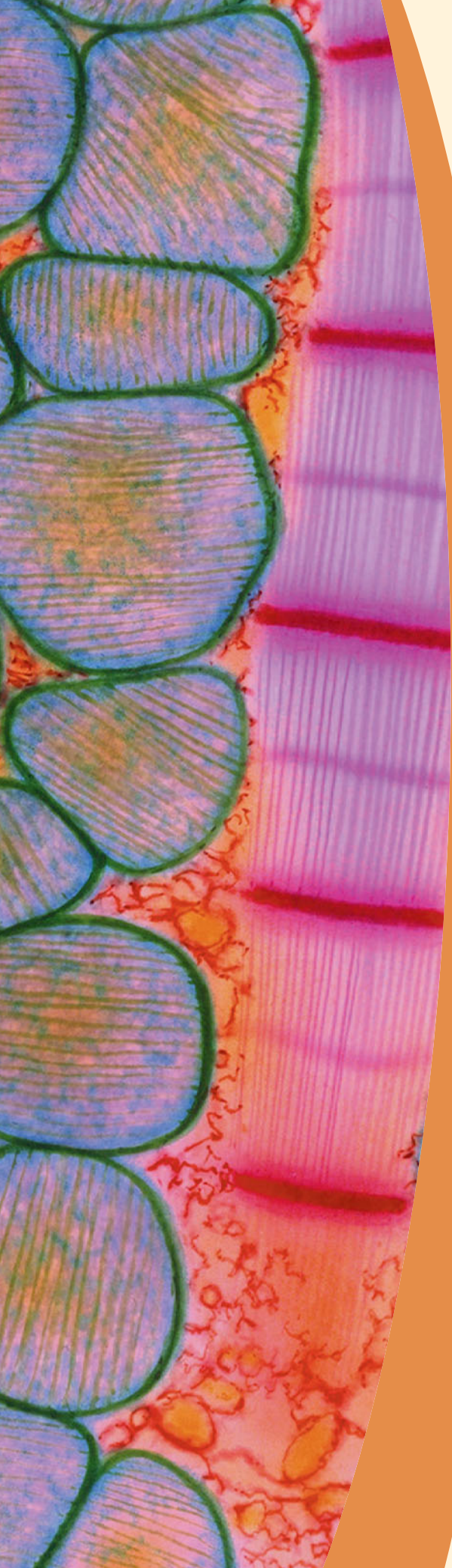


The Heart 19

Healthy cardiac muscle (colored TEM, longitudinal section).

The ceaselessly beating heart in the thorax has intrigued people for thousands of years. The ancient Greeks believed the heart was the seat of intelligence. Others thought it was the source of emotions. Although these theories have proved false, emotions certainly affect heart rate. Only when your heart pounds or occasionally skips a beat do you become acutely aware of this dynamic organ.

The heart is a muscular double pump with two functions: (1) Its right side receives oxygen-poor blood from the body tissues and then pumps this blood to the lungs to pick up oxygen and dispel carbon dioxide, and (2) its left side receives the oxygenated blood returning from the lungs and pumps this blood throughout the body to supply oxygen and nutrients to the body tissues **(Figure 19.1)**. The blood vessels that carry blood to and from the lungs form the **pulmonary circuit** (*pulmonos* = lung), whereas the vessels that transport blood to and from all body tissues form the **systemic circuit.** The heart has two receiving chambers, the *right atrium* and *left atrium* (*atrium* = entranceway), that receive blood returning from the systemic and pulmonary circuits. The heart also has two main pumping chambers, the *right ventricle* and *left ventricle* ("hollow belly"), that pump blood around the two circuits.

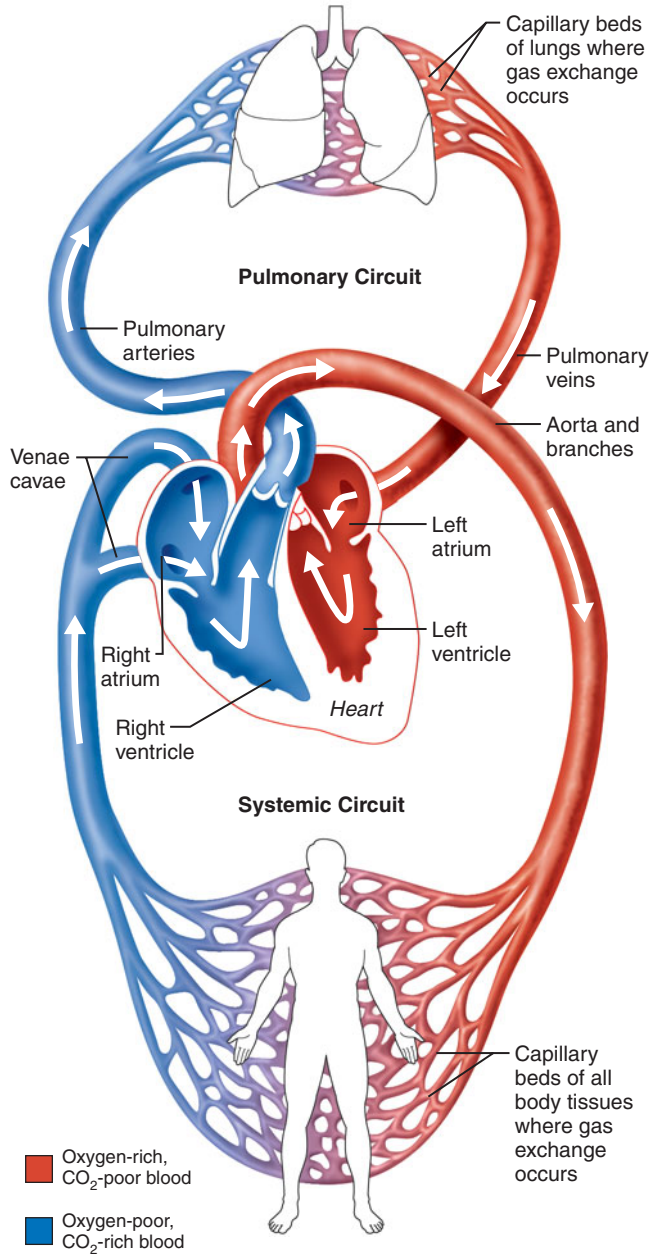


FIGURE 19.1 The heart as a double pump. The right side of the heart pumps blood through the pulmonary circuit. The left side of the heart pumps blood to all the body tissues via the systemic circuit.

LOCATION AND ORIENTATION WITHIN THE THORAX

➤ Describe the orientation, location, and surface anatomy of the heart in the thorax.

The heart's modest size belies its incredible strength and durability. Only about the size of a fist, this hollow, cone-shaped organ looks enough like the popular valentine image to satisfy the sentimentalists among us. Typically it weighs between 250 and 350 grams—less than a pound.

The heart lies in the thorax posterior to the sternum and costal cartilages and rests on the superior surface of the diaphragm **(Figure 19.2a)**. It is the largest organ in the mediastinum, which is the region between the two lungs (and pleural cavities) (Figure 19.2b and c). The heart assumes an oblique position in the thorax, with its pointed **apex** lying to the left of the midline and anterior to the rest of the heart (Figure 19.2c and d). If you press your fingers between the fifth and sixth ribs just inferior to the left nipple, you may feel the beating of your heart where the apex contacts the thoracic wall. Cone-shaped objects have a base as well as an apex, and the heart's **base** is its broad posterior surface.

The heart is said to have four corners defined by four points projected onto the anterior thoracic wall; you may sketch these points on Figure 19.2a. The second rib is easily palpated just lateral to the sternal angle. Use this landmark to help you locate the four corners of the heart. The *superior right* point lies where the costal cartilage of the third rib joins the sternum. The *superior left* point lies at the costal cartilage of the second rib, a finger's breadth lateral to the sternum. The *inferior right* point lies at the costal cartilage of the sixth rib, a finger's breadth lateral to the sternum. Finally, the *inferior left* point (the apex point) lies in the fifth intercostal space at the midclavicular line—that is, at a line extending inferiorly from the midpoint of the left clavicle. The imaginary lines that connect these four corner points delineate the normal size and location of the heart. Clinicians must know these normal landmarks, because an enlarged or displaced heart as viewed on an X-ray or other medical image can indicate heart disease or other disease conditions.

check your understanding

1. Which side of the heart receives and pumps deoxygenated blood?

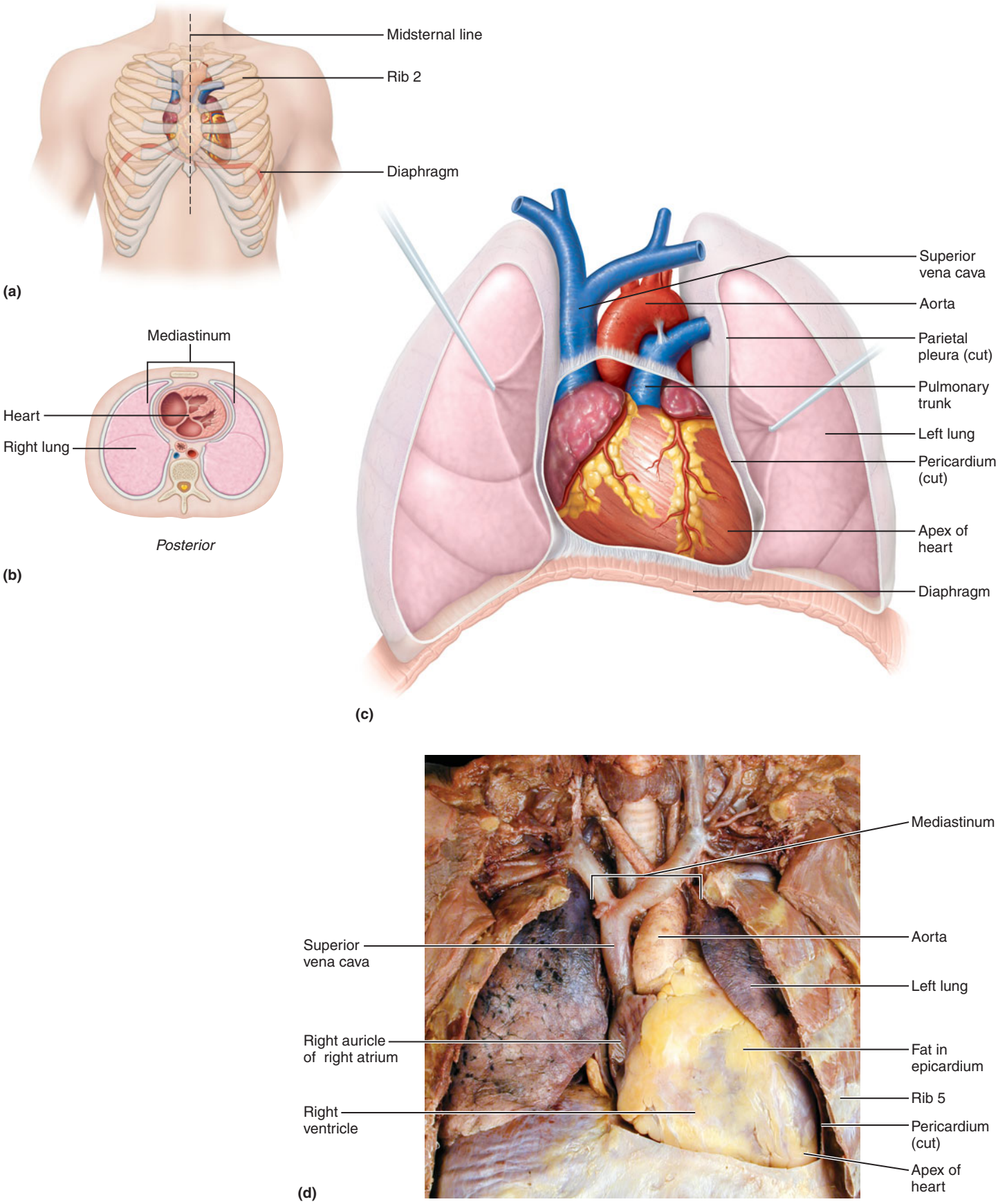


FIGURE 19.2 Location of the heart in the thorax. **(a)** Relation of the heart to the sternum and ribs in a person who is lying down. (In a standing person, the heart lies slightly inferior to this position.) **(b)** Inferior view of a cross section showing the heart's relative position in the thorax. **(c)** Relationship of the heart and great vessels to the lungs. **(d)** Photograph of the heart in the mediastinum. (See *A Brief Atlas of the Human Body,* Second Edition, Figure 56.)

2. Where is the apex of the heart located in reference to the anterior thoracic wall?

For answers, see Appendix B.

STRUCTURE OF THE HEART

- Describe the layers of the pericardium and the tissue layers of the heart wall.
- List the important structural features of each heart chamber: right and left atria, and right and left ventricles.

Coverings

The **pericardium** (per″ ĭ-kar′de-um; "around the heart") is a triple-layered sac that encloses the heart **(Figure 19.3)**. The outer layer of this sac, called the **fibrous pericardium,** is a strong layer of dense connective tissue. It adheres to the diaphragm inferiorly, and superiorly it is fused to the roots of the great vessels that leave and enter the heart. The fibrous pericardium acts as a tough outer coat that holds the heart in place and keeps it from overfilling with blood.

Deep to the fibrous pericardium is the double-layered **serous pericardium,** a closed sac sandwiched between the fibrous pericardium and the heart. The outer, **parietal layer of the serous pericardium** adheres to the inner surface of the fibrous pericardium. The parietal layer is continuous with the **visceral layer of the serous pericardium,** or **epicardium,** which lies on the heart and is considered a part of the heart wall (discussed shortly). Between the parietal and visceral layers of serous pericardium is a slitlike space, called the **pericardial cavity.** The epithelial cells of the serous pericardium that line the pericardial cavity secrete a lubricating film of serous fluid into the pericardial cavity. This fluid reduces friction between the beating heart and the outer wall of the pericardial sac. The pericardial cavity is a division of the embryonic coelom that separates the inner tube from the outer tube.

PERICARDITIS AND CARDIAC TAMPONADE

Infection and inflammation of the pericardium, or **pericarditis,** can lead to a roughening of the serous lining of the pericardial cavity. As a consequence, the beating heart produces a creaking sound called pericardial friction rub, which can be heard with a stethoscope. Pericarditis is characterized by pain behind the sternum. Over time, it can lead to adhesions of the heart to the outer pericardial wall, or the pericardium can scar and thicken inhibiting the heart's movements.

In severe acute cases of pericarditis, large amounts of fluid resulting from the inflammatory response exude into the pericardial cavity. Because the fibrous pericardium is a tough, inflexible tissue, the excess fluid compresses the heart, limiting the expansion of the heart between beats and diminishing its ability to pump blood. This condition is called **cardiac tamponade** (tam″po-nād′; "a heart plug"). Physicians treat it by inserting a hypodermic needle into the pericardial cavity to drain the excess fluid. Cardiac tamponade also results if blood accumulates inside the pericardial cavity, as occurs when a penetrating wound to the heart (such as a stab wound) allows blood to leak out of the heart and into the pericardial cavity.

Layers of the Heart Wall

The wall of the heart has three layers: a superficial *epicardium,* a middle *myocardium,* and a deep *endocardium* (Figure 19.3). All three layers are richly supplied with blood vessels.

