Bradyarrhythmias are the result of either (1) extrinsic factors slowing the normally functioning sinus node and conduction pathways or (2) failure of the sinus node and conduction pathways because of intrinsic causes of degeneration of the conduction system.

**EXTRINSIC CAUSES**

Clues to an extrinsic cause of a bradyarrhythmia are a history of a new cardioactive medication, conditions that activate vagal tone, and extreme electrolyte imbalances, specifically potassium (Table 58.1). The associated rhythms are typically those that are due to dysfunction of the sinus or atrioventricular (AV) node:

- Sinus bradycardia, arrest (Figs. 58.1 to 58.3)
- Type I second-degree AV nodal block (Fig. 58.4)
- Third-degree heart block (see Fig. 58.6)

**INTRINSIC CAUSES**

Certain conditions result in failure of elements of the conduction system because of aging, ischemia or infarction, surgical trauma, or infiltration (Table 58.2). The latter cause encompasses a large group of conditions that include infectious and rheumatologic diseases. The associated rhythms are typically those that indicate failure of one element of the conduction system rather than failure to generate a rhythm:

- Second-degree AV block (either type I or type II) (Fig. 58-5; also see Fig. 58.4)
- Third-degree heart block (Fig. 58.6)
- Associated fascicular or bundle branch blocks (see Fig. 58.5)

**RELATIVE BRADYCARDIA**

Relative bradycardia is the presence of a heart rate that is inappropriately slow for the clinical findings. Though often caused by coexisting beta-blocker therapy or age-related blunting of sinus node automaticity, it is also a diagnostic feature of a number of infectious disorders. Relative bradycardia is most useful in differentiating infectious diseases that resemble each other; for example, legionnaires’ disease from *Mycoplasma* pneumonia and psittacosis or Q fever from tularemia pneumonia.

**PRESENTING SIGNS AND SYMPTOMS**

Cardiac output is dependent on the patient’s stroke volume and heart rate. Bradycardias are symptomatic only to the extent that they affect cardiac output, which is a product of...
the heart rate and stroke volume. In the setting of a normal stroke volume, heart rates as low as 20 to 30 beats/min can sustain a reasonable cardiac output. A heart rate of 40 beats/min may be physiologically normal for some individuals, whereas a rate of 60 beats/min may be inadequate for patients with conditions that compromise stroke volume, such as cardiomyopathies, bleeding, or sepsis.

Symptoms of low cardiac output are due to hypoperfusion of vital organs and muscle tissue. Mild reductions in cardiac output usually cause exertional fatigue and dyspnea. Greater reductions in cardiac output can produce signs and symptoms of cerebral ischemia, congestive heart failure, mesenteric ischemia, and renal insufficiency. Complete heart block with slow escape rhythms or bradycardia in the setting of profound reductions in stroke volume can manifest as syncope, pulmonary edema, and cardiogenic shock.

Alternatively, a profound bradycardia that does not result in hemodynamic compromise does not need to be treated but should be considered an important diagnostic clue. The classic example is a patient with intracranial hemorrhage and Cushing reflex, which will be manifested as hypertension and often profound bradycardia. In this case the bradycardia is a sign of elevated intracranial pressure, and treatment should focus on reducing it.

### Differential Diagnosis and Medical Decision Making

The key to diagnosis and treatment is accurate interpretation of the 12-lead electrocardiogram (ECG). Though not a comprehensive review of all bradycardic rhythms, the following are those commonly encountered.

#### Sinus Bradycardia

For the diagnosis of sinus bradycardia, P waves must be present at a heart rate of less than 60 beats/min. The morphology of the P wave must be consistent with a sinus beat (upright in leads I and II), and each P wave must be followed by a QRS complex with a fixed PR interval.

#### Sick Sinus Syndrome

Sick sinus syndrome consists of a group of diseases characterized by dysfunction of the sinus node that can have a variety of appearances on an ECG. The most common ECG finding is severe, inappropriate sinus bradycardia. Another frequently encountered form is sinus arrest with a prolonged period (>2.5 seconds) and no atrial activity. The final form of sick sinus syndrome is tachycardia-bradycardia...
**Fig. 58.2** Sinus bradycardia with a ventricular response rate of 59 beats/min. There is a P wave for every QRS complex and normal PR intervals.

