

Lecture series

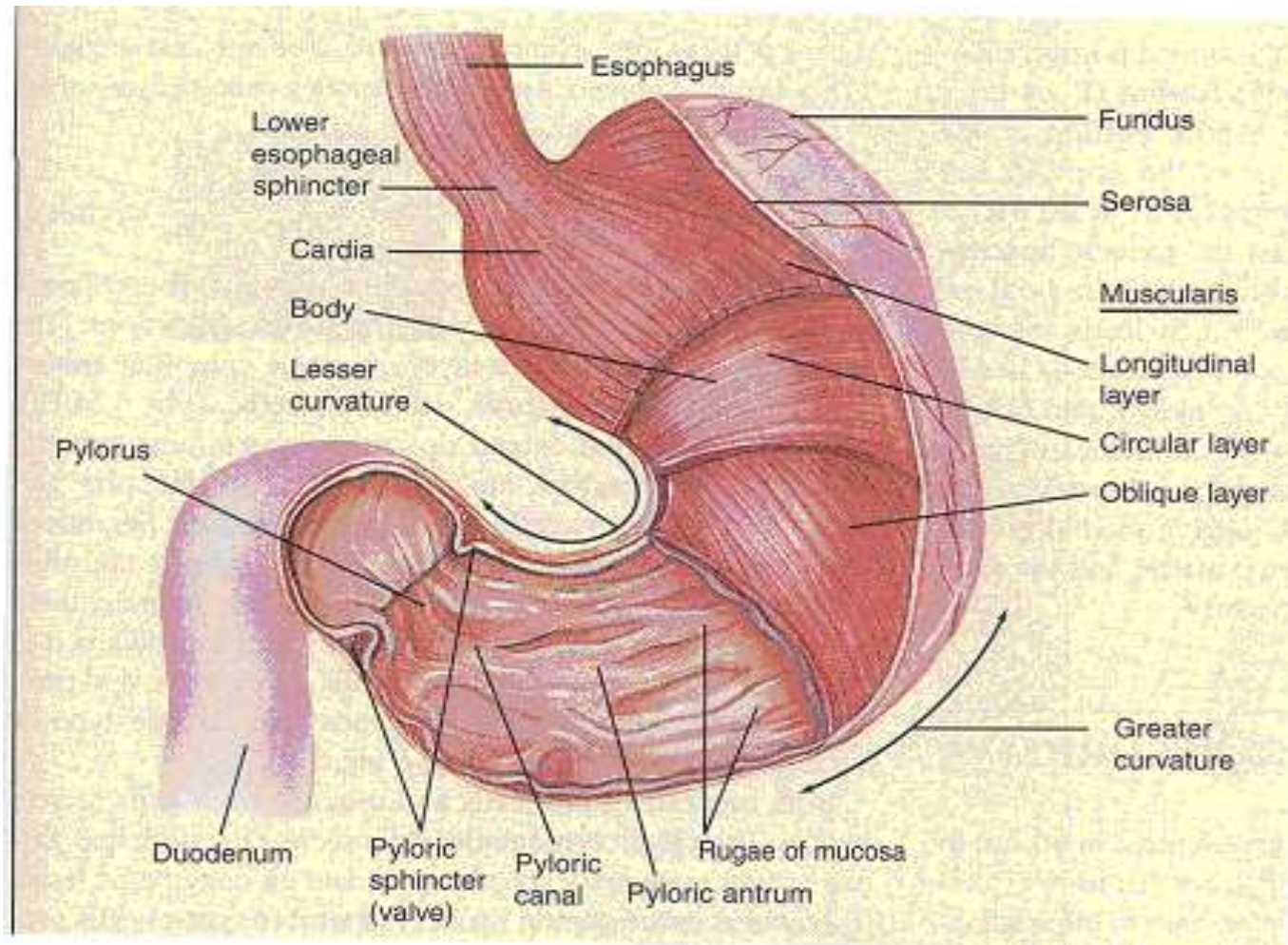
Gastrointestinal tract



