CHAPTER OBJECTIVES

1. Describe the basic design of the cardiovascular system and the function of the heart.
2. Describe the structure of the subdivisions of the pericardium and discuss its functions.
3. Identify and describe the epicardium, myocardium, and endocardium of the heart.
4. Identify important differences between cardiac muscle tissue and skeletal muscle tissue.
5. Discuss the structure and function of the fibrous skeleton of the heart.
6. Identify and describe the external form and surface features of the heart.
7. Describe the structural and functional specializations of each chamber of the heart.
8. Identify the major arteries and veins of the pulmonary and systemic circuits that are connected to the heart.
9. Trace the path of blood flow through the heart.
10. Describe the structure and function of each of the heart valves.
11. Locate the coronary blood vessels and identify their origins and major branches.
12. Name and trace the components of the conduction pathway of the heart.
13. Describe the function of the conduction pathway.
14. Discuss the events that take place during the cardiac cycle.
15. Describe the cardiac centers and discuss their functions in regulating the heart.
Every living cell relies on the surrounding interstitial fluid as a source of oxygen and nutrients and as a place for the disposal of wastes. Levels of gases, nutrients, and waste products in the interstitial fluid are kept stable through continuous exchange between the interstitial fluid and the circulating blood. The blood must stay in motion to maintain homeostasis. If blood stops flowing through a tissue, its oxygen and nutrient supplies are exhausted quickly, its capacity to absorb wastes is soon reached, and neither hormones nor white blood cells can get to their intended targets. Thus, all of the functions of the cardiovascular system ultimately depend on the heart, because it is the heart that keeps blood moving. This muscular organ beats approximately 100,000 times each day, propelling blood through the blood vessels. Each year the heart pumps more than 1.5 million gallons of blood, enough to fill 200 train tank cars.

For a practical demonstration of the heart’s pumping abilities, turn on the faucet in the kitchen and open it all the way. To deliver an amount of water equal to the volume of blood pumped by the heart in an average lifetime, that faucet would have to be left on for at least 45 years. Equally remarkable, the volume of blood pumped by the heart can vary widely, between 5 and 30 liters per minute. The performance of the heart is closely monitored and finely regulated by the nervous system to ensure that gas, nutrient, and waste levels in the peripheral tissues remain within normal limits, whether one is sleeping peacefully, reading a book, or involved in a vigorous racquetball game.

We begin this chapter by examining the structural features that enable the heart to perform so reliably, even in the face of widely varying physical demands. We will then consider the mechanisms that regulate cardiac activity to meet the body’s ever-changing needs.

**An Overview of the Cardiovascular System** [Figure 21.1]

Despite its impressive workload, the heart is a small organ; your heart is roughly the size of your clenched fist. The heart’s four muscular chambers, the right and left atria (Ā-trē-ə; singular, atrium “chamber”) and right and left ventricles (VEN-tri-kls; “little belly”), work together to pump blood through a network of blood vessels between the heart and the peripheral tissues. The network can be subdivided into two circuits: the **pulmonary circuit**, which carries carbon dioxide-rich blood from the heart to the gas-exchange surfaces of the lungs and returns oxygen-rich blood to the heart; and the **systemic circuit**, which transports oxygen-rich blood from the heart to the rest of the body’s cells, returning carbon dioxide-rich blood back to the heart. The right atrium receives blood from the systemic circuit, and the right ventricle discharges blood into the pulmonary circuit. The left atrium collects blood from the pulmonary circuit, and the left ventricle ejects blood into the systemic circuit. When the heart beats, the atria contract first, followed by the ventricles. The two ventricles contract at the same time and eject equal volumes of blood into the pulmonary and systemic circuits.

Each circuit begins and ends at the heart. **Arteries** transport blood away from the heart; **veins** return blood to the heart (Figure 21.1). Blood travels through these circuits in sequence. For example, blood returning to the heart in the systemic veins must complete the pulmonary circuit before reentering the systemic arteries. **Capillaries** are small, thin-walled vessels that interconnect the smallest arteries and veins. Capillaries are called **exchange vessels** because their thin walls permit exchange of nutrients, dissolved gases, and waste products between the blood and surrounding tissues.

**The Pericardium** [Figure 21.2]

The heart is located near the anterior chest wall (Figure 21.2a), directly posterior to the sternum in the **pericardial** (per-i-KAR-dé-al) **cavity**, a portion of the ventral body cavity. The pericardial cavity is situated between the pleural cavities, in the mediastinum, which also contains the thymus, esophagus, and trachea. The position of the heart relative to other structures in the mediastinum is shown in Figure 21.2c,d.

The **pericardium** is the serous membrane lining the pericardial cavity. To visualize the relationship between the heart and the pericardial cavity, imagine pushing your fist toward the center of a large balloon (Figure 21.2b). The wall of the balloon corresponds to the pericardium, and your fist is the heart. The pericardium is divided into the **visceral pericardium** (the part of the balloon in contact with your fist) and the **parietal pericardium** (the rest of the balloon). Your wrist, where the balloon folds back upon itself, corresponds to the **base** of the heart (so named because it is where the heart is attached to the major vessels and bound to the mediastinum).

The loose connective tissue of the visceral pericardium, or **epicardium**, is bound to the cardiac muscle tissue of the heart. The serous membrane of the parietal pericardium is reinforced by an outer layer of dense, irregular connective tissue containing abundant collagen fibers. This reinforcing layer is known as the **fibrous pericardium**. Together, the parietal pericardium and the fibrous pericardium form the tough **pericardial sac**. At the base of the heart, the collagen fibers of the fibrous pericardium stabilize...
Figure 21.2 Location of the Heart in the Thoracic Cavity
The heart is situated within the middle portion of the mediastinum, immediately posterior to the sternum. (a) Anterior view of the open chest cavity, showing the position of the heart and major vessels relative to the lungs. The sectional plane indicates the orientation of part (c). (b) Relationships between the heart and the pericardial cavity. The pericardial cavity surrounds the heart like the balloon surrounds the fist (right). (c) Diagrammatic view showing the position of the heart and the location of other organs within the mediastinum. In this sectional view, the heart is shown intact so you can see the orientation of the major vessels. (d) Superior view of a horizontal section through the trunk at the level of vertebra T8.
the positions of the pericardium, heart, and associated vessels in the mediastinum. The slender gap between the opposing parietal and visceral surfaces is the pericardial cavity. This cavity normally contains 10–20 ml of pericardial fluid secreted by the pericardial membranes. Pericardial fluid acts as a lubricant, reducing friction between the opposing surfaces. The moist pericardial lining prevents friction as the heart beats, and the collagen fibers binding the base of the heart to the mediastinum limit movement of the major vessels during a contraction.

Structure of the Heart Wall [Figure 21.3]

A section through the wall of the heart (Figure 21.3a,b) reveals three distinct layers: (1) an outer epicardium (visceral pericardium), (2) a middle myocardium, and (3) an inner endocardium.

1. The **epicardium** is the visceral pericardium; it forms the external surface of the heart. The epicardium is a serous membrane consisting of a mesothelium covering a supporting layer of areolar connective tissue.

2. The **myocardium** consists of multiple, interlocking layers of cardiac muscle tissue, with associated connective tissues, blood vessels, and nerves. The relatively thin atrial myocardium contains layers that form figure-eights as they pass from atrium to atrium. The ventricular myocardium is much thicker, and the muscle orientation changes from layer to layer. Superficial ventricular muscles wrap around both ventricles; deeper muscle layers spiral around and between the ventricles from the attached base toward the free tip, or apex, of the heart (Figure 21.3a–c).

3. The inner surfaces of the heart, including the valves, are covered by a simple squamous epithelium, known as the **endocardium** (en-do-kar-đé-um; endo-, inside). The endocardium is continuous with the endothelium of the attached blood vessels.

Cardiac Muscle Tissue [Figure 21.3b–e]

The unusual histological characteristics of cardiac muscle tissue give the myocardium its unique functional properties. Cardiac muscle tissue was introduced in Chapter 3, and its properties were briefly compared with those of other muscle types. Cardiac muscle cells, or **cardiocytes**, are relatively small, averaging 10–20 μm in diameter and 50–100 μm in length. A typical cardiocyte has a single, centrally placed nucleus (Figure 21.3b–d).

Although they are much smaller than skeletal muscle fibers, cardiac muscle cells resemble skeletal muscle fibers in that each cardiac muscle cell contains organized myofibrils, and the alignment of their sarcomeres produces striations. However, cardiac muscle cells differ from skeletal muscle fibers in several important respects:

1. Cardiac muscle cells are almost totally dependent on aerobic respiration to obtain the energy needed to continue contracting. The sarcoplasm of a cardiac muscle cell thus contains hundreds of mitochondria and abundant reserves of myoglobin (to store oxygen). Energy reserves are maintained in the form of glycogen and lipid inclusions.

