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# Abnormalities of the P Wave and PR Interval

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## The Normal P Wave

The P wave represents atrial depolarization, as well as the start of the cardiac electrical cycle. Atrial activation is normally initiated in or immediately around the sinoatrial (SA) node in the superior right atrium and proceeds toward the left atrium and the atrioventricular (AV) node. The duration of the normal P wave is less than 0.12 seconds (s), and the amplitude should be no greater than 0.25 millivolts (mV) in the frontal-plane leads and 0.15 mV in the transverse-plane leads.

The normal frontal-plane P-wave axis may be anywhere between 0 and +75° but typically ranges from +45 to +60°. The normal P wave is always positive in leads I and II and is usually positive in lead aVF. It is always negative in lead aVR. Regarding the transverse-plane leads, the P wave is typically biphasic in the right precordial leads (i.e. V1 and sometimes V2). The initial positive deflection represents right atrial activation and is followed by a negative deflection, as left atrial activation dominates the terminal portion of the wave and proceeds away from surface leads V1 and V2. The remainder of the precordial leads should display a positive P wave.

## The Abnormal P Wave

### Atrial Abnormality

The terms *right* and *left atrial abnormality* are preferred to such labels as right and left atrial enlargement, hypertrophy, overload, and conduction delay, as the P-wave changes attributed to these states are not specific for just one, and substantial crossover exists (Table 7.1.1) [1]. Differentiation of these conditions based on the surface electrocardiogram is fraught with error and should generally be avoided. Numerous studies have examined the sensitivity and specificity of various criteria for right and left atrial *enlargement* using a reference standard of either echocardiography or cardiac magnetic resonance imaging. These studies have uniformly shown poor sensitivity but good specificity for right atrial enlargement, whereas criteria for left atrial enlargement have reasonable sensitivity and specificity (Table 7.1.2) [2, 3, 4, 5].

Because the SA node resides in the right atrium, this chamber is activated first and is represented by the initial portion of the P wave. Subsequent left atrial activation comprises the terminal portion of the P wave. Consequently, right atrial abnormality affects the early P wave, while left atrial abnormality affects the latter portion. Of the two, only left atrial abnormality should prolong the *duration* of the P wave (Figure 7.1.1).

#### Right Atrial Abnormality

Right atrial abnormality produces a tall (greater than 0.25 mV), peaked P wave in lead II and may shift the P-wave axis rightward (greater than +75°) (Figure 7.1.2a). This results in a P wave that is larger in lead III than in lead I. In addition, the initial positive P-wave deflection in leads V1 and V2 is amplified (greater than 0.15 mV) (Figure 7.1.2b).

#### Left Atrial Abnormality

Left atrial abnormality manifests as P-wave prolongation (greater than or equal to 0.12 s) with notching of the P wave in leads with similarly directed right and left P wave forces (i.e. those without biphasic P waves). A delay of greater than 0.04 s between the peaks of the notched P wave increases the specificity of this finding to near 100% (Figure 7.1.3a) [4]. In lead V1, the increased left atrial forces pull the terminal (negative) portion of the P wave further downward, resulting in an amplitude deeper than 0.10 mV

*Electrocardiogram in Clinical Medicine*, First Edition. Edited by William J. Brady, Michael J. Lipinski, Andrew E. Darby, Michael C. Bond, Nathan P. Charlton, Korin Hudson, and Kelly Williamson.

**Table 7.1.1** Atrial abnormality may result from atrial volume/pressure overload, hypertrophy, enlargement, or conduction delay.

| Right atrial abnormality | Left atrial abnormality |
|---|---|
| • Acute pulmonary illness<br>– Pulmonary embolism<br>– Asthma exacerbation<br>• Chronic pulmonary disease (e.g. COPD)<br>• Chronic pulmonary hypertension<br>• Structural heart disease<br>– Atrial septal defect<br>– Ebstein anomaly<br>– Pulmonic stenosis<br>– Tricuspid stenosis | • Cardiomyopathy<br>– Dilated<br>– Hypertrophic<br>– Restrictive<br>• Coronary artery disease<br>• Systemic hypertension (chronic)<br>• Valvular disease<br>– Mitral stenosis or insufficiency<br>– Aortic stenosis or insufficiency |

COPD = chronic obstructive pulmonary disease.

**Table 7.1.2** Diagnostic criteria for atrial abnormality.

*Right Atrial Abnormality*
- Tall (>0.25 mV), peaked P waves in frontal-plane leads (best seen in II, III, aVF)[a]
- P-wave amplitude >0.15 mV in lead V1 and/or V2
- P-wave duration <0.12 s (normal)
- May have P-wave axis +75° or further rightward

*Left Atrial Abnormality*
- Wide P waves (≥0.12 s)
- Notched P waves in frontal-plane leads (best seen in I, II, aVL, V5, V6; inter-peak distance >0.04 s)[a]
- Prominent terminal negative P-wave deflection in lead V1 (>0.10 mV, >0.04 s)
- May have P wave axis +15° or further leftward

*Biatrial Abnormality*
- Components of both right and left atrial abnormality
- Increased P-wave amplitude (>0.25 mV) and width (≥0.12 s) in frontal-plane leads
- Peaked P wave (positive amplitude >0.15 mV) with prominent terminal negative deflection in lead V1 (>0.10 mV and/or >0.04 s)
- Wide (≥0.12 s), notched P waves in frontal-plane or lateral transverse-plane (V5 or V6) leads with tall, peaked P wave (positive amplitude >0.15 mV) in V1 and/or V2

[a] Most specific criterion (sensitivity of criteria vary widely among studies).

and an increase in the duration of this negative deflection to greater than 0.04 s (Figure 7.1.3b) [6]. The P-wave axis is directed leftward (generally left of +15°), producing a P wave in lead I that is larger than that in lead III.

### Biatrial Abnormality

Biatrial abnormality manifests as a combination of the findings seen with right atrial abnormality and left atrial abnormality. The limb leads may show a prolonged (greater than or equal to 0.12 s) P wave with increased amplitude (greater than 0.25 mV) (Figure 7.1.4). Another diagnostic criterion for biatrial abnormality requires a peaked P wave taller than 0.15 mV in lead V1 with a terminal portion deeper than 0.10 mV and/or wider than 0.04 s. Finally, a wide, notched P wave in the limb leads or lateral transverse-plane leads (V5 and V6), in combination with a tall (>0.15 mV), peaked P wave in the right precordial leads, also qualifies as biatrial abnormality.

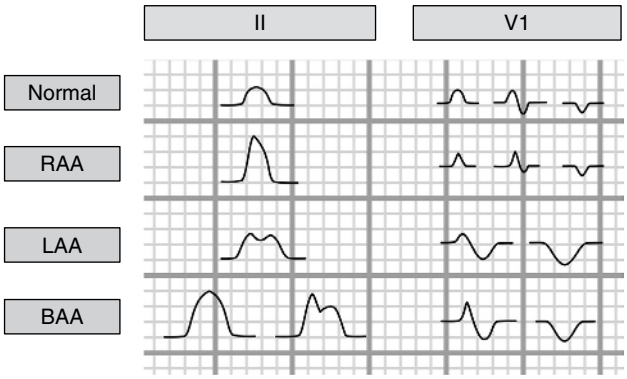



**Figure 7.1.1** Atrial abnormality. Note the various P-wave morphologies seen with right, left, and bi-atrial abnormality in leads II and V1. Normal P-wave morphologies are also provided for comparison. RAA = right atrial abnormality, LAA = left atrial abnormality, BAA = biatrial abnormality.

