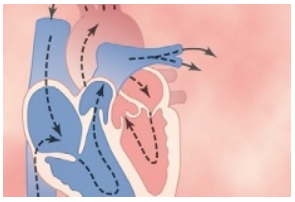
Cardiac Muscle; The Heart as a Pump and Function of the Heart Valves



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 cardiac 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 cardiac 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. The specialized excitatory and conductive fibers, however, 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 the histology 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 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.

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 rapid 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

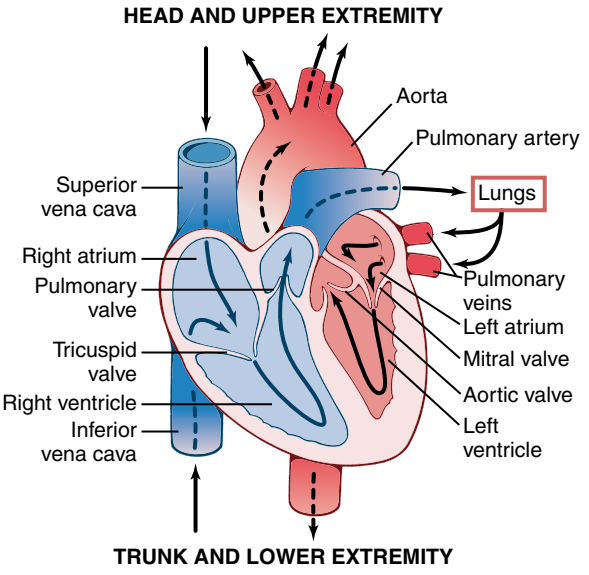


Figure 9-1 Structure of the heart, and course of blood flow through the heart chambers and heart valves.

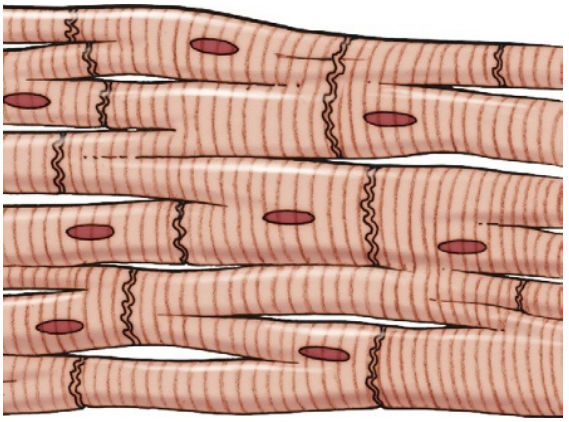


Figure 9-2 "Syncytial," interconnecting nature of cardiac muscle fibers.

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, from cell to cell throughout the latticework interconnections.

The heart actually is composed of two syncytiums: the *atrial syncytium,* which constitutes the walls of the two atria, and the *ventricular syncytium,* which 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 address the questions: Why is the action potential of cardiac muscle so long and why does it have a plateau, whereas that of skeletal muscle

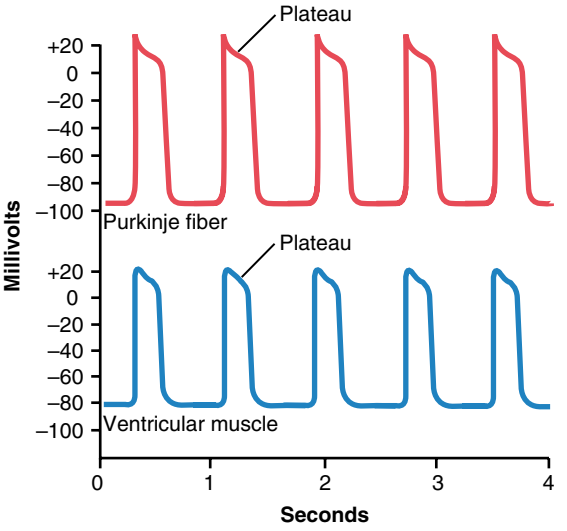


Figure 9-3 Rhythmical action potentials (in millivolts) from a Purkinje fiber and from a ventricular muscle fiber, recorded by means of microelectrodes.

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 ½₅₀ 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 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).

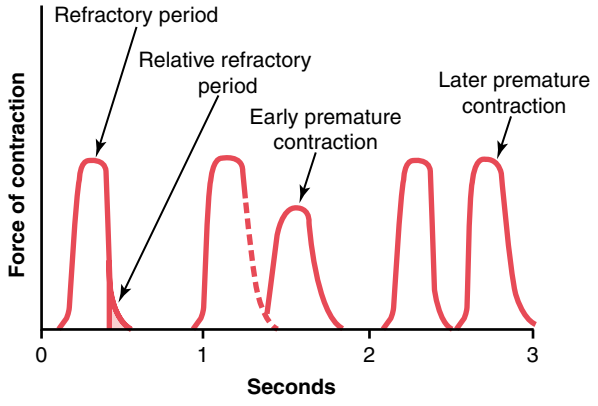


Figure 9-4 Force of ventricular heart muscle contraction, showing also duration of the refractory period and relative refractory period, plus the effect of premature contraction. Note that premature contractions do not cause wave summation, as occurs in skeletal muscle.

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 heart 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, calcium ions also diffuse into the sarcoplasm from the T tubules themselves at the time of the action potential, which opens voltage-dependent calcium channels in the membrane of the T tubule (Figure 9-5). Calcium entering the cell then activates *calcium release channels,* also called *ryanodine receptor channels,* in the sarcoplasmic reticulum membrane, triggering the release of calcium into the sarcoplasm. Calcium ions in the sarcoplasm then interact with troponin to initiate cross-bridge formation and contraction by the same basic mechanism as described for skeletal muscle in Chapter 6.

Without the 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. The T tubules of cardiac muscle, however, 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. In fact, a heart placed in a calcium-free solution will quickly stop beating. 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

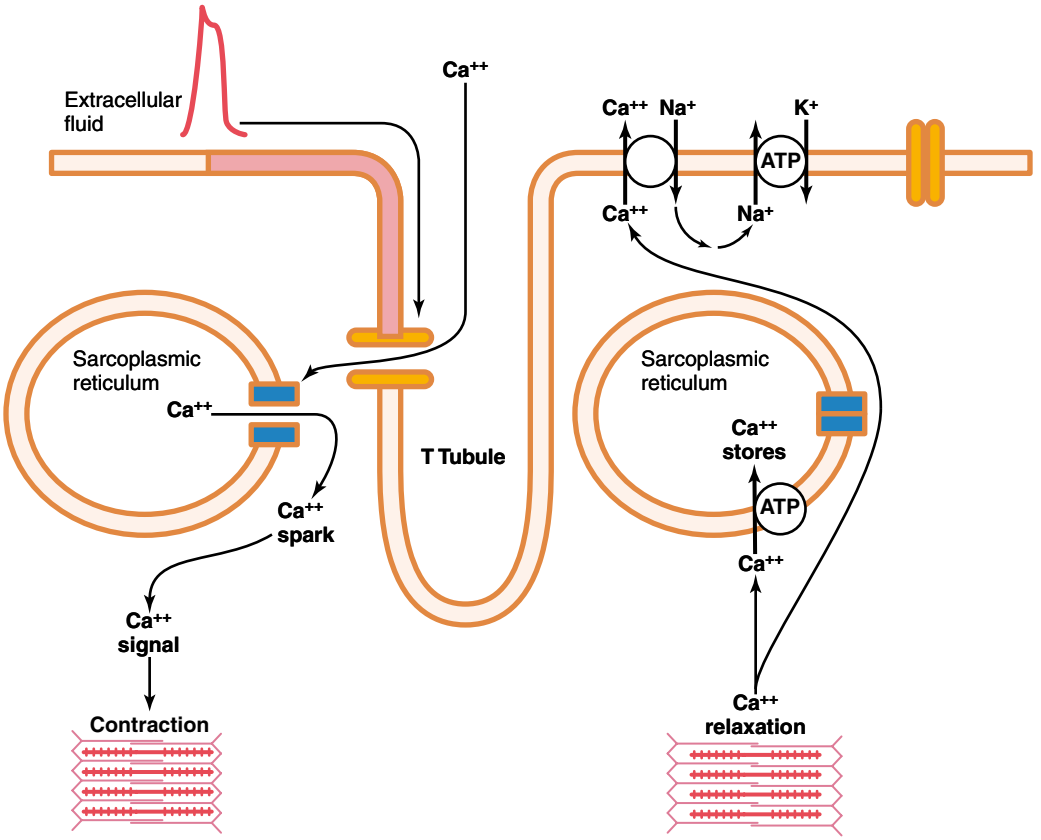


Figure 9-5 Mechanisms of excitation-contraction coupling and relaxation in cardiac muscle.

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 (i.e., the availability of calcium ions to cause cardiac muscle contraction) depends to a great extent on the extracellular fluid calcium ion concentration.

In 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.

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. Transport of calcium back into the sarcoplasmic reticulum is achieved with the help of a calcium-ATPase pump (see Figure 9-5). Calcium ions are also removed from the cell by a sodium-calcium exchanger. The sodium that enters the cell during this exchange is then transported out of the cell by the sodium-potassium ATPase pump. 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.

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.*

The total *duration of the cardiac cycle,* including systole and diastole, is the reciprocal of the heart rate. For example, if heart rate is 72 beats/min, the duration of the cardiac cycle is 1/72 beats/min—about 0.0139 minutes per beat, or 0.833 second per beat.

Figure 9-6 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 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.

Effect of Heart Rate on Duration of Cardiac Cycle. When heart rate increases, the duration of each cardiac cycle decreases, including the contraction and relaxation phases. The duration of the action potential and the period of contraction (systole) also decrease, but not by as great a percentage as does the relaxation phase (diastole). At a normal heart rate of 72 beats/min, systole comprises about 0.4 of the entire cardiac cycle. At three times the normal heart rate, systole is about 0.65 of the entire cardiac cycle. This means that the heart beating at a very fast rate does not remain relaxed long enough to allow complete filling of the cardiac chambers before the next contraction.

Relationship of the Electrocardiogram to the Cardiac Cycle

The electrocardiogram in Figure 9-6 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 percent of the blood flows directly through the atria into the ventricles even before the atria contract. Then, atrial contraction usually causes an additional 20 percent filling of the ventricles. Therefore, the atria simply function as primer pumps that increase the ventricular pumping effectiveness as much as 20 percent. However, the heart can continue to operate under most conditions

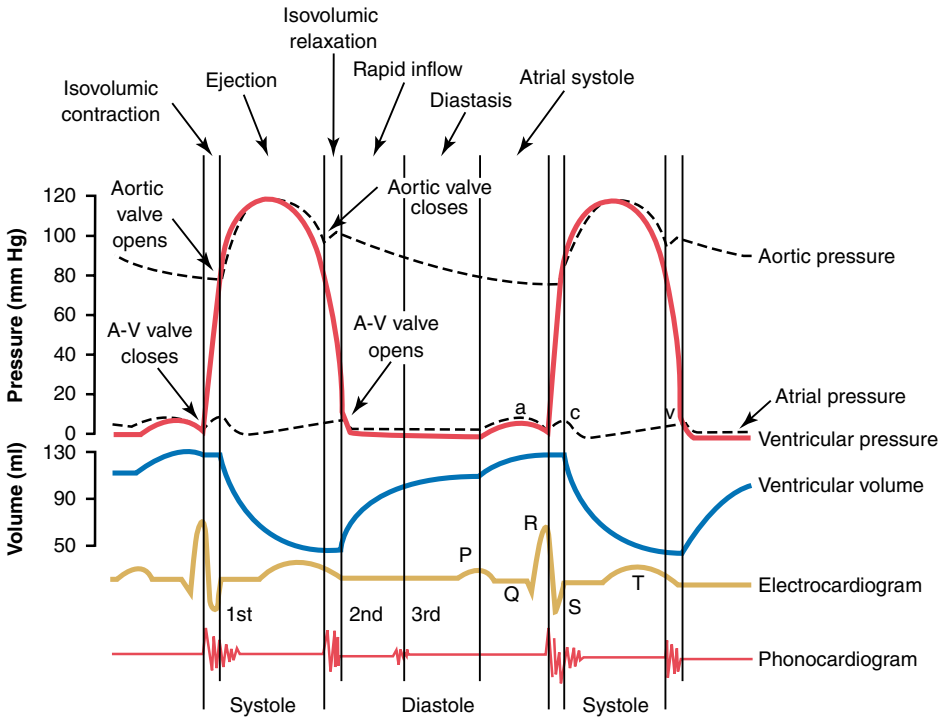


Figure 9-6 Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram.

even without this extra 20 percent effectiveness because it normally has the capability of pumping 300 to 400 percent 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—a, c, and v Waves. In the atrial pressure curve of Figure 9-6, 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 Diastole. 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 9-6. 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 percent 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 9-6, 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 percent of the blood emptying occurring during the first third of the period of ejection and the remaining 30 percent 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 ml. This volume is called the *end-diastolic volume.* Then, as the ventricles empty during systole, the volume decreases about 70 ml, which is called the *stroke volume output.* The remaining volume in each ventricle, about 40 to 50 ml, 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 percent.

When the heart contracts strongly, the end-systolic volume can be decreased to as little as 10 to 20 ml. 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 ml 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 9-7 for the left ventricle, close and open *passively.* That is, they close when a backward pressure gradient pushes

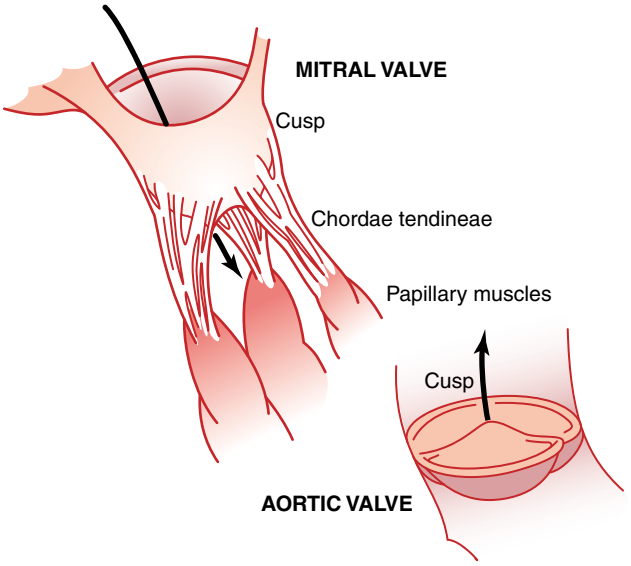


Figure 9-7 Mitral and aortic valves (the left ventricular valves).

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 9-7 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.

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 9-7) 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 9-6, 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 percent 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 percent of the total work output may be required to create kinetic energy of blood flow.

Graphical Analysis of Ventricular Pumping

Figure 9-8 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.

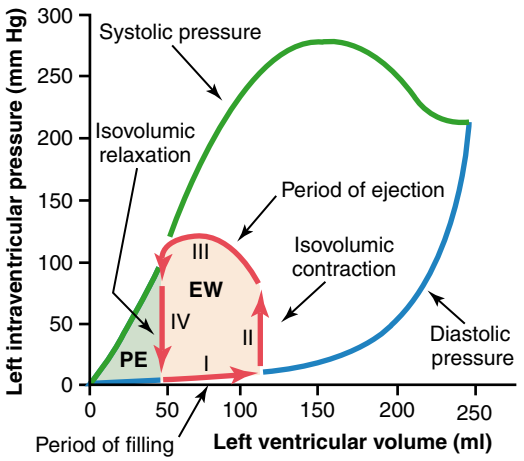


Figure 9-8 Relationship between left ventricular volume and intraventricular pressure during diastole and systole. Also shown by the heavy red lines is the "volume-pressure diagram," demonstrating changes in intraventricular volume and pressure during the normal cardiac cycle. EW, net external work.

Until the volume of the noncontracting ventricle rises above about 150 ml, the "diastolic" pressure does not increase greatly. Therefore, up to this volume, blood can flow easily into the ventricle from the atrium. Above 150 ml, 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 ml. 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 9-8, 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 9-8 form a loop called the *volume-pressure diagram* of the cardiac cycle for normal function of the *left* ventricle. A more detailed version of this loop is shown in Figure 9-9. 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 50 ml and a diastolic pressure of 2 to 3 mm Hg. The amount of blood that remains in the ventricle after the previous heartbeat, 50 ml, is called the *end-systolic volume.* As venous blood flows into the ventricle from the left atrium, the ventricular volume normally increases to about 120 ml, called the *end-diastolic volume,* an increase of 70 ml. Therefore, the volume-pressure diagram during phase I extends along the line labeled "I," from point A to point B, with the volume increasing to 120 ml and the diastolic pressure rising to about 5 to 7 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 point C.

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," or "period of ejection," 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 (point D), 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 50 ml of blood left in the ventricle and at an atrial pressure of 2 to 3 mm Hg.

Readers well trained in the basic principles of physics will recognize that the area subtended by this functional

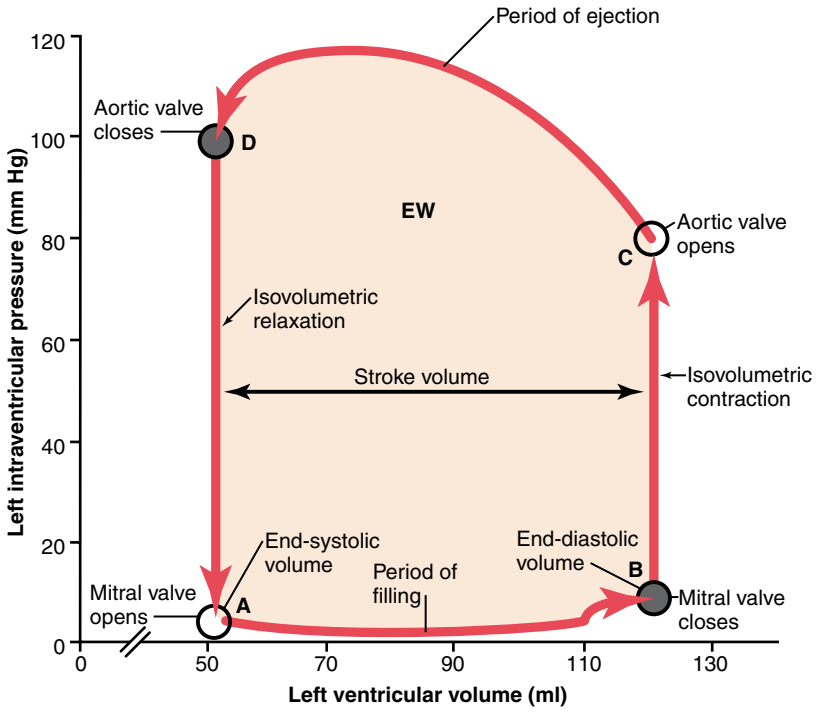


Figure 9-9 The "volume-pressure diagram" demonstrating changes in intraventricular volume and pressure during a single cardiac cycle (*red line*). The tan shaded area represents the net external work (*EW*) output by the left ventricle during the cardiac cycle.

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 aorta leading from the ventricle. In Figure 9-8, 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. Approximately 70 to 90 percent of this energy is normally derived from oxidative metabolism of fatty acids with about 10 to 30 percent coming from 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.

Experimental studies have shown that oxygen consumption of the heart and the chemical energy expended during contraction are directly related to the total shaded area in Figure 9-8. This shaded portion consists of the *external work* (EW) as explained earlier and an additional portion called the *potential energy,* labeled PE. The potential energy represents additional work that could be accomplished by contraction of the ventricle if the ventricle should empty completely all the blood in its chamber with each contraction.

Oxygen consumption has also been shown to be nearly proportional to the *tension* that occurs in the heart muscle during contraction multiplied by the *duration of time* that the contraction persists, called the *tension-time index*. Because tension is high when systolic pressure is high, correspondingly more oxygen is used. Also, much more chemical energy is expended even at normal systolic pressures when the ventricle is abnormally dilated because the heart muscle tension during contraction is proportional to pressure times the diameter of the ventricle. This becomes especially important in heart failure where the heart ventricle is dilated and, paradoxically, the amount of chemical energy required for a given amount of work output is greater than normal even though the heart is already failing.

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 percent. In heart failure, this can decrease to as low as 5 to 10 percent.

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 normally 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 Otto Frank and Ernest Starling, two great physiologists of a century ago. Basically, the Frank-Starling mechanism means that the greater the heart muscle is stretched during filling, the greater is the force of contraction and the greater the quantity of blood pumped into the aorta. Or, stated another way: *Within physiologic limits, the heart pumps all the blood that returns to it by the way of the veins.*

What Is the Explanation of the Frank-Starling Mechanism? When an extra amount of blood flows into the ventricles, the cardiac muscle itself is stretched to greater length. This in turn causes the muscle to contract with increased force because the actin and myosin filaments are brought to a more nearly optimal degree of overlap for force generation. Therefore, the ventricle, because of its increased pumping, automatically pumps the extra blood into the arteries.

This ability of stretched muscle, up to an optimal length, to contract with increased work output is characteristic of all striated muscle, as explained in Chapter 6, and is not simply a characteristic of cardiac muscle.

In addition to the important effect of lengthening the heart muscle, still another factor increases heart pumping when its volume is increased. Stretch of the right atrial wall directly increases the heart rate by 10 to 20 percent; this, too, helps increase the amount of blood pumped each minute, although its contribution is much less than that of the Frank-Starling mechanism.

Ventricular Function Curves

One of the best ways to express the functional ability of the ventricles to pump blood is by *ventricular function curves,* as shown in Figures 9-10 and 9-11. Figure 9-10 shows a type of ventricular function curve called the *stroke work output curve.* Note that as the atrial pressure for each side of the heart increases, the stroke work output for that side increases until it reaches the limit of the ventricle's pumping ability.

Figure 9-11 shows another type of ventricular function curve called the *ventricular volume output curve.* The two curves of this figure represent function of the two ventricles of the human heart based on data extrapolated from lower animals. As the right and left atrial pressures increase, the respective ventricular volume outputs per minute also increase.

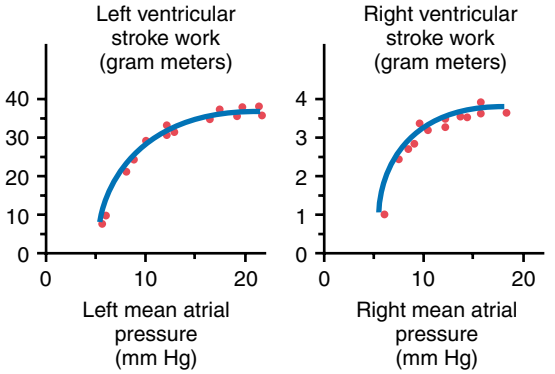


Figure 9-10 Left and right ventricular function curves recorded from dogs, depicting *ventricular stroke work output* as a function of left and right mean atrial pressures. (Curves reconstructed from data in Sarnoff SJ: Myocardial contractility as described by ventricular function curves. Physiol Rev 35:107, 1955.)

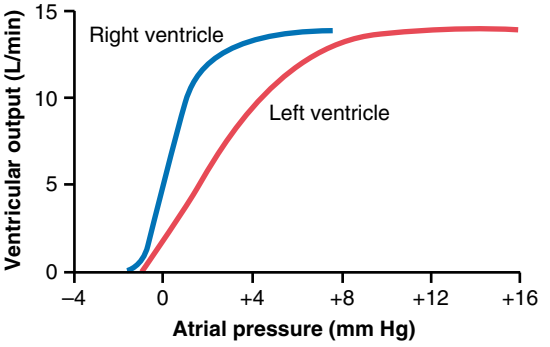


Figure 9-11 Approximate normal right and left *ventricular volume output curves* for the normal resting human heart as extrapolated from data obtained in dogs and data from human beings.

Thus, *ventricular function curves* are another way of expressing the Frank-Starling mechanism of the heart. That is, as the ventricles fill in response to higher atrial pressures, each ventricular volume and strength of cardiac muscle contraction increase, causing the heart to pump increased quantities of blood into the arteries.

Control of the Heart by the Sympathetic and Parasympathetic Nerves

The pumping effectiveness of the heart also is controlled by the *sympathetic* and *parasympathetic (vagus)* nerves, which abundantly supply the heart, as shown in Figure 9-12. For given levels of atrial pressure, the amount of blood pumped each minute *(cardiac output)* often can be increased more than 100 percent by sympathetic stimulation. By contrast, the output can be decreased to as low as zero or almost zero by vagal (parasympathetic) stimulation.

Mechanisms of Excitation of the Heart by the Sympathetic Nerves. Strong sympathetic stimulation can increase the heart rate in young adult humans from the normal rate of 70 beats/min up to 180 to 200 and, rarely, even 250 beats/min. Also, sympathetic stimulation increases the force of heart contraction to as much as double normal, thereby increasing the volume of blood pumped and increasing the ejection pressure. Thus, sympathetic stimulation often can increase the maximum cardiac output as much as twofold to threefold, in addition to the increased output caused by the Frank-Starling mechanism already discussed.

