#3 Physiology of the stomach and regulation of gastric secretions

objectives :

- Functions of stomach
- Gastric secretion
- Mechanism of HCl formation
- Gastric digestive enzymes
- Neural & hormonal control of gastric secretion
- Phases of gastric secretion
- Motor functions of the stomach
- Stomach Emptying

Doctors' notes

Extra

Important

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Resources: 435 Boys' & Girls' slides | Guyton and Hall 12th & 13th edition

Editing file

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Anatomy & physiology of the stomach :

Stomach

Physiologically

Orad, Reservoir part (Fundus & upper ⅔ of the Body) (tonic contraction)

The main functions of the (Reservoir part) :

- **Maintain a continuous compression.**
- **To accommodate the received food (storage of food).**

Caudad, Antral pump part (lower ⅓ of Body & Antrum) (Phasic contraction)

Why do we divide the stomach physiologically into two parts?

Because the two parts are totally different in their function :

1.Reservoir part : Storage function, in fundus and upper two third of the body.

2.antral part : Represent the real function of secreting gastric juice.

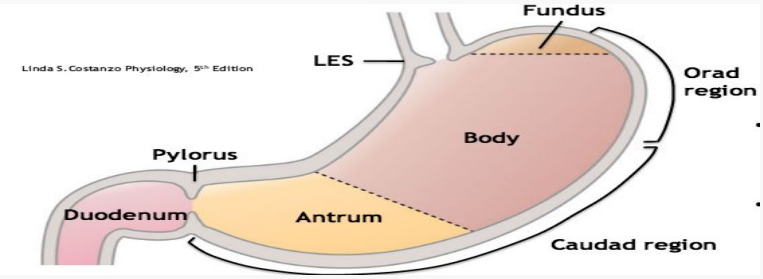
Muscular wall

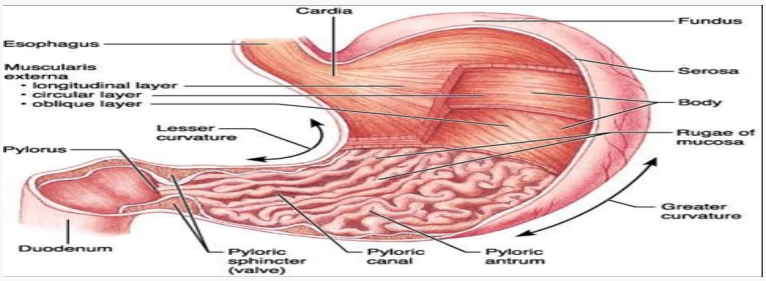
- **Longitudinal (outer)**
- **Circular (middle)**
- **Oblique (inner)**

3 layers of muscles! = Imp. role in digestion

Anatomically

- **Fundus** "receptive relaxation"
- **Body**
- **Antrum**
- **Pylorus**





- **Rugae = large folds.**

  The purpose of the gastric rugae is to allow for expansion of the stomach after the consumption of food and liquids.

- **Mucus = protects lining of stomach** (rich in alkaline)

Functions of the Stomach

Storage of food

-The stomach accomadate for the recived food without significant gaastric wall distention or pressure

- When food enters the fundus (receptive relaxation), it relaxes to accommodate the food.(Maintain a continuous compression)

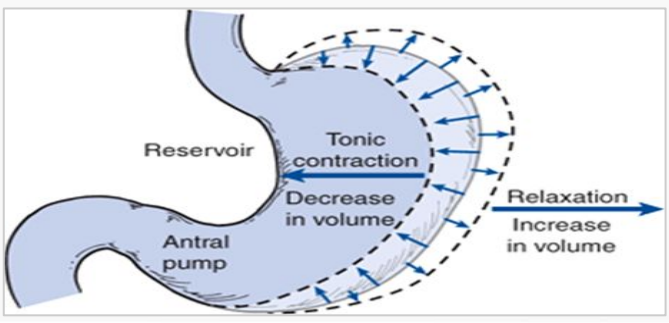
Digestion

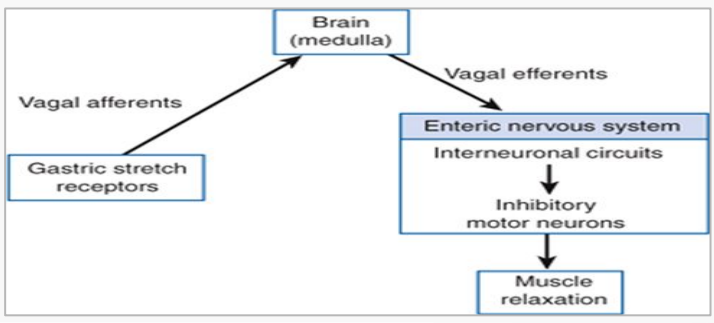
Chemical:
Protein digestion

Mechanical:
Mix - churn
Mixed with enzyme

Relaxation reflexes in reservoir part:

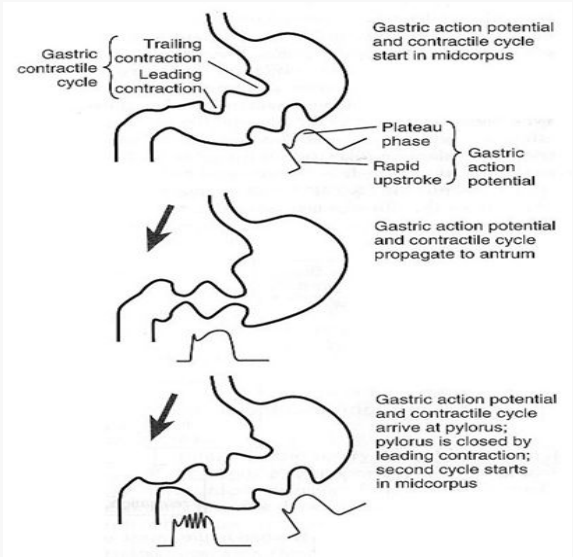
	Receptive relaxation reflex	Adaptive relaxation (vagovagal reflex)	Feedback relaxation:
Triggered by	swallowing reflex	stretch receptors	small intestine (due to presence of nutrient in it)
Action	• increase the volume through the inhibition of myenteric plexus	• reduce the tone in the stomach wall.	• Relaxation
Notes	• Relax LES (Lower esophageal sphincter).	• This reflex is lost in vagotomy (lead to decrease wall compliance & decrease threshold for sensation of pain and fullness.	• Done by neural (ENS) or hormonal pathway. • Can involve local reflex between receptors in small intestine.

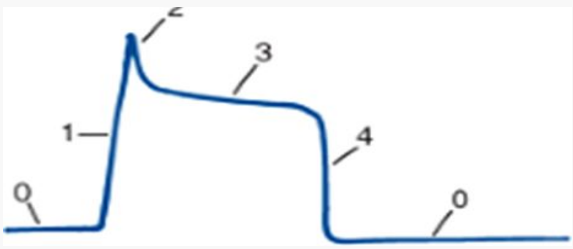




Electrical action potential in GI muscles : (Gastric action potential)

0	RMP; outward K+ current
1	Depolarization; activation of Ca^{2+} & K+ voltage-gated channels .
2	-
3	Plateau phase; balance of Ca^{2+} inward & K+ outward current.
4	Repolarization; inactivation of voltage-gated Ca^{2+}channel & activation of calcium-gated channels.



