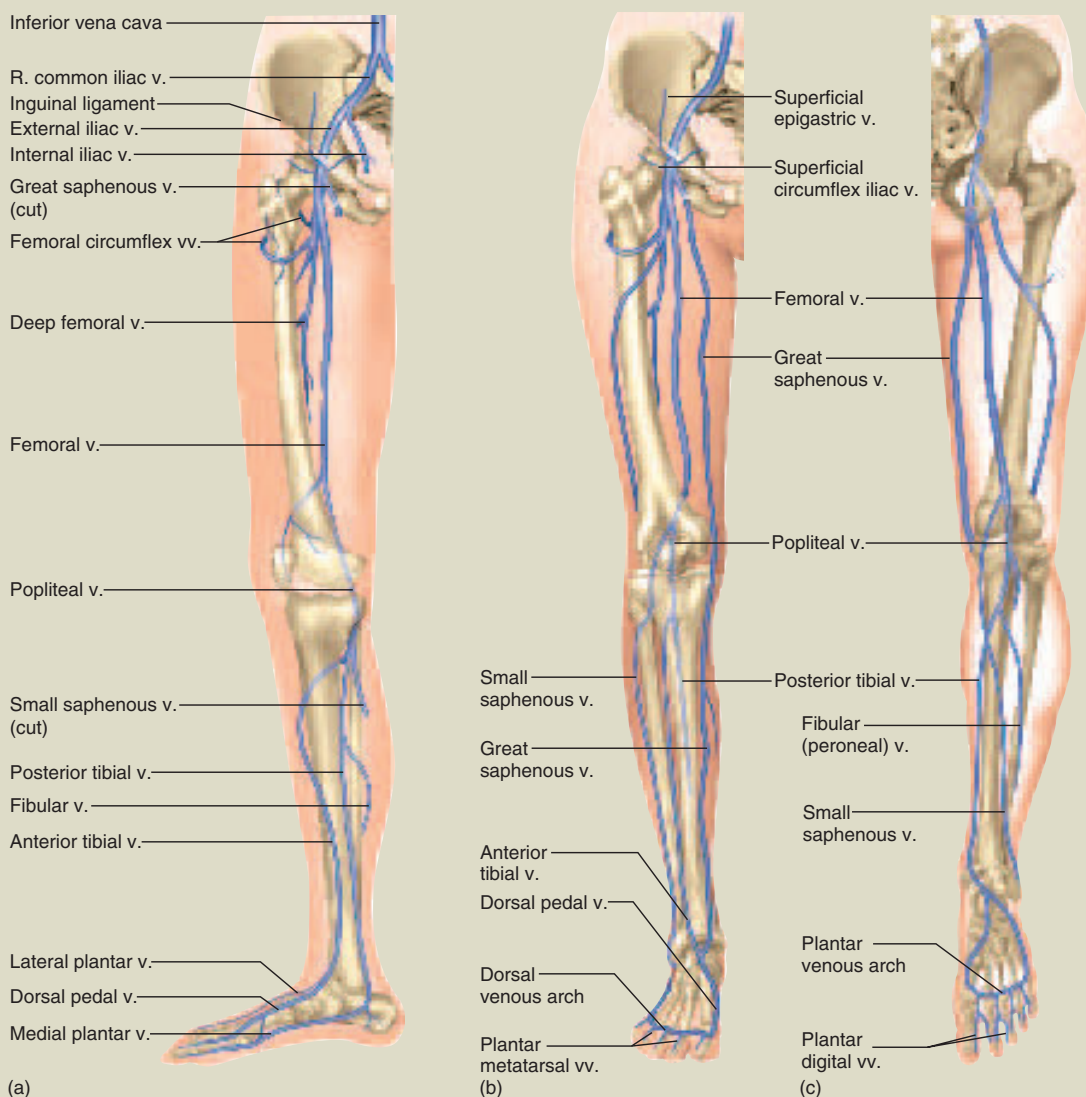


Figure 20.38 Veins Draining the Lower Limb. (a) Deep veins, anteromedial view of the right limb. (b) Anterior aspect of the right limb and dorsal aspect of the foot. (c) Posterior aspect of the right limb and plantar aspect of the foot. *(continued)*

