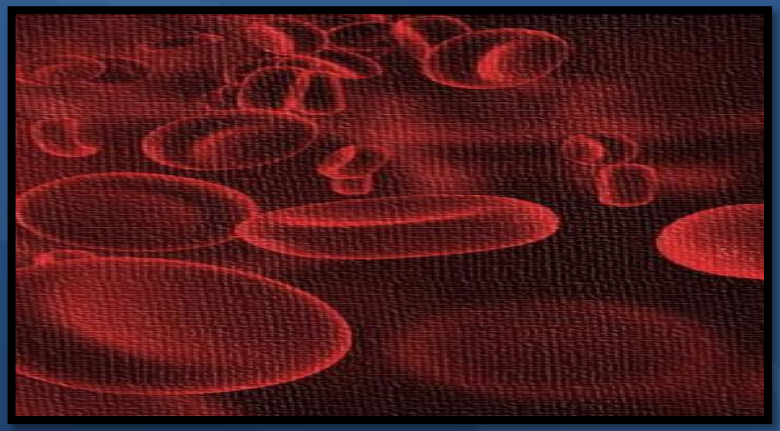
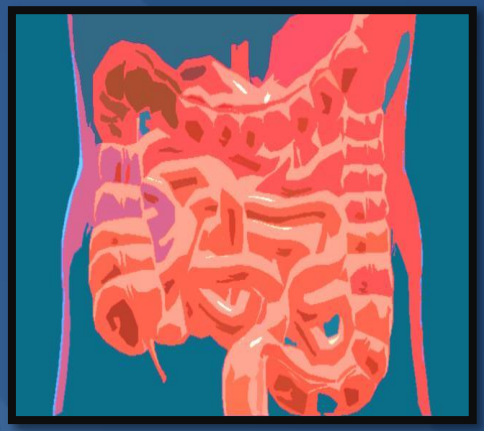
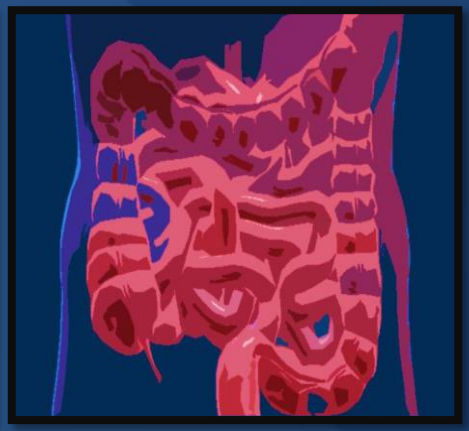
Physiology Team

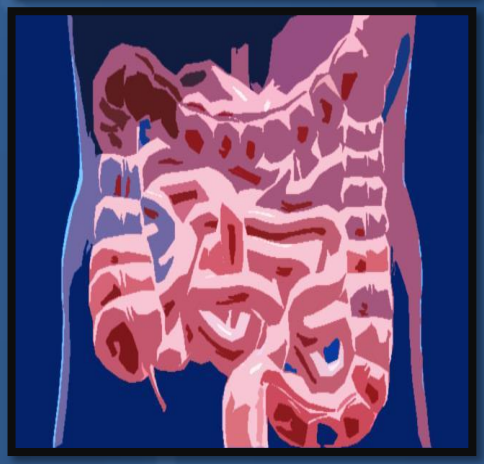
LECTURE 4

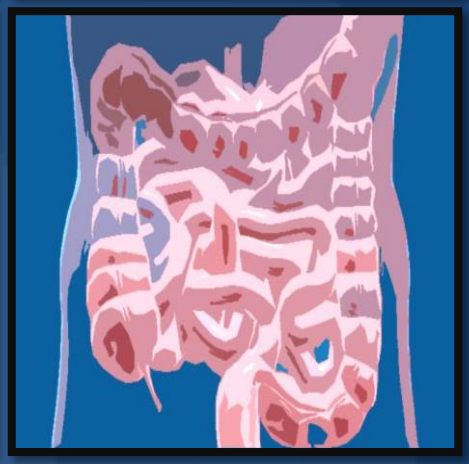
Physiology of the Stomach and Regulation of Gastric Secretions











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GASTOINTESTINAL TRACT

OBJECTIVES

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At the end of this lecture, student should be able to describe:

- Functions of stomach.
- Gastric secretion
- Mechanism of HCl formation.
- Gastric digestive enzymes.
- Neural and hormonal control of gastric secretion.
- Phases of gastric secretion.
- Motor functions of stomach.
- Stomach emptying
- Gastric factors that promote stomach emptying.
- Duodenal factors that inhibit stomach emptying.

DON'T WORRY ABOUT THE # OF THE SLIDS !
..
THERE ARE 5 SLIDES FOR SUMMARY
&
3 SLIDES FOR QUESTIONS
☺

MIND MAP

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Function of the Stomach

Motor

- Reservoir part and it's Reflexes
- Antral pump part
- Action potential of the stomach
- Stomach empting

Secretion

- Phases of Gastric secretion
- HCL Formation
- Gastric enzymes
- Control of Secretion

Anatomical and physiological divisions of the stomach

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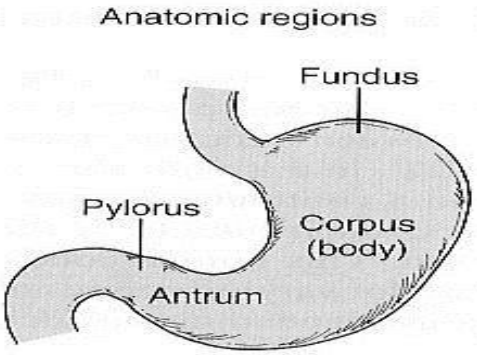
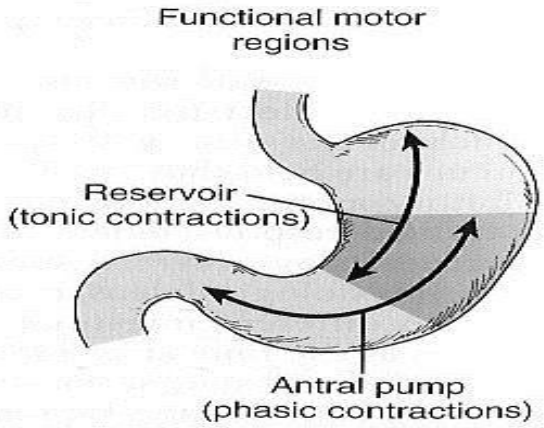
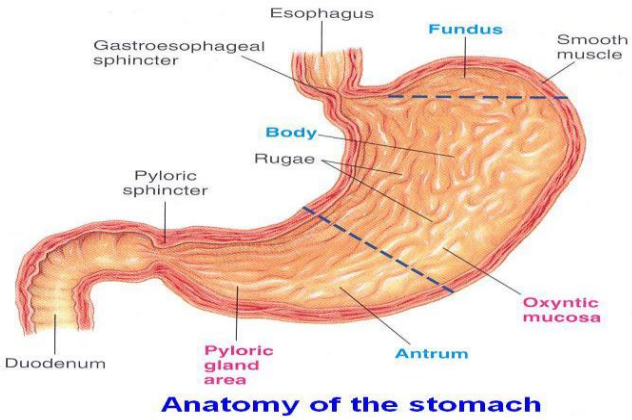
Anatomically the stomach is composed of the fundus, body and the antrum.

Physiologically it is composed of:

- The orad portion (fundus and upper two thirds of the body – proximal region of stomach) considered as Reservoir "storage" part and undergo tonic contraction.
- The caudal (lower third of the body plus antrum – distal region)- considered as Antral pump and undergo phasic contraction.







Anatomy of the stomach

Slides | Important | Females' Notes | Explanation | Males' Notes

Functions of stomach:

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1- It stores food as it is the most dilated part of GIT & regulates its passage to small intestine after being partly digested and mixed with gastric juice.
2- It secretes juice that liquefies & partly digests food.
3- It produces intrinsic factor necessary for vitamin B12 absorption.
4- Gastric HCl which has several functions:
- ✓Kills ingested bacteria because it provides highly acidic medium.
- ✓Is necessary for iron * & Ca++ ** absorption.
- ✓Catalyzes cleavage of inactive pepsinogen into active pepsin.

