1. The Basics

In this chapter you will learn:

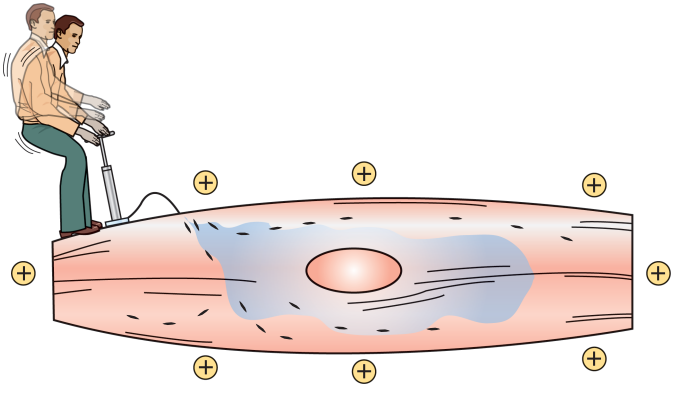
1. how the electrical current in the heart is generated
2. how this current is propagated through the four chambers of the heart
3. that the movement of electricity through the heart produces predictable wave patterns on the EKG
4. how the EKG machine detects and records these waves
5. that the EKG looks at the heart from 12 different perspectives, providing a remarkable three-dimensional electrical map of the heart
6. that you are now able to recognize and *understand* all the lines and waves on the 12-lead EKG
7. that trusting the EKG machine to interpret your patient's EKG is nothing but an invitation to trouble!

Electricity and the Heart

Electricity, an innate biologic electricity, is what makes the heart go. The EKG is nothing more than a recording of the heart's electrical activity, and it is through perturbations in the normal electrical patterns that we are able to diagnose many different cardiac and noncardiac disorders.

All You Need to Know About Cellular Electrophysiology in a Few Brief Paragraphs

Cardiac cells, in their resting state, are electrically polarized; that is, their insides are negatively charged with respect to their outsides. This electrical polarity is maintained by membrane pumps that ensure the appropriate distribution of ions (primarily potassium, sodium, chloride, and calcium) necessary to keep the insides of these cells relatively electronegative. These ions pass into and out of the cell through special ion channels in the cell membrane.



The resting cardiac cell maintains its electrical polarity by means of a membrane pump. This pump requires a constant supply of energy, and the gentleman above, were he real rather than a metaphor, would soon be flat on his back.

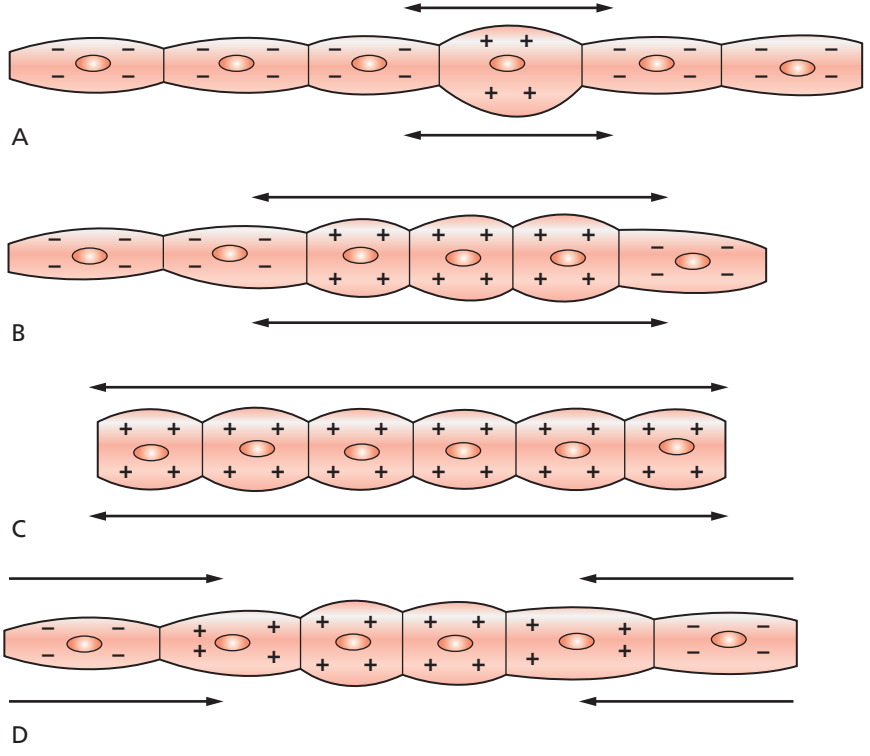
Sometimes lethal disturbances in the conduction of electricity through the heart occur because of an inherited disorder of these transmembrane ion channels. Fortunately, these so-called *channelopathies* are quite rare. Many different genetic mutations affecting the cardiac ion channels have been identified, and more are being discovered every year.

Cardiac cells can lose their internal negativity in a process called *depolarization*. **Depolarization is the fundamental electrical event of the heart**. In some cells, known as pacemaker cells, it occurs spontaneously. In others, it is initiated by the arrival of an electrical impulse that causes positively charged ions to cross the cell membrane.

Depolarization is propagated from cell to cell, producing a wave of depolarization that can be transmitted across the entire heart. This wave of depolarization represents a flow of electricity, an electrical current, that can be detected by electrodes placed on the surface of the body.

After depolarization is complete, the cardiac cells restore their resting polarity through a process called *repolarization*. Repolarization is accomplished by the membrane pumps, which reverse the flow of ions. This process can also be detected by recording electrodes.

All of the different waves that we see on an EKG are manifestations of these two processes: depolarization and repolarization.

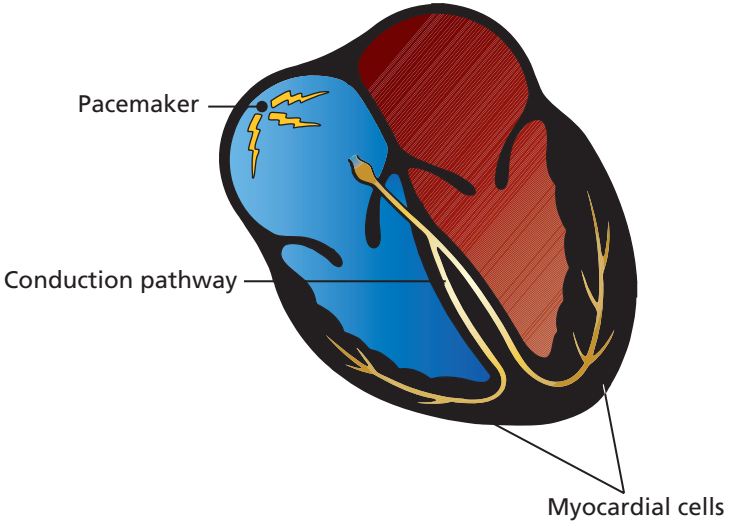


In (*A*), a single cell has depolarized. A wave of depolarization then propagates from cell to cell (*B*) until all are depolarized (*C*). Repolarization (*D*) then restores each cell's resting polarity.

The Cells of the Heart

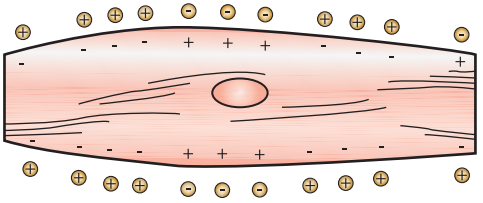
From the standpoint of the electrocardiographer, the heart consists of three types of cells:

- *Pacemaker cells*—under normal circumstances, the electrical power source of the heart
- *Electrical conducting cells*—the hard wiring of the heart
- *Myocardial cells*—the contractile machinery of the heart



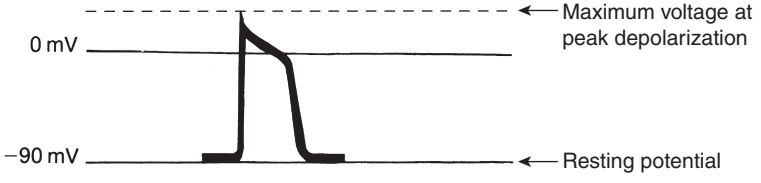
Pacemaker Cells

Pacemaker cells are small cells approximately 5 to 10 µm long, about the same as the width of a single strand of a spider's web. These cells are able to depolarize spontaneously over and over again. The rate of depolarization is determined by the innate electrical characteristics of the cell and by external neurohormonal input. Each spontaneous depolarization serves as the source of a wave of depolarization that initiates one complete cycle of cardiac contraction and relaxation.



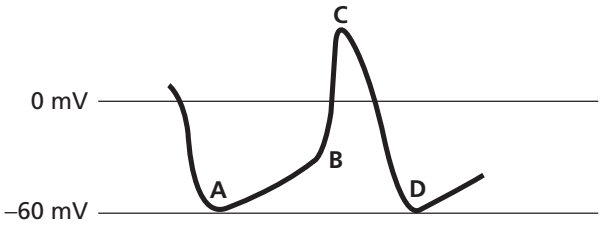
A pacemaker cell depolarizing spontaneously.

If we record one electrical cycle of depolarization and repolarization from a single cell, we obtain an electrical tracing called an **action potential**. With each spontaneous depolarization, a new action potential is generated, which in turn stimulates neighboring cells to depolarize and generate their own action potential, and so on and on, until the entire heart has been depolarized.



A typical action potential.

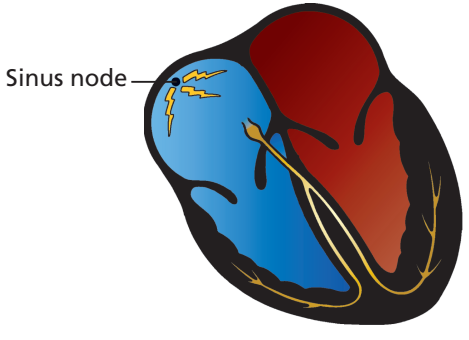
The action potential of a cardiac pacemaker cell looks a little different than the generic action potential shown here. A pacemaker cell does *not* have a true resting potential. Its electrical charge drops to a minimal negative potential of approximately –60 mV, which it maintains for just a moment (it does not rest there), and then gradually rises until it reaches the threshold for the sudden depolarization that is an action potential. These events are illustrated on the following tracing.



The electrical depolarization–repolarization cycle of a cardiac pacemaker cell. Point *A* is the minimal negative potential. The gentle rising slope between points *A* and *B* represents a slow, gradual depolarization. At point *B*, the threshold is crossed and the cell dramatically depolarizes (as seen between points *B* and *C*); that is, an action potential is produced. The downslope between points *C* and *D* represents repolarization. This cycle will repeat over and over for, let us hope, many, many years.

The dominant pacemaker cells in the heart are located high up in the right atrium. This group of cells is called the **sinoatrial (SA) node**, or **sinus node** for short. These cells typically fire at a rate of 60 to 100 times per minute, but the rate can vary tremendously depending upon the activity of the autonomic nervous system (*e.g.*, sympathetic stimulation from catecholamines, such as epinephrine and norepinephrine, accelerates the sinus node, whereas vagal stimulation slows it) and the demands of the body for increased cardiac output (exercise raises the heart rate, whereas a restful afternoon nap lowers it).

Pacemaker cells are really good at what they do. They will continue firing in a donor heart even after it has been harvested for transplant and before it has been connected to the new recipient. The transplanted heart, devoid of normal vagal stimulation (the nerves are cut when the new heart is implanted), beats at an average rate of 100 beats per minute (bpm).



In a resting individual, the sinus node typically fires 60 to 100 times per minute, producing a regular series of action potentials, each of which initiates a wave of depolarization that will spread through the heart.

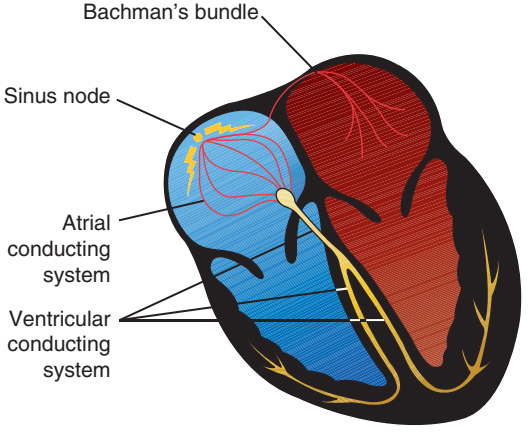
Actually, *every* cell in the heart has the ability to behave like a pacemaker cell. This so-called *automatic ability* is normally suppressed unless the dominant cells of the sinus node fail or if something in the internal or external environment of a cell (sympathetic stimulation, cardiac disease, *etc.*) stimulates its automatic behavior. This topic assumes greater importance later on and is discussed under *Ectopic Rhythms* in Chapter 3.

Electrical Conducting Cells

Electrical conducting cells are long, thin cells. Like the wires of an electrical circuit, these cells carry current rapidly and efficiently to distant regions of the heart. They are, in effect, the electrical highway of the heart.

