of urine. Blood pressure is also used to stiffen erectile tissues in both sexes during sexual intercourse. In the arms, the circulatory system includes vascular heat exchangers that can short-circuit heat flux, helping reduce energy costs by limiting heat loss into chilly environments (see Figure 10.34).

Ancient people undoubtedly were as aware as we are that when the body is cut, it oozes blood regardless of where the cut is made. This observation was the first sign that blood at high pressure streams through every region of tissue. The idea that the blood makes a round-trip through the body—the concept that it circulates—was first put forth by William Harvey in 1628. However, Harvey and his contemporaries could not possibly have understood the circulation as we do today, because blood capillaries were unknown in his time, and O_2 was not identified as a defined chemical element for another 150 years. From Harvey's time to the present, scientists have learned steadily more about the tasks that are accomplished by the circulation of the blood.

The very word *circulation* has taken on a progressively different meaning as knowledge has expanded. For Harvey, *circulation* meant round-trip blood flow in major blood vessels. A generation after Harvey, early microscopists discovered that minute blood vessels just barely wider than red blood cells—the capillaries—weave among the cells of every tissue. *Circulation* then included tissue blood flow, and bleeding from every cut could finally be understood. In the nineteenth century, scientists demonstrated that the blood brings O_2 to cells (see Box Extension 7.1). In the twentieth century, hormones, antibodies, and immune cells were discovered, and the concept of *circulation* was expanded to include the transport of these agents from one region of the body to another.

To define **circulation** today in a way that is relevant to all kinds of animals, two perspectives can be taken. From the perspective of mechanics, circulation is the pressure-driven bulk flow of a body fluid called **blood** through a system of tubular vessels or other passages that brings the fluid to all parts of the body. The system of vessels or other blood passages—plus the blood itself—is called the **circulatory system**. When we think of circulation, however, we usually do not think of it in only these mechanical terms. From a second perspective, circulation is defined by what it accomplishes. Thus, for us today, circulation is a pressure-driven bulk flow of fluid that rapidly transports O_2, CO_2, nutrients, organic wastes, hormones, agents of the immune system, heat, and other commodities throughout the body and that often provides a source of hydraulic pressure for organ function.

The *speed* of transport by the circulation is one of its most central and defining attributes. As we first saw in discussing Table 5.1, diffusion through aqueous solutions is too slow to transport commodities at biologically significant rates over distances exceeding 1 mm or so. Only very small animals, therefore, can depend on diffusion as their sole means of internal transport (see Box 22.1). Convective transport—transport by bulk flow of body fluids—is intrinsically far faster than diffusion. Consequently, as stressed in Chapter 22 (see Figure 22.7), animals that are larger than 1 mm or so generally require blood circulation (or some other form of bulk flow of body fluids) to move commodities from place to place in their bodies at adequate rates.

As we study the circulation, both of the defining perspectives we have identified will be important. Looking back at either of the athletes in our opening photograph, for example, one of our two key questions must be how her circulatory system itself works: How do her heart and vascular system function to bring blood to and from the cells in her skeletal muscles and all the other parts of her body at the rate required, and how are these processes regulated? Our second key question must focus on consequences: What functions are accomplished by her circulation?

In humans and in most other types of animals, the transport of O_2 is by far the most pressing and urgent function performed by the circulation.[1] That is, of all the commodities that tissues require to be brought to them by the circulation of blood, O_2 is the one that, by far, the tissues can least afford to have brought more slowly. This observation has both evolutionary and immediate implications. Speaking of evolution, when we compare animal taxonomic groups, we in general see a clear positive correlation between *metabolic intensity* and the *peak ability of the circulatory system to transport O_2 rapidly*. These two properties have clearly coevolved over the course of evolutionary time. Similarly, in the day-to-day life of an individual animal, it is often true that tissue O_2 needs drive changes in blood-flow rates: The rate of blood flow rises and falls as the metabolic need for O_2 increases and decreases. These principles explain why O_2 transport often receives paramount attention in the study of circulatory systems, even though circulation is essential for a great many functions.

25.1 Hearts

A logical starting point for study of the circulation is the hearts of animals. A **heart** is a discrete, localized pumping structure. Some animals that have a circulatory system lack a heart; in many annelid worms, for example, the blood is propelled through the circulatory system entirely by peristaltic contractions of blood vessels. Hearts are very common in circulatory systems, however, and often assume principal responsibility for driving the flow of blood through the blood vessels.

In some types of animals, such as arthropods, the heart is **single-chambered**, consisting of a single muscular tube or sac. In others, such as vertebrates, the heart is composed of two or more compartments through which blood passes in sequence, and thus is **multichambered**. Many types of animals, in addition to their principal heart, possess other hearts that assist with the pumping of blood through localized parts of the body. Such secondary or local hearts are called **accessory hearts** or **auxiliary hearts**.

The muscle tissue of a heart, composed of *cardiac muscle*, is known as the **myocardium** (*myo*, "muscle"; *cardium*, "heart"). **Cardiac muscle**, one of the major types of muscle, typically has distinctive structural and physiological properties in comparison with other types of muscle (e.g., skeletal muscle). **BOX 25.1** highlights the distinctive properties of vertebrate cardiac muscle.

[1] Insects and other tracheate arthropods are dramatic exceptions to this statement, as discussed in **BOX 25.4**.

