C H A P T E R 9

# Heart Muscle; The Heart as a Pump and Function of the Heart Valves

With this chapter we begin discussion of the heart and circulatory system. The heart, shown in Figure 9–1, is actually two separate pumps: a *right heart* that pumps blood through the lungs, and a *left heart* that pumps blood through the peripheral organs. In turn, each of these hearts is a pulsatile two-chamber pump composed of an *atrium* and a *ventricle.* Each atrium is a weak primer pump for the ventricle, helping to move blood into the ventricle. The ventricles then supply the main pumping force that propels the blood either (1) through the pulmonary circulation by the right ventricle or (2) through the peripheral circulation by the left ventricle.

Special mechanisms in the heart cause a continuing succession of heart contractions called cardiac rhythmicity, transmitting action potentials throughout the heart muscle to cause the heart's rhythmical beat. This rhythmical control system is explained in Chapter 10. In this chapter, we explain how the heart operates as a pump, beginning with the special features of heart muscle itself.

## Physiology of Cardiac Muscle

The heart is composed of three major types of cardiac muscle: *atrial muscle, ventricular muscle,* and specialized *excitatory* and *conductive muscle* fibers. The atrial and ventricular types of muscle contract in much the same way as skeletal muscle, except that the duration of contraction is much longer. Conversely, the specialized excitatory and conductive fibers contract only feebly because they contain few contractile fibrils; instead, they exhibit either automatic rhythmical electrical discharge in the form of action potentials or conduction of the action potentials through the heart, providing an excitatory system that controls the rhythmical beating of the heart.

### Physiologic Anatomy of Cardiac Muscle

Figure 9–2 shows a typical histological picture of cardiac muscle, demonstrating cardiac muscle fibers arranged in a latticework, with the fibers dividing, recombining, and then spreading again. One also notes immediately from this figure that cardiac muscle is *striated* in the same manner as in typical skeletal muscle. Further, cardiac muscle has typical myofibrils that contain *actin* and *myosin filaments* almost identical to those found in skeletal muscle; these filaments lie side by side and slide along one another during contraction in the same manner as occurs in skeletal muscle (see Chapter 6). But in other ways, cardiac muscle is quite different from skeletal muscle, as we shall see.

**Cardiac Muscle as a Syncytium.** The dark areas crossing the cardiac muscle fibers in Figure 9–2 are called *intercalated discs;* they are actually cell membranes that separate individual cardiac muscle cells from one another. That is, cardiac muscle fibers are made up of many individual cells connected in series and in parallel with one another.

**Figure 9–1**

Structure of the heart, and course of blood flow through the heart chambers and heart valves.

**Figure 9–2**

"Syncytial," interconnecting nature of cardiac muscle fibers.

At each intercalated disc the cell membranes fuse with one another in such a way that they form permeable "communicating" junctions (gap junctions) that allow almost totally free diffusion of ions. Therefore, from a functional point of view, ions move with ease in the intracellular fluid along the longitudinal axes of the cardiac muscle fibers, so that action potentials travel easily from one cardiac muscle cell to the next, past the intercalated discs. Thus, cardiac muscle is a *syncytium* of many heart muscle cells in which the cardiac cells are so interconnected that when one of these cells becomes excited, the action potential spreads to all of them, spreading from cell to cell throughout the latticework interconnections.

**Figure 9–3**

Rhythmical action potentials (in millivolts) from a Purkinje fiber and from a ventricular muscle fiber, recorded by means of microelectrodes.

The heart actually is composed of two syncytiums: the *atrial syncytium* that constitutes the walls of the two atria, and the *ventricular syncytium* that constitutes the walls of the two ventricles. The atria are separated from the ventricles by fibrous tissue that surrounds the atrioventricular (A-V) valvular openings between the atria and ventricles. Normally, potentials are not conducted from the atrial syncytium into the ventricular syncytium directly through this fibrous tissue. Instead, they are conducted only by way of a specialized conductive system called the *A-V bundle,* a bundle of conductive fibers several millimeters in diameter that is discussed in detail in Chapter 10.

This division of the muscle of the heart into two functional syncytiums allows the atria to contract a short time ahead of ventricular contraction, which is important for effectiveness of heart pumping.

## Action Potentials in Cardiac Muscle

The *action potential* recorded in a ventricular muscle fiber, shown in Figure 9–3, averages about 105 millivolts, which means that the intracellular potential rises from a very negative value, about −85 millivolts, between beats to a slightly positive value, about +20 millivolts, during each beat. After the initial *spike,* the membrane remains depolarized for about 0.2 second, exhibiting a *plateau* as shown in the figure, followed at the end of the plateau by abrupt repolarization. The presence of this plateau in the action potential causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle.

**What Causes the Long Action Potential and the Plateau?** At this point, we must ask the questions: Why is the action potential of cardiac muscle so long, and why does it have a plateau, whereas that of skeletal muscle does not? The basic biophysical answers to these questions were presented in Chapter 5, but they merit summarizing here as well.

At least two major differences between the membrane properties of cardiac and skeletal muscle account for the prolonged action potential and the plateau in cardiac muscle. First, the *action potential of skeletal muscle* is caused almost entirely by sudden opening of large numbers of so-called *fast sodium channels* that allow tremendous numbers of sodium ions to enter the skeletal muscle fiber from the extracellular fluid. These channels are called "fast" channels because they remain open for only a few thousandths of a second and then abruptly close. At the end of this closure, repolarization occurs, and the action potential is over within another thousandth of a second or so.

In *cardiac muscle, the action potential* is caused by opening of two types of channels: (1) the same *fast sodium channels* as those in skeletal muscle and (2) another entirely different population of *slow calcium channels,* which are also called *calcium-sodium channels.* This second population of channels differs from the fast sodium channels in that they are slower to open and, even more important, remain open for several tenths of a second. During this time, a large quantity of both calcium and sodium ions flows through these channels to the interior of the cardiac muscle fiber, and this maintains a prolonged period of depolarization, *causing the plateau* in the action potential. Further, the calcium ions that enter during this plateau phase activate the muscle contractile process, while the calcium ions that cause skeletal muscle contraction are derived from the intracellular sarcoplasmic reticulum.

The second major functional difference between cardiac muscle and skeletal muscle that helps account for both the prolonged action potential and its plateau is this: Immediately after the onset of the action potential, the permeability of the cardiac muscle membrane for potassium ions *decreases* about fivefold, an effect that does not occur in skeletal muscle. This decreased potassium permeability may result from the excess calcium influx through the calcium channels just noted. Regardless of the cause, the decreased potassium permeability greatly decreases the outflux of positively charged potassium ions during the action potential plateau and thereby prevents early return of the action potential voltage to its resting level. When the slow calcium-sodium channels do close at the end of 0.2 to 0.3 second and the influx of calcium and sodium ions ceases, the membrane permeability for potassium ions also increases rapidly; this rapid loss of potassium from the fiber immediately returns the membrane potential to its resting level, thus ending the action potential.

