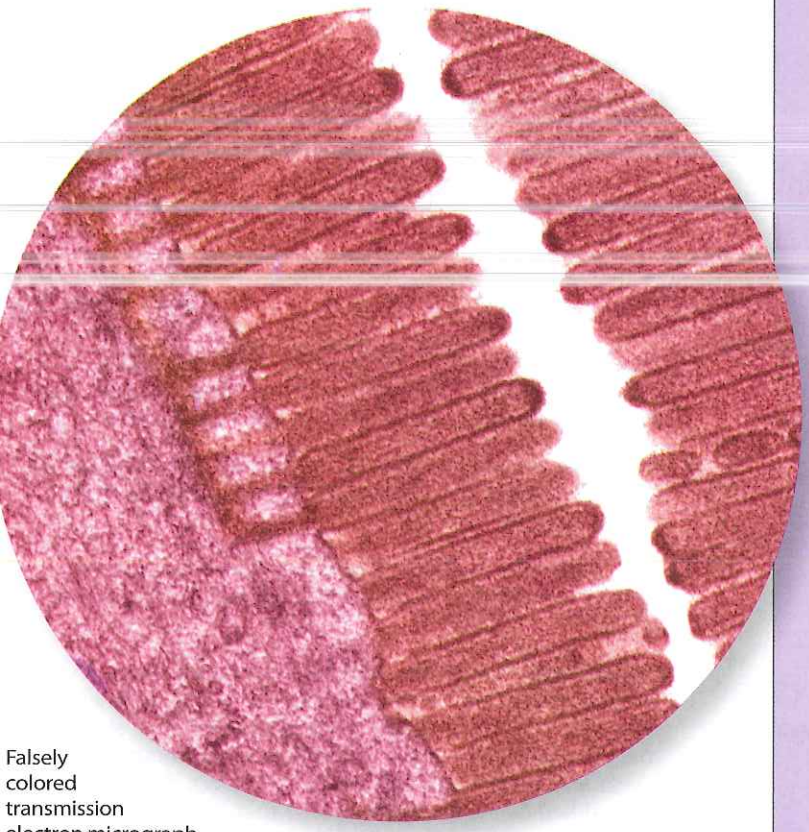
Unit V

17 Digestive System



Falsely colored transmission electron micrograph (TEM) of microvilli on intestinal cells. The cytoplasm of the cell on the left shows as purple.

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Module 12: Digestive System

Learning Outcomes

After you have studied this chapter, you should be able to:

17.1 Introduction

1 Describe the general functions of the digestive system. (p. 649)
2 Name the major organs of the digestive system. (p. 649)

17.2 General Characteristics of the Alimentary Canal

3 Describe the structure of the wall of the alimentary canal. (p. 649)
4 Explain how the contents of the alimentary canal are mixed and moved. (p. 651)

17.3 Mouth

5 Describe the functions of the structures associated with the mouth. (p. 653)
6 Describe how different types of teeth are adapted for different functions, and list the parts of a tooth. (p. 655)

17.4–17.10 Salivary Glands—Large Intestine

7 Locate each of the organs and glands; then describe the general function of each. (pp. 658–686)
8 Identify the function of each enzyme secreted by the digestive organs and glands. (pp. 658–679)
9 Describe how digestive secretions are regulated. (pp. 658–684)
10 Explain control of movement of material through the alimentary canal. (pp. 660–686)
11 Describe the mechanisms of swallowing, vomiting, and defecating. (pp. 661, 667, 686)
12 Explain how the products of digestion are absorbed. (p. 680)

17.11 Life-Span Changes

13 Describe aging-related changes in the digestive system. (p. 686)

LEARN PRACTICE ASSESS

Understanding Words

aliment-, food: *aliment*ary canal—tubelike part of the digestive system.

cari-, decay: dental *cari*es—tooth decay.

cec-, blindness: *cec*um—blind-ended sac at the origin of the large intestine.

chym-, juice: *chyme*—semifluid paste of food particles and gastric juice formed in the stomach.

decidu-, falling off: *decidu*ous teeth—teeth shed during childhood.

frenul-, bridle, restraint: *frenul*um—membranous fold that anchors the tongue to the floor of the mouth.

gastr-, stomach: *gastr*ic gland—part of the stomach that secretes gastric juice.

hepat-, liver: *hepat*ic duct—duct that carries bile from the liver to the bile duct.

hiat-, opening: esophageal *hiat*us—opening through which the esophagus penetrates the diaphragm.

lingu-, tongue: *lingu*al tonsil—mass of lymphatic tissue at the root of the tongue.

peri-, around: *peri*stalsis—wavelike ring of contraction that moves material along the alimentary canal.

pyl-, gatekeeper, door: *pyl*oric sphincter—muscle that serves as a valve between the stomach and small intestine.

rect-, straight: *rect*um—distal part of the large intestine.

sorpt-, to soak up: ab*sorpt*ion—uptake of substances.

vill-, hairy: *vill*i—tiny projections of mucous membrane in the small intestine.

The Gut Microbiome

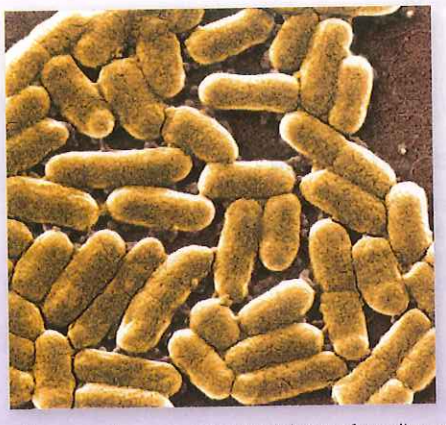
The Human Microbiome Project is cataloging the bacterial residents of five areas: the skin, mouth, nasal passages, digestive system, and urogenital tract. Researchers identify these bacteria from bacterial DNA sequences.

More than 600 species of bacteria make their home in human mouths; each person's mouth has about 200 of these species. The other end of the digestive tract, the "distal gut microbial community," includes more than 6,800 species, with each person's large intestine harboring 500 to 1,000 of these species.

To track the establishment of the human gut microflora, researchers probed the DNA in a year's worth of stool collected daily from the soiled diapers of fourteen infants. Bacteria in the stool varied greatly from baby to baby at the outset, but by their first birthdays, the gut communities were more alike and more closely resembled the microbial communities in adults.

Some of the microorganisms that live in our intestines are crucial to our health. They produce more than eighty types of enzymes that digest plant polysaccharides that our bodies cannot break down, and they help our bodies process certain sugars, synthesize essential vitamins and amino acids, and break down certain toxins and drugs. The observation that the composition of the gut microbiome varies somewhat from person to person may be one reason why some people can eat a great deal and not gain weight, yet others gain weight easily.

Unusual bacterial communities in our bodies can reflect disease. Specific microfloral profiles are associated with colorectal cancer, diarrhea, inflammatory bowel disease, and peptic ulcers. A new focus of drug development is to target our microbial residents. Also, we can add bacteria to foods to prevent certain infections, an approach called probiotics. For example, certain *Lactobacillus* strains added to yogurt can help protect against *Salmonella* foodborne infection. ■



Several million microorganisms are normal residents of our digestive tracts. *Escherichia coli*, pictured here (6,800×), produce vitamin K, and if present in low numbers, will not cause diarrhea.

17.1 INTRODUCTION

Digestion (di-jest'yun) is the mechanical and chemical breakdown of foods into forms that cell membranes can absorb. *Mechanical digestion* breaks large pieces into smaller ones without altering their chemical composition. *Chemical digestion* breaks food into simpler chemicals. The organs of the **digestive system** carry out these processes, as well as ingestion, propulsion, absorption, and defecation.

The digestive system consists of the **alimentary canal** (al"i-men'tar-e kah-nal'), extending from the mouth to the anus, and several accessory organs, which release secretions into the canal. The alimentary canal includes the mouth, pharynx, esophagus, stomach, small intestine, large intestine, and anal canal. The accessory organs include the salivary glands, liver, gallbladder, and pancreas. Figure 17.1 and reference plates 4, 5, and 6 (pp. 42–44) show the major organs of the digestive system.

The digestive system originates from the inner layer (endoderm) of the embryo, which folds to form the tube of the alimentary canal. The accessory organs develop as buds from the tube.

PRACTICE

1 What are the general functions of the digestive system?

2 Which organs constitute the digestive system?

17.2 GENERAL CHARACTERISTICS OF THE ALIMENTARY CANAL

The alimentary canal is a muscular tube about 8 meters long that passes through the body's thoracic and abdominopelvic cavities (fig. 17.2). The structure of its wall, how it moves food, and its innervation are similar throughout its length.

Structure of the Wall

The wall of the alimentary canal consists of four distinct layers developed to different degrees from region to region. Although the four-layered structure persists throughout the alimentary canal, certain regions are specialized for particular functions. Beginning with the innermost tissues, these layers, shown in figure 17.3, include the following:

1. The **mucosa** (mu-ko'sah), or **mucous membrane** is formed of surface epithelium, underlying connective tissue (lamina propria), and a small amount of smooth muscle (muscularis mucosae). In some regions, the mucosa is folded, with tiny projections that extend into the passageway, or **lumen**, of the digestive tube. These folds increase the absorptive surface area. The mucosa also has glands that are tubular invaginations into which the lining cells secrete mucus and digestive enzymes. The mucosa protects the tissues beneath it and carries on secretion and absorption.

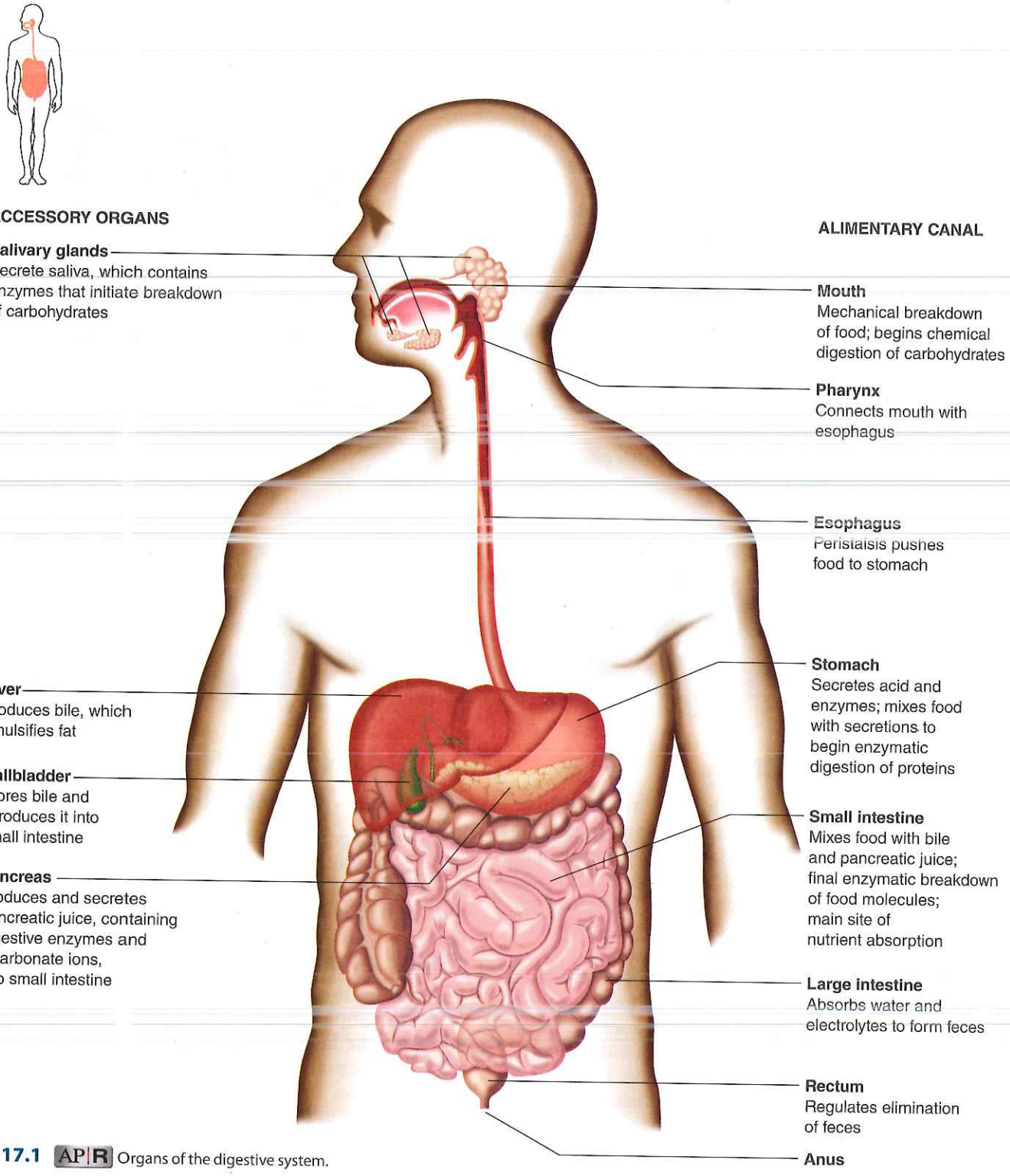


FIGURE 17.1 AP|R Organs of the digestive system.

2. The **submucosa** (sub″mu-ko′sah) contains considerable loose connective tissue as well as glands, blood vessels, lymphatic vessels, and nerves. Its vessels nourish the surrounding tissues and carry away absorbed materials.
3. The **muscular layer**, which provides movements of the tube, consists of two coats of smooth muscle tissue. The fibers of the inner coat encircle the tube. When these *circular fibers* contract, the diameter of the tube decreases. The fibers of the outer muscular coat run lengthwise. When these *longitudinal fibers* contract, the tube shortens. Coordinated contractions of both muscle layers cause movements associated with digestion and absorption of food.
4. The **serosa** (ser-o′sah), or **serous layer,** is the outer covering of the tube. It is composed of the *visceral peritoneum,* which is formed of epithelium on the

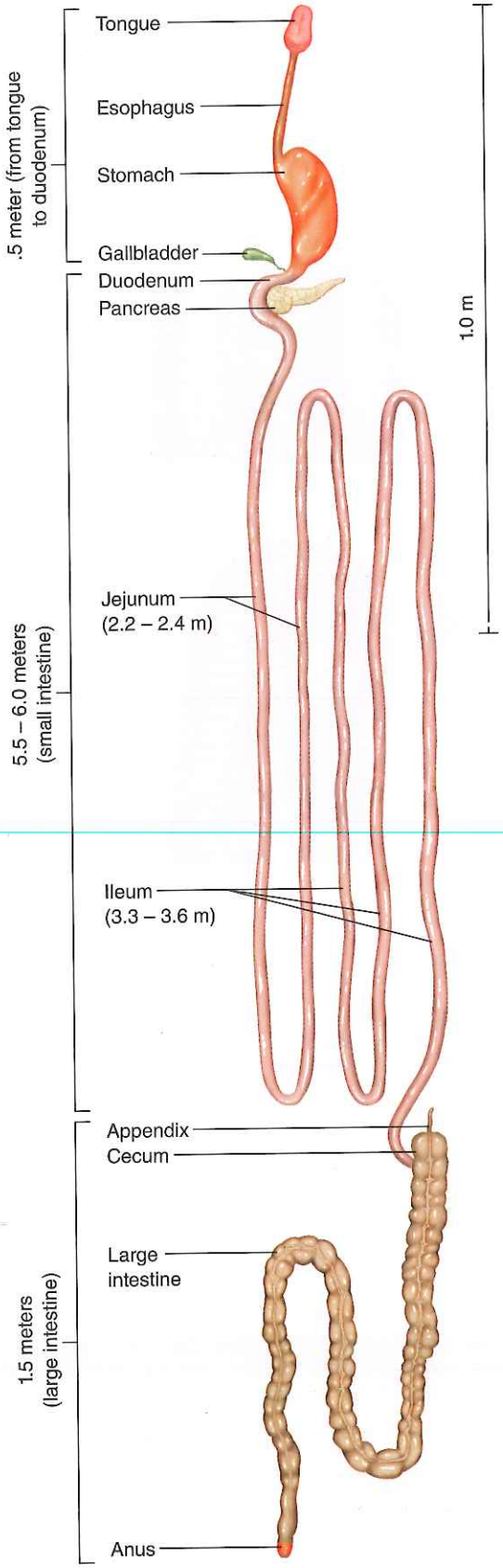


FIGURE 17.2 The alimentary canal is a muscular tube about 8 meters long.

Q: *What is the distance from the tongue to the duodenum, in English units?*

Answer can be found in Appendix G on page 938.

outside and connective tissue beneath. The cells of the serosa protect underlying tissues and secrete serous fluid, which moistens and lubricates the tube's outer surface. As a result, the organs in the abdominal cavity slide freely against one another.

Table 17.1 summarizes the characteristics of the layers of the alimentary canal wall.

Movements of the Tube

The motor functions of the alimentary canal are of two basic types—*mixing movements* and *propelling movements*. Mixing occurs when smooth muscles in small segments of the tube contract rhythmically. For example, when the stomach is full, waves of muscular contractions move along its wall from one end to the other (fig. 17.4*a*). These waves pass every twenty seconds or so, and they mix foods with the digestive juices that the mucosa secretes. In the small intestine, **segmentation** is a type of movement that aids mixing by alternately contracting and relaxing the smooth muscle in segments of the organ (fig. 17.4*b*). Because segmentation does not follow a set pattern, materials are not moved along the tract in one direction.

Propelling movements include a wavelike motion called **peristalsis** (per″i-stal′sis), in which a ring of contraction occurs in the wall of the tube and moves progressively along its length (fig. 17.4*c*). At the same time, the muscular wall just ahead of the ring relaxes—a phenomenon called *receptive relaxation*. As the wave of contraction moves along the tube, it pushes the contents of the tube ahead of it. Peristalsis begins when food expands the tube. It causes the sounds that can be heard through a stethoscope applied to the abdominal wall.

A "GI camera" the size of a large vitamin pill can image the alimentary canal, revealing blockages and sites of bleeding. The patient swallows the capsule, which contains a camera, a light source, radio transmitter, and batteries. Peristalsis moves it along, and about six hours after swallowing, it transmits images from the small intestine to a device worn on the physician's belt. The information goes to a computer, which downloads still or video images. The device, which is disposable, leaves the body in the feces within a day or two.

Innervation of the Tube

Branches of the sympathetic and parasympathetic divisions of the autonomic nervous system extensively innervate the alimentary canal. Some of these postganglionic fibers, which are associated with the tube's muscular layer, maintain muscle tone and regulate the strength, rate, and velocity of muscular contractions. Many of the postganglionic fibers are organized into a network or nerve plexus within the wall of the canal (see fig. 17.3). The *submucosal plexus* is important in controlling secretions by the gastrointestinal tract. The *myenteric plexus* of the muscular layer is more extensive and controls gastrointestinal motility. The plexuses also include sensory neurons.

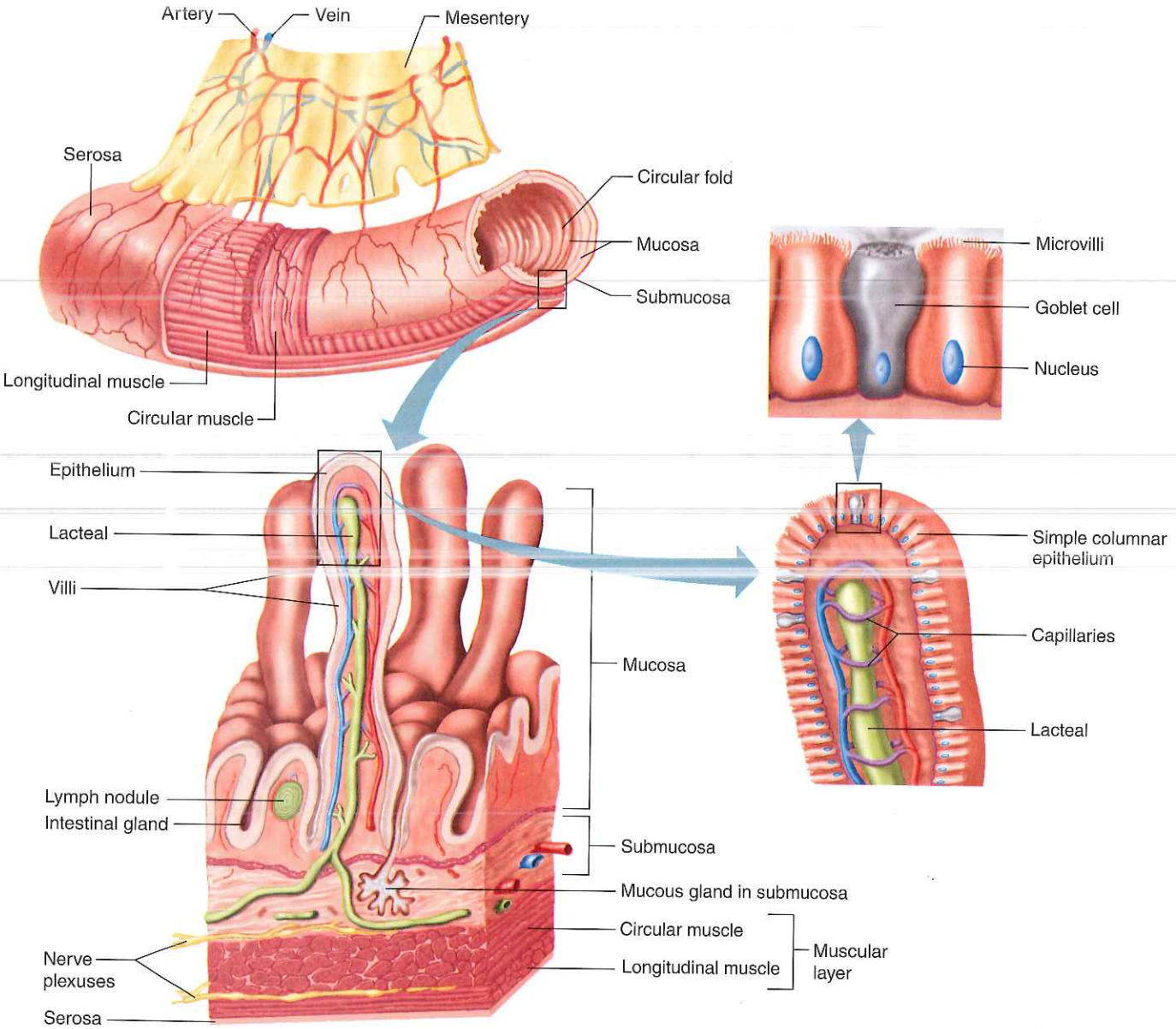
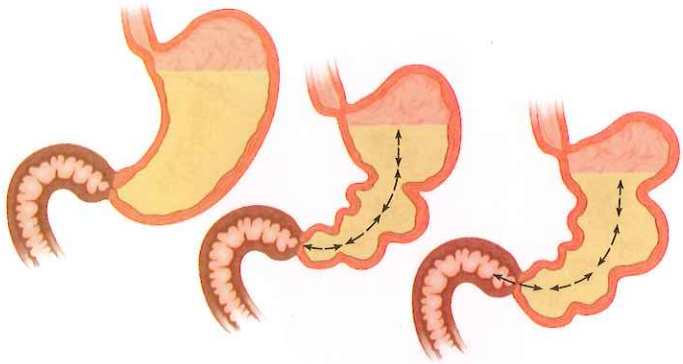


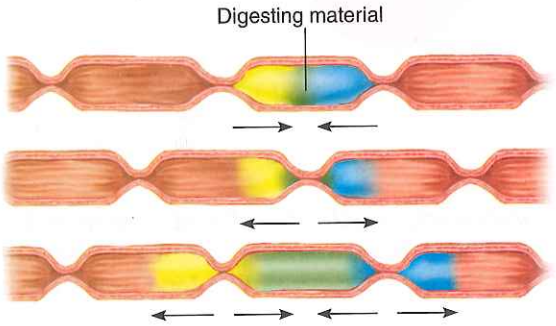
FIGURE 17.3 The wall of the small intestine, as in other portions of the alimentary canal, consists of four layers: an inner mucosa, a submucosa, a muscular layer, and an outer serosa.