2. The relatively short T-tubules of cardiac muscle cells do not form triads with the sarcoplasmic reticulum.

3. The circulatory supply of cardiac muscle tissue is more extensive than that of red skeletal muscle tissue.

4. Cardiac muscle cells contract without instructions from the nervous system; their contractions will be discussed later in this chapter.

5. Cardiac muscle cells are interconnected by specialized cell junctions called **intercalated discs** (Figure 21.3c–e).

The Intercalated Discs [Figure 21.3b–e]

Cardiac muscle cells are connected to neighboring cells at specialized cell junctions known as intercalated (in-TER-ka-lā-ted) discs. Intercalated discs are unique to cardiac muscle tissue (Figure 21.3b–e). The jagged appearance is due to the extensive interlocking of opposing sarcolemmal membranes. At an intercalated disc,

1. The sarcolemmas of two cardiac muscle cells are bound together by desmosomes (maculae adherens). This locks the cells together and helps maintain the three-dimensional structure of the tissue.

2. Myofibrils in these muscle cells anchor firmly to the sarcolemma at the intercalated disc. The intercalated disc thus ties together the myofibrils of adjacent cells. As a result, the two muscle cells “pull together” with maximum efficiency.

3. Cardiac muscle cells are also connected by gap junctions. Ions and small molecules can move between cells at gap junctions, thereby creating a direct electrical connection between the two muscle cells. As a result, the stimulus for contraction—an action potential—can move from one cardiac muscle cell to another as if the sarcolemmas were continuous.

Because cardiac muscle cells are mechanically, chemically, and electrically connected to one another, cardiac muscle tissue functions like a single, enormous muscle cell. The contraction of any one cell will trigger the contraction of several others, and the contraction will spread throughout the myocardium. For this reason, cardiac muscle has been called a **functional syncytium** (sin-SISH-ē-um; “fused mass of cells”).

The Fibrous Skeleton [Figures 21.3b/21.7]

The connective tissues of the heart include large numbers of collagen and elastic fibers (Figure 21.3b). Each cardiac muscle cell is wrapped in a strong but elastic sheath, and adjacent cells are tied together by fibrous cross-links, or “struts.” In turn, each muscle layer has a fibrous wrapping, and fibrous sheets separate the superficial and deep muscle layers. These connective tissue layers are continuous with dense bands of fibroelastic tissue that encircle (1) the bases of the pulmonary trunk and aorta and (2) the valves of the heart. This extensive connective tissue network is called the **fibrous skeleton** of the heart (Figure 21.7).

The fibrous skeleton has the following functions:

1. Stabilizing the positions of the muscle cells and valves in the heart.

2. Providing physical support for the cardiac muscle cells and for the blood vessels and nerves in the myocardium.

3. Distributing the forces of contraction.

4. Reinforcing the valves and helping prevent overexpansion of the heart.

5. Providing elasticity that helps return the heart to its original shape after each contraction.

6. Physically isolating the atrial muscle cells from the ventricular muscle cells; as you will see in a later section, this isolation is vital for the coordination of cardiac contractions.
Figure 21.3  Histological Organization of Muscle Tissue in the Heart Wall
(a) Anterior view of the heart showing several important landmarks.
(b) A diagrammatic section through the heart wall showing the structure of the epicardium, myocardium, and endocardium. (c) and (d) Histological and diagrammatic views of cardiac muscle tissue. Distinguishing characteristics of cardiac muscle cells include (1) small size; (2) a single, centrally placed nucleus; (3) branching interconnections between cells; and (4) the presence of intercalated discs. (e) The structure of an intercalated disc.
Orientation and Superficial Anatomy of the Heart

[Figures 21.2b/21.4/21.5]

Although advertisements and cartoons often show the heart at the center of the chest, a midsagittal section would not cut the heart in half. This is because the heart (1) lies slightly to the left of the midline, (2) sits at an angle to the longitudinal axis of the body, and (3) is rotated toward the left side.

1. The heart lies slightly to the left of the midline: The heart is located within the mediastinum, between the two lungs. Because the heart lies slightly to the left of the midline, the notch within the medial surface of the left lung is considerably deeper than the corresponding notch in the medial surface of the right lung. The base is the broad superior portion of the heart, where the heart is attached to the major arteries and veins of the systemic and pulmonary circuits. The base of the heart includes both the origins of the major vessels and the superior surfaces of the two atria. In terms of our balloon analogy, the base corresponds to the wrist (Figure 21.2b). The base sits posterior to the sternum at the level of the third costal cartilage, centered about 1.2 cm (0.5 in.) to the left side (Figure 21.4). The apex (À-peks) is the inferior, rounded tip of the heart, which points laterally at an oblique angle. A typical adult heart measures approximately 12.5 cm (5 in.) from the attached base to the apex. The apex reaches the fifth intercostal space approximately 7.5 cm (3 in.) to the left of the midline.

2. The heart sits at an oblique angle to the longitudinal axis of the body: The base forms the superior border of the heart. The right border of the heart is formed by the right atrium; the left border is formed by the left ventricle and a small portion of the left atrium. The left border extends to the apex, where it meets the inferior border: The inferior border is formed mainly by the inferior wall of the right ventricle.

3. The heart is rotated slightly toward the left: As a result of this rotation, the anterior surface, or sternocostal (ster-nô-KOS-tal) surface, consists primarily of the right atrium and right ventricle (Figure 21.5a). The posterior and inferior wall of the left ventricle forms much of the sloping posterior surface, or diaphragmatic surface, that extends between the base and the apex of the heart (Figure 21.5b).

The four internal chambers of the heart are associated with grooves or sulci visible on its external surface (Figure 21.5). A shallow interatrial groove separates the two atria, while the deeper coronary sulcus marks the border between the atria and the ventricles. The division between the left and right ventricles is indicated by linear depressions on the anterior surface (the anterior interventricular sulcus) and the posterior surface (the posterior interventricular sulcus). The connective tissue of the epicardium at the coronary and interventricular sulci usually contains substantial amounts of adipose tissue that must be removed to expose the underlying grooves. These sulci also contain the arteries and veins that supply blood to the cardiac muscle of the heart.

The atria and the ventricles have very different functions—the atria receive venous blood that must continue on to the ventricles, whereas the ventricles must propel blood around the systemic and pulmonary circuits. These functional differences are of course linked to external and internal structural differences. Examine Figure 21.5, which details the superficial anatomy of the heart, and note the distinguishing characteristics of the atria and ventricles.
Figure 21.5  **Superficial Anatomy of the Heart**  
(a) Anterior view of the heart and great vessels. In the photo, the pericardial sac has been cut and reflected to expose the heart and great vessels.  
(b) Posterior view of the heart and great vessels; the coronary vessels have been injected with colored latex (see Figure 21.8).
An atrium functions to collect blood returning to the heart and deliver it to the attached ventricle. The functional demands placed on the right and left atria are very similar, and the two chambers look almost identical. The demands placed on the right and left ventricles are very different, and there are significant structural differences between the two.

The Right Atrium [Figures 21.5/21.6a,c]

The right atrium receives oxygen-poor venous blood from the systemic circuit through the superior vena cava (VE-na CĀ-va) and the inferior vena cava (Figures 21.5 and 21.6a,c). The superior vena cava, which opens into the posterior, superior portion of the right atrium, delivers venous blood from the head, neck, upper limbs, and chest. The inferior vena cava, which opens into the posterior and inferior portion of the right atrium, delivers venous blood from the tissues and organs of the abdominal and pelvic cavities, and the lower limbs. The veins of the heart itself, called coronary veins, collect blood from the heart wall and deliver it to the coronary sinus (Figure 21.5b). This collecting vessel opens into the posterior wall of the right atrium, inferior to the opening of the inferior vena cava. (The coronary blood vessels will be described under a separate heading.)

Prominent muscular ridges, the pectinate muscles (pectin, comb), or musculi pectinati, extend along the inner surface of the right auricle and across the adjacent anterior atrial wall. The interatrial septum separates the right and left atria. From the fifth week of embryonic development until birth, there is an oval opening, the foramen ovale, in this septum. (See Embryology Summaries in Chapter 28.) The foramen ovale permits blood flow directly from the right atrium to the left atrium while the lungs are developing and nonfunctional. At birth the lungs begin functioning and the foramen ovale closes; after 48 hours it is permanently sealed. A small depression, the fossa ovalis, persists at this site in the adult heart. Occasionally the foramen ovale does not close, and it remains patent (open). As a result, blood recirculates into the pulmonary circuit, reducing the efficiency of systemic circulation and elevating blood pressure in the pulmonary vessels. This can lead to cardiac enlargement, fluid buildup in the lungs, and eventual heart failure.