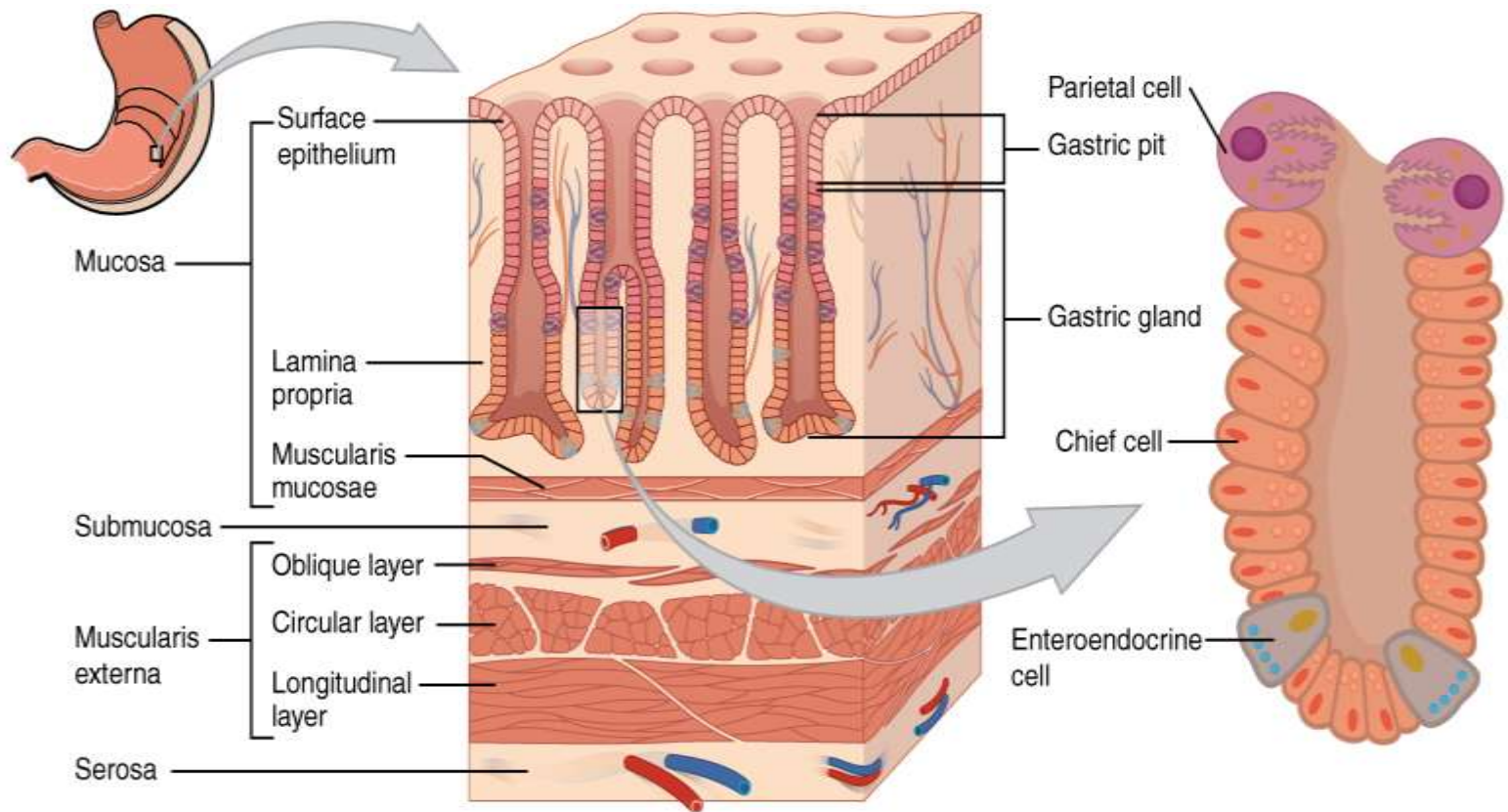
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STOMACH AND ITS SECRETIONS

