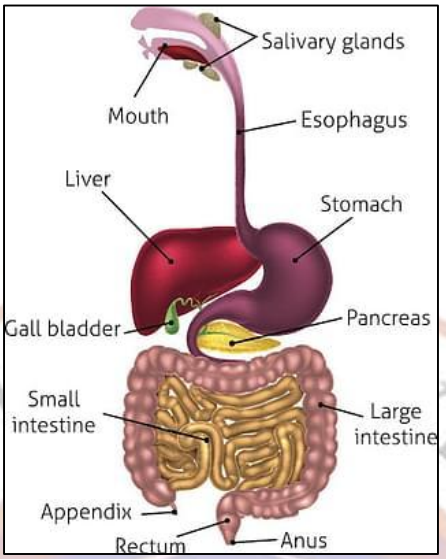
DIGESTIVE SYSTEM



Digestive System

- Digestive system is a system that consists of a group of organs that break down the food we eat into smaller molecules that can be used by body cells.

Components of Digestive System

The digestive system includes:

1. Gastrointestinal tract (GIT) or alimentary canal
2. Accessory organs

Basic Processes / Functions of Digestive System

1. Ingestion

- It is the taking of food into the mouth or alimentary tract.
- It includes eating or drinking.

2. Secretion

- It is the release of water, acids, enzymes, buffers, and salts into the lumen of the GIT.
- Cells within the walls of the GIT and accessory digestive organs secrete a total of about **7 liters** of water, acid, buffers, and enzymes into the lumen of the tract.

3. Propulsion / Motility

- It is the movement of food through the digestive tract.
- Includes:
  - Swallowing (voluntary)
  - Peristalsis (involuntary)
- Alternating contractions and relaxations of smooth muscle in the walls of the GIT mix food and secretions and move them toward the anus.
- This capability is called **motility**.

4. Digestion

- It is the process of breaking down ingested food into small molecules that can be used by body cells.
- Two types:
  1. Mechanical digestion
  2. Chemical digestion

Mechanical Digestion

- Physical digestion carried out by teeth.
- Teeth cut and grind food before swallowing (chewing).

Chemical Digestion

- Breakdown of complex compounds into absorbable forms.
- Large carbohydrate, lipid, protein, and nucleic acid molecules are split into smaller molecules by **hydrolysis**.
- Occurs by action of enzymes present in secretions of glands and accessory organs.

5. Absorption

- Movement of products of digestion from lumen of GI tract into blood or lymph.
- Enables circulation and use by body cells.

6. Elimination

- Removal of indigestible wastes through the anus in the form of feces.
- Also called **defecation**.
- Eliminated material is called **feces or stool**.

ORGANS OF THE DIGESTIVE SYSTEM

Alimentary Canal / Gastrointestinal Tract (GIT)

- A continuous long tube through which food passes.
- Extends from mouth to anus.

- Length in adults: **5–7 meters**.
- Structurally similar but functionally different organs.

Organs of GIT

- Mouth
- Pharynx
- Oesophagus
- Stomach
- Small intestine
- Large intestine
- Rectum and anal canal

Accessory Organs

- Teeth – aid in physical breakdown of food
- Tongue – assists in chewing and swallowing
- Salivary glands – produce or store secretion that flows to GIT
- Liver – aids in chemical breakdown of food
- Gall bladder
- Pancreas

Layers of GIT / Basic Structure of Alimentary Canal

- Wall of GIT from lower esophagus to anal canal has **four layers**.
- From deep to superficial:
    1. Mucosa
    2. Submucosa
    3. Muscle layer (Muscularis)
    4. Serosa / Adventitia

MUCOSA

- Inner lining of GIT; a mucous membrane.
- Composed of:
    1. Epithelium – direct contact with contents of GIT
    2. Connective tissue – lamina propria
    3. Thin smooth muscle layer – muscularis mucosae

Epithelium

- Mouth, pharynx, esophagus, anal canal:
    - Non-keratinized stratified squamous epithelium (protection)
- Stomach and intestines:
    - Simple columnar epithelium (secretion and absorption)

- Exocrine cells:
  - Secrete mucus and fluid into lumen
- Endocrine cells:
  - Enteroendocrine cells secrete hormones

Lamina Propria

- Areolar connective tissue containing blood and lymph vessels
- Supports epithelium and binds it to muscularis mucosae
- Contains MALT (mucosa-associated lymphoid tissue)
- Provides immunity and protection
- Present throughout GIT, especially:
  - Tonsils
  - Small intestine
  - Appendix
  - Large intestine

Muscularis Mucosae

- Throws mucous membrane of stomach and small intestine into many small folds
- Increases surface area for digestion and absorption

SUBMUCOSA

- Made of loose areolar connective tissue
- Binds mucosa to muscularis
- Contains:
  - Blood vessels
  - Lymph vessels
  - Glands
  - Lymph tissue
- Glands are present in this layer
- Contains **submucosal plexus**
  - Network of neurons
  - Includes sympathetic and parasympathetic nerves
  - Supplies mucosal lining

MUSCULARIS (MUSCLE LAYER)

- Mouth, pharynx, superior and middle parts of esophagus, external anal sphincter:
  - Skeletal muscle (voluntary swallowing and defecation)
- Rest of GIT:
  - Smooth muscle (involuntary)

Layers

- Inner circular muscle fibers
- Outer longitudinal muscle fibers

Myenteric Plexus

- Located between circular and longitudinal layers
- Contains blood vessels, lymph vessels, sympathetic and parasympathetic fibers
- Controls:
    - Contraction and relaxation
    - Rhythmic waves called **peristalsis**
    - Mixing of food
    - Propulsion of contents

Sphincters

- Thickened rings of circular muscle
- Act like valves
- Control onward movement of food

ADVENTITIA / SEROSA

- Superficial layer of parts of GIT in abdominal cavity
- Composed of:
    1. Simple squamous epithelium
    2. Areolar connective tissue
- Serosa is also called **visceral peritoneum**
- Esophagus lacks serosa
- Instead, it has a single layer of areolar connective tissue called **adventitia**

PERITONEUM

- Largest serous membrane of the body
- Lines abdominal cavity and covers abdominal organs
- Forms a closed sac
- Made of:
    - Simple squamous epithelium
    - Supporting areolar connective tissue

Layers

- Parietal peritoneum – lines abdominal wall
- Visceral peritoneum – covers organs and forms serosa
- Space between layers:
    - Peritoneal cavity

- Contains lubricating serous fluid

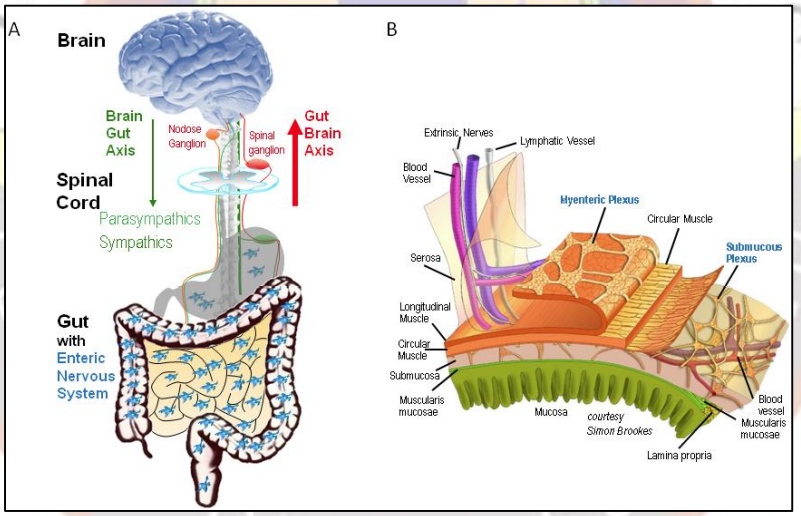
Functions

- Contains folds and loops
- Binds organs to each other and to abdominal wall
- Contains blood vessels, lymph vessels, and nerves

Organ Position

- Intraperitoneal organs:
  - Almost completely covered by visceral peritoneum
  - Examples: stomach, intestines, liver
- Retroperitoneal organs:
  - Covered only on anterior side
  - Examples: kidneys, spleen, pancreas

NERVE SUPPLY OF GIT



Regulation by:

1. Intrinsic neurons – Enteric Nervous System (ENS)
2. Extrinsic neurons – Autonomic Nervous System (ANS)

ENTERIC NERVOUS SYSTEM (Brain of Gut)

- Contains about **100 million neurons**
- Extends from esophagus to anus
- Arranged in two plexuses:
  1. Myenteric (Auerbach's) plexus

2. Submucosal (Meissner's) plexus

Neurons present

- Motor neurons
- Sensory neurons
- Interneurons

Functions

- Myenteric plexus:
  - Controls GIT motility
- Submucosal plexus:
  - Controls secretion
- Sensory neurons supply mucosal epithelium

Sensory Receptors

1. Chemoreceptors – respond to chemicals in food
2. Mechanoreceptors – respond to stretch of GI walls

AUTONOMIC NERVOUS SYSTEM

Parasympathetic Supply

- Vagus nerves supply most of alimentary canal and accessory organs
- Sacral nerves supply distal half of large intestine

Effects:

- Increases peristalsis
- Increases GI secretions

Sympathetic Supply

- Arises from thoracic and lumbar spinal cord
- Forms plexuses in thorax, abdomen, pelvis

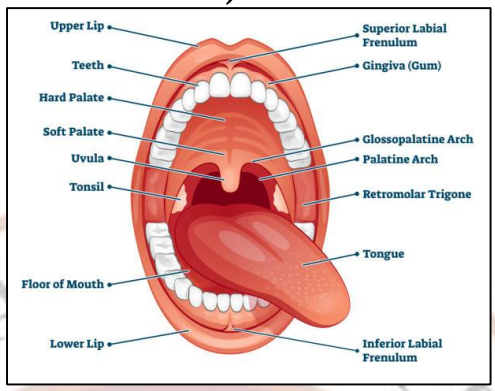
Effects:

- Decreases muscular activity
- Decreases glandular secretion

GASTROINTESTINAL REFLEX PATHWAYS

- ENS neurons form GI reflex pathways
- Regulate GI secretion and motility in response to lumen stimuli

I. MOUTH (ORAL / BUCCAL CAVITY)



Boundaries

- Anterior: Lips
- Posterior: Oropharynx
- Lateral: Cheek muscles
- Superior: Hard and soft palate
- Inferior: Tongue and floor of mouth

Lining

- Stratified squamous epithelium
- Mucus-secreting glands present

Cheeks

- External surface: skin
- Internal surface: non-keratinized stratified squamous epithelium

Lips (Labia)

- External: skin
- Internal: mucous membrane

Labial Frenulum

- Midline fold of mucous membrane
- Attaches inner surface of lip to gum

Vestibule

- Space between lips/cheeks and gums/teeth

Oral Cavity Proper

- Remaining internal space

PALATE

- Forms roof of mouth

Hard Palate

- Anterior
- Formed by maxilla and palatine bones
- Arch shaped

Soft Palate

- Posterior
- Muscular
- Blends with pharyngeal walls

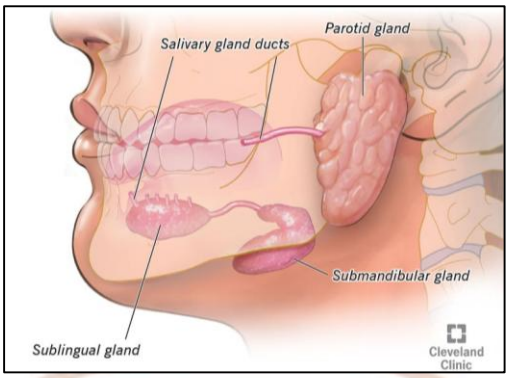
Uvula

- Finger-like muscular projection
- Hangs from soft palate

Palatine Arches

- Four mucosal folds from uvula
  - Two anterior
  - Two posterior
- Palatine tonsils located between arches

SALIVARY GLANDS



Types

1. Major salivary glands
2. Minor salivary glands

Major Salivary Glands (3 pairs)

1. Parotid glands
2. Submandibular glands
3. Sublingual glands

Minor Salivary Glands

- Numerous
- Scattered throughout mouth and tongue

Parotid Glands

- Located below external acoustic meatus
- Parotid duct opens opposite second upper molar

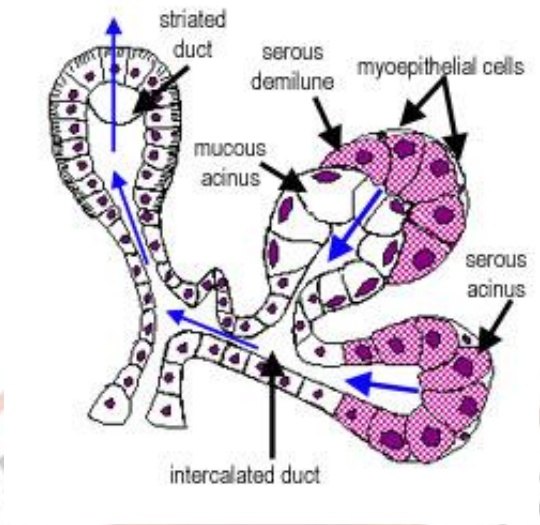
Submandibular Glands

- Located under angle of jaw
- Ducts open on floor of mouth near frenulum of tongue

Sublingual Glands

- Beneath tongue
- Superior to submandibular glands
- Multiple small ducts open into floor of mouth

STRUCTURE OF SALIVARY GLANDS



- Surrounded by fibrous capsule
- Contain three types of cells:
  1. Acinar cells
  2. Ductal cells
  3. Myoepithelial cells
- All salivary glands have:
  - Branched ducts opening into oral cavity
  - Secretory acini producing saliva
- Saliva released into ductules → larger ducts → mouth

COMPOSITION OF SALIVA

- Saliva is a combination of secretions from:
  - Salivary glands
  - Mucus-secreting glands of oral mucosa
- Average daily secretion: **1.5 litres**

Composition

- **99.5% water**
- **0.5% solutes**, which include:
  - Ions: Na^+, K^+, Cl^-, HCO_3^-, PO_4^{3-}
  - Dissolved gases
  - Organic substances: urea, uric acid
  - Mucus
  - Immunoglobulins
  - Lysozyme – bacteriolytic enzyme
  - Salivary amylase – digestive enzyme

SECRETION OF SALIVA

- Process of secretion of saliva is called **salivation**
- Controlled by **Autonomic Nervous System**

Parasympathetic Stimulation

- Promotes profuse secretion of watery saliva
- Releases low amounts of enzymes

Sympathetic Stimulation

- Dominates during stress
- Causes dryness of mouth

Nerves Involved

- Facial nerve (VII)
- Glossopharyngeal nerve (IX)

Reflexes

- Reflex secretion occurs when food enters mouth
- Conditioned reflex:
  - Sight
  - Smell
  - Sound
  - Thought of food

