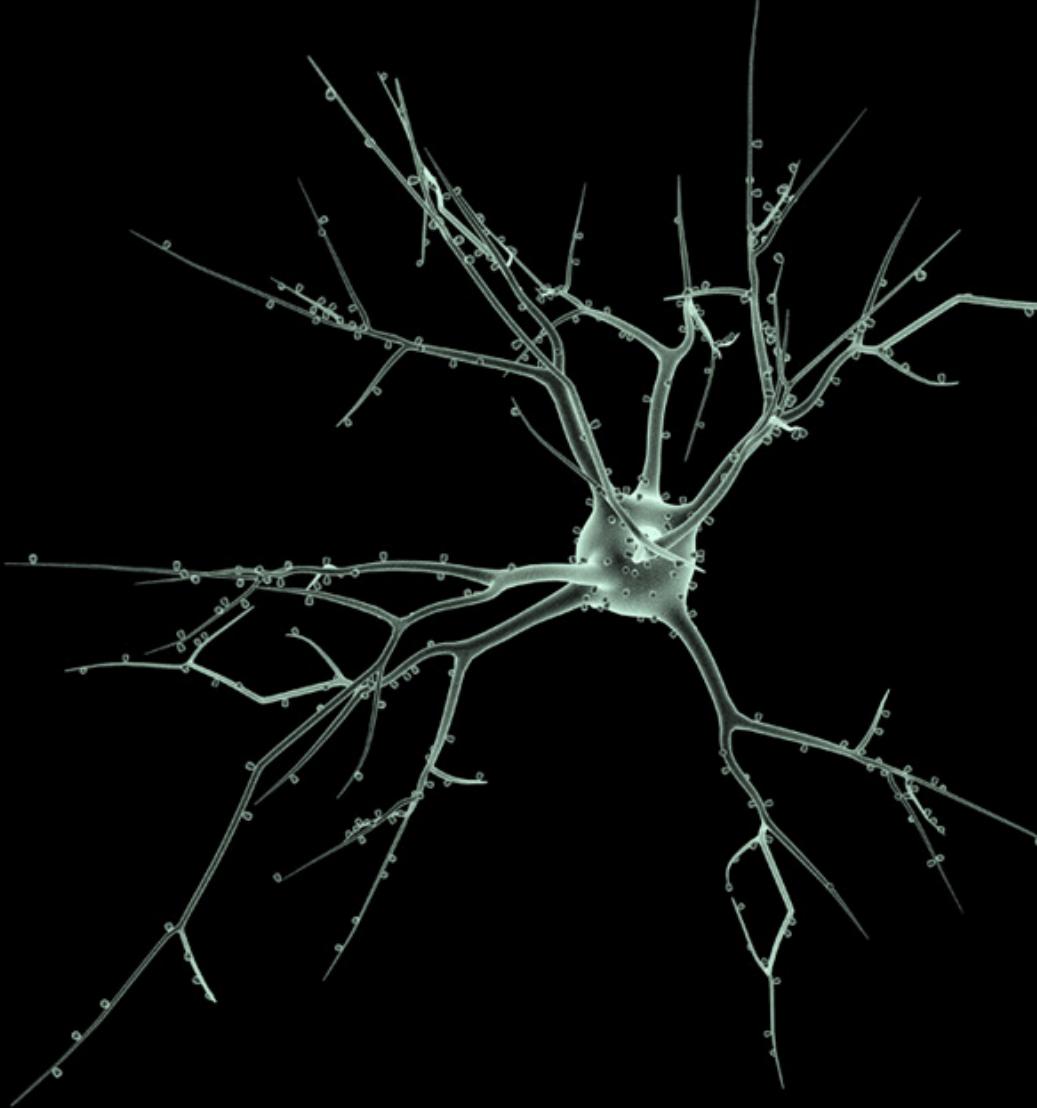


ANT 3120 Physiology Exam 5

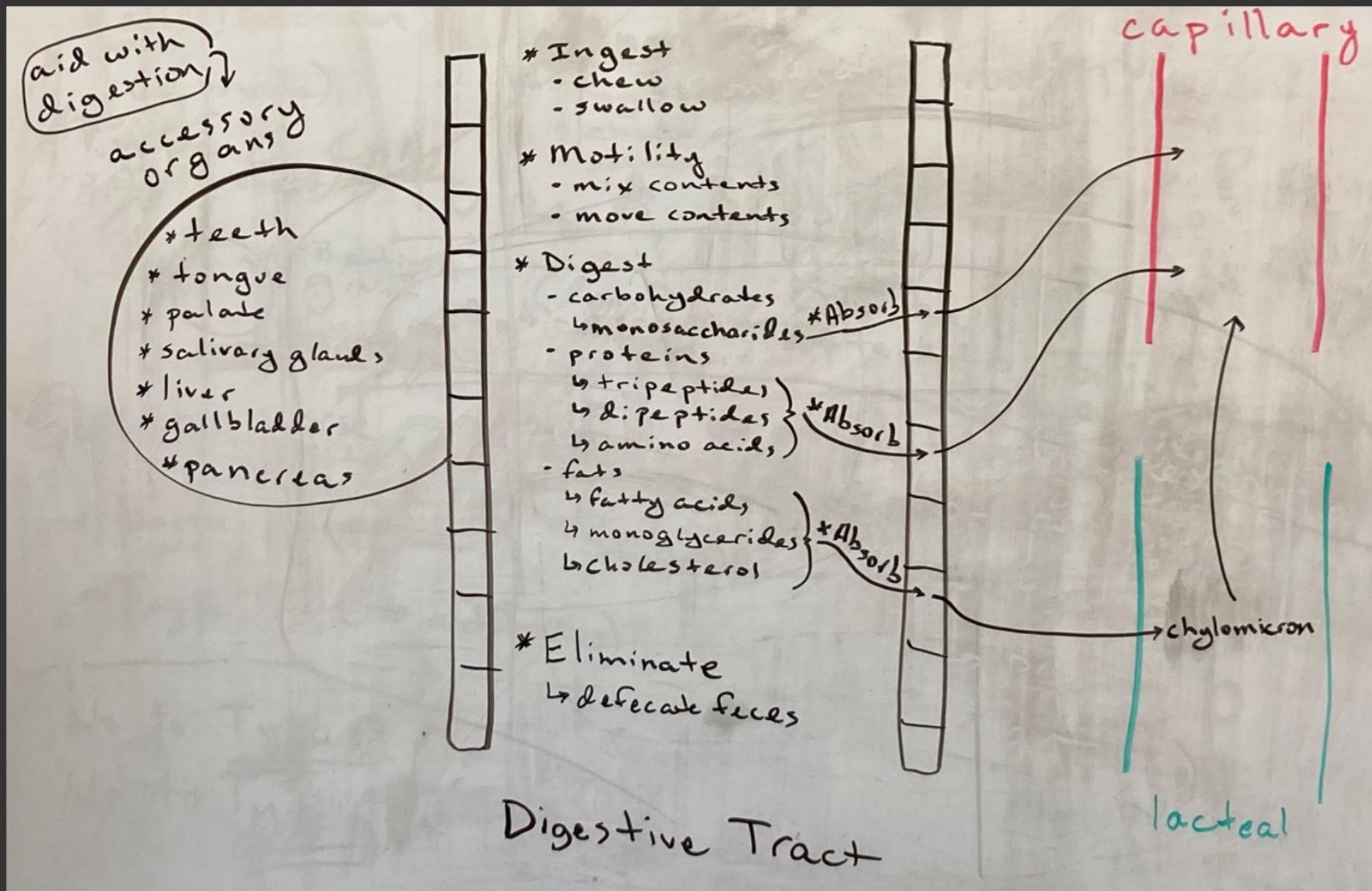
Combined Notes
14APR2022



Five Major Processes of the Digestive System

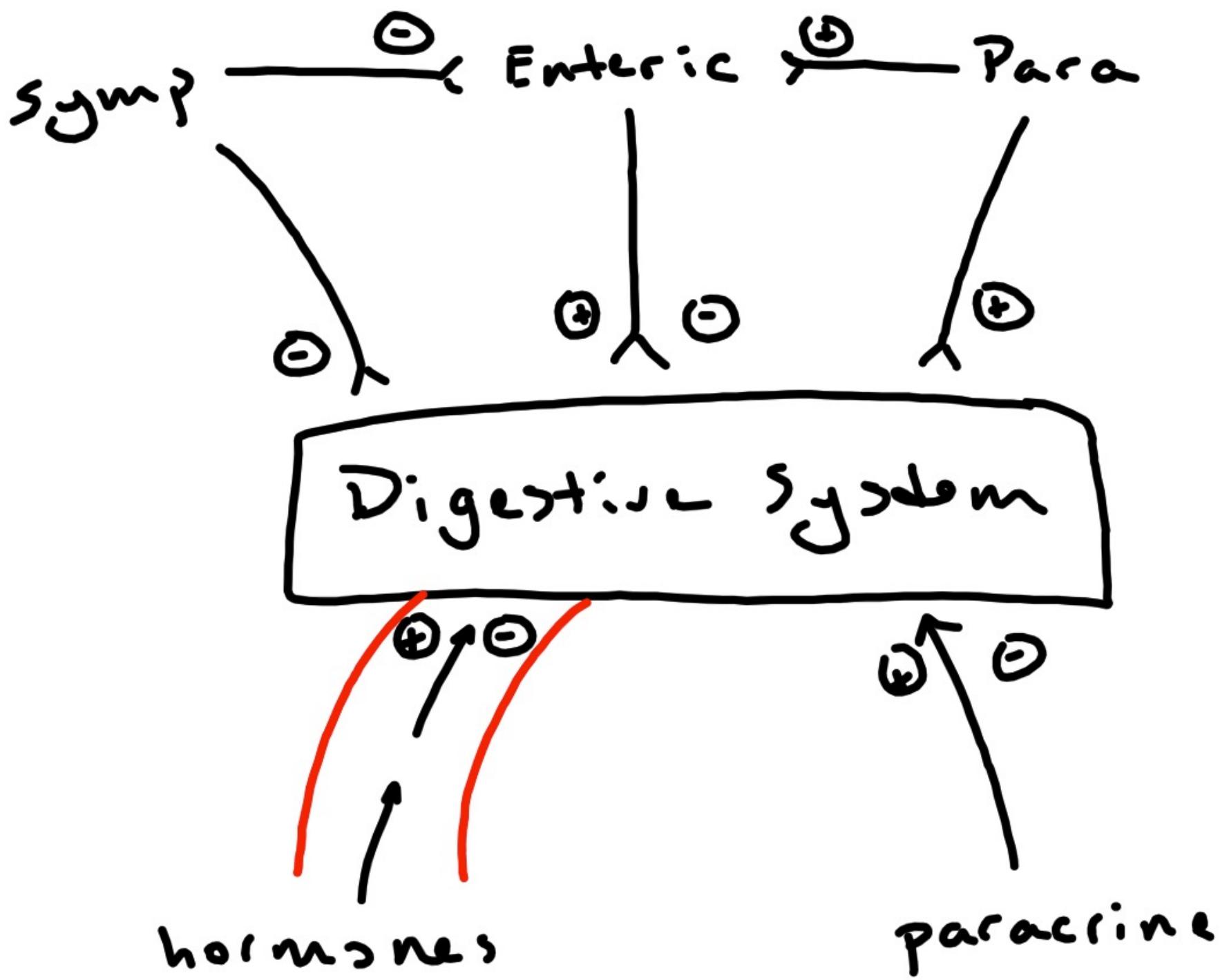
- Ingestion
 - Mastication (chewing) of food and deglutition (swallowing)
- Motility
 - Propelling of contents through the digestive tract
 - Mixing of contents within the digestive tract
- Digestion
 - Break down of food into their simplest forms (nutrients)
 - Mechanical digestion
 - Physical break down
 - Mastication / Chewing
 - Mixing
 - Chemical Digestion
 - Break down of large molecules into small molecules
 - Carbohydrates into monosaccharides
 - Proteins into tripeptides , dipeptides , and amino acids
 - Lipids into fatty acids , monoglycerides , and cholesterol
 - Done by specific digestive secretions
 - Some foods are ingested in forms that do not require digestion
 - Already in their simplest forms (eg , vitamins , minerals , simple sugars , water)
- Absorption
 - Transport of nutrients to the circulatory system (blood and lymph)
 - Allows nutrients to be utilized by the body
- Elimination
 - Removal of undigested and unabsorbed material from the body

Five Major Processes of the Digestive System



Regulation of the Digestive System

- Processes that regulate motility , digestion , absorption , and elimination
 - Nervous System
 - Hormones
 - Paracrines



Regulation of the Digestive System - Autonomic Nervous System

- Enteric Nervous System
 - Submucosal plexus and myenteric plexus
 - Influenced by the autonomic nervous system
 - Can operate autonomously
 - Functions to stimulate or inhibit the digestive system
- Sympathetic
 - Functions to inhibit the digestive system
- Parasympathetic
 - Functions to stimulate the digestive system

Walls of the Digestive Tract Consist of Four Layers

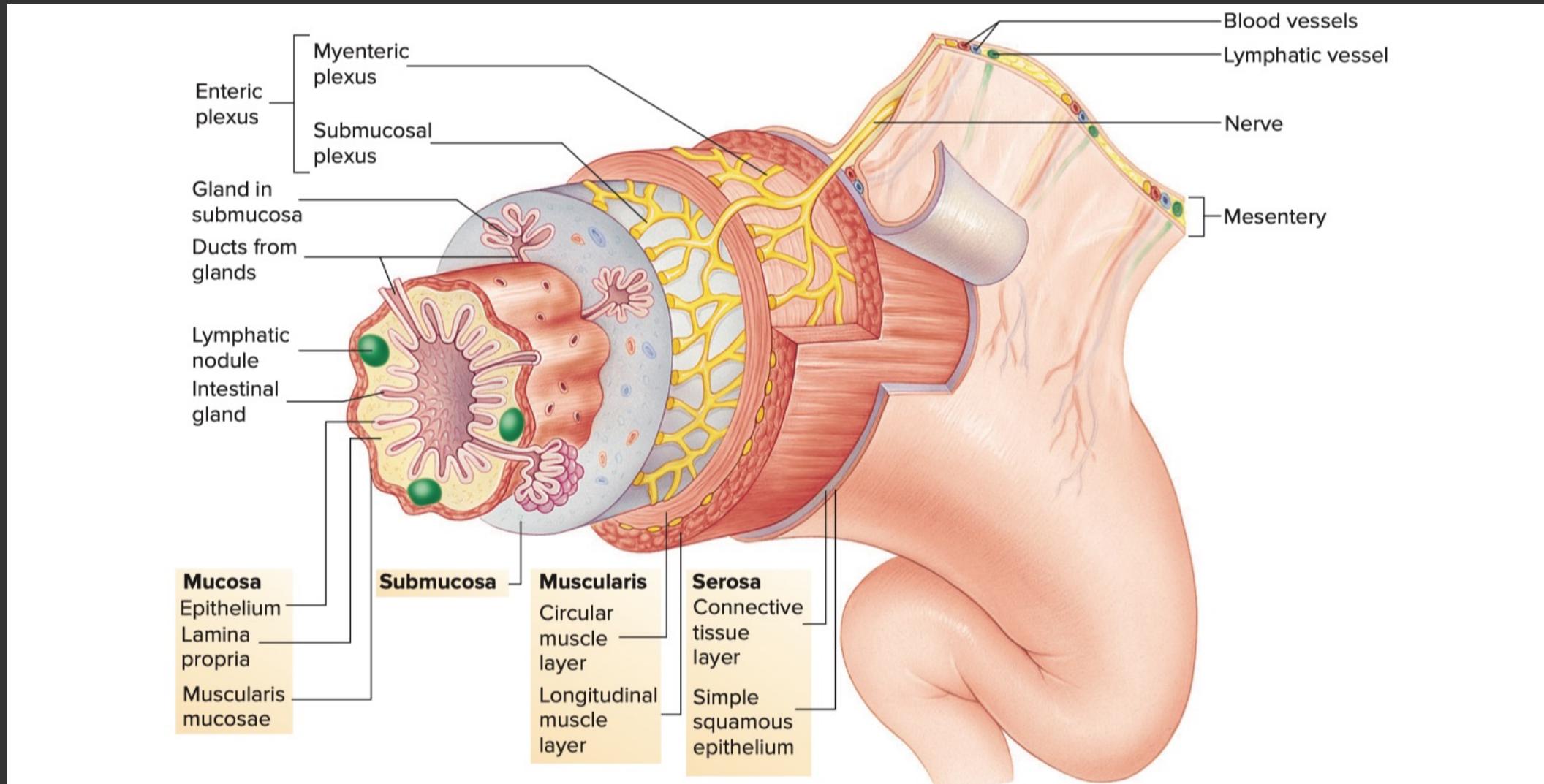


FIGURE 24.4 Digestive Tract Histology

The four tunics are the mucosa, the submucosa, the muscularis, and a serosa or an adventitia. In this image, the serosa is also called the visceral peritoneum, which forms part of the mesentery. Glands may exist along the digestive tract as part of the epithelium, as glands within the submucosa, or as large glands outside the digestive tract.

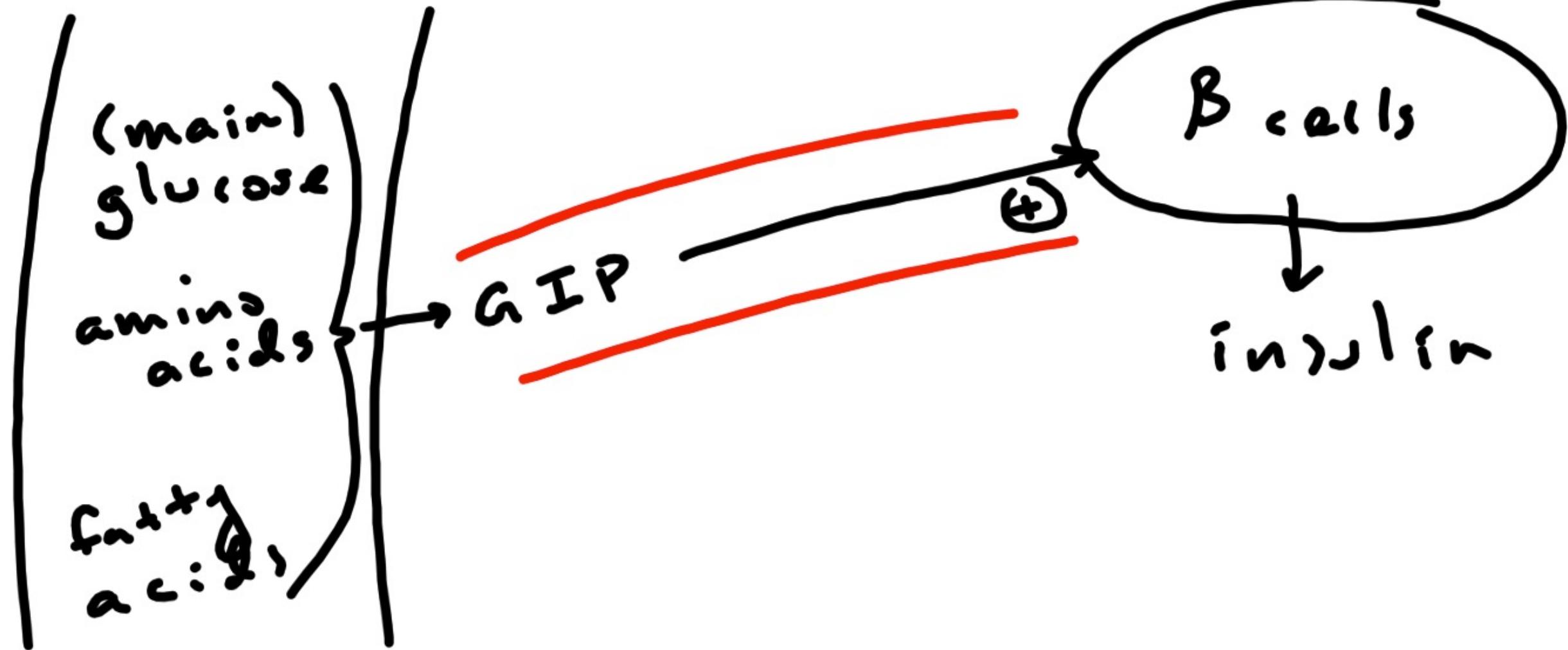
Regulation of the Digestive System - Hormones

- Gastrin = secreted by stomach , duodenum , and jejunum
 - Functions :
 - Stimulates gastric acid secretion
 - Stimulates gastric motility
 - Stimulation of Release :
 - Protein
 - Distension of Stomach
 - Parasympathetics
- Cholecystokinin (CCK) = secreted by duodenum , jejunum , and ileum
 - Functions :
 - Causes gallbladder to contract
 - Stimulates secretion of pancreatic enzymes
 - Inhibits gastric emptying
 - Potentiates effect of secretin on secretion of HCO_3^- from pancreas
 - Stimulation of Release :
 - Protein in the small intestine
 - Lipid in the small intestine
 - Decrease in small intestine pH

Regulation of the Digestive System - Hormones

- Secretin = secreted by duodenum , jejunum , and ileum
 - Functions :
 - Stimulates secretion of HCO_3^- from pancreas
 - Stimulates secretion of HCO_3^- from liver
 - Inhibits release of gastrin
 - Inhibits gastric emptying
 - Stimulation of Release :
 - Decrease in small intestine pH
- Glucose-Dependent Insulinotropic Peptide (GIP) :
 - Secreted by duodenum
 - Functions :
 - Stimulates secretion of insulin
 - Stimulation of Release :
 - Glucose (mainly glucose) , lipid , and amino acids in small intestine

Small intestine



Regulation of the Digestive System - Paracrines

- Somatostatin = secreted by the stomach
 - Function = Inhibits gastric acid secretion
 - Stimulation of Release = decreased gastric pH
- Histamine = secreted by stomach
 - Function = Stimulates gastric acid secretion
 - Stimulation of Release = Parasympathetics

Ingestion

- Mastication / Chewing
- Deglutition / Swallowing

Mastication / Chewing

- Function :
 - Mechanical Digestion
 - Reduces size of food
 - Mixes food with saliva to form bolus
 - Semi-solid mass that is swallowed

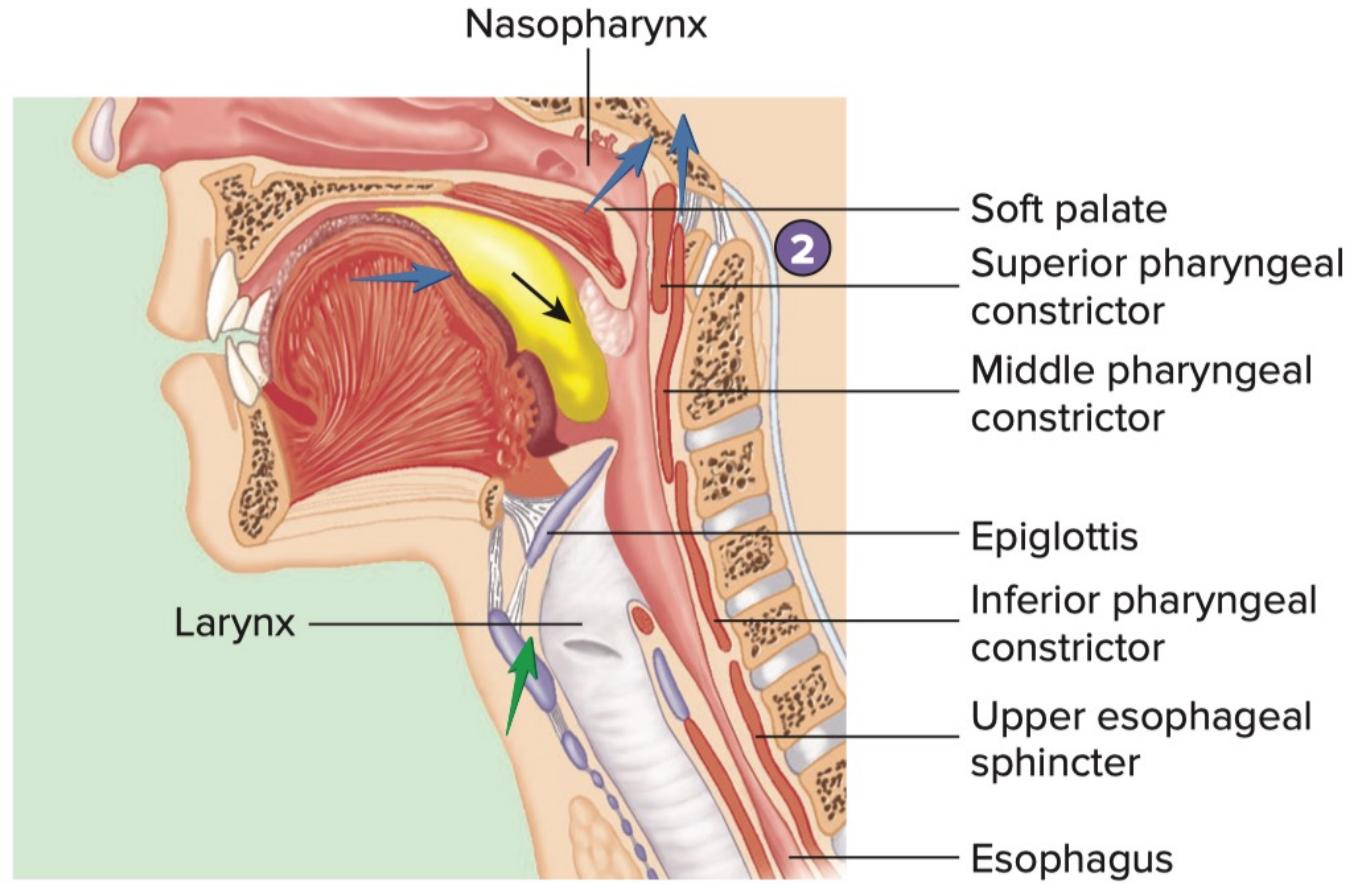
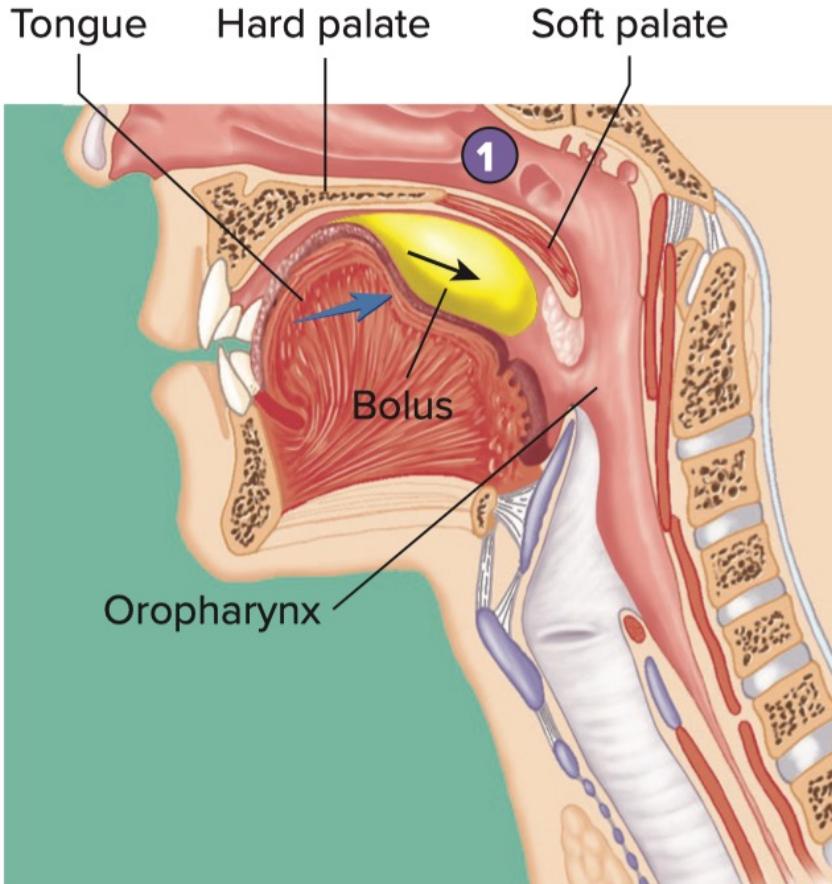


Deglution / Swallowing

- Divided into 3 Phases
- Oral Phase (voluntary)
 - Tongue pushes bolus to back of throat (oropharynx)
- Pharyngeal Phase (involuntary)
 - Initiated by bolus in oropharynx
 - Causes activation of swallowing center in medulla
 - Motor output from swallowing center
 - Causes soft palate to pull upward
 - Keeps bolus from entering nasopharynx
 - Causes epiglottis to cover the glottis
 - Keeps bolus from entering the larynx
 - Causes upper esophageal sphincter (UES) to relax
 - Bolus passes from laryngopharynx to esophagus
 - Breathing is inhibited during the pharyngeal phase
- Esophageal Phase (involuntary)
 - Initiated by bolus in the esophagus
 - Once bolus passes into esophagus , UES closes
 - Prevents reflux of bolus into pharynx
 - Bolus travels the length of the esophagus
 - Bolus enters stomach by passing through lower esophageal sphincter

Oral	Pharyngeal	Esophageal
Tongue pushes bolus to back of throat	Initiated by bolus in back of throat	Initiated by bolus in the esophagus

