Chapter 3

Electrocardiography

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In 1902, the Dutch physiologist Wilhelm Einthoven recorded the first ECG signals from humans. Since then, the number of recording leads has increased from 3 to 12, but the basic principles underlying electrocardiography are unchanged. Electrocardiography records from the body surface the voltage gradients created as myocardial cells sequentially depolarize and repolarize. It is the most commonly used technique to detect and diagnose cardiac disease and to monitor therapies that influence the electrical behavior of the heart. It is noninvasive, virtually risk free, and relatively inexpensive. Since its introduction, a large database has been assembled that correlates the ECG waveform recorded from the body surface to the clinical presentation of the patient, providing insight into the underlying electrical behavior of the heart and its modification by physiologic, pharmacologic, and pathologic events. This chapter discusses the relation of the ECG waveform to the underlying electrophysiologic properties of the heart and illustrates the changes in the ECG waveform induced by various events.

LEADS

Twelve leads are routinely used to record the body surface ECG: three bipolar limb leads: leads I, II, and III; three augmented limb leads: leads aVR, aVL, and aVF; and six unipolar chest leads: leads V1 through V6 (Fig. 3-1). In the bipolar limb leads, the negative pole for each of the leads is different, whereas in the unipolar chest leads, the negative pole is constant and created by the three limb leads. The positive chest lead is, in effect, an exploring lead that can be placed anywhere, provided the reader of the ECG knows its position. In children, for example, routine electrocardiography often includes placing leads on the right side of the chest wall in the positions referred to as V3R and V4R. Similar right-sided chest leads are often used in adults to diagnose right ventricular infarction, and one or more leads positioned on the back are sometimes used to diagnose posterior wall infarction.

The chest leads are much closer to the heart than are the limb leads and are influenced by the electrical activity directly under the recording lead. Changes in the relation of the individual chest lead to the heart may cause significant changes in the ECG waveform. For instance, if the lead is placed an interspace too high or too low, or if the patient is in a sitting rather than a supine position, the relation of the leads to the heart and the ECG waveform will change, potentially leading to misinterpretation unless the reader of the ECG is aware of the change from normal position.

ELECTROCARDIOGRAPHIC WAVEFORM

The ECG waveform consists of a P wave, a PR interval, a QRS complex, an ST segment, and T and U waves. Their relation to the underlying electrophysiologic events is shown in Figure 3-2. The P wave reflects depolarization of the atria, the QRS complex reflects depolarization of the ventricles, and the ST segment and T wave reflect repolarization of the ventricles. The cause of the U wave remains unclear. Sinus node depolarization occurs before the onset of the P wave, but the voltage gradients associated with sinus node depolarization are too small to be recorded on the body surface by the clinically used ECG machine. Therefore, this event is electrocardiographically silent. Similarly, the electrical activity of the atrioventricular (AV) junction, which occurs during the PR interval, is also electrocardiographically silent. Figure 3-3 is an example of a normal ECG.

P Wave

The P wave is caused by voltage gradients created by the sequential depolarization of atrial cells, indicated in Figure 3-2 by the upstroke of the atrial action potential. The sequence of atrial depolarization and time required to depolarize
Electrocardiographic Leads and Reference Lines

**Limb Leads**
- Lead I
- Lead II
- Lead III

**Augmented Limb Leads**
- Lead aVR
- Lead aVL
- Lead aVF

**Precordial Leads**
- V1
- V2
- V3
- V4
- V5
- V6

When current flows toward red arrowheads, upward deflection occurs in ECG.
When current flows away from red arrowheads, downward deflection occurs in ECG.
When current flows perpendicular to red arrows, no deflection or biphasic deflection occurs.
Figure 3-2  
Relation of Action Potential From the Various Cardiac Regions to the Body Surface ECG

Figure 3-3  
Normal ECG

Example of a normal ECG recorded from a 24-year-old woman. Note that the P wave is upright in leads I and II and inverted in aVR. The QRS complex gradually changes from negative to $V_1$ to positive $V_6$. Note that the polarity of the T wave is similar to that of the QRS complex.
all cells of the two atria are reflected in the shape and duration of the P wave. Impulses arising in the sinus node depolarize the right atrium before the left atrium. For this reason, the vectorial direction of atrial depolarization is from right to left, from superior to inferior, and from anterior to posterior. This results in a P wave that is characteristically upright or positive in leads I, II, V5, and V6 and inverted in aVR (Fig. 3-3). In V1, the P wave may be upright, biphasic, or inverted.