**Fig. 58.3** Sick sinus syndrome. This syndrome has many variants, but all have the common feature of abrupt dysfunction of the sinus node. This electrocardiogram shows a normal sinus rhythm with abrupt absence of P waves.

**Fig. 58.4** Mobitz type I second-degree heart block. This is sinus rhythm at 70 beats/min with a slower ventricular rate because of a progressive delay in atrioventricular (AV) nodal conduction and, finally, a dropped beat. Note that the PR interval after the dropped beat is shorter than the PR interval just before the dropped beat. The hallmark feature of this type of second-degree AV block is the presence of a normal sinus rhythm with a progressive delay in AV nodal conduction.
Table 58.2 Intrinsic Causes of Bradyarrhythmias

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
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<tbody>
<tr>
<td>Ischemia and infarction</td>
<td>Inferior wall MI—sinus bradycardia, type I second-degree heart block, third-degree heart block</td>
</tr>
<tr>
<td></td>
<td>Anterior wall MI—type II second-degree heart block, third-degree heart block</td>
</tr>
<tr>
<td>Infection</td>
<td>Viral—varicella, mononucleosis, hepatitis, mumps, rubella, rubeola</td>
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<tr>
<td></td>
<td>Bacterial—endocarditis, diphtheria, Lyme disease</td>
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<tr>
<td></td>
<td>Parasitic—Chagas disease</td>
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<tr>
<td>Malignancy</td>
<td>Lymphoma, sarcoma</td>
</tr>
<tr>
<td>Rheumatologic</td>
<td>Systemic lupus erythematosus, rheumatoid arthritis, Reiter syndrome, systemic sclerosis</td>
</tr>
<tr>
<td>Other</td>
<td>Sarcoïd, amyloid, Lev disease (spread of fibrosis and calcification from the adjacent cardiac skeleton), Lenègre disease (idiopathic fibrotic degeneration of the His-Purkinje system)</td>
</tr>
</tbody>
</table>

Fig. 58.5 Mobitz type II second-degree atrioventricular block. Careful review of this electrocardiogram shows a sinus rhythm with a left anterior fascicular block and a right bundle branch block. Importantly, there is evidence of an anterior wall infarct with ST-segment elevation in the anterior leads. This demonstrates a significant complication of anterior wall infarcts involving the septum, with necrosis of the right bundle branch and left anterior fascicle and intermittent failure of the left bundle branch.

Fig. 58.6 Complete atrioventricular block. This electrocardiogram shows an underlying sinus rhythm that is regular at approximately 80 to 90 beats/min. The ventricular escape rhythm is slow and regular with no apparent relationship between the P waves and the QRS complexes.
syndrome, which is characterized by alternating periods of severe bradycardia interrupted by paroxysms of supraventricular tachycardia, usually atrial fibrillation.

**SECOND-DEGREE HEART BLOCK**
These heart blocks will be manifested as bradycardia only if the sinus rhythm is slow or a significant percentage of the sinus beats are not conducted.9

**Type I Second-Degree Atrioventricular Block**
The key ECG feature of a Mobitz type I second-degree AV block (i.e., a Wenckbach block) is progressive lengthening of the PR interval leading to a dropped QRS complex. The PR interval of the first-conducted QRS complex after a nonconducted beat has the shortest or a relatively normal PR interval. These blocks are usually indicative of a medication effect, AV node disease, or ischemia, but the rhythm is rarely the source of the instability10 (Fig. 58.7; also see Fig. 58.4).

**Type II Second-Degree Atrioventricular Conduction Block**
Diagnosis of a Mobitz type II heart block is determined by the presence of an intact sinus pacemaker with intermittent failure of conductance to the ventricles. This is due to failure of either the bundle of His or one of the Purkinje fibers. This rhythm is indicative of significant disease of the infranodal conducting system and is likely to progress to a complete heart block. These blocks are often due to myocardial ischemia and are symptomatic (see Fig. 58.5).