The Right Ventricle [Figures 21.5/21.6]

Oxygen-poor venous blood travels from the right atrium into the right ventricle through a broad opening bounded by three fibrous flaps. These flaps, or cusps, form the right atrioventricular (AV) valve, or tricuspid valve (tri-KUS-pid; tri, three) (Figure 21.6). The free edges of the cusps are attached to bundles of collagen fibers, the chordae tendineae (KOR-dē TEN-di-nē-ē; “tendinous cords”). These bundles arise from the papillary (PAP-i-lē-rē) muscles, cone-shaped muscular projections of the inner ventricular surface. The chordae tendineae limit the movement of the cusps and prevent backflow of blood from the right ventricle into the right atrium; the mechanism will be detailed in a later section.

The internal surface of the ventricle contains a series of irregular muscular folds, the trabeculae carneae (tra-BEK-u-lē CAR-nē-ē; carneus, fleshy). The moderator band is a band of ventricular muscle that extends from the interventricular septum, a thick, muscular partition that separates the two ventricles, to the anterior wall of the right ventricle and the bases of the papillary muscles.

The superior end of the right ventricle tapers to a smooth-walled, cone-shaped pouch, the conus arteriosus, which ends at the pulmonary valve (pulmonary semilunar valve). This valve consists of three thick semilunar (half moon–shaped) cusps. As blood is ejected from the right ventricle, it passes...
Figure 21.6  Sectional Anatomy of the Heart
(a) Diagrammatic frontal section through the relaxed heart, showing major landmarks and the path of blood flow through the atria and ventricles (arrows). (b) Photograph of papillary muscles and chordae tendineae supporting the right AV valve. The picture was taken inside the right ventricle, looking toward a light shining from the right atrium. (c) Anterior view of a frontally sectioned heart, showing internal features and valves. The cardiac arteries and veins have been injected with latex; the arteries are red, the veins blue. (d) Horizontal section through the heart at the level of vertebra T8.
through this valve to enter the pulmonary trunk, the start of the pulmonary circuit. The arrangement of cusps in this valve prevents the backflow of blood into the right ventricle when that chamber relaxes. From the pulmonary trunk, blood flows into both the left and right pulmonary arteries (Figures 21.5 and 21.6). These vessels branch repeatedly within the lungs before supplying the pulmonary capillaries, where gas exchange occurs.

**The Left Atrium** [Figures 21.5/21.6a]

From the pulmonary capillaries, the blood, now oxygen-rich, flows into small veins that ultimately unite to form four pulmonary veins, two from each lung. These left and right pulmonary veins empty into the posterior portion of the left atrium (Figures 21.5 and 21.6a). The left atrium lacks pectinate muscles, but it has an auricle. Blood flowing from the left atrium into the left ventricle passes through the left atrioventricular (AV) valve, also known as the mitral (Mi-tral; mitre, a bishop’s hat) valve or the bicuspid (bi-KUS-pid) valve. As the name bicuspid implies, this valve contains a pair of cusps (bi-, two) rather than a trio (tri-, three). The left atrioventricular valve permits the flow of oxygen-rich blood from the left atrium into the left ventricle, but prevents blood flow in the reverse direction.

**The Left Ventricle** [Figures 21.5/21.6a,c,d]

The left ventricle has the thickest wall of any heart chamber. The extra-thick myocardium enables it to develop enough pressure to force blood around the entire systemic circuit; by comparison the right ventricle, which has a relatively thin wall, must push blood to the lungs and then back to the heart, a total distance of only about 30 cm (1 ft). The internal organization of the left ventricle resembles that of the right ventricle (Figure 21.6a,c,d). However, the trabeculae carneae are more prominent than they are in the right ventricle, there is no moderator band, and since the mitral valve has two cusps, there are two large papillary muscles rather than three.

Blood leaves the left ventricle by passing through the aortic valve (aortic semilunar valve) into the ascending aorta. The arrangement of cusps in the aortic valve is the same as in the pulmonary semilunar valve. Saccular dilations of the base of the ascending aorta occur adjacent to each cusp. These sacs, called aortic sinuses, prevent the individual cusps from sticking to the wall of the aorta when the valve opens. The right and left coronary arteries, which deliver blood to the myocardium, originate at the aortic sinus. The aortic valve prevents the backflow of blood into the left ventricle once it has been pumped out of the heart and into the systemic circuit. From the ascending aorta, blood flows on through the aortic arch and into the descending aorta (Figures 21.5 and 21.6a). The pulmonary trunk is attached to the aortic arch by the ligamentum arteriosum, a fibrous band that is the remnant of an important fetal blood vessel. Cardiovascular changes that occur at birth are described in Chapter 22.

**Structural Differences between the Left and Right Ventricles** [Figure 21.6a,c,d]

Anatomical differences between the left and right ventricles are best seen in three-dimensional or sectional views (Figure 21.6a,c,d). The lungs partially enclose the pericardial cavity, and the base of the heart lies between the left and right lungs. As a result, the pulmonary arteries and veins are relatively short and wide, and the right ventricle normally does not need to push very hard to propel blood through the pulmonary circuit. The wall of the right ventricle is relatively thin, and in sectional view it resembles a pouch attached to the massive wall of the left ventricle. When the right ventricle contracts, it moves toward the wall of the left ventricle. This compresses the blood within the right ventricle, and the rising pressure forces the blood through the pulmonary valve and into the pulmonary trunk. This mechanism moves blood very efficiently at relatively low pressures, which are all that one needs to move blood around the pulmonary circuit. Higher pressures would actually be dangerous, because the pulmonary capillaries are very delicate. Pressures as high as those found in systemic capillaries would both damage the pulmonary vessels and force fluid into the alveoli of the lungs.

A comparable pumping arrangement would not be suitable for the left ventricle, because six to seven times as much force must be exerted to propel blood through the systemic circuit. The left ventricle, which has an extremely thick muscular wall, is round in cross section. When the left ventricle contracts, two things happen: The distance between the base and apex decreases, and the diameter of the ventricular chamber decreases. If you imagine the effects of simultaneously squeezing and rolling up the end of a toothpaste tube, you will get the idea. The forces generated are quite powerful, more than enough to force open the aortic valve and eject blood into the ascending aorta. As the powerful left ventricle contracts, it also bulges into the right ventricular cavity. This intrusion improves the efficiency of the right ventricle’s efforts. Individuals whose right ventricular musculature has been severely damaged may continue to survive because of the extra push provided by the contraction of the left ventricle.

**Concept Check**

1. What is the name of the groove separating the atria from the ventricles?
2. What are some external characteristics that distinguish the atria from the ventricles?

See blue "Answers" tab at back of book.

**The Structure and Function of Heart Valves** [Figures 21.6/21.7]

Details of the structure and function of the four heart valves are shown in Figures 21.6 and 21.7.

The atrioventricular (AV) valves are situated between the atria and the ventricles. Each AV valve has four components: (1) a ring of connective tissue that attaches to the fibrous skeleton of the heart; (2) connective tissue cusps, which function to close the opening between the heart chambers; and (3) chordae tendineae that attach the margins of the cusps to (4) the papillary muscles of the heart wall.

There are two semilunar valves guarding the outflow from the two ventricles. These valves get their names from the shape of their three valvules or cusps, which resemble half moon–shaped pockets. The pulmonary valve is found at the exit of the pulmonary trunk from the right ventricle, while the aortic valve is found at the exit of the aorta from the left ventricle.

**Valve Function during the Cardiac Cycle**

The chordae tendineae and papillary muscles associated with the AV valves play an important role in the normal function of the AV valves during the cardiac cycle. During the period of ventricular relaxation (ventricular diastole) the ventricles are filling with blood, the papillary muscles are relaxed, and the open AV valve offers no resistance to the flow of blood from atrium to ventricle. Over this period the semilunar valves are closed; the semilunar valves do not need chordae tendineae because the relative positions of the
Figure 21.7  Valves of the Heart

Red (oxygenated) and blue (deoxygenated) arrows indicate blood flow into or out of a ventricle; black arrows, blood flow into an atrium; and green arrows, ventricular contraction. (a) When the ventricles are relaxed, the AV valves are open and the semilunar valves are closed. The chordae tendineae are loose, and the papillary muscles are relaxed. (b) When the ventricles are contracting, the AV valves are closed and the semilunar valves are open. In the frontal section notice the attachment of the left AV valve to the chordae tendineae and papillary muscles. (c) The aortic valve in the open (left) and closed (right) positions. The individual cusps brace one another in the closed position.
Clinical Note

**Mitral Valve Prolapse** Minor abnormalities in valve shape are relatively common. For example, an estimated 10 percent of normal individuals age 14–30 have some degree of mitral valve prolapse (MVP). In this condition the mitral valve cusps do not close properly. The problem may involve abnormally long (or short) chordae tendineae or malfunctioning papillary muscles. Because the valve does not work perfectly, some regurgitation may occur during left ventricular systole. The surges, swirls, and eddies that occur during regurgitation create a rushing, gurgling sound known as a heart murmur. Most of these individuals are completely asymptomatic, and they live normal, healthy lives unaware of any circulatory malfunction. However, regurgitation may increase the risk of valve infection after dental (or some medical) procedures.

cusps are stable, and the three symmetrical cusps support one another like the legs of a tripod.