The electrical conducting cells of the ventricles form distinct electrical pathways. The ventricular conducting fibers constitute what is called the *Purkinje system*.

The conducting pathways in the atria have more anatomic variability; prominent among these are fibers at the top of the intra-atrial septum in a region called Bachmann's bundle that allow for rapid activation of the left atrium from the right.

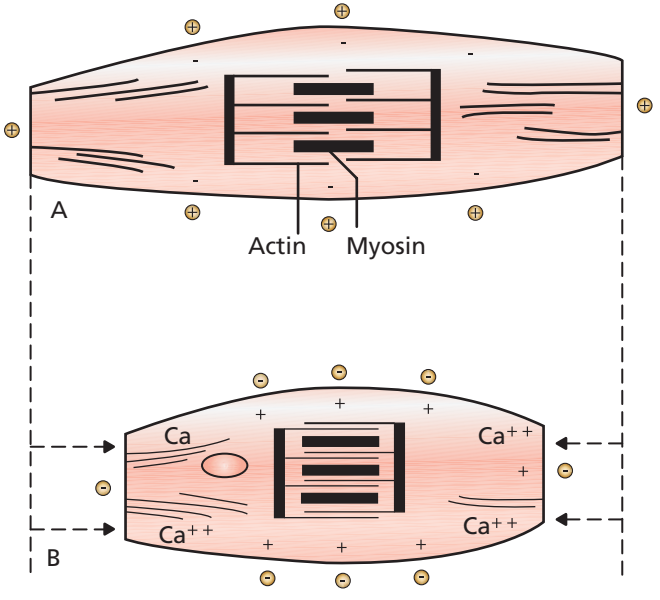


The hard wiring of the heart.

Myocardial Cells

The *myocardial cells* constitute by far the largest part of the heart tissue. They are responsible for the heavy labor of repeatedly contracting and relaxing, thereby delivering blood to the rest of the body. These cells are about 50 to 100 μm in length and contain an abundance of the contractile proteins actin and myosin.

When a wave of depolarization reaches a myocardial cell, calcium is released within the cell, causing the cell to contract. This process, in which calcium plays the key intermediary role, is called *excitation–contraction coupling*.



Depolarization causes calcium to be released within a myocardial cell. This influx of calcium allows actin and myosin, the contractile proteins, to interact, causing the cell to contract. (*A*) A resting myocardial cell. (*B*) A depolarized, contracted myocardial cell.

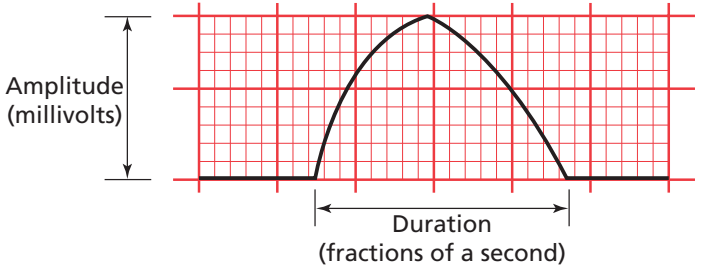
Myocardial cells can transmit an electrical current just like electrical conducting cells, but they do so far less efficiently. Thus, a wave of depolarization, upon reaching the myocardial cells, will spread slowly across the entire myocardium.

Time and Voltage

The waves that appear on an EKG primarily reflect the electrical activity of the *myocardial cells*, which make up the vast bulk of the heart. Pacemaker activity and transmission by the conducting system are generally not seen on the EKG; these events simply do not generate sufficient voltage to be recorded by surface electrodes.

The waves produced by myocardial depolarization and repolarization are recorded on EKG paper and, like any wave, have three chief characteristics:

1. *Duration*, measured in fractions of a second
2. *Amplitude*, measured in millivolts (mV)
3. *Configuration*, a more subjective criterion referring to the shape and appearance of a wave



A typical wave that might be seen on any EKG. It is two large squares (or 10 small squares) in amplitude, three large squares (or 15 small squares) in duration, and slightly asymmetric in configuration.

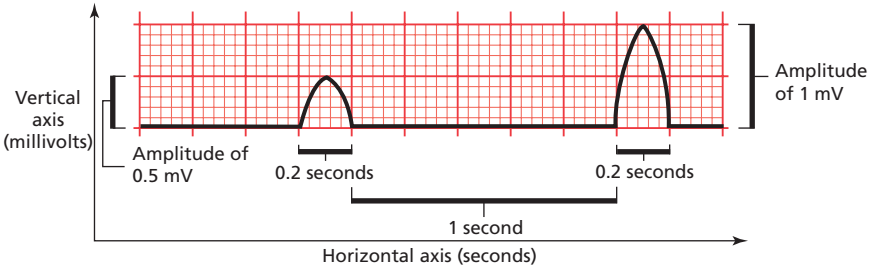
EKG Paper

EKG paper is a long, continuous roll of graph paper, usually pink (but any color will do), with light and dark lines running vertically and horizontally. The light lines circumscribe small squares of 1 × 1 mm; the dark lines delineate large squares of 5 × 5 mm.

The horizontal axis measures time. The distance across one small square represents 0.04 seconds. The distance across one large square is five times greater, or 0.2 seconds.

The vertical axis measures voltage. The distance along one small square represents 0.1 mV, and along one large square, 0.5 mV.

You will need to memorize these numbers at some point, so you might as well do it now.



Both waves are one large square in duration (0.2 seconds), but the second wave is twice the voltage of the first (1 mV compared with 0.5 mV). The flat segment connecting the two waves is five large squares (5 × 0.2 seconds = 1 second) in duration.

P Waves, QRS Complexes, T Waves, and Some Straight Lines

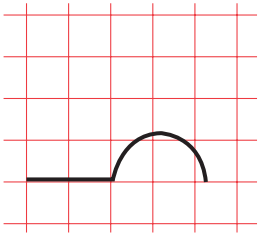
Let's follow one cycle of cardiac contraction (systole) and relaxation (diastole), focusing on the electrical events that produce the basic waves and lines of the standard EKG.

Atrial Depolarization

The sinus node fires spontaneously (an event not visible on the EKG), and a wave of depolarization begins to spread outward into the atrial myocardium, much as if a pebble were dropped into a calm lake. Depolarization of the atrial myocardial cells results in atrial contraction.

Each cycle of normal cardiac contraction and relaxation begins when the sinus node depolarizes spontaneously. The wave of depolarization then propagates through both atria, causing them to contract.

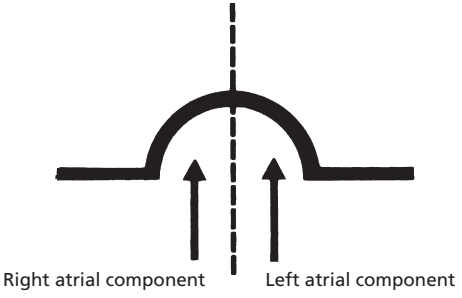
During atrial depolarization and contraction, electrodes placed on the surface of the body record a small burst of electrical activity lasting a fraction of a second. This is the *P wave*. It is a recording of the spread of depolarization through the atrial myocardium from start to finish.



The EKG records a small deflection, the P wave.

Because the sinus node is located in the right atrium, the right atrium begins to depolarize before the left atrium and finishes earlier as well. Therefore, the first part of the P wave predominantly represents right atrial depolarization, and the second part left atrial depolarization.

Once atrial depolarization is complete, the EKG again becomes electrically silent.



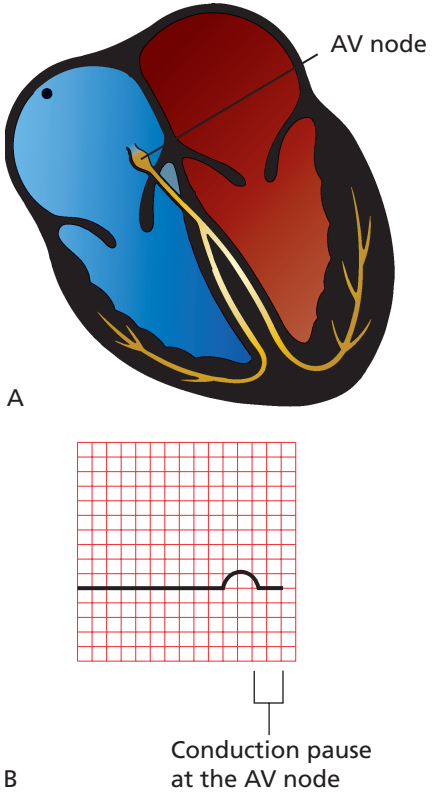
The components of the P wave.

A Pause Separates Conduction From the Atria to the Ventricles

In healthy hearts, there is an electrical gate at the junction of the atria and the ventricles. The wave of depolarization, having completed its journey through the atria, is prevented from communicating with the ventricles by the heart valves that separate the atria and ventricles. Electrical conduction must be funneled along the interventricular septum, the wall that separates the right and left ventricles. Here, a structure called the *atrioventricular* (*AV*) *node* slows conduction to a crawl. This pause lasts only a fraction of a second.

This physiologic delay in conduction is essential to allow the atria to finish contracting before the ventricles begin to contract. This clever electrical wiring of the heart permits the atria to empty their volume of blood completely into the ventricles before the ventricles contract.

Like the sinus node, the AV node is also under the influence of the autonomic nervous system. Vagal stimulation slows the current even further, prolonging the delay, whereas sympathetic stimulation accelerates the current through the AV node.



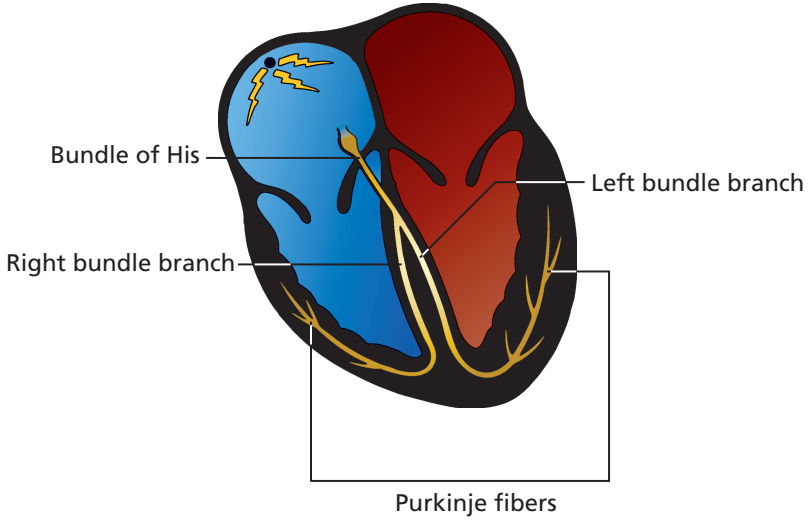
(*A*) The wave of depolarization is briefly held up at the AV node. (*B*) During this pause, the EKG falls silent; there is no detectable electrical activity.

Ventricular Depolarization

After about one-tenth of a second, the depolarizing wave escapes the AV node and is swept rapidly down the ventricles along specialized electrical conducting cells.

This ventricular conducting system has a complex anatomy but essentially consists of three parts:

1. Bundle of His
2. Bundle branches
3. Terminal *Purkinje fibers*

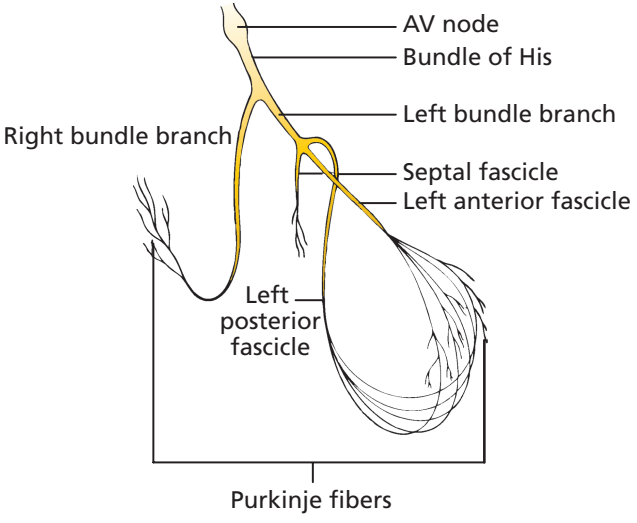


The *bundle of His* emerges from the AV node and almost immediately divides into right and left bundle branches. The *right bundle branch* carries the current down the right side of the interventricular septum all the way to the apex of the right ventricle. The *left bundle branch* is more complicated. It divides into three major fascicles:

1. *Septal fascicle*, which depolarizes the interventricular septum (the wall of muscle separating the right and left ventricles) in a left-to-right direction

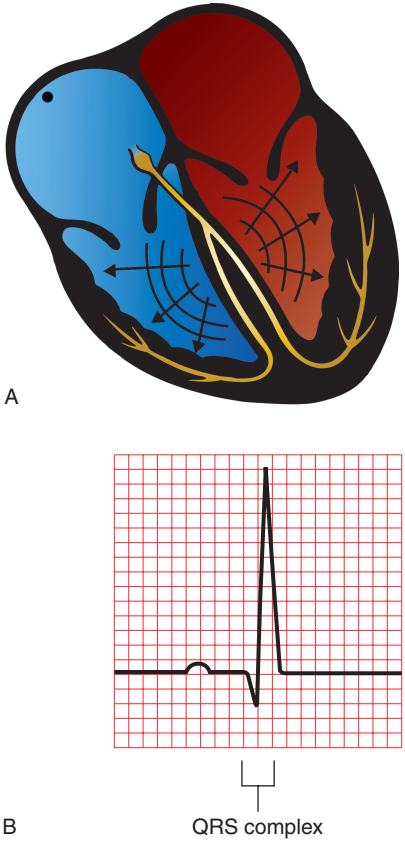
2. *Anterior fascicle*, which runs along the anterior wall of the left ventricle
3. *Posterior fascicle*, which sweeps over the posterior wall of the left ventricle

The right bundle branch and the left bundle branch and its fascicles terminate in countless tiny Purkinje fibers, which resemble little twigs coming off the branches of a tree. These fibers deliver the electrical current into the ventricular myocardium.



