18

The Cardiovascular System: The Heart

WHY THIS
MATTERS

In this chapter, you will learn that

The heart pumps blood through the pulmonary and systemic circuits

starting with

then looking at

18.1 Anatomy of the heart

Physiology of the heart

then asking

starting with

18.2 Why does the heart have valves?

18.3 What path does blood take through the heart?

18.4 How do cardiac muscle fibers differ from skeletal muscle fibers?

18.5 Electrical events

which cause

18.6 Mechanical events

then exploring

18.7 How is pumping regulated?

and finally, exploring

Developmental Aspects of the Heart

Electron micrograph of a mitral valve of the human heart.

O ur ceaselessly beating heart has intrigued people for cen-
turies. The ancient Greeks believed the heart was the
seat of intelligence. Others thought it was the source of
emotions. While these ideas have proved false, we do know that
emotions affect heart rate. When your heart pounds or skips a
beat, you become acutely aware of how much you depend on
this dynamic organ for your very life.

Despite its vital importance, the heart does not work alone.
Indeed, it is only part of the cardiovascular system, which
includes the miles of blood vessels that run through your body.
Day and night, tissue cells take in nutrients and oxygen and
excrete wastes. Cells can make such exchanges only with their
immediate environment, so some means of changing and
renewing that environment is necessary to ensure a continual
supply of nutrients and prevent a buildup of wastes. The car-
diovascular system provides the transport system "hardware"
that keeps blood continuously circulating to fulfill this critical
homeostatic need.

18.1 The heart has four chambers and pumps blood through the pulmonary and systemic circuits
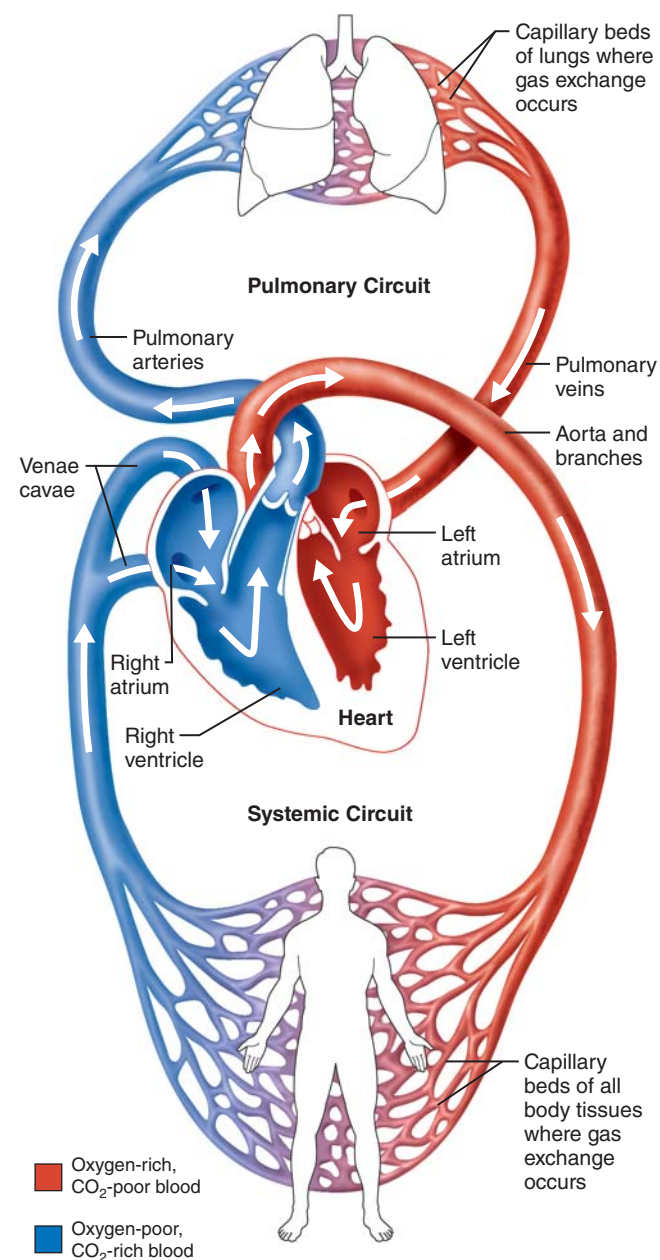
→ **Learning Objectives**

☐ Describe the size, shape, location, and orientation of the heart in the thorax.

☐ Name the coverings of the heart.

☐ Describe the structure and function of each of the three layers of the heart wall.

☐ Describe the structure and functions of the four heart chambers. Name each chamber and provide the name and general route of its associated great vessel(s).

The Pulmonary and Systemic Circuits

Stripped of its romantic cloak, the **heart** is no more than the transport system pump, and the blood vessels are the delivery routes. In fact, the heart is actually two pumps side by side (**Figure 18.1**).

- The *right side* of the heart receives oxygen-poor blood from body tissues and then pumps this blood to the lungs to pick up oxygen and dispel carbon dioxide. The blood vessels that carry blood to and from the lungs form the **pulmonary circuit** (*pulmo* = lung).

- The *left side* of the heart receives the oxygenated blood returning from the lungs and pumps this blood throughout the body to supply oxygen and nutrients to body tissues. The blood vessels that carry blood to and from all body tissues form the **systemic circuit**.
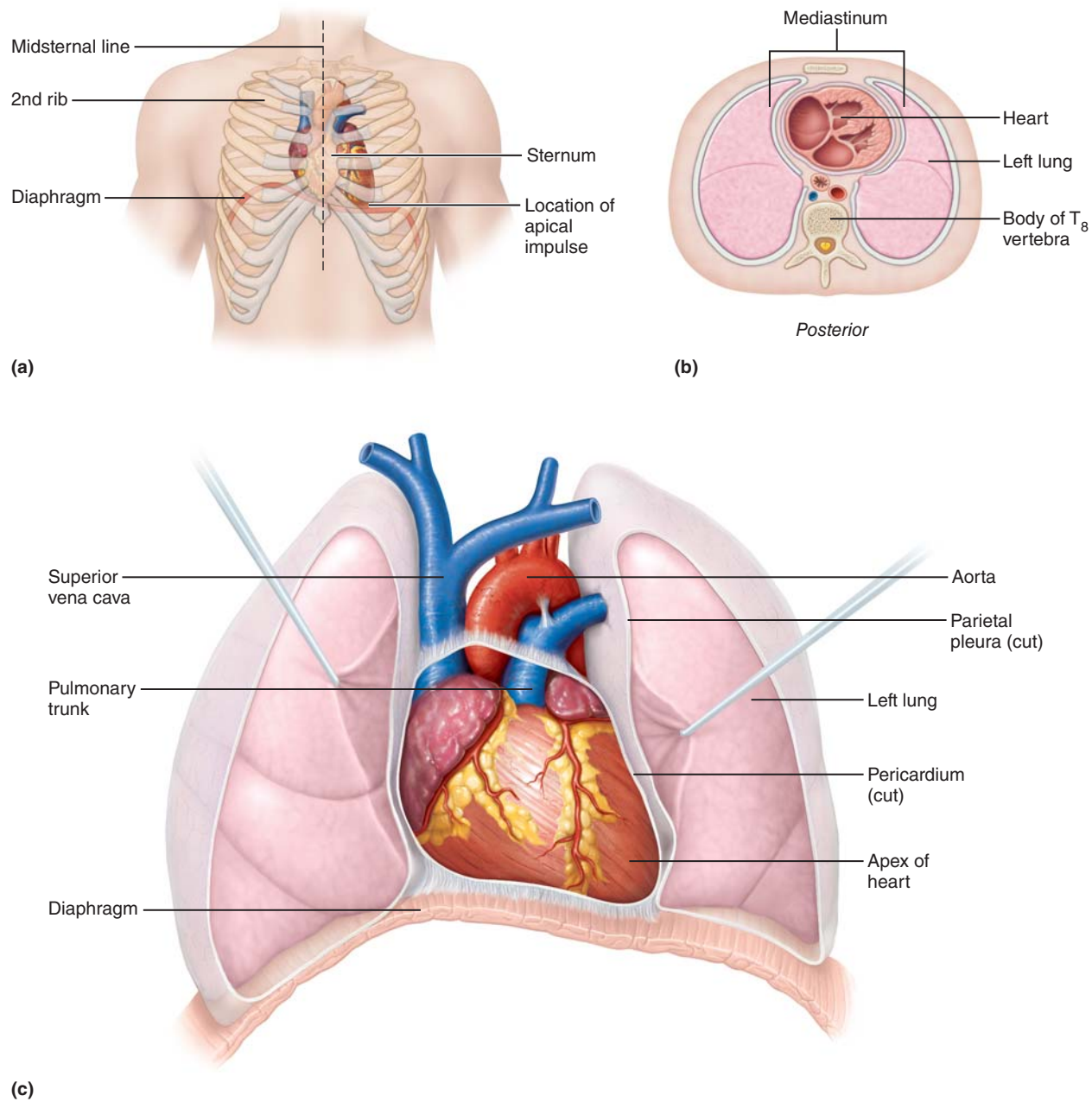
The heart has two receiving chambers, the *right atrium* and *left atrium*, that receive blood returning from the systemic and pulmonary circuits. The heart also has two main pumping chambers, the *right ventricle* and *left ventricle*, that pump blood around the two circuits.

Figure 18.1 The systemic and pulmonary circuits. The right side of the heart pumps blood through the pulmonary circuit and the left side of the heart pumps blood through the systemic circuit. The arrows indicate the direction of blood flow. For simplicity, the actual number of two pulmonary arteries and four pulmonary veins has been reduced to one each.

Size, Location, and Orientation of the Heart

The modest size and weight of the heart belie its incredible strength and endurance. About the size of a fist, the hollow, cone-shaped heart has a mass of 250 to 350 grams—less than a pound (**Figure 18.2**).

Figure 18.2 Location of the heart in the mediastinum. (a) Relationship of the heart to the sternum, ribs, and diaphragm in a person who is lying down (the heart is slightly inferior to this position in a standing person). **(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.

Snugly enclosed within the **mediastinum** (me″de-ah-sti′num), the medial cavity of the thorax, the heart extends obliquely for 12 to 14 cm (about 5 inches) from the second rib to the fifth intercostal space (Figure 18.2a). As it rests on the superior surface of the diaphragm, the heart lies anterior to the vertebral column and posterior to the sternum. Approximately two-thirds of its mass lies to the left of the midsternal line; the balance projects to the right. The lungs flank the heart laterally and partially obscure it (Figure 18.2b, c).

Its broad, flat **base**, or posterior surface, is about 9 cm (3.5 in) wide and directed toward the right shoulder. Its **apex** points inferiorly toward the left hip. If you press your fingers between the fifth and sixth ribs just below the left nipple, you can easily feel the **apical impulse** caused by your beating heart's apex where it touches the chest wall.

Coverings of the Heart

The heart is enclosed in a double-walled sac called the **pericardium** (per″ĭ-kar′de-um; *peri* = around, *cardi* = heart) (**Figure 18.3**). The loosely fitting superficial part of this sac is the **fibrous pericardium**. This tough, dense connective tissue layer (1) protects the heart, (2) anchors it to surrounding structures, and (3) prevents overfilling of the heart with blood.

Deep to the fibrous pericardium is the **serous pericardium**, a thin, slippery, two-layer serous membrane that forms a closed sac around the heart (see Figure 1.10, p. 18). Its **parietal layer** lines the internal surface of the fibrous pericardium. At the superior margin of the heart, the parietal layer attaches to the large arteries exiting the heart, and then turns inferiorly and continues over the external heart surface as the **visceral layer**, also called the *epicardium* ("upon the heart"), which is an integral part of the heart wall.

Between the parietal and visceral layers is the slitlike **pericardial cavity**, which contains a film of serous fluid. The serous membranes, lubricated by the fluid, glide smoothly past one another, allowing the mobile heart to work in a relatively friction-free environment.

HOMEOSTATIC IMBALANCE 18.1   CLINICAL

Pericarditis, inflammation of the pericardium, roughens the serous membrane surfaces. Consequently, as the beating heart rubs against its pericardial sac, it creates a creaking sound (*pericardial friction rub*) that can be heard with a stethoscope. Pericarditis is characterized by pain deep to the sternum. Over time, it may lead to adhesions in which the visceral and parietal pericardia stick together and impede heart activity.

In severe cases, large amounts of inflammatory fluid seep into the pericardial cavity. This excess fluid compresses the heart and limits its ability to pump blood, a condition called *cardiac tamponade* (tam″pŏ-nād′), literally, "heart plug." Physicians treat cardiac tamponade by inserting a syringe into the pericardial cavity and draining off the excess fluid. **+**
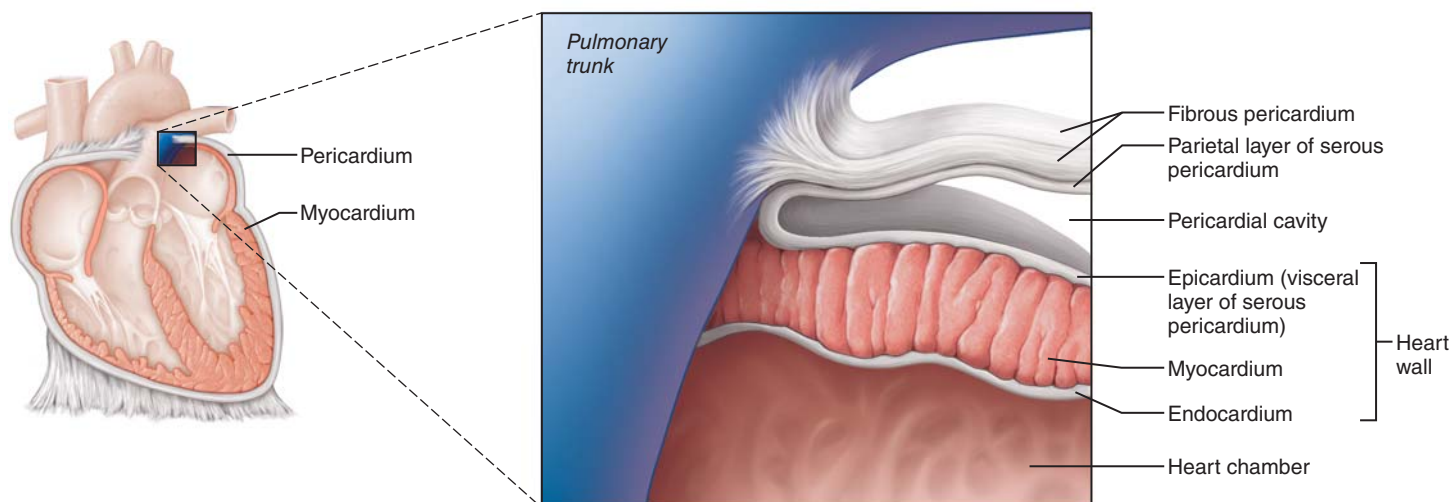
Layers of the Heart Wall

The heart wall, richly supplied with blood vessels, is composed of three layers: the epicardium, myocardium, and endocardium (Figure 18.3).

As we have noted, the superficial **epicardium** is the visceral layer of the serous pericardium. It is often infiltrated with fat, especially in older people.
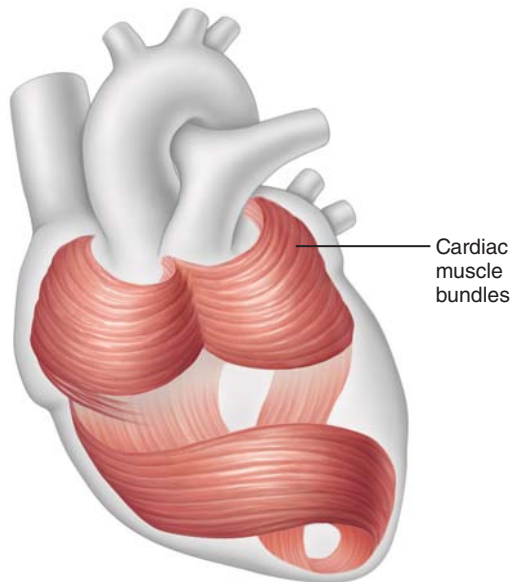
The middle layer, the **myocardium** ("muscle heart"), is composed mainly of cardiac muscle and forms the bulk of the heart. This is the layer that contracts. In the myocardium, the branching cardiac muscle cells are tethered to one another by crisscrossing connective tissue fibers and arranged in spiral or circular *bundles* (**Figure 18.4**). These interlacing bundles effectively link all parts of the heart together.

The connective tissue fibers form a dense network, the fibrous **cardiac skeleton**, that reinforces the myocardium internally and anchors the cardiac muscle fibers. This network of collagen and elastic fibers is thicker in some areas than others. For example, it constructs ropelike rings that provide additional support where the great vessels issue from the heart and around the heart valves (see Figure 18.6a, p. 671). Without this support, the vessels and valves might eventually become stretched because of the continuous stress of blood pulsing through them. Additionally, because connective tissue is not electrically excitable, the cardiac skeleton limits the spread of action potentials to specific pathways in the heart.

The third layer of the heart wall, the **endocardium** ("inside the heart"), is a glistening white sheet of endothelium (squamous epithelium) resting on a thin connective tissue layer. Located on the inner myocardial surface, it lines the heart chambers and covers the fibrous skeleton of the valves. The endocardium is

Figure 18.3 The layers of the pericardium and of the heart wall.

Figure 18.4 caption below the figure.

Figure 18.4 The circular and spiral arrangement of cardiac muscle bundles in the myocardium of the heart.

continuous with the endothelial linings of the blood vessels leaving and entering the heart.

Chambers and Associated Great Vessels

The heart has four chambers (**Figure 18.5e** on p. 670)—two superior **atria** (a′tre-ah) and two inferior **ventricles** (ven′trĭ-klz). The internal partition that divides the heart longitudinally is called the **interatrial septum** where it separates the atria, and the **interventricular septum** where it separates the ventricles. The right ventricle forms most of the anterior surface of the heart. The left ventricle dominates the inferoposterior aspect of the heart and forms the heart apex.

Two grooves visible on the heart surface indicate the boundaries of its four chambers and carry the blood vessels supplying the myocardium. The **coronary sulcus** (Figure 18.5b, d), or *atrioventricular groove*, encircles the junction of the atria and ventricles like a crown (*corona* = crown). The **anterior interventricular sulcus**, cradling the anterior interventricular artery, marks the anterior position of the septum separating the right and left ventricles. It continues as the **posterior interventricular sulcus**, which provides a similar landmark on the heart's posteroinferior surface.

Atria: The Receiving Chambers

Except for small, wrinkled, protruding appendages called **auricles** (or′ĭ-klz; *auricle* = little ear), which increase the atrial volume somewhat, the right and left atria are remarkably free of distinguishing surface features. Internally, the right atrium has two basic parts (Figure 18.5c): a smooth-walled posterior part and an anterior portion in which bundles of muscle tissue form ridges in the walls. These muscle bundles are called **pectinate**

muscles because they look like the teeth of a comb (*pectin* = comb). The posterior and anterior regions of the right atrium are separated by a C-shaped ridge called the *crista terminalis* ("terminal crest").

In contrast, the left atrium is mostly smooth and pectinate muscles are found only in the auricle. The interatrial septum bears a shallow depression, the **fossa ovalis** (o-vă′lis), that marks the spot where an opening, the *foramen ovale*, existed in the fetal heart (Figure 18.5c, e).

Functionally, the atria are receiving chambers for blood returning to the heart from the circulation (*atrium* = entryway). The atria are relatively small, thin-walled chambers because they need to contract only minimally to push blood "downstairs" into the ventricles. They contribute little to the propulsive pumping activity of the heart.

Blood enters the *right atrium* via three veins (Figure 18.5c–e):

- The **superior vena cava** returns blood from body regions superior to the diaphragm.
- The **inferior vena cava** returns blood from body areas below the diaphragm.
- The **coronary sinus** collects blood draining from the myocardium.

Four **pulmonary veins** enter the *left atrium*, which makes up most of the heart's base. These veins, which transport blood from the lungs back to the heart, are best seen in a posterior view (Figure 18.5d).

Ventricles: The Discharging Chambers

Together the ventricles (*ventr* = underside) make up most of the volume of the heart. As already mentioned, the right ventricle forms most of the heart's anterior surface and the left ventricle dominates its posteroinferior surface. Irregular ridges of muscle called **trabeculae carneae** (trah-bek′u-le kar′ne-e; "crossbars of flesh") mark the internal walls of the ventricular chambers. Other muscle bundles, the **papillary muscles**, which play a role in valve function, project into the ventricular cavity (Figure 18.5e).
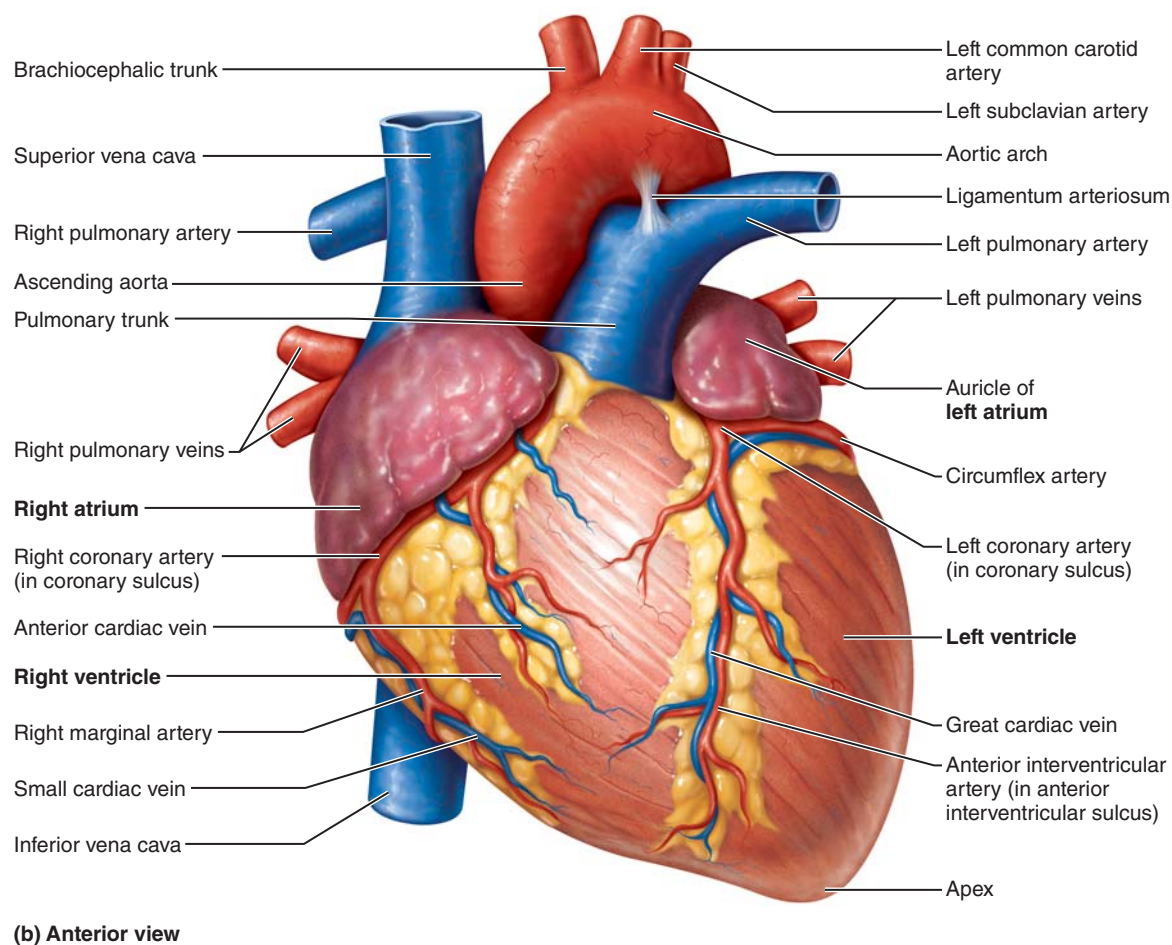
The ventricles are the discharging chambers, the actual pumps of the heart. Their walls are much more massive than the atrial walls, reflecting the difference in function between the atria and ventricles (Figure 18.5e and f). When the ventricles contract, they propel blood out of the heart into the circulation. The right ventricle pumps blood into the **pulmonary trunk**, which routes the blood to the lungs where gas exchange occurs. The left ventricle ejects blood into the **aorta** (a-or′tah), the largest artery in the body.

