Conduction Overview

As mentioned in the Basic Beat chapter, the PR interval is the interval from the beginning of the P wave to the beginning of the QRS complex. It represents the time frame from the beginning of atrial depolarization to the beginning of ventricular depolarization.

Let’s break down the events that occur during the PR interval. Figure 10-1 shows how the electrical impulse relates to the ECG. First, the atria begin to depolarize by the transmission of the electrical impulse through the specialized conduction pathway of the atria, the Bachmann bundles, to the atrial myocytes. The impulse reaches the AV node before all of the atrial myocytes have depolarized because of the faster transmission down the Bachmann bundles. The depolarization of all of the atrial myocytes represents a larger electrical force than depolarization of the AV node, so the force seen on the ECG tracing is the P wave.

In the AV node, the conduction slows momentarily. (See dashed rectangle; note that the rectangle is superimposed under the P wave, representing this electrocardiographically silent event.) This physiologic slowing is needed to allow the mechanical emptying of atrial blood into the ventricles. Without this block, the atria and the ventricles would beat simultaneously and the ventricles would fill only by the passive inflow of blood during diastole. This would result in a decreased volume entering the ventricles and, hence, a smaller amount ejected from the ventricles. This lack of an “atrial kick” may lead to shock in many patients.

The His bundles, the next to be activated, transmit the impulse down the left and right bundle branches. Finally, the impulse reaches the individual Purkinje fibers, which will then innervate the ventricular myocytes. This is represented by the QRS complex on the ECG tracing.
PACs may have a shorter or longer PR interval. Why does this occur? To answer that question, look at the previous page and analyze the components of the PR interval. There are several locations where we can gain or lose a few milliseconds. We gain a few milliseconds, making the PR interval shorter, if the origin of the P wave is near the AV node, or it bypasses the AV node with its physiologic block altogether. We can lose a few milliseconds, making the PR interval longer, by having an ectopic atrial impulse transmitted from cell to cell directly rather than through the internodal pathways. Some other factors that can alter the PR interval involve prolongation of the physiologic block by vagal stimulation, drugs, or electrolyte abnormalities. The PR interval will also lengthen with prolongation of the conduction in the His bundles, the bundle branches, or in the Purkinje system caused by the same factors — or by the presence of anatomic blocks to the impulse path.

**Figure 10-2**: PR segment positions in relation to the baseline.

1. The baseline of the ECG is measured from TP segment to ____.
2. Should the PR segment fall on the baseline?
3. Should the ST segment fall on the baseline?
4. The baseline cannot always be measured because of rapid tachycardias that do not show a clear TP segment. True or False.
5. PR segment elevation is a common occurrence seen on most ECGs. True or False.
PR Depression

An example of PR depression is shown in Figure 10-3. The differential diagnosis of PR depression includes:

1. Normal variant
   The PR segment is usually on the baseline. However, it is sometimes found to be slightly depressed. In order for it to be considered normal, it cannot be depressed more than 0.8 mm below the baseline. This normal variant is due to atrial repolarization, which pulls the PR segment downward. The atrial repolarization wave is called the Tp wave. It is usually not seen because it is buried in the QRS wave.

2. Pericarditis
   Pericarditis is an inflammation of the pericardium, the fibrous sac that encircles and protects the heart. At this point, we only want you to concentrate on the PR segment when you look at the next few examples. Just remember that pericarditis is a pathological process that may or may not have PR depression that is greater than or equal to 0.8 mm. When you revisit this area as a graduate to Level 2, you will learn the other criteria.

3. Atrial infarction
   This is very rare. You see it when there is significant PR depression in an ECG with signs of infarction and without any of the criteria for pericarditis.

When pericarditis is present, it presents electrocardiographically with one or more of these signs:

1. Tachycardia
2. PR depression
3. Diffuse ST segment elevation. Note that the ST elevations are usually concave up with a scooped-out appearance.
4. Notching of the terminal portion of the QRS complex, especially in the lateral precordial leads

Look at the example ECGs that follow in the next few pages. Can you find one or more of the pericarditis criteria in any of them? The history will be very helpful in these cases, as the patient usually presents with sharp chest pain that hurts more on inspiration, coughing, or lying back. The pain will be relieved when sitting forward.

The Tp wave is usually buried inside the QRS complex and, therefore, is not seen. You can sometimes see it as the ST depression that occurs in very rapid supraventricular tachycardias, especially rapid sinus tachycardias. In general, these cases have poor baselines with TP segments that are not clearly identifiable.

Atrial infarctions are rare because of the relatively small pressures encountered in the atria and the thinness of the atrial walls. In addition, the circulation to the atria includes thebesian veins that carry blood directly to the tissues. These small veins originate in the atrial or ventricular cavities and bypass the coronary system.
ECG CASE STUDY  PR Interval Depression

ECG 10-1  Because this is the chapter on PR intervals, we want you to concentrate on them in these examples. In the following example, where is the baseline? Take a piece of paper and place the edge on the TP segments surrounding the complex you want to examine. If you placed the paper below the complex, you should not be able to see the PR interval because it is depressed. Now put the paper’s edge so that the paper is on the top. (This should hide most of the QRS complex.) You should now be able to see the PR segment and calculate the amount of depression. In this case, it is about a whole block, or 1.0 mm. When you see PR depression, think of pericarditis or atrial infarct. We will discuss pericarditis further in the ST segment chapter. How long is the PR interval in this ECG? Is it prolonged?

REMINDER

Use a straight edge or ECG ruler to calculate the baseline. Remember — it extends from TP segment to TP segment.

