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Cardiac Muscle I

M1- Cardiovascular/Respiratory Sequence
Louis D’Aleyc, Ph.D.

Fall 2008
Tuesday 10/28/08, 10:00
Cardiac Muscle I
19 Slides, 50 min

1. CM structure
2. CM contractile function
3. Ca^{++} induced Ca^{++} release
4. Isometric contraction
5. Isotonic contraction
6. Afterloaded contraction
General Structure = muscle!

- Superior vena cava
- Sinoatrial node
- Atrioventricular node
- Bundle of His

4 Chambers
4 Valves
Syncytium Conducting system
ALL MUSCLE

- Right bundle branch
- Purkinje fibers
- Left bundle branch

Different Conduction velocities
<table>
<thead>
<tr>
<th>Requirement</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Synchronized</td>
<td>not arrhythmic</td>
</tr>
<tr>
<td>2) Valves open fully</td>
<td>not stenotic</td>
</tr>
<tr>
<td>3) Valves don't leak</td>
<td>not insufficient</td>
</tr>
<tr>
<td></td>
<td>or regurgitant</td>
</tr>
<tr>
<td>4) Forceful</td>
<td>not failing</td>
</tr>
<tr>
<td>5) Must fill</td>
<td>Not &quot;dry&quot;</td>
</tr>
</tbody>
</table>
Intercalated Disc =

- Firm mechanical connection
- Low resistance electrical connection

Cardiac myocyte

All these are muscle cells

SA node - pacemaker

Purkinje fibers

AV node

One heart - ~ 1 billion cells

Source Undetermined (All Images)
Functional syncytium
<table>
<thead>
<tr>
<th></th>
<th>SKELETAL</th>
<th>CARDIAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanism of excitation</td>
<td>Neuromuscular transmission</td>
<td>Pacemaker potentials</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Electrotonic depolarization via gap junctions</td>
</tr>
<tr>
<td>Electrical activity of muscle cell</td>
<td>Action potential spikes</td>
<td>Action potential plateaus</td>
</tr>
<tr>
<td>Ca²⁺ sensor</td>
<td>Troponin</td>
<td>Troponin</td>
</tr>
<tr>
<td>Excitation-contraction coupling</td>
<td>L-type Ca²⁺ channel (DHP receptor) in T-tubule membrane coupling to Ca²⁺ release channel (ryanodine receptor) in SR</td>
<td>Ca²⁺ entry via L-type Ca²⁺ channel (DHP receptor) triggers Ca²⁺-induced Ca²⁺ release from SR</td>
</tr>
<tr>
<td>Terminates contraction</td>
<td>Breakdown of ACh by acetylcholinesterase</td>
<td>Action potential repolarization</td>
</tr>
<tr>
<td>Twitch duration</td>
<td>20–200 msec</td>
<td>200–400 msec</td>
</tr>
<tr>
<td>Regulation of force</td>
<td>Frequency and multifiber summation</td>
<td>Regulation of calcium entry</td>
</tr>
<tr>
<td>Metabolism</td>
<td>Oxidative, glycolytic</td>
<td>Oxidative</td>
</tr>
</tbody>
</table>
CALCIUM-INDUCED CALCIUM RELEASE

1. “Excitation” (Depolarization of plasma membrane)
2. Opening of voltage-sensitive Ca$^{++}$ channels in transverse tubules
3. Flow of Ca$^{++}$ into cytosol (small amount ~20%)
4. Ca$^{++}$ binds to Ca$^{++}$ receptors (Ryanodine receptor) on the external surface of the sarcoplasmic reticulum within the cell
5. Opening of Ca$^{++}$ channels (large amount of calcium release ~ 80%)
6. Flow of Ca$^{++}$ into cytosol
7. Cytosolic Ca$^{++}$ conc. increases (10$^{-7}$ M to ~10$^{-5}$ M)
8. Contraction

0.1 µM to 100 µM
Calcium-Induced Calcium Release

Essentially defines ‘contractility’.

