I. CARDIOVASCULAR SYSTEM: THE HEART

- transport of blood. Heart is a pump that provides pressure to move blood through arteries to capillaries. Capillaries are where we exchange with tissues. Veins return blood to heart.

* 2 circuits, so oxygenated & deoxygenated blood won't mix: systemic and pulmonary.

* main point: blood flow cannot slow down, or tissues die due to build-up of acids and wastes. The system must maintain enough pressure to maintain blood flow.

- blood pressure can't be too high *(hypertension)*, or the capillaries are damaged.
- blood pressure can't be too low *(hypotension)*, or wastes build up near tissues, pH changes.

- Congestive heart failure = slowed/stopped blood flow (often due to too low blood pressure, or a thrombus/embolism), leading to cell death. Causes: chronic hypertension, arteriosclerosis, damage to heart muscle.
A. Anatomy of the Heart

1. Location and Size: less than a pound in weight. Thoracic cavity.
   - **Location**
     - Thorax between the lungs in the inferior mediastinum
   - **Orientation**
     - Pointed apex directed toward left hip
     - Base points toward right shoulder
   - **About the size of your fist**

2. Coverings and Wall
      - **Pericardium—a double-walled sac**
      - **Visceral pericardium**
        - Next to heart; also known as the epicardium
      - **Parietal pericardium**
        - Outside layer
      - **Serous fluid fills the space between the layers of pericardium**

Pericarditis
b. Myocardium: myocardium, made of cardiocytes, arranged in bundles.

c. Endocardium

Certain *tendinous* structures are made from this endocardium
- Overview of the Anatomy of the 2 circuits: **The heart as 2 separate pumps!**

1. **Systemic Circulation:** blood flows from left side of heart through body & tissues back to the right side of the heart.
2. **Pulmonary Circulation:** blood flows from right side of heart through lungs back to the left side of the heart.

**Blood Flow Through the Heart**

- Superior and inferior venae cavae dump blood into the right atrium
- From right atrium, through the tricuspid valve, blood travels to the right ventricle
- From the right ventricle, blood leaves the heart as it passes through the pulmonary semilunar valve into the pulmonary trunk
- Pulmonary trunk splits into right and left pulmonary arteries that carry blood to the lungs
- This affects the rest of the anatomy:

- Oxygen is picked up and carbon dioxide is dropped off by blood in the lungs
- Oxygen-rich blood returns to the heart through the four pulmonary veins
- Blood enters the left atrium and travels through the bicuspid valve into the left ventricle
- From the left ventricle, blood leaves the heart via the aortic semilunar valve and aorta
3. Chambers and Associated Vessels

- **Right and left side act as separate pumps.**
- **4 chambers : 2 pumps (one for each circuit) and 2 "receiving chambers" called atria.**

![Heart diagram with labeled parts]

a. **Atria** : thin myocardium.

b. **Ventricles** : thick myocardium, pump into arteries.

c. **Superior/Inferior Venae Cavae** : return to the right side (bring deoxygenated blood to the pulmonary pump).

d. **Pulmonary Arteries** : take deoxygenated blood to the capillaries of the lungs.

e. **Pulmonary Veins** : return oxygenated blood to the “systemic pump” on the left side.

f. **Aorta** : take oxygenated blood to the systemic capillaries.
4. Valves: stop back-flow when the ventricles pump. Keeps blood flowing the correct direction.

a. Atrioventricular (AV) Valves

*Make sure the blood isn’t pumped back up into atria when ventricles squeeze.*

Open during heart relaxation and closed during ventricular contraction

Anchored in place by chordae tendineae

i. Bicuspid (Mitral) Valve

ii. Tricuspid Valve

b. Semilunar Valves

*Make sure blood doesn’t fall back into the ventricles after contraction.*

Closed during heart relaxation but open during ventricular contraction

i. Pulmonary Semilunar Valve

ii. Aortic Semilunar Valve

- myocardial infarct (coronary or heart attack). Slowed blood flow, cardiocytes die. Immune attack on damaged tissue. Angina pectoris = common first symptom in women.

B. Physiology of the Heart

- heart beats about once per second. Each beat = contraction of ventricles, which pumps the blood volume (about 6 quarts or 5.2 liters) about 1,000 times per day.