FUNCTIONS OF SALIVA

1. Chemical Digestion of Polysaccharides

- Chloride ions activate salivary amylase
- Starch → disaccharide (maltose)
- Optimum pH: **6.8**
- Normal pH range: **5.8 – 7.4**

2. Lubrication of Food

- Moistens food
- Helps form bolus

3. Cleaning and Lubrication of Mouth

- Prevents damage to mucous membrane by rough food

4. Non-Specific Defence

- Lysozyme and immunoglobulins destroy microbes

5. Taste

- Taste buds stimulated only by chemicals in solution

6. Removal of Waste Molecules

TONGUE

- Accessory digestive organ
- Composed of **skeletal (voluntary) muscle**
- Covered by mucous membrane
- Attached to:
  - Hyoid bone
  - Styloid process of temporal bone
  - Mandible
- Frenulum:
  - Fold of mucous membrane
  - Attaches tongue to floor of mouth

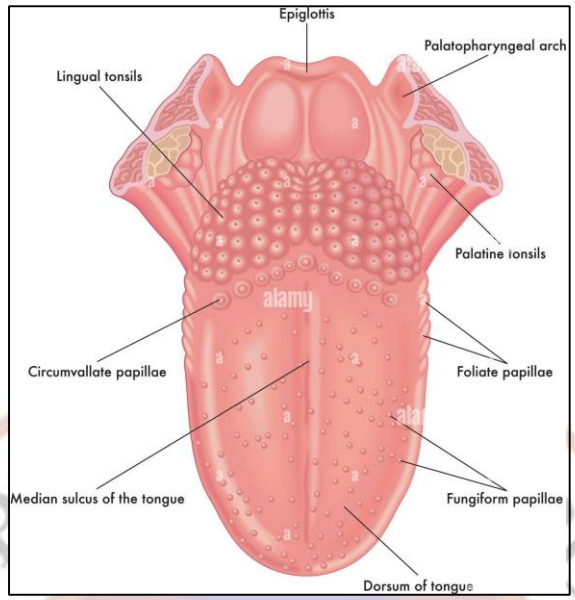
Surface Features

- Superior surface:
  - Stratified squamous epithelium
  - Numerous papillae
  - Taste buds present
- Divided into symmetrical halves by median septum

Muscles

- **Extrinsic muscles**
  - Move tongue side to side and in/out
  - Maneuver food for chewing and swallowing
- **Intrinsic muscles**
  - Alter shape and size
  - Important for speech and swallowing

FUNCTIONS OF TONGUE



- Chewing (mastication)
- Swallowing (deglutition)
- Speech
- Taste

TEETH

- Teeth (dentes) are accessory digestive organs
- Embedded in alveoli of mandible and maxilla

Dentitions

1. Temporary (deciduous) teeth
2. Permanent teeth

Temporary Teeth

- Total: **20 teeth**
- 10 in each jaw
- Eruption: 6 months
- Completed by: 24 months
- Lost between: 6–12 years

Permanent Teeth

- Replace deciduous teeth between 6–13 years
- Total: **32 teeth**
- Completed by age 20

- **Third molars (wisdom teeth) erupt last**

Jaws	Deciduous Teeth	Permanent Teeth	Functions
Upper Jaw	Incisors (c) – 2 Incisors (L) – 2	Incisors (c) – 2 Incisors (L) – 2	To bite off pieces of food
Lower Jaw	Incisors (c) – 2 Incisors (L) – 2	Incisors (c) – 2 Incisors (L) – 2	
Upper Jaw	Canines – 2	Canines – 2	To grasp and tear food
Lower Jaw	Canines – 2	Canines – 2	
Upper Jaw	Premolars – 0	Premolars – 4	To grind food particles
Lower Jaw	Premolars – 0	Premolars – 4	
Upper Jaw	Molars – 4	Molars – 6	To grind food particles
Lower Jaw	Molars – 4	Molars – 6	
Total	**20**	**32**	

DENTAL FORMULA AND FUNCTIONS

Upper Jaw

- Incisors: 4 (cut food)
- Canines: 2 (tear food)
- Premolars: 4 (grind food)
- Molars: 6 (grind food)

Lower Jaw

- Same distribution as upper jaw

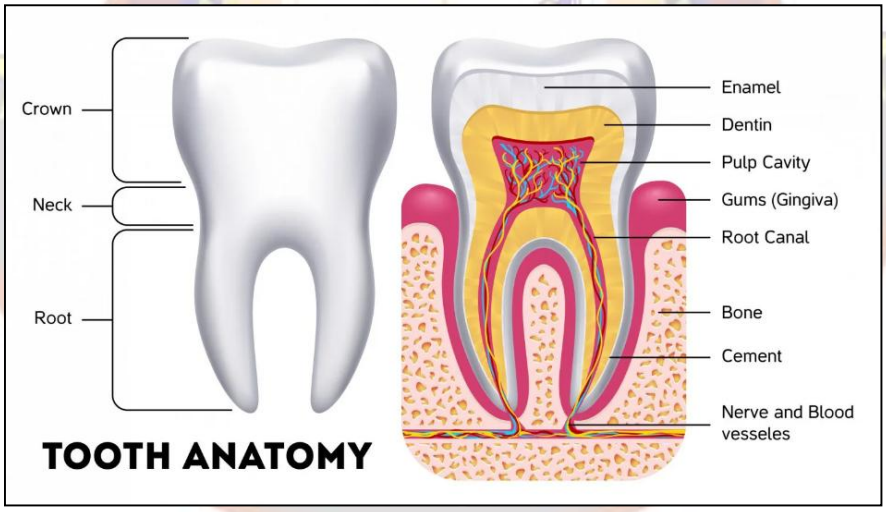
Functions

- Incisors – biting
- Canines – tearing
- Premolars – grinding
- Molars – grinding

STRUCTURE / PARTS OF A TOOTH

Parts

1. **Crown**
   - Visible part above gum
2. **Root**
   - Embedded in bone socket
3. **Neck**
   - Constricted region between crown and root



Internal Structure

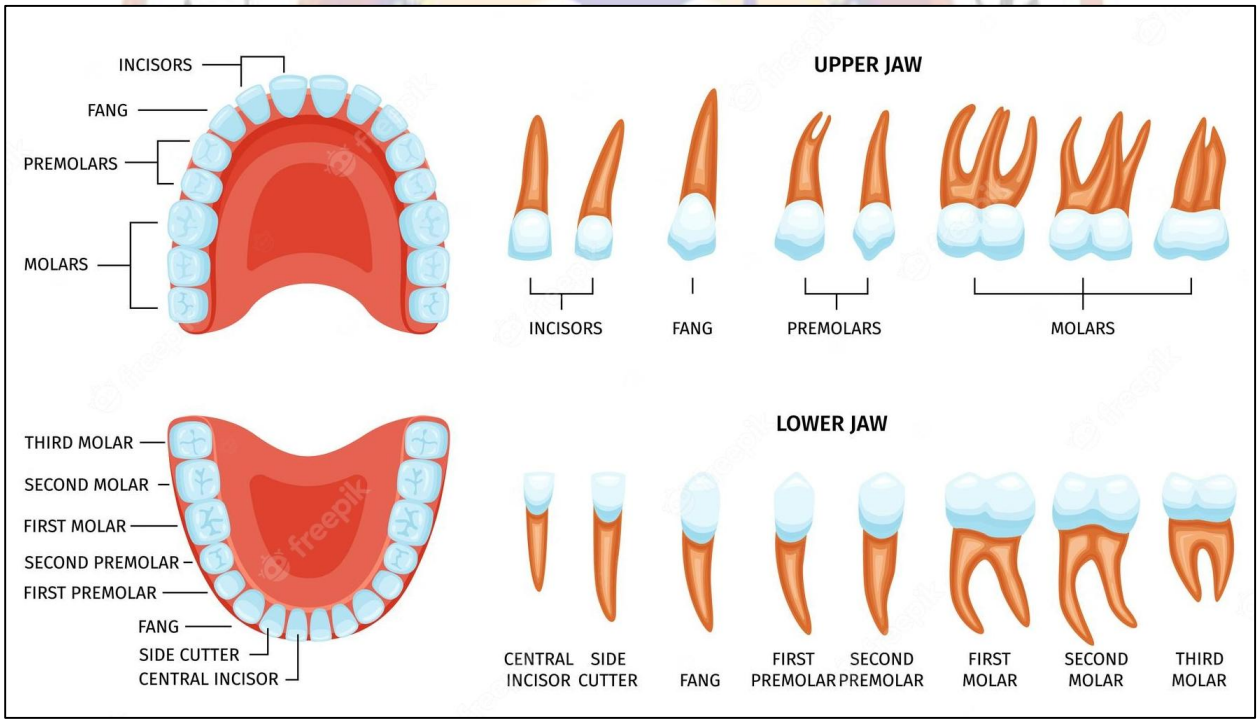
- Pulp cavity (upper)
- Root canal (lower)
- Apical foramen:
  - Opening at base of root
  - Entry of blood vessels, lymph vessels, nerves

Tissues

- Pulp:
  - Connective tissue with vessels and nerves
- Dentine:
  - Hard ivory-like substance
  - Forms bulk of tooth
- Enamel:
  - Covers crown
  - Hardest substance in body
  - Made of calcium phosphate and calcium carbonate
- Cementum:
  - Covers dentine of root

Functions

- Cutting
- Tearing
- Shredding
- Grinding
- Chewing



DIGESTION IN MOUTH

Mechanical Digestion

- Mastication by teeth
- Tongue manipulates food

- Food mixed with saliva
- Forms **bolus**

Chemical Digestion

- Enzymes involved:
  - Salivary amylase
  - Lingual lipase

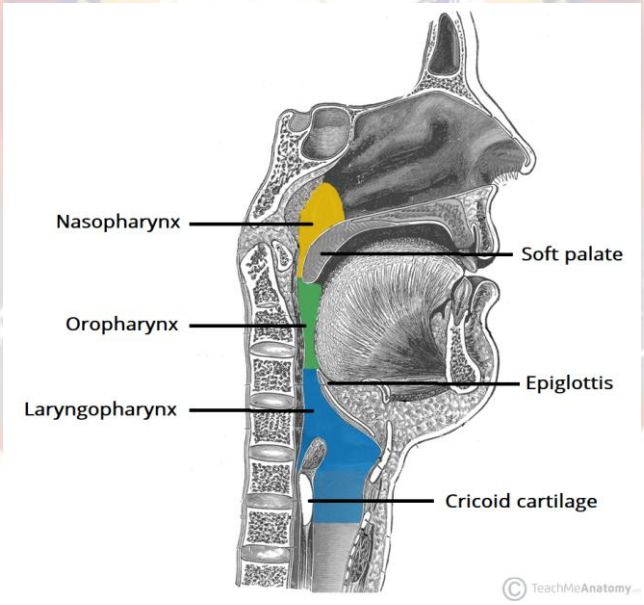
Salivary Amylase

- Initiates starch digestion
- Starch → maltose, maltotriose, α-dextrins

Lingual Lipase

- Activated in acidic environment of stomach
- Triglycerides → fatty acids + diglycerides

II. PHARYNX



- Funnel-shaped muscular tube
- Extends from internal nares to:
  - Esophagus posteriorly
  - Larynx anteriorly
- Lined by stratified squamous epithelium

Divisions

1. Nasopharynx – respiration only
2. Oropharynx – digestion and respiration
3. Laryngopharynx – digestion and respiration

- Swallowed food passes:
  - Mouth → oropharynx → laryngopharynx → esophagus
- Muscular contractions propel food downward

III. ESOPHAGUS

General Features

- Collapsible muscular tube
- Length: **25 cm**
- Diameter: **2 cm**
- Located:
  - Anterior to vertebral column
  - Posterior to trachea
- Passes through diaphragm via **esophageal hiatus**
- Curves upward before entering stomach to prevent reflux

Sphincters

Upper Esophageal Sphincter

- Regulates entry of food
- Prevents air entry and aspiration

Lower Esophageal Sphincter

- Regulates entry into stomach
- Prevents reflux of gastric contents

Structure of Esophagus

- **Adventitia**
  - Areolar connective tissue
- **Muscularis**
  - Upper 1/3 – skeletal muscle
  - Middle 1/3 – skeletal + smooth muscle
  - Lower 1/3 – smooth muscle
- **Submucosa**
  - Areolar connective tissue
  - Blood vessels

    - Mucous glands
- **Mucosa**
    - Non-keratinized stratified squamous epithelium
    - Lamina propria
    - Muscularis mucosa

Functions

- Secretes mucus
- Transports food
- No digestion
- No absorption

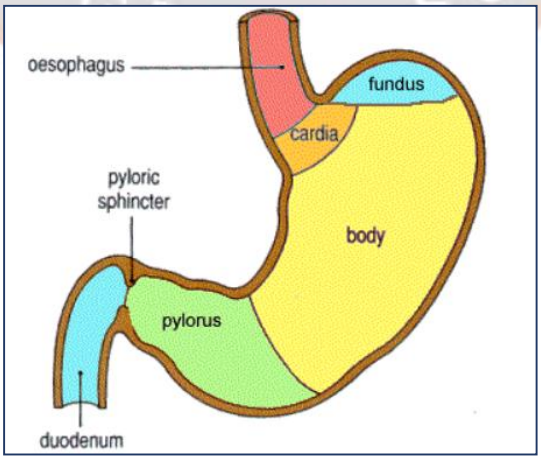
DEGLUTITION (SWALLOWING)

