Introduction to GI System

Introduction to GI System

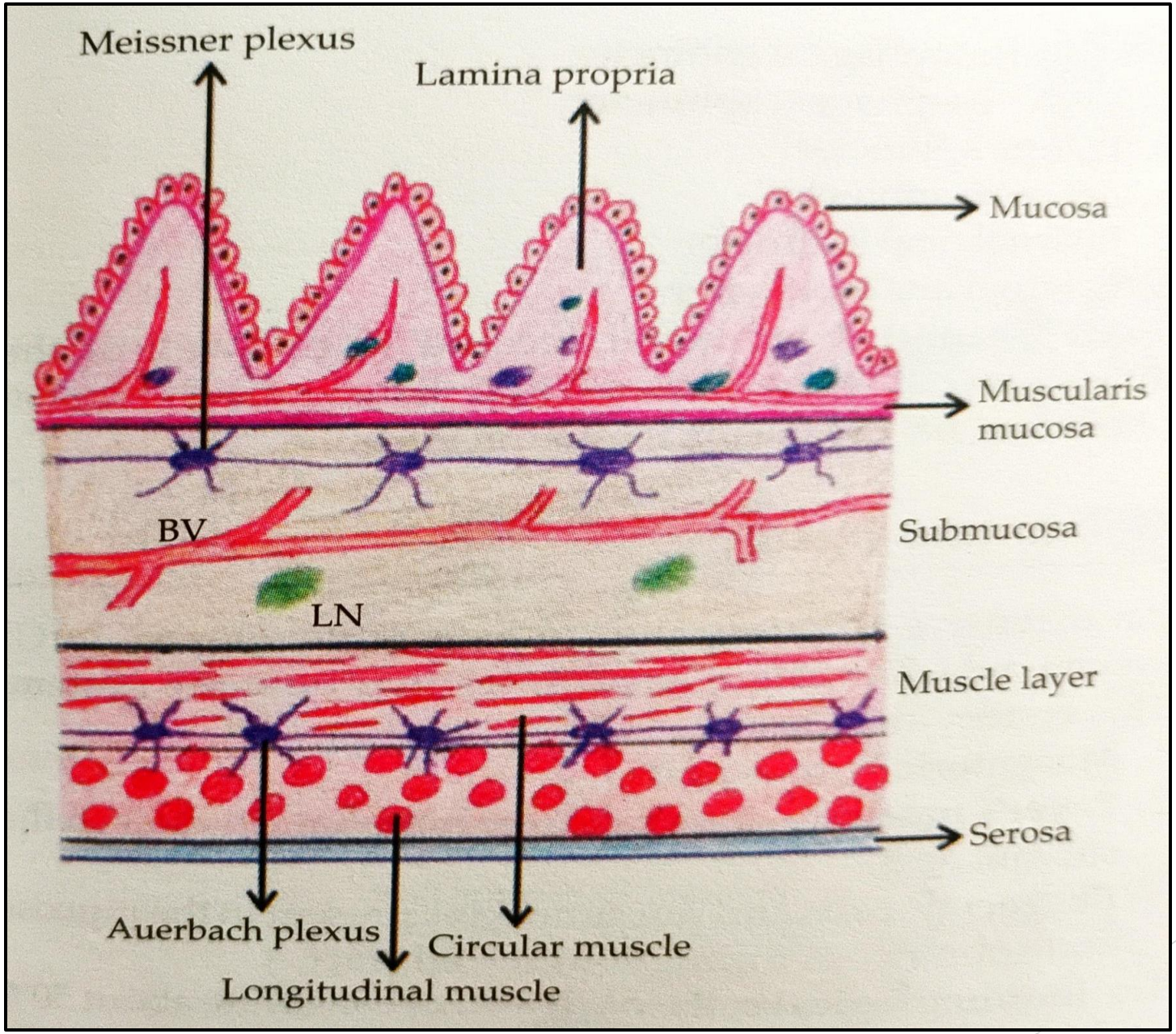
FUNCTIONS

- Digestion & Absorption of food
- Excretion of waste materials
- Fluid & Electrolyte balance-consume 1.5 litres of water , 7L secreted into GI tract as secretions of various GI tracts.
- 99% reabsorbed.100ml of liquid excreted in feces per day.

Introduction to GI System

FUNCTIONS

- IMMUNITY
- GALTS
- Acidic secretion of stomach
- INTESTINAL BACTERIA FLORA-Non pathogenic bacteria-Essential for many intestinal functions


Meissner plexus
Lamina propria
Mucosa
Muscularis mucosa
BV
Submucosa
LN
Muscle layer
Serosa
Auerbach plexus
Circular muscle
Longitudinal muscle

PARTS OF GI SYSTEM

- Mouth,
- Pharynx,
- Oesophagus,
- Stomach,
- small intestine,
- colon, rectum and anus.
- Glandular structures-salivary glands, liver, exocrine pancreas, intestinal glands

INNERVATION OF GI TRACT

1.**SYMPATHETIC**

- Inhibitory
- Via noradrenergic fibres having cell bodies in prevertebral & paravertebral ganglia
- Post ganglionic fibres originate from celiac, superior & inferior mesenteric ganglia.
- Sympathetic fibres end on intrinsic plexus of enteric nervous system.
- Effects-Relaxation of GI smooth muscles

INNERVATION OF GI TRACT

- Contraction of sphincters
- Inhibition of GI secretions.
- PARASYMPATHETIC INNERVATION
- Stimulatory
- Mainly via VAGUS nerve-from oral cavity to transverse colon
- Remaining parts from PELVIC nerves
- End on intrinsic plexus of ENS.
- Increased motility& exocrine secretion of GI tract

