150 Practice ECGs: Interpretation and Review

Third Edition

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Part I: How to Interpret ECGs
   Chapter 1: Baseline Data
   Chapter 2: Morphologic Changes in P, QRS, ST, and T
Part II: 150 Practice ECGs
Part III: Interpretation and Comments
For Marilyn
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Your problem as a student of electrocardiography is that you may not get enough practice to become good at it. The best way to get experience is to read ECGs from the hospital’s daily accumulation, commit your interpretation to paper, then look over the shoulder of the experienced person who is reading those ECGs for the record.

Unfortunately, most students and residents do not have that opportunity. Training programs are placing an ever-increasing clinical load on their faculties. One-on-one teaching experiences are hard to program. It is the rare institution that provides most of its students and residents headed for primary care practice with an adequate ECG reading experience.

This book is intended as an ECG curriculum that emphasizes practice. My goal is to have you reading ECGs as quickly as possible. The introductory chapters are shorter than those found in the usual beginner’s manual, but there is plenty there to get you started. Where you want additional depth, refer to an encyclopedic text in the library.

The practice ECGs include clinical data and questions that are designed to make teaching points. My brief discussion emphasizes daily issues in clinical medicine, as well as material that you may encounter on Board exams (Internal Medicine, Family Practice, Flex, and National Boards). Spend five evenings with these practice ECGs, and you will be far more comfortable than the average house officer with this basic part of the clinical examination.

Credit for the high quality of ECG reproduction in this book goes to Gordon Grindy and his colleagues at Marquette Electronics, Inc. My partner, Wes Moses, proofread the text and ECG interpretations, and I am also grateful to Dr. Hans Traberg who made useful suggestions for the 3rd edition. I again acknowledge that Marilyn Taylor is a patient woman, and I appreciate her forbearance during this writing adventure.

G.J.T.
How to Interpret ECGs

Normal Intervals

Heart Rate 60–99 beats/min
  bradycardia <60 beats/min
  tachycardia >100 beats/min
PR 0.12–0.21 sec
PR prolongation ≥0.22 sec
QRS < 0.12
QRS axis −30° to +105°
QTc the corrected QT interval (calculated as QT + \sqrt{\text{RR} \text{ interval}}).
It varies with age and gender, but is roughly <0.45 sec.
A Protocol for Reading ECGs

The protocol that you should follow when reading ECGs is outlined in Table 1.1. It is the approach cardiologists have taught generations of students, and it works. After reading ECGs for decades—and for a living—I still use it. With experience, I am good at pattern recognition. I glance at an ECG and promptly recognize major abnormalities. As you gain experience, you will develop this ability, and you will be tempted to focus immediately on the gross abnormalities that seem to jump out of the page. Resist that temptation! Do what the pros do, and make yourself follow the steps outlined in Table 1.1. Regardless of your ability and experience, if you do not focus on the rate, rhythm, intervals, and axis, you will miss subtle and important abnormalities. This is one of those areas of clinical medicine where you should not cut corners. Not addressing intervals, for example, would be like omitting the family history from a history and physical exam.

That analogy is a good one. The beauty of the history and physical examination format is that it allows you to collect meaningful data, even when the patient has an illness that you do not understand. Collecting basic data from the ECG serves a similar purpose for the novice.

How to Use This Book

First, read the introductory chapters that explain ECG findings and provide diagnostic criteria. Although useful, this exercise will not teach you how to read ECGs. You will take that step when you work through the practice tracings in Part II of this book.

When reading the unknown ECGs in Part II, write your interpretation. First, record rate, rhythm, intervals, and QRS axis. Then, analyze QRS and ST-T wave morphologies, and record your impression beginning with “ECG abnormal due to...” If you do not commit yourself on paper, it does not count! Finally, check your interpretation with mine, which is in Part III. Read five to ten tracings, or more, before checking answers. You will get into a kind of rhythm when you read ECGs without interruption.
Basic clinical data are provided with the ECGs, and I ask questions about management and diagnosis that go beyond the formal ECG report. Reading ECGs is a great opportunity to think (and teach) about heart disease, and I will not miss that opportunity here.

The remainder of this and the next chapter deal with each item on the ECG reading protocol (see Table 1.1). This book is for the near-beginner; most of you have had some introduction to the ECG. I will avoid lengthy description of technical areas such as the origin of lead systems. My goal is to provide brief yet clear explanations, and to get you through the introductory material as quickly as possible. Then it’s on to the practice ECGs.

