Physiology of the heart.

Plan.

1. **Functions of the heart.**
2. **Electrical Activity of the heart.**
3. **Cardiac cycle and its phases.**
4. **Heart sounds and their diagnostic value.**
5. **Physiological and technical aspects of electrocardiography.**

Functions of the heart.

Three main functions of the heart are:

Generating blood pressure. Contractions of the heart generate blood pressure, which is needed for blood flow through the blood vessels.

Routing blood. The heart is two pumps, moving blood through the pulmonary and systemic circulations. The pulmonary circulation is the flow of blood from the heart through the lungs back to the heart. Blood in the pulmonary circulation picks up oxygen and releases carbon dioxide in the lungs. The systemic circulation is the flow of blood from the heart through the body back to the heart. Blood in the systemic circulation delivers oxygen and nutrients to the cell and picks up carbon dioxide and waste products from the body's tissues (figure 17.1).

Regulating blood supply. Changes in the rate and force of heart contraction match blood flow to the changing metabolic needs of the tissues during rest, exercise, and changes in body position.

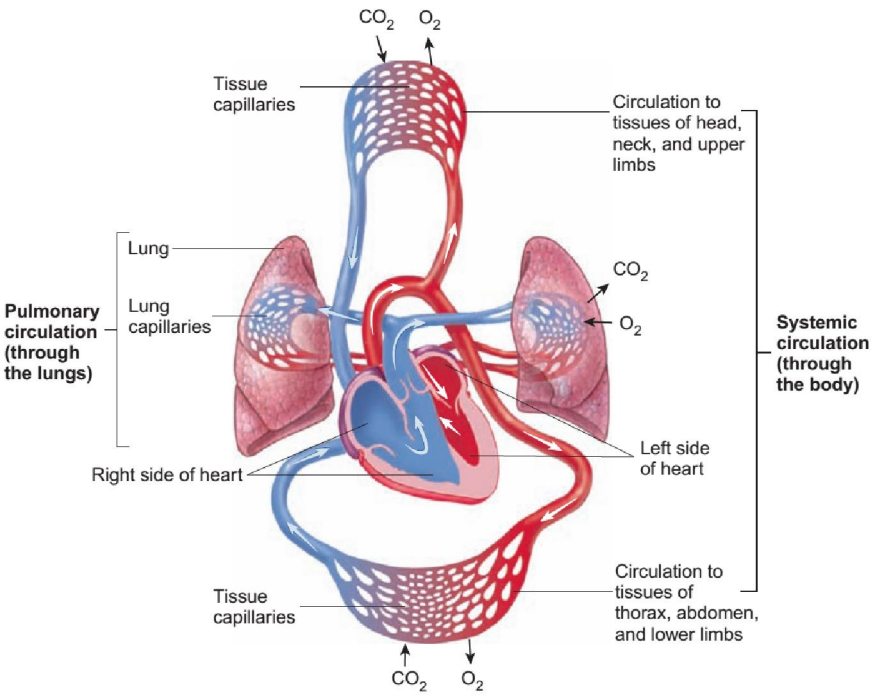


Figure 17.1 Systemic and Pulmonary Circulation AP|R
The circulatory system consists of the pulmonary and systemic circulations. The right side of the heart pumps blood through vessels to the lungs and back to the left side of the heart through the pulmonary circulation. The left side of the heart pumps blood through vessels to the tissues of the body and back to the right side of the heart through the systemic circulation.

The healthy heart is able to pump the circulating blood volume through both the pulmonary and systemic systems. When the heart is damaged (by myocardial infarctions or long-standing high blood pressure, for example), it is unable to maintain the delicate balance between blood volume and the ability to pump. Fluid backs up in the lungs when the left ventricle fails, resulting in

shortness of breath, cough, and respiratory distress. When the right ventricle weakens, fluid retains in the peripheral tissues, leading to edema (swelling in the extremities) and liver engorgement.

Electrical Activity of the Heart.

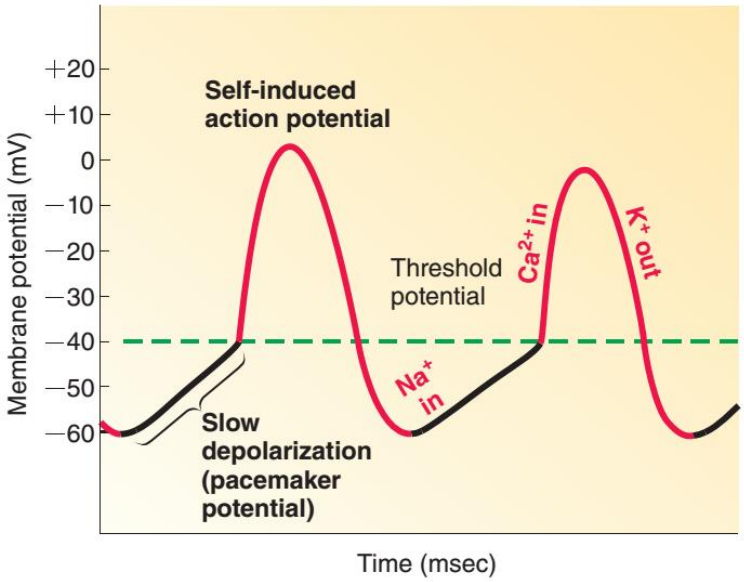
There are two specialized types of cardiac muscle cells:

1. **Contractile cells**, which are 99% of the cardiac muscle cells, do the mechanical work of pumping. These working cells normally do not initiate their own action potentials.
2. In contrast, **the autorhythmic (noncontractile) cells**, the small but extremely important remainder of the cardiac cells, do not contract but instead are specialized for initiating and conducting the action potentials responsible for contraction of the working cells.

Pacemaker activity of the cardiac autorhythmic cells.

In contrast to nerve and skeletal muscle cells, in which the membrane remains at constant resting potential unless the cell is stimulated, the cardiac autorhythmic cells do not have a resting potential. Instead, they display pacemaker activity; that is, their membrane potential slowly depolarizes, or drifts, between action potentials until threshold is reached, at which time the membrane fires or has an action potential.

An autorhythmic cell membrane's slow drift to threshold is called the **pacemaker potential** (Figure 9-6;). A unique voltage-gated channel found only in cardiac autorhythmic cells plays a key role in producing the pacemaker potential. These unusual channels open when the potential becomes more negative (hyperpolarizes) at the end of repolarization from the previous action potential. Because of this unusual behavior, they are sometimes called **funny channels**. When one action potential ends and the funny channels open, the resultant depolarizing net inward Na^+ movement starts immediately moving the pacemaker cell's potential toward threshold once again. Once threshold is reached, the rising phase of the action potential occurs in response to opening of voltage gated Ca^{2+} channels and the resulting entry of Ca^{2+} , in contrast to nerve and skeletal muscle cells where Na^+ entry rather than Ca^{2+} entry swings the potential in the positive direction. The falling phase occurs as usual by K^+ leaving the pacemaker cell as a result of opening of voltage-gated K^+ channels. Through repeated cycles of drift and fire, these autorhythmic cells cyclically initiate action potentials, which then spread throughout the heart to trigger rhythmic beating without any nervous stimulation



