Blood Flow and Blood Pressure, Chapter 14

Outline of class notes:
Objectives: After studying this chapter you should be able to:
1. Describe the quick and slow response mechanisms that adjust to an increase and decrease in blood pressure.
2. Explain what total peripheral resistance (TPR) is and the three parameters that affect TPR in blood vessels:
3. Describe the intrinsic and extrinsic mechanisms that control arteriole diameter.
4. Describe the changes in blood flow that occur during exercise focusing on the intrinsic and extrinsic control of arteriolar radius.
5. Explain how the kidneys regulate blood volume focusing on antidiuretic hormone and the Renin-Angiotensin-Aldosterone System.
6. Describe the distribution of water within the body.
7. Describe the characteristics of primary and secondary hypertension
8. Describe the four main types of circulatory shock
9. Explain the forces/mechanisms involved in blood volume regulation at the capillaries and then define edema and describe the various causes of edema.

Blood Pressure
- Blood pressure is the pressure exerted by circulating blood upon the walls of blood vessels.
  - When used without further specification, "blood pressure" usually refers to the arterial pressure in the systemic circulation.
  - Blood pressure varies depending on situation, activity, and disease states.
  - It is regulated by the nervous and endocrine systems and the kidneys.

Overview: Blood Pressure Regulation
- There are two basic mechanisms for regulating blood pressure:
  1. Quick response mechanisms: Regulate vessel diameter, heart rate and heart contractility.

Quick Response Regulation of Elevated Blood Pressure
- Rising blood pressure causes the arterial walls to stretch which stimulates the baroreceptors in the carotid sinus and aortic arch.
  - Baroreceptors are specialized sensory neurons in the walls of the aortic arch and the carotid sinuses (dilations of the carotid arteries in the neck).
  - Baroreceptors measure BP by monitoring the degree of stretch in the vessel wall.
- Increased blood pressure causes baroreceptors to increase their rate of impulses being sent to the cardiac centers of the medulla oblongata.
- The medulla oblongata responds by:
  - Increased parasympathetic activity (vagus nerve) which lowers heart rate.
  - Decreased sympathetic activity (cardiac nerves) which reduces heart rate and force of contraction and increases the diameter of the arterioles. Remember that decreased sympathetic activity dilates arteriolar diameter.
- The result is a lowering of blood pressure to normal levels.
**Quick Response Regulation of Lowered Blood Pressure**

- A sudden decrease in blood pressure is detected by **baroreceptors** in the **carotid sinus** and **aortic arch**.
- **Baroreceptors** decrease their rate of impulses that are being sent to the medulla oblongata.
- The **medulla oblongata** responds by:
  - **Decreased parasympathetic activity** (vagus nerve).
  - **Increased sympathetic activity** which:
    1. Increases heart rate and force of contraction - higher cardiac output
    2. Causes vasoconstriction of arterioles.
    3. Stimulates adrenal glands to release the hormones epinephrine and norepinephrine which enhances heart rate, contractility, and vasoconstriction.

The result is an increase of blood pressure to normal levels.

**Slow Response Regulation of Blood Pressure**

- Slow or long-term regulation of blood pressure is primarily accomplished by altering blood volume and is mediated by the **kidneys** and the **hypothalamus**.
- Changes in blood volume due to hemorrhage, accident, or donating a pint of blood will lower blood pressure and trigger processes to restore blood volume.
  - Note these will also activate the quick regulatory mechanisms as well.