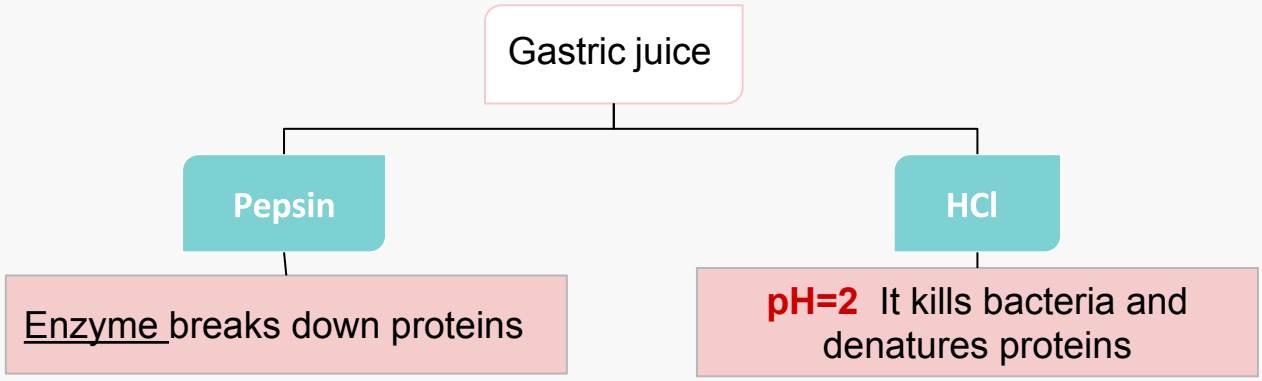


Gastric action potential triggers two kinds of contraction at the antrum:

❖ Leading contraction	❖ Trailing contraction
• Constant amplitude, associated with **Phase 1** • have negligible amplitude as they propagate to the pylorus • causing closing of the orifice between stomach & duodenum.	• variable amplitude, associated with **phase 3** • In trailing contraction;due to closed pylorus, gastric content forced into a small volume and high pressure, this will lead to retropulsion of food particle through trailing contraction which will help in decreasing the size of food particle **(Retropulsion phenomena)**

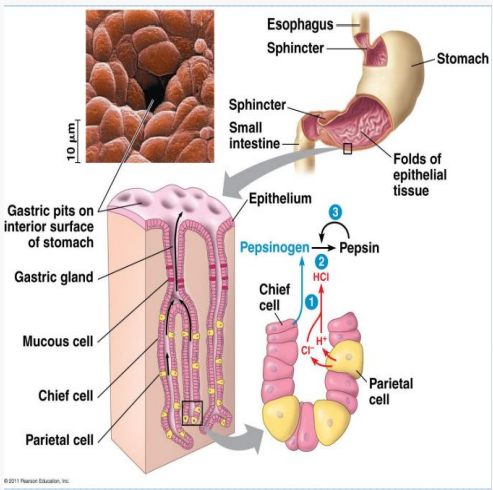
Gastric juice: converts meal to acidic chyme (due to the very acidic HCl)

"**Chyme:** murky semi-fluid or paste composed of food that is thoroughly mixed with gastric secretion"

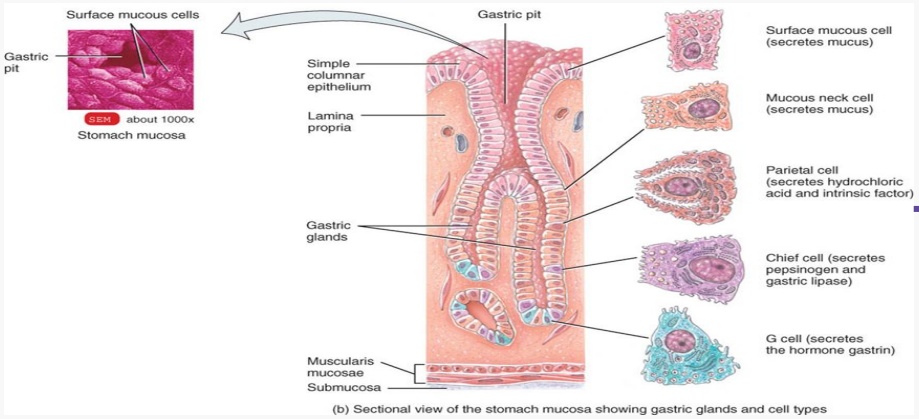


Gastric secretion :

- **Gastric glands** empty into the bottom of **gastric pits,** which are numerous openings in the gastric mucosa, synthesis of all secretions happen inside these glands.
- **Glands are composed of 4 functionally different cell types:**
  - Mucous cells (HCO3&Mucus)
  - Chief cells (releases Pepsinogen)
  - Parietal cells (releases HCl and Intrinsic factor)
  - Enteroendocrine cells (release hormones)
  - Enterochromaffin-like cells (secrete histamine).



Enterochromaffin-like cells are **enteroendocrine and neuroendocrine** cells also known for their similarity to chromaffin cells secreting histamine, which stimulates G cells to secrete gastrin.
G Cells: Gastrin (hormone): increases HCl secretion
D Cells: Somatostatin:decreases HCl secretion

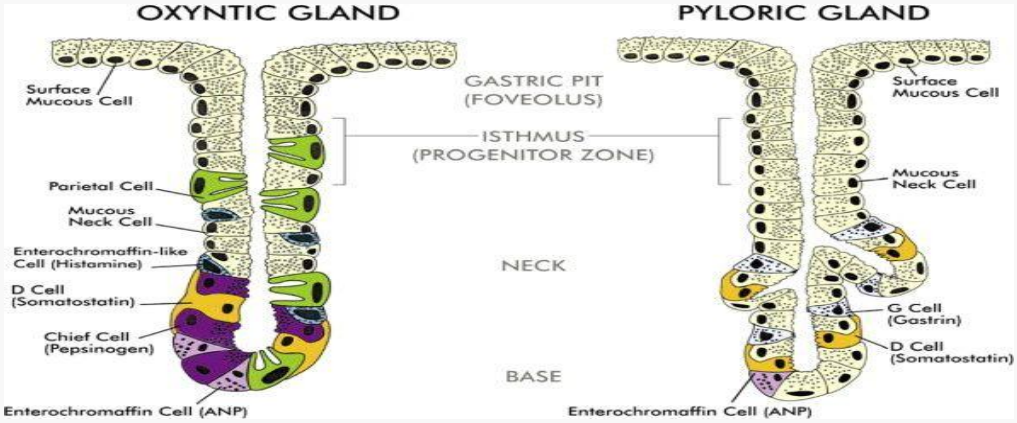


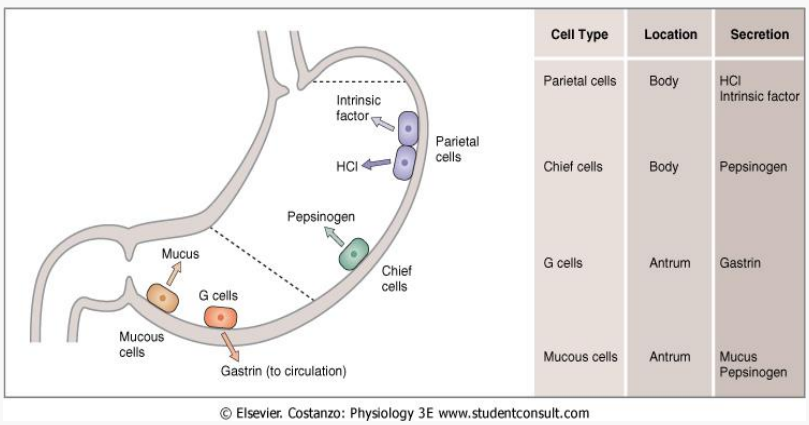
(b) Sectional view of the stomach mucosa showing gastric glands and cell types

Guyton corner :
Parietal cell (also called an oxyntic cell) contains large branching intracellular canaliculi. HCl is formed at the villus-like projections inside these canaliculi and is then conducted through the canaliculi to the secretory end of the cell.

