Arrhythmias

Definition:

Deviation from regular sinus rhythm of 60-100bpm. May be one or more of: irregular, faster (tachyarrhythmias), slower (bradyarrhythmias), or not generated from SA node (ectopic).

Conduction system

- SA Node:
  - Junction of RA & SVC. Linked to AV node by atrial internodal pathways
  - Supplied by RCA in 55-60%, otherwise L circumflex
  - Innervated by symp & parasymp.
  - Native discharge rate is 90-100bpm (↓ by parasymp tone to ~70-80bpm)
- AV Node
  - RA myocardium nr septal leaflet of TV in otherwise non-conducting annular fibrosis
  - Supplied by RCA in 90%, 10% by LC circumflex
  - Slower conduction 0.05m/s to allow for atrial contraction
- Remainder of conduction system
  - Bundle of His runs in membranous septum
  - Splits into right & left bundles. The latter splits into ant. & post. Fascicles.
  - Right bundle & L ant. Fascicle supplied by AV nodal art. in 50% & LAD in rest.
  - Left post. fasc supplied by AV nodal only in 50% & by both AV nodal & LAD in rest.
  - Bundles/fascicles are composed of fast conducting Purkinje fibres.
  - Embryonic myocardium remnants around the AV node can → accessory pathways.

Arrhythmia Mechanisms

- Tachyarrhythmias
  - Re-entry circuits – unidirectional, can be micro (AF, VF) or macro (PSVT)
  - Enhanced automaticity – ectopic foci
  - After polarizations – oscillations of mem pot → another depolarization. E.g. TdP
- Bradyarrhythmias
  - Depression of SA node
  - Conduction system block

Causes

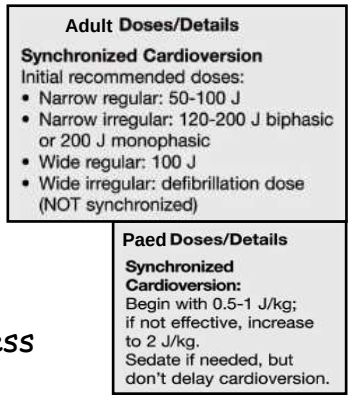
- IHD – most common of serious acute arrhythmias
- CHD – valvular, myocardial defects, HOCM, prolonged QT syndromes
- Structural – cardiomyopathies, acq. Valve defects, HT
- Electrolyte disturbance – high or low K+, low Mg2+ or Ca2+
- Metabolic – hypoxia, hypercarbia, acidosis, hypothermia, hyperthyroidism, phaeo.
- Drugs – antiarrhythmics, cocaine, amphetamines, TCA, Na+ or K+ channel blockers
- Trauma – commotio cordis – VF from blunt chest trauma.

Principles of management

- Triaged to an area where they can be monitored and sequelae of arrhythmia managed.
- Resuscitate/ABCs - ?arrest, shock, APO, ↓GCS. If unstable, cardioversion/pacing likely.
- IVC + bloods for UEC, CMP, FBC ± cardiac biomarkers ± TFTs ± drug levels (e.g. digoxin)
- ECG (classify & diagnose arrhythmia) ± CXR
- Rx aimed at restoring adequate cerebral perfusion, a reg. rhythm & treat precip conds.

Management Options

- No Rx:
  - If no resus required.
  - May be approp: sinus arrhythmia, atrial ectopics, non R-on-T ventricular ectopics, accel idioventricular rhythm, AF in assoc with hypothermia, Mobitz type I 2° block.
- Non-Pharmacological
  - Vagal manoeuvres – Valsalva, carotid sinus massage, diving reflex. Avoid ocular massage, PR.
  - Precordial thump – immediate response to monitored VF/pulseless VT
- Pharmacological
  - Electrolyte correction
  - Usual first line if haemodynamically stable and arrhythmia needs treatment.
- Electrical
  - Cardioversion
    - Haemodynamically unstable or drug-resistant tachyarrhythmias.
  - Pacing
    - Transcutaneous, transvenous, overdrive
    - Drug-resistant brady & tachyarrhythmias.



