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The Clinical Challenge of Bradycardia: Diagnosis, Evaluation, and Intervention in the Emergency Department

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Bradycardic patients present unique challenges for the emergency department (ED) physician. First, the clinician must determine the stability of the patient and promptly institute any necessary resuscitation measures before proceeding with further evaluation and work-up of the dysrhythmia. Naturally, consideration always should be given to age-related and/or situational parameters associated with bradycardic syndromes. For example, the athletic, 24-year-old female with an ankle sprain, a heart rate of 44, and a blood pressure of 90/60 mmHg requires a much different approach than the 74 year-old presenting with chest pain and the same vital signs. After hemodynamic stability is achieved, the etiology of the bradydysrhythmia can be investigated in more detail.

Defining a bradycardic rhythm requires a working knowledge of the cardiac conduction system and an understanding of the various electrocardiographic manifestations of bradycardia. Moreover, bradycardia is associated with an extensive differential diagnosis. In this regard, after such critical entities as acute myocardial infarction, sepsis, hypoxia, hypoglycemia, and hypothermia have been considered, the differential expands to include a wide variety of other cardiac and systemic conditions. Finally, evaluation and treatment options should be revisited to ensure the underlying condition was addressed in the therapeutic plan.

The following review of the emergency evaluation and treatment of bradycardia includes a practically oriented discussion of the relevant anatomy, followed by a discussion of the electrocardiographic spectrum of bradydysrhythmias. The etiology and pathophysiology of bradycardia will be discussed, considering both cardiac and extracardiac causes. Finally, a review of pharmacologic and nonpharmacologic treatment modalities will be presented.

— The Editor

Cardiac Conduction System: Anatomy and Electrophysiology

A basic understanding of the cardiac conduction system requires a knowledge of relevant structures and their connections, as well as the blood supply to these structures. *(See Table 1.)* Normally, impulse conduction commences at the sinoatrial (SA) node, which is located at the junction of the superior vena cava and the right atrium. The SA node is generously innervated by both branches of the autonomic nervous system. The cardiac impulse next traverses intra-atrial fibers to reach the atrioventricular (AV) node, which is located above the ostium of the

coronary sinus at the base of the interatrial septum on the posteromedial portion of the right atrial wall.

Emerging from the AV node is the bundle of His, which enters the fibrous skeleton of the heart and runs anteriorly across the membranous interventricular septum. The His bundle then subdivides into right and left bundle branches. The right bundle branch courses along the interventricular septum toward the base of the anterior papillary muscle of the right ventricle. The flatter, broader left bundle branch runs along the septal wall toward the apex of the heart, dividing into an anterior and posterior fascicle. The former passes along the left ventricular outflow tract toward the anterior papillary muscle of the left ventricle, and the latter courses toward the posterior papillary muscle. The His-Purkinje system is composed of the myriad branches of the left and right bundle branches.[1,2]

The electrophysiologic manifestations of the anatomical configuration of the conduction system are observed on the electrocardiogram. The duration of the P wave reflects the duration of atrial activation, which proceeds from right atrium to left. The PR interval represents the time interval between the onset of atrial depolarization and the beginning of ventricular depolarization. During this time, the impulse travels from the atria (not including the time it took to move from the SA node itself to the right atrium), through the AV node, the bundle of His, the bundle branches, and the Purkinje fibers, until the ventricular myocardium begins to depolarize.

Ventricular activation is reflected in the duration of the QRS complex.[3] The frequency of P waves, the relationship between P waves and other activation sites, or the absence of the P waves, analyzed along with length of the PR interval, are the most important parameters for assessing bradydysrhythmias. Stated differently, bradydysrhythmias are the result of sinus node dysfunction or AV conduction disorders; these, in turn, are affected by diseases (e.g., myocardial ischemia), conditions (e.g., hypothermia), or drugs (e.g., digoxin) that affect the automaticity and refractoriness of cardiac cells, and conduction of impulses within the system.[1]

Electrocardiographic Analysis

Regular Bradycardia. Sinus bradycardia (SB) is defined as the presence of sinus rhythm with a rate less than 60 beats per minute (bpm); that is, the P wave morphology, the PR interval, and the P-P intervals are all uniform and normal.[1] *(See Figure 1.)* The parameters of so-called "normality" must be flexible, however, inasmuch as rates less than 60 bpm may be normal for some individuals. For example, highly trained endurance athletes may exhibit a resting sinus bradycardia of 30 to 40 bpm, due to increased vagal tone.[1,3]

In some cases, lower pacemakers may assume control of rhythm in the absence of sinus node activity. A junctional rhythm is by definition bradycardic, and is characterized by a regular, narrow QRS complex rhythm at a rate between 40 to 60 bpm *(See Figure 2.)* There may be no evidence of P waves, or retrograde P waves may be seen. Retrograde refers to conduction from, rather than to, the AV node, and the retrograde P may come before, after, or during the QRS complex on the ECG.[3] Junctional rhythms may emanate from the AV node, in which case the rate is higher (45 to 60 bpm). In contrast, slower, narrow complex junctional rhythms (35 to 45 bpm) have been found to originate in the His bundle.[3] An idioventricular rhythm *(see Figure 3)* usually is between 30 and 40 bpm, but may be as slow as 20 or as rapid as 50 bpm. The QRS complexes are widened abnormally. If the new pacemaker site is located in the ventricular septum, however, the complexes may be close to the upper limits of normal in width. Preexisting intraventricular conduction delays can make a slow junctional escape rhythm indistinguishable from a fast idioventricular rhythm.[3]

Traditionally, the various degrees of AV block (first-, second-, and third-degree) are considered together in a discussion of AV conduction defects. It may be useful to consider bradycardias as regular vs. irregular, as we do the tachycardias. Accordingly, first- and third-degree AV block can be considered here. First-degree AV block *(see Figure 4)* is a regular rhythm;