☑ Check Your Understanding

1. The heart is in the mediastinum. Just what is the mediastinum?
2. From inside to outside, list the layers of the heart wall and the coverings of the heart.
3. What is the purpose of the serous fluid inside the pericardial cavity?

For answers, see Answers Appendix.

(Text continues on p. 671.)

Left subclavian artery

Left common carotid artery

Brachiocephalic trunk

Ascending aorta

Right atrium

Right coronary artery
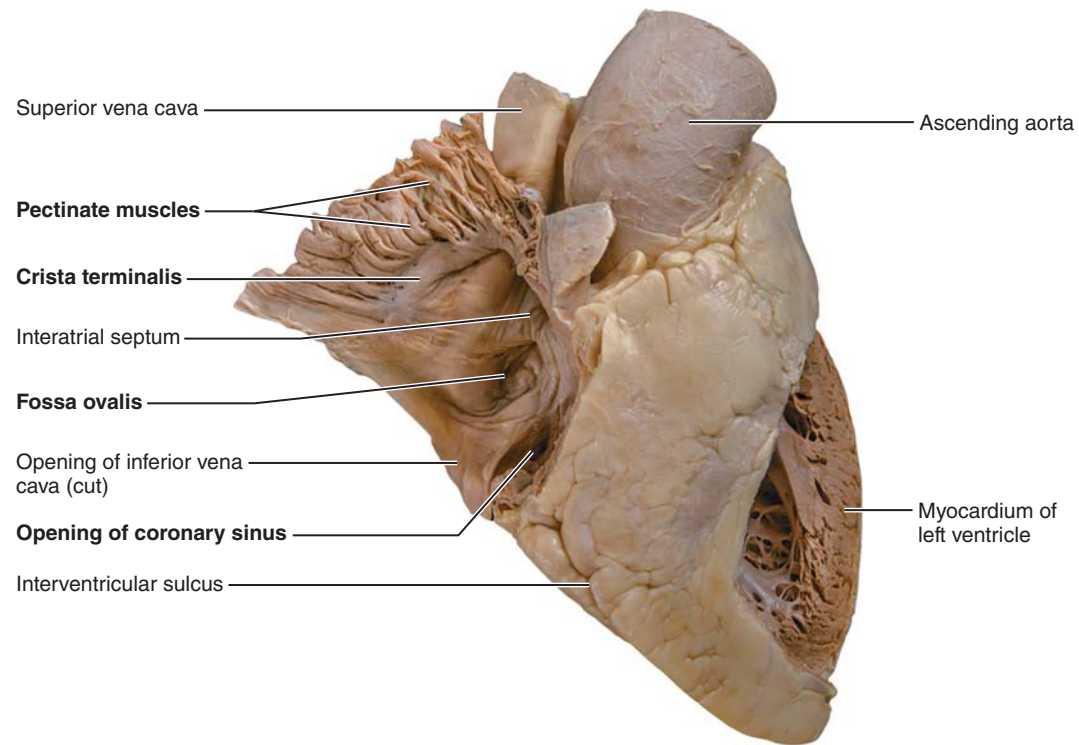(in coronary sulcus)

Right ventricle

Aortic arch

Ligamentum arteriosum

Pulmonary trunk

Auricle of left atrium

Anterior interventricular artery
(in anterior interventricular
sulcus)

Additional branch off left
coronary artery, normal
variation

Left ventricle

Apex of heart
(left ventricle)

(a) Anterior aspect (pericardium removed)

Brachiocephalic trunk

Superior vena cava

Right pulmonary artery

Ascending aorta

Pulmonary trunk

Right pulmonary veins

Right atrium

Right coronary artery
(in coronary sulcus)

Anterior cardiac vein

Right ventricle

Right marginal artery

Small cardiac vein

Inferior vena cava

Left common carotid
artery

Left subclavian artery

Aortic arch

Ligamentum arteriosum

Left pulmonary artery

Left pulmonary veins

Auricle of
left atrium

Circumflex artery

Left coronary artery
(in coronary sulcus)

Left ventricle

Great cardiac vein

Anterior interventricular
artery (in anterior
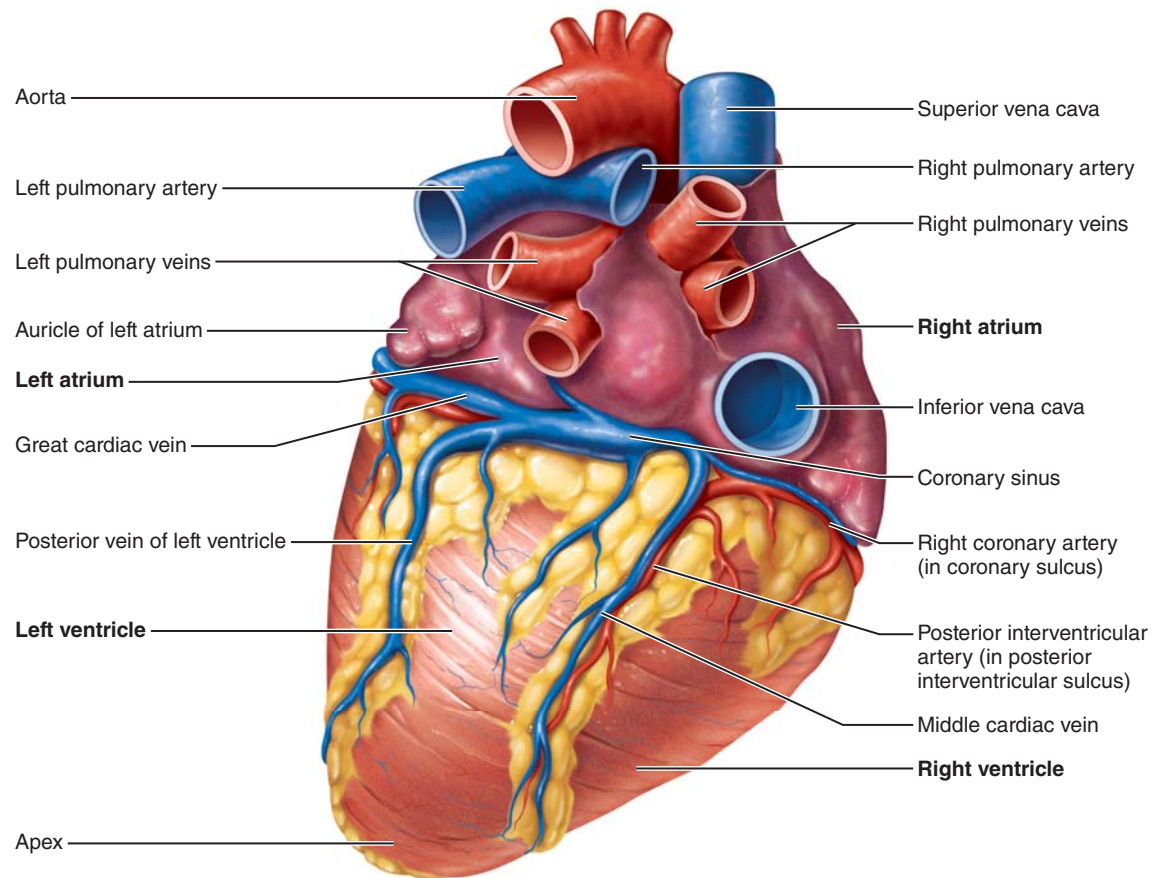interventricular sulcus)

Apex

(b) Anterior view

Figure 18.5 Gross anatomy of the heart. In diagrammatic views, vessels transporting
oxygen-rich blood are red; those transporting oxygen-poor blood are blue.

Practice art labeling
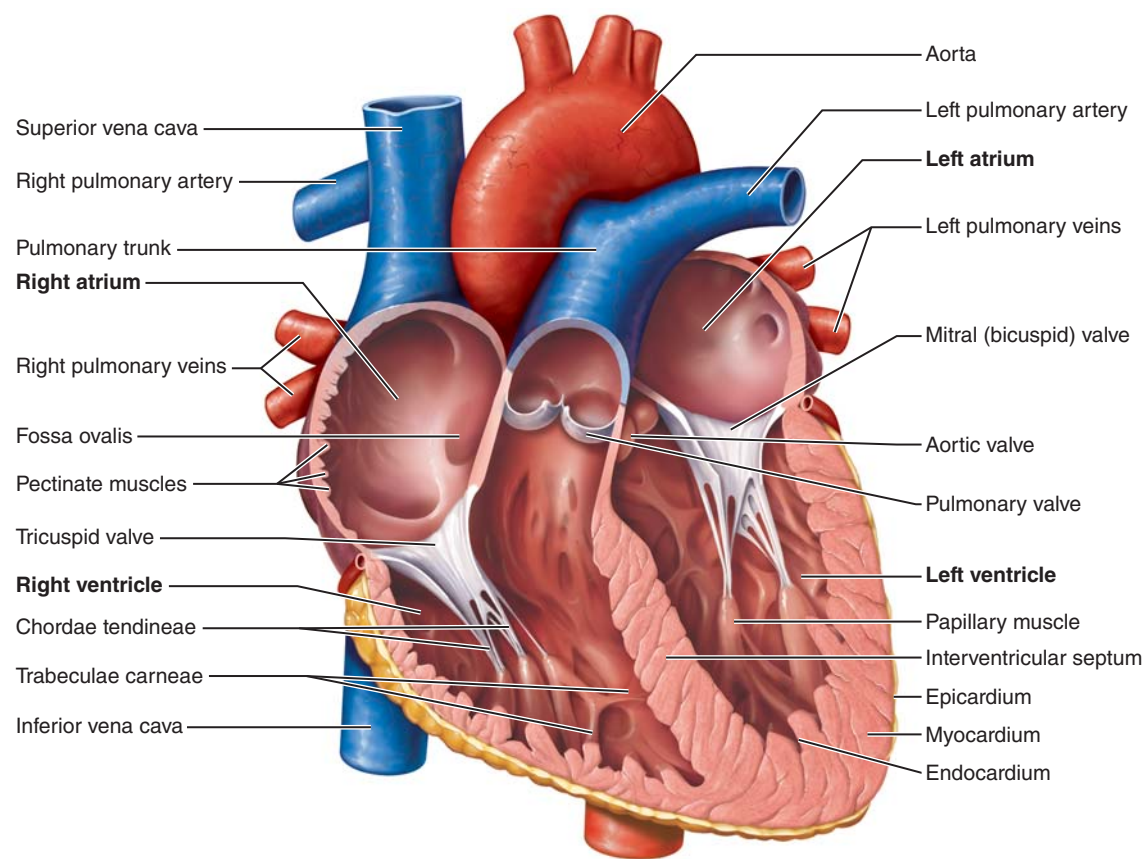MasteringA&P®>Study Area>Chapter 18

Superior vena cava —

Ascending aorta

Pectinate muscles —

Crista terminalis —

Interatrial septum —

Fossa ovalis —

Opening of inferior vena cava (cut) —

Myocardium of left ventricle

Opening of coronary sinus —

Interventricular sulcus —

(c) Internal aspect of the right atrium, anterior view

Aorta —

Superior vena cava

Left pulmonary artery —

Right pulmonary artery

Left pulmonary veins —

Right pulmonary veins

Auricle of left atrium —

Right atrium

Left atrium —

Inferior vena cava

Great cardiac vein —

Coronary sinus

Posterior vein of left ventricle —

Right coronary artery (in coronary sulcus)

Left ventricle —

Posterior interventricular artery (in posterior interventricular sulcus)

Middle cardiac vein
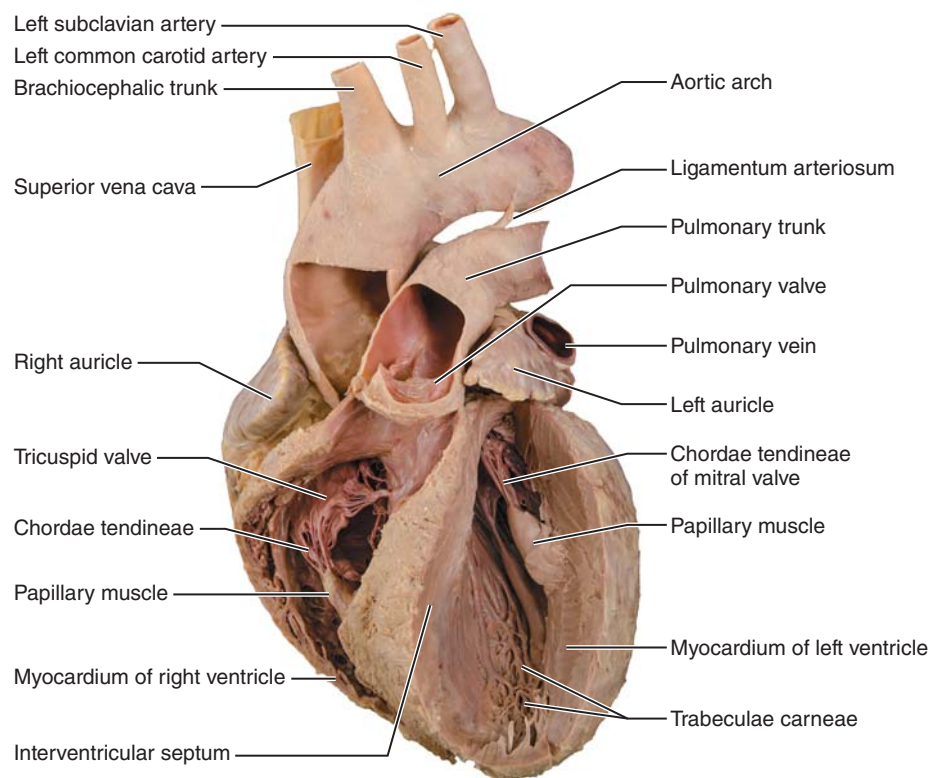
Right ventricle

Apex —

(d) Posterior surface view

18

Figure 18.5 *(continued)* In (c), the anterior wall of the atrium has been opened and folded superiorly.

Aorta

Superior vena cava

Left pulmonary artery

Right pulmonary artery

Left atrium

Pulmonary trunk

Left pulmonary veins

Right atrium

Right pulmonary veins

Mitral (bicuspid) valve

Fossa ovalis

Aortic valve

Pectinate muscles

Pulmonary valve

Tricuspid valve

Left ventricle

Right ventricle

Papillary muscle

Chordae tendineae

Interventricular septum

Trabeculae carneae

Epicardium

Inferior vena cava

Myocardium

Endocardium

(e) Frontal section

Left subclavian artery

Left common carotid artery

Brachiocephalic trunk

Aortic arch

Ligamentum arteriosum

Superior vena cava

Pulmonary trunk

Pulmonary valve

Pulmonary vein

Right auricle

Left auricle

Tricuspid valve

Chordae tendineae
of mitral valve

Chordae tendineae

Papillary muscle

Papillary muscle

Myocardium of left ventricle

Myocardium of right ventricle

Trabeculae carneae

Interventricular septum

(f) Internal aspect of ventricles; dissection of view similar to (e)

Figure 18.5 *(continued)* **Gross anatomy of the heart.**

18.2   Heart valves make blood flow in one direction

→ **Learning Objective**

☐ **Name the heart valves and describe their location, function, and mechanism of operation.**
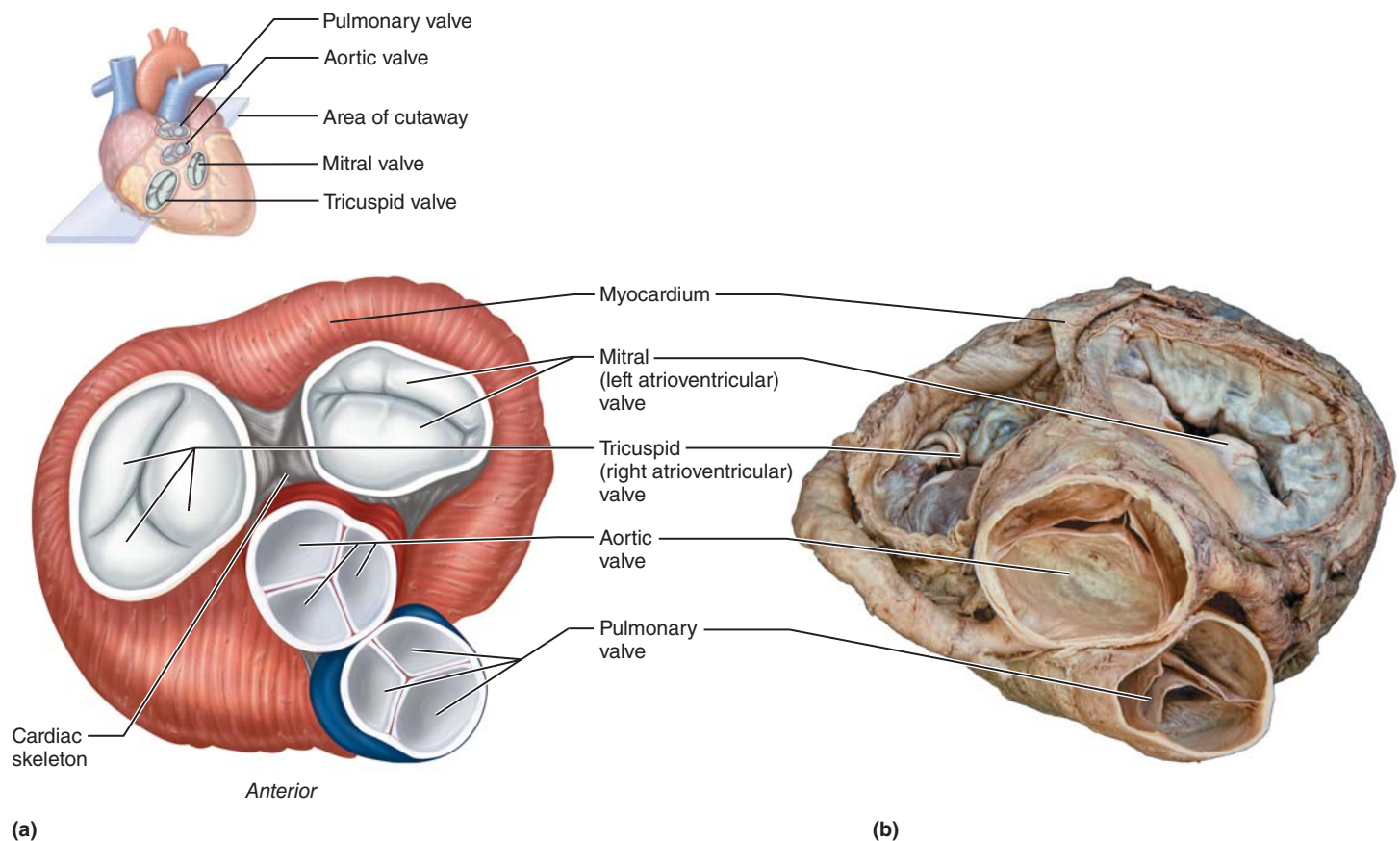
Blood flows through the heart in one direction: from atria to ventricles and out the great arteries leaving the superior aspect of the heart. Four valves enforce this one-way traffic (Figure 18.5e and **Figure 18.6**). They open and close in response to differences in blood pressure on their two sides.
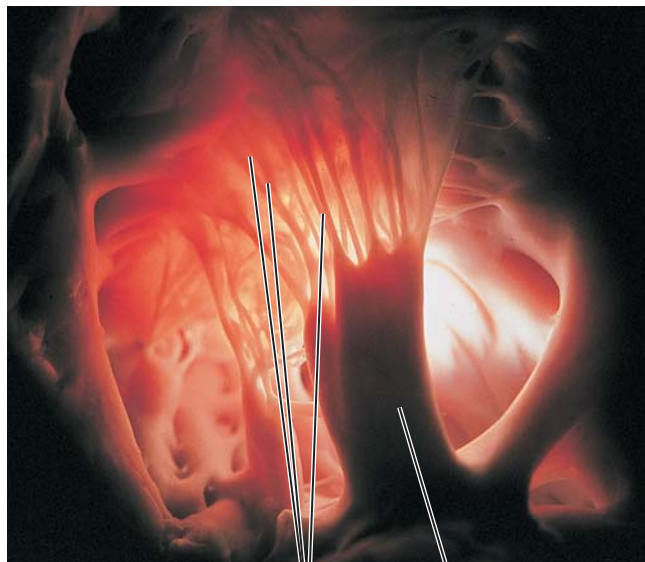
Atrioventricular (AV) Valves

The two **atrioventricular (AV) valves**, one located at each atrial-ventricular junction, prevent backflow into the atria when the ventricles contract.

- The right AV valve, the **tricuspid valve** (tri-kus′pid), has three flexible cusps (flaps of endocardium reinforced by connective tissue cores).
- The left AV valve, with two cusps, is called the **mitral valve** (mi′tral) because it resembles the two-sided bishop's miter (tall, pointed hat). It is sometimes called the *bicuspid valve*.