Vaughan-Williams Classification of antiarrhythmics – limited usefulness

- Class I (Fast Na+ channel blockers)
  - Class Ia (slow conduction, ↑QRS & ↑QT)
    - Procainamide: IV 17mg/kg (12mg/kg if APO) load then 2.8mg/kg/hr (halve if APO). SE: ↓BP, ↓HR, lupus-like syndrome
    - Disopyramide: PO. Slow Abs. 1/3 Metab liver. Renal excr. Neg. ionotrope.
    - Quinidine: Rare in Aus. 200-400mg q2h PO to max 1g or 600-800mg PO stat. SE: cinchonism (tinnitus, vis.dist., headache, vertigo, confusion)
  - Class Ib (prolong refractory period)
    - Lignocaine: 1.5mg/kg+bol. max 3mg/kg. Maint 2mg/min IV. SE: ↓BP,CNS tox
    - Phenytoin
  - Class Ic (depress conductivity, ↑QRS, ↑PR, ↑AV block)
    - Flecainide: 2mg/kg over 20min. SE: proarrhythmic in structural/IHD
- Class II (β-blockers) e.g. atenolol, metoprolol, esmolol, sotalol
- Class III (prolong repol. by K^+ efflux, ↑QRS, ↑PR & ↑QT)
  - Sotalol: 0.5-1.5mg/kg IV/PO. CI: COAD, DM, PVD, LVF, hypoK+, bradycardic
  - Amiodarone (also class I, II & IV): 2-5mg/kg over 5min (resus) to 1hr in 5%Dex. (via CVC if poss.) Maint: 15mg/kg in 500ml 5%dex over 24h. Ok in LVF. Long $T\frac{1}{2}$. SE: Proarrhythmic. Long term (vis.dist, photosens, grey skin, abn. LFT/TFT, pulm fibrosis, tremors), unpred effect with warfarin, ↑dig,phenytoin,theophylline levels
- Class IV (calcium channel blockers, prolong AV node cond, ↑PR) e.g. verapamil

Brugada Syndrome (See ECG Article for more details)

Cause of sudden cardiac death & 60% of idiopathic VF. Aut.Dom defect in Na^+ channel. More common in Asian males. Ave. age at Dx=30y. ECG features (may be transient) partial RBBB, downsloping ST elevation & ↑or↓T in V1-3. In addition syndrome reqs also one of: syncope, VF, polymorphic VT, FamHx of sudden death<45y, or ST elevation in current family members.

Bradyarrhythmias

Definitions

- Strictly: <60bpm in adults, age dependent in children
- Relative: too slow for haemodynamic state of patient (may be >60bpm)
- Features: If symptoms, may include light-headedness, palpitations, syncope

Types

- Sinus bradycardia
  - Causes:
    - High resting vagal tone e.g. athletes, young adults
    - Patients on negative chronotropes
      - β blockers, CCB, digoxin, amiodarone, theophylline, clonidine
    - Hypothyroidism
    - Hypothermia
    - Cushing reflex (↑ICP)
    - Bezold-Jarish reflex - parasympathetic stimulation in early inferior MI
    - Pericardial tamponade
    - Adrenal insufficiency
    - Dive reflex in young children (vagal)
    - Severe jaundice
    - Pleural/peritoneal stimulation
    - Rarely carotid hypersensitivity (tight shirt collar) or infection (e.g. typhoid - relative bradycardia)
  - Management:
    - Rule out and treat above causes or myocardial ischaemia
    - If symptomatic (often not until HR<40) treat as below
- 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, sinus exit block or sinus arrest
  - SA node fails to produce an impulse or not conducted to atria
  - ECG typically shows absent P waves with escape rhythm
    - Junctional - narrow complexes @ 40-60bpm, may be accelerated to 60-100
    - (Idio)Ventricular - wide complexes @ 30-40bpm
  - Usual causes: Ischaemia, hyperK+, vagal tone, negative chronotopes (see above)
  - Treat if symptomatic
- Sick sinus syndrome aka tachy-brady syndrome
  - Causes: SA/AV nodal fibrosis, ischaemia, CHD, tumours, surgery, cardiomyopathy
  - ECG may intermittently show SA block, sinus bradycardia or arrest with bursts of atrial tachycardia (usually AF, but also junctional tachycardia, SVT, atrial flutter)
  - Management:
    - Treat any symptomatic acute arrhythmia
    - Most require permanent pacemaker for the bradycardia component & an antiarrhythmic (e.g. digoxin or verapamil) to suppress tachycardias.

- AV blocks
  - First degree
    - Slow AV node conduction
    - ECG: PR>200ms
    - All atrial impulses conducted to ventricles
    - Benign, but may be assoc with vagal tone, inf MI, digoxin tox, myocarditis
  - 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
      - Usually asymptomatic and treatment not required
      - Treat if symptomatic or in context of inferior MI
    - Mobitz Type II
      - Conduction defect generally below AV node
      - Degenerative Lev or Lenegre disease
      - ECG: constant PR with intermittent QRS absence
      - Risk of progression to third degree block
      - Atropine often ineffective as block usually below AV node
      - Permanent pacing is usually required
    - 2:1 block
      - 2 P waves per QRS complex. Types I & II indistinguishable on ECG.
      - 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 & QRS junctional/escape complexes occur independently
    - Treat as below - though atropine is unlikely to be effective(unless a nodal block) and a permanent pacemaker will be needed
    - Myocardial fibrosis is commonest cause
    - Associations:
      - Inferior AMI
      - Sick sinus syndrome
      - Mobitz type II
      - Second degree block plus new bundle branch or fascicular block
  - With all AV blocks additionally rule out
    - Negative chronotropes (as for sinus bradycardia)
    - Lyme disease
    - Myocarditis/endocarditis
    - SLE
    - Chagas disease
    - Myxoedema,
    - Amyloid
    - Cardiac surgery/tumours