Secretory functions of the stomach:

In addition to **mucus-secreting cells** that line the stomach and secrete alkaline mucus, there is two important types of **tubular glands**:

	oxyntic (gastric) glands	Pyloric glands
Secretion	• Hydrochloric acid (HCL) • Intrinsic factor Both secreted by parietal (oxyntic) cells	• Gastrin Secreted by G cells (a type of enteroendocrine cells)
	• Pepsinogen • Mucus - protection - (HCO3-)	
Location	proximal **80%** of stomach (body & fundus)	distal **20%** of stomach (antrum and pylorus)
Illustration (extra pic)		

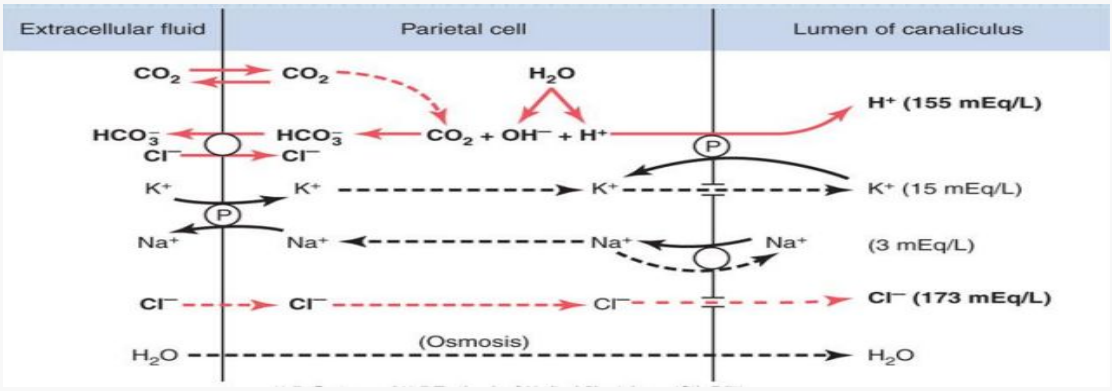


Cell Type	Location	Secretion
Parietal cells	Body	HCl Intrinsic factor
Chief cells	Body	Pepsinogen
G cells	Antrum	Gastrin
Mucous cells	Antrum	Mucus Pepsinogen

Guyton corner :

The pyloric glands are structurally similar to the oxyntic glands but contain few peptic cells and almost no parietal cells. Instead, they contain mostly mucous cells that are identical with the mucous neck cells of the oxyntic glands. These cells secrete a small amount of pepsinogen, as discussed earlier, and especially large amount of thin mucus that helps to lubricate food movement, as well as to protect the stomach wall from digestion by the gastric enzymes.

HCl production and secretion :



P Active pumps

Free diffusion and osmosis

Very helpful video !

1. **Chloride ion is actively transported from the cytoplasm of the parietal cell into the lumen of the canaliculus, and sodium ions are actively transported out of the canaliculus into the cytoplasm of the parietal cell.**

2. **Water becomes dissociated into *hydrogen ions* and *hydroxyl ions in the cell cytoplasm. The* hydrogen ions are then actively secreted into the canaliculus in exchange for potassium ions.**

3. **Carbon dioxide, either formed during metabolism in the cell or entering the cell from the blood, combines under the influence of carbonic anhydrase with the hydroxyl ions to form bicarbonate ions. These then diffuse out of the cell cytoplasm into the extracellular fluid in exchange for chloride ions that enter the cell.**

❖ HCl production:

- **Depends on H/K ATPase**
- **Inhibited by: omeprazole**
- **H/K pump depends on $[K]_{out}$**
- **[HCl] drives water into gastric content to maintain osmolality**
- **During gastric acid secretion: amount of HCO_3^- in blood = amount of HCl being secreted**
- **Alkaline tide** (refers to a condition, normally encountered after eating a meal, where during the production of HCl by parietal cells in the stomach, the parietal cells secrete bicarbonate ions across their basolateral membranes and into the blood, causing a temporary increase in pH.)

Guyton corner :

1. Water inside the parietal cell becomes dissociated into H+ and hydroxide (OH−) in the cell cytoplasm. The H+ is then actively secreted into the canaliculus in exchange for K+, an active exchange process that is catalyzed by H+-K+ ATPase. Potassium ions transported into the cell by the Na+-K+ ATPase pump on the basolateral (extracellular) side of the membrane tend to leak into the lumen but are recycled back into the cell by the H+-K+ ATPase. The basolateral Na+-K+ ATPase creates low intracellular Na+, which contributes to Na+ reabsorption from the lumen of the canaliculus. Thus, most of the K+ and Na+ in the canaliculus is reabsorbed into the cell cytoplasm, and hydrogen ions take their place in the canaliculus.
2. The pumping of H+ out of the cell by the H+-K+ ATPase permits OH− to accumulate and form bicarbonate (HCO3−) from CO2, either formed during metabolism in the cell or while entering the cell from the blood. This reaction is catalyzed by carbonic anhydrase. The HCO3− is then transported across the basolateral membrane into the extracellular fluid in exchange for chloride ions, which enter the cell and are secreted through chloride channels into the canaliculus, giving a strong solution of hydrochloric acid in the canaliculus. The hydrochloric acid is then secreted outward through the open end of the canaliculus into the lumen of the gland.3. Water passes into the canaliculus by osmosis because of extra ions secreted into the canaliculus.

Neural & Hormonal Control of Gastric Secretion

- excitatory effect-

Vagus nerve

(Neural effector)

1) Direct activation of parietal cells:

by releasing Ach

2) Indirect activation:

by releasing GRP (Gastrin-releasing peptide)

In the parasympathetic system, the postganglion will be close to the organ, it releases GRP (specific for G cells) -> blood circulation -> stimulation of parietal cells

Gastrin

(Hormonal effector)

Guyton corner :
When meats or other foods containing protein reach the antral end of the stomach, some of the proteins from these foods have a special stimulatory effect on the gastrin cells in the pyloric glands to cause release of gastrin into the blood to be transported to the ECL cells of the stomach. The vigorous mixing of the gastric juices transports the gastrin rapidly to the ECL cells in the body of the stomach, causing release of histamine directly into the deep oxyntic glands. The histamine then acts quickly to stimulate gastric hydrochloric acid secretion.

1) Distention → local stimulation → sensory → myenteric plexus → release of Ach → G cell stimulation → gastrin release

2) Pepsinogen → Pepsin → breakdown of proteins → amino acid → touches the G-cells → gastrin release

Enterochromaffin-like cells (ECL cells)

Located close to the parietal cells

Release Histamine → activates H2 receptor (parietal cells) → increases acid secretion

Cimetidine: (inhibitory effect)

It is a H2 receptor blocker, used for peptic ulcer and gastroesophageal reflux.

1) Prostaglandins and prostacyclin inhibit parietal cells . This is why when you use NSAIDs, you ultimately stop prostaglandin release, which leads to HCL increase → ulcer

2) People with high acidity use cimetidine to block H2 receptors which inhibits HCl secretion. This can be used as a treatment for ulcers.

3) Inhibitors on proton pump also inhibits HCl secretion

The Rate of Secretion Modify the Composition of Gastric Juice :

- **At low secretion rate:** gastric juice contains **high** concentrations of Na+ and Cl- and **low** concentrations of K+ and H+.

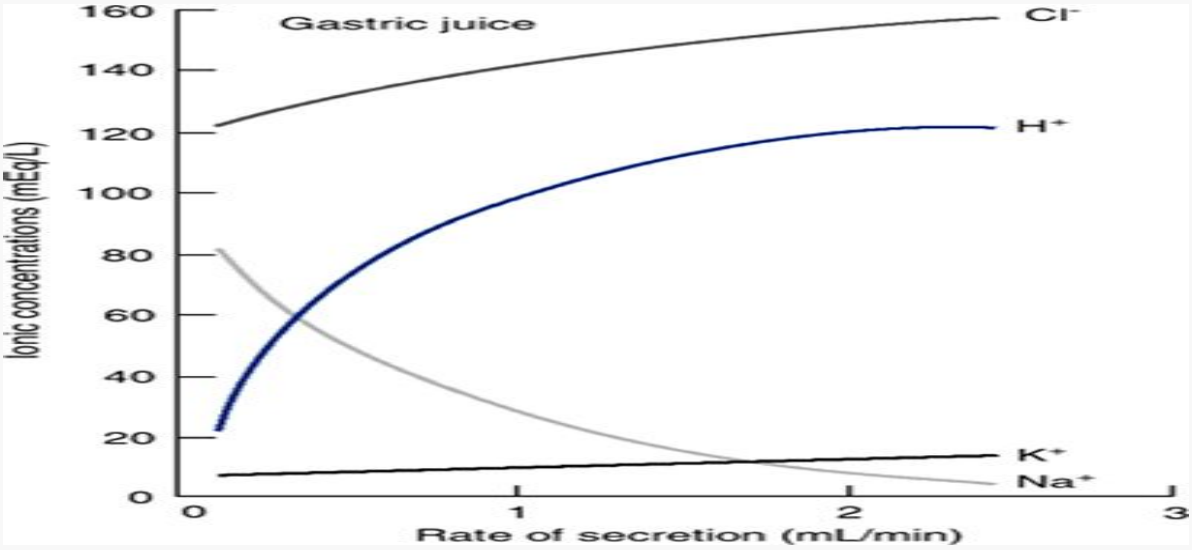
- **When the secretion rate increases (high):** the concentration of Na+ **decreases** whereas that of **H+ increases significantly** ,coupled with this increase in gastric secretion is an **increase** in Cl- concentration .