When the period of ventricular contraction (ventricular systole) begins, blood leaving the ventricles opens the semilunar valves, while blood moving back toward the atria swings the cusps of the AV valves together. Tension in the papillary muscles and chordae tendineae keeps the cusps from swinging farther and opening into the atria. Thus, the chordae tendineae and papillary muscles are essential to prevent the backflow, or regurgitation, of blood into the atria each time the ventricles contract.

Serious valvular abnormalities can interfere with cardiac function; the timing and intensity of the related heart sounds can provide useful diagnostic information. Physicians use an instrument called a stethoscope (STETH-ə-scop) to listen to normal and abnormal heart sounds. Valve sounds may be muffled as they pass through the pericardium, surrounding tissues, and the chest wall. As a result, the stethoscope placement does not always correspond to the position of the valve under review.

**Coronary Blood Vessels** [Figure 21.8]

The heart works continuously, and cardiac muscle cells require reliable supplies of oxygen and nutrients. The coronary circulation supplies blood to the muscle tissue of the heart. During maximum exertion, the oxygen demand rises considerably, and the blood flow to the heart may increase to nine times that of resting levels.

The coronary circulation ([Figure 21.8]) includes an extensive network of coronary blood vessels. The left and right coronary arteries originate at the base of the ascending aorta, within the aortic sinus, as the first branches of this vessel. Blood pressure here is the highest found anywhere in the systemic circuit, and this pressure ensures a continuous flow of blood to meet the demands of active cardiac muscle tissue.

**The Right Coronary Artery** [Figure 21.8]

The right coronary artery (RCA) branches off the ascending aorta, turns to the right, and passes between the right auricle and the pulmonary trunk. It then continues within the coronary sulcus. Although many variations may occur, the branches of the right coronary artery typically supply blood to (1) the right atrium, (2) a portion of the left atrium, (3) the interatrial septum, (4) the entire right ventricle, (5) a variable portion of the left ventricle, (6) the posteroinferior one-third of the interventricular septum, and (7) portions of the conducting system of the heart. The major branches are shown in Figure 21.8.

1. **Atrial branches**: As it curves across the anterior surface of the heart, the right coronary artery gives rise to atrial branches that supply the myocardium of the right atrium and a portion of the left atrium.

2. **Ventricular branches**: Near the right border of the heart, the right coronary artery usually gives rise to the right marginal branch that extends toward the apex along the anterior surface of the right ventricle. It then continues across the posterior surface of the heart, supplying the posterior interventricular branch, or posterior descending artery, which runs toward the apex within the posterior interventricular sulcus. This branch supplies blood to the interventricular septum and adjacent portions of the ventricles.

3. **Branches to the conducting system**: A small branch near the base of the right coronary artery penetrates the atrial wall to reach the sinoatrial (SA) node, also known as the cardiac pacemaker. A small branch to the atrioventricular (AV) node, another part of the conducting system of the heart, originates from the right coronary artery near the posterior interventricular branch. These nodes and their role in the regulation of the heartbeat will be the topic of a later section.

**The Left Coronary Artery** [Figure 21.8]

The left coronary artery commonly supplies blood to (1) most of the left ventricle, (2) a narrow slip of the right ventricle, (3) most of the left atrium, and (4) the anterior two-thirds of the interventricular septum. As it reaches the anterior surface of the heart, it gives rise to a circumflex branch and an anterior interventricular branch (Figure 21.8). The circumflex branch curves to the left within the coronary sulcus, giving rise to one or more diagonal branches as it curves toward the posterior surface of the heart. It usually gives rise to a left marginal branch, and on reaching the posterior surface of the heart it forms a posterior left ventricular branch. The distal portions of the circumflex artery often meet and fuse with small branches of the right coronary artery. The much larger anterior interventricular branch, or left anterior descending branch, runs along the anterior surface within the anterior interventricular sulcus. This artery supplies the anterior ventricular myocardium and the anterior two-thirds of the interventricular septum. Small branches from the anterior interventricular branch of the left coronary artery are continuous with those of the posterior interventricular branch of the right coronary artery. Such interconnections between arteries are called anastomoses (a-nas-tô-MÔ-ses; anastomosis, outlet). Because the arteries are interconnected in this way, the blood supply to the ventricular muscle remains relatively constant, regardless of pressure fluctuations within the left and right coronary arteries.

**The Cardiac Veins** [Figures 21.5b/21.8a,b]

The great cardiac vein and middle cardiac vein collect blood from smaller veins draining the myocardial capillaries; they deliver this venous blood to the coronary sinus, a large thin-walled vein that lies in the posterior portion of the coronary sulcus (Figures 21.5b and 21.8a,b). As noted earlier in the chapter, the coronary sinus drains into the right atrium inferior to the opening of the inferior vena cava.
Figure 21.8  Coronary Circulation
(a) Coronary vessels supplying the anterior surface of the heart. (b) Coronary vessels supplying the posterior surface of the heart. (c) A cast of the coronary vessels, showing the complexity and extent of the coronary circulation. Coronary vessels are also seen in Figure 21.5. (d) Coronary angiogram showing the coronary arteries, left lateral projection.
**Clinical Note**

**Coronary Artery Disease** The term *coronary artery disease (CAD)* refers to degenerative changes in the coronary circulation. Cardiac muscle fibers need a constant supply of oxygen and nutrients, and any reduction in coronary circulation produces a corresponding reduction in cardiac performance. Such reduced circulatory supply, known as *coronary ischemia* (ìs-KÉ-mé-a), usually results from partial or complete blockage of the coronary arteries. The usual cause is the formation of a fatty deposit, or *plaque*, in the wall of a coronary vessel. The plaque, or an associated thrombus, then narrows the passageway and reduces or stops blood flow. Spasms in the smooth muscles of the vessel wall can further decrease blood flow or even stop it altogether. A variety of imaging procedures can be used to visualize coronary circulation, including *coronary angiography* (Figure 21.8d) and DSA (Digital Subtraction Angiography) scans (Figure 21.9a,b).

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**Figure 21.9** *Coronary Circulation and Clinical Testing*

(a) A color-enhanced DSA image of a healthy heart. The ventricular walls have an extensive circulatory supply. (The atria are not shown.) (b) A color-enhanced DSA image of a damaged heart. Most of the ventricular myocardium is deprived of circulation. (c) Balloon angioplasty can sometimes be used to remove a circulatory blockage. The catheter is guided through the coronary arteries to the site of blockage and inflated to press the soft plaque against the vessel wall. (d) A stent is often inserted after balloon angioplasty to help prevent plaques from recurring. This scan shows a stent in the anterior interventricular branch of the left coronary artery.
Clinical Note (continued)

One of the first symptoms of CAD is often angina pectoris (an-JL-na PEK-tor-iss, angina, pain spasm + pectoris, of the chest). In the most common form of angina, temporary insufficiency of oxygen delivery and ischemia develop when the workload of the heart increases. Although the individual may feel comfortable at rest, any unusual exertion or emotional stress can produce a sensation of pressure, chest constriction, and pain that may radiate from the sternal area to the arms, back, and neck.

Angina can often be controlled by a combination of drug treatment and changes in lifestyle. Lifestyle changes to combat angina include (1) limiting activities known to trigger angina attacks, such as strenuous exercise, and avoiding stressful situations while doing moderate, regular exercise within tolerated limits; (2) stopping smoking; and (3) modifying the diet to lower fat consumption. Medications useful for controlling angina include drugs that block sympathetic stimulation (propranolol or metoprolol); vasodilators, such as nitroglycerin (ni-trō-GLIS-er-in) or atrial natriuretic peptide; or drugs that block calcium movement into the cardiac muscle fibers (calcium channel blockers). Drugs that lower cholesterol and lipid levels in the blood may prevent plaque growth or even cause plaque regression.

Angina can also be treated surgically. A single, soft plaque may be removed with the aid of a slender, elongated catheter (KATH-er-ter). The catheter, a small-diameter tube, is inserted into a large artery (generally the femoral) and guided to the plaque in a coronary artery. A variety of different surgical tools can be slid into the catheter, and the plaque can then be removed with laser beams or chewed to pieces by a miniature version of the Roto-Rooter machine. Debris created during plaque destruction is sucked up by the catheter, preventing blockage of smaller vessels.