**QRS Complex**

The QRS complex reflects ventricular depolarization. Normally, depolarization of both ventricles occurs simultaneously, spreading from endocardium to epicardium and from apex to base. Because the left ventricle is three times the size of the right ventricle, its depolarization overshadows and largely obscures right ventricular (RV) depolarization. The spatial vector of the QRS complex reflects this left ventricular (LV) dominance and is directed to the left and posteriorly. The QRS complex is usually upright or positive in leads I, V5, and V6, the left-sided and more posterior leads, and negative or inverted in leads aVR and V1, the most right-sided and more anterior leads (Fig. 3-3). It is only in situations such as right bundle branch block and profound RV hypertrophy that the electrical activity associated with RV depolarization can be identified.

**ST Segment**

During the ST segment, all ventricular action potentials are at their plateau voltage of approximately 0 mV, and no voltage gradients are generated. Therefore, the ST segment is at the same level on the ECG as the PR and TP segments, during which time the ventricular action potentials are at their resting phase of approximately –85 mV.

**T Wave**

The T wave occurs as the result of sequential repolarization of the ventricular cells. If the repolarizing sequence were the same as the depolarizing sequence, the T wave would be opposite in direction to the QRS complex. However, the normal T wave is generally upright (positive) in leads with an upright or positive QRS complex (leads I, V5, and V6) and inverted (negative) in leads with an inverted QRS complex (aVR and V1) (Fig. 3-3). The QRS and T wave vectorial directions are similar because the sequence of repolarization is reversed, relative to the sequence of depolarization. This occurs because the duration of epicardial action potentials is shorter than that of the action potentials in the mid myocardium and subendocardium. Therefore, the cells on the epicardium are the first to repolarize, though they are the last to depolarize. The shorter duration of the epicardial action potential is attributed to two primary factors: The repolarizing ionic currents are slightly different in the epicardium, and cells of the specialized conducting systems have longer action potentials than the ventricular fibers and tend to prolong the action potentials of endocardial cells.

**FACTORS THAT ALTER COMPONENTS OF THE BODY SURFACE ELECTROCARDIOGRAM**

Factors that alter the sequence of depolarization and/or influence the upstroke of the action potential influence and alter the shape, duration, and vectorial direction of the P wave or the QRS complex, whereas factors that alter the sequence of repolarization and/or the phase of rapid repolarization influence the shape, duration, and vectorial direction of the T wave. The ST, TP, and PR segments are elevated or depressed by factors that introduce voltage gradients during these portions of the action potential. The interval from the onset of the QRS complex to the end of the T wave (the QT interval) is affected by factors that alter the time required for ventricular repolarization to occur, either by lengthening or shortening the plateau phase of the action potential, thereby influencing the duration of the ST segment, or by speeding or slowing the phase of rapid repolarization, thereby influencing the duration of the T wave. The route and the speed of conduction from the atria to the ventricles, which usually occurs via the AV node and specialized conducting system, influence the PR interval. Slowing of the impulse conduction anywhere in this pathway, but especially within the AV node, lengthens the PR interval. If bypass tracts that circumvent the AV nodal conduction pathway are present, conduction to the ventricles requires less time and the PR interval shortens.
**P Wave**

The duration of the P wave is lengthened by factors that prolong impulse propagation in the atria, such as fibrosis or hypertrophy. The shape of the P wave is modified by atrial hypertrophy, by the position of the heart within the chest, and by the site of origin of the impulses initiating atrial activation. For instance, in COPD, the diaphragm is depressed and the heart assumes a more vertical position. In this situation, the P wave will be altered. When the left atrium is hypertrophied, or when intra-atrial conduction is slowed, the terminal component of the P wave, which represents left atrial depolarization, will be affected and the P wave will change.

Impulses arising from an ectopic focus within the atria are associated with P waves in which the shape depends on the location of the focus and the sequence of atrial depolarization. If the ectopic focus is close to the sinus node, the P wave will resemble a normal sinus P wave. The further the ectopic focus is from the sinus node, the more abnormal will be the P-wave configuration. For instance, impulses arising in the inferior portion of the atria or in the AV node will depolarize the atria in a retrograde, superiorly oriented direction. The P wave will reflect this superior orientation and will be inverted in leads II, III, and aVF (Fig. 3-4).