TABLE 17.1 | Layers of the Wall of the Alimentary Canal

Layer	Composition	Function
Mucosa	Epithelium, connective tissue, smooth muscle	Protection, secretion, absorption
Submucosa	Loose connective tissue, blood vessels, lymphatic vessels, nerves	Nourishes surrounding tissues, transports absorbed materials
Muscular layer	Smooth muscle fibers in circular and longitudinal groups	Movements of the tube and its contents
Serosa	Epithelium, connective tissue	Protection, lubrication



(a)



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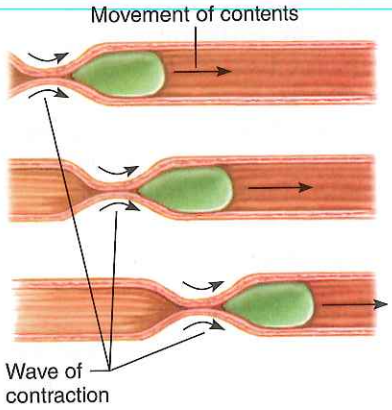


(c)

FIGURE 17.4 Movements through the alimentary canal. (*a*) Mixing movements occur when small segments of the muscular wall of the stomach rhythmically contract. (*b*) Segmentation mixes the contents of the small intestine. (*c*) Peristaltic waves move the contents along the canal.

Parasympathetic impulses generally increase the activities of the digestive system. Some of these impulses originate in the brain and are conducted through branches of the vagus nerves to the esophagus, stomach, pancreas, gallbladder, small intestine, and proximal half of the large intestine. Other parasympathetic impulses arise in the sacral region of the spinal cord and supply the distal half of the large intestine.

The effects of sympathetic impulses on digestive actions usually oppose those of the parasympathetic division. That is, sympathetic impulses inhibit certain digestive actions. For example, such impulses inhibit mixing and propelling movements, but stimulate contraction of the sphincter muscles in the wall of the alimentary canal, blocking movement of materials through the tube.

PRACTICE

3 Describe the wall of the alimentary canal.

4 Name the types of movements in the alimentary canal.

5 How do parasympathetic nerve impulses affect digestive actions? What effect do sympathetic nerve impulses have?

17.3 MOUTH

The **mouth,** the first portion of the alimentary canal, receives food and begins digestion by mechanically breaking up solid particles into smaller pieces and mixing them with saliva. This action is called *mastication* (mas″tĭ-ka′shun). The mouth also functions as an organ of speech and sensory reception. It is surrounded by the lips, cheeks, tongue, and palate and includes a chamber between the palate and tongue called the *oral cavity,* as well as a narrow space between the teeth, cheeks, and lips called the *vestibule* (fig. 17.5 and reference plate 9, p. 47).

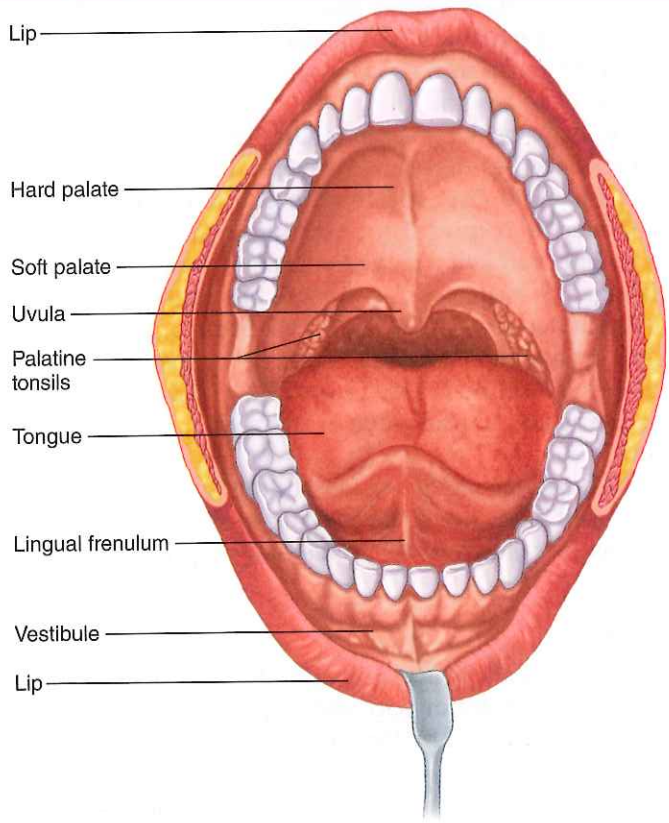


FIGURE 17.5 The mouth is adapted for ingesting food and beginning digestion, both mechanically and chemically.

Cheeks and Lips

The **cheeks** form the lateral walls of the mouth. They consist of outer layers of skin, pads of subcutaneous fat, muscles associated with expression and chewing, and inner linings of moist, stratified squamous epithelium.

Cells lining the cheek are sampled to provide DNA for genetic testing. A person scrapes the inside of the cheek with a cotton swab or swishes with mouthwash and expectorates into a small tube. The sample is sent to a lab, the DNA extracted, and clinically meaningful gene variants identified.

The **lips** are highly mobile structures that surround the mouth opening. They contain skeletal muscles and sensory receptors useful in judging the temperature and texture of foods. Their normal reddish color is due to the many blood vessels near their surfaces. The external borders of the lips mark the boundaries between the skin of the face and the mucous membrane that lines the alimentary canal.

Tongue

The **tongue** (tung) is a thick, muscular organ that occupies the floor of the mouth and nearly fills the oral cavity when the mouth is closed. Mucous membrane covers the tongue, and a membranous fold called the **lingual frenulum** (ling'gwahl fren'u-lum) connects the midline of the tongue to the floor of the mouth.

The *body* of the tongue is largely composed of skeletal muscle fibers that run in several directions. Muscular action mixes food particles with saliva during chewing and moves food toward the pharynx during swallowing. The tongue also helps move food underneath the teeth for chewing. The surface of the tongue has rough projections, called **papillae** (pah-pil'a) (fig. 17.6). Some of these provide friction, which helps handle food. Other papillae contain most of the taste buds (see chapter 12, p. 454). Some taste buds are scattered elsewhere in the mouth, particularly in children.

The posterior region, or *root,* of the tongue is anchored to the hyoid bone. It is covered with rounded masses of lymphatic tissue called **lingual tonsils** (ton'silz) (fig. 17.7).

Palate

The **palate** (pal'at) forms the roof of the oral cavity and consists of a hard anterior part and a soft posterior part. The *hard palate* is formed by the palatine processes of the maxillae in front and the horizontal portions of the palatine bones in back. The *soft palate* forms a muscular arch, which extends posteriorly and downward as a cone-shaped projection called the **uvula** (u'vu-lah).

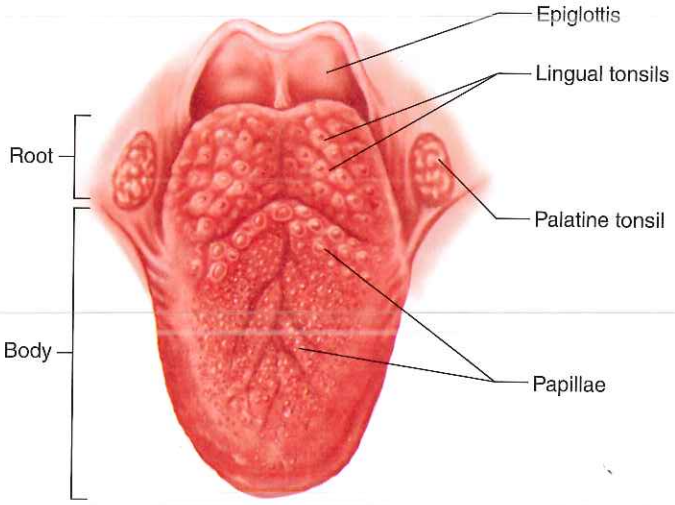


FIGURE 17.6 The surface of the tongue, superior view.

During swallowing, muscles draw the soft palate and the uvula upward. This action closes the opening between the nasal cavity and the pharynx, preventing food from entering the nasal cavity.

In the back of the mouth, on either side of the tongue and closely associated with the palate, are masses of lymphatic tissue called **palatine** (pal'ah-tīn) **tonsils** (fig. 17.7). These structures lie beneath the epithelial lining of the mouth and, like other lymphatic tissues, help protect the body against infections (see chapter 16, p. 621).

Other masses of lymphatic tissue, called **pharyngeal** (fah-rin'je-al) **tonsils,** or *adenoids,* are on the posterior wall of the pharynx, above the border of the soft palate (fig. 17.7). If the adenoids enlarge and block the passage between the nasal cavity and pharynx, they may be surgically removed.

The palatine tonsils are common sites of infection and become inflamed in *tonsillitis.* Infected tonsils may swell so greatly that they block the passageways of the pharynx and interfere with breathing and swallowing. Because the mucous membranes of the pharynx, auditory tubes, and middle ears are continuous, an infection can spread from the throat into the middle ears (otitis media).

When tonsillitis recurs and does not respond to antibiotic treatment, the tonsils may be surgically removed. Such tonsillectomies are done less often today than they were a generation ago because the tonsils' role in immunity is now recognized.

PRACTICE

6 What are the functions of the mouth?

7 How does the tongue function as part of the digestive system?

8 Where are the tonsils located?

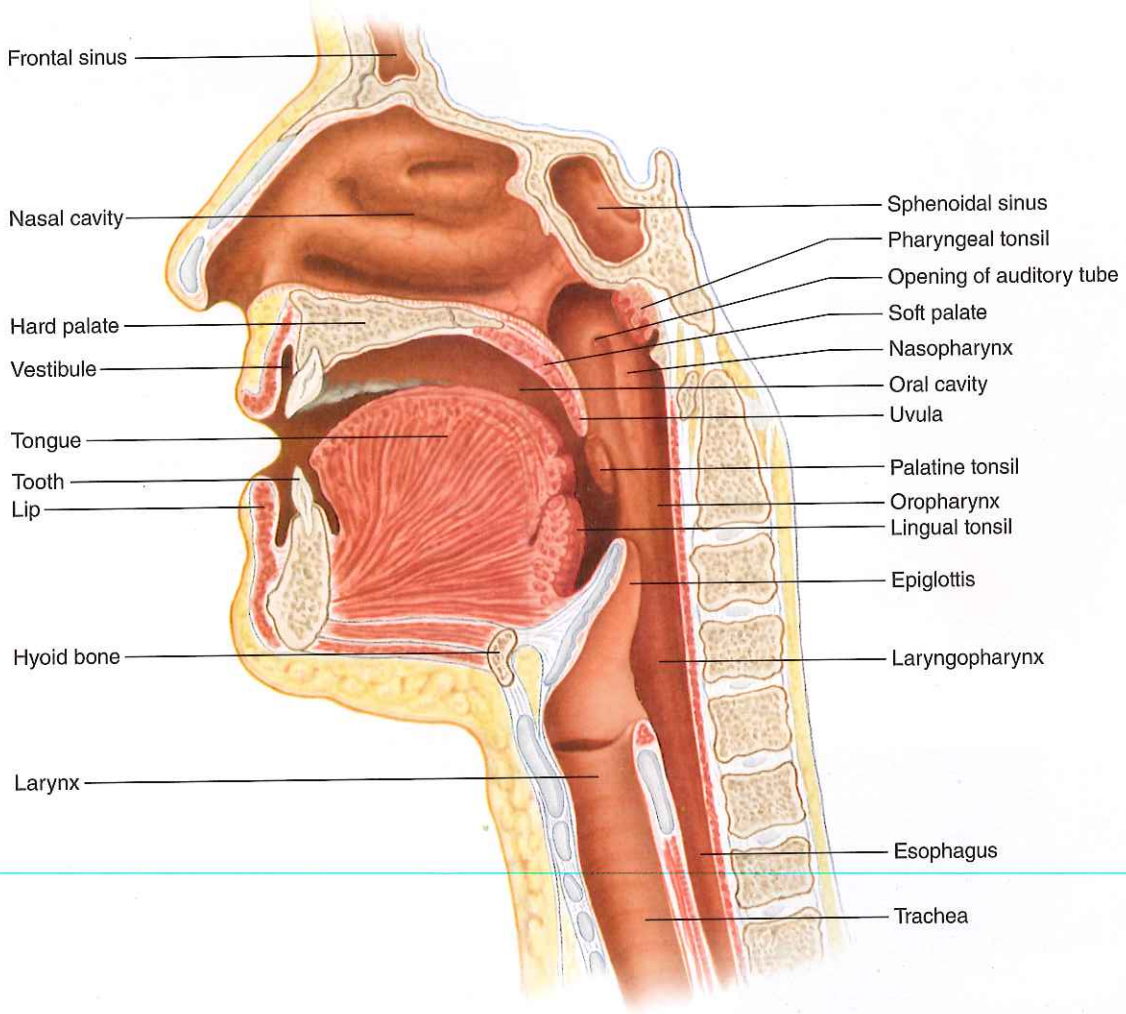


FIGURE 17.7 AP|R Sagittal section of the mouth, nasal cavity, and pharynx.

Teeth

The **teeth** are the hardest structures in the body. They are not considered part of the skeletal system because they have at least two types of proteins that are not found in bone, and their structure is different from that of bone.

Teeth develop in sockets in the alveolar processes of the mandible and maxillae. Teeth are unique structures in that two sets form during development (fig. 17.8). The first set, the *primary teeth* (deciduous teeth), usually erupt through the gums (gingiva) at regular intervals between the ages of six months and two to four years. The ten primary teeth are anchored in each jaw from the midline toward the sides in the following sequence: central incisor, lateral incisor, canine (cuspid), first molar, and second molar.

The primary teeth are usually shed in the same order as they erupted. After their roots are resorbed, the *secondary* (permanent) *teeth* push the primary teeth out of their sockets. This secondary set consists of thirty-two teeth—sixteen in

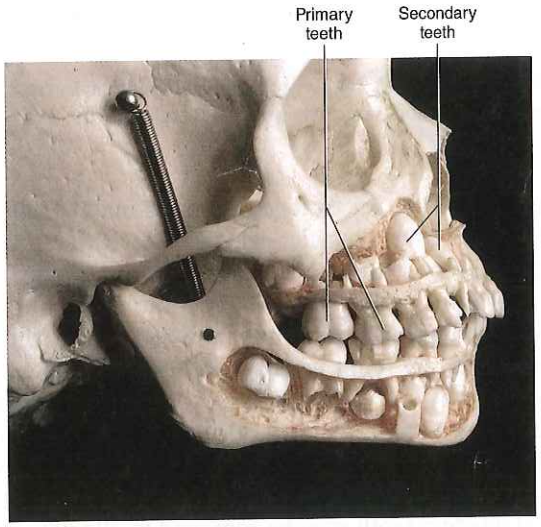


FIGURE 17.8 This partially dissected child's skull reveals primary and developing secondary teeth in the maxilla and mandible.

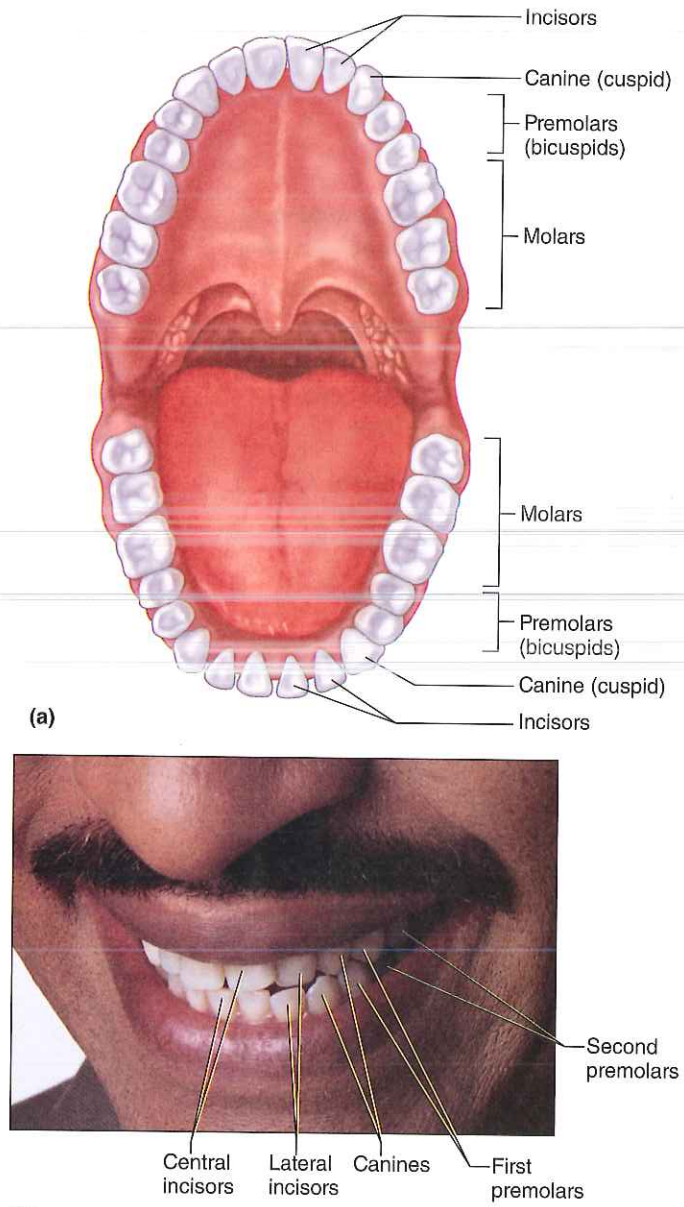


FIGURE 17.9 AP|R Permanent teeth. (*a*) The secondary teeth of the upper and lower jaws. (*b*) Anterior view of the secondary teeth.

TABLE 17.2 | Primary and Secondary Teeth

Primary Teeth (Deciduous)		Secondary Teeth (Permanent)	
Type	*Number*	*Type*	*Number*
Incisor		Incisor	
Central	4	Central	4
Lateral	4	Lateral	4
Canine (cuspid)	4	Canine (cuspid)	4
		Premolar (bicuspid)	
		First	4
		Second	4
Molar		Molar	
First	4	First	4
Second	4	Second	4
		Third	4
Total	20	Total	32

each jaw—and they are arranged from the midline as follows: central incisor, lateral incisor, canine (cuspid), first premolar (bicuspid), second premolar (bicuspid), first molar, second molar, and third molar (fig. 17.9). Table 17.2 summarizes the types and numbers of primary and secondary teeth.

The secondary teeth usually begin to erupt at six years, but the set may not be completed until the third molars emerge between seventeen and twenty-five years. Sometimes these third molars, also called wisdom teeth, become wedged in abnormal positions in the jaws and fail to erupt. Such *impacted* wisdom teeth may be removed to alleviate pain.

The teeth break food into smaller pieces, which begins mechanical digestion. Chewing increases the surface area of the food particles, enabling digestive enzymes to interact more effectively with nutrient molecules.

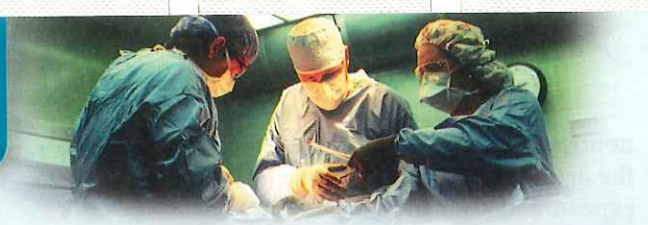
Different teeth are adapted to handle food in different ways. The sharp edges of the chisel-shaped *incisors* bite off large pieces of food. The cone-shaped *canines* grasp and tear food. The flattened surfaces of the *premolars* and *molars* grind food particles.

Each tooth consists of two main parts—the *crown,* which projects beyond the gum, and the *root,* which is anchored to the alveolar process of the jaw. These structures meet at the *neck* of the tooth. Glossy, white *enamel* covers the crown. Enamel mainly consists of calcium salts and is the hardest substance in the body. If abrasive action or injury damages enamel, it is not replaced. Enamel may wear away with age.

The bulk of a tooth beneath the enamel is composed of a living cellular tissue called *dentin,* which is similar to bone but harder. Dentin, in turn, surrounds the tooth's central cavity (pulp cavity), which contains blood vessels, nerves, and connective tissue, collectively called pulp. Blood vessels and nerves reach the central cavity through tubular *root canals,* which extend into the root. Tooth loss is most often associated with diseases of the gums (gingivitis) and the dental pulp (endodontitis).

A thin layer of bonelike material called *cementum,* surrounded by a *periodontal ligament* (periodontal membrane), encloses the root. This ligament, composed of collagen, passes between the cementum and the bone of the alveolar process, firmly attaching the tooth to the jaw. The ligament also contains blood vessels and nerves near the surface of the cementum-covered root (fig. 17.10). Clinical Application 17.1 describes the effect of bacteria on teeth. Table 17.3 summarizes the mouth parts and their functions.

17.1 CLINICAL APPLICATION



Dental Caries

Sticky foods, such as caramel, lodge between the teeth and in the crevices of molars, feeding bacteria such as *Actinomyces, Streptococcus mutans,* and *Lactobacillus.* These microbes metabolize carbohydrates in the food, producing acid by-products that destroy tooth enamel and dentin (fig. 17A). The bacteria also produce sticky substances that hold them in place.

If a person eats candy, for example, but does not brush the teeth soon afterward, the acid-forming bacteria may cause tooth decay, creating a condition called *dental caries.* Unless a dentist cleans and fills the resulting cavity that forms where enamel is destroyed, the damage will spread to the underlying dentin.

Dental caries can be prevented in several ways:

1. Brush and floss teeth regularly.
2. Have regular dental exams and cleanings.
3. Talk with your dentist about receiving a fluoride treatment. Fluoride is added to the water supply in many communities. Fluoride is incorporated into the enamel's chemical structure, strengthening it.
4. The dentist may apply a sealant to children's and adolescents' teeth where crevices might hold onto decay-causing bacteria. The sealant is a coating that keeps acids from eating away at tooth enamel. ■

FIGURE 17A *Actinomyces* bacteria (falsely colored) clinging to teeth release acids that decay tooth enamel (1,250×).

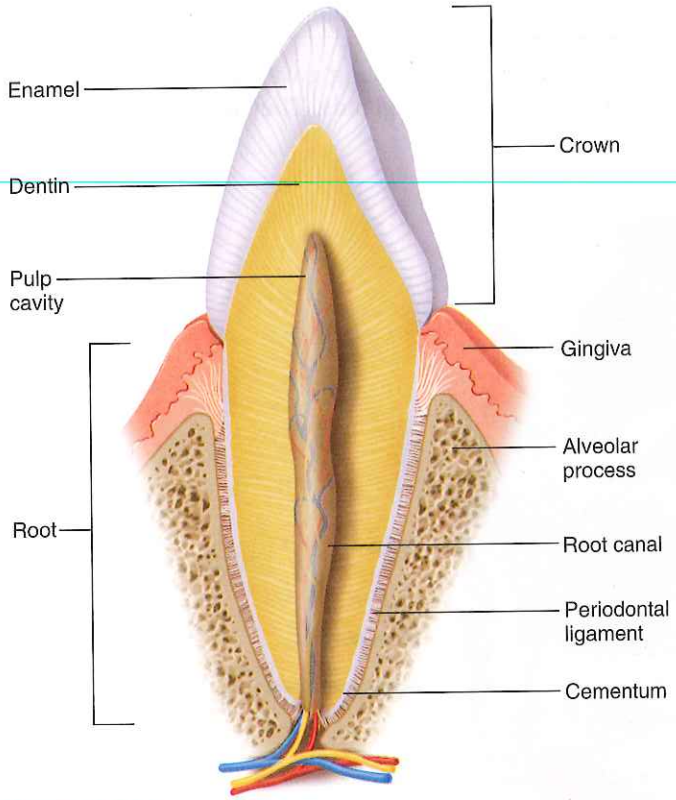


FIGURE 17.10 A section of a tooth.