BOX 25.1 The Structure and Function of Vertebrate Cardiac Muscle

Vertebrate cardiac muscle has distinctive traits that suit it for its specialized role. Some of these traits are discussed fully in this chapter. Others are discussed in detail in Chapters 12, 13, and 20. Here we briefly note the properties that receive detailed treatment in the other chapters.

Structurally, cardiac muscle is a type of striated muscle, and in this respect it resembles skeletal muscle (see Table 20.3 in the main text). Cardiac muscle is structurally very different from skeletal muscle, however, in that adjacent muscle cells (muscle fibers) are joined by specialized *intercalated discs* (FIGURE A; see Section 20.7 in the main text) that impart important mechanical and electrical properties. Where two cells meet at an intercalated disc, they are joined together by strong mechanical adhesions, so that mechanical forces developed in one cell are transmitted to the other, helping achieve mutually reinforcing force generation. The two cells that meet at an intercalated disc are also joined to each other there by gap junctions (see Figures 2.7 and 13.2 in the main text), meaning that the cytoplasm of each cell is continuous with that of the other cell. At the gap junctions, an action potential (wave of cell-membrane depolarization) in one cell is transmitted electrically—and therefore very rapidly—to the other cell (see Section 13.1 in the main text). Thus when one cardiac muscle cell generates an action potential and contracts, adjacent cells quickly generate action potentials and contract almost synchronously. Because of the control of contraction in this way, all the cells in the wall of a heart chamber contract together.

Individual action potentials in cardiac muscle cells are long, drawn-out events by comparison with those in neurons (FIGURE B). During an action potential in a cardiac muscle cell, depolarization of the cell membrane—which is the immediately effective stimulus for contraction—lasts about 100–500 milliseconds (ms), whereas depolarization in neurons typically lasts less than 1 ms. The mechanism of the long depolarization is discussed in Chapter 12 (see Figure 12.22 in the main text). Its function is to ensure that contraction is prolonged, rather than being just a brief twitch. Cardiac muscle cells must contract for about 100–500 ms during each heartbeat for the heart to pump blood effectively.

The cellular process that initiates each heartbeat is also a property of the cardiac muscle. The pacemaker, which initiates each beat, is composed of specialized muscle cells. Although we say much about this topic in this chapter, the electrical properties of the cells that compose the pacemaker are discussed in Chapter 12 in the main text. As explained there (see the discussion of pacemaker potentials in Section 12.5), the membrane potential across the cell membrane in these cells does not stay at a stable resting value between beats. Instead, after a heartbeat, the membrane potential spontaneously drifts in the direction of ever-increasing depolarization. Because of this drift, the membrane potential eventually becomes depolarized enough to initiate a new action potential, which triggers the next heartbeat.

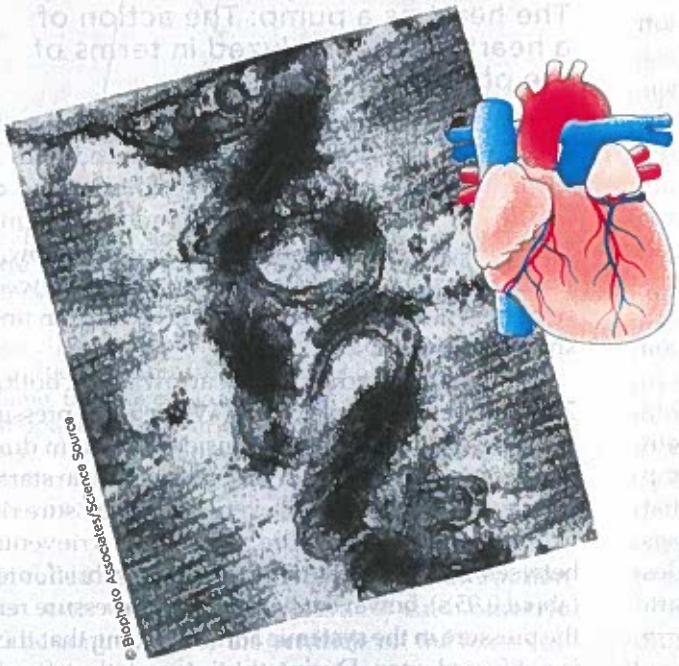


FIGURE A Human cardiac muscle cells with intercalated discs

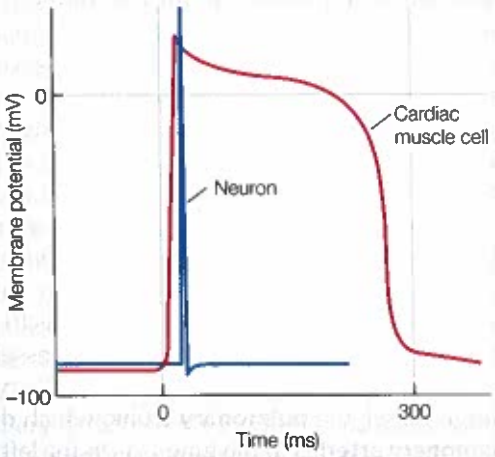


FIGURE B Action potentials An action potential occurs when, as time passes, the cell membrane transiently becomes less negative (a change referred to as a depolarization).

