Electrocardiography for the Family Physician

The Essentials

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

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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 competency and 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 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 Re-certification.

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.

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 (digitalis, quinidine)
- Evidence of abnormal blood electrolytes (potassium, calcium, magnesium)
- 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)

Electrocardiography is not indicated for screening of healthy subjects without symptoms of heart disease, hypertension, or other risk factors for the development of heart disease.

ELECTROCARDIOGRAPHIC INTERPRETATION

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

1. Calibration standard (half or full standard)
2. Rate (normal, greater than normal, less than normal)
3. Rhythm (presence of arrhythmia)
4. Axis (normal axis, left axis deviation, right axis deviation)
5. Intervals (P-R, QRS, Q-T)
6. Signs of atrial enlargement or ventricular hypertrophy (P wave morphology, greater that normal magnitudes of QRS complexes)
7. Signs of ischemia and infarct (ST segment elevation or depression, Q waves)

If there is a previous EKG in the patient's file, the current EKG 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 EKG interpretation.

ELECTROCARDIOGRAPH PAPER

The electrocardiogram is recorded on graph paper with divisions as indicated in Figure 1-1. Usually a small square is 0.04 seconds wide and one millimeter (0.1 mV) high. A large square is 0.2 seconds wide and five mm (0.5 mV) high. Paper speed is 25 mm per second.

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 as above. When recorded at half standard because of large QRS voltages, the calibration standard is 5 mm, 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.

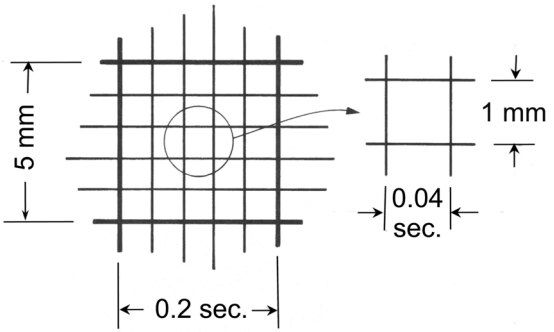

Figure 1-1. Electrocardiograph paper dimensions.

CONDUCTION SYSTEM OF THE HEART

Electrical activation of the myocardium 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*, which divides into the *right* and *left bundle branches* (Figure 1-2). The left

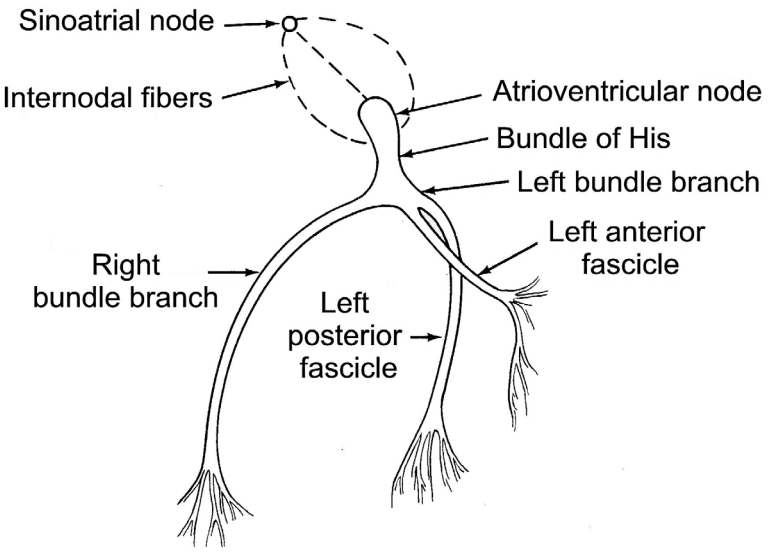

Figure 1-2. The conduction system of the heart.

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*, or restoration, of the electrical potential of the myocardial 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 results in various deflections recorded on EKG paper. From this recording, various waves, intervals, and segments also 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.

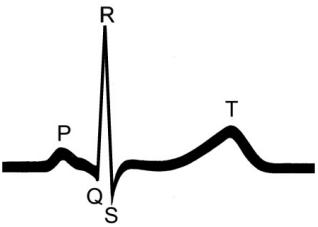

Figure 1-3. The deflections of the electrocardiogram generated by the heart during depolarization and repolarization.

P Wave

The *P wave* is normally largest in lead II and is upright in all leads except aVR. 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 and others
- **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 downward stroke following an upward stroke is designated as an *S wave*. A downward deflection without any upward deflections, before or after, is known as a *QS wave*.

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.

Causes of QRS widening include

- Drug effect (quinidine)
- Electrolyte effect (hyperkalemia)

- 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 aVR. T waves are variable in the other leads. T waves up to 2.5 mm in height are considered normal. The direction normally follows the direction of the main QRS deflection.

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

- **Tall T waves.** Hyperkalemia, very early myocardial infarction, and left bundle branch block
- **Flat or small T waves.** Ischemia, evolving myocardial infarction, bundle branch blocks, 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, digitalis, athletic heart syndrome, and acute cerebral disease

In young children, T waves 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*. It represents the last phase of ventricular repolarization and its normal direction 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. U waves are best seen in leads V_2 and V_3. Their exact significance is unknown.

U waves may be seen in:

- Cerebral accidents (occasionally)
- Hyperthyroidism
- Hypokalemia
- Left ventricular hypertrophy (right precordial leads with deep S waves)
- Medications (quinidine and other type 1A antiarrhythmics, phenothiazines)
- Sinus bradycardia (accentuates the U wave)

Inverted U waves may be seen with:

- Hypertension
- Ischemia (angina or exercise-induced ischemia)
- Myocardial infarction (in leads with pathologic Q waves)
- Prinzmetal's angina
- Some cases of ventricular hypertrophy (usually in leads with prominent R waves)

Low Voltage Deflections

On occasion an EKG will be seen in which all of the deflections from baseline are much less than expected … the low-voltage EKG. Several factors lead to low voltage deflections. These include:

- Diffuse myocardial fibrosis
- Emphysema (over-expanded lungs act to insulate the heart)
- Half standard (when full standard is incorrectly expected)
- Hypothyroidism (bradycardia may also be present)
- Infiltration of the heart muscle with substances such as amyloid
- Normal variant
- Obesity (fat tissue between the heart and the chest wall)

- Pericardial effusion (fluid between the heart and the chest wall)

Intervals

The P-R, QRS, and Q-T intervals fall within well-defined limits (Figure 1-4).

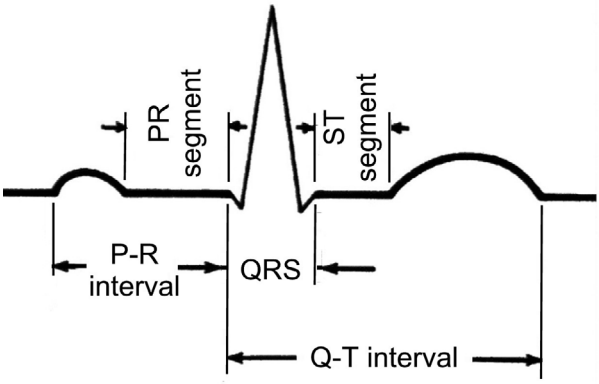

Figure 1-4. Intervals and segments in the electrocardiogram.

P-R Interval

The *P-R 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 P-R 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 P-R interval may be unusually short with:

- Electrolyte abnormalities (hypokalemia, hypocalcemia)
- Type II glycogen storage disease (Pompe's disease)
- Hypertension
- Hypertrophic cardiomyopathy
- Junctional rhythm
- Low-atrial pacemaker
- Normal variant
- 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 indicates some blockage of the electrical action in the conducting system. This may be due to:

- Electrolyte abnormality (hyperkalemia)
- Hypothermia
- Ischemia
- Medications (flucainamide, tricyclics)
- Necrosis of the conducting tissue (bundle branch blocks)
- Nonspecific intraventricular conduction defect

Q-T Interval

The *Q-T 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 Q-T interval varies with heart rate. Fast rates shorten the Q-T interval and slow heart rates lengthen it.

The Q-T interval represents about 40 percent of the total time between QRS complexes (the *R-R interval*). In most cases the Q-T

interval lasts from between 0.34 to 0.42 seconds.

At normal heart rates, the Q-T interval is considered abnormal if it is greater than 0.40 sec (10 small squares) for males and 0.44 sec (11 small squares) for females. For every 10 beats per minute (bpm) above 70 bpm, subtract 0.02 seconds. Add 0.02 seconds for every 10 bpm below 70.

Long QT syndrome (LQTS) is a disorder of the heart's electrical system. The condition leaves affected individuals vulnerable to fast, chaotic heartbeats that may lead to fainting, and in some cases cardiac arrest and sudden death. Long Q-T syndrome can be genetic or caused by more than 50 medications, as well as electrolyte abnormalities and various medical conditions.

The Q-T interval may be prolonged with:

- Certain medications, including amiodarone, chlorpromazine, clarithromycin, erythromycin, haloperidol, procainamide, quinidine, thioridazine
- Congestive heart failure
- Electrolyte abnormalities (hypokalemia, hypocalcemia, hypomagnesemia)
- Hereditary diseases (Romano Ward syndrome, Jervill-Lang Nielson syndrome)
- Hypothyroidism
- Myocardial ischemia and infarction
- Myocarditis
- Organophosphate insecticide poisoning
- Severe CNS events (CVA, seizures, intracranial hemorrhage)
- Slow heart rates
- Type Ia antiarrhythmic agents (quinidine, procainamide, disopyramide), as well as other antiarrhythmics

The Q-T interval may be shortened with:

- Congenital disorder (Short QT syndrome)
- Electrolytes (hypercalcemia, hyperkalemia)
- Fast heart rates
- Hyperthyroidism
- Medications (digitalis)

Short QT syndrome is a genetic disease of the electrical system of the heart. Structurally, the heart appears normal. The disorder appears to be inherited in an autosomal dominant pattern.

Individuals with short QT syndrome frequently complain of palpitations and may have syncope that is unexplained. Most individuals will have family members with a history of palpitations, atrial fibrillation, or unexplained or sudden death at a young age. Death is most likely due to ventricular fibrillation.

The characteristic finding of short QT syndrome on EKG is a short QT interval, typically less than or equal to 300 milliseconds, that doesn't significantly change with the heart rate. Tall, peaked T waves also may be noted. Individuals also may have atrial fibrillation.

Segments

The PR and ST segments fall within well defined limits (Figure 1-4).

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. The PR segment may be depressed in 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. Abnormality of electrolytes may also affect the ST segment length. Measurement of the length of the ST segment alone is usu-

ally not of any clinical use; however, ST segment depression and elevation can be clinically important.

ST segment depression can indicate:

- Acute posterior myocardial infarction
- Classical angina
- Drug effects (digitalis, quinidine)
- Electrolyte effects (hypokalemia, hypercalcemia, hypomagnesium)
- 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 and 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
- Transmural 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 (Figure 1-4). 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 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. It is the point of intersection between the end of the QRS complex and the onset of the ST segment (Figure 1-5). As such, it is an essential landmark for measuring QRS duration. At times the J point can be difficult to identify.

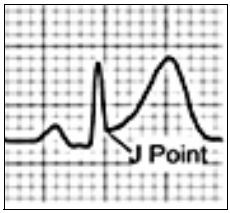

Figure 1-5. The J Point.

Chapter 2

Leads and the Normal Electrocardiogram

LEADS

Two types of arrangements of leads are used … bipolar and unipolar 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 two electrodes. 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.

A *unipolar lead* is one in which the electrical potential at an electrode is compared to a reference point that averages electrical activity rather than to that of another electrode. In this instance, the single electrode, termed the *exploring electrode*, is the positive electrode.

Figure 2-1 illustrates the process by which an electrocardiogram is generated during depolarization. In this figure, the electrical activity of a hypothetical strip of cardiac muscle is monitored at each end by a positive electrode. 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 electro-

cardiogram tracings have returned to zero.

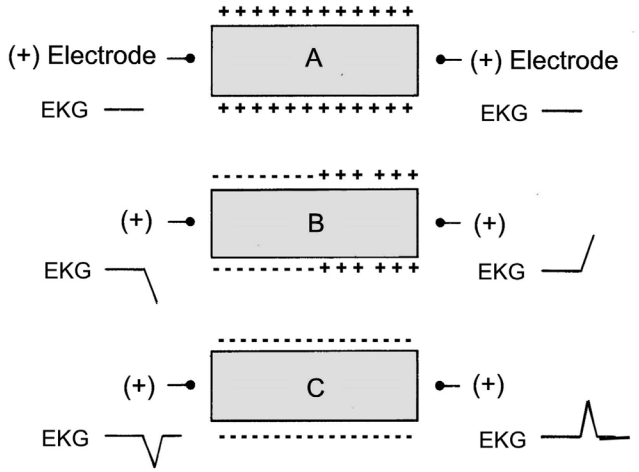

Figure 2-1. Depolarization of a hypothetical strip of cardiac muscle and corresponding generation of the electrocardiogram. (A) The muscle strip is in a polarized state. (B) The depolarization wave travels from left to right. (C) The muscle strip is depolarized.

The Limb Leads

The six limb leads (I, II, III, aVR, aVL, aVF) explore the electrical activity of the heart in a frontal plane; that is, the orientation of the heart seen when looking directly at the anterior chest.

Standard Limb Leads

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

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

The right arm and left arm electrodes alternatively may be placed on the right and left shoulders, respectively.

The standard limb leads are bipolar leads and form a set of axes 60 degrees apart (Figure 2-2).

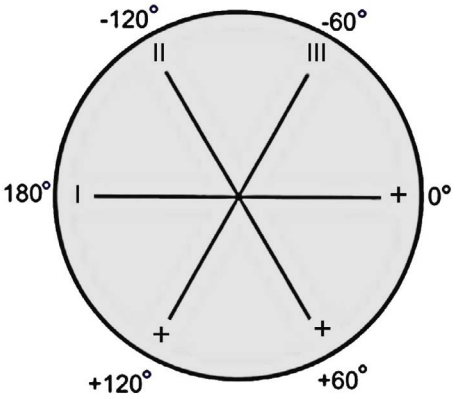

Figure 2-2. Axes of the three bipolar limb leads (I, II, III).

Augmented Limb Leads

A second set of limb leads (aVR, aVL, AVF) are unipolar leads, and also form a set of axes 60 degrees apart. However, these axes are rotated 30 degrees from the axes of the standard limb leads. This very conveniently gives a lead every 30 degrees (Figure 2-3).

Leads aVR, aVL, and aVF are referred to as *augmented leads* because an electrical manipulation is done to increase the size of the voltage recordings. The "a" stands augmented, "V" for voltage, "R" for right arm (shoulder), "L" for the left arm (shoulder), and "F" for the foot.

- **aVR.** Created by connecting the left arm and the left foot (leg) **e**lectrodes together to form the negative "electrode." The voltage of this average electrode is compared to the right arm electrode (positive electrode). This means that if electricity is moving in a normal leftward direction, lead aVR will record a downward wave.
- **aVL.** Created by connecting the right arm and the foot together to form a negative "electrode," then comparing this

"average" electrode to the left arm electrode. The left arm electrode is positive, meaning that electricity moving to the left will cause an upward motion of the EKG stylus in aVL

- **aVF.** Created by connecting the two arms together to create an "average" electrode. To the EKG machine, this combination looks like a single negative electrode midway between the two arms (directly in the center of the body above the heart). This "average" electrode is connected through the EKG machine to the left foot electrode. The foot is the positive electrode, so a downward motion of electricity will make the EKG stylus move upward on the paper in aVF.

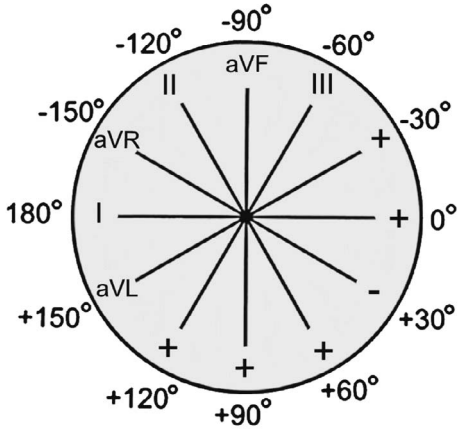

Figure 2-3. Axes of all six limb leads (I, II, III, aVL, aVR, aVF).

The Chest Leads

The six *chest (precordial) leads* (V_1, V_2, V_3, V_4, V_5, V_6) are unipolar leads that explore the electrical activity of the heart in a horizontal plane; that is, as if looking down on a cross section of the body at the level of the heart. The reference point is obtained by connecting the left arm, right arm, and left leg electrodes together.

The standard positions for the exploring chest electrodes are located with reference to various landmarks of the bony thorax:

- V_1 is in the fourth intercostal space just to the right of the sternum.

- V_2 is at the fourth intercostal space just to the left of the sternum.
- V_3 is halfway between V_2 and V_4.
- V_4 is at the fifth intercostals space in the midclavicular line.
- V_5 is in the anterior axillary line at the same level as V_4.
- V_6 is at the same level in the midaxillary line.

Generation of the depolarization wave of the ventricles is shown schematically in Figure 2-4. Left ventricular muscle mass is much greater than that of right ventricular mass and as a result the forces of left ventricular depolarization normally mask those of right ventricular depolarization. Leads V_1 and V_2 monitor the electrical activity of the heart from the right side, V_3 and V_4 from the anterior aspect, and V_5 and V_6 from the left side.

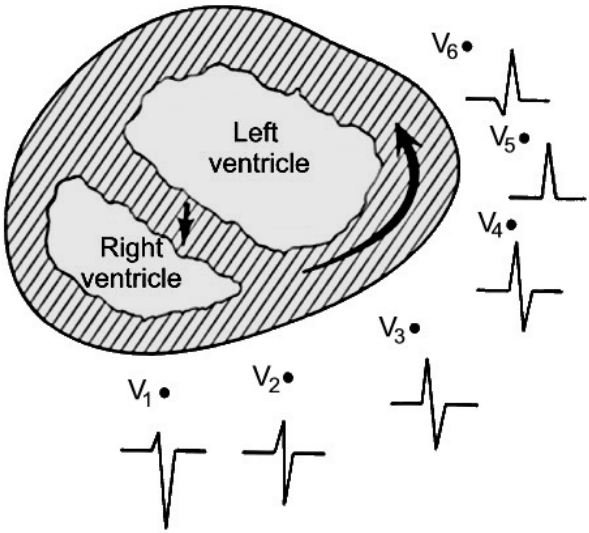

Figure 2-4. QRS waves in the six chest leads. The recorded signal becomes positive as the depolarization wave moves toward an electrode and negative as it moves away from an electrode.

It should be noted that leads V_1 and V_6 essentially monitor the electrical activity from opposite sides of the heart, hence registering reciprocal voltages. The QRS complexes in these two leads develop during depolarization as follows: Since initial myocardial depolarization occurs in the septum from left to right

(small arrow), the electrical signal travels toward V_1 and away from V_6, causing an initial small upward deflection in V_1 and a downward deflection in V_6. After septal depolarization, the wave sweeps downward around the lower part of the heart and then back up the myocardium (large arrow) away from V_1 and toward V_6, causing a downward deflection in V_1 and an upward deflection in V_6. As depolarization is completed, the recording returns to baseline.

R-Wave Progression

V_1 normally consists of a small R wave and a large S wave, whereas V_6 consists of a small Q wave and a large R wave. Since V_3 and V_4 are located midway between V_1 and V_6, the QRS complex would be expected to be nearly isoelectric in one of these leads; that is, the positive deflection and the negative deflection will be about equal (Figure 2-4). The magnitude of the deflection of V_2 lies between that of V_1 and V_3 and the magnitude of the deflection of V_5 between that of V_4 and V_6. Hence, a progression of the R wave occurs, getting progressively more positive from V_1 to V_6. R-wave progression may be abnormal in anterior myocardial infarction and a number of other conditions.

Lead Combinations

The various limb and chest leads can be grouped in combinations depending on the anatomical area of the heart they monitor.

- **Lateral leads.** The lateral leads (I, aVL, V_4-V_6) monitor the electrical activity of the lateral aspect of the heart.
- **Inferior (diaphragmatic) leads.** The inferior leads (II, III, aVF) monitor the electrical activity of the underside of the heart.
- **Anteroseptal.** The anteroseptal leads (V_1-V_4) monitor the right side and the anterior aspect of the heart, with V_1 and V_2 being septal leads and V_3 and V_4 being anterior leads.

