CARDOVASCULAR SYSTEM: The Heart

I. THE HEART & BLOOD FLOW – Functional Anatomy of the heart

- CARDIOVASCULAR SYSTEM - heart + blood vessels.

- HEART = muscular pump; keeps blood CONSTANTLY moving through system.

  * Recall from the Blood Chapter:

  Blood Flow: In order to understand heart anatomy, it helps to understand BLOOD FLOW; that is, the flow of blood through the circulatory system.

  * Oxygenated blood: high oxygen, low CO\(_2\) (always red on diagrams)

  * Deoxygenated blood: low oxygen, high CO\(_2\) (always blue on diagrams)

  * Arteries: take blood away from the heart. May carry oxygenated or deoxygenated blood.

  * Veins: carry blood towards the heart. May carry oxygenated or deoxygenated blood.

- Ventricle = the heart’s pump.

  * Myocardium: heart muscle

- HEART = 2 muscular pumps (ventricles); keeps blood CONSTANTLY moving through system at a perfect rate for the tissues to be supplied with O\(_2\) and not have CO\(_2\) build up.

  * It maintains a pressure within the cardiovascular system that is sufficient to keep blood moving past the tissues, but not high enough to burst the narrow-walled capillaries.

- We will look at blood vessels and the heart together, as heart anatomy is determined by 3 criteria that must be met for the cardiovascular system to get its job done:

  A) Criterion #1: Recall from the blood chapter that blood has several major functions. Two of these functions must be kept isolated from each other:

  **Functional consideration #1.** Take oxygenated blood to the body’s cells, where it will be used in cellular respiration (aerobic):

  \[ \text{O}_2 + \text{Glucose} \rightarrow \text{ATP} + \text{CO}_2 + \text{H}_2\text{O} \]

  * This occurs in the Systemic Circuit

  **Functional consideration #2.** Take deoxygenated blood to the lungs, where the CO\(_2\) will be disposed of.

  * This occurs in the Pulmonary Circuit
- The 2 circuits are continuous with each other. This allows blood to easily be oxygenated right after it has been deoxygenated.

**The heart is not a single pump, but two separate pumps. The left ventricle pumps blood into the systemic circuit, while the right ventricle pumps blood into the pulmonary circuit.**

- The pumps must pump at the same time, so the blood returning from one circuit has somewhere to go, and we don’t get back-pressure!

**We will have a special built-in controlling structure, called the Intrinsic Conduction System, to assure that the pumping is coordinated. See later section.**

B) **Criterion #2:** Oxygenated & Deoxygenated blood must not mix!

- These 2 circuits must be kept isolated from each other, or oxygen and carbon dioxide will diffuse down their concentration gradients, lower the oxygen content and increasing the carbon dioxide content of blood going to the Systemic Circuit.

1. So, we cannot have backflow; blood flow must be one-way. **In order to prevent backflow, we have valves:**

   The pumps move blood into arteries, which take blood to the body’s capillaries. To understand the rest, you must realize that the arteries help to move blood.

   As blood is pumped into the arteries, they swell, and act like a bellows, moving blood through the capillaries while the ventricles relax, waiting to fill for the next pump!
2. Also, there is an interventricular septum.

![Interventricular Septum](image)

**C) Criterion #3:** These two circuits must have the same volume of blood pumped into them, but at different pressures.

- We must always be oxygenating about the same amount of blood as we are deoxygenating, or we will lose oxygen in the system, and build up CO₂.

But, the lungs are closer to the heart, and fragile. We must pump blood at a higher pressure to the Systemic Circuit. **The left ventricle (space) is the same size as the right, but has a much thicker myocardium.**

Left myocardium much thicker than the right myocardium (see above diagram).

**- SUMMARY of Functional Anatomy:**

Follow the numbers on the diagram to the right, recalling:

* Left side = high in [O₂], low [CO₂], high pressure.

* Right side = low in [O₂], high [CO₂], low pressure.
II. **GROSS ANATOMY**

- Heart = size of fist, found in the MEDIASTINUM
  * Enclosed in the PERICARDIUM (serosal membrane surrounding the heart).

  * 2 layers: VISCERAL & PARIETAL PERICARDIA; w/PERICARDIAL CAVITY between them.

  * EPICARDIUM: another name of the visceral pericardium.

  * PERICARDITIS - inflammation of sac (bacterial pneumonia, etc.).

- Decrease in production of serous fluid in pericardial cavity, heart rubs against sac; can be heard with a stethoscope.

- Increase in production of fluid increases compression against heart = CARDIAC TAMPONADE; liquid removed by inserting syringe.

- MYOCARDIUM - muscle of heart. Contains the heart muscle cells (cardiocytes, myocardiocytes)

- ENDOCARDIUM - "Inside heart" - lines inner chamber; continuous w/ endothelial lining of blood vessels.

  * Forms the Blood-Heart barrier, and plays an important role in regulating the functioning of the myocardium!
4 chambers:
* 2 ATRIA (w/ outer AURICLES) - receiving chambers; DO NOT pump (much), so thinner myocardium.
* 2 VENTRICLES ("pumps") - most of mass; = 2 separate pumps, pumping into 2 separate circuits. Note thick myocardium around ventricles, and interventricular septum.

- Between the atria & ventricles = ATRIOVENTRICULAR (AV) VALVES = stop backflow into the atria when the ventricles pump.
  * left AV = BICUSPID or MITRAL VALVE.
  * right AV = TRICUSPID VALVE.
  * CHORDAE TENDINEAE = tendons attaching valves to myocardium; prevent valves from flipping backwards when ventricles pump. Attach to papillary muscles.