Conversely, *inhibition* of the sympathetic nerves to the heart can decrease cardiac pumping to a moderate extent in the following way: Under normal conditions, the sympathetic nerve fibers to the heart discharge continuously at a slow rate that maintains pumping at about 30 percent above that with no sympathetic stimulation. Therefore, when the activity of the sympathetic nervous system is depressed below normal, this decreases both heart rate and strength of ventricular muscle contraction, thereby decreasing the level of cardiac pumping as much as 30 percent below normal.

Parasympathetic (Vagal) Stimulation of the Heart. Strong stimulation of the parasympathetic nerve fibers in the vagus nerves to the heart can stop the heartbeat for a few seconds, but then the heart usually "escapes" and beats at a rate of 20 to 40 beats/min as long as the parasympathetic stimulation continues. In addition, strong vagal stimulation can decrease the strength of heart muscle contraction by 20 to 30 percent.

The vagal fibers are distributed mainly to the atria and not much to the ventricles, where the power contraction of the heart occurs. This explains the effect of vagal stimulation mainly to decrease heart rate rather than to decrease greatly the strength of heart contraction. Nevertheless, the great decrease in heart rate combined with a slight decrease in heart contraction strength can decrease ventricular pumping 50 percent or more.

Effect of Sympathetic or Parasympathetic Stimulation on the Cardiac Function Curve. Figure 9-13 shows four cardiac function curves. They are similar to the ventricular function curves of Figure 9-11. However, they represent function of the entire heart rather than of a single ventricle; they show the relation between right atrial pressure at the input of the right heart and cardiac output from the left ventricle into the aorta.

The curves of Figure 9-13 demonstrate that at any given right atrial pressure, the cardiac output increases during increased sympathetic stimulation and decreases during increased parasympathetic stimulation. These changes in output caused by autonomic nervous system stimulation result both from *changes in heart rate* and from *changes in contractile strength of the heart* because both change in response to the nerve stimulation.

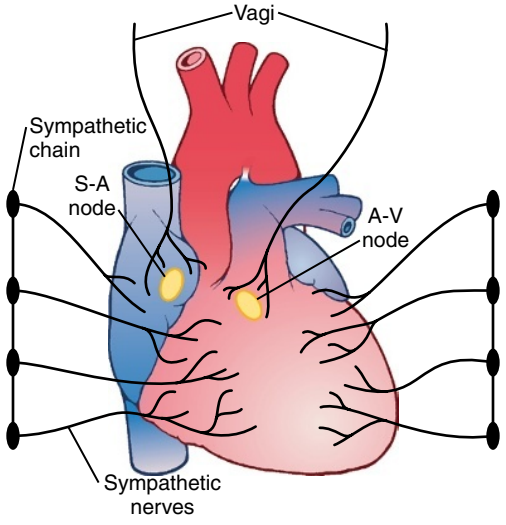


Figure 9-12 Cardiac *sympathetic* and *parasympathetic* nerves. (The vagus nerves to the heart are parasympathetic nerves.)

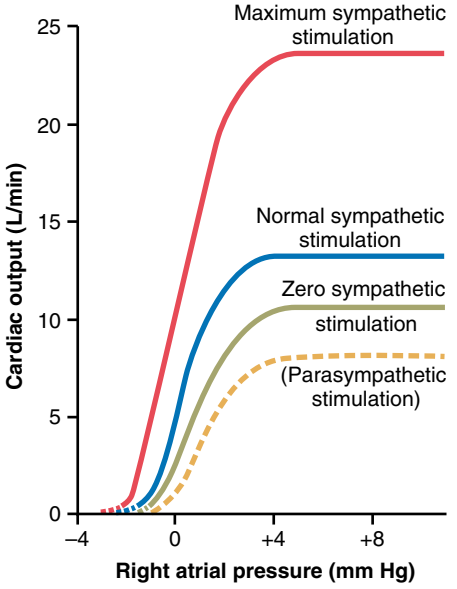


Figure 9-13 Effect on the cardiac output curve of different degrees of sympathetic or parasympathetic stimulation.

Effect of Potassium and Calcium Ions on Heart Function

In the discussion of membrane potentials in Chapter 5, it was pointed out that potassium ions have a marked effect on membrane potentials, and in Chapter 6 it was noted that calcium ions play an especially important role in activating the muscle contractile process. Therefore, it is to be expected that the concentration of each of these two ions in the extracellular fluids should also have important effects on cardiac pumping.

Effect of Potassium Ions. Excess potassium in the extracellular fluids causes the heart to become dilated and flaccid and also slows the heart rate. Large quantities also can block conduction of the cardiac impulse from the atria to the ventricles through the A-V bundle. Elevation of potassium concentration to only 8 to 12 mEq/L—two to three times the normal value—can cause such weakness of the heart and abnormal rhythm that death occurs.

These effects result partially from the fact that a high potassium concentration in the extracellular fluids decreases the resting membrane potential in the cardiac muscle fibers, as explained in Chapter 5. That is, high extracellular fluid potassium concentration partially depolarizes the cell membrane, causing the membrane potential to be less negative. As the membrane potential decreases, the intensity of the action potential also decreases, which makes contraction of the heart progressively weaker.

Effect of Calcium Ions. An excess of calcium ions causes effects almost exactly opposite to those of potassium ions, causing the heart to go toward spastic contraction. This is caused by a direct effect of calcium ions to initiate the cardiac contractile process, as explained earlier in the chapter.

Conversely, deficiency of calcium ions causes cardiac *flaccidity*, similar to the effect of high potassium. Fortunately, calcium ion levels in the blood normally are regulated within a very narrow range. Therefore, cardiac effects of abnormal calcium concentrations are seldom of clinical concern.

Effect of Temperature on Heart Function

Increased body temperature, as occurs when one has fever, causes a greatly increased heart rate, sometimes to double normal. Decreased temperature causes a greatly decreased heart rate, falling to as low as a few beats per minute when a person is near death from hypothermia in the body temperature range of 60° to 70°F. These effects presumably result from the fact that heat increases the permeability of the cardiac muscle membrane to ions that control heart rate, resulting in acceleration of the self-excitation process.

Contractile strength of the heart often is enhanced temporarily by a moderate increase in temperature, as occurs during body exercise, but prolonged elevation of temperature exhausts the metabolic systems of the heart and eventually causes weakness. Therefore, optimal function of the heart depends greatly on proper control of body temperature by the temperature control mechanisms explained in Chapter 73.

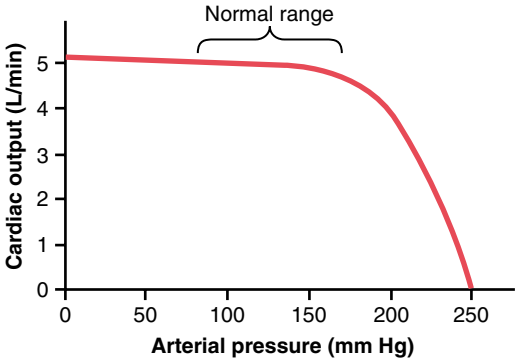


Figure 9-14 Constancy of cardiac output up to a pressure level of 160 mm Hg. Only when the arterial pressure rises above this normal limit does the increasing pressure load cause the cardiac output to fall significantly.

Increasing the Arterial Pressure Load (up to a Limit) Does Not Decrease the Cardiac Output

Note in Figure 9-14 that increasing the arterial pressure in the aorta does not decrease the cardiac output until the mean arterial pressure rises above about 160 mm Hg. In other words, during normal function of the heart at normal systolic arterial pressures (80 to 140 mm Hg), the cardiac output is determined almost entirely by the ease of blood flow through the body's tissues, which in turn controls *venous return* of blood to the heart. This is the principal subject of Chapter 20.

Bibliography

Bers DM: Altered cardiac myocyte Ca regulation in heart failure, *Physiology (Bethesda)* 21:380, 2006.

Bers DM: Calcium cycling and signaling in cardiac myocytes, *Annu Rev Physiol* 70:23, 2008.

Brette F, Orchard C: T-tubule function in mammalian cardiac myocytes, *Circ Res* 92:1182, 2003.

Chantler PD, Lakatta EG, Najjar SS: Arterial-ventricular coupling: mechanistic insights into cardiovascular performance at rest and during exercise, *J Appl Physiol* 105:1342, 2008.

Cheng H, Lederer WJ: Calcium sparks, *Physiol Rev* 88:1491, 2008.

Clancy CE, Kass RS: Defective cardiac ion channels: from mutations to clinical syndromes, *J Clin Invest* 110:1075, 2002.

Couchonnal LF, Anderson ME: The role of calmodulin kinase II in myocardial physiology and disease, *Physiology (Bethesda)* 23:151, 2008.

Fuchs F, Smith SH: Calcium, cross-bridges, and the Frank-Starling relationship, *News Physiol Sci* 16:5, 2001.

Guyton AC: Determination of cardiac output by equating venous return curves with cardiac response curves, *Physiol Rev* 35:123, 1955.

Guyton AC, Jones CE, Coleman TG: *Circulatory Physiology: Cardiac Output and Its Regulation*, 2nd ed, Philadelphia, 1973, WB Saunders.

Kang M, Chung KY, Walker JW: G-protein coupled receptor signaling in myocardium: not for the faint of heart, *Physiology (Bethesda)* 22:174, 2007.

Knaapen P, Germans T, Knuuti J, et al: Myocardial energetic and efficiency: current status of the noninvasive approach, *Circulation* 115:918, 2007.

Mangoni ME, Nargeot J: Genesis and regulation of the heart automaticity, *Physiol Rev* 88:919, 2008.

Korzick DH: Regulation of cardiac excitation-contraction coupling: a cellular update, *Adv Physiol Educ* 27:192, 2003.

Olson EN: A decade of discoveries in cardiac biology, *Nat Med* 10:467, 2004.

Rudy Y, Ackerman MJ, Bers DM, et al: Systems approach to understanding electromechanical activity in the human heart: a National Heart, Lung, and Blood Institute workshop summary, *Circulation* 118:1202, 2008.

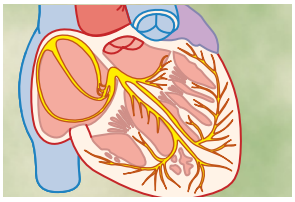
Saks V, Dzeja P, Schlattner U, et al: Cardiac system bioenergetics: metabolic basis of the Frank-Starling law, *J Physiol* 571:253, 2006.

Sarnoff SJ: Myocardial contractility as described by ventricular function curves, *Physiol Rev* 35:107, 1955.

Starling EH: *The Linacre Lecture on the Law of the Heart*, London, 1918, Longmans Green.

This page intentionally left blank

Rhythmical Excitation of the Heart



The heart is endowed with a special system for (1) generating rhythmical electrical impulses to cause rhythmical contraction of the heart muscle and (2) conducting these impulses rapidly through the heart. When this system functions normally, the atria contract about one sixth of a second ahead of ventricular contraction, which allows filling of the ventricles before they pump the blood through the lungs and peripheral circulation. Another special importance of the system is that it allows all portions of the ventricles to contract almost simultaneously, which is essential for most effective pressure generation in the ventricular chambers.

This rhythmical and conductive system of the heart is susceptible to damage by heart disease, especially by ischemia of the heart tissues resulting from poor coronary blood flow. The effect is often a bizarre heart rhythm or abnormal sequence of contraction of the heart chambers, and the pumping effectiveness of the heart often is affected severely, even to the extent of causing death.

Specialized Excitatory and Conductive System of the Heart

Figure 10-1 shows the specialized excitatory and conductive system of the heart that controls cardiac contractions. The figure shows the sinus node (also called sinoatrial or S-A node), in which the normal rhythmical impulses are generated; the internodal pathways that conduct impulses from the sinus node to the atrioventricular (A-V) node; the A-V node, in which impulses from the atria are delayed before passing into the ventricles; the A-V bundle, which conducts impulses from the atria into the ventricles; and the left and right bundle branches of Purkinje fibers, which conduct the cardiac impulses to all parts of the ventricles.

Sinus (Sinoatrial) Node

The sinus node (also called *sinoatrial node*) is a small, flattened, ellipsoid strip of specialized cardiac muscle about 3 millimeters wide, 15 millimeters long, and 1 millimeter thick. It is located in the superior posterolateral wall of the right atrium immediately below and slightly lateral to the opening of the superior vena cava. The fibers of this node have almost no contractile muscle filaments and are each only 3 to 5 micrometers in diameter, in contrast to a diameter of 10 to 15 micrometers for the surrounding atrial muscle fibers. However, the sinus nodal fibers connect directly with the atrial muscle fibers so that any action potential that begins in the sinus node spreads immediately into the atrial muscle wall.

Automatic Electrical Rhythmicity of the Sinus Fibers

Some cardiac fibers have the capability of *self-excitation*, a process that can cause automatic rhythmical discharge and contraction. This is especially true of the fibers of the heart's specialized conducting system, including the fibers of the sinus node. For this reason, the sinus node ordinarily controls the rate of beat of the entire heart, as discussed in detail later in this chapter. First, let us describe this automatic rhythmicity.

Mechanism of Sinus Nodal Rhythmicity. Figure 10-2 shows action potentials recorded from inside a sinus nodal fiber for three heartbeats and, by comparison, a single ventricular muscle fiber action potential. Note that the "resting membrane potential" of the sinus nodal fiber between discharges has a negativity of about −55 to −60 millivolts, in comparison with −85 to −90 millivolts for the ventricular muscle fiber. The cause of this lesser negativity is that the cell membranes of the sinus fibers are naturally leaky to sodium and calcium ions, and positive charges of the entering sodium and calcium ions neutralize some of the intracellular negativity.

Before attempting to explain the rhythmicity of the sinus nodal fibers, first recall from the discussions of Chapters 5 and 9 that cardiac muscle has three types of membrane ion channels that play important roles in causing the voltage changes of the action potential. They are (1) *fast sodium channels*, (2) *slow sodium-calcium channels*, and (3) *potassium channels*.

Opening of the fast sodium channels for a few 10,000ths of a second is responsible for the rapid upstroke spike of the action potential observed in ventricular muscle, because of rapid influx of positive sodium ions to the

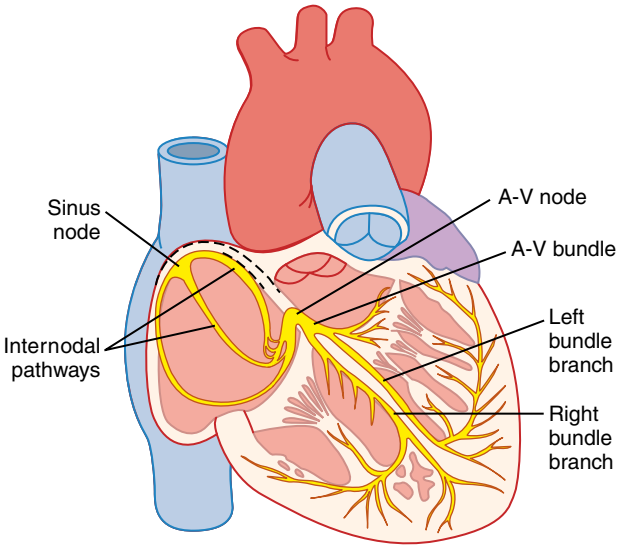


Figure 10-1 Sinus node and the Purkinje system of the heart, showing also the A-V node, atrial internodal pathways, and ventricular bundle branches.

interior of the fiber. Then the "plateau" of the ventricular action potential is caused primarily by slower opening of the slow sodium-calcium channels, which lasts for about 0.3 second. Finally, opening of potassium channels allows diffusion of large amounts of positive potassium ions in the outward direction through the fiber membrane and returns the membrane potential to its resting level.

But there is a difference in the function of these channels in the sinus nodal fiber because the "resting" potential is much less negative—only –55 millivolts in the nodal fiber instead of the –90 millivolts in the ventricular muscle fiber. At this level of –55 millivolts, the fast sodium channels mainly have already become "inactivated," which means that they have become blocked. The cause of this is that any time the membrane potential remains less negative than about –55 millivolts for more than a few milliseconds, the inactivation gates on the inside of the cell membrane that close the fast sodium channels become closed and remain so. Therefore, only the slow sodium-calcium channels can open (i.e., can become "activated")

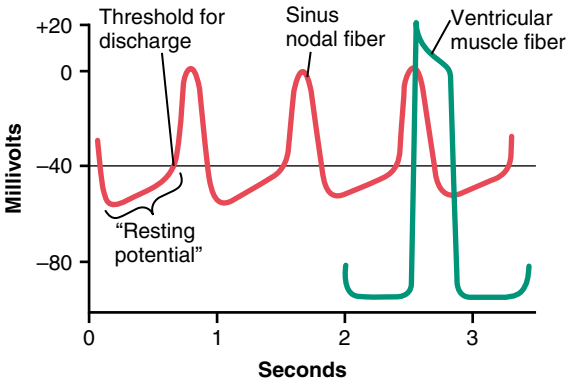


Figure 10-2 Rhythmical discharge of a sinus nodal fiber. Also, the sinus nodal action potential is compared with that of a ventricular muscle fiber.

and thereby cause the action potential. As a result, the atrial nodal action potential is slower to develop than the action potential of the ventricular muscle. Also, after the action potential does occur, return of the potential to its negative state occurs slowly as well, rather than the abrupt return that occurs for the ventricular fiber.

Self-Excitation of Sinus Nodal Fibers. Because of the high sodium ion concentration in the extracellular fluid outside the nodal fiber, as well as a moderate number of already open sodium channels, positive sodium ions from outside the fibers normally tend to leak to the inside. Therefore, between heartbeats, influx of positively charged sodium ions causes a slow rise in the resting membrane potential in the positive direction. Thus, as shown in Figure 10-2, the "resting" potential gradually rises and becomes less negative between each two heartbeats. When the potential reaches a threshold voltage of about –40 millivolts, the sodium-calcium channels become "activated," thus causing the action potential. Therefore, basically, the inherent leakiness of the sinus nodal fibers to sodium and calcium ions causes their self-excitation.

Why does this leakiness to sodium and calcium ions not cause the sinus nodal fibers to remain depolarized all the time? The answer is that two events occur during the course of the action potential to prevent this. First, the sodium-calcium channels become inactivated (i.e., they close) within about 100 to 150 milliseconds after opening, and second, at about the same time, greatly increased numbers of potassium channels open. Therefore, influx of positive calcium and sodium ions through the sodium-calcium channels ceases, while at the same time large quantities of positive potassium ions diffuse out of the fiber. Both of these effects reduce the intracellular potential back to its negative resting level and therefore terminate the action potential. Furthermore, the potassium channels remain open for another few tenths of a second, temporarily continuing movement of positive charges out of the cell, with resultant excess negativity inside the fiber; this is called *hyperpolarization.* The hyperpolarization state initially carries the "resting" membrane potential down to about –55 to –60 millivolts at the termination of the action potential.

Why is this new state of hyperpolarization not maintained forever? The reason is that during the next few tenths of a second after the action potential is over, progressively more and more potassium channels close. The inward-leaking sodium and calcium ions once again overbalance the outward flux of potassium ions, and this causes the "resting" potential to drift upward once more, finally reaching the threshold level for discharge at a potential of about –40 millivolts. Then the entire process begins again: self-excitation to cause the action potential, recovery from the action potential, hyperpolarization after the action potential is over, drift of the "resting" potential to threshold, and finally re-excitation to elicit another cycle. This process continues indefinitely throughout a person's life.

Internodal Pathways and Transmission of the Cardiac Impulse Through the Atria

The ends of the sinus nodal fibers connect directly with surrounding atrial muscle fibers. Therefore, action potentials originating in the sinus node travel outward into these atrial muscle fibers. In this way, the action potential spreads through the entire atrial muscle mass and, eventually, to the A-V node. The velocity of conduction in most atrial muscle is about 0.3 m/sec, but conduction is more rapid, about 1 m/sec, in several small bands of atrial fibers. One of these, called the *anterior interatrial band,* passes through the anterior walls of the atria to the left atrium. In addition, three other small bands curve through the anterior, lateral, and posterior atrial walls and terminate in the A-V node; shown in Figures 10-1 and 10-3, these are called, respectively, the *anterior, middle, and posterior internodal pathways*. The cause of more rapid velocity of conduction in these bands is the presence of specialized conduction fibers. These fibers are similar to even more rapidly conducting "Purkinje fibers" of the ventricles, which are discussed as follows.

Atrioventricular Node and Delay of Impulse Conduction from the Atria to the Ventricles

The atrial conductive system is organized so that the cardiac impulse does not travel from the atria into the ventricles too rapidly; this delay allows time for the atria to empty their blood into the ventricles before ventricular contraction begins. It is primarily the A-V node and its adjacent conductive fibers that delay this transmission into the ventricles.

The A-V node is located in the posterior wall of the right atrium immediately behind the tricuspid valve, as shown in Figure 10-1. And Figure 10-3 shows diagrammatically the different parts of this node, plus its connections with the entering atrial internodal pathway fibers and the exiting A-V bundle. The figure also shows the approximate intervals of time in fractions of a second between initial onset of the cardiac impulse in the sinus node and its subsequent appearance in the A-V nodal system. Note that the impulse, after traveling through the internodal pathways, reaches the A-V node about 0.03 second after its origin in the sinus node. Then there is a delay of another 0.09 second in the A-V node itself before the impulse enters the penetrating portion of the A-V bundle, where it passes into the ventricles. A final delay of another 0.04 second occurs mainly in this penetrating A-V bundle, which is composed of multiple small fascicles passing through the fibrous tissue separating the atria from the ventricles.

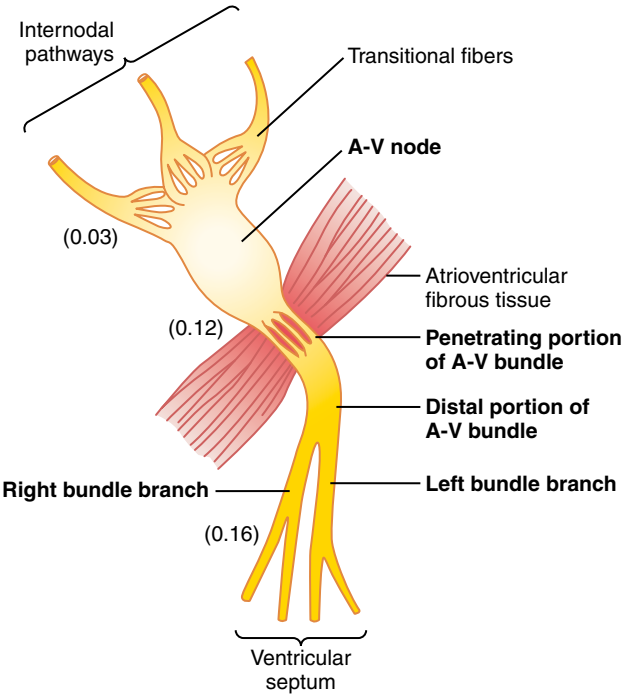


Figure 10-3 Organization of the A-V node. The numbers represent the interval of time from the origin of the impulse in the sinus node. The values have been extrapolated to human beings.

Thus, the total delay in the A-V nodal and A-V bundle system is about 0.13 second. This, in addition to the initial conduction delay of 0.03 second from the sinus node to the A-V node, makes a total delay of 0.16 second before the excitatory signal finally reaches the contracting muscle of the ventricles.

Cause of the Slow Conduction. The slow conduction in the transitional, nodal, and penetrating A-V bundle fibers is caused mainly by diminished numbers of gap junctions between successive cells in the conducting pathways, so there is great resistance to conduction of excitatory ions from one conducting fiber to the next. Therefore, it is easy to see why each succeeding cell is slow to be excited.

Rapid Transmission in the Ventricular Purkinje System

Special Purkinje fibers lead from the A-V node through the A-V bundle into the ventricles. Except for the initial portion of these fibers where they penetrate the A-V fibrous barrier, they have functional characteristics that are quite the opposite of those of the A-V nodal fibers. They are very large fibers, even larger than the normal ventricular muscle fibers, and they transmit action potentials at a velocity of 1.5 to 4.0 m/sec, a velocity about 6 times that in the usual ventricular muscle and 150 times that in some of the A-V nodal fibers. This allows almost instantaneous transmission of the cardiac impulse throughout the entire remainder of the ventricular muscle.

The rapid transmission of action potentials by Purkinje fibers is believed to be caused by a very high level of permeability of the gap junctions at the intercalated discs between the successive cells that make up the Purkinje fibers. Therefore, ions are transmitted easily from one cell to the next, thus enhancing the velocity of transmission. The Purkinje fibers also have very few myofibrils, which means that they contract little or not at all during the course of impulse transmission.