### The ECG is a Voltmeter

It measures the small amount of voltage generated by depolarization of heart muscle. The vertical, or \(y\) axis, on the ECG is *voltage*, with each millimeter (mm) of paper equal to 0.1 millivolt (mV) (Fig 1.1). For practical purposes, we often refer to the amplitude, or height, of an ECG complex in millimeters of paper rather than in millivolts. At the beginning or end of the ECG, you may see a square wave, machine induced, that is 10 mm tall; this is a 1-mV current entered by the machine for calibration. The gain can be changed so that high-voltage complexes fit on the paper, or so that low-voltage complexes are magnified. Changing the gain is uncommon, but it would be apparent from the calibration marker.

Voltage may have either a negative or a positive value. This is because voltage is a *vector* force with *direction* as well as amplitude. All the rules of vector analysis apply.

Note that the wave of depolarization moves through the heart in three dimensions, but that each ECG lead records it in just one dimension, between two poles. Having 12 leads grouped in frontal and horizontal planes allows us to reconstruct electrical events in three dimensions (Fig 1.2). The vectorcardiogram, popular 40 years ago and seldom used now, displayed the wave of depolarization in three dimensions, using \(x\), \(y\), and \(z\) axes.

On the ECG, when the wave of depolarization moves toward the positive pole of an individual lead the deflection is upright, or positive. For example, if depolarization progresses from the right side of the heart to the left, the net voltage is positive in lead I (Fig 1.2). Downward deflections are negative. The general direction of the wave of depolarization, the orientation of its vector in space, is referred to as the electrical *axis*. Depolarization of the atria progresses from the upper right toward the lower left, so the
The square wave at the beginning is a 1-mV calibration marker. At full standard, 10 mm of paper = 1 mV of current. The ECG paper runs at 25 mm/sec. Thus, each millimeter = 0.04 second, and each large square (5 mm) = 0.2 second. The time between two positive deflections, or R waves, is the RR interval. If that is 1 second, the heart rate is 60 beats/min. This patient’s heart rate is 100 beats/min (60 seconds per minute ÷ 0.6 second per beat).

Spatial orientation of the 12 ECG leads. Each of the ECG leads functions as a voltmeter and has spatial orientation (as voltage is a vector force). Leads that have an inferior orientation are best at detecting changes from the inferior surface of the heart. Anterior precordial leads are most sensitive in detecting anterior wall changes, and the lateral leads, lateral wall abnormalities.
normal P wave axis is about 60°. Measurement of the QRS axis is discussed at the end of this chapter.

The ECG records the voltage generated by depolarization of the different regions of the heart through time. Following discharge of the sinoatrial (SA) node, the atria are depolarized (the P wave, Fig 1.3). Current then passes through the atroventricular (AV) node, where there is delay (the PR interval). When the wave of depolarization exits the AV node, it passes through the His bundle, then the bundle branches, and on to the ventricles. Discharge of the muscular ventricles produces the QRS complex. This is followed by repolarization of the ventricles (T wave).

**Measuring Heart Rate**

On older ECG machines, the paper moved at an arbitrarily set speed of 25 mm/sec. On current machines the paper is stationary and the stylus moves at 25 mm/sec, yet we customarily refer to “paper speed.” At this speed, each millimeter of ECG paper is equal to 1/25, or 0.04 second (see Fig 1.1). ECG paper is boldly ruled at 5-mm, or 0.2-second, intervals. And 5 of these large (5-mm) squares equals 1 second—straightforward arithmetic. Using this, there are a couple of fast ways to calculate heart rate when the rhythm is regular.
1. Check the distance (that is to say, the time) between two R waves. (The R wave is the dominant and easily identified positive (upright) wave or deflection in the QRS complex [see Fig 1.1].) That is the time for one cardiac cycle, or one heartbeat, and it is called the **RR interval**. If the RR interval is 5 large squares, or 1 second, then one heartbeat takes 1 second, and the rate is 60 beats/min. If the RR interval is 4 squares, or 0.8 sec/beat, then the heart rate is 60 sec/min divided by 0.8 sec/beat, which equals 75 beats/min. Three squares: \(60 \div 0.6 = 100\) beats/min.

2. A simpler way to do the arithmetic, and the way I determine rate quickly, is to measure the number of large squares between R waves, then divide that into 300: for 2 large squares, rate = 150 beats/min; 3 squares, rate = 100 beats/min; 4 squares, rate = 75/min; 5 squares, rate = 60/min; 6 squares, rate = 50/min; 5.5 squares, rate = between 50 and 60/min. When the rhythm is regular, I select an easily identifiable R wave that falls on, or near, a boldly scored line, then count the number of large squares to the next R wave. It is a crude but fast way to measure rate, but it does not work when the rhythm is grossly irregular. In most cases, it allows you to determine quickly whether the patient has a normal rate, **bradycardia** (less than 60 beats/min), or **tachycardia** (more than 100 beats/min).

## Intervals

After emphasizing the importance of following the reading protocol (see Table 1.1), I am already violating it by considering intervals before rhythm. This is useful, however, because the intervals are at times necessary to determine rhythm. First, let us review events of the normal cardiac cycle and the basic ECG nomenclature.