### Ectopic Atrial Foci

Atrial pacemakers distant from the SA node produce P waves of varying axis and morphology. These may be positive, biphasic, or negative in a given lead, depending on the particular location of the focus. If the pacemaker is low in the atria, the P wave will likely be negative in leads II, III, and aVF due to retrograde conduction away from the inferior leads (Figure 7.1.5).

### Biphasic P Waves

Biphasic P waves are normal in leads V1 and V2 and may also be seen less commonly in leads III, aVL, and aVF; however, they should be completely positive or negative in the remaining leads (Figure 7.1.6). In the case of blocked conduction through Bachmann's bundle (which connects the right and left atria), an impulse generated in the superior right atrium in or around the SA node proceeds toward the AV node, then must cross to the left atrium and travel superiorly to activate the remainder of that chamber. This is known as interatrial block and often yields wide (at least 0.12 s) and biphasic (positive–negative) P waves in leads II, III, and aVF, as the left atrium is activated in a retrograde fashion away from those inferior leads [7]. Wide, notched P waves result from partial interatrial block in which conduction through Bachmann's bundle is slowed but not blocked entirely (Figure 7.1.7) [8, 9].

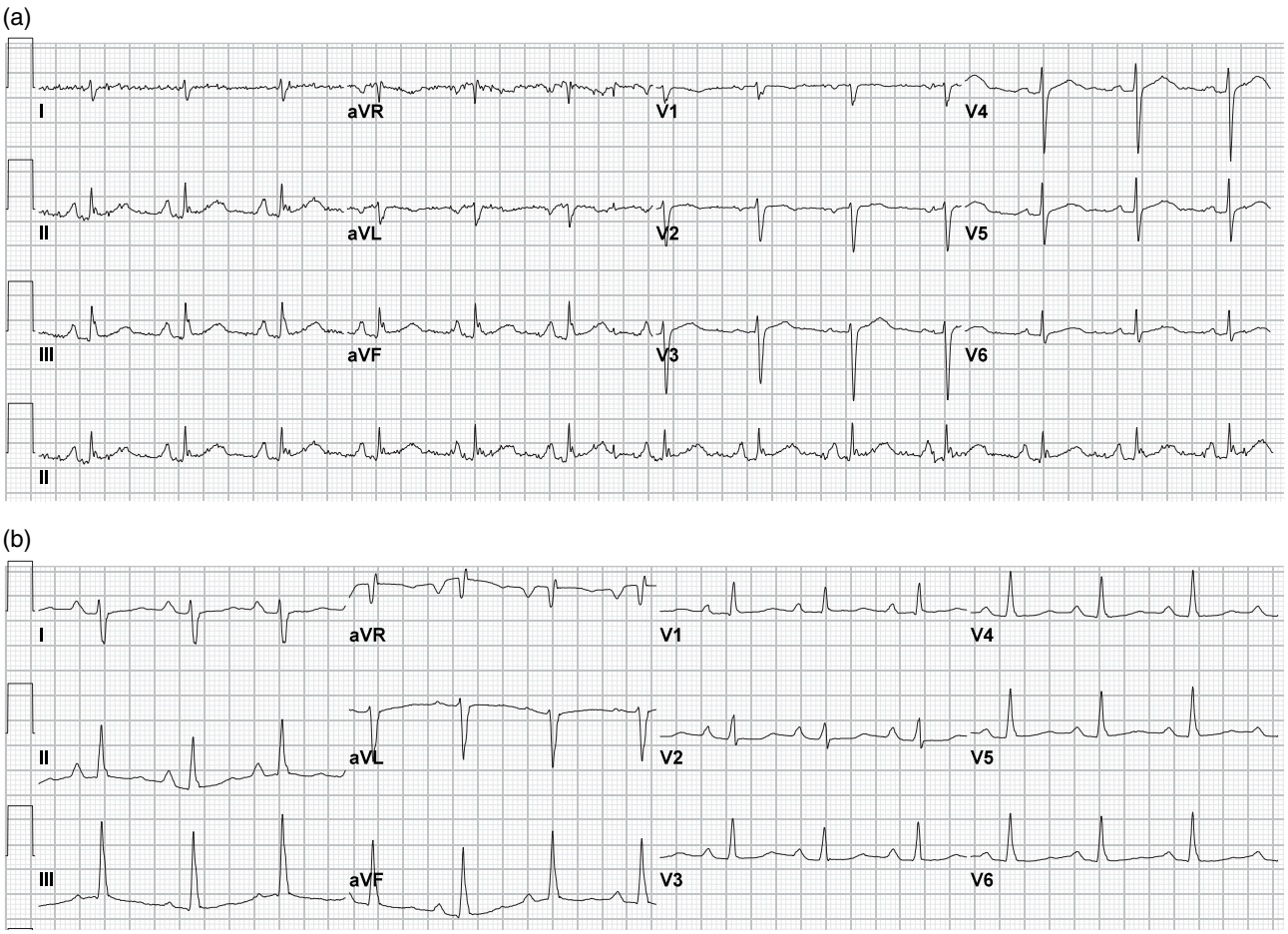



**Figure 7.1.2** Right atrial abnormality. (a) Note the tall P waves ("P pulmonale" morphology) in lead II. Specifically, the amplitude of the upright P wave is >0.25 mV. In addition, the P-wave amplitude in lead III is greater than that in lead I, indicating a rightward shift of the P-wave axis. (b) Note the entirely positive P waves in leads V1 and V2 that are greater than 0.15 mV in amplitude.

### Multifocal Atrial Tachycardia

Multifocal atrial tachycardia (MAT) is a chaotic atrial tachydysrhythmia that manifests with multiple (at least three) different P-wave morphologies that represent various atrial foci responsible for impulse generation. By definition, the ventricular rate must be greater than 100 bpm. Without a dominant focus in the SA node or atrial tissue, the rhythm may arise from anywhere in the atria, which leads to variability in the PP, PR, and RR intervals. This rhythm is most commonly seen transiently during periods of acute illness in patients with chronic lung disease (Figure 7.1.8). In order to differentiate this rhythm from atrial fibrillation, which is commonly seen in these same patients, an isoelectric baseline must be confirmed. If an isoelectric baseline is identified, and more than three P-wave morphologies exist, then the rhythm is a multifocal atrial rhythm. If no isoelectric baseline is present and it is difficult to identify distinct P waves, then the rhythm more likely represents atrial fibrillation.

## The Normal PR Interval

The PR interval represents the time from the start of atrial activation to the onset of ventricular activation and can be shortened or lengthened by factors that affect the speed of conduction from the atria to the ventricles (Figure 7.1.9). It is measured from the start of the P wave through the start of the QRS complex and, therefore, includes both the P wave and the PR segment. The normal PR interval is 0.12–0.20 s (Figure 7.1.6). Infants and small children may have normal conduction with a PR interval shorter than this, though it is typically greater than 0.09 s.