One-Way Conduction Through the A-V Bundle. A special characteristic of the A-V bundle is the inability, except in abnormal states, of action potentials to travel backward from the ventricles to the atria. This prevents re-entry of cardiac impulses by this route from

the ventricles to the atria, allowing only forward conduction from the atria to the ventricles.

Furthermore, it should be recalled that everywhere, except at the A-V bundle, the atrial muscle is separated from the ventricular muscle by a continuous fibrous barrier, a portion of which is shown in Figure 10-3. This barrier normally acts as an insulator to prevent passage of the cardiac impulse between atrial and ventricular muscle through any other route besides forward conduction through the A-V bundle itself. (In rare instances, an abnormal muscle bridge does penetrate the fibrous barrier elsewhere besides at the A-V bundle. Under such conditions, the cardiac impulse can re-enter the atria from the ventricles and cause a serious cardiac arrhythmia.)

Distribution of the Purkinje Fibers in the Ventricles—The Left and Right Bundle Branches. After penetrating the fibrous tissue between the atrial and ventricular muscle, the distal portion of the A-V bundle passes downward in the ventricular septum for 5 to 15 millimeters toward the apex of the heart, as shown in Figures 10-1 and 10-3. Then the bundle divides into left and right bundle branches that lie beneath the endocardium on the two respective sides of the ventricular septum. Each branch spreads downward toward the apex of the ventricle, progressively dividing into smaller branches. These branches in turn course sidewise around each ventricular chamber and back toward the base of the heart. The ends of the Purkinje fibers penetrate about one third of the way into the muscle mass and finally become continuous with the cardiac muscle fibers.

From the time the cardiac impulse enters the bundle branches in the ventricular septum until it reaches the terminations of the Purkinje fibers, the total elapsed time averages only 0.03 second. Therefore, once the cardiac impulse enters the ventricular Purkinje conductive system, it spreads almost immediately to the entire ventricular muscle mass.

Transmission of the Cardiac Impulse in the Ventricular Muscle

Once the impulse reaches the ends of the Purkinje fibers, it is transmitted through the ventricular muscle mass by the ventricular muscle fibers themselves. The velocity of transmission is now only 0.3 to 0.5 m/sec, one sixth that in the Purkinje fibers.

The cardiac muscle wraps around the heart in a double spiral, with fibrous septa between the spiraling layers; therefore, the cardiac impulse does not necessarily travel directly outward toward the surface of the heart but instead angulates toward the surface along the directions of the spirals. Because of this, transmission from the endocardial surface to the epicardial surface of the ventricle requires as much as another 0.03 second, approximately equal to the time required for transmission through the entire ventricular portion of the Purkinje system. Thus, the total time for transmission of the cardiac impulse from the initial bundle branches to the last of the ventricular muscle fibers in the normal heart is about 0.06 second.

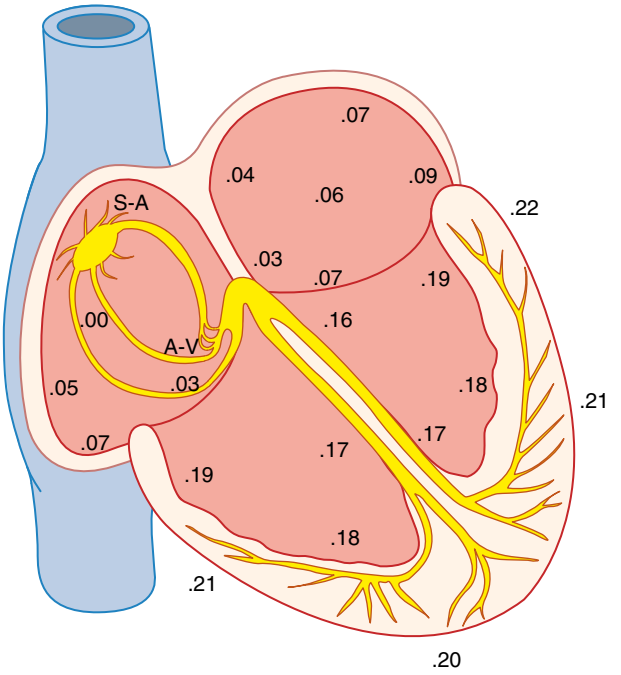


Figure 10-4 Transmission of the cardiac impulse through the heart, showing the time of appearance (in fractions of a second after initial appearance at the sinoatrial node) in different parts of the heart.

Summary of the Spread of the Cardiac Impulse Through the Heart

Figure 10-4 shows in summary form the transmission of the cardiac impulse through the human heart. The numbers on the figure represent the intervals of time, in fractions of a second, that lapse between the origin of the cardiac impulse in the sinus node and its appearance at each respective point in the heart. Note that the impulse spreads at moderate velocity through the atria but is delayed more than 0.1 second in the A-V nodal region before appearing in the ventricular septal A-V bundle. Once it has entered this bundle, it spreads very rapidly through the Purkinje fibers to the entire endocardial surfaces of the ventricles. Then the impulse once again spreads slightly less rapidly through the ventricular muscle to the epicardial surfaces.

It is important that the student learn in detail the course of the cardiac impulse through the heart and the precise times of its appearance in each separate part of the heart, because a thorough quantitative knowledge of this process is essential to the understanding of electrocardiography, which is discussed in Chapters 11 through 13.

Control of Excitation and Conduction in the Heart

Sinus Node as the Pacemaker of the Heart

In the discussion thus far of the genesis and transmission of the cardiac impulse through the heart, we have noted that the impulse normally arises in the sinus node. In some

abnormal conditions, this is not the case. Other parts of the heart can also exhibit intrinsic rhythmical excitation in the same way that the sinus nodal fibers do; this is particularly true of the A-V nodal and Purkinje fibers.

The A-V nodal fibers, when not stimulated from some outside source, discharge at an intrinsic rhythmical rate of 40 to 60 times per minute, and the Purkinje fibers discharge at a rate somewhere between 15 and 40 times per minute. These rates are in contrast to the normal rate of the sinus node of 70 to 80 times per minute.

Why then does the sinus node rather than the A-V node or the Purkinje fibers control the heart's rhythmicity? The answer derives from the fact that the discharge rate of the sinus node is considerably faster than the natural self-excitatory discharge rate of either the A-V node or the Purkinje fibers. Each time the sinus node discharges, its impulse is conducted into both the A-V node and the Purkinje fibers, also discharging their excitable membranes. But the sinus node discharges again before either the A-V node or the Purkinje fibers can reach their own thresholds for self-excitation. Therefore, the new impulse from the sinus node discharges both the A-V node and the Purkinje fibers before self-excitation can occur in either of these.

Thus, the sinus node controls the beat of the heart because its rate of rhythmical discharge is faster than that of any other part of the heart. Therefore, the sinus node is virtually always the pacemaker of the normal heart.

Abnormal Pacemakers—"Ectopic" Pacemaker. Occasionally some other part of the heart develops a rhythmical discharge rate that is more rapid than that of the sinus node. For instance, this sometimes occurs in the A-V node or in the Purkinje fibers when one of these becomes abnormal. In either case, the pacemaker of the heart shifts from the sinus node to the A-V node or to the excited Purkinje fibers. Under rarer conditions, a place in the atrial or ventricular muscle develops excessive excitability and becomes the pacemaker.

A pacemaker elsewhere than the sinus node is called an "*ectopic*" *pacemaker*. An ectopic pacemaker causes an abnormal sequence of contraction of the different parts of the heart and can cause significant debility of heart pumping.

Another cause of shift of the pacemaker is blockage of transmission of the cardiac impulse from the sinus node to the other parts of the heart. The new pacemaker then occurs most frequently at the A-V node or in the penetrating portion of the A-V bundle on the way to the ventricles.

When A-V block occurs—that is, when the cardiac impulse fails to pass from the atria into the ventricles through the A-V nodal and bundle system—the atria continue to beat at the normal rate of rhythm of the sinus node, while a new pacemaker usually develops in the Purkinje system of the ventricles and drives the ventricular muscle at a new rate somewhere between 15 and 40 beats per minute. After sudden A-V bundle block, the Purkinje system does not begin to emit its intrinsic rhythmical impulses until 5 to 20 seconds later because, before the blockage, the Purkinje fibers had been "overdriven" by the rapid sinus impulses and, consequently, are in a suppressed state. During these 5 to 20 seconds, the ventricles fail to pump blood, and the person faints after the first 4 to 5 seconds because of lack of blood flow to the brain. This delayed pickup of the heartbeat is called *Stokes-Adams syndrome*. If the delay period is too long, it can lead to death.

Role of the Purkinje System in Causing Synchronous Contraction of the Ventricular Muscle

It is clear from our description of the Purkinje system that normally the cardiac impulse arrives at almost all portions of the ventricles within a narrow span of time, exciting the first ventricular muscle fiber only 0.03 to 0.06 second ahead of excitation of the last ventricular muscle fiber. This causes all portions of the ventricular muscle in both ventricles to begin contracting at almost the same time and then to continue contracting for about another 0.3 second.

Effective pumping by the two ventricular chambers requires this synchronous type of contraction. If the cardiac impulse should travel through the ventricles slowly, much of the ventricular mass would contract before contraction of the remainder, in which case the overall pumping effect would be greatly depressed. Indeed, in some types of cardiac debilities, several of which are discussed in Chapters 12 and 13, slow transmission does occur, and the pumping effectiveness of the ventricles is decreased as much as 20 to 30 percent.

Control of Heart Rhythmicity and Impulse Conduction by the Cardiac Nerves: Sympathetic and Parasympathetic Nerves

The heart is supplied with both sympathetic and parasympathetic nerves, as shown in Figure 9-10 of Chapter 9. The parasympathetic nerves (the vagi) are distributed mainly to the S-A and A-V nodes, to a lesser extent to the muscle of the two atria, and very little directly to the ventricular muscle. The sympathetic nerves, conversely, are distributed to all parts of the heart, with strong representation to the ventricular muscle, as well as to all the other areas.

Parasympathetic (Vagal) Stimulation Can Slow or Even Block Cardiac Rhythm and Conduction—"Ventricular Escape." Stimulation of the parasympathetic nerves to the heart (the vagi) causes the hormone *acetylcholine* to be released at the vagal endings. This hormone has two major effects on the heart. First, it decreases the rate of rhythm of the sinus node, and second, it decreases the excitability of the A-V junctional fibers between the atrial musculature and the A-V node, thereby slowing transmission of the cardiac impulse into the ventricles.

Weak to moderate vagal stimulation slows the rate of heart pumping, often to as little as one-half normal.

And strong stimulation of the vagi can stop completely the rhythmical excitation by the sinus node or block completely transmission of the cardiac impulse from the atria into the ventricles through the A-V mode. In either case, rhythmical excitatory signals are no longer transmitted into the ventricles. The ventricles stop beating for 5 to 20 seconds, but then some small area in the Purkinje fibers, usually in the ventricular septal portion of the A-V bundle, develops a rhythm of its own and causes ventricular contraction at a rate of 15 to 40 beats per minute. This phenomenon is called *ventricular escape.*

Mechanism of the Vagal Effects. The acetylcholine released at the vagal nerve endings greatly increases the permeability of the fiber membranes to potassium ions, which allows rapid leakage of potassium out of the conductive fibers. This causes increased negativity inside the fibers, an effect called *hyperpolarization,* which makes this excitable tissue much less excitable, as explained in Chapter 5.

In the sinus node, the state of hyperpolarization decreases the "resting" membrane potential of the sinus nodal fibers to a level considerably more negative than usual, to –65 to –75 millivolts rather than the normal level of –55 to –60 millivolts. Therefore, the initial rise of the sinus nodal membrane potential caused by inward sodium and calcium leakage requires much longer to reach the threshold potential for excitation. This greatly slows the rate of rhythmicity of these nodal fibers. If the vagal stimulation is strong enough, it is possible to stop entirely the rhythmical self-excitation of this node.

In the A-V node, a state of hyperpolarization caused by vagal stimulation makes it difficult for the small atrial fibers entering the node to generate enough electricity to excite the nodal fibers. Therefore, the safety factor for transmission of the cardiac impulse through the transitional fibers into the A-V nodal fibers decreases. A moderate decrease simply delays conduction of the impulse, but a large decrease blocks conduction entirely.

Effect of Sympathetic Stimulation on Cardiac Rhythm and Conduction. Sympathetic stimulation causes essentially the opposite effects on the heart to those caused by vagal stimulation, as follows: First, it increases the rate of sinus nodal discharge. Second, it increases the rate of conduction, as well as the level of excitability in all portions of the heart. Third, it increases greatly the force of contraction of all the cardiac musculature, both atrial and ventricular, as discussed in Chapter 9.

In short, sympathetic stimulation increases the overall activity of the heart. Maximal stimulation can almost triple the frequency of heartbeat and can increase the strength of heart contraction as much as twofold.

Mechanism of the Sympathetic Effect. Stimulation of the sympathetic nerves releases the hormone *norepinephrine* at the sympathetic nerve endings. Norepinephrine in turn stimulates *beta-1 adrenergic receptors*, which mediate the effects on heart rate. The precise mechanism by which beta-1 adrenergic stimulation acts on cardiac muscle fibers is somewhat unclear, but the belief is that it increases the permeability of the fiber membrane to sodium and calcium ions. In the sinus node, an increase of sodium-calcium permeability causes a more positive resting potential and also causes increased rate of upward drift of the diastolic membrane potential toward the threshold level for self-excitation, thus accelerating self-excitation and, therefore, increasing the heart rate.

In the A-V node and A-V bundles, increased sodium-calcium permeability makes it easier for the action potential to excite each succeeding portion of the conducting fiber bundles, thereby decreasing the conduction time from the atria to the ventricles.

The increase in permeability to calcium ions is at least partially responsible for the increase in contractile strength of the cardiac muscle under the influence of sympathetic stimulation, because calcium ions play a powerful role in exciting the contractile process of the myofibrils.

Bibliography

Barbuti A, DiFrancesco D: Control of cardiac rate by "funny" channels in health and disease, ***Ann N Y Acad Sci*** 1123:213, 2008.

Baruscotti M, Robinson RB: Electrophysiology and pacemaker function of the developing sinoatrial node, ***Am J Physiol Heart Circ Physiol*** 293:H2613, 2007.

Cheng H, Lederer WJ: Calcium sparks, ***Physiol Rev*** 88:1491, 2008.

Chien KR, Domian IJ, Parker KK: Cardiogenesis and the complex biology of regenerative cardiovascular medicine, ***Science*** 322:1494, 2008.

Dobrzynski H, Boyett MR, Anderson RH: New insights into pacemaker activity: promoting understanding of sick sinus syndrome, ***Circulation*** 115:1921, 2007.

James TN: Structure and function of the sinus node, AV node and His bundle of the human heart: part I—structure, ***Prog Cardiovasc Dis*** 45:235, 2002.

James TN: Structure and function of the sinus node, AV node and His bundle of the human heart: part II—function, ***Prog Cardiovasc Dis*** 45:327, 2003.

Kléber AG, Rudy Y: Basic mechanisms of cardiac impulse propagation and associated arrhythmias, ***Physiol Rev*** 84:431, 2004.

Lakatta EG, Vinogradova TM, Maltsev VA: The missing link in the mystery of normal automaticity of cardiac pacemaker cells, ***Ann N Y Acad Sci*** 1123:41, 2008.

Leclercq C, Hare JM: Ventricular resynchronization: current state of the art, ***Circulation*** 109:296, 2004.

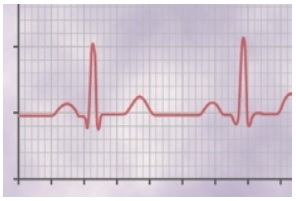
Mangoni ME, Nargeot J: Genesis and regulation of the heart automaticity, ***Physiol Rev*** 88:919, 2008.

Mazgalev TN, Ho SY, Anderson RH: Anatomic-electrophysiological correlations concerning the pathways for atrioventricular conduction, ***Circulation*** 103:2660, 2001.

Schram G, Pourrier M, Melnyk P, et al: Differential distribution of cardiac ion channel expression as a basis for regional specialization in electrical function, ***Circ Res*** 90:939, 2002.

Yasuma F, Hayano J: Respiratory sinus arrhythmia: why does the heartbeat synchronize with respiratory rhythm? ***Chest*** 125:683, 2004.

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 necessary.

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

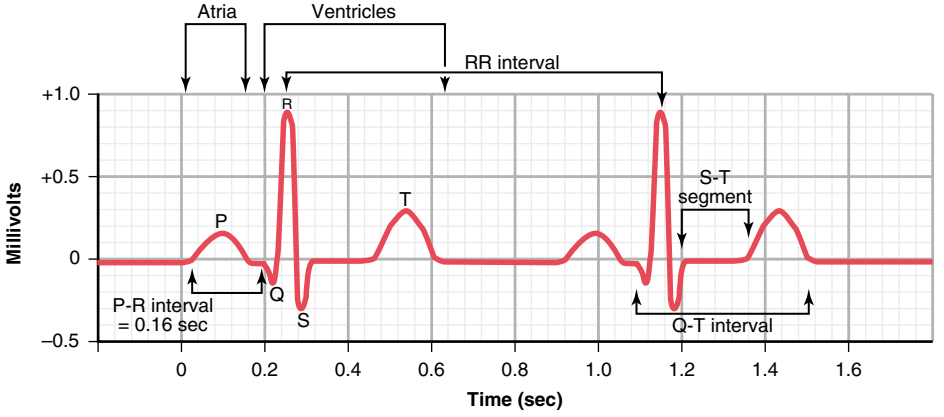


Figure 11-1 Normal electrocardiogram.

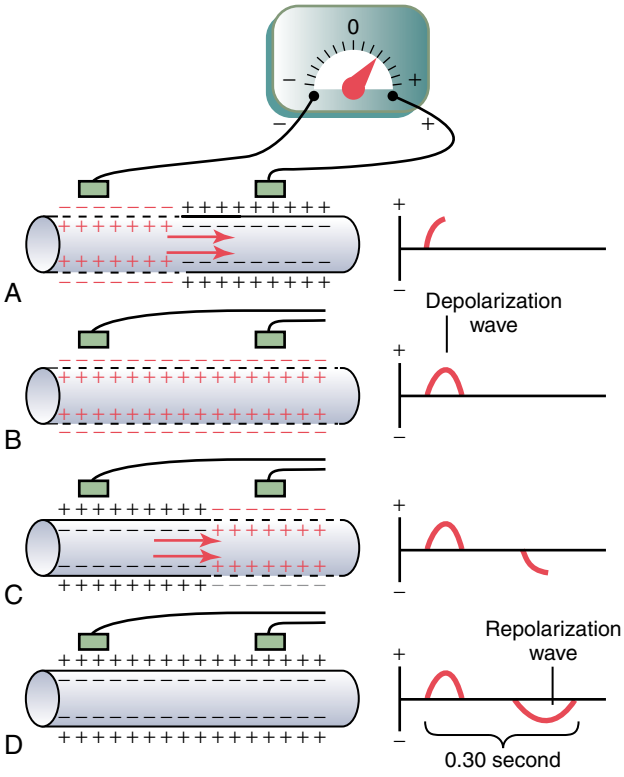


Figure 11-2 Recording the depolarization wave (*A* and *B*) and the repolarization wave (*C* and *D*) from a cardiac muscle fiber.

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 11-2*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

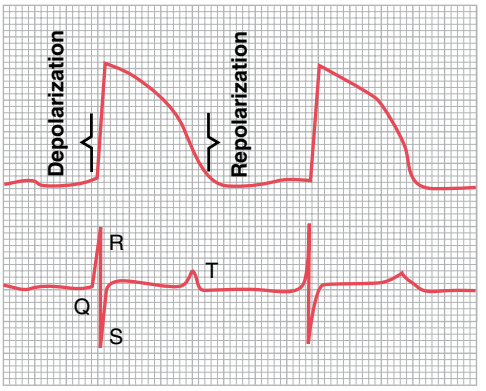


Figure 11-3 *Above,* Monophasic action potential from a ventricular muscle fiber during normal cardiac function, showing rapid depolarization and then repolarization occurring slowly during the plateau stage but rapidly toward the end. *Below,* Electrocardiogram recorded simultaneously.

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 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. A typical electrocardiogram is run at a paper speed of 25 millimeters per second, although faster speeds are sometimes used. Therefore, each 25 millimeters in the horizontal direction is 1 second, and each 5-millimeter segment, indicated by the dark vertical lines, represents 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 millivolts 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 millivolts; and that of the T wave is between 0.2 and 0.3 millivolts.

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.

Recorders for Electrocardiographs

Many modern clinical electrocardiographs use computer-based systems and electronic display, whereas 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. 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

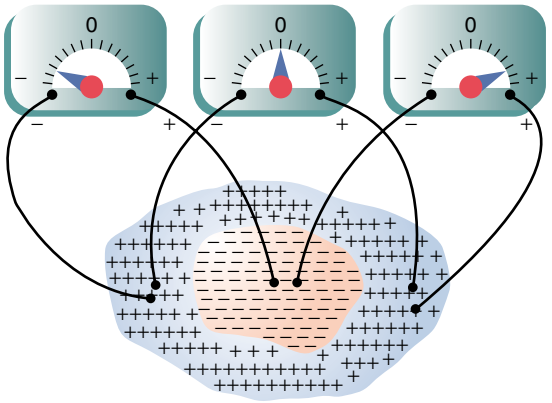


Figure 11-4 Instantaneous potentials develop on the surface of a cardiac muscle mass that has been depolarized in its center.

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 remainder, electrical current flows from the depolarized area to the polarized area in large circuitous routes, as noted in the figure.

It should be recalled from the discussion of the Purkinje system in Chapter 10 that the cardiac impulse first arrives in the ventricles in the septum and shortly thereafter spreads to the inside surfaces of the remainder of the ventricles, as shown by the red areas and the negative signs in Figure 11-5. This provides electronegativity on the insides of the ventricles and electropositivity on the outer walls of the ventricles, with electrical current flowing through the fluids surrounding the ventricles along elliptical paths, as demonstrated by the curving arrows in the figure. If one algebraically averages all the lines of current flow (the elliptical lines), one finds that *the average current flow occurs with negativity toward the base of the heart and with positivity toward the apex.*

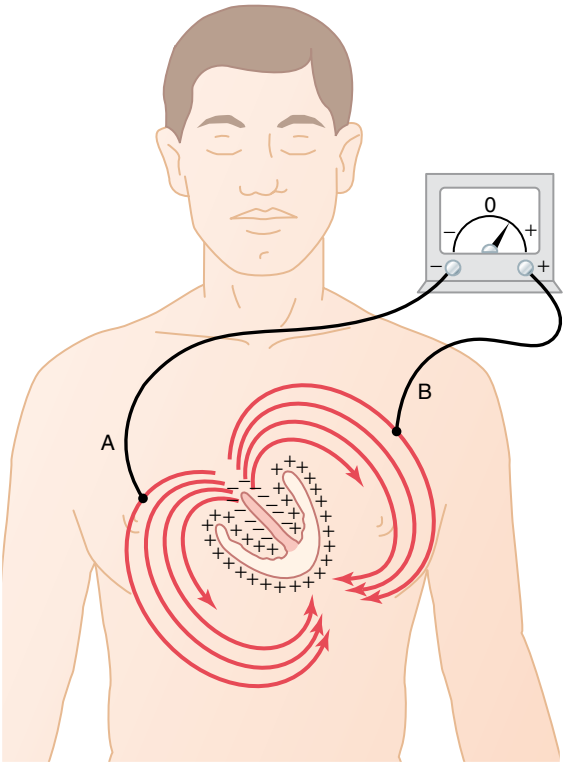


Figure 11-5 Flow of current in the chest around partially depolarized ventricles.

During most of the remainder of the depolarization process, current also continues to flow in this same direction, while depolarization spreads from the endocardial surface outward through the ventricular muscle mass. Then, immediately before depolarization has completed its course through the ventricles, the average direction of current flow reverses for about 0.01 second, flowing from the ventricular apex toward the base, because the last part of the heart to become depolarized is the outer walls of the ventricles near the base of the heart.

Thus, in normal heart ventricles, current flows from negative to positive primarily in the direction from the base of the heart toward the apex during almost the entire cycle of depolarization, except at the very end. And if a meter is connected to electrodes on the surface of the body as shown in Figure 11-5, the electrode nearer the base will be negative, whereas the electrode nearer the apex will be positive, and the recording meter will show positive recording in the electrocardiogram.