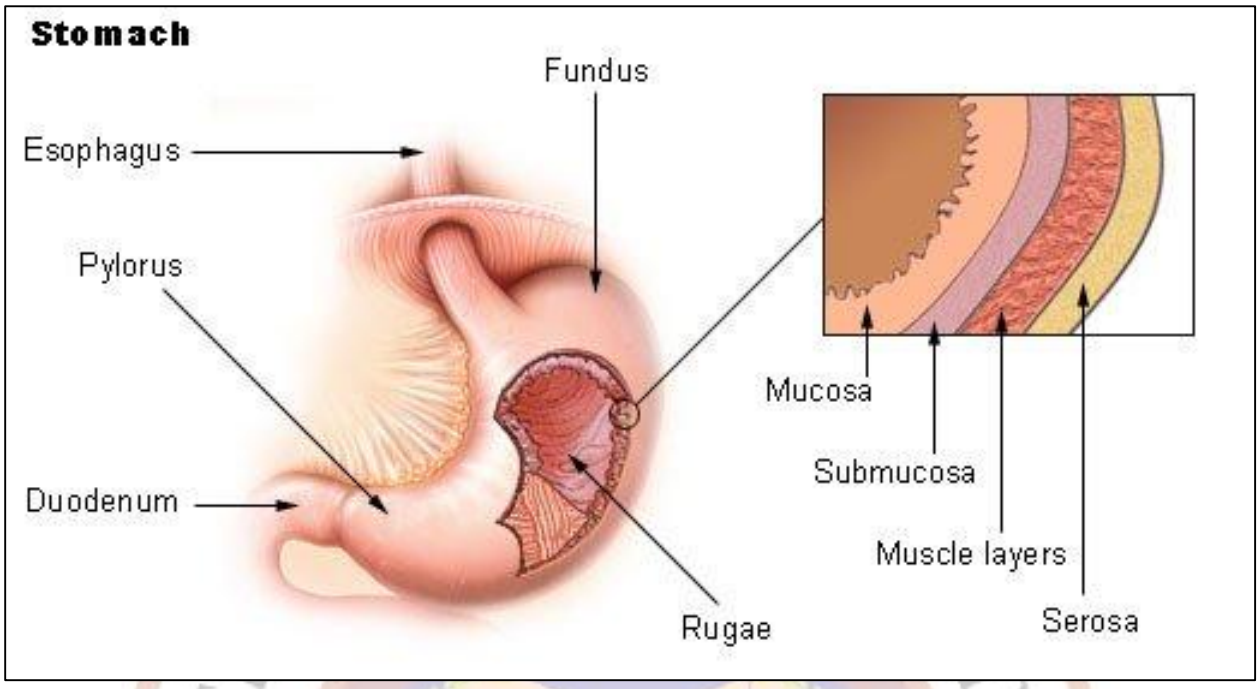
- Movement of food from mouth to stomach
- Involves mouth, pharynx, esophagus
- Assisted by saliva and mucus

Stages

1. **Voluntary stage**
    - Tongue pushes bolus into oropharynx
2. **Pharyngeal stage**
    - Involuntary passage to esophagus
3. **Esophageal stage**
    - Peristalsis moves bolus to stomach
    - Lower sphincter relaxes
    - Prevents reflux

STOMACH





General Features

- J-shaped
- Most distensible part of GIT
- Connects esophagus to duodenum
- Mixing chamber and reservoir
- Converts bolus to liquid chyme
- Protein digestion begins

Relations

- Anterior: liver, abdominal wall
- Posterior: pancreas, spleen, kidney
- Superior: diaphragm
- Inferior: transverse colon
- Left: spleen
- Right: liver, duodenum

Anatomy of Stomach

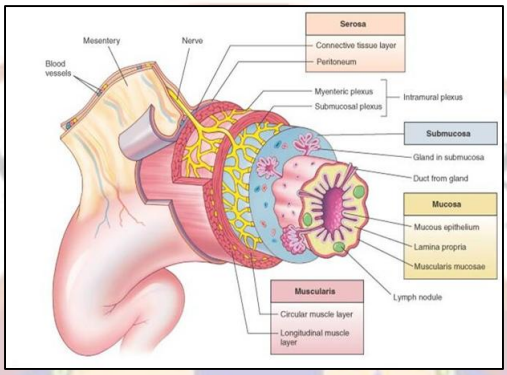
- Four regions:

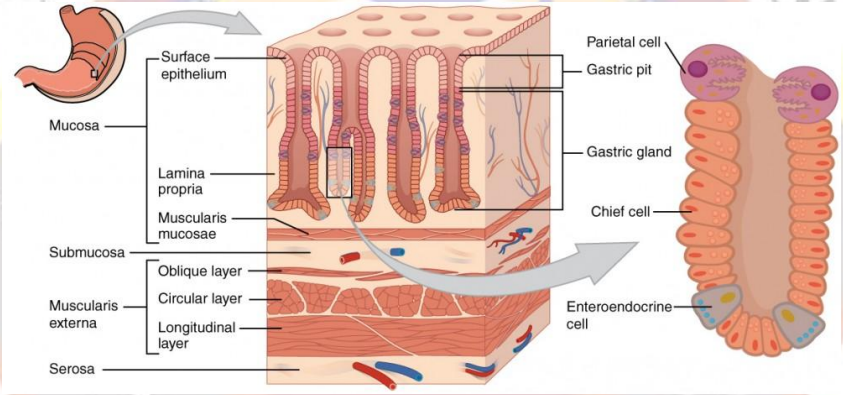
1. Cardia
2. Fundus
3. Body
4. Pylorus

Features

- Rugae present when empty
- Lesser curvature – concave
- Greater curvature – convex
- Pyloric sphincter regulates exit

Histology





Mucosa

- Simple columnar epithelium
- Gastric pits and gastric glands
- Cells:
    - Surface mucous cells – mucus
    - Parietal cells – HCl and intrinsic factor
    - Chief cells – pepsinogen, gastric lipase
    - Enteroendocrine cells – gastrin

Submucosa

- Areolar connective tissue
- Submucosal plexus

Muscularis

- Three smooth muscle layers:
  - Outer longitudinal
  - Middle circular
  - Inner oblique
- Enables churning and peristalsis

Serosa

- Visceral peritoneum

Functions of Stomach

- Mechanical digestion
- Reservoir for food
- Secretion of gastric juice
- Chemical digestion
- Non-specific defence
- Regulation of chyme release

GASTRIC JUICE

- About **2 L/day**
- Components:
  - Water
  - Mineral salts
  - Mucus
  - HCl
  - Intrinsic factor
  - Pepsinogen
  - Lipase
- Parasympathetic stimulation ↑ secretion
- Sympathetic stimulation ↓ secretion

- Food may remain in fundus ~1 hour unmixed

ACID (HCl) PRODUCTION IN STOMACH

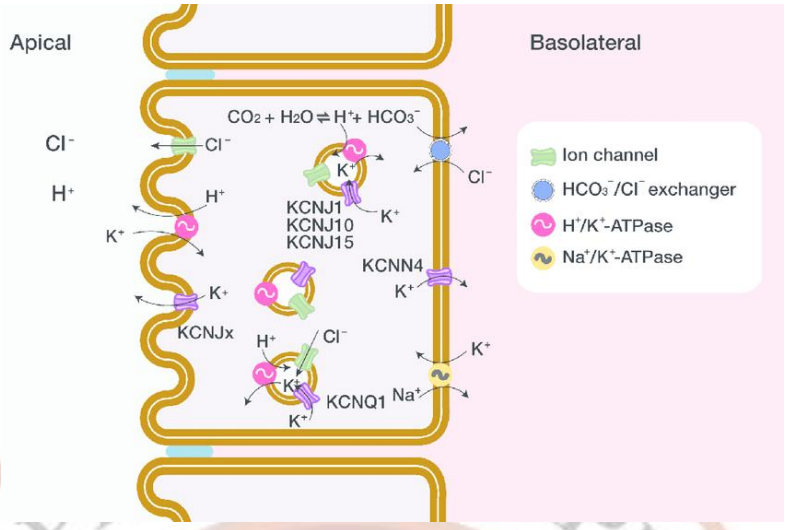
- **Parietal cells** secrete **H^+ and Cl^- ions separately** into the lumen of the stomach.
- Net result is formation of **hydrochloric acid (HCl)**.
- **Carbonic anhydrase (CA)** enzyme present in parietal cells catalyzes:
  - $CO_2 + H_2O \rightarrow H_2CO_3$
- Carbonic acid (H_2CO_3) dissociates into:
  - H^+
  - HCO_3^-
- H^+ serves as a ready source for proton pumps.
- HCO_3^- accumulates in cytosol and exits the cell in exchange for Cl^- via **Cl^-–HCO_3^- antiporter** in the basolateral membrane.
- HCO_3^- diffuses into nearby capillaries causing **temporary alkaline pH of blood** (alkaline tide).
- **Proton pumps (H^+–K^+ ATPase)** actively transport H^+ into the gastric lumen while K^+ enters the cell.
- At the same time:
  - Cl^- diffuses into lumen through Cl^- channels
  - K^+ diffuses through K^+ channels
- In the lumen, H^+ and Cl^- combine to form **HCl**.

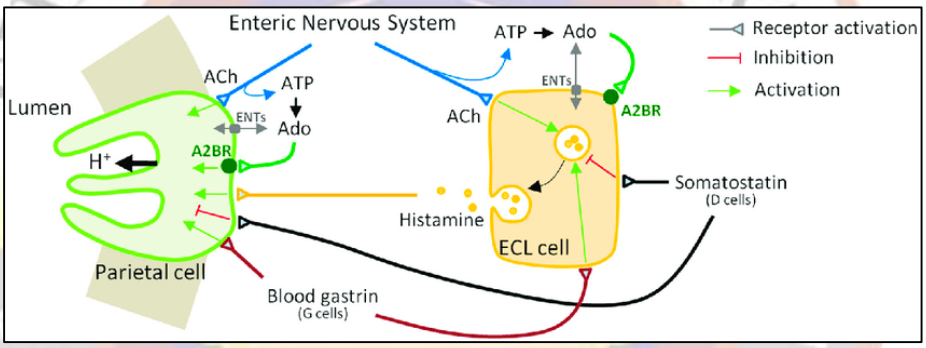
REGULATION OF HCl SECRETION

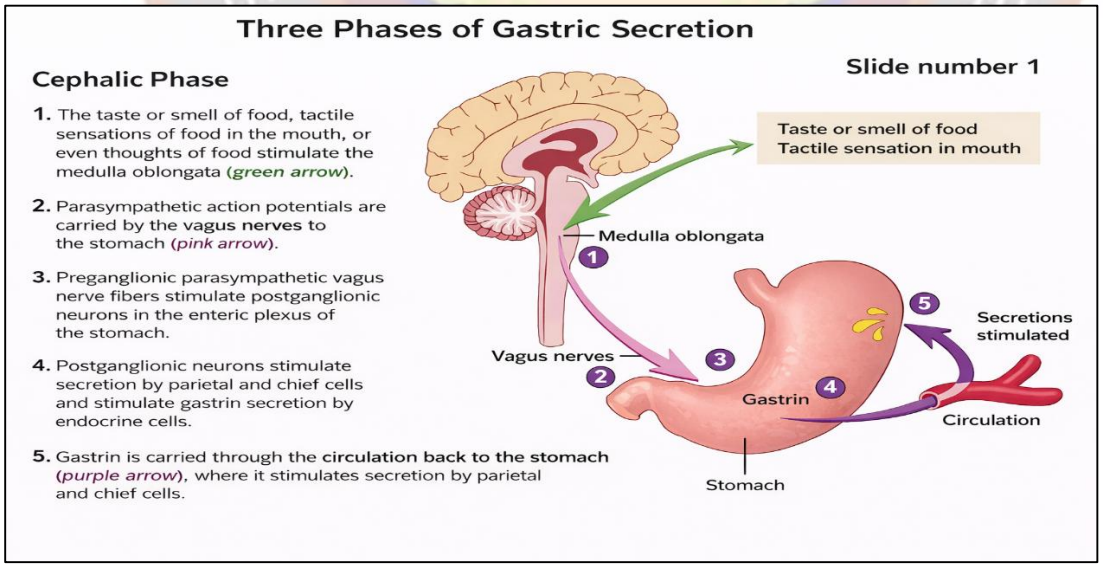
HCl secretion by parietal cells is stimulated by:

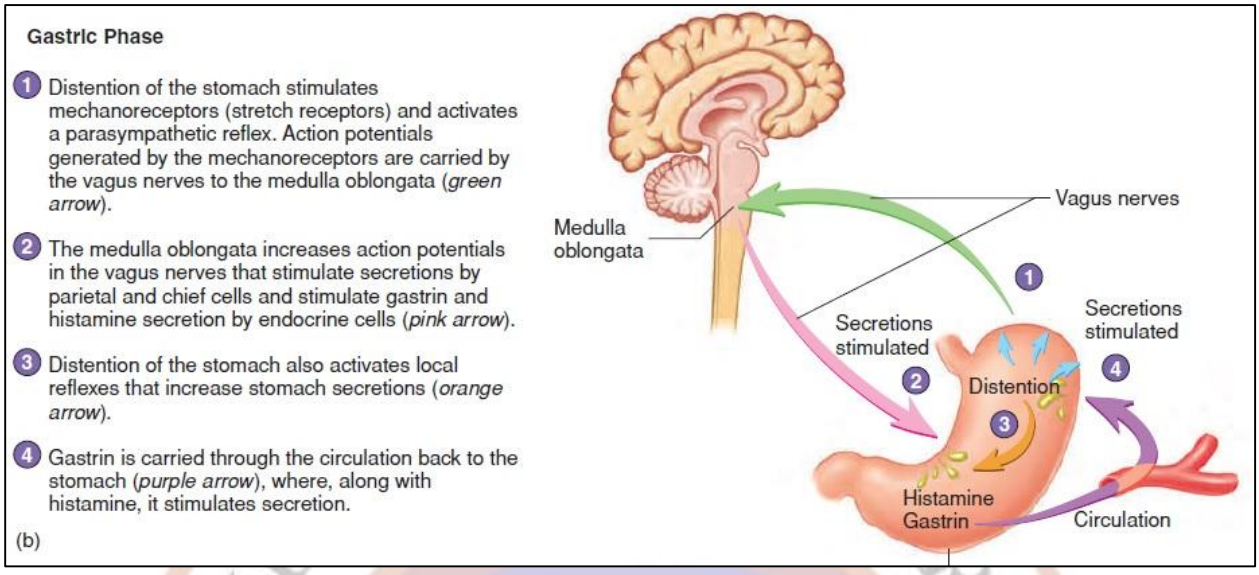
1. **Acetylcholine**
   - Released by parasympathetic neurons
2. **Gastrin**
   - Secreted by G-cells
3. **Histamine**
   - Released by mast cells

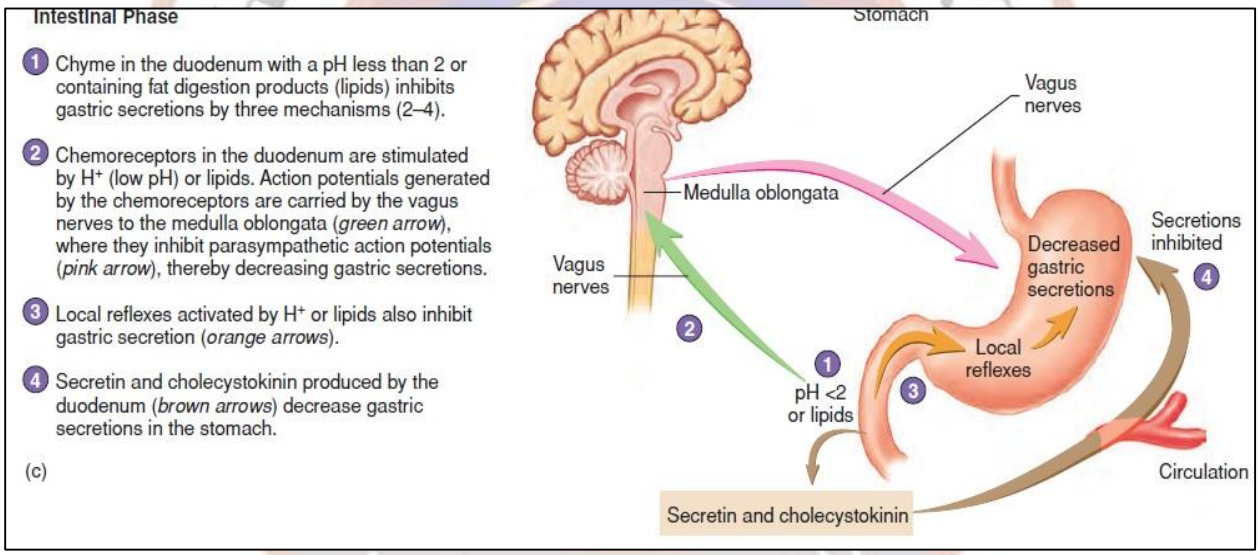
- **Acetylcholine and gastrin together** stimulate parietal cells to secrete more HCl **in the presence of histamine**.


