CHAPTER 24: DIGESTIVE SYSTEM

MOVEMENTS

ORGANS      SECRETIONS

DISORDERS      ABSORPTION

DIGESTIVE SYSTEM

DEVELOPMENT      EXCRETION

AGING

DIGESTION: breaking down of larger food particles into molecules small enough to enter body cells

ABSROPTION: passage of smaller food molecules into blood and lymph

DIGESTIVE SYSTEM: composed by the organs that collectively perform digestion and absorption

DIGESTIVE SYSTEM

ALIMENTARY CANAL

(MOUTH, PHARYNX, ESOPHAGUS, STOMACH, SMALL INTESTINE, LARGE INTESTINE, ANUS)

ACCESSORY ORGANS

(TEETH, TONGUE, SALIVARY GLANDS, LIVER, GALL BLADDER, PANCREAS)
DIGESTIVE PROCESSES:

INGESTION  MIXING & PROPULSION

ABSORPTION

SECRETION  DIGESTION  DEFECATION (EGESTION)

MECHANICAL  CHEMICAL

IMPORTANCE OF FOOD:

1. CONTAINS VARIETY OF NUTRIENTS (MOLECULES NEEDED FOR BUILDING NEW & REPAIRING DAMAGED BODY TISSUES & SUSTAINING NEEDED CHEMICAL REACTIONS).

2. SOURCE OF ENERGY THAT
   i. drives chemical reactions occurring in every cell
   ii. is needed for muscle contraction
   iii. is needed for conduction of nerve impulses
   iv. is needed for secreting & absorptive activities of many cells

INTRODUCTION:

Food contains substances and energy the body needs to form all cellular components. It must be broken down through digestion to molecular size before they can be absorbed by the digestive system & used by cells.

The organs that collectively perform these functions constitute the digestive system (Fig. 24.1, p 853)

The medical professions that deal with the structures, functions, and disorders of the alimentary canal are called Gastroenterology for the upper end of the system and Proctology for the lower end.
OVERVIEW:
ORGANIZATION:

1. Two major sections perform the processes required to prepare food for the use in the body.
2. The GI (gastrointestinal) tract is open at both ends for the transit of food during processing; it includes mouth, esophagus, stomach, small intestine, large intestine, and anus.
3. The accessory structures that contribute to the food processing include teeth, tongue, salivary glands, liver, gall bladder, and pancreas.

BASIC ACTIVITIES:

1. **Ingestion**: food intake (eating)
2. **Secretion**: Cells within walls of GI tract and accessory organs secrete approximately 7 liters of water, acid, buffers, and enzymes into the lumen of the tract.
3. **Mixing & propulsion**: Alternating contraction and relaxation of smooth muscles in the muscularis mix food & secretions & propel them toward the anus.
4. **Mechanical digestion**: includes mastication (chewing) & various movements (peristalsis, segmentation, churning) of the GI tract that aid in chemical digestion.
5. **Chemical digestion**: includes series of hydrolytic (catabolic) reactions that break down macromolecules (carbohydrates, proteins, and lipids) into smaller molecules that are usable by the body cells.
6. **Absorption**: passage of the end products of digestion from the GI tract into blood & lymph for distribution to cells.
7. **Defecation (egestion)**: emptying of the rectum – eliminating indigestible substances from the GI tract.
LAYERS OF GI TRACT: (Fig. 24.2, p 854)

Basic arrangement of layers in the GI tract from inside out includes *mucosa, submucosa, muscularis, & serosa.*

**Composition of mucosa:**

1. **Epithelium:** non-keratinized stratified squamous epithelium in the mouth cavity, pharynx, esophagus, and anal region; in stomach, small and large intestines – simple columnar epithelium containing mucus-secreting, enzyme-secreting, HCl-secreting, and some enteroendocrine cells that secrete hormones (help regulate digestive processes).

2. **Lamina propria:** 3 components – (a) areolar tissue that adheres the epithelium to lower layers, (b) system of blood and lymph vessels for absorption and transportation of food, and (c) nerves & sensors. The lymph system is part of the MALT (mucosa associated lymphatic tissues) that monitors and produces an immune response to pathogens (passing with food).

3. **Muscularis mucosae:** causes local folding of the mucosal layer, consisting of smooth muscle fibers (inner circular and outer longitudinal), increasing surface area for digestion and absorption.

**Submucosa:**

Consists of areolar connective tissue and binds the mucosa to the muscularis, highly vascular containing a portion of the submucosal plexus (plexus of Meissner) – part of autonomic nerve supply to smooth muscle cells of muscularis mucosae and blood vessels, may also contain glands and lymphatic tissue. [Examples: Brunner’s glands in duodenum, Peyer’s patches in ileum]

**Muscularis:** (also called muscularis externa)

Mouth, pharynx, & superior part of the esophagus contain skeletal muscle producing voluntary swallowing and the external anal sphincter offers voluntary control of defecation. In the rest of the tract it is formed of smooth muscle fibers, generally, in two sheets, inner sheet of circular fibers and outer sheet of longitudinal fibers (stomach has an additional innermost oblique layer). It provides
involuntary contractions that help break down food physically mix it with digestive secretions, propel it along the GI tract. The major nerve supply includes myenteric plexus (plexus of Auerbach) which controls the motility of the GI tract.

**Serosa: (Visceral peritoneum)**
Layer of those portions of GI tracts that are suspended in the abdominopelvic cavity; visceral membrane is made of connective tissue and simple squamous epithelium; serosa is absent in esophagus, instead it has adventitia composed of areolar connective tissue.