Depolarization of the SA node normally initiates the cardiac cycle (see Fig 1.3). This neural structure is small, and its depolarization generates a small amount of current that cannot be seen on the surface ECG (e.g., the 12-lead ECG measured from the surface of the body). The wave of depolarization spreads through both left and right atria, producing the P wave (see Fig 1.3).

Although the atria and ventricles have a broad area of surface contact, they are effectively insulated from each other by connective tissue. The wave of depolarization from the atrium is funneled through what I think of as a hole in the insulation, but it is actually specialized conducting tissue called the atrioventricular (AV) node. Current moves rapidly along nerves and fairly quickly through heart muscle. But the AV node puts the brakes on the wave of depolarization. This slowing creates a delay between atrial depolarization and ventricular depolarization. A pause in the AV node gives the atria time to contract, providing the final increment of ventricular filling. According to Dr. Starling, that is important; he discovered that greater ventricular volume—or individual muscle fiber length—at the beginning of ventricular contraction produces stronger contraction.

### PR Interval

The interval that includes a measure of the AV node conduction delay is the PR interval (see Fig 1.3). It is often easier to identify the beginning of the P wave than its end,
and by convention, this interval is measured from the start of the P wave. The interval thus includes the time of atrial depolarization, the P wave itself, and the delay during AV node conduction (roughly the time from the end of the P wave until the beginning of the QRS complex). However, when the PR interval is prolonged, it is usually a result of delayed AV node conduction; I know of no condition that lengthens the P wave enough to cause prolongation of the PR interval.

A common question is which ECG lead to use for measuring the PR or other intervals. What you are trying to measure with the PR interval is the time from initiation of atrial depolarization until the beginning of ventricular depolarization. There are slight variations in the sensitivities of particular ECG leads for recording the onset of the P wave, and which lead is most sensitive will vary from patient to patient. It makes sense to use the lead that records atrial depolarization earliest and ventricular depolarization earliest.

Do you get the feeling that these are rough measurements, despite the fact that we are dealing with milliseconds and microvolts? The truth is that they are, and that the surface ECG is a crude tool. As a practical matter, measure intervals from a lead where the onset of the waves—P and QRS—is well defined, and where the interval seems longest. This general rule applies to the measurement of all intervals.

The normal PR interval ranges from 0.12 to 0.22 second (see page 1). First-degree atrioventricular block (1° AV block) is defined as a PR interval of 0.22 second or more.

**QRS Duration**

Ventricular depolarization produces the QRS complex, the largest deflection on the ECG (Fig. 1.4, and see Fig 1.3). As a rule, the voltage generated is proportional to the amount of muscle depolarized, and the ventricles contain the bulk of cardiac muscle.

![QRS nomenclature](image)

**FIGURE 1.4** QRS nomenclature. Any positive deflection is an R wave. An initial negative deflection is a Q wave. A negative deflection following an R wave is an S wave. Small, low-voltage deflections may be designated with lowercase letters. When there are two R waves separated by an S wave, the second may be referred to as R' (R prime).
QRS nomenclature may seem confusing at first, but it follows quite simple conventions (Fig 1.4).

The QRS duration, or interval, is a measure of the time it takes to depolarize the two ventricles. Look again at Figure 1.3. Current exits the AV node and the His bundle and moves simultaneously through the infranodal bundle branches. Normally, the ventricles are activated at the same time, and the time of ventricular depolarization is roughly the duration of the QRS.

On the surface ECG, measure the QRS duration where it seems longest and where the beginning and end of the QRS are obvious. The normal duration is less than 0.12 second (3 mm). There are no illnesses that cause pathologic shortening of the QRS complex.

**T Wave and the QT Interval**

Repolarization, or the return of muscle to its resting state, spontaneously follows depolarization in heart muscle. Repolarization of the thin-walled atrium produces no apparent deflection on the surface ECG. Repolarization of the ventricles produces the T wave. This usually has the same axis as the QRS complex; that is to say, in ECG leads where the QRS complex is positive, the T wave is positive as well.

The QT interval is measured from the beginning of the QRS complex to the end of the T wave (see Fig 1.3). Why measure from the beginning of the QRS, apart from convention? It is probably because the beginning of the QRS often is easier to identify than the end, and the QRS complex is short relative to the duration of the QT interval. Measure the QT interval using the lead where it seems longest.

