Heart Anatomy

- Approximately the size of a fist

- Location
  - In mediastinum
  - On superior surface of diaphragm
  - Anterior to vertebral column
  - Posterior to sternum
  - 1/3 on right, 2/3 on left

- Enclosed in pericardium, a double-walled sac
Heart Anatomy

(a) Midsternal line
2nd rib
Sternum
Diaphragm
Point of maximal intensity (PMI)

(b) Right lung
Heart

Anterior

(c) Superior vena cava
Left lung
Aorta
Parietal pleura (cut)
Pulmonary trunk
Pericardium (cut)
Apex of heart
Diaphragm
Coverings of the Heart: Pericardium

double-walled sac surrounding heart

- **Fibrous pericardium** (superficial)
  - Protects, anchors, prevents overfilling of heart with blood

- **Serous pericardium** (deep)
  - double layer serous membrane with serous fluid between
  - **Parietal** layer lines internal surface of the fibrous pericardium
  - **Visceral** layer or **epicardium** lines external surface of the heart
  - Separated by fluid-filled **pericardial cavity** - contains serous fluid for lubrication – decreases friction
Pericardial Layers of the Heart

- Fibrous pericardium
- Parietal layer of serous pericardium
- Pericardial cavity
- Epicardium (visceral layer of serous pericardium)
- Myocardium
- Endocardium
- Heart chamber

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Pericarditis

- inflammation of the pericardium
  - Can be caused by infection, MI, or other unknown causes
- Can cause rubbing – very painful
  - Can be heard with stethoscope
- Severe cases can develop into cardiac tamponade
  - Large amounts of inflammatory fluid compresses the heart, limiting its ability to pump blood
  - Treatment: needle into the pericardial cavity to drain off the excess fluid
Layers of the Heart Wall

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

- **Epicardium** (outermost)
  - visceral layer of the serous pericardium

- **Myocardium**
  - Spiral bundles of cardiac muscle cells - held together by elastic & collagen fibers that form a dense network called the *Fibrous skeleton* of the heart

- **Endocardium** (innermost)
  - endothelial layer of the inner myocardial surface that is continuous with blood vessel linings
    - Creates a smooth surface for easy blood flow
Cardiac Muscle Bundles
Layers of the Heart Wall

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

- Four chambers
  - Two atria
    - Separated internally by interatrial septum
    - Coronary sulcus (AV groove) at A-V junction
    - Walls are ridged by pectinate muscles
    - The receiving chambers
      - To right atrium: sup & inf vena cava, coronary sinus
      - To left atrium: R & L pulmonary veins
  - Two ventricles
    - Separated by interventricular septum
    - Walls are ridged by trabeculae carneae; papillary muscles
    - The discharging chambers
      - Leaving R vent: pulmonary trunk
      - Leaving L vent: aorta
Aorta
Left pulmonary artery
Left pulmonary veins
Auricle of left atrium
Left atrium
Great cardiac vein
Posterior vein of left ventricle
Left ventricle

Superior vena cava
Right pulmonary artery
Right pulmonary veins
Right atrium
Inferior vena cava
Coronary sinus
Right coronary artery (in coronary sulcus)
Posterior interventricular artery (in posterior interventricular sulcus)
Middle cardiac vein
Right ventricle

(d) Posterior surface view
(e) Frontal section

- Superior vena cava
- Right pulmonary artery
- Pulmonary trunk
- Right atrium
- Right pulmonary veins
- Fossa ovalis
- Pectinate muscles
- Tricuspid valve
- Right ventricle
- Chordae tendineae
- Trabeculae carnea
- Inferior vena cava
- Aorta
- Left pulmonary artery
- Left atrium
- Left pulmonary veins
- Mitral (bicuspid) valve
- Aortic valve
- Pulmonary valve
- Papillary muscle
- Interventricular septum
- Epicardium
- Myocardium
- Endocardium
Atria of the Heart

- areas that **receive blood** from the vessels
- have characteristic structures:
  - **Auricles** (appendages)
  - **Pectinate** (combed) muscles
  - **fossa ovalis** marks spot where an opening - **foramen ovale** – existed in fetal heart
    - Allowed pulmonary circuit bypass in fetus
- Small thin walls require little contraction
  - gravity helps too
Atria of the heart

