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Evaluation And Management Of Bradydysrhythmias In The Emergency Department

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Abstract

Bradydysrhythmias represent a collection of cardiac conduction abnormalities that span the spectrum of emergency presentations, from relatively benign conditions to conditions that represent serious, life-threatening emergencies. This review presents the electrocardiographic findings seen in common bradydysrhythmias and emphasizes prompt recognition of these patterns. Underlying etiologies that may accompany these conduction abnormalities are discussed, including bradydysrhythmias that are reflex mediated (including trauma induced) and those with metabolic, environmental, infectious, and toxicologic causes. Evidence regarding the management of bradydysrhythmias in the emergency department is limited; however, there are data to guide the approach to the unstable bradycardic patient. When decreased end-organ perfusion is present, the use of atropine, beta agonists, and transcutaneous or transvenous pacing may be required.

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CME Objectives

Upon completion of this article, you should be able to:

1. Recognize the electrocardiographic features of common bradydysrhythmias.
2. Consider a variety of pathologies that give rise to bradydysrhythmias.
3. Identify the emergent therapies for the unstable patient with bradycardia.
4. Be familiar with common antidotes for acute toxicities that result in bradydysrhythmias.

Prior to beginning this activity, see the back page for faculty disclosures and CME accreditation information.

Case Presentations

It is about 20 minutes into your shift when EMS arrives with a pleasant 80-year-old woman who has had a syncopal event. She describes standing in her home earlier today and becoming lightheaded and falling to the ground. She is now resting comfortably, and her vital signs are: blood pressure, 140/72 mm Hg; pulse rate, 74 beats/min; respiratory rate, 14 breaths/min; and oxygen saturation, 99% on room air. You have just begun to take her history when you are interrupted and called to your next patient...

You approach the bedside of a 27-year-old woman who is pale, diaphoretic, and writhing in pain. The only history you are able to obtain is that she has had mild lower abdominal pain for a few days that acutely worsened today. Initial vital signs are: blood pressure, 70/40 mm Hg; pulse rate, 58 beats/min; respiratory rate, 20 breaths/min; and oxygen saturation, 99% on room air. Your brief exam is significant for diffuse abdominal tenderness and guarding. You then hear a flurry of activity from the hallway...

Your next patient is being rushed down the hall on a stretcher. Brought in by a family member for intermittent lightheadedness and shortness of breath, this 64-year-old man is pale and diaphoretic, with depressed mental status. A quick check of his radial artery demonstrates a weak pulse with a palpable rate of approximately 40 beats/min. You quickly place him on the cardiac monitor and notice what appears to be a third-degree heart block. Initial vitals are: blood pressure, 82/40 mm Hg; pulse rate, 38 beats/min; respiratory rate, 18 breaths/min; and oxygen saturation, 98% on room air.

These 3 cases represent some of the variable presentations of patients with bradydysrhythmias. The underlying pathology for these patients ranges from the benign to the life threatening. You approach each case in a systematic manner, knowing that prompt evaluation, recognition, and treatment can make the difference.

Introduction

During the course of practice, all emergency clinicians will be faced with evaluating and managing patients with bradydysrhythmias. These disorders result from a wide range of underlying pathologies that must all be considered in the evaluation of patients who present to the emergency department (ED) with bradydysrhythmias. Assessments will be focused on identifying cardiovascular etiologies, but the thorough evaluation should not overlook traumatic, intracranial, and intra-abdominal processes. Prompt electrocardiogram (ECG) analysis often represents a key point of the emergent evaluation, and familiarity with ECG patterns is critical to clinical decision-making.

There is always the risk that patients with bradydysrhythmias will be hemodynamically unstable upon arrival or that they will become unstable during the course of their evaluation and treatment. While these scenarios may not occur on a regular basis, emergency clinicians must be prepared to provide emergent interventions, as needed, to stabilize these patients. Familiarity with resuscitation techniques and medications as well as procedures (including transvenous pacer placement) is central to management. This issue of *Emergency Medicine Practice* provides a systematic review of the literature on bradydysrhythmias and presents recommendations based on the best available evidence.

Critical Appraisal Of The Literature

A review of the literature on bradydysrhythmias proved to be challenging due to the vast number of articles mentioning relevant terms. The review was initially performed through PubMed and Ovid MEDLINE®, using the keywords *bradydysrhythmias, sinus bradycardia, sick sinus syndrome, tachy-brady syndrome, sinoatrial block, sinus arrest, first-degree heart block, second-degree heart block, Mobitz type I, Mobitz type II, Wenckebach, third-degree heart block, complete heart block, complete AV block, reflex-mediated bradycardia, relative bradycardia,* and *paroxysmal bradycardia.* While several hundred randomized controlled trials were initially identified, most involved long-term pacemaker implantation for patients with known cardiac dysfunction. Very few large trials involving the treatment of bradydysrhythmias in the emergency setting could be identified.

The Cochrane Database of Systematic Reviews was searched for entries regarding arrhythmias, and a single review was found that investigated the long-term use of medications and pacemakers for reflex-mediated syncope, although the data were insufficient to produce a firm conclusion. The consensus guidelines, recommendations, and algorithms that have been developed were adapted primarily from statements by the American College of Emergency Physicians, the American College of Cardiology, and the American Heart Association, as published by the National Guideline Clearinghouse (www.guideline.gov). Overall, the literature is sparse regarding high-quality studies for the evaluation and treatment of bradydysrhythmias in the emergency setting, and many of the recommendations are extrapolated from larger studies investigating the long-term management of these patients.

Pathophysiology And Rhythm Analysis

Bradydysrhythmias represent a variety of electrical abnormalities with a diverse set of etiologies and include ischemia and infarction, infiltrative processes, hypothermia, toxin-mediated causes, electrolyte abnormalities, age-related degeneration, and

many others. The unifying principle in each of these disorders (when symptomatic) is the inability of the cardiac conduction system to generate and/or transmit the appropriate electrical signals to generate sufficient cardiac output. It is this relative deficiency in perfusion, when compared to metabolic demands, that results in the patient's symptoms, which range from fatigue and malaise to altered mentation and frank syncope. Based on rhythm analysis, bradydysrhythmias can be classified into 2 broad categories: (1) sinus node dysfunction, and (2) atrioventricular blocks. **(See Table 1.)**

Sinus Node Dysfunction

Sinus node dysfunction, also referred to as *sick sinus syndrome*, includes a collection of electrical abnormalities that are characterized by the failure to generate appropriate cardiac potentials from the sinus node. This class of dysfunction, described as early as 1968, includes sinus bradycardia and sinus arrest.[1] Sinus bradycardia is defined by a ventricular rate < 60 beats/min with regular, identifiable P waves preceding each QRS and a normal PR interval. **(See Figure 1.)** In comparison, sinus arrest is defined by absent atrial depolarization for a period of time before the resumption of regular cardiac conduction. **(See Figure 2.)** Sinus node dysfunction is predominantly observed in the seventh and eighth decades of life, suggesting that age-related degeneration plays a causative role.[2]

One variant of sinus node dysfunction is tachy-brady syndrome, in which episodes of sinus bradycardia or sinus arrest are interspersed with episodes of supraventricular tachycardia. **(See Figure 3.)** During the periods of supraventricular tachycardia, atrial fibrillation is commonly the underlying rhythm, although other etiologies of supraventricular tachycardia can be observed. There appears to be a strong correlation between atrial fibrillation and sinus node dysfunction, with the annual incidence of chronic atrial fibrillation as high as 6% in patients with sinus node dysfunction.[3,4] It is the oscillation between periods dominated by supraventricular tachycardia and periods dominated by sinus node dysfunction that result in the characteristic rhythm.