7- Absorption of water and lipid-soluble substances (alcohol and drugs)
The main side of absorption in the GIT is the small intestines
8- It has endocrine function, e.g. It produces gastrin and somatostatin.

* Iron ingested from food in the form of ferric which must be converted into ferrous by the help of HCl to be absorbed.
** Ca++ absorption rate is more in acidic medium while in alkaline medium it will precipitate and form salt.

Histologically gastric mucosa is divided into 3 areas:-

1- The cardiac area (10 % of mucosa):

- At gastroesophageal junction and contain mucous cells only.
- Most of cells secrete mucus.

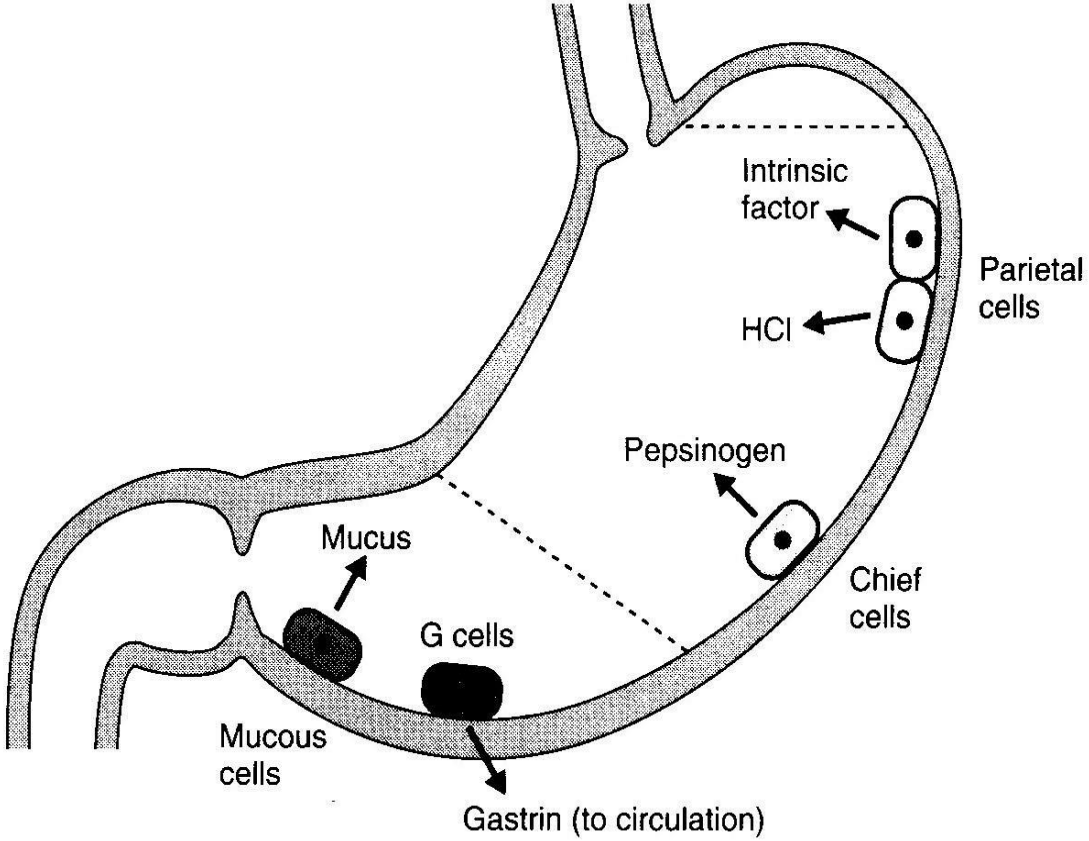
2- The main gastric area (70-80 %):

- Includes mucosa of fundus & body.
- Its oxyntic glands secrete all constituents of gastric juice.
  - Parietal (oxyntic) cells secrete HCl & intrinsic factor.
  - Peptic (chief) cells secrete pepsinogen.
  - Mucous neck cells secrete mucus & HCO3-.

3- The pyloric area (15 %):

- Most of its cells are mucous cells. Contains G- cells that secrete gastrin.

Normal locations of gastric cells:



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

Gastric Juice:

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Volume about 2-3 L/day

Main constituents are:

1. HCl
2. Digestive enzymes (Pepsinogen)
3. Mucus (mucus gel layer)
4. Electrolytes
5. Intrinsic factor

- Highly acidic – pH can be 0,8
- All secretions are isotonic except saliva which is hypotonic.

Gastric HCL:

- Secreted by parietal cells.
- They are pyramidal in shape.
- Their structure is unique in that they have an abundance of mitochondria* and intracellular canaliculi "spaces between microvilli" that are continuous with the lumen of the oxyntic gland.
- HCl is secreted across the parietal cell microvillar membrane and flows out of the intracellular canaliculi into the oxyntic gland lumen.

*so they are very active cells as they have important role to concentrate H+ thousands times in order to form HCL .

Mechanism of HCL formation

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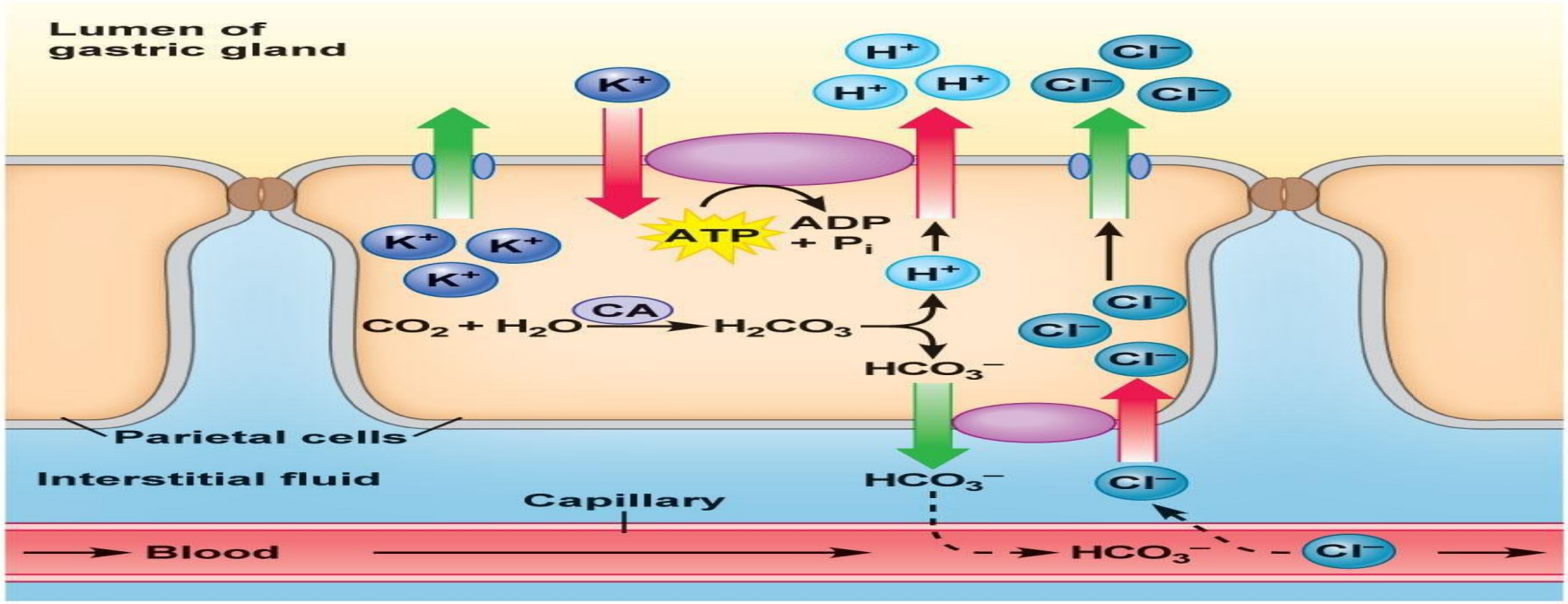

A very helpful animation demonstrates the formation of gastric HCL and we strongly advise you to have a look before reading the next slide ^_^

Just click on

Formation of Gastric HCL

Mechanism of HCL formation

Important

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Source of Cl- :

- *Cl- is actively transported from cytoplasm into luminal canaliculi. This create –ve potential which causes passive diffusion by electrical gradient of K+ from cytoplasm into canaliculi. Thus K+ & Cl- enters canaliculi. Now lumen contain K+ and Cl-

Sources of H+ :

1. Intracellular H2O dissociates into H+ & OH-.

- H+ is actively transported across canalicular membrane against concentration gradient by H+-K+ ATPase which exchange H+ with K+. It can be inhibited by omeprazole (proton pump inhibitors).