Apical
Basolateral
Cl⁻
H⁺
CO₂ + H₂O ⇌ H⁺ + HCO₃⁻
KCNJ1
KCNJ10
KCNJ15
KCNN4
KCNJx
KCNQ1
Ion channel
HCO₃⁻/Cl⁻ exchanger
H⁺/K⁺-ATPase
Na⁺/K⁺-ATPase


Enteric Nervous System
Lumen
ACh
ATP
Ado
ENTs
A2BR
H⁺
Parietal cell
Histamine
ECL cell
Somatostatin
(D cells)
Blood gastrin
(G cells)
Receptor activation
Inhibition
Activation

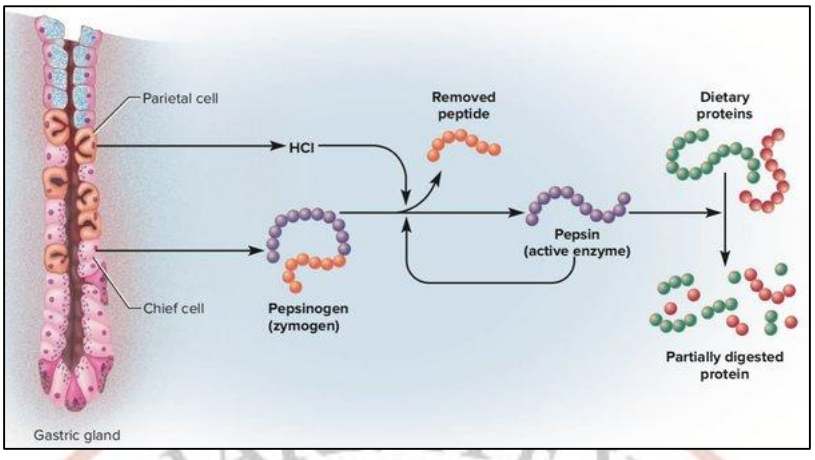

Three Phases of Gastric Secretion
Slide number 1
Cephalic Phase
1. The taste or smell of food, tactile sensations of food in the mouth, or even thoughts of food stimulate the medulla oblongata (green arrow).
2. Parasympathetic action potentials are carried by the vagus nerves to the stomach (pink arrow).
3. Preganglionic parasympathetic vagus nerve fibers stimulate postganglionic neurons in the enteric plexus of the stomach.
4. Postganglionic neurons stimulate secretion by parietal and chief cells and stimulate gastrin secretion by endocrine cells.
5. Gastrin is carried through the circulation back to the stomach (purple arrow), where it stimulates secretion by parietal and chief cells.
Taste or smell of food
Tactile sensation in mouth
Medulla oblongata
Vagus nerves
Gastrin
Secretions stimulated
Circulation
Stomach





ROLE OF PEPSIN IN PROTEIN DIGESTION

- Enzymatic digestion of proteins **begins in the stomach**.
- HCl partially **denatures proteins**, exposing peptide bonds.
- **Pepsin** is the only proteolytic enzyme in the stomach.
- Secreted by **chief cells**.
- Acts on proteins and breaks peptide bonds forming **smaller peptide fragments**.
- Optimal activity at **very acidic pH (~2)**.
- Becomes inactive at higher pH.



Protection of Stomach Cells

1. Pepsin is secreted as **inactive pepsinogen**.
    - Converted to pepsin only in presence of HCl.
2. Gastric epithelium protected by **1–3 mm thick alkaline mucus layer** secreted by:
    - Surface mucus cells
    - Mucous neck cells

ABSORPTION IN STOMACH

- Only **small amounts of nutrients** absorbed in stomach.
- After **2–4 hours** following a meal:
    - Stomach empties contents into duodenum.
- Chyme passes into small intestine.
- Chemical digestion in small intestine depends on:
    - Pancreas
    - Liver
    - Gall bladder

PANCREAS

- Creamy pink gland
- Length: **12–15 cm**
- Thickness: **2.5 cm**
- Lies posterior to greater curvature of stomach

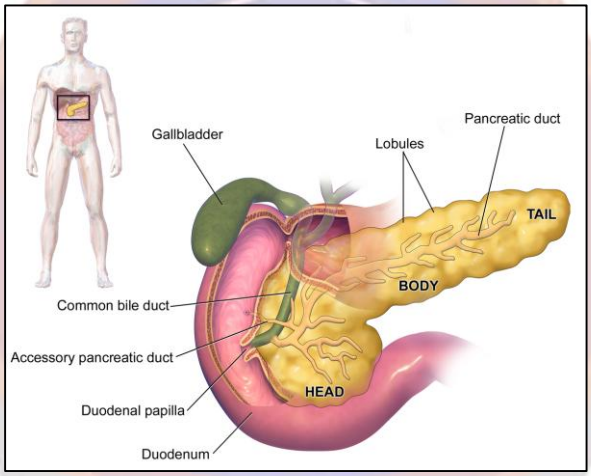
Parts

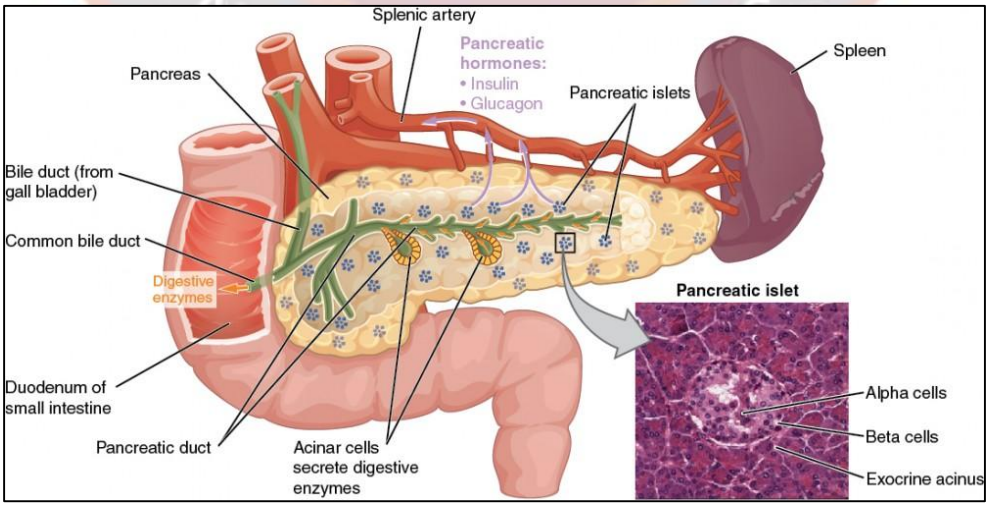
1. Head
    - Broad expanded portion

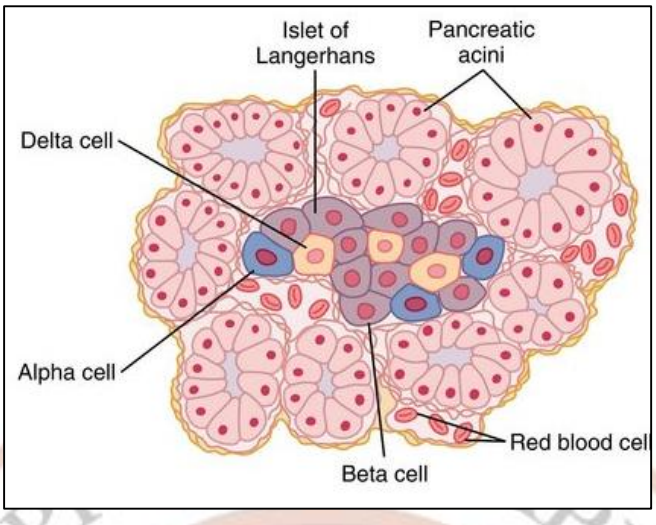
   - Lies in curve of duodenum
2. Body
   - Lies superior and left to head
3. Tail
   - Narrow and tapering

Ducts

- Connected to duodenum by:
  1. Hepatopancreatic ampulla
  2. Accessory duct
- Pancreas is both **exocrine and endocrine**.







EXOCRINE PANCREAS (99%)

- Consists of clusters of glandular epithelial cells called **acini**.
- Acini form lobules.
- Acinar cells secrete **pancreatic juice** (fluid + enzymes).
- Secretions pass into:
    - Small ducts → larger ducts

Pancreatic Duct System

1. **Pancreatic duct (duct of Wirsung)**
    - Joins common bile duct
    - Forms hepatopancreatic ampulla
2. **Accessory duct**
    - Opens into duodenum about 2.5 cm above ampulla

- Opening of ampulla controlled by **hepatopancreatic sphincter (sphincter of Oddi)**.

FUNCTIONS OF PANCREAS – PANCREATIC JUICE

- Produces **1.2–1.5 L/day** of pancreatic juice.
- Composition:
    - Water
    - Salts
    - Sodium bicarbonate ($NaHCO_3$)
    - Digestive enzymes

Role of $NaHCO_3$

- Slightly alkaline pH: **7.1–8.2**
- Neutralizes acidic chyme
- Provides optimal pH for enzyme action

Pancreatic Enzymes

1. **Pancreatic amylase** – digests starch
2. **Proteolytic enzymes**
    - Trypsin
    - Chymotrypsin
    - Carboxypeptidase
    - Elastase
3. **Pancreatic lipase**
    - Main triglyceride digesting enzyme in adults
4. **Nucleases**
    - Ribonuclease
    - Deoxyribonuclease

ACTIVATION OF PANCREATIC ENZYMES

- Protein-digesting enzymes are secreted in **inactive forms**:

1. Trypsinogen → Trypsin
    - Activated by **enterokinase** (brush border enzyme)
2. Chymotrypsinogen
3. Procarboxypeptidase
4. Proelastase

ENDOCRINE PANCREAS (1%)

- Groups of specialized cells called **pancreatic islets (Islets of Langerhans)**.
- Secrete hormones:
    - Insulin
    - Glucagon

LIVER AND GALL BLADDER

Liver

- Largest and heaviest gland in the body
- Weight: **1–2.3 kg**
- Located inferior to diaphragm

- Occupies:
  - Right hypochondriac region
  - Part of epigastric region

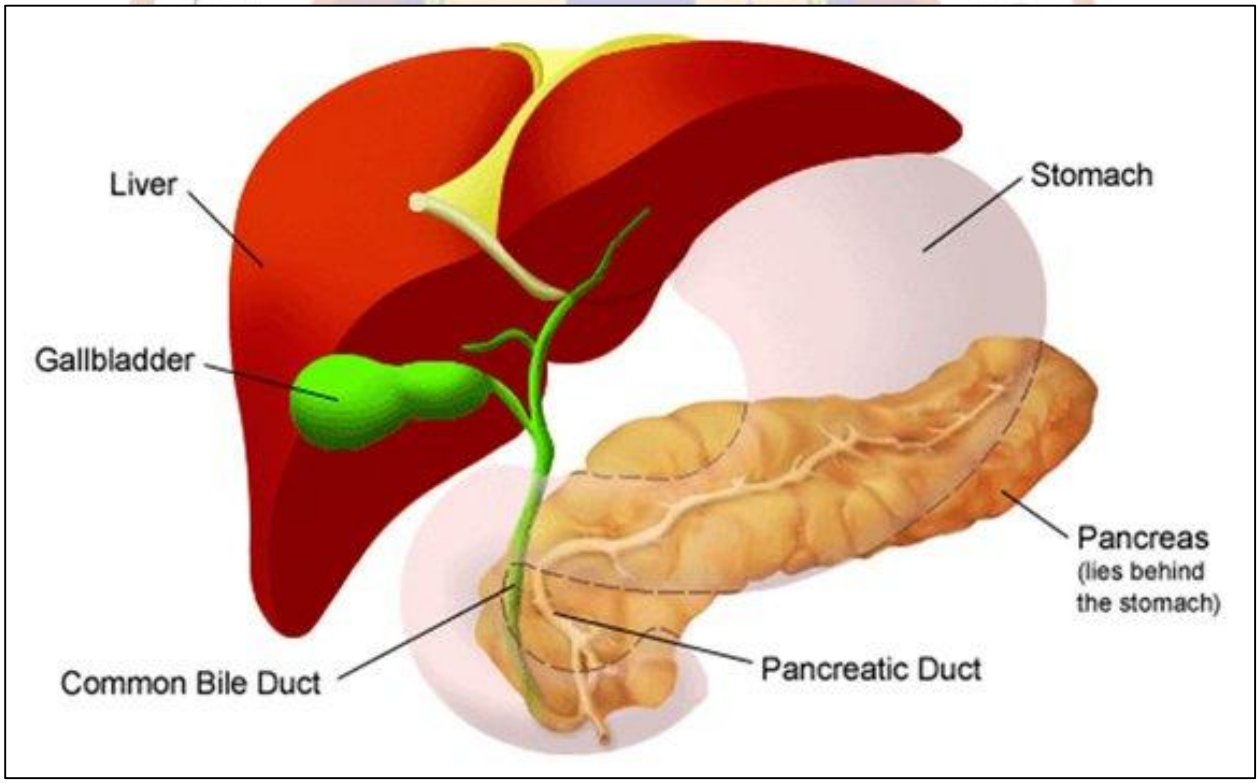
Gall Bladder

- Pear-shaped sac
- Located in depression on posterior surface of liver

Relations of Liver

- Superior & anterior: diaphragm, anterior abdominal wall
- Inferior: stomach, duodenum, bile ducts, right kidney, adrenal gland
- Posterior: esophagus, inferior vena cava, aorta, gall bladder, vertebral column
- Lateral: ribs and diaphragm

STRUCTURE OF LIVER & GALL BLADDER



Liver

- Almost completely covered by visceral peritoneum
- Divided into:

  - Right lobe (large)
  - Left lobe (small)
  - Caudate lobe
  - Quadrate lobe
- Right and left lobes separated by **falciform ligament**