Electrocardiographic Leads

Three Bipolar Limb Leads

Figure 11-6 shows electrical connections between the patient's limbs and the electrocardiograph for recording electrocardiograms from the so-called *standard bipolar*

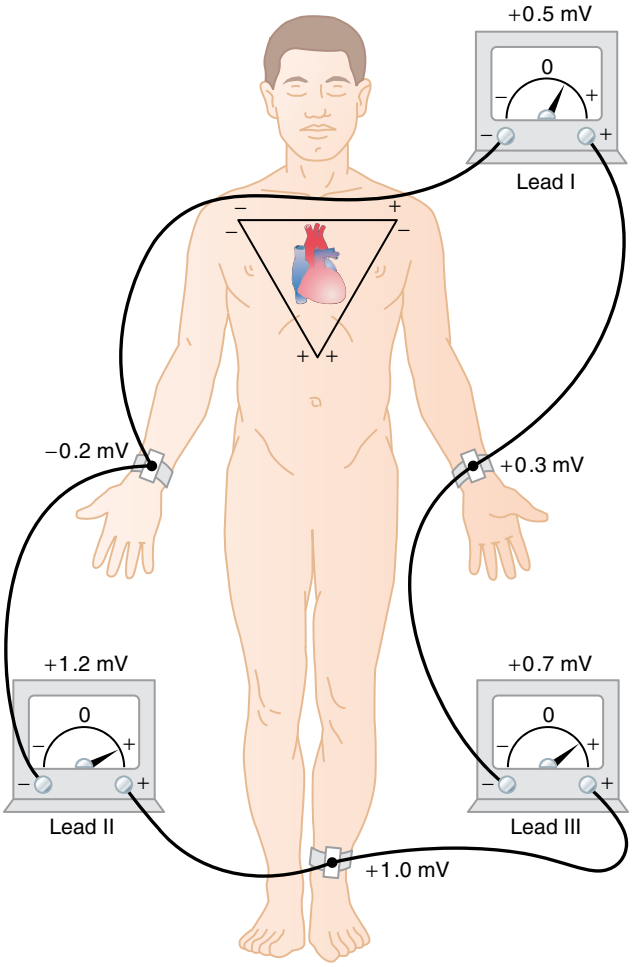


Figure 11-6 Conventional arrangement of electrodes for recording the standard electrocardiographic leads. Einthoven's triangle is superimposed on the chest.

limb leads. The term "bipolar" means that the electrocardiogram is recorded from two electrodes located on different sides of the heart—in this case, on the limbs. Thus, a "lead" is not a single wire connecting from the body but a combination of two wires and their electrodes to make a complete circuit between the body and the electrocardiograph. The electrocardiograph in each instance is represented by an electrical meter in the diagram, although the actual electrocardiograph is a high-speed recording meter with a moving paper.

Lead I. In recording limb lead I, the negative terminal of the electrocardiograph is connected to the right arm and the positive terminal to the left arm. Therefore, when the point where the right arm connects to the chest is electronegative with respect to the point where the left arm connects, the electrocardiograph records positively, that is, above the zero voltage line in the electrocardiogram. When the opposite is true, the electrocardiograph records below the line.

Lead II. To record limb lead II, the negative terminal of the electrocardiograph is connected to the right arm and the positive terminal to the left leg. Therefore, when the right arm is negative with respect to the left leg, the electrocardiograph records positively.

Lead III. To record limb lead III, the negative terminal of the electrocardiograph is connected to the left arm and the positive terminal to the left leg. This means that the electrocardiograph records positively when the left arm is negative with respect to the left leg.

Einthoven's Triangle. In Figure 11-6, the triangle, called *Einthoven's triangle,* is drawn around the area of the heart. This illustrates that the two arms and the left leg form apices of a triangle surrounding the heart. The two apices at the upper part of the triangle represent the points at which the two arms connect electrically with the fluids around the heart, and the lower apex is the point at which the left leg connects with the fluids.

Einthoven's Law. Einthoven's law states that if the electrical potentials of any two of the three bipolar limb electrocardiographic leads are known at any given instant, the third one can be determined mathematically by simply summing the first two. Note, however, that the positive and negative signs of the different leads must be observed when making this summation.

For instance, let us assume that momentarily, as noted in Figure 11-6, the right arm is −0.2 millivolts (negative) with respect to the average potential in the body, the left arm is +0.3 millivolts (positive), and the left leg is +1.0 millivolts (positive). Observing the meters in the figure, one can see that lead I records a positive potential of +0.5 millivolts because this is the difference between the −0.2 millivolts on the right arm and the +0.3 millivolts on the left arm. Similarly, lead III records a positive potential of +0.7 millivolts, and lead II records a positive potential of +1.2 millivolts because these are the instantaneous potential differences between the respective pairs of limbs.

Now, note that the sum of the voltages in leads I and III equals the voltage in lead II; that is, 0.5 plus 0.7 equals 1.2. Mathematically, this principle, called Einthoven's law, holds true at any given instant while the three "standard" bipolar electrocardiograms are being recorded.

Normal Electrocardiograms Recorded from the Three Standard Bipolar Limb Leads. Figure 11-7 shows recordings of the electrocardiograms in leads I, II, and III. It is obvious that the electrocardiograms in these three leads are similar to one another because they all record positive P waves and positive T waves, and the major portion of the QRS complex is also positive in each electrocardiogram.

On analysis of the three electrocardiograms, it can be shown, with careful measurements and proper observance of polarities, that at any given instant the sum of the potentials in leads I and III equals the potential in lead II, thus illustrating the validity of Einthoven's law.

Because the recordings from all the bipolar limb leads are similar to one another, it does not matter greatly which lead is recorded when one wants to diagnose different cardiac arrhythmias, because diagnosis of arrhythmias depends mainly on the time relations between the different waves of the cardiac cycle. But when one wants to diagnose damage in the ventricular or atrial muscle or in the Purkinje conducting system, it matters greatly which

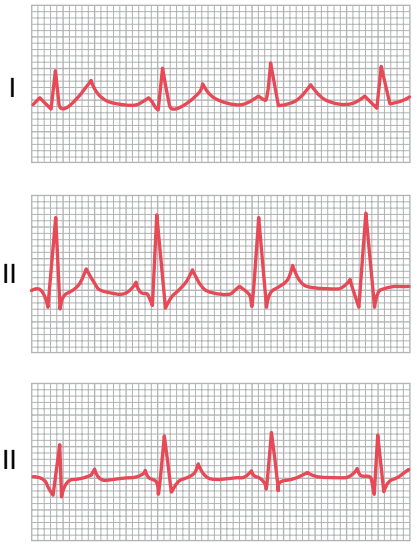


Figure 11-7 Normal electrocardiograms recorded from the three standard electrocardiographic leads.

leads are recorded, because abnormalities of cardiac muscle contraction or cardiac impulse conduction do change the patterns of the electrocardiograms markedly in some leads yet may not affect other leads. Electrocardiographic interpretation of these two types of conditions—cardiac myopathies and cardiac arrhythmias—is discussed separately in Chapters 12 and 13.

Chest Leads (Precordial Leads)

Often electrocardiograms are recorded with one electrode placed on the anterior surface of the chest directly over the heart at one of the points shown in Figure 11-8. This electrode is connected to the positive terminal of the electrocardiograph, and the negative electrode, called the *indifferent electrode,* is connected through equal electrical resistances to the right arm, left arm, and left leg all at the same time, as also shown in the figure. Usually six standard chest leads are recorded, one at a time, from the anterior chest wall, the chest electrode being placed sequentially at the six points shown in the diagram. The different recordings are known as leads V1, V2, V3, V4, V5, and V6.

Figure 11-9 illustrates the electrocardiograms of the healthy heart as recorded from these six standard chest leads. Because the heart surfaces are close to the chest wall, each chest lead records mainly the electrical potential of the cardiac musculature immediately beneath the electrode. Therefore, relatively minute abnormalities in the ventricles, particularly in the anterior ventricular wall, can cause marked changes in the electrocardiograms recorded from individual chest leads.

In leads V1 and V2, the QRS recordings of the normal heart are mainly negative because, as shown in Figure 11-8, the chest electrode in these leads is nearer to the base of the heart than to the apex, and the base of the heart is the direction of electronegativity during most of

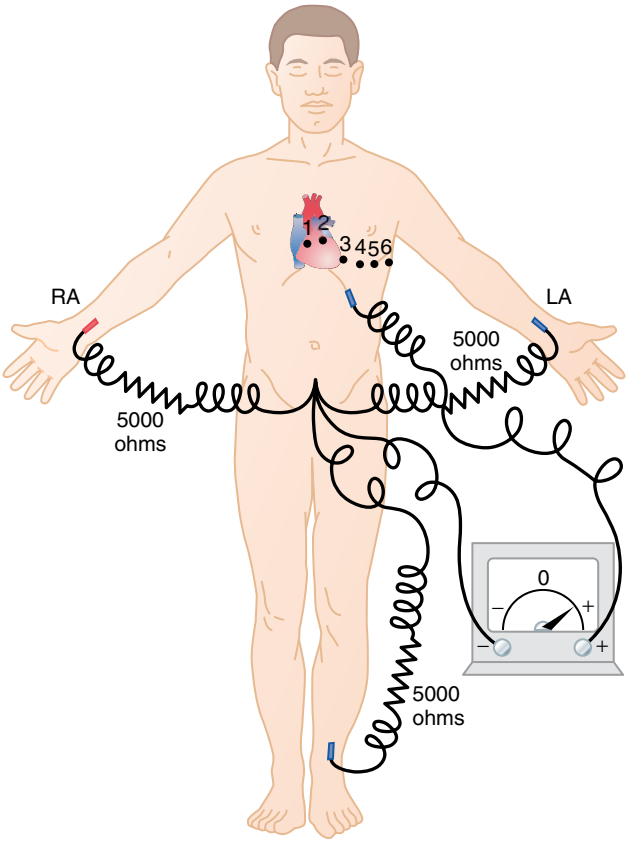


Figure 11-8 Connections of the body with the electrocardiograph for recording chest leads. LA, left arm; RA, right arm.

the ventricular depolarization process. Conversely, the QRS complexes in leads V4, V5, and V6 are mainly positive because the chest electrode in these leads is nearer the heart apex, which is the direction of electropositivity during most of depolarization.

Augmented Unipolar Limb Leads

Another system of leads in wide use is the *augmented unipolar limb lead*. In this type of recording, two of the limbs are connected through electrical resistances to the negative terminal of the electrocardiograph, and the third limb is connected to the positive terminal. When the positive terminal is on the right arm, the lead is known as the aVR lead; when on the left arm, the aVL lead; and when on the left leg, the aVF lead.

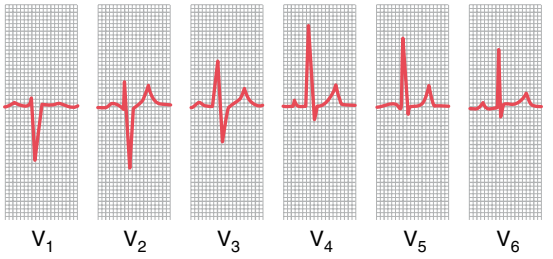


Figure 11-9 Normal electrocardiograms recorded from the six standard chest leads.

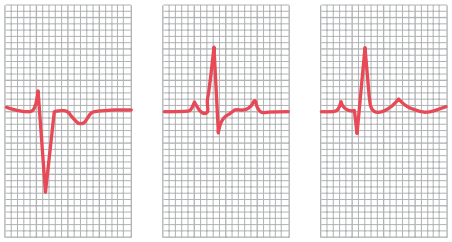


Figure 11-10 Normal electrocardiograms recorded from the three augmented unipolar limb leads.

Normal recordings of the augmented unipolar limb leads are shown in Figure 11-10. They are all similar to the standard limb lead recordings, except that the recording from the aVR lead is inverted. (Why does this inversion occur? Study the polarity connections to the electrocardiograph to determine this.)

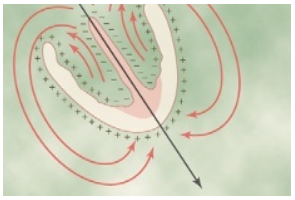
Bibliography

See bibliography for Chapter 13.

This page intentionally left blank

CHAPTER 12

Electrocardiographic Interpretation of Cardiac Muscle and Coronary Blood Flow Abnormalities: Vectorial Analysis



From the discussion in Chapter 10 of impulse transmission through the heart, it is obvious that any change in the pattern of this transmission can cause abnormal electrical potentials around the heart and, consequently, alter the shapes of the waves in the electrocardiogram. For this reason, most serious abnormalities of the heart muscle can be diagnosed by analyzing the contours of the waves in the different electrocardiographic leads.

Principles of Vectorial Analysis of Electrocardiograms

Use of Vectors to Represent Electrical Potentials

Before it is possible to understand how cardiac abnormalities affect the contours of the electrocardiogram, one must first become thoroughly familiar with the concept of *vectors* and *vectorial analysis* as applied to electrical potentials in and around the heart.

Several times in Chapter 11 it was pointed out that heart current flows in a particular direction in the heart at a given instant during the cardiac cycle. A vector is an arrow that points in the direction of the electrical potential generated by the current flow, *with the arrowhead in the positive direction.* Also, by convention, the length of the arrow is drawn *proportional to the voltage of the potential.*

"Resultant" Vector in the Heart at Any Given Instant. Figure 12-1 shows, by the shaded area and the negative signs, depolarization of the ventricular septum and parts of the apical endocardial walls of the two ventricles. At this instant of heart excitation, electrical current flows between the depolarized areas inside the heart and the nondepolarized areas on the outside of the heart, as indicated by the long elliptical arrows. Some current also flows inside the heart chambers directly from the depolarized areas toward the still polarized areas. Overall, considerably more current flows downward from the base of the ventricles toward the apex than in the upward direction. Therefore, the summated vector of the generated potential at this particular instant, called the *instantaneous mean vector,* is represented by the long *black* arrow drawn through the center of the ventricles in a direction from base toward apex. Furthermore, because the summated current is considerable in quantity, the potential is large and the vector is long.

Direction of a Vector Is Denoted in Terms of Degrees

When a vector is exactly horizontal and directed toward the person's left side, the vector is said to extend in the direction of 0 degrees, as shown in Figure 12-2. From this zero reference point, the scale of vectors rotates clockwise: when the vector extends from above and straight downward, it has a direction of +90 degrees; when it extends from the person's left to right, it has a direction of +180 degrees; and when it extends straight upward, it has a direction of –90 (or +270) degrees.

In a normal heart, the average direction of the vector during spread of the depolarization wave through the ventricles, called the *mean QRS vector,* is about +59 degrees, which is shown by vector *A* drawn through the center of Figure 12-2 in the +59-degree direction. This means that during most of the depolarization wave, the apex of the heart remains positive with respect to the base of the heart, as discussed later in the chapter.

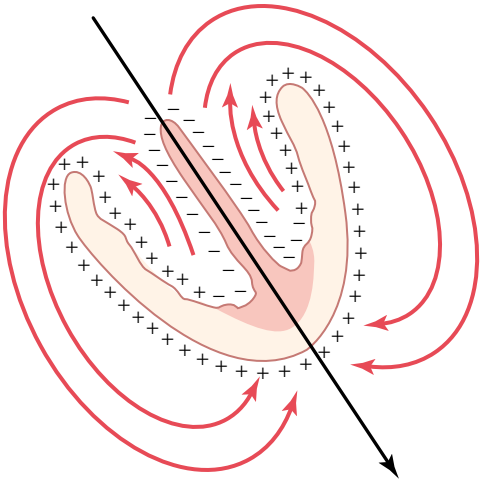


Figure 12-1 Mean vector through the partially depolarized ventricles.

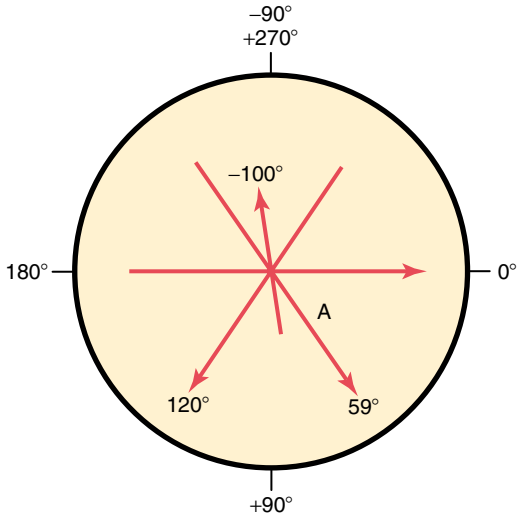


Figure 12-2 Vectors drawn to represent potentials for several different hearts, and the "axis" of the potential (expressed in degrees) for each heart.

Axis for Each Standard Bipolar Lead and Each Unipolar Limb Lead

In Chapter 11, the three standard bipolar and the three unipolar limb leads are described. Each lead is actually a pair of electrodes connected to the body on opposite sides of the heart, and the direction from negative electrode to positive electrode is called the "axis" of the lead. Lead I is recorded from two electrodes placed respectively on the two arms. Because the electrodes lie exactly in the horizontal direction, with the positive electrode to the left, the axis of lead I is 0 degrees.

In recording lead II, electrodes are placed on the right arm and left leg. The right arm connects to the torso in the upper right-hand corner and the left leg connects in the lower left-hand corner. Therefore, the direction of this lead is about +60 degrees.

By similar analysis, it can be seen that lead III has an axis of about +120 degrees; lead aVR, +210 degrees; aVF, +90 degrees; and aVL –30 degrees. The directions of the axes of all these leads are shown in Figure 12-3, which is known as the *hexagonal reference system.* The polarities of the electrodes are shown by the plus and minus signs in the figure. *The reader must learn these axes and their polarities, particularly for the bipolar limb leads I, II, and III, to understand the remainder of this chapter.*

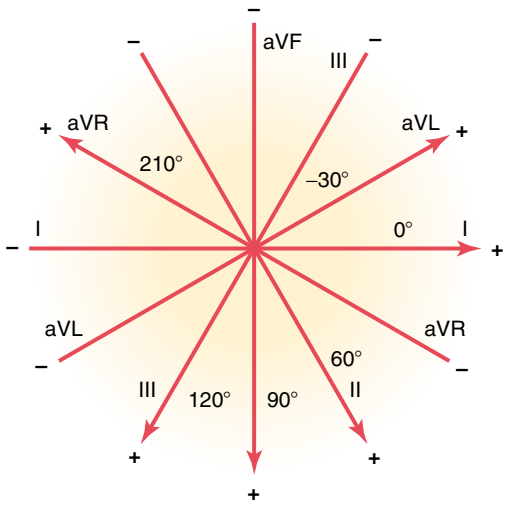


Figure 12-3 Axes of the three bipolar and three unipolar leads.

Vectorial Analysis of Potentials Recorded in Different Leads

Now that we have discussed, first, the conventions for representing potentials across the heart by means of vectors and, second, the axes of the leads, it is possible to use these together to determine the instantaneous potential that will be recorded in the electrocardiogram of each lead for a given vector in the heart, as follows.

Figure 12-4 shows a partially depolarized heart; vector *A* represents the instantaneous mean direction of current flow in the ventricles. In this instance, the direction of the vector is +55 degrees, and the voltage of the potential, represented by the length of vector *A,* is 2 mv. In the diagram below the heart, vector *A* is shown again, and a line is drawn to represent the axis of lead I in the 0-degree direction. To determine how much of the voltage in vector *A* will be recorded in lead I, a line perpendicular to the axis of lead I is drawn from the tip of vector *A* to the lead I axis, and a so-called *projected vector (B)* is drawn along the lead I axis. The arrow of this projected vector points toward the positive end of the lead I axis, which means that the record momentarily being recorded in the electrocardiogram of lead I is positive. And the instantaneous recorded voltage will be equal to the length of *B* divided by the length of *A* times 2 millivolts, or about 1 millivolt.

Figure 12-5 shows another example of vectorial analysis. In this example, vector *A* represents the electrical potential and its axis at a given instant during ventricular depolarization in a heart in which the left side of the heart depolarizes more rapidly than the right. In this instance, the instantaneous vector has a direction of 100 degrees, and its voltage is again 2 millivolts. To determine the potential actually recorded in lead I, we draw a perpendicular line from the tip of vector *A* to the lead I axis and find projected vector *B.* Vector *B* is very short and this time in the negative direction, indicating that at this

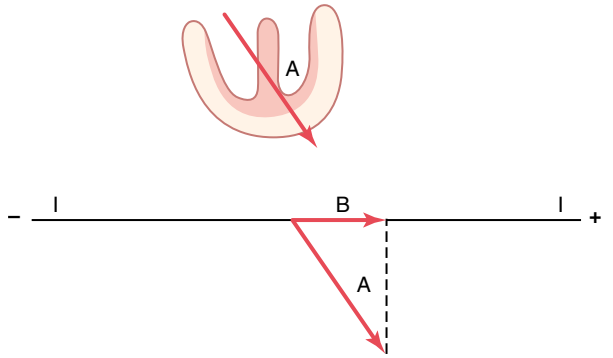


Figure 12-4 Determination of a projected vector B along the axis of lead I when vector A represents the instantaneous potential in the ventricles.

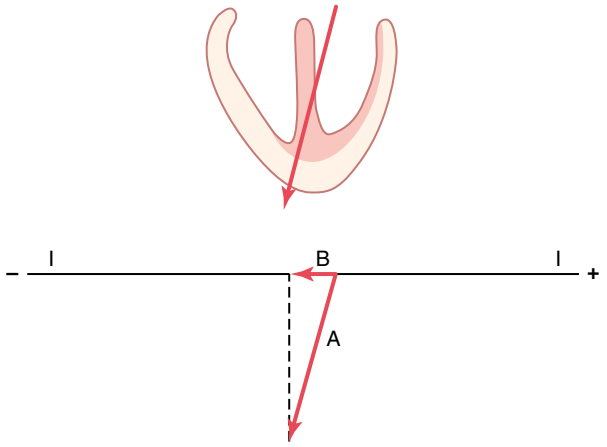


Figure 12-5 Determination of the projected vector B along the axis of lead I when vector A represents the instantaneous potential in the ventricles.

particular instant, the recording in lead I will be negative (below the zero line in the electrocardiogram), and the voltage recorded will be slight, about –0.3 millivolts. This figure demonstrates that *when the vector in the heart is in a direction almost perpendicular to the axis of the lead, the voltage recorded in the electrocardiogram of this lead is very low.* Conversely, *when the heart vector has almost exactly the same axis as the lead axis, essentially the entire voltage of the vector will be recorded.*

Vectorial Analysis of Potentials in the Three Standard Bipolar Limb Leads. In Figure 12-6, vector *A* depicts the instantaneous electrical potential of a partially depolarized heart. To determine the potential recorded at this instant in the electrocardiogram for each one of the three standard bipolar limb leads, perpendicular lines (the dashed lines) are drawn from the tip of vector *A* to the three lines representing the axes of the three different standard leads, as shown in the figure. The projected vector *B* depicts the potential recorded at that instant in lead I, projected vector *C* depicts the potential in lead II, and projected vector *D* depicts the potential in lead III. In each of these, the record in the electrocardiogram is positive—that is, above the zero line—because the projected vectors point in the positive directions along the axes of all the leads. The potential in lead I (vector *B*) is about one-half that of the actual potential in the heart (vector *A*); in lead II (vector *C*), it is almost equal to that in the heart; and in lead III (vector *D*), it is about one-third that in the heart.

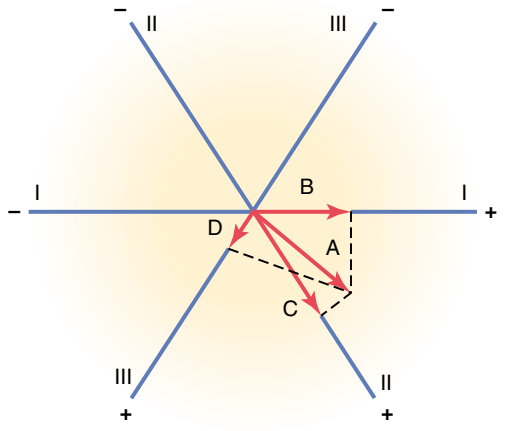


Figure 12-6 Determination of projected vectors in leads I, II, and III when vector A represents the instantaneous potential in the ventricles.

An identical analysis can be used to determine potentials recorded in augmented limb leads, except that the respective axes of the augmented leads (see Figure 12-3) are used in place of the standard bipolar limb lead axes used for Figure 12-6.