2. intracellular CO2, either formed during metabolism in the cell or entering the cell from the blood, combines under the influence of carbonic anhydrase with the OH- to form HCO3-.

- HCO3- diffuses from the cell to plasma (Alkaline tide) and *CL- enters the cytoplasm via a carrier mechanism that facilitates exchange between the 2 ions.

Control of HCL secretion at the level of parietal cells:

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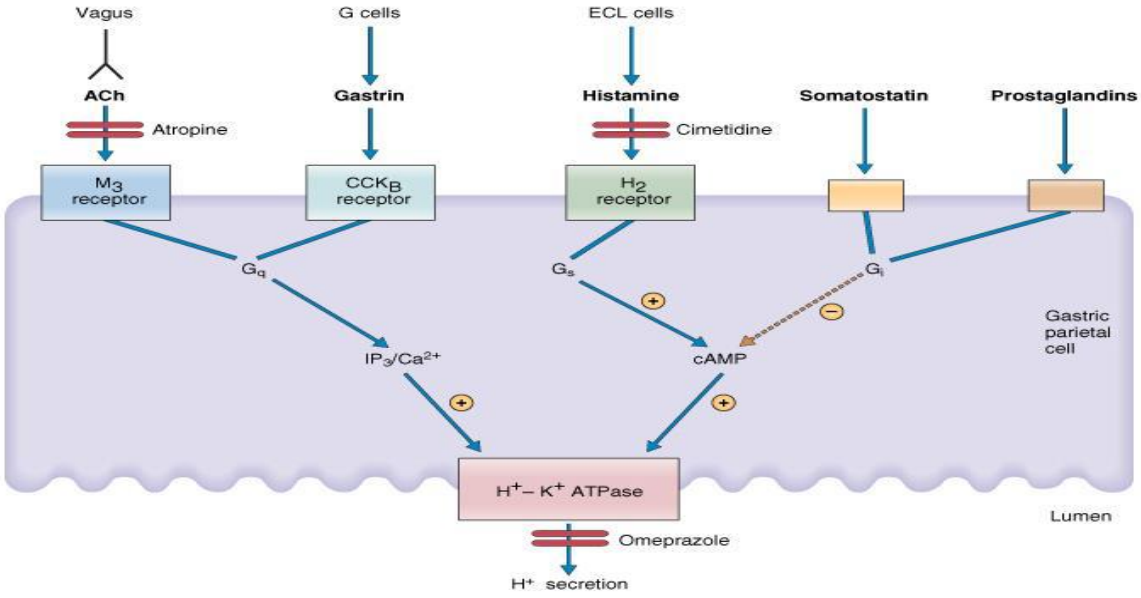
- **Vagus nerve**: or Ach released from nerve ending increases acid secretion. Either direct activation of parietal cells or by releasing Gastrin releasing peptide, GRP (indirect activation). (neural effector)
- **Gastrin** (hormonal effector): increases acid secretion.
- **Histamine**: Enterochromaffin-like cells release Histamine→activates H_2 receptor (parietal cells) → increases acid secretion.
- somatostatin and prostaglandins: are both inhibit H+ secretion by gastric parietal cells



Gastric digestive enzymes (pepsin and lipase)

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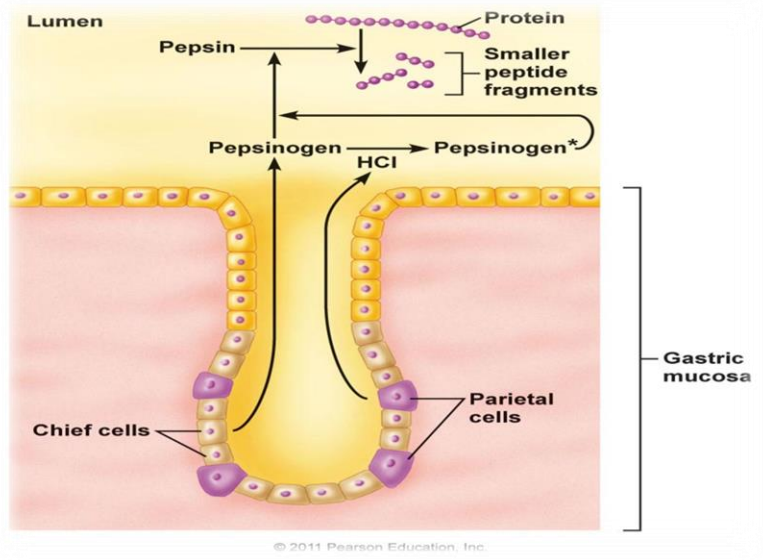
Pepsin enzyme

- Several types of pepsinogen secreted from chief cells. They are activated by HCl into pepsin and once activated, they can activate more pepsinogen "autoactivation = autocatalytic".
- The optimum pH is 1.5-3.5.
- Pepsin breaks down proteins into peptones & polypeptides "proteins of shorter chain".
- Pepsinogen secretion is stimulated by Ach "vagal stimulation", acid, hormones like gastrin, secretin & CCK.



Lipase enzyme

- Secreted from fundic mucosa. It hydrolyses TAG into MG (monoacylglycerol) & FA (Fatty Acid).
- Its activity is less than pancreatic lipase

Gastric digestive enzymes (pepsin and lipase)

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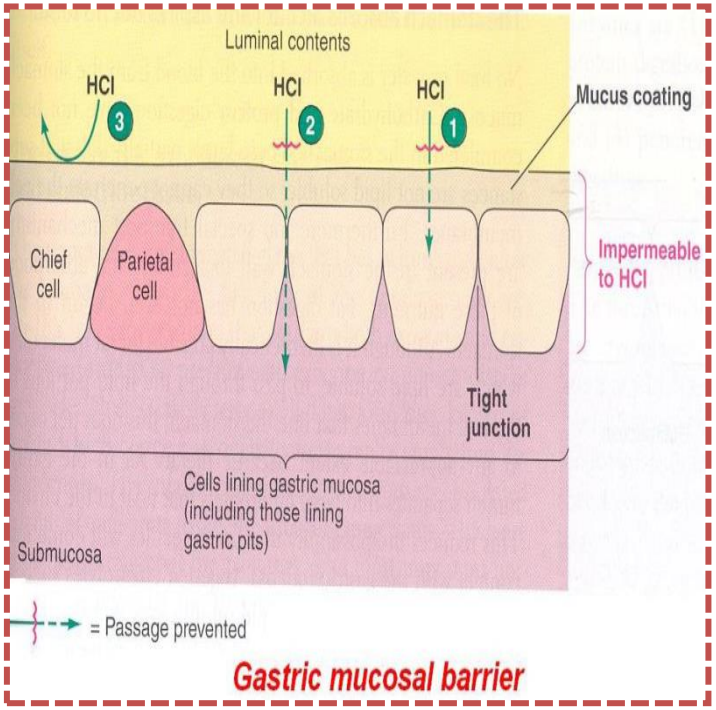
Gastric mucus:

- It is glycoprotein. Its secretion is stimulated by mechanical & chemical irritation of mucosa.
- It is about 0.2 mm thick and separate surface epithelial cells from acidic contents thus it allows neutral pH at epithelial cells despite luminal pH about 2. to protect luminal surface of gastric mucosa.