**Kidneys: Effects of the Renin-Angiotensin-Aldosterone System**

- We will discuss this system in detail later

**Hypothalamus: Effects of Osmolarity on Blood Pressure**

- Dehydration due to sweating, diarrhea, or excessive urine flow will cause an increase in osmolarity of blood and a **decrease in blood volume and pressure**.
  - **Osmolarity**: Measure of solute concentration; the number of particles in a solution.
- The **hypothalamus** detects an increase in blood osmolarity and responds by:
  - Activating the **hypothalamic thirst centers** to stimulate the individual to drink more water.
  - Sends a signal to the posterior pituitary to **release antidiuretic hormone (ADH)**.
  - ADH increases the reabsorption of water in the collecting ducts of the kidney.
Mean Arterial Pressure

- **Mean arterial pressure (MAP)** is the main driving force for propelling blood to the tissues and must be closely regulated.
  - Must be high enough for the brain and other organs to receive adequate flow.
  - Must be low enough so that it doesn’t damage small blood vessels or create extra work for the heart.
- **Mean arterial pressure** is the blood pressure that is monitored and regulated in the body and consists of a number of factors as indicated by the diagram.
  - Review MAP diagram

Overview of the Determinants of Mean Arterial Pressure

Review of the Determinants of Mean Arterial Pressure

- Review figure as you read through the following. Numbers in text refer to number on figure.
- Mean arterial pressure depends on cardiac output and total peripheral resistance (1).
- Cardiac output depends on heart rate and stroke volume (2).
- Heart rate depends on the relative balance of parasympathetic activity (3), which decreases heart rate, and sympathetic activity (including epinephrine throughout this discussion) (4), which increases heart rate.
- Stroke volume increases in response to sympathetic activity (5) (extrinsic control of stroke volume).
Stroke volume also increases as venous return increases (6) (intrinsic control of stroke volume according to the Frank-Starling law of the heart).

Venous return is enhanced by sympathetically induced venous vasoconstriction (7), the skeletal muscle pump (8), the respiratory pump (9) and cardiac “suction” (10) – actually creates a vacuum and not due to “suction”.

The effective circulating blood volume also influences how much blood is returned to the heart (11). The blood volume depends on the short term on the size of passive bulk-flow fluid shifts between plasma and interstitial fluid across the capillary walls (12). In the long term, the blood volume depends on salt and water balance (13), which are hormonally controlled by the renin-angiotensin-aldosterone system and vasopressin (ADH), respectively (14).

The other major determinant of mean arterial pressure, total peripheral resistance, depends on the radius of all arterioles as well as blood viscosity (15). The main factor determining blood viscosity is the number of red blood cells (16). However, arteriole radius is the more important factor determining total peripheral resistance.

Arteriolar radius is influenced by local (intrinsic) metabolic controls that match blood flow with metabolic needs (17). For example, local changes that take place in active skeletal muscles cause local arteriolar vasodilation and increased blood flow to these muscles (18).

Arteriolar radius is also influenced by sympathetic activity (19), an extrinsic control mechanism that causes arteriolar vasoconstriction (20) to increase total peripheral resistance and mean arterial blood pressure.

Arteriolar radius is also extrinsically controlled by the hormones vasopressin (ADH) and angiotensin II, which are potent vasoconstrictors (21) as well as being important in salt and water balances (22).

Factors Affecting Mean Arterial Pressure
The following information will focus on each aspect of the factors affecting MAP

Blood Flow through Vessels is affected by Pressure and Total Peripheral Resistance

Pressure gradient: Blood flows from an area of higher pressure to an area of lower pressure down a pressure gradient.
- Contraction of the heart is the main driving force for blood flow through a vessel.
- The pressure in a vessel increases then

Total Peripheral Resistance: Resistance is the opposition to blood flow through a vessel.
- As resistance increases

Total Peripheral Resistance in the blood vessels is affected by three parameters:
1. Viscosity of the blood: The greater the
- Viscosity refers to the friction developed between the molecules of the fluid as they slide over each other.
- Primarily determined by the number of red blood cells.
- Excessive RBCs result in slower blood flow and increased blood pressure.
2. **Length of the vessel:** The longer the vessel the

- Blood “rubs” against the lining of the vessel as it flows past so the longer the total length of all the body’s blood vessels, the higher the blood pressure must be to push the blood.
- Because vessel length remains relatively constant under normal circumstances this is not a major factor to changes in blood pressure.
- Obesity causes increased resistance as the total length of the body’s blood vessels increases to supply the adipose tissue (~2 miles of additional blood vessels for each pound of fat).