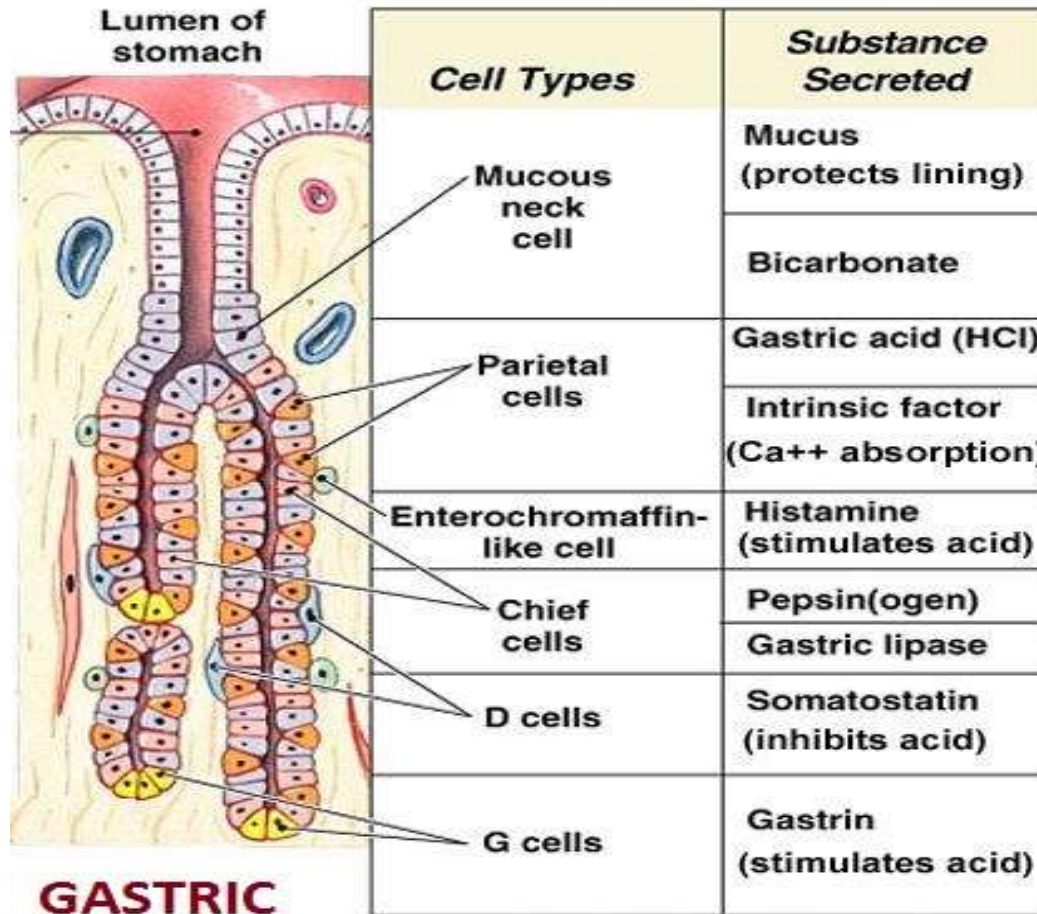
Anatomical consideration



Histological representation of gastric wall



Physiological view of gastric glands



<i>Cell Types</i>	<i>Substance Secreted</i>
Mucous neck cell	Mucus (protects lining)
	Bicarbonate
Parietal cells	Gastric acid (HCl)
	Intrinsic factor (Ca ⁺⁺ absorption)
Enterochromaffin-like cell	Histamine (stimulates acid)
Chief cells	Pepsin(ogen)
	Gastric lipase
D cells	Somatostatin (inhibits acid)
G cells	Gastrin (stimulates acid)

**GASTRIC
GLANDS**

Basic functions of the stomach

- Food is stored in the stomach
- Mixed with acid mucus and pepsin
- Stomach also add significant amount of digestive juices to meal
- As cephalic phase of gastric secretion start earlier
- This food is released in steady in to the duodenum