## The Abnormal PR Interval

### Short PR Interval

An abnormally short PR interval results from conditions that allow faster conduction from the atria to the ventricles. This can occur with accessory conduction

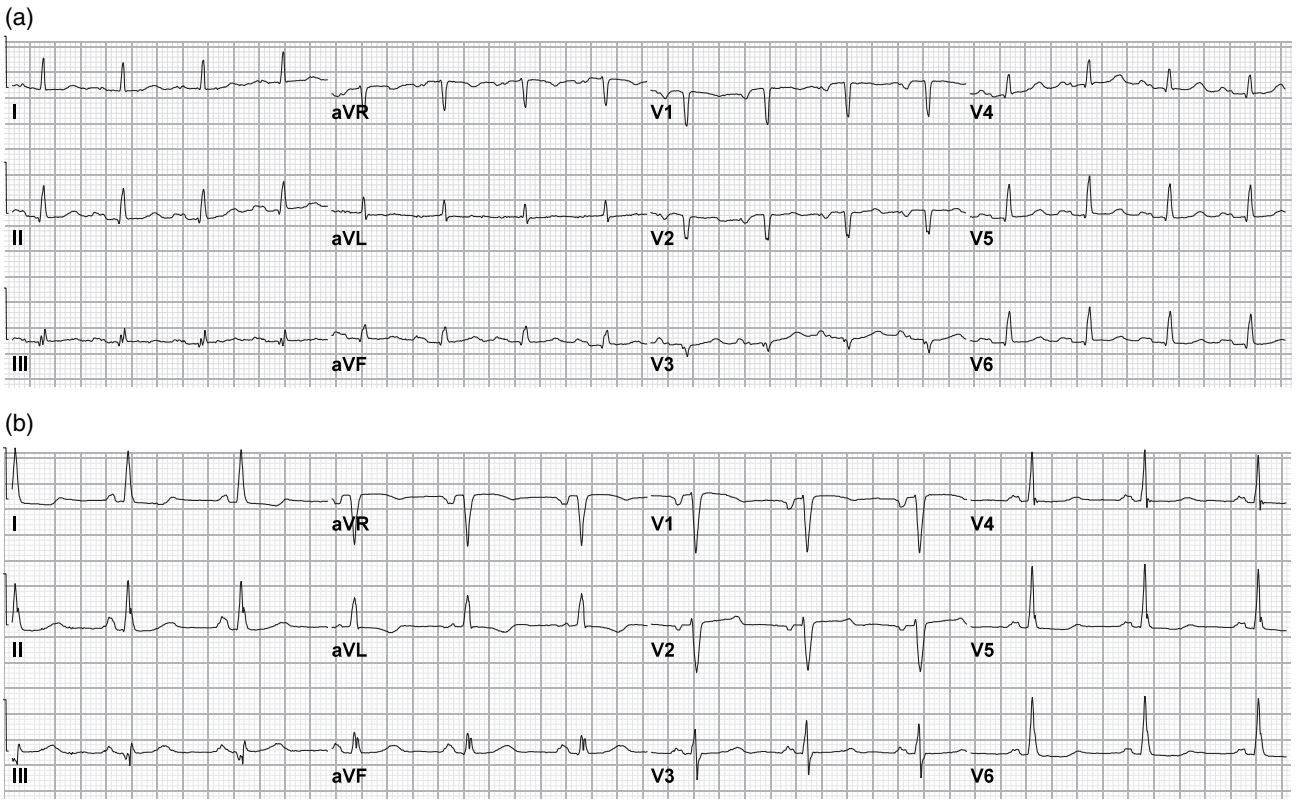



**Figure 7.1.3** Left atrial abnormality. (a) Note the notched P wave ("P mitrale" pattern) in lead II. The two humps of the notched P wave are greater than 0.04 sec apart, which improves the specificity of this finding. (b) Note the deep and entirely negative P waves in lead V1. Some of the P waves in other leads are notched, but the distance between the humps is not greater than 0.04 sec, rendering this finding less reliable in confirming left atrial abnormality.

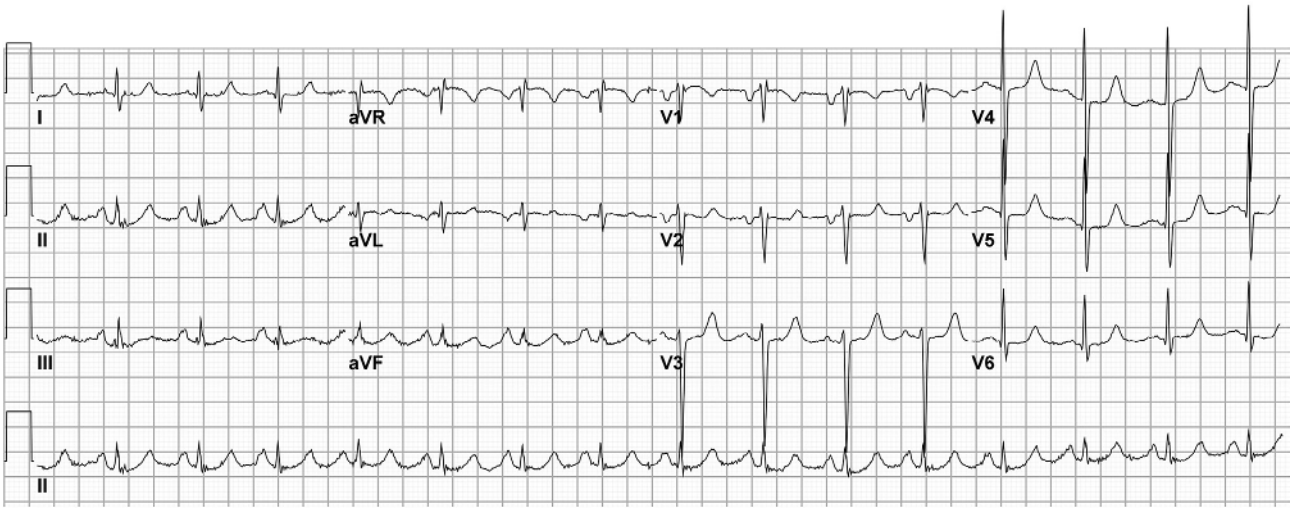



**Figure 7.1.4** Biatrial abnormality. 70-year-old female with a history of diastolic heart failure presented with acute-on-chronic dyspnea. Biatrial enlargement was confirmed by echocardiography. Note the tall and wide P waves in lead II and the deep, almost entirely negative P waves in lead V1.

pathways that facilitate ventricular preexcitation by shortcircuiting normal conduction through the AV node or with exercise-induced sinus tachycardia. Other causes of a short PR interval likely to be seen on a resting ECG include junctional rhythms and ectopic atrial rhythms in which the site of atrial activation is in close proximity to the AV node (Table 7.1.3, Figure 7.1.10).