3. **Diameter or radius of vessel:** The smaller the diameter the greater the resistance.
   - Of all of the factors that affect blood flow, the diameter of the blood vessel has the greatest effect.
   - The body controls blood flow to specific areas of the body by controlling the diameter of arterioles servicing those areas.

**Importance of Arterioles in Determining Blood Flow**
- Arterioles offer the most resistance to blood flow and thus help regulate arterial blood pressure and tissue perfusion.
- **Arterioles:**
   - Smooth muscle controls vasoconstriction and vasodilation
   - Innervated by sympathetic nerves
   - Respond to local chemical changes, hormones, and mechanical factors such as stretch (pressure).
Control of Arteriole Diameter

- Control of arteriolar vasoconstriction or vasodilation fall into two categories:
  1. 
  2. 

Intrinsic (Local) Controls of Blood Flow

- **Local control**: Ability of an organ to regulate its own blood flow regardless of what may be happening elsewhere in the body.
  - Two basic types of intrinsic control are **metabolic control** and **myogenic control**.

- **Local Metabolic Control**:
  - Arteriole diameter increases or decreases in response to the increased or decreased metabolic activity, respectively.
    - Increased blood flow in response to enhanced tissue activity is called **active hyperemia** (emia means blood)
  - The following factors produce vasodilation of arterioles in response to increased metabolic activity:
    - Increased adenosine, prostaglandin, and nitric oxide (NO₂) release
    - Increased K⁺ in tissue fluid due to repeated action potentials that outpace the ability the Na⁺/K⁺ pump to restore the resting membrane gradients of active muscle and neural tissue.
• **Increased heat production**
  • Can be used clinically to increase blood flow to an area
  • Conversely, applying ice packs to an inflamed area produces vasoconstriction, which reduces swelling.
  – Increase in blood flow eventually lowers the levels of metabolites thus removing the stimulus for vasodilation and the arteriole returns to its original diameter.

• **Clinical Note: Local Histamine Release Dilates Arterioles**
  – Not released in response to local metabolic changes and is not considered to control blood flow.
  – Tissue damage or allergic reactions cause
  – Causes vasodilation of arterioles and increases blood flow into the area that produces redness and contributes to swelling.

**Extrinsic Controls of Blood Flow**

• Control of **arteriolar diameter** by both the **autonomic nervous system** and the **endocrine system**.

• **Autonomic Nervous System Control**
  – Arterioles are generally enervated only by the **sympathetic nervous system**.
    • Parasympathetic innervation of arterioles is not significant except in the arterioles of genital erectile tissue (penis and clitoris).
  – Sympathetic nerve fibers
    • Bind with **α1 adrenergic receptors** on smooth muscles of blood vessels.
    • Cerebral arterioles do not have α1 receptors and are almost entirely controlled by local mechanisms that maintain a constant blood flow.
  – Arterioles servicing tissues at rest receive a baseline amount of sympathetic stimulation and thus are slightly constricted (vessel b in the figure).
  – This baseline level of constriction is called **Vascular Tone**. Vasodilation is accomplished by decreasing sympathetic stimulation below baseline (vessel a).
  – Vasoconstriction is accomplished by increasing sympathetic stimulation above
- **Endocrine System Control: Epinephrine, Norepinephrine, Antidiuretic Hormone and Angiotensin II**
  - Epinephrine (adrenaline) and norepinephrine (noradrenaline) are hormones secreted by the adrenal medulla in response to sympathetic stimulation and have a significant effect on the diameter of blood vessels.
  - Norepinephrine combines with α1 receptors to produce general vasoconstriction – similar to
  - Epinephrine combines with both α1 and β-2 adrenergic receptors but has greater affinity for β-2 receptors.
  - Epinephrine can cause either vasoconstriction or vasodilation of blood vessels depending on the type of receptor found in the smooth muscle of a particular vessel.
  - The binding of epinephrine to α1 receptors leads to vasoconstriction.
    - α1 receptors are found in all
  - Conversely, the binding of epinephrine to β-2 receptors leads to vasodilation but not all tissues have β-2 receptors.
    - β-2 receptors are found predominantly in arterioles servicing skeletal muscle and heart muscle.
    - Arterioles in digestive organs and kidneys, in contrast, are equipped only with α1 receptors
  - During a full-blown sympathetic response (fight or flight), the released epinephrine combine with β2 receptors in the heart and skeletal muscle to reinforce local vasodilation mechanisms in these tissues.
    - In the rest of the circulatory system (which has α1 receptors) epinephrine causes
  - **Note:** Skeletal and cardiac muscles have the most powerful local metabolic control mechanisms with which to override generalized sympathetic vasoconstriction.
  - **Antidiuretic Hormone (vasopressin)**
    - Released by
    - A potent vasoconstrictor at high concentrations
    - Regulates water balance in blood plasma by controlling amount of water retained by kidneys – will discuss details later
  - **Angiotensin II**
    - Part of the renin-angiotensin-aldosterone system.
    - Regulates salt balance in blood plasma by controlling amount of salt retained by the kidneys and thus water retention.
    - Is a potent vasoconstrictor – will discuss details later.
Exercise and Blood Flow