Gastric secretions

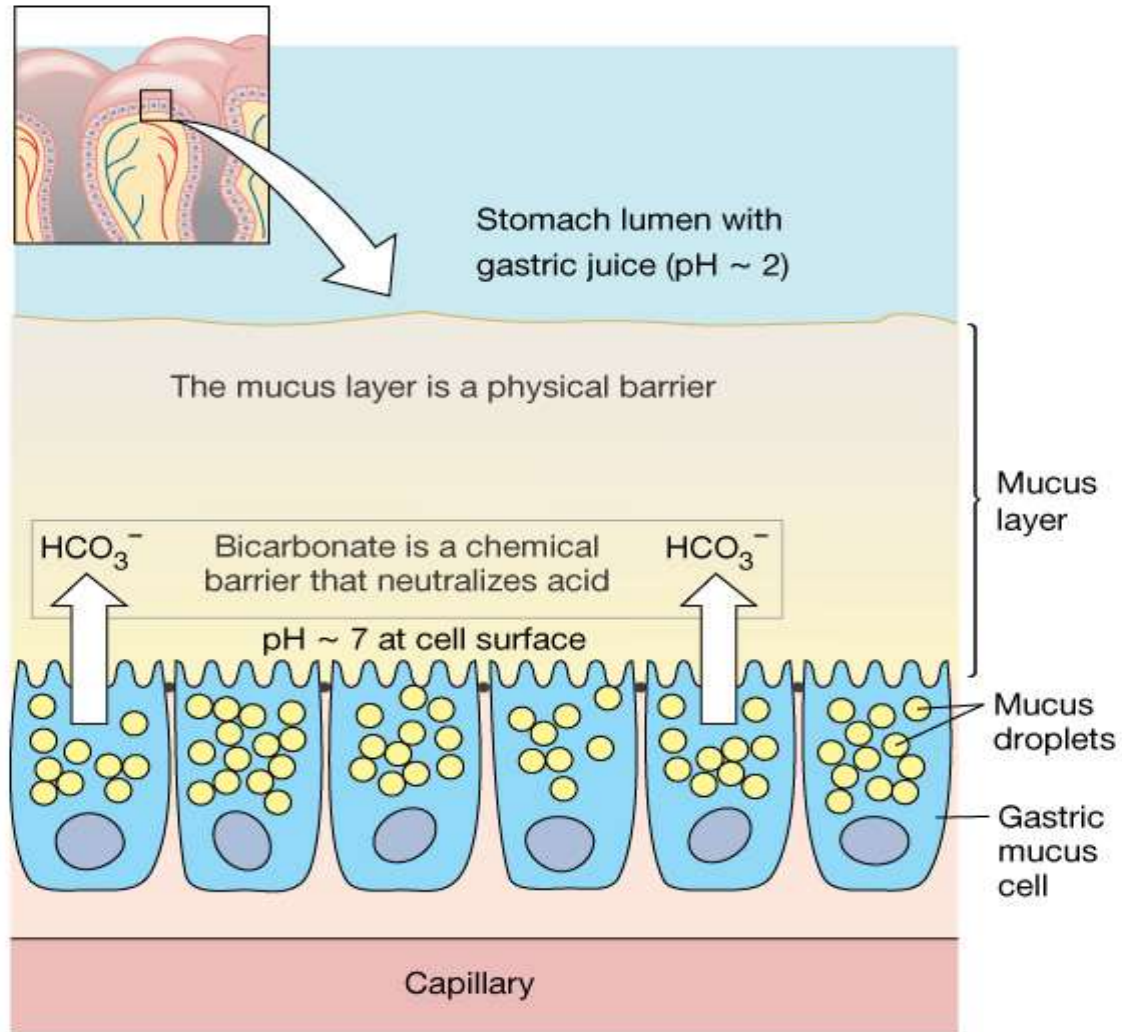
It is a colorless, watery, acidic, digestive fluid produced in the stomach

- Pale yellow in colour ,
- pH is 1-3 ,
- Per day secretion is 2-3 L .