The normal duration of the QT interval varies with heart rate. The corrected QT (QTc) is calculated using Dr. Bazett’s formula:

\[ \text{QTc} = \text{QT} + \sqrt{\text{RR interval}} \]

The RR interval, or the duration of one cardiac cycle, is a measure of heart rate. Therefore, when the heart rate is 60 beats/min, and the RR interval is 1 second, the QTc equals the measured QT. When the heart rate is greater than 60 beats/min and the RR interval is less than 1 second, the QTc will be greater than the measured QT. Most ECG manuals provide tables that give the top-normal QT (measured) for a given heart rate, and these tables are based on Bazett’s formula with a top normal QTc that is roughly 0.45 sec. The normal range varies with age and gender.

There is a quick and easy method for determining whether the QT interval is normal, and it is the method I use when plowing through a stack of ECGs. If the measured QT is less than half the RR interval, then it is probably normal. If it is clearly longer, then it is probably abnormal. Using this shortcut, my ECG interpretation usually reads “QT normal for the rate” or “QT prolonged for the rate.” In borderline situations I calculate the QTc. The QTc provided by the ECG computer is occasionally inaccurate. Particularly with rapid heart rates, there is a tendency to overdiagnose QT prolongation, even with careful measurement.

The T wave may contain a second hump, or even a separate wave, which is called
the U wave, and this is a part of the ventricular repolarization process. It may be a normal finding. There is general agreement that it should be considered a part of the T wave when thinking of QT, or QTU, prolongation. Hypokalemia, especially in combination with hypomagnesemia, causes an increase in U wave amplitude and prolongation of the QTU interval.

QT interval prolongation is important. You will miss it unless you look for it on every ECG you read. One place you will see it is on Board exams. Conditions and drugs that prolong the QT interval are summarized in Table 1.2.

<table>
<thead>
<tr>
<th>Interval</th>
<th>Condition</th>
<th>Drugs/metabolic abnormality</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR</td>
<td>1’ AV block</td>
<td>Digoxin, β-adrenergic blockers, calcium channel blockers, intravenous adenosine</td>
</tr>
<tr>
<td>QRS</td>
<td>Ventricular conduction abnormalities, including bundle branch block; pre-excitation</td>
<td>Quinidine, flecainide, propafenone and other antiarrhythmics; extreme hyperkalemia</td>
</tr>
<tr>
<td>QT/QTU</td>
<td>Myocardial ischemia, hypothermia, intracranial bleeding, long QT syndrome</td>
<td>Quinidine, procainamide, disopyramide, sotalol, amiodarone, phenothiazine and phenothiazine derivatives, erythromycin; hypokalemia, hypomagnesemia, hypocalcemia</td>
</tr>
</tbody>
</table>

**PATHOPHYSIOLOGY**

Recall the shape of the action potential of isolated nerve or muscle cells. Repolarization (the return of the cell membrane to resting potential after depolarization) is a brief event, a sharp downward deflection. However, the repolarization wave on the surface ECG is broad. That is because the T wave is generated by repolarization of the large population of cardiac cells, some of which repolarize early and others much later. Doesn’t the T wave look like a bell-shaped curve? In a sense it is, with the average cell repolarizing at the peak of the T wave. A broader T wave indicates greater heterogeneity of the repolarization process among cardiac muscle cells so that it takes longer (electrophysiologists call this temporal dispersion of refractoriness).

This is clinically important because increased heterogeneity of repolarization is the substrate for reentry, which is the mechanism of most ventricular tachyarrhythmias. A longQT interval (a measure of the duration of repolarization) may identify the patient at risk for ventricular arrhythmias and sudden death (Table 1.2).

The QT prolongation of hypocalcemia is an exception, with somewhat less risk. That is probably because the heterogeneity of ventricular repolarization is less affected. This is the only cause of QT prolongation where the duration of the T wave is not prolonged—a normal T wave just occurs later.
Rhythm

My purpose is to help the student read through a stack of ECGs in the heart station (and look good to the attending). I will review selected rhythms common in this setting, but I will not attempt a comprehensive discussion of the rhythm abnormalities that you will encounter in telemetry units.

Sinus Rhythm and Sinus Arrhythmia

Normal sinus rhythm is a regular rhythm between 60 and 100 beats/min, with a P wave before each QRS complex and a QRS after each P wave. A faster rate defines tachycardia and a slower rate, bradycardia. The term sinus indicates that the rhythm originates in the sinoatrial (SA) node, that there is atrial depolarization (a P wave before each QRS), and that atrial contraction precedes ventricular contraction.

When you are excited, or when you walk up stairs and are short winded and your pulse is 120 beats/min, you have sinus tachycardia. This usually is a benign rhythm, but not always. It is a normal response of healthy people to exercise. But sinus tachycardia in a patient who is at rest and pain free the day after an MI may indicate severe left ventricular dysfunction. Cardiac output = stroke volume × heart rate. A depressed left ventricle generates less stroke volume, and increasing the rate is the first compensatory response to maintain output. Although a heart rate >90 beats/minute does not require specific treatment, it is a marker of decompensation and poor prognosis in patients who have had an MI and in those with congestive heart failure. Do not overlook other illnesses that may cause sinus tachycardia, such as thyrotoxicosis, anemia, and fever. It may also be caused by drugs, such as thyroid hormone, catecholamines, caffeine, and amphetamines.

