

Evaluation of Bradyarrhythmias in Hospitalized Patients

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What is bradycardia?

Bradycardia is any rhythm that results in a heart rate less than 60 bpm. The number 60, however, is a somewhat arbitrary choice, so a pulse less than 60 bpm may be due to normal physiologic variation rather than disease or illness. In fact, in young or otherwise healthy persons, bradycardia is a manifestation of good health and fitness. Symptomology is key to defining a pathologic state. A slow heartbeat can take origin in several parts of the heart and can be the result of physiologic adaptation, medication effect, or congenital or acquired illness. In general bradycardia arises by one of two pathophysiologic mechanisms: decrease in impulse formation or decrease in conduction system function.

Etiology

Slow heart rates may result from multiple causes including specific cardiac diseases, response to systemic disease, degenerative diseases and medications. Systemic causes include hypoxia, increased intracranial pressure, hypothermia, hypo-thyroidism, electrolyte disorders, and sleep apnea. Cardiac diseases that can cause bradycardia include infiltrative heart disease (sarcoid and amyloid), degenerative disease of the cardiac conduction system, ischemic heart disease, Lyme disease and rheumatic heart disease. Degenerative disease of the conduction system may be isolated to the conduction system (Lenegre's disease) or represent generalized calcification of the cardiac skeleton that includes aortic and mitral valve involvement (Lev's disease). Many drugs can also slow cardiac conduction and result in bradycardia. Drugs that commonly have this side effect are beta-blockers, calcium channel blockers, and digoxin, but these are only a few of the many drugs that can have this side effect.

Initial questions to address

1. *What are the other vitals signs?* A patient with low blood pressure and bradycardia requires more urgent care than a patient with bradycardia and a normal BP.

2. *What is the overall pattern of heart rate since admission?* The ranges of normal heart rates are wide and influenced by many factors including activity, age, medications, pain, fever and level of fitness of patient.
3. *Is the patient symptomatic?*

History

The presence of symptoms with bradycardia often determines the extent of treatment. Sinus bradycardia is most often asymptomatic. Symptoms associated with bradycardia include:

- Syncope
- Dizziness/lightheadedness/pre-syncope
- Congestive heart failure
- Chest pain or anginal equivalent

Pertinent elements of history are previous cardiac history, medications, toxic exposures, and prior illnesses.

Physical Examination

Cardiac auscultation and palpation of peripheral pulses often reveal a slow, regular heart rate. Physical examination can reveal the following:

- Altered mental status
- Cyanosis
- Peripheral edema
- Pulmonary vascular congestion
- Dyspnea and poor perfusion

Differential Diagnosis

The differential diagnosis of bradycardia may be classified by the regularity of the heart rate.

Regular rate:

- Sinus bradycardia
- Complete heart block
- 2:1 AV block
- sinus arrest with escape rhythm
- regularized slow atrial fibrillation

Irregular rate:

- sick sinus syndrome
- second degree AV block (type I and II)
- slow atrial fibrillation

I. Sinus bradycardia-Defined as a sinus node rhythm below 60 bpm. Changes in sinus rate are modulated primarily by

parasympathetic nervous system tone. Many of the causes are listed below:

1. Increased parasympathetic tone- i.e. vasovagal events with venipuncture.

2. Sinoatrial node dysfunction- the presence of bradycardia at inappropriate times. Sinus node dysfunction can also be manifested by periods of sinoatrial node arrest or sinoatrial exit block, or by alternating sinus bradycardia and tachyarrhythmias, called sick sinus syndrome.

3. Myocardial infarction- frequently seen with inferior wall infarctions involving the proximal portion of the right coronary artery and its branch to the sinoatrial node. The presence of sinus bradycardia during the early phases of an acute myocardial infarction is, in general, a good prognostic sign. It does not require therapy as long as left ventricular cardiac output is adequate, and hypotension or CHF are absent.

4. Cushing's reflex- sinus bradycardia with hypertension associated with increased intracranial pressure.

5. Medical disorders- hypothyroidism, hypothermia, OSA, age, carotid sinus hypersensitivity, and infiltrative disease.

6. Drug effect

II. Atrioventricular Node Blocks-

Atrioventricular (AV) block can be defined as a delay or interruption in the transmission of an impulse from the atria to the ventricles due to an anatomic or functional impairment in the conduction system. The conduction disturbance can be transient or permanent. The conduction can be delayed, intermittent, or absent. The commonly used terminology includes first degree (slowed conduction without missed beats), second degree (missed beats, often in a regular pattern such as 2:1, 3:2, or higher degrees of block), and third degree/complete AV block.

A. Mobitz type I second degree AV block-

Rhythm described by Wenckebach where progressive PR interval prolongation precedes a non-conducted P wave. There is usually a gradually falling R-R interval with an eventual dropped beat. Causes include:

1. Acute Myocardial infarction- most commonly with inferior wall infarcts related to ischemia affecting the atrioventricular nodal branch of the right coronary artery.