Extracted primary and wisdom teeth may one day provide stem cells that can be used to regenerate tooth roots and supporting periodontal ligaments or even replace teeth. The stem cells are in the pulp of the developing tooth.

TABLE 17.3 | Mouth Parts and Their Functions in Digestion

Part	Location	Function
Cheeks	Form lateral walls of mouth	Hold food in mouth; muscles chew food
Lips	Surround mouth opening	Contain sensory receptors used to judge characteristics of foods
Tongue	Floor of mouth	Mixes food with saliva; moves food toward pharynx; contains taste receptors
Palate	Forms roof of mouth	Holds food in mouth; directs food to pharynx
Teeth	In sockets of mandibular and maxillary bones	Break food particles into smaller pieces; help mix food with saliva during chewing

PRACTICE

9 How do primary teeth differ from secondary teeth?

10 How are types of teeth adapted to provide specialized functions?

11 Describe the structure of a tooth.

12 Explain how a tooth is attached to the bone of the jaw.

17.4 SALIVARY GLANDS

The **salivary** (sal'ĭ-ver-e) **glands** secrete *saliva.* This fluid moistens food particles, helps bind them, and begins the chemical digestion of carbohydrates. Saliva is also a solvent, dissolving foods so that they can be tasted, and it helps cleanse the mouth and teeth. Bicarbonate ions (HCO_3^-) in saliva help buffer acid in the mouth, keeping the pH near

neutral, between 6.5 and 7.5. This is a favorable range for the action of the salivary enzyme and protects the teeth from exposure to acids in foods.

Many minor salivary glands are scattered throughout the mucosa of the tongue, palate, and cheeks. They continuously secrete fluid, moistening the lining of the mouth. The three pairs of major salivary glands are the parotid glands, the submandibular glands, and the sublingual glands.

Salivary Secretions

The different salivary glands have varying proportions of two types of secretory cells, *serous cells* and *mucous cells*. Serous cells produce a watery fluid that contains a digestive enzyme, **salivary amylase** (am′ĭ-lās). This enzyme splits starch and glycogen molecules into disaccharides, starting the chemical digestion of carbohydrates. Mucous cells secrete a thick liquid called **mucus,** which binds food particles and acts as a lubricant during swallowing.

Branches of both sympathetic and parasympathetic nerves innervate the salivary glands, as they do other digestive structures. Impulses arriving on sympathetic fibers stimulate the gland cells to secrete a small volume of viscous saliva. Parasympathetic impulses, on the other hand, elicit the secretion of a large volume of watery saliva. Such parasympathetic impulses are activated reflexly when a person sees, smells, tastes, or even thinks about pleasant foods. Conversely, if food looks, smells, or tastes unpleasant, parasympathetic activity is inhibited. Less saliva is produced, and swallowing may become difficult.

Major Salivary Glands

The **parotid** (pah-rot′id) **glands** are the largest of the major salivary glands. Each gland lies anterior to and somewhat inferior to each ear, between the skin of the cheek and the masseter muscle. A *parotid duct* (Stensen's duct) passes from the gland inward through the buccinator muscle, entering the mouth just opposite the upper second molar on either side of the jaw. The parotid glands secrete a clear, watery fluid rich in salivary amylase because their secretory cells are primarily serous cells (figs. 17.11 and 17.12*a*).

The **submandibular** (sub″man-dib′u-lar) **glands** are in the floor of the mouth on the inside surface of the lower jaw. The secretory cells of these glands are about equally serous and mucous. Consequently, the submandibular glands secrete a more viscous fluid than the parotid glands (see figs. 17.11 and 17.12*b*). The ducts of the submandibular glands (Wharton's ducts) open inferior to the tongue, near the lingual frenulum.

The **sublingual** (sub-ling′gwal) **glands** are the smallest of the major salivary glands. They are on the floor of the

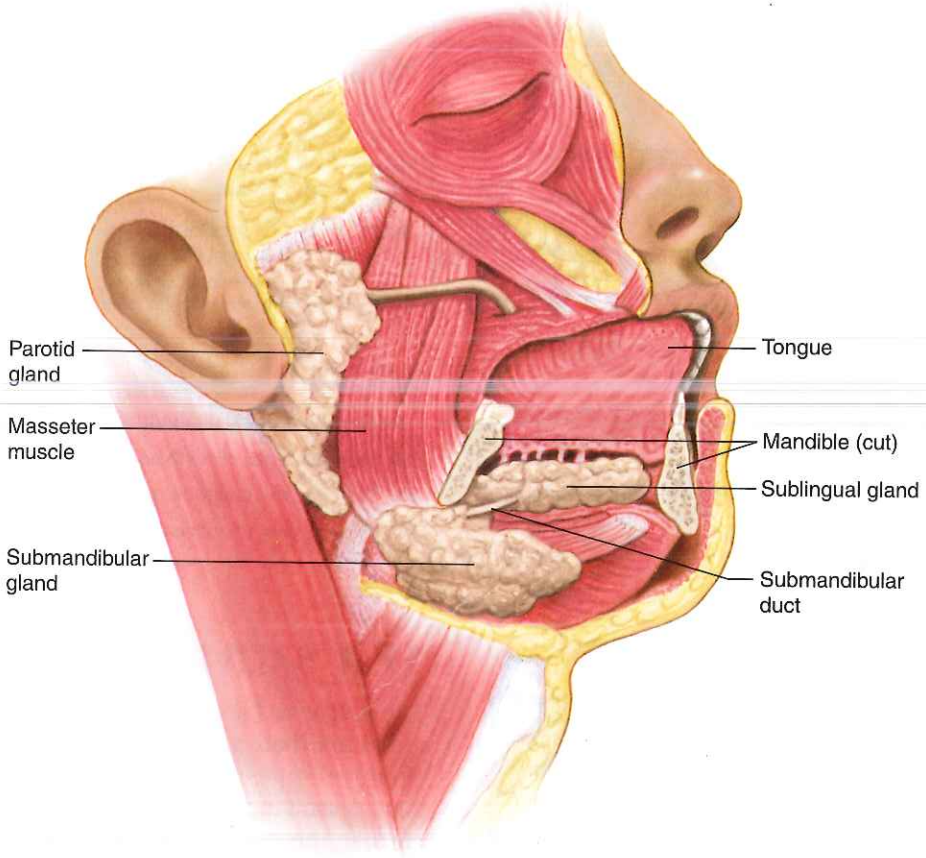


FIGURE 17.11 AP|R Locations of the major salivary glands.

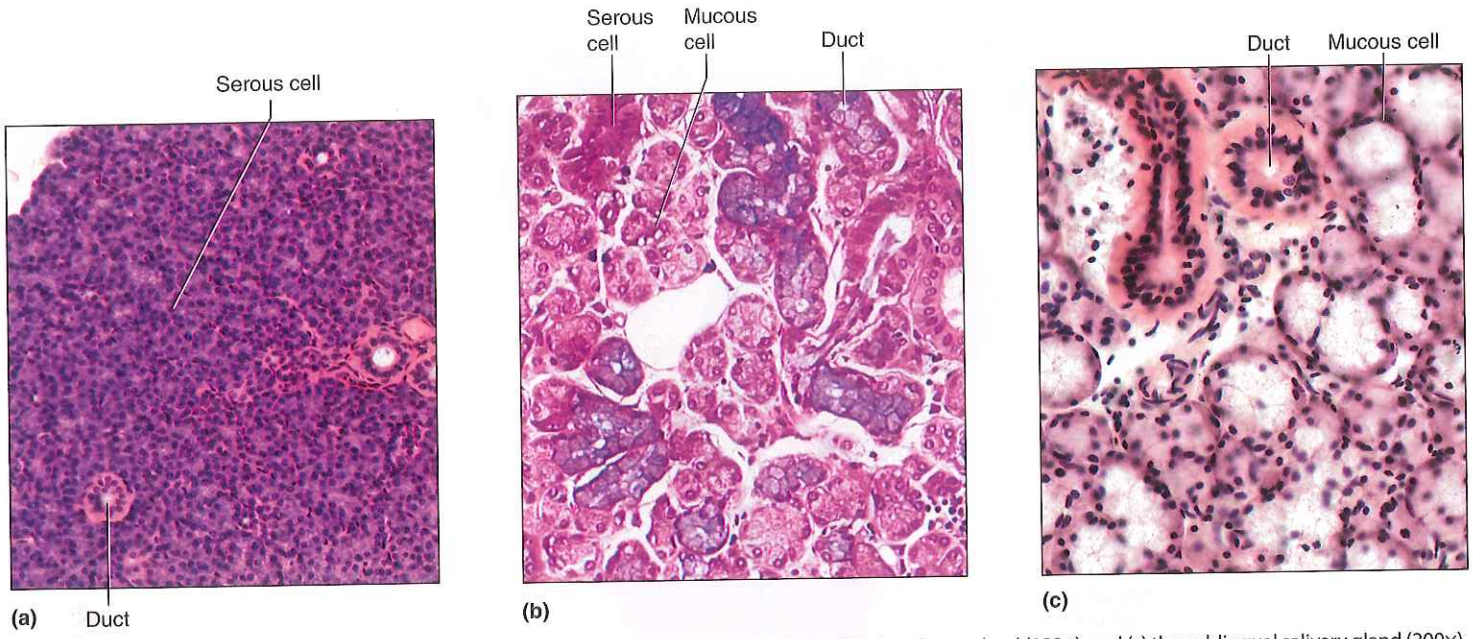


FIGURE 17.12 Light micrographs of (*a*) the parotid salivary gland (100×), (*b*) the submandibular salivary gland (180×), and (*c*) the sublingual salivary gland (200×).

mouth inferior to the tongue. Because the secretory cells of the sublingual glands are primarily the mucous type, their secretions, which enter the mouth through many separate ducts (Rivinus's ducts), are thick and stringy (see figs. 17.11 and 17.12*c*). Table 17.4 summarizes the characteristics of the major salivary glands.

PRACTICE

13 What is the function of saliva?

14 What stimulates the salivary glands to secrete saliva?

15 Where are the major salivary glands located?

TABLE 17.4 | The Major Salivary Glands

Gland	Location	Duct	Type of Secretion
Parotid glands	Anterior to and somewhat inferior to the ears between the skin of the cheeks and the masseter muscles	Parotid ducts pass through the buccinator muscles and enter the mouth opposite the upper second molars	Clear, watery serous fluid, rich in salivary amylase
Submandibular glands	In the floor of the mouth on the inside surface of the mandible	Ducts open inferior to the tongue near the frenulum	Some serous fluid with some mucus; more viscous than parotid secretion
Sublingual glands	In the floor of the mouth inferior to the tongue	Many separate ducts	Primarily thick, stringy mucus

17.5 PHARYNX AND ESOPHAGUS

The pharynx is a cavity posterior to the mouth from which the tubular esophagus leads to the stomach. The pharynx and the esophagus do not digest food, but both are important passageways, and their muscular walls function in swallowing.

Structure of the Pharynx

The **pharynx** (far'ingks) connects the nasal and oral cavities with the larynx and esophagus (see fig. 17.7). It can be divided into the following parts:

1. The **nasopharynx** (na"zo-far'ingks) is superior to the soft palate. It communicates with the nasal cavity and provides a passageway for air during breathing. The auditory tubes, which connect the pharynx with the middle ears, open through the walls of the nasopharynx (see chapter 12, p. 458).
2. The **oropharynx** (o"ro-far'ingks) is posterior to the mouth. It is posterior to the soft palate and inferior to the nasopharynx, projecting downward to the upper border of the epiglottis. This portion is a passageway for food moving downward from the mouth and for air moving to and from the nasal cavity.
3. The **laryngopharynx** (lah-ring"go-far'inks) is just inferior to the oropharynx. It extends from the upper border of the epiglottis downward to the lower border of the cricoid cartilage of the larynx and is a passageway to the esophagus.

The muscles in the walls of the pharynx form inner circular and outer longitudinal groups (fig. 17.13). The circular muscles, called *constrictor muscles*, pull the walls inward during swallowing. The *superior constrictor muscles*,

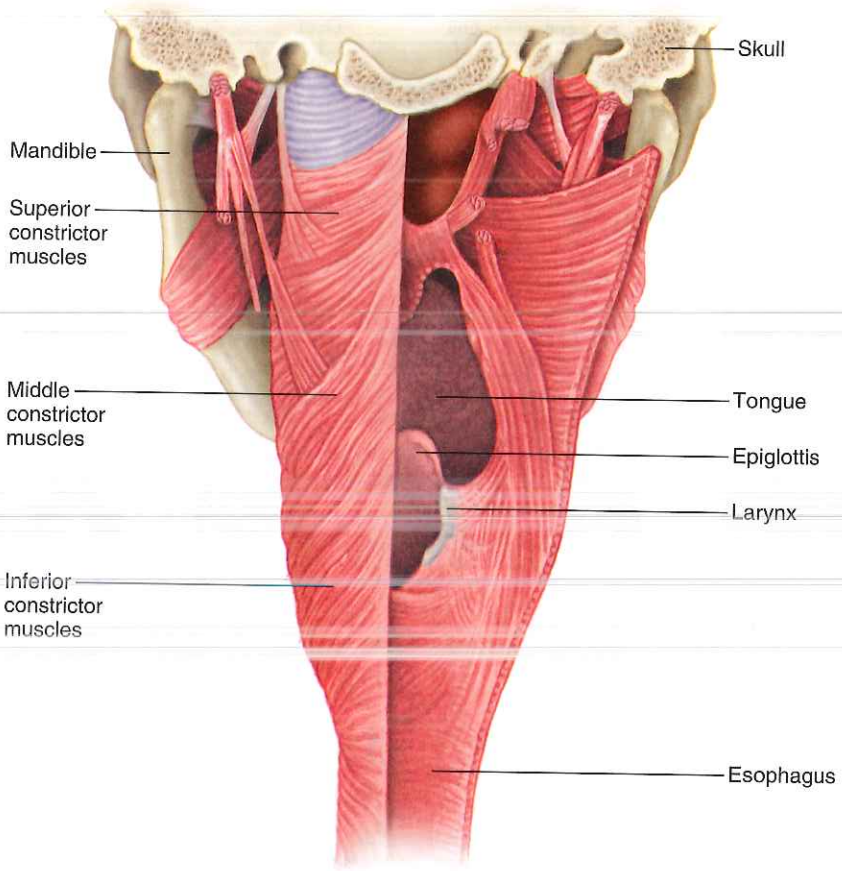


FIGURE 17.13 Muscles of the pharyngeal wall, posterior view.

attached to bony processes of the skull and mandible, curve around the upper part of the pharynx. The *middle constrictor muscles* arise from projections on the hyoid bone and fan around the middle of the pharynx. The *inferior constrictor muscles* originate from cartilage of the larynx and pass around the lower portion of the pharyngeal cavity. Some of the lower inferior constrictor muscle fibers contract most of the time, which prevents air from entering the esophagus during breathing.

The pharyngeal muscles are skeletal muscles, but they are under voluntary control only in the sense that swallowing (deglutition) can be voluntarily initiated. Complex reflexes control the precise actions of these muscles during swallowing.

Swallowing Mechanism

Swallowing can be divided into three stages. In the first stage, which is voluntary, food is chewed and mixed with saliva. Then, the tongue rolls this mixture into a mass, or **bolus,** and forces it into the oropharynx.

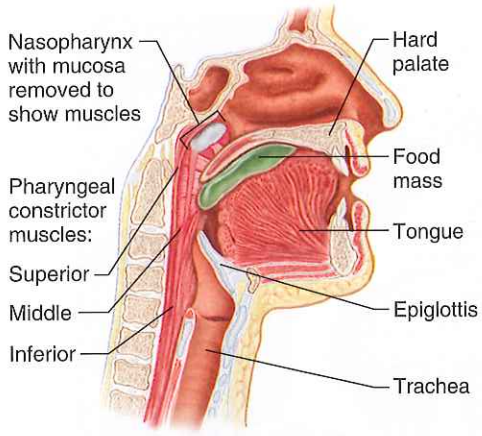
The second stage of swallowing begins as food reaches the oropharynx and stimulates sensory receptors around the pharyngeal opening. This triggers the swallowing reflex, illustrated in figure 17.14, which includes the following actions:

1. The soft palate (including the uvula) raises, preventing food from entering the nasal cavity.
2. The hyoid bone and the larynx are elevated. A flaplike structure attached to the larynx, called the *epiglottis* (ep″ĭ-glot′is), closes off the top of the trachea so that food is less likely to enter the trachea.
3. The tongue is pressed against the soft palate and uvula, sealing off the oral cavity from the nasal cavity.
4. The longitudinal muscles in the pharyngeal wall contract, pulling the pharynx upward toward the food.
5. The lower portion of the inferior constrictor muscles relaxes, opening the esophagus.
6. The superior constrictor muscles contract, stimulating a peristaltic wave to begin in other pharyngeal muscles. This wave forces the food into the esophagus.

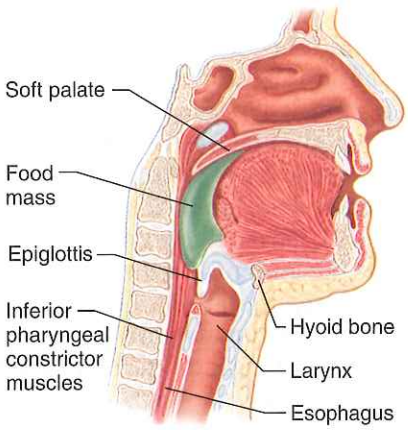
The swallowing reflex momentarily inhibits breathing. Then, during the third stage of swallowing, peristalsis transports the food in the esophagus to the stomach.

Esophagus

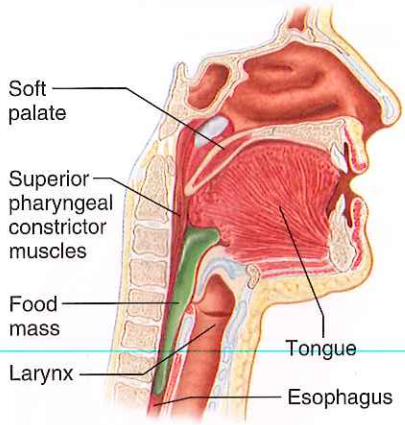
The **esophagus** (ĕ-sof′ah-gus) is a straight, collapsible tube about 25 centimeters long. It provides a passageway for food, and its muscular wall propels food from the pharynx to



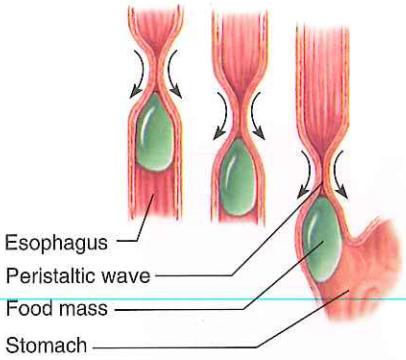
(a) The tongue forces food into the pharynx.



(b) The soft palate, hyoid bone, and larynx are raised, the tongue is pressed against the palate, the epiglottis closes, and the inferior constrictor muscles relax so that the esophagus opens.



(c) Superior constrictor muscles contract and force food into the esophagus.



(d) Peristaltic waves move food through the esophagus to the stomach.

FIGURE 17.14 Steps in the swallowing reflex. (The mucosa in (*a*), (*b*), and (*c*) has been removed to reveal the underlying muscles.)

the stomach. The esophagus begins at the base of the laryngopharynx and descends through the thorax posterior to the trachea, passing through the mediastinum. It penetrates the diaphragm through an opening, the *esophageal hiatus* (ĕ-sof″ah-je′al hi-a′tus), and is continuous with the stomach on the abdominal side of the diaphragm (fig. 17.15 and reference plates 17 and 23, pp. 52 and 55).

In a *hiatal hernia,* part of the stomach protrudes through a weakened area of the diaphragm, through the esophageal hiatus and into the thorax. Regurgitation (reflux) of gastric juice into the esophagus becomes more likely. This may inflame the esophageal mucosa, causing "heartburn," difficulty in swallowing, or ulceration and blood loss. In response to the destructive action of gastric juice, columnar epithelium may replace the squamous epithelium that normally lines the esophagus (see chapter 5, page 156). This condition, called *Barrett's esophagus,* increases the risk of developing esophageal cancer.

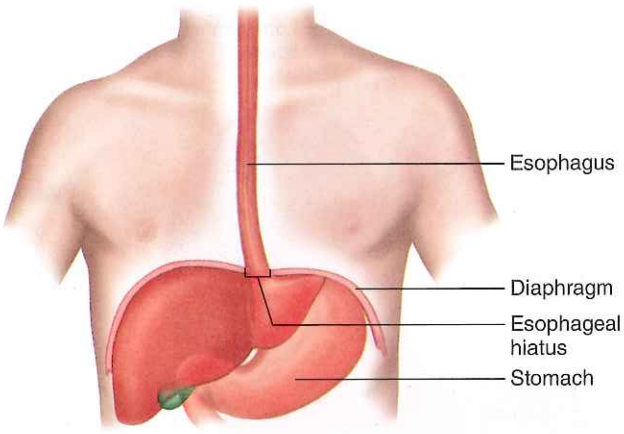


FIGURE 17.15 AP|R The esophagus is a passageway between the laryngopharynx and the stomach.

Mucous glands are scattered throughout the submucosa of the esophagus (fig. 17.16). Their secretions moisten and lubricate the inner lining of the tube.

Just superior to the point where the esophagus joins the stomach, some of the circular muscle fibers have increased sympathetic muscle tone, forming the **lower esophageal sphincter** (loh'er ĕ-sof"ah-je'al sfingk'ter), or cardiac sphincter (fig. 17.17). These fibers usually remain contracted, and they close the entrance to the stomach. In this way, they help prevent regurgitation of the stomach contents into the esophagus. When peristaltic waves reach the stomach, the muscle fibers that guard its entrance temporarily relax and allow the swallowed food to enter.

PRACTICE

16 Describe the regions of the pharynx.

17 List the major events of swallowing.

18 What is the function of the esophagus?

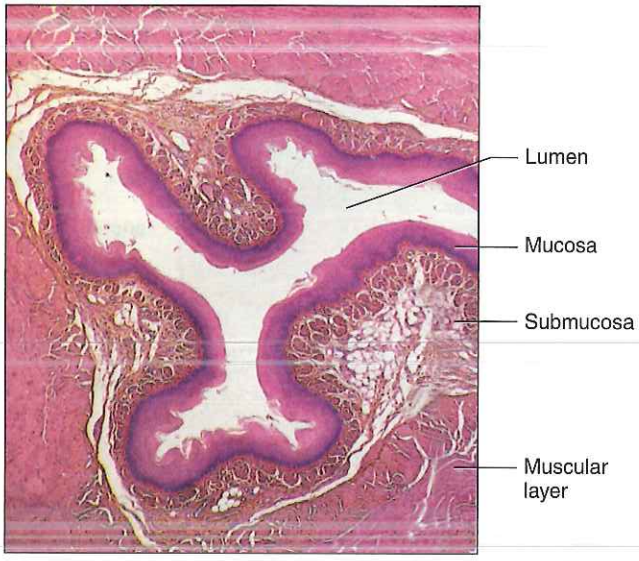
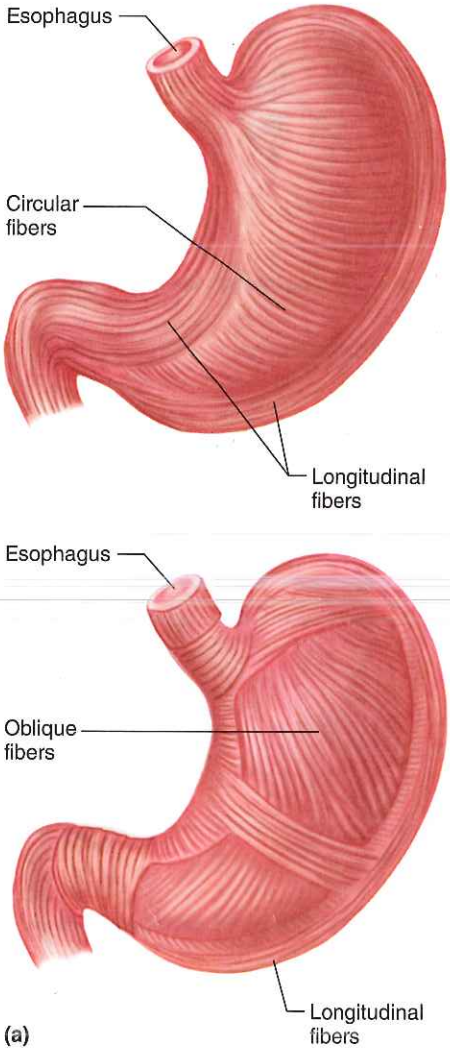


FIGURE 17.16 AP|R This cross section of the esophagus shows its muscular wall (10×).