Sinus bradycardia is a common finding. In the absence of conduction abnormalities, when all the intervals are normal, bradycardia at rest is a normal variant. It usually indicates good cardiovascular fitness, and it is common in trained athletes. It can be a drug effect (digitalis, β-adrenergic blockers, or the calcium channel blockers diltiazem and verapamil). A variety of illnesses can cause sinus slowing, including the sick sinus syndrome, hypothyroidism, sleep apnea, and other conditions that cause hypoxemia. Vasovagal attacks may include profound sinus bradycardia, sinus pauses, and syncope.

Sinus Arrhythmia

During the respiratory cycle, the vagus nerve is intermittently activated, producing a beat-to-beat variation in heart rate. On the 12-lead ECG (which is a relatively short rhythm strip), this is seen as a variable RR interval. When pronounced, it may affect your quick and easy calculation of heart rate using the technique just described. Be aware of this, but do not worry as long as the rate is within the normal limits.

Sinus arrhythmia usually indicates good cardiovascular health. It disappears when the heart is sick, as in the case of heart failure. The autonomic nervous system
compensates for low cardiac output by suppressing the parasympathetic nervous system as well as increasing sympathetic tone. Resting heart rate increases. In addition, the vagus nerve is not activated during the respiratory cycle, so there is little if any variation in RR intervals. The rhythm becomes perceptibly more regular.

Precise quantification of sinus arrhythmia, or heart rate variability (HRV), has emerged as a noninvasive test for increased risk of ventricular arrhythmias. It is not that vagal activity prevents dangerous ventricular arrhythmias. Rather, an active vagus nerve indicates good left ventricular function and therefore a low risk of arrhythmias. Those with low HRV (reduced vagal tone) usually have poor left ventricular function and an increased risk of ventricular arrhythmias and sudden cardiac death. HRV may be measured by calculating the mean and standard deviation of a large number of RR intervals; the standard deviation serves as a measure of the variability.

Heart Block

Block can be a confusing term in cardiovascular medicine. Blocked arteries, blocked valves, and blocked nerve conduction are different illnesses, and they may be confused by patients (and medical students). The term heart block usually refers to interruption of nerve conduction. It is an electrical problem, not one of fuel lines or valves (although these conditions may coexist).

Nerve conduction can be interrupted, or blocked, at any level of the cardiac nervous system (Fig 1.3). Block is uncommon within the SA node or in the body of the atrium. But it is quite common in the AV node and in the nerves below the AV node (Fig 1.3). These infranodal nerves include the His bundle, the bundle branches and their major divisions, and the small terminal Purkinje fibers. The infranodal nerves may be referred to as the His-Purkinje system.

Blocked conduction may alter intervals and may cause bradycardia. When block is complete, there is no transmission to structures distal to the block, but the heart rarely stops. Instead, an auxiliary pacemaker just below the level of block takes over. The intrinsic rate (the rate of spontaneous depolarization) of the takeover pacemaker is progressively slower the farther it is from the SA node. Control of heart rate reminds me of the children’s game, King of the Mountain. Pacers highest on the mountain, nearest the SA node, get the first chance to rule. When they fail, those just below take over. As you go lower down the mountain, the pacers are slower.

For example, when complete block occurs in the AV node, a pacemaker in the His bundle, just below the AV node, takes over with an intrinsic rate of 30 to 45 beats/
min. It would be hard to exercise with a heart rate that slow, but syncope is uncommon. If complete block occurs farther down, within the septum and beyond the division of the two bundle branches (see Fig 1.3), the takeover pacemaker is in the body of the ventricles. These deeper pacers have a much slower intrinsic rate, occasionally as slow as 10 to 20 beats/min. In this case, syncope and even sudden death are more likely.

From this outline of general principles, you begin to see that the level of block determines prognosis, and identification of this level is critical. Now we turn to specific ECG findings and clinical situations.

**First-Degree AV Block**

First-degree AV block is defined as a PR interval of 0.22 second or more, and without variation (Fig 1.5). It is caused by a delay in conduction in the AV node. Increased vagal tone, hyperkalemia, digitalis, calcium blockers (particularly diltiazem and verapamil), and β-adrenergic blockers all may slow AV node conduction. It is common in elderly patients, who may have degeneration of the AV node in the absence of ischemic heart disease. In other patients, ischemia may injure the AV node and either delay or block conduction. The right coronary artery usually supplies the AV node as well as the inferior wall of the heart, and AV nodal block is common with inferior myocardial infarction (MI).

