CARDIAC PHYSIOLOGY

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IONIC BASES OF TRANSMEMBRANE POTENTIALS

- The RMP is attributed mainly to the equilibrium potential of potassium. The RMP is affected more by potassium than by any other ion.
- Ionic Mechanisms in the different phases of the action potential.
  Cardiac tissues maybe classified as:
  - slow fiber
  - fast fiber
<table>
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<th>Characteristics</th>
<th>Examples</th>
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<td>Slow conduction velocity</td>
<td>SA node</td>
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<tr>
<td>Low RMP</td>
<td>AV node</td>
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<tr>
<td>Slow phase 0 depolarization</td>
<td>Fibers in AV ring</td>
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<td>Fast conduction velocity</td>
<td>Atrial and Ventricular muscle fibers</td>
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<td>Higher RMP</td>
<td>Purkinje fibers</td>
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<table>
<thead>
<tr>
<th>Phase of AP</th>
<th>SLOW fiber</th>
<th>FAST fiber</th>
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<tr>
<td>Phase 4</td>
<td>Due to decreasing permeability of K+</td>
<td>Due to decreasing permeability of K+</td>
</tr>
<tr>
<td></td>
<td>Due to inward current of Ca+</td>
<td></td>
</tr>
<tr>
<td>Phase 0</td>
<td>Due to inward Ca+ current</td>
<td>Mainly due to an inward Na+ current</td>
</tr>
<tr>
<td></td>
<td></td>
<td>To a lesser degree, due to inward Ca+ current</td>
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<tr>
<td>Phase 1</td>
<td>Not prominent</td>
<td>Due to inward current of Ca+</td>
</tr>
<tr>
<td>Phase 2</td>
<td>Due to continuing permeability of the membrane to Ca+</td>
<td>Due to continuing permeability of the membrane to Ca+</td>
</tr>
<tr>
<td>Phase 3</td>
<td>Due to efflux of K+ from the cell</td>
<td>Due to efflux of K+ from the cell</td>
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EFFECTS OF IONS ON TRANSMEMBRANE POTENTIALS

- Effects of ions on the RMP
  - Increase extracellular K⁺
    - Reduction in the RMP. This enhances the excitability because the resting potential becomes closer to the threshold or firing level. The intensity of the stimulus required to excite the cell becomes lower.
    - Further increase in K⁺ concentration results in loss of excitability probably due to the mechanism of inactivation.
  - Decreased extracellular K⁺
    - Paradoxically, also results in reduction in RMP. The depolarizing effect of decreasing extracellular K⁺ concentration is aided by simultaneously raising extracellular Ca++ concentration and counteracted by lowering the Ca++ concentration.

EFFECTS OF IONS ON TRANSMEMBRANE POTENTIALS

- Effects of ions on the action potential of non-automatic cells
  - Increased extracellular K⁺
    - Reduction in the velocity of phase 0 depolarization
    - Decreased amplitude
    - Decreased duration of the action potential
  - Decreased extracellular K⁺
    - Increased amplitude
    - Prolonged duration
    - Eventually results in supraventricular and ventricular ectopic beats, ectopic tachycardia and ventricular fibrillation
  - Decreased extracellular sodium
    - Decreased velocity of phase 0 depolarization
    - Decreased amplitude
    - Severe reduction leads to complete loss of excitability
  - Increased extracellular sodium
    - Little direct effect in action potential configuration
  - Increased extracellular Ca++
    - Ventricular action potential becomes prolonged
    - Inhibition of Ca++ influx causes the ventricular AP to become like that of the atrium
      - Ca++ has little effect on the membrane potential and the amplitude of the action potential.
EFFECTS OF IONS ON TRANSMEMBRANE POTENTIALS

- Effects of ions on the action potential of automatic cells
  - Decreased extracellular K+
    - Increased slope (steepness) of phase 4 depolarization probably due to decreased permeability to K+
    - Decreased in maximum diastolic potential
    - Reduction in threshold
  - Increased extracellular K+
    - Decreased slope of phase 4 depolarization probably because of increased permeability to K+ which then decreases
    - Decreased magnitude of maximum diastolic potential of SA node which increases firing rate
  - Other ions such as Na+ and Cl- have little effect on automatic cells
  - Calcium may play an important role in the automaticity of the cells.

The Physiologic Properties of the Heart

- Irritability: Excitation of the Heart
- Specialized excitatory and conductive system of the Heart
  - Sinus node
  - Internodal pathways
  - AV node
  - AV Bundle
  - LBB and RBB, Purkinje fibers
Automatic cells are found in

- SA node
  - normal pacemaker
- Around the opening of the coronary sinus
- AV node and its junction with the Bundle of His
- Specialized conducting tissue of the ventricles

SA node pacemaker because

1) Highest frequency of discharge
   - Other cells with low frequency of discharge
     - Called latent or potential pacemakers; abnormal or ectopic pacemakers
     - Become pacemaker when:
       - Develop rhythmical discharge rate that is more rapid than SA node
       - Develop excessive excitability
       - Blockage of transmission of the impulses from the SA node to other parts of the heart