* so: first we must conduct electricity (electrical events of the myocardium), then we must contract (the cardiac cycle).

- as with all muscle tissue, generate an AP causes release of Ca++ which causes the cardiocytes to contract.
- Cardiocytes spontaneously generate APs ... heart will beat by itself, without stimulation of nervous system.

Differences: Ca++ comes from the extracellular fluids, and the cardiocytes only do aerobic respiration (very red tissue = lots of mitochondria).

** Calcium blockers lower the contractility of the myocardium, if it is beating too hard.
1. Conduction System of the Heart

- cardiocytes spontaneously generate APs ... heart will beat by itself, without stimulation of nervous system.

* Occasionally Na+ channels open by themselves, start a depolarization, which leads to an AP.

- Start depolarizations up on the right atrium. Then, bundled-up cardiac tissue, acting like an "internal nervous system", takes the depolarizations to the apex, so the beat will start from the bottom and move upward.

- although all cardiocytes will spontaneously generate an AP, some tissue will do it faster = "pacemaker" for the rest of the myocardium (sets the beat).

i. Sinoatrial (SA) Node (Pacemaker) : although all cardiocytes will spontaneously generate an AP, some tissue will do it faster = "pacemaker" for the rest of the myocardium (sets the beat).
* then, due to gap junctions (intercalated disks under the microscope), AP is passed throughout the myocardium, causing it to act as a unit.

ii. Atrioventricular (AV) Node: “portal” to the ventricles.

* heart block: block movement of depolarizations at the AV node, usually due to scarring.

  ** AV node’s spontaneous generation is enough to maintain life, but can’t get lower than this.

  ** If heart rate gets too low, we put in a pacemaker.

iii. Atrioventricular Bundle (Bundle of His)

iv. Bundle Branches

v. Purkinje Fibers
* Contraction follows a moment later. This coordinates contraction, so the heart will pump from bottom upward, pushing blood into the arteries.
* EKG follows these depolarizations through the myocardium.

- **P wave = atrial depolarization**

- **QRS Complex = ventricular depolarization**
- T wave = ventricular repolarization
2. Neural control (from the outside of the system)

**EXTRINSIC CONTROL**

Nerves of the Autonomic Nervous System:
- Slows down or speeds up heart rate.
- **Parasympathetic (vagus)** slows it down using ACh,
- **Sympathetic** speeds it up, using epinephrine.

3. Problems with Control
- **Tachycardia**—rapid heart rate over 100 beats per minute
- **Bradycardia**—slow heart rate less than 60 beats per minutes

* **Fibrillation**: irregular electrical events, causing uncoordinated pumping, lowered blood flow.

**An infarct is only deadly if it trips the patient into fibrillation.**

* Why start the depolarizations at the top, and make it flow to the apex? This puts a "lag" in the contraction, which gives the ventricles time to fill before pumping starts.

* **Heart block**: block movement of depolarizations at the AV node, usually due to scarring.

**AV node’s spontaneous generation is enough to maintain life, but can’t get lower than this.**

**If heart rate gets too low, we put in a pacemaker.**
C. Heart Contraction

1. Cardiac Cycle and Heart Sounds: the pumping of the ventricles. We listen to the sounds of the valves opening & closing to determine if they are coordinated with each other.

   * both valves must pump at the same time, and the same volume of blood.

   * systole = contraction, diastole = relaxation. Traditionally, we are referring to the left ventricle.

   a. Mid-to-Late Diastole: ventricle completely relaxed. Ventricles are filling.

   b. Ventricular Systole: ventricle contracting. AV valves slam shut, stopping back flow (stopping the ventricles from pumping into the atria).

   c. Early Diastole: Semilunar valves snap shut, stopping back flow of blood back into the ventricles from arteries.

- “lub-dub”: the heart sounds. Lub = AV valves, dub = SL valves.

   * Murmur = irregular heart sounds.

   * Stenosis = turbulence caused by a thickened valve.

   * Incompetent valve: valve doesn’t close tightly.

   * Prolapse (not here in book): valve extends out the wrong way.
2. Cardiac Output: amount of blood pumped by each ventricle in 1 minute.

* We use this to estimate the efficiency of the heart.

