CARDIOVASCULAR PHYSIOLOGY:
THE HEART
Cardiovascular System Overview

• Cardiovascular system components
  – Heart
  – Blood vessels
  – Blood

• Cardiovascular system functions
  – Transportation of substances
    • Respiration
    • Nutrition
    • Excretion
    • Hormones
  – Regulation
  – Protection
Cardiac Muscle

• Characteristics
  – Some cells in the atria secrete a peptide hormone called *atrial natriuretic factor* (ANF)
    • Causes natriuresis
    • Vasodilation

• Conducting system
  – 1% of cells
  – Initiates heartbeat and spreads the impulse throughout the heart

• Innervation

• Blood supply
  – Coronary circulation
Heartbeat Coordination

• SA node is the pacemaker of the heart
  – Initializes depolarization
  – Determines heart rate

• Pathway
  – SA node
  – Across atria, then down
  – AV node
  – Bundle of His
    • R/L bundle branches
  – Purkinje fibers
Sequence of Cardiac Excitation

Atrial excitation:
- Begins
- Complete

Ventricular excitation:
- Begins
- Complete

Ventricular relaxation:

SA node
AV node
Atrial relaxation

Electrocardiogram:
- Time
- Time
- Time
Myocardial Action Potential

- vg Na⁺ channels open (depolarization)
- **L-type Ca^{2+} channels** open
- Membrane remains depolarized
  - Ca^{2+} influx sustains depolarization
  - K⁺ channels remain closed
- vg K⁺ channels open (depolarization)

RMP = -90 mV
Threshold = -60 mV
Nodal Cell Action Potential

- Pacemaker potential
  - Slow depolarization
  - Automaticity (spontaneous, rhythmical)
- Voltage-gated $K^+$ channels close
- **$F$-type $Na^+$ channels** open when the membrane potential is at negative values
- **$T$-type $Ca^{2+}$ channels** open briefly
  - Inward $Ca^{2+}$ current
  - Final depolarizing boost to threshold
Electrical Events of the Heart

- **Electrocardiogram (ECG)**
  - Measures the currents generated in the ECF by the changes in many cardiac cells

- **P wave**
  - Atrial depolarization

- **QRS complex**
  - Ventricular depolarization
  - Atrial repolarization

- **T wave**
  - Ventricular repolarization
Excitation-Contraction Coupling

- Ca\(^{2+}\) entering through L-type Ca\(^{2+}\) channels triggers the release of more Ca\(^{2+}\) from the ryanodine receptors in the SR
  - *Calcium induced calcium release*

- Cross-bridge cycling occurs

- Contraction ends when Ca\(^{2+}\) is pumped back into the SR by Ca\(^{2+}\)/ATPase pumps and Na\(^+\)/Ca\(^{2+}\) counter-transporters
Refractory Period of the Heart

- Long absolute refractory period prevents tetany
  - Muscle can not be stimulated in time to produce summation
- Absolute refractory period for cardiac muscle is 20-200 ms
  - Skeletal muscle 1-2 ms
Mechanical Events of the Heart

- Cardiac cycle
  - Pressure and volume changes that occur during the cardiac cycle
  - Average heart rate 72 bpm
  - Each cardiac cycle lasts 0.8 s
    - 0.3 s in systole
    - 0.5 s in diastole

- 2 alternating phases
  - **Systole**
    - Ventricular contractions and blood ejection
  - **Diastole**
    - Ventricular relaxation and blood filling
Cardiac Cycle

(a) Systole

- **Isovolumetric ventricular contraction**
- **Ventricular ejection**

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(b) Diastole

- **Isovolumetric ventricular relaxation**
- **Ventricular filling**

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Cardiac Cycle
Systole

• *Isovolumetric Ventricular Contraction*
  – Ventricle contracting
    • Muscle fibers developing tension
    • Muscle fibers do not shorten
    • Increasing pressure inside the ventricles
  – All valves closed
  – No blood ejection
  – Ventricular volume remains the same
Cardiac Cycle

Systole

• **Ventricular Ejection**
  – Pressure in the ventricles exceed pressure in aorta/pulmonary trunk
  – Semilunar valves open
  – Blood forced into aorta/pulmonary trunk
  – Muscle fibers shorten

