Physiology Exam 10 Study Guide

Heart Contraction Pattern Continued…

-**Purkinje fibers** (PF) – depolarize ventricle's cells

-Work from bottom to top → squeeze from bottom up b/c outlets are up (LV to aorta, RV to pulmonary artery) – needed to get proper blood flow

-External measure: Electrocardiogram (EKG) – pattern of electrical activity being registered

-Y axis = overall electrical charge (NOT V_m of a single cell like in APs)

-X axis = time

-Electrical Activity:

1. **P wave** – when first contraction is occurring → atrial depolarization

-Top-down, pushing blood into ventricle

*Delay due to hiding in bundles of His

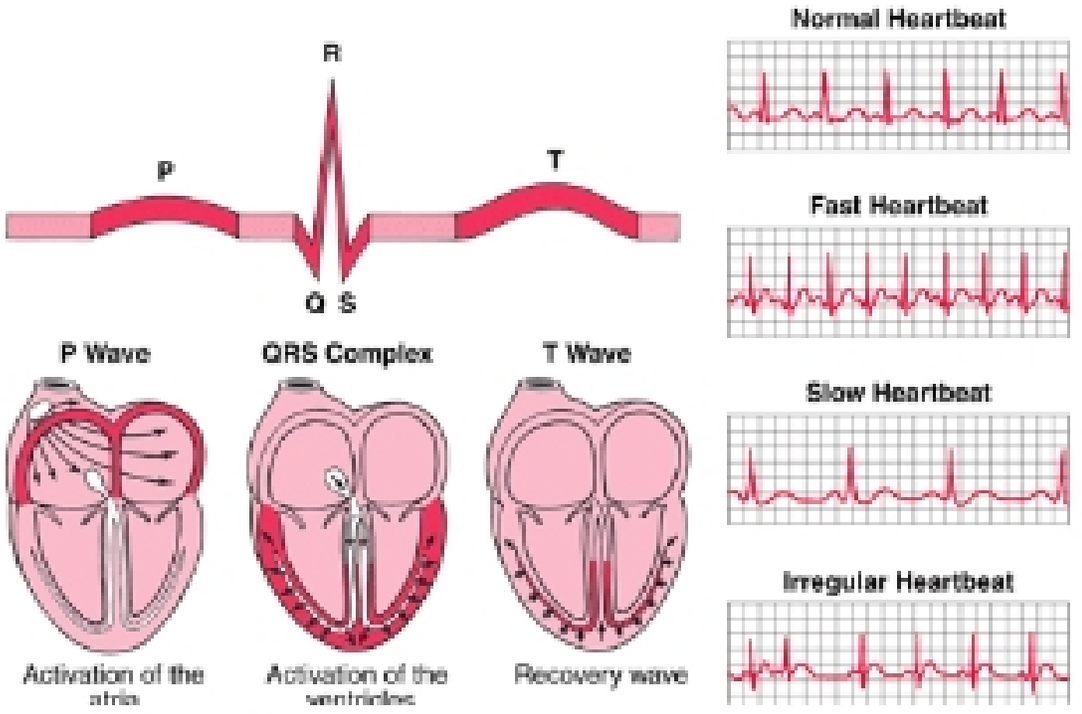
2. **QRS complex** – Ventricular depolarization

-Ventricles contracting from bottom-up (also obscures atrial repolarization)

3. **T wave** – Ventricular repolarization → ends contraction in ventricles

**What about atrial relaxation?

-Very small, times out in QRS system (it obscures atrial repolarization also occurring during this time)



-*Must finish one cycle before next one can start*

-Sounds = Lub-Dub

-Lub: post QRS – ventricle squeezes blood up / closes cuspid valves to prevent backflow

-Sounds are the valves closing – can only go up through semilunar valves and not back into atria

-Bicuspid valve in LV, tricuspid valve in RV

-Dub: post T – blood going out aorta/pulmonary artery / relaxing ventricles

-Close semilunar valves to maintain 1 way flow (prevent backflow into ventricles)

*Heart murmur – doors not closing all the way → blood flows in wrong direction (backflow)

-Fail to completely close the valve (Lub-**sh**-dub, Lub-dub-**sh**) = backflow

-Based on pattern, we know if it is semilunar or cuspid, but not which side

-Cardiologists listen to different parts of heart to find specific problematic valve

Cardiac Cycle

-Heart creates BP = the force getting blood moving to create convection needed to create distance movement that diffusion can't do on its own

-**Blood Pressure** – contraction of space in the heart

-PV=nRT (Inverse relationship of P and V → nRT are all constants in heart)

-2 phases: fill then empty = 1 cycle

-**Systole** (1/3 of time) – squeezing/contracting for ejecting blood

-**Diastole** (2/3 of time) – relax for filing

*Takes longer to relax (fill heart) than contract

-**Stroke Volume** = EDV – ESV = amount of blood ejected in 1 cycle

-EDV = end diastolic volume → pre-contract volume

-When heart most opened/filled with blood – average = 135 mL

-ESV = end systolic volume = post-contract volume

-Can't empty all blood → best squeeze leaves average of 65 mL

-Heart rate (HR) = cycles/min

-**Cardiac Output (CO)** = SV x HR = what heart is doing overtime (1 min)

