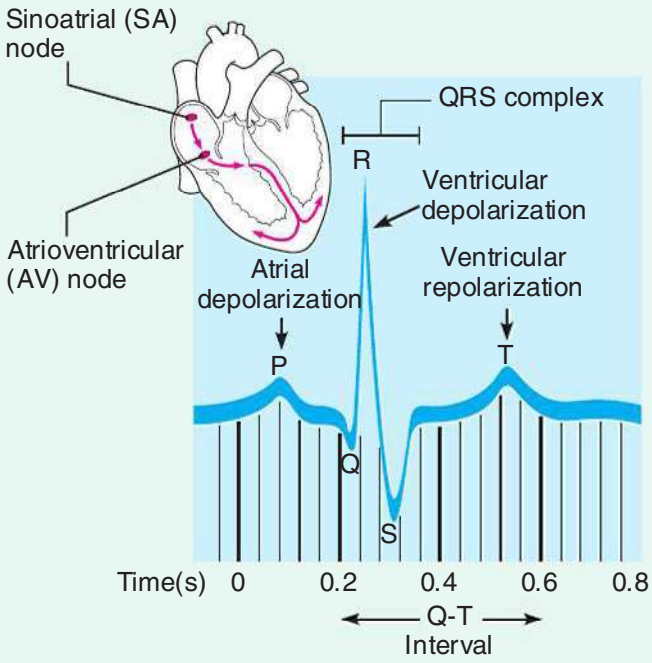
A Closer Look

Electrocardiography: (Don't) Be Still My Heart

When impulses pass through the heart, electrical currents are generated that spread throughout the body. These impulses can be detected on the body surface and recorded with an *electrocardiograph.* The recording that is made, the **electrocardiogram (ECG),** traces the flow of current through the heart. The illustration shows a normal ECG tracing.

The typical ECG has three recognizable waves. The first wave, the **P wave,** is small and signals the depolarization of the atria immediately before they contract. The large **QRS complex,** which results from the depolarization of the ventricles, has a complicated shape. It precedes the contraction of the ventricles. The **T wave** results from currents flowing during the repolarization of the ventricles. (Atrial repolarization is generally hidden by the large QRS complex, which is being recorded at the same time.)

Abnormalities in the shape of the waves and changes in their timing send signals that something may be wrong with the intrinsic conduction system, or they may indicate a *myocardial infarct* (present or past). A myocardial infarct is an area of heart tissue in which the cardiac cells have died; it is generally a result of *ischemia.* During *fibrillation,* the normal pattern of the ECG is totally lost, and the heart ceases to act as a functioning pump.



An electrocardiogram tracing showing the three normally recognizable deflection waves—P, QRS, and T.

Cardiac Output

✓ Describe the effect of each of the following on heart rate: stimulation by the vagus nerve, exercise, epinephrine, and various ions.

Cardiac output (CO) is the amount of blood pumped out by *each* side of the heart (actually each ventricle) in 1 minute. It is the product of the **heart rate (HR)** and the **stroke volume (SV).** Stroke volume is the volume of blood pumped out by a ventricle with each heartbeat. In general, stroke volume increases as the force of ventricular contraction increases. If we use the normal resting

values for heart rate (75 beats per minute) and stroke volume (70 ml per beat), the average adult cardiac output can be easily figured:

CO = HR (75 beats/min) × SV (70 ml/beat)
CO = 5250 ml/min

The normal adult blood volume is about 6000 ml, so the entire blood supply passes through the body once each minute. Cardiac output varies with the demands of the body. It rises when the stroke volume is increased or the heart beats faster or both; it drops when either or both of these factors decrease. Let's take a look at how stroke volume and heart rate are regulated.

Regulation of Stroke Volume A healthy heart pumps out about 60 percent of the blood present in its ventricles. As noted above, this is approximately 70 ml (about 2 ounces) with each heartbeat. According to *Starling's law of the heart,* the critical factor controlling stroke volume is how much the cardiac muscle cells are stretched just before they contract. The more they are stretched, the stronger the contraction will be. The important factor stretching the heart muscle is *venous return,* the amount of blood entering the heart and distending its ventricles. If one side of the heart suddenly begins to pump more blood than the other, the increased venous return to the opposite ventricle will force it to pump out an equal amount, thus preventing backup of blood in the circulation.