(continued)

Table 20.14 Venous Drainage of the Lower Limb and Pelvic Organs (continued)

5. The **popliteal vein** is formed at the back of the knee by the union of the anterior and posterior tibial veins.
6. The **femoral vein** is a continuation of the popliteal vein into the thigh. It receives drainage from the deep thigh muscles and femur.
7. The **external iliac vein**, superior to the inguinal ligament, is formed by the union of the femoral vein and great saphenous vein (one of the superficial veins described next).
8. The **internal iliac vein** follows the course of the internal iliac artery and its distribution. Its tributaries drain the gluteal muscles; the medial aspect of the thigh; the urinary bladder, rectum, prostate, and ductus deferens in the male; and the uterus and vagina in the female.
9. The **common iliac vein** is formed by the union of the external and internal iliac veins; it also receives blood from the ascending lumbar vein. The right and left common iliacs then unite to form the inferior vena cava.

Superficial Veins

1. The **dorsal venous arch** is visible through the skin on the dorsum of the foot. It has numerous anastomoses similar to the dorsal venous network of the hand.
2. The **great saphenous**²⁷ (sah-FEE-nus) **vein**, the longest vein in the body, arises from the medial side of the dorsal venous arch. It traverses the medial aspect of the leg and thigh and terminates by emptying into the femoral vein, slightly inferior to the inguinal ligament. It is commonly used as a site for the long-term administration of intravenous fluids; it is a relatively accessible vein in infants and in patients in shock whose veins have collapsed. Portions of this vein are commonly excised and used as grafts in coronary bypass surgery.
3. The **small saphenous vein** arises from the lateral side of the dorsal venous arch, courses up the lateral aspect of the foot and through the calf muscles, and terminates at the knee by emptying into the popliteal vein. It has numerous anastomoses with the great saphenous vein. The great and small saphenous veins are among the most common sites of varicose veins.

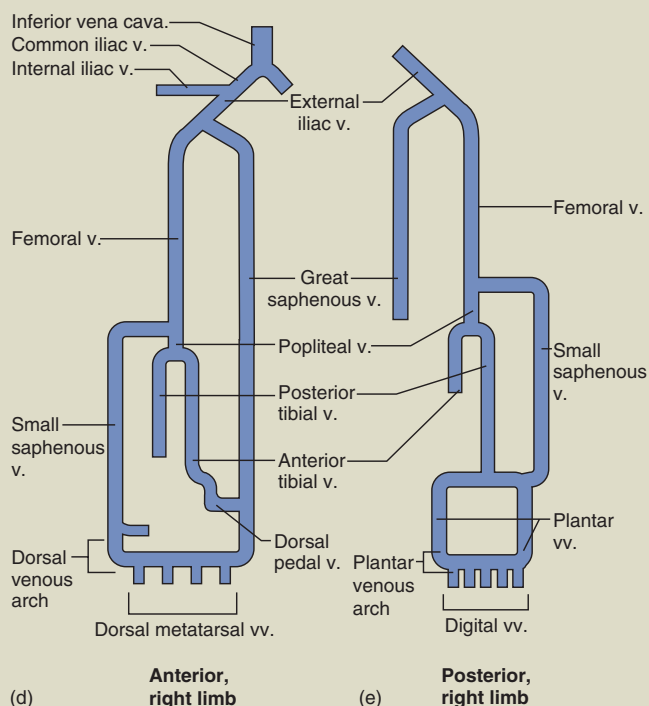


Figure 20.38 Veins Draining the Lower Limb (continued). (d) Flowchart of venous drainage of the right limb, anterior aspect. (e) Flowchart of the same limb, posterior aspect.