- Between the ventricles & arteries = SEMILUNAR VALVES (SL VALVES) = stop backflow into the ventricles when the ventricles relax.
  * PULMONARY and AORTIC SL VALVES.
- 2 special regions we will discuss:
  * APEX – “pointed” area.
  * BASE – Where blood vessels enter/leave.

- Myocardium supplied by its own arteries: LEFT & RIGHT CORONARY ARTERIES & VEINS, and their branches.

* MYOCARDIAL INFARCT - Prolonged coronary blockage. The medical term for an event commonly known as a “HEART ATTACK” or “CORONARY”.

* Infarct – tissue death due to a lack of oxygen.

It happens when blood stops flowing properly to part of the heart and the heart muscle is injured due to not getting enough oxygen. Usually this is because one of the coronary arteries that supplies blood to the heart develops a blockage. The event is called "acute" if it is sudden and serious.

Cell death leads to scar tissue formation, which leads to permanent damage (heart cells are generally thought to be amitotic as adults, so they can’t replace themselves)

* ANGINA PECTORIS (“strangling chest”) - chest pain caused by a decreased blood delivery to the myocardium; no cells die.

We will return to this after ECG discussion.
III. HISTOLOGY

- Cardiac muscle is similar to skeletal muscle tissue. Striated, with sarcomeres, sliding filament theory applies. Review sliding filament theory image to the right.

*Sequence of electrical and contraction events = similar to skeletal muscle tissue:

1. If reach threshold, influx of Na+ ions from extracellular fluid initiates a + feedback loop; produces an AP by opening voltage-regulated Na+ and K+ channels.

2. AP reaches T-tubules (not shown). SR releases Ca++ into sarcoplasm. SR re-pumps Ca++ back into itself.

3. Ca++ diffuses to troponin, increase x-bridge activity, followed by the “power stroke”. Myofilaments slide, shortening sarcomeres. ATP is used.

4. Contraction is maintained as long as there is Ca++ in the sarcoplasm (i.e., as long as there is an AP on the sarcolemma), and ATP is present.

- However, with some differences:

A) Contraction and Energy Requirements

- Some differences between the 2 cell types:

1. Cardiac cells are fat, short, branched, interconnected, uni- or dual-nucleate.

2. Cardiac cells are arranged in circular bands around ventricles. Contraction leads to “squeezing of the ventricles”.

3. AEROBIC RESPIRATION only.

(see image “Tetany and fatigue are bad in cardiac muscle tissue” on next page)

* Characteristic: do not go into tetany, prolonged contraction w/out fatigue.

* Lots of mitochondria, numerous myoglobin molecules, & a good blood supply.

* Intracellular space filled with matrix called ENDOMYSIUM with lots of capillaries.

* Can use glucose, fat, etc., even lactic acid produced by skeletal muscle.
Tetany and fatigue are bad in cardiac muscle tissue:

By Dr. S. Giord, Anton Becker; Own work

B) Excitability and Contraction of Myocardioocytes

* Excitable, just like skeletal muscle cells. As with skeletal muscle tissue, contraction occurs a moment later.

But, they exhibit spontaneous depolarization.

Depolarize without neurotransmitter or any other external stimulus. Occasionally, PM channels open by themselves, allowing ions to enter and depolarize the membrane. There are no "sub-threshold stimuli”; they always lead to an AP.

* INTERCALATED DISKS - cells interlock w/ DESMOSOMES & GAP JUNCTIONS, allowing ions to pass freely from cell to cell; AP's pass from cell to cell. Myocardium contracts as a single unit!!!!

- AUTORHYTHMIC REGION - small region that exhibits SPONTANEOUS DEPOLARIZATION (depolarizes without neurotransmitter contacting plasma membrane) = "PACEMAKER" of the heart.

* All myocardioocytes can do this, but we will see they differ in the rates that they spontaneously depolarize. The cells that depolarize the fastest cause the others to depolarize, so they set the "pace".

**Gap Junctions:** Protein tubes that run between cells. Act as an open passageway.
- There are some differences in how depolarizations and contractions happen:

1. Due to the presence of gap junctions, the ALL OR NOTHING LAW applies to the entire organ instead of just a single cell.

2. Length of absolute refractory period (see below) is lengthened to about the same as the contraction period, preventing tetany.

- Absolute Refractory Period is the time during which an AP is impossible if there is another stimulus. Basically, it is the time that an AP is occurring on a cell.

- How lengthened? Ca++ also enters the cell when depolarization occurs, as Ca++ gates are opened on the PM.

- This "draws out" the absolute refractory period as Ca++ is a large "double positive" ion that moves slowly, dragging out repolarization.

**Graph of the AP (spontaneous) of a myocardiocyte**

- Sometimes, the heart is contracting too hard. Its dependence on extracellular Ca++ can be used to lessen the contraction strength.

  * Why would a heart be contracting too hard? Hypertrophy of cardiac muscle tissue, and loss of elasticity of the arteries. Person in danger of stroke or heart attack by destroying capillaries.

  * Enlargement of the heart (cardiomegaly): Can be "hypertrophic" (enlarged cells) or enlarged, stretched-out ventricles (more common).

  * Like skeletal muscle cells, myocardiocytes become fairly amitotic. But, capable of increasing size & width by increasing myofilaments if exercised ("made to work hard").

  * Left ventricular enlargement (hypertrophy) is usually caused by high blood pressure. The heart works harder, and the muscle responds by enlarging the cells.

  * **Calcium Blockers** – a family of drugs that lower the permeability of the PM to Ca++. They can be used to lessen contraction of heart without affecting the contraction of skeletal muscle.