

The Heart

I. Heart anatomy.

A. Size and location.

- about the size of a fist, 250-300 grams.
- located within the mediastinum (medial cavity of the thorax).

B. Coverings of the heart.

- enclosed by double walled fibroserous sac, pericardium.
- 1. Fibrous pericardium: tough, dense connective tissue; protects the heart and anchors it to the surrounding structures, also prevents overfilling.
- 2. Serous pericardium: parietal pericardium, lines the internal surface of fibrous pericardium; visceral pericardium, is the outer heart surface.

C. Layers of the heart wall.

1. Epicardium: visceral pericardium.
2. Myocardium: forms the bulk of the heart wall; mostly composed of cardiac muscle fibers arranged in spiral, circular bundles; connective tissue fibers interspersed between cardiac cells, form fibrous skeleton of heart.
3. Endocardium: made up of endothelium.

D. Chambers and associated large vessels.

- 4 chambers: 2 atria and 2 ventricles; separated by interatrial septum and interventricular septum.
- the atria are relatively small.
- the right ventricle (RV) forms most of the anterior surface.
- the left ventricle (LV) forms most of the inferior-posterior aspects and the apex.
- 1. Atria: functions as receiving chambers, conduits for blood into ventricles.
- posterior walls are smooth; anterior walls are rigged because of muscle bundles.
- atrial contraction contributes very little to ventricular filling.
- blood enters the atria from the superior vena cava, the inferior vena cava, and the coronary sinus.

2. Ventricles: the discharging chambers, the pumps.

- the internal walls have muscle ridges, trabeculae carneae, and muscle bundles, papillary muscles.
- RV pumps blood to the pulmonary circuit; LV pumps blood to the systemic circuit.

E. Pathway of blood through the heart.

1. Pulmonary circuit: blood vessels that carry blood to and from the lungs; gas exchange function.

- RA --> RV --> pulmonary arteries --> lung capillaries --> pulmonary veins --> LA

2. Systemic circuit - blood vessels carrying blood to and from body tissues.

- LA --> LV --> aorta --> elastic arteries --> muscular arteries --> capillaries --> venules --> veins
- note that equal volumes of blood are flowing in the pulmonary and systemic circuit.

F. Heart valves: connective tissue structures that allow unidirectional flow of blood through the heart.

1. Atrioventricular valves (AV).

- found between the atria and ventricles, the right or tricuspid; and left or bicuspid (mitral).
- the valve flaps are anchored to papillary muscles by collagen cords, chordae tendineae
- the heart is relaxed during atrial filling, the AV valves open, blood flowing from the atria to ventricles. Ventricles contract increasing internal pressure and forcing the AV valves to close, preventing entrance of blood into atria during ventricular systole.

2. Semilunar valves (SLV).

- found between RV and pulmonary trunk, and between LV and aorta.
- prevent backflow of blood into the ventricles during ventricular relaxation.
- during rapid filling, semilunar valves are closed; ventricle contract.
- when ventricular pressure is greater than atrial pressure SLV open.
- as ventricles relax, arterial pressure is greater than ventricular pressure and as blood begins to move back, is caught in the cusps, and the valves close.
- the opening and closing of valves explains the lub/dub sounds heard from the heart.

G. Coronary circulation.

- the heart is completely dependent on aerobic metabolism, and NEEDS oxygen.
- functional blood supply of the heart is provided by the right and left coronary arteries; these stem from the base of the aorta and encircle the heart at the atrioventricular groove.
- coronary arteries --> coronary veins --> coronary sinus --> right atrium.
- blockage of coronary artery causes angina pectoralis and/or myocardial infarctions.

II. Properties of cardiac muscle fibers.

A. Microscopic anatomy.

- striated, contractions occur via sliding filament.
- unlike skeletal muscle, cardiac cells are short, branched, interconnected, and have 1-2 nuclei.
- intercellular spaces are filled with connective tissue.
- skeletal muscle cells are interdependent structurally and functionally.
- cardiac muscle cells are structurally and functionally connected through intercalated disks (gap junctions); form a functional syncytium since all cardiac fibers are electrically coupled.
- many mitochondria are present in cardiac muscle; sarcomeres present.
- T-tubules at Z-lines, weakly developed SR.

