



Western  
UNIVERSITY • CANADA

# Midterm #3 Review

## Sections 09/010

TA: Greydon Gilmore  
Physiology 2130  
Feb 24<sup>th</sup>, 2020

# Your TA Reminding You....

- 3<sup>rd</sup> Midterm – Feb 28<sup>th</sup> @ 6pm (15%)
- 4<sup>th</sup> Assignment – Mar 27<sup>th</sup> (1.5%)
  - Post 2 MC questions: Mar 25<sup>th</sup> @ midnight
- 3<sup>rd</sup> Quiz – due Feb 25<sup>th</sup> @ 4pm (1%)
  - Opens: Feb 24<sup>th</sup> @ 4pm
  - Closes: Feb 25<sup>th</sup> @ 4pm
- 4<sup>th</sup> Quiz – due March 31<sup>st</sup> @ 4pm (1%)
  - Opens: March 30<sup>th</sup> @ 4pm
  - Closes: March 31<sup>st</sup> @ 4pm

# Midterm Information

- **When:** Feb 28<sup>th</sup> from 6pm-7pm
- **What:** 35 multiple choice
  - Renal Physiology – 17 questions
  - Respiratory Physiology – 14 questions
  - GI physiology – 4 questions
- **Where:** TBD

# Factors that minimize simple diffusion (p. 307)

$$\text{Rate of dissusion} = \frac{\text{gradient} * \text{surface area}}{\text{thickness}}$$

1. Huge surface area (300+ million alveoli)
2. Blood flow velocity (slower flow = better reabsorption)
3. High pressure gradient
4. Thin membrane (BGB)
5. Rate of diffusion

# Effects of Changing Ventilation (p. 324)

Changing ventilation affects arterial  $PO_2$ ,  $PCO_2$  and pH

A. Holding breath without changing metabolic activity

- $\downarrow PO_2$
- $\uparrow PCO_2$  ( $\uparrow H^+ = \downarrow pH$ )

Less  $O_2$  coming in at alveolar capillaries,  
Less  $CO_2$  leaving at alveolar capillaries (i.e. build up in blood)

B. Hyperventilating without changing metabolic activity

- $\uparrow PO_2$
- $\downarrow PCO_2$  ( $\downarrow H^+ = \uparrow pH$ )

More  $O_2$  coming in at alveolar capillaries,  
More  $CO_2$  leaving at alveolar capillaries (i.e. less in blood)

C. Increase metabolic activity without changing ventilation

- $\downarrow PO_2$
- $\uparrow PCO_2$  ( $\uparrow H^+ = \downarrow pH$ )

More  $O_2$  leaving at systemic capillaries (i.e. less in blood),  
More  $CO_2$  entering at systemic capillaries

# Chapter 8: Renal

## 17 Questions on Exam

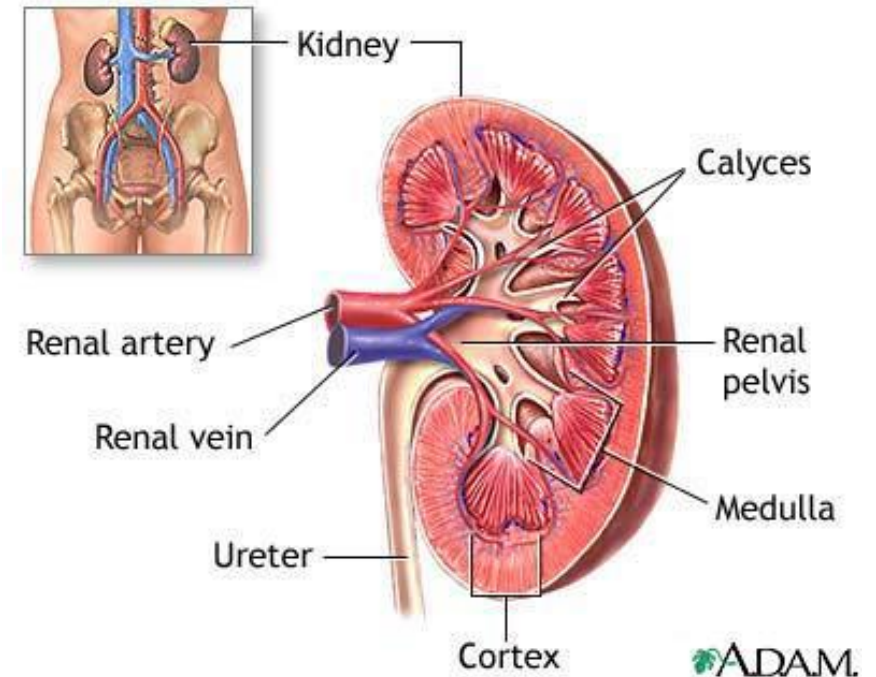
Professor: Dr. Woods

# Overview of Chapter

1. Renal anatomy
2. Glomerular filtration
3. Transport mechanisms
4. Water and sodium handling
5. No Renin System
6. ANP

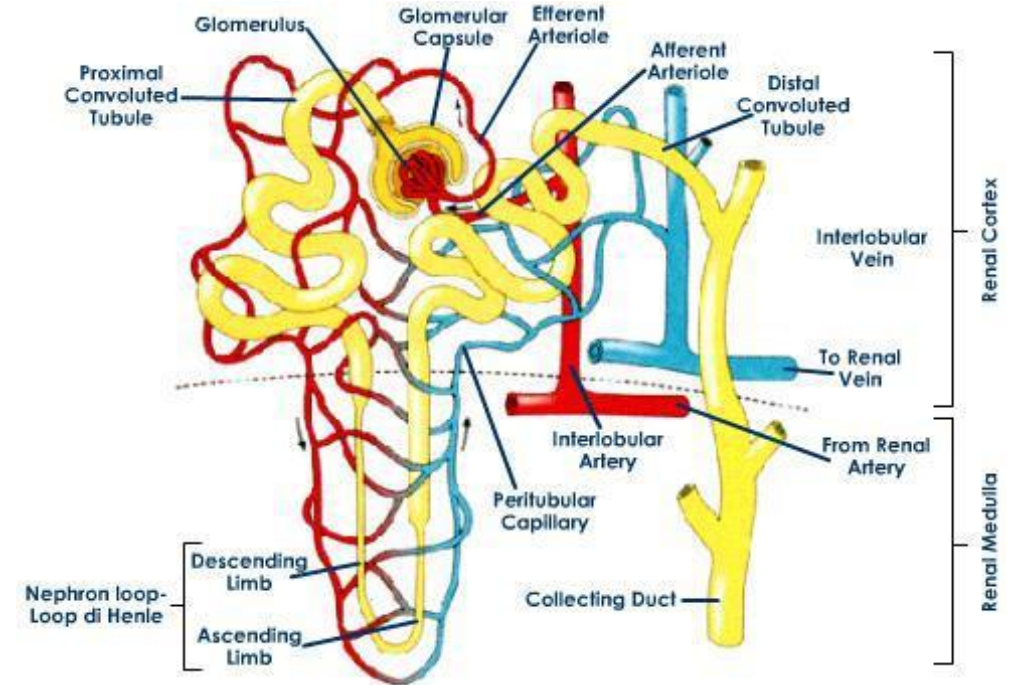
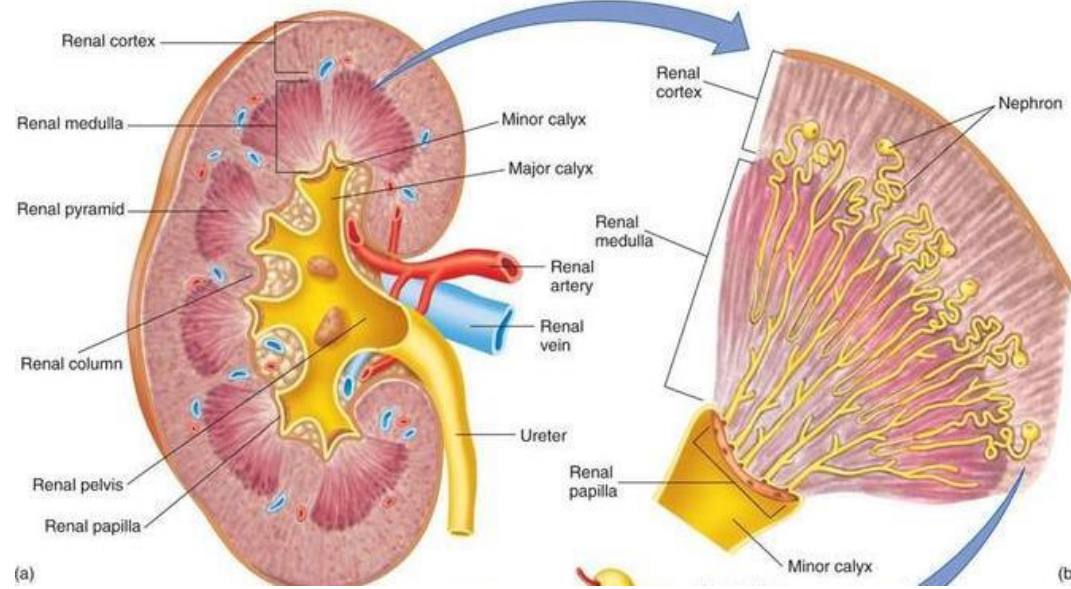
# Kidney Anatomy

- Two kidneys that sit posterior and outside of abdominal cavity (i.e. retroperitoneal)
- **Cortex** = outer portion
- **Medulla** = inner portion
- Fluid collected into minor calyces → major calyces → renal pelvis → ureter
- Renal artery carries blood to kidneys
- Renal vein carries blood away from kidneys



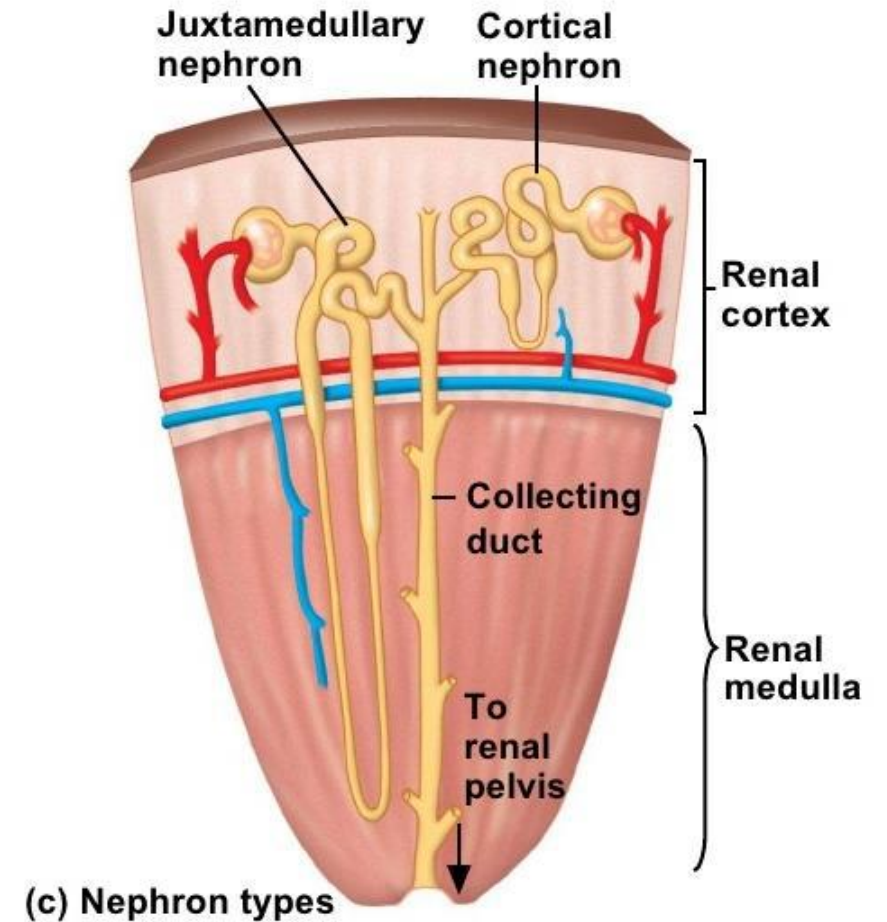
# Nephron Organization In The Kidney

- Renal corpuscle located in cortex
- Loop of Henle projects into and out of renal medulla
- 4-5 nephrons share a collecting duct
- Collecting ducts drain into minor calyx
- In 3D, the ascending limb is found near the glomerulus



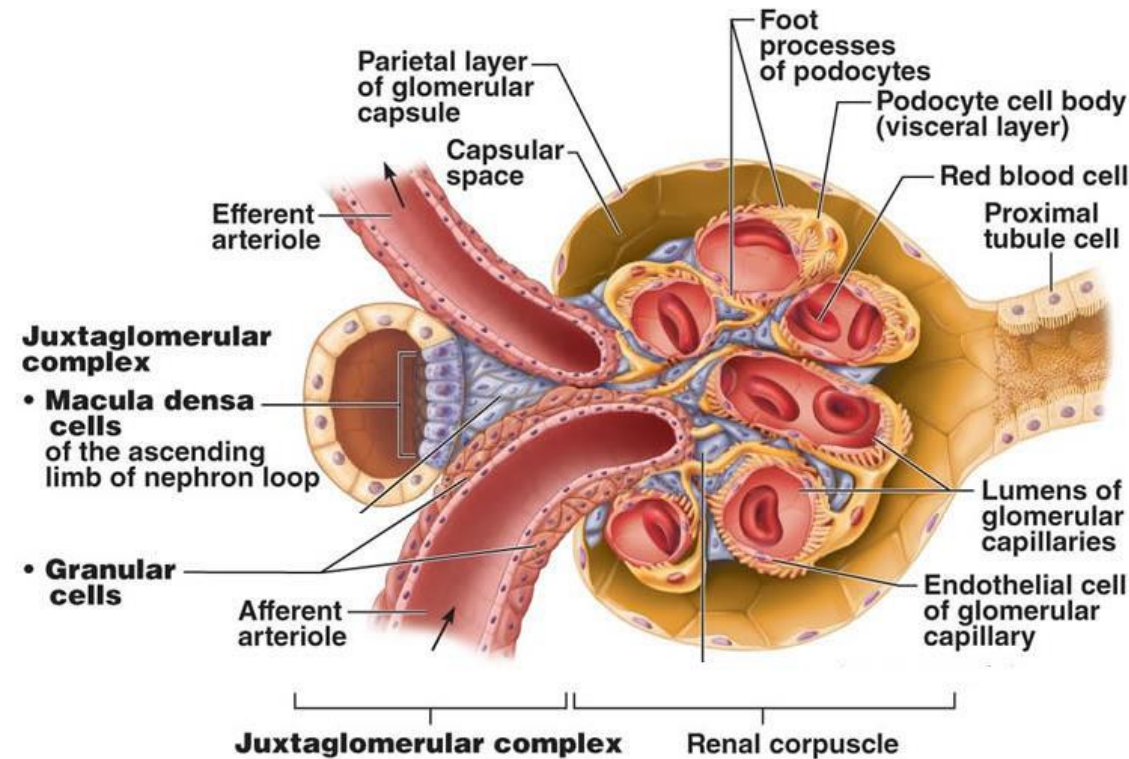
# Types of Nephrons

	Juxamedullary Nephron	Cortical Nephron
# in Kidney	Few	Many
Ability to concentrate urine	Good	Bad
Ability to filter blood	Good	Good
Location of Corpuscle in Cortex	Low	High
Loop of Henle	Long	Short
Capillaries	Vasa recta	Peritubular



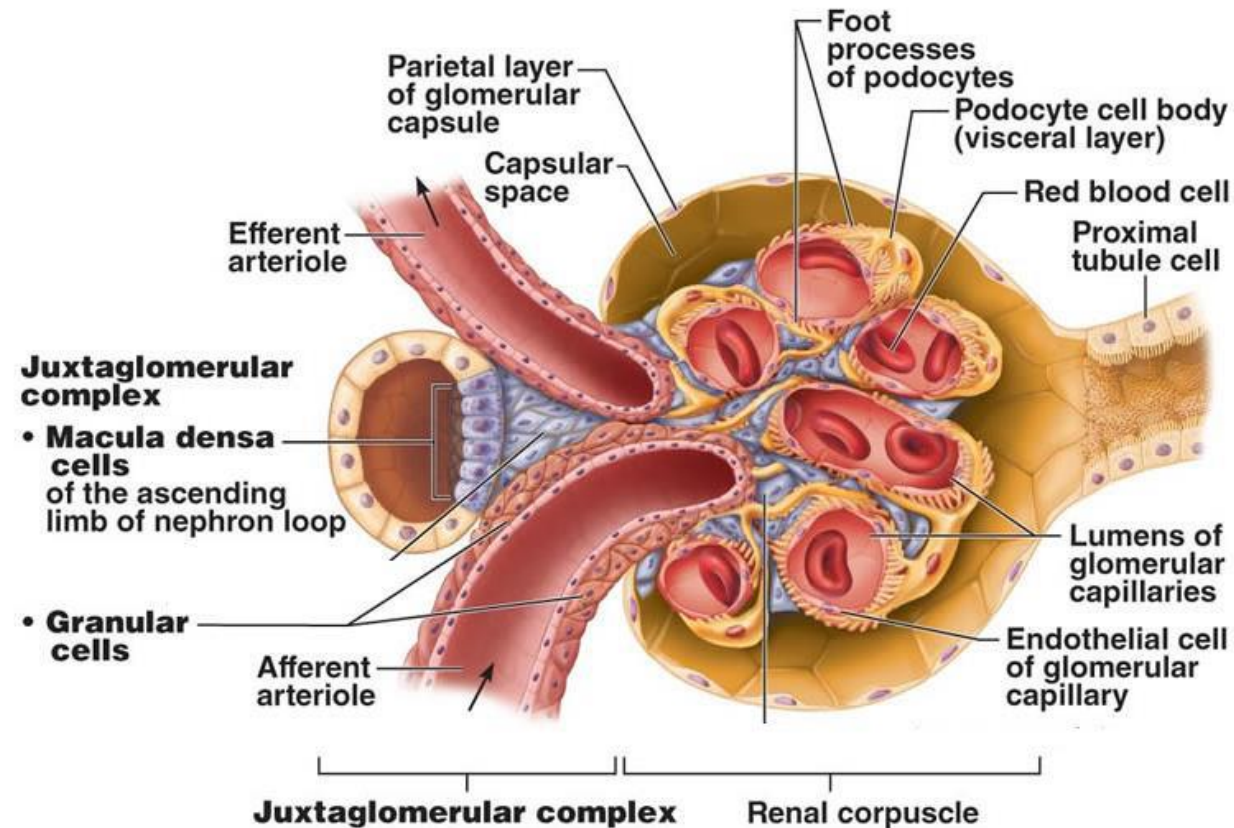
# Renal Corpuscle

- **Glomerulus:** Group of fenestrated capillaries
- **Fenestrations** allow passage of many substances (ions, water, etc.) into Bowman's space (except red/white blood cells)
- Cells of Bowman's capsule (outer layer) are **simple squamous epithelial cells**
- Cells of Bowman's capsule (inner layer) are called **podocytes**
- Cells of glomerular capillaries are called **endothelial cells**
- Endothelial cells are fused with podocytes by **basal lamina**



# Renal Corpuscle

- **JGA:** part of ascending Loop of Henle, containing macula densa
- **Macula Densa:** detect changes in  $\text{Na}^+$  and  $\text{Cl}^-$  levels in filtrate, cause vasoconstriction of afferent arteriole.
- **Granular Cells:** release renin



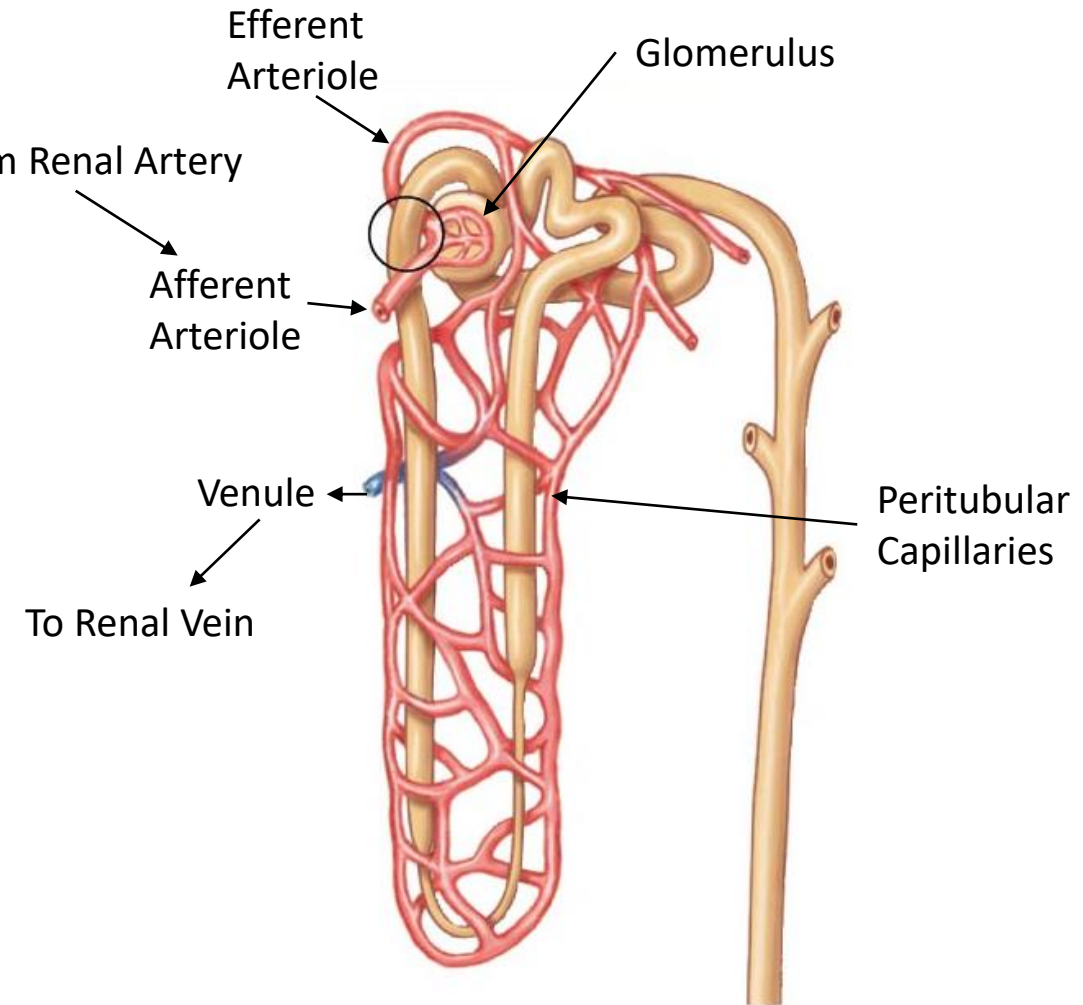
# Blood Flow to the Kidneys

Typical blood flow to organs is as follows:

Heart → Artery → Arteriole → Capillary →  
Venule → Vein → Heart

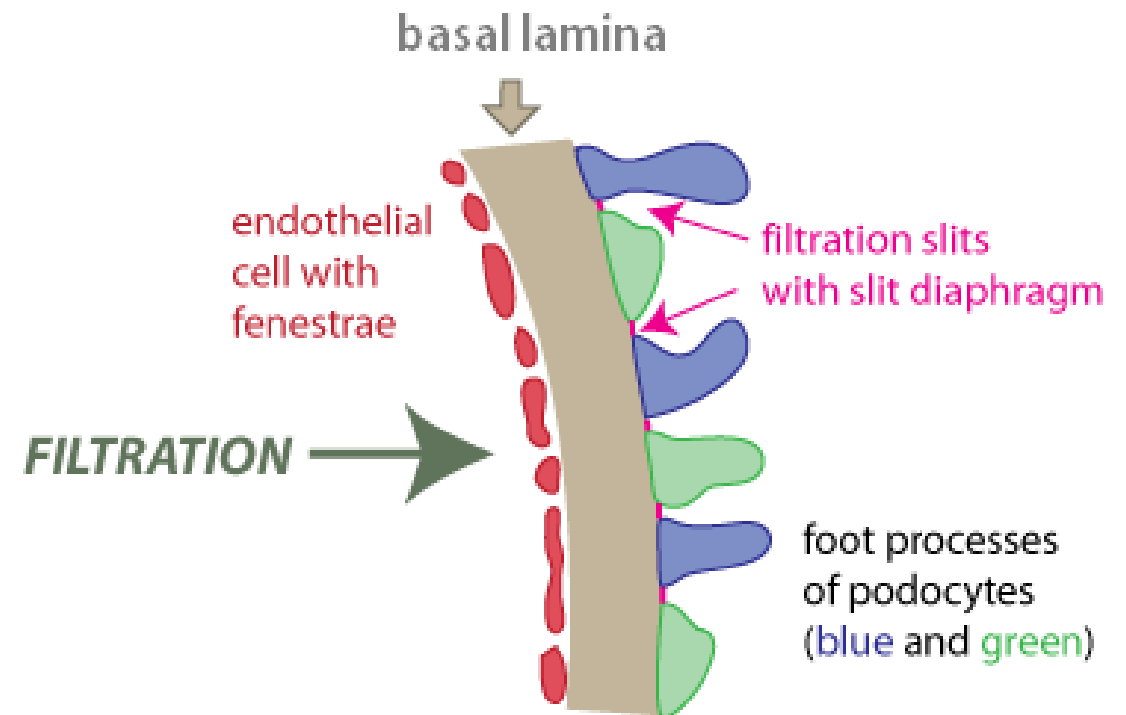
Blood flow to kidney different:

Heart → Artery → Arteriole → Capillary →  
Arteriole → Capillary → Venule → Vein →  
Heart



# Barriers to Filtration

1. Size of glomerular gaps/fenestrations
2. Gaps in basal lamina
3. Space between podocytes



# Barriers to Filtration

- Of all the blood that arrives at kidney, only 20% is filtered
- Net filtration pressure (NFP): Sum of forces that affect filtration
  - $\text{NFP} > 0$  : filtration
  - $\text{NFP} \leq 0$  : no filtration

	Hydrostatic Pressure of Glomerular Capillaries	Colloid Osmotic Pressure of Glomerular Capillaries	Hydrostatic Pressure of Bowman's Capsule	Colloid Osmotic Pressure of Bowman's Capsule
<b>Abbreviation</b>	$P_{GC}$	$\pi_{GC}$	$P_{BC}$	$\pi_{BC}$
<b>Caused by</b>	Blood flowing into glomerulus	Presence of proteins in glomerulus	Filtrate remaining in Bowman's space	Presence of proteins in Bowman's space
<b>Filtration</b>	Promotes	Inhibits	Inhibits	Promotes
<b>mmHg</b>	55	30	15	0

# NFP Calculation

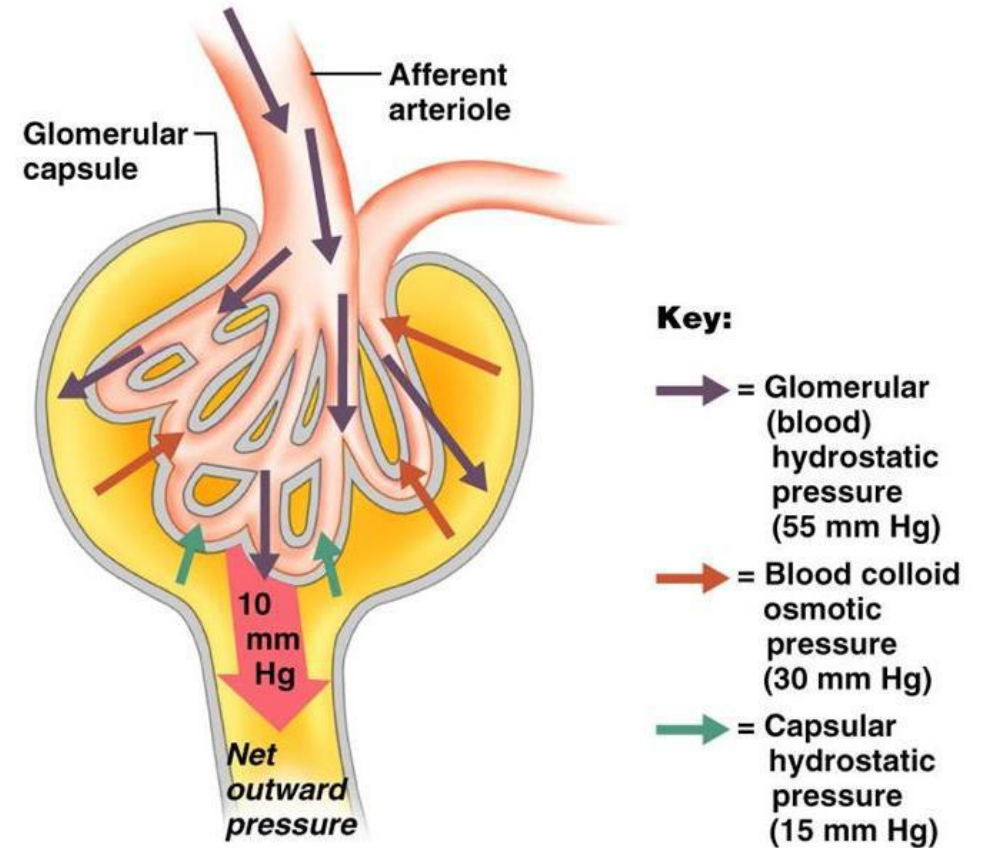
- NFP = Promotes Filtration – Inhibits Filtration

$$= (P_{GC} + \pi_{BC}) - (P_{BC} + \pi_{GC})$$

$$= (55 + 0) - (15 + 30)$$

$$= 10 \text{ mmHg}$$

	$P_{GC}$	$\pi_{GC}$	$P_{BC}$	$\pi_{BC}$
<b>Filtration</b>	Promotes	Inhibits	Inhibits	Promotes
<b>mmHg</b>	55	30	15	0



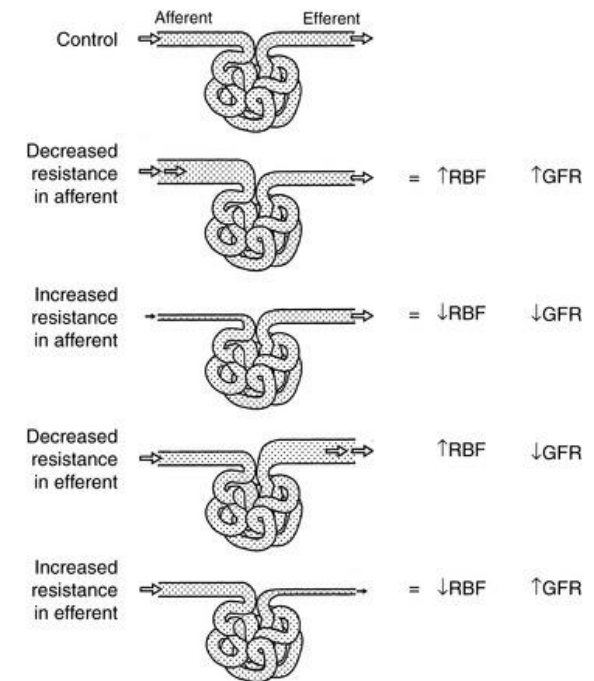
# Glomerular Filtration Rate (GFR)

- Volume of fluid filtered per day by the kidneys
- Normal: 180 L/day
- Important to maintain a constant GFR throughout the day
- Affected by:
  1. Net Filtration Pressure (NFP)
    - Mostly affected by renal blood flow and pressure ( $P_{GC}$ )
  2. Filtration Coefficient
    - Mostly affected by podocytes and basal lamina

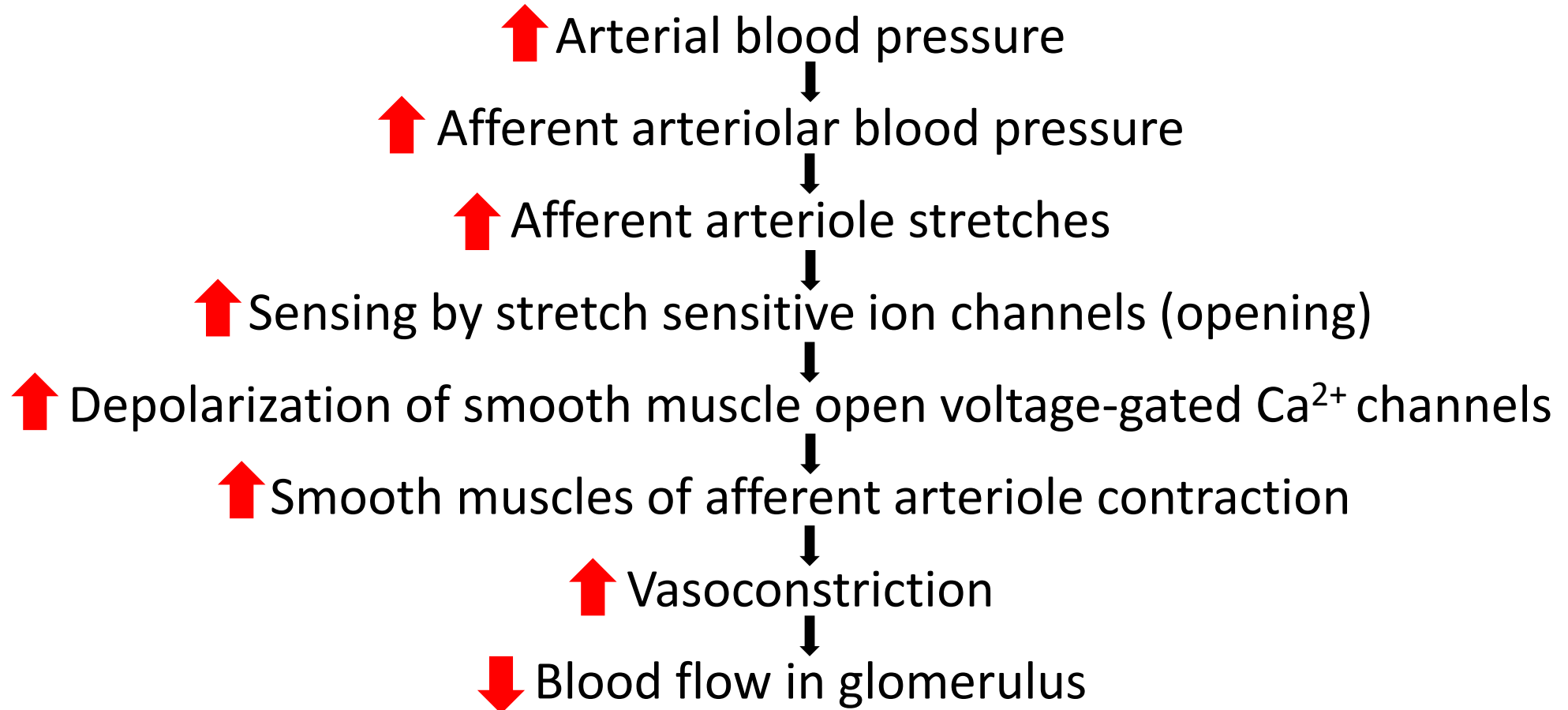
# GFR Regulation: Overview

- Both the myogenic response and tubuloglomerular response are used to increase and decrease GFR
- Their combined goal is to mediate a constant GFR throughout the day

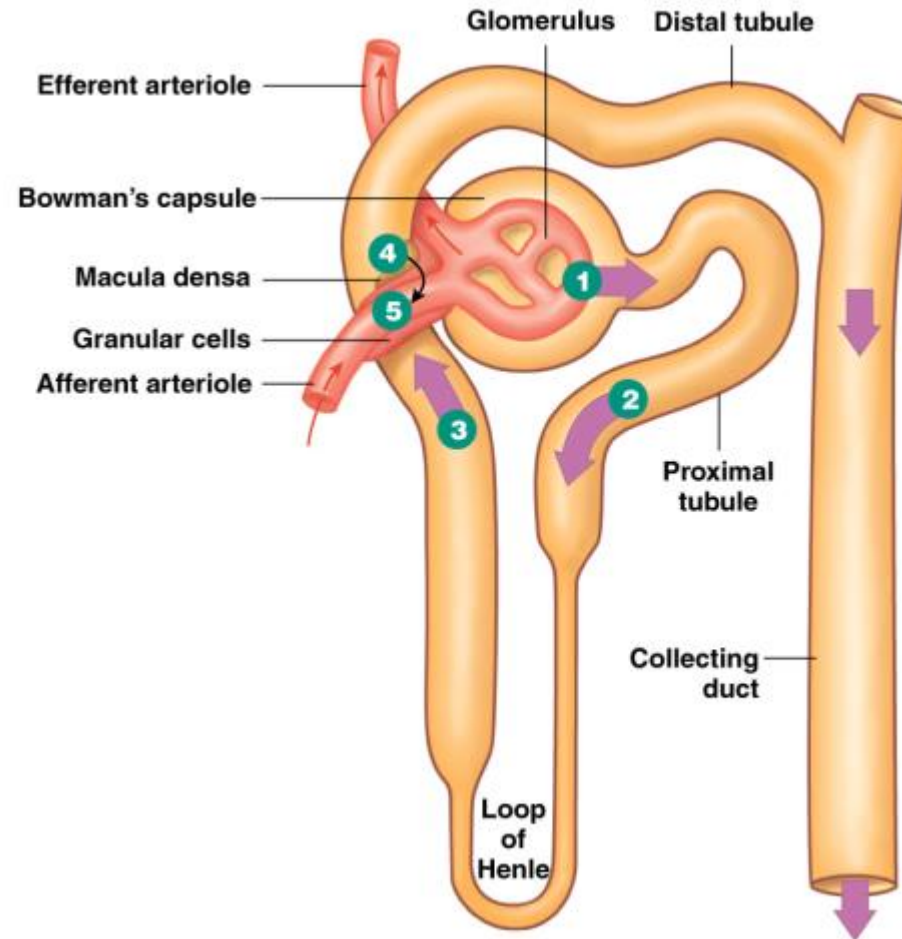
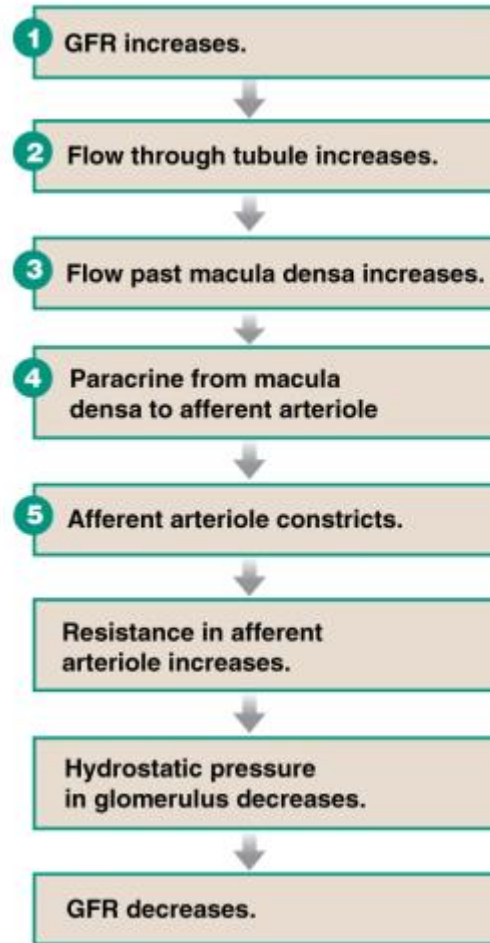
	Afferent Arteriole		Efferent Arteriole	
Smooth Muscle	Dilate	Constrict	Dilate	Constrict
GFR	↑	↓	↓	↑



# GFR Regulation: Myogenic Response



# GFR Regulation: Tubuloglomerular feedback



# Measurement of GFR

- In order to measure GFR, you want to choose a substance that is **excreted, but not reabsorbed**
- Bad substances: glucose, ions, water
- Best substance: **creatinine**
- Rate of creatinine excretion from the body is equivalent to GFR
- $\text{GFR (L/day)} = ([\text{Creatinine}]_{\text{urine}} \times \text{Urine/day}) / [\text{Creatinine}]_{\text{plasma}}$   
= (90 mg/L x 2 L/day) / (1 mg/L)  
= 180 L/day

# Renal Handling

## Example: Urea

- Filtered Load of Urea =  $[\text{Urea}]_{\text{plasma}} \times \text{GFR} = 0.31 \times 180 = 56 \text{ g/day}$
- % Excreted =  $(\text{Amount Excreted} / \text{Filtered Load}) \times 100\% = (28/56) \times 100\% = 50 \text{ g/day}$
- % Reabsorbed =  $100 - \% \text{ Excreted} = 100 - 50 = 50\%$

Substance	Concentration (in plasma)	Filtered Load	Amount Excreted	% Excreted	% Reabsorbed
Sodium	3.5 g/L	630 g/day	3.2 g	0.5%	99.5%
Glucose	1.0 g/L	180 g/day	0 g	0%	100%
Urea	0.31 g/l	56 g/day	28 g	50%	50%

# What is true regarding reabsorption in the kidney?

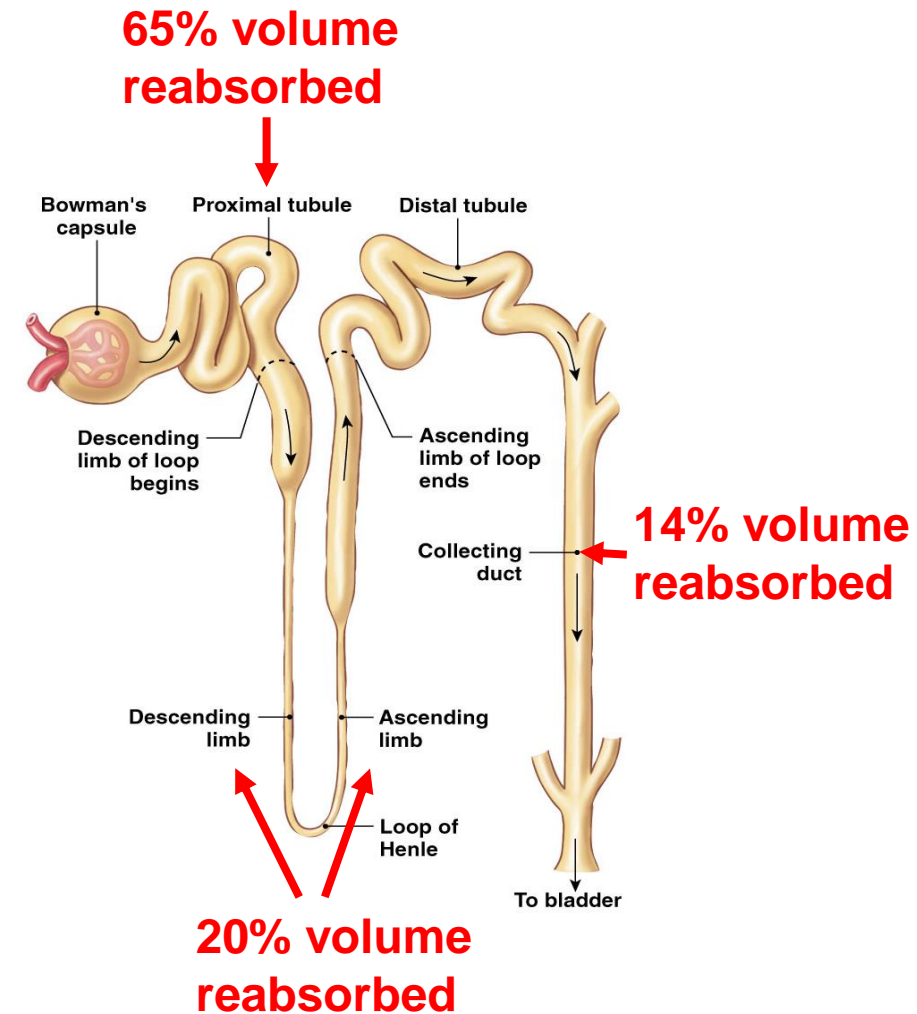
- a) all potassium is reabsorbed in the collecting duct
- b) glucose is reabsorbed by a type of primary active transporter
- c) all sodium is reabsorbed by a protein carrier transporter
- d) water reabsorption can be both paracellular and transcellular in the proximal tubule

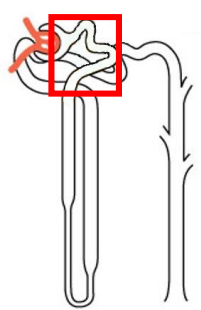
# What is true regarding the action of hormones on the nephron?

- a) anti-diuretic hormone (ADH) increases sodium reabsorption in the proximal tubule
- b) aldosterone increases sodium reabsorption in the collecting duct
- c) angiotensinogen increases sodium reabsorption in the collecting duct
- d) angiotensin II increases water reabsorption in the proximal tubule

# Reabsorption Along The Tubule

Segment of Tubule	Substances	Hormone Regulation	Percent
Proximal Tubule	Glucose, H <sub>2</sub> O, Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>	Yes	65%
Descending Limb of LOH	H <sub>2</sub> O and minimal Na <sup>+</sup>	No	20 %
Ascending Limb of LOH	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup>	No	
Distal Tubule	Na <sup>+</sup> , K <sup>+</sup> , Cl <sup>-</sup> , Ca <sup>2+</sup>	Yes	14%
Collecting Duct	H <sub>2</sub> O, Na <sup>+</sup>	Yes	



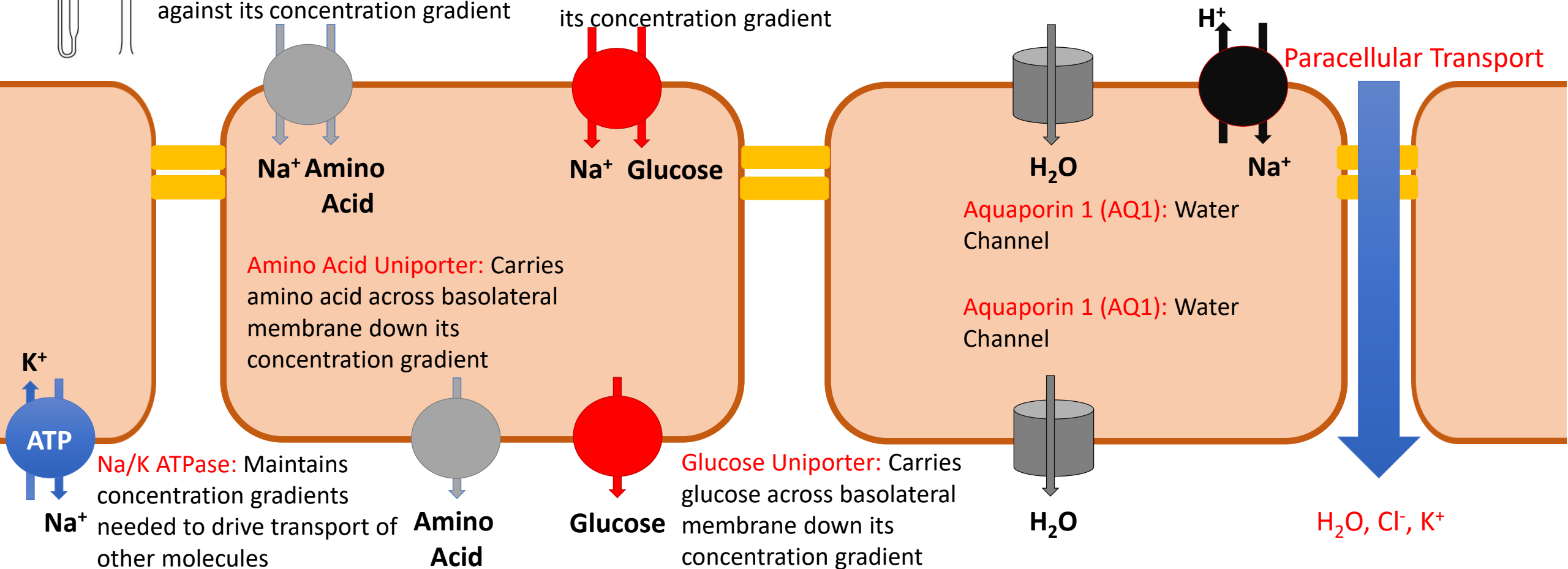


# Proximal Tubule

**Na<sup>+</sup>/Amino Acid Symporter:** Uses Na<sup>+</sup> gradient to carry an amino acid across luminal membrane against its concentration gradient

