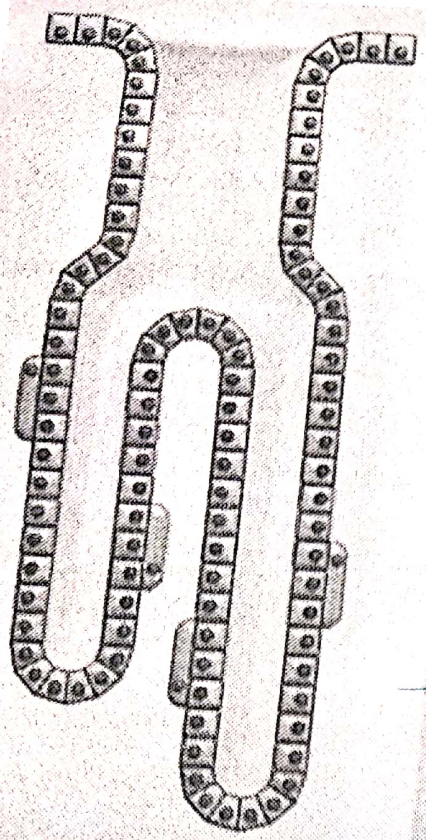
Types of Gastric secretion



Cell Types	Substance Secreted
① Goblet cells	Mucus (protects stomach lining)
② Parietal cells	Gastric acid (e.g. hydrochloric acid)
③ Chief cells	Pepsinogen (protease precursor)
④ D cells	Somatostatin (inhibits acid secretion)
⑤ G cells	Gastrin (stimulates acid secretion)

thin thick

Mucus (protects stomach lining) barrier

Gastric acid (e.g. hydrochloric acid) HCl or oxyntic

+ intrinsic Factor

activate

Pepsinogen (protease precursor) → inactive

Delta

Somatostatin (inhibits acid secretion) inhibit

by receptor found in partial

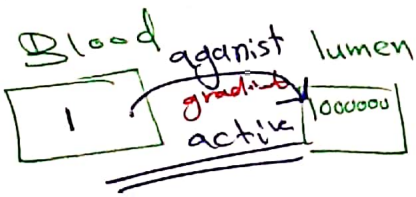
Gastrin (stimulates acid secretion) → hormon from stomach.

regulator

activat parital cell

1

2



3/31/2021

(1) HCL secretion:

So it active secreted

-Hcl is secreted by the oxyntic (parietal) cells.

-Concentration of H+ ions in gastric juice is one million times the conc. in plasma. So, H+ ions is secreted against a very high gradient. → lumen بموجود

-Mechanism of Hcl secretion :

1.Co2 from metabolism or from blood → Co2 + H2O H2 Co3

2.H2 Co3 → H+ + HCo3 .The bicarbonate diffuse to blood in exchange with CL-. Buffer

3.H2O in cytoplasm → H+ + OH-. The H+ is secreted in lumen in exchange with K+ by H+ - K+ pump (proton pump) and OH- form H2O with H+ from carbonic acid.

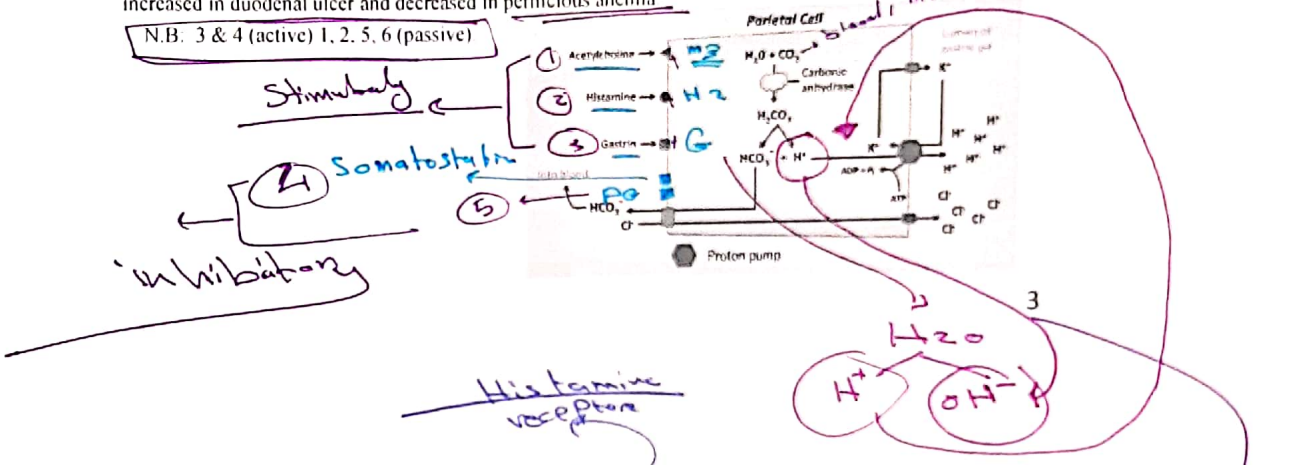
4.CL- is actively secreted into the lumen (the lumen is – 70 mvol.) to unites with H+ → HCL.