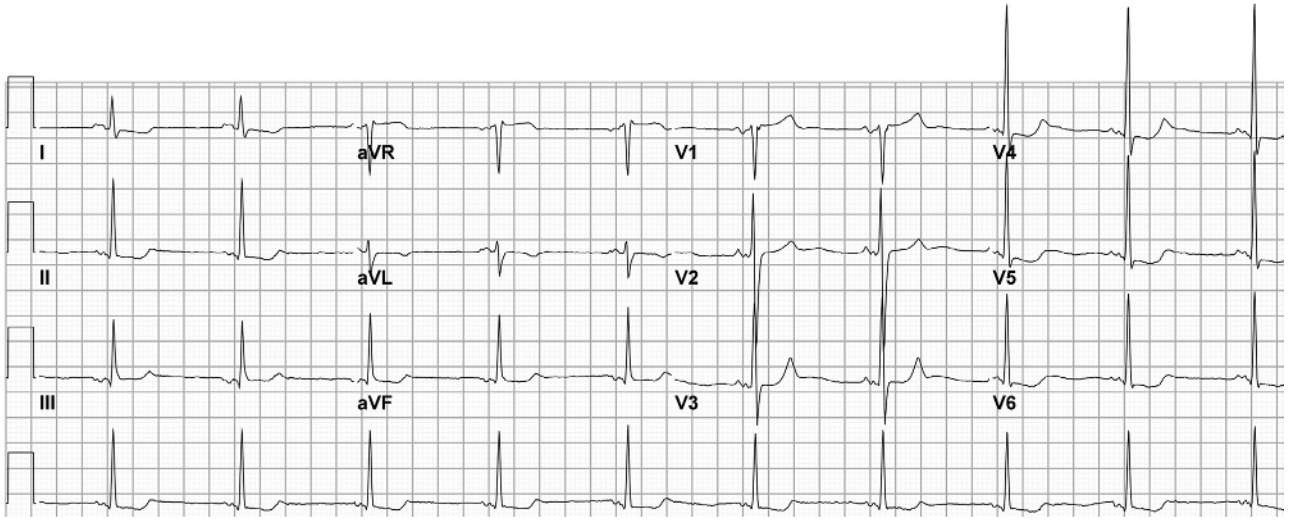



**Figure 7.1.5** Ectopic atrial rhythm. Note the atypical P-wave morphology and axis. All of the P waves have the same morphology, suggesting that they all originate from the same ectopic focus.

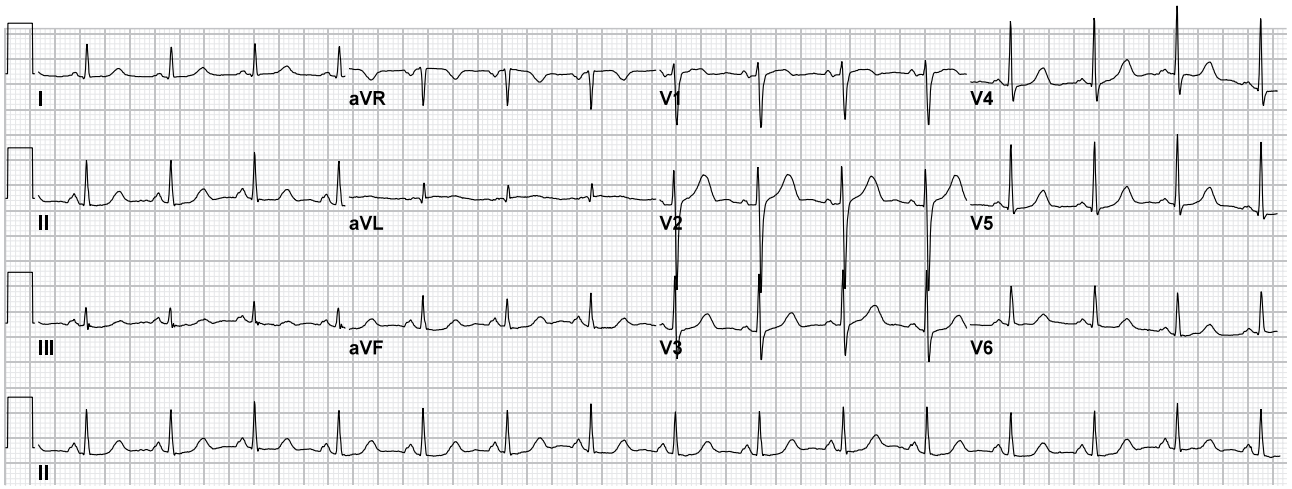
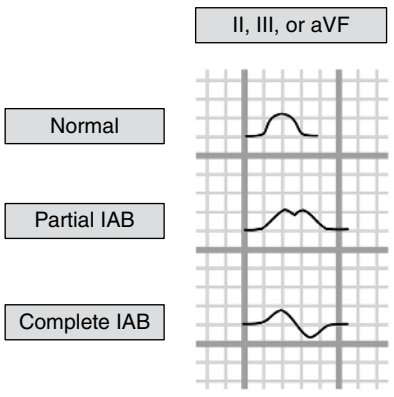



**Figure 7.1.6** Normal P-wave morphology. Note the biphasic P waves in lead V1 and the negative P waves in lead aVR. Biphasic P waves would also be considered normal in leads V2, III, aVL, and aVF. The P wave duration is less than 0.12 sec, and the amplitude neither exceeds 0.25 mV in the frontal-plane leads nor 0.15 mV in the transverse-plane leads. Additionally, the PR interval is normal.

**Figure 7.1.7** Interatrial block. Delayed conduction from the right atrium to the left atrium may manifest as changes in the P-wave morphology of the inferior leads. Partial interatrial block results in a prolonged (≥0.12 sec) P wave, often notched. Complete interatrial block causes retrograde atrial activation after the normal impulse reaches the tissue near the AV node. This retrograde activation is represented as a negative terminal deflection in the inferior leads. IAB = interatrial block.



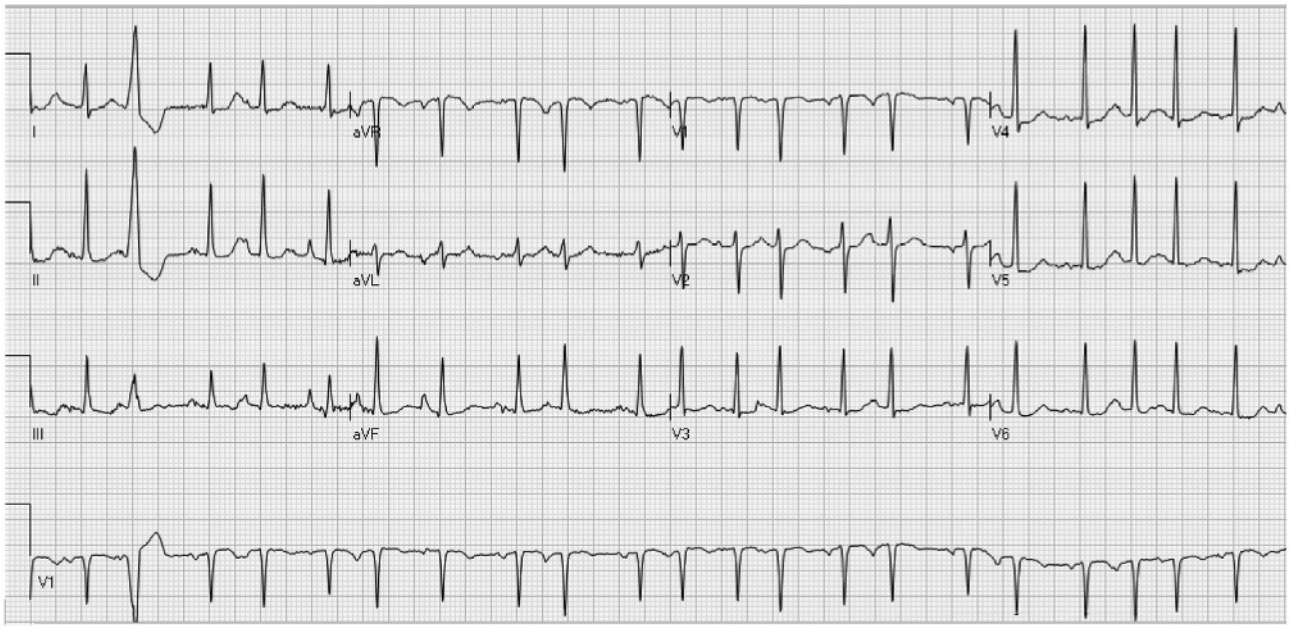



**Figure 7.1.8** Multifocal atrial tachycardia (MAT). Note the irregular rhythm and multiple P-wave morphologies (at least 3 required for diagnosis).