Attached to each AV valve flap are tiny white collagen cords called **chordae tendineae** (kor′de ten″di′ne-e; "tendinous cords"), "heart strings" which anchor the cusps to the papillary muscles protruding from the ventricular walls (Figure 18.6c, d).
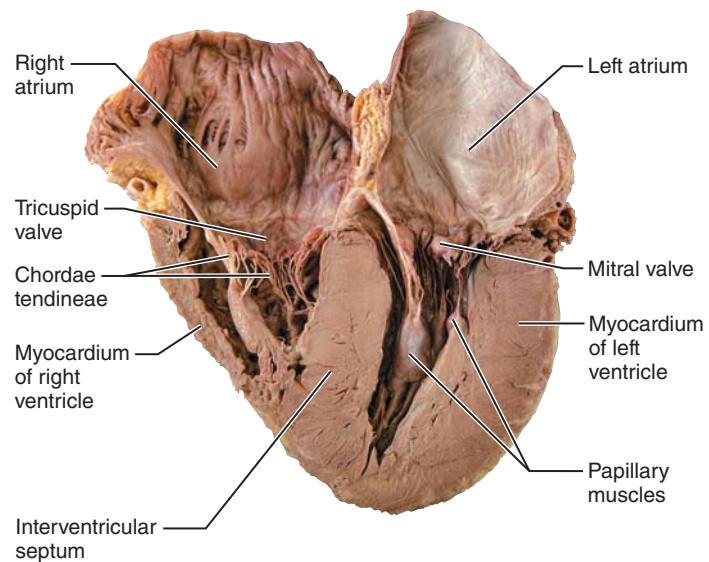

(a)

(b)

Figure 18.6   Heart valves. (a) Superior view of the two sets of heart valves (atria removed). The paired atrioventricular valves are located between atria and ventricles; the two semilunar valves are located at the junction of the ventricles and the arteries issuing from them. **(b)** Photograph of the heart valves, superior view.

Explore human cadaver
MasteringA&P®Study Area>PAL

(c)

Right atrium

Left atrium

Tricuspid valve

Chordae tendineae

Mitral valve

Myocardium of right ventricle

Myocardium of left ventricle

Interventricular septum

Papillary muscles

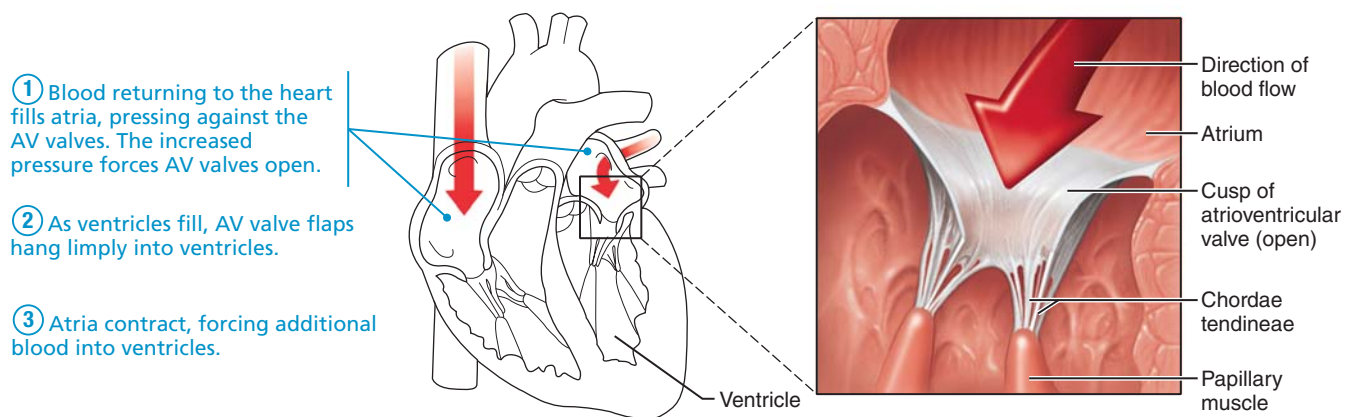Chordae tendineae attached to tricuspid valve flap
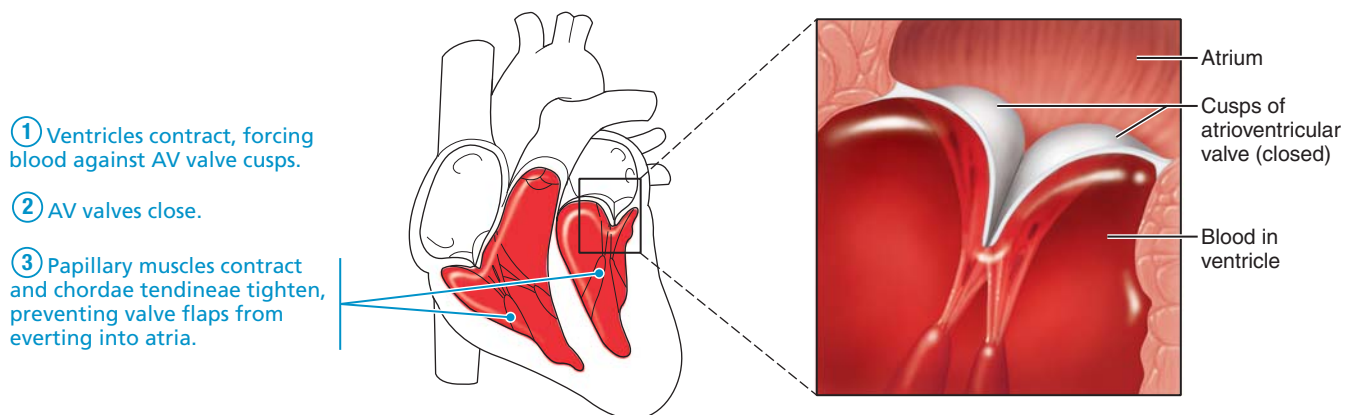
Papillary muscle

(d)

Figure 18.6 *(continued)* **Heart valves. (c)** Photograph of the tricuspid valve. This bottom-to-top view shows the valve as seen from the right ventricle. **(d)** Frontal section of the heart. (For related images, see *A Brief Atlas of the Human Body*, Figures 58, 60, and 61.)

① Blood returning to the heart fills atria, pressing against the AV valves. The increased pressure forces AV valves open.

② As ventricles fill, AV valve flaps hang limply into ventricles.

③ Atria contract, forcing additional blood into ventricles.

Direction of blood flow

Atrium

Cusp of atrioventricular valve (open)

Chordae tendineae

Papillary muscle

Ventricle

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

① Ventricles contract, forcing blood against AV valve cusps.

② AV valves close.

③ Papillary muscles contract and chordae tendineae tighten, preventing valve flaps from everting into atria.

Atrium

Cusps of atrioventricular valve (closed)

Blood in ventricle

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

Figure 18.7 **The function of the atrioventricular (AV) valves.**

18

When the heart is completely relaxed, the AV valve flaps hang limply into the ventricular chambers below. During this time, blood flows into the atria and then through the open AV valves into the ventricles (**Figure 18.7a**). When the ventricles contract, compressing the blood in their chambers, the intraventricular pressure rises, forcing the blood superiorly against the valve flaps. As a result, the flap edges meet, closing the valve (Figure 18.7b).

The chordae tendineae and the papillary muscles serve as guy-wires that anchor the valve flaps in their *closed* position. If the cusps were not anchored, they would be blown upward (everted) into the atria, in the same way an umbrella is blown inside out by a gusty wind. The papillary muscles contract with the other ventricular musculature so that they take up the slack on the chordae tendineae as the full force of ventricular contraction hurls the blood against the AV valve flaps.

Semilunar (SL) Valves

The **aortic** and **pulmonary (semilunar**, **SL) valves** guard the bases of the large arteries issuing from the ventricles (aorta and pulmonary trunk, respectively) and prevent backflow into the associated ventricles. Each SL valve is fashioned from three pocketlike cusps, each shaped roughly like a crescent moon (*semilunar* = half-moon).

Like the AV valves, the SL valves open and close in response to differences in pressure. When the ventricles contract and intraventricular pressure rises above the pressure in the aorta and pulmonary trunk, the SL valves are forced open and their cusps flatten against the arterial walls as blood rushes past them (**Figure 18.8a**). When the ventricles relax, and the blood flows backward toward the heart, it fills the cusps and closes the valves (Figure 18.8b).
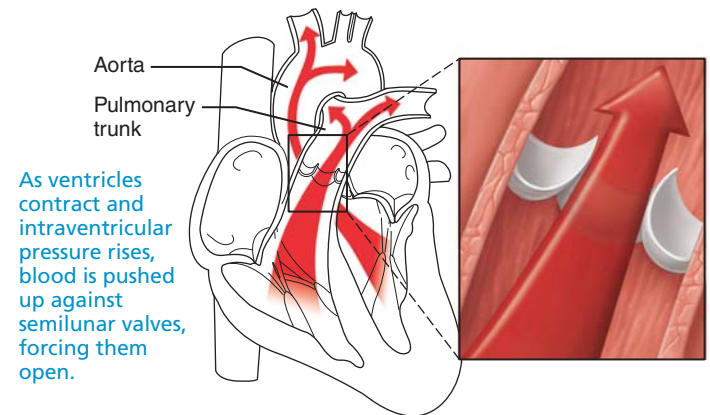
We complete the valve story by noting what seems to be an important omission—there are no valves guarding the entrances of the venae cavae and pulmonary veins into the right and left atria, respectively. Small amounts of blood *do* spurt back into these vessels during atrial contraction, but backflow is minimal because of the inertia of the blood and because as it contracts, the atrial myocardium compresses (and collapses) these venous entry points.

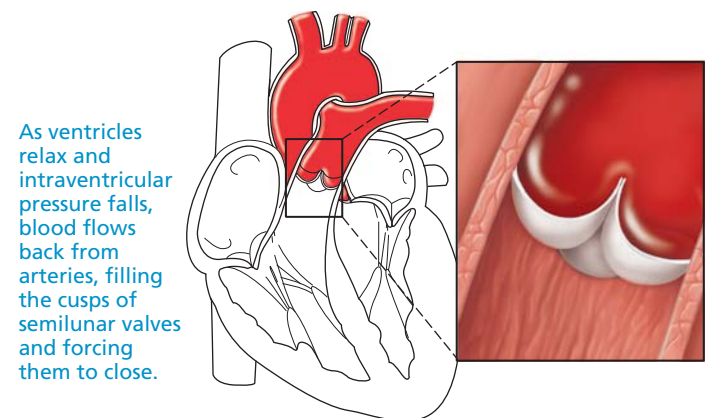> ✒ **HOMEOSTATIC IMBALANCE 18.2**   CLINICAL
>
> Heart valves are simple devices, and the heart—like any mechanical pump—can function with "leaky" valves as long as the impairment is not too great. However, severe valve deformities can seriously hamper cardiac function.
>
> An *incompetent*, or *insufficient*, *valve* forces the heart to repump the same blood over and over because the valve does not close properly and blood backflows. In valvular *stenosis* ("narrowing"), the valve flaps become stiff (typically due to calcium salt deposits or scar tissue that forms following endocarditis) and constrict the opening. This stiffness compels the heart to contract more forcibly than normal. Both conditions increase the heart's workload and may weaken the heart severely over time.
>
> The faulty valve (most often the mitral valve) can be replaced with a mechanical valve, a pig or cow heart valve chemically

Aorta
Pulmonary trunk

As ventricles contract and intraventricular pressure rises, blood is pushed up against semilunar valves, forcing them open.

(a) Semilunar valves open

As ventricles relax and intraventricular pressure falls, blood flows back from arteries, filling the cusps of semilunar valves and forcing them to close.

(b) Semilunar valves closed

Figure 18.8 The function of the semilunar (SL) valves.

treated to prevent rejection, or cryopreserved valves from human cadavers. Heart valves tissue-engineered from a patient's own cells grown on a biodegradable scaffold are being developed. ✚

☑ Check Your Understanding

4. What is the function of the papillary muscles and chordae tendineae?

5. Name the valve that has just two cusps.
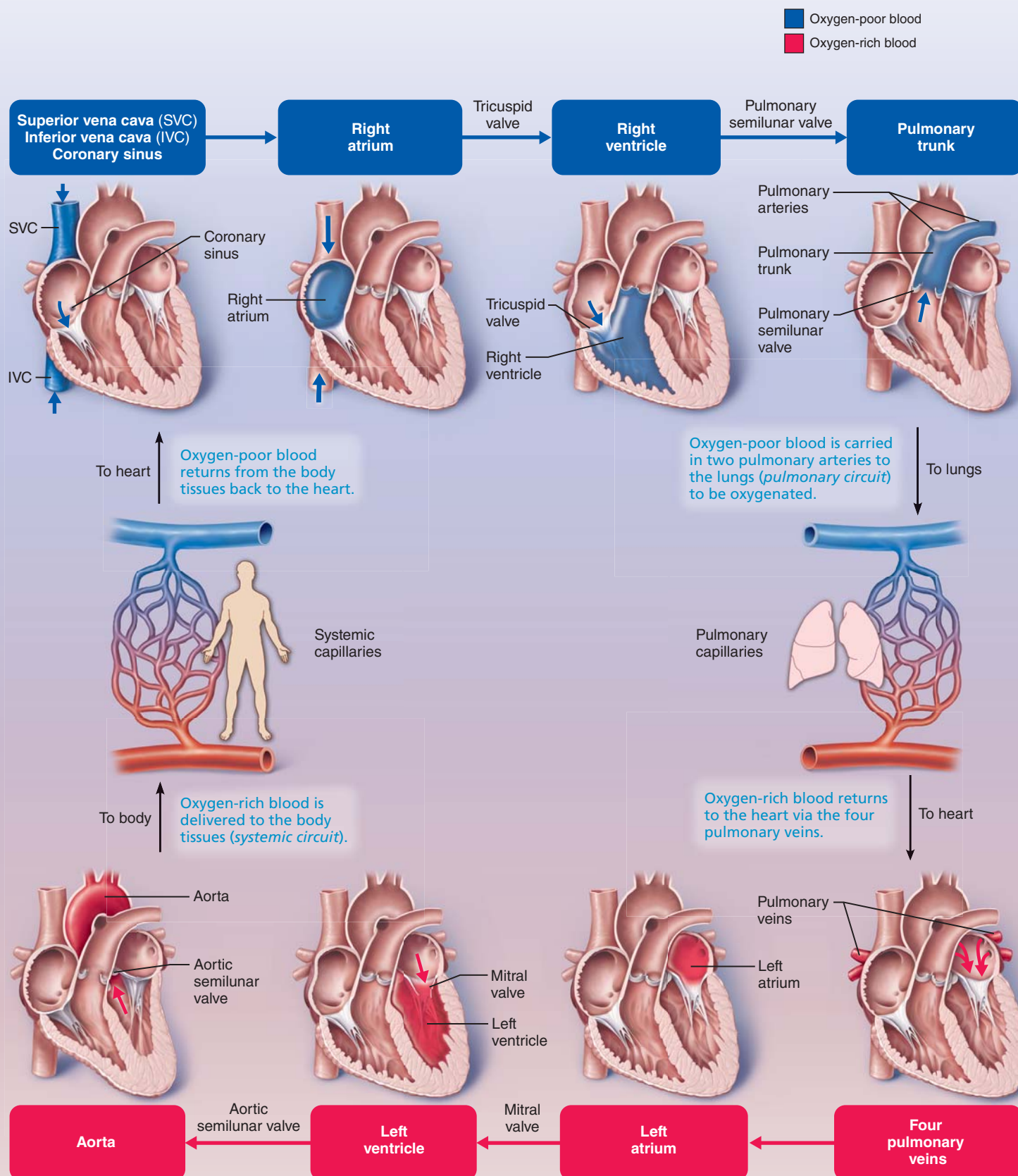
For answers, see Answers Appendix.

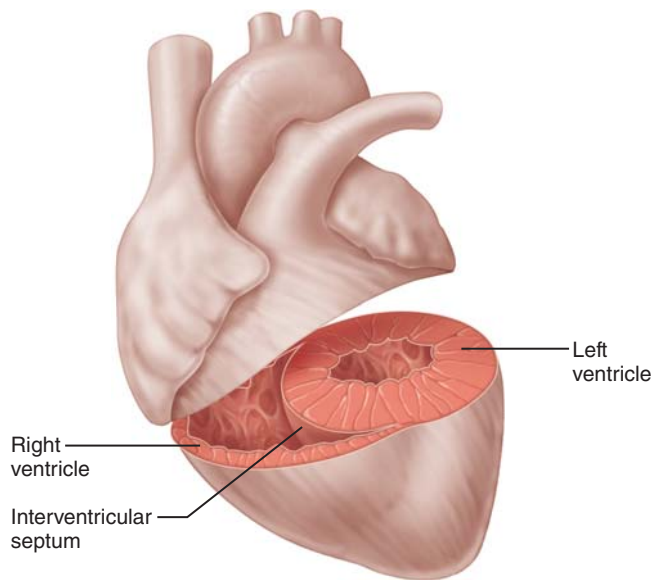## 18.3 Blood flows from atrium to ventricle, and then to either the lungs or the rest of the body

→ **Learning Objectives**
☐ **Trace the pathway of blood through the heart.**
☐ **Name the major branches and describe the distribution of the coronary arteries.**

Having covered the basic anatomy of the heart, we can now follow the path that blood takes through the heart and its associated circuits. *Focus on Blood Flow through the Heart*

Focus Figure 18.1 The heart is a double pump, each side supplying its own circuit.

- Oxygen-poor blood
- Oxygen-rich blood

Superior vena cava (SVC)
Inferior vena cava (IVC)
Coronary sinus

SVC
Coronary sinus
IVC

Right atrium

Right atrium

Tricuspid valve

Right ventricle

Tricuspid valve
Right ventricle

Pulmonary semilunar valve

Pulmonary trunk

Pulmonary arteries
Pulmonary trunk
Pulmonary semilunar valve

To heart
Oxygen-poor blood returns from the body tissues back to the heart.

Oxygen-poor blood is carried in two pulmonary arteries to the lungs (*pulmonary circuit*) to be oxygenated.
To lungs

Systemic capillaries

Pulmonary capillaries

To body
Oxygen-rich blood is delivered to the body tissues (*systemic circuit*).

Oxygen-rich blood returns to the heart via the four pulmonary veins.
To heart

Aorta
Aortic semilunar valve

Mitral valve
Left ventricle

Left atrium

Pulmonary veins
Left atrium

Aorta

Aortic semilunar valve

Left ventricle

Mitral valve

Left atrium

Four pulmonary veins

Figure 18.9 Anatomical differences between the right and left ventricles. The left ventricle has a thicker wall and its cavity is basically circular. The right ventricle cavity is crescent shaped and wraps around the left ventricle.

(**Focus Figure 18.1**) follows a single "spurt" of blood as it passes through all four chambers of the heart and both blood circuits in its ever-repeating journey.

As you work your way through this figure, keep in mind that the left side of the heart is the *systemic circuit pump* and the right side is the *pulmonary circuit pump*. Notice how unique the pulmonary circuit is. Elsewhere in the body, veins carry relatively oxygen-poor blood to the heart, and arteries transport oxygen-rich blood from the heart. The opposite oxygenation conditions exist in veins and arteries of the pulmonary circuit.

Equal volumes of blood are pumped to the pulmonary and systemic circuits at any moment, but the two ventricles have very unequal workloads. The pulmonary circuit, served by the right ventricle, is a short, low-pressure circulation. In contrast, the systemic circuit, associated with the left ventricle, takes a long pathway through the entire body and encounters about five times as much friction, or resistance to blood flow.

This functional difference is revealed in the anatomy of the two ventricles (Figure 18.5e and **Figure 18.9**). The walls of the left ventricle are three times thicker than those of the right ventricle, and its cavity is nearly circular. The right ventricular cavity is flattened into a crescent shape that partially encloses the left ventricle, much the way a hand might loosely grasp a clenched fist. Consequently, the left ventricle can generate much more pressure than the right and is a far more powerful pump.

Coronary Circulation

Although the heart is continuously filled with various amounts of blood, this blood provides little nourishment to heart tissue. (The myocardium is too thick to make diffusion a practical

means of delivering nutrients.) How, then, does the heart get nourishment? It does so through the **coronary circulation**, the functional blood supply of the heart, and the shortest circulation in the body.

Coronary Arteries

The *left* and *right coronary arteries* both arise from the base of the aorta and encircle the heart in the coronary sulcus. They provide the arterial supply of the coronary circulation (**Figure 18.10a**).

The **left coronary artery** runs toward the left side of the heart and then divides into two major branches:
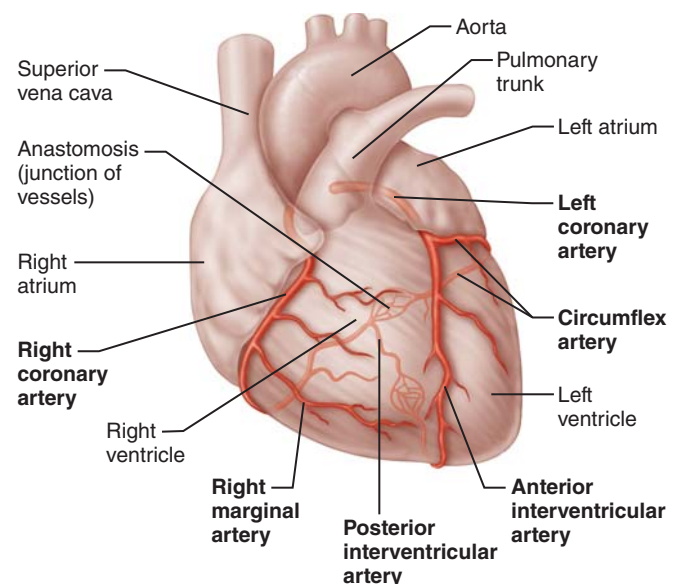
- The **anterior interventricular artery** (also known clinically as the *left anterior descending artery*) follows the anterior interventricular sulcus and supplies blood to the interventricular septum and anterior walls of both ventricles.
- The **circumflex artery** supplies the left atrium and the posterior walls of the left ventricle.

The **right coronary artery** courses to the right side of the heart, where it also gives rise to two branches:

- The **right marginal artery** serves the myocardium of the lateral right side of the heart.
- The **posterior interventricular artery** runs to the heart apex and supplies the posterior ventricular walls. Near the apex of the heart, this artery merges (anastomoses) with the anterior interventricular artery.

Together the branches of the right coronary artery supply the right atrium and nearly all the right ventricle.

The arterial supply of the heart varies considerably. For example, in 15% of people, the left coronary artery gives rise to *both* interventricular arteries. In about 4% of people, a single

Figure 18.10 Coronary circulation. (a) The major coronary arteries. Lighter-tinted vessels are more posterior in the heart.

coronary artery supplies the whole heart. Additionally, there may be both right and left marginal arteries. There are many anastomoses (junctions) among the coronary arterial branches. These fusing networks provide additional (*collateral*) routes for blood delivery to the heart muscle, but are not robust enough to supply adequate nutrition when a coronary artery is suddenly occluded (blocked). Complete blockage leads to tissue death and heart attack.