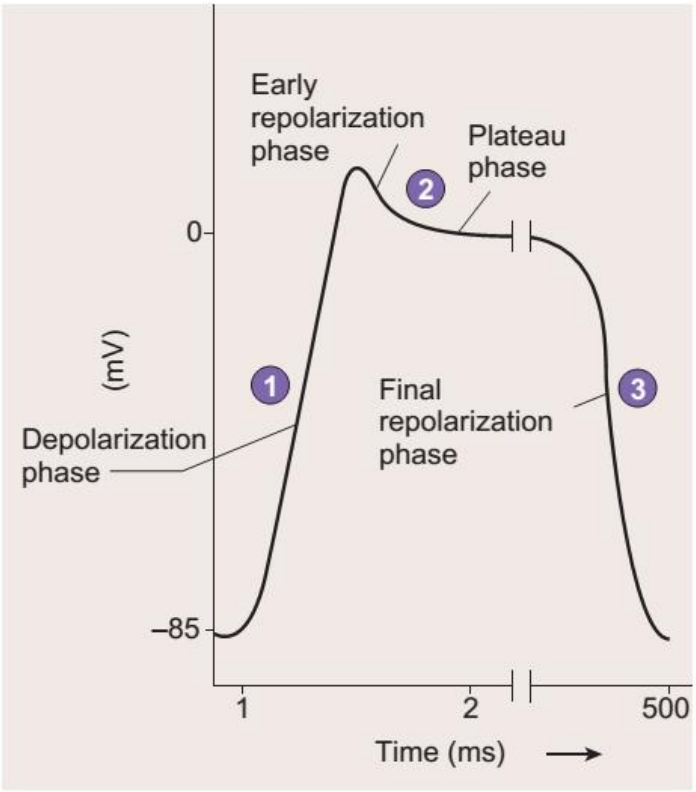
● **FIGURE 9-6 Pacemaker activity of cardiac autorhythmic cells.** The pacemaker potential (slow, self-induced depolarization to threshold) is largely caused by Na^+ entry through unique funny channels that open in response to hyperpolarization at the end of the previous action potential. Once threshold is reached, the rising phase of the action potential is the result of Ca^{2+} entry on opening of Ca^{2+} channels, whereas the falling phase is the result of K^+ exit on opening of K^+ channels.

The action potential of cardiac contractile cells.

The action potential in cardiac contractile cells, although initiated by the nodal pacemaker cells, is considerably different in ionic mechanisms and shape from the SA node potential (Figures

9-9). Unlike the membrane of autorhythmic cells, the membrane potential of contractile cells remains at rest on level of about –85 millivolts (mV), until excited by electrical activity propagated from the pacemaker. Once the membrane of a ventricular myocardial contractile cell is excited, the membrane potential rapidly reverses to a positive value of +30 mV as a result of activation of voltage-gated Na^+ channels and Na^+ subsequently rapidly enters the cell, as it does in other excitable cells.

In cardiac muscle, each action potential consists of a **depolarization phase** followed by a rapid but partial **early repolarization phase.**This is followed by a longer period of slow repolarization, called the **plateau phase**.At the end of the plateau phase, a more rapid **final repolarization phase** takes place. During the final repolarization phase, the membrane potential achieves its maximum degree of repolarization.



(b)

Permeability changes during an action potential in cardiac muscle:

1. **Depolarization phase**
   - Voltage-gated Na^+ channels open.
   - Voltage-gated K^+ channels close.
   - Voltage-gated Ca^{2+} channels begin to open.
2. **Early repolarization and plateau phases**
   - Voltage-gated Na^+ channels close.
   - Some voltage-gated K^+ channels open, causing early repolarization.
   - Voltage-gated Ca^{2+} channels are open, producing the plateau by slowing further repolarization.
3. **Final repolarization phase**
   - Voltage-gated Ca^{2+} channels close.
   - Many voltage-gated K^+ channels open.

Opening and closing of voltage-gated ion channels are responsible for the changes in the permeability of the plasma membrane that produce action potentials. The depolarization phase of the action potential results from three permeability changes. Sodium ion channels open, increasing the permeability of the plasma membrane to Na^+. Sodium ions then diffuse into the cell, causing depolarization. This depolarization causes K^+ channels to close quickly, decreasing the permeability of the plasma membrane to K^+. The decreased diffusion of K^+ out of the cell causes further depolarization. Calcium ion channels slowly open, increasing the permeability of the plasma membrane to Ca^{2+}. Calcium ions then diffuse into the cell and cause depolarization. It is not until the plateau phase that most of the Ca^{2+} channels open.

Early repolarization occurs when the Na^+ channels close and a small number of K^+ channels open. Diffusion of Na^+ into the cell stops, and there is some movement of K^+ out of the cell, which produces a small repolarization. The plateau phase occurs as Ca^{2+} channels continue to open, and the diffusion of Ca^{2+} into the cell counteracts the potential change produced by the diffusion of K^+ out of the cell. The plateau phase ends and final repolarization begins as the Ca^{2+} channels close, and many K^+ channels open. Diffusion of Ca^{2+} into the cell decreases and diffusion of K^+ out of the cell increases. These changes cause the membrane potential to repolarize during the final repolarization phase.

Refractory periods and their role in the heart pump function.

Cardiac muscle, like skeletal muscle, has **absolute** and **relative refractory periods** associated with its action potentials. The absolute refractory period begins with depolarization and extends into the final repolarization phase. During the absolute refractory period, the cardiac muscle cell is completely insensitive to further stimulation. The relative refractory period occurs during the last part of the final repolarization phase. During the relative refractory period, the cell is sensitive to stimulation, but a greater stimulation than normal is required to cause an action potential. The absolute refractory period is much longer in cardiac muscle than in skeletal muscle because of the long duration of the plateau phase in cardiac muscle. The long absolute refractory period ensures that contraction and most of relaxation are complete before another action potential can be initiated. This prevents tetanus in cardiac muscle and is responsible for rhythmic contractions.

Conducting System of the Heart.

Effective pumping of blood through the heart depends on coordinated contraction of the atria and ventricles. The atria contract, moving blood into the ventricles. Then the ventricles contract, moving blood to the lungs and body. **The conducting system** of the heart, which consists of specialized cardiac muscle cells, stimulates the atria and ventricles to contract by relaying action potentials through the heart (figure 17.13).

The sinoatrial (SA) node is located medial to the opening of the superior vena cava. Action potentials originating in the SA node spread over the right and left atria, causing them to contract. Some of these action potentials reach the atrioventricular (AV) node, located in the lower portion of the right atrium. Action potentials propagate slowly through the AV node before moving on to stimulate the ventricles to contract. This delay lasts approximately 0.11 sec and it allows the atria to complete their contraction before the ventricles contract.

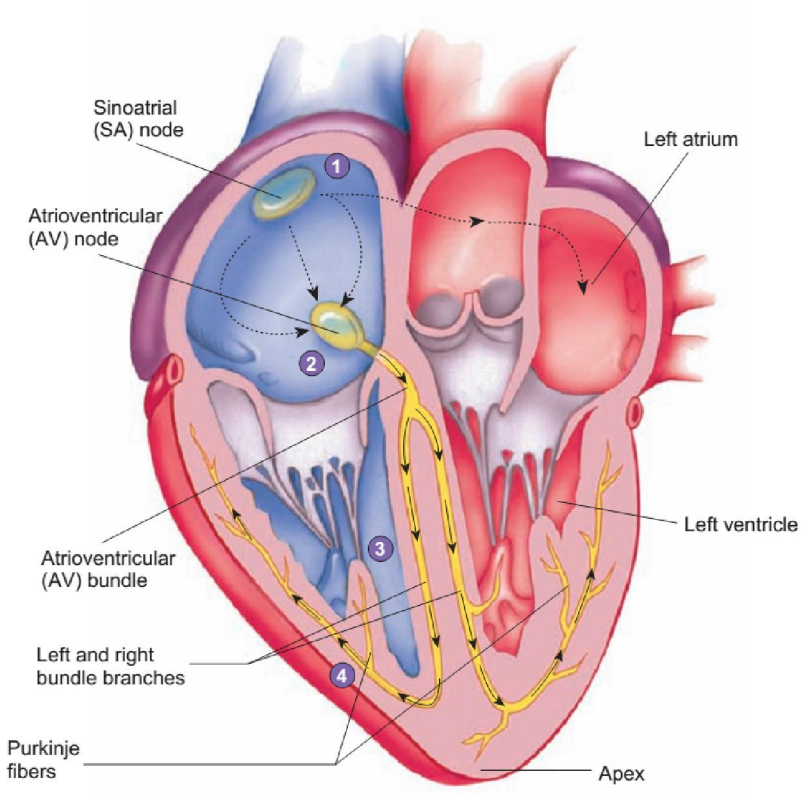
The AV node gives excitation to the atrioventricular (AV) bundle, which passes through a small opening in the fibrous skeleton of the heart to reach the interventricular septum. The fibrous skeleton electrically separates the atria and ventricles so that action potentials spreading through the atria do not cause the ventricles to contract at the same time as the atria. Thus, only action potentials transmitted through the conducting system of the heart normally cause the ventricles to contract, allowing atria and ventricular contractions to be coordinated.