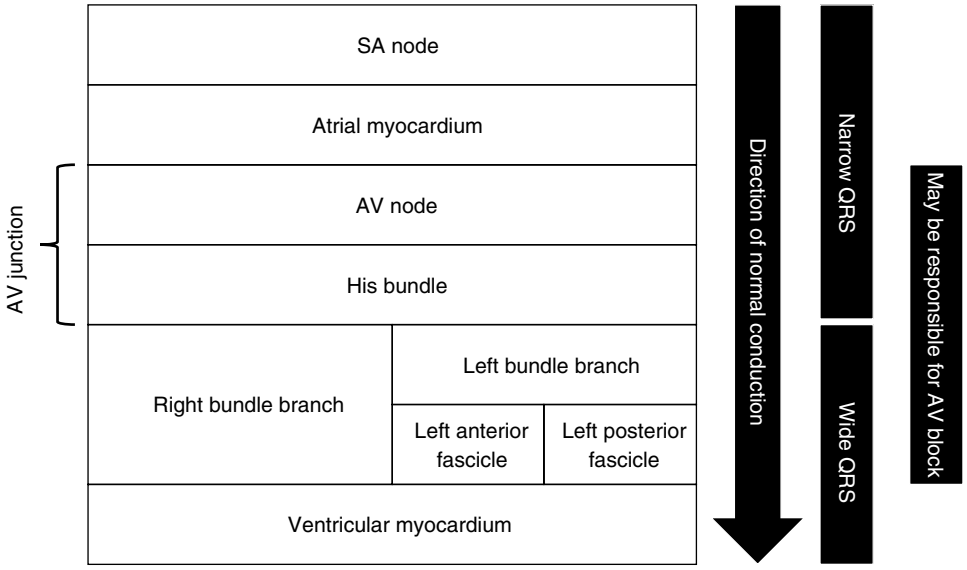



**Figure 7.1.9** Normal atrioventricular conduction. Note that generation of an impulse from structures including and proximal to the His bundle can result in a narrow QRS complex (if no conduction delay is present distally), while those that originate distal to the His bundle produce a wide QRS complex. The "AV junction" refers to the AV node and His bundle. AV block can result from conduction delay or failure at any of the structures in the conduction system between the atrial and ventricular myocardia.

**Preexcitation**

Accessory pathways that allow for rapid conduction of atrial impulses to the ventricular myocardium result in a syndrome of ventricular preexcitation. One such example is the Wolff-Parkinson-White (WPW) pattern in which the so-called "bundle of Kent" allows for a portion of the ventricle to be activated sooner than the remainder of the ventricle that is dependent upon depolarization via the AV-His-Purkinje system. This yields a slurred QRS upstroke, known as a *delta wave,* which results in a shortened PR interval and (sometimes) a prolonged QRS interval (Figure 7.1.11). Individuals exhibiting these findings on a resting ECG are at risk for tachydysrhythmias.

**Table 7.1.3** Causes of abnormal PR interval.

| Short PR interval | Long PR interval[a] |
|---|---|
| • Exercise-induced sinus tachycardia<br>• Junctional rhythms<br>• Preexcitation<br>• Thyrotoxicosis [10]<br>• Pheochromocytoma [11]<br>• Pompe disease [12]<br>• Fabry disease [13] | • Acute MI<br>– Anterior (usually permanent)<br>– Inferior (usually temporary)<br>• Congenital heart disease<br>– Atrial septal defect<br>– Ebstein anomaly<br>• Connective tissue disease<br>– Lupus (neonatal or adult) [14]<br>– Marfan syndrome<br>– Scleroderma<br>• Hyperkalemia<br>• Hypothermia<br>• Hypothyroidism<br>• Iatrogenic<br>– Post ablation<br>– Post valve surgery<br>• Infection<br>– Endocarditis<br>– Lyme carditis [15]<br>– Myocarditis<br>– Toxoplasmosis<br>• Infiltration<br>– Amyloidosis<br>– Sarcoidosis<br>• Medications<br>– Amiodarone<br>– Beta-adrenergic receptor antagonists<br>– Calcium channel antagonists[b]<br>– Class IA and IC sodium channel antagonists<br>– Digoxin<br>• Neuromuscular disease (inherited)<br>– Kearns-Sayre syndrome<br>– Myotonic dystrophy [16]<br>• Valve disease, calcific (aortic & mitral) |

[a] The items listed can produce AV blocks of varying degrees.
[b] Non-dihydropyridine agents, such as diltiazem or verapamil
AV = atrioventricular; MI = myocardial infarction.

Similar preexcitation states are facilitated by Brechenmacher fibers, which extend from atrial tissue to the His bundle, and by James fibers, which serve as a bridge from the proximal AV node to the distal AV node. These both produce a short PR interval but no delta wave or widened QRS complex because the ventricular myocardium is ultimately activated through the His-Purkinje system; in such cases, at best, only the AV node is bypassed (Figure 7.1.12).

#### Ectopic Atrial Foci

Atrial impulses originating from ectopic sites distant from the SA node and near the AV node may also produce a short PR interval, simply due to the shorter distance for the impulse to travel prior to reaching the AV node. These are often labeled as having come from the AV junction (Figure 7.1.10).

#### Junctional Rhythms

If the electrical impulse is initiated within the AV junction (AV node or His bundle), conduction proceeds in both directions simultaneously – back up toward the atria and down toward the ventricles. If atrial activation occurs before ventricular activation, then a P wave may be seen, but it will appear immediately before the QRS complex and will likely be inverted, as the direction of conduction is away from the surface ECG leads. In many cases, the P wave is entirely obscured by the more dominant QRS complex, as ventricular activation often begins at the same time, or slightly before, atrial activation. In some cases, a P wave may even be seen in the terminal portion of the QRS complex, as the retrograde activation of the atria leads to depolarization, which occurs just after ventricular depolarization and in a direction away from the surface ECG leads (Figure 7.1.13). Certain types of junctional tachycardias produce similar findings, but clues other than the PR interval may be more helpful in guiding the clinician to these diagnoses.

### Long PR Interval

#### First-Degree Atrioventricular Block

First-degree AV block is defined by a PR interval longer than 0.20 s with an otherwise normal appearing P wave of sinus origin. In addition, every P wave must be followed by a QRS complex, and every QRS complex is preceded by a P wave (Figure 7.1.14a and b). A variety of medical conditions and therapeutic agents are associated with PR prolongation (Table 7.1.3), and many of these conditions and medications may also lead to higher-degree AV blocks, which are discussed below. Asymptomatic and isolated first-degree AV block is of no immediate clinical significance.

#### Second-Degree Atrioventricular Block

Second-degree AV block differs from first-degree AV block in that not every P wave is followed by a QRS complex. There are two principal types of second-degree AV block – type I (Wenckebach or Mobitz type I) and type II (Mobitz type II).

In type I second-degree AV block, the PR interval progressively lengthens from beat to beat until a single QRS complex is dropped (Figure 7.1.15). That is, there is a P wave that is not followed immediately by a QRS complex.

