Heart

- Pericardium
- Myocardium
- Fibrous pericardium
- Parietal layer of serous pericardium
- Pericardial cavity
- Visceral layer of serous pericardium (epicardium)
- Myocardium
- Endocardium
- Heart chamber
- Heart wall
Heart

- Brachiocephalic artery
- Superior vena cava
- Right pulmonary artery
- Ascending aorta
- Pulmonary trunk
- Right pulmonary veins
- Right atrium
- Right coronary artery (in right atrioventricular groove)
- Anterior cardiac vein
- Right ventricle
- Marginal artery
- Small cardiac vein
- Inferior vena cava
- Left common carotid artery
- Left subclavian artery
- Aortic arch
- Ligamentum arteriosum
- Left pulmonary artery
- Left pulmonary veins
- Left atrium
- Auricle
- Circumflex artery
- Left coronary artery (in left atrioventricular groove)
- Left ventricle
- Great cardiac vein
- Anterior interventricular artery (in anterior interventricular sulcus)
- Apex

(b)
Capillary beds of lungs where gas exchange occurs

Pulmonary Circuit
- Pulmonary arteries
- Pulmonary veins
- Aorta and branches

Venae cavae

Left atrium
Left ventricle
Right atrium
Right ventricle
Heart

Systemic Circuit

Key:
- Red = Oxygen rich, CO2-poor blood
- Blue = Oxygen poor, CO2-rich blood

Capillary beds of all body tissues where gas exchange occurs
Heart

(a) Blood returning to the heart fills atria, putting pressure against atrioventricular valves; atrioventricular valves forced open

(b) As ventricles fill, atrioventricular valve flaps hang limply into ventricles

(a) Atria contract, forcing additional blood into ventricles

(b) Ventricles contract, forcing blood against atrioventricular valve cusps

2 Atrioventricular valves close

3 Papillary muscles contract and chordae tendineae tighten, preventing valve flaps from evert ing into atria
Heart

As ventricles contract and intraventricular pressure rises, blood is pushed up against semilunar valves, forcing them open

(a) Semilunar valve open

As ventricles relax and intraventricular pressure falls, blood flows back from arteries, filling the cusps of semilunar valves and forcing them to close

(b) Semilunar valve closed
Properties of Cardiac Muscle Fibers

• Microscopic Anatomy
  
  – Branched
  
  – Centrally located nucleus
  
  – Actin and myosin arranged into sarcomeres
  
  – Sarcoplasmic reticulum and T tubules not well organized
  
  – Cells joined by intercalated discs, heart functions as a syncytium
Properties of Cardiac Muscle Fibers
Properties of Cardiac Muscle Fibers

• Mechanisms and Events of Contraction
  
  – Means of Stimulation
    
    • Some self exciteable (automaticity or autorhythmicity)
  
  – Organ vs Motor Unit Contraction
    
    • Skeletal muscle each motor unit contracts independently
    
    • Entire heart either contracts or not
Properties of Cardiac Muscle Fibers

- Length of Absolute Refractory Period
  - Much longer than skeletal muscle
  - Prevents tetany
Properties of Cardiac Muscle Fibers

- Events in the Contraction of Cardiac Muscle

  • Voltage-regulated Fast Na⁺ Channels open, initiating an action potential

  • Depolarization travels down T tubules resulting in the release of Ca²⁺
    - 20 – 30% of Ca²⁺ needed for contraction comes from outside the cell; this acts as the stimulus to release Ca²⁺ from the sarcoplasmic reticulum
    - For Ca²⁺ to enter the cell depolarization of cell membrane via Na⁺ channels opening, opens slow Ca²⁺ channels
      » Ca²⁺ enters the cell causing Ca²⁺ channels to open in the sarcoplasmic reticulum, releasing Ca²⁺
Properties of Cardiac Muscle Fibers

(a) Membrane potential (mV) vs. Time (ms)
- Action potential
- Plateau
- Tension development (contraction)
- Absolute refractory period

(b) Relative membrane permeability vs. Time (ms)
- Na$^+$ permeability (inward flux)
- Ca$^{2+}$ permeability (inward flux)
- K$^+$ permeability (outward flux)
Properties of Cardiac Muscle Fibers

- **Energy Requirements**
  
  - Possess a large number of mitochondria (needs large amounts of oxygen)
  
  - Cannot function anaerobically for very long, unlike skeletal muscle
  
  - Utilizes multiple fuels – even lactic acid generated by skeletal muscle contraction
Heart Physiology

- **Electrical Events**
  - Autorhythmicity of Cells – important to understand, some cardiac drugs work at this level