**Velocity of Signal Conduction in Cardiac Muscle.** The velocity of conduction of the excitatory action potential signal along both *atrial and ventricular muscle fibers* is about 0.3 to 0.5 m/sec, or about $^1/_{250}$ the velocity in very large nerve fibers and about $^1/_{10}$ the velocity in skeletal muscle fibers. The velocity of conduction in the specialized heart conductive system—in the *Purkinje fibers*—is as great as 4 m/sec in most parts of the system, which allows reasonably rapid conduction of the excitatory signal to the different parts of the heart, as explained in Chapter 10.

**Refractory Period of Cardiac Muscle.** Cardiac muscle, like all excitable tissue, is refractory to restimulation during the action potential. Therefore, the refractory period of the heart is the interval of time, as shown to the left in Figure 9–4, during which a normal cardiac impulse cannot re-excite an already excited area of cardiac muscle. The normal refractory period of the ventricle is 0.25 to 0.30 second, which is about the duration of the prolonged plateau action potential. There is an additional *relative refractory period* of

**Figure 9–4**

Force of ventricular heart muscle contraction, showing also duration of the refractory period and relative refractory period, plus the effect of premature contraction. Note that premature contractions do not cause wave summation, as occurs in skeletal muscle.

about 0.05 second during which the muscle is more difficult than normal to excite but nevertheless can be excited by a very strong excitatory signal, as demonstrated by the early "premature" contraction in the second example of Figure 9–4. The refractory period of atrial muscle is much shorter than that for the ventricles (about 0.15 second for the atria compared with 0.25 to 0.30 second for the ventricles).

#### Excitation-Contraction Coupling—Function of Calcium Ions and the *Transverse Tubules*

The term "excitation-contraction coupling" refers to the mechanism by which the action potential causes the myofibrils of muscle to contract. This was discussed for skeletal muscle in Chapter 7. Once again, there are differences in this mechanism in cardiac muscle that have important effects on the characteristics of cardiac muscle contraction.

As is true for skeletal muscle, when an action potential passes over the cardiac muscle membrane, the action potential spreads to the interior of the cardiac muscle fiber along the membranes of the transverse (T) tubules. The T tubule action potentials in turn act on the membranes of the longitudinal sarcoplasmic tubules to cause release of calcium ions into the muscle sarcoplasm from the sarcoplasmic reticulum. In another few thousandths of a second, these calcium ions diffuse into the myofibrils and catalyze the chemical reactions that promote sliding of the actin and myosin filaments along one another; this produces the muscle contraction.

Thus far, this mechanism of excitation-contraction coupling is the same as that for skeletal muscle, but there is a second effect that is quite different. In addition to the calcium ions that are released into the sarcoplasm from the cisternae of the sarcoplasmic reticulum, a large quantity of extra calcium ions also diffuses into the sarcoplasm from the T tubules themselves at the time of the action potential. Indeed, without this extra calcium from the T tubules, the strength of cardiac muscle contraction would be reduced considerably because the sarcoplasmic reticulum of cardiac muscle is less well developed than that of skeletal muscle and does not store enough calcium to provide full contraction. Conversely, the T tubules of cardiac muscle have a diameter 5 times as great as that of the skeletal muscle tubules, which means a volume 25 times as great. Also, inside the T tubules is a large quantity of mucopolysaccharides that are electronegatively charged and bind an abundant store of calcium ions, keeping these always available for diffusion to the interior of the cardiac muscle fiber when a T tubule action potential appears.

The strength of contraction of cardiac muscle depends to a great extent on the concentration of calcium ions in the extracellular fluids. The reason for this is that the openings of the T tubules pass directly through the cardiac muscle cell membrane into the extracellular spaces surrounding the cells, allowing the same extracellular fluid that is in the cardiac muscle interstitium to percolate through the T tubules as well. Consequently, the quantity of calcium ions in the T tubule system—that is, the availability of calcium ions to cause cardiac muscle contraction—depends to a great extent on the extracellular fluid calcium ion concentration.

(By way of contrast, the strength of skeletal muscle contraction is hardly affected by moderate changes in extracellular fluid calcium concentration because skeletal muscle contraction is caused almost entirely by calcium ions released from the sarcoplasmic reticulum *inside* the skeletal muscle fiber itself.)

At the end of the plateau of the cardiac action potential, the influx of calcium ions to the interior of the muscle fiber is suddenly cut off, and the calcium ions in the sarcoplasm are rapidly pumped back out of the muscle fibers into both the sarcoplasmic reticulum and the T tubule–extracellular fluid space. As a result, the contraction ceases until a new action potential comes along.

**Duration of Contraction.** Cardiac muscle begins to contract a few milliseconds after the action potential begins and continues to contract until a few milliseconds after the action potential ends. Therefore, the duration of contraction of cardiac muscle is mainly a function of the duration of the action potential, *including the plateau*—about 0.2 second in atrial muscle and 0.3 second in ventricular muscle.

## The Cardiac Cycle

The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the *cardiac cycle.* Each cycle is initiated by spontaneous generation of an action potential in the *sinus node*, as explained in Chapter 10. This node is located in the superior lateral wall of the right atrium near the opening of the superior vena cava, and the action potential travels from here rapidly through both atria and then through the A-V bundle into the ventricles. Because of this special arrangement of the conducting system from the atria into the ventricles, there is a delay of more than 0.1 second during passage of the cardiac impulse from the atria into the ventricles. This allows the atria to contract ahead of ventricular contraction, thereby pumping blood into the ventricles before the strong ventricular contraction begins. Thus, the atria act as *primer pumps* for the ventricles, and the ventricles in turn provide the major source of power for moving blood through the body's vascular system.

### Diastole and Systole

The cardiac cycle consists of a period of relaxation called *diastole,* during which the heart fills with blood, followed by a period of contraction called *systole.*

Figure 9–5 shows the different events during the cardiac cycle for the left side of the heart. The top three curves show the pressure changes in the aorta, left ventricle, and left atrium, respectively. The fourth curve depicts the changes in left ventricular volume, the fifth

**Figure 9–5**

Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram.

the electrocardiogram, and the sixth a phonocardiogram, which is a recording of the sounds produced by the heart—mainly by the heart valves—as it pumps. It is especially important that the reader study in detail this figure and understand the causes of all the events shown.

