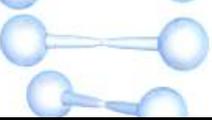


# Arrhythmia

- **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



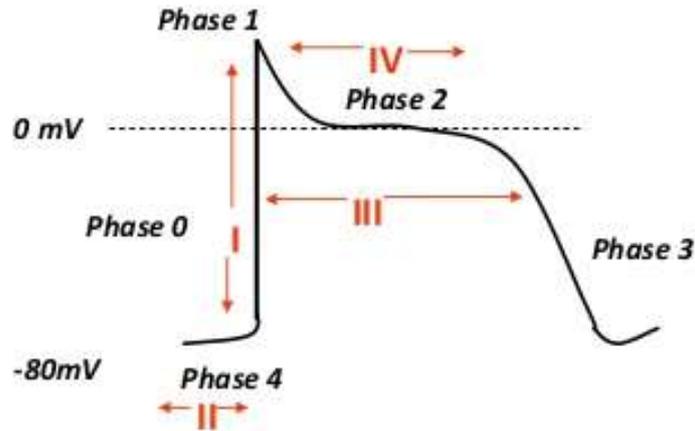


# Mechanism of arrhythmias

- **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

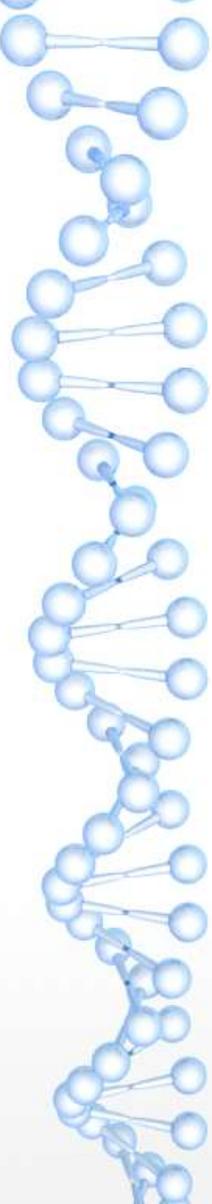
# Phases of action potential of cardiac cells

- **Phase 0** rapid depolarisation (inflow of  $\text{Na}^+$ )
- **Phase 1** partial repolarisation (inward  $\text{Na}^+$  current deactivated, outflow of  $\text{K}^+$ )
- **Phase 2** plateau (slow inward calcium current)
- **Phase 3** repolarisation (calcium current inactivates,  $\text{K}^+$  outflow)
- **Phase 4** pacemaker potential (Slow  $\text{Na}^+$  inflow, slowing of  $\text{K}^+$  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 ( $\alpha$  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