Chapter 14 Outline

- Cardiac Output
- Blood Volumes
- Vascular Resistance to Blood Flow
- Blood Flow to the Heart and Skeletal Muscles
- Blood Flow to the Brain and Skin
- Blood Pressure

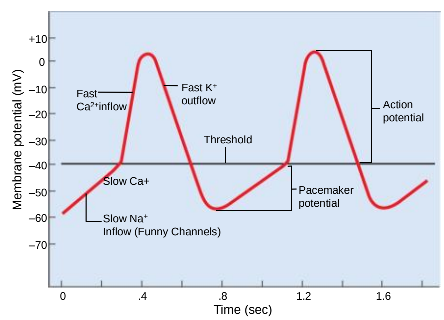
Cardiac Output (CO)

- Is volume of blood pumped/min by each ventricle
- Stroke volume (SV) = blood pumped/beat by each ventricle
- Heart rate (HR) = the number of beats/minute
- CO = SV x HR
- Total blood volume is about 5.5L

Regulation of Cardiac Rate

- Without neuronal influences, SA node will initiate HR
- Autonomic innervation of SA node is main controller of HR
- Cardiac control center of medulla oblongata coordinates activity of autonomic innervation
  - Symp and Parasymp nerve fibers modify rate of spontaneous depolarization
  - Parasympathetic (Ach – inhibitory)
  - Sympathetic (NE – excitatory)

SA Node Potentials (Physiology)



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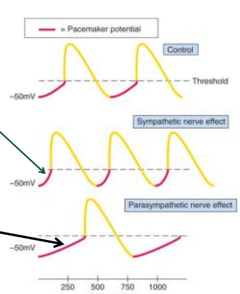
Regulation of Cardiac Rate

- Sympathetic:
- Norep. & epineph. stimulate opening of funny channels & Ca+ channels
  - depolarizes SA node faster, increasing HR
- Parasympathetic:
- ACh 1) promotes opening of K^+ channels & 2) decreases Ca+ permeability

1. K+channels open:
   - Hyperpolarizing cell
   - Pacemaker potential begins at more neg. value
2. Ca+ less permeable:
   - Slows pacemaker pot. to reach threshold



Multiple Factors Influence Stroke Volume

- Review: Systole & Diastole?

Stroke volume is directly related to force of contraction of the ventricle

Ventricular contraction influenced by:

1. length of muscle fibers at beginning of contraction (influenced by amount of blood in ventricle)
2. contractility of the heart (intrinsic ability of fiber to contract at a given fiber length)

Multiple Factors Influence Stroke Volume

- Stroke Volume is determined by 3 variables:
  1. End diastolic volume (EDV) = volume of blood in ventricles at end of diastole – just before contraction
  2. Total peripheral resistance (TPR) = resistance to blood flow in arteries
  3. Contractility = strength of ventricular contraction

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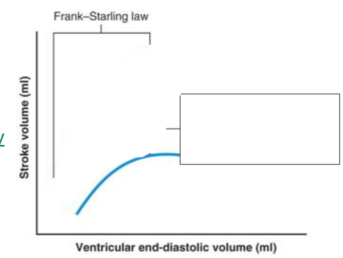
Frank-Starling Law of the Heart

- Strength of ventricular contraction varies directly with EDV
  - As EDV increases, myocardium is stretched more, causing greater contraction and SV
  - An intrinsic property of myocardium



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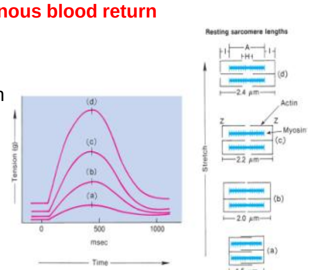
Frank-Starling Law of the Heart

- When is tension of cells greatest?
- According to Frank-Starling Law: stroke volume increases with increased EDV
- **EDV is determined by Venous blood return**

3 Factors influence venous return.

1. Contraction/compression of veins returning blood (skeletal muscle pump)
2. pressure changes in abdomen and thorax during breathing (respiratory pump)
3. sympathetic innervation



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Extrinsic Control of Contractility (Nervous and endocrine system)

1. Sympathetic:

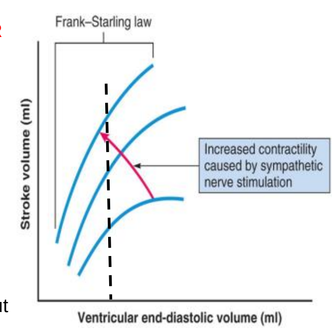
Norepi. & Epinephrine produce increase in **HR and contraction**