## Relationship of the Electrocardiogram to the Cardiac Cycle

The electrocardiogram in Figure 9–5 shows the *P, Q, R, S,* and *T waves,* which are discussed in Chapters 11, 12, and 13. They are electrical voltages generated by the heart and recorded by the electrocardiograph from the surface of the body.

The *P wave* is caused by *spread of depolarization* through the atria, and this is followed by atrial contraction, which causes a slight rise in the atrial pressure curve immediately after the electrocardiographic P wave.

About 0.16 second after the onset of the P wave, the *QRS waves* appear as a result of electrical depolarization of the ventricles, which initiates contraction of the ventricles and causes the ventricular pressure to begin rising, as also shown in the figure. Therefore, the QRS complex begins slightly before the onset of ventricular systole.

Finally, one observes the *ventricular T wave* in the electrocardiogram. This represents the stage of repolarization of the ventricles when the ventricular muscle fibers begin to relax. Therefore, the T wave occurs slightly before the end of ventricular contraction.

## Function of the Atria as Primer Pumps

Blood normally flows continually from the great veins into the atria; about 80 per cent of the blood flows directly through the atria into the ventricles even before the atria contract. Then, atrial contraction usually causes an additional 20 per cent filling of the ventricles. Therefore, the atria simply function as

primer pumps that increase the ventricular pumping effectiveness as much as 20 per cent. However, the heart can continue to operate under most conditions even without this extra 20 per cent effectiveness because it normally has the capability of pumping 300 to 400 per cent more blood than is required by the resting body. Therefore, when the atria fail to function, the difference is unlikely to be noticed unless a person exercises; then acute signs of heart failure occasionally develop, especially shortness of breath.

**Pressure Changes in the Atria—The a, c, and v Waves.** In the atrial pressure curve of Figure 9–5, three minor pressure elevations, called the *a, c,* and *v atrial pressure waves,* are noted.

The *a wave* is caused by atrial contraction. Ordinarily, the *right* atrial pressure increases 4 to 6 mm Hg during atrial contraction, and the *left* atrial pressure increases about 7 to 8 mm Hg.

The *c wave* occurs when the ventricles begin to contract; it is caused partly by slight backflow of blood into the atria at the onset of ventricular contraction but mainly by bulging of the A-V valves backward toward the atria because of increasing pressure in the ventricles.

The *v wave* occurs toward the end of ventricular contraction; it results from slow flow of blood into the atria from the veins while the A-V valves are closed during ventricular contraction. Then, when ventricular contraction is over, the A-V valves open, allowing this stored atrial blood to flow rapidly into the ventricles and causing the v wave to disappear.

## Function of the Ventricles as Pumps

**Filling of the Ventricles.** During ventricular systole, large amounts of blood accumulate in the right and left atria because of the closed A-V valves. Therefore, as soon as systole is over and the ventricular pressures fall again to their low diastolic values, the moderately increased pressures that have developed in the atria during ventricular systole immediately push the A-V valves open and allow blood to flow rapidly into the ventricles, as shown by the rise of the left *ventricular volume curve* in Figure 9–5. This is called the *period of rapid filling of the ventricles.*

The period of rapid filling lasts for about the first third of diastole. During the middle third of diastole, only a small amount of blood normally flows into the ventricles; this is blood that continues to empty into the atria from the veins and passes through the atria directly into the ventricles.

During the last third of diastole, the atria contract and give an additional thrust to the inflow of blood into the ventricles; this accounts for about 20 per cent of the filling of the ventricles during each heart cycle.

### Emptying of the Ventricles During Systole

**Period of Isovolumic (Isometric) Contraction.** Immediately after ventricular contraction begins, the ventricular pressure rises abruptly, as shown in Figure 9–5, causing the A-V valves to close. Then an additional 0.02 to 0.03 second is required for the ventricle to build up sufficient pressure to push the semilunar (aortic and pulmonary) valves open against the pressures in the aorta and pulmonary artery. Therefore, during this period, contraction is occurring in the ventricles, but there is no emptying. This is called the period of *isovolumic* or *isometric contraction,* meaning that tension is increasing in the muscle but little or no shortening of the muscle fibers is occurring.

**Period of Ejection.** When the left ventricular pressure rises slightly above 80 mm Hg (and the right ventricular pressure slightly above 8 mm Hg), the ventricular pressures push the semilunar valves open. Immediately, blood begins to pour out of the ventricles, with about 70 per cent of the blood emptying occurring during the first third of the period of ejection and the remaining 30 per cent emptying during the next two thirds. Therefore, the first third is called the *period of rapid ejection,* and the last two thirds, the *period of slow ejection.*

**Period of Isovolumic (Isometric) Relaxation.** At the end of systole, ventricular relaxation begins suddenly, allowing both the right and left *intraventricular pressures* to decrease rapidly. The elevated pressures in the distended large arteries that have just been filled with blood from the contracted ventricles immediately push blood back toward the ventricles, which snaps the aortic and pulmonary valves closed. For another 0.03 to 0.06 second, the ventricular muscle continues to relax, even though the ventricular volume does not change, giving rise to the period of *isovolumic* or *isometric relaxation.* During this period, the intraventricular pressures decrease rapidly back to their low diastolic levels. Then the A-V valves open to begin a new cycle of ventricular pumping.

**End-Diastolic Volume, End-Systolic Volume, and Stroke Volume Output.** During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 milliliters. This volume is called the *end-diastolic volume.* Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the *stroke volume output.* The remaining volume in each ventricle, about 40 to 50 milliliters, is called the *end-systolic volume.* The fraction of the end-diastolic volume that is ejected is called the *ejection fraction*—usually equal to about 60 per cent.

When the heart contracts strongly, the end-systolic volume can be decreased to as little as 10 to 20 milliliters. Conversely, when large amounts of blood flow into the ventricles during diastole, the ventricular end-diastolic volumes can become as great as 150 to 180 milliliters in the healthy heart. By both increasing the end-diastolic volume and decreasing the end-systolic volume, the stroke volume output can be increased to more than double normal.

## Function of the Valves

**Atrioventricular Valves.** The *A-V valves* (the *tricuspid* and *mitral* valves) prevent backflow of blood from the ventricles to the atria during systole, and the *semilunar valves* (the *aortic* and *pulmonary artery* valves) prevent backflow from the aorta and pulmonary arteries into the ventricles during diastole. These valves, shown in Figure 9–6 for the left ventricle, close and open *passively.* That is, they close when a backward pressure gradient pushes blood backward, and they open when a forward pressure gradient forces blood in the forward direction. For anatomical reasons, the thin, filmy A-V valves require almost no backflow to cause closure, whereas the much heavier semilunar valves require rather rapid backflow for a few milliseconds.