Functions of gastric mucus:

1. It protects the mucosa against mechanical injury by lubricating the chyme.
2. It protects the mucosa against chemical injury by acting together with HCO3- as a barrier to HCl & pepsin. It also neutralize HCl and arrest action of pepsin.

→ Aspirin (NSAIDs): inhibit secretion of both mucus and HCO3-, Prolonged use of these drugs may produce gastritis or ulcer.



Gastric mucosal barrier

Intrinsic factor:

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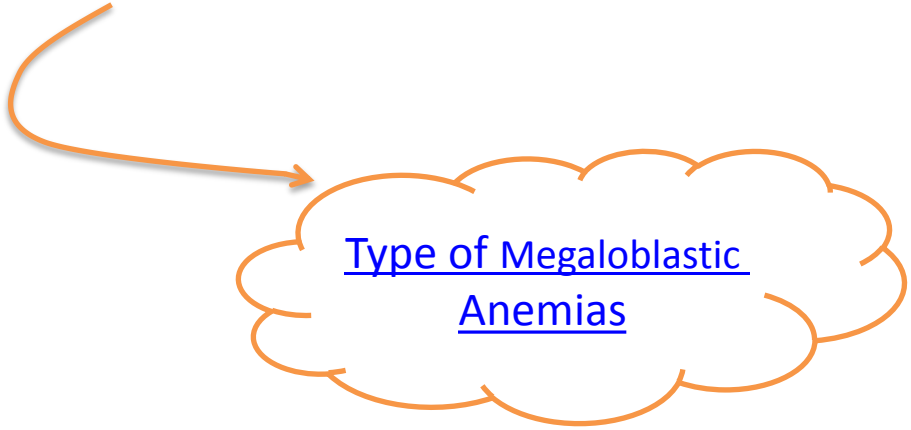
- It is glycoprotein secreted by parietal cells.
- It is the only essential function of stomach other function of stomach can be compensated as it is essential for vitamin B12 absorption.
- Atrophy of gastric mucosa leads to pernicious anemia



Gastric secretion occurs in three phases:

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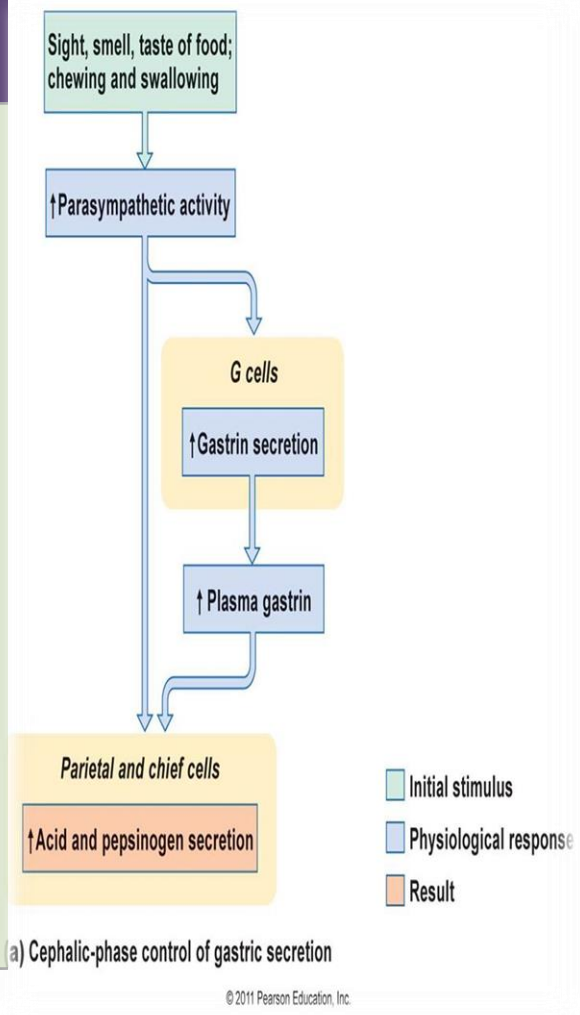
Phase	% of HCL section	Stimuli	mechanism
1- Cephalic phase	30%	• **Excitatory** • Conditioning • Seeing • Smelling, • chewing • swallowing food	By Vagal nerve (Ach) afferent impulses to *vagal nucleus* which sends impulses via the vagus nerves to parietal and "G" cells in the stomach. • **direct:** The nerve endings release ACh, which directly stimulates acid secretion from parietal cells. • **Indirect** : The vagal nerves also release gastrin-releasing peptide (GRP), which stimulates "G" cells to release gastrin, indirectly stimulating parietal cell acid secretion.



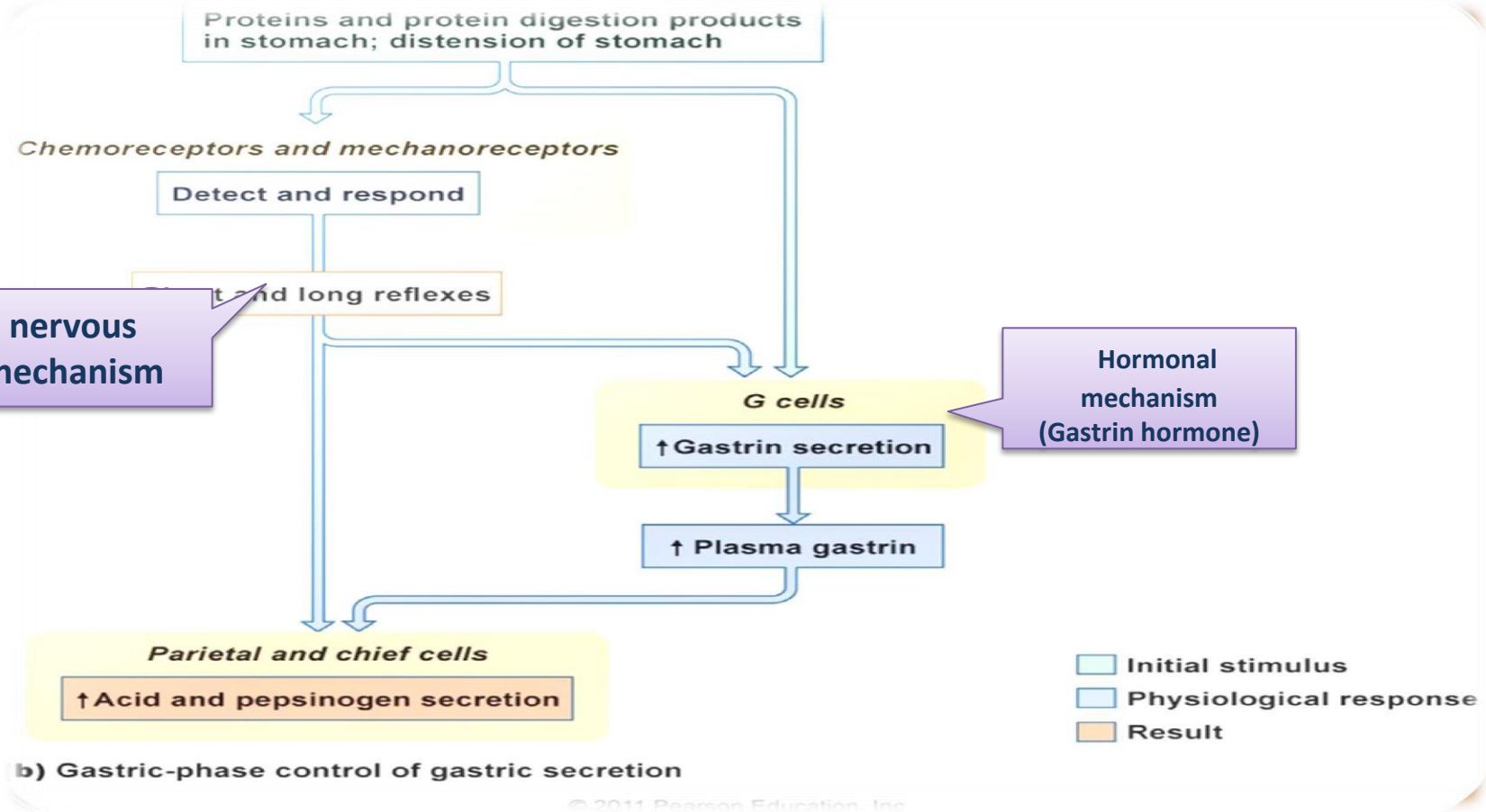
Gastric secretion occurs in three phases:

Physiology Team

Phase	% of HCL section	Stimuli	mechanism

2-gastric phase	60%	• **excitatory**
		• It is elicited by distention and presence of food (amino acid and small peptide = **digested proteins**) in stomach.
		• Several other chemicals, such as alcohol and caffeine, stimulate gastric acid secretion through mechanisms that are not well understood.
			It is mediated by nervous & hormonal mechanisms.
			• **nervous mechanism :** Distension of either body or antrum of stomach stimulates mechanoreceptors in gastric wall. which stimulate the parietal cells directly through:
			  • long vagovagal reflex
			  • short intramural cholinergic reflexes (ENS)
			• **Hormonal mechanism (Gastrin hormone) :** Gastrin is secreted from "G" cells in antrum, enters the blood and then stimulates gastric glands.

