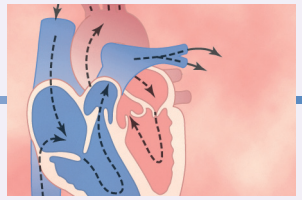


CHAPTER 9

UNIT III

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

Our discussion of the heart and circulatory system begins in this chapter. 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 systemic circulation that provides blood flow to the other organs and tissues of the body. 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 systemic 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.

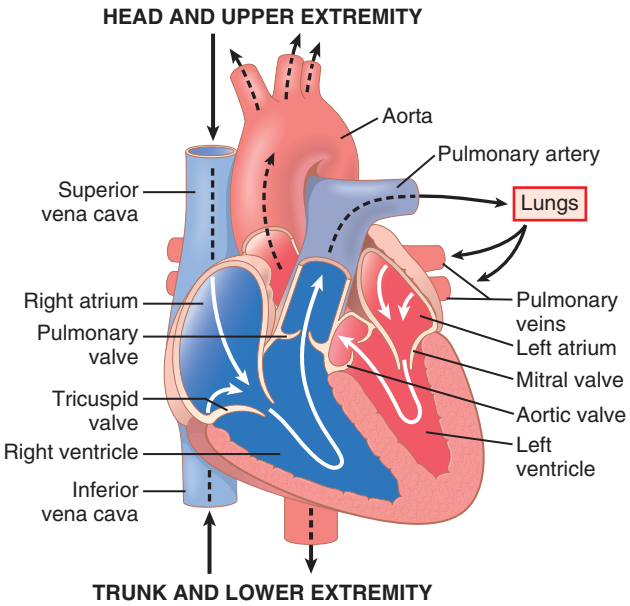


Figure 9-1. Structure of the heart and course of blood flow through the heart chambers and heart valves.

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 of the heart, 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. Note 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 during contraction in the same manner as occurs in skeletal muscle (see Chapter 6). In other ways, however, cardiac muscle is quite different from skeletal muscle, as we shall see.

Cardiac Muscle Is 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 to 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 action potentials

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

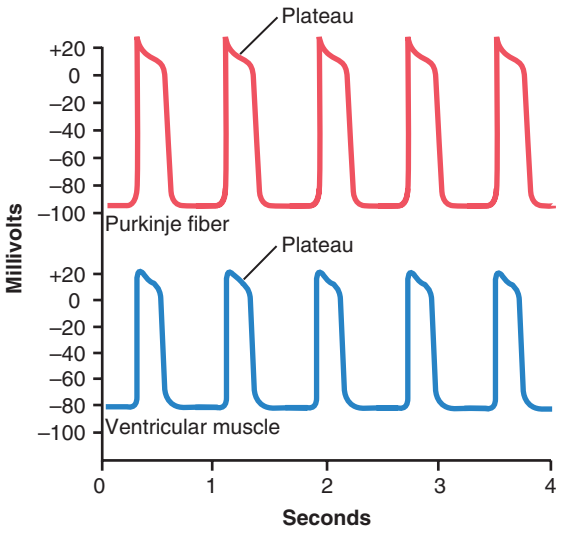


Figure 9-3. Rhythmical action potentials (in millivolts) from a Purkinje fiber and from a ventricular muscle fiber, recorded by means of microelectrodes.

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 cell becomes excited, the action potential rapidly spreads to all of them.

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 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*, 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? Why is the action potential of cardiac muscle so long, why does it have a plateau, when action potential of skeletal muscle does not have a plateau? The basic biophysical answers to these questions were presented in Chapter 5, but they merit recapitulating 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 *activation of skeletal muscle* is caused almost entirely by the opening of large numbers of *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 *voltage-activated sodium channels* as those in skeletal muscle and (2) another entirely different population of *slow L-type calcium channels*. These *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, 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 activity 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 contraction process, whereas most of the calcium ions that cause skeletal muscle contraction are derived from the intracellular sarcoplasmic reticulum.

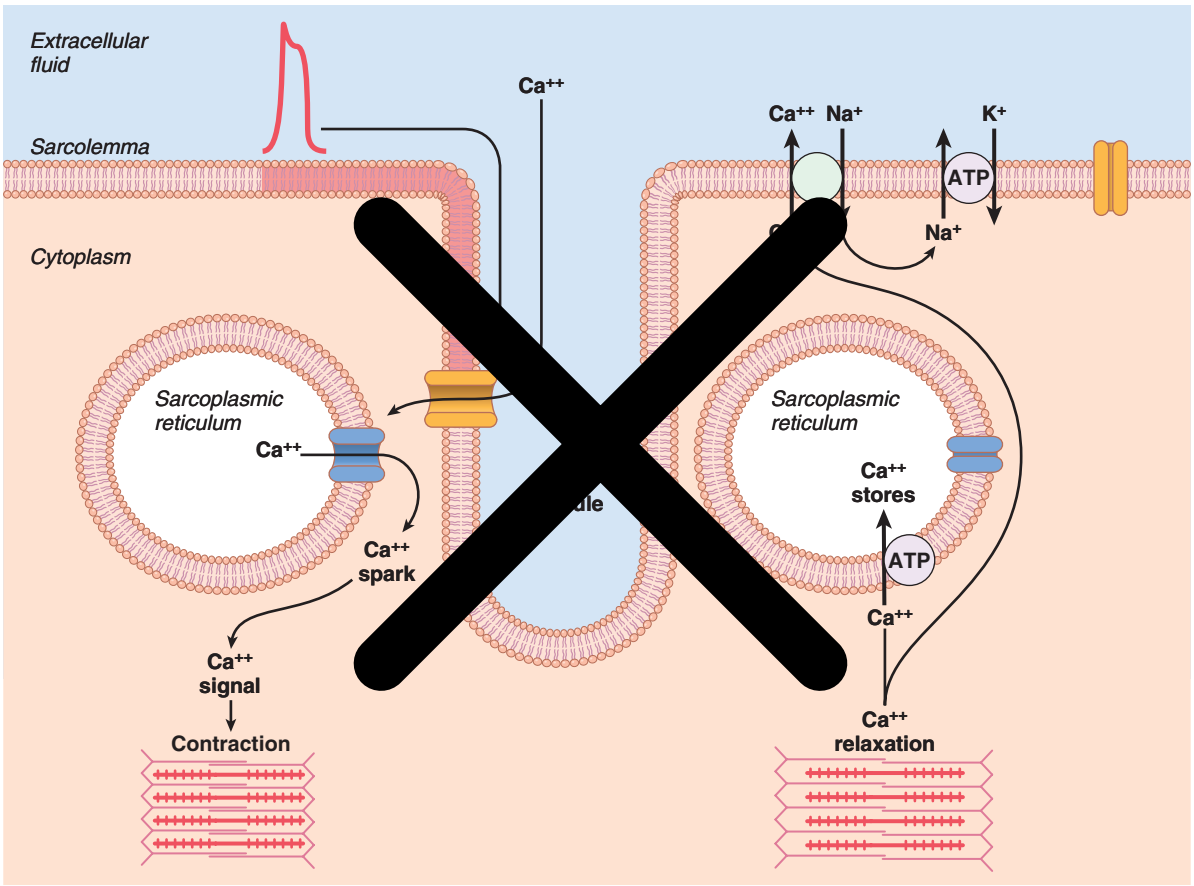


Figure 9-6. Mechanisms of excitation-contraction coupling and relaxation in cardiac muscle. ATP, adenosine triphosphate.

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 delay 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 min/beat—about 0.0139 minutes per beat, or 0.833 second per beat.

Figure 9-7 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 depicts the electrocardiogram, and the sixth depicts 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.

Increasing Heart Rate Decreases 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

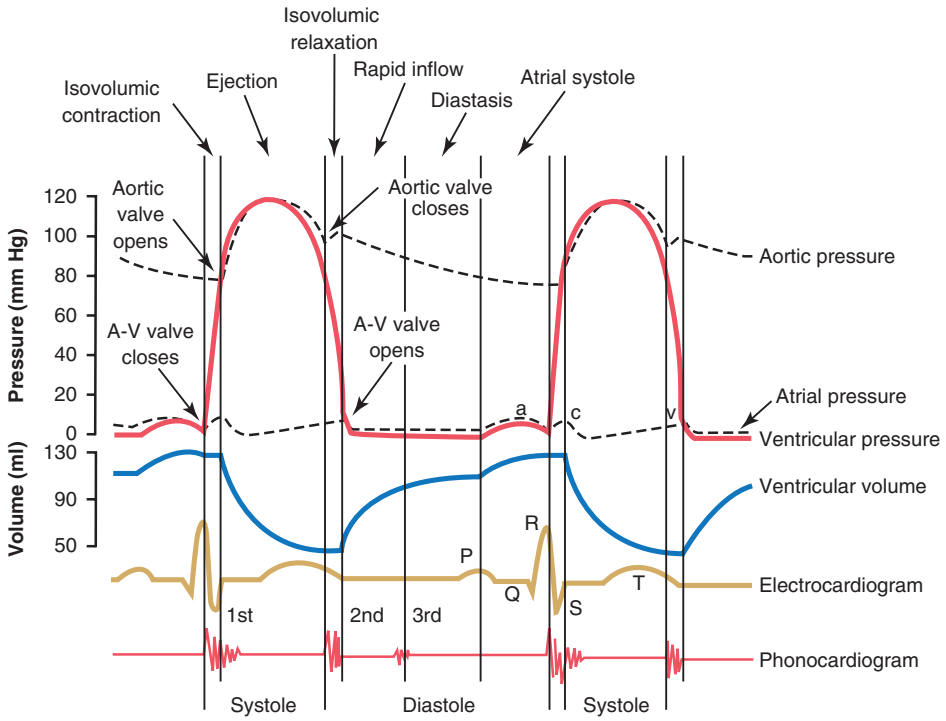


Figure 9-7. Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram. A-V, atrioventricular.

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 9-7** 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 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. Therefore, the QRS complex begins slightly before the onset of ventricular systole.

Finally, the *ventricular T wave* 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.

The Atria Function as Primer Pumps for the Ventricles

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 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 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 9-7**, three minor pressure elevations, called the *a, c,* and *v atrial pressure waves,* are shown.

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

The Ventricles Fill With Blood 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 9-7**. This period 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 mechanism accounts for about 20 percent of the filling of the ventricles during each heart cycle.

Outflow of Blood From the Ventricles During Systole

Period of Isovolumic (Isometric) Contraction. Immediately after ventricular contraction begins, the ventricular pressure rises abruptly, as shown in **Figure 9-7**, 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 no emptying occurs. This period is called the period of *isovolumic* or *isometric contraction,* meaning that cardiac muscle tension is increasing 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 rises slightly above 8 mm Hg), the ventricular pressures push the semilunar valves open. Immediately, blood begins to pour out of the ventricles. Approximately 60 percent of the blood in the ventricle at the end of diastole is ejected during systole; about 70 percent of this portion flows out during the first third of the ejection period, with 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 are called 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 rapidly decrease back to their low diastolic levels. Then the A-V valves open to begin a new cycle of ventricular pumping.

End-Diastolic Volume, End-Systolic Volume, and Stroke Volume Output. During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 milliliters. This volume is called the *end-diastolic volume.* Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the *stroke volume output.* The remaining volume in each ventricle, about 40 to 50 milliliters, is called the *end-systolic volume.* The fraction of the end-diastolic volume that is ejected is called the *ejection fraction*—usually equal to about 0.6 (or 60 percent).

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

THE HEART VALVES PREVENT BACKFLOW OF BLOOD DURING SYSTOLE

Atrioventricular Valves. The *A-V valves* (i.e., the *tricuspid* and *mitral* valves) prevent backflow of blood from the ventricles to the atria during systole, and the *semilunar valves* (i.e., the *aortic* and *pulmonary artery* valves) prevent backflow from the aorta and pulmonary arteries into the ventricles during diastole. These valves, shown in **Figure 9-8** for the left ventricle, close and open *passively.* That is, they close when a backward pressure gradient pushes blood backward, and they open when a forward pressure gradient forces blood in the forward direction. For anatomical reasons, the thin, filmy A-V valves require almost no backflow to cause closure, whereas the much heavier semilunar valves require rather rapid backflow for a few milliseconds.

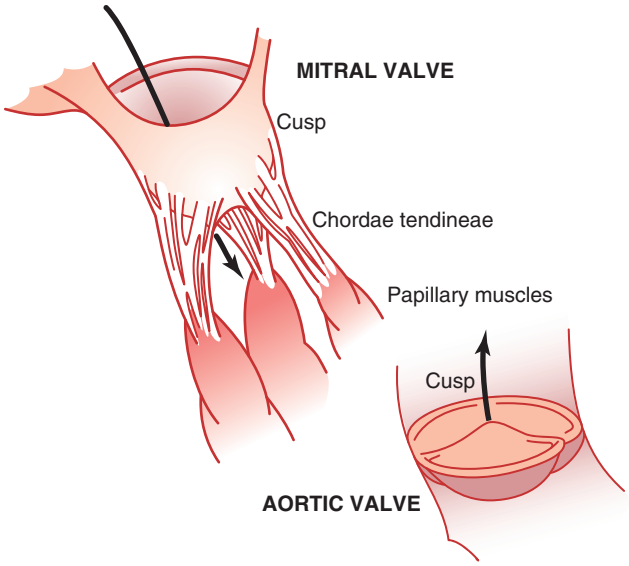


Figure 9-8. Mitral and aortic valves (the left ventricular valves).

Function of the Papillary Muscles. Figure 9-8 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 closed, 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 9-8) that they must be constructed with an especially strong yet very pliable fibrous tissue 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 during systole 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.

An *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 pitch is low 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, which 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. In certain abnormal conditions, however, 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 9-9 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.

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

During ventricular contraction, the systolic pressure increases even at low ventricular volumes and reaches a maximum at a ventricular volume of 150 to 170 milliliters. Then, as the volume increases still further, the systolic pressure actually decreases under some conditions, as demonstrated by the falling systolic pressure curve in **Figure 9-9**, 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.

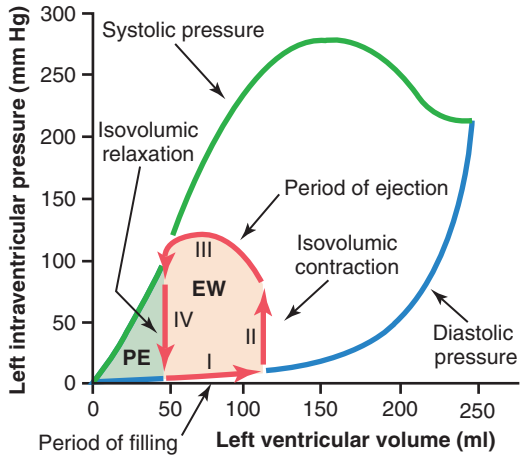


Figure 9-9. Relationship between left ventricular volume and intraventricular pressure during diastole and systole. Also shown by the red lines is the "volume-pressure diagram," demonstrating changes in intraventricular volume and pressure during the normal cardiac cycle. EW, net external work; PE, potential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

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 9-9** 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 9-10**. It is divided into four phases.

