Gastric secretions

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Stomach

Functions of the stomach

- Storage of food.
- Slow evacuation of meal to allow good digestion
- Partial digestion of proteins and fats.
- Sterilization of ingested food by high acidity.
- Secretion of HCl, enzymes,….
- Help defecation by gastro-colic reflex.

Gastric secretion

- **Gastric secretion**

It is 2.5 – 3 L/day of acidic juice (pH may reach 1)

It is secreted from the **gastric glands**

- **Gastric glands**

- Simple tubular glands open at the mucosal surface at the gastric pits.
- In these glands, many types of cells are present:
  1) Mucous neck cells (Goblet) → Mucus.
  2) Chief cells → Pepsinogen & enzymes.
  3) Oxyntic (parietal) cells → HCL & intrinsic factor (essential for life for absorption of vit.B12).
  4) G. cells → Gastrin H.
  5) D. cells → Somatostatin.
  6) Entero-chromaffin like cells → histamine
  - The pyloric canal and cardiac region contain goblet cells only.
  - The body & fundus contain all types of cells except **G. cells.**
  - The antrum of pyloric area contains **1, 2, 4& 5 types of cells.**

HCL secretion:

- **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.
- ***Mechanism of HCl secretion*:**

1. In parietal cell CO2 (from metabolism)→ CO2 + H2O → H2 CO3 under effect of Carbonic anhydrase enzyme.

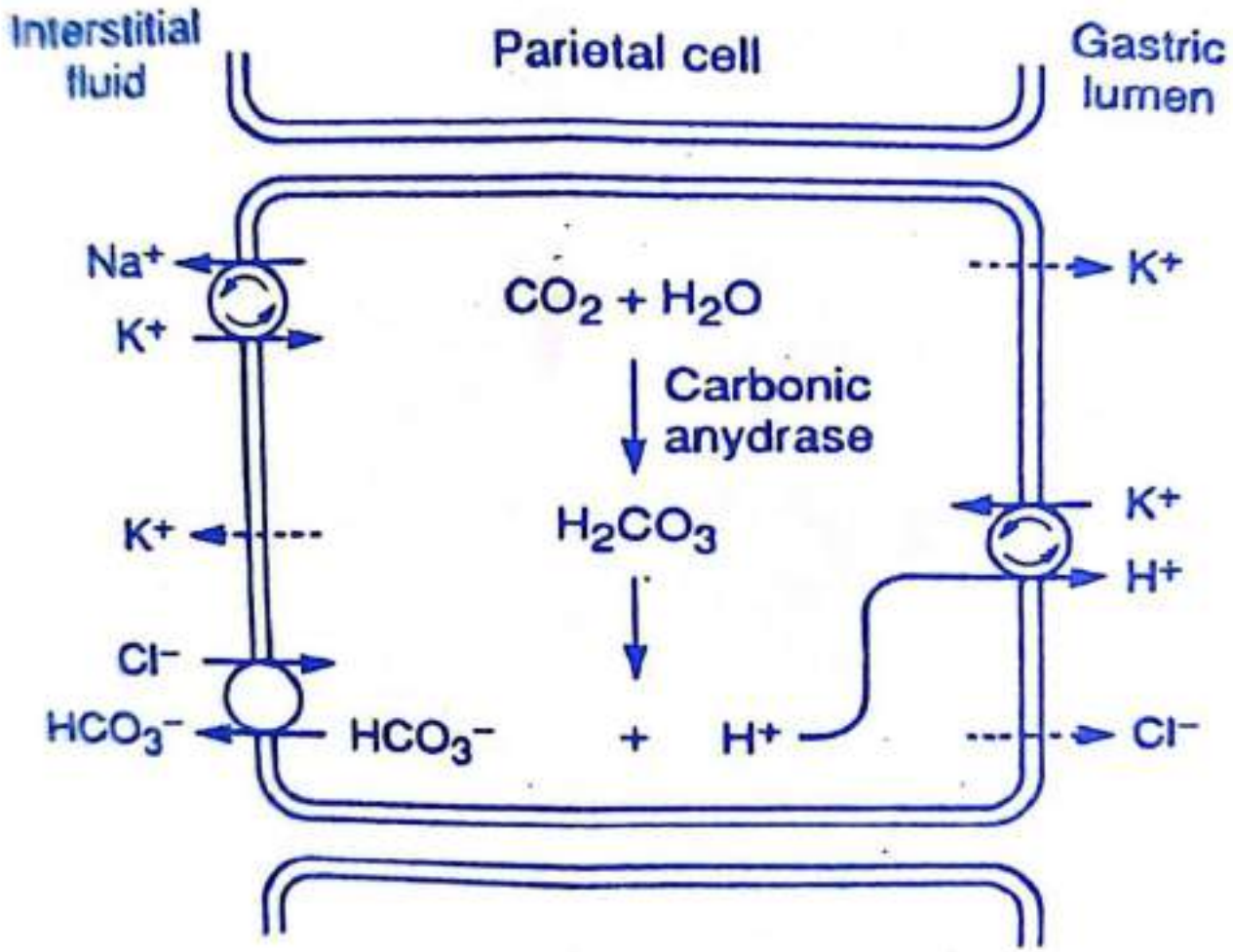
2. H2 Co3 → **H+** + **HCo3-**

