Electrocardiography for the Family Physician

Electrocardiography for the Family Physician: The Essentials

Second Edition

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The Essentials, Second Edition

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PREFACE

The electrocardiogram can serve as an independent identifier of myocardial disease or reflect anatomic, metabolic, hemodynamic, or electrophysiological alterations in the heart. It can provide information that is often essential for the proper diagnosis and treatment of a variety of disorders and is without equal as a method for diagnosing cardiac arrhythmias. It is the procedure of choice for patients who present with chest pain, dizziness, syncope, or symptoms that may indicate risk of myocardial infarction or sudden death.

Family physicians are often the first, and sometimes the only, point of contact for many patients within the health care system. The standard 12-lead electrocardiogram is one of the most common tests obtained and interpreted by the family physician, with most physicians reading their own recordings and basing clinical decisions on their findings. It has been shown that family physicians can achieve proficiency in the interpretation of over 95 percent of all electrocardiogram findings seen in the primary care setting.

Although computerized interpretation is widely available, it is considered unreliable in up to 20 percent of the cases, making interpretation by family physicians an essential skill. This book provides the necessary skills for family physicians to use in interpreting electrocardiograms, both in their offices and in the emergency rooms of their hospitals. It also should prove of value to other primary care physicians, as well as medical students and residents of nearly all medical specialties.

As the subtitle states, this book is about the essential elements involved in electrocardiographic interpretation. It is not all inclusive; however, it does cover the abnormalities most likely to be seen by family physicians in their everyday practice of medicine.

This book is an outgrowth of a course I taught in the De-

partment of Family Medicine at the University of Mississippi School of Medicine and five articles titled *Electrocardiography for the Family Physician* I subsequently published in *Family Practice Recertification.*

In short, this book is the one I wish I had access to during the many years I actively practiced family medicine and when I was a resident in family medicine.

I have made several changes in the second edition. These include adding sections on hypertrophic cardiomyopathy, the Sgarbossa criteria for diagnosing myocardial infarction in the face of left bundle branch block, left ventricular aneurysm, myocarditis, bigeminy, electrical alternans, Takotsubo cardiomyopathy, Brugada syndrome, and upgrading the terminology for acute coronary syndrome.

I currently teach an ECG course to family medicine residents in the EC-Healthnet Family Medicine Residency Program in Meridian, Mississippi.

H. Thomas Milhorn, M.D., Ph.D.

Contents

Chapter 1

The Electrocardiogram

Electrocardiography is a test that measures the electrical signals that control the rhythm of the heartbeat. The graph that shows the results is called an *electrocardiogram* (EKG, ECG).

An electrocardiogram may show:

- Abnormal conduction of cardiac impulses due to damage of the conducting system
- Abnormally slow, fast, or irregular heart rhythms
- Adverse effects on the heart from certain lung conditions, such as emphysema and pulmonary embolus
- Adverse effects on the heart from various cardiovascular or systemic diseases, such as high blood pressure and thyroid conditions
- Certain congenital heart abnormalities
- Changes in the electrical activity of the heart caused by medication (digoxin, type 1a antiarrhythmics such as quinidine)
- Evidence of abnormal blood electrolytes (potassium, calcium)
- Evidence of an acute impairment of blood flow to the heart (angina)
- Evidence of an acute, evolving, or prior myocardial infarction
- Evidence of atrial enlargement or ventricular hypertrophy
- Evidence of inflammation of the heart (myocarditis) or its lining (pericarditis).

ELECTROCARDIOGRAPH PAPER

The electrocardiogram is recorded on graph paper with divisions as indicated in Figure 1-1. Since the ECG paper speed is ordinarily 25 mm/second, a small square is 0.04 seconds wide. A small square is one millimeter (0.1 mV) high. A large square is 0.2 seconds wide and five millimeters (0.5 mV) high.