The **epicardium** ("upon the heart") is the visceral layer of the serous pericardium, as previously mentioned. This serous membrane is often infiltrated with fat, especially in older people (see Figure 19.2d). The **myocardium** ("muscle heart") forms the bulk of the heart. It consists of cardiac

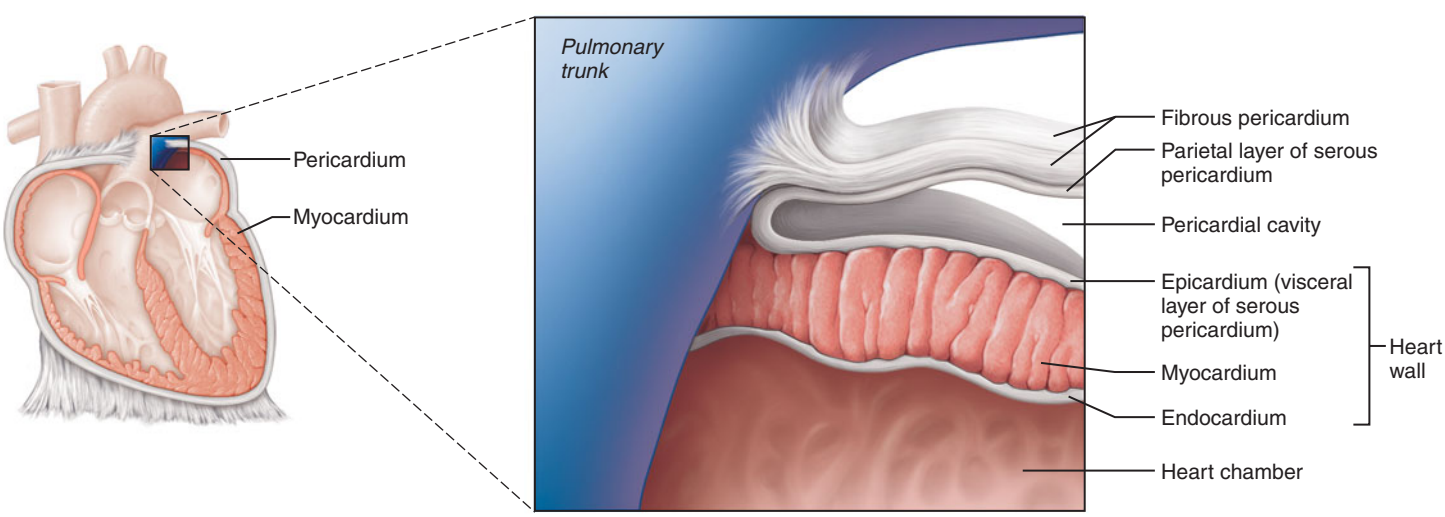


FIGURE 19.3 The layers of the pericardium and of the heart wall.

muscle tissue and is the layer that actually contracts. Surrounding the cardiac muscle cells in the myocardium are connective tissues that bind these cells together into elongated, circularly and spirally arranged networks called *bundles* **(Figure 19.4)**. These bundles function to squeeze blood through the heart in the proper directions: inferiorly through the atria and superiorly through the ventricles. The connective tissues of the myocardium form the *fibrous skeleton of the heart,* which reinforces the myocardium internally and anchors the cardiac muscle fibers. The histological structure and function of cardiac muscle tissue is covered in detail later in this chapter (p. 568). The **endocardium** ("inside the heart"), located deep to the myocardium, is a sheet of simple squamous epithelium resting on a thin layer of connective tissue. Endocardium lines the heart chambers and covers the heart valves.

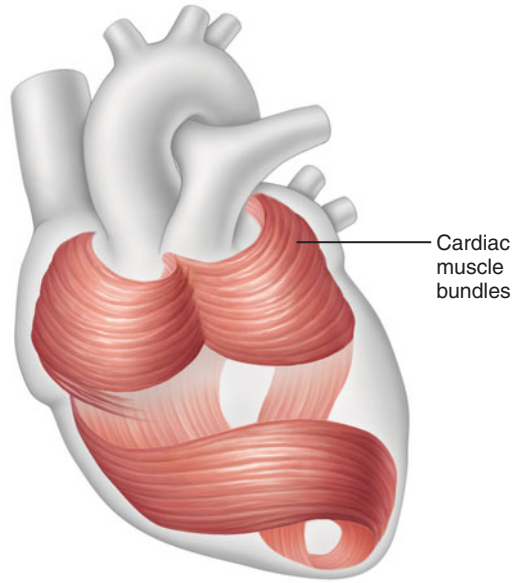


FIGURE 19.4 The circular and spiral arrangement of cardiac muscle bundles in the myocardium of the heart.

Heart Chambers

The four heart chambers are the *right* and *left atria* (singular, *atrium*) superiorly, and the *right* and *left ventricles* inferiorly **(Figure 19.5)**. Internally, the heart chambers are divided longitudinally by a partition called, depending on which chambers it separates, either the **interatrial septum** ("wall between the atria," see Figure 19.5c) or the **interventricular septum** ("wall between the ventricles," Figure 19.5e and f). Externally, the boundaries of the four chambers are marked by two grooves. The first, the **coronary sulcus** (Figure 19.5b), forms a "crown" by circling the boundary between the atria and the ventricles (*corona* = crown). The second groove consists of (1) the **anterior interventricular sulcus** (Figure 19.5a and b), which marks the anterior position of the interventricular septum between the two ventricles, and (2) the **posterior interventricular sulcus** (Figure 19.5d), which separates the two ventricles on the heart's inferior surface. Recall that the heart is oriented obliquely within the thorax (Figure 19.2d); the "posterior" of the heart lies against the diaphragm and is thus its inferior surface.

Right Atrium

The **right atrium** forms the entire right border of the human heart. It is the receiving chamber for oxygen-poor blood returning from the systemic circuit (Figure 19.1). The right atrium receives blood via three veins: the *superior vena cava* and *inferior vena cava* (Figure 19.5b and d) and the *coronary sinus* (Figure 19.5c and d).

Externally, the **right auricle,** a small flap shaped like a dog's ear (*auricle* = little ear) projects anteriorly from the superior corner of the atrium (Figure 19.5a). Internally, the right atrium has two parts (Figure 19.5c): a smooth-walled posterior part and an anterior part lined by horizontal ridges called the **pectinate muscles** (*pectin* = comb). These two parts of the atrium are separated by a large, C-shaped ridge called the **crista terminalis** ("terminal crest"). The crista is an important landmark in locating the sites where veins enter the right atrium: The superior vena cava opens into the atrium just posterior to the superior bend of the crista; the inferior vena cava opens into the atrium just posterior to the inferior bend of the crista; and the coronary sinus opens into the atrium just anterior to the inferior end of the crista. Additionally, just posterior to this end of the crista is the **fossa ovalis,** a depression in the interatrial septum that marks the spot where an opening existed in the fetal heart (the *foramen ovale;* see discussion of fetal circulation on p. 611).

Inferiorly and anteriorly, the right atrium opens into the right ventricle through the *tricuspid valve (right atrioventricular valve)* (Figure 19.5e and f).

Right Ventricle

The **right ventricle** forms most of the anterior surface of the heart. It receives blood from the right atrium and pumps it into the pulmonary circuit via an artery called the **pulmonary trunk** (Figure 19.5a and e). Internally, the ventricular walls are marked by irregular ridges of muscle called **trabeculae carneae** (trah-bek′u-le kar′ne-e; "little beams of flesh"). Additionally, cone-shaped **papillary muscles** project from the walls into the ventricular cavity (*papilla* = nipple).

Thin, strong bands called **chordae tendineae** (kor′de ten′dĭ-ne-e; "tendinous cords"), the "heart strings," project superiorly from the papillary muscles to the flaps (cusps) of the tricuspid (right atrioventricular) valve. Superiorly, the opening between the right ventricle and the pulmonary trunk contains the *pulmonary semilunar valve* (or simply, *pulmonary valve*).

Left Atrium

The **left atrium** makes up most of the heart's posterior surface, or base. It receives oxygen-rich blood returning from the lungs through two right and two left **pulmonary veins** (Figure 19.5d). The only part of the left atrium visible anteriorly is its triangular left auricle (Figure 19.5a and b). Internally, most of the atrial wall is smooth, with pectinate muscles lining the auricle only. The left atrium opens into the left ventricle through the *mitral valve (left atrioventricular valve)* (Figure 19.5e).

Left Ventricle

The **left ventricle** forms the apex of the heart and dominates the heart's inferior surface (Figure 19.5d and e). It pumps

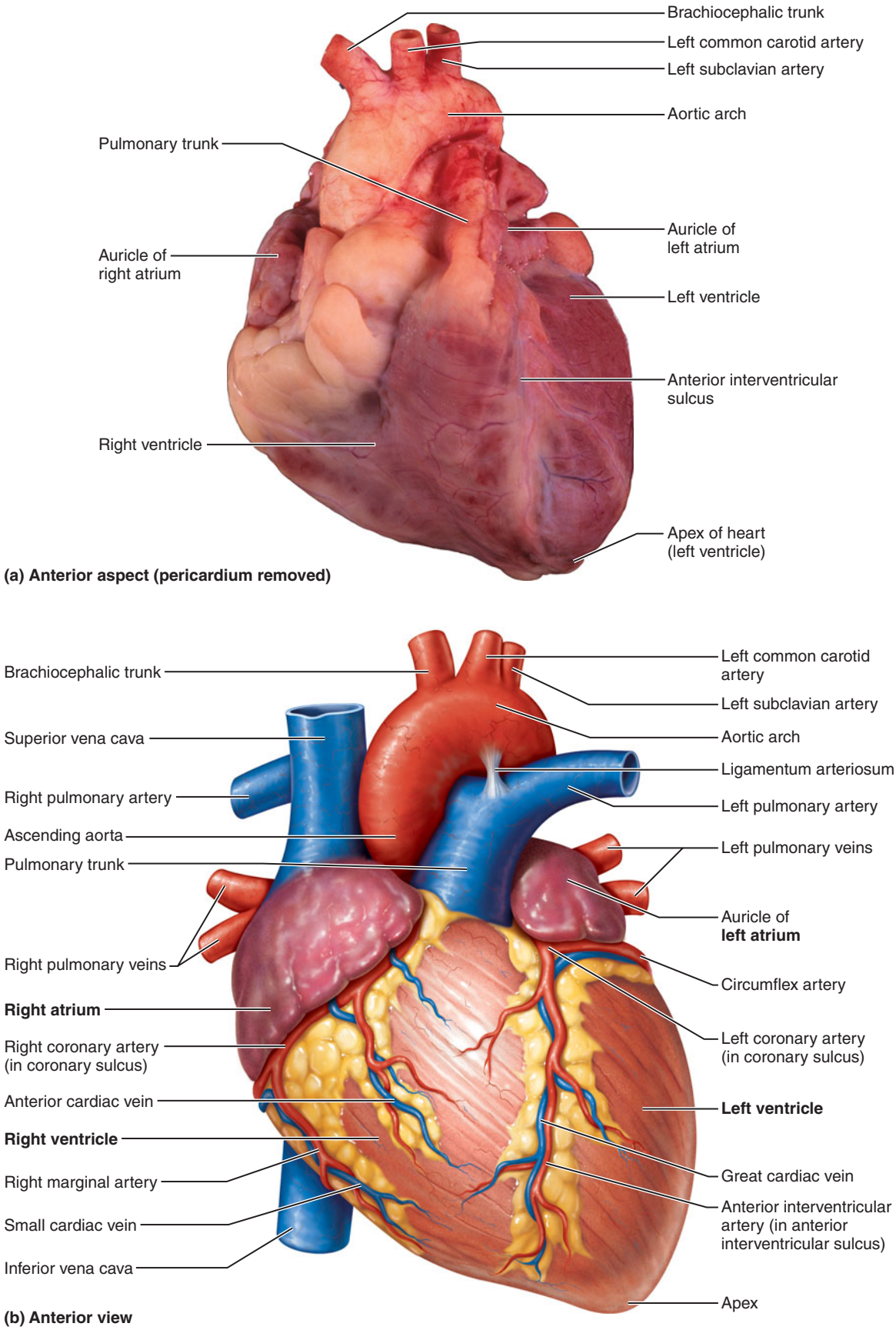
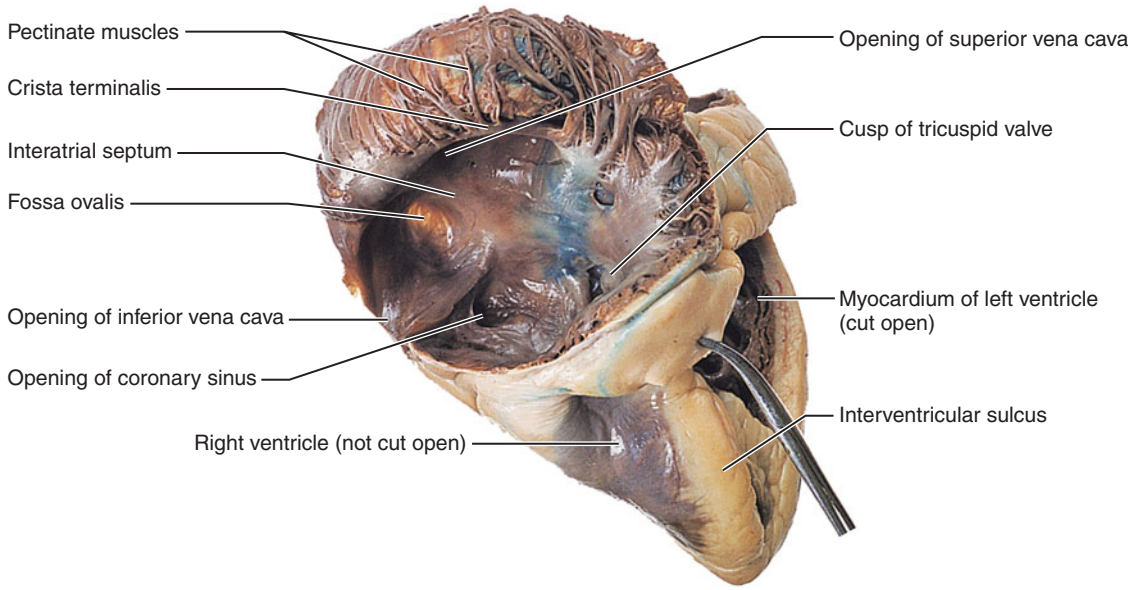
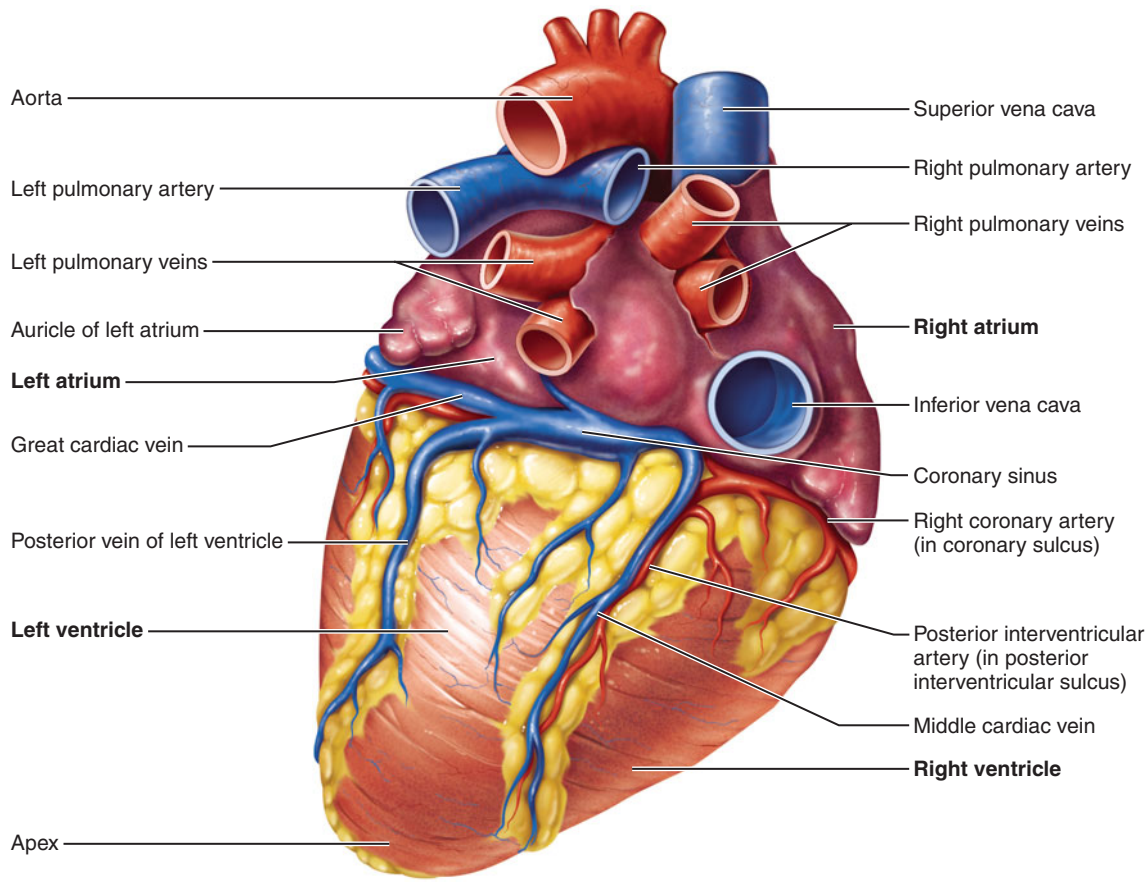


FIGURE 19.5 Gross anatomy of the heart. In diagrammatic views, vessels carrying oxygen-rich blood are red; those carrying oxygen-poor blood are blue. (See *A Brief Atlas of the Human Body,* Second Edition, Figure 57.)

