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Bradydysrhythmias

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Rapid Access

Approach to the Critical Patient

Connect the patient to a monitor, obtain IV access, and place pacer pads.

Have resuscitation cart, intubation supplies, and transvenous pacer kit at the bedside.

Obtain a 12-lead ECG.

If the patient is unstable, lay the patient flat to increase cerebral perfusion.

- **Atropine 0.5-1 mg IV q3-5min** until resolution or the maximum dose (3 mg) is reached **or**

If atropine is ineffective or for Mobitz II or third-degree heart block, proceed to transcutaneous pacing.

- **Transcutaneous Pacing**

  Adjust energy delivery of the pacing stimulus.

  - Start at 80 mA and reduce to the lowest energy that consistently initiates pacing.

  Adjust pacing rate.

  - Rates of 80-100 per min are appropriate.

  Determine patient response.

  Electrical capture

  - Pacing spike is consistently followed by a widened QRS complex.

  Mechanical capture

  - Patient demonstrates a palpable pulse corresponding to each electrically paced complex.
  - Patient's perfusion improves.

If patient does not respond to atropine or transcutaneous pacing, administer:

- **Epinephrine 20-50 µg IV bolus or** epinephrine 2-10 µg/min IV infusion.

Transcutaneous pacing is a bridge to **transvenous pacing** or resolution of the bradycardia.

If there is no response to the above measures or if hyperkalemia is a consideration, administer **1 g calcium chloride or 3 g calcium gluconate IV**.

Additional medication considerations

- **Dobutamine** 2-20 µg/kg/min IV infusion

  May cause vasodilation and hypotension

- **Dopamine** 2-20 µg/kg/min IV infusion

  May cause vasodilatation at low doses and vasoconstriction at higher doses

- **Isoproterenol** (pure beta agonist) 2-10 µg/min IV, titrate to effect
- **Overdose/medication toxicity (eg, beta blocker, calcium channel blocker)**

  Glucagon 5-10 mg IV bolus over 1 min followed by continuous infusion at 1-5 mg/h

Calcium chloride 1 g (10 mL of 10% solution) IV **or** calcium gluconate 3 g (30 mL of 10% solution) IV bolus at 1 mL/min; may repeat dose if needed. Calcium chloride should be given through a central line.

High-dose insulin 1 unit/kg IV push followed by 0.5-1 unit/kg/h with adequate glucose repletion.

IV lipid emulsion 20% at 1.5 mL/kg IV bolus followed by continuous infusion of 0.25 mL/kg/min over 30-60 min.

All patients should have a **core temperature,** fingerstick glucose, and lab studies including electrolytes, troponin, thyroid studies, coagulation panel, and complete blood count with cultures if an infection is suspected; treatments may include extracorporeal membrane oxygenation, percutaneous intervention, thyroxine, hydrocortisone, or antibiotics.

If indicated, obtain a head computed tomography (CT) scan to rule out increased intracranial pressure (ICP).

EM:RAP Links

Bradycardia- Initial Approach

ECG Course: Too Slow

Key Concepts

PEARLS

Bradycardia is defined as a heart rate of less than 50-60 beats/min (bpm) in adults (non-well-conditioned athletes) or a sinus pause >2-3 s.

Causes of bradycardia can be either intrinsic or extrinsic, all of which ultimately impair the conduction system of the heart, ie, sinoatrial or atrioventricular (AV) node.

The patient with bradycardia should be approached first on the basis of hemodynamic stability.

An unstable bradycardia occurs when there is a reduction in the heart rate that results in a low cardiac output requiring immediate intervention. Evidence of instability includes

- Hypotension with hypoperfusion
- Altered mental status
- Anginal chest pain
- Dyspnea due to pulmonary congestion

PEARLS

In unstable patients, the goal of treatment is to increase the heart rate to achieve an adequate cardiac output to perfuse vital organs.

Treatment of unstable bradydysrhythmias requires temporizing the patient with an external pacemaker, atropine, and/or adrenergic drugs to increase the heart rate **until the underlying cause is identified and treated**.

PITFALLS

- Atropine is a vagolytic and will not work for non-parasympathetic causes of bradycardia or infranodal blocks. It is not useful in most cases.

- Epinephrine works on the entire myocardium and will increase the rate and contractility of the heart. If it is readily available, start with epinephrine instead of atropine.

