The Cardiovascular System

Part 2

Properties of cardiac muscle

- Cardiac muscle
  - Striated
  - Short
  - Wide
  - Branched
  - Interconnected

- Skeletal muscle
  - Striated
  - Long
  - Narrow
  - Cylindrical

Properties of Cardiac Muscle

- Cells connected to each other by intercalated discs
  - Passage of ions between cells
  - Allows entire heart to work as a syncytium
- Numerous large mitochondria
  - 25–35% of cell volume
  - Increases cell capacity to do what?
- Able to utilize many food molecules
  - Example: lactic acid
  - Most cells primarily use what?

Some cells are myogenic

- Contractile impulse originates in non-contractile pacemaker cells, not in nerve cells
- Cardiac control center in medulla can increase or decrease rate through ANS
- Primary innervation is inhibitory through vagus nerve
  - Sympathetic or PNS?
- Because cells are joined by gap junctions, spontaneous depolarization of pacemaker cells initiates depolarization of contractile cells too
Properties of cardiac muscle

- Heart contracts as a unit or not at all (video)
  - Sodium channels leak slowly in specialized cells
  - Spontaneous depolarization
  - Leakage rate sets rhythm
  - Compare to skeletal muscle

Action Potential of Contractile Cardiac Muscle Cells

- Depolarization is due to fast opening of Na⁺ channels. A positive feedback rapidly opens many more Na⁺ channels, reversing the membrane potential. Channel inactivation ends this phase.
- Plateau is due to slow Ca⁺ influx through slow Ca⁺ channels.
- Repolarization is due to Ca⁺ channels inactivating and K⁺ channels opening. This allows K⁺ efflux, which brings the membrane potential back to its most negative voltage.

Conduction System of the Heart

- Terms
  - Systole
  - Contraction of ventricles
  - Diastole
  - Relaxation of ventricles

Conduction system of the heart

- SA node (pacemaker)
- Atrial depolarization and contraction
- AV node
- Bundle of His
- Right and left bundle branches
- Purkinje fibers
- Myocardium
- Contraction of ventricles
1. Sinoatrial (SA) node (pacemaker)
   - Generates impulses about 70-75 times/minute (sinus rhythm)
   - Depolarizes faster than any other part of the myocardium
   - Cut the vagus nerve = HR immediately increases by ~25bpm

2. Atrioventricular (AV) node
   - Delays impulses approximately 0.1 second
   - Depolarizes 50 times per minute in absence of SA node input

3. Atrioventricular (AV) bundle (bundle of His)
   - Only electrical connection between the atria and ventricles

4. Right and left bundle branches
   - Two pathways in the interventricular septum that carry the impulses toward the apex of the heart

5. Purkinje fibers
   - Complete the pathway into the apex and ventricular walls

Conduction Pathway
(a) Anatomy of the intrinsic conduction system showing the sequence of electrical excitation (Intrinsic Innervation)

Conduction System of the Heart

Conduction system of the heart

Conduction System of the heart
• Intrinsic innervation
• External influences
• Normal:
  - Average = 70 bpm
  - Range = 60-100 bpm

Conduction System of the heart
• Sympathetic stimulation
  - Enhances Ca²⁺ movement in contractile cells
  - Increases HR, contractility
    • Stroke volume decreases due to less ventricular filling time
  - Speeds relaxation
**Conduction System of the Heart**

- Parasympathetic stimulation
  - Dominant signal under resting conditions
  - Cardiac response mediated by acetylcholine
  - Opens K+ channels, hyperpolarizes cells
  - Decreases HR
  - Innervation of ventricles is sparse = little effect on contractility

**Other influences**

- Blood pressure
- Atrial reflex
  - Increased rate of blood returning to heart -> atrial stretching -> increased HR
- Hormonal
  - Epinephrine, thyroxine = increase HR
- Ion balance
- Exercise
- Temperature
- Exercise
- Age

**Homeostatic Imbalances**

Defects in the intrinsic conduction system may result in

1. Arrhythmias: irregular heart rhythms
2. Bradycardia < 60 bpm
3. Tachycardia >100 bpm
4. Maximum is about 300 bpm

**Electrocardiography**

- A composite of all the action potentials generated by nodal and contractile cells at a given time
  - Represents movement of ions = bioelectricity
  - Three waves
    1. P wave: electrical depolarization of atria
    2. QRS complex: ventricular depolarization
    3. T wave: ventricular repolarization

**Figure 18.16**

- Sinoatrial node
- Atrioventricular node
- Atrial depolarization, initiated by the SA node, causes the P wave.
- Ventricular depolarization begins at the AV node.
- Ventricular depolarization spreads to the ventricles.
- Ventricular repolarization begins at the apex.
- Ventricular repolarization is complete, causing the T wave.
- Depolarization is complete, and the QRS complex is complete.