A square-wave *calibration signal* is placed on every electrocardiogram. When recorded with a normal calibration the signal is 10 mm high and represents 1.0 mV. When recorded at half standard because of large QRS voltages, the calibration standard is 5 millimeters, also representing 1.0 mV. The calibration standard should always be noted first when interpreting an electrocardiogram. The full-standard calibration is used throughout this book.

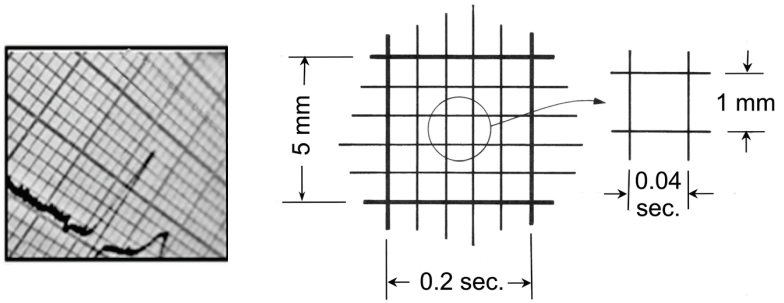

Figure 1-1. Electrocardiograph paper dimensions.

CONDUCTION SYSTEM OF THE HEART

Electrical activation of the atrial and ventricular muscle is termed *depolarization*. Initiation of depolarization normally occurs in the *sinoatrial node (SA node)*. The current then travels through the *internodal tracts* of the atria to the *atrioventricular node (AV node)*. From there the depolarization wave passes down the *bundle of His (atrioventriclular bundle)*, which divides into the *right* and *left bundle branches* (Figure 1-2).

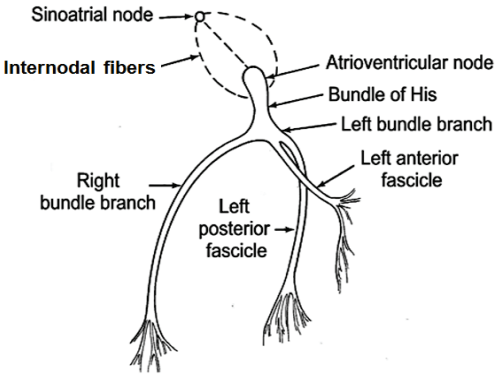

Figure 1-2. The conduction system of the heart. The bundle of His is also known as the atrioventricular bundle

The left bundle branch, in turn, divides into the *left anterior* and *left posterior fascicles*. The right bundle branch is not divided and supplies the right ventricle. The left bundle branch supplies the left ventricle. The AV node, bundle of His, and right and left bundle branches are known collectively as the *Purkinje* system. The depolarization wave rapidly spreads out from these pathways, causing contraction of the myocardial muscle. *Repolarization* of the electrical potential of the cardiac muscle cells follows.

PARTS OF THE ELECTROCARDIOGRAM

Because the body is a conductor of electrical current, the electrical activity of the heart can be monitored by the use of a galvanometer and electrodes placed on the surface of the skin. Depolarization and repolarization result in various deflections recorded on ECG paper. From this recording, various waves, intervals, and segments can be identified.

Deflections

The *P wave* reflects atrial depolarization, the *QRS complex* reflects ventricular depolarization, and the *T wave* reflects ventricular repolarization (Figure 1-3). Atrial repolarization occurs during ventricular depolarization and, therefore, is obscured by the QRS complex.

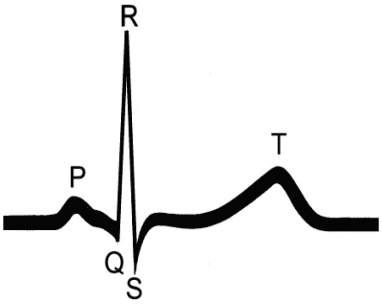

Figure 1-3. The deflections of the electrocardiogram generated by the heart during depolarization and repolarization.