2) Of overdrive suppression
Overdrive suppression

- The greater rhythmicity of the SA node forces the other automatic cells to fire off at a faster rate than their natural discharge rate. This causes depression of their rhythmicity.
- SA node rhythmical discharge rate = 70-80/min
- AV node = 40-60/min
- P fibers = 15-40/min

The frequency of discharge of impulses from the pacemaker is a function of:

- The slope of slow diastolic depolarization (pre potential)
- The value of the maximum diastolic potential (beginning of phase 4)
- The value of the threshold voltage or the critical firing level
- Duration of the period of recovery
CONDUCTIVITY

- spread of excitation
- Excitation – originates from the SA node
  - Conduction velocity in atrial muscle = 0.3 to 0.5 m/sec
  - Conduction is faster in the interatrial bundles (presence of specialized conduction fibers)

CONDUCTIVITY

- Excitation reaches the AV node through the internodal tracts
  - Conduction is slowed down (AV nodal delay)
    - due to slow conduction velocity
    - assures proper coordination between atrial and ventricular contraction
  - As the AP spreads to the N zone there is progressive reduction in the:
    - RMP
    - Amplitude and overshoot of the AP
    - Velocity of phase 0 depolarization
    - Conduction velocity

Conduction velocity through the AV node = 0.1 to 0.2 m/sec (primarily unidirectional)
CONDUCTIVITY

- Conduction velocity:
  - 0.03 m/sec internodal pathway to AV node
  - 0.09 m/sec AV node itself
  - 0.04 m/sec penetrating AV bundle
- Total delay in the AV nodal and AV bundle system = 0.13 m/sec + 0.03 m/sec from SA to AV node = 0.16 m/sec

CONDUCTIVITY

- Cause of slow conduction in the transitional, nodal, and penetrating AV bundle fibers:
  1) Their sizes are considerably smaller than the sizes of the normal atrial muscle fibers
  2) All these fibers have RMP that are much less negative than the normal RMP of other cardiac muscle
  3) Few gap junctions connect the successive muscle cells in the pathway
CONDUCTIVITY

- Excitation reaches the Bundle of His
  - Velocity of conduction = 3-4 m/sec
  - Increased magnitude of the AP; increased velocity of phase 0 depolarization; increased duration of the AP
  - Excitation transmitted to the RBB and LBB and fascicles then to the ventricular muscle

CONDUCTIVITY

- The rapid transmission of APs by the P fibers is believed to be caused by:
  1) A high level of permeability of the gap junction at the intercalated discs
  2) They have few myofibrils
- From the time the cardiac impulse enters the bundle branches in the ventricular septum until it reaches the terminations of the P fibers, the total time that elapses average only 0.03 seconds.
CONDUCTIVITY

- Excitation of the ventricles begin at the left side of the ventricular septum, to the epicardial surface of the RV near the apex, travels concentrically towards the base of the heart.
  - The last area to be depolarized is the posterobasal portion of the ventricles near the septum.
  - Conduction velocities = 0.35 to 0.40 m/sec.
- Note: AP of endocardial cells lasts longer than that of epicardial cells so that depolarization proceeds from endo to epicardial surface but repolarization travels from epicardial to endocardial surface.

CONDUCTIVITY

- Effects of repetitive stimulation:
  - Production of heart blocks
  - Overdrive suppression
  - Overdrive enhancement
  - Changes in the AP are:
    - Increasing RMP which was initially low
    - Increasing amplitude of AP
    - Increasing velocity of phase 0 depolarization
    - Development of phase 4 slow diastolic depolarization
CONTRACTILITY

- EXCITATION-CONTRACTION COUPLING
  - The mechanism by which the action potential causes the myofibrils to contract
Differences with that of Skeletal Muscle

- A large quantity of extra Ca++ diffuses into the sarcoplasm from the T tubules themselves at the time of the AP. Without this extra Ca++ from the T tubules, the strength of cardiac muscle contraction would be considerably reduced because:
  - the sarcoplasmic reticulum of cardiac muscle is less well developed and does not store enough Ca++ to provide full contraction
  - the T tubules of cardiac muscle have a diameter of 5x as great as that of skeletal muscle tubules which means a volume 25x as great
  - inside the T tubules is a large quantity of mucopolysaccharides that are electronegatively charged and bind an abundant store of even more Ca++, keeping them always available for diffusion to the interior of the cardiac muscle fiber when the T-tubule AP occurs.
- The strength of contraction of cardiac muscle depends to a great extent on the concentration of calcium ions in the ECF.

Some Characteristics of Cardiac Muscle Contraction

- All-or-none response
  - Due to stimulation of all fibers in the cardiac tissue. This is made possible through rapid spread of excitation from cell to cell
- Tetanic contractions cannot be produced
  - The cardiac muscle membrane is in absolute refractory period up to early phase 3 of the AP. Related to muscle contraction, the period would extend to the end of muscle contraction. Therefore, no response can be elicited until the muscle is in the relaxation period.
- Effect of initial stretch: the Frank-Starling law.
  - Within certain limits, stretching the muscle fiber results in increased force of contraction. Translated to the ventricle, if more blood returns to it before it contracts (increased ventricular filling, increased EDV), the subsequent contraction is more forceful and more blood is ejected into the circulation.
Thank You!

For Listening?