Phase I: Period of filling. Phase I in the volume-pressure diagram begins at a ventricular volume of about 50 milliliters and a diastolic pressure of 2 to 3 mm Hg. The amount of blood that remains in the ventricle after the previous heartbeat, 50 milliliters, 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 milliliters, called the *end-diastolic volume,* an increase of 70 milliliters. Therefore, the volume-pressure diagram during phase I extends along the line in **Figure 9-9** labeled "I," and from point A to point B in **Figure 9-10**, with the volume increasing to 120 milliliters 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 (**Figure 9-10**).

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.

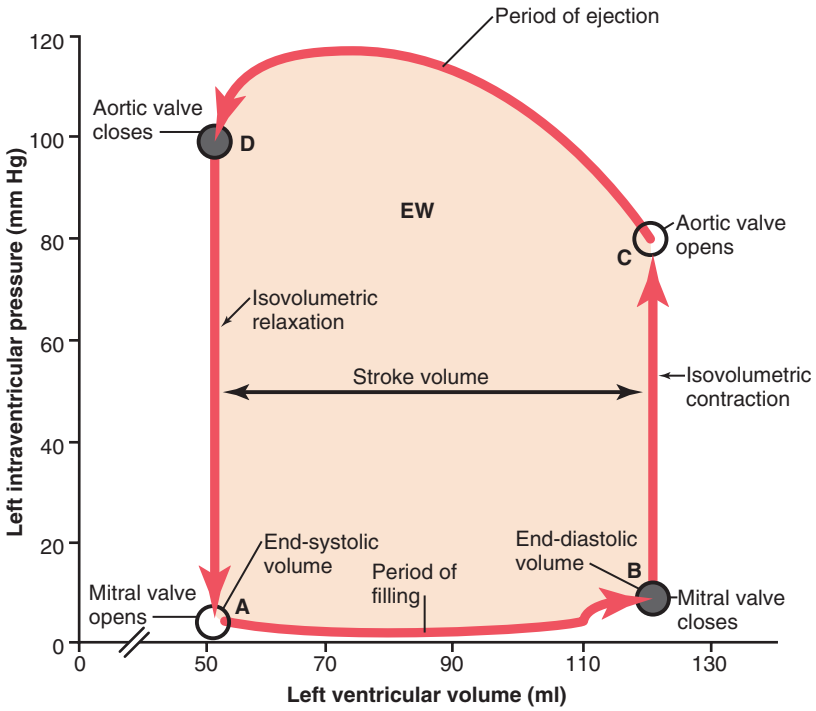


Figure 9-10. The volume-pressure diagram demonstrating changes in intraventricular volume and pressure during a single cardiac cycle *(red line)*. The shaded area represents the net external work (*EW*) output by the left ventricle during the cardiac cycle.

Therefore, in **Figure 9-9** 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; **Figure 9-10**), the aortic valve closes and the ventricular pressure falls back to the diastolic pressure level. The line labeled "IV" (**Figure 9-9**) traces this decrease in intraventricular pressure without any change in volume. Thus, the ventricle returns to its starting point, with about 50 milliliters of blood left in the ventricle and at an atrial pressure of 2 to 3 mm Hg.

The area subtended by this functional volume-pressure diagram (the 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.

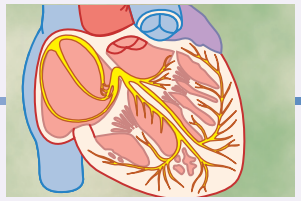
The *afterload* of the ventricle is the pressure in the aorta leading from the ventricle. In **Figure 9-9**, this corresponds to the systolic pressure described by the phase III curve of the volume-pressure diagram. (Sometimes the afterload is loosely considered to be the resistance in the circulation 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 altered from normal to a severe degree.

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 the energy is normally derived from oxidative metabolism of fatty acids, with the remaining 10 to 30 percent coming from other nutrients, principally 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 work. The different chemical reactions that liberate energy are discussed in Chapters 68 and 69.

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 9-9**. This shaded portion consists of the *external work* (EW) as explained earlier and an additional



CHAPTER 10

UNIT III

Rhythmical Excitation of the Heart

The human heart has a special system for rhythmic self-excitation and repetitive contraction approximately 100,000 times each day, or 3 billion times in the average human lifetime. This impressive feat is performed by a system that (1) generates rhythmical electrical impulses to initiate rhythmical contraction of the heart muscle and (2) conducts 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 the 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 capability 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 we attempt to explain the rhythmicity of the sinus nodal fibers, first recall from the discussions of Chapters 5 and 9 that cardiac muscle has three main 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) *L-type calcium channels (slow sodium-calcium channels)*, and (3) *potassium channels*.

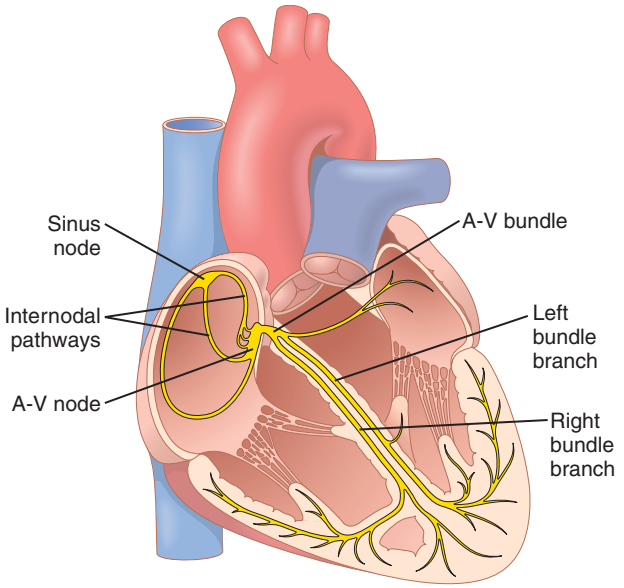


Figure 10-1. Sinus node and the Purkinje system of the heart, showing also the atrioventricular *(A-V)* node, atrial internodal pathways, and ventricular bundle branches.

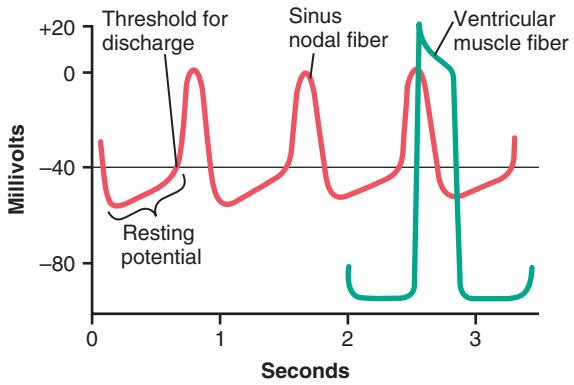


Figure 10-2. Rhythmical discharge of a sinus nodal fiber. Also, the sinus nodal action potential is compared with that of a ventricular muscle fiber.

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 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.

However, 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") 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 L-type 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? Two events occur during the course of the action potential to prevent such a constant state of depolarization. First, the L-type 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 L-type 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 process 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, which 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 throughout a person's life.

INTERNODAL AND INTERATRIAL PATHWAYS TRANSMIT CARDIAC IMPULSES 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 bands, 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.

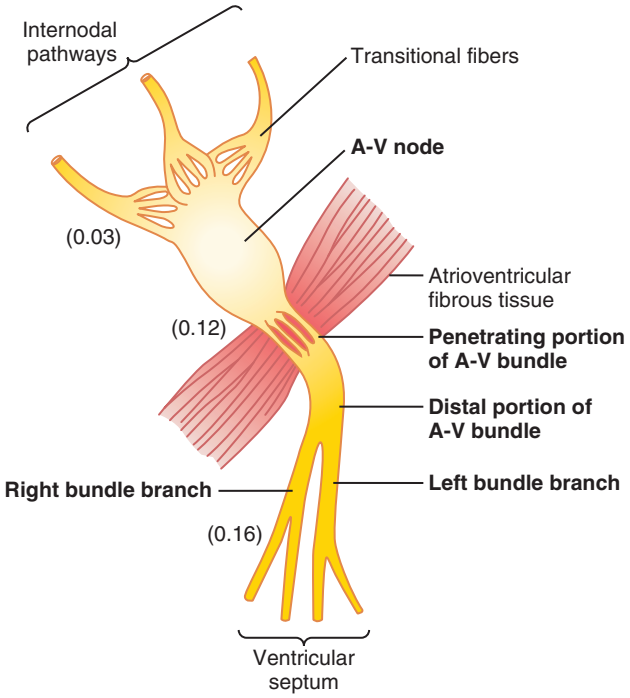


Figure 10-3. Organization of the atrioventricular *(A-V)* node. The numbers represent the interval of time from the origin of the impulse in the sinus node. The values have been extrapolated to human beings.

THE ATRIOVENTRICULAR NODE DELAYS 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**. **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. This figure also shows the approximate intervals of time in fractions of a second between the 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.

Thus, the total delay in the A-V nodal and A-V bundle system is about 0.13 second. This delay, 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 six

times that in the usual ventricular muscle and 150 times that in some of the A-V nodal fibers. This velocity 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 characteristic 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. (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 serious cardiac arrhythmias.)

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.

The total elapsed time averages only 0.03 second from the time the cardiac impulse enters the bundle branches in the ventricular septum until it reaches the terminations of the Purkinje fibers. 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 angulation, 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.

SUMMARY OF THE SPREAD OF THE CARDIAC IMPULSE THROUGH THE HEART

Figure 10-4 summarizes 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; 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

THE SINUS NODE IS THE NORMAL 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

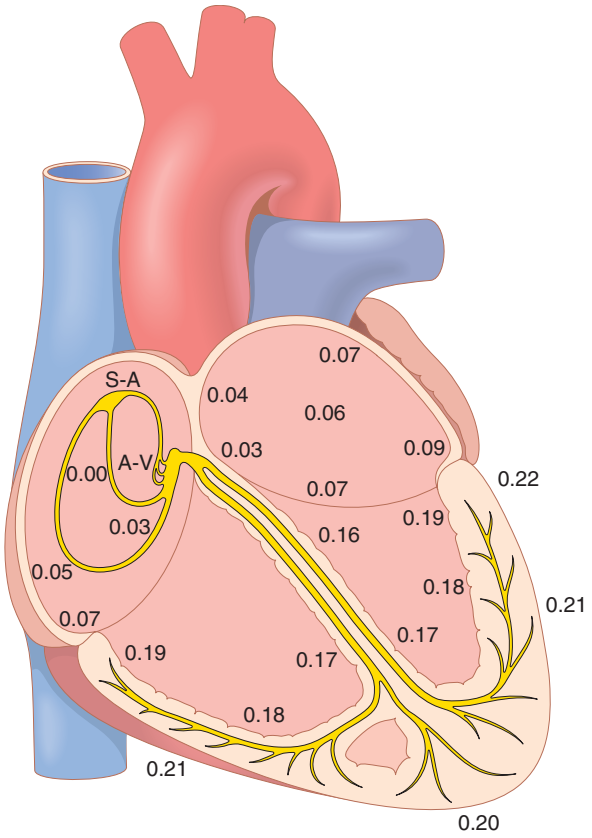


Figure 10-4. Transmission of the cardiac impulse through the heart, showing the time of appearance (in fractions of a second after initial appearance at the sinoatrial node) in different parts of the heart. A-V, atrioventricular; S-A, sinoatrial.

parts of the heart can also exhibit intrinsic rhythmical excitation in the same way that the sinus nodal fibers do; this capability 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. However, 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 sites.

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 almost 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 development 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.

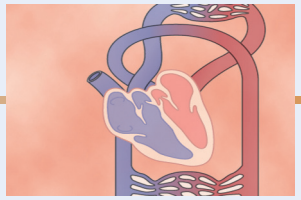
A pacemaker elsewhere than the sinus node is called an *"ectopic" pacemaker*. An ectopic pacemaker causes an abnormal sequence of contraction of the different parts of the heart and can cause significant debility of heart pumping.

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

The rapid conduction of the Purkinje system normally permits the cardiac impulse to arrive 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 timing 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.



CHAPTER 14

UNIT IV

Overview of the Circulation; Biophysics of Pressure, Flow, and Resistance

The function of the circulation is to serve the needs of the body tissues—to transport nutrients to the body tissues, to transport waste products away, to transport hormones from one part of the body to another and, in general, to maintain an appropriate environment in all the tissue fluids of the body for survival and optimal function of the cells.

The rate of blood flow through many tissues is controlled mainly in response to their need for nutrients. In some organs, such as the kidneys, the circulation serves additional functions. Blood flow to the kidney, for example, is far in excess of its metabolic requirements and is related to its excretory function, which requires that a large volume of blood be filtered each minute.

The heart and blood vessels, in turn, are controlled to provide the necessary cardiac output and arterial pressure to cause the needed tissue blood flow. What are the mechanisms for controlling blood volume and blood flow, and how does this process relate to the other functions of the circulation? These are some of the topics and questions that we discuss in this section on the circulation.

PHYSICAL CHARACTERISTICS OF THE CIRCULATION

The circulation, shown in **Figure 14-1**, is divided into the *systemic circulation* and the *pulmonary circulation.* Because the systemic circulation supplies blood flow to all the tissues of the body except the lungs, it is also called the *greater circulation* or *peripheral circulation.*

Functional Parts of the Circulation. Before discussing the details of circulatory function, it is important to understand the role of each part of the circulation.

The function of the *arteries* is to transport blood *under high pressure* to the tissues. For this reason, the arteries have strong vascular walls, and blood flows at a high velocity in the arteries.

The *arterioles* are the last small branches of the arterial system; they act as *control conduits* through which blood is released into the capillaries. Arterioles have strong muscular walls that can close the arterioles completely or can, by relaxing, dilate the vessels severalfold, thus having the capability of vastly altering blood flow in each tissue in response to its needs.

The function of the *capillaries* is to exchange fluid, nutrients, electrolytes, hormones, and other substances between the blood and the interstitial fluid. To serve this role, the capillary walls are thin and have numerous minute *capillary pores* permeable to water and other small molecular substances.

The *venules* collect blood from the capillaries and gradually coalesce into progressively larger veins.

The *veins* function as conduits for transport of blood from the venules back to the heart; equally important, they serve as a major reservoir of extra blood. Because the pressure in the venous system is very low, the venous walls are thin. Even so, they are muscular enough to contract or expand and thereby serve as a controllable reservoir for the extra blood, either a small or a large amount, depending on the needs of the circulation.

Volumes of Blood in the Different Parts of the Circulation. **Figure 14-1** gives an overview of the circulation and the percentage of the total blood volume in major segments of the circulation. For instance, about 84 percent of the entire blood volume of the body is in the systemic circulation and 16 percent in the heart and lungs. Of the 84 percent in the systemic circulation, approximately 64 percent is in the veins, 13 percent is in the arteries, and 7 percent in the systemic arterioles and capillaries. The heart contains 7 percent of the blood, and the pulmonary vessels, 9 percent.