- This gastric juice is derived from **parietal** cells and **nonparietal** cells.

- Secretion from nonparietal cells is probably constant; therefore, it is parietal secretion (HCl secretion) that contributes mainly to the changes in electrolyte composition with higher secretion rates.

Briefly :

- **Low** secretion rate (between meals) - high NaCl
- **High** secretion rate (after a meal)- high HCl
- **Always isotonic**



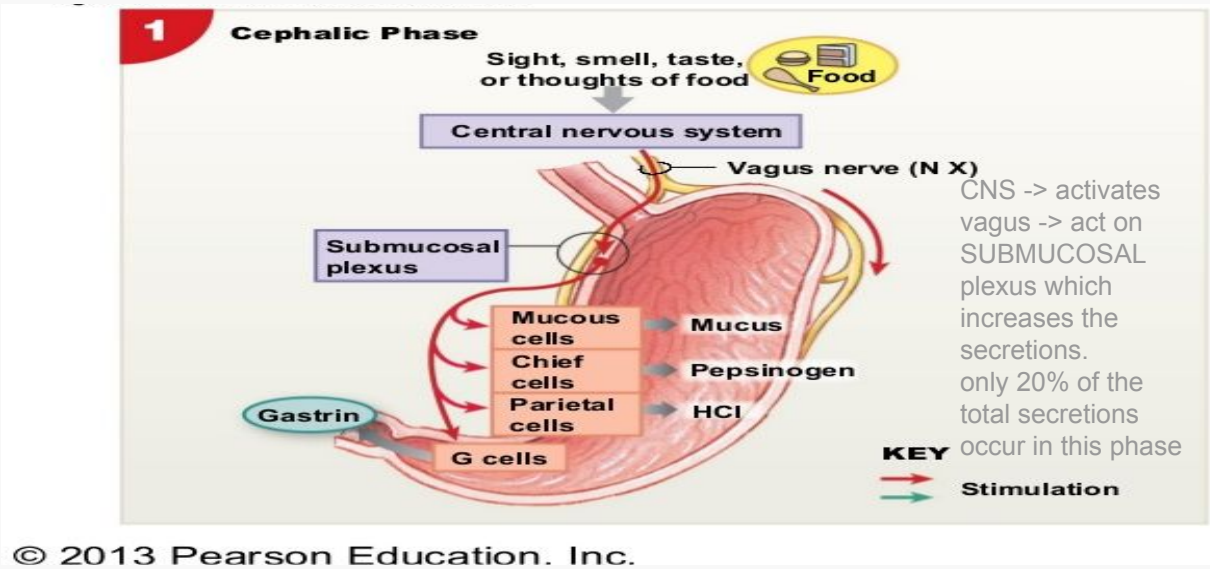
Phases of gastric secretion

(according to the phase in which you are taking in the food)

The videos are from the doctor's slides

1) Cephalic Phase:

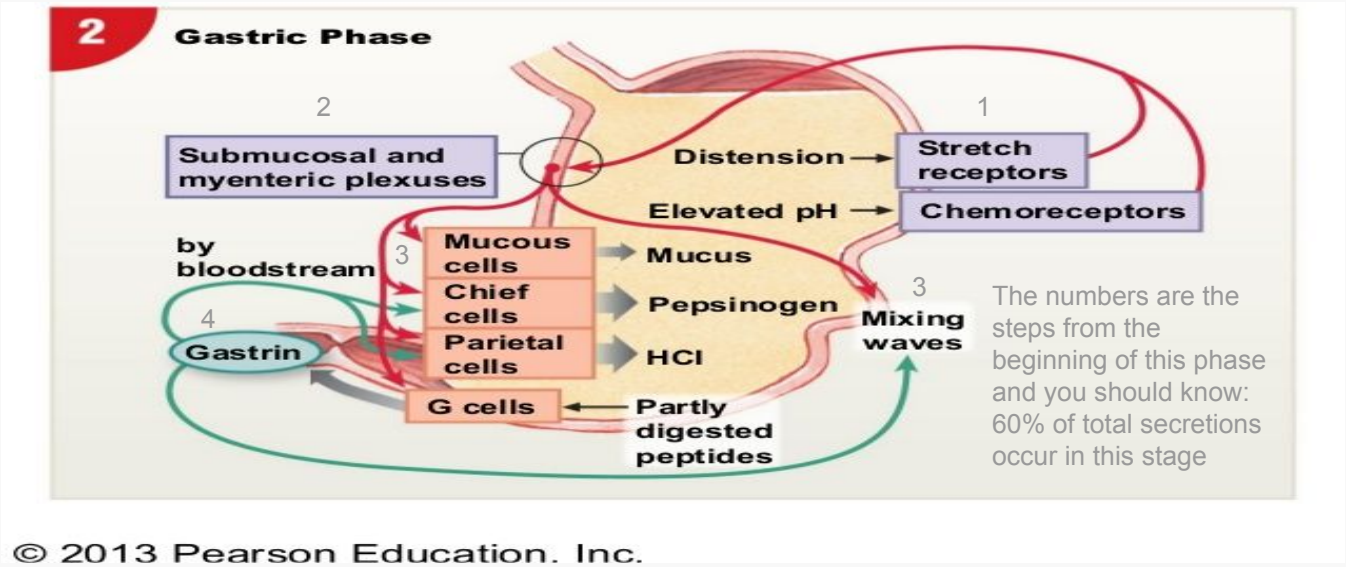
Before eating: you are seeing and smelling and tasting, or even just thinking of the food.



Guyton corner :

The cephalic phase of gastric secretion occurs even before food enters the stomach, especially while it is being eaten. It results from the sight, smell, thought, or taste of food, and the greater the appetite, the more intense is the stimulation. Neurogenic signals that cause the cephalic phase of gastric secretion originate in the cerebral cortex and in the appetite centers of the amygdala and hypothalamus. They are transmitted through the dorsal motor nuclei of the vagi and thence through the vagus nerves to the stomach. This phase of secretion normally accounts for about 30 percent of the gastric secretion associated with eating a meal