The coronary arteries provide an intermittent, pulsating blood flow to the myocardium. These vessels and their main branches lie in the epicardium and send branches inward to nourish the myocardium. They deliver blood when the heart is relaxed, but are fairly ineffective when the ventricles are contracting because they are compressed by the contracting myocardium. Although the heart represents only about 1/200 of the body's weight, it requires about 1/20 of the body's blood supply. As might be expected, the left ventricle receives the most plentiful blood supply.

Coronary Veins

After passing through the capillary beds of the myocardium, the venous blood is collected by the **cardiac veins**, whose paths roughly follow those of the coronary arteries. These veins join to form an enlarged vessel called the **coronary sinus**, which empties the blood into the right atrium. The coronary sinus is obvious on the posterior aspect of the heart (Figure 18.10b).

The sinus has 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 right inferior margin. Additionally, several **anterior cardiac veins** empty directly into the right atrium anteriorly.

Superior vena cava

Anterior cardiac veins

Great cardiac vein

Coronary sinus

Small cardiac vein   Middle cardiac vein

Figure 18.10 *(continued)* **Coronary circulation. (b)** The major cardiac veins. Lighter-tinted vessels are more posterior in the heart.

Blockage of the coronary arterial circulation can be serious and sometimes fatal. **Angina pectoris** (an-ji′nah pek′tor-is; "choked chest") is thoracic pain caused by a fleeting deficiency in blood delivery to the myocardium. It may result from stress-induced spasms of the coronary arteries or from increased physical demands on the heart. The myocardial cells are weakened by the temporary lack of oxygen but do not die.

Prolonged coronary blockage is far more serious because it can lead to a **myocardial infarction (MI)**, commonly called a **heart attack**, in which cells *do* die. Since adult cardiac muscle is essentially amitotic, most of the dead tissue is replaced with noncontractile scar tissue. Whether or not a person survives a myocardial infarction depends on the extent and location of the damage. Damage to the left ventricle—the systemic pump—is most serious. ✚

☑ Check Your Understanding

6. Which side of the heart acts as the pulmonary pump? The systemic pump?

7. Which of the following statements are true? (a) The left ventricle wall is thicker than the right ventricle wall. (b) The left ventricle pumps blood at a higher pressure than the right ventricle. (c) The left ventricle pumps more blood with each beat than the right ventricle. Explain.

8. Name the two main branches of the right coronary artery.

For answers, see Answers Appendix.

18.4 Intercalated discs connect cardiac muscle fibers into a functional syncytium
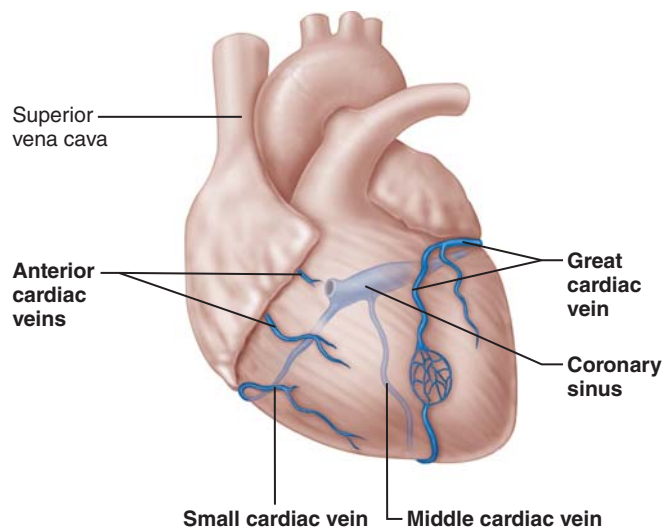
→ **Learning Objectives**

☐ Describe the structural and functional properties of cardiac muscle, and explain how it differs from skeletal muscle.

☐ Briefly describe the events of excitation-contraction coupling in cardiac muscle cells.
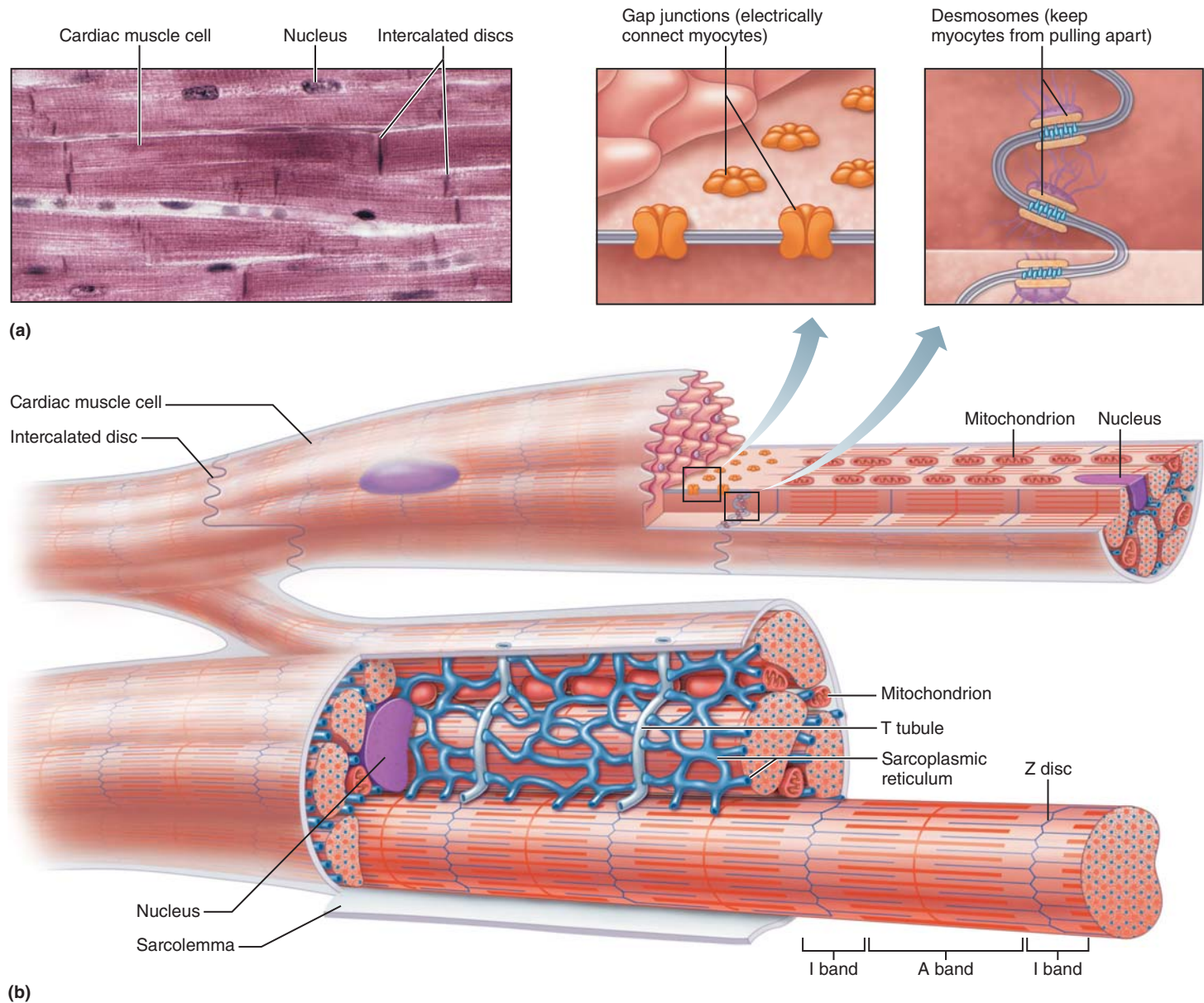
Although similar to skeletal muscle, cardiac muscle displays some special anatomical features that reflect its unique blood-pumping role.

Microscopic Anatomy

Like skeletal muscle, **cardiac muscle** is striated and contracts by the sliding filament mechanism. However, in contrast to the long, cylindrical, multinucleate skeletal muscle fibers, cardiac cells are short, fat, branched, and interconnected. Each fiber contains one or at most two large, pale, *centrally* located nuclei (**Figure 18.11a**). The intercellular spaces are filled with a loose connective tissue matrix (the *endomysium*) containing numerous capillaries. This delicate matrix is connected to the fibrous cardiac skeleton, which acts both as a tendon and as an insertion, giving the cardiac cells something to pull or exert their force against.

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Figure 18.11 Microscopic anatomy of cardiac muscle. (a) Photomicrograph of cardiac muscle (290×). Notice that the cardiac muscle cells are short, branched, and striated. The dark-staining areas are intercalated discs, or junctions, between adjacent cells. **(b)** Components of intercalated discs and cardiac muscle fibers.

Skeletal muscle fibers are independent of one another both structurally and functionally. In contrast, the plasma membranes of adjacent cardiac cells interlock like the ribs of two sheets of corrugated cardboard at dark-staining junctions called **intercalated discs** (in-ter′kah-la″ted; *intercala* = insert) (Figure 18.11). Intercalated discs contain anchoring *desmosomes* and *gap junctions* (cell junctions discussed in Chapter 3). The desmosomes prevent adjacent cells from separating during contraction, and the gap junctions allow ions to pass from cell to cell, transmitting current across the entire heart. Because gap junctions electrically couple cardiac cells, the myocardium *behaves* as a single coordinated unit, or **functional syncytium** (sin-sit′e-um; *syn* = together, *cyt* = cell).

Large mitochondria account for 25–35% of the volume of cardiac cells (compared with only 2% in skeletal muscle), a characteristic that makes cardiac cells highly resistant to fatigue. Most of the remaining volume is occupied by myofibrils composed of fairly typical sarcomeres. The sarcomeres have Z discs, A bands, and I bands that reflect the arrangement of the thick (myosin) and thin (actin) filaments composing them. However, in contrast to skeletal muscle, the myofibrils of cardiac muscle cells vary greatly in diameter and branch extensively, accommodating the abundant mitochondria between them. This difference produces a banding pattern less dramatic than that seen in skeletal muscle.

The system for delivering Ca^{2+} is less elaborate in cardiac muscle cells. The T tubules are wider and fewer than in skeletal muscle and they enter the cells once per sarcomere at the

Z discs. (Recall that T tubules are invaginations of the sarcolemma. In skeletal muscle, the T tubules invaginate twice per sarcomere, at the A band–I band junctions.) The cardiac sarcoplasmic reticulum is simpler and lacks the large terminal cisterns seen in skeletal muscle. Consequently, cardiac muscle fibers do not have *triads*.

How Does the Physiology of Skeletal and Cardiac Muscle Differ?

Both skeletal muscle and cardiac muscle are contractile tissues, and in both types of muscle the contraction is preceded by a depolarization in the form of an action potential (AP). Transmission of the depolarization wave down the T tubules (ultimately) causes the sarcoplasmic reticulum (SR) to release Ca^{2+} into the sarcoplasm. Excitation-contraction coupling occurs as Ca^{2+} provides the signal (via troponin binding) for cross bridge activation. This sequence of events couples the depolarization wave to the sliding of the myofilaments in both skeletal and cardiac muscle cells. However, cardiac muscle fibers differ from skeletal muscle fibers as summarized in **Table 18.1** and described below.

- **Some cardiac muscle cells are self-excitable.** The heart contains two kinds of myocytes. Almost all of the myocytes are *contractile cardiac muscle cells*, responsible for the heart's pumping activity. However, certain locations in the heart contain special noncontractile cells, called *pacemaker cells*, that spontaneously depolarize. Because heart cells are electrically joined together by gap junctions, these cells can initiate not only their own depolarization, but also that of the rest of the heart. No neural input is required. As demonstrated by transplanted hearts, you can cut all of the nerves to the heart and it still beats. In contrast, each skeletal muscle fiber must be stimulated by a nerve ending to contract, and cutting the nerves results in paralysis.

- **The heart contracts as a unit.** As we just learned, gap juctions tie cardiac muscle cells together to form a functional syncytium. This allows the wave of depolarization to travel from cell to cell across the heart. As a result, either all fibers in the heart contract as a unit or the heart doesn't contract at all. In skeletal muscle, on the other hand, impulses do not spread from cell to cell. Only skeletal muscle fibers that are individually stimulated by nerve fibers contract, and the strength of the contraction increases as more motor units are recruited. Such recruitment cannot happen in the heart because it acts as a single huge motor unit. Contraction of all of the cardiac myocytes ensures effective pumping by the heart—a half-hearted contraction would just not do.

- **The influx of Ca^{2+} from extracellular fluid triggers Ca^{2+} release from the SR.**

Recall that in skeletal muscle, the wave of depolarization directly causes release from the SR of all the Ca^{2+} required for contraction. In cardiac muscle, depolarization opens special Ca^{2+} channels in the plasma membrane. These *slow Ca^{2+} channels* allow entry of 10–20% of the Ca^{2+} needed for contraction. Once inside, this influx of Ca^{2+} triggers Ca^{2+}-sensitive channels in the SR to release bursts of Ca^{2+} ("calcium sparks") that account for the other 80–90% of the Ca^{2+} needed for contraction.

- **Tetanic contractions cannot occur in cardiac muscles.** The absolute refractory period is the period during an action potential when another action potential cannot be triggered. In skeletal muscle, the absolute refractory period is much shorter than the contraction, allowing multiple contractions to summate (tetanic contractions). If the heart were to contract tetanically, it would be unable to relax and fill, and so would be useless as a pump. To prevent tetanic contractions, the absolute refractory period in the heart is nearly as long as the contraction itself. We will examine the mechanism underlying the long refractory period of contractile cardiac myocytes shortly.

- **The heart relies almost exclusively on aerobic respiration.** Cardiac muscle has more mitochondria than skeletal muscle does, reflecting its greater dependence on oxygen for its energy metabolism. The heart relies almost exclusively on aerobic respiration, so cardiac muscle cannot operate effectively for long without oxygen. This is in contrast to skeletal muscle, which can contract for prolonged periods by carrying out anaerobic respiration, and then restore its reserves of oxygen and fuel using excess postexercise oxygen consumption (EPOC).

    Both types of muscle tissue use multiple fuel molecules, including glucose and fatty acids. But cardiac muscle is much more adaptable and readily switches metabolic pathways to use whatever nutrients are available, including lactic acid

Table 18.1	Key Differences between Skeletal and Cardiac Muscle	
	SKELETAL MUSCLE	**CARDIAC MUSCLE**
Structure	Striated, long, cylindrical, multinucleate	Striated, short, branched, one or two nuclei per cell
Gap junctions between cells	No	Yes
Contracts as a unit	No, motor units must be stimulated individually	Yes, gap junctions create a functional syncytium
T tubules	Abundant	Fewer, wider
Sarcoplasmic reticulum	Elaborate; has terminal cisterns	Less elaborate; no terminal cisterns
Source of Ca^{2+} for contraction	Sarcoplasmic reticulum only	Sarcoplasmic reticulum and extracellular fluid
Ca^{2+} binds to troponin	Yes	Yes
Pacemaker cells present	No	Yes
Tetanus possible	Yes	No
Supply of ATP	Aerobic and anaerobic (fewer mitochondria)	Aerobic only (more mitochondria)

generated by skeletal muscle activity. Consequently, the real danger of an inadequate blood supply to the myocardium is not lack of nutrients, but lack of oxygen.

☑ Check Your Understanding

9. For each of the following, state whether it applies to skeletal muscle, cardiac muscle, or both: (a) refractory period is almost as long as the contraction; (b) source of Ca^{2+} for contraction is *only* SR; (c) has troponin; (d) has triads.

For answers, see Answers Appendix.

18.5 Pacemaker cells trigger action potentials throughout the heart

→ **Learning Objectives**

☐ **Describe and compare action potentials in cardiac pacemaker and contractile cells.**

☐ **Name the components of the conduction system of the heart, and trace the conduction pathway.**

☐ **Draw a diagram of a normal electrocardiogram tracing. Name the individual waves and intervals, and indicate what each represents. Name some abnormalities that can be detected on an ECG tracing.**

Although the ability of the heart to depolarize and contract is intrinsic (no nerves required), the healthy heart *is* supplied with autonomic nerve fibers that alter its basic rhythm. In this module, we examine how the basic rhythm is generated and modified.

Setting the Basic Rhythm: The Intrinsic Conduction System

The independent, but coordinated, activity of the heart is a function of (1) the presence of gap junctions, and (2) the activity of the heart's "in-house" conduction system. The **intrinsic cardiac conduction system** consists of noncontractile cardiac cells specialized to initi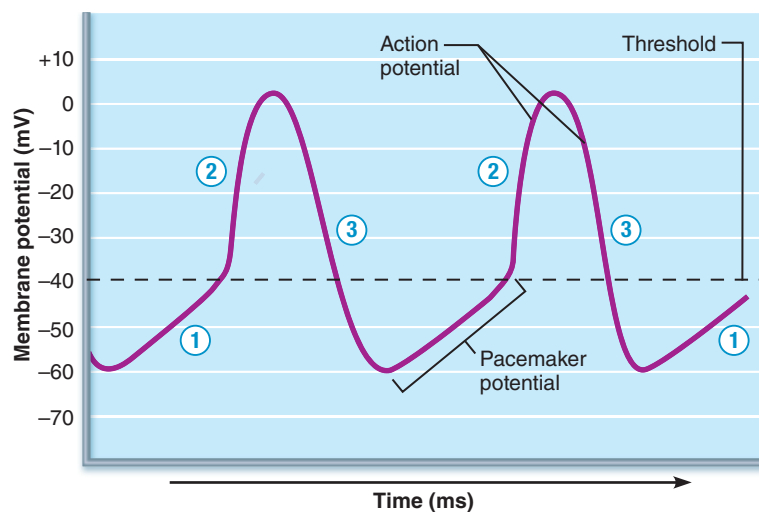ate and distribute impulses throughout the heart, so that it depolarizes and contracts in an orderly, sequential manner. Let's look at how this system works.

Action Potential Initiation by Pacemaker Cells

Unstimulated contractile cells of the heart (and neurons and skeletal muscle fibers) maintain a stable resting membrane potential. However, about 1% of cardiac fibers are autorhythmic ("self-rhythmic") **cardiac pacemaker cells**, having the special ability to depolarize spontaneously and thus pace the heart. Pacemaker cells are a part of the intrinsic conduction system. They have an *unstable resting potential* that continuously depolarizes, drifting slowly toward threshold. These spontaneously changing membrane potentials, called **pacemaker potentials** or **prepotentials**, initiate the action potentials that spread throughout the heart to trigger its rhythmic contractions. Let's look at the three parts of an action potential in typical pacemaker cells as shown in **Figure 18.12**.

① **Pacemaker potential.** The pacemaker potential is due to the special properties of the ion channels in the sarcolemma. In these cells, hyperpolarization at the end of an action potential both closes K^+ channels and opens slow Na^+ channels. The Na^+ influx alters the balance between K^+ loss and Na^+ entry, and the membrane interior becomes less and less negative (more positive).

② **Depolarization.** Ultimately, at threshold (approximately −40 mV), **Ca^{2+} channels** open, allowing explosive entry of Ca^{2+} from the extracellular space. As a result, in pacemaker cells, it is the influx of Ca^{2+} (rather than Na^+) that produces the rising phase of the action potential and reverses the membrane potential.

③ **Repolarization.** Ca^{2+} channels inactivate. As in other excitable cells, the falling phase of the action potential and repolarization reflect opening of K^+ channels and K^+ efflux from the cell.

Once repolarization is complete, K^+ channels close, K^+ efflux declines, and the slow depolarization to threshold begins again.

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① **Pacemaker potential** This slow depolarization is due to both opening of Na^+ channels and closing of K^+ channels. Notice that the membrane potential is never a flat line.

② **Depolarization** The action potential begins when the pacemaker potential reaches threshold. Depolarization is due to Ca^{2+} influx through Ca^{2+} channels.

③ **Repolarization** is due to Ca^{2+} channels inactivating and K^+ channels opening. This allows K^+ efflux, which brings the membrane potential back to its most negative voltage.

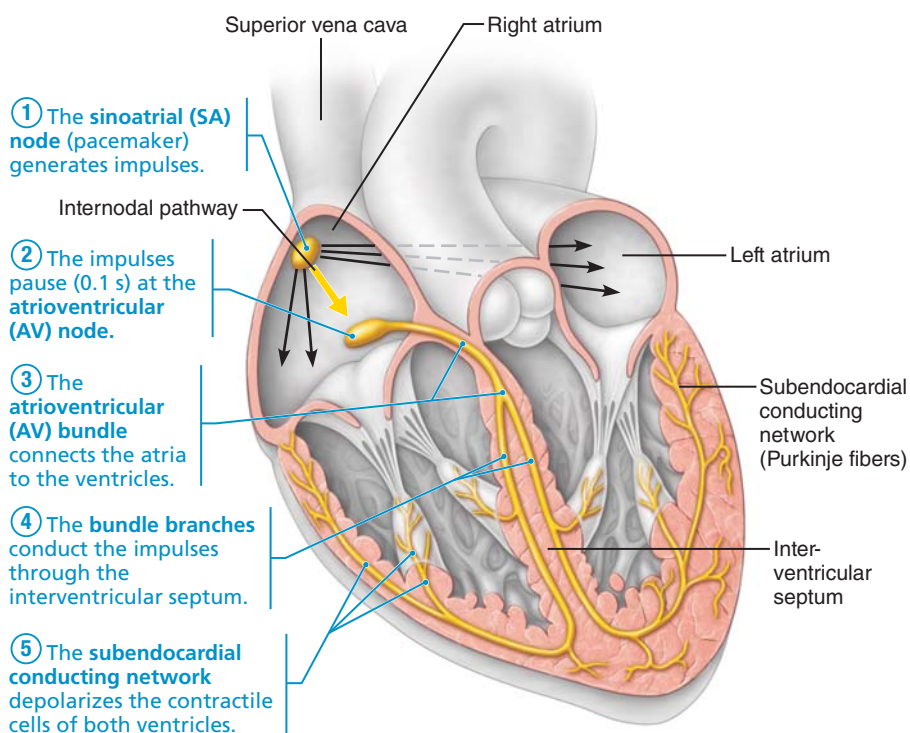**Figure 18.12 Pacemaker and action potentials of typical cardiac pacemaker cells.**

Sequence of Excitation

Typical cardiac pacemaker cells are found in the sinoatrial (si″no-a′tre-al) and atrioventricular nodes (**Figure 18.13**). In addition, cells of the atrioventricular bundle, right and left bundle branches, and subendocardial conducting network (Purkinje fibers) can sometimes act as pacemakers. Impulses pass across the heart in order from ① to ⑤ following the yellow pathway in Figure 18.13a.
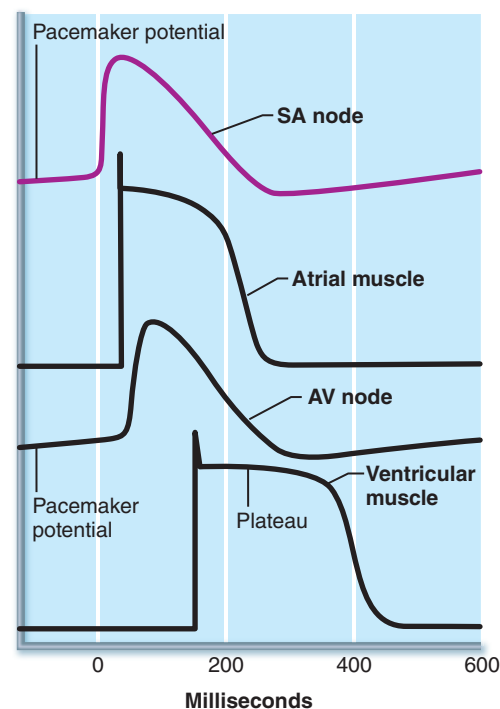
① **Sinoatrial (SA) node.** The crescent-shaped **sinoatrial node** is located in the right atrial wall, just inferior to the entrance of the superior vena cava. A minute cell mass with a mammoth job, the SA node typically generates impulses about 75 times every minute. The SA node sets the pace for the heart as a whole because no other region of the conduction system or the myocardium has a faster depolarization rate. For this reason, it is the heart's **pacemaker**, and its characteristic rhythm, called **sinus rhythm**, determines heart rate.