Gastric secretion occurs in three phases:



Gastrin hormone:

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Stimuli of gastrin release:

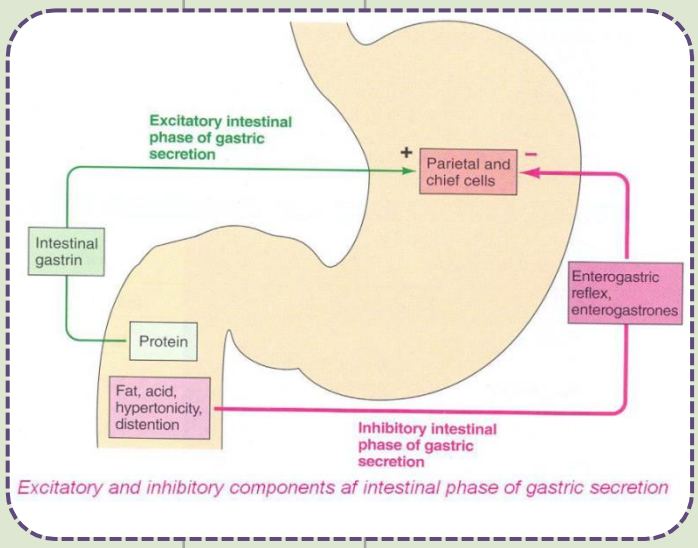
1- The presence of amino acids & peptides. (in gastric phase)
2- Gastric distension, (in gastric phase)
3- Vagal excitation. (in cephalic phase)
4- Rising of pH of gastric juice. (alkaline stimulate gastrein which stimulate parietal cells to secret HCL to decrease PH)
5- Alcohol & caffeine

Actions of gastrin: (it stimulate all secretions and motilities)
1- It stimulates gastric acid secretion, secretion of pepsin and intrinsic factor.
2- It stimulates intestinal secretion.
3- It stimulates pancreatic secretion of enzyme & HCO3-.
4- It stimulates biliary secretion of HCO3- & H2O.
5- It stimulates gastric motility.
6- It stimulates intestinal motility & relaxes ileocaecal sphincter.
7- It contract LES.
8- It has trophic effect on gastric mucosa. (growth and development of gastric mucosa)

Gastric secretion occurs in three phases:

Physiology Team

Phase	% of HCL section	Stimuli	mechanism
3-Intestinal	10%	• Excitatory at the beginning then inhibitory. • Duodenal distension • Amino acids • small peptide	The presence of chyme in duodenum causes neural & hormonal responses that first stimulates & later inhibits gastric acid secretion. **1-excitatory:** secretion is enhanced by:- A. Distension of duodenum stimulates G.A.(gastric acid) secretion by means of : ▪ Neural : vagovagal reflex ▪ Hormonal : the release of the hormone entero-oxyntin from intestinal endocrine cells that stimulates parietal & G- cells. B. Presence of protein digestive products ▪ In duodenum stimulates G- cells in duodenum & proximal jejunum to release gastrin. **2- the inhibitory mechainsm which limit G.A secretion :** A. The presence of food in small intestine initiates enterogastric reflexs (LAST SLIDE), transmitted through ENS (enteric or intrensic nervous system)& autonomic NS (extrinsic nervous system) that inhibits G.A secretion. B. Drop the pH in pyloric antrum to < 2.5 reduces G.A secretion via release of somatostatin from antral & duodenal "D" cells. C. The presence of acid, fat, protein digestive products, hypertonic solution in upper intestine inhibits G.A secretion. These effects are mediated mainly by hormonal mechanisms.



Excitatory and inhibitory components af intestinal phase of gastric secretion

Slides | Important | Females' Notes | Explanation | Males' Notes

Enterogastrones hormone:

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Are Inhibitory hormones released from intestine and affect Gastric Acid secretion as:-

1. Bulbogastrone
2. Gastric inhibitory peptide (GIP)) in duodenum.
3. Secretin (from S-cells) in duodenum
4. CCK (cholecyctokinin) chloe =bile → it inhibit gastric acid secretion and stimulate bile and pancreatic secretions)
5. Pancreatic glucagone.
6. Other peptides as VIP, somatostatin (from D-cells) in antrum and certain types of prostaglandins.

The functional purpose of the inhibition of G.A secretion by intestinal factors is to slow the release of chyme from stomach when the small intestine is already filled.

Motor functions of the Stomach:

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1- Motor behavior of the upper part of the Stomach (reservoir part):

The main functions of the upper part of the stomach:

1-To maintain a continuous compression (tonic contraction).

2-To accommodate the received food without significant gastric wall distention or pressure (Storage of food).

The stomach can store 0.8-1.5 L of food.

- **Chyme: Is a murky semi-fluid or paste composed of food that is thoroughly mixed with gastric secretions** (in the antrum we call the food bolus "chyme")

Relaxation reflexes in gastric reservoir part :

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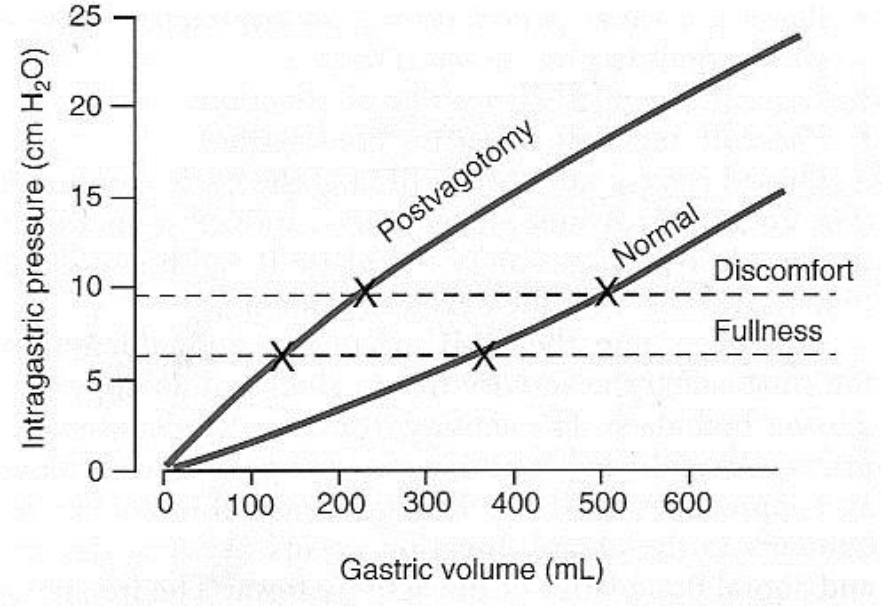
Three Kinds of Relaxation Occur in the Gastric Reservoir:

A- Receptive Relaxation Reflex:	B- Adaptive relaxation:	C- Feedback Relaxation:
• Triggered by **swallowing reflex.** • When the esophageal peristaltic waves reach the stomach, a **vagovagal reflex** is initiated from the stomach to the brain stem and back to the muscular wall of the stomach resulting in reduction in muscular wall tone and the stomach relaxes through **inhibition of myenteric neurons.** which prepares the stomach to receive the food that is propelled into the esophagus during swallowing.	• Triggered by **stretch receptors** (**vago-vagal reflex**). • 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 gastric muscular wall so that the wall bulges outward, accommodating greater quantities of food up to a limit (0.8 to 1.5 L). • **This reflex is lost in vagotomy**.	•The **presence of nutrients in the small intestine** triggers feedback relaxation. •It can involve both local reflex connections between receptors in the small intestine and the gastric ENS or hormones that are released from endocrine cells in the small intestinal mucosa and signal the gastric ENS and stimulate firing in vagal afferent terminals in the stomach.