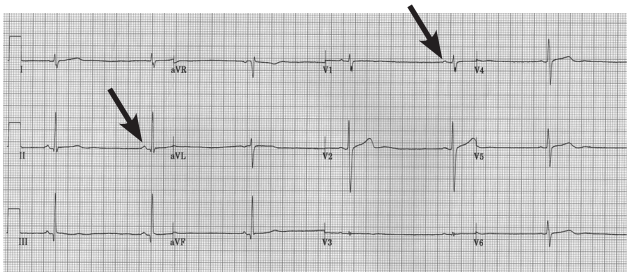
A final dysrhythmia that can be classified as sinus node dysfunction includes a more ambiguous process referred to as *chronotropic incompetence*. This

Table 1. Categories Of Bradydysrhythmias

Bradydysrhythmia Category	Bradydysrhythmia Type
Sinus node dysfunction	• Sinus bradycardia • Sinus arrest • Tachy-brady syndrome • Chronotropic incompetence
AV blocks	• First-degree AV block • Second-degree AV block (Mobitz type I or Wenckebach) • Second-degree AV block (Mobitz type II) • Third-degree AV block (complete heart block)

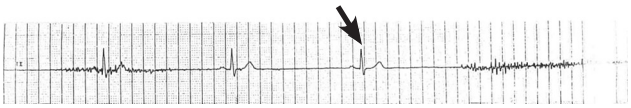
Abbreviation: AV, atrioventricular.

Figure 1. Sinus Bradycardia Electrocardiogram Tracing



Arrows point out regular P waves followed by normal PR intervals.
Source: http://cdn.lifeinthefastlane.com/wp-content/uploads/2011/12/sinus-brady-anorexia-nervosa.jpg.
Used with permission from: www.lifeinthefastlane.com

Figure 2. Sinus Arrest Electrocardiogram Tracing



Arrow points out regular complexes, followed by a long pause without identifiable P waves.
Source: http://cdn.lifeinthefastlane.com/wp-content/uploads/2012/01/sinus-arrest.jpg.
Used with permission from: www.lifeinthefastlane.com.

Figure 3. Tachy-Brady Syndrome Electrocardiogram Tracing

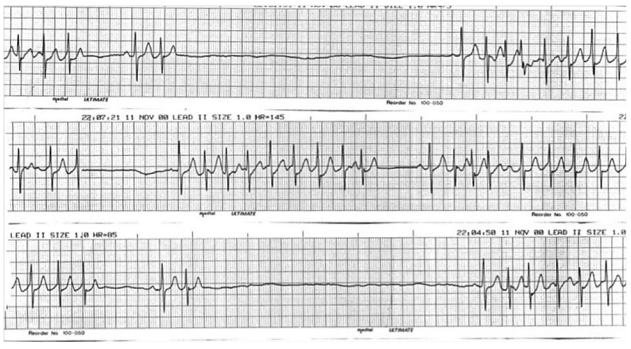


Image shows periods of sinus arrest followed by episodes of tachycardia with irregular RR intervals.
Source: http://lifeinthefastlane.com/wp-content/uploads/2010/09/ecg-exigency-003-a.jpg.
Used with permission from: www.lifeinthefastlane.com

dysrhythmia is best explained as the inability of the sinus node to modulate the heart rate in response to changes in metabolic demands (such as exercise). While it rarely causes symptoms in resting patients, symptoms of exercise intolerance or limitations in physical abilities may be described. Certain medications that have negative chronotropic actions (including beta blockers) may be the underlying cause of this dysfunction.

Atrioventricular Blocks

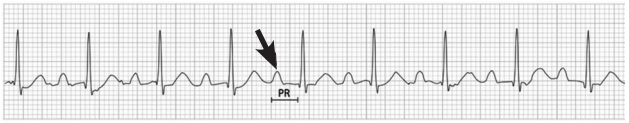
Atrioventricular blocks represent a second broad category of disease that may result in bradydysrhythmia. These occur when conduction from the atria to the atrioventricular node and into the bundle of His is disrupted. Anatomically, these blocks may occur above, within, or below the bundle of His and are further distinguished based on characteristic ECG patterns.

Although it rarely causes patients to seek medical care, first-degree atrioventricular block bears mentioning as a means of comparison to the more severe atrioventricular blocks. First-degree atrioventricular block is defined by a prolongation of the PR interval beyond the normal upper limit of 0.2 seconds. **(See Figure 4.)** Its overall prevalence has been estimated to be between 0.65% and 1.1%.[5] Although it was previously assumed to be a benign disorder, recent research has demonstrated that patients with first-degree atrioventricular block are twice as likely to develop atrial fibrillation and have a moderate increase in all-cause mortality.[6]

Second-degree atrioventricular blocks are distinguished by the intermittent failure of atrial impulses to be conducted into the ventricles. Originally described as early as 1924, they can be further subdivided into type I and type II based on whether or not variance in PR interval is noted.[7] Type I second-degree block (also referred to as Mobitz type I or Wenckebach periodicity) demonstrates progressive elongation of the PR interval before a nonconducted beat occurs. **(See Figure 5.)** In comparison, type II second-degree block (or Mobitz type II) has fixed PR segments, with nonconducted beats usually occurring at regular intervals. **(See Figure 6.)** Recognition of type II second-degree heart block can have prognostic value, as this rhythm is far more likely to degrade to third-degree or complete heart block.[8]

Third-degree atrioventricular block (complete heart block) represents the most severe type of atrioventricular block. This rhythm demonstrates no coordination between atrial activity and ventricular activity, with atrial and ventricular depolarizations occurring at regular (but unrelated) rates. **(See Figure 7.)**

Figure 4. First-Degree Atrioventricular Block Electrocardiogram Tracing



Arrow points to prolonged PR interval approaching 0.28 seconds.
Source: http://cdn.lifeinthefastlane.com/wp-content/uploads/2011/02/1st-degree-heart-block-on-call.jpg.
Used with permission from: www.lifeinthefastlane.com.

