Bradyarrhythmias

**Differential diagnosis of bradyarrhythmias**

- **First degree heart block**  
  - All P waves are conducted to the ventricles but there is a persistent delay resulting in a prolonged PR interval (>0.2 s)

- **Second degree heart block**  
  - Some P waves are conducted to the ventricles but not all; there are two main types
  - Mobitz type I (Wenckebach): progressive prolongation of the PR interval until a P wave fails to be conducted
  - Mobitz type II: constant PR interval but occasionally a P wave fails to be conducted; this can occur in a regular (e.g. 2:1 or 3:1) or irregular fashion

- **Third degree (complete) heart block**  
  - No P waves are conducted to the ventricles; the atria and ventricles are electrically disconnected
  - Both P waves and QRS complexes may be seen occurring at a regular but different rate, with no relationship between them; the precise rate and duration of QRS complexes depends on where they arise from; foci more proximal to the atrioventricular (AV) node will result in narrower, faster complexes eg 50 bpm, whereas foci distal to the AV node will result in broader, slower complexes eg 30 bpm

- **Sick sinus syndrome**  
  - Ischaemia or fibrosis leads to degeneration of the sino-atrial (SA) node; results in sinus pauses or arrest; escape rhythms may emerge, known as ‘tachy-brady syndrome’

**Aetiology of bradyarrhythmias**

- **Physiological**  
  - Particularly in young, cardiovascularly fit people

- **Cardiac**  
  - Post-myocardial infarction (MI)
  - Sinus node disease
  - AV block

- **Non-cardiac**  
  - Vasovagal
  - Hypothermia
  - Hypothyroidism
  - Raised intracranial pressure in association with hypertension

- **Drug-induced**  
  - Calcium channel blockers
  - Beta blockers
  - Digoxin
  - Amiodarone

**Clinical features of bradyarrhythmias**

- **Adverse features**  
  - Shock: hypotension, diaphoresis, pallor, increased capillary refill time (CRT)
  - Syncope: transient loss of consciousness
  - Myocardial ischaemia: ischaemic chest pain and/or ischaemic electrocardiogram (ECG) changes
Cardiac failure: orthopnoea, paroxysmal nocturnal dyspnoea, bibasal crepitations, raised jugular venous pressure (JVP)

### Initial investigation of bradyarrhythmias
- 12 lead ECG
- Venous blood gas (VBG)
- Bloods
  - Full blood count
  - Urea & electrolytes
  - Magnesium
  - Bone profile (particularly noting calcium and phosphate)
  - Thyroid function tests
  - Other: liver function (useful pre-medication); coagulation (may need anticoagulation)
- Chest radiograph (CXR)

### Further investigation of bradyarrhythmias
- Echocardiogram (echo)

### Initial management of bradyarrhythmias
- Assess patient from an ABCDE perspective
- Maintain a patent airway
  - Use manoeuvres, adjuncts, supraglottic or definitive airways as indicated
- Controlled oxygen
  - Maintain saturations ($\text{SpO}_2$) 94-98%
- Attach monitoring
  - Pulse oximetry
  - Non-invasive blood pressure
  - Three-lead cardiac monitoring
  - Defibrillator pads
- Request 12 lead ECG
- Obtain intravenous (IV) access and take bloods
- Give IV fluid challenge if appropriate and repeat as necessary
- Identify and treat any reversible causes e.g. electrolyte abnormalities on initial VBG

- If adverse features are present [shock, syncope, myocardial ischaemia, heart failure]:
  - Atropine 0.5 mg IV
  - Repeat even few minutes up to a maximum dose of 3 mg
- If adverse features persist despite atropine, attempt transcutaneous pacing under general anaesthesia or conscious sedation
  - Once ready, set the defibrillator to pacing mode, select a pacing rate of 60-90 bpm
  - Select the lowest current initially
  - Gradually increase until both electrical capture (QRS complex after each pacing stimulus) and mechanical capture (palpable pulse corresponding to each QRS complex) are achieved
    - Once capture achieved, increase the ampage by 10mv to ensure capture.
  - This is only a temporary intervention an arrangements should be made for prompt insertion of a transvenous pacing wire
  - If there is a delay in achieving transcutaneous pacing, other pharmacological measures can be used in the interim e.g. adrenaline 2-10 mcg/min IVI
- If adverse features are not present
o Observe the patient unless there is a risk of asystole [recent asystole, Mobitz type II, complete heart block with broad QRS, ventricular pauses >3 s], in which case treat as if there were adverse features present.

Further management of bradyarrhythmias:
- Request 12 lead ECG
- Identify and correct any underlying cause if not already done so
- Arrange for permanent pacemaker (PPM) if indicated