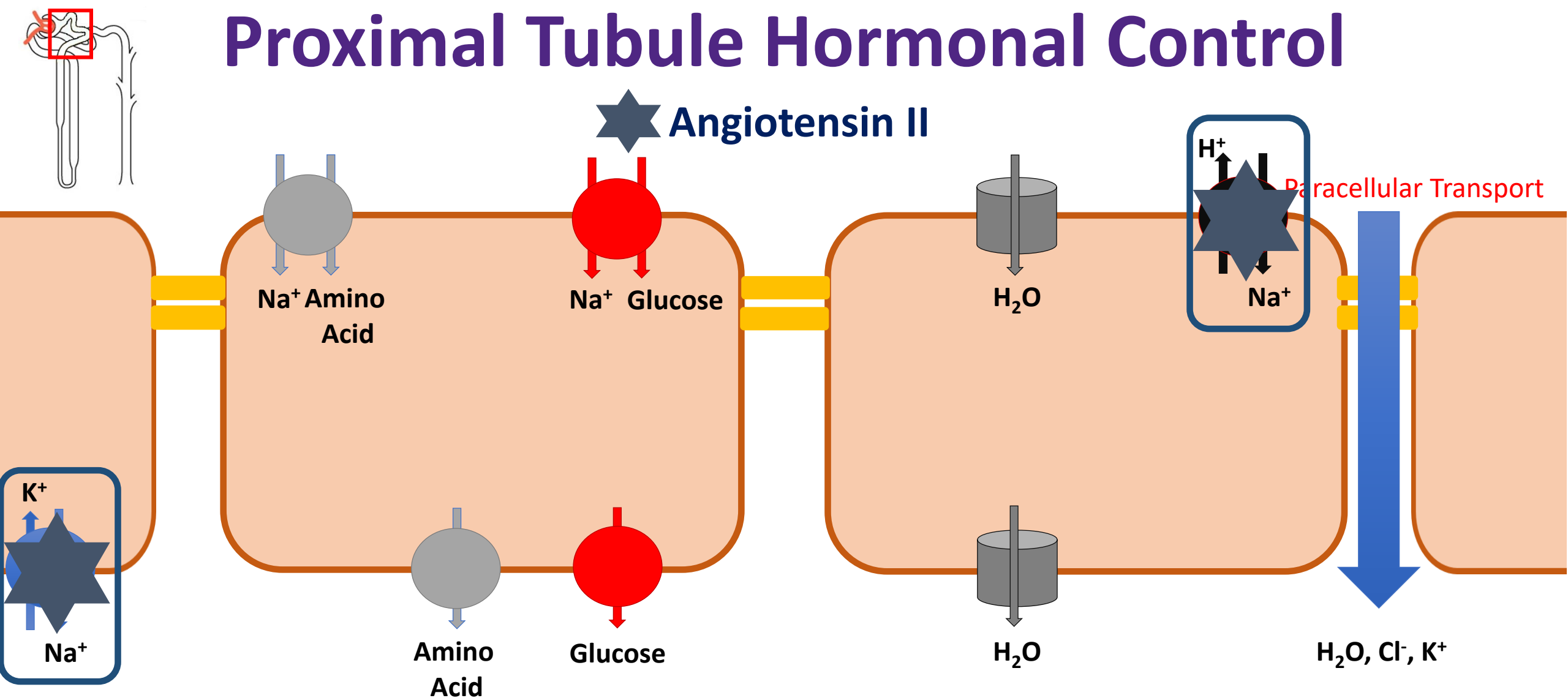
**Na<sup>+</sup>/Glucose Symporter:** Uses Na<sup>+</sup> gradient to carry glucose across luminal membrane against its concentration gradient

**Na/H+Antiporter/Exchanger:** Uses Na gradient to carry H<sup>+</sup> across luminal membrane into filtrate



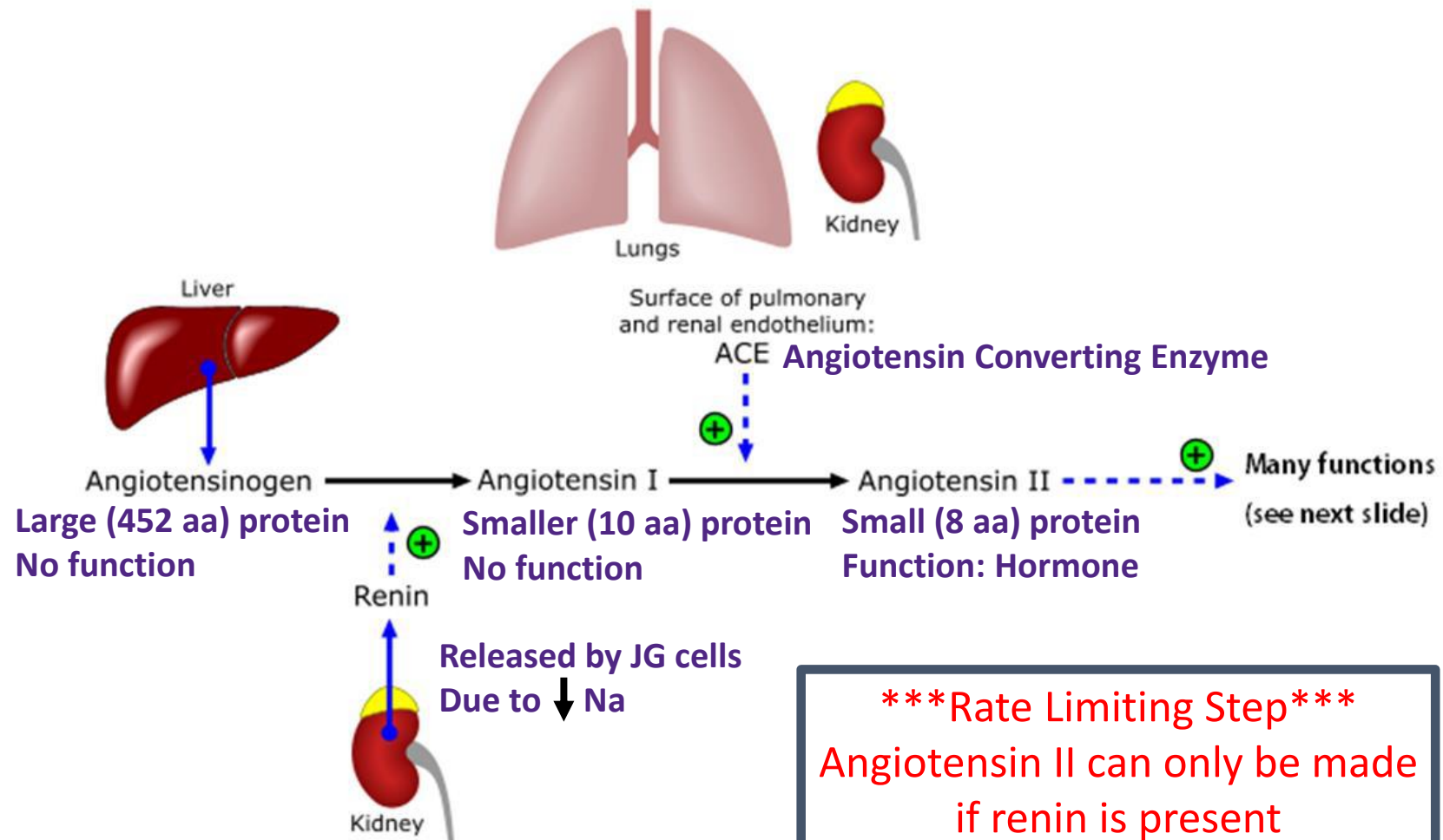
Blood vessel near proximal tubule

# Proximal Tubule Hormonal Control



Blood vessel near proximal tubule

# RAAS



# Angiotensin II

- Made by cleavage of:  
angiotensinogen  $\Rightarrow$  angiotensin I  $\Rightarrow$  angiotensin II
- Peptide hormone (= requires cell-surface receptor on luminal membrane)

## Stimulus

- Release of renin by JG cells due to  $\downarrow$   $\text{Na}^+$

## Goal

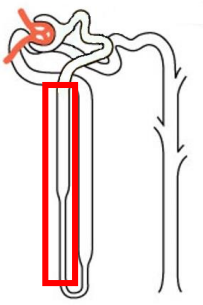
- Increase  $\text{Na}^+$  reabsorption in proximal tubule

## How

1. Increase activity of  $\text{Na}^+/\text{H}^+$  exchanger and  $\text{Na}^+/\text{K}^+$  ATPase in proximal tubule
2. Decrease GFR by constricting afferent arteriole

# Renin

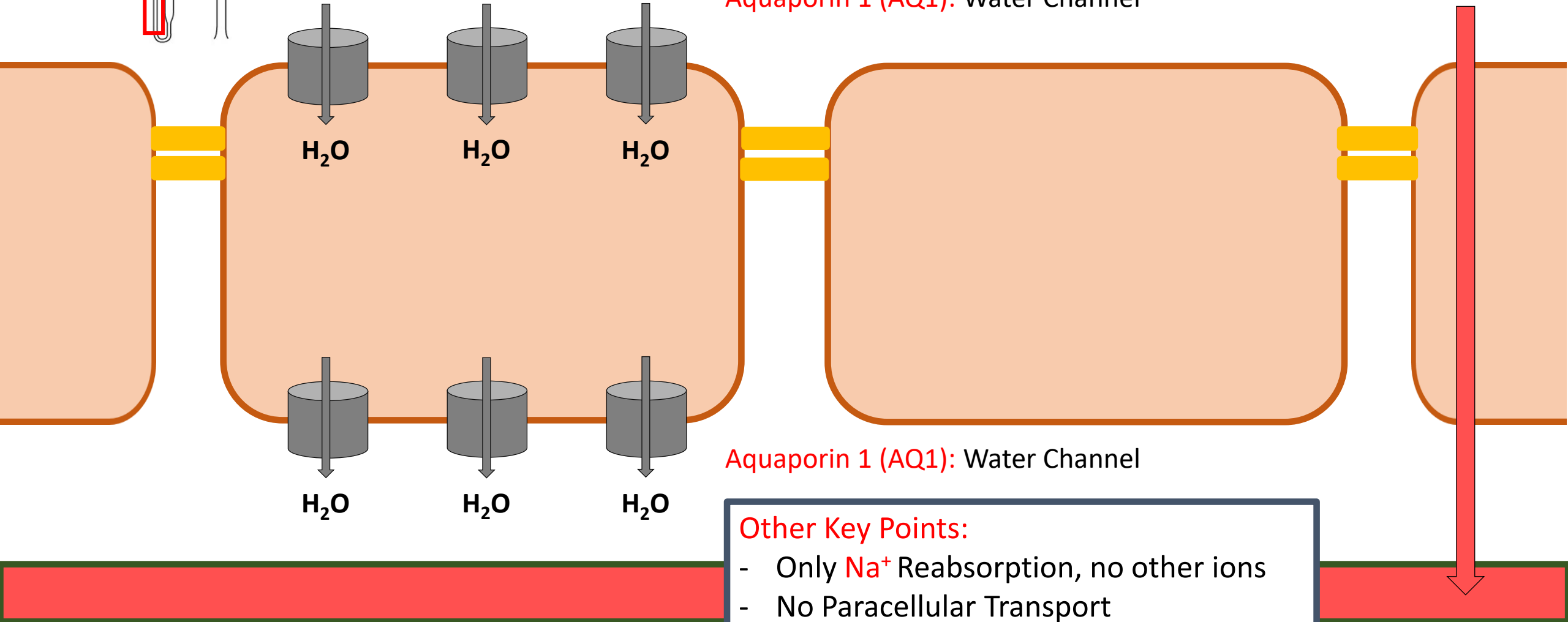
- Renin released due to low sodium levels



# Descending LOH

Overall Goal:  
**REABSORPTION**

Aquaporin 1 (AQ1): Water Channel



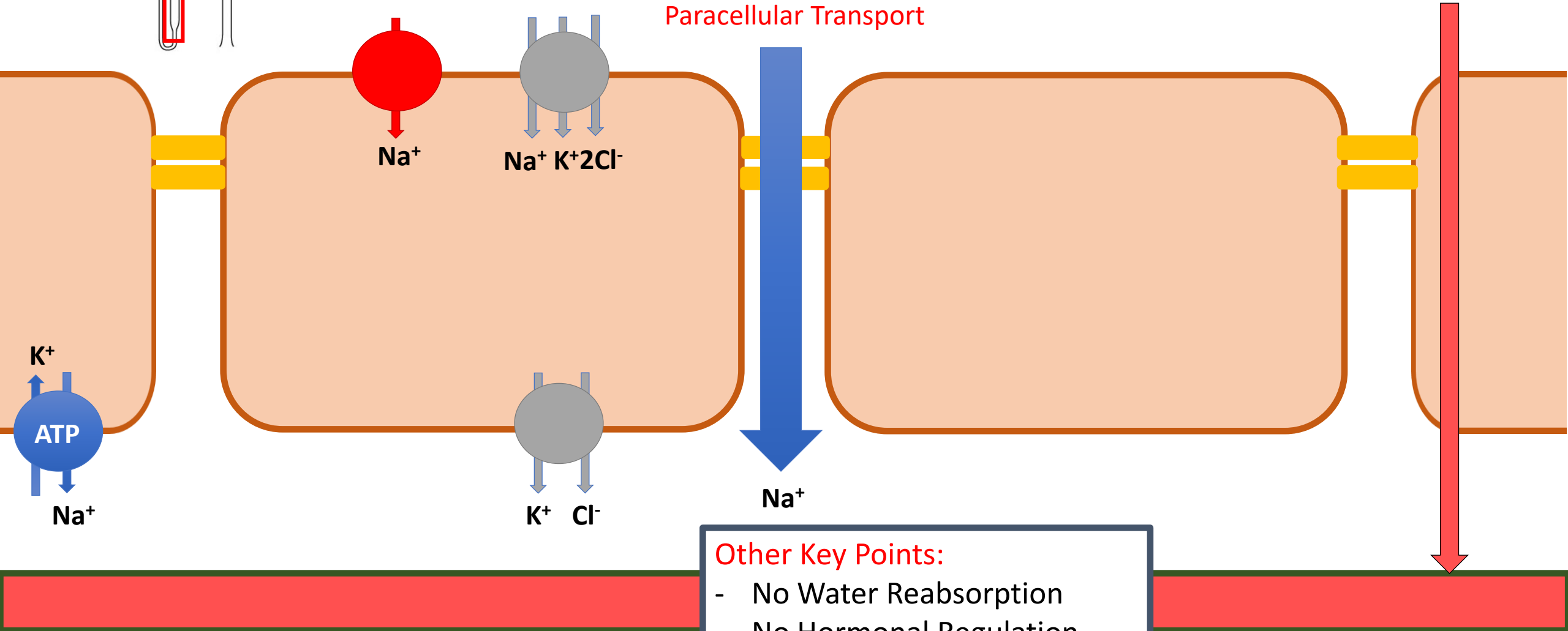
Aquaporin 1 (AQ1): Water Channel

## Other Key Points:

- Only  $Na^+$  Reabsorption, no other ions
- No Paracellular Transport
- No Hormonal Regulation

# Ascending LOH

Overall Goal:  
REABSORPTION



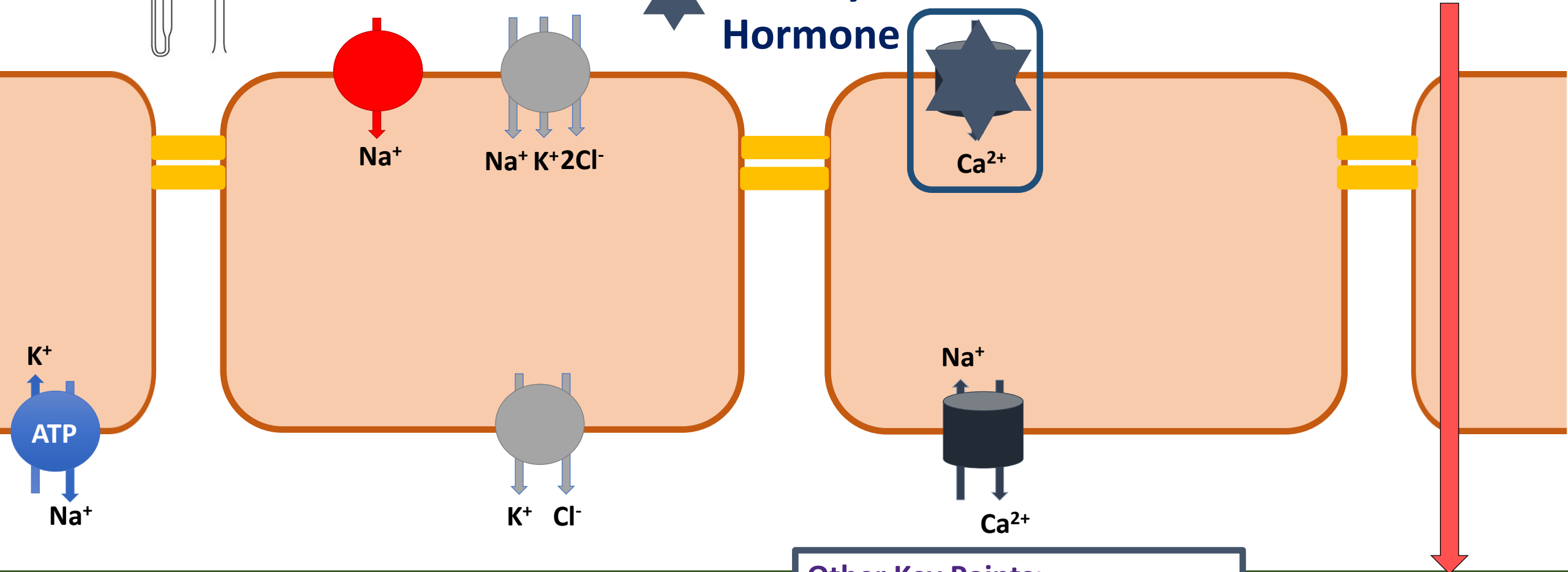
## Other Key Points:

- No Water Reabsorption
- No Hormonal Regulation

# Distal Convoluted Tubule

Overall Goal:  
**REABSORPTION**

★ Parathyroid  
Hormone



## Other Key Points:

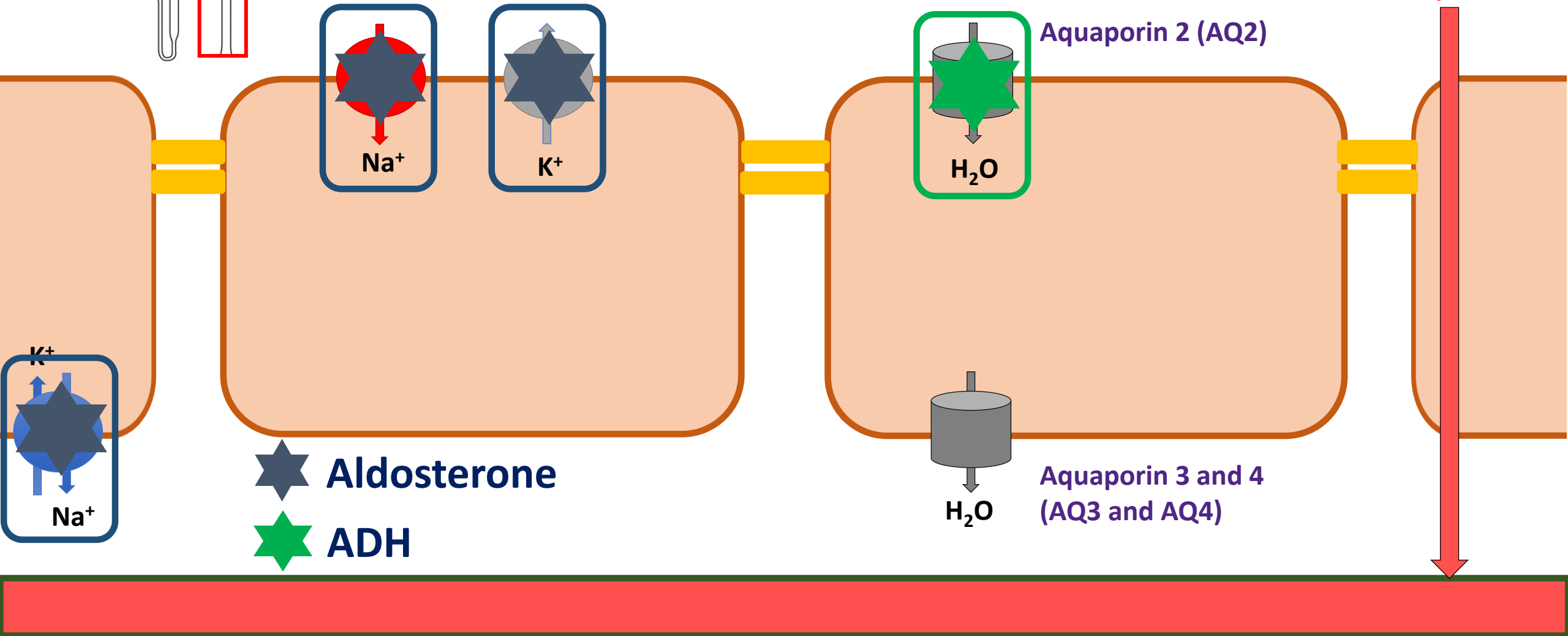
- No Water Reabsorption
- No Paracellular Transport

**Which of the following is a transporter that increases activity in the presence of aldosterone?**

- a) sodium/potassium ATPase in the proximal tubule**
- b) sodium/potassium ATPase in the collecting duct**
- c) sodium/hydrogen exchanger in the proximal tubule**
- d) sodium/hydrogen exchanger in the collecting duct**

# Collecting Duct

Overall Goal:  
Fine Tuning of  $\text{H}_2\text{O}$   $\text{Na}^+$   
Reabsorption



# Aldosterone

- Made by adrenal gland
- Steroid hormone (= intracellular receptor)

## Stimulus

- Angiotensin II, High  $K^+$ , ACTH

## Goal

- Increase  $Na^+$  reabsorption in collecting duct

## How

1. ↑  $Na^+$  and  $K^+$  channels in luminal membrane: by translocation and protein expression
2. ↑  $Na^+ / K^+$  ATPase activity and protein expression

# Anti-Diuretic Hormone (aka Vasopressin)

- Made by hypothalamus (by neuroendocrine cells)
  - stored/released by posterior pituitary
- Peptide hormone (= requires cell-surface receptor on basolateral membrane)