2. Drug effect- many drugs such as beta-blockers, digoxin, amiodarone, and calcium channel blockers can cause this finding.

3. Infections- particularly the initial phases of rheumatic fever and Lyme disease.

4. Other causes- can be seen in asymptomatic normal adults with a high level of parasympathetic nervous system tone, such as sleeping, highly trained aerobic athletes.

B. Mobitz type II block- the PR interval remains unchanged prior to a P wave that fails to conduct to the ventricles. The PR interval may be normal or prolonged in Mobitz type II block. Mobitz type II block is almost always below the AV node, occurring in the bundle of His in about 20 percent of cases and in the bundle branches in the remainder. Patients with bundle branch involvement also have axis shifts and QRS widening depending upon the location of the block. In addition, at least two-thirds of patients with this disorder have bifascicular or trifascicular disease. Causes include:

1. Acute myocardial infarction- large anterior wall infarcts can cause this rhythm.

2. Degenerative fibrosing diseases- involving the His bundle

3. Infections diseases- like viral myocarditis, rheumatic fever and Lyme disease.

C. Third-degree AV block- No atrial impulses reach the ventricle in third degree or complete heart block. The block can exist in the AV node or in the infranodal specialized conduction system. A His-bundle electrocardiographic study can determine the site of block quite accurately, but the escape rhythm provides important clues. As a general rule, the more distal the block, the slower will the escape rhythm. The P wave and QRS complexes are independent of one another. This rhythm is associated with the same conditions as Mobitz type II heart block.

D. Malfunctioning pacemaker

Diagnostic Evaluation and Assessment

A 12-lead ECG is the first diagnostic test. One should first look for the presence of P waves. The absence of a P wave may indicate failure of the SA node to fire (sinus arrest) or failure of the SA node to excite the atria (sinus exit block). These two conditions are indistinguishable by ECG and are referred to as sick sinus syndrome. If the syndrome is advanced, a regular escape rhythm may appear. P waves are completely

absent in atrial fibrillation. In regularized atrial fibrillation, complete heart block is present at the AV node and a lower escape pacemaker begins to fire (resulting in a rate of 40-60 bpm). This particular rhythm is seen in digoxin toxicity and will appear as an irregularly irregular bradycardia with no P waves. After assessment for conduction block as mentioned above, look for ST segment depression or elevation, T wave inversion, and new Q waves to evaluate the patient for ischemic events. To assess for pacemaker function, look for pacer spikes.

Lab Studies

If the cause of bradycardia could be due to electrolytes, drugs, or toxins, laboratory studies may be helpful. In cases of sick sinus syndrome, routine laboratory studies are rarely of specific value. Reasonable screening studies - especially if the patient is symptomatic, and this is the initial presentation - include the following: electrolytes, glucose, calcium, magnesium, thyroid function tests and toxicologic screening. Routine imaging studies are rarely of value in the absence of specific indications. Other important diagnostic considerations are sleep apnea, infectious diseases, head injuries, hypothermia, and hypoglycemia

Treatment

If no underlying heart disease is detected, the heart's response to exercise is normal, and there are no symptoms of low cardiac output, treatment may not be required. It may be necessary to monitor the heart rate and rhythm periodically. People with cardiac symptoms and conditions should be treated. Symptomatic bradycardia treatment should be undertaken immediately after diagnosis. Symptomatic sinus bradycardia, first-degree AV block, or Mobitz I second-degree AV block may respond acutely to atropine treatment, 0.5-2.0 mg IV. For irreversible causes of symptomatic bradycardia, pacemaker therapy should be considered. Treatment for symptomatic bradycardia should also include discontinuation of medications that slow the heart rate. Temporary pacing is best achieved by insertion of a temporary transvenous pacemaker, although placement of an external transthoracic unit can be used. Temporary pacing is indicated for: 1) Mobitz type II second-degree or third-degree AV

block associated with an acute MI; 2) Symptomatic AV block associated with drug toxicity that is likely to be prolonged (amiodarone toxicity); 3) Sinus bradycardia with severe congestive heart failure; 4) Prolonged sinus pauses (>3.5 sec) associated with syncope. Contemporary pacemakers can maintain AV synchrony and adapt the rate of pacing to mimic the normal physiologic heart rate response to exertion. Before a pacemaker is implanted, the patient must be free of any active infections, and anticoagulation issues must be carefully considered. Hematomas over the pacemaker pocket are most often seen in patients who are receiving anti-coagulants and may require surgical intervention in severe cases. Indications for permanent pacemakers are discussed fully in the recommendations from AHA, ACC and the NASEP (*Circulation* 106:2145, 2002). Class I indications for permanent pacemakers are: 1) Symptomatic sinus bradycardia or AV block; 2) Sinus bradycardia as a result of necessary drug therapy; 3) Symptomatic chronotropic incompetence; 4) Advanced AV block; 5) Intermittent complete heart block; 6) Intermittent type II second-degree block; 7) Alternating bundle branch block; and 8) Recurrent syncope with carotid sinus massage causing asystole > or equal to 3 seconds.

References

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