The NORMAL EKG

Mean Electrical Activity

The *QRS axis* is the "average" direction of electrical activity during ventricular depolarization (*mean electrical activity*). The mean electrical axis may shift due to physical change in the position of the heart, chamber hypertrophy, or conduction block.

The mean electrical activity of ventricular depolarization in the frontal plane can be represented by a vector. The length of this vector indicates the magnitude of the activity. The mean direction of the depolarization wave is represented by the angle of the vector in regard to the six limb leads.

In Figure 2-5, the mean vector is determined from the magnitudes of the QRS voltages in leads I and aVF. The normal mean vector usually lies between 0 and 90 degrees, averaging 58 degrees as shown. The mean vector can be determined using the magnitudes of QRS complexes in other leads to project lines at 90 degrees from their tips to a crossing point, which is the tip of the mean vector.

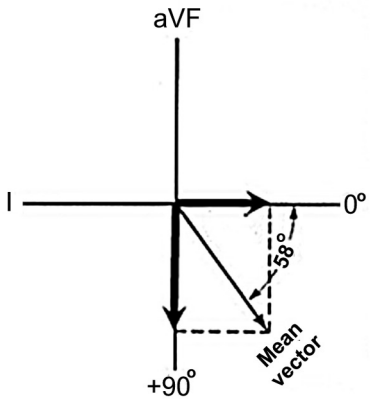

Figure 2-5. The mean vector of ventricular depolarization as determined from the QRS magnitudes in leads I and aVF.

Criteria

A normal 12-lead electrocardiogram is shown in Figure 2-6. A *rhythm strip* is displayed along the bottom. Parameters associated with a normal electrocardiogram include:

1. Pulse rate is between 60 and 100 beats per minute (bpm).

2. Rhythm is regular except for minor variations with respiration, usually no more than 10 percent.
3. A P wave precedes every QRS complex.
4. P waves are normally positive or biphasic in all leads except aVR, and sometimes V_1.
5. P waves in lead II should be upright. Otherwise sinus rhythm is not present.
6. P-R interval is the time required for completion of atrial depolarization; conduction through the AV note, bundle of His, and bundle branches; and arrival at the ventricular myocardial cells. The normal P-R interval is 0.12 to 0.20 seconds.
7. The QRS interval represents the time required for ventricular cells to depolarize. The normal duration is 0.06 to 0.10 seconds.
8. The Q-T interval is the time required for depolarization and repolarization of the ventricles. The time required is inversely proportional to the heart rate; the faster the heart rate the shorter the Q-T interval. With slower heart rates, the Q-T interval is longer. Normally. the Q-T interval represents about 40 percent of the total time between the QRS complexes (the R-R interval). In most cases, the Q-T interval lasts between 0.34 and 0.42 seconds.
9. Lead I is a mirror image of aVR.
10. The QRS deflections in leads I and III approximately equal that of lead II. Similarly, the sum of The QRS deflections in aVR, aVL, and aVF should approximately equal zero. When this is not true, there is reason to suspect the electrodes were placed incorrectly or that recordings were mixed up during mounting.
11. R-wave progression occurs in the chest leads, with the transition zone (point of equal positive and negative voltages) occurring somewhere between V_3 and V_4.

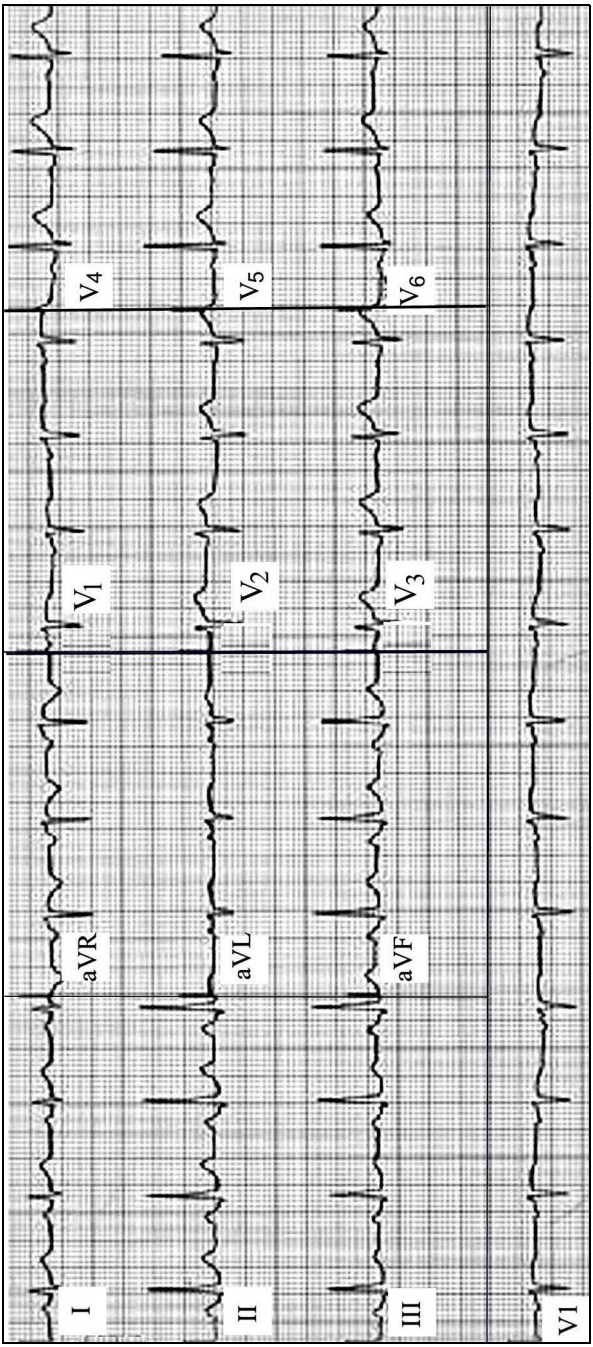

Figure 2-6. A normal 12-lead electrocardiogram.

Chapter 3

Heart Rate and Axis

HEART RATE

A normal *heart rate* is defined as 60 to 100 bpm. The rate is normally set by the rhythm of the sinoatrial node, which is located in the posterior wall of the right atrium. When the sinoatrial node fails to function as the pacemaker, another area of the heart assumes the pacemaker role. This area may be another area of the atria, the atrioventricular node, or an area of the ventricles. When an area of the atria other than the sinoatrial node becomes the pacemaker, the rate is usually about the same as that of the sinoatrial node. Should the atrioventricular node become the pacemaker, the rate will be about 60 bpm. If an area of the ventricles assumes the role, the rate will be 30 to 40 bpm.

Heart rate can be estimated in straight-forward fashion from an electrocardiogram tracing (Figure 3-1). The R-R interval is inversely proportional to heart rate. At a normal paper speed of 25 mm per second, an R-R interval of 0.2 seconds (one large square) indicates a rate of 300 bpm. An R-R interval of two large squares indicates a rate of half as fast, or 150 bpm. Similarly, three large squares represent 100 bpm, four large squares 75 bpm, five large squares 60 bpm, and six large squares 50 bpm.

Heart rates of less that 50 bpm are estimated as follows: The electrocardiogram has small vertical markings above the graph portion at three-second intervals. Heart rate is estimated by multiplying the number and portions of cycles in a six-second interval

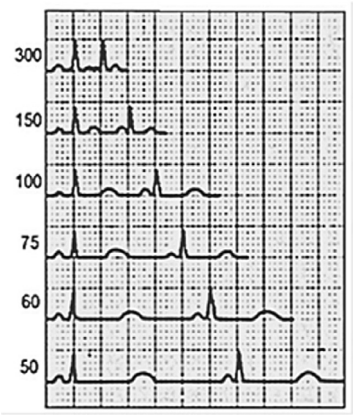

Figure 3-1. Heart rates between 50 and 300 bpm can be estimated from the number of large squares in an R-R interval.

by ten. For example, if there are 3-1/2 cycles in a six-second interval, the heart rate is 35 bpm (3-1/2 x 10 = 35). Some examples of heart rates of less than 50 bpm are shown in Figure 3-2.

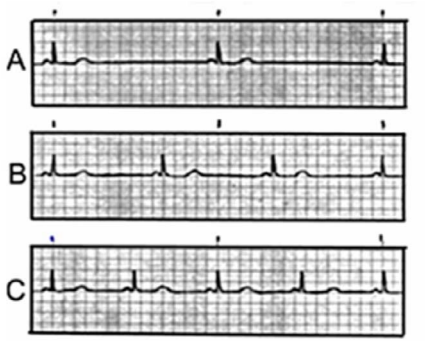

Figure 3-2. Heart rates less than 50 bpm can be estimated with the aid of markings at three-second intervals along the top of the graph paper: (A) 20 bpm, (B) 30 bpm, and (C) 40 bpm.

AXIS

Axis refers to the direction of the mean vector of ventricular depolarization (Figure 3-3). An axis between +90 degrees and 0 degrees is normal, and an axis between 0 degrees and -30 degrees is in a "gray" zone that is usually considered normal. More negative than -30 is called left axis deviation. More positive than +90 is called right axis deviation.

Right Axis Deviation

When the direction of the mean vector is greater than +90 degrees, *right axis deviation (RAD)* is present. Rarely, extreme RAD may be present when the direction of the mean vector lies between 180 degrees and -90 degrees. RAD in this range is much less common than left axis deviation.

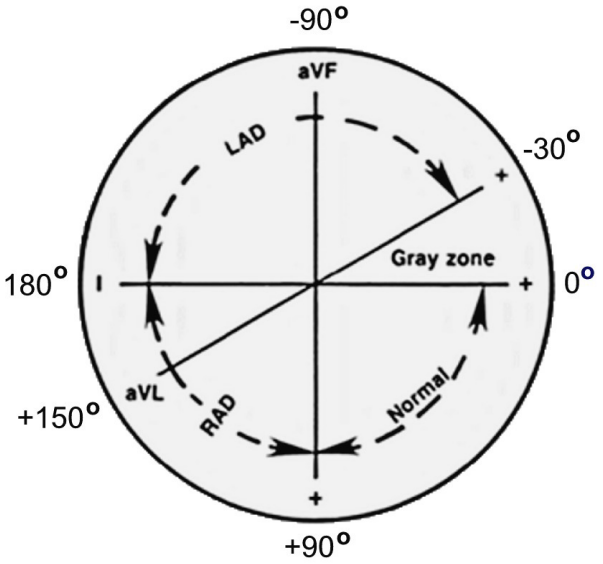

Figure 3-3. The mean vector of ventricular depolarization may show right or left axis deviation (RAD, LAD) or fall in a normal or gray zone.

Causes of right axis deviation include:

- Acute pulmonary embolus (sudden shift to the right)
- Anterolateral myocardial infarction
- Atrial septal defect
- Chronic lung disease (emphysema, chronic bronchitis)
- Dextrocardia
- Left posterior hemiblock
- Normal finding in children and tall thin adults
- Reversed arm leads
- Right ventricular hypertrophy
- Ventricular septal defect
- Wolff-Parkinson-White syndrome with left-sided accessory pathway

Left Axis Deviation

An axis more negative than -30 is called *left axis deviation*. Causes of left axis deviation include:

- Emphysema
- Hyperkalemia
- Hypertension
- Left anterior hemiblock
- Left bundle branch block
- Left ventricular hypertrophy
- Ostium primum atrial septal defect
- Past anterior myocardial infarction
- Subaortic stenosis
- Valvular heart disease (aortic stenosis or regurgitation, mitral regurgitation, tricuspid atresia)
- Ventricular pacing
- Wolff-Parkinson-White syndrome (right-sided accessory pathway)

An axis between -90 and 180 degrees has been designated the *Northwest Axis* or *No Man's Land* because either RAD or LAD may be present in this zone, although LAD is far more common. Therefore, an axis in this area will be considered LAD without evidence that RAD should be considered.

Northwest axis is most commonly caused by:

- Artificial cardiac pacing
- Emphysema
- Hyperkalemia
- Lead transposition
- Ventricular tachycardia

Determining the Axis of the Mean Vector

Several methods exist for determining the *axis of the mean vector*. Two will be discussed here.

Method One

The first method consists of going through a series of steps. Step one is to determine if a normal axis is present. If not, first RAD, then the gray zone, and then LAD are checked for.

The axes of most mean vectors lie between 0 degrees and +90 degrees (Figure 3-4). This means that the QRS complexes for

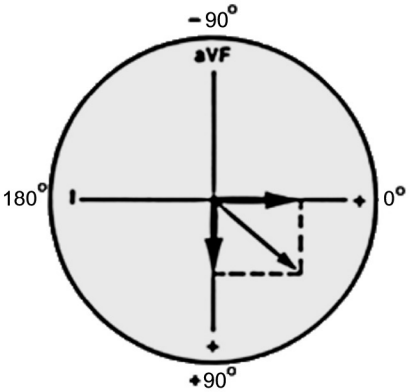

Figure 3-4. Normal axis. If the QRS complexes for leads aVF and I are positive, then the mean vector lies between 0 and +90 degrees.

lead I and lead aVF will be positive (that is, upward deflection is greater than downward deflection). Hence, it suffices merely to check the electrocardiogram to see if these leads are both positive. If so, a normal axis is present. However, if lead aVF is positive and lead I is negative, RAD is present (Figure 3-5).

On the other hand, if lead aVF is negative, LAD may be present or the mean vector may lie in the gray zone. If lead II is positive, the axis lies within the gray zone (Figure 3-6). If lead II is negative, then LAD is present (Figure 3-7).

To summarize, the axis of the mean vector can be determined by the following steps (Table 3-1): First check lead aVF. If it is positive, then observe lead I. If lead I also is positive, then a normal axis is present. If lead I is negative, then RAD is present. If lead aVF is negative, then observe lead II. If lead II is positive,

then the axis lies within the gray zone. However, if lead II is negative, LAD is present.

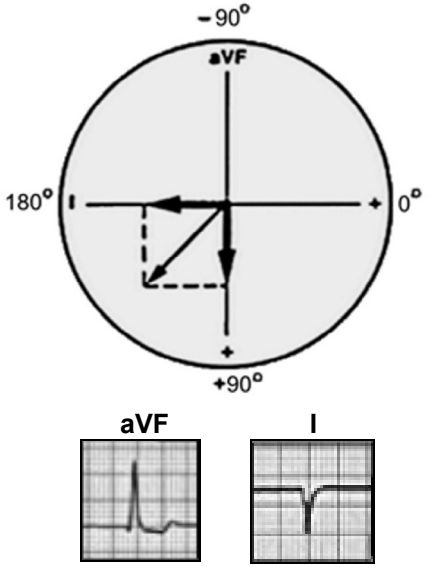

Figure 3-5. If the QRS complexes for leads aVF and I are positive and negative respectively, the mean vector shows right axis deviation.

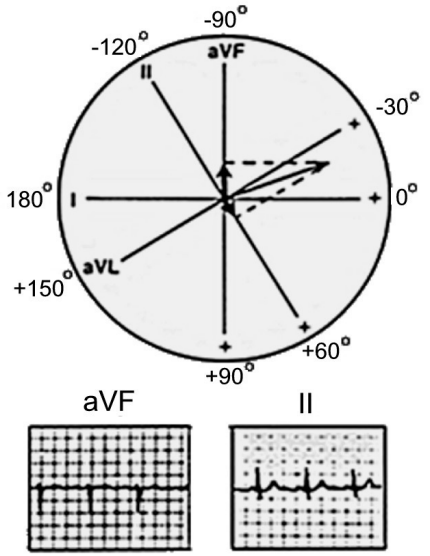

Figure 3-6. If the QRS complexes for leads aVF and II are negative and positive, respectively, the axis lies within the gray zone.

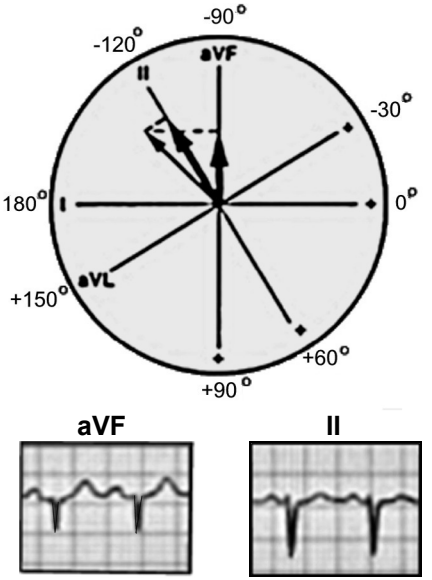

Figure 3-7. If the QRS complexes for leads aVF and II are both negative, there is left axis deviation.

Table 3-1. How to determine the axis of the mean vector.

CHECK LEAD aVF

- **If lead aVF is positive, check lead I**
  - **If lead I is positive, axis is in normal zone**
  - **If lead I is negative, there is right axis deviation**
- **If lead aVF is negative, check lead II**
  - **If lead II is positive, axis is in gray zone**
  - **If lead II is negative, there is left axis deviation**

Method Two

A second method for determining axis is to look for a limb lead in which the QRS complex is isoelectric (negative deflection = positive deflection). The axis of the mean vector must lie at an angle of 90 degrees to this lead. For example, if the QRS complex in lead II is isoelectric, the mean vector must lie in the direction of lead aVL. Problems inherent in this method are that an isoelectric lead may not be present and that the direction of the mean vector

is not specified; that is, whether it points in the positive or the negative direction. Other leads must be observed to determine this.

Chapter 4

Atrial Enlargement and Ventricular Hypertrophy

ATRIAL ENLARGEMENT

Atrial depolarization is reflected in the P wave of the electrocardiogram. The normal P wave is less than 2.5 mm high and 0.08 to 0.1 seconds in duration. Because lead V_1 is the closest to the atria, it gives the most accurate information about atrial enlargement. Lead II is also a useful lead to observe because it is approximately parallel to the forces of atrial depolarization.

Figure 4-1 illustrates P-wave configurations for normal atria, right atrial enlargement, and left atrial enlargement. The right atrium is responsible for the left-hand portion of the P wave and the left atrium for the right-hand portion. For practical purposes, the diagnosis of atrial enlargement can be made if either atrial component is prominent.

Right Atrial Enlargement

Criteria for *right atrial enlargement (RAE)* include:

- P is greater than 2.5 mm tall in II and/or greater than 1.5 mm in V_1
- P is biphasic in lead V_1 with initial portion greater in amplitude

The accuracy of RAE criteria is greatly improved in the pres-

ence of right ventricular hypertrophy.

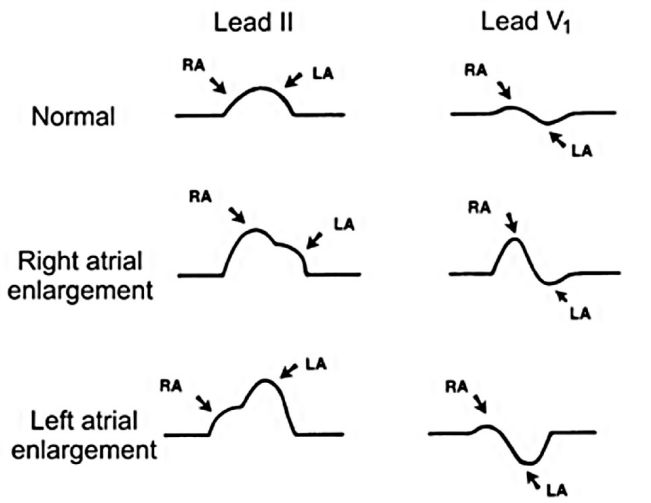

Figure 4-1. P-wave configurations from leads II and V_1 provide clues to atrial enlargement.

Right atrial enlargement may occur in a variety of clinical settings, including:

- Right ventricular enlargement
- Pulmonary disease (emphysema, chronic bronchitis)
- Heart disease (pulmonary stenosis, atrial septal defect, mitral stenosis or regurgitation, tetralogy of Fallot).

Because RAE is so frequently seen in chronic pulmonary disease, the peaked P wave is often called *P pulmonale*.

Left Atrial Enlargement

Criteria for *left atrial enlargement (LAE)* include:

- P is greater than 2.5 mm high in any lead
- P is double humped (notched) in any lead (humps at least one mm apart)
- Negative deflection of terminal portion of P in V_1

Left atrial enlargement occurs in a variety of clinical settings. It may be secondary to:

- Left ventricular enlargement (congestive heart failure, hypertensive heart disease, aortic stenosis or regurgitation)
- Mitral valve disease (stenosis, regurgitation).