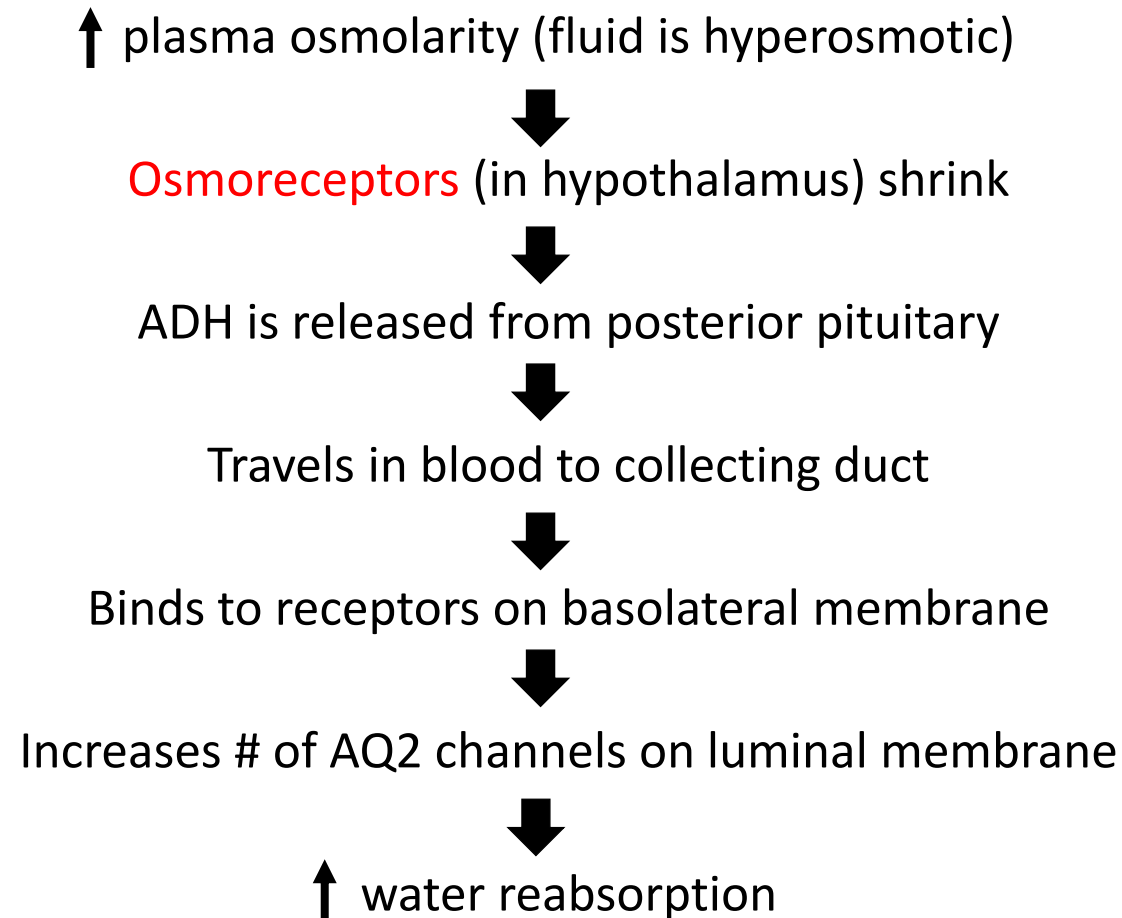
## Stimulus

High plasma osmolarity = Low ECF volume = Low BP

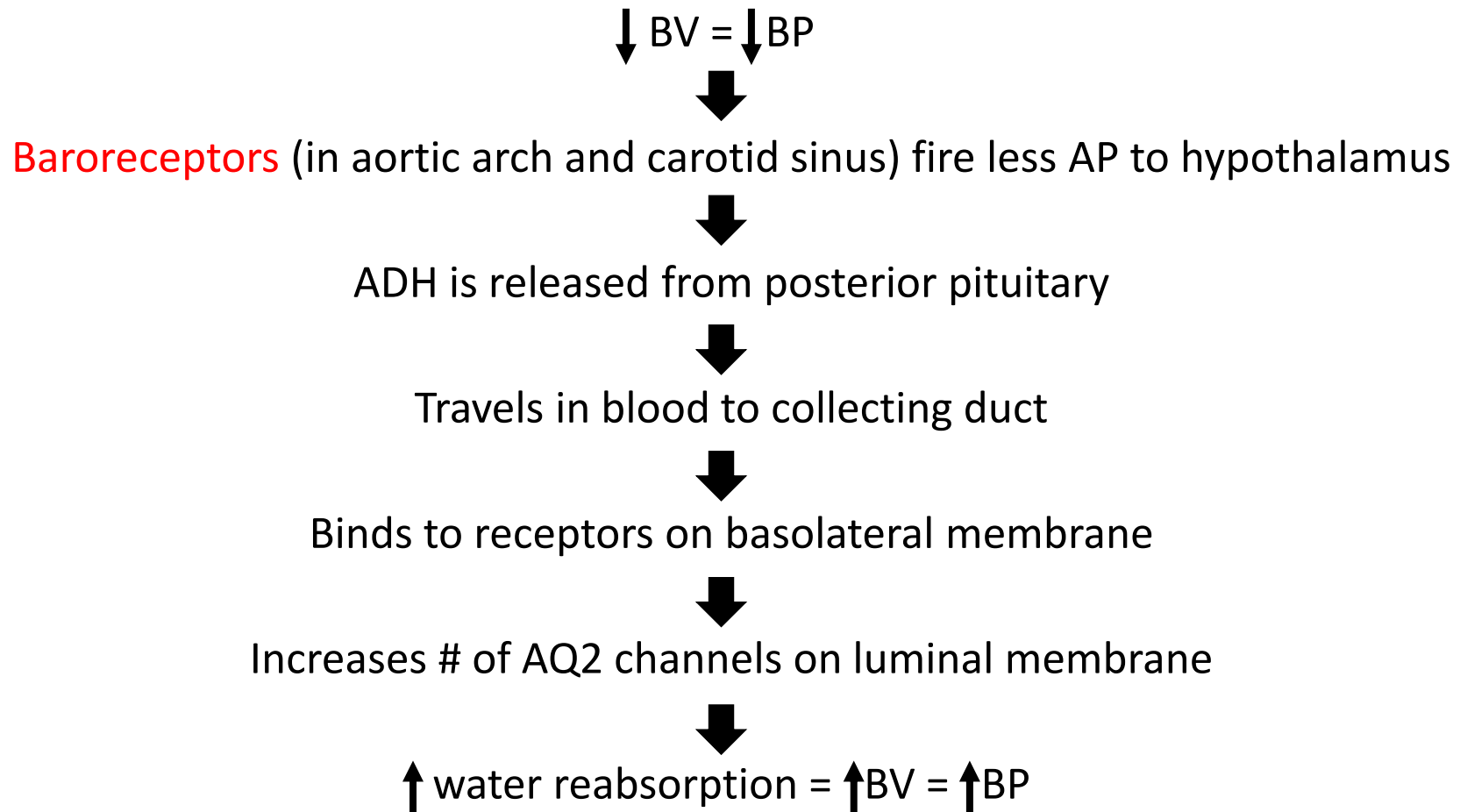
## Goal

↑ water reabsorption = ↑ ECF volume = ↑ BP

# Anti-Diuretic Hormone (aka Vasopressin)



# Anti-Diuretic Hormone (aka Vasopressin)



# Atrial Natriuretic Peptide (ANP)

- Made by **cardiac atrial cells**
- Peptide hormone

## Stimulus

- High blood pressure

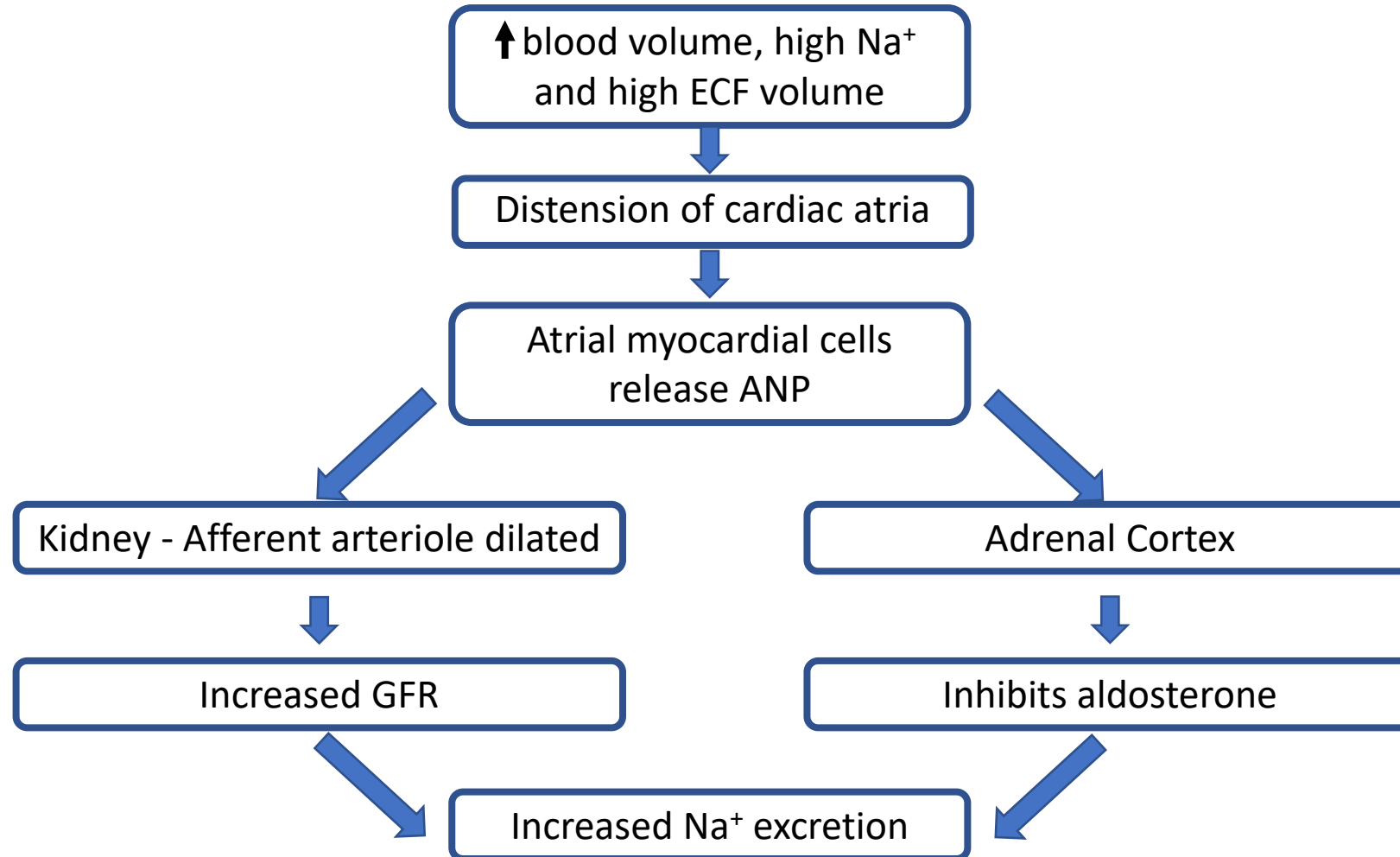
## Goal

- Decrease  $\text{Na}^+$  reabsorption

## How

- Inhibits aldosterone secretion by adrenal glands
- Dilates afferent arterioles → increasing GFR
  - This increases flow of filtrate through tubule, leads to increase in  $\text{Na}^+$  excretion

# Atrial Natriuretic Peptide (ANP)



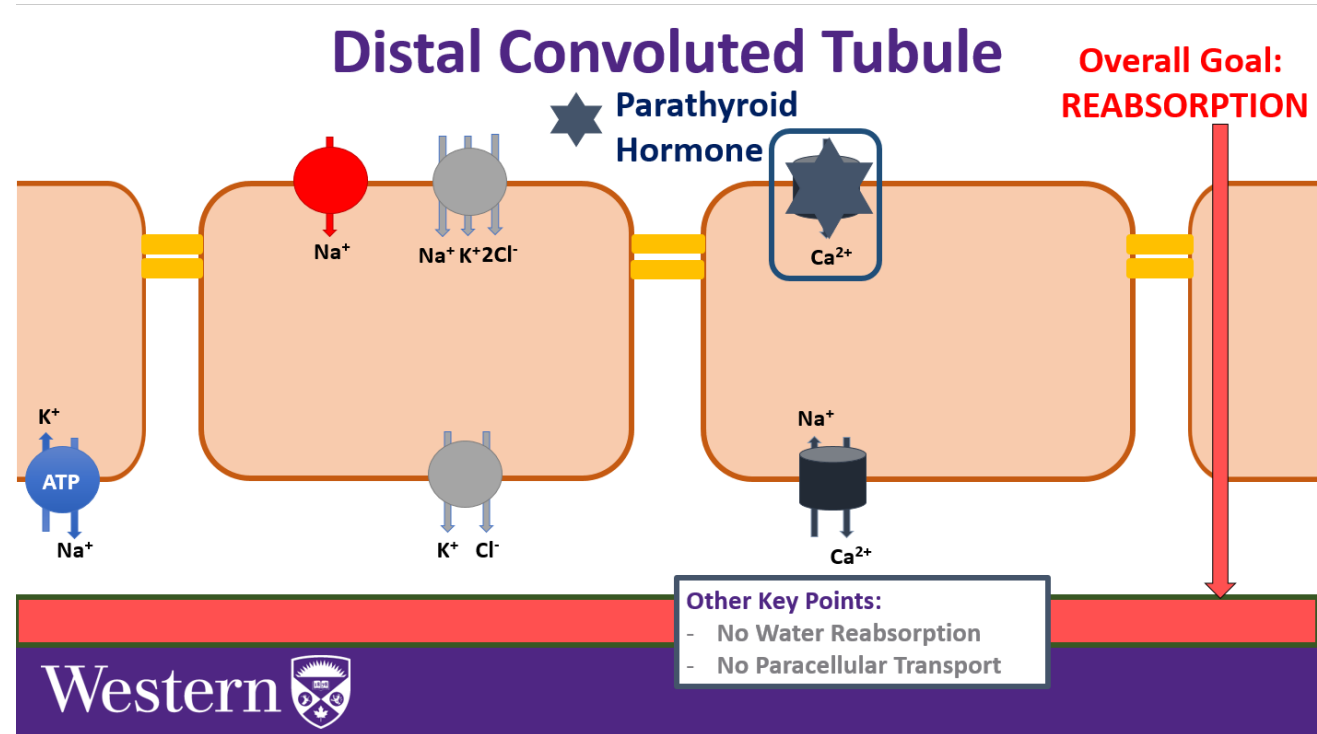
## Based on the following information, what segment of the tubule is being described?

- Incapable of paracellular transport
  - No water reabsorption
  - Some hormonal control
- 
- A. Proximal tubule
  - B. Ascending limb of the loop of Henle
  - C. Distal convoluted tubule
  - D. Collecting duct

# Based on the following information, what segment of the tubule is being described?

- Incapable of paracellular transport
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- A. Proximal tubule
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- D. Collecting duct



# Summary of Transport

	Proximal Tubule	Descending Limb	Ascending Limb	Distal Tubule	Collecting Duct
Goal	Reabsorption of everything	Water reabsorption	Ion reabsorption	Ion reabsorption	Fine tuning (water and Na)
Water	Yes	Yes	No	No	Yes
Ions	Yes (Na <sup>+</sup> , Cl <sup>-</sup> , K <sup>+</sup> )	Minimal (Na <sup>+</sup> )	Yes (Na <sup>+</sup> , Cl <sup>-</sup> , K <sup>+</sup> )	Yes (Na <sup>+</sup> , Cl <sup>-</sup> , K <sup>+</sup> , Ca <sup>2+</sup> )	Yes (Na <sup>+</sup> )
Paracellular Transport	Yes	No	Yes	No	No
Hormone Regulation	Angiotensin II	-	-	PTH	Aldosterone ADH

# Summary of Hormonal Regulation

Hormone	Made By	Hormone Type	Stimulus	Response
<b>Renin</b>	Kidneys	N/A	Increased sodium	N/A
<b>Angiotensin II</b>	Angiotensin Converting Enzyme	Peptide	Renin – released due to low sodium	Increase sodium reabsorption in proximal tubule: <ol style="list-style-type: none"> <li>1. Increase activity of <math>\text{Na}^+/\text{H}^+</math> exchanger and <math>\text{Na}^+/\text{K}^+</math> ATPase</li> <li>2. Constrict afferent arteriole (decreasing GFR)</li> </ol>
<b>Aldosterone</b>	Adrenal Gland	Steroid	Angiotensin II and high $\text{K}^+$	Increase sodium reabsorption in collecting duct: <ol style="list-style-type: none"> <li>1. Increase <math>\text{Na}^+</math> and <math>\text{K}^+</math> channels in luminal membrane</li> <li>2. Increase activity of <math>\text{Na}^+/\text{K}^+</math> ATPase</li> </ol>
<b>Atrial Natriuretic Peptide</b>	Cardiac atrial cells	Peptide	High blood pressure	Decrease sodium reabsorption: <ol style="list-style-type: none"> <li>1. Inhibit aldosterone secretion from adrenal glands</li> <li>2. Dilates afferent arteriole (increasing GFR)</li> </ol>

# Chapter 9: Respiratory

## 14 Questions on Exam

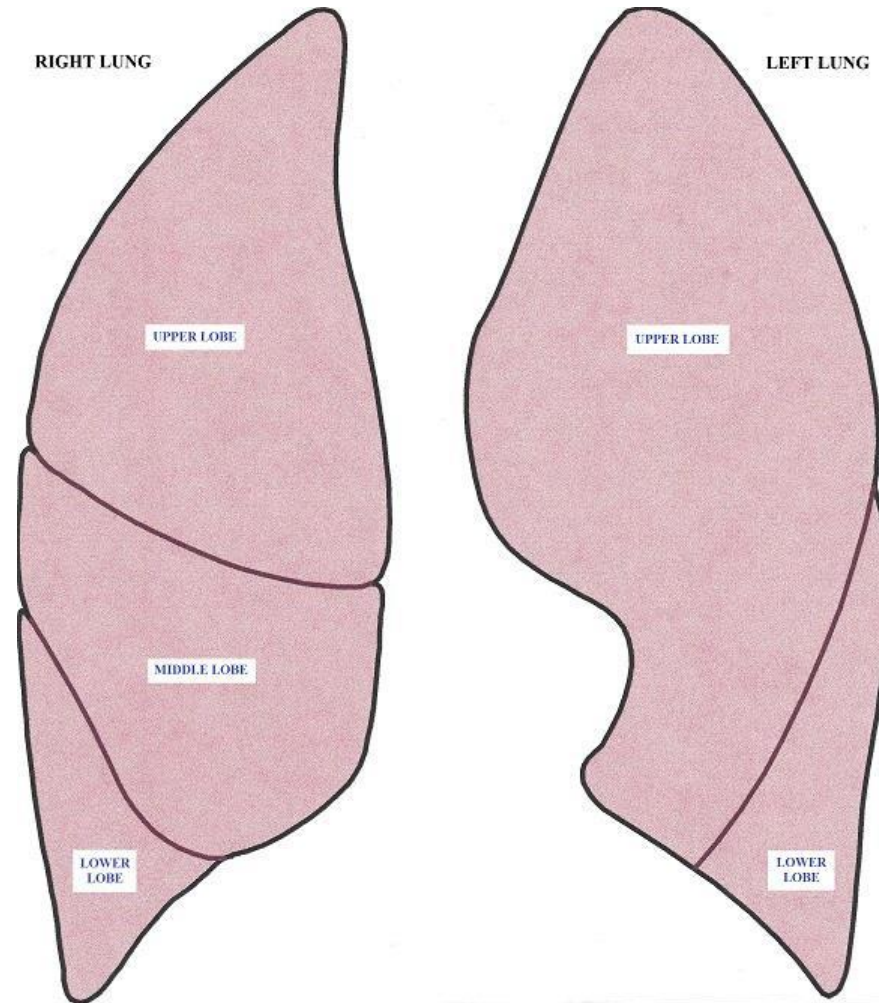
Professor: Dr. Beye

# Overview of Chapter

1. Anatomy of respiratory system
2. Lung volume measurements
3. Partial pressures and gas exchange
4. Carbon dioxide transport

# Lung Anatomy

Right Lung  
3 Lobes



Left Lung  
2 Lobes

Why? The  
heart sits on  
the left side  
of thoracic  
cavity

# Lung Anatomy

## Muscles

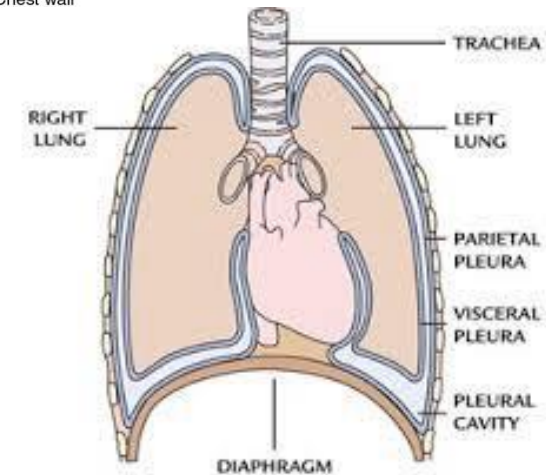
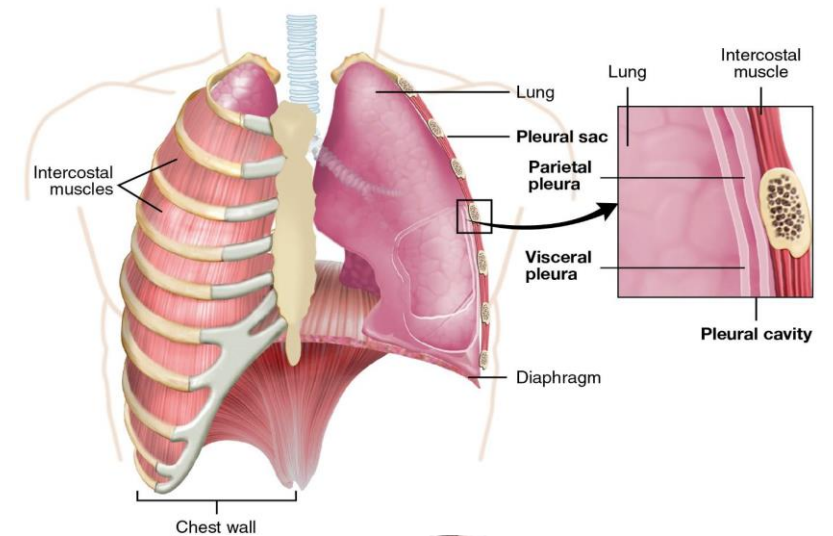
- Intercostal muscles (between ribs)
- Diaphragm (bottom)

## Pleural Layers

- Visceral pleura (against lungs)
- Intrapleural space/cavity
- Parietal pleura (against chest wall)

Visceral = Very close

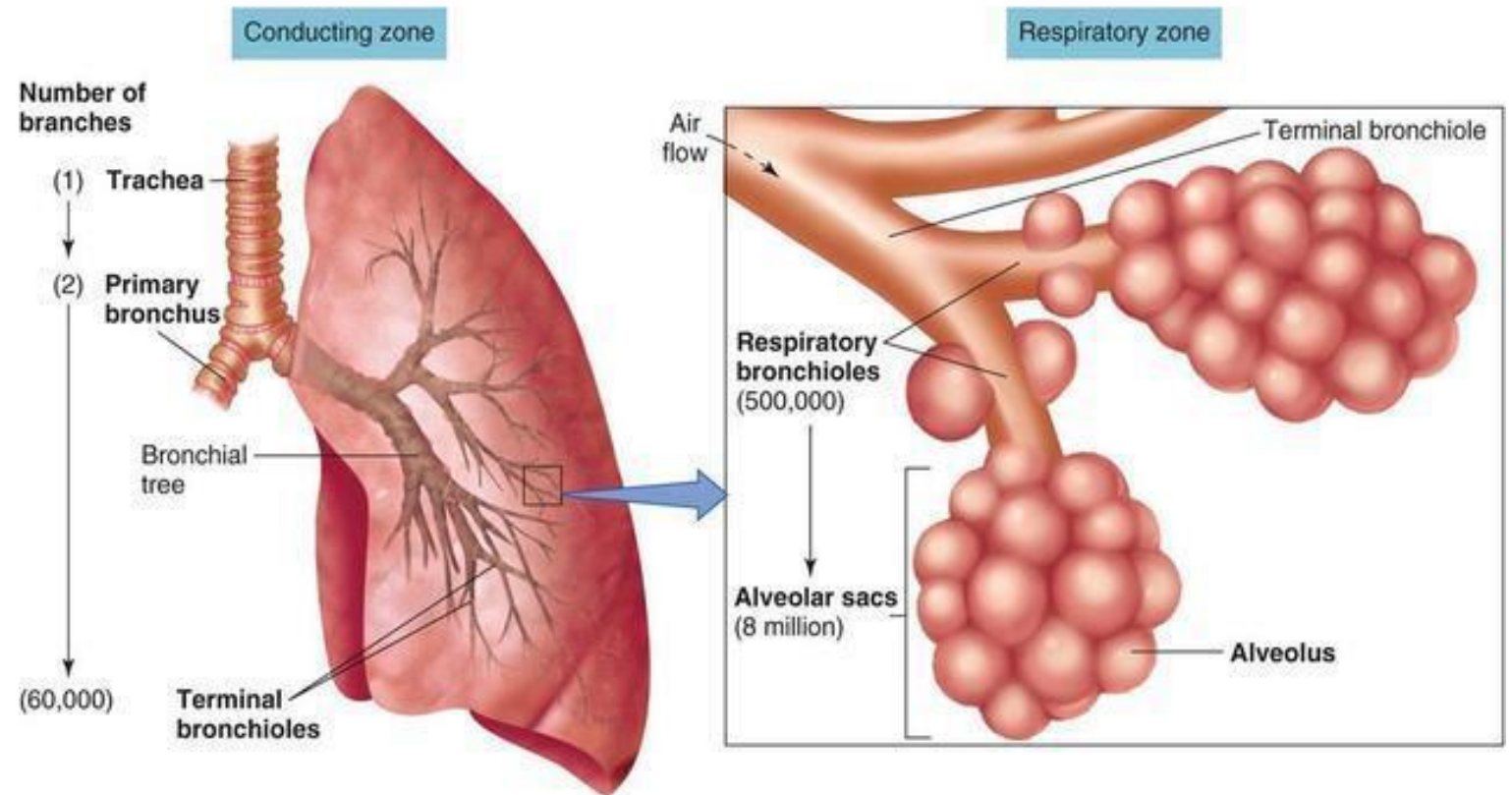
Parietal = Pretty close



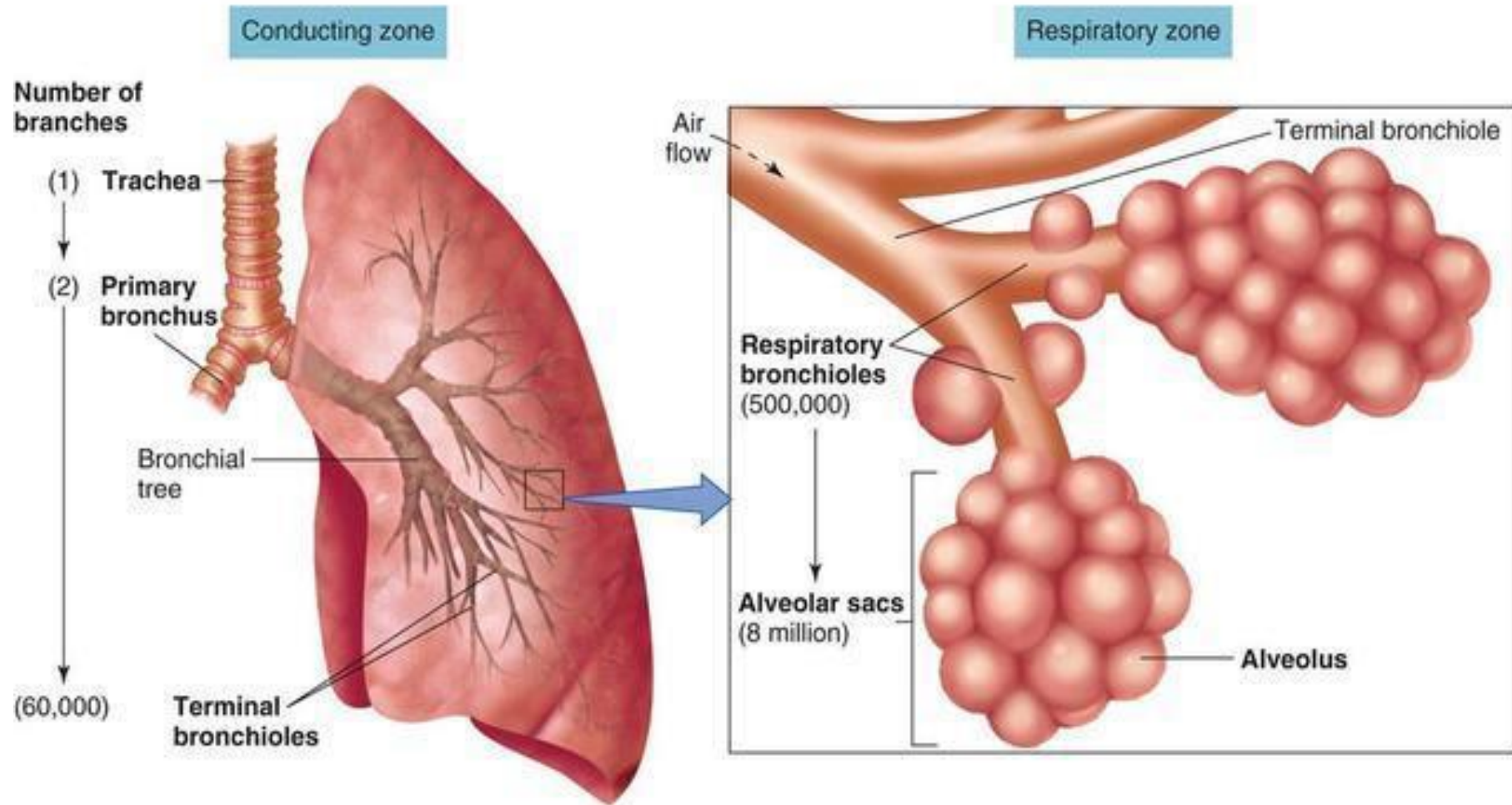
# Lung Anatomy

Respiratory tract can be divided into two sections:

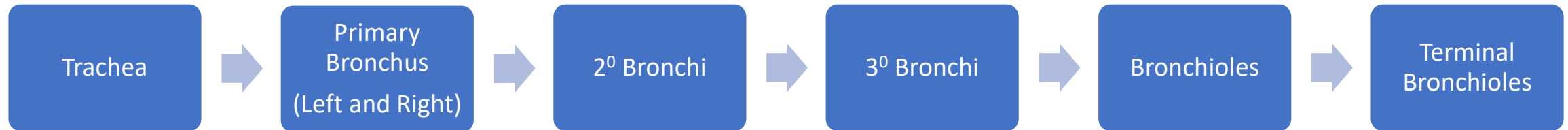
1. Conducting Zone
2. Respiratory Zone



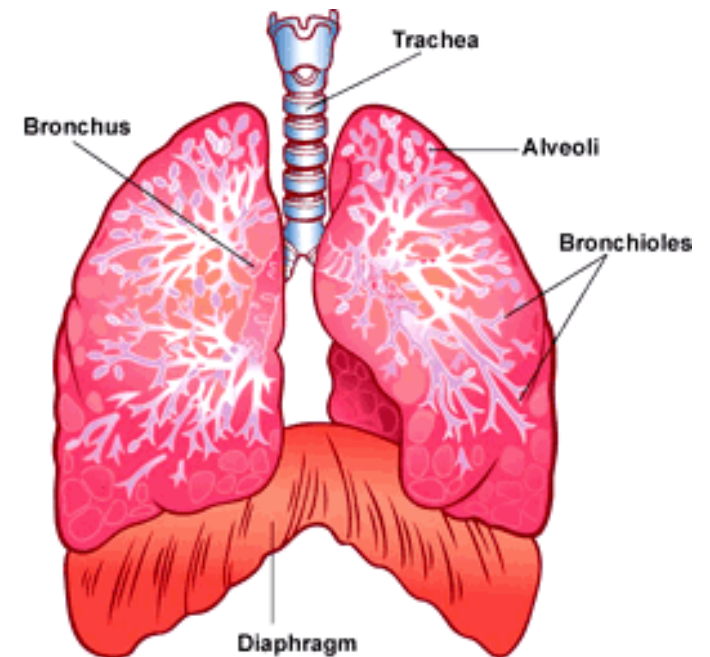
# Zones of Respiratory Tract



# Conducting Zone



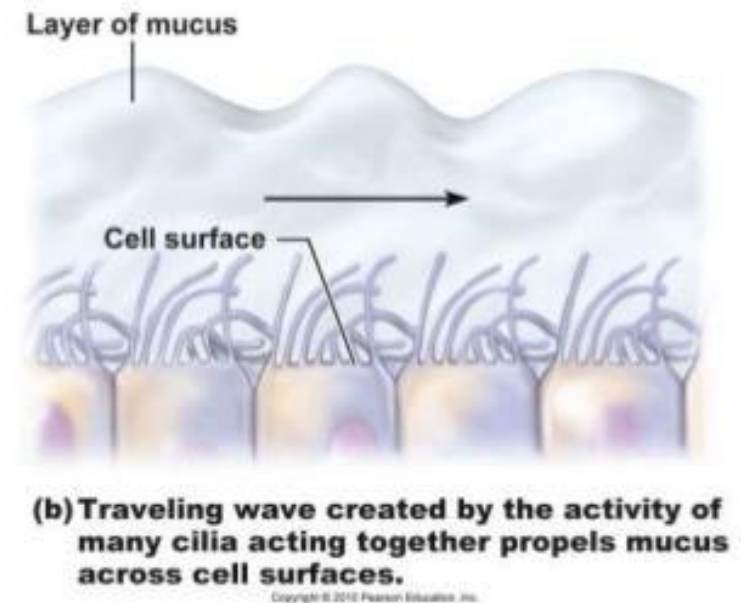
“Conducting zone **terminates** at **terminal** bronchioles”



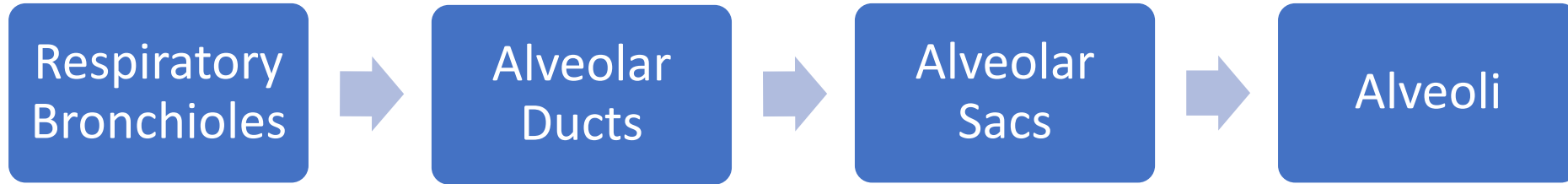
# Conducting Zone

## Functions

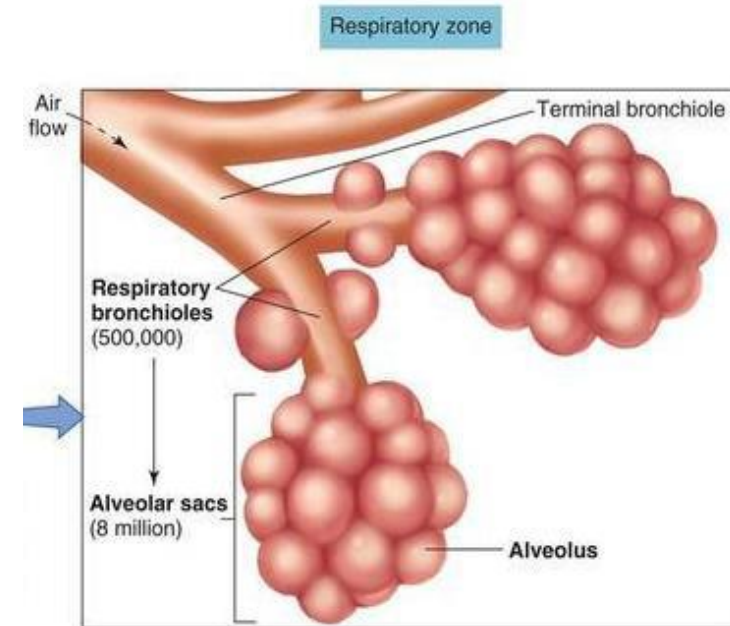
- Transport air to lungs
- Filter, warm and moisten air
- Microbial defense:
  - Bronchial epithelial cells are ciliated
  - **Cilia** sweeps mucus with trapped micro-organisms towards the trachea
  - Smoking reduces function of cilia



# Respiratory Zone



“**Respiratory** zone begins at **respiratory** bronchioles”



**The walls of the alveoli are composed of two types of cells, type I and II. The function of type II is to \_\_\_\_\_.**

- A. Secrete surfactant
- B. Trap dust and other debris
- C. Replace mucus in the alveoli
- D. Protect the lungs from bacterial invasion

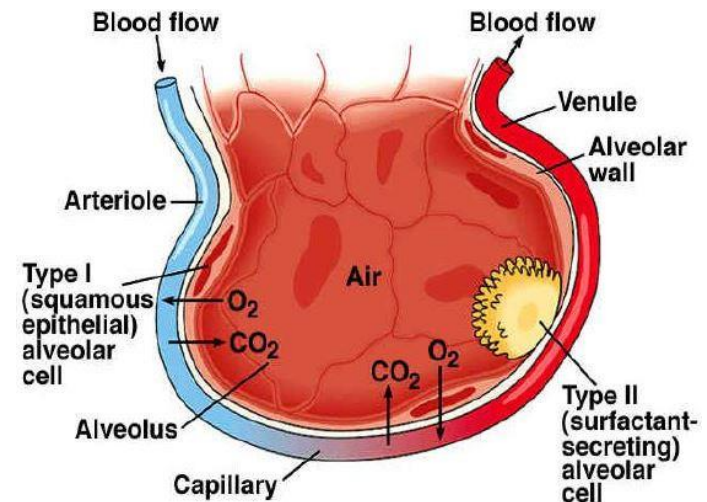
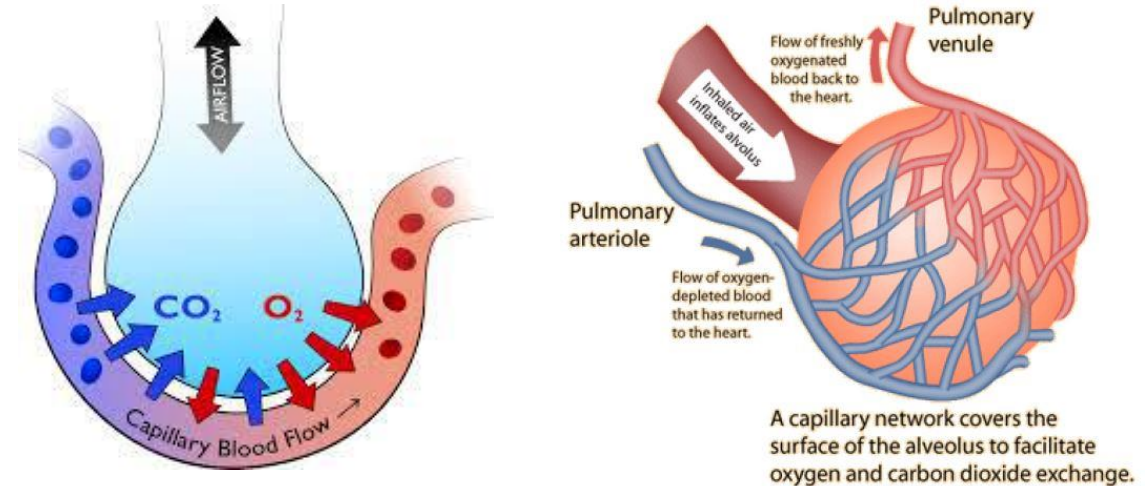
# Respiratory Zone

## Function

- Gas exchange in the alveoli at the blood gas barrier
  - $\text{CO}_2$  leaves blood supply into air
  - $\text{O}_2$  leaves air into blood supply

## Cells

- **Type 1 cell:** flat and thin cells that form alveolar wall and allow for easy gas exchange between alveoli and capillary
- **Type 2 cell:** secrete surfactant
- **Macrophages:** destroy microorganisms



**A 200 pound 20 year old man has a tidal volume of 500 mL and a respiratory rate of 10 breaths per minute. What is his anatomical dead space ventilation per minute?**

- A. 5 liters
- B. 2 liters
- C. 3000 ML
- D. 200 mL

$$V_D = \text{Weight} \times \text{Respiratory Rate}$$

# Ventilation Calculations

- **Tidal Volume**: Amount of air entering lungs in one breath during normal inhalation
- **Respiratory Rate**: # of breaths per minute
- **Pulmonary Ventilation ( $V_E$ )**: Amount of air entering lungs (both zones) per minute

$$V_E = \text{Tidal Volume} \times \text{Respiratory Rate}$$

- **Anatomical Dead Space Ventilation ( $V_D$ )**: Amount of air not involved in gas exchange (Anatomical dead space = 1 mL/pound)

$$V_D = \text{Weight} \times \text{Respiratory Rate}$$

- **Alveolar Ventilation ( $V_A$ )**: Amount of air entering only the respiratory zone per minute

$$V_A = V_E - V_D$$

# Ventilation Calculations

Example: For a 150 lb individual with respiratory rate of 30 breaths/min and tidal volume of 200 mL/breath

$$\begin{aligned} V_E &= \text{Tidal Volume} \times \text{Respiratory Rate} \\ &= 200 \times 30 \\ &= 6000 \text{ mL/min} \end{aligned}$$

$$\begin{aligned} V_D &= \text{Weight} \times \text{Respiratory Rate} \\ &= 150 \times 30 \\ &= 4500 \text{ mL/min} \end{aligned}$$

$$\begin{aligned} V_A &= V_E - V_D \\ &= 6000 - 4500 \\ &= 1500 \text{ mL/min} \end{aligned}$$

By changing your pattern of breathing, you can alter how much air is actually available for gas exchange ( $V_A$ )

**You head out for a run. About 5 minutes in, you notice you are breathing pretty hard. What is happening in your thoracic cavity?**

- a) your external intercostals are contracting during exhalation
- b) your diaphragm is relaxing during inhalation
- c) your intrapulmonary pressure will match the atmospheric pressure during exhalation
- d) your internal intercostal muscles will pull your ribcage down and inwards during exhalation

# Inhalation

Diaphragm and external intercostal muscles contract (Diaphragm moves down and intercostals pull ribcage up and out)



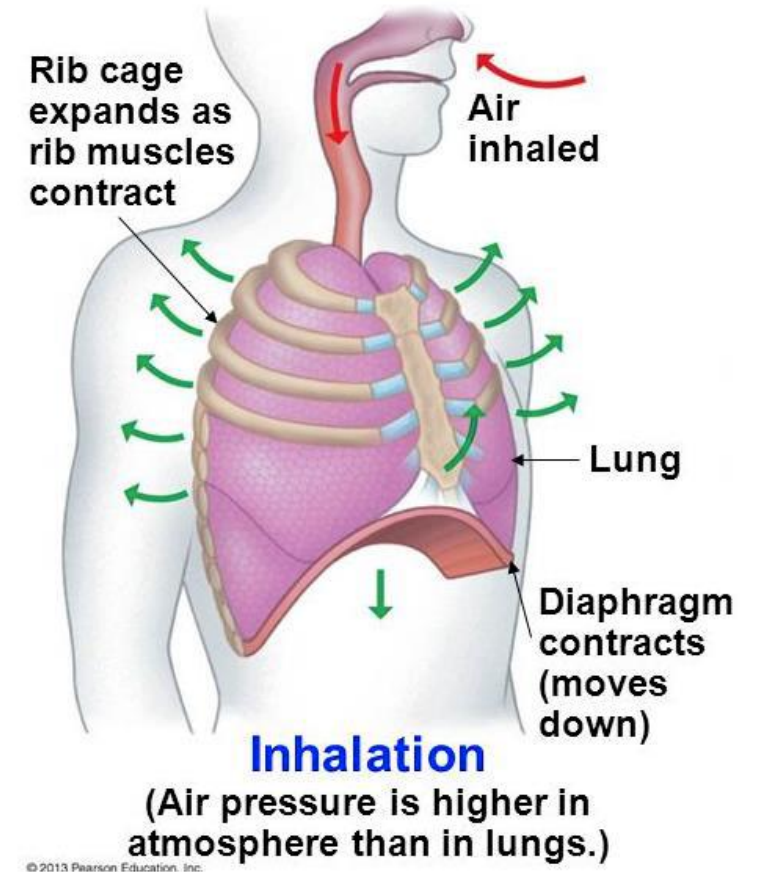
Thoracic cavity expands (Increase volume)



Decrease intrapulmonary pressure by Boyle's law ( $<760$  mmHg)



Air moves in



# Exhalation (Passive)

Diaphragm and external intercostal muscles relax (Diaphragm moves up and ribcage gets smaller)



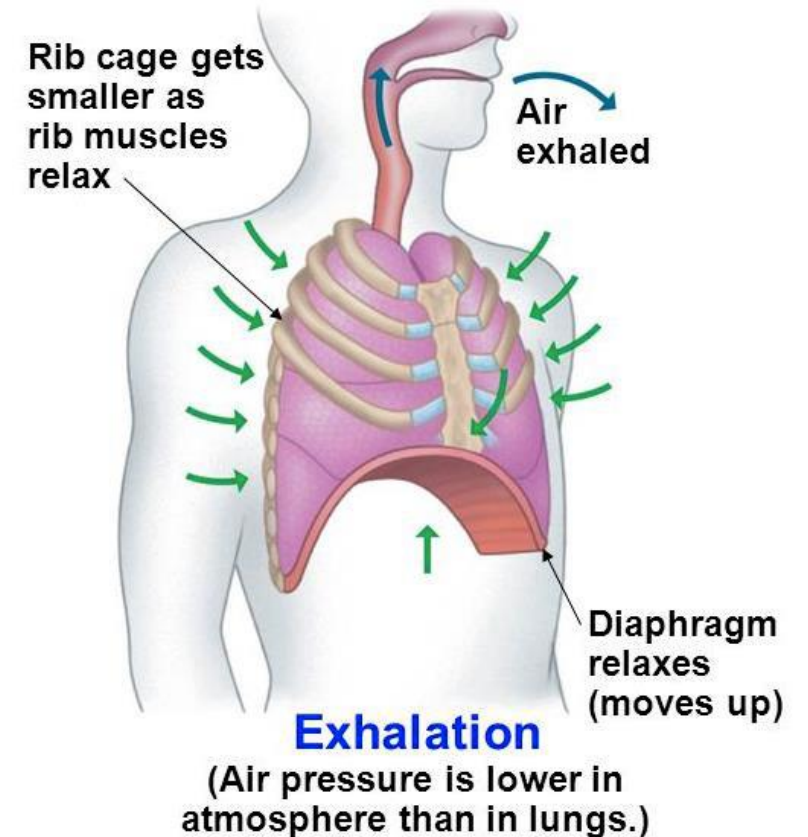
Thoracic cavity decreases in volume



Increase intrapulmonary pressure by Boyle's law ( $>760$  mmHg)



Air moves out



# Exhalation (Active - Exercise)

Diaphragm and external intercostal muscles relax

Internal intercostals, obliques and rectus abdominus contract



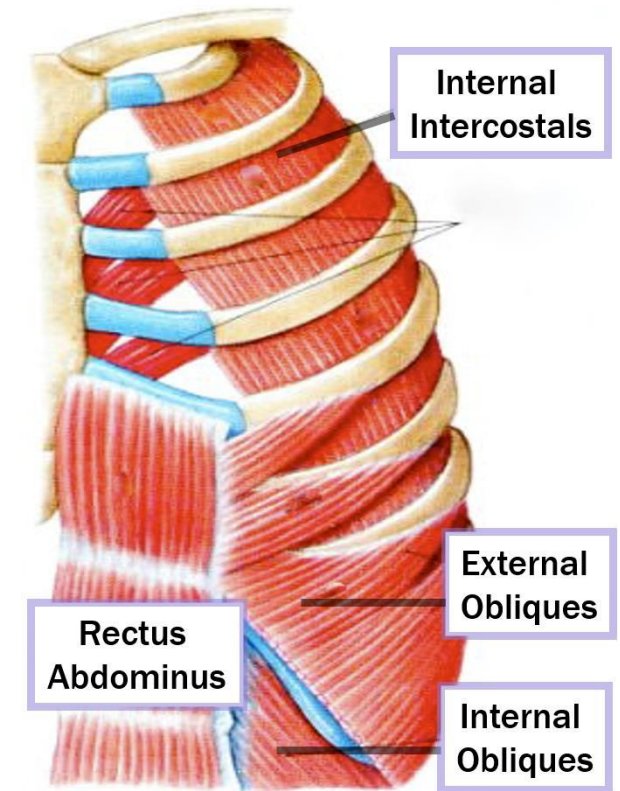
Thoracic cavity decreases in volume



Increase intrapulmonary pressure by Boyle's law  
( $>760$  mmHg)



Air moves out

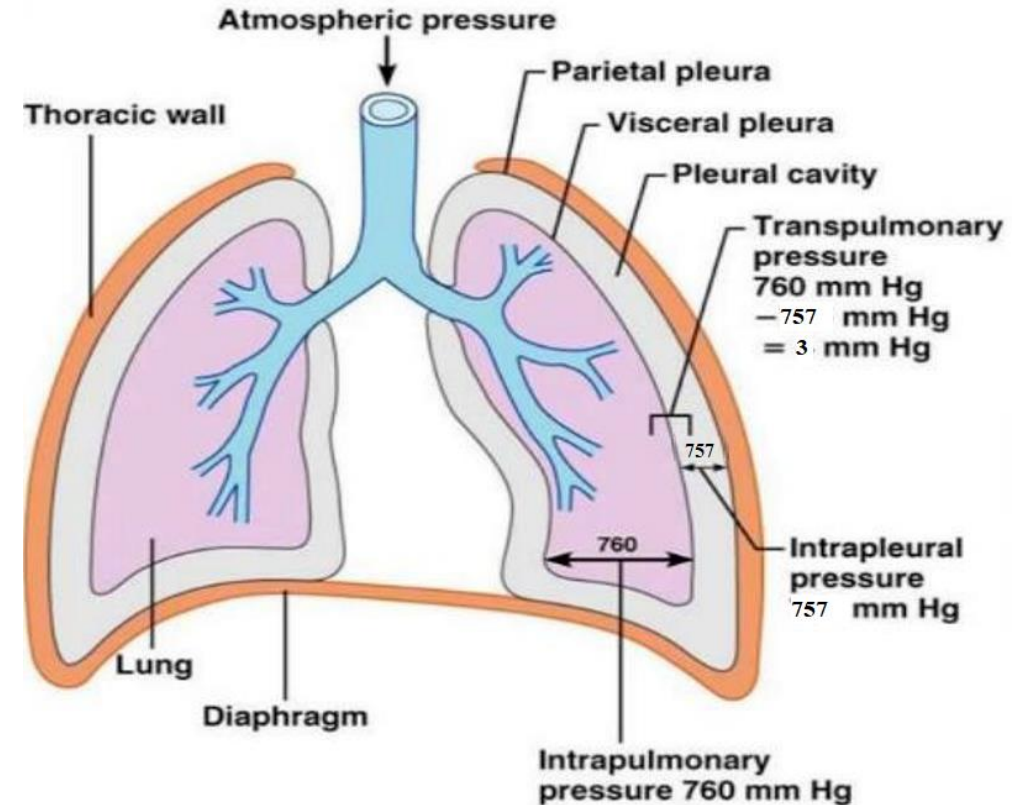


# During active exhalation (exercise), which of the following muscles are contracting?

1. Obliques
  2. Diaphragm
  3. Internal intercostals
  4. External intercostals
- 
- A. 1, 2 and 3 are correct
  - B. 1 and 3 are correct
  - C. 2 and 4 are correct
  - D. Only 4 is correct
  - E. All are correct

# Intrapleural and Transpulmonary Pressure

- **Intrapleural Pressure:** Pressure in intrapleural space; 757 mmHg
  - Prevents lung from collapsing at the end of expiration
  - Allows for easy expansion of the lung
- **Transpulmonary Pressure:** Pressure across the lung; +3 mmHg
  - Intrapulmonary Pressure (760) – Intrapleural Pressure (757)

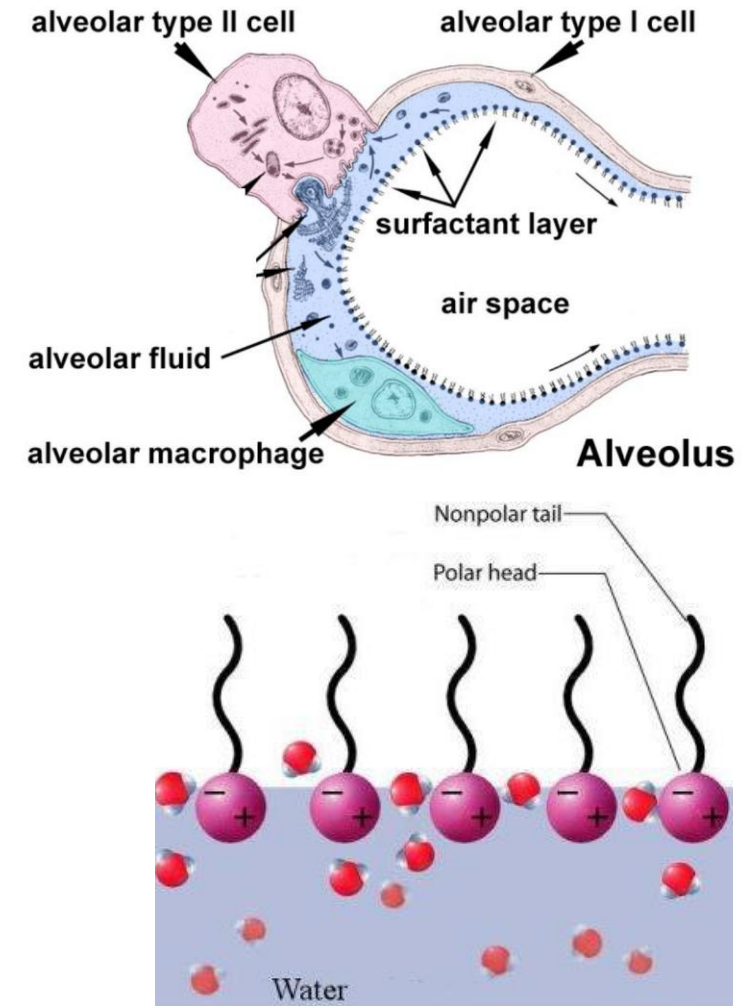


# Lung Compliance

- Compliance is the “stretchability” of the lung
- Compliance = Change in volume / Change in pressure
- Affected by 2 factors:
  1. Elastic tissue of lungs (33%)
    - Caused by presence of **elastin** and **collagen**
  2. Surface tension in alveoli (66%)
    - **Surfactant** reduces surface tension
- An increase in these 2 factors decreases compliance and increases likelihood of lung collapse