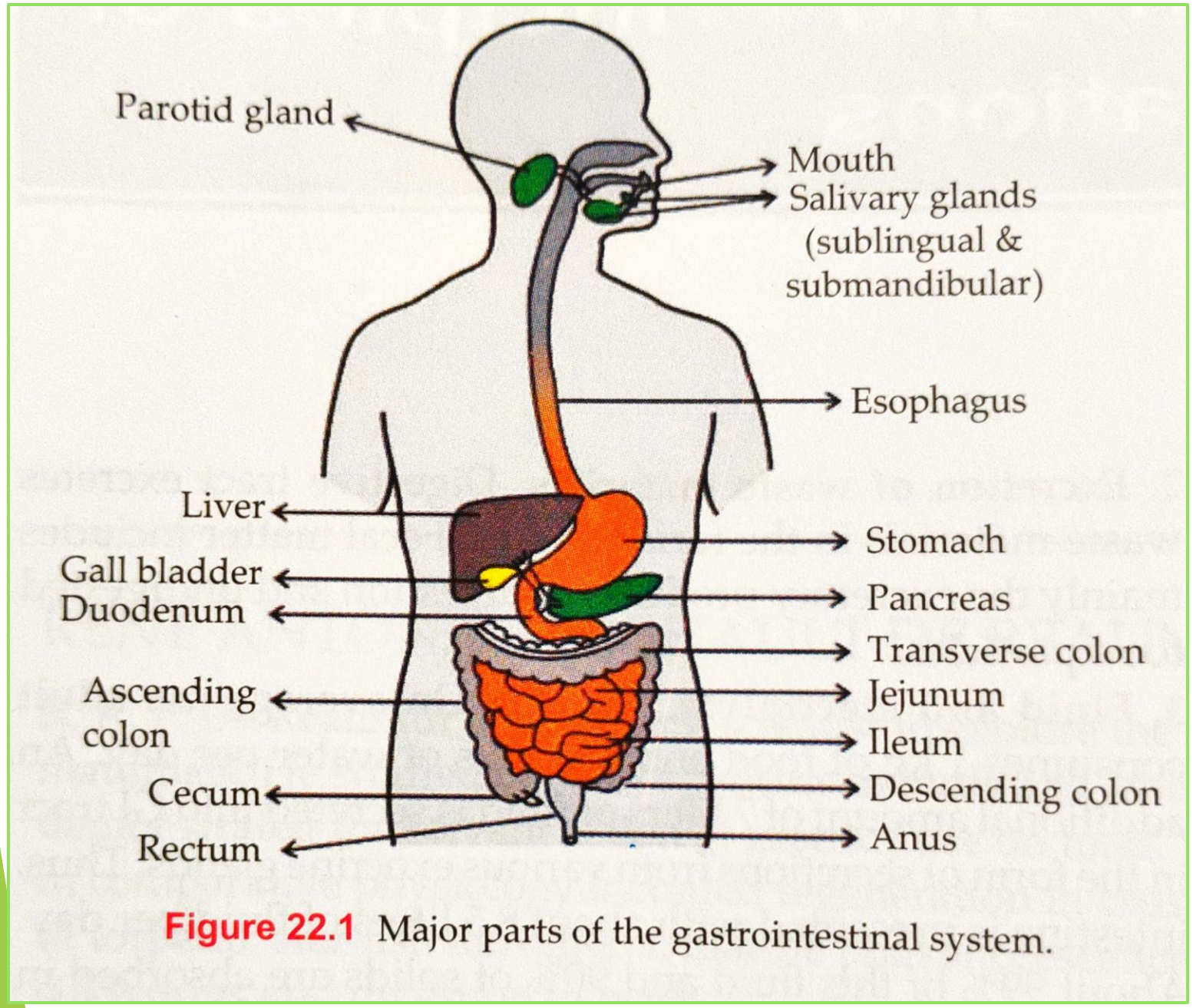


Figure 22.1 Major parts of the gastrointestinal system.

SALIVARY GLANDS

- Parotid gland, Submandibular(Submaxillary),Sublingual gland.
- Minor glands are also there.

COMPOSITION OF SALIVA

- Variable
- Mixed salivary secretion contain,
  - Water-99.5%
  - Organic & Inorganic constituents
- INORGANIC
  - Carbonic acid, Bicarbonate, Phosphate, Sodium, Pottassium, Calcium, Magnesium, chloride ions

COMPOSITION OF SALIVA

ORGANIC

- MUCIN-Specially from sublingual gland.
  - Lubricate food, Protect oral mucosa.
- Alpha Amylase(Ptyalin)
  - Split cooked starch
  - Chloride ions for activation
  - No action on cellulose

COMPOSITION OF SALIVA

LINGUAL LIPASE

- Produced by glands of tongue (Ebners glands)
- Active only in stomach
- Digest 30% of triglycerides

LYSOZYME

- Bactericidal

KALLIKREIN

COMPOSITION OF SALIVA

BLOOD GROUP ANTIGENTS IN SECRETORS

- IgA, IgM-Act against bacteria

PAROTIN-Local hormone from parotid & Submaxillary gland-Deposition of calcium in teeth.

LACTOFERRIN- Binds with Iron, Bacteriostatic.

PROLEIN RICH PROTEIN -Binds calcium ions &Toxic Tannins.