**PR Interval**

The PR interval is prolonged by factors that slow AV nodal conduction, including an increase in vagal tone (because the AV node is richly supplied by vagal fibers) and by drugs that enhance vagal tone or diminish sympathetic tone, such as the digitalis glycosides and the β-adrenergic-blocking agents. Drugs that inhibit or block the calcium inward current, calcium channel blockers, also cause PR prolongation because calcium ions rather than sodium ions are responsible for the upstroke of the action potential in cells comprising the AV node upper portion. Diseases involving the AV node are another cause of PR prolongation. The PR interval is shortened when impulses reach the ventricles via a bypass tract to cause ventricular preexcitation.

**QRS Complex**

The QRS complex is altered both in shape and duration by abnormalities in the sequence of ventricular activation, such as right and left bundle branch block (Fig. 3-5A). Ventricular pre-excitation (as occurs in the Wolff-Parkinson-White Syndrome) also changes the sequence of ventricular activation and the shape and duration of the QRS complex, mimicking a bundle branch block (Fig. 3-5B). Loss of ventricular muscle also results in an abnormal QRS shape. ECG changes accompanying myocardial infarction are examples of this phenomenon (Fig. 3-6). Infarction results in abnormalities in the early portion of the QRS complex with creation of an abnormal Q wave in leads overlying the infarcted region. In this way, the ECG abnormality localizes the infarction and suggests the vessel responsible for the infarct.

Drugs that block the sodium inward current, such as the type I antiarrhythmic drugs, slow the rate at which individual cells depolarize. This slows impulse propagation throughout the ventricle and causes diffuse lengthening of the QRS complex. However, the sequence of activation is not altered, so the QRS complex maintains its normal waveform. An increase in extracellular potassium, which makes the resting membrane potential of the individual action potential less negative, also slows interventricular conduction and the rate of cellular depolarization, causing uniform lengthening of the QRS complex and also characteristic peaking of T waves (Fig. 3-7). The QRS complex is also changed by ectopic beats and rhythms originating from an ectopic focus in the ventricle. The shape and duration of these ectopic beats reflect the site of origin.

The amplitude of the QRS complex is subject to a variety of factors: thickness of the LV and RV walls, presence of pericardial or pleural fluid, and amount of tissue between the heart and the chest wall. Age, sex, and race may also affect QRS amplitude. For instance, young adults have greater QRS voltages than older individuals, men have a greater QRS voltage than women, and black individuals tend to have greater QRS voltages than white individuals. In LV hypertrophy, the magnitude of left and posterior forces associated with LV depolarization increases, causing an increase in the positive QRS voltage, that is, the R wave, in the left-sided leads, V₅ and V₆, and an increase in the negative QRS voltage, that is, the S wave, in the right-sided chest leads.
QRS duration may increase, reflecting the increased thickness of the left ventricle and there may be repolarization changes (Fig. 3-8). Pericardial and pleural effusion decreases QRS voltage in all leads. Infiltrative diseases, such as amyloidosis, may also decrease QRS voltage.

**ST Segment and T Wave**

The ST segment and T wave reflect the action potential plateau (ST segment), and the phase of rapid repolarization (T wave). These two components are often affected simultaneously by factors such as LV hypertrophy; cardioactive drugs, such as digitalis and the type I and type III antiarrhythmic agents; and a decreased concentration of serum potassium. In these situations, ST-segment and T-wave changes both occur. However, the ST segment and T wave may also be affected separately, resulting in ST-segment changes without T-wave changes or T-wave changes without ST-segment changes. The ST segment is altered by factors that induce voltage gradients during the plateau phase of the action potential. Acute myocardial ischemia causes the plateau voltage to become more negative in cells located within the ischemic zone, creating voltage gradients during the plateau phase between the ischemic and nonischemic regions. This phenomenon leads to generation of injury currents across the ischemic margin, which can cause either ST-segment elevation or depression, depending on whether acute ischemia is transmural or nontransmural (see Figs. 3-6 and 3-9). Acute pericarditis usually involves the entire precordial surface but does not affect deeper layers. Thus, the injury current generated is between the epicardium and deeper layers and, generally, leads to diffuse ST-segment elevation. There are also normally occurring differences in the early portion of the plateau of the action potential in cells from the epicardial and deeper layers. These differences may cause voltage gradients and result in ST-segment elevation. This form of ST-segment deviation, which occurs most frequently in younger males, is a normal variant and referred to as “early repolarization.”

