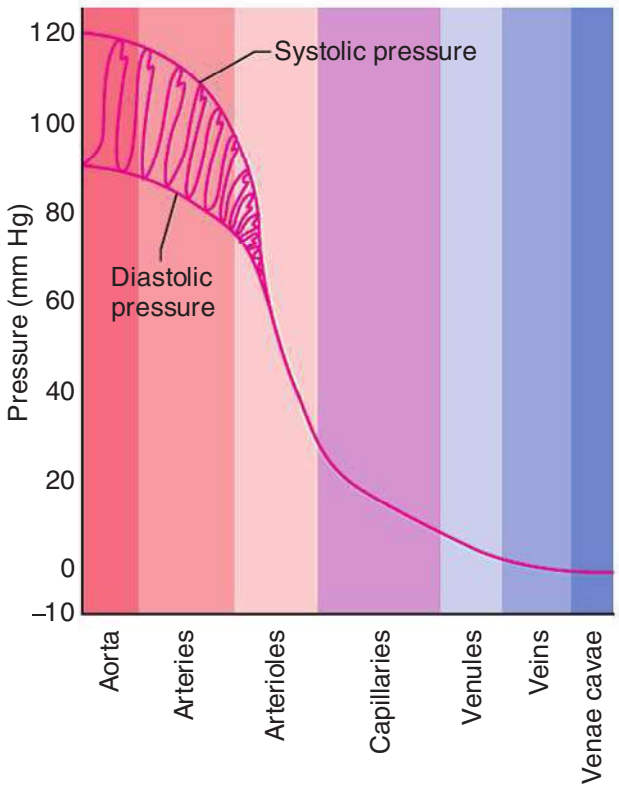


Figure 11.20 **Blood pressure in various areas of the cardiovascular system.**

Unless stated otherwise, the term *blood pressure* is understood to mean the pressure within the large systemic arteries near the heart.

Blood Pressure Gradient When the ventricles contract, they force blood into large, thick-walled elastic arteries close to the heart that expand as the blood is pushed into them. The high pressure in these arteries forces the blood to continuously move into areas where the pressure is lower. The pressure is highest in the large arteries and continues to drop throughout the systemic and pulmonary pathways, reaching either zero or negative pressure at the venae cavae (**Figure 11.20**). Recall that the blood flows into the smaller arteries, then arterioles, capillaries, venules, veins, and finally back to the large venae cavae entering the right atrium of the heart. It flows continuously along a pressure gradient (from high to low pressure) as it makes its circuit day in and day out. Notice that if venous return depended entirely on a high blood pressure throughout the system, blood would probably never be able to complete its circuit back to the heart. This is why the valves in the larger veins, the milking activity of the skeletal muscles, and pressure changes in the thorax are so important.

The pressure differences between arteries and veins become very clear when these vessels are cut. If a vein is cut, the blood flows evenly from the wound; a lacerated artery produces rapid spurts of blood.

Continuous blood flow absolutely depends on the stretchiness of the larger arteries and their ability to recoil and keep exerting pressure on the blood as it flows off into the circulation. Think of a garden hose with relatively hard walls. When the water is turned on, the water spurts out under high pressure because the hose walls don't expand. However, when the water faucet is suddenly turned off, the flow of water stops just as abruptly. The reason is that the walls of the hose cannot recoil to keep pressure on the water; therefore, the pressure drops and the flow of water stops. The importance of the elasticity of the arteries is best appreciated when it is lost, as happens in *arteriosclerosis*. We discuss arteriosclerosis, also called "hardening of the arteries," in "A Closer Look" on pp. 388–389.

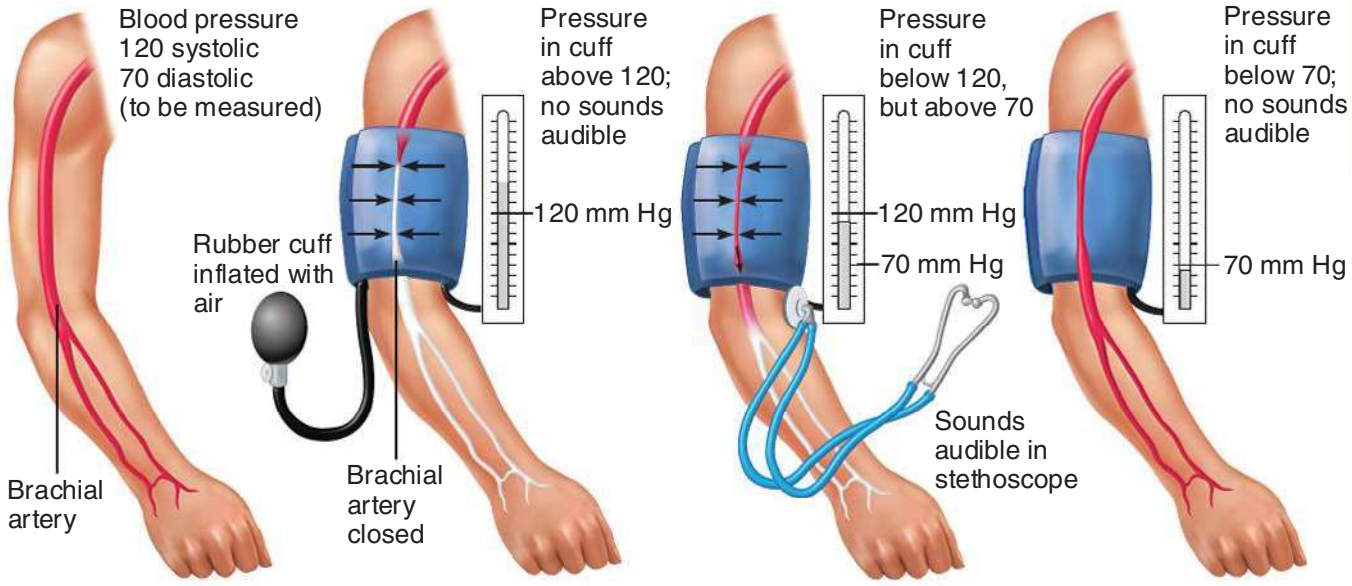
DID YOU GET IT?