The ventricular conduction system, shown in detail. Below the bundle of His, the conduction system divides into right and left bundle branches. The right bundle branch remains intact, whereas the left divides into three separate fascicles.

Ventricular myocardial depolarization causes ventricular contraction. It is marked by a large deflection on the EKG called the *QRS complex*. The amplitude of the QRS complex is much greater than that of the atrial P wave because the ventricles have so much more muscle mass than the atria. The QRS complex is also more complicated and variable in shape than the P wave, reflecting the greater intricacy of the pathway of ventricular depolarization.



(*A*) Ventricular depolarization generates (*B*) a complicated waveform on the EKG called the QRS complex.

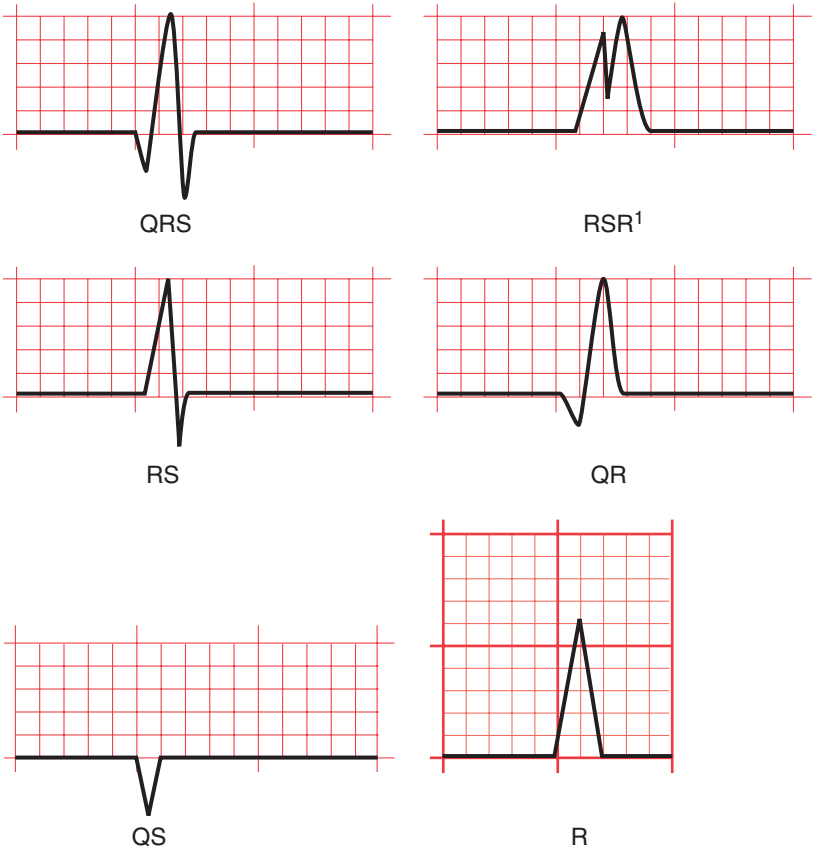
The Parts of the QRS Complex

The QRS complex consists of several distinct waves, each of which has a name. Because the precise configuration of the QRS complex can vary so greatly, a standard format for naming each component has been devised. It may seem a bit arbitrary to you right now, but it actually makes good sense.

1. If the first deflection is downward, that part of the QRS complex is called a *Q wave*.
2. The first upward deflection is called an *R wave*.
3. If there is a second upward deflection, it is called *R′* ("R-prime").

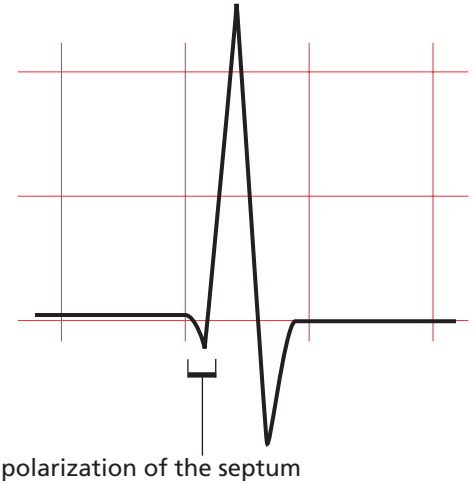
4. The first downward deflection following an upward deflection is called an *S wave*. Therefore, if the first wave of the complex is an R wave, the ensuing downward deflection is called an S wave, not a Q wave. A downward deflection can only be called a Q wave if it is the first wave of the complex. Any other downward deflection is called an S wave.
5. If the entire configuration consists solely of one downward deflection, the wave is called a *QS wave*.

Here are several of the most common QRS configurations, with each wave component named.



The earliest part of the QRS complex represents depolarization of the interventricular septum by the septal fascicle of the left bundle

branch. The right and left ventricles then depolarize at about the same time, but most of what we see on the EKG represents left ventricular activation because the muscle mass of the left ventricle is about three times that of the right ventricle.



The initial part of the QRS complex represents septal depolarization. Sometimes, this septal depolarization may appear as a small, discrete, negative deflection, a Q wave.

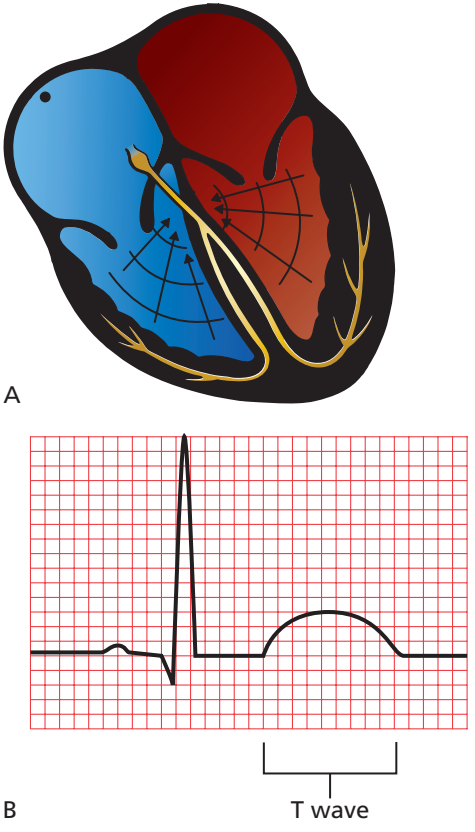
Repolarization

After myocardial cells depolarize, they pass through a brief refractory period during which they are resistant to further stimulation. They then *repolarize*; that is, they restore the electronegativity of their interiors so that they can be restimulated.

Just as there is a wave of depolarization, there is also a wave of repolarization. This, too, can be seen on the EKG. Ventricular repolarization inscribes a third wave on the EKG, the *T wave*.

Note: There is a wave of atrial repolarization as well, but it coincides with ventricular depolarization and is hidden by the much more prominent QRS complex.

Ventricular repolarization is a much slower process than ventricular depolarization. Therefore, the T wave is broader than the QRS complex. Its configuration is also simpler and more rounded, like the silhouette of a gentle hill compared to the sharp, jagged, and often intricate contour of the QRS complex. Except with certain pathologic conditions, which we will explore later, the voltage of the T wave is less than that of the Q wave.

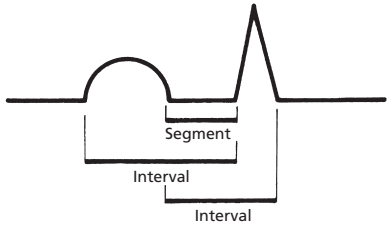


(*A*) Ventricular repolarization generates (*B*) a T wave on the EKG.

Naming the Straight Lines

The different straight lines connecting the various waves have also been given names. Thus, we speak of the *PR interval*, the *ST segment*, the *QT interval*, and so on.

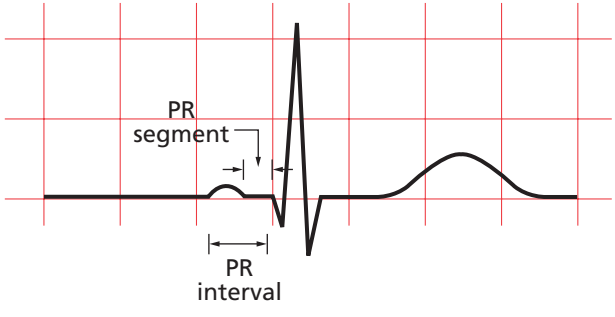
What differentiates a segment from an interval? A segment is a straight line connecting two waves, whereas an interval encompasses at least one wave plus, in most instances, the connecting straight line.



The *PR interval* includes the P wave and the straight line connecting it to the QRS complex. It therefore measures the time from the start of atrial depolarization to the start of ventricular depolarization.

The *PR segment* is the straight line running from the end of the P wave to the start of the QRS complex. It therefore measures the time from the end of atrial depolarization to the start of ventricular depolarization.

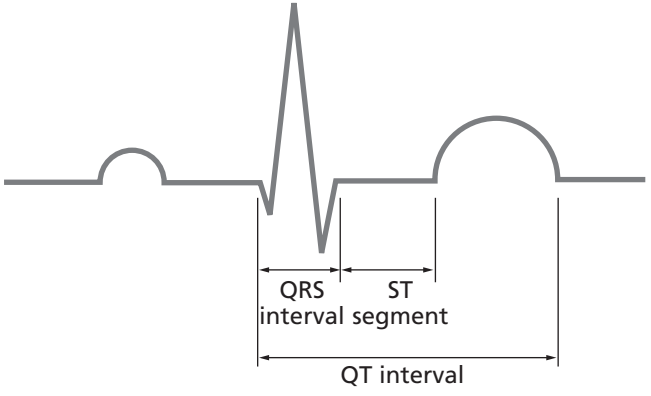
You might be wondering: if the QRS complex has a Q wave, shouldn't the PR segment be called the PQ segment, and the PR interval the PQ interval? Well, yes, but they're not. These straight lines are always called the PR segment and PR interval no matter what the configuration of the QRS complex.



The *ST segment* is the straight line connecting the end of the QRS complex with the beginning of the T wave. It measures the time from the end of ventricular depolarization to the start of ventricular repolarization.

The *QT interval* includes the QRS complex, the ST segment, and the T wave. It therefore measures the time from the beginning of ventricular depolarization to the end of ventricular repolarization. And, yes, the term QT interval is used even if a particular QRS complex does not have a Q wave.

The term *QRS interval* is used to describe the duration of the QRS complex alone without any connecting segments. Obviously, it measures the duration of ventricular depolarization.



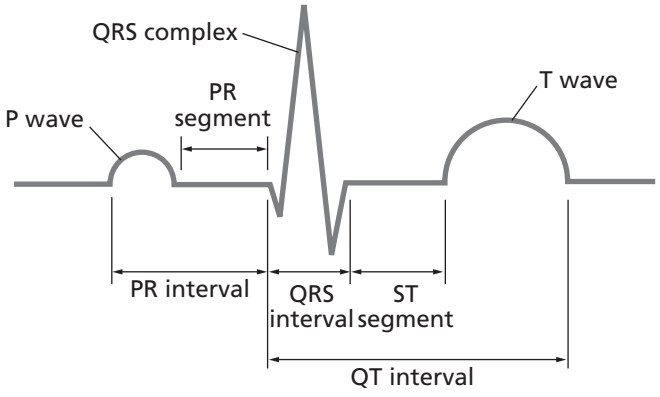
SUMMARY

The Waves and Straight Lines of the EKG

1. Each cycle of cardiac contraction and relaxation is initiated by spontaneous depolarization of the sinus node. This event is not seen on the EKG.
2. The P wave records atrial depolarization and contraction. The first part of the P wave reflects right atrial activity; the second part reflects left atrial activity.
3. There is a brief pause when the electrical current reaches the AV node and the EKG falls silent (the PR segment).
4. The wave of depolarization then spreads along the ventricular conducting system (bundle of His, bundle branches, and Purkinje fibers) and out into the ventricular myocardium. The first part of the ventricles to be depolarized is the interventricular septum. Ventricular depolarization generates the QRS complex.
5. The T wave records ventricular repolarization. Atrial repolarization is not seen.
6. Various segments and intervals describe the time between these events:
   a. The PR interval measures the time from the start of atrial depolarization to the start of ventricular depolarization.
   b. The PR segment measures the time from the end of atrial depolarization to the start of ventricular depolarization.
   c. The ST segment records the time from the end of ventricular depolarization to the start of ventricular repolarization.

d. The QT interval measures the time from the start of ventricular depolarization to the end of ventricular repolarization.

e. The QRS interval measures the time of ventricular depolarization.