ECG 10-1  Notice that all of the criteria for acute pericarditis are present on this ECG, except tachycardia:

1. Diffuse ST segment elevations, which are scooped and upwardly concave
2. PR depression
3. Notching of the S wave

When you see ST elevation in the inferior and the precordials from V3 to V6, you should think of an inferolateral acute myocardial infarction (AMI). If the ST elevation includes V2, it is indicative of a special kind of AMI known as an apical AMI. This is usually due to a very large right coronary dominant system.
CHAPTER 10  ■  The PR Interval

ECG 10-1
**Measuring the Interval**

The normal PR interval, shown in Figure 10-4, is from 0.12 seconds to 0.20 seconds in length. The PR interval is considered short when it is less than or equal to 0.11 seconds (Figure 10-5), and prolonged when it is more than 0.20 seconds (Figure 10-6). The interval should be measured in the lead with the widest P wave and the widest QRS complex in order to avoid the inadvertent omission of an isoelectric portion of a P wave. If your calculation does not take into account this isoelectric portion, it will give you a falsely shortened PR interval. You avoid the problems with isoelectric portions by using the lead with the longest PR interval to take your measurement. Remember that intervals should be the same throughout all of the leads. This will become more evident in future sections.

The PR interval is shortened in sinus tachycardia and in kids. It is usually longer in the elderly.

* Most books refer to a normal PR interval being from 0.12 to 0.20 seconds and first-degree heart block as more than 0.20 seconds. However, in their examples, they include 0.20 seconds as prolonged. In this book, we will consider 0.20 seconds as borderline PR prolongation.
CLINICAL PEARL

When you have a prolonged PR interval, take a quick look at the rest of the intervals. If they are all prolonged, there may be a metabolic problem causing it; commonly it is a high potassium level.

QUICK REVIEW

1. The PR interval can be normal, short, or _______.
2. The normal PR interval is from _______ to _______.
3. The PR interval is considered short if it is less than or equal to _______ seconds long.
4. Tachycardia will lengthen the PR interval. True or False.
5. The PR interval can be measured in any lead. True or False.
6. Intervals can vary from one lead to another. True or False.

1. Prolonged 2. 0.12 to 0.19 sec 3. 0.11 4. False 5. False 6. False. They can appear shorter or longer but the intervals will always be the same.

1. Can you think of the differential diagnosis for a shortened PR interval?
2. Can you think of the differential diagnosis for a prolonged PR interval?
3. Why do we have isoelectric sections in the different leads?
Short PR Interval

The PR interval is considered short if it is less than or equal to 0.11 seconds. There are three major mechanisms that cause a short PR interval:

1. Retrograde junctional P waves
2. Lown-Ganong-Levine syndrome (LGL)
3. Wolff-Parkinson-White pattern and syndrome (WPW)

We discussed retrograde P waves in the previous chapter. Go back and review it if you need to. It is an important point that you will run into again and again.

LGL syndrome is a benign condition associated with a short PR interval, a normal P wave, and a normal QRS. Some authors believe that it must be associated with tachycardias, but others disagree. Just keep in mind that the possibility of paroxysmal tachycardia or other tachycardias exists. The explanation for the short PR interval is that the impulse is transmitted through a bypass tract called James fibers, shown in Figure 10-7. These fibers bypass the upper and central portions of the AV node where the normal physiologic block occurs. The impulse thus bypasses the normal physiologic block, shortening the PR interval. The QRS complex is normal because conduction through the His bundles and bundle branches proceeds normally.

We will discuss WPW syndrome shortly.

QUICK REVIEW

1. What are the major causes of a short PR interval?
2. Retrograde P waves are easily identifiable on an ECG because the P waves are inverted in leads II, III, and aVF. True or False.
3. What is the name of the syndrome that features a short PR interval with a normal QRS complex?
4. What is the name of the bypass tract associated with LGL syndrome?
5. Would you be surprised if a patient with a short PR interval and a normal QRS complex reported episodes of very rapid heart rate?

There are two other types of bypass tracts besides James fibers. Can you name them? They are the Kent bundle and the Mahaim fibers. Mahaim fibers are a short bypass tract that connects the lower AV node or the His bundle with the interventricular septum. The Mahaim fibers are associated with a delta wave and can account for some of the cases of WPW. These two fiber tracts can coexist in the same patients, although it is rare.

Note that in patients with Mahaim fibers, the PR interval should be normal because the normal physiologic block has been maintained. The Kent bundle bypasses the AV node, and thus can have a shortened PR interval.
**ECG CASE STUDY**

**Short PR Interval**

**ECG 10-2** That’s a short PR interval! It is about 0.8 seconds long. This is an example of LGL syndrome. It contains a very short PR interval and normal waves in the complex. What is the significance of this? Not much, except that it may be associated with tachycardias. Why are we spending the time to go over LGL and WPW (next section)? Simply because they are conditions that are commonly overlooked. We had one patient who presented to the emergency department 36 times with a complaint of syncope (fainting). He had about 20 ECGs done during those visits. The man was sent to a psychiatrist, who placed him on antidepressants and antipsychotics. This all led to a downward spiral in the patient’s life that could have been avoided by recognizing WPW.