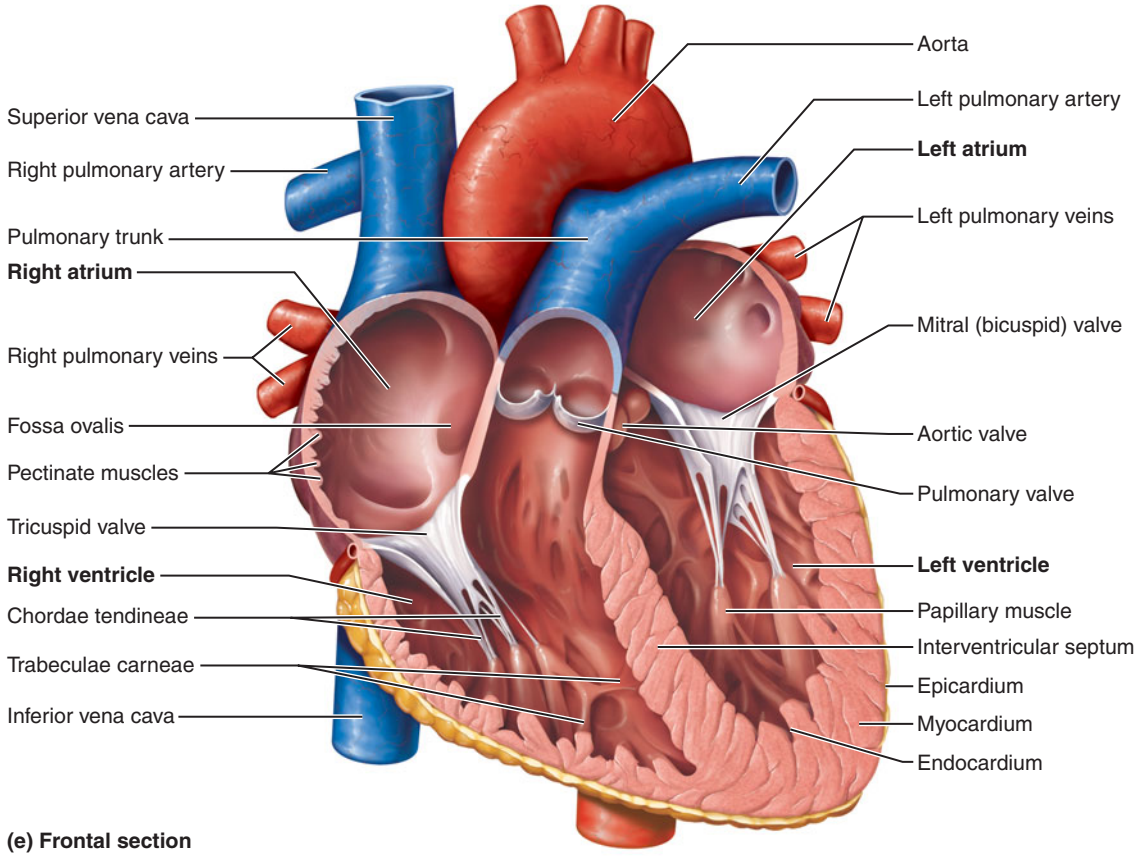


(c) Right anterior view of the internal aspect of the right atrium

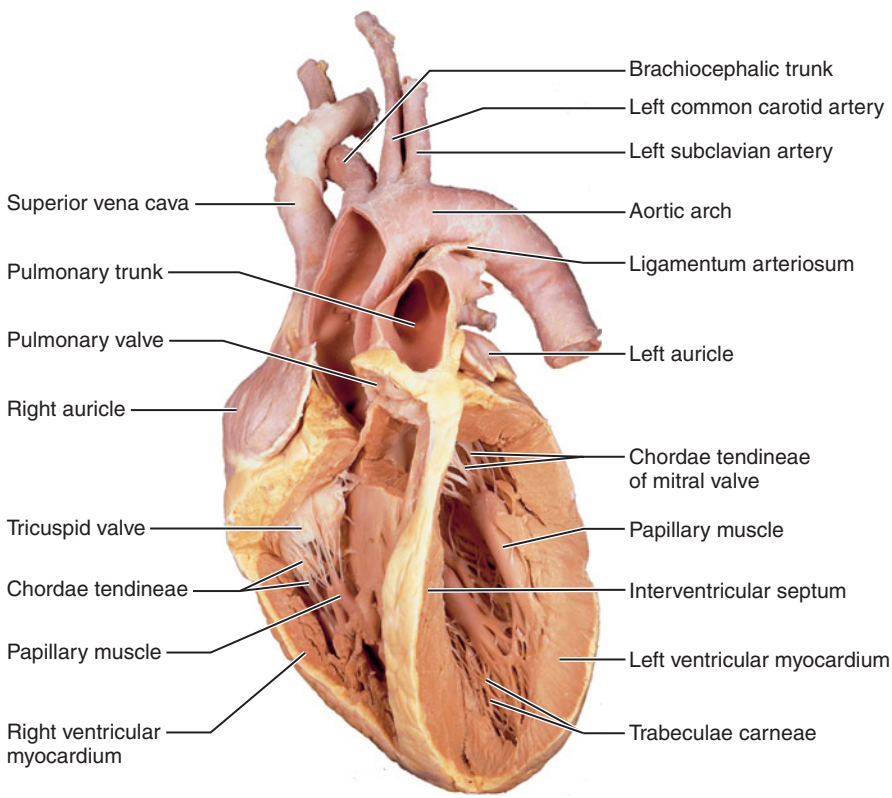


(d) Inferior view; surface shown rests on the diaphragm.

FIGURE 19.5 Gross anatomy of the heart, *continued.* (c), the anterior wall of the atrium has been opened and reflected superiorly. (See *A Brief Atlas of the Human Body,* Second Edition, Figure 59.)



(e) Frontal section



(f) Photograph; view similar to (e)

FIGURE 19.5 Gross anatomy of the heart, *continued.* (See *A Brief Atlas of the Human Body,* Second Edition, Figure 58.)

blood into the systemic circuit. Like the right ventricle, it contains trabeculae carneae, papillary muscles, chordae tendineae, and the cusps of an atrioventricular *(mitral)* valve. Superiorly, the left ventricle opens into the stem artery of the systemic circulation (the aorta) through the *aortic semilunar valve* (or simply, *aortic valve*).

check your understanding

3. What is another name for the epicardium?
4. Identify the heart chamber or chambers that contain the listed structures: (a) pectinate muscles; (b) papillary muscles; (c) fossa ovalis; (d) trabeculae carneae.
5. Name three vessels that empty into the right atrium. Are these vessels arteries or veins?

For answers, see Appendix B.

HEART VALVES

- Name the heart valves, and describe their locations and functions. Indicate where on the chest wall each of the valves is heard.
- Describe the fibrous skeleton of the heart, and explain its functions.

Valve Structure

The heart valves—the paired atrioventricular (AV) and semilunar valves—enforce the one-way flow of blood through the heart, from the atria to the ventricles and into the great arteries that leave the superior part of the heart. Each heart valve consists of two or three *cusps,* which are flaps of endocardium reinforced by cores of dense connective tissue **(Figure 19.6)**. Located at the junctions of the atria and their respective ventricles are the atrioventricular valves: the **right atrioventricular (tricuspid) valve,** which has three cusps, and the **left atrioventricular (bicuspid) valve,** which has only two cusps. The latter is also called the **mitral** (mi′tral) **valve** because its cusps resemble the two sides of a bishop's hat, or miter. Located at the junctions of the ventricles and the great arteries are the **aortic** and **pulmonary (semilunar) valves,** each of which has three pocketlike cusps shaped roughly like crescent moons (*semilunar* = half moon) (Figures 19.6a and b, and 19.5e and f).

The **fibrous skeleton** of the heart lies in the plane between the atria and the ventricles and surrounds all four heart valves rather like handcuffs (Figure 19.6a). Composed of dense connective tissue, it has four functions:

1. It anchors the valve cusps.
2. It prevents overdilation of the valve openings as blood pulses through them.
3. It is the point of attachment for the bundles of cardiac muscle in the atria and ventricles (see Figure 19.4).
4. It blocks the direct spread of electrical impulses from the atria to the ventricles (discussed shortly). This function is critical for the proper coordination of atrial and ventricular contractions.

Valve Function

Heart valves open (to allow blood flow) and close (to prevent the backflow of blood) in response to differences in blood pressure on each side of the valves. The two *atrioventricular valves* prevent the backflow of blood into the atria during contraction of the ventricles **(Figure 19.7)**. When the ventricles are relaxed, the cusps of the AV valves hang limply into the ventricular chambers while blood flows into the atria and down through the open AV valves into the ventricles (Figure 19.7a). When the ventricles start to contract, the pressure within them rises and forces the blood superiorly against the valve cusps, pushing the edges of the cusps together and closing the AV valves. The chordae tendineae and papillary muscles that attach to these valves look like the cords of an open parachute, limiting the closed cusps so they cannot fly up and allow reflux of ventricular blood into the atria. The papillary muscles begin to contract slightly before the rest of the ventricle contracts, pulling on the chordae tendineae and preventing the AV valves from everting (Figure 19.7b). If the cusps were not anchored in this manner, they would be forced superiorly into the atria.

The two *semilunar valves* prevent backflow from the great arteries into the ventricles (**Figure 19.8**, p. 566). When the ventricles contract and raise the intraventricular pressure, the semilunar valves are forced open, and their cusps are flattened against the arterial walls as the blood rushes past them. When the ventricles relax, blood that tends to flow back toward the heart fills the cusps of the semilunar valves and forces them shut.

Heart Sounds

The closing of the valves causes vibrations in the adjacent blood and heart walls that account for the familiar "lub-dup" sounds of each heartbeat: The "lub" sound is produced by the closing of the AV valves at the start of ventricular contraction; the "dup" is produced by the closing semilunar valves at the end of ventricular contraction. The mitral valve closes slightly before the tricuspid closes, and the aortic valve generally closes just before the pulmonary valve closes. Because of these slight differences in timing, all four valve sounds are discernible when the clinician listens through a stethoscope placed on the anterior chest wall.

Even though all four heart valves lie in roughly the same plane (see Figure 19.6a)—the plane of the coronary sulcus—the clinician does not listen directly over the respective valves, because the sounds take oblique paths through the heart chambers to reach the chest wall. Each valve is best heard near a different heart corner: the pulmonary valve near the superior left point, the aortic valve near the superior right point, the mitral valve at the apex