21. How does blood pressure change throughout the systemic circulatory pathway?

For the answer, see Appendix D.

Measuring Blood Pressure Because the heart alternately contracts and relaxes, the off-and-on flow of blood into the arteries causes the blood pressure to rise and fall during each beat. Thus, two arterial blood pressure measurements are usually made: **systolic** (sis-to′lik) **pressure,** the pressure in the arteries at the peak of ventricular contraction, and **diastolic** (di″us-to′lik) **pressure,** the pressure when the ventricles are relaxing. Blood pressures are reported in millimeters of mercury (mm Hg), with the systolic pressure written first—120/80 (120 over 80) translates to a systolic pressure of 120 mm Hg and a diastolic pressure of 80 mm Hg. Most often, systemic arterial blood pressure is measured indirectly by the **auscultatory** (os-kul′tuh-tor-e) **method.** This procedure, as used to measure blood pressure in the brachial artery of the arm, is illustrated and described in **Figure 11.21**.

Effects of Various Factors on Blood Pressure Arterial blood pressure (BP) is directly related to cardiac output (CO; the amount of blood pumped out of



(a) The course of the brachial artery of the arm. Assume a blood pressure of 120/70 in a young, healthy person.

(b) The blood pressure cuff is wrapped snugly around the arm just above the elbow and inflated until the cuff pressure exceeds the systolic blood pressure. At this point, blood flow into the arm is stopped, and a brachial pulse cannot be felt or heard.

(c) The pressure in the cuff is gradually reduced while the examiner listens (auscultates) for sounds in the brachial artery with a stethoscope. The pressure read as the first soft tapping sounds are heard (the first point at which a small amount of blood is spurting through the constricted artery) is recorded as the systolic pressure.

(d) As the pressure is reduced still further, the sounds become louder and more distinct; when the artery is no longer constricted and blood flows freely, the sounds can no longer be heard. The pressure at which the sounds disappear is recorded as the diastolic pressure.

Figure 11.21 **Measuring blood pressure.**

the left ventricle per minute) and peripheral resistance (PR). This relationship is expressed by the equation BP = CO × PR. We have already considered regulation of cardiac output, so we will concentrate on peripheral resistance here.

Peripheral resistance is the amount of friction the blood encounters as it flows through the blood vessels. Many factors increase peripheral resistance, but probably the most important is the constriction, or narrowing, of blood vessels, especially arterioles, as a result of either sympathetic nervous system activity or atherosclerosis. Increased blood volume or increased blood viscosity (thickness) also raises peripheral resistance. Any factor that increases either cardiac output or peripheral resistance causes an almost immediate reflex rise in blood pressure. Many factors can alter blood pressure—age, weight, time of day, exercise, body position, emotional state, and various drugs, to name a few. The influence of a few of these factors is discussed next.

1. **Neural factors: the autonomic nervous system.** The parasympathetic division of the autonomic nervous system has little or no effect on blood pressure, but the sympathetic division is important. The major action of the sympathetic nerves on the vascular system is to cause **vasoconstriction** (vas″o-kon-strik′shun), or narrowing of the blood vessels, which increases the blood pressure. The sympathetic center in the medulla of the brain is activated to cause vasoconstriction in many different