- **Right Atrium**
  - deoxygenated blood drain here via:
    - Superior vena cava
    - Inferior vena cava
    - Coronary sinus

- **Left atrium**
  - pulmonary veins bring oxygenated blood from the lungs to here
Ventricles of the Heart

- Largest chambers
  - left vent. is most powerful and larger than right
- discharging chambers of the heart
- **Papillary** (cone shaped) **muscles** projecting into ventricular cavities connected to valve chordae tendineae
- **trabeculae carneae** (ridged) wall muscles
- Right ventricle → pulmonary trunk
- Left ventricle → aorta
Right and Left Ventricles

- Right ventricle
- Left ventricle
- Muscular interventricular septum
Heart Valves

- Pulmonary valve
- Aortic valve
- Area of cutaway
- Mitral valve
- Tricuspid valve

(a) Anterior

Myocardium

Tricuspid (right atrioventricular) valve

Mitral (left atrioventricular) valve

Aortic valve

Pulmonary valve

(b)
Heart Valves

Figure 18.8c, d

- Chordae tendineae attached to tricuspid valve flap
- Papillary muscle
- Opening of superior vena cava
- Mitral valve
- Chordae tendineae
- Tricuspid valve
- Myocardium of right ventricle
- Interventricular septum
- Myocardium of left ventricle
- Pulmonary valve
- Aortic valve
- Area of cutaway
- Mitral valve
- Tricuspid valve

(d)
Heart Valves

- Ensure unidirectional blood flow through heart

- Atrioventricular (AV) valves
  - Prevent backflow into atria when ventricles contract
    - Tricuspid valve (right) – 3 flaps
    - Mitral valve (bicuspid; left) – 2 flaps
  - Anchored to papillary muscles via chordae tendineae

- Semilunar valves
  - Prevent backflow into the ventricles when they relax
    - Aortic semilunar valve (left)
    - Pulmonary semilunar valve (right)
Atrioventricular Valve Function

1. Blood returning to the heart fills atria, putting pressure against atrioventricular valves; atrioventricular valves are forced open.
2. As ventricles fill, atrioventricular valve flaps hang limply into ventricles.
3. Atria contract, forcing additional blood into ventricles.

(a) AV valves open; atrial pressure greater than ventricular pressure

1. Ventricles contract, forcing blood against atrioventricular valve cusps.
2. Atrioventricular valves close.
3. Papillary muscles contract and chordae tendineae tighten, preventing valve flaps from everting into atria.

(b) AV valves closed; atrial pressure less than ventricular pressure
Semilunar Valve Function

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

(a) Semilunar valves 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 valves closed
Valve disorders

- **Incompetent valve**
  - valve does not close completely and backflow of blood occurs

- **Stenosis**
  - valve flaps become stiff and constricts the opening
  - heart has to work harder to pump blood
  - under these conditions valve is usually replaced

- Often the mitral valve
  - Why do you think???
Pathway of Blood Through the Heart

- **Pulmonary circuit:** Right atrium → tricuspid valve → right ventricle → pulmonary semilunar valve → pulmonary arteries → Lungs → pulmonary veins → left atrium
  - Short, low pressure

- **Systemic circuit:** Left atrium → bicuspid (mitral) valve → left ventricle → aortic semilunar valve → aorta → to the body → vena cava → right atrium
  - Long, higher pressure (higher resistance)
Capillary beds of lungs where gas exchange occurs

Pulmonary Circuit

Pulmonary arteries
Venae cavae

Right atrium
Right ventricle

Heart

Systemic Circuit

Pulmonary veins
Aorta and branches

Left atrium
Left ventricle

Oxygen-rich, CO₂-poor blood

Oxygen-poor, CO₂-rich blood

Capillary beds of all body tissues where gas exchange occurs

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Coronary Circulation

- Blood supply to the heart muscle itself
- Accomplished via **right and left coronary arteries** that arise from the aorta
- Blood is drained by the **cardiac veins** into the **coronary sinus** into the **right atrium**
- The heart requires 1/20\(^{th}\) of the blood supply - requires lots of O2
Coronary Circulation: Arterial Supply