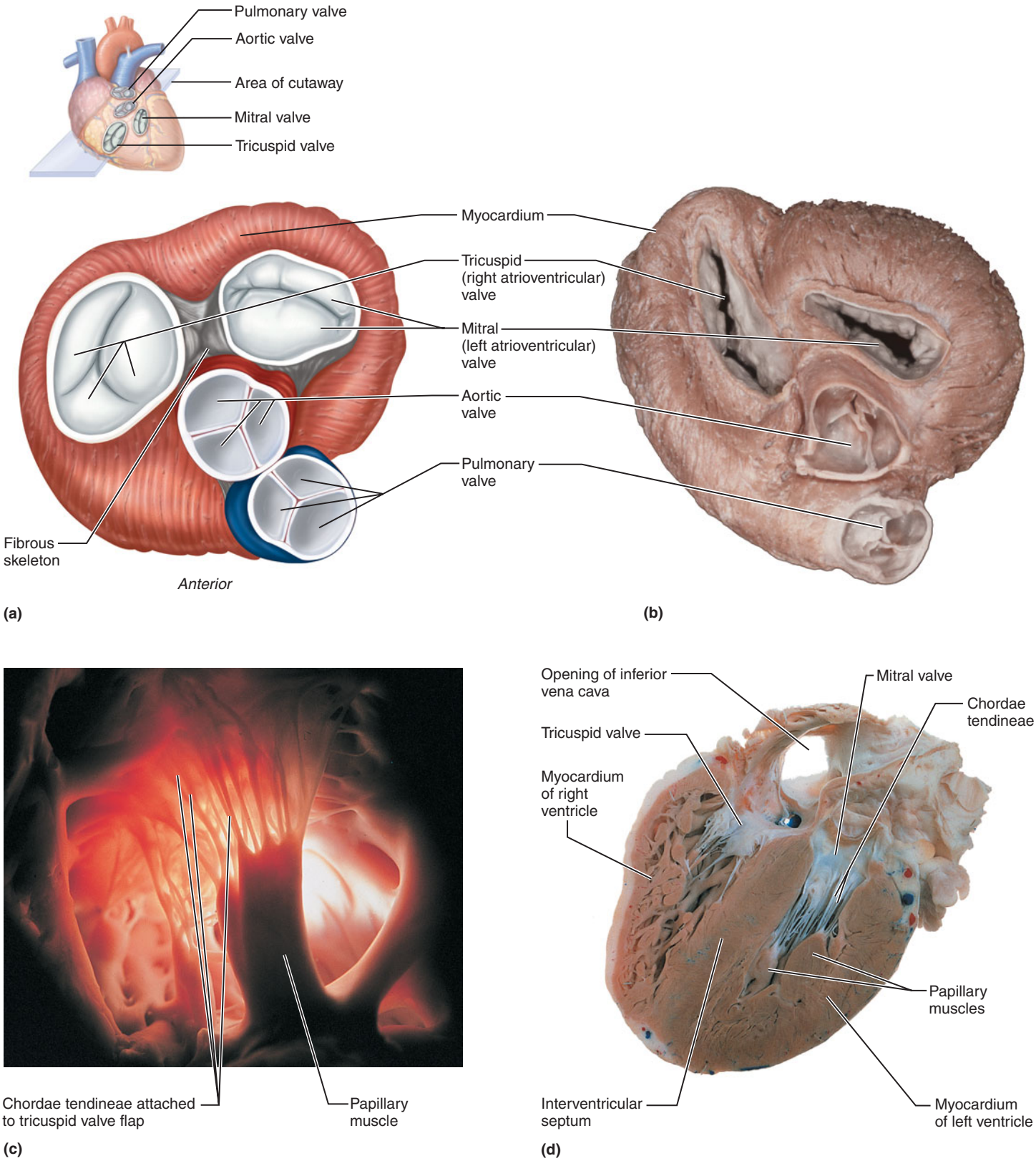
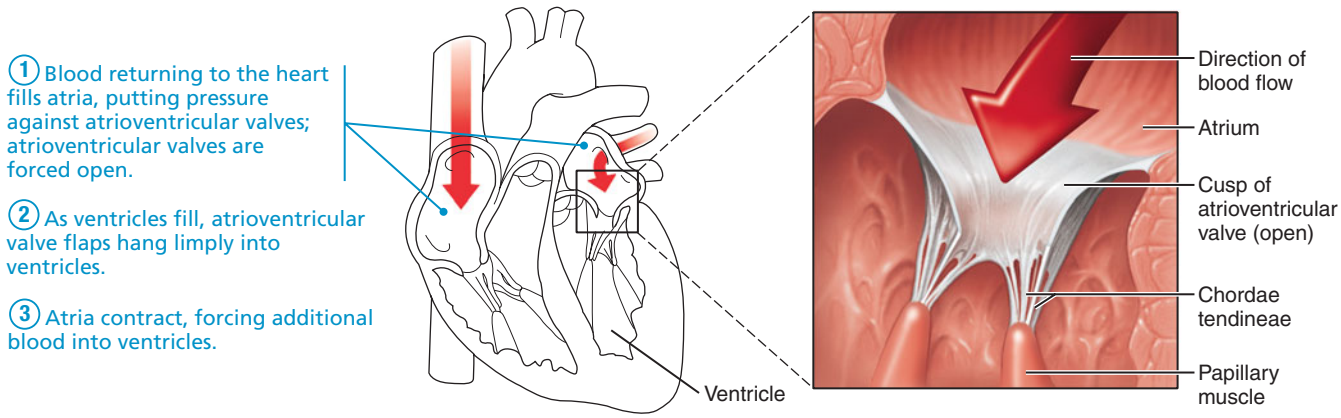
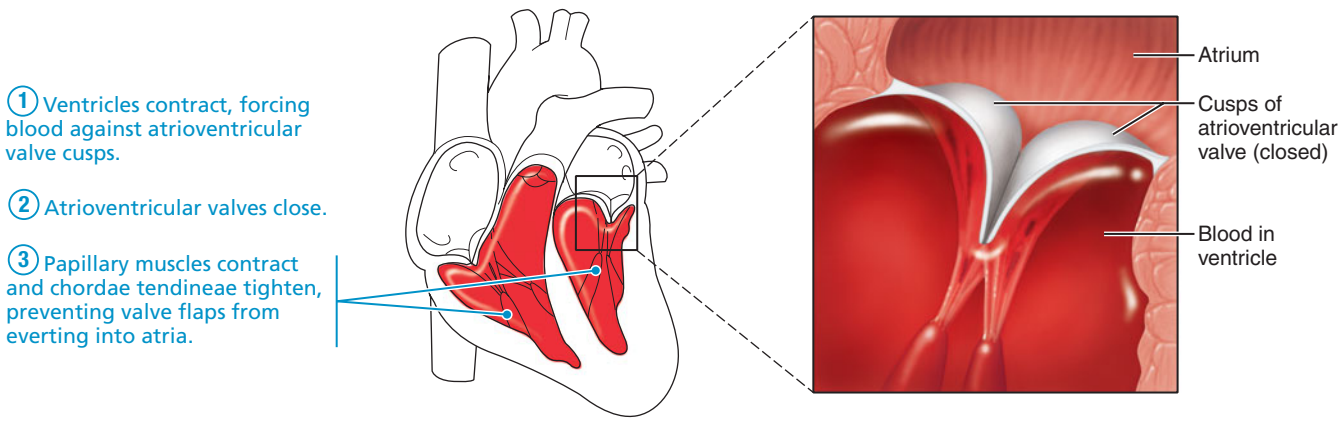


FIGURE 19.6 Heart valves. (a) Superior view of the two sets of heart valves (atria removed). The inset shows the plane of section through the heart. **(b)** Photograph of heart valves, superior view. **(c)** Photograph of the tricuspid valve. This inferior-to-superior view shows the valve as seen from the right ventricle. **(d)** Coronal section of the heart. (See *A Brief Atlas of the Human Body,* Second Edition, Figures 60 and 61.)



(a) AV valves open; atrial pressure greater than ventricular pressure



(b) AV valves closed; atrial pressure less than ventricular pressure

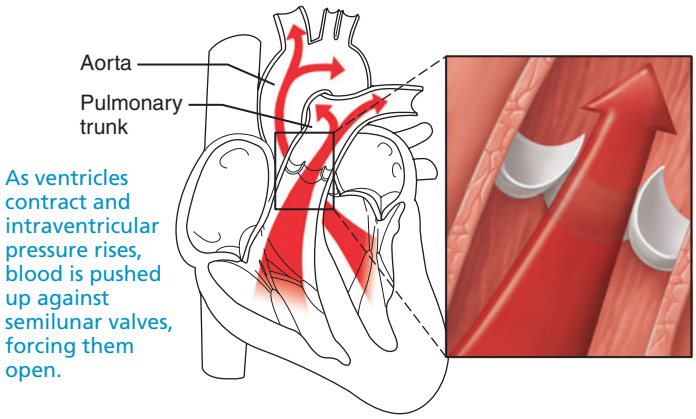
FIGURE 19.7 Function of the atrioventricular valves.

point, and the tricuspid near the inferior right point (Figure 19.9). Abnormal heart sounds often indicate disorders of the heart valves.

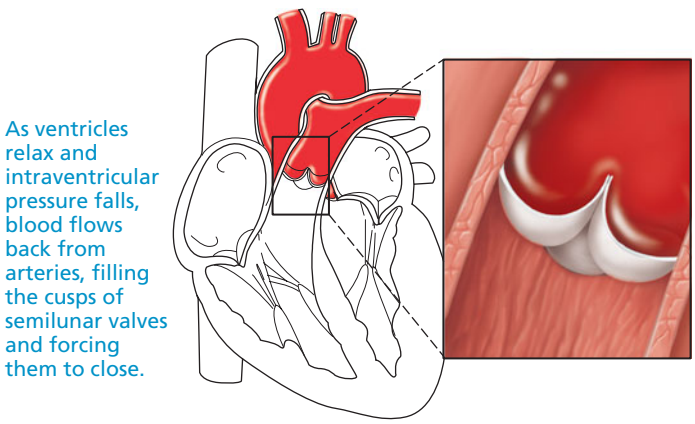
VALVE DISORDERS Defects of the heart valves are common and have a number of causes, ranging from congenital and genetic abnormalities, to an inadequate blood supply to the valves (due to a heart attack), to bacterial infection of the endocardium. Valvular defects lead to valvular disorders, most of which are classified as one of two types. Valves that leak because they fail to close properly are considered **incompetent** (or are said to exhibit **insufficiency**). An incompetent valve produces a distinct blowing sound after the valve closes. By contrast, valves with narrowed openings, such as occur when cusps have fused or become stiffened by calcium deposits, are termed **stenotic.** Stiffened valves cannot open properly. Stenosis of the aortic valve produces a distinctive "click" sound during ventricular systole as blood passing through the constricted opening becomes turbulent and vibrates. Both incompetent and stenotic valves lessen the heart's efficiency and thus increase its workload; ultimately the heart may weaken severely.

The mitral valve, and the aortic valve to a lesser extent, are most often involved in valve disorders because they are subjected to the great forces resulting from contraction of the powerful left ventricle. In **mitral valve prolapse,** an inherited weakness of the collagen in the valve and chordae tendineae allows one or both cusps of this valve to "flop" into the left atrium during ventricular systole. This is the most common heart valve disorder (affecting 2.5% to 5% of the population); it is characterized by a distinctive heart sound—a click followed by a swish (backflow of blood). Most cases are mild and harmless, but severe cases may lead to heart failure or a disruption of heart rhythm.

Treatment of valve disorders typically involves surgical repair of damaged valves. When this approach is unsuccessful, valves are replaced with either synthetic or pig valves. Replacement techniques are neither permanent nor problem-free.



(a) Semilunar valves open



(b) Semilunar valves closed

FIGURE 19.8 Function of the semilunar valves.

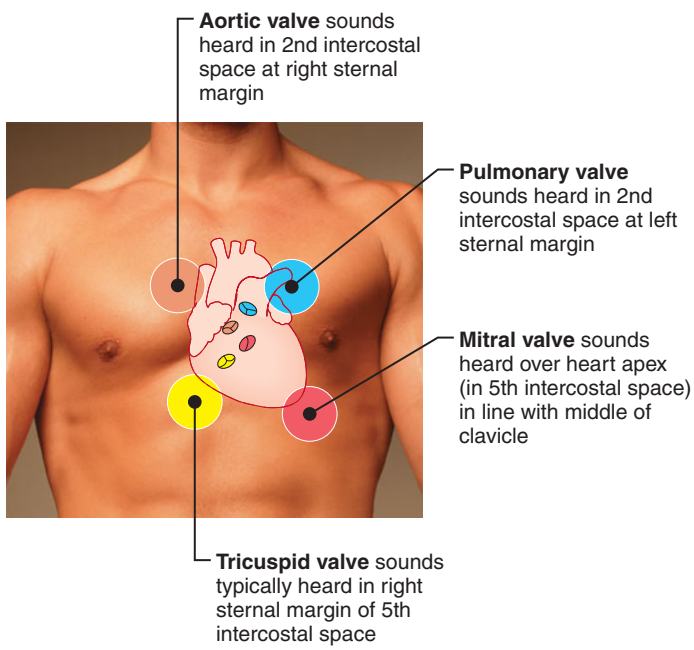


FIGURE 19.9 Areas on the thoracic surface where heart sounds are heard most clearly.

PATHWAY OF BLOOD THROUGH THE HEART

➤ Describe the path of a drop of blood through the four chambers of the heart and the systemic and pulmonary circuits.

Figure 19.10, *Focus on Blood Flow Through the Heart,* illustrates the path of blood around the pulmonary and systemic circuits, beginning with oxygen-poor systemic blood as it arrives at the right side of the heart. Blood coming from body regions superior to the diaphragm (excluding the heart wall) enters the right atrium via the superior vena cava (SVC); blood returning from body regions inferior to the diaphragm enters via the inferior vena cava (IVC); and blood draining from the heart wall itself is collected by and enters through the coronary sinus. The blood passes from the right atrium through the tricuspid valve to the right ventricle, propelled by gravity and the contraction of the right atrium. Then, the right ventricle contracts, propelling the blood through the pulmonary semilunar valve into the pulmonary trunk and to the lungs through the pulmonary circuit for oxygenation. The freshly oxygenated blood returns via the four pulmonary veins to the left atrium and passes through the mitral valve to the left ventricle, propelled by gravity and the contraction of the left atrium. The left ventricle then contracts and propels the blood through the aortic semilunar valve into the aorta and its branches. After delivering oxygen and nutrients to the body tissues through the systemic capillaries, the oxygen-poor blood returns through the systemic veins to the right atrium—and the whole cycle repeats continuously.

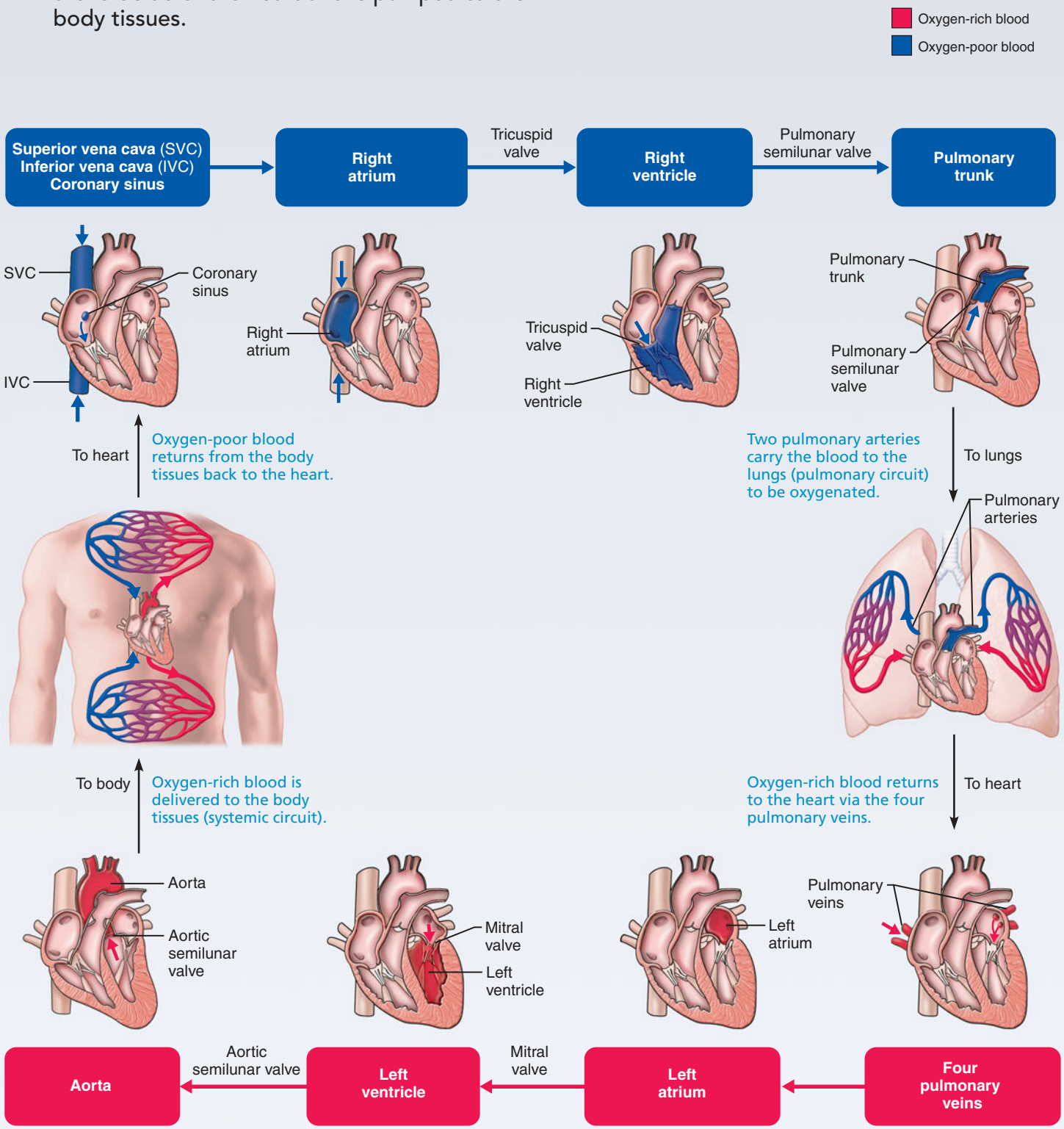
Although a given drop of blood passes through the heart chambers sequentially (one chamber after another), the four chambers do not contract in that order. Rather, the two atria always contract *together,* followed by the simultaneous contraction of the two ventricles.