Chemical composition;

- It contains inorganic salts ,
- and organic components that include ,mucin, digestive enzymes hormones, intrinsic factors

Goblet cells or mucus cells

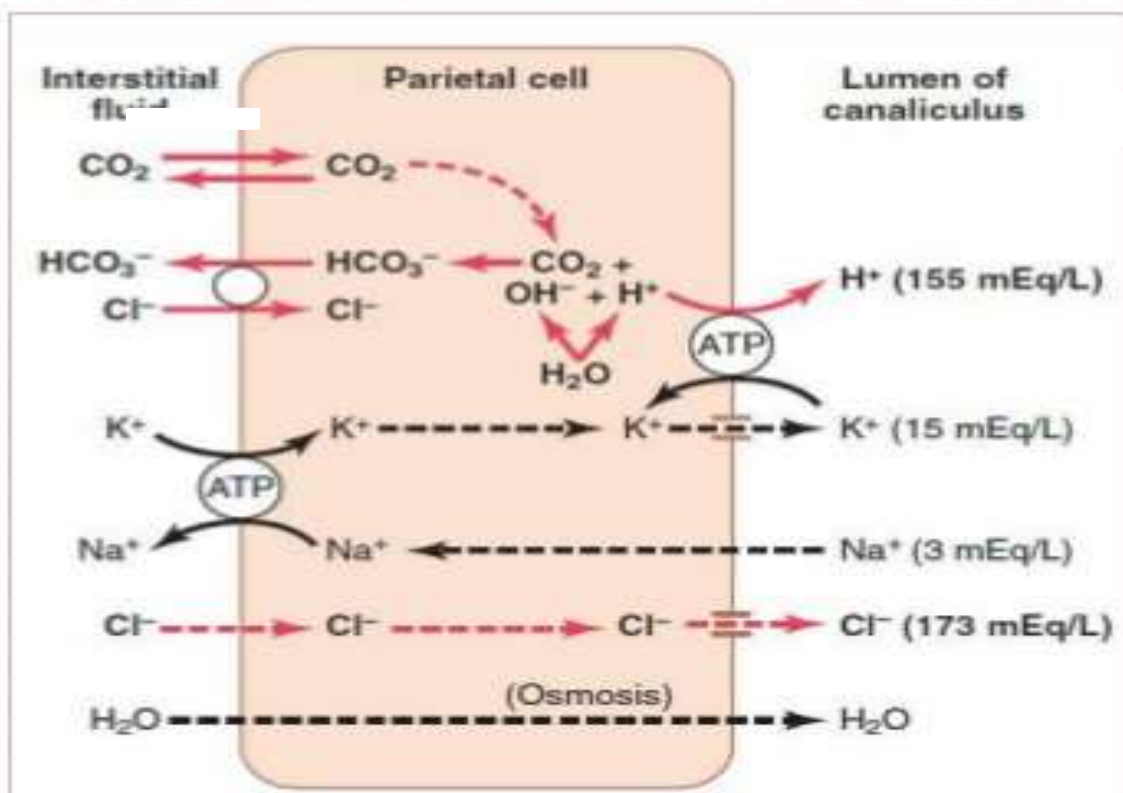


Parietal cells:

- They secrete **HCl** into the stomach
 - This acid is important for activation of pepsinogen, inactivation of microorganisms ,
 - It also secretes the intrinsic factor, necessary for intestinal absorption of vitamin B12.

Gastric Secretion

- Basic Mechanism of HCl secretion



Chief cells:

- **Gastric Lipase** is also secreted by chief cells, responsible for the initiation of fat digestion
- It secretes **pepsinogen(zymogen)**. Once secreted, pepsinogen is activated by stomach acid into the active protease pepsin,

G cells

- G cells secrete **Gastrin** hormone which in turn stimulates chief, parietal and ECL cells
- G cells are activated by GRP and inhibited by somatostatin

ECL cells and D cells

- ECL cells secrete **Histamine** ↑ HCl sec.
- D cells secrete **somatostatin** by the influence of HCl
- Somatostatin inhibits G cell