**CLINICAL INSIGHT**

An underappreciated physical finding that accompanies PR interval prolongation is a soft first heart sound ($S_1$). Because of delayed conduction between atria and ventricles, contraction of the ventricle is much later than usual. During this delay, atrial contraction finishes, and the mitral and tricuspid valves drift toward the closed position. When the ventricles finally contract the valves do not have as far to travel, so the closure sound is softer. This is one of the few causes of a soft $S_1$.

**Second-Degree Heart Block, Mobitz I and II Block**

With second-degree AV block, some beats pass through the AV node to the ventricles but others do not. This follows a pattern: when every other P wave captures the
ventricle (producing a QRS complex), the patient is said to have 2:1 block. When every third P wave is conducted through the AV node, it is 3:1 block; and when two of three P waves are conducted, it is 3:2 block.

Second-degree heart block is further classified into two types: Mobitz I and II. This is a source of confusion. I find it easier to remember without the Mobitz designations, instead thinking anatomically of where the conduction system block occurs.

Mobitz I block occurs within the AV node (exceptions are rare.) Injury to the node causes it to tire with each succeeding beat until it is so tired that a P wave is completely blocked. On the ECG, we observe the Wenckebach phenomenon: progressive

![Figure 1.5](image1.png)

**FIGURE 1.5** First-degree AV block. The PR interval is longer than 0.22 second, and it does not vary.

![Figure 1.6](image2.png)

**FIGURE 1.6** Second-degree AV block, Mobitz type I (or Wenckebach). The level of block is the AV node. There is progressive lengthening of the PR interval until the P wave is not conducted. After the dropped beat, the PR interval is short (the AV node has had time to recuperate). An additional feature, not mentioned in the text, is progressive shortening of the RR interval before the dropped beat. Note that the QRS complex is narrow, more evidence that the level of block is the AV node.

**CLINICAL INSIGHT**

Think of Mobitz I or Wenckebach when you see group beating (groups of QRS complexes regularly separated by pauses). Then look for progressive prolongation of the PR, then a P wave not followed by a QRS, and then a shorter PR interval following the blocked beat. The diagnosis is supported by a narrow QRS complex, since the level of block is the AV node.
prolongation of the PR interval until there is a P wave that is blocked and not followed by a QRS (Fig 1.6). Notice that the PR interval of the beat following the blocked beat, or pause, is shorter. The AV node apparently recuperates during the pause. Often this short PR is the best evidence of Wenckebach, and can be used to make the diagnosis even when progressive PR prolongation is subtle and not certain.

Mobitz I block occurs at the level of the AV node, and the conduction system below the node may be normal. In fact, a normal QRS duration excludes block below the AV node. On the other hand, a wide QRS does not define block as infranodal. It is possible for a patient with a preexisting intraventricular conduction abnormality (and wide QRS) to develop AV nodal disease.

Mobitz II block, also a form of second-degree block, is caused by block below the AV node. The AV node may be healthy. With Mobitz II there is no progressive prolongation of the PR interval in the beats preceding the blocked beat (Fig 1.7). Because the infranodal conduction system is diseased, the QRS is wide, usually meeting criteria for bundle branch block. A narrow QRS excludes infranodal heart block. Mobitz II block often precedes symptomatic, complete heart block (see Fig 1.7) and is an indicator for pacemaker therapy.

2:1 AV Block—Is It Mobitz I or II?
Mobitz I second-degree AV block can be severe enough that every other beat is blocked (e.g., 2:1 AV block). This would eliminate the variable PR interval as a diagnostic marker. In fact, 2:1 AV block is a common rhythm with digitalis toxicity; it is Mobitz I, as the level of block is the AV node. How do you know whether 2:1 block is Mobitz I or Mobitz II? One way is to get a long rhythm strip: with Mobitz I block, there may be brief sections where block will be less severe, with 3:2 or 4:3 conduction and typical PR interval findings of the Wenckebach phenomenon.

Another way to tell is to focus on the QRS duration. I repeat this because it is important (it will be the key to answering a Board question). When block occurs at the level of the AV node, the infranodal conduction system usually is healthy, the ventricles are activated in the normal sequence—that is, simultaneously—so the QRS duration is normal. When block occurs below the AV node (Mobitz II), the patient invariably has an intraventricular conduction abnormality such as bundle branch block, and the QRS duration is long.
Here is how the electrophysiology laboratory assesses heart block. As noted, an elderly person with infranodal disease—bundle branch block—may develop a sick AV node and Mobitz I block. Whether it is really Mobitz I or II block can be sorted out in the electrophysiology lab, especially when there is uncertainty about a need for pacemaker therapy.