1. Action potentials originate in the sinoatrial (SA) node (the pacemaker) and travel across the wall of the atrium (*arrows*) from the SA node to the atrioventricular (AV) node.

2. Action potentials pass through the AV node and along the atrioventricular (AV) bundle, which extends from the AV node, through the fibrous skeleton, into the interventricular septum.

3. The AV bundle divides into right and left bundle branches, and action potentials descend to the apex of each ventricle along the bundle branches.

4. Action potentials are carried by the Purkinje fibers from the bundle branches to the ventricular walls and papillary muscles.



Process Figure 17.13 Conducting System of the Heart AP|R

The AV bundle divides at the interventricular septum to form the **right and left bundle branches**, which extend to the apex of the heart. Many small bundles of **Purkinje fibers** pass from the tips of the right and left bundle branches to the apex of the heart and then extend superiorly to the cardiac muscle of the ventricular walls. The AV bundle, the bundle branches, and the Purkinje fibers are composed of specialized cardiac muscle cells that conduct action potentials very rapidly. Consequently, ventricular contraction begins at the apex and progresses superiorly throughout the ventricles. This action pushes blood superiorly out the pulmonary trunk and aorta.

Cardiac cycle and its phases

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The heart can be viewed as two separate pumps represented by the right and left halves of the heart. Each pump consists of a primer pump—the atrium—and a power pump—the ventricle. The atria act as **primer pumps** because they complete the filling of the ventricles with blood, and the ventricles act as **power pumps** because they produce the major force that causes blood to flow through the pulmonary and systemic circulations. The term cardiac cycle refers to the repetitive pumping process that begins with the onset of cardiac muscle contraction and ends with the beginning of the next contraction (figure 17.15). Pressure changes produced within the heart chambers as a result of cardiac muscle contraction are responsible for blood movement because blood moves from areas of higher pressure to areas of lower pressure.

Atrial systole refers to contraction of the two atria, which occurs simultaniously. **Ventricular systole** refers to contraction of the two ventricles, which also occurs simultaniously. **Atrial diastole** refers to relaxation of the two atria, and **ventricular diastole** refers to relaxation of the two ventricles. When the terms systole and diastole are used without reference to the atria or ventricles, they refer to ventricular contraction or relaxation.

Just before ventricular systole begins, the atria and ventricles are relaxed, the ventricles are filled with blood, the semilunar valves are closed, and the AV valves are open. As systole begins, contraction of the ventricles increases ventricular pressures, causing blood to flow toward the atria and close the AV valves. As contraction proceeds, ventricular pressures continue to rise, but no blood flows from the ventricles because all the valves are closed. Thus, ventricular volume does not change, even though the ventricles are contracting (see figure 17.15, step 1). This period of cardiac cycle is called – **isovolumetric contraction**.

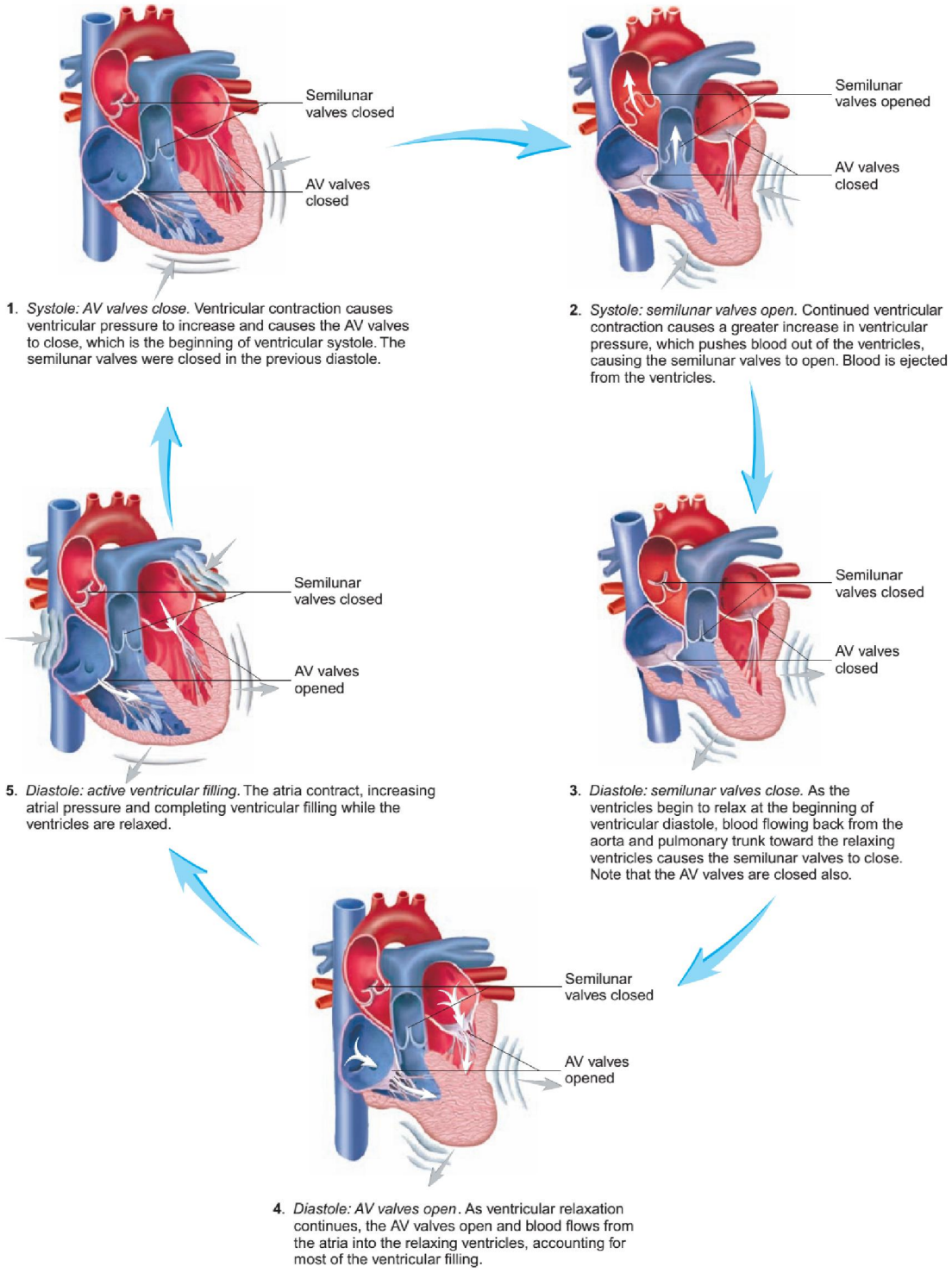
As the ventricles continue to contract, ventricular pressures become greater than the pressures in the pulmonary trunk and aorta. As a result, the semilunar valves are pushed open and blood flows from the ventricles into those arteries (see figure 17.15, step 2). This period of cardiac cycle is called **ejection phase**.