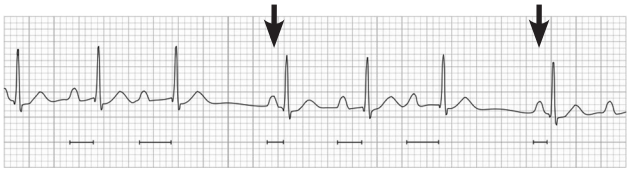
Etiology And Differential Diagnosis

Emergency clinicians should consider a broad differential in the evaluation of patients with bradydysrhythmias, as a wide range of conditions can result in these cardiac abnormalities. These conditions are organized into categories based on similarity of the disease processes. **(See Table 2.)**

Ischemia- And Infarction-Related Causes

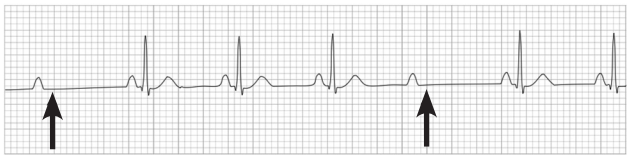
Emergency clinicians are well acquainted with the identification and treatment of acute coronary syndromes, as they represent a common presenting complaint to the ED. Bradydysrhythmias (including sinus bradycardia and complete atrioventricular blocks) are relatively common complications of

Figure 5. Second-Degree Atrioventricular Block, Mobitz Type I, Wenckebach Electrocardiogram Tracing



Note elongation of the PR interval prior to a nonconducted beat.
Source: http://cdn.lifeinthefastlane.com/wp-content/uploads/2011/04/wenckebach.jpg.
Used with permission from: www.lifeinthefastlane.com.

Figure 6. Second-Degree Atrioventricular Block, Mobitz Type II Electrocardiogram Tracing



Note constant length of the PR interval with intermittent, nonconducted beats
Source: http://cdn.lifeinthefastlane.com/wp-content/uploads/2011/04/mobitz_II_rhythm_strip.jpg.
Used with permission from: www.lifeinthefastlane.org.

myocardial infarctions, especially in patients with inferior ischemia patterns. These conditions are more correlated with right coronary artery occlusions than other coronary artery lesions.[9,10] In a retrospective study of 216 consecutive patients presenting with inferior ST-elevation myocardial infarction due to right coronary artery occlusion, bradydysrhythmias and hypotension were present in 31% and 33% of patients, respectively. It was also noted that lesions of the right coronary artery proximal to the right ventricle and atrial branches appear to have statistically higher incidences of hypotension, suggesting that right ventricle dysfunction is central to this process.[11] Emergency clinicians should consider acute ischemia and infarction in patients presenting with these bradydysrhythmias.

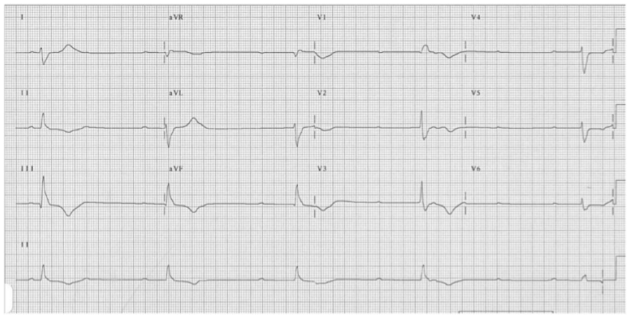
Neurocardiogenic (Reflex-Mediated) Causes

Reflex-mediated etiologies represent another category of bradydysrhythmias that must be considered. One of the more common syndromes that can cause acute reductions in heart rate is that of vasovagal reflex or neurocardiogenic syncope. Estimated to account for 10% to 40% of syncopal episodes, vasovagal (or neurocardiogenic) syncope can occur in response to a myriad of situations, including pain, anxiety, strong emotion, and defecation.[12] It is postulated that, in response to these scenarios, the dysregulated autonomic nervous system induces a sudden excess in parasympathetic tone (or a sudden reduction in sympathetic tone) in what manifests as bradycardia, hypotension, and potential syncope.[13,14]

Hypersensitive carotid sinus syndrome can also result in bradycardia (as well as other bradydysrhythmias, including sinus arrest or atrioventricular blockade) that occurs in response to direct carotid sinus stimulation. This syndrome, mediated by excessive parasympathetic activity, can be further subdivided into cardioinhibitory, vasodepressor, or mixed categories. Untreated, up to 25% of these patients may exhibit recurrent syncope.[15,16] This syndrome may be differentiated from the vasovagal reflex described earlier by the history of direct compression or massage of the carotid sinus and the ability to reproduce symptoms with similar maneuvers. Repeating these maneuvers should be performed cautiously in some patient populations (especially those with atherosclerotic disease), as there is a theoretical risk of dislodging plaque that can occlude downstream vessels.

An interesting phenomenon of relative or paradoxical bradycardia has been observed in trauma patients, and it offers greater insight into the potential mechanisms of reflex-mediated bradycardia. The initial work identified relative bradycardia in roughly 1% to 2% of all trauma patients and in 29% to 44% of hypotensive trauma patients, with a slight improvement in mortality compared to patients exhibiting tachycardia.[17] Subsequent subgroup analyses demonstrated survival benefit in patients with a heart rate between 60 and 90 beats/min but also markedly higher mortality in patients with a heart rate of < 60 beats/min.[18] A variety of mechanisms have been suggested to explain this effect. In case reports and series, it has been observed that patients with hemoperitoneum have exhibited relative bradycardia even in the setting of profound hypotension, leading to the conclusion that irritation of the vagus nerve or the pelvic parasympathetic nerves may result in increased parasympathetic tone.[19,20] This parasympathetic stimulation from intra-abdom-

Figure 7. Third-Degree Atrioventricular Block, Complete Heart Block Electrocardiogram Tracing



Note regular PP and RR intervals with no coordination between atrial and ventricular activity.

Source: http://lifeinthefastlane.com/ecg-library/basics/complete-heart-block/.

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Table 2. Differential Diagnosis For Bradydysrhythmias

Category	Disease Process
Ischemia and infarction	• Inferior myocardial infarction, especially involving the right coronary artery
Neurocardiogenic or reflex-mediated	• Vasovagal reflex • Hypersensitive carotid sinus syndrome • Intra-abdominal hemorrhage • Increased intracranial pressure
Metabolic, endocrine, and environmental	• Hypothyroidism • Hyperkalemia • Hypothermia
Infectious and postinfectious	• Chagas disease *(Trypanosoma cruzi)* • Lyme disease *(Borrelia* species*)* • Viral agents (parvovirus B19, coxsackievirus B, etc) • Syphilis *(Treponema pallidum)*
Toxicologic	• Therapeutic doses of prescribed drugs, overdoses of drugs, or poisoning

inal injuries may result in the cardioinhibitory effects evidenced by absolute or relative bradycardia.

Reflex-mediated bradycardia may also be seen in cases of increased intracranial pressure. Described in 1901 as the Cushing reflex, increased intracranial pressure, increased mean arterial pressure, and bradycardia may be exhibited in cases of intracranial hemorrhage, stroke, intracranial masses, or any other course by which increased intracranial pressure is observed.[21] In a prospective case series of 31 patients presenting with nontraumatic intracerebral hemorrhage, sinus bradycardia was demonstrated in 16% of patients.[22] It is postulated that increased intracranial pressure results in initial sympathetic activation (which causes increased cardiac contractility and vascular tone), while subsequent baroreceptor activation in the carotid sinus results in the observed bradycardia.[23] Emergency clinicians should include intracranial etiologies in their differentials for reflex-mediated bradycardia.