Depolarization of the proximal bundle of His, adjacent to the AV node, generates a small current that is not apparent on the surface ECG. This “H spike” can be measured with an electrode that is near it, using an electrode catheter positioned in the lower right atrium next to the tricuspid valve. The H wave allows partition of the PR interval into its nodal, and infranodal, or infra-His spike segments (Fig 1.8). Block in the node causes A-H interval prolongation, and block below the node—below the proximal His—causes H-V interval prolongation. Infranodal block identified by a long H-V interval is an indication for a pacemaker.

**FIGURE 1.8** His bundle recordings from three patients. The goal of EPS when evaluating heart block is to determine the anatomic level of conduction delay. The His (H) spike is generated by depolarization of the His bundle, just below and adjacent to the AV node, and is recorded with a bipolar catheter positioned next to the tricuspid valve. The H spike essentially divides the PR interval into its AV nodal (the AH interval) and infranodal (HV) portions. Patient A: the PR interval is normal as is the HV interval (<55 msec). Patient B: A patient with first-degree AV block (long PR). Marked prolongation of the AH interval indicates that the level of block is the AV node. Infranodal conduction (the HV interval) is normal. Patient C: Another with first-degree AV block. The AH interval is normal, so there is no delay in conduction in the AV node. The prolonged HV interval indicates infranodal conduction delay. With infranodal disease there is a higher risk of developing symptomatic heart block. (Reproduced with permission from Taylor C.J. Primary care management of heart disease. St. Louis, MO: Mosby. 2000.)
Third-Degree, Complete Heart Block

Complete atrioventricular block is just that; nothing gets through to the ventricles. There are P waves and QRS complexes, but they are unrelated; this is called AV dissociation (Fig 1.9). The term is used when P waves are not followed by QRS complexes; the atria and ventricles operate independently. Complete heart block is just one of the conditions where this occurs, and you will encounter other examples in Chapter 2.

How do you know whether block occurs within the AV node itself, or in the infranodal conduction system? The issues are those discussed above with 2:1 AV block. When block is at the level of the AV node, the takeover pacemaker is just below the node, within the His bundle and before the division into the bundle branches (see Fig 1.3). The sequence of ventricular activation is therefore normal, and the QRS duration is normal (unless there is coexisting bundle branch block). Furthermore, the takeover pacemaker is relatively high in the conduction system and has an intrinsic rate ranging from 35 to 45 beats/min. The rate would probably increase with catecholamine infusion or administration of atropine.

When complete block develops in the infranodal conduction system, the takeover pacemaker is in the body of the ventricle, the QRS is wide, and the rate is low. This may be called an idioventricular rhythm, but it should not be mistaken as a ventricular

![Figure 1.9](image_url)

**Figure 1.9** Three patients with complete heart block. The atria are being discharged at a regular rate (P waves) and the ventricles at a regular rate (QRS complexes). The two rhythms appear unrelated; there is AV dissociation. You may be tempted to say that P waves coming before QRS complexes could be conducted, but these do not alter the regularity of the ventricular escape rhythm. Patients A and B have wide QRS complexes and slow ventricular rates; they probably have block below the AV node, with a takeover pacemaker in the body of the ventricle. Patient C has a more rapid escape rate (55 beats/min), and the QRS complex is narrow; the level of block is the AV node.
arrhythmia. Suppressing it with an antiarrhythmic agent could lead to asystole and death. The idioventricular pacing rate does not increase following treatment with atropine or catecholamines.

Complete heart block may develop at the level of the AV node, but is uncommon. The usual situation is congenital heart block that is detected on a routine ECG in an asymptomatic young person (Fig 1.9C). The patient usually has a history of slow pulse, as the takeover pacemaker in the upper His system has a rate in the mid-40s. The heart rate may increase with exercise, although the response is subnormal. The QRS duration is normal. Pacemaker therapy is not required in the absence of symptoms.

**Heart Block with Acute Myocardial Infarction**

Heart block is a common complication of inferior MI, and it is uncommon with anterior MI (Fig 1.10). It helps to remember how the location of block determines the severity of the arrhythmia and prognosis. Heart block with *inferior MI* probably has dual causation. First, patients with inferior MI have high vagal tone, possibly related to the Bezold-Jarisch reflex. Second, there may be ischemia of the AV node, as the AV node is supplied by the same artery that feeds the inferior wall. You may expect the usual features of AV nodal block: PR interval prolongation and Mobitz I patterns are common, the QRS is narrow, and the takeover pacer is fairly rapid if block is complete. In addition, the AV node usually has collateral blood flow from other arteries, so permanent injury is uncommon and recovery of normal conduction is the rule. Permanent pacemaker therapy is rarely needed, although temporary pacing is indicated for symptomatic bradycardia. The heart rate usually increases with atropine therapy.