Anything that increases the volume or speed of venous return also increases stroke volume and force of contraction (**Figure 11.9**). For example, a slow heartbeat allows more time for the ventricles to fill. Exercise speeds venous return because it results in increased heart rate and force. The enhanced squeezing action of active skeletal muscles on the veins returning blood to the heart, the so-called *muscular pump,* also plays a major role in increasing the venous return. In contrast, low venous return, such as might result from severe blood loss or an extremely rapid heart rate, decreases stroke volume, causing the heart to beat less forcefully.

Factors Modifying Basic Heart Rate In healthy people, stroke volume tends to be relatively constant. However, when blood volume drops suddenly or when the heart has been seriously weakened, stroke volume declines, and cardiac output is maintained by a faster heartbeat. Although heart contraction does not depend on the nervous system, its rate *can* be changed temporarily by the autonomic nerves. Indeed, the most important external influence on heart rate is the activity of the autonomic nervous system. Heart rate is also modified by various chemicals, hormones, and ions. Some of these factors are summarized in Figure 11.9, and several are discussed next.

1. **Neural (ANS) controls.** During times of physical or emotional stress, the nerves of the *sympathetic division* of the autonomic nervous system more strongly stimulate the SA and AV nodes and the cardiac muscle itself. As a result, the heart beats more rapidly. This is a familiar phenomenon to anyone who has ever been frightened or has had to run to catch a bus. As fast as the heart pumps under ordinary conditions, it really speeds up when special demands are placed on it. Because a faster blood flow increases the rate at which fresh blood reaches body cells, more oxygen and glucose are made available to them during periods of stress. When demand declines, the heart adjusts. *Parasympathetic nerves,* primarily the vagus nerves, slow and steady the heart, giving it more time to rest during noncrisis times. In patients with *congestive heart failure,* a condition in which the heart is nearly "worn out" because of age, hypertensive heart disease, or another pathological process, the heart pumps weakly. For those patients, the drug digitalis is routinely prescribed. It enhances contractile force and stroke volume of the heart, resulting in greater cardiac output.
2. **Hormones and ions.** Various hormones and ions can have a dramatic effect on heart activity. *Epinephrine,* which mimics the effect of the sympathetic nerves and is released in response to sympathetic nerve stimulation, and *thyroxine* both increase heart rate. Electrolyte imbalances pose a real threat to the heart. For example, reduced levels of ionic calcium in the blood depress the heart, whereas excessive blood calcium causes such prolonged contractions that the heart may stop entirely. Either excess or lack of needed ions such as sodium and potassium also modifies