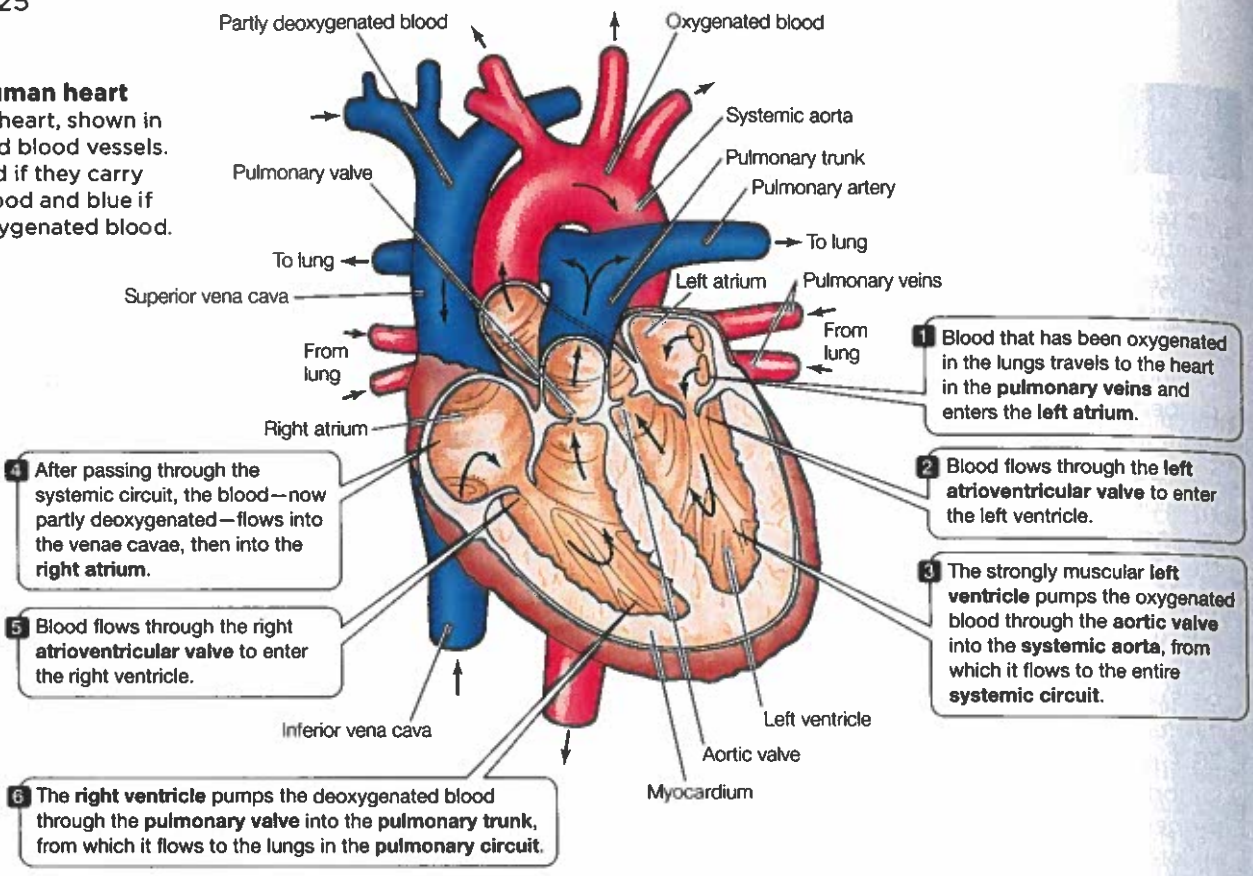


FIGURE 25.1 The human heart
A section through the heart, shown in relation to the attached blood vessels. Vessels are colored red if they carry freshly oxygenated blood and blue if they carry partly deoxygenated blood.

To study the morphology of a heart in detail, there is no more appropriate example than our own (FIGURE 25.1). The hearts of other mammals and of birds are similar. The left side of the human heart, which consists of two chambers—a weakly muscular **atrium** and a strongly muscular **ventricle**—receives freshly oxygenated blood from the lungs and pumps it to the systemic tissues of the body.[2] Blood arrives in the **left atrium** via the **pulmonary veins** that drain the lungs.[3] It leaves the **left ventricle** via a single massive artery, the **systemic aorta**, which branches to send arterial vessels to the head, arms, abdomen, and all other body regions, even the myocardium itself. Passive valves, consisting of flaps of connective tissue covered with endothelial tissue, are positioned between the atrium and ventricle (the **left atrioventricular valve**) and between the ventricle and aorta (the **aortic valve**); these valves allow blood to flow freely in the correct direction and prevent it from flowing backward. After blood leaves the systemic aorta, it passes through the **systemic circuit**—the blood vessels that take blood to and from the systemic tissues—and ultimately returns in the great collecting veins (**venae cavae**; singular *vena cava*) to the heart, where it enters the **right atrium** and then the **right ventricle**. The function of the right side of the heart is to pump blood through the **pulmonary circuit**—the blood vessels that take blood to and from the lungs. The right ventricle propels blood into a large vessel, the **pulmonary trunk**, which divides to form the **pulmonary arteries** to the lungs. As in the left heart, passive flap valves prevent backward flow in the right heart; these valves are positioned between the atrium and ventricle (the **right atrioventricular valve**) and between the ventricle and pulmonary trunk (the **pulmonary valve**). After blood has been oxygenated in the lungs, it returns to the left atrium.

