

Heart Block

Sept 2014

1st degree heart block

Causes

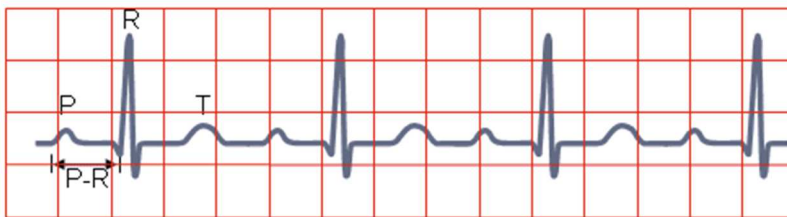
Beta-blockers, Ca channel antagonists, digoxin, inf MI, incr vagal tone, AV disease, myocarditis, RF

ECG

PR >200

Management

No trt needed; stop contributing agents; correct electrolyte abnormalities (eg. hyperK)



2nd degree HB

Mobitz I/Wenckebach

Causes

Inf MI, digoxin, incr vagal tone, myocarditis

5% normal subjects during sleep

Pathophysiology

Above bundle of His, in/above AVN

ECG

Progressive prolongation of PR until drops one; RR pattern shows grouped beating

Management

Will respond to atropine; rarely requires treatment



Mobitz II

Pathophysiology

25% in BOH (QRS width normal, but may be BBB - wide QRS); in/below AVN

ECG

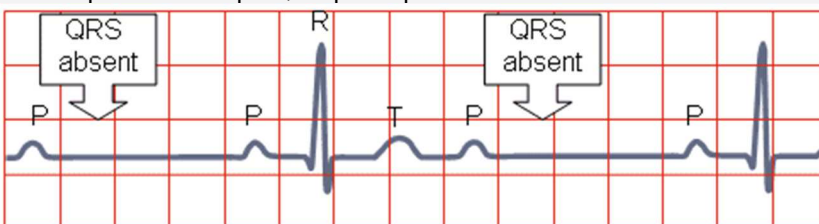
Regular atrial rhythm; no PR prolongation prior to dropped beat

Complications

CVA, Stokes-Adams attack, sudden cardiac death

Management

60% respond to atropine; requires pacemaker insertion



3rd degree heart block

Causes

Degenerative (most common); inf MI (usually nodal, will usually resolve spontaneously), ant MI (usually infranodal, worse prognosis), myocardial fibrosis

OE

Canon A waves on JVP (AF has lack of a waves)

ECG

Atrial rate > ventricular rate; no relationship between P and QRS; regular rhythm of QRS; may masquerade as 2:1 in short recordings when A rate is 2x V rate

Escape rhythm

Morphology/rate of QRS depends of origin of escape rhythm; escape rhythm should originate from just distal to site of block (40-60bpm = nodal, QRS normal, better prognosis = junctional; <40bpm = infranodal, QRS wide, unstable = ventricular); likely deterioration if slow QRS rates, broad QRS

Management

Atropine 300 – 600mcg IV to max 3mg; rarely effective unless incr vagal tone; less success if infranodal block (ie. Mobitz II, CHB); caution if MI as incr HR - incr infarct

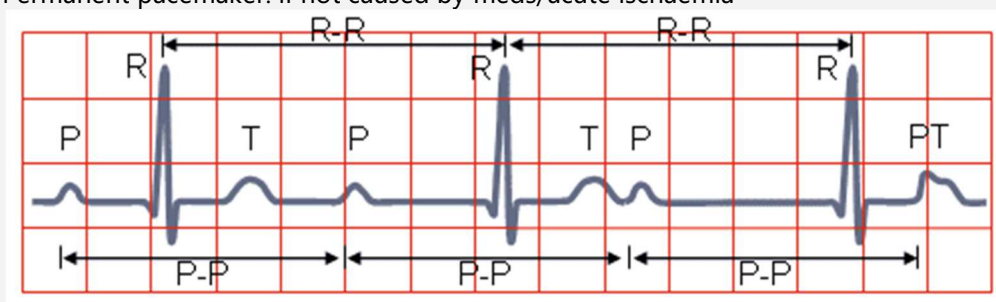
Isoprenaline 20-40mcg IV bolus - 0.5-20mcg/min (same dose as adrenaline); CI in digitoxicity

Fist pacing: if nothing else available; at 60bpm

Transcutaneous pacing: starting at 100mA and titrating up

Transvenous pacing: if resistant to isoprenaline, failure of transcutaneous, high risk of progression

Permanent pacemaker: if not caused by meds/acute ischaemia



RBBB

rSR pattern in right precordial leads (V1-2)

OR: single wide R wave, qR pattern

S wave on lateral leads (I, aVL, V5 V6) is slightly wide

QRS > 0.12 secs

If QRS < 0.12 secs and all other criteria met = incomplete RBBB

LBBB

QRS > 0.12 secs

LAD

Broad monophasic R wave in I and V6

Deep wide S wave in V1 (often no R wave)

ST segments/T waves in opposite direction to main QRS vector ("appropriate discordance")

LAFB

LAD

qR complex (small q large R) or R wave in I and aVL

rS complex (small r large S) in III

Absence of other causes of LAD

LAD causes: LAFB, LBBB, Inferior AMI, LVH, Ventricular ectopy, Paced beats, WPW



LPFB

Less common than LAFB

Usually occurs with RBBB

RBBB + Fascicular block = "Bifascicular block"

RAD

qR (small q large R) in III

Absence of other causes of RAD

RAD causes:

LPFB, Lateral AMI, RVH

Acute lung disease: PE, Chronic lung disease: COAD

Ventricular ectopics, Hyperkalaemia, OD of Na⁺ blockers (eg TCA)

Young slim people – heart more horizontal

Trifascicular Block

= Complete Right Bundle Branch Block

+ Left Anterior Hemiblock

+ Long PR interval