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)

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

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

Extrinsic Control of Contractility (Nervous and endocrine system)

1. **Sympathetic:**
   - Norepi. & Epinepherine 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)

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

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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Veins hold most of blood in body (~70%)
- Have thin walls and stretch easily without increased pressure (higher compliance)
- Have only 0-10 mm Hg pressure

Blood Volume
- Constitutes small fraction of total body fluid
- 2/3 of body H₂O is in intracellular compartment
- 1/3 total body H₂O is in extracellular compartment
- 80% of extracellular H₂O 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)
  \[
  (P_c + \pi_t) - (P_i + \pi_p) \]
  - \(P_c\): Hydrostatic pressure in capillary
  - \(\pi_t\): Colloid osmotic pressure of interstitial fluid
  - \(P_i\): Hydrostatic pressure in interstitial fluid
  - \(\pi_p\): Colloid osmotic pressure of blood plasma
Regulation of Blood Volume

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

ADH (vasopressin)

- ADH released by Post.Pit.
- Osmoreceptors in hypothalamus detect high osmolality (low blood volume - dehydrated)
- Excess salt intake/dehydration
- Causes thirst
- Stimulates H₂O reabsorption from urine

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-angiotensin-aldosterone system

Renin-Angiotension-Aldosterone System

- Increased blood volume detected by stretch receptors in Atrium
- Atrium releases ANP - inhibits aldosterone - promotes salt/H₂O excretion (i.e., lowers blood volume)

Atrial Natriuretic Peptide (ANP)
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 ($\Delta P$) at its two ends.

Flow rate is directly proportional to difference ($\Delta P = P_1 - P_2$).

Flow rate is inversely proportional to resistance.

Resistance is affected by:
  - length of vessel ($L$)
  - radius of vessel
  - viscosity of blood ($\eta$)

Sum of all vascular resistances within the systemic circulation is total peripheral resistance.

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.

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.

Parasympathetic can cause vasodilation.

But Parasymp is not as important as Symp.

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 $O_2$ and high $CO_2$ cause NO release
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
- **Metabolic control mechanism** matches blood flow to local tissue needs
  - If Low O\textsubscript{2} or pH or high CO\textsubscript{2}, adenosine, or K+
    - (all often caused from high metabolism)
  - Vasoconstriction occurs via paracrine regulators
    - i.e., if metabolically active more blood goes to area
    - particularly important in brain, heart, skel. mms.

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

Circulatory Changes During Exercise

- At rest, flow through skeletal muscles is low
  - because vessels are constricted
- At beginning of exercise, Sympathetic activity causes vasoconstriction via Epinephrine 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

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\textsubscript{2} levels (Metabolic regulation)
  - If high CO\textsubscript{2}, cerebral arterioles dilate
  - If low they constrict (why we get light headed when we hyperventilate)
Cutaneous Blood Flow

- Skin serves as a heat exchanger for thermoregulation.
- Skin blood flow is adjusted to keep deep-body at 37°C.
  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.

Baroreceptor Reflex

- 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.

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.
Measurement of Blood Pressure

- Via **auscultation** (to examine by listening)
- No sound is heard during **laminar flow** (normal, quiet, smooth blood flow)
- **Korotkoff sounds** can be heard when **sphygmomanometer** cuff pressure > diastolic but < systolic pressure
  - Cuff constricts artery creating noise (**turbulent flow**) 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

- 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