**THIRD-DEGREE ATRIOVENTRICULAR BLOCK**
This rhythm should be considered first in a patient with bradyarrhythmia because it is always clinically significant, often hemodynamically compromising, and unstable (Fig. 58.8; also see Fig. 58.6). The ventricular rate is typically very slow, but the actual rate varies according to the inherent rate of the escape rhythm. In this rhythm there is evidence of independent activity because of no connection between the atria and ventricles. It is important to “march out” the P waves to see if they are regular because some are most likely buried within or are part of the QRS complex or T wave. It should be apparent that there is no fixed relationship between the P waves and the QRS complexes. The QRS complexes can be narrow or wide, depending on the location of the escape pacemaker. Junctional escape pacemakers are faster (50 to 60 beats/min), narrow, and relatively stable in comparison with a ventricular escape rhythm, which is slow (30 to 40 beats/min), wide, and unstable. Often, the most helpful feature of the ECG for this rhythm is that the QRS complexes should be absolutely regular. This is a rhythm initiated by a slower pacemaker exhibiting spontaneous automaticity that has “escaped” the influence of the overriding sinus rhythm. Escape rhythms fire automatically at their inherent rate and are regular. Any irregularity in the escape rhythm suggests a high-grade incomplete AV block or an inherently unstable escape rhythm.

**BRADYCARDIA ASSOCIATED WITH A WIDE QRS COMPLEX**
Bradycardia associated with a “new” widening of the QRS complex suggests a cause that results in diffuse slowing of depolarization and conduction and that usually implicates agents that block the fast sodium channels or their ability to repolarize. This bradycardia is typically seen in patients with poisoning from agents such as tricyclic antidepressants, in patients with hypothermia, or in those with hyperkalemia (Figs. 58.9 and Fig. 58.10).

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![Fig. 58.7](image_url) This electrocardiogram shows diffuse ST elevations involving both the inferior and anterior walls. The ventricular rate is irregular, which indicates that the rhythm is originating from the sinus node and is not an escape rhythm. Though not the typical pattern of a Mobitz type I second-degree heart block, the progressive lengthening of the PR intervals suggests that the block is occurring at the level of the atrioventricular node. This conduction disturbance may be responsive to atropine.
Fig. 58.8 Acute myocardial infarction and third-degree atrioventricular block. The sinus rhythm is regular and slow. The lead II rhythm strip shows that there is no fixed relationship between the P waves and the QRS complex. The ventricular response is also regular and narrow, thus suggesting a junctional escape rhythm in the setting of an inferior wall myocardial infarction. The block is at the level of the atrioventricular node and may be responsive to atropine.

Fig. 58.9 Hyperkalemia. This electrocardiogram (ECG) is from a 52-year-old man who complained of generalized weakness in the previous week. The ECG shows a very slow rhythm that is regular and has a wide QRS complex. No obvious P waves are present, and the morphology of the QRS complexes is not typical for a right or left bundle branch block. The differential diagnosis includes sinus arrest with an escape rhythm and electrolyte abnormalities. After treatment with intravenous calcium gluconate, insulin, and 5% dextrose in water, the heart returned to a normal sinus rhythm with a narrow QRS complex. The patient’s serum potassium value was found to be 7.1 mEq/L.
**Fig. 58.10** Electrocardiogram showing sinus bradycardia with Osborn waves distorting the QRS complexes. These are classic findings in patients with clinically significant hypothermia. Treatment consists of rewarming and monitoring the patient’s condition via telemetry.

