With normal cardiac anatomy (see Chapter 1), the only means of electrical communication between the atria and ventricles is via the specialized conduction system of the heart. This relay network comprises the atrioventricular (AV) node, which is connected to the His bundle, which in turn is connected to the bundle branches (Fig. 17-1). The atria and ventricles are otherwise electrically isolated from each other by connective tissue in the indented rings (grooves) between the upper and lower chambers. The key exception occurs with Wolff-Parkinson-White (WPW) preexcitation syndrome, described in Chapters 12 and 14.

The slight physiologic delay, reflected in the normal PR interval, between atrial and ventricular activation allows the ventricles optimal time to fill with blood during and after atrial contraction. Excessive slowing or actual interruption of electrical signal propagation across the heart’s conduction system is abnormal and termed AV (atrioventricular) block or heart block. The closely related (and often confusing to students and experienced clinicians!) topic of AV dissociation is discussed at the end of this chapter and in Chapter 10.

**WHAT IS THE DEGREE OF AV BLOCK?**

Depending on the severity of conduction impairment, there are three major degrees of AV block:

- **First-degree (PR interval prolongation):** uniform slowing of conduction between the atria and ventricles (an increase in the normal AV delay described earlier), but without its interruption
- **Second-degree:** intermittent interruption of conduction, which may be further designated as Mobitz I (AV Wenckebach) or Mobitz II varieties
- **Third-degree:** complete interruption of AV conduction, with a nodal or infranodal escape rhythm, or with asystole

Two other important subtypes of second-degree AV block, namely 2:1 block and high-grade block (also referred to as “advanced second-degree AV block”) will also be discussed.

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**Key Points**

Clinicians should try to answer two key questions when examining the ECG of a patient with apparent AV heart block:

1. **What is the degree of block:** is it first, second, or third degree (complete)?
2. **What is the most likely level of the block:** is it in the AV node (nodal) or below the AV node, in the His–bundle branch system (infranodal)?
**First-Degree AV Block**

Figure 17-2. With first-degree atrioventricular (AV) “block,” the PR interval is uniformly prolonged above 0.20 sec (200 msec) with each electrical cycle.

**Mobitz Type I (Wenckebach) Second-Degree AV Block**

Figure 17-3. Sinus rhythm is present. The PR interval lengthens progressively with successive beats until one P wave is not conducted at all. Then the cycle repeats itself. Notice that the PR interval following the nonconducted P wave is shorter than the PR interval of the beat just before it.

**Mobitz Type I (Wenckebach) Second-Degree AV Block**

Figure 17-4. Sinus rhythm is present. Notice the progressive increase in PR intervals, with the third P wave in each sequence not followed by a QRS complex. Mobitz type I (Wenckebach) block produces a characteristically syncopated rhythm with grouping of the QRS complexes (“group beating”).

**Prolonged PR Interval (First-Degree AV Block)**

First-degree AV block (Fig. 17-2) is characterized by a P wave (usually sinus in origin) followed by a QRS complex with a uniformly prolonged PR interval greater than 200 msec. The preferred term is PR interval prolongation because the signal is not really blocked, but rather it is delayed. The PR interval can be slightly prolonged (e.g., 240 msec) or it can become markedly long (up to 400 msec or longer).

**Second-Degree AV Block Syndromes**

Second-degree AV block is characterized by intermittently “dropped” QRS complexes. There are two major subtypes of second-degree AV block: Mobitz type I (AV Wenckebach) and Mobitz type II.

With Mobitz type I, the classic AV Wenckebach pattern (Figs. 17-3 and 17-4), each stimulus from the atria has progressive difficulty traversing the AV node to the ventricles (i.e., the node becomes increasingly refractory). Finally, the atrial stimulus is not conducted at all, such that the expected QRS
complex is blocked (“dropped QRS”). This cycle is followed by relative recovery of the AV junction, and then the whole cycle starts again.

The characteristic ECG signature of AV Wenckebach block, therefore, is progressive lengthening of the PR interval from beat to beat until a QRS complex is dropped. The PR interval following the nonconducted P wave (the first PR interval of the new cycle) is always shorter than the PR interval of the beat just before the nonconducted P wave.

The number of P waves occurring before a QRS complex is “dropped” may vary. The nomenclature is in terms of a ratio that gives the number of P waves to QRS complexes in a given cycle. The numerator is always one higher than the denominator. In many cases just two or three conducted P waves are seen before one is not conducted (e.g., 3:2, 4:3 block). In other cases, longer cycles are seen (e.g., 5:4, 10:9, etc.).