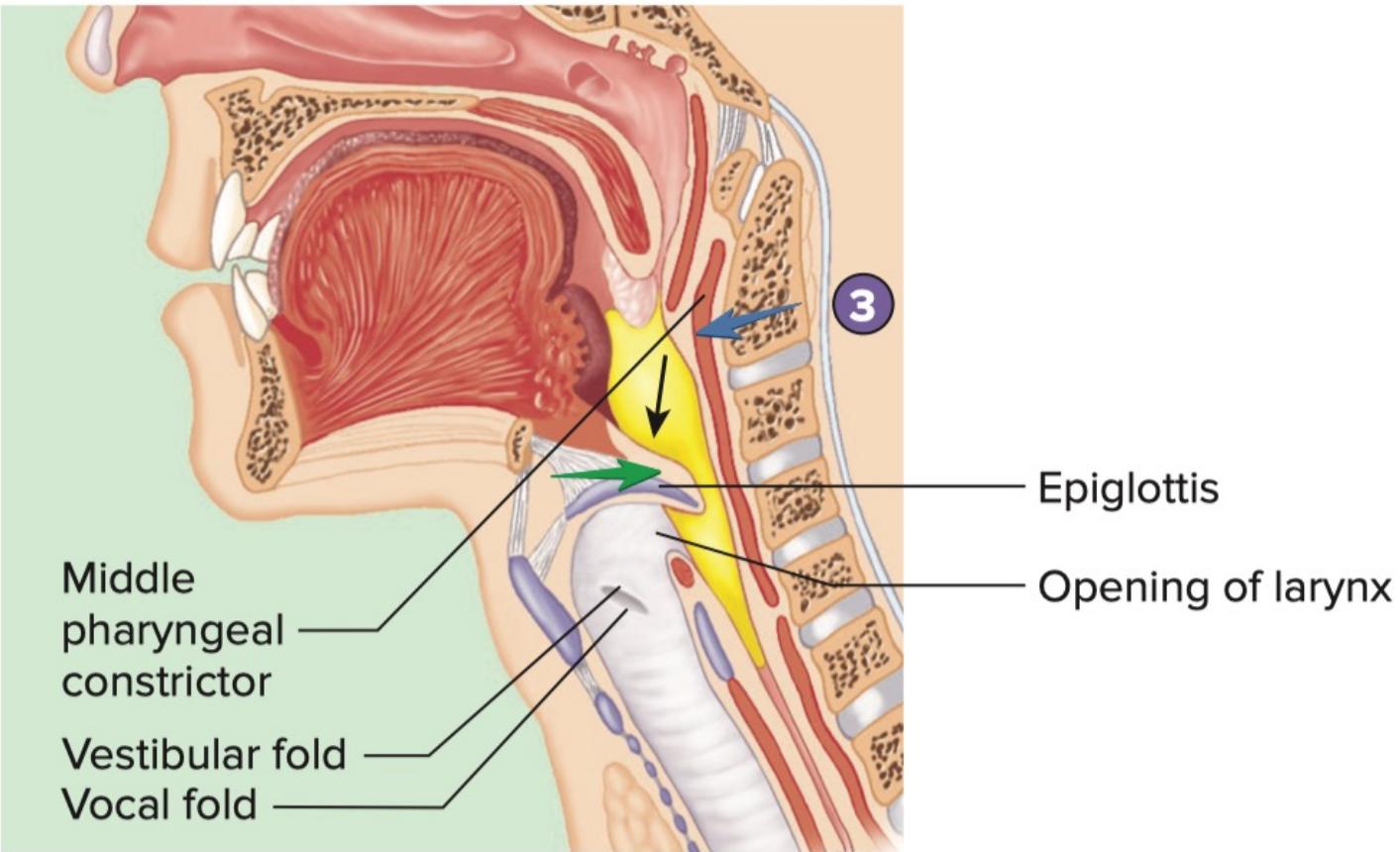
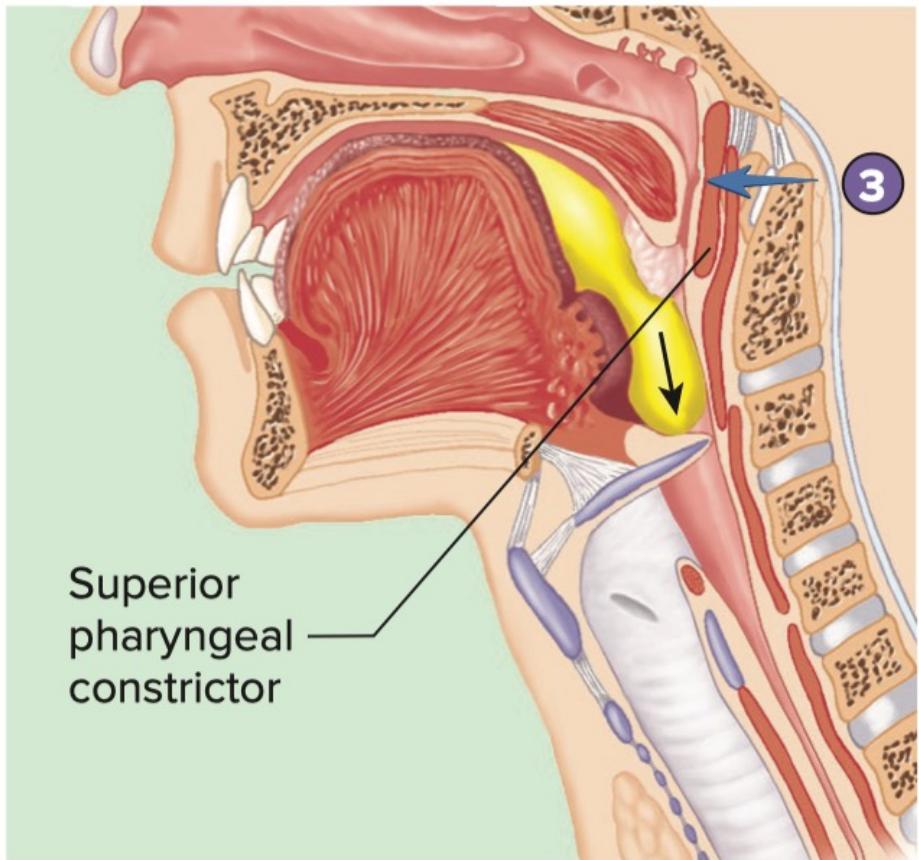
Oral and Pharyngeal Phases



1 During the **voluntary phase**, a bolus of food (yellow) is pushed by the tongue against the hard and soft palates and posteriorly toward the oropharynx (blue arrow indicates tongue movement; black arrow indicates movement of the bolus). Tan: bone; purple: cartilage; red: muscle.

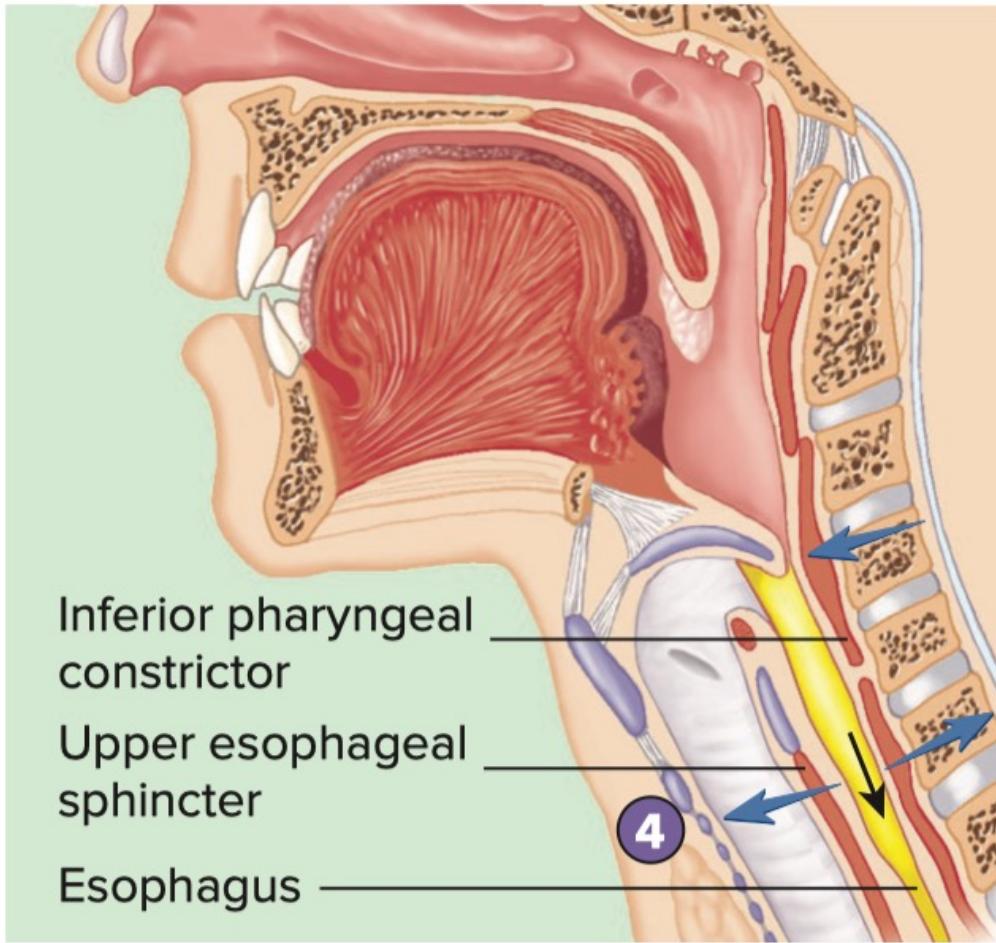
2 During the **pharyngeal phase**, the soft palate is elevated, closing off the nasopharynx. The pharynx and larynx are elevated (blue arrows indicate muscle movement; green arrow indicates elevation of the larynx).

Oral and Pharyngeal Phases

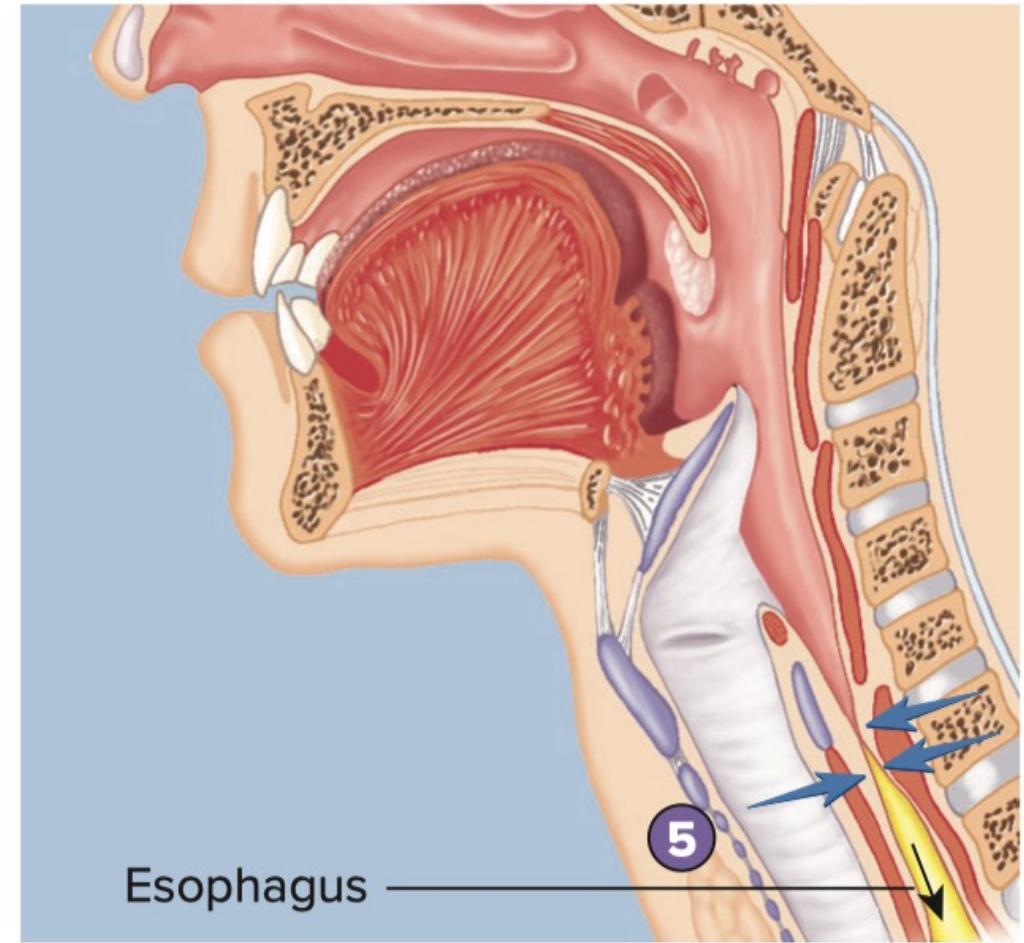


- ③ Successive constriction of the pharyngeal constrictors from superior to inferior (*blue arrows*) forces the bolus through the pharynx and into the esophagus. As this occurs, the vestibular and vocal folds expand medially to close the passage of the larynx. The epiglottis (*green arrow*) is bent down over the opening of the larynx largely by the force of the bolus pressing against it.

Esophageal Phase



- 4 As the inferior pharyngeal constrictor contracts, the upper esophageal sphincter relaxes (outwardly directed *blue arrows*), allowing the bolus to enter the esophagus.



- 5 During the **esophageal phase**, the bolus is moved by peristaltic contractions of the esophagus toward the stomach (inwardly directed *blue arrows*).

Motility

- Peristalsis
 - Wavelike contractions of the digestive tract that propel contents
 - Circular layer contracts behind and relaxes in front of contents
 - Longitudinal layer relaxes behind and contracts in front of contents
- Mixing
 - Series of contractions and relaxations of circular layer that mix the contents
- Sphincters
 - Ring of smooth muscle
 - Exception is UES and external anal sphincter , are a ring of skeletal muscle
 - Exhibit sustained contractility , which keeps them closed
 - When relaxed , allows for the passage of digestive tract contents
 - Sphincters are relaxed by peristalsis
 - Sphincter tone influences motility
 - Higher tone decreases motility
 - Lower tone increases motility

* Motility

- move

↳ peristalsis

pharynx
esophagus
stomach
small intestine
large intestine



↳ mass movement : large intestine

- mix

↳ stomach : mix

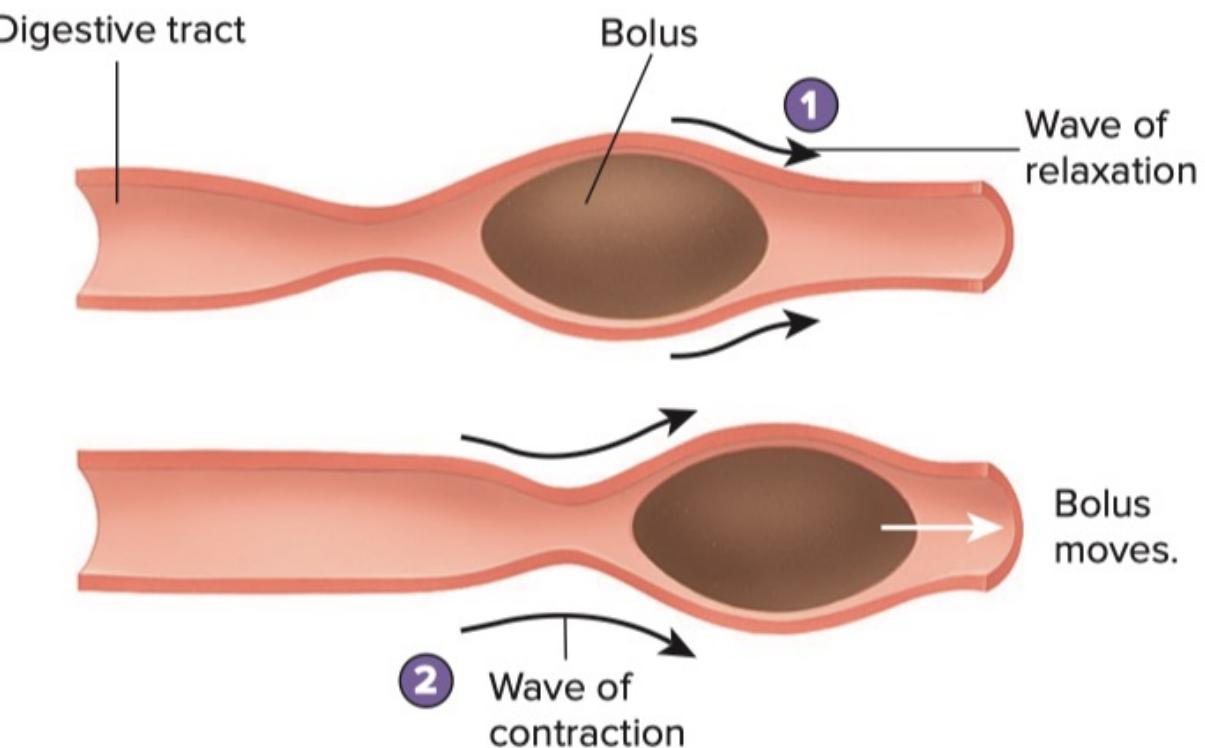
↳ small intestine : segmentation

↳ large intestine : haustration

Motility

① A wave of smooth muscle relaxation moves ahead of the bolus, allowing the digestive tract to expand.

② A wave of contraction of the smooth muscle behind the bolus propels it through the digestive tract.

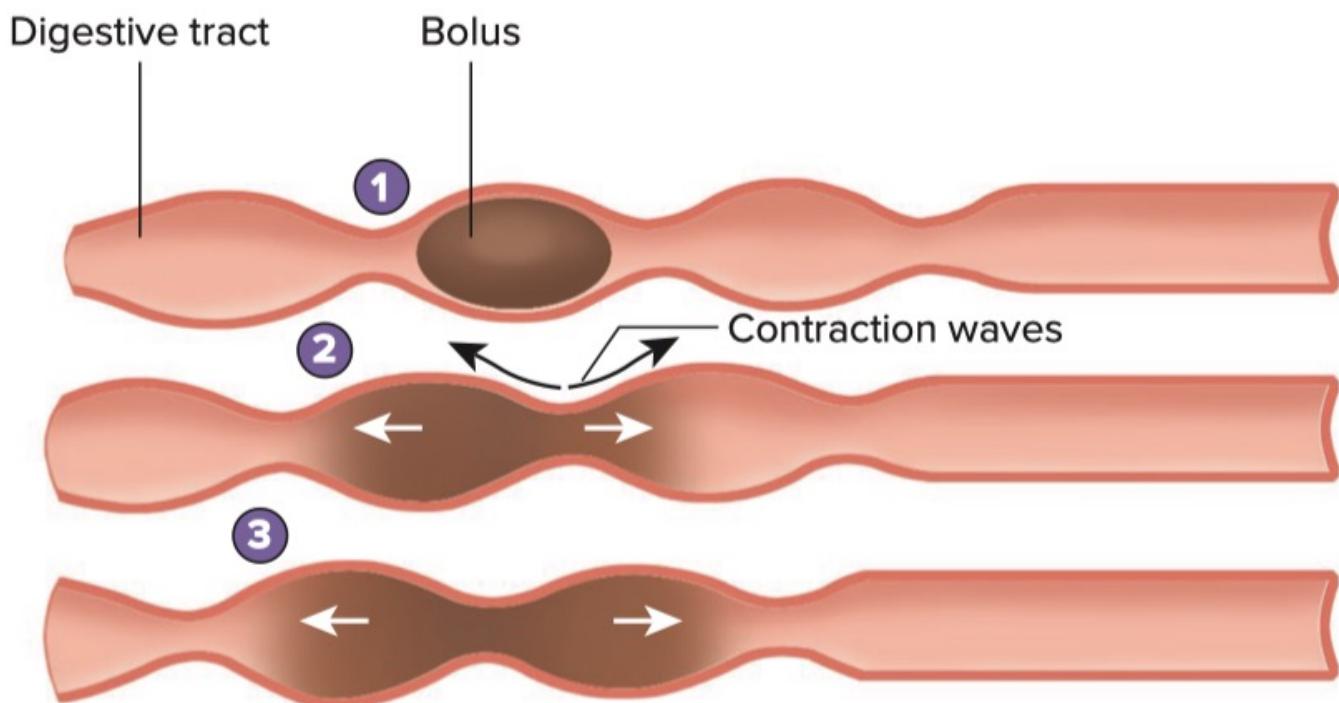


PROCESS FIGURE 24.2 Peristalsis and Segmental Contractions

Waves of smooth muscle contraction push food and waste through the digestive tract.

Motility

- 1 A bolus within the tract begins at one location.
- 2 Segments of the digestive tract alternate between contraction and relaxation.
- 3 The bolus spreads out in both directions.



PROCESS FIGURE 24.3 Segmental Contractions

Smooth muscle contractions in the wall of the small intestine disperse digesting food throughout its lumen.

circular muscle relaxes

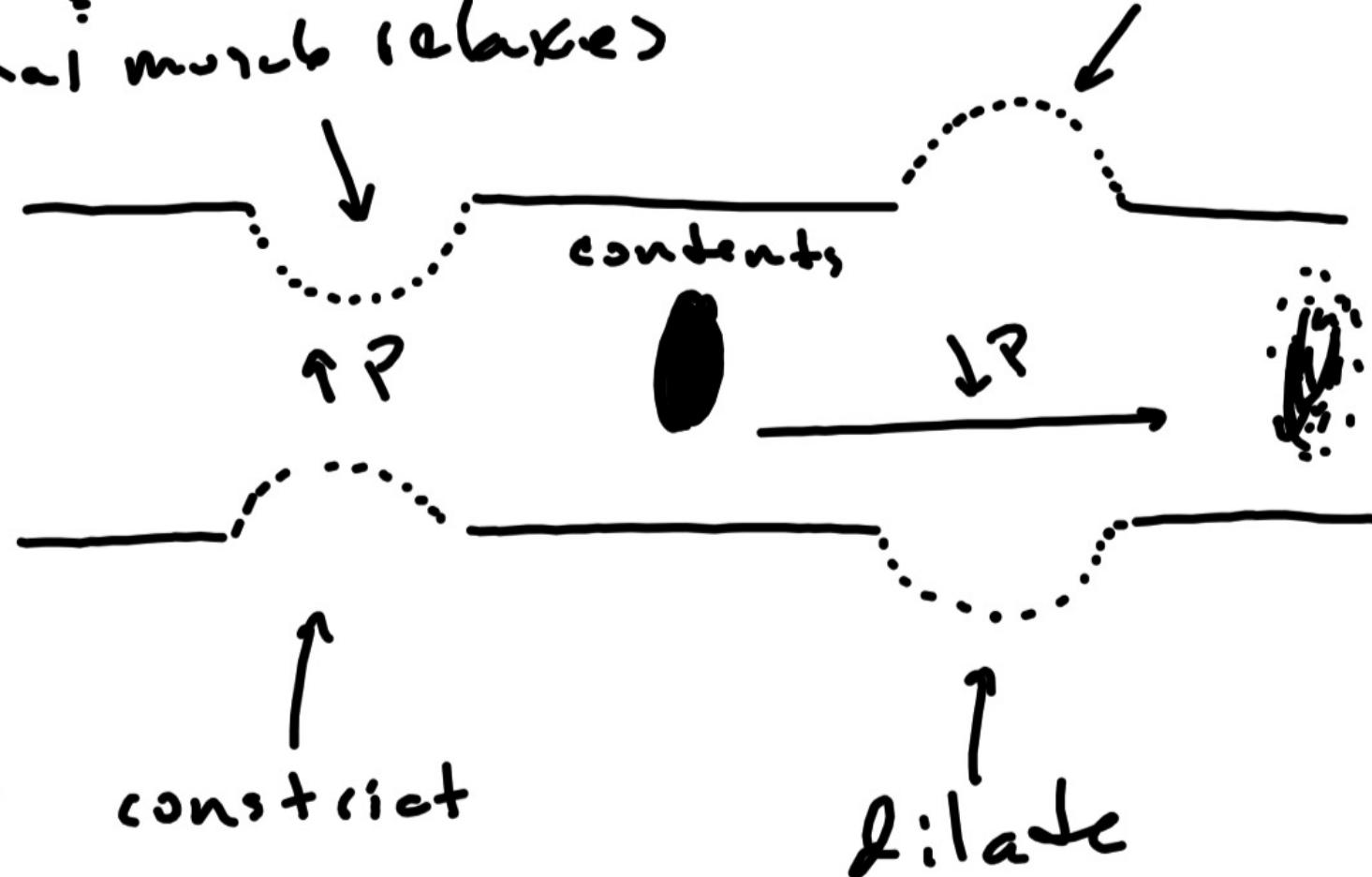
longitudinal muscle contracts

circular muscle contracts

longitudinal muscle relaxes

peristalsis

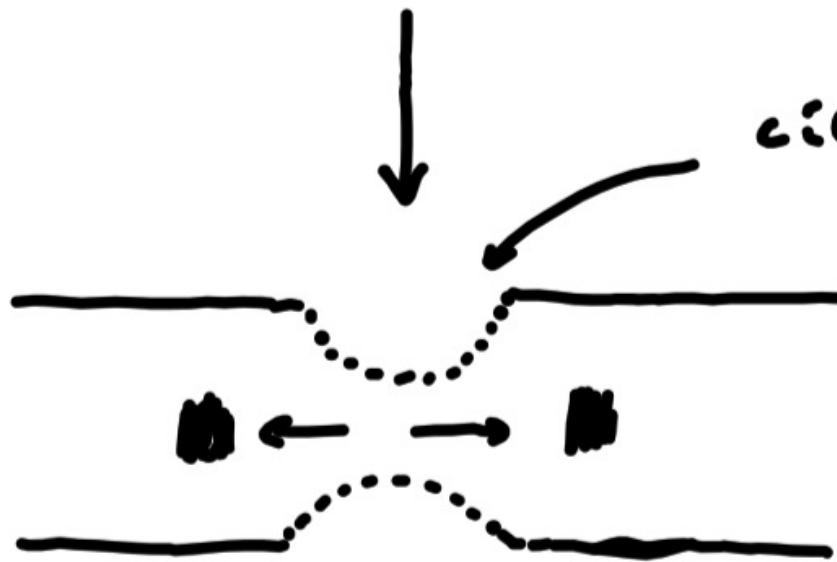
caused by presence of contents



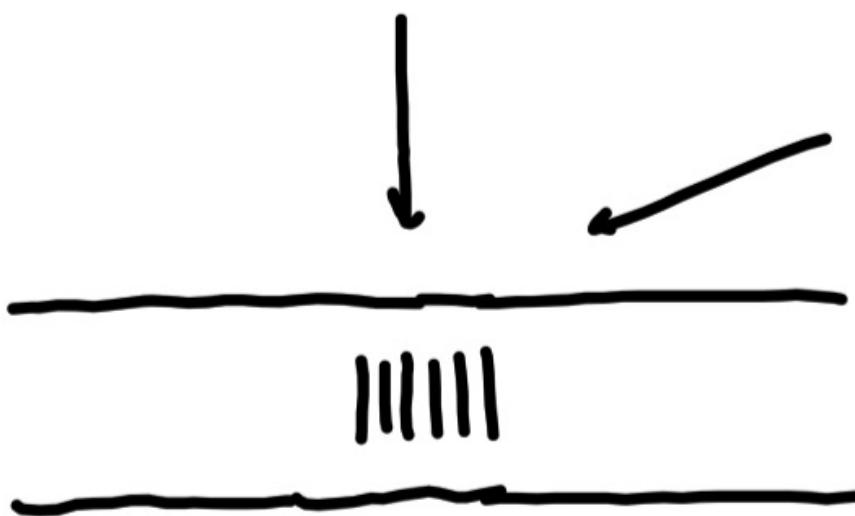
Esophageal Motility

- Peristalsis propels bolus toward the stomach during esophageal phase of swallowing
 - Bolus takes approximately 3 to 10 seconds to move through the esophagus
 - Peristalsis causes relaxation of lower esophageal sphincter (LES)
 - Allows contents of esophagus to empty into the stomach
 - Should be closed in the absence of esophageal peristalsis
 - Prevents contents of stomach from refluxing into esophagus
 - Gastroesophageal Reflux Disease (GERD)