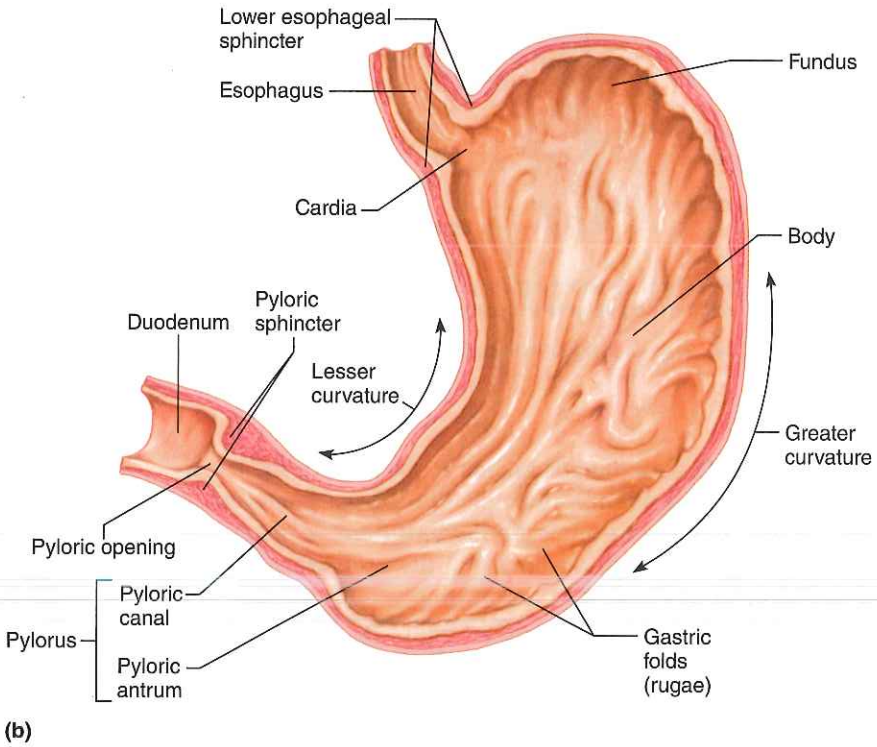


FIGURE 17.17 AP|R Stomach. (*a*) Some parts of the stomach have three layers of muscle fibers. (*b*) Major regions of the stomach and its associated structures.

17.6 STOMACH AP|R

The **stomach** (stum'ak) is a J-shaped, pouchlike organ, about 25–30 centimeters long, which hangs inferior to the diaphragm in the upper-left portion of the abdominal cavity (see figs. 17.1 and 17.15; reference plates 4 and 5, pp. 42–43). The stomach has a capacity of about one liter or more. Its inner lining has thick gastric folds (rugae) of the mucosal and submucosal layers that unfold when the wall is distended. The stomach receives food from the esophagus, mixes it with gastric juice, initiates the digestion of proteins, carries on limited absorption, and moves food into the small intestine.

In addition to the two layers of smooth muscle—an inner circular layer and an outer longitudinal layer—which are also in other regions of the alimentary canal, some parts of the stomach have an additional inner layer of oblique fibers, which strengthen the stomach wall and help the mixing and churning. This third innermost muscular layer is most highly developed near the opening of the esophagus and in the body of the stomach (fig. 17.17).

Parts of the Stomach

The stomach, shown in figure 17.17 and figure 17.18, can be divided into the cardia, fundus, body, and pylorus. The *cardia* is a small area near the esophageal opening. The *fundus*, which balloons superior to the cardia, is a temporary storage area and sometimes fills with swallowed air. This produces a gastric air bubble, which may be used as a landmark on a radiograph of the abdomen. The dilated *body*, which is the main part of the stomach, lies between the fundus and pylorus. The *pyloric antrum* is a funnel-shaped portion that narrows and becomes the *pyloric canal* as it approaches the small intestine.

At the end of the pyloric canal, the circular layer of fibers in its muscular wall thickens, forming a powerful muscle, the **pyloric sphincter.** This muscle is a valve that controls gastric emptying.

Hypertrophic pyloric stenosis is a birth defect in which muscle overgrowth blocks the pyloric canal. The newborn vomits, with increasing force. To diagnose the condition, a radiograph is taken of the area after the infant drinks formula containing a radiopaque barium compound. Surgical splitting of the muscle blocking the passageway from stomach to small intestine is necessary to enable the infant to eat normally. Pyloric stenosis can occur later in life as a result of ulcers or cancer.

Gastric Secretions

The mucous membrane that forms the inner lining of the stomach is thick. Its surface is studded with many small openings, called *gastric pits*, that are located at the ends of tubular **gastric glands** (oxyntic glands) (fig. 17.19). Although their structure and the composition of their secretions vary in different parts of the stomach, gastric glands

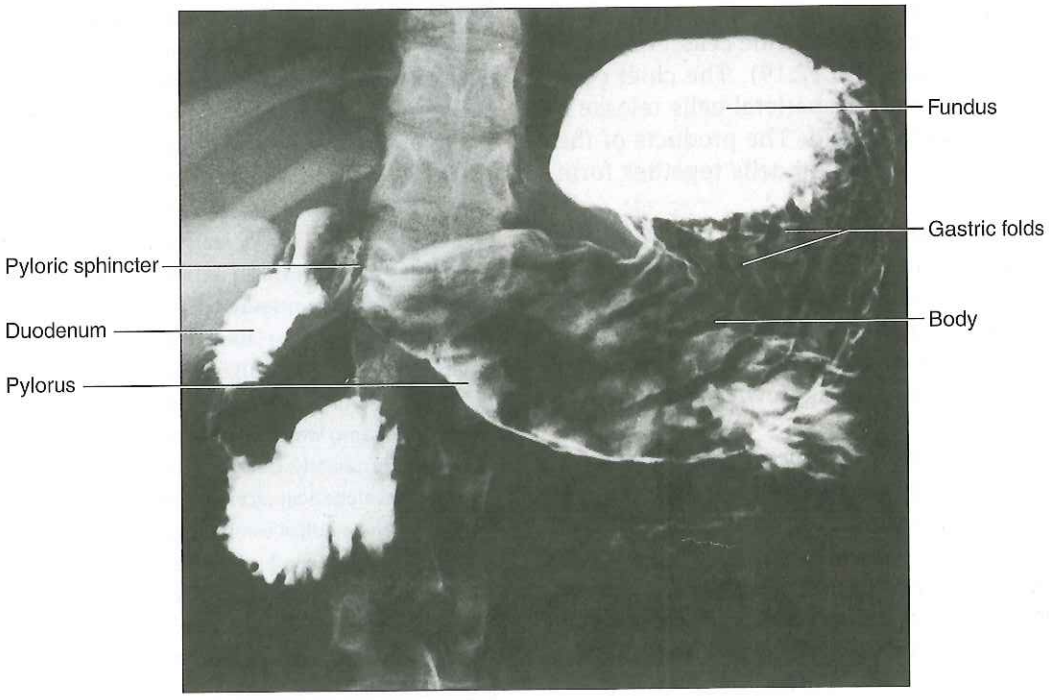


FIGURE 17.18 Radiograph of a stomach. (*Note:* A radiopaque compound the patient swallowed appears white in the radiograph.)

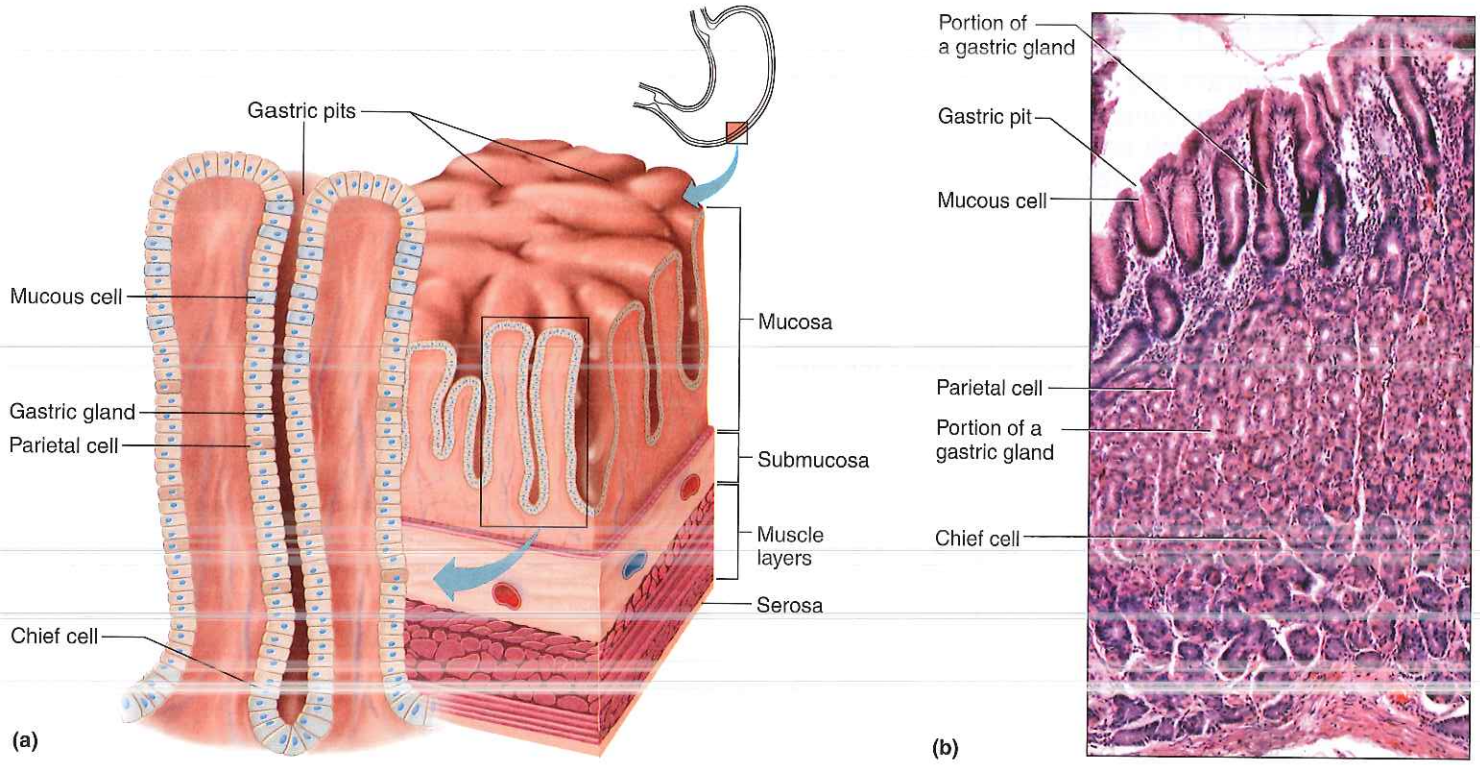


FIGURE 17.19 AP|R Lining of the stomach. (*a*) Gastric glands include mucous cells, parietal cells, and chief cells. The mucosa of the stomach is studded with gastric pits that are the openings of the gastric glands. (*b*) A light micrograph of cells associated with the gastric glands (60×).

generally contain three types of secretory cells. One type, the *mucous cell*, found in the necks of the glands near the openings of the gastric pits, secretes mucus. The other types, *chief cells* (peptic cells) and *parietal cells* (oxyntic cells), reside in the deeper parts of the glands (fig. 17.19). The chief cells secrete digestive enzymes, and the parietal cells release a solution containing hydrochloric acid. The products of the mucous cells, chief cells, and parietal cells together form **gastric juice** (gas'trik jo͞os).

Pepsin is by far the most important of the digestive enzymes in gastric juice. The chief cells secrete pepsin as an inactive, nonerosive enzyme precursor called **pepsinogen.** When pepsinogen contacts the hydrochloric acid from the parietal cells, however, it breaks down rapidly, forming pepsin. Pepsin, in turn, can also break down pepsinogen to release more pepsin.

Pepsin begins the digestion of nearly all types of dietary protein into polypeptides. This enzyme is most active in an acidic environment, which is provided by the hydrochloric acid in gastric juice.

Gastric juice contains small quantities of a fat-splitting enzyme, *gastric lipase.* However, its action is weak due in part to the low pH of gastric juice. Gastric lipase acts mainly on butterfat.

Much of what we know about the stomach's functioning comes from a French-Canadian explorer, Alexis St. Martin, who in 1822 accidentally shot himself in the abdomen. His extensive injuries eventually healed, but a hole, called a fistula, was left, allowing observers to look at his stomach in action. A U.S. Army surgeon, William Beaumont, spent eight years watching food digesting in the stomach, and noted how the stomach lining changed in the process.

In 1984, our knowledge of digestive function expanded when medical resident Barry Marshall at Royal Perth Hospital in western Australia performed a daring experiment. His mentor, J. Robin Warren, had hypothesized that a bacterial infection causes gastritis (inflammation of the stomach lining) and peptic ulcers (sores in the lining of the esophagus, stomach, or small intestine). At the time, these conditions were attributed to poor diet and stress. Marshall drank "swamp water"—billions of bacteria. He developed gastritis, which, fortunately, cleared up. A colleague who repeated the experiment developed an ulcer and required antibiotics. After a decade of debate, the medical community finally concurred that the bacterium *Helicobacter pylori,* which thrives under acidic conditions, causes many cases of gastritis and peptic ulcers. A short course of antibiotics and acid-lowering drugs has replaced lifelong treatments. Marshall and Warren were awarded a Nobel Prize in 2005 for their discovery.

The mucous cells of the gastric glands *(mucous neck cells)* and the mucous cells, associated with the stomach's inner surface, release a viscous, alkaline secretion that coats the inside of the stomach wall. This coating is especially important because pepsin can digest the proteins of stomach tissues, as well as those in foods. The coating normally prevents the stomach from digesting itself.

Another component of gastric juice is **intrinsic factor** (in-trin'sik fak'tor). The parietal cells of the gastric glands secrete intrinsic factor, required for vitamin B_{12} absorption from the small intestine. Table 17.5 summarizes the components of gastric juice.

PRACTICE

19 Where is the stomach located?

20 What are the secretions of the chief cells and parietal cells?

21 Why doesn't the stomach digest itself?

Regulation of Gastric Secretions

Gastric juice is produced continuously, but the rate varies considerably and is controlled both neurally and hormonally. In the gastric glands, specialized cells closely associated with the parietal cells secrete the hormone *somatostatin,* which inhibits acid secretion. However, acetylcholine (ACh) released from nerve endings in response to parasympathetic impulses arriving on the vagus nerves suppresses the secretion of somatostatin and stimulates the gastric glands to secrete abundant gastric juice, which is rich in hydrochloric acid and pepsinogen. These parasympathetic impulses also stimulate certain stomach cells, mainly in the pyloric region, to release a peptide hormone called **gastrin,** which increases the secretory activity of gastric glands (fig. 17.20). Furthermore, parasympathetic impulses and gastrin promote release of *histamine* from gastric mucosal cells, which, in turn, stimulates additional gastric secretion.

Gastrin stimulates cell growth in the mucosa of the stomach and intestines, except where gastrin is produced. This cell growth helps replace mucosal cells damaged by normal stomach function, disease, or medical treatments.

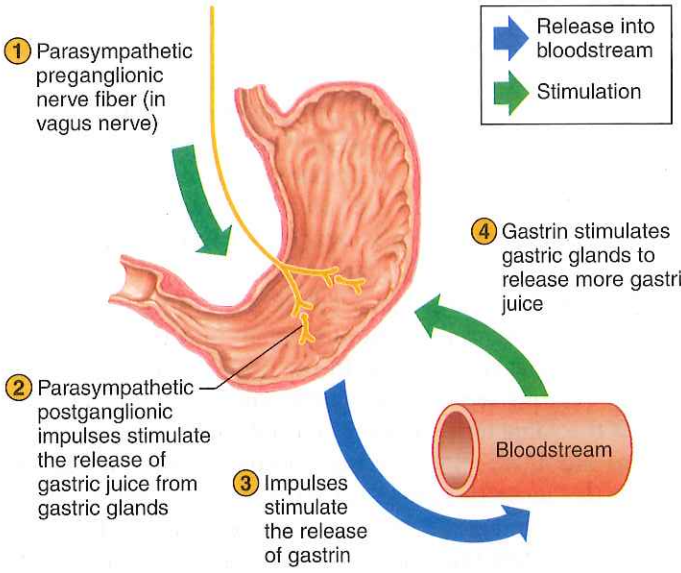


FIGURE 17.20 The secretion of gastric juice is regulated in part by parasympathetic impulses that stimulate the release of gastric juice and gastrin.

Gastric secretion occurs in three stages—the cephalic, gastric, and intestinal phases. The *cephalic phase* begins before food reaches the stomach and possibly even before eating. In this stage, parasympathetic reflexes operating through the vagus nerves stimulate gastric secretion at the taste, smell, sight, or thought of food. The greater the hunger, the greater the gastric secretion.

The *gastric phase* of gastric secretion, which accounts for most of the secretory activity, starts when food enters the stomach. The presence of food and the distension of the stomach wall trigger the stomach to release gastrin, which stimulates production of more gastric juice.

As food enters the stomach and mixes with gastric juice, the pH of the contents rises, which enhances gastrin secretion. Consequently, the pH of the stomach contents drops. As the pH approaches 3.0, secretion of gastrin is inhibited. When the pH reaches 1.5, gastrin secretion ceases.

For the stomach to secrete hydrochloric acid, hydrogen ions are actively transported into the stomach. Negatively charged chloride ions, attracted by the positively charged hydrogen ions, move from the blood into the stomach. An

TABLE 17.5 | Major Components of Gastric Juice AP|R

Component	Source	Function
Pepsinogen	Chief cells of the gastric glands	Inactive form of pepsin
Pepsin	Formed from pepsinogen in the presence of hydrochloric acid	A protein-splitting enzyme that digests nearly all types of dietary protein into polypeptides
Hydrochloric acid	Parietal cells of the gastric glands	Provides the acid environment needed for production and action of pepsin
Mucus	Mucous cells	Provides a viscous, alkaline protective layer on the stomach's inner surface
Intrinsic factor	Parietal cells of the gastric glands	Aids in vitamin B_{12} absorption in the small intestine

equivalent number of alkaline bicarbonate ions are released into the blood. Following a meal, the blood concentration of bicarbonate ions increases, and the urine excretes excess bicarbonate ions. This phenomenon is called the *alkaline tide*.

The *intestinal phase* of gastric secretion begins when food leaves the stomach and enters the small intestine. When food first contacts the intestinal wall, it stimulates intestinal cells to release a hormone, *intestinal gastrin*, that briefly enhances gastric gland secretion.

The intestinal phase primarily inhibits gastric juice secretion. As more food moves into the small intestine, a sympathetic reflex triggered by acid in the upper part of the small intestine inhibits secretion of gastric juice from the stomach wall. At the same time, proteins and fats in this region of the intestine stimulate release of the peptide hormone **cholecystokinin** (ko″le-sis″to-ki′nin) (**CCK**) from the intestinal wall, which decreases gastric motility. Similarly, fats in the small intestine stimulate intestinal cells to release *intestinal somatostatin*, which inhibits release of gastric juice. Overall, these actions decrease gastric secretion and motility as the small intestine fills with food. Table 17.6 summarizes the phases of gastric secretion.

TABLE 17.6 | Phases of Gastric Secretion AP|R

Phase	Action
Cephalic phase	The sight, taste, smell, or thought of food triggers parasympathetic reflexes. Gastric juice is secreted in response.
Gastric phase	Food in stomach chemically and mechanically stimulates release of gastrin, which, in turn, stimulates secretion of gastric juice; reflex responses also stimulate gastric juice secretion.
Intestinal phase	As food enters the small intestine, it stimulates intestinal cells to release intestinal gastrin, which, in turn, briefly promotes the secretion of gastric juice from the stomach wall. This phase primarily inhibits gastric juice secretion.

PRACTICE

22 What controls gastric juice secretion?

23 Distinguish among the cephalic, gastric, and intestinal phases of gastric secretion.

24 What is the function of cholecystokinin?

Gastric Absorption

Gastric enzymes begin breaking down proteins, but the stomach wall is not well-adapted to absorb digestive products. The stomach absorbs only small volumes of water and certain salts, as well as certain lipid-soluble drugs. Most nutrients are absorbed in the small intestine. Alcohol, which is not a nutrient, is absorbed both in the small intestine and stomach.

Mixing and Emptying Actions

Food stretches the smooth muscles of the stomach wall. The stomach may enlarge, but its muscles maintain their tone, and internal pressure of the stomach normally is unchanged. When a person eats more than the stomach can comfortably hold, the internal pressure may rise enough to stimulate pain receptors. The result is a stomachache. Clinical Application 17.2 discusses this common problem along with its associated indigestion.

Following a meal, the mixing movements of the stomach wall aid in producing a semifluid paste of food particles and gastric juice called **chyme** (kīm). Peristaltic waves push the chyme toward the pylorus of the stomach, and as chyme accumulates near the pyloric sphincter, this muscle begins to relax. Stomach contractions push chyme a little (5–15 milliliters) at a time into the small intestine. These stomach contraction waves push most of the chyme backward into the stomach, mixing it further. The lower esophageal sphincter prevents reflux of stomach contents into the esophagus. Figure 17.21 illustrates this process.

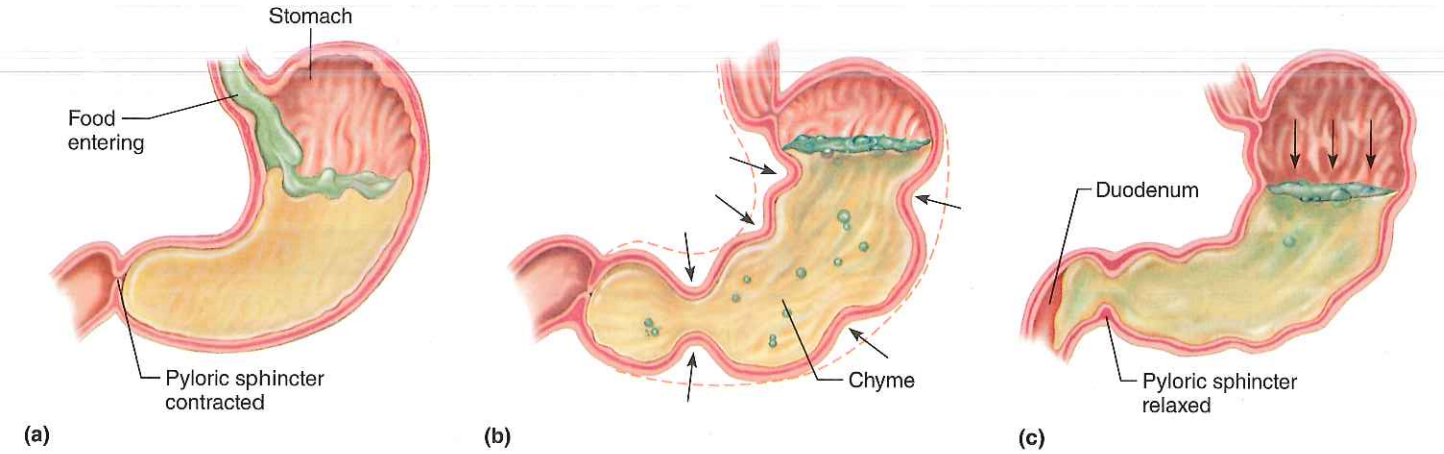


FIGURE 17.21 Stomach movements. (*a*) As the stomach fills, its muscular wall stretches, but the pyloric sphincter remains closed. (*b*) Mixing movements combine food and gastric juice, creating chyme. (*c*) Peristaltic waves move the chyme toward the pyloric sphincter, which relaxes and admits some chyme into the duodenum.

