

The approach to Bradycardia

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1. Definition:

Bradycardia is defined by a heart rate less than the lower limit of normal for age.

Guidelines for bradycardia based on a 12-lead ECG recorded during the awake state are as follows:

- 0 – 3 years: <100 bpm
- 3 – 9 years: < 60 bpm
- 9 – 16years: < 50 bpm

Guidelines for bradycardia based on 24-hour Holter monitoring are as follows:

- 0 – 2 years: < 60 bpm while asleep, < 80 while awake
- 2 – 6 years: < 60 pbm during sleep or awake
- 6 – 11 years: < 45 bpm during sleep or awake
- Older than 11 years: < 40 bpm during sleep or awake

2. Background

Bradycardia can be caused by intrinsic dysfunction of or injury to the heart's conduction system, or by extrinsic factors acting on a normal heart. Both intrinsic and extrinsic factors can affect any part of the heart's conduction system, including the SA node, AV node or bundle of His.

3. Basic Anatomy and Physiology

The SA node

The SA node is the pacemaker of the heart. In sinus arrest, there is failure of the SA node to generate an impulse; whereas in SA block there is an interruption in the transmission of the impulse in the atrium.

The AV node

The AV node conducts the electrical impulse from the atria to the ventricles via the bundle of His. There are three forms of AV block: 1st degree block, 2nd degree block, or 3rd degree block. In 1st degree, there is a prolonged P-R interval; however all atrial impulses are conducted to the ventricle (not a cause of bradycardia, but included for completeness). There are two types of 2nd degree AV block: Mobitz type I and II. In Mobitz type I (Also known as Wenckebach), the P-R interval progressively lengthens until a P wave is not conducted. In Mobitz type II, occasional atrial beats are not conducted to the ventricle. In 3rd degree block, or complete block, atrial impulses are not conducted to the ventricle. Complete block has a high mortality rate.

Increased parasympathetic tone via the vagus nerve both decreases sinus node pacing rate and slows conduction through the AV node.

4. Presentation

Whether or not the child is symptomatic depends on the severity of the bradycardia, any related cardiac problems and on the child's age. While the infant may present with non-specific symptoms of failure to thrive or exhaustion with feeds, the older child may complain of exercise intolerance, dizziness and/or syncope.

Severe bradycardia may present with cardiogenic shock (poor cardiac output and decrease in perfusion, measured in decreased mental status, poor blood pressure and decreased urine output). Refer to PALS guidelines for management of acute symptomatic bradycardia.

5. Questions to Ask

- Is the child able to increase heart rate with exercise?
- History of prematurity or small for gestational age.
- Associated symptoms? Ie. Fatigue? Lightheadedness. Syncope.
- Previous history of cardiac surgery or cardiac catheterization.
- History of medications that could affect the conduction system (directly or via activation of PNS)
- Family History of autoimmune disease eg. a maternal history of SLE.
- Family history of congenital heart disease.
- Family history of unexplained sudden death.

6. Differential Diagnosis

Sinus bradycardia is simply a slowing of the normal heart rhythm. Reversible causes of sinus bradycardia include hypothermia, hypothyroidism, anorexia nervosa, malnutrition, hypokalemia, and hypoxia. In an otherwise well child sinus bradycardia can be a non-pathological finding. For this diagnosis the 12-lead ECG must be normal, with normal P-waves, but with the rate below normal for age. Children with benign sinus bradycardia are asymptomatic, and follow a benign course. These children show normal heart rate reactivity to exercise (able to increase heart rate to above 100 bpm). Up to 35% of all individuals have sinus bradycardia, but the incidence is higher in well trained athletes.

In **sick sinus syndrome**, an irregular tachycardia is followed by a slowed discharge from the sinus node. There is failure of the heart rate to elevate in response to exercise or stress. In children, this is most commonly seen following surgical correction of congenital heart defects. Myocardial diseases such as cardiomyopathies, inflammatory or ischemic myocardial diseases as well as conduction abnormalities due to Kawasaki's disease can also cause SA node dysfunction. There are rare familial causes, including mutations of the SCN5A sodium channel gene.

Medications that can cause sinus nodal dysfunction, include digoxin, beta blockers, calcium channel blockers, lithium and clonidine.

Increased Vagal tone, as mentioned, decreases heart rate via its effects at both the SA and AV nodes. Examples include cholinergic medications (phenylephrine, neostigmine), sedatives (morphine) and nasopharyngeal or esophageal stimulation (eg. due to gastric reflux, breath holding, vomiting, coughing or iatrogenic causes including intubation, placement of nasopharyngeal tube, suctioning, etc.)

AV nodal block can be caused by intrinsic or extrinsic causes. Intrinsic causes of AV block include congenital heart defects such as Ebstein's anomaly, ASD and AVSD. Extrinsic damage to the AV node can be post-surgical/post-cardiac catheterization or due to inflammation, such as in rheumatic fever, infective endocarditis, myocarditis, Lyme disease and diphtheria. Maternal lupus leads to congenital heart block due to the presence of Anti-Ro and Anti-La maternal antibodies

that crossed the placenta antenatally. Digoxin toxicity is an important extrinsic cause of AV block.

7. Procedures for Investigation

Bradycardia and the specific conduction abnormality can be diagnosed using the 12-lead ECG or the 24-hour holter monitor (ambulatory monitoring).

Exercise stress testing is not needed in the diagnosis of bradycardia but may be helpful to determine chronotropic competence. In cases of pathological causes of bradycardia there is a suboptimal response in heart rate to exercise, while in sinus bradycardia or in bradycardia due to an increased vagal tone, there should be a normal response of heart rate to exercise.

8. References

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