P Wave

The *P wave* normally is largest in lead II and is upright in all leads except aV_R. If the P wave is not upright in lead II, you should suspect:

- Dextrocardia
- Ectopic atrial rhythm
- Reversed arm electrodes

The P wave normally lasts less than 0.11 seconds (just less than three small squares). An abnormally long P wave occurs whenever it takes extra time for the electrical wave to travel over the entire atrium, such as in atrial enlargement. The height of the P wave is normally less than 2.5 small squares (0.25 mV).

An abnormally tall P wave is seen when larger amounts of electricity are moving over the atrium than normally, such as also occurs in atrial enlargement. Abnormal P waves can be:

- **Widened.** Treatment with a Class Ia antiarrhythmic agent, such as quinidine
- **Inverted.** Direction opposite the predominant QRS deflection. Retrograde atrial depolarization; that is, depolarization originating in the atrioventricular junction and traveling backward up the atria
- **Notched.** Atrial enlargement
- **Small or Absent.** Hyperkalemia

QRS Complex

The *QRS complex* represents depolarization of the ventricles. By definition, the *Q wave* is the first downward stroke of the QRS complex and is usually followed by an *R wave*, which is the first upward deflection of the QRS complex. A QRS complex may not necessarily contain a Q wave, an R wave, or an S wave, and may contain more than one R wave (Figure 1-4).

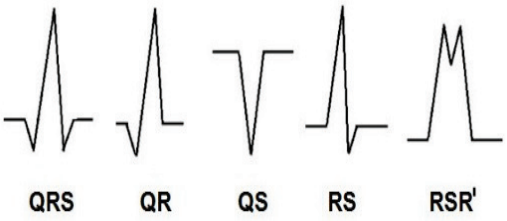

Figure 1-4. Examples of various QRS complex morphologies and their nomenclatures

If a second upward deflection is seen, it is called an R-prime (R′) wave. R-prime waves are never normal, but indicate a problem in the ventricular conduction system.

Common causes of QRS widening include:

- Drug effect (procainamide, tricyclic antidepressants, cocaine)
- Electrolyte effect (hyperkalemia, hypermagnesemia)
- Premature ventricular contractions
- Right and left bundle branch blocks
- Supraventricular beats with aberration
- Ventricular escape beats
- Ventricular pacemaker beats
- Wolff-Parkinson-White syndrome

T Wave

The *T wave* represents the wave of repolarization as the ventricle muscle prepares for firing again. It is normally upright in leads I, II, and V_3-V_6. It is normally inverted in lead aV_R. T waves are variable in the other leads. Height is normally less than 5 mm in the standard limb leads and less than 10 mm in the precordial

leads. The direction normally follows the direction of the main QRS deflection.

T wave abnormalities may be seen with or without ST segment abnormalities. T wave abnormalities include:

- **Tall T waves.** Hyperkalemia, very early myocardial infarction, and left ventricular hypertrophy
- **Flat or small T waves.** Ischemia, evolving myocardial infarction, myocarditis, pulmonary embolus, hypokalemia, thick chest wall, emphysema, pericardial effusion, cardiomyopathy, constrictive pericarditis, hypothyroidism, hypoadrenalism, hypocalcemia, and nonspecific causes
- **Inverted T waves.** Ischemia, infarction, late in pericarditis, left ventricular hypertrophy, bundle branch blocks, digoxin, athletic heart syndrome, and acute cerebral disease

In young children, T waves normally may be inverted in the right precordial leads (V_1 to V_3). Occasionally, these T wave inversions persist into young adulthood.

U Wave

When present, a second wave following the T wave is called a *U wave*. Its direction usually is the same as that of the T wave. Its amplitude is usually less than 1/3 of the T wave amplitude in the same lead (Figure 1-5).

Figure 1-5. The U wave.