17.2 CLINICAL APPLICATION

A Common Problem: Stomachache

If a person eats large amounts of food too quickly, perhaps because it can take up to 20 minutes for the brain's hypothalamus to sense that the stomach is full, a stomachache can result. A feeling of fullness becomes abdominal pain, then heartburn, as stomach contents back up into the esophagus.

An antacid product may provide temporary relief from a stomachache by quickly raising the pH of the stomach contents. Most antacids include a compound containing sodium, calcium, magnesium, or aluminum. Some products also contain simethicone, which breaks up gas bubbles in the digestive tract.

Avoiding acid indigestion and heartburn is a more healthful approach than gorging and then reaching for medication. Some tips:

- Avoid large meals. The more food, the more acid the stomach produces.
- Eat slowly so that stomach acid secretion is more gradual.
- Do not lie down immediately after eating, being upright enables gravity to help food move along the alimentary canal.
- If prone to indigestion or heartburn, avoid caffeine, which increases stomach acid secretion.
- Cigarettes and alcohol irritate the stomach lining and relax the lower esophageal sphincter. This makes it easier for food to return to the esophagus, causing heartburn. ■

The rate at which the stomach empties depends on the fluidity of the chyme and the type of food. Liquids usually pass through the stomach rapidly, but solids remain until they are well mixed with gastric juice. Fatty foods may remain in the stomach three to six hours; foods high in proteins move through more quickly; carbohydrates usually pass through more rapidly than either fats or proteins.

As chyme fills the duodenum, internal pressure on the organ increases, stretching the intestinal wall. These actions stimulate sensory receptors in the wall, triggering an **enterogastric reflex** (en-ter-o-gas'trik re'fleks). The name of this reflex, like those of other digestive reflexes, describes the origin and termination of reflex impulses. That is, the enterogastric reflex begins in the small intestine *(entero)* and ends in the stomach *(gastro)*. As a result of the enterogastric reflex, fewer parasympathetic impulses arrive at the stomach, decreasing peristalsis, and intestinal filling slows (fig. 17.22). If chyme entering the intestine is fatty, the intestinal wall releases the hormone cholecystokinin, which further inhibits peristalsis.

Vomiting results from a complex reflex that empties the stomach in the reverse of the normal direction. Irritation or distension in the stomach or intestines can trigger vomiting. Sensory impulses travel from the site of stimulation to the *vomiting center* in the medulla oblongata, and motor responses follow. These include taking a deep breath, raising the soft palate and thus closing the nasal cavity, closing the opening to the trachea (glottis), relaxing the circular muscle fibers at the base of the esophagus, contracting the diaphragm so it presses downward over the stomach, and contracting the abdominal wall muscles to increase pressure inside the abdominal cavity. These motor responses squeeze the stomach from all sides, forcing its contents upward and out through the esophagus, pharynx, and mouth.

Several types of stimuli can promote activity in the vomiting center. These include certain drugs (emetics); toxins in contaminated foods; rapid changes in body motion; and sensory impulses from the labyrinths of the inner ears, which can produce motion sickness. Stimulation of higher brain centers through sights, sounds, odors, tastes, emotions, or mechanical stimulation of the back of the pharynx can also trigger vomiting.

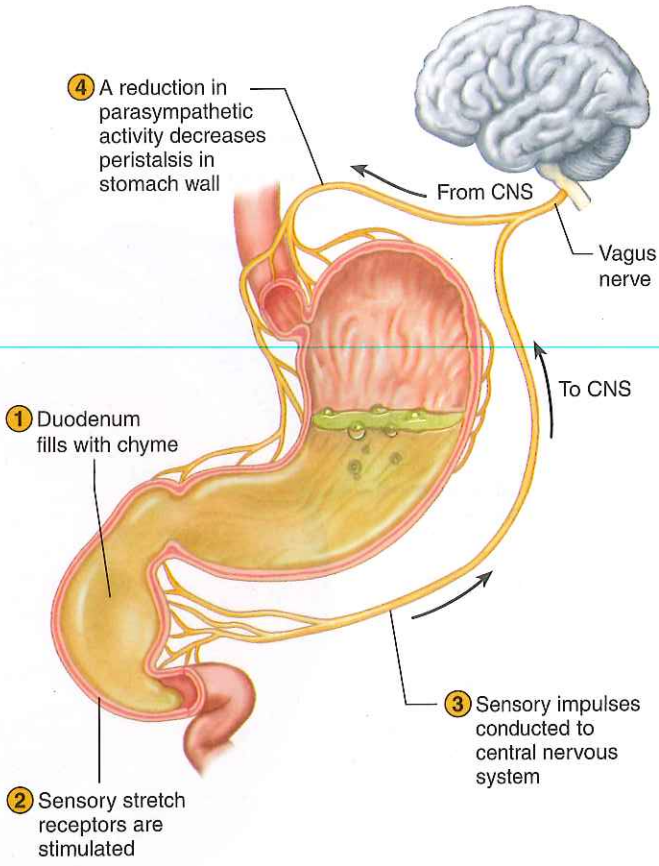


FIGURE 17.22 The enterogastric reflex partially regulates the rate at which chyme leaves the stomach.

Nausea emanates from activity in the vomiting center or in nerve centers near it. During nausea, stomach movements usually are diminished or absent, and duodenal contents may move back into the stomach.

PRACTICE

25 How is chyme produced?
26 What factors influence how quickly chyme leaves the stomach?
27 Describe the enterogastric reflex.
28 Describe the vomiting reflex.

17.7 PANCREAS

The **pancreas** was discussed as an endocrine gland in chapter 13 (pp. 513–515). It also has an exocrine function—secretion of a digestive fluid called **pancreatic juice** (pan″kre-at′ik jo͞os).

Structure of the Pancreas

The pancreas is closely associated with the small intestine and is posterior to the parietal peritoneum. It extends horizontally across the posterior abdominal wall, with its head in the C-shaped curve of the duodenum (portion of the small intestine) and its tail against the spleen (fig. 17.23 and reference plate 19, p. 53).

The cells that produce pancreatic juice, called *pancreatic acinar cells,* make up the bulk of the pancreas. These cells form clusters called *acini* (*acinus,* sing.) around tiny tubes into which they release their secretions. The smaller tubes unite to form larger ones, which, in turn, give rise to a *pancreatic duct* extending the length of the pancreas and transporting pancreatic juice to the small intestine. The pancreatic duct usually connects with the duodenum at the same place where the bile duct from the liver and gallbladder joins the duodenum, although other connections may be present (see figs. 13.34, p. 513 and 17.23).

The pancreatic and bile ducts join at a short, dilated tube called the *hepatopancreatic ampulla* (ampulla of Vater).

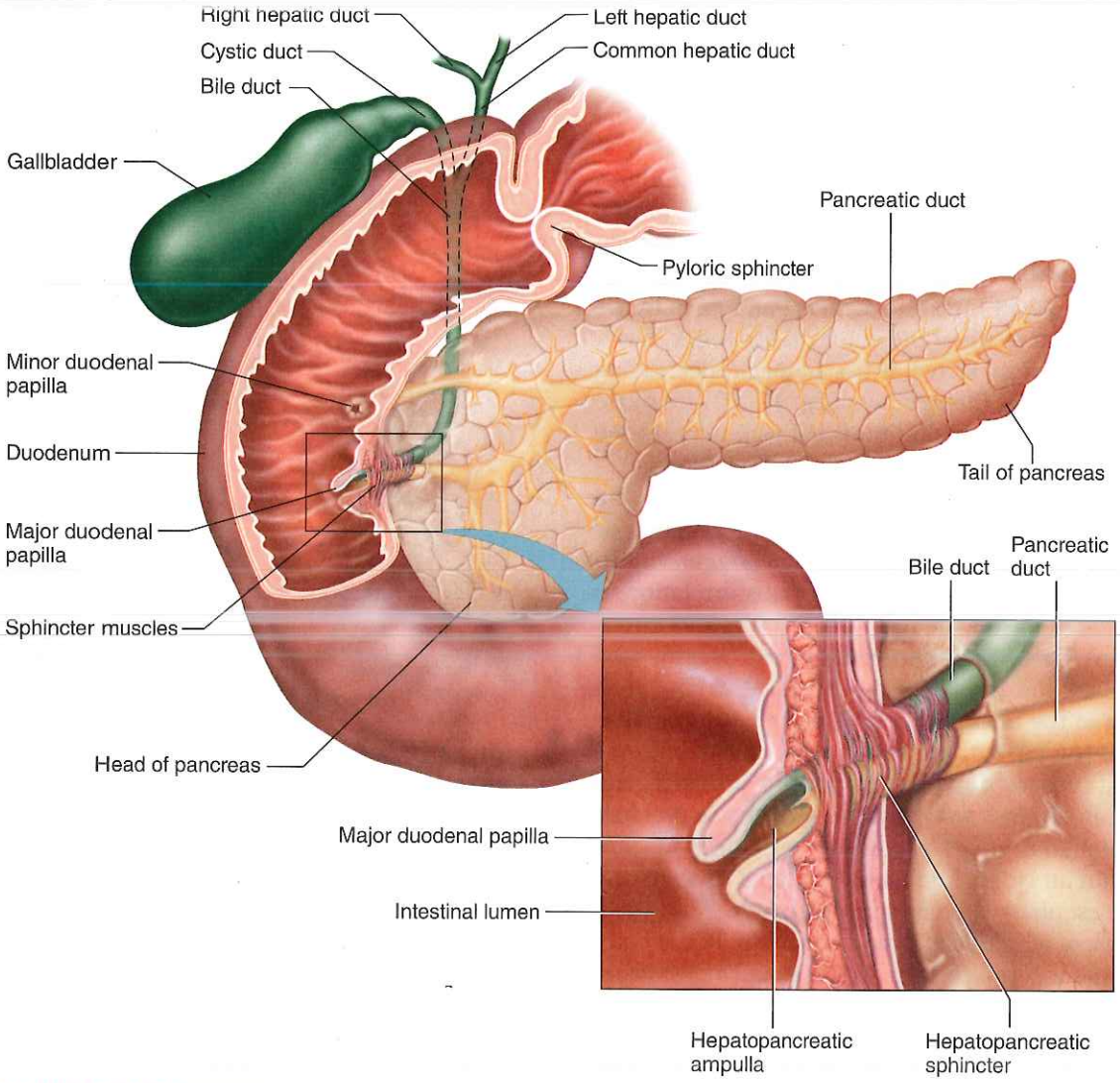


FIGURE 17.23 AP|R The pancreas is closely associated with the duodenum.

A band of smooth muscle, called the *hepatopancreatic sphincter* (sphincter of Oddi), surrounds this ampulla. This sphincter controls the movement of pancreatic juice and bile into the small intestine.

Pancreatic Juice

Pancreatic juice contains enzymes that digest carbohydrates, fats, proteins, and nucleic acids. The carbohydrate-digesting enzyme, **pancreatic amylase,** splits molecules of starch or glycogen into disaccharides. The fat-digesting enzyme, **pancreatic lipase,** breaks triglyceride molecules into fatty acids and monoglycerides. (A monoglyceride molecule consists of one fatty acid bound to glycerol.)

The protein-splitting (proteolytic) enzymes are **trypsin, chymotrypsin,** and **carboxypeptidase.** Each of these enzymes splits the bonds between particular combinations of amino acids in proteins. No single enzyme can split all possible amino acid combinations. Therefore, several enzymes are necessary to completely digest protein molecules.

The proteolytic enzymes are stored in tiny cellular structures called *zymogen granules.* These enzymes, like gastric pepsin, are secreted in inactive forms and must be activated by other enzymes after they reach the small intestine. For example, the pancreatic cells release inactive **trypsinogen,** which is activated to trypsin when it contacts the enzyme *enterokinase,* which the mucosa of the small intestine secretes. Trypsin, in turn, activates chymotrypsin and carboxypeptidase. This mechanism prevents enzymatic digestion of proteins in the secreting cells and the pancreatic ducts.

Painful *acute pancreatitis* results when pancreatic enzymes become active before they are secreted, and digest parts of the pancreas. This can be caused by blockage in the release of pancreatic juice. Alcoholism, gallstones, certain infections, traumatic injuries, or the side effects of some drugs can cause pancreatitis.

Pancreatic juice contains two types of **nucleases,** which are enzymes that break down nucleic acid molecules into nucleotides. A high concentration of bicarbonate ions makes the juice alkaline, which provides a favorable environment for the actions of the digestive enzymes and helps neutralize acidic chyme as it arrives from the stomach. The alkaline environment in the small intestine also blocks the action of pepsin, which might otherwise damage the duodenal wall.

Regulation of Pancreatic Secretion

The nervous and endocrine systems regulate release of pancreatic juice, much as they regulate gastric and small intestinal secretions. For example, during the cephalic and gastric phases of gastric secretion, parasympathetic impulses stimulate the pancreas to release digestive enzymes. A peptide hormone, **secretin,** stimulates the pancreas to secrete abundant fluid when acidic chyme enters the duodenum. Secretin is released into the blood from the duodenal mucous membrane in response to the acid in chyme. The pancreatic juice secreted at this time contains few, if any, digestive enzymes but has a high concentration of bicarbonate ions. These ions neutralize the acid in chyme (fig. 17.24).

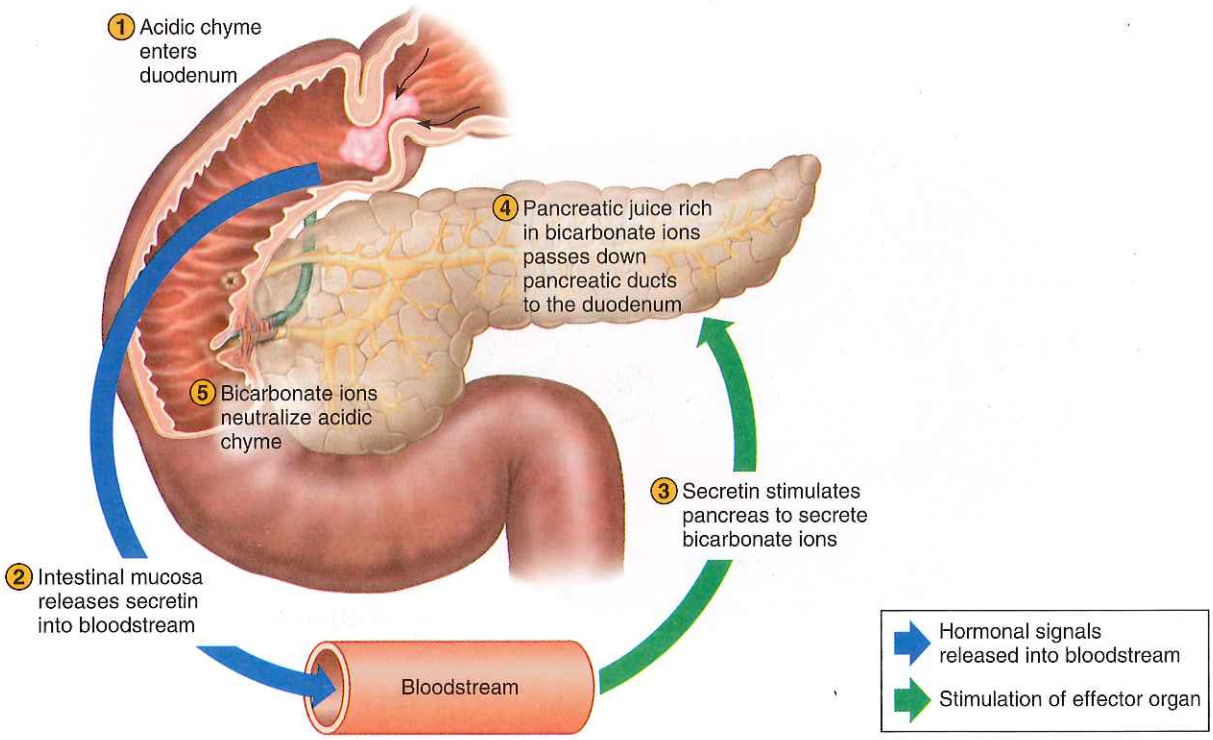


FIGURE 17.24 Acidic chyme entering the duodenum from the stomach stimulates the release of secretin, which, in turn, stimulates the release of pancreatic juice.

Proteins and fats in chyme in the duodenum also stimulate the release of *cholecystokinin* from the intestinal wall. As in the case of secretin, cholecystokinin reaches the pancreas in the bloodstream. Pancreatic juice secreted in response to cholecystokinin has a high concentration of digestive enzymes.

PRACTICE

29 Where is the pancreas located?
30 List the enzymes in pancreatic juice.
31 What are the functions of the enzymes in pancreatic juice?
32 What regulates secretion of pancreatic juice?

17.8 LIVER

The **liver,** the largest internal organ, is located in the upper right quadrant of the abdominal cavity, just inferior to the diaphragm. A fold of visceral peritoneum called the *coronary ligament* attaches the liver to the diaphragm on its superior surface. The liver is partially surrounded by the ribs and extends from the level of the fifth intercostal space to the lower margin of the ribs. It is reddish brown in color and well supplied with blood vessels (**figs. 17.25, 17.26, and 17.27** and reference plates 8, 17, and 24, pp. 46, 52, and 56).

Liver Structure

A fibrous capsule encloses the liver, and connective tissue divides the organ into a large *right lobe* and a smaller *left lobe.* The *falciform ligament* is a fold of visceral peritoneum that separates the lobes and fastens the liver to the abdominal wall anteriorly. The liver also has two minor lobes, the *quadrate lobe,* near the gallbladder, and the *caudate lobe,* close to the vena cava (see fig. 17.26). The area where the four lobes meet and blood vessels and ducts enter or exit the liver is the *porta hepatis.*

Each lobe is separated into many tiny **hepatic lobules,** which are the liver's functional units (fig. 17.27). A lobule consists of many *hepatic cells* radiating outward from a *central vein.* Vascular channels called **hepatic sinusoids** separate platelike groups of these cells from each other. Blood from the digestive tract, carried in the *hepatic portal vein* (see chapter 15, pp. 603 and 605), brings newly absorbed nutrients into the sinusoids **(fig. 17.28)**. At the same time, oxygenated blood from the hepatic artery mixes freely with the blood containing nutrients, then flows through the liver sinusoids and nourishes the hepatic cells.

Often blood in the hepatic portal vein contains some bacteria that have entered through the intestinal wall. However, large **Kupffer cells,** fixed to the inner lining (endothelium) of the hepatic sinusoids, remove most of the bacteria from the blood by phagocytosis. Then the blood passes into the *central veins* of the hepatic lobules and exits the liver via the hepatic vein.

Within the hepatic lobules are many fine *bile canaliculi,* which carry secretions from hepatic cells to *bile ductules.* The ductules of neighboring lobules unite to form intrahepatic bile ducts, which then converge to become the **hepatic ducts.** These ducts merge, in turn, to form the **common hepatic duct.**

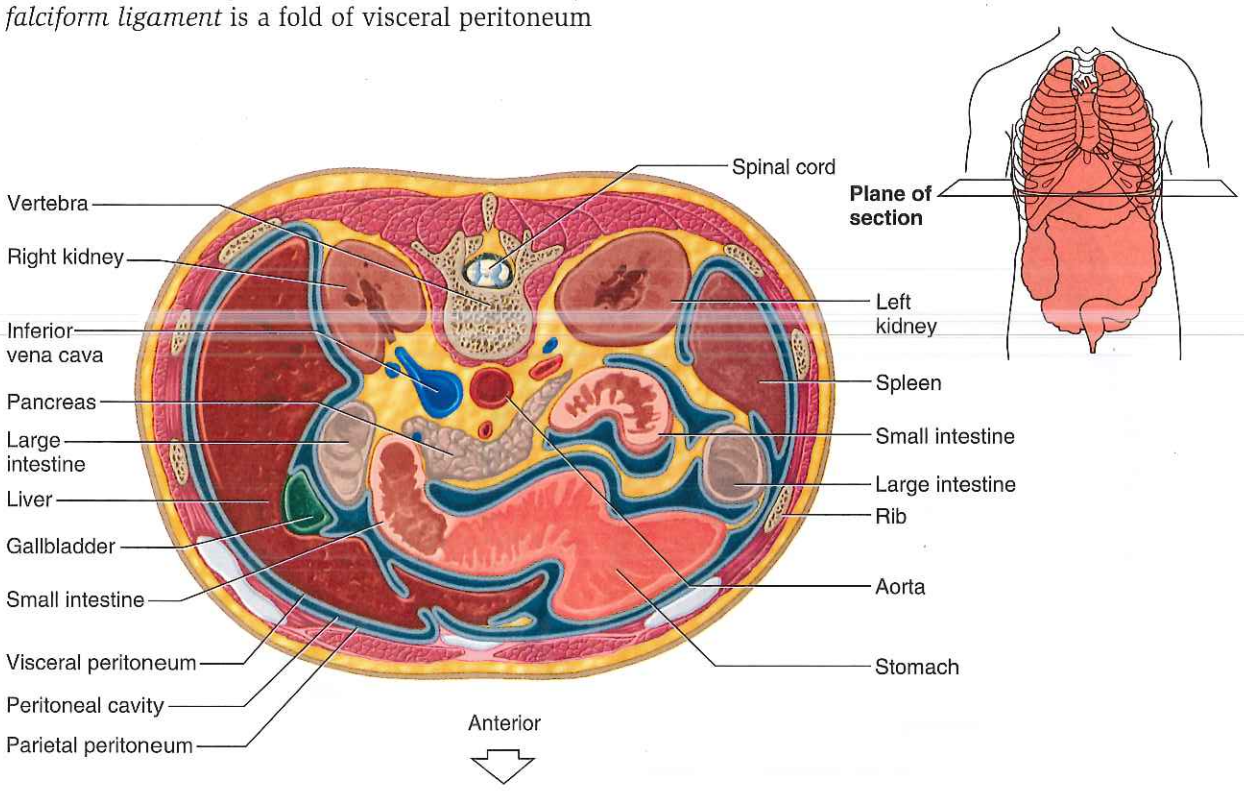


FIGURE 17.25 AP|R This transverse section of the abdomen reveals the liver and other organs in the upper part of the abdominal cavity.