Metabolic, Endocrine, And Environmental Causes

Bradydysrhythmias are also observed in a collection of disorders and conditions that include endocrine diseases such as hypothyroidism, electrolyte abnormalities, and environmental situations such as hypothermia. Hypothyroidism is a relatively uncommon cause of bradydysrhythmia, but when it is associated with bradycardia, the most common arrhythmia is sinus bradycardia with first-degree atrioventricular block.[24,25] Higher-degree atrioventricular blocks are far less common, and they may respond to thyroid supplementation.[26]

Abnormalities in the extracellular levels of potassium have been demonstrated to result in bradydysrhythmias. Patients with end-stage renal disease or acute kidney injury frequently present with elevated potassium levels, and typical ECG findings of peaked T waves, widening of the QRS complex, and eventual sine wave morphology have been documented.[27] However, sinus bradycardia, sinus arrest, and intraventricular blocks can be seen in patients with significant hyperkalemia (> 8.0 mmol/L), particularly in the setting of renal failure.[28] These bradydysrhythmias are often resistant to standard therapy for bradycardia, and atropine and emergent hemodialysis may be required.[29]

Bradycardia can also be seen with hypothermia (as defined by a drop in the patient's temperature to < 35°C, typically occurring as a result of environmental exposure). Sinus bradycardia is most commonly associated with hypothermia, but atrial and ventricular fibrillation can also occur.[30] The classic ECG finding of J-point elevation (or Osborn waves) may or may not be present. **(See Figure 8.)** Many of these cardiac dysrhythmias will improve once normothermia has been achieved.

Infectious And Postinfectious Causes

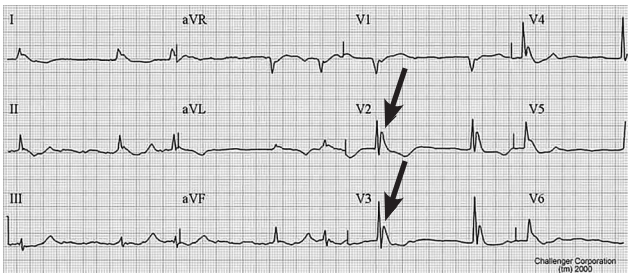
A range of infectious agents including bacteria, viruses, and parasites can lead to bradydysrhythmias. One of the most common infectious agents worldwide is *Trypanosoma cruzi*, which causes Chagas disease. Estimated to affect 10 million people globally, this parasite enters the skin with the help of an insect vector, where it multiplies and primarily targets the cardiac and gastrointestinal systems.[31] Up to 30% of infected individuals will have cardiac dysfunction due to direct infiltration, destruction of cardiac myocytes, and inflammatory damage. Dilated cardiomyopathy and subsequent heart failure as well as progressive atrioventricular blocks and sinus arrest are commonly observed.[32,33]

Lyme disease, caused by bacteria from the genus *Borrelia*, can also lead to bradydysrhythmias. Lyme disease has a reported incidence in the United States of roughly 13 cases per 100,000 persons. Retrospective reviews of Lyme disease have suggested that, in up to 8% of patients, the infection will progress to cardiac involvement.[34,35] Atrioventricular block (including complete atrioventricular block) is the most common form of cardiac impairment, and it is observed in 90% of patients with cardiac effects.[36]

Viral infections have been implicated in up to 50% of all cases of myocarditis. Coxsackievirus B and parvovirus B19 represent the 2 most common offenders.[37,38] During the disease process, direct invasion and destruction of cardiac myocytes lead to progressive heart failure and conduction abnormalities, including atrioventricular blocks.[39]

Syphilis has demonstrated a resurgence in the United States recently, with an annual incidence of 2.7 cases per 100,000 individuals in 2004 and rising.[40] Caused by *Treponema pallidum*, progression of this disease to tertiary syphilis can involve the cardiovascular system. The most common manifestations include aortitis, aortic aneurysms, and valvular

Figure 8. Osborn Waves Electrocardiogram Tracing



Note bradycardia associated with J-point elevation in multiple leads (arrows), as commonly observed in hypothermia.

Source: http://upload.wikimedia.org/wikipedia/commons/2/22/HypothermiaECG.jpg. Reproduced under Creative Commons License 3.0.

abnormalities, although cardiac conduction abnormalities can also be observed.[41,42]

Prehospital Care

Patients with symptomatic bradydysrhythmias may be transported to the ED via emergency medical services (EMS). The initial focus of EMS efforts is the appropriate and timely recognition of the dysrhythmia. Once identified, an evaluation is performed, looking for evidence of hemodynamic instability, including hypotension and altered mentation. Patients classified as unstable may be treated in the field and en route to the ED by protocols and procedures that match closely with the Advanced Cardiac Life Support (ACLS) algorithm for adult bradycardia.[43]

Because many cases of bradydysrhythmia require specific, definitive therapy, additional consideration should be given to choosing the best destination for each patient. For example, if acute ischemia and infarction are expected, transportation to facilities with interventional catheterization services would be optimal. Likewise, patients suffering from potential intracranial or intra-abdominal causes of their bradycardia should be transported to hospitals where appropriate surgical teams are available. Not all EMS agencies will have these options, and many patients will lack clear etiologies for their symptoms; however, when it is feasible, transport to the optimal receiving facility can better match resources to the patient's needs and shorten the time to definitive therapy.

Emergency Department Evaluation

The Basics

A central focus of the ED evaluation is the ability to promptly identify the unstable patient and administer critical therapy, as indicated. A quick assessment of the patency of the airway, the adequacy of ventilations, and the circulatory status can help determine if patients are stable. By rapidly assessing the pulse rate, providers may be able to identify marked bradycardia as well as irregularity of the beats that can help indicate the presence of a dysrhythmia. Initial vital signs can also help identify hemodynamic instability, and frequent reassessment will aid the provider in recognizing response to therapy.

Focused History

A thorough history may provide clues to the underlying disease process that has resulted in the bradydysrhythmia. **(See Table 3.)** For patients who arrive via EMS, pertinent historical elements may be provided by the first responders, especially in the case of disease that has resulted in only transient symptoms. Family or friends may also have additional insight into the patient's condition and may identify subtle changes unrecognized by the patient. Finally, in an era of electronic patient health information, a comprehensive review of previous medical records, if available, can be very helpful in identifying preexisting disease or progression over time.