2) Gastric Phase: food is already in the stomach

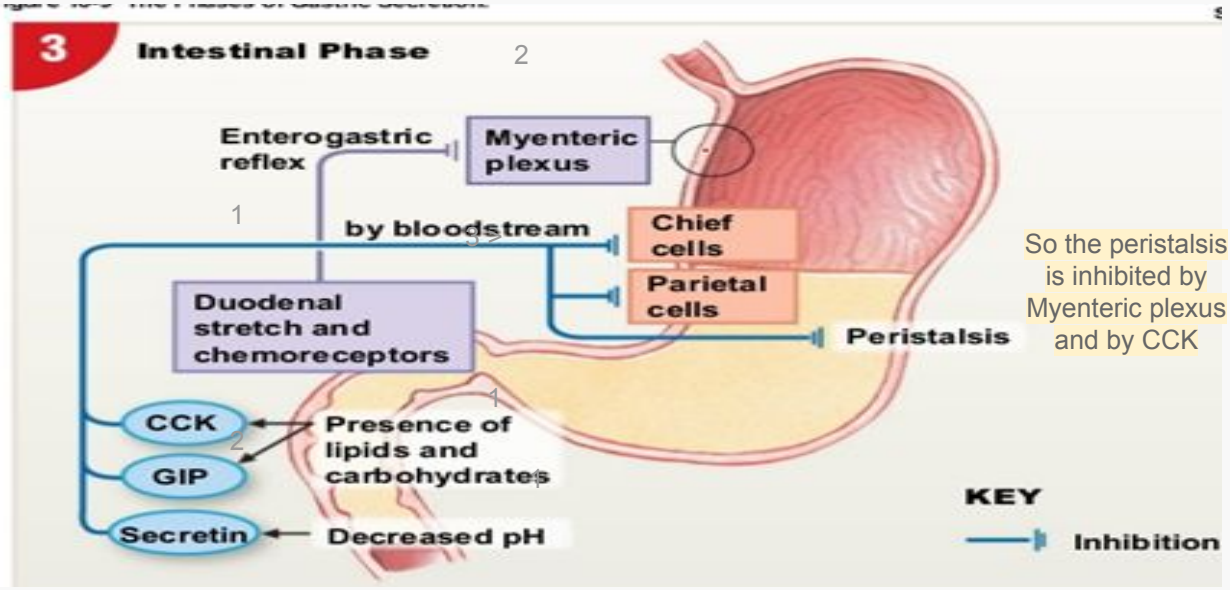


Guyton corner :

Gastric Phase: Once food enters the stomach, it excites (1) long vagovagal reflexes from the stomach to the brain and back to the stomach, (2) local enteric reflexes, and (3) the gastrin mechanism, all of which cause secretion of gastric juice during several hours while food remains in the stomach. The gastric phase of secretion accounts for about 60 percent of the total gastric secretion associated with eating a meal and therefore accounts for most of the total daily gastric secretion of about 1500 milliliter.

Phases of gastric secretion (cont.)

3) Intestinal Phase: mainly inhibitory (after digestion) the myenteric plexus inhibits the contraction. Also called enterogastric reflex.



So the peristalsis is inhibited by Myenteric plexus and by CCK

Guyton corner :

Intestinal Phase: The presence of food in the upper portion of the small intestine, particularly in the duodenum, will continue to cause stomach secretion of small amounts of gastric juice, probably partly because of small amounts of gastrin released by the duodenal mucosa. This secretion accounts for about 10 percent of the acid response to a meal.

Inhibition of Gastric Secretion by Other Intestinal Factors:
Although intestinal chyme slightly stimulates gastric secretion during the early intestinal phase of stomach secretion, it paradoxically inhibits gastric secretion at other times.This inhibition results from at least two influences:
1- Presence of food in the small intestine initiates a reverse enterogastric reflex (check first lecture)
2- Sence of acid, fat, protein breakdown products, hyperosmotic or hypo-osmotic fluids, or any irritating factor in the upper small intestine causes release of several intestinal hormones.
The purpose of intestinal factors that inhibit gastric secretion is presumably to slow passage of chyme from the stomach when the small intestine is already filled or already overactive.

Extra:

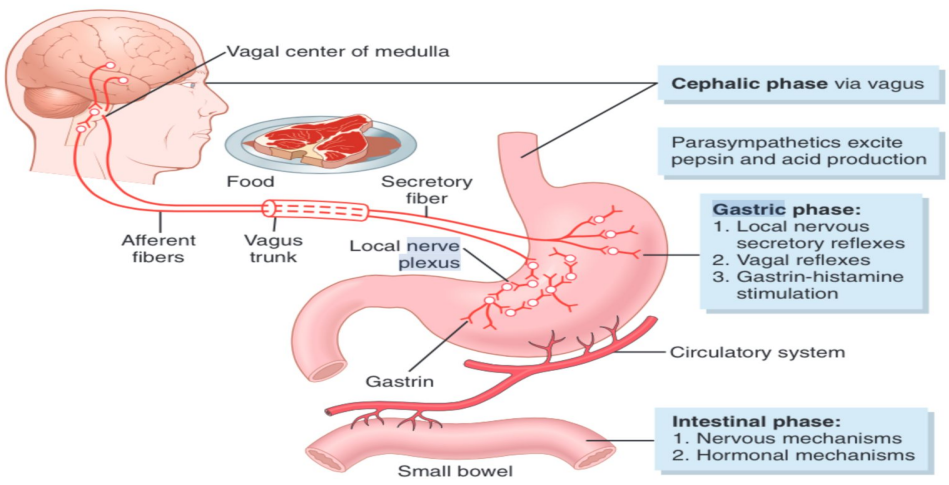
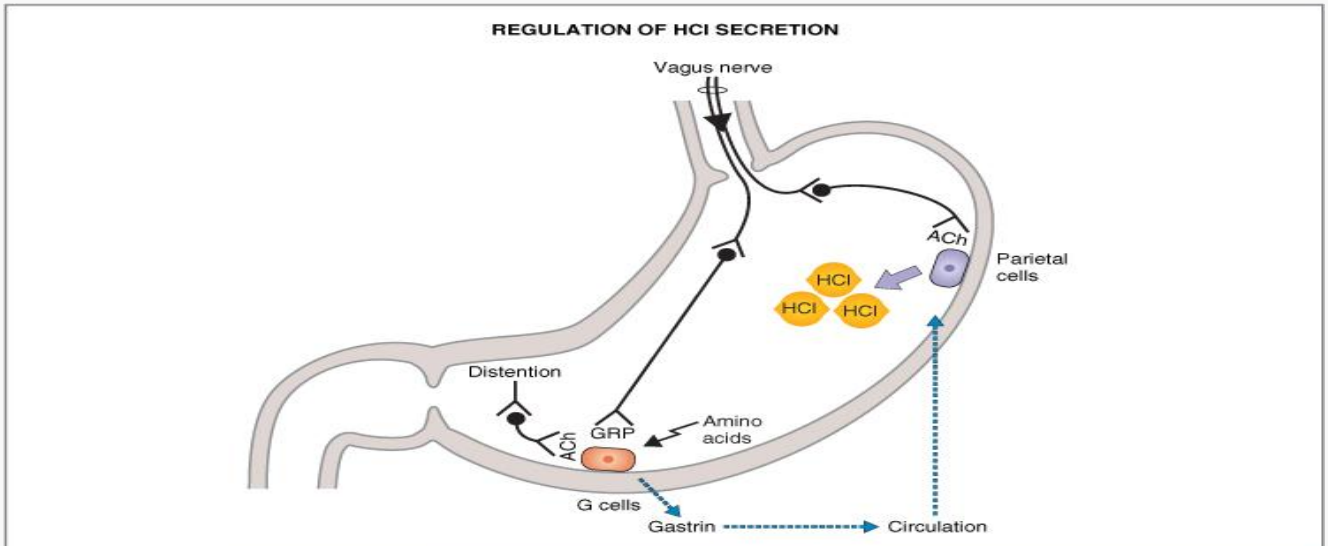


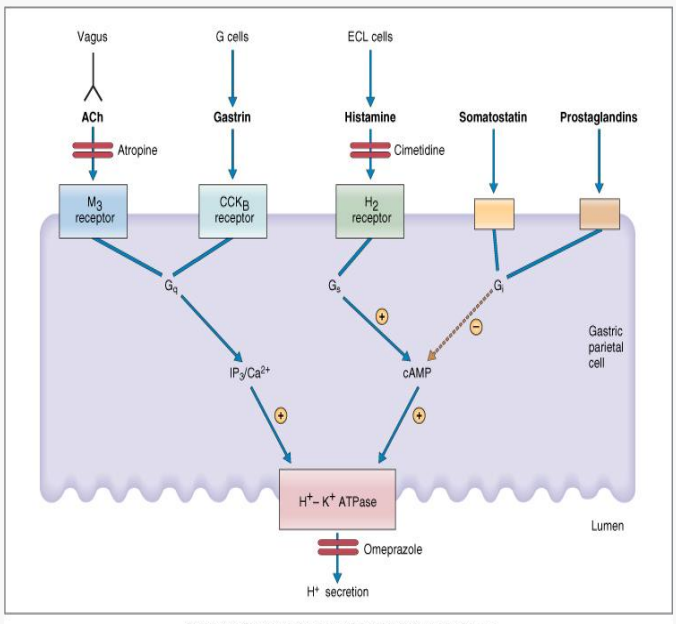
Figure 65-7. Phases of gastric secretion and their regulation.