Most surprising is how little blood volume in the capillaries. It is here, however, that the most important function of the circulation occurs—diffusion of substances back and forth between the blood and the tissues. This function is discussed in detail in Chapter 16.

Cross-Sectional Areas and Velocities of Blood Flow. If all the *systemic vessels* of each type were put side by side, their approximate total cross-sectional areas for the average human being would be as follows:

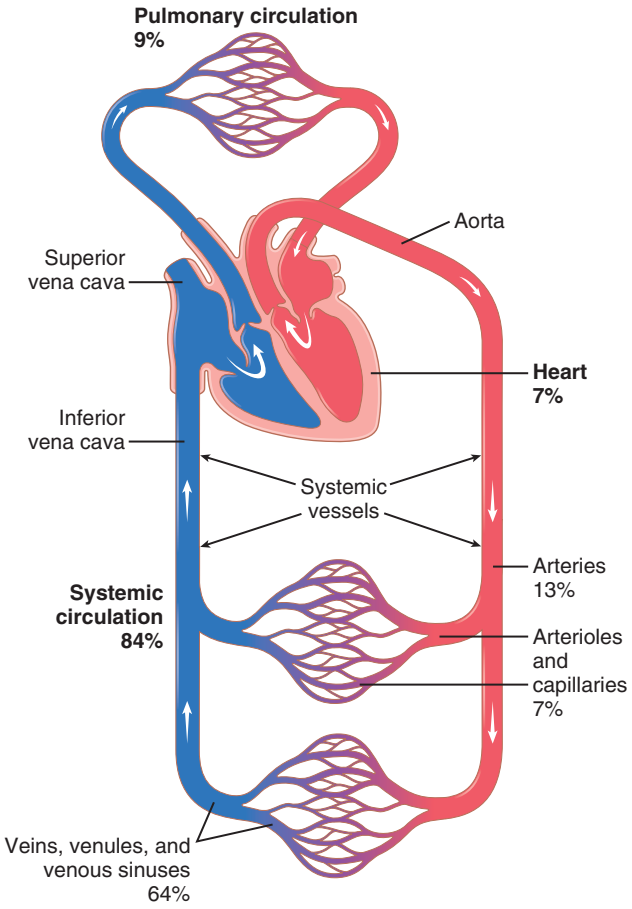


Figure 14-1. Distribution of blood (in percentage of total blood) in the different parts of the circulatory system.

Vessel	Cross-Sectional Area (cm^2)
Aorta	2.5
Small arteries	20
Arterioles	40
Capillaries	2500
Venules	250
Small veins	80
Venae cavae	8

Note particularly that the cross-sectional areas of the veins are much larger than those of the arteries, averaging about 4 times those of the corresponding arteries. This difference means the large blood storage capacity of the venous system in comparison with the arterial system.

Because the entire volume of blood flow (F) must pass through each segment of the circulation each minute, the velocity of blood flow (v) is inversely proportional to vascular cross-sectional area (A):

$$v = \frac{F}{A}$$

Thus, under resting conditions, the velocity averages about 33 cm/sec in the aorta but is only 1/1000 as rapid in the capillaries—about 0.3 mm/sec. However, because the capillaries have a typical length of only 0.3 to 1 mm, the blood remains in the capillaries for only 1 to 3 seconds, which is surprising because diffusion of nutrient and waste substances and electrolytes that occurs through the capillary walls must be performed in this short time.

Pressures in the Various Portions of the Circulation. Because the heart pumps blood continually into the aorta, the mean pressure in the aorta is high, averaging about 100 mm Hg. Also, because heart pumping is pulsatile, the arterial pressure alternates between a *systolic pressure level* of 120 mm Hg and a *diastolic pressure level* of 80 mm Hg, as shown on the left side of Figure 14-2.

As the blood flows through the *systemic circulation,* its mean pressure falls progressively to about 0 mm Hg by the time it reaches the termination of the superior and inferior venae cavae where they empty into the right atrium of the heart.

The pressure in the systemic capillaries varies from as high as 35 mm Hg near the arteriolar ends to as low as 10 mm Hg near the venous ends, but their average "functional" pressure in most vascular beds is about 17 mm Hg, a pressure high enough that little of the plasma leaks through the capillary *pores* of the capillary walls, even though nutrients *diffuse* easily through these same pores to the outlying tissue cells.

Note at the far right of Figure 14-2 the respective pressures in the different parts of the *pulmonary circulation.* In the pulmonary artery the pressure is pulsatile, just as in the aorta, but the pressures are far less: *pulmonary artery systolic pressure* averages only 25 mm Hg and *diastolic pressure* averages about 8 mm Hg, with a mean pulmonary arterial pressure of only 16 mm Hg. The mean pulmonary capillary pressure averages only 7 mm Hg. Yet, the total blood flow through the lungs each minute is the same as through the systemic circulation. The low pressures of the pulmonary system are in accord with the needs of the lungs because all that is required is to expose the blood in the pulmonary capillaries to oxygen and other gases in the pulmonary alveoli.

BASIC PRINCIPLES OF CIRCULATORY FUNCTION

Although the details of circulatory function are complex, three basic principles underlie all functions of the system.

1. *Blood flow to most tissues is controlled according to the tissue need.* When tissues are active, they need a greatly increased supply of nutrients and therefore much more blood flow than when at rest—occasionally as much as 20 to 30 times the resting level. Yet, the heart normally cannot increase its cardiac output more than four to seven times greater than resting levels. Therefore, it is not possible simply to increase blood flow everywhere in the body when a particular tissue demands increased flow. Instead, the microvessels of each

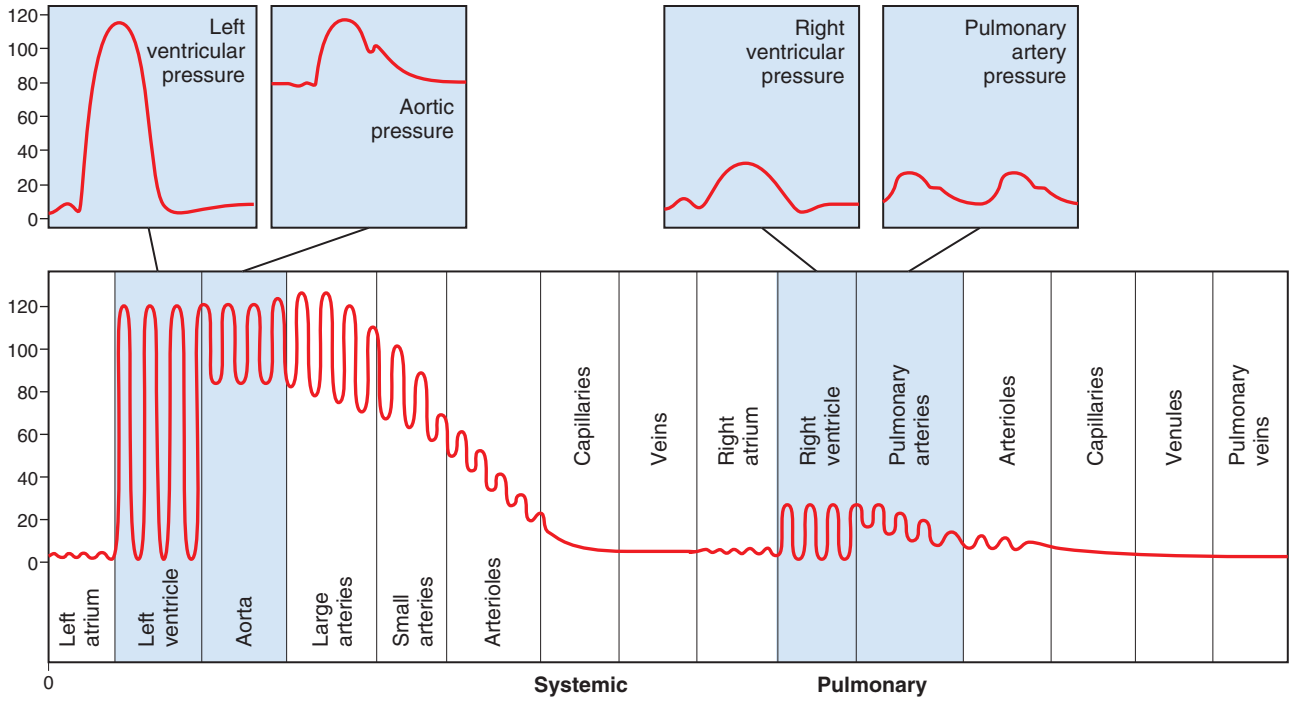


Figure 14-2. Normal blood pressures in the different portions of the circulatory system when a person is lying in the horizontal position.

tissue continuously monitor tissue needs, such as the availability of oxygen and other nutrients and the accumulation of carbon dioxide and other tissue waste products, and these microvessels in turn act directly on the local blood vessels, dilating or constricting them, to control local blood flow precisely to that level required for the tissue activity. Also, nervous control of the circulation from the central nervous system and hormones provide additional help in controlling tissue blood flow.

2. *Cardiac output is the sum of all the local tissue flows.* When blood flows through a tissue, it immediately returns by way of the veins to the heart. The heart responds automatically to this increased inflow of blood by pumping it immediately back into the arteries. Thus, the heart acts as an automaton, responding to the demands of the tissues. The heart, however, often needs help in the form of special nerve signals to make it pump the required amounts of blood flow.
3. *Arterial pressure regulation is generally independent of either local blood flow control or cardiac output control.* The circulatory system is provided with an extensive system for controlling the arterial blood pressure. For instance, if at any time the pressure falls significantly below the normal level of about 100 mm Hg, within seconds a barrage of nervous reflexes elicits a series of circulatory changes to raise the pressure back toward normal. The nervous signals especially (a) increase the force of heart pumping, (b) cause contraction of the large venous reservoirs to provide more blood to the heart, and (c) cause generalized constriction of the arterioles in many tissues so that more blood accumulates in the large arteries to increase the arterial pressure. Then, over more prolonged periods—hours and days—the kidneys play an additional major role in pressure control both by secreting pressure-controlling hormones and by regulating the blood volume.

Thus, the needs of the individual tissues are served specifically by the circulation. In the remainder of this chapter, we begin to discuss the basic details of the management of tissue blood flow and control of cardiac output and arterial pressure.

INTERRELATIONSHIPS OF PRESSURE, FLOW, AND RESISTANCE

Blood [illegible] through a blood vessel is dete[illegible]ed by two fac[illegible]) *pressure difference* of the bl[illegible]etween the two en[illegible]he vessel, also sometim[illegible]ed "pressure gradient[illegible] the vessel, whic[illegible]es the blood through the vess[illegible] (2) the imp[illegible]nt to blood flow through the vessel, [illegible] is c[illegible] *ascular resistance.* **Figure 14-3** demonst[illegible]elationships, showing a blood vessel segment [illegible]anywhere in the circulatory system.

P_1 represents the [illegible]re at [illegible]gin of the vessel; at the other end, th[illegible]sure is P_2. [illegible]nce occurs as a result of frict[illegible]ween the flowing [illegible]nd the intravascular e[illegible]um all along the insi[illegible]he vessel. The flo[illegible]gh the vessel can be calcula[illegible]he following [illegible]ula, which is called *Ohm's law*:

$$F = \frac{\Delta P}{R}$$

$$Re = \frac{v \cdot d \cdot \rho}{\eta}$$

where *Re* is *Reynolds' number* and is the measure of the tendency for turbulence to occur, v is the mean velocity of blood flow (in centimeters/second), d is the vessel diameter (in centimeters), ρ is density, and η is the viscosity (in poise). The viscosity of blood is normally about 1/30 p[illegible] and the density is only slightly gre[illegible]han 1. When [illegible]lds' number rises above 200 to [illegible]rbulent flow will [illegible]t some branches of ve[illegible]ut will die out along th[illegible]th portions of th[illegible]els. However, when Reynolds [illegible]er rises abo[illegible]oximately 2000, turbulence will us[illegible]ccur e[illegible]a straight, smooth vessel.

Reynolds' number fo[illegible] the vascular system normally rises to 200 to 4[illegible]rge arteries; as a result there is almost alw[illegible]me [illegible]ence of flow at the branches of thes[illegible]s. In the p[illegible]l portions of the aorta and pul[illegible]y artery, Reyno[illegible]mber can rise to several t[illegible]d during the rapid pi[illegible]ejection by the ven[illegible] which causes considerabl[illegible]lence in the p[illegible]l aorta and pulmonary artery [illegible] many conditions are appropriate for turbulence: (1) high velocity of blood flow, (2) pulsatile nature of the flow, (3) sudden change in vessel diameter, and (4) large vessel diameter. However, in small vessels, Reynolds' number is almost never high enough to cause turbulence.

BLOOD PRESSURE

Standard Units of Pressure. Blood pressure almost always is measured in millimeters of mercury (mm Hg) because the mercury manometer has been used as the standard reference for measuring pressure since its invention in 1846 by Poiseuille. Actually, blood pressure means the *force exerted by the blood against any unit area of the vessel wall*. When one says that the pressure in a vessel is 50 mm Hg, this means that the force exerted is sufficient to push a column of mercury against gravity up to a level 50 millimeters high. If the pressure is 100 mm Hg, it will push the column of mercury up to 100 millimeters.

Occasionally, pressure is measured in *centimeters of water (cm H_2O)*. A pressure of 10 cm H_2O means a pressure sufficient to raise a column of water against gravity to a height of 10 centimeters. *One millimeter of mercury pressure equals 1.36 centimeters of water pressure* because the specific gravity of mercury is 13.6 times that of water, and 1 centimeter is 10 times as great as 1 millimeter.

High-Fidelity Methods for Measuring Blood Pressure. The mercury in a manometer has so much inertia that it cannot rise and fall rapidly. For this reason, the mercury manometer, although excellent for recording steady pressures, cannot respond to pressure changes that occur more rapidly than about one cycle every 2 to 3 seconds.

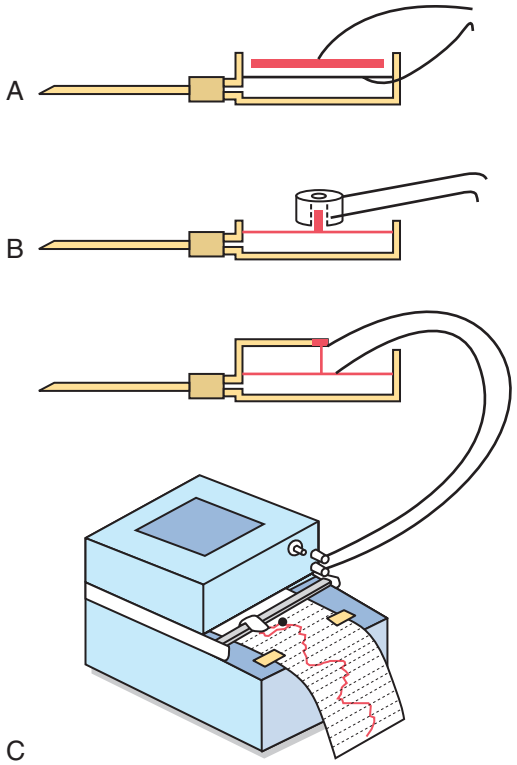


Figure 14-7. Principles of three types of electronic transducers for recording rapidly changing blood pressures (explained in the text).