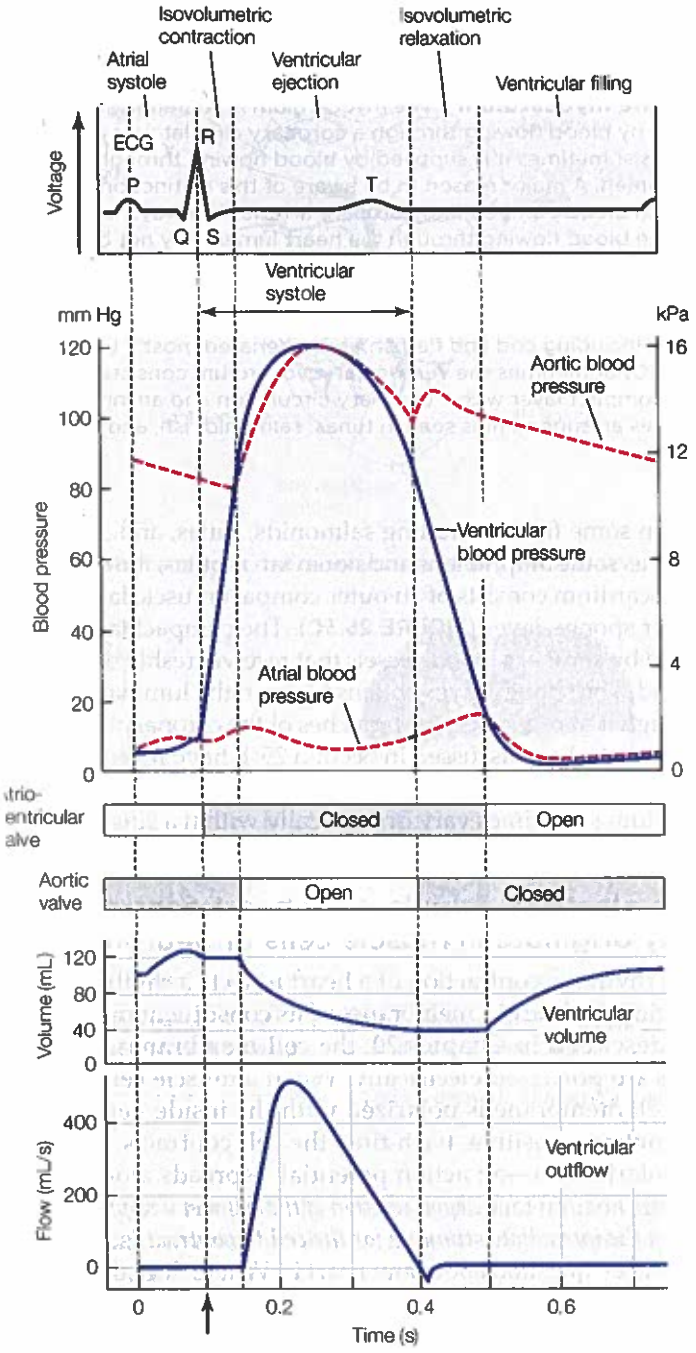
[2] The **systemic tissues** are all the tissues other than the tissues of the breathing organs.

[3] By definition, **veins** are vessels that carry blood toward the heart, and **arteries** are vessels that carry blood away from the heart.

The heart as a pump: The action of a heart can be analyzed in terms of the physics of pumping

During the beating cycle of any type of heart, the period of contraction is called **systole** (pronounced with a long *e*: *sis-tuh-lee*), and the period of relaxation is termed **diastole** (*dy-as-tuh-lee*). The heart is a pump, and we can understand its workings as a pump by analyzing pressure, flow, and volume during these periods. Here, as an example, we analyze the workings of the human left heart (left atrium and ventricle), shown in FIGURE 25.2.

At the time marked by the arrow at the bottom of Figure 25.2, ventricular systole begins. Whereas the pressure inside the ventricle was lower than that inside the atrium during the time just before the arrow, as soon as the ventricle starts to contract (marked by the arrow), the ventricular pressure rises abruptly to exceed the atrial pressure, causing the atrioventricular valve between the chambers to flip shut. For a brief interval of time (about 0.05 s), however, the ventricular pressure remains below the pressure in the systemic aorta, meaning that the aortic valve is not forced open. During this interval, therefore, both the inflow and outflow valves of the ventricle are shut. The volume of blood in the ventricle during this time is thus constant, and



IGURE 25.2 The heart as a pump: The dynamics of the eft side of the human heart The heart cycle is divided into ve phases, labeled at the top and demarcated by the vertical ashed lines that run through the diagram. The diagram shows ne synchronous changes that occur in left ventricular blood pres- ure, systemic aortic blood pressure, left atrial blood pressure, entricular volume, the rate of blood flow out of the ventricle, and ne closing and opening of the atrioventricular and aortic valves ı humans at rest. The arrow at the bottom marks the start of ven- ricular systole. The ECG (see top panel) is the electrocardiogram, iscussed later in Section 25.1.