**ECG 10-2** The underlying rhythm is a sinus arrhythmia. There is not a lot more to say about this ECG. So, we’re going to talk about the need to know and remember the differential diagnosis of the various findings. To be a great clinician, you have to think of all of the possibilities related to the patient’s condition. The only way to make the correct diagnosis is to have thought about it. Use the information you have to rule in or out the specific conditions. Make some 3×5 cards with the differentials we give you in this book and carry them with you. Review the cards for a few days and you’ll never forget them.
Wolff-Parkinson-White Syndrome (WPW)
The syndrome of Wolff-Parkinson-White is defined by:

1. Shortened PR interval (< 0.12 seconds) with a normal P wave
2. Wide QRS complex (≥ 0.11 seconds)
3. The presence of a delta wave
4. ST-T wave changes or abnormalities
5. Association with paroxysmal tachycardias

Patients with WPW have a tract that bypasses the AV node altogether known as the Kent bundle, shown in Figure 10-8. Now imagine the impulse traveling down through the atria. It reaches the Kent bundle and the AV node just about simultaneously. The impulse travels down the AV node and is met by the normal physiologic block. The impulse also travels down the Kent bundle, doesn’t meet any block, and so begins to spread through the ventricular myocardium. This progression is slow and gives a wide pattern on the ECG tracing. This is, in reality, the same as saying that a premature ventricular contraction (PVC) (wide, bizarre complex) is starting at the terminal point of the Kent bundle. Now, remember that impulse traveling down the AV node? It starts down the normal conduction pathway and depolarizes the myocardium that has not already been depolarized by the Kent bundle impulse. Because the AV nodal impulse is much faster than transmission of the Kent bundle impulse through the myocardium, the two waves meet and extinguish each other because of the refractoriness of the two areas. The slow Kent bundle impulse is superimposed or fused on the normal impulse and forms a fusion beat with a delta wave as shown in Figure 10-9. The actual delta wave is the initial slurring of the QRS; it represents the small amount of tissue that was stimulated by the Kent bundle impulse wave.

If the patient has all of the above findings except for tachycardia, it is known as the WPW pattern. In addition, 12% of patients have a normal PR interval. Why the big deal and the full page devoted to WPW, you ask? Well, WPW is associated with tachycardias, as mentioned above. These tachycardias can be wide (> 0.12 sec), regular or irregular, and very, very fast. The distinction between a supraventricular pattern and a ventricular tachycardia pattern is difficult, sometimes impossible. Treatment for these tachycardias is beyond the scope of this book, but we highly recommend that you spend the time to fully understand the treatment strategies and why they are important. Just remember that you should treat a wide-complex tachycardia as if it is ventricular tachycardia, until proven otherwise.

**Figure 10-8:** The Kent bundle.

**Figure 10-9:** The delta wave.
**ECG CASE STUDIES**  

**WPW**

**ECG 10-3** This is a classic example of a WPW pattern on an ECG. Notice there is a short PR interval and very distinct delta waves. In this case, the delta waves are seen in all of the leads. This doesn’t always occur, because some leads are isoelectric to the delta wave component.  
Do leads III and aVF have delta waves? Yes, but they are negative (deflected downward). If you have followed the format of the book, you should already have reviewed Q waves, and the basic infarct patterns at the end of the book. When you look at leads III and aVF, they are similar to — and sometimes confused with — Q waves. This similarity to Q waves has given rise to the term **pseudoinfarct pattern**. Please remember that this is not a true infarct.  
Take a look at the ST and T wave changes in this ECG. Look at the ST elevation in V1 to V3, and the flipped Ts in I, aVL, and V4 to V6. Are they a sign of ischemia? Not in a patient with WPW. What happens is that, because part of the depolarization wave travels down the accessory pathway, it causes the repolarization also to be abnormal. This abnormal repolarization gives rise to all sorts of ST and T wave abnormalities. It is therefore very hard, if not impossible, to diagnose AMI based on the standard criteria in patients with WPW. Let the history guide you in making the diagnosis, and consult a cardiologist as soon as possible if you suspect an AMI.

**REMINDER**

Lown-Ganong-Levine (LGL) is usually benign. Wolff-Parkinson-White (WPW) can be life threatening!
CHAPTER 10  ■  The PR Interval

ECG CASE STUDIES CONTINUED

ECG 10-3

Fig. 1284-1_01_0158A
Fig. ECG 10.03

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CHAPTER 10  ■  The PR Interval

ECG CASE STUDIES CONTINUED

ECG 10-3
WPW Syndrome — Advanced Information

There are 3 types of WPW:

Type A: In this type of WPW, the QRS complexes are primarily upright in all of the precordial leads. A good way to remember it is to look at V₁: in type A, you can draw a small line across the QRS complex and it resembles an “A” (Figure 10-10, top). Type B, on the other hand, is negative in V₁ and V₂ and — if you use your imagination — can look like a “b” (Figure 10-10, bottom). It can sometimes resemble a right bundle branch block with an RSR’ pattern, for which it is usually mistaken. The ST-T wave repolarization abnormalities are seen usually in the right precordials, and present as ST depressions and T wave inversions. Type A is usually associated with a Kent bundle on the left side of the heart.

Type B: In type B, the QRS complexes are negative in V₁ and V₂, and upright in the left-sided precordial leads. It can be mistaken for a left bundle branch block because of this pattern. The repolarization abnormalities are seen in the left precordials.

Type C: In this type of WPW, the complexes are upright in V₁ to V₄, and negative in V₅ to V₆. It starts off like WPW type A, but does not maintain positive complexes all the way to the lateral leads. This type is very rare.

All types of WPW can be mistaken for infarcts when the delta wave is negative, because it resembles a Q wave. This is especially prominent when the deflections are negative in the inferior leads. This pattern is called pseudoinfarct because it is not associated with a myocardial infarction (see type B diagram). Another possible relationship with AMI presents with type A, which can resemble a posterior infarction because of the tall R wave in V₁.

