

Arrhythmia

- **Definition:**

- Disturbances in the heart rate, rhythm, impulse generation or conduction of electrical impulses responsible for membrane depolarization
- These disturbances can lead to alterations in overall cardiac function that can be life threatening.

- **Antiarrhythmic drugs:**

- Compounds used to prevent or treat cardiac arrhythmias



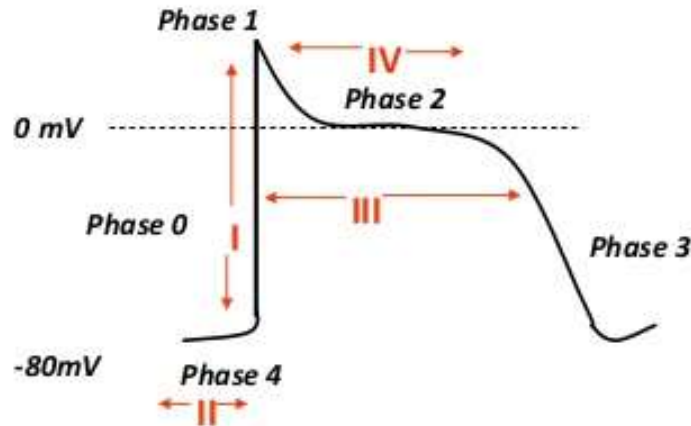


Mechanism of arrhythmias

- **Disturbances in impulse generation** may be due to
 - Abnormal automaticity
 - Delayed after depolarizations
- **Disturbances of impulse conduction**
 - The impulse may recirculate in heart causing repeated activation (re-entry)
 - Conduction blocks

Phases of action potential of cardiac cells

- **Phase 0** rapid depolarisation (inflow of Na^+)
- **Phase 1** partial repolarisation (inward Na^+ current deactivated, outflow of K^+)
- **Phase 2** plateau (slow inward calcium current)
- **Phase 3** repolarisation (calcium current inactivates, K^+ outflow)
- **Phase 4** pacemaker potential (Slow Na^+ inflow, slowing of K^+ outflow) 'autorhythmicity'
- **Refractory period** (phases 1-3)



Class I: Sodium channel blockers

- **IA: Prolong repolarization**
 - Quinidine, procainamide, disopyramide, morcizine
- **IB: Shorten repolarization**
 - Lignocaine, mexiletine, phenytoin
- **1C: Little effect on repolarization**
 - Encainide, flecainide, propafenone



Quinidine

- **D- isomer of quinine** obtained from cinchona bark
- **MOA:** blocks sodium channels
 - ↓ automaticity , conduction velocity and prolongs repolarization
 - ↓ phase 0 depolarization , ↑ APD & ↑ ERP
- **Other actions:**
 - ↓ BP (α block), skeletal muscle relaxation
- **Uses:** Atrial and ventricular arrhythmias
- **Adverse effects:**
 - Arrhythmias and heart block , hypotension, QT prolongation
 - GIT , thrombocytopenia, hepatitis , idiosyncratic reactions
 - High doses – cinchonism like quinine