Source Undetermined
ISOMETRIC LENGTH-TENSION
The difference between "Total" and "Passive" tension.
Optimized stretching

Good

“Over stretched”
Bad
Passive stretch & Isometric contraction

“At rest” Diastole

Inactive                      Active

Contracting Systole

Same tension (load) but shorter length.
Isotonic = Shortening at same tension

1 to 3 Isotonic at 1 g tension

2.9 MH
1-2-3 Isotonic

1-4-5 Afterloaded

"COUNTER CLOCKWISE ROTATION"

Terms Related to Cardiac Performance

**Preload** - The ventricular wall tension at the end of diastole.

**Afterload** -- The ventricular wall tension during contraction; the resistance that must be overcome for the ventricle to eject its contents. Approximated clinically by systolic ventricular or arterial pressure.
1. Afterloaded contraction (length-tension)
2. Afterloaded contraction (volume-pressure)
3. LaPlace
4. Wiggers diagram
5. Stroke volume & Ejection Fraction
6. Cardiac Output
7. Right pump
8. Preload (Frank-Starling), Afterload, & Contractility

3.3 MH
Pressure increases as radius decreases.

Ejection Fraction = \( \frac{70}{130} = 54\% \)
Law of La Place

\[ T = P \times r \]  (see page 44 of M&H)

The tension (T) in the ventricular wall depends upon both the pressure (P) in the chamber and the radius (r) of the chamber.

Thus as the ventricle gets smaller during ejection the pressure within increases even at the same muscle tension.

Same \( T = P \times r \)
A PRESSURE-VOLUME LOOP

Aortic valve closes

Aortic valve opens

Left ventricular pressure (mm Hg)

Stroke volume

End-systolic volume

End-diastolic volume

Mitral valve opens

Mitral valve closes

COUNTER CLOCKWISE ROTATION
Ventricular

A = diastole

B = ventricular systole

C = isovolumetric contraction

D = isovolumetric relaxation

Three Pressures

mmHg

Textbooks vary in definitions but the more common uses of the unmodified terms “systole” and “diastole” are:

**Systole** is the period from the closing of the atrio-ventricular valve (mitral) to the closing of the aortic valve (ventricular contraction).

**Diastole** is the period from the closing of the aortic valve to the closing of the atrio-ventricular valve (ventricular relaxation and filling).

M & H NOTE: Your text distinguishes **ventricular systole** from **arterial systole**:

**Ventricular systole** is the period from the closing of the atrio-ventricular valve (mitral) until its opening. (Fig 3.1 M &H)

**Arterial systole** is the period from the opening of the aortic valve until its closing.
3.1 MH

Ventricular Filling (volume mL)
Flow mL/min

LV end-diastolic Volume

Small P & V
Contribution from Atrial contraction.
Heart is a Pressure Pump but also pumps volume/time.

Stroke Volume = volume pumped with each beat of the heart.

Heart Rate X Stroke Volume = Cardiac Output

\[ \text{HR} \times \text{SV} = \text{CO} \]
\[ \text{b/min} \times \text{mL} / \text{b} = \text{mL} / \text{min} \]
Volume Pumped

RV

\[ \text{Lungs} \]

= Volume Pumped \ LV

\[ \text{Capillaries} \]
Not 125 mmHg

Figure 3.2 Cardiac cycle—right heart.
Factors influencing heart rate

+ and (-) CHRONOTROPIC EFFECTS

- Plasma epinephrine
  - Activity of sympathetic nerves to heart
  - Activity of parasympathetic nerves to heart

SA node

- Heart rate
Terms Related to Cardiac Performance

**Preload** - The ventricular wall tension at the end of diastole.

**Afterload** -- The ventricular wall tension during contraction; the resistance that must be overcome for the ventricle to eject its contents. Approximated by systolic ventricular or arterial pressure.

**Contractility** -- Property of heart muscle that accounts for changes in strength of contraction independent of preload and afterload.
Complex interactions so we will treat each separately with others held constant.
Increased Preload Increases Stroke Volume
Frank-Starling

Contractility & Afterload ~ CONSTANT

Increased Preload ~ Increases SV
(Frank-Starling Mechanism)

LV Pressure

Contractility & Afterload ~ CONSTANT

However! Excessive Diastolic Volume or Pressure Decreases Developed Tension

![Graph showing the relationship between diastolic volume and pressure, with arrows indicating the decrease in pressure as diastolic volume increases.](image)
Increased Afterload Decreases SV

Contractility & Preload ~ CONSTANT

Increased Afterload Decreases SV

Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6\textsuperscript{th} ed.
Stroke Volume

Contractility

Preload

Afterload

Complex interactions so we will treat each separately with others held constant.
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