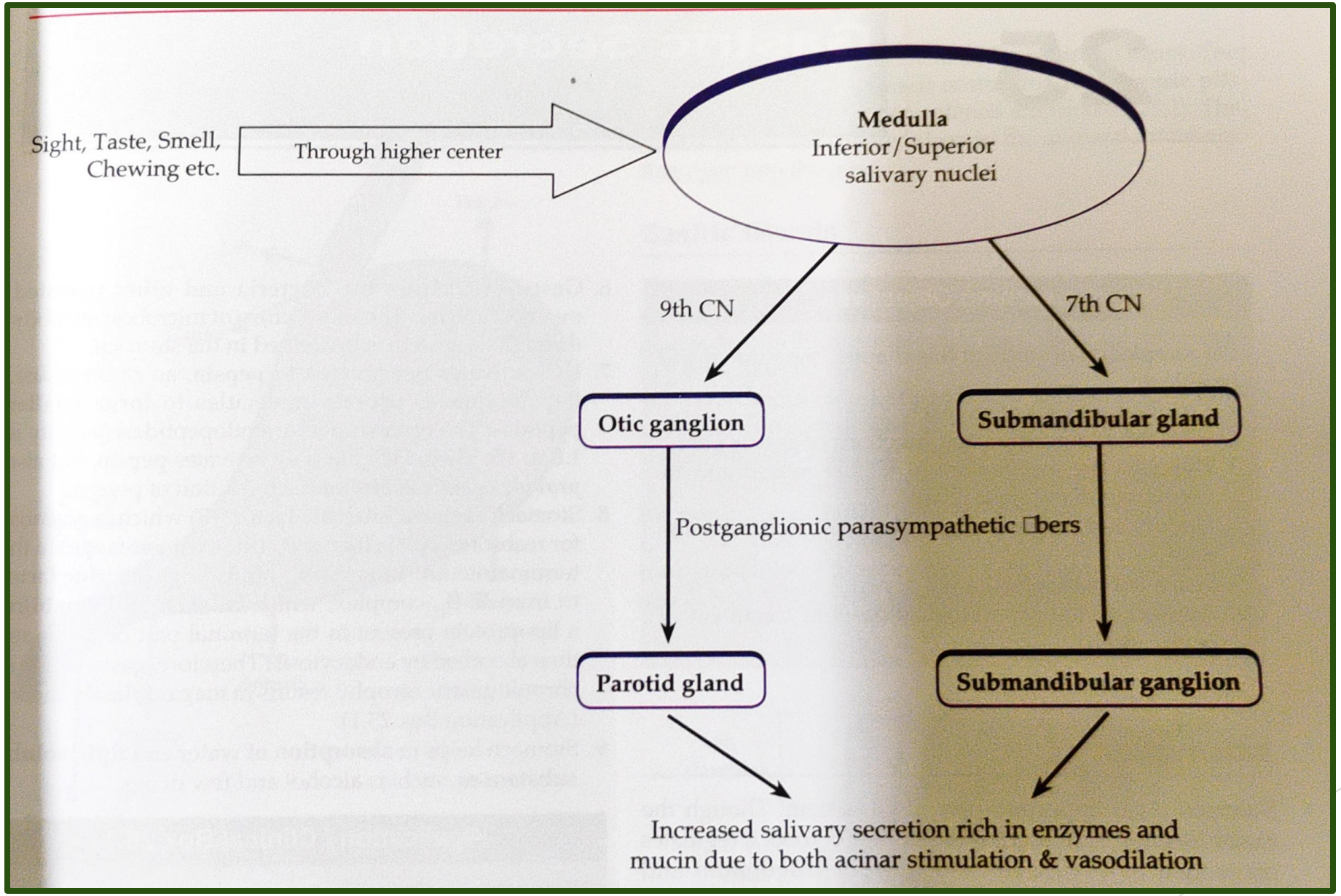
FUNCTIONS OF SALIVA

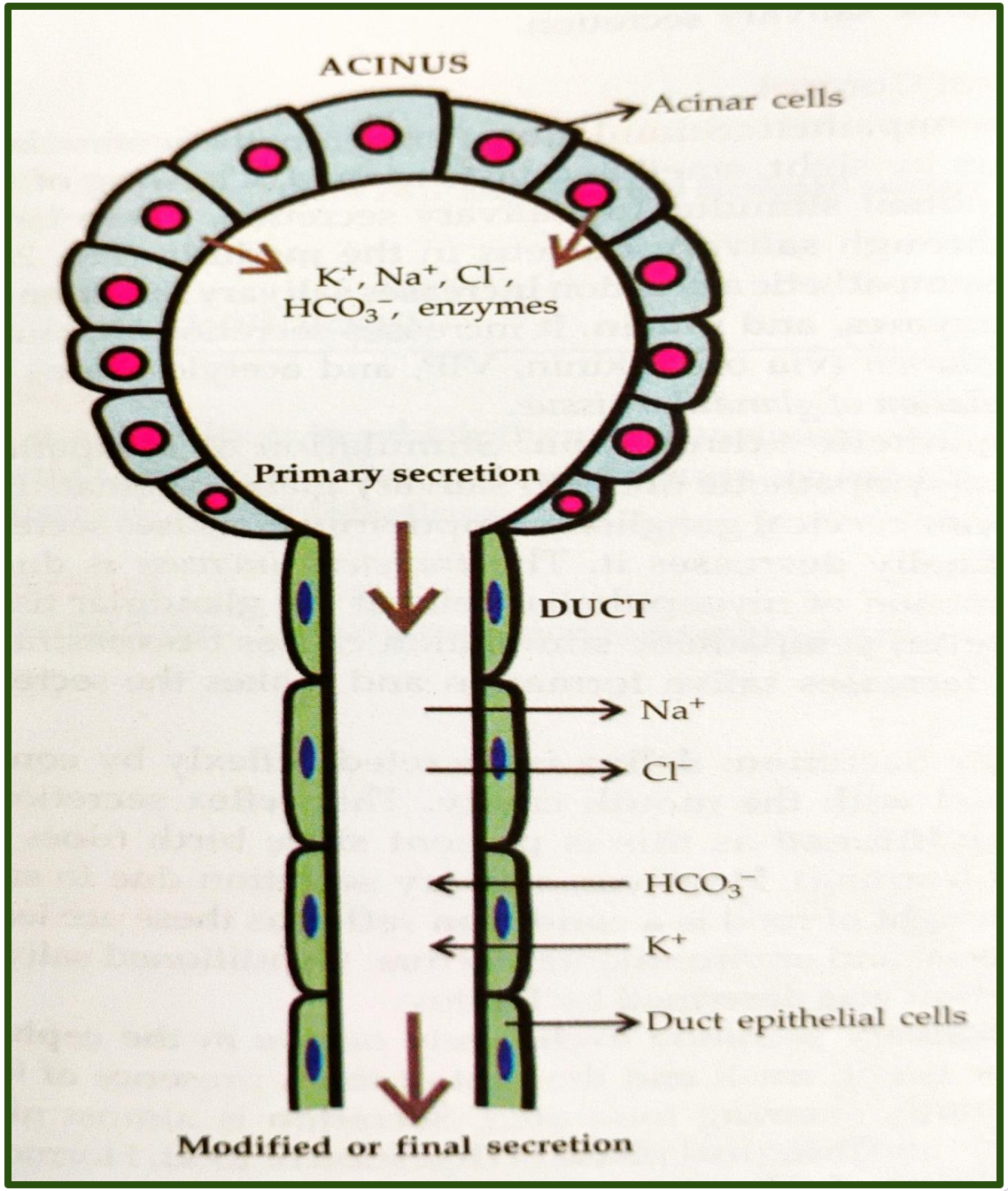
- Moistening & cleansing function-Keep oral& pharyngeal mucosa moist
- Antibacterial LYSOZYME is present.
- Helps in mastication, swallowing, speech.
- Mucin act as lubricant
- DIGESTION
  - SALIVARY AMYLASE convert cooked starch into MALTOSE
  - LINGUAL LIPASE