mixing

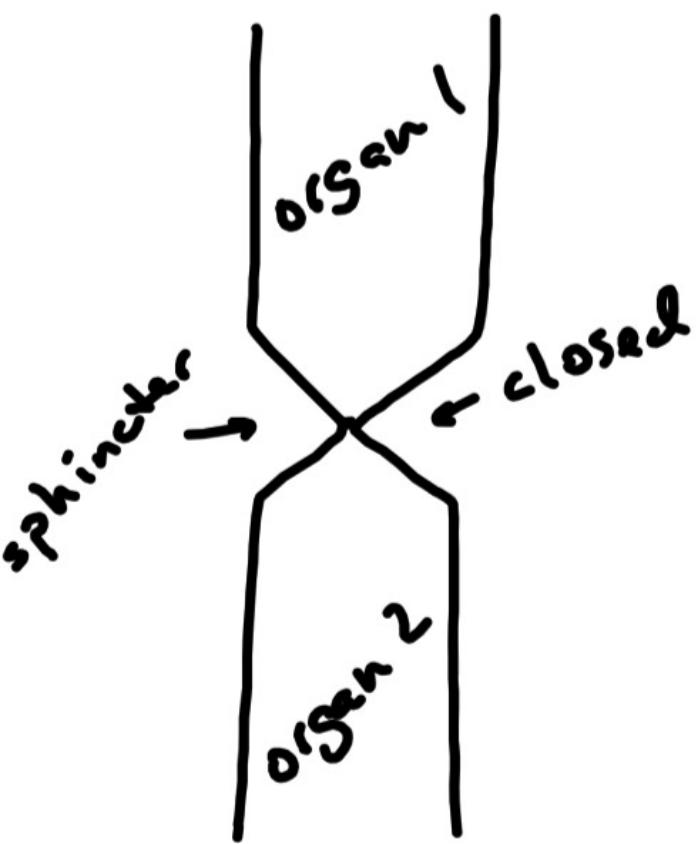


circular muscle contracts



circular muscle relaxes

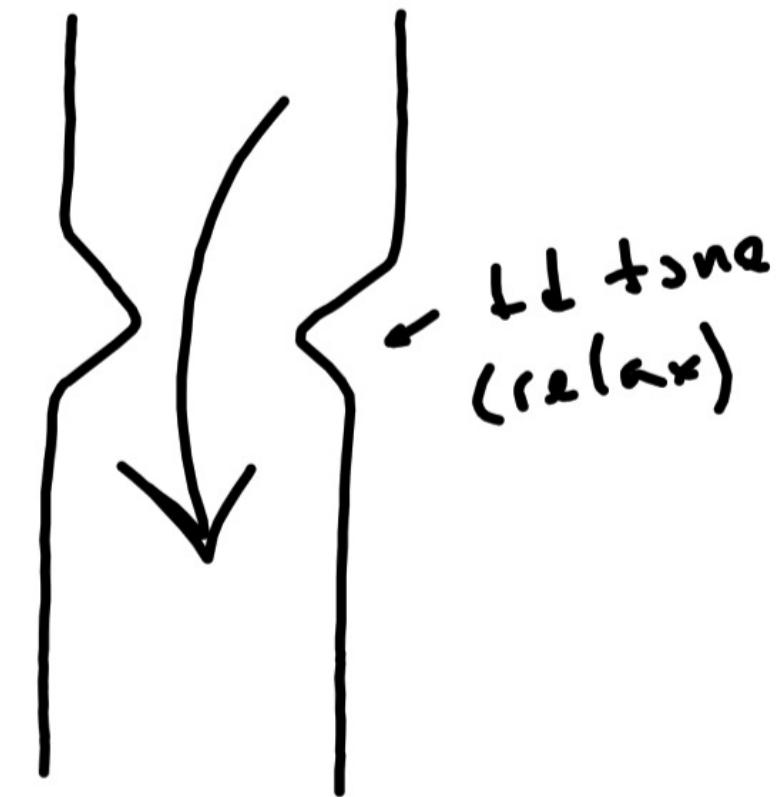
peristalsis causes sphincters to relax



∅ motility



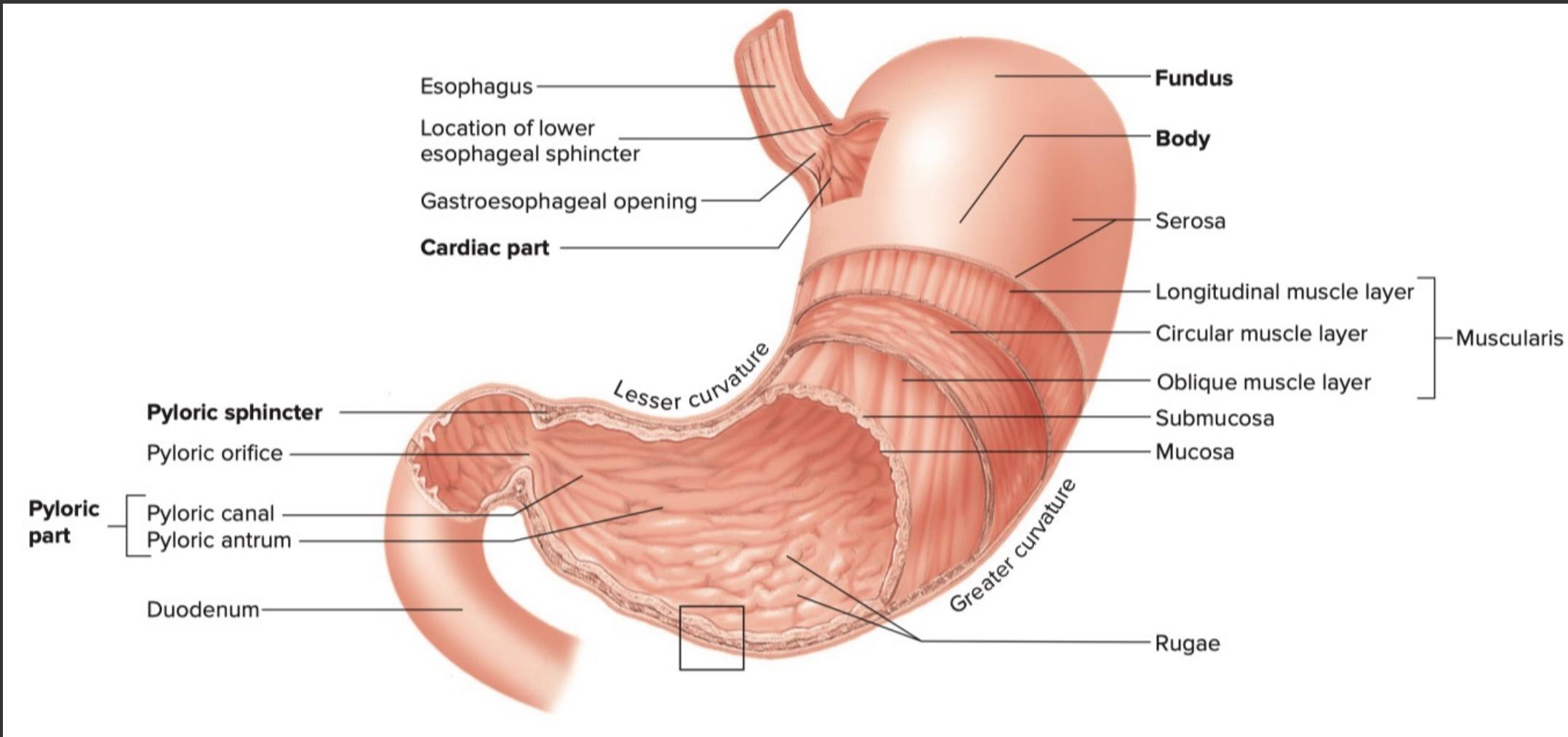
↑ motility



↑↑ motility

Stomach Motility

- Main function is mixing
- Mixes bolus with gastric juices to form chyme

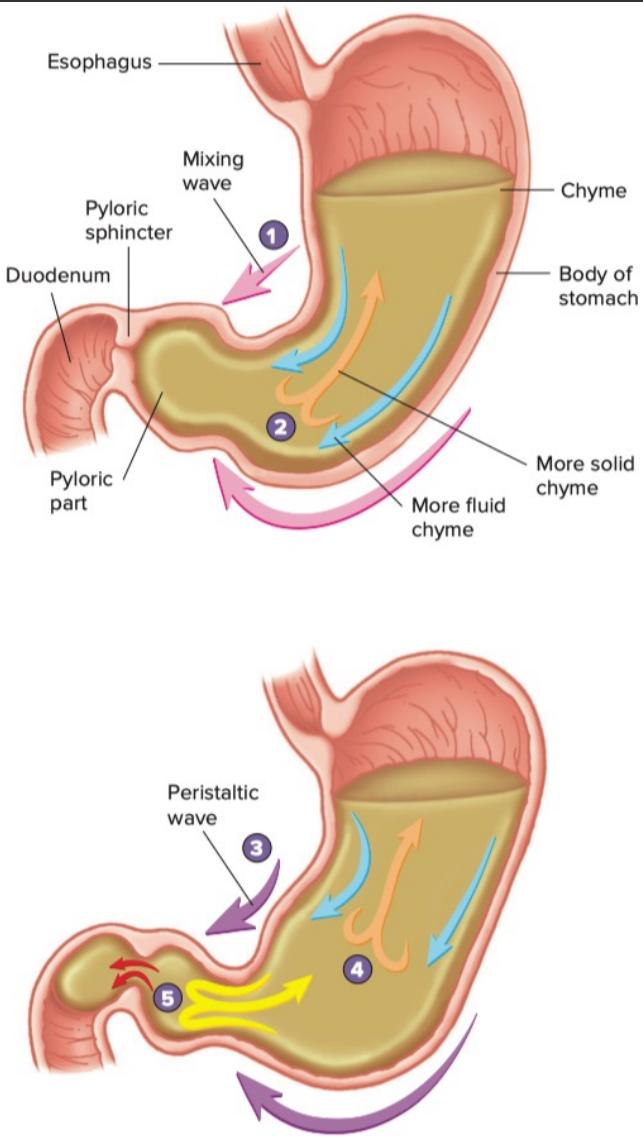


Stomach Motility

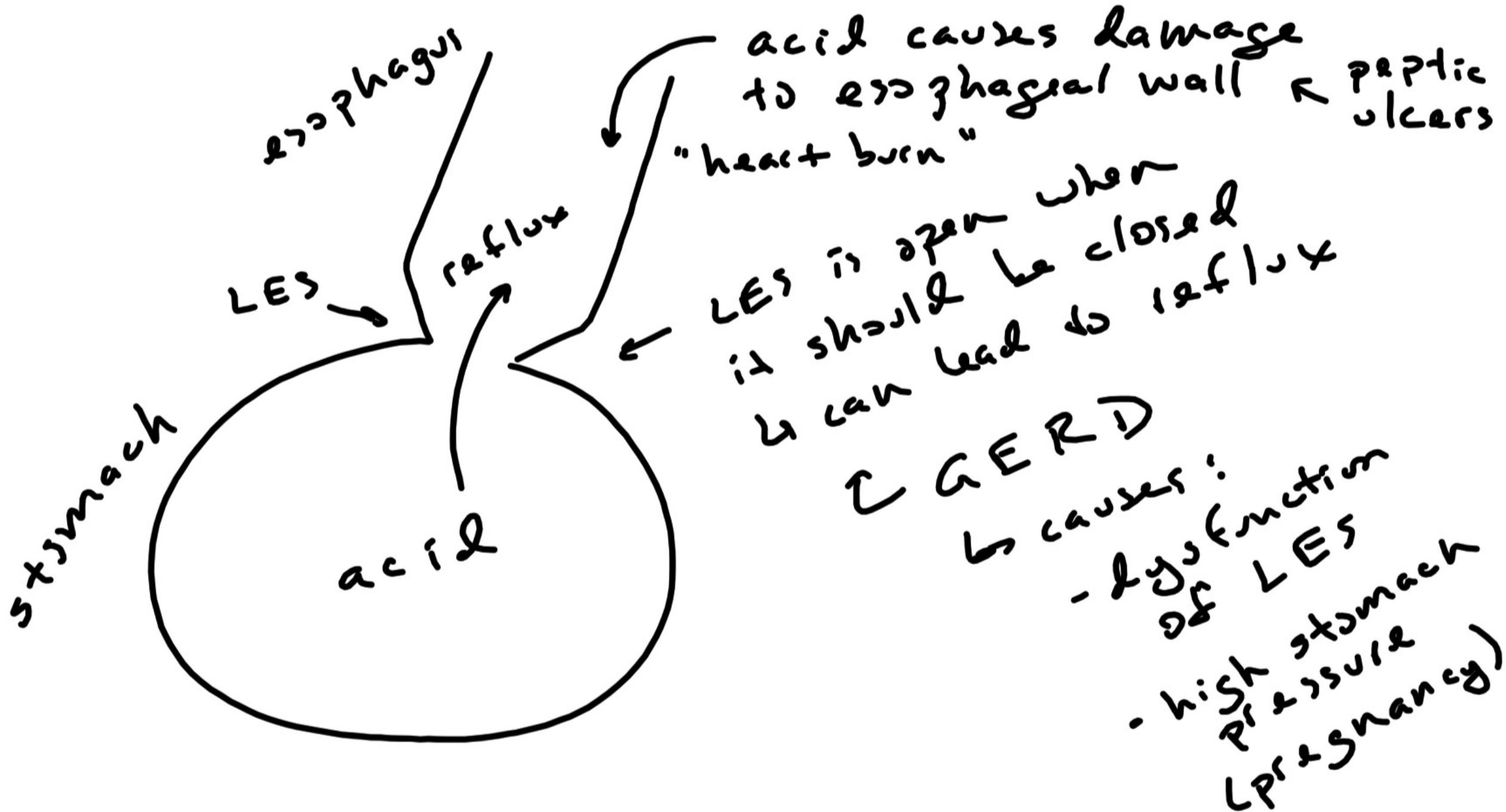
- Gastric emptying via peristalsis
 - Each peristaltic wave empties approximately 1% of stomach contents
 - Approximately three peristaltic waves per minute
 - Causes opening of pyloric sphincter , so contents empty into duodenum
 - Takes approximately 2 to 3 hours to empty a "normal" sized meal
 - Rate of emptying dependent on contents
 - Carbohydrates empty the fastest
 - Lipids empty the slowest

Stomach Motility

- 1 A mixing wave initiated in the body of the stomach progresses toward the pyloric sphincter (pink arrows directed inward).
- 2 The more fluid part of the chyme is pushed toward the pyloric sphincter (blue arrows), whereas the more solid center of the chyme squeezes past the peristaltic constriction back toward the body of the stomach (orange arrow).
- 3 Peristaltic waves (purple arrows) move in the same direction and in the same way as the mixing waves but are stronger.
- 4 Again, the more fluid part of the chyme is pushed toward the pyloric region (blue arrows), whereas the more solid center of the chyme squeezes past the peristaltic constriction back toward the body of the stomach (orange arrow).
- 5 Peristaltic contractions force a few milliliters of the mostly fluid chyme through the pyloric opening into the duodenum (small red arrows). Most of the chyme, including the more solid portion, is forced back toward the body of the stomach for further mixing (yellow arrow).

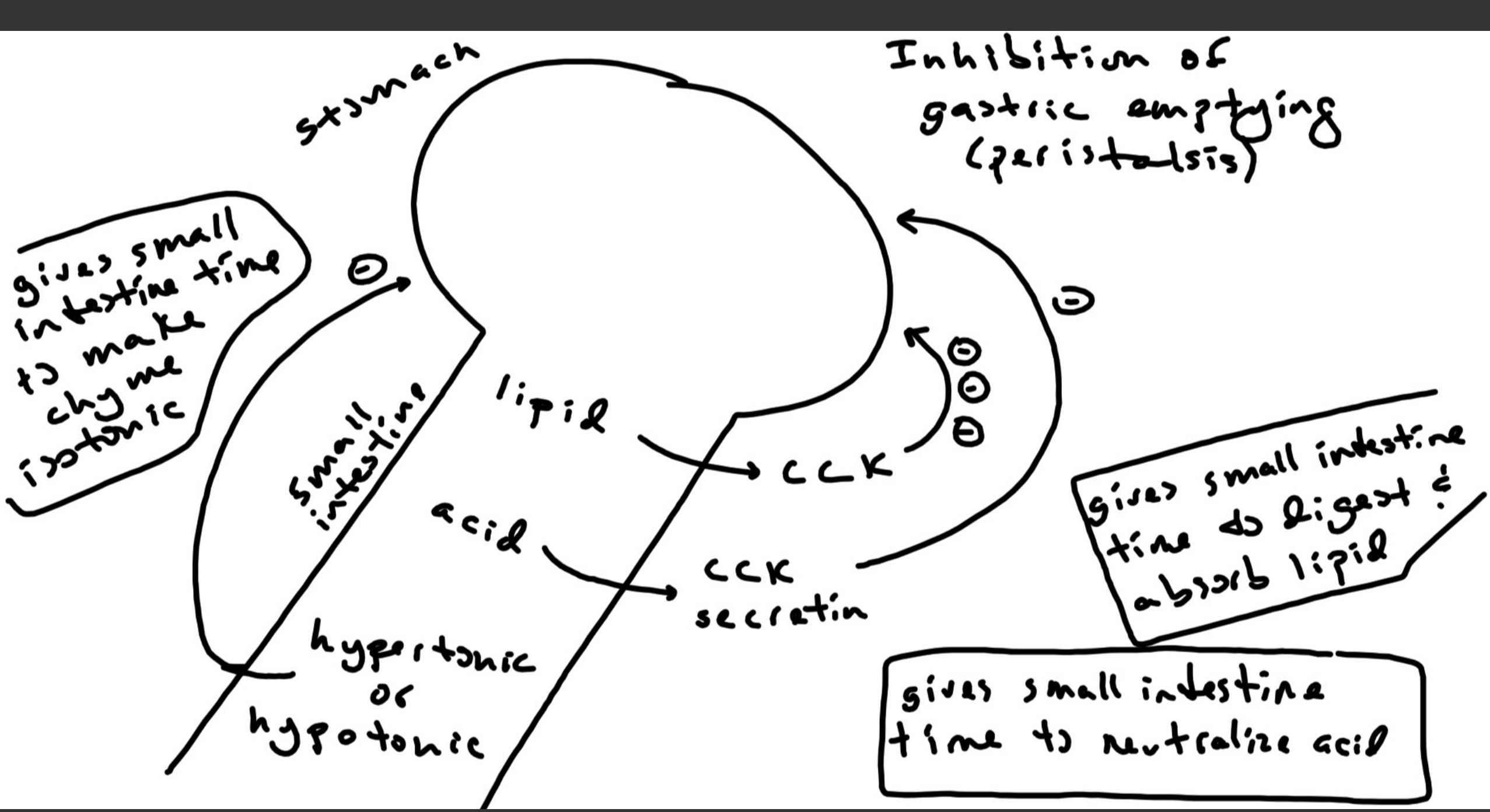


- **Stimulation of gastric emptying**
 - Increased volume of contents in stomach
 - Increased fluidity of chyme in stomach
- **Inhibition of gastric emptying**
 - Decreased fluidity of chyme in stomach
 - Lipids in duodenum (most potent inhibitor)
 - Mediated by cholecystokinin
 - Digestion and absorption of lipids in small intestine is slow
 - Gives small intestine more time to digest and absorb lipid
- **Chyme below pH 2.0 in duodenum**
 - Mediated by secretin and cholecystokinin
 - Gives small intestine more time to neutralize acid
- **Hypertonic or hypotonic contents in duodenum**
 - Gives duodenum more time to make chyme isotonic

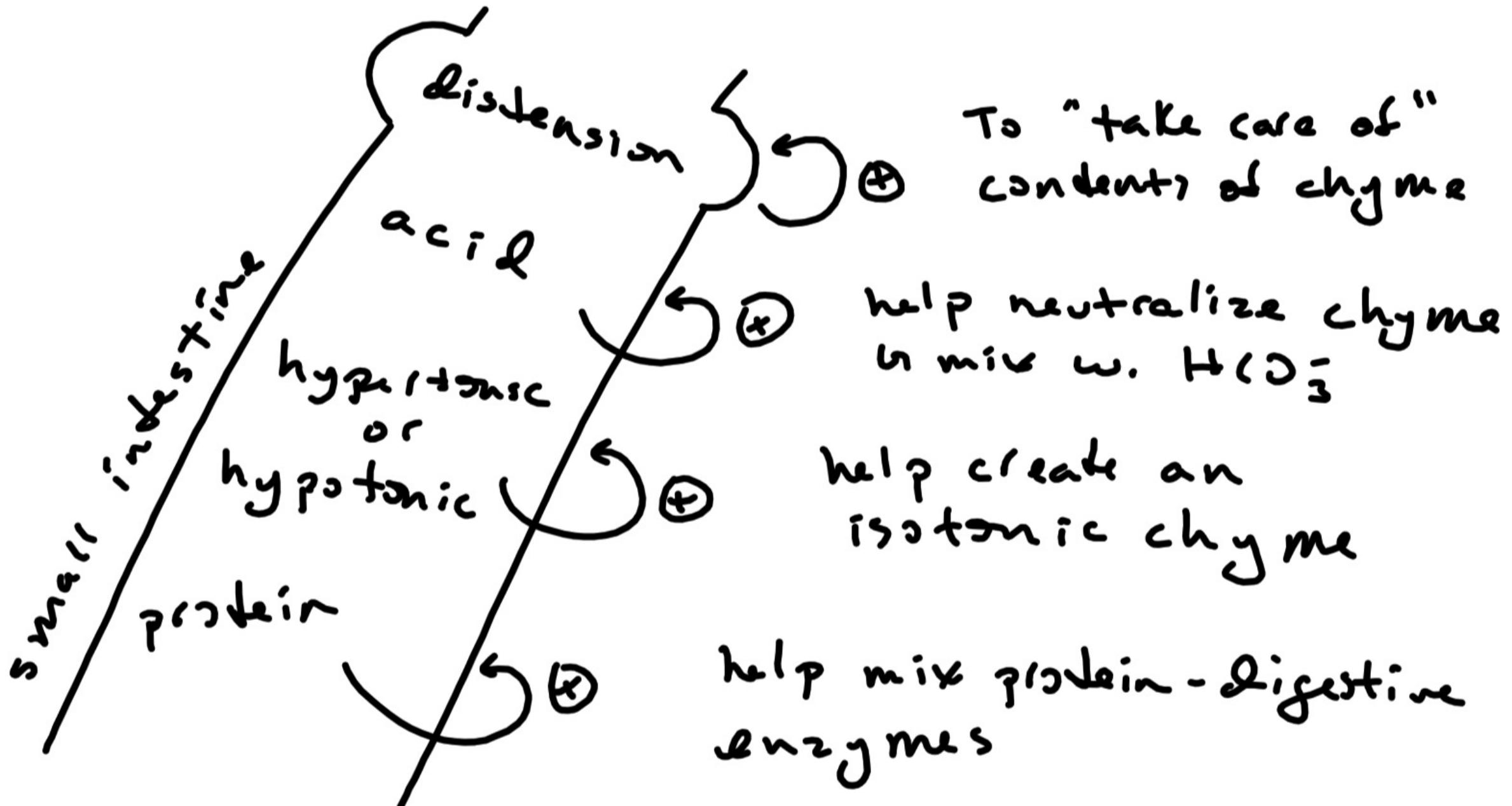


Small Intestine Motility

- Segmentation / Mixing
 - Most prevalent movement of the small intestines
- Peristalsis
 - Takes approximately 4 to 6 hours for chyme to move through small intestine
- Stimulation of Motility
 - Distension (by presence of emptied chyme from stomach) of duodenum
 - Hypertonic or hypotonic chyme in duodenum
 - Low pH of chyme in duodenum
 - Protein digested contents in the duodenum



Stimulation of Segmentation

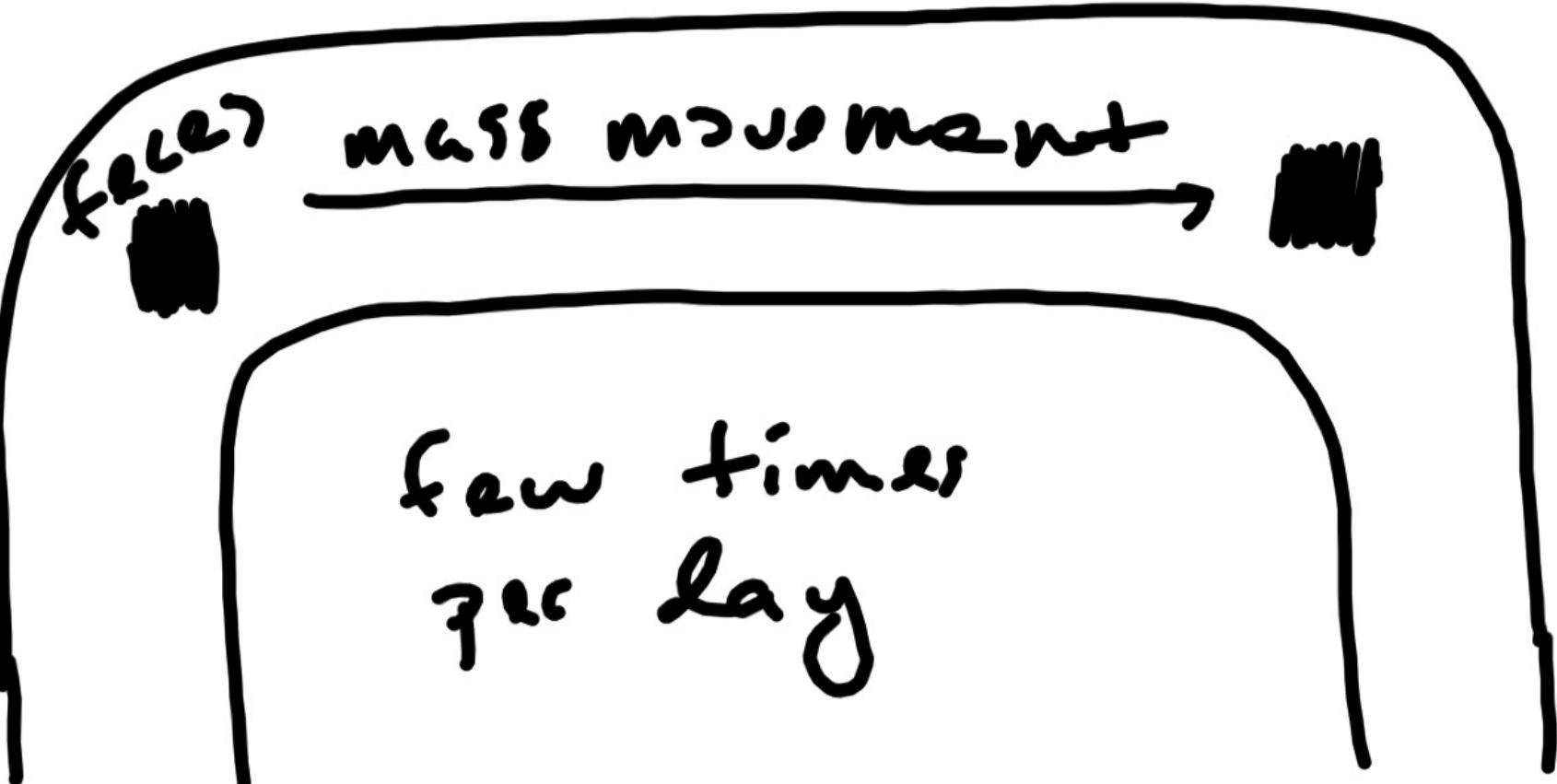


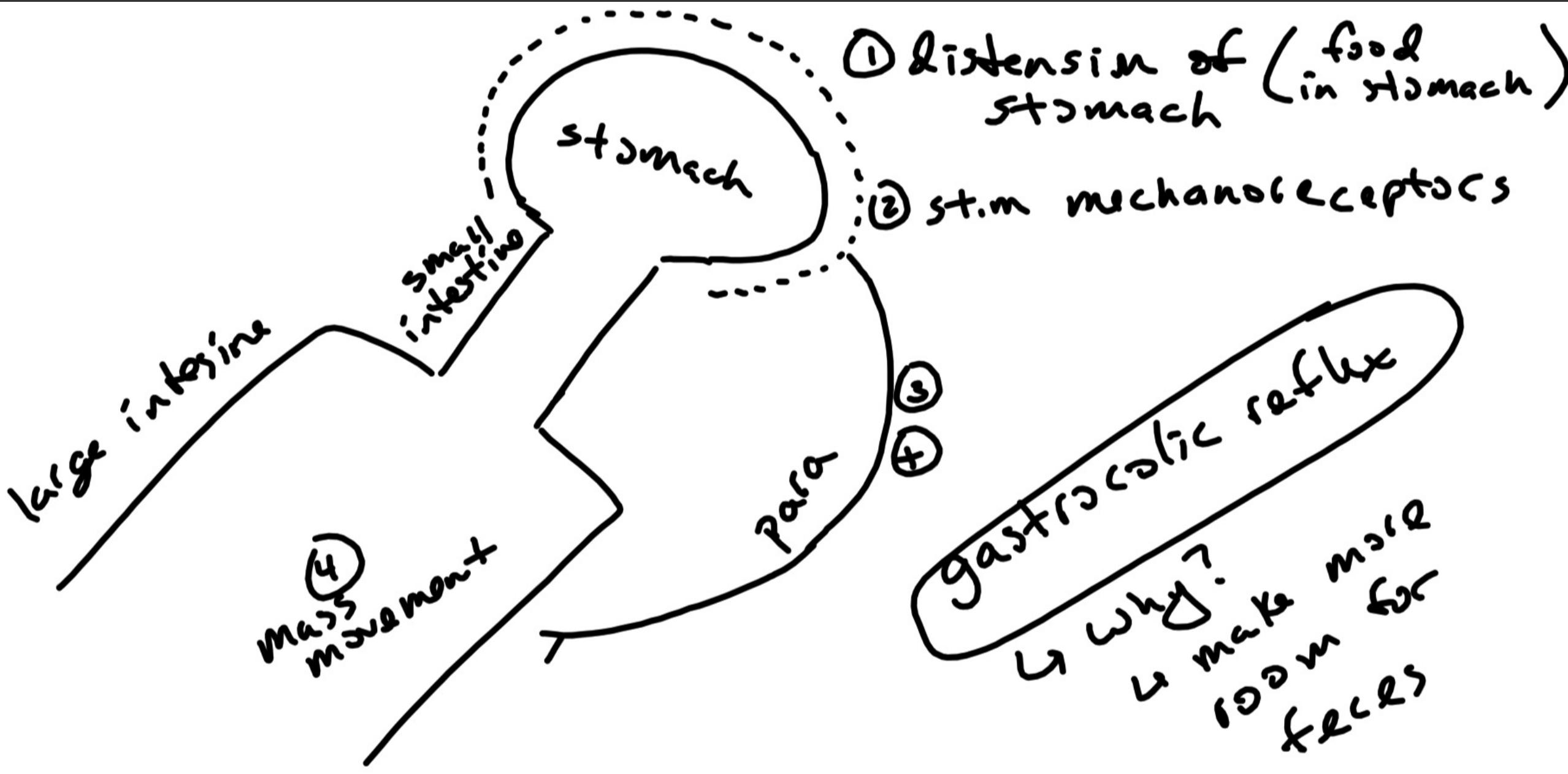
Large Intestine Motility

- Haustration / Mixing of Contents
 - Aids in formation of feces
 - Water , undigested material , mucous , flora (30% of dry weight)
 - Brown due to bile pigments (ie , bilirubin)
- Peristalsis
 - Very little peristalsis (occurs in the ascending colon)
- Mass Movement (not peristalsis)
 - Typically occurs a few times per day
 - Gastric Reflex :
 - Initiated by distension of stomach when food is present
 - Activates parasympathetic reflex that stimulates large intestine
 - Causes mass movement
 - Very slow compared to the rest of the digestive tract
 - Normally , 18 to 24 hours for material to pass through large intestine
 - Slow movement allows for the incubation of flora ("good" bacteria)
 - Flora feed off of nutrients left in chyme
 - Flora produce vitamin K and certain B vitamins
- Elimination
 - Rectosphincteric Reflex :
 - Initiated by distention of the rectum
 - Parasympathetics reflexively contract the rectal walls
 - Parasympathetics reflexively relax the internal anal sphincter
 - Voluntary relaxation of the external anal sphincter expels feces
 - Defecation : feces expelled (not part of rectosphincteric reflex)
 - Voluntary contraction of abdominal muscles
 - Voluntary relaxation of external anal sphincter

