Atrioventricular Conduction Disturbances

Morning Report
7/25/03
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Etiology

- Fibrosis and sclerosis of the cardiac conduction system (50%)
  - Lenegre’s disease: progressive sclerosis of the cardiac conduction system in young patients
  - Lev’s disease: occurs in older people, progressive fibrosis of the left side of the cardiac skeleton, also involving the mitral and aortic rings
Etiology

- Ischemia (40%)
  - Up to 20% of AMI develop AV block
  - Inferior MI: RCA supplies inf-post. Walls, AVN (90%) and SA (50%)
  - Mobitz I usually transient because the Left heart supplies collaterals
    - Complete infarction associated with increased mortality presumed due to larger infarct size.
Etiology

- Drugs: Digoxin, CCBs (Verapamil>Diltiazem), Amiodarone, Beta Blockers
- Familial AV Conduction Block
  - AD, associated with mutation in Na channel
- Increased Vagal Tone (Mobitz I): Pain, Carotid Sinus Massage/hypersensitivity, Well-trained athletes
- Congenital heart disease
- Valvular Heart disease, also associated with valvular replacement or repair.
Etiology

- HOCM
- Amyloid
- Sarcoid
- Infection: diphtheria, virus/coxsackie, toxoplasmosis, endocarditis, syphilis, Chagas
- Hyperkalemia
- Hodgkins, lymphoma, myeloma
- Rheum: SLE, dermatomyositis, RA
- Endo: hyper/hypothyroid, Paget’s
First Degree Heart Block

- PR >200ms
- No symptoms except below:
- PseudoPacemaker syndrome:
  - Most commonly occurs with marked first degree block or Mobitz II with high ratios, i.e., greater than 5:4
  - Pacemaker syndrome classically occurs with ventricular pacing, in which the atria contract against a closed valve.
  - Mechanism is atrial conduction in close proximity to preceding ventricular systole, resulting in incomplete atrial filling. This results in compromised ventricular filling, increased PCWP, and decreased CO.
Second Degree Heart Block

- **Mobitz Type I (Wenckebach):** progressive PR prolongation precedes a non-conducted p wave. Involves the AV node and less likely to progress.
- **Mobitz Type II:** constant PR interval prior to failed conduction of p wave. Usually below the AV node and can progress
- Can not distinguish the two in 2:1 blockade. Must obtain a long rhythm strip or use atropine to induce 3:2
- **Advanced second degree heart block:** block of 2 consecutive p waves
Wenckebach

- Does not produce symptoms unless the sinus rate is slow or pseudopacemaker syndrome
- 70-75% occurs in AVN, especially the classic pattern
- 25-30% His bundle, bundle branches or fascicles
Wenckebach

- Classic Pattern: most commonly in the AVN
  - Usually has ratios of 3:2, 4:3 or 5:4
  - The absolute increase in PR interval becomes smaller with each successive beat in a cycle.
  - R-R interval becomes smaller throughout the cycle.
  - Occasionally there is an escape beat between cycles

- Atypical Pattern: more likely to be distal block
  - Ratio exceeds 6:5. With larger ratios, the increase in PR becomes logarithmic and the PR becomes constant.
  - The PR is still shortest the first beat of the cycle and longest the last beat.
Mobitz II

- Block almost always occurs below the AVN; bundle of his (20%), bundle branch or fascicles in others.
- 2/3 also have bifascicular or trifascicular block
- PR may be normal or slightly prolonged
- Increase in HR can worsen block, allowing less time for the bundle of his to recover (implications for using atropine)
Third Degree Heart Block

- No atrial impulse is propagated to the ventricle
- Symptoms: syncope (Stokes-Adams attacks), presyncope, heart failure, angina
- May develop VT/VF
- Escape rhythm provides important clues to site of block
Clues to Site of block in AV conduction disturbances

- **EKG**: can not determine site reliably
- **The His bundle EKG**
  - Obtained during EPS
  - A=atrial depol., H=his depol., V=ventricular depol., AH=AVN conduction time, HV=His-Purkinje conduction time
  - AH becomes progressively long if blockage is in the AVN
  - If block is in the bundle of His or lower, there is often an H’
    - H-H’ interval becomes progressively longer in Mobitz I
    - H-H’, AH, VH all constant, with sudden drop in V in Mobitz II
Clues to Site of block in AV conduction disturbances

- **AVN**
  - Mobitz I (70%) especially classic pattern
  - PR >240
  - Junctional escape (narrow QRS), faster rate
  - HR responds to atropine/exercise, vagal
- **Bundle Branch/fascicle**
  - Concomitant BBB
  - Slower escape rhythm with wide QRS
  - 80% of Mobitz II
  - No response to Atropine
Indications for Pacer

■ Class I
  ■ Symptomatic bradycardia, CHF
  ■ Medical conditions which require use of AVN blocking agents
  ■ Asystole of at least 3 seconds
  ■ Escape rate of <40 BPM
  ■ Neuromuscular disease
  ■ Bifascicular or trifascicular block and Mobitz II
Indications for Pacer

- **Class II**
  - Asymptomatic Mobitz II
  - Asymptomatic Mobitz I with block at His or distally
  - Asymptomatic third degree heart block
  - Pseudopacemaker syndrome

- **Class III**
  - First degree AVB
  - Asymptomatic Mobitz I with block at supra-his level
# Pacing Code

<table>
<thead>
<tr>
<th>I. Chamber paced</th>
<th>II. Chamber sensed</th>
<th>III. Response to sensing</th>
<th>IV. Programmability, Rate Modulation</th>
<th>V. Antitachyarrhythmia Functions</th>
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<td>I, Inhibited</td>
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<td>R, Rate modulation</td>
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Pacers

- VVI (ventricular sensed, paced and inhibited) used in past but lose synchrony between atrial and ventricular contraction
- DDD (senses and paces both atria and ventricles) used most often, especially for physically active or those with borderline hemodynamic reserve.
- DDDR (automatically adjust ventricular pacing rate to sensed indicator of exertion) required in SA node dysfunction or afib.
- VAT (atrial sensed, ventricular paced) or VDD if SA node normal