Electrical Activity of Cardiac Nerve & Muscle Cells

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Two components of Electrical Activity in the Heart

- Electrical activity of individual nerves and muscle cells
  - Recorded with an intracellular electrode
  - Seen as cell membrane potentials

- Electrical activity of the whole heart
  - Recorded with electrodes on the chest and limbs
  - Seen as the electrocardiogram (ECG)

Resting Membrane Potential results from:

1. Unequal distribution of electrolytes across the cell wall

2. Movement of electrolytes along their concentration (chemical) and electrical gradients
Ventricular Muscle Action Potential – Ionic Basis

Phase 2 $\leftrightarrow$ Ca$^{2+}$ influx (L Channels) $\equiv$ K$^+$ efflux

Phases 3 & 4:
Driving force on K$^+$ decreases whilst that on Na$^+$ increases $\rightarrow$ membrane repolarization.

Phase 0 = 2 ms
Phases 2+3 = 200 ms
At a heart rate of 75 bpm, whole AP = 800 ms

Properties of the Inward Rectifier Potassium channel

• A channel that is "inwardly-rectifying" is one that passes current (positive charge) more easily in the inward direction (into the cell)

• K$^+$ efflux is small or absent toward peak of AP of cardiac muscle

• Allows K$^+$ influx at membrane potentials of -70 to -100 and beyond, which:
  • Opposes effect of Delayed Rectifier & Transient Outward K$^+$ channels

NB: $+I_k$ => Outward Current (efflux)
$-I_k$ => Inward Current (influx)
SA & AV nodes Action Potential

- 0 $\leftrightarrow$ Ca++ influx (via L channels)

- 2 $\leftrightarrow$ K+ efflux (esp inc Ito) > Ca$^{2+}$ influx (L Channels)

- 3 $\leftrightarrow$ K+ efflux (via Delayed Rectifier; No K+ inward rectifier in nodes)

- 4 $\leftrightarrow$ Closure of K+ channels

Ionic basis of SA Node Prepotential

Prepotential has 2 phases:
- An early D1 Phase
- A later D2 Phase
Parasympathetic stimulation (by vagus nerve) increases hyperpolarization and decreases prepotential gradient → Decreased heart rate

Ach activates M₂ muscarinic receptors → G protein activation → opening of K⁺ channels ($I_{K\text{Ach}}$) to counter the K⁺ decay & $I_{Ca}$

Sympathetic stimulation decreases hyperpolarization & increases prepotential gradient → Increase in heart rate

NE activates β₁ receptors → Inc. cAMP → Inc. $I_{Ca}$ & $I_{Na}$
### Ventricular Muscle AP
- Greater RMP (i.e., more negative)
- Bigger potential size
- More rapid onset
- Obvious plateau in Phase 2

### Refractory Periods
- \( \leftarrow \) inactivated Na\(^+\) & Ca\(^++\) channels
  - prevent tetanic contraction of the heart

### Action Potentials of Cardiac Nerve & Muscle Cells have different speeds

<table>
<thead>
<tr>
<th>Conduction Speeds:</th>
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<tbody>
<tr>
<td>0.05 m/s</td>
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<tr>
<td>• SA node</td>
</tr>
<tr>
<td>• AV node</td>
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<tr>
<td>1 m/s</td>
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<tr>
<td>• Atrial pathways</td>
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<tr>
<td>• Bundle of His</td>
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<tr>
<td>• Ventricular muscle</td>
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<tr>
<td>4 m/s</td>
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<tr>
<td>• Purkinje system</td>
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<td>cp 60 m/s in median nerve</td>
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Clinical Correlates

- **Arrhythmias**
  - ectopic pacemaker e.g. atrial muscle, ventricular muscle or AV node
  - Block of normal conduction pathway → re-entry phenomenon

- **Rx: use drugs that block**
  1. Fast sodium channels (dec Phase 0 of AP)
  2. Beta adrenergic receptors (dec prepotential of slow AP)
  3. K+ channels (prolong depolarization and refractory period of cells)
  4. Ca2+ channels (both L- and T- types affect slow AP)