As diastole begins, the ventricles relax and ventricular pressures decrease below the pressures in the pulmonary trunk and aorta. Blood begins to flow back toward the ventricles, causing the semilunar valves to close (see figure 17.15, step 3). With closure of the semilunar valves, all the heart valves are closed and no blood flows into the relaxing ventricles. This period of cardiac cycle is called – **isovolumetric relaxation** because no change of ventricular volume occurs.

Throughout systole and the beginning of diastole, the atria relax and blood flows into them from the veins. When ventricular pressures become lower than atrial pressures, the AV valves open and blood flows from the atria into the relaxed ventricles (see figure 17.15, step 4). Most ventricular filling occurs as a result of this passive flow of blood during the first two-thirds of diastole. This period of cardiac cycle is called **rapid filling phase**. The remainder of ventricular filling occurs when the atria contract and push blood into the ventricles during **reduced filling phase** (see figure 17.15, step 5). During exercise, atrial contraction is more important for ventricular filling because, as heart rate increases, less time is available for passive ventricular filling.

Events occurring during ventricular systole.

Figure 17.16 displays the main events of the cardiac cycle in graphic form and should be examined from top to bottom for systole and diastole. An ECG indicates the electrical events that cause contraction and relaxation of the atria and ventricles. The pressure graph shows the pressure changes within the left atrium, left ventricle, and aorta resulting from atrial and ventricular contraction and relaxation. Although pressure changes in the right side of the heart are not shown,



Process Figure 17.15 Cardiac Cycle AP|R

The cardiac cycle is a repeating series of contraction and relaxation that moves blood through the heart (*AV* = atrioventricular).

they are similar to those in the left side, only lower. The volume graph presents the changes in left ventricular volume as blood flows into and out of the left ventricle as a result of the pressure changes. The sound graph records the closing of valves caused by blood flow.

Ventricular depolarization produces the QRS complex and initiates contraction of the ventricles. Ventricular pressure rapidly increases, resulting in closure of the AV valves. During the previous ventricular diastole, the ventricles were filled with blood, which is called the **end-diastolic volume**. At first, ventricular volume does not change because all the heart valves are closed. As soon as ventricular pressures exceed the pressures in the aorta and pulmonary trunk, the semilunar valves open. The aortic semilunar valve opens at approximately 80 mm Hg ventricular pressure, whereas the pulmonary semilunar valve opens at approximately 8 mm Hg. Although the pressures are different, both valves open at nearly the same time. As blood flows from the ventricles, the left

ventricular pressure continues to climb to approximately 120 mm Hg, and the right ventricular

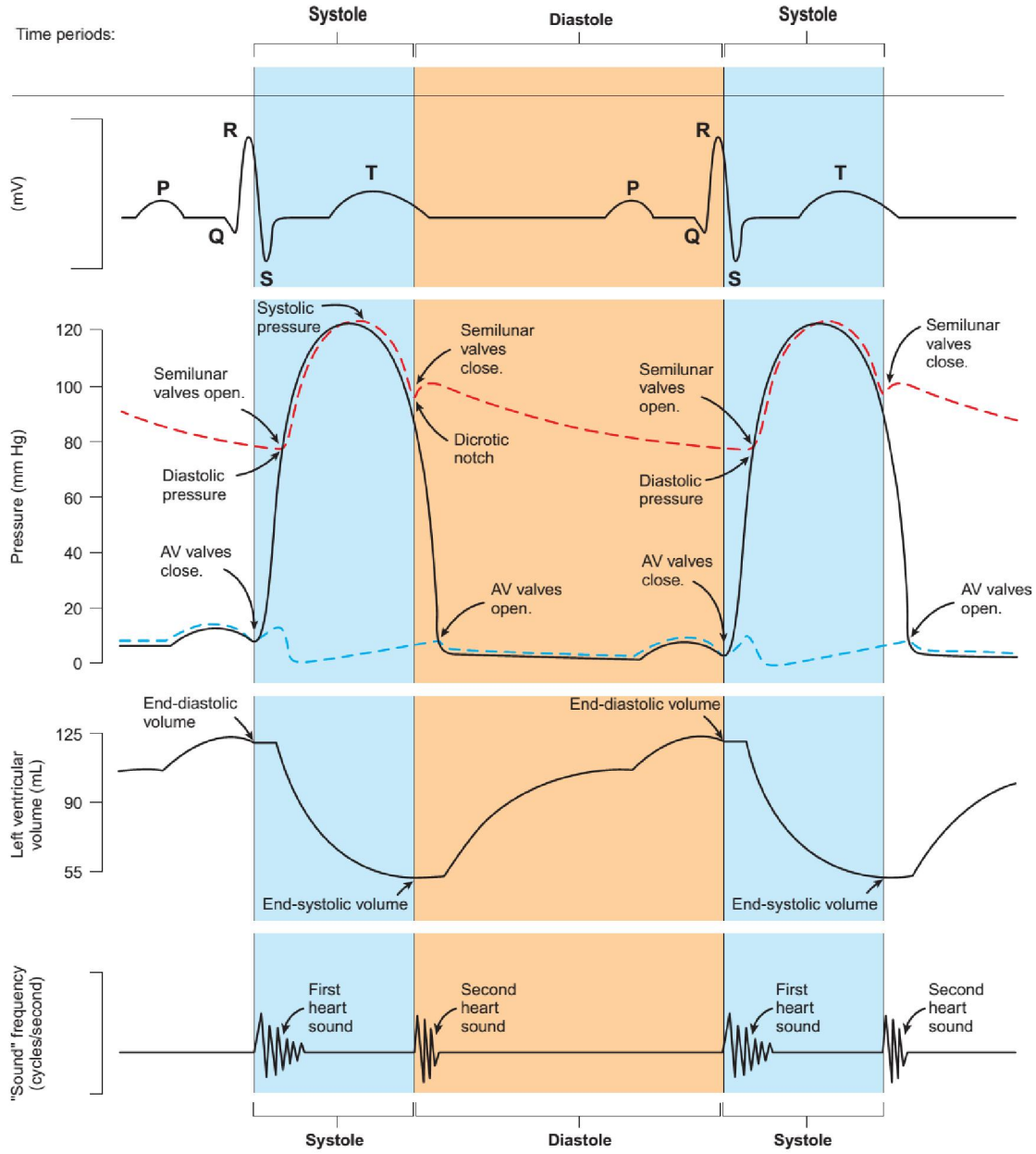


Figure 17.16 Events Occurring During the Cardiac Cycle
The cardiac cycle is divided into ventricular systole and diastole (see *top of figure*). Within systole and diastole, four graphs are presented. From *top* to *bottom*, the electrocardiogram; pressure changes for the left atrium (*blue line*), left ventricle (*black line*), and aorta (*red line*); left ventricular volume curve; and heart sounds are illustrated.

pressure increases to approximately 22 mm Hg. The higher pressure generated by the left ventricle is necessary to move blood through the larger systemic circulation. A lower pressure is adequate to move blood through the smaller pulmonary circulation. At first, blood flows rapidly out of the ventricles. Toward the end of systole, very little blood flow occurs, which causes the ventricular pressure to decrease despite continued ventricular contraction. As systole ends, the volume of blood remaining in the ventricle is called **the end-systolic volume**.

Events occurring during ventricular diastole.

Ventricular repolarization produces the T wave and the ventricles relax. The already decreasing ventricular pressure falls very rapidly as the ventricles suddenly relax. When the ventricular pressures fall below the pressures in the aorta and pulmonary trunk, the recoil of the elastic arterial walls, which were stretched during the period of ejection, forces the blood to flow back toward the ventricles, thereby closing the semilunar valves. Ventricular volume does not change at this time because all the heart valves are closed.

When ventricular pressure drops below atrial pressure, the atrioventricular valves open and blood flows from the area of higher pressure in the veins and atria toward the area of lower pressure

in the relaxed ventricles, which decreases to nearly 0 mm Hg. Approximately 70% of ventricular fi lling occurs during the first two-thirds of diastole.