(a) The major coronary arteries

- Superior vena cava
- Anastomosis (junction of vessels)
- Right atrium
- Right coronary artery
- Right ventricle
- Right marginal artery
- Posterior interventricular artery
- Anterior interventricular artery
- Left atrium
- Pulmonary trunk
- Left coronary artery
- Circumflex artery
- Left ventricle
Coronary Circulation: Arterial Supply

- **L. coronary artery** → 2 branches supply L heart
  - *anterior interventricular:* supplies interventricular septum, anterior walls of ventricles
  - *circumflex artery:* supplies left atrium, posterior walls of the left ventricle

- **R. coronary artery** → 2 branches supply R heart
  - *Marginal artery:* supplies myocardium of the right side
  - *Posterior interventricular:* supplies apex of the heart & posterior ventricle
    - Merges with anterior interventricular artery (anastomosis)
(a) The major coronary arteries

- Superior vena cava
- Aorta
- Pulmonary trunk
- Left atrium
- Left coronary artery
- Circumflex artery
- Left ventricle
- Anterior interventricular artery
- Right coronary artery
- Right atrium
- Right ventricle
- Right marginal artery
- Posterior interventricular artery
Coronary Circulation: Venous Supply

Returning blood flow from the capillaries is via:

- **Cardiac veins**
- coronary sinus
- right atrium

(b) The major cardiac veins

- Superior vena cava
- Anterior cardiac veins
- Great cardiac vein
- Coronary sinus
- Small cardiac vein
- Middle cardiac vein
Homeostatic Imbalances

- **Angina pectoris**
  - pain due to deficient supply of blood to the myocardium
  - Caused by: stress-induced spasms of arteries, increased physical demands on the heart or arteriosclerosis
  - Cells are weakened

- **Myocardial infarction (MI)**
  - heart attack
  - Prolonged coronary blockage = prolonged lack of oxygen to the heart muscles = cardiac muscle cell death
  - These cells are AMITOTIC; replaced with non-contractile scar tissue
  - May be repairable depending on extent of damage and time
Cardiac Muscle Characteristics

- Striated, branched, interconnected
- Numerous large mitochondria
- Intercalated discs: anchoring junctions between cardiac cells
  - Desmosomes: prevent cell separation during contraction
  - Gap junctions: all ions to pass; electrically couples cells
    - Ensures heart contracts as a unit
- About 1% of cardiac cells have automaticity (are self-excituble)
- Depolarization of the heart is rhythmic and spontaneous
Cardiac Muscle Contraction

- Electrical events of the heart are independent of the nervous system (intrinsic, automaticity)
  - but can be modulated by the autonomic nervous system (extrinsic control)
Cardiac Intrinsic Conduction

- Intrinsic rhythms are modulated by autorhythmic cells found in the nodal tissue
  - These cells have an unstable RMP and will spontaneously depolarize
    - Sinoatrial node (SA node - pacemaker)
    - Atrioventricular node (AV node)
    - Atrioventricular (AV) bundles (bundle of His)
    - Right and left bundle branches
    - Ventricular Purkinje fibers
Cardiac Intrinsic Conduction

1. The sinoatrial (SA) node (pacemaker) generates impulses.

2. The impulses pause (0.1 s) at the atrioventricular (AV) node.

3. The atrioventricular (AV) bundle connects the atria to the ventricles.

4. The bundle branches conduct the impulses through the interventricular septum.

5. The Purkinje fibers depolarize the contractile cells of both ventricles.
Sinoatrial node

- Located on the upper right atrium
- Sinus rhythm is fastest
  - Generates impulses about 75 times/minute
  - “pacemaker” of the heart
- Depolarizes faster than any other part of myocardium
- Causes atrial contractions via gap junctions & stimulates the AV node
Atrioventricular Node