A single sequence of atrial contraction followed by ventricular contraction is called a **heartbeat,** and the heart of an average person at rest beats 70–80 times per minute. The term that describes the contraction of a heart chamber is **systole** (sis′to-le; "contraction"); the time during which a heart chamber is relaxing and filling with blood is termed **diastole** (di-as′to-le; "expansion"). Thus, both atria and ventricles experience systole and diastole. Be aware, however, that in common medical usage, diastole and systole most often refer to ventricular filling and contraction, respectively. For example, a blood pressure reading measures the ventricular systolic pressure over the ventricular diastolic pressure (systolic/diastolic) in the systemic circuit, that is, the left ventricle. Because the ventricle is relaxed during diastole, elevation of pressure during this portion of the heartbeat is a symptom of hypertension (high blood pressure).

Now that you know how the heart pumps blood, you can readily understand why the muscular walls of its different chambers differ in thickness. The walls of the atria are much thinner than those of the ventricles (Figure 19.5e) because much of ventricular filling is done by gravity, and thus the

FIGURE 19.10

Oxygen-poor blood enters the right side of the heart and is pumped to the lungs where gas exchange occurs. Oxygen-rich blood returns to the left side of the heart and is pumped to the body tissues.



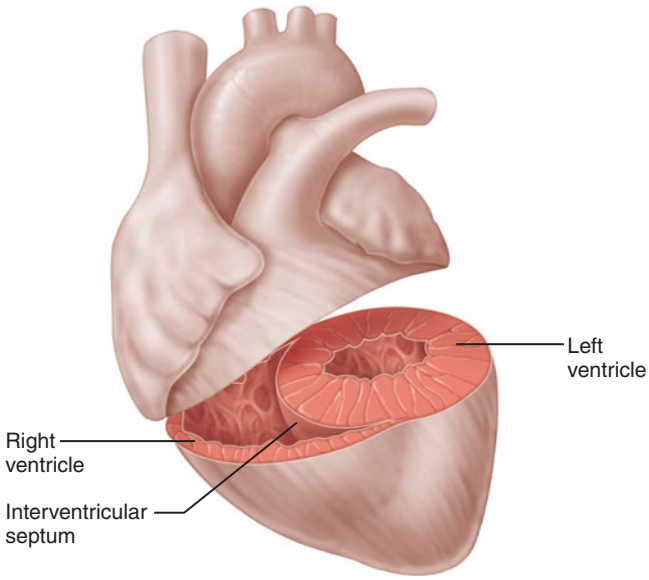


FIGURE 19.11 Anatomical differences between the right and left ventricles. The thicker, circular wall of the left ventricle encroaches on the cavity of the thinner-walled right ventricle, making the cavity of the right ventricle crescent-shaped.

atria exert little effort to propel blood inferiorly into the ventricles. Furthermore, the wall of the left ventricle (the systemic pump) is at least three times as thick as that of the right ventricle (the pulmonary pump) (Figure 19.11). Consequently, the left ventricle can generate much more force than the right and pumps blood at a much higher pressure. The higher pressure in the systemic circuit reflects the fact that the systemic circuit is much longer than the pulmonary circuit and offers greater resistance to blood flow. The thick wall of the left ventricle gives this chamber a circular shape and flattens the cavity of the adjacent right ventricle into the shape of a crescent.

check your understanding

6. Where does blood travel after passing through the aortic semilunar valve? Is this blood oxygenated or deoxygenated?
7. During ventricular systole, are the AV valves open or closed? Are the semilunar valves open or closed during this period?
8. Differentiate a stenotic valve from an incompetent valve.

For answers, see Appendix B.

CARDIAC MUSCLE TISSUE

- Describe the structure of cardiac muscle tissue.
- Describe the structure of intercalated discs and discuss their importance in the contraction of cardiac muscle.

Thus far, we have described the gross anatomy of the heart and how these structures function to move blood through the heart. To understand how the heart produces forceful contractions, we must examine the structure of cardiac muscle tissue.

Cardiac muscle tissue forms the thick myocardium of the heart wall. It contains cardiac muscle cells and the connective tissues that surround these cells. The contractions of cardiac muscle cells pump blood through the heart and into and through the blood vessels of the circulatory system. Cardiac muscle tissue, like skeletal muscle tissue, is striated, and it contracts by the sliding filament mechanism.

Unlike a skeletal muscle cell, which is long, multinucleated, and cylindrically shaped, a cardiac muscle cell is a short, branching cell (Figure 19.12a) with one or two large, centrally located nuclei. Each cell averages about 25 μm in diameter and 120 μm in length. Adjacent cardiac muscle cells are joined together at their ends to form cellular networks. These branching networks of cardiac muscle cells are called *cardiac fibers.*

The complex junctions that join cardiac muscle cells are called **intercalated discs** (in-ter′kah-la″ted; "inserted between"). At these junctions, the sarcolemmas of adjacent cells interlock through meshing "fingers," like one empty egg carton stacked inside another (Figure 19.12b). Intercalated discs have two distinct regions: Transverse regions contain desmosome-like junctions called **fasciae adherens** (singular: **fascia adherens**) that function to bind adjacent cells together and transmit the contractile force to adjacent cells. Longitudinal regions contain gap junctions that allow ions to pass between cells, transmitting the contractile signal to adjacent cells. The free movement of ions between cells allows the direct transmission of an electrical impulse through the entire network of cardiac muscle cells. This impulse in turn stimulates all the muscle cells in a heart chamber (atria or ventricles) to contract at the same time.

In the intercellular spaces around each cardiac fiber is a loose fibrous connective tissue, the *endomysium,* which aids to bind adjacent cardiac fibers together and contains the vessels and nerves that serve the muscle cells. Groups of cardiac fibers form the cardiac muscle bundles in the myocardium (Figure 19.4). The connective tissues surrounding the cardiac fibers merge with the fibrous skeleton of the heart and thus function to anchor the muscle cells and transmit the contractile forces produced by the muscle cells, similar to the tendinous origins and insertions of skeletal muscles.

The striated cardiac muscle cells contain myofibrils with typical sarcomeres composed of A and I bands, H zones, titin, and Z discs and M lines (Figure 19.12b and Figure 19.13). Striations are less apparent in cardiac muscle tissue than in skeletal muscle tissue, especially when viewed by light microscopy, because of the branching of the myofibrils and the great abundance of mitochondria surrounding them (Figure 19.13). The abundant mitochondria make large amounts of ATP by aerobic metabolism; thus, cardiac muscle is highly resistant to fatigue.

The molecular mechanism for contraction in cardiac muscle is similar to that in skeletal muscle. Cardiac muscle

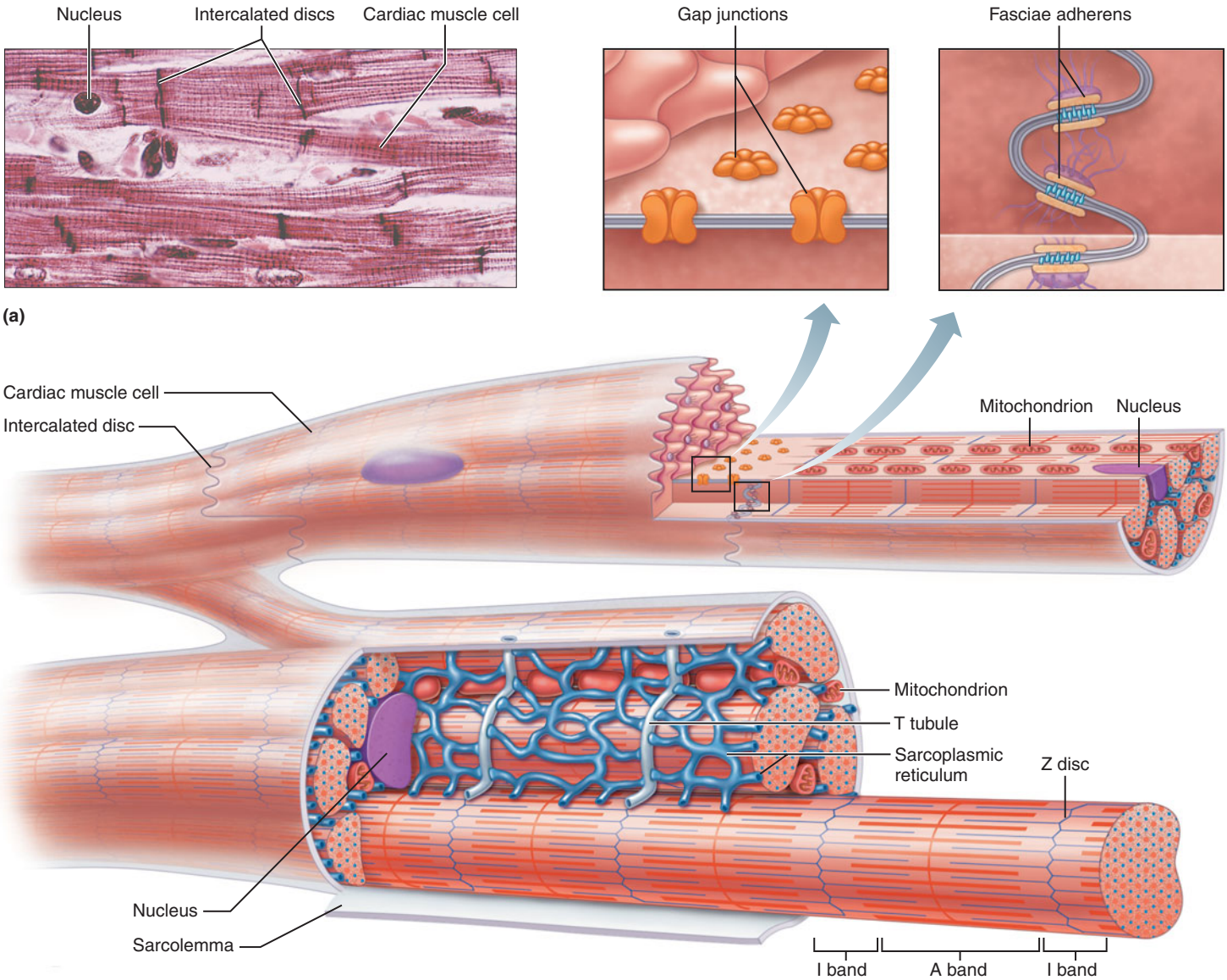


FIGURE 19.12 Microscopic anatomy of cardiac muscle. (a) Photomicrograph of cardiac muscle tissue (430×). Notice that the cardiac muscle cells are short, branched, and striated. The dark-staining areas are intercalated discs, junctions between adjacent cells. **(b)** Diagram of a cardiac muscle cell, partially sectioned, and enlargements of the intercalated disc.

cells are triggered to contract by ionic calcium (Ca^{2+}) entering the sarcoplasm. In response to an action potential, a small amount of Ca^{2+} from the extracellular tissue fluid enters the cardiac muscle cell through the sarcolemma. This rise in intracellular calcium signals the sarcoplasmic reticulum to release its stored Ca^{2+}. These ions diffuse into the sarcomeres and trigger the sliding of the filaments. Reuptake of calcium by the sarcoplasmic reticulum ends contraction. Compared to skeletal muscle, the sarcoplasmic reticulum of cardiac muscle cells is less complex, and the T tubules (recall from skeletal muscle that T tubules are invaginations of the sarcolemma that carry the electrical signal deep into the cell) are less abundant—occurring at Z discs instead of at the A-I junctions (Figure 19.12b and Table 10.2, pp. 254–255).

As with skeletal muscle, the amount of force that cardiac muscle cells can generate depends on their length. Significantly, these cells normally remain slightly *shorter* than their optimal length. Therefore, when they are stretched by a greater volume of blood returning to the heart, their contraction force increases, and they can pump the additional blood.

Unlike cells of skeletal muscle tissue, not all cells of cardiac muscle tissue are innervated. In fact, an isolated cardiac muscle cell will contract rhythmically without any innervation at all. This inherent rhythmicity of cardiac muscle cells is the basis of the rhythmic heartbeat, as explained next.

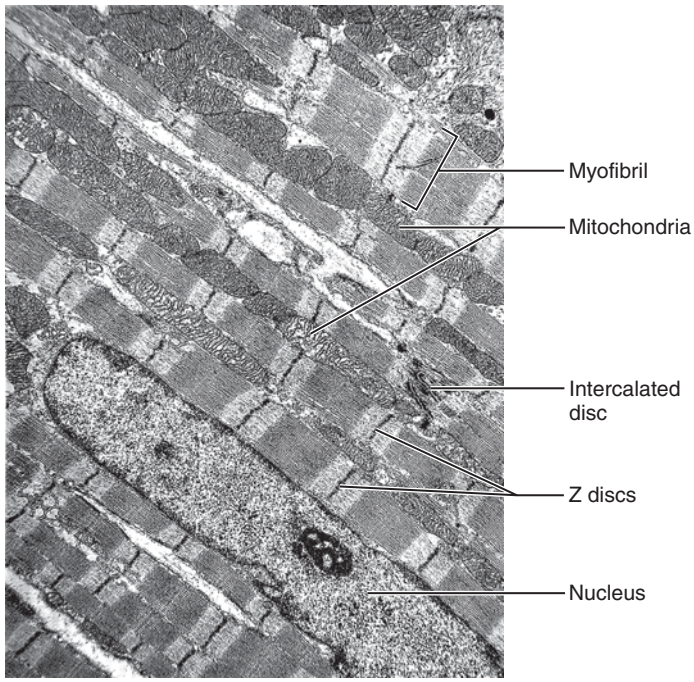


FIGURE 19.13 Electron micrograph of a portion of a cardiac muscle cell. Notice the large number of mitochondria between each myofibril and the irregular size of the myofibrils (2000×).

CONDUCTING SYSTEM AND INNERVATION

➤ Name the components of the conducting system of the heart, and describe the conduction pathway.

Conducting System

Cardiac muscle cells have an intrinsic ability to generate and conduct electrical impulses that stimulate these same cells to contract rhythmically. These properties are intrinsic to the heart muscle itself and do not depend on extrinsic nerve impulses. Even if all nerve connections to the heart are severed, the heart continues to beat rhythmically. Perhaps you remember a beating heart in a dissected frog from your high school biology class.

The **conducting system** of the heart is a series of specialized cardiac muscle cells that carries impulses throughout the heart musculature, signaling the heart chambers to contract in the proper sequence. It also initiates each contraction sequence, thereby setting basic heart rate.

The impulse that signals each heartbeat begins at the **sinoatrial (SA) node** (Figure 19.14, ①), a crescent-shaped mass of muscle cells that lies in the wall of the right atrium, just inferior to the entrance of the superior vena cava. The SA node sets the basic heart rate by generating 70–80 electrical impulses per minute. It is the heart's pacemaker. The signal initiated by the SA node spreads throughout the myocardium through the gap junctions in the intercalated discs.