- Due to increased Ca^{2+} availability
- More cross-bridging
- Greater force of contraction

- Parasympathetic influence

- SA node (slower APs)

- not direct affect on contraction strength (but recall F-S Law)



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Venous Return

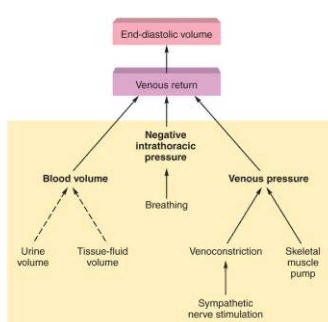
- **Return of blood to heart via veins**
- **Controls EDV and thus SV and CO**
- **Dependent on:**
  - **Blood volume and venous pressure**

Aided by

1. Sympathetic nerves – contraction of smooth mms

2. Skeletal muscle pumps

3. Pressure drop during inhalation



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EDV and Arterial Blood Pressure

1. ***Recall - Preload*** degree of stretch on heart prior to contraction
   - Strength of contraction varies directly with EDV
2. **Afterload** Total peripheral resistance (TPR) = EDV & arterial resistance –
   - **- impedes ejection from ventricle**
   - - i.e., more blood left in ventricle – (stretched cells!)
   - SV is inversely proportional to Total Periph. Res.
     - - Increase TPR and SV lowers
     - - Decrease TPR and SV increases

- ***BUT after a few beats heart corrects this!!!!!!!!!!!!***

- i.e., in response to low SV (increased TPR) heart beats more strongly – because more blood was left in the ventricle.

- Frank-Starling Law of the Heart

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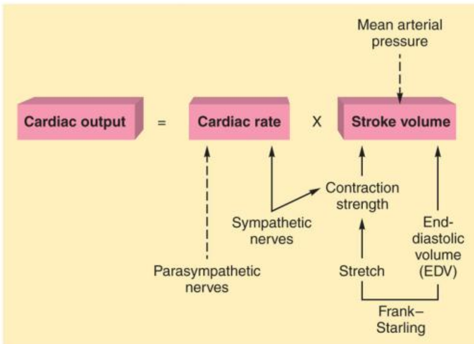
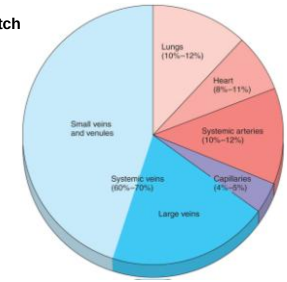
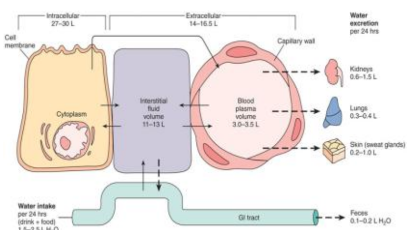


- **Veins hold most of blood in body (~70%)**
  - **Have thin walls and stretch easily W/O increased pressure (= higher compliance)**
    - **Have only 0-10 mm Hg pressure**



Blood Volume

- **Constitutes small fraction of total body fluid**
- **2/3 of body H_2O is in intracellular compartment**
- **1/3 total body H_2O is in extracellular compartment**
  - **80% of extracellular H_2O is interstitial fluid; 20% is blood plasma**



Exchange of Fluid between Capillaries and Tissues

- Distribution of ECF between blood and interstitial compartments is in state of dynamic equilibrium
- Movement in or out of capillaries is driven by
  1. hydrostatic pressure
  2. colloid osmotic pressure

- Hydrostatic pressure (pressure exerted on inside walls):
  - Promotes formation of tissue fluid

- Colloid osmotic pressure:
  - osmotic pressure exerted by proteins in fluid
    - Greater in caps than ECF –
    - i.e., water wants to move into caps.