In balloon angioplasty (AN-jē-ō-plas-tē; anjeion, vessel), the catheter tip contains an inflatable balloon. Once in position, the balloon is inflated, compressing the plaque against the vessel walls (Figure 21.9c). This procedure works best on small (under 10 mm), soft plaques. Because restenosis, or repeated narrowing, may develop, metal stents, or sleeves, can often be put into the artery to hold it open (Figure 21.9d).

Coronary bypass surgery involves taking a small section from either a small artery (often the internal thoracic artery) or a peripheral vein, such as a branch of the femoral vein, and using it to create a detour around the obstructed portion of a coronary artery. As many as four coronary arteries can be rerouted this way during a single operation. The procedures are named according to the number of vessels repaired, so one speaks of single, double, triple, or quadruple coronary bypass operations. Current recommendations are that coronary bypass surgery should be reserved for cases of severe angina that do not respond to other treatment.

Cardiac veins that empty into the great cardiac vein or the coronary sinus include (1) the posterior vein of the left ventricle, draining the area served by the circumflex artery; (2) the middle cardiac vein, draining the area supplied by the posterior interventricular artery; and (3) the small cardiac vein, which receives blood from the posterior surfaces of the right atrium and ventricle. The anterior cardiac veins, which drain the anterior surface of the right ventricle, empty directly into the right atrium.

The Coordination of Cardiac Contractions [Figure 21.11]

The function of any pump is to develop pressure and move a particular volume of fluid in a specific direction at an acceptable speed. The heart works in cycles of contraction and relaxation, and the pressure within each chamber alternately rises and falls. The AV and semilunar valves help ensure a one-way flow of blood despite these pressure oscillations. Blood will flow out of an atrium only as long as the AV valve is open and atrial pressure exceeds ventricular pressure. Similarly, blood will flow from a ventricle into an arterial trunk only as long as the semilunar valve is open and ventricular pressure exceeds the arterial pressure. The proper functioning of the heart thus depends on proper timing of atrial and ventricular contractions. The elaborate pacemaking and conduction systems normally provide the required timing.

Unlike skeletal muscle, cardiac muscle tissue contracts on its own, in the absence of neural or hormonal stimulation. This inherent ability to generate and conduct impulses is called automaticity or autorhythmicity. (Automaticity is also characteristic of some types of smooth muscle tissue discussed in Chapter 25.) Neural or hormonal stimuli can alter the basic rhythm of contraction, but even a heart removed for a heart transplant will continue to beat unless steps are taken to prevent it.

Each contraction follows a precise sequence: The atria contract first and then the ventricles. If the contractions follow another sequence, the normal pattern of blood flow is disturbed. For example, if the atria and ventricles contract at the same time, the closing of the AV valves prevents blood flow between the atria and ventricles. Cardiac contractions are coordinated by specialized conducting cells, cardiac muscle cells that are incapable of undergoing powerful contractions. There are two distinct populations of these
Nodal cells are responsible for establishing the rate of cardiac contraction, and conducting fibers distribute the contractile stimulus to the general myocardium (Figure 21.11).

### The Sinoatrial and Atrioventricular Nodes [Figure 21.11]

Nodal cells are unusual because their cell membranes spontaneously depolarize to threshold. Nodal cells are electrically coupled to one another, to conducting fibers, and to normal cardiac muscle cells. As a result, when an action potential appears in a nodal cell, it sweeps through the conducting system, reaching all of the cardiac muscle tissue and causing a contraction. In this way, nodal cells determine the heart rate.

Not all nodal cells depolarize at the same rate, and the normal rate of contraction is established by the nodal cells that reach threshold first; the impulse they produce will bring all other nodal cells to threshold. These rapidly depolarizing cells are called pacemaker cells. They are found in the sinoatrial (sī-nō-Á-trē-al) node (SA node), or cardiac pacemaker. The SA node is embedded in the posterior wall of the right atrium, near the entrance of the superior vena cava (Figure 21.11). Isolated pacemaker cells depolarize rapidly and spontaneously, generating 80–100 action potentials per minute.

Each time the SA node generates an impulse, it produces a heartbeat, so theoretically the resting heart rate would be 80–100 beats per minute (bpm). However, any factor that changes either the resting potential or the rate of spontaneous depolarization at the SA node will alter the heart rate. For example, nodal cell activity is affected by the activity of the autonomic nervous system. When acetylcholine (ACh) is released by parasympathetic neurons, it slows the rate of spontaneous depolarization and lowers the heart rate. In contrast, when norepinephrine (NE) is released by sympathetic neurons, the rate of depolarization increases, and the heart rate accelerates. Under normal resting conditions, parasympathetic activity reduces the heart rate from the inherent nodal rate of 80–100 impulses per minute to a more leisurely 70–80 beats per minute.

A number of clinical problems are the result of abnormal pacemaker function. Bradycardia (brād-ē-KAR-dē-a; brady, slow) is the term used to indicate a heart rate that is slower than normal, whereas a faster-than-normal heart rate is termed tachycardia (tak-ē-KAR-dē-a; tachys, swift). Both terms are relative, and in clinical practice the definition varies depending on the normal resting heart rate and conditioning of the individual.

### The Conducting System of the Heart [Figure 21.11]

The cells of the SA node are electrically connected to those of the larger atrioventricular (ā-trē-ō-ven-TRIK-û-lar) node (AV node) through conducting fibers in the atrial walls (Figure 21.11). As the signal for contraction passes from the SA node to the AV node via the internodal pathways, the conducting fibers also pass the contractile stimulus to cardiac muscle cells of both atria. The action potential then spreads across the atrial sur-
CHAPTER 21 · The Cardiovascular System: The Heart

Internodal pathways
Purkinje fibers
Left bundle branch
AV bundle
Right bundle branch
Moderator band

(a) Nodes and conducting fibers

**Figure 21.11 The Conducting System of the Heart**
(a) The stimulus for contraction is generated by pacemaker cells at the SA node. From there, impulses follow three different paths through the atrial walls to reach the AV node. After a brief delay, the impulses are conducted to the bundle of His (AV bundle), and then on to the bundle branches, the Purkinje cells, and the ventricular myocardial cells. (b) The movement of the contractile stimulus through the heart is shown in **STEPS 1–5**.

faces through cell-to-cell contact. The stimulus affects only the atria, because the fibrous skeleton electrically isolates the atrial myocardium from the ventricular myocardium.

The AV node sits within the floor of the right atrium near the opening of the coronary sinus. Due to differences in the shape of the nodal cells, the impulse slows as it passes through the AV node. From there, the impulse travels to the AV bundle, also known as the bundle of His (HISS). This rather massive bundle of conducting fibers travels along the interventricular septum a short distance before dividing into a right bundle branch and a left bundle branch that extend toward the apex and then radiate across the inner surfaces of both ventricles. At this point, Purkinje (pur-KIN-jë) cells (Purkinje fibers) convey the impulses very rapidly to the contractile cells of the ventricular myocardium. The conducting fibers of the moderator band relay the stimulus to the papillary muscles, which tense the chordae tendineae before the ventricles contract.

The stimulus for a contraction is generated at the SA node, and the anatomical relationships among the contracting cells, the nodal cells, and the conducting fibers distribute the impulse so that (1) the atria contract together, before the ventricles, and (2) the ventricles contract together in a wave that begins at the apex and spreads toward the base. When the ventricles contract in this way, blood is pushed toward the base of the heart and out into the aortic and pulmonary trunks.

**Embryology Summary**
For a summary of the development of the cardiovascular system, see Chapter 28 (Embryology and Human Development).
Clinical Note

Cardiac Arrhythmias, Artificial Pacemakers, and Myocardial Infarctions

Cardiac Arrhythmias

There are many different types of cardiac arrhythmias, or abnormal cardiac rhythms, and they range from inconsequential to lethal. Many people have mild cardiac arrhythmias. For example, children and young adults commonly exhibit a quickening of their heart rate during inhalation, and a slowing during exhalation.

Healthy adults may occasionally have premature atrial contractions (PACs) that vary in duration and frequency. In a PAC, the normal atrial rhythm is momentarily interrupted by an “extra” atrial contraction. Stress, caffeine, and various drugs may increase the incidence of PACs, presumably by increasing the permeabilities of the SA pacemakers. The impulse spreads along the conduction pathway normally, and a typical ventricular contraction follows the atrial beat.