3. The **bicarbonate diffuse to blood** in exchange with **CL-**

4. **The H+ is secreted** in lumen in exchange with **K+** by **H+ - K+ pump**

5. **CL-** **Diffuse** into the lumen to unites with **H+** → HCL

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

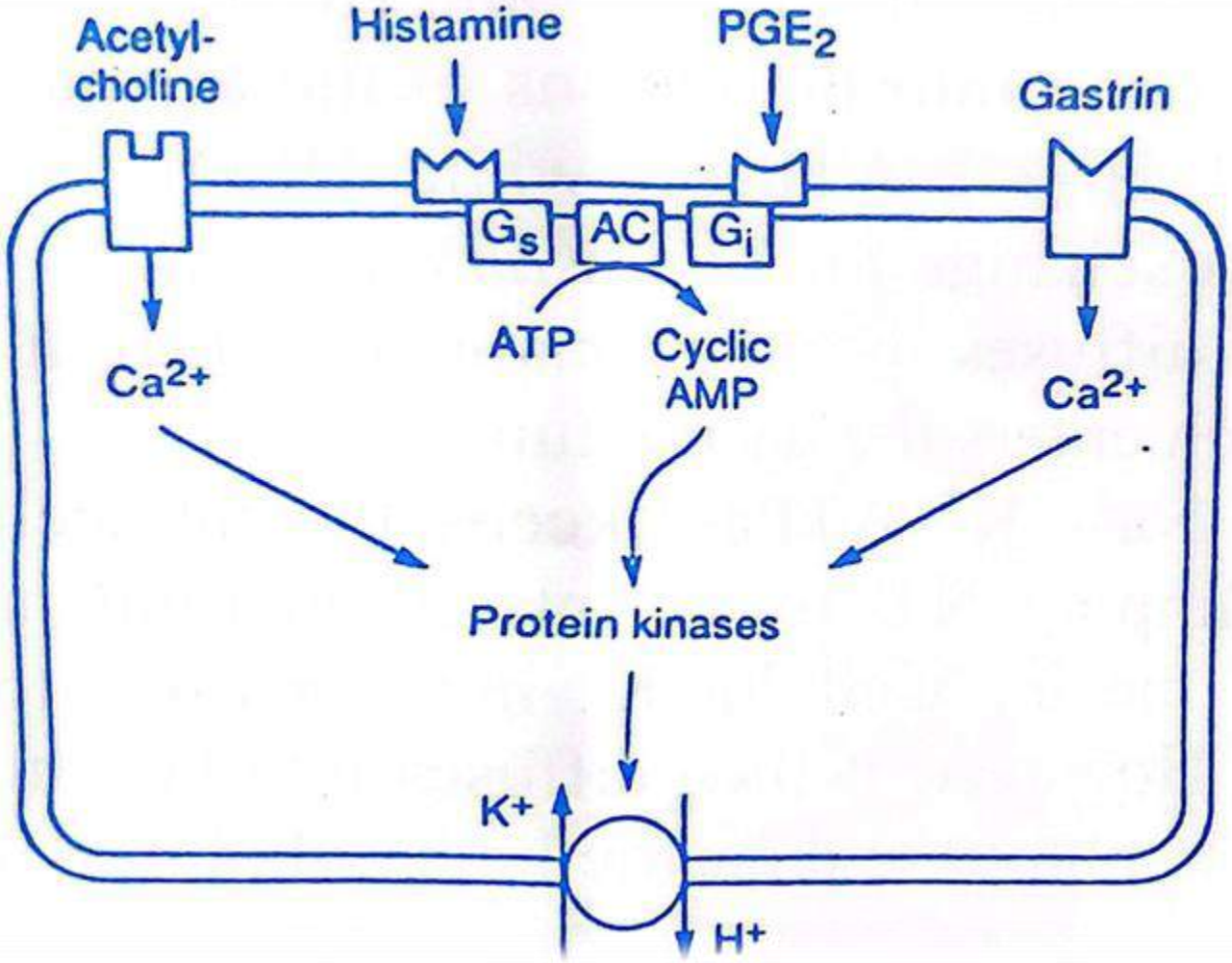


Mechanism of HCL formation in the parietal cells

Factors affecting HCL secretion (Receptors on parietal cells)

- Histamine →↑ **HCl** secretion via stimulation of **H2 receptors** by ↑ cAMP (these receptors are blocked by cimetidine).
- Acetyl choline →↑ **HCl** secretion via muscarinic receptors (**M3**) by ↑ intra-cellular Ca+2 & this effect is blocked by atropine.
- Gastrin →↑ **HCl** secretion via special gastrin receptors by ↑ intra-cellular Ca+2.
- Prostaglandin E2 causes **decrease HCl** secretion via

↓ cAMP (used in treatment of peptic ulcer)



Regulation of HCL secretion

Functions of HCL

- Sterilization : by acidity (So, in infants less HCL secretion→ more gastroenteritis .
- Digestion of protein by activation of **pepsinogen** → pepsin & give optimum pH of its effect (hydrolysis of protein)
- HCl enters the duodenum → ↑ **secretin** hormone →↑ bile and pancreatic secretion.
- Produces curdling of milk.
- Initiate **entero-gastric inhibitory reflex** →↓ gastric secretion and evacuation.
- ↑ absorption of iron (by converting ferric state into ferrous)

Secretion of enzymes

A- *pepsinogens : (I & II)*