Phase	% of HCl Secretion	Stimuli	Mechanisms
Cephalic	30%	Smell, taste, conditioning	Vagus → parietal cell Vagus → gastrin → parietal cell
Gastric	60%	Distention	Vagus → parietal cell Vagus → gastrin → parietal cell
		Distention of antrum	Local reflex → gastrin → parietal cell
		Amino acids, small peptides	Gastrin → parietal cell

Agents that stimulate and inhibit H^+ secretion by gastric parietal cells

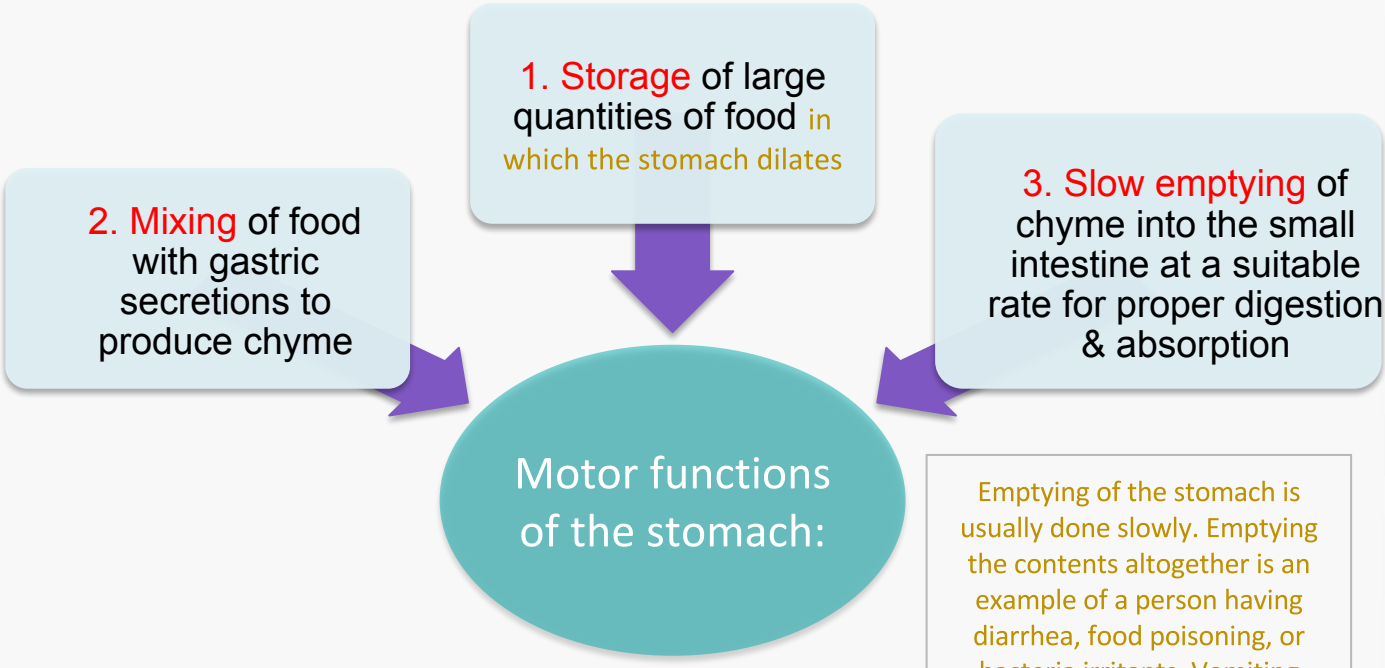
It's important to know the Omeprazole proton pump



Inhibitory hormones(Enterogastrones):

- **Somatostatin (D-cells) in antrum.**
- **Secretin (S-cells) in duodenum.**
- **Glucose-dependent insulinotropic peptide (GIP) in duodenum.**

You will find the same illustration explained in details in the 1st pharmacology lecture



Emptying of the stomach is usually done slowly. Emptying the contents altogether is an example of a person having diarrhea, food poisoning, or bacteria irritants. Vomiting happens when the toxins are very high in the body.

Storage function:

When food stretch the stomach a vagovagal reflex from the stomach to the brain stem Back to the stomach to reduce the tone in the muscular wall in the body of the stomach.

Stomach wall bulges progressively

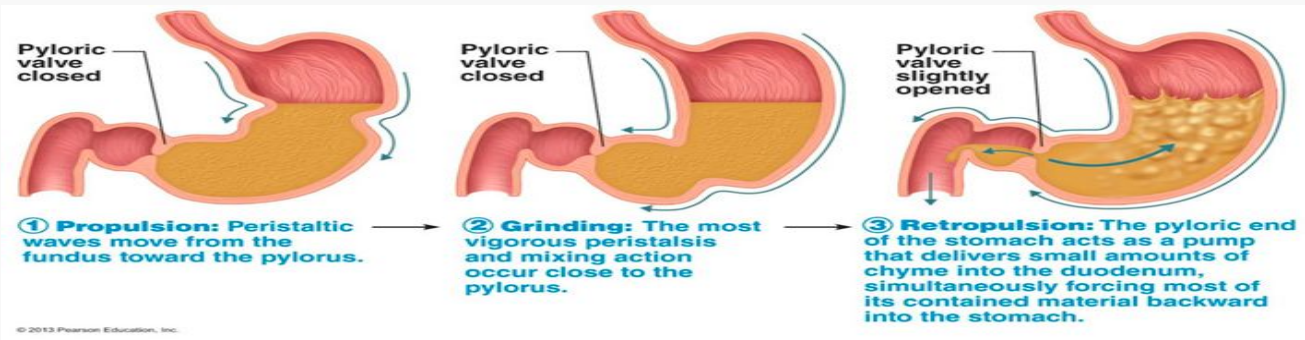
Stomach will accommodate up to 0.8-1.5 liters and pressure will remain low,may remain unmixed for 1 Hour in the corpus

Guyton corner : As food enters the stomach, it forms concentric circles of the food in the orad portion of the stomach, the **newest food** lying closest to the **esophageal opening** and the **oldest food** lying nearest the **outer wall of the stomach**. Normally, when food stretches the stomach, a **"vagovagal reflex"** from the stomach to the brain stem and then back to the stomach reduces the tone in the muscular wall of the body of the stomach so that the wall bulges progressively outward, accommodating greater and greater quantities of food up to a limit in the completely relaxed stomach of **0.8 to 1.5 liters**. **The pressure in the stomach remains low until this limit is approached.**

Mixing and propulsion function: (by the Antrum mainly)

Location	Result
constrictor ring in mid to upper portions of the stomach	• As long as food is in the stomach, **weak peristaltic constrictor waves (mixing waves)** begin in the mid to upper portions of the stomach wall and move toward the antrum once every 15-20 seconds or 3 times/min. "propulsion" • It is initiated by the gut wall basic electrical rhythm **(slow waves)**.
constrictor ring moving in antrum	• As the constrictor waves move into the antrum they become more intense. "grinding" • Some become extremely intense providing **peristaltic action potential-driven constrictor rings** that force antral contents **under higher pressure toward the pylorus**. • Motor behavior of the antral pump is initiated by a dominant pacemaker (ICC) & Gastric action potentials determine the duration and strength of the phasic contractions of the antral pump. • Electrical syncytial properties of the gastric musculature account for propagation of the action potentials from the pacemaker site to the gastroduodenal junction..

2. Mixing of food with gastric secretions:	3. Stomach emptying (Pyloric pump):
• Most of the time contractions are weak and cause mixing of food with gastric secretions "grinding & retropulsion"	• 20% of the time, contractions are in the form of tight ringlike constrictions that are 6 times as powerful as mixing waves, these cause stomach emptying. • Stomach emptying is promoted by intense peristaltic contractions in the antrum.