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Table 1. Vascular Supply of the Cardiac Conduction System[1,2]

STRUCTURE	IMMEDIATE BLOOD SUPPLY	CORONARY VASCULAR SUPPLY
SA node	sinus nodal artery	RCA 60% LCX 40%
AV node	AV nodal artery	PDA (of RCA) 90% LCX 10%
Bundle of His	AV nodal artery	PDA (of RCA) 90% LCX 10%
	Septal perforating arteries	LAD
Right bundle branch	AV nodal artery	PDA (of RCA) 90% LCX 10%
	Septal perforating arteries	LAD
Left bundle branch		
Anterior fascicle	Septal perforating arteries	LAD
Posterior fascicle		LAD PDA (of RCA)

Key:
RCA = right coronary artery
LCX = left circumflex artery
PDA = posterior descending artery
LAD = left anterior descending artery
Percentages refer to inter-, rather than intra-individual variations in blood supply.

the P-P and R-R intervals are uniform. The PR interval is prolonged, by definition, to greater than 0.20 seconds; this may be uniform on a tracing or there may be some variability in the degree of prolongation, which may be linked to changes in heart rate and vagal tone.[3] Electrophysiologically, the delay in first-degree AV *block* may be in the atrial fibers, the AV node, or the His-Purkinje system.

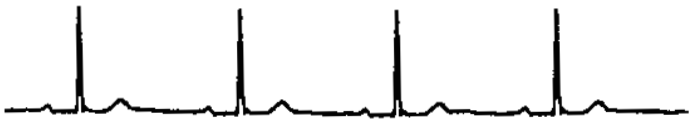
Third-degree AV block (complete heart block) also is a regular rhythm. *(See Figure 5.)* The P-P and R-R intervals are constant (unless there is sinus arrhythmia, with some variability then being seen in the P-P intervals), but the PR intervals are variable. This is considered by some to be a type of AV dissociation, as the two chambers are functioning independently of each other. Others state that true AV dissociation exists only when increased automaticity of a lower pacemaker renders its rate faster than that of the atria, leaving the ventricle refractory to activation from the slower atrial impulses, as in the case of ventricular tachycardia.[3] In complete heart block, the atrial pacemaker can be either sinus or ectopic, and may be tachycardic or bradycardic, but it is faster than the lower pacemaker, which can be either junctional or ventricular. *(See Figure 6.)* The location of that lower pacemaker determines the width of the QRS complexes and the rate of ventricular contraction, as previously discussed.[1,3]

Irregular Bradycardias. Sinus arrhythmia is defined as normal, consistent P wave morphology with variation in the P-P interval of greater than 0.16 seconds. This arrhythmia is seen more commonly in the context of slow rates, so it deserves discussion within the context of sinus bradyarrhythmias. Two types exist: respiratory sinus arrhythmia is the more common variant, in which the rate slows with expiration and speeds up with inspiration. This phasic change results from pulmonary reflex-mediated changes in vagal tone. The less common, non-respiratory sinus arrhythmia has an unknown mechanism, and is more often seen in patients with heart disease.[3] It should be noted that sinus arrhythmia also can present with non-bradycardic rhythms.

Sinus pause and sinus arrest are related entities, primarily differing in the degree rate slowing. Sinus pause is characterized by a transient cessation of impulse generation from the SA node; if this is prolonged, it is classified as sinus arrest. The pathophysiological distinction is not always clear.[3] The sinus pause may be terminated by a sinus beat, an AV junctional complex, or a ventricular escape beat, depending on the length of the pause. Thus, the rhythm is irregular, and bradycardic during the period of pause. There may be periods of other rhythms on a prolonged rhythm strip (e.g., normal sinus rhythm), which are punctuated by bradycardic interludes.

Sinoatrial block (SA block, or SA exit block) is a somewhat confusing entity. The confusion probably stems from the fact

Figure 1. Sinus Bradycardia



Sinus bradycardia is noted when the ventricular rate is less than 60 bpm, the QRS complex is narrow, the rhythm is regular, and each P wave is associated with a QRS complex.

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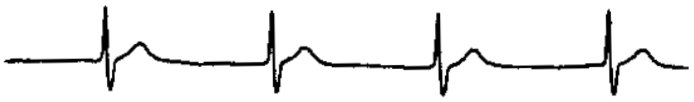
Figure 2. Junctional Bradycardia



A junctional rhythm is noted with a narrow-QRS complex and ventricular rates of 45-60 bpm.

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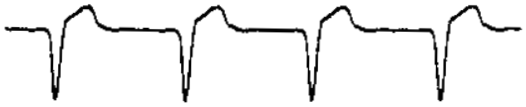
Figure 3. Idioventricular Bradycardia



An idioventricular rhythm is noted if the focus of the escape rhythm is found in the His-bundle branch block system; the QRS complex is wide with a rate of 30-45 bpm.

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Figure 4. First-Degree Atrioventricular Block

The PR interval is prolonged with a duration greater than 0.20 sec and is constant without progressive change with both a normal P wave and QRS complex. Every atrial impulse is conducted to the ventricles.

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that it is difficult to detect on the normal 12-lead surface ECG; in fact, it can only be inferred from the P wave activity.[3] As with AV block, there are three degrees of SA block. Only one variant (second-degree SA block; see next section) can be detected in the ED using 12-lead ECG. First-degree SA block cannot be distinguished on a standard ECG; this entity reflects prolonged conduction time from the SA node to the surrounding atrial tissue, but the P-P, R-R, and PR intervals are uniform. There is a gap in time between SA node impulse formation and P wave occurrence, but the emergency physician cannot detect this by evaluating the ECG at the bedside. Similarly, third-degree SA block cannot be distinguished from sinus arrest at the bedside.