When there is a tachycardia present, the impulse can either travel down the Kent bundle and back up the AV node. This type of circus movement gives rise to a wide-complex tachycardia that is difficult to distinguish from ventricular tachycardia. Antidromic tachycardias can be very fast, especially in cases of atrial flutter and atrial fibrillation wherein transmission can be on a one-to-one basis.

The other type of tachycardia pattern, known as orthodromic, represents transmission of the impulse down the AV node and a return to the atria through the Kent bundle. This usually presents as a narrow-complex tachycardia and is less dangerous because the AV node still exerts its influence through the physiologic block. Therefore, the tachycardia is usually slower and more controlled than it is in antidromic tachycardia.

CLINICAL PEARL

The differential diagnosis of a tall R wave in V₁ includes:

1. Right bundle branch block
2. Posterior myocardial infarction
3. Right ventricular hypertrophy
4. WPW type A
5. Normal in adolescents and young children
In this example, we again see the delta waves typical of WPW. But what about the PR interval? Is this a short PR interval? In this case, the PR interval is about 0.12 seconds. About 12% of WPW patients do not have a short PR interval. In some cases, there can even be first-degree heart block. Why does this happen? Remember that the delta wave just hides the underlying PR interval (see Figure 10-9). If the underlying problem is a prolonged PR interval, then the patient will have a normal or prolonged PR interval when the delta wave is superimposed.

This patient has the pseudoinfarct pattern on lead aVF, and ST-T wave abnormalities that are common to WPW.

This ECG and ECG 10-3 are both examples of WPW type A; the delta wave of the QRS complex in lead V1 is positive. Type B has a negative delta wave. In general, type A is associated with accessory pathways in the left side of the heart, and type B corresponds to pathways on the right. This is not exactly true, however, because many patients have more than one pathway. The best way to find the pathway is with electrophysiologic studies.

This is yet another example of a WPW pattern. It has some interesting variations, however. Can you pick them out? Don’t come back until you’ve really looked at the ECG carefully.

First of all, the sixth complex is a PAC that is conducted mostly through the AV node. How do we know this? The delta wave is smaller in this complex. That means that most of the conduction occurred through the AV node.

Second, being an expert on P waves by now, you immediately see that the P waves are different in many of the complexes. In addition, the PR and RR intervals are different in many cases. Use your calipers. This is an example of wandering atrial pacemaker in a patient with WPW.

This ECG and ECG 10-3 are both examples of WPW type A; the delta wave of the QRS complex in lead V1 is positive. Type B has a negative delta wave. In general, type A is associated with accessory pathways in the left side of the heart, and type B corresponds to pathways on the right. This is not exactly true, however, because many patients have more than one pathway. The best way to find the pathway is with electrophysiologic studies.

This is an example of type B WPW. Note that the delta wave is negative in lead V1. There is also a nice pseudoinfarct pattern in leads III, aVF, and V1. Type B WPW is often misdiagnosed as an anterior AMI or a left bundle branch. Be careful.

Q waves are not always pathological.
**ECG 10-6** Here is another example of WPW. In this case, it is easy to see the delta wave in various leads. Once again, the PR interval is longer than expected for a WPW. Are those Q waves in leads II, III, and aVF? No. Remember that a delta wave in the inferior leads can mimic the Q waves of an inferior myocardial infarction.

**ECG 10-6** This is once again WPW, but is it type A or type B? Well, type A has the delta wave in a positive direction in lead V1. The problem is it should be positive in all of the precordials. Type B should have a negative delta wave in V1, so this is obviously not the right answer. This is type C. It starts off like type A, but then has negative deltas in the left lateral precordial leads. This is a very rare form of a rare syndrome. The important thing in these cases is to diagnose the WPW and then refer the patient to a cardiologist specializing in EPS.

**ECG 10-7** This is a different format of ECG. Note that there are calibration blocks at the start of most leads, and that there is no rhythm strip at the bottom. When you are confronted with a different format than the one that you are used to, just break it down into its components and note the leads. Although not labeled, the format for the leads is the same we are used to. If the order of the leads were different, it would have to be stated on the ECG.

This is a patient with WPW. The traditional delta wave is easy to pick out on most leads. Take a look at III and aVF. What’s going on in these leads? Well, the P wave is isoelectric in these leads, or close to it, and you don’t see it clearly. What you do see is a small QRS complex with a significant notch. The first part of the complex is not the P wave.

This is an example of an isolated intraventricular conduction delay. It is isolated because it does not cause any widening of the QRS complex, and you only see it in some leads. The reason the complex is so bizarre is that the conduction takes place aberrantly (through an abnormal pathway) and gives rise to a different morphology on the ECG. If the conduction disturbance occurred earlier, nearer the AV node, the length of the QRS complex could be widened and there would be more generalized changes in the QRS morphology. We will discuss this in greater detail when we get to bundle branch blocks.

**Clinical Pearl**

Remember, there is a difference between having a WPW ECG pattern and having the WPW syndrome. The syndrome is associated with paroxysmal tachycardias.
CHAPTER 10  ■  The PR Interval
Both this ECG and the one on the previous page are examples of type A WPW. This ECG has some interesting findings. In addition to the delta wave and ST-T wave abnormalities normally found in WPW, we have the presence of a scooping ST segment with a large upward concavity; the concave segment faces the positive part of the ECG. This concavity looks like someone just scooped it out with an ice cream scoop, as shown in Figure 10-11, doesn’t it? If you don’t see it, look at V₅ and V₆. This scooping is classic for digoxin drug therapy. The patient was on digoxin at the time the ECG was taken. This scooped ST segment occurs in all circumstances, not just WPW.