② **Atrioventricular (AV) node.** From the SA node, the depolarization wave spreads via gap junctions throughout the atria and via the *internodal pathway* to the **atrioventricular node**, located in the inferior portion of the interatrial septum immediately above the tricuspid valve. At the AV node, the impulse is delayed for about 0.1 second, allowing the atria to respond and complete their contraction before the ventricles contract. This delay reflects the smaller diameter of the fibers here and the fact that they have fewer gap junctions for current flow. Consequently, the AV node conducts impulses more slowly than other parts of the system, just as traffic slows when cars are forced to merge from four lanes into two. Once through the AV node, the signaling impulse passes rapidly through the rest of the system.

③ **Atrioventricular (AV) bundle.** From the AV node, the impulse sweeps to the **atrioventricular bundle** (also called the **bundle of His**) in the superior part of the interventricular septum. Although the atria and ventricles are adjacent to each other, they are *not* connected by gap junctions. The AV bundle is the *only* electrical connection between them. The fibrous cardiac skeleton is nonconducting and insulates the rest of the AV junction.

④ **Right and left bundle branches.** The AV bundle persists only briefly before splitting into two pathways—the **right** and **left bundle branches**, which course along the interventricular septum toward the heart apex.

⑤ **Subendocardial conducting network.** Essentially long strands of barrel-shaped cells with few myofibrils, the **subendocardial conducting network**, also called **Purkinje fibers** (pur-kin′je), completes the pathway through the interventricular septum, penetrates into the heart apex, and then turns superiorly into the ventricular walls. The bundle branches excite the septal cells, but the bulk of ventricular depolarization depends on the large fibers of the conducting network and, ultimately, on cell-to-cell transmission of the impulse via gap junctions between the ventricular muscle cells. Because the left ventricle is much larger than the

① The **sinoatrial (SA) node** (pacemaker) generates impulses.

② The impulses pause (0.1 s) at the **atrioventricular (AV) node.**

③ The **atrioventricular (AV) bundle** connects the atria to the ventricles.

④ The **bundle branches** conduct the impulses through the interventricular septum.

⑤ The **subendocardial conducting network** depolarizes the contractile cells of both ventricles.

(a) Anatomy of the intrinsic conduction system showing the sequence of electrical excitation

(b) Comparison of action potential shape at various locations

Figure 18.13 Intrinsic cardiac conduction system and action potential succession during one heartbeat.

right, the subendocardial conducting network is more elaborate in that side of the heart.

The total time between initiation of an impulse by the SA node and depolarization of the last of the ventricular muscle cells is approximately 0.22 s (220 ms) in a healthy human heart.

Ventricular contraction almost immediately follows the ventricular depolarization wave. The wringing motion of contraction begins at the heart apex and moves toward the atria, following the direction of the excitation wave through the ventricle walls. This contraction ejects some of the contained blood *superiorly* into the large arteries leaving the ventricles.

The various cardiac pacemaker cells have different rates of spontaneous depolarization. The SA node normally drives the heart at a rate of 75 beats per minute. Without SA node input, the AV node would depolarize only about 50 times per minute. Without input from the AV node, the atypical pacemakers of the AV bundle and the subendocardial conducting network would depolarize only about 30 times per minute. Note that these slower pacemakers cannot dominate the heart unless faster pacemakers stop functioning.

The cardiac conduction system coordinates and synchronizes heart activity. Without it, impulses would travel much more slowly. This slower rate would allow some muscle fibers to contract long before others, reducing pump effectiveness.

HOMEOSTATIC IMBALANCE 18.4
CLINICAL

Defects in the intrinsic conduction system can cause irregular heart rhythms, or **arrhythmias** (ah-rith′me-ahz). They may also cause uncoordinated atrial and ventricular contractions, or even **fibrillation**, a condition of rapid and irregular or out-of-phase contractions in which control of heart rhythm is taken away from the SA node by rapid activity in other heart regions. The heart in fibrillation has been compared with a squirming bag of worms. Fibrillating ventricles are useless as pumps; and unless the heart is defibrillated quickly, circulation stops and brain death occurs.

Defibrillation is accomplished by electrically shocking the heart, which interrupts its chaotic twitching by depolarizing the entire myocardium. The hope is that "with the slate wiped clean" the SA node will begin to function normally and sinus rhythm will be reestablished. Implantable cardioverter defibrillators (ICDs) can continually monitor heart rhythms and slow an abnormally fast heart rate or emit an electrical shock if the heart begins to fibrillate.

A defective SA node may have several consequences. An **ectopic focus** (ek-top′ik) (an abnormal pacemaker) may appear and take over the pacing of heart rate, or the AV node may become the pacemaker. The pace set by the AV node (**junctional rhythm**) is 40 to 60 beats per minute, slower than sinus rhythm but still adequate to maintain circulation.

Occasionally, ectopic pacemakers appear even when the SA node is operating normally. A small region of the heart becomes hyperexcitable, sometimes as a result of too much caffeine or nicotine, and generates impulses more quickly than the SA node. This leads to a *premature contraction* or **extrasystole** (ek″strah-sis′to-le) before the SA node initiates the next contraction. Then, because the heart has a longer time to fill, the next (normal) contraction is felt as a thud. As you might guess, premature *ventricular* contractions (PVCs) are most problematic.

The only route for impulse transmission from atria to ventricles is through the AV node, AV bundle, and bundle branches. Damage to any of these structures interferes with the ability of the ventricles to receive pacing impulses, and may cause **heart block**. In total heart block, no impulses get through and the ventricles beat at their intrinsic rate, which is too slow to maintain adequate circulation. In partial heart block, only some of the atrial impulses reach the ventricles. In both cases, artificial pacemakers are implanted to recouple the atria to the ventricles as necessary. These programmable devices speed up in response to increased physical activity just as a normal heart would, and many can send diagnostic information to the patient's doctor. ✚

Modifying the Basic Rhythm: Extrinsic Innervation of the Heart

Although the intrinsic conduction system sets the basic heart rate, fibers of the autonomic nervous system modify the march-like beat and introduce a subtle variability from one beat to the next. The sympathetic nervous system (the "accelerator") increases both the rate and the force of the heartbeat. Parasympathetic activation (the "brakes") slows the heart. We explain these neural controls later—here we discuss the anatomy of the nerve supply to the heart.

The cardiac centers are located in the medulla oblongata. The **cardioacceleratory center** projects to sympathetic neurons in the T_1–T_5 level of the spinal cord. These preganglionic neurons, in turn, synapse with postganglionic neurons in the cervical and upper thoracic sympathetic trunk (**Figure 18.14**). From there, postganglionic fibers run through the cardiac plexus to the heart where they innervate the SA and AV nodes, heart muscle, and coronary arteries.

The **cardioinhibitory center** sends impulses to the para-sympathetic dorsal vagus nucleus in the medulla, which in turn sends inhibitory impulses to the heart via branches of the vagus nerves. Most parasympathetic postganglionic motor neurons lie in ganglia in the heart wall and their fibers project most heavily to the SA and AV nodes.
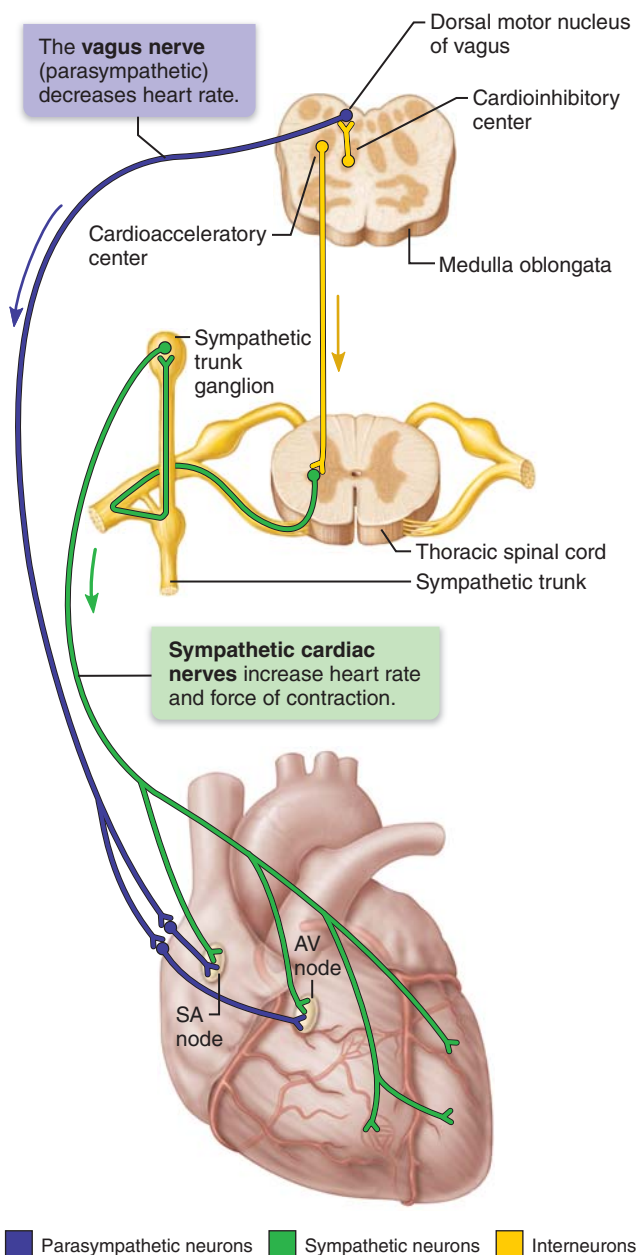
Action Potentials of Contractile Cardiac Muscle Cells

The bulk of heart muscle is composed of *contractile muscle fibers* responsible for the heart's pumping activity. As we have seen, the sequence of events leading to contraction of these cells is similar to that in skeletal muscle fibers. However, the action potential has a characteristic "hump" or *plateau* as shown in **Figure 18.15**.
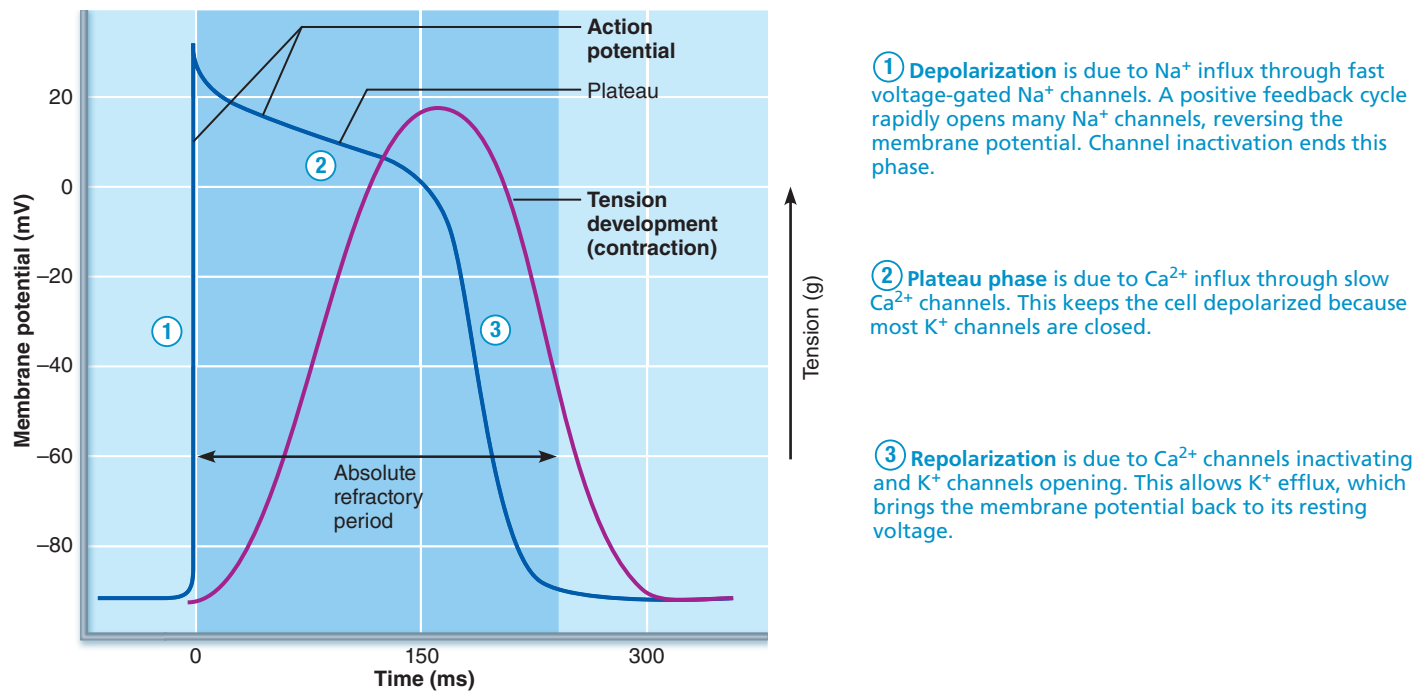
① Depolarization opens a few **fast voltage-gated Na^+ channels** in the sarcolemma, allowing extracellular Na^+ to enter. This influx initiates a positive feedback cycle that causes the rising phase of the action potential (and reversal of the membrane potential from −90 mV to nearly +30 mV). The period of Na^+ influx is very brief, because the sodium channels quickly inactivate and the Na^+ influx stops.

② When Na^+-dependent membrane depolarization occurs, the voltage change also opens channels that allow Ca^{2+} to enter from the extracellular fluid. These channels are called **slow Ca^{2+} channels** because their opening is delayed a bit. The Ca^{2+} surge across the sarcolemma prolongs the depolarization, producing a **plateau** in the action potential tracing. Not many voltage-gated K^+ channels are open yet, so the plateau is prolonged. As long as Ca^{2+} is entering, the cells continue to contract. Notice in Figure 18.15 that muscle tension develops during the plateau, and peaks just after the plateau ends.

③ After about 200 ms, the slope of the action potential tracing falls rapidly. This repolarization results from inactivation of Ca^{2+} channels and opening of voltage-gated K^+ channels. The rapid loss of potassium from the cell through K^+ channels restores the resting membrane potential. During repolarization, Ca^{2+} is pumped back into the SR and the extracellular space.

Notice that the action potential and contractile phase lasts much longer in cardiac muscle than in skeletal muscle. In skeletal muscle, the action potential typically lasts 1–2 ms and the contraction (for a single stimulus) 15–100 ms. In cardiac muscle, the action potential lasts 200 ms or more (because of the plateau), and tension development persists for 200 ms or more. This long plateau in cardiac muscle has two consequences:

- It ensures that the contraction is sustained so that blood is ejected efficiently from the heart.
- It ensures that there is a long refractory period, so that tetanic contractions cannot occur and the heart can fill again for the next beat.

The **vagus nerve** (parasympathetic) decreases heart rate.

Dorsal motor nucleus of vagus

Cardioinhibitory center

Cardioacceleratory center

Medulla oblongata

Sympathetic trunk ganglion

Thoracic spinal cord

Sympathetic trunk

Sympathetic cardiac nerves increase heart rate and force of contraction.

AV node

SA node

■ Parasympathetic neurons ■ Sympathetic neurons ■ Interneurons

Figure 18.14 Autonomic innervation of the heart.

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① **Depolarization** is due to Na⁺ influx through fast voltage-gated Na⁺ channels. A positive feedback cycle rapidly opens many Na⁺ channels, reversing the membrane potential. Channel inactivation ends this phase.

② **Plateau phase** is due to Ca²⁺ influx through slow Ca²⁺ channels. This keeps the cell depolarized because most K⁺ channels are closed.

③ **Repolarization** is due to Ca²⁺ channels inactivating and K⁺ channels opening. This allows K⁺ efflux, which brings the membrane potential back to its resting voltage.

Figure 18.15 The action potential of contractile cardiac muscle cells. Relationship between the action potential, period of contraction, and absolute refractory period in a single ventricular cell.
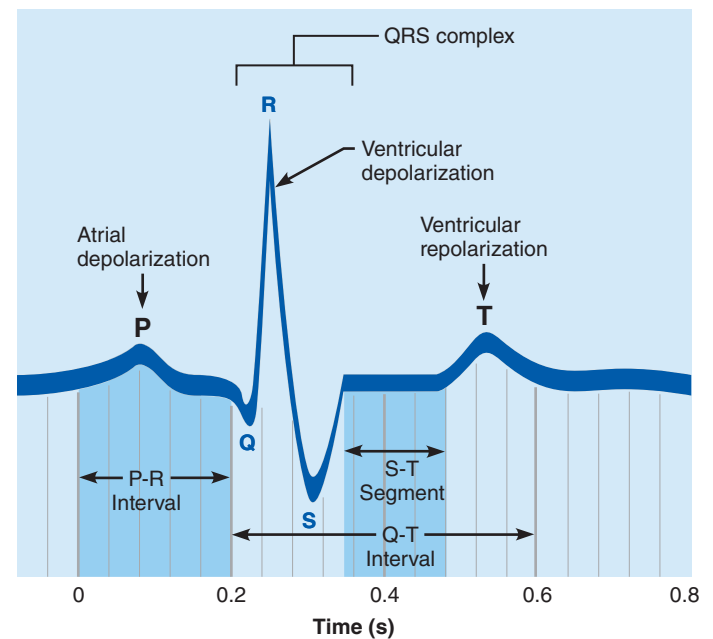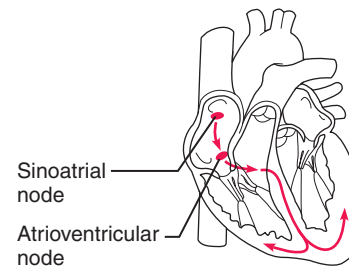
Electrocardiography

The electrical currents generated in and transmitted through the heart spread throughout the body and can be detected with a device called an **electrocardiograph**. An **electrocardiogram (ECG)** is a graphic record of heart activity. An ECG is a composite of all the action potentials generated by nodal and contractile cells at a given time (**Figure 18.16**)—*not*, as sometimes assumed, a tracing of a single action potential.
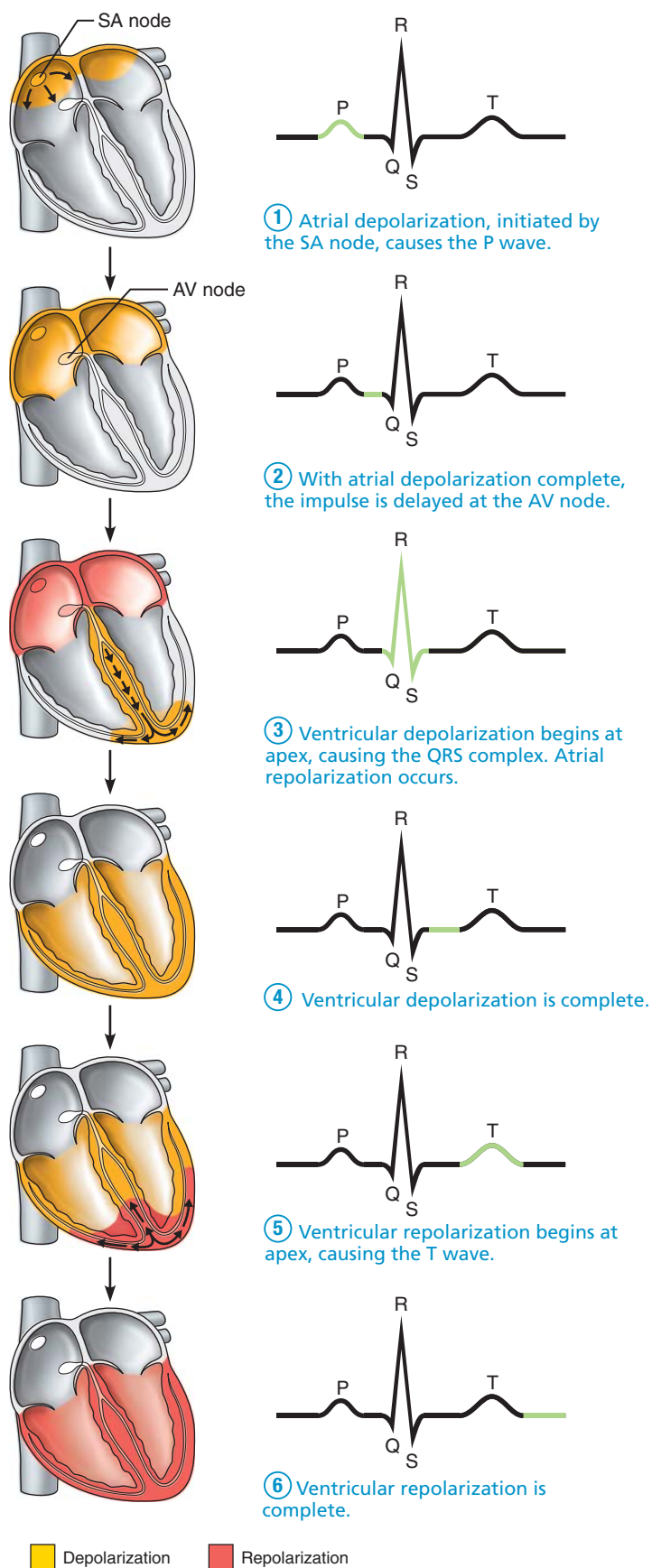
To record an ECG, recording electrodes are placed at various sites on the body surface. In a typical 12-lead ECG, three electrodes form bipolar leads that measure the voltage difference either between the arms or between an arm and a leg, and nine form unipolar leads. Together the 12 leads provide a comprehensive picture of the heart's electrical activity.