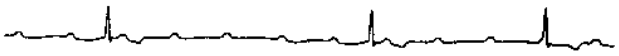
The only SA block that can be distinguished on a standard 12-lead ECG is an irregular rhythm, second-degree SA block. In this conduction disturbance, there is intermittent failure to conduct sinus impulses to the surrounding atrial tissue, and an intermittent absence of the P-QRS-T sequence. The "long cycle" between P waves usually is close to a multiple of the basic P-P interval, the duration of the long cycle being just short of the exact multiple of the P-P interval. As such, it may be difficult to distinguish from marked sinus arrhythmia, blocked premature atrial contractions, and periods of sinus pause.[1,3]

Second-degree AV block is usually, but not always, manifested by an irregular rhythm. There are two types of second-degree AV block: Mobitz Type I and Mobitz Type II. Both feature intermittent failure of atrial impulses to reach the ventricles (i.e., some of the P waves are not followed by QRS complexes). Mobitz Type I features "grouped beating," meaning the QRS complexes appear in groups, separated by a pause, which produces the irregularity. *(See Figure 7.)* The morphology of the P waves and the QRS complexes generally are normal. However, the PR interval progressively lengthens until an atrial impulse cannot reach the ventricle, in which case a "dropped beat" occurs; this represents a nonconducted P wave. An additional feature is a progressive shortening of the RR interval as the PR interval gets progressively longer.

Taken together, these characteristics are known as the Wenkebach phenomenon. In some cases, there is Type I second-degree AV block without the typical Wenkebach phenomenon, i.e. there is no progressive lengthening of the PR intervals before the dropped QRS. The PR interval may be variable and the RR interval may not progressively shorten. However, in this variety of Mobitz Type I, the biggest increase in the PR interval may be seen just before the nonconducted P, and the shortest PR interval occurs after the nonconducted P.[3] The less commonly encountered Mobitz type II AV block also features intermittently blocked P waves, but in contrast to Type I, the PR intervals are constant in the conducted beats; whether normal or prolonged, they are uniform. *(See Figure 8.)* Most patients with Type II AV block have an associated bundle branch block, meaning the block is distal to the His bundle; rarely (approximately one-third of the time) the block is in the bundle of His or in the AV node, rendering a narrow complex QRS.[3] The occasional dropped beats lead to interludes of intermittent bradydysrhythmia.

The magnitude of AV block is expressed as a ratio of P waves to QRS complexes, termed the conduction ratio. When the conduction ratio is 2:1, it is impossible to differentiate the two types of second-degree AV block, since the conduction of every other P wave is blocked; thus, the PR interval cannot be assessed for lengthening. *(See Figure 9.)* This may simulate SB, especially if

Figure 5. Third-Degree Atrioventricular Block with Narrow QRS Complex



No meaningful relationship is noted between the P waves and the QRS complex. The P waves will appear in a regular rhythm and will "match" through the rhythm strip at a specific atrial rate. The QRS complexes should appear in a regular fashion and will also "march" through the rhythm strip. The duration of the QRS complex and the ventricular escape rhythm is located near the His bundle, the rate is greater than 40 bpm, and the QRS complexes tend to be narrow.

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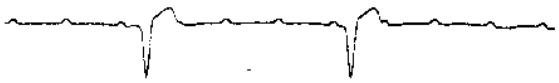
Figure 6. Third-Degree AVB with Widened QRS Complex



When the site of escape is distal to the His bundle, the rate tends to be less than 40 bpm and the QRS complexes tend to be wide.

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the nonconducted P waves are buried in the preceding T waves.[3] Moreover, like SB, 2:1 AV block is a generally regular rhythm; if there is variability of the conduction ratio over time (on a long rhythm strip), irregularity will become evident, and the differentiation between Mobitz I and II may be clearer.

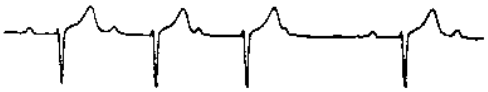
Differential Diagnosis: Cardiac and Non-Cardiac Causes of Bradycardia

The differential diagnosis of bradycardia is extensive, and can be considered in a number of ways. The following discussion will examine cardiac causes (ischemic and nonischemic), which will be followed by a section on extracardiac causes.

Ischemia. A variety of bradydysrhythmias (bradycardia and conduction block) may complicate acute myocardial infarction (AMI). SB occurs in about 40% of patients with AMI; junctional (20%) and idioventricular (15%) dysrhythmias occur far less frequently. First-, second- (Mobitz Type I), and third-degree AV blocks occur in approximately 15%, 12%, and 8% of AMIs, respectively; Mobitz Type II AV block is very unusual.[4,5] When considering only the unstable bradydysrhythmias, third-degree AV block is the most frequently encountered in this setting (40%). SB (25%) and junctional rhythm (20%) follow complete heart block in frequency of unstable rhythms in AMI.[4,5]

The pathophysiology of bradydysrhythmia in AMI can be multifactorial. In most cases, ischemia or infarction of cardiac tissue is the underlying cause. Other mechanisms that may be associated with the AMI include: hyperkalemia, hypoxia, acidosis, local increase in adenosine concentration, variations in autonomic control, and pharmacotherapy (especially calcium channel antagonists and beta adrenergic blockers). Occurrence of bradydysrhythmia in AMI also varies depending upon the location of the coronary vascular lesion. Inferior AMI is more frequently complicated by bradydysrhythmias; in most cases, this results from occlusion of the right coronary artery (RCA).[3] During the first six hours post-AMI, patients also may abruptly develop compromising bradydysrhythmias due to increased parasympathetic tone; this is characterized by a slow ventricular escape rate, which usually responds well to atropine. After six hours, bradydysrhythmias tend to develop slowly, and may consist of slow ventricular escape rates, but with a poorer response to pharmacotherapy.[6]

Figure 7. Second-Degree, Type I Atrioventricular Block (Wenkebach block)



The PR interval is often normal in the first beat of the series. Progressive PR interval lengthening with subsequent beats is ovserved until an impulse is unable to reach the ventricles, resulting in a nonconducted P wave. After the dropped beat, the PR interval returns to normal and the cycle repeats. P-waves and QRS complexes are usually normal in terms of morphology and total duration. A pattern to the RR interval is also seen with RR interval becoming increasingly shorter. Following the dropped beat, the RR interval in the subsequent beats tend to shorten. One will also notice on the rhythm strip a grouping of beats, referred to as grouped beating of Wenkebach.