If arrhythmias are occasional and brief in duration, they are rarely of any importance. However, if an arrhythmia persists, or occurs frequently, it merits medical attention. In clinical diagnosis, arrhythmias are classified as:

1. Alterations in heart rate, with normal nodal and conducting pathway function. These conditions, which usually indicate abnormal function at the SA node and atria, are often relatively harmless and may go undetected.
2. Abnormal origination or distribution of the cardiac action potential within the ventricles. These conditions are dangerous and potentially lethal.

Alterations in Heart Rate

Tachycardia is usually defined as a heart rate of more than 100 beats per minute. Under some situations, as during exercise or excitement, tachycardia is quite normal. However chronic tachycardia, even at rest, indicates abnormal activity at the cardiac pacemaker. This type of arrhythmia increases the workload on the heart. Cardiac performance suffers at very high heart rates, because the ventricles do not have enough time to refill with blood before the next contraction occurs. Chronic or acute incidents of tachycardia may be controlled by drugs that affect the permeability of pacemaker membranes or block the effects of sympathetic stimulation.

In paroxysmal (par-ok-SIZ-mal) atrial tachycardia, or PAT, a premature atrial contraction triggers a flurry of atrial activity. The ventricles are still able to keep pace, and the heart rate jumps to about 180 beats per minute. In atrial flutter, the atria contract in a coordinated manner, but the contractions occur very frequently. During a bout of atrial fibrillation (fi-bri-L), the impulses move over the atrial surface at rates of perhaps 500 beats per minute. The atrial wall quivers instead of producing an organized contraction. The ventricular rate in atrial flutter or atrial fibrillation cannot follow the atrial rate and may remain within normal limits. Despite the fact that the atria are now essentially nonfunctional, the condition may go unnoticed, especially in older individuals who lead sedentary lives. In chronic atrial fibrillation, blood clots may form near the atrial walls. Pieces of the clot may break off, creating emboli and increasing the risk of stroke. As a result, most people diagnosed with this condition are placed on anticoagulant therapy. PACs, PAT, atrial flutter, and even atrial fibrillation are not considered very dangerous, unless they are prolonged or associated with some more serious indications of cardiac damage, such as coronary artery disease or valve problems.

Abnormal Origination or Conduction of Impulses

These conditions result in abnormal ventricular activity, which directly affects cardiac output. Many of these ventricular arrhythmias are potentially lethal. Because the conduction system functions in one direction only, from atria to ventricle, ventricular arrhythmias are not linked to atrial activities. Premature ventricular contractions (PVCs) occur when a Purkinje cell or ventricular myocardial cell depolarizes to threshold and triggers a premature contraction. The cell responsible for triggering the contraction is called an ectopic pacemaker. The frequency of PVCs can be increased by exposure to epinephrine, to other stimulatory drugs, or to ionic changes that depolarize cardiac muscle cell membranes. The abnormal ventricular contraction is strong, and after each abnormal beat, there is a pause before the next beat. Single PVCs are common and not dangerous, but they can be unsettling if they occur often enough that the individual starts noticing them.

Ectopic pacemaker activity, potentially enhanced by environmental factors, is probably responsible for periods of ventricular tachycardia (defined as four or more PVCs without intervening normal beats). This condition is also known as VT or V-tach. Multiple PVCs and VT often precede the most serious arrhythmia, ventricular fibrillation (VF). During ventricular fibrillation, the cardiac muscle cells are overly sensitive to stimulation, and the impulses are traveling from cell to cell, around and around the ventricular walls. A normal rhythm cannot become established, because the ventricular muscle cells are stimulating one another at such a rapid rate. If untreated, death will occur within minutes; the condition is commonly called cardiac arrest.

Artificial Pacemakers

Bradycardia is usually defined as a heart rate of less than 50 beats per minute. As with tachycardia, bradycardia under certain conditions (deep sleep, for example) is not abnormal. But chronic bradycardia indicates that the heart is unable to respond to commands for increased cardiac output; when the body’s need for oxygen goes up, the heart doesn’t respond by working harder. Symptoms of severe bradycardia include weakness, fatigue, fainting, and confusion. Drug therapies are seldom helpful, but artificial pacemakers can be used with considerable success. Wires are run to the atria, the ventricles, or both, depending on the nature of the problem, and the unit delivers small electrical pulses to stimulate the myocardium. Internal pacemakers are surgically implanted, batteries and all (Figure 21.12). These units last seven to eight years or more before another operation is required to change the battery.

Significant technological advances have been made in artificial pacemakers in the past 10–15 years. Modern pacemakers improve the patient’s quality of life by performing different functions under specific conditions, thanks to the introduction of microprocessors. New, more sophisticated “smart” pacemakers stimulate the atria and ventricles in sequence and may vary the rate of stimulation to adjust to changing circulatory demands, such as during exercise. Others are able to monitor cardiac activity and respond whenever the heart begins to function abnormally.

One type of smart pacemaker was implanted in Vice President Cheney during his term in office. This smart pacemaker is an automatic implantable cardioverter/defibrillator, which is commonly referred to as an AICD. An AICD is a device that continuously monitors the heart patient’s cardiac rhythm. If the AICD detects an abnormally fast heart rhythm, it will automatically pace the heart electrically and attempt to slow the patient’s heart rate. If the abnormal cardiac rhythm persists, the
AICD will deliver a small electrical shock to the heart in an attempt to re-store normal heart rhythm. The patient rarely feels the AICD rapidly pacing the heart in an attempt to return the cardiac rhythm to normal. However, if the electrical shock is used, it is felt as a strong jolt in the chest. The de-vice is normally used for the instantaneous treatment of immediately life-threatening heart rhythms (i.e., ventricular tachycardia and ventricular fibrillation) that can’t wait for treatment until an ambulance arrives.

An external defibrillator has two electrodes that are placed in con-tact with the chest, and a powerful electrical shock is administered. The electrical stimulus depolarizes the entire myocardium simultaneously. With luck, after repolarization, the SA node will be the first area of the heart to reach threshold. Thus, the primary goal of defibrillation is not just to stop the fibrillation, but to give the ventricles a chance to respond to normal SA commands.

Early defibrillation can result in dramatic recovery of an unconscious cardiac-arrest victim. Automatic external defibrillators (AEDs) are easily used, portable machines that can detect lethal ventricular rhythms in people who have collapsed and administer a defibrillating shock. These devices are increasingly being placed on planes, in airports, and in other public areas.

**Clinical Note (continued)**

Myocardial Infarction

In a myocardial (mɪ-ˈoʊ-KAR-dəl) infarction (MI), or heart attack, the coronary circulation becomes blocked and the cardiac muscle cells die from lack of oxygen. The affected tissue then degenerates, creating a nonfunctional area known as an infarct. Heart attacks most often result from severe coronary artery disease. The consequences depend on the site and nature of the circulatory blockage. If it occurs near the base of one of the coronary arteries, the damage will be widespread and the heart will probably stop beating. If the blockage involves one of the smaller arterial branches, the individual may survive the immediate crisis, but there are many potential complications, all unpleasant. As scar tissue forms in the damaged area, the heartbeat may become irregular and less

![Image of an artificial pacemaker](image1)

**Figure 21.12** An artificial pacemaker.

![Image of a coronary angiogram](image2)

(a) A coronary angiogram. (b) An echocardium (left) with interpretive drawing (right). (c) A three-dimensional CT scan of an oblique section and (d) of a posterior-superior view of the heart and great vessels.

![Image of a three-dimensional CT scan](image3)
effective as a pump, and other vessels can become constricted, creating additional cardiovascular problems such as angina.

Myocardial infarctions are most often associated with fixed blockages, such as those seen in CAD. When the crisis develops because of thrombus (stationary clot) formation at an area of plaque, the condition is called coronary thrombosis. A vessel already narrowed by plaque formation may also become blocked by a sudden spasm in the smooth muscles of the vascular wall. The individual then experiences intense pain, similar to that of an angina attack but persisting even at rest.

Roughly 25 percent of MI patients die before obtaining medical assistance, and 65 percent of MI deaths among those under age 50 occur within an hour after the initial infarct. The goals of treatment are to limit the size of the infarct and prevent additional complications by preventing irregular contractions, improving circulation with vasodilators, providing additional oxygen, reducing the cardiac workload, and, if possible, eliminating the cause of the circulatory blockage. Anticoagulants may help prevent the formation of additional thrombi. Chewing an aspirin early in the course of an MI is helpful, and clot-dissolving enzymes may reduce the extent of the damage if they are administered within 6 hours after the MI has occurred.

There are roughly 1.3 million MIs in the United States each year, and half of the victims die within a year of the incident. A number of factors have been identified that increase the risk of a heart attack. They include smoking, high blood pressure, high blood cholesterol levels, diabetes, and obesity. There are also hereditary factors that may predispose an individual to coronary artery disease. The presence of two risk factors more than doubles the risk, so eliminating as many risk factors as possible will improve one’s chances of preventing or surviving a heart attack. For example, changes in eating habits to limit dietary cholesterol, exercise to lower weight, and seeking treatment for high blood pressure are relatively easy steps in the right direction, and the benefits are considerable.