The duration of the ST segment and, thereby, the duration of the QT interval may be altered by changes in heart rate and by changes in extracellular calcium. Hypocalcemia and bradycardia lengthen the plateau of the action potential and cause lengthening of the ST segment and of the QT interval (Fig. 3-10). Hypercalcemia and tachycardia have the opposite effect. They shorten the plateau duration, the ST segment, and the QT interval.

The T wave can be influenced independent of the ST segment by factors that alter the sequence of repolarization. For example, sudden changes in heart rate may cause some
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(A) Electrocardiogram showing left bundle branch block. It was recorded from a 73-year-old man. Note that the QRS complex is diffusely widened and is notched in leads V3, V4, V5, and V6. Note also that the T wave is directed opposite to the QRS complex. This is an example of a secondary T-wave change.

(B) ECG showing ventricular preexcitation. It is recorded from a 28-year-old woman. Note the short PR interval (0.9 seconds) and the widened QRS complex (0.134 seconds). The initial portion of the QRS complex appears slurred. This is referred to as a delta wave. This combination of short PR interval and widened QRS complex with a delta wave is characteristic of ventricular pre-excitation. Note also that the T wave is abnormal, another example of a secondary T-wave change.

Figure 3-5

Bundle Branch Block

Ventricular Preexcitation

action potentials to shorten or lengthen more rapidly and to a greater extent than other action potentials. This is an example of a functional rather than a pathologic T-wave change. Pathologic T-wave changes are those that occur with disease entities such as myocarditis and some cardiomyopathies. Inverted T waves may also persist after an ischemic event or myocardial infarction (Fig. 3-11). Changes in the sequence of repolarization also result from changes in the sequence of depolarization. These obligatory changes in repolarization result in “secondary” T-wave changes and are responsible for the ST-segment and T-wave changes accompanying bundle branch blocks and ventricular preexcitation (Figs. 3-5A and 3-5B).
Myocardial Ischemia, Injury, and Infarction

Ischemia causes inversion of T wave due to altered repolarization.

Muscle injury causes elevation of S–T segment.

Death (infarction) of muscle causes Q or QS waves due to absence of depolarization current from dead tissue and opposing currents from other parts of the heart.

During recovery (subacute and chronic stages) S–T segment often is first to return to normal, then T wave, due to disappearance of zones of injury and ischemia.

Reciprocal effects on opposite side of infarct.

Figure 3-6

Changes Associated With Hyperpotassemia

Example of the ECG changes associated with hyperpotassemia. It is recorded from a 29-year-old woman with chronic renal disease. The P wave is broad and difficult to identify in some leads. The QRS is diffusely widened (0.188 seconds) and the T wave is peaked and symmetrical. These changes are characteristic of severe hyperpotassemia and, in this patient, the serum potassium concentration was 8.2 mM.
**Figure 3-8**

**ECG Changes of LV Hypertrophy**

Example of the ECG changes of LV hypertrophy. It is recorded from an 83-year-old woman with aortic stenosis and insufficiency. Note the increase in QRS amplitude, the slight increase in QRS duration to 100 ms, and the ST-segment and T-wave changes.

**Figure 3-9**

**ST-Segment Changes Associated With an Acute Ischemic Event**

Example of ST-segment changes associated with an acute ischemic event. It is recorded from a 43-year-old man with chest pain. Note the ST-segment elevation in leads V1, aVL, and V2 through V6, and the ST-segment depression in leads III and aVF.