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Bradycardic conduction disturbances, such as the various AV blocks, also may develop in anterior and anteroseptal infarction, which typically are due to occlusion of the left main coronary artery, the left anterior descending artery (LAD), or its branches. These cases are associated with a poorer response to pharmacotherapy and a worse prognosis.[3] The risk of developing complete heart block, regardless of infarct site, has been found to directly correlate with the degree of conduction disease evidenced by the 12-lead ECG in AMI; the more conduction defects (first- or second-degree AV blocks, bundle branch, or fascicular blocks) occurring on the tracing, the higher the risk of progression to complete heart block.[7] This can have implications for the use of prophylactic ventricular pacing in the setting of AMI.

Nonischemic Cardiac Causes. These disorders are not necessarily directly related to acute cardiac ischemia, injury, or infarct. Sick sinus syndrome (SSS) is a disorder of sinus node impulse formation, or transmission to the atria. It has a variety of electrocardiographic manifestations, including: 1) severe bradycardia; 2) periods of sinus pause or arrest; 3) periods of sinus exit

Figure 8. Second-Degree, Type II Atrioventricular Block with 3:2 Conduction

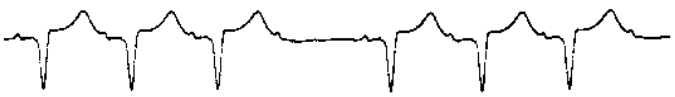

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Figure 9. Second-Degree, Type II Atrioventricular Block with 2:1 Conduction

The PR interval is constant without progressive lengthening and may be either normal or prolonged. The QRS complex is widened in most instances. The magnitude of the AVB is expressed as a ration of P waves to QRS complexes. For example, if there are two P waves for every QRS complex, then it is a 2:1 block as seen here. Distinguishing type I from type II block is relatively straightforward unless there is 2:1 conduction as seen in this example. In this situation, there is no way to compare the PR intervals for the conducted beats and hence make the distinction between Mobitz Type I and type II AVBs. The width of the QRS complex in Mobitz Type II block, however, may give the clinician information as to the level of block; Type II AVB is usually characterized by an escape rhythm with a widened QRS comples. Nonetheless, in situations of 2:1 conduction the physician should initially assume the block is a Mobitz II unless proven otherwise, owing to the relatively malignant couse of type II AVB.

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block (SA block); 4) alternating bursts of varying atrial tachycardias and bradycardia (the tachy-brady syndrome); and 5) AV junctional escape rhythms.[3,8,9] These manifestations may occur alone or in combination and are often intermittent, making ambulatory electrocardiographic monitoring a more sensitive tool than a single 12-lead ECG for detection of SSS.[3] SSS is more common in the elderly population, but can be seen in younger patients.

Commonly, there is histopathological evidence of fibrous destruction of nodal and internodal tissue, which is often independent of coronary artery disease.[3,4,5,8-10] Causes of SSS include primary, idiopathic disease as well as ischemic cardiac disease and a multitude of other factors (e.g., rheumatologic, oncologic, metabolic, infectious, structural, and iatrogenic) discussed below.[3] Lenegre's disease is an idiopathic degenerative condition featuring fibrotic changes of the conduction system that may advance to calcification; bradycardia and varying degrees of AV block may be found on the ECG, along with other conduction disease manifestations, such as right bundle branch block and left anterior fascicular block.

Due to similar presentations, Lenegre's disease is often lumped with Lev's disease, another fibrosclerotic process. In this condition, normal sclerotic changes of the cardiac skeletal structures seen with advanced aging also involve the adjacent conduction system.[3,10,11] Although rare (approximately 1 in 22,000 live births), congenital complete AV block has been described, and has been associated with maternal rheumatologic disease, especially systemic lupus erythematosus.[12]

Pharmacologic/Toxicologic. Several medications, most of which are used for treatment of cardiovascular disease, can cause bradycardia when taken in therapeutic amounts. These include some calcium channel antagonists, beta-adrenergic blockers, digoxin, alpha$_2$-adrenergic agonists (e.g., clonidine, methyldopa), and cholinergic agents. It follows that these same agents have the propensity to cause profound bradydysrhythmias when ingested in toxic amounts; indeed, pharmacotoxicity, together with ischemic disease and degenerative processes, account for the majority of cases of AV block.[9]

Virtually all calcium antagonists and beta-adrenergic blockers can cause severe bradycardia and advanced heart block in overdose;[13] among the former group, verapamil has been found to more frequently and profoundly cause these bradydysrhythmias when compared to diltiazem and nifedipine in overdose situations.[14] Amlodipine also has been reported to cause bradycardia in a poisoning scenario.[15]