Whenever it is desired to record rapidly changing pressures, some other type of pressure recorder is necessary. **Figure 14-7** demonstrates the basic principles of three electronic pressure *transducers* commonly used for converting blood pressure and/or rapid changes in pressure into electrical signals and then recording the electrical signals on a high-speed electrical recorder. Each of these transducers uses a very thin, highly stretched metal membrane that forms one wall of the fluid chamber. The fluid chamber in turn is connected through a needle or catheter inserted into the blood vessel in which the pressure is to be measured. When the pressure is high, the membrane bulges slightly, and when it is low, it returns toward its resting position.

In **Figure 14-7*A*,** a simple metal plate is placed a few hundredths of a centimeter above the membrane. When the membrane bulges, the membrane comes closer to the plate, which increases the *electrical capacitance* between these two, and this change in capacitance can be recorded using an appropriate electronic system.

In **Figure 14-7*B***, a small iron slug rests on the membrane, and this slug can be displaced upward into a center space inside an electrical wire coil. Movement of the iron into the coil increases the *inductance* of the coil, and this, too, can be recorded electronically.

Finally, in **Figure 14-7*C***, a very thin, stretched resistance wire is connected to the membrane. When this wire is stretched greatly, its resistance increases; when it is stretched less, its resistance decreases. These changes, too, can be recorded by an electronic system.

is spent in diastole than is systole; thus, the arterial pressure remains nearer to diastolic pressure than to systolic pressure during the greater part of the cardiac cycle. The mean arterial pressure is therefore determined about 60 percent by the diastolic pressure and 40 percent by the systolic pressure. Note in **Figure 15-8** that the mean pressure *(solid green line)* at all ages is nearer to the diastolic pressure than to the systolic pressure. However, at very high heart rates, diastole comprises a smaller fraction of the cardiac cycle and the mean arterial pressure is more closely approximated as the average of systolic and diastolic pressures.

VEINS AND THEIR FUNCTIONS

The veins provide passageways for flow of blood to the heart, but they also perform other special functions that are necessary for operation of the circulation. Of special importance is that they are capable of constricting and enlarging and thereby storing either small or large quantities of blood and making this blood available when it is required by the remainder of the circulation. The peripheral veins can also propel blood forward by means of a so-called *venous pump,* and they even help to regulate cardiac output, an exceedingly important function that is described in detail in Chapter 20.

VENOUS PRESSURES—RIGHT ATRIAL PRESSURE (CENTRAL VENOUS PRESSURE) AND PERIPHERAL VENOUS PRESSURES

To understand the various functions of the veins, it is first necessary to know something about pressure in the veins and what determines the pressure.

Blood from all the systemic veins flows into the right atrium of the heart; therefore, the pressure in the right atrium is called the *central venous pressure.*

Right atrial pressure is regulated by a balance between (1) the ability of the heart to pump blood out of the right atrium and ventricle into the lungs and (2) the tendency for blood to flow from the peripheral veins into the right atrium. If the right heart is pumping strongly, the right atrial pressure decreases. Conversely, weakness of the heart elevates the right atrial pressure. Also, any effect that causes rapid inflow of blood into the right atrium from the peripheral veins elevates the right atrial pressure. Some of the factors that can increase this venous return and thereby increase the right atrial pressure are (1) increased blood volume, (2) increased large vessel tone throughout the body with resultant increased peripheral venous pressures, and (3) dilation of the arterioles, which decreases the peripheral resistance and allows rapid flow of blood from the arteries into the veins.

The same factors that regulate right atrial pressure also contribute to regulation of cardiac output because the amount of blood pumped by the heart depends on both the ability of the heart to pump and the tendency for blood to flow into the heart from the peripheral vessels. Therefore, we discuss regulation of right atrial pressure in much more depth in Chapter 20 in connection with regulation of cardiac output.

The *normal right atrial pressure* is about 0 mm Hg, which is equal to the atmospheric pressure around the body. It can increase to 20 to 30 mm Hg under very abnormal conditions, such as (1) serious heart failure or (2) after massive transfusion of blood, which greatly increases the total blood volume and causes excessive quantities of blood to attempt to flow into the heart from the peripheral vessels.

The lower limit to the right atrial pressure is usually about –3 to –5 mm Hg below atmospheric pressure, which is also the pressure in the chest cavity that surrounds the heart. The right atrial pressure approaches these low values when the heart pumps with exceptional vigor or when blood flow into the heart from the peripheral vessels is greatly depressed, such as after severe hemorrhage.

Venous Resistance and Peripheral Venous Pressure

Large veins have so little resistance to blood flow *when they are distended* that the resistance then is almost zero and is of almost no importance. However, as shown in **Figure 15-9**, most of the large veins that enter the thorax are compressed at many points by the surrounding tissues so that blood flow is impeded at these points. For instance, the veins from the arms are compressed by their sharp angulations over the first rib. Also, the pressure in the neck veins often falls so low that the atmospheric pressure on the outside of the neck causes these veins to collapse. Finally, veins coursing through the abdomen are often compressed by different organs and by the intra-abdominal

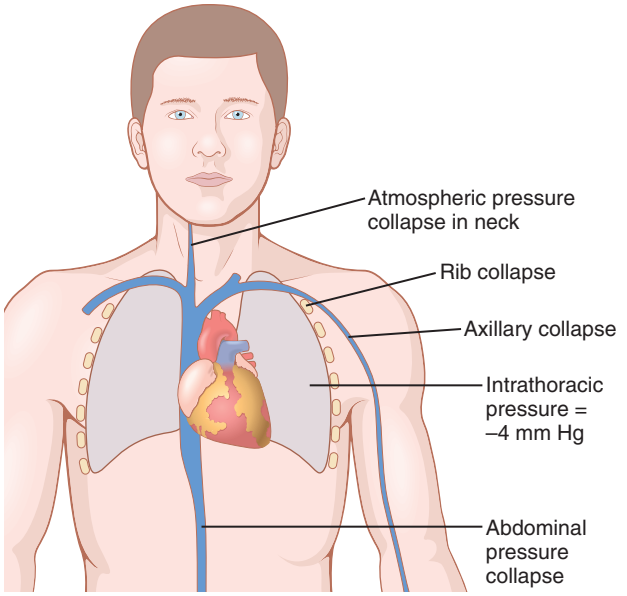


Figure 15-9. Compression points that tend to collapse the veins entering the thorax.

pressure, so they usually are at least partially collapsed to an ovoid or slit-like state. For these reasons, the *large veins do usually offer some resistance to blood flow,* and thus the pressure in the more peripheral small veins in a person lying down is usually +4 to +6 mm Hg greater than the right atrial pressure.

Effect of High Right Atrial Pressure on Peripheral Venous Pressure. When the right atrial pressure rises above its normal value of 0 mm Hg, blood begins to back up in the large veins. This backup of blood enlarges the veins, and even the collapse points in the veins open up when the right atrial pressure rises above +4 to +6 mm Hg. Then, as the right atrial pressure rises still further, the additional increase causes a corresponding rise in peripheral venous pressure in the limbs and elsewhere. Because the heart must be weakened significantly to cause a rise in right atrial pressure as high as +4 to +6 mm Hg, the peripheral venous pressure is not noticeably elevated even in the early stages of heart failure as long as the person is at rest.

Effect of Intra-abdominal Pressure on Venous Pressures of the Leg. The pressure in the abdominal cavity of a recumbent person normally averages about +6 mm Hg, but it can rise to +15 to +30 mm Hg as a result of pregnancy, large tumors, abdominal obesity, or excessive fluid (called "ascites") in the abdominal cavity. When the intra-abdominal pressure rises, the pressure in the veins of the legs must rise *above* the abdominal pressure before the abdominal veins will open and allow the blood to flow from the legs to the heart. Thus, if the intra-abdominal pressure is +20 mm Hg, the lowest possible pressure in the femoral veins is also about +20 mm Hg.

Effect of Gravitational Pressure on Venous Pressure

In any body of water that is exposed to air, the pressure at the surface of the water is equal to atmospheric pressure, but the pressure rises 1 mm Hg for each 13.6 millimeters of distance below the surface. This pressure results from the weight of the water and therefore is called *gravitational pressure* or *hydrostatic pressure.*

Gravitational pressure also occurs in the vascular system of the human being because of weight of the blood in the vessels, as shown in **Figure 15-10**. When a person is standing, the pressure in the right atrium remains about 0 mm Hg because the heart pumps into the arteries any excess blood that attempts to accumulate at this point. However, in an adult *who is standing absolutely still,* the pressure in the veins of the feet is about +90 mm Hg simply because of the gravitational weight of the blood in the veins between the heart and the feet. The venous pressures at other levels of the body are proportionately between 0 and 90 mm Hg.

In the arm veins, the pressure at the level of the top rib is usually about +6 mm Hg because of compression

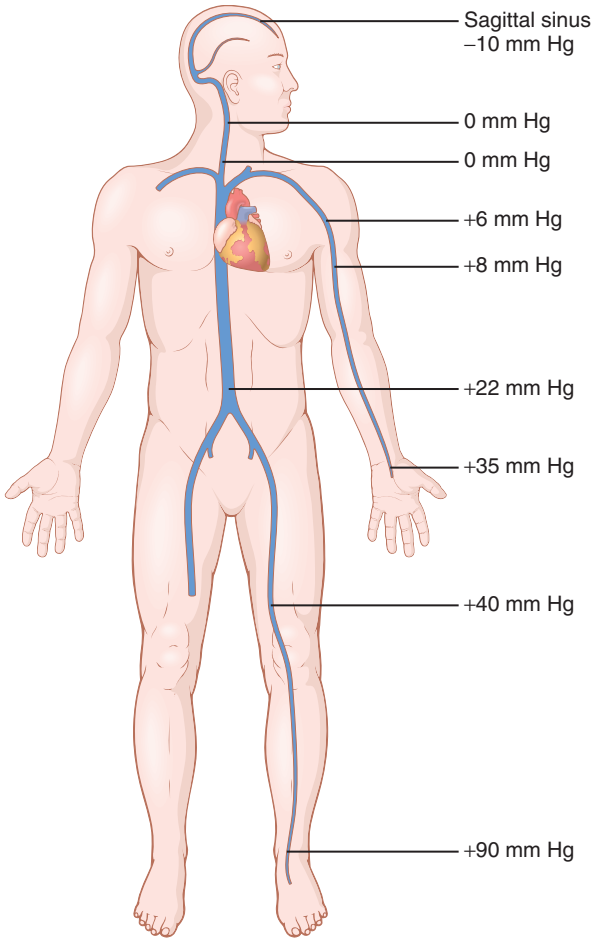


Figure 15-10. Effect of gravitational pressure on the venous pressures throughout the body in the standing person.

of the subclavian vein as it passes over this rib. The gravitational pressure down the length of the arm then is determined by the distance below the level of this rib. Thus, if the gravitational difference between the level of the rib and the hand is +29 mm Hg, this gravitational pressure is added to the +6 mm Hg pressure caused by compression of the vein as it crosses the rib, making a total of +35 mm Hg pressure in the veins of the hand.

The neck veins of a person standing upright collapse almost completely all the way to the skull because of atmospheric pressure on the outside of the neck. This collapse causes the pressure in these veins to remain at zero along their entire extent. Any tendency for the pressure to rise above this level opens the veins and allows the pressure to fall back to zero because of flow of the blood. Conversely, any tendency for the neck vein pressure to fall below zero collapses the veins still more, which further increases their resistance and again returns the pressure back to zero.

The veins inside the skull, on the other hand, are in a noncollapsible chamber (the skull cavity) and thus they cannot collapse. Consequently, *negative pressure can exist in the dural sinuses of the head;* in the standing position, the venous pressure in the sagittal sinus at the top of the

brain is about −10 mm Hg because of the hydrostatic "suction" between the top of the skull and the base of the skull. Therefore, if the sagittal sinus is opened during surgery, air can be sucked immediately into the venous system; the air may even pass downward to cause air embolism in the heart and death.

Effect of the Gravitational Factor on Arterial and Other Pressures. The gravitational factor also affects pressures in the peripheral arteries and capillaries. For instance, a standing person who has a mean arterial pressure of 100 mm Hg at the level of the heart has an arterial pressure in the feet of about 190 mm Hg. Therefore, when one states that the arterial pressure is 100 mm Hg, this statement generally means that 100 mm Hg is the pressure at the gravitational level of the heart but not necessarily elsewhere in the arterial vessels.

Venous Valves and the "Venous Pump": Their Effects on Venous Pressure

Were it not for valves in the veins, the gravitational pressure effect would cause the venous pressure in the feet always to be about +90 mm Hg in a standing adult. However, every time one moves the legs, one tightens the muscles and compresses the veins in or adjacent to the muscles, which squeezes the blood out of the veins. However, the valves in the veins, shown in **Figure 15-11**, are arranged so that the direction of venous blood flow can be only toward the heart. Consequently, every time a person moves the legs or even tenses the leg muscles, a certain amount of venous blood is propelled toward the heart. This pumping system is known as the "venous pump" or "muscle pump," and it is efficient enough that under ordinary circumstances the venous pressure in the feet of a walking adult remains less than +20 mm Hg.

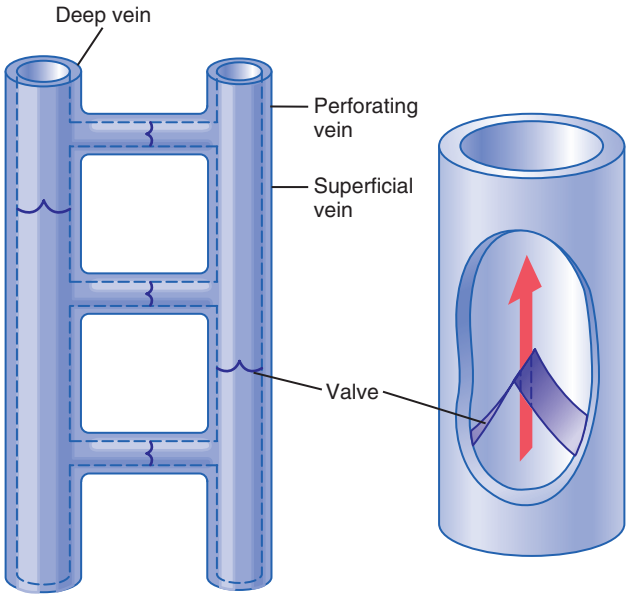


Figure 15-11. Venous valves of the leg.

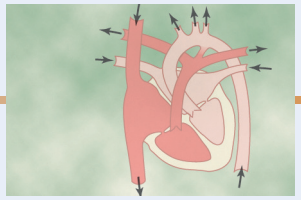
If a person stands perfectly still, the venous pump does not work, and the venous pressures in the lower legs increase to the full gravitational value of 90 mm Hg in about 30 seconds. The pressures in the capillaries also increase greatly, causing fluid to leak from the circulatory system into the tissue spaces. As a result, the legs swell and the blood volume diminishes. Indeed, 10 to 20 percent of the blood volume can be lost from the circulatory system within the 15 to 30 minutes of standing absolutely still, which may lead to fainting as sometimes occurs when a soldier is made to stand at rigid attention. This situation can be avoided by simply flexing the leg muscles periodically and slightly bending the knees, thus permitting the venous pump to work.