Many techniques can be used to examine the structure and performance of the heart (Figure 21.13). No single diagnostic procedure provides the complete picture, so the test used will vary with the suspected nature of the problem.

### CONCEPT CHECK

1. If the cells of the SA node were not functioning, what effect would this have on heart rate?
2. If norepinephrine is released at the heart, what is the effect on heart rate?
3. How do nodal cells coordinate cardiac muscle contractions?

See blue “Answers” tab at back of book.

### The Electrocardiogram (ECG)

[Figure 21.14]

The electrical events associated with the depolarization and repolarization of the heart are powerful enough to be detected by electrodes placed on the body surface. A recording of these electrical activities constitutes an electrocardiogram (ē-lek-trō-KAR-dē-ō-gram), also called an ECG or EKG. During each cardiac cycle, a wave of depolarization radiates through the atria, reaches the AV node, travels down the interventricular septum to the apex, turns, and spreads through the ventricular myocardium toward the base (Figure 21.14). This electrical activity can be monitored from the body surface. By comparing the information obtained from electrodes placed at different locations, one can monitor the performance of specific nodal, conducting, and contractile components. For example, when a portion of the heart has been damaged, as after an MI, these cardiac muscle cells can no longer conduct action potentials, so an ECG will reveal an abnormal pattern of electrical conduction.

The appearance of the ECG tracing varies, depending on the placement of the monitoring electrodes, or leads. Figure 21.14 shows the important features of a representative electrocardiogram. The P wave accompanies the depolarization of the atria. The QRS complex appears as the ventricles depolarize. This electrical signal is relatively strong because the mass of the ventricular muscle is much larger than that of the atria. The smaller T wave indicates ventricular repolarization. You do not see a deflection corresponding to atrial repolarization because it occurs while the ventricles are depolarizing, and the electrical events are masked by the QRS complex.

Analysis of an ECG usually focuses on the size and duration of the P wave, the QRS complex, and the ST segment (between the QRS and T waves). For example, a smaller-than-normal electrical signal may mean that the mass of the heart muscle has decreased; in contrast, excessively large signals may mean that the heart muscle has become enlarged. Changes in the size and shape of the T wave may indicate a condition that slows ventricular repolarization.

Despite the variety of sophisticated equipment available to assess or visualize cardiac function, in the vast majority of cases, the electrocardiogram provides the first and most important diagnostic information. ECG analysis is especially useful in detecting and diagnosing cardiac arrhythmias (ā-rith-mē-əz) (ā-RITH-mē-əz), abnormal patterns of cardiac activity. Clinical problems appear when the arrhythmias reduce the pumping efficiency of the heart. Serious arrhythmias may indicate damage to the myocardial musculature, injuries to the pacemakers or conduction pathways, or other factors.

### Autonomic Control of Heart Rate [Figure 21.15]

The basic heart rate is established by the pacemaker cells of the SA node, but this intrinsic rate can be modified by the autonomic nervous system (ANS). The sympathetic and parasympathetic divisions of the ANS provide innervation to the heart through the cardiac plexus (anatomical details were presented in Chapter 17). See p. 464 Both ANS divisions innervate the SA and AV nodes as well as the atrial and ventricular cardiac muscle cells and smooth muscle in the walls of the cardiac blood vessels (Figure 21.15).

The effects of NE and ACh on nodal tissues were detailed earlier in this chapter, and may be summarized as follows:

- NE release produces an increase in both heart rate and force of contractions through the stimulation of beta receptors on nodal cells and contractile cells;
- ACh release produces a decrease in both heart rate and force of contractions through the stimulation of muscarinic receptors of nodal cells and contractile cells.
The cardiac centers of the medulla oblongata contain the autonomic centers for cardiac control. Stimulation of the cardioacceleratory center activates the necessary sympathetic neurons; the nearby cardioinhibitory center governs the activities of the parasympathetic neurons. The cardiac centers receive inputs from higher centers, especially from the parasympathetic and sympathetic headquarters in the hypothalamus. (These centers were described in Chapter 15.)

Information concerning the status of the cardiovascular system arrives at the cardiac centers from visceral sensory fibers that monitor baroreceptors sensitive to blood pressure and chemoreceptors sensitive to dissolved gas concentrations. These receptors are innervated by the glossopharyngeal (N IX) and vagus (N X) nerves. In response to this information, the cardiac centers adjust the cardiac performance to maintain adequate circulation to vital organs, such as the brain. These centers respond very quickly to changes in blood pressure and to the amount of dissolved oxygen and carbon dioxide in arterial blood. For example, a drop in blood pressure or an increase in carbon dioxide concentration usually indicates that the heart must work harder to meet the demands of peripheral tissues. The cardiac centers then respond by increasing the heart rate and force of contraction by activating the sympathetic nervous system.
angina pectoris (an-JĪ-na PEK-tor-is): A condition in which exertion or stress can produce severe chest pain, resulting from temporary circulatory insufficiency and ischemia when the heart’s workload increases.

bradycardia (bră-dē-KAR-dē-a): A heart rate that is slower than normal.

cardiac arrhythmias (a-RITH-mē-az): Abnormal patterns of cardiac contraction.

cardiac tamponade: A condition resulting from pericardial irritation and inflammation, in which fluid collects in the pericardial sac and restricts cardiac output.

cardiomyopathies (kar-dē-o-mi-OP-a-thēz): A group of diseases characterized by the progressive, irreversible degeneration of the myocardium.

carditis (kar-DĪ-tis): A general term indicating inflammation of the heart.

coronary artery disease (CAD): Degenerative changes in the coronary circulation.

coronary thrombosis: A blockage due to the formation of a clot (thrombus) at a plaque in a coronary artery.

heart failure: A condition in which the heart weakens and peripheral tissues suffer from oxygen and nutrient deprivation.

heart murmur: A rushing, gurgling sound caused by blood regurgitation back through faulty heart valves.

mitral valve prolapse: A condition in which the mitral valve cusps do not close properly because of abnormally long (or short) chordae tendineae or malfunctioning papillary muscles.

myocardial (mi-ô-KAR-dē-al) infarction (MI): A condition in which the coronary circulation becomes blocked and the cardiac muscle cells die from oxygen starvation; also called a heart attack.

rheumatic heart disease (RHD): A disorder in which the heart valves become thickened and stiffen into a partially closed position, affecting the efficiency of the pumping action of the heart.

tachycardia (tak-e-KAR-dē-a): A heart rate that is faster than normal.

valvular stenosis (ste-NŌ-sis): A condition in which the opening between the heart valves is narrower than normal.
5. The heart has the following surfaces: The sternocostal surface is formed by the anterior surfaces of the right atrium and ventricle; the diaphragmatic surface is formed primarily by the posterior, inferior wall of the left ventricle. (see Figure 21.5)

Internal Anatomy and Organization of the Heart 554
1. The atria are separated by the interatrial septum, and the ventricles are divided by the interventricular septum. The openings between the atria and ventricles contain folds of connective tissue covered by endocardium; these valves maintain a one-way flow of blood. (see Figure 21.6)

The Right Atrium 554
2. The right atrium receives blood from the systemic circuit through two great veins, the superior vena cava and inferior vena cava. The atrial walls contain prominent muscular ridges, the pectinate muscles. The coronary veins return blood to the coronary sinus, which opens into the right atrium. During embryonic development an opening called the foramen ovale penetrates the interatrial septum. This opening closes after birth, leaving a depression termed the fossa ovalis. (see Figure 21.6)

The Right Ventricle 554
3. Blood flows from the right atrium through the right atrioventricular (AV) valve, or tricuspid valve. This valve consists of three cusps of fibrous tissue braced by the tendinous chordae tendineae that are connected to papillary muscles. (see Figure 21.6a,c)

4. Blood leaving the right ventricle enters the pulmonary trunk after passing through the pulmonary valve. The pulmonary trunk divides to form the left and right pulmonary arteries. (see Figure 21.6)

The Left Atrium 556
5. The left atrium receives oxygenated blood from the left and right pulmonary veins; it has thicker walls than those of the right atrium. (see Figure 21.6a,c)

6. Blood leaving the left atrium flows into the left ventricle through the left atrioventricular (AV) valve (mitral or bicuspid valve).