# Pulmonary Surfactant

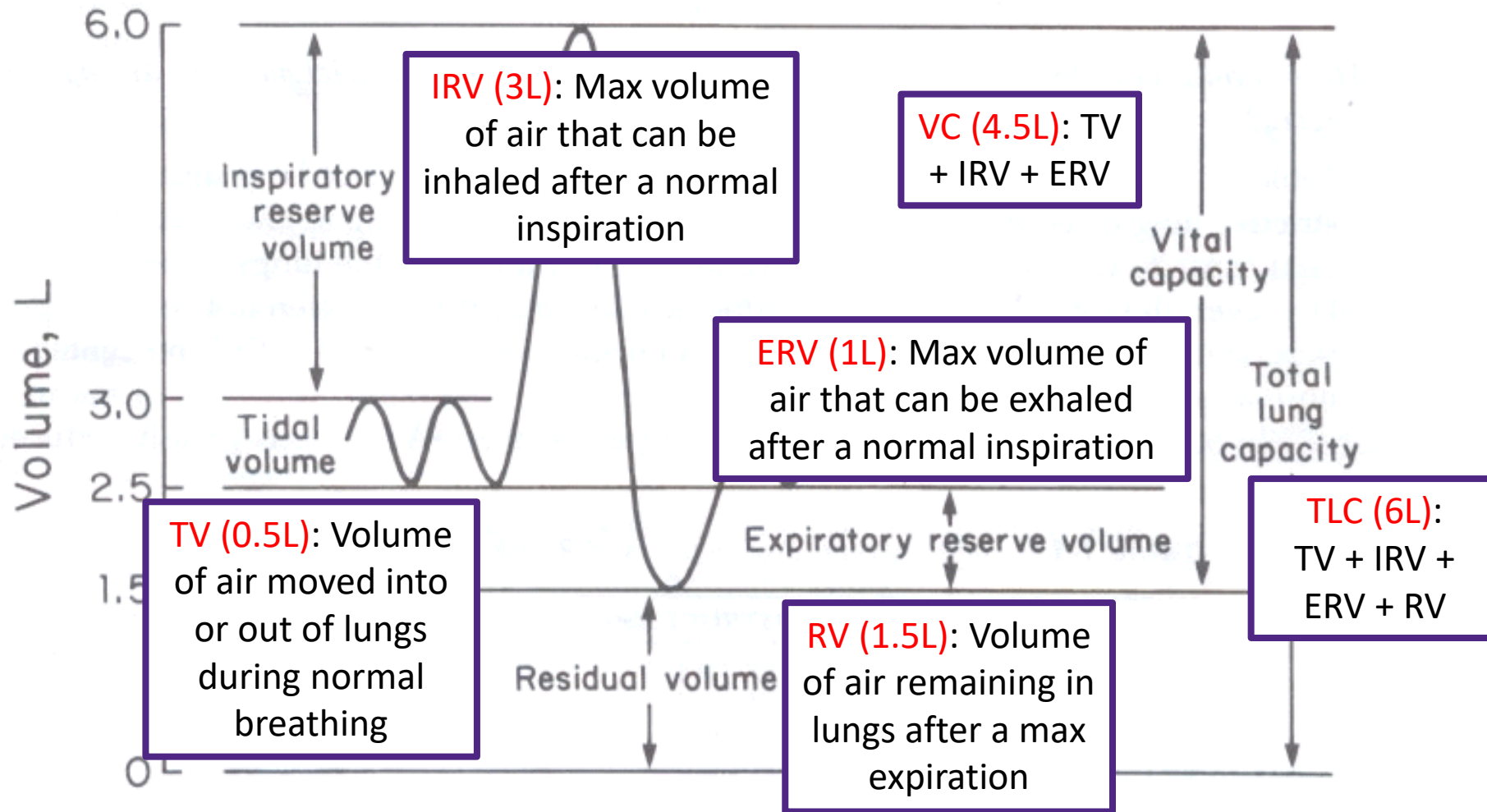
- Released by **type 2 cells**
- Layer is spread across air-water interface in alveoli
- **Surfactant** = phospholipids + proteins
- Phospholipids: hydrophilic head towards water; hydrophobic head towards air
- **Proteins**: help with microbial defense
- Functions:
  1. **Reduce surface tension**
    - Improve compliance and prevent alveolar collapse
  2. **Improves microbial defense function**
    - Proteins help identify foreign particles for macrophages
- **Neonatal respiratory distress syndrome (nRDS)**: premature infants lack mature surfactant system, which leads to poor lung function, alveolar collapse and hypoxemia (treatment = surfactant)



**If in one breath a person takes in a larger tidal volume than they normally would, which one of the following statements would be correct?**

- a) they are likely be breathing in less than 500 mL (0.5 litres)
- b) they would have a smaller IRV (inspiratory reserve volume)
- c) they would have a larger total lung capacity
- d) they would have a smaller FEV1

# Spirometry

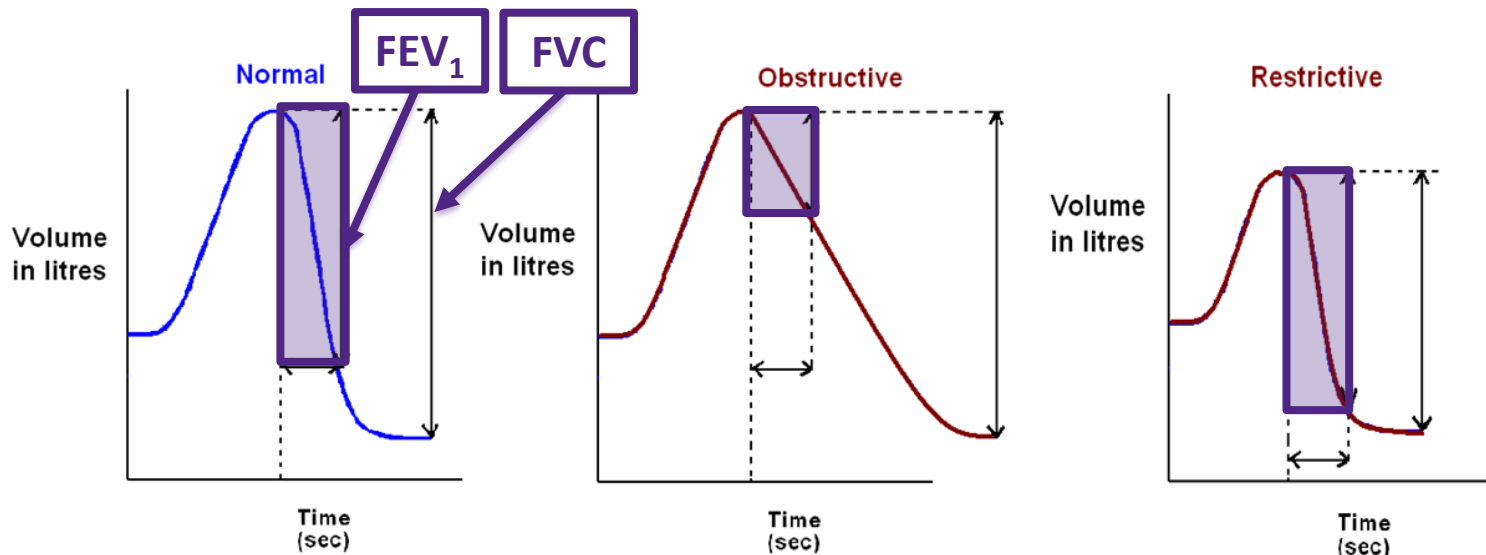


# What is true of a restrictive lung disease?

- A. Emphysema is an example of a restrictive lung disease
- B. Lungs can become floppy due to Loss of elastin
- C. FEV1 decreases
- D. FVC decreases

# Other Lung Measurements

- **Forced vital capacity (FVC)**: how much air a person can exhale as fast as possible during a forced breath
- **Forced expiratory volume ( $FEV_1$ )**: volume of exhalation over 1 second



Normal	Obstructive	Restrictive
FEV1	Decreases	No Change
FVC	No Change	Decreases
$FEV1/FVC = 80\%$	$FEV1/FVC < 80\%$	$FEV1/FVC > 80\%$

# Lung Diseases: Obstructive

- Air flow obstruction during exhalation
- Diameter of bronchioles decreases (lumen constricted)

	Cause	Effect
<b>Asthma</b>	Spasms triggered by exercise, air pollution and allergies	<ul style="list-style-type: none"><li>• Airway inflammation and hyper-responsiveness</li></ul>
<b>Chronic Bronchitis</b>	Smoking	<ul style="list-style-type: none"><li>• Excessive mucus and inflammation</li></ul>
<b>Emphysema</b>	Smoking	<ul style="list-style-type: none"><li>• Alveolar wall break down creates large air sacs (↓surface area = poor gas exchange)</li><li>• Loss of elastin reduces elastic recoil (↑compliance so lungs fill but can't empty)</li></ul>

# Lung Diseases: Restrictive

- Air flow restriction during inhalation

	Cause	Effect
<b>Pulmonary Fibrosis</b>	Chronic inhalation of asbestos, coal, dust, pollution or sometimes unknown	<ul style="list-style-type: none"><li>• Fibrous scar tissue (thickened tissue) in alveoli and other lung tissue</li><li>• Due to thick walls, poor gas exchange (similar to emphysema but different reason)</li><li>• Lungs become stiff due to ↑in collagen = ↓compliance (opposite to emphysema)</li></ul>

**In the pulmonary vein, what is the PO<sub>2</sub> for a person at rest?**

- a) 40 mmHg
- b) 46 mmHg
- c) 100 mmHg
- d) 159 mmHg

# Erthropoiesis

Process of producing new RBCs (2 million made and die each day)

- Occurs in bone marrow
- Regulated by the hormone, **erythropoietin (EPO)** from kidneys
- Normally, EPO levels are low for balanced RBC production to loss ratio
- When  $O_2$  levels to kidney drop, EPO is released
- Drop in  $O_2$  can be caused by:
  - ↓ in # of RBCs (↓carrying capacity)
  - ↓ in cardiac output (↓blood flow = ↓ $O_2$ )
  - lung disease
  - high altitude

# Regulation of Ventilation

**Stimulus:** Altered  $\text{PO}_2$  and  $\text{PCO}_2$  levels



**Sensors:** Chemoreceptors sense change in P



**Control centre:** Info is sent to respiratory centre in medulla



**Effector:** Respiratory muscles change force of contraction and relaxation to alter pulmonary ventilation



**Effect:**  $\text{PO}_2$  and  $\text{PCO}_2$  levels return to normal

# Chemoreceptors

	Central	Peripheral
Location	Medulla	Aortic arch and carotid body
Respond to changes in	pH only Note: $H^+$ cannot cross the blood brain barrier, but $CO_2$ can. So $CO_2$ is converted into bicarbonate and $H^+$ in CSF for detection	$PO_2$ $PCO_2$ pH

1. Gases move from high pressure to low pressure
2. Gases move until equilibrium
3. Gases only move at capillaries

Atmospheric  $PO_2 = 160$  mmHg  
Atmospheric  $PCO_2 = 0.3$  mmHg

Alveolar  $PO_2 = 100$  mmHg  
Alveolar  $PCO_2 = 40$  mmHg

$O_2$  enters capillaries from alveoli

Pulmonary artery  
 $PO_2 = 40$  mmHg  
 $PCO_2 = 46$  mmHg

Pulmonary circulation  
capillaries

Pulmonary vein  
 $PO_2 = 100$  mmHg  
 $PCO_2 = 40$  mmHg

$CO_2$  enters alveoli from capillaries

Systemic veins  
Venous  $PO_2 = 40$  mmHg  
Venous  $PCO_2 = 46$  mmHg

Systemic circulation  
capillaries

Systemic arteries  
Arterial  $PO_2 = 100$  mmHg  
Arterial  $PCO_2 = 40$  mmHg

$CO_2$  enters capillaries from body tissues

Body tissues  
Tissue  $PO_2 = 40$  mmHg (or less)  
Tissue  $PCO_2 = 46$  mmHg (or more)

$O_2$  leaves capillaries into body tissues

# Oxygen Transport

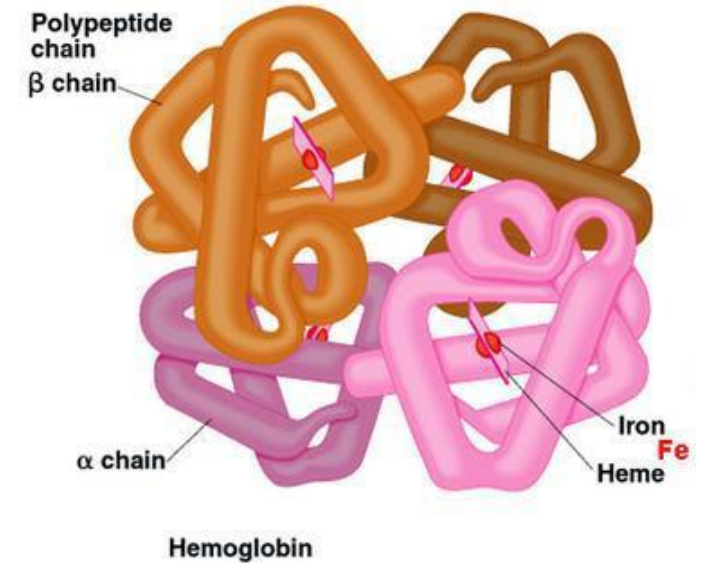
## Two Transport Mechanisms in Blood

### 1. Dissolved in plasma (1.5%)

- Very inadequate

### 2. Carried by hemoglobin (98.5%)

- $O_2 + Hb \leftrightarrow HbO_2$  (oxyhemoglobin)
- Hb is found in RBCs
- 4 heme groups (each Fe binds a  $O_2$ ) + 4 globins (polypeptide chain)
- Note: Hb transports BOTH  $O_2$  and  $CO_2$  (at different sites!)



# Carbon Dioxide Transport

## Three Transport Mechanisms in Blood

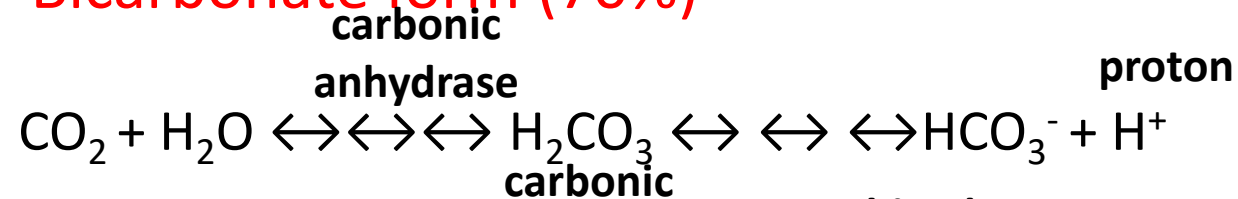
### 1. Dissolved in plasma (10%)

- More soluble than O<sub>2</sub>

### 2. Carried by hemoglobin (20%)

- CO<sub>2</sub> + Hb  $\leftrightarrow$  HbCO<sub>2</sub> (carbamino hemoglobin)
- Attached to “**globin**” not heme

### 3. Bicarbonate form (70%)



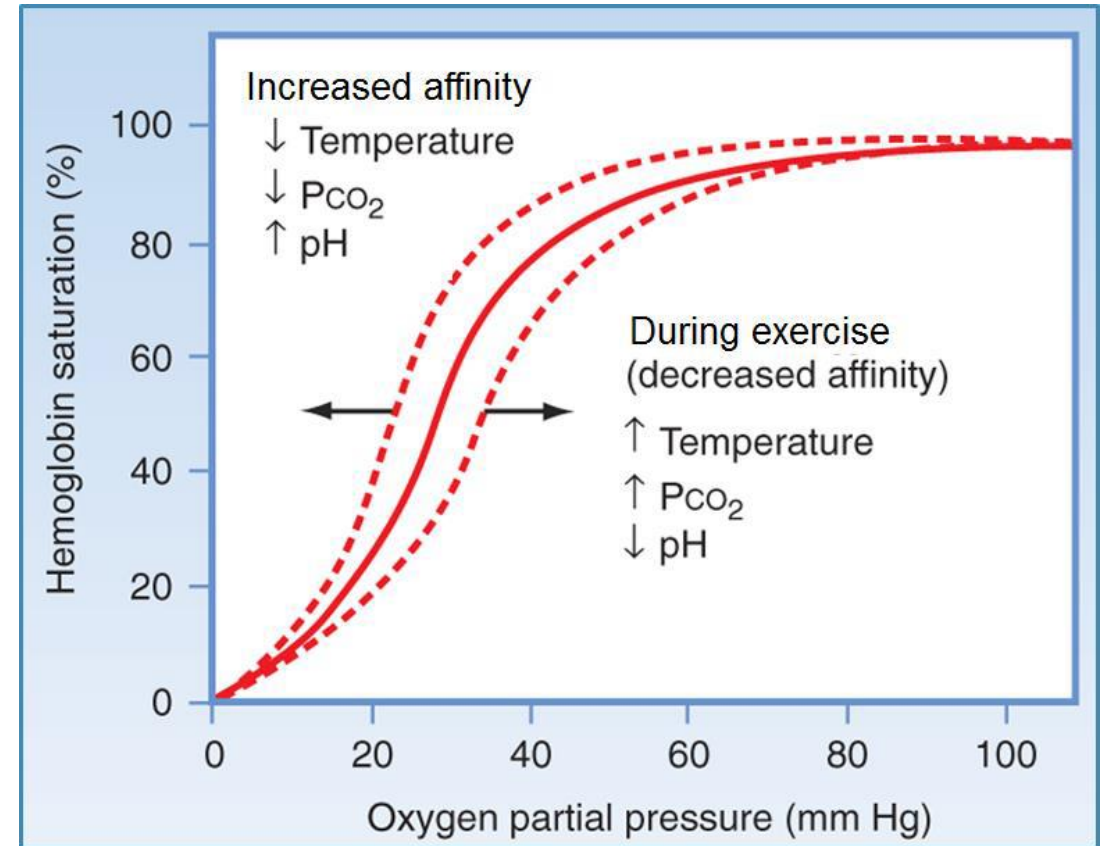
- Carbonic anhydrase is an enzyme found in RBCs

# Oxyhemoglobin Dissociation Curve

How to read this graph:

- At resting cell (40 mmHg), Hb is 80% saturated with  $O_2$
- At exercising cell (20 mmHg), Hb is 25% saturated with  $O_2$
- At alveoli (100 mmHg), Hb is 98% saturated with  $O_2$
- The steep slope means that a small change in  $PO_2$  drastically effects Hb saturation

Thus, at lungs, Hb is highly saturated with  $O_2$ . At tissues, Hb is releasing  $O_2$ .

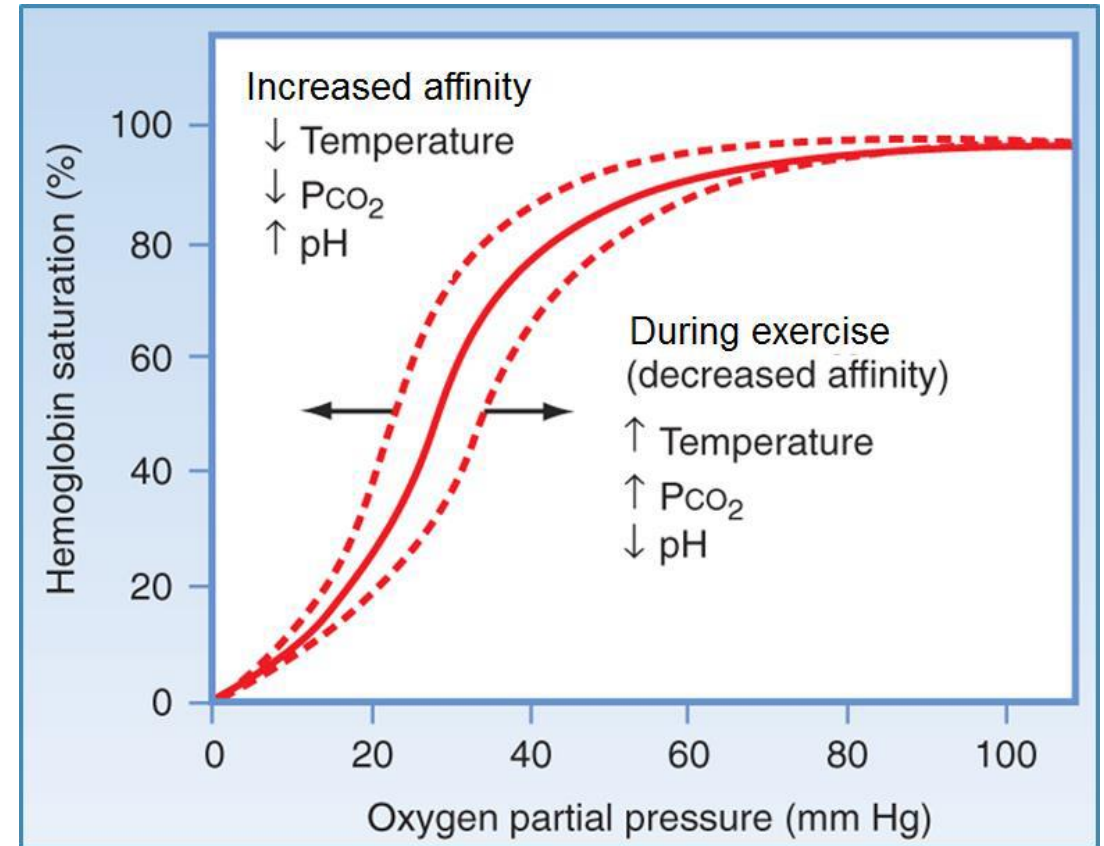


# Oxyhemoglobin Dissociation Curve

## Bohr Effect

- In the presence of  $\text{CO}_2$  or protons, Hb has a decreased affinity for oxygen
- The curve shifts to the RIGHT
- Example: now at 40 mmHg, Hb is decreased from 80% to 60% saturation with  $\text{O}_2$
- In other words, a right shift promotes  $\text{O}_2$  offloading

Thus, during exercise (when temp,  $\text{CO}_2$  and  $\text{H}^+$  levels are high), more  $\text{O}_2$  is delivered to tissues