**Function of the Papillary Muscles.** Figure 9–6 also shows papillary muscles that attach to the vanes of the A-V valves by the *chordae tendineae.* The papillary muscles contract when the ventricular walls contract, but contrary to what might be expected, they *do not* help the valves to close. Instead, they pull the vanes of the valves inward toward the ventricles to prevent their bulging too far backward toward the atria during ventricular contraction. If a chorda tendinea becomes ruptured or if one of the papillary muscles becomes paralyzed, the valve bulges far backward during ventricular contraction, sometimes so far that it leaks severely and results in severe or even lethal cardiac incapacity.

**Figure 9–6**

Mitral and aortic valves (the left ventricular valves).

**Aortic and Pulmonary Artery Valves.** The aortic and pulmonary artery semilunar valves function quite differently from the A-V valves. First, the high pressures in the arteries at the end of systole cause the semilunar valves to snap to the closed position, in contrast to the much softer closure of the A-V valves. Second, because of smaller openings, the velocity of blood ejection through the aortic and pulmonary valves is far greater than that through the much larger A-V valves. Also, because of the rapid closure and rapid ejection, the edges of the aortic and pulmonary valves are subjected to much greater mechanical abrasion than are the A-V valves. Finally, the A-V valves are supported by the chordae tendineae, which is not true for the semilunar valves. It is obvious from the anatomy of the aortic and pulmonary valves (as shown for the aortic valve at the bottom of Figure 9–6) that they must be constructed with an especially strong yet very pliable fibrous tissue base to withstand the extra physical stresses.

## Aortic Pressure Curve

When the left ventricle contracts, the ventricular pressure increases rapidly until the aortic valve opens. Then, after the valve opens, the pressure in the ventricle rises much less rapidly, as shown in Figure 9–5, because blood immediately flows out of the ventricle into the aorta and then into the systemic distribution arteries.

The entry of blood into the arteries causes the walls of these arteries to stretch and the pressure to increase to about 120 mm Hg.

Next, at the end of systole, after the left ventricle stops ejecting blood and the aortic valve closes, the elastic walls of the arteries maintain a high pressure in the arteries, even during diastole.

A so-called *incisura* occurs in the aortic pressure curve when the aortic valve closes. This is caused by a short period of backward flow of blood immediately before closure of the valve, followed by sudden cessation of the backflow.

After the aortic valve has closed, the pressure in the aorta decreases slowly throughout diastole because the blood stored in the distended elastic arteries flows continually through the peripheral vessels back to the veins. Before the ventricle contracts again, the aortic pressure usually has fallen to about 80 mm Hg (diastolic pressure), which is two thirds the maximal pressure of 120 mm Hg (systolic pressure) that occurs in the aorta during ventricular contraction.

The pressure curves in the *right ventricle* and *pulmonary artery* are similar to those in the aorta, except that the pressures are only about one sixth as great, as discussed in Chapter 14.

# Relationship of the Heart Sounds to Heart Pumping

When listening to the heart with a stethoscope, one does not hear the opening of the valves because this is a

relatively slow process that normally makes no noise. However, when the valves close, the vanes of the valves and the surrounding fluids vibrate under the influence of sudden pressure changes, giving off sound that travels in all directions through the chest.

When the ventricles contract, one first hears a sound caused by closure of the A-V valves. The vibration is low in pitch and relatively long-lasting and is known as the *first heart sound.* When the aortic and pulmonary valves close at the end of systole, one hears a rapid snap because these valves close rapidly, and the surroundings vibrate for a short period. This sound is called the *second heart sound.* The precise causes of the heart sounds are discussed more fully in Chapter 23, in relation to listening to the sounds with the stethoscope.

## Work Output of the Heart

The *stroke work output* of the heart is the amount of energy that the heart converts to work during each heartbeat while pumping blood into the arteries. *Minute work output* is the total amount of energy converted to work in 1 minute; this is equal to the stroke work output times the heart rate per minute.

Work output of the heart is in two forms. First, by far the major proportion is used to move the blood from the low-pressure veins to the high-pressure arteries. This is called *volume-pressure work* or *external work.* Second, a minor proportion of the energy is used to accelerate the blood to its velocity of ejection through the aortic and pulmonary valves. This is the *kinetic energy of blood flow* component of the work output.

Right ventricular external work output is normally about one sixth the work output of the left ventricle because of the sixfold difference in systolic pressures that the two ventricles pump. The additional work output of each ventricle required to create kinetic energy of blood flow is proportional to the mass of blood ejected times the square of velocity of ejection.

Ordinarily, the work output of the left ventricle required to create kinetic energy of blood flow is only about 1 per cent of the total work output of the ventricle and therefore is ignored in the calculation of the total stroke work output. But in certain abnormal conditions, such as aortic stenosis, in which blood flows with great velocity through the stenosed valve, more than 50 per cent of the total work output may be required to create kinetic energy of blood flow.

### Graphical Analysis of Ventricular Pumping

Figure 9–7 shows a diagram that is especially useful in explaining the pumping mechanics of the *left* ventricle. The most important components of the diagram are the two curves labeled "diastolic pressure" and "systolic pressure." These curves are volume-pressure curves.

The diastolic pressure curve is determined by filling the heart with progressively greater volumes of blood and then measuring the diastolic pressure immediately before ventricular contraction occurs, which is the *end-diastolic pressure* of the ventricle.

The systolic pressure curve is determined by recording the systolic pressure achieved during ventricular contraction at each volume of filling.

**Figure 9–7**

Relationship between left ventricular volume and intraventricular pressure during diastole and systole. Also shown by the heavy red lines is the "volume-pressure diagram," demonstrating changes in intraventricular volume and pressure during the normal cardiac cycle. EW, net external work.

Until the volume of the noncontracting ventricle rises above about 150 milliliters, the "diastolic" pressure does not increase greatly. Therefore, up to this volume, blood can flow easily into the ventricle from the atrium. Above 150 milliliters, the ventricular diastolic pressure increases rapidly, partly because of fibrous tissue in the heart that will stretch no more and partly because the pericardium that surrounds the heart becomes filled nearly to its limit.

During ventricular contraction, the "systolic" pressure increases even at low ventricular volumes and reaches a maximum at a ventricular volume of 150 to 170 milliliters. Then, as the volume increases still further, the systolic pressure actually decreases under some conditions, as demonstrated by the falling systolic pressure curve in Figure 9–7, because at these great volumes, the actin and myosin filaments of the cardiac muscle fibers are pulled apart far enough that the strength of each cardiac fiber contraction becomes less than optimal.

Note especially in the figure that the maximum systolic pressure for the normal *left* ventricle is between 250 and 300 mm Hg, but this varies widely with each person's heart strength and degree of heart stimulation by cardiac nerves. For the normal *right* ventricle, the maximum systolic pressure is between 60 and 80 mm Hg.