Digoxin has been reported to cause bradycardia, sinus arrest, SA block, and all degrees of AV block in the setting of overdose; conversely, it also may cause supraventricular or ventricular tachydysrhythmias. Early in acute digoxin toxicity, the depression of SA node function or impairment of AV nodal conduction may respond to atropine; later this response may be lost. One indication for administration of digoxin-specific antibody fragments is progressive bradydysrhythmia unresponsive to atropine.[16] A variety of botanicals with cardiac glycoside activity also have been reported to cause bradydysrhythmias, among them: foxglove, lily-of-the-valley, yew berry, dogbane, Siberian ginseng, and squill.[16-18] Although classically linked with wide-complex tachydysrhythmias, the Type Ia antiarrhythmics (quinidine, disopyramide, and procainamide) and drugs that may behave like them in overdose (e.g., Type Ic antiarrhythmics, tricyclic antidepressants, carbamazepine, quinine, chloroquine, propoxyphene, amantadine, cyclobenzaprine, and thioridazine) may produce heart block.[19]

Opioids, sedative-hypnotics, and alpha$_2$-adrenergic agonists act via central nervous system-mediated mechanisms to produce bradycardia.[19] Methyldopa has been postulated to cause both direct cardiac and central nervous system-mediated bradycardia and first-and second-degree AV block.[19,20] Other pharmacologic agents linked with bradycardia include the following: organophosphates, cholinesterase inhibitors, lithium, phenothiazines, and cocaine.[10,19,21] The latter drug, although classically associated with a hyperadrenergic state and tachycardia, also has been found to cause bradycardia at low doses, and may behave like a type Ia antiarrhythmic in toxic situations.[22] Cardiac conduction abnormalities are not a feature of oral phenytoin overdose;[23] however, the propylene glycol carrier it is mixed with for intravenous delivery has been implicated for concentration- and infusion-rate related bradycardia and heart block.[24]

Neurologic Disorders. Traumatic as well as non-traumatic neurological processes may result in bradydysrhythmias; among the former, trauma to the brain and spinal cord have been implicated. Severe head trauma leading to profound injury

Table 2. Infectious Causes of Myocarditis with Potential for Bradydysrhythmia[10,21,28-32]

VIRAL		
Coxsackie B virus	Influenza	Mononucleosis
Hepatitis	Mumps	Rubella
Rubeola	Varicella	Respiratory syncytial virus
BACTERIAL		
Streptococcus	Meningococcus	Mycoplasma
Staphylococcus	Diphtheria	
OTHER		
Trypanosomiasis (Chagus disease)	Syphilis	Lyme disease

Table 3. Rheumatologic Diseases Affecting the Heart with Potential for Bradydysrhythmia[33-35]

- Rheumatoid arthritis
- Scleroderma
- Systemic lupus erythematosus
- Polymyositis
- Reiter's syndrome
- Ankylosing spondylitis
- Sjögren's syndrome
- Behcet's disease
- Wegener's granulomatosis

and increased intracranial pressure may be accompanied by systemic hypertension and bradycardia (i.e., the Cushing's reflex, which carries a poor prognosis).[10] Severe bradycardia also can be seen after acute injury to the cervical cord; the bradycardia is mediated by disruption of the autonomic nervous system. Specifically, disruption of the sympathetic fibers in this region can cause unopposed parasympathetic effects, leading to slowing of the heart rate.[10] Studies have shown that patients with profound injury to the cervical cord display bradycardia for up to 2-6 weeks post-trauma; vagal stimulatory maneuvers (e.g., suctioning) may exacerbate this bradycardia, resulting in sinus pauses.[25]

Other neurologic diseases associated with bradydysrhythmias include cerebrovascular accident, subarachnoid hemorrhage, seizure (ictal bradycardia syndrome), and Guillain Barré syndrome.[10,26,27] In the first two entities, increased intracranial pressure must be suspected when hypertension also is present. This also should be suspected if vital signs consistent with the Cushing's reflex are seen after seizure and there is a possibility of occult head trauma. Most cases of ictal bradycardia syndrome are seen with temporal lobe seizures; the conduction pauses that may occur can make it difficult to discern whether loss of consciousness was due to a primary cardiac event or the ictal bradycardia syndrome.[26] Autonomic nervous system dysfunction is felt to be responsible for the brady- or tachydysrhythmias, which are associated with severe cases of Guillain Barré syndrome; sinus bradycardia and AV block are both potential cardiac manifestations of the disease.[27]

Infectious Etiologies. Myocarditis can produce a variety of electrocardiographic manifestations, including ST segment and T wave changes, and less commonly, AV block of varying degrees.[3,21,28] A variety of infectious agents cause these changes via differing mechanisms, including direct tissue involvement, immune-mediated toxicity, and toxin-mediated effects. Myriad infectious agents have been linked to myocarditis. *(See Table 2.)* Chagas disease can cause both a congestive cardiomyopathy and dysrhythmias, including ventricular dysrhythmias and conduction disease such as AV block.[29] Lyme disease deserves special attention. Up to 10% of patients will develop clinically evident, usually transient, cardiac involvement, and the most common cardiac pathology is AV block. Degrees of heart block may fluctuate within a given patient, including the development of third-degree AV block.[21,30-32]

Rheumatologic Conditions. Most connective tissue disorders can feature involvement of the heart (see Table 3), and although pericarditis is the most common rheumatologic manifestation in some (e.g., rheumatoid arthritis, systemic lupus erythematosus, and scleroderma), varying degrees of conduction system disease may occur, including AV block.[33-35] Heart block may be seen in up to 25% of patients with Reiter's syndrome, and up to 10% of those with rheumatoid arthritis.[33]