he interval is called the phase of **isovolumetric contraction** "contraction with unchanging volume") or **isometric ontraction**. The contraction of the ventricle on the fixed vol- ıme of blood within causes the blood pressure inside the ven- ricle to rise rapidly. As soon as the ventricular pressure rises ıgh enough to exceed the aortic pressure, the aortic valve flips open, and the blood in the ventricle accelerates extremely rapidly, gushing out into the aorta (thus increasing aortic pressure). The opening of the aortic valve marks the start of the phase of **ventricular ejection**. Toward the end of this phase, the aortic pressure comes to exceed the ventricular pressure slightly, but ejection of blood into the aorta continues for a while—at a rapidly falling rate—because of blood momentum. Ultimately, the ventricle starts to relax. The ventricular pressure then falls rapidly away from the aortic pressure, and the aortic valve shuts. A period of **isovolumetric relaxation** follows, as ventricular pressure falls with both the inflow and outflow valves shut. When the ventricular pressure drops below the atrial pressure, the atrioventricular valve opens inward to the ventricle, and **ventricular filling** begins. Most filling of the ventricle occurs *before* atrial systole—that is, before the atrial muscle contracts; the motive force for this filling is the pressure built up by *accumulation* of pulmonary venous blood in the atrium. When atrial systole occurs, it forces some additional blood into the ventricle just before the next ventricular systole.

In thinking of any heart as a pump, its most important attribute is the volume of blood it pumps per unit of time, known as the **cardiac output**. (In the case of the mammalian or avian heart, the term *cardiac output* refers specifically to the output of the left ventricle into the systemic aorta unless stated otherwise.) The cardiac output is the product of the heart rate and the **stroke volume**, the volume of blood pumped per heart cycle:

$$\underset{\text{(mL/minute)}}{\text{Cardiac output}} = \underset{\text{(beats/minute)}}{\text{heart rate}} \times \underset{\text{(mL/beat)}}{\text{stroke volume}} \quad (25.1)$$

The circulation must deliver O_2 to the myocardium

The myocardium of any heart (the "heart muscle") performs sustained, vigorous work, and its cells therefore are especially dependent on a steady O_2 supply. In most vertebrates, the ventricular myocardium is second only to the brain in its reliance on aerobic catabolism and in the urgency with which it requires O_2.

In mammals and birds, the ventricular myocardium is classified as **compact** because its muscle cells are packed closely together, much as cells are in other sorts of muscles. Blood passing through the *ventricular lumen*—the open central cavity of the right or left ventricle—cannot flow directly among the myocardial muscle cells because of their close packing. The myocardium therefore is not oxygenated by the blood flowing through the heart lumen. Instead, the ventricular myocardium in mammals and birds is supplied with tissue blood flow and O_2 by a system of blood vessels called the *coronary circulation* (FIGURE 25.3A). **Coronary arteries** branch from the systemic aorta at its very beginning and carry freshly oxygenated blood to capillary beds throughout the myocardium; the blood then flows into **coronary veins**, which carry it out of the myocardium and into the right atrium. If a coronary artery becomes blocked, the part of the myocardium it supplies quickly deteriorates because of O_2 deprivation, explaining why occlusions in the coronary arteries are extremely dangerous.

The process by which depolarization spreads through the vertebrate heart or any other myogenic heart is known as **conduction**. Critical details of conduction in the mammalian heart depend on key structural features of the heart. The myocardium of the two atria of the heart is separated, for the most part, from the myocardium of the two ventricles by a layer of fibrous connective tissue across which myocardial cells are not electrically coupled by gap junctions and through which depolarization therefore cannot pass. In the mammalian heart, the one "electrical window" through this fibrous layer is provided by a **conducting system** composed of specialized muscle cells. As shown in Figure 25.4A, the conducting system starts with a group of cells in the right atrial wall known as the **atrioventricular (A-V) node**. Emanating from this node is a bundle of cells called the **atrioventricular bundle** (*common bundle, bundle of His*), which penetrates the fibrous layer and enters the *interventricular septum*—the wall of tissue that separates the right and left ventricles. Once in the septum, the atrioventricular bundle divides into right and left portions, the **bundle branches**, which travel along the right and left surfaces of the septum and connect with systems of large, distinctive muscle cells, the **Purkinje fibers**, that branch into the ventricular myocardium on each side.

The conducting system of the mammalian heart has two key *functional* properties: (1) Depolarization in the right atrial muscle enters the A-V node and traverses the node relatively slowly, and (2) depolarization spreads down the atrioventricular bundle, bundle branches, and systems of Purkinje fibers much more rapidly than it could travel through ordinary ventricular muscle. The implications of these properties become apparent when we consider the sequence of events during a heartbeat, shown in FIGURE 25.4B. Steps ❶ and ❷ show that once the sinoatrial (S-A) node initiates a heartbeat by depolarizing spontaneously, the depolarization spreads rapidly throughout the muscle of both atria, leading to atrial contraction. Spread into the ventricular muscle does not occur as rapidly, however, because it is dependent on activation of the conducting system, and the spread of depolarization into and through the initial part of the conducting system—the A-V node—is relatively slow (step ❷). This slowness of depolarization of the A-V node is responsible for the sequencing of contraction: atrial contraction distinctly first, ventricular contraction distinctly second. Once the A-V node is activated, depolarization sweeps rapidly down the conducting system into the ventricles (step ❸), precipitating wholesale ventricular depolarization and contraction (step ❹). The rapid delivery of the depolarizing wave to far-flung parts of the ventricular tissue by the conducting system ensures that all parts of the ventricular myocardium contract approximately together.

