ELECTROCARDIOGRAPHY
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A GUIDE FOR PHYSICIANS, MEDICAL STUDENTS, NURSE PRACTITIONERS, AND OTHER HEALTHCARE PROVIDERS

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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.

Primary care 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 primary care physician, with most physicians reading their own recordings and basing clinical decisions on their findings. It has been shown that primary care 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 primary care physicians an essential skill. This book provides the necessary skills for primary care physicians to use in interpreting electrocardiograms, both in their offices and in the emergency departments of their hospitals.

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 primary care physicians in their everyday practice of medicine.

This book is the result of a course I taught in the Department 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.

Although this book was written with the primary care physician in mind, it should prove useful to medical students, residents in all primary care specialties, primary care nurse practitioners, and physician assistants. It is an outgrowth of my prior book Electrocardiography for the Family Physician.

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

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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.

![Electrocardiograph paper dimensions (full standard).](image)

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* (*atrioventricular bundle*), which divides into the *right* and *left bundle branches* (Figure 1-2).
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.

The blood supply of the SA and AV nodes usually originates from the right coronary artery. However, ten percent of the time the blood supply to the AV node arises from the circumflex artery.

**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 is normally largest in lead II and positive in leads I, II and $V_3-V_6$. It is normally negative in lead aVR and may be biphasic in leads $V_1$ and $V_2$. 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 low in the atria or 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. An *S wave* is an upward deflection that is preceded by a downward deflection (Figure 1-4).

![Figure 1-4. Components of the QRS complex.](image)

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 1-5).

![Figure 1-5. Examples of various QRS complex morphologies and their nomenclatures](image)

If a second upward deflection is seen, it is called an R-prime (R’) wave. An R followed by an extension below the baseline is an S, giving an RSR’. RR’ and RSR’ waves are never normal in adults but indicate a problem in the ventricular conduction system.

Causes of an RR’ or RSR’ include bundle branch block and incomplete bundle branch block. An RSR’ configuration can be normal in the right-most chest lead (V1) of young children.

The width of the QRS complex 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 duration 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 all leads except \( aV_R \) and \( V_1 \). It is normally inverted in lead \( aV_R \).

The height of the T wave is normally less than five millimeters (0.5 mV) in the standard limb leads and less than 10 mm (1.0 mV) 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.

Concordance and discordance

Concordance and discordance have to do with the direction of the T wave in relation to the direction of the QRS complex).

- **Concordance**: The direction of the T wave is in the same direction as that of the main QRS deflection (Figure 1-6A)
- **Discordance**: The direction of the T wave is in the opposite direction than that of the main QRS deflection (Figure 1-6B)

![Figure 1-6. (A) Concordance and (B) discordance.](image)

U Wave

When present, a second wave following the T wave is called a *U wave* (Figure 1-7). 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 1-7. The U wave.](image)

U waves are most prominent in leads V2 and V3. Their Usual direction is the same as the T wave; however, in some cases they may be inverted.

The most common cause of prominent U waves is bradycardia, generally becoming visible when the heart rate falls below 65 bpm. Other causes of positive U waves are:
Inverted U waves may be seen with:

- Coronary artery spasm (Prinzmetal's angina)
- Myocardial infarction
- During episode of acute ischemia
- Some cases of left ventricular hypertrophy or right ventricular hypertrophy
- Some patients with long QT syndrome

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 1-8).

![Figure 1-8. Intervals and segments in the electrocardiogram.](image-url)
PR Interval

The \textit{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 AV node
- Digoxin, beta blockers, some calcium channel blockers (diltiazem, verapamil)
- Electrolyte abnormalities (hyperkalemia, hypercalcemia)
- Hypothermia
- Hyperthyroidism

The PR interval may be unusually short with:

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

QT Interval

The \textit{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. A way to compensate for changes in the QT interval with heart rate is to use a formula such as Hodge’s formula:

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

where QTc is the corrected QT interval. The formula corrects QT for all ECGs to a heart rate of 60 bpm.

The corrected QT interval (QTc) should be less than 0.44 seconds in males and 0.46 seconds in females. If the QTc is prolonged there is a risk of ventricular arrhythmia, in particular Torsades de pointes. Torsades de pointes is a ventricular tachycardia characterized by fluctuation of the QRS complex magnitudes around the electrocardiographic baseline (Figure 1-9).

Females normally have a QT interval slightly longer than that of males.

The QT interval may be prolonged with:

- Over 50 medications, including amiodarone, chlorpromazine, clarithromycin, erythromycin, haloperidol, thioridazine, type Ia antiarrhythmic agents (quinidine, procainamide, disopyramide), and other antiarrhythmics.
- Congestive heart failure
- Hypothyroidism
- Hypothermia
- Electrolyte abnormalities (hypokalemia, hypocalcemia, hypomagnesemia)
- Myocardial ischemia and infarction
- Myocarditis
- Organophosphate insecticide poisoning
- Severe CNS events (seizures, CVA, intracranial hemorrhage)
- Hereditary diseases (Jervell and Lang Nielson syndrome, Romano Ward syndrome)
Other formulas for calculating QTc are:

- **Linear formula**
  - Framingham formula: \( QTc = QT + 0.154 (1 – RR) \)

- **Logarithmic formulas**
  - Bazett’s formula: \( QTc = \frac{QT}{(RR)^{0.5}} \)
  - Fridericia’s formula: \( QTc = \frac{QT}{(RR)^{0.33}} \)
  - Baseline correction: \( QTc = \frac{QT}{(RR)^{0.37}} \)

All formulas give similar results.

**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, hypermagnesiu)m
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 (Figure 1-10).

Figure 1-10. Sagging and flattening of the ST segment

J Point

The J point marks the end of ventricular depolarization (Figure 1-11). 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 1-11. The J Point.

ELECTROCARDIOGRAPHIC INTERPRETATION

In interpreting an ECG, one looks in order at eight 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, etc.)
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)
8. Other findings

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 1 Quiz

Electrocardiograph paper (questions 1 to 4)

5. PR interval
6. QT interval
7. QRS width
8. PR segment
9. ST segment

10. What is the magnitude of the J-point in mV)?
18. If the heart rate is 120 bpm and QT is 0.44 seconds, what is the corrected QT (QTc) using Hodge’s formula?

19. Is the corrected QT (QTc) above:
   A. Normal
   B. Prolonged
   C. Short
   D. Both prolonged and short (Schrödinger’s QTc)