Notice the pseudoinfarct pattern in the inferior leads.

This is an example of a wide-complex tachycardia. This patient came into the emergency department with a known history of WPW, which made his management easier. Once again, please review the management of WPW and its associated tachyarrhythmias in a medical textbook.

What is the bundle branch block pattern associated with this tachycardia? It is a right bundle branch block pattern (RBBB). The slurred S waves in leads I and V₆ are clearly evident, as are the rabbit ears — or RSR’ — in V₁. Note the ECG on the next page. This is the ECG of the same patient after he was converted. The patient has a WPW type B pattern. Remember that patients with WPW type B usually have the Kent bundle on the right side. This is therefore an example of antidromic conduction leading to the RBBB pattern of the tachycardia.

A simple mnemonic is: The B of type B WPW and the R for right-sided Kent bundle are similar (Figure 10-12).

Figure 10-12: The B representing type B WPW and the R representing right-sided Kent bundle create a simple mnemonic to help remember that patients with WPW type B usually have the Kent bundle on the right side.
ECG 10-8

CHAPTER 10  The PR Interval
**ECG 10-10**  This ECG represents the patient’s ECG after he was converted from the wide-complex tachycardia seen on the previous page. If we were to have seen this ECG by itself, it would have been easy to call it a simple left bundle branch block. This is a common problem, and we have to keep the differential diagnosis of a left bundle branch block (LBBB) pattern when we look at an ECG for the first time. The delta waves are difficult to spot because they are small, but they can be seen in many leads.

Beware of any tachycardia that is over 250 BPM, especially a wide one. If the heart rate is above 250, there is usually a bypass tract associated with it. Any tachycardia at a rate of 300 has to be associated with a bypass tract, because it is much faster than any that can be transmitted through the AV node.

When the tachycardia is above 250 BPM, it is difficult to differentiate any of the components of the complex, so diagnosis will be difficult at best. The key is to remember that there could be a bypass tract involved; you want to be careful in the drugs you use to treat this patient. A drug that further slows conduction through the AV node may worsen an already poor situation. Our advice: don’t be afraid to sedate and electrically cardiovert the patient. In this situation, it is safer than the unknown problems that IV medications can induce.

**ECG 10-11** It’s pretty obvious that this patient is very tachycardic at about 280 BPM. Remember that we mentioned that WPW is associated with fast tachycardias? Well, this is another example. Note the difference between this example and those on the two previous pages. This one is a narrow-complex tachycardia, meaning that the QRS complex is less than 0.12 seconds wide. The other one is an example of a wide-complex tachycardia with a QRS complex width more than 0.12 seconds. Take a look at the next ECG. It belongs to the same patient, except that it is much slower at this point.

**ECG 10-11** This patient has a heart rate of about 280 BPM. As mentioned earlier, if the heart rate is above 250, think about a bypass tract. This patient spontaneously converted and was found to have intermittent WPW (see next ECG). This is an example of orthodromic conduction causing a narrow-complex tachycardia. There are no clearly discernible P waves, so the rhythm could be paroxysmal supraventricular tachycardia (PSVT) or 1:1 conduction of an atrial flutter. The atrial flutter would have to be slower than the traditional 300 BPM because the heart rate is about 280 BPM. There is ST depression everywhere on this ECG, which is probably subendocardial ischemia secondary to the tachycardia.

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**REMINDER**

Be careful not to confuse a normal intrinsicoid deflection with a delta wave.
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ECG CASE STUDIES

ECG 10-10

The PR Interval
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ECG 10-11
ECG 10-12 This is the same patient as that of the previous ECG after his rhythm converted. So what is going on in this one? What is the rhythm? Well, this is a very tricky rhythm to figure out because it is irregularly irregular with a lot of different looking complexes throughout. The ECG complexes with the star on top show a more positive QRS complex and some slurring at the onset. If this were your only ECG, you would have a tough call, but knowing that the patient just came out of a very fast tachycardia makes it easier to diagnose intermittent WPW. What is the rhythm? Atrial flutter with variable block. Look at the P waves in V1 marked by the vertical black lines, and it will be clearer.

ECG 10-12 This ECG shows atrial flutter with variable block, along with intermittent WPW. The atrial rate is identical to the tachycardic rate of the previous ECG, making 1:1 conduction of an atrial flutter the answer to the rhythm in that previous ECG. V1 is your only clue to the diagnosis. Look at the P waves and map them out with your calipers. The variability of the response to the P waves makes the morphology of the QRS complexes different. You can still see some of the delta component breaking through in some of the complexes. There is still some ST depression globally, which could be ischemia versus rate-related changes.

ECG 10-13 Take a really good look at the ECG below. Do you see anything unusual about the QRS complexes? This is an example of intermittent WPW. What is happening is that this patient’s impulses occasionally conduct down the AV node, and at other times down the Kent bundle. The ones that conduct normally are the ones with the asterisks. It would be difficult to pick it up from the rhythm strip, but not in leads III, aVL, and V2. In these leads, the conduction gives rise to markedly different QRS complexes.

Does it make sense that the QRS complexes of normally transmitted impulses and those transmitted through the Kent bundles are different? Sure it does! Think about the routes of transmission to the ventricles. Impulses go through two different anatomic areas to get there. They thus give rise to two different axes, because the partial transmission through the Kent bundles alters the original axis. How transmission through the Kent bundles will affect the axis depends on the anatomic location of the bundles and the size of the delta wave.