# Oxyhemoglobin Dissociation Curve

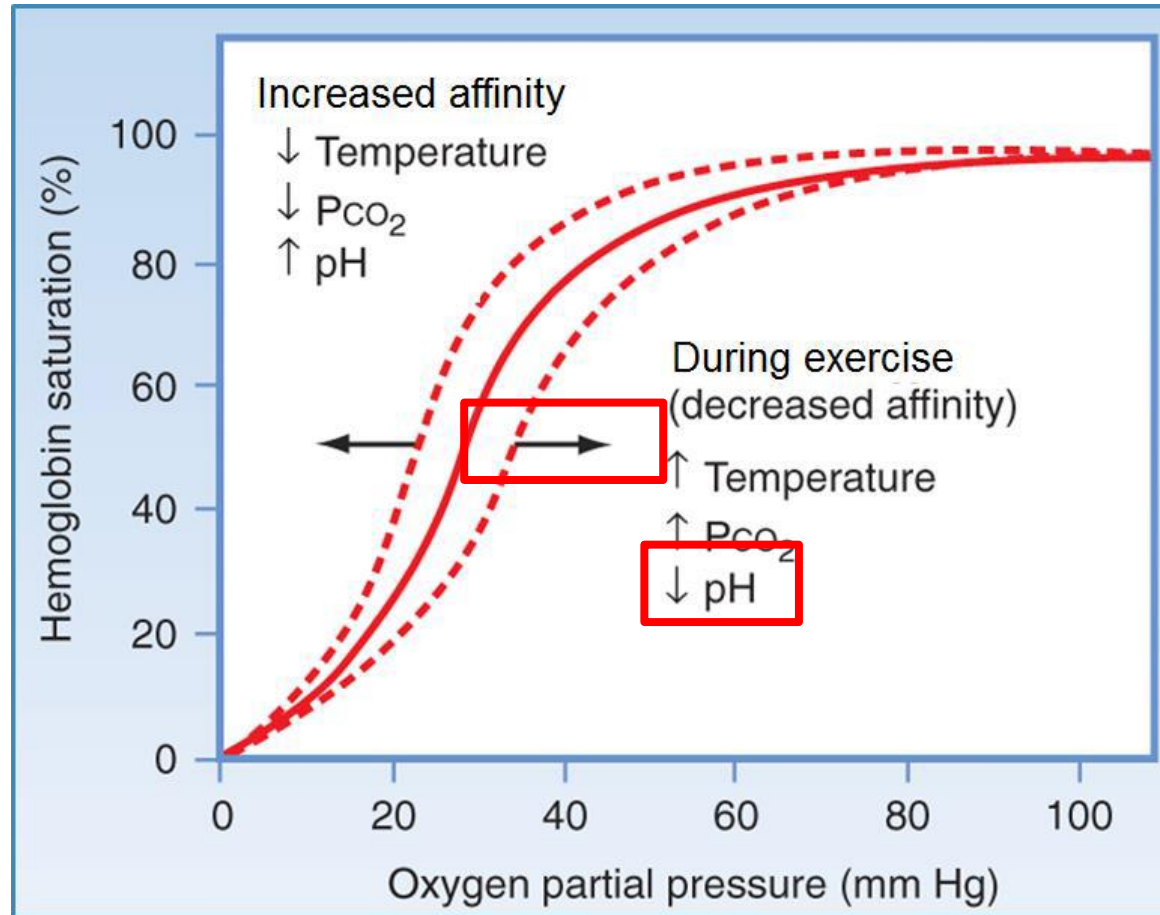
## At Body tissues:

- CO<sub>2</sub> begins to bind & form carbonic acid
- Curve shifts to right
- This enhances O<sub>2</sub> off loading at tissues

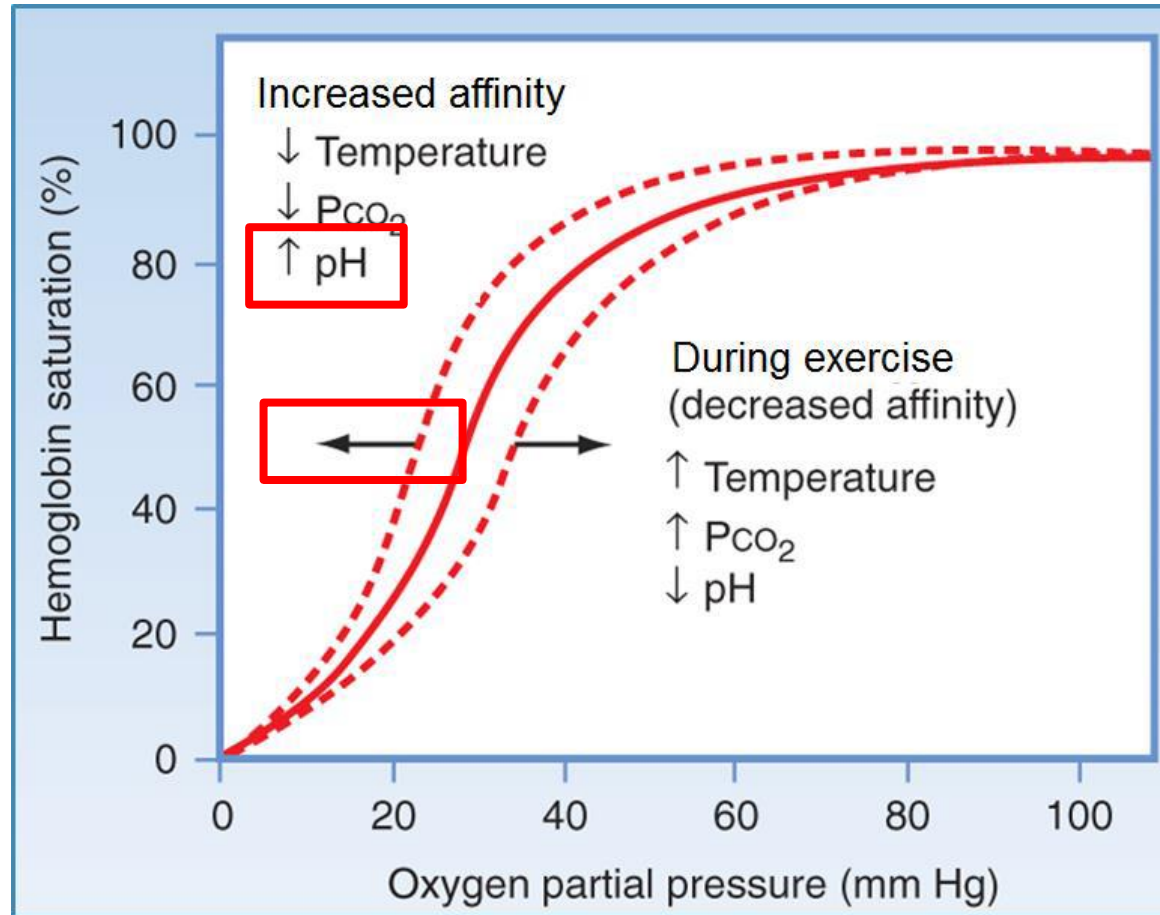
## At Lungs:

- Blood passing through lungs gives up CO<sub>2</sub> & H<sup>+</sup> ions in the form of carbonic acid
- Bicarbonate will combine with the proton to form CO<sub>2</sub> and water and CO<sub>2</sub> will diffuse out of the blood stream at the lungs
- This shifts O<sub>2</sub> dissociation curve to left
- During this process the pH will increase and the affinity for oxygen by Hb will increase, allowing for binding of O<sub>2</sub>

# Shift Right = Decreased Affinity and increased off-loading

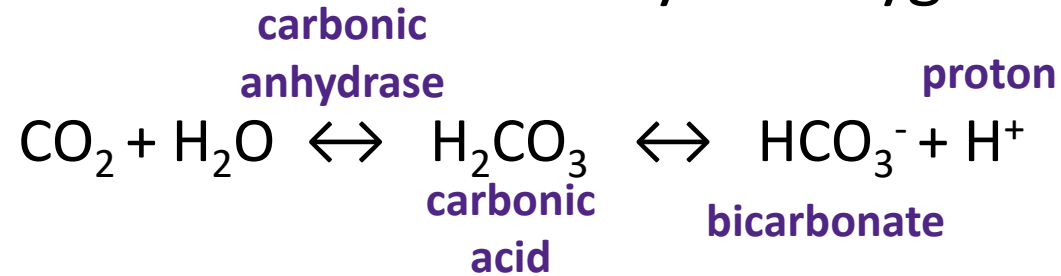


# Shift Left = Increased Affinity

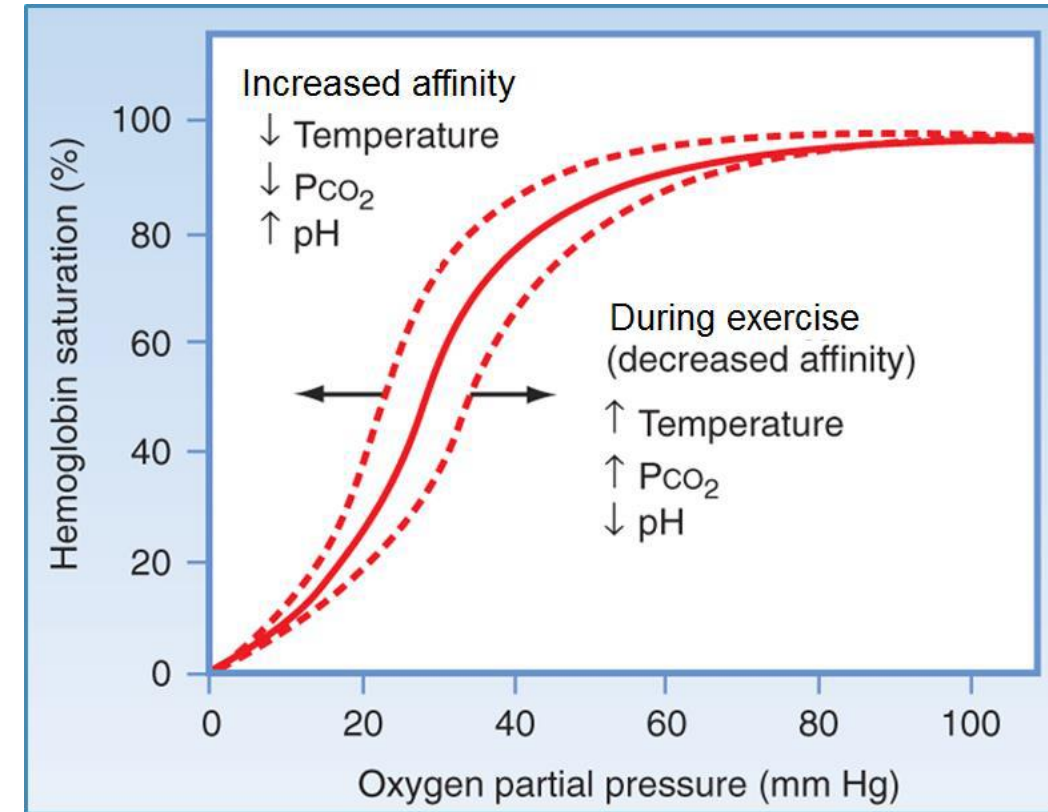


# Bohr Effect

- In the presence of  $\text{CO}_2$  or protons, Hb has a decreased affinity for oxygen



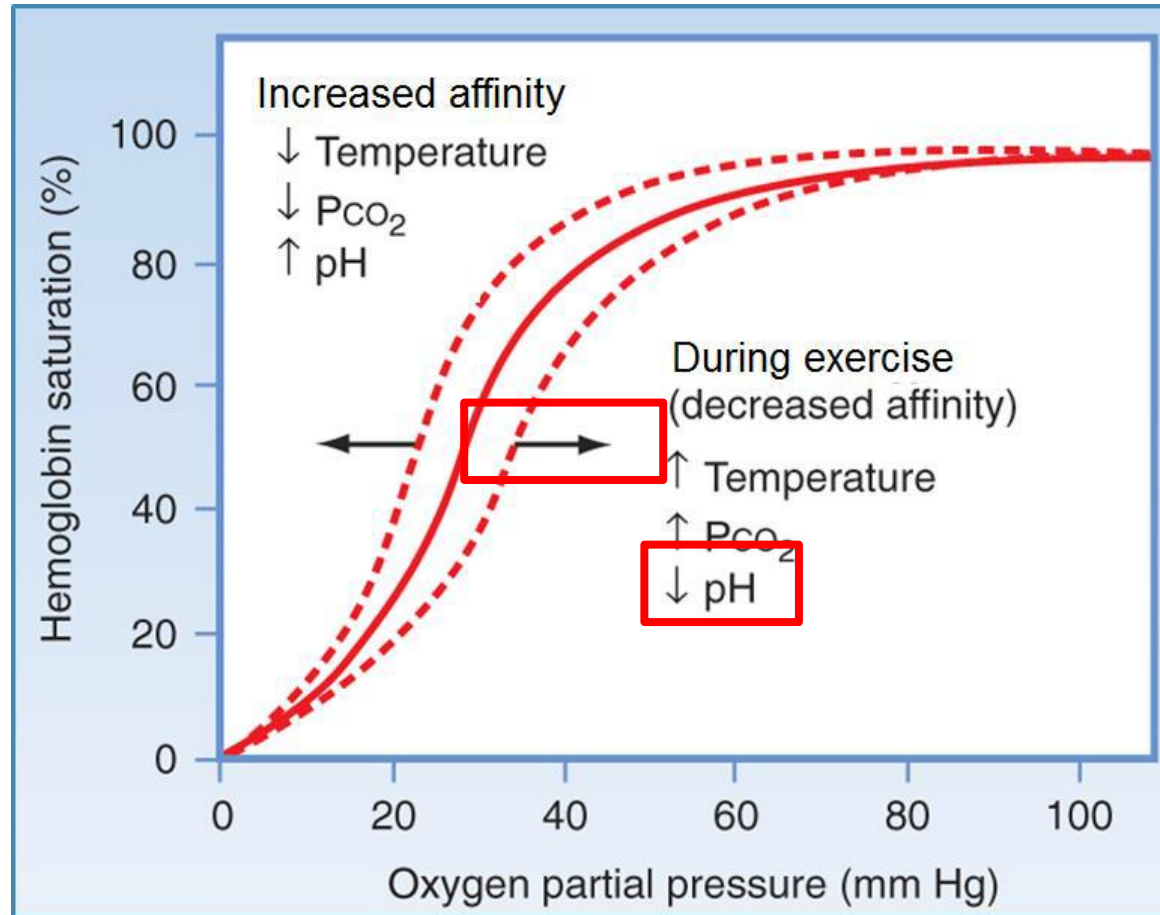
- Within the red blood cells  $\text{CO}_2$  is converted to bicarbonate
- The pH of blood will change and cause the  $\text{O}_2$  dissociation curve to shift right, causing more  $\text{O}_2$  offloading at tissues



# Chloride Shift

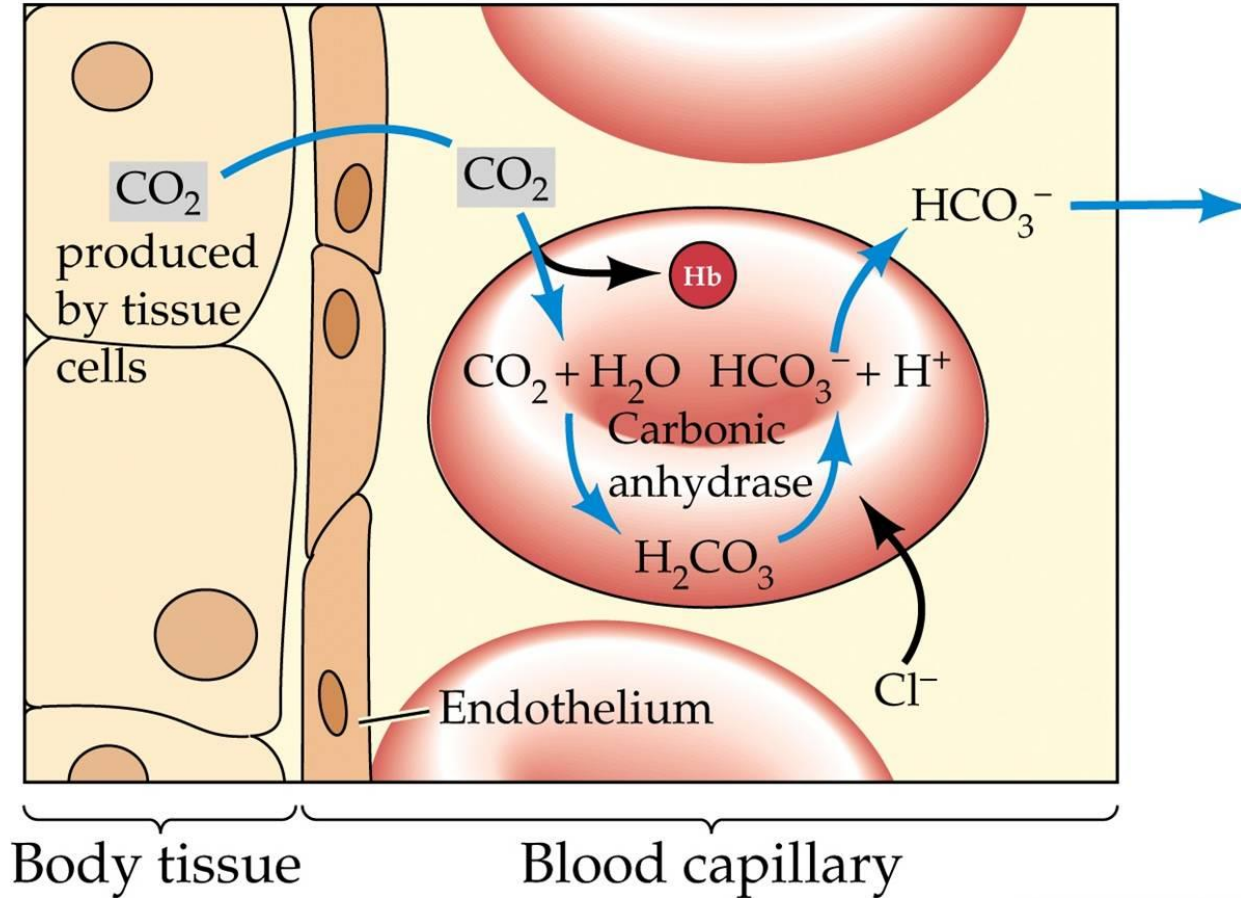
- As  $\text{CO}_2$  leaves body tissue it enters the red blood cells
- $\text{CO}_2$  is converted into bicarbonate in the red blood cell and a proton is formed
- The pH will decrease and oxygen affinity of Hb will decrease, allowing off-loading into the tissues
- As bicarbonate exits the red blood cell a chloride molecule will enter to maintain the charge balance
  - To maintain an electrically neutral state

# Shift Right = Decreased Affinity and increased off-loading



# Chloride Shift: Body Tissues

(a) In body tissue



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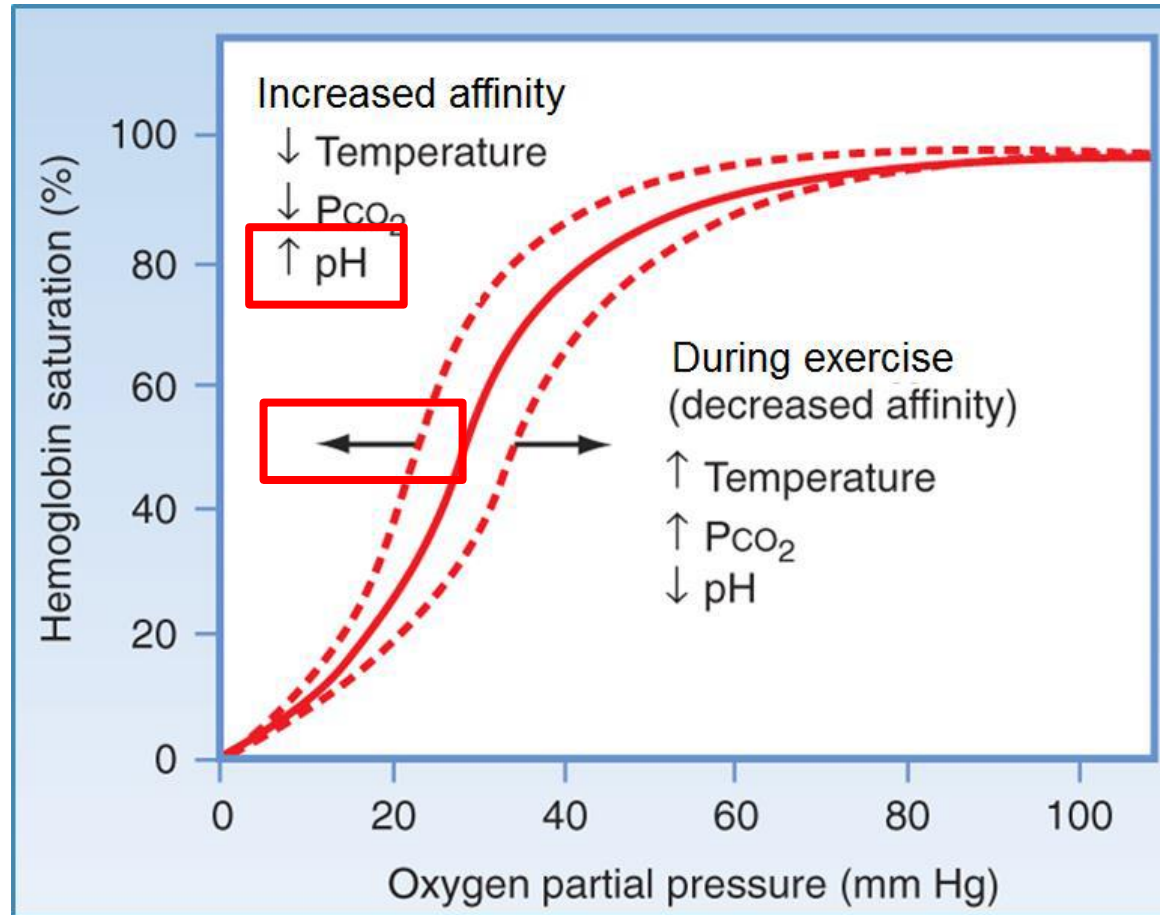
# Chloride Shift: At Lungs

- Takes place in reverse

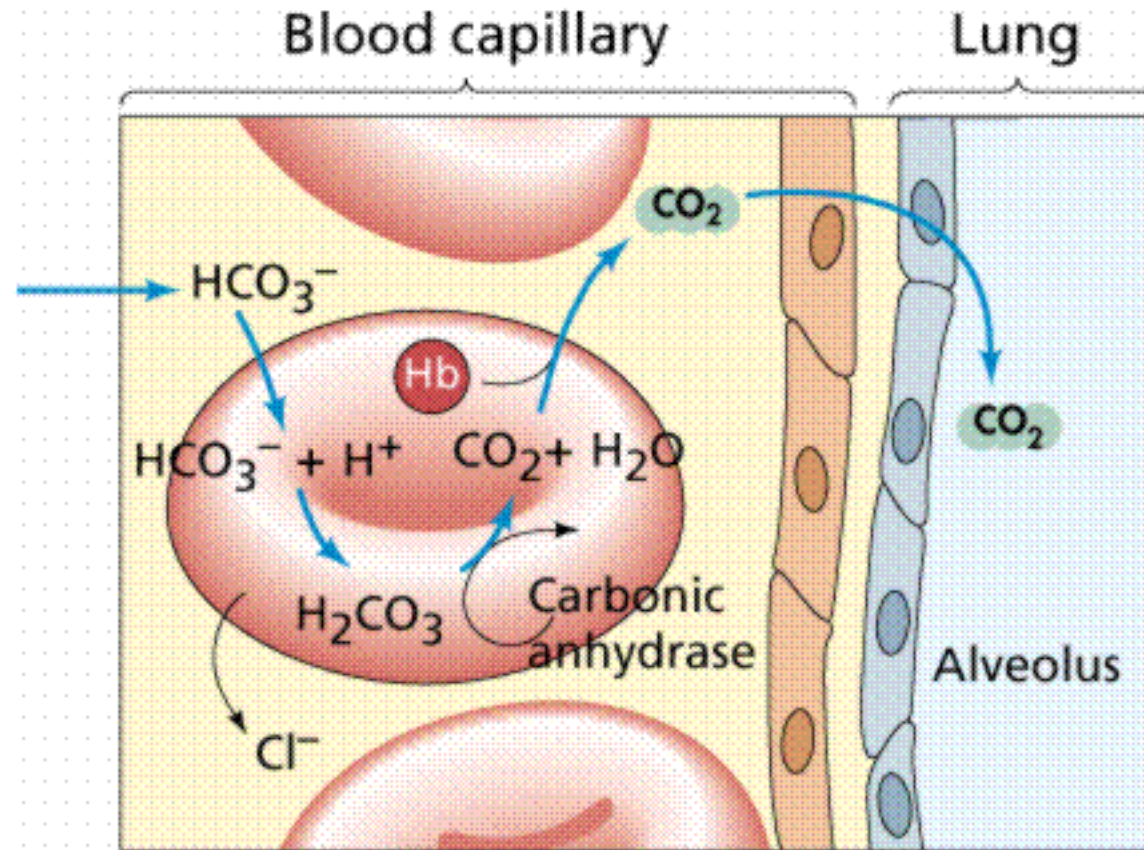


- At pulmonary capillaries, the bicarbonate ion will enter the red blood cell and a chloride will leave the red blood cell
- Bicarbonate will combine with the proton to form  $\text{CO}_2$  and water and  $\text{CO}_2$  will diffuse out of the blood stream at the lungs
- During this process the pH will increase and the affinity for oxygen by Hb will increase, allowing for binding of  $\text{O}_2$

# Shift Left = Increased Affinity



# Chloride Shift: At Lungs



# Intro to Gastrointestinal Physiology

**Chapter 10: Dr. Woods**

# Four Processes of Digestive System

## 1. Secretion

- Exocrine into lumen of GI tract
- Endocrine into blood

## 2. Digestion

- Chemical (enzymes)
- Mechanical

## 3. Motility

- Propels food through each segment of GI tract
- Can participate in mechanical digestion