**"Volume-Pressure Diagram" During the Cardiac Cycle; Cardiac Work Output.** The red lines in Figure 9–7 form a loop called the *volume-pressure diagram* of the cardiac cycle for normal function of the *left* ventricle. It is divided into four phases.

Phase I: *Period of filling.* This phase in the volume-pressure diagram begins at a ventricular volume of about 45 milliliters and a diastolic pressure near 0 mm Hg. Forty-five milliliters is the amount of

blood that remains in the ventricle after the previous heartbeat and is called the *end-systolic volume.* As venous blood flows into the ventricle from the left atrium, the ventricular volume normally increases to about 115 milliliters, called the *end-diastolic volume,* an increase of 70 milliliters. Therefore, the volume-pressure diagram during phase I extends along the line labeled "I," with the volume increasing to 115 milliliters and the diastolic pressure rising to about 5 mm Hg.

Phase II: *Period of isovolumic contraction.* During isovolumic contraction, the volume of the ventricle does not change because all valves are closed. However, the pressure inside the ventricle increases to equal the pressure in the aorta, at a pressure value of about 80 mm Hg, as depicted by the arrow end of the line labeled "II."

Phase III: *Period of ejection.* During ejection, the systolic pressure rises even higher because of still more contraction of the ventricle. At the same time, the volume of the ventricle decreases because the aortic valve has now opened and blood flows out of the ventricle into the aorta. Therefore, the curve labeled "III" traces the changes in volume and systolic pressure during this period of ejection.

Phase IV: *Period of isovolumic relaxation.* At the end of the period of ejection, the aortic valve closes, and the ventricular pressure falls back to the diastolic pressure level. The line labeled "IV" traces this decrease in intraventricular pressure without any change in volume. Thus, the ventricle returns to its starting point, with about 45 milliliters of blood left in the ventricle and at an atrial pressure near 0 mm Hg.

Readers well trained in the basic principles of physics should recognize that the area subtended by this functional volume-pressure diagram (the tan shaded area, labeled EW) represents the *net external work output* of the ventricle during its contraction cycle. In experimental studies of cardiac contraction, this diagram is used for calculating cardiac work output.

When the heart pumps large quantities of blood, the area of the work diagram becomes much larger. That is, it extends far to the right because the ventricle fills with more blood during diastole, it rises much higher because the ventricle contracts with greater pressure, and it usually extends farther to the left because the ventricle contracts to a smaller volume—especially if the ventricle is stimulated to increased activity by the sympathetic nervous system.

**Concepts of Preload and Afterload.** In assessing the contractile properties of muscle, it is important to specify the degree of tension on the muscle when it begins to contract, which is called the *preload,* and to specify the load against which the muscle exerts its contractile force, which is called the *afterload.*

For cardiac contraction, the *preload* is usually considered to be the end-diastolic pressure when the ventricle has become filled.

The *afterload* of the ventricle is the pressure in the artery leading from the ventricle. In Figure 9–7, this corresponds to the systolic pressure described by the phase III curve of the volume-pressure diagram. (Sometimes the afterload is loosely considered to be the resistance in the circulation rather than the pressure.)

The importance of the concepts of preload and afterload is that in many abnormal functional states of the heart or circulation, the pressure during filling of the ventricle (the preload), the arterial pressure against which the ventricle must contract (the afterload), or both are severely altered from normal.

## Chemical Energy Required for Cardiac Contraction: Oxygen Utilization by the Heart

Heart muscle, like skeletal muscle, uses chemical energy to provide the work of contraction. This energy is derived mainly from oxidative metabolism of fatty acids and, to a lesser extent, of other nutrients, especially lactate and glucose. Therefore, the rate of oxygen consumption by the heart is an excellent measure of the chemical energy liberated while the heart performs its work. The different chemical reactions that liberate this energy are discussed in Chapters 67 and 68.

**Efficiency of Cardiac Contraction.** During heart muscle contraction, most of the expended chemical energy is converted into *heat* and a much smaller portion into *work output.* The ratio of work output to total chemical energy expenditure is called the *efficiency of cardiac contraction,* or simply *efficiency of the heart.* Maximum efficiency of the normal heart is between 20 and 25 per cent. In heart failure, this can decrease to as low as 5 to 10 per cent.

## Regulation of Heart Pumping

When a person is at rest, the heart pumps only 4 to 6 liters of blood each minute. During severe exercise, the heart may be required to pump four to seven times this amount. The basic means by which the volume pumped by the heart is regulated are (1) intrinsic cardiac regulation of pumping in response to changes in volume of blood flowing into the heart and (2) control of heart rate and strength of heart pumping by the autonomic nervous system.

### Intrinsic Regulation of Heart Pumping—The Frank-Starling Mechanism

In Chapter 20, we will learn that under most conditions, the amount of blood pumped by the heart each minute is determined almost entirely by the rate of blood flow into the heart from the veins, which is called *venous return.* That is, each peripheral tissue of the body controls its own local blood flow, and all the local tissue flows combine and return by way of the veins to the right atrium. The heart, in turn, automatically pumps this incoming blood into the arteries, so that it can flow around the circuit again.

This intrinsic ability of the heart to adapt to increasing volumes of inflowing blood is called the *Frank-Starling mechanism of the heart,* in honor of Frank and

CHAPTER 11

# The Normal Electrocardiogram

When the cardiac impulse passes through the heart, electrical current also spreads from the heart into the adjacent tissues surrounding the heart. A small portion of the current spreads all the way to the surface of the body. If electrodes are placed on the skin on opposite sides of the heart, electrical potentials generated by the current can be recorded; the recording is known as an *electrocardiogram.* A normal electrocardiogram for two beats of the heart is shown in Figure 11–1.

## Characteristics of the Normal Electrocardiogram

The normal electrocardiogram (see Figure 11–1) is composed of a P wave, a QRS complex, and a T wave. The QRS complex is often, but not always, three separate waves: the Q wave, the R wave, and the S wave.

The P wave is caused by electrical potentials generated when the atria depolarize before atrial contraction begins. The QRS complex is caused by potentials generated when the ventricles depolarize before contraction, that is, as the depolarization wave spreads through the ventricles. Therefore, both the P wave and the components of the QRS complex are *depolarization waves.*

The T wave is caused by potentials generated as the ventricles recover from the state of depolarization. This process normally occurs in ventricular muscle 0.25 to 0.35 second after depolarization, and the T wave is known as a *repolarization wave.*

Thus, the electrocardiogram is composed of both depolarization and repolarization waves. The principles of depolarization and repolarization are discussed in Chapter 5. The distinction between depolarization waves and repolarization waves is so important in electrocardiography that further clarification is needed.