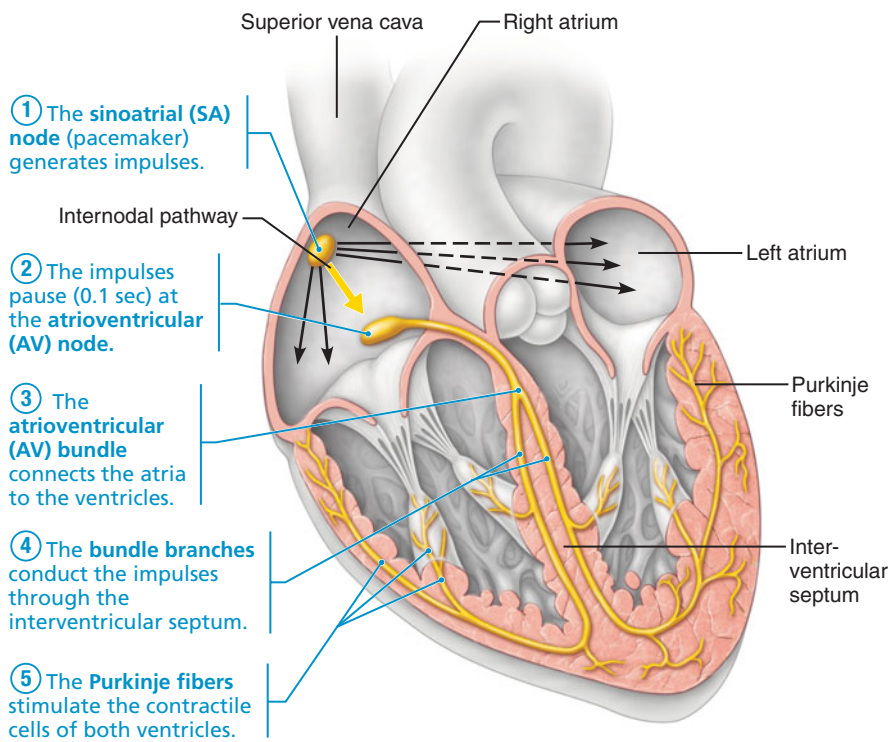


FIGURE 19.14 The intrinsic conducting system of the heart. This system consists of specialized cardiac muscle cells, not nerves.

From the SA node, impulses spread in a wave along the cardiac muscle fibers of the atria, signaling the atria to contract. Some of these impulses travel along an **internodal pathway** to the **atrioventricular (AV) node** in the inferior part of the interatrial septum, where they are delayed for a fraction of a second (Figure 19.14, ②). After this delay, the impulses race through the **atrioventricular bundle** (formerly, *bundle of His*), which enters the interventricular septum and divides into right and left **bundle branches,** or *crura* ("legs"). About halfway down the septum, the crura become **subendocardial branches,** commonly called **Purkinje** (Purkin′je) **fibers,** which approach the apex of the heart and then turn superiorly into the ventricular walls (Figure 19.14, ③–⑤). This arrangement ensures that the contraction of the ventricles begins at the apex of the heart and travels superiorly, so that ventricular blood is ejected superiorly into the great arteries. The brief delay of the contraction-signaling impulses at the AV node enables the ventricles to fill completely before they start to contract. Because the fibrous skeleton between the atria and ventricles is nonconducting, it prevents impulses in the atrial wall from proceeding directly to the ventricular wall. As a result, only those signals that go through the AV node can continue on.

Examination of the microscopic anatomy of the heart's conducting system reveals that the cells of the nodes and AV bundle are small, but otherwise typical, cardiac muscle cells. Each Purkinje fiber, by contrast, is a long row of special, large-diameter, barrel-shaped cells called *Purkinje myocytes.* These muscle cells contain relatively few myofilaments because they are adapted more for conduction than for contraction. Their large diameter maximizes the speed of impulse conduction. Purkinje fibers are located in the deepest part of the ventricular endocardium, between the endocardium and myocardium layers.

DAMAGE TO THE CONDUCTING SYSTEM

Because the atria and ventricles are insulated from each other by the electrically inert fibrous skeleton, the only route for impulse transmission from the atria to the ventricles is through the AV node and AV bundle. Therefore, damage to either of these, called a **heart block,** interferes with the ability of the ventricles to receive the pacing impulses.

Without these signals, the ventricles beat at an intrinsic rate that is slower than that of the atria—and too slow to maintain adequate circulation. In such cases, an artificial pacemaker set to discharge at the appropriate rate is usually implanted. For other conditions involving damage to the conducting system, see the section on disorders of the heart (p. 573).

Innervation

Although the heart's inherent rate of contraction is set by the SA node, this rate can be altered by extrinsic neural controls

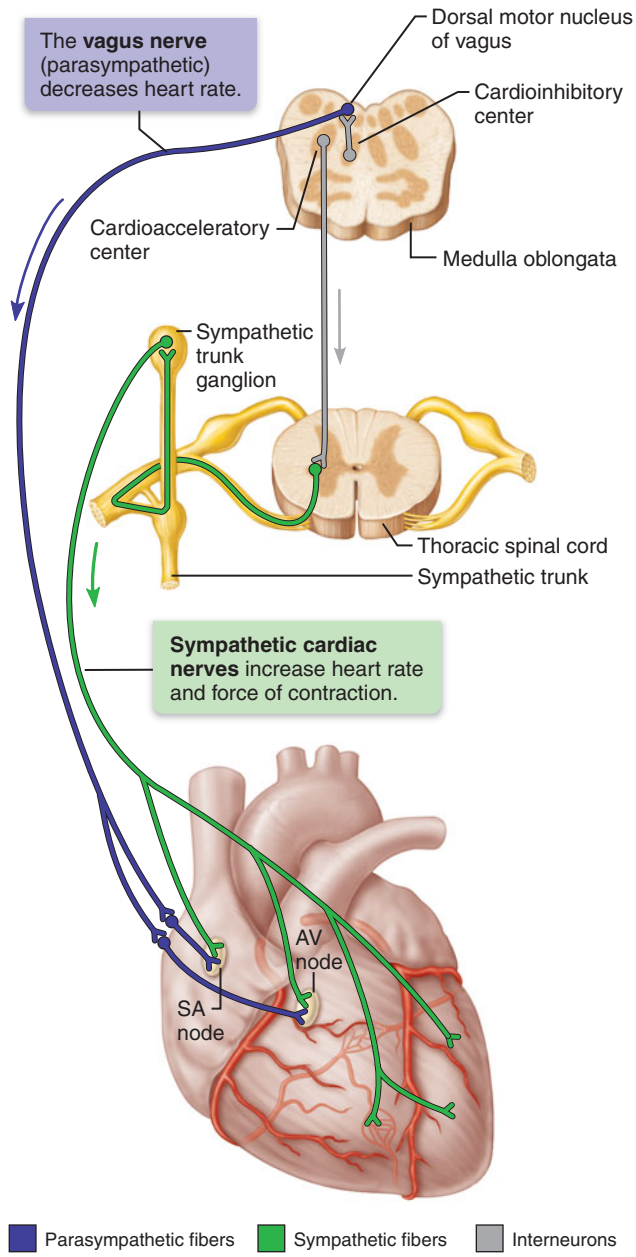


FIGURE 19.15 Autonomic innervation of the heart.

(Figure 19.15). The nerves to the heart consist of *visceral sensory* fibers, *parasympathetic* fibers that slow heart rate, and *sympathetic* fibers that increase the rate and force of heart contractions. The parasympathetic nerves arise as branches of the vagus nerve in the neck and thorax, whereas the sympathetic nerves travel to the heart from the cervical and upper thoracic chain ganglia (shown in Figure 15.5, p. 468). All nerves serving the heart pass through the cardiac plexus on the trachea before entering the heart. Parasympathetic innervation of the heart, which influences heart rate, is restricted to the SA and AV nodes and the coronary arteries. Sympathetic fibers innervate these same areas and also project to the cardiac musculature throughout the heart. These sympathetic fibers affect both rate and strength of contraction.

As mentioned previously (see p. 385), the autonomic input to the heart is controlled by *cardiac centers* in the reticular

formation of the medulla of the brain. In the medulla, the **cardioinhibitory center** influences parasympathetic neurons, whereas the **cardioacceleratory center** influences sympathetic neurons. These medullary cardiac centers, in turn, are influenced by such higher brain regions as the hypothalamus, periaqueductal gray matter, amygdala, and insular cortex (discussed in Chapter 13).

check your understanding

9. What is the significance of the gap junctions in the intercalated discs?
10. What is the pacemaker of the heart, and where is it located? What type of tissue forms the structures of the conducting system of the heart?
11. How do the autonomic nerves that innervate the heart influence heart function?

For answers, see Appendix B.

BLOOD SUPPLY TO THE HEART

➤ Describe the locations of the coronary arteries and cardiac veins on the heart surface.

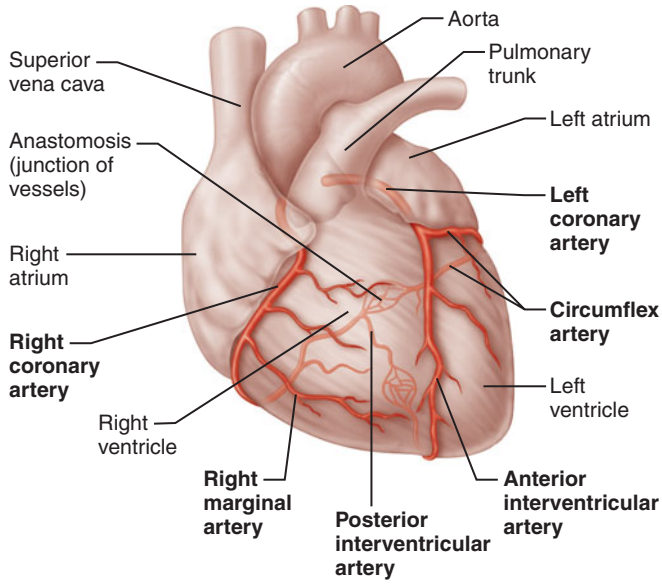
Although the heart is filled with blood, the heart walls are too thick to obtain much nutrition by diffusion from this contained blood. Instead, blood supply to the muscular walls and tissues of the heart is delivered by the right and left *coronary arteries* **(Figure 19.16a)**. These systemic arteries arise from the base of the aorta and run in the coronary sulcus.

The **left coronary artery** arises from the left side of the aorta, passes posterior to the pulmonary trunk, then divides into two branches: the *anterior interventricular* and *circumflex arteries.* The **anterior interventricular artery** descends in the anterior interventricular sulcus toward the apex of the heart, sending branches into the interventricular septum and onto the anterior walls of both ventricles. The **circumflex artery** follows the coronary sulcus posteriorly and supplies the left atrium and the posterior part of the left ventricle.

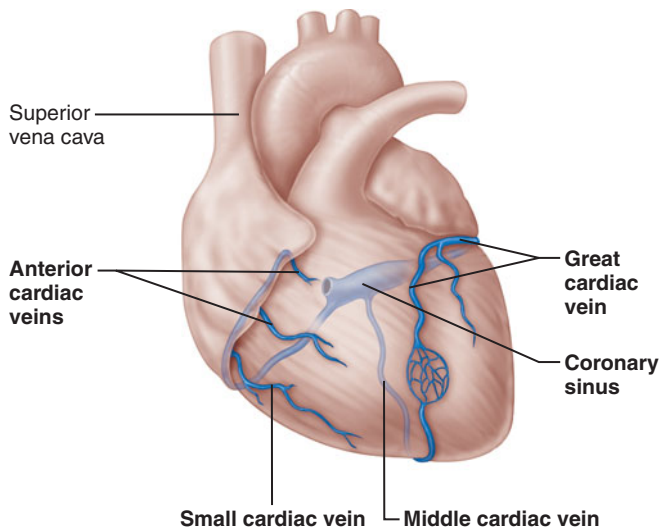
The **right coronary artery** emerges from the right side of the aorta and descends in the coronary sulcus on the anterior surface of the heart, between the right atrium and right ventricle. At the inferior border of the heart, it branches to form the **marginal artery.** Continuing into the posterior part of the coronary sulcus, the right coronary artery gives off a large branch called the **posterior interventricular artery** in the posterior interventricular sulcus. Overall, the branches of the right coronary artery supply the right atrium and almost all of the right ventricle.

The arrangement of the coronary arteries varies considerably among individuals. For example, in about 15% of people, the left coronary artery gives rise to *both* interventricular arteries. In other people (4%), a single coronary artery emerges from the aorta and supplies the entire heart.

Cardiac veins, which carry deoxygenated blood from the heart wall into the right atrium, also occupy the sulci on the heart surface (Figure 19.16b). The largest of these veins, the **coronary sinus,** occupies the posterior part of the coronary sulcus and returns almost all the venous blood from the heart to the right atrium. Draining into the coronary sinus are three large tributaries: the **great cardiac vein** in the anterior interventricular sulcus, the **middle cardiac vein** in the posterior interventricular sulcus, and the **small cardiac vein** running along the heart's inferior right margin. The anterior surface of the right ventricle contains several horizontal **anterior cardiac veins** that empty directly into the right atrium.



(a) The major coronary arteries



(b) The major cardiac veins

FIGURE 19.16 Coronary circulation. In both illustrations, the lighter-colored vessels lie more posteriorly on the heart.

DISORDERS OF THE HEART

➤ Define coronary artery disease, heart failure, and atrial and ventricular fibrillation.

Coronary Artery Disease

Atherosclerosis is an accumulation of fatty deposits in the inner lining of the body's arteries that can block blood flow through these arteries (see p. 610 for details). When atherosclerosis affects the coronary arteries, it leads to **coronary artery disease (CAD),** in which the arteries supplying the heart wall are narrowed or blocked. A common symptom of this disease is **angina pectoris** (an-ji′nah pek′tor-us; "choked chest"), thoracic pain caused by inadequate oxygenation of heart muscle cells, which weaken but do not die. Although the pain of angina usually results directly from tissue hypoxia, it can also result from stress-induced spasms of the atherosclerotic coronary arteries. Angina attacks occur most often during exercise, when the vigorously contracting heart may demand more oxygen than the narrowed coronary arteries can deliver. The onset of angina should be considered a warning sign of other, more serious conditions. Prevention and treatments for CAD are described in A Closer Look on p. 574.

If the blockage of a coronary artery is more complete or prolonged, the oxygen-starved cardiac muscle cells die—a condition called **myocardial infarction,** or a heart attack. Few experiences are more frightening than a heart attack: A sharp pain strikes with lightning speed through the chest (and sometimes the left arm and left side of the neck) and does not subside. Death from cardiac arrest occurs almost immediately in about one-third of cases. Heart attacks kill either directly (due to severe weakening of the heart) or indirectly (due to heart-rhythm disruptions caused by damage to the conducting system). Because cardiac muscle tissue has no satellite cells as skeletal muscle does, it does not regenerate effectively, and thus myocardial damage from a heart attack is irreversible.

For many people a painless but fatal heart attack is the first and only clear symptom of coronary artery disease. Such individuals are victims of **silent ischemia,** a condition in which blood flow to the heart is interrupted often, exactly as in angina, but without any pain to provide warning. Silent ischemia, which can also occur in some heart-attack survivors, can be detected by measuring heart rhythm through electrocardiography (ECG) during exercise, a procedure called a graded exercise test (or treadmill test).

Heart Failure

Heart failure is a progressive weakening of the heart as it fails to keep pace with the demands of pumping blood and thus cannot meet the body's need for oxygenated blood. This condition may be due to weakened ventricles (damaged, for example, by a heart attack), to failure of the ventricles to fill completely during diastole (for example, as a result of mitral valve stenosis), or to overfilling of the ventricles (for example, because of stenosis or insufficiency of the aortic valve). Most commonly, however, congestive heart failure is the cause of this progressive weakening.

In **congestive heart failure,** the heart enlarges greatly while its pumping efficiency progressively declines. This condition affects 5 million Americans and is increasing in frequency. Its cause is unknown. One hypothesis is that it may involve a destructive positive feedback loop: An initially weakened heart causes the sympathetic nervous system to stimulate the heart to pump harder. This increased demand further weakens the heart, which again causes stimulation of the sympathetic system, and so on.

A condition called **pulmonary arterial hypertension** is the enlargement and sometimes ultimate failure of the right ventricle resulting from elevated blood pressure in the pulmonary circuit. A blockage or constriction of the vessels in the lungs increases resistance to blood flow, which increases blood pressure and forces the right ventricle to work harder. Whereas acute cases of pulmonary hypertension may develop suddenly from an embolism in the pulmonary vessels, chronic cases are usually associated with chronic lung diseases such as emphysema.