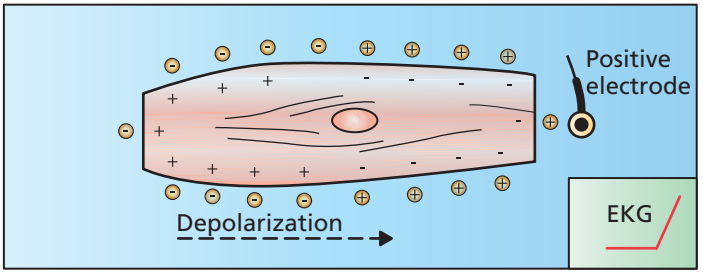


Making Waves

Electrodes can be placed anywhere on the surface of the body to record the heart's electrical activity. If we do this, we quickly discover that the waves recorded by a positive electrode on the left arm look very different from those recorded by a positive electrode on the right arm (or right leg, left leg, *etc.*).

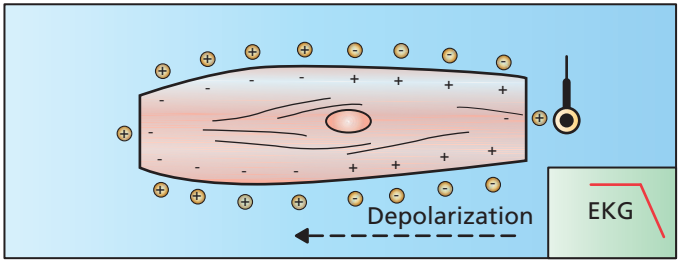
It's easy to see why. A wave of depolarization moving *toward* a positive electrode causes a *positive* deflection on the EKG. A wave of depolarization moving *away* from a positive electrode causes a *negative* deflection.

Look at the figure below. The wave of depolarization is moving left to right, *toward* the electrode. The EKG records a positive deflection.



A wave of depolarization moving toward a positive electrode records a positive deflection on the EKG.

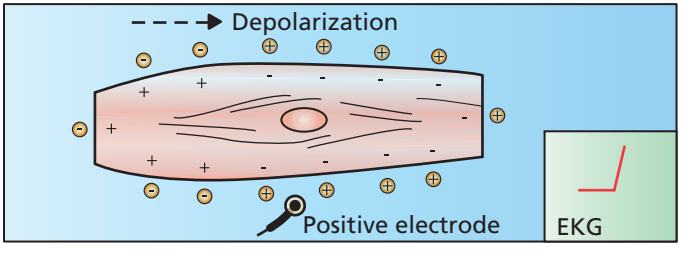
Now look at the following figure. The wave of depolarization is moving right to left, *away* from the electrode. The EKG therefore records a negative deflection.



A wave of depolarization moving away from a positive electrode records a negative deflection on the EKG.

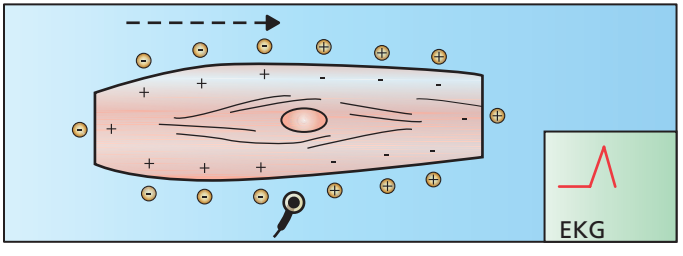
What will the EKG record if the positive electrode is placed near the middle of the cell?

Initially, as the wavefront approaches the electrode, the EKG records a positive deflection.



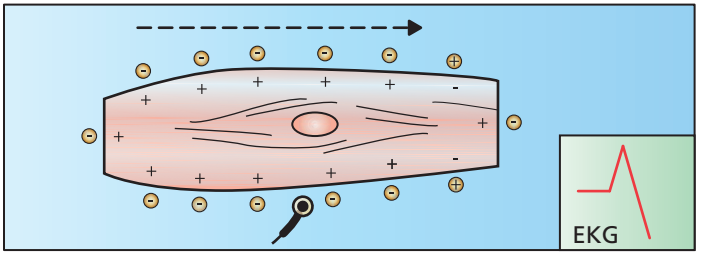
Depolarization begins, generating a positive deflection on the EKG.

Then, at the precise moment that the wave reaches the electrode, the positive and negative charges are balanced and essentially cancel each other out. The EKG recording returns to baseline.



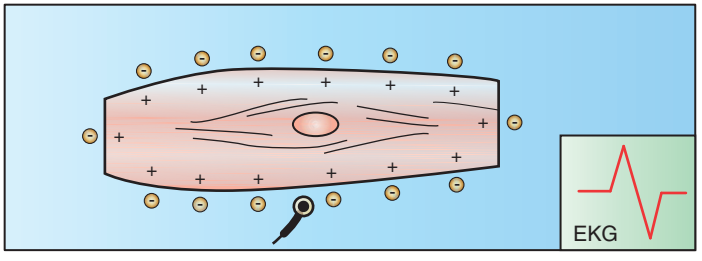
The wavefront reaches the electrode. The positive and negative charges are balanced, and the EKG returns to baseline.

As the wave of depolarization recedes, a negative deflection is inscribed.



The wave of depolarization begins to recede from the electrode, generating a negative deflection.

The EKG finally returns to baseline once again when depolarization is complete.

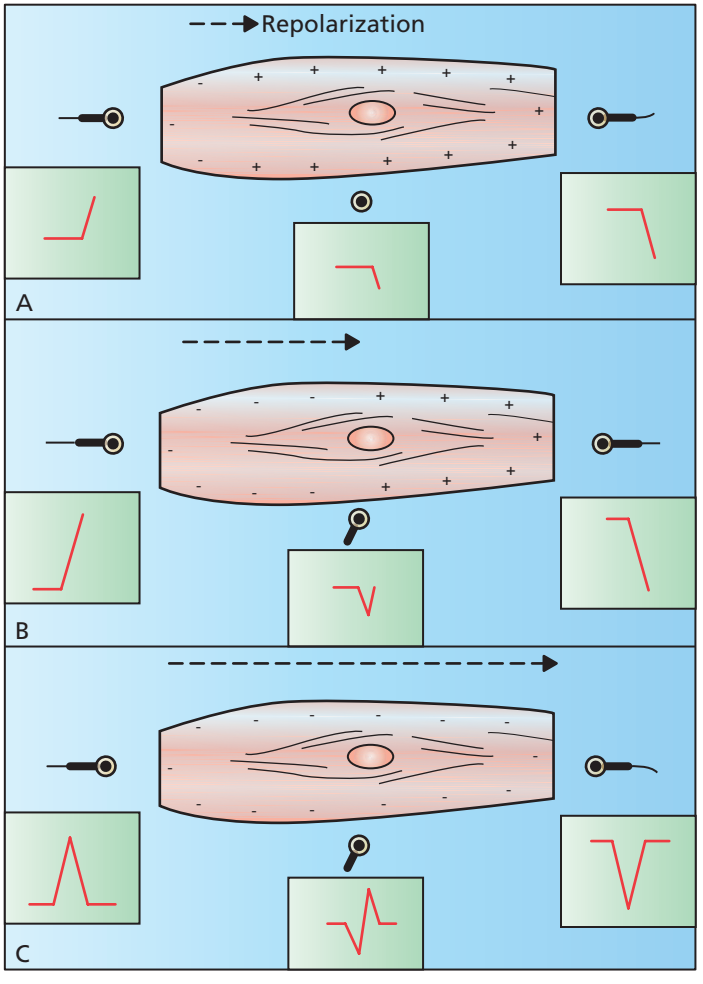


The cell is fully depolarized, and the EKG once again returns to baseline.

The final inscription of a depolarizing wave moving perpendicularly to a positive electrode is therefore a *biphasic wave*.

What would the tracing look like if the recording electrode were placed over a region of pacemaker cells sufficient to generate a detectable current? The tracing would show a downward, negative deflection, since all the current is moving away from the origin where you are recording.

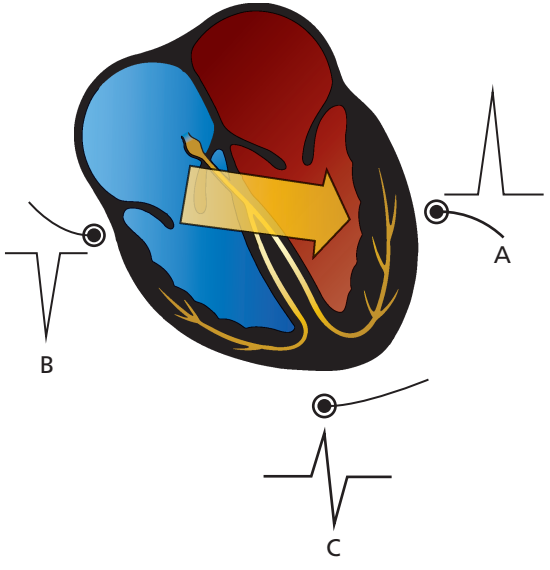
The effects of repolarization on the EKG are similar to those of depolarization, except that the charges are reversed. A wave of repolarization moving *toward* a positive electrode inscribes a *negative* deflection on the EKG. A wave of repolarization moving *away* from a positive electrode produces a *positive* deflection on the EKG. A perpendicular wave produces a *biphasic wave;* however, the negative deflection of the biphasic wave now *precedes* the positive deflection.



A wave of repolarization moving through muscle tissue is recorded by three different positive electrodes: (*A*) Early repolarization. (*B*) Late repolarization. (*C*) Repolarization is complete.

We can easily apply these concepts to the entire heart. Electrodes placed on the surface of the body will record waves of depolarization and repolarization as they sweep through the heart.

If a wave of depolarization passing through the heart is moving toward a surface electrode, that electrode will record a positive deflection (electrode *A*). If the wave of depolarization is moving away from the electrode, the electrode will record a negative deflection (electrode *B*). If the wave of depolarization is moving perpendicularly to the electrode, the electrode will record a biphasic wave (electrode *C*). The effects of repolarization are precisely the opposite of those of depolarization, as you would expect.



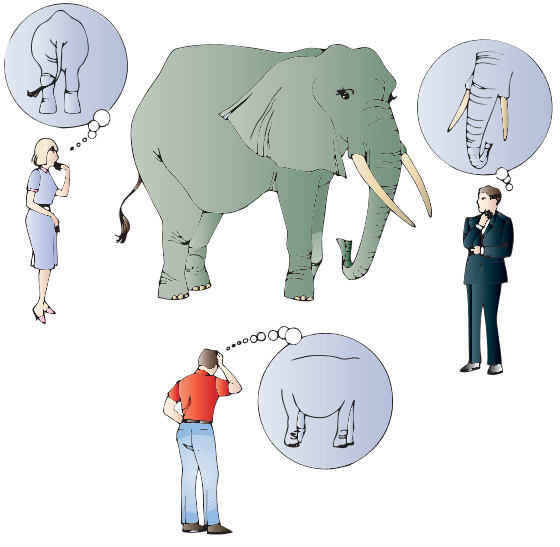
A wave of depolarization moving through the heart (*large arrow*). Electrode *A* records a positive deflection, electrode *B* records a negative deflection, and electrode *C* records a biphasic wave.

The 12 Views of the Heart

If the heart were as simple as a single myocardial cell, a couple of recording electrodes would give us all the information we need to describe its electrical activity. However, as we have already seen, the heart is *not* so simple—a burden to you, a boon to authors of EKG books.