ECG CASE STUDIES continued

1. The WPW pattern is always visible in a patient with WPW syndrome. True or False.
2. The WPW pattern is never intermittent. True or False.
3. The delta wave is caused by an early impulse transmission through the Kent bundle. True or False.

Quick Review

1. True
2. False
3. True

Most patients with WPW have a concealed pathway.
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ECG 10-13

The PR Interval
ECG 10-14  This is one of our all-time favorite ECGs. It stumps about 98% of the people who try to interpret it. Can you figure it out?

It is simpler to interpret in this book because it is in the PR interval section and, in particular, the WPW section. The key to interpreting this ECG is to look at the rhythm strip. Look especially at the last two complexes. This is another example of intermittent WPW with the transition to the normal beat occurring in those last two complexes. What makes this ECG so hard to analyze is that these two complexes are at the transition points to V₄ to V₆.

The WPW in this ECG is type B. There is a pseudoinfarct pattern in lead aVL and the usual ST-T wave changes are scattered throughout. Note that the PR interval is not shortened.

Remember, to analyze an ECG you need to be thorough and methodical. Because you are at Level 3, you should already have some method established. If you do not, we recommend that you review the chapter, Putting It All Together.
A Few Words About Atrioventricular Blocks . . .

AV blocks are conduction disturbances in the AV node or the bundle of His. They cause abnormalities or prolongation of the PR interval, or in the extreme case, a complete disruption of impulse transmission to the ventricles. Don’t get them confused with bundle branch blocks. These are blocks in either the left or right bundles or their fascicles (left anterior or left posterior), or a combination of blocks.

First-degree AV block is a prolongation of the normal physiologic block. It usually occurs at the level of the AV node itself and is caused by organic heart disease. However, it can also be caused by drug toxicity (digoxin, calcium channel blockers, tricyclic antidepressants), hypercalcemia, hypothermia, and instances of increased vagal stimulation such as inferior wall myocardial infarctions.

There are two kinds of second-degree AV blocks: Mobitz I, or Wenckebach, and Mobitz II. Mobitz I is caused by a defective AV node that has a long refractory period. When the first P waves reach the node, it gets slowed down. Because the SA node is functioning normally, it starts another beat that now reaches the AV node earlier in its refractory period. The result is that the PR interval is longer because it takes that much more time to transmit. The next P reaches it earlier and takes longer to transmit, and so on. This continues until one of the P waves reaches the node at a point when it will not conduct the impulse, so it drops a QRS. This leads you to the Wenckebach pattern, which is grouped beatings with prolongation of the PR interval until one is not transmitted.

The ratio of Ps to QRSs is variable and can be 2:1, 3:1, 4:1, or more. Whenever you see grouped beating, think of Wenckebach. Some additional criteria that may help you: the R-R interval will get shorter until the dropped beat, and the distance between the QRS complexes with the dropped beats is less than twice the shortest R-R interval in the group.

Mobitz II is more dangerous and is a possible harbinger of complete block. In this type, the PR interval remains constant, but there are still intermittent dropped QRS complexes.

Note that when there is a 2:1 complex, you cannot tell if it is Mobitz I or Mobitz II. When you see such a pattern, obtain a long rhythm strip and see if there are any other groups that may help you determine the type of block. Normally, the type of block will be continuous throughout the strip.

In third-degree block, there is a complete block of the impulse at the AV node, and the P waves and the QRS complexes are dissociated from each other. Each is marching to its own drummer, so to speak. The usual atrial beat is sinus rhythm or sinus tachycardia. The ventricular beats are either junctional or ventricular in origin, and so may be either narrow or wide. There are always more P waves than QRS complexes. If there are the same number of Ps and QRS complexes, we say it is AV dissociation, not third-degree heart block. This is a fine nomenclature problem. Once again, we are not going to go into treatment, but just in case, have a temporary pacer nearby.
Prolonged PR Interval

A prolonged PR interval is one that is longer than 0.20 seconds. When you are confronted by a prolonged PR interval, ask yourself a few questions:

1. Are all of the PR intervals and P waves the same? If they are, you are probably dealing with first-degree heart block. If they are not, you have to think of premature atrial complexes, wandering pacemaker, multifocal atrial tachycardia, or another type of block.

2. Do the PR intervals vary consistently?
   (a) Are all of the Ps the same?
   (b) Are the PR intervals progressively lengthening?
   (c) Do you have grouped beating (Figure 10-13)?
   (d) Are the Ps and QRSs dissociated? If the P waves are all different, you are definitely talking about wandering pacemaker or multifocal atrial tachycardia (MAT). If the Ps are the same, start thinking about what type of block is present. Is it Mobitz I or II? Is it third-degree AV block or AV dissociation? Should I get a rhythm strip? Finally, and most importantly, what does the patient look like? You need to put it all together to obtain the right answer.

Look at some examples on the following pages and see if you can come up with the right answer. By the way, if you disagree with us on any of the ECGs, that’s OK. You’re wrong, but it’s OK. (Just kidding.) Remember, there are always disagreements about interpretation . . . even between your own interpretations on different days. This is a scientific fact verified in multiple studies.

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ECG 10-15 How long is the PR interval? It is a little over 0.20 seconds. This is an example of first-degree heart block. The P waves show some left atrial enlargement in V_1, but otherwise the Ps aren’t remarkable. There is some slight PR depression in leads III and aVF, but these are not found in any other leads, so pericarditis is probably not present.