Depolarization of the SA node generates action potentials that spread over the atria, producing the P wave and stimulating both atria to contract (atrial systole). The atria contract during the last one-third of diastole and complete ventricular filling. Under most conditions, the atria function primarily as reservoirs, and the ventricles can pump sufficient blood to maintain homeostasis even if the atria do not contract at all. During exercise, however, the heart pumps 300%–400% more blood than during resting condition. It is under these conditions that the pumping action of the atria becomes important in maintaining the pumping efficiency of the heart.

Aortic Pressure Curve.

As the ventricles contract and push blood into the aorta, aortic pressure increases to a maximum value called the **systolic pressure**, which is approximately 120 mm Hg (see figure 17.16). Even though ventricular pressure drops below aortic pressure as the ventricle relaxes, the forward momentum of the ejected blood maintains aortic pressure and prevents the backflow of blood into the ventricle. The higher pressure in the aorta eventually slows and then reverses the flow of blood, which moves back toward the ventricle, closing the aortic semilunar valve. Elastic recoil of the aorta prevents a large increase in aortic pressure and moves blood through the systemic circulation. Elastic recoil also moves blood back toward the left ventricle and pressure within the aorta ncreases slightly, producing a **dicrotic notch** in the aortic pressure curve (see figure 17.16).

Aortic pressure gradually falls throughout the rest of ventricular diastole as blood flows through the peripheral vessels. Aortic pressure decreases to a minimum value called **diastolic pressure**,which is approximately 80 mm Hg. The blood pressure in the aorta fluctuates between systolic and diastolic pressure. Blood pressure measurements performed for clinical purposes reflect the pressure changes that occur in the aorta rather than in the left ventricle. Arteriosclerosis is a hardening of the arteries that occurs with age. The decreased elasticity of the aorta results in less expansion of the aorta when blood is injected into it, producing high blood pressure.

Heart sounds and their diagnostic value.

Two major heart sounds normally can be heard with a stethoscope during the cardiac cycle. The first heart sound is low pitched, soft, and relatively long—often said to sound like "lub." The second heart sound has a higher pitch and is shorter and sharper—often said to sound like "dup." Thus , one normally hears "lub-dup-lub-dup-lub-dup . . . ." The first heart sound is associated with closure of the AV valves, whereas the second sound is associated with closure of the semilunar valves. Opening of valves does not produce any sound.

The sounds are caused by vibrations set up within the walls of the ventricles and major arteries during valve closure, not by the valves snapping shut. Because the AV valves close at

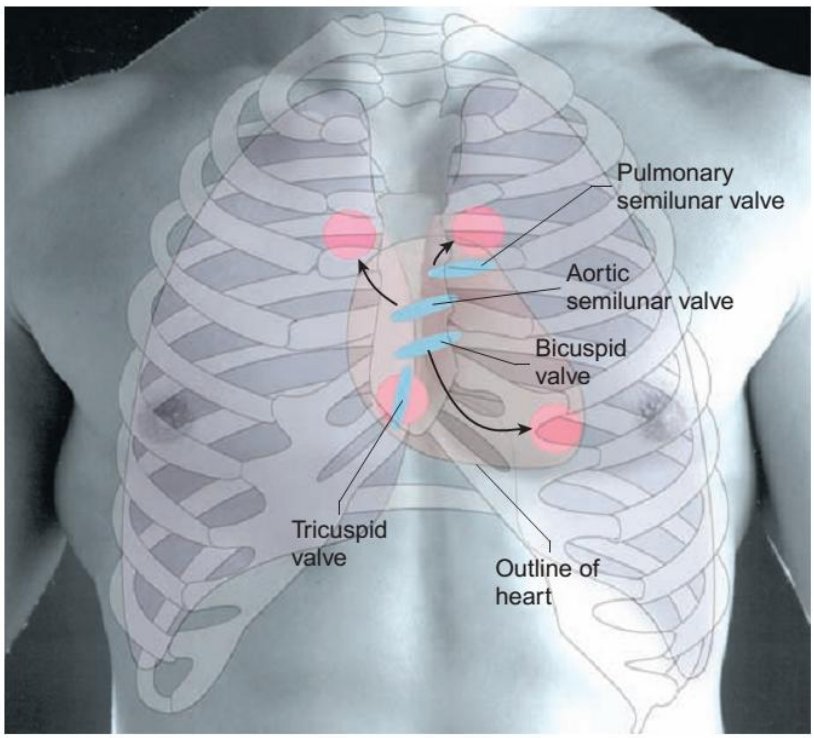


Figure 17.17 Location of the Heart Valves in the Thorax
Surface markings of the heart in the male. The positions of the four heart valves are indicated by *blue ellipses,* and the sites where the sounds of the valves are best heard with the stethoscope are indicated by *pink circles.*

the onset of ventricular contraction, when ventricular pressure first exceeds atrial pressure, the first heart sound signals the onset of ventricular systole. The semilunar valves close at the onset of ventricular relaxation, as the left and right ventricular pressures fall below the aortic and pulmonary artery pressures, respectively. The second heart sound, therefore, signals the onset of ventricular diastole.

Abnormal heart sounds, or **murmurs**, are usually (but not always) associated with cardiac disease. Murmurs not involving heart pathology, so-called functional murmurs, are more common in young people.

Blood normally flows in a laminar fashion; that is, layers of the fluid slide smoothly over each other. Laminar flow does not produce any sound. When blood flow becomes turbulent, however, a sound can be heard. Such an abnormal sound is the result of vibrations that the turbulent flow creates in the surrounding structures.

Stenotic and insufficient valves.

The most common cause of turbulence is valve malfunction, either a stenotic or an insufficient valve. A stenotic valve is a stiff , narrowed valve that does not open completely. Blood must be forced through the constricted opening at tremendous velocity, resulting in turbulence that produces an abnormal whistling sound similar to the sound produced when you force air rapidly through narrowed lips to whistle.

An insufficient,or incompetent, valve is one that cannot close completely, usually because the valve edges are scarred and do not fit together properly. Turbulence is produced when blood flows backward through the insufficient valve and collides with blood moving in the opposite direction, creating a swishing or gurgling murmur. Such backflow of blood is known as **regurgitation**. An insufficient heart valve is often called a leaky valve, because it lets blood leak back through at a time when the valve should be closed.

Most often, both valvular stenosis and insufficiency are caused by rheumatic fever, an autoimmune ("immunity against self") disease triggered by a streptococcus bacterial infection. Antibodies formed against toxins produced by these bacteria. interact with many of the body's own tissues, resulting in immunological damage. The heart valves are among the most susceptible tissues in this regard. Large, hemorrhagic, fibrous lesions form along the inflamed edges of an affected heart valve, causing the valve to become thickened, stiff, and scarred. Sometimes the leaflet edges permanently adhere to each other. Depending on the extent and specific nature of the lesions, the valve may become either stenotic or insufficient or some degree of both. On occasion children are born with malfunctioning valves.

The valve involved and the type of defect can usually be detected by the location and timing of the murmur. Each heart valve can be heard best at a specific location on the chest (fig.17.17). Noting where a murmur is loudest helps the diagnostician tell which valve is involved.