- Lower R. atrium in the interatrial septum
- Receives impulse from SA node > stimulates ventricular contractions by:
  - Stimulating the AV bundle (bundle of His) > R & L bundle branches > Purkinje fibers > Cell to cell transmission via gap junctions to all cells
  - There are direct connections to the papillary muscles—these contract first to support the valve flaps
- [Conduction Animation]
Homeostatic Imbalances

- **Arrhythmias**: irregular heart rhythms
- **Fibrillation**: quivering instead of ordered contraction
  - Can be atrial or ventricular
  - Fibrillation of ventricles most dangerous
- **Defibrillation**: electrical shock to reset circuit
- **Defective SA node (pacemaker)** may have several consequences:
  - **Ectopic focus**: abnormal pacemaker takes over
  - **Extrasystole**: pre mature contractions linked to localized hyper-excitability of tissue
    - can be caused by caffeine or other stimulants
Homeostatic Imbalances

- Defective AV node
  - Results in heart block
    - Partial block: some transmission come through
      - may require a demand rate pacemaker
    - Total block: no transmissions coming through
      - requires a fixed, stationary pacemaker
  - Few or no impulses from SA node reach ventricles
    - Ventricles beat at their slower intrinsic rate
Extrinsic Innervation of the Heart

- Heartbeat is modified by ANS
- Cardiac centers are located in the medulla oblongata
  - Cardioacceleratory center
    - Innervates SA & AV node, heart muscle, and coronary arteries via sympathetic neuron activation
  - Cardioinhibitory center
    - Inhibits SA and AV nodes through parasympathetic fibers in vagus nerves
      - If vagus nerves cut > inc HR by ~ 25 bpm
Electrocardiography

- Electrocardiogram (ECG or EKG)
  - Composite of all action potentials generated by nodal and contractile cells at a given time
    - Leads record electrical impulse
    - Produce deflection waves - depolarization or repolarization
  - Three waves
    - P-wave: depolarization of SA node
    - QRS complex: ventricular depolarization
    - T-wave: ventricular repolarization
Electrocardiography

- Sinoatrial node
- Atrioventricular node
- Atrial depolarization
- Ventricular depolarization
- QRS complex
- Ventricular repolarization
- P-Q Interval
- S-T Segment
- Q-T Interval
Heart Excitation Related to ECG

1. Atrial depolarization, initiated by the SA node, causes the P wave.

2. With atrial depolarization complete, the impulse is delayed at the AV node.

3. Ventricular depolarization begins at apex, causing the QRS complex. Atrial repolarization occurs.

4. Ventricular depolarization is complete.

5. Ventricular repolarization begins at apex, causing the T wave.

6. Ventricular repolarization is complete.
Heart Excitation Related to ECG

- P wave corresponds to depolarization of SA node and atria
- Takes 0.08 sec
- Followed by 0.1 sec delay
Heart Excitation Related to ECG

- P wave corresponds to depolarization of SA node and atria
- Takes 0.08 sec
- Followed by 0.1 sec delay
Heart Excitation Related to ECG

• P wave corresponds to depolarization of SA node and atria

• Takes 0.08 sec

• Followed by 0.1 sec delay
Heart Excitation Related to ECG

- QRS complex corresponds to ventricular depolarization (0.1 sec delay after SA)
- lasts 0.08 seconds
- Atrial repolarization record is masked by the larger QRS complex
- T wave corresponds to ventricular repolarization - 0.16 sec
Heart Sounds

- Heart sounds (lub-dup) are associated with closing of heart valves
  - First sound (S1; lub) occurs as AV valves close and signifies beginning of systole (contraction)
  - Second sound (S2; dup) occurs when SL valves close at beginning of ventricular diastole (relaxed)

- Heart murmurs: abnormal heart sounds
  - Most often indicate valve problems or thin walls
Heart Sounds

- **Aortic valve** sounds heard in 2nd intercostal space at right sternal margin.
- **Pulmonary valve** sounds heard in 2nd intercostal space at left sternal margin.
- **Mitral valve** sounds heard over heart apex (in 5th intercostal space) in line with middle of clavicle.
- **Tricuspid valve** sounds typically heard in right sternal margin of 5th intercostal space.
The Cardiac Cycle