²⁷saphen = standing

Table 20.15 Some Disorders of the Arteries and Veins

<i>Dissecting aneurysm</i>	Splitting of the layers of an arterial wall from each other because of the accumulation of blood between layers. Results from either a tear in the tunica intima or rupture of the vasa vasorum.	
<i>Fat embolism</i>	The presence of fat globules traveling in the bloodstream. Globules originate from bone fractures, fatty degeneration of the liver, and other causes and may block cerebral or pulmonary blood vessels.	
<i>Orthostatic hypotension</i>	A decrease in blood pressure that occurs when one stands, often resulting in blurring of vision, dizziness, and syncope (fainting). Results from sluggish or inactive baroreflexes.	
<i>Disorders described elsewhere</i>		
Aneurysm 754	Embolism 707	Stroke 766
Atherosclerosis 741	Hypertension 792	Transient ischemic attack 766
Circulatory shock 765	Hypotension 754	Varicose veins 753
Edema 762		

Chapter 29 describes the effects of aging on the circulatory system, and table 20.15 lists some disorders of the blood vessels. Disorders of the blood and heart are tabulated in chapters 18 and 19.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

- If you were dissecting a cadaver, where would you look for the internal and external jugular veins? What muscle would help you distinguish one from the other?
- How do the vertebral veins differ from the vertebral arteries in their superior terminations?
- By what route does blood from the abdominal wall reach the superior vena cava?
- Trace one possible path of an RBC from the fingertips to the right atrium and name the veins along the way.
- State two ways in which the great saphenous vein has special clinical significance. Where is this vein located?

Insight 20.4 Clinical Application

Hypertension—The “Silent Killer”

Hypertension, the most common cardiovascular disease, affects about 30% of Americans over age 50 and 50% by age 74. It is a “silent killer” that can wreak its destructive effects for 10 to 20 years before its effects are first noticed. Hypertension is the major cause of heart failure, stroke, and kidney failure. It damages the heart because it increases the afterload, which makes the ventricles work harder to expel blood. The myocardium enlarges up to a point (the *hypertrophic response*), but eventually it becomes excessively stretched and less efficient. Hypertension strains the blood vessels and tears the endothelium, thereby creating lesions that become focal points of atherosclerosis. Atherosclerosis then worsens the hypertension and establishes an insidious positive feedback cycle.

Another positive feedback cycle involves the kidneys. Their arterioles thicken in response to the stress, their lumens become narrower, and renal blood flow declines. When the kidneys detect the resulting drop in blood pressure, they release renin, which leads to the formation of the vasoconstrictor angiotensin II and the release of aldosterone, a hormone that promotes salt retention (described in detail in chapter 24). These effects worsen the hypertension that already existed. If diastolic pressure exceeds 120 mmHg, blood vessels of the eye hemorrhage, blindness ensues, the kidneys and heart deteriorate rapidly, and death usually follows within 2 years.

Primary hypertension, which accounts for 90% of cases, results from such a complex web of behavioral, hereditary, and other factors that it is difficult to sort out any specific underlying cause. It was once considered such a normal part of the “essence” of aging that it continues to be called by another name, *essential hypertension*. That term suggests a fatalistic resignation to hypertension as a fact of life, but this need not be. Many risk factors have been identified, and most of them are controllable.

One of the chief culprits is obesity. Each pound of extra fat requires miles of additional blood vessels to serve it, and all of this added vessel length increases peripheral resistance and blood pressure. Just carrying around extra weight, of course, also increases the workload on the heart. Even a small weight loss can significantly reduce blood pressure. Sedentary behavior is another risk factor. Aerobic exercise helps to reduce hypertension by controlling weight, reducing emotional tension, and stimulating vasodilation.

Dietary factors are also significant contributors to hypertension. Diets high in cholesterol and saturated fat contribute to atherosclerosis. Potassium and magnesium reduce blood pressure; thus, diets deficient in these minerals promote hypertension. The relationship of salt intake to hypertension has been a very controversial subject. The kidneys compensate so effectively for excess salt intake that dietary salt has little effect on the blood pressure of most people. Reduced salt intake may, however, help to control hypertension in older people and in people with reduced renal function.

Nicotine makes a particularly devastating contribution to hypertension because it stimulates the myocardium to beat faster and harder; it also stimulates vasoconstriction and thus increases the afterload against which the myocardium must work. Just when the heart needs extra oxygen, nicotine causes coronary vasoconstriction and promotes myocardial ischemia.