Vectorial Analysis of the Normal Electrocardiogram

Vectors That Occur at Successive Intervals during Depolarization of the Ventricles—the QRS Complex

When the cardiac impulse enters the ventricles through the atrioventricular bundle, the first part of the ventricles to become depolarized is the left endocardial surface of the septum. Then depolarization spreads rapidly to involve both endocardial surfaces of the septum, as demonstrated by the darker shaded portion of the ventricle in Figure 12-7*A*. Next, depolarization spreads along the endocardial surfaces of the remainder of the two ventricles, as shown in Figure 12-7*B* and *C*. Finally, it spreads through the ventricular muscle to the outside of the heart, as shown progressively in Figure 12-7*C, D,* and *E.*

At each stage in Figure 12-7, parts *A* to *E,* the instantaneous mean electrical potential of the ventricles is represented by a red vector superimposed on the ventricle in each figure. Each of these vectors is then analyzed by the method described in the preceding section to determine the voltages that will be recorded at each instant in each of the three standard electrocardiographic leads. To the right in each figure is shown progressive development of the electrocardiographic QRS complex. *Keep in mind that a positive vector in a lead will cause recording in the electrocardiogram above the zero line, whereas a negative vector will cause recording below the zero line.*

Before proceeding with further consideration of vectorial analysis, it is essential that this analysis of the successive normal vectors presented in Figure 12-7 be understood. Each of these analyses should be studied in detail by the procedure given here. A short summary of this sequence follows.

In Figure 12-7*A,* the ventricular muscle has just begun to be depolarized, representing an instant about 0.01 second after the onset of depolarization. At this time, the vector is short because only a small portion of the ventricles—the septum—is depolarized. Therefore, all electrocardiographic voltages are low, as recorded to the right of the ventricular muscle for each of the leads. The voltage in lead II is greater than the voltages in leads I and III because the heart vector extends mainly in the same direction as the axis of lead II.

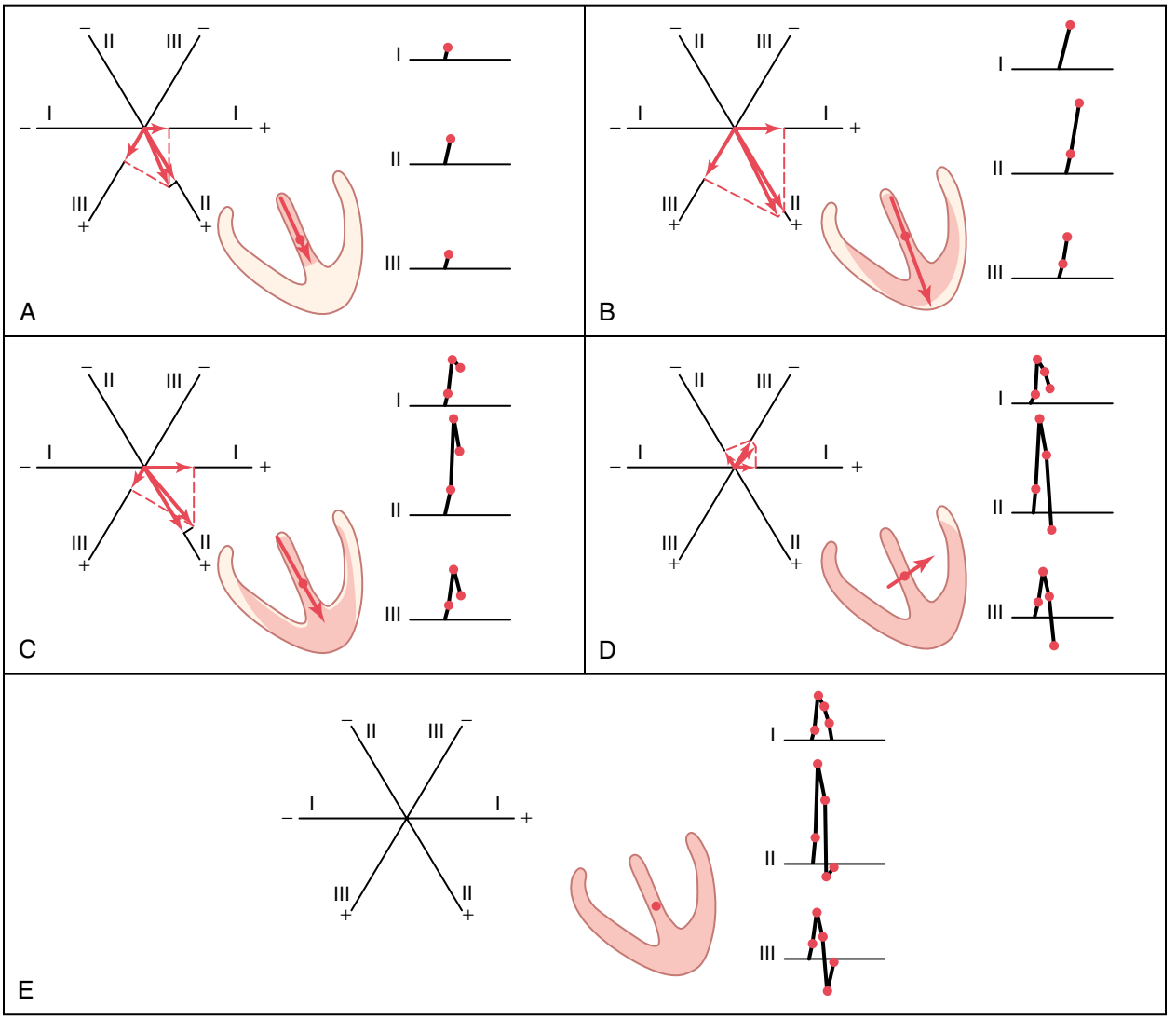


Figure 12-7 Shaded areas of the ventricles are depolarized (–); nonshaded areas are still polarized (+). The ventricular vectors and QRS complexes 0.01 second after onset of ventricular depolarization *(A)*; 0.02 second after onset of depolarization *(B)*; 0.035 second after onset of depolarization *(C)*; 0.05 second after onset of depolarization *(D)*; and after depolarization of the ventricles is complete, 0.06 second after onset *(E)*.

In Figure 12-7*B*, which represents about 0.02 second after onset of depolarization, the heart vector is long because much of the ventricular muscle mass has become depolarized. Therefore, the voltages in all electrocardiographic leads have increased.

In Figure 12-7*C*, about 0.035 second after onset of depolarization, the heart vector is becoming shorter and the recorded electrocardiographic voltages are lower because the outside of the heart apex is now electronegative, neutralizing much of the positivity on the other epicardial surfaces of the heart. Also, the axis of the vector is beginning to shift toward the left side of the chest because the left ventricle is slightly slower to depolarize than the right. Therefore, the ratio of the voltage in lead I to that in lead III is increasing.

In Figure 12-7*D*, about 0.05 second after onset of depolarization, the heart vector points toward the base of the left ventricle, and it is short because only a minute portion of the ventricular muscle is still polarized positive. Because of the direction of the vector at this time, the voltages recorded in leads II and III are both negative—that is, below the line—whereas the voltage of lead I is still positive.

In Figure 12-7*E*, about 0.06 second after onset of depolarization, the entire ventricular muscle mass is depolarized so that no current flows around the heart and no electrical potential is generated. The vector becomes zero, and the voltages in all leads become zero.

Thus, the QRS complexes are completed in the three standard bipolar limb leads.

Sometimes the QRS complex has a slight negative depression at its beginning in one or more of the leads, which is not shown in Figure 12-7; this depression is the Q wave. When it occurs, it is caused by initial depolarization of the left side of the septum before the right side, which creates a weak vector from left to right for a fraction of a second before the usual base-to-apex vector occurs. The major positive deflection shown in Figure 12-7 is the R wave, and the final negative deflection is the S wave.

Electrocardiogram during Repolarization—the T Wave

After the ventricular muscle has become depolarized, about 0.15 second later, repolarization begins and proceeds until complete at about 0.35 second. This repolarization causes the T wave in the electrocardiogram.

Because the septum and endocardial areas of the ventricular muscle depolarize first, it seems logical that these areas should repolarize first as well. However, this is not the usual case because the septum and other endocardial areas have a longer period of contraction than most of the external surfaces of the heart. Therefore, *the greatest portion of ventricular muscle mass to repolarize first is the entire outer surface of the ventricles, especially near the apex of the heart.* The endocardial areas, conversely, normally repolarize last. This sequence of repolarization is postulated to be caused by the high blood pressure inside the ventricles during contraction, which greatly reduces coronary blood flow to the endocardium, thereby slowing repolarization in the endocardial areas.

Because the outer apical surfaces of the ventricles repolarize before the inner surfaces, the positive end of the overall ventricular vector during repolarization is toward the apex of the heart. *As a result, the normal T wave in all three bipolar limb leads is positive, which is also the polarity of most of the normal QRS complex.*

In Figure 12-8, five stages of repolarization of the ventricles are denoted by progressive increase of the light tan areas—the repolarized areas. At each stage, the vector extends from the base of the heart toward the apex until it disappears in the last stage. At first, the vector is relatively small because the area of repolarization is small. Later, the vector becomes stronger because of greater degrees of repolarization. Finally, the vector becomes weaker again because the areas of depolarization still persisting become so slight that the total quantity of current flow decreases. These changes also demonstrate that the vector is greatest when about half the heart is in the polarized state and about half is depolarized.

The changes in the electrocardiograms of the three standard limb leads during repolarization are noted under each of the ventricles, depicting the progressive stages of repolarization. Thus, over about 0.15 second, the period of time required for the whole process to take place, the T wave of the electrocardiogram is generated.

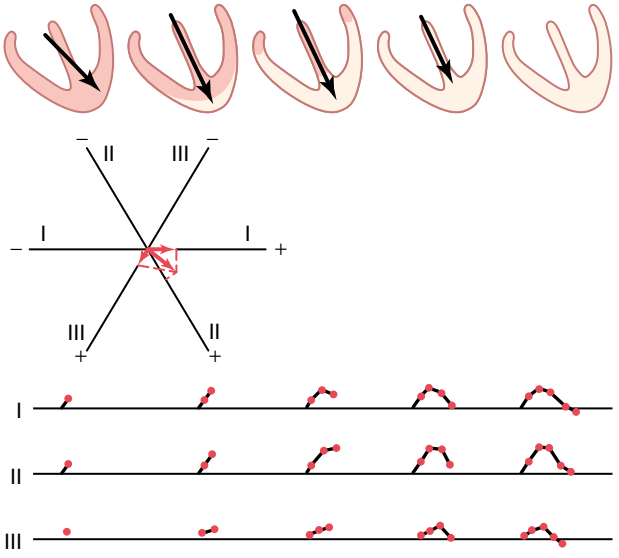


Figure 12-8 Generation of the T wave during repolarization of the ventricles, showing also vectorial analysis of the first stage of repolarization. The total time from the beginning of the T wave to its end is approximately 0.15 second.

Depolarization of the Atria—the P Wave

Depolarization of the atria begins in the sinus node and spreads in all directions over the atria. Therefore, the point of original electronegativity in the atria is about at the point of entry of the superior vena cava where the sinus node lies, and the direction of initial depolarization is denoted by the black vector in Figure 12-9. Furthermore, the vector remains generally in this direction throughout the process of normal atrial depolarization. Because this direction is generally in the positive directions of the axes of the three standard bipolar limb leads I, II, and III, the electrocardiograms recorded from the atria during depolarization are also usually positive in all three of these leads, as shown in Figure 12-9. This record of atrial depolarization is known as the atrial P wave.

Repolarization of the Atria—the Atrial T Wave. Spread of depolarization through the atrial muscle is *much slower than in the ventricles* because the atria have no Purkinje system for fast conduction of the depolarization signal. Therefore, the musculature around the sinus node becomes depolarized a long time before the musculature in distal parts of the atria. Because of this, *the area in the atria that also becomes repolarized first is the sinus nodal region, the area that had originally become depolarized first.* Thus, when repolarization begins, the region around the sinus node becomes positive with respect to the rest of the atria. Therefore, the atrial repolarization

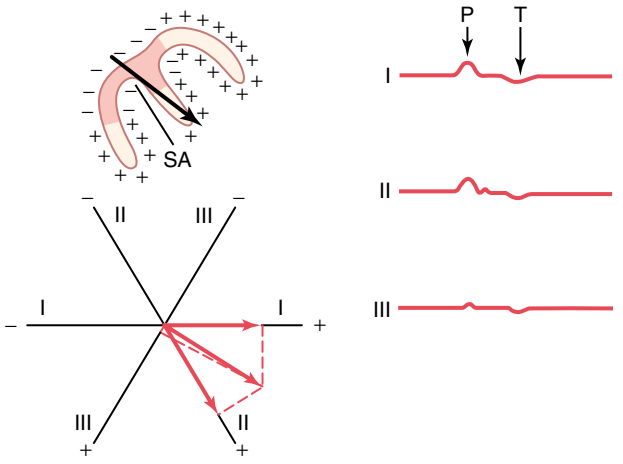


Figure 12-9 Depolarization of the atria and generation of the P wave, showing the maximum vector through the atria and the resultant vectors in the three standard leads. At the right are the atrial P and T waves. SA, sinoatrial node.

vector is *backward to the vector of depolarization.* (Note that this is opposite to the effect that occurs in the ventricles.) Therefore, as shown to the right in Figure 12-9, the so-called atrial T wave follows about 0.15 second after the atrial P wave, but this T wave is on the opposite side of the zero reference line from the P wave; that is, it is normally negative rather than positive in the three standard bipolar limb leads.

In the normal electrocardiogram, the *atrial* T wave appears at about the same time that the QRS complex of the ventricles appears. Therefore, it is almost always totally obscured by the large *ventricular* QRS complex, although in some very abnormal states, it does appear in the recorded electrocardiogram.

Vectorcardiogram

It has been noted in the discussion up to this point that the vector of current flow through the heart changes rapidly as the impulse spreads through the myocardium. It changes in two aspects: First, the vector increases and decreases in length because of increasing and decreasing voltage of the vector. Second, the vector changes direction because of changes in the average direction of the electrical potential from the heart. The so-called *vectorcardiogram* depicts these changes at different times during the cardiac cycle, as shown in Figure 12-10.

In the large vectorcardiogram of Figure 12-10, point 5 is the *zero reference point,* and this point is the negative end of all the successive vectors. While the heart muscle is polarized between heartbeats, the positive end of the vector remains at the zero point because there is no vectorial electrical potential. However, as soon as current begins to flow through the ventricles at the beginning of ventricular depolarization, the positive end of the vector leaves the zero reference point.

When the septum first becomes depolarized, the vector extends downward toward the apex of the ventricles, but it is relatively weak, thus generating the first portion of the ventricular vectorcardiogram, as shown by the positive end of vector 1. As more of the ventricular muscle becomes depolarized, the vector becomes stronger and stronger, usually swinging slightly to one side. Thus, vector 2 of Figure 12-10 represents the state of depolarization of the ventricles about 0.02 second after vector 1. After another 0.02 second, vector 3 represents the potential, and vector 4 occurs in another 0.01 second. Finally, the ventricles become totally depolarized, and the vector becomes zero once again, as shown at point 5.

The elliptical figure generated by the positive ends of the vectors is called the *QRS vectorcardiogram.* Vectorcardiograms can be recorded on an oscilloscope by connecting body surface electrodes from the neck and lower abdomen to the vertical plates of the oscilloscope and connecting chest surface electrodes from each side of the heart to the horizontal plates. When the vector changes, the spot of light on the oscilloscope follows the course of the positive end of the changing vector, thus inscribing the vectorcardiogram on the oscilloscopic screen.

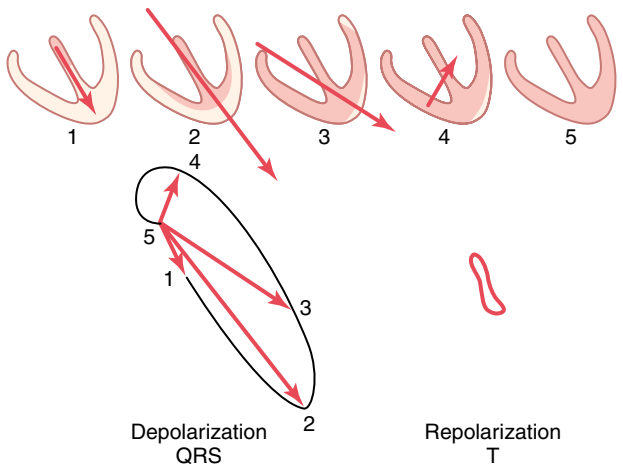


Figure 12-10 QRS and T vectorcardiograms.

Mean Electrical Axis of the Ventricular QRS—and Its Significance

The vectorcardiogram during ventricular depolarization (the QRS vectorcardiogram) shown in Figure 12-10 is that of a normal heart. Note from this vectorcardiogram that the preponderant direction of the vectors of the ventricles during depolarization is mainly toward the apex of the heart. That is, during most of the cycle of ventricular depolarization, the direction of the electrical potential (negative to positive) is from the base of the ventricles toward the apex. This preponderant direction of the potential during depolarization is called the *mean electrical axis of the ventricles.* The mean electrical axis of the normal ventricles is 59 degrees. In many pathological conditions of the heart, this direction changes markedly, sometimes even to opposite poles of the heart.

Determining the Electrical Axis from Standard Lead Electrocardiograms

Clinically, the electrical axis of the heart is usually estimated from the standard bipolar limb lead electrocardiograms rather than from the vectorcardiogram. Figure 12-11 shows a method for doing this. After recording the standard leads, one determines the net potential and polarity of the recordings in leads I and III. In lead I of Figure 12-11, the recording is positive, and in lead III, the recording is mainly positive but negative during part of the cycle. If any part of a recording is negative, *this negative potential is subtracted from the positive part of the potential* to determine the *net potential* for that lead, as shown by the arrow to the right of the QRS complex for lead III. Then each net potential for leads I and III is plotted on the axes of the respective leads, with the base of the potential at the point of intersection of the axes, as shown in Figure 12-11.

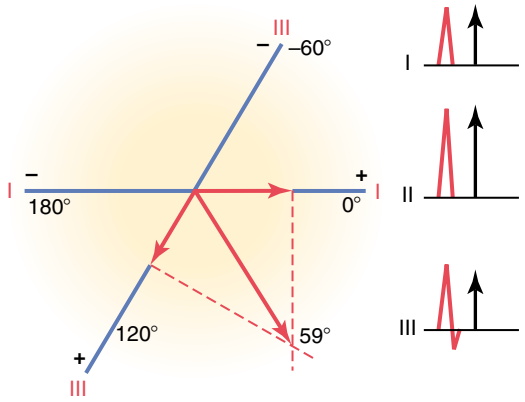


Figure 12-11 Plotting the mean electrical axis of the ventricles from two electrocardiographic leads (leads I and III).

If the net potential of lead I is positive, it is plotted in a positive direction along the line depicting lead I. Conversely, if this potential is negative, it is plotted in a negative direction. Also, for lead III, the net potential is placed with its base at the point of intersection, and, if positive, it is plotted in the positive direction along the line depicting lead III. If it is negative, it is plotted in the negative direction.

To determine the vector of the total QRS ventricular mean electrical potential, one draws perpendicular lines (the dashed lines in the figure) from the apices of leads I and III, respectively. The point of intersection of these two perpendicular lines represents, by vectorial analysis, the apex of the *mean* QRS vector in the ventricles, and the point of intersection of the lead I and lead III axes represents the negative end of the mean vector. Therefore, the *mean QRS vector* is drawn between these two points. The approximate average potential generated by the ventricles during depolarization is represented by the length of this mean QRS vector, and the mean electrical axis is represented by the direction of the mean vector. Thus, the orientation of the mean electrical axis of the normal ventricles, as determined in Figure 12-11, is 59 degrees positive (+59 degrees).

Abnormal Ventricular Conditions That Cause Axis Deviation

Although the mean electrical axis of the ventricles averages about 59 degrees, this axis can swing even in the normal heart from about 20 degrees to about 100 degrees. The causes of the normal variations are mainly anatomical differences in the Purkinje distribution system or in the musculature itself of different hearts. However, a number of abnormal conditions of the heart can cause axis deviation beyond the normal limits, as follows.

Change in the Position of the Heart in the Chest. If the heart itself is angulated to the left, the mean electrical axis of the heart also *shifts to the left*. Such shift occurs (1) at the end of deep expiration, (2) when a person lies down, because the abdominal contents press upward against the diaphragm, and (3) quite frequently in obese people whose diaphragms normally press upward against the heart all the time due to increased visceral adiposity.

Likewise, angulation of the heart to the right causes the mean electrical axis of the ventricles to *shift to the right.* This occurs (1) at the end of deep inspiration, (2) when a person stands up, and (3) normally in tall, lanky people whose hearts hang downward.

Hypertrophy of One Ventricle. When one ventricle greatly hypertrophies, *the axis of the heart shifts toward the hypertrophied ventricle* for two reasons. First, a far greater quantity of muscle exists on the hypertrophied side of the heart than on the other side, and this allows generation of greater electrical potential on that side. Second, more time is required for the depolarization wave to travel through the hypertrophied ventricle than through the normal ventricle. Consequently, the *normal* ventricle becomes depolarized considerably in advance of the *hypertrophied* ventricle, and this causes a strong vector from the normal side of the heart toward the hypertrophied side, which remains strongly positively charged. Thus, the axis deviates toward the hypertrophied ventricle.

Vectorial Analysis of Left Axis Deviation Resulting from Hypertrophy of the Left Ventricle. Figure 12-12 shows the three standard bipolar limb lead electrocardiograms. Vectorial analysis demonstrates left axis deviation with mean electrical axis pointing in the –15-degree direction. This is a typical electrocardiogram caused by increased muscle mass of the left ventricle. In this instance, the axis deviation was caused by *hypertension* (high arterial blood pressure), which caused the left ventricle to hypertrophy so that it could pump blood against elevated systemic arterial pressure. A similar picture of left axis deviation occurs when the

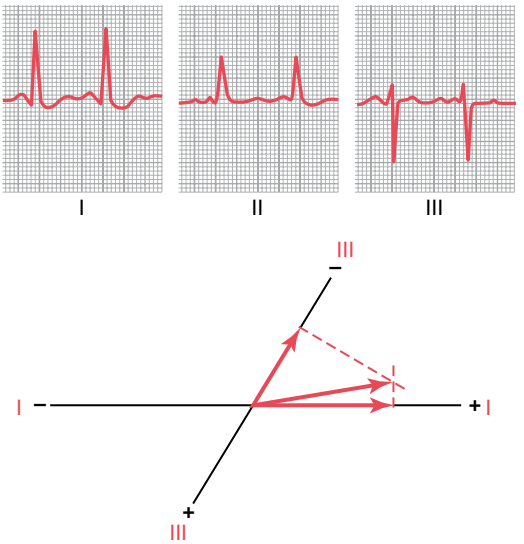


Figure 12-12 Left axis deviation in a ***hypertensive heart (hypertrophic left ventricle)***. Note the slightly prolonged QRS complex as well.

left ventricle hypertrophies as a result of *aortic valvular stenosis, aortic valvular regurgitation,* or any number of *congenital heart conditions* in which the left ventricle enlarges while the right ventricle remains relatively normal in size.

Vectorial Analysis of Right Axis Deviation Resulting from Hypertrophy of the Right Ventricle. The electrocardiogram of Figure 12-13 shows intense right axis deviation, to an electrical axis of 170 degrees, which is 111 degrees to the right of the normal 59-degree mean ventricular QRS axis. The right axis deviation demonstrated in this figure was caused by hypertrophy of the right ventricle as a result of *congenital pulmonary valve stenosis.* Right axis deviation also can occur in other congenital heart conditions that cause hypertrophy of the right ventricle, such as *tetralogy of Fallot* and *interventricular septal defect.*

Bundle Branch Block Causes Axis Deviation. Ordinarily, the lateral walls of the two ventricles depolarize at almost the same instant because both the left and the right bundle branches of the Purkinje system transmit the cardiac impulse to the two ventricular walls at almost the same instant. As a result, the potentials generated by the two ventricles (on the two opposite sides of the heart) almost neutralize each other. But if only one of the major bundle branches is blocked, the cardiac impulse spreads through the normal ventricle long before it spreads through the other. Therefore, depolarization of the two ventricles does not occur even nearly simultaneously, and the depolarization potentials do not neutralize each other. As a result, axis deviation occurs as follows.

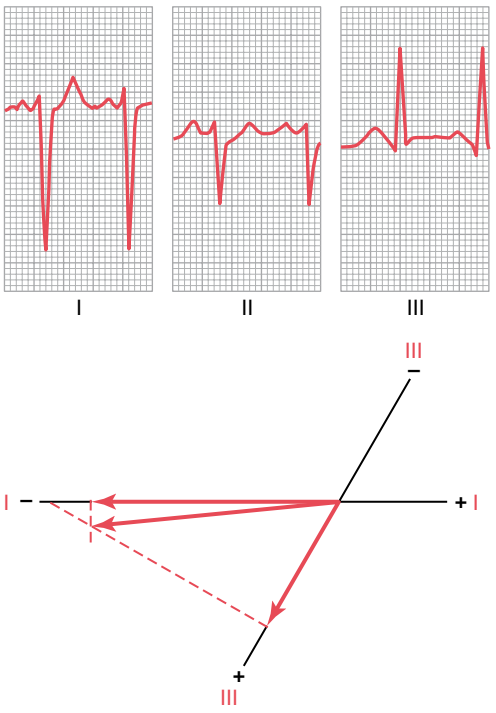


Figure 12-13 High-voltage electrocardiogram in *congenital pulmonary valve stenosis* ***with right ventricular hypertrophy.*** Intense right axis deviation and a slightly prolonged QRS complex also are seen.

