The Heart
Objectives

• Describe the organization of the cardiovascular system

• Compare the properties of cardiac, smooth and skeletal muscle. What specializations of cardiac muscle facilitate heart function?

• What are the ionic mechanisms of the cardiac RP and AP?

• Describe the cardiac cycle with respect to chamber pressures, vessel pressures, EKG, and heart sounds

• Describe the structure and function of the electrical system of the heart
The heart as a pump

- 2 parallel pumps, right and left heart
- Each has an atrium and a ventricle
- Atria are primer pumps
- Ventricles provide main pumping force
- Pressure gradient exists in a tubular system
- Cardiac muscle/fibrous connective tissue
- Specialized muscle fibers and autonomic nerves connect atria and ventricles
The heart as a pump

- Pericardium prevents overlengthening of sarcomeres - heart contractions grow stronger as its volume increases
- Spontaneous pacemaker activity
- Excitable properties of cardiac cells vary with location
How does blood flow through the heart?
Name the valves
Pulmonary and systemic circulation
Nerve fibers, electrical connections

Figure 8-1 Structure of the heart and course of blood flow through the heart chambers.
Cardiac Muscle

- Branched
- Striated
- Syncytial
- Actin/myosin
- T-tubules open to SR

Figure 8-2 The “syncytial,” interconnecting nature of cardiac muscle.
Cardiac Muscle

- Design a heart using skeletal muscle
Cardiac Muscle

- Design a heart using skeletal muscle
- Pacemaker?
- Interconnections?
- Fatigue?
- Response to hormones?
<table>
<thead>
<tr>
<th>Property</th>
<th>Cardiac</th>
<th>Smooth</th>
<th>Skeletal</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Striated</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>(b) Contraction by sliding filaments</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>(c) Contraction regulated by Ca, ATP</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>(d) Prominent sarcoplasmic reticulum</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>(e) Cells electrically coupled</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>(f) AP due to increased gCa</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>(g) AP duration, msec</td>
<td>500</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>(h) Refractory period</td>
<td>long</td>
<td>moderate</td>
<td>short</td>
</tr>
<tr>
<td>(i) Tetany</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>(j) Tension regulated by neurotransmitters</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>(k) Tension regulated by hormones</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>(l) Spontaneous depolarization</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>
Ionic mechanisms of cardiac AP

- Rp is -85 to -95 MV in atrial fibers; -90-100 mv in purkinje fibers
- Ap controlled by
  - Fast Na /Slow Ca channels
  - Channels stay open longer - creates plateau
  - Prolonged depolarization
  - Refractory period
Plateau/ conduction velocity/ refractory period

- Plateau prevents tetany but allows orderly spread of contraction
- AV fibers 0.3-0.5 m/s
- Purkinje fibers variable velocity depending on location
- Refractory period varies depending on location
Figure 8-3 Rhythmic action potentials from a Purkinje fiber and from a ventricular muscle fiber, recorded by means of microelectrodes.
Plateau (maintained depolarization) due to opening of voltage-gated slow Ca\textsuperscript{2+} channels and closing of some K\textsuperscript{+} channels

1. Rapid depolarization due to opening of voltage-gated fast Na\textsuperscript{+} channels

2. Repolarization due to opening of voltage-gated K\textsuperscript{+} channels and closing of Ca\textsuperscript{2+} channels

0.3 sec = 300 msec

- Depolarization
- Repolarization
- Refractory period
- Contraction

(a) Action potential, refractory period, and contraction

(b) Membrane permeability (P) changes
Excitation contraction coupling

- **Ca sources:**
  - T tubules connected to Sarcoplasmic membrane
  - ECF Ca influences contraction
  - Larger T tubules hold more Ca
- **Neg. charged molecules sequester Ca**
(a) Cardiac muscle fibers

- Desmosomes
- Mitochondrion
- Cardiac muscle fiber
- Nucleus
- Sarcolemma
- Intercalated discs
- Opening of transverse tubule
- Gap junctions
(b) Cardiac myofibrils based on an electron micrograph
Cell types coordinate function