Remember, at this point you should only be looking at the sections of the ECG that we have reviewed in detail: the P waves and the PR intervals. When you revisit this ECG at Level 3, you will be in for some other juicy findings.

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ECG 10-15 So what do you want to do with this patient? He just has some mild first-degree heart block, right? WRONG. This patient has changes consistent with an AMI in the inferior leads, and possibly involving the right ventricle. The patient has significant Q waves in II, III, and aVF, with ST segment elevation, as well. In addition, the patient has ST depression in aVL. There is some ST elevation in V_1 to V_5, with poor RR progression. The ST segment elevation in V_1 with an inferior AMI is classic for right ventricular involvement. Right-sided leads are recommended even though the ST elevation in V_1 is only about 0.5 mm.

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Figure 10-13: Grouped beats.
ECG 10-16 Here is another example of first-degree heart block. What we have tried to do in the three representative examples on first-degree block is to show you a progression of PR prolongation. Remember that this interval can vary significantly.

Did you evaluate the P waves? If you did, you saw the P-pulmonale that is present on this ECG. When you continue to go through this book, try to evaluate the ECG for all of the items covered previously. That way, when you reach the end of the book, you will be better prepared to go on to Level 3 if you wish.

ECG 10-17 The right half of the ECG below shows a long first-degree heart block. How long is the PR interval in this ECG? About 0.48 seconds, which makes this a very long PR interval. Now, let’s look at the first half of the ECG. The first complete complex is similar to the ones at the end of the ECG and represents a normal complex for this patient. Then there is a much longer pause between the first and second complexes. In addition, this second complex has a shorter PR interval, making you think that this was not normally conducted. It appears to be a sinus escape beat. The pause between the second and third complexes is again long, but this time the PR interval for the third complex is normal. This is not a sinus arrhythmia as it encompasses only one complex.

ECG 10-17 What kind of block does this patient have? It is definitely a right bundle branch block with slurred S waves in V5 and an RSR’ complex in V1. The axis is in the extreme right quadrant. It is a wide block and has some bizarre ST-T wave abnormalities. Look at V1 and V2. Can you make any statements about the ST depression and the T waves? Well, you can say that the ST segments are depressed and that the T waves are concordant; they are in the same direction as the last part of the QRS complex. Could this represent a posterior AMI? Sure it could. You would need some clinical correlation and an old ECG to tell definitively.

REMINDER

AV blocks and bundle branch blocks are different.
ECG 10-18 For those of you who were astute enough to pick it up, this is the same patient that was reviewed in the first-degree heart block section earlier. Now the rhythm is completely different. Do you see any grouping on this ECG? Yes, there are two full groups of three complexes each. Now, let’s look at the PR intervals . . . are they the same? No, they seem to get longer in each succeeding complex. In addition, the RR intervals are shorter in each successive complex in a set. This is an example of Mobitz I or Wenckebach second-degree heart block in someone with a prolonged PR interval. Do you see the P wave of the dropped beat? No, because that P is buried inside the T wave of the third QRS complex. Whenever you see a grouped beating situation, you have to think of second-degree heart block!

ECG 10-18 What is the differential diagnosis of tall R waves in the right precordial leads?

1. Normal young children and adolescents
2. Right bundle branch block
3. Wolff-Parkinson-White syndrome
4. Right ventricular hypertrophy
5. Posterior myocardial infarction

How do you tell the difference between them? Look at the company they keep! Is the patient young? Do you have slurred S waves or delta waves? Is there any evidence of right atrial enlargement (RAE) or RAD? Does the patient look like a chronic obstructive pulmonary disease (COPD) patient or one having an AMI?

ECG 10-19 First of all, don’t panic. This is yet another ECG format, and it is not much different from the ones you are used to. If you look at the top four strips and mentally erase the other two, you have the format that we usually use in this book. This format is useful in that you have three rhythm strips, and all of them are occurring simultaneously. (Note that the same beats are reflected at the same moment in time in all six strips.) This multiple-rhythm-strip capability is very helpful in studying rhythms.

Do you see groupings? Yes, they occur in sets of two complexes. Are there P waves? Yes. Are the PR intervals getting longer? Yes. Are there nonconducted P waves? Yes, the third one in each group. What is the rhythm? Mobitz I or Wenckebach second-degree heart block. Piece of cake! By the way, the patient also has first-degree heart block.

ECG 10-19 Take a look at the third P wave in each set, and look at them in all of the leads. In which lead is it easiest to see them? Leads aVL, V1, and V2. Can you figure out why? Because these are the leads in which the T wave is the flattest or most isoelectric. The P wave can come out in all of its glory in these leads. This concept is helpful when you order a rhythm strip. If you are looking for P waves, order a strip that includes those leads. That is what we have done in this case.

When using a rhythm strip, use the leads that will yield the most useful information. You can find out which ones by getting a standard 12-lead.
**ECG 10-20** Let’s analyze this ECG. The first thing to do is to find P waves that you can clearly identify. Now place your calipers between two of these in the first part of the ECG. Walk your calipers back and forth, identifying the rest of the P waves in the ECG. When you do that on the section covered by the blue line, you notice that the first eight beats are on time and as scheduled. The beats marked by green arrows are of a different morphology and timing than the rest. Then they go back to the same P wave morphology as the first group but at a different rate. What occurred is that two beats from an ectopic source fired and reset the underlying sinus node rate. Now look at the association between the P waves and the QRS complexes. Is there any association? NO. This is a third-degree AV block.