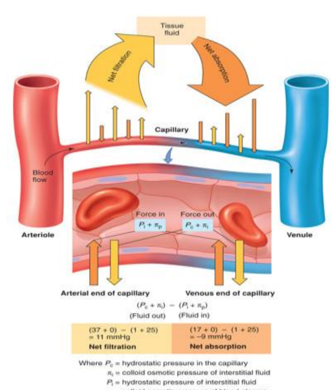
Difference between Hydrostatic and osmotic pressure determine if and how much leaves or enters capillaries

Overall Fluid Movement

- Determined by net filtration pressure and forces opposing it (Starling forces)

$(P_c + \pi_I) – (P_i + \pi_p)$
[fluid out] – [fluid in]

- P_c = Hydrostatic pressure in capillary
- π_i = Colloid osmotic pressure of interstitial fluid
- P_i = Hydrostatic pressure in interstitial fluid
- π_p = Colloid osmotic pressure of blood plasma



Regulation of Blood Volume

- Kidneys and hormones influence blood volume
  - Water and ions
  - Hormones
    - Antidiuretic Hormone
    - Aldosterone

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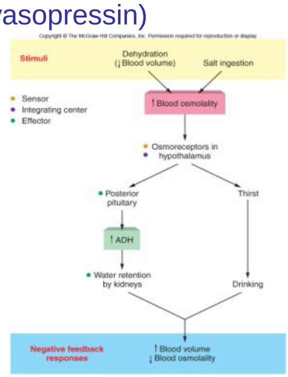
ADH (vasopressin)

- ADH released by Post.Pit.
- Osmoreceptors in hypothalamus detect high osmolality (low blood volume - dehydrated)
  - Excess salt intake/dehydration
  - Causes thirst
  - Stimulates H_2O reabsorption from urine



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Aldosterone

- Steroid hormone secreted by adrenal cortex
- Helps maintain blood volume/pressure through reabsorption and retention of Na+ and water
  - Release stimulated by low Na+, low blood volume, and low Blood pressure
  - Renin-angiotenson-aldosterone system

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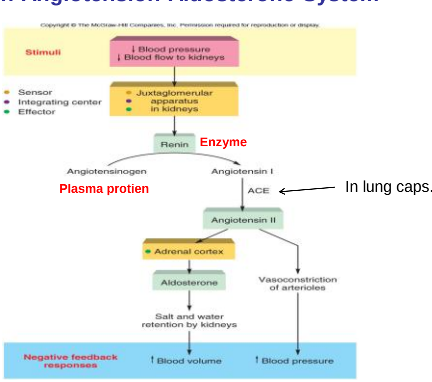
Renin-Angiotension-Aldosterone System



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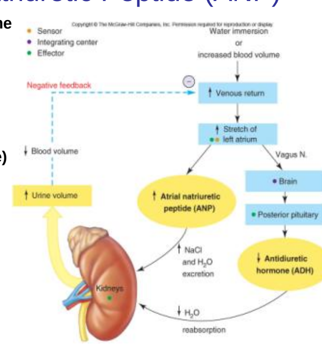
Atrial Natriuretic Peptide (ANP)

- **Increased blood volume detected by stretch receptors in Atrium**
- **Atrium releases ANP**
  - **- inhibits aldosterone**
  - **- promotes salt/H2O excretion**

(i.e, lowers blood volume)



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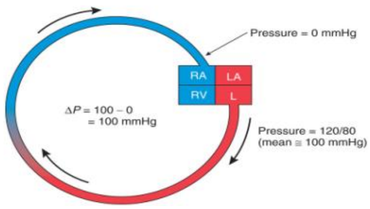
Physical Laws Describing Blood Flow

- Vascular resistance determines how much blood flows through a tissue or organ
  - Vasodilation decreases resistance, increases blood flow
  - Vasoconstriction does opposite
- Blood flows through vascular system when there is pressure difference (ΔP) at its two ends
  - Flow rate is directly proportional to difference ($\Delta P = P_1 - P_2$)



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Physical Laws Describing Blood Flow

- **Flow rate is inversely proportional to resistance**
  - **Resistance is affected by:**
    - **length of vessel (L)**
    - **radius of vessel**
    - **viscosity of blood (η)**
- **Sum of all vascular resistances within the systemic circulation is total peripheral resistance**

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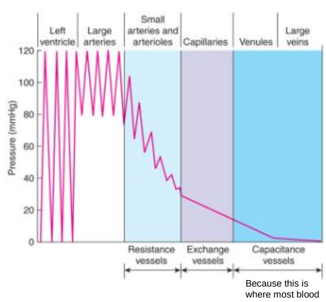
Blood Flow