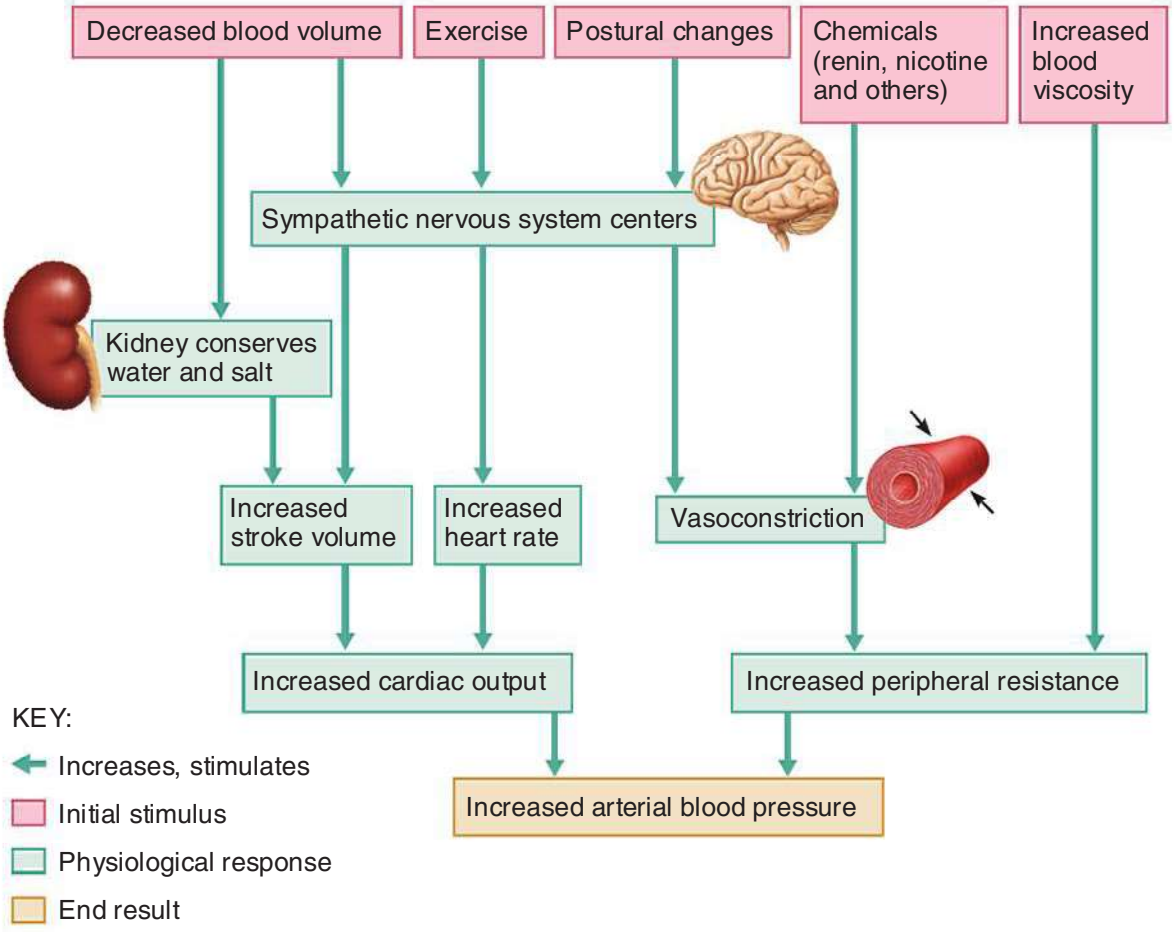


Figure 11.22 **Summary of factors that increase arterial blood pressure.**

circumstances (**Figure 11.22**). For example, when we stand up suddenly after lying down, the effect of gravity causes blood to pool in the vessels of the legs and feet, and blood pressure drops. This activates *pressoreceptors* (also called *baroreceptors*) in the large arteries of the neck and chest. They send off warning signals that result in reflexive vasoconstriction, increasing blood pressure back to homeostatic levels.

When blood volume suddenly decreases, as in hemorrhage, blood pressure drops, and the heart begins to beat more rapidly as it tries to compensate. However, because blood loss reduces venous return, the heart also beats weakly and inefficiently. In such cases, the sympathetic nervous system causes vasoconstriction to increase the blood pressure so that (hopefully) venous return increases and circulation can continue.

The final example concerns sympathetic nervous system activity when we exercise vigorously or are frightened and have to make a hasty escape. Under these conditions, there is a generalized vasoconstriction *except* in the skeletal muscles. The vessels of the skeletal muscles dilate to increase the blood flow to the working muscles. (It should be noted that the sympathetic nerves *never* cause vasoconstriction of blood vessels of the heart or brain.)

2. **Renal factors: the kidneys.** The kidneys play a major role in regulating arterial blood pressure by altering blood volume. As blood pressure (and/or blood volume) increases beyond normal, the kidneys allow more water to leave the body in the urine. Because the source of this water is the bloodstream, blood volume decreases, which in turn decreases blood pressure. However, when arterial blood pressure falls, the kidneys retain body water, increasing blood volume, and blood pressure rises (see Figure 11.22).

In addition, when arterial blood pressure is low, certain kidney cells release the enzyme *renin* into the blood. Renin triggers a series of

chemical reactions that result in the formation of *angiotensin II,* a potent vasoconstrictor chemical. Angiotensin also stimulates the adrenal cortex to release aldosterone, a hormone that enhances sodium ion reabsorption by the kidneys. As sodium moves into the blood, water follows. Thus, blood volume and blood pressure both rise.

3. **Temperature.** In general, cold has a *vasoconstricting* effect. This is why your exposed skin feels cold to the touch on a winter day and why cold compresses are recommended to prevent swelling of a bruised area. Heat has a *vasodilating effect,* and warm compresses are used to speed the circulation into an inflamed area.
4. **Chemicals.** The effects of chemical substances, many of which are drugs, on blood pressure are widespread and well known in many cases. We will give just a few examples here. **Epinephrine** increases both heart rate and blood pressure. *Nicotine* increases blood pressure by causing vasoconstriction. Both *alcohol* and *histamine* cause vasodilation and decrease blood pressure. The reason a person who has "one too many" becomes flushed is that alcohol dilates the skin vessels.
5. **Diet.** Although medical opinions tend to change and are at odds from time to time, it is generally believed that a diet low in salt, saturated fats, and cholesterol helps to prevent *hypertension,* or high blood pressure.

DID YOU GET IT?

22. What is the effect of hemorrhage on blood pressure? Why?

For the answer, see Appendix D.

Variations in Blood Pressure In normal adults at rest, systolic blood pressure varies between 110 and 140 mm Hg, and diastolic pressure between 70 and 80 mm Hg—but blood pressure varies considerably from one person to another and cycles over a 24-hour period, peaking in the morning. What is normal for you may not be normal for your grandfather or your neighbor. Blood pressure varies with age, weight, race, mood, physical activity, and posture. Nearly all these variations can be explained in terms of the factors affecting blood pressure that we have already discussed.