**PERITONEUM: (Fig. 24.3, pp.856-857)**

It is the largest serous membrane in the body.

1. **Parietal peritoneum**: lines the wall of the abdominal cavity.
2. **Visceral peritoneum**: covers most of the abdominal organs and forms their serosa.
3. **Peritoneal cavity**: contains serous fluid.
4. **Extensions of peritoneum**: include mesentery, mesocolon, falciform ligament, lesser and greater omentum.
5. **Large folds of peritoneum** weave between the viscera, functioning to support organs and to contain blood and lymphatic vessels, & nerves of the abdominal organs.

*(Peritonitis: inflammation of peritoneum)*

**MOUTH: (Fig. 24.4, p 858)**

Formed by cheeks, hard and soft palates, lips and tongue.

**Oral cavity proper**: space extending from the gums & teeth to the fauces (opening between oral cavity and the pharynx).

**Vestibule**: bounded externally by the cheeks and lips and internally by the gums and teeth.

**Tongue** together with associated muscle forms the floor of the oral cavity.

  Skeletal muscles are covered with mucous membrane; extrinsic and intrinsic muscles are responsible for tongue movement, food manipulation for chewing & swallowing and speech.
Lingual frenulum – membrane attached to the midline of the undersurface of the tongue; if too short, a condition called ankyloglossia, it limits tongue’s movement impairing speech, surgically correctable.
Upper surface and sides of tongue are covered with papillae (filiform, fungiform, & circumvallate, some contain taste buds).
Ducts of lingual glands (von Ebner’s) surround circumvallate papillae.
Dorsum of tongue contains lingual glands that secrete lingual lipase – initiates digestion of triglycerides into fatty acids and monoglycerides

SALIVARY GLANDS: (Fig. 24.5, p 859)

Major portion of saliva is secreted by salivary glands; lie outside the mouth and pour their contents into the ducts and empty into the oral cavity; remainder comes from buccal glands in the mucus membrane that lines the mouth.

There are three pairs of salivary glands – parotid (largest), submandibular (submaxillary), and sublingual.

Functions of saliva: (a) lubricates and dissolves food and starts digestion of carbohydrates (by ptyalin – amylolytic enzyme or salivary amylase); (b) keeps mucous membranes of mouth and throat moist.

Mumps: inflammation and enlargement of parotid glands caused by infection with myxovirus (mumps virus). If mumps is contracted by a male past puberty, there is a possibility of inflammation of testis (orchitis) and, occasionally, sterility.

Chemical composition of saliva:
- 99.5% water
- 0.5% solutes {e.g., salts, dissolved gases, various organic substances, and enzymes (salivary amylase, lysozymes)}

Lysozymes are bacteriolytic.
Saliva is hypotonic to other body fluids; pH is slightly acidic, varies from 6.35 to 6.85. Salivary amylase, also called ptyalin, begins digestion of carbohydrates (starch is converted to maltose, a disaccharide)
Each salivary gland supplies different ingredients to saliva:

**Parotid glands**: secrete watery serous liquid containing salivary amylase.

**Submandibular glands**: some cells are similar to parotid gland and secrete salivary amylase, but some cells secrete mucus; secretion thickened with mucus and quite a bit of enzymes.

**Sublingual glands**: formed mostly of mucous cells; secrete much thicker fluid because of mucus and only a small amount of amylase.

Salivation is entirely under nervous control (by ANS); parasympathetic stimulation promotes continuous secretion of saliva, while sympathetic stimulation dominates during stress resulting in dryness of mouth.

Daily average saliva production: 1,000-1,500 ml

**PHYSIOLOGY OF DIGESTION IN THE MOUTH:**

1. Through mastication (chewing) food is mixed with saliva and shaped into a bolus that can be swallowed easily.
2. Salivary amylase converts polysaccharides (starches) to disaccharides (maltose). Food is usually swallowed too quickly for all starches to be reduced to disaccharides in the mouth. Salivary amylase continues to act on starches in the stomach for about an hour or so before HCl inactivates it.
3. Lingual lipase, secreted by lingual glands, initiates the digestion of triglycerides into fatty acids and monoglycerides.

(LEARN ALSO SUMMARY OF DIGESTIVE ACTIVITIES IN THE MOUTH ON TABLE 24.1 ON PAGE 863!)

**PHARYNX**: (Fig. 24.8, p 864)
It is funnel-shaped tube that extends from the internal nostrils to the esophagus posteriorly and the larynx anteriorly; composed of skeletal muscle and lined by mucous membrane which is a nonkeratinized stratified squamous epithelium. It is common chamber for digestive as well as respiratory systems.
It is divisible into three regions: nasopharynx functions in respiration only, whereas oropharynx or buccopharynx and laryngopharynx have digestive and respiratory functions.

**PHYSIOLOGY OF DEGLUTITION:**

1. Deglutition moves a bolus from the mouth to the stomach, facilitated by saliva and mucus; it involves mouth, pharynx, and esophagus.
2. Consists of 3 stages: i. voluntary stage, ii. pharyngeal stage (involuntary), and iii. esophageal stage (involuntary). [Fig. 24.8 p 864]
3. Receptors in the oropharynx stimulate the deglution center in the medulla and the lower pons of the brain stem.

**ESOPHAGUS:** (Fig. 24.8, p 864; Figs 24.9 & 24.10, p 865)

Collapsible, muscular tube that lies behind the trachea and connects the pharynx to the stomach.