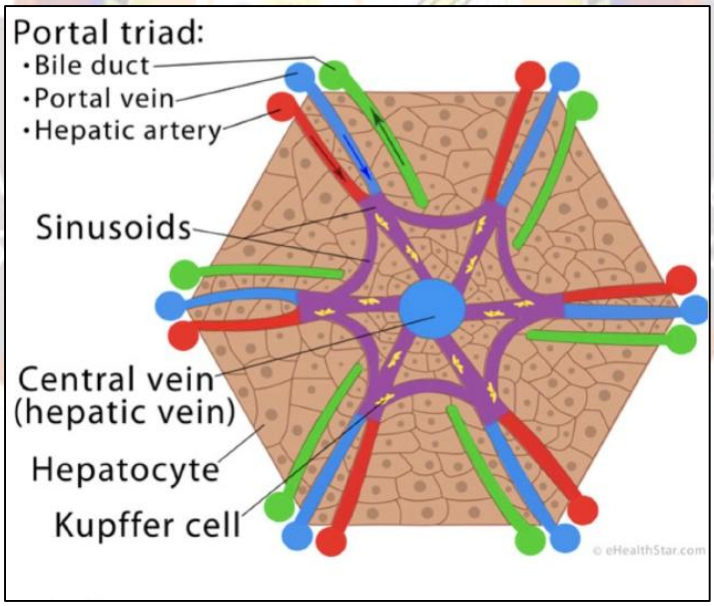
Gall Bladder

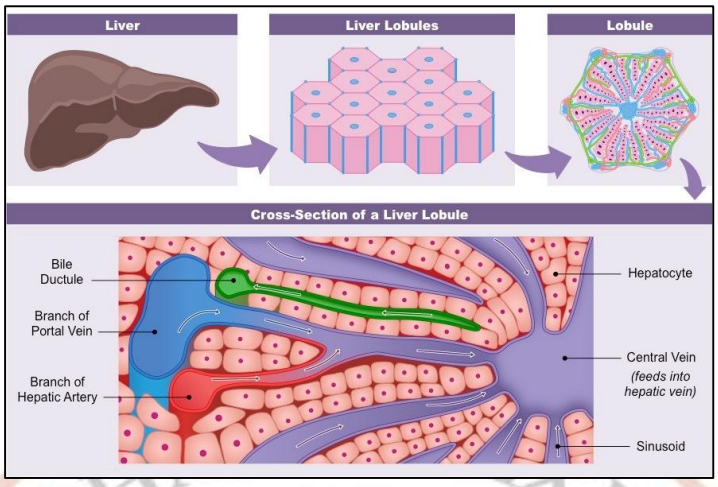
- Regions:
  1. Fundus
  2. Body
  3. Neck (continues as cystic duct)

Portal Fissure

- Region where structures enter and leave liver
- Portal vein and hepatic artery enter
- Hepatic ducts, lymph vessels, nerves leave

HISTOLOGY OF LIVER





- Liver lobes composed of **hepatic lobules**.
- Lobules:
  - Hexagonal
  - Made of hepatocytes arranged in pairs radiating from central vein

Sinusoids

- Located between hepatocyte columns
- Carry mixed blood:
  - Oxygenated blood from hepatic artery
  - Nutrient-rich blood from portal vein

Kupffer Cells

- Macrophages lining sinusoids
- Phagocytize:
  - Worn-out RBCs
  - Foreign particles

Stellate Cells

- Quiescent fibroblasts
- Activated during inflammation
- Synthesize collagen

Blood Flow

- Sinusoids → central veins → hepatic veins → inferior vena cava

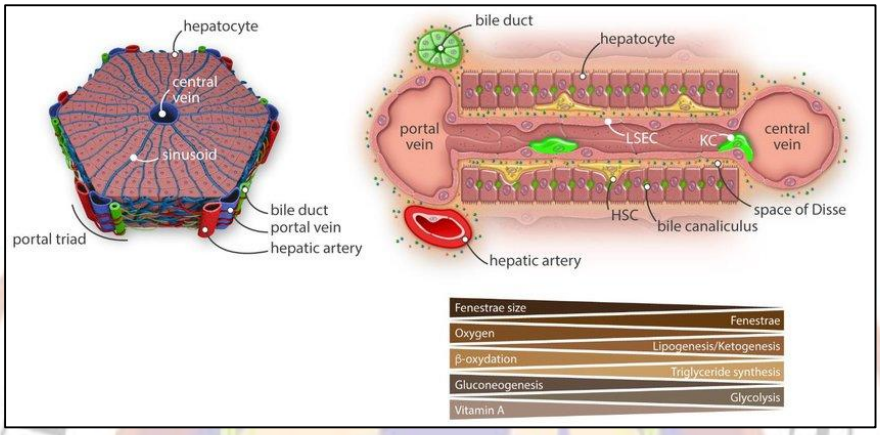
Bile Canaliculi

- Tiny bile channels between hepatocytes

- Drain bile into right and left hepatic ducts

Portal Triad

- Bile duct
- Branch of hepatic artery
- Branch of portal vein

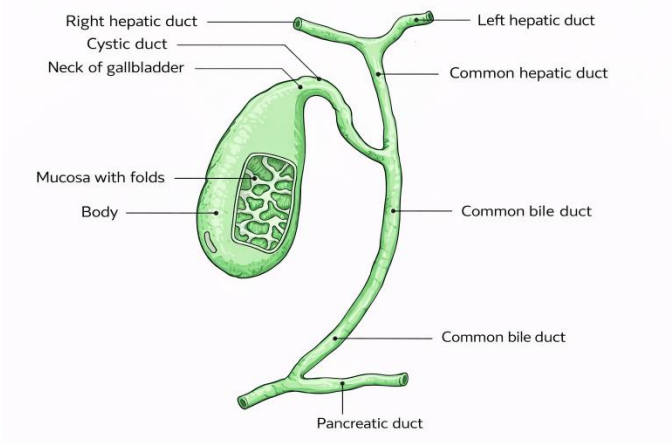


GALL BLADDER STRUCTURE & BILE DUCTS

- Same tissue layers as GIT
- Covered by peritoneum on inferior surface only
- Has additional oblique muscle layer

Bile Duct Pathway

- Right and left hepatic ducts → common hepatic duct
- Joins cystic duct → common bile duct
- Joins pancreatic duct → hepatopancreatic ampulla
- Opens into duodenum at duodenal papilla
- Controlled by sphincter of Oddi



FUNCTIONS OF LIVER

Carbohydrate Metabolism

- Maintains blood glucose level
- Low glucose:
  - Glycogen → glucose (glucagon)
- High glucose:
  - Glucose → glycogen and triglycerides (insulin)

Lipid Metabolism

- Stores triglycerides
- Breaks down fatty acids
- Synthesizes cholesterol and bile salts
- Forms lipoproteins

Protein Metabolism

- Deamination → urea formation
- Transamination → non-essential amino acids
- Synthesizes plasma proteins (albumin, globulin, clotting factors)

Detoxification

- Alcohol, drugs, toxins
- Drugs excreted in bile: penicillin, erythromycin

Hormone Inactivation

- Thyroid hormones

- Steroid hormones
- Insulin and glucagon

Bilirubin Excretion

- From breakdown of hemoglobin
- Secreted into bile

Bile Salt Synthesis

- Required for fat digestion

Phagocytosis

- Kupffer cells remove aged RBCs and bacteria

Vitamin D Activation

- Liver participates with skin and kidney

Heat Production

- Major heat-producing organ

Storage

- Glycogen
- Vitamins A, D, E, K
- Vitamin B_{12}
- Iron and copper

SECRETION OF BILE

- Produced by hepatocytes: **500–1000 ml/day**
- Composition:
  - Water
  - Mineral salts
  - Mucus
  - Bile pigments (bilirubin)
  - Bile salts
  - Cholesterol
- pH: **7.6–8.8**
- Bile is both:
  - Excretory

- Digestive secretion

FUNCTIONS OF BILE

Fat Digestion

- Bile salts emulsify fats
- Increase surface area for lipase action
- Aid absorption of fats and fat-soluble vitamins

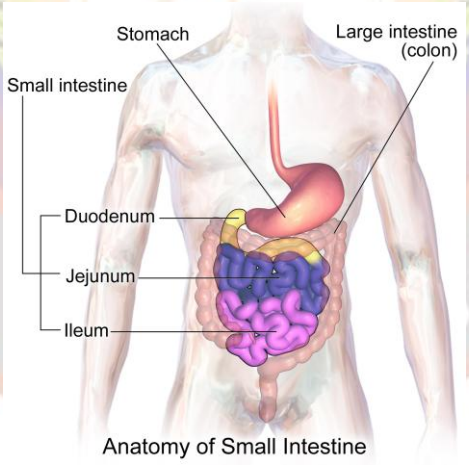
Bilirubin Excretion

- Converted by bacteria to stercobilin
- Gives brown color to feces

FUNCTIONS

1. Storage of bile
2. Concentration of bile (10–15 times)
3. Release of bile into duodenum

STRUCTURE OF SMALL INTESTINE



Anatomy of Small Intestine

Small intestine is divided into **three regions**:

1. Duodenum

- Length: **25 cm**
- C-shaped, curves around pancreas
- Continuous with jejunum

2. Jejunum

- Middle portion
- Length: **1–2 m**

3. Ileum

- Final and longest region
- Length: **2–3 m**
- Joins large intestine at:
    - **Ileocaecal sphincter / valve**
    - Controls flow of contents from ileum to caecum

WALL OF SMALL INTESTINE

Wall consists of **four layers**:

1. Mucosa

- Composed of:
    - Epithelium
    - Lamina propria
    - Muscularis mucosa

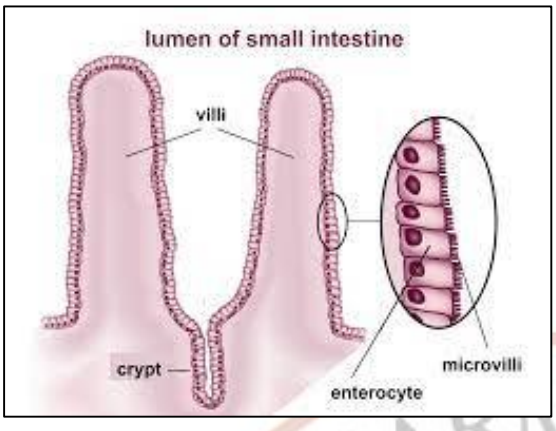
Surface Area Modifications

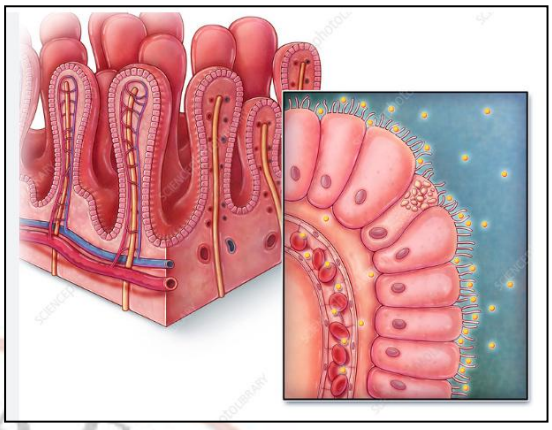
- Permanent circular folds – promote mixing of chyme
- **Villi and microvilli (brush border)** – increase surface area

Epithelium

- Simple columnar epithelium
- Major cell types:
    1. **Absorptive cells (Enterocytes)**
        - Possess microvilli
        - Contain digestive enzymes
        - Absorb nutrients
    2. **Goblet cells**
        - Secrete mucus

Villi



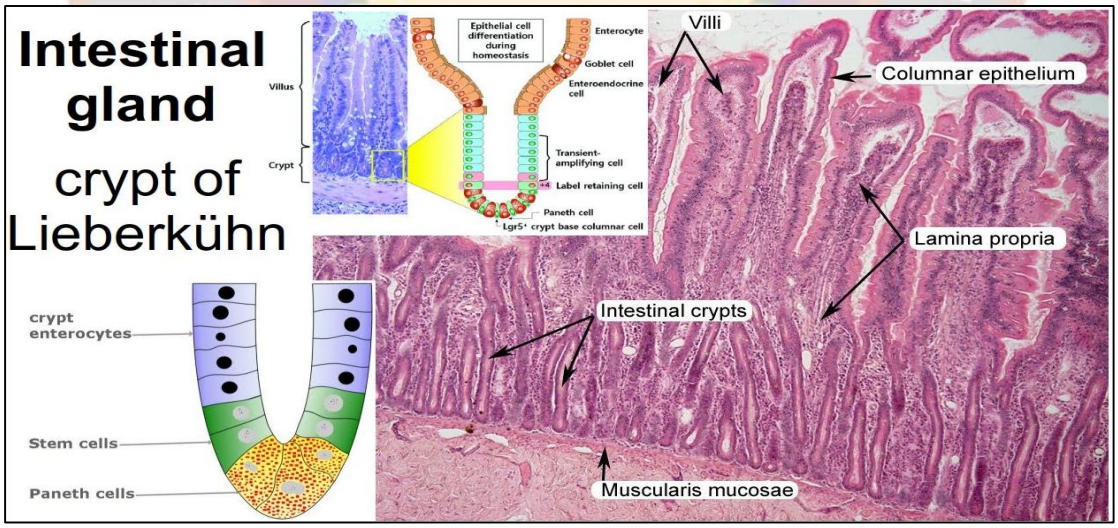


- Finger-like projections (0.5–1 mm long)
- Contain:
  - Network of blood capillaries
  - Central lymph capillary called **lacteal**

Microvilli

- Present on enterocytes
- Length: **~1 µm**
- Form brush border

INTESTINAL GLANDS



- Simple tubular glands located between villi
- Secrete **intestinal juice**

Special Cells

- **Paneth cells**
  - Secrete lysozyme
- **Enteroendocrine cells**
  - S-cells – secrete **secretin**
  - CCK cells – secrete **cholecystokinin**
  - K-cells – secrete **GIP (Glucose-dependent insulinotropic peptide)**

Other Features

- Entire epithelium replaced every **3–5 days**
- Numerous lymph nodes along length of small intestine
- Aggregated lymph follicles (20–30) in distal ileum called:
  - **Peyer's patches**

SUBMUCOSA

- In duodenum contains **Brunner's glands**
- Secrete **alkaline mucus**

MUSCULARIS

- Two smooth muscle layers:
  - Outer longitudinal (thin)
  - Inner circular (thick)

SEROSA

- Completely surrounds small intestine except major part of duodenum
- Forms **mesentery**
  - Double layer of peritoneum
  - Suspends intestine and carries blood vessels, lymphatics, nerves

INTESTINAL JUICE

- Volume: **1–2 L/day**
- Clear yellow fluid
- Composition:
  - Water
  - Mucus
  - Bicarbonate (HCO_3^-)
- pH: **~7.6 (slightly alkaline)**