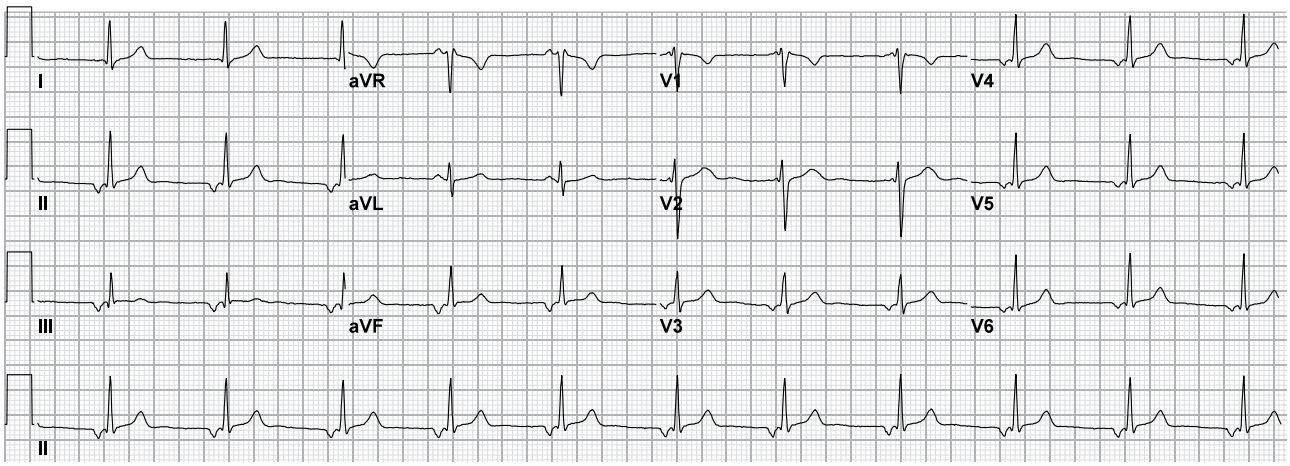



**Figure 7.1.10** Ectopic atrial rhythm with short PR interval. This 16-year-old patient presented with a syncopal episode and was found to have an ectopic atrial rhythm with a short PR interval (0.10 sec). Repeat ECG several days later showed normal sinus rhythm with a normal PR interval.

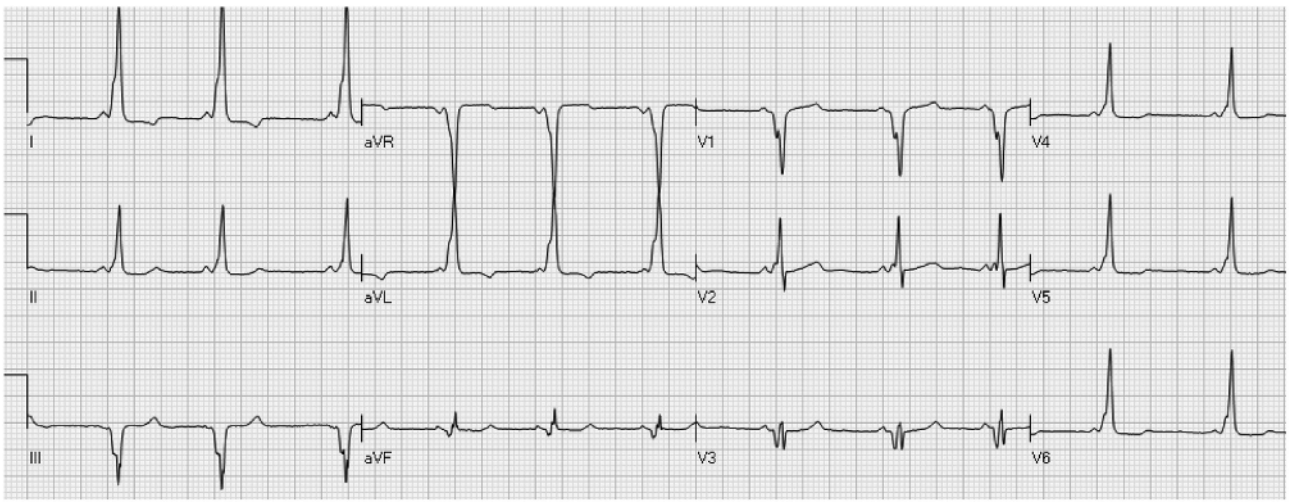



**Figure 7.1.11** Wolff-Parkinson-White (WPW) pattern. Note the short PR interval, prolonged QRS interval, and prominent "delta wave." The latter is most easily seen in lead II and represents the ventricular preexcitation that defines this condition.

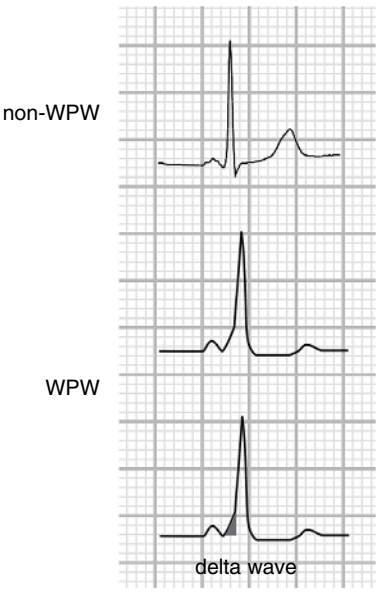



**Figure 7.1.12** Ventricular preexcitation. The upper tracing is an example of a non-Wolff-Parkinson-White pattern of preexcitation in which the PR interval is shortened, but the QRS duration is normal and no delta wave is present. In this case, the accessory pathway delivers the impulse proximal to the ventricle after bypassing a portion of the normal conduction pathway. The middle and lower tracings show the classic pattern of WPW in which an atrioventricular accessory pathway results in a delta wave, a shortened PR interval, and a prolonged QRS duration.

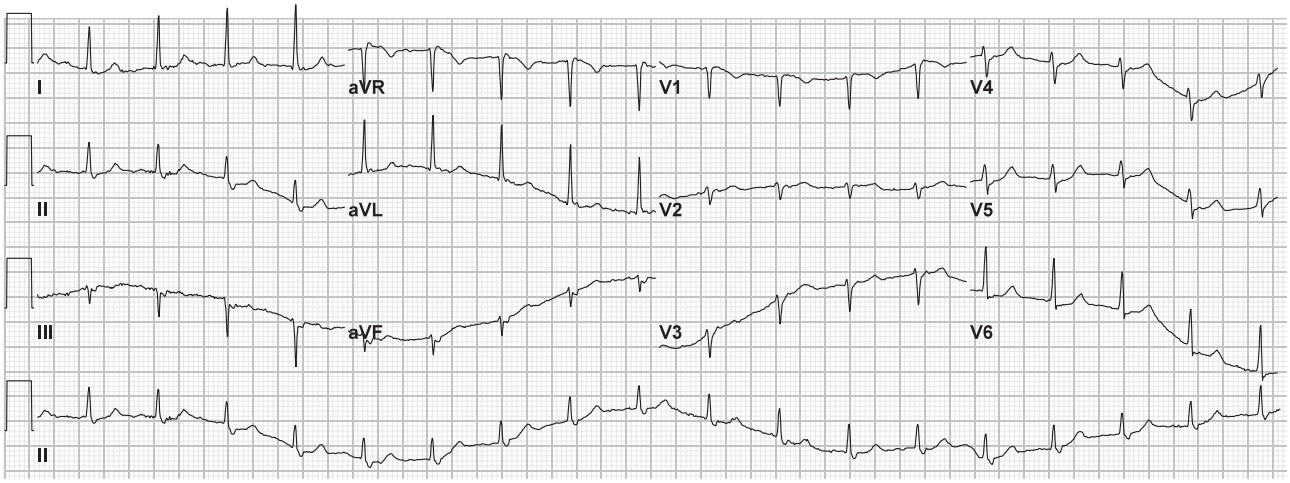



**Figure 7.1.13** Accelerated junctional rhythm. Note the inverted P waves in the inferior leads that appear to merge with the end of the QRS complexes. Also, note the upright P waves (at the end of the QRS complexes) in lead aVR.