FUNCTIONS OF SALIVA

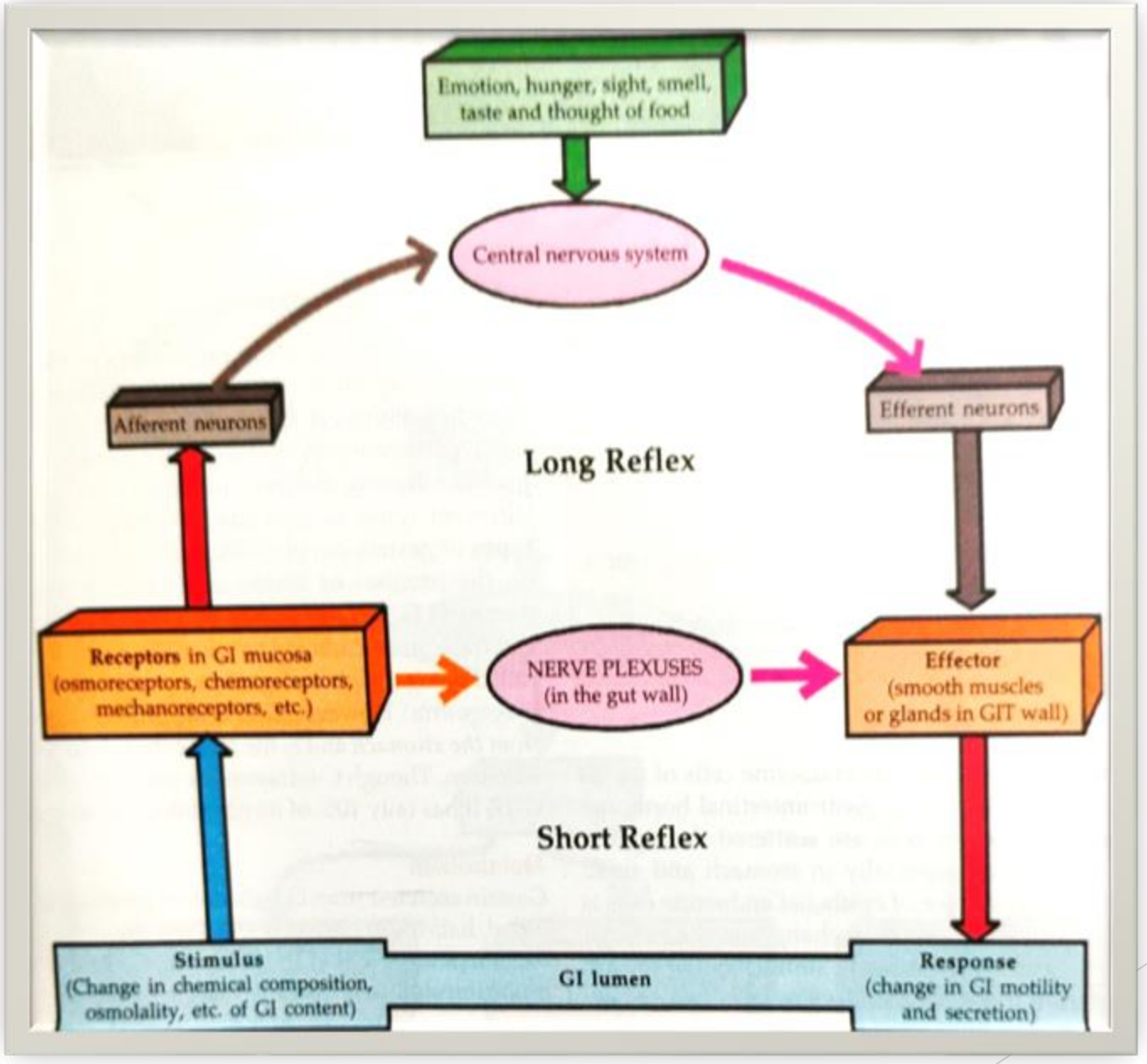
- BUFFERING ACTION- Bicarbonate, Phosphate & mucin,
  - Decrease in PH mobilise Calcium from teeth.
  - Increase PH -Calcium precipitation in teeth-TARTAR
  - Buffers neutralise acid regurgitation from stomach to oesophagus.
- SOLVENT- Stimulate taste buds.
- EXCRETORY FUNCTION-

  Lead, Mercury, Thiocyanate, Morphin, Streptomycin, Viruses of Rabies ,Mumps, Poliomyelitis.
- HELPS IN WATER BALANCE- Dryness of mouth-Thirst.

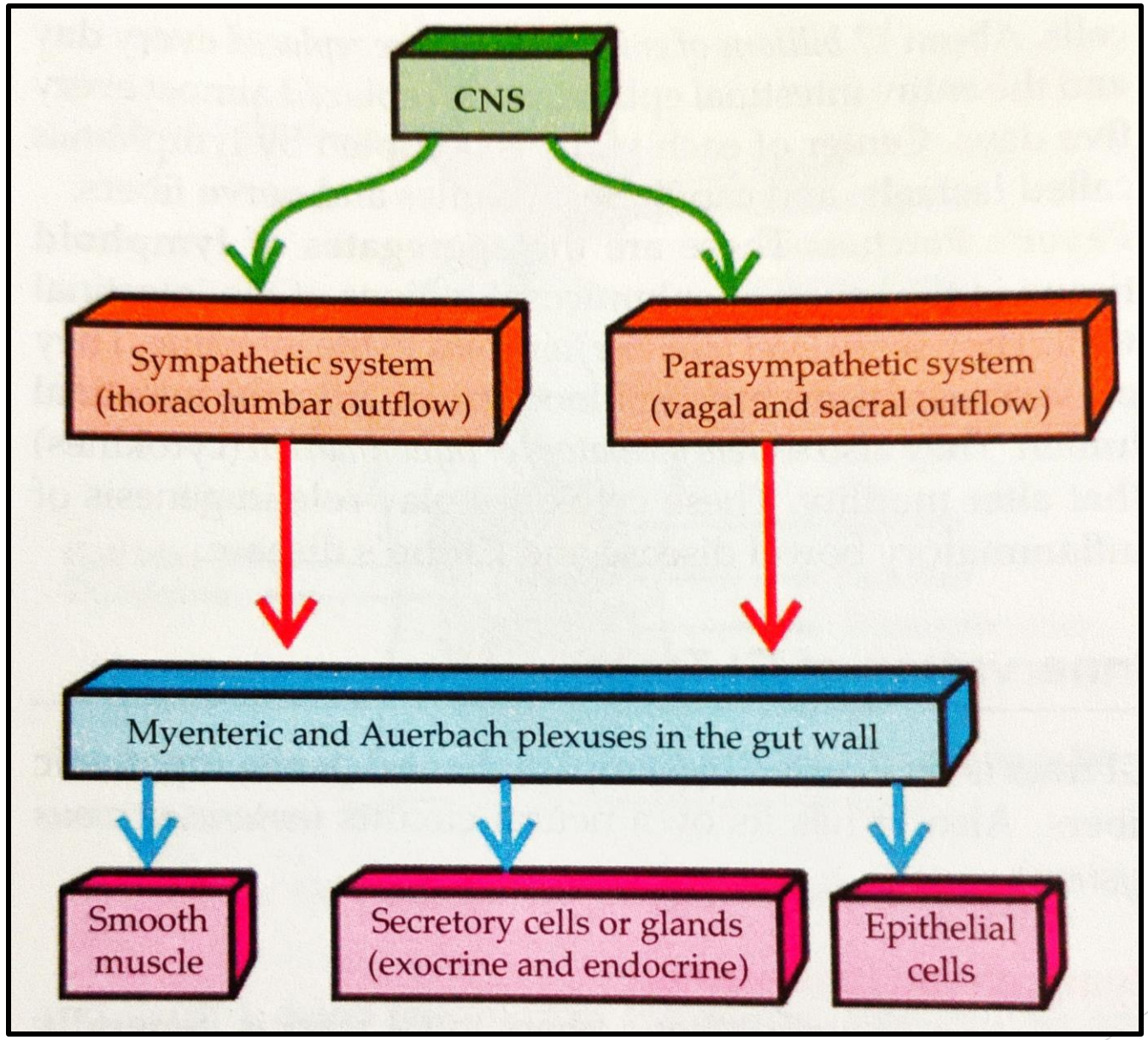

Sight, Taste, Smell, Chewing etc.
Through higher center
Medulla
Inferior/Superior salivary nuclei
9th CN
7th CN
Otic ganglion
Submandibular gland
Postganglionic parasympathetic fibers
Parotid gland
Submandibular ganglion
Increased salivary secretion rich in enzymes and mucin due to both acinar stimulation & vasodilation