5.Water diffused to lumen → iso-osmotic HCL acid.

6.Diffusion of HCo3 to blood → Na HCo3 → post prandial alkaline tide (↑ pH in blood and urine after gastric secretion).

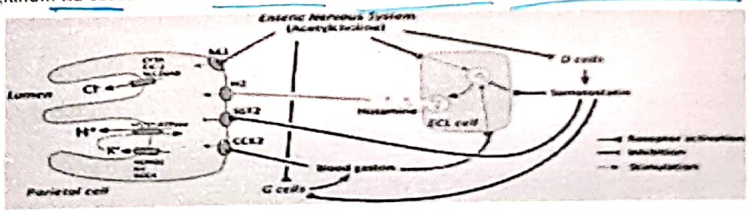
7.The rate of unstimulated secretion is 2mEq/h. The normal maximum rate is 5 mEq/h. This rate increased in duodenal ulcer and decreased in pernicious anemia

N.B: 3 & 4 (active) 1, 2, 5, 6 (passive)



-Factors affecting HCL secretion (receptors on pariatal cells):

1. •Histamine →↑ HCl secretion via stimulation of H2 receptors by ↑ cAMP (these receptors are blocked by cimetidine) = acidity
2. •Acetyl choline →↑ HCl secretion via muscarinic receptors by ↑ Ca+2 & this effect is blocked by atropin. acid HCl
3. •Gastrin →↑ HCl secretion via G cells by ↑ intra cellular Ca+2.
4. •Prostaglandin E2 causes decrease HCl secretion via ↓ cAMP (used in treatment of peptic ulcer)



5 Somatostatin inhibitory

- Functions of HCL

1) Sterilization : by acidity (So, in infants less HCL secretion→ more gastroenteritis (kill bacteria)

2) Digestion of protein by activation of pepsinogen → pepsin & give optimum pH of its effect and hydrolysis of protein.

3) HCl enters the duodenum → ↑ secretin hormone →↑ bile and pancreatic secretion. محفز

4) Produces curdling of milk. بكتيريا → تخثر الحليب

5) Initiate enterogastric inhibitory reflex →↓ gastric secretion and evacuation.

6) ↑ absorption of iron (by converting ferric state into ferrous) and calcium (by prevention of calcium salts precipitation)

Fe+3 Fe+2

Hcl secretion

1) CO_2 → (inside the cell) which come from metabolism of the cell (from its cytoplasm) or from blood

CO_2 (+) H_2O (come from cytoplasm) $\xrightarrow{C.A}$ H_2CO_3

2) Rapidly H_2CO_3 $\xrightarrow{\text{Carbonic anhydrase}}$ H^+ (+) HCO_3^-

- diffuse (passive) to blood in exchange with Cl^-
- it react with Na^+ to give $NaHCO_3$ (it is a Buffer) → which called post prandial Alkaline tide ↑ pH in blood + urin

H^+ (from CO_2) + OH^- (From H_2O) → H_2O

3) H_2O in cytoplasm → H^+ + OH^-

H^+ → secreted in Lumen in exchange with K^+ by H^+-K^+ active pump which also called (proton pump)

4) Cl^- actively secreted in lumen unites with H^+ to give HCl (in Lumen)

* H^+ that may be finally Hcl come from H_2O not CO_2

(2) Secretion of enzymes :

A- pepsinogens : (I & II) : Secreted by chief (peptic) cells as Inactive pepsinogen → active pepsin. by (Autoactivation + HCl)

-Of optimum pH 1.6 →3.2.

-Digest proteins → proteases & polypeptides.

-Pepsinogen- I is large amount, secreted by chief cells and its secretion is linked with HCL secretion

-Pepsinogen- II is less amount, secreted by mucosal cells and not linked with HCL secretion.

B-Gelatinase : which liquefies gelatin.

C-Gastric lipase: act on short chain fat. Its optimum pH = 3.

D-Amylase (from saliva).