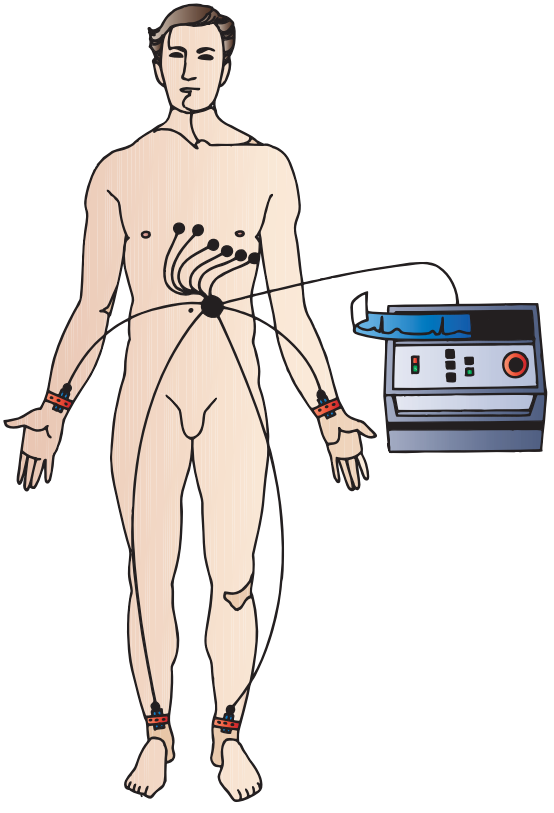
The heart is a three-dimensional organ, and its electrical activity must be understood in three dimensions as well. A couple of electrodes are not adequate to do this, a fact that the original electrocardiographers recognized well over a century ago when they devised the first limb leads. Today, the standard EKG consists of 12 leads, with each lead determined by the placement and orientation of various electrodes on the body. Each lead views the heart at a unique angle, enhancing its sensitivity to a particular region of the heart at the expense of others. The more views, the more the information provided.

To read an EKG and extract as much information as possible, you need to understand the 12-lead system.



Three curious observers get three very different impressions of this consummate example of the *Loxodonta africana*. One observer sees the trunk, another sees the body, and the third sees the tail. If you wanted the best description of the elephant, who would you ask? All three, of course.

To prepare a patient for a 12-lead EKG, two electrodes are placed on the arms and two on the legs. These provide the basis for the six *limb leads*, which include the three *standard leads* and the three *augmented leads* (these terms will make more sense in a moment). Six electrodes are also placed across the chest, forming the six *precordial leads*.

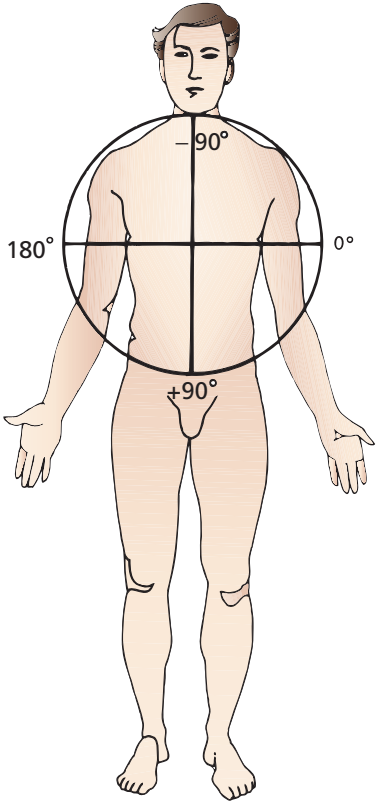


The electrical recordings will vary depending on the precise placement of the electrodes. Therefore, adherence to standard positioning protocols is very important to allow comparison between EKGs taken at different times in different settings.

The Six Limb Leads

The limb leads are created by putting electrodes on all four extremities. They view the heart in a vertical plane called the *frontal plane*. The frontal plane can be envisioned as a giant circle superimposed on the

patient's body. This circle is then marked off in degrees. The limb leads view electrical forces (waves of depolarization and repolarization) moving up and down and left and right through this circle.



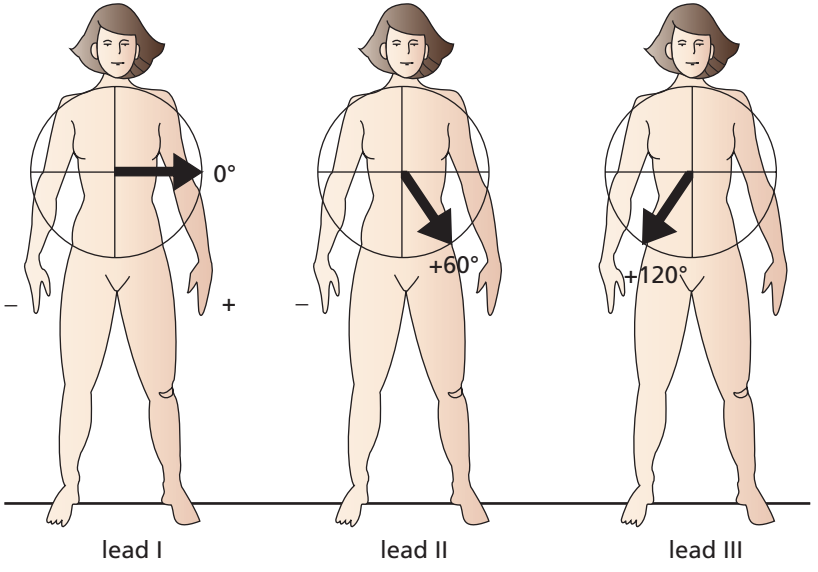
The frontal plane is a coronal plane. The limb leads view electrical forces moving up and down and left and right on the frontal plane.

To produce the six leads of the frontal plane, each of the electrodes is variably designated as positive or negative (this is done automatically by circuitry inside the EKG machine).

Each lead has its own specific view of the heart, or *angle of orientation*. The angle of each lead can be determined by drawing a line from the negative electrode(s) to the positive electrode(s). The resultant angle is then expressed in degrees by superimposing it on the 360° circle of the frontal plane. This is far less complicated than it sounds. Let's look at each limb lead individually.

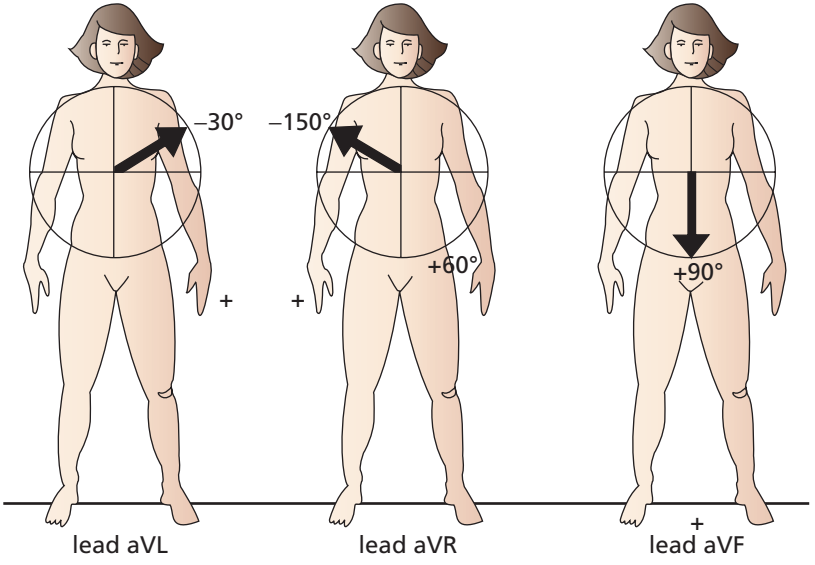
The three standard limb leads are defined as follows:

1. Lead I is created by making the left arm positive and the right arm negative. Its angle of orientation is 0°.
2. Lead II is created by making the legs positive and the right arm negative. Its angle of orientation is 60°.
3. Lead III is created by making the legs positive and the left arm negative. Its angle of orientation is 120°.

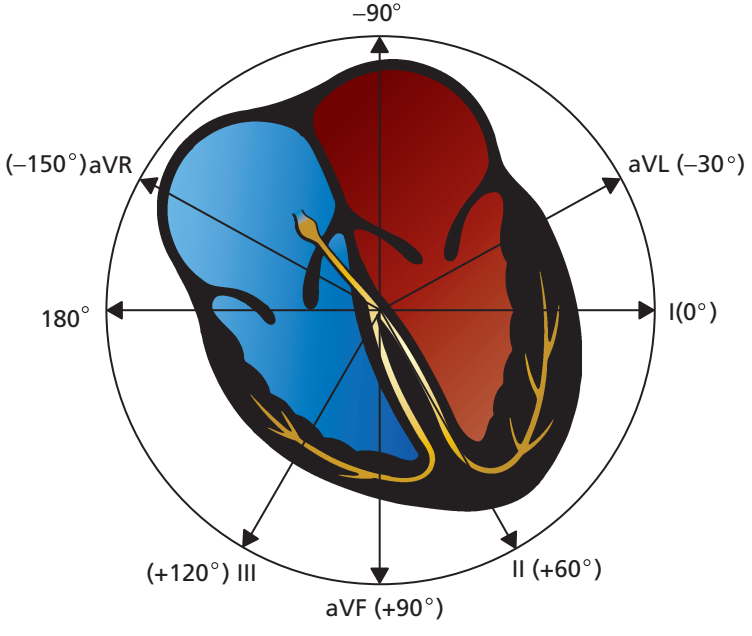


The three augmented limb leads are created somewhat differently. A single lead is chosen to be positive, and all the others are made negative, with their average essentially serving as the negative electrode (common ground). They are called *augmented leads* because the EKG machinery must amplify the tracings to get an adequate recording.

1. Lead aVL is created by making the left arm positive and the other limbs negative. Its angle of orientation is –30°.
2. Lead aVR is created by making the right arm positive and the other limbs negative. Its angle of orientation is –150°.
3. Lead aVF is created by making the legs positive and the other limbs negative. Its angle of orientation is +90°.



In the next figure, all six leads of the frontal plane are indicated with their appropriate angles of orientation. Just as our three inquisitive observers each looked at the elephant from his or her own unique perspective, each lead perceives the heart from its own unique point of view.

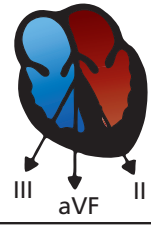
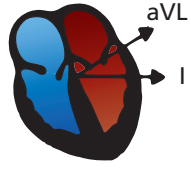
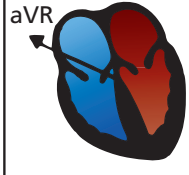


Leads II, III, and aVF are called the *inferior leads* because they most effectively view the inferior surface of the heart. The inferior surface, or wall, of the heart is an anatomic term for the bottom of the heart, the portion that rests on the diaphragm.

Leads I and aVL are often called the *left lateral leads* because they have the best view of the left lateral wall of the heart.

aVR is a loner among the limb leads. It is the only true *right-sided limb lead*.

Now is as good a time as any to memorize these six leads and their angles.

Lead	Angle	
Inferior Leads		
Lead II	+60°	
Lead III	+120°	
Lead aVF	+90°	
Left Lateral Leads		
Lead I	+0°	
Lead aVL	–30°	
Right-sided Lead		
Lead aVR	–150°	

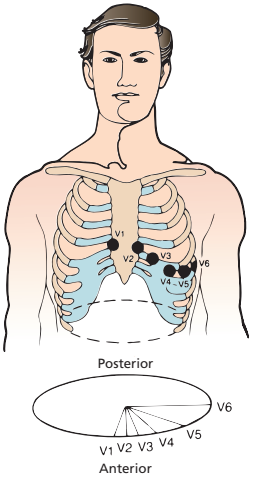
Of six limb leads, three are standard (I, II, and III), and three are augmented (aVR, aVL, and aVF). Each lead views the heart from its own particular angle of orientation.

The Six Precordial Leads

The six precordial leads, or chest leads, are even easier to understand. They are arranged across the chest in a *horizontal plane* as illustrated below. Whereas the leads of the frontal plane view electrical forces moving up and down and left and right, the precordial leads record forces moving anteriorly and posteriorly.

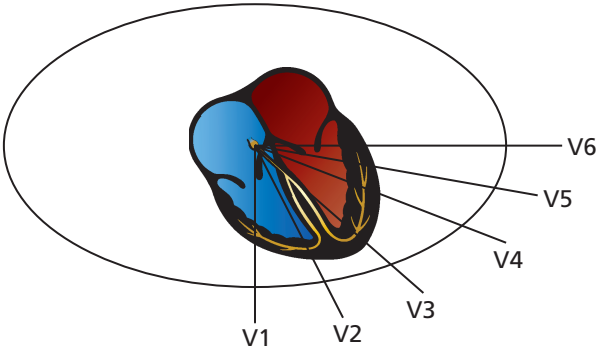
To create the six precordial leads, each chest electrode is made positive in turn, and the whole body is taken as the common ground. The six positive electrodes, creating the precordial leads V1 through V6, are positioned as follows:

- V1 is placed in the fourth intercostal space to the right of the sternum.
- V2 is placed in the fourth intercostal space to the left of the sternum.
- V3 is placed between V2 and V4.
- V4 is placed in the fifth intercostal space in the midclavicular line.
- V5 is placed between V4 and V6.
- V6 is placed in the fifth intercostal space in the midaxillary line.



The precordial leads define a horizontal or transverse plane and view electrical forces moving anteriorly and posteriorly.

Just like the limb leads, each precordial lead has its own particular line of sight and region of the heart that it views best.