Physical Examination

The physical examination can help confirm the suspicions that the emergency clinician may have regarding the hemodynamic stability of the patient. For this reason, ongoing assessment of perfusion status is essential in all patients with bradydysrhythmias. Perfusion can be confirmed with the identification of strong peripheral pulses, brisk capillary refill, and warm extremities. The examination may also aid in identifying the underlying pathology that has led to the dysrhythmia, as many of these diseases result in concomitant heart failure as well as bradydysrhythmia. Evidence of heart failure may be suggested by lower-extremity edema, elevated jugular venous distention, or rales in the lower lung fields. The presence of a dialysis catheter or a fistula can indicate a patient with end-stage renal disease, which suggests hyperkalemia. A tender abdomen (especially if guarding is present) can suggest an intra-abdominal insult with resultant reflex-mediated bradycardia. Also, the presence of an implanted device such as an automatic implantable cardioverter defibrillator (AICD)/pacemaker may suggest previous occurrences of similar pathology.

Diagnostic Studies

The Electrocardiogram

The most meaningful study that can be obtained in the ED is the 12-lead ECG. Initial rhythm analysis

Table 3. Assessing The History Of The Patient With Bradydysrhythmia

History	Possible Underlying Pathology
Preceding angina symptoms	Myocardial ischemia/infarction
Fevers, travel to endemic areas, tick bite	Infectious agent
Cold intolerance, weight gain, increased fatigue	Hypothyroidism
Headache, mental status change, recent head trauma, falls	Intracranial causes, including intracranial hemorrhage
Abdominal pain or distention	Intra-abdominal hemorrhage
Recent additions or changes to medications	Drug toxicity
History of end-stage renal disease, receiving dialysis	Hyperkalemia
Cancer history, receiving treatment	Acute or long-term toxicity from chemotherapeutic agents
Severe pain, anxiety, strong emotion preceding the event	Vasovagal reflex, neurocardiogenic

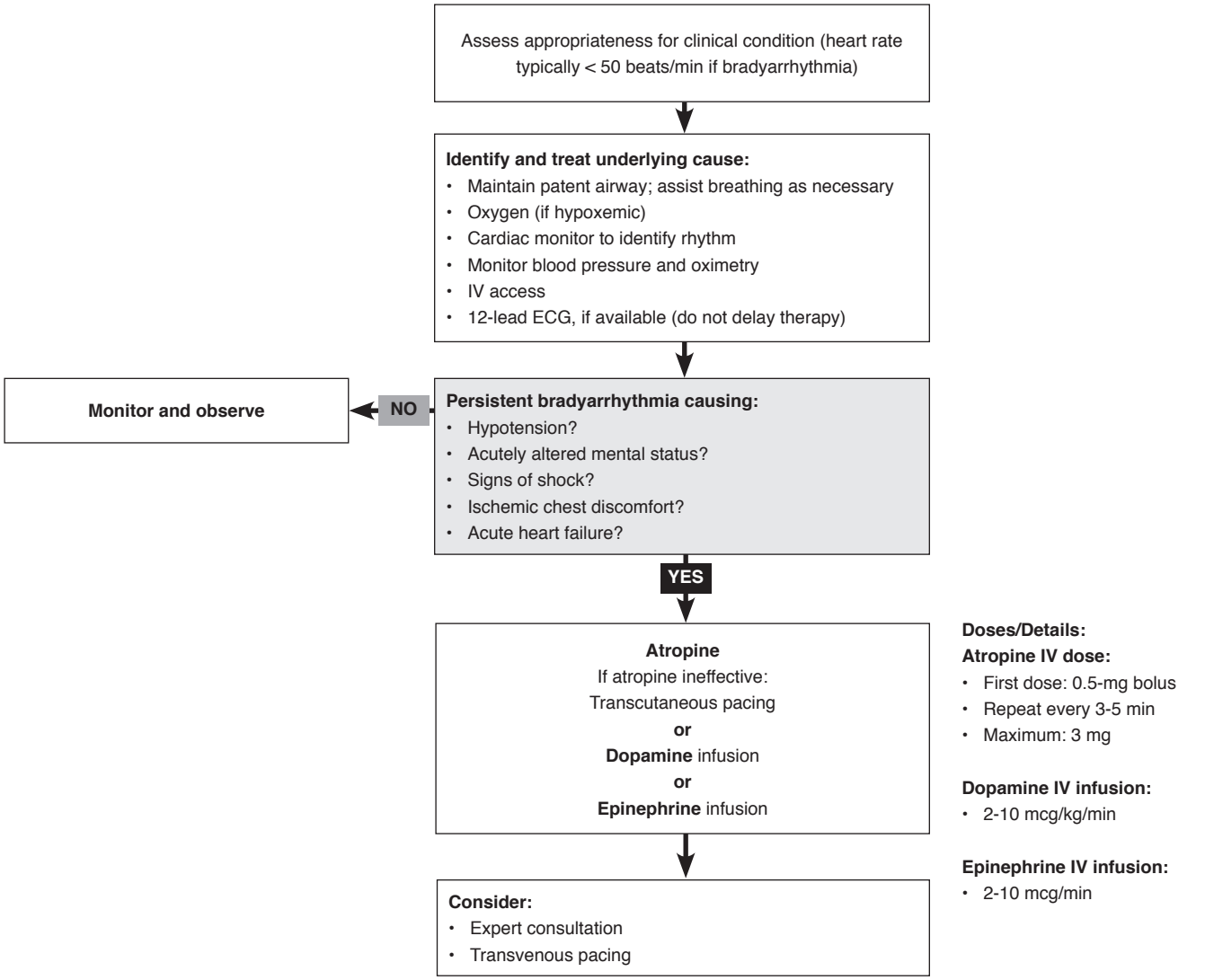
can explain the symptoms the patient is experiencing, especially if an abnormality is captured on the ECG. The importance of repeat analysis cannot be overstated, as some bradydysrhythmias will appear intermittently and the evolution over time can be critical for acute diagnosis. Because of the possibility of intermittent or infrequent dysrhythmias, many emergency clinicians will choose to place patients on continuous cardiac monitoring while in the ED. This can also aid in the diagnosis, especially if symptoms correlate with changes noted on the cardiac tracing.

Laboratory Studies

Serum studies may confirm underlying diagnoses and help guide therapy. Abnormalities in electrolyte levels, especially potassium, may be responsible for the patient's condition or may exacerbate other disease processes and should be investigated. A drug level should be obtained on all patients who are currently taking digoxin because of its narrow therapeutic window. Thyroid function testing (including thyroid stimulating hormone and free T3 or T4) can be obtained if hypothyroidism is suspected.

Infectious markers (such as a rapid plasma reagin [RPR] for syphilis or Lyme titers) are indicated if an infectious etiology is possible or when a

Clinical Pathway For Adult Bradycardia (With Pulse)



Abbreviations: ECG, electrocardiogram; IV, intravenous.

Reprinted from: Robert W. Neumar, Charles W. Otto, Mark S. Link, et al. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 122(18 Suppl 3), pages 729-767. Used with permission of Wolters Kluwer Health.

search for more-likely etiologies has not yielded a diagnosis, but they are unlikely to be helpful in the acute setting. Cardiac biomarkers, including troponin, creatine kinase (CK), CK-MB, myoglobin, and B-type natriuretic peptide (BNP) can also be helpful in diagnosing infarction and/or heart failure.