U waves are most prominent in leads V_2 and V_3. The most common cause of prominent U waves is bradycardia, generally becoming visible when the heart rate falls below 65 bpm. Other causes of U waves are:

- CNS disease
- Drugs (amiodarone, disopyramide, digoxin, epinephrine, phenothiazines, procainamide, quinidine)
- Electrolyte imbalance (hypokalemia, hypomagnesemia, hypercalcemia)
- Hyperthyroidism
- Left ventricular hypertrophy
- Long QT syndrome
- Mitral valve prolapse

Inverted U waves may be seen with:

- During episode of acute myocardial ischemia
- During coronary artery spasm (Prinzmetal's angina)
- Some cases of left or right ventricular hypertrophy

The exact significance of U waves is unknown, but they may be due to repolarization of the papillary muscles or Purkinje fibers.

Intervals

The PR, QRS, and QT intervals fall within well-defined limits (Figure 1-6).

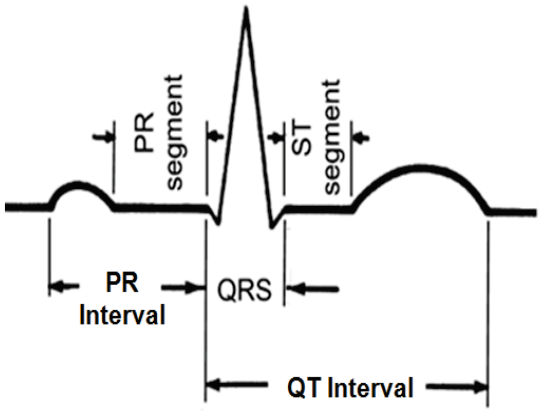

Figure 1-6. Intervals and segments in the electrocardiogram.

PR Interval

The *PR interval* is the time required for the depolarization wave to complete atrial depolarization; be conducted through the AV node, bundle of His and bundle branches; and arrive at the ventricular myocardial cells. It is the time from the beginning of the P wave to the beginning of the QRS complex. It is normally between 0.12 and 0.2 seconds (three to five small squares) in length.

The PR interval may be prolonged when conduction of the electrical wave through the AV node is slow. This may be seen with:

- Degenerative disease of the node (heart blocks)
- Digoxin
- Electrolyte abnormalities (hyperkalemia, hypercalcemia)
- Hypothermia
- Hyperthyroidism (occasionally)

The PR interval may be unusually short with:

- Electrolyte abnormalities (hypokalemia, hypocalcemia)
- Type II glycogen storage disease (Pompe's disease)
- Hypertension
- Hypertrophic cardiomyopathy
- Junctional rhythm
- Pacing
- Preexcitation syndromes (Wolff-Parkinson-White syndrome, Lown-Ganong-Levine syndrome)

QRS Interval

The *QRS interval* is the time required for the ventricular cells to depolarize. The normal duration is 0.06 to 0.10 seconds (1-1/2 to 2-1/2 small squares).

Lengthening of the QRS interval usually indicates some blockage of the electrical activity in the conducting system. Some causes of increased QRS interval include:

- Drug effect (procainamide, tricyclic antidepressants, cocaine)
- Electrolyte effect (hyperkalemia, hypermagnesemia)
- Premature ventricular contractions
- Right and left bundle branch blocks
- Supraventricular beats with aberration
- Ventricular escape beats
- Ventricular pacemaker beats
- Wolff-Parkinson-White syndrome

QT Interval

The *QT interval* is the time required for depolarization and repolarization of the ventricles, measured from the beginning of the QRS complex to the end of the T wave. The normal QT interval varies with heart rate. Fast rates shorten the QT interval and slow heart rates lengthen it.

At normal heart rates the QT interval lasts between 0.34 and 0.42 seconds. It is considered abnormally long if it is greater than 0.40 seconds (10 small squares) for males and 0.44 seconds (11 small squares) for females.

A way to compensate for changes in the QT interval with heart rate is to use Hodge's formula:

$$QTc = QT + 0.00175(\text{heart rate} - 60)$$

where QTc is the corrected QT interval. Normally it should be less than 0.44 seconds. If the QTc is prolonged there is a risk of ventricular arrhythmia, in particular Torsades de pointes. Females have a QT interval slightly longer than that of males.