Stable patients may not require intervention.

PEARLS

- **Treat the patient, not the number (ie, heart rate)**.

PITFALLS

- **Caution is advised for severely bradycardic and normotensive patients as endogenous sympathetic responses result in vasoconstriction. Look for other markers of shock, i.e. altered mental status, ischemic chest pain, and pulmonary congestion**

- **Avoid treating hypertension in patients with severe bradycardia and hypertension. Vasodilation can lead to hemodynamic collapse.**

Bradycardia caused by metabolic/toxic insults (eg, hypoglycemia, hypothermia, hyperkalemia, or hypothyroidism) will respond poorly or not at all to standard therapy (ie, electrical pacing, atropine administration). The underlying disease must be managed first.

PEARLS

- **Consider hyperkalemia early, and if possible, obtain bedside potassium levels.**

Diagnosis

The majority of bradycardic patients will be asymptomatic.

In patients with bradycardia-impaired cardiac output, clinical presentations may vary and can include lightheadedness, near syncope, syncope, chest pain, pulmonary edema, heart failure, altered mental status, and cognitive slowing.

Causes

Bradydysrhythmias result from intrinsic (ie, cardiac electrical system-based) dysfunction (49%) or extrinsic causes (51%).

Intrinsic (sinoatrial and AV node dysfunction)

- Aging is the most common cause
- Ischemic heart disease
- Infiltrative disorders
- Surgery
- Trauma

Extrinsic (including non-electrical system cardiac tissues)

- Acute coronary syndrome (ACS)
- Medications, illicit drugs, and toxins
- Metabolic (particularly electrolyte disorders such as hyperkalemia)
- Implanted pacemaker dysfunction
- Infectious agents and infections
- Endocrinopathies
- Increased ICP

Classification

Bradyarrhythmias are **classified anatomically** by either the location of the dysfunction (usually sinoatrial node dysfunction and/or other areas of the myocardium taking the role of the pacemaker) or the location of the block along the electrical conduction system of the heart.

ECG can help in localizing the anatomical dysfunction or block.

In the ED, investigating the underlying clinical causes of bradyarrhythmias is a more useful approach than anatomical classification, in terms of management and treatment.

ECG

- The ECG is the first critical step in diagnosis.

- When reviewing the ECG, noting the following characteristics will aid in identification of the rhythm:

    Atrial rate (P waves)

    P-R interval length from beat-to-beat

    Ventricular rate (QRS complex)

    Association of P waves with QRS complex

    Width of the QRS complex

    Irregular or regular rhythm

Treatment

Treat the patient, not the number (heart rate).

Be prepared.

- Perform ECG monitoring.
- Obtain IV access.
- Place resuscitation cart and airway equipment at bedside.
- **Place pacer pads on the patient's chest.**

If patient is stable, have pacing pads in place but do not activate.

PEARLS

If patient is unstable, initiate either atropine or transcutaneous pacing.

- For unstable sinus bradycardia or Mobitz I, administer atropine 0.5-1 mg IV q3-5min until resolution or the maximum dose (3 mg) is reached.
- If atropine is ineffective or in cases of Mobitz II or third-degree AV block, initiate transcutaneous pacing.

    PEARLS

    If there is no response to atropine and/or transcutaneous pacing, administer **epinephrine** 20-50 µg IV bolus **or** epinephrine 2-10 µg/min IV infusion.

Transcutaneous pacing

- Place pads on the patient's chest at base and apex locations (or anterior/posterior).
- Activate pacing.

    Adjust energy delivery of the pacing stimulus.

PEARLS

- Start at 80 mA and reduce to the lowest energy that consistently initiates pacing.

Adjust pacing rate.

- Rates of 80-100 per min are appropriate.

Determine patient response.

Electrical capture.

- Pacing spike is consistently followed by a widened QRS complex.

Mechanical capture.

- Patient demonstrates a palpable pulse corresponding to each electrically paced complex.
- Patient's perfusion improves.

- Sedate and/or treat pain **if possible** for the patient's status (perfusion and airway).

Such interventions are not always possible if the patient is in shock or has a compromised airway/breathing status.

Prepare for transvenous pacing, as transcutaneous pacing is only a temporary bridge to transvenous pacing or resolution of bradycardia.