![Graph showing electrical events in the heart](image)
Heart Physiology

- Sequence of Excitation

(a) Diagram of heart showing sequence of excitation:
- Superior vena cava
- ① Sinoatrial (SA) node (pacemaker)
- Internodal pathway
- ② Atrioventricular (AV) node
- ③ Atrioventricular (AV) bundle (Bundle of His)
- ④ Bundle branches
- ⑤ Purkinje fibers
- Right atrium
- Left atrium
- Purkinje fibers
- Inter-ventricular septum

(b) Graph showing electrical activity:
- SA node
- Atrial muscle
- AV node
- Ventricular muscle
- Milliseconds
Heart Physiology

- **Modifying the Basic Rhythm: Extrinsic Innervation of the Heart**

![Diagram of heart physiology and neural connections]

- **Key:**
  - Blue: Parasympathetic fibers
  - Green: Sympathetic fibers
  - Red: Interneurons
  - Yellow: Cardiac acceleratory center (sympathetic)
  - Green: Sympathetic trunk
  - Orange: Thoracic spinal cord
  - White: Sympathetic chain ganglion
  - Purple: Medulla oblongata
  - Black: Dorsal motor nucleus of vagus
  - Orange: Cardioinhibitory center (parasympathetic)
  - Blue: Vagus nerve
  - Red: Sympathetic cardiac nerve
  - Pink: SA node
  - Pink: AV node
Heart Physiology

- Electrocardiography

![Diagram of heart physiology with labeled parts: Sinoatrial node, Atrioventricular node, QRS complex, Ventricular depolarization, Atrial depolarization, Ventricular repolarization, P-Q interval, S-T segment, Q-T interval.](image)
Heart Physiology

- **SA node generates impulse; atrial excitation begins**
- **Impulse delayed at AV node**
- **Impulse passes to heart apex; ventricular excitation begins**
- **Ventricular excitation complete**
Heart Physiology

(a)  

(b)  

(c)  

(d)
Heart Sounds

Sounds of aortic semilunar valve are heard in 2nd intercostal space at right sternal margin.

Sounds of pulmonary semilunar valve are heard in 2nd intercostal space at left sternal margin.

Sounds of mitral valve are heard over heart apex, in 5th intercostal space in line with middle of clavicle.

Sounds of tricuspid valve are typically heard in right sternal margin of 5th intercostal space; variations include over sternum or over left sternal margin in 5th intercostal space.
Mechanical Events: The Cardiac Cycle
Cardiac Output

- **Cardiac output is equal to the stroke volume times the heart rate**
  - Stroke volume is the volume of blood pumped by either the right or left ventricle during one ventricular contraction – it equals the end-diastolic volume minus the end-systolic volume

\[
SV = EDV - ESV
\]

\[
70 = 125 - 55
\]

\[
CO = SV \times HR
\]

\[
5250 = 70 \text{ ml/b} \times 75 \text{ b/m} \times (\text{bpm})
\]

\[
CO = 5.25 \text{L/min}
\]
Cardiac Output

- Regulation of Stroke Volume
  - Preload: Degree of Stretch of Heart Muscle (Frank-Starling Law of the Heart) – greatest factor influencing stretch is venous return
  - Contractility – strength of contraction
    - Increased Ca$^{2+}$ the result of sympathetic nervous system
Cardiac Output

- Other chemicals can affect contractility
  » Positive inotropic agents – glucagon, epinephrine, thyroxine, digitals
  » Negative inotropic agents – acidosis, rising K⁺ levels, calcium channel blockers

- Afterload: Back Pressure Exerted by Arterial Blood

- Regulation of Heart Rate
  • Autonomic Nervous System
  • Chemical Regulation
    - Hormones
      » Epinephrine
      » Thyroxine
    - Ions
Cardiac Output

Crisis stressors (exercise, physical or emotional trauma, increased body temperature)

Increased activity of muscular pump and respiratory pump

Increased renal activity (conservation of Na⁺ and water)

Low blood pressure, low blood volume (hemorrhage, excessive sweating)

Increased blood volume

High blood pressure

Chemicals: Bloodborne thyroxine, epinephrine, excess Ca²⁺

(Short-term effects only)

Venous return

Increased contractility of cardiac muscle

Sympathetic nervous system activity

Crisis has passed

Parasympathetic nervous system controls via cardioinhibitory center and vagus nerves

Stroke volume (SV) (ml/beat)

EDV  EDV  ESV

Heart rate (HR) (beats/min)

Cardiac output (CO) (ml/min)

Increases, stimulates

Reduces, inhibits

Initial stimulus

Physiological response

Result
Cardiac Output

- Other Factors
  - Age
    » Infants faster
  - Gender
    » Women faster (Why?)
  - Exercise/Body Temperature

Read Homeostatic Imbalances on your own along with “A Closer Look” on pages 704-705 – If you have questions ASK !!!!