Chapter 20 The Circulatory System: Blood Vessels and Circulation 793

Some risk factors cannot be changed at will—race, heredity, and sex. Hypertension runs in some families. A person whose parents or siblings have hypertension is more likely than average to develop it. The incidence of hypertension is about 30% higher, and the incidence of strokes about twice as high, among blacks as among whites. From ages 18 to 54, hypertension is more common in men, but above age 65, it is more common in women. Even people at risk from these factors, however, can minimize their chances of hypertension by changing risky behaviors.

Treatments for primary hypertension include weight loss, diet, and certain drugs. Diuretics lower blood volume and pressure by promoting urination. ACE inhibitors block the formation of the vasoconstrictor

angiotensin II. Beta-blockers such as propranolol block the vasoconstrictive action of the sympathetic nervous system. Calcium channel blockers such as verapamil and nifedipine inhibit the inflow of calcium into cardiac and smooth muscle, thus inhibiting their contraction and promoting vasodilation and reduced cardiac workload.

Secondary hypertension, which accounts for about 10% of cases, is high blood pressure that is secondary to (results from) other identifiable disorders. These include kidney disease (which may cause renin hypersecretion), atherosclerosis, hyperthyroidism, Cushing syndrome, and polycythemia. Secondary hypertension is corrected by treating the underlying disease.

Connective Issues

Interactions Between the CIRCULATORY SYSTEM and Other Organ Systems

- ← indicates ways in which this system affects other systems
- indicates ways in which other systems affect this one

All Systems

Circulatory system delivers O₂ and nutrients to all other systems and carries away wastes; carries heat from deeper organs to skin for elimination

Integumentary System

- ← Dermal blood flow affects sweat production
- Serves as blood reservoir; helps to regulate blood temperature

Skeletal System

- ← Provides minerals for bone deposition; delivers erythropoietin to bone marrow and delivers hormones that regulate skeletal growth
- Skeleton provides protective enclosure for heart and thoracic vessels; serves as reservoir of calcium needed for heart contractions; bone marrow carries out hemopoiesis

Muscular System

- ← Removes heat generated by exercise
- Helps to regulate blood temperature; respiratory and limb muscles aid venous return; aerobic exercise enhances circulatory efficiency

Nervous System

- ← Endothelial cells of blood vessels maintain blood-brain barrier and help to produce CSF
- Modulates heart rate, strength of contraction, and vasomotion; governs routing of blood flow; monitors blood pressure and composition and activates homeostatic mechanisms to regulate these

Endocrine System

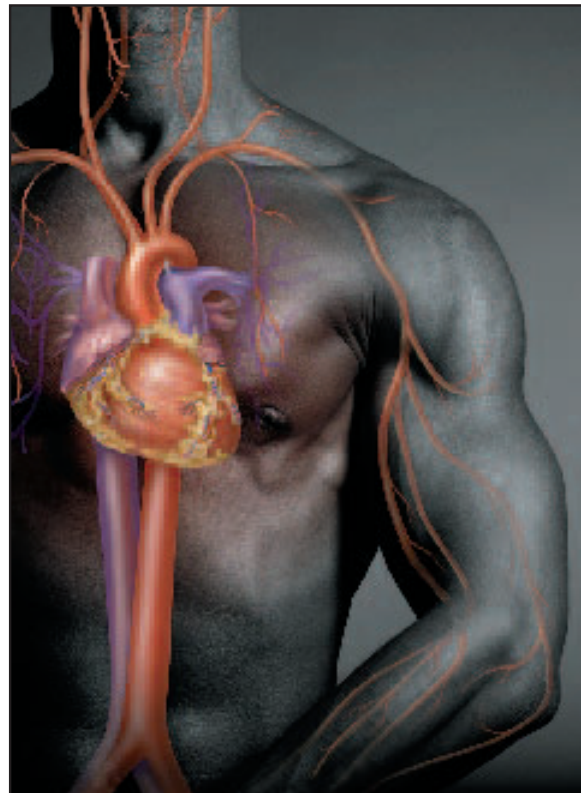
- ← Transports hormones to their target cells
- Regulates blood volume and pressure; stimulates hemopoiesis