Disorders of the Conduction System

This section covers a type of *arrhythmia*—a variation from the normal rhythm of the heartbeat—called fibrillation.

In **ventricular fibrillation,** the ventricles are unable to pump blood into the arteries because rapid, random firing of electrical impulses within ventricular cardiac muscle prevents coordinated contraction of the ventricle. The fibrillating ventricles can be likened to a quivering bag of worms. Ventricular fibrillation results from a crippled conducting system and is the most common cause of cardiac arrest and sudden death in patients with hearts damaged by coronary artery disease.

In **atrial fibrillation,** multiple waves of impulses circle within the atrial myocardium, randomly stimulating the AV node, which then signals the ventricles to contract quickly and irregularly. The resulting lack of smooth movement of blood through the heart can promote the formation of clots, parts of which can break off, reach the brain, and cause strokes. The fibrillations are usually discontinuous, occurring in episodes characterized by palpitations (sensations of an unduly quick and irregular heartbeat), anxiety, fatigue, and shortness of breath. The cause of this condition, which affects 5% of individuals over age 65 and 10% over 75, is unknown, but it is often associated with coronary artery or heart-valve disease. Intermittent rhythm disturbances can be identified by ECG monitoring during normal activity, a procedure called an ambulatory ECG or Holter monitoring. Treatment typically involves drug therapy and administration of anticoagulants to prevent clotting.

check your understanding

12. What vessel supplies blood to the left ventricle?
13. What are the risk factors for coronary artery disease? What is one common symptom of this disorder?

For answers, see Appendix B.

THE HEART THROUGHOUT LIFE

- Explain how the heart develops, and describe some congenital heart defects.
- List some effects of aging on the heart.

a closer look

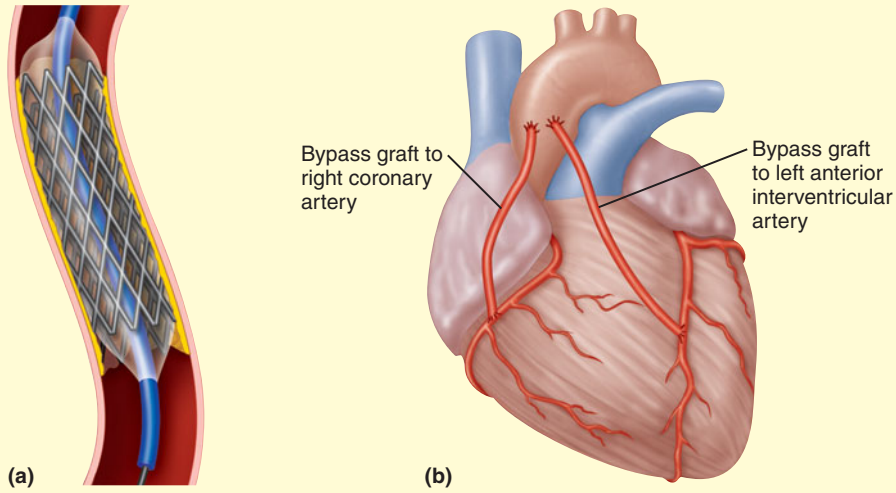
Coronary Artery Disease

Out for her morning walk, Barbara, 63, started to feel sharp, squeezing pains in her chest. The pain subsided when she sat down and rested. Her immediate thought was heart attack. A trip to the emergency room showed that fortunately there was no damage to the heart muscle. Barbara had suffered an angina attack, a clear sign of coronary artery disease. Coronary artery disease (CAD) is caused by a buildup of fatty plaque (atherosclerosis) in the coronary arteries, resulting in decreased blood supply to the myocardium of the heart and diminished heart function.

Once coronary artery disease is detected, the first step is to reduce risk factors through lifestyle modification. Risk factors include high blood pressure, smoking, high cholesterol levels, diabetes, inactivity, and family history of the disease. Changes in diet and exercise routine are primary. Medication may be required to control risk factors, decrease workload on the heart, and increase blood flow through coronary vessels.

When medication and lifestyle changes are inadequate to maintain blood supply to the heart, surgical interventions may be needed. For many years, coronary artery bypass graft (CABG) was the only intervention available. The first bypass occurred in 1967 and was a standard, although risky, treatment requiring major surgery. Other, less invasive treatments have been developed in the last 25 years.

Angioplasty (percutaneous transluminal coronary angioplasty, PTCA), developed in the late 1970s, involves inserting a catheter into the patient's arm or leg and threading it through the arteries until it enters the blocked coronary artery. When the catheter reaches the blockage, a balloon located on the tip of the catheter is inflated to exert pressure on the plaque-filled wall of the vessel. This pressure compresses the plaque and increases the diameter of the vessel lumen, thus improving blood flow to the myocardium.



(a) Angioplasty with insertion of a stent. (b) Coronary artery bypass graft (CABG) performed on two vessels; double bypass surgery.

This procedure revolutionized the treatment of coronary artery disease. However, in some cases angioplasty weakened the wall of the coronary vessel, causing collapse of the coronary artery after the balloon was deflated. In 1993 the use of stents to prevent arterial collapse and reblockage was approved by the FDA. Following the opening of the artery with balloon angioplasty, a metal mesh tube called a bare metal stent (BMS) is inserted into the artery at the site of blockage (see figure a). The stent serves as scaffolding to hold the arterial lumen open. Anticlotting medications are prescribed for at least 6 months after stent insertion to decrease the risk of thrombosis.

Stents improved the success of angioplasty; however, restenosis, the thickening of the vessel wall in response to the stent, continues to be a challenge for some patients. The endothelial cells and smooth muscle cells of the wall grow and divide. This, along with inflammation, results in thickening of the arterial wall and diminished blood flow through the artery.

Drug-eluting stents, first approved in 2003, slowly release drugs that disrupt various portions of the cell cycle. These drugs prevent cells in the vessel wall from proliferating in response to the stent and thus prevent restenosis.

These catheterization procedures are much less invasive than coronary bypass surgery and are appropriate treatments for many individuals. However, some patients may have a better outcome with a coronary artery bypass graft (CABG) procedure. In coronary bypass, vessels from another part of the body are used to reroute blood to the heart. A portion of a vessel is removed from its original location and grafted between the aorta and the heart wall, thus supplying an alternate route for blood to the heart muscle (see figure b). Vessels commonly used for bypass surgery are the internal thoracic artery, which normally serves the anterior body wall; the saphenous vein from the leg; and the radial artery in the forearm.

Most important, the long-term success of any of these treatments depends on lifestyle changes by the patient. Better knowledge of lifestyle factors and improved medical intervention, both pharmacological and surgical, have influenced the prevention of and the prognosis for living with coronary artery disease.

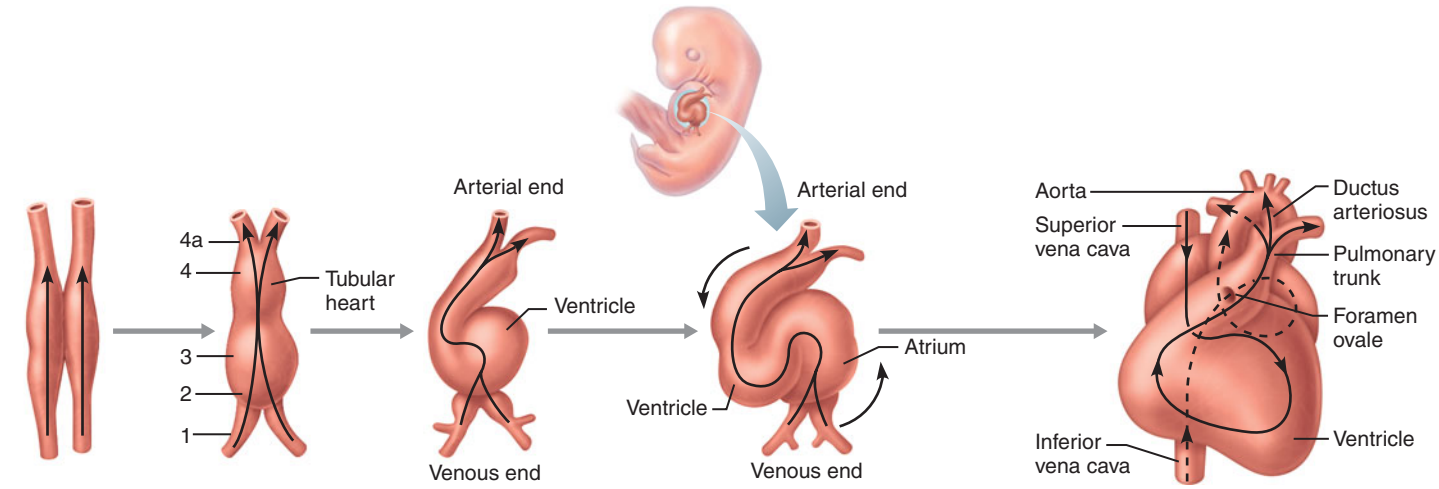


FIGURE 19.17 Heart development. Ventral views, with the cranial direction toward the top of the figures. Arrows show the direction of blood flow. Days are approximate. In (b), 1 is the sinus venosus; 2, the atrium; 3, the ventricle; 4, the bulbus cordis; and 4a, the truncus arteriosus.

Development of the Heart

An understanding of heart development is clinically important because congenital abnormalities of the heart account for nearly half of all deaths from birth defects. One of every 150 newborns has some congenital heart defect.

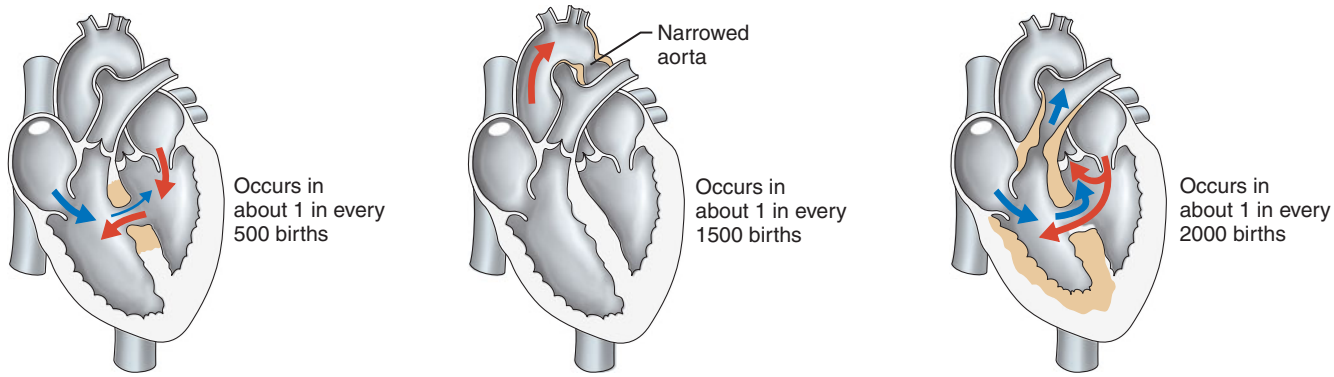
As explained in Chapter 18 (p. 550), all blood vessels begin as condensations of mesodermal mesenchyme called blood islands. Subsequently, the blood islands destined to become the heart form in the splanchnic mesoderm around the future head and neck of the embryonic disc. The heart folds neatly into the thorax region when the flat embryonic disc lifts up off the yolk sac to assume its three-dimensional body shape around day 20 or 21 (this folding is described on p. 55).

When the embryonic heart first reaches the thorax, it is a pair of endothelial tubes in the body midline. These tubes fuse into a single tube on about day 20 **(Figure 19.17a)**. The heart starts pumping about day 22, by which time four bulges have developed along the heart tube (Figure 19.17b). These bulges are the earliest heart chambers and are unpaired. From tail to head, following the direction of blood flow, the four chambers are the *sinus venosus, atrium, ventricle,* and *bulbus cordis:*

1. **Sinus venosus** (ven-o′sus; "of the vein"). This chamber, which initially receives all blood from the veins of the embryo, will become the smooth-walled part of the right atrium and the coronary sinus; it also gives rise to the sinoatrial node. Furthermore, recent evidence indicates that the sinus venosus also contributes to the back wall of the *left* atrium (which mostly derives from the bases of the developing pulmonary veins).
2. **Atrium.** This embryonic chamber eventually becomes the ridged parts of the right and left atria—specifically, the parts lined by pectinate muscles.
3. **Ventricle.** The strongest pumping chamber of the early heart, the embryonic ventricle gives rise to the *left* ventricle.
4. **Bulbus cordis.** This chamber and its most cranial extension, the *truncus arteriosus,* give rise to the pulmonary trunk and first part of the aorta. The bulbus cordis also gives rise to the *right* ventricle.

At the time these four chambers appear, the heart starts bending into an S shape (Figure 19.17c and d). The ventricle moves caudally and the atrium cranially, assuming their adult positions. This bending occurs because the ventricle and bulbus cordis grow quickly, and the heart is unable to accommodate elongation within the confines of the pericardial sac.

During month 2 of development, the heart divides into its four definitive chambers by the formation of its midline septum and valves. These structures develop from cardiac cushions, which are regional thickenings of the endocardium, the inner lining of the heart wall. For example, most of the interatrial septum forms by growing caudally from the heart's roof, and most of the interventricular septum forms by growing cranially from the heart's apex. Additionally, neural crest cells (pp. 52 and 56) migrate into the area where atrium meets ventricle. Here, these cells contribute to the developing heart valves and to the bases of the pulmonary trunk and ascending aorta, the great arteries that form by the splitting of the bulbus cordis. These month-2 events are so complex that perfect orchestration does not always occur, and developmental defects result.



(a) Ventricular septal defect. The superior part of the interventricular septum fails to form; thus, blood mixes between the two ventricles. More blood is shunted from left to right because the left ventricle is stronger.

(b) Coarctation of the aorta. A part of the aorta is narrowed, increasing the workload of the left ventricle.

(c) Tetralogy of Fallot. Multiple defects (*tetra* = four): (1) Pulmonary trunk too narrow and pulmonary valve stenosed, resulting in (2) hypertrophied right ventricle; (3) ventricular septal defect; (4) aorta opens from both ventricles.

FIGURE 19.18 Congenital heart defects. The defects are shown according to relative frequency of occurrence, from the most frequent to the least. Tan areas indicate the locations of the defects.

Most other details of heart development are beyond the scope of this book, but one thing should be mentioned: The two atria remain interconnected by a hole in the interatrial septum—the foramen ovale—until birth, at which time this hole closes to become the fossa ovalis (Figure 19.5c). The foramen ovale plays an important role in blood circulation before birth, and is discussed in the section on fetal circulation in the next chapter (p. 611).

Figure 19.18 illustrates and explains the common congenital heart defects, almost all of which can be traced to month 2 of development. The most common of these is a **ventricular septal defect** (Figure 19.18a), in which the superior (cranial) region of the interventricular septum fails to form, leaving a hole between the two ventricles. As you study this and the other defects in the figure, note that they produce two basic kinds of effects in newborns: Either inadequately oxygenated blood reaches the body tissues (because oxygen-poor systemic blood mixes with oxygenated pulmonary blood), or the ventricles labor under an increased workload (because of narrowed valves and vessels), or both effects occur. Modern surgical techniques can usually correct these congenital defects.

The Heart in Adulthood and Old Age

In the absence of congenital heart problems, the resilient heart usually functions well throughout life. In individuals who exercise regularly and vigorously, the heart gradually adapts to the increased demand by increasing in strength and size. Aerobic exercise also helps clear fatty deposits from the walls of the coronary vessels, thereby retarding the process of atherosclerosis. Barring some chronic illness, this beneficial response to exercise persists into old age.