As you can see from the examples, the Wenckebach cycle also produces a distinct clustering of QRS complexes separated by a pause (the dropped beat). Any time you encounter an ECG with this type of group beating, you should suspect AV Wenckebach block and look for the diagnostic pattern of lengthening PR intervals and the presence of a nonconducted P wave. As discussed in the following text, infranodal second-degree AV block (Mobitz type II) also demonstrates grouped beating with dropped QRS complexes, but without significant progressive PR interval prolongation (Fig. 17-5).

**Caution!** Be careful not to mistake group beating due to blocked atrial premature beats (APBs) for second-degree AV block. In the former, the nonconducted P waves come “early”; in the latter they come “on time” (see Chapter 14).

**Mobitz type II AV block** is a rarer and more serious form of second-degree heart block. Its characteristic feature is the sudden appearance of a single, nonconducted sinus P wave without (1) the progressive prolongation of PR intervals seen in classic Mobitz type I AV block, and (2) without the noticeable (≤40 msec) shortening of the PR interval in the beat following the nonconducted P wave versus the PR before, as seen with type I block.

A subset of second-degree heart block occurs when there are multiple consecutive nonconducted P waves present (e.g., P-QRS ratios of 3:1, 4:1, etc.). This finding is referred to as high-degree (or advanced) AV block. It can occur at any level of the conduction system (Fig. 17-6). A common mistake is to call this pattern Mobitz II block.

**Third-Degree (Complete) AV Block**

First- and second-degree heart blocks are examples of incomplete block because the AV junction conducts some stimuli to the ventricles. With third-degree, or complete, heart block, no stimuli are transmitted from the atria to the ventricles. Instead, the atria and ventricles are paced independently. The atria often continue to be paced by the sinoatrial (SA) node. The ventricles, however, are paced by a nodal or infranodal escape pacemaker located somewhere below the point of block. The resting ventricular rate with complete heart block may be around 30 beats/min or lower or as high as 50 to 60 beats/min. This situation, when there is no “cross-talk” between the atria and ventricles and
Advanced Second-Degree AV Block

**Figure 17-6.** Modified lead II recorded during a Holter monitor ECG in a patient complaining of intermittent lightheadedness. The ECG shows sinus rhythm with 2:1 atrioventricular (AV) block alternating with 3:1 AV block (i.e., two consecutive nonconducted P waves followed by a conducted one). The term high-grade or advanced second-degree AV block is applied when the ECG shows two or more nonconducted P waves in a row.

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**BOX 17-1 ECG with Sinus Rhythm and Complete Heart Block: Three Key Features**

- P waves (upright in lead II) are present, with a relatively regular sinus rate that is typically much faster than the ventricular rate.
- QRS complexes are present, with a slow (usually near-constant) ventricular rate.
- The P waves bear no relation to the QRS complexes; thus, the PR intervals are variable.

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In general, block at the level of the AV node:
- Is often caused by reversible factors (Box 17-2)
- Progresses more slowly, if at all
- In the case of complete heart block, is associated with a relatively stable escape rhythm

In contrast, infranodal block:
- Is usually irreversible (Box 17-3)
- May progress rapidly and unexpectedly to complete heart block with a slow, unstable escape mechanism

Therefore, infranodal block (even second-degree) generally requires pacemaker implantation.

Clues to nodal versus infranodal mechanisms of AV block include the following:
- **Onset and progression of block**
  - Nodal block usually occurs gradually. Conduction through the AV nodal cells is relatively sluggish (relying on slowly depolarizing calcium channels) and accounts for most of the PR interval duration. As the block progresses, a significant additional PR interval prolongation usually occurs before conduction fails completely with second- or third-degree block (similar to stretching of an elastic band before it snaps).
  - In contrast, infranodal block usually happens abruptly. Conduction through the infranodal structures is relatively fast (relying on rapidly conducting sodium channels) and therefore accounts for only a very small portion of the PR interval. As a consequence, when infranodal block develops there is minimal or no visible PR prolongation and the block (second- or third-degree) appears abruptly (similar to the snap of a metal chain).
- **Escape rhythms**
  - Because the AV node is located on the very top (proximal part) of the specialized conduction system, there are multiple potential escape or
CHAPTER 17  What Is the Location of the Block? Nodal vs. Infranodal

Third-Degree (Complete) AV Block

**Figure 17-7.** Complete heart block is characterized by independent atrial (P) and ventricular (QRS complex) activity. The atrial rate (sinus rate, here) is always faster than the ventricular rate. The PR intervals are completely variable. Some sinus P waves fall on the T wave, distorting its shape. Others may fall in the QRS complex and be “lost.” Notice that the QRS complexes are of normal width, indicating that the ventricles are being paced from the atrioventricular junction. Compare this example with Figure 17-8, which shows complete heart block with wide, very slow QRS complexes because the ventricles are most likely being paced from below the atrioventricular junction (idioventricular pacemaker).