Composition and function of gastric secretions

HCl converts pepsinogen to pepsin for chemical digestion

- provides optimal pH environment for pepsin
- destroys some bacteria
- stimulates the small intestinal mucosa to release Secretin and CCK
- promotes the absorption of Ca^{2+} and Fe^{2+} in small intestine

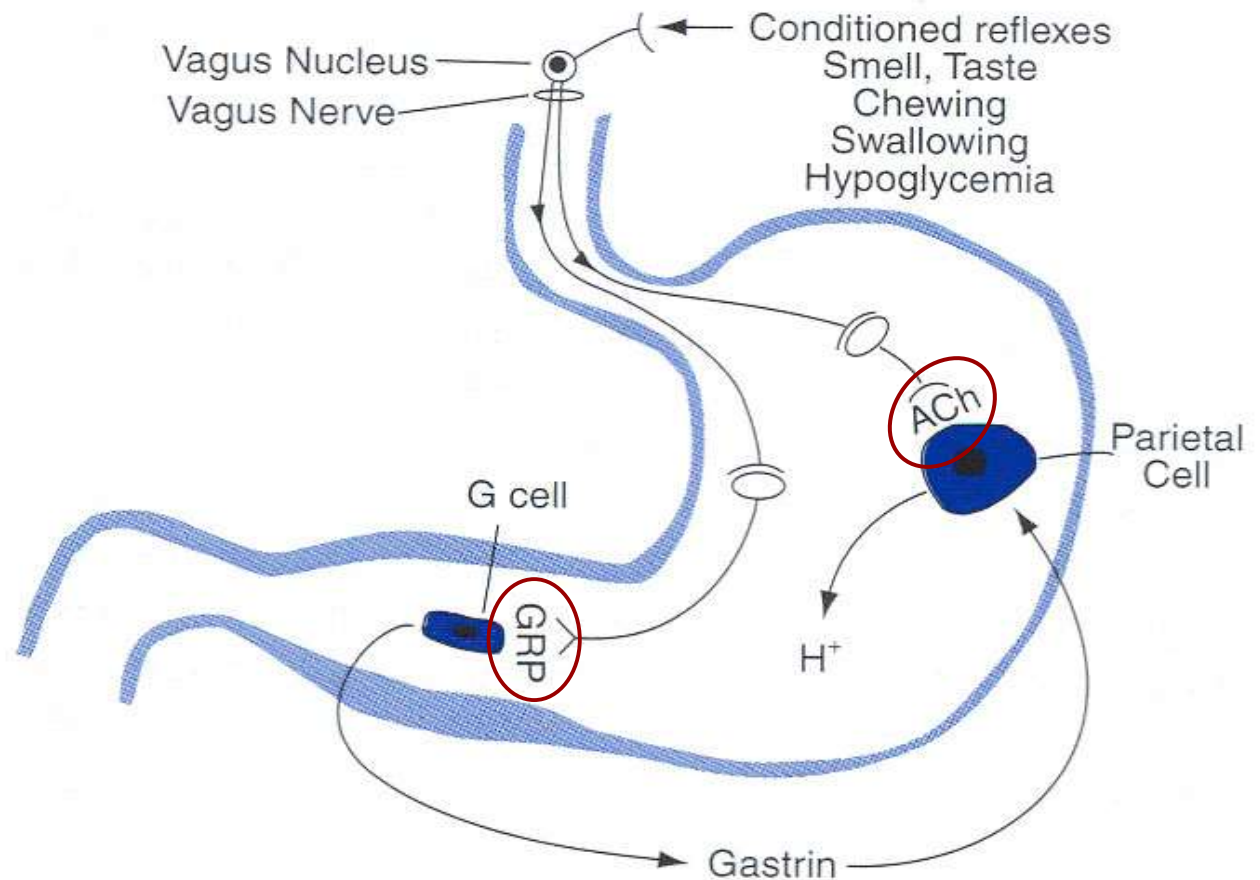
Gastric secretion phases

Gastric acid secretion can be divided into three phases:

- **Cephalic phase** mediated by the CNS and triggered by smelling, chewing or even the thought of food. Mediated by the vagus and accounts for 10- 30% of the acid secreted).
- **Gastric phase triggered by the presence of food in the stomach** Accounts for 70-90% of the acid secretion
-
- **Intestinal phase.** Presence of chyme, most probably amino acids, in the intestine triggers approximately 5% of the gastric acid secretion.