- All events associated with blood flow through the heart during one complete heart beat
  - Systole
    - contraction of heart muscle
  - Diastole
    - relaxation of heart muscle
- Followed by a succession of pressure and volume changes
- Blood flow is controlled by pressure gradients moving from areas of high to low
Phases of Cardiac cycle

- Three main phases to the cardiac cycle
  - Ventricular filling (mid to late diastole)
  - Ventricular systole (isovolumetric contraction)
  - Isovolumetric relaxation (early diastole)

- [VIDEO LINK]
Phases of the Cardiac Cycle

- Ventricular filling – takes place mid-to-late diastole
  - Atrial contraction
  - AV valves are open
  - 80% of blood passively flows into ventricles
    - Remaining 20% delivered with atrial systole
  - Heart blood pressure is low as blood enters atria and flows into ventricles
  - End diastolic volume (EDV): volume of blood in each ventricle at the end of ventricular diastole
Phases of the Cardiac Cycle

- Ventricular systole (contraction)
  - Atria relax; ventricles begin to contract
  - Rising ventricular pressure results in closing of AV valves
  - Isovolumetric contraction phase (all valves are closed)
    - Pressure builds up in the ventricles until the pressure there is greater than the pressure in the arteries
  - In ejection phase, ventricular pressure exceeds pressure in the large arteries, forces SL valves open
  - End systolic volume (ESV): volume of blood remaining in each ventricle
Phases of the Cardiac Cycle

- **Isovolumetric relaxation** – occurs in early diastole
  - Ventricles relax
  - Backflow of blood in aorta and pulmonary trunk closes semilunar valves
    - Causes Dicrotic notch: brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves

- Heart is in a relaxed state through half of the cycle (quiescent period)
Cardiac Output (CO)

- Volume of blood pumped by each ventricle in one minute

- $\text{CO} = \text{heart rate (HR)} \times \text{stroke volume (SV)}$
  - HR: heart beats per minute
  - SV: volume of blood pumped out by a ventricle with each beat (typically 70 ml/beat)
Cardiac Output: Example

- **At rest**
  - \( \text{CO (ml/min)} = \text{HR (75 beats/min)} \times \text{SV (70 ml/beat)} \)
  - \( = 5250 \text{ ml/min (5.25 L/min)} \)
  - If HR or SV goes up so does CO same is true for going down

- There is an average of 5L of blood in an adult, with 5.25L passing through each ventricle per min
  - Maximal CO is 4-5 times resting CO in non-athletic people
  - Maximum CO may reach 35 L/min in trained athletes

- Cardiac reserve: difference between resting and maximal CO; also higher is trained athletes
Regulation of Stroke Volume (SV)

- **SV** = **EDV** - **ESV**
  - **EDV**: end diastolic volume
    - amount of blood collected in a ventricle during diastole
  - **ESV**: end systolic volume
    - amount of blood remaining in a ventricle after contraction

- Three main factors affect SV:
  - Preload
  - Contractility
  - Afterload
Regulation of Stroke Volume

- **Preload**: degree of stretch in cardiac muscle cells before they contract (Frank-Starling law of heart)
  - $\uparrow$ EDV > $\uparrow$ stretch > $\uparrow$ contractility

- **Contractility**: contractile strength at a given muscle length, independent of stretch and EDV
  - caused frequently by extrinsic factors SNS, hormones, drugs
  - $\uparrow$ contractility > $\uparrow$ SV (↓ ESV)

- **Afterload**: pressure that must be overcome for ventricles to eject blood (hypertension increases afterload)
  - $\uparrow$afterload > $\uparrow$ESV OR $\downarrow$afterload > $\downarrow$ESV
Preload and Afterload

(a) Preload
(b) Afterload
If a patient had a HR of 85 and a SV of 70ml what would his CO be? (CO=HR x SV)
- What would happen to CO if either SV or HR went up or down?