Venous Valve Incompetence Causes "Varicose" Veins. The valves of the venous system may become "incompetent" or even be destroyed when the veins have been overstretched by excess venous pressure lasting weeks or months, which can occur in pregnancy or when one stands most of the time. Stretching the veins increases their cross-sectional areas, but the leaflets of the valves do not increase in size. Therefore, the leaflets of the valves no longer close completely. When this lack of complete closure occurs, the pressure in the veins of the legs increases greatly because of failure of the venous pump, which further increases the sizes of the veins and finally destroys the function of the valves entirely. Thus, the person develops "varicose veins," which are characterized by large, bulbous protrusions of the veins beneath the skin of the entire leg, particularly the lower leg.

Whenever people with varicose veins stand for more than a few minutes, the venous and capillary pressures become very high and leakage of fluid from the capillaries causes constant edema in the legs. The edema in turn prevents adequate diffusion of nutritional materials from the capillaries to the muscle and skin cells, so the muscles become painful and weak and the skin may even become gangrenous and ulcerate. The best treatment for such a condition is continual elevation of the legs to a level at least as high as the heart. Tight binders or long "compression" stockings on the legs also can be of considerable assistance in preventing the edema and its sequelae.

Clinical Estimation of Venous Pressure. Venous pressure often can be estimated by simply observing the degree of distention of the peripheral veins—especially of the neck veins. For instance, in the sitting position, the neck veins are never distended in the normal quietly resting person. However, when the right atrial pressure becomes increased to as much as +10 mm Hg, the lower veins of the neck begin to protrude, and at +15 mm Hg atrial pressure, essentially all the veins in the neck become distended.

Direct Measurement of Venous Pressure and Right Atrial Pressure. Venous pressure can also be measured



CHAPTER 23

UNIT IV

Heart Valves and Heart Sounds; Valvular and Congenital Heart Defects

Function of the heart valves was discussed in Chapter 9, where we pointed out that *closing* of the valves causes audible sounds. Ordinarily, no audible sounds occur when the valves open. In this chapter, we first discuss the factors that cause the sounds in the heart under normal and abnormal conditions. Then we discuss the overall circulatory changes that occur when valvular or congenital heart defects are present.

HEART SOUNDS

NORMAL HEART SOUNDS

When listening to a normal heart with a stethoscope, one hears a sound usually described as "lub, dub, lub, dub." The "lub" is associated with closure of the atrioventricular (A-V) valves at the beginning of systole, and the "dub" is associated with closure of the semilunar (aortic and pulmonary) valves at the end of systole. The "lub" sound is called the *first heart sound,* and the "dub" is called the *second heart sound,* because the normal pumping cycle of the heart is considered to start when the A-V valves close at the onset of ventricular systole.

The First Heart Sound Is Associated with Closure of the A-V Valves. The earliest explanation for the cause of the heart sounds was that the "slapping" together of the valve leaflets sets up vibrations. However, this closure of the valve leaflets has been shown to cause little, if any, of the sound, because the blood between the leaflets cushions the slapping effect and prevents significant sound. Instead, the cause is *vibration of the taut valves immediately after closure,* along with *vibration of the adjacent walls of the heart and major vessels around the heart.* That is, in generating the first heart sound, contraction of the ventricles first causes sudden backflow of blood against the A-V valves (the tricuspid and mitral valves), causing them to close and bulge toward the atria until the chordae tendineae abruptly stop the back bulging. The elastic tautness of the chordae tendineae and of the valves then causes the back-surging blood to bounce forward again into each respective ventricle. This mechanism causes the blood and the ventricular walls, as well as the taut valves, to vibrate and causes vibrating turbulence in the blood. The vibrations travel through the adjacent tissues to the chest wall, where they can be heard as sound by using the stethoscope.

The Second Heart Sound Is Associated with Closure of the Aortic and Pulmonary Valves. The second heart sound results from sudden closure of the semilunar valves (i.e., the aortic and pulmonary valves) at the end of systole. When the semilunar valves close, they bulge backward toward the ventricles and their elastic stretch recoils the blood back into the arteries, which causes a short period of reverberation of blood back and forth between the walls of the arteries and the semilunar valves, as well as between these valves and the ventricular walls. The vibrations occurring in the arterial walls are then transmitted mainly along the arteries. When the vibrations of the vessels or ventricles come into contact with a "sounding board," such as the chest wall, they create sound that can be heard.

Duration and Pitch of the First and Second Heart Sounds. The duration of each of the heart sounds is slightly more than 0.10 second, with the first sound about 0.14 second and the second about 0.11 second. The reason for the shorter second sound is that the semilunar valves are more taut than the A-V valves, so they vibrate for a shorter time than do the A-V valves.

The audible range of frequency (pitch) in the first and second heart sounds, as shown in **Figure 23-1**, begins at the lowest frequency the ear can detect, about 40 cycles/sec, and goes up above 500 cycles/sec. When a special electronic apparatus is used to record these sounds, by far a larger proportion of the recorded sound is at frequencies and sound levels below the audible range, going down to 3 to 4 cycles/sec and peaking at about 20 cycles/sec, as illustrated by the lower shaded area in **Figure 23-1**. For this reason, major portions of the heart sounds can be recorded electronically in phonocardiograms even though they cannot be heard with a stethoscope.

The second heart sound normally has a higher frequency than the first heart sound for two reasons: (1) the tautness of the semilunar valves in comparison with the much less taut A-V valves, and (2) the greater elastic coefficient of the taut arterial walls that provide the principal

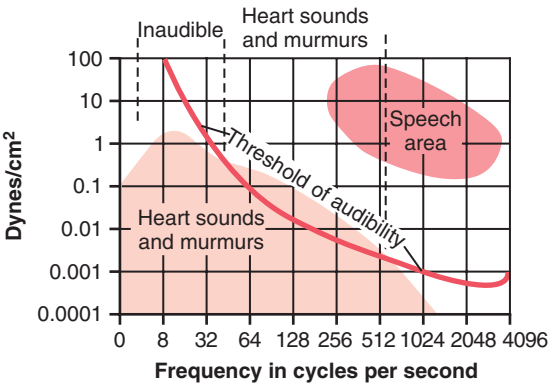


Figure 23-1. Amplitude of different-frequency vibrations in the heart sounds and heart murmurs in relation to the threshold of audibility, showing that the range of sounds that can be heard is between 40 and 520 cycles/sec. *(Modified from Butterworth JS, Chassin JL, McGrath JJ: Cardiac Auscultation, 2nd ed. New York: Grune & Stratton, 1960.)*

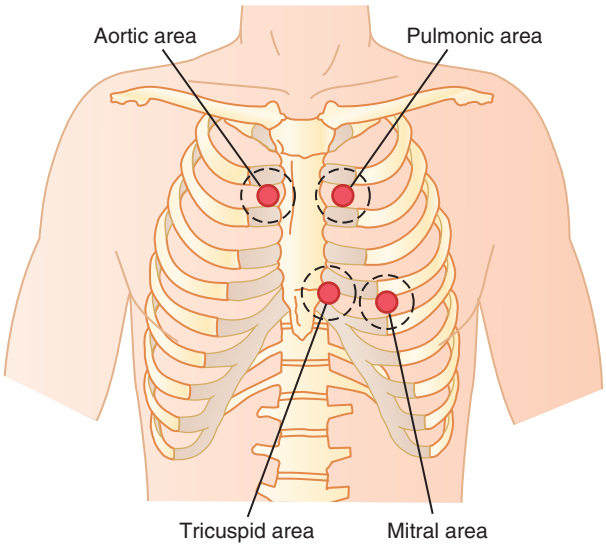


Figure 23-2. Chest areas from which sound from each valve is best heard.

vibrating chambers for the second sound, in comparison with the much looser, less elastic ventricular chambers that provide the vibrating system for the first heart sound. The clinician uses these differences to distinguish special characteristics of the two respective sounds.

The Third Heart Sound Occurs at the Beginning of the Middle Third of Diastole. Occasionally a weak, rumbling third heart sound is heard at the beginning of the *middle third of diastole.* A logical but unproved explanation of this sound is oscillation of blood back and forth between the walls of the ventricles initiated by inrushing blood from the atria. This occurrence is analogous to running water from a faucet into a paper sack, with the inrushing water reverberating back and forth between the walls of the sack to cause vibrations in its walls. The reason the third heart sound does not occur until the middle third of diastole is believed to be that in the early part of diastole, the ventricles are not filled sufficiently to create even the small amount of elastic tension necessary for reverberation. The frequency of this sound is usually so low that the ear cannot hear it, yet it can often be recorded in the phonocardiogram. The third heart sound may be normally present in children, adolescents, and young adults but generally indicates systolic heart failure in older adults.

Atrial Contraction Sound (Fourth Heart Sound). An atrial heart sound can sometimes be recorded in the phonocardiogram, but it can almost never be heard with a stethoscope because of its weakness and very low frequency—usually 20 cycles/sec or less. This sound occurs when the atria contract, and presumably, it is caused by the inrush of blood into the ventricles, which initiates vibrations similar to those of the third heart sound. A fourth heart sound is common in persons who derive benefit from atrial contraction for ventricular filling as a result of decreased ventricular wall compliance and increased resistance to ventricular filling. For example, a fourth heart sound is often heard in older patients with left ventricular hypertrophy.

Chest Surface Areas for Auscultation of Normal Heart Sounds. Listening to the sounds of the body, usually with the aid of a stethoscope, is called *auscultation.* **Figure 23-2** shows the areas of the chest wall from which the different heart valvular sounds can best be distinguished. Although the sounds from all the valves can be heard from all these areas, the cardiologist distinguishes the sounds from the different valves by a process of elimination. That is, he or she moves the stethoscope from one area to another, noting the loudness of the sounds in different areas and gradually picking out the sound components from each valve.

The areas for listening to the different heart sounds are not directly over the valves themselves. The aortic area is upward along the aorta because of sound transmission up the aorta, and the pulmonic area is upward along the pulmonary artery. The tricuspid area is over the right ventricle, and the mitral area is over the apex of the left ventricle, which is the portion of the heart nearest the surface of the chest; the heart is rotated so that the remainder of the left ventricle lies more posteriorly.

Phonocardiogram. If a microphone specially designed to detect low-frequency sound is placed on the chest, the heart sounds can be amplified and recorded by a high-speed recording apparatus. The recording is called a *phonocardiogram,* and the heart sounds appear as waves, as shown schematically in **Figure 23-3**. **Recording A** is an example of normal heart sounds, showing the vibrations of the first, second, and third heart sounds and even the very weak atrial sound. Note specifically that the third and atrial heart sounds are each a very low rumble. The

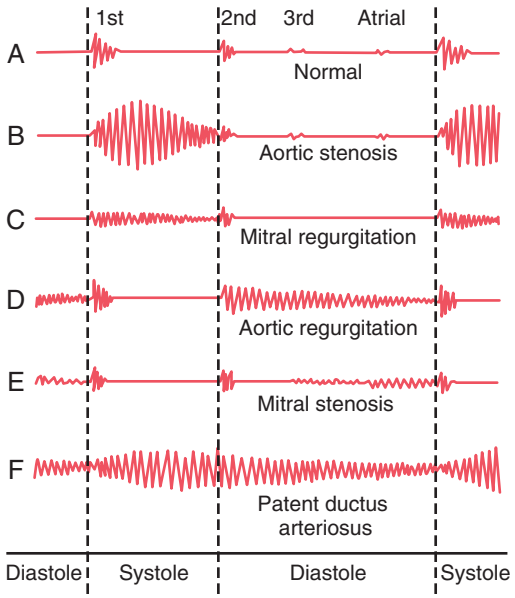


Figure 23-3. Phonocardiograms from normal and abnormal hearts.

third heart sound can be recorded in only one third to one half of all people, and the atrial heart sound can be recorded in perhaps one fourth of all people.

VALVULAR LESIONS

Rheumatic Valvular Lesions

By far the greatest number of valvular lesions results from *rheumatic fever.* Rheumatic fever is an autoimmune disease in which the heart valves are likely to be damaged or destroyed. The disease is usually initiated by a streptococcal toxin.

The sequence of events almost always begins with a preliminary streptococcal infection caused specifically by group A hemolytic streptococci. These bacteria initially cause a sore throat, scarlet fever, or middle ear infection. However, the streptococci also release several different proteins against which the person's reticuloendothelial system produces *antibodies.* The antibodies react not only with the streptococcal protein but also with other protein tissues of the body, often causing severe immunologic damage. These reactions continue to take place as long as the antibodies persist in the blood—1 year or more.

Rheumatic fever particularly causes damage in certain susceptible areas, such as the heart valves. The degree of heart valve damage is directly correlated with the concentration and persistence of the antibodies. The principles of immunity that relate to this type of reaction are discussed in Chapter 35, and it is noted in Chapter 32 that acute glomerular nephritis of the kidneys has a similar immunologic basis.

In persons with rheumatic fever, large hemorrhagic, fibrinous, bulbous lesions grow along the inflamed edges of the heart valves. Because the mitral valve undergoes more trauma during valvular action than any of the other valves, it is the one most often seriously damaged, and the aortic valve is the second most frequently damaged. The right heart valves—that is, the tricuspid and pulmonary valves—are usually affected much less severely, probably because the low-pressure stresses that act on these valves are slight compared with the high-pressure stresses that act on the left heart valves.

Scarring of the Valves. The lesions of acute rheumatic fever frequently occur on adjacent valve leaflets simultaneously, so the edges of the leaflets become stuck together. Then, weeks, months, or years later, the lesions become scar tissue, permanently fusing portions of adjacent valve leaflets. Also, the free edges of the leaflets, which are normally filmy and free-flapping, often become solid, scarred masses.

A valve in which the leaflets adhere to one another so extensively that blood cannot flow through it normally is said to be *stenosed.* Conversely, when the valve edges are so destroyed by scar tissue that they cannot close as the ventricles contract, *regurgitation* (backflow) of blood occurs when the valve should be closed. Stenosis usually does not occur without the coexistence of at least some degree of regurgitation, and vice versa.

Other Causes of Valvular Lesions. Stenosis or lack of one or more leaflets of a valve also occurs occasionally as a *congenital defect.* Complete lack of leaflets is rare; *congenital stenosis* is more common, as is discussed later in this chapter.

Heart Murmurs Are Caused by Valvular Lesions

As shown by the phonocardiograms in **Figure 23-3**, many abnormal heart sounds, known as "heart murmurs," occur when abnormalities of the valves are present, as follows.

