Bradydcardia

Bradycardia is strictly defined in adults as a pulse rate below 60 beats per minute (bpm). However, few individuals are symptomatic unless the heart rate is below 50 bpm. Top endurance athletes may have a resting heart rate in the very low 30s without needing any intervention with anticholinergics, isoprenaline, adrenaline (epinephrine), chest compressions or the insertion of an emergency pacemaker.\(^1\)

Physiologically, heart rate can vary in normal adults from 40 bpm up to 180 bpm. However, a relative bradycardia may be greater than 60 bpm if that rate is too slow for the haemodynamic requirements of the patient.

Children tend to have a higher resting pulse rate than adults and generally the smaller they are, the faster the heart rate. Hence, in a neonate, bradycardia may be defined as a rate below 100 bpm.

Epidemiology

It is impossible to give meaningful figures on incidence and prevalence. In most young people bradycardia is physiological and represents athletic training. The incidence of pathological bradycardia rises with age as the underlying causes become more frequent.

Presentation

Bradycardia may be asymptomatic but can present with syncope, fatigue or dizziness. Ischaemic chest pain, Stokes-Adams attacks, hypoxic seizures, congestive heart failure, cardiovascular collapse and sudden cardiac death may occur, depending on the underlying cause of bradycardia.\(^2\)

History

- Establish whether there is any history of chest pain or collapse.
- Ask whether there is any shortness of breath on exertion. It is important to ask about exercise and tolerance of exercise. An athlete with a resting bradycardia will raise heart rate and cardiac output to meet demand. Anyone who can sustain even moderate exercise without undue distress is most unlikely to have a pathological cause.
- Ask whether the patient is aware of heart rate - whether it is always slow or just at times; whether there are any ‘thumps’ in the chest, extra or missed beats.
- Establish whether there is a history of faintness, dizziness, nausea or chronic fatigue.
- It is important to ask about medication, as drugs are an important cause of bradycardia.

Examination

- Note whether the patient looks well or there are signs of inadequate cardiac output - for example, cold peripheries, peripheral cyanosis or features of congestive cardiac failure.
- Examination of jugular venous pressure (JVP) may reveal:
  - Elevation of the JVP in heart failure.
  - Cannon waves, which may be seen in complete atrioventricular (AV) dissociation. This is an intermittent very high jugular pulse as the atrium contracts against a closed tricuspid valve.

- Examine the pulse carefully and count for at least 30 seconds (the slower the rate, the longer the duration). An athlete may show sinus arrhythmia but this is rare beyond 30.
- Note any shift in the cardiac apex.
Aetiology

Sinus bradycardia may be physiological with a high resting vagal tone - eg, in athletes or the diving reflex.

Sinus bradycardia

The causes of a pathological sinus bradycardia include:

- Drugs (negative chronotropes):
  - Betablockers.
  - Calcium-channel blockers.
  - Digoxin.
  - Amiodarone.
  - Clonidine.
  - Verapamil.

- Hypoxia.
- Pain (may also cause tachycardia).
- Hypothyroidism.
- Hypothermia.
- Cushing's reflex (systemic response to raised intracranial pressure, leading to widening pulse pressure, irregular breathing and bradycardia).
- Acute myocardial infarction.
- Sick sinus syndrome.
- Pericardial tamponade.
- Adrenal insufficiency.
- Severe obstructive jaundice.
- Pleural or peritoneal stimulation.
- Rarely, infection (eg, typhoid may cause relative bradycardia).

Ectopic atrial rhythm or wandering atrial pacemaker

- Ectopic atrial foci, if ≥3 foci = wandering atrial pacemaker.
- Different P-wave morphologies ± PR duration variations.
- Not normally clinically significant.

Sinoatrial (SA) block or sinus exit block

- SA node fails to produce an impulse or not conducted to atria.
- ECG typically shows absent P waves with escape rhythm:
  - Junctional - narrow complexes at 45-60 bpm.
  - Ventricular - wide complexes at 30-45 bpm.

- Usual causes:
  - Ischaemia.
  - Hyperkalaemia.
  - Excess vagal tone.
  - Negative chronotropes.

- Treat if symptomatic.

Sick sinus syndrome

See also the separate Sick Sinus Syndrome article.

- Tachycardia-bradycardia syndrome (characterised by bursts of atrial tachycardia interspersed with periods of bradycardia; paroxysmal atrial flutter or fibrillation may also occur).\\(^{[2]}\\)
- Causes:
  - SA and AV nodal fibrosis.
  - Myocardial ischaemia.
  - Congenital heart disease.

- ECG may show SA block, sinus or atrial bradycardia with bursts of atrial tachycardia (usually atrial fibrillation).
- Management:
  - Treat any symptomatic acute arrhythmia.
  - Most require a permanent pacemaker for the bradycardia component ± anti-arrhythmic medication (eg, digoxin or verapamil) to suppress tachycardias.