Stimulation of acid secretion – *cephalic phase*

- blocked by vagotomy
- sham feeding
- hypoglycemia
- role of GRP (bombesin)



Intestinal phase

- **Presence of chyme, most** probably amino acids, in the intestine triggers approximately 5% of the gastric acid secretion.

Gastric acid secretion is controlled by three mechanisms:

- ▶ Neurocrine (denoting an endocrine influence on or by the nerves).
- ▶ Endocrine (gastrin)
- ▶ Paracrine (histamine) in contrast to true endocrines these hormones are not released into the bloodstream but into the surrounding tissues and act in the immediate vicinity, e.g. intestinal mucosal hormones.

APPLIED PHYSIOLOGY

Dysphasia :- dysphagia means difficulty in swallowing.

causes :

- 1) Mechanical obstruction of esophagus due to tumor, stricture, diverticular hernia (out pouching of the wall).
- 2) Decreased movement of esophagus due to neurological disorder such as parkinsonism.
- 3) Muscular disorder leading to difficulty in swallowing during oral stage or esophageal stage.

ESOPHAGEAL ACHALASIA

it is due to the failure of lower esophageal (cardiac)sphincter to relax during swallowing. The accumulated food substance cause dilatation of esophagus.

- The feature of disease are :
 - Dysphagia
 - Chest pain
 - Weight loss
 - cough

GASTRITIS

- Inflammation of gastric mucous membrane is called gastritis.
- It may be acute or chronic
- **Acute gastritis** is characterized by inflammation of superficial layers of mucous membrane and infiltration with leukocytes, mostly neutrophils.
- **Chronic gastritis** involves inflammation of even the deeper layers and infiltration with more lymphocytes. it results in the atrophy of the gastric mucosa with loss of chief cells and parietal cells of gland. therefore The secretion of gastric juice decreases.

GASTRIC ATROPHY

- Gastric atrophy is the condition in which the muscles of the stomach shrink and become weak.
- The gastric glands also shrink resulting in the deficiency of gastric juice.
- Cause by: Loss of gastric gland

Peptic Ulcer Disease

- Peptic ulcers:
 - Erosions of the mucous membranes of the stomach or duodenum produced by action of HCl. Disruption of mucus barrier
- Zollinger-Ellison syndrome:
 - Ulcers of the duodenum are produced by excessive gastric acid secretions. Due to gastrinomas
- Helicobacter pylori:
 - Bacterium that resides in GI tract that may produce ulcers.
- Acute gastritis:
 - **Histamine released by tissue damage and inflammation stimulate further acid secretion.**

Management of Ulcers

- Proton pump inhibitors (omeprazole)
- Antibiotics assist in eradicating H. pylori bacteria.
- Histamine 2 (H2) receptor antagonists (Ranitidine)
- Local antacids
- Life Style Changes

STOMACH CARCINOMA

Types- Adenocarcinoma(most common)

-Lymphoma

-Mesenchymal tumour

*Risk factors-*1.smoking,spicy food

-2.obesity

-3.Helicobacter pylori

-4.genetics

*SITE-*Body of stomach near greater curvature(Mc)

-Pylorus

Symptoms

Early-Heartburn, upper GI pain, nausea
loss of appetite

Late-weight loss, vomiting, difficulty in swallowing, blood in stool

Diagnostic-Biopsy

Prevention & Treatment- healthy lifestyle
surgery, chemotherapy

Radiotherapy in advanced stages

References

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- Medical Physiology, Updated second edition (walter F. Boron, MD, phd)
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- Ganong's Review of Medical Physiology, 26 t h e d i t i o n