Because LAE is so frequently seen with mitral valve disease, a broad notched P wave is often called *P mitrale.*

Biatrial Enlargement

Biatrial enlargement (BAE) presents with:

- Features of both RAE and LAE
- The P wave in lead II is greater than 2.5 mm tall and greater than or equal to 0.12 seconds in duration
- The initial positive component of the P wave in V_1 is greater than 1.5 mm tall and a prominent terminal P component is present

VENTRICULAR HYPERTROPHY

The electrocardiogram normally reflects left ventricular depolarization because left ventricular muscle mass is much greater than right ventricular mass. However, when right ventricular muscle mass becomes great enough, it causes alterations in the positivity of the right chest leads of the electrocardiogram that can be interpreted as right ventricular hypertrophy.

Right Ventricular Hypertrophy

In the absence of myocardial infarction or right bundle branch block, the diagnosis of *right ventricular hypertrophy* (RVH) can be made when:

- Right axial deviation is present and
- R is greater than S in lead V_1 ($R/S > 1$) or S is greater then R in lead V_6 ($R/S < 1$)

Other findings in RVH may include R greater than 7 mm high in V_1, taller R waves in the right precordial leads (V_1-V_3), and deeper S waves in the left precordial leads (V_4-V_6). The T wave may be inverted in V_1, and often in V_2. Signs or RAE may be present.

An example of RVH is given in Figure 4-2.

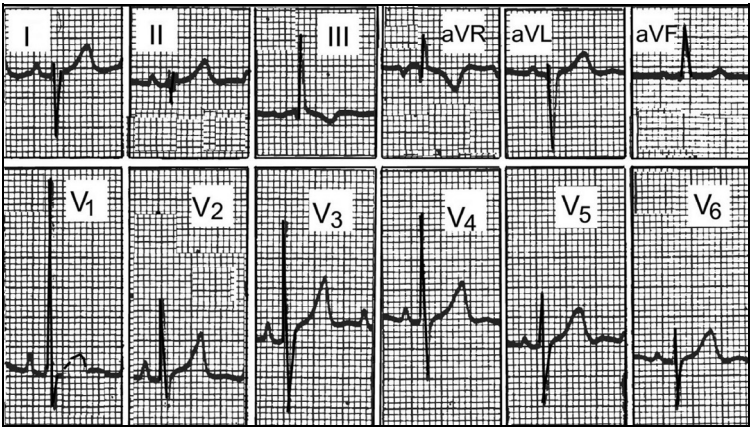

Figure 4-2. Right ventricular hypertrophy.

Right ventricular hypertrophy occurs in a variety of clinical conditions, including:

- Chronic pulmonary diseases (emphysema, chronic bronchitis, cystic fibrosis)
- Congenital heart disease (pulmonary stenosis, atrial septal defect, tetralogy of Fallot)
- Pulmonary embolus
- Pulmonary hypertension secondary to mitral stenosis
- Ventricular septal defect

Left Ventricular Hypertrophy

Left ventricular hypertrophy (LVH) causes an increase in the height and depth of the QRS complexes (Figure 4-3). Several criteria, based on chest leads or limb leads, are available for probable diagnosis of LVH. These criteria are more accurate for

individuals greater than or equal to 35 years of age. Four of these criteria are as follow:

- Sum of the S wave in V_1 or V_2 (whichever is larger) and the R wave in V_5 or V_6 (whichever is larger) is equal to 35 mm or more ($SV_{1-2} + RV_{5-6} \geq 35$ mm).
- Sum of the R wave in lead I and the S wave in lead III is equal to or greater than 25 mm (RI + SIII $\geq$ 25 mm).
- R wave in aVL is > 11 mm.
- R in aVL plus S in V_3 is greater than 28 mm in men or 20 mm in women [RaVL + SV_3 > 28(M) or 20(W)].

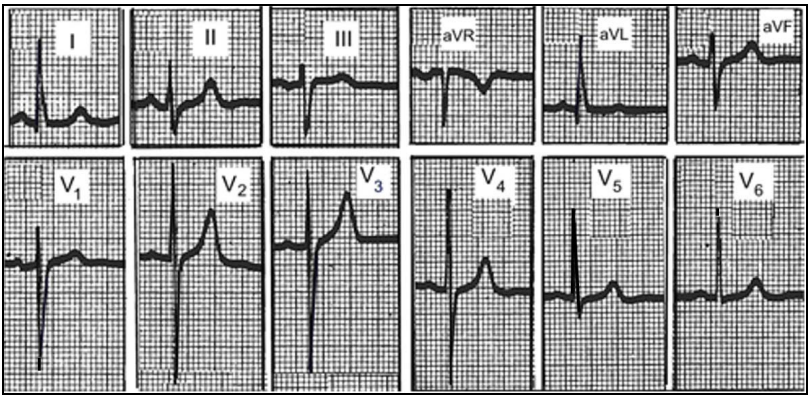

Figure 4-3. Left ventricular hypertrophy. Note that all four criteria are met.

The fact that one set of criteria has not universally been agreed upon means that the use of any lacks satisfactory accuracy. The diagnosis of LVH is best made by echocardiography.

Left axis deviation is often present with left ventricular hypertrophy but is of little significance and is not considered part of the criteria for diagnosis. Signs of LAE may be present.

The electrocardiograms of thin young men often meet the criteria for left ventricular hypertrophy without ventricular hypertrophy actually being present. Individuals with emphysema or pericardial effusion may have reduced QRS voltages and, hence, masked left ventricular hypertrophy.

The causes of left ventricular hypertrophy are numerous. The most common causes are:

- Chronic cocaine or methamphetamine use
- Hypertension
- Hypertrophic cardiomyopathy
- Valvular heart disease (aortic stenosis, aortic regurgitation, mitral regurgitation)

Ventricular Overload (Strain)

Ventricular hypertrophy may be the result of systolic or diastolic overload. Systolic overload is due to impairment of blood flow out of the ventricle, such as occurs with aortic stenosis, pulmonary stenosis, or hypertension. Diastolic overload is due to overfilling of the ventricle during diastole, such as occurs with aortic insufficiency or atrial septal defect.

Right Ventricular Systolic Overload

The chest leads of an electrocardiogram with *right ventricular systolic overload* are shown in Figure 4-4. In addition to the right ventricular hypertrophy criteria, T-wave inversion and usually ST segment depression are present in the right chest leads.

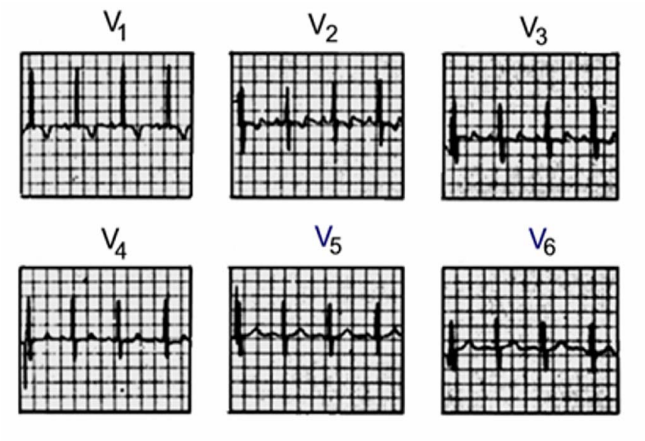

Figure 4-4. Right ventricular hypertrophy with strain.

Left Ventricular Systolic Overload

The diagnosis of LVH is made more accurately in the presence of *left ventricular systolic overload* as shown in Figure 4-5. In addition to the criteria for left ventricular hypertrophy already discussed, T-wave inversion and ST segment depression occur in the left chest leads.

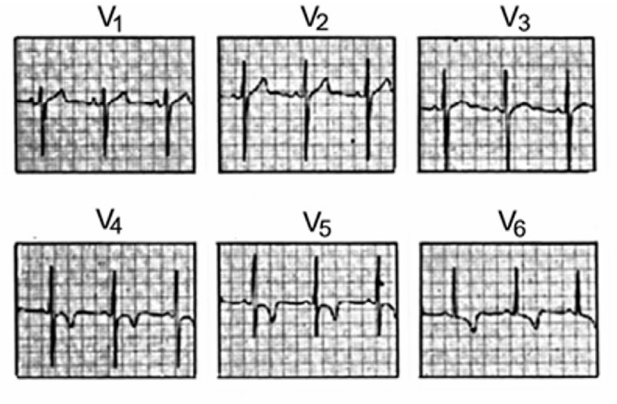

Figure 4-5. Left ventricular hypertrophy with strain.

Biventricular Hypertrophy

Biventricular Hypertrophy is a difficult EKG diagnosis to make. In the presence of LAE any one of the following suggests this diagnosis:

- R/S ratio in V_5 or $V_6 < 1$
- S in V_5 or $V_6 > 6$ mm
- RAD is present

Other suggestive EKG findings include:

- Criteria for LVH and RVH are both met
- LVH criteria is met and RAD or RAE is present

Chapter 5

Intraventricular Conduction Defects

In the normal process of ventricular depolarization, the electrical stimulus originates in the sinoatrial node, and then passes through the atria before reaching the atrioventricular node and junction. From there three pathways conduct the signal to the tissue of the ventricles: The *right bundle branch*, the *left anterior fascicle*, and the *left posterior fascicle*. The combined left anterior and left posterior fascicles are referred to as the *left bundle*.

Normally, the entire process of ventricular depolarization occurs in less than 0.1 seconds. Any process that interferes with normal depolarization of the ventricles may prolong the QRS width.

Bundle branch block occurs when electricity is not conducted down one or more of the conducting pathways in the ventricles. This may occur due to degeneration with age, or may be due to specific pathology, such as myocardial infarction.

Bundle branch block widens the QRS complex because electricity passes slower through cardiac muscle than through the special conducting pathways. The altered depolarization path may also cause the form or shape of the QRS complex to change.

RIGHT BUNDLE BRANCH BLOCK

Blockage of conduction in the right bundle branch results in delayed depolarization of the right ventricle. The sequence of ventricular depolarization in *right bundle branch block (RBBB)* is shown in Figure 5-1. Septal depolarization is normally initiated by

a branch of the left bundle branch and, hence, occurs from left to right, resulting in a small R wave in lead V_1. This is unchanged by RBBB. After septal depolarization, left ventricular depolarization takes place in a normal fashion, causing an S wave in lead V_1. Delayed depolarization of the right ventricle produces a second R wave in lead V_1 known as R′.

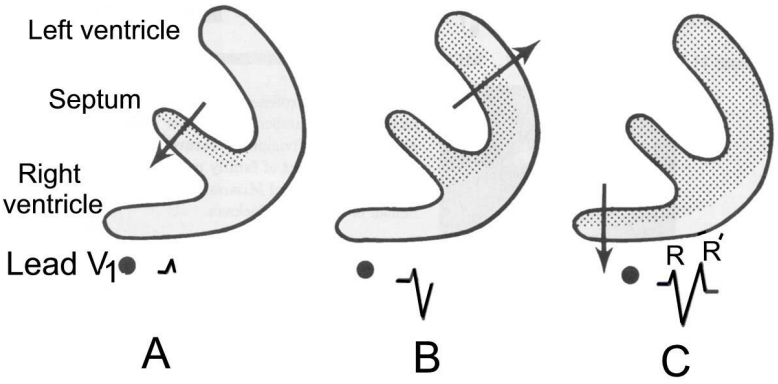

Figure 5-1. Sequence of ventricular depolarization in right bundle branch block. (A) Septal depolarization results in a small R wave in lead V_1. (B) Left ventricular depolarization results in an S wave. (C) Right ventricular depolarization produces a second R wave (R′).

The delayed depolarization of the right ventricle causes an increased width of the QRS complex to at least 0.12 seconds. Hence, RBBB is characterized by an R-R' configuration in lead V_1 or V_2 with a QRS complex equal to or greater than 0.12 seconds wide.

RBBB may be seen with:

- Atrial septal defect (especially ostium secundum)
- Cardiac surgery (especially in children)
- Congenital disorders (Brugada syndrome)
- Healthy people (common)
- Myocardial ischemia

Brugada syndrome is a congenital syndrome involving an abnormality of right bundle branch block with ST segment elevation

in the right chest leads. It is a rare cause of sudden death in otherwise healthy young men.

EKG changes of RBBB are shown in Figure 5-2.

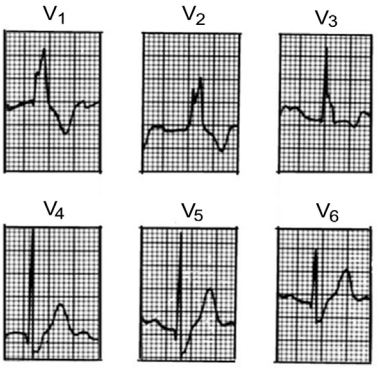

Figure 5-2. Electrocardiogram tracings typical of right bundle branch block. Note the R-R' configuration in the right chest leads and the wide QRS.

LEFT BUNDLE BRANCH BLOCK

Blockage of conduction in the left bundle branch, prior to its bifurcation into anterior and posterior fascicles, results primarily in delayed depolarization of the left ventricle. The sequence of ventricular depolarization with *left bundle branch block (LBBB)* is shown in Figure 5-3. The septum depolarizes from right to left

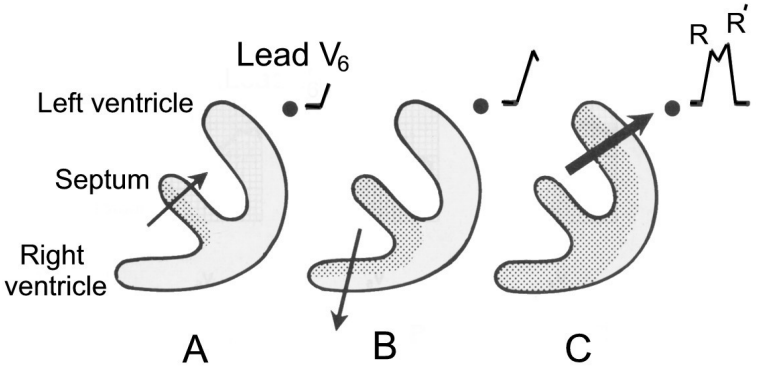

Figure 5-3. Ventricular depolarization in left bundle branch block results in an R-R' configuration in lead V_6. (A) Septal depolarization initiated by the right bundle branch occurs from right to left. (B) Right ventricular depolarization. (C) Left ventricular depolarization.

since its depolarization is now initiated by the right bundle branch. Next, the right ventricle depolarizes, followed by delayed depolarization of the left ventricle, giving an R-R' configuration in lead V_5 or V_6 and a QRS interval equal to or greater than 0.12 seconds.

Occasionally, right ventricular depolarization precedes septal depolarization, resulting in a small R wave in V_1. T waves in LBBB are usually oriented opposite the largest QRS deflection; that is, where large R waves are seen T waves will be inverted. ST segment depression may occur. An example of left bundle branch block is shown in Figure 5-4.

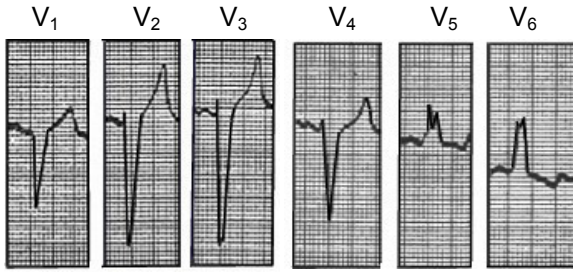

Figure 5-4. Electrocardiogram tracing typical of left bundle branch block. Note the and R-R' configuration in the lateral chest leads and the wide QRS.

Unlike RBBB, which is occasionally seen in normal subjects, LBBB is always a sign of organic heart disease. LBBB is seen in:

- Chronic degenerative changes in the ventricular conduction systems (elderly patients)
- Long-standing hypertension or valvular lesions, such as aortic stenosis or aortic regurgitation, that produce chronic strain on the left ventricle
- Coronary artery disease, since the blood supply to the conduction system arises from the coronary arteries
- Myocardial infarction, which is often a forerunner of complete heart block

INCOMPLETE BUNDLE BRANCH BLOCK

Incomplete right bundle branch block shows the same QRS pattern as RBBB; that is, an R-R' in lead V_1 or V_2. However, the QRS duration is between 0.1 and 0.12 seconds.

Incomplete left bundle branch block shows the same QRS pattern as LBBB; that is, an R-R' in lead V_5 or V_6. However, the QRS duration is between 0.1 and 0.12 seconds.

HEMIBLOCKS

The two major branches of the left bundle may be blocked individually. When only one branch is blocked, this is called *hemiblock* … either *left anterior hemiblock (LAH)* or *left posterior hemiblock (LPH)*, depending on whether the anterior or posterior fascicle is involved. The main effect of a hemiblock is to markedly change the QRS axis without changing the shape or duration of the QRS wave form.

Left anterior hemiblock results in left axis deviation (-30 degrees or more). S waves may be larger than R waves in II, III, and aVF.

Left posterior hemiblock results in right axis deviation (+90 degrees or more).

LAH is relatively common, while LPH is rare. In general, findings of isolated hemiblock are of little clinical significance. Hemiblock should only be considered after more common causes of axis deviation have been ruled out.

BIFASCICULAR BLOCK

Blockage of one of the subdivisions of the left bundle branch in the presence of RBBB can occur and is known as *bifasicular block*. With RBBB, the axis is usually within normal limits. The configuration of the QRS complex of RBBB is not altered by left anterior or left posterior hemiblock.

RBBB plus LAH produces an electrocardiographic picture of RBBB with left axis deviation. Similarly, *RBBB plus LPH* produces an electrocardiographic picture of RBBB with right axis deviation. These combinations are potentially significant since the presence of either means the ventricles are being depolarized via only one fascicle of the left bundle branch and subject to complete heart block.

NONSPECIFIC INTRAVENTRICULAR CONDUCTION DEFECTS

Nonspecific Intraventricular Conduction Defects (NIVCDs) have a QRS duration greater than 0.10 seconds, indicating slowed conduction in the ventricles. However, criteria for specific bundle branch or fascicular blocks are not met. Causes of nonspecific NIVCDs include:

- Drugs (especially class IA and IC antiarrhythmics such as quinidine)
- Hyperkalemia
- Myocardial infarction (so called *peri-infarction blocks*)
- Ventricular hypertrophy (especially LVH)

Chapter 6

Myocardial Ischemia, Myocardial Infarction, and Pseudoinfarction Syndromes

MYOCARDIAL ISCHEMIA

Myocardial ischemia is due to insufficient oxygen supply to the ventricular muscle. It may be transient, causing angina pectoris or more severe, causing the death of a portion of heart muscle (myocardial infarction). The following discussion refers to ischemia and infarction of the left ventricle since it is the predominant cardiac chamber.

Left ventricular muscle is divided into an outer layer (*epicardium*) and an inner layer (*subendocardium*) as shown in Figure 6-1. This distinction is important since ischemia is sometimes limited to the inner layer, and sometimes affects the entire thickness of the ventricular wall.

Subendocardial ischemia produces *classic angina* and *subendocardial myocardial infarction*. Transmural ischemia produces *Prinzmetal's angina* and *transmural myocardial infarction*.

Classic Angina

Angina pectoris is usually of the classic type; that is, it is brought on by exertion, stress, or exposure to cold. It is relieved by rest and nitroglycerin. Classic angina is thought to be due to transient subendocardial ischemia. The most common electrocardiographic finding with classic angina is transient ST segment de-

pression, except in lead aVR which may show a reciprocal ST segment elevation.

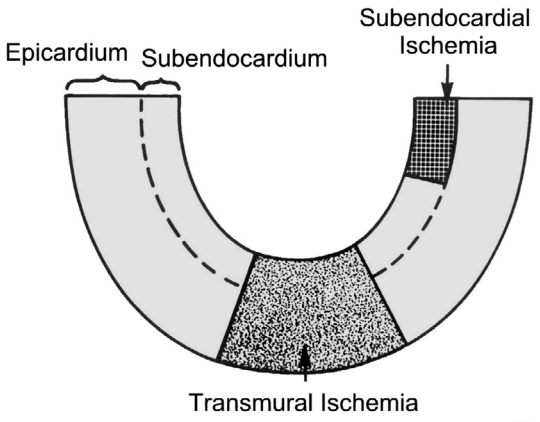

Figure 6-1. Ventricular muscle is divided into an outer layer (epicardium) and an inner layer (subendocardium). Ischemia may affect both layers or the subendocardium layer only.