Wall of esophagus contains mucosa, submucosa, muscularis, and adventitia.

Passes bolus into stomach by peristalsis – progressive, wavelike contractions of the muscularis
- controlled by medulla oblongata
- circular muscle fibers of the section lying just above the bolus contract, constricting the esophageal wall & squeezing the bolus toward the stomach
- longitudinal muscle fibers inferior to the bolus contract, shortening the inferior section and pushing its walls outward so that it can receive the bolus

[Passage of bolus (solid or semisolid food) takes 4-8 seconds to reach the stomach from mouth.]

Contains an upper and lower esophageal sphincters.

**Achalasia:** a malfunction of the myenteric plexus (plexus of Auerbach) prevents normal relaxation of the lower esophageal sphincter, thus impeding entrance of food into the stomach.
Gastroesophageal Reflux Disease (GERD): If the lower esophageal sphincter fails to close adequately after food has entered the stomach, the stomach content can back up or reflux into the inferior part of the esophagus. This condition is known as GERD. Hydrochloric acid (HCl) from the stomach contents irritate the esophageal wall, resulting in the burning sensation called \textit{heartburn}, because it is experienced in a region very near the heart, even though it is unrelated to any heart problem. (ALSO LEARN THE SUMMARY OF DIGESTIVE ACTIVITIES IN THE PHARYNX AND ESOPHAGUS IN TABLE 24.2 ON PAGE 866!)

\textbf{STOMACH}: (Figs. 24.11 & 24.12, pp. 867-869)

\textbf{Anatomy (gross)}:

1. \textbf{J-shaped enlargement of the GI tract} which begins at the bottom of the esophagus (\textit{lower esophageal} or \textit{cardiac sphincter}) and ends at the pyloric sphincter. (Fig. 24.11)
2. It serves as a mixing and holding area for food, begins the digestion of proteins, and continues the digestion of triglycerides; converting a bolus to a liquid called chyme.
3. \textbf{4 anatomical subdivisions: cardia, fundus, body, and pylorus.}
   1. When empty (about the size of a large sausage), it remains in collapsed condition and the mucosa lies in folds called rugae.
   4. \textbf{Pylorospasm} and \textbf{pyloric stenosis} are two abnormalities of the pyloric sphincter that can occur in newborns; both functionally block or partially block the exit of food from the stomach into the duodenum and must be treated with drugs (relax the muscle fibers of the sphincter for the treatment of pylorospasm) or surgery (for pyloric stenosis).

\textbf{HISTOLOGY} [Fig 24.12, pp. 868-869]

Same organization as the GI tract: mucosa, submucosa, muscularis, and serosa.
Mucosa lies in large folds, rugae; it consists of a layer of simple columnar epithelial cells, lamina propria, and muscularis mucosae. Epithelial cells extend down into lamina propria, forming gastric pits and gastric glands. Gastric glands consist of 4 types of exocrine cells and one type of enteroendocrine cells.

Exocrine cells are: (i) surface mucous cells, secreting mucus, (ii) mucous neck cells, secreting mucus, (iii) parietal or oxyntic cells, secreting HCl and intrinsic factor (essential for absorption of Vitamin B₁₂ in the small intestine), and (iv) chief or zymogenic cells, secreting pepsinogen and gastric lipase. Enteroendocrine cells known as G cells secrete the hormone gastrin into the bloodstream.

The submucosa is formed of areolar connective tissue. The muscularis has three layers of smooth muscle: inner oblique, middle circular and outer longitudinal layers. The serosa is a part of the visceral peritoneum – at the lesser curvature it becomes lesser omentum, and at the greater curvature it becomes greater omentum.

Secretion of HCl by parietal cells (Fig. 24.13, p 870): These cells secrete hydrogen ions (H⁺) and chloride ions (Cl⁻) separately, but the net effect is the secretion of hydrochloric acid (HCl). Proton pumps powered by H⁺/K⁺ ATPases actively transport H⁺ into the lumen while bringing K⁺ into the cell. At the same time Cl⁻ and K⁺ diffuse out through Cl⁻ and K⁺ channels in the apical membrane (apical membrane). Carbonic anhydrase (CA), plentiful in parietal cells, catalyzes formation of H₂CO₃ from water and CO₂. As carbonic acid dissociates, it provides a ready source of H⁺ for the proton pumps and also generates HCO₃⁻. As HCO₃⁻ builds up in the cytosol, it exits the parietal cell in exchange for Cl⁻ via Cl⁻ / HCO₃⁻ antiporters in the basolateral membrane (next to lamina propria). [HCO₃⁻ diffuses into nearby blood capillaries. This “alkaline tide” of bicarbonate ions entering the blood stream after a meal may be large enough to elevate slightly blood pH and make urine more alkaline.]
PHYSIOLOGY OF DIGESTION & ABSORPTION IN THE STOMACH

MECHANICAL DIGESTION: consists of peristaltic movements and churning called mixing waves.

CHEMICAL DIGESTION: consists mostly of conversion of proteins into peptides by pepsin (most effective in a very acidic environment (pH 2.0). HCl is produced by parietal cells. This acid activates pepsinogen to pepsin. Other enzymes contribute to digestion in the stomach: (i) gastric lipase splits certain molecules of butterfat of milk into fatty acids and monoglycerides and has a limited role in the adult stomach; (ii) lingual lipase digests fats in the stomach (increased acidity inactivates this enzyme); (iii) rennin (milk-curdling enzyme) aids in digestion of milk in infants. (Summary of digestive activities in the stomach is given in Table 24.3, p 871)

ACID CHYME, a thin liquid (semisolid), is formed in the stomach as a result of mechanical and chemical activities of the stomach.