Note that the right ventricle lies anteriorly and medially within the body cavity, and the left ventricle lies posteriorly and laterally. Lead V1 lies directly over the right ventricle, V2 and V3 over the interventricular septum, V4 over the apex of the left ventricle, and V5 and V6 over the lateral left ventricle.

Leads V2 through V4 are often referred to as the *anterior leads*, V5 and V6 join I and aVL as *left lateral leads*, and leads aVR and V1 are the *right ventricular leads*.

Leads	Group
V2, V3, V4	Anterior
I, aVL, V5, V6	Left lateral
II, III, aVF	Inferior
aVR, V1	Right ventricular

What Happens If You Misplace the Electrodes?

Perhaps you can already guess the answer to this question. If you reverse the limb electrodes—the most common mistake is to reverse the electrodes on the right and left arms—the EKG machine has no way of knowing that you have messed up. It can't adjust and correct your error. Therefore, current moving left to right or right to left will have its electrical recording reversed 180°. In other words, a lead that would normally show a tall, positive wave will now show a deep negative one, and vice versa. Your interpretation of the EKG will be seriously flawed, and you may think that a perfectly healthy patient has a serious heart condition.

You must also be careful to place the precordial leads, V1 through V6, as carefully as possible on the chest wall. Even a slight misplacement can lead to a mistaken interpretation, indicating, for example, that a previous heart attack has taken place where none has occurred. Placing these precordial leads on the chest can be particularly challenging in an obese patient or in someone with large, pendulous breasts. You may also be tempted from time to time to move some of the chest wall electrodes just a smidgeon to avoid patches of thick hair on a hirsute patient. And it is true that those sticky EKG electrode pads we typically use today do not adhere well when there is a bunch of hair between the pads and the chest wall. Do not take the easy way out and give in to temptation. Shave off the little spot of skin that you need. The hair will grow back quickly.

As many as 4% of all EKGs are run with incorrect lead placement by well-intentioned health care providers who are simply not being careful or who are understandably in a hurry (the emergency department and cardiac intensive care unit can be very stimulating environments!).

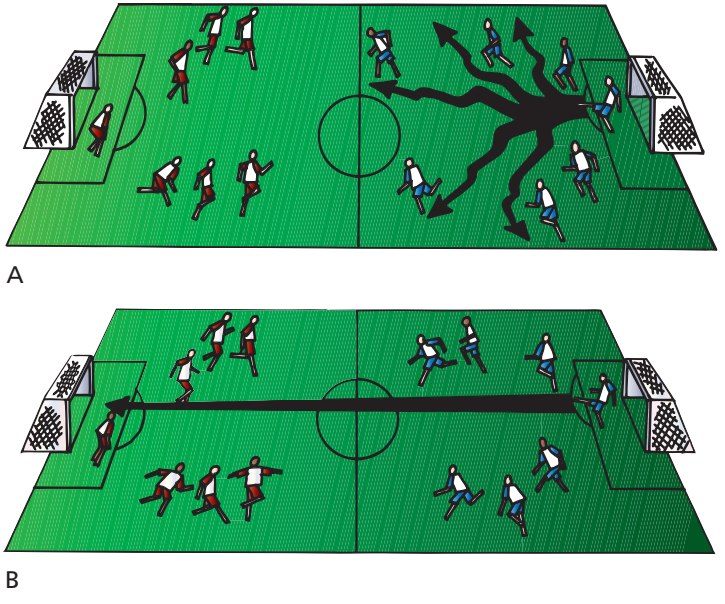
Does My Patient Have To Be Lying Down?

Yes, and as flat as possible. Some patients won't be able to lie perfectly flat—perhaps they have congestive heart failure and lying down makes them short of breath (orthopnea), or perhaps they have cervical arthritis and lying flat causes neck pain. Well, we are not tyrants—give your patient a pillow or elevate the head of the bed or examining table. But don't raise them up more than is necessary. Why does it matter? Changes in body posture affect the position of the heart within the body, and this in turn affects the voltage measurements on the EKG. Among the subtle changes that can occur are variations in the ST segments and the appearance of new Q waves, both of which, as we will discuss in Chapter 6, can be critical in diagnosing a myocardial infarction.

A Word About Vectors

It is important to recognize that each EKG electrode records only the *average* current flow at any given moment. Thus, although tiny swirls of current may simultaneously be going off in every direction, each lead records only the instantaneous average of these forces. In this way, out of chaos, some very simple patterns emerge.

This concept is really quite simple; an analogy may be helpful. During the course of a soccer match, a goalie may kick the ball many different times to various members of his team (or, if our hypothetical goalie is not very good, the other team). Some balls will go left, others right, and still others straight down the field. However, by the end of the game, the *average direction* of all of the goalie's kicks and tosses is likely to be straight ahead, toward the opposing net. This average direction can be represented by a single arrow, or *vector*.



(*A*) The directions of each of the goalie's kicks during the course of the game. (*B*) A single vector represents the average direction and distance of all of these kicks.

This vector is precisely what our EKG electrodes record when measuring the electrical flow within the heart. The vector's angle of orientation represents the average *direction* of current flow, and its length represents the voltage (*amplitude*) attained.

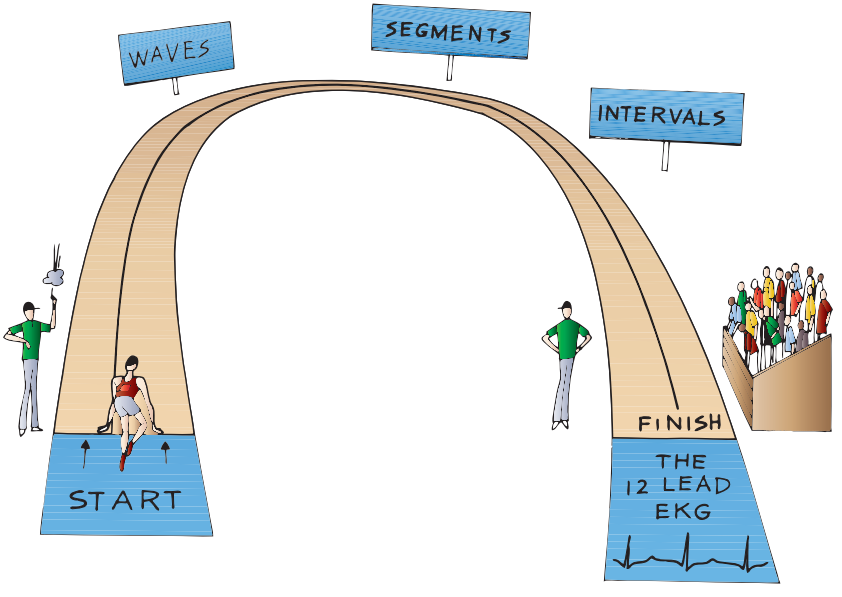
At any given moment, the electrical forces moving within the heart can be represented by a single vector. Furthermore, over any particular period of time during the cardiac cycle (*e.g.*, atrial depolarization), these individual vectors can be summed into a kind of *vector of vectors,* which describes the average direction and magnitude of current flow during that time period (*e.g.*, during atrial depolarization, corresponding, let us say, to all the goalie's kicks over the first half of the game). Thus, a particular wave (in this case, the wave of atrial depolarization) can be described by a single vector of given direction and magnitude. You will see how this works and how it simplifies our understanding of the 12-lead EKG in the following section.

The Normal 12-Lead EKG

You now know the three things necessary to derive the normal 12-lead EKG:

1. The normal pathway of cardiac electrical activation and the names of the segments, waves, and intervals that are generated
2. The orientation of all 12 leads, 6 in the frontal plane and 6 in the horizontal plane
3. The simple concept that each lead records the average current flow at any given moment

All we need to do now is to take what you already know and figure out what each wave looks like in each of the 12 leads.



The P Wave

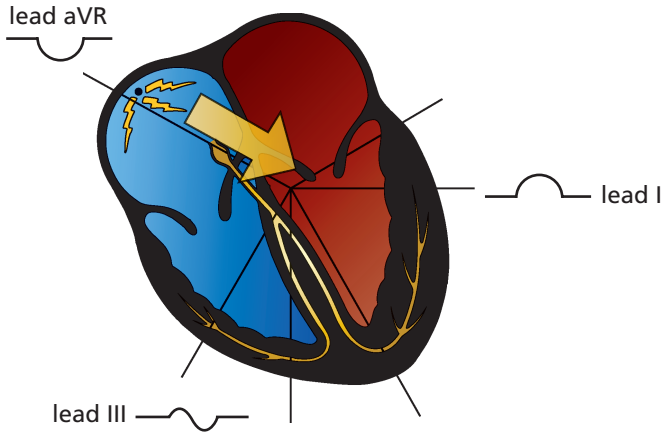
Atrial depolarization begins at the sinus node, high up in the right atrium. The right atrium depolarizes first, and then the left atrium.

The vector of current flow for atrial depolarization, therefore, points from right to left and slightly inferiorly (*large arrow*).

Any lead that views the wave of atrial depolarization as moving toward it will record a positive deflection on the EKG paper. The left lateral and inferior leads clearly fit this description. In the *frontal plane,* these leads include the left lateral leads I and aVL and the inferior leads II and aVF.

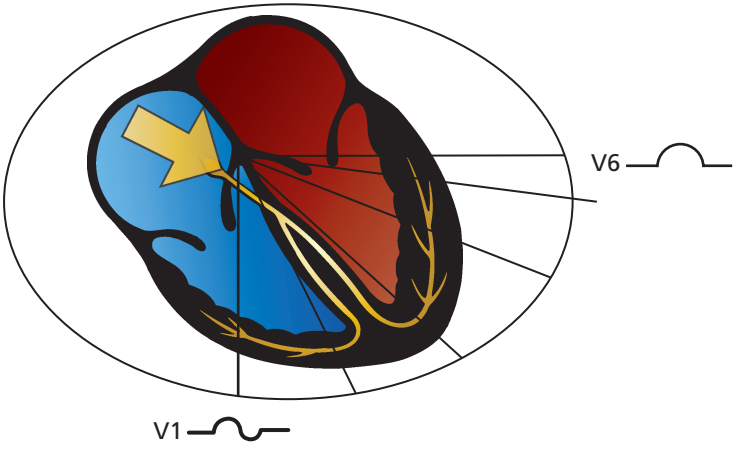
Lead III, which is also one of the inferior leads, is positioned a bit differently. It is the most rightward (orientation +120°) of the inferior leads and actually lies nearly perpendicular to the atrial current. Predictably, lead III frequently records a biphasic P wave.

Lead aVR, the most rightward of all the leads of the frontal plane (orientation –150°), sees the electrical current as moving away; hence, it records a purely negative deflection.



The vector of atrial depolarization points leftward and inferiorly. Therefore, lead I records a positive wave, aVR records a negative wave, and lead III records a biphasic wave.

In the *horizontal plane*, the left lateral leads V5 and V6 record a positive deflection, just as leads I and aVL did in the frontal plane. Lead V1, lying over the right heart, is oriented perpendicularly to the direction of current flow and records a biphasic wave, just like lead III. Leads V2 through V4 are variable.

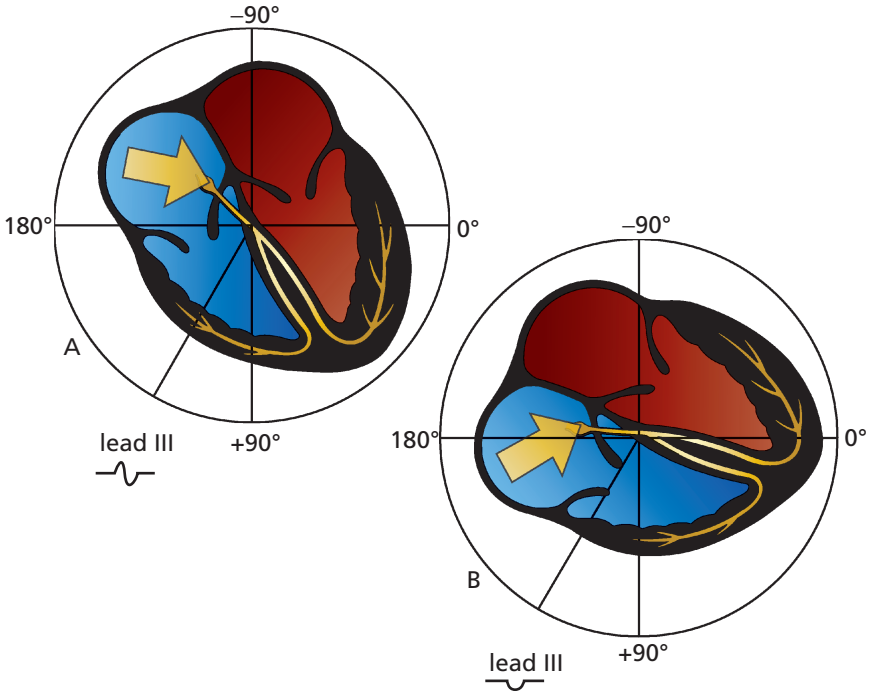


Atrial depolarization in the horizontal plane. V1 records a biphasic wave, and V6 records a positive wave.