Segments

PR Segment

The *PR segment* is the portion of the tracing falling between the end of the P wave and the beginning of the QRS complex. During this time, the electrical wave moves slowly through the atrioventricular node. The PR segment is not routinely measured, but may be commented on if it is depressed or elevated. A common cause of PR segment depression is pericarditis.

ST Segment

The *ST segment* is the portion of the tracing falling between the end of the QRS complex and the beginning of the T wave. During this time, the ventricle is contracting, but no electricity is flowing. The ST segment is therefore usually at the baseline. ST segment elevation or depression is generally measured at a point two small squares beyond the end of the QRS complex.

The length of the ST segment shortens with increasing heart rate. Measurement of the length of the ST segment alone is usually not of any clinical use; however, ST segment depression and elevation can be clinically important.

ST segment depression can occur with:

- Acute posterior myocardial infarction
- Angina
- Drug effects (digoxin, quinidine)
- Electrolyte effects (hypokalemia, hypercalcemia, hypermagnesium)
- Hypothermia
- Left bundle branch block
- Pulmonary embolus
- Reciprocal changes representing cardiac injury in other leads
- Supraventricular tachycardia
- Ventricular hypertrophy with strain

ST segment elevation can occur with:

- Acute pericarditis
- Myocarditis
- Athletic heart syndrome
- Brugada syndrome (congenital abnormality)
- Cardiomyopathy
- CNS events, such as subarachnoid hemorrhage
- Early repolarization
- Hyperkalemia

- Left ventricular aneurysm
- Reciprocal changes due to ischemia in other leads
- ST-segment elevation myocardial infarction
- Vasospasm (Prinzmetal's angina, cocaine or methamphetamine abuse)

ST-T Complex

The *repolarization complex* (ST-T) is the most sensitive part of the electrocardiogram. It consists of the ST segment and the T wave. ST-T complexes can change in duration, amplitude, and sign, or combinations of these. The ST-T complex can be influenced by many nonpathological factors, including temperature, hyperventilation, and anxiety.

The diagnosis of *nonspecific ST-T abnormality* is made when the repolarization complex is abnormal, but not suggestive of a specific diagnosis. The most common nonspecific ST-T abnormality is low T wave voltages with slight sagging or flattening of the ST segment.

J Point

The *J point* marks the end of ventricular depolarization (Figure 1-7). It is the point of intersection between the end of the QRS complex and the onset of the ST segment. As such, it is an essential landmark for measuring QRS duration. At times, the J point can be difficult to identify.

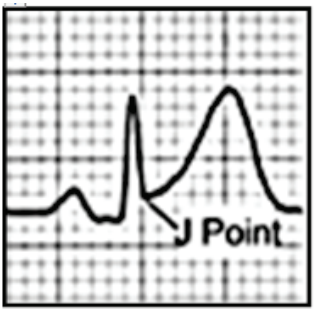

Figure 1-7. The J Point.

ELECTROCARDIOGRAPHIC INTERPRETATION

In interpreting an ECG one looks in order at seven areas on each ECG:

1. Calibration standard (half or full standard)
2. Rate (normal, greater than normal, less than normal)
3. Rhythm (regular, regularly irregular, irregularly irregular)
4. Axis (normal axis, left axis deviation, right axis deviation)
5. Intervals (PR, QRS, QT), segments (PR, ST)
6. Signs of atrial enlargement or ventricular hypertrophy (P wave morphology, greater than normal magnitudes of QRS complexes)
7. Signs of ischemia and infarction (ST segment elevations and depressions, Q waves)

If there is a previous ECG in the patient's file, the current ECG should be compared with it to see if any significant changes have occurred.

From all of the above information, taking into account the patient's symptoms and history, we arrive at an ECG interpretation.

Chapter 2

Leads and the Normal Electrocardiogram

LEADS

Two types of arrangements of leads are used—bipolar leads and unipolar leads.