ABSORPTION IN THE STOMACH: stomach wall is usually impermeable to most substances, but some water, electrolytes, certain drugs (e.g., aspirin), and alcohol can be absorbed through the gastric lining.

REGULATION OF GASTRIC SCRETION AND MOTILITY

Gastric secretion is regulated by neural and humoral (hormonal) mechanisms. There are three phases: cephalic, gastric, and intestinal. (Fig. 24.14, p 871)

Cephalic phase stimulates gastric secretion and motility by way of vagus nerve (parasympathetic).

Gastric phase begins when food enters the stomach: the increase in pH and distension of the stomach wall stimulate the chemoreceptors and mechanoreceptors (stretch receptors)
respectively and result in secretion of gastrin by the G cells and increased peristalsis by the muscularis. Neural negative feedback regulates the pH of gastric juice and gastric motility during the gastric phase. [Fig 24.15, p 872]

Gastrin secretion is stimulated when pH rises and inhibited when the pH of gastric juice goes below 2.0 – this negative feedback mechanism helps provide an optimal low pH for the functioning of pepsin, killing of microbes, and denaturing of proteins in the stomach.

Gastrin stimulates growth of the gastric glands (its target cells are parietal and chief cells) and secretion of large amounts of gastric juice; it also strengthens contraction of lower esophageal sphincter, increases motility of the stomach, and relaxes the pyloric and ileocecal sphincters.

ACh (acetylcholine), released by parasympathetic fibers and gastrin by G cells stimulate parietal cells to secrete more HCl when histamine is present. Certain drugs, e.g., Zantac (ranitidine hydrochloride), Tagamat, etc. are used to block histamine receptors on parietal cells to prevent hypersecretion of HCl as in peptic ulcers.

When partially digested food enters duodenum, it triggers enterogastric reflex and secretion of secretin and CCK (cholecystokinin) by the enteroendocrine cells of the intestinal mucosa. The effect is the inhibition of gastrin secretion by G cells.

REGULATION OF GASTRIC EMPTYING

- Gastric emptying is the periodic release of chyme from the stomach into the duodenum. It is stimulated by two factors: (i) nerve impulses in response to distension of stomach and (ii) stomach gastrin in response to the presence of certain types of food. (Fig. 24.16, p 873)
- Most food leaves stomach 2-6 hours after ingestion, carbohydrates earliest, followed by proteins and fats.
- Gastric emptying is inhibited by enterogastric reflex and by hormones, secretin and CCK.
• Vomiting: Forcible expulsion of contents of upper GI tract (stomach and sometimes duodenum) through mouth (prolonged vomiting could be serious, especially in infants and elderly people, because loss of gastric juice and fluids can lead to disturbances in fluid and acid-base balance).

PANCREAS

GROSS ANATOMY: (FIG. 24.17, P 875)
• divided into 3 parts: head, body, and tail; connected to the duodenum via pancreatic duct (duct of Wirsung) and accessory duct (duct of Santorini)

HISTOLOGY: (Fig. 18.18, p 615)
formed of pancreatic acini and islets of Langerhans
Acini secrete a mixture of fluid and digestive enzymes – (i) starch-splitting enzyme – pancreatic amylase; (ii) protein-splitting enzymes – trypsin, chymotrypsin, & carboxypeptidase; (iii) fat-splitting enzyme – pancreatic lipase; (iv) nucleic acid-splitting enzymes – ribonuclease and deoxyribonuclease; (v) sodium bicarbonate - bicarbonate ions neutralize acid chyme and raise pH to slightly alkaline (pH 7.1-8.0), halt stomach pepsin activity and promote activity of pancreatic enzymes.

Islets of Langerhans are formed of alpha, beta, delta, and F cells secreting respectively glucagon, insulin, somatostatin (GHIH), and pancreatic polypeptide.

Inflammation of pancreas is called pancreatitis.

REGULATION OF PANCREATIC SECRETION

Pancreatic secretion is regulated by neural and hormonal mechanisms. (Fig.24.18, p 876)
• During cephalic and gastric phases of gastric digestion, parasympathetic impulses are transmitted along the vagus nerve to the pancreas that stimulate increased secretion of pancreatic enzymes.
• Acidic chyme containing partially digested fats and proteins enters small intestine.
• In response to fatty acids and amino acids, some enteroendocrine cells (CCK cells) in the small intestine secrete cholecystokinin (CCK) into blood. Other enteroendocrine cells (S cells) in the small intestinal epithelium release secretin into blood.
• CCK stimulates a pancreatic secretion that is rich in digestive enzymes and secretin stimulates the flow of pancreatic juice that is rich in bicarbonate ions.

LIVER: (Fig. 24.17, p 875; Figures 24.19-24.21, pp. 878-879)

ANATOMY & HISTOLOGY: right and left lobes separated by falciform ligament (more in the lab); each lobe of liver is made of lobules (roughly hexagonal); each lobule consists of hepatocytes, sinusoids (capillaries), stellate reticuloendothelial cells called Kupffer’s cells (phagocytes), and a central vein; hepatocytes secrete bile, which is transported to gall bladder for concentration and temporary storage.

