

ANTI-ARRHYTHMIC DRUGS:

Cardiac Arrhythmias:

- 25% of death with digitalis
- 50% Arrhythmia patients
- 80% patient with Atrial HT
- lead to D.C.D
- It's using drugs, surgery, pacemaker.

⇒ Tonic basis of membrane electric Activity?

- Transmembrane potential of cardiac cells is determined by the concentration of the following ions:

- Sodium, potassium, calcium.
- The more

CARDIAC ACTION POTENTIAL:

The phases of cardiac Action Potential:

Phase 0:

AP depolarize cells to threshold $-70mV$.

Phase 1:

Phase 3 - Repolarization

Rapid depolarization.

- Caused by a transient opening of Na^+ channels.
- Decrease caused direct depolarizing Na^+ currents.
- Generate fast response (AP₀)
- Plateau phase prolongs AP duration as AP is never at skeletal muscle.

Refractory Period

- ⇒ Once a AP is initiated there is a period that new AP can not be initiated
- ⇒ effective or absolute refractory period
- ⇒ stimulation of cell by adjacent cell depolarization cell
- ⇒ Prevent reinitiated by AP from occurring if wave front of depolarization of H.R.

Arrhythmia → Absence of Rhythms.

1) Result form:

- 1) disturbance in impulse formation
- Disturbance in impulse conduction
- Block Result form severely depressed conduction
- Re-entry (circus movement) / delayed impulse

NOA in cardiac Arrhythmias:

Result from disorder of impulse formation & conduction and

causes of Arrhythmias:

- cardiac ischemia
- excessive discharge
- exposure to toxic substance
- electrolyte disturbance
- Re-entrant tachycardia

Right Bundle Branch Block (RBBB)

→ Right ventricle gets a delayed impulse.

Depolarization spreads from the left

→ The tissue should be deflected after the PRS complex.

This is taken as asynchronous wave character with bundle branch block.

→ Parameter of dysmorphic cells.

Left Bundle Branch Block (LBBB)

Disorder of impulse conduction:

Many Results in Bundle branch block, Prolonged QT, eg. EXOTIC (1805)