### Depolarization Waves Versus Repolarization Waves

Figure 11–2 shows a single cardiac muscle fiber in four stages of depolarization and repolarization, the color red designating depolarization. During depolarization, the normal negative potential inside the fiber reverses and becomes slightly positive inside and negative outside.

In Figure 11–2*A,* depolarization, demonstrated by red positive charges inside and red negative charges outside, is traveling from left to right. The first half of the fiber has already depolarized, while the remaining half is still polarized. Therefore, the left electrode on the outside of the fiber is in an area of negativity, and the right electrode is in an area of positivity; this causes the meter to record positively. To the right of the muscle fiber is shown a record of changes in potential between the two electrodes, as recorded by a high-speed recording meter. Note that when depolarization has reached the halfway mark in Figure 11–2*A,* the record has risen to a maximum positive value.

In Figure 11–2*B*, depolarization has extended over the entire muscle fiber, and the recording to the right has returned to the zero baseline because both electrodes are now in areas of equal negativity. The completed wave is a *depolarization wave* because it results from spread of *depolarization* along the muscle fiber membrane.

**Figure 11–1**

Normal electrocardiogram.

Figure 11–2*C* shows halfway repolarization of the same muscle fiber, with positivity returning to the outside of the fiber. At this point, the left electrode is in an area of positivity, and the right electrode is in an area of negativity. This is opposite to the polarity in Figure 11–2*A*. Consequently, the recording, as shown to the right, becomes negative.

In Figure 11–2*D*, the muscle fiber has completely repolarized, and both electrodes are now in areas of positivity, so that no potential difference is recorded between them. Thus, in the recording to the right, the potential returns once more to zero. This completed negative wave is a *repolarization wave* because it results from spread of repolarization along the muscle fiber membrane.

**Relation of the Monophasic Action Potential of Ventricular Muscle to the QRS and T Waves in the Standard Electrocardiogram.** The monophasic action potential of ventricular muscle, discussed in Chapter 10, normally lasts between 0.25 and 0.35 second. The top part of Figure 11–3 shows a monophasic action potential recorded from a microelectrode inserted to the inside of a single ventricular muscle fiber. The upsweep of this action potential is caused by *depolarization,* and the return of the potential to the baseline is caused by *repolarization.*

Note in the lower half of the figure a simultaneous recording of the electrocardiogram from this same ventricle, which shows the QRS waves appearing at

**Figure 11–2**

Recording the *depolarization wave* (*A* and *B*) and the *repolarization wave* (*C* and *D*) from a cardiac muscle fiber.

**Figure 11–3**

*Above,* Monophasic action potential from a ventricular muscle fiber during normal cardiac function, showing rapid depolarization and then repolarization occurring slowly during the plateau stage but rapidly toward the end. *Below,* Electrocardiogram recorded simultaneously.

the beginning of the monophasic action potential and the T wave appearing at the end. Note especially that *no potential is recorded in the electrocardiogram when the ventricular muscle is either completely polarized or completely depolarized.* Only when the muscle is partly polarized and partly depolarized does current flow from one part of the ventricles to another part, and therefore current also flows to the surface of the body to produce the electrocardiogram.

## Relationship of Atrial and Ventricular Contraction to the Waves of the Electrocardiogram

Before contraction of muscle can occur, depolarization must spread through the muscle to initiate the chemical processes of contraction. Refer again to Figure 11–1; the P wave occurs at the *beginning of contraction of the atria,* and the QRS complex of waves occurs at the *beginning of contraction of the ventricles.* The ventricles remain contracted until after repolarization has occurred, that is, until after the end of the T wave.

The atria repolarize about 0.15 to 0.20 second after termination of the P wave. This is also approximately when the QRS complex is being recorded in the electrocardiogram. Therefore, the atrial repolarization wave, known as the *atrial T wave,* is usually obscured by the much larger QRS complex. For this reason, an atrial T wave seldom is observed in the electrocardiogram.

The ventricular repolarization wave is the T wave of the normal electrocardiogram. Ordinarily, ventricular muscle begins to repolarize in some fibers about 0.20 second after the beginning of the depolarization wave (the QRS complex), but in many other fibers, it takes as long as 0.35 second. Thus, the process of ventricular repolarization extends over a long period, about 0.15 second. For this reason, the T wave in the normal electrocardiogram is a prolonged wave, but the voltage of the T wave is considerably less than the voltage of the QRS complex, partly because of its prolonged length.

## Voltage and Time Calibration of the Electrocardiogram

All recordings of electrocardiograms are made with appropriate calibration lines on the recording paper. Either these calibration lines are already ruled on the paper, as is the case when a pen recorder is used, or they are recorded on the paper at the same time that the electrocardiogram is recorded, which is the case with the photographic types of electrocardiographs.

As shown in Figure 11–1, the horizontal calibration lines are arranged so that 10 of the small line divisions upward or downward in the standard electrocardiogram represent 1 millivolt, with positivity in the upward direction and negativity in the downward direction.

The vertical lines on the electrocardiogram are time calibration lines. Each inch in the horizontal direction is 1 second, and each inch is usually broken into five segments by dark vertical lines; the intervals between these dark lines represent 0.20 second. The 0.20 second intervals are then broken into five smaller intervals by thin lines, each of which represents 0.04 second.

**Normal Voltages in the Electrocardiogram.** The recorded voltages of the waves in the normal electrocardiogram depend on the manner in which the electrodes are applied to the surface of the body and how close the electrodes are to the heart. When one electrode is placed directly over the ventricles and a second electrode is placed elsewhere on the body remote from the heart, the voltage of the QRS complex may be as great as 3 to 4 millivolts. Even this voltage is small in comparison with the monophasic action potential of 110 millivolts recorded directly at the heart muscle membrane. When electrocardiograms are recorded from electrodes on the two arms or on one arm and one leg, the voltage of the QRS complex usually is 1.0 to 1.5 millivolt from the top of the R wave to the bottom of the S wave; the voltage of the P wave is between 0.1 and 0.3 millivolt; and that of the T wave is between 0.2 and 0.3 millivolt.

**P-Q or P-R Interval.** The time between the beginning of the P wave and the beginning of the QRS complex is the interval between the beginning of electrical excitation of the atria and the beginning of excitation of the ventricles. This period is called the P-Q interval. The normal P-Q interval is about 0.16 second. (Often this interval is called the P-R interval because the Q wave is likely to be absent.)

**Q-T Interval.** Contraction of the ventricle lasts almost from the beginning of the Q wave (or R wave, if the Q wave is absent) to the end of the T wave. This

interval is called the Q-T interval and ordinarily is about 0.35 second.