Liver receives double blood supply – from hepatic artery and hepatic portal vein. Hepatic veins drain the liver. (Fig. 24.20, p 879)

DIGESTIVE FUNCTION OF LIVER: makes bile from breakdown of erythrocytes (RBCs) - bile is partially an excretory product (containing components of worn-out RBCs) and partially a digestive secretion.
Function of bile: (i) detergent action decreases surface tension of fat globules in food, (ii) mixing action (agitation) emulsifies fat to increase surface area for chemical digestion.

CONTROL OF BILE SECRETION: Rate of bile secretion is regulated by neural and hormonal mechanisms as well as by volume of hepatic blood flow and concentration of bile salts in the blood. (Fig. 24.21, p 879)

OTHER FUNCTIONS OF LIVER:
carbohydrate, protein, and fat metabolism
removal of drugs and hormones from blood
storage of vitamins and mineral
phagocytosis
activation of Vitamin D

Inflammation of liver is called **hepatitis**, which is often associated with jaundice, yellowish coloration of the white (sclera) of eyes, skin, and mucous membranes due to buildup of bilirubin, the principal pigment of bile in the body.

**GALL BLADDER** (Fig. 24.17, p 875)

It is located in the fossa of the visceral surface of the liver.

**FUNCTION:** it stores and concentrates bile.

**STRUCTURE**

smooth muscle in wall
mucosa thrown into rugae
cystic duct joins common hepatic duct to form the common bile duct

**CONTENTS OF BILE IN GALL BLADDER:**

- Water
- Bile salts
- Cholesterol
- Bile pigments
  - Biliverdin
  - Bilirubin

Under the influence of CCK bile is ejected into the common bile duct via the cystic duct. (Fig. 24.21, p 879)

**EFFECTS OF MAJOR DIGESTIVE HORMONES**

Gastrin, secretin, and CCK regulate gastric secretion and motility, as well as secretion of the pancreas and liver and release of bile from the gall bladder. Each hormone is secreted in response to specific chemical conditions of the chyme and stimulates or inhibits the appropriate secretion or contraction in the requisite organs in order to maintain the chemical conditions within the homeostatic range. (Table 24.4, p 881)
MECHANISM OF RELEASE OF BILE FROM GALL BLADDER

Secretin is released into duodenum and taken up by tributaries of hepatic portal vein that carries it to liver. Outcomes: (a) increased bile secretion from liver, (b) with the action of CCK the gall bladder and the cystic duct contract, (c) relaxation of the sphincter of Oddi (discussed in the lab), (d) bile released into duodenum.

CAUSES OF GALLSTONES:

1. Too much absorption of water from bile
2. Too much absorption of bile acids from bile
3. Too much cholesterol in bile
4. Inflammation of epithelium

SMALL INTESTINE (Figs. 24.22-24.26, pp. 882-890)

GROSS ANATOMY: [Fig 24.22, p 882]
Extends from pyloric sphincter to ileocecal sphincter.
3 divisions: duodenum, jejunum, and ileum.

HISTOLOGY: [Fig 24.23 & Fig 24.24, pp. 883-885]
Same four layers: mucosa (simple columnar epithelium), submucosa, muscularis, and serosa.
Highly adapted for digestion and absorption.
Its glands produce enzymes and mucus, and the microvilli, villi, and circular folds (plicae circulares) provide a large surface area for digestion and absorption.

DUODENUM:
Major Functions:
- Collection place for 4 juices – chyme, bile, pancreatic juice, and intestinal juice.
• Brunner’s glands (in the submucosa) secrete alkaline mucus, which helps neutralize gastric acid in the chyme.

JEJUNUM:
Major Functions:
• Mechanical digestion
  Peristalsis
  Segmentation (peristalsis without directional movement)
• Chemical digestion

Different Enzymes:
  Brushborder Enzymes:
  • Enterokinase: converts trypsinogen to trypsin.
  • Peptidase: converts polypeptides and dipeptides into amino acids.
  • Sucrase: converts sucrose to glucose and fructose.
  • Maltase: converts maltose to 2 glucose molecules.
  • Lactase: converts lactose into glucose and galactose. Some people develop lactose intolerance because of their inability to produce lactase.
  • Galactase: converts galactose to glucose. Deficiency causes buildup of galactose in blood, a condition called galactosemia.

Mucus production by goblet cells is enhanced by the acid chyme.

[Learn the digestive enzymes from Table 24.5, p 887!]

ILEUM:
Structural Makeup: Peyer’s patches (aggregates of lymphatic nodules) in the submucosa; plicae circulares – mucosa contains circular folds.
Major Function: Absorption

REGULATION OF INTESTINAL SECRETION AND MOTILITY
• Most important mechanism is the action of local reflexes in response to the presence of chyme.
• Hormones (VIP – vasoactive intestinal polypeptide) assume a role.
• Parasympathetic impulses increase motility, whereas sympathetic impulses decrease it.

**PHYSIOLOGY OF ABSORPTION IN SMALL INTESTINE** (Fig 24.25, pp. 888-889)

• Absorption is the passage of the end products of digestion into blood and lymph.
• Absorption occurs by diffusion, facilitated diffusion, osmosis, and active transport.
• Monosaccharides, amino acids, and short-chain fatty acids pass into the blood capillaries.
• Long chain fatty acids and monoglycerides are absorbed as part of **micelles**, resynthesized into triglycerides, and formed into protein-coated spherical masses called **chylomicrons**.