Age-related changes that affect the heart include the following:

1. **Hardening and thickening of the cusps of the heart valves.** These changes occur particularly in those valves subjected to the highest blood pressures (mitral and aortic valves). Thus, abnormal heart sounds are more common in elderly people.
2. **Decline in cardiac reserve.** Although the passage of years seems to cause few changes in the resting heart rate, the aged heart is slower at increasing its output to pump more blood. Sympathetic control of the heart becomes less efficient, and heart rate gradually becomes more variable. Maximum heart rate declines, although this problem is much less severe in seniors who are physically active.
3. **Fibrosis of cardiac muscle.** As one ages, more and more cardiac muscle cells die and are replaced by fibrous scar tissue. This fibrosis, which is much more extensive in men than in women, lowers the maximum amount of blood that the heart can pump per unit time. Also, in conjunction with the aging of muscle-cell membranes, fibrosis hinders the initiation and transmission of contraction-signaling impulses, leading to abnormal heart rhythms and other conduction problems.

check your understanding

14. How would incomplete formation of the interventricular septum alter blood flow through the heart?
15. What chamber of the heart is formed from the embryonic ventricle?
16. What is the single most important factor for maintaining a healthy heart throughout life?

For answers, see Appendix B.

RELATED CLINICAL TERMS

ASYSTOLE (a-sis′to-le; *a* = without) Failure of the heart to contract.

CARDIAC CATHETERIZATION Diagnostic procedure in which a fine catheter (tube) is passed from a blood vessel at the body surface to the heart. Blood-oxygen content, blood pressure, and blood flow are measured, and heart structures can be visualized. Findings help detect problems with the heart valves, heart deformities, and other cardiac malfunctions.

CARDIOMYOPATHY (kar′de-o-mi-op′ah-the) Any disease of the myocardium that weakens the heart's ability to pump.

ECHOCARDIOGRAPHY (ek″o-kar″de-og′rah-fe) Ultrasound imaging of the heart; used not only for imaging but also for measuring blood flow through the heart.

ENDOCARDITIS (en″do-kar-di′tis) Inflammation of the endocardium, usually confined to the endocardium of the heart valves. Endocarditis often results from infection by bacteria that have entered the bloodstream, but may result from fungal infections or an autoimmune response. Drug addicts may develop endocarditis by injecting themselves with contaminated needles. Additionally, the bacteria can enter during routine dentistry and ear-piercing procedures.

HYPERTROPHIC CARDIOMYOPATHY Inherited condition in which the wall of the left ventricle (especially the interventricular septum) is abnormally thick and composed of disorganized muscle cells. Affects 1 of every 500 people. Because the lumen of the left ventricle is too small to hold all the atrial blood returning to it, blood pressure in the pulmonary circuit is elevated. Additionally, the grossly thickened myocardium becomes ischemic because it cannot get enough oxygen from the coronary vessels that supply it. Exercising can lead to fainting, chest pain, or even sudden death.

MYOCARDITIS (mi′o-kar-di′tis) Inflammation of the heart's myocardium. Sometimes follows an untreated streptococcal infection in children; may be extremely serious because it can weaken the heart and impair its ability to pump blood.

PERCUSSION Tapping the thorax or abdomen wall with the fingertips and using the nature of the resulting sounds to estimate the location, density, and size of the underlying organs. Percussion of the thoracic wall can be used to estimate the size of a patient's heart.

CHAPTER SUMMARY

You can use the following media study tool for additional help when you review specific key topics in Chapter 19.

PAL = **Practice Anatomy Lab™**

1. The heart is a double pump whose right side pumps blood to the lungs for oxygenation and whose left side pumps blood throughout the body to nourish body tissues; that is, the right side is the pump of the pulmonary circuit, and the left side is the pump of the systemic circuit.
2. The four chambers of the heart are right and left atria (receiving chambers) and right and left ventricles (main pumping chambers).

Location and Orientation Within the Thorax (pp. 556–558)

3. The cone-shaped human heart, about the size of a fist, lies obliquely within the mediastinum. Its apex points anteriorly and to the left. Its base is its posterior surface.
4. From an anterior view, the heart is said to have four corners. The locations of its four corner points are described on p. 556.

Structure of the Heart (pp. 558–563)

Coverings (p. 558)

5. The pericardium encloses the heart. It consists of a superficial layer (fibrous pericardium and parietal layer of the serous pericardium), and a deeper layer that covers the heart surface (visceral layer of the serous pericardium, or epicardium). The pericardial cavity, between the two layers, contains lubricating serous fluid.

Layers of the Heart Wall (pp. 558–559)

6. The layers of the heart wall, from external to internal, are the epicardium, the myocardium (which consists of cardiac muscle tissue), and the endocardium (endothelium and connective tissue).

Heart Chambers (pp. 559–563)

7. On the external surface of the heart, several sulci separate the four heart chambers: the coronary sulcus and the anterior and posterior interventricular sulci. Internally, the right and left sides of the heart are separated by the interatrial and interventricular septa.
8. The right atrium has the following features: right auricle and pectinate muscles anteriorly; a smooth-walled posterior part; crista terminalis; openings of the coronary sinus and the superior and inferior venae cavae; SA and AV nodes; and fossa ovalis.
9. The right ventricle contains the following features: trabeculae carneae, papillary muscles, chordae tendineae, and right atrioventricular (tricuspid) valve. The pulmonary valve lies at the base of the pulmonary trunk.
10. The left atrium has a large, smooth-walled, posterior region into which open the four pulmonary veins. Anteriorly, its auricle is lined by pectinate muscles.
11. The left ventricle, like the right ventricle, contains papillary muscles, chordae tendineae, trabeculae carneae, and an atrioventricular valve (mitral). The aortic valve lies at the base of the aorta.

Heart Valves (pp. 563–576)

Valve Structure (p. 563)

12. The four heart valves are the atrioventricular valves (tricuspid and mitral) and the semilunar valves (aortic and pulmonary). All except the mitral have three cusps.
13. The fibrous skeleton of the heart surrounds the valves between the atria and ventricles. It anchors the valve cusps, is the point of insertion of the heart musculature, and blocks the direct spread of electrical impulses from the atria to the ventricles.

Valve Function (p. 563)

14. The atrioventricular valves prevent backflow of blood into the atria during contraction of the ventricles. The pulmonary and aortic semilunar valves prevent backflow into the ventricles during relaxation of the ventricles.

Heart Sounds (pp. 563–565)

15. Using a stethoscope, physicians can listen to the sounds produced by the closing atrioventricular and semilunar valves. Each valve is best heard near a different heart corner on the anterior chest wall (see Figure 19.9).

Pathway of Blood Through the Heart (pp. 566–568)

16. A drop of blood circulates along the following path: right atrium to right ventricle to pulmonary circuit to left atrium to left ventricle to systemic circuit to right atrium.

17. In each heartbeat, both atria contract together, followed by simultaneous contraction of both ventricles.

18. The wall of the left ventricle is thicker than that of the right ventricle, resulting in higher pressure in the systemic circuit.

Cardiac Muscle Tissue (pp. 568–569)

19. Cardiac muscle tissue forms the thick myocardium of the heart wall. It contains cardiac muscle cells and connective tissues that surround these cells.

20. Cardiac muscle cells are short, branching, striated cells with one or two central nuclei. They contain myofibrils made of typical sarcomeres. Cardiac muscle contracts by the sliding filament mechanism.

21. Adjacent cardiac cells are connected by intercalated discs to form cardiac fibers. Intercalated discs contain two types of junctions: desmosome-like fasciae adherens and gap junctions. Gap junctions between cells allow the direct transmission of an electrical impulse through the entire network of cardiac muscle cells.

22. Cardiac muscle cells have many mitochondria and depend on aerobic respiration to form ATP. They are very resistant to fatigue.

23. Contraction of cardiac muscle is triggered by Ca^{2+}. Some of this calcium comes from the extracellular fluid. The sarcoplasmic reticulum and T tubules are somewhat simpler than those of skeletal muscle.

24. Cardiac muscle cells contract with their own inherent rhythm, the basis of the heartbeat.

PAL **Human Cadaver/Cardiovascular System/Heart**

Conducting System and Innervation (pp. 570–572)

Conducting System (pp. 570–571)

25. The conducting system of the heart is an interconnected series of specialized cardiac muscle cells that initiates each heartbeat, sets the basic rate of the heartbeat, and coordinates the contraction of the heart chambers. The impulse that signals heart contraction travels from the SA node (the pacemaker) through the atrial myocardium and internodal pathway to the AV node, to the atrioventricular bundle, bundle branches, Purkinje fibers, and the ventricular musculature.

Innervation (pp. 571–572)

26. Heart innervation consists of visceral sensory fibers, heart-slowing vagal parasympathetic fibers, and sympathetic fibers that increase heart rate and force of contraction.

Blood Supply to the Heart (p. 572)

27. The coronary vessels that supply the heart wall are located in the coronary sulcus and interventricular sulci. The right and left coronary arteries branch from the base of the aorta to supply the heart wall. Venous blood, collected by the cardiac veins (great, middle, small, and anterior), empties into the coronary sinus, which opens into the right atrium.

Disorders of the Heart (pp. 572–574)

28. Coronary artery disease is caused by atherosclerotic blockage of the coronary arteries, and can lead to angina pectoris and myocardial infarction (heart attack). Heart failure is a weakening of the heart, resulting in its inability to pump blood fast enough to meet the body's needs. Atrial and ventricular fibrillations are rapid, irregular, and uncoordinated contractions of the respective heart chambers, resulting from damage to the conducting system.

The Heart Throughout Life (pp. 573–576)

Development of the Heart (pp. 573–576)

29. The heart develops from splanchnic mesoderm around the head and neck of the embryonic disc. When the heart folds into the thorax, it is a double tube that soon fuses into one and starts pumping blood (day 22). It soon bends into an S shape. Its four earliest chambers are the sinus venosus, atrium, ventricle, and bulbus cordis.

30. The four final heart chambers are defined during month 2 through the formation of valves and dividing walls. Failures in this complex process account for most congenital defects of the heart (see Figure 19.18).

The Heart in Adulthood and Old Age (p. 576)

31. Age-related changes in the heart include hardening and thickening of the valve cusps, decline in cardiac reserve, fibrosis of cardiac muscle, and atherosclerosis.

REVIEW QUESTIONS

Multiple Choice/Matching Questions

For answers, see Appendix B.

1. The most external part of the pericardium is the (a) parietal layer of serous pericardium, (b) fibrous pericardium, (c) visceral layer of serous pericardium, (d) pericardial cavity.

2. Which heart chamber forms most of the heart's inferior surface? (a) right atrium, (b) right ventricle, (c) left atrium, (d) left ventricle.

3. How many cusps does the right atrioventricular valve have? (a) two, (b) three, (c) four.

4. The sequence of contraction of the heart chambers is (a) random, (b) left chambers followed by right chambers, (c) both atria followed by both ventricles, (d) right atrium, right ventricle, left atrium, left ventricle.

5. The middle cardiac vein runs with which artery? (a) marginal artery, (b) aorta, (c) coronary sinus, (d) anterior interventricular artery, (e) posterior interventricular artery.

6. The base of the heart (a) is its posterior surface, (b) lies on the diaphragm, (c) is the same as its apex, (d) is its superior border.

7. Which of the following is an *in*correct statement about the crista terminalis of the right atrium? (a) It separates the smooth-walled part from the part with pectinate muscles. (b) It is shaped like the letter C. (c) The coronary sinus and inferior vena cava open near its inferior part. (d) It lies mostly in the interatrial septum.
8. The aortic valve closes (a) at the same time the mitral valve closes, (b) just after the atria contract, (c) just before the ventricles contract, (d) just after the ventricles contract.
9. The ventricle of the embryonic heart gives rise to what adult structure(s)? (a) bulbus cordis, (b) both ventricles, (c) left ventricle, (d) the aorta, (e) none of these.
10. Which layer of the heart wall is the thickest? (a) endocardium, (b) myocardium, (c) epicardium, (d) endothelium.
11. The inferior left corner of the heart is located at the (a) second rib slightly lateral to the sternum, (b) third rib at the sternum, (c) sixth rib slightly lateral to the sternum, (d) fifth intercostal space at the midclavicular line.

Short Answer Essay Questions

12. Ben was annoyed when the teaching assistant who ran the discussion section of his anatomy class said that the atria pump blood to the lungs, and the ventricles pump blood throughout the body. Correct this error.
13. Describe the location of the heart within the thorax.
14. Trace a drop of blood through all the heart chambers and heart valves, and through the basic vascular circuits, from the time it enters the left atrium until it returns to the left atrium again.
15. (a) Name the elements of the heart's conducting system in order, beginning with the pacemaker. (b) Is the conducting system made of nerves? Explain. (c) What are the functions of this conducting system?
16. Sketch the heart and draw all the coronary vessels in their correct locations. (Alternatively, you could locate these vessels on a realistic diagram of the heart.)
17. On a diagram of a frontally sectioned heart, indicate the location of the fibrous skeleton.
18. When you view a heart's *anterior* surface, which of its four chambers appears largest?
19. Make a drawing of the adult heart and the associated large vessels. Then color and label the adult regions that derive from each *embryonic* heart chamber: (a) sinus venosus, (b) embryonic atrium, (c) embryonic ventricle, (d) bulbus cordis.
20. How do the right and left ventricles differ structurally, and how do these structural differences reflect functional differences?
21. Which is more resistant to fatigue, cardiac muscle or skeletal muscle? What is the anatomical basis for this difference, and why is it important?
22. Describe the structure and function of an intercalated disc.
23. Compare and contrast the structure of cardiac muscle tissue with skeletal muscle tissue.

CRITICAL REASONING & CLINICAL APPLICATION QUESTIONS

1. After studying Figure 19.17, classify the three congenital heart defects depicted according to whether they produce (1) mixing of oxygenated and unoxygenated blood, (2) increased workload for the ventricles, or (3) both of these problems.
2. Ms. Hamad, who is 73 years old, is admitted to the coronary care unit of a hospital with a diagnosis of left ventricular failure resulting from a myocardial infarction. Her heart rhythm is abnormal. Explain what a myocardial infarction is, how it is likely to have been caused, and why the heart rhythm is affected.
3. You have been called on to demonstrate where to listen for heart sounds. Explain where on the chest wall you would place a stethoscope to listen for (a) incompetence of the aortic valve and (b) stenosis of the mitral valve.
4. After a man was stabbed in the chest, his face became blue and he lost consciousness from lack of oxygenated blood to the brain. The diagnosis was cardiac tamponade rather than severe blood loss through internal bleeding. What is cardiac tamponade, how did it cause the observed symptoms, and how is it treated?
5. A heroin addict felt tired, weak, and feverish, with vague pains and aches. Finally, he went to a doctor, terrified that he had AIDS. Instead, the doctor found an abnormal heart sound, and the final diagnosis was not AIDS but endocarditis. What is the most likely way that this patient contracted endocarditis?
6. Another patient had an abnormal heart sound that indicated a stenotic valve. Define this condition, and contrast it with an incompetent heart valve.
7. Assume that the four corner points of a patient's heart, as revealed on an X-ray film, relate to the rib cage as follows: second right rib at sternum, tip of xiphoid process, seventh intercostal space lateral to the left nipple, and second left rib at sternum. Are the size and position of this patient's heart normal?
8. During a lethal heart attack, a blood clot lodges in the first part of the circumflex branch of the left coronary artery, blocking blood flow through this vessel. What regions of the heart will become ischemic and die?
9. Blood from the ventricles is "squeezed" out of the heart from the apex toward the great arteries. What features of the heart contribute to this sequence of contraction?