Vectorial Analysis of Left Axis Deviation in Left Bundle Branch Block. When the left bundle branch is blocked, cardiac depolarization spreads through the right ventricle two to three times as rapidly as through the left ventricle. Consequently, much of the left ventricle remains polarized for as long as 0.1 second after the right ventricle has become totally depolarized. Thus, the right ventricle becomes electronegative, whereas the left ventricle remains electropositive during most of the depolarization process, and a strong vector projects from the right ventricle toward the left ventricle. In other words, there is intense left axis deviation of about –50 degrees because the positive end of the vector points toward the left ventricle. This is demonstrated in Figure 12-14, which shows typical left axis deviation resulting from left bundle branch block.

Because of slowness of impulse conduction when the Purkinje system is blocked, in addition to axis deviation, the duration of the QRS complex is greatly prolonged because of extreme slowness of depolarization in the affected side of the heart. One can see this by observing the excessive widths of the QRS waves in Figure 12-14. This is discussed in greater detail later in the chapter. This extremely prolonged QRS complex differentiates bundle branch block from axis deviation caused by hypertrophy.

Vectorial Analysis of Right Axis Deviation in Right Bundle Branch Block. When the right bundle branch is blocked, the left ventricle depolarizes far more rapidly than the right ventricle, so the left side of the ventricles becomes electronegative as long as 0.1 second before the right. Therefore, a strong vector develops, with its negative end toward the left ventricle and its positive end toward the right ventricle. In other words, intense right axis deviation occurs. Right axis deviation caused by right bundle branch block is demonstrated, and its vector is

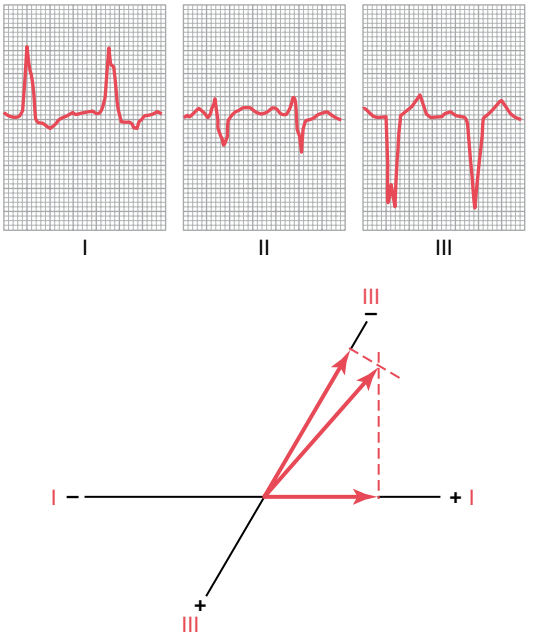


Figure 12-14 Left axis deviation caused by *left bundle branch block.* Note also the greatly prolonged QRS complex.

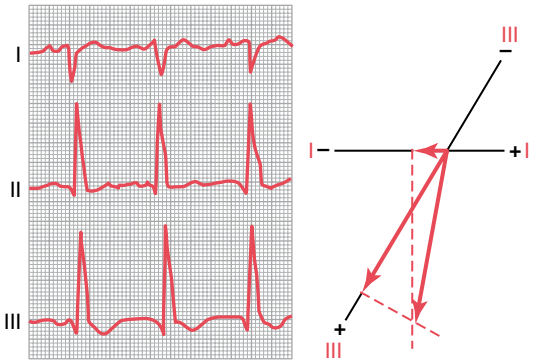


Figure 12-15 Right axis deviation caused by *right bundle branch block*. Note also the greatly prolonged QRS complex.

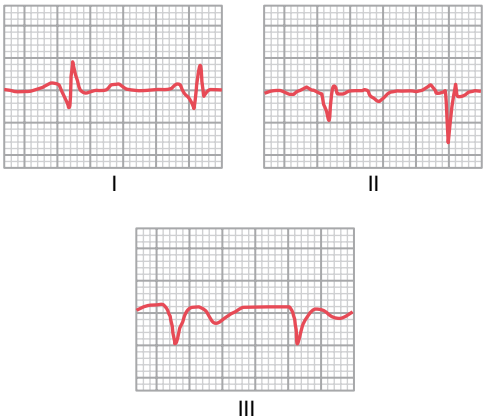


Figure 12-16 Low-voltage electrocardiogram following local damage throughout the ventricles caused by *previous myocardial infarction*.

analyzed, in Figure 12-15, which shows an axis of about 105 degrees instead of the normal 59 degrees and a prolonged QRS complex because of slow conduction.

Conditions That Cause Abnormal Voltages of the QRS Complex

Increased Voltage in the Standard Bipolar Limb Leads

Normally, the voltages in the three standard bipolar limb leads, as measured from the peak of the R wave to the bottom of the S wave, vary between 0.5 and 2.0 millivolts, with lead III usually recording the lowest voltage and lead II the highest. However, these relations are not invariable, even for the normal heart. In general, when the sum of the voltages of all the QRS complexes of the three standard leads is greater than 4 millivolts, the patient is considered to have a high-voltage electrocardiogram.

The cause of high-voltage QRS complexes most often is increased muscle mass of the heart, which ordinarily results from *hypertrophy of the muscle* in response to excessive load on one part of the heart or the other. For example, the right ventricle hypertrophies when it must pump blood through a stenotic pulmonary valve, and the left ventricle hypertrophies when a person has high blood pressure. The increased quantity of muscle causes generation of increased quantities of electricity around the heart. As a result, the electrical potentials recorded in the electrocardiographic leads are considerably greater than normal, as shown in Figures 12-12 and 12-13.

Decreased Voltage of the Electrocardiogram

Decreased Voltage Caused by Cardiac Myopathies. One of the most common causes of decreased voltage of the QRS complex is a series of *old myocardial infarctions* with resultant *diminished muscle mass.* This also causes the depolarization wave to move through the ventricles slowly and prevents major portions of the heart from becoming massively depolarized all at once. Consequently, this condition causes some prolongation of the QRS complex along with the decreased voltage. Figure 12-16 shows a typical low-voltage electrocardiogram with prolongation of the QRS complex, which is common after multiple small infarctions of the heart have caused local delays of impulse conduction and reduced voltages due to loss of muscle mass throughout the ventricles.

Decreased Voltage Caused by Conditions Surrounding the Heart. One of the most important causes of decreased voltage in electrocardiographic leads is *fluid in the pericardium.* Because extracellular fluid conducts electrical currents with great ease, a large portion of the electricity flowing out of the heart is conducted from one part of the heart to another through the pericardial fluid. Thus, this effusion effectively "short-circuits" the electrical potentials generated by the heart, decreasing the electrocardiographic voltages that reach the outside surfaces of the body. *Pleural effusion,* to a lesser extent, also can "short-circuit" the electricity around the heart so that the voltages at the surface of the body and in the electrocardiograms are decreased.

Pulmonary emphysema can decrease the electrocardiographic potentials, but for a different reason than that of pericardial effusion. In pulmonary emphysema, conduction of electrical current through the lungs is depressed considerably because of excessive quantity of air in the lungs. Also, the chest cavity enlarges, and the lungs tend to envelop the heart to a greater extent than normally. Therefore, the lungs act as an insulator to prevent spread of electrical voltage from the heart to the surface of the body, and this results in decreased electrocardiographic potentials in the various leads.

Prolonged and Bizarre Patterns of the QRS Complex

Prolonged QRS Complex as a Result of Cardiac Hypertrophy or Dilatation

The QRS complex lasts as long as depolarization continues to spread through the ventricles—that is, as long as part of the ventricles is depolarized and part is still polarized.

Therefore, *prolonged conduction* of the impulse through the ventricles always causes a prolonged QRS complex. Such prolongation often occurs when one or both ventricles are hypertrophied or dilated, owing to the longer pathway that the impulse must then travel. The normal QRS complex lasts 0.06 to 0.08 second, whereas in hypertrophy or dilatation of the left or right ventricle, the QRS complex may be prolonged to 0.09 to 0.12 second.

Prolonged QRS Complex Resulting from Purkinje System Blocks

When the Purkinje fibers are blocked, the cardiac impulse must then be conducted by the ventricular muscle instead of by way of the Purkinje system. This decreases the velocity of impulse conduction to about one third of normal. Therefore, if complete block of one of the bundle branches occurs, the duration of the QRS complex is usually increased to 0.14 second or greater.

In general, a QRS complex is considered to be abnormally long when it lasts more than 0.09 second; when it lasts more than 0.12 second, the prolongation is almost certainly caused by pathological block somewhere in the ventricular conduction system, as shown by the electrocardiograms for bundle branch block in Figures 12-14 and 12-15.

Conditions That Cause Bizarre QRS Complexes

Bizarre patterns of the QRS complex most frequently are caused by two conditions: (1) destruction of cardiac muscle in various areas throughout the ventricular system, with replacement of this muscle by scar tissue, and (2) multiple small local blocks in the conduction of impulses at many points in the Purkinje system. As a result, cardiac impulse conduction becomes irregular, causing rapid shifts in voltages and axis deviations. This often causes double or even triple peaks in some of the electrocardiographic leads, such as those shown in Figure 12-14.

Current of Injury

Many different cardiac abnormalities, especially those that damage the heart muscle itself, often cause part of the heart to remain partially or totally *depolarized all the time.* When this occurs, current flows between the pathologically depolarized and the normally polarized areas even between heartbeats. This is called a *current of injury.* Note especially that *the injured part of the heart is negative, because this is the part that is depolarized and emits negative charges into the surrounding fluids, whereas the remainder of the heart is neutral or positive polarity.*

Some abnormalities that can cause current of injury are (1) *mechanical trauma,* which sometimes makes the membranes remain so permeable that full repolarization cannot take place; (2) *infectious processes* that damage the muscle membranes; and (3) *ischemia of local areas of heart muscle caused by local coronary occlusions,* which is by far the most common cause of current of injury in the heart. During ischemia, not enough nutrients from the coronary blood supply are available to the heart muscle to maintain normal membrane polarization.

Effect of Current of Injury on the QRS Complex

In Figure 12-17, a small area in the base of the left ventricle is newly infarcted (loss of coronary blood flow). Therefore, during the T-P interval—that is, when the normal ventricular muscle is totally polarized—abnormal *negative* current still flows from the infarcted area at the base of the left ventricle and spreads toward the rest of the ventricles.

The vector of this "current of injury," as shown in the first heart in Figure 12-17, is in a direction of about 125 degrees, with the base of the vector, the *negative end,* toward the injured muscle. As shown in the lower portions of the figure, even before the QRS complex begins, *this vector causes an initial record in lead I below the zero potential line,* because the projected vector of the current of injury in lead I points toward the negative end of the lead I axis. In lead II, the record is above the line because the projected vector points more toward the positive terminal of the lead. In lead III, the projected vector points in the same direction as the positive terminal of lead III so that the record is positive. Furthermore, because the vector lies almost exactly in the direction of the axis of lead III, the voltage of the current of injury in lead III is much greater than in either lead I or lead II.

As the heart then proceeds through its normal process of depolarization, the septum first becomes depolarized; then the depolarization spreads down to the apex and back toward the bases of the ventricles. The last portion of the ventricles to become totally depolarized is the base of the right ventricle, because the base of the left ventricle is already totally and permanently depolarized. By vectorial analysis, the successive stages of electrocardiogram generation by the depolarization wave traveling through the ventricles can be constructed graphically, as demonstrated in the lower part of Figure 12-17.

When the heart becomes totally depolarized, at the end of the depolarization process (as noted by the next-to-last stage in Figure 12-17), all the ventricular muscle is in a negative state. Therefore, at this instant in the electrocardiogram, no current flows from the ventricles to the electrocardiographic electrodes because now both the injured heart muscle and the contracting muscle are depolarized.

Next, as repolarization takes place, all of the heart finally repolarizes, except the area of permanent depolarization in the injured base of the left ventricle. Thus, repolarization causes a return of the current of injury in each lead, as noted at the far right in Figure 12-17.

The J Point—the Zero Reference Potential for Analyzing Current of Injury

One might think that the electrocardiograph machines for recording electrocardiograms could determine when no current is flowing around the heart. However, many

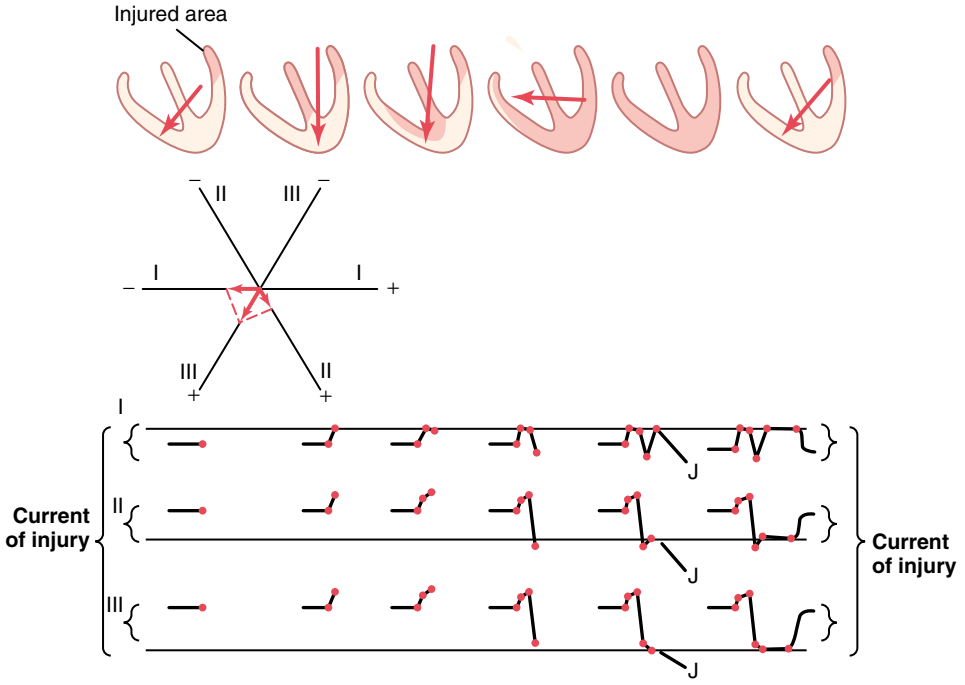


Figure 12-17 Effect of a current of injury on the electrocardiogram.

stray currents exist in the body, such as currents resulting from "skin potentials" and from differences in ionic concentrations in different fluids of the body. Therefore, when two electrodes are connected between the arms or between an arm and a leg, these stray currents make it impossible to predetermine the exact zero reference level in the electrocardiogram.

For these reasons, the following procedure must be used to determine the zero potential level: First, one notes *the exact point at which the wave of depolarization just completes its passage through the heart,* which occurs at the end of the QRS complex. At exactly this point, all parts of the ventricles have become depolarized, including both the damaged parts and the normal parts, so no current is flowing around the heart. Even the current of injury disappears at this point. Therefore, the potential of the electrocardiogram at this instant is at zero voltage. This point is known as the "J" point in the electrocardiogram, as shown in Figure 12-18.

Then, for analysis of the electrical axis of the injury potential caused by a current of injury, a horizontal line is drawn in the electrocardiogram for each lead at the level of the J point. This horizontal line is then the *zero potential level* in the electrocardiogram from which all potentials caused by currents of injury must be measured.

Use of the J Point in Plotting Axis of Injury Potential. Figure 12-18 shows electrocardiograms (leads I and III) from an injured heart. Both records show injury potentials. In other words, the J point of each of these two electrocardiograms is not on the same line as the T-P segment. In the figure, a horizontal line has been drawn through the J point to represent the zero voltage level in each of the two recordings. The injury potential in each lead is the difference between the voltage of the electrocardiogram immediately before onset of the P wave and the zero voltage level determined from the J point. In lead I, the recorded voltage of the injury potential is above the zero potential level and is, therefore, positive. Conversely, in lead III, the injury potential is below the zero voltage level and, therefore, is negative.

At the bottom in Figure 12-18, the respective injury potentials in leads I and III are plotted on the coordinates of these leads, and the resultant vector of the injury potential for the whole ventricular muscle mass

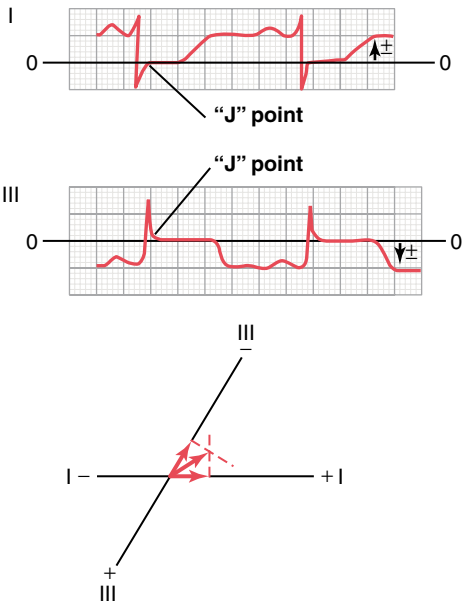


Figure 12-18 J point as the zero reference potential of the electrocardiograms for leads I and III. Also, the method for plotting the axis of the injury potential is shown by the lowermost panel.

is determined by vectorial analysis as described. In this instance, the resultant vector extends from the right side of the ventricles toward the left and slightly upward, with an axis of about –30 degrees. If one places this vector for the injury potential directly over the ventricles, *the negative end of the vector points toward the permanently depolarized, "injured" area of the ventricles.* In the example shown in Figure 12-18, the injured area would be in the lateral wall of the right ventricle.

This analysis is obviously complex. However, it is essential that the student go over it again and again until he or she understands it thoroughly. No other aspect of electrocardiographic analysis is more important.

Coronary Ischemia as a Cause of Injury Potential

Insufficient blood flow to the cardiac muscle depresses the metabolism of the muscle for three reasons: (1) lack of oxygen, (2) excess accumulation of carbon dioxide, and (3) lack of sufficient food nutrients. Consequently, repolarization of the muscle membrane cannot occur in areas of severe myocardial ischemia. Often the heart muscle does not die because the blood flow is sufficient to maintain life of the muscle even though it is not sufficient to cause repolarization of the membranes. As long as this state exists, an injury potential continues to flow during the diastolic portion (the T-P portion) of each heart cycle.

Extreme ischemia of the cardiac muscle occurs after coronary occlusion, and a strong current of injury flows from the infarcted area of the ventricles during the T-P interval between heartbeats, as shown in Figures 12-19 and 12-20. Therefore, one of the most important diagnostic features of electrocardiograms recorded after acute coronary thrombosis is the current of injury.

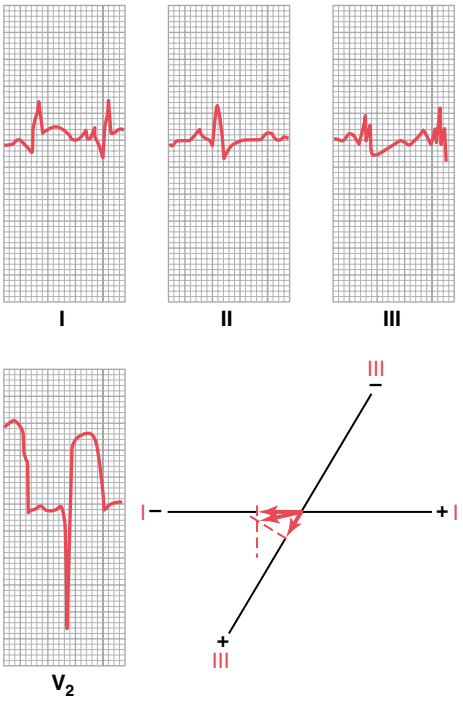


Figure 12-19 Current of injury in *acute anterior wall infarction*. Note the intense injury potential in lead V_2.

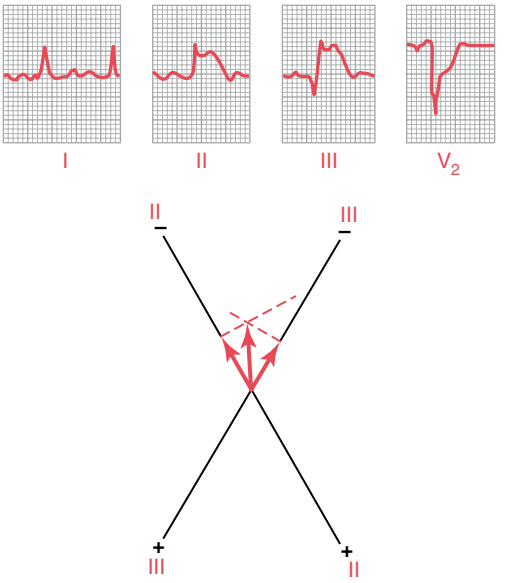


Figure 12-20 Injury potential in ***acute posterior wall, apical infarction.***

Acute Anterior Wall Infarction. Figure 12-19 shows the electrocardiogram in the three standard bipolar limb leads and in one chest lead (lead V_2) recorded from a patient with acute anterior wall cardiac infarction. The most important diagnostic feature of this electrocardiogram is the intense injury potential in chest lead V_2. If one draws a zero horizontal potential line through the J point of this electrocardiogram, a strong *negative* injury potential during the T-P interval is found, which means that the chest electrode over the front of the heart is in an area of strongly negative potential. In other words, the negative end of the injury potential vector in this heart is against the anterior chest wall. This means that the current of injury is emanating from the anterior wall of the ventricles, which diagnoses this condition as *anterior wall infarction.*

Analyzing the injury potentials in leads I and III, one finds a negative potential in lead I and a positive potential in lead III. This means that the resultant vector of the injury potential in the heart is about +150 degrees, with the negative end pointing toward the left ventricle and the positive end pointing toward the right ventricle. Thus, in this particular electrocardiogram, the current of injury is coming mainly from the left ventricle, as well as from the anterior wall of the heart. Therefore, one would conclude that this anterior wall infarction almost certainly is caused by thrombosis of the anterior descending branch of the left coronary artery.

Posterior Wall Infarction. Figure 12-20 shows the three standard bipolar limb leads and one chest lead (lead V_2) from a patient with posterior wall infarction. The major diagnostic feature of this electrocardiogram is also in the chest lead. If a zero potential reference line is drawn through the J point of this lead, it is readily apparent that during the T-P interval, the potential of the current of injury is positive. This means that the positive end of the vector is in the direction of the anterior chest wall, and the negative end (injured end of the vector) points away

from the chest wall. In other words, the current of injury is coming from the back of the heart opposite to the anterior chest wall, which is the reason this type of electrocardiogram is the basis for diagnosing posterior wall infarction.

If one analyzes the injury potentials from leads II and III of Figure 12-20, it is readily apparent that the injury potential is negative in both leads. By vectorial analysis, as shown in the figure, one finds that the resultant vector of the injury potential is about −95 degrees, with the negative end pointing downward and the positive end pointing upward. Thus, because the infarct, as indicated by the chest lead, is on the posterior wall of the heart and, as indicated by the injury potentials in leads II and III, is in the apical portion of the heart, one would suspect that this infarct is near the apex on the posterior wall of the left ventricle.

Infarction in Other Parts of the Heart. By the same procedures demonstrated in the preceding discussions of anterior and posterior wall infarctions, it is possible to determine the locus of any infarcted area emitting a current of injury, regardless of which part of the heart is involved. In making such vectorial analyses, it must be remembered that *the positive end of the injury potential vector points toward the normal cardiac muscle, and the negative end points toward the injured portion of the heart that is emitting the current of injury.*

Recovery from Acute Coronary Thrombosis. Figure 12-21 shows a V_3 chest lead from a patient with acute posterior wall infarction, demonstrating changes in the electrocardiogram from the day of the attack to 1 week later, 3 weeks later, and finally 1 year later. From this electrocardiogram, one can see that the injury potential is strong immediately after the acute attack (T-P segment displaced positively from the S-T segment). However, after about 1 week, the injury potential has diminished considerably, and after 3 weeks, it is gone. After that, the electrocardiogram does not change greatly during the next year. This is the usual recovery pattern after acute myocardial infarction of moderate degree, showing that the *new collateral coronary blood flow* develops enough to re-establish appropriate nutrition to most of the infarcted area.