Not all patients with coronary artery disease show ST segment depression during chest pain. Many patients with coronary artery disease have a normal electrocardiogram while at rest but develop ST segment depression with exertion because of the increased myocardial oxygen requirement. To assist in the diagnosis of coronary artery disease, the electrocardiogram can be recorded while the patient exercises under controlled conditions (*stress testing*).

Horizontal or down-sloping ST segments as seen in Figure 6-2 may be indicative of coronary heart disease. ST segment depression is considered significant if the ST segment is at least one small square below baseline, as measured two small squares after the end of the QRS complex (J point). The location of the ischemia is reflected in the leads in which the ST depression occurs.

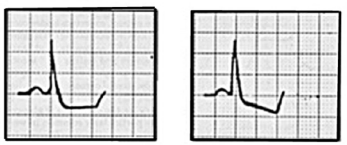

Figure 6-2. ST segment depression with classic angina. Depressions of at least one mm at 2 small squares past the J point are considered significant.

Chest pain may or may not occur and is not necessary for the diagnosis.

A more unpredictable or severe form of angina is defined as *unstable angina*. Chest pain may occur while resting or even sleeping (*nocturnal angina*), and the discomfort may last longer and be more intense than that of stable angina. Unstable angina may be a sign of impending myocardial infarction.

About 10 percent of normal men will have a false-positive test for angina. False-positive tests are particularly common in women. False-negative tests also occur.

Other Causes of ST Segment Depression

Other conditions to consider in the presence of ST segment depression include:

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

Prinzmetal's Angina

Prinzmetal's angina is atypical angina that occurs at rest or at night and results in ST segment elevation, as opposed to the ST segment depression of classic angina. The cause is thought to be transient transmural ischemia due to vasospasm. Prinzmetal's angina may occur in individuals with otherwise normal coronary arteries.

The height of the ST segment is measured at a point 2 squares after the end of the QRS complex (J point). ST segment elevation is considered significant if it exceeds one millimeter in a limb lead or two millimeters in a precordial lead.

Other Causes of ST Segment Elevation

Other conditions to consider in the presence of ST segment elevation include:

- Acute pericarditis or myocarditis
- Athletic heart syndrome
- Cardiomyopathy
- CNS events, such as subarachnoid hemorrhage
- Early repolarization
- Hyperkalemia
- Left ventricular aneurysm
- Reciprocal changes due to ischemia in other leads
- Transmural myocardial infarction
- Vasospasm due to cocaine or methamphetamine abuse

MYOCARDIAL INFARCTION

Myocardial infarction is diagnosed electrocardiographically by examining the QRS complexes, ST segments, and T waves. Combinations of abnormalities are usually required to make the diagnosis. Changes over time also may be required to firmly diagnose infarction.

Transmural Myocardial Infarction

With *transmural myocardial infarction*, the infarcted area remains in a depolarized (negative) state after the rest of the myocardium has repolarized. The loss of positivity in the infarcted area is responsible for the characteristic Q waves that develop in the leads exploring the infarcted area. However, it must be kept in mind that a normal electrocardiogram may exhibit small Q waves in leads I, aVR, V_5, and V_6 that represent only normal septal depolarization. Small Qs are also generally innocent in lead III and lead V_1 if no other abnormality is seen.

Q waves, to be considered diagnostic of acute myocardial infarction, must (1) have a duration of at least 0.04 seconds or (2) have a depth greater than 25 percent or more of the height of the R wave ($Q \geq 1/4R$).

Hyperacute T wave changes (increased T-wave amplitude and width) may occur early on with transmural myocardial infarction. The hyperacute T wave is noted as early as 30 minutes after the onset of transmural infarction. It tends to be a short-lived phenomenon that evolves rapidly to ST segment elevation. Q waves then follow within a few hours.

Other conditions to consider in the presence of Q waves include:

- Hypertrophic cardiomyopathy
- Left bundle branch block
- Left ventricular hypertrophy

ST segment elevations that occur after a myocardial infarction are seen in the leads that explore the infarcted area. Reciprocity (ST segment depressions) occur in other leads. Electrocardiographic diagnosis of acute myocardial infarction in the presence of left bundle branch block is extremely difficult.

The time sequence of a myocardial infarction can be divided into three stages, as shown in Figure 6-3: (A) acute phase, (B) evolving phase, and (C) resolving phase. In the *acute phase*, ST segment elevations may be the first sign of a myocardial infarction, generally appearing within a few minutes. Q waves may then appear in the leads showing the ST segment elevations. The T wave is incorporated into the ST segment elevation. The ST segment elevation may last for three to four days.

Figure 6-3. QRS and ST-T changes during three phases of transmural myocardial infarction: (A) acute, (B) evolving, and (C) resolving.

During the *evolving phase*, the ST segments begin returning toward the baseline and the T waves become inverted.

In the weeks to months that follow, during the *resolving phase*, the T waves again return to the upright position. In most cases, however, abnormal Q waves persist for months and even years. It is not unusual to see abnormal Q waves in a patient's electrocardiogram that suggest a previous infarction without a clinical history of a definite myocardial infarction, especially in the elderly. These are referred to as *silent infarctions*.

A normal electrocardiographic variant, *early repolarization*, often occurs in younger individuals and may be confused with myocardial infarction. With early repolarization, however, the T wave is distinct from the elevated ST segment, whereas with myocardial infarction it is incorporated into it. Early repolarization is discussed in Chapter 8.

Anterior Infarction

Anterior infarctions may be subdivided into (1) strictly anterior, (2) anteroseptal and (3) anterolateral infarctions. *Strictly anterior infarction* causes diagnostic changes in V_3 and V_4. *Anteroseptal infarction* results in loss of the normal small septal R waves in leads V_1 and V_2 as well as diagnostic changes in leads V_3 and V_4. *Anterolateral infarction* results in changes in more laterally situated chest leads (V_5, V_6) and the left lateral limb leads (I, aVL), as well as the anterior leads V_3 and V_4.

An anterior myocardial infarction is shown in Figure 6-4.

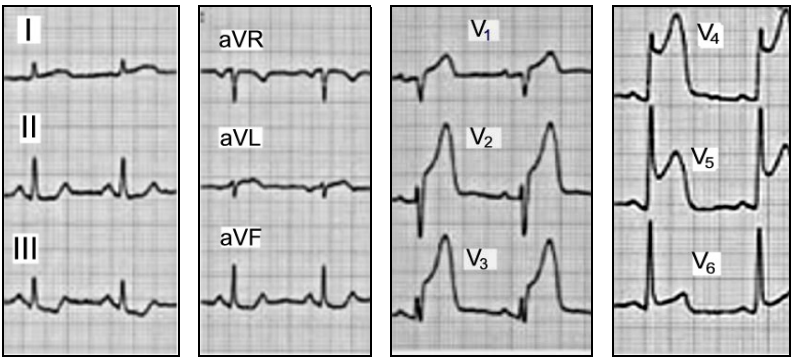

Figure 6-4. Electrocardiogram tracing typical of an anterior myocardial infarction. Note diagnostic Q waves and ST segment elevations.

Anterior infarction interrupts the normal R-wave progression in the chest leads, so R-wave progression should be noted when reading an electrocardiogram. However, poor R-wave progression is not specific for anterior myocardial infarction. Other conditions to consider in the presence of poor R-wave progression include:

- Cardiomyopathy
- Chest wall deformity
- Complete or incomplete LBBB
- Diffuse infiltrative cardiac disease
- Lead misplacement
- Left anterior fascicular block
- Left anterior hemiblock
- Normal variant
- Pulmonary disease (COPD, chronic asthma)
- Ventricular hypertrophy (LVH, RVH)
- Wolff-Parkinson-White syndrome

Inferior Infarction

Inferior infarction involves the diaphragmatic portion of the left ventricle and is indicated by changes in leads that explore the heart from below (II, III, aVF). An electrocardiogram tracing from someone with an inferior infarction is shown in Figure 6-5.

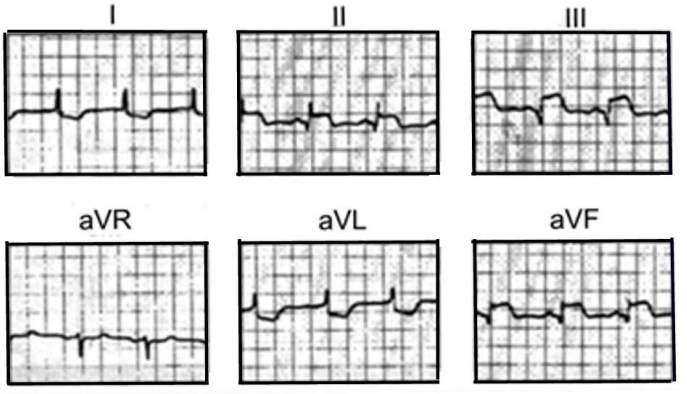

Figure 6-5. Electrocardiogram tracings typical of an inferior myocardial infarction. Note diagnostic changes in leads II, III, and aVF.

Posterior Infarction

Posterior infarction involves the posterior wall of the left ventricle. It does not generate Q-wave formation or ST segment elevation in the conventional 12-lead electrocardiogram since there are no posterior exploring electrodes. Instead, the EKG changes of posterior infarction are reciprocal changes; that is, they are seen up side down on the front of the heart in lead V_1 (Q waves become big R waves, ST elevation is seen as depression, T-wave inversion is seen as an upright T).

Therefore, in posterior infarction, the R waves in V_1 and V_2 become taller than the S waves (R/S > 1**).** Unlike right ventricular hypertrophy, right axis deviation is not present. ST segment depression may occur in these leads (Figure 6-6).

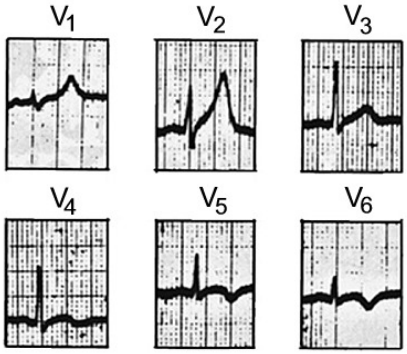

Figure 6-6. Electrocardiogram tracings typical of a posterior myocardial infarction. Note diagnostic changes in leads V_1 and V_2.

Other conditions to consider in the presence of tall R waves in V_1 include:

- Dextrocardia (rare)
- Duchenne muscular dystrophy (rare)
- Right bundle branch block
- Right ventricular hypertrophy
- Some normal children and young adults
- Wolff-Parkinson-White syndrome (with left ventricular insertion of the accessory pathway)

Subendocardial Infarction

Subendocardial infarction affects only repolarization (ST-T complex) and not depolarization (QRS complex). Hence, Q waves are not characteristic of subendocardial infarction. When subendocardial infarction occurs, the electrocardiogram may show persistent ST segment depression (Figure 6-7) instead of the transient depression seen with classic angina. Persistent T-wave inversion without ST segment depression may also occur. The ST-T changes slowly return to normal as the infarction resolves.

Electrocardiographic findings must be combined with the clinical circumstance and cardiac enzymes to make a diagnosis of subendocardial infarction.

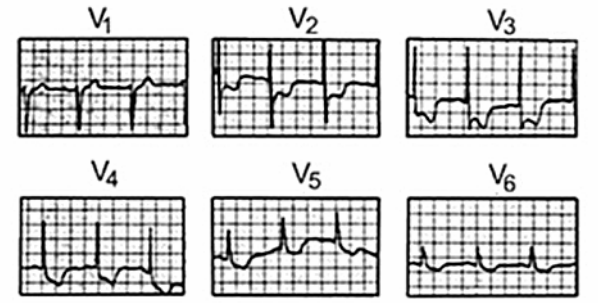

Figure 6-7. Electrocardiogram tracing typical of subendocardial myocardial infarction. Note the ST-T changes.

PSEUDOINFARCTION SYNDROMES

Several specific pathological conditions may present electrocardiographic findings similar to those of myocardial infarction. They are called *pseudoinfarction syndromes*. Pseudoinfarction syndromes include:

- Complete or incomplete LBBB (QS waves or poor R-wave progression in leads V_{1-3})
- Dramatic alterations of ST segments and T waves may occur with increased intracranial pressure due to changes in repolarization that result from enhanced sympathetic nervous system activity.
- Hyperkalemia (ST segment elevation and peaked T waves).

- LAH (may see small Q waves in anterior chest leads)
- Left ventricular aneurysm after extensive infarction may show persistent ST segment elevation
- LVH (may have QS pattern or poor R-wave progression in leads V_{1-3})
- Patients with hypertrophic cardiomyopathy may have significant Q waves on their electrocardiograms due to distortion of the normal pattern of depolarization because of the asymmetrical, hypertrophied ventricular muscle.
- Pericarditis may show ST segment elevation and subsequent T-wave inversion. However, with pericarditis there is no Q-wave formation.
- Pneumothorax (loss of right precordial R waves)
- Pulmonary emphysema and cor pulmonale may show loss of R waves in V_{1-3} and/or inferior Q waves with right axis deviation.
- RVH (tall R waves in V_1 or V_2 may mimic true posterior myocardial infarction).

Chapter 7

Rhythm Disturbances

Rhythm disturbances may involve the sinoatrial node, the atria, the atrioventricular junction, and the ventricles. They can be categorized as follow:

- **Regular.** R-R interval constant (except for minor variation with respiration)
- **Basically regular.** Regular except for occasional premature beats or escape beats
- **Regularly irregular.** R-R interval variable but with a definite pattern (normal beats and ectopic beats grouped together and repeating over and over)
- **Irregularly irregular.** R-R interval variable with no pattern (totally irregular)

NORMAL SINUS RHYTHM

Sinus rhythm originates in the *sinoatrial node*. Diagnosing sinus rhythms requires examining leads II and aVR for the correct polarity of the P waves. The P wave is normally positive in lead II and negative in lead aVR. A P wave should precede each QRS complex and the P-R interval should be relatively constant.

Normal sinus rhythm (NSR) is defined a sinus rhythm with a heart rate between 60 and 100 bpm (Figure 7-1). The rate may vary slightly in a phasic manner due to respiration, increasing with inspiration and decreasing with expiration.

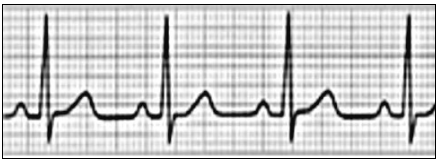

Figure 7-1. Normal sinus rhythm with a rate of about 75 bpm.

SINUS ARRHYTHMIAS

Occasionally a variable sinus rhythm is seen in which the heart rate varies independent of the respiratory cycle or varies greater than 10 percent with the respiratory cycle. Such a rhythm is referred to as *sinus arrhythmia*.

Sinus Tachycardia

Sinus tachycardia is a sinus rhythm with a heart rate greater than 100 bpm (Figure 7-2). With sinus tachycardia at very fast rates, the P waves may merge with the preceding T waves and be indistinct. For this reason, tachycardia originating above the ventricles (sinoatrial node, atrial muscle, atrioventricular junction) is often referred to as *supraventricular tachycardia* without specifying its site of origin.

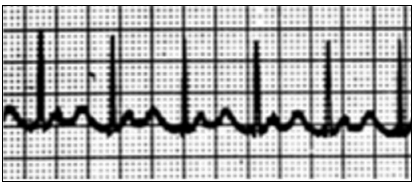

Figure 7-2. Sinus tachycardia with a rate of about 140 bpm.

Sinus tachycardia occurs for many reasons, including:

- Anxiety
- Congestive heart failure
- Hyperthyroidism
- Hypotension
- Physical activity
- Sympathomimetic drugs

Sinus Bradycardia

Sinus bradycardia is a sinus rhythm with a heart rate less than 60 bpm (Figure 7-3).

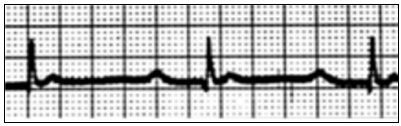

Figure 7-3. Sinus bradycardia with a rate of 50-55 bpm.

Sinus bradycardia occurs for many reasons, including:

- Drugs that increase vagal tone (digitalis)
- Drugs that decrease sympathetic tone (beta-blockers)
- Hypothyroidism
- Physical conditioning. World-class marathon runners may have resting heart rates as low as 35 bpm.

Sinoatrial Block

Sinoatrial block refers to failure of the sinus node to function for one or more beats. In this condition there is simply one or more missing beats; that is, no P waves or QRS complexes are seen (Figure 7-4). Fortunately, when the sinus fails to function for a

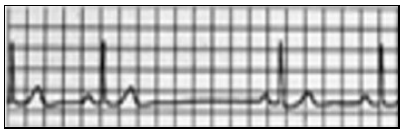

Figure 7-4. Sinoatrial block. Note the missing beat.

significant period of time (*sinus arrest*), another part of the conduction system usually assumes the role of pacemaker. These pacing beats are referred to as *escape beats* and may come from the atrial muscle, the atrioventricular junction, or the ventricles. When the sinoatrial node fails to function and other areas of the heart do not take over the pacemaker role, *asystole* occurs and the electrocardiogram shows only a straight-line pattern.

Sinoatrial block can occur for a variety of reasons, including:

- Digitalis toxicity
- Hyperkalemia
- Hypoxemia

Sick Sinus Syndrome

In elderly people the sinus node may undergo degenerative changes and fail to function effectively. Periods of sinus arrest, sinus tachycardia, or sinus bradycardia may occur, leading to episodes of lightheadedness or even syncope. This has been termed *sick sinus syndrome* (Figure 7-5). Treatment of symptomatic individuals may require a permanent pacemaker to control the periods of bradycardia. This is combined with drugs such as digitalis or a beta-blocker to control the periods of tachycardia.

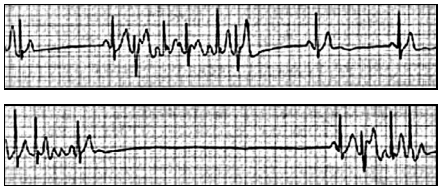

Figure 7-5. Sick sinus syndrome.

NON-SINUS ATRIAL ARRHYTHMIAS

Non-sinus atrial arrhythmias include premature atrial beats, paroxysmal atrial tachycardia, multifocal atrial tachycardia, atrial flutter, and atrial fibrillation. Because the stimuli arise above the level of the ventricles, the QRS pattern is usually normal.

Premature Atrial Contraction

A *premature atrial contraction (PAC)* is an ectopic beat arising from somewhere in either atrium, but not in the sinoatrial node. Premature atrial contractions occur before the next normal beat is due, and a slight pause usually follows the contraction (Figure 7-6).

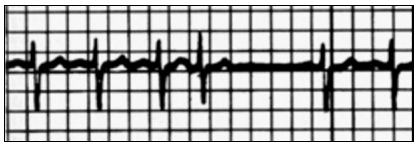

Figure 7-6. Premature atrial contraction. Note the pause following the PAC.

The P wave of the premature beat may have a configuration

different than the normal P wave. It may even be of opposite polarity if it arises low in the atria, causing the atrial depolarization wave to travel backwards toward the sinoatrial node. Occasionally the P wave will not be seen because it is lost in the preceding T wave. The P-R interval of the premature atrial contraction may be shorter than the normal P-R interval.

If the premature atrial depolarization wave reaches the atrioventricular node before the node has had time to repolarize, the impulse may not be conducted. In this case, a premature, abnormal P wave will be seen without a subsequent QRS complex.

Premature atrial beats are sometimes conducted to ventricular tissue during the process of ventricular repolarization. In such cases, the subsequent ventricular depolarization may take place by an abnormal pathway, generating a wide, bizarre QRS complex. This is referred to as *aberrant ventricular depolarization*, and is discussed later in this chapter.

Paroxysmal Atrial Tachycardia

Paroxysmal atrial tachycardia (PAT) is defined as three or more consecutive premature atrial contractions. In some cases, the run of paroxysmal atrial tachycardia may be brief and self-limited. In other cases, it may be sustained for hours or even days and require termination by intervention. Paroxysmal atrial tachycardia occurs at a regular rate, usually between 150 and 250 bpm (Figure 7-7).

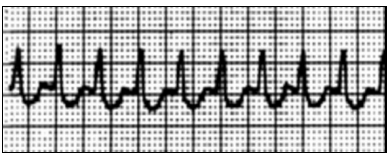

Figure 7-7. Paroxysmal atrial tachycardia with a rate of about 225 bpm.