**[Micelle:** a spherical aggregate of bile salts that dissolves fatty acids and monoglycerides so that they can be absorbed into small intestinal epithelial cells.]
• **Chylomicrons** are taken up by the lacteal (special lymph capillary) of a villus.
• From the lacteal they enter lymphatic system and then to cardiovascular system, finally reaching the liver or adipose tissue.
• Plasma lipids (fatty acids, triglycerides, and cholesterol) are insoluble in water and body fluids. To be transported in blood and utilized by the cells, lipids must be combined with transporters called lipoproteins to make them soluble (e.g., HDL – high-density lipoproteins, LDL – low-density lipoproteins, and VLDL – very low-density lipoproteins).
• Absorption also includes water, electrolytes, and vitamins. Fat-soluble vitamins are Vitamin A, Vitamin D, Vitamin E and K; water-soluble vitamins are Vitamin B complexes and Vitamin C. **[Learn Table 24.6, p 891!]**
LARGE INTESTINE (Fig. 24.26-Fig 24.28, pp. 890-894)

STRUCTURAL DIVISIONS:
Ascending colon
  Cecum, appendix, right colic flexure (hepatic flexure)
Transverse colon
  Left colic flexure (splenic flexure)
Descending colon
Sigmoid colon
Rectum (not a part of the colon)
  Anal canal: lower part of the rectum
  Anal columns: mucous membrane arranged in longitudinal folds
    (contain a network of arteries and veins)
  Anus: opening of the anal canal to the exterior
  Internal anal sphincter: smooth muscle wrapped around anus
  External anal sphincter: skeletal muscle wrapped around anus distal to the internal anal sphincter

HISTOLOGY: All four layers
  Mucosa: no villi, simple columnar epithelium, lamina propria, and muscularis mucosae; epithelium consists mostly of absorptive and goblet cells, the former absorb water and the latter secrete mucus that lubricates the colonic contents as they pass through.

STRUCTURAL CHARACTERISTICS:
  Haustra: pockets of large intestinal wall, involved in hastral churning (a type mechanical mixing of chyme).
  Taenia coli: 3 bands of longitudinal smooth muscle running lengthwise along outer surface of the large intestine.

FUNCTIONS OF LARGE INTESTINE:
- Vitamin production (by microorganisms)
  Vitamin K (helps in thrombin production)
Vitamin B$_{12}$

- Storage of wastes
- Formation of feces
  Absorption of water
  Removal of water and electrolytes
  [500-800 ml of chyme enters colon but only a volume of 100-200 ml is left to form feces]
- Bacterial action of *E. coli*
  Breaks up chyme
  Ferments carbohydrates
  Produces gas (flatus)
  [Fermentation: anaerobic digestion of carbohydrates into carbon dioxide and alcohol]

$$C_6H_{12}O_6 = 2CO_2 + 2C_2H_5OH$$
  Produces odor due to skatole, indole, and H$_2$S
  [Bad odor is a natural selective process]

**CONTENTS OF FECES:**
- Epithelial cells
- Microorganisms
- Chyme
- Water
- Bile pigment (bilirubin, gives the color of feces)
- Methane (contributes to global warming)

**ACTIONS OF MUCUS IN LARGE INTESTINE:**
- Protects wall from autodigestion
- Holds fecal material together
- Lubricates the passage for easier movement of feces toward the rectum

{DISCUSS DAILY VOLUMES OF FLUID INGESTED, SECRETED, ABSORBED, AND EXCRETED FROM GI TRACT IN FIG. 24.26, p 890}

[Table 24.7, p 896 for Summary of Digestive Activities in the Large Intestine]
MECHANISM OF DEFCATION:

A. Stretching of lower rectum by bulk of feces initiates defecation reflex.

B. Involuntary reflexes:
   - Nerve impulses pass to spinal cord and brain
   - Nerve impulses go to hypothalamus
     - Result: Relaxation of internal anal sphincter
   - Some impulses go to cerebral cortex to alert the individual for the “next move”.

C. Voluntary reflexes:
   - Individual voluntarily positions himself or herself to defecate.
   - Motion of feces continues to stretch lower rectum beyond the internal anal sphincter.
   - Cerebrum releases voluntary constraints on external anal sphincter, as a result, sphincter relaxes and opens.
   - Intraabdominal pressure increases by contraction of the abdominal wall muscles.
   - Pressure is exerted on rectum, as a result, feces exit anal canal through anus.
   - Discharge of feces removes original stretch in rectum and stops further process of defecation.

DIAGNOSES FROM FECES:

Worms, such as, pinworms, tapeworms, roundworms can be detected from fecal examination. Black feces indicate iron or bleeding; bloody stool may indicate cancer or bleeding fissure; green stool is obtained in case of diarrhea in children – the green color is due to the pigment biliverdin which could not be transformed into bilirubin due to too fast passage of feces.

AGING AND THE GI TRACT:

Decreasing secretory mechanisms, decreasing motility of the digestive organs, loss of strength and tone of digestive muscular tissue, are some of the changes associated with aging.

Specific changes include loss of taste, periodontal disease, hiatal hernia, cancer of the esophagus, gastritis, peptic ulcer, gastric cancer,
duodenal ulcers, appendicitis, gall bladder problem, acute pancreatitis, constipation, colon and rectal cancer, hemorrhoids, diverticular diseases of the colon, etc.