E-Rennin: milk clotting enzymes (not present in humans).

maybefound inanimal

لكنه لا يعمل في المعدة لأنه يحتاج pH الى 6.3

5

(3) Secretion of intrinsic factor :

-It is a glycoprotein secreted from oxyntic cells with HCL.

-It is essential for vit B12 absorption in ileum.

-In gastritis → pernicious anemia (↓ B12 anemia).

الأنيميا الخبيثة

(4) Secretion of Mucus:

There are two types of mucus:

-**Soluble thin mucus**: secreted by mucus neck cells by vagal as muco-proteins to lubricate gastric chyme.

-**Insoluble thick mucus:** Secreted by the surface epithelium.

-Viscid alkaline mucus layer to protect gastric wall from digestion & acidity.

Goblet cell

يشكل بيئة في gastric mucosal barrier

when it may be deficient in someone → it increase risk of gastric ulcer

6

3

(5) Secretion of gastrin hormone : From G cell

-It is a polypeptide of 3 types according to number of amino acids G34, G17 (most important) and G14.

-It is secreted from: **G-cells**: in pyloric antrum, flask – shaped cells and have microvilli contains receptors. (chemoreceptor)

T.G cells: in mucosa of stomach and small intestine → G34.

growth of epithelial mucosa

-Action of gastrin on:

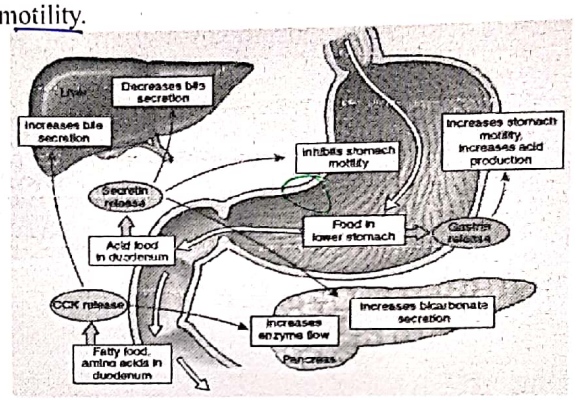
-Stomach: ↑ growth & secretion & motility.

-Pancreas:

↑ exocrine and endocrine secretion.

-Sphincters:

- Lower esoph. → Contraction.
- Ileocecal → Relaxation.



7

V.I Take Care

Regulation of Gastrin Hormone secretion

Open Close

	Stimulation	Inhibition
• **Chemical factors**	• Polypeptides, amino acids , caffeine and alcohol	• ↑acidity (pH<2) -ve F.B via release of somatostatin → D cell
• **Luminal**	• Distension of the stomach. (result from food)	
• **Blood born**	• Calcium, adrenaline. (with stress)	• Secretin, GIP, VIP, calcitonin, glucagon
• **Neural**	• Vagal by gastrin releasing peptide. • Sympathetic (as in anxiety and anger).	sympathetic (as in fear and depression).