- The changes in blood flow that occur during exercise provide an excellent illustration of intrinsic and extrinsic control of arteriolar radius.
- The vascular tone of arterioles found in skeletal muscle is relatively high, consequently blood flow to resting muscles is low (20-25% of total blood flow).
  - However, during heavy exercise, blood flow to the skeletal muscles increases significantly (up to 80-85% of total blood flow).
- The increase in blood flow to skeletal muscles during exercise is mediated by three factors:
  - (1) an increase in cardiac output
  - (2) vasodilation of
  - (3) vasoconstriction of

**An increase in cardiac output.**
- Exercise activates sympathetic nervous system output to the heart causing
  - Exercise increases venous return of blood to the heart via the skeletal muscle pump and the respiratory pump.
  - An increase in venous return leads to an increase in end-diastolic volume (EDV), which in turn, causes an increase in stroke volume (Frank Starling effect).

**Vasodilation of skeletal muscle arterioles.**
- The most important factor governing flow of blood to exercising muscles is local metabolic control (active hyperemia).
  - As muscular activity increases, metabolites build up and
  - Additionally, beta-adrenergic (β-2 receptors) stimulation by epinephrine causes vasodilation of arterioles in skeletal and cardiac muscle.

**Vasoconstriction of arterioles in the viscera and skin.**
- As a result of alpha-adrenergic sympathetic stimulation, arterioles in the
  - However, as exercise progresses and body temperature rises, cutaneous arterioles dilate in order to radiate heat and reduce body temperature.
Important Concept!

- **Statement:** Tonic sympathetic activity induces vasoconstriction (with the exception of those vessels in the brain) and reduces blood flow to organs which results in elevating blood pressure.
- **Question:** Why is it beneficial (via the sympathetic nervous system’s actions) to increase blood pressure while reducing blood flow to the organs by vasoconstriction? Seems counterproductive!
- **Answer:** The extent to which each organ actually receives blood flow is determined by intrinsic (local) arteriolar adjustments that
  - Therefore, each organ receives the appropriate amount of blood dependent on its metabolic activity.
  - The sympathetically induced arteriolar responses help maintain the appropriate driving pressure (MAP) on which organs can utilize as needed through local mechanisms that control arteriolar radius.

**Regulation of Blood Volume by the Kidneys**

- The kidneys produce ~180 L (50 gal) per day of blood filtrate.
  - ~98% of the filtrate is reabsorbed back into the vascular system.
- **Hormones that regulate blood volume in the kidney include:**
  - Antidiuretic Hormone (ADH)
  - Aldosterone
  - Renin-Angiotensin-Aldosterone System