- A transvenous pacer is best placed by a clinician experienced in this procedure, with appropriate fluoroscopic guidance.
- If such resources are not available, consider local capabilities and a potential placement using either ECG or ultrasound guidance. (See chapter: Cardiac Pacing)

Management of specific situations:

- If an **extrinsic cause** is identified, treat the underlying etiology.

Myocardial infarction (MI): Reperfusion therapy.

Electrolytes: Treat the primary derangement.

Hypothermia: Rewarming.

Sepsis: Resuscitation with IV fluids, antibiotics, and source control.

Hypoglycemia: Dextrose and manage etiology.

Intracranial hemorrhage: Management as directed by the type of hemorrhage and sequelae.

- **Intrinsic causes**

Narrow QRS bradycardias usually have a better prognosis and frequently do not require pacing. These bradycardias are frequently vagally induced or occur as a result of a reversible cause (eg, beta blocker overdose).

- Examples:
  - Sinus bradycardia
  - Other sinus node dysfunction
  - First-degree AV block
  - Second-degree AV block, type 1 (Mobitz I, Wenckebach)
  - Third-degree block AV block with narrow QRS complex escape

Wide QRS bradycardias are concerning for infranodal block or disease. These patients will likely need resuscitation and may potentially require permanent pacemaker placement.

- Examples:
  - Second-degree type 2 AV block (**Mobitz II**)
  - **Third-degree AV block** with wide QRS complex
  - Idioventricular rhythm (ventricular escape rhythm)

Disposition

The presence of a chronotropic response (increasing heart rate with movement) is reassuring.

Discharge home can be considered when:

- The patient is asymptomatic and without a concerning ECG or labs. All patients require outpatient follow-up.
- Patients may benefit from outpatient monitoring.

Patients with the following features require admission:

- Symptomatic (hypotension, lightheadedness, altered mental status, near syncope, angina, shortness of breath, loss of consciousness).
- Bradycardia as the result of severe high degree AV block.
- Underlying cause not yet reversed (ie, infections, hypothermia, hypothyroidism).
- Patients should be admitted to a closely monitored setting, such as a cardiac ICU, in preparation for placement of a permanent internal pacemaker.

Deep Dive

Background

Epidemiology

In patients over 65 y of age, 1 in 600 develop sinus node dysfunction per year.

Intrinsic causes (primary cardiac electrical system dysfunction) of bradyarrhythmia are much more common in the older adult population (age 50 y and older).

- Examples in older adults: Sick sinus syndrome, Lenegre's disease. Lev's disease.
- Example in infants: Congenital heart block in an infant born to a mother with systemic lupus erythematosus.

Extrinsic causes occur in a range of patient types and vary depending on the causative event or syndrome.

- Acute coronary syndrome, particularly inferior ST-elevated myocardial infarction, is a very common cause of sinus bradycardia and AV block.
- Ingestion, purposeful or accidental, of cardioactive medications and agents is another very common cause in children and adults.
- Among metabolic causes, hyperkalemia is a frequent inciting factor in the development of bradycardia and should be excluded before considering other etiologies.

Pathophysiology

The cardiac electrical (conduction) system is a group of specialized cells present in the walls of the myocardium.

- Sinoatrial node is located in the right atrium
- AV node is located in the interatrial septum
- Bundle of His
- Bundle branches
- Purkinje fibers

Impulses originate in the sinoatrial node, resulting in atrial contraction, and then travel to the AV node. After a delay in the AV node, the signal moves to the bundle of His, bundle branches, and Purkinje system, leading to ventricular contraction.

- The sinoatrial node receives its blood supply from the sinoatrial nodal artery, which arises from the right coronary artery in 60% of people. In the remaining 40%, the sinoatrial nodal artery arises from the left circumflex artery.

- The AV node receives its blood supply from the AV nodal branch of the right coronary artery in 80-90% of patients and the left circumflex artery in 10% of cases.

Bradycardia occurs with abnormalities of the sinoatrial node, atrial tissue, AV node, and conduction system.

The underlying **etiology of conduction abnormalities in bradycardia** can be classified as either intrinsic or extrinsic.

- With intrinsic causes, the conductive tissue is replaced by fibrous tissue (eg, infarction, collagen vascular diseases, infection, infiltrative diseases, and surgical trauma).
- Extrinsic causes are broad and include those related to autonomic dysfunction, medications and toxins, and insults to other organ systems.