- Mean arterial pressure and vascular resistance are 2 major factors regulating blood flow
  - Blood is shunted from one organ to another by degree of constriction of their arterioles



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Extrinsic Regulation of Blood Flow

- Extrinsic Regulation – control by ANS & endocrine sys.
- Sympathetic activation causes increased CO & Total Peripheral Resistance
  - **Increased Blood flows to skeletal muscles:**
    1. **Arterioles to skin and viscera vasoconstict in response to Epinepherine (adrenal medulla)**
    2. **Sympathetic fibers release Norepinepherin further constricts arterioles**
- blood is shunted away from viscera and skin to muscles

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Extrinsic Regulation of Blood Flow

- Parasympathetic can cause vasodilation
  - But Parasymp is not as important as Symp

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Paracrine Regulation of Blood Flow

- Paracrine regulators: molecules produced by tissue of one organ to help regulate tissue of same organ
  - i.e., local regulation
- Endothelium produces several paracrine regulators that promote smooth muscle relaxation:
  - Nitric oxide, bradykinin, prostacyclin
    - NO is involved in setting resting "tone" of vessels
      - Levels are increased by Parasymp activity
      - Low 0_2 and high CO_2 cause NO release

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Intrinsic Regulation of Blood Flow (Autoregulation)

- Maintains fairly constant blood flow despite BP variation
- Myogenic control mechanisms occur in some tissues to maintain proper B.P.
  - smooth muscle of arterioles contract when **stretched** and **relaxes** when not stretched
  - e.g. increased BP in arteriole causes constriction
    - and resistance by the arteriole
    - and decreased blood flow through vessel

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Intrinsic Regulation of Blood Flow (Autoregulation)

- Metabolic control mechanism matches blood flow to local tissue needs
- If Low O_2 or pH or high CO_2, adenosine, or K+
  - (all often caused from high metabolism)
  - Vasodilation occurs via paracrine regulators
  - i.e., if metabolically active more blood goes to area
  - particularly important in brain, heart, skel. mms.

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Aerobic Requirements of the Heart

- Heart (and brain) must receive adequate blood supply at all times
  - Aerobic
  - Contains lots of mitochondria
- During systole, coronary vessels are squeezed
  - Heart gets around this by having lots of myoglobin
    - During diastole oxygen is stored in myoglobin
    - During systole myoglobin releases it

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Circulatory Changes During Exercise

- **At rest, flow through skeletal muscles is low**
  - **because vessels are constricted**
- **At beginning of exercise, Sympathetic activity causes vasodilation via Epinepherine and local ACh release**
  - **Blood flow is shunted from periphery and viscera to active skeletal muscles**
  - **Blood flow to brain stays same**
- **As exercise continues, intrinsic metabolic control is major vasodilator**
- **Symp effects cause SV and CO to increase**

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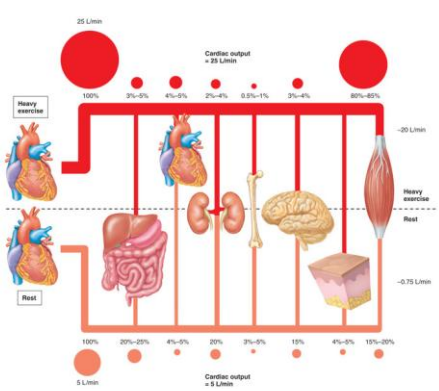

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Cerebral Circulation

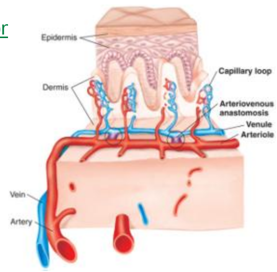
- Gets about 15% of total resting CO
- Regulated almost exclusively by intrinsic (myogenic regulation) mechanisms
  - When BP increases, cerebral arterioles constrict (protects finer vessels "down stream")
  - when BP decreases, arterioles dilate
- Brain is also responsive to CO_2 levels (Metabolic regulation)
  - If high CO_2, cerebral arterioles dilate
  - If low they constrict (why we get light headed when we hyperventilate)

14-50

Cutaneous Blood Flow

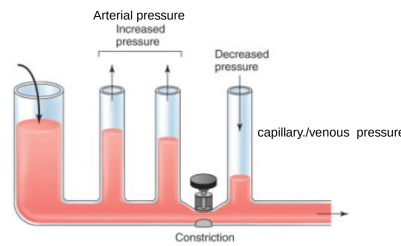
- Skin serves as a heat exchanger for thermoregulatior
- Skin blood flow is adjusted to keep deep-body at 37ºC