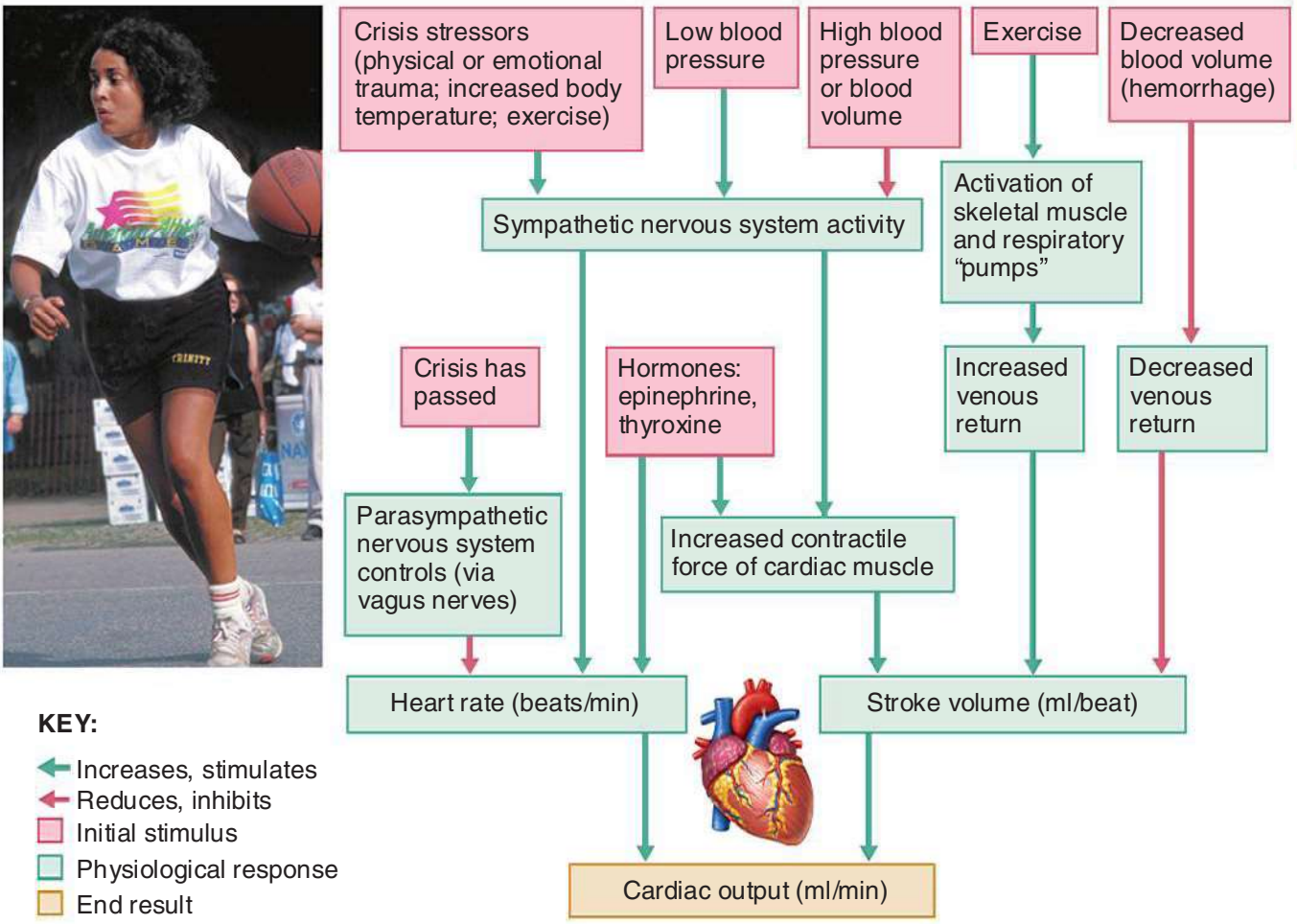


Figure 11.9 **Influence of selected factors on cardiac output.**

heart activity. A deficit of potassium ions in the blood, for example, causes the heart to beat feebly, and abnormal heart rhythms appear.

3. **Physical factors.** A number of physical factors, including age, gender, exercise, and body temperature, influence heart rate. Resting heart rate is fastest in the fetus (140–160 beats per minute) and then gradually decreases throughout life. The average heart rate is faster in females (72–80 beats per minute) than in males (64–72 beats per minute). Heat increases heart rate by boosting the metabolic rate of heart cells. This explains the rapid, pounding heartbeat you feel when you have a high fever and accounts in part for the effect of exercise on heart rate (remember, working muscles generate heat). Cold has the opposite effect; it directly decreases heart rate. As noted above, exercise acts through nervous system controls (sympathetic division) to increase heart rate (and also, through the action of the muscular pump, to increase stroke volume).

HOMEOSTATIC IMBALANCE

The pumping action of the healthy heart maintains a balance between cardiac output and venous return. But when the pumping efficiency of the heart is depressed so that circulation is inadequate to meet tissue needs, **congestive heart failure (CHF)** occurs. Congestive heart failure is usually a progressive condition that reflects weakening of the heart by *coronary atherosclerosis* (clogging of the coronary vessels with fatty buildup), persistent high blood pressure, or multiple myocardial infarctions (leading to repair with noncontracting scar tissue).