**Antidiuretic Hormone (ADH)**

- **Antidiuretic hormone (ADH)**
  - Synthesized by the **hypothalamus** and released by the **posterior lobe** of the **pituitary gland**
  - Released in response to increased plasma concentrations (osmolality) that can be due to dehydration or by excessive salt intake.
    - Stimulation of osmoreceptors produces sensations of thirst and increased water intake, which increases blood volume.
    - ADH stimulates water reabsorption from the kidney filtrate and thus a smaller volume of urine is excreted and maintains blood volume.
Regulation of Blood Volume by Aldosterone

- **Aldosterone**: Stimulates kidneys to reabsorb salt (specifically Na+).
  - Retention of salt causes

- **Aldosterone** increases blood volume, but, unlike ADH, it does not produce a change in **plasma osmolality** because salt and water are reabsorbed in proportional amounts.

- **Aldosterone** secretion (by adrenal cortex) is stimulated by the **Renin-Angiotensin-Aldosterone System** which is responsive to:
  - Decreased Na+ concentrations

**Renin-angiotensin-aldosterone system**
Renin-Angiotensin-Aldosterone System Mechanism of Action

- **Steps in this magnificent process**

  1. Decrease in blood pressure (BP) and/or a decrease in Na+ concentration is sensed by specialized cells of the juxtaglomerular apparatus.
    - **Juxtaglomerular apparatus** contains:
      - **Macula densa cells** located at the top of the ascending limb of the renal tubule.
      - Function to monitor
      - **Juxtaglomerular cells** (granular cells) are modified smooth muscle cells located in the wall of the afferent arterioles.
      - Function as baroreceptors to sense changes in blood volume or pressure and secrete the enzyme renin.
      - **Juxtaglomerular cells**, in communication with the macula densa cells, release renin into the blood in response to a decrease in
  2. **Renin** enters the blood stream and converts the protein

  3. **Angiotensin I** is converted to **angiotensin II** by an angiotension-converting enzyme (ACE) as it passes through the capillaries of the lungs.
    - Thus, conditions of salt deprivation, low blood volume, and low blood pressure cause increased production of angiotension II in the blood.

  4. **Angiotensin II causes blood pressure and volume to rise by the following:**
    - **For Increased Blood Pressure:**
      - Is a powerful vasoconstrictor of small arteries and arterioles which increases
    - **For increased blood volume (and thus BP):**
      - Stimulates the adrenal cortex to release aldosterone, a hormone that promotes Na+ and water retention at the kidneys and thus increases blood volume and BP
      - Stimulates ADH release by the posterior pituitary which increases blood volume and thus BP
      - Activates the body’s thirst center within the hypothalamus;
        - Thirsty person drinks more - increased fluid intake causes

  5. **High salt intake**, leading to high blood volume and pressure, **inhibits renin secretion**.
    - Less aldosterone means less salt is retained by the kidneys and
Clinical Application: ACE Inhibitors

• Hypertension (high blood pressure) can be treated with angiotensin converting enzyme (ACE) inhibitors that:
  – Block the formation of Angiotensin II, thus reducing its vasoconstrictor effect.
  – Increase activity of bradykinin, a chemical that promotes vasodilation which decreases total peripheral resistance (TPR).
  – Good for treatment of

Blood Pressure Abnormalities

• Hypertension
  – Resting blood pressure above 140/90 mm Hg.
  – Occurs in one in three adults (33%).
  – 74.5 million people in the USA over the age of 20
  – 2 broad classes
    • Primary hypertension
    • Secondary hypertension
• Hypotension
  – Resting blood pressure below 100/60 mm Hg

Primary Hypertension

• Primary (essential) hypertension accounts for 90% of cases
  – Catchall category where the underlying cause is unknown.
  – Potential causes being investigated
    • Defects in salt management by the kidneys
    • Excessive salt intake: Salt osmotically retains water thus raising plasma volume
    • Diets low in K+ and Ca2+: Diets low in fruits, vegetables, and dairy products.
    • Plasma membrane abnormalities such as defective Na+-K+ pumps which are crucial to salt management by the kidneys
    • Variation in gene that encodes for angiotensinogen which causes excess production of angiotensinogen.
    • Endogenous digitalis-like substances increase cardiac contractility.
    • Abnormalities (decreases) in NO in blood vessels inhibits vasodilation abilities