1. arterial dilation
2. arteriovenous anastomoses
    - Symp activity closes during cold and fight-or-flight, opens in heat and exercise



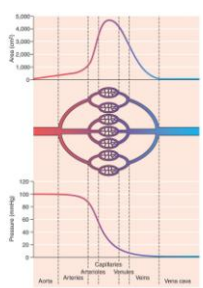
Blood Pressure (BP)

- Arterioles play role in blood distribution and control of BP
- Blood flow & BP to capillaries controlled by diameter of arterioles



Blood Pressure (BP)

- Capillary BP is low because of large total cross-sectional area

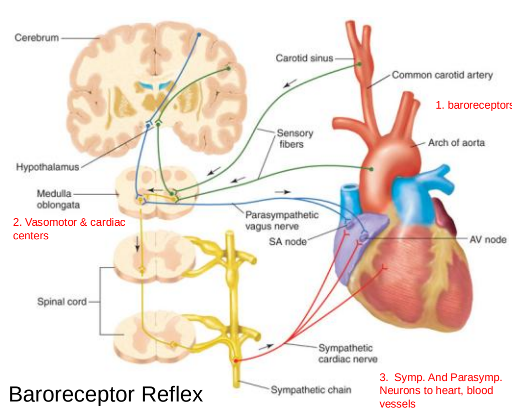


Blood Pressure (BP)

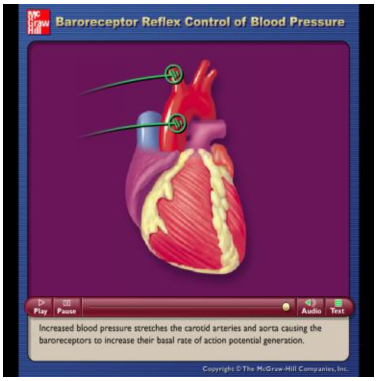
- Controlled mainly by HR, SV, and Total peripheral resistance
    - An increase in any of these can result in increased BP
- Sympathetic activity raises BP via arteriole vasoconstriction and by increased CO
- Kidney plays role in BP by regulating blood volume and thus stroke volume

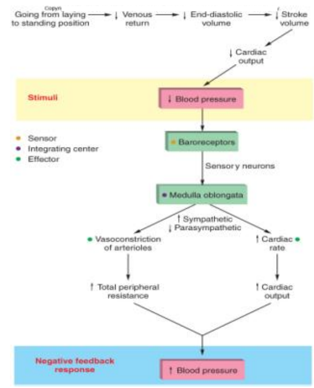
Baroreceptor Reflex

- Is activated by changes in BP
    - baroreceptors (stretch receptors) in aortic arch and carotid arteries
        - Increase in BP causes walls of these regions to stretch, increasing frequency of APs
            - to vasomotor control center and cardiac control centers in medulla oblongata
                - Vasomotor center: regulates vasoconstriction & dilation (i.e., TPR)
                - Cardiac control center: regulates cardiac rate



Baroreceptor Reflex





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Measurement of Blood Pressure

- Via <u>auscultation</u> (to examine by listening)
- No sound is heard during <u>laminar flow</u> (normal, quiet, smooth blood flow)
- <u>Korotkoff sounds</u> can be heard when <u>sphygmomanometer</u> cuff pressure > diastolic but < systolic pressure
  - Cuff constricts artery creating noise (<u>turbulent flow</u>) noise as blood passes constriction during systole and is blocked during diastole
  - 1st sound is heard at pressure blood is 1st able to pass thru cuff (systole)
  - last sound when cuff pressure = diastolic pressure

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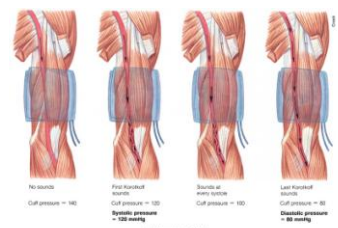
Measurement of Blood Pressure

- Blood pressure cuff is inflated above systolic pressure, occludes artery (no sound)
- Cuff pressure is lowered, blood flows only when systolic pressure is above cuff pressure, producing Korotkoff sounds
- Sounds are heard until cuff pressure equals diastolic pressure



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