**Rate of Heartbeat as Determined from the Electrocardiogram.** The rate of heartbeat can be determined easily from an electrocardiogram because the heart rate is the reciprocal of the time interval between two successive heartbeats. If the interval between two beats as determined from the time calibration lines is 1 second, the heart rate is 60 beats per minute. The normal interval between two successive QRS complexes in the adult person is about 0.83 second. This is a heart rate of 60/0.83 times per minute, or 72 beats per minute.

## Methods for Recording Electrocardiograms

Sometimes the electrical currents generated by the cardiac muscle during each beat of the heart change electrical potentials and polarities on the respective sides of the heart in less than 0.01 second. Therefore, it is essential that any apparatus for recording electrocardiograms be capable of responding rapidly to these changes in potentials.

### Pen Recorder

Many modern clinical electrocardiographs use computer-based systems and electronic display, while others use a direct pen recorder that writes the electrocardiogram with a pen directly on a moving sheet of paper. Sometimes the pen is a thin tube connected at one end to an inkwell, and its recording end is connected to a powerful electromagnet system that is capable of moving the pen back and forth at high speed. As the paper moves forward, the pen records the electrocardiogram. The movement of the pen is controlled by appropriate electronic amplifiers connected to electrocardiographic electrodes on the patient.

Other pen recording systems use special paper that does not require ink in the recording stylus. One such paper turns black when it is exposed to heat; the stylus itself is made very hot by electrical current flowing through its tip. Another type turns black when electrical current flows from the tip of the stylus through the paper to an electrode at its back. This leaves a black line on the paper where the stylus touches.

## Flow of Current Around the Heart During the Cardiac Cycle

### Recording Electrical Potentials from a Partially Depolarized Mass of Syncytial Cardiac Muscle

Figure 11–4 shows a syncytial mass of cardiac muscle that has been stimulated at its centralmost point.

**Figure 11–4**

Instantaneous potentials develop on the surface of a cardiac muscle mass that has been depolarized in its center.

Before stimulation, all the exteriors of the muscle cells had been positive and the interiors negative. For reasons presented in Chapter 5 in the discussion of membrane potentials, as soon as an area of cardiac syncytium becomes depolarized, negative charges leak to the outsides of the depolarized muscle fibers, making this part of the surface electronegative, as represented by the negative signs in Figure 11–4. The remaining surface of the heart, which is still polarized, is represented by the positive signs. Therefore, a meter connected with its negative terminal on the area of depolarization and its positive terminal on one of the still-polarized areas, as shown *to the right* in the figure, records positively.

Two other electrode placements and meter readings are also demonstrated in Figure 11–4. *These should be studied carefully, and the reader should be able to explain the causes of the respective meter readings.* Because the depolarization spreads in all directions through the heart, the potential differences shown in the figure persist for only a few thousandths of a second, and the actual voltage measurements can be accomplished only with a high-speed recording apparatus.

### Flow of Electrical Currents in the Chest Around the Heart

Figure 11–5 shows the ventricular muscle lying within the chest. Even the lungs, although mostly filled with air, conduct electricity to a surprising extent, and fluids in other tissues surrounding the heart conduct electricity even more easily. Therefore, the heart is actually suspended in a conductive medium. When one portion of the ventricles depolarizes and therefore becomes electronegative with respect to the

C H A P T E R 1 3

# Cardiac Arrhythmias and Their Electrocardiographic Interpretation

Some of the most distressing types of heart malfunction occur not as a result of abnormal heart muscle but because of abnormal rhythm of the heart. For instance, sometimes the beat of the atria is not coordinated with the beat of the ventricles, so that the atria no longer function as primer pumps for the ventricles.

The purpose of this chapter is to discuss the physiology of common cardiac arrhythmias and their effects on heart pumping, as well as their diagnosis by electrocardiography. The causes of the cardiac arrhythmias are usually one or a combination of the following abnormalities in the rhythmicity-conduction system of the heart:

1. Abnormal rhythmicity of the pacemaker
2. Shift of the pacemaker from the sinus node to another place in the heart
3. Blocks at different points in the spread of the impulse through the heart
4. Abnormal pathways of impulse transmission through the heart
5. Spontaneous generation of spurious impulses in almost any part of the heart

## Abnormal Sinus Rhythms

### Tachycardia

The term "tachycardia" means *fast heart rate,* usually defined in an adult person as faster than 100 beats per minute. An electrocardiogram recorded from a patient with tachycardia is shown in Figure 13–1. This electrocardiogram is normal except that the heart rate, as determined from the time intervals between QRS complexes, is about 150 per minute instead of the normal 72 per minute.

The general causes of tachycardia include *increased body temperature, stimulation of the heart by the sympathetic nerves, or toxic conditions of the heart.*

The heart rate increases about 10 beats per minute for each degree Fahrenheit (18 beats per degree Celsius) increase in body temperature, up to a body temperature of about 105°F (40.5°C); beyond this, the heart rate may decrease because of progressive debility of the heart muscle as a result of the fever. Fever causes tachycardia because increased temperature increases the rate of metabolism of the sinus node, which in turn directly increases its excitability and rate of rhythm.

Many factors can cause the sympathetic nervous system to excite the heart, as we discuss at multiple points in this text. For instance, when a patient loses blood and passes into a state of shock or semishock, sympathetic reflex stimulation of the heart often increases the heart rate to 150 to 180 beats per minute.

Simple weakening of the myocardium usually increases the heart rate because the weakened heart does not pump blood into the arterial tree to a normal extent, and this elicits sympathetic reflexes to increase the heart rate.

### Bradycardia

The term "bradycardia" means a slow heart rate, usually defined as fewer than 60 beats per minute. Bradycardia is shown by the electrocardiogram in Figure 13–2.

**Bradycardia in Athletes.** The athlete's heart is larger and considerably stronger than that of a normal person, which allows the athlete's heart to pump a large stroke volume output per beat even during periods of rest. When the athlete is at rest, excessive quantities of blood pumped into the arterial tree with each beat initiate feedback circulatory reflexes or other effects to cause bradycardia.

**Figure 13–1**

Sinus tachycardia (lead I).

**Figure 13–2**

Sinus bradycardia (lead III).

**Figure 13–3**

Sinus arrhythmia as recorded by a cardiotachometer. To the left is the record when the subject was breathing normally; to the right, when breathing deeply.