Diagnostic Considerations

Clinical Presentation

Ranges from asymptomatic to shock, including impending cardiac arrest.

Cardiac output = Heart rate x stroke volume

- When a low heart rate is no longer compensated by increases in stroke volume, the cardiac output decreases, causing symptoms.

Hypoperfusion can result in shock, manifested by

- Hypotension with abnormal perfusion
- Altered mental status
- Ischemic chest pain
- Pulmonary congestion (shortness of breath)

History

Obtaining a detailed **history** will assist in discovering the underlying etiology of the patient's bradycardia.

Physical Examination

First, assess hemodynamic stability and confirm adequate perfusion.

- Reassuring findings include brisk capillary refill, strong peripheral pulses, and warm extremities.

It is imperative to **obtain a core temperature** (rectal preferable), especially if hypothermia is suspected.

- Bradycardia due to hypothermia will not respond well to electrical impulse or atropine. In fact, pacing may be harmful because the cold cardiac membrane is unstable in hypothermia and prone to refractory ventricular fibrillation.

The exam can also provide guidance for identifying the underlying pathology.

- Heart failure

  Lower extremity edema

  Elevated jugular venous distention

  Rales in the lower lung fields

- Renal failure/impairment

  Look for a dialysis catheter or fistula

- Evidence of trauma (head or abdominal)
- Endocrine/environmental causes

  Low core temperature

  Non-pitting lower extremity edema, obesity (hypothyroidism)

- Electrolyte disturbances

  Weakness, tremor, muscle contractions, Chvostek sign, Trousseau sign, dysphagia, nystagmus, psychosis, spasms, depression

ECG

ECG is the first and most critical diagnostic tool in the evaluation of the bradycardic patient.

Narrow QRS bradycardias usually have a better prognosis and do not require permanent pacemakers. These cases are usually vagally induced or the result of a reversible cause (eg, beta blocker overdose).

Patients with a second-degree AV block type II (Mobitz II), complete AV block, or ventricular pause >3 seconds, as well as patients with wide complex bradycardias without a reversible underlying cause, will usually require aggressive resuscitation and eventual permanent pacemaker insertion..

Radiographic Evaluation

Use the clinical presentation and evaluation to guide imaging decisions.

Obtain a head CT if there is concern for increased ICP, hemorrhage, or mass.

Order a chest X-ray if there is concern for decompensated heart failure, pacemaker lead fracture or migration (see chapter: **Pacemakers**), or evidence of sarcoidosis.

Perform a bedside ultrasound (ie, focused assessment with sonography in trauma exam) and CT of the abdomen if there is concern for intra-abdominal hemorrhage.

Transthoracic echocardiogram (TTE)

- Transthoracic echocardiogram is a level-one recommendation of the American College of Cardiology/American Heart Association/Heart Rhythm Society guidelines for patients who are bradycardic and have the following:

  Newly identified left bundle branch block

  Second-degree Mobitz type II AV block

  High-grade AV block or

  Third-degree AV block with or without apparent structural heart disease or coronary artery disease

- Transthoracic echocardiogram should be part of the initial evaluation in patients whose symptoms are suspected to be cardiac in origin (eg, MI, structural disease).
- A bedside echo in the ED can be performed once the patient is stable in order to observe for cardiac motion abnormalities, valvular abnormalities, etc.
- The patient may need a formal ECG study during their stay and work-up.
- Transthoracic echocardiogram assists with determining appropriate pacemaker capture.
- Transthoracic echocardiogram should also evaluate for wall motion abnormalities in the setting of suspected myocardial ischemia. Patients with symptomatic bradycardia related to cardiac ischemia will often display right ventricular wall motion abnormalities (due to the vascular supply of the sinoatrial node and moderator band).

Laboratory Evaluation

Consider hyperkalemia early in an evaluation, and if possible, obtain bedside potassium levels.

- Elevated potassium levels can present with profound bradycardia and can mimic heart blocks (especially third-degree) on an ECG.
- Examine the ECG for signs of hyperkalemia, such as peaked T waves, widened QRS complexes, right axis deviation, and a prolonged P-R interval.
- At-risk populations include patients with renal impairment, hypoperfusion states such as shock, or possible compartment syndrome.
- However, no specific potassium cut-off levels are associated with bradycardia or specific ECG findings (eg, peaked T waves).