Because the atria are small, the voltage they can generate is also small. The amplitude of the P wave does not normally exceed 0.25 mV (2.5 mm, or two and one-half small squares) in any lead. The P wave amplitude is usually most positive in lead II and most negative in lead aVR.

But People Are Individuals

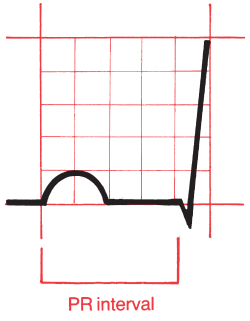
A word of caution is needed. Variations in anatomy and orientation of the heart from person to person make absolute rules impossible. For example, although the P wave in lead III is usually biphasic, it is not uncommon for it to be negative in perfectly normal hearts. All it takes is a change of a few degrees in the vector of current flow to turn a biphasic wave into a negative one. This can happen, for instance, if the patient's heart is angled slightly differently in the chest cavity. For this reason, the normal angle of orientation of current vectors is given in ranges, not precise numbers. For example, the normal range of the P wave vector is 0° to 70°.



Rotation of the heart within the chest cavity redirects the perceived direction of current flow. Lead III is normally oriented perpendicularly to atrial depolarization. With the apex of the heart turned leftward, lead III will view atrial depolarization as receding and will record a wave that is largely negative.

The PR Interval

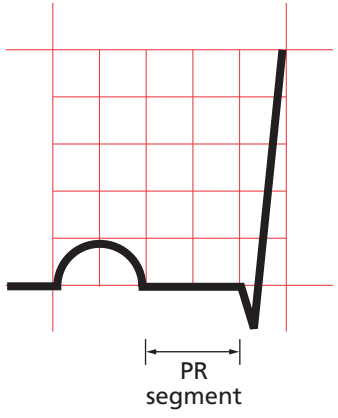
The PR interval represents the time from the start of atrial depolarization to the start of ventricular depolarization. It includes the delay in conduction that occurs at the AV node. The PR interval normally lasts from 0.12 to 0.2 seconds (3 to 5 mm on the EKG paper).



The normal PR interval lasts 0.12 to 0.2 seconds.

The PR Segment

The PR segment represents the time from the end of atrial depolarization to the beginning of ventricular depolarization. The PR segment is usually horizontal and runs along the same baseline as the start of the P wave.



The PR segment is horizontal.

The QRS Complex Is Complex, But Not Complicated

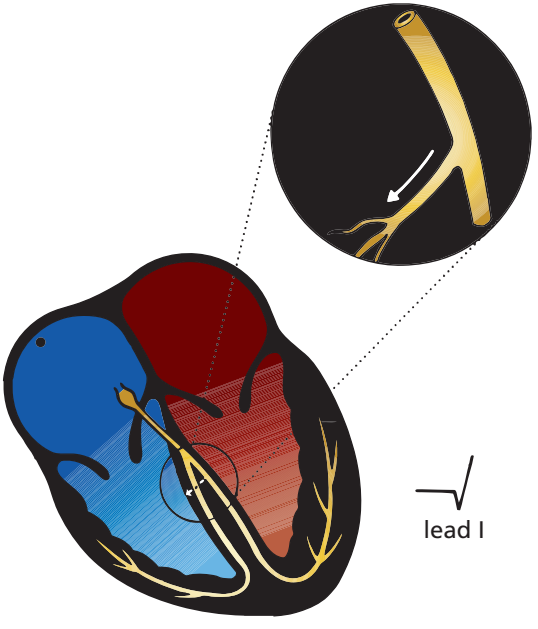
Our wave of electrical depolarization, emerging from the AV node, is now ready to enter the ventricles.

Septal Q Waves

The interventricular septum, the wall of muscle separating the right and left ventricles, is the first to depolarize, and it does so in a left-to-right direction. The tiny septal fascicle of the left bundle branch is responsible for rapidly delivering the wave of depolarization to this region of the heart.

Septal depolarization is not always visible on the EKG, but when it is, this small left-to-right depolarization inscribes a tiny negative deflection in one or several of the left lateral leads. This initial negative deflection, or Q wave, may therefore be seen in leads I, aVL, V5, and V6. Sometimes, small Q waves may also be seen in the inferior leads and in V3 and V4.

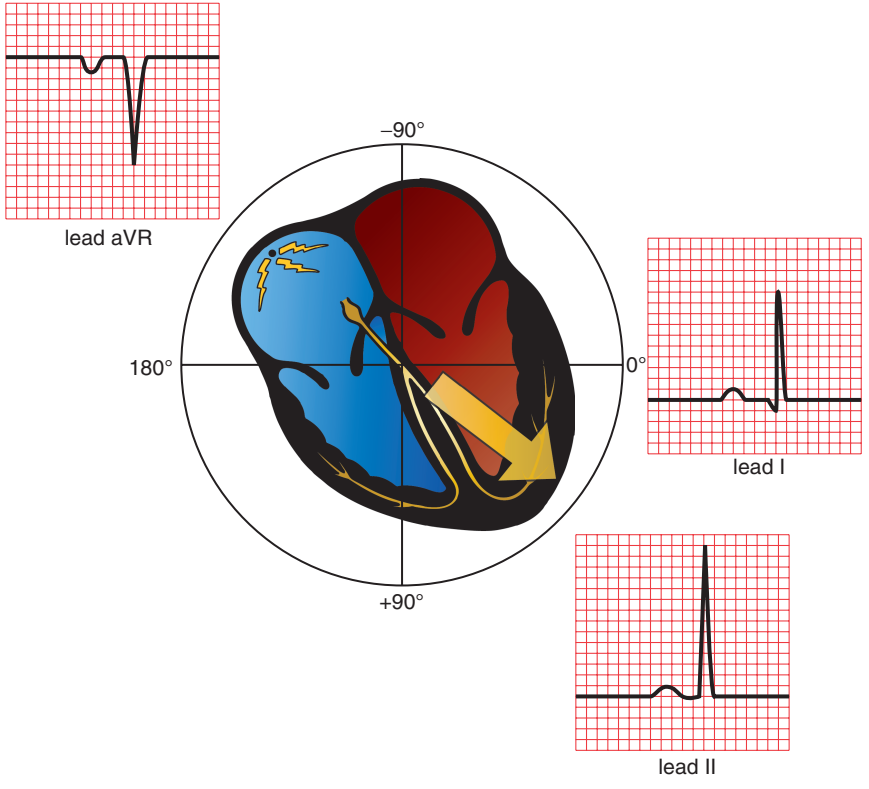
Normal septal Q waves have an amplitude of not greater than 0.1 mV.



The left lateral leads view left-to-right septal depolarization as moving away; therefore, they record a small initial negative deflection, or Q wave. Small Q waves are also sometimes seen in the inferior leads; these are normal.

The Rest of the Ventricular Myocardium Depolarizes

The remainder of the ventricles, the vast bulk of the myocardium, depolarizes next. Because the left ventricle is so much more massive than the right ventricle, it dominates the remainder of the QRS complex, and the average vector of current flow swings leftward. Normally, this vector points anywhere from 0° to +90°. In the frontal plane, therefore, large positive deflections (R waves) may be seen in many of the left lateral and inferior leads. Lead aVR, lying rightward, records a deep negative deflection (S wave).

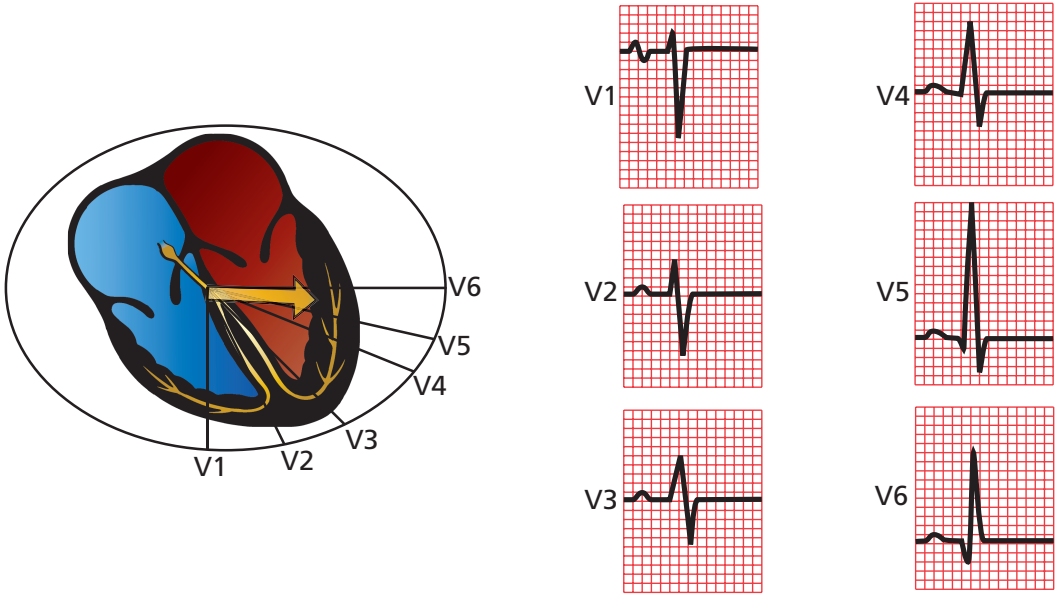


Ventricular depolarization as seen in leads I, II, and aVR. Lead I records a small Q wave due to septal depolarization and a tall R wave. Lead II also records a tall R wave and, less often, a small Q wave. The QRS complex in lead aVR is also deeply negative.

In the horizontal plane, lead V1, which overlies the right ventricle, usually records a deep S wave because the current is moving leftward, away from it. Conversely, leads V5 and V6, lying over the left ventricle, record tall positive R waves. Leads V3 and V4 represent a *transition zone*, and usually one of these leads records a biphasic wave, that is, an R wave and an S wave of nearly equal amplitude.

This pattern of progressively increasing R wave amplitude moving right to left in the precordial leads is called *R wave progression*. Lead V1 has the smallest R wave and lead V5 the largest (the R wave in lead V6 is normally a little smaller than that in lead V5). We also speak of a *transition zone*, the precordial lead or leads in which the QRS complex goes from being predominantly negative to predominantly positive. The normal transition zone occurs at leads V3 and V4.

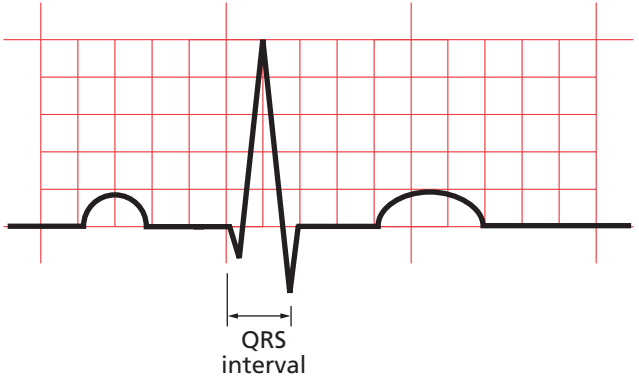
The amplitude of the QRS complex is much greater than that of the P wave because the ventricles, having so much more muscle mass than the atria, can generate a much greater electrical potential.



Ventricular depolarization in the precordial leads. Note the normal pattern of R-wave progression. The wave in lead V3 is biphasic.

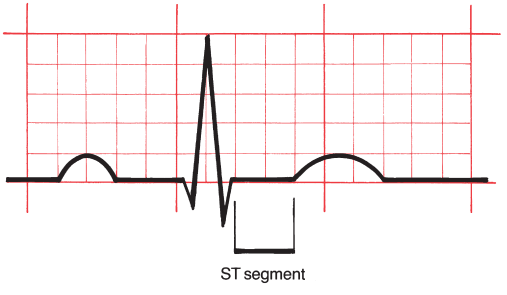
The QRS Interval

A normal QRS interval, representing the duration of the QRS complex, is 0.06 to 0.1 seconds in duration.



The ST Segment

The ST segment is usually horizontal or gently upsloping in all leads. It represents the time from the end of ventricular depolarization to the start of ventricular repolarization.



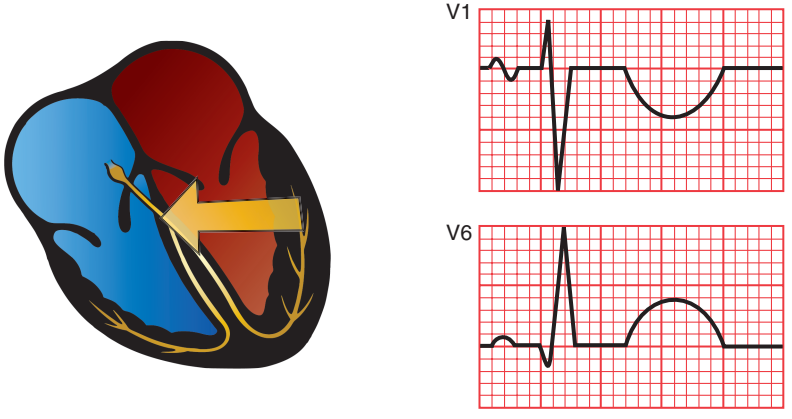
The T Wave

The T wave represents ventricular *repolarization*.