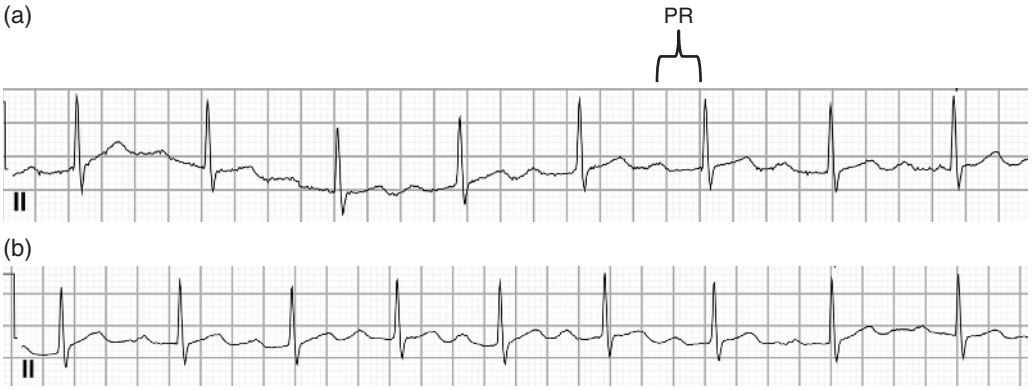



**Figure 7.1.14** (a) First-degree AV block. This 17-year-old male presented to the emergency department with complaint of palpitations. Note the prolonged PR interval, which measures 0.31 sec. (b) The PR interval shortens to 0.26 sec after the same patient engages in low-intensity exercise during the same visit. His PR interval was normal at a subsequent follow-up visit with cardiology. The cause for his first-degree AV block was not determined.

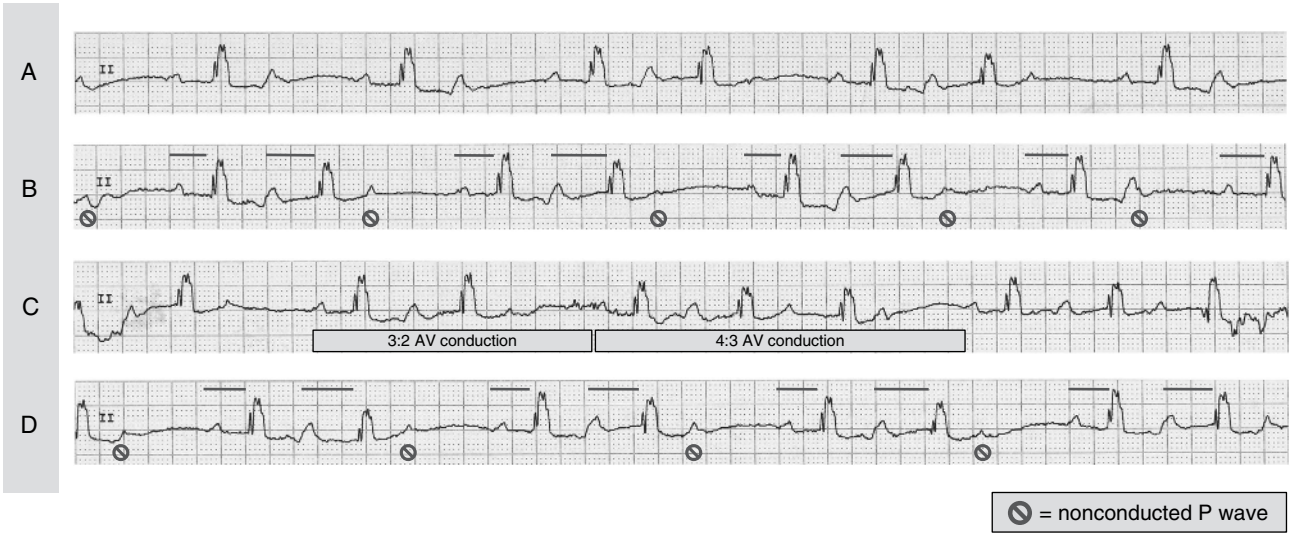



**Figure 7.1.15** Second-degree AV block, Mobitz type I (Wenckebach). **Strip A** begins with second-degree AV block with 2:1 conduction, then displays Mobitz type I pattern. **Strip B** demonstrates 3:2 AV conduction with lengthening PR intervals until the third P wave in each series is dropped; it ends with reversion to 2:1 AV conduction. **Strip C** shows periods of both 3:2 and 4:3 AV conduction. **Strip D** shows consistent 3:2 AV conduction with the lengthening PR intervals and non-conducted P waves highlighted again.

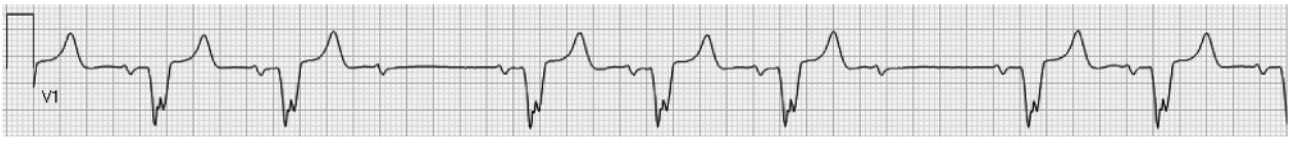



**Figure 7.1.16** Second-degree AV block, Mobitz type II. This rhythm strip demonstrates a constant PR interval with failure to conduct every fourth beat. This would be described as 4:3 AV conduction, meaning that for every four atrial impulses, only three are conducted to the ventricles. Note that the QRS complexes are wide (left bundle branch block pattern), which is typical of Mobitz type II.

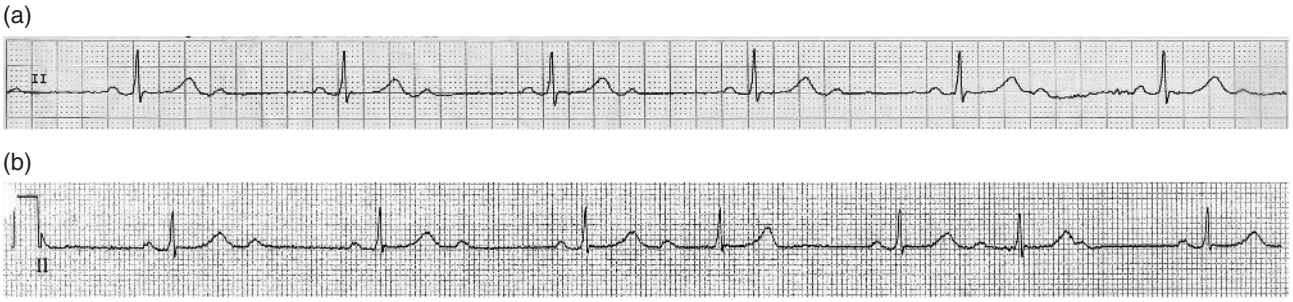



**Figure 7.1.17** (a and b) Second-degree AV block with 2:1 AV conduction. Note that the first half of this strip does not allow differentiation between type I and type II second-degree AV block, but the latter half of the strip shows prolongation of the PR interval, followed by a non-conducted P wave, confirming a type I AV block.