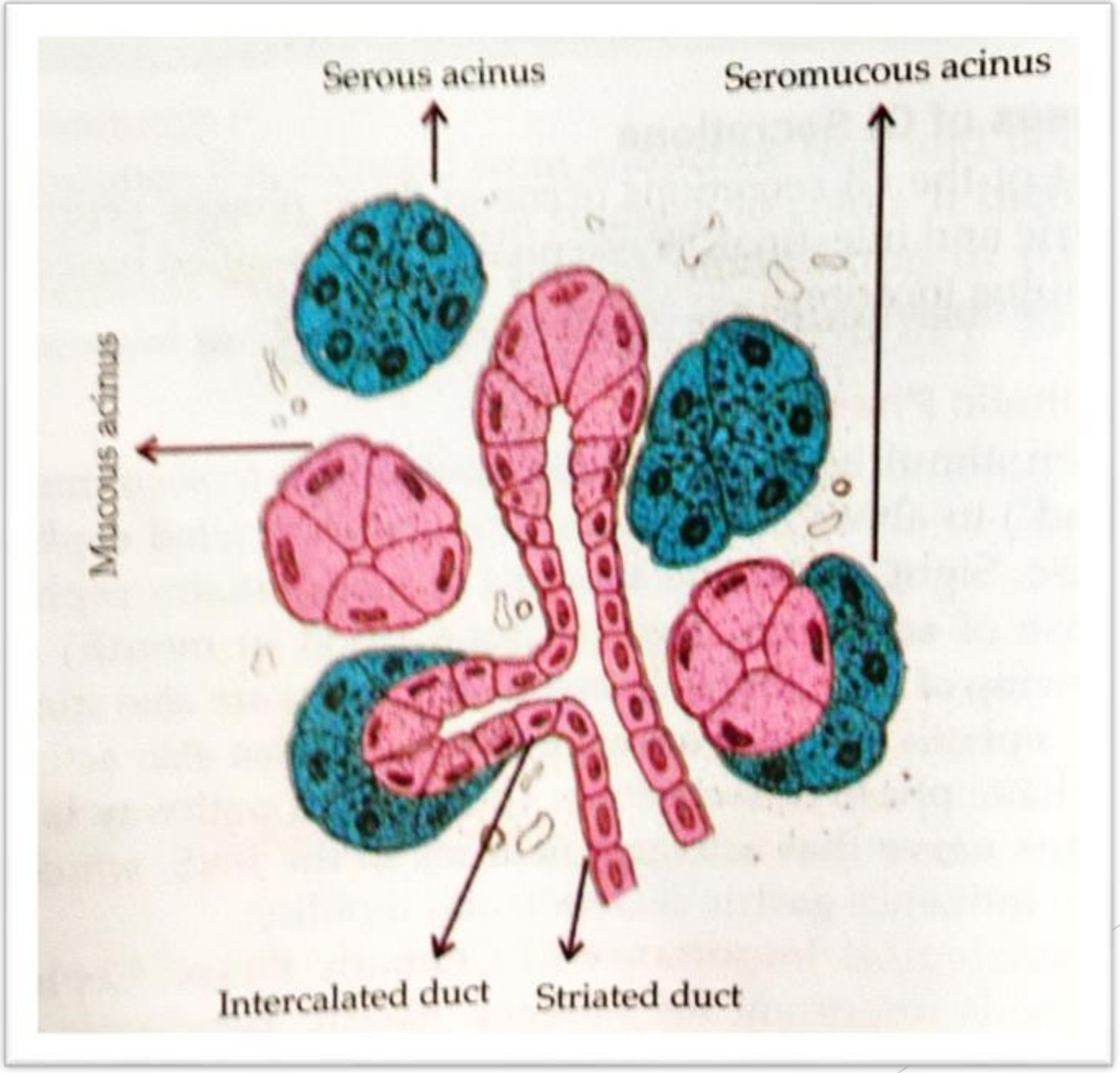

ACINUS
Acinar cells
K^+, Na^+, Cl^-,
HCO_3^-, enzymes
Primary secretion
DUCT
Na^+
Cl^-
HCO_3^-
K^+
Duct epithelial cells
Modified or final secretion