B. Energy requirements.

- completely dependent on oxygen for metabolism; aerobic respiration; cardiac muscle flexible in terms of fuel source; glucose; fat and lactic acid can be metabolized.

C. The action potential in a cardiac cell.

- heart contraction caused by action potentials (AP) sweeping across the cardiac cell membranes.
- 1% of cardiac fibers are autorhythmic, special ability to depolarize spontaneously.
- once an AP is started, it is propagated from cell to cell due to functional syncytium.
- sequence of ionic events: electrical activity --> contraction:

1. $\text{Na}^+/\text{Ca}^{++}$ ions from adjacent cell pass across the gap junction; RMP less negative; triggers the opening of voltage gated Na^+ channels, rising phase of AP; these quickly close.

2. Drastic change in voltage causes opening of voltage gated Ca^{++} channels (slow channels); calcium ion permeability increases; simultaneously, potassium channels close completely and potassium permeability decreases; these events produce plateau phase.

3. Recall sodium gates are closed; slope of plateau decreases, calcium gates close, potassium gates open and there is an outflux of potassium, repolarization occurs; Ca^{++} ions are pumped back into the SR and extracellular space.

D. Excitation contraction coupling.

- depolarization --> wave --> opening calcium channels --> calcium influx --> calcium released from the SR, sliding filament.

E. Functional differences between skeletal muscle and cardiac muscle.

1. All or none law: in skeletal muscle applies to the single cell/motor unit; in cardiac muscle applies to organ.

2. Means of stimulation: each skeletal muscle cell must be independently stimulated to contract by nerve endings; in cardiac muscle, 1% of cells are self excitable and initiate AP's, electrical activity is passed to other cells.

3. ARP length in skeletal muscle cells is 1-2 ms; ARP ends before muscle develops peak tension, twitches can summate and tetanus can occur.

- ARP length in cardiac muscle cells is 250 ms; the muscle has developed peak tension by the time ARP ends, thus no twitches can summate.

III. Heart physiology.

A. Electrical events.

- ability of cardiac muscle to depolarize and contract is intrinsic; innervation only alters the basic rhythm.

- basis of independent, coordinated activity of the heart:

- autorhythmicity of some cardiac cells.

- conduction system in heart -- composed of noncontractile cardiac cells.

- electrical coupling of the cardiac muscle cells.

1. Autorhythmicity of cardiac cells.

- 1% of cardiac cells are autorhythmic (depolarize spontaneously).

- have unstable resting potentials, pacemaker potentials.

- mechanism:
- pacemaker potential results from gradually reduced potassium permeability, while sodium permeability goes unchanged; RMP gradually becomes less negative (prepotential).
- when threshold is reached voltage gated calcium ion channels open, causing massive calcium ion influx, depolarization; this leads to opening of voltage-gated potassium ion channels, increased potassium permeability and repolarization; once repolarization is complete, voltage-gated potassium channels close.
- autorhythmic cardiac cells are localized in sinoatrial node, atrioventricular node, atrioventricular bundle, right/left bundle branches, and ventricular walls (Purkinje fibers).
- in normal situations the quickest rate of depolarization is that of the SA node - sinus rhythm
- intrinsic conduction system:
 - a. SA node.
 - b. AV node.
 - c. AV bundle
 - d. right/left bundle branches.
 - e. Purkinje Fibers.
- intrinsic conduction system coordinates manner in which electrical activity is passed from the atria to ventricles.

Disorders:

- arrhythmias: uncoordinated atrial and ventricular contractions.
- fibrillation: condition of very rapid out of phase ventricular contractions.
- ectopic focus: SA node dysfunction causes abnormal pacemaker to appear and assume control of the heart rate; or the AV node takes over (reduces heart rate to ~40-60 beats/min).
- heart block: damage to the AV node causes inability to transmit impulses from atria to ventricles.

2. Modifications of autorhythmicity.

- the ANS via the cardioaccelerator (CA) and cardioinhibitory (CI) centers can modify the activity of the intrinsic conduction system.
 - CA: sympathetic fibers, increases HR and force of contraction (contractility).