As with sinus tachycardia, P waves may or may not be seen, and it may be difficult to differentiate paroxysmal atrial tachycardia from sinus tachycardia. Paroxysmal atrial tachycardia may occur in normal individuals as well as in those with organic heart disease.

Multifocal Atrial Tachycardia

Multifocal atrial tachycardia (MFAT) results from the presence of multiple, different atrial pacemaker foci. This rhythm disturbance is characterized by a tachycardia with beat-to-beat variations of the P-wave morphology (Figure 7-8). Multifocal atrial tachycardia most commonly occurs in individuals with chronic pulmonary disease.

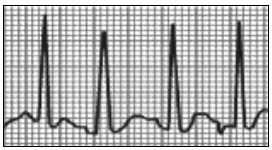

Figure 7-8. Multifocal atrial tachycardia. Note the varying P-wave morphology.

Atrial Flutter

Atrial flutter is also an ectopic atrial rhythm. Instead of P waves, characteristic sawtooth waves are seen. The atrial rate in atrial flutter is usually about 300 bpm. However, the atrioventricular junction is unable to conduct at this rapid rate so the ventricular rate is less, usually 150 bpm, 100 bpm, 75 bpm, and so on.

Atrial flutter with a ventricular rate of 150 bpm is called two-to-one flutter because of the ratio of the atrial rate to the ventricular rate (300 bpm/150 bpm = 2/1); a ventricular rate of 100 bpm is three-to-one flutter (300 bpm/100 bpm = 3/1), and a ventricular rate of 75 bpm is a four-to-one flutter (300 bpm/75 bpm = 4/1). The ventricular rate may abruptly change from one rate to another as the degree of block at the atrioventricular junction changes.

Atrial flutter can occur with:

- Alcohol abuse
- COPD
- Mitral valve disease
- Myocardial infarction
- Pulmonary embolism
- Thyrotoxicosis

The sawtooth pattern of atrial flutter is best seen in the inferior leads (II, III, aVF). An example of atrial flutter is shown in Figure 7-9. Although atrial flutter is not specific for any particular type of heart disease, it is rarely seen in individuals with normal hearts.

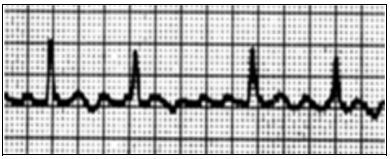

Figure 7-9. Atrial flutter with abrupt change from 3:1 flutter to 5:1 and back again.

Atrial Fibrillation

In *atrial fibrillation (AF)* the atria are depolarized at an extremely rapid rate, greater than 400 bpm. This fibrillatory activity produces a characteristic irregular, wavy base-line pattern instead of normal P waves. Because the atrioventricular junction is refractory to most of the impulses reaching it, it allows only a fraction of them to pass through to the ventricles. The ventricular rate is, therefore, only 110 to 180 bpm.

Also characteristic of atrial fibrillation is a haphazardly irregular ventricular rhythm that results from the random stimulation of the atrioventricular junction (variable R-R intervals). An example of atrial fibrillation is shown in Figure 7-10.

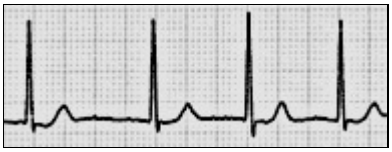

Figure 7-10. Atrial fibrillation. Note the variable R-R intervals and lack of P waves.

Atrial fibrillation may occur paroxysmally, lasting only a few minutes, hours, or days, or it may be chronic and persist for years. Atrial fibrillation may occur in normal individuals and in patients with a variety of cardiac diseases. Common causes of atrial fibrillation are:

- Acute alcohol intoxication (holiday heart, Saturday night heart)
- Acute pulmonary processes
- Congestive heart failure
- Coronary artery disease
- Hypertensive heart disease
- Hyperthyroidism
- Narcotic abuse
- Valvular heart disease

JUNCTIONAL RHYTHMS

Junctional rhythms arise in the atrioventricular junction. P waves, when seen, are opposite their normal polarity; that is, negative in lead II and positive in lead aVR. These are called *retrograde P waves* because they result from the atrial depolarization wave traveling backward from the atrioventricular junction through the atria toward the sinoatrial node. Retrograde P waves may precede, be buried in, or follow the QRS complex. Since the stimulus arises above the level of the ventricles, the QRS complex is usually of normal configuration.

Premature Junctional Contractions

Since the atrioventricular junction also may serve as an ectopic pacemaker, premature junctional beats can occur. *Premature junctional contractions* are similar to premature atrial contractions in that they occur before the next beat is due and a slight pause follows the premature beat (Figure 7-11).

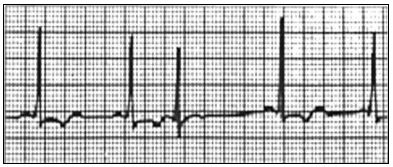

Figure 7-11. Premature junctional contraction. Note the pause after the premature beat.

Junctional Tachycardia

Atrioventricular *junctional tachycardia* is simply a run of three or more premature junctional beats (Figure 7-12). Junctional tachycardia has about the same rate as paroxysmal atrial tachycardia and often cannot be distinguished from it. The difference is not clinically significant.

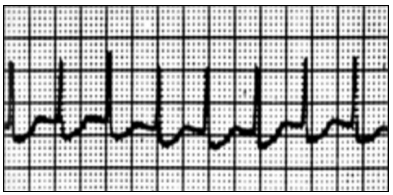

Figure 7-12. Junctional tachycardia at a rate of about 150 bpm.

Junctional Escape Rhythm

An atrioventricular *junctional escape beat* is an escape beat that occurs after a pause in the normal sinus rhythm. Atrial pacing usually resumes after the junctional beat. A *junctional escape rhythm*, defined as a consecutive run of atrioventricular junctional beats, may develop if the sinoatrial node does not resume the pacemaker role. Junctional escape rhythm has a rate between 40 and 60 bpm (Figure 7-13).

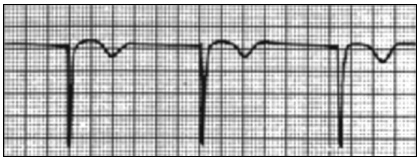

Figure 7-13. Junctional escape rhythm at a rate of 50 bpm.

Atrioventricular junctional escape rhythms may be due to several causes, including:

- Acute myocardial infarction
- Digitalis toxicity
- Hyperkalemia
- Hypoxia

VENTRICULAR RHYTHM DISTURBANCES

Ventricular tissue is capable of spontaneous depolarization. Because the depolarization wave arises in the myocardium, it does not follow the normal path of ventricular depolarization. Therefore, the QRS complex is prolonged and bizarre in shape.

In addition to premature ventricular contractions, ectopic ventricular beats produce ventricular tachycardia and sometimes ventricular fibrillation. Ventricular escape rhythms also occur.

Premature Ventricular Contractions

Premature ventricular contractions (PVCs) are premature beats arising in the ventricles. They are analogous to premature atrial contractions and premature junctional contractions. Premature ventricular contractions have two major characteristics:

- They are premature and arise before the next normal beat is expected (a P wave is not seen before a premature ventricular contraction).
- They are aberrant in appearance. The QRS complex is always abnormally wide, usually greater than 0.12 seconds.

The T wave and the QRS complex usually point in the opposite direction. As with a premature atrial contraction or a premature junctional contraction, a premature ventricular contraction is usually followed by a compensatory pause before the supraventricular mechanism resumes control (Figure 7-14).

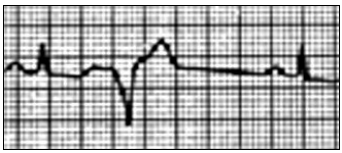

Figure 7-14. Premature ventricular contraction. Note the pause after the PVC.

PVCs may be unifocal or multifocal. *Unifocal PVCs* arise from the same ventricular site and, as a result, have the same appearance in a given electrocardiogram. *Multifocal PVCs*, on the other hand, arise from different ventricular foci and have different QRS patterns.

PVCs can be seen with virtually any kind of heart disease, particularly after an acute myocardial infarction where they may be the forerunner of ventricular tachycardia and even ventricular fibrillation.

Should a PVC fall on a T wave, serious dysrhythmias can occur, since the T wave represents repolarization of the ventricle. If a PVC should occur when the ventricle is only partially repolarized, ventricular tachycardia can occur and can then progress to ventricular fibrillation, which is fatal if left untreated.

Benign PVCs are common to all age groups. Benign premature ventricular contractions are due to:

- Anxiety
- Drugs (epinephrine, aminophylline, digitalis)
- Excessive caffeine use

Ventricular Tachycardia

Ventricular tachycardia (*V-tach)* is defined as a run of three or more premature ventricular contractions (Figure 7-15). It may occur as a single isolated burst or paroxysmally, or it may persist until stopped by intervention. The heart rate is usually 160 to 240 bpm. Ventricular tachycardia is a life-threatening arrhythmia because many patients are not able to maintain an adequate blood pressure with the rapid rate and because it often degenerates into ventricular fibrillation. The most common cause of V-tach is coronary artery disease. Other triggers include ischemia, electrolyte abnormalities, and stimulant abuse (cocaine, methamphetamine).

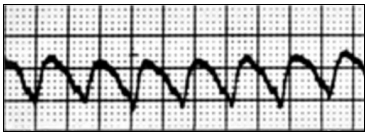

Figure 7-15. Ventricular tachycardia at a rate of about 225 bpm.

Torsades de Pointes

Torsades de pointes (Figure 7-16) is a medical condition, the name of which means in French "twisting of the points." It is a potentially deadly form of ventricular tachycardia. On the electrocardiogram it presents like ventricular tachycardia, but the QRS complexes swing up and down around the baseline in a chaotic fashion, which prompted the name.

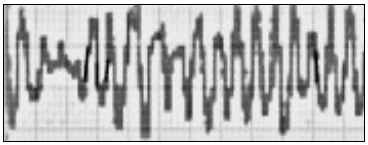

Figure 7-16. Torsades de pointes.

Factors associated with increased risk for medication-induced torsades de pontes include congenital and acquired factors that prolong the Q-T interval. These include:

- Cardiac abnormalities (bradycardia, recent atrial defibrillation, congestive heart failure)
- Endocrine factors
- Female sex (Q-T interval is naturally longer in females)
- Electrolytes (hypokalemia, hypomagnesemia such as occurs in alcoholics)

- Hypoxia/acidosis
- Intracranial factors
- Nutritional disorders
- Organophosphate pesticide toxicity

Some medications known to cause torsades de pointes in susceptible individuals are listed in Table 7-1.

Table 7-1. Some medications that can prolong the Q-T interval.

More Commonly		Less Commonly
Amiodarone	Droperidol	Pentamidine
Arsenic trioxide	Erythromycin	Pimozide
Bepridil	Halofantrine	Procainamide
Chlorpromazine	Haloperidol	Quinidine
Cisapride	Ibutilide	Sotalol
Clarithromycin	Levomethadyl	Sparfloxacin
Disopyramide	Lidoflazine	Thioridazine
Dofetilide	Mesoridazine	
Domperidone	Methadone	

Ventricular Fibrillation

In *ventricular fibrillation (V-fib)* the ventricles do not beat in a coordinated fashion, but instead twitch asynchronously. Ventricular fibrillation is sometimes divided into coarse and fine rhythms. V-fib is a potentially deadly arrhythmia because there is no cardiac output. An illustration of ventricular fibrillation is found in Figure 7-17.

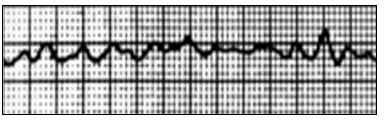

Figure 7-17. Ventricular fibrillation.

Idioventricular Rhythm

Since ventricular tissue is capable of spontaneous depolarization at an intrinsically slower rate than other areas of the heart, a ventricular focus may initiate depolarization when a faster pace-

maker does not control the rate. Like junctional escape beats, ventricular escape beats occur after a pause in the regular rhythm. If a higher focus fails to pick up the rhythm, ventricular escape beats may continue. When this occurs, the rhythm is called *idioventricular rhythm* and has a rate usually less than 100 bpm. The QRS complex is wide and bizarre. P waves will, of course, not be present. Idioventricular rhythms are usually of short duration and as a rule require no intervention. An example of idioventricular rhythm is shown in Figure 7-18.

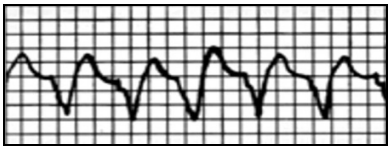

Figure 7-18. Idioventricular rhythm with a rate of about 80 bpm.

Supraventricular Beat with Aberrancy

There are two possible etiologies for a wide and premature ventricular depolarization. The first etiology, premature ventricular contraction, has already been discussed. The second etiology is referred to as *aberrant ventricular depolarization*. In this instance, the depolarization wave is initiated above the ventricular level and, because it is premature, reaches the ventricles when they are still in a partially depolarized state, resulting in a wide QRS complex. The following rules can be used to help differentiate aberrant ventricular depolarization from premature ventricular contraction:

1. The beat is aberrant if a P wave precedes the wide QRS complex.
2. The preceding R-R interval is usually longer than the other ones.
3. Most aberrant beats are conducted via the left bundle branch, giving the appearance of right bundle branch block in lead V_1.
4. The initial deflection of the wide QRS is in the same direction as that of the normal QRS complex.

An example of a supraventricular beat with aberrancy is shown in Figure 7-19

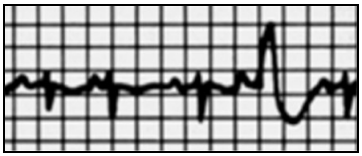

Figure 7-19. Supraventricular beat with aberrancy. Note the P wave preceding the QRS complex.

ATRIOVENTRICULAR HEART BLOCK

Heart block occurs in three forms … first degree, second degree, and third degree. Second-degree heart block is divided into two types: Mobitz type 1 and Mobitz type 2. There are numerous causes of heart block, and many factors can produce any of the three degrees. Some of these causes are:

- Acute myocardial infarction, particularly inferior infarctions because the blood supply to the inferior wall and the atrioventricular junction arises from the same source
- Digitalis
- Ischemic heart disease

First-degree Heart Block

The electrocardiographic abnormality of *first-degree heart block* is simply a prolonged P-R interval to greater than 0.2 seconds (Figure 7-20). In addition to the factors already mentioned, hyperkalemia may cause first-degree heart block. First-degree heart block may also be present in normal individuals.

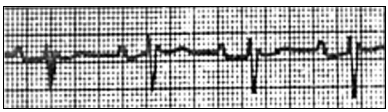

Figure 7-20. First-degree atrioventricular block. Note the prolonged P-R interval.

Second-degree Heart Block

There are two types of second-degree atrioventricular block … Mobitz type 1 and Mobitz type 2. When the P-R interval becomes progressively longer until a QRS complex is dropped and then the process repeats, this is a second degree atrioventricular block known as *Mobitz* type I block or *Wenckebach* phenomenon (Figure 7-21).

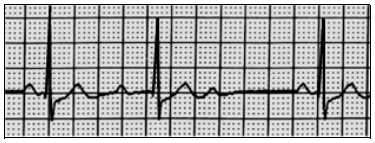

Figure 7-21. Second-degree A-V block, Mobitz type 1. Note the progressively increasing P-R interval until a QRS is dropped.

If the QRS complex is periodically dropped without lengthening of the P-R interval, this is called a *Mobitz type 2 block*. A dropped beat is seen as a P wave that is not followed by a QRS complex. Mobitz type 2 heart block is a more severe form of second-degree block since it often progresses to complete heart block.

The characteristic electrocardiogram picture of Mobitz type 2 is that of a series of non-conducted P waves. For example, with a two-to-one block, every other P wave is conducted; with a three-to-one block, every third P wave is conducted; and with a four-to-one block, every fourth P wave is conducted. An example of Mobitz type 2 block is shown in Figure 7-22.

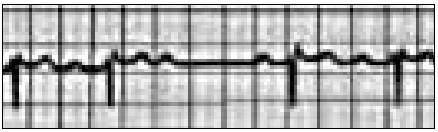

Figure 7-22. Second-degree A-V block, Mobitz type 2. Note the P wave that is not followed by a QRS complex.

Third-degree Heart Block

Third-degree heart block is also referred to as *complete heart block* because the atrioventricular junction does not conduct any stimuli from the atria to the ventricles. Instead, the atria and ventricles are paced independently. The characteristic electrocardiogram picture of complete heart block is:

- P waves are present and occur at a faster rate than the ventricular rate.
- QRS complexes are present and occur at a regular rate, usually less than 60 bpm.
- The P waves bear no relationship to the QRS complexes. Thus, the P-R intervals are completely variable.

With third-degree heart block, the QRS complex may be of normal or abnormal width, depending on the location of the blockage in the atrioventricular junction. If the blockage is in the first

part of the junction (atrioventricular node), the QRS complex will be of normal width. If the blockage is lower in the junction (bundle of His), the ventricles will be paced by an idioventricular pacemaker and the QRS complexes will be abnormally wide. An example of third-degree heart block is shown in Figure 7-23.

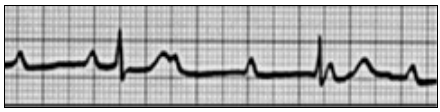

Figure 7-23. Third-degree (complete) heart block.

Complete heart block resulting from blockage of the right bundle branch and both fascicles of the left bundle branch is known as *trifascicular block*.

Complete heart block is commonly seen in older individuals who have degenerative changes in their conduction system. Fainting attacks from complete heart block is known as *Stokes-Adams syndrome*.

BIGEMINY

Bigeminy is an alternating pattern of combinations of atrial-atrial, atrial-ventricular, or ventricular-ventricular beats. Trigeminy and even *quadrigeminy* also can occur. Figure 7-24 shows bigeminy consisting of a series of atrial beats, each followed by a ventricular beat (PVC).

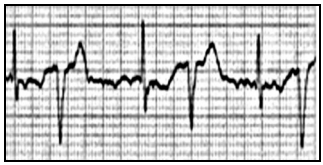

Figure 7-24. Bigeminy (couplets of an atrial beat followed by a PVC).

Chapter 8

Preexcitation Syndromes, Early Repolarization, Pulmonary Embolus, and Pericarditis

As we saw in Chapter 1, normally the depolarization wave is initiated by the sinoatrial node. From there it passes down internodal fibers to the atrioventricular node, through the bundle of His, and down the right and left bundle branches. It should be remembered that this process normally requires 0.12 to 0.20 seconds, mostly because of slow passage in the atrioventricular node.

PREEXCITATION SYNDROMES

Preexcitation syndromes refer to clinical conditions in which the wave of depolarization initially bypasses the atrioventricular node as it passes from the atria to the ventricles. Because conduction through an accessory pathway is faster than that through the atrioventricular node, the time required for the wave to leave the sinoatrial node and arrive at ventricular muscle (P-R interval) is shortened.

Two important preexcitation syndromes will be discussed: The Wolff-Parkinson-White syndrome and the Lown-Ganong-Levine syndrome.

Wolff-Parkinson-White Syndrome

Patients with *Wolff-Parkinson-White (WPW) syndrome* possess an accessory pathway of depolarization known as the *bundle*

of Kent (Figure 8-1). The bundle of Kent originates in atrial tissue and ends in ventricular tissue. It is capable of rapidly conducting the depolarization wave, partially bypassing the delay in the atrioventricular node.

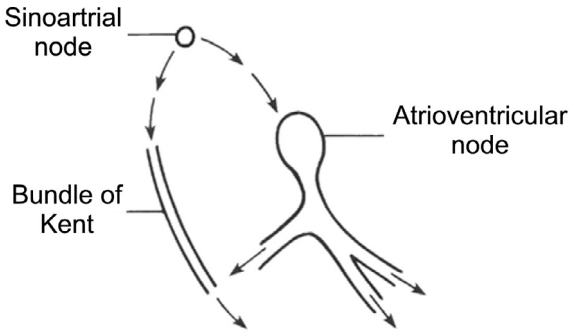

Figure 8-1. Accessory pathway of Wolff-Parkinson-White syndrome (bundle of Kent).

With Wolff-Parkinson-White syndrome, as atrial depolarization is being completed, the depolarization wave arrives simultaneously at the atrioventricular node and the atrial end of the bundle of Kent. Conduction through the atrioventricular node is delayed, but conduction passes rapidly through the bundle of Kent. After the initial focus of ventricular depolarization via the bundle of Kent has occurred, the remainder of the ventricular muscle is depolarized via the wave from the atrioventricular bundle. The net result is a QRS complex that is a composite of the initial premature ventricular depolarization and the later depolarization of the remaining myocardium via the normal conducting system. The early depolarization produces a slurring of the initial portion of the QRS complex called a *delta wave* as illustrated in Figure 8-2.