NEUROGENIC HEARTS The defining feature of neurogenic hearts is that the rhythmic depolarization responsible for initiating the heartbeats originates in nervous tissue. The hearts of lobsters are well-documented examples. Each muscle cell in a lobster heart is innervated and typically contracts when and only when stimulated to do so by nerve impulses (neuronal action potentials). As shown in FIGURE 25.5, a **cardiac ganglion** consisting of nine neurons is attached to the inside of the dorsal wall of the heart. The axonal processes of the five most anterior neurons (numbered 1–5) innervate the heart muscle. Those of the four posterior neurons (numbered 6–9) are confined to the ganglion and make synaptic contact with the five anterior neurons. One of the posterior neurons ordinarily assumes the role of pacemaker. This neuron functions as a cellular oscillator and central pattern generator (see Section 19.2): Periodically and spontaneously, the neuron produces a train of impulses, which excite the other posterior neurons. The impulses from the posterior neurons activate the five anterior neurons, which in turn send trains of impulses to the muscle cells of the heart, causing the latter to contract approximately in unison. If the ganglion and heart muscle are dissected apart, the ganglion continues to produce bursts of impulses periodically, but the muscle ordinarily stops contracting! Other animals known or believed to have neurogenic hearts include other decapod crustaceans (e.g., crabs, shrimps, and crayfish), horseshoe crabs (*Limulus*), and spiders and scorpions.

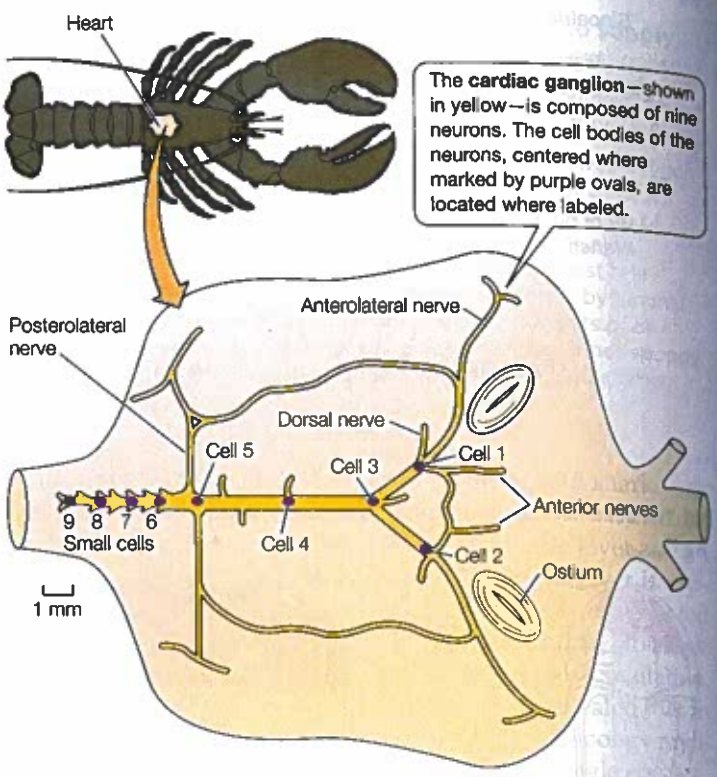


FIGURE 25.5 The neurogenic heart of a lobster and the cardiac ganglion that initiates and controls its contractions A dorsal view of the heart of the American lobster (*Homarus americanus*), showing the cardiac ganglion, which is positioned on the inside of the dorsal heart wall. Neuronal processes go out from each of the nine cell bodies in the ganglion and together form the structure of the ganglion. The posterior four neurons (numbers 6–9) are small, whereas the anterior five are large. Neuronal processes exit the ganglion to innervate the cells of the heart muscle. Regulatory neurons from the central nervous system enter the ganglion in the dorsal nerve. The ostia (slitlike openings through the heart wall) are discussed later in Section 25.8. (After D. K. Hartline. 1967. *J. Exp. Biol.* 47: 327–346.)

A heart produces an electrical signature, the electrocardiogram

When a mass of heart muscle is *in the process* of being depolarized, such that some regions of cells are depolarized already and others await depolarization, a difference in electrical potential exists between the extracellular fluids in the depolarized regions of the muscle and those in the undepolarized regions (FIGURE 25.6A). A voltage difference of this sort within the heart muscle sets up ionic currents, not only in the muscle but also in the tissues and body fluids surrounding the heart. In this way, the voltage difference within the heart induces voltage differences elsewhere in the body, even between various parts of the external body surface. **Electrocardiograms (ECGs, EKGs)** are measurements over time of voltage differences of this sort. They are recorded using extracellular electrodes, usually placed on the body surface. To record an elementary ECG of a person, one can simply use two electrodes, placed at any two distinctly different places on the skin surface.[7] The electrodes detect voltage differences on the skin surface that are induced by voltage differences within the heart muscle.

The ECGs of two species, human and octopus, are shown in FIGURE 25.6B. The waveforms in the human ECG are named with letters (FIGURE 25.6C). The **P wave** is produced by the depolarization of the myocardium of the two atria (= atrial contraction). The Q, R, and S waves, together known as the **QRS complex**, arise from the depolarization of the myocardium of the two ventricles (= ventricular contraction). Repolarization of the ventricles generates the **T wave**.[8] Figure 25.2 shows the relation of the ECG waveforms to mechanical events during the heart pumping cycle.