- Secreted by chief (peptic) cells.
- Inactive pepsinogen $\xrightarrow{HCL}$ active pepsin.
- Of optimum pH 1.6 – 3.2.
- Digest proteins → proteases & polypeptides.
- Pepsinogen I is large amount, secreted by the 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.

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).

Secretion of Mucus

There are **two types** of mucus

1.Soluble thin mucus:

Secreted by **mucus neck cells** by vagal stimulation as muco-proteins to lubricate gastric chyme.

2. Insoluble thick mucus:

Secreted by **the surface epithelium**
Viscid alkaline mucus layer to protect gastric wall from digestion & acidity.

N.B: the gastric mucosal barrier is protected by:

1) The insoluble thick alkaline (1 mm layer).
2) The mucosal cells are **impermeable** to H+ which is pumped to the lumen .
3) Prostaglandins stimulate the previous two factors and **antagonist HCL secretion**
4) The tight junction between mucosal cells to prevent passing HCL in between cells.

N.B: Duodenum is protected by mucosal barrier + pancreatic alkaline secretion.

Secretion of gastrin hormone

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

Action of gastrin

- **Stomach**: ↑ growth & secretion & motility.
- **Pancreas**: ↑ exocrine and endocrine secretion.
- **Sphincters**: - Lower esoph. → Contraction.
  - Ileocecal → Relaxation.

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 nuclei.