Lymphatic/Immune System

- ← Produces tissue fluid, which becomes lymph; provides the WBCs and plasma proteins employed in immunity
- Lymphatic and circulatory systems jointly regulate fluid balance; lymphatic system returns fluid to bloodstream; spleen acts as RBC and platelet reservoir; lymphatic tissues produce lymphocytes; immune cells protect circulatory system from pathogens

Respiratory System

- ← Delivers and carries away respiratory gases; low capillary blood pressure keeps alveoli dry
- Site of exchange for blood gases; helps to regulate blood pH; thoracic pump aids venous return



Urinary System

- ← Blood pressure maintains kidney function
- Controls blood volume, pressure, and composition; initiates renin-angiotensin-aldosterone mechanism; regulates RBC count by producing erythropoietin

Digestive System

- ← Carries away absorbed nutrients; helps to reabsorb and recycle bile salts and minerals from intestines
- Provides nutrients for hemopoiesis; affects blood composition

Reproductive System

- ← Distributes sex hormones; vasodilation causes erection
- Estrogens may slow development of atherosclerosis in women; testosterone stimulates erythropoiesis

Chapter Review

Review of Key Concepts

General Anatomy of the Blood Vessels (p. 748)

1. Blood flows away from the heart in arteries and back to the heart in veins.
2. Between the arteries and veins, it normally flows through one capillary bed. Portal systems and anastomoses are exceptions to this rule.
3. The wall of a blood vessel has three layers: *tunica externa*, *tunica media*, and *tunica intima*. The tunica intima is lined with a simple squamous *endothelium*.
4. Arteries are classified as *conducting*, *distributing*, and *resistance arteries* from largest to smallest. Conducting arteries are subject to the highest blood pressure and have the most elastic tissue; distributing and resistance arteries contain more smooth muscle relative to their size.
5. The smallest of the resistance arteries are *arterioles*. *Metarterioles* link arterioles with capillaries.
6. Capillaries are the primary point of fluid exchange with the tissues. Their wall is composed of endothelium and basement membrane only.
7. Capillaries are arranged in networks called capillary beds, supplied by a single metarteriole. Precapillary sphincters regulate blood flow through a capillary bed.
8. The two types of capillaries are *continuous* and *fenestrated*. *Sinusoids* are irregular blood spaces of either the continuous or fenestrated type.
9. The smallest veins, or *venules*, also exchange fluid with the tissues. They converge to form medium veins, and medium veins converge to form large veins.
10. Veins have relatively low blood pressures and therefore have thinner walls and less muscular and elastic tissue. Medium veins of the limbs have valves to prevent the backflow of blood.

Blood Pressure, Resistance, and Flow (p. 753)

1. *Blood flow* (mL/min) and *perfusion* (flow/g of tissue) vary with the metabolic needs of a tissue.
2. Flow (F) is directly proportional to the pressure difference between two points (ΔP) and inversely proportional to resistance (R): $F \propto \Delta P/R$.
3. Blood pressure (BP) is usually measured with a sphygmomanometer. Arterial pressures are expressed as systolic over diastolic pressure—for example, 120/80 mmHg.
4. *Pulse pressure* is systolic minus diastolic pressure. *Mean arterial pressure* is the average pressure in a vessel over the course of a cardiac cycle, estimated as diastolic pressure + $1/3$ of pulse pressure.
5. Chronic, abnormally high BP is *hypertension* and low BP is *hypotension*.
6. The expansion and contraction of arteries during the cardiac cycle reduces the pulse pressure and eases the strain on smaller arteries, but arterial blood flow is nevertheless pulsatile. In capillaries and veins, flow is steady (without pulsation).
7. *Peripheral resistance* is resistance to blood flow in the blood vessels. Resistance is directly proportional to blood viscosity and vessel length, and inversely proportional to vessel radius to the fourth power (r^4). Changes in vessel radius (*vasomotion*) thus have the greatest influence on flow from moment to moment.
8. Blood flow is fastest in the aorta, slowest in the capillaries, and speeds up somewhat in the veins.
9. Blood pressure is controlled mainly by local, neural, and hormonal control of vasomotion.
10. *Autoregulation* is the ability of a tissue to regulate its own blood supply. Over the short term, local vasomotion is stimulated by *vasoactive chemicals* (histamine,

nitric oxide, and others). Over the long term, autoregulation can be achieved by *angiogenesis*, the growth of new vessels.