Heart action is modulated by hormonal, nervous, and intrinsic controls

Heart action is subject to hormonal, nervous, and intrinsic controls. The controls we are typically most aware of are hormonal. When we are frightened and our heart pounds and races, the hormones epinephrine and norepinephrine, secreted by the adrenal medullary glands, are in part responsible for the heart stimulation we experience.

Nearly all hearts—whether myogenic or neurogenic—are innervated by neurons coming from the central nervous system, termed *regulatory neurons*. Some of these neurons stimulate increased heart action, whereas others are inhibitory. In the mammalian heart, both the sinoatrial node—the pacemaker—and the muscle cells of the myocardium are profusely innervated by the sympathetic and parasympathetic divisions of the autonomic nervous system. Sympathetic impulses delivered to the S-A node increase the frequency of spontaneous depolarization by the pacemaker cells (by affecting ion channel proteins) and thus raise the heart rate, whereas

[7] For recording more advanced electrocardiograms used for diagnostic purposes, a physician or nurse attaches multiple skin electrodes, permitting the waves of potential difference sweeping across the skin surface to be recorded in multiple specific dimensions.

[8] The waveform produced by repolarization of the atria is typically not seen because it is obscured by the QRS complex.

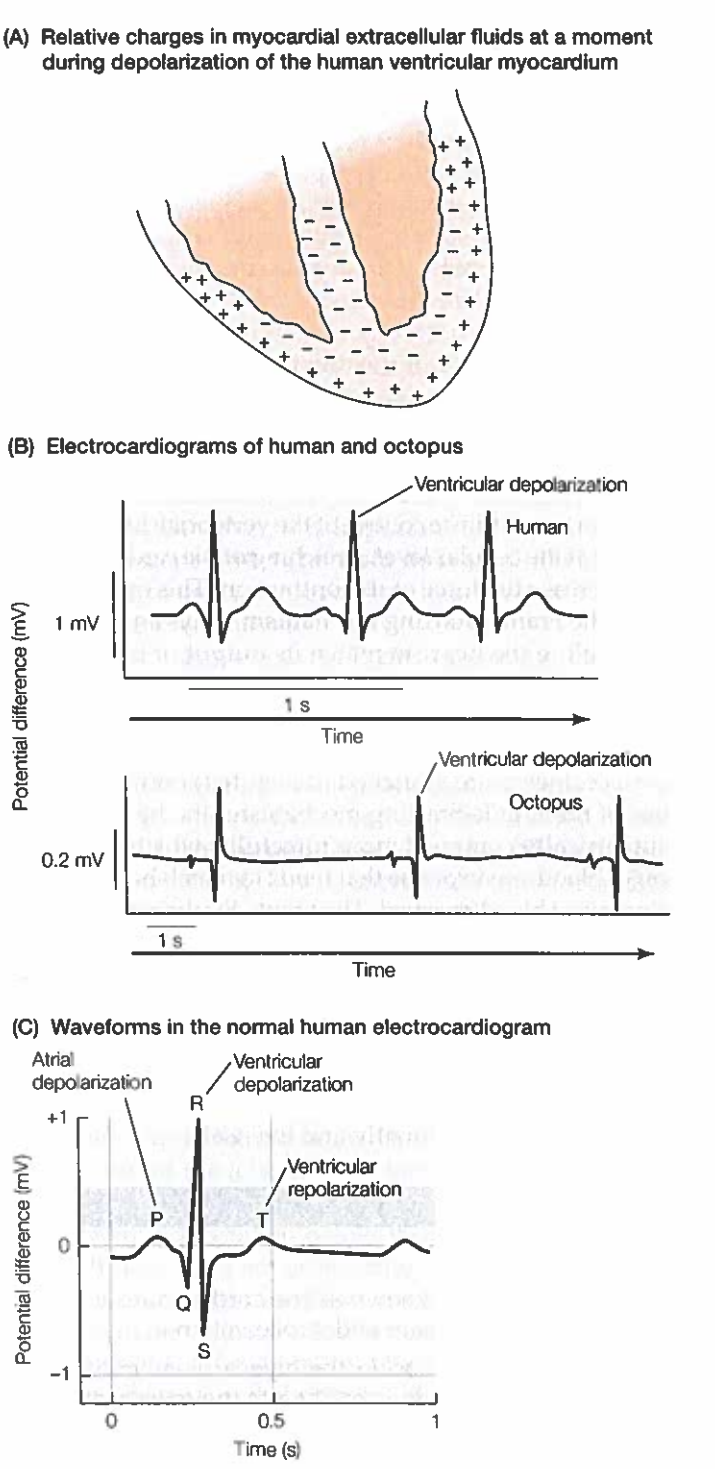


FIGURE 25.6 Electrocardiography (A) Relative electrical charges in the extracellular fluids of the human ventricular myocardium at an instant during passage of a wave of depolarization. The part of the ventricular myocardium lying nearest the ventricular chambers depolarizes first because it is the part activated immediately by the branches of the conducting system (see Figure 25.4). (B) Electrocardiograms of a human and an octopus (*Eledone cirrhosa*) during three heartbeats. The human ECG was obtained using electrodes placed on the skin surface of the right arm and left leg. Electrodes attached to the surface of the ventricle (main body) of the systemic heart were used to record the octopus ECG. (C) A human ECG during one heartbeat with waveforms identified. (Octopus ECG after P. J. S. Smith. 1981. *Comp. Biochem. Physiol. A.* 70: 103–105.)

parasympathetic impulses exert opposite effects. Sympathetic impulses delivered to the cells of the myocardium markedly enhance the force and speed of their contraction, whereas parasympathetic impulses reduce the force and speed of contraction. When people exercise, sympathetic stimulation of the heart is increased. Most vertebrates are similar to mammals in that their hearts receive both sympathetic excitatory innervation and parasympathetic inhibitory innervation.[9] In lobsters, the cardiac ganglion is innervated by both excitatory and inhibitory regulatory neurons; these neurons modulate both the frequency and intensity of the bursts of impulses generated by the ganglion and thus affect the heart rate and the force of heart contraction.