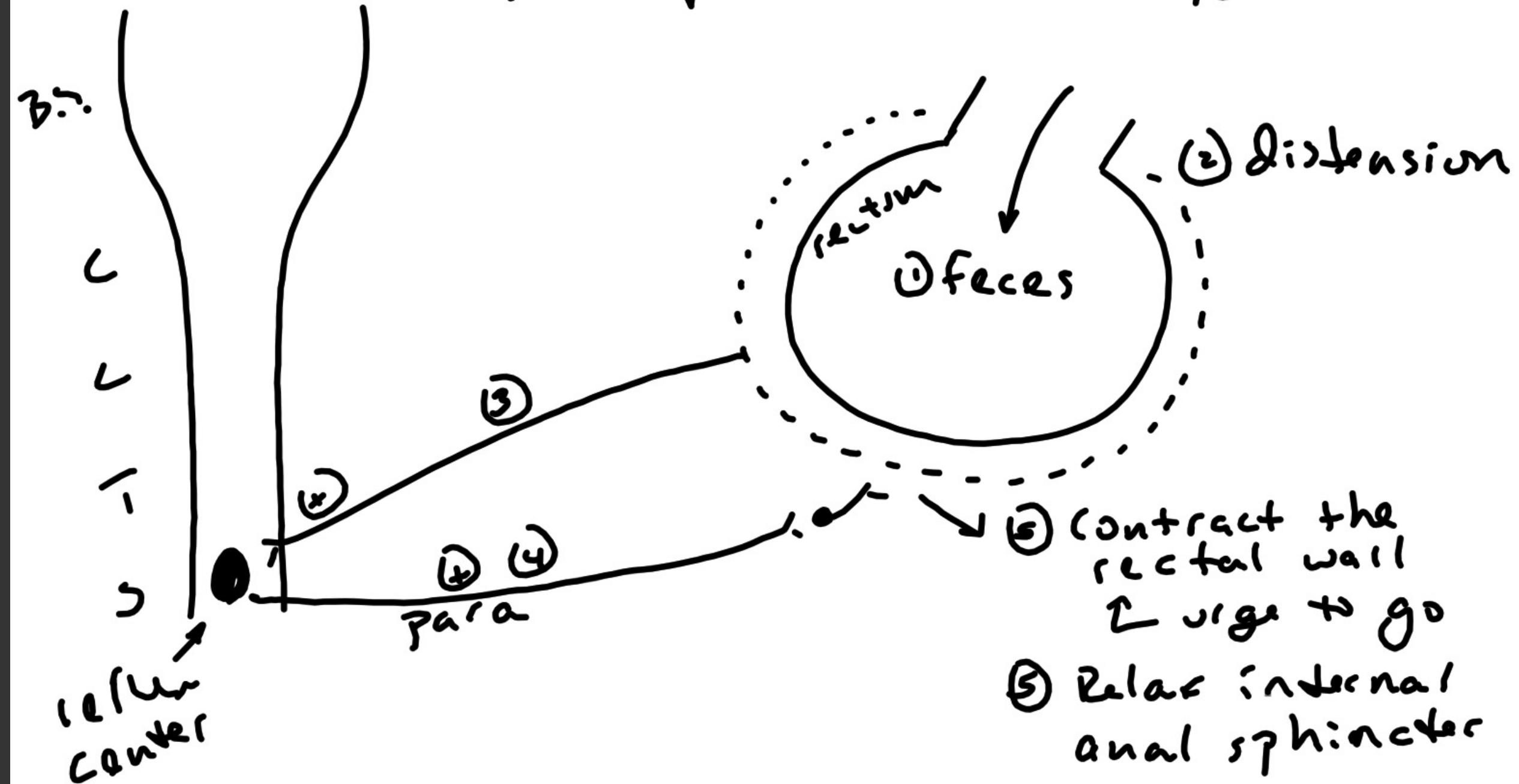
transverse

as long as





Rectosphincteric Reflex



Oral Cavity

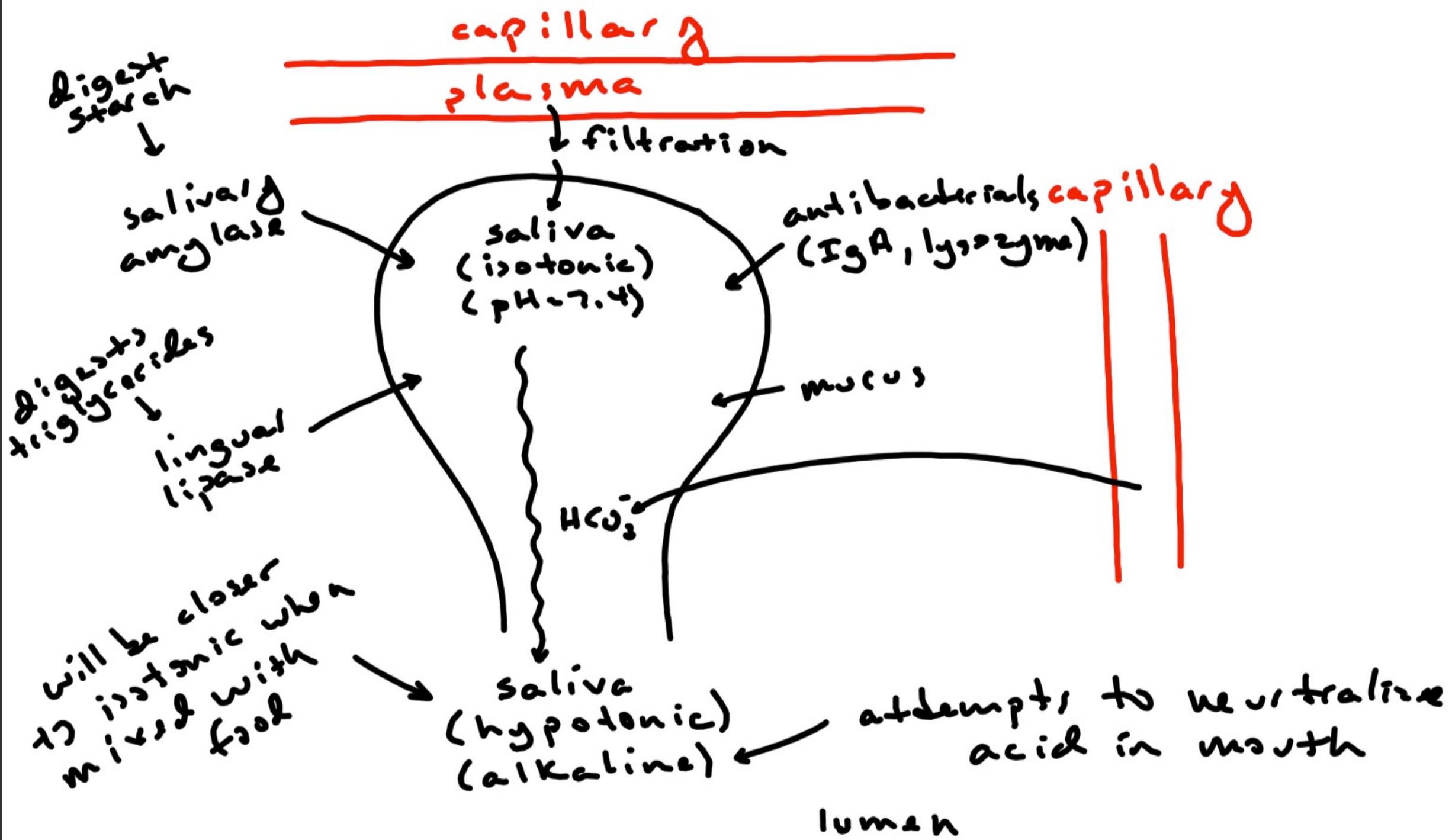
- Teeth
 - Mechanically break down food
- Tongue
 - Mechanically breaks down food
 - Presses food against the hard palate
- Salivary Glands
 - Production of saliva (1 to 1.5 liters per day)
 - Aqueous component
 - Organic component

Saliva - Aqueous Component

- Initial step in formation is an isotonic filtrate of the plasma
- Final aqueous composition of saliva is hypotonic and alkaline
 - Aids in neutralizing oral cavity acids

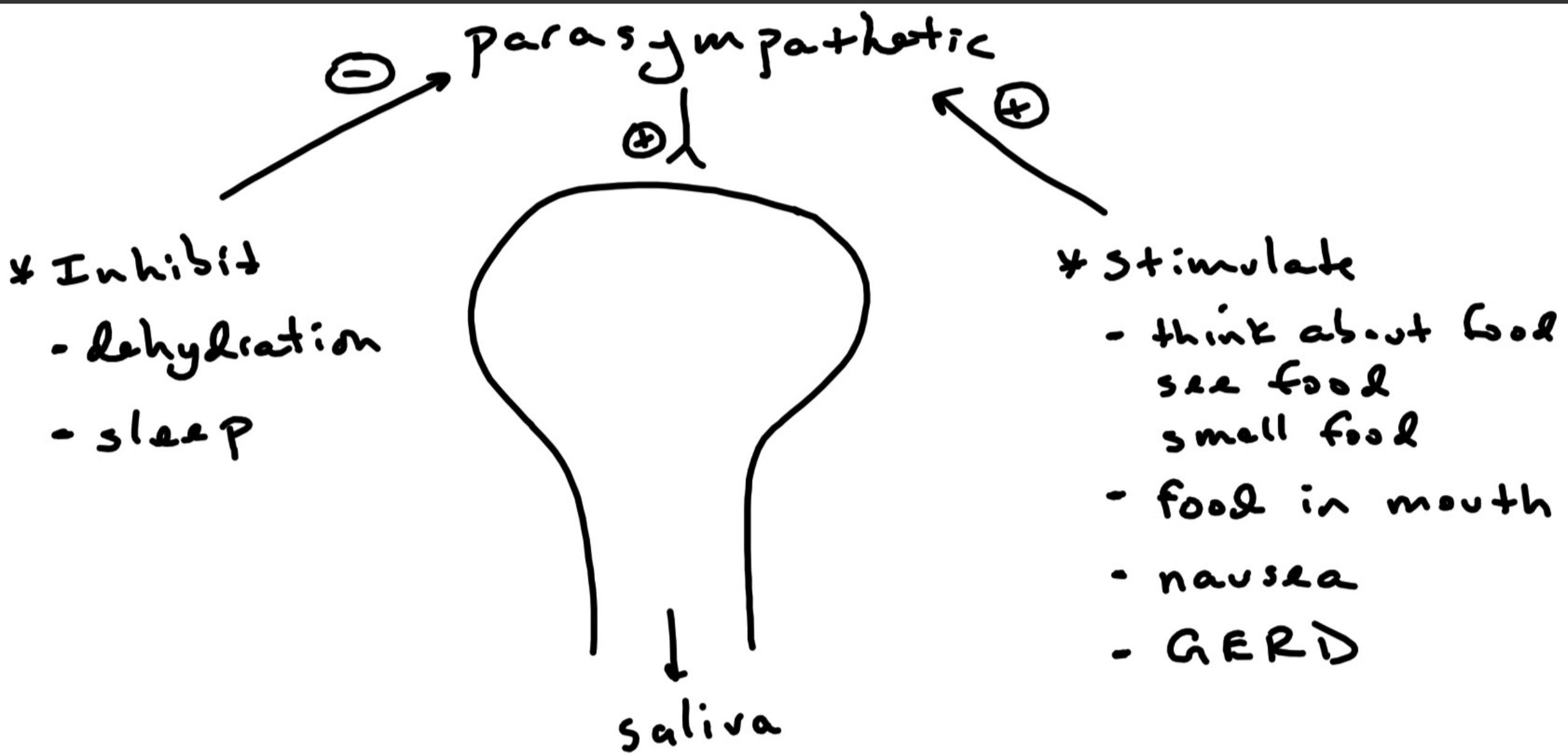
Saliva - Organic Component

- Salivary Amylase
 - Begins the chemical digestion of starch
 - Inactivated by low pH of the stomach
- Lingual Lipase
 - Digests triglycerides in emulsified fats
 - (eg , fats in creams , egg yolks)
 - Continuous to work in the stomach
 - Minor role of lipid digestion in adults
- Mucus
 - Lubricates mouth and esophagus
 - Softens food to help form a bolus
- Antibacterials
 - Antibodies (IgA) and lysozyme



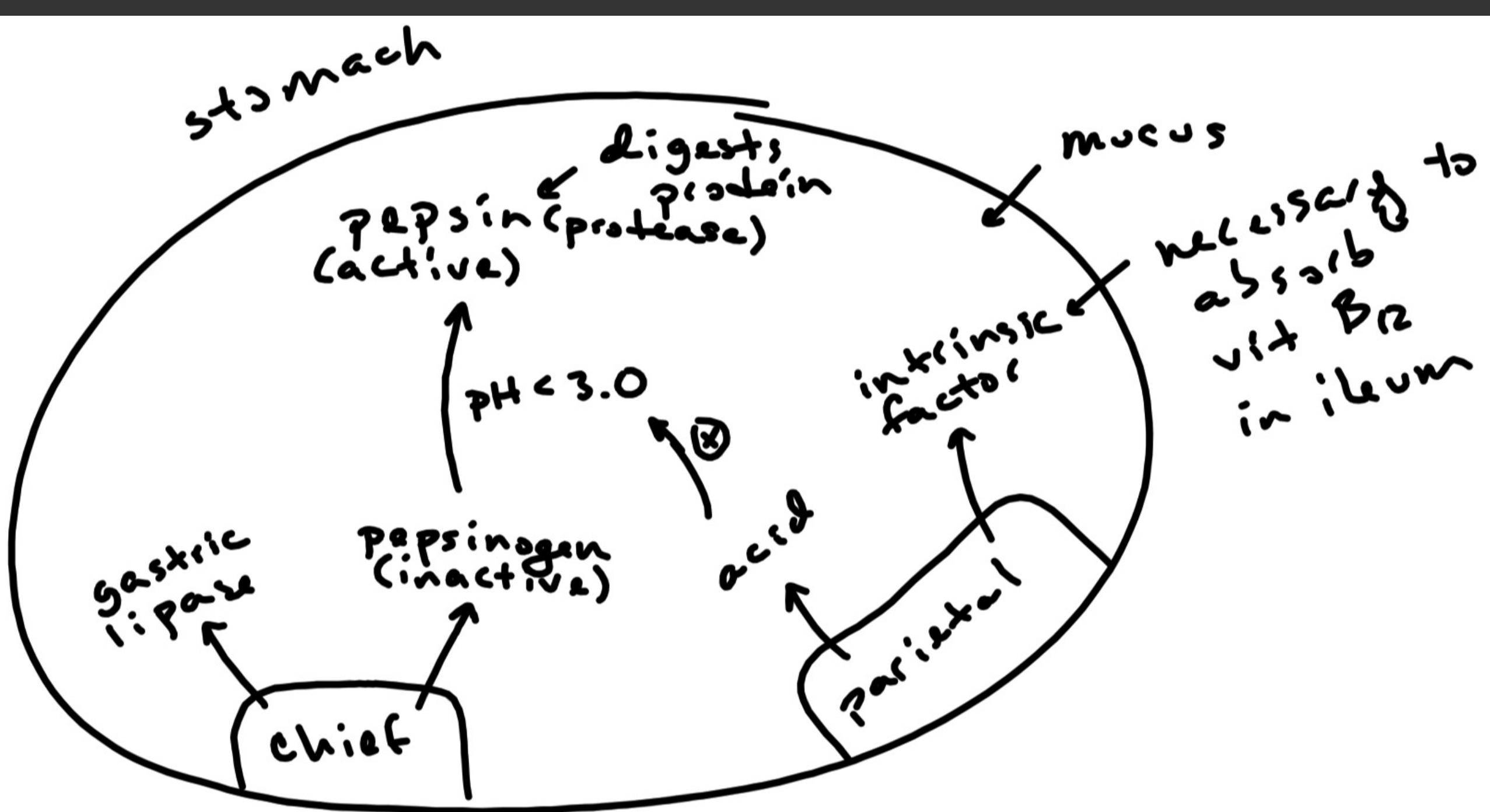
Regulation of Saliva Production

- Parasympathetic Nervous System (main influence)
 - Stimulates aqueous and enzyme secretions
- Sympathetic Nervous System
 - Stimulates mucous secretion
- Stimulation of Release
 - Sight and smell of food
 - Presence of anything in the mouth
 - Nausea
 - GERD
- Inhibition of Release
 - Dehydration
 - Sleep



Stomach

- Very little digestion occurs in the stomach

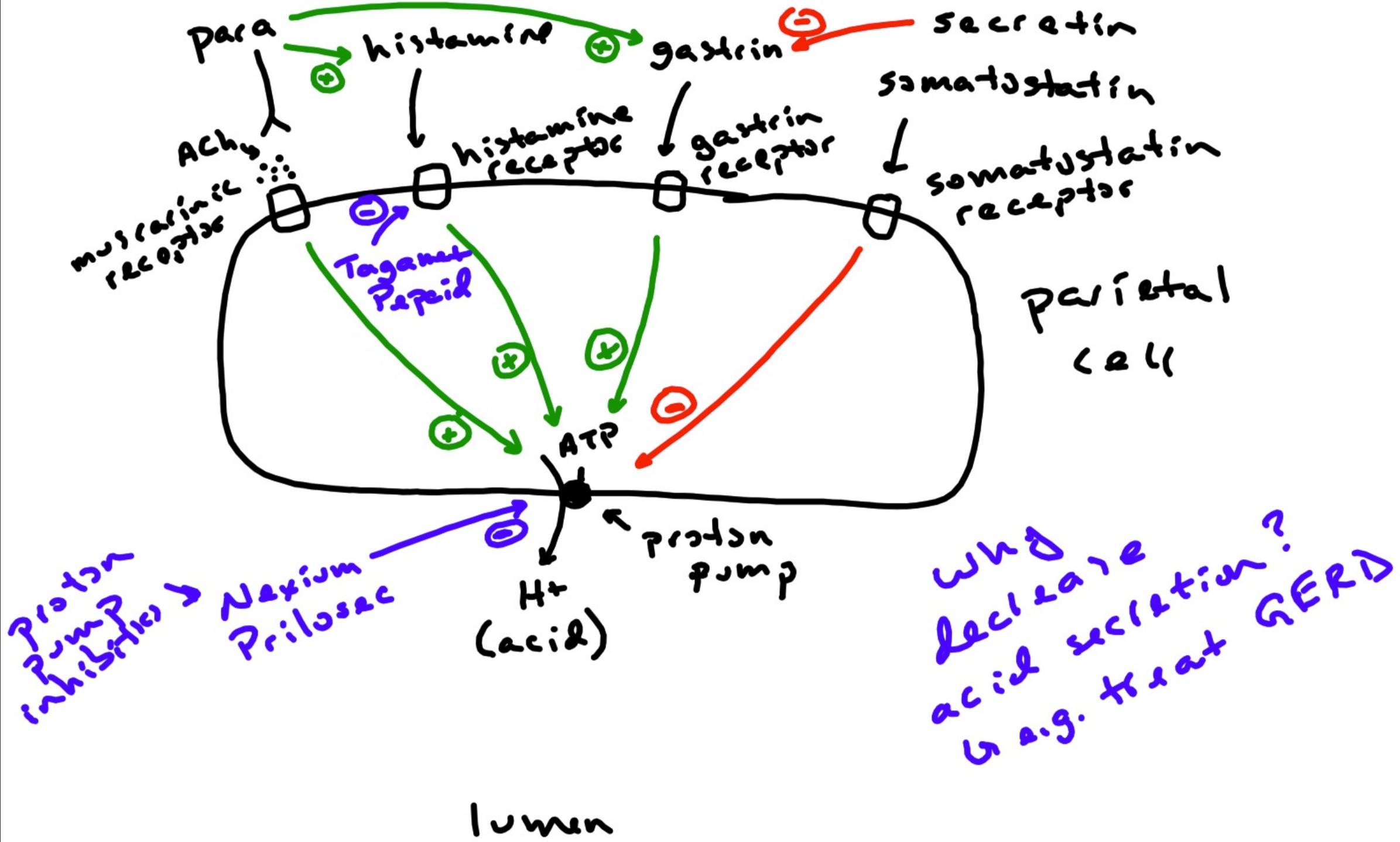


Stomach - Secretions

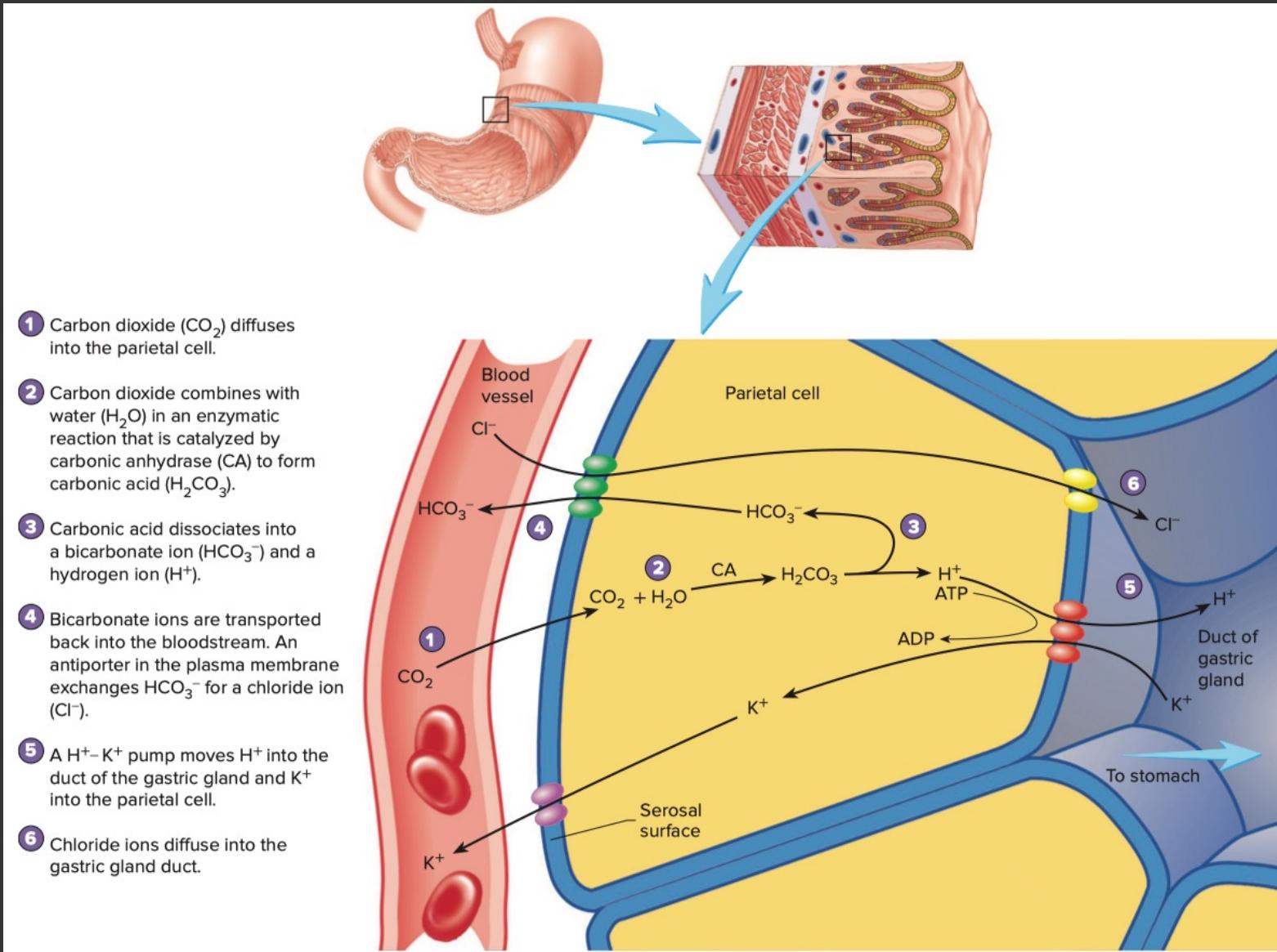
- Pepsin
 - Protease (enzyme that digests protein) released by chief cells
 - Produced in its inactive form (pepsinogen)
 - Activated when pH is below 3.0
- Gastric Lipase
 - Released by chief cells
 - Digests triglycerides in emulsified fats
 - Minor role of lipid digestion in adults
- Intrinsic Factor
 - Released by parietal cells
 - Necessary for proper vitamin B₁₂ absorption in the small intestine
- Mucous
 - Protects stomach from gastric acid and enzymes

Stomach - Secretions

- Gastric Acid
 - Released by parietal cells
 - Formation of gastric acid
 - H⁺ / K⁺ Pump (proton pump) transports H⁺ into lumen
 - Decreases gastric pH to as low as 1.0
 - Inhibited by proton pump inhibitors (eg , Prilosec , Nexium)
- Functions
 - Decreases pH to activate pepsin
 - Breaks down connective tissue in meat
 - Kills bacteria



Formation of Gastric Acid



PROCESS FIGURE 24.12 Hydrochloric Acid Production by Parietal Cells in the Gastric Glands of the Stomach

A series of steps involving carbonic anhydrase, a proton pump, and a HCO₃⁻-Cl⁻ antiporter produce HCl in the gastric gland.

Regulation of Stomach Secretions

- Parasympathetics
 - Stimulates release of gastric acid
 - Stimulates gastrin and histamine
- Gastrin
 - Stimulates release of gastric acid
- Histamine
 - Stimulates release of gastric acid
 - Blocked by H₂ antagonists
 - eg , Tagamet
 - eg , Pepcid
- Somatostatin
 - Inhibits release of gastric acid
- Secretin
 - Inhibits gastrin (therefore , indirectly inhibits gastric acid)

Three Phases of Gastric Acid Secretion

- Cephalic Phase
- Gastric Phase
- Intestinal Phase

Gastric Acid Secretion - Cephalic Phase

- Taste , smell , or thought of food stimulates medulla oblongata
 - Stimulates parasympathetics
 - Stimulates gastric acid , gastrin , and histamine
 - Gastrin and histamine stimulate gastric acid
- Accounts for 30% of gastric acid secretion

Gastric Acid Secretion - Cephalic Phase

Taste , smell or thought of food stimulates medulla oblongata



Parasympathetics



gastric acid , gastrin , and histamine

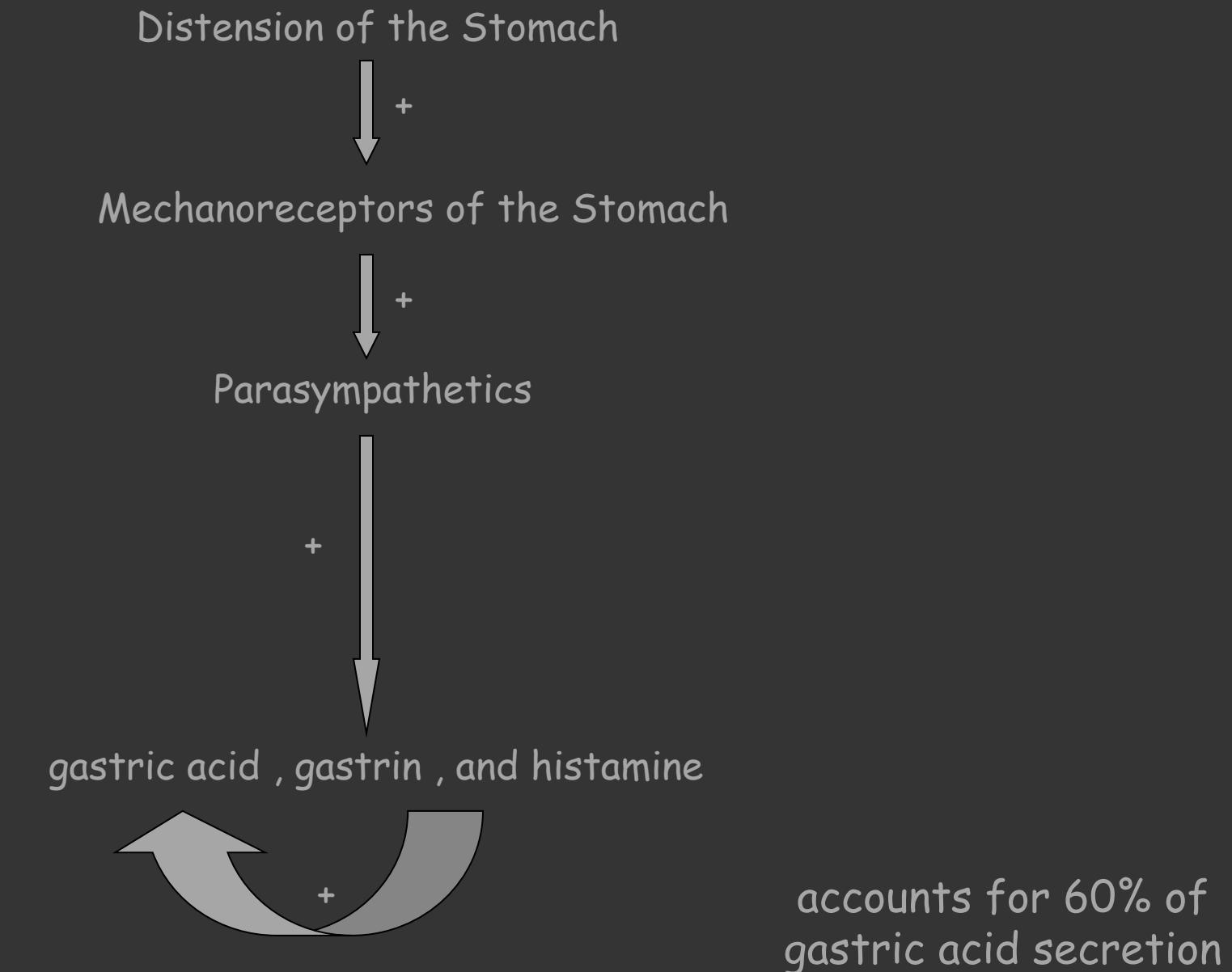


accounts for 30% of
gastric acid secretion

Gastric Acid Secretion - Gastric Phase

- Distension of the stomach stimulates mechanoreceptors
 - Mechanoreceptors stimulate parasympathetics
 - Stimulates gastric acid , gastrin , and histamine
 - Gastrin and histamine stimulate gastric acid
- Accounts for 60% of gastric acid secretion

Gastric Acid Secretion - Gastric Phase



Gastric Acid Secretion - Intestinal Phase

- Chyme first emptied by the stomach
 - Stimulates duodenum to release gastrin
 - Stimulates the release of gastric acid
- Accounts for 10% of gastric acid secretion