Conversely, in some patients with myocardial infarction, the infarcted area never redevelops adequate coronary blood supply. Often, some of the heart muscle

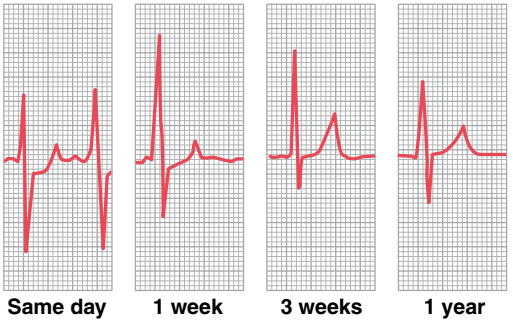


Figure 12-21 Recovery of the myocardium after ***moderate posterior wall infarction,*** demonstrating disappearance of the injury potential that is present on the first day after the infarction and still slightly present at 1 week.

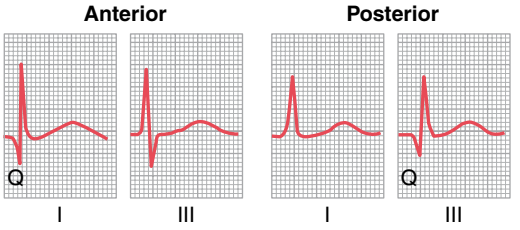


Figure 12-22 Electrocardiograms of anterior and posterior wall infarctions that occurred about 1 year previously, showing a Q wave in lead I in anterior wall infarction and a Q wave in lead III in ***posterior wall infarction.***

dies, but if the muscle does not die, it will continue to show an injury potential as long as the ischemia exists, particularly during bouts of exercise when the heart is overloaded.

Old Recovered Myocardial Infarction. Figure 12-22 shows leads I and III after *anterior infarction* and leads I and III after *posterior infarction* about 1 year after the acute heart attack. The records show what might be called the "ideal" configurations of the QRS complex in these types of recovered myocardial infarction. Usually a Q wave has developed at the beginning of the QRS complex in lead I in anterior infarction because of loss of muscle mass in the anterior wall of the left ventricle, but in posterior infarction, a Q wave has developed at the beginning of the QRS complex in lead III because of loss of muscle in the posterior apical part of the ventricle.

These configurations are certainly not found in all cases of old cardiac infarction. Local loss of muscle and local points of cardiac signal conduction block can cause very bizarre QRS patterns (especially prominent Q waves, for instance), decreased voltage, and QRS prolongation.

Current of Injury in Angina Pectoris. "Angina pectoris" means pain from the heart felt in the pectoral regions of the upper chest. This pain usually also radiates into the left neck area and down the left arm. The pain is typically caused by moderate ischemia of the heart. Usually, no pain is felt as long as the person is quiet, but as soon as he or she overworks the heart, the pain appears.

An injury potential sometimes appears in the electrocardiogram during an attack of severe angina pectoris because the coronary insufficiency becomes great enough to prevent adequate repolarization of some areas of the heart during diastole.

Abnormalities in the T Wave

Earlier in the chapter, it was pointed out that the T wave is normally positive in all the standard bipolar limb leads and that this is caused by repolarization of the apex and outer surfaces of the ventricles ahead of the intraventricular surfaces. That is, the T wave becomes abnormal when the normal sequence of repolarization does not occur. Several factors can change this sequence of repolarization.

Effect of Slow Conduction of the Depolarization Wave on the Characteristics of the T Wave

Referring to Figure 12-14, note that the QRS complex is considerably prolonged. The reason for this prolongation is *delayed conduction in the left ventricle* resulting from left bundle branch block. This causes the left ventricle to become depolarized about 0.08 second after depolarization of the right ventricle, which gives a strong mean QRS vector *to the left.* However, the refractory periods of the right and left ventricular muscle masses are not greatly different from each other. Therefore, the right ventricle begins to repolarize long before the left ventricle; this causes strong positivity in the right ventricle and negativity in the left ventricle at the time that the T wave is developing. In other words, the mean axis of the T wave is now deviated *to the right,* which is opposite the mean electrical axis of the QRS complex in the same electrocardiogram. Thus, when conduction of the depolarization impulse through the ventricles is greatly delayed, the T wave is almost always of opposite polarity to that of the QRS complex.

Shortened Depolarization in Portions of the Ventricular Muscle as a Cause of T Wave Abnormalities

If the base of the ventricles should exhibit an abnormally short period of depolarization, that is, a shortened action potential, repolarization of the ventricles would not begin at the apex as it normally does. Instead, the base of the ventricles would repolarize ahead of the apex, and the vector of repolarization would point from the apex toward the base of the heart, opposite to the standard vector of repolarization. Consequently, the T wave in all three standard leads would be negative rather than the usual positive. Thus, the simple fact that the base of the ventricles has a shortened period of depolarization is sufficient to cause marked changes in the T wave, even to the extent of changing the entire T wave polarity, as shown in Figure 12-23.

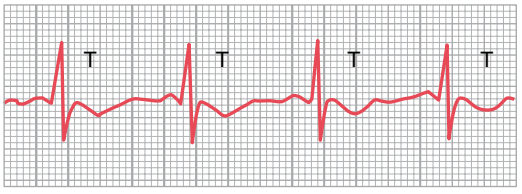


Figure 12-23 Inverted T wave resulting from mild *ischemia at the apex* of the ventricles.

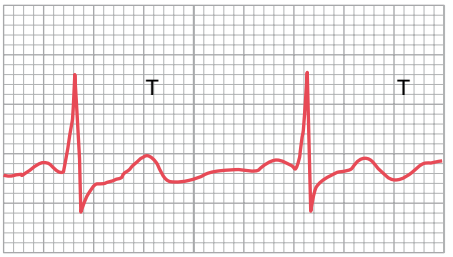


Figure 12-24 Biphasic T wave caused by *digitalis toxicity*.

Mild ischemia is by far the most common cause of shortening of depolarization of cardiac muscle because this increases current flow through the potassium channels. When the ischemia occurs in only one area of the heart, the depolarization period of this area decreases out of proportion to that in other portions. As a result, definite changes in the T wave can take place. The ischemia might result from chronic, progressive coronary occlusion; acute coronary occlusion; or relative coronary insufficiency that occurs during exercise.

One means for detecting mild coronary insufficiency is to have the patient exercise and to record the electrocardiogram, noting whether changes occur in the T waves. The changes in the T waves need not be specific because any change in the T wave in any lead—inversion, for instance, or a biphasic wave—is often evidence enough that some portion of the ventricular muscle has a period of depolarization out of proportion to the rest of the heart, caused by mild to moderate coronary insufficiency.

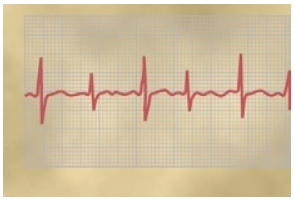
Effect of Digitalis on the T Wave. As discussed in Chapter 22, digitalis is a drug that can be used during coronary insufficiency to increase the strength of cardiac muscle contraction. But when overdosages of digitalis are given, depolarization duration in one part of the ventricles may be increased out of proportion to that of other parts. As a result, nonspecific changes, such as T wave inversion or biphasic T waves, may occur in one or more of the electrocardiographic leads. A biphasic T wave caused by excessive administration of digitalis is shown in Figure 12-24. Therefore, changes in the T wave during digitalis administration are often the earliest signs of digitalis toxicity.

Bibliography

See bibliography for Chapter 13.

CHAPTER 13

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 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/min. 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.

Some 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/min for each degree of 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/min.

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/min. 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

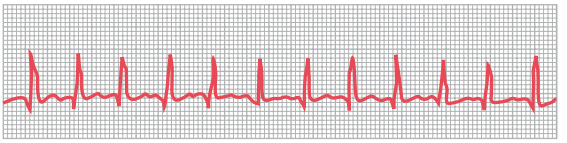


Figure 13-1 Sinus tachycardia (lead I).

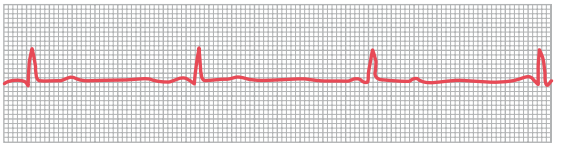


Figure 13-2 Sinus bradycardia (lead III).

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.

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 percent 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 percent.

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.

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

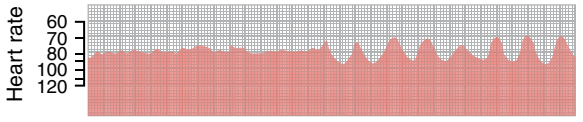


Figure 13-3 Sinus arrhythmia as recorded by a cardiotachometer. To the left is the record when the subject was breathing normally; to the right, when breathing deeply.

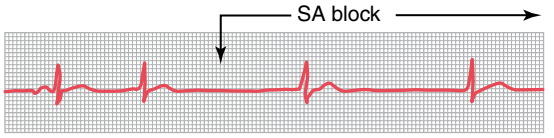


Figure 13-4 Sinoatrial nodal block, with A-V nodal rhythm during the block period (lead III).

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 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 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

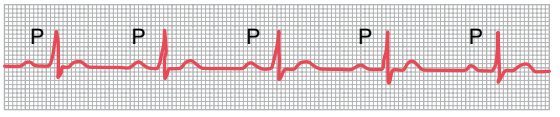


Figure 13-5 Prolonged P-R interval caused by first degree A-V heart block (lead II).

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 is sometimes strong enough to pass through the bundle into the ventricles and sometimes 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 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 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.

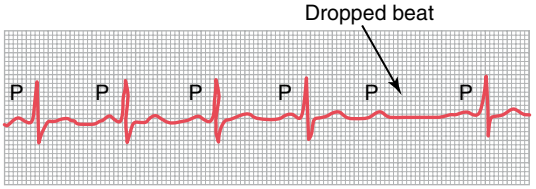


Figure 13-6 Second degree A-V block, showing occasional failure of the ventricles to receive the excitatory signals (lead V_3).

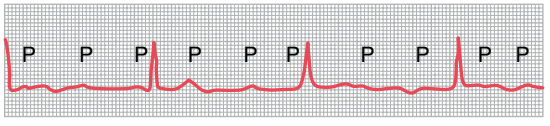


Figure 13-7 Complete A-V block (lead II).

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.*

Because the brain cannot remain active for more than 4 to 7 seconds without blood supply, most patients faint a few seconds after complete block occurs because the heart does not pump any blood for 5 to 30 seconds, until the ventricles "escape." After escape, however, the slowly beating ventricles usually pump enough blood to allow rapid recovery from the faint and then to sustain the person. These periodic fainting spells are known as the *Stokes-Adams syndrome.*

Occasionally the interval of ventricular standstill at the onset of complete block is so long that it becomes detrimental to the patient's health or even causes death. Consequently, most of these patients are provided with an *artificial pacemaker,* a small battery-operated electrical stimulator planted beneath the skin, with electrodes usually connected to the right ventricle. The pacemaker provides continued rhythmical impulses that take control of the ventricles.

Incomplete Intraventricular Block—Electrical Alternans

Most of the same factors that can cause A-V block can also block impulse conduction in the peripheral ventricular Purkinje system. Figure 13-8 shows the condition known as *electrical alternans,* which results from partial intraventricular block every other heartbeat.

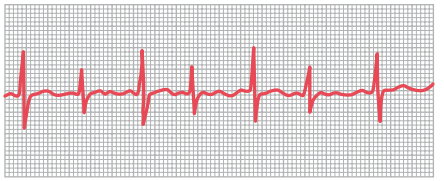


Figure 13-8 Partial intraventricular block—"electrical alternans" (lead III).

This electrocardiogram also shows *tachycardia* (rapid heart rate), which is probably the reason the block has occurred, because when the rate of the heart is rapid, it may be impossible for some portions of the Purkinje system to recover from the previous refractory period quickly enough to respond during every succeeding heartbeat. Also, many conditions that depress the heart, such as ischemia, myocarditis, or digitalis toxicity, can cause incomplete intraventricular block, resulting in electrical alternans.

Premature Contractions

A premature contraction is a contraction of the heart before the time that normal contraction would have been expected. This condition is also called *extrasystole, premature beat,* or *ectopic beat.*

Causes of Premature Contractions. Most premature contractions result from *ectopic foci* in the heart, which emit abnormal impulses at odd times during the cardiac rhythm. Possible causes of ectopic foci are (1) local areas of ischemia; (2) small calcified plaques at different points in the heart, which press against the adjacent cardiac muscle so that some of the fibers are irritated; and (3) toxic irritation of the A-V node, Purkinje system, or myocardium caused by drugs, nicotine, or caffeine. Mechanical initiation of premature contractions is also frequent during cardiac catheterization; large numbers of premature contractions often occur when the catheter enters the right ventricle and presses against the endocardium.

Premature Atrial Contractions

Figure 13-9 shows a single premature atrial contraction. The P wave of this beat occurred too soon in the heart cycle; the P-R interval is shortened, indicating that the ectopic origin of the beat is in the atria near the A-V node. Also, the interval between the premature contraction and the next succeeding contraction is slightly prolonged, which is called a *compensatory pause.* One of the reasons for this is that the premature contraction originated in the atrium some distance from the sinus node, and the impulse had to travel through a considerable amount of atrial muscle before it discharged the sinus node. Consequently, the sinus node discharged late in the premature cycle, and this made the succeeding sinus node discharge also late in appearing.

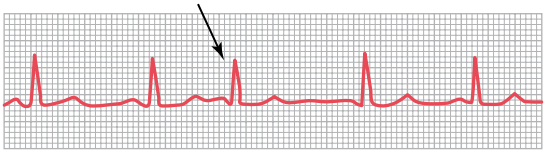


Figure 13-9 Atrial premature beat (lead I).

Premature atrial contractions occur frequently in otherwise healthy people. Indeed, they often occur in athletes whose hearts are in very healthy condition. Mild toxic conditions resulting from such factors as smoking, lack of sleep, ingestion of too much coffee, alcoholism, and use of various drugs can also initiate such contractions.

Pulse Deficit. When the heart contracts ahead of schedule, the ventricles will not have filled with blood normally, and the stroke volume output during that contraction is depressed or almost absent. Therefore, the pulse wave passing to the peripheral arteries after a premature contraction may be so weak that it cannot be felt in the radial artery. Thus, a deficit in the number of radial pulses occurs when compared with the actual number of contractions of the heart.

A-V Nodal or A-V Bundle Premature Contractions

Figure 13-10 shows a premature contraction that originated in the A-V node or in the A-V bundle. The P wave is missing from the electrocardiographic record of the premature contraction. Instead, the P wave is superimposed onto the QRS-T complex because the cardiac impulse traveled backward into the atria at the same time that it traveled forward into the ventricles; this P wave slightly distorts the QRS-T complex, but the P wave itself cannot be discerned as such. In general, A-V nodal premature contractions have the same significance and causes as atrial premature contractions.

Premature Ventricular Contractions

The electrocardiogram of Figure 13-11 shows a series of premature ventricular contractions (PVCs) alternating with normal contractions. PVCs cause specific effects in the electrocardiogram, as follows:

1. The QRS complex is usually considerably prolonged. The reason is that the impulse is conducted mainly through slowly conducting muscle of the ventricles rather than through the Purkinje system.
2. The QRS complex has a high voltage for the following reasons: when the normal impulse passes through the heart, it passes through both ventricles nearly simultaneously; consequently, in the normal heart, the depolarization waves of the two sides of the heart—mainly of opposite polarity to each other—partially neutralize each other in the electrocardiogram. When a PVC

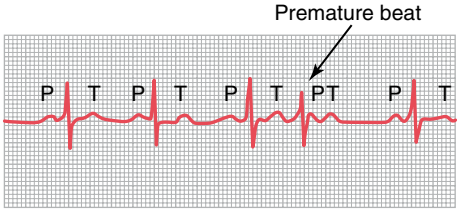


Figure 13-10 A-V nodal premature contraction (lead III).

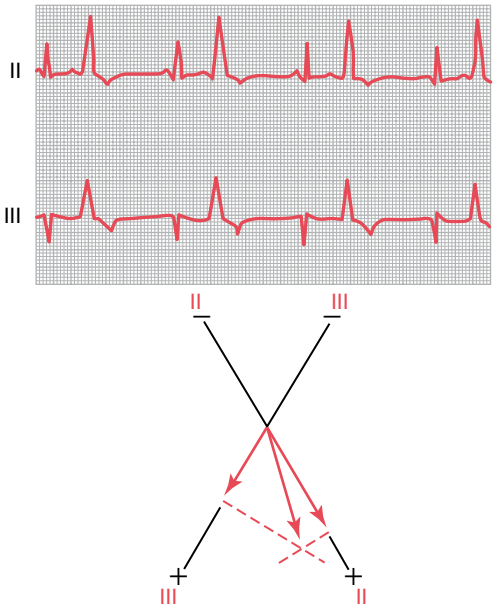


Figure 13-11 Premature ventricular contractions (PVCs) demonstrated by the large abnormal QRS-T complexes (leads II and III). Axis of the premature contractions is plotted in accordance with the principles of vectorial analysis explained in Chapter 12; this shows the origin of the PVC to be near the base of the ventricles.

occurs, the impulse almost always travels in only one direction, so there is no such neutralization effect, and one entire side or end of the ventricles is depolarized ahead of the other; this causes large electrical potentials, as shown for the PVCs in Figure 13-11.

3. After almost all PVCs, the T wave has an electrical potential polarity exactly opposite to that of the QRS complex because the *slow conduction of the impulse* through the cardiac muscle causes the muscle fibers that depolarize first also to repolarize first.

Some PVCs are relatively benign in their effects on overall pumping by the heart; they can result from such factors as cigarettes, excessive intake of coffee, lack of sleep, various mild toxic states, and even emotional irritability. Conversely, many other PVCs result from stray impulses or re-entrant signals that originate around the borders of infarcted or ischemic areas of the heart. The presence of such PVCs is not to be taken lightly. Statistics show that people with significant numbers of PVCs have a much higher than normal chance of developing spontaneous lethal ventricular fibrillation, presumably initiated by one of the PVCs. This is especially true when the PVCs occur during the vulnerable period for causing fibrillation, just at the end of the T wave when the ventricles are coming out of refractoriness, as explained later in the chapter.

Vector Analysis of the Origin of an Ectopic Premature Ventricular Contraction. In Chapter 12, the principles of vectorial analysis are explained. Applying these principles, one can determine from the electrocardiogram in Figure 13-11 the point of origin of the PVC as follows: Note that the potentials of the premature contractions in leads II and III are both strongly positive. Plotting these potentials on the axes of leads II and III and solving by vectorial analysis for the mean QRS vector in the heart, one finds that the vector of this premature contraction has its negative end (origin) at the base of the heart and its positive end toward the apex. Thus, the first portion of the heart to become depolarized during this premature contraction is near the base of the ventricles, which therefore is the locus of the ectopic focus.

Disorders of Cardiac Repolarization—The Long QT Syndromes. Recall that the Q wave corresponds to ventricular depolarization while the T wave corresponds to ventricular repolarization. The Q-T interval is the time from the Q point to the end of the T wave. Disorders that delay repolarization of ventricular muscle following the action potential cause prolonged ventricular action potentials and therefore excessively long Q-T intervals on the electrocardiogram, a condition called *long QT syndrome* (LQTS).

The major reason that the long QT syndrome is of concern is that delayed repolarization of ventricular muscle increases a person's susceptibility to develop ventricular arrhythmias called *torsades de pointes,* which literally means "twisting of the points." This type of arrhythmia has the features shown in Figure 13-12. The shape of the QRS complex may change over time with the onset of arrhythmia usually following a premature beat, a pause, and then another beat with a long Q-T interval, which may trigger arrhythmias, tachycardia, and in some instances ventricular fibrillation.

Disorders of cardiac repolarization that lead to LQTS may be inherited or acquired. The congenital forms of LQTS are rare disorders caused by mutations of sodium or potassium channel genes. At least 10 different mutations of these genes that can cause variable degrees of Q-T prolongation have been identified.

More common are the acquired forms of LQTS that are associated with plasma electrolyte disturbances, such as hypomagnesemia, hypokalemia, or hypocalcemia, or with administration of excess amounts of antiarrhythmic drugs such as quinidine or some antibiotics such as fluoroquinolones or erythromycin that prolong the Q-T interval.

Although some people with LQTS show no major symptoms (other than the prolonged Q-T interval), others exhibit fainting and ventricular arrhythmias that may be precipitated by physical exercise, intense emotions such as fright or anger, or when startled by a noise. The ventricular arrhythmias associated with LQTS can, in some cases, deteriorate into ventricular fibrillation and sudden death.

Treatment for LQTS may include magnesium sulfate for acute LQTS, and for long-term LQTS antiarrhythmia medications, such as beta-adrenergic blockers, or surgical implantation of a cardiac defibrillator are used.

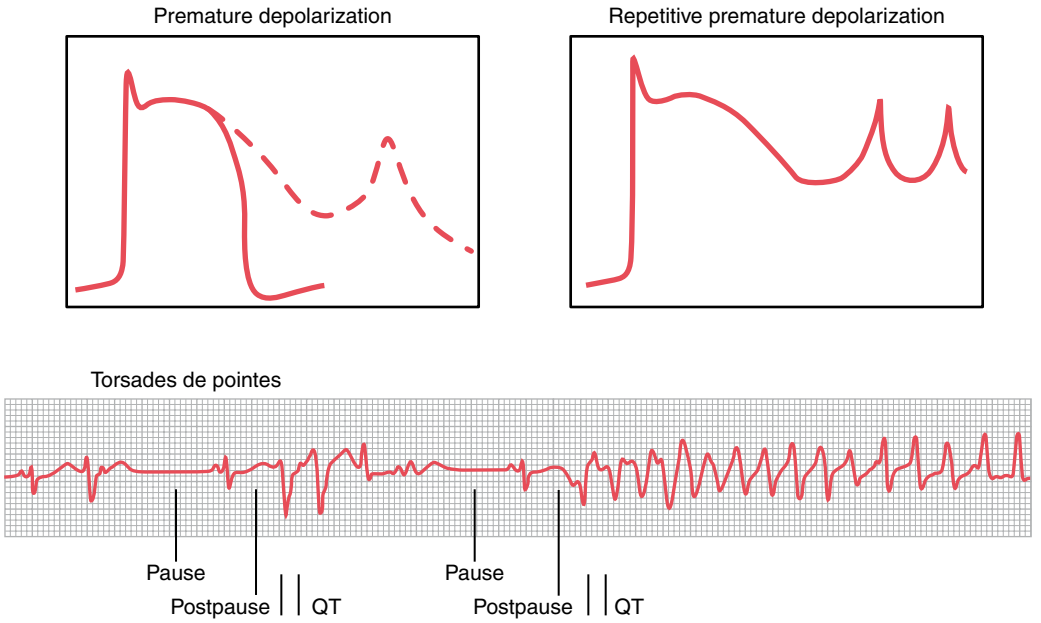


Figure 13-12 Development of arrhythmias in long QT syndrome (LQTS). When the ventricular muscle fiber action potential is prolonged as a result of delayed repolarization, a premature depolarization (***dashed line in top left figure***) may occur before complete repolarization. Repetitive premature depolarizations (***right top figure***) may lead to multiple depolarizations under certain conditions. In torsades de pointes (***bottom figure***), premature ventricular beats lead pauses, postpause prolongation of the Q-T interval, and arrhythmias. (Redrawn from Murray KT, Roden DM: Disorders of cardiac repolarization: the long QT syndromes. In: Crawford MG, DiMarco JP [eds]: Cardiology. London: Mosby, 2001.)

Paroxysmal Tachycardia

Some abnormalities in different portions of the heart, including the atria, the Purkinje system, or the ventricles, can occasionally cause rapid rhythmical discharge of impulses that spread in all directions throughout the heart. This is believed to be caused most frequently by re-entrant circus movement feedback pathways that set up local repeated self–re-excitation. Because of the rapid rhythm in the irritable focus, this focus becomes the pacemaker of the heart.

The term "paroxysmal" means that the heart rate becomes rapid in paroxysms, with the paroxysm beginning suddenly and lasting for a few seconds, a few minutes, a few hours, or much longer. Then the paroxysm usually ends as suddenly as it began, with the pacemaker of the heart instantly shifting back to the sinus node.

Paroxysmal tachycardia often can be stopped by eliciting a vagal reflex. A type of vagal reflex sometimes elicited for this purpose is to press on the neck in the regions of the carotid sinuses, which may cause enough of a vagal reflex to stop the paroxysm. Various drugs may also be used. Two drugs frequently used are quinidine and lidocaine, either of which depresses the normal increase in sodium permeability of the cardiac muscle membrane during generation of the action potential, thereby often blocking the rhythmical discharge of the focal point that is causing the paroxysmal attack.