Third-Degree (Complete) Heart Block

**Figure 17-8.** This example of sinus rhythm with complete heart block shows a very slow, idioventricular (note wide QRS) rhythm and a faster independent atrial (sinus) rhythm.

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**BOX 17-2** Some Conditions That May Cause Temporary AV Conduction Impairment

- Autonomic factors (increased vagal tone with vasovagal syncope or sleep apnea). Trained athletes at rest may show a prolonged atrioventricular (AV) interval and even AV Wenckebach with sinus bradycardia that resolve with exercise.
- Medications (especially, beta blockers; digoxin, certain calcium channel blockers) and electrolyte abnormalities (especially hyperkalemia)
- Acute myocardial infarction, especially inferior (see text discussion)
- Inflammatory processes (e.g., myocarditis, rheumatic fever, lupus)
- Certain infections (e.g., Lyme disease, toxoplasmosis)

**BOX 17-3** Some Causes of Permanent AV Conduction System Damage

- Acute myocardial infarction, especially anterior wall
- Infiltrative diseases (e.g., amyloid, sarcoid, lymphomas)
- Degeneration of the conduction system, usually with advanced age (Lenègre’s disease) or associated with cardiac calcification around the aortic and mitral valves (Lev’s disease)
- Hereditary neuromuscular diseases (e.g., myotonic dystrophy, Kearns-Sayre syndrome, Erb’s dystrophy)
- Iatrogenic damage to the conduction system as the result of valve surgery or arrhythmia ablations in the area of atrioventricular (AV) node and His bundle; ethanol septal ablation for obstructive hypertrophic cardiomyopathy
“backup” pacemakers located below the level of block, i.e., in the lower parts of the AV node as well as the His bundle and its branches. Pure nodal and His bundle escape rhythms have narrow QRS complexes and a moderately low rate (e.g., 40-60 beats/min). However, the QRS complexes may be wide if there is an associated bundle branch block. As a result, in the case of complete block occurring at the nodal level, there is usually a hemodynamically adequate escape rhythm present.

- In contrast, when the site of block is infranodal (in the His bundle, Purkinje system, or ventricular myocardium), there are fewer and less reliable potential escape pacemakers below that level. Idioventricular escape mechanisms usually produce regular, wide QRS complexes with a very slow rate (i.e., 40 beats/min or less). In addition, the abrupt onset of the block can produce a life-threatening period of asystole.

- **Autonomic and drug influences**
  - The physiology of the AV node is similar to that of the sinus node and their functions tend to change in parallel. Both are sensitive to the autonomic (sympathetic and parasympathetic) stimulation, as well as drugs affecting the autonomic nervous system (e.g., beta blockers, atropine, digoxin, and certain calcium channel blockers). For example, vagal stimulation can simultaneously produce both sinus bradycardia and AV block. This combination is commonly seen in vasovagal (neurocardiogenic) syncope, obstructive sleep apnea, or during normal sleep.
  - Almost all medications causing sinus bradycardia (see Chapter 13) also worsen AV nodal conduction and can induce various degrees of heart block at the level of the AV node. Of note, adenosine has very potent suppressive activity on the AV (and SA) nodes and can induce transient complete heart block, an important effect to be aware of when it is used for differential diagnosis and termination of supraventricular arrhythmias (see Chapter 14). Stimulation with sympathomimetic (e.g., dopamine, isoproterenol, and epinephrine) and anticholinergic drugs (atropine) increases the sinus rate and improves AV conduction.
  - In contrast, infranodal conduction does not respond to sympathetic/parasympathetic stimulation or most drugs. (Rarely, antiarrhythmic sodium channel blockers such as quinidine, flecainide, or propafenone can produce infranodal block. They can also markedly worsen or unmask preexisting infranodal disease.) Infranodal block often worsens with an increase in heart rate. Drugs causing tachycardia such as atropine and sympathomimetics are likely to worsen conduction in infranodal block (although beta agonists are useful in speeding up the rate of an idioventricular escape pacemaker in cases of a complete infranodal block in emergency settings).