The three electrocardiographic criteria for Wolff-Parkinson-White syndrome are:

- A short P-R interval (0.12 seconds or less)
- A wide QRS complex
- A delta wave (the QRS complex is widened by the delta wave exactly the same amount as the P-R interval is shortened)

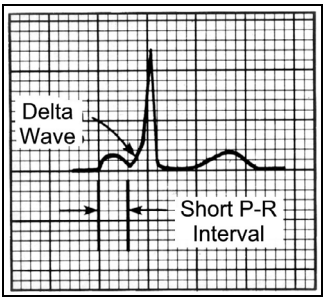

Figure 8-2. Electrocardiographic illustration of the Wolff-Parkinson-White syndrome.

The major clinical manifestation of Wolff-Parkinson-White syndrome is recurrent tachycardia due to the fact that the bundle of Kent allows the establishment of a continuous reentry cycle (Figure 8-3). The QRS complex may be normal or wide and bizarre, depending on the direction of the reentry wave. If the atrioventricular node is activated in an antegrade fashion (normal direction) and the bundle of Kent is activated retrograde, a normal QRS complex results. However, if the bundle of Kent is depolarized in an antegrade fashion, with retrograde depolarization of the atrioventricular node, a wide, bizarre QRS complex results.

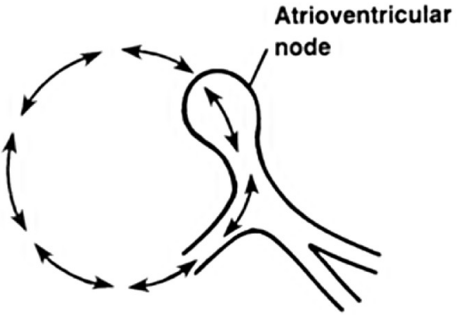

Figure 8-3. Reentry cycle of Wolff-Parkinson-White Syndrome. The cycle may be antegrade or retrograde through the atrioventricular node.

Lown-Ganong-Levine Syndrome

The *Lown-Ganong-Levine* syndrome is the result of some of the internodal fibers, called *James fibers*, bypassing the major portion of the atrioventricular node and terminating in the bundle of His (Figure 8-4), as opposed to the ventricle muscle as in the WPW syndrome.

The major conduction delay in the atrioventricular node is partially bypassed, resulting in a short P-R interval of 0.12 seconds or

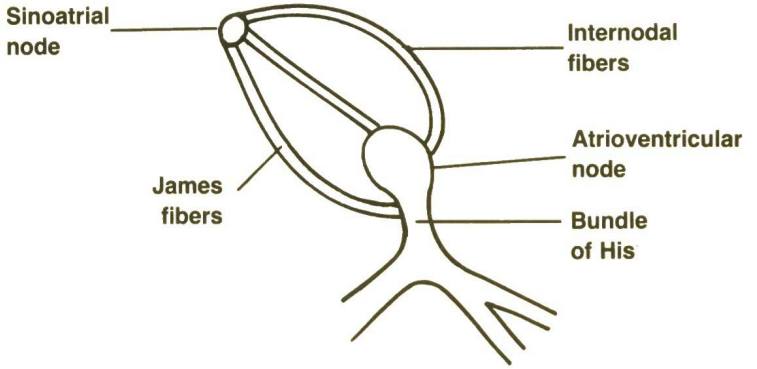

Figure 8-4. James fibers of Lown-Ganong-Levine syndrome terminating in the bundle of His.

less as shown in Figure 8-5. Ventricular depolarization takes place via the normal conduction pathway. Hence, the QRS is of normal configuration. The aberrant termination of internodal fibers can lead to a reentry depolarization wave, resulting in a supraventricular tachycardia with a normal-appearing QRS complex (Figure 8-6).

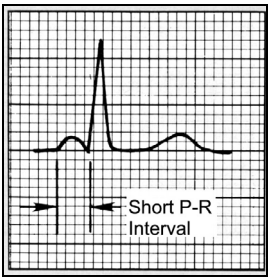

Figure 8-5. Electrocardiographic illustration of the Lown-Ganong-Levine syndrome.

The three criteria for Lown-Ganong-Levine syndrome are:

- A short P-R interval (0.12 seconds or less) without a delta wave
- A normal QRS complex
- A recurrent paroxysmal tachycardia

It should be noted that, unlike Wolff-Parkinson-White syndrome, episodes of tachycardia are required for the diagnosis of Lown-Ganong-Levine syndrome.

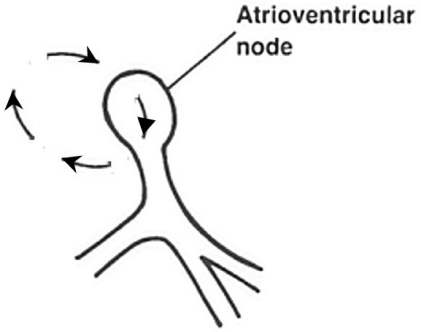

Figure 8-6. Reentry cycle of Lown-Ganong-Levine syndrome.

EARLY REPOLARIZATION

Early repolarization (Figure 8-7) is characterized by a concave ST segment elevation in the lateral leads (V_5, V_6, I, aVL). It is a benign condition that is more common in young, healthy, black men.

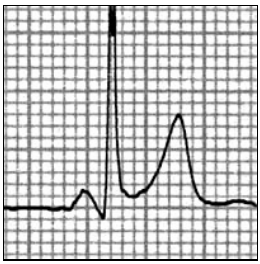

Figure 8-7. Early repolarization. Note the elevated J point.

PULMONARY EMBOLUS

The EKG is abnormal with *pulmonary embolus* in more than two thirds of the cases, but findings are not sensitive, nor specific; and classic findings are seen in fewer than 20 percent of the cases. The most common abnormal findings are sinus tachycardia and nonspecific ST segment and T-wave changes. The $S_1Q_3T_3$ pattern is a classic finding:

- S Wave in Lead I
- Q Wave in Lead III
- T Wave Inversion in Lead III

Other common findings include:

- Atrial fibrillation or other atrial arrhythmia (new onset)
- Findings that mimic myocardial infarction (ST segment and T wave changes)
- Right axis deviation
- Right bundle branch block (transient)
- Right-sided strain pattern
- T-wave inversion in precordial leads V_1 through V_4

The bottom line is that the EKG is a poor diagnostic test for pulmonary embolism. The greatest utility of the procedure in the patient with suspected pulmonary embolism is ruling out other potential life-threatening diagnoses such as myocardial infarction.

PERICARDITIS

Abnormal EKG findings are present in 90 percent of people with *pericarditis*. In general, changes may include ST segment changes, PR segment changes, low voltage QRS complexes, and *electrical alternans*, which is a broad term that describes alternate-beat variation in the direction, amplitude, and duration of any component of the EKG waveform.

Stages of Pericarditis

The EKG in acute viral pericarditis typically shows changes that evolve over a period of three to four weeks through four stages, although only about 50 percent of patients with pericarditis demonstrate all four phases.

Stage I

Stage I pericarditis (Figure 8-8A) is characterized by an onset of one to two days and a duration of up to two weeks. Electrocar-

diographic changes include (1) diffuse, concave ST segment elevation without a distinct J point; (2) ST segment depression in leads aVR or V_1 with concordant T wave changes; and (3) PR segment depression in leads II, aVF, and V_4 to V_6. There are no T-wave inversions at this stage.

Stage II

Stage II pericarditis (Figure 8-8B) is characterized by a duration of days to weeks. During this period of time the ST segments return toward baseline and the T waves flatten.

Stage III

Stage III pericarditis (Figure 8-8C) is characterized by an onset by week two or three and a duration of two to three weeks. Electrocardiographic findings include a return of ST segments to baseline and deep T wave inversion in leads II, aVF, and V_4 to V_6.

Stage IV

Stage IV pericarditis (Figure 8-8D) is characterized by a duration of up to three months, with gradual resolution of T wave inversion and return to normal.

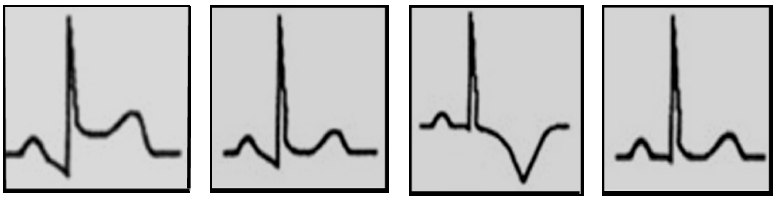

Figure 8-8. Stages of Pericarditis.

Acute Pericarditis versus Myocardial Infarction versus Early Repolarization

Comparisons of pericarditis to myocardial infarction and early repolarization are given in Table 8-1.

Table 8-1. Comparison of EKG changes associated with acute pericarditis, myocardial infarction, and early repolarization.

EKG Finding	Acute Pericarditis	Myocardial Infarction	Early Repolarization
ST segment shape	Concave upward	Convex upward	Concave upward
Q waves	Absent	Present	Absent
Reciprocal ST segment changes	Absent	Present	Absent
Location of ST segment elevation	Limb and chest leads	Area of involved myocardium	Chest leads
PR segment depression	Present	Absent	Absent

Chapter 9

Athletic Heart Syndrome, Ventricular Pacemaker, Drug Effects, Electrolyte Effects, EKG Worksheet, and Practice EKG

ATHLETIC HEART SYNDROME

A variety of abnormal EKG patterns occur in 40 percent of athletes. A small subgroup of these shows striking EKG abnormalities that suggest cardiovascular disease. However, these changes are likely an innocent consequence of long-term, intense athletic training and, therefore, just another component of *athlete heart syndrome*.

Resting sinus bradycardia is the most frequent abnormal finding in EKGs of well-conditioned athletes. Marathon runners may have a heart rate as low as 35 bpm. Athletes also have a considerably higher incidence of first and second degree atrioventricular block, more premature atrial beats, and slightly more premature ventricular beats.

Increased QRS complex height may be present, giving the appearance of left or right ventricular hypertrophy. Widened QRS complexes may simulate incomplete right bundle branch block. Repolarization changes may include ST segment elevation indicative of early repolarization. Flipped T waves may also occur. Despite these findings, athletic heart syndrome is a benign condition.

A typical EKG of a well-trained athlete is shown is Figure 9-1.

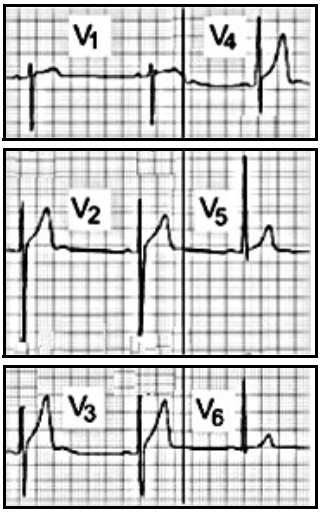

Figure 9-1. EKG from a well-trained athlete showing athletic heart syndrome. Note the marked bradycardia, increased QRS voltages, and ST segment elevation.

VENTRICULAR PACEMAKER

Ventricular pacemakers are battery-powered, implantable devices that electrically stimulate the heart, causing the ventricles to contract and thus to pump blood throughout the body. A pacemaker consists of a pager-sized device which contains a battery and the electronic circuitry that runs the pacemaker and one or two long thin wires that travel through a vein in the chest to the heart. Pacemakers are usually implanted in patients in whom the heart's sinoatrial node is no longer functioning normally. The electrocardiogram of someone with a pacemaker shows a spike at the time the pacemaker fires, followed by a wide QRS complex (Figure 9-2).

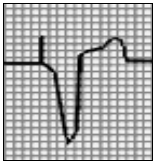

Figure 9-2. Electrocardiogram evidence of a ventricular pacemaker. Note the spike created by the pacemaker firing and the wide QRS complex.

DRUG EFFECTS

Digitalis

Changes in the electrocardiogram due to *digitalis* include modification of the ST-T contour, slowing of atrioventricular conduction, and enhancement of ectopic automaticity. Digitalis may produce a characteristic scooping of the ST-T complex (Figure 9-3). The ST segment and T wave are fused together and it is impossible to tell where one ends and the other begins.

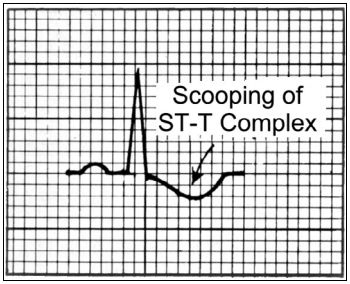

Figure 9-3. Effects of digitalis.

Digitalis effect may occur in the therapeutic range and should be distinguished from digitalis toxicity. *Digitalis toxicity* can cause virtually any arrhythmia and all degrees of atrioventricular block.

Quinidine

The effect of *quinidine* on the myocardium is to increase repolarization time, and, hence, prolong the Q-T interval and unmask a U wave. Quinidine, in toxic dose, may widen the QRS complex and cause ST segment depression, as can be seen in Figure 9-4.

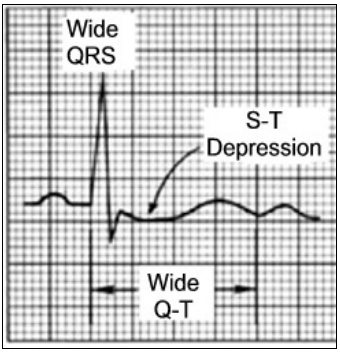

Figure 9-4. Effects of quinidine.

ELECTROLYTE EFFECTS

Potassium

Abnormally high *potassium* concentrations in the blood (hyperkalemia) produce tall, peaked T waves; widening of the QRS complex; and prolongation of the P-R interval. In the latter stages, it produces loss of P waves and spread of the QRS complex into a smooth, continuous sine wave. There is a rough correlation between the degree of change in the electrocardiogram and the concentration of potassium in the serum, as can be seen in Figure 9-5.

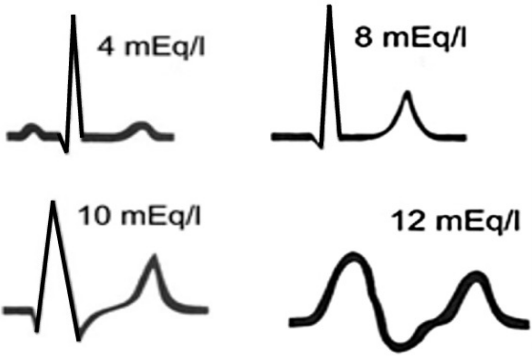

Figure 9-5. Correlations between serum potassium levels and the electrocardiogram in hyperkalemia.

Abnormally low potassium concentrations in the blood (hypokalemia) produce flattening of the T waves, which may unmask U waves as illustrated by Figure 9-6. T waves may become inverted, and ST segment depression may also occur.

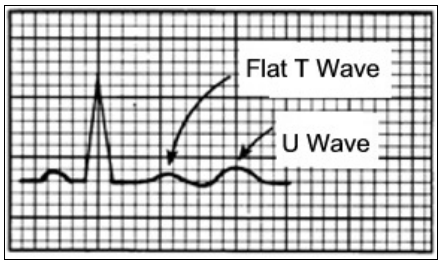

Figure 9-6. Effects of hypokalemia on the electrocardiogram.

Calcium

Excessive *calcium* in the blood (*hypercalcemia*) shortens ventricular repolarization time, resulting in a shortened Q-T interval (Figure 9-7A). *Hypocalcemia*, on the other hand, prolongs the Q-T interval (Figure 9-7B).

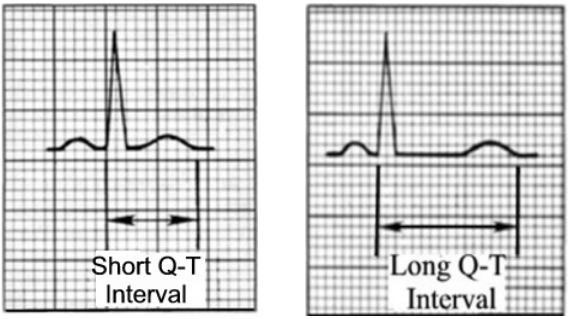

Figure 9-7. Effects of (A) hypercalemia and (B) hypocalcemia on the electrocardiogram.

EKG WORKSHEET

Electrocardiograms must be read in an orderly fashion. It is helpful to use a worksheet, such as the one presented in Table 9-1. After the calibration signal is observed (full or half standard) the usual procedure is to determine (1) rate, (2) rhythm, (3) intervals (4) axis, (5) ventricular hypertrophy, (6) atrial enlargement, and (7) signs of infarction or ischemia.

Table 9-1. A worksheet for collating electrocardiographic findings to facilitate interpretation.

1. Rate (bpm) **Atrial _____** **Ventricular _____**	**2. Rhythm** **Regular _____** **Irregular _____**
3. Intervals (seconds) **PR_____** **QRS _____** **QT_____**	**4. Axis** **Normal: yes____ no ____** **Right: yes ____ no____** **Left: yes____ no____**

5. Ventricular hypertrophy **Right ventricle: yes ___ no ___** **Left ventricle: yes ____ no ____**	**6. Atrial enlargement** **RA: yes ____ no ____** **LA: yes____ no ____**
7. Infarction or ischemia **Significant Q waves: leads _____** **ST segment elevation: leads _____** **ST segment depression: leads _____** **T-wave inversion: leads ___ _____** **R-wave progression: normal _____abnormal _____**	
8. Interpretation _____ **_____**	

PRACTICE EKG

Practice reading the EKG in Figure 9-8. Record you answers in table 9-1. The history is that the patient, a 56-year-old white male, presented to the emergency room of his local hospital 45 minutes after the onset of severe chest pain and nausea, but no vomiting. The results are given in Table 9-2.

There are a number of websites you can use to practice reading EKGs. Six of the most useful ones are:

- 12-lead ECG.com, http://www.12leadecg.com/.
- Dan Lemkin and Gary Plotnick, EKG Review, Sample arrhythmias, Introduction to EKGs, http://davidge2.umaryland.edu/~emig/ekg01.html
- Dean Jenkins and Stephen Gerred, http://www.ecglibrary.com/ecghome.html, October 12, 2002.
- ECG Rounds, MDchoice.com, http://www.mdchoice.com/ekg/ekg.asp.
- Emergency electrocardiography, Online Training Module, EMEDU.com, http://www.emedu.org/ecg/voz.php.
- Practice EKGs. What's your diagnosis? http://www.uga.edu/~lam/Barton/practiceECG.pdf.

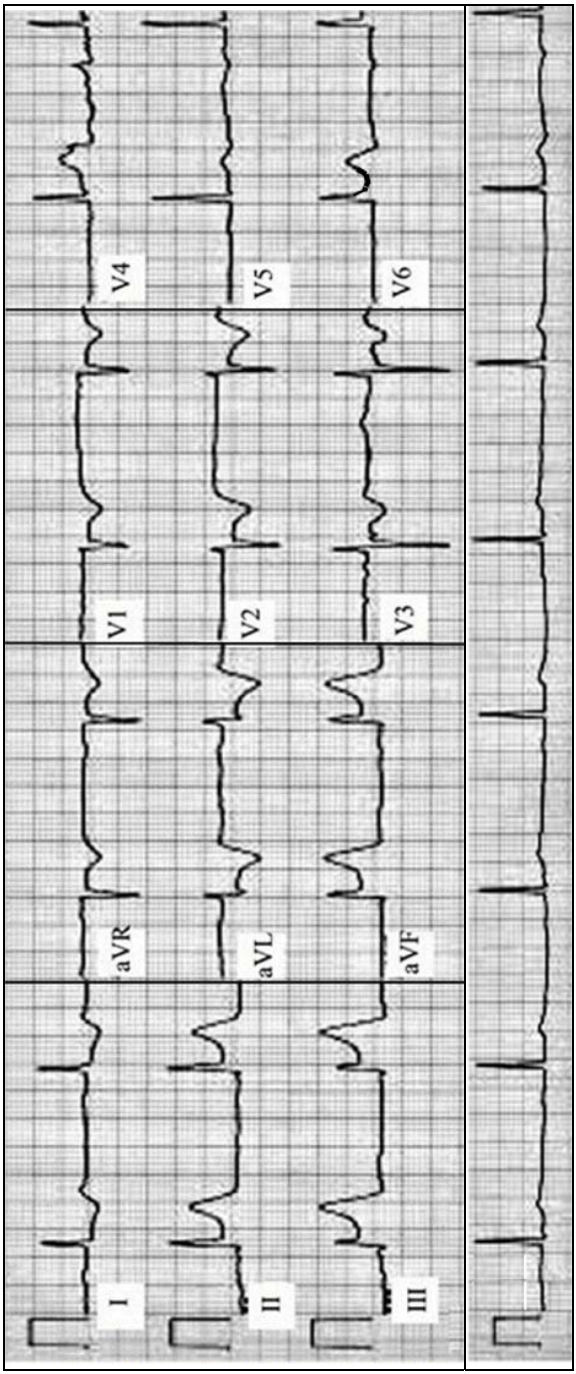

Figure 9-8. Practice EKG. Record your results in Table 9-1.