Hypotension, or low blood pressure, is generally considered to be a systolic blood pressure below 100 mm Hg. In many cases, it simply reflects individual differences and is no cause for concern. In fact, low blood pressure is an expected result of physical conditioning and is often associated with long life and an old age free of illness.

HOMEOSTATIC IMBALANCE

Elderly people may experience temporary low blood pressure and dizziness when they rise suddenly from a reclining or sitting position—a condition called **orthostatic hypotension.** Because an aging sympathetic nervous system reacts more slowly to postural changes, blood pools briefly in the lower limbs, reducing blood pressure and, consequently, blood delivery to the brain. Making postural changes more slowly to give the nervous system time to make the necessary adjustments usually prevents this problem.

Chronic hypotension (not explained by physical conditioning) may hint at poor nutrition and inadequate levels of blood proteins. Because blood viscosity is low, blood pressure is also lower than normal. Acute hypotension is one of the most important warnings of **circulatory shock,** a condition in which the blood vessels are inadequately filled and blood cannot circulate normally. The most common cause is blood loss.

A brief elevation in blood pressure is a normal response to fever, physical exertion, and emotional upset, such as anger and fear. Persistent **hypertension,** or **high blood pressure,** is pathological and is defined as a condition of sustained elevated arterial pressure of 140/90 or higher.

HOMEOSTATIC IMBALANCE

Chronic hypertension is a common and dangerous disease that warns of increased peripheral resistance. Although it progresses without symptoms for the first 10 to 20 years, it slowly and surely strains the heart and damages the arteries. For this reason, hypertension is often called the "silent killer." Because the heart is forced to pump against increased resistance, it must work harder, and in time, the myocardium enlarges. When finally strained beyond its capacity to respond, the heart weakens and its walls become flabby. Hypertension also ravages blood vessels, causing small tears in the endothelium that accelerate the progress of atherosclerosis (the early stage of arteriosclerosis).

(Continues on page 390)

A Closer Look

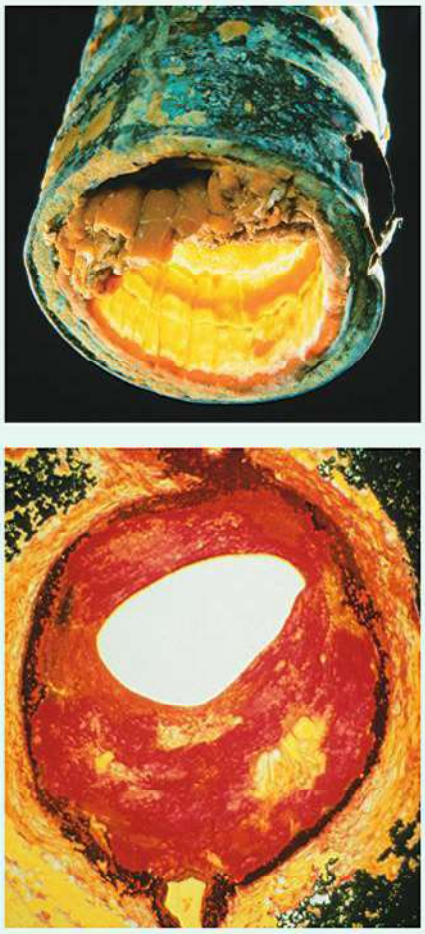
Atherosclerosis? Get Out the Cardiovascular Drāno!

When pipes get clogged, it is usually because something that shouldn't be there gets stuck in them—a greasy mass or a hair ball (see top photo). But when arteries are narrowed by **atherosclerosis,** the damming-up process occurs from the inside out: the walls of the vessels thicken and then protrude into the vessel lumen. Once this happens, it does not take much to close the vessel completely. A roaming blood clot or arterial spasms can do it.

Although all blood vessels are susceptible to atherosclerosis, for some unknown reason the aorta and the coronary arteries serving the heart are most often affected. The disease progresses through many stages before the arterial walls actually become hard and approach the stage of the rigid tube system described in the text, but some of the earlier stages are just as lethal.

Onset and Stages of Atherosclerosis

What triggers this scourge of blood vessels that indirectly causes half of the deaths in the Western world? According to the *response to injury hypothesis,* the initial event is damage to the tunica intima caused by bloodborne chemicals such as carbon monoxide (present in cigarette smoke or auto exhaust); by bacteria or viruses; or by physical factors such as a blow or persistent hypertension. Once a break has occurred, blood platelets cling to the injured site and initiate clotting to prevent blood loss. The injured endothelium sets off the alarm, summoning the immune system and the inflammatory process to repair the damage. If it is a one-time injury, when it's over, it's over. But most plaques grow slowly, through a series of injuries that heal, only to be ruptured again and again. As the plaque grows, the injured endothelial cells release chemotactic agents and chemicals that increase the permeability of the endothelium to fats and cholesterol, allowing them to take up residence just deep to the tunica intima. Monocytes attracted to the area migrate beneath the endothelium, where they become macrophages that gorge themselves on the fat in particular. These cells can become so engorged with oxidized fats that they are transformed into lipid-laden "foam cells" that lose their ability to act as scavengers. Soon they are joined by smooth muscle cells migrating from the tunica media of the blood vessel wall. These cells deposit collagen and elastin fibers in the area and also take in fat, becoming foam cells. The result is the erroneously named **fatty streak stage,** characterized by thickening of the tunica intima by greasy gray to yellow lesions called **fibrous** or **atherosclerotic plaques.** When these small, fatty mounds of muscle begin to protrude into the vessel wall (and ultimately the vessel lumen), the condition is called *atherosclerosis* (see bottom photo).