In the unconditioned reflex: Direct contact of food stimulate taste buds which give afferent to the vagal nuclei ,then the vagal nuclei stimulate gastric secretion by:
1.Direct stimulation of gastric glands **(ACh)** 2.Release of gastrin hormone
This is proved by :

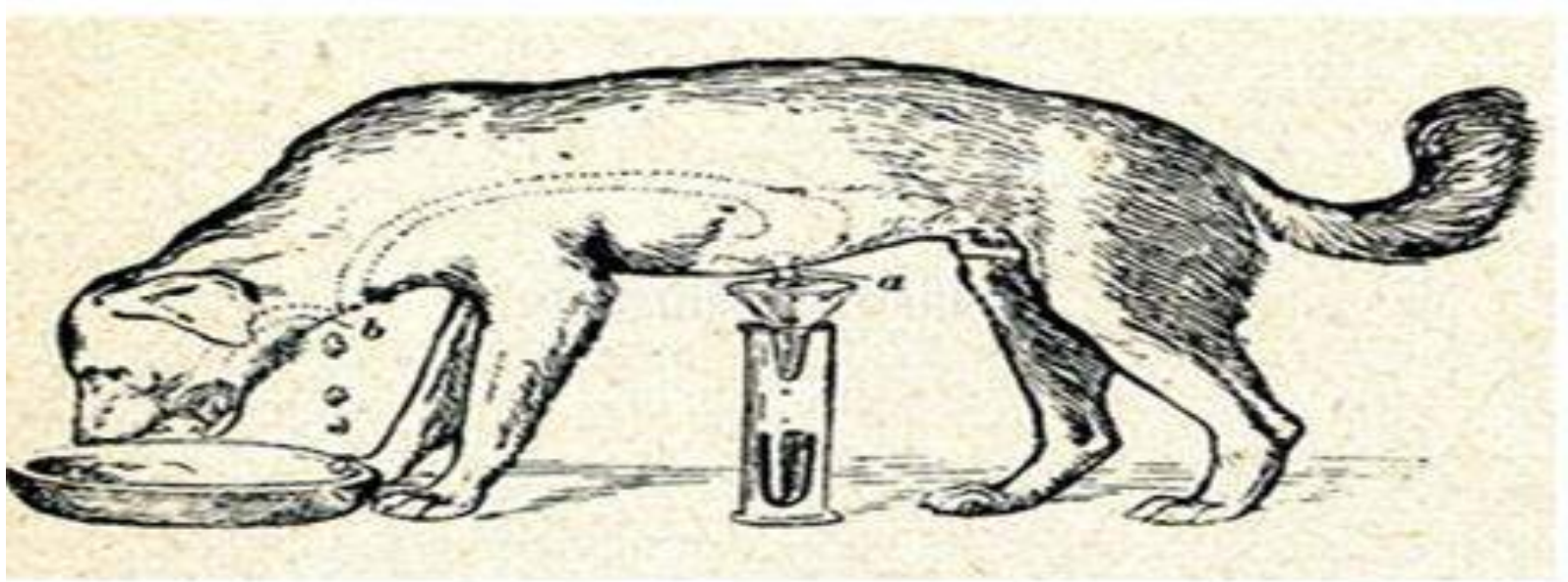
- **Sham feeding:**

The esophagus is make to open in neck, so the food swallowed will pass to outside through this fistula & part of the stomach is made into a pouch to outside.
The blood & nerve supply is kept intact.

- **Denervated pouch:**

The nerve supply to the pouch is cut but the blood supply is intact.

Sham feeding increases gastric secretion but not in **Denervated pouch**. So, secretion depends on **intact vagal nerve**.



Sham feeding

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
- **Local nerve plexus**: by distension or polypeptides → stimulate Meissner's plexus →↑ secretion.
- **Vago-vagal long reflex**: food in stomach → afferent vagus to vagal nuclei & efferent vagal → increase in gastric secretion so inhibited by **atropine**.

C-Intestinal phase:

The presence of food in the **duodenum** ,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 many ways: local 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.

2- Hormonal mechanism (Enterogastrone hormone)

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

N.B. :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.


THank you