Table 9-2. Practice EKG results.

1. Rate (bpm) **Atrial** __*50*_____ **Ventricular** __*50*_____	**2. Rhythm** **Regular** ___*X*____ **Irregular** _____
3. Intervals (seconds) **PR**_____*0.28*_____ **QRS** ____*0.08*_____ **QT**_____ *0.4*_____	**4. Axis** **Normal: yes**__*X*_ **no** ____ **Right: yes** ____ **no**_____ **Left: yes**____ **no**_____
5. Ventricular hypertrophy **Right ventricle: yes** ____ **no** __*X*_ **Left ventricle: yes** ____ **no** __*X*_	**6. Atrial enlargement** **RA: yes** ____ **no** _*X*__ **LA: yes**____ **no** _*X*__
7. Infarction or ischemia **Significant Q waves: leads** _____*none*_____ **ST segment elevation: leads** ___*II, III, aVF*_____ **ST segment depression: leads**_____*aVL, V2,* _____ **T-wave inversion: leads** ___ __*aVR, aVL, V1, V2, V3*_____ **R-wave progression: normal** ___*normal*_____	
8. Interpretation *Early inferior myocardial infarction (Q waves not yet present)* _____ _____	

Appendix

A Quick Review of Electrocardiography

EKG Paper

The horizontal axis represents time. Large squares are 0.2 seconds in duration, while small squares are 0.04 seconds in duration. The vertical axis represents voltage. Usually large squares are 5 mm (0.5 mV), while small squares represent 1 mm (0.1 mV). Paper speed is 25 mm/second. Normal calibration = 10 mm (full standard) or 5 mm (half standard).

Conduction System of the Heart

Depolarization originates in the sinoatrial (SA) node; current travels through internodal tracts of the atria to the atrioventricular (AV) node; then through the Bundle of His, which divides into right and left bundle branches. The left bundle branch divides into left anterior and left posterior fascicles.

Deflections, Intervals, and Segments

P Wave. EKG deflection representing atrial depolarization. Atrial repolarization occurs during ventricular depolarization and is obscured. P wave normally largest in lead II. Upright in all leads except aVR. Less than 0.11 seconds long and 2.5 mV high.

QRS Complex. EKG deflection representing ventricular depolarization.

T Wave. EKG defection representing ventricular repolarization. Normally upright in all leads except aVR and less than 2.5 mm high.

U Wave. Second wave following T wave. Normally less than 1/3 of height of T wave. Significance unknown.

Low Voltage Deflections. Error occurs if interpreting EKG at full standard when it was recorded at half standard. Low voltage deflections are present in some normal individuals as well as pathological states that decrease the magnitude of the signal.

P-R Interval. 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. Beginning of P wave to beginning of QRS complex. Normally 0.12 to 0.2 seconds

QRS Interval. Time required for the ventricular cells to depolarize. Normal duration 0.06 to 0.10 seconds.

Q-T interval. Beginning of QRS complex to end of T wave. Inversely proportional to heart rate. The faster the heart rate, the shorter the Q-T interval. The Q-T interval represents about 40 percent of the total time between QRS complexes (the R-R interval). In most cases, the Q-T interval lasts between 0.34 and 0.42 seconds.

PR Segment. Portion of the tracing falling between the end of the P wave and the beginning of the QRS complex.

ST Segment. Portion of the tracing falling between the end of the QRS complex and the beginning of the T wave. The ST segment is normally even with the baseline.

ST-T Complex. Repolarization complex. The most sensitive part of the electrocardiogram. Consists of the ST segment and the T wave. 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 ST-T abnormalities are low T-wave voltages with slight sagging or flattening of the ST segment.

J Point. Intersection between the end of the QRS complex and the onset of the ST segment.

Leads

Limb Leads. I, II, III, aVR, aVL, aVF explore the electrical activity of the heart in a frontal plane; that is, the orientation of the heart seen when looking directly at the anterior chest.

Standard Limb Leads. Leads I, II, III form a set of axes 60° apart.

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

Augmented Voltage Leads. Leads aVR, aVL aVF form a set of axes 60° apart, but are rotated 30° from the axes of the standard limb leads.

- **aVR.** Exploring electrode located at the right arm. Negative "electrode" is formed by connecting the left arm and left foot electrodes together.
- **aVL.** Exploring electrode located at the left arm. Negative "electrode" is formed by connecting the right arm and left foot electrodes together.
- **aVF.** Exploring electrode located at the left foot (leg). Negative "electrode" is formed by connecting the electrodes of the two arms together.

Right arm and left arm electrodes may be placed on the right shoulder and left shoulder, respectively.

Chest Leads. Leads V_1, V_2, V_3, V_4, V_5, V_6 explore the electrical activity of the heart in the horizontal plane; that is, as if looking down on a cross section of the body at the level of the heart.

- **V_1.** Positioned in the 4th intercostal space just to the right of the sternum
- **V_2.** Positioned in the 4th intercostal space just to the left of the sternum
- **V_3.** Positioned halfway between V_2 and V_4
- **V_4.** Positioned at the 5th intercostal space in the mid-clavicular line
- **V_5.** Positioned in the anterior axillary line at the same level as V_4
- **V_6.** Positioned in the mid axillary line at the same level as V_4 and V_5

R-Wave Progression. V_1 Consists of a small R wave and a large S wave, whereas V_6 consists of a small Q wave and a large R wave. Since V_3 and V_4 are located midway between V_1 and V_6, the QRS complex in a normal EKG is nearly isoelectric in one of these leads; that is, the positive and negative deflections are about equal.

Lead Combinations

- **Lateral leads.** I, aVL, V_{4-6}
- **Inferior leads.** II, III, aVF
- **Anteroseptal leads.** V_1-V_4 (right side of heart), with V_1-V_2 (septum) and V_3-V_4 (anterior area of heart)

Normal EKG

Mean Electrical Activity. Can be represented by a vector. The length of the vector represents the magnitude of the activity (positive QRS deflection minus negative QRS deflection) and the angle represents the mean direction of the vector. A normal mean vector lies between 0 and 90 degrees, averaging 58 degrees.

Criteria

1. Pulse rate is between 60 and 100 bpm.
2. Rhythm is regular except for minor variations with respiration, usually no more than 10 percent.
3. P waves precede every QRS complex.
4. P waves are normally positive or biphasic in all leads, except aVR, and sometimes V_1.
5. P waves in lead II should be upright. Otherwise sinus rhythm is not present.
6. P-R interval is the time required for completion of atrial depolarization; conduction through the AV note, bundle of His, and bundle branches; and arrival at the ventricular myocardial cells. The normal P-R interval is 0.12 to 0.20 seconds.
7. The QRS interval represents the time required for ventricular cells to depolarize. The normal duration is 0.06 to 0.10 seconds.
8. The Q-T interval is the time required for depolarization and repolarization of the ventricles. The time required is inversely proportional to the heart rate. The faster the heart rate the shorter the Q-T interval. With slow heart rates, the Q-T interval is longer. The Q-T interval represents about 40 percent of the total time between the QRS complexes (the R-R interval). In most cases, the Q-T interval lasts between 0.34 and 0.42 seconds.
9. Lead I is mirror image of aVR.
10. The QRS deflections in leads I and III approximately equal that of lead II. Similarly, the sum of The QRS deflections in aVR, aVL, and aVF should approximately equal zero. When this is not true, there is reason to suspect the electrodes were placed incorrectly or that recordings were mixed up during mounting.
11. R-wave progression occurs in the chest leads, with the transition zone (point of equal positive and negative voltages) occurring somewhere between V_3 and V_4.

Estimation of Heart Rate

Rates of 50 to 300 bpm. Estimated from the number of large squares in an R-R interval. One large square = 300 bpm, two large squares = 150 bpm, three large squares = 100 bpm, and so forth, down to 50 bpm.

Rates of < 50 bpm. Estimated with the aid of markings at 3-second intervals along the graph paper. To calculate the rate, the cycles and partial cycles in a 6-second interval (two 3-second markings) are multiplied by 10.

Determination 0f Axis

Axis. Defined as the mean vector of ventricular depolarization.

Normal Axis. A mean vector between 0 and 90 degrees. Average = 58 degrees.

- **Right Axis Deviation (RAD).** A mean vector greater than 90 degrees.
- **Gray Zone**. A mean vector between 0 and -30 degrees. Usually considered normal.
- **Left Axis Deviation (LAD).** A mean vector more negative than -30 degrees.

Northwest axis. RAD or LAD. Indicated by a mean vector between -90 and 180 degrees. LAD much more common. Considered to be LAD in absence of reason to consider RAD.

Determining the Axis of the Mean Vector

Check lead aVF.

- If aVF is positive, check lead I
  - If lead I is positive, axis is normal.
  - If lead I is negative, there is right axis deviation
- If aVF is negative, check lead II.
  - If lead II is positive, axis is in the gray zone.
  - If lead II is negative, there is left axis deviation.

Atrial Enlargement

Normal P wave is < 2.5 high and 0.08-0.1 seconds in duration. To evaluate atrial enlargement, look at the P waves in leads II and V_1. The right atrium generates the left portion of the P wave, the left atrium generates the right portion.

- **Lead II.** Normally parallel to the axis of the atrial depolarization vector force. P wave configuration is positive deflection from baseline.
- **Lead V_1.** Normally closest to the atria and perpendicular to the axis of the atrial depolarization vector force. A positive deflection and then a negative deflection from the baseline, resulting in a sinusoidal appearing curve.

Right Atrial Enlargement (RAE). Generates an accentuated left-sided portion of the P wave. P > 2.5 mm in lead II and or greater than 1.5 mm in lead V_1. Biphasic P in V_1 with initial portion greater in amplitude than terminal portion.

Left Atrial Enlargement (LAE). Results in an accentuated right-sided portion of the P wave > 2.5 mm in any lead. P is double humped in any lead. Negative deflection of terminal portion of P in V_1.

Biatrial Enlargement

Features of both RAE and LAE. The P wave in lead II is greater than 2.5 mm tall and greater than or equal to 0.12 seconds in duration. The initial positive component of the P wave in V_1 is greater than 1.5 mm tall and a prominent terminal P component is present

Ventricular Hypertrophy

The EKG normally reflects left ventricular depolarization because left ventricular mass is much greater than right ventricular mass.

Right Ventricular Hypertrophy (RVH). When right ventricular muscle mass becomes great enough, it causes alterations in the positivity of the right chest leads. In the absence of myocardial infarction or right bundle branch block, the diagnosis of RVH can

be made when right axial deviation is present and when R > S in lead V_1 (R/S > 1) or S > R in lead V_6 (R/S < 1).

Left Ventricular Hypertrophy (LVH). Hypertrophy of the left ventricle causes an increase in the height and depth of the QRS complexes. LVH may be present when the sum of the S wave in V_1 or V_2 (whichever is larger) and the R wave in V_5 or V_6 (whichever is larger) $\geq$ 35 mm ($SV_{1-2} + RV_{5-6} \geq 35$). Accuracy in diagnosing LVH can be improved by considering limb lead criteria; that is, if the sum of the R wave in lead I and the S wave in lead III equal or greater than 25 mm (RI + SIII $\geq$ 25). Other criteria include R wave in aVL is > 11 mm and $RaVL + SV_3 > 28$ (M) or 20 (F). Criteria apply to individuals 35 years of age and older. Diagnosis is best made by echocardiography.

RVH with Strain (systolic overload). In addition to RVH criteria, T wave inversion and usually ST segment depression are present in the right chest leads.

LVH with Strain (systolic overload). In addition to criteria for LVH, T wave inversion and ST segment depression occur in the left chest leads.

Biventricular Hypertrophy

Biventricular Hypertrophy is a difficult EKG diagnosis to make. In the presence of LAE any one of the following suggests this diagnosis: R/S ratio in V_5 or V_6 < 1, S in V_5 or V_6 > 6 mm, or RAD is present. Other suggestive EKG findings include: Criteria for LVH and RVH are both met or LVH criteria is met and RAD or RAE is present.

Intraventricular Conduction Defects

Normally the entire process of ventricular depolarization occurs in less than 0.1 seconds. Any process that interferes with normal depolarization of the ventricles may prolong the QRS width.

Right Bundle Branch Block (RBBB). Septal depolarization results in a small R wave in V_1. Left ventricular depolarization results in an S wave. Right ventricular depolarization produces a second R wave (R′). The delayed depolarization of the right ventricle causes an increased width of the QRS complex to at least 0.12 seconds. Hence, RBBB is characterized by an R-R′ configuration in lead V_1 or V_2 with a QRS complex of 0.12 seconds or greater.

Incomplete RBBB. This shows the same QRS pattern as a complete RBBB; however, the QRS duration is between 0.1 and 0.12 seconds.

Left Bundle Branch Block (LBBB). Blockage of conduction in the left bundle branch prior to its bifurcation results primarily in delayed depolarization of the left ventricle. In LBBB, the septum depolarizes from right to left, since its depolarization now is initiated by the right bundle branch. Next the right ventricle depolarizes, followed by delayed depolarization of the left ventricle, giving an R-R′ configuration in lead V_5 or V_6 and a QRS interval 0.12 seconds or greater.

Incomplete LBBB. This shows the same QRS pattern as a complete LBBB; however, the QRS duration is between 0.1 and 0.12 seconds.

Fascicular Blocks (hemi-blocks). These are blockages of transmission that occur in the anterior or posterior branches (fascicles) of the left bundle branch. The main effect of a fascicular block is to markedly change the QRS axis without changing the shape or duration of the QRS wave form.

- **Left Anterior Hemiblock (LAH).** Results in left axis deviation (-30 degrees or more).
- **Left Posterior Hemiblock (LPH).** Results in right axis deviation (+90 degrees or more).

RBBB plus Hemiblock (bifascicular block). Blockage of right bundle branch plus LAH or LPH. Potentially significant since the

presence of either means the ventricles are being depolarized via only on fascicle of the left bundle branch and subject to complete heart block.

- **RBBB plus LAH.** Produces EKG picture of RBBB plus LAD.
- **RBBB plus LPH.** Produces EKG picture of RBBB plus RAD.

Nonspecific Intraventricular Conduction Defects. QRS duration greater than 0.10 seconds. Criteria for specific bundle branch or fascicular block not met.

Myocardial Ischemia

Myocardial ischemia is due to insufficient oxygen supply to the ventricular muscle. It may be transient, causing angina pectoris, or more severe, causing the death of a portion of heart muscle (myocardial infarction).

- **Subendocardial Ischemia.** Produces classic angina and subendocardial myocardial infarction. Involves the inner layer of ventricular muscle.
- **Transmural Ischemia.** Produces Prinzmetal's angina and transmural myocardial infarction. Involves the entire thickness of the ventricular wall.

Classic Angina, Produces transient ST segment depression (except in lead aVR, which may show reciprocal ST segment elevation). Not all patients with coronary artery disease show ST segment depression during chest pain.

Unstable Angina. Chest pain may occur while resting or even sleeping (*nocturnal angina*), and the discomfort may last longer and be more intense than that of stable angina. Unstable angina may be a sign of impending myocardial infarction.

Prinzmetal's Angina. Atypical angina that occurs at rest or at night and results in ST segment elevation. Thought to be caused by transient transmural ischemia due to vasospasm. May occur in individuals with otherwise normal coronary arteries.

Myocardial Infarction

Transmural Infarction. The infarcted area remains in a depolarized (negative) state. The loss of positivity in the infarcted area is responsible for the characteristic Q waves that develop in the leads exploring the infarcted area. A normal EKG may exhibit small Q waves in leads I, V_5, and V_6 that represent normal septal depolarization. Q waves, to be considered diagnostic of acute myocardial infarction, must (1) have a duration of at least 0.04 seconds or (2) have a depth equal to 25 percent or more of the height of the R wave.

Time Sequence of Myocardial Infarction

- **Acute Phase.** ST segment elevations generally appear within a few minutes and may last 3 to 4 days. During this period of time, Q waves appear in the leads showing the ST segment elevations.
- **Evolving Phase.** ST segments begin returning toward their baseline, and the T waves become inverted.
- **Resolving Phase.** In the weeks to months that follow, the T waves again return to the upright position. In most cases, the abnormal Q waves persist for months or even years.

Localization of Myocardial Infarction. Myocardial infarctions tend to be localized to left ventricular areas supplied by particular branches of the coronary arteries. They are described by their locations … anterior, inferior, and posterior.

- **Anterior Infarction.** Subdivided into strictly anterior, anteroseptal, and anterolateral infarctions. Strictly anterior infarction results in diagnostic changes in V_3 and V_4. Anteroseptal infarction results in loss of the normal small septal R waves in V_1 and V_2 as well as diagnostic changes in V_3 and V_4. Anterolateral infarction results in changes V_3 and V_4 and the more laterally situated chest leads (V_5, V_6) as well as the left lateral limb leads (I, aVL).
- **Inferior Infarction.** Produces changes in the leads that explore the heart from below (II, III, aVF).

- **Posterior Infarction.** Does not generate Q wave formation or ST segment elevation in the conventional 12-lead EKG since there are no posterior exploring electrodes. Instead, subtle reciprocal changes in the magnitude of R waves in V_1 and V_2 may occur. The R waves in V_1 and V_2 become taller than or equal to the S waves ($R/S \geq 1$). Unlike RVH, right axis deviation is not present. ST segment depression also may occur in these leads.

Silent Myocardial Infarction. Incidental finding of Q waves where they shouldn't be without a history of having had a prior myocardial infarction.

Subendocardial Infarction. Affects only repolarization (ST-T complex) and not depolarization (QRS complex). Hence, Q waves are not characteristic of subendocardial infarction. When subendocardial infarction occurs, the EKG may show persistent ST segment depression instead of the transient depression seen with classic angina. Persistent T wave inversion without ST segment depression may occur. The ST-T change slowly returns to normal as the infarction resolves.

Pseudoinfarction Syndromes. Some conditions may show EKG characteristics that can be confused with myocardial infarction:

- Complete or incomplete LBBB (QS waves or poor R wave progression in leads V_{1-3}).
- Dramatic alterations of ST segments and T waves may occur with increased intracranial pressure due to changes in repolarization that result from enhanced sympathetic nervous system activity.
- Hyperkalemia (ST segment elevation and peaked T waves).
- LAH (may see small Q waves in anterior chest leads).
- Left ventricular aneurysm after extensive infarction may show persistent ST segment elevation.
- LVH (may have QS pattern or poor R-wave progression in leads V_{1-3}).
- Patients with hypertrophic cardiomyopathy may have significant Q waves on their electrocardiograms due to distortion of

the normal pattern of depolarization because of the asymmetrical hypertrophied ventricular muscle.
- Pericarditis may show ST segment elevation and subsequent T-wave inversion. However, with pericarditis there is no Q-wave formation.
- Pneumothorax (loss of right precordial R waves).
- Pulmonary emphysema and cor pulmonale may show loss of R waves in V_{1-3} and/or inferior Q waves with right axis deviation.
- RVH (tall R waves in V_1 or V_2 may mimic true posterior MI).

Rhythm Disturbances

Sinus Rhythms. Sinus rhythms originate in the sinoatrial node. Diagnosis of sinus rhythms requires examining leads II and aVR for the correct polarity of the P waves. The P wave is always positive in lead II and negative in lead aVR. A P wave will precede each QRS complex, and the P-R interval should be relatively constant.

Categories of Arrhythmias

- **Regular.** R-R interval constant (except for minor variation with respiration)
- **Basically regular.** Regular except for occasional premature beats or escape beats
- **Regularly irregular.** R-R interval variable but with a definite pattern. (normal beats and ectopic beats grouped together and repeating over and over)
- **Irregularly irregular.** R-R interval variable with no pattern (totally irregular)

Sinus Arrhythmias. Initiated by SA node. Rhythm is irregular or varies more than 10 percent with respiration.

