mechanisms of arrhythmia

- blocks and bradys are caused by impaired automaticity or conduction - if one pacemaker fails another generally takes over at a lower rate
- factors that impair pacemaker automaticity or myocardial impulse conduction:
  1. hypoxia
  2. drugs (eg beta blockers)
  3. electrolyte & pH disturbances
  4. myocardial ischaemia
  5. anything that enhances parasympathetic tone (eg carotid sinus hypersensitivity)

1. fast or slow?
2. ventricular or supraventricular?
3. compromised or not?
4. does arrhythmia need management?
5. what is underlying substrate predisposing?
6. what is trigger?
7. will arrhythmia recur?

- factors contributing to arrhythmogenenesis
  - Structural influences:
    (i) myocardial infarction - acute, healed, aneurysm
    (ii) hypertrophy
    (iii) myopathic ventricle - dilation, fibrosis
  - Transient influences:
    (i) transient ischaemia / reperfusion
    (ii) systemic factors
      - hypoxia
      - acidosis
      - electrolyte abnormalities
    (iii) neurophysiological factors
      - autonomic tone
      - endogenous catecholamines
    (iv) toxicity
      - proarrhythmic drugs
      - exogenous catecholamines

Vaughan-Williams classification of anti-arrhythmics

class I - sodium channel blockade
class II - beta adrenergic blockade
class III - prolongation of repolarisation often due to potassium channel blockade
class IV - calcium channel blockade

factors facilitating anti-arrhythmic proarrrhythmia

factors contributing to arrhythmogenesis

increased automaticity

- key concept is "afterdepolarisation" where after a normal action potential, the cellular transmembrane potential suddenly swings positive again, if the upswing is sufficient, a full depolibration may occur again & again
- there are (at least) two different mechanisms of triggered activity and these result in:
  1. early afterdepolarisations (EADs)
  2. delayed afterdepolarisations (DADs)

triggered activity

ventricular or supraventricular?