If a patient had an ESV of 35ml and an EDV of 105ml what would the SV be? (SV=EDV-ESV)
- What would happen to SV if ESV or EDV went up or down?
Chemical Regulation of Heart Rate

- **Hormones**
  - **Epinephrine**: Released by adrenal medulla during SNS activation
    - Enhances HR and contractility
    - Causes threshold to be reached more quickly
  - **Thyroxine**: Thyroid gland hormone which inc metabolic rate & body heat production
    - Released in large amounts, causes a sustained inc in HR
    - Enhances effect of epinephrine
    - Hyperthyroidism > weakened heart

- **Ions**
  - **Intra- and extracellular ion concentrations (esp Ca & K)**
    - Plasma electrolyte imbalances = heart danger
Intra- and extracellular ion concentrations must be maintained for normal heart function

- **Hypocalcemia**: lower than normal blood Ca levels
  - depresses the heart
- **Hypercalcemia**: higher than normal blood Ca levels
  - increase heart irritability and leads to spastic heart contractions
- **Hyperkalemia**: higher than normal K
  - interferes with depolarization by lowering the resting potential
    - Possible heart block and cardiac arrest
- **Hypokalemia**: lower than normal K
  - causes weak heart beat and arrhythmias
Homeostatic Imbalances:
Variations in Heart Rate

- HR varies with changes in activity, but persistent rate changes signal cardiovascular disease
  - Tachycardia
    - rapid rate, ≥ 100 bpm
    - Causes: elevated body temp, stress, stimulants, heart disease
    - can lead to fibrillation
  - Bradycardia
    - slow rate, ≤ 60 bpm
    - Causes: low body temp, certain drugs, parasympathetic activation
    - being physically fit > heart hypertrophy > increases SV (&...CO)
      - > lowers resting HR with same CO (heart doesn’t have to beat as much)
Homeostatic Imbalances:

Congestive Heart Failure (CHF)

- Progressive condition where CO is so low that blood circulation is inadequate to meet tissue needs

Caused by:

- **Coronary atherosclerosis**
  - Heart deprived of O2 > weakened contractions

- **Persistent high blood pressure**
  - Heart must work harder to maintain SV & CO; weakens muscle

- **Multiple myocardial infarcts**
  - Tissue dies from lack of O2/nutrients; weakens heart

- **Dilated cardiomyopathy (DCM)**
  - Often idiopathic (unknown cause)
  - Ventricles stretch and become flabby
Homeostatic Imbalances: Congestive Heart Failure (CHF)

- The heart is a “double pump” and each side can initially fail independently of the other
  - **Left side**
    - Pulmonary congestion
      - blood backing up into the lungs > pulmonary edema
      - can lead to suffocation
  - **Right side**
    - Peripheral congestion
      - blood backs up at the tissue level > edema in the extremities
      - can lead to tissue hypoxia
Homeostatic Imbalances:
Related Clinical Terms

- Review all clinical terms pg. 709, especially:

  - **Commotio cordis** “concussion of heart”
    - Situation in which a mild blow to the chest causes heart failure & sudden death
      - This occurs when heart is repolarizing – very vulnerable

  - **Mitral valve prolapse**
    - One or more flaps become incompetent
      - may be hereditary

  - **Myocarditis**
    - inflammation of the cardiac muscle layer
      - can result from bacterial infection
Developmental Aspects of the Heart

- Fetal heart structures that bypass pulmonary circulation (because lungs are not operational)
  - **Foramen ovale**
    - connects the two atria
    - after birth this closes and becomes the *fossa ovalis*
  - **Ductus arteriosus**
    - connects pulmonary trunk and the aorta
    - after birth this closes and becomes the *ligamentum arteriosum*
Age-Related Changes Affecting the Heart

- Sclerosis (hardening) and thickening of valve flaps
  - occurs in older individuals most often the mitral valve

- Decline in cardiac reserve
  - sympathetic control goes ↓; more difficult to adjust HR as needed

- Fibrosis of cardiac muscle
  - cells die are replaced by fibrous connective tissue (scar tissue)
  - SV / CO goes down; also can affect conduction in the heart

- Arteriosclerosis (narrowing/hardening of the arteries)
  - as a result of plaque deposits of saturated fats and cholesterol
    - atherosclerosis
  - arteries becoming fibrous: Inc with smoking, stress, inactivity inc risk, diet