Because the heart is a double pump, each side can fail independently of the other. If the left heart fails, *pulmonary congestion* occurs. The right side of the heart continues to propel blood to the lungs, but the left side is unable to eject the returning blood into the systemic circulation. As blood vessels within the lungs become swollen with blood, the pressure within them increases, and fluid leaks from the circulation into the lung tissue, causing **pulmonary edema.** If untreated, the person suffocates.

If the right side of the heart fails, *peripheral congestion* occurs as blood backs up in the systemic circulation. Edema is most noticeable in the distal parts of the body: The feet, ankles, and fingers become swollen and puffy. Failure of one side of the heart puts a greater strain on the opposite side, and eventually the whole heart fails.

DID YOU GET IT?

9. What does the term *cardiac output* mean?

10. What would you expect to happen to the heart rate of an individual with a fever? Why?

11. What is the most important factor affecting stroke volume?

For answers, see Appendix D.

Blood Vessels

✓ Compare and contrast the structure and function of arteries, veins, and capillaries.

Blood circulates inside the blood vessels, which form a closed transport system, the so-called **vascular system.** The idea that blood circulates, or "makes rounds," through the body is only about 300 years old. The ancient Greeks believed that blood moved through the body like an ocean tide, first moving out from the heart and then ebbing back to it in the same vessels to get rid of its impurities in the lungs. It was not until the seventeenth century that William Harvey, an English physician, proved that blood did, in fact, move in circles.

Like a system of roads, the vascular system has its freeways, secondary roads, and alleys. As the heart beats, it propels blood into the large **arteries** leaving the heart. Blood then moves into successively smaller and smaller arteries and then into the **arterioles** (ar-ter′e-ōlz), which feed the **capillary** (kap′ĭ-lar″e) **beds** in the tissues. Capillary beds are drained by **venules** (ven′ulz), which in turn empty into **veins** that finally empty into the great veins (venae cavae) entering the heart. Thus arteries, which carry blood away from the heart, and veins, which drain the tissues and return the blood to the heart, are simply conducting vessels—the freeways and secondary roads. Only the tiny hairlike capillaries, which extend and branch through the tissues and connect the smallest arteries (arterioles) to the smallest veins (venules), directly serve the needs of the body cells. The capillaries are the side streets or alleys that intimately intertwine among the body cells and provide access to individual "homes." It is only through their walls that exchanges between the tissue cells and the blood can occur.

Notice that this book routinely depicts arteries in red and veins in blue because, by convention, red indicates oxygen-rich blood, the normal status of blood in most of the body's arteries, and blue indicates relatively oxygen-depleted, carbon dioxide–rich blood, the normal status of blood in most of the veins. However, there are exceptions to this convention. For instance, we have seen that oxygen-poor blood is carried in the pulmonary trunk, an artery, while oxygen-rich blood is transported back to the heart in pulmonary veins. An easy way to remember this difference is the following device: *arteries are red and veins are blue, but for the lungs there's an exception of two.* We will point out other exceptions as we encounter them.

Microscopic Anatomy of Blood Vessels

Tunics

Except for the microscopic capillaries, the walls of blood vessels have three coats, or tunics (**Figure 11.10**). The **tunica intima** (tu′nĭ-kah in-tim′ah), which lines the lumen, or interior, of the vessels, is a thin layer of endothelium (squamous epithelial cells) resting on a basement membrane. Its cells fit closely together and form a slick surface that decreases friction as blood flows through the vessel lumen.

The **tunica media** (me′de-ah) is the bulky middle coat. It is mostly smooth muscle and elastic fibers. Some of the larger arteries have *elastic*