Other electrolytes

- Magnesium

  A level below 0.75 mmol/L is defined as hypomagnesemia. Magnesium acts similarly to a calcium channel blocker on the myocardial membrane action potential. Furthermore, magnesium plays a role in the action of the Na/K ATPase. Depletion of magnesium renders a cell unable to retain potassium intracellularly, leading to intracellular potassium depletion. Hypomagnesemia also affects the kidney's ability to retain potassium and results in hypokalemia. Magnesium depletion can cause arrhythmias (initially tachydysrhythmias, then bradydysrhythmias) and sudden death.

  Populations at risk for hypomagnesemia include elderly patients with decreased magnesium consumption, patients on loop or thiazide diuretics, and patients with acute or chronic use of alcohol.

  The ECG findings of **hypomagnesemia** are primarily a **prolonged QT,** which can lead to torsade de pointes.

- Calcium

  The normal concentration of total extracellular calcium is 9-10.5 mg/dL, half of which is found in the free ionized form, Ca++. The other half is protein-bound.

  A level below 8.8 mg/dL is defined as hypocalcemia. Levels above 10.5 mg/dL define hypercalcemia.

  Calcium levels are inversely proportional to QT length.

  Hypercalcemia is associated with **short QT syndrome** and arrhythmias, especially if the patient is on digoxin.

  Hypocalcemia results in a **prolonged ST segment and QT interval.**

  Bradyarrhythmia can result from either type of calcium abnormality.

  - Hypocalcemia impairs myocardial contractility. The myocardial sarcoplasmic reticulum does not store a large amount of calcium, and thus, it depends on extracellular calcium levels. Prolonged hypocalcemia can result in heart failure.
  - Hypercalcemia has been reported to cause bradyarrhythmias as well, possibly due to calcification and dysfunction of the AV node.

  Populations at risk include patients with renal impairment, vitamin D deficiency, acute pancreatitis, magnesium deficiency, hyperparathyroidism, post total thyroidectomy, or cancer.

Glucose

- Hypoglycemia alters the physiology of the myocardium. It has proarrhythmic effects and prolongs the QTc, which can lead to torsades de pointes. Hypoglycemia can also induce hypokalemia, which further causes arrhythmias and can lead to sudden death.

- One study showed that 47% of patients with type 2 diabetes who presented with severe hypoglycemia had hypokalemia as well. The median level of blood glucose in that study was 34 mg/dL thyroid-stimulating hormone.
- Hypothyroidism can cause sinus node dysfunction, AV blocks, and even torsade de pointes.

Check blood urea nitrogen and creatinine, as patients with acute and chronic renal failure are at high risk of electrolyte imbalances.

Other labs should be guided by clinical presentation:

- Troponins, creatine kinase, myoglobin, brain natriuretic peptide (BNP), or N-terminal-proBNP if there is concern for cardiac ischemia or other cardiac disease.
- Serum drug levels (e.g., digoxin).
- Infectious titers (rapid plasma reagin for syphilis or Lyme).

Therapeutic Considerations

Management of bradydysrhythmias is based on various factors including patient stability, severity of symptoms, and the risk of progression to third-degree AV block or asystole.

When treating a patient with bradyarrhythmia, it is paramount to determine the following:

- 1. If the patient is stable or unstable.

    a. “Unstable” is defined by any of the following: hypotension, decreased level of consciousness, ischemic chest pain or anginal equivalent, or acute heart failure.

    b. If any of the above are presumed *to be due to* bradycardia, then the patient is assumed to have an “unstable bradyarrhythmia” and requires immediate treatment.

- 2. The ECG rhythm.
- 3. The underlying cause of the bradyarrhythmia.

    Management of bradyarrhythmia depends on the underlying etiology.

Managing the Patient with Unstable Bradyarrhythmia

Unstable patients are generally defined as patients with hypotension with poor perfusion, altered mental status, ischemic chest pain, and dyspnea from acute heart

failure. These patients likely require urgent intervention to improve perfusion.

If patient is unstable, initiate either atropine or transcutaneous pacing.

- Atropine 0.5-1 mg IV q3-5min until resolution or the maximum dose (3 mg) is reached, should be used for unstable sinus bradycardia or Mobitz I.
- Transcutaneous pacing should be used if atropine is ineffective or for Mobitz II or third-degree AV block.

  Patients may respond to atropine with total higher initial (eg, 1 mg) and maximal doses (3 mg).