Unlike depolarization, which is largely passive, repolarization requires the expenditure of a great deal of cellular energy (remember the membrane pump). The T wave is therefore highly susceptible to all kinds of influences, both cardiac and noncardiac (*e.g.*, hormonal, neurologic), and is therefore variable in its appearance.

Nevertheless, certain general statements can be made. In the normal heart, repolarization usually begins in the last area of the heart to have been depolarized and then travels backward, in a direction opposite that of the wave of depolarization (*large arrow*). Because both an approaching wave of depolarization and a receding wave of repolarization generate a positive deflection on the EKG, the same electrodes that recorded a *positive* deflection during *depolarization* (appearing as a tall R wave) will also generally record a *positive* deflection during *repolarization* (appearing as a positive T wave). **It is therefore typical and normal to find positive T waves in the same leads that have tall R waves**.

The amplitude, or height, of a normal T wave is one-third to two-thirds that of the corresponding R wave.

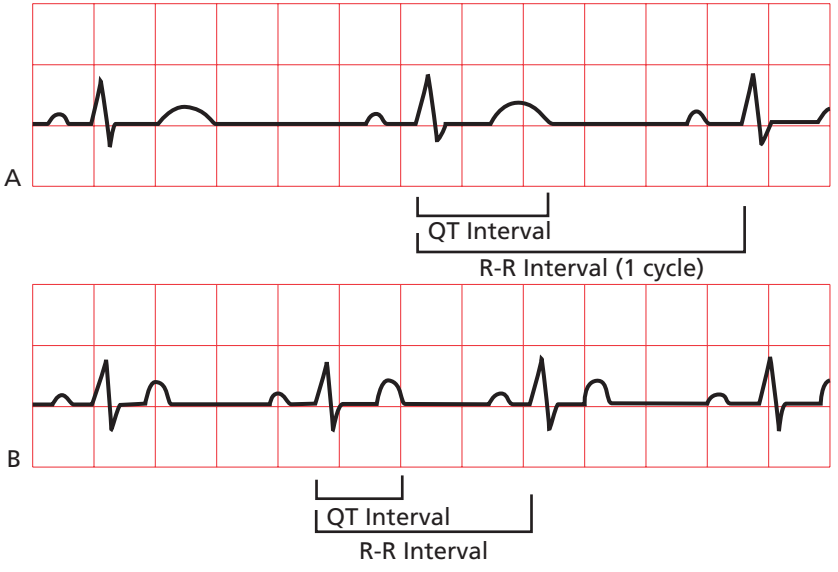


Ventricular repolarization generates a T wave on the EKG. The T wave is usually positive in leads with tall R waves.

The QT Interval

The QT interval encompasses the time from the beginning of ventricular depolarization to the end of ventricular repolarization. It therefore includes all of the electrical events that take place in the ventricles. From the standpoint of time, more of the QT interval is devoted to ventricular *repolarization* than depolarization (*i.e.*, the T wave is wider than the QRS complex).

The duration of the QT interval is proportionate to the heart rate. The faster the heart beats, the faster it must repolarize to prepare for the next contraction; thus, the shorter the QT interval. Conversely, when the heart is beating slowly, there is little urgency to repolarize, and the QT interval is long. In general, the QT interval constitutes about 40% of the normal cardiac cycle, as measured from one R wave to the next.



The QT interval constitutes about 40% of each cardiac cycle (R–R interval). The faster the heart beats, the shorter the QT interval. The heart rate in *B* is considerably faster than that in *A*, and the QT interval is correspondingly much shorter (less than one and one-half boxes vs. two full boxes).

SUMMARY

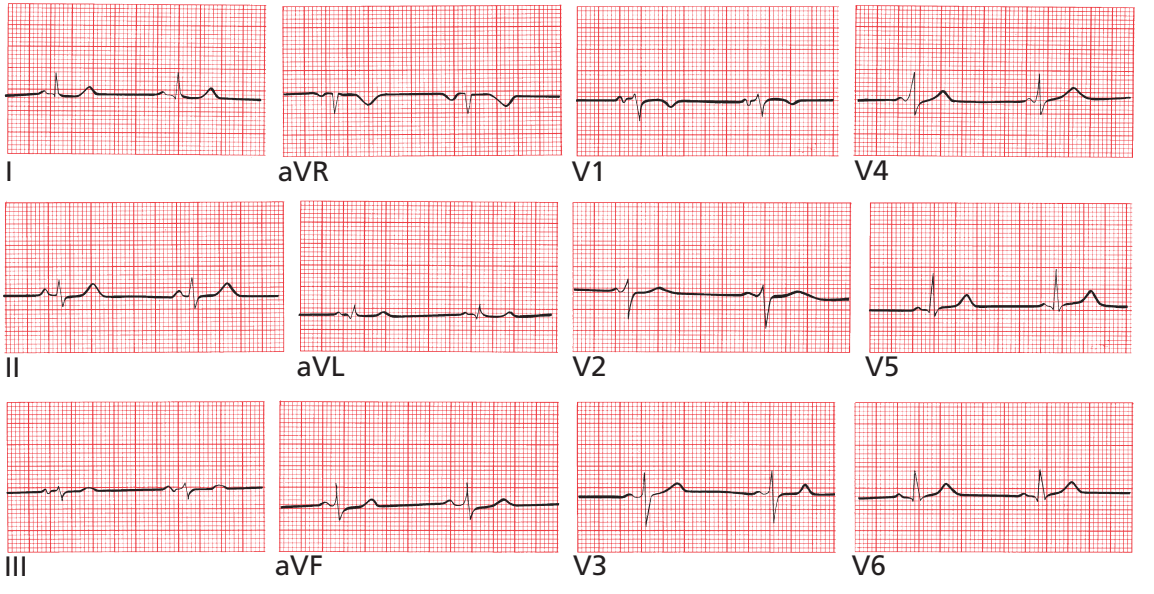
Orientation of the Waves of the Normal EKG

1. The P wave is small and usually positive in the left lateral and inferior leads. It is often biphasic in leads III and V1. It is usually most positive in lead II and most negative in lead aVR.

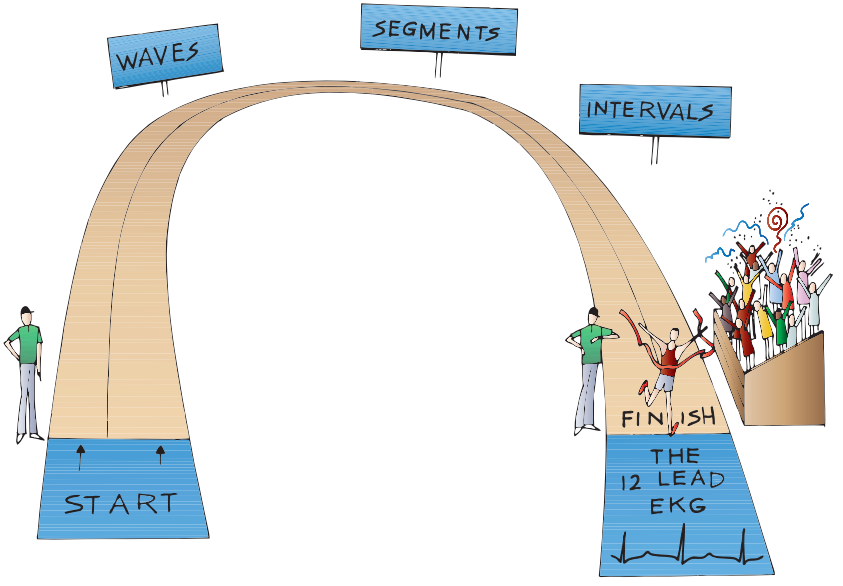
2. The QRS complex is large, and tall R waves (positive deflections) are usually seen in most left lateral and inferior leads. R-wave progression refers to the sequential enlargement of R waves as one proceeds across the precordial leads from V1 to V5. A small initial Q wave, representing septal depolarization, can often be seen in one or several of the left lateral leads, and sometimes in the inferior leads.

3. The T wave is variable, but it is usually positive in leads with tall R waves.

4. Now, take a good look at the following EKG. Does it seem familiar?



Of course it seems familiar. It's a normal 12-lead EKG, identical to the one that began the book.



Congratulations! You have successfully traversed the most difficult terrain in this book. Everything that follows builds logically from the few basic principles you have just mastered.

Some Really Important Stuff That You May Be Wondering: Part 1: Why Not Just Let the Computer Do It?

Before we leave the cozy confines of the normal EKG for the more harrowing realm of the abnormal, let's squarely face an important and frequently neglected issue. When you use a modern EKG machine, you get more than just a tracing. You get an interpretation straight from the computer's, er, mouth. Since we all know how smart computers are, why bother to learn to read EKG's at all?

There are two major reasons:

1. The computer often doesn't get it right. It does certain things very well, such as measuring intervals and identifying obvious deviations from the norm. But often it will overcall subtle but normal findings, sending you into a tizzy. Or, on the opposite side of the coin, it will miss slight abnormalities that may actually have huge consequences for your patient. And sometimes it will just waffle, calling something a *possible* abnormality, which doesn't help you at all.

2. You have one important thing going for you that the computer lacks—clinical context. You know your patient, how he or she looks and feels, the past medical history, the risk for cardiac disease, and so on. You also should know for certain whether the electrodes have been placed correctly. The best EKG interpretation will always be one that takes the actual patient into prime consideration. Borderline findings are common on EKGs, and only by considering the EKG as just one tool among many in your overall assessment are you likely to avoid unnecessary panic and start ordering needless additional tests or calling 911 to get your patient to the emergency department. On the other hand, a finding on the EKG that the machine just yawns at and dismisses as "nonspecific" may strike you quite differently if your patient is right in front of you clutching his or her chest in pain, complaining of shortness of breath and too light-headed to stand up.

To quote (in English translation) the ancient Greek playwright, Euripides: "*Much effort, much prosperity.*" In the chapters that follow, you will learn how to outsmart even the best EKG machine.

Some Really Important Stuff That You May Be Wondering: Part 2: How Do I Get the Goop Off My Patient's Chest Once the EKG Is Finished?

Most EKG electrodes are attached to the body with special pads impregnated with adhesive. Pulling them off can be painful, especially if there are hairs caught between the pads and the skin. Usually, a quick yank is your best bet to minimize your patient's discomfort. However, there may still be some adhesive residue left behind where the pads were placed. Happily, there are all sorts of ways to remove it—alcohol pads, petroleum jelly, baby oil, and mineral oil can all get the job done. Even after the goop has been removed, some patients may still be left with red marks where the electrode pads were placed. Assure your patient that these will resolve quickly, often within hours, sometimes days. If they find these spots intolerably itchy, a mild over-the-counter hydrocortisone cream will quickly set things right.

Coming Attractions

You are now ready to use the EKG to diagnose an extraordinary variety of cardiac and noncardiac disorders. We shall group these disorders into five categories.

Hypertrophy and Enlargement (Chapter 2). The EKG can reveal whether a particular atrial or ventricular chamber is enlarged or hypertrophied. Valvular diseases, sustained hypertension, and both inherited and acquired cardiac muscle disorders can affect the heart in this way, and the EKG can therefore help to recognize and evaluate these disorders.

Abnormalities of Rhythm (Chapter 3). The heart can beat too fast or too slow, fibrillate chaotically, or come to a sudden standstill. The EKG is still the best means to assess such rhythm disturbances, which, at their most severe, can lead to sudden death.

Abnormalities of Conduction (Chapters 4 and 5). If the normal pathways of cardiac electrical conduction become blocked, the heart rate can fall precipitously. The result can be syncope, a faint caused by a sudden decrease in cardiac output. Syncope is one of the leading causes of hospital admission. Conduction can also be accelerated along short circuits that bypass the normal delay in the AV node; we will look at these, too.

Myocardial Ischemia and Infarction (Chapter 6). The diagnosis of myocardial ischemia and infarction is one of the most important roles for the EKG. There are many reasons why a patient may have chest pain, and the EKG can help sort these out.

Electrolyte Disturbances, Drug Effects, and Miscellaneous Disorders (Chapter 7). Because all of the electrical events of the heart are dependent on electrolytes, it stands to reason that various electrolyte disorders can affect cardiac conduction and even lead to sudden death if untreated. Medications such as digitalis, antidepressants, antiarrhythmic agents, and even antibiotics can profoundly alter the EKG. A number of cardiac and noncardiac diseases can also cause dramatic shifts in the EKG. In each of these instances, a timely glance at an EKG may be diagnostic and sometimes lifesaving. We will also look at the controversial roles of the EKG in screening young athletes before sports participation and in evaluating a patient's risks before going for surgery.