Bipolar Leads

A *bipolar lead* is one in which the electrical activity at one electrode is compared with that of another, the net result being the measurement of electrical activity between the two electrodes (Figure 2-1).

Figure 2-1. Formation of a bipolar lead.

By convention, a positive electrode is one in which the electrocardiograph records a positive (upward) signal when the electrical impulse flows toward it and a negative (downward) signal when the electrical impulse flows away from it.

Figure 2-2 shows depolarization of a hypothetical strip of cardiac muscle and the corresponding generation of the electrocardiogram tracing. In segment A the muscle is in its normal state of polarization. Hence, the electrocardiogram recorded from both ends is at zero. In segment B the depolarization wave is traveling away from the electrode at the left and toward the one at the right. This results in a negative deflection in the left electrode and

a positive deflection in the right electrode. In segment C the muscle strip is completely depolarized so the electrocardiogram tracings have returned to zero.

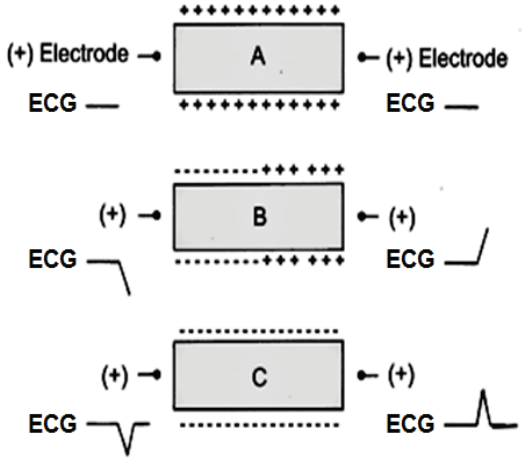

Figure 2-2. Depolarization of a hypothetical strip of cardiac muscle and the corresponding generation of the electrocardiogram.

The Standard Limb Leads

By attaching electrodes to the left arm, right arm, and a leg, we obtain the three bipolar *standard limb leads*, named I, II, and III.

The standard limb lead placements are formed as follows:

- **Lead I:** Negative electrode placed on the right arm and positive electrode placed on the left arm
- **Lead II:** Negative electrode placed on the right arm and positive electrode placed on the left leg
- **Lead III:** Negative electrode placed on the left arm and positive electrode placed on the left leg

The right arm and left arm electrodes alternatively may be placed on the right and left shoulders, respectively. The leg electrode can be placed on the thigh. The standard limb leads form a set of axes 60 degrees apart (Figure 2-3).

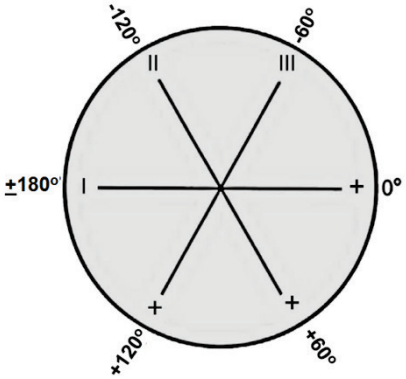

Figure 2-3. Axes of the three bipolar limb leads (I, II, III).

Unipolar Leads

A *unipolar lead* is one in which the electrical potential at an electrode is compared to a reference point that averages electrical activity of combined leads. The single electrode, termed the *exploring electrode*, is the positive electrode.

There are two sets of unipolar leads—the augmented limb leads and the chest (precordial) leads.

Augmented Limb Leads

A second set of limb leads (aV_R, aV_L, AV_F) are unipolar leads. The "a" stands augmented, "V" for voltage, "R" for right arm, "L" for the left arm, and "F" for the foot. They are referred to as augmented leads because an electrical manipulation is done to increase the size of the voltage recordings.

In actuality, augmented limb leads use the same electrodes as leads I, II, and III. The ECG machine switches and rearranges the electrode designations.

Each augmented lead is formed by combining the potentials from two electrodes to form the exploring electrode, which becomes the positive electrode (Figure 2-4).