- **Sinus Tachycardia.** Defined as sinus rhythm with a rate > 100 bpm. With fast rates, P waves may merge with preceding T waves and be indistinct.

- **Sinus Bradycardia.** Defined as sinus rhythm with a rate < 60 bpm.
- **Sinoatrial Block.** Refers to failure of the sinus node to function for one or more beats. In this condition, there are one or more missing beats; that is, there are no P waves or QRS complexes seen during this period of time.
- **Sick Sinus Syndrome.** In elderly people, the sinus node may undergo degenerative changes and fail to function effectively. Periods of sinus arrest, sinus tachycardia, or sinus bradycardia may occur.

Non-sinus Atrial Arrhythmias. Non-sinus atrial arrhythmias include premature atrial beats, paroxysmal atrial tachycardia, multifocal atrial tachycardia, atrial flutter, and atrial fibrillation. Because the stimuli arise above the level of the ventricles, the QRS pattern usually is normal.

- **Premature Atrial Contraction (PAC).** An ectopic beat arising somewhere in either atrium, but not in the sinoatrial node. Occurs before the next normal beat is due, and a slight pause usually follows. The P wave may have a configuration different from the normal P wave and may even be of opposite polarity. Occasionally, the P wave will not be seen because it is lost in the preceding T wave. The P-R interval may be shorter then normal. If the premature atrial depolarization wave reaches the AV node before the node has had a chance to repolarize, it may not be conducted, and what may be seen is an abnormal P wave without a subsequent QRS complex. These premature atrial depolarization waves also may be conducted to ventricular tissue before complete repolarization has occurred, and in such cases, the subsequent ventricular depolarization may take place by an abnormal pathway, generating a wide, bizarre QRS complex (aberrancy).
- **Paroxysmal Atrial Tachycardia (PAT).** Defined as three or more consecutive PACs. PAT usually occurs at a regular rate, most commonly between 150 and 250 bpm. P waves may or may not be seen and the condition may be difficult to differentiate from sinus tachycardia.

- **Multi-Focal Atrial Tachycardia (MFAT).** Results from the presence of multiple, different atrial pacemaker foci. This rhythm disturbance is characterized by a tachycardia with beat-to-beat variation of the P wave morphology.
- **Atrial Flutter.** An ectopic atrial rhythm. Instead of P waves, characteristic sawtooth waves are seen. The atrial rate in atrial flutter is usually about 300 bpm. However, the AV junction is unable to contract at this rapid rate, so the ventricular rate is less … usually 150, 100, 75 bpm and so on. Atrial flutter with a ventricular rate of 150 bpm is called a two-to-one flutter because of the ratio of the atrial rate to the ventricular rate, a ventricular rate of 100 bpm is a three-to-one flutter because of the ratio of the atrial rate to the ventricular rate, and so on.
- **Atrial Fibrillation (AF).** With AF the atria are depolarized at an extremely rapid rate, greater then 400 bpm. This produces a characteristic wavy baseline pattern instead of normal P waves. Because the AV junction is refractory to most of the impulses reaching it, only a fraction of them are allowed to reach the ventricles. The ventricular rate, therefore, is only 110-180 bpm. Also characteristic of atrial fibrillation is a haphazardly irregular ventricular rhythm (variable R-R intervals).

Junctional Rhythms. Three types of junctional rhythms occur: Premature junctional contractions, junctional tachycardia, and junctional escape rhythms. Junctional rhythms arise in the AV junction. P waves, when seen, are opposite their normal polarity. They are called retrograde P waves. These P waves may precede, be buried in, or follow the QRS complex. Since the stimulus arises above the level of the ventricles, the QRS complex is usually of normal configuration.

- **Premature Junctional Contractions.** Can occur since the AV junction may also serve as an ectopic pacemaker. These are similar to PACs, in that they occur before the next beat is due and a slight pause follows the premature beat.
- **Junctional Tachycardia.** A run of three or more premature junctional beats. Has about the same rate as PAT and often cannot be distinguished from it. The difference is not clinically significant.

- **Junctional Escape Beat.** An escape beat that occurs after a pause in the normal sinus rhythm. Atrial pacing usually resumes after the junctional beat. A junctional escape rhythm, defined as a consecutive run of atrioventricular junctional beats, may develop if the SA node does not resume the pacemaker role. Junctional escape rhythm has a rate between 40 and 60 bpm.

Ventricular Rhythm Disturbances. Ventricular tissue is capable of spontaneous depolarization. When this occurs, a premature ventricular contraction (PVC) is initiated. Because the depolarization wave arises in the myocardium, it does not follow the normal path of ventricular depolarization. Therefore, the QRS complex is prolonged and bizarre in shape. In addition to PVCs, ectopic ventricular beats produce ventricular tachycardia and sometimes ventricular fibrillation. Ventricular escape rhythms also occur.

- **Premature Ventricular Contractions (PVCs).** PVCs are premature beats arising from the ventricles and are analogous to premature atrial contractions and premature junctional contractions. PVCs have two major characteristics: (1) They are premature and arise before the next normal beat is expected (a P wave is not seen before a PVC), and (2) they are aberrant in appearance. The QRS complex always is abnormally wide; the T wave and the QRS complex usually point in opposite directions. A PVC usually is followed by a compensatory pause. PVCs may be unifocal or multifocal. Unifocal PVCs arise from the same ventricular site, and as a result have the same appearance on a given EKG lead. Multifocal PVCs arise from different foci and give rise to different QRS patterns.
- **Ventricular Tachycardia (V-tach).** This is defined as a run of three or more PVCs and may occur in bursts or paroxysmally. V-tach may be persistent until stopped by intervention. The heart rate is usually 120 to 200 bpm. Ventricular tachycardia is a life-threatening arrhythmia. Torsades de pointes is a form of V-tach in which the QRS complexes swing up and down around the baseline in a chaotic pattern. The disorder is related to a prolonged Q-T interval. Some medications and

electrolyte imbalances may precipitate this in susceptible individuals.

- **Ventricular Fibrillation (V-fib).** This occurs when ventricles fail to beat in a coordinated fashion and instead twitch asynchronously. The beats are sometimes divided into coarse and fine rhythms.
- **Ventricular Escape Beats.** A ventricular focus may initiate depolarization when a faster pacemaker does not control the rate. It occurs after a pause in the regular rhythm. If a higher focus fails to pick up the rhythm, ventricular escape beats may continue. When this occurs, the rhythm is called *idioventricular* and has a rate usually less than 100 bpm. The QRS complex is wide and bizarre and P waves are not present. Idioventricular rhythms are usually of short duration and as a rule require no intervention.

Supraventricular Beat with Aberration. The depolarization wave is initiated above the ventricular level and, because it is premature, reaches the ventricles when they are in a partially depolarized state, resulting in a wide QRS complex resembling a PVC. The following rules can be used to determine aberrant ventricular depolarization: (1) The beat is aberrant if a P wave precedes the wide QRS complex, (2) the preceding R-R interval usually is longer than the other ones, (3) most aberrant beats are conducted via the left bundle branch, giving the appearance of right bundle branch block in lead V_1, and (4) the initial deflection of the wide QRS is in the same direction as that of the normal QRS complex.

Atrioventricular Heart Block. Heart block occurs in three forms: First degree, second degree, and third degree. Second degree heart block is divided into two types: Mobitz type 1 and Mobitz type 2.

- **First Degree Heart Block.** The EKG abnormality is simply a prolonged P-R interval to greater than 0.2 seconds.
- **Second Degree Heart Block, Mobitz Type 1.** The characteristic EKG is progressive lengthening of the P-R interval until finally a beat is dropped. The dropped beat is seen as a P wave that is not followed by a QRS complex.

- **Second Degree Heart Block, Mobitz Type 2.** A more severe form of second degree block, since it often progresses to complete heart block. The characteristic EKG picture is that of a series of non-conducted P waves; that is, 2:1, 3:1, 4:1 block.
- **Third Degree Heart Block.** Also known as *complete heart block*. The atrioventricular junction does not conduct any stimuli from the atria to the ventricles. Instead, the atria and the ventricles are paced independently. The characteristic EKG picture is: (1) P waves are present and occur at a rate faster than the ventricular rate; (2) QRS complexes are present and occur at a regular rate, usually < 60 bpm; and (3) the P waves bear no relationship to the QRS complexes. Thus, the P-R intervals are completely variable. The QRS complex may be of normal or abnormal width, depending on the location of the blockage in the AV junction (high or low). Trifascicular heart block is a form of third-degree heart block in which The right bundle and both branches of the left are blocked.

Bigeminy. *Bigeminy* is an alternating pattern of combinations of atrial-atrial, atrial-ventricular, and ventricular-ventricular beats. *Trigeminy* and even *quadrigeminy* also can occur.

Preexcitation Syndromes

Preexcitation syndromes refer to clinical conditions in which the wave of depolarization partially bypasses the atrioventricular node as it passes from the atria to the ventricles. The time required for the wave to leave the sinoatrial node and arrive at ventricular muscle (P-R interval) is, therefore, shortened. Two important preexcitation syndromes are (1) the Wolff-Parkinson-White syndrome, and (2) the Lown-Ganong-Levine syndrome.

Wolff-Parkinson-White Syndrome (WPW). Patients with WPW possess an accessory pathway of depolarization (bundle of Kent). The three electrocardiographic criteria for WPW are: (1) A short P-R interval, (2) a wide QRS complex, and (3) a delta wave. The QRS complex is widened by the delta wave in exactly the same amount as the P-R interval is shortened. The delta wave is a slurring of the initial portion of the QRS complex produced by early

depolarization of a portion of the ventricles. The major clinical manifestation of WPW is recurrent tachycardia with either normal or wide and bizarre QRS complexes depending on whether reentry via the AV node is antegrade or retrograde, respectively. Tachycardia is not required to make the diagnosis of WPW.

Lown-Ganong-Levine Syndrome (LGL). LGL is the result of some of the internodal fibers' (James fibers) bypassing the major portion of the atrioventricular node and terminating in the bundle of His. The three criteria for LGL are: (1) a short P-R interval without a delta wave, (2) a normal QRS, and (3) recurrent paroxysmal tachycardia. It should be noted that, unlike in WPW, episodes of tachycardia are required for the diagnosis of LGL.

Early Repolarization

Early repolarization is characterized by concave ST segment elevation in lateral leads (V_5, V_6, I, aVL). It is a benign condition that is more common in young, healthy, black men.

Pulmonary Embolus

The EKG is abnormal in more than two thirds of the cases, but findings are not sensitive, nor specific; and classic findings are seen in under 20 percent of the cases. The most common abnormal findings are sinus tachycardia and nonspecific ST-segment and T-wave changes. The $S_1Q_3T_3$ pattern is a classic finding (S Wave in Lead I, Q Wave in Lead III, T Wave inversion in Lead III). Other common findings include (1) atrial fibrillation or other atrial arrhythmia (new onset), (2) findings that mimic myocardial infarction (ST segment and T wave changes), (3) right axis deviation, (4) right bundle branch block (transient), (4) right sided strain pattern, and (5) T-wave inversion in precordial leads V_1 through V_4. The EKG is a poor diagnostic test for pulmonary embolism. The greatest utility of the procedure in the patient with suspected pulmonary embolism is ruling out other potential life-threatening diagnoses, such as myocardial infarction.

Pericarditis

The EKG in acute viral pericarditis typically shows changes that evolve over a period of three to four weeks through four stages, although only about 50 percent of patients with pericarditis demonstrate all four phases.

- **Stage I.** Characterized by an onset of one to two days and a duration of up to two weeks. Electrocardiographic changes include (1) diffuse, concave ST segment elevation without a distinct J point; (2) ST segment depression in leads aVR or V_1 with concordant T wave changes; and (3) PR segment depression in leads II, aVF, and V_4 to V_6. There are no T-wave inversions at this stage.
- **Stage II.** Characterized by a duration of days to weeks. During this period of time the ST segments return toward baseline and the T waves flatten.
- **Stage III.** Characterized by an onset by week two or three and a duration of two to three weeks. Electrocardiographic findings include a return of ST segments to baseline and deep T wave inversion in leads II, aVF, and V_4 to V_6.
- **Stage IV.** Characterized by a duration of up to three months, with gradual resolution of T wave inversion and return to normal.

Athletic Heart Syndrome

Resting sinus bradycardia is the most frequent abnormal finding in EKGs of well-conditioned athletes. Marathon runners may have a heart rate as low as 35 bpm. Athletes also have a considerably higher incidence of first and second degree atrioventricular block, more premature atrial beats, and slightly more premature ventricular beats. Increased QRS complex height may be present, giving the appearance of left or right ventricular hypertrophy. Widened QRS complexes may mimic incomplete right bundle branch block. Repolarization changes may include ST segment elevation indicative of early repolarization. Flipped T waves may also occur. Despite these findings, athletic heart syndrome is a benign condition.

Ventricular Pacemaker

The electrocardiogram of someone with a ventricular pacemaker shows a spike at the time the pacemaker fires, followed by a wide QRS complex.

Drug Effects

The drugs, digitalis and quinidine, produce major effects on an EKG that have considerable clinical significance.

- **Digitalis.** Changes include modification of the ST-T contour, slowing of AV conduction, and enhancement of ectopic automaticity. Digitalis may produce characteristic scooping of the ST-T complex. The ST segment and T wave are fused together, and it is impossible to tell where one ends and the other begins. This may occur even when digitalis is in the therapeutic range. With toxicity, digitalis can cause virtually any arrhythmia and all degrees of atrioventricular block.
- **Quinidine.** Increases repolarization time and, hence, prolongs the Q-T interval. In toxic doses, it may widen the QRS complex and cause ST segment depression.

Electrolyte Effects

Two electrolytes, potassium and calcium, produce significant EKG effects.

- **Potassium.** *Hyperkalemia* produces tall, peaked T waves, widening of the QRS complex, and prolongation of the P-R interval. *Hypokalemia* produces flattening of the T waves, which may unmask U waves. T waves may become inverted, and ST segment depression may occur. Marked decreased magnesium levels are usually associated with potassium depletion and the EKG demonstrates the characteristic changes of hypokalemia. Hypomagnesemia is usually associated with uremia and severe alcoholism.
- **Calcium.** *Hypercalcemia* shortens ventricular repolarization time, resulting in a shortened Q-T interval. *Hypocalcemia* prolongs the Q-T interval.

EKG Worksheet

1. Determine the calibration standard (full or half).
2. Determine the rate.
3. Determine the rhythm.
4. Determine the P-R, QRS, and Q-T intervals.
5. Determine the axis.
6. Determine whether atrial enlargement and/or ventricular hypertrophy exists.
7. Determine the presence or absence of infarction or ischemia.
8. Make an interpretation.

References

1. Argyle, Bruce, MicroEKG, http://www.madsci.com/manu/indexekg.htm.
2. Blake, T. M. Introduction to Electrocardiography. Appleton-Century Crofts, New York, 1972.
3. Chapter 8: Cardiology and Vascular Disease, TheDoctorsLounge.net, http://www.thedoctorslounge.net/linlounge/diseases/cardiology/.
4. Drugs which cause QT interval prolongation, DrugIntel Newsletter, http://www.drugintel.com/drugs/qt_arrhythmia.htm, February 14, 2003.
5. Dubin, D. Rapid Interpretation of EKG's. Cover Publishing Col, Tampa, 1976.
6. ECG Library, Contents, http://www.ecglibrary.com/ecghome.html.
7. EKG, FamilyPracticeNotebook.com, http://www.fpnotebook.com/CV52.htm.
8. Electrocardiogram in Pericarditis, FamilyPracticeNotebook.com, http://www.fpnotebook.com/CV76.htm.
9. Electrocardiograms, Interpretation of (Position Paper), Policy & Advocacy, AAFP, http://www.aafp.org/x6765.xml.
10. Electrocardiography, Echo services, http://www.echoservices.com.au/EchoSpeak/FAQ/Electrocardiography.aspx, 2002.
11. Electrocardiography, Health Guide A - Z, WebMD, http://my.webmd.com/hw/heart_disease/hw213248.asp.
12. Electrocardiography: An Overview, Catcha.com.My, http://www.catcha.com.my/channels/health/content_page.phtml?main=cardiology20.my.
13. Froom, J. and P. T. Tchao. A Curriculum in Electrocardiography for Family Physicians, J. Fam. Pract. 5:857-863, 1981.

14. Goldberger, A. L. and E. Goldberger. Clinical Electrocardiography, C. V. Mosby Co., St. Louis, 1981.
15. Gottlieb, S. H. D. D. Zieve, and G. C. Voight. Arrhythmias, In Principles of Ambulatory Medicine. Ed. by L. R. Barker, J. R. Burton, and J. R. Zieve. Williams and Wilkins Co., 1982.
16. Grauer, Ken, 12-lead ECGs: A Pocket Brain for Easy Interpretation (Third Edition), Kg/EKG Press, Gainesville, 2005.
17. Haddad, A. and D. C. Dean. Interpreting EKGs. Medical Economics Co. Oradell, New Jersey, 1981.
18. Hals, Gary D. and Stephen C. Carleton, Pericardial Disease, http://www.hypertension-consult.com/Secure/textbook articles/Textbook/59_pericardial.htm.
19. Iqbal, Itif, EKG Case #2 – Acute Pericarditis, Albany Medical Review, http://www.amc.edu/amr/archives/200408/ ekg2_ans.html, August 2004.
20. Jenkins, Dean and Stephen Gerred. ECG Library, Contents, http://www.ecglibrary.com/.
21. Marriott, Henry J. L., Practical Electrocardiography, 8th Edition, The Williams & Wilkins Co., Baltimore, 1988.
22. Martin, Gary and Arnold S. Baas, S-T-Elevation/Q-Wave Myocardial Infarction, Best Practice of Medicine, January 2004.
23. Meter, M. V. and P. G. Lavine. Reading EKGs Correctly. Nursing 77 Books, Intermed Communications, Jenkintown, Pennsylvania, 1981.
24. Milhorn, H. T. Jr. Electrocardiography for the Family Physician: Part I. Family Practice Recertification, 5(2)69-94, 1983.
25. Milhorn, H. T. Jr. Electrocardiography for the Family Physician: Part II. Family Practice Recertification, 5(3)105-130, 1983.
26. Milhorn, H. T. Jr. Electrocardiography for the Family Physician: Part III. Family Practice Recertification, 5(4)35-57, 1983.
27. Milhorn, H. T. Jr. Electrocardiography for the Family Physician: Part IV. Family Practice Recertification, 5(5)101-124, 1983.
28. Milhorn, H. T. Jr. Electrocardiography for the Family Physician: Part V. Family Practice Recertification, 5(6)121-137, 1983.

29. Mudge, G. H. Manual of Electrocardiography. Little-Brown and Co., Boston, 1981.
30. Outline of interactive electrocardiography, EKG Section, Loyola University Chicago, Stritch School of Medicine, http://www.meddean.luc.edu/lumen/MedEd/MEDICINE/medclerk/ekg.htm.
31. Scheidt, Steven, Basic Electrocardiography, Ciba-Geigy, West Caldwell, N.J., 1986.
32. Torsade de Pointe, eMedicine.com, http://www.emedicine.com/MED/topic2286.htm#section~workup, 2005.
33. Yanowitz, Frank G., Alan E. Lindsay Learning Center in Cyberspace, http://medstat.med.utah.edu/kw/ecg/index.html, May 24, 2005.

Figures 1-1 to 1-4; 2-1 to 2-5; 3-1 to 3-7; 4-1, 4.4, 4-5; 5-1 to 5-4; 6-1 to 6-3, 6-5 to 6-7; 7-2 to 7-4, 7-6, 7-7, 7-9, 7-11 to 7-15, 7-19 to 7-22; 8-1 to 8-8,; 9-3 to 9-7 are based on Milhorn, H. T., Electrocardiography for the Family Physician: Part I, Family Practice Recertification, Vol. 5(2),69-94, 1983; Part II, 5(3)105-130, 1983; Part III, 5(4)35-57, 1983; Part IV, 5(5)101-123, 1983; and Part V, 5(6)121-137, 1983.

Table 8-1 is based on Iqbal, Itif, EKG Case #2 – Acute Pericarditis, Albany Medical Review, http://www.amc.edu/amr/ archives/ 200408/EKG2_ans.html, August 2004.

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