Enzymes

- Enzymes are **not secreted into juice**
- Present in enterocytes:
  - α-dextrinase
  - Maltase
  - Sucrase
  - Lactase
  - Peptidases
  - Nucleosidase
  - Phosphatase

FUNCTIONS OF SMALL INTESTINE

1. Onward movement of contents by **peristalsis**
   - Increased by parasympathetic stimulation
2. Secretion of intestinal juice
3. Completion of chemical digestion of:
   - Carbohydrates
   - Proteins
   - Fats
4. Protection against infection
   - Solitary and aggregated lymph follicles
5. Secretion of hormones:
   - CCK
   - Secretin
6. Absorption of nutrients

MECHANICAL DIGESTION IN SMALL INTESTINE

- **Segmentation**
  - Localized mixing contractions
- **Migrating motility complexes**
  - Type of peristalsis

CHEMICAL DIGESTION IN SMALL INTESTINE

- Acidic chyme mixes with:
  - Pancreatic juice
  - Bile
  - Intestinal juice
- Digestion completed at brush border

End Products

- Carbohydrates → Monosaccharides
- Proteins → Amino acids
- Fats → Fatty acids + glycerol

DIGESTION SUMMARY

Mouth

- Salivary amylase:
  - Starch → Maltose, maltotriose, α-dextrins

Stomach

- Pepsin:
  - Proteins → Peptides
- Lingual and gastric lipase:
  - Triglycerides → fatty acids + mono/diglycerides

DIGESTION OF CARBOHYDRATES

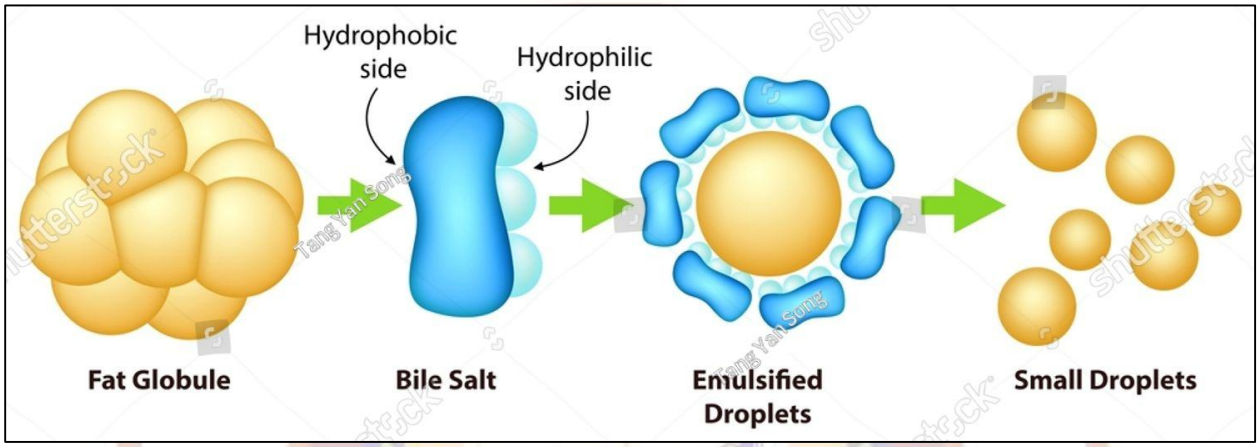
- Pancreatic amylase:
  - Starch, glycogen → maltose, maltotriose, α-dextrins
- α-dextrinase:
  - α-dextrin → glucose
- Sucrase:
  - Sucrose → glucose + fructose
- Maltase:
  - Maltose/maltotriose → glucose
- Lactase:
  - Lactose → glucose + galactose
- End product: **monosaccharides**

DIGESTION OF PROTEINS

- Pepsin:
  - Proteins → peptides
- Pancreatic proteases:
  - Trypsin
  - Chymotrypsin
  - Carboxypeptidase

  - Elastase
- Peptidases:
  - Aminopeptidase
  - Dipeptidase
- Protein digestion completed in small intestine

DIGESTION OF LIPIDS



- Dietary lipids mainly triglycerides
- Lipases involved:
  - Lingual
  - Gastric
  - Pancreatic (major)

Pancreatic Lipase

- Triglycerides → fatty acids + monoglycerides

Emulsification

- Large lipid globules broken into small globules
- Carried out by **bile salts**

Bile Salts

- Amphipathic
- Hydrophobic end binds fat
- Hydrophilic end binds water
- Increase surface area for lipase action

DIGESTION OF NUCLEIC ACIDS

- Pancreatic nucleases:
  - Ribonuclease
  - Deoxyribonuclease
- Break RNA and DNA into nucleotides
- Further digested by:
  - Nucleosidases
  - Phosphatases
- End products:
  - Pentose sugars
  - Phosphate
  - Nitrogenous bases

ABSORPTION IN SMALL INTESTINE

- Absorption = passage of digested nutrients into blood or lymph
- Occurs mainly through enterocytes

Mechanisms

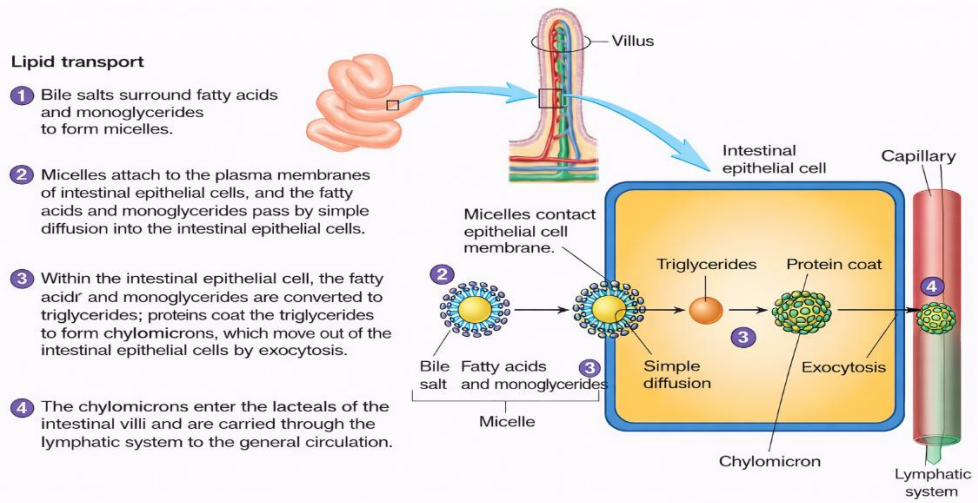
- Diffusion
- Facilitated diffusion
- Osmosis
- Active transport

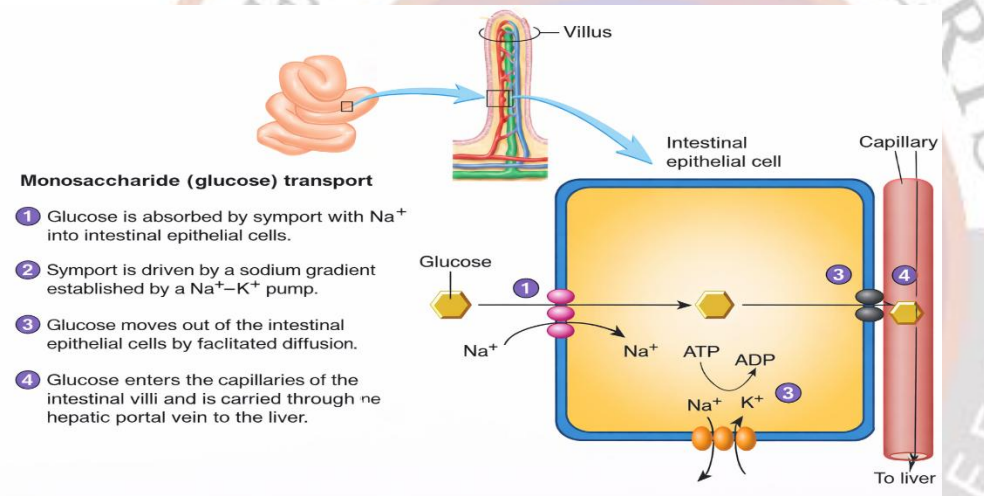
Extent

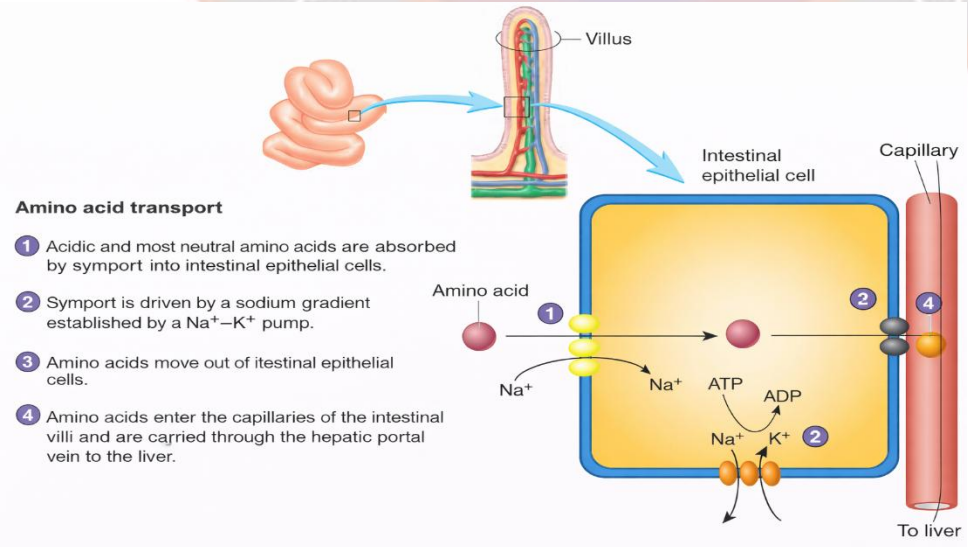
- 90% absorption – small intestine
- 10% – stomach and large intestine
- Undigested material (~1500 ml) enters large intestine

ABSORPTION OF MONOSACCHARIDES

- Fructose:
  - Facilitated diffusion
- Glucose and galactose:
  - Secondary active transport with Na^+
  - Cotransport (1 glucose + 2 Na^+)
- Exit via facilitated diffusion into blood capillaries


Lipid transport
1 Bile salts surround fatty acids and monoglycerides to form micelles.
2 Micelles attach to the plasma membranes of intestinal epithelial cells, and the fatty acids and monoglycerides pass by simple diffusion into the intestinal epithelial cells.
3 Within the intestinal epithelial cell, the fatty acidr and monoglycerides are converted to triglycerides; proteins coat the triglycerides to form chylomicrons, which move out of the intestinal epithelial cells by exocytosis.
4 The chylomicrons enter the lacteals of the intestinal villi and are carried through the lymphatic system to the general circulation.
Villus
Intestinal epithelial cell
Capillary
Micelles contact epithelial cell membrane.
Triglycerides
Protein coat
Bile salt
Fatty acids and monoglycerides
Micelle
Simple diffusion
Exocytosis
Chylomicron
Lymphatic system


Monosaccharide (glucose) transport
1 Glucose is absorbed by symport with Na+ into intestinal epithelial cells.
2 Symport is driven by a sodium gradient established by a Na+–K+ pump.
3 Glucose moves out of the intestinal epithelial cells by faclitated diffusion.
4 Glucose enters the capillaries of the intestinal villi and is carried through ne hepatic portal vein to the liver.
Villus
Intestinal epithelial cell
Capillary
Glucose
Na+
Na+
ATP
ADP
Na+
K+
To liver

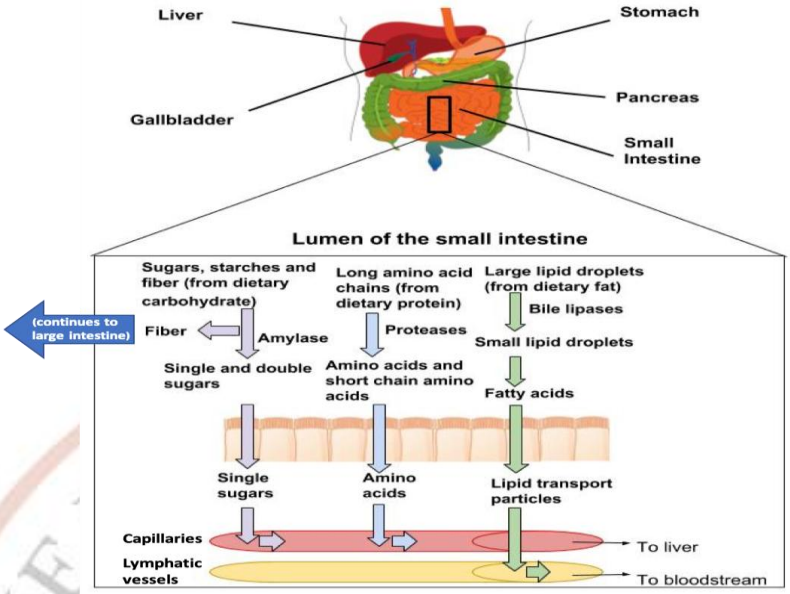

Amino acid transport
1 Acidic and most neutral amino acids are absorbed by symport into intestinal epithelial cells.
2 Symport is driven by a sodium gradient established by a Na+–K+ pump.
3 Amino acids move out of itestinal epithelial cells.
4 Amino acids enter the capillaries of the intestinal villi and are carried through the hepatic portal vein to the liver.
Villus
Intestinal epithelial cell
Capillary
Amino acid
Na+
Na+
ATP
ADP
Na+
K+
To liver

ABSORPTION OF AMINO ACIDS

- Absorbed mainly in duodenum and jejunum
- Mechanisms:
  - Active transport
  - Cotransport with Na^+
- Exit cells by diffusion
- Some proteins absorbed unchanged:
  - Antibodies in breast milk
  - Oral vaccines (polio)

ABSORPTION OF LIPIDS & BILE SALTS

- Fatty acids and glycerol:
  - Diffuse into lacteals
  - Enter lymph → thoracic duct → circulation
- Transport forms:
  - Micelles
  - Chylomicrons



ABSORPTION OF ELECTROLYTES

- Na^+:
  - Active transport (Na^+–K^+ ATPase)
- Cl^-, HCO_3^-, I^-, NO_3^{2-}:
  - Passive or active transport
- Ca^{2+}:
  - Active transport
- Fe, K^+, Mg^{2+}, phosphate:
  - Active transport