**FACTS AND FORMULAS**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Sinus bradycardia (adults)</td>
<td>Sinus rhythm &lt; 60 beats/min.</td>
</tr>
<tr>
<td>Junctional escape rhythm</td>
<td>Ventricular response rate of 40 to 60 beats/min. No P waves. Regular narrow QRS (unless preexisting BBB present).</td>
</tr>
<tr>
<td>Idioventricular rhythm</td>
<td>Ventricular response rate of 30 to 45 beats/min. No P waves. Regular wide QRS (usually does not appear as a typical right or left BBB pattern).</td>
</tr>
<tr>
<td>Type I second-degree heart block</td>
<td>Sinus rhythm with progressive PR prolongation and a nonconducted beat. The PR interval of the conducted beat following the blocked beat is the shortest interval in the sequence. The QRS complex is usually narrow.</td>
</tr>
<tr>
<td>Type II second-degree heart block</td>
<td>Normal sinus rhythm with fixed PR intervals. Random sinus beats are not conducted to the ventricle. The QRS complex is often wide.</td>
</tr>
<tr>
<td>Third-degree heart block</td>
<td>Sinus rhythm with P waves visible. The ventricular response rate is regular but slower than the atrial rate with no visible relationship between the P waves and QRS complexes. The QRS rate and width are dependent on the site of the escape rhythm. The junctional escape rate is 40 to 60 beats/min and the QRS complex is narrow. The ventricular escape rhythm is 30 to 45 beats/min and the QRS complex is wide.</td>
</tr>
</tbody>
</table>

**RED FLAGS**

- Bradycardia is relative. When cardiac stroke volume is normal, perfusion (e.g., blood pressure and cerebral perfusion) can be perfectly adequate even with ventricular response rates in the 30s. When a patient is symptomatic with a bradycardia, it is often multifactorial in etiology and attention must be paid to optimizing the stroke volume with fluids, reversing the ischemia, or adding inotropic agents in addition to increasing the heart rate.
- Bradycardia not associated with hypotension or signs of hypoperfusion may be normal.
- Bradycardia not associated with hypotension or signs of hypoperfusion may be an important clue to potentially serious conditions such as elevated intracranial pressure, electrolyte disturbances, or cardiac toxicity from medications.
- Bradycardias associated with a wide QRS complex should be reviewed carefully for the presence of a complete heart block or medication or electrolyte effects.
- Bradycardia or conduction disturbances in the setting of acute myocardial infarction should be looked for and recognized as a potential clinically significant complication.

**TREATMENT**

**PREHOSPITAL CARE**

Respiratory disorders and myocardial ischemia are often associated with bradycardia, and prehospital care should focus on optimizing oxygenation, ventilation, and blood pressure and on relieving ischemia if present. Treatment of bradycardia is indicated only for patients who have a bradycardia that is inappropriate for the clinical condition and the patient is symptomatic (Fig. 58.11).

1. Optimize oxygenation (supplemental oxygen) and/or ventilation (continuous positive airway pressure) if needed.
2. Establish intravenous access.
3. Place the patient on telemetry and obtain a 12-lead ECG if possible.
4. If the absolute ventricular response rate is the probable cause of the hemodynamic instability, the following interventions may be helpful:
   a. Atropine, 0.5 mg intravenously (IV) up to 3 mg, may be effective for sinus bradycardia, Mobitz type I second-degree AV block, and third-degree heart block.
   b. Glucagon, 1 mg IV, may be effective for beta-blocker and calcium channel blocker toxicity.
   c. Calcium gluconate, 10 mL of a 10% solution IV, may be effective for calcium channel blocker toxicity or hyperkalemia.

HOSPITAL CARE
On arrival at the hospital, a slow heart rate will be readily identified while obtaining triage vital signs. The patient should be assessed rapidly for conditions that would cause the bradycardia through a screening examination for respiratory distress, medication toxicities, or myocardial ischemia. Once identified, disease-specific treatment plans will usually treat the bradycardia as well. Mainstays of management include the following:
1. Optimize oxygenation and ventilation.
2. Establish intravenous access and administer bolus intravenous fluid if the patient is hypotensive without signs of congestive heart failure.
3. Obtain a 12-lead ECG and rhythm strip.
4. Interventions:
   a. The patient remains hemodynamically compromised by the heart rate:
      - Sinus bradycardia, type I second-degree heart block, third-degree heart block:
        - Atropine, 0.5 mg IV up to 3 mg; atropine dosing should be aimed at obtaining the lowest heart rate that will optimize perfusion. In the setting of myocardial ischemia, the clinician must carefully balance the priority of maintaining perfusion to vital organs without exacerbating the myocardial ischemia.
        - Glucagon, 1 mg IV, may be effective for beta-blocker and calcium channel blocker toxicity.
      - Calcium gluconate, 10 mL of a 10% solution IV, may be effective for calcium channel blocker toxicity or hyperkalemia.
      - Temporary pacemaker.
   b. The patient is not hemodynamically compromised by the heart rate:
      - Sinus bradycardia, type I second-degree heart block:
        - Treat the underlying cause—myocardial infarction (MI), myocardial ischemia.
        - Discontinue the offending medication.
        - Optimize electrolytes and volume status.
      - Type II second-degree heart block, third-degree heart block:
        - Temporary pacemaker.
        - Cardiology consultation.
• Treat the underlying cause—MI, myocardial ischemia.
• Optimize electrolytes and volume status.