Gastric Acid Secretion - Intestinal Phase

Chyme first emptied by stomach



+

Release of gastrin



+

Release of gastric acid

accounts for 10% of gastric acid secretion

Small Intestine

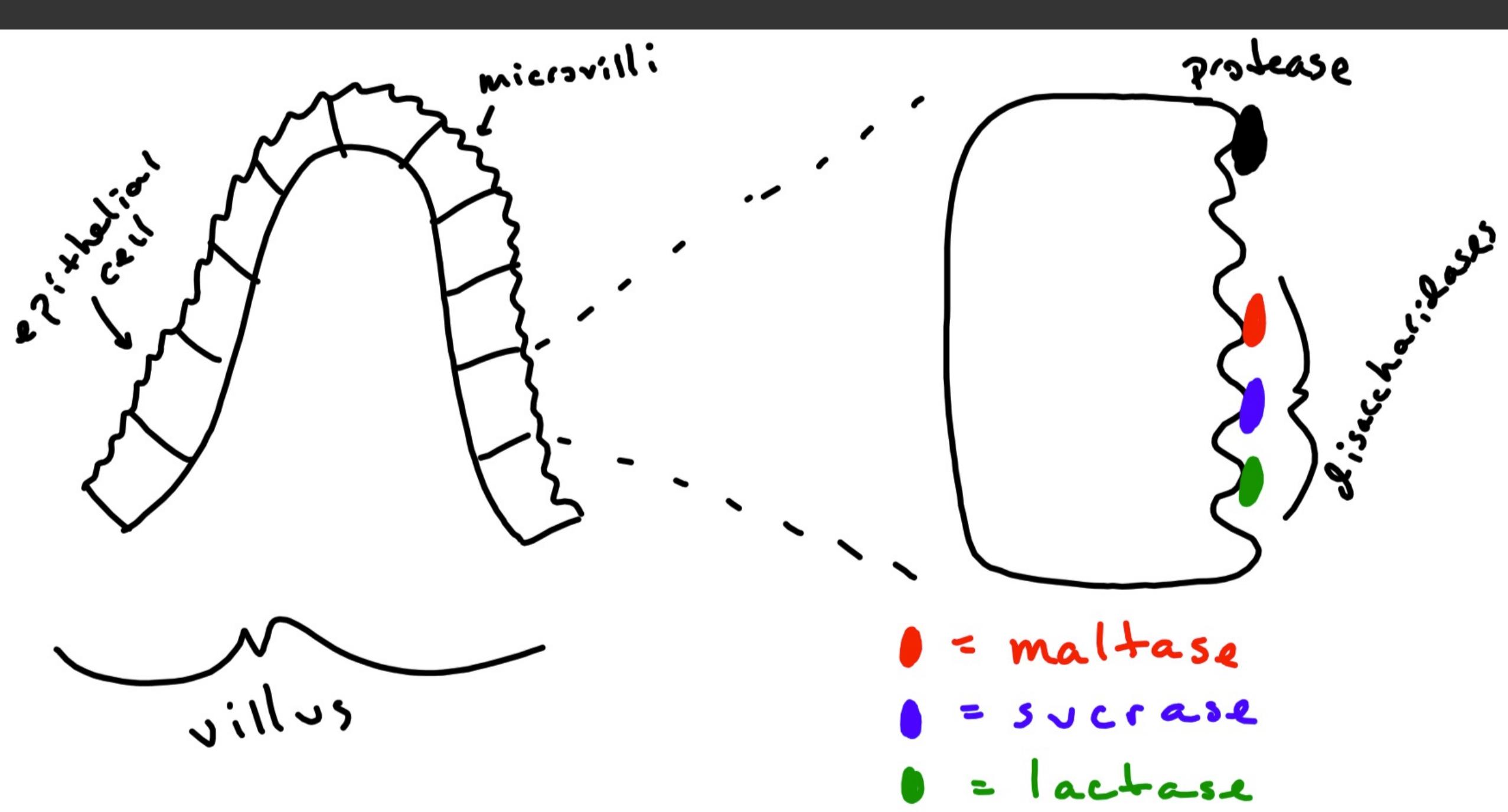
- Most digestion occurs in the small intestine
 - Lining of small intestine greatly increases surface area for digestion
- Contains various surface-bound enzymes
 - Disaccharidases (digests disaccharides)
 - Proteases (digests protein)

Small Intestine

- Lysosomal Enzymes
 - Released by intestinal glands
 - Glands that extend down between villi into the mucosa
 - Help maintain fluidity of chyme
- Mucous
 - Protects lining from acidic chyme and digestive enzymes

Small Intestine - Produced Enzymes

- Disaccharidases for the final digestion of carbohydrates
 - Surface-bound to microvilli
 - Maltase
 - Digests maltose into glucose + glucose
 - Sucrase
 - Digests sucrose into glucose + fructose
 - Lactase
 - Digests lactose into glucose + galactose
- Proteases for the digestion of protein
 - Surface-bound to microvilli



Large Intestine

- Flora digest approximately 10% of soluble fiber
 - Produces short-chain fatty acids (acetate , propionate , butyrate)
 - Have anti-inflammatory properties

Accessory Organs

- Teeth
- Tongue
- Salivary Glands
- Liver
- Gall Bladder
- Pancreas

Liver and Gallbladder

- The liver produces bile
 - Approximately 600 to 1,000 mL per day
 - Comprised of :
 - Electrolytes (namely HCO_3^- to neutralize the acidic chyme)
 - Bile Acids
 - Combine with amino acids to become bile salts
- Functions of Bile
 - Neutralize acidic chyme via HCO_3^-
 - Protects the duodenum from acidic chyme
 - Necessary for proper enzyme function
 - HCO_3^- release is stimulated by secretin
 - Emulsify lipids via bile salts

Liver and Gallbladder

- Bile release from the liver to the gallbladder
- Bile is stored , concentrated , and released by the gallbladder
 - Cholecystokinin causes gallbladder to contract
 - Causes release of bile into duodenum
 - In response to acid and lipids in small intestine

Other Functions of the Liver

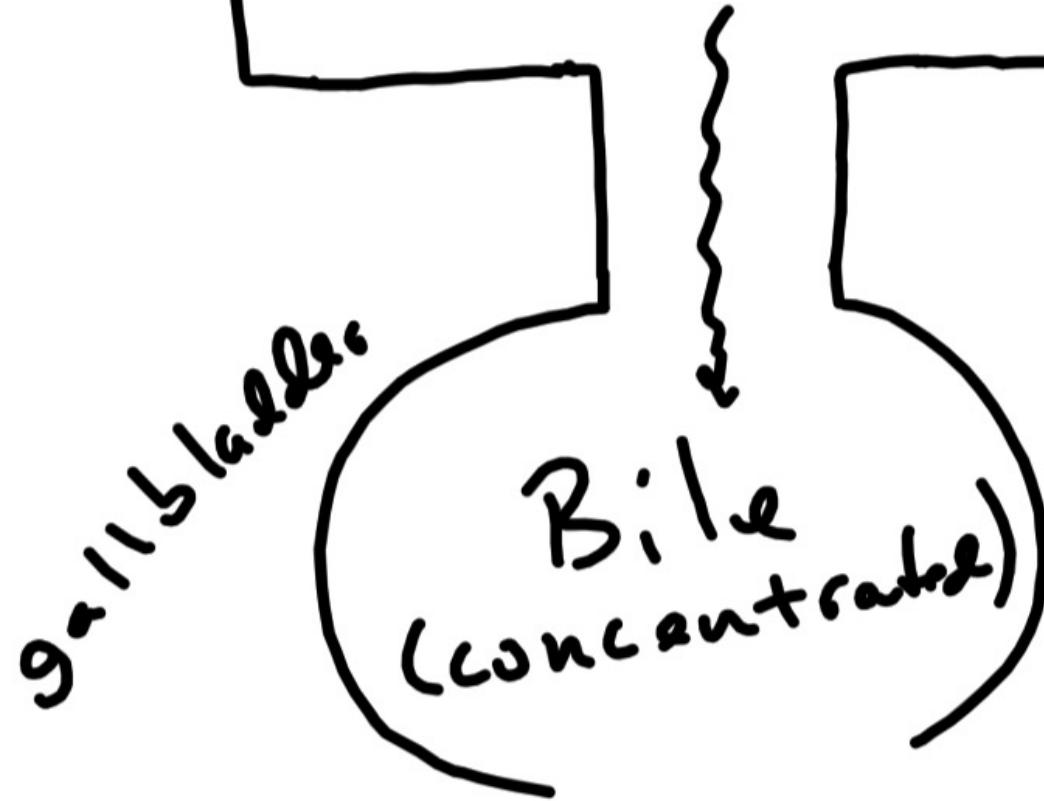
- **Glycogenesis**
 - Converts glucose into glycogen for storage
- **Glycogenolysis**
 - Breaks down glycogen into glucose for use
- **Gluconeogenesis**
 - Production of glucose from non-carbohydrate sources
 - Amino acids , lactate , glycerol , and pyruvate
- **Lipogenesis**
 - Production of lipids from glucose and amino acids
- Stores glycogen , iron (in the form of ferritin) and vitamins A , D , and B₁₂
- Removes nutrients from blood
- Adds nutrients to blood
- Produces cholesterol and many plasma proteins
- Detoxifies alcohol and many drugs and toxins that enter the body
- Phagocytizes bacteria and worn out red blood cells
- Deaminates amino acids (ammonia produced as a consequence)
- Converts ammonia into urea

Liver

- * Bile

- bile acids / salts

- HCO_3^-



- * bile acids / salts

- * bile acids / salts
fat

- ↳ emulsify
(digest)

- * HCO_3^-
↳ neutralize
acid

Pancreas

- Pancreatic secretions released into the duodenum
- HCO_3^- neutralizes acidic chyme
 - Protects duodenum from acidic chyme
 - Necessary for proper enzyme function
 - Release stimulated by secretin
 - CCK potentiates effects

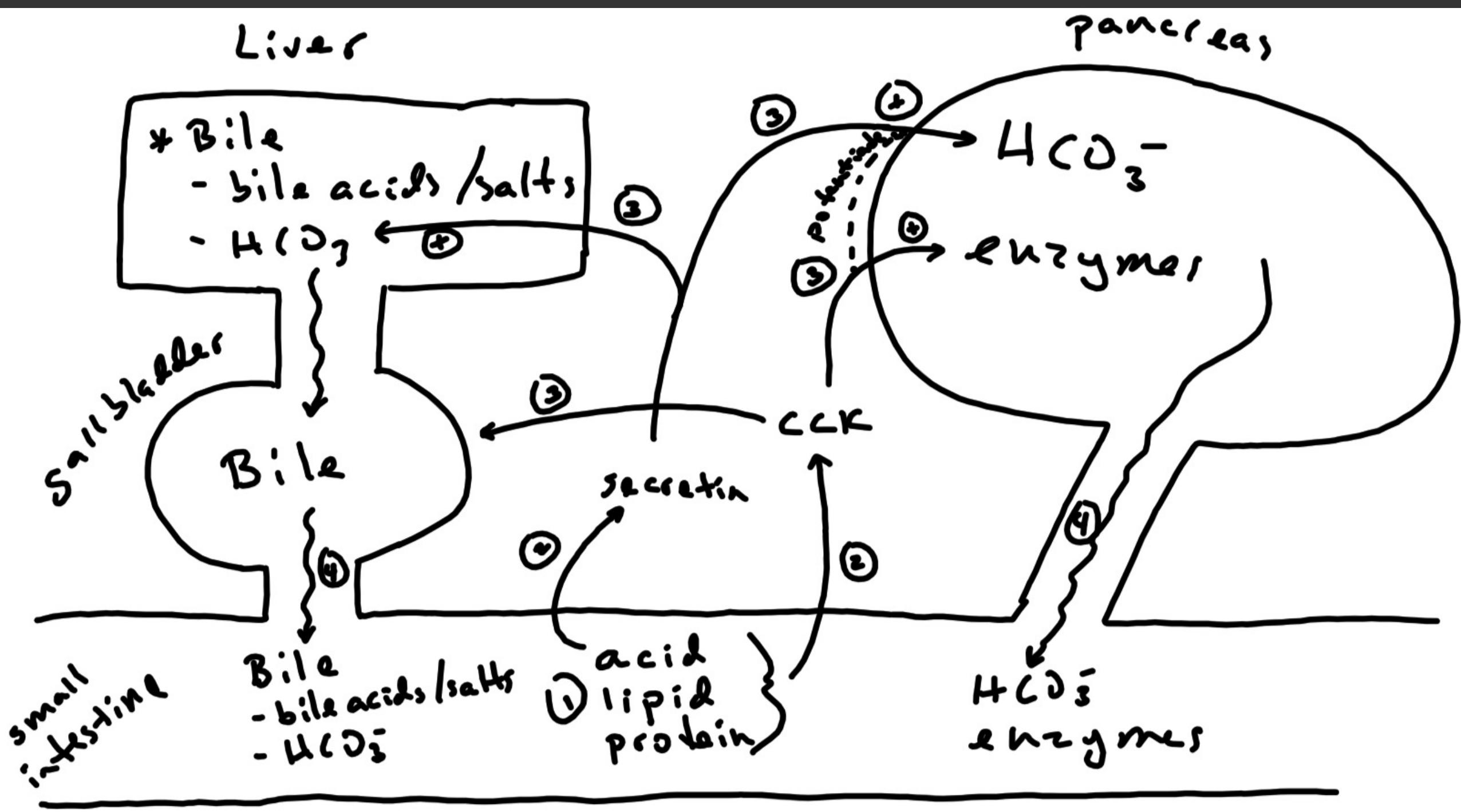
Pancreas - Organic Secretions

- Pancreatic Amylase
 - Continues digestion of starch into disaccharides
- Pancreatic Lipase
 - Digestion of lipid
 - Digests triglycerides into fatty acids and monoglycerides
- Cholesterol Esterase
 - Digests cholesterol esters into cholesterol and fatty acids
- Proteases
 - Digest protein into polypeptides , tripeptides , dipeptides , and amino acids
- Pancreatic Secretions Release stimulated by cholecystokinin

pancreas

HCO₃⁻

pancreatic amylase
pancreatic lipase
cholesterol esterase
pancreatic proteases



Summary of Carbohydrate Digestion

- Mouth
 - Salivary Amylase
 - Polysaccharide (starch) into disaccharide (maltose)
- Small Intestine
 - Pancreatic Amylase
 - Polysaccharide (starch) into disaccharide (maltose)
 - Maltase surface-bound to microvilli
 - Disaccharide (maltose) into monosaccharide (glucose)
 - Sucrase surface-bound to microvilli
 - Disaccharide (sucrose) into monosaccharides (glucose + fructose)
 - Lactase surface-bound to microvilli
 - Disaccharide (lactose) into monosaccharides (glucose + galactose)
 - Lactose intolerance = lack of lactase
 - Cannot digest lactose
 - Can lead to hypertonic chyme
 - Can lead to osmotic diarrhea
 - Flora feed off of lactose and produce gas

\circ = glucose

\square = fructose

Δ = galactose

Starch



↓ salivary amylase (mouth)

↓ pancreatic amylase (small intestine)



↓ maltase (small intestine)



maltose



↓ maltase (small intestine)



sucrose



↓ sucrase (small intestine)



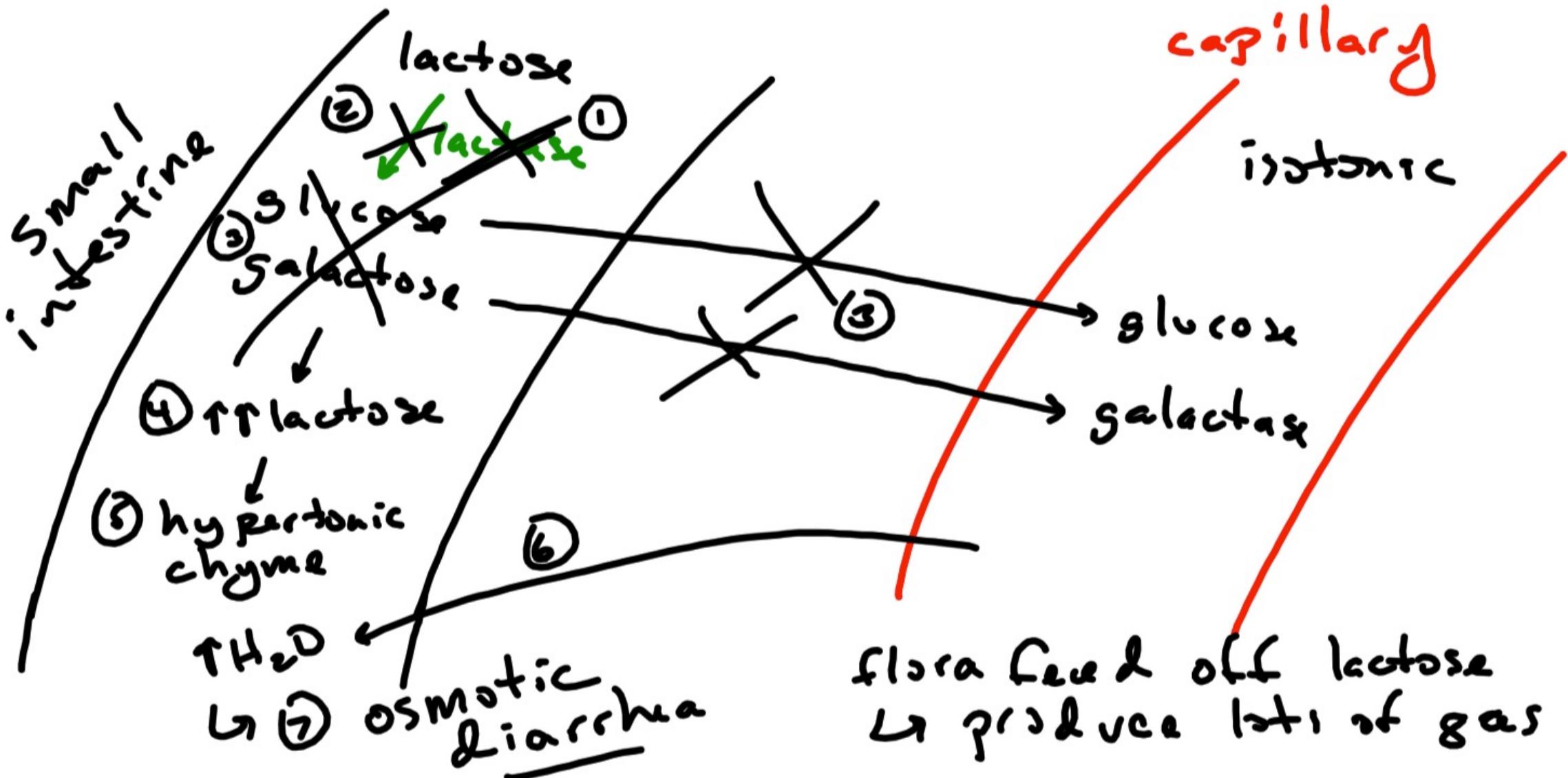
lactose



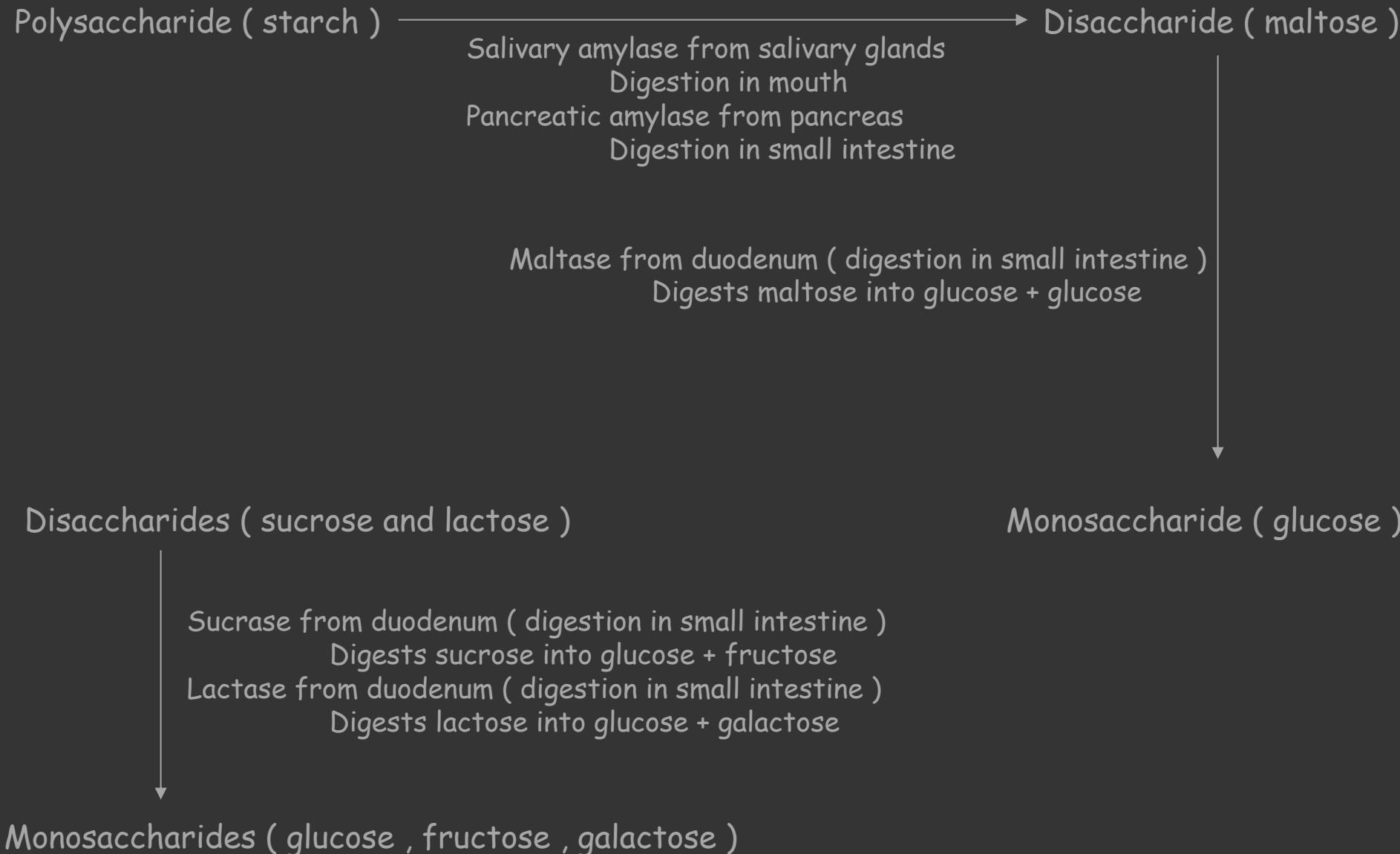
↓ lactase
small intestine



Lactose Intolerance



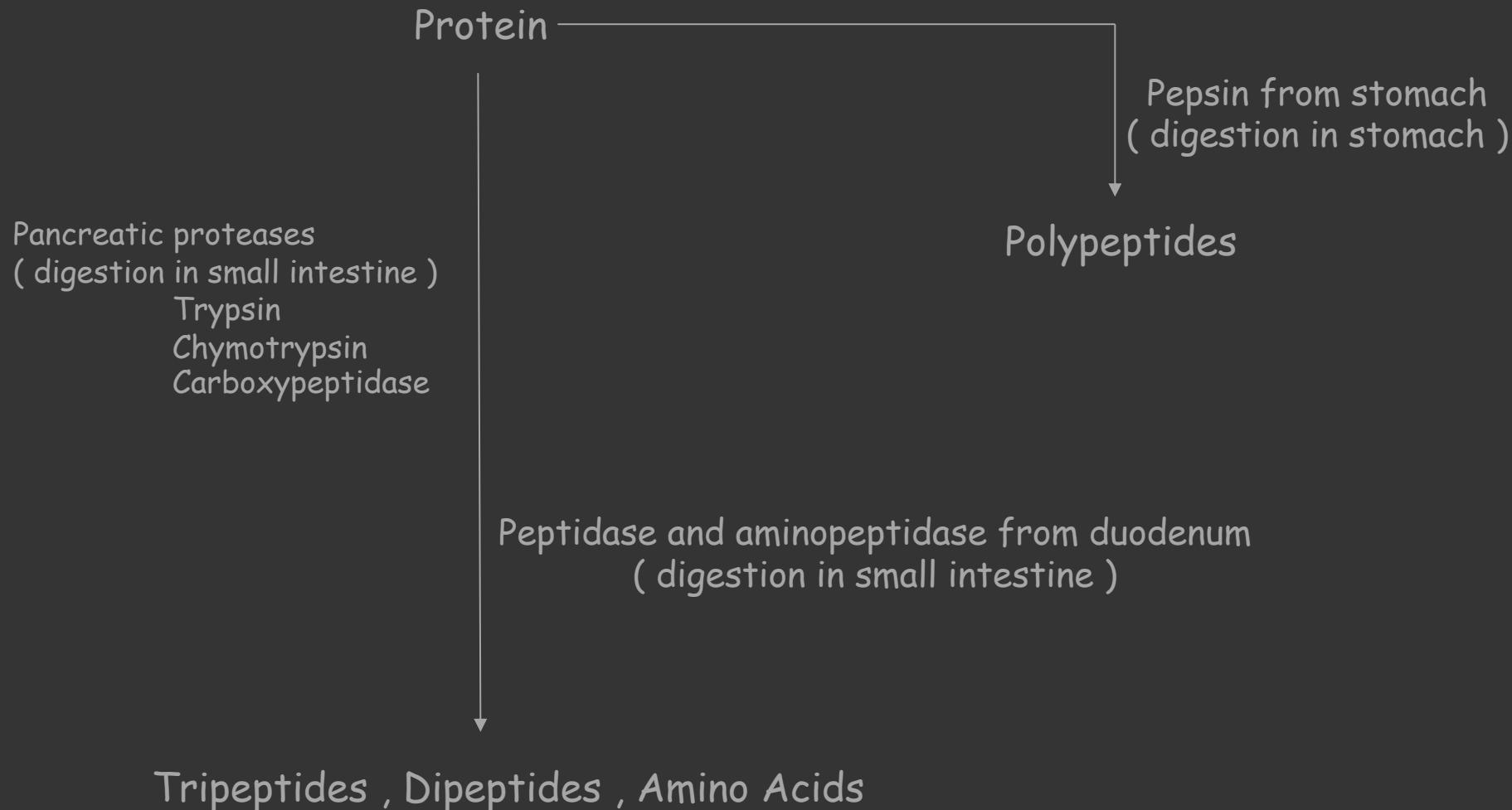
Carbohydrate Digestion



Summary of Protein Digestion

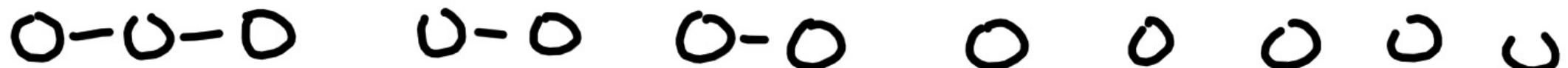
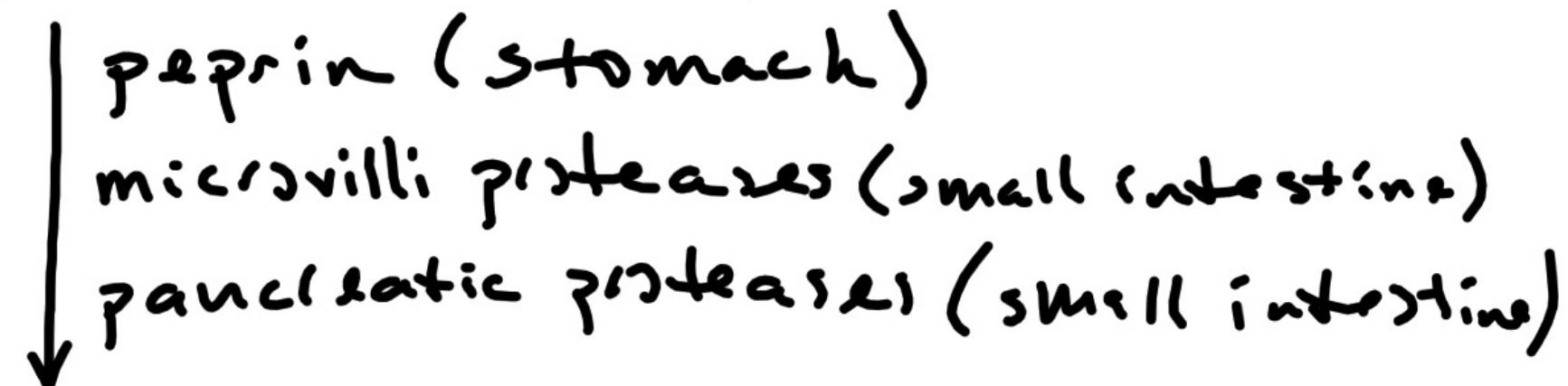
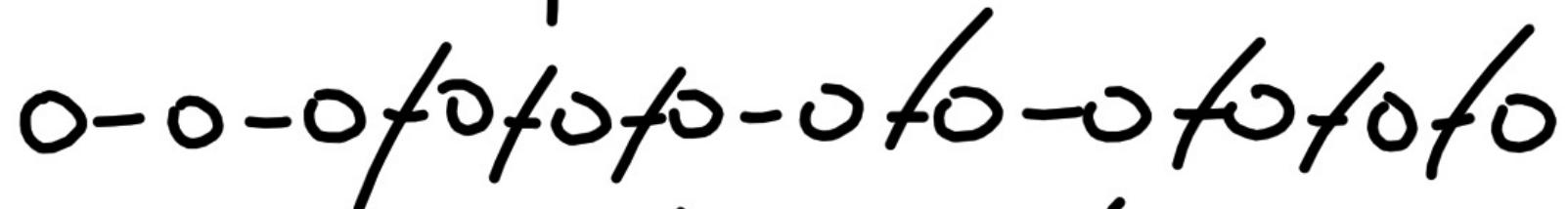
- Stomach
 - Pepsin
 - Proteins into polypeptides
- Small Intestine
 - Proteases surface-bound to microvilli
 - Proteins into tripeptides , dipeptides , and amino acids
 - Pancreatic Proteases
 - Proteins into polypeptides , tripeptides , dipeptides , and amino acids