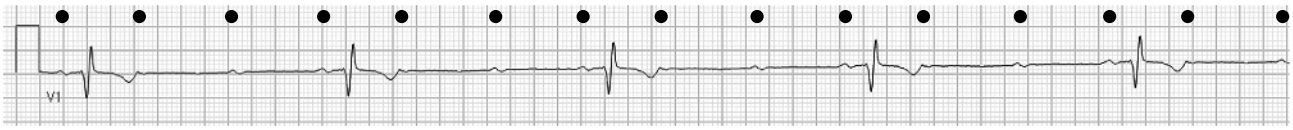



**Figure 7.1.18** High-grade AV block with 3:1 conduction ratio. Note that there are three P waves for each QRS and that the PR interval of the conducted beats is constant. The fact that more than one consecutive P wave is not conducted renders this a "high-grade" or "advanced" AV block and differentiates it from a Mobitz II block. The black circles mark the P waves.

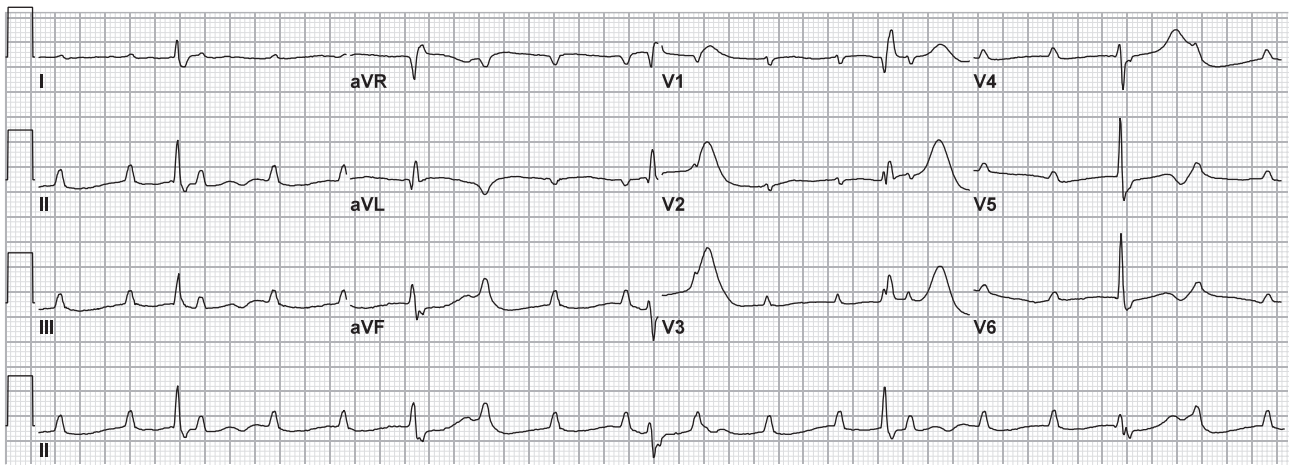



**Figure 7.1.19** Third-degree AV block (complete heart block). This 67-year-old female presented to the emergency department after a syncopal episode and was experiencing periods of intermittent weakness and near syncope. This ECG was obtained during one of those periods and displays sinus tachycardia with complete heart block and a junctional escape rhythm with right bundle branch block. She was diagnosed with a non-ischemic cardiomyopathy of uncertain etiology and required permanent pacemaker placement.

The QRS duration is typically normal but may be wide, especially in conditions that also affect conduction via the His-Purkinje system or in the case of preexisting bundle branch block. The opposite is true of type II second-degree AV block in that the QRS complex is most often wide, but may be normal, as in the case of type II block at the His bundle (Figure 7.1.9).

In type II second-degree AV block, the PR interval is constant on every conducted beat, but not every P wave conducts to produce a QRS complex. The PR interval may be

long or normal. The QRS complexes that do conduct are most often wide because the lesion is typically farther down the conduction system (usually in the bundle branches) than in type I block and, therefore, affects the QRS interval. In judging the severity of the block, it is useful to describe the conduction ratio in terms of the number of P waves generated to the number of QRS complexes that result. For example, if three of every four P waves are conducted, this would be described as an AV conduction ratio of 4 : 3 (Figure 7.1.16). Ratios of 4 : 3 or 3 : 2 are most common, but others may be seen. In addition, the ratio need not be constant over time, as a variety of dynamic factors influence AV conduction (e.g. sympathetic and parasympathetic tone, ischemia, circulating concentration of calcium channel or beta-adrenergic antagonists).

If the AV conduction ratio is seen to be 2 : 1, it is usually impossible to differentiate type I from type II second-degree AV block, as the progressive PR-interval lengthening that is characteristic of type I block cannot be seen (Figure 7.1.17a). Sometimes, a long rhythm strip reveals a variable conduction ratio over time. In this case, PR interval lengthening in consecutive cycles may be seen (Figure 7.1.17b). Because QRS complexes may be narrow or wide with either of these scenarios, QRS duration does not definitively identify the precise type of block, either. Autonomic influences that accelerate the atrial rate (e.g. anticholinergic medications, exercise) often improve AV conduction in the setting of type I block, while having no effect on, or potentially worsening, the conduction in type II block. On the contrary, vagal maneuvers may improve type II block by slowing the atrial rate and allowing more time for recovery of the infranodal conduction system between impulses. Demonstration of such changes in the severity of the block with these maneuvers may assist with the diagnosis, though expert consultation is advised.

So-called "high-grade" or "advanced" AV block can result from either type I or type II second-degree AV block and indicates significant risk for progression to complete heart block. Differentiation of the type of block that preceded the high-grade block (type I versus type II) is not immediately important. This condition is defined by an AV conduction ratio of 3 : 1 or greater. In other words, for every three sinus beats, only one QRS complex is seen (Figure 7.1.18). Similarly, a 4 : 1 AV conduction ratio would indicate that of four sinus beats, only one QRS complex results. Both the PR interval (of conducted beats) and the QRS duration in a high-grade block may be normal or prolonged.

#### Third-Degree Atrioventricular (Complete Heart) Block

In complete heart block, there is no meaningful communication between the atria and the ventricles (Figure 7.1.19). The atrial rate is independent of the ventricular rate, and the P waves and QRS complexes display no relationship to one another; this is referred to as AV dissociation. It is important to recognize, though, that third-degree AV block is but one type of AV dissociation; other types include junctional and ventricular tachycardias. The PP interval and the RR interval should be relatively constant, though the former may vary some as in the case of sinus arrhythmia. The PR interval, however, is variable. The QRS duration and ventricular rate are determined by the site of the escape focus. If this focus is within the His bundle, the QRS complex may be narrow, and the rate may be 40–60 bpm. Pacemakers distal to the His bundle typically generate a ventricular rate less than 40 beats per minute with wide QRS complexes.

## Conclusion

While the P wave and PR interval are often overlooked as less important components of the electrocardiogram, when examined carefully, they can provide the clinician with critical information that directly impacts patient management. Indeed, close inspection of the P wave's morphology, amplitude, and duration, as well as its relation to the other components of the tracing (e.g. QRS complex and associated PR interval), reveals the anatomic focus of the rhythm in the majority of cases. This is certainly crucial for the electrocardiographic diagnosis and is often central to determining the origin of the patient's symptomatology.

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