CONSULTATION
Cardiology consultation is recommended for bradyarrhythmias that require pacemaker support and those that are due to failure of the infranodal conduction system or occur in the context of MI. Temporary cardiac pacing (TCP) can be an effective bridge to permanent pacemaker placement. The limited data on the effectiveness of TCP in patients with symptomatic bradycardia show that it is comparable with atropine and dopamine when hospital survival is used as an outcome.11 Its clinical utility is limited by the fact that it is poorly tolerated by awake patients and capture may be unreliable over time. It is reasonable to initiate TCP in unstable patients who do not respond to oxygen, fluids, and pharmacologic interventions as noted earlier. The decision regarding implantation of a pacemaker depends on the underlying cause and the likelihood that the AV block will be permanent. Many conditions affecting the conduction system will predictably resolve, such as electrolyte abnormalities, medication effects, hypothermia, and inferior wall MI, and will require at most TCP. Conversely, pacemaker implantation is indicated, even without hemodynamic instability, for conditions that are likely to progress and place the patient at risk for failure of the conduction system.12,13

Bradycardias caused by intrinsic disease of the myocardial conduction system are typically type II second-degree heart blocks and third-degree heart block and will usually require permanent pacemaker placement. Type I second-degree heart blocks rarely result in clinically significant bradycardia, but pacemaker therapy is warranted in patients who progress to complete heart block that does not respond to pharmacologic interventions.

MI is associated with significant bradyarrhythmia 25% to 30% of the time.14 The underlying mechanism is either direct ischemic injury to the sinus node, AV node, or conduction system or exaggerated vagally mediated reflexes. The bradyarrhythmias and conduction blocks associated with inferoposterior infarcts are commonly due to vagal reflexes, are responsive to atropine, and are usually transient5,16 (see Figs. 58.7 and 58.8). Sinus bradycardia and AV nodal blockade in the context of an inferior wall MI have been attributed to a higher density of cardiac afferent receptors in the inferoposterior portions of the heart. For hemodynamically compromising bradycardias, atropine is an excellent initial therapy, particularly in the first 6 hours. Doses should be administered in 0.5-mg increments with a maximum dose of 0.04 mg/kg or 3 mg. Doses lower than 0.5 mg should be avoided because of the risk for a paradox bradycardic response. When atropine is ineffective, transcutaneous or transvenous pacing is indicated. It is uncommon for type I second-degree and third-degree heart block to be the primary cause of hypotension in a patient with acute MI. Hypotension is usually due to poor stroke volume from the MI, and therapeutic effort should focus on restoring blood flow to the myocardium.

The conduction blocks associated with anterior infarcts typically involve the septum and the bundle branches that run through it. These blocks are not responsive to atropine and generally require permanent pacemaker support. Conduction blocks are typically those that are due to type II second-degree heart block or third-degree heart block. The anterior circulation perfuses the septum containing the His-Purkinje fibers. Ischemia and infarction of the septum may result in failure of one of the fascicles to conduct, usually the right bundle and left anterior fascicle, which lie in the anterior portion of the septum. Fascicular failure alone does not result in bradycardia as long as there is one functioning fascicle in sinus rhythm. Bradycardia may occur with either intermittent failure of the one remaining fascicle to conduct or failure of the bundle of His, which results in a type II second-degree heart block or third-degree heart block. Treatment is placement of a permanent pacemaker.12,13

PRIORITY ACTIONS

1. Determine whether the patient is hemodynamically compromised by the bradyarrhythmia. If the patient is hypertensive, determine whether it is due to the absolute heart rate or associated conditions that will cause a relative bradycardia to be symptomatic, such as volume loss or left ventricular dysfunction.