Effect of Vagotomy:

- **Adaptive relaxation** is lost in patients who have undergone a **vagotomy**. Following a vagotomy, increased tone in the musculature of the reservoir decreases the wall compliance, which in turn **affects the responses of gastric stretch receptors** to distention of the reservoir.
- **The loss of adaptive relaxation after a vagotomy is associated with a lowered threshold for sensations of fullness and pain.**

- معناه ان الـ adaptive reflex يسمح للمعدة بالاتساع لحد معين ثم يشعر الانسان بالشبع، واذا استمر بالاكل سيشعر بالألم، فإذا فقد الانسان هذا الـReflex سيشعر بالشبع اسرع من الانسان السليم.



2-Motor behavior of the Antral pump region (phasic conteraction):

- Major mixing activities take place in the antrum:

- Contact of gastric chyme with the mucosal surface of the stomach, causes weak peristaltic constrictor waves called mixing waves, initiated by the basic electrical rhythm, once every 15-20 sec.

- These waves progress from the body to the antrum and become intense forcing the chyme to mix and move under high pressure from the antrum toward the pylorus.

- Each time a wave passes from to the antrum to the pylorus, few millimeters of antral content move into the duodenum through the pyloric sphincter.

Gastric action potentials :

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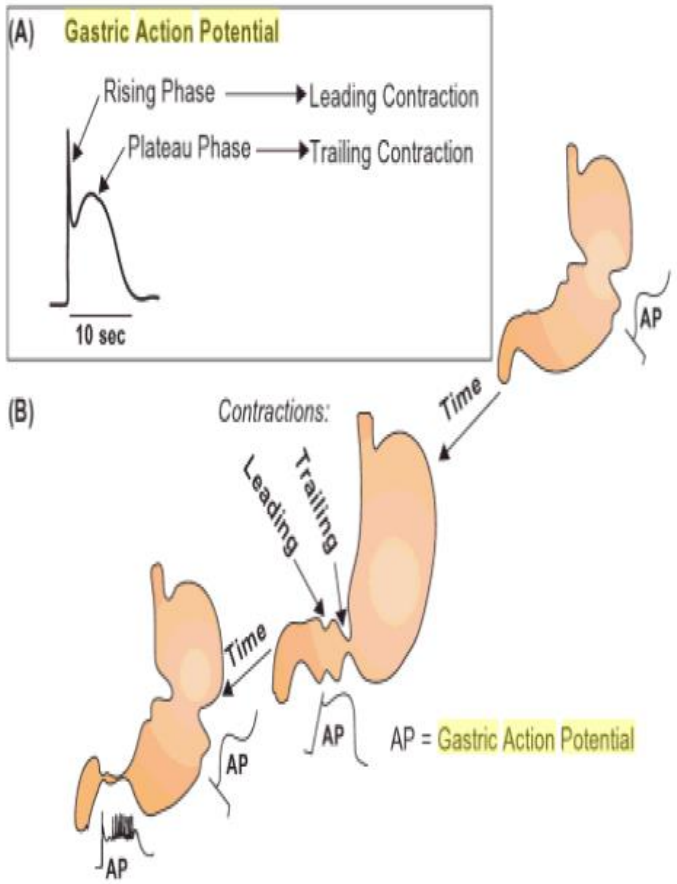
- Gastric action potentials are initiated by a dominant pacemaker (ICC) at a frequency of 3/min and lasts about 5 seconds . They propagate rapidly around the gastric circumference and trigger a ring-like contraction.

- Electrical syncytial properties of the gastric musculature account for propagation of the action potentials (and associated ring-like contraction) to the gastroduodenal junction

Gastric Action Potentials are characterized by :
An initial rapidly rising upstroke (depolarization) >> followed by a plateau phase >> then a falling phase (repolarization) >> back to the baseline membrane potential.



Gastric action potentials :

Males' Slide

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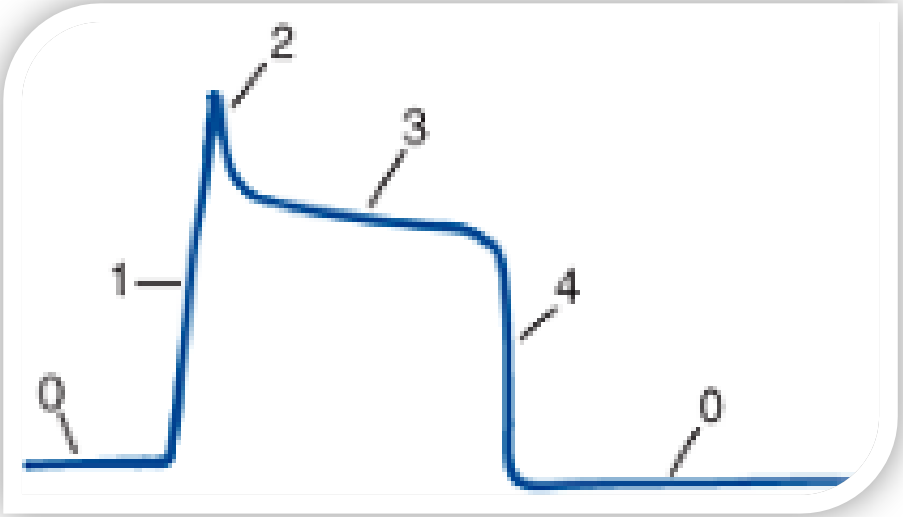
Electrical action potentials in gastrointestinal muscles occur in four phases, determined by specific ionic mechanisms:

1. **Phase 0: Resting membrane potential → outward potassium current**
2. **Phase 1: Rising phase (upstroke depolarization) → activation of voltage-gated calcium channels and voltage-gated potassium channels.**
3. **Phase 3: Plateau phase → balance of inward calcium current and outward potassium current.**
4. **Phase 4: Falling phase (repolarization) → inactivation of voltage-gated calcium channels and activation of calcium-gated potassium channels.**



Gastric action potentials :

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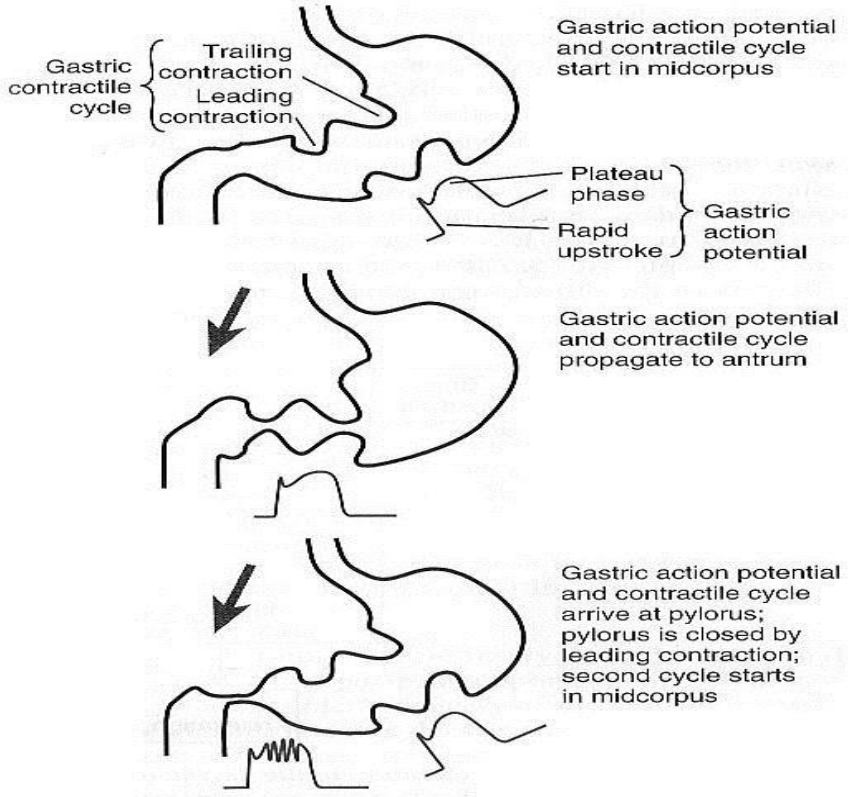
The gastric action potential trigger 2 types of contractions :

(1) A leading contraction	(2) A trailing contraction
which has relatively **constant amplitude**, is associated with the **rising phase** of the action potential	of **variable amplitude**, is associated with the **plateau phase**.