Imaging Modalities

There is limited utility to imaging modalities in the ED for the evaluation of the patient with bradydysrhythmias. If there is suspicion of an intracranial hemorrhage or mass resulting in increased intracranial pressure, computed tomography (CT) of the head may be warranted. The emergency clinician may also consider abdominal imaging (such as CT or ultrasound) for patients with abdominal findings on examination who exhibit absolute or relative bradycardia. A chest x-ray may also lend support for an underlying diagnosis of heart failure if there is evidence of an enlarged cardiac silhouette or pulmonary edema; however, it has limited utility otherwise. Heart failure may be further confirmed with an echocardiogram. While the echocardiogram is unlikely to provide additional information that will alter care in the ED, it may be useful for the patient's continued course of care.

Treatment

The approach to treating a patient with a bradydysrhythmia begins with identifying whether the patient is stable or unstable. Evidence of instability in the patient's condition includes hypotension, altered mentation, acute heart failure, or poor distal perfusion. Unstable patients require emergent intervention to improve cardiac output and prevent end-organ damage. After acute stabilization efforts for the unstable patient have been made, efforts to diagnose any underlying etiology of the bradydysrhythmias should commence. While not all causes will be amenable to treatment in the ED, the identification and reversal of the underlying pathology is the central goal of these patients' continued care.

The Unstable Patient

Therapy for unstable patients begins with airway control, oxygen administration, and ventilatory assistance, if necessary. Atropine administration remains the first-line medication for unstable and symptomatic patients with bradydysrhythmias, and it has been shown to significantly improve heart rate and conduction blocks (Class IIa, Level of Evidence [LOE] B).[44,45] **(See Table 4.)** The recommended dose is 0.5 mg intravenously every 3 to 5 minutes, with a maximum dose of 3 mg. Note that, in patients who have undergone cardiac transplantation, atropine is unlikely to be effective, as the expected vagal innervation to the heart no longer exists.[46] It is also important to recognize that second-degree type II atrioventricular blocks or third-degree blocks also may not respond to atropine.

If atropine administration has been ineffective, a beta-adrenergic agent (such as dopamine, epinephrine, or isoproterenol) should be considered. Dopamine infusion should be initiated at 2 to 10 mcg/kg/min in patients who have failed to improve with atropine (Class IIb, LOE B). In a randomized controlled trial in the prehospital setting, 82 unstable patients with bradycardia were randomized to transcutaneous pacing versus dopamine infusion after fluid resuscitation and atropine therapy failed. Dopamine demonstrated equivalent survival outcomes and adverse events to transcutaneous pacing without the associated chest discomfort associated with pacing.[47] While known to have beta-adrenergic effects, epinephrine and isoproterenol are less well studied in the setting of unstable bradycardia, but they can be considered in the setting of symptomatic bradycardia when atropine has failed (Class IIb, LOE B).

Transcutaneous and transvenous pacing, described as early as the 1950s, remain important options for the treatment of unstable bradycardia (Class IIa, LOE B).[48,49] In a few feasibility studies, pacing has been demonstrated to be equal in effectiveness to beta-adrenergic agents in augmenting the heart rate.[47,50] Limitations for transcutaneous and transvenous pacing include patient comfort and the need for central venous access, respectively; however, pacing should be initiated in unstable patients who have not responded to atropine or beta-adrenergic agents (Class IIa, LOE B).

Technique For Transcutaneous Pacing

There are 2 placement configurations recommended for optimal capture in transcutaneous pacing. One option is to center a pad over the apex of the heart and place the other pad on the right upper chest. Alternatively, a pad may be placed over the V3 lead position while the other is placed between the left scapula and the thoracic spine. Transcutaneous pacing can be quite uncomfortable for patients who are awake, as it requires the discharge of electrical

Table 4. American Heart Association Classification Of Recommendations[43]

Size of Treatment Effect

Class I: Benefits *significantly* greater than risks
Class IIa: Benefits *somewhat* greater than risks
Class IIb: Benefits *may* be greater or equal to risk
Class III: Risks *greater* than benefits

Strength of Evidence

Level A: Multiple randomized trials
Level B: Single randomized trial or nonrandomized studies
Level C: Case studies or expert opinion

impulses through the skin and chest wall muscles. Therefore, procedural sedation should be considered in order to reduce this discomfort. Some medications used for sedation have the potential to lower blood pressure and can potentially worsen the patient's condition. For this reason, caution is advised when considering these medications.

Once effective sedation has been achieved (or in patients whose clinical condition is too tenuous to allow for sedation), cardiac pacing should be initiated. An initial pacing rate should be set at least as high as the intrinsic heart rate of the patient (although a standard rate of 80 beats/min can be selected), with the current set to minimal output. Initially, pacer spikes may be visualized without resultant cardiac depolarization. The current can be increased 5 to 10 mA at a time until a clear QRS complex and T wave is demonstrated following each pacer spike. Check the patient's pulse at this point to confirm that the electrical "capture" has resulted in a mechanical response. This level is defined as the pacing threshold, and it will be found between 40 and 80 mA for most healthy patients. Final current output should be set to 5 to 10 mA above the threshold level to ensure continued capture. For a video demonstration of the technique, see **Figure 9**.

Technique For Transvenous Pacing

Several anatomic sites may be used to access the central venous system for transvenous pacing, but the right internal jugular vein is most commonly used in the ED. This also preserves the left subclavian vein for the placement of a permanent pacer, should one be needed. A single lumen sheath catheter may be placed and secured using the standard technique. The pacing catheter contains a small balloon at the distal tip that must be checked for leaks prior to insertion. This can be accomplished by filling the balloon with air while the balloon rests in a container of saline. The presence of bubbles in the saline indicates a leak in the balloon and the need for a new catheter.

Once the balloon has been confirmed to be intact, connect the positive and negative electrodes to the external pacemaker unit and advance it through the introducer sheath to a depth that ensures that the balloon has emerged beyond the distal segment of the sheath. At this point, the balloon may be inflated with air. Recommended volumes may differ, based on the specific brand of product used, and emergency clinicians are encouraged to refer to product packaging for the appropriate volumes. The pacer may be turned on at this point, with an initial setting at least as high as the intrinsic heart rate of the patient (although a standard rate of 80 beats/min is often selected) and the maximal current output (usually 20 mA). The sensitivity dial should be adjusted to the "asynchronous" setting.

While the pacer wire is slowly advanced, the ECG monitor should be watched closely for evidence of capture. Pacer spikes (very narrow vertical signals) will begin to be apparent, followed by a widened QRS with a similar appearance to a left bundle branch block. This indicates contact between the electrode and the wall of the right ventricle. At this point, the balloon can be deflated and the catheter secured in place. To determine a threshold level, electrical current settings may be reduced until failure to capture is demonstrated. Final settings should be roughly twice the threshold value to ensure continued capture.

Ultrasound has now been advocated as an additional dynamic method for determining lead placement and capture. Further verification of placement is accomplished by chest radiograph demonstrating the catheter tip over the inferior border of the cardiac shadow. For a video demonstration of the technique, see **Figure 10**.