**ECG 10-21** This is an example of third-degree heart block. Notice that the sinus beat is much faster than the ventricular beats. The ventricular rhythm appears to be a junctional escape beat with a rate of about 35 BPM. Note that you cannot rule out a ventricular escape rhythm in this case, but the morphology is suggestive of a supraventricular origin.

Look at the first two complexes. Could you have diagnosed the block from these two? You could if you were looking closely at the two humps on the T waves and you noticed that the two T waves are not identical. Whenever you see two humps on a T wave you should ask yourself, “Could this be a superimposed P wave?” Use your calipers and see if it falls on the middle, or at a multiple, of the P to P interval. If the answer is yes, then it is a superimposed P wave.

**ECG 10-20** The patient has an underlying RBBB morphology with slurred S waves in I and aV_{1} and RSR’ complex in V_{6}. The T waves are symmetrical and somewhat peaked in leads V_{3} and aVF; these Ts are as tall or taller than the QRS complexes accompanying them. Whenever you see Ts like these, especially when there is an underlying block, you should think about hyperkalemia. We don’t know clinically if this patient has hyperkalemia, but you’d better think about it and treat it if it is present.

Hyperkalemic T waves are only classically tall, peaked, and narrow in 22% of cases.

**ECG 10-21** This ECG, in addition to the beautiful example of third-degree heart block, shows a bifascicular block. The patient has a RBBB and left anterior hemiblock (LAH) pattern on his ECG. If you can just imagine that the patient has significant myocardial damage to the conduction system, enough to cause a bifascicular block, then the amount of ischemia or infarction needed to complete the block would be very little. Remember, if you have any patient with ischemia and a bifascicular block you need to keep the possibility of a complete AV block in mind. What should you do with this patient? You should have an external pacemaker available at the bedside, just in case.
CHAPTER 10  ■  The PR Interval

ECG CASE STUDIES CONTINUED

I  aVR  V₁  V₄
II  aVL  V₂  V₅
III  aVF  V₃  V₆

ECG 10-20
ECG 10-22  What a mess! First things first: can you identify the P waves? You should be able to see them clearly on the rhythm strip. Use your calipers and map them out. Are they regular? Yes. Do they have any association with the QRS complexes? No. Are there more P waves than QRS complexes? Yes. This is an example of third-degree heart block.

Now, turn your attention to the QRS complexes. First of all, how fast is it going? The ventricular rate is about 20 BPM. Are the QRS complexes wide or narrow? Really wide. A wide-complex rhythm at a rate this slow is a ventricular escape rhythm known as an idioventricular rhythm.

ECG 10-22  We really try to stay away from treatment in this book, but occasionally we will make a comment for you to think about. If you had the choice of using either atropine or an external pacemaker on this patient, which would you choose? The ACLS guidelines suggest the intervention sequence to be atropine 0.5–1.0 mg IV, then a transcutaneous pacemaker if available. Atropine is fast and easy to administer, if you have intravenous access. However, the resultant rhythm after atropine administration is variable and difficult to predict. Transcutaneous pacemaking (if available quickly and if capture is achieved) may offer an added level of control over the subsequent heart rate and may be preferable in certain clinical scenarios.

**REMEMBER**

Remember that you can have multiple rhythm abnormalities on the same strip. For example, ECG 10-22 contains a sinus tachycardia as the underlying atrial rhythm and an idioventricular rhythm. These together form a third-degree heart block because the atrial rate is faster than the ventricular rate. All of this information can be put together into the correct and most complete label that you can give this abnormality: a paroxysmal atrial tachycardia with block. In this case, the block leads to the idioventricular rhythm.
CHAPTER 10  ■ The PR Interval
1. The PR interval represents the time frame from the beginning of atrial depolarization to the end of ventricular repolarization. True or False.

2. The differential diagnosis of PR depression includes:
   A. Normal variant
   B. Pericarditis
   C. Atrial infarction
   D. All of the above
   E. None of the above

3. If the PR interval in lead II is 0.18 seconds long and in V₁ it is 0.22 seconds long, what is the true PR interval?
   A. 0.18 seconds long
   B. 0.20 seconds long
   C. 0.22 seconds long
   D. 0.24 seconds long
   E. None of the above

4. The differential diagnosis of a short PR interval includes:
   A. Retrograde junctional P waves
   B. Lown-Ganong-Levine syndrome
   C. Wolff-Parkinson-White syndrome
   D. All of the above
   E. None of the above

5. Which of the following is incorrect when discussing WPW syndrome:
   A. Shortened PR interval is always present
   B. Widened QRS complex ≥ 0.11 seconds
   C. Delta waves are present
   D. Associated with ST-T wave abnormalities
   E. Associated with paroxysmal tachycardias

6. If you see a wide-complex tachycardia, you can assume it is secondary to WPW syndrome. True or False.

7. Q waves in the inferior leads of patients with WPW are always caused by a prior myocardial infarction. True or False.

8. AV blocks and bundle branch blocks are the same. This is just a nomenclature issue. True or False.

9. Grouped beating that has progressively prolonging PR intervals until a ventricular complex is dropped is:
   A. First-degree heart block
   B. Mobitz I second-degree heart block, or Wenckebach
   C. Mobitz II second-degree heart block
   D. Third-degree heart block
   E. AV dissociation

10. If the sinus rate is 100 BPM, the ventricular rate is 38 BPM, and they are dissociated, we refer to this rhythm as:
    A. AV dissociation
    B. Third-degree heart block
    C. Both A and B are correct
    D. None of the above