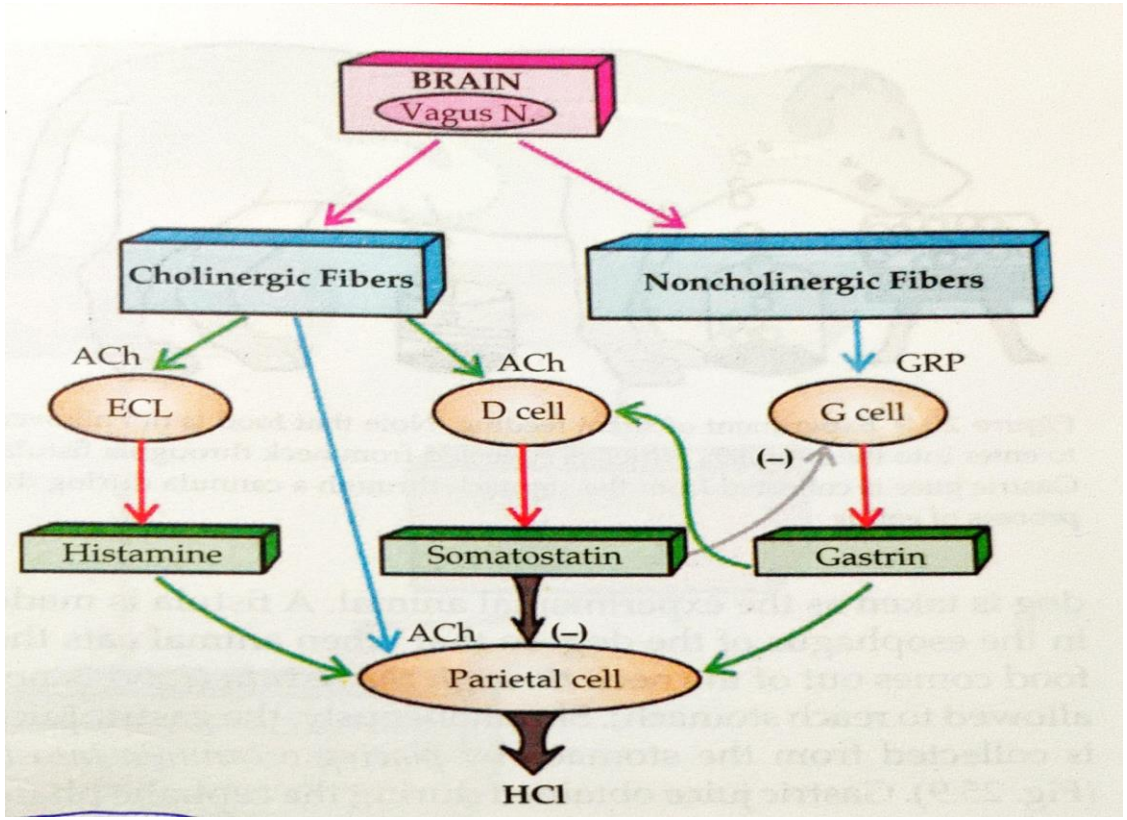
Short and Long reflexes of GI functions



Innervation of GI tract




Serous acinus
Seromucous acinus
Mucous acinus
Intercalated duct
Striated duct


BRAIN
Vagus N.
Cholinergic Fibers
Noncholinergic Fibers
ACh
ACh
GRP
ECL
D cell
G cell
(–)
Histamine
Somatostatin
Gastrin
ACh
(–)
Parietal cell
HCl

Gastric Secretions

STOMACH & ITS SECRETIONS

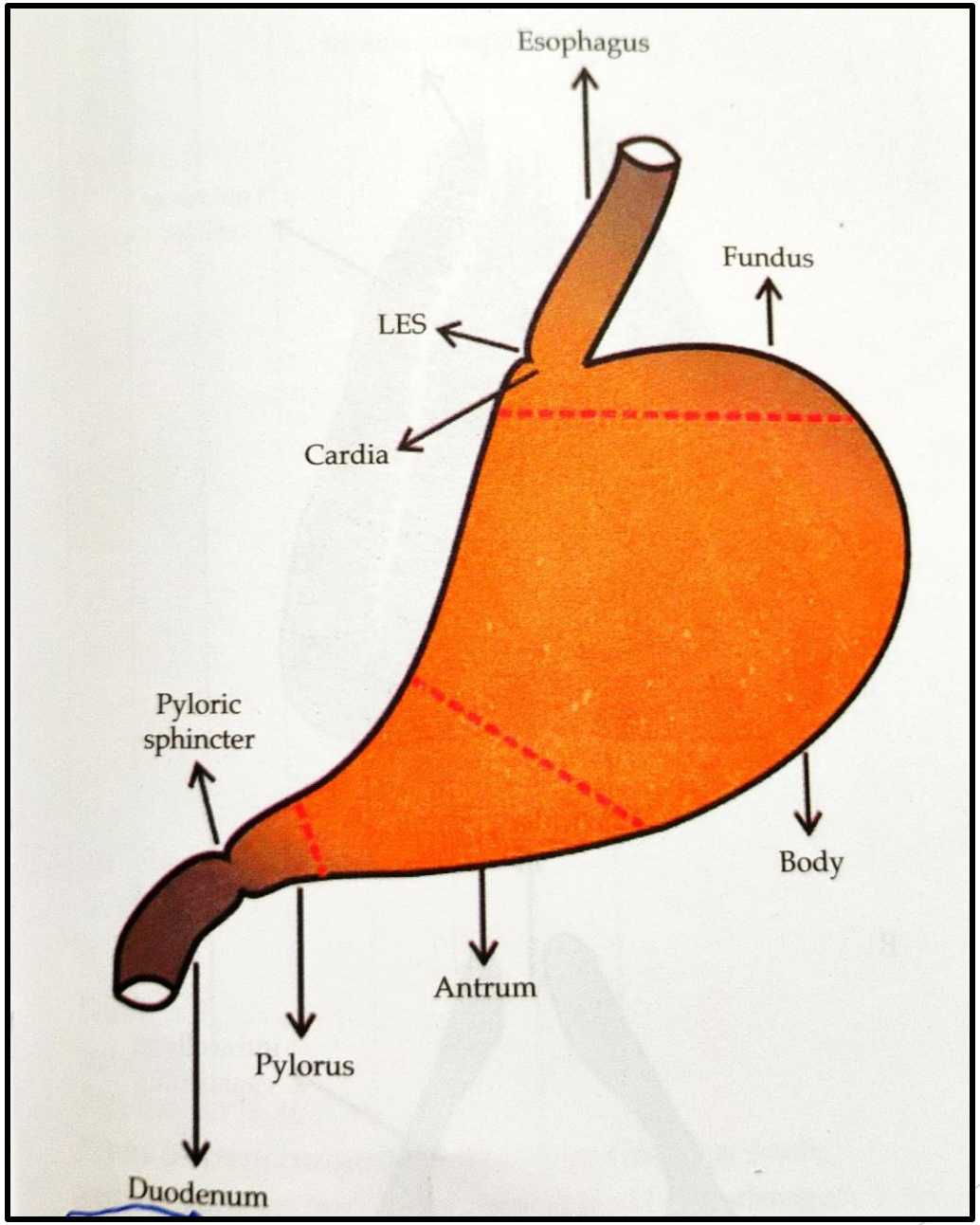
FUNCTIONAL ANATOMY

- FUNDUS, BODY, PYLORUS(Pyloric antrum,Pyloric canal)
- Cardiac end of stomach- Oesophagus opens.
- Pyloric end- Opens to duodenum.
- 2 curvatures- greater& Lesser.

HISTOLOGY

- Mucosa- Submucosa-Muscular layer, Serosa

Stomach


Esophagus
Fundus
LES
Cardia
Pyloric sphincter
Body
Antrum
Pylorus
Duodenum

GASTRIC GLANDS

1. FUNDIC GLANDS

- Most numerous
- Consist of body & neck
- Body contain,
  a) Chief or Peptic cell-Produce Pepsinogen
  b) Parietal or Oxyntic cell-secret HCl & Intrinsic factor
  c) Neck cell produce soluble MUCIN
- Mucous membrane in between glands lined by surface epithelial cells which produce Thick mucous & Bicarbonate

GASTRIC GLANDS

2. PYLORIC GLAND

- Structure similar
- No parietal cell.G cell present.
- NO HCl, Rich in Mucin & Gastrin

- ECL CELLS
- Vesicle& Granule containing cells
- Lie close to Gastric glands
- Contain receptors for GASTRIN& ACETYL CHOLINE.
- Release HISTAMINE- Increase HCl secretion

- NERVE SUPPLY
- Parasympathetic via VAGUS-synapse with ENS
- Supply different cells of gastric glands& stomach muscle.
- STIMULATION OF VAGAL FIBRES.