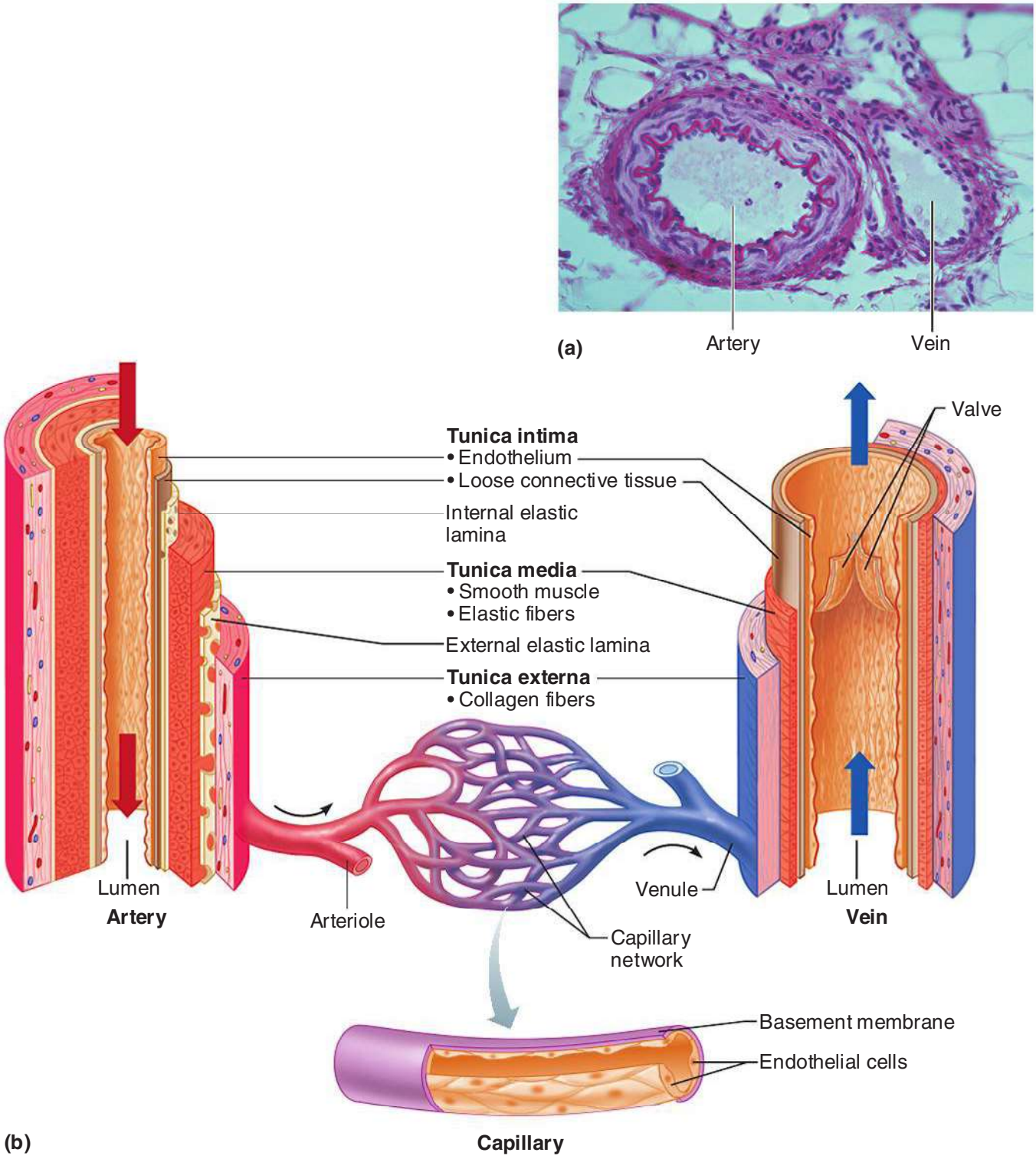


Figure 11.10 **Structure of blood vessels.** **(a)** Light photomicrograph of a muscular artery and the corresponding vein in cross section (140×). **(b)** The walls of arteries and veins are composed of three tunics: the tunica intima, tunica media, and tunica externa. Capillaries—between arteries and veins in the circulatory pathway—are composed only of the tunica intima. Notice that the tunica media is thick in arteries and relatively thin in veins.