The Left Ventricle 556
7. The left ventricle is the largest and thickest of the four chambers because it must pump blood to the entire body. Blood leaving the left ventricle passes through the aortic valve and into the systemic circuit via the ascending aorta. Blood passes from the ascending aorta through the aortic arch and into the descending aorta. (see Figure 21.6a,c)

Structural Differences between the Left and Right Ventricles 556
8. The right ventricle has thin walls and develops low pressure when pumping into the pulmonary circuit to and from the adjacent lungs. Functionally, low pressure is necessary because the pulmonary capillaries at the gas-exchange surfaces of the lungs are very delicate. The left ventricle has a thick wall because it pumps blood throughout the systemic circuit. Anatomical differences between the left and right ventricles are shown in Figure 21.6.

The Structure and Function of Heart Valves 556
9. The AV valves have four components: (1) a ring of connective tissue attached to the fibrous skeleton of the heart, (2) cusps, (3) chordae tendineae, and (4) papillary muscles.

10. There are two semilunar valves, the aortic valve and the pulmonary valve, guarding the exits of the left and right ventricles. (see Figures 21.6 and 21.7)

11. Valves normally permit blood flow in only one direction, preventing the regurgitation (backflow) of blood.

Coronary Blood Vessels 558
12. The coronary circulation supplies blood to the muscles of the heart to meet the high oxygen and nutrient demands of cardiac muscle cells. The coronary arteries originate at the base of the ascending aorta, and each gives rise to two branches. The right coronary artery gives rise to both a right marginal branch and a posterior interventricular branch. The left coronary artery gives rise to both a circumflex branch and an anterior interventricular branch. Interconnections between arteries called anastomoses ensure a constant blood supply.

13. The great and middle cardiac veins carry blood from the coronary capillaries to the coronary sinus. (see Figure 21.8)

14. Other cardiac veins that empty into the great cardiac vein or the coronary sinus are the posterior vein of the left ventricle, draining the areas served by the circumflex branch of the LCA; the middle cardiac vein, draining the areas supplied by the posterior interventricular branch of the LCA; and the small cardiac vein, draining blood from the posterior surfaces of the right atrium and ventricle.

15. The anterior cardiac veins drain the anterior surface of the right ventricle and empty directly into the right atrium.

The Cardiac Cycle 561
1. The cardiac cycle consists of periods of atrial and ventricular systole (contraction) and atrial and ventricular diastole (relaxation/filling). (see Figure 21.10)

The Coordination of Cardiac Contractions 561
2. Cardiac muscle tissue contracts on its own, without neural or hormonal stimulation. This is called automaticity or autorhythmicity.

3. Nodal cells establish the rate of cardiac contraction, and conducting fibers distribute the contractile stimulus to the general myocardium. (see Figure 21.11)

The Sinoatrial and Atrioventricular Nodes 562
4. Pacemaker cells found in the sinoatrial (SA) node (cardiac pacemaker) normally establish the rate of contraction. (see Figure 21.11)

5. From the SA node, the stimulus travels over the internodal pathways to the atrioventricular (AV) node, then to the AV bundle, which divides into a right and left bundle branch. From here Purkinje cells convey the impulses to the ventricular myocardium. (see Figure 21.11)

The Electrocardiogram (ECG) 566
1. A recording of electrical activities in the heart is an electrocardiogram (ECG or EKG). Important landmarks of an ECG include the P wave (atrial depolarization), QRS complex (ventricular depolarization) and T wave (ventricular repolarization). ECG analysis can detect cardiac arrhythmias, which are abnormal patterns of cardiac activity (see Figure 21.14).

Autonomic Control of Heart Rate 567
2. The basic heart rate is established by the pacemaker cells, but it can be modified by the ANS. Norepinephrine produces an increase in heart rate and force of contraction, while acetylcholine produces a decrease in heart rate and contraction.

3. The cardioacceleratory center in the medulla oblongata activates sympathetic neurons; the cardioinhibitory center governs the activities of the parasympathetic neurons. The cardiac centers receive inputs from higher centers and from receptors monitoring blood pressure and the concentrations of dissolved gases in the blood. (see Figure 21.15)
Level 1 Reviewing Facts and Terms

Match each numbered item with the most closely related lettered item. Use letters for answers in the spaces provided.

<table>
<thead>
<tr>
<th>Column A</th>
<th>Column B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. cardiocytes</td>
<td>a. vein to the left atrium</td>
</tr>
<tr>
<td>2. bradycardia</td>
<td>b. covers the outer surface of heart</td>
</tr>
<tr>
<td>3. diastole</td>
<td>c. supplies blood to heart muscle</td>
</tr>
<tr>
<td>4. coronary circulation</td>
<td>d. lines inner surface of pericardial sac</td>
</tr>
<tr>
<td>5. visceral pericardium</td>
<td>e. slow heart rate</td>
</tr>
<tr>
<td>6. systole</td>
<td>f. cardiac muscle cells</td>
</tr>
<tr>
<td>7. myocardium</td>
<td>g. muscular wall of the heart</td>
</tr>
<tr>
<td>8. right pulmonary vein</td>
<td>h. relaxation phase of the cardiac cycle</td>
</tr>
<tr>
<td>9. superior vena cava</td>
<td>i. vein to the right atrium</td>
</tr>
<tr>
<td>10. parietal pericardium</td>
<td>j. contraction phase of the cardiac cycle</td>
</tr>
</tbody>
</table>

11. The heart lies in the (a) pleural cavity (b) peritoneal cavity (c) abdominopelvic cavity (d) pericardial cavity
12. The atrioventricular valve that is located on the side of the heart that receives blood from the superior vena cava is the (a) mitral valve (b) bicuspid valve (c) tricuspid valve (d) aortic valve
13. The functions of the fibrous pericardium include (a) returning blood to the atria (b) pumping blood into circulation (c) anchoring the heart to surrounding structures (d) providing blood flow to the myocardium
14. All of the following are true of intercalated discs except (a) they provide additional strength from cells bound together by tight junctions (b) they have a smooth junction between the sarcolemmata of apposed muscle cells (c) they have the myofibrils of the interlocking muscle fibers anchored at the membrane (d) the cardiac muscle fibers at the intercalated discs are connected by gap junctions
15. The heart is innervated by (a) only parasympathetic nerves (b) only sympathetic nerves (c) both sympathetic and parasympathetic nerves (d) only splanchnic nerves
16. The pacemaker cells of the heart are located in (a) the SA node (b) the wall of the left ventricle (c) the Purkinje fibers (d) both the left and right ventricles
17. The muscle fibers of the atria are isolated physically from those of the ventricles (a) by the epicardium (b) by the fibrous skeleton of the heart (c) but not electrically, as they all contract at exactly the same time (d) by the coronary blood vessels
18. The two main branches of the right coronary artery are the (a) circumflex branch and the left marginal branch (b) anterior interventricular branch and the left anterior descending branch (c) right marginal branch and the posterior interventricular branch (d) great and middle cardiac veins
19. Analysis of ECG data is useful in the detection and diagnosis of (a) hypertension (b) cardiac arrhythmias (c) cerebrovascular accidents (d) all of the above
20. The mitral or bicuspid valve is located (a) in the opening of the aorta (b) between the left atrium and left ventricle (c) between the right atrium and right ventricle (d) in the opening of the pulmonary trunk

Level 2 Reviewing Concepts

1. If the sinoatrial node is damaged, what will happen to the heartbeat? (a) it will be generated by the bundle branches, at a much lower rate (b) the heart will stop (c) the atrioventricular node will take over setting the pace, at a speed somewhat slower than normal (d) the heartbeat will increase in rate, but not in forcefulness
2. If the papillary muscles fail to contract, (a) blood will not enter the atria (b) the ventricles will not pump blood (c) the AV valves will not close properly (d) the semilunar valves will not open
3. If there were damage to the sympathetic innervation to the heart, what would happen to the heartbeat rate under the influence of the remaining autonomic nervous system stimulation? (a) it would increase (b) it would not change (c) it would decrease (d) it would first increase and then decrease
4. How is cardiac muscle similar to skeletal muscle?
5. Why do semilunar valves lack muscular braces like those found in AV valves?
6. Define a pacemaker cell, and list the group of cells that normally serve as the heart’s pacemaker, as well as those other cells that have the potential to serve as a pacemaker.
7. What is the function of the pericardial fluid?
8. Which chamber of the heart has the thickest walls? Why are its walls so thick?
9. Why are nodal cells unique? What is their function?
10. What is the effect of NE release on cardiac function?

Level 3 Critical Thinking

1. Harvey has a heart murmur in his left ventricle that produces a loud “gurgling” sound at the beginning of systole. What do you suspect to be the cause of this sound?
2. Lee is brought to the emergency room of a hospital suffering from a cardiac arrhythmia. In the emergency room he begins to exhibit tachycardia and as a result loses consciousness. His wife asks you why he lost consciousness. What would you tell her?
3. If the cardiac centers detect an abundance of oxygen in the blood, what complications might be feared to occur up to as many as 10 to 20 years later?