Miscellaneous. Due to its multisystem, infiltrative course, the granulomatous disease sarcoidosis has a propensity to cause heart block. Twenty-five percent of sarcoid patients have histologic evidence of cardiac involvement, with only about one-fifth of those showing clinical cardiac manifestations.[28,36] Although a variety of cardiac pathologies can be seen with sarcoidosis (e.g., conduction system disease—congestive and restrictive cardiomyopathy, supraventricular and ventricular dysrhythmias—the most common presenting cardiac manifestation is complete heart block. The disease has a predilection for involving the interventricular septum.[36] Amyloidosis, a rare systemic infiltrative disease, usually affects the heart by causing a restrictive cardiomyopathy, and the vascular system by inducing orthostatic hypotension. However, it also can cause bradycardia, SSS, and AV block.[28]

Hypothyroidism and hypothermia may produce bradycardia. Hypoadrenalism, hyperparathyroidism, and acromegaly all have been described with bradycardia. Marked electrolytes elevations, including hyperkalemia, hypermagnesemia, and hypercalcemia, also can cause bradydysrhythmias.[10] Hypoglycemia is an unusual cause of compromising bradycardia; neurologic and endocrinologic mediation has been postulated.[37] Among iatrogenic causes, radiation therapy has induced conduction system disease, including heart block, but more commonly damages the pericardium.[38,39] Rejection and ischemia are felt to be responsible for bradydysrhythmias occasionally seen after orthotopic heart transplant.[40]

Reflex-Mediated Causes. Finally, a number of neuroreflexive processes should be considered in the differential diagnosis of bradydysrhythmias; these are not disease processes, but rather cardiac responses to stimuli via neurologic pathways. Most commonly encountered is the vasovagal event. Here, tran-

sient bradycardia sometimes leading to syncope occurs, and usually is associated with a painful, stressful, or emetic event.[9,41]

There are two types of hypersensitive carotid sinus syndrome: cardioinhibitory and vasodepressor. The former is manifested by more than three seconds of ventricular asystole during stimulation of the carotid sinus, and results from an oversensitivity of the afferent carotid sinus nerves leading to the efferent vagal response. Cardioinhibitory and vasodepressor responses can occur independently or concomitantly.[9,10] Less commonly seen reflex-mediated bradydysrhythmias sharing a common afferent (fifth cranial nerve) and efferent (10th cranial nerve) pathway include the diving reflex (cold water to the upper face); oculocardiac reflex (trauma or pressure applied to the globe); and maxillofacial reflex (surgical manipulation of the maxillofacial or temperomandibular regions).[10,42,43] Other reflexive mechanisms known to cause bradycardia include rectoprostatic massage; deglutition, micturition, and defecation syncope; and glossopharyngeal neuralgia.[10]

Pharmacotherapy of Bradycardia

Atropine. Generally the first-line therapy for symptomatic bradydysrhythmia, atropine enhances the automaticity of the SA node and AV nodal conduction via a vagolytic mechanism. Indications for atropine include seriously symptomatic sinus bradycardia or AV block, and asystolic cardiac arrest. In adults, the initial dose is 0.5 mg to 1.0 mg intravenously, repeated every five minutes if needed to a maximum vagolytic dose of 0.04 mg/kg (3 mg in a 75 kg patient).[44] The pediatric dose is 0.01 mg/kg. Generally, patients are more likely to respond to the first dose than to subsequent doses, and bradycardia responds more readily than does AV block.[4,5]

There are two major concerns when considering administration of atropine to a patient with symptomatic bradydysrhythmia. One concern is the potential exacerbation of underlying coronary ischemia and precipitation of malignant ventricular dysrhythmias, and the other is the possibility of paradoxical slowing of the rhythm after atropine is given. Regarding the first issue, although atropine may worsen ischemia if given during an acute coronary event,[44,45] so too might hypoperfusion from an unstable bradydysrhythmia adversely affect outcome. Recent data did not reveal evidence of intensification of cardiac ischemia in patients receiving atropine for compromising bradydysrhythmias.[4,5] Development of malignant ventricular dysrhythmias has been found infrequently after administration of atropine for unstable bradydysrhythmia in the prehospital setting, and has a reported incidence of 2-4%.[4,5,46] Knowing the minimal risk associated with this therapy, the emergency physician should nevertheless be prepared and use the agent cautiously.[47-50]

Paradoxical slowing of the heart rate after administration of atropine for unstable bradydysrhythmia has been found rarely in patients with infranodal AV block—Mobitz Type II second-degree AV block and third-degree AV block with a wide QRS complex. The majority of patients with these rhythms do not manifest this paradoxical reaction, however; an awareness of this possible adverse effect in this subgroup of bradydysrhythmias is advisable.[4,5,46,49,51-54]

Isoproterenol. A non-specific beta-adrenergic agonist, isoproterenol causes an increase in chronotropy, inotropy, and conduction velocity via beta-$_1$ receptor stimulation, and smooth muscle relaxation and bronchodilation via beta-$_2$ stimulation. Physiologically, the result is an increase in cardiac output, systolic blood pressure, and myocardial contractility, with a decrease in diastolic blood pressure, systemic vascular resistance, and pulmonary vascular resistance. Although the mean arterial pressure should not be significantly affected, myocardial oxygen demand is increased (due to beta$_1$ effects), and coronary perfusion pressure is decreased (due to beta$_2$ effects). Therefore, isoproterenol is not the drug of choice in situations of acute coronary ischemia, and is relegated to a reserve role—as a bridge, only if needed, between atropine failure or maximization, and pacing, which is actually preferred.