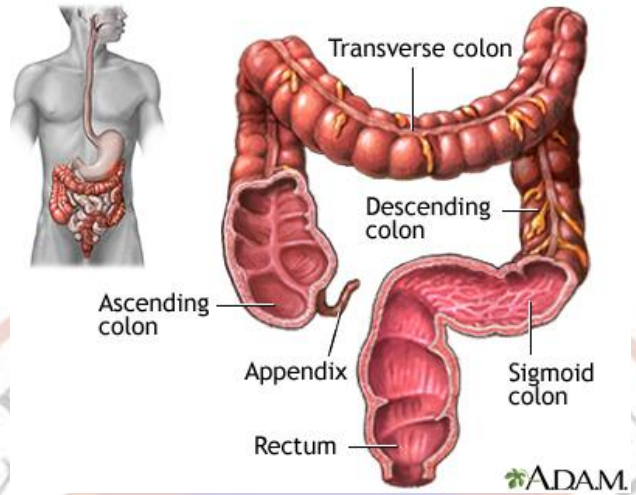
ABSORPTION OF VITAMINS

- Fat-soluble vitamins:
  - Absorbed with fats into lacteals
- Vitamin B_{12}:
  - Absorbed with intrinsic factor
  - Site: terminal ileum

ABSORPTION OF WATER

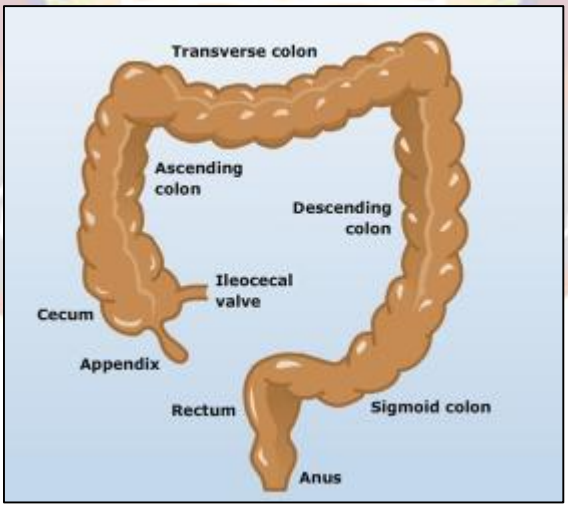
- Occurs by osmosis
- Depends on absorption of solutes
- Total fluid entering intestine: **9.3 L**
- Absorbed in small intestine: **8.3 L**
- Remaining enters large intestine

VI. LARGE INTESTINE



- Terminal part of GIT
- Length: **1.5 m**, Diameter: **6.5 cm Forms arch around coiled up small intestine**
- Extends from ileum to anus
- Surrounds small intestine
- Attached by **mesocolon**

Four Regions



1. Caecum
2. Colon
3. Rectum
4. Anal canal

Caecum

- Ileocaecal valve guards entry
- Blind pouch ~6 cm long
- Vermiform appendix:
    - 8 cm long
    - Attached to caecum

Colon

- Ascending colon
- Transverse colon
- Descending colon
- Sigmoid colon

ANAL CANAL

- Short terminal passage of large intestine
- Length: **3.8 cm**
- Extends from rectum to exterior
- External opening is **anus**

Sphincters

- **Internal anal sphincter**
    - Smooth muscle
    - Involuntary
- **External anal sphincter**
    - Skeletal muscle
    - Voluntary
- Anal sphincter normally remains closed

STRUCTURE OF LARGE INTESTINE

Large intestine consists of **four layers** in all regions:

1. MUCOSA

- Epithelium:
    - Simple columnar epithelium
- Colon and upper rectum:
    - Numerous **goblet cells**
    - Secrete mucus
    - Lubricates passage of solid feces
- Anal canal:

    - Stratified squamous epithelium
- Upper part of anal canal:
    - Mucous membrane forms **6–10 vertical folds**
    - Called **anal columns**

2. SUBMUCOSA

- Contains **more lymphatic tissue** than other parts of GIT
- Provides protection against:
    - Resident bacteria
    - Invading bacteria

3. MUSCULARIS

- Inner layer: Circular muscle fibers
- Outer layer: Longitudinal muscle fibers

Taeniae Coli

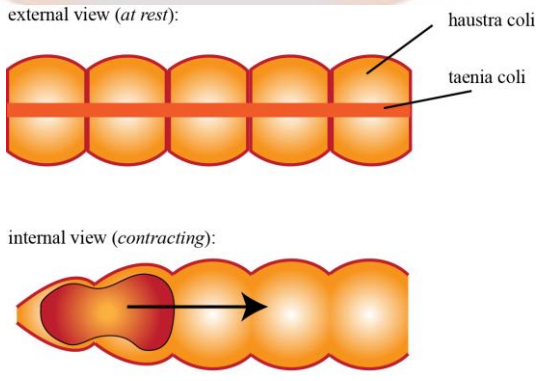
- Longitudinal fibers do not form a continuous layer
- Collected into **three distinct bands**
- Called **taenia coli**
- Run along caecum and colon

Haustra

- Tonic contraction of taenia coli
- Colon gathered into pouch-like sacs
- Called **haustra**
- Gives colon puckered appearance

4. SEROSA

- Forms part of **visceral peritoneum**



MECHANICAL DIGESTION IN LARGE INTESTINE

- Large intestine **does not exhibit peristalsis**
- Movement occurs by:
    - **Mass movements**

CHEMICAL DIGESTION IN LARGE INTESTINE

- Occurs due to **bacterial activity**
- No digestive enzymes secreted by intestine
- Mucus is secreted

Bacterial Actions

- Ferment remaining carbohydrates
- Release gases:
    - Hydrogen (H_2)
    - Carbon dioxide (CO_2)
    - Methane (CH_4)
- Excess gas leads to **flatulence**
- Convert remaining proteins into:
    - Amino acids
    - Indole
    - Hydrogen sulphide
    - Fatty acids
- Responsible for odor of feces
- Decompose bilirubin into simpler pigments

FUNCTIONS OF LARGE INTESTINE

1. Absorption

- Contents are still fluid
- Water absorbed by **osmosis**
- Leads to semisolid feces
- Absorption of:
    - Mineral salts
    - Vitamins
    - Some drugs

2. Microbial Activity

- Heavily colonized by bacteria
- Bacteria synthesize:
  - Vitamin K
  - Fatty acids

Examples of Bacteria

- *Escherichia coli*
- *Enterobacter aerogenes*
- *Streptococcus faecalis*
- *Clostridium perfringens*
- These bacteria are **commensals**
- Can become pathogenic in other parts
  - Example: *E. coli* → cystitis

MASS MOVEMENT

- Haustral churning and mass peristalsis move contents to rectum
- No true peristalsis
- Occurs **4–6 times per day** in adults
- Strong waves sweep contents from transverse colon to sigmoid colon
- Controlled by **gastrocolic reflex**

FAECES FORMATION AND DEFAECATION

- Mass movements push feces into rectum
- Stretch receptors in rectal wall stimulated

Defaecation Reflex

- Normally under voluntary control
- Brain can inhibit reflex until convenient

Process Involves

- Involuntary contraction of rectal muscles
- Relaxation of internal anal sphincter
- Voluntary relaxation of external anal sphincter
- Contraction of abdominal muscles
- Lowering of diaphragm during forced expiration
- Increased intra-abdominal pressure
- Promotes expulsion of feces

Constipation

- Occurs when defaecation is postponed repeatedly
- Excess water absorbed
- Feces become hard

COMPOSITION OF FAECES

- Semisolid brown mass (color due to **stercobilin**)

Constituents

- Water – **60–70%**
- Dietary fiber (indigestible material)
- Dead and live microorganisms
- Shed epithelial cells
- Fatty acids
- Mucus – aids lubrication

ROLE OF DIETARY FIBER

- Also called **roughage**
- Non-starch polysaccharides
- Ensures bulky feces
- Stimulates defaecation
- Prevents constipation

Diseases of the Mouth

Inflammatory and infectious conditions

Thrush (oral candidiasis)

Gingivitis

Recurrent aphthous ulceration

Viral infections

- Acute herpetic gingivostomatitis

- Seconday or recurrent herpes lesions (cold sores)

Tumours of the mouth

Squamous cell carcinoma

Congenital disorders

Cleft palate and cleft lip (harelip)

Dental carInflammatory and infectious conditions

Food & other ingested substances – corrosive, abrasive, or excessively hot or cold – injury to tissues in and around the mouth

Large number and variety of harmless commensals in the mouth

Saliva – antimicrobial action limit the growth

Presence of dental plaque & food residues (sugars) – promote infection

Stomatitis – inflammation of mouth

Gingivitis – inflammation of the gumsies

Thrush (oral candidiasis)

- Fungal infection caused by yeast *Candida albicans*
- Commensal microbe grows in white patches on the tongue and oral mucosa

Gingivitis

- Also called gum inflammation
- It is a form of gum disease that causes inflamed gums
- Cause – poor oral hygiene
- Untreated – loss of tooth and other serious conditions
- Symptoms – gums that are swollen, puffy, receding, sometimes tender and bleed easily

Recurrent aphthous ulceration

- Extremely painful ulcers – occur singly or in crops in any part of the mouth
- Cause – unknown

Viral Infections

- Usually caused by one type of Herpes Simplex Virus (HSV-1)

Acute herpetic gingivostomatitis

- Most common oral viral infection
- Inflammation of mouth & gums
- Extensive & painful ulceration

Secondary or recurrent herpes lesions (cold sores)

- Lesions occur round the nose and on the lips
- Initial outbreak – virus dormant

- Later outbreaks – stimulated by exposure to UV rays & impaired immunity

Tumours of the mouth

Squamous cell carcinoma

- Most common type of malignant tumor in the mouth
- Affects older adults
- Usual sites – floor of mouth, edge of the tongue
- Frequent ulceration, early spread to surrounding tissues, cervical lymph nodes

Congenital disorders

Cleft palate and cleft lip (harelip)

- During embryonic development, roof of mouth (hard palate) develops as separate halves
- Before birth, two halves normally fuse along midline
- Fusion incomplete – a cleft (division) remains, minor or substantial
- Cleft lip - minor notch in upper lip to more extensive condition, upper lip is completely split in one or two places & nose is involved
- Cleft palate – gap between two halves of palate
- Factors – genetic abnormalities, certain drugs or poor nutrition b/w 7-12 weeks of pregnancy

Dental Caries

- Tooth decay starts with discoloration, then formation of cavities or caries
- Occurs when bacteria in plaque on teeth act on sugars – form acids – destroy hard parts of teeth
- Prevented by good oral hygiene
- Without treatment – infection & inflammation of soft tissues of mouth & tooth loss

<u>Diseases of the Pharynx</u>

Tonsilitis

- ➢ Inflammation of the palatine tonsils, palatine arches and walls of the pharynx
- ➢ Causes
- Viruses
- *Streptococcus pyogenes*
- ➢ Severe infection – suppuration and abscess formation

- After acute tonsilitis – swelling subsides and return to normal
- Repeated infections – chronic inflammation, fibrosis and permanent enlargement
- Endotoxins from tonsilitis by bacteria – development of rheumatic fever and glomerulonephritis

Diphtheria

- Bacterial infection of the pharynx – may extend to the nasopharynx and trachea
- Caused by Corynebacterium diphtheriae
- A thick, fibrous membrane forms over the area and may obstruct the airway
- Pathogen produces powerful exotoxins that may severely damage cardiac and skeletal muscle, liver, kidneys and adrenal glands
- In countries where immunization is widespread, it is rare condition

Diseases of salivary glands

Mumps (parotitis)

- Acute inflammatory condition of salivary glands, esp. parotids
- Caused by mumps virus, spread by inhalation of infected droplets
- Most infectious for 1-2 days before and 5 days after symptoms appear
- Most common between 5 and 15 years of age, also affects adults & is more serious infection
- 25% adult males – testicular inflammation (orchitis) – cause atrophy and occasional sterility
- Complications – meningitis and meningoencephalitis
- Vaccination for children in preschool years

Tumors of salivary glands

Salivary adenoma

- Benign tumor occurs mainly in parotid gland
- Most common tumor
- Occasional malignancy

Carcinoma

- Malignant tumor – mostly parotid glands

Diseases of the esophagus

Esophageal varices

- Varices are localized dilations of veins
- Cirrhosis or venous thrombosis – blood flow to liver via portal vein impeded – BP in portal system rises (portal hypertension)
- blood forced into anastomotic veins – blood redirected (shunted) to systemic circulation, bypassing the liver
- Rise in pressure in veins too
- One route – veins of lower esophagus – distended & weakened by abnormal high volume of blood
- Weakest regions of vessel wall bulge into lumen – develop varices
- Thin-walled and fragile – easily eroded or traumatised by swallowed food
- Bleeding – minor to chronic (iron deficiency anaemia)
- Sudden rupture – life-threatening haemorrhage

Inflammatory and infectious conditions

Acute esophagitis

- Occurs when –

1. Caustic materials swallowed
2. Acquiring of severe fungal infections by immunocompromised people – candidiasis or
3. Viral infections – herpes simplex

- Dysphagia – usually present

Gastro-esophageal reflux disease (GERD)

- ✓ Most common cause of indigestion (heartburn)
- ✓ Caused by persistent regurgitation of acidic gastric juice into esophagus – cause irritation, inflammation and painful ulceration
- ✓ Reflux occurs –

1. Increase in intra-abdominal pressure – pregnancy, constipation, obesity
2. Low levels of gastrin – reduce activity of lower esophageal sphincter
3. Presence of hiatus hernia

- ✓ When blood vessels eroded – haemorrhage
- ✓ Persistent reflux – chronic inflammation
- ✓ Extensive damage – healing by fibrosis – shrinkage cause stricture of esophagus – in chronic, leads to Barrett's esophagus

Barretts' esophagus

- It is damage to lower esophagus by acid reflux, which causes the lining to thicken and become red
- Premalignant condition arises with long-standing GERD
- Columnar cells like that in stomach replace the squamous epithelium of lower esophagus

Achalasia

- Peristalsis of lower esophagus impaired, lower esophageal sphincter fails to relax during swallowing
- Cause accumulation of food and drink in esophagus – difficulty in passing to stomach
- Also dysphagia, regurgitation of gastric contents and aspiration pneumonia
- Esophagus dilated, autonomic nerve to esophagus muscle impaired

Tumors of the esophagus

- Benign tumors are rare

Malignant tumors

- More often in males than females
- Most common in lower esophagus, often associated with Barrett's esophagus
- Begins as ulcer that spreads round the circumference – stricture – results in dysphagia

<u>Diseases of the stomach</u>

Gastritis

- It is inflammation of the stomach – acute or chronic

Acute gastritis

- Occurs in response to irritant drugs or alcohol

1. Drugs – NSAIDS ***ex:*** aspirin
2. ***Helicobacter pylori*** infection
3. Physiological stress – ***ex:*** extensive burns and multiple organ failure

Severity

- Mild – asymptomatic, or nausea and vomiting
- Loss of superficial layers of gastric mucosa – called erosions
- Serious cases – multiple erosions – life threatening haemorrhage, haematemesis and melaena

Chronic gastritis

- Milder but lasts longer
- Usually associated with H. pylori infection; also autoimmune or chemical injury