Top A pipe clogged by accumulated deposits. **Bottom** Atherosclerotic plaques nearly close a human artery.

Arteriosclerosis is the end stage of the disease. As enlarging plaques hinder diffusion of nutrients from the blood to the deeper tissues of the

artery wall, smooth muscle cells in the tunica media die and the elastic fibers deteriorate and are gradually replaced by nonelastic scar tissue. Then, calcium salts are deposited in the lesions, forming **complicated plaques.** Collectively, these events cause the arterial wall to fray and ulcerate, conditions that encourage thrombus formation. The increased rigidity of the vessels leads to hypertension. Together, these events increase the risk of myocardial infarctions, strokes, and aneurysms.

However, the popular view that most heart attacks are the consequence of severe vessel narrowing and hardening is now being challenged, particularly since some 70 percent of heart attacks are caused by much smaller obstructions, too small to be seen on an arteriogram or to cause any symptoms in most cases. It now appears that the body's defense system betrays it. The inflammatory process that occurs in the still soft, unstable, cholesterol-rich plaques changes the biology of the vessel wall and makes the plaques susceptible to rupture, exploding off fragments that trigger massive clots that can cause lethal heart attacks. The victim appears perfectly healthy until he or she drops dead!

Treatment and Prevention

The *vulnerable plaque hypothesis* described above has attracted many medical converts, but the question of what to do about it remains. Some medical centers test heart patients for elevated levels of cholesterol and C-reactive protein, a marker of inflammation. Electron beam CT scans may be able to identify people at risk by detecting calcium deposits in their coronary arteries. Antibiotics and anti-inflammatory drugs are being tested as preventive measures, and *statins,* cholesterol-reducing drugs, show promise for reducing C-reactive protein levels. Even the humble aspirin is gaining new respect, and more cardiologists recommend that people at high risk take one baby aspirin (81 mg) daily.

So what can help when the damage is done and the heart is at risk because of atherosclerotic coronary vessels? In the past, the only choice has been coronary artery bypass surgery, in which vessels removed from the legs or thoracic cavity are implanted in the heart to restore circulation. More recently, devices threaded through blood vessels to obstructed sites have become part of the ammunition of cardiovascular medicine. *Balloon angioplasty* uses a catheter with a balloon packed into its tip. When the catheter reaches the blockage, the balloon is inflated, and the fatty mass is compressed against the vessel wall. However, this procedure is useful to clear only very localized obstructions. A newer catheter device uses a laser beam to vaporize the arterial clogs. Although these intravascular devices are faster, cheaper, and less risky than bypass surgery, they carry with them the same major shortcoming: they do nothing to stop the underlying disease, and in time new blockages occur in 30 to 50 percent of cases. Sometimes after angioplasty, a metal-mesh tube called a stent is placed in the artery to keep it open.

When a blood clot is trapped by the diseased vessel walls, the answer may be a *clot-dissolving agent,* for example, *tissue plasminogen activator (tPA),* a naturally occurring substance now being produced by genetic engineering techniques. Injecting tPA directly into the heart restores blood flow quickly and puts an early end to many heart attacks in progress.

There is little doubt that lifestyle factors—emotional stress, smoking, obesity, high-fat and high-cholesterol diets, and lack of exercise—contribute to both atherosclerosis and hypertension. If these are the risk factors (and indeed they are), then why not just have patients at risk change their lifestyle? This is not as easy as it seems. Although taking antioxidants (E and C vitamins and beta carotene) and exercising more may "undo" some of the damage, old habits die hard, and North Americans like their burgers and butter. Can atherosclerosis be reversed to give the heart a longer and healthier life? If so, many more people with diseased arteries may be more willing to trade lifelong habits for a healthy old age!

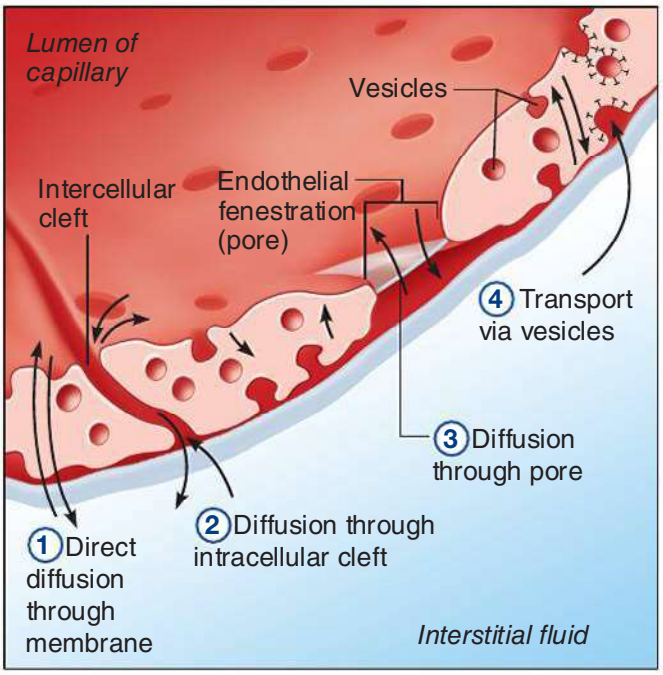


Figure 11.23 Capillary transport mechanisms. The four possible pathways or routes of transport across the wall of an endothelial cell of a capillary. (The endothelial cell is illustrated as if cut in cross section.)