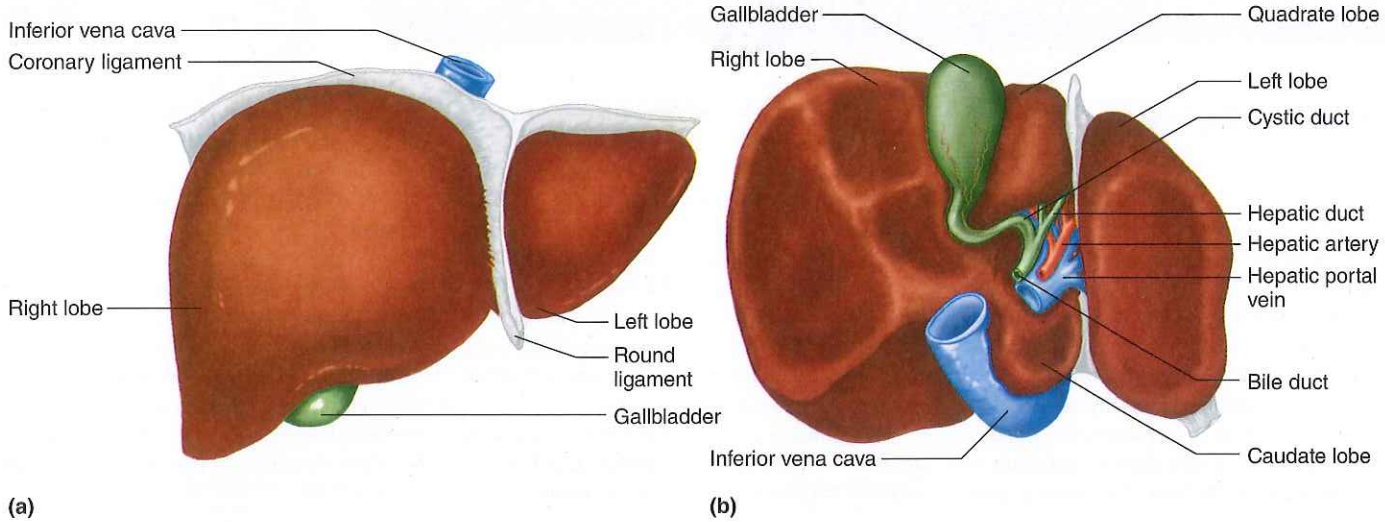


FIGURE 17.26 AP|R Lobes of the liver, viewed (*a*) anteriorly and (*b*) inferiorly.

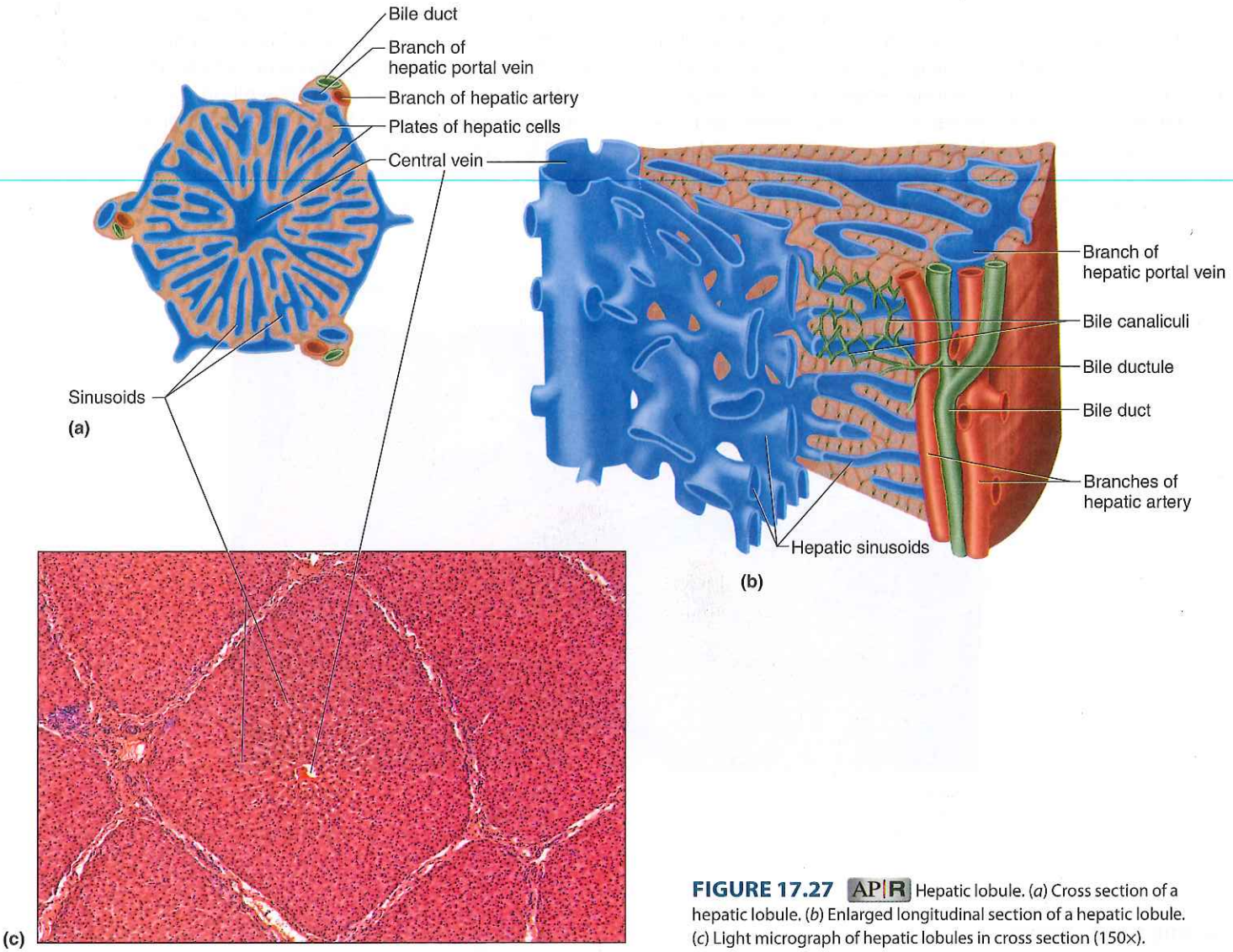


FIGURE 17.27 AP|R Hepatic lobule. (*a*) Cross section of a hepatic lobule. (*b*) Enlarged longitudinal section of a hepatic lobule. (*c*) Light micrograph of hepatic lobules in cross section (150×).

17.1 FROM SCIENCE TO TECHNOLOGY

Replacing the Liver

Life without a liver is not possible. Although the liver is capable of regeneration if even a small part of it (25 - 30%) remains healthy, a person can survive only a few days once the liver stops functioning. In fulminant hepatic failure, for example, the liver of an otherwise healthy young person suddenly fails. This condition is caused by exposure to a toxin, reaction to a drug, or a viral infection. Jaundice and fatigue progress rapidly to coma and death. In another example of the liver's importance, once cancer spreads to the liver, survival is generally only weeks or a few months.

Livers are in great demand for transplant, but they are scarce. Each year in the United States, only about 4,500 of the 12,000 or so individuals requiring livers survive long enough to undergo a transplant. A person can receive part of a liver donated by a living relative or other close match. The donor lives because only a portion of the liver is needed for survival, and enough is left by the procedure to allow regeneration to occur.

A promising solution to the liver shortage is an "extracorporeal liver assist device" (ELAD). It can take over the liver's blood-cleansing function until a cadaver organ becomes available, or enable remaining functional liver tissue to stimulate enough regeneration to restore health.

An ELAD is a "bioartificial" liver because it has synthetic as well as biological components. The device consists of four cartridges filled with hollow fibers that house millions of human liver cells (hepatocytes). A patient's plasma is separated from the blood and passed through the device, where the liver cells remove toxins and add liver-secreted products, such as clotting factors. The plasma is then reunited with the formed elements of the blood, and the blood reinfused into the patient. It is in clinical trials in several countries. ■

Liver Functions

The liver carries on many important metabolic activities. From Science to Technology 17.1 discusses a bioengineered liver. Recall from chapter 13 (p. 514) that the liver plays a key role in carbohydrate metabolism by helping maintain concentration of blood glucose within the normal range. Liver cells responding to hormones such as insulin and glucagon lower the blood glucose level by polymerizing glucose to glycogen and raise the blood glucose level by breaking down glycogen to glucose or by converting noncarbohydrates into glucose.

The liver's effects on lipid metabolism include oxidizing fatty acids at an especially high rate (see chapter 18, p. 699); synthesizing lipoproteins, phospholipids, and cholesterol; and converting portions of carbohydrate and protein molecules into fat molecules. The blood transports fats synthesized in the liver to adipose tissue for storage.

The most vital liver functions are probably those related to protein metabolism. They include deaminating amino acids; forming urea (see chapter 18, p. 700); synthesizing plasma

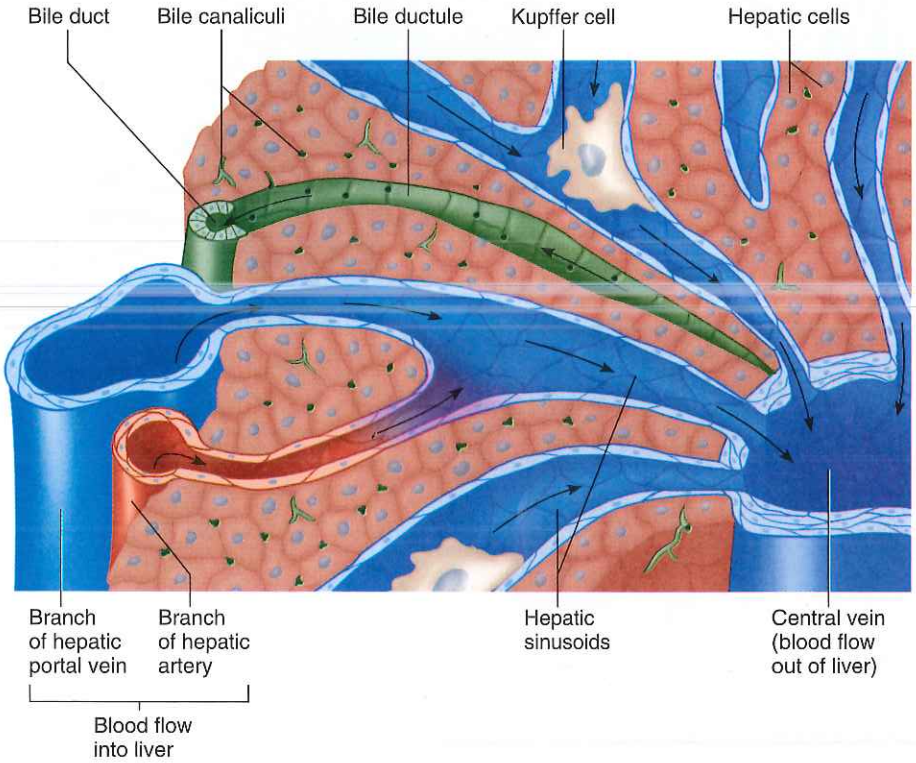
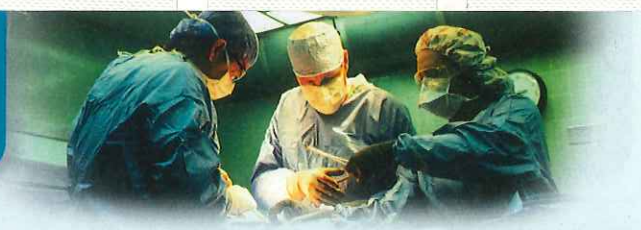


FIGURE 17.28 AP|R The paths of blood and bile in a hepatic lobule.

17.3 CLINICAL APPLICATION



Hepatitis

Hepatitis is an inflammation of the liver. It has several causes, but the various types have similar symptoms.

For the first few days, hepatitis may resemble the flu, producing mild headache, low fever, fatigue, lack of appetite, nausea and vomiting, and sometimes stiff joints. By the end of the first week, more distinctive symptoms arise: a rash, pain in the upper right quadrant of the abdomen, dark and foamy urine, and pale feces. The skin and sclera of the eyes begin to turn yellow due to accumulating bile pigments (jaundice). Great fatigue may continue for two or three weeks, and then gradually the person begins to feel better.

This is hepatitis in its most common, least dangerous, acute form. About half a million people develop hepatitis in the United States each year, and 6,000 die. In a rare form called *fulminant hepatitis,* symptoms are sudden and severe, along with altered behavior and personality. Medical attention is necessary to prevent kidney or liver failure or coma. Hepatitis that persists for more than six months is termed chronic.

Only rarely does hepatitis result from alcoholism, autoimmunity, or the use of certain drugs. Usually, one of several types of viruses causes hepatitis. Viral types are distinguished by the route of infection, surface features, and whether the viral genetic material is DNA or RNA. Hepatitis B virus has DNA; the others have RNA. The viral types are classified as follows:

Hepatitis A spreads by contact with food or objects contaminated with virus-containing feces, including diapers. The course of hepatitis A is short and mild.

Hepatitis B spreads by contact with virus-containing body fluids, such as blood, saliva, or semen. It may be transmitted by blood transfusions, hypodermic needles, or sexual activity.

Hepatitis C accounts for about half of all known cases of hepatitis. This virus is primarily transmitted in blood—by sharing razors or needles, from pregnant woman to fetus, or through blood transfusions or use of blood products. Ten to twenty percent of people known to be infected with hepatitis C virus develop symptoms.

Hepatitis D occurs in people already infected with the hepatitis B virus. It is blood-borne and associated with blood transfusions and intravenous drug use. About 20% of individuals infected with this virus die from the infection.

Hepatitis E virus is usually transmitted in water contaminated with feces. It most often affects visitors to developing nations.

Hepatitis G is rare but seems to account for a significant percentage of cases of fulminant hepatitis. In people with healthy immune systems, it produces symptoms so mild that they may not even be noticed.

A virus usually causes hepatitis, so antibiotic drugs, effective against bacteria, are not helpful. Usually, the person must just wait out the symptoms. Many cases of hepatitis C, resolve on their own or respond to a 6 to 12 month course of a form of interferon or either of two new drugs. As many as 300 million people worldwide are hepatitis carriers. They do not have symptoms but can infect others. Five percent of carriers eventually develop liver cancer. ■

proteins such as clotting factors (see chapter 14, p. 539); and converting certain amino acids into other amino acids.

> Bacteria in the intestine produce ammonia, which is carried in the blood to the liver, where it reacts to yield urea. When this liver function fails, concentration of blood ammonia sharply rises, causing *hepatic coma,* a condition that can lead to death.

The liver also stores many substances, including glycogen, iron, and vitamins A, D, and B_{12}. Extra iron from the blood combines with a protein (apoferritin) in liver cells, forming *ferritin.* The iron is stored in this form until blood iron concentration falls, when some of the iron is released. Thus, the liver is important in iron homeostasis.

Liver cells help destroy damaged red blood cells and phagocytize foreign antigens. The liver removes toxic substances such as alcohol and certain drugs from the blood (detoxification). The liver can also serve as a blood reservoir, storing 200 to 400 milliliters of blood. The liver's role in digestion is to secrete bile. Table 17.7 summarizes the major functions of the liver. Clinical Application 17.3 discusses hepatitis, an inflammation of the liver.

PRACTICE

33 Locate the liver.

34 Review liver functions.

35 How does the liver aid in digestion?

TABLE 17.7 | Major Functions of the Liver

General Function	Specific Function
Carbohydrate metabolism	Polymerizes glucose to glycogen; breaks down glycogen to glucose; converts noncarbohydrates to glucose
Lipid metabolism	Oxidizes fatty acids; synthesizes lipoproteins, phospholipids, and cholesterol; converts portions of carbohydrate and protein molecules into fats
Protein metabolism	Deaminates amino acids; forms urea; synthesizes plasma proteins; converts certain amino acids into other amino acids
Storage	Stores glycogen, vitamins A, D, and B_{12}, iron, and blood
Blood filtering	Removes damaged red blood cells and foreign substances by phagocytosis
Detoxification	Removes toxins from the blood
Secretion	Produces and secretes bile

Composition of Bile

Bile (bīl) is a yellowish-green liquid that is continuously secreted from hepatic cells. In addition to water, bile contains *bile salts, bile pigments, cholesterol,* and *electrolytes.* Bile salts are the most abundant and are the only bile components that have a digestive function.

Hepatic cells use cholesterol to produce bile salts, and in secreting these salts, they release some cholesterol into the bile. Cholesterol by itself has no special function in bile or in the alimentary canal.

17.4 CLINICAL APPLICATION

Gallbladder Disease

Molly G., an overweight, forty-seven-year-old college administrator and mother of four, had pain in the upper-right quadrant of her abdomen (see fig. 1.24b, p. 32). Sometimes the discomfort seemed to radiate around to her back and move upward into her right shoulder. She usually felt this pain after her evening meal, but it occasionally happened at night, awakening her. After an episode of severe pain accompanied by sweating (diaphoresis) and nausea, Molly visited her physician, who discovered tenderness in the epigastric region (see fig. 1.24a, p. 32). She decided that Molly's symptoms could indicate *acute cholecystitis*—an inflammation of the gallbladder. Molly could be diagnosed with ultrasonography of her gallbladder or by an X ray of the gallbladder, called a *cholecystogram*.

Molly took tablets containing a contrast medium the night before the X-ray procedure, which allowed time for the small intestine to absorb the substance and for it to reach the liver and be excreted into the bile. Later, contrast medium would be concentrated in the bile and stored in the gallbladder and would make the contents of the gallbladder opaque to X rays.

Molly's cholecystogram revealed several stones (calculi) in her gallbladder, a condition called *cholelithiasis* (see fig. 17.29). Molly's symptoms were worsening, so her physician recommended *cholecystectomy*—surgical removal of the gallbladder.

An incision was made in Molly's right subcostal region and her gallbladder excised from the liver. Fortunately, the cystic duct and hepatic ducts did not have stones (see fig. 17.26).

Unfortunately, Molly's symptoms persisted following her recovery from surgery. So her surgeon ordered a *cholangiogram*—an X-ray series of the bile ducts. This study showed a residual stone at the distal end of Molly's bile duct (see fig. 17.23).

The surgeon extracted the residual stone using a *fiber-optic endoscope*, a long, flexible tube passed through the esophagus and stomach and into the duodenum. This instrument enables a surgeon to observe features of the gastrointestinal tract directly through the eyepiece of the endoscope or on a monitor. A surgeon can also perform manipulations using specialized tools passed through the endoscope to its distal end.

In Molly's case, the surgeon performed an *endoscopic papillotomy*—an incision of the hepatopancreatic sphincter made by applying an electric current to a wire extending from the end of the endoscope (see fig. 17.23). She then removed the exposed stone by manipulating a tiny basket at the tip of the endoscope. Many patients undergo only the endoscopic procedure to remove the gallbladder, performed on an outpatient basis. ■

Bile pigments (bilirubin and biliverdin) are breakdown products of hemoglobin from red blood cells (see chapter 14, p. 532). These pigments are normally excreted in the bile. The yellowish skin, sclerae, and mucous membranes of jaundice result from excess deposition of bile pigments.

> Jaundice has several causes. In *obstructive jaundice,* bile ducts are blocked, perhaps by gallstones or tumors. In *hepatocellular jaundice,* the liver is diseased, as in cirrhosis or hepatitis. In *hemolytic jaundice,* red blood cells are destroyed too rapidly, as happens with an incompatible blood transfusion or a blood infection.

Bile salts, bile pigments, and cholesterol become increasingly concentrated as the gallbladder lining reabsorbs water and electrolytes. The cholesterol normally remains in solution, but under certain conditions it may precipitate and form solid crystals. If cholesterol continues to come out of solution, the crystals enlarge, forming *gallstones* (fig. 17.29). This may happen if the bile is too concentrated, hepatic cells secrete too much cholesterol, or the gallbladder is inflamed (cholecystitis). Gallstones in the bile duct may block the flow of bile, causing obstructive jaundice and considerable pain. Clinical Application 17.4 discusses disorders of the gallbladder.

Gallbladder

The **gallbladder** is a pear-shaped sac in a depression on the inferior surface of the liver. It is connected to the **cystic duct,** which, in turn, joins the common hepatic duct (see fig. 17.23 and reference plate 19, p. 53). The gallbladder has a capacity of 30–50 milliliters, is lined with columnar epithelial cells, and has a strong muscular layer in its wall. It stores bile between meals, concentrates bile by reabsorbing water, and contracts to release bile into the small intestine.

The **bile duct** is formed by the union of the common hepatic and cystic ducts. It leads to the duodenum, where the hepatopancreatic sphincter muscle guards its exit (see fig. 17.23). This sphincter normally remains contracted, so as bile collects in the common bile duct it backs up into the cystic duct. When this happens, the bile flows into the gallbladder, where it is stored.

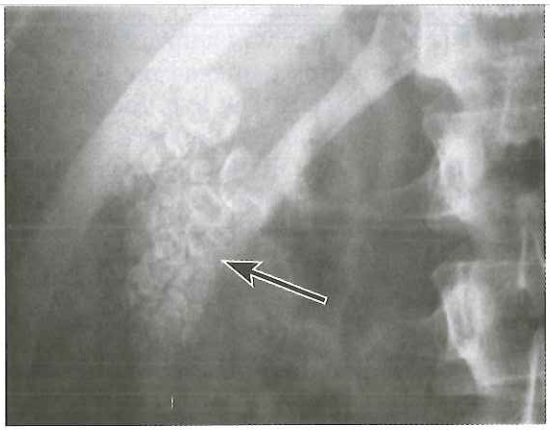


FIGURE 17.29 Radiograph of a gallbladder that contains gallstones (arrow).

Regulation of Bile Release

Normally, bile does not enter the duodenum until *cholecystokinin* stimulates the gallbladder to contract. The intestinal mucosa releases this hormone in response to proteins and fats in the small intestine. (Recall from earlier in this chapter, p. 669, that cholecystokinin also stimulates pancreatic enzyme secretion.) The hepatopancreatic sphincter usually remains contracted until a peristaltic wave in the duodenal wall approaches. Just before the wave hits, the sphincter relaxes, and bile squirts into the duodenum (fig. 17.30). Table 17.8 summarizes the hormones that control digestion.

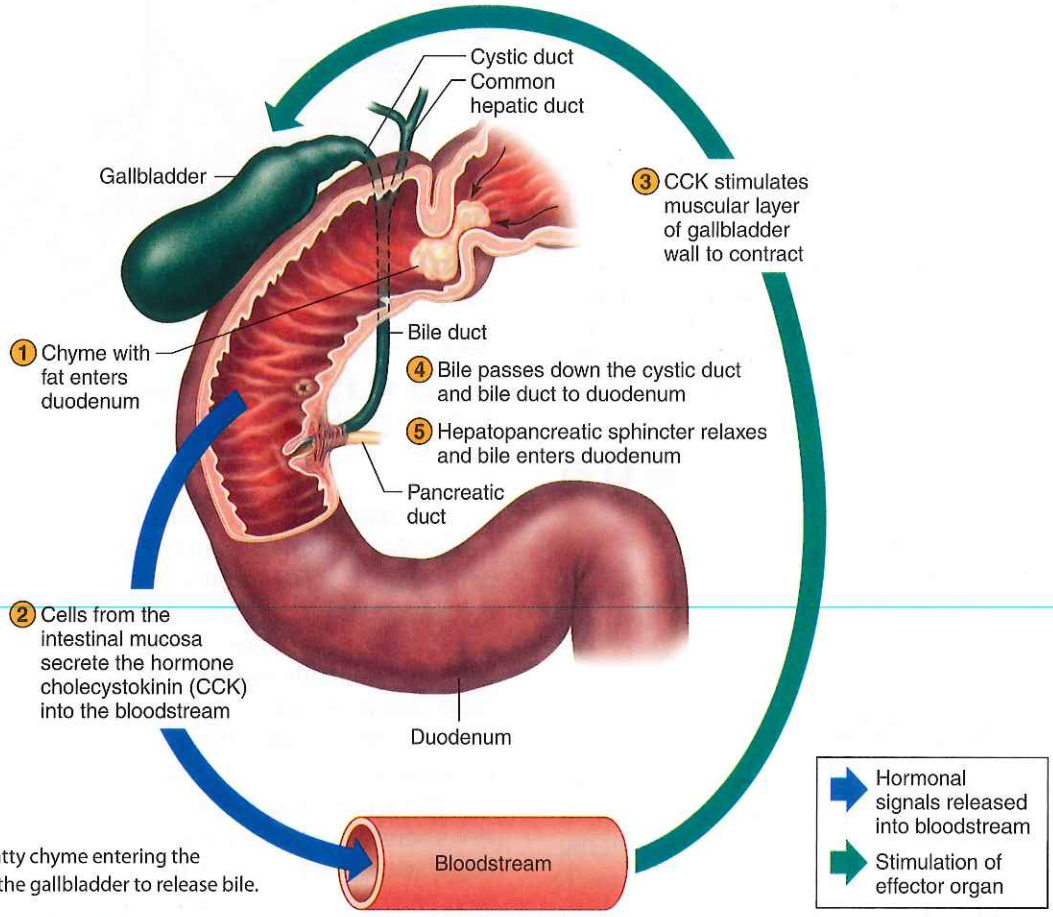


FIGURE 17.30 Fatty chyme entering the duodenum stimulates the gallbladder to release bile.