Protein Digestion



O = amino acid

protein

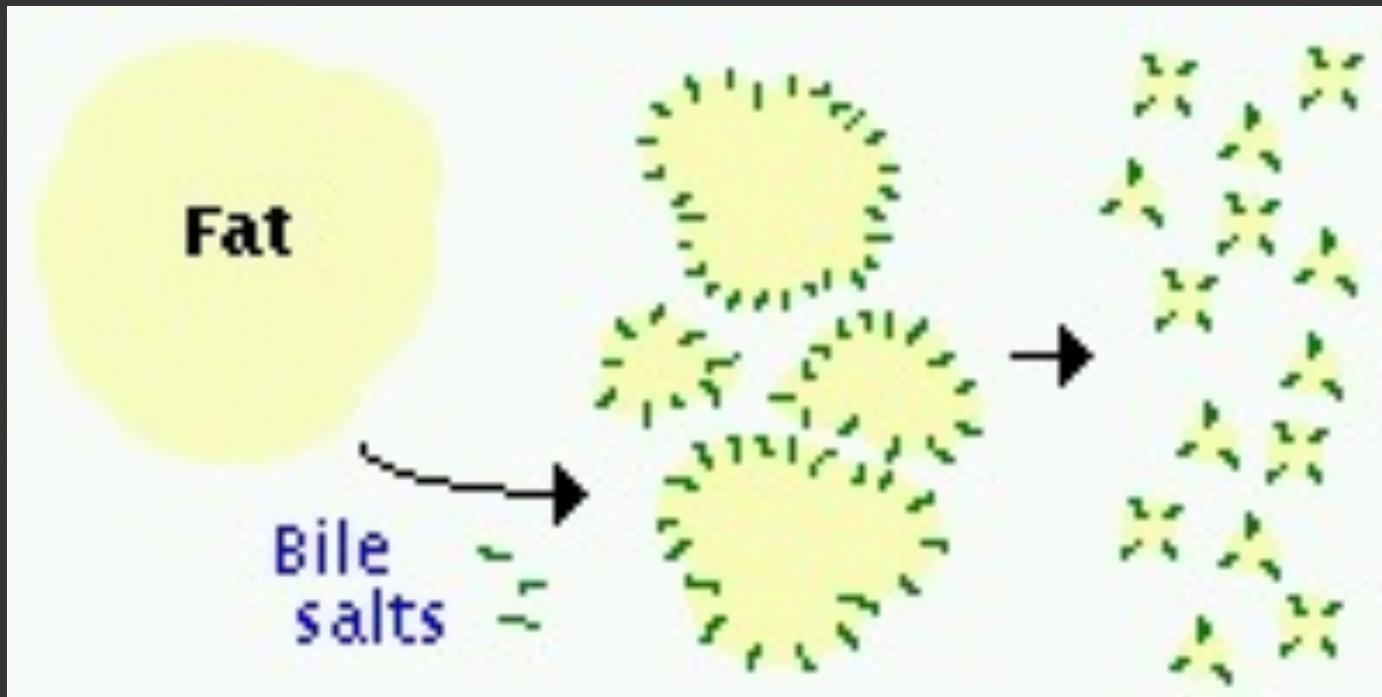


tripeptides dipeptides amino acids

Summary of Fat / Lipid Digestion

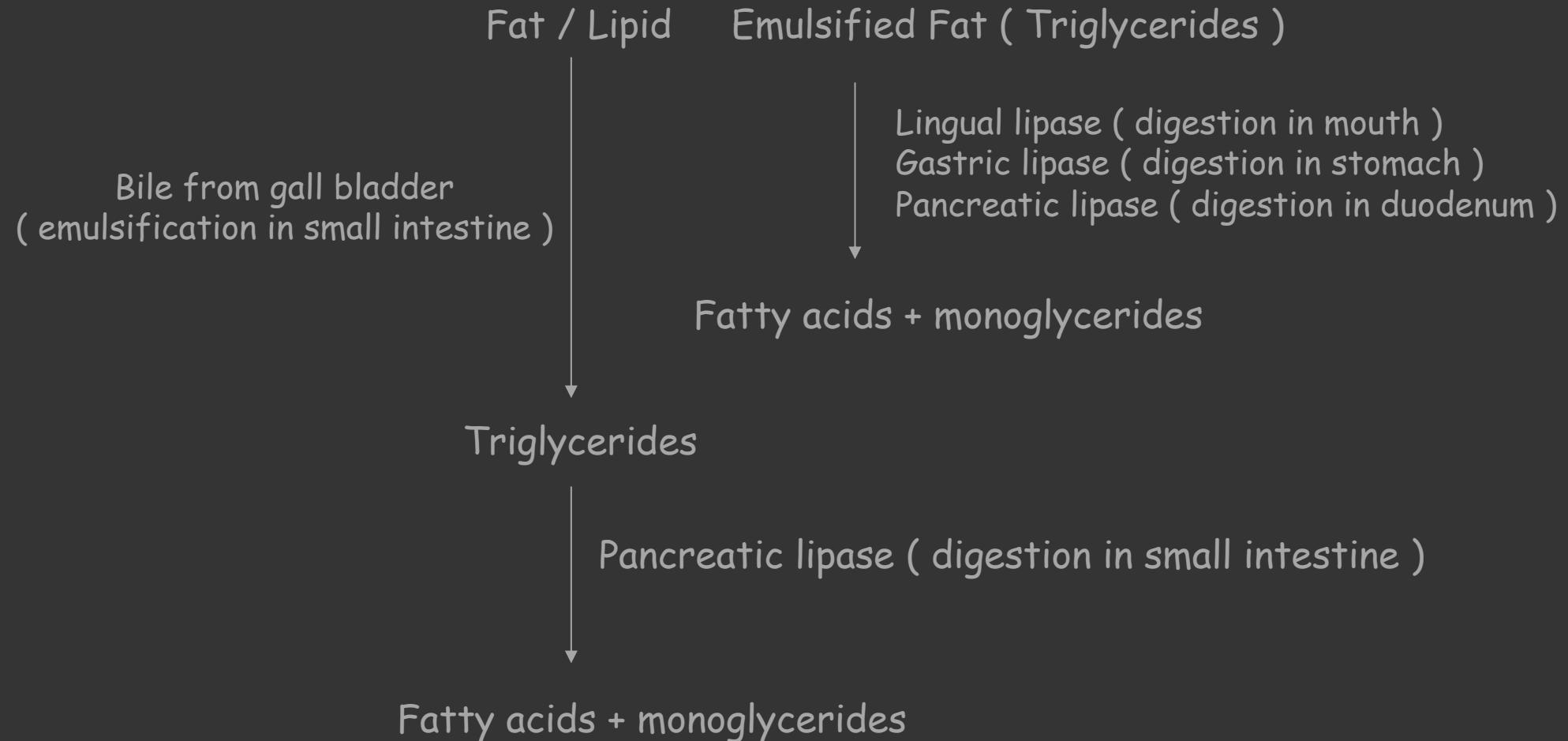
- Mouth
 - Lingual Lipase
 - Emulsified lipid (ie , triglycerides) into monoglycerides and fatty acids
- Stomach
 - Gastric Lipase
 - Emulsified lipid (ie , triglycerides) into monoglycerides and fatty acids
- Small Intestine
 - Bile
 - Lipids into emulsified lipid (ie , triglycerides)
 - Pancreatic Lipase
 - Emulsified lipid (ie , triglycerides) into fatty acids + monoglycerides
 - Cholesterol Esterase
 - Cholesterol ester into cholesterol + fatty acids

Emulsification



Bile salts surround dietary lipid / fat to form small , lipid / fat droplets (triglycerides)

Fat Digestion



+ triglyceride



lingual lipase (mouth)

gastric lipase (stomach)

↓ pancreatic lipase (small intestine)



monoglyceride

free fatty acids

cholesterol ester

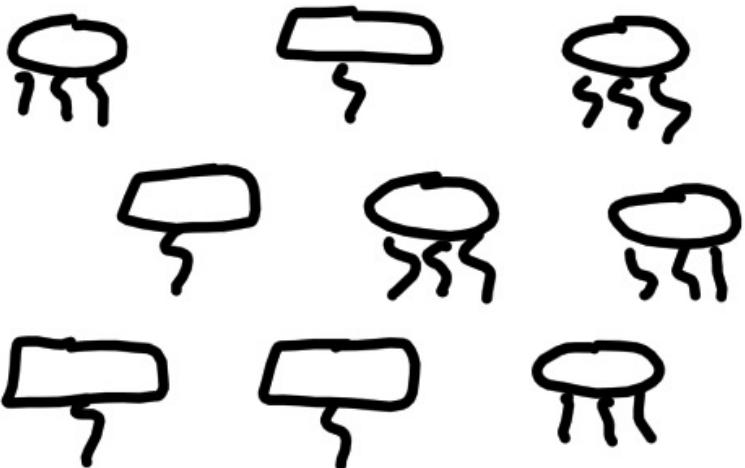
cholesterol

↓ cholesterol esterase (small intestine)

cholesterol

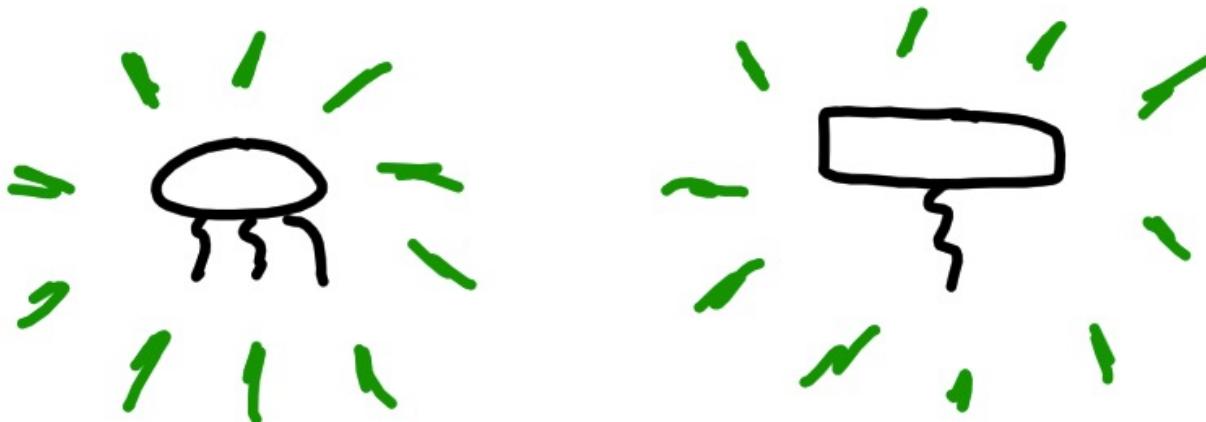
}

Animal Fat



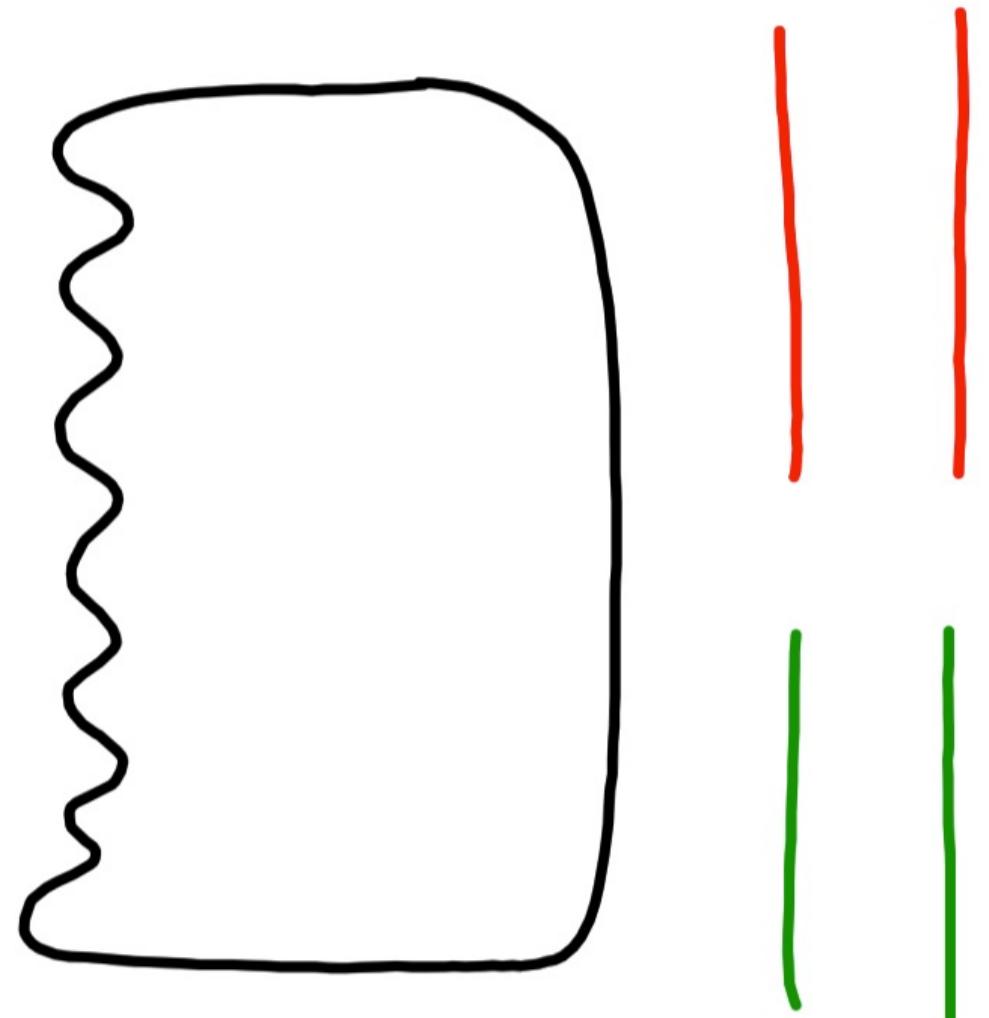
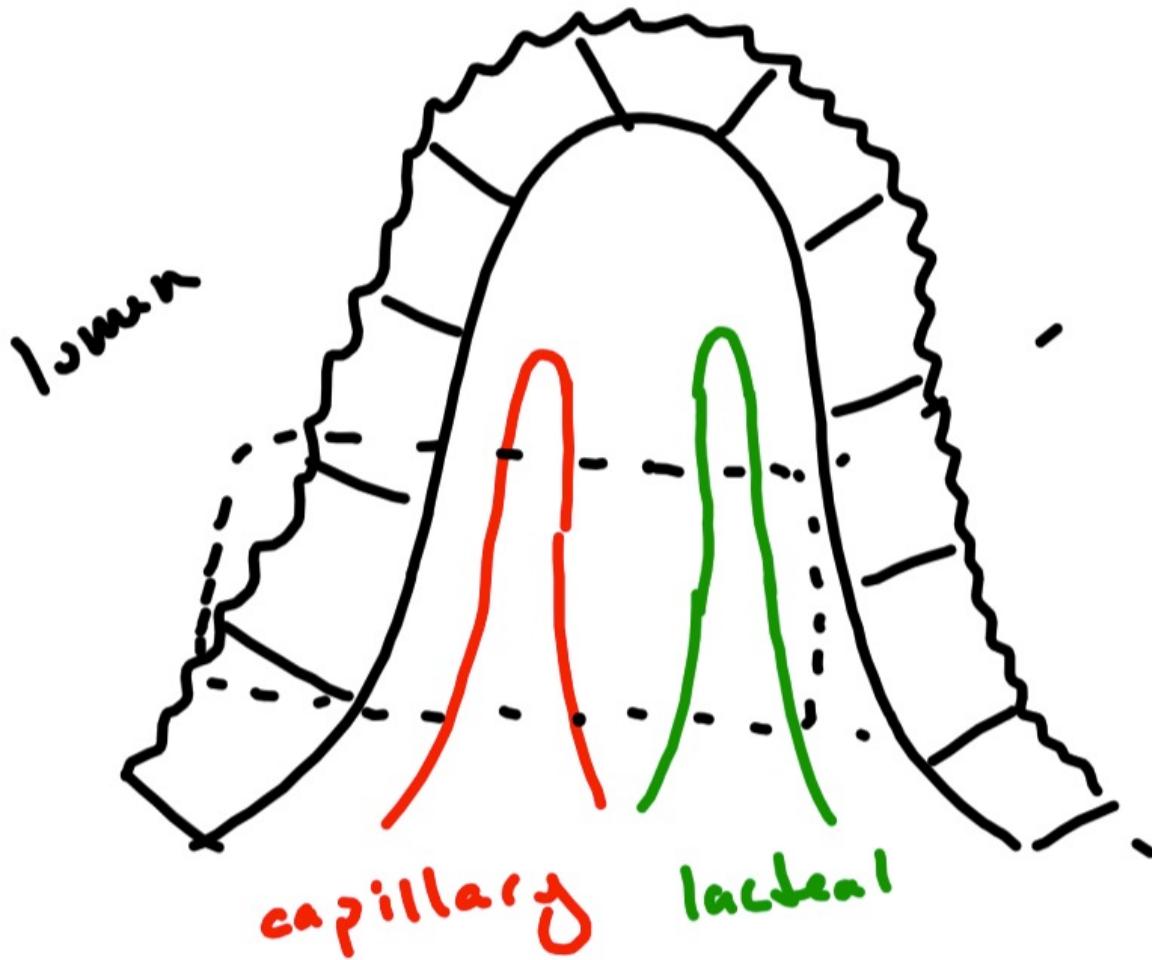
bile acid/salt
/

emulsify | bile acids/salts (small intestine)



Absorption of Nutrients

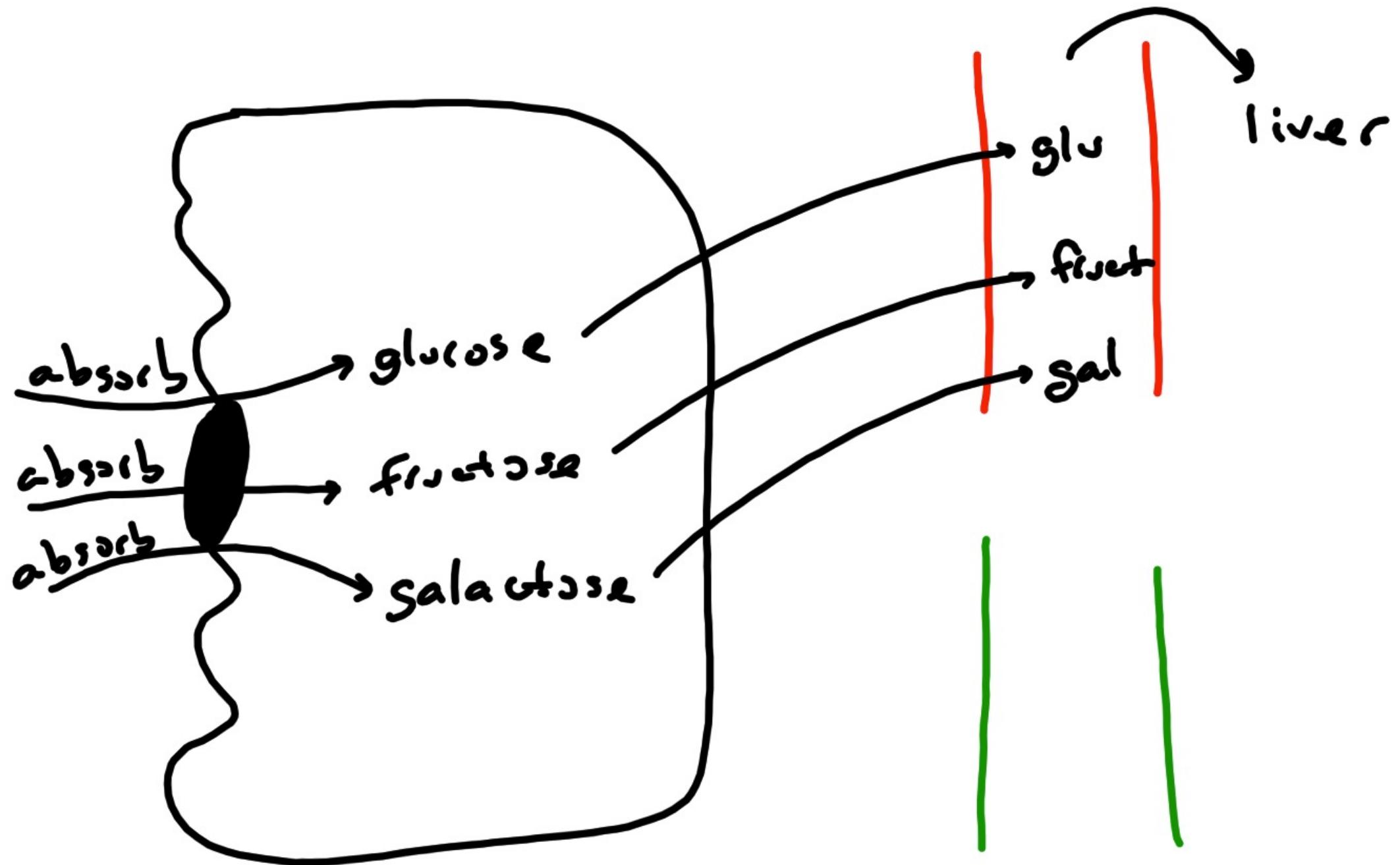
- Stomach
 - Very little absorption takes place in the stomach
 - Some water and electrolytes as well as lipid and alcohol absorbed
- Small Intestine
 - Most absorption occurs in the small intestine through small intestine epithelial cells
- Large Intestine
 - Approximately 1.5 liters of chyme enters the large intestine per day
 - 90% of that volume is absorbed as water and electrolytes
 - Absorption of vitamin K and biotin (vitamin B7) and short-chain fatty acids



Absorption of Monosaccharides

- Monosaccharides from carbohydrate digestion
 - Absorbed by epithelial cells of villi via secondary active transport
 - Enter blood capillaries via facilitated diffusion
 - Enter the hepatic portal system and transported to the liver

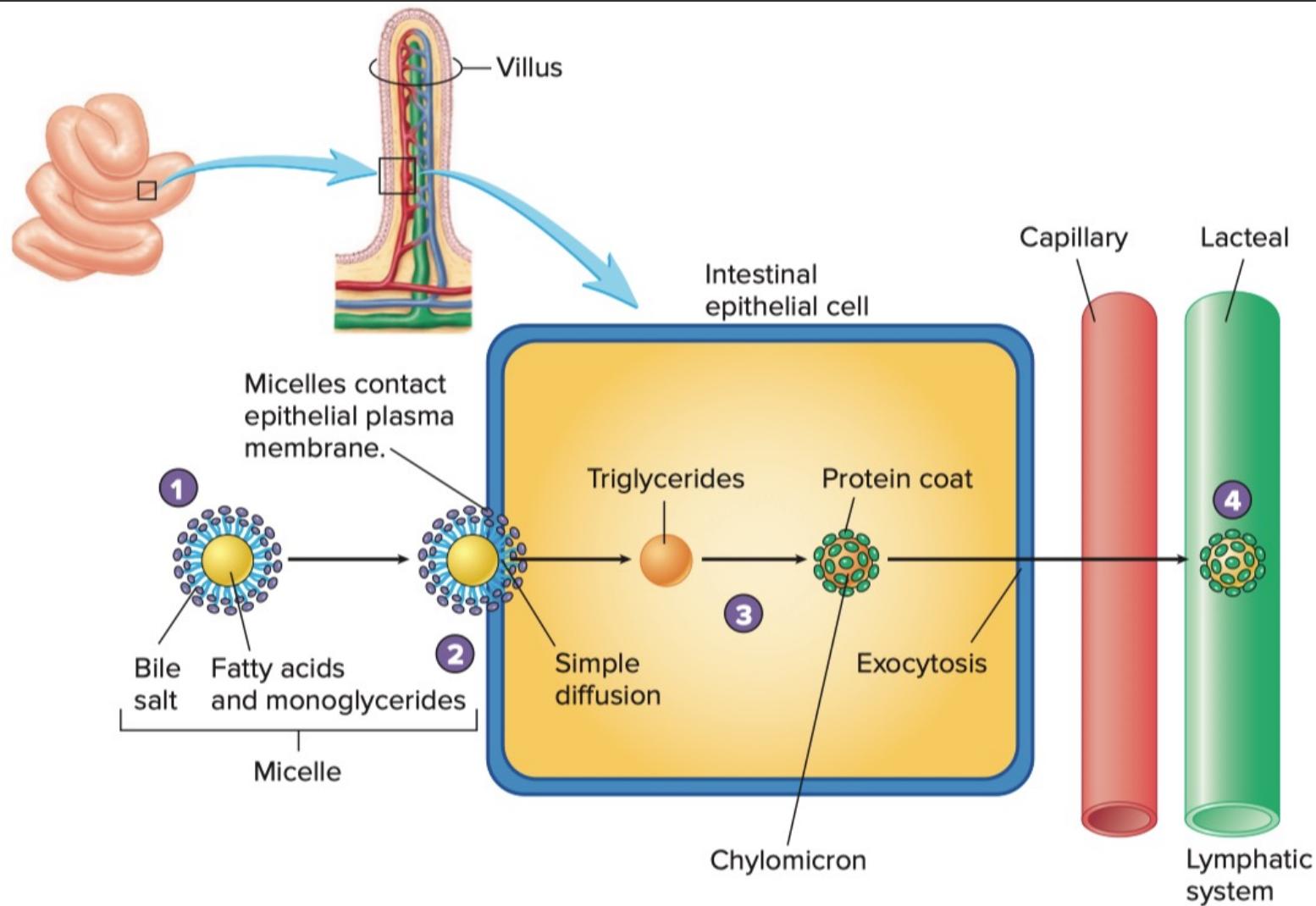
tumor



Absorption of Monosaccharides

Lipid transport

- 1 Bile salts surround fatty acids and monoglycerides to form micelles.
- 2 Micelles attach to the plasma membranes of intestinal epithelial cells, and the fatty acids and monoglycerides pass by simple diffusion into the intestinal epithelial cells.
- 3 Within the intestinal epithelial cell, the fatty acids and monoglycerides are converted to triglycerides; proteins coat the triglycerides to form chylomicrons, which move out of the intestinal epithelial cells by exocytosis.
- 4 The chylomicrons enter the lacteals of the intestinal villi and are carried through the lymphatic system to the general circulation.



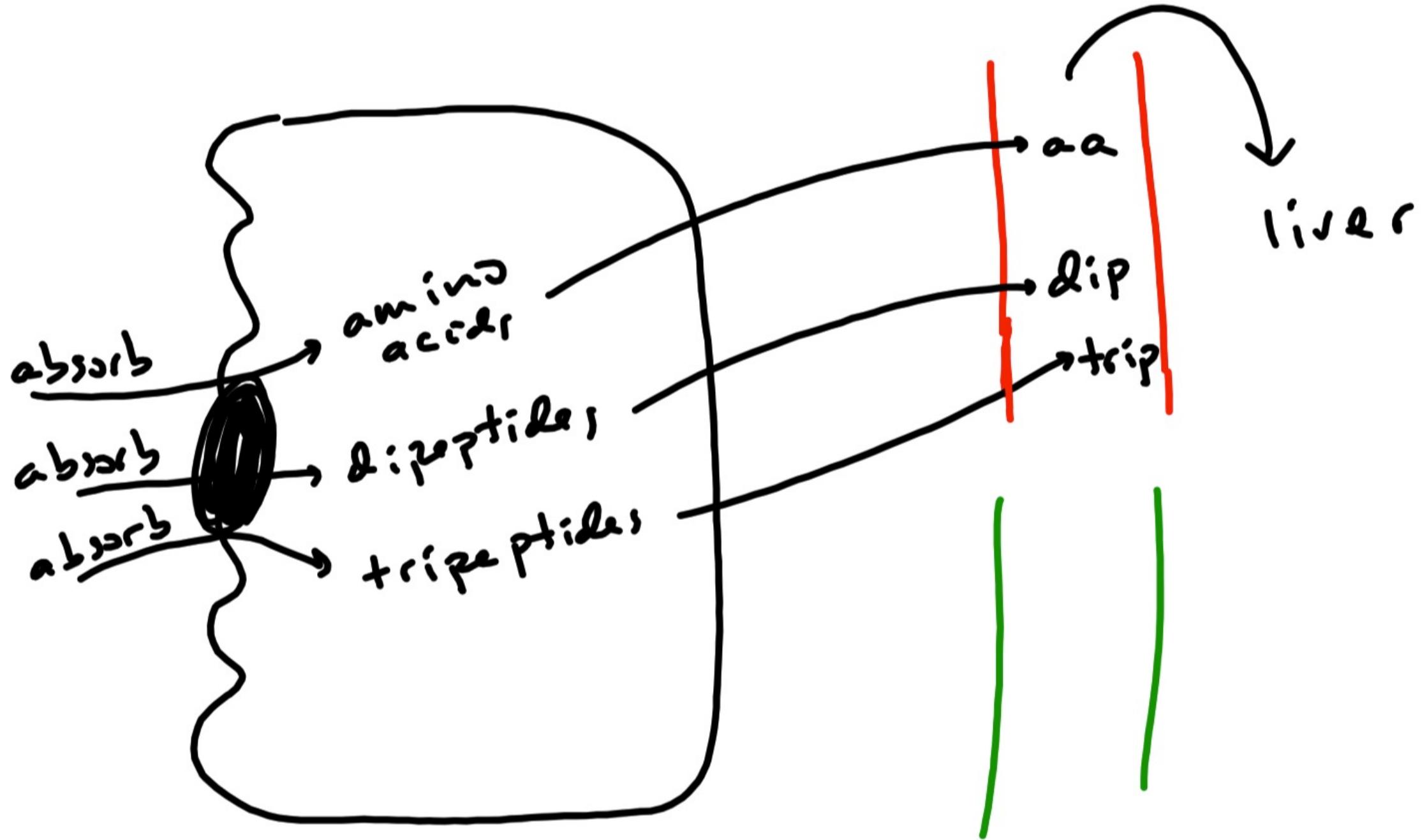
PROCESS FIGURE 24.30 Transport of Lipids Across the Intestinal Epithelium

Lipid absorption occurs when micelles enter intestinal epithelial cells and exit by exocytosis.