Although hypertension and atherosclerosis are often linked, it is difficult to blame hypertension on any distinct anatomical pathology. In fact, about 90 percent of hypertensive people have **primary,** or **essential, hypertension,** which cannot be attributed to any specific organic cause. However, factors such as diet, obesity, heredity, race, and stress appear to be involved. For instance, more women than men and more blacks than whites are hypertensive. Hypertension runs in families. The child of a hypertensive parent is twice as likely to develop high blood pressure as is a child of parents with normal blood pressure. High blood pressure is common in obese people because the total length of their blood vessels is relatively greater than that in thinner individuals. For each pound of fat, miles of additional blood vessels are required, making the heart work harder to pump blood over longer distances.

Capillary Exchange of Gases and Nutrients

✓ Describe the exchanges that occur across capillary walls.

Capillaries form an intricate network among the body's cells and no substance has to diffuse very far to enter or leave a cell. The substances to be exchanged diffuse through an intervening space filled with **interstitial fluid (tissue fluid).**

Substances tend to move to and from body cells according to their concentration gradients. Thus, oxygen and nutrients leave the blood and move into the tissue cells, and carbon dioxide and other wastes exit the tissue cells and enter the blood. Basically, substances entering or leaving the blood may take one of four routes across the plasma membranes of the single layer of endothelial cells forming the capillary wall (**Figure 11.23**).

1. **Direct diffusion through membrane.** As with all cells, substances can diffuse directly through (cross) their plasma membranes if the substances are lipid-soluble (like the respiratory gases).
2. **Diffusion through intercellular clefts.** Limited passage of fluid and small solutes is allowed by **intercellular clefts** (gaps or areas of plasma membrane not joined by tight junctions). It is safe to say that, with the exception of brain capillaries—which are entirely secured together by tight junctions (the basis of the blood-brain barrier described in Chapter 7)—most of our capillaries have intercellular clefts.
3. **Diffusion through pores.** Very free passage of small solutes and fluids is allowed by **fenestrated capillaries.** These unique capillaries are found where absorption is a priority (intestinal capillaries or capillaries serving endocrine glands) or where filtration occurs (the kidney). A fenestra is an oval pore (*fenestra* = window) or opening and is usually covered by a delicate membrane (see Figure 11.23). Even so, a fenestra is much more permeable than other regions of the plasma membrane.
4. **Transport via vesicles.** Certain lipid-insoluble substances may enter or leave the blood and/or pass through the plasma membranes of endothelial cells within vesicles, that is, by endocytosis or exocytosis.

Only substances unable to pass by one of these routes are prevented from leaving (or entering) the capillaries. These include protein molecules (in plasma or interstitial fluid) and blood cells.

Fluid Movements at Capillary Beds

Besides the exchanges made via passive diffusion through capillary endothelial cell plasma

membranes, clefts, or fenestrations, and via vesicles there are active forces operating at capillary beds. Because of their intercellular clefts and fenestrations, some capillaries are leaky, and bulk fluid flows occur across their plasma membranes. Hence, blood pressure tends to force fluid (and solutes) out of the capillaries, and osmotic pressure tends to draw fluid into them because blood has a higher solute concentration (due to its plasma proteins) than does interstitial fluid. Whether fluid moves out of or into a capillary depends on the difference between the two pressures. As a rule, blood pressure is higher than osmotic pressure at the arterial end of the capillary bed, and lower than osmotic pressure at the venous end. Consequently, fluid moves out of the capillaries at the beginning of the bed and is reclaimed at the opposite (venule) end (**Figure 11.24**). However, not quite all of the fluid forced out of the blood is reclaimed at the venule end. Returning that lost fluid to the blood is the chore of the lymphatic system, discussed in Chapter 12.

DO YOU GET IT?

23. Would you expect fluid to be entering or leaving the capillaries at the venous end of a capillary bed?

For the answer, see Appendix D.

Developmental Aspects of the Cardiovascular System

- ✓ Briefly describe the development of the cardiovascular system.
- ✓ Name the fetal vascular modifications, or "fetal shunts," and describe their function before birth.
- ✓ Describe changes in the cardiovascular system with aging and list several factors that help maintain cardiovascular health.

The heart begins as a simple tube in the embryo. It is beating and busily pumping blood by the fourth week of pregnancy. During the next three weeks, the heart continues to change and mature, finally becoming a four-chambered structure capable of acting as a double pump—all without missing a beat! During fetal life, the collapsed lungs and nonfunctional liver are mostly bypassed by the blood, through special vascular shunts. After the seventh week of development, few changes other than growth occur in the fetal circulation

Q: *Assume there is a bacterial infection in the interstitial fluid. How would this affect fluid flows across the capillary walls in the area?*

