Chapter 59
Approach to Bradycardia in the ICU

59.1 Introduction

Bradycardia is defined as a heart rate originating from the sinus node at less than 60 beats per minute. It can be physiological, as seen in well-trained athletes, or pathological, indicating underlying cardiac or systemic issues. While asymptomatic and physiological bradycardia generally does not require intervention, symptomatic bradycardia demands immediate attention to prevent potential catastrophic outcomes such as syncope, heart failure, or sudden cardiac death. New-onset bradycardia, especially when accompanied by symptoms like dizziness, fatigue, or chest pain, often signals underlying cardiac pathology and necessitates prompt evaluation and management [1–3] [Ref: Algorithm 59.1].

59.2 Pathophysiology and Etiology

Bradycardia results from impaired impulse generation in the sinoatrial (SA) node, impaired conduction through the atrioventricular (AV) node, or both. The causes of bradycardia are diverse and can be categorized into several groups:

59.2.1 Drug-Induced Bradycardia

Medications are a common cause of bradycardia. Drugs such as beta-blockers, calcium channel blockers, digoxin, antiarrhythmic agents, and certain antiepileptics can depress SA node activity or AV node conduction. Overdose of these medications can lead to severe bradycardia requiring emergency intervention.

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[α-2 agonist (clonidine, dexmedetomidine), α-blocker (prazosin), cholinergic agents (neostigmine), beta blockers (metoprolol, bisoprolol, labetalol, carvedilol), antiarrhythmics (amiodarone, diltiazem, verapamil, flecainide), ivabradine, digoxin, and rarely anti-seizure medicines (carbamazepine, lacosamide, phenytoin), Propofol, Lithium].

59.2.2 Metabolic and Endocrine Causes

Metabolic disturbances like hyperkalemia, hypokalemia, hypocalcemia, and hypoxia can affect cardiac conduction. Hypothyroidism slows metabolic processes and can lead to bradycardia. Hypothermia also decreases heart rate by slowing the rate of depolarization in cardiac pacemaker cells. Uremia, advanced liver disease, hypermagnesemia, hypercapnia, and acidemia are the other metabolic causes that can lead to bradycardia.

59.2.3 Cardiovascular Causes

Intrinsic cardiac diseases such as acute myocardial infarction (especially inferior wall MI), anterior wall myocardial infarction, myocarditis, acute hypertension, and infiltrative diseases like amyloidosis, hemochromatosis, and sarcoidosis can damage the conduction system. AV blocks of varying degrees can result from ischemia or fibrosis of the conduction pathways.

59.2.4 Infections and Inflammatory Conditions

Infections such as Lyme disease, endocarditis, and viral myocarditis can involve the cardiac conduction system. Systemic inflammatory diseases like systemic lupus erythematosus and scleroderma may also affect cardiac conduction.

59.2.5 Increased Vagal Tone and Other Causes

Conditions that increase vagal tone, such as vasovagal syncope or increased intracranial pressure, can lead to bradycardia. Sleep apnea is another important cause, particularly in patients experiencing nocturnal bradycardia. Sudden pain, sick sinus, acute stressful vomiting, coughing, straining, micturition (Valsalva), surgeries, and raised intra-cranial pressure (tumor, traumatic brain injury, intracranial hemorrhage,

large acute ischemic stroke, and chronic liver disease) can also lead to bradycardia.

59.3 Clinical Presentation

Patients with bradycardia may present with a variety of symptoms, ranging from mild fatigue and dizziness to syncope and heart failure. Symptoms are often a result of decreased cardiac output due to the slow heart rate. Key clinical features include:

- Dizziness or lightheadedness.
- Syncope or near-syncope.
- Fatigue.
- Shortness of breath.
- Chest pain.
- Confusion or altered mental status.

59.4 Evaluation

59.4.1 Immediate Assessment and Stabilization

A rapid and systematic approach is essential in evaluating a patient with bradycardia. Look for hypotension, acute onset altered mentation, signs of shock, ischemic discomfort, and acute heart failure as these signs mandate immediate treatment.