A typical ECG has three almost immediately distinguishable waves or *deflections*: the P wave, the QRS complex, and the T wave (Figure 18.16). The first, the small **P wave**, lasts about 0.08 s and results from movement of the depolarization wave from the SA node through the atria. Approximately 0.1 s after the P wave begins, the atria contract.

The large **QRS complex** results from ventricular depolarization and precedes ventricular contraction. It has a complicated shape because the paths of the depolarization waves through the ventricular walls change continuously, producing corresponding changes in current direction. Additionally, the time required for each ventricle to depolarize depends on its size relative to the other ventricle. Average duration of the QRS complex is 0.08 s.

Figure 18.16 An electrocardiogram (ECG) tracing. The labels identify the three normally recognizable deflections (waves) and the important intervals.

① Atrial depolarization, initiated by the SA node, causes the P wave.

② With atrial depolarization complete, the impulse is delayed at the AV node.

③ Ventricular depolarization begins at apex, causing the QRS complex. Atrial repolarization occurs.

④ Ventricular depolarization is complete.

⑤ Ventricular repolarization begins at apex, causing the T wave.

⑥ Ventricular repolarization is complete.

☐ Depolarization   ☐ Repolarization

Figure 18.17 The sequence of depolarization and repolarization of the heart related to the deflection waves of an ECG tracing.

The **T wave,** caused by ventricular repolarization, typically lasts about 0.16 s. Repolarization is slower than depolarization, so the T wave is more spread out and has a lower amplitude (height) than the QRS complex. Because atrial repolarization takes place during the period of ventricular excitation, the wave representing atrial repolarization is normally obscured by the large QRS complex being recorded at the same time.

The **P-R interval** is the time (about 0.16 s) from the beginning of atrial excitation to the beginning of ventricular excitation. If the Q wave is visible (which is often not the case), it marks the beginning of ventricular excitation, and for this reason this interval is sometimes called the **P-Q interval**. The P-R interval includes atrial depolarization (and contraction) as well as the passage of the depolarization wave through the rest of the conduction system.

During the **S-T segment** of the ECG, when the action potentials of the ventricular myocytes are in their plateau phases, the entire ventricular myocardium is depolarized. The **Q-T interval**, lasting about 0.38 s, is the period from the beginning of ventricular depolarization through ventricular repolarization.

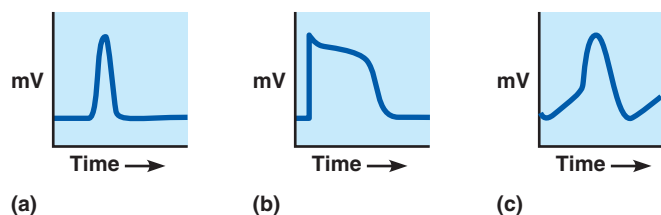**Figure 18.17** relates the parts of an ECG to the sequence of depolarization and repolarization in the heart.

HOMEOSTATIC IMBALANCE 18.5   CLINICAL

In a healthy heart, the size, duration, and timing of the deflection waves tend to be consistent. Changes in the pattern or timing of the ECG may reveal a diseased or damaged heart or problems with the heart's conduction system (**Figure 18.18**). For example, an enlarged R wave hints of enlarged ventricles, an S-T segment that is elevated or depressed indicates cardiac ischemia, and a prolonged Q-T interval reveals a repolarization abnormality that increases the risk of ventricular arrhythmias. +

☑ Check Your Understanding

10. Cardiac muscle cannot go into tetany. Why?

11. Which part of the intrinsic conduction system directly excites ventricular myocardial cells? In which direction does the depolarization wave travel across the ventricles?

12. Describe the electrical event in the heart that occurs during each of the following: (a) the QRS wave of the ECG; (b) the T wave of the ECG; (c) the P-R interval of the ECG.

13. MAKING connections Below are drawings of three different action potentials. Two of these occur in the heart, and one occurs in skeletal muscle (as you learned in Chapter 9).

(a)   (b)   (c)

Which one comes from a contractile cardiac muscle cell? A skeletal muscle cell? A cardiac pacemaker cell? For each one, state which ion is responsible for the depolarization phase and which ion is responsible for the repolarization phase.

For answers, see Answers Appendix.

(a) Infant undergoing an electrocardiogram (ECG)

(b) **Normal sinus rhythm**	(c) **Junctional rhythm**
Normal ECG trace (sinus rhythm)	The SA node is nonfunctional. As a result: • P waves are absent. • The AV node paces the heart at 40–60 beats per minute.

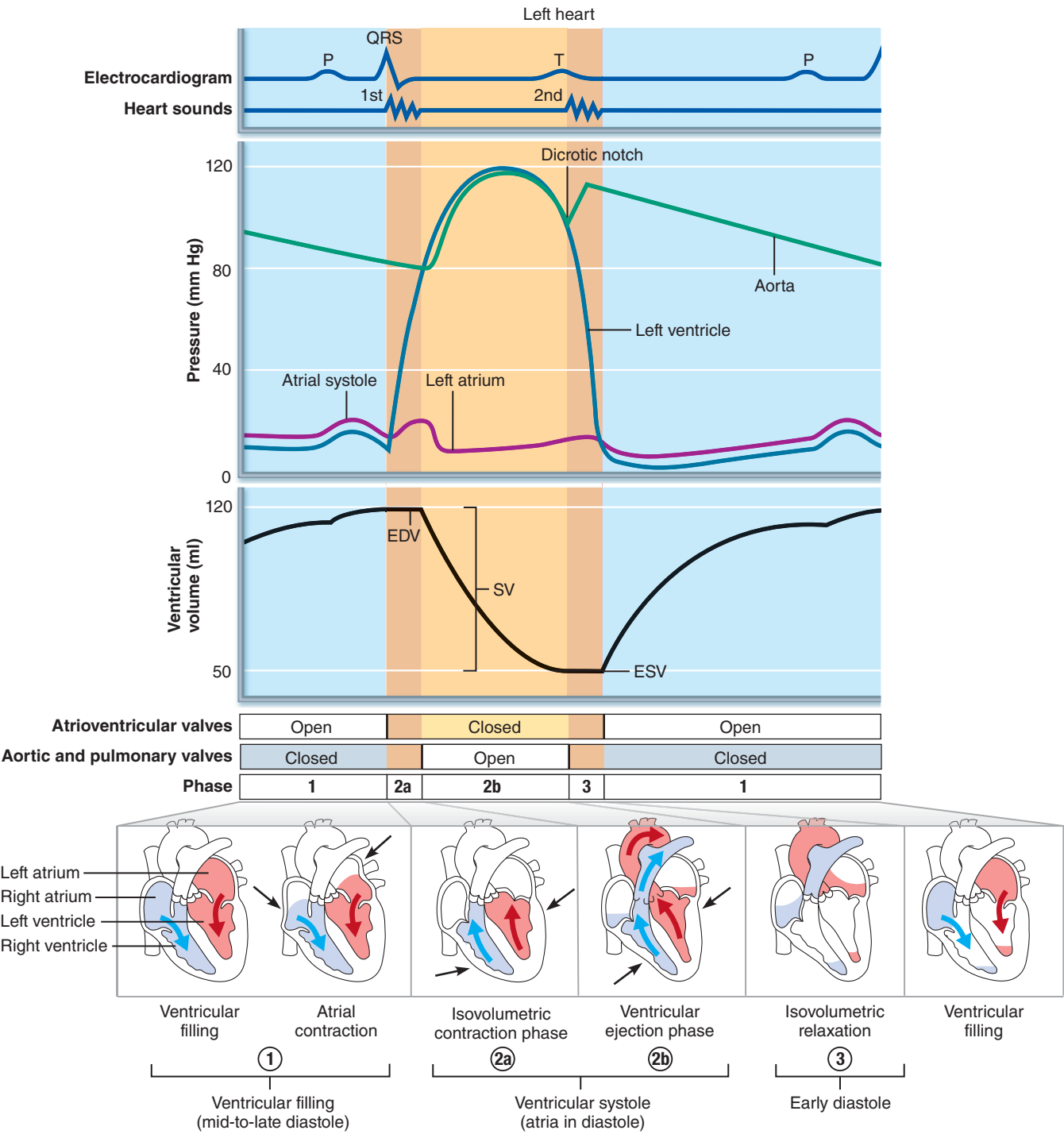| (d) **Second-degree heart block**	(e) **Ventricular fibrillation**
The AV node fails to conduct some SA node impulses. • As a result, there are more P waves than QRS waves. • In this tracing, there are usually two P waves for each QRS wave.	Electrical activity is disorganized. Action potentials occur randomly throughout the ventricles. • Results in chaotic, grossly abnormal ECG deflections. • Seen in acute heart attack and after an electrical shock.

Figure 18.18 Normal and abnormal ECG tracings.

18.6 The cardiac cycle describes the mechanical events associated with blood flow through the heart

→ **Learning Objectives**

☐ Describe the timing and events of the cardiac cycle.
☐ Describe normal heart sounds, and explain how heart murmurs differ.

The heart undergoes some dramatic writhing movements as it alternately contracts, forcing blood out of its chambers, and then relaxes, allowing its chambers to refill with blood. The term **systole** (sis′to-le) refers to these periods of contraction, and **diastole** (di-as′to-le) refers to those of relaxation. The **cardiac cycle** includes *all* events associated with the blood flow through the heart during one complete heartbeat—atrial systole and diastole followed by ventricular systole and diastole. These mechanical events always *follow* the electrical events seen in the ECG.

18

Left heart

Electrocardiogram

Heart sounds

P   QRS   T   P

1st   2nd

Pressure (mm Hg)

120   Dicrotic notch

80   Aorta

40   Left ventricle

Atrial systole   Left atrium

0

Ventricular volume (ml)

120   EDV

SV

50   ESV

Atrioventricular valves   Open   Closed   Open

Aortic and pulmonary valves   Closed   Open   Closed

Phase   1   2a   2b   3   1

Left atrium
Right atrium
Left ventricle
Right ventricle

Ventricular filling   Atrial contraction   Isovolumetric contraction phase   Ventricular ejection phase   Isovolumetric relaxation   Ventricular filling

①   2a   2b   ③

Ventricular filling (mid-to-late diastole)   Ventricular systole (atria in diastole)   Early diastole

Figure 18.19 Summary of events during the cardiac cycle. An ECG tracing (*top*) correlated with graphs of pressure and volume changes (*center*) in the left side of the heart. Pressures are lower in the right side of the heart. Timing of heart sounds is also indicated. (*Bottom*) Events of phases 1 through 3 of the cardiac cycle. (EDV = end diastolic volume, ESV = end systolic volume, SV = stroke volume)

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The cardiac cycle is marked by a succession of pressure and blood volume changes in the heart. Because blood circulates continuously, we must choose an arbitrary starting point for one turn of the cardiac cycle. As shown in **Figure 18.19**, which outlines what happens in the left side of the heart, we begin with

the heart in total relaxation: Atria and ventricles are quiet, and it is mid-to-late diastole.

① **Ventricular filling: mid-to-late diastole.** Pressure in the heart is low, blood returning from the circulation is flowing passively through the atria and the open AV valves into the

ventricles, and the aortic and pulmonary valves are closed. More than 80% of ventricular filling occurs during this period, and the AV valve flaps begin to drift toward the closed position. (The remaining 20% is delivered to the ventricles when the atria contract toward the end of this phase.)

Now the stage is set for atrial systole. Following depolarization (P wave of ECG), the atria contract, compressing the blood in their chambers. This causes a sudden slight rise in atrial pressure, which propels residual blood out of the atria into the ventricles. At this point the ventricles are in the last part of their diastole and have the maximum volume of blood they will contain in the cycle, an amount called the *end diastolic volume* (*EDV*). Then the atria relax and the ventricles depolarize (QRS complex). Atrial diastole persists through the rest of the cycle.

② **Ventricular systole (atria in diastole).** As the atria relax, the ventricles begin contracting. Their walls close in on the blood in their chambers, and ventricular pressure rises rapidly and sharply, closing the AV valves. The split-second period when the ventricles are completely closed chambers and the blood volume in the chambers remains constant as the ventricles contract is the **isovolumetric contraction phase** (i″so-vol″u-met′rik).

Ventricular pressure continues to rise. When it finally exceeds the pressure in the large arteries issuing from the ventricles, the isovolumetric stage ends as the SL valves are forced open and blood rushes from the ventricles into the aorta and pulmonary trunk. During this ventricular ejection phase, the pressure in the aorta normally reaches about 120 mm Hg.

③ **Isovolumetric relaxation: early diastole.** During this brief phase following the T wave, the ventricles relax. Because the blood remaining in their chambers, referred to as the *end systolic volume* (*ESV*), is no longer compressed, ventricular pressure drops rapidly and blood in the aorta and pulmonary trunk flows back toward the heart, closing the SL valves. Closure of the aortic valve raises aortic pressure briefly as backflowing blood rebounds off the closed valve cusps, an event beginning at the **dicrotic notch** shown on the pressure graph. Once again the ventricles are totally closed chambers.

All during ventricular systole, the atria have been in diastole. They have been filling with blood and the intra-atrial pressure has been rising. When blood pressure on the atrial side of the AV valves exceeds that in the ventricles, the AV valves are forced open and ventricular filling, phase ①, begins again. Atrial pressure drops to its lowest point and ventricular pressure begins to rise, completing the cycle.

Assuming the average heart beats 75 times each minute, the cardiac cycle lasts about 0.8 s, with atrial systole accounting for 0.1 s and ventricular systole 0.3 s. The remaining 0.4 s is a period of total heart relaxation, the **quiescent period**.

Notice two important points: (1) Blood flow through the heart is controlled entirely by pressure changes, and (2) blood flows down a pressure gradient through any available opening. The pressure changes, in turn, reflect the alternating contraction

and relaxation of the myocardium and cause the heart valves to open, which keeps blood flowing in the forward direction.
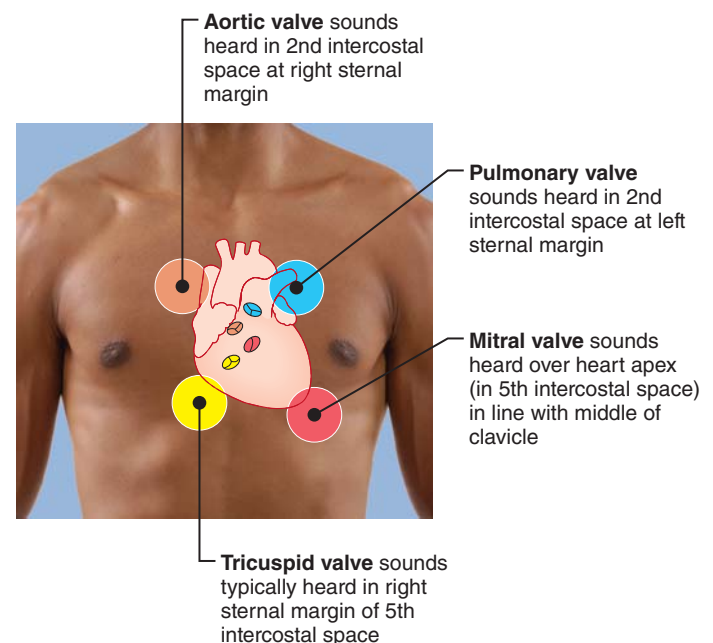
The situation in the right side of the heart is essentially the same as in the left side *except* for pressure. The pulmonary circulation is a low-pressure circulation as evidenced by the much thinner myocardium of its right ventricle. So, typical systolic and diastolic pressures for the pulmonary artery are 24 and 10 mm Hg, compared to systolic and diastolic pressures of 120 and 80 mm Hg, respectively, for the aorta. However, the two sides of the heart eject the same blood volume with each heartbeat.

Heart Sounds

Auscultating (listening to) the thorax with a stethoscope will reveal two sounds during each heartbeat. These **heart sounds**, often described as lub-dup, are associated with the heart valves closing. (The top of Figure 18.19 shows the timing of heart sounds in the cardiac cycle.)

The basic rhythm of the heart sounds is lub-dup, pause, lub-dup, pause, and so on, with the pause indicating the period when the heart is relaxing. The first sound occurs as the AV valves close. It signifies the point when ventricular pressure rises above atrial pressure (the beginning of ventricular systole). The first sound tends to be louder, longer, and more resonant than the second. The second sound occurs as the SL valves snap shut at the beginning of ventricular relaxation (diastole), resulting in a short, sharp sound.

Because the mitral valve closes slightly before the tricuspid valve does, and the aortic SL valve generally snaps shut just before the pulmonary valve, it is possible to distinguish the individual valve sounds by auscultating four specific regions of the thorax (**Figure 18.20**). Notice that these four points, while not directly superficial to the valves (because the sounds take oblique paths

Aortic valve sounds heard in 2nd intercostal space at right sternal margin

Pulmonary valve sounds heard in 2nd intercostal space at left sternal margin

Mitral valve sounds heard over heart apex (in 5th intercostal space) in line with middle of clavicle

Tricuspid valve sounds typically heard in right sternal margin of 5th intercostal space

Figure 18.20 Areas of the thoracic surface where the sounds of individual valves are heard most clearly.

to reach the chest wall), handily define the four corners of the normal heart. Knowing normal heart size and location is essential for recognizing an enlarged (and often diseased) heart.

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Blood flows silently as long as its flow is smooth and uninterrupted. If blood strikes obstructions, however, its flow becomes turbulent and generates abnormal heart sounds, called **heart murmurs**, that can be heard with a stethoscope. Heart murmurs are fairly common in young children (and some elderly people) with perfectly healthy hearts, probably because their heart walls are relatively thin and vibrate with rushing blood.

Most often, however, murmurs indicate valve problems. An *insufficient* or *incompetent* valve fails to close completely. There is a swishing sound as blood backflows or regurgitates through the partially open valve *after* the valve has (supposedly) closed.

A *stenotic* valve fails to open completely and its narrow opening restricts blood flow *through* the valve. In a stenotic aortic valve, for instance, a high-pitched sound or click can be detected when the valve should be wide open during ventricular contraction, but is not. ✚

☑ **Check Your Understanding**

14. The second heart sound is associated with the closing of which valve(s)?

15. If the mitral valve were insufficient, would you expect to hear the murmur (of blood flowing through the valve that should be closed) during ventricular systole or diastole?

16. During the cardiac cycle, there are two periods when all four valves are closed. Name these two periods.

For answers, see Answers Appendix.

18.7 Stroke volume and heart rate are regulated to alter cardiac output

→ **Learning Objectives**

☐ Name and explain the effects of various factors regulating stroke volume and heart rate.

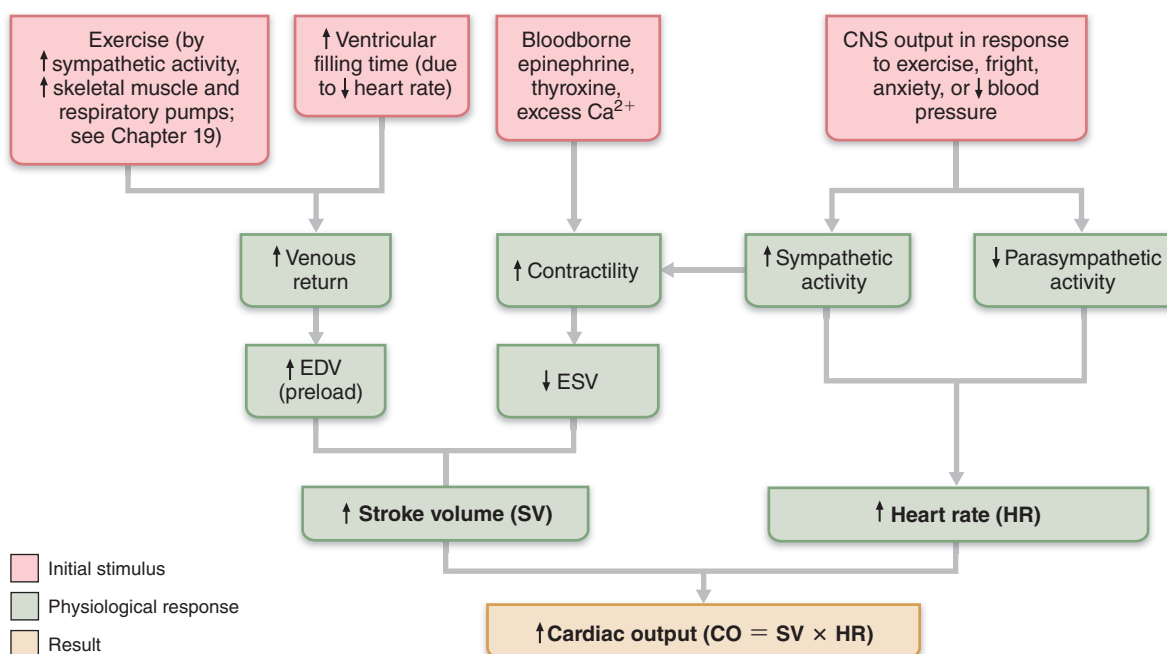☐ Explain the role of the autonomic nervous system in regulating cardiac output.

Cardiac output (CO) is the amount of blood pumped out by *each* ventricle in 1 minute. It is the product of heart rate (HR) and stroke volume (SV). **Stroke volume** is defined as the volume of blood pumped out by one ventricle with each beat. In general, stroke volume correlates with the force of ventricular contraction.

Using normal resting values for heart rate (75 beats/min) and stroke volume (70 ml/beat), the average adult cardiac output can be computed:

$$CO = HR \times SV = \frac{75 \text{ beats}}{\text{min}} \times \frac{70 \text{ ml}}{\text{beat}}$$
$$= \frac{5250 \text{ ml}}{\text{min}} = \frac{5.25 \text{ L}}{\text{min}}$$

The normal adult blood volume is about 5 L (a little more than 1 gallon). As you can see, the entire blood supply passes through each side of the heart once each minute.

Figure 18.21 Factors involved in determining cardiac output. (EDV = end diastolic volume, ESV = end systolic volume)

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Notice that cardiac output varies directly with SV and HR. This means that CO increases when the stroke volume increases or the heart beats faster or both, and it decreases when either or both of these factors decrease.

Cardiac output is highly variable and increases markedly in response to special demands, such as running to catch a bus. **Cardiac reserve** is the difference between resting and maximal CO. In nonathletic people, cardiac reserve is typically four to five times resting CO (20–25 L/min), but CO in trained athletes during competition may reach 35 L/min (seven times resting CO).

How does the heart accomplish such tremendous increases in output? To understand this feat, let's look at how stroke volume and heart rate are regulated. See **Figure 18.21** for an overview of the factors that affect stroke volume and heart rate, and consequently, cardiac output.