• **Stroke volume (SV)**
  • Volume of blood ejected during systole
  • $SV = 135 \text{ mL} (EDV) - 65 \text{ mL} (ESV)$
  • Average $SV$ is 70 mL/beat (0.07 L/min)
Cardiac Cycle

Diastole

• *Isovolumetric Ventricular Relaxation*
  – Ventrices begin to relax
  – Semilunar valves close
  – AV valves closed
  – No blood entering or leaving the ventricles
  – Ventricular volume remains the same
Cardiac Cycle
Diastole

• **Ventricular Filling**
  – AV valves open
  – Blood flows from atria to ventricles
  – 80% of ventricular filling is passive
  – Atrial contraction occurs at the end of diastole
    • Atrial kick moves the remaining 20% of blood in atria into ventricles
Cardiac Cycle
Volumes

• *End-diastolic volume*
  – *EDV*
  – Volume in the ventricles at the end of diastole

• *End-systolic volume*
  – *ESV*
  – Volume in the ventricles at the end of systole
Pressure Changes

Volume Changes

ECG
Heart Sounds

- **Lub**
  - Soft sound
  - Closing of the AV valves
  - Onset of systole

- **Dup**
  - Louder sound
  - Closing of the semilunar valves
  - Onset of diastole
Cardiac Output (CO)

- Volume of blood pumped out of the ventricles expressed as L/min
  - Volume of blood flowing through either the pulmonary or systemic circuit per minute
- \( \text{CO} = \text{HR} \times \text{SV} \)
- \( \text{CO} = 72 \text{ beats/min} \times 0.07 \text{ L/beat} \)
- \( \text{CO} = 5.0 \text{ L/min} \)
- Total blood volume is pumped around the circuit once each minute
  - 1,440 per day!
Control of Heart Rate

*HR is a variable that determines CO*

- 100 BPM without nerve or hormone influence on the SA node
- However, SA node is under constant influence of nerves and hormones
  - Activity of the parasympathetic nerves causes a decrease in heart rate
  - Activity of sympathetic nerves causes an increase in heart rate
Control of Heart Rate

*HR is a variable that determines CO*

- **Sympathetic stimulation**
  - Increases slope
  - Increases F-type Na\(^+\) channel permeability
  - Faster depolarization

- **Parasympathetic stimulation**
  - Slope decreases
  - Hyperpolarizes plasma membrane of SA node
  - Increases K\(^+\) permeability
Control of Heart Rate

*HR is a variable that determines CO*

- **Epinephrine**
  - Increases HR
  - Binds to beta-adrenergic receptors in the SA node

- **Heart rate is also sensitive to changes in:**
  - Body Temperature
  - Plasma electrolyte concentrations
    - $K^+$
    - $Ca^{2+}$
Control of Stroke Volume

SV is a variable that determines CO

• Ventricles do not completely empty during contraction
• More forceful contraction can produce an increase in SV by causing greater emptying
• 3 main factors
  1. Changes in EDV (*preload*)
  2. Changes in contractility
  3. Changes in *afterload*
     • arterial pressures against which the ventricles pump
     • Increase in total peripheral resistance (TPR)
Starling’s Law of the Heart

Relationship between EDV and SV

• Ventricles contract more forcefully during systole when it has been filled to a greater degree during diastole

• **SV increases as EDV increases**

• Increase in venous return forces an increase in CO by increasing EDV which increases SV
Sympathetic Regulation

- Sympathetic nerves innervate the entire myocardium
- NE and Epi bind to beta-adrenergic receptors to increase *contractility*
  - Increases strength of contraction at any given EDV
  - Independent of EDV
  - Leads to an increase in ejection fraction
Ejection Fraction (EF)

- EF quantifies contractility
- EF = SV/EDV
- Under resting conditions, average is between 50–75%
- Increased contractility causes increased EF
Sympathetic Regulation

• Increased sympathetic activity
  – Increases HR without decreasing CO
  – Increases contractility
  – Ventricles contract more forcefully to compensate for the increase in HR
Sympathetic Regulation of Myocardial Contractility
Control of Cardiac Output Summary

Begin

End-diastolic ventricular volume

Activity of sympathetic nerves to heart

Activity of parasympathetic nerves to heart

Plasma epinephrine

Cardiac muscle

SA node

Cardiac output = Stroke volume x Heart rate