Bradyarrhythmias

General Management

- ABC, O_2
- IV access
- Bloods: UEC, CMP, Troponin/CK, FBC, TFT or digoxin level as appropriate
- ECG
- Treat any underlying cause (cease negative chronotrope, correct electrolytes, etc.)
    - Treat bradycardia only if HR<50 and hypoperfusion (syncope, sysBP <90mmHg, HF)
- Drugs:
    - Atropine: 0.5-1.0mg (0.02mg/kg, minimum of 0.1mg in child) repeated up to 2-3mg
        - Only useful if increase vagal tone i.e. problem at/above AV node.
    - Isoprenaline (isoproterenol): bolus 20-40mcg IV, infusion 0.5mcg/min of 2mg/100ml N.Saline
        - Caution: As a pure beta agonist can cause beta2 vasodilatation in muscle beds leading to hypotension
    - Adrenaline: infusion 2-10mcg/min
        - Useful if hypotension an issue. Ideally needs a central line.
    - Others:
        - Dopamine: 5-20mcg/kg/min through central line.
        - Aminophylline
        - Glucagon (beta blocker/calcium channel OD)
        - Digoxin Fab (digoxin toxicity)
- Pacing:
    - Temporary if drug therapy fails (likely with Mobitz II 2nd & 3rd degree AV blocks)
        - Transcutaneous
            - Via fist (!) or defibrillator pacing function
            - Will normally require analgesia/sedation
            - Temporising measure before:
        - Transvenous wires inserted
    - Permanent pacemaker

Disposition

- Admit to monitored bed if:
    - Symptomatic
    - Sick sinus
    - Mobitz II second degree block
    - Third degree block

Tachyarrhythmias

Common symptoms: rapid palpitations, chest discomfort, dyspnoea.

Types

- Narrow complex (QRS ≤ 120ms, origin atrial or nodal)
  - Regular
    - *Sinus tachycardia*
      - Rates 100 to ~180.
      - HR variability is maintained unlike SVT.
      - Causes:
        - physiological - exercise, anxiety
        - pharmacological - stimulant use, anticholinergics, β-agonists
        - pathological - ↑T, ↓Hb, PE, hypoxia, hypovolaemia, myocarditis
    - *Supraventricular tachycardia* – see separate article
    - *Atrial flutter* – rare in absence of heart disease
      - Atrial rate ~300, ±2:1 or more AV block. Sawtooth waves: Inf+V1.
      - Causes: IHD (2% of MI), CCF, PE, myocarditis, chest trauma, dig toxic
      - Rx underlying cond. If unstable DC 25-50J or atrial overdrive pacing (>400bpm). Rate control as for AF. Reversion (amiodarone, flecainide)
    - *Atrial (ectopic) tachycardia* – <10% of SVT. Dig tox. Rx as for AF.
    - *Junctional tachycardia* – causes e.g. dig toxic, inf MI, RhF
  - Irregular
    - *Atrial fibrillation* – see separate article
    - *Atrial flutter with variable block*
    - *Multifocal atrial tachycardia*
      - 3^+ atrial foci (diff p wave shapes), HR>100, variable PP, PR, RR ints
      - Causes: Sev COAD, dig/theophylline tox, large PE, ↓↓O_2, DM, sepsis
      - Mx: Rx underlying cond, electrolytes, Mg^{2+}, metoprolol (?CI in COAD)
- Broad complex tachycardias (QRS>120ms, ventricular origin or atrial+aberrant cond.)
  - Regular
    - *Ventricular tachycardia* – see separate article
    - *Antidromic AVRT* – see SVT article
    - *Narrow complex tachycardia (ST, SVT, A. flutter) with aberrant conduction*
  - Irregular
    - *Torsade de pointes* – see VT article
    - *AF with aberrant conduction*
    - *WPW + AF* – see SVT article
    - *VF*
- Others
  - *Accelerated junctional tachycardia*
    - Regular narrow complex AV nodal escape rhythm @ 60-100bpm.
  - *Accelerated idioventricular tachycardia*
    - Regular broad complex ectopic rhythm @ 40-110bpm.
    - Highly specific for myocardial disease e.g. AMI
    - Only rarely causes haem. instability.
    - Occ atropine used to increase sinus rate & suppress ectopy.