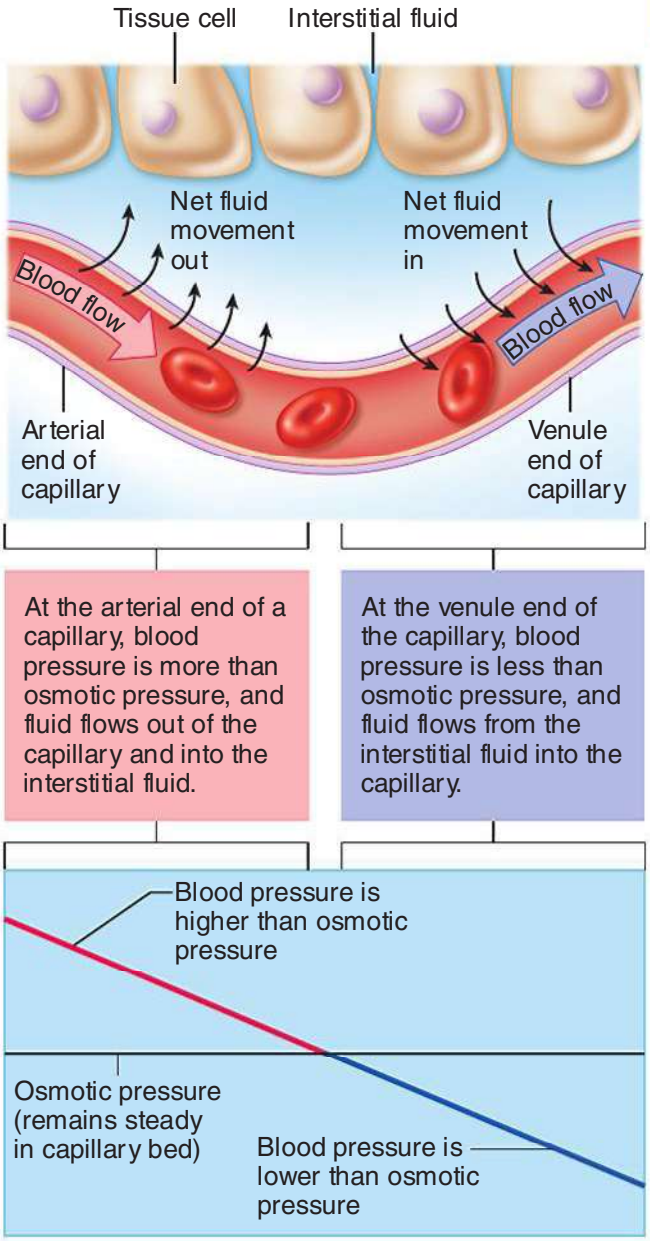
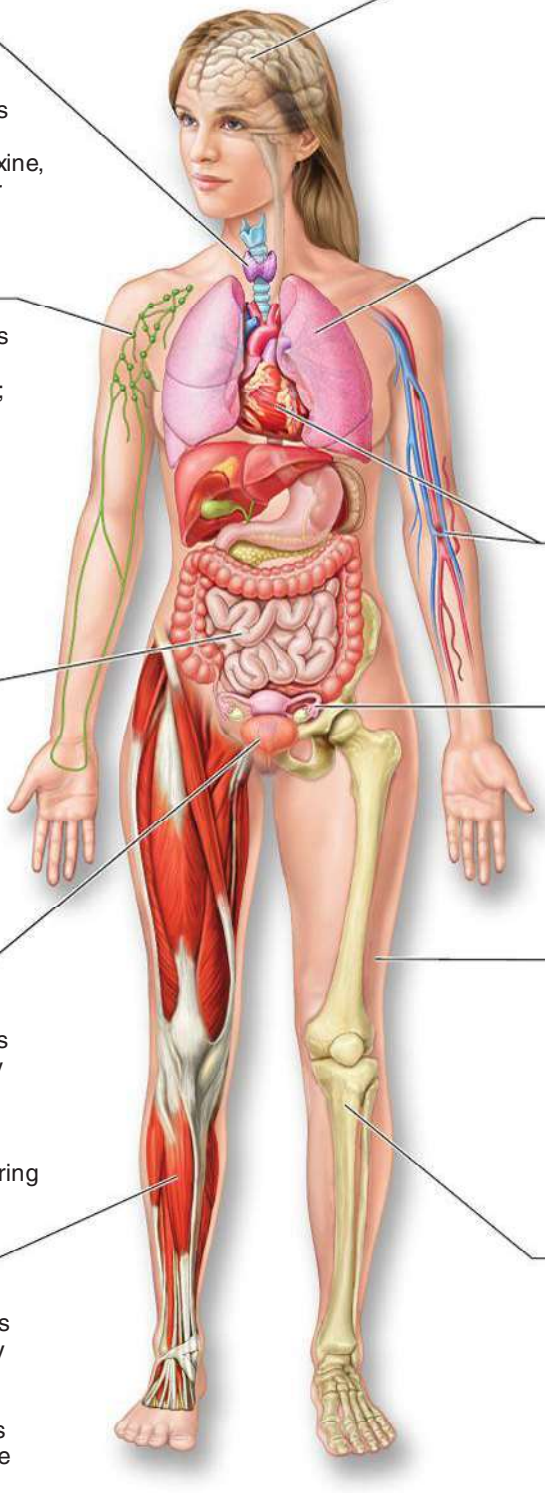


Figure 11.24 **Bulk fluid flows across capillary walls depend largely on the difference between the blood pressure and the osmotic pressure at different regions of the capillary bed.**

A: *It would increase it because the osmotic pressure of the interstitial fluid would rise as inflammatory molecules and debris accumulated in the area.*

SYSTEMS IN SYNC

Homeostatic Relationships between the **Cardiovascular System** and Other Body Systems