**U Wave**

The U wave follows the T wave. It may also arise within the terminal portion of the T wave and be difficult to distinguish from a notched T wave. Although the precise etiology of the U wave is not clear, an increase in its magnitude or a change in its polarity occurs with several clinical entities. An increase in U-wave amplitude is frequently associated with hypokalemia and with some direct-acting cardiac drugs (Fig. 3-12A). Notching of the T wave, resembling an increase in U-wave amplitude and lengthening of the QT-U interval, also occurs in patients with congenital long QT syndrome (Fig. 3-12B), reflecting a genetic abnormality of one or more ionic channels responsible for repolarization.
ST-segment and QT-interval changes associated with hypocalcemia. It is recorded from a 53-year-old man with chronic renal disease. The ST segment is prolonged, but the T wave is normal. The QT interval reflects ST-segment lengthening and is prolonged.

T-wave changes induced by a recent ischemic event, recorded from a 70-year-old man. The QT interval is prolonged and the T waves are markedly inverted in the precordial leads (V₁ through V₆). These changes gradually evolved over several days, and coronary angiography recorded the day this tracing was taken revealed a subtotal occlusion of the left anterior descending coronary artery.

**Arrhythmias**

Electrocardiography is indispensable in the diagnosis of brady- and tachyarrhythmias. For instance, a heart rate greater than 100 beats/min may have multiple causes, including sinus tachycardia, atrial and AV junctional tachycardia (Fig. 3-13A), atrial flutter, atrial fibrillation (Fig. 3-13B), and ventricular tachycardia (Fig. 3-13C). The rate and configuration of the P wave, its relation to the QRS complexes, and the shape and duration of the QRS complex establish the correct diagnosis. Abnormally slow heart rates may also be caused by several entities, including sinus bradycardia or sinoatrial or AV block (Fig. 3-13D). Again, the diagnosis can be established by noting the rate, regularity, and configuration of the P wave and
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Changes Associated With Hypopotassemia

(A) Example of the changes associated with hypopotassemia. It is recorded from a 44-year-old man who was receiving long-term thiazide therapy. The QT interval is prolonged due to the presence of a U wave, which interrupts the descending limb of the T wave and is of equal amplitude to the T wave. In this patient, the serum potassium concentration was 2.7 mM.

(B) Recorded from a 16-year-old girl with syncopal episodes that were documented to be due to rapid ventricular tachycardia. It is an example of long QT syndrome. The T wave is notched and prolonged in much the same way as was shown in the patient with hypopotassemia. However, in this patient, the serum potassium concentration was normal.

QRS complexes, the relation of the P wave to the QRS complexes, and the PR interval.

Irregular rhythms may be due to atrial and ventricular premature beats (Figs. 3-14A and 3-14B), atrial fibrillation (Fig. 3-13B), and incomplete (second degree) sinoatrial or AV block (Fig. 3-14C).

FUTURE DIRECTIONS

The ECG provides a window into the basic electrophysiologic properties of the heart and their modification by physiologic, pharmacologic, and pathologic causes. The ECG is relatively simple to obtain, reasonably inexpensive, and,
when correctly interpreted, of inestimable help in the diagnosis and treatment of a wide variety of cardiac diseases. Many proposed approaches have the goal of obtaining more precise, predictive information from the baseline ECG. Signal-averaged ECGs (SAECG) were developed as an attempt to more accurately predict the propensity of development of ventricular arrhythmias in an individual and to gauge the effectiveness of pharmacologic therapy. It has become evident that the SAECG offers only a limited amount of incremental information. There is much interest in computerized analysis of T-wave features as markers for the same events. It is likely that more powerful computerized analysis of ECG morphology will increase the usefulness of this test and its prognostic value, and that detailed analysis of the ECG will become increasingly important.
**Irregular Cardiac Rhythms**

**Atrial Premature Beats**

(A) Atrial premature beats (shown with an arrow) recorded from a 77-year-old man. In this example, there is an atrial premature beat after every two sinus beats. This is referred to as atrial trigeminy. Note that the shape of the premature P wave is different than that of the sinus P waves, reflecting its ectopic location.

**Ventricular Premature Beats**

(B) Ventricular premature beats recorded from a 30-year-old man with no known heart disease.

**Type I Second-Degree AV Block**

(C) Type I second-degree AV block with Wenckebach periodicity recorded from a 74-year-old man. There is progressive prolongation of the PR interval, followed by a blocked or nonconducted P wave. This leads to irregular groups of QRS complexes. In this example, there is 5:4 and 4:3 AV block. The atrial rate is 110 beats/min, and the ventricular rate is 90 beats/min.

**REFERENCES**