**Vagal Stimulation as a Cause of Bradycardia.** Any circulatory reflex that stimulates the vagus nerves causes release of acetylcholine at the vagal endings in the heart, thus giving a parasympathetic effect. Perhaps the most striking example of this occurs in patients with *carotid sinus syndrome.* In these patients, the pressure receptors (baroreceptors) in the carotid sinus region of the carotid artery walls are excessively sensitive. Therefore, even mild external pressure on the neck elicits a strong baroreceptor reflex, causing intense vagal-acetylcholine effects on the heart, including extreme bradycardia. Indeed, sometimes this reflex is so powerful that it actually stops the heart for 5 to 10 seconds.

### Sinus Arrhythmia

Figure 13–3 shows a *cardiotachometer* recording of the heart rate, at first during normal and then (in the second half of the record) during deep respiration. A cardiotachometer is an instrument that records *by the height of successive spikes* the duration of the interval between the successive QRS complexes in the electrocardiogram. Note from this record that the heart rate increased and decreased no more than 5 per cent during quiet respiration (left half of the record). Then, *during deep respiration*, the heart rate increased and decreased with each respiratory cycle by as much as 30 per cent.

Sinus arrhythmia can result from any one of many circulatory conditions that alter the strengths of the sympathetic and parasympathetic nerve signals to the heart sinus node. In the "respiratory" type of sinus arrhythmia, as shown in Figure 13–3, this results mainly from "spillover" of signals from the medullary respiratory center into the adjacent vasomotor center during inspiratory and expiratory cycles of respiration. The spillover signals cause alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart.

**Figure 13–4**

Sinoatrial nodal block, with A-V nodal rhythm during the block period (lead III).

## Abnormal Rhythms That Result from Block of Heart Signals Within the Intracardiac Conduction Pathways

### Sinoatrial Block

In rare instances, the impulse from the sinus node is blocked before it enters the atrial muscle. This phenomenon is demonstrated in Figure 13–4, which shows sudden cessation of P waves, with resultant standstill of the atria. However, the ventricles pick up a new rhythm, the impulse usually originating spontaneously in the atrioventricular (A-V) node, so that the rate of the ventricular QRS-T complex is slowed but not otherwise altered.

### Atrioventricular Block

The only means by which impulses ordinarily can pass from the atria into the ventricles is through the *A-V bundle,* also known as the *bundle of His.* Conditions that can either decrease the rate of impulse conduction in this bundle or block the impulse entirely are as follows:

1. *Ischemia of the A-V node or A-V bundle fibers* often delays or blocks conduction from the atria to the ventricles. Coronary insufficiency can cause ischemia of the A-V node and bundle in the same way that it can cause ischemia of the myocardium.
2. *Compression of the A-V bundle* by scar tissue or by calcified portions of the heart can depress or block conduction from the atria to the ventricles.
3. *Inflammation of the A-V node or A-V bundle* can depress conductivity from the atria to the ventricles. Inflammation results frequently from

**Figure 13–5**

Prolonged P-R interval caused by first degree A-V heart block (lead II).

different types of myocarditis, caused, for example, by diphtheria or rheumatic fever.

4. *Extreme stimulation of the heart by the vagus nerves* in rare instances blocks impulse conduction through the A-V node. Such vagal excitation occasionally results from strong stimulation of the baroreceptors in people with *carotid sinus syndrome,* discussed earlier in relation to bradycardia.

## Incomplete Atrioventricular Heart Block

**Prolonged P-R (or P-Q) Interval—First Degree Block.** The usual lapse of time between *beginning* of the P wave and *beginning* of the QRS complex is about 0.16 second when the heart is beating at a normal rate. This so-called *P-R interval* usually decreases in length with faster heartbeat and increases with slower heartbeat. In general, when the P-R interval increases to greater than 0.20 second, the P-R interval is said to be prolonged, and the patient is said to have *first degree incomplete heart block.*

Figure 13–5 shows an electrocardiogram with prolonged P-R interval; the interval in this instance is about 0.30 second instead of the normal 0.20 or less. Thus, first degree block is defined as a *delay* of conduction from the atria to the ventricles but not actual blockage of conduction. The P-R interval seldom increases above 0.35 to 0.45 second because, by that time, conduction through the A-V bundle is depressed so much that conduction stops entirely. One means for determining the severity of some heart diseases—*acute rheumatic heart disease,* for instance—is to measure the P-R interval.

**Second Degree Block.** When conduction through the A-V bundle is slowed enough to increase the P-R interval to 0.25 to 0.45 second, the action potential sometimes is strong enough to pass through the bundle into the ventricles and sometimes is not strong enough. In this instance, there will be an atrial P wave but no QRS-T wave, and it is said that there are "dropped beats" of the ventricles. This condition is called *second degree heart block.*

Figure 13–6 shows P-R intervals of 0.30 second, as well as one dropped ventricular beat as a result of failure of conduction from the atria to the ventricles.

At times, every other beat of the ventricles is dropped, so that a "2:1 rhythm" develops, with the atria beating twice for every single beat of the ventricles. At other times, rhythms of 3:2 or 3:1 also develop.

**Complete A-V Block (Third Degree Block).** When the condition causing poor conduction in the A-V node or A-V bundle becomes severe, complete block of the impulse

**Figure 13–6**

Second degree A-V block, showing occasional failure of the ventricles to receive the excitatory signals (lead $V_3$).

**Figure 13–7**

Complete A-V block (lead II).

from the atria into the ventricles occurs. In this instance, the ventricles spontaneously establish their own signal, usually originating in the A-V node or A-V bundle. Therefore, the P waves become dissociated from the QRS-T complexes, as shown in Figure 13–7. Note that the *rate of rhythm of the atria* in this electrocardiogram is about 100 beats per minute, whereas the *rate of ventricular beat* is less than 40 per minute. Furthermore, there is no relation between the rhythm of the P waves and that of the QRS-T complexes because the ventricles have "escaped" from control by the atria, and they are beating at their own natural rate, controlled most often by rhythmical signals generated in the A-V node or A-V bundle.

**Stokes-Adams Syndrome—Ventricular Escape.** In some patients with A-V block, the total block comes and goes; that is, impulses are conducted from the atria into the ventricles for a period of time and then suddenly impulses are not conducted. The duration of block may be a few seconds, a few minutes, a few hours, or even weeks or longer before conduction returns. This condition occurs in hearts with borderline ischemia of the conductive system.

Each time A-V conduction ceases, the ventricles often do not start their own beating until after a delay of 5 to 30 seconds. This results from the phenomenon called *overdrive suppression.* This means that ventricular excitability is at first in a suppressed state because the ventricles have been driven by the atria at a rate greater than their natural rate of rhythm. However, after a few seconds, some part of the Purkinje system beyond the block, usually in the distal part of the A-V node beyond the blocked point in the node, or in the A-V bundle, begins discharging rhythmically at a rate of 15 to 40 times per minute and acting as the pacemaker of the ventricles. This is called *ventricular escape.*