Isoproterenol does have a role in the patient with symptomatic bradydysrhythmia and a denervated, transplanted heart.[44] It should be avoided in digoxin-toxic patients, as increased ventricular ectopy may result.[16] The dose is 2.0 mcg/min, with titration to hemodynamic effect, to a maximum infusion of 10 mcg/min. The half life and duration of effect are short; side effects are predictable from the pharmacology of the drug: tachydysrhythmias, palpitations, ischemia, flushing, dizziness, tremor, and nausea.[44]

Glucagon. This naturally occurring hormone has positive inotropic and chronotropic effects, actions that are not mediated by adrenergic receptors. Inotropic effects exceed chronotropic effects in the failing heart; the reverse is true under normal conditions (in both cases with limited expense of myocardial oxygen consumption).[55] Glucagon has a demonstrated beneficial effect in bradydysrhythmias resulting from calcium channel blocker and beta adrenergic antagonist toxicity;[13,14,56,57] many consider it first-line therapy in these cases, yet reports of its success have been mixed.[55] Recommended dose ranges in the setting of toxicity from these drugs are variable; generally, an initial bolus of 2-10 mg intravenously is suggested, with continuous infusion if necessary at 2-5 mg/hr. In cases requiring large doses, the phenol diluent that accompanies the preparation should be replaced by sterile water, saline, or D_5W to avoid phenol toxicity. In infants, the bolus dose is 50 mcg/kg. Adverse effects include nausea, emesis, hypokalemia, and inconsequential hyperglycemia.[55]

Aminophylline. Because adenosine has been postulated to contribute to some conduction abnormalities seen during acute myocardial infarction, aminophylline—its competitive inhibitor—may play a role in the management of some bradydysrhythmias in this setting.[58] Adenosine appears to be released from ischemic myocardial cells, resulting in prolongation of AV nodal conduction time. The dose is 5-15 mg/kg infused over five minutes. As with isoproterenol, it functions as a medical bridge to more definitive therapy of atropine-resistant bradydysrhythmias—namely, cardiac pacing.

Emergency Cardiac Pacing

Transcutaneous Cardiac Pacing. This technique has evolved in both simplicity and efficacy over time. Indications are strati-

Table 4. Electrocardiographic Waveforms Seen During Transvenous Pacemaker Placement[65-67]

LOCATION	WAVEFORM	
	P wave	***QRS complex***
Superior vena cava	negative	negative
High right atrium	larger/negative	smaller/negative
Mid-right atrium	larger/biphasic	smaller/negative
Low right atrium	smaller/positive	larger/negative
Right ventricle, free	smaller/positive	larger/negative
Right ventricle, against wall		ST segment elevation
Inferior vena cava	smaller/positive	smaller again/negative

fied into those for prophylactic placement without active pacing, and those for active pacing. Prophylactic placement of transcutaneous pacing apparatus is indicated for stable bradydysrhythmias or conduction disturbances with potential for progression to instability, such as those occurring during acute myocardial infarction. Examples include significant sinus node dysfunction, Mobitz Type II second-degree AV block, third-degree AV block, and new conduction delays (e.g., bundle branch blocks or bifascicular block). Active pacing is indicated for bradydysrhythmias with instability (e.g., hypotension/shock, anginal chest pain or dyspnea, altered sensorium, pulmonary edema) that are unresponsive to medical therapy, and for bradyasystolic arrest.[44]

Pacing of asystolic cardiac arrest is often futile, however, and if instituted, should be employed early to optimize chances of a better outcome.[59,60] Prehospital transcutaneous pacing by emergency medical technicians for asystolic patients (before intubation and/or drug administration by later-arriving paramedics) has not been found to be effective.[61] It has been used in the prehospital arena with success, however, in patients with hemodynamically significant bradycardia.[62] Transcutaneous cardiac pacing is an interim therapy, and should serve only as a bridge to definitive transvenous pacing. Dog studies with pacing for up to 60 minutes have not shown enzyme, electrocardiographic, or histologic evidence of myocardial damage.[63]

Transcutaneous pacemakers generally require two connections to the patient: a set of pads for delivering the pacing current, and leads for monitoring the patient; some units allow both defibrillation and pacing through the same pads. The placement of the pads is generally anterior/posterior, with the anterior pad being as close as possible to the point of maximum cardiac impulse, and the posterior pad being directly opposite the anterior, in the left parathoracic region. When pacing an awake patient, current should be slowly increased until capture is attained—a palpable pulse that correlates with the waveform seen on the unit. In cardiac arrest, the maximum current should be applied initially to assure rapid capture; this can be titrated down to the minimal current that guarantees capture if the patient regains consciousness. Conscious patients may need intravenous medication for pain and anxiety management, as the current necessary may cause significant discomfort. Complications include pain, failure to detect underlying ventricular dysrhythmias (due to technical difficulties in systems without adequate dampening capability for pacing artifact), and theoretically, induction of dysrhythmias. Minor local tissue damage is possible; the magnitude of electrical shock delivered to health care personnel, even during CPR, is trivial.[64]

Transvenous Cardiac Pacing. This procedure yields more definitive control of bradydysrhythmias unresponsive to medical therapy or accompanied by serious signs and symptoms as discussed above. Although it is invasive, the actual delivery of current to pace the heart is better tolerated in most patients than with the transcutaneous method. Central venous access is the first step, and is optimally accomplished via the right internal jugular approach (leaving the left side clear for possible future permanent pacemaker placement). The right subclavian approach may be technically more difficult; the right femoral approach is another possibility, especially in coagulopathic patients in whom a compressible vessel is more desirable.