The "timing" of the murmur refers to the part of the cardiac cycle during which the murmur is heard. Recall that the first heart sound signals the onset of ventricular systole, and the second

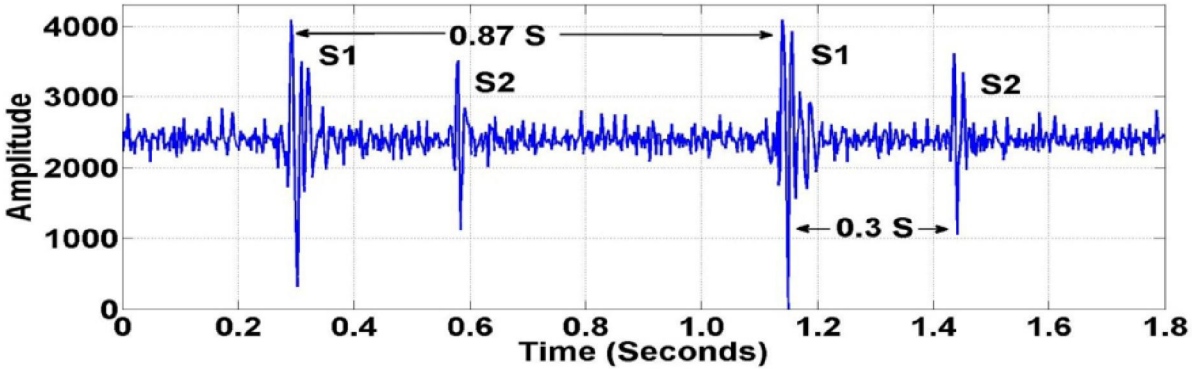
▲ TABLE 9-2 Timing and Type of Murmur Associated with Various Heart Valve Disorders

Pattern Heard on Auscultation	Type of Valve Defect	Timing of Murmur	Valve Disorder	Comment
Lub-Whistle-Dup	Stenotic	Systolic	Stenotic semilunar valve	A whistling systolic murmur signifies that a valve that should be open during systole (a semilunar valve) does not open completely.
Lub-Dup-Whistle	Stenotic	Diastolic	Stenotic AV valve	A whistling diastolic murmur signifies that a valve that should be open during diastole (an AV valve) does not open completely.
Lub-Swish-Dup	Insufficient	Systolic	Insufficient AV valve	A swishy systolic murmur signifies that a valve that should be closed during systole (an AV valve) does not close completely.
Lub-Dup-Swish	Insufficient	Diastolic	Insufficient semilunar valve	A swishy diastolic murmur signifies that a valve that should be closed during diastole (a semilunar valve) does not close completely.

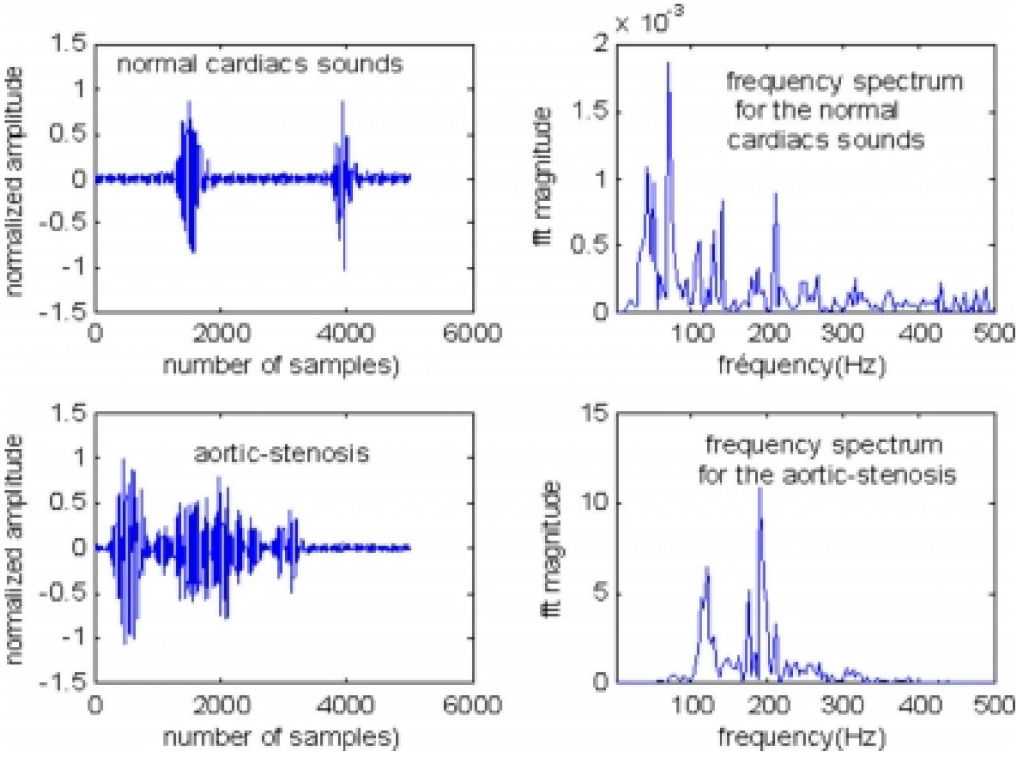
heart sound signals the onset of ventricular diastole. Thus, a murmur between the first and second heart sounds (lub-murmur-dup, lub-murmur-dup) is a **systolic murmur**. **A diastolic murmur**, in contrast, occurs between the second and first heart sound (lub-dup-murmur, lub-dupmurmur). The sound of the murmur characterizes it as either a **stenotic** (whistling) murmur or an **insufficient** (swishy) murmur. Armed with these facts, one can determine the cause of a valvular murmur (Table 9-2). As an example, a whistling murmur (denoting a stenotic valve) occurring between the first and second heart sounds (denoting a systolic murmur) indicates stenosis in a valve that should be open during systole. It could be either the aortic or the pulmonary semilunar valve through which blood is being ejected. Identifying which of these valves is stenotic is accomplished by finding where the murmur is best heard.

Phonocardiography is a method of graphical recording of the heart sounds using a special electrical microphone placed above the projection of the heart valves.

The typical normal phonocardiogram recorded on the heart apex is shown on the figure.



The pathological phonocardiogram recorded from a patient with aortic valve stenosis is shown below.

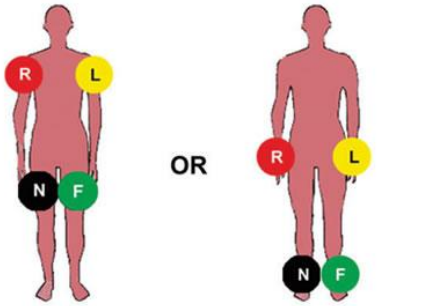


Phonocardiography allows to determine the localization and timing of the heart sounds and murmurs more precisely then an auscultation.

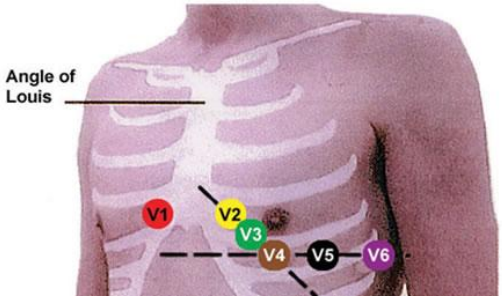
Physiological and technical aspects of electrocardiography.

Electrocardiography (ECG) is a method of recording of the heart electrical activity from the special electrodes attached to the surface of the skin on the standard places. Each pair of electrodes is considered as a "lead". The term lead may refer to **the tracing of the voltage difference between two of the electrodes** and is what is actually produced by the ECG recorder. Each lead have a specific name. A 12-lead ECG system is the most popular for today. Ten electrodes are used for a 12-lead ECG. The electrodes usually consist of a conducting gel, embedded in the middle of a self-adhesive pad onto which cables clip. Sometimes the gel also forms the adhesive. They are labeled and placed on the patient's body as follows.

RA
LA
RL



V1
V2
V3
V4
V5
V6



The limb electrodes

- On the right arm, avoiding thick muscle
– On the left arm this time.
- On the right leg, lateral calf muscle
LL- On the left leg this time.

The 6 chest electrodes

- Fourth intercostal space, right sternal border.
- Fourth intercostal space, left sternal border.
- Midway between V2 and V4.
- Fifth intercostal space, left midclavicular line.
- Level with V4, left anterior axillary line.
- Level with V4, left mid axillary line.

What means each of 12 leads of standard ECG?