* Cannot be measured directly, so we use the following equation:

\[ \text{CO} = \text{SV} \times \text{HR} \]

\( \text{SV} = \) stroke volume = amount pumped by a ventricle per beat

\( \text{HR} = \) heart rate, as determined by pulse

* resting CO is about 5250 ml/min, which is about equal to blood volume (that is, your heart pumps about you total blood volume of 5 liters every minute).

* if this number doesn’t change much, why do we care? Our test of efficiency is the amount you can increase CO if your system needs to (exercise).

  - notice: by increasing either SV or HR, we will increase CO. However, increasing HR by itself won’t work, because it lowers the amount of time we have to fill the ventricles. This is why long-term exercise leads to a lowered HR.

a. Regulation of Stroke Volume: anything that increases SV increases CO.

  - Starlings Law: the amount of stretch of the cardiac muscle cells affects SV.

    * The more the cardiac muscle is stretched before it pumps, the harder it will pump!

    * Best way: increase venous return.

      - Increased activity = increase muscles, which “squeezes” blood back to the heart (the “muscular pump”).

      - Also, lowered HR!!

    * also, certain hormones (epinephrine & norepinephrine) and drugs.
b. Regulation of Heart Rate: autonomic innervation, along with some hormones and drugs. However, ends up lowering venous return, so HR doesn’t increase CO without also increasing SV.

* Increasing HR. Sympathetic nerves innervate the SA and AV nodes.

  ** Epinephrine mimics this effect.

  ** Thyroxine.

  ** Hypercalcemia (calcium), hyperkalemia (potassium) & hypernatremia (sodium).

* Deceasing HR: Parasympathetic innervation (vagus). Under normal resting conditions, vagus is inhibiting HR.

  ** Hypocalcemia, hypokalemia and hyponatremia.

  ** Digitalis is a drug that lowers Ca++ permeability of cardiocytes.
II. CARDIOVASCULAR SYSTEM: BLOOD VESSELS

- vascular system = enclosed, in order to prevent blood loss. Any blood that is lost from vessels is picked up by lymphatics or lost via evaporation.

- arteries take blood away from the heart. Veins return it to the heart. In between, capillaries provide the surface area needed for diffusion with the tissues.

  * arteries branch into smaller arterioles, which branch into capillary beds, which converge into larger venules, which converge into veins, which lead to the atria of the heart.

  * the entire system is lined on the inside with the endothelium, a special epithelial layer.
A. Microscopic Anatomy of Blood Vessels

- layers they all have:
  
  a. Tunica Intima: endothelium + loose connective tissue. Next to lumen. Lowers friction, participates in clotting (see blood chapter). In capillaries, becomes the diffusion surface.

  b. Tunica Media: muscular + elastic tissue. Sympathetic innervation. Vasodilation = relaxing this layer, vasoconstriction = contracting this layer. Thicker in arteries.

  c. Tunica Externa: fibrous connective tissue that supports the vessel. Thicker in veins, so they don't collapse when pressure drops.

- veins have specializations that help get blood back to the heart:
  
  a. They are deep in muscles (muscular pump)

  b. They have “venous valves”, stopping blood from flowing downward in between pumps of the heart.

  * varicosities ("varicose veins") = break-downs in the valves.

  c. Thick tunica externa = they don't collapse.

  d. They are larger diameter than arteries = lower pressure.

- capillaries are found in capillary beds (not in book), which can be opened or closed. Tissues with capillary beds are “Vascularized”.

\[\text{Diagram of blood vessels with varicosities marked.}\]
B. Gross Anatomy of Blood Vessels - **DONE IN LAB**

1. Major Arteries of the Systemic Circulation
2. Major Veins of the Systemic Circulation
3. Special Circulations

C. Physiology of Circulation

1. Basics.

   - **NOTE:** the heart does not pump blood all the way through the system.

   * If it tried, the capillaries would blow out!

   * Also, blood flow would stop while we waited for the ventricles to fill for the next beat!

   - instead, the system maintains a “blood pressure gradient”, from high to low, that keeps blood flowing.

   ![Pressure vs. Blood Vessel Diagram](image)

   * Blood Pressure (BP): The heart forces blood into the arteries, which stretch out, and act as a “bellows”, while the heart relaxes and begins filling for the next beat.
Blood pressure is measured in mmHg (millimeters of mercury).