59.4.2 ABCDE Assessment

- Airway and Breathing: Ensure the airway is open and assess breathing. Provide supplemental oxygen to maintain adequate oxygenation.
- Circulation: Assess hemodynamic status by checking blood pressure, heart rate, and peripheral perfusion. Aim to maintain a mean arterial pressure (MAP) greater than 65 mm Hg.
- Disability: Evaluate neurological status, including level of consciousness and signs of stroke.
- Exposure: Fully expose the patient to assess for any signs of trauma, rash, or other physical findings that may indicate the cause of bradycardia.

Vital Sign Monitoring: Continuous monitoring of vital signs is crucial. Place the patient on cardiac monitors to observe electrocardiogram (ECG) rhythms, measure blood pressure (BP) regularly, and monitor oxygen saturation (SpO_2).

59.4.3 Identification of Reversible Causes

It is important to identify and correct reversible causes promptly. A checklist of potential factors includes:

- Hypoxia.
- Electrolyte imbalances (e.g., hyperkalemia, hypokalemia).
- Hypothermia.
- Toxins or drug overdose.
- Cardiac tamponade.
- Tension pneumothorax.
- Thrombosis (pulmonary or coronary).
- Trauma.

59.5 History and Physical Examination

A thorough history and physical examination help identify potential causes.

59.5.1 History

- Review medications, including recent changes or overdoses.
- Ask about chest pain, dyspnea, palpitations, or syncope.
- Inquire about recent infections, trauma, or exposure to toxins.
- Explore family history of cardiac conditions.
- Assess sleep patterns to evaluate for sleep apnea.

59.5.2 Physical Examination

- Observe general appearance for signs of distress.
- Check for cyanosis or pallor.
- Auscultate the heart for murmurs or gallops.
- Assess lung fields for crackles or rales, indicating heart failure.
- Examine for peripheral edema.
- Perform a neurological assessment.

59.6 Investigations

59.6.1 Electrocardiogram (ECG)

A 12-lead ECG is essential to evaluate the type of bradycardia and identify conduction abnormalities.

- Sinus Bradycardia: Normal P waves with a rate less than 60 bpm.
- First-Degree AV Block: Prolonged PR interval (>200 ms) without dropped beats.
- Second-Degree AV Block Type I (Mobitz I/Wenckebach): Progressive prolongation of PR interval until a QRS complex is dropped.
- Second-Degree AV Block Type II (Mobitz II): Constant PR intervals with intermittent dropped QRS complexes.
- Third-Degree AV Block: Complete dissociation between atrial and ventricular activity.
- Left Bundle Branch Block (LBBB): Widened QRS complexes with specific patterns; requires further evaluation.
- Junctional Rhythm/Escape: Constant RR interval with rate < 60 (around 40), retrograde P-waves may be seen.
- Ventricular Escape: Wide QRS with rate of 30–40 beats per minute.

59.6.2 Laboratory Tests

- Complete Blood Count (CBC).
- Basic Metabolic Panel: Electrolytes (Na^+, K^+, Ca^{2+}, Mg^{2+}), renal function, glucose.
- Thyroid Function Tests: To assess for hypothyroidism.
- Cardiac Enzymes: Troponin levels to evaluate for myocardial infarction.
- Arterial Blood Gas (ABG): To assess oxygenation and acid-base status.
- Drug Levels: If overdose is suspected.
- Genetic Testing: In cases of suspected inherited arrhythmias.

59.6.3 Imaging and Advanced Diagnostics

- Chest X-ray: To assess for cardiac enlargement or pulmonary pathology.
- Echocardiography: Especially important in patients with LBBB or suspected structural heart disease to evaluate ventricular function and structural abnormalities.

- Ambulatory ECG Monitoring (Holter Monitor): For patients with intermittent symptoms to detect transient arrhythmias.
- Sleep Studies: In patients with nocturnal bradycardia or suspected sleep apnea.