GASTRIC JUICE- COMPOSITION

- Mixture of secretions of Parietal cells, Peptic cells, mucous secreting cells & surface epithelial cells.
- Volume-1-1.5 L
- PH-1.5-6
- Ingredients-99.5% water ,0.5% solids.
- **INORGANIC-**

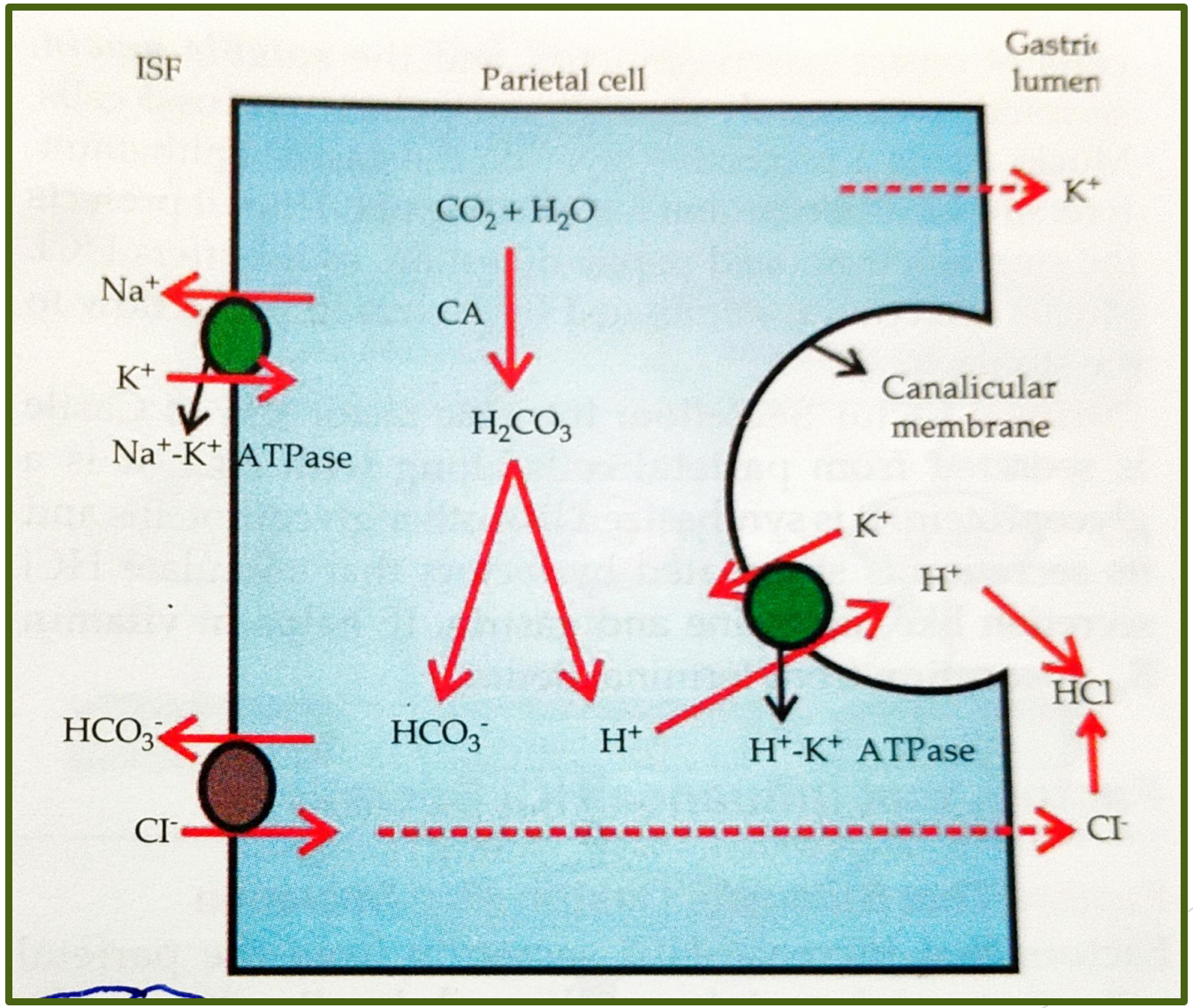
  HCl, Bicarbonate, Magnesium, Sodium, Potassium, Phosphate & Sulphate ions.
- **ORGANIC-**

  Enzymes, Mucous, Intrinsic factor.

MECHANISM OF HCl SECRETION

- HCl secreted from parietal cells located in fundus& body of stomach
- In the cytosol of parietal cell H^+ is derived from the break down of Carbonic acid.
- $CO_2 + H_2O \rightarrow H_2CO_3$
- Carbonic acid split into HCO_3^- & H^+.
- CO_2 is derived from intracellular metabolism& plasma.HCO3 formed is exchanged with Cl^- on the basolateral membrane of the cell by HCO_3 Cl^- exchanger

Mechanism of HCl Secretion



Mechanism of HCl Secretion

- Cl^- that enters parietal cell is passively transported into gastric lumen.
- In lumen Cl^- combines with H^+ forming HCl.
- For each H^+ secreted into gastric lumen, one HCO3 is reabsorbed into plasma.
- After a heavy meal → Increase HCl secretion → Increase HCO3 diffuse to blood→Excreted in urine → POST PRANDIAL ALKALINE TIDE
- H^+ K^+ pump actively pumps H^+ into gastric lumen. Potassium enters the cell.

REGULATION OF GASTRIC SECRETION

- NEURAL
- Local reflexes.
- Impulses from CNS via VAGUS NERVE.
- HORMONAL
- Stimulants, Inhibitors

PHASES OF SECRETION

1. **CEPHALIC PHASE**

- Vagally mediated responses induced by activity in CNS.
- 10% of total secretion.
- Starts before food reaches stomach & ends as food reaches stomach.
- Sight, smell, thought or food in mouth reflexly stimulate gastric juice secretion.

Mechanism-

- Due to conditioned reflex(sight, smell, thought of food) & Unconditioned reflex(when food is in mouth)

PHASES OF SECRETION

Afferent limb starts from receptors in Retina,Nasal cavity,Mouth.

Centre-Dorsal motor nucleus of vagus nerve.

Efferent limb-Vagal parasympathetic

Effector sites-ECL Cells,Parietal cells,Gcells.

Emotions & cephalic phase-

Anger& hostility increase secretion &Fear& depression decrease secretion.

HISTORY-At the end of 19^{th} century EVAN PAVLOV-SHAM FEEDING TECHNIQUE

PHASES OF SECRETION

GASTRIC PHASE

- 70% of gastric juice secretion.
- Neural &Hormonal mechanism
- NEURAL
- Distension of stomach wall by food→Stimulation of stretch receptors → Increase gastric juice secretion. It is by 2 reflexes

PHASES OF SECRETION

SHORT REFLEXES-

Reflex arc with in stomach wall.