-Typical: 70 mL/beat x 70 bpm = 4.9 L/min

<u>Regulating of CO</u>

-Changes possible: HR or SV (relatively independent)

-Biggest change → should change both

-<u>Regulation of HR</u>

-Autonomic:

-Sympathetic (fight or flight): Increases HR

1. Input to SA node increased (more APs)

*Must tell heart to contract/be ready to do so (complete previous cycle)

2. Increases conduction velocity → ready for next cycle faster → more cycles

-Parasympathetic = exact opposite: Decreases HR

-Decrease input to SA node, decreases conduction velocity

-Others: indirectly through autonomic system

-Epinephrine – try to increase heart activity (See in hospital shows after crash)

-Body temperature changes rate of flow

-Cold = keeps blood flow in core, warm = flow to surface to dissipate as heat)

*Only in extreme conditions does heart must work harder

-Heart doesn't play a big role in normal temperatures

-Electrolytes affecting V_m (calcium, sodium, potassium) – affect electrical activity

-Off-balance of any ions are the heart can have impacts

-Endocrines – Sex hormones when in love

-Estrogen/testosterone gets heart doing more

-Sexual elders usually don't have high levels → heart problems

<u>-Regulation of SV</u>

1. Increase EDV → fill up heart more

-Increase contract force (ejects more blood → bigger SV) when stretched **a little**

Ex: Stretching before game (myosin pulling against actin – string out to get most connections possible) → increase potential for cross-bridge cycling

*Only a little → stretching too much or will eliminate cross bridge from occurring by eliminating overlap (like water balloon) → no contraction

-Too big of stretch = Decrease force (myosin can't grab actin) → no contraction

-Congestive heart failure – heart overfilled/stretched too far → need to relax

2. Change in arterial pressure (where blood is sent to)

-Ventricle ejects to aorta on left side (artery)

-Pumping against pressure gradient out in system = resistance

*Daughter pushing chair → can't when Dr. B is in it

3. Sympathetic Nervous System (affects both SV and HR)

-Epi/Norepi will increase ability to heart to squeeze regardless of volumes

*Better squeeze = Decrease ESV = Increased SV

-SNS input increases strength of ventricular contraction regardless of EDV

Blood Vessels

-All types are lined with endothelium (plasma always inside tubes)

-Tight junctions between them to protect blood/ act as barrier between blood and IF

-**Arteries**: away from the heart (defined based on direction)

-Carry oxygenated blood usually, except for pulmonary artery

-Flexible/highly elastic: decreases variation

-Yes/no process (either pumps or doesn't) – blood goes out or it doesn't

-Want continuous flow of blood to keep cells aerobic → elasticity makes it possible by pushing on sides → rebound occurs in diastolic (resting)

*Allows less variation, keep continuous flow going

*Hardening in artery → increased variation → issues with cells (sometimes force cells to be anaerobic)

-Smooth muscle surrounds arteries – can change the size of vessels (changes BP)

-□□□□hydrostatic pressure (BP = the starting factor)

-Fluid pushes on walls to make vessel circular

-Ex: Like a fire hose → flat when no water, swell when filled with water

-2 components: BP = horizontal pressure

Hydrostatic pressure = perpendicular pressure

*More surface area = less hydrostatic pressure (like snowshoes)

-Hydrostatic pressure very high b/c very small wall (low SA) and high BP

*Aneurysm – blood flows out of wall due to build up of pressure

-**Arterioles:** smaller, but more

-Less elastic

-Smooth muscle

-□ hydrostatic pressure (much lower than arteries → further from the heart, more SA)

-**Capillaries**: smallest (allow only 1 RBC to go through at a time – not compressible cells)

-Very thin walls – 1 endothelial cell thick

-Allows diffusion to occur!!

-Site exchange (distance critical) of oxygen, CO_2, glucose

-Increased surface area → low hydrostatic pressure (necessary b/c walls so thin)

-Decrease flow rate to do necessary exchange → slowest movement

*Like food traveling on a conveyer belt – need time to get food

-□□□ hydrostatic pressure – b/c high SA in combination with low BP

Venules: Returning blood (lower [oxygen])

-Thin walled

-Low hydrostatic pressure (SA still relatively high, far from heart) – Don't need walls to be that strong

*Buildings in cbus not built as strong as in Chicago/LA → invest more due to shaky weather conditions – need stronger walls

*Hydrostatic pressure is beginning to go back up at this point in flow

Veins: bigger returning (arterioles converging)

-□□ hydrostatic pressure → but still increasing b/c bigger, fewer vessels = less SA

*Regular BP dropping – not impacting hydrostatic pressure here

-Hydrostatic pressure difference: artery spurts blood, while vein oozes

-Smooth muscle (regulating flow returning to the heart)

-Must go against gravity (lie down if issues to help flow)