59.7 Management

- Management of bradycardia focuses on symptomatic relief, stabilization, and addressing the underlying cause.

59.7.1 General Measures

- Ensure Airway and Breathing: Provide oxygen and support ventilation if necessary.
- Establish IV Access: For medication administration.
- Continuous Monitoring: ECG, blood pressure, and SpO_2.

59.7.2 Pharmacological Treatment

59.7.2.1 Atropine

Atropine is the first-line medication for symptomatic bradycardia.

- Dosage: Administer 1 mg IV bolus.
- Repeat: Every 3–5 minutes as needed.
- Maximum Dose: Do not exceed a total of 3 mg.

59.7.2.2 If Atropine Is Ineffective

When atropine fails to improve the heart rate, consider the following:
Dopamine Infusion.

- Dosage: Start at 5–20 mcg/kg/min.
- Titration: Adjust based on patient response.
- Tapering: Slowly reduce the dose as the patient's condition stabilizes.

Epinephrine Infusion.

- Dosage: Administer 2–10 mcg/min.
- Adjustment: Titrate according to clinical response.

59.7.2.3 Second-Line Medications

Aminophylline.

- Used in refractory cases where standard agents are ineffective.

Glucagon.

- Especially useful for bradycardia induced by beta-blocker or calcium channel blocker overdose.
- Dosage: Initial bolus followed by an infusion as needed. 50 micrograms/kg iv loading dose, followed by a continuous infusion of 1–15 mg/h

Isoproterenol.

- Considered when bradycardia is due to drug toxicity and pacing is unavailable.
- Caution: Use with care due to potential arrhythmogenic effects.

59.7.3 Pacing

- Pacing is indicated when pharmacological therapy is ineffective or contraindicated.

59.7.3.1 Transcutaneous Pacing

- Indication: Symptomatic high-degree AV blocks (second or third degree) unresponsive to atropine.
- Procedure: Apply pacing pads and initiate pacing while preparing for transvenous pacing if necessary.
- Analgesia and Sedation: Consider for patient comfort.

59.7.3.2 Transvenous Pacing

- Indication: Persistent bradyarrhythmias not responsive to medications or transcutaneous pacing.
- Procedure: Insertion of a pacing catheter into the right ventricle.
- Monitoring: Continuous ECG and hemodynamic monitoring.

59.7.3.3 Permanent Pacing

- Indication: Patients with recurrent or severe symptoms, or irreversible causes of bradycardia.

- Device: Implantation of a permanent pacemaker.
- Considerations: Evaluate patient's suitability and preference.

59.7.3.4 Advanced Pacing Options

His-Bundle Pacing

- Targets the His-Purkinje conduction system to maintain physiological ventricular activation.
- Reduces the risk of pacing-induced cardiomyopathy associated with right ventricular pacing.

Cardiac Resynchronization Therapy (CRT)

- Beneficial for patients with heart failure and intraventricular conduction delays.
- Improves ventricular synchrony and cardiac output.

59.7.4 Management of Conduction Delays

Patients with LBBB on ECG require special attention:

- Echocardiography.
- Early assessment of left ventricular function.
- Identifies structural heart disease.
- Consider Advanced Pacing.
- His-bundle pacing or CRT to minimize dysynchrony and preserve cardiac function.

59.7.5 Addressing Underlying Causes

- Medication Review.
- Discontinue or adjust medications contributing to bradycardia.
- Electrolyte Correction.

Normalize levels of potassium, calcium, and magnesium.

- Manage Hypothyroidism.

Initiate thyroid hormone replacement therapy.

- Treat Infections.

Appropriate antimicrobial therapy for conditions like Lyme disease or endocarditis.

- Manage Sleep Apnea

Referral for sleep studies and initiation of continuous positive airway pressure (CPAP) therapy if indicated.

59.8 Patient-Centered Care

- Effective management of bradycardia involves engaging the patient in decision-making.