**DISORDERS: HOMEOSTATIC IMBALANCES** [Learn from pp. 899-901]

Some of the disorders are already mentioned above. There are several types of hepatitis e.g., Hepatitis A (infectious hepatitis), Hepatitis B, Hepatitis C, Hepatitis D, and Hepatitis E. Anorexia nervosa is a chronic eating disorder characterized by self-induced weight loss, body image and other perceptual disturbances, and physiologic changes that result from nutritional depletion.

**DEVELOPMENTAL ANATOMY:**
Endoderm forms the epithelium and glands of most of the GI tract; mesoderm forms the smooth muscle and connective tissue of the GI tract.

**CHAPTER 25 OVERVIEW**

**FOOD INTAKE**

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**METABOLISM**

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**BASAL METABOLIC RATE**

**METABOLISM:** All the biochemical reactions that occur within an organism. It includes the synthetic (anabolic) and decomposition (catabolic) reactions.

**BASAL METABOLIC RATE (BMR):** The rate of metabolism measured under standard or basal conditions (awake, at rest, and fasting).
**NUTRIENT:** A chemical substance in food that provides energy, forms new body components, or assists in the functioning of various body processes. [Nutrients include carbohydrates, lipids, proteins, vitamins, minerals, and water.]

**METABOLISM & LIVER FUNCTIONS**

**TYPES:**

A. **Anabolism:** buildup of larger molecules from smaller ones.

Examples:
- Glucose + Glucose + E → Maltose + water
- Glucose + ...+ Glucose + E → Glycogen + water

Reaction Type: **endergonic**, always requires energy

II. **Catabolism:** breakdown of larger molecules into smaller ones.

Examples:
- Maltose + water → Glucose + Glucose + E
- Sucrose + water → Fructose + Glucose + E

Reaction Type: **exergonic**, always produces energy

**Importance:**
- Production of body heat
- Energy production for anabolic reactions

**METABOLISM OF CARBOHYDRATES**

The liver is the main monitor of blood sugar (glucose) level. As blood passes through the liver, it senses the level of sugar, whether the level is too high or low. Accordingly, the liver deletes or adds sugar to or from blood.

**Processes of metabolism include:**

1. **Glycogenesis:** when glucose is too high in blood (above 100-mg/100 ml), the excess glucose is converted into glycogen in the liver and thus removed from blood.
2. **Glycogenolysis**: when glucose is too low in blood, it is added from
the liver to blood through breakdown of glycogen and thus blood
glucose level goes back to normal.

3. **Gluconeogenesis**: when glucose is not available at all, glucose
breakdown products are formed from noncarbohydrates (lipids and
proteins).

4. **Glycolysis**: occurs in all cells; gives energy to cell. It is a series of
chemical reactions in the cytosol of a cell in which a molecule of
 glucose is converted to two molecules of pyruvic acid with
production of 2 ATPs.

**METABOLISM OF FATS**
The liver oversees the level of fat in the blood by breaking down
neutral fats to fatty acids and glycerol. It then may use fatty acids as
energy source, or convert them to new fats that may then be needed by
the cells.

**Importance of fats:**
- Components of cell membrane.
- Components of hormones
- Secondary energy source after carbohydrates
- Insulation

**METABOLISM OF PROTEINS**
- Amino acids, brought to liver, are used to make larger proteins,
rather than being used as an energy source.
- Harmful effects of protein catabolism can occur when their
breakdown products accumulate, especially ammonia.

**DETOXIFICATION OF NITROGENOUS WASTES**
- Poisonous ammonia is released in protein metabolism.
- Liver converts ammonia to urea to carry away via urine.

\[ \text{CO}_2 + 2\text{NH}_3 = \text{CONH}_2\text{NH}_2 + \text{H}_2\text{O} \]

**MANUFACTURE OF PLASMA PROTEINS:**
- Albumin
- Fibrinogen
• Globulin
• Prothrombin

STORAGE OF VITAMINS:

Vitamin A, E, D, and B₁₂

CONJUGATION OF STEROID HORMONES

This is a procedure, where the liver chemically converts steroid hormones to water-soluble forms so that they can pass into blood, then to kidney, and subsequently to be excreted in the urine.

BILIRUBIN FORMATION

When red blood cells die, hemoglobin is broken down into heme and globin. The heme is converted to bilirubin (bile pigment).

CONSEQUENCES OF GLUCOSE FOLLOWING ABSORPTION INTO BLOOD:

Once glucose enters blood from small intestine, it enters the tributaries of superior mesenteric vein and then flows into the hepatic portal vein before entering the liver. Liver monitors blood sugar levels, noting when it is above or below its normal level (average 100mg/100ml):

A. When glucose level reaches above 100mg/100ml, the following occurs:
   Glycogenesis: formation of glycogen from glucose. Excess glucose is converted to glycogen by hepatocytes.

   Insulin
   Glucose → Glucose-6-phosphate
   Glucokinase
   Glycogen synthetase
Glucose-6-phosphate → Glycogen

[Glucokinase is a hexokinase and a phosphorylating enzyme.]

Insulin promotes phosphorylation (addition of phosphate) to glucose. This is a necessary step to make glycogen. The greater is the amount of glucose above 100mg/100ml, greater the rate of glycogenesis.

B. **When glucose level is below 100mg/100ml**, the following occurs:

**Glycogenolysis**: catabolism (breakdown) of glycogen to glucose.

Phosphorylase

Glycogen → Glucose-6-phosphate

Glucose-6-phosphatase

Glucose-6-phosphate → Glucose

Since glucose is reformed, this raises blood glucose level to normal.