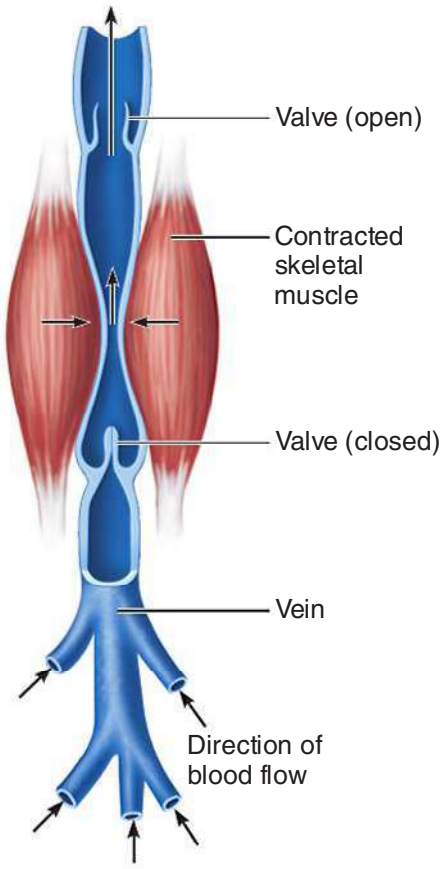


Figure 11.11 Operation of the muscular pump. When skeletal muscles contract and press against the flexible veins, the valves proximal to the area of contraction are forced open, and blood is squeezed toward the heart. The valves distal to the point of contraction are closed by the backflowing blood.

laminae, sheets of elastic tissue, in addition to the scattered elastic fibers. The smooth muscle, which is controlled by the sympathetic nervous system, is active in changing the diameter of the vessels. As the vessels constrict or dilate, blood pressure increases or decreases, respectively.

The **tunica externa** (eks′tern-ah) is the outermost tunic. This layer is composed largely of fibrous connective tissue, and its function is basically to support and protect the vessels.

Structural Differences in Arteries, Veins, and Capillaries

The walls of arteries are usually much thicker than those of veins. The arterial tunica media, in particular, tends to be much heavier. This structural difference is related to a difference in function of these two types of vessels. Arteries, which are closer to the pumping action of the heart, must be able to expand as blood is forced into them and then recoil passively as the blood flows off into the circulation during diastole. Their walls must be strong and stretchy enough to take these continuous changes in pressure (see Figure 11.20).

Veins, in contrast, are far from the heart in the circulatory pathway, and the pressure in them tends to be low all the time. Thus veins have thinner walls. However, because the blood pressure in veins is usually too low to force the blood back to the heart, and because blood returning to the heart often flows against gravity, veins are modified to ensure that the amount of blood returning to the heart (*venous return*) equals the amount being pumped out of the heart (*cardiac output*) at any time. The lumens of veins tend to be much larger than those of corresponding arteries, and the larger veins have **valves** that prevent backflow of blood (see Figure 11.10).

- To see the effect of venous valves, perform the following simple experiment on yourself: Allow one hand to hang by your side for a minute or two, until the blood vessels on its dorsal aspect become distended (swollen) with blood. Place two fingertips side by side against one of the distended veins. Then, pressing firmly, move your proximal finger along the vein toward your heart. Now release that finger. As you can see, the vein remains collapsed in spite of gravity. Now remove your distal finger, and watch the vein fill rapidly with blood.

Skeletal muscle activity also enhances venous return. As the muscles surrounding the veins contract and relax, the blood is squeezed, or "milked," through the veins toward the heart (**Figure 11.11**). Finally, when we inhale, the drop in pressure that occurs in the thorax causes the large veins near the heart to expand and fill. Thus, the "respiratory pump" also helps return blood to the heart (see Figure 11.9).

The transparent walls of the capillaries are only one cell layer thick—just the tunica intima. Because of this exceptional thinness, exchanges are easily made between the blood and the tissue cells. The tiny capillaries tend to form interweaving networks called *capillary beds.* The flow of blood from an arteriole to a venule—

that is, through a capillary bed—is called **microcirculation.** In most body regions, a capillary bed consists of two types of vessels: (1) a **vascular shunt,** a vessel that directly connects the arteriole and venule at opposite ends of the bed, and (2) true capillaries, the actual *exchange vessels* (**Figure 11.12**).