11. Neural control of blood vessels is based in the *vasomotor center* of the medulla oblongata. This center integrates *baroreflexes*, *chemoreflexes*, and the *medullary ischemic reflex*, and issues signals to the blood vessels by way of sympathetic nerve fibers.
12. Blood pressure is regulated in various ways by the hormones angiotensin II, aldosterone, atrial natriuretic peptide, antidiuretic hormone, epinephrine, and norepinephrine.
13. Vasomotion often shifts blood flow from organs with less need of perfusion at a given time, to organs with greater need—for example, away from the intestines and to the skeletal muscles during exercise.

Capillary Exchange (p. 761)

1. *Capillary exchange* is a two-way movement of water and solutes between the blood and tissue fluids across the walls of the capillaries and venules.
2. Materials pass through the vessel wall by diffusion, transcytosis, filtration, and reabsorption, passing through intercellular clefts, fenestrations, and the endothelial cell cytoplasm.
3. Fluid is forced out of the vessels by blood pressure and the negative hydrostatic pressure of the interstitial space. The force drawing fluid back into the capillaries is colloid osmotic pressure. The difference between the outward and inward forces is an outward *net filtration pressure* or an inward *net reabsorption pressure*.
4. Capillaries typically give off fluid at the arterial end, where the relatively high blood pressure overrides reabsorption; they reabsorb about 85% as much fluid at the venous end, where colloid osmotic pressure overrides the lower blood pressure.

- About 15% of the tissue fluid is reabsorbed by the lymphatic system.
- Fluid exchange dynamics vary from place to place in the body (some capillaries engage solely in filtration and some solely in reabsorption) and from moment to moment (as vasomotion shifts the balance between filtration and reabsorption).
- Accumulation of excess tissue fluid is *edema*. It results from increased capillary filtration, reduced reabsorption, or obstructed lymphatic drainage.

Venous Return and Circulatory Shock (p. 763)

- Venous return*, the flow of blood back to the heart, is driven by the venous blood pressure gradient, gravity, the skeletal muscle pump (aided by valves in the veins of the limbs), the thoracic pump, and cardiac suction.
- Exercise increases venous return because the vessels dilate, the thoracic pump and skeletal muscle pump work more energetically, and cardiac output is elevated.
- Inactivity allows blood to accumulate in low points in the body by gravity; this is called *venous pooling*. It can result in *syncope* (fainting) if too much blood drains away from the brain.
- Circulatory shock* is any state of inadequate cardiac output. Its two basic categories are *cardiogenic shock* and *low venous return (LVR) shock*.
- The main forms of LVR shock are *hypovolemic*, *obstructed venous return*, and *venous pooling shock*.
- Septic shock* and *anaphylactic shock* combine elements of hypovolemia and venous pooling.
- Compensated shock* is corrected by the body's homeostatic mechanisms. *Decompensated shock* is life-threatening, incapable of self-correction, and requires clinical intervention.

Special Circulatory Routes (p. 766)

- The brain receives a relatively stable total blood flow of about 700 mL/min, but flow shifts rapidly from one part of the brain to another during varying cerebral activities.

- The brain regulates its own blood flow in response to changes in BP and pH.
- Transient ischemic attacks* result from brief periods of cerebral ischemia (poor blood flow). A *cerebral vascular accident* (stroke) results from a permanent loss of perfusion due to arterial blockage or rupture.
- Skeletal muscles receive highly variable flow depending on their state of activity. Most muscle capillary beds are shut down at rest. During exercise, flow increases in response to muscle metabolites and sympathetic vasodilation.
- The pulmonary circuit is the only route in which arteries carry less oxygen than veins do.
- Pulmonary arteries have relatively low BP and slow flow, which allows ample time for gas exchange and promotes capillary reabsorption. The latter prevents fluid from accumulating in the lungs.
- Pulmonary arteries, unlike systemic arteries, constrict in response to hypoxia, so less blood is sent to poorly ventilated areas of the lung.