  Atropine is vagolytic and will only affect the sinoatrial node and proximal AV node. Bradyarrhythmias below the AV will not respond well to atropine (wide QRS).

  - Atropine is unlikely to benefit post-cardiac transplant patients and should be avoided.
- If there is no response to atropine and/or transcutaneous pacing, administer epinephrine 20-50 µg IV bolus or epinephrine 2-10 µg/min infusion.
- Transcutaneous pacing serves as a bridge to more definitive therapy or an improvement in the patient's condition. Pacing pads should be placed on the patient regardless of whether pacing is immediately indicated.

Pharmacotherapy

- Atropine is a reasonable first choice.

  Administer atropine 0.5-1 mg IV q3-5min until resolution or the maximum dose (3 mg) is reached.

  Atropine is an anticholinergic medication that works by parasympathetic blockade and direct vagolytic action. Some conditions (eg, heart transplant, hypothermia, some Mobitz II, third-degree heart block cases) will have a poor response to atropine; for this reason, epinephrine is recommended as the first-line drug of choice in these situations.
- Epinephrine is a reasonable second-choice agent.

  Administer epinephrine 20-50 µg IV bolus **or** epinephrine 2-10 µg/min IV infusion or atropine IV 0.5-1 mg if epinephrine is not readily available. Epinephrine exerts both positive inotropic and chronotropic effects on the heart; unlike atropine, it exerts its effect directly on the myocardium.
- Consider isoproterenol as a third-line agent. It is a pure beta agonist (beta 1 and beta 2) and exerts its effects directly on the myocardium.

  Isoproterenol, if available, 2-10 µg/min IV, titrate to effect.

  In ACS presentations, isoproterenol can exacerbate the ischemic process.

As stabilization occurs, efforts should be made to identify, diagnose, and reverse the underlying cause. Once the cause is identified, immediately initiate therapy for the specific condition(s) to improve bradyarrhythmia and increase cardiac output.

Managing the Stable Bradyarrhythmia Patient

Stable patients are asymptomatic with a normal blood pressure and no signs of heart failure, altered mental status, chest pain, or lightheadedness.

The underlying cause of bradyarrhythmia should be identified; this may occur in either an inpatient or outpatient setting.

Caution is advised for severely bradycardic and normotensive patients.

- Endogenous sympathetic responses result in vasoconstriction.
- Patients may have a low cardiac output and be in shock despite a 'normal' blood pressure. Always consider the following indicators of instability and potential shock:

  Hypotension with abnormal perfusion

  Altered mental status

  Ischemic chest pain

  Pulmonary congestion (shortness of breath)

Avoid treating hypertension in patients with severe bradycardia and hypertension. Vasodilation can lead to hemodynamic collapse.

Other Pharmacologic Considerations

Dobutamine is an effective beta agonist but has vasodilatory effects and should be avoided in the hypotensive, crashing bradycardic patient.

Dopamine has chronotropic properties and can be used if other agents are unavailable or ineffective.

- Dopamine has potent vasoconstrictive effects and may cause skin necrosis with extravasation.

Intralipids should be considered in cases of bradyarrhythmia due to medication toxicities such as lidocaine infusion or in patients with recent nerve blocks, beta-blocker or calcium channel blocker toxicity/overdose, etc.

- **Therapy for drug of toxin-related bradycardia**

Disposition

Decisions regarding patient disposition should be based on the etiology of the underlying cause of bradydysrhythmias and the overall patient condition.

Discharge

Patients with non-concerning, extrinsic bradydysrhythmia causes may be discharged if the patient is asymptomatic with normal vital signs and the underlying cause has been reversed (eg, drug overdose, vagal stimulation, hypoxia).

Patients need clear follow-up and discharge instructions (eg, do not strain while defecating, discontinue a certain medication until follow-up, etc.).

Hospital Admission

Most intrinsic bradydysrhythmias will require hospital admission, as they are frequently not readily reversible and require additional specialized management of the underlying condition. Insertion of a permanent pacemaker is the definitive treatment.

Symptomatic patients (ie, hypotensive, altered mental status, chest pain, etc.) with intrinsic or extrinsic causes, as well as those with unresolved underlying conditions, should be admitted to a closely monitored setting.

Additional Information

Links to EM:RAP

Bradycardia- Initial Approach

References

show