Regulation of Stroke Volume

Mathematically, stroke volume (SV) represents the difference between **end diastolic volume (EDV)**, the amount of blood that collects in a ventricle during diastole, and **end systolic volume (ESV)**, the volume of blood remaining in a ventricle *after* it has contracted. The EDV, determined by how long ventricular diastole lasts and by venous pressure, is normally about 120 ml. (An increase in either factor *raises* EDV.) The ESV, determined by arterial blood pressure and the force of ventricular contraction, is approximately 50 ml. (The higher the arterial blood pressure, the higher the ESV.) To figure normal stroke volume, simply plug these values into this equation:

$$SV = EDV - ESV = \frac{120 \text{ ml}}{\text{beat}} - \frac{50 \text{ ml}}{\text{beat}} = \frac{70 \text{ ml}}{\text{beat}}$$

As you can see, each ventricle pumps out about 70 ml of blood with each beat, which is about 60% of the blood in its chambers.
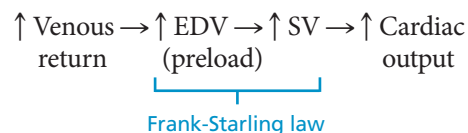
So what is important here—how do we make sense out of this alphabet soup (SV, ESV, EDV)? Although many factors affect SV by altering EDV or ESV, the three most important are *preload, contractility*, and *afterload*. As we describe in detail next, preload affects EDV, whereas contractility and afterload affect the ESV.

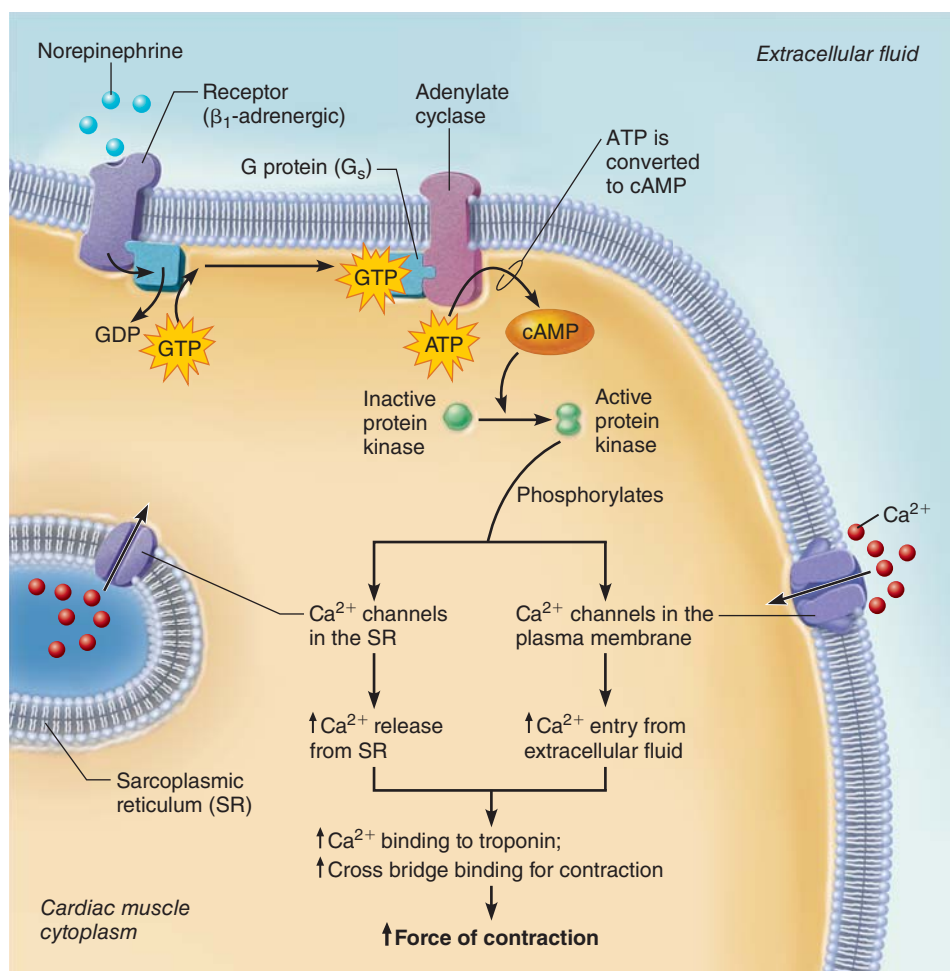### Preload: Degree of Stretch of Heart Muscle

The degree to which cardiac muscle cells are stretched just before they contract, called the **preload**, controls stroke volume. In a normal heart, the higher the preload, the higher the stroke volume. This relationship between preload and stroke volume is called the **Frank-Starling law of the heart**. Recall that at an *optimal length* of muscle fibers (and sarcomeres) (1) the maximum number of active cross bridge attachments is possible between actin and myosin, and (2) the force of contraction is maximal (see Figure 9.19, p. 305). Cardiac muscle, like skeletal muscle, exhibits a *length-tension relationship*.

Resting skeletal muscle fibers are kept near optimal length for developing maximal tension while resting cardiac cells are normally *shorter* than optimal length. As a result, stretching cardiac cells can produce dramatic increases in contractile force. The most important factor stretching cardiac muscle is **venous return**, the amount of blood returning to the heart and distending its ventricles.

Anything that increases venous return increases EDV and, consequently, SV and contraction force (Figure 18.21). Basically:

↑ Venous → ↑ EDV → ↑ SV → ↑ Cardiac
 return (preload) output

Frank-Starling law

Both exercise and increased filling time increase EDV. Exercise increases venous return because both increased sympathetic nervous system activity and the squeezing action of the skeletal muscles compress the veins, decreasing the volume of blood they contain and returning more blood to the heart. During vigorous exercise, SV may double

Figure 18.22 Norepinephrine increases heart contractility via a cyclic AMP second messenger system. Cyclic AMP activates protein kinases that phosphorylate proteins that determine cytoplasmic Ca^{2+} concentrations.

as a result of increased venous return. Conversely, low venous return might result from severe blood loss or an extremely rapid heart rate that does not allow enough time for ventricular filling. Low venous return decreases EDV, causing the heart to beat less forcefully and lowering SV.

Because the systemic and pulmonary circulations are in series, the intrinsic mechanism we just described ensures equal outputs of the two ventricles and proper distribution of blood volume between the two circuits. If one side of the heart suddenly begins to pump more blood than the other, the increased venous return to the opposite ventricle forces that ventricle—through increased cardiac muscle stretch—to pump out an equal volume, preventing backup or accumulation of blood in the circulation.

Contractility

EDV is the major *intrinsic factor* influencing SV, but *extrinsic factors* that increase heart muscle contractility can also enhance SV. **Contractility** is defined as the contractile strength achieved at a given muscle length. Note in Figure 18.21 that contractility is *independent* of muscle stretch and EDV. Contractility rises when more Ca^{2+} enters the cytoplasm from the extracellular

fluid and the SR. Enhanced contractility means more blood is ejected from the heart (greater SV), hence a lower ESV.

Increased sympathetic stimulation increases contractility. As noted on p. 682, sympathetic fibers serve not only the intrinsic conduction system but the entire heart. One effect of norepinephrine or epinephrine binding is to initiate a cyclic AMP second-messenger system that increases Ca^{2+} entry, which in turn promotes more cross bridge binding and enhances ventricular contractility (**Figure 18.22**).

A battery of other chemicals also influence contractility. Substances that increase contractility are called *positive inotropic agents* (*ino* = muscle, fiber). The hormones epinephrine, thyroxine, and glucagon; the drug digitalis; and high levels of extracellular Ca^{2+} are all positive inotropic agents. *Negative inotropic agents,* which impair or decrease contractility, include acidosis (excess H^+), rising extracellular K^+ levels, and drugs called calcium channel blockers.

Afterload: Back Pressure Exerted by Arterial Blood

Afterload is the pressure that the ventricles must overcome to eject blood. It is essentially the back pressure that arterial blood exerts on the aortic and pulmonary valves—about 80 mm Hg in the aorta and 10 mm Hg in the pulmonary trunk.

In healthy individuals, afterload is not a major determinant of stroke volume because it is relatively constant. However, in people with hypertension (high blood pressure), afterload is important because it reduces the ability of the ventricles to eject blood. Consequently, more blood remains in the heart after systole, increasing ESV and reducing stroke volume.

Regulation of Heart Rate

Given a healthy cardiovascular system, SV tends to be relatively constant. However, when blood volume drops sharply or the heart is seriously weakened, SV declines and CO is maintained by increasing HR and contractility. Temporary stressors can also influence HR—and consequently CO—by acting through homeostatic mechanisms induced neurally, chemically, and physically. Factors that increase HR are called *positive chronotropic* (*chrono* = time) factors, and those that decrease HR are *negative chronotropic* factors.

Autonomic Nervous System Regulation of Heart Rate

The autonomic nervous system exerts the most important extrinsic controls affecting heart rate, as shown on the right side

of Figure 18.21. When emotional or physical stressors (such as fright, anxiety, or exercise) activate the sympathetic nervous system, sympathetic nerve fibers release norepinephrine at their cardiac synapses. Norepinephrine binds to β_1-adrenergic receptors in the heart, causing threshold to be reached more quickly. As a result, the SA node fires more rapidly and the heart responds by beating faster.

Sympathetic stimulation also enhances contractility and speeds relaxation. It does this by enhancing Ca^{2+} movements in the contractile cells as we described above and in Figure 18.22. Enhanced contractility lowers ESV, so SV does not decline as it would if only heart *rate* increased. (Remember, when the heart beats faster, there is less time for ventricular filling and so a lower EDV.)

The parasympathetic division opposes sympathetic effects and effectively reduces heart rate when a stressful situation has passed. Parasympathetic-initiated cardiac responses are mediated by acetylcholine, which hyperpolarizes the membranes of its effector cells by *opening* K^+ channels. Because vagal innervation of the ventricles is sparse, parasympathetic activity has little effect on cardiac contractility.

Under resting conditions, both autonomic divisions continuously send impulses to the SA node, but the *dominant* influence is inhibitory. For this reason, the heart is said to exhibit **vagal tone**, and heart rate is generally slower than it would be if the vagal nerves were not innervating it. Cutting the vagal nerves results in an almost immediate increase in heart rate of about 25 beats/min, reflecting the inherent rate (100 beats/min) of the pacemaking SA node.

When sensory input from various parts of the cardiovascular system activates either division of the autonomic nervous system more strongly, the other division is temporarily inhibited. Most such sensory input is generated by *baroreceptors* which respond to changes in systemic blood pressure, as we will discuss in Chapter 19. Another example, the **atrial (Bainbridge) reflex**, is an autonomic reflex initiated by increased venous return and increased atrial filling. Stretching the atrial walls increases heart rate by stimulating both the SA node and the atrial stretch receptors. Stretch receptor activation triggers reflexive adjustments of autonomic output to the SA node, increasing heart rate.

Increased or decreased CO results in corresponding changes to systemic blood pressure, so blood pressure regulation often involves reflexive controls of heart rate. In Chapter 19 we describe in more detail neural mechanisms that regulate blood pressure.

Chemical Regulation of Heart Rate

Chemicals normally present in the blood and other body fluids may influence heart rate.

- **Hormones.** *Epinephrine*, liberated by the adrenal medulla during sympathetic nervous system activation, produces the same cardiac effects as norepinephrine released by the sympathetic nerves: It enhances heart rate and contractility.

  Thyroxine is a thyroid gland hormone that increases metabolic rate and production of body heat. When released in large quantities, it causes a sustained increase in heart rate. Thyroxine acts directly on the heart but also *enhances* the effects of epinephrine and norepinephrine.

- **Ions.** Normal heart function depends on having normal levels of intracellular and extracellular ions. Plasma electrolyte imbalances pose real dangers to the heart.

Reduced Ca^{2+} blood levels (*hypocalcemia*) depress the heart. Conversely, above-normal levels (*hypercalcemia*) increase heart rate and contractility—up to a point. Very high Ca^{2+} levels disrupt heart function and can cause life-threatening arrhythmias.

High or low blood K^+ levels are particularly dangerous and arise in a number of clinical conditions. Excessive K^+ (*hyperkalemia*) alters electrical activity in the heart by depolarizing the resting potential, and may lead to heart block and cardiac arrest. *Hypokalemia* is also life threatening, in that the heart beats feebly and arrhythmically. ✚

Other Factors That Regulate Heart Rate

Age, gender, exercise, and body temperature also influence HR, although they are less important than neural factors. Resting heart rate is fastest in the fetus (140–160 beats/min) and gradually declines throughout life. Average heart rate is faster in females (72–80 beats/min) than in males (64–72 beats/min).

Exercise raises HR by acting through the sympathetic nervous system (Figure 18.21). Exercise also increases systemic blood pressure and routes more blood to the working muscles. However, resting HR in the physically fit tends to be substantially lower than in those who are out of condition, and in trained athletes it may be as slow as 40 beats/min. We explain this apparent paradox below.

Heat increases HR by enhancing the metabolic rate of cardiac cells. This explains the rapid, pounding heartbeat you feel when you have a high fever and also accounts, in part, for the effect of exercise on HR (remember, working muscles generate heat). Cold directly decreases heart rate.

HR varies with changes in activity, but marked and persistent rate changes usually signal cardiovascular disease.

Tachycardia (tak″e-kar′de-ah; "heart hurry") is an abnormally fast heart rate (more than 100 beats/min) that may result from elevated body temperature, stress, certain drugs, or heart disease. Persistent tachycardia is considered pathological because tachycardia occasionally promotes fibrillation.

Bradycardia (brad″e-kar′de-ah; *brady* = slow) is a heart rate slower than 60 beats/min. It may result from low body temperature, certain drugs, or parasympathetic nervous activation. It is a known, and desirable, consequence of endurance training. With physical and cardiovascular conditioning, the heart hypertrophies and SV increases, allowing a lower resting heart rate while still providing the same cardiac output. However, in poorly conditioned people persistent bradycardia may result in grossly inadequate blood circulation to body tissues, and bradycardia is often a warning of brain edema after head trauma. ✚

18

<div style="color:teal">CLINICAL</div>

Homeostatic Imbalance of Cardiac Output

The heart's pumping action ordinarily maintains a balance between cardiac output and venous return. Were this not so, a dangerous damming up of blood (blood congestion) would occur in the veins returning blood to the heart.

In **congestive heart failure (CHF)**, the heart is such an inefficient pump that blood circulation is inadequate to meet tissue needs. This progressively worsening disorder reflects weakening of the myocardium by various conditions that damage it in different ways. The most common causes include:

- **Coronary atherosclerosis.** Coronary atherosclerosis, essentially fatty buildup that clogs the coronary arteries, impairs blood and oxygen delivery to cardiac cells. The heart becomes increasingly hypoxic and begins to contract ineffectively.

- **Persistent high blood pressure.** Normally, pressure in the aorta during diastole is 80 mm Hg, and the left ventricle exerts only slightly over that amount of force to eject blood from its chamber. When aortic diastolic blood pressure rises to 90 mm Hg or more, the myocardium must exert more force to open the aortic valve and pump out the same amount of blood. If afterload is chronically elevated, ESV rises and the myocardium hypertrophies. Eventually, the stress takes its toll and the myocardium becomes progressively weaker.

- **Multiple myocardial infarctions.** A succession of MIs (heart attacks) depresses pumping efficiency because noncontractile fibrous (scar) tissue replaces the dead heart cells.

- **Dilated cardiomyopathy (DCM).** In this condition, the ventricles stretch and become flabby and the myocardium deteriorates, often for unknown reasons. Drug toxicity or chronic inflammation may be involved.

Because the heart is a double pump, each side can initially fail independently of the other. If the left side fails, **pulmonary congestion** occurs. The right side continues to propel blood to the lungs, but the left side does not adequately eject the returning blood into the systemic circulation. Blood vessels in the lungs become engorged with blood, the pressure in them increases, and fluid leaks from the circulation into the lung tissue, causing pulmonary edema. If the congestion is untreated, the person suffocates.

If the right side of the heart fails, **peripheral congestion** occurs. Blood stagnates in body organs, and pooled fluids in the tissue spaces impair the ability of body cells to obtain adequate nutrients and oxygen and rid themselves of wastes. The resulting edema is most noticeable in the extremities (feet, ankles, and fingers).

Failure of one side of the heart puts a greater strain on the other side, and ultimately the whole heart fails. A seriously weakened, or *decompensated*, heart is irreparable. Treatment is directed primarily toward (1) removing the excess leaked fluid with *diuretics* (drugs that increase the kidneys' excretion of Na^+ and water), (2) reducing afterload with drugs that drive down blood pressure, and (3) increasing contractility with digitalis derivatives. Heart transplants and other surgical or mechanical remedies to replace damaged heart muscle provide additional hope for some cardiac patients.

☑ **Check Your Understanding**

17. After running to catch a bus, Josh noticed that his heart was beating faster than normally and was pounding forcefully in his chest. How did his increased HR and SV come about?

18. What problem of cardiac output might ensue if the heart beats far too rapidly for an extended period, that is, if tachycardia occurs? Why?

For answers, see Answers Appendix.

Developmental Aspects of the Heart

The human heart, derived from mesoderm and guided by powerful signaling molecules, begins as two simple endothelial tubes. They quickly fuse to form a single chamber or heart tube that is busily pumping blood by the 22nd day of gestation (**Figure 18.23**).

The tube develops four slightly bulged areas that represent the earliest heart chambers. From tail to head, following the direction of blood flow, the four primitive chambers are the following (Figure 18.23b):

1. **Sinus venosus** (ven-o′sus). This chamber initially receives all the venous blood of the embryo. It will become the smooth-walled part of the right atrium and the coronary sinus. It also gives rise to the sinoatrial node, which "takes the baton" and sets heart rate early in embryonic development.

2. **Atrium.** This embryonic chamber eventually becomes the pectinate muscle–ridged parts of the atria.

3. **Ventricle.** This is the strongest pumping chamber of the early heart and becomes the *left* ventricle.

4. **Bulbus cordis.** This chamber plus its cranial extension, the *truncus arteriosus* (labeled 4a in Figure 18.23b), give rise to the pulmonary trunk, the first part of the aorta, and most of the *right* ventricle.

During the next three weeks, the heart "tube" contorts and major structural changes convert it into a four-chambered organ capable of acting as a double pump—all without missing a beat! After the second month, few changes other than growth occur until birth.
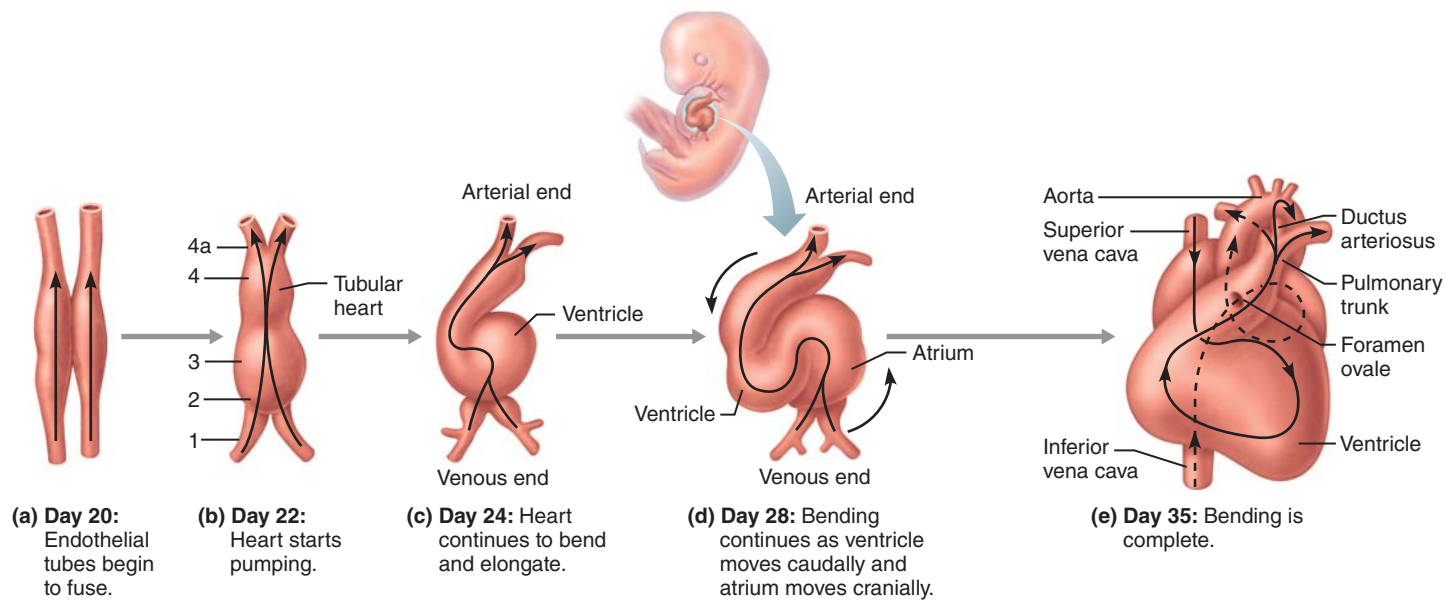
The interatrial septum of the fetal heart is incomplete. The **foramen ovale** (literally, "oval door") connects the two atria and allows blood entering the right heart to bypass the pulmonary circuit and the collapsed, nonfunctional fetal lungs (Figure 18.23e). Another lung bypass, the **ductus arteriosus**, exists between the pulmonary trunk and the aorta. At or shortly after birth, these shunts close, completing the separation between the right and left sides of the heart. We give a more complete description of the fetal and newborn circulation in Chapter 28 (see Figure 28.13).

<div style="color:red">HOMEOSTATIC IMBALANCE 18.9</div> <div style="color:teal">CLINICAL</div>

Building a perfect heart is difficult. Each year about 40,000 infants are born in the U.S. with one or more of 30 different **congenital heart defects**, making them the most common of

(a) **Day 20:** Endothelial tubes begin to fuse.

(b) **Day 22:** Heart starts pumping.

(c) **Day 24:** Heart continues to bend and elongate.