Anatomy of the Pulmonary Circuit (p. 767)

- The route of blood flow in the pulmonary circuit is right ventricle of the heart → pulmonary trunk → pulmonary arteries → lobar arteries → alveolar capillary beds → venules → pulmonary veins → left atrium of the heart.
- The pulmonary circuit serves only to exchange CO₂ for O₂ in the blood. The metabolic needs of the lung tissue are met by a separate systemic blood supply to the lungs, via the bronchial arteries.

Anatomy of the Systemic Arteries (p. 767)

- The systemic circulation begins with the ascending aorta. Table 20.3 describes the major branches of the aorta.
- The head and neck receive blood from the common carotid and vertebral arteries. Table 20.4 describes the branches of these arteries.

- The upper limbs receive blood from the subclavian arteries. Table 20.5 describes the branches of these arteries in the limb.
- The thoracic organs receive blood from several small branches of the thoracic aorta and the subclavian and axillary arteries. Table 20.6 describes these branches.
- After passing through the diaphragm, the descending aorta gives off a series of branches to the abdominal viscera. Table 20.7 describes these.
- At its inferior end, the abdominal aorta forks into two common iliac arteries, whose distal branches supply the pelvic region and lower limb. Table 20.8 describes these.
- Arteries tend to be deeper than veins, but there are several places where they come close enough to the surface to be palpated. These sites serve for taking a pulse and as emergency *pressure points* where compression can stop arterial bleeding.

Anatomy of the Systemic Veins (p. 781)

- In venous circulation, blood flows through smaller veins that join to form progressively larger ones. Veins that merge to create a larger one are called *tributaries*.
- The head and neck are drained by the jugular and vertebral veins, which ultimately converge to form the *superior vena cava* leading to the right atrium of the heart. Table 20.9 describes the tributaries that drain the head and neck.
- Table 20.10 describes tributaries in the upper limb that converge to drain the limb via the axillary and subclavian veins.
- The thoracic viscera are drained by the *azygos system*, described in table 20.11.
- The abdominal viscera are drained by tributaries of the *inferior vena cava (IVC)*, described in table 20.12.
- The digestive system is drained by the *hepatic portal system* of veins, described in table 20.13.
- Table 20.14 describes tributaries of the lower limbs, which ultimately converge on the *common iliac veins*. The two common iliac veins join to form the IVC.

Selected Vocabulary

portal system 748
anastomosis 748
endothelium 750
artery 750
arteriole 750
continuous capillary 752

fenestrated capillary 752
sinusoid 752
venule 752
perfusion 753
systolic pressure 754

diastolic pressure 754
vasoconstriction 755
vasodilation 755
angiogenesis 757
baroreceptor 758

capillary exchange 761
oncotic pressure 762
edema 762
circulatory shock 765
anaphylactic shock 765