Negative Feed back inhibitory

Gastroinhibitory peptin

التوتر الشديد

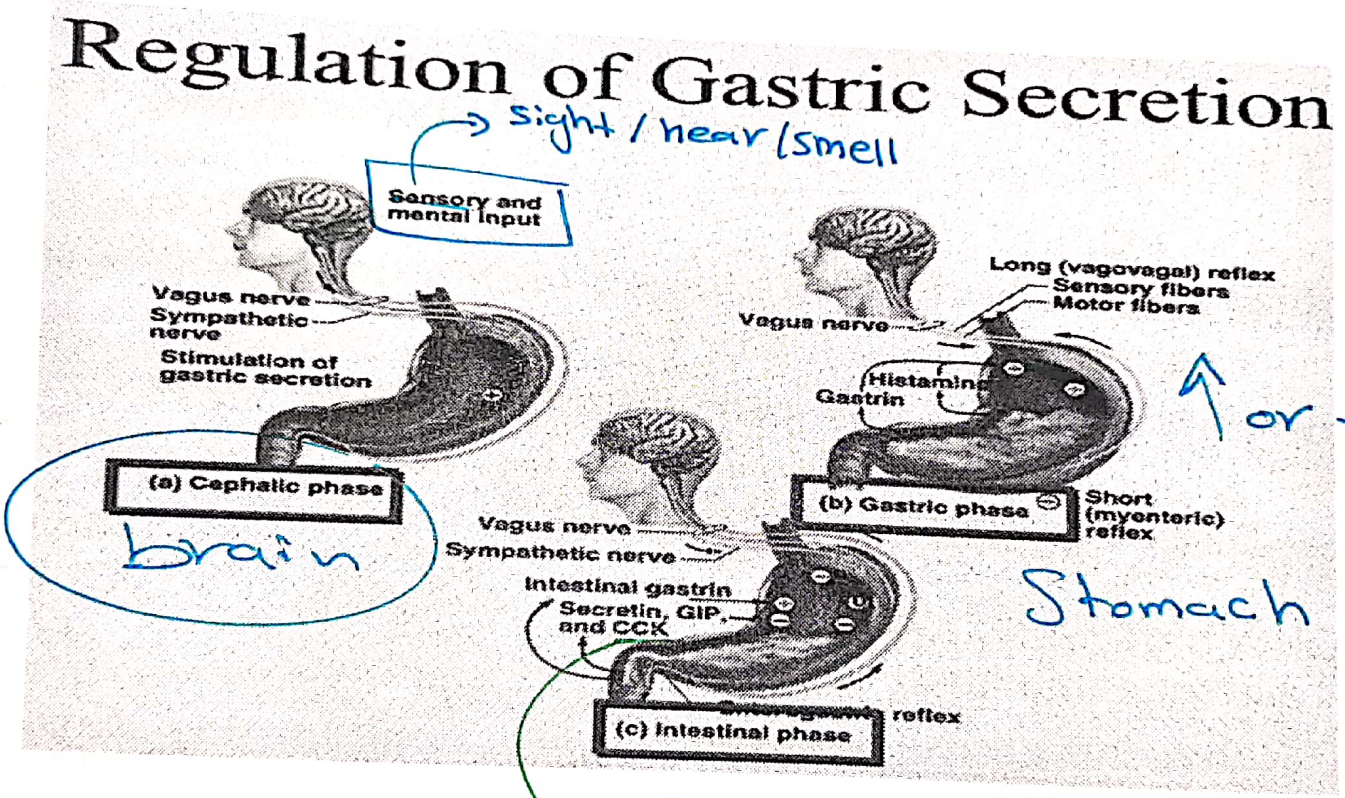
الاكتئاب والخوف

8

brain/centers

Control of gastric secretion:

•Nervous and hormonal.



•**Three phases:**

A-Cephalic phase: (25 %)

- It is a nervous phase activated by conditioned and unconditioned reflexes:

.In the conditioned reflex: Psychic stimulation of cerebral cortex will stimulate the vagal center.

.In the unconditioned reflex: direct contact of food stimulate taste buds which give afferent to the vagal center.

-The vagal nuclei stimulate gastric secretion by:

1.Direct stimulation of gastric glands (ACh).

2.Release of gastrin hormone (Gastrin releasing peptide).

-This phase increases by anxiety and decreases in depression.

11

B-Gastric phase : (70 %)

The presence of food in the stomach → increase gastric secretion by mechanical, chemical and neural stimuli as the following:

•Gastrin secretion: by direct stimuli as polypeptides, alcohol and caffeine or via local and vago-vagal reflex to inhibit the vagal center.

•Local nerve plexus: by distension or polypeptides → stimulate Meissner's plexus →↑ secretion.

•Vago-vagal long reflex: food in stomach → afferent vagus to vagal center & efferent vagal increase in gastric secretion so inhibited by atropine.

N.B : hypoglycemia →↑ vagal stimuli →↑ secretion.

12

C-Intestinal phase:

The presence of food in the duodenum may inhibit the gastric secretion: as the following:

In the duodenum: presence of acid, fats or hyperosmotic solution in the duodenum will inhibit the gastric secretion via:

1- Nervous mechanism (Entero-gastric reflex):

- It is stimulated by presence of acid, fats or hyperosmotic solution in the duodenum or distention of the duodenum will inhibit the gastric secretion.
- The reflex is conducted in the three ways: local, ganglionic or vago – vagal reflex.
- The response and the importance:

1. Inhibition of gastric secretion and motility
2. Protection of duodenum from over distention by increase in the tone of pyloric sphincter → delay the emptying.
3. Protection of duodenum from hyperacidity (till neutralized by alkaline duodenal secretion).
4. Insure protein digestion.
5. Prevent rapid electrolyte changes during intestinal absorption.

13

2- Hormonal mechanism (Entero-gastrone hormone):

- It is stimulated by the presence of fats and fatty acids → the release of 4 hormones from the duodenum [cholecystokinin (CCK), secretin, gastric inhibitory peptide (GIP) & VIP] → hormonal feed – back inhibition of gastric secretion and motility for complete digestion of fat.

-Gastric-inhibitory peptide (GIP): is a duodenal hormone secreted in response to presence of glucose and fat in the duodenum and causes inhibition of gastric function and stimulate the insulin hormone release from pancreas.

14