C. **When glucose is needed for energy**, then the following occurs:

**Glycolysis**: Breakdown of glucose to two molecules of pyruvic acid.

Glucose → 2 pyruvic acid + 2 ATP

[Oxygen is not needed, but insulin is indirectly needed because it promotes uptake of glucose into the liver so that the reaction can take place.]

This process occurs anytime, anywhere if energy is needed to do cellular work. Basically, it supplies the energy to activate the Krebs cycle that occurs during aerobic cellular respiration.

**Aerobic cellular respiration:**
2 Pyruvic acid + 6 O₂ → Krebs Cycle → Electron Transport → 6H₂O + 6 CO₂ + 36 ATP
Pyruvic acid must first be converted to Acetyl CoenzymeA (Actyl CoA) in order to enter Krebs Cycle.

D. **When glucose is not immediately available at all**, then the following occurs:

**Gluconeogenesis**: formation of glucose breakdown products from non-glucose precursors.

**Situations causing need for gluconeogenesis**:

- Starvation or excessive fasting
- Chronic disease situations, such as, arthritis, polio, cancers, pneumonia, etc.
- Diabetic shock

**Breakdown products of gluconeogenesis**:

- Pyruvic acid
- Acetyl Coenzyme A
- Acetoacetic acid

**METABOLIC INTERRELATIONS BETWEEN CARBOHYDRATES, FATS, AND PROTEINS**

PROTEINS GLYCOGEN NEUTRAL FAT
AMINO ACIDS

GLUCOSE

GLYCOLYSIS

GLYCEROL + FATTY ACIDS

ATP

R-NH₂

NH₃

PYRUVIC ACID

UREA

CO₂

ACETYL

COENZYMME A

KREBS CYCLE → CO₂

CARRIER HYDROGEN

OXIDATIVE

O₂ → PHOSPHORYLATION → H₂O

ATP

ACTION OF GLUCOSE IN SKELETAL MUSCLES:

• If glucose is not immediately needed, the following happens:

Glycogenesis

• If glucose is needed for energy, the following incidents occur:
**Glycogenolysis** to yield glucose, making it immediately available for glycolysis

Glycolysis follows as soon as glucose available

**ACTION OF GLUCOSE IN OTHER TISSUES:**

Glucose is used in cells for energy via glycolysis. Excess glucose is converted into fat (lipogenesis). [This process is promoted by insulin; it is how one gets fat by eating too much sugar.]

**FATE OF EXCESS GlUCOSE:**

A. **Hepatic glycogen threshold activated:**

[A threshold is the limit to which a process can occur.] When no more glycogen can be made, excess glucose is anabolized, converted into fat (lipogenesis). Conversion to fat occurs only over long-term over-ingestion of carbohydrates.

B. **Renal threshold for glucose:**

Too much intake of glucose at one time overwhelms the liver’s ability to make glycogen; extra glucose stays in the blood and passes to the kidneys. Renal tubules can only absorb up to **180mg/100ml** (blood) of glucose; any extra above this level remains in the renal filtrate and flows into urine (glycosuria).

**ACTION OF HORMONES ON CARBOHYDRATE METABOLISM**

I. **Insulin:** manufactured by the beta cells of pancreatic islets

Stimulus for secretion: glucose level above 100mg/100ml

Higher the glucose level, the more insulin is secreted.]

**Insulin promotes:**

Glucose uptake by cells.
Glycogenesis: principally occurs in hepatocytes, but also occurs in all types of muscle.
Lipogenesis: very common in adult-type diabetes. {Insulin inhibits breakdown of fats.}
Active transport of amino acids into cells, mainly muscle cells. [Once amino acids enter cells, they can be built up into new proteins.]

II. **Glucagon**: manufactured by the alpha cells of pancreatic islets.
Stimulus for secretion: extremely low blood glucose levels.
When blood glucose levels are very low, as in insulin shock, a serious stress occurs and this causes a sympathetic response in which epinephrine (adrenalin) is secreted by adrenal medulla. Epinephrine, in turn, causes the release of glucagon.

Functions of glucagon in carbohydrate metabolism:
- Stimulates glycogenolysis (this occurs only in liver).
- Stimulates gluconeogenesis.

I. **Epinephrine**: produced by the chromaffin cells of the adrenal medulla.
Stimulus for secretion: any stressful situation.

Functions of epinephrine in carbohydrate metabolism:
- Stimulates glycogenolysis.
- Stimulates gluconeogenesis.
- Stimulates breakdown of fats.

**DISEASES OF CARBOHYDRATE METABOLISM:**

**Diabetes mellitus**: It is disease in which the liver fails to adequately monitor the glucose level of the blood; insulin is not properly metabolized, and blood sugar levels remain high.
Two types:
1. **Type I Diabetes**: Insulin-dependent
2. **Type II Diabetes**: Non-insulin dependent
Detrimental carbohydrate metabolism:

- Inability to use glucose due to lack of insulin.
- Hyperglycemia: occurs when glucose builds up in blood.
- Glycosuria: occurs when glucose level surpasses renal threshold in kidneys, and spills into the urine.
- Continued need for energy via cellular respiration: since glucose is not available to cells (due to lack of insulin) despite a very high level of glucose in blood, fat begins to break down (gluconeogenesis).
- Increased fat catabolism for energy.
- Acidosis (ketoacidosis) due to keto acid buildup.
- Coma and unconsciousness follow.