Testing Your Recall

- Blood normally flows into a capillary bed from
 - the distributing arteries.
 - the conducting arteries.
 - a metarteriole.
 - a thoroughfare channel.
 - the venules.
- Plasma solutes enter the tissue fluid most easily from
 - continuous capillaries.
 - fenestrated capillaries.
 - arteriovenous anastomoses.
 - collateral vessels.
 - venous anastomoses.
- A blood vessel adapted to withstand a high pulse pressure would be expected to have
 - an elastic tunica media.
 - a thick tunica intima.
 - one-way valves.
 - a flexible endothelium.
 - a rigid tunica media.
- The substance most likely to cause a rapid drop in blood pressure is
 - epinephrine.
 - norepinephrine.
 - angiotensin II.
 - serotonin.
 - histamine.
- A person with a systolic blood pressure of 130 mmHg and a diastolic pressure of 85 mmHg would have a mean arterial pressure of about
 - 85 mmHg.
 - 100 mmHg.
 - 108 mmHg.
 - 115 mmHg.
 - 130 mmHg.
- The velocity of blood flow decreases if
 - vessel radius increases.
 - blood pressure increases.
 - viscosity increases.
 - afterload increases.
 - vasomotion decreases.
- Blood flows faster in a venule than in a capillary because venules
 - have one-way valves.
 - exhibit vasomotion.
 - are closer to the heart.
 - have higher blood pressures.
 - have larger diameters.
- In a case where interstitial hydrostatic pressure is negative, the only force causing capillaries to reabsorb fluid is
 - colloid osmotic pressure of the blood.
 - colloid osmotic pressure of the tissue fluid.
 - capillary hydrostatic pressure.
 - interstitial hydrostatic pressure.
 - net filtration pressure.
- Intestinal blood flows to the liver by way of
 - the superior mesenteric artery.
 - the celiac trunk.
 - the inferior vena cava.
 - the azygos system.
 - the hepatic portal system.
- The brain receives blood from all of the following vessels *except* the ____ artery or vein.
 - basilar
 - vertebral
 - internal carotid
 - internal jugular
 - anterior communicating
- The highest arterial blood pressure attained during ventricular contraction is called ____ pressure. The lowest attained during ventricular relaxation is called ____ pressure.
- The capillaries of skeletal muscles are of the structural type called ____.
- ____ shock occurs as a result of exposure to an antigen to which one is hypersensitive.
- The role of breathing in venous return is called the ____.
- The difference between the colloid osmotic pressure of blood and that of the tissue fluid is called ____.
- Movement across the capillary endothelium by the uptake and release of fluid droplets is called ____.
- All efferent fibers of the vasomotor center belong to the ____ division of the autonomic nervous system.
- The pressure sensors in the major arteries near the head are called ____.
- Most of the blood supply to the brain comes from a ring of arterial anastomoses called ____.
- The major superficial veins of the arm are the ____ on the medial side and ____ on the lateral side.

True or False

Determine which five of the following statements are false, and briefly explain why.

1. In some circulatory pathways, blood can get from an artery to a vein without going through capillaries.
2. In some cases, a blood cell may pass through two capillary beds in a single trip from left ventricle to right atrium.
3. The body's longest blood vessel is the great saphenous vein.
4. Arteries have a series of valves that ensure a one-way flow of blood.
5. If the radius of a blood vessel doubles and all other factors remain the same, blood flow through that vessel also doubles.
6. The femoral triangle is bordered by the inguinal ligament, sartorius muscle, and adductor longus muscle.
7. The lungs receive both pulmonary and systemic blood.
8. Blood capillaries must reabsorb all the fluid they emit, or else edema will occur.
9. An aneurysm is a ruptured blood vessel.
10. Anaphylactic shock is a form of hypovolemic shock.

Answers in Appendix B

Testing Your Comprehension

1. It is a common lay perception that systolic blood pressure should be 100 plus a person's age. Evaluate the validity of this statement.
2. Calculate the net filtration or reabsorption pressure at a point in a hypothetical capillary assuming a hydrostatic blood pressure of 28 mmHg, an interstitial hydrostatic pressure of -2 mmHg, a blood COP of 25 mmHg, and an interstitial COP of 4 mmHg. Give the magnitude (in mmHg) and direction (in or out) of the net pressure.
3. Aldosterone secreted by the adrenal gland must be delivered to the kidney immediately below. Trace the route that an aldosterone molecule must take from the adrenal gland to the kidney, naming all major blood vessels in the order traveled.
4. People in shock commonly exhibit paleness, cool skin, tachycardia, and a weak pulse. Explain the physiological basis for each of these signs.
5. Discuss why it is advantageous to have baroreceptors in the aortic arch and carotid sinus rather than in some other location such as the common iliac arteries.

Answers at the Online Learning Center

Answers to Figure Legend Questions

- 20.2 Veins are subjected to less pressure than arteries and have less need of elasticity.
- 20.17 Nothing would happen if he lifted his finger from point *O* because the valve at that point would prevent blood from flowing downward and filling the vein. If he lifted his finger from point *H*, blood would flow upward, fill the vein, and the vein between *O* and *H* would stand out.
- 20.22 Aorta \rightarrow left common carotid a. \rightarrow external carotid a. \rightarrow superficial temporal a.
- 20.30 The deep and superficial palmar arches.
- 20.34 The cephalic, basilic, and median cubital vv.

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