Secondary Hypertension

• Secondary hypertension accounts for 10% of cases
  – Examples of secondary hypertension
    • Renal hypertension: Could be due to atherosclerotic lesions protruding into the renal artery or external compression by a tumor that reduces blood flow through the kidney and thus blood pressure.
      – Initiates the rennin-angiotensin-aldosterone pathway that promotes salt and water retention
    • Endocrine hypertension: Pheochromocytoma is an adrenal medullary tumor that secretes excessive epinephrine and norepinephrine causing high cardiac output and peripheral vasoconstriction.

Complications of Hypertension

• Congestive heart failure
• Stroke
• Heart attack
• Spontaneous hemorrhage
Hypotension
- Resting low blood pressure (below 100/60 mm Hg)
- Occurs when
  - Heart is too weak to drive the blood
- Orthostatic (postural) hypotension
  - Transient hypotensive condition resulting from insufficient compensatory responses to gravitational shifts in blood when person moves from horizontal to vertical position

Circulatory Shock
- Circulatory shock occurs when blood pressure falls so low that adequate blood flow to the tissues can no longer be maintained
- Four main types:
  1. Hypovolemic ("low volume") shock
     - Caused by severe hemorrhage or through loss of fluids (severe diarrhea, excessive urination, extensive sweating)
  2. Cardiogenic ("heart produced") shock
  3. Vasogenic ("vessel produced") shock
     - Widespread vasodilation triggered by vasodilator substances – two types:
       - Septic shock: Massive infection - infectant releases vasodilator substances.
       - Anaphylactic shock: Extensive histamine release is severe allergic reactions.
  4. Neurogenic ("nerve produced") shock
     - Loss of sympathetic vascular tone leads to generalized vasodilation.
Summary of the effects of the parasympathetic and sympathetic nervous systems on the factors that influence mean arterial blood pressure.

**Bonus Question Information**

**Intracellular vs. Extracellular Water Distribution**

- Distribution of water between the interstitial fluid and the blood plasma is determined at the capillaries
  - Total body water: ~ 44 L (11 Gallons)
  - Intracellular compartment: ~ 2/3 of total body water
  - Extracellular compartment: ~ 1/3 of total body water.
    - 80% as interstitial fluid
    - 20% as blood plasma
Blood Volume Regulation

- **Blood Volume is Regulated by:**
  - The exchange of fluid between capillaries and tissues
  - The kidneys:
    - Antidiuretic Hormone (ADH)
    - Renin-Angiotensin-Aldosterone System

Capillary Blood Pressure

- Blood pressure is relatively low
- Low capillary blood pressure is advantageous because:
  - Capillaries are fragile and High BP could cause them to burst
  - Capillaries are very permeable.
    - High BP could cause excess fluid loss

Mechanisms of Capillary Exchange

- **Capillaries:** Exchange sites between the blood and tissue fluid.
  - Water, nutrients, wastes, signaling molecules, lipid-soluble materials, and gases are exchanged primarily by diffusion
- Four forces affect the movement of fluid at the capillary.
  - **Capillary hydrostatic pressure**
  - **Capillary colloid osmotic pressure**
  - **Interstitial fluid hydrostatic pressure**
  - **Interstitial fluid osmotic pressure**

Capillary Hydrostatic Pressure

- **Capillary hydrostatic pressure:** Blood pressure within the capillaries.
  - Hydrostatic pressure pushes fluid out of the capillary and into the interstitial space by filtration.
  - Pressure declines by ~50% from the arterial end of the capillary to the venous end of the capillary.
    - Pressure at the Arterial end of the capillary is ~ 37 mm Hg and falls to ~17 mm Hg at the start of the venous system.

Capillary Colloid Osmotic Pressure

- **Capillary (plasma) colloid osmotic pressure (~25 mm Hg):** Osmotic pressure exerted primarily by plasma proteins (mainly albumin).
  - Is significantly greater than the colloid osmotic pressure of interstitial fluid.
- Opposes capillary hydrostatic pressure
- Promotes fluid movement into the capillary from the interstitial space.