Absorption of Amino Acids , Dipeptides , and Tripeptides

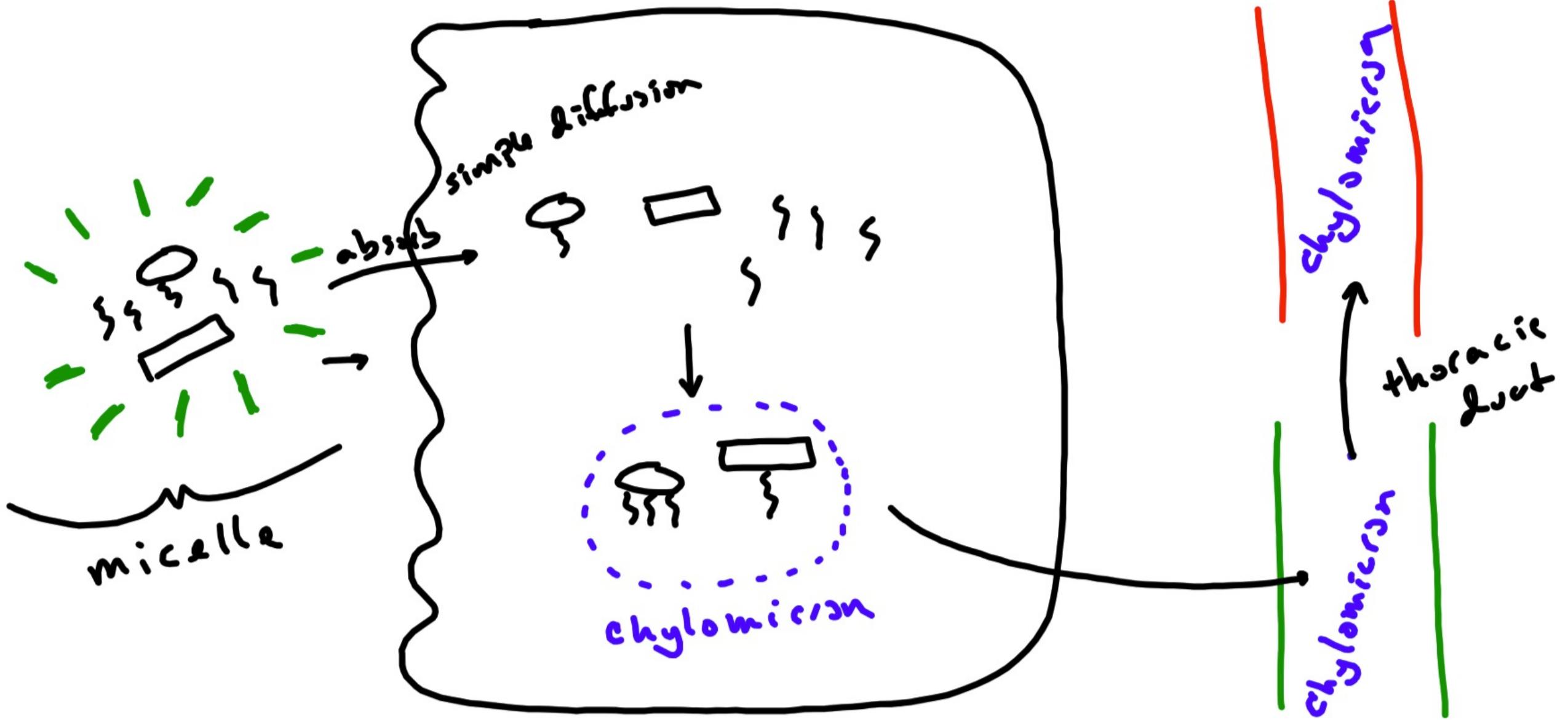
- Amino acids , dipeptides , and tripeptides from protein digestion
 - Absorbed by epithelial cells of villi via secondary active transport
 - Most dipeptides and tripeptides broken down into individual amino acids
 - Amino acids , dipeptides , and tripeptides enter capillaries via facilitated diffusion
 - Enter the hepatic portal system and transported to the liver

lumen



Absorption of Fatty Acids and Monoglycerides

- Surrounded by bile salts in the small intestines to form micelles
- Monoglycerides , fatty acids , and cholesterol absorbed by epithelial cells of villi via simple diffusion
 - Fatty acids and monoglycerides combine to form triglycerides
 - Fatty acids and cholesterol combine to form cholesterol esters
 - Triglycerides and cholesterol esters used to make chylomicrons (lipoprotein)
 - Chylomicrons exit epithelial cells via exocytosis
 - Chylomicrons then endocytosed into lacteals
 - Enter lymphatic system and subsequently carried to the blood



Absorption of Water

- Approximately 9 liters of water enters the digestive tract each day
(~ 99% absorbed)
 - Approximately 5% absorbed in the stomach
 - Approximately 85% absorbed in the small intestine
 - Approximately 10% absorbed in the large intestine
- Movement of water dictated by movement of nutrients
 - As nutrients are absorbed , water follows osmotically

Defining pH

$$\text{pH} = -\log [\text{H}^+]$$

Increase [H⁺] → decrease pH

Decrease [H⁺] → increase pH

pH scale: 0 - 14

pH 7.0 10^{-7} or 0.0000001 moles H⁺ / L

pH 7.2 $10^{-7.2}$ or 0.000000063 moles H⁺ / L

pH 7.4 $10^{-7.4}$ or 0.000000040 moles H⁺ / L

pH 7.6 $10^{-7.6}$ or 0.000000025 moles H⁺ / L

pH 8.0 10^{-8} or 0.00000001 moles H⁺ / L

Body must maintain blood pH within its normal range

Normal arterial pH range: 7.35 to 7.45

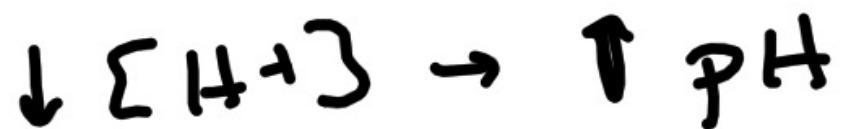
Abnormal: pH < 7.35 (acidosis)

Abnormal: pH > 7.45 (alkalosis)

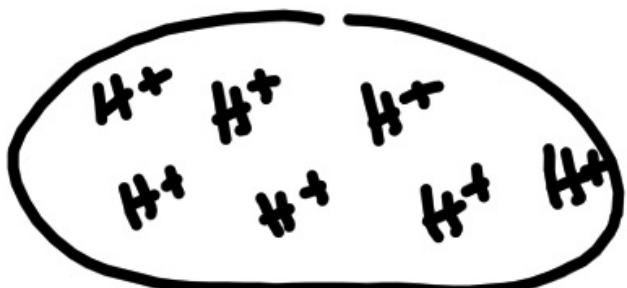
pH changes minimized by pH buffers

pH is regulated (compensated) towards normal by lungs and/or kidneys

* pH is the measure of free $[H^+]$



A



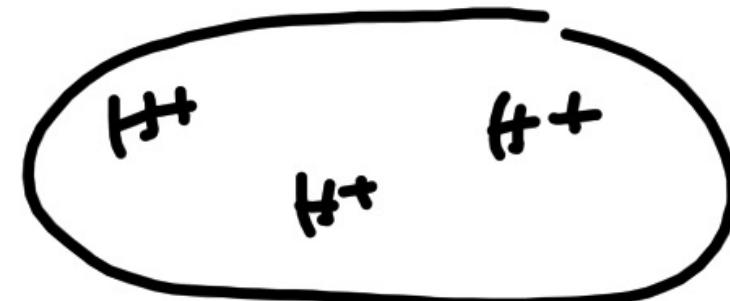
higher $[H^+]$

lower pH

e.g. $pH = 7.02$

vs

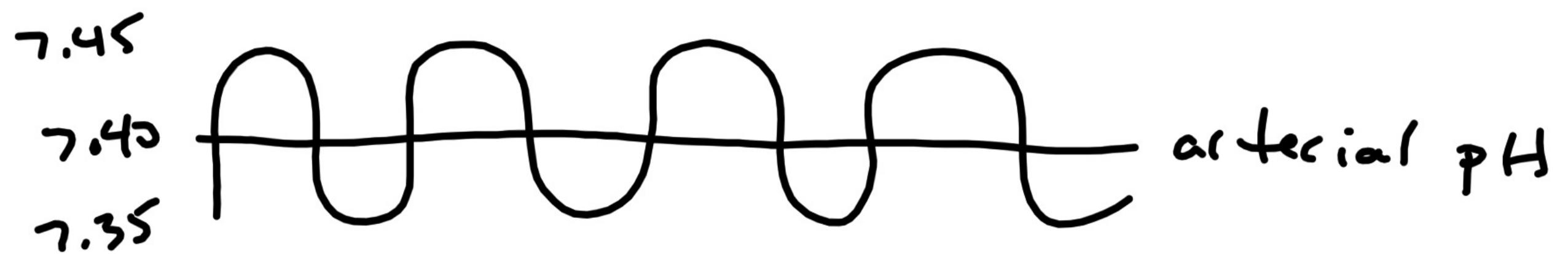
B



lower $[H^+]$

higher pH

e.g. $pH = 7.81$



Control of pH

- pH Buffering Systems
 - pH Regulation
 - Lungs
 - Kidneys

Control of pH

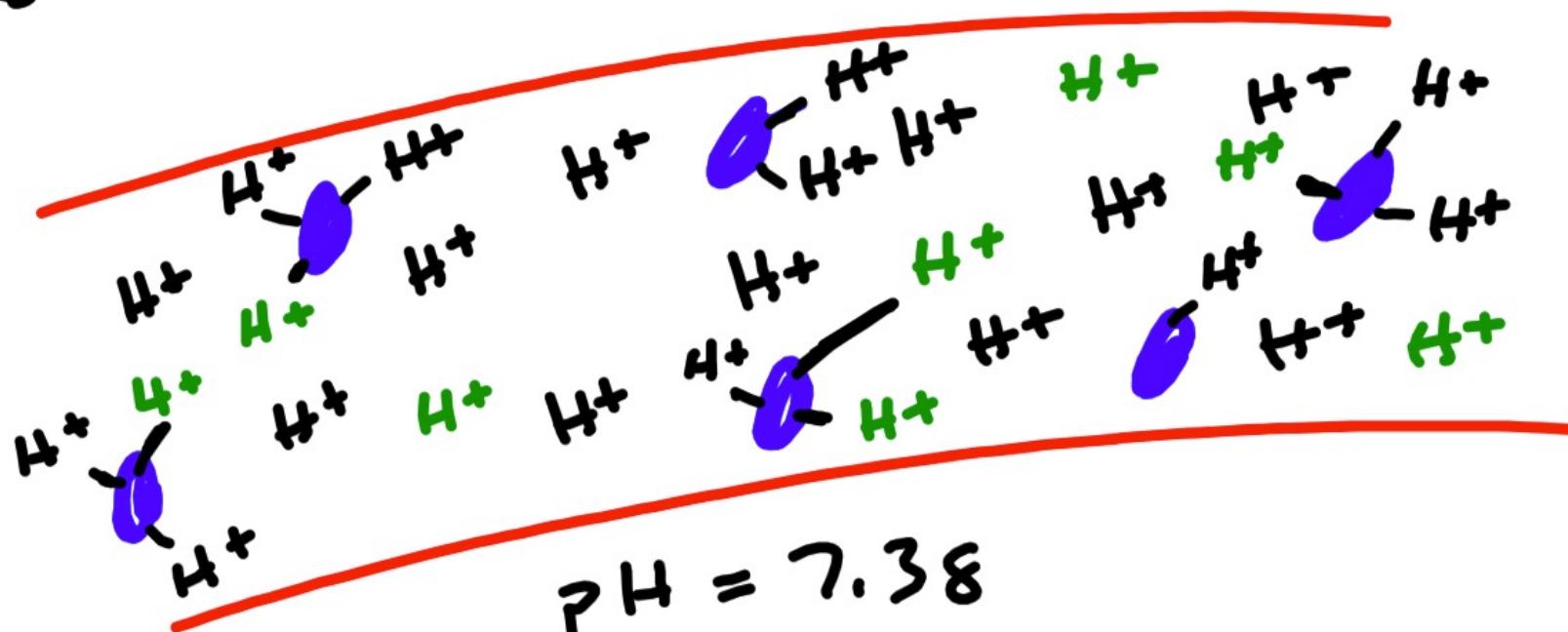
- pH Buffers :
 - Minimize changes in pH with added acid or base
 - Buffers bind H⁺ with added acid
 - Buffers release H⁺ with added base
 - pH buffers **DO NOT** regulate / compensate pH changes
- Types of Buffering Systems :
 - Carbon Dioxide / Bicarbonate Buffer System
 - Strongest buffering system

* What keeps pH within normal range?

↳ 2 - things:

① pH buffering

② pH regulation (via lungs & kidneys)



• = buffer

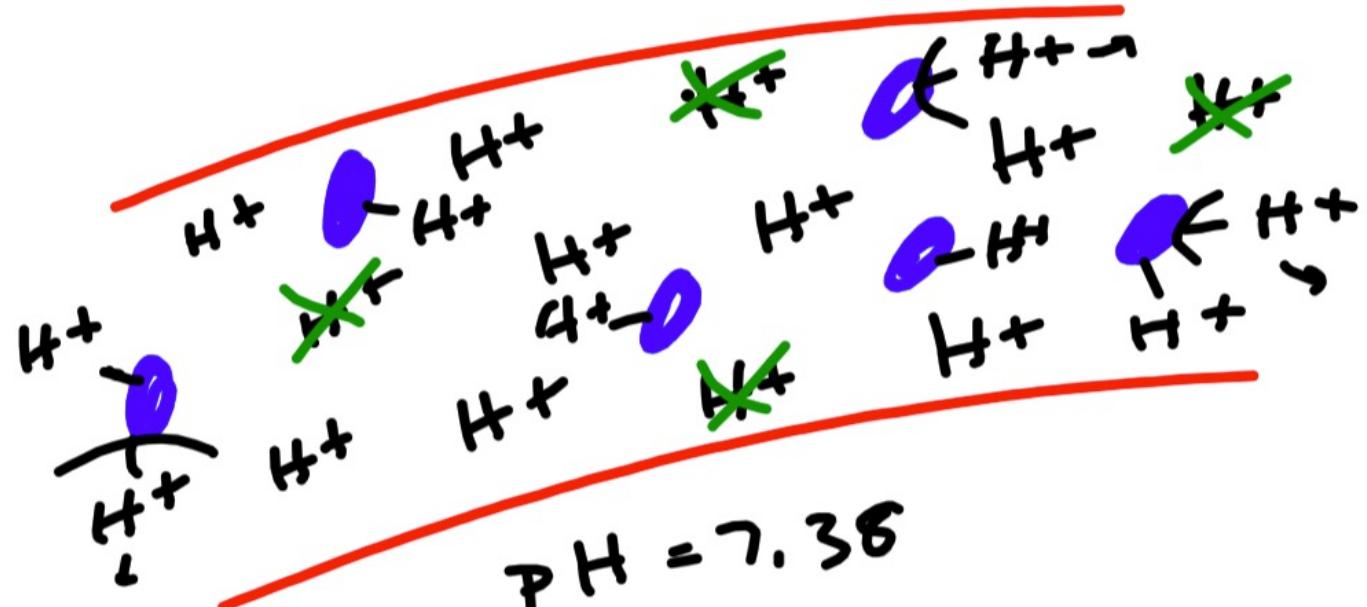
$$pH = 7.38$$

↳ add acid

↳ w/o buffers: 7.04

↳ w. buffers: 7.35

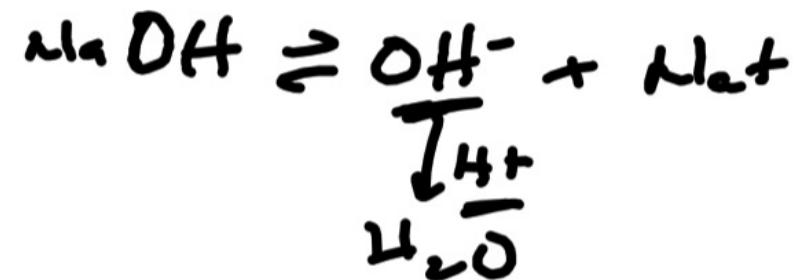
base \rightarrow $\downarrow [\text{H}^+]$

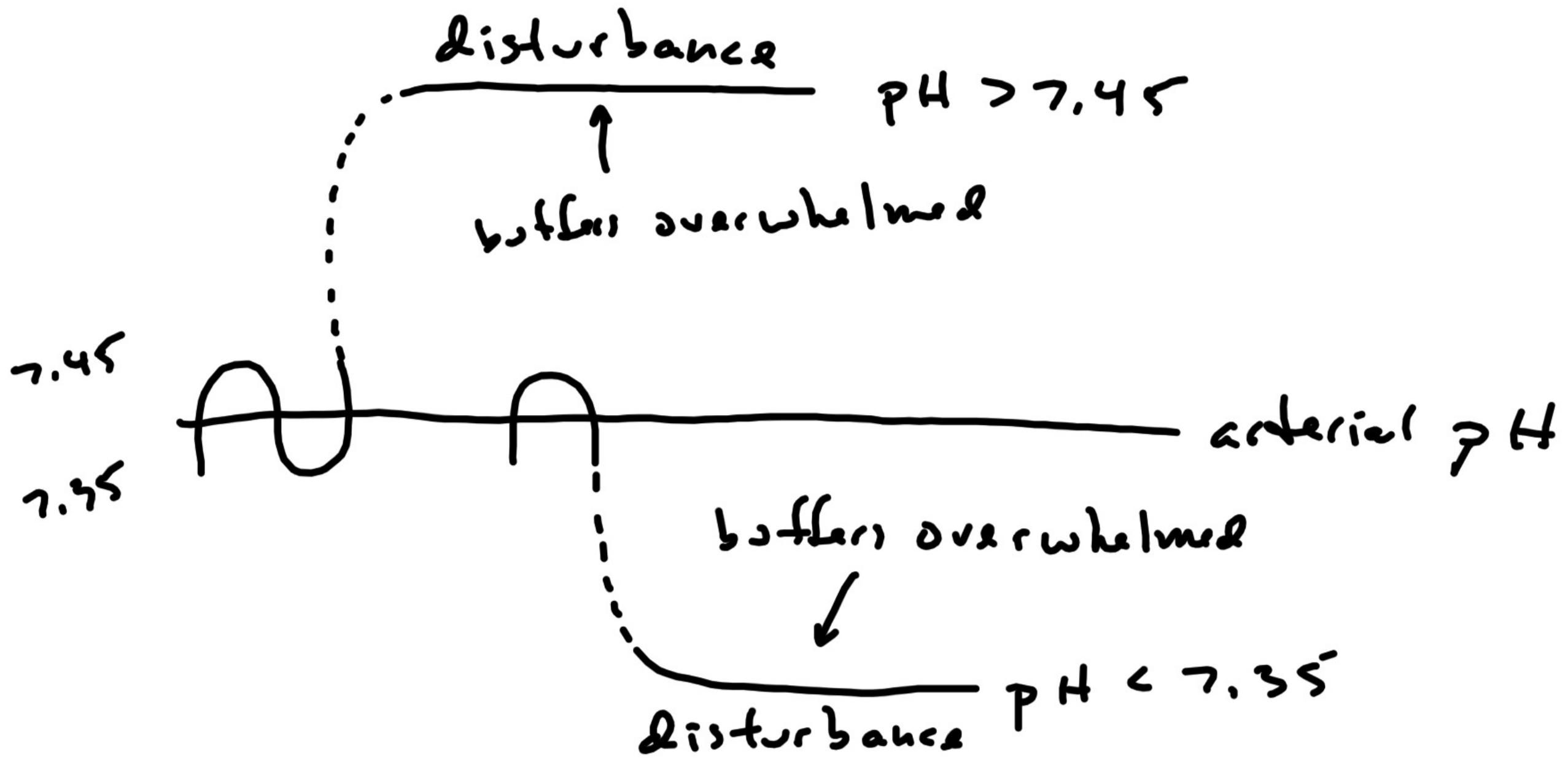


→ add base

↳ w/o buffers: 7.74

↳ w. buffers: 7.43





pH Regulation

- Arterial Blood pH ~ 7.40
 - Range : 7.35 - 7.45
- Arterial Blood $[HCO_3^-] \sim 24 \text{ mEq / L}$
 - Range : 22 - 26 mEq / L
- Arterial Blood $P_{CO_2} (P_{aCO_2}) \sim 40 \text{ mm Hg}$
 - Range : 35 - 45 mm Hg
- $CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$
 - Ratio of $[HCO_3^-]$ to $[CO_2]$ dictates pH ... calculated by the Henderson-Hasselbach equation. Simplified form :

$$pH \propto \frac{[HCO_3^-]}{P_{CO_2}}$$

$$pH = pK + \log \frac{[HCO_3^-]}{(x_{CO_2})(P_{CO_2})}$$

$$pH \sim \frac{[HCO_3^-]}{P_{CO_2}}$$

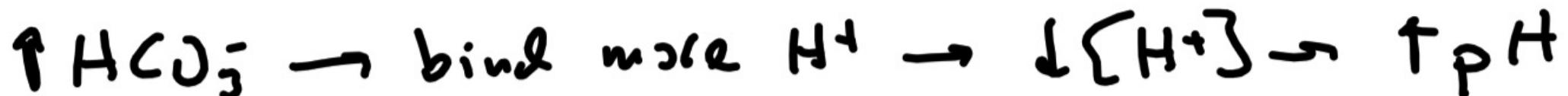
pH Regulation - Changing pH

- $\uparrow PCO_2 \rightarrow \uparrow H^+ formation \rightarrow \uparrow [H^+] \rightarrow \downarrow pH$
- $\downarrow PCO_2 \rightarrow \downarrow H^+ formation \rightarrow \downarrow [H^+] \rightarrow \uparrow pH$
- $\uparrow [HCO_3^-] \rightarrow binds\ more\ H^+ \rightarrow \downarrow [H^+] \rightarrow \uparrow pH$
- $\downarrow [HCO_3^-] \rightarrow binds\ less\ H^+ \rightarrow \uparrow [H^+] \rightarrow \downarrow pH$
- $\uparrow acid\ (eg.\ ketoacid) \rightarrow \uparrow [H^+] \ and \ \downarrow [HCO_3^-] \rightarrow \downarrow pH$



$$pH \approx \frac{[HCO_3^-]}{P_{CO_2}}$$

[CO_2 is acid HCO_3^- is a base]

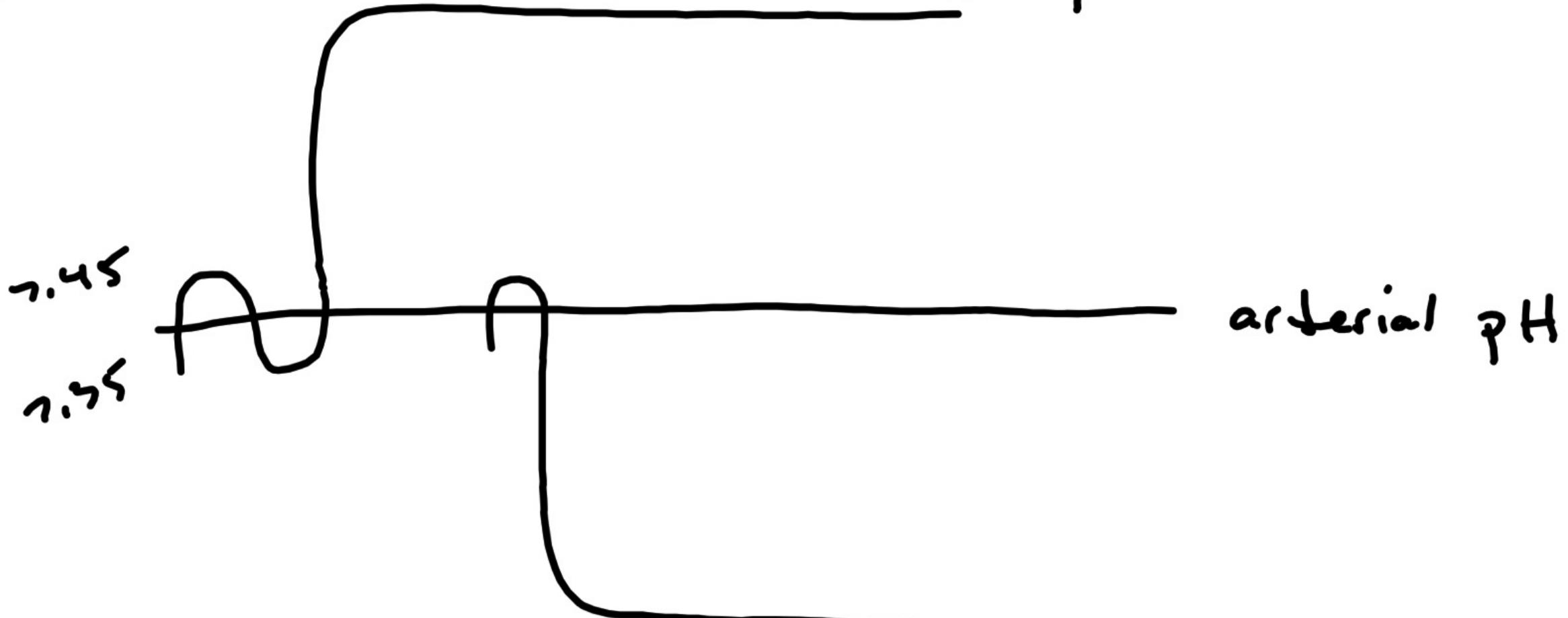


Acid - Base Disturbances

- Respiratory Alkalosis
 - Decreased level of arterial P_{CO_2} (less than 35 mm Hg)
 - Causes a pH > 7.45
 - Caused by hyperventilation
 - eg , Hyperthyroidism
 - Stimulates respiratory center via increased T_3 and T_4
 - eg , Anxiety Attack
 - Stimulates respiratory center via hypothalamus
 - eg , Acute aspirin toxicity
 - Stimulates respiratory center
- Respiratory Acidosis
 - Increased level of arterial P_{CO_2} (greater than 45 mm Hg)
 - Causes a pH < 7.45
 - Caused by hypoventilation or poor gas exchange at the lungs
 - eg , Pulmonary diseases
 - Decrease ventilation and poor gas exchange
 - eg , Hypothyroidism
 - Decreased T_3 and T_4 depresses respiratory center
 - eg , Opiates (eg , morphine) benzodiazepines (eg , Xanax)
 - Inhibit respiratory center