2. Mixing of food with gastric secretions:

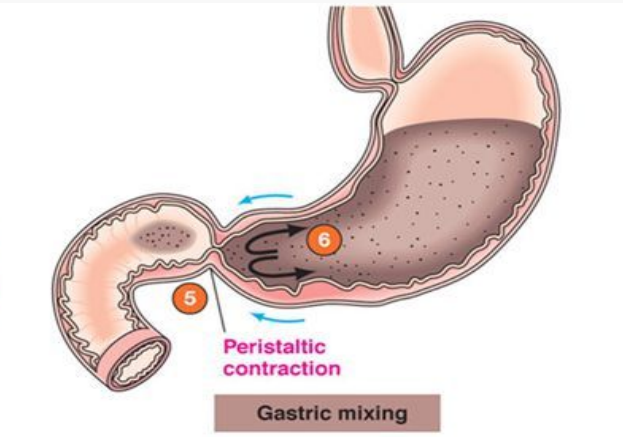
Constrictor rings play an important role in mixing the stomach contents:

- Each time, it digs deeply into the food contents in the antrum.
- The opening of the pylorus allows only a few mls of antral contents to be expelled into the duodenum with each wave.
- As each wave approaches the pylorus, the pyloric muscle contracts.
- Most of the antral content are squeezed **upstream** through the peristaltic ring toward the body
- The moving peristaltic ring (grinding) + upstream squeezing action (Retropulsion) is an important mixing mechanism

3. Stomach emptying (Pyloric pump):

Role of pylorus in controlling emptying:

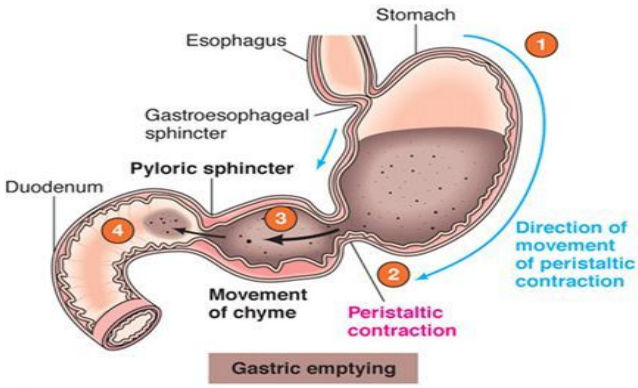
- Pylorus is the distal opening of the stomach, It is named the pyloric sphincter.
- Thickness of circular muscles is 50-100% greater than in the antrum.
- It is slightly tonically contracted almost all the time. Emptying is thus opposed by resistance to passage of chyme at the pylorus.
- It is usually open enough to allow water & fluids.
- **It is controlled by nervous and humoral reflexes from the stomach and duodenum.**



Gastric mixing

5 When the peristaltic contraction reaches the pyloric sphincter, the sphincter is tightly closed and no further emptying takes place.

6 When chyme that was being propelled forward hits the closed sphincter, it is tossed back into the antrum. Mixing of chyme is accomplished as chyme is propelled forward and tossed back into the antrum with each peristaltic contraction.



Gastric emptying

1 A peristaltic contraction originates in the upper fundus and sweeps down toward the pyloric sphincter.

2 The contraction becomes more vigorous as it reaches the thick-muscled antrum.

3 The strong antral peristaltic contraction propels the chyme forward.

4 A small portion of chyme is pushed through the partially open sphincter into the duodenum. The stronger the antral contraction, the more chyme is emptied with each contractile wave.

Hunger contractions:

- Occurs when stomach is empty for several hours
- They are rhythmic peristaltic contractions in the body of the stomach
- When successive contractions become extremely strong they fuse into a continuing tetanic contractions (these are painful) that lasts for 2-3 min
- Sometimes they cause mild pain (hunger pangs).
- They begin 12-24 hrs after last meal, and reach greatest intensity in starvation after 3-4 days.

Guyton corner: Hunger contractions are most intense in young, healthy people who have high degrees of gastrointestinal tonus; they are also greatly increased by the person's having lower than normal levels of blood sugar.

Regulation of stomach emptying:

1. Gastric factors:

***Effect of gastric food volume:** when volume is increased, it increases emptying due to stretch of stomach wall which initiate l**ocal myenteric reflexes** causing:

- Increase activity of pyloric pump
- Inhibit the pylorus (sphincter)

***Effect of Gastrin hormone** released from **antral** mucosa.

- Enhance the activity of pyloric pump.
- Promotes the secretion of gastric acidic juices (HCL) by stomach gastric glands(oxyntic glands)

2. Duodenal factors (more potent):

Digestion is deactivated in the duodenum due to change in the pH level

Factor:	Inhibitory enterogastric nervous reflexes	Hormonal feedback:
Stimulus	**Factors that initiate these reflexes are:** • **Duodenal distention** • **Irritation of mucosa** • **Acidity of duodenal chyme** • **Breakdown products such as proteins & fats.** • **Osmolality of the chyme**	**The main stimulus for releasing these inhibitory hormones is fat in the duodenum through receptors on epithelial cells**
Mediated by:	• **Directly from duodenum to stomach via enteric nervous system in the gut wall** • **Extrinsic nerves to sympathetic ganglia** • **Vagus nerves to the brain stem**	**Released hormones carried by blood to the stomach, they include:** • **The most potent hormone, CCK released from jejunum by fat** • **Secretin released from duodenal mucosa in response to acid** • **Gastric inhibitory peptide (GIP) from upper small intestine mainly by fat in chyme and carbohydrates**
Effect	**All these reflexes strongly:** (in contrast to gastric factors) • **inhibit pyloric pump** (emptying) • **increase tone (contraction) of the pyloric sphincter** **why? When the small intestines wants to deal with the fluid passing by, it slows down the movement.**	

Guyton corner : **Summary of the control of stomach emptying.**
Emptying of the stomach is controlled only to a moderate degree by stomach factors such as the degree of filling in the stomach and the excitatory effect of gastrin on stomach peristalsis. Probably the more important control of stomach emptying resides in inhibitory feedback signals from the duodenum, including both enterogastric inhibitory nervous feedback reflexes and hormonal feedback by CCK. These feedback inhibitory mechanisms work together to slow the rate of emptying when (1) too much chyme is already in the small intestine or (2) the chyme is excessively acidic, contains too much unprocessed protein or fat, is hypotonic or hypertonic, or is irritating.

Digestion :

Digestion of Carbohydrates in stomach and mouth:

Saliva contains **ptyalin (an α amylase)** which is secreted by parotid gland → mixed with food → hydrolyses **starch to maltose** → It continues in stomach for 1 hr only due to its inactivation by gastric acid.

Digestion of proteins in stomach:

Pepsin	Hydrochloric acid (HCl)
• **Secreted by chief (peptic) cells** • **It is active at pH 2-3 (e.g. stomach) and inactive at pH 5 (e.g. intestine)** • **Initiate protein digestion (10-20% of protein digestion),including collagen.**	• **secreted by parietal (oxyntic) cells.** • without HCl, pepsinogen won't be activated into pepsin • Note that parietal cells don't secrete HCl combined. They secrete H+ and Cl separately which then combine in the lumen away from the cells, to prevent the damage of mucosal membrane by HCl (mucus also helps in mucosal lining protection).