The *true capillaries* number 10 to 100 per capillary bed, depending on the organ or tissues served. They usually branch off the proximal end of the shunt and return to the distal end, but occasionally they spring from the **terminal arteriole** and empty directly into the **postcapillary venule.** A cuff of smooth muscle fibers, called a **precapillary sphincter,** surrounds the root of each true capillary and acts as a valve to regulate the flow of blood into the capillary. Blood flowing through a terminal arteriole may take one of two routes: through the true capillaries or through the shunt. When the precapillary sphincters are relaxed (open), blood flows through the true capillaries and takes part in exchanges with tissue cells. When the sphincters are contracted (closed), blood flows through the shunts and bypasses the tissue cells.

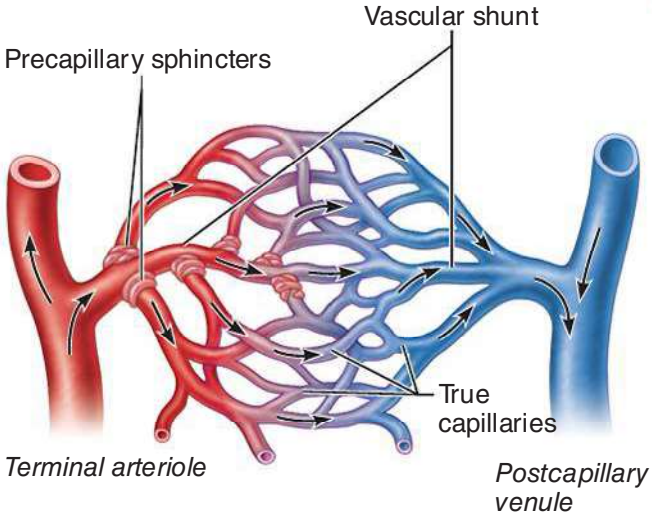
HOMEOSTATIC IMBALANCE

Varicose veins are common in people who stand for long periods of time (for example, dentists and hairdressers) and in obese (or pregnant) individuals. The common factors are the pooling of blood in the feet and legs and inefficient venous return resulting from inactivity or pressure on the veins. In any case, the overworked valves give way, and the veins become twisted and dilated. A serious complication of varicose veins is **thrombophlebitis** (throm″bo-fle-bi′tis), inflammation of a vein that results when a clot forms in a vessel with poor circulation. Because all venous blood must pass through the pulmonary circulation before traveling through the body tissues again, a common consequence of thrombophlebitis is clot detachment and **pulmonary embolism,** which is a life-threatening condition.

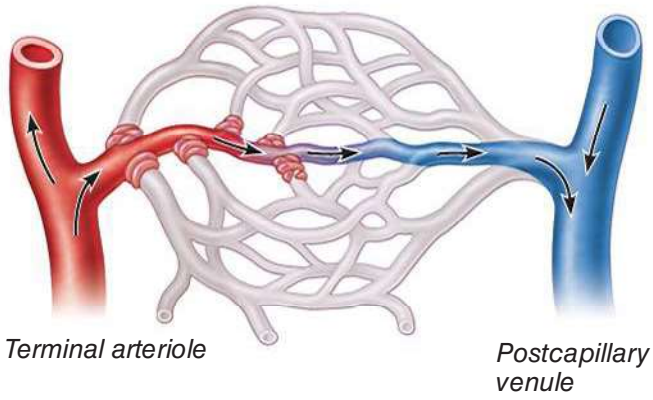
DID YOU GET IT?

12. Assume you are viewing a blood vessel under the microscope. It has a lopsided lumen, relatively thick externa, and a relatively thin media. Which kind of blood vessel is this?

Q: *Assume the capillary bed depicted here is in the biceps brachii muscle of your arm. What condition would the capillary bed be in (**a** or **b**) if you were doing push-ups at the gym?*



(a) Sphincters open; blood flows through true capillaries.



(b) Sphincters closed; blood flows through vascular shunt.

Figure 11.12 Anatomy of a capillary bed. The vascular shunt bypasses the true capillaries when precapillary sphincters controlling blood entry into the true capillaries are constricted.

13. Arteries lack valves, but veins have them. How is this structural difference related to blood pressure?

14. How is the structure of capillaries related to their function in the body?

For answers, see Appendix D.

A: ***a,*** *The true capillaries would be flushed with blood to serve the working muscle cells.*