Systolic Murmur of Aortic Stenosis. In persons with aortic stenosis, blood is ejected from the left ventricle through only a small fibrous opening of the aortic valve. Because of the resistance to ejection, sometimes the blood pressure in the left ventricle rises as high as 300 mm Hg, while the pressure in the aorta is still normal. Thus, a nozzle effect is created *during systole,* with blood jetting at tremendous velocity through the small opening of the valve. This phenomenon causes *severe turbulence* of the blood in the root of the aorta. The turbulent blood impinging against the aortic walls causes intense vibration, and a loud murmur (see **recording B, Figure 23-3**) occurs during systole and is transmitted throughout the superior thoracic aorta and even into the large arteries of the neck. This sound is harsh, and in persons with severe stenosis it may be so loud that it can be heard several feet away from the patient. Also, the sound vibrations can often be felt with the hand on the upper chest and lower neck, a phenomenon known as a *thrill.*

Diastolic Murmur of Aortic Regurgitation. In aortic regurgitation, no abnormal sound is heard during systole, but *during diastole,* blood flows backward from the high-pressure aorta into the left ventricle, causing a "blowing" murmur of relatively high pitch with a swishing quality heard maximally over the left ventricle (see **recording D, Figure 23-3**). This murmur results from *turbulence* of blood jetting backward into the blood already in the low-pressure diastolic left ventricle.

Systolic Murmur of Mitral Regurgitation. In persons with mitral regurgitation, blood flows backward through the mitral valve into the left atrium *during systole.* This backward flow also causes a high-frequency "blowing," swishing sound (see **recording C, Figure 23-3**) similar to that of aortic regurgitation but occurring during systole rather than diastole. It is transmitted most strongly into the left atrium. However, the left atrium is so deep within the chest that it is difficult to hear this sound directly over the atrium. As a result, the sound of mitral regurgitation is transmitted to the chest wall mainly through the left ventricle to the apex of the heart.

Diastolic Murmur of Mitral Stenosis. In persons with mitral stenosis, blood passes with difficulty through the stenosed mitral valve from the left atrium into the left ventricle, and because the pressure in the left atrium seldom rises above 30 mm Hg, a large pressure differential forcing blood from the left atrium into the left ventricle does not develop. Consequently, the abnormal sounds heard in mitral stenosis (see **recording E, Figure 23-3**) are usually weak and of very low frequency, so most of the sound spectrum is below the low-frequency end of human hearing.

During the early part of diastole, a left ventricle with a stenotic mitral valve has so little blood in it and its walls are so flabby that blood does not reverberate back and forth between the walls of the ventricle. For this reason, even in persons with severe mitral stenosis, no murmur may be heard during the first third of diastole. Then, after partial filling, the ventricle has stretched enough for blood to reverberate and a low rumbling murmur begins.

Phonocardiograms of Valvular Murmurs. Phonocardiograms B, C, D, and E of **Figure 23-3** show, respectively, idealized records obtained from patients with aortic stenosis, mitral regurgitation, aortic regurgitation, and mitral stenosis. It is obvious from these phonocardiograms that the aortic stenotic lesion causes the loudest murmur, and the mitral stenotic lesion causes the weakest murmur. The phonocardiograms show how the intensity of the murmurs varies during different portions of systole and diastole, and the relative timing of each murmur is also evident. Note especially that the murmurs of aortic stenosis and mitral regurgitation occur only during systole, whereas the murmurs of aortic regurgitation and mitral stenosis occur only during diastole. If the reader does not understand this timing, extra review should be undertaken until it is understood.

ABNORMAL CIRCULATORY DYNAMICS IN VALVULAR HEART DISEASE

DYNAMICS OF THE CIRCULATION IN AORTIC STENOSIS AND AORTIC REGURGITATION

In *aortic stenosis,* the contracting left ventricle fails to empty adequately, whereas in *aortic regurgitation,* blood flows backward into the ventricle from the aorta after the ventricle has just pumped the blood into the aorta. Therefore, in either case, the *net stroke volume output* of the heart is reduced.

Several important compensations take place that can ameliorate the severity of the circulatory defects. Some of these compensations are described in the following sections.

Hypertrophy of the Left Ventricle. In both aortic stenosis and aortic regurgitation, the left ventricular musculature hypertrophies because of the increased ventricular workload.

In *regurgitation,* the left ventricular chamber also enlarges to hold all the regurgitant blood from the aorta. Sometimes the left ventricular muscle mass increases fourfold to fivefold, creating a tremendously large left side of the heart.

When the aortic valve is seriously *stenosed,* the hypertrophied muscle allows the left ventricle to develop as much as 400 mm Hg of intraventricular pressure at systolic peak.

In persons with severe aortic regurgitation, sometimes the hypertrophied muscle allows the left ventricle to pump a stroke volume output as great as 250 milliliters, although as much as three fourths of this blood returns to the ventricle during diastole, and only one fourth flows through the aorta to the body.

Increase in Blood Volume. Another effect that helps compensate for the diminished net pumping by the left ventricle is increased blood volume. This increased volume results from (1) an initial slight decrease in arterial pressure, plus (2) peripheral circulatory reflexes induced by the decrease in pressure. These mechanisms together diminish renal output of urine, causing the blood volume to increase and the mean arterial pressure to return to normal. Also, red blood cell mass eventually increases because of a slight degree of tissue hypoxia.

The increase in blood volume tends to increase venous return to the heart, which, in turn, causes the left ventricle to pump with the extra power required to overcome the abnormal pumping dynamics.

Aortic Valvular Lesions May be Associated with Inadequate Coronary Blood Flow. When a person has stenosis of the aortic valve, the ventricular muscle must develop a high tension to create the high intraventricular pressure needed to force blood through the stenosed valve. This action increases work load and oxygen consumption of the ventricle, necessitating increased coronary blood flow to deliver this oxygen. The high wall tension of the ventricle, however, causes marked decreases in coronary flow during systole, particularly in the subendocardial vessels. Intraventricular diastolic pressure is also increased when there is aortic valve stenosis, and this increased pressure may cause compression of the inner layers of the heart muscle and reduced coronary blood flow. Thus, severe aortic valve stenosis often causes ischemia of the heart muscle.

With aortic regurgitation the intraventricular diastolic pressure also increases, compressing the inner layer of the heart muscle and decreasing coronary blood flow. Aortic diastolic pressure decreases during aortic regurgitation, which can also decrease coronary blood flow and cause ischemia of the heart muscle.

Eventual Failure of the Left Ventricle and Development of Pulmonary Edema. In the early stages of aortic stenosis or aortic regurgitation, the intrinsic ability of the left ventricle to adapt to increasing loads prevents significant abnormalities in circulatory function in the person during rest, other than increased work output required of the left ventricle. Therefore, considerable degrees of aortic stenosis or aortic regurgitation often occur before the person knows that he or she has serious heart disease (such as a resting left ventricular systolic pressure as high as 200 mm Hg in persons with aortic stenosis or a left ventricular stroke volume output as high as double normal in persons with aortic regurgitation).

Beyond a critical stage in these aortic valve lesions, the left ventricle finally cannot keep up with the work demand. As a consequence, the left ventricle dilates and cardiac output begins to fall; blood simultaneously dams up in the left atrium and in the lungs behind the failing left ventricle. The left atrial pressure rises progressively, and at mean left atrial pressures above 25 to 40 mm Hg, serious edema appears in the lungs, as discussed in detail in Chapter 39.

DYNAMICS OF MITRAL STENOSIS AND MITRAL REGURGITATION

In persons with mitral stenosis, blood flow from the left atrium into the left ventricle is impeded, and in persons with mitral regurgitation, much of the blood that has flowed into the left ventricle during diastole leaks back into the left atrium during systole rather than being pumped into the aorta. Therefore, either of these conditions reduces net movement of blood from the left atrium into the left ventricle.

Pulmonary Edema in Mitral Valvular Disease. The buildup of blood in the left atrium causes progressive increase in left atrial pressure, eventually resulting in the development of serious pulmonary edema. Ordinarily, lethal edema does not occur until the mean left atrial pressure rises above 25 mm Hg and sometimes as high as 40 mm Hg, because the lung lymphatic vessels enlarge manyfold and can rapidly carry fluid away from the lung tissues.

Enlarged Left Atrium and Atrial Fibrillation. The high left atrial pressure in mitral valvular disease also causes progressive enlargement of the left atrium, which increases the distance that the cardiac electrical excitatory impulse must travel in the atrial wall. This pathway may eventually become so long that it predisposes to the development of excitatory signal *circus movements,* as discussed in Chapter 13. Therefore, in late stages of mitral valvular disease, especially in mitral stenosis, atrial fibrillation often occurs. This development further reduces the pumping effectiveness of the heart and causes further cardiac debility.

Compensation in Early Mitral Valvular Disease. As also occurs in aortic valvular disease and in many types of congenital heart disease, the blood volume increases in mitral valvular disease principally because of diminished excretion of water and salt by the kidneys. This increased blood volume increases venous return to the heart, thereby helping to overcome the effect of the cardiac debility. Therefore, after compensation, cardiac output may fall only minimally until the late stages of mitral valvular disease, even though the left atrial pressure is rising.

As the left atrial pressure rises, blood begins to dam up in the lungs, eventually all the way back to the pulmonary artery. In addition, incipient edema of the lungs causes pulmonary arteriolar constriction. These two effects together increase systolic pulmonary arterial pressure and also right ventricular pressure, sometimes to as high as 60 mm Hg, which is more than double normal. This increased pressure, in turn, causes hypertrophy of the right side of the heart, which partially compensates for its increased workload.

CIRCULATORY DYNAMICS DURING EXERCISE IN PATIENTS WITH VALVULAR LESIONS

During exercise, large quantities of venous blood are returned to the heart from the peripheral circulation. Therefore, all the dynamic abnormalities that occur in the different types of valvular heart disease become tremendously exacerbated. Even in persons with mild valvular heart disease, in which the symptoms may be unrecognizable at rest, severe symptoms often develop during heavy exercise. For instance, in patients with aortic valvular lesions, exercise can cause acute left ventricular failure

followed by *acute pulmonary edema.* Also, in patients with mitral disease, exercise can cause so much damming of blood in the lungs that serious or even lethal pulmonary edema may ensue in as little as 10 minutes.

Even in mild to moderate cases of valvular disease, the patient's *cardiac reserve* diminishes in proportion to the severity of the valvular dysfunction. That is, the cardiac output does not increase as much as it should during exercise. Therefore, the muscles of the body fatigue rapidly because of too little increase in muscle blood flow.

ABNORMAL CIRCULATORY DYNAMICS IN CONGENITAL HEART DEFECTS

Occasionally, the heart or its associated blood vessels are malformed during fetal life; the defect is called a *congenital anomaly.* There are three major types of congenital anomalies of the heart and its associated vessels: (1) *stenosis* of the channel of blood flow at some point in the heart or in a closely allied major blood vessel; (2) an anomaly that allows blood to flow backward from the left side of the heart or aorta to the right side of the heart or pulmonary artery, thus failing to flow through the systemic circulation, which is called a *left-to-right shunt;* and (3) an anomaly that allows blood to flow directly from the right side of the heart into the left side of the heart, thus failing to flow through the lungs—called a *right-to-left shunt.*

The effects of the different stenotic lesions are easily understood. For instance, *congenital aortic valve stenosis* results in the same dynamic effects as aortic valve stenosis caused by other valvular lesions, namely, cardiac hypertrophy, heart muscle ischemia, reduced cardiac output, and a tendency to develop serious pulmonary edema.

Another type of congenital stenosis is *coarctation of the aorta,* often occurring near the level of the diaphragm. This stenosis causes the arterial pressure in the upper part of the body (above the level of the coarctation) to be much greater than the pressure in the lower body because of the great resistance to blood flow through the coarctation to the lower body; part of the blood must go around the coarctation through small collateral arteries, as discussed in Chapter 19.

PATENT DUCTUS ARTERIOSUS IS A LEFT-TO-RIGHT SHUNT

During fetal life, the lungs are collapsed, and the elastic compression of the lungs that keeps the alveoli collapsed keeps most of the lung blood vessels collapsed as well. Therefore, resistance to blood flow through the lungs is so great that the pulmonary arterial pressure is high in the fetus. Also, because of low resistance to blood flow from the aorta through the large vessels of the placenta, the pressure in the aorta of the fetus is lower than normal—in fact, lower than in the pulmonary artery. This phenomenon causes almost all the pulmonary arterial

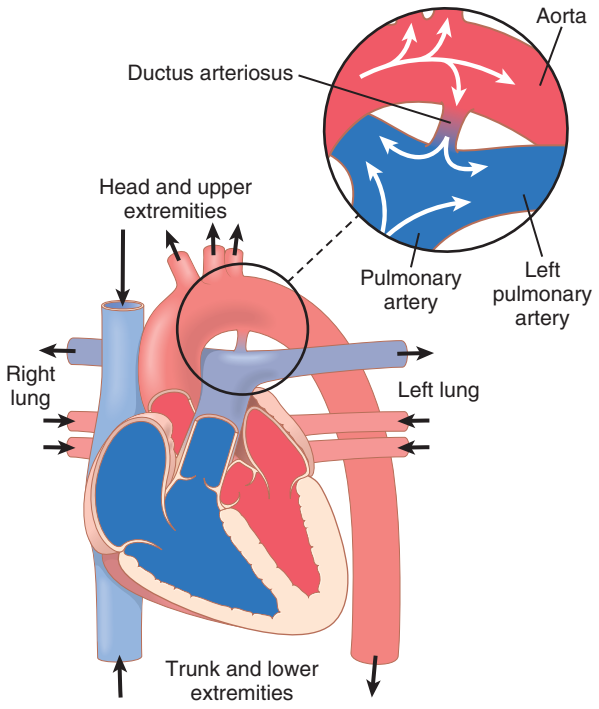


Figure 23-4. Patent ductus arteriosus, showing by the blue color that venous blood changes into oxygenated blood at different points in the circulation. The right-hand diagram shows backflow of blood from the aorta into the pulmonary artery and then through the lungs for a second time.

blood to flow through a special artery present in the fetus that connects the pulmonary artery with the aorta (**Figure 23-4**), called the *ductus arteriosus,* thus bypassing the lungs. This mechanism allows immediate recirculation of the blood through the systemic arteries of the fetus without the blood going through the lungs. This lack of blood flow through the lungs is not detrimental to the fetus because the blood is oxygenated by the placenta.

Closure of the Ductus Arteriosus After Birth. As soon as a baby is born and begins to breathe, the lungs inflate; not only do the alveoli fill with air, but also the resistance to blood flow through the pulmonary vascular tree decreases tremendously, allowing the pulmonary arterial pressure to fall. Simultaneously, the aortic pressure rises because of sudden cessation of blood flow from the aorta through the placenta. Thus, the pressure in the pulmonary artery falls, while that in the aorta rises. As a result, forward blood flow through the ductus arteriosus ceases suddenly at birth, and in fact, blood begins to flow backward through the ductus from the aorta into the pulmonary artery. This new state of backward blood flow causes the ductus arteriosus to become occluded within a few hours to a few days in most babies, so blood flow through the ductus does not persist. The ductus is believed to close because the oxygen concentration of the aortic blood now flowing through it is about twice as high as that of the blood flowing from the pulmonary artery into the ductus during fetal life. The oxygen presumably constricts the

muscle in the ductus wall. This phenomenon is discussed further in Chapter 84.