*Anterior MI* may injure the interventricular septum below the AV node, so the pattern of heart block is infranodal: Mobitz II block is the rule, the QRS is wide, and, when block is complete, the escape rhythm is slow. An anterior infarction large enough to cause infranodal block is usually huge, spontaneous recovery from the heart block is rare, and the prognosis is terrible. These patients need pacemakers, but despite pacing they do poorly because of the degree of LV injury.

**Atrial Arrhythmias**

Atrial arrhythmias may be chronic or acute. They are common, and you will see them almost daily when reading routine ECGs.
Heart block after MI. It is important to distinguish between block at the level of the AV node and block below the AV node. The takeover pacemaker with AV nodal block has an adequate intrinsic rate and responds to atropine. Deep ventricular pacemakers that take over after infranodal block are less responsive and are too slow.

**FIGURE 1.10**

<table>
<thead>
<tr>
<th>Location of Heart Block</th>
<th>Classification and ECG Appearance</th>
<th>QRS Duration</th>
<th>Origin of Escape Rhythm with Complete Heart Block</th>
<th>Prognosis</th>
<th>MI Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAN BLOCK AVN</td>
<td>1. First degree AV Block PR $\geq$ 0.22 sec</td>
<td>Normal (70-120 sec) rarely has associated bundle branch block</td>
<td>AV Node Heart rate $\leq$ 40 beats/min</td>
<td>Good (Usually spontaneous recovery)</td>
<td>Inferior MI (Right coronary artery supplies the AV node in 85%)</td>
</tr>
<tr>
<td>AV Block</td>
<td>2. Wenckebach, Mobitz 1, Block P</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>3. Complete heart block P P P P</td>
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<tr>
<td>Inferoanodal Block</td>
<td>1. Mobitz II P P P P P P P P</td>
<td>Wide (70.12 sec)</td>
<td>Ventricular Conduction System Usually Heart rate $\leq$ 20 beats/min</td>
<td>Poor (Large MI, heart failure)</td>
<td>Anterior MI (Septal ischemia)</td>
</tr>
<tr>
<td></td>
<td>2. Complete Heart Block P P P P P P P</td>
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</tbody>
</table>

**FIGURE 1.11**

Two patients with premature atrial contractions. **A**: An ectopic P wave is seen before the premature QRS. **B**: Blocked PAC, causing a pause. The ectopic wave is seen as a distortion of the preceding T wave. This is a common cause of pauses. In most cases, the ectopic P wave is harder to see; look for subtle changes in the preceding T wave.

**Premature Atrial Contractions**

Usually premature atrial contractions (PACs), also called PABs (beats) or APBs, are easy to recognize. The premature beat has a narrow QRS, and the QRS is identical to normal beats. A misshapen, ectopic P wave may precede it (Fig 1.11).
A blocked PAC may be the cause of a pause on the ECG or rhythm strip, a pause that may be felt by the patient (see Fig 1.11). This happens when the PAC is early enough that the AV node is refractory and will not conduct it. The P wave that is blocked may be buried in the T wave of the preceding complex, making it hard to see. Look for subtle alteration in the T wave just before the pause. This is a favorite board question.

PACs are common in young, healthy people and do not indicate heart disease.

**Paroxysmal Supraventricular Tachycardia**

Paroxysmal supraventricular tachycardia (PSVT) is a rapid, regular rhythm with a rate of 120 to 200 beats/min. Most cases are caused by reentry within the AV node.

**Reentry:** Before going further with PSVT, we should discuss reentry as a mechanism of premature beats and tachyarrhythmias. The concept is one that is often misunderstood, but is actually quite simple (Fig 1.12). The reentrant “focus” is an island of cardiac tissue that is protected, or insulated, from surrounding tissue. Current enters one end of the focus and exits the other (conduction is unidirectional). Within the focus, conduction is much slower than conduction through the surrounding tissue. By the time current exits the focus, the surrounding tissue has depolarized and has had

**FIGURE 1.12** Reentry. Follow the sequence of events. **A:** The wave of depolarization comes from above (the atrium in the case of atrial arrhythmias, the ventricle in this case of ventricular reentry). **B:** As current moves through the myocardium, it also enters the reentrant focus, a region that is insulated from the surrounding tissue. **C:** Depolarization of the surrounding myocardium happens quickly, but conduction through the reentrant focus is slow. **D:** By the time current exits the reentrant focus, the surrounding tissue has been repolarized and is vulnerable. That is to say, it can be stimulated. This produces the ectopic beat. **E:** If the timing is perfect, current from the ectopic beat reenters the protected focus, travels through it, and again finds the surrounding tissue vulnerable when it exits. A circuit is established and the result is repetitive beats.

Characteristics of the reentrant focus that make this possible are as follows: (1) insulation from surrounding tissue, (2) unidirectional conduction, and (3) slow conduction.