The transvenous wire optimally is placed with fluoroscopic guidance, which, as a rule, is often not available to the emergency physician who must perform the procedure emergently. Alternatively, the wire can be passed either blindly or semi-blindly with electrocardiographic guidance. The blind approach should be reserved for situations where electrocardiographic guidance is not readily available and the patient is in extremis. The operator should advance the balloon-tipped catheter approximately 10 cm into the venous port, and then turn the pulse generator to the "sense" mode, advancing into the ventricle until cardiac activity is sensed. At this time, the balloon is deflated, the generator is switched to the "pace" mode, and the current is advanced from a minimal setting, used during the first phase (e.g., < 0.2 mA), to a current that is likely to capture the ventricle (e.g., 4 to 5 mA). The monitor is then watched for electrical capture as the wire is advanced up to 10 cm further. If capture does not occur, the wire should be withdrawn the 10 cm distance, and readvanced. If the patient is asystolic, there is no reason to advance the wire in the "sense" mode; the pacing generator should be in the "pace" mode, asynchronous, at maximum output, and advanced looking for evidence of ventricular capture.[65-67]

In a patient with a heart beat, semi-blind placement of the pacing wire with electrocardiographic guidance is a better alternative when fluoroscopy is not available. In this case, the patient should be connected to the limb leads of the ECG machine. A precordial "V" lead from the ECG machine is then connected to the distal, negative terminal of the pacer wire, and serves as an intracardiac electrocardiographic monitor to ascertain location. Table 4 describes the waveforms seen at different points as the wire advances. The general concept is that wave forms grow larger as the chamber they correspond to is entered, and vector cardiographic rules are in place. The balloon should be deflated once in the right ventricle to avoid straying into the right ventricular outflow tract. The wire should then be advanced while monitoring the V lead, with the goal of engaging the wall of the right

ventricular apex. Once the operator is satisfied that the right ventricular wall is engaged, the V lead should be disconnected and the pacing generator attached to both pacing wire leads, and the patient can be paced. Chest radiography is used to verify positioning and exclude iatrogenic pneumothorax. On the anteroposterior film, the tip of the wire should be seen in the right ventricle, crossing the midline to the left. If a lateral film can be obtained, the tip of the catheter should be seen curling toward the distal aspect of the sternum. Complications include inappropriate positioning of the wire, induction of dysrhythmias, infection, thrombosis, phlebitis, and perforation of cardiovascular structures traversed en route. The complications related to central venous access also must be anticipated.[65-67]

Finally, pacemaker function should be assessed in terms of both pacing threshold and sensitivity. Pacing threshold is the smallest amount of current necessary to cause ventricular contraction. It is determined by setting the rate at least 10 bpm above the intrinsic heart rate, and then dialing the amperage down until capture is lost. With a properly positioned wire, this should be < 1.0 mA. This process should be repeated two or three times for reliability assessment. Once the threshold has been determined, the output should be set at approximately 2.5 times the threshold to assure capture. If the patient has an underlying rhythm, pacing sensitivity should be assessed. This is defined as the pacemaker's ability to sense adequate cardiac activity, and thus be inhibited. To test this, the pacemaker rate (in demand mode) should be dialed down to 10 to 20 bpm below the intrinsic heart rate; if sensing adequately, paced impulses should disappear from the monitor. Like pacing threshold, this too should be determined two or three times, looking for equivalent results. A post-procedure 12-lead ECG tracing should reveal a left bundle branch block pattern during paced beats if the wire is in the right ventricle.[65-67]

Summary

Bradydysrhythmias are frequently encountered in the ED, and at times require emergent therapy for stabilization. There are a wide variety of bradycardic rhythms, which are best assessed with a 12-lead EKG, and can be stratified into regular and irregular rhythms. While stabilizing the patient and defining the rhythm, the EP must consider a vast differential diagnosis of causality that includes much more than ischemia and infarction, although these are a paramount consideration. Treatment options include observing the rhythm, pharmacotherapy, and cardiac pacing, both transcutaneous and transvenous. Disposition decisions are best made when the nature of the rhythm and its etiology are determined, with consideration of the extent of therapy given to that point, and an assessment of the potential for instability in the immediate future.

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Physician CME Questions

41. Second-degree AV block, Mobitz Type I:
    A. is by definition unstable.
    B. features progressive shortening of the PR interval before nonconducted P wave appears.
    C. should be treated with atropine if it occurs during acute myocardial infarction, regardless of patient stability.
    D. is more commonly encountered than the Mobitz II variation.

42. Sinus bradycardia:
    A. is defined as the presence of sinus rhythm with a rate of less than 60 bpm.
    B. is frequently seen with phenylpropanolamine overdose.
    C. is usually unresponsive to atropine in acute myocardial infarction.
    D. is usually unresponsive to isoproterenol in acute myocardial infarction.

43. Which of the following may be a defining feature of sick sinus syndrome?
    A. Second-degree AV block, Mobitz Type I
    B. Ventricular parasystole
    C. Periods of sinus exit block
    D. Periods of nonsustained ventricular tachycardia

44. The only type of sinoatrial block (sinus exit block) readily distinguishable on the standard 12-lead ECG is:
    A. first-degree.
    B. irregular rhythm, second-degree.
    C. third-degree.
    D. fourth-degree.

45. The blood supply of the sinus nodal artery principally stems from:
    A. the left anterior decending coronary artery.
    B. a direct branch of the left main coronary artery.
    C. the right coronary or left circumflex coronary artery.
    D. the posterior decending branch of the left circumflex coronary artery.

46. While placing a transvenous cardiac pacemaker wire, a large biphasic P wave with an associated small negative QRS complex is seen on the V-lead being used to guide placement via the right internal jugular approach. The tip of the wire is most likely in the:
    A. superior vena cava.
    B. inferior vena cava.
    C. high right atrium.
    D. middle right atrium.

47. Transcutaneous cardiac pacing:
    A. should not be done in awake patients.
    B. ideally serves as a bridge to more definitive therapy with isoproterenol.
    C. is highly successful in asystolic cardiac arrest, if instituted by emergency medical technicians in the field.
    D. is best viewed as an interim therapy until more definitive transvenuous cardiac pacing can be instituted.

48. Which of the following drugs is associated with severe bradydysrhythmias after oral overdose?
    A. Verapamil
    B. Phenytoin
    C. Metformin
    D. Captopril

In Future Issues:

Pneumonia in the Geriatric Adult