

conduction disturbances
(created by Paul Young 13/10/07)

sinus node abnormalities

- sinus bradycardia:
- can be divided into appropriate and inappropriate pathological bradycardia
 - pathological bradycardia does not increase appropriately with exercise
 - medications are the most common cause of inappropriate sinus bradycardia; other causes include autonomic influences, electrolyte abnormalities, increased vagal tone (eg due to ischaemia) and intrinsic structural disorders

sinus arrest:

- sinus arrest occurs when the pacemaker cells in the SA node fail to depolarise
- pauses of less than 3 seconds may be normal; however, pauses of longer than 3 seconds are usually pathological and should be evaluated

carotid sinus hypersensitivity:

- diagnosed when ventricular asystole greater than 3 seconds of a drop in systolic BP >50mmHg occurs in response to carotid sinus massage
- treatment is permanent pacing in symptomatic individuals

post-surgical bradydysrhythmia:

- bradyarrhythmias are common after cardiac surgery
- valve surgery and septal myectomy can cause significant damage to the conduction system; prolonged ischaemia during heart transplant can also cause sinus node or conduction system damage
- medication and reversible ischaemia are often implicated therefore the decision to place a permanent pacemaker should be deferred until 5-7 days post-operatively
- permanent pacing is required in 2% of patients after valve surgery & approximately 10% of heart transplant recipients

1st degree AV block:

- characterised by a PR interval of greater than 0.2s in adults and 0.18s in children
- potential causes of 1st degree AVB include:
 - (i) delayed conduction through the atria from the SA to the AV node
 - typically due to structural causes such as right atrial enlargement or ASD
 - (ii) delay in AV nodal conduction
 - most common cause of 1st degree AVB
 - (iii) prolonged infranodal conduction
 - typically associated with a wide QRS due to disease in the fascicles or bundle of His
 - typically benign and seen in 0.5% of young adults without heart disease; in older patients is most commonly due to idiopathic degenerative disease

2nd degree block type I:

- Mobitz type I or Wenkebach rhythm is defined by progressive PR interval prolongation followed by a dropped beat
- caused by a block in AV conduction
- a stable rhythm
- if due to a medication it will resolve on discontinuation of the medication

2nd degree block type II:

- 2nd degree block type II (or Mobitz type II) is characterised by a sudden non-conducted beat
- this is an inherently unstable & may be associated with serious pathology
- almost always due to infranodal conduction disturbance
- is described as a high degree AV block with P to QRS ratios of 3:1 to 4:1 & can progress to complete heart block

2:1 AVB

- can be caused by Mobitz type I (usually narrow QRS) or Mobitz type II (usually wide QRS)

3rd degree AVB:

- characterised by complete AV dissociation
- ventricular complexes may be junctional (narrow QRS complex at 40-60) or ventricular (wide QRS at <40)
- congenital 3rd degree AVB is more common in females and often has an escape rhythm with an adequate rate
- acquired 3rd degree AVB usually requires permanent pacing and occurs most often in males. Specific causes include medication, ischaemia, progression from Mobitz type II rhythm and infarction. Third degree heart block complicating inferior MI is usually temporary and can be treated with temporary pacing whereas when it complicates anterior infarction it usually requires a permanent pacemaker

- medical therapy for AV block consists of atropine & adrenergic agents
- atropine decreases vagal tone & is useful in inferior myocardial infarction than anterior myocardial infarction (because it is not helpful for AV node ischaemia)
- atropine will not improve 3rd degree heart block or Mobitz type II block if the pathology is below the AV node & it is not effective in heart transplant patients
- digibind should be used in symptomatic patients with digoxin-induced AV block.

Therapy

normal cardiac conduction

- the electrical impulse leading to cardiac contraction is generated by specialised groups of cells with the ability to depolarise spontaneously
- the SA node is located in the posterior & superior portion of right atrium & its depolarisation is not visible on an ECG
- the p wave is generated by spread of the electrical impulse through the atria
- the impulse reaches the AV node approximately half way through the p wave with remainder of the p wave due to left atrial depolarisation
- in the normal heart the atria and ventricles are electrically isolated except at the AV node which is located in the atrial septum
- conduction through the AV node accounts for the majority of the PR interval
- from the AV node the impulse travels through the bundle of His and from there travels down the right and left bundle branches and their fascicles to the Purkinje network which causes ventricular contraction

causes of conduction failure

- narrow QRS complexes suggest supranodal site of block
- wide QRS complexes suggest and infranodal site of block
- AV nodal blocks are usually reversible except where infarction permanently damages a portion of the conduction pathway
- Causes of AV blocks include:
 - (i) drugs:
 - digoxin
 - beta blockers
 - calcium channel blockers
 - (ii) primary cardiac disease
 - ischaemic heart disease
 - fibrosis of the conduction system
 - congenital heart disease
 - calcific valvular heart disease
 - cardiomyopathy
 - (iii) metabolic
 - hyperkalaemia
 - hypermagnesaemia
 - (iv) infiltrative disease
 - (v) infectious / inflammatory disease
 - (vi) collagen vascular disease
 - (vii) Addison's disease
 - (viii) radiation
 - (ix) neurally mediated conditions
 - carotid sinus syndrome
 - vasovagal syncope
 - neuromyopathic disorders
- infranodal blocks are usually caused by structural heart disease & anatomical disruptions of the conduction system
- rare causes of infranodal block include disruption of the Bundle of His from aortic valve calcification & Chagas disease

clinical presentation

- syncope and presyncope are the most dramatic symptoms of conduction disturbances; palpitations, dyspnoea, angina & fatigue are also seen
- a significant number of patients develop bradydysarrhythmias after an AMI