(d) **Day 28:** Bending continues as ventricle moves caudally and atrium moves cranially.
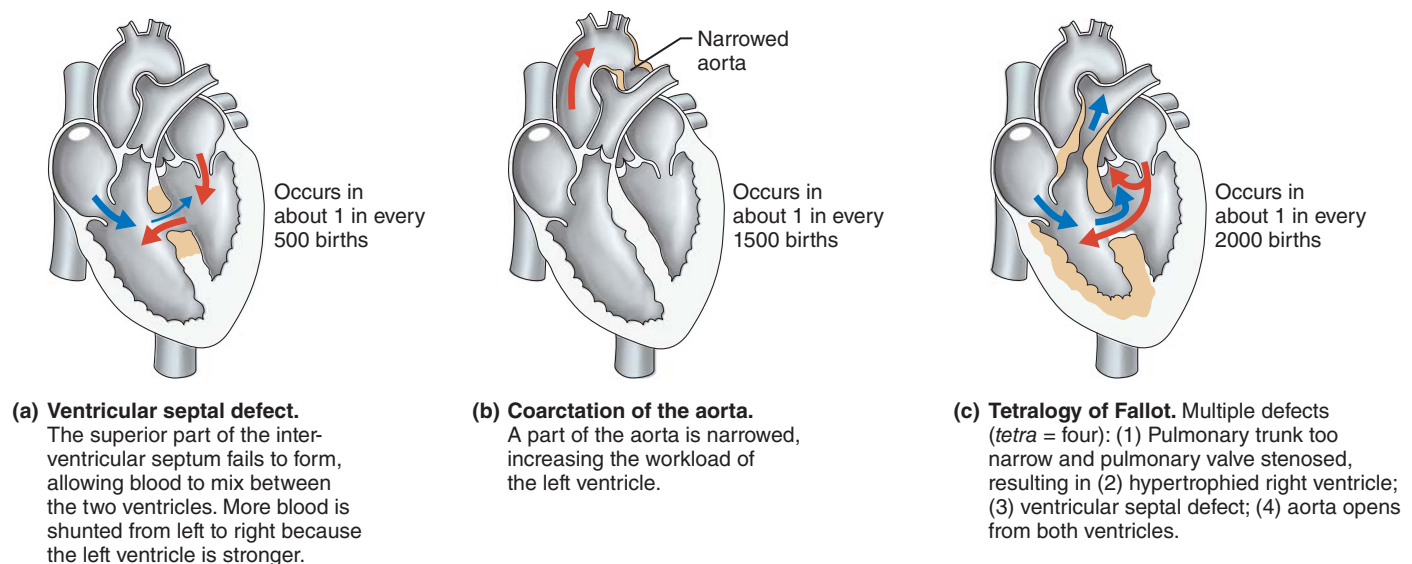
(e) **Day 35:** Bending is complete.

Figure 18.23 Development of the human heart. Ventral views, with the cranial direction toward the top of the figures. Arrows show the direction of blood flow. Days are approximate. **(b)** 1 is the sinus venosus; 2, the atrium; 3, the ventricle; 4, the bulbus cordis; and 4a, the truncus arteriosus.

all birth defects. Some congenital heart problems are traceable to environmental influences, such as maternal infection or drug intake during month 2 when the major events of heart formation occur.

The most prevalent abnormalities produce two basic kinds of disorders in the newborn. They either (1) lead to mixing of oxygen-poor blood with oxygenated blood (so that inadequately oxygenated blood reaches the body tissues) or (2) involve narrowed valves or vessels that greatly increase the workload on the heart.

Examples of the first type of defect are *septal defects* (**Figure 18.24a**) and *patent ductus arteriosus*, in which the connection between the aorta and pulmonary trunk remains open. *Coarctation of the aorta* (Figure 18.24b) is an example of the second type of problem. *Tetralogy of Fallot* (te-tral′o-je ov fal-o′), a serious condition in which the baby becomes cyanotic within minutes of birth, encompasses both types of disorders (Figure 18.24c). Surgery can usually correct these congenital defects. +

(a) **Ventricular septal defect.** The superior part of the inter-ventricular septum fails to form, allowing blood to mix between the two ventricles. More blood is shunted from left to right because the left ventricle is stronger.

Occurs in about 1 in every 500 births

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

Occurs in about 1 in every 1500 births

(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.

Occurs in about 1 in every 2000 births

Figure 18.24 Three examples of congenital heart defects. Tan areas indicate the locations of the defects.

Heart Function throughout Life

In the absence of congenital heart problems, the heart functions admirably throughout a long lifetime for most people. Homeostatic mechanisms are normally so efficient that people rarely notice when the heart is working harder.

In people who exercise regularly and vigorously, the heart gradually adapts to the increased demand by enlarging and becoming more efficient and more powerful. Aerobic exercise also helps clear fatty deposits from blood vessel walls throughout the body, retarding atherosclerosis and coronary heart disease. Barring some chronic illnesses, this beneficial cardiac response to exercise persists into old age.

The key word on benefiting from exercise is *regularity*. Regular exercise gradually enhances myocardial endurance and strength. For example, 30 minutes a day of moderately vigorous activity (brisk walking, biking, or yard work) offers significant health benefits to most adults. However, intermittent vigorous exercise, enjoyed by weekend athletes, may push an unconditioned heart beyond its ability to respond to the unexpected demands and bring on a myocardial infarction.

Because of the incredible amount of work the heart does over the course of a lifetime, certain structural changes are inevitable. Age-related changes include the following:

- **Valve flaps thicken and become sclerotic (stiff).** This change occurs particularly where the stress of blood flow is greatest (mitral valve). For this reason, heart murmurs are more common in elderly people.

- **Cardiac reserve declines.** Although the passing years seem to cause little change in resting heart rate, the aged heart is less able to respond to both sudden and prolonged stressors that demand increased output. In addition, the maximum HR declines as sympathetic control of the heart becomes less efficient. These changes are less of a problem in physically active seniors.

- **Cardiac muscle becomes fibrosed (scarred).** As a person ages, more and more cardiac cells die and are replaced with fibrous tissue. As a result, the heart stiffens and fills less efficiently, reducing stroke volume. The nodes of the heart's conduction system may also become fibrosed, which increases the incidence of arrhythmias and other conduction problems.

- **Atherosclerosis.** The insidious progress of atherosclerosis begins in childhood, but inactivity, smoking, and stress accelerate it. The most serious consequences to the heart are hypertensive heart disease and coronary artery occlusion, both of which increase the risk of heart attack and stroke. Although the aging process itself leads to changes in blood vessel walls that promote atherosclerosis, many investigators feel that diet, not aging, is the single most important contributor to cardiovascular disease. We can lower our risk by consuming less animal fat, cholesterol, and salt.

• • •

The heart is an exquisitely engineered double pump that operates with precision to propel blood into the large arteries leaving its chambers. However, continuous circulation of blood also depends critically on the pressure dynamics in the blood vessels. Chapter 19 considers the structure and function of these vessels and relates this information to the work of the heart to provide a complete picture of cardiovascular functioning.

CHAPTER SUMMARY

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(MAP) For more chapter study tools, go to the Study Area of MasteringA&P®.

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18.1 The heart has four chambers and pumps blood through the pulmonary and systemic circuits (pp. 664–670)

1. The right side of the heart is the pulmonary circuit pump. It pumps blood through the lungs, where the blood picks up oxygen and dumps carbon dioxide. The left side of the heart is the systemic circuit pump. It pumps blood through the body's tissues, supplying them with oxygen and nutrients and removing carbon dioxide.

2. The human heart, about the size of a clenched fist, is located obliquely within the mediastinum of the thorax.

3. The heart is enclosed within a double sac made up of the outer fibrous pericardium and the inner serous pericardium (parietal and visceral layers). The pericardial cavity between the serous layers contains lubricating serous fluid.

4. Layers of the heart wall, from the interior out, are the endocardium, myocardium (reinforced by a fibrous cardiac skeleton), and epicardium (visceral layer of the serous pericardium).

5. The heart has two superior atria and two inferior ventricles. Functionally, the heart is a double pump.

6. Entering the right atrium are the superior vena cava, inferior vena cava, and coronary sinus. Four pulmonary veins enter the left atrium.

7. The right ventricle discharges blood into the pulmonary trunk; the left ventricle pumps blood into the aorta.

18.2 Heart valves make blood flow in one direction (pp. 671–673)

1. The atrioventricular (AV) valves (tricuspid and mitral) prevent backflow into the atria when the ventricles are contracting; the semilunar (SL) valves (pulmonary and aortic) prevent backflow into the ventricles when the ventricles are relaxing.

18.3 Blood flows from atrium to ventricle, and then to either the lungs or the rest of the body (pp. 673–676)

1. Oxygen-poor systemic blood enters the right atrium, passes into the right ventricle, through the pulmonary trunk to the lungs, and back to the left atrium via the pulmonary veins. Oxygen-laden blood entering the left atrium from the lungs flows into the left ventricle and then into the aorta, which provides the functional supply of all body organs. Systemic veins return the oxygen-depleted blood to the right atrium.
2. The right and left coronary arteries branch from the aorta and via their main branches (anterior and posterior interventricular, right marginal, and circumflex arteries) supply the heart itself. Venous blood, collected by the cardiac veins (great, middle, and small), empties into the coronary sinus.
3. Blood delivery to the myocardium occurs during heart relaxation.

iP Cardiovascular System; Topic: Anatomy Review: The Heart, pp. 1–8.

18.4 Intercalated discs connect cardiac muscle fibers into a functional syncytium (pp. 676–679)

1. Cardiac muscle cells are branching, striated, generally uninucleate cells. They contain myofibrils consisting of typical sarcomeres.
2. Intercalated discs containing desmosomes and gap junctions connect adjacent cardiac cells. The myocardium behaves as a functional syncytium because of electrical coupling provided by gap junctions.
3. Ca^{2+} released by the SR and entering from the extracellular space couples the action potential to sliding of the myofilaments. Compared to skeletal muscle, cardiac muscle has a prolonged refractory period that prevents tetany.
4. Cardiac muscle has abundant mitochondria and depends almost entirely on aerobic respiration to form ATP.

18.5 Pacemaker cells trigger action potentials throughout the heart (pp. 679–685)

1. Certain noncontractile cardiac muscle cells exhibit automaticity and rhythmicity and can independently initiate action potentials. Such cells have an unstable resting potential called a pacemaker potential that gradually depolarizes, drifting toward threshold for firing. These cells compose the intrinsic conduction system of the heart.
2. The conduction system of the heart consists of the SA and AV nodes, the AV bundle and bundle branches, and the subendocardial conducting network. This system coordinates the depolarization of the heart and ensures that the heart beats as a unit. The SA node has the fastest rate of spontaneous depolarization and acts as the heart's pacemaker; it sets the sinus rhythm.
3. Defects in the intrinsic conduction system can cause arrhythmias, fibrillation, and heart block.
4. The autonomic nervous system innervates the heart. Cardiac centers in the medulla include the cardioacceleratory center, which projects to the T_1–T_5 region of the spinal cord, which in turn projects to the cervical and upper thoracic sympathetic trunk. Postganglionic fibers innervate the SA and AV nodes and the cardiac muscle fibers. The cardioinhibitory center exerts its influence via the parasympathetic vagus nerves (X), which project to the heart wall. Most parasympathetic fibers serve the SA and AV nodes.
5. The membrane depolarization of contractile myocytes causes opening of sodium channels and allows sodium to enter, which

is responsible for the rising phase of the action potential curve. Depolarization also opens slow Ca^{2+} channels; Ca^{2+} entry prolongs the period of depolarization (creates the plateau).

iP Cardiovascular System; Topic: Cardiac Action Potential, pp. 11–18.

6. An electrocardiogram (ECG) is a graphic representation of the cardiac conduction cycle. The P wave reflects atrial depolarization. The QRS complex indicates ventricular depolarization; the T wave represents ventricular repolarization.

iP2 Cardiovascular System; Topic: Intrinsic Conduction System.

18.6 The cardiac cycle describes the mechanical events associated with blood flow through the heart (pp. 685–688)

1. A cardiac cycle consists of the events occurring during one heartbeat. During mid-to-late diastole, the ventricles fill and the atria contract. Ventricular systole consists of the isovolumetric contraction phase and the ventricular ejection phase. During early diastole, the ventricles are relaxed and are closed chambers until the atrial pressure exceeds the ventricular pressure, forcing the AV valves open. Then the cycle begins again. At a normal heart rate of 75 beats/min, a cardiac cycle lasts 0.8 s.
2. Pressure changes promote blood flow and valve opening and closing.

iP2 Cardiovascular System; Topic: Cardiac Cycle.

3. Normal heart sounds arise chiefly from turbulent blood flow during the closing of heart valves. Abnormal heart sounds, called murmurs, usually reflect valve problems.

18.7 Stroke volume and heart rate are regulated to alter cardiac output (pp. 688–692)

1. Cardiac output, typically 5 L/min, is the amount of blood pumped out by each ventricle in 1 minute. Stroke volume is the amount of blood pumped out by a ventricle with each contraction. Cardiac output = heart rate × stroke volume.
2. Stroke volume depends to a large extent on the degree to which venous return stretches cardiac muscle. Approximately 70 ml, it is the difference between end diastolic volume (EDV) and end systolic volume (ESV). Anything that influences heart rate or blood volume influences venous return, hence stroke volume.
3. Activation of the sympathetic nervous system increases heart rate and contractility; parasympathetic activation decreases heart rate but has little effect on contractility. Ordinarily, the heart exhibits vagal tone.
4. Chemical regulation of the heart is effected by hormones (epinephrine and thyroxine) and ions (particularly potassium and calcium). Ion imbalances severely impair heart activity.
5. Other factors influencing heart rate are age, sex, exercise, and body temperature.
6. Congestive heart failure occurs when the pumping ability of the heart cannot provide adequate circulation to meet body needs. Right heart failure leads to systemic edema; left heart failure results in pulmonary edema.

iP2 Cardiovascular System; Topic: Cardiac Output.

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Developmental Aspects of the Heart (pp. 692–694)

1. The heart begins as a simple (mesodermal) tube that is pumping blood by the fourth week of gestation. The fetal heart has two lung bypasses: the foramen ovale and the ductus arteriosus.
2. Congenital heart defects are the most common of all birth defects. The most common of these disorders lead to inadequate oxygenation of blood or increase the workload of the heart.
3. Age-related changes include sclerosis and thickening of the valve flaps, declines in cardiac reserve, fibrosis of cardiac muscle, and atherosclerosis.
4. Risk factors for cardiac disease include dietary factors, excessive stress, cigarette smoking, and lack of exercise.

REVIEW QUESTIONS

Multiple Choice/Matching

(Some questions have more than one correct answer. Select the best answer or answers from the choices given.)

1. When the semilunar valves are open, which of the following are occurring? **(a)** 2, 3, 5, 6, **(b)** 1, 2, 3, 7, **(c)** 1, 3, 5, 6, **(d)** 2, 4, 5, 7.
   - _____ **(1)** coronary arteries fill
   - _____ **(2)** AV valves are closed
   - _____ **(3)** ventricles are in systole
   - _____ **(4)** ventricles are in diastole
   - _____ **(5)** blood enters aorta
   - _____ **(6)** blood enters pulmonary arteries
   - _____ **(7)** atria contract

2. The portion of the intrinsic conduction system located in the superior interventricular septum is the **(a)** AV node, **(b)** SA node, **(c)** AV bundle, **(d)** subendocardial conducting network.

3. An ECG provides information about **(a)** cardiac output, **(b)** movement of the excitation wave across the heart, **(c)** coronary circulation, **(d)** valve impairment.

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 fact that the left ventricular wall is thicker than the right reveals that it **(a)** pumps a greater volume of blood, **(b)** pumps blood against greater resistance, **(c)** expands the thoracic cage, **(d)** pumps blood through a smaller valve.

6. The chordae tendineae **(a)** close the atrioventricular valves, **(b)** prevent the AV valve flaps from everting, **(c)** contract the papillary muscles, **(d)** open the semilunar valves.

7. In the heart, which of the following apply? **(1)** Action potentials are conducted from cell to cell across the myocardium via gap junctions, **(2)** the SA node sets the pace for the heart as a whole, **(3)** spontaneous depolarization of cardiac cells can occur in the absence of nerve stimulation, **(4)** cardiac muscle can continue to contract for long periods in the absence of oxygen. **(a)** all of the above, **(b)** 1, 3, 4, **(c)** 1, 2, 3, **(d)** 2, 3.

8. The activity of the heart depends on intrinsic properties of cardiac muscle and on neural factors. Thus, **(a)** vagus nerve stimulation of the heart reduces heart rate, **(b)** sympathetic nerve stimulation of the heart decreases time available for ventricular filling, **(c)** sympathetic stimulation of the heart increases its force of contraction, **(d)** all of the above.

9. Freshly oxygenated blood is first received by the **(a)** right atrium, **(b)** left atrium, **(c)** right ventricle, **(d)** left ventricle.

Short Answer Essay Questions

10. Describe the location and position of the heart in the body.

11. Describe the pericardium and distinguish between the fibrous and the serous pericardia relative to histological structure and location.

12. Trace one drop of blood from the time it enters the right atrium until it enters the left atrium. What is this circuit called?

13. **(a)** Describe how heart contraction and relaxation influence coronary blood flow. **(b)** Name the major branches of the coronary arteries, and note the heart regions served by each.

14. The refractory period of cardiac muscle is much longer than that of skeletal muscle. Why is this a desirable functional property?

15. **(a)** Name the elements of the intrinsic conduction system of the heart in order, beginning with the pacemaker. **(b)** What is the important function of this conduction system?

16. Draw a normal ECG pattern. Label and explain the significance of its deflection waves.

17. Define cardiac cycle, and follow the events of one cycle.

18. What is cardiac output, and how is it calculated?

19. Discuss how the Frank-Starling law of the heart helps to explain the influence of venous return on stroke volume.

20. **(a)** Describe the common function of the foramen ovale and the ductus arteriosus in a fetus. **(b)** What problems result if these shunts remain patent (open) after birth?

Critical Thinking and Clinical Application Questions

CLINICAL

1. You have been called upon to demonstrate the technique for listening to valve sounds. **(a)** Explain where you would position your stethoscope to auscultate (1) the aortic valve of a patient with severe aortic valve insufficiency and (2) a stenotic mitral valve. **(b)** During which period(s) would you hear these abnormal valve sounds most clearly? (During atrial diastole, ventricular systole, ventricular diastole, or atrial systole?) **(c)** What cues would you use to differentiate between an insufficient and a stenotic valve?

2. Florita Santos, a middle-aged woman, is admitted to the coronary care unit with a diagnosis of left ventricular failure resulting from a myocardial infarction. Her history indicated that she was aroused in the middle of the night by severe chest pain. Her skin is pale and cold, and moist sounds are heard over the lower regions of both lungs. Explain how failure of the left ventricle can cause these signs and symptoms.

3. Hannah, a newborn baby, needs surgery because she was born with an aorta that arises from the right ventricle and a pulmonary trunk that issues from the left ventricle, a condition called transposition of the great vessels. What are the physiological consequences of this defect?

4. Gabriel, a heroin addict, feels tired, is weak and feverish, and has vague aches and pains. Terrified that he has AIDS, he goes to a doctor and is informed that he is suffering not from AIDS, but from a heart murmur accompanied by endocarditis. What is the

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most likely way that Gabriel contracted endocarditis? (Hint: See Related Clinical Terms.)

5. As Cara worked at her dissection, she became frustrated that several of the structures she had to learn about had more than one common name. Provide another name for each of these structures: (a) atrioventricular groove, (b) tricuspid valve, (c) bicuspid valve (give two synonyms), and (d) atrioventricular bundle.

AT THE CLINIC

Related Clinical Terms

Asystole (a-sis'to-le) Situation in which the heart fails to contract.

Cardiac catheterization Diagnostic procedure that involves passing a fine catheter (tubing) through a blood vessel into the heart. Oxygen content of blood, blood flow, and pressures within the heart can be measured. Findings help to detect valve problems, heart deformities, and other heart malfunctions.

Commotio cordis ("concussion of the heart") Situation in which a relatively mild blow to the chest causes heart failure and sudden death because it occurs during a vulnerable interval (2 ms) when the heart is repolarizing. Explains those rare instances when youngsters drop dead on the playing field after being hit in the chest by a ball.

Cor pulmonale (kor pul-mun-nă'le; *cor* = heart, *pulmo* = lung) A condition of right-sided heart failure resulting from elevated blood pressure in the pulmonary circuit (pulmonary hypertension). Acute cases may develop suddenly due to a pulmonary embolism; chronic cases are usually associated with chronic lung disorders such as emphysema.

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 infection or an autoimmune response. Drug addicts may develop endocarditis by injecting themselves with contaminated needles.

Heart palpitation A heartbeat that is unusually strong, fast, or irregular so that the person becomes aware of it; may be caused by certain drugs, emotional stress ("nervous heart"), or heart disorders.

Hypertrophic cardiomyopathy (HCM) The leading cause of sudden death in young athletes, this condition, which is usually inherited, causes the cardiac muscle cells to enlarge, thickening the heart wall. The heart pumps strongly but doesn't relax well during diastole when the heart is filling.

Mitral valve prolapse Valve disorder affecting up to 1% of the population; most often seen in young women. It appears to have a genetic basis resulting in abnormal chordae tendineae or a malfunction of the papillary muscles. One or more of the mitral valve flaps become incompetent and billow into the left atrium during ventricular systole, allowing blood regurgitation. Occasionally requires valve replacement surgery.

Myocarditis (mi"o-kar-di'tis; *myo* = muscle, *card* = heart, *itis* = inflammation) Inflammation of the cardiac muscle layer (myocardium) of the heart; sometimes follows an untreated streptococcal infection in children. May weaken the heart and impair its ability to pump effectively.

Paroxysmal atrial tachycardia (PAT) Bursts of atrial contractions with little pause between them.

Ventricular tachycardia (VT *or* **V-tac)** Rapid ventricular contractions that are not coordinated with atrial activity.

Clinical Case Study
Cardiovascular System: The Heart

Donald Ayers, a 49-year-old male, was the driver of the bus involved in the accident on Route 91. He was brought into the ER with blunt trauma to the chest. Paramedics noted that the driver's seatbelt had broken and that he was found lying under the instrument panel. Initially unresponsive, Mr. Ayers regained consciousness and complained of chest, epigastric, and left upper quadrant pain. Examination revealed mild tachycardia (110 bpm) and a blood pressure of 105/75 mm Hg. An exam 10 minutes later showed a rapid change in blood pressure (80/55 mm Hg) and HR (130 bpm) along with muffled heart sounds, a thready (weak) pulse, and bulging neck veins. Soon after, the patient began to complain of a sudden onset of pain that radiated into his back from the injury site. The patient described the pain as "sharp, stabbing, and tearing" and it continued to increase.

1. Mr. Ayers's pulse is described as "thready." What might this indicate with respect to this patient's stroke volume?

2. Mr. Ayers's HR increased from 110 to 130 bpm. What effect will this have on his cardiac output? Explain your reasoning.

Mr. Ayers's blood pressure continued to drop, so doctors ordered a chest X ray, ECG, and spiral CT scan (a rapid CT technique). These diagnostic tests revealed four fractured ribs, an enlarged mediastinum, and pericardial effusions (fluid in the pericardium) producing cardiac tamponade.

Mr. Ayers was scheduled for emergency surgery.

3. Beginning with the concept of end diastolic volume (EDV), explain the effect that the fluid in the pericardium is having on the stroke volume of Mr. Ayers's heart.

4. Muffled heart sounds are quieter and less distinct. Explain how changes in EDV can result in muffled heart sounds.

5. The final diagnosis in this case is a dissection (tear) of the aorta. From what you know about the anatomy of the heart, where in the aorta do you think the tear is located? Explain your answer.

6. Why did Mr. Ayers's neck veins bulge?

For answers, see Answers Appendix.