2. Establish intravenous access.

3. If it is determined that the absolute ventricular response rate is inappropriate for the clinical condition, the following interventions may be helpful:
   • Atropine, 0.5 mg intravenously up to 3 mg, may be effective for sinus bradycardia, Mobitz type I second-degree heart block (particularly in the setting of inferior wall myocardial infarction)
   • Glucagon, 1 mg intravenously, for beta-blocker and calcium channel blocker toxicity
   • Calcium gluconate, 10 mL of a 10% solution intravenously, for calcium channel blocker toxicity

4. Indications for temporary transcutaneous pacemaker placement include the following:
   • Symptomatic bradycardia unresponsive to pharmacologic therapy
   • Mobitz type II second-degree block
   • Third-degree heart block

FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION

ADMISSION
Admission for patients with bradycardia is dependent on the underlying cause, the extent to which the bradycardia is clinically significant, and whether interventions require in-hospital monitoring, treatment, or both.

PROGNOSIS
The prognosis varies with the cause of the bradyarrhythmia. Bradyarrhythmias secondary to extrinsic causes are reactive and usually recoverable once the offending agent or condition is removed. Intrinsic causes often suggest disease of the cardiac conduction system and require permanent pacing. Rarely is a recognized bradyarrhythmia a primary cause of death.
Bradyarrhythmias are a symptom, not a disease. Frequently, the underlying cause is clearly identified, and documentation is fairly simple—for example, the patient is experiencing effects of a new blood pressure medication. When the cause is less clear, the documentation must clearly include or exclude variables that must be considered in the work-up and treatment:

- Determine the clinical significance of the bradycardia. Did the patient pass out or complain of chest pain, exertional fatigue, or dyspnea? Precipitating factors such as nausea, head turning, tight ties, and new medications are important clues to the underlying cause.
- Does the patient have underlying cardiac disease, specifically left ventricular dysfunction, that would contribute to a bradyarrhythmia being symptomatic?
- The medical history should include a detailed medication list and recent dosing history.
- The physical examination may provide clues to the cause of the bradycardia:
  - Lethargy and hypertension—elevated intracranial pressure
  - Dialysis catheter or shunt—hyperkalemia
  - Obesity and sonorous breathing—sleep apnea
  - The electrocardiogram and frequently multiple electrocardiograms, particularly when the patient reports symptoms, often specify the diagnosis.

PATIENT TEACHING TIPS

Patients have the ability to exacerbate symptoms in specific conditions that cause bradycardia and thus should be given guidelines to avoid doing so if possible. These generally fall into the category of extrinsic causes of bradycardia:

- Patients with cough, micturition, and bradycardia or syncope should be advised to seek treatment to minimize triggers and to have access to support rails. Patients should lie down when lightheaded and should call 911 for persistent or particularly severe symptoms.
- Patients with carotid sinus hypersensitivity should avoid wearing tight ties and buttoned collars.
- Patients with documented sensitivity to medications that affect the sinus node or the atrioventricular node (or both) should report this fact when being prescribed any new medications.

REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.

PITFALLS

Bradycardia is readily identified because of the heart rate being an essential component of triage and monitoring of vital signs. The diagnosis is dependent on accurate interpretation of the ECG in the context of the patient’s overall medical findings. Treatment “failures” are often due to the clinician’s focus on the bradyarrhythmia without consideration of the underlying cause. When the bradycardia is not causing hemodynamic instability, it does not need to be “treated” but remains an important clue to underlying conditions, such as medication effects, elevated intracranial pressure, pulmonary hypertension, or cardiac ischemia.
REFERENCES