LEADS I, II, III are formed by voltage tracing between the limb electrodes. These are only bipolar leads. All together they are called the **limb leads** or the Einthoven triangle (after name of scientist who had been recorded first ECG in 1903).

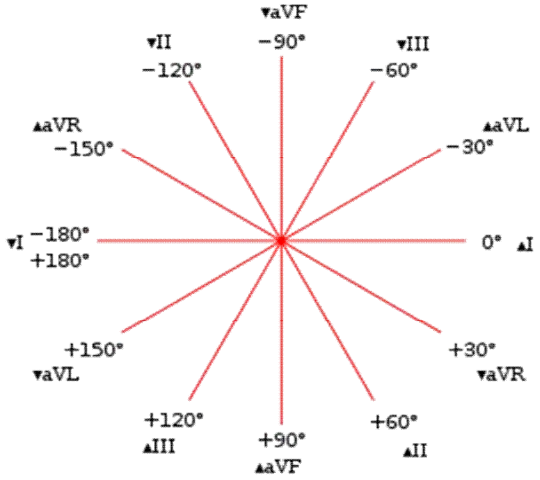
I lead is voltage tracing between the electrodes .RA(-) and LA(+)

II lead is voltage tracing between the electrodes .RA(-) and LL(+)

III lead is voltage tracing between the electrodes .LA(-) and LL(+)

LEADS aVR, aVL, aVF also derived from the limb electrodes. They measure the electric potential at the point where the are placed with respect to a null point, which is considered as "-" . Three other electrodes are considered as "+". These leads are called **augmented limb leads**.

LEADS V1,V2,V3,V4,V5,V6 are placed directly on the chest. Because of their close proximity of the heart, they do not require augmentation. Each electrode from this group is

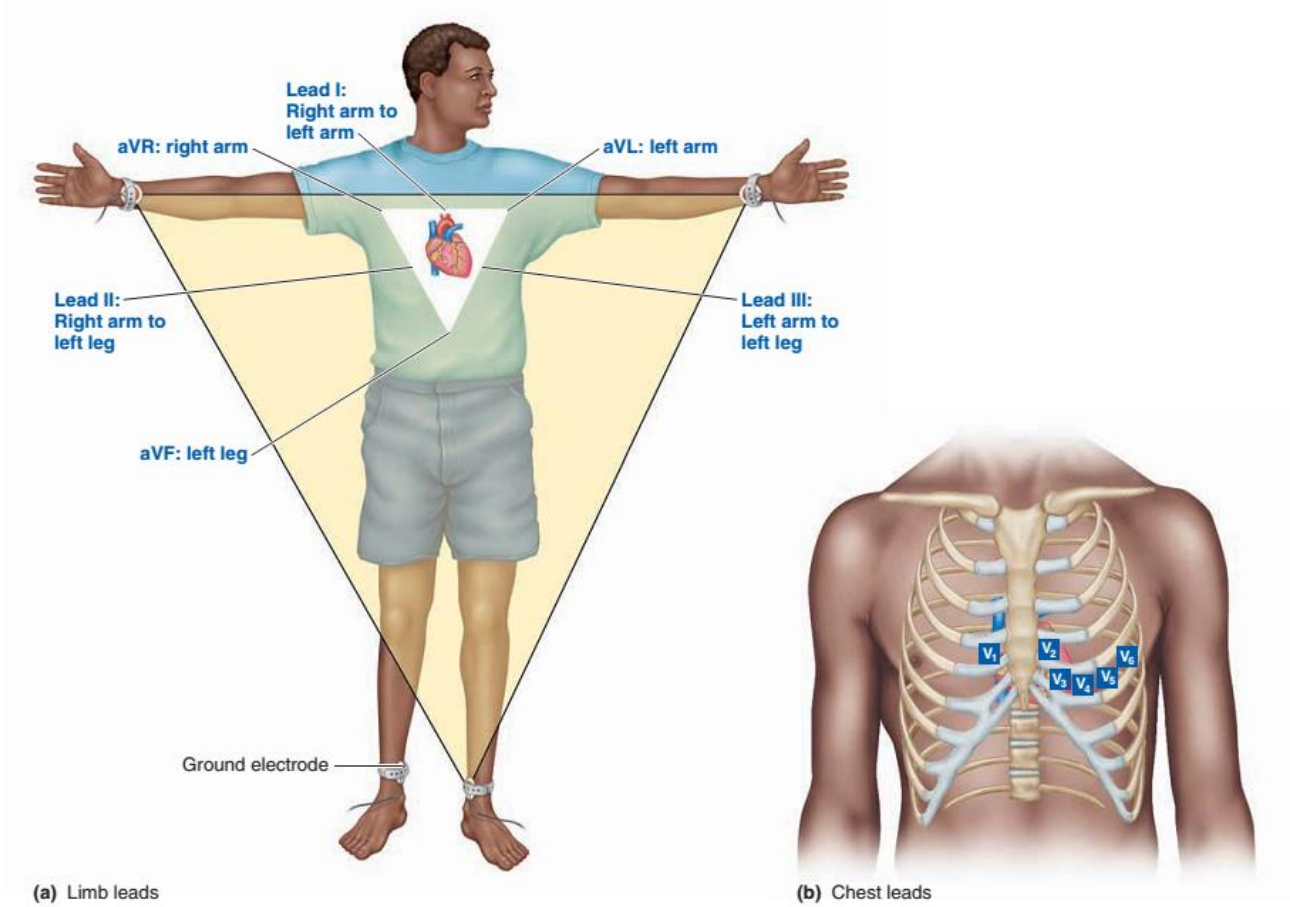


considered as “+”. They are called **precordial leads**.

Interpretation of the ECG relies on the idea that different leads (meaning the ECG leads I, II, III, aVR, aVL, aVF and the chest leads) "view" the heart from different angles. The concept of “lead axis” allows to use this idea in order to explain the form of ECG curve in each lead. Lead axis can be determined as an imaginary line connecting the places where electrodes are attached with point of **heart null potential**. This point divides each axis on positive and negative part depending on polarity of attached electrodes.

The hexaxial reference system showing the orientation of each lead is presented on the figure above. : For example, if the bulk of heart muscle is oriented at +60 degrees with respect to the SA node, lead II will show the greatest deflection and aVL the least.

Leads axes help to determine heart’s electrical axis. The limb leads and the augmented limb leads form **the frontal plane**. The precordial leads form **the horizontal plane**.



An accordance between leads and anatomical structure of heart is presented in the table. Changes in the group of leads reflect the topic of the myocardium damage.

Leads	Anatomical representation of the heart
V1, V2, V3, V4	Anterior
I, aVL, V5, V6	left lateral
II, III, aVF	inferior
aVR, V1	Right atrium

A concept of **integral heart vector** is useful for understanding of the ECG waveform. According to this concept, excitation of each individual cardiomyocyte can be considered as elementary electrical vector, which has certain direction and magnitude at each time point. The elementary vector reflects a potential difference between excited (-) and unexcited (+) cardiac fibers. A geometric sum of these elementary vectors forms the integral heart vector. This vector is changing during the heart cycle because of the wave of excitation travels throughout the heart

conduction system to contractile myocardium. Its direction depends from the excitation sequence of different heart parts, but its magnitude depends mainly from myocardium mass involved. There are 5 basic heart vectors during the cardiac cycle:

Vector P reflects the propagation of the action potential from sinoatrial node to atrioventricular node through atria. It directs from top to bottom, from right to left, and from back to front. It lasts about of 0,1 sec.

No integral vector is formed during next 0,05-0,1 sec, when the excitation travels along the conduction system between AV node and ventricular myocardium. The relative mass of conduction system is too small comparing to contractile myocardium (less then 1% total myocardium mass) in order to generate a distinguishable potential difference.