Absorption :

Stomach is a poor absorptive area of GIT, due to:

- **It lacks the villous type of absorptive membrane.**
- **It has tight junctions between epithelial cells.**
- **Only a few highly-lipid soluble substances can be absorbed such as Alcohol and Aspirin.**

SUMMARY

Type of glands	oxyntic	pyloric	Cardiac glands
Special secretion	HCl- Intrinsic factor	Gastrin	
General secretion	Pepsinogen - mucus		
Location	Body and fundus 80%	Antrum 20%	

Function of stomach		
storage	**Mixing of food with gastric secretions**	**Pyloric pump(stomach emptying)**
-Stomach will accommodate up to 0.8-1.5 liters . -Stretch receptors in stomach initiate the vagovagal reflexes.	Three stages: 1.**propulsion**: from fundus toward the pylorus. 2.**grinding**: mixing action occur near to pylorus. 3.**retropulsion**: small amount of chyme move to the duodenum.	controlled by nervous and humoral reflexes from the stomach and duodenum by their effect on pyloric sphincter

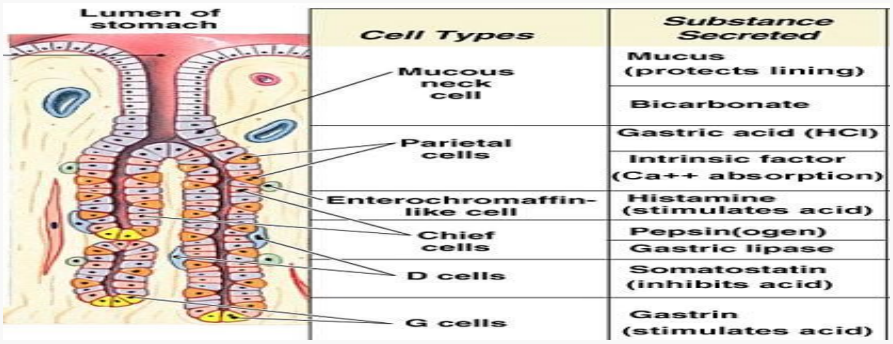
Regulation of stomach emptying			
	Gastric factors	**Duodenal factors**	
Factors	*** gastric food volume.** ***Gastrin hormone**	***Inhibitory enterogastric nervous reflexes:** **Mediated by :** **-ENS.** **-Extrinsic nervous system** **-vagus nerve.**	**Hormonal feedback:** **-Stimulated by fat in duodenum** **-mediated by release hormones:** **CCk:released from jejunum by fat.** **Secretin:released from duodenal mucosa in response to acid** **GIP:from upper small intestine mainly by fat in chyme and carbohydrates**
Effect	Increase activity of pyloric pump	-inhibit pyloric pump (emptying) - increase tone (contraction) of the pyloric sphincter	

SUMMARY

<table>
<tr><td>Gastric juices :</td><td colspan="3">HCL ,Pepsinogen, Electrolytes, Intrinsic factor, Mucus (mucus gel layer)</td></tr>
<tr><td rowspan="4">Gastric secretion phases :</td><td colspan="2">Neural pathway</td><td>hormonal pathway</td></tr>
<tr><td colspan="2">• By stmulation of the vagus .N :
• either by releasing Ach (direct activation of parietal cells) or by releasing Gastrin releasing peptide, GRP (indirect activation).</td><td>• Gastrin (hormonal effector)
• Enterochromaffin-like cells release Histamine activates H_2 receptor (parietal cells) that will increases acid secretion</td></tr>
<tr><td>1-Cephalic phase(30%)</td><td>2-Gastric phase (60%)</td><td>3-Intestinal phase (10%)</td></tr>
<tr><td>Smelling, Chewing and swallowing
Stimulate parietal G-Cells (via GRP)</td><td>gastric distention
proteins</td><td>digested proteins</td></tr>
<tr><td colspan="3">Cimetidine (H_2 receptor blocker)⟶ ↓ peptic ulcer and gastroesophageal reflux by lowering gastric secretions .</td></tr>
<tr><td rowspan="2">Composition of Gastric Juice & the secretion rate</td><td>(low) / Dereasing of secretion rate result in</td><td colspan="2">(high) / Increasing of secretion rate result in</td></tr>
<tr><td>↑Na & Cl concentration
↓ H & K concentration</td><td colspan="2">↑ significant concentration of H
↑ Cl concentration
↓ Na concentration</td></tr>
<tr><td>HCL Secretion</td><td colspan="3">• Depends on H/K ATPase
• - H/K pump depends on $[K]_{out}$
• [HCl] drives water into gastric content to maintain osmolality
• amount of HCO_3^- in blood = amount of HCl being secreted
• Controlled by through neural(vagus .N) and hormonal pathways (gastrin & special H_2 receptors)
• Alkaline tide</td></tr>
<tr><td>Inhibition of Acid Secretion</td><td colspan="3">• <u>Inhibitory hormones (Enterogastrones):</u>
• <u>Somatostatin</u> (D-cells) in antrum
• <u>Secretin</u> (S-cells) in duodenum
• <u>Glucose-dependent insulinotropic peptide</u> (GIP) in duodenum</td></tr>
</table>

SUMMARY

Hormone	Site of Secretion	Stimuli for Secretion	Actions
Gastrin	G cells of the stomach	Small peptides and amino acids Distention of the stomach Vagal stimulation (GRP)	↑ Gastric H^+ secretion Stimulates growth of gastric mucosa
Cholecystokinin (CCK)	I cells of the duodenum and jejunum	Small peptides and amino acids Fatty acids	↑ Pancreatic enzyme secretion ↑ Pancreatic HCO_3^- secretion Stimulates contraction of the gallbladder and relaxation of the sphincter of Oddi Stimulates growth of the exocrine pancreas and gallbladder Inhibits gastric emptying
Secretin	S cells of the duodenum	H^+ in the duodenum Fatty acids in the duodenum	↑ Pancreatic HCO_3^- secretion ↑ Biliary HCO_3^- secretion ↓ Gastric H^+ secretion Inhibits trophic effect of gastrin on gastric mucosa
Glucose-Dependent Insulinotropic Peptide (GIP)	K cells of the Duodenum and jejunum	Fatty acids Amino acids Oral glucose	↑ Insulin secretion from pancreatic β cells ↓ Gastric H^+ secretion
Motilin	M cells of the duodenum and jejunum	Fat Acid Nerve	Stimulates: Gastric motility Intestinal motility



D cells have the same effect as prostaglandins.

MCQs

Q1\ Which one of the following cells is responsible for the secretion of HCL and Intrinsic factor.

A. Mucus cells.
B. Parietal cells
C. G cells.
D. Peptic (chief) cells.

Q2\ in rule of stomach emptying , gastric factors facilitate the emptying if there is :

A. Duodenal distention.
B. Disturbed the balance of chyme's osmolality.
C. Increased gastric food volume.
D. Mucosal irritation.

Q3\ in case of hormonal feedback, CCk, secretin, and gastric inhibitory peptide released . the result will be:

A. Inhibition of pyloric sphincter.
B. Facilitation of pyloric pump.
C. Increase gastric secretion.
D. Strongly contraction of pyloric sphincter.

Q4\ Mechanism of HCl production depend on :

A. H/K ATPase
B. Na/K ATPase pump
C. Both A & B
D. None of above

Q5\ Which of gastric secretion involve distention of the stomach through vagovagal reflexes :

A. Cephalic phase
B. Gastric phase
C. Intestinal phase

Q6\ Which cells in the antrum secrete Somatostatin :

A. D-cells
B. S-cells
C. M cells
D. Parietal cells

answer key: 1.B, 2.C, 3.D, 4.C, 5.B, 6.A .

خولة العمَّاري
نجود الحيدري
نورة الطويل
لولوة الصغير
لجين السواط
رزان السبتي
ربى السليمي
ديما الفارس
خولة العريني
ملاك الشريف
منيرة الحسيني
مروج الحربي
أفنان المالكي
دلال الحزيمي
رناد القحطاني
سارة الخليفة
فرح مندوزا
مي العقيل
نورة الخراز
سارة الخليفة
نورة الخيال
رغد النفيسة
منيرة السلولي
نوف العبدالكريم
سها العنزي
نورة القحطاني

عمر آل سليمان
عبدالعزيز الحماد
عبدالرحمن السياري
محمد أبونيان
عبدالرحمن البركه
إبراهيم النفيسه
محمد البشر
عمر العتيبي
حمزة الفعر
عبدالله الجعفر
عبدالله الضحيان
حسن البلادي
حسن الشماسي
عبدالله الضبيب
محمد الفواز
محمد السحيباني
وائل العود
رواف الرواف
عمر الشهري