Interstitial Fluid Hydrostatic Pressure

- **Hydrostatic pressure within the interstitial fluid:** Promotes fluid movement into the capillary from the interstitial space.
Interstitial Fluid Osmotic Pressure
- **Interstitial Fluid Osmotic Pressure**: Promotes fluid movement into the interstitial space from the capillary
  - Relatively low due to the low protein content of interstitial fluid.
  - Can be neglected (0 mmHg)

Capillary Fluid Movement
- Fluid movement can be determined by the following:
  - Expression to the left of the minus sign represents the sum of forces acting to move fluid out of the capillary
  - Expression to the right represents the sum of forces acting on the capillary.
    - Is a positive number at the arteriole end and a negative number at the venular end.

Summary of Capillary Fluid Movement
- Sum all the forces acting to move fluid out of and into the capillary you find that there is an overall net movement of fluid out of the capillary at the **arterial end** and an overall net movement of fluid into the capillary at the **venous end**.
  - Example: In skeletal muscle capillaries, the arterial end of the capillary has +11 mm Hg which favors fluid moving out; at the venous end of the capillary there is a -9 mm Hg, which favors fluid moving back into the capillary.
- Normally filtration (movement of fluid out of the capillary) slightly outweighs reabsorption (movement of fluid into the capillary).
  - About 24 L/day move out of the capillaries and about 20.4 L/day is reabsorbed. **What happens to the other 3.6 L?**
  - Lymphatic vessels functions to drain excess interstitial fluid and proteins and return it back to the vascular system.

Clinical Consideration: Edema
- **Edema**: Excessive accumulation of interstitial fluid.
  - Disturbance in the capillary and/or problems with lymphatic drainage.
- **Causes of Edema.**
  - High arterial pressure: Increases capillary pressure and causes excessive filtration.
    - Hypertension
  - Increased venous pressure: Produces a congestive increase in capillary pressure
    - Thrombosis (thrombus forms in a vein)
    - Pregnancy (mechanical compression of veins)
    - Varicose veins (blood pulls in veins)
    - Congestive heart failure
  - Decreased plasma protein concentration: Less capillary colloid osmotic pressure to pull fluid back in.
    - Liver disease (decrease in albumin production)
    - Kidney disease (excessive loss of proteins in urine)
    - Starvation (decrease in abumin production)
      - In children, fluid accumulates in the abdominopelvic cavity, producing the swollen bellies typical of starvation victims.
  - Increased capillary permeability allows leakage of plasma proteins into the interstitial fluid: Less colloid osmotic capillary pressure to pull fluid back in.
    - Tissue injury or allergic reactions (histamine increase capillary permeability)
  - Obstruction of lymphatic drainage: Prevents return of excess interstitial fluid
    - Filariasis: Filaria roundworm infection – transmitted by mosquito.
      - Elephantiasis
      - Roundworms block lymphatic drainage
Measurements of Arterial Blood Pressure

• Details will be Covered in Lab

Important Formulas

• **Pulse Rate** =

• **Pulse Pressure** =
  - Felt in an artery as the difference between systolic and diastolic pressure.

• **Mean Arterial Pressure (MAP)** = Diastolic Pressure + 1/3 Pulse Pressure
  - The average pressure driving blood forward into the tissues.
  - **Primary determinants**
    - Cardiac output

  - **Formula bases**: ~2/3 of the cardiac cycle is spent in diastole and only ~1/3 in systole.
Test Your Knowledge
• Suppose you measured the pulse rate and pulse pressure at the carotid artery and at the tibial artery.
  – Would pulse rate be the same in both places?
  – What about pulse pressure?
• What is the pulse pressure of someone with a BP of 130/95 mm Hg?
• If systolic BP is 118 mm Hg and pulse pressure is 41 mm Hg, what’s the diastolic BP?

Test Your Knowledge
• If your BP is 120/80 mm Hg, what is your mean arterial pressure?