- Fast response cells:
  - Atria and ventricles
  - Rp -80 to -90mv
  - Do not spontaneously depolarize
  - Short refractory period
  - Fast conduction velocity

- Slow response cells:
  - Conductive system
  - Rp -55 to -60 mV
  - Spontaneously depolarize
  - Long refractory period
  - Slow conduction velocity
Cardiac cycle

• Describe the cardiac cycle with respect to chamber pressures, vessel pressures, EKG, and heart sounds
Electrical system of the heart

**Figure 9-1** The sinus node and the Purkinje system of the heart, showing also the A-V node, the A-V bundle, the atrial internodal pathways, and the ventricular bundle branches.

**Figure 9-4** Transmission of the cardiac impulse through the heart, showing the time of appearance (in fractions of a second) of the impulse in different parts of the heart.
Electrical system of the heart

1. SINOATRIAL (SA) NODE
2. ATRIOVENTRICULAR (AV) NODE
3. ATRIOVENTRICULAR (AV) BUNDLE (BUNDLE OF HIS)
4. RIGHT AND LEFT BUNDLE BRANCHES
5. CONDUCTION MYOFIBERS (PURKINJE FIBERS)

Anterior view of frontal section
Cardiac cycle of the heart

 contraction  Relaxation
Electrocardiogram

• P wave
  - Atrial depolarization
• QRS waves
  - Ventricular depolarizations
• T wave
  - Ventricular repolarization
Cardiac cycle of the heart

- Contraction
- Relaxation

- Ejection
-Isovolumic contraction
- Aortic valve opens
- Aortic valve closes
- A-V valve opens
- A-V valve closes
- Diastasis
- Atrial systole
- Isovolumic relaxation
- Rapid inflow

Pressure (mm Hg)

Volume (ml)

Electrocardiogram

Phonocardiogram

Systole

Diastole

1st

2nd

3rd

a
c
v

Atrial pressure
Ventricular pressure
Ventricular volume

P
Q
S
T

contraction
Relaxation
Figure 8–5 Mitral and aortic valves.
Regulation of heart pumping

• Intrinsic control
  • Frank Starling Mechanism
  • Venous return determines output
  • Greater return stretches heart more - yields stronger contraction

• Autonomic control
  • Sympathetic and parasympathetic nerve inputs
  • Cardiac output increases with sympathetic stimulation
  • Cardiac output decreases with parasympathetic stimulation (vagal stimulation)
Ventricular force curve

- Pressure rises
- Ventricular output increases

**Figure 8-6** Approximate normal right and left ventricular output curves for the unstimulated human heart, as extrapolated from data obtained in dogs.
Regulation of heart pumping

- **Intrinsic control**
  - Frank Starling Mechanism
  - Venous return determines output
  - Greater return stretches heart more - yields stronger contraction

- **Autonomic control**
  - Sympathetic and parasympathetic nerve inputs
  - Cardiac output increases with sympathetic stimulation
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Figure 8-7 The cardiac nerves. (A-V = atrioventricular; S-A = sinoatrial.)
Cardiac output and extrinsic control

Figure 8-8: Effect on the cardiac output curve of different degrees of sympathetic and parasympathetic stimulation.
K/Ca and heart function

- K dramatically changes membrane potentials
- Excess K - heart dilated and flaccid
- Excess Ca - heart shows spastic contractions
Figure 9-2 Rhythmical discharge of a sinus nodal fiber. Also, the sinus nodal action potential is compared with that of a ventricular muscle fiber.
Figure 9-1 The sinus node and the Purkinje system of the heart, showing also the A-V node, the A-V bundle, the atrial internodal pathways, and the ventricular bundle branches.
Figure 9-3 Organization of the A-V node. The numbers represent the intervals of time from the origin of the impulse in the sinus node. The values have been extrapolated to the human being.
Key:
- Blue: Atrial contraction
- Yellow: Ventricular contraction

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