AV blocks

See also the separate ECG Identification of Conduction Disorders article.
• With all AV blocks additionally rule out:
  • Myocardial ischaemia or infarction.
  • Lyme disease.
  • Myocarditis or endocarditis.
  • Systemic lupus erythematosus (SLE).

• First-degree:
  • Slow AV node conduction.
  • ECG: PR interval >200 ms.
  • All atrial impulses conducted to ventricles.
  • Benign.

• Second-degree:
  Some atrial impulses are not conducted to ventricles:
  • Mobitz type I (Wenckebach):
    • AV node conduction defect.
    • ECG: repeated lengthening of PR until a P is not followed by a QRS complex.
    • Usually asymptomatic and treatment not required.
    • Treat if symptomatic or in the context of inferior myocardial infarction.
  
  • Mobitz type II:
    • Conduction defect below AV node.
    • Degenerative: Lev's disease (Lenègre-Lev syndrome), which is an acquired complete heart block due to idiopathic fibrosis and calcification of the electrical conduction system.
    • ECG: constant PR with intermittent QRS absence.
    • Risk of progression to third-degree block.
    • Atropine ineffective as block below AV node.
    • Permanent pacing is required if symptomatic.

• 2:1 block:
  • ECG: 2 P waves for every QRS complex.
  • May occur in digoxin toxicity or ischaemia.
  • Needs further electrophysiological tests to determine treatment.
• **Third-degree:**
  - AV dissociation.
  - Atrial impulses not conducted to ventricles.
  - ECG: both P waves and QRS escape complexes may be present but occur independently.
  - Treat as below, although atropine is unlikely to be effective and a permanent pacemaker will be required.
  - Myocardial fibrosis is the most common cause.
  - Associations:
    - Inferior acute myocardial infarction.
    - Sick sinus syndrome.
    - Mobitz type II.
    - Second-degree block plus new bundle branch or fascicular block.
Investigations

Consider:

- A 12-lead ECG will indicate the true nature of the rhythm and possibly show ischaemic changes, myocardial infarction and other conduction defects like bundle branch block.
- If the bradycardia is variable, ambulatory ECG monitoring may be necessary.
- Coronary heart disease may require investigation.
- Electrolytes, glucose, calcium, magnesium, TFTs and toxicology to exclude causes as above.

General management

- Rule out and treat any underlying causes of sinus bradycardia.
- Treatment is indicated if significantly symptomatic (syncope, hypotension, cardiac failure).
- Diagnose and treat myocardial ischaemia.
- If symptomatic (often not until heart rate <40), treat as below.

Initial management

- Resuscitation.
- Intravenous access with cannula insertion.
- Blood tests:
  - FBC.
  - U&Es.
  - LFTs, including albumin and total protein.
  - Blood glucose.
  - Calcium.
  - Creatinine.
  - Cardiac enzymes (troponin).
  - TFTs.
  - Digoxin level if appropriate.

- ECG.
- Treat the underlying cause if present (cease negative chronotrope, correct electrolytes, etc).
- Treat bradycardia if adverse features:
  - Shock
  - Syncope
  - Myocardial ischaemia
  - Heart failure

- Drugs:
  - Atropine: 0.5 mg IV.
  - If response is not satisfactory, repeat atropine up to a maximum total dose of 3 mg OR transcutaneous pacing OR consider alternative drugs, such as isoprenaline infusion or adrenaline (epinephrine) infusion.
  - Further alternatives include:
    - Aminophylline
    - Dopamine: 5-20 micrograms/kg/minute through central line.
    - Glucagon (if bradycardia is caused by a beta-blocker or a calcium-channel blocker).
    - Glycopyrrolate (may be used instead of atropine).

- Once there is a satisfactory response, continue observation if there is a risk of asystole (recent asystole, Mobitz II AV block, complete heart block with broad QRS, ventricular pause >3 seconds).

- Pacing may be required if symptomatic and drug therapy fails.
  - Temporary pacing
  - Permanent pacemaker

- With excessive vagal tone it can be corrected either by ceasing the activity that caused excessive vagal tone or by giving a drug such as atropine.
- Hypothyroidism, hypothermia, raised intracranial pressure, etc, need treatment of the underlying condition.
- Infective causes usually recover spontaneously.
- Where heart block or sick sinus is the cause, implantation of a pacemaker may be required.

Prevention

- Appropriate treatment of any underlying cause - eg, hypothyroidism.
- Glycopyrrolate (antimuscarinic agent) is used in anaesthesia to prevent the bradycardia induced by cholinergic drugs such as the anticholinesterases.

Further reading & references
3. Adult Bradycardia Algorithm; Resuscitation Council (UK), 2015
4. Guidelines on cardiac pacing and cardiac resynchronization therapy; European Society of Cardiology (2013)

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