Cardiovascular System

Endocrine System

- The cardiovascular system delivers oxygen and nutrients; carries away wastes; blood serves as a transport vehicle for hormones
- Several hormones influence blood pressure (epinephrine, ANP, thyroxine, ADH); estrogen maintains vascular health in women

Lymphatic System/Immunity

- The cardiovascular system delivers oxygen and nutrients to lymphatic organs, which house immune cells; transports lymphocytes and antibodies; carries away wastes
- The lymphatic system picks up leaked fluid and plasma proteins and returns them to the cardiovascular system; its immune cells protect cardiovascular organs from specific pathogens

Digestive System

- The cardiovascular system delivers oxygen and nutrients; carries away wastes
- The digestive system provides nutrients to the blood including iron and B vitamins essential for RBC (and hemoglobin) formation

Urinary System

- The cardiovascular system delivers oxygen and nutrients; carries away wastes; blood pressure maintains kidney function
- The urinary system helps regulate blood volume and pressure by altering urine volume and releasing renin

Muscular System

- The cardiovascular system delivers oxygen and nutrients; carries away wastes
- Aerobic exercise enhances cardiovascular efficiency and helps prevent arteriosclerosis; the muscle "pump" aids venous return

Nervous System

- The cardiovascular system delivers oxygen and nutrients; removes wastes
- ANS regulates cardiac rate and force; sympathetic division maintains blood pressure and controls blood distribution according to need

Respiratory System

- The cardiovascular system delivers oxygen and nutrients; carries away wastes
- The respiratory system carries out gas exchange: loads oxygen and unloads carbon dioxide from the blood; respiratory "pump" aids venous return

Reproductive System

- The cardiovascular system delivers oxygen and nutrients; carries away wastes
- Estrogen maintains vascular health in women

Integumentary System

- The cardiovascular system delivers oxygen and nutrients; carries away wastes
- The skin's blood vessels provide an important blood reservoir and a site for heat loss from body

Skeletal System

- The cardiovascular system delivers oxygen and nutrients and carries away wastes
- Bones are the site of hematopoiesis; protect cardiovascular organs by enclosure; provide a calcium depot

until birth. Shortly after birth, the bypass structures become blocked, and the special umbilical vessels stop functioning.

HOMEOSTATIC IMBALANCE

Congenital heart defects account for about half of infant deaths resulting from all congenital defects. Environmental interferences, such as maternal infection and ingested drugs during the first three months of pregnancy (when the embryonic heart is forming), seem to be the major causes of such problems. Congenital heart defects may include a ductus arteriosus that does not close, septal openings, and other structural abnormalities of the heart. Such problems can usually be corrected surgically.

In the absence of congenital heart problems, the heart usually functions smoothly throughout a long lifetime for most people. Homeostatic mechanisms are so effective that we rarely are aware of when the heart is working harder. The heart will hypertrophy and its cardiac output will increase substantially if we exercise regularly and aerobically (that is, vigorously enough to force it to beat at a higher-than-normal rate for extended periods of time). The heart becomes not only a more powerful pump but also a more efficient one: pulse rate and blood pressure decrease. An added benefit of aerobic exercise is that it clears fatty deposits from the blood vessel walls, helping to slow the progress of atherosclerosis. However, let's raise a caution flag here: The once-a-month or once-a-year tennis player or snow shoveler has not built up this type of heart endurance and strength. When such an individual pushes his or her heart too much, it may not be able to cope with the sudden demand. This is why many weekend athletes are myocardial infarction victims.

As we get older, more and more signs of cardiovascular system disturbances start to appear. In some, the venous valves weaken, and purple, snakelike varicose veins appear. Not everyone has varicose veins, but we all have progressive atherosclerosis. Some say the process begins at birth, and there's an old saying that goes, "You are only as old as your arteries," referring to this degenerative process. The gradual loss in elasticity in the blood vessels leads to hypertension and hypertensive heart disease. The insidious filling of the blood vessels with fatty, calcified deposits leads most commonly to **coronary artery disease.** Also, as described in Chapter 10, the roughening of the vessel walls encourages thrombus formation. At least 30 percent of the population in the United States has hypertension by the age of 50, and cardiovascular disease causes more than one-half of the deaths in people over age 65. Although the aging process itself contributes to changes in the walls of the blood vessels that can lead to strokes or myocardial infarctions, most researchers feel that diet, not aging, is the single most important contributing factor to cardiovascular diseases. There is some agreement that the risk is lowered if people eat less animal fat, cholesterol, and salt. Other recommendations include avoiding stress, eliminating cigarette smoking, and taking part in a regular, moderate exercise program.

Summary

Access more review material and fun learning activities online—visit **www.anatomyandphysiology.com** and select Essentials of Human Anatomy & Physiology, 10th edition. In addition, references to Interactive Physiology are included below.

iP = Interactive Physiology

The Heart (pp. 357–370)

1. The heart, located in the thorax, is flanked laterally by the lungs and enclosed in a pericardium.
2. The bulk of the heart (myocardium) is composed of cardiac muscle. The heart has four hollow chambers—two atria (receiving chambers) and two ventricles (discharging chambers), each lined with endocardium. The heart is divided longitudinally by a septum.

   iP Cardiovascular System Topic: Anatomy Review: The Heart, pp. 3–5.
3. The heart functions as a double pump. The right heart is the pulmonary pump (right heart to lungs to left heart). The left heart is the systemic pump (left heart to body tissues to right heart).