## 4. Absorption

- Movement of macronutrients into cells of GI tract and then into blood

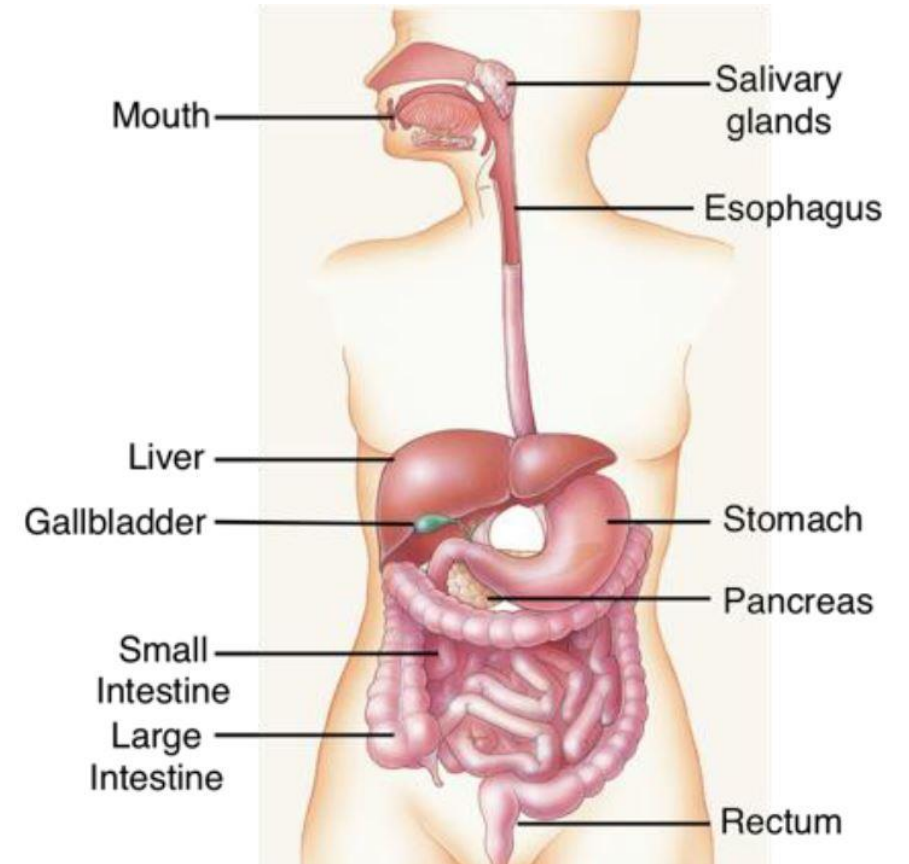
# GI Tract

## Main pathway

- Mouth and salivary glands
- Esophagus
- Stomach
- Small Intestine
- Large Intestine
- Rectum

## Accessory Organs

- Liver (produces bile)
- Gall bladder (stores biles)
- Common bile duct
- Pancreas (endocrine and exocrine secretions)



# Salivary Glands

## Function

- Produce saliva (water, mucus, ions and salivary amylase; lipase in babies)

## Parotid Gland

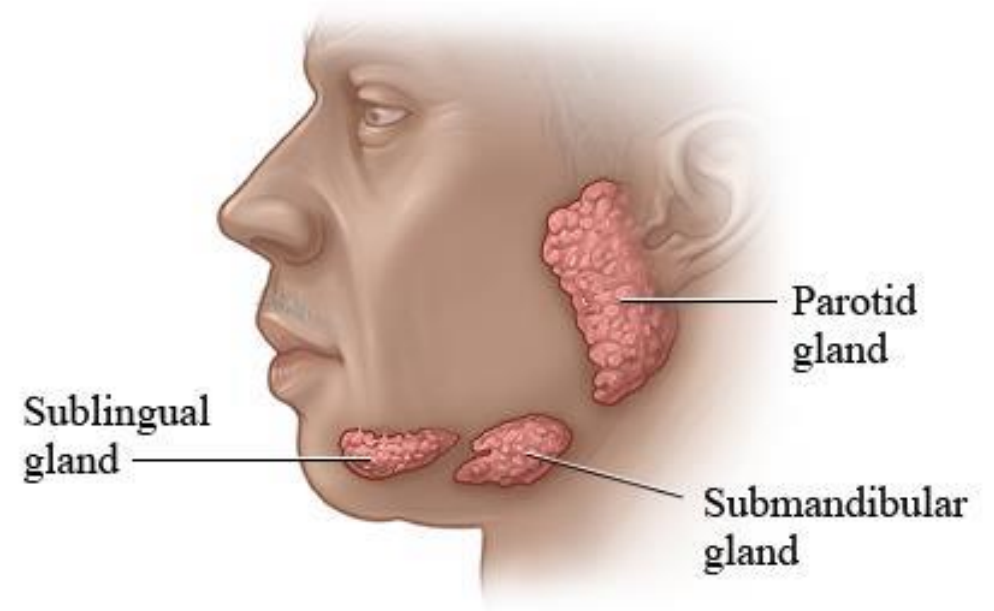
- Watery liquid that contains **amylase** and **lingual lipase**

## Submandibular Gland

- Thicker liquid that contains **mucus** with less **amylase** and less **lingual lipase**

## Sublingual Gland

- A lot of **mucus** and very little enzymes



# Mastication, Swallowing and Peristalsis

## Mastication

- Mechanical manipulation of food into a lump of food (**bolus**)

## Swallowing → 3 Stages:

1. Voluntary stage
2. Pharyngeal stage (involuntary): propagation of food into pharynx; close trachea and nasal cavity
3. Esophageal (involuntary): propagation of food into esophagus

## Peristalsis

- Movement of bolus down the esophagus by contraction of muscles
- Involuntary control by **medulla**
- Second peristaltic wave if food is still lodged

# Mouth: Summary of Four Processes

## 1. Secretion

- Saliva

## 2. Digestion

- Chemical (amylase and lipase)
- Mechanical (mastication)

## 3. Motility

- Mastication in mouth
- Peristalsis in esophagus

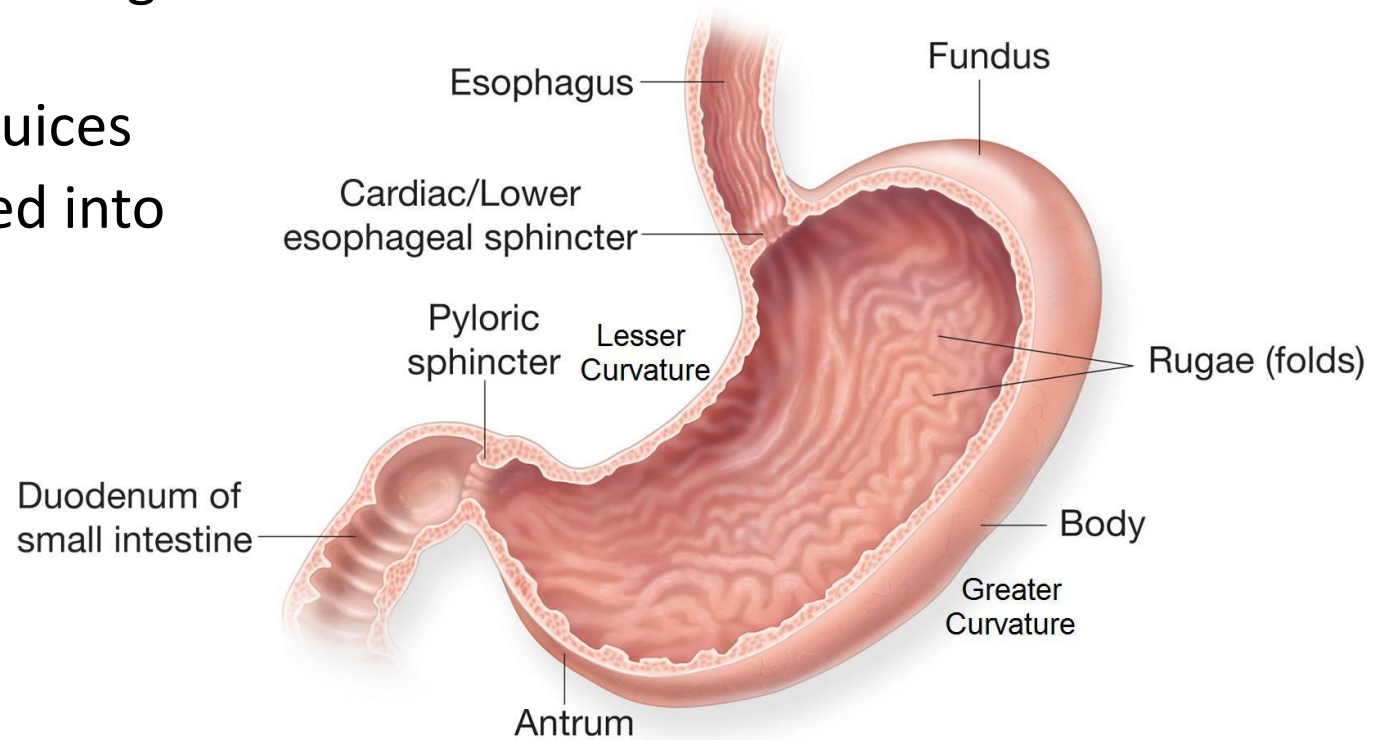
## 4. Absorption

- None

# Stomach

## Function

- Reservoir for food before entering intestines for absorption
- Bolus is liquefied by gastric juices
- 2-3 L of gastric juices secreted into stomach per day



# Layers of The Stomach

## Mucosa

- Single layer of cells that can be endocrine or exocrine
- Large folds called **rugae** and invaginations called **pits**

## Submucosa

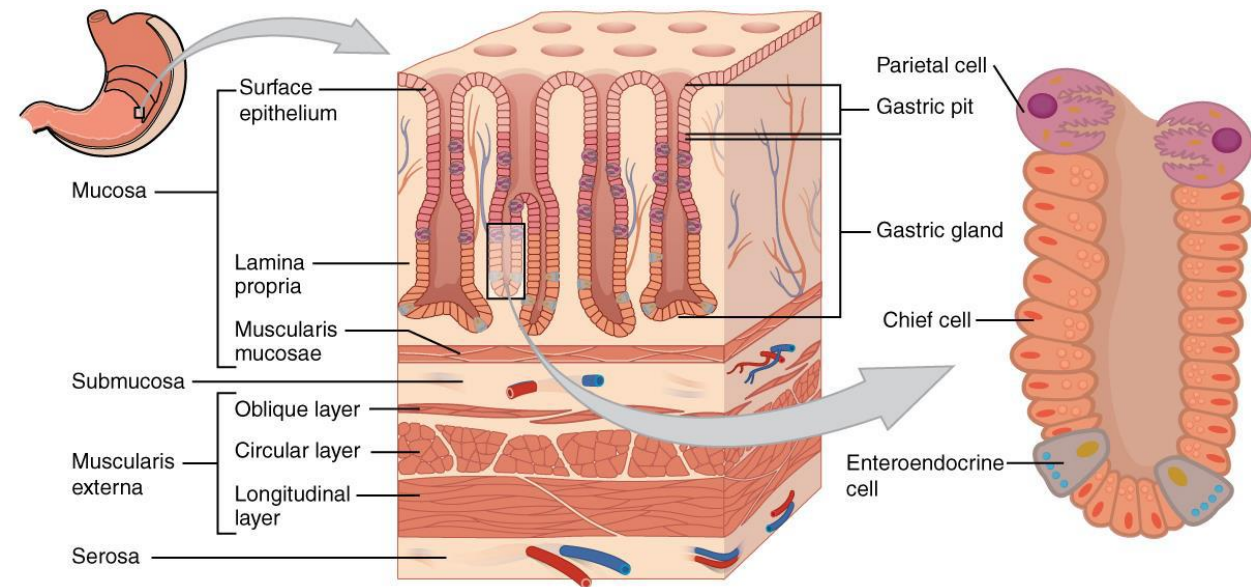
- Nervous plexus: **submucosal plexus** (detects food in stomach and sends signals to mucosa for response)
- Connective tissue to adhere mucosa to smooth muscle

## Smooth muscle (Muscularis externa)

- Circular and longitudinal muscle to change shape of the stomach
- Nervous plexus: **myenteric plexus** (controls muscle)

## Serosa

- External layer of dense connective tissue



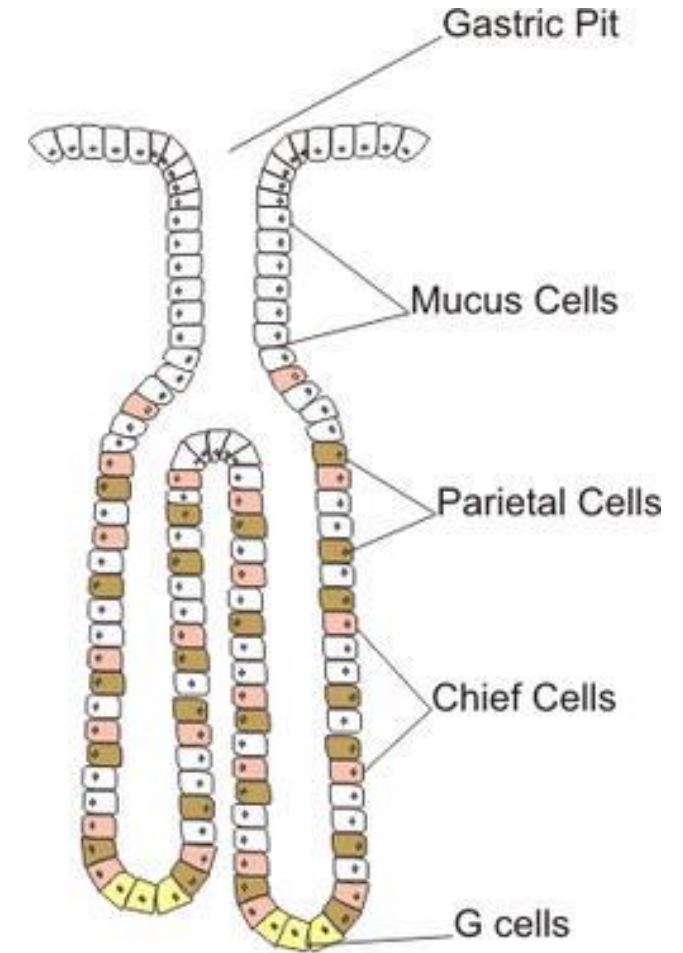
# Secretions In The Stomach

## Exocrine

Cell	Secretion	Function
Mucus Neck Cells	Mucus	Protects stomach from acid
Chief Cells	Pepsinogen	Inactive enzyme
	Gastric Lipase	Breakdown fats
Parietal Cells	Intrinsic Factor	Allows absorption of vitamin B12
	HCL acid	See slide 10.

## Endocrine

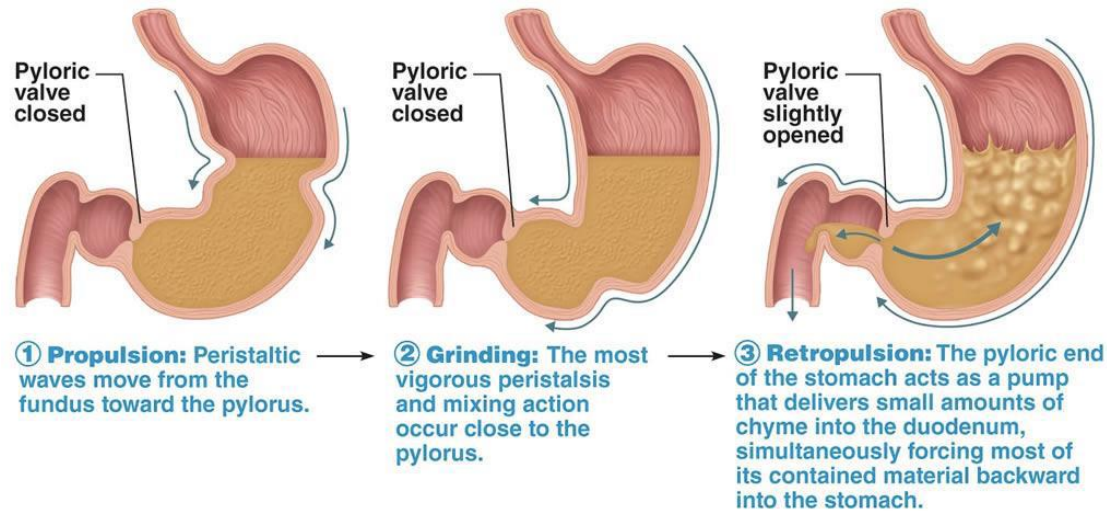
Cell	Secretion	Function
G Cell	Gastrin	Promotes gastric motility and function



Frank Bourghrey M.D. 2009

# Mechanical Digestion in the Stomach

- Gentle mixing waves(every 15-25 seconds), pyloric sphincter closed
- Vigorous mixing from body to pylorus
- Slight opening of **pyloric sphincter**, very small amount of chyme exits to the duodenum (**retropulsion**)
- Most chyme pushed back into stomach body for more mixing



# Chemical Digestion in the Stomach

- HCl (parietal cells) cleaves pepsinogen (chief cells) to pepsin
- **Pepsin**: protein digestion
- **Gastric lipase** (chief cells; activated by HCl): lipid digestion
- **Lingual lipase** (activated by HCl): lipid digestion
- **Salivary amylase** (inactivated by HCl): carbohydrate digestion stops

# HCl Acid in the Stomach

## Functions:

- Activation of pepsin
- Activation of lingual and gastric lipase
- Inactivation of salivary amylase
- Kills microbes
- Denatures proteins
- Stimulates secretion of hormones

# Anatomy of the Small Intestine

## Duodenum

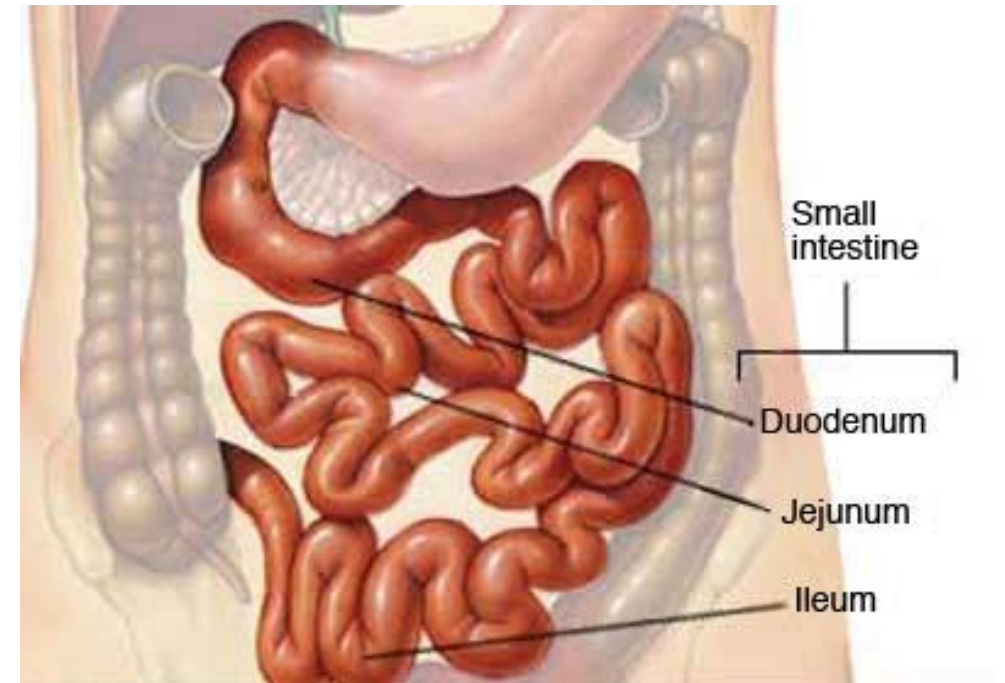
- Location of enzymes mixing with chyme, most **digestion** occurs here
- Can increase or decrease motility to optimize chemical digestion

## Jejunum

- Many villi to increase surface area for optimal absorption
- Most **absorption** occurs here

## Ileum

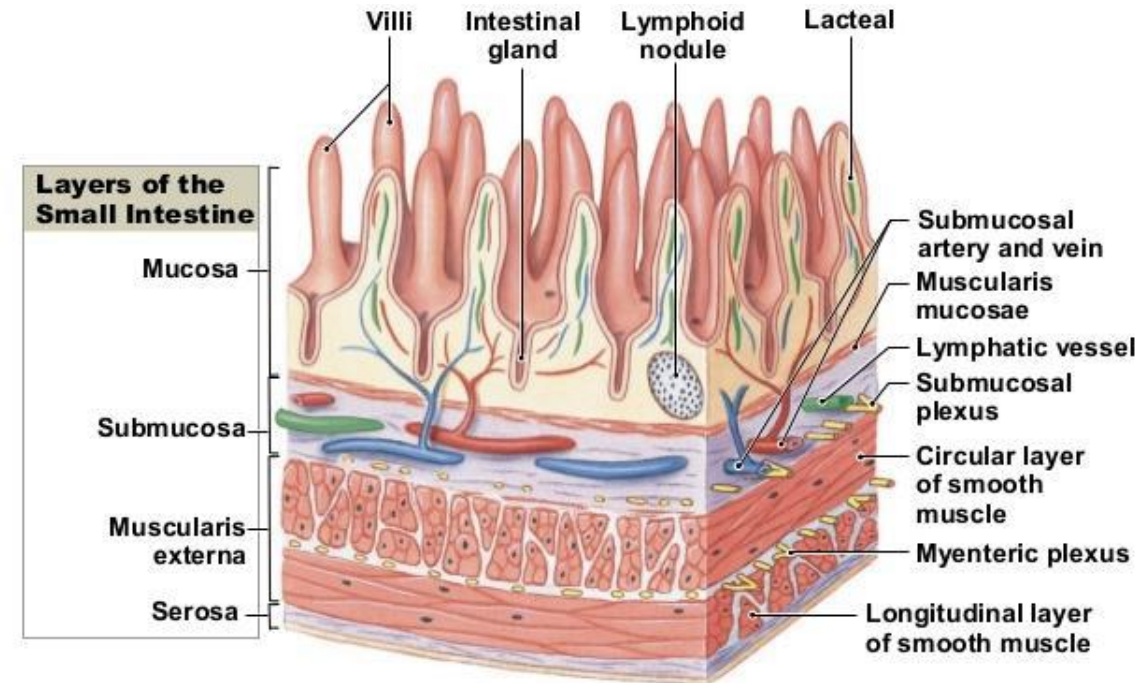
- Less villi but can still absorb nutrients if necessary



# Layers of the Small Intestine

4 layers (similar to stomach)

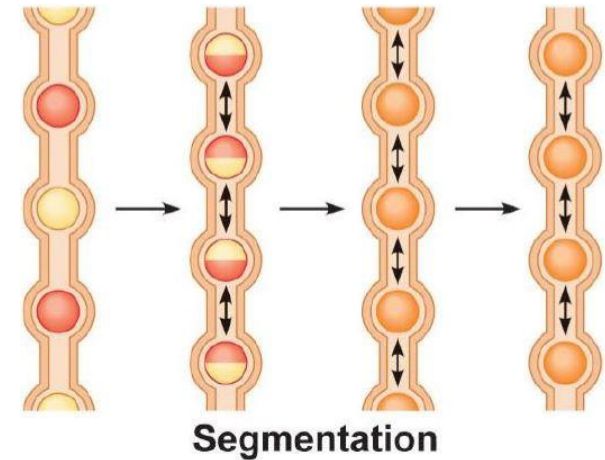
- Mucosa
  - Submucosa
    - Submucosal plexus
  - Muscularis externa
    - Myenteric plexus
  - Serosa
- SI has villi and microvilli that increases surface area for absorption
  - Note: villi in SI  $\neq$  rugae in stomach



# Motility of the Small Intestine

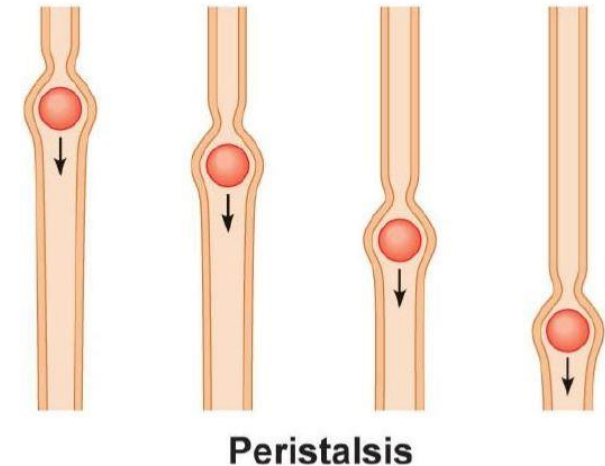
## Segmentation

- Localized contractions for mixing chyme with digestive juices
- Increases the interactions of particles of food in chyme with absorptive cells of the mucosa layer



## Peristalsis

- Propels chyme forward to next portion of SI (from pyloric sphincter to large intestine)



# Cells in the Small Intestine

## Exocrine

Cell	Secretion	Function
Goblet Cells	Mucus	Protects small intestine from acid
Intestinal Gland cells	Intestinal Juice (alkaline)	Neutralize acid from stomach
Paneth Cells	Lysozyme	Anti-bacterial enzyme

## Endocrine

Cell	Secretion	Function
S Cell	Secretin	Stimulates release of bicarbonate from pancreas
CCK Cells	CCK: Cholecystokinin	Stimulates release of enzymes from pancreas and bile from gallbladder
K cells	GIP: Glucose Dependent Insulinotrophic peptide	Stimulates insulin release from the pancreas

# What Questions Do You Have?

You can ask in the **Owl forums** as well!

Also anonymously ask questions in the **online dropbox!!**