* Arterial Pulse: the arteries are "pulsatile" (have a pulse). That is, they have 2 pressures (highest = systolic, lowest = diastolic).

** Pulse points: points where we feel the pressure applied.

* normal systolic: 95 - 140 mmHg
* normal diastolic: 70 - 85 mmHg
* always reported as systolic/diastolic (e.g.: 120/80 mmHg)
* auscultatory method of determining blood pressure.
* hyper- versus hypotension.
- the “steepest” part of the gradient is between the arterioles and the venules. This keeps blood flowing through the capillaries.

* as the blood passes through the capillaries, a lot of pressure is lost (“Peripheral Resistance” or PR).

** The small gradient between veins and the heart is not enough for venous return to the heart. This is why the veins need the adaptations to return blood (see earlier section).

* if blood flow is too fast, not enough time for diffusion with the tissues. If blood flow is too slow, wastes and acids build up, leading to tissue death.
2. Effects of Various Factors on Blood Pressure

a. Peripheral Resistance

- anything that increases/decreases PR or increases/decreases CO greatly affects the system by increasing/decreasing BP.

* gaining weight adds capillaries, which increases PR

* decrease elasticity of arteries loss of elastin with aging and arteriosclerosis

* break down of autonomic nervous system with aging (see below)

* increase viscosity by increasing hematocrit

* increased/decreased blood volume by retaining/losing water from blood

* increasing or decreasing vasoconstriction / vasodilation

b. Neural Factors: The Autonomic Nervous System

- symapthetic only.  Vasoconstrictor causes an increase in PR, and an increase in HR. Controlled by medulla of brain.

* example: stand up = lower pressure as gravity pulls on blood.  Pressoreceptors send signal to brain, start vasoconstriction reflex. Individual turns pale.

* blood loss through hemorrhage causes same result.

c. Renal Factors: The Kidneys

- control blood pressure by regulating water loss.

* diuretics: any hormone/chemical that increases water loss (putting it into the urine = less in blood, decreasing BP).

* anti-diuretics: hormone/chemical that conserves water in blood (= concentrate urine).
* Renin: hormone produced by kidney cells. If BP drops, renin is released, which causes angiotensin II to be formed (liver protein that is a vasoconstrictor) and aldosterone to be released by adrenals (increases sodium & water conservation). Blood volume and pressure rises.

d. Temperature: cold causes vasoconstriction, heat causes vasodilation.

e. Chemicals:

i. epinephrine increases BP in many ways (increase HR & vasoconstriction).

ii. Nicotine is a vasoconstrictor.

iii. Alcohol decreases BP by 1) vasodilation and 2) inactivating the main hormone that causes the kidney to conserve water (therefor, it is put into the urine).

* inactivates ADH (antidiuretic hormone, released by the posterior pituitary).

f. Diet: controversy over effects of sodium & fats in diet. Protein intake increases BP and increases water loss in kidneys. In general: if kidneys are working, these substances probably affect BP long-term.

3. Variations in Blood Pressure

- race, age, sex, mood, posture, position, etc. affect BP.

- hypotension: systolic below 100 mmHg. Rare in healthy individual; usually diet-related. Happens with age.

  * orthostatic hypotension. Slow-down in sympathetic response. Stand up = BP drops, fainting occurs. Also, hot showers, etc. will cause this effect, as vasodilation occurs.

- hypertension: more than 100 causes. Systolic over 140 for any length of time.

  * chronic = increase in PR. Causes an enlargement in myocardium, as heart works harder. Myocardial infarct, stroke, kidney failure as capillaries are damaged.
4. Capillary Exchange

- Movement in/out of capillaries, over the endothelial surface or between cells.

* Diffusion, osmosis, exocytosis, endocytosis.

* Concentration gradients must be maintained, which is why blood flow is so important.

* Wastes move out of interstitial fluids into blood, nutrients move opposite direction.

* Areas where filtration is important have lots of intracellular clefts. Special capillaries are called “Fenestrated capillaries”. Kidneys, intestines, etc.

  ** fenestration = pore.

* Increase movement of ions, water into tissues = edema. Increase movement of ions/water into capillaries = dehydration.

  ** Chemicals increase/decrease permeability of capillary walls, such as HISTAMINE

** Lymphatics return water to the veins.