Intrinsic controls of heart action are controls that occur without the mediation of hormones or extrinsic neurons. One vitally important intrinsic control of the vertebrate heart is that, by an effect at the cellular level, stretching of the cardiac muscle tends to increase the force of its contraction. This mechanism, known as the **Frank–Starling mechanism**, plays an important role in enabling the heart to match its output of blood to its input. Consider, for example, what happens when the rate of blood flow into a heart chamber is increased. Because the heart chamber then tends to take in more blood in the time between beats, it becomes more stretched (distended) between beats. Because of the Frank–Starling mechanism, the heart muscle then intrinsically contracts more forcefully, which enhances ejection of blood—a response that tends to match heart output to the increased blood received. The Frank–Starling mechanism is important in all vertebrate hearts studied. Lobster hearts function similarly to vertebrate hearts in that they intrinsically increase both the rate and force of their contraction as they are stretched. The mechanism of this response in lobsters is, at least in part, quite different from that in vertebrates, however, because the cardiac ganglion is involved. Stretch induces the ganglion to fire more frequently and intensely.

SUMMARY

Hearts

- The output of a heart, known as the cardiac output, depends on the heart rate and stroke volume.
- The cells in the heart muscle, the myocardium, must have means of receiving O_2. In some hearts the myocardium is spongy, and blood flowing through the heart chambers flows through the spongy spaces, supplying O_2 to the cells. In other hearts, including those of mammals, the myocardium is compact and is supplied with blood and O_2 by means of coronary blood vessels.
- A heart is myogenic if the depolarization impulses required for heartbeats originate in muscle cells or modified muscle cells. A heart is neurogenic if the impulses originate in neurons. Vertebrate hearts are myogenic. Hearts of adult decapod crustaceans are neurogenic.
- In the mammalian heart, the sinoatrial node in the wall of the right atrium acts as pacemaker, initiating waves of depolarization. Conduction from the atria to the ventricles occurs through the conducting system, which ensures both that the ventricles contract later than the atria and that the entire ventricular myocardium contracts approximately at once.
- When a part of the myocardium is in the process of contracting, voltage differences exist in the extracellular fluids and are transmitted to the body surface. An electrocardiogram is a recording of such differences as a function of time.
- The rate and force of heart contraction are governed by nervous, endocrine, and intrinsic controls.

[9] Some teleost (bony) fish are exceptions in that they have only parasympathetic inhibitory innervation. In hagfish (primitive jawless fish), the heart seems to lack any innervation.

25.2 Principles of Pressure, Resistance, and Flow in Vascular Systems

Having discussed the fundamental features of the hearts of animals, we now turn our attention to the perfusion of the vascular system. **Perfusion** refers to the forced flow of blood through blood vessels.

The **blood pressure** produced by the heart—or, in some animals, by other muscular activity—is the principal factor that drives blood to flow through the vascular system. What we mean by *blood pressure* is the amount by which the pressure of the blood exceeds the ambient pressure (i.e., pressure in the organism's surroundings). Blood pressure is often expressed in *kilopascals* (*kPa*) by physiologists, but usually in *millimeters of mercury* (*mm Hg*) in medicine and related disciplines.[10]

In arteries, the blood pressure rises and falls over the heart cycle. The highest pressure attained at the time of cardiac contraction is termed the **systolic pressure**, whereas the lowest pressure reached during cardiac relaxation is the **diastolic pressure**. In young adult humans at rest, the systolic pressure in the systemic aorta is usually about 16 kPa or 120 mm Hg, and the aortic diastolic pressure is about 10 kPa or 75 mm Hg. When these pressures are measured for clinical reasons, the results are often expressed as a pseudo-ratio—for example, 120/75 ("120 over 75"). The **mean pressure** in an artery is obtained by averaging the pressure over the entire cardiac cycle; it usually does not equal the average of the two extreme pressures, systolic and diastolic, because the systolic and diastolic phases are not the same in duration. In resting young adults, the mean pressure in the systemic aorta is ordinarily about 12.7 kPa or 95 mm Hg.

In addition to the pressures produced dynamically by the beating of the heart, pressures resulting from *fluid-column effects* can also be important in circulatory systems. Any unobstructed vertical column of fluid exerts a pressure—termed a *hydrostatic* pressure—that increases as its height increases (FIGURE 25.7A). Because blood in the vessels of an animal forms fluid columns, fluid-column pressures are present in circulatory systems. The pressure produced by the beating of the heart is added (in an algebraic sense discussed in the next paragraph) to the fluid-column pressures that are present in

[10] Appendix A and Footnote 9 in Section 22.5 discuss the relations among these units.