**Figure 18.17**

- P wave: electrical depolarization of atria
- QRS complex: ventricular depolarization
- T wave: ventricular repolarization
Figure 18.18

(a) Normal sinus rhythm.

NORMAL HEART RHYTHM

(b) Junctional rhythm. The SA node is nonfunctional, P waves are absent, and heart is paced by the AV node at 40 - 60 beats/min.

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(c) Second-degree heart block. Some P waves are not conducted through the AV node; hence more P than QRS waves are seen. In this tracing, the ratio of P waves to QRS waves is mostly 2:1.

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(d) Ventricular fibrillation. These chaotic, grossly irregular ECG deflections are seen in acute heart attack and electrical shock.

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Atrial fibrillation. Rapid, irregular heartbeat. Note the absence of P waves (which represent depolarization of the top of the heart).

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Normal Atrial fibrillation

Video
The Cardiac Cycle

- All events associated with blood flow through the heart during one complete heartbeat
  - Systole — ventricular contraction (ejection of blood)
  - Diastole — ventricular relaxation (receiving of blood)

The Cardiac Cycle

- Three events
  1) Recordable bioelectrical disturbances (EKG)
  2) Contraction of cardiac muscle
  3) Generation of pressure and volume changes

- Blood flows from areas of higher pressure to areas of lower pressure
  - Valves prevent backflow

The Cardiac Cycle

- Ventricular filling → ventricular systole
  - Backpressure closes AV valves = isovolumetric contraction
  - Ventricular pressure becomes greater than aorta & pulmonary artery pressure = semilunar valves open
    - Portion of ventricular contents ejected

Heart Sounds

- Two sounds associated with closing of heart valves
  - First sound occurs as AV valves close and signifies beginning of systole = Lub
  - Second sound occurs when SL valves close at the beginning of ventricular diastole = Dub
- Heart murmurs = abnormal heart sounds
  - Most often indicative of valve problems
Heart sounds

• Valvular insufficiency (regurgitation)

Heart Sounds

• Mitral stenosis

Heart sounds

• Example: mitral valve prolapse

Cardiac Output (CO)

• Definition: volume of blood pumped by each ventricle in one minute
  ~ AKA: Minute volume

• 2 major factors
  ~ Stroke volume (SV)
  ~ Heart rate (HR)

Cardiac output

• CO (ml/min) = heart rate (HR) x stroke volume (SV)
  ~HR = number of beats per minute
  ~SV = volume of blood pumped out by a ventricle with each beat (ml/min)
Cardiac output

- Stroke volume
  - Difference between EDV and ESV
  - EDV - ESV = SV
  - Average is about 75 ml

Cardiac Output (CO)

- At rest
  - CO (ml/min) = HR (75 beats/min) × SV (70 ml/beat)
    = 5.25 L/min
  - Maximal CO is 4–5 times resting CO in nonathletic people
  - CO may reach 30 L/min in trained athletes
  - Cardiac reserve
    - Difference between resting and maximal CO

Regulation of stroke volume

- Stroke volume
  - Three main factors affect SV
    - Preload
    - Contractility
    - Afterload

Regulation of Stroke Volume

- Preload
  - Frank-Starling law of the heart: the heart can change its CO according to the incoming volume of blood
    - At rest, cardiac muscle cells are shorter than optimal length
    - Must be a degree of stretch of cardiac muscle cells before they contract
    - Slow heartbeat and exercise increase venous return
    - Increased venous return distends (stretches) the ventricles and increases contraction force
Regulation of Stroke Volume

- **Contractility**
  - Contractile strength at a given muscle length, independent of muscle stretch and EDV
  - **Agents increasing contractility**
    - Increased Ca\(^{2+}\) influx
    - Sympathetic stimulation
    - Hormones
      - Thyroxin, glucagon, and epinephrine
  - **Agents decreasing contractility**
    - Calcium channel blockers

- **Sympathetic nervous system**
  - Norepinephrine
    - Increased SA node firing rate
    - Faster conduction through AV node
    - Increases excitability of heart by increasing Ca\(^{2+}\) availability

- **Parasympathetic nervous system**
  - Vagus nerve
    - Decreases HR

Who dominates at rest?

Control of Heart rate

- **Sympathetic nervous system**
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Other factors...
Cardiac Output

Cardiac output

Heart rate

Stroke volume

Usually set by SA node

Preload

Contractility

Afterload

Rate of venous return

Ca²⁺, hormones

Blood pressure

Autonomic nervous system (among other things)