Receptors----Meissners& Auebachs Plexus-------Synapse in ganglion cells------Post ganglionic fibres supply PARIETAL CELLS,CHIEF CELLS,MUCOUS CELLS,G CELLS

PHASES OF SECRETION

LONG VAGOVAGAL REFLEX

Receptors→Impulses via VAGUS to DORSAL MOTOR NUCLEUS OF VAGUS → VAGUS → GANGLION CELLS OF ENS → Post ganglionic fibres → Different cells of Gastric gland → Increase secretion

PHASES OF SECRETION

HORMONAL OR CHEMICAL REGULATION

- Amino acids, Polypeptides, Distension of pyloric antrum increase gastric acid secretion due to release of GASTRIN.
- Caffeine, alcohol also increase secretion.

PHASES OF SECRETION

INTESTINAL PHASE

- Partially digested food(peptides, amino acids)→increase gastrin secretion.
- Increased acidity → increase secretin → decrease HCl secretion.
- Increase fat in gastric chyme → decrease HCl secretion.
- Mediators → GIP,VIP,CCK-PZ, Neurotensin

FACTORS INFLUENCING HCl SECRETION

STIMULANTS

1) **HISTAMINE:**

- Secreted from ECL cells.
- It act on H2 receptors on parietal cells.
- Increase intracellular cyclic AMP $\rightarrow$ Stimulate H+K+ ATPse $\rightarrow$ Increase HCl secretion.
- Histamine release stimulated by gastrin& Acetyl choline.
- H2 blockers – Treatment for Peptic ulcer.

FACTORS INFLUENCING HCl SECRETION

2. GASTRIN

- Most powerful stimulant .
- From G Cells in pyloric glands of stomach & in first part of DUODENUM.
- Direct action on parietal cells, but mainly act via ECL Cells.
- Action by increasing intra cellular Calcium.
- Gastrin absorbed from GIT → Venous blood → Heart → Aorta → Gastric artery → Parietal cell → HCl secretion.
- Products of Protein digestion increase Gastrin secretion

FACTORS INFLUENCING HCl SECRETION

3. ACETYL CHOLINE

Vagal parasympathetic terminate on

a) Parietal cell-neurotransmitter-Acetyl choline.

b) ECL CELL—neurotransmitter-Acetyl choline.

c) G CELLS—neurotransmitter—GRP.

FACTORS INFLUENCING HCl SECRETION

- INHIBITION OF HCl SECRETION
- SOMATOSTATIN
- SECRETIN
- ACIDIC GASTRIC CHYME
- FAT IN CHYME
- HYPEROSMOTIC CHYME.

FUNCTIONS OF HCl

- Conversion of Pepsinogen to Pepsin,
- Bacteriostatic,
- Stimulate flow of bile,
- Stimulate secretion of some GI hormones
- Preventive role against Carcinoma stomach

Other constituents of Gastric Juice

- PEPSINOGEN is converted to pepsin in presence of HCl.
- Products of pepsin digestion-Polypeptides of diverse size.
- Pepsinogen secretion is stimulated by Gastrin& Histamine.

Other constituents of Gastric Juice

- **Mucus**
  - Secreted from mucus secreting cells in the neck region of gastric glands.
  - Forms a protective layer on the gastric epithelium.
  - Contain Bicarbonate ,protect stomach from Acid peptic digestion.

Other constituents of Gastric Juice

INTRINSIC FACTOR OF CASTLE

- Secreted from parietal cells along with HCl.
- Secretion stimulated by Histamine & Gastrin.
- It helps in Vitamin B_{12} absorption from terminal ileum.

PEPTIC ULCER

GASTRIC OR DUODENAL ULCER

PATHOPHYSIOLOGY

It is caused by

1. Decreased mucosal defence
2. Hypersecretion of Acid
3. Infection

CAUSES OF PEPTIC ULCER

1. DECREASED MUCOSAL DEFENCE

- Defence by mucous coat on the gastric epithelium.
- Mucus is a viscous gel containing MUCIN, PHOSPHOLIPID, ELECTROLYTES (mainly HCO_3) and WATER.
- Secreted by mucous cells.
- 0.2mm thick.
- Separates bicarbonate rich secretion of epithelial cells from acidic content of stomach.
- It protects mucosal epithelium from injury caused by acidic chyme.

1. DECREASED MUCOSAL DEFENCE

- When secretion of mucous impaired or Bicarbonate production decreased or when mucosal coat is mechanically damaged ACID & PEPSIN cause ulcer.
- Use of NSAIDS-Inhibit secretion of Mucous & Bicarbonate.
- Catecholamines inhibit mucous secretion→STRESS ULCER in chronic stress.

2.HYPERSECRETION OF ACID (HYPERCHLORHYDRIA)

- In chronic ANXIETY gasric acid secretion increases.
- Intake of spicy food increase acid secretion → Increased acid damage mucosal barrier
- ZOLLINGER-ELLISON SYNDROME → Gastrin secreting tumour of pancrease.

3.HELICOBACTER PYLORI INFECTION

- The major cause of peptic ulcer.
- Gram negative Bacillus that secrete UREASE.
- Urease convert urea to Ammonia& CO_2 .Ammonia buffer the acid surrounding bacteria.
- Bacteria colonizes the antral mucosa & cause local inflammation & disrupt immune responses.
- It inhibit somatostatin secretion from D cells $\rightarrow$ Facilitate Gastrin $\rightarrow$ Increased HCl secretion $\rightarrow$ Gastritis& later ulcer.
- So antibiotic therapy to kill H.pylori.

Features of Peptic Ulcer

- Upper abdominal pain(Epigastric pain)
- Hemetemesis, Vomiting, Malena.

Treatment

1. **Specific treatment.**

   H_2 Receptor Antogonist Ex: Ranitidine

   Block H_2 receptors & inhibit Histamine secretion.

2. **Proton pump blockers(H+ K+ ATPase inhibitor)**

   The final step of acid secretion is inhibited. Most effective medicine. Ex: Omeprazole.

3. **Sucralfate.** Provide protective layer on the ulcer**.**

Treatment

4.Gastrin blockers -Proglumide-Gastrin blocker.

5.Antibiotics-Amoxicillin.

6.Muscarinic blockers- Noncholinergic vagal innervation dominate over the cholinergic innervation for acid secretion. So muscarinic blockers are not used in peptic ulcer treatment.

Non-Specific measures

- Antacids
- Reduce stress-Yoga
- Adequate sleep, rest, regulation of diet, withdrawl of drugs like NSAIDS
- Use of cold milk, avoidance of spicy food& alcohol.

Surgical treatment

- Vagotomy
- Gastrectomy-Partial gastrectomy removes the antral portion of stomach(contain G Cells)