Vector Q reflects the depolarization of the interventricular septum. It directs from bottom to

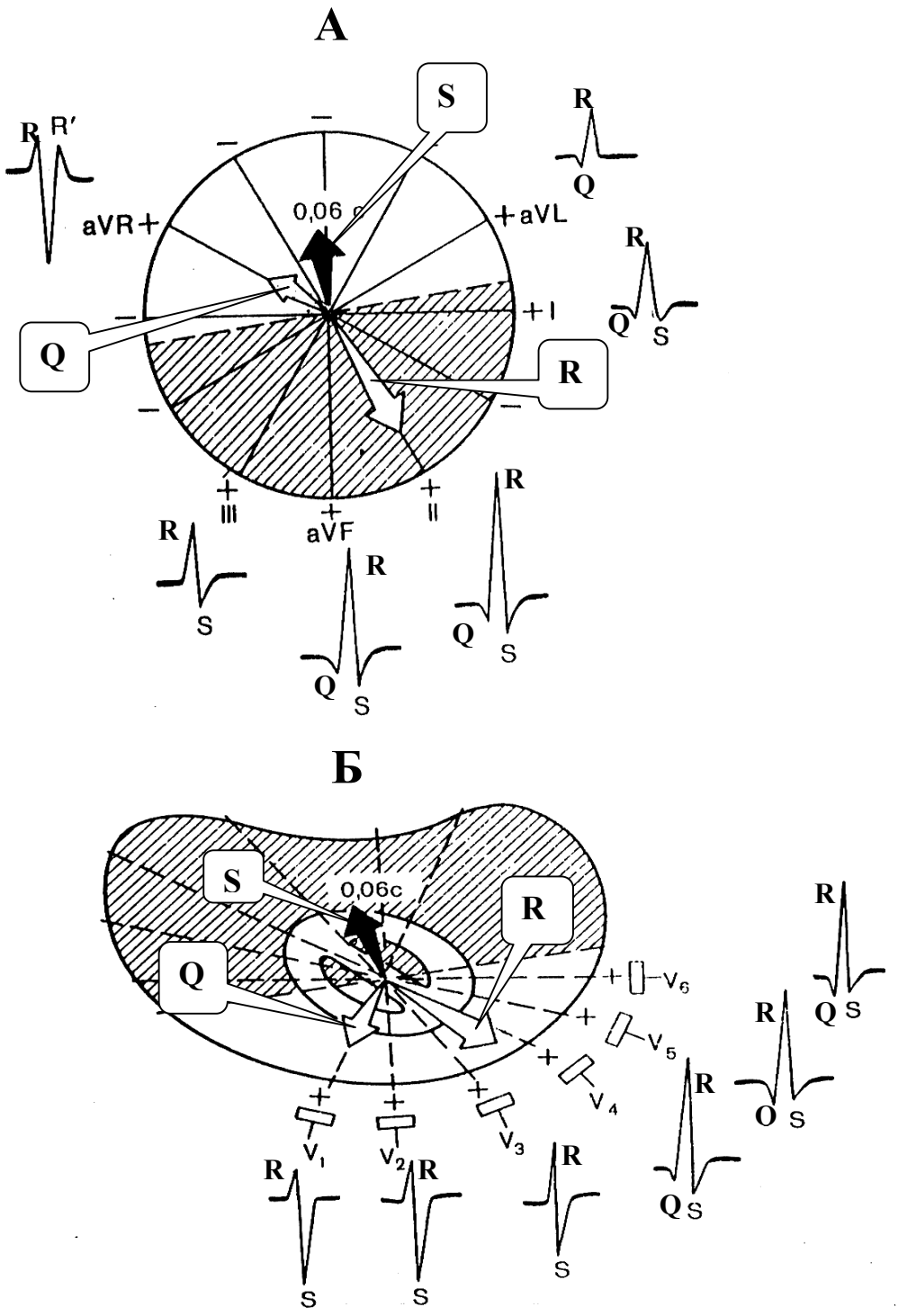


Fig.103. Forming of ECG ventricular complex by projection of the Q, R and S vectors on the axes of frontal and horizontal plane

top, from left to right, and from back to front. It lasts about of 0,02-0,04 sec.

Vector R is the biggest integral heart vector. It reflects the depolarization of the myocardium in main part left and right ventricles. It directs like vector P from top to bottom, from right to left, and from back to front. It lasts about of 0,04-0,06 sec.

Vector S reflects the depolarization of the myocardium located at base of left and right ventricles. It directs from bottom to top, from left to right, and from front to back. It lasts about of 0,03-0,05 sec.

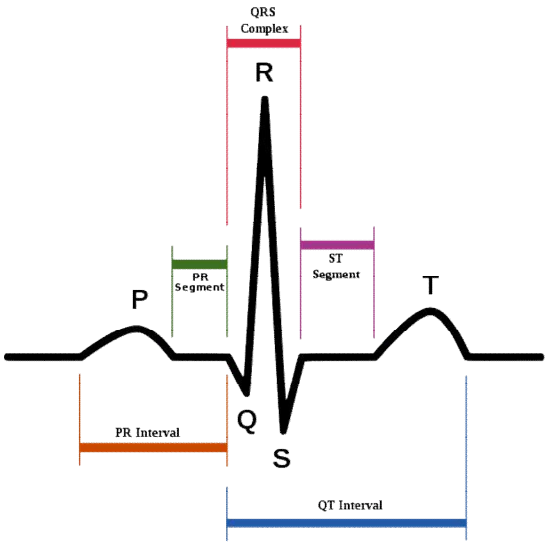
Next 0,1 sec all the ventricular fibers are in the plateau phase. It means that no potential difference between any myocardial fibers exists and accordingly no integral vector is formed.

Vector T, the last heart integral vector, reflects the repolarization wave of both ventricles. It directs like vector P and R from top to bottom, from right to left, and from back to front. It lasts about of 0,16-0,24 sec.

After the ending vector T the electrical pause occurs because of all contractile fibers are in rest phase and no potential difference between them exists.

ECG waveform is the tracing of the projection of integral heart vector on the lead's axis. (fig 103)., Positive-going deflection is recorded, if the projection refers to positive part of the axis. Negative-going deflection is recorded, if the projection refers to negative part of the axis. Because of different axis angles, each vector gives a different waveform of ECG for different leads. For example, R wave in I standard lead is positive, but in AVR - it is negative.

The typical ECG in a II standard limb lead and its main components are shown on the fig. below.



A normal ECG consists of a P wave, a QRS complex, and a T wave.

The P wave results from depolarization of the atrial myocardium, and the beginning of the P wave precedes the onset of atrial contraction.

The QRS complex consists of three individual waves: the Q, R, and S waves. The QRS complex results from depolarization of the ventricles, and the beginning of the QRS complex precedes ventricular contraction.

The T wave represents repolarization of the ventricles, and the beginning of the T wave precedes ventricular relaxation.

A wave representing repolarization of the atria cannot be seen because it occurs during the QRS complex.

The time between the beginning of the P wave and the beginning of the QRS complex is the PQ interval, commonly called the PR interval because the Q wave is very small. During the PQ interval the atria contract and begin to relax. At the end of the PQ interval the ventricles begin to depolarize.

The QT interval extends from the beginning of the QRS complex to the end of the T wave and represents the length of time required for ventricular depolarization and repolarization.

The ECG as a diagnostic tool.

The ECG is not a direct measurement of mechanical events in the heart, and neither the force of contraction nor the blood pressure can be determined from it. Each deflection in the ECG record, however, indicates an electrical event within the heart and correlates with a subsequent mechanical event. Consequently, it is an extremely valuable diagnostic tool in identifying a number of cardiac abnormalities, particularly because it is painless, is easy to record, and does not require surgical procedures.

Abnormal heart rates or rhythms; abnormal conduction pathways, such as blockages in the conduction pathways; hypertrophy or atrophy of portions of the heart; and the approximate location of damaged cardiac muscle can be determined from the analysis of an ECG