- **QRS duration**
  - The width of the QRS complexes depends in part on the location of the block. If the block is in the AV node proper, the ventricles are stimulated normally by a nodal pacemaker below the point of block and the QRS complexes are narrow (≤120 msec) (see Fig. 17-5), unless the patient has an underlying bundle branch block. If the block is within, or particularly below, the bundle of His, the ventricles are paced by an infranodal pacemaker, usually producing wide (>120 msec) QRS complexes (see Fig. 17-6). As a general clinical rule, complete heart block with wide QRS complexes tends to be less stable than complete heart block with narrow QRS complexes because the ventricular escape pacemaker is usually slower and less consistent.
  - With infranodal disease, there are often (but not always) other signs present (bundle branch blocks, hemiblocks, or nonspecific QRS widening).

### 2:1 AV Block: A Special and Often Confusing Subtype of Second-Degree Heart Block

2:1 AV block occurs when every other QRS complex is “dropped” or, equivalently, every other P wave is not conducted. In such cases, it becomes difficult or impossible from the surface ECG to tell Mobitz I from Mobitz II type block simply because there are not two consecutive conducted PR intervals to compare with the subsequent nonconducted one. Very prolonged PR interval in conducted beats (>280 msec) strongly suggests nodal (type I) block (Fig. 17-9), although a relatively short PR interval (≤140 msec), especially in association with QRS widening, suggests
infranodal (type II) block (Fig. 17-10). Intermediate values are not diagnostic.*

**Cautions:** 2:1 AV block may present a very common pitfall in ECG analysis when the non-conducted P wave is hidden in the preceding T wave (see Chapter 23). The rhythm may be misdiagnosed as “normal sinus” or “sinus bradycardia.” If the PR interval of conducted beats is not prolonged (as usually seen in infranodal block) the presence of AV block can be completely missed while the patient, in fact, urgently needs a permanent pacemaker.

Blocked atrial bigeminy (see Chapter 14) can appear similar to 2:1 AV block, but PP interval differences usually allow you to distinguish between these two distinct diagnoses. In 2:1 AV block, the P waves come “on time,” but with atrial bigeminy and blocked APBs, every other P wave is early.

**ATRIAL FIBRILLATION OR FLUTTER WITH AV HEART BLOCK**

With atrial fibrillation or flutter, the diagnosis of AV heart block is complicated. It is impossible to diagnose first- or second-degree AV block in the presence of these arrhythmias because of the lack of discrete P waves. However, with complete heart block, clues are as follows:

- Regularization
- Marked slowing (<50 beats/min or slower) of the ventricular rate (sometimes with QRS widening and change in morphology suggesting an escape pacemaker rather than conducted beats) (see Chapter 15)

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*In uncertain cases, in which the need for a pacemaker is being assessed, an invasive electrophysiologic study can be done to record directly the signals from the conduction system of the heart. In Mobitz I block the signal blocks in the AV node without reaching the His bundle area. In Mobitz II block the signal reaches the His bundle, producing a typical deflection on the intracardiac recording.*
Important Clinical Considerations

Symptoms

Symptoms of heart block vary depending on its degree as well as the time course of its development.

PR interval prolongation (first-degree AV block) is usually asymptomatic (see Box 17-4). Occasionally, if the PR interval becomes so long that the P waves move close to the preceding QRS complexes, the patient might feel pulsation in the neck and dizziness, similar to that seen in the pacemaker syndrome, due to near simultaneous atrial and ventricular contractions (see Chapter 21).

Second-degree block can produce sensations of skipped beats and exertional dyspnea due to inability to augment heart rate with exercise.

Development of a complete heart block can be a life-threatening event presenting with presyncope or syncope (Adams-Stokes attacks) due to a very slow escape rate or even to prolonged asystole. This severe bradycardia is more likely to happen with infranodal complete blocks due to their more abrupt onset and the slower rate of the escape rhythms (see Chapter 13).

In addition, very slow rates can induce severe QT interval prolongation and torsades de pointes ventricular tachycardia (see Chapter 16), culminating in cardiac arrest due to ventricular fibrillation (see Chapter 19). If the patient survives this initial episode of complete heart block, the primary complaints are usually severe exertional dyspnea and fatigue due to inability to augment heart rate and cardiac output with exercise, similar to that of second-degree AV block.*

Treatment Considerations

The initial, emergency approach to a symptomatic patient with complete heart block should follow the current ACLS algorithms and appropriate measures, including preparation for transcutaneous pacing if indicated. If the patient is hemodynamically stable, the level of block should be determined and potential causes reviewed (see Boxes 17-2 and 17-3).

AV HEART BLOCK IN ACUTE MYOCARDIAL INFARCTION

AV block of any degree can develop during acute myocardial infarction because of the interruption

*Rarely, patients may have congenital complete heart block (which is usually at the AV nodal level, associated with a narrow and not excessively slow QRS escape mechanism). These individuals may be asymptomatic (other than noting a slow pulse) because of adaptations including increased left ventricular dimension and stroke volume.

