PROPERTIES OF CARDIAC MUSCLES

LECTURE OBJECTIVES
By the end of this lecture students should be able to:
• Define the type of cardiac muscle cells
• Define the different properties of cardiac muscles
• Understand the implications of the properties of cardiac muscles
• Understand the mechanism of pacemaker activity of the heart

TYPES OF CARDIAC MUSCLES
There are two types of cardiac muscles:
• Structural/contractile
• Pacemaker cells

CARDIAC MUSCLE CELLS

CONTRACTILE CELLS:
• Similar to skeletal muscle
• 3 differences:
  1. Longer action potential
  2. Entry of extracellular calcium ions through voltage-regulated calcium channels
    • Delay depolarization
    • Initiate contraction
    • Trigger release of Ca2+ from reserves in sarcoplasmic reticulum
  3. Longer contraction

PROPERTIES OF CARDIAC MUSCLE CELLS
• Contractile cells: Contractility
• **Excitable cells**: Automacity

• *Twitch summation does not occur: Tetanus is not possible*

**Automaticity (Autorhythmicity)**
The ability to produce spontaneous rhythmic excitation without external stimulus.

(1) Intrinsic rhythm of excitable cells
- Purkinje fiber: **15 – 40 /min**
- Atrioventricular node: **40 – 60 /min**
- Sinoatrial node: **90 – 100 /min**

(2) NORMAL PACEMAKER OF THE HEART: **SA NODE (SINO ATRIAL NODE)**
Exhibits:
1) The capture effect
2) Overdrive suppression

**FACTORS DETERMINING AUTOMATICITY**
1) Depolarization rate of phase 4

2) Threshold potential
3) The maximal repolarization potential

**CONDUCTIVITY**

**ACTION POTENTIAL OF CARDIAC MUSCLE CELLS (CONTRACTILE MUSCLES)**

- **Inactivating K channels** ($I_{TO}$)
- **"Slow" K channels** ($I_{KS}$)
- Voltage-gated Na Channels
- Voltage-gated Ca Channels

Ventricular muscle membrane potential (mV)

- TP
- $I_{K1}$

Time scales:
- 100 ms
- 200 msec
SA NODE ACTION POTENTIAL

SA node membrane potential (mV)

REFRACTORY PERIOD

- **Absolute Refractory Period** – regardless of the strength of a stimulus, the cell cannot be depolarized.
- **Relative Refractory Period** – stronger than normal stimulus can induce depolarization.
**REFRACTORY PERIOD**

- The plateau phase of the cardiac cell AP increases the duration of the AP to 300 msec.
- The refractory period of cardiac cells is long (250 msec).
  - compared to 1-5 msec in neurons and skeletal muscle fibers.

**IMPORTANCE OF REFRACTORY PERIOD**

- Long refractory period prevents tetanic contractions.
• systole and diastole occur alternately.
• It is very important for pumping blood to arteries.

**SUPRANORMAL PERIOD**

• The cells can be restimulated and the threshold is actually lower than normal.
• Occurs early in phase 4 and is usually accompanied by positive after-potentials as some potassium channels close.
• *Can be source of reentrant arrhythmias especially when phase 3 is delayed as in long Q-T syndrome*
PREMATURE EXCITATION, PREMATURE CONTRACTION AND COMPENSATORY PAUSE
CONDUCTING SYSTEM OF HEART

FLOW OF CARDIAC ELECTRICAL ACTIVITY (ACTION POTENTIALS)

Pacing (sets heart rate)

- **Atrial Muscle**: 0.4 m/s
- **AV node**: 0.02 m/s Delay
- **Purkinje System**: 4 m/s Rapid, uniform spread
- **Ventricular Muscle**: 1 m/s
CHARACTERISTICS OF CONDUCTION IN HEART

- Delay in transmission at the A-V node (150 –200 ms)
- This determines the sequence of the atrial and ventricular contraction

- Rapid transmission of impulses in the Purkinje system
- This synchronize contraction of entire ventricles

FACTORS DETERMINING CONDUCTIVITY

- Anatomical factors

- Physiological factors

ANATOMICAL FACTORS

- A. Gap junction between working cells
  - functional atrial and ventricular syncytium
  - Diameter of the cardiac cell – causes conductive resistance –

COORDINATING THE PUMP: ELECTRICAL SIGNAL FLOW
PHYSIOLOGICAL FACTORS

Neural and humoral control of the cardiac function
• Sympathetic
• parasympathetic
• T3, T4

EFFECT OF AUTONOMIC NERVE ACTIVITY ON THE HEART

<table>
<thead>
<tr>
<th>Region Effected</th>
<th>Sympathetic Nerve</th>
<th>Parasympathetic Nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>SA node</td>
<td>Increased rate of diastole depolarization ; increased cardiac rate</td>
<td>Decreased rate of diastole depolarization ; Decreased cardiac rate</td>
</tr>
<tr>
<td>AV node</td>
<td>Increase conduction rate</td>
<td>Decreased conduction rate</td>
</tr>
<tr>
<td>Atrial muscle</td>
<td>Increase strength of contraction</td>
<td>Decreased strength of contraction</td>
</tr>
<tr>
<td>Ventricular muscle</td>
<td>Increased strength of contraction</td>
<td>No significant effect</td>
</tr>
</tbody>
</table>
Ach ON ATRIAL ACTION POTENTIAL

0 mv
Voltage

Time

(↑) K⁺ Conductance (Efflux)
EFFECTS OF SYMPATHETIC NERVE

• Postganglionic sympathetic nerves (epinephrine) and adrenal gland (epinephrine and norepinephrine)
• Binds with $\beta_1$ receptor on cardiac cells $\rightarrow$ increase the Ca2+ channel permeability $\rightarrow$ Ca2+ channel permeability increase
• *Increase the spontaneous depolarization rate at phase 4*
• Automaticity of SA node cell rise
• heart rate increase
• **Positive chronotropic effect**

ISOVOLUMETRIC CONTRACTION

• During the time period between the closure of the AV valves and the opening of the aortic and pulmonic valves
• Ventricular pressure rises rapidly without a change in ventricular volume (i.e., no ejection occurs).
• *Ventricular volume does not change because all valves are closed during this phase. Contraction, therefore, is said to be "isovolumic" or "isovolumetric."*

ISOVOLUMETRIC RELAXATION
All Valves Closed

• When the intraventricular pressures fall sufficiently at the end of phase 4, the aortic and pulmonic valves abruptly close causing the beginning of isovolumetric relaxation.
• *After valve closure, the aortic and pulmonary artery pressures rise slightly followed by a slow decline in pressure.*
• The rate of pressure decline in the ventricles is determined by the rate of relaxation of the muscle fibers.
• This relaxation is regulated largely by the sarcoplasmic reticulum that are responsible for rapidly re-sequestering calcium following contraction

REFERENCES
• GUYTON AND HALL text book of medical physiology
• GANONG’s review of medical physiology
• www.medscape.com

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