Q: *Which other organ, besides the gallbladder, responds to cholecystokinin stimulation, and what is the response of that organ to cholecystokinin stimulation?*

Answer can be found in Appendix G on page 938.

TABLE 17.8 | Hormones of the Digestive Tract

Hormone	Source	Function
Gastrin	Gastric cells, in response to food	Increases secretory activity of gastric glands
Intestinal gastrin	Cells of small intestine, in response to chyme	Increases secretory activity of gastric glands
Somatostatin	Gastric cells	Inhibits secretion of acid by parietal cells
Intestinal somatostatin	Intestinal wall cells, in response to fats	Inhibits secretion of acid by parietal cells
Cholecystokinin	Intestinal wall cells, in response to proteins and fats in the small intestine	Decreases secretory activity of gastric glands and inhibits gastric motility; stimulates pancreas to secrete fluid with a high digestive enzyme concentration; stimulates gallbladder to contract and release bile
Secretin	Cells in the duodenal wall, in response to acidic chyme entering the small intestine	Stimulates pancreas to secrete fluid with a high bicarbonate ion concentration

Functions of Bile Salts

Bile salts aid digestive enzymes. Molecules of fats clump into *fat globules*. Bile salts reduce surface tension and break fat globules into droplets, much like the action of soap or detergent. This process is called **emulsification.** Monoglycerides that form from the action of pancreatic lipase on triglyceride molecules aid emulsification. Overall, emulsification greatly increases the total surface area of the fatty substance, and the resulting droplets mix with water. Lipases can then digest the fat molecules more effectively.

Bile salts enhance absorption of fatty acids and cholesterol by forming complexes (micelles) that are very soluble in chyme and that epithelial cells can more easily absorb. The fat-soluble vitamins A, D, E, and K are also absorbed in the presence of bile salts. Lack of bile salts results in poor lipid absorption and vitamin deficiencies.

The mucous membrane of the small intestine reabsorbs nearly all of the bile salts, along with fatty acids. The blood carries bile salts to the liver, where hepatic cells resecrete them into the bile ducts. Liver cells synthesize bile salts, which replace the small amounts lost in the feces.

PRACTICE

36 Explain how bile forms.

37 Describe the function of the gallbladder.

38 How is secretion of bile regulated?

39 How do bile salts function in digestion?

17.9 SMALL INTESTINE

The **small intestine** is a tubular organ that extends from the pyloric sphincter to the beginning of the large intestine. With its many loops and coils, the small intestine fills much of the abdominal cavity (see fig. 17.1 and reference plates 4 and 5, pp. 42–43). The small intestine is 5.5–6.0 meters (18–20 feet) long in a cadaver when the muscular wall lacks tone, but it may be only half this long in a living person.

The small intestine receives secretions from the pancreas and liver. It also completes digestion of the nutrients in chyme, absorbs the products of digestion, and transports the remaining residue to the large intestine.

Parts of the Small Intestine

The small intestine, shown in figures 17.31 and 17.32 and in reference plates 12 and 18 (pp. 49 and 52), consists of three portions: the duodenum, the jejunum, and the ileum.

The **duodenum** (du″o-de′num), which is about 25 centimeters long and 5 centimeters in diameter, lies posterior to the parietal peritoneum (retroperitoneal). It is the shortest and most fixed portion of the small intestine. The duodenum follows a C-shaped path as it passes anterior to the right kidney and the upper three lumbar vertebrae.

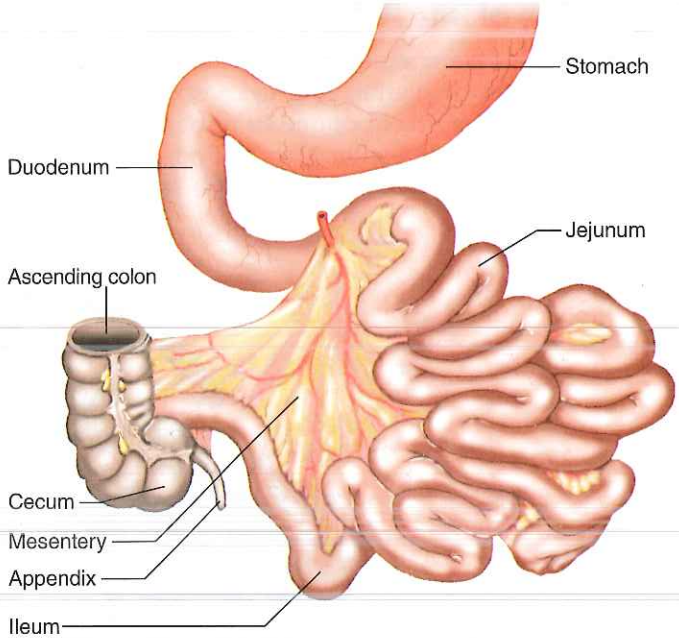


FIGURE 17.31 The three parts of the small intestine are the duodenum, the jejunum, and the ileum.

The remainder of the small intestine is mobile and lies free in the peritoneal cavity. The proximal two-fifths of this portion of the small intestine is the **jejunum** (jĕ-jo͞o′num), and the remainder is the **ileum** (il′e-um). There is no distinct separation between the jejunum and ileum, but the diameter of the jejunum is usually greater, and its wall is thicker, more vascular, and more active than that of the ileum. The ileum has more lymph nodules (Peyer's patches, p. 621) and a greater bacterial population.

The jejunum and ileum are suspended from the posterior abdominal wall by a double-layered fold of peritoneum called **mesentery** (mes′en-ter″e) (fig. 17.33). The mesentery supports the blood vessels, nerves, and lymphatic vessels that supply the intestinal wall (see fig. 17.31 and reference plate 5, p. 43).

A filmy, double fold of peritoneal membrane called the *greater omentum* drapes like an apron from the stomach over the transverse colon and the folds of the small intestine (fig. 17.34). If the wall of the alimentary canal becomes infected, cells from the omentum may adhere to the inflamed region and help seal it off, lowering the risk that the infection will spread to the peritoneal cavity.

Structure of the Small Intestinal Wall

The inner wall of the small intestine has a velvety appearance throughout its length, due to many tiny projections of mucous membrane called **intestinal villi** (figs. 17.35 and 17.36; see fig. 17.3). These structures are most numerous in the duodenum and the proximal jejunum. They project into the lumen of the alimentary canal, contacting the intestinal

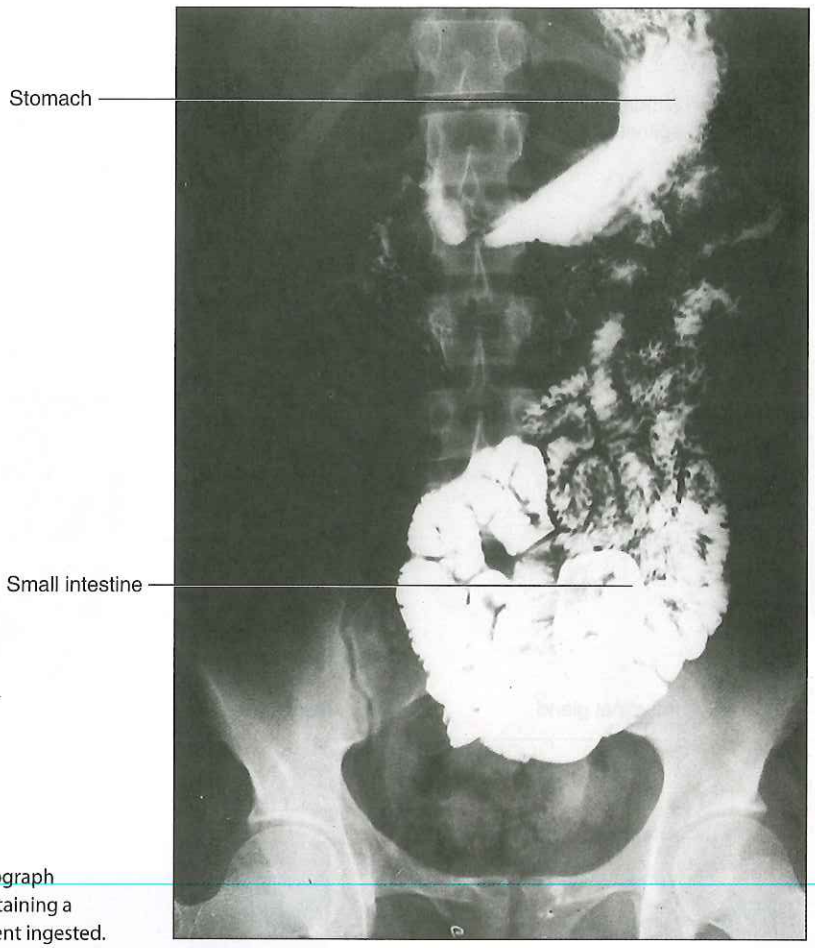


FIGURE 17.32 AP|R Radiograph showing normal small intestine containing a radiopaque substance that the patient ingested.

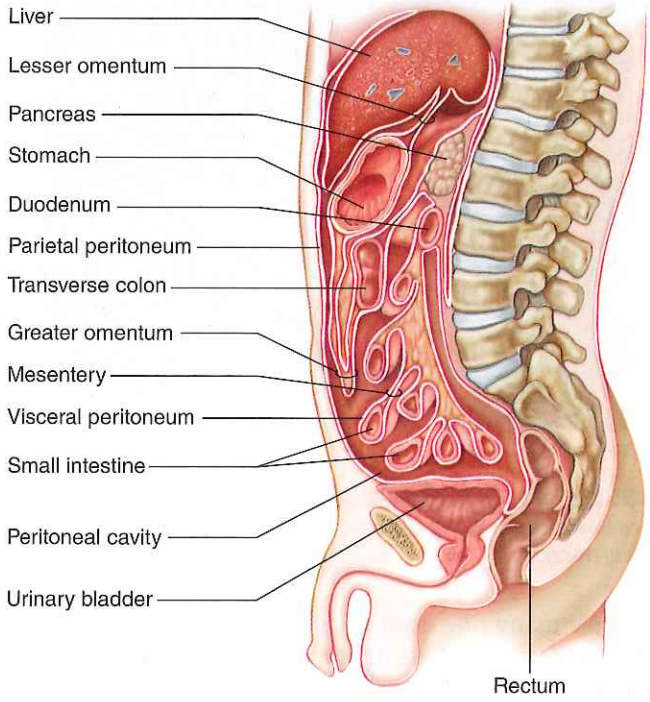


FIGURE 17.33 AP|R Mesentery formed by folds of the peritoneal membrane suspends parts of the small intestine from the posterior abdominal wall.

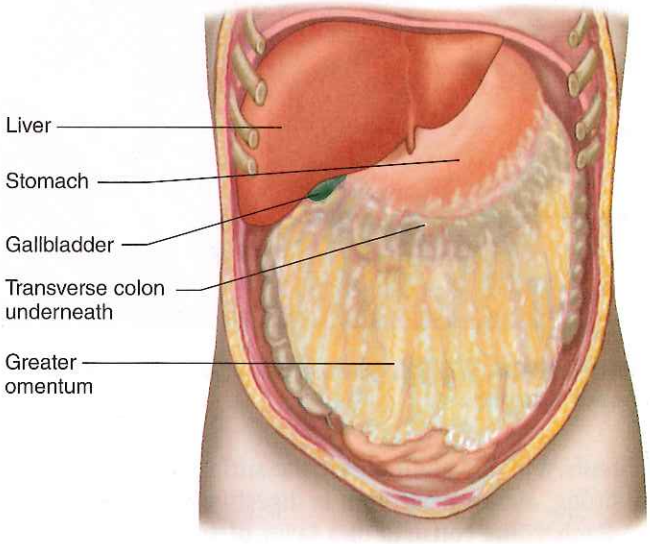


FIGURE 17.34 AP|R The greater omentum hangs like an apron over the abdominal organs.

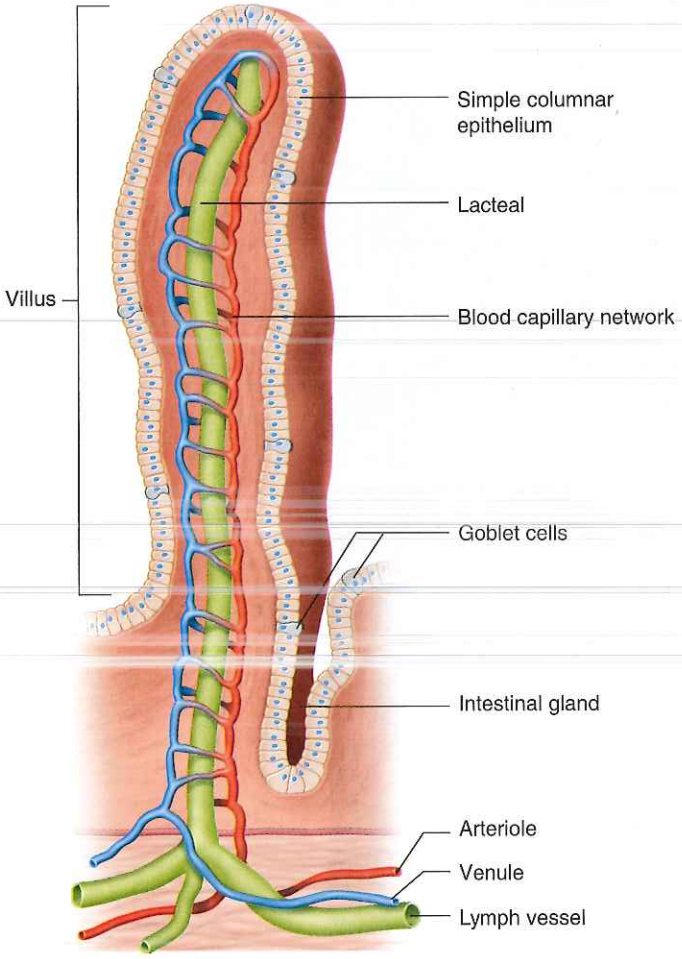


FIGURE 17.35 AP|R Structure of a single intestinal villus.

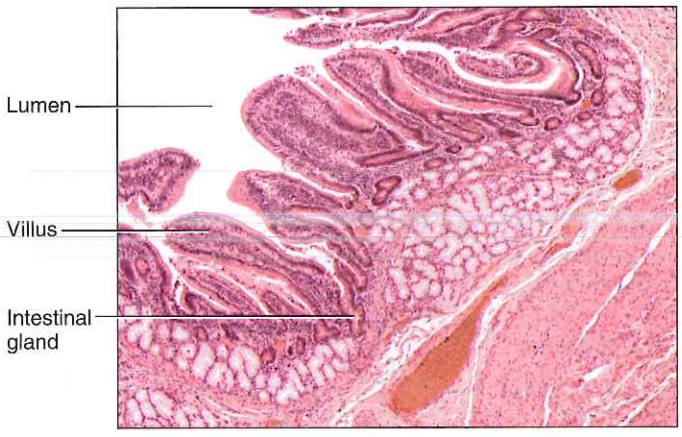


FIGURE 17.36 AP|R Light micrograph of intestinal villi from the wall of the duodenum (50×).

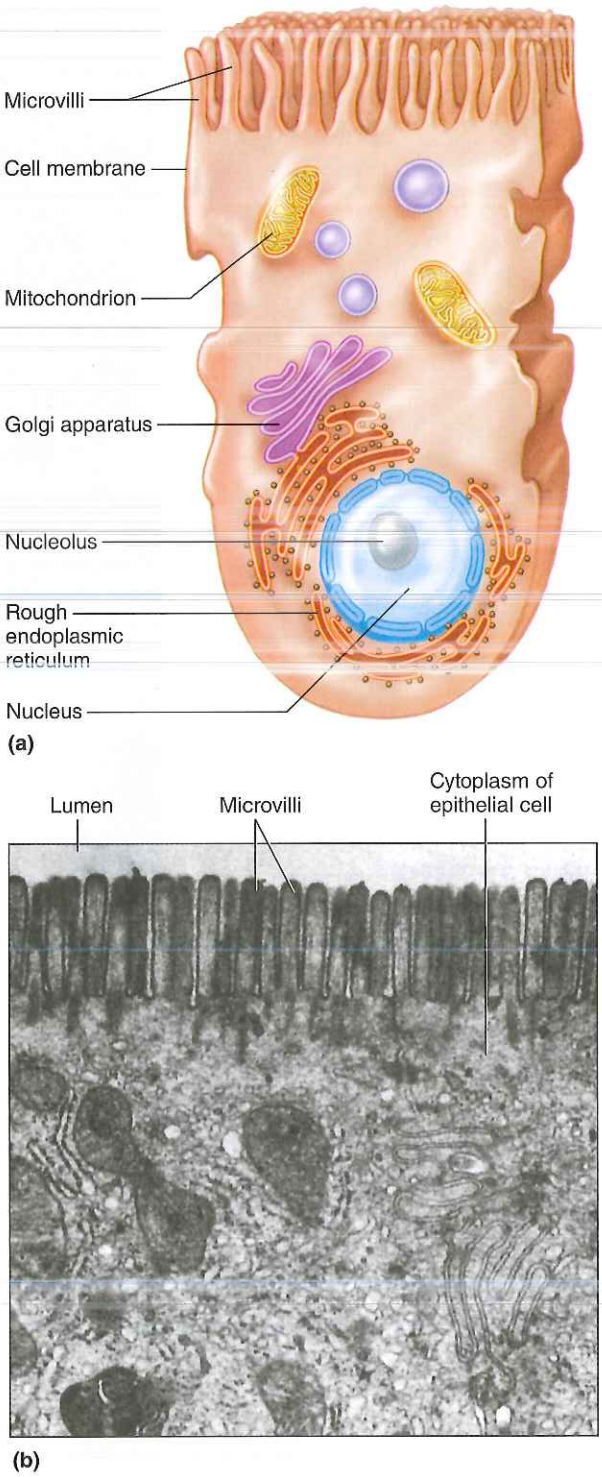


FIGURE 17.37 AP|R Intestinal epithelium. (*a*) Microvilli increase the surface area of intestinal epithelial cells. (*b*) Transmission electron micrograph of microvilli (16,000×).

contents. Villi greatly increase the surface area of the intestinal lining, aiding absorption of digestive products.

Each villus consists of a layer of simple columnar epithelium and a core of connective tissue containing blood capillaries, a lymphatic capillary called a **lacteal**, and nerve fibers (see fig. 17.35). At their free surfaces, the epithelial cells have many fine extensions called *microvilli* that form a brushlike border and greatly increase the surface area of the intestinal cells, further enhancing absorption (see fig. 17.3 and fig. 17.37). The blood capillaries and lacteals carry away absorbed nutrients, and impulses transmitted by the nerve fibers can stimulate or inhibit villus activities.

Between the bases of adjacent villi are tubular **intestinal glands** (crypts of Lieberkühn) that dip into the mucous membrane. The deeper layers of the small intestinal wall are much like those of other parts of the alimentary canal in that they include a submucosa, a muscular layer, and a serosa.

The lining of the small intestine has many circular folds of mucosa, called *plicae circulares,* that are especially well developed in the lower duodenum and upper jejunum (fig. 17.38). With the villi and microvilli, these folds help increase the surface area of the intestinal lining.

The epithelial cells that form the lining of the small intestine are continually replaced. New cells form in the intestinal glands by cell division and migrate outward onto the villus surface. When the migrating cells reach the tip of the villus, they are shed. As a result, nearly one-quarter of the bulk of feces consists of dead epithelial cells from the small intestine. This *cellular turnover* renews the small intestine's epithelial lining every three to six days.

Secretions of the Small Intestine

Mucus-secreting goblet cells are abundant throughout the mucosa of the small intestine. In addition, many specialized *mucus-secreting glands* (Brunner's glands) in the submucosa in the proximal portion of the duodenum secrete a thick, alkaline mucus in response to certain stimuli.

The intestinal glands at the bases of the villi secrete large volumes of a watery fluid (see fig. 17.35). The villi rapidly reabsorb this fluid, which carries digestive products into the villi. The fluid the intestinal glands secrete has a nearly neutral pH (6.5–7.5), and it lacks digestive enzymes. However, the epithelial cells of the intestinal mucosa have digestive enzymes embedded in the membranes of the microvilli on their luminal surfaces. These enzymes break down food molecules just before absorption takes place. The enzymes include **peptidases,** which split peptides into their constituent amino acids; **sucrase, maltase,** and **lactase,** which split the disaccharides sucrose, maltose, and lactose into the monosaccharides glucose, fructose, and galactose; and **intestinal lipase,** which splits fats into fatty acids and glycerol. Table 17.9 summarizes the sources and actions of the major digestive enzymes.

Regulation of Small Intestinal Secretions

Mucus protects the intestinal wall in the same way it protects the stomach lining. Therefore, mucus secretion increases in response to mechanical stimulation and the presence of irritants, such as gastric juice. Stomach contents entering the small intestine stimulate the duodenal mucous glands to release mucus.

Direct contact with chyme chemically and mechanically stimulates goblet cells and intestinal glands to secrete their products. Distension of the intestinal wall activates the nerve plexuses therein and stimulates parasympathetic reflexes that also trigger release of small intestine secretions.

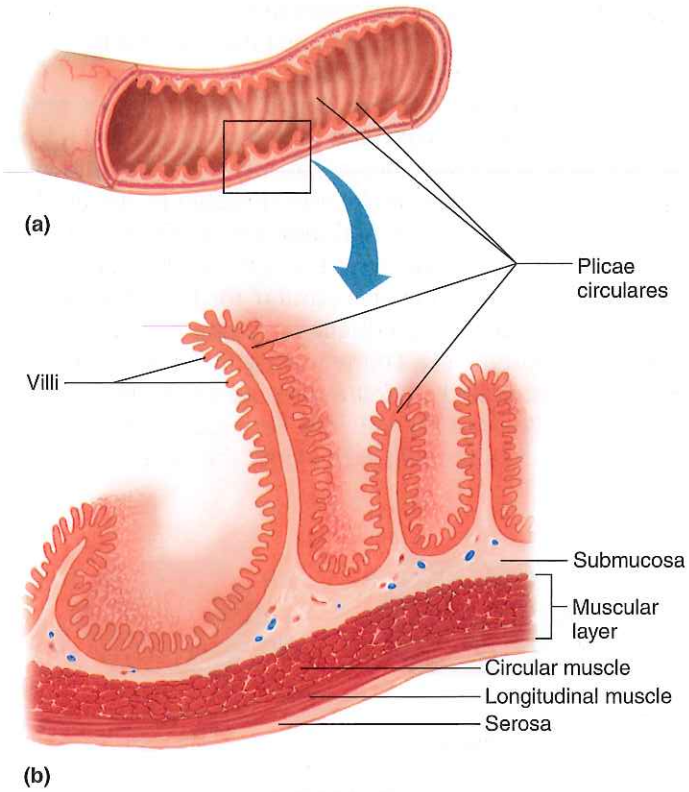


FIGURE 17.38 Section of small intestine. (*a*) The inner lining of the small intestine contains many circular folds, the plicae circulares. (*b*) A longitudinal section through some of these folds.