Goals Of Treatment For Unstable Patients

Treatment directed toward unstable patients aims to restore appropriate cardiac output as quickly as possible to ensure continued end-organ perfusion. In the event that emergent therapy is not required, the underlying pathology should be investigated. Treatment for these patients may begin in the ED, but it will often continue once the patient is admitted and may include support from consulting services.

Figure 9. Link For Demonstration Video Of Transcutaneous Pacing

To access the video, scan the QR code with a smartphone or visit:
http://www.youtube.com/watch?v=qkSGaJkNqvg

Figure 10. Link For Demonstration Video Of Transvenous Pacing

To access the video, scan the QR code with a smartphone or visit:
http://www.youtube.com/watch?v=5BiQQYjw6no

The Stable Patient

Many patients who present to the ED with a bradydysrhythmia or who have experienced a recent transient event of bradycardia are stable. Evidence of stability in the patient's condition is continued evidence of end-organ perfusion, including appropriate mental status. Most of these patients do not require the emergent interventions described earlier. For the stable patient, the role of the emergency clinician moves away from the provision of critical care and towards that of making a diagnosis.

Efforts should be made to identify the underlying etiology of the bradydysrhythmia. Some etiologies are readily identifiable and treatable, especially in toxicological cases or for electrolyte abnormalities. For these patients, therapy to address the central process begins in the ED; however, some diseases (including infectious myocarditis or immune-mediated mechanisms) may not be readily identifiable, may require additional testing to uncover, or may have treatments that cannot easily be initiated in the ED. It is reasonable to expect that these patients will be admitted to the hospital for continued care.

It is imperative for the emergency clinician to keep in mind that not all causes of bradycardia are cardiac in origin. As described previously, intra-abdominal and intracranial insults may result in absolute or relative bradycardia. If these processes have been identified, early consultation with other medical services and possible operative management can remove the causative process and improve cardiac status.

There are a number of patients who experience bradydysrhythmias for which no treatment is necessary. Most of these patients will have transient symptoms, often from reflex-mediated causes, including vasovagal syncope or hypersensitive carotid sinus syndrome. These patients may be asymptomatic once they arrive to the ED and—especially for first-time occurrences—no further therapy is warranted. Once the underlying etiology has been addressed, most of these patients can be discharged from the ED with reassurance.

Drug Toxicity

Many medications, in therapeutic doses or in overdose, are common culprits of bradydysrhythmias, and emergency clinicians should be prepared to identify and address these conditions as quickly as possible. Beta blockers and calcium channel blockers are the most likely medications to present as bradydysrhythmias, and they represent roughly 1% of all calls to poison centers.[51] Many of these ingestions are unintentional overdoses of the patient's prescribed medications, and a thorough review of a patient's medication list may reveal the offending drug.[51,52] Digoxin is another common medication that can result in bradydysrhythmia, with up to 35% of patients exhibiting some degree of atrioventricular block.[54] General toxicology principles of limiting exposure, decontamination, and supportive care are critical to appropriate treatment of these patients; however, some compounds have particular antidotes and therapies that can improve or reverse the bradydysrhythmias exhibited. **(See Table 5.)**

Disposition

The majority of the patients who present with symptomatic bradydysrhythmias are likely to be admitted to the hospital for continued monitoring and further treatment. Patients who were initially identified as unstable and required emergent care (including the use of atropine, a beta-adrenergic agent, or cardiac pacing) should be admitted to an intensive care unit. In addition, patients with ECG analysis confirming complete heart block or Mobitz type II second-degree heart block should also be admitted to an intensive care setting, as there is a significant concern for continued deterioration. For patients not admitted to intensive care settings, it is recommended to admit them to units capable of continuous cardiac telemetry. As many bradydysrhythmias are intermittent by nature, continuous telemetry will capture the offending rhythm, should it recur.

In smaller or rural hospitals, there may not be readily available access to cardiology consultation. In those circumstances, the emergency clinician should strongly consider transferring the patient to a hospital where these services are available.

Table 5. Specific Antidotes And Therapies For Toxicological Causes Of Bradydysrhythmias

Drug or Toxin	Antidote or Therapy
Beta blockers	Glucagon: 5 mg IV; can be repeated every 10 min, up to 3 doses
Calcium channel blockers	• Calcium gluconate 10%, 30 to 60 cc IV • Insulin (regular): 1 unit/kg bolus + 0.5 units/kg/h infusion; supplement glucose as needed
Digitalis (digoxin)	Digoxin immune Fab (Digibind® or DigiFab®): empirically, 10-20 vials (if serum digoxin level is available, product insert can be used for more exact dosing guide)
Opioids	Naloxone: 0.4 mg IV, then 2 mg IV if no response
Organophosphates	Atropine: 2 mg IV; double every 3-5 min until pulmonary secretions are manageable
	Pralidoxime (2-PAM): 2 g IV over 10-15 min, repeated every 6 h

Abbreviations: Fab, fragment antigen-binding; IV, intravenously.

Risk Management Pitfalls For Bradydysrhythmias In The Emergency Department

1. **"The initial ECG looks fine, and I'm not that impressed with the history. I'm sure the patient is fine."**
   Many bradydysrhythmias are transient and intermittent in nature. Upon initial evaluation, it is not uncommon to have a normal ECG and an asymptomatic patient. Don't make any final decisions based on a single ECG. Two ECGs are better than one, and continuous monitoring in the ED is even better.

2. **"I know she passed out at home, but she looks fine now, and she really wants to go home."**
   It can be tempting to discharge an asymptomatic patient home, especially if she is eager to leave. Remember that the absence of symptoms now does not mean there will be no recurrence. Make sure that there is a clearly identifiable (and reversible or avoidable) cause of symptoms if you plan on discharging a patient home.

3. **"I didn't check the medical record because he said this has never happened before."**
   With the pervasiveness of electronic health records, it is becoming difficult to justify failure to review a patient's records of previous encounters. Patients may not be able to accurately answer whether they have had dysrhythmias in the past. A quick review of the record may uncover additional history that can make a big difference.

4. **"I was so focused on the bradycardia that I totally missed the ST changes in the inferior leads."**
   Inferior ischemia and myocardial infarction are frequently associated with bradydysrhythmias. Don't forget to scan through the inferior leads of the ECG to make sure the patient doesn't need emergent revascularization.

5. **"It looks like second-degree type II block on the ECG. We can probably admit him to the floor."**
   Even if the patient appears asymptomatic now and a majority of the beats are being conducted, remember that, in certain settings, second-degree type II blocks can rapidly degrade to complete heart block. Strongly consider admitting the patient to an intensive care unit for closer monitoring.

6. **"I didn't even think to ask about travel history or tick bites."**
   Infectious causes of bradydysrhythmias, including Chagas or Lyme disease, may not be common if your ED is not within an endemic area; however, for patients who have lived in or traveled to endemic areas, asking about this may identify the underlying cause.

7. **"We were so busy focusing on the therapy that I overlooked the fistula in the patient's arm."**
   Clues to the underlying cause of the conduction abnormality may be evident on examination. Make sure you look for evidence of dialysis catheters or fistulas if you are considering the likelihood of hyperkalemia.