Retropulsion phenomenon:

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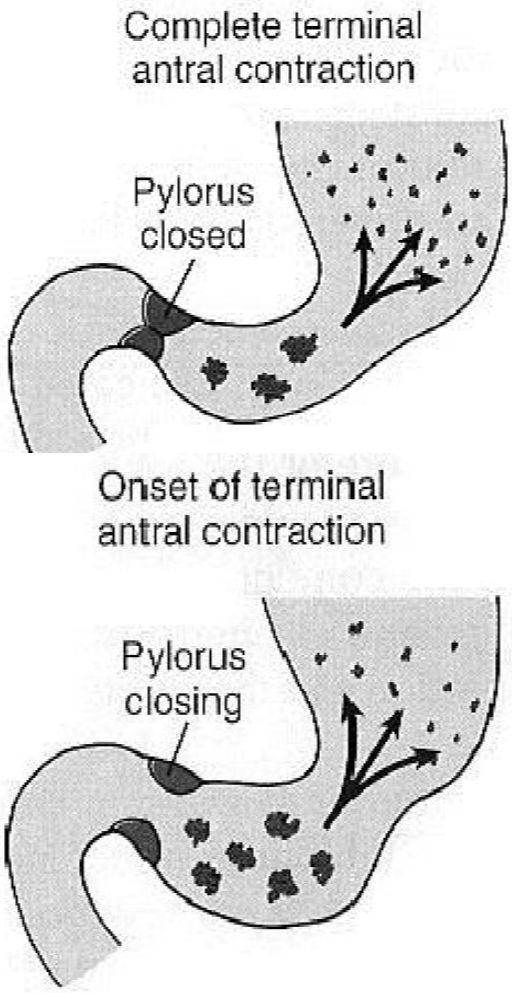
- The leading contractions have negligible amplitude as they propagate to the pylorus. As the rising phase reaches the terminal antrum and spreads into the pylorus, contraction of the pyloric muscle closes the orifice between the stomach and duodenum. The trailing contraction follows the leading contraction by a few seconds.
- As the trailing contraction approaches the closed pylorus, the gastric contents are forced into the antrum of decreasing volume and progressively increasing pressure.
- This results in jet-like retropulsion is formed by the trailing contraction through the pyloric orifice at 3 cycles/min to reduce particle size before they can be emptied into the duodenum.
- These intense peristaltic contractions that cause emptying increase the pressure in the stomach.



Pylorus has a certain diameter , so not all the particles will be able to pass through it's orifice. Only the small one will pass , and large particles > 7 will return back to stomach to complete their digesting and that's why food take long time to go into duodenum.

Hanger contraction:

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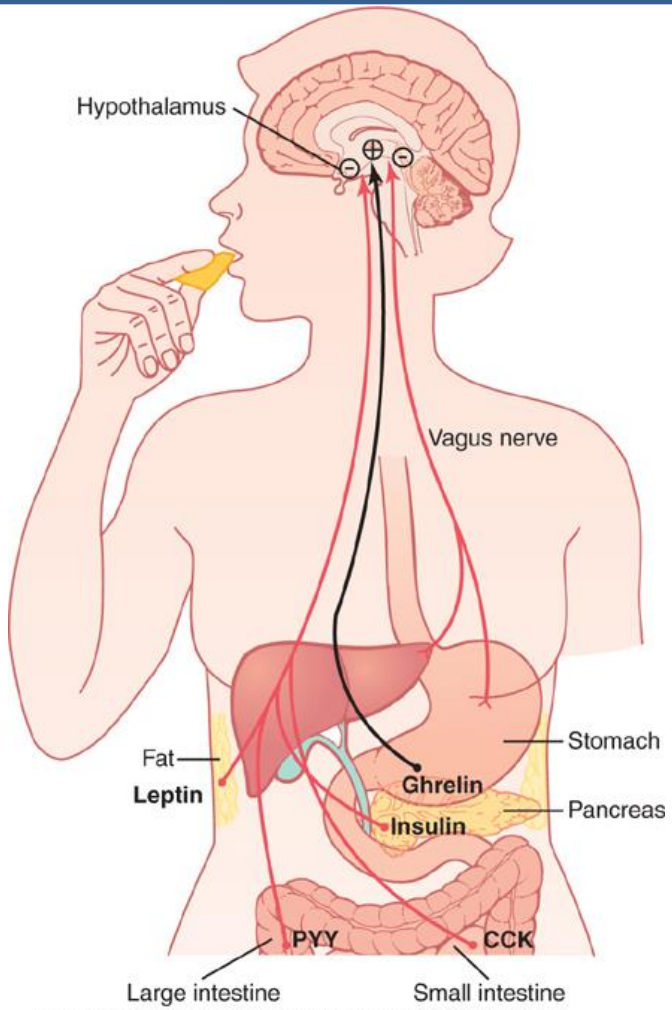
Occur when the stomach has been empty for several hours.

→ These are rhythmical peristaltic contractions that can become very strong and fuse to form a continuing tetanic contraction lasting 2-3 minutes.

→ They are intense in young healthy people and increase by low blood glucose levels.

Hunger pain can begin after 12-24 hr of last food ingestion



Hall: Guyton and Hall Textbook of Medical Physiology, 12th Edition

The Migrating motor complex (MMC) :

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- It is bursts of depolarization accompanied by peristaltic contraction that occur in empty stomach during interdigestive period.

- MMC moves on a long whole length of small intestine to reach ileocaecal valve after 1.5-2 h where it disappears. A new wave of MMC starts.

- The activity of MMC terminates as soon as food is ingested.

- The function of MMC : is to sweep remnants in stomach & small intestine into colon.

- This process occurs in between .
- Start from stomach > through the whole length of small intestine.
- When almost all the food is digested and moved to the small then large intestine , it cleans the area after digestion from the remnants and remove them to colon (زي المكنسة) ☺

Stomach empting:

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- Stomach empting is the results from intense peristaltic antral contractions against resistance to passage of chyme at the pylorus.
- The rate of stomach emptying is controlled by signals from the duodenum and stomach.
- The signals from the duodenum are far stronger and control emptying of chyme at a rate that allows the proper digestion and absorption in the small intestines.

Gastric factors that promote the stomach empting (محفزة دائماً) :

1. Gastric Food Volume:	2. Gastrin Hormone:
An increase in gastric food volume results in increased stretch " into a certain limit "in the stomach wall which elicits local myenteric reflexes that increase the activity of the pyloric pump and inhibit the tonic contraction of the pyloric sphincter leading to increased stomach emptying.	Gastrin increases the activity of the pyloric pump and motor stomach function (moderate effect) and probably promotes stomach emptying.

Stomach empting (Con.):

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The duodenal Factors that can inhibit stomach empting include (مثبطة):

(1) Duodenal distention. to give time for food to be emptying from intestine
(2) Duodenal irritation.
(3) Duodenal acidity activates "S" cells to release Secretin which constricts the antrum. Due to Marked drop in PH
(4) Hyperosmotic chyme in the duodenum. Excess electrolytes
(5) Protein content of the chyme in the duodenum.
(6) Fat (monoglycerides) in the duodenum activates different cells to produce CCK and GIP that inhibit the pyloric pump and increase the tone of the pyloric sphincter thus decreasing stomach emptying.
CCK also acts as an inhibitor to block increased stomach motility caused by gastrin.