H. Pylori associated gastritis

Autoimmune chronic gastritis

- A progressive disease, destructive inflammation of surface of mucous membrane – extend to whole thickness, gastric glands
- Secretion of HCl and IF reduced
- Autoimmunity – destruction of parietal cells
- Secondary consequences –

1. *Pernicious anaemia – lack of IF*
2. *Increased risk of cancer of stomach*

Peptic ulcer disease

- Occur when stomach acid damages the lining of the GIT
- Affects full thickness of GI mucosa, penetrate the muscle layer of stomach and duodenum
- Caused by disruption of normal balance between corrosive effect of gastric juice and the protective effect of mucus on the gastric epithelial cells
- Damage to cells – leads to ulceration; protected by good blood supply, adequate mucus secretion and epithelial cell replacement
- Most common sites – stomach & first few cm of duodenum; also rarely in esophagus and small intestine

Causes

1. *H. pylori infection – most common*
2. *Use of NSAIDS*
3. *Smoking – delays the healing*

- Incidence – more in men than women

Acute ulcers

- Lesions may be single or multiple
- Found in many sites of stomach and duodenum
- Associated with gastritis, severe stress, shock, burns, severe emotional disturbance after major surgery
- Stressor removed – heal without fibrosis
- Complication – haemorrhage

Chronic ulcers

- Ulcers 2-3 times more common in duodenum than in stomach
- Occur singly in pylorus of stomach or in duodenum
- Heal with formation of fibrous tissue – shrinkage causes

1. Stricture of lumen of stomach
2. Gastric outflow obstruction or stenosis of pyloric sphincter
3. Adhesion to adjacent structures – pancreas, liver or transverse colon

Complications of ulcers

1. ***Perforation*** – contents enter peritoneal cavity causing peritonitis
2. ***Haemorrhage*** – erosion of major artery
3. ***Anaemia***
4. ***Gastric outflow obstruction*** – also called pyloric stenosis – results in vomiting
5. ***Malignancy***

Tumors of the stomach

- Benign tumors are rare

Malignant tumors

- More in men than in women H. pylori infection, smoking, alcohol and diets high in salted, smoked and pickled food – might be reasons
- Local growth – gradual destruction of normal tissue – leads to achlorhydria and pernicious anaemia
- Further growth of tumors – ulceration of surface and infection
- Poor prognsis

Congenital pyloric stenosis

- There is spasmodic constriction of pyloric sphincter – projectile vomiting, fail to put on weight
- To overcome spasms, hypertrophy of muscle of pylorus – cause pyloric obstruction 2-3 wks after birth
- More common in boys

Diseases of the intestines

Appendicitis

- A condition in which appendix become inflammed and filled with pus, causing pain
- Initial cause not known. Microbial infection + obstruction by hard fecal matter, kinking, or fo reign body
- Initial stage – pain localised to central area of abdomen
- Severe cases – progression of microbial growth – leads to suppuration, abscess formation
- Rising pressure – occlusion of local veins, arteries – cause ischaemia – followed by gangrene and perforation
- Peritonitis, abscess formation and adhesions (bands of fibrous scar tissue) – are the complications

Gastrointestinal infections

- Caused by microorganisms in the gut; highly contagious
- Most of these infections spread by faecal-oral route via contaminated food & water
- Preventive measures

1. Clean and safe drinking water
2. Effective sewage disposal
3. Safe food hygiene practices
4. Meticulous hand washing after defaecation and contact with contaminated materials

- Contaminated drinking water – diarrhoea – major cause of illness in all age groups

Typhoid and paratyphoid (enteric) fever

- Caused by *Salmonella typhi* and *S. paratyphi A or B* resp.
- Spread by the faecal-oral route from food, water or any contaminated individuals
- Incubation period – 10-14 days; bacteria invade peyer's patches
- After, microbes spread via blood to liver, spleen and gall bladder

- Febrile illness + malaise, headache, drowsiness and aching limbs
- Inflammation of LI lymphoid tissue, splenomegaly, red spots on the skin
- Typhoid fever serious & fatal without treatment
- Complications – pneumonia, meningitis and typhoid cholecystitis
- Few become carriers
- Paratyphoid fever also similar but milder and of short duration

Other Salmonella infections

- *Salmonella typhimurium* and *S. enteritidis* – confined to GIT & cause enteritis
- Hosts – humans, domestic animals and birds
- In meat, poultry, eggs and milk – infection if cooking does not achieve sterilisation
- Abdominal pain, diarrhoea, vomitings (sometimes)

***Escherichia coli* food poisoning**

- Common source – under-cooked meat & unpasteurized milk
- Adequate cooking, pasteurization – kill *E.coli*

Staphylococcal food poisoning

- Ingested *Staphylococcus aureus* in contaminated food – release toxins – cause acute gastroenteritis
- Acute inflammation with violent vomiting 2-4 hr after ingestion, cause dehydration and electrolyte imbalance

***Clostridium perfringens* food poisoning**

- Anaerobic commensal in intestines of humans and animals
- Ingestion in large numbers – cause food poisoning (spores are source)
- Large outbreaks – in schools and hospitals
- Meat/ poultry cooled slowly/ stored in unrefrigerated conditions/ prepared in advance and kept warm for several hours before serving – dormant spores – viable bacteria
- Release of powerful toxins – watery diarrhoea and severe abdominal pain

Antibiotic-associated diarrhoea

- Antibiotic therapy – death of commensals – *C. difficile* (already present in LI) proliferate – release toxins – damage mucosa – profound diarrhoea
- Hospitalised older adults commonly affected

Campylobacter food poisoning

- Common cause of gastroenteritis, affects mainly young adults and children under 5 yrs
- Present in intestines of birds and animals – transmitted in under-cooked poultry and meat, also spraed in contaminated water and milk

Cholera

- Caused by *Vibrio cholerae*; spread by contaminated drinking water, faeces, vomit, food, hands and fomites
- A very powerful toxin released by bacteria, stimulates intestinal glands to secrete large quantities of water, bicarbonate and chloride
- Persistent watery diarrhoea, severe dehydration and electrolyte imbalance
- Death due to hypovolaemic shock

Dysentery

Bacillary dysentery

- Infection of large intestine by bacteria of Shigella group
- S. dysenteriae causes most severe infection
- Organisms spread by faecal contamination of food, drink, hands and any items handled by infected individuals (fomites)
- Inflammation of mucosa, ulceration, edema with excess mucus secretion
- Severe – acute diarrhoea, with blood and excessive mucus
- Dehydation, electrolyte imbalance and anaemia

Amoebiasis (amoebic dysentery)

- Caused by protozoa *Entamoeba histolytica*; method of spread similar
- Grow, divide and invade mucosal cells – inflammation of colon
- Without treatment – diarrhoea and abdominal pain

Viral gatroenteritis

- Rotavirus and norovirus – cause vomiting and/ or diarrhoea
- Rotavirus – major cause of diarrhoea in young children worldwide
- Norovirus – also called winter vomiting virus; vomiting is main symptom

Inflammatory bowel disease

- Includes two chronic conditions – Crohn's disease and ulcerative colitis

Crohn's disease

- Chronic inflammatory condition of terminal ileum and rectum, usually occurs in young adults
- May affect any part of GIT
- Patchy inflammation with edema of full thickness of wall, partial obstruction of lumen
- Diarrhoea, abdominal pain and weight loss

Ulcerative colitis

- Chronic inflammatory disease of mucosa of colon and rectum – ulcerate and become infected
- Begins in rectum and spread to colon
- Main symptom – bloody diarrhoea

Diverticular disease

- Diverticula – small pouches of mucosa protrude (herniate) into peritoneal cavity through circular muscle fibres of colon
- Occur at weakest portions of intestinal wall
- Diverticulitis occurs as a consequence of diverticulosis when faeces become trapped in diverticula

Tumors of small and large intestines

- Benign and malignant tumors of small intestine are rare

Colorectal cancer

- Most common malignancy of alimentary tract in developed countries
- Most important factor – diet; large quantities of red meat, saturated animal fat, insufficient fiber
- Predisposing diseases – ulcerative colitis and some benign tumors

Hernias

- Hernia – a protrusion of an organ or part of an organ through a weak point or aperture in the surrounding structures
- Hernias of digestive system – a piece of bowel protrudes through a weak point in either the musculature of anterior abdominal wall or an existing opening
- Sites – inguinal, femoral, umbilical, incisional, hiatus, peritoneal and congenital diaphragmatic hernias

Volvulus

- Occurs when a loop of bowel twists, occluding its lumen and resulting in intestinal obstruction
- Usually occurs when there is interruption in blood supply, causing gangrene
- Most common sites – sigmoid colon in adults and small intestine in children

Intussusception

- A length of intestine is invaginated into itself, causing intestinal obstruction
- Most common in children when a piece of terminal ileum is pushed through ileocaecal valve

Intestinal obstruction

- Not a disease but arises as a consequence of many other conditions
- ***Mechanical causes*** – strangulated hernia, intissusception, volvulus or adhesions, stenosis of intestinal wall etc.
- ***Neurological causes*** – partial or complete loss of peristaltic activity
- ***Vascular causes*** – blood supply to a segment of bowel is cut off, ischaemia followed by infarction and gangrene – damaged bowel unable to function
- ***Effects*** – abdominal pain, vomiting and costipation

Malabsorption

- Impaired absorption of nutrients and water – not a disease
- It is the result of abnormal changes of one or more of following:

1. Villi in small intestine – coeliac disease, tropical sprue
2. Digestion of food
3. Absorption or transport of nutrients from the small intestine

Coeliac disease

- Main cause of malabsorption
- Due to abnormal, autoimmune reaction to protein gluten present in wheat, barley and rye
- Marked villous atrophy in jejunim
- Malabsorption characterised by passage of loose, pale-colored, fatty stools (steatorrhoea)

Tropical sprue

- Malabsorption syndrome characterised by chronic diarrhoea, weight loss and malabsorption of nutrients
- Partial villous atrophy

Diseases of the pancreas

Pancreatitis

- Inflammation of pancreas due to activation of precursor enzymes in the pancreas
- Acute or chronic

Acute pancreatitis

- Cause – gall stones, excessive alcohol intake and others
- Mild – common, damage of cells near the duct, complete recovery
- Sever – widespread damage with necrosis and haemorrhage

Chronic pancreatitis

- Either due to repeated attacks of acute pancreatitis or may arise gradually without evidence of pancreatic disease
- Irreversible structural changes, fibrosis and distortion of main pancreatic duct
- Most common cause – excessive alcohol consumption, dietary factors and malnutrition

Cystic fibrosis (mucoviscidosis)

- An inherited life-threatening disorder that damages lungs and digestive tract
- Secretions of exocrine glands are abnormally thick due to abnormal chloride transport across secretory epithelia of pancreas
- Highly viscous mucus secreted by walls of ducts – obstruction, parenchymal cell damage, formation of cysts, and defective enzyme secretion

Tumors of the pancreas

- Benign are very rare

Malignant tumors

- Common and affect more men than women
- Cause – smoking, alcohol intake in excess, use of aspirin and coexsiting conditions lie diabetes mellitus and chronic pancreatitis
- Tumors affect exocrine head, development of jaundice with itching, weight loss

Diseases of the liver

- In most liver diseases, hepatocellular failure and portal hypertension are present

Acute hepatitis

- It is a wide variety of conditions characterized by acute inflammation of hepatic parenchyma or injury to hepatocytes

- Groups of hepatocytes die by necrosis
- Causes

1. *Viral infections* – viral hepatitis (A-E)
2. *Toxic substances* – drug and its metabolites – ex: paracetamol, indomethacin etc.
3. *Circulatory disturbances* – hypoxia by fibrosis in liver, compression of portal vein, hepatic artery etc.

Chronic hepatitis

- Any form of hepatitis that persists for more than 6 months
- May be caused by viruses, alcohol or drugs
- Continuous progressive inflammation with cell necrosis and formation of fibrous tissue – leads to cirrhosis of the liver

Cirrhosis of the liver

- Chronic liver damage resulting in inflammation and necrosis and replacement with fibrous tissue
- Most common causes

1. Hepatitis B and C infections
2. Excessive alcohol consumption
3. Recurrent obstruction of the biliary tract

- Hyperplasia of hepatocytes adjacent to damaged cells – formation of nodules
- Liver failure when cell regeneration do no keep pace with destruction

Liver Failure

- Occurs when liver function is markedly impaired
- Acute or chronic
- Causes – acute viral hepatitis, extensive necrosis due to poisoning (overdose of drugs, hepatotoxic chemicals etc), cirrhosis of liver
- Liver failure causes

1. Hepatic encephalopathy (disorientation, confusion, coma)
2. Blood coagulation defects
3. Oliguria and renal failure
4. Edema and ascites

5. Jaundice

Tumors of the liver

Malignant tumors

- Frequently occurs due to cirrhosis
- Also after hepatitis B and C

Diseases of Gall bladder and Bile ducts

Gallstones (cholelithiasis)

- Consist of deposits of the constituents of bile, most commonly cholesterol
- Many small stones or one or more large stones may form
- Causes include – changes in composition of bile, high blood cholesterol, female gender, obesity, pregnancy, diabetes mellitus

Cholecystitis

- Inflammation of the gall bladder due to the presence of gallstones

Acute cholecystitis

- Occurs when a gallstone becomes stuck in the cystic duct, often after a fatty meal
- Strong peristaltic contractions – severe acute abdominal pain
- Bile unable to leave gall bladder – inflammation occurs
- Severe attack – nausea and vomiting
- If bacterial infection – risk of perforation and peritonitis

Cholangitis

- Inflammation of bile ducts caused by bacterial infection – accompanied by abdominal pain, fever and jaundice
- Occurs due to gall bladder diseases
- Infection can spread upwards causing liver abscesses

Tumors of the biliary tract

Malignant tumors

- Rare
- Gallstones are always present

Jaundice

- Not a disease in itself

- Abnormal bilirubin metabolism and excretion – yellowing of the skin and mucous membrane
- Conjugated in liver and excreted in bile
- Unconjugated bilirubin – toxic effect on brain cells
- Serum bilirubin rise to 40-50 µmol/L before yellow coloration is evident in skin and conjunctive
- Accompanied by itching (pruritis) due to irritating effects of bile salts on skin

Types of jaundice

- Depending on the stage of bilirubin processing – end result rise in blood bilirubin levels

Prehepatic jaundice

- Due to increased hemolysis of RBC

Neonatal haemolytic jaundice

- Occurs in many babies, esp. in premature

Intrahepatic jaundice

- Result of damage to liver itself by viral hepatitis, toxic substances amoebiasis and cirrhosis
- Excess bilirubin in liver – conjugated form – excreted in urine – dark in color

Posthepatic jaundice

- Obstruction to flow of bile in biliary tract due to gallstones in common bile duct, tumor of head of pancreas etc.