8. **"He said he had been on the same digoxin dose for the past few years, so I didn't check."**
   In the setting of digoxin therapy (which is known to cause bradydysrhythmias), don't forget to check drug levels. If recognized as the offending toxin, antibody antidote therapy may be the only treatment for the dysrhythmia.

9. **"A heart rate of 45 beats/min in a 25-year-old? I can't find a reason for her to have any cardiac disease."**
   Not all causes of bradydysrhythmias are cardiac in nature. Don't overlook intra-abdominal pathology as a potential cause. Broaden your differential to include these reflex-mediated syndromes.

10. **"I was so pleased I could explain the patient's syncope with the sinus bradycardia that I completely missed the other injuries."**
    Although the evaluation and identification of syncope are important to signal bradydysrhythmia as a potential factor in trauma, do not overlook other injuries the trauma patient may have sustained. Especially in the elderly, be sure to evaluate for extremity fractures, head trauma, and other injuries following syncope and falls.

There may be a small subset of patients who can be discharged home following their ED encounter for bradydysrhythmia. Patients suitable for discharge are likely patients who are have been diagnosed with a transient reflex-mediated bradycardia that has subsequently resolved; they should be otherwise healthy and without risk factors predisposing them to continued or future dysrhythmias. Patients discharged home should be given strict instructions to return to the ED if symptoms recur as well as recommendations to follow up promptly with a primary care provider.

Summary

Bradydysrhythmias represent a collection of cardiac conduction abnormalities that emergency clinicians are likely to encounter. When evaluating patients with bradydysrhythmias, consider a wide variety of underlying conditions and diseases, including acute myocardial infarction, electrolyte abnormalities, drug toxicities, and infectious causes. These patients have the potential to present with hemodynamic instability and will require emergent intervention, including atropine, beta-agonist therapy, or cardiac pacing. With an organized and systematic approach, emergency clinicians can provide the life-saving therapy that these patients require.

Case Conclusions

When you returned to complete the history on your 80-year-old female patient who had the syncopal event at home, you found that she had no prodrome before the syncopal event, although she did describe recent episodes over the past few weeks where she suddenly became very lightheaded. These episodes apparently resolved spontaneously. She appeared stable upon arrival to the ED. After being placed on cardiac telemetry in the ED, a couple of episodes with sinus arrest were recorded, and some were followed by sinus tachycardia. Based on her brief telemetry monitoring in the ED, you suspected tachy-brady syndrome, and you admitted her to the hospital for continued monitoring, cardiology consultation, and consideration of pacemaker placement.

Your 27-year-old female patient with abdominal pain, hypotension, and bradycardia had a history of lower abdominal pain and had experienced significant worsening of the pain within the last few hours. You inquired as to pregnancy, and she confirmed a recent positive pregnancy test. Her last menstrual period placed her around 8 weeks' gestation. Her vital signs were notable for profound hypotension with paradoxical bradycardia. As intravenous access was obtained, you prepared for fluid resuscitation as well as blood transfusion. A rapid FAST exam confirmed your suspicions of hemoperitoneum. A quick call to the in-house gynecologist requested emergent surgery for this patient with a ruptured ectopic pregnancy.

Meanwhile, your 64 year-old patient with bradycardia was noted to be pale, diaphoretic, and altered. Initial ECG analysis demonstrated third-degree heart block. You quickly secured IV access and gave him IV atropine, but he had minimal-to-no response in the conduction block. You initiated transcutaneous pacing, capture was demonstrated, and you noted marked improvement of his distal perfusion and mental status. Because of the discomfort associated with transcutaneous pacing, you obtained central venous access and transitioned the patient to a transvenous pacer. From his health records, you learned that he had long-standing bundle branch block from dilated cardiomyopathy. With the transvenous pacer, he appeared to be doing much better, and he was admitted to the cardiac critical care unit with a plan to place a permanent pacer/AICD soon.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available.

Time- And Cost-Effective Strategies

1. If you are administering atropine or dopamine for the unstable patient with bradycardia, prepare to pace early. Don't wait for the medications to fail before considering transcutaneous or transvenous pacing. Equipment must be set up appropriately (especially with the transvenous modality) before the therapy can be initiated. In these critical patients, plan ahead and have the supplies ready before you need them.
2. In the patient with absolute or relative bradycardia and suspicion for intraperitoneal hemorrhage, utilize the focused assessment with sonography for trauma (FAST) scan if you are considering intra-abdominal pathology. CT can take significantly longer to obtain—time the patient may not have. If you suspect intra-abdominal hemorrhage, a quick look with the ultrasound may be all that is necessary to decide on a trip to the operating room.

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CME Questions

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1. Which bradydysrhythmia is best described as "intermittent periods of sinus tachycardia interspersed with periods of sinus arrest or sinus bradycardia?"
a. Sinus arrest
b. First-degree AV block
c. Third-degree AV block
d. Tachy-brady syndrome

2. What bradydysrhythmia is best described as "prolongation of the PR interval followed by a nonconducted beat?"
a. First-degree AV block
b. Second-degree AV block, type I
c. Second-degree AV block, type II
d. Third-degree AV block

3. All of the following mechanisms may result in bradycardia EXCEPT:
a. Pressure applied to the carotid sinus
b. Lumbar spinal cord lesions
c. Increased intracranial pressure
d. Intraperitoneal hemorrhage

4. Overactivity of which nerve carrying parasympathetic fibers to the heart is suspected to occur in many reflex-mediated bradydysrhythmias?
a. Hypoglossal
b. Peroneal
c. Phrenic
d. Vagus

5. Which electrolyte abnormality is most commonly associated with bradydysrhythmias?
a. Hypokalemia
b. Hyperkalemia
c. Hypocalcemia
d. Hypermagnesemia

6. **Which abnormality on an ECG can be found in hypothermic patients exhibiting bradycardia?**
   a. Biphasic T waves in V1 and V2
   b. PR depression
   c. U wave
   d. J-point elevation (Osborn wave)

7. **Estimated to affect 10 million people, this is the most common infectious cause of brady-dysrhythmias worldwide:**
   a. *Mycobacterium tuberculosis* (tuberculosis)
   b. *Plasmodium falciparum* (malaria)
   c. *Treponema pallidum* (syphilis)
   d. *Trypanosoma cruzi* (Chagas disease)

8. **What is the first-line therapy for the unstable patient presenting with bradycardia?**
   a. Adenosine
   b. Atropine
   c. Dopamine
   d. Epinephrine

9. **Which of the following medications should be considered if atropine has failed to improve the bradycardia?**
   a. Beta agonists (dopamine or epinephrine)
   b. Insulin
   c. IV calcium
   d. IV magnesium

10. **Below is a list of drugs that can produce brady-dysrhythmias during acute intoxication. Which is correctly paired with its antidote?**
   a. Beta blockers/glucagon
   b. Calcium channel blockers/pralidoxime
   c. Digoxin/calcium
   d. Organophosphates/adenosine

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