BOX 17-4  Infections and Heart Block

- Progressive PR prolongation in a patient with infective endocarditis is an ominous sign, suggesting the development of a peri-valvular abscess.
- Lyme disease can produce any degree of heart block at the level of AV node, including complete heart block, often associated with severe symptoms. Occasionally syncope can be the first presentation of the disease. Almost always the block resolves with antibiotic therapy, but sometimes temporary pacing is required.

Key Point

Because septal branches are proximal divisions of LAD artery, development of RBBB or complete heart block with acute anterior myocardial infarction is an ominous sign, suggesting a very large area of myocardium at risk. Prompt restoration of blood flow through the occluded coronary artery by angioplasty and stenting or thrombolysis often resolves infranodal block in anterior myocardial infarction.
Acute Inferior MI with Second Degree AV Block

Figure 17-11. Sinus tachycardia with acute inferior (and probably posterolateral) ST segment elevation myocardial infarction (MI) with 3:2 atrioventricular (AV) Wenckebach block. Arrows point to the sinus P waves at rate of about 100 beats/min. There is a 3:2 conduction pattern with AV Wenckebach block, indicating Mobitz I block. Note the subtle group beating pattern. The ST segment depressions in leads V1-V3 are consistent with reciprocal change to the ST segment elevations laterally and probably posteriorly.

Acute/Evolving Anterior MI and AV Heart Block

Figure 17-12. High-degree AV block (Mobitz II and complete) in acute anterior ST segment elevation MI (STEMI). Multiple sinus nonconducted P waves are present. Third and fourth QRS complexes (narrow) appear relatively early and therefore are likely to be conducted. Wider QRS complexes (with right bundle branch block [RBBB] and right axis morphology) at a regular slow rate represent an idioventricular (fascicular) escape rhythm. The anterior and inferior ST segment elevations are present in both conducted and escape complexes. Q waves in anterior and inferior leads are consistent with evolving extensive anterior myocardial infarction, possibly due to a very proximal occlusion of a large “wrap-around” left anterior descending coronary artery. This situation requires emergent temporary pacing and reperfusion therapy.
AV DISSOCIATION SYNDROMES

Cardiologists use the term AV dissociation in two related though not identical ways. This classification continues to cause considerable (and understandable) confusion among students and clinicians.

• AV dissociation is widely used as a general term for any arrhythmia in which the atria and ventricles are controlled by independent pacemakers. The definition includes complete heart block, as described previously, as well as some instances of ventricular tachycardia or accelerated idioventricular rhythm in which the atria remain in sinus rhythm (see Chapter 16).

• AV dissociation is also used as a more specific term to describe a particular family of arrhythmias that are often mistaken for complete heart block. With this type of AV dissociation, the SA node and AV junction appear to be “out of synch”; thus, the SA node loses its normal control of the ventricular rate. As a result the atria and ventricles are paced independently—the atria from the SA node, the ventricles from the AV junction. This situation is similar to what occurs with complete heart block. However, in this instance, the ventricular rate is the same as or slightly faster than the atrial rate. When the atrial and ventricular rates are almost the same, the term isorhythmic AV dissociation is used. (Iso is the Greek root for “same.”)

The critical difference between AV dissociation (resulting from “desynchronization” of the SA and AV nodes) and complete heart block (resulting from actual conduction failure) is as follows: with AV dissociation (e.g., isorhythmic) a properly timed P wave can be conducted through the AV node, whereas with complete heart block, no P wave can stimulate the ventricles.

AV dissociation (Fig. 17.13) when used in this more specific context, therefore, can be regarded as a “competition” between the SA node and the AV node for control of the heartbeat. It may occur either when the SA node slows down (e.g., because of the effects of beta blockers or calcium channel blockers or with increased vagal tone) or when the AV node is accelerated (e.g., by ischemia or digitalis toxicity). Not uncommonly, isorhythmic AV dissociation is seen in healthy young individuals, particularly when they are sleeping.

Figure 17-14 presents an example of isorhythmic AV dissociation, a common benign arrhythmia.
easily confused with complete heart block. Notice the P waves with a variable PR interval because the ventricular (QRS) rate is nearly the same as the atrial rate. At times the P waves may merge with the QRS complexes and become imperceptible for several beats. If the sinus rate speeds up sufficiently (or the AV junctional rate slows), the atrial stimulus may be able to penetrate the AV junction, reestablishing sinus rhythm.