Presence of protein and fat > to give time to the product to be digested and absorbed.

- These duodenal factors initiate the enterogastric inhibitory reflexes (see the next slide)

Enterogastric inhibitory reflexes: (→ Doctor said it's important)

When food enters the duodenum, multiple nervous reflexes are initiated from the duodenal wall and pass back to the stomach to regulate stomach emptying depending on the volume of chyme in the duodenum. These duodenal reflexes are mediated by three routes:

- directly from the duodenum to stomach through the enteric nervous system in the gut wall
- through extrinsic nerves that go to the prevertebral sympathetic ganglia and then back through inhibitory sympathetic nerve fibers to the stomach
- probably to a slight extent through the vagus nerves reflex to the brain stem to inhibit the normal excitatory signals that are transmitted to the stomach through the vagus nerves.

These reflexes inhibit the pyloric pump and increase the tone of the pyloric sphincter thus decreasing stomach emptying.

Role of the Pylorus in Controlling Stomach Emptying:

The distal opening is the pylorus. The pyloric sphincter is characterized by strong circular muscle (as compared to the antrum) and remains tonically contracted most of the time. However, during pyloric constriction, watery chyme can still pass through the pylorus into the duodenum, but not food particles. Pyloric constriction is determined by nervous and humoral reflex signals from the stomach and the duodenum.

SUMMARY

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Functions of stomach: storage, digestion, absorption, gradual release of chyme to the duodenum.

Types of cells in stomach:

1. Mucous neck cells: secrete mucus & bicarbonate.
2. G cells: secrete gastrin which stimulates HCL secretion (Gastrin is secreted from stomach, duodenum & pancreas).
3. D cells: secrete somatostatin which decrease HCL secretion.
4. Enterochromaffin – like cells: secrete Histamine.
5. parietal cells (oxyntic cells): secrete IF, HCL.
6. Chief cells (peptic cells): secrete pepsinogen which starts digestion of protein & also secrete gastric lipase.

Factors increase stomach emptying:

parasympathetic & gastrin (increases antrum contraction) .. Distention of stomach.

Factors inhibit stomach emptying:

1. CCK & GIP (stimulated by fat presence in duodenum)
2. secretin (constrict the antrum): secretin secretion stimulated by presence of HCL in duodenum.
3. sympathetic.
4. hyperosmotic or hyposmotic solutions.
5. distention of duodenum.
6. amino acids elicit inhibitory enterogastric reflexes.

SUMMARY

- Intestinal phase (presence of food in intestine) is always inhibitory of gastric emptying.
- Blocking of H2 receptors >> HCL secretion from stomach (as histamine stimulate HCL secretion.
- Gastric HCL is secreted by parietal cells.
- The main gastric area (70-80 %) Includes mucosa of fundus & body.
- HCl is secreted across the parietal cell microvillar membrane and flows out of the intracellular canaliculi into the oxyntic gland lumen.
- Physiological division of stomach:
  1. the orad portion (resirvoir part) > tonic contraction
  2. the caudal portion (antral pump) > phasic contraction

Mechanism of 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 HCO3- in blood = amount of HCl being secreted
- Alkaline tide

Factors promote constriction of pyloric sphincter:

1. Cholecystokinin (CCK)
2. Secretin
3. Glucose-dependent insulinotropic peptide (GIP)
4. Sympathetic innervation

Powerful Duodenal Factors That Inhibit Stomach Emptying :	Gastric Factors That Promote Emptying od stomach:
At the presence of food in the duodenum, multiple nervous reflexes are initiated from the duodenal wall that pass back to the stomach to slow or even stop stomach emptying via one of the following routes: **(1) Directly through ENS,** **(2) Or through extrinsic nerves** **(3) Through the vagus nerves.**	**1) Food Volume**: Increased food volume in the stomach promotes emptying from the stomach (inhibits the pylorus). **2) Gastrin hormone:** enhances the activity of the pyloric pump. Thus, it, too, probably promotes stomach emptying.

SUMMARY

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Phase		Stimuli	Mechanism	
Cephalic phase Excitatory 30% of HCL production		Conditioning ,Seeing, smelling, chewing, and swallowing food .	Direct > vagus > paraital cells	
			Indirect >vagus >(GRP)>gastrin >parietal cells	
Gastric phase Excitatory 60% of HCL production		Distention	Neural (long) : vagal >parietal Vagal > gastrin > parietal	
		Distention of antrum	Neural (short):Local reflex >gastrin> parietal	
		Amino acid and small peptide	Hormonal: Gastrin > parital	
Intestinal phase Excitatory then inhibitory 10% of HCL production	**Excitatory**	Duodenal distension	Neural: vagovagal reflex .	Hormonal : entero-oxyntin hormone > stimulates parietal & G- cells
		Amino acids, small peptide	stimulates G- cells in duodenum & proximal jejunum to release gastrin.	
	Inhibitory	The presence of food in small intestine	enterogastric reflex > ENS & autonomic NS that inhibits G.A secretion.	
		Drop the pH in pyloric antrum to < 2.5.	somatostatin from antral & duodenal "D" cells > reduces G.A secretion	
		The presence of acid, fat, protein digestive products, hypertonic solution in upper intestine	hormonal mechanisms.> inhibits G.A secretion	

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 • 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

1. **which one of the following stimulate the secretion of pepsin enzyme :**

A. Ach
B. Gastrin
C. CCK
D. all of the above

2. **the only essential function of stomach is :**

A. intrinsic factor
B. storage of food
C. endocrine function
D. absorption

3- the intrinsic factor essential for the absorption of :

A. lipids
B. proteins
C. vitamin B12
D. calcium

4- Most of gastric acid secretion occur in :

A. cephalic phase
B. gastric phase
C. intestinal phase

5- which one of the following is NOT true about the action of Gastrin :

A. stimulate intestinal secretion
B. stimulate gastric motility
C. stimulate the biliary secretion
D. relaxing the LES

6- the effect of intestinal phase on the gastric acid secretion :

A. stimulate
B. inhibit
C. first stimulate then inhibit
D. no effect

1-D
2-A
3-C
4-B
5-D
6-C

7- which one of the following is an enterogastrone hormone :

A. Gastrin
B. pancreatic glucagon
C. entero-oxyntin
D. non of the above

8- stomach can store :

A. 0.8 – 1.5 L
B. 0.3 - 0.5 L
C. 2-3 L
D. 3.5 – 4 L

9- the adaptive relaxation of gastric reservoir part triggered by :

A. swallowing reflex
B. stretch receptors
C. presence of chym in small intestine
D. A+B

10- the leading contraction of stomach is ……. And associated with ……. :

A. constant amplitude ,platue phase
B. constant amplitude ,rising phase
C. variable amplitude ,platue phase
D. variable amplitude ,rising phase

11- Hunger contraction increasing by :

A. low blood glucose level
B. low gastric acid secretion
C. increase pancreatic secretion
D. decrease intestinal secretion

7- B
8- A
9- B
10- B
11- A
12- C

12. **Which one of these cells secret HCL ?**
    A. Parietal cells
    B. Chief cells
    C. G Cells
    D. Enterochromaffin cell

13. **Which one of these is Cephalic phase stimulus ?**
    A. Distention
    B. Amino acid
    C. Chewing
    D. Drop the pH

14. **Trailing contraction, is associated ?**
    A. Resting membrane potential
    B. Plateau phase
    C. Falling phase
    D. Rising phase

15. **In vagotomy which reflex we lose ?**
    A. Receptive Relaxation Reflex
    B. Adaptive relaxation
    C. Feedback Relaxation

16. **Upper part of the Stomach function is ?**
    A. Mixing of food.
    B. Digestion of food.
    C. Pushing the food into duodenum.
    D. Storage of food.

12- A
13- C
14- B
15- B
16- D

THE END

Physiology Team

If there are any Problems or Suggestions,
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THANK YOU

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