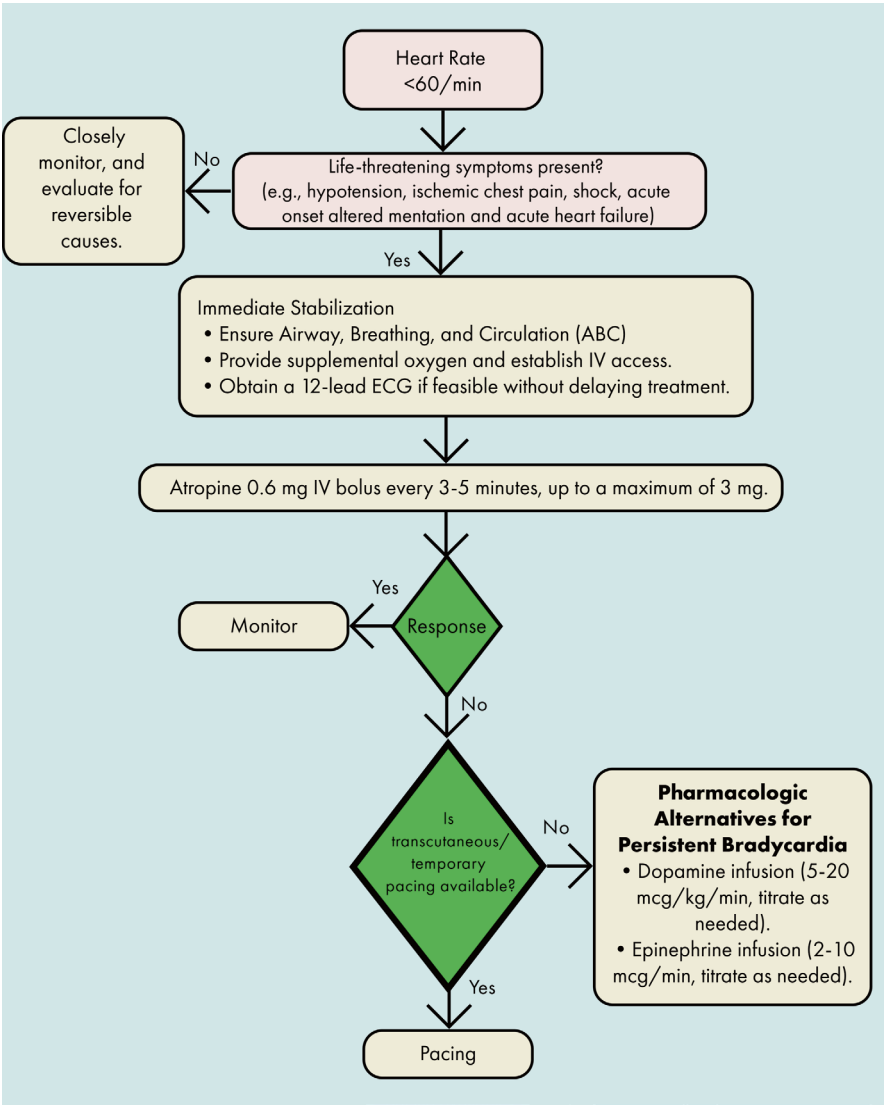
59.9 Shared Decision-Making

- Discussion of Options.
- Explain the benefits and risks of pharmacological therapy versus pacing.
- Patient Preferences.
- Consider the patient's quality of life, occupation, and personal goals.
- Informed Consent.
- Essential before initiating temporary or permanent pacing procedures.

59.10 Conclusion

Bradycardia can range from a benign finding to a life-threatening condition requiring immediate intervention. A systematic approach that includes rapid assessment, identification of reversible causes, appropriate pharmacological therapy, and pacing interventions is essential. Advanced diagnostics and therapies, including His-bundle pacing and CRT, offer improved outcomes for patients with structural heart disease. Engaging patients in their care through shared decision-making enhances adherence and satisfaction with treatment. Recognizing and managing bradycardia effectively can prevent serious complications and improve patient quality of life.

Algorithm 59.1: Approach to bradycardia in the ICU



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