PRACTICE

40 Describe the parts of the small intestine.
41 What is the function of an intestinal villus?
42 Distinguish between intestinal villi and microvilli.
43 What is the function of the intestinal glands?
44 List intestinal digestive enzymes.

Adults who have *lactose intolerance* do not produce sufficient lactase to adequately digest lactose, or milk sugar. Undigested lactose increases osmotic pressure of the intestinal contents and draws water into the intestines. At the same time, intestinal bacteria metabolize undigested sugar, producing organic acids and gases. The overall result is bloating, intestinal cramps, and diarrhea.

Genetic evidence suggests that lactose intolerance may be the "normal" condition, with the ability to digest lactose the result of a mutation that occurred recently in our evolutionary past and became advantageous when agriculture brought dairy foods to human populations. The trait of ability to digest lactose has increased in parallel to increased use of dairy foods at least three times in history, in different populations.

TABLE 17.9 | Summary of the Major Digestive Enzymes AP R

Enzyme	Source	Digestive Action
Salivary Enzyme		
Salivary amylase	Salivary glands	Begins carbohydrate digestion by breaking down starch and glycogen to disaccharides
Gastric Enzymes		
Pepsin	Gastric glands	Begins protein digestion
Gastric lipase	Gastric glands	Begins butterfat digestion
Pancreatic Enzymes		
Pancreatic amylase	Pancreas	Breaks down starch and glycogen into disaccharides
Pancreatic lipase	Pancreas	Breaks down fats into fatty acids and glycerol
Trypsin, chymotrypsin	Pancreas	Breaks down proteins or partially digested proteins into peptides
Carboxypeptidase	Pancreas	Breaks down peptides into amino acids
Nucleases	Pancreas	Breaks down nucleic acids into nucleotides
Intestinal Enzymes		
Peptidase	Mucosal cells	Breaks down peptides into amino acids
Sucrase, maltase, lactase	Mucosal cells	Breaks down disaccharides into monosaccharides
Intestinal lipase	Mucosal cells	Breaks down fats into fatty acids and glycerol
Enterokinase	Mucosal cells	Converts trypsinogen into trypsin

Absorption in the Small Intestine

Villi greatly increase the surface area of the intestinal mucosa, making the small intestine the most important absorbing organ of the alimentary canal. The small intestine is so effective in absorbing digestive products, water, and electrolytes, that very little absorbable material reaches the organ's distal end.

RECONNECT

To Chapter 2, Organic Substances, pages 70–77.

Carbohydrate digestion begins in the mouth with the activity of salivary amylase and is completed in the small intestine by enzymes from the intestinal mucosa and pancreas (fig. 17.39). The resulting monosaccharides are absorbed by facilitated diffusion or active transport into the villi and enter blood capillaries (see chapter 3, pp. 101 and 103).

Protein digestion begins in the stomach as a result of pepsin activity and is completed in the small intestine by enzymes from the intestinal mucosa and the pancreas (fig. 17.40). Large protein molecules are ultimately broken down into amino acids, which are then absorbed into the villi by active transport and enter the circulation.

Fat molecules are digested almost entirely by enzymes from the intestinal mucosa and pancreas (fig. 17.41). The resulting fatty acid molecules are absorbed in the following steps: (1) The fatty acid molecules dissolve in the epithelial cell membranes of the villi and diffuse through them. (2) The endoplasmic reticula of the cells use the fatty acids to resynthesize fat molecules similar to those previously digested. (3) These fats collect in clusters that become encased in protein. (4) The resulting large molecules of lipoprotein are called *chylomicrons*, and they make their way to the lacteals of the villi. (5) Periodic contractions of smooth muscles in the villi help empty the lacteals into the

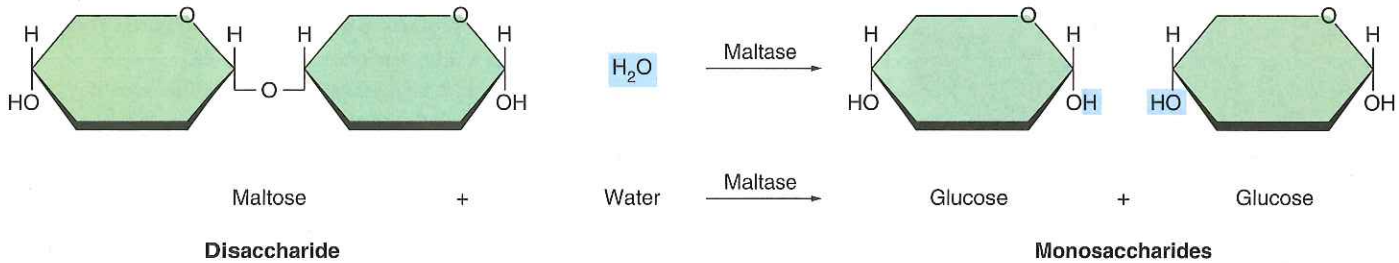


FIGURE 17.39 Digestion breaks down complex carbohydrates into disaccharides, which are then broken down into monosaccharides, which are small enough for intestinal villi to absorb. The monosaccharides then enter the bloodstream.

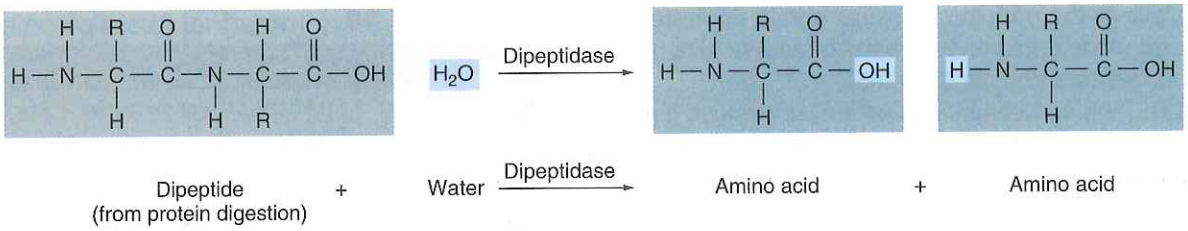


FIGURE 17.40 The amino acids that result from dipeptide digestion are absorbed by intestinal villi and enter the blood.

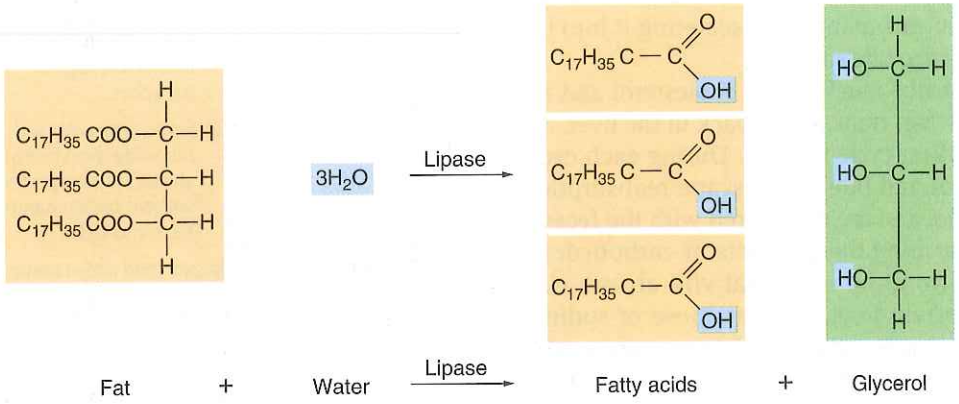


FIGURE 17.41 Fatty acids and glycerol result from fat digestion. Intestinal villi absorb them, and most are resynthesized into fat molecules before they enter the lacteals.

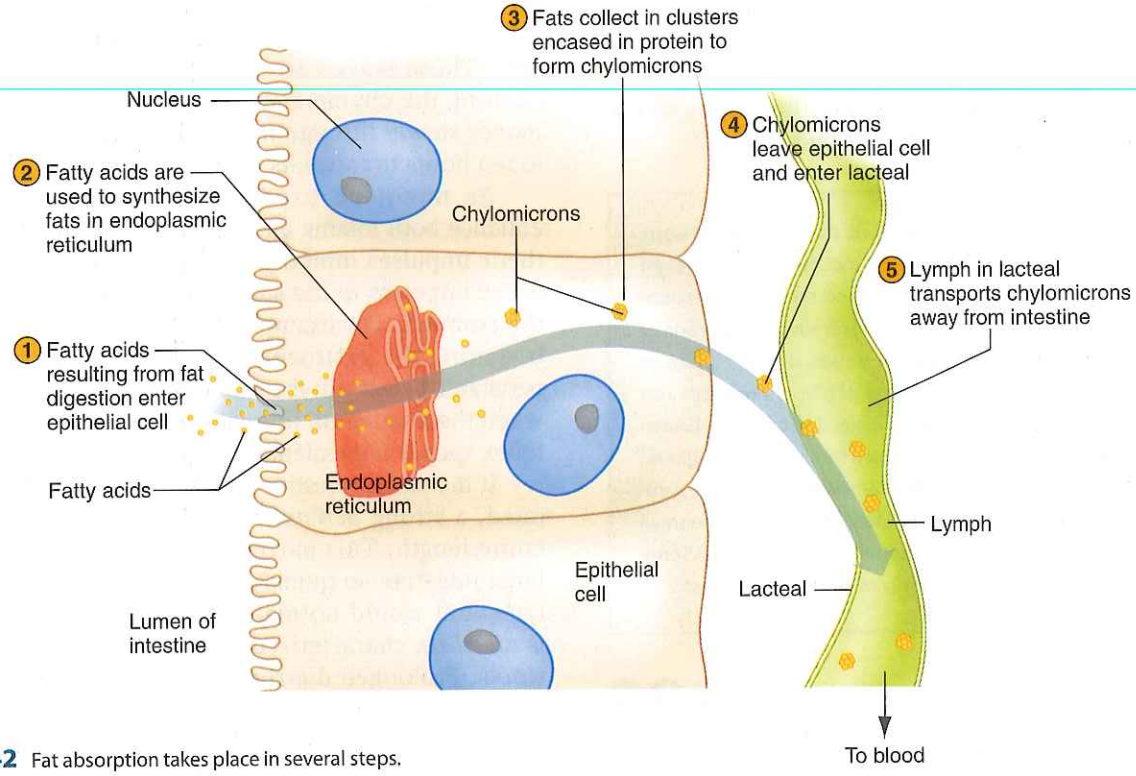


FIGURE 17.42 Fat absorption takes place in several steps.

cysterna chyli (see fig. 16.6*a*, p. 619), which is an expansion of the thoracic duct. The lymph carries the chylomicrons to the bloodstream (fig. 17.42).

Chylomicrons in the blood transport dietary fats to muscle and adipose cells. Similarly, very-low-density lipoprotein (VLDL) molecules, produced in the liver, transport triglycerides synthesized from excess dietary carbohydrates. As VLDL molecules reach adipose cells, an enzyme, *lipoprotein lipase,* catalyzes reactions that unload their triglycerides, converting VLDL to low-density lipoprotein (LDL) molecules. Because most of the triglycerides have been removed, LDL molecules have a higher cholesterol content than the original VLDL

molecules. Cells in the peripheral tissues obtain cholesterol by using receptor-mediated endocytosis to remove LDL particles from plasma (see chapter 3, p. 105).

Unlike LDL, which delivers cholesterol to tissues, high-density lipoprotein (HDL) removes cholesterol from tissues and delivers it to the liver. The liver produces the basic HDL framework and secretes HDL molecules into the bloodstream. Circulating HDL picks up cholesterol from peripheral tissues and returns it to the liver, where it enters cells by receptor-mediated endocytosis. The liver disposes of the cholesterol it obtains in this manner by secreting it into bile or by using it to synthesize bile salts.

The intestine reabsorbs much of the cholesterol and bile salts in bile, which are then transported back to the liver, and the secretion-reabsorption cycle repeats. During each cycle, some of the cholesterol and bile salts escape reabsorption, reach the large intestine, and are eliminated with the feces.

In addition to absorbing the products of carbohydrate, protein, and fat digestion, the intestinal villi absorb electrolytes and water. Certain ions, such as those of sodium, potassium, chloride, nitrate, and bicarbonate, are readily absorbed. Certain others, including ions of calcium, magnesium, and sulfate, are poorly absorbed.

Electrolytes are usually absorbed by diffusion and active transport, and water by osmosis. Thus, even though the intestinal contents may be hypertonic to the epithelial cells at first, as nutrients and electrolytes are absorbed, the contents become slightly hypotonic to the cells. Then, water follows the nutrients and electrolytes into the villi by osmosis. Table 17.10 summarizes the absorption process.

TABLE 17.10 | Intestinal Absorption of Nutrients

Nutrient	Absorption Mechanism	Means of Transport
Monosaccharides	Facilitated diffusion and active transport	Blood in capillaries
Amino acids	Active transport	Blood in capillaries
Fatty acids and glycerol	Facilitated diffusion of glycerol; diffusion of fatty acids into cells	
	(a) Most fatty acids are resynthesized into fats and incorporated in chylomicrons for transport.	Lymph in lacteals
	(b) Some fatty acids with relatively short carbon chains are transported without being changed back into fats.	Blood in capillaries
Electrolytes	Diffusion and active transport	Blood in capillaries
Water	Osmosis	Blood in capillaries

In *malabsorption* the small intestine digests, but does not absorb, some nutrients. Symptoms of malabsorption include diarrhea, weight loss, weakness, vitamin deficiencies, anemia, and bone demineralization. Causes of malabsorption include surgical removal of a portion of the small intestine, obstruction of lymphatic vessels due to a tumor, or interference with the production and release of bile as a result of liver disease. Another cause of malabsorption is a reaction to *gluten*. Gluten is a composite of two types of proteins that are found in certain grains, such as wheat, barley, and rye. This condition, called *celiac disease*, damages or even destroys microvilli, reducing the surface area of the small intestine, preventing adequate absorption of some nutrients. Some grocery stores and restaurants offer a variety of gluten-free products.

PRACTICE

45 Which substances resulting from digestion of carbohydrate, protein, and fat molecules does the small intestine absorb?

46 Which ions does the small intestine absorb?

47 Describe how fatty acids are absorbed and transported.

Movements of the Small Intestine

The small intestine carries on mixing movements and peristalsis, like the stomach. In the major mixing movement—segmentation—periodic small, ringlike contractions cut the chyme into segments and move it back and forth. Segmentation also slows the movement of chyme through the small intestine.

Peristaltic waves propel chyme through the small intestine. These waves are usually weak, and they stop after pushing the chyme a short distance. Consequently, chyme moves slowly through the small intestine, taking from three to ten hours to travel its length.

As might be expected, parasympathetic impulses enhance both mixing and peristaltic movements, and sympathetic impulses inhibit them. Reflexes involving parasympathetic impulses to the small intestine sometimes originate in the stomach. For example, food distends the stomach wall, triggering the gastroenteric reflex, which greatly increases peristaltic activity in the small intestine. Another reflex begins when the duodenum fills with chyme, stretching its wall. This reflex speeds movement through the small intestine.

If the small intestine wall becomes overdistended or irritated, a strong *peristaltic rush* may pass along the organ's entire length. This movement sweeps the contents into the large intestine so quickly that water, nutrients, and electrolytes that would normally be absorbed are not. The result is *diarrhea*, characterized by frequent defecation and watery stools. Prolonged diarrhea causes imbalances in water and electrolyte concentrations.

At the distal end of the small intestine, the **ileocecal sphincter** joins the small intestine's ileum to the large intestine's cecum. Normally, this sphincter remains constricted, preventing the contents of the small intestine from entering the large intestine, and at the same time keeping the contents of the large intestine from backing up into the ileum. However, after a meal, a gastroileal reflex increases peristalsis in the ileum and relaxes the sphincter, forcing some of the contents of the small intestine into the cecum.

PRACTICE

48 Describe the movements of the small intestine.

49 What is a peristaltic rush?

50 What stimulus relaxes the ileocecal sphincter?

17.10 LARGE INTESTINE

The **large intestine** is so named because its diameter is greater than that of the small intestine. This part of the alimentary canal is about 1.5 meters long, and it begins in the lower right side of the abdominal cavity where the ileum joins the cecum. From there, the large intestine ascends on the right side, crosses obliquely to the left, and descends into the pelvis. At its distal end, it opens to the outside of the body as the anus.

The large intestine absorbs ingested water and electrolytes remaining in the alimentary canal. Additionally it reabsorbs and recycles water and remnants of digestive secretions. The large intestine also forms and stores feces.

Parts of the Large Intestine

The large intestine consists of the cecum, the colon, the rectum, and the anal canal. Figures 17.43 and 17.44 and reference plates 11, 12, 18, and 25 (p. 48, 49, 52, and 57) depict the large intestine.

The **cecum,** at the beginning of the large intestine, is a dilated, pouchlike structure that hangs slightly inferior to the ileocecal opening. Projecting downward from it is a narrow tube with a closed end called the **appendix.** The human appendix has no known digestive function. However, it contains lymphatic tissue. One suggested function of the appendix is that it stores useful bacteria that might otherwise be lost when a person has diarrhea.

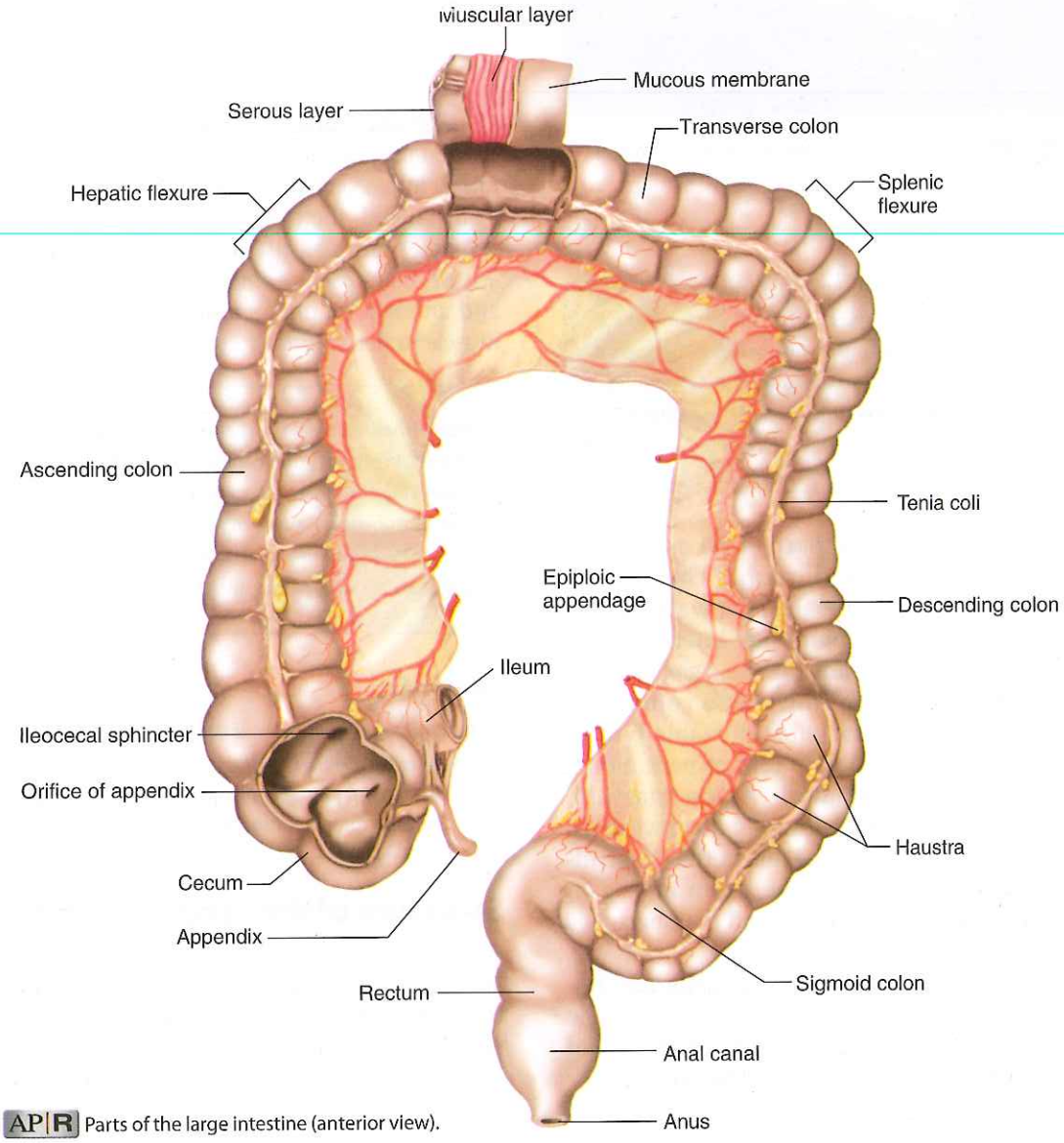


FIGURE 17.43 AP|R Parts of the large intestine (anterior view).

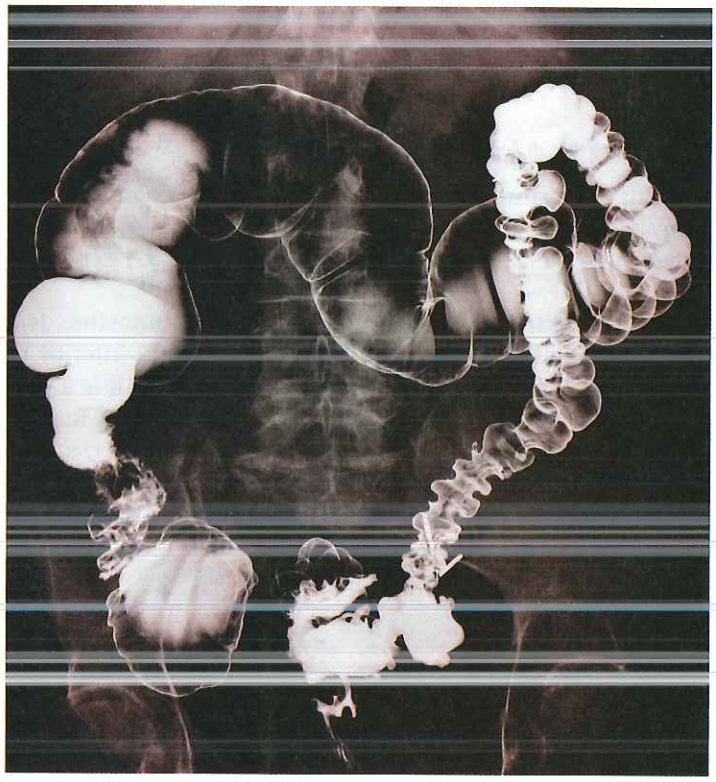


FIGURE 17.44 AP|R Radiograph of the large intestine containing a radiopaque substance that the patient ingested.

In *appendicitis,* the appendix becomes inflamed and infected. Surgery is often required to remove the appendix before it ruptures. If it does rupture, this may allow contents of the large intestine to enter the abdominal cavity and cause a serious infection of the peritoneum called *peritonitis.*

The **colon** is divided into four parts—the ascending, transverse, descending, and sigmoid colons. The **ascending colon** begins at the cecum and extends upward against the posterior abdominal wall to a point just inferior to the liver. There it turns sharply to the left (as the right colic, or hepatic, flexure) and becomes the **transverse colon.** The transverse colon is the longest and most movable part of the large intestine. It is suspended by a fold of peritoneum and sags in the middle below the stomach. As the transverse colon approaches the spleen, it turns abruptly downward (as the left colic, or splenic, flexure) and becomes the **descending colon.** At the brim of the pelvis, the colon makes an S-shaped curve, called the **sigmoid colon,** and then becomes the rectum.

The **rectum** lies next to the sacrum and generally follows its curvature. The peritoneum firmly attaches it to the sacrum, and it ends about 5 centimeters inferior to