Atrial Paroxysmal Tachycardia

Figure 13-13 demonstrates in the middle of the record a sudden increase in the heart rate from about 95 to about 150 beats per minute. On close study of the

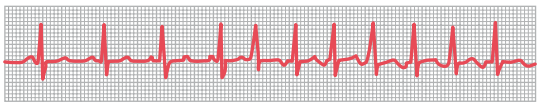


Figure 13-13 Atrial paroxysmal tachycardia—onset in middle of record (lead I).

electrocardiogram during the rapid heartbeat, an inverted P wave is seen before each QRS-T complex, and this P wave is partially superimposed onto the normal T wave of the preceding beat. This indicates that the origin of this paroxysmal tachycardia is in the atrium, but because the P wave is abnormal in shape, the origin is not near the sinus node.

A-V Nodal Paroxysmal Tachycardia. Paroxysmal tachycardia often results from an aberrant rhythm that involves the A-V node. This usually causes almost normal QRS-T complexes but totally missing or obscured P waves.

Atrial or A-V nodal paroxysmal tachycardia, both of which are called *supraventricular tachycardias,* usually occurs in young, otherwise healthy people, and they generally grow out of the predisposition to tachycardia after adolescence. In general, supraventricular tachycardia frightens a person tremendously and may cause weakness during the paroxysm, but only seldom does permanent harm come from the attack.

Ventricular Paroxysmal Tachycardia

Figure 13-14 shows a typical short paroxysm of ventricular tachycardia. The electrocardiogram of ventricular paroxysmal tachycardia has the appearance of a series of

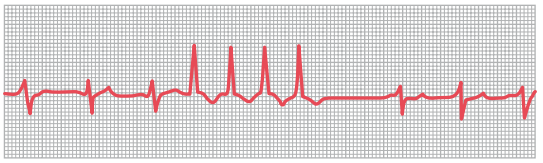


Figure 13-14 Ventricular paroxysmal tachycardia (lead III).

ventricular premature beats occurring one after another without any normal beats interspersed.

Ventricular paroxysmal tachycardia is usually a serious condition for two reasons. First, this type of tachycardia usually does not occur unless considerable ischemic damage is present in the ventricles. Second, *ventricular tachycardia frequently initiates the lethal condition of ventricular fibrillation* because of rapid repeated stimulation of the ventricular muscle, as we discuss in the next section.

Sometimes intoxication from the heart treatment drug *digitalis* causes irritable foci that lead to ventricular tachycardia. Conversely, *quinidine,* which increases the refractory period and threshold for excitation of cardiac muscle, may be used to block irritable foci causing ventricular tachycardia.

Ventricular Fibrillation

The most serious of all cardiac arrhythmias is ventricular fibrillation, which, if not stopped within 1 to 3 minutes, is almost invariably fatal. Ventricular fibrillation results from cardiac impulses that have gone berserk within the ventricular muscle mass, stimulating first one portion of the ventricular muscle, then another portion, then another, and eventually feeding back onto itself to re-excite the same ventricular muscle over and over—never stopping. When this happens, many small portions of the ventricular muscle will be contracting at the same time, while equally as many other portions will be relaxing. Thus, there is never a coordinate contraction of all the ventricular muscle at once, which is required for a pumping cycle of the heart. Despite massive movement of stimulatory signals throughout the ventricles, the ventricular chambers neither enlarge nor contract but remain in an indeterminate stage of partial contraction, pumping either no blood or negligible amounts. Therefore, after fibrillation begins, unconsciousness occurs within 4 to 5 seconds for lack of blood flow to the brain, and irretrievable death of tissues begins to occur throughout the body within a few minutes.

Multiple factors can spark the beginning of ventricular fibrillation—a person may have a normal heartbeat one moment, but 1 second later, the ventricles are in fibrillation. Especially likely to initiate fibrillation are (1) sudden electrical shock of the heart or (2) ischemia of the heart muscle, of its specialized conducting system, or both.

Phenomenon of Re-entry—"Circus Movements" as the Basis for Ventricular Fibrillation

When the *normal* cardiac impulse in the normal heart has traveled through the extent of the ventricles, it has no place to go because all the ventricular muscle is refractory and cannot conduct the impulse farther. Therefore, that impulse dies, and the heart awaits a new action potential to begin in the atrial sinus node.

Under some circumstances, however, this normal sequence of events does not occur. Therefore, let us explain more fully the background conditions that can initiate re-entry and lead to "circus movements," which in turn cause ventricular fibrillation.

Figure 13-15 shows several small cardiac muscle strips cut in the form of circles. If such a strip is stimulated at the 12 o'clock position *so that the impulse travels in only one direction,* the impulse spreads progressively around the circle until it returns to the 12 o'clock position. If the originally stimulated muscle fibers are still in a refractory state, the impulse then dies out because refractory muscle cannot transmit a second impulse. But there are three different conditions that can cause this impulse to continue to travel around the circle, that is, to cause "re-entry" of the impulse into muscle that has already been excited. This is called a "circus movement."

First, if the *pathway around the circle is too long,* by the time the impulse returns to the 12 o'clock position, the originally stimulated muscle will no longer be refractory and the impulse will continue around the circle again and again.

Second, if the length of the pathway remains constant but the *velocity of conduction becomes decreased* enough, an increased interval of time will elapse before the impulse returns to the 12 o'clock position. By this time, the originally stimulated muscle might be out of the refractory state, and the impulse can continue around the circle again and again.

Third, *the refractory period of the muscle might become greatly shortened.* In this case, the impulse could also continue around and around the circle.

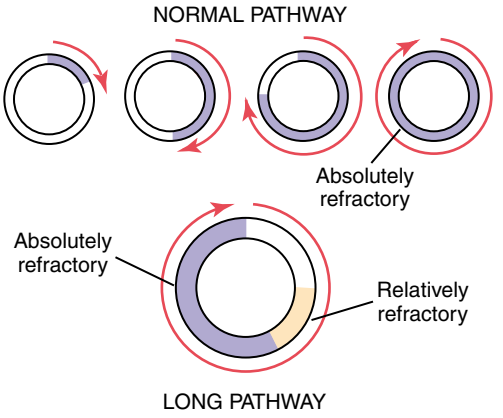


Figure 13-15 Circus movement, showing annihilation of the impulse in the short pathway and continued propagation of the impulse in the long pathway.

All these conditions occur in different pathological states of the human heart, as follows: (1) A long pathway typically occurs in dilated hearts. (2) Decreased rate of conduction frequently results from (a) blockage of the Purkinje system, (b) ischemia of the muscle, (c) high blood potassium levels, or (d) many other factors. (3) A shortened refractory period commonly occurs in response to various drugs, such as epinephrine, or after repetitive electrical stimulation. Thus, in many cardiac disturbances, re-entry can cause abnormal patterns of cardiac contraction or abnormal cardiac rhythms that ignore the pace-setting effects of the sinus node.

Chain Reaction Mechanism of Fibrillation

In ventricular fibrillation, one sees many separate and small contractile waves spreading at the same time in different directions over the cardiac muscle. The re-entrant impulses in fibrillation are not simply a single impulse moving in a circle, as shown in Figure 13-15. Instead, they have degenerated into a series of multiple wave fronts that have the appearance of a "chain reaction." One of the best ways to explain this process in fibrillation is to describe the initiation of fibrillation by electric shock caused by 60-cycle alternating electric current.

Fibrillation Caused by 60-Cycle Alternating Current. At a central point in the ventricles of heart A in Figure 13-16, a 60-cycle electrical stimulus is applied through a stimulating electrode. The first cycle of the electrical stimulus causes a depolarization wave to spread in all directions, leaving all the muscle beneath the electrode in a refractory state. After about 0.25 second, part of this muscle begins to come out of the refractory state. Some portions come out of refractoriness before other portions. This state of events is depicted in heart A by many lighter patches, which represent excitable cardiac muscle, and dark patches, which represent still refractory muscle. Now, continuing 60-cycle stimuli from the electrode can cause impulses to travel only in certain directions through the heart but not in all directions. Thus, in heart A, certain impulses travel for short distances, until they reach refractory areas of the heart, and then are blocked. But other impulses pass between the refractory areas and continue to travel in the excitable areas. Then, several events transpire in rapid succession, all occurring simultaneously and eventuating in a state of fibrillation.

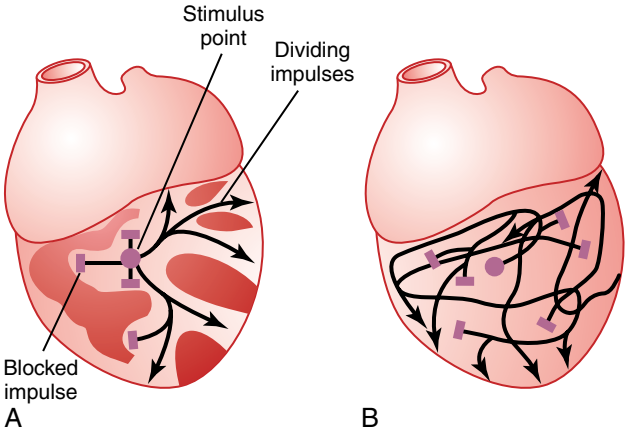


Figure 13-16 *A,* Initiation of fibrillation in a heart when patches of refractory musculature are present. *B,* Continued propagation of *fibrillatory impulses* in the fibrillating ventricle.

First, block of the impulses in some directions but successful transmission in other directions creates one of the necessary conditions for a re-entrant signal to develop—that is, *transmission of some of the depolarization waves around the heart in only some directions but not other directions.*

Second, the rapid stimulation of the heart causes two changes in the cardiac muscle itself, both of which predispose to circus movement: (1) The *velocity of conduction through the heart muscle decreases,* which allows a longer time interval for the impulses to travel around the heart. (2) The *refractory period of the muscle is shortened,* allowing re-entry of the impulse into previously excited heart muscle within a much shorter time than normally.

Third, one of the most important features of fibrillation is the *division of impulses,* as demonstrated in heart A. When a depolarization wave reaches a refractory area in the heart, it travels to both sides around the refractory area. Thus, a single impulse becomes two impulses. Then, when each of these reaches another refractory area, it, too, divides to form two more impulses. In this way, many new wave fronts are continually being formed in the heart by progressive *chain reactions* until, finally, there are many small depolarization waves traveling in many directions at the same time. Furthermore, this irregular pattern of impulse travel causes *many circuitous routes for the impulses to travel, greatly lengthening the conductive pathway, which is one of the conditions that sustains the fibrillation.* It also results in a continual irregular pattern of patchy refractory areas in the heart.

One can readily see when a vicious circle has been initiated: More and more impulses are formed; these cause more and more patches of refractory muscle, and the refractory patches cause more and more division of the impulses. Therefore, any time a single area of cardiac muscle comes out of refractoriness, an impulse is close at hand to re-enter the area.

Heart B in Figure 13-16 demonstrates the final state that develops in fibrillation. Here one can see many impulses traveling in all directions, some dividing and increasing the number of impulses, whereas others are blocked by refractory areas. In fact, a single electric shock during this vulnerable period frequently can lead to an odd pattern of impulses spreading multidirectionally around refractory areas of muscle, which will lead to fibrillation.

Electrocardiogram in Ventricular Fibrillation

In ventricular fibrillation, the electrocardiogram is bizarre (Figure 13-17) and ordinarily shows no tendency toward a regular rhythm of any type. During the first few seconds

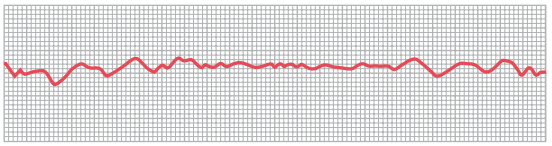


Figure 13-17 Ventricular fibrillation (lead II).

of ventricular fibrillation, relatively large masses of muscle contract simultaneously, and this causes coarse, irregular waves in the electrocardiogram. After another few seconds, the coarse contractions of the ventricles disappear, and the electrocardiogram changes into a new pattern of low-voltage, very irregular waves. Thus, no repetitive electrocardiographic pattern can be ascribed to ventricular fibrillation. Instead, the ventricular muscle contracts at as many as 30 to 50 small patches of muscle at a time, and electrocardiographic potentials change constantly and spasmodically because the electrical currents in the heart flow first in one direction and then in another and seldom repeat any specific cycle.

The voltages of the waves in the electrocardiogram in ventricular fibrillation are usually about 0.5 millivolt when ventricular fibrillation first begins, but they decay rapidly so that after 20 to 30 seconds, they are usually only 0.2 to 0.3 millivolt. Minute voltages of 0.1 millivolt or less may be recorded for 10 minutes or longer after ventricular fibrillation begins. As already pointed out, because no pumping of blood occurs during ventricular fibrillation, this state is lethal unless stopped by some heroic therapy, such as immediate electroshock through the heart, as explained in the next section.

Electroshock Defibrillation of the Ventricles

Although a moderate alternating-current voltage applied directly to the ventricles almost invariably throws the ventricles into fibrillation, a strong high-voltage alternating electrical current passed through the ventricles for a fraction of a second can stop fibrillation by throwing all the ventricular muscle into refractoriness simultaneously. This is accomplished by passing intense current through large electrodes placed on two sides of the heart. The current penetrates most of the fibers of the ventricles at the same time, thus stimulating essentially all parts of the ventricles simultaneously and causing them all to become refractory. All action potentials stop, and the heart remains quiescent for 3 to 5 seconds, after which it begins to beat again, usually with the sinus node or some other part of the heart becoming the pacemaker. However, the same re-entrant focus that had originally thrown the ventricles into fibrillation often is still present, in which case fibrillation may begin again immediately.

When electrodes are applied directly to the two sides of the heart, fibrillation can usually be stopped using 110 volts of 60-cycle alternating current applied for 0.1 second or 1000 volts of direct current applied for a few thousandths of a second. When applied through two electrodes on the chest wall, as shown in Figure 13-18, the

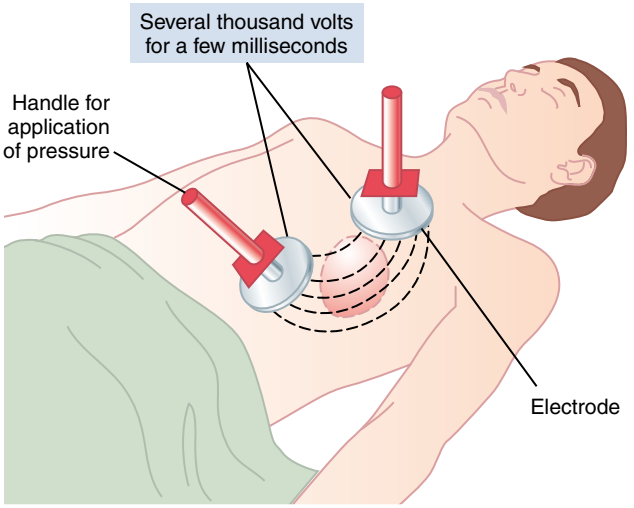


Figure 13-18 Application of electrical current to the chest to stop ventricular fibrillation.

usual procedure is to charge a large electrical capacitor up to several thousand volts and then to cause the capacitor to discharge for a few thousandths of a second through the electrodes and through the heart.

Hand Pumping of the Heart (Cardiopulmonary Resuscitation) as an Aid to Defibrillation

Unless defibrillated within 1 minute after fibrillation begins, the heart is usually too weak to be revived by defibrillation because of the lack of nutrition from coronary blood flow. However, it is still possible to revive the heart by preliminarily pumping the heart by hand (intermittent hand squeezing) and then defibrillating the heart later. In this way, small quantities of blood are delivered into the aorta and a renewed coronary blood supply develops. Then, after a few minutes of hand pumping, electrical defibrillation often becomes possible. Indeed, fibrillating hearts have been pumped by hand for as long as 90 minutes followed by successful defibrillation.

A technique for pumping the heart without opening the chest consists of intermittent thrusts of pressure on the chest wall along with artificial respiration. This, plus defibrillation, is called *cardiopulmonary resuscitation,* or CPR.

Lack of blood flow to the brain for more than 5 to 8 minutes usually causes permanent mental impairment or even destruction of brain tissue. Even if the heart is revived, the person may die from the effects of brain damage or may live with permanent mental impairment.

Atrial Fibrillation

Remember that except for the conducting pathway through the A-V bundle, the atrial muscle mass is separated from the ventricular muscle mass by fibrous tissue. Therefore, ventricular fibrillation often occurs without atrial fibrillation. Likewise, fibrillation often occurs in the

atria without ventricular fibrillation (shown to the right in Figure 13-20).

The mechanism of atrial fibrillation is identical to that of ventricular fibrillation, except that the process occurs only in the atrial muscle mass instead of the ventricular mass. A frequent cause of atrial fibrillation is atrial enlargement resulting from heart valve lesions that prevent the atria from emptying adequately into the ventricles, or from ventricular failure with excess damming of blood in the atria. The dilated atrial walls provide ideal conditions of a long conductive pathway, as well as slow conduction, both of which predispose to atrial fibrillation.

Pumping Characteristics of the Atria during Atrial Fibrillation. For the same reasons that the ventricles will not pump blood during ventricular fibrillation, neither do the atria pump blood in atrial fibrillation. Therefore, the atria become useless as primer pumps for the ventricles. Even so, blood flows passively through the atria into the ventricles, and the efficiency of ventricular pumping is decreased only 20 to 30 percent. Therefore, in contrast to the lethality of ventricular fibrillation, a person can live for months or even years with atrial fibrillation, although at reduced efficiency of overall heart pumping.

Electrocardiogram in Atrial Fibrillation. Figure 13-19 shows the electrocardiogram during atrial fibrillation. Numerous small depolarization waves spread in all directions through the atria during atrial fibrillation. Because the waves are weak and many of them are of opposite polarity at any given time, they usually almost completely electrically neutralize one another. Therefore, in the electrocardiogram, one can see either no P waves from the atria or only a fine, high-frequency, very low voltage wavy record. Conversely, the QRS-T complexes are normal unless there is some pathology of the ventricles, but their timing is irregular, as explained next.

Irregularity of Ventricular Rhythm during Atrial Fibrillation. When the atria are fibrillating, impulses arrive from the atrial muscle at the A-V node rapidly but also irregularly. Because the A-V node will not pass a second impulse for about 0.35 second after a previous one, at least 0.35 second must elapse between one ventricular contraction and the next. Then an additional but variable interval of 0 to 0.6 second occurs before one of the irregular atrial fibrillatory impulses happens to arrive at the A-V node. Thus, the interval between successive ventricular contractions varies from a minimum of about 0.35 second to a maximum of about 0.95 second, causing a very irregular heartbeat. In fact, this irregularity, demonstrated by the variable spacing of the heartbeats in the electrocardiogram of Figure 13-19, is one of the clinical findings used to diagnose the condition. Also, because of the rapid rate of the fibrillatory impulses in the atria, the ventricle is driven at a fast heart rate, usually between 125 and 150 beats per minute.

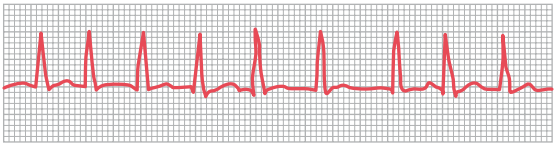


Figure 13-19 Atrial fibrillation (lead I). The waves that can be seen are ventricular QRS and T waves.

Electroshock Treatment of Atrial Fibrillation. In the same manner that ventricular fibrillation can be converted back to a normal rhythm by electroshock, so too can atrial fibrillation be converted by electroshock. The procedure is essentially the same as for ventricular fibrillation conversion—passage of a single strong electric shock through the heart, which throws the entire heart into refractoriness for a few seconds; a normal rhythm often follows *if the heart is capable of this.*

Atrial Flutter

Atrial flutter is another condition caused by a circus movement in the atria. It is different from atrial fibrillation, in that the electrical signal travels as a single large wave always in one direction around and around the atrial muscle mass, as shown to the left in Figure 13-20. Atrial flutter causes a rapid rate of contraction of the atria, usually between 200 and 350 beats per minute. However, because one side of the atria is contracting while the other side is relaxing, the amount of blood pumped by the atria is slight. Furthermore, the signals reach the A-V node too rapidly for all of them to be passed into the ventricles, because the refractory periods of the A-V node and A-V bundle are too long to pass more than a fraction of the atrial signals. Therefore, there are usually two to three beats of the atria for every single beat of the ventricles.

Figure 13-21 shows a typical electrocardiogram in atrial flutter. The P waves are strong because of contraction of semicoordinate masses of muscle. However, note

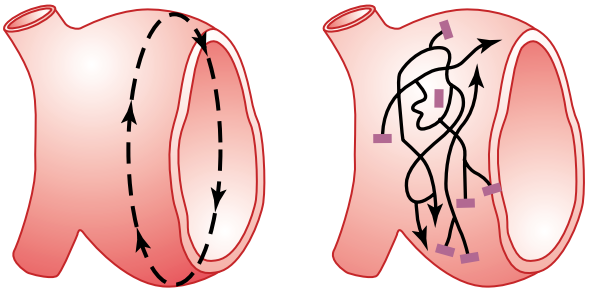


Figure 13-20 Pathways of impulses in atrial flutter and atrial fibrillation.

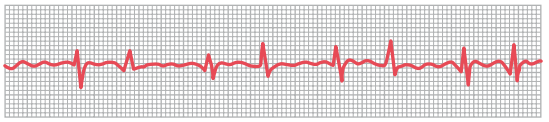


Figure 13-21 Atrial flutter—2:1 and 3:1 atrial to ventricle rhythm (lead I).

in the record that a QRS-T complex follows an atrial P wave only once for every two to three beats of the atria, giving a 2:1 or 3:1 rhythm.

Cardiac Arrest

A final serious abnormality of the cardiac rhythmicity-conduction system is *cardiac arrest.* This results from cessation of all electrical control signals in the heart. That is, no spontaneous rhythm remains.

Cardiac arrest may occur *during deep anesthesia,* when many patients develop severe hypoxia because of inadequate respiration. The hypoxia prevents the muscle fibers and conductive fibers from maintaining normal electrolyte concentration differentials across their membranes, and their excitability may be so affected that the automatic rhythmicity disappears.

In most instances of cardiac arrest from anesthesia, prolonged cardiopulmonary resuscitation (many minutes or even hours) is quite successful in re-establishing a normal heart rhythm. In some patients, severe myocardial disease can cause permanent or semipermanent cardiac arrest, which can cause death. To treat the condition, rhythmical electrical impulses from an *implanted electronic cardiac pacemaker* have been used successfully to keep patients alive for months to years.

Bibliography

Antzelevitch C: Role of spatial dispersion of repolarization in inherited and acquired sudden cardiac death syndromes, *Am J Physiol Heart Circ Physiol* 293:H2024, 2007.

Awad MM, Calkins H, Judge DP: Mechanisms of disease: molecular genetics of arrhythmogenic right ventricular dysplasia/cardiomyopathy, *Nat Clin Pract Cardiovasc Med* 5:258, 2008.

Barbuti A, DiFrancesco D: Control of cardiac rate by "funny" channels in health and disease, *Ann N Y Acad Sci* 1123:213, 2008.

Cheng H, Lederer WJ: Calcium sparks, *Physiol Rev* 88:1491, 2008.

Dobrzynski H, Boyett MR, Anderson RH: New insights into pacemaker activity: promoting understanding of sick sinus syndrome, *Circulation* 115:1921, 2007.

Elizari MV, Acunzo RS, Ferreiro M: Hemiblocks revisited, *Circulation* 115:1154, 2007.

Jalife J: Ventricular fibrillation: mechanisms of initiation and maintenance, *Annu Rev Physiol* 62:25, 2000.

Lubitz SA, Fischer A, Fuster V: Catheter ablation for atrial fibrillation, *BMJ* 336:819, 2008.

Maron BJ: Sudden death in young athletes, *N Engl J Med* 349:1064, 2003.

Morita H, Wu J, Zipes DP: The QT syndromes: long and short, *Lancet* 372:750, 2008.

Murray KT, Roden DM: Disorders of cardiac repolarization: the long QT syndromes. In Crawford MG, DiMarco JP, editors: Cardiology, London, 2001, Mosby.

Myerburg RJ: Implantable cardioverter-defibrillators after myocardial infarction, *N Engl J Med* 359:2245, 2008.

Passman R, Kadish A: Sudden death prevention with implantable devices, *Circulation* 116:561, 2007.

Roden DM: Drug-induced prolongation of the QT interval, *N Engl J Med* 350:1013, 2004.

Sanguinetti MC: Tristani-Firouzi M: hERG potassium channels and cardiac arrhythmia, *Nature* 440:463, 2006.

Swynghedauw B, Baillard C, Milliez P: The long QT interval is not only inherited but is also linked to cardiac hypertrophy, *J Mol Med* 81:336, 2003.

Wang K, Asinger RW, Marriott HJ: ST-segment elevation in conditions other than acute myocardial infarction, *N Engl J Med* 349:2128, 2003.

Zimetbaum PJ, Josephson ME: Use of the electrocardiogram in acute myocardial infarction, *N Engl J Med* 348:933, 2003.