$P_{CO_2} < 35 \rightarrow$ Respiratory Alkalosis: pH > 7.45

$HCO_3 > 26 \rightarrow$ Metabolic Alkalosis: pH > 7.45

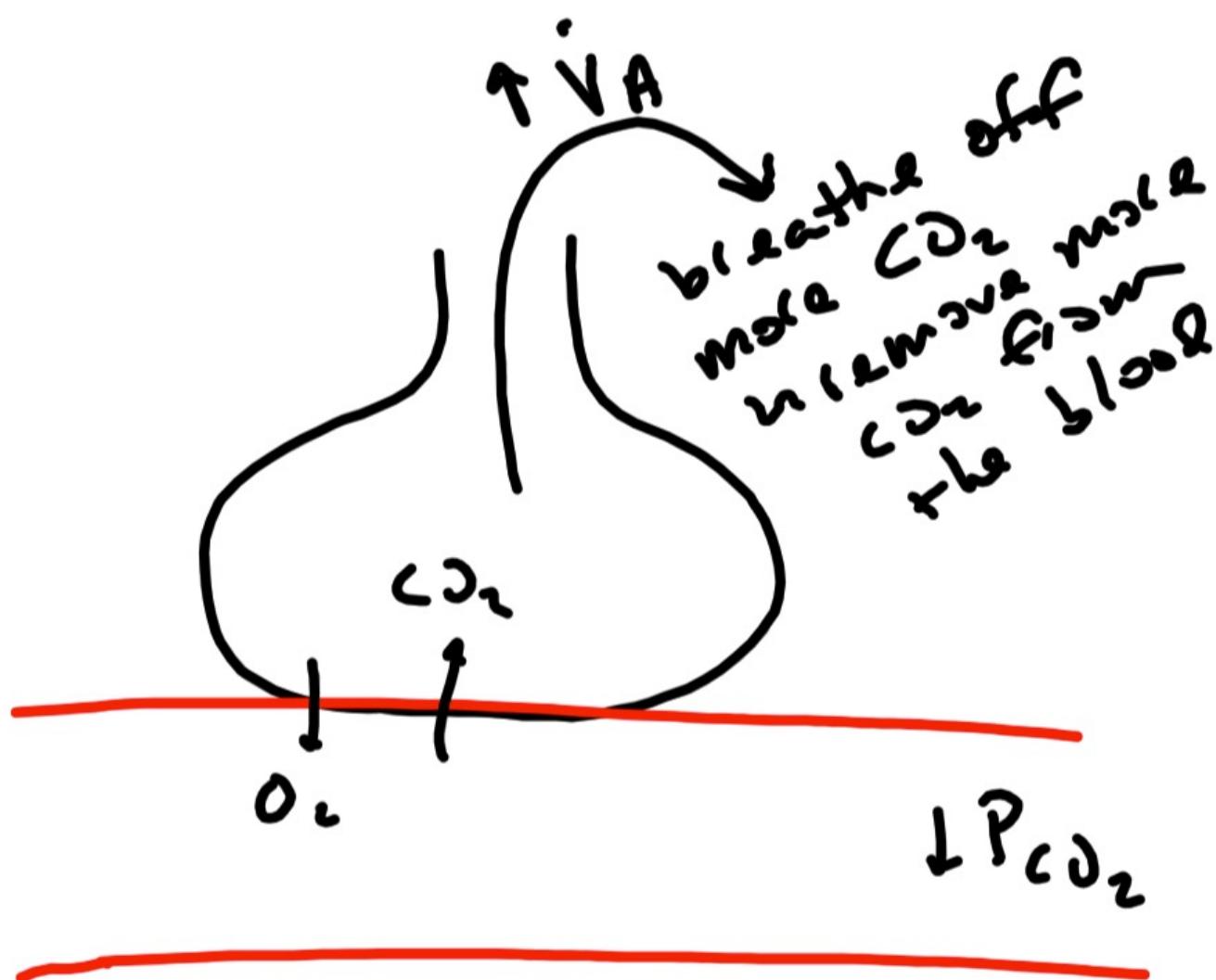


$P_{CO_2} > 45 \rightarrow$ Respiratory Acidosis: pH < 7.35

$HCO_3 < 22 \rightarrow$ Metabolic Acidosis: pH < 7.35

Respiratory = CO_2
Metabolic = HCO_3^-

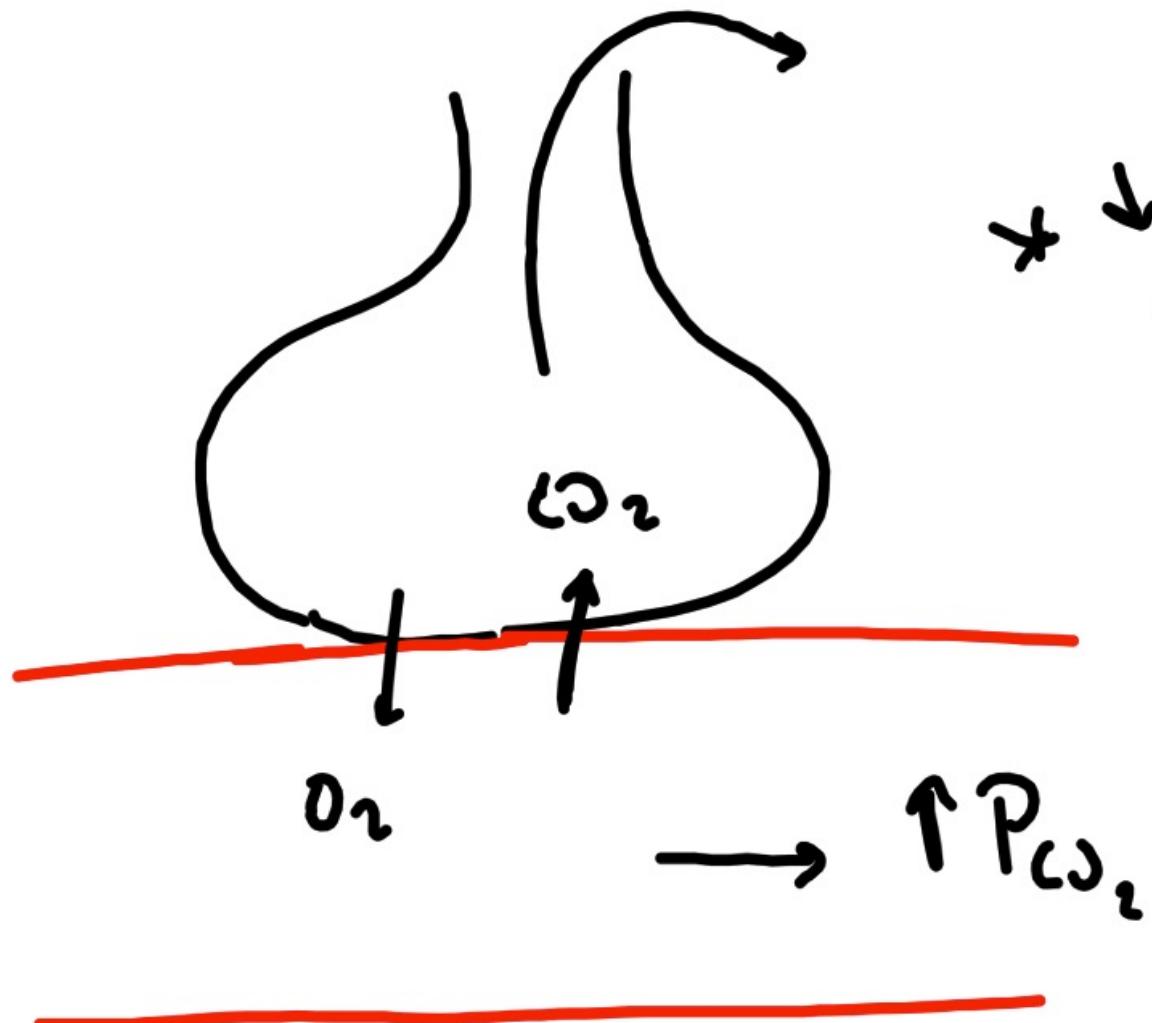
Respiratory Alkalosis



due to
hyperventilation
($\uparrow \dot{V}A$)

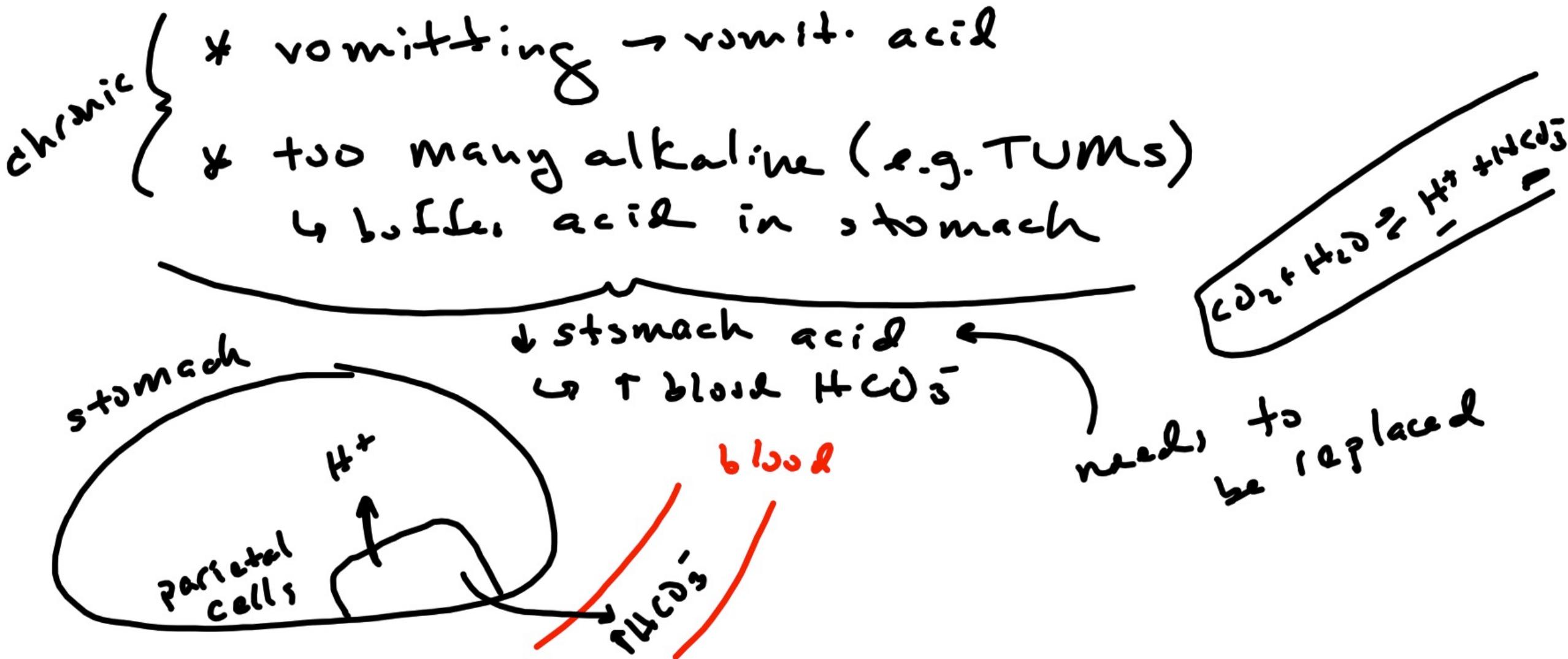
pulmonary capillary

Respiratory Acidosis ($P_{CO_2} > 45$)



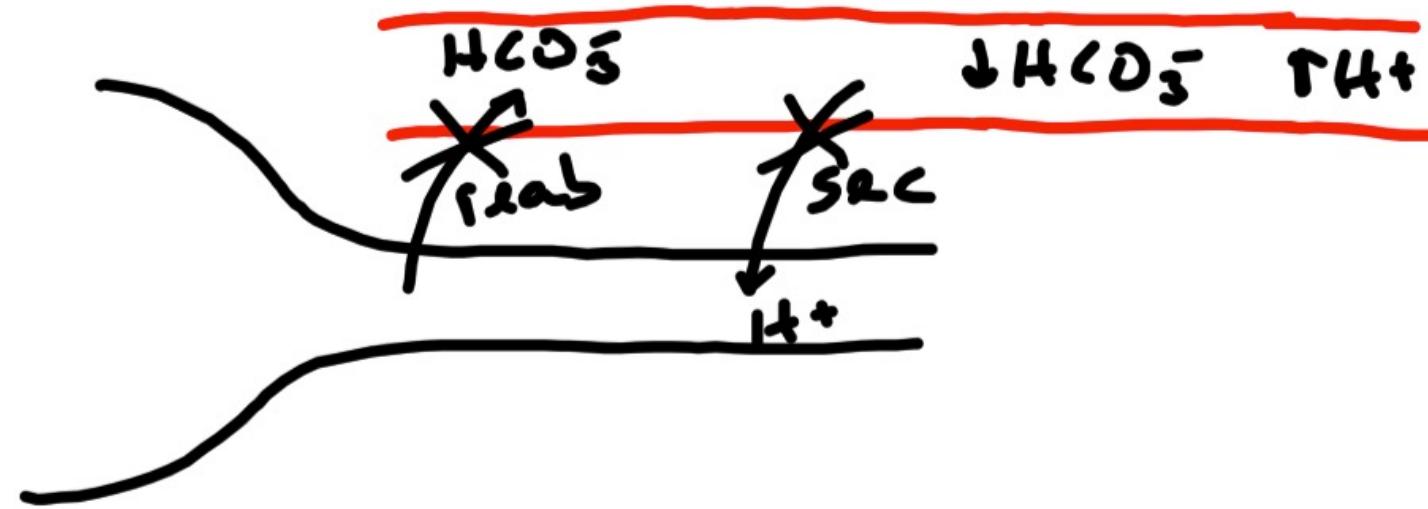
- * ↓ V_A
- * ↓ B_L + the off
- ↳ ↑ O₂
- ↳ ↑ P_{CO₂}
- * sometimes Gas exchange
- ↳ ↑ CO₂ out
- ↳ less blood
- ↳ ↓ P_{CO₂}

* Metabolic Alkalosis ($HCO_3^- > 26$)



* Metabolic Acidosis ($\underline{\text{HCO}_3^-} < 22$)

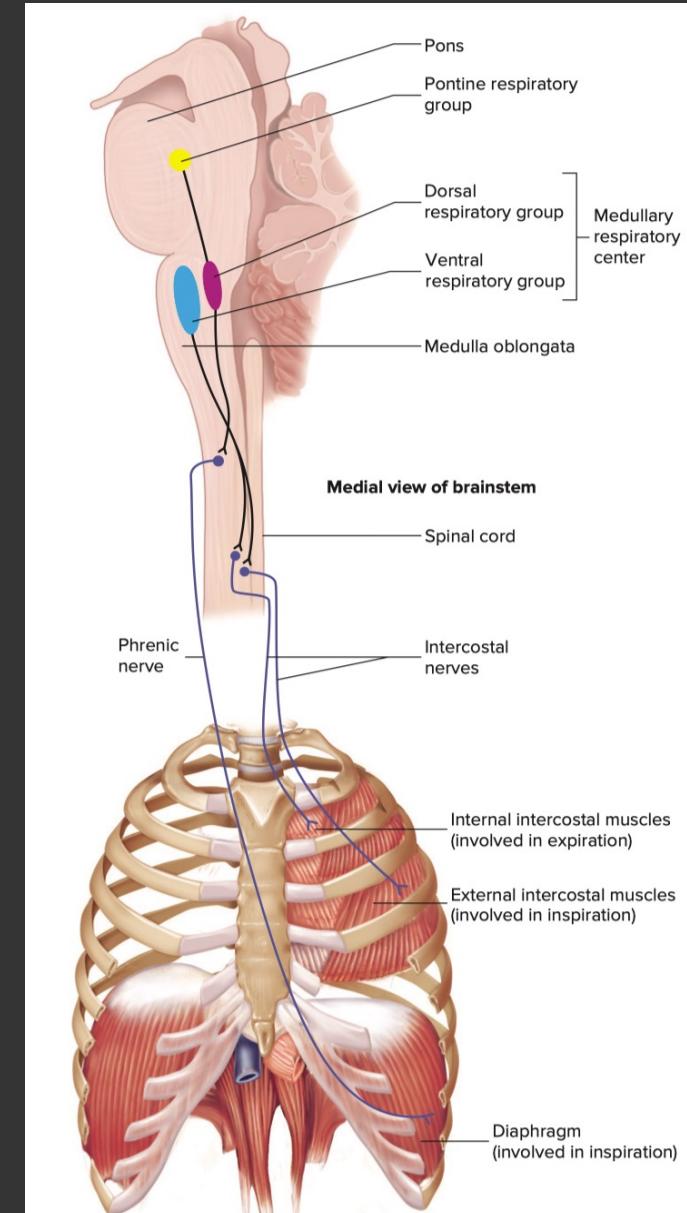
* Kidney disease



* Chronic Diarrhea \rightarrow loss of digestive tract HCO_3^- needs to be replaced
↳ in part get it from the blood
↳ ↓ blood HCO_3^-

Respiratory Center

- Bilateral network of neurons with pacemaker-like activity in the medulla
- Dorsal Respiratory Group :
 - Innervate and drive the respiratory muscles
- Ventral Respiratory Group :
 - Innervate and drive the respiratory muscles
 - Contains pacemaker neurons
 - pre-Bötzinger complex



Acid - Base Disturbances - Metabolic Alkalosis

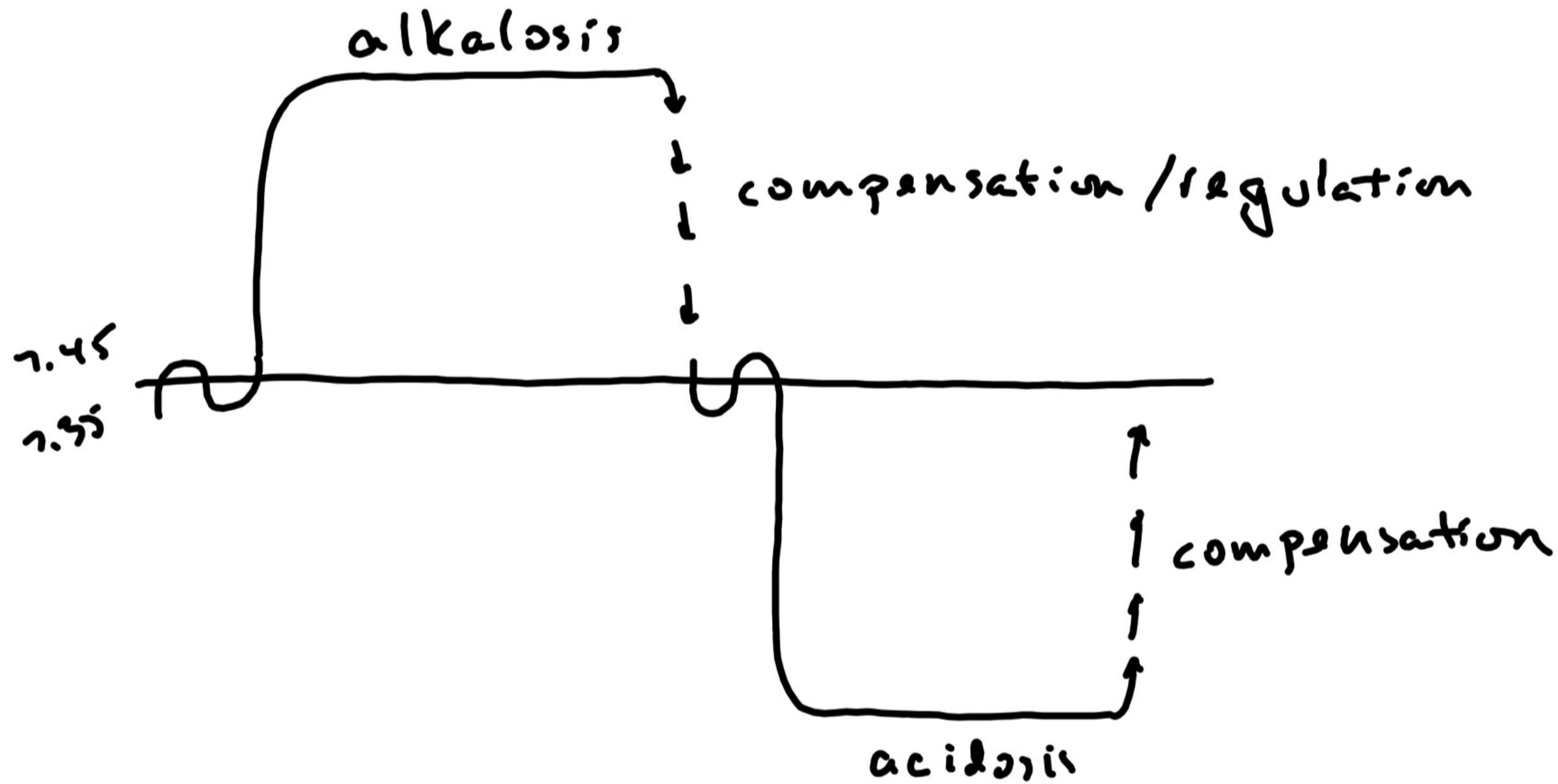
- Failure of the kidneys to maintain a normal arterial blood HCO_3^-
- Increased level of arterial HCO_3^- (greater than 26 mEq / L)
 - Causes a pH > 7.45
- Caused by :
 - eg , Gastric vomiting
 - Loss of stomach acids results in increased stomach pH
 - More gastric acid produced to replace lost acid
 - Results in HCO_3^- transport into the blood
 - eg , Ingestion of too many alkaline drugs
 - Increases stomach pH
 - More gastric acid produced to normalize pH
 - Results in HCO_3^- transport into the blood

Acid - Base Disturbances - Metabolic Acidosis

- Failure of the kidneys to maintain a normal arterial blood HCO_3^-
- Decreased level of arterial HCO_3^- (less than 22 mEq / L)
 - Causes a pH < 7.35
- Caused by :
 - eg , Kidney disease
 - Failure to reabsorb HCO_3^- and / or secrete H^+
 - eg , Diarrhea
 - Rapid excretion of HCO_3^- from digestive tract
 - Lost HCO_3^- acquired from the plasma
 - eg , Acetylsalicylic acid (aspirin) toxicity
 - eg , Ketoacidosis

Compensation of Acid - Base Disturbances

- Regulation of pH back towards normal
 - Performed by the lungs and / or kidneys
 - With a metabolic disturbance :
 - If due to kidney disease , only the lungs can compensate
 - If kidneys are not damage , kidneys can compensate too
 - If there is a respiratory disturbance , the kidneys will compensate



Compensation of Acid - Base Disturbances

- **Respiratory Compensation** (ie , lungs regulate pH back to normal) :
 - Performed by the lungs via changes in alveolar ventilation
 - Lungs compensate in minutes to hours
 - Lungs compensate metabolic disturbances
 - Disturbance : **Metabolic Alkalosis** (ie , increase in HCO_3^-)
 - Body responds with respiratory compensation
 - Decrease ventilation → increases P_{CO_2}
 - Decrease pH back towards normal
 - Disturbance : **Metabolic Acidosis** (ie , decrease in HCO_3^-)
 - Body responds with respiratory compensation
 - Increase ventilation → decreases P_{CO_2}
 - Increase pH back toward normal

* Respiratory Compensation

$$pH = \frac{HCO_3^-}{P_{CO_2}}$$

- lungs compensate in seconds / minutes
- lungs compensate metabolic disturbances
- lungs DO NOT compensate respiratory disturbance
- How? → change $\dot{V}A$
 - * met alkalosis ($\uparrow HCO_3^-$)
 - ↳ $\downarrow \dot{V}A \rightarrow \uparrow P_{CO_2}$
 - * met acidosis ($\downarrow HCO_3^-$)
 - ↳ $\uparrow \dot{V}A \rightarrow \downarrow P_{CO_2}$

Compensation of Acid - Base Disturbances

- Metabolic Compensation (ie , kidneys regulate pH back to normal)
 - Performed by the kidneys via changes in reabsorption and secretion
 - Kidneys take days to compensate
 - Compensate respiratory disturbances
 - Compensate metabolic disturbances if not due to kidney issue
- Disturbance : *Respiratory Alkalosis* (ie , decreases in P_{CO_2})
 - Body responds with metabolic compensation
 - Decrease secretion of H^+
 - Decrease reabsorption of HCO_3^-
 - Above decrease pH back towards normal
- Disturbance : *Respiratory Acidosis* (ie , increase in P_{CO_2})
 - Body responds with metabolic compensation
 - Increase secretion of H^+
 - Increase reabsorption of HCO_3^-
 - Above increase pH back towards normal

Compensation of Acid - Base Disturbances

- Metabolic compensation (ie , kidneys regulate pH back to normal)
 - Performed by the kidneys via changes in reabsorption and secretion
 - Kidneys take days to compensate
 - Compensate respiratory disturbances
 - Compensate metabolic disturbances if not due to kidney issue
 - Disturbance : *Metabolic Alkalosis* (ie , increase in HCO_3^-)
 - Body responds with metabolic compensation
 - ONLY if there is no kidney injury or disease
 - Decrease secretion of H^+
 - Decrease reabsorption of HCO_3^-
 - Above decrease pH back towards normal
 - Disturbance : *Metabolic Acidosis* (ie , decrease in HCO_3^-)
 - Body responds with metabolic compensation
 - ONLY if there is no kidney injury or damage
 - Increase secretion of H^+
 - Increase reabsorption of HCO_3^-
 - Above increase pH back towards normal

* Metabolic Compensation

$$\text{pH} \sim \frac{\text{HCO}_3^-}{\text{PCO}_2}$$

- Kidneys take days to compensate
- Kidneys compensate respiratory disturbances
- Kidneys can compensate metabolic disturbance if they're not the problem
- HOW? → change HCO_3^- reab and H^+ secretion

* resp acidosis ($\uparrow \text{PCO}_2$)

↳ reab more HCO_3^- and secrete more H^+

* resp alkalosis ($\downarrow \text{PCO}_2$)

↳ reab less HCO_3^- and secrete less H^+

if kidney's not the problem
↳ reab more HCO_3^- : secrete more H^+

↳ reab less HCO_3^- : secrete less H^+

Compensation of Acid - Base Disturbances

- No Compensation :
 - Disturbance has yet to be regulated
 - pH is abnormal and either P_{CO_2} or HCO_3^- is also abnormal
 - If P_{CO_2} is abnormal , then HCO_3^- will be normal
 - If HCO_3^- is abnormal , then P_{CO_2} will be normal
 - Certain disturbances where pH , P_{CO_2} and HCO_3^- are all abnormal
 - If P_{CO_2} is increased , HCO_3^- will be decreased
 - If P_{CO_2} is decreased , HCO_3^- will be increased
- Partial Compensation :
 - Disturbance is being regulated toward normal
 - pH , P_{CO_2} , and HCO_3^- will be abnormal
 - P_{CO_2} and HCO_3^- will both be increased or both will be decreased
- Full Compensation :
 - Disturbance has been regulated to normal
 - pH will be normal but both P_{CO_2} and HCO_3^- will be abnormal
 - P_{CO_2} and HCO_3^- will both be increased or both will be decreased

* Respiratory alkalosis ($\downarrow \text{PCO}_2$; $\uparrow \text{pH}$)
leads to $\uparrow \text{VA}$

$$\text{pH} = \frac{\text{HCO}_3^-}{\text{PCO}_2}$$

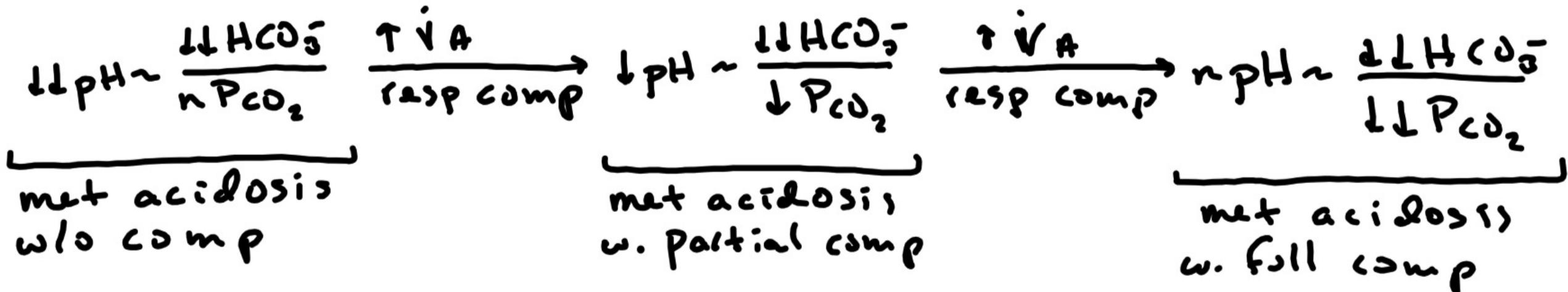
$$\uparrow \text{pH} \sim \frac{n\text{HCO}_3^-}{\downarrow \text{PCO}_2} \quad \begin{array}{c} \text{trab HCO}_3^- \\ \downarrow \text{sec H}^+ \\ \text{met comp} \end{array} \quad \text{pH} \sim \frac{\downarrow \text{HCO}_3^-}{\downarrow \text{PCO}_2} \quad \begin{array}{c} \text{trab HCO}_3^- \\ \downarrow \text{sec H}^+ \\ \text{met comp} \end{array} \quad n\text{pH} \sim \frac{\downarrow \text{HCO}_3^-}{\downarrow \text{PCO}_2}$$

resp alkalosis
w. no comp

resp alkalosis
partial comp

resp alkalosis
w. full comp

* Metabolic Acidosis ($\downarrow \text{HCO}_3^-$; $\downarrow \text{pH}$) $\text{pH} = \frac{\text{HCO}_3^-}{\text{P}_{\text{CO}_2}}$



* If kidneys not issue

↳ ↑ reab of HCO_3^- and ↑ secretion of H^+



Examples of Acid - Base Disturbances

Resp Acidosis w. no comp

pH 7.15
 P_{CO_2} 65 mm Hg
 $[HCO_3^-]$ 22 mEq/L

Resp Acidosis w. partial comp

pH 7.26
 P_{CO_2} 65 mm Hg
 $[HCO_3^-]$ 28 mEq/L

Resp Acidosis w. full comp.

pH 7.35
 P_{CO_2} 65 mm Hg
 $[HCO_3^-]$ 35 mEq/L

Met Acidosis w. no comp

pH 7.18
 P_{CO_2} 39 mm Hg
 $[HCO_3^-]$ 14 mEq/L

Met Acidosis w. partial comp

pH 7.29
 P_{CO_2} 30 mm Hg
 $[HCO_3^-]$ 14 mEq/L

Met Acidosis w. full comp.

pH 7.37
 P_{CO_2} 25 mm Hg
 $[HCO_3^-]$ 14 mEq/L

Resp Alkalosis w. no comp

pH 7.57
 P_{CO_2} 26 mm Hg
 $[HCO_3^-]$ 23 mEq/L

Resp Alkalosis w. partial comp

pH 7.51
 P_{CO_2} 26 mm Hg
 $[HCO_3^-]$ 20 mEq/L

Resp Alkalosis w. full comp.

pH 7.41
 P_{CO_2} 26 mm Hg
 $[HCO_3^-]$ 16 mEq/L

Met Alkalosis w. no comp

pH 7.54
 P_{CO_2} 42 mm Hg
 $[HCO_3^-]$ 35 mEq/L

Met Alkalosis w. partial comp

pH 7.49
 P_{CO_2} 49 mm Hg
 $[HCO_3^-]$ 35 mEq/L

Met Alkalosis w. full comp.

pH 7.43
 P_{CO_2} 55 mm Hg
 $[HCO_3^-]$ 35 mEq/L

Examples of Acid - Base Disturbances

➤ Mixed Disturbance :

➤ When two or more disturbances occur simultaneously

➤ eg , respiratory acidosis with a simultaneous metabolic acidosis

pH	7.01
P_{CO_2}	58 mm Hg
$[HCO_3^-]$	14 mEq/L

➤ eg , respiratory alkalosis with a simultaneous metabolic alkalosis

pH	7.79
P_{CO_2}	23 mm Hg
$[HCO_3^-]$	34 mEq/L

➤ And then things can get very complicated :

- eg , metabolic acidosis with a respiratory alkalosis
- eg , metabolic alkalosis with a respiratory acidosis
- eg , metabolic acidosis with a metabolic alkalosis
- eg , respiratory acidosis with metabolic acidosis with metabolic alkalosis

pH 7.11 ↓↓

P_{CO₂} 38 n

HCO₃⁻ 15 ↓↓

met^x
acidosis
w/ D comp

$$\text{pH} \sim \frac{\text{HCO}_3^-}{\text{P}_{\text{CO}_2}}$$

pH: 7.35 - 7.45
P_{CO₂}: 35 - 45
HCO₃⁻: 22 - 26

pH: 7.26 ↓

P_{CO₂}: 31 ↓

HCO₃⁻: 15 ↓↓

met^x
acidosis
w/ Partial comp?

pH: 7.37 n

P_{CO₂}: 24 ↓↓

HCO₃⁻: 15 ↓↓

met^x
acidosis
w/ full comp or

(as P)
alkalosis
w/ full comp

pH: 7.24 //

P_{CO₂}: 20 //

HCO₃: 4 ↓↓↓↓↓↓

met acidosis
w. partial comp

pH: 7.73 ↑↑

P_{CO₂}: 61 ↑↑

HCO₃: 15 //

NOT POSSIBLE

pH: 7.10 ↓↓

P_{CO₂}: 61 ↑↑

HCO₃: 18 ↓↓

$$\text{pH} \approx \frac{\text{HCO}_3^-}{\text{P}_{\text{CO}_2}}$$

(27) ^{met} acidosis
mixed disturbance

pH

7.79

P_{CO_2} is (\dot{V}_A)
 HCO_3 is (\downarrow reabs HCO_3)

resp alk
w. partial
comp