- CI: parasympathetic fibers, decrease HR.

3. Electrocardiography.

- recording of electrical activity of the heart monitored from electrodes on the surface of the skin.
- bipolar leads: measure voltage difference between two points of the body.
- unipolar leads -- measures electrical activity of the heart as seen from one point in the body.
- typical EKG:
 - P-wave: atrial depolarization.
 - QRS complex: ventricular depolarization.
 - T-wave: ventricular repolarization.
 - P-R interval: beginning of atrial excitation to beginning of ventricular excitation (beginning of P to end of R)
 - Q-T interval: beginning of ventricular depolarization, through ventricular repolarization (beginning of Q to end of T).

B. Mechanical events.

- cardiac cycle: all events associated with flow of blood through the heart during one complete heartbeat.
- blood flow through the heart is controlled entirely by pressure gradients; blood flows from areas of high pressure to areas of low pressure through any available openings.

1. Period of ventricular filling.

- atrial pressure is greater than ventricular pressure, therefore the AV valves open.
- aortic pressure is greater than ventricular pressure, therefore SL valves closed.
- atria provide conduit to blood entering ventricles; ventricular volume increases and atrial pressure constant.
- P wave is followed by atrial systole, remaining 30% of blood is delivered to the ventricles.
- produces increased ventricular volume along with a small increase in atrial pressure.
- atria begin to repolarize while ventricles depolarize (QRS begins).

2. Isovolumetric contraction/ejection.

- ventricular systole begins.
- ventricular pressure rises; when it is greater than atrial pressure, the AV valve closes.
- ventricles become closed chambers (the SLV are still closed).
- ventricles keep contracting; the ventricular pressure increases-- note that this is an isovolumetric contraction and there is no net change in ventricular volume.
- when ventricular pressure is greater than aortic pressure, the SL valves open, and blood is ejected, period of ejection.
- T wave; ventricular repolarization.

3. Isovolumetric relaxation.

- ventricular pressure decreases.
- when ventricular pressure is less than aortic pressure the SLV closes causing a temporary increase in aortic pressure.
- recall that the AV valves are still closed (ventricular pressure is still greater than atrial pressure), the ventricles are again closed chambers.
- relaxation continues, ventricular pressure decreases, isovolumetric relaxation.

4. Filling and diastasis.

- during ventricular systole, the atria have been in diastole slowly filling with blood, atrial pressure increases.
- when atrial pressure is greater than ventricular pressure the AV valves open, rapid filling occurs.

C. Cardiac output (CO).

- the amount of blood pumped by the heart per minute; highly variable; $CO = HR \times SV$.
- stroke volume (SV) is the amount of blood pumped by the ventricle in one beat.

1. Regulation of stroke volume.

- $SV = \text{End Diastolic Volume (EDV)} - \text{End Systolic Volume (ESV)}$.
- EDV is determined by the length of ventricular diastole and venous pressure.
- ESV is determined by atrial blood pressure and force of ventricular contraction.
- the three most important factors affecting SV are preload, contractility, afterload.

a. Preload (on EDV).

- Starling's law of the heart: the greater the stretch placed on cardiac muscle, the more vigorous the contraction.
- can increase stretch by increasing EDV.

b. Effects of contractility (on ESV).

- contractility is an increase in force of contraction independent of muscle stretch and EDV.
- can be changed by any factor that influences calcium permeability of cardiac cells: sympathetic stimulation , drugs.
- positive inotropes: factors that increase contractility.
- negative inotropes: factors that decrease contractility

c. Effects of afterload (on ESV).

- in normal individuals, afterload does not limit SV; however, in individuals with high blood pressure, it does.

2. Regulation of heart rate (HR)

- heart under vagal tone (parasympathetic).

a. positive chronotropes: factors that increase the rate at which potassium permeability spontaneously decreases in SA node cells

i. sympathetic stimulation.

ii. epinephrine, thyroxine.

iii. ions.

b. negative chronotropes: factors that decrease the rate at which potassium permeability spontaneously decreases in SA node cells.

i. parasympathetic stimulation.