Unfortunately, in about 1 of every 5500 babies, the ductus does not close, causing the condition known as *patent ductus arteriosus,* which is shown in **Figure 23-4**.

Dynamics of the Circulation with a Persistent Patent Ductus

During the early months of an infant's life, a patent ductus usually does not cause severely abnormal function. However, as the child grows older, the differential between the high pressure in the aorta and the lower pressure in the pulmonary artery progressively increases, with corresponding increase in backward flow of blood from the aorta into the pulmonary artery. Also, the high aortic blood pressure usually causes the diameter of the partially open ductus to increase with time, making the condition even worse.

Recirculation Through the Lungs. In an older child with a patent ductus, one half to two thirds of the aortic blood flows backward through the ductus into the pulmonary artery, then through the lungs, and finally back into the left ventricle and aorta, passing through the lungs and left side of the heart two or more times for every one time that it passes through the systemic circulation. People with this condition *do not show cyanosis until later in life, when the heart fails or the lungs become congested.* Indeed, early in life, the arterial blood is often better oxygenated than normal because of the extra times it passes through the lungs.

Diminished Cardiac and Respiratory Reserve. The major effects of patent ductus arteriosus on the patient are decreased cardiac and respiratory reserve. The left ventricle is pumping about two or more times the normal cardiac output, and the maximum that it can pump after hypertrophy of the heart has occurred is about four to seven times normal. Therefore, during exercise, the net blood flow through the remainder of the body can never increase to the levels required for strenuous activity. With even moderately strenuous exercise, the person is likely to become weak and may even faint from momentary heart failure.

The high pressures in the pulmonary vessels caused by excess flow through the lungs may also lead to pulmonary congestion and pulmonary edema. As a result of the excessive load on the heart, and especially because the pulmonary congestion becomes progressively more severe with age, most patients with uncorrected patent ductus die from heart disease between ages 20 and 40 years.

Heart Sounds: Machinery Murmur

In a newborn infant with patent ductus arteriosus, occasionally no abnormal heart sounds are heard because the quantity of reverse blood flow through the ductus may be insufficient to cause a heart murmur. But as the baby grows older, reaching age 1 to 3 years, a harsh, blowing murmur begins to be heard in the pulmonary artery area of the chest, as shown in recording F, **Figure 23-3**. This sound is much more intense during systole when the aortic pressure is high and much less intense during diastole when the aortic pressure falls low, so that the murmur waxes and wanes with each beat of the heart, creating the so-called *machinery murmur.*

Surgical Treatment

Surgical treatment of patent ductus arteriosus is simple; one need only ligate the patent ductus or divide it and then close the two ends. In fact, this procedure was one of the first successful heart surgeries ever performed.

TETRALOGY OF FALLOT IS A RIGHT-TO-LEFT SHUNT

Tetralogy of Fallot is shown in **Figure 23-5**; it is the most common cause of "blue baby." Most of the blood bypasses the lungs, so the aortic blood is mainly unoxygenated venous blood. In this condition, four abnormalities of the heart occur simultaneously:

1. The aorta originates from the right ventricle rather than the left, or it overrides a hole in the septum, as shown in **Figure 23-5**, receiving blood from both ventricles.
2. Because the pulmonary artery is stenosed, much lower than normal amounts of blood pass from the right ventricle into the lungs; instead, most of the blood passes directly into the aorta, thus bypassing the lungs.

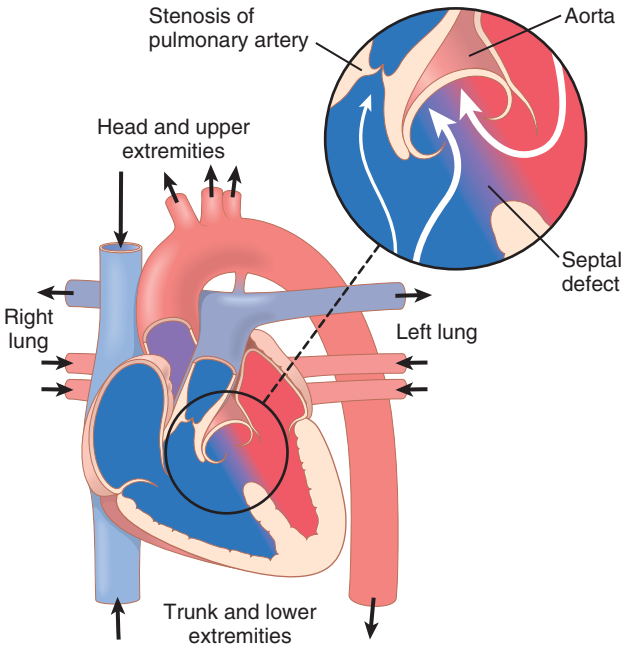


Figure 23-5. Tetralogy of Fallot, showing by the blue color that most of the venous blood is shunted from the right ventricle into the aorta without passing through the lungs.

3. Blood from the left ventricle flows either through a ventricular septal hole into the right ventricle and then into the aorta or directly into the aorta that overrides this hole.
4. Because the right side of the heart must pump large quantities of blood against the high pressure in the aorta, its musculature is highly developed, causing an enlarged right ventricle.

Abnormal Circulatory Dynamics. It is readily apparent that the major physiological difficulty caused by tetralogy of Fallot is the shunting of blood past the lungs without its becoming oxygenated. As much as 75 percent of the venous blood returning to the heart passes directly from the right ventricle into the aorta without becoming oxygenated.

A diagnosis of tetralogy of Fallot is usually based on (1) the fact that the baby's skin is *cyanotic* (blue); (2) measurement of high systolic pressure in the right ventricle, recorded through a catheter; (3) characteristic changes in the radiological silhouette of the heart, showing an enlarged right ventricle; and (4) angiograms (x-ray pictures) showing abnormal blood flow through the interventricular septal hole and into the overriding aorta, but much less flow through the stenosed pulmonary artery.

Surgical Treatment. Tetralogy of Fallot can usually be treated successfully with surgery. The usual operation is to open the pulmonary stenosis, close the septal defect, and reconstruct the flow pathway into the aorta. When surgery is successful, the average life expectancy increases from only 3 to 4 years to 50 or more years.

CAUSES OF CONGENITAL ANOMALIES

Congenital heart disease is not uncommon, occurring in about 8 of every 1000 live births. One of the most common causes of congenital heart defects is a viral infection in the mother during the first trimester of pregnancy when the fetal heart is being formed. Defects are particularly prone to develop when the expectant mother contracts German measles.

Some congenital defects of the heart are hereditary because the same defect has been known to occur in identical twins, as well as in succeeding generations. Children of patients surgically treated for congenital heart disease have about a 10 times greater chance of having congenital heart disease than do other children. Congenital defects of the heart are also frequently associated with other congenital defects of the baby's body.

USE OF EXTRACORPOREAL CIRCULATION DURING CARDIAC SURGERY

It is almost impossible to repair intracardiac defects surgically while the heart is still pumping. Therefore, many types of artificial *heart-lung machines* have been developed to take the place of the heart and lungs during the course of an operation. Such a system is called *extracorporeal circulation.* The system consists principally of a pump and an oxygenating device. Almost any type of pump that does not cause hemolysis of the blood seems to be suitable.

Methods used for oxygenating blood include (1) bubbling oxygen through the blood and removing the bubbles from the blood before passing it back into the patient, (2) dripping the blood downward over the surfaces of plastic sheets in the presence of oxygen, (3) passing the blood over surfaces of rotating discs, or (4) passing the blood between thin membranes or through thin tubes that are permeable to oxygen and carbon dioxide.

The different systems have all been fraught with difficulties, including hemolysis of the blood, development of small clots in the blood, likelihood of small bubbles of oxygen or small emboli of antifoam agent passing into the arteries of the patient, the necessity for large quantities of blood to prime the entire system, failure to exchange adequate quantities of oxygen, and the need to use heparin to prevent blood coagulation in the extracorporeal system. Heparin also interferes with adequate hemostasis during the surgical procedure. Yet despite these difficulties, in the hands of experts, patients can be kept alive on artificial heart-lung machines for many hours while operations are performed on the inside of the heart.

HYPERTROPHY OF THE HEART IN VALVULAR AND CONGENITAL HEART DISEASE

Hypertrophy of cardiac muscle is one of the most important mechanisms by which the heart adapts to increased workloads, whether these loads are caused by increased pressure against which the heart muscle must contract or by increased cardiac output that must be pumped. Some physicians believe that the increased strength of contraction of the heart muscle causes the hypertrophy; others believe that the increased metabolic rate of the muscle is the primary stimulus. Regardless of which of these is correct, one can calculate approximately how much hypertrophy will occur in each chamber of the heart by multiplying ventricular output by the pressure against which the ventricle must work, with emphasis on pressure. Thus, hypertrophy occurs in most types of valvular and congenital disease, sometimes causing heart weights as great as 800 grams instead of the normal 300 grams.

Detrimental Effects of Late Stages of Cardiac Hypertrophy. Although the most common cause of cardiac hypertrophy is hypertension, almost all forms of cardiac diseases, including valvular and congenital disease, can stimulate enlargement of the heart.

"Physiological" cardiac hypertrophy is generally considered to be a compensatory response of the heart to increased workload and is usually beneficial for maintaining cardiac output in the face of abnormalities that impair the heart's effectiveness as a pump. However, extreme degrees of hypertrophy can lead to heart failure. One of the reasons for this is that the coronary vasculature typically does not increase to the same extent as the mass of cardiac muscle increases. The second reason is that fibrosis often develops in the muscle, especially in the subendocardial muscle where the coronary blood flow is poor, with fibrous tissue replacing degenerating muscle fibers. Because of the disproportionate increase in muscle mass relative to coronary blood flow, relative ischemia may develop as the cardiac muscle hypertrophies, and coronary blood flow insufficiency may ensue. Anginal pain is therefore a frequent accompaniment of cardiac hypertrophy associated with valvular and congenital heart diseases. Enlargement of the heart is also associated with greater risk for developing arrhythmias, which in turn can lead to further impairment of cardiac function and sudden death because of fibrillation.

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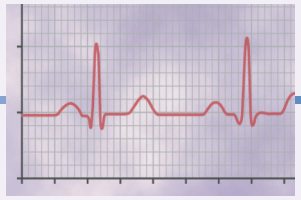

CHAPTER 11

UNIT III

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 (ECG)*. A normal ECG for two beats of the heart is shown in **Figure 11-1**.

CHARACTERISTICS OF THE NORMAL ELECTROCARDIOGRAM

The normal ECG (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. The T wave is known as a *repolarization wave.*

Thus, the ECG 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, with 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, which causes the meter to record positively. To the right of the muscle fiber is shown a record of changes in potential between the two electrodes, as recorded by a high-speed recording meter. Note that when depolarization has reached the halfway mark in **Figure 11-2*A***, the record has risen to a maximum positive value.

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

Figure 11-2*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 polarity is opposite to the polarity in **Figure 11-2*A***. Consequently, the recording, as shown to the right, becomes negative.

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

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

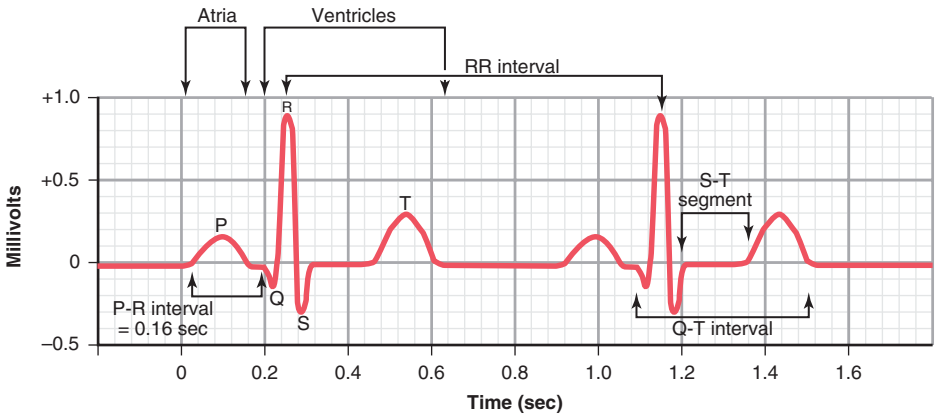

Figure 11-1. Normal electrocardiogram.

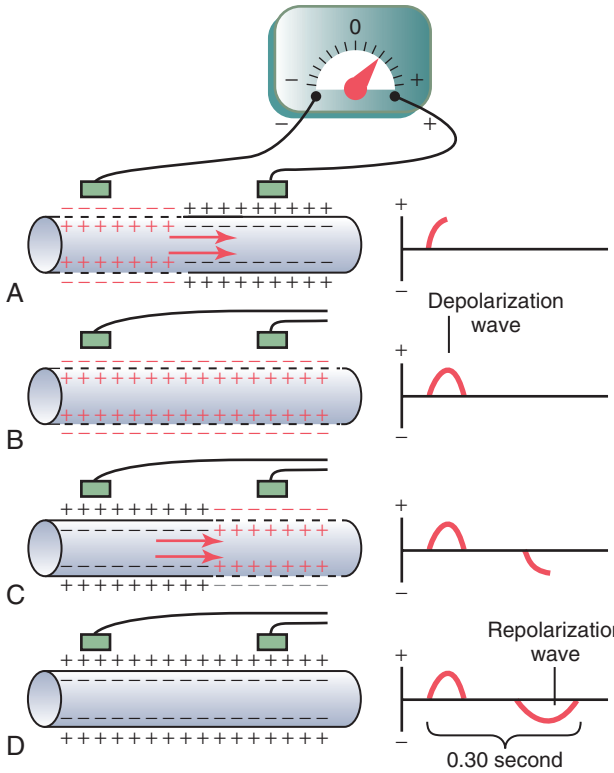

Figure 11-2. Recording the depolarization wave (**A** and **B**) and the repolarization wave (**C** and **D**) from a cardiac muscle fiber.

return of the potential to the baseline is caused by repolarization.

The lower half of **Figure 11-3** shows a simultaneous recording of the ECG from this same ventricle. Note that the QRS waves appear at the beginning of the monophasic action potential and the T wave appears at the end. Note especially that *no potential is recorded in the ECG 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 ECG.

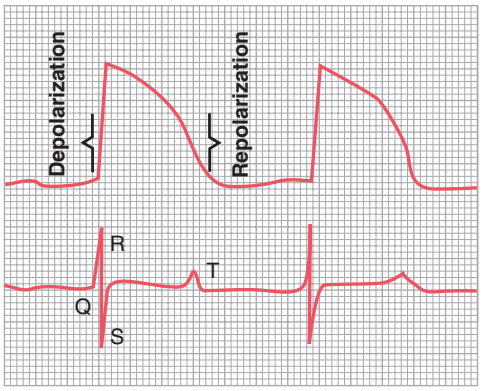

Figure 11-3. Top, Monophasic action potential from a ventricular muscle fiber during normal cardiac function showing rapid depolarization and then repolarization occurring slowly during the plateau stage but rapidly toward the end. **Bottom,** Electrocardiogram recorded simultaneously.

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, which is also approximately when the QRS complex is being recorded in the ECG. 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 on the ECG.

The ventricular repolarization wave is the T wave of the normal ECG. 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 ECG 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 ECGs are made with appropriate calibration lines on the recording paper. Either these calibration lines are already ruled on the paper, as is the case with the pen recorder is used, or they are recorded on the paper at the same time that the ECG is recorded, which is the case with the photographic types of electrocardiographs.

As shown in Figure [illegible], the horizontal calibration lines are arranged so that the small line divisions upward or downward from the isoelectric standard ECG represent 1 millivolt, with positivity in the upward direction and negativity in the downward direction.

The vertical lines on the ECG are time calibration lines. A typical ECG 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 ECG 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 100 millivolts recorded directly at the heart muscle membrane. When ECGs are recorded from electrodes on the two arms or on the 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 the voltage 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 to the end of the R wave, if the Q wave is absent. The end of the T wave. This interval is called the *Q-T interval* and ordinarily is about 0.35 second.

Rate of Heartbeat Determined from the Electrocardiogram. The rate of the heartbeat can be determined easily from the ECG because the heart rate is the reciprocal of the time interval between two successive heartbeats. The interval between 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 an adult is about 0.83 second, which is a heart rate of 60/0.83 times per minute, or 72 beats/min.

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 center by a shock point. Before stimulation, the exteriors of the muscle cells had been positive and the interiors had been negative. For reasons presented in Chapter 5 in the discussion of membrane potentials, as soon as the area of cardiac syncytium becomes depolarized, positive charges leak to the outsides of the depolarized muscle fibers, making this part of the surface electronegative, as represented by the minus signs in Figure 11-4. The remaining surface of the heart, which is still polarized, is represented by the plus 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.

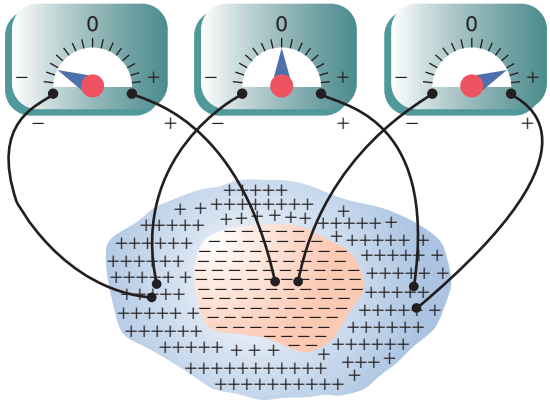

Figure 11-4. Instantaneous potentials develop on the surface of a cardiac muscle mass that has been depolarized in its center.

Two other electrode placements and meter readings are also demonstrated in **Figure 11-4**. These placements and readings 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 shows the ventricul muscle lying within the chest. the lungs, altho mostly filled with air, conduct elect to a su g extent, and fluids in other tissues su di heart conduct electricity even more easily. e, the heart is actually suspended in a condu dium. When one portion of the ventricles de izes therefore becomes electronegative with ect to the inder, electrical current flows from depolarized are he polarized area in large ci us routes, as noted i figure.

It d be recalled from the dis on 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 process 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 arrow the figure. If one algebraical verages all the lines o ent flow (the elliptical), one finds that *the averag rent flow occurs w egativity toward the base of the h nd with po y toward the apex.*

During most he re er of the depolarization process, current al es to flow in this same direction, while depolari spreads from the endocardial surface outward g ventricular muscle mass. Then, immedi before arization has completed its course th the ventricl average direction of current fl verses for about 0. ond, flowing from the ve lar apex toward the base, use the last part of the eart to become depolarized is e 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. 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 ECG.

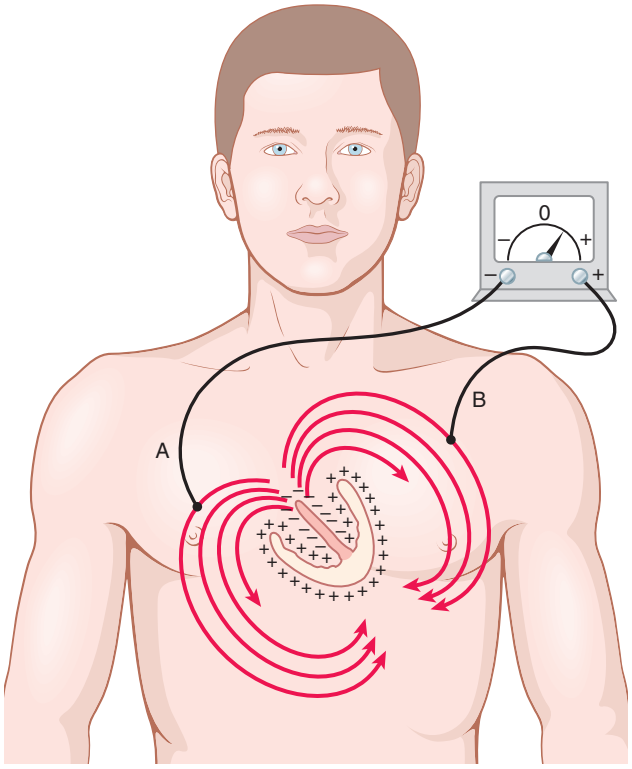

Figure 11-5. Flow of current in the chest around partially depolarized ventricles. *A* and *B* are electrodes.

ELECTROCARDIOGRAPHIC LEADS

THREE BIPOLAR LIMB LEADS

Figure 11-6 shows electrical connections between the patient's limbs and the electrocardiograph for recording ECGs from the so-called *standard bipolar 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 computer-based system with an electronic display.

Lead I. In recording limb lead I, the negative terminal of the electrocardiograph is connected to the right arm and the positive terminal is connected 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 ECG. When the opposite is true, the electrocardiograph records below the line.

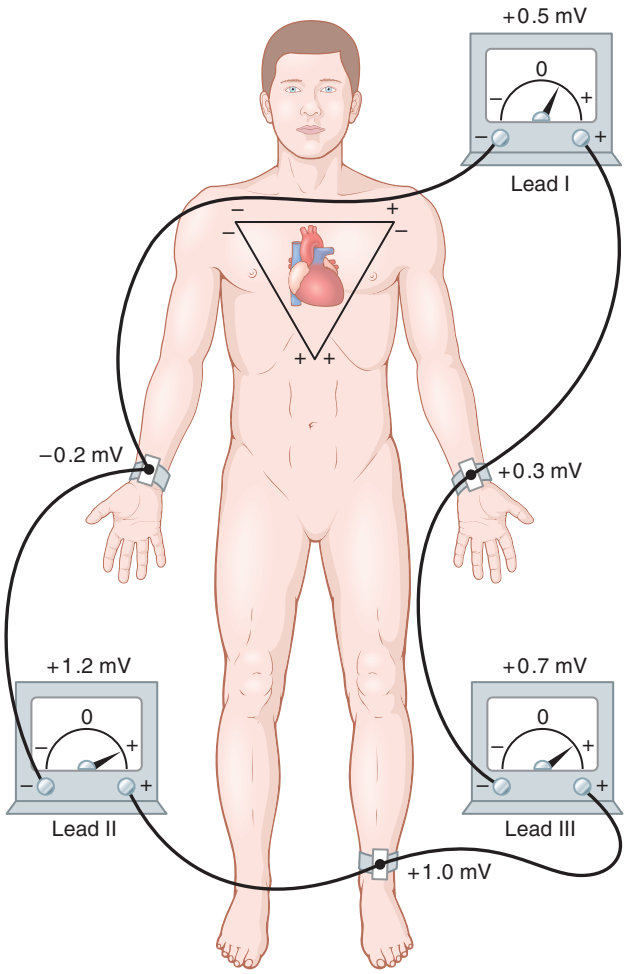

Figure 11-6. Conventional arrangement of electrodes for recording the standard electrocardiographic leads. Einthoven's triangle is superimposed on the chest.

Lead II. To record limb lead II, the negative terminal of the electrocardiograph is connected to the right arm and the positive terminal is connected 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 is connected to the left leg. This configuration 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 triangle 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.

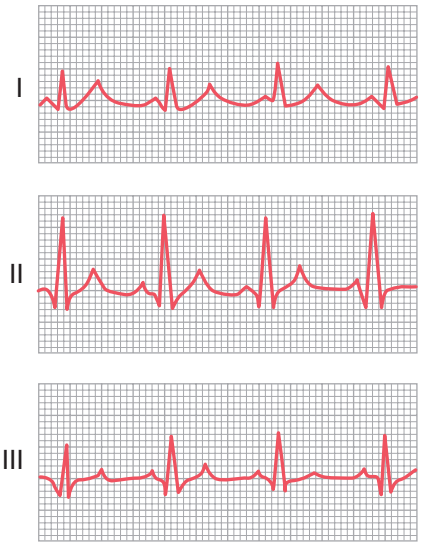

Figure 11-7. Normal electrocardiograms recorded from the three standard electrocardiographic leads.

Einthoven's Law. Einthoven's law states that if the ECGs are recorded simultaneously with the three limb leads, the sum of the potentials recorded in leads I and III will equal the potential in lead II.

Lead I potential + Lead III potential = Lead II potential

In other words, 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 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 ECGs are being recorded.

Normal Electrocardiograms Recorded from the Three Standard Bipolar Limb Leads. **Figure 11-7** shows recordings of the ECGs in leads I, II, and III. It is obvious that the ECGs 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 ECG.

On analysis of the three ECGs, 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. However, when one wants to diagnose damage in the ventricular or atrial muscle or in the Purkinje conducting system, it matters greatly which leads are recorded because abnormalities of cardiac muscle contraction or cardiac impulse conduction change the patterns of the ECGs 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 ECGs 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, with the chest electrode being placed sequentially at the six points shown in the diagram. The different recordings are known as leads V_1, V_2, V_3, V_4, V_5, and V_6.

Figure 11-9 illustrates the ECGs 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 ECGs recorded from individual chest leads.

In leads V_1 and V_2, 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 the ventricular depolarization process. Conversely, the QRS complexes in leads V_4, V_5, and V_6 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.

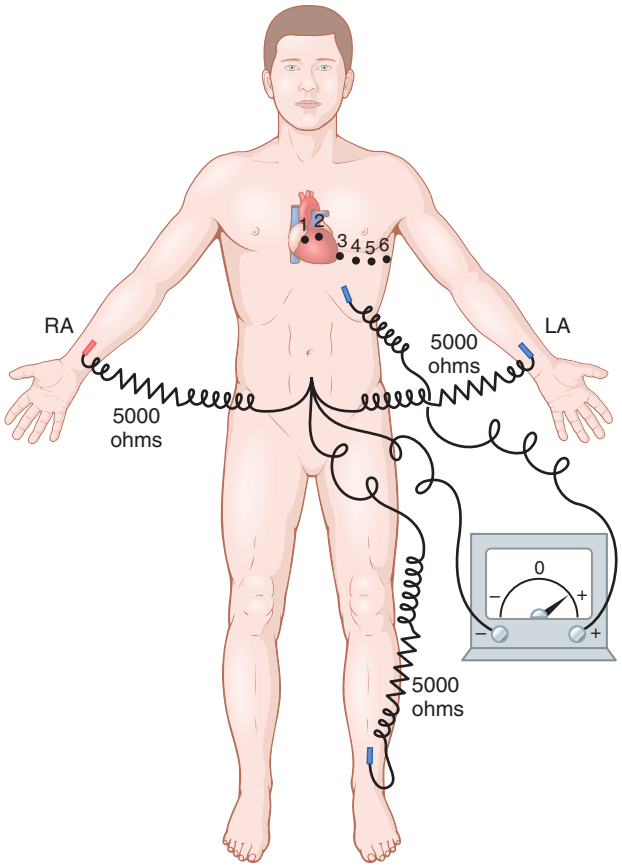

Figure 11-8. Connections of the body with the electrocardiograph for recording chest leads. LA, left arm; RA, right arm.

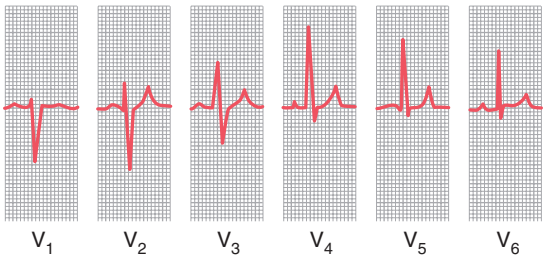

Figure 11-9. Normal electrocardiograms recorded from the six standard chest leads.

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, it is known as the aVL lead; and when on the left leg, it is known as the aVF lead.

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 the answer to this question.)

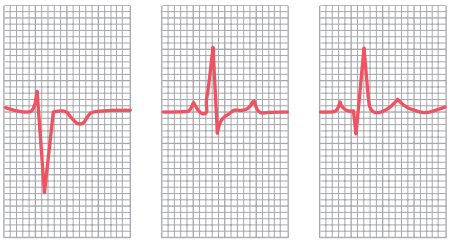

Figure 11-10. Normal electrocardiograms recorded from the three augmented unipolar limb leads.

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 ECGs be capable of responding rapidly to these changes in potentials. Modern clinical electrocardiographs use computer-based systems and electronic display.

Ambulatory Electrocardiography

Standard ECGs provide assessment of cardiac electrical events over a brief duration, usually while the patient is resting. In conditions associated with infrequent but important abnormalities of cardiac rhythms, it may be useful to examine the ECG over a longer period, thereby permitting evaluation of changing cardiac electrical phenomena that are transient and may be missed with a standard ECG. Extending the ECG to allow assessment of cardiac electrical events while the patient is ambulating during normal daily activities is called *ambulatory electrocardiography*.

Ambulatory ECG monitoring is typically used when a patient demonstrates symptoms that are thought to be caused by transient arrhythmias or other transient cardiac abnormalities. These symptoms may include chest pain, syncope (fainting) or near syncope, dizziness, and irregular heartbeats. The crucial information needed to diagnose serious, transient arrhythmias or other cardiac conditions is a recording of an ECG during the precise time that the symptom is occurring. Because the day-to-day variability in the frequency of arrhythmias is substantial, detection often requires ambulatory ECG monitoring throughout the day.

There are two categories of ambulatory ECG recorders: (1) continuous recorders, typically used for 24 to 48 hours to investigate the relationship of symptoms and ECG events that are likely to occur within that time frame, and (2) intermittent recorders, which are used for longer periods (weeks to months) to provide brief, intermittent recordings for detection of events that occur infrequently. In some cases a small device, about the size of a pack of chewing gum and called an *implantable loop recorder,* is implanted just under the skin in the chest to monitor the heart's electrical activity intermittently for as long as 2 to 3 years. The device can be programmed to initiate a recording when the heart rate fall below, or rises above, a predetermined level, or it can be activated manually by the patient when a symptom such as dizziness occurs. Improvements in solid-state digital technology and recorders equipped with microprocessors now permit continuous or intermittent transmission of digital ECG data over phone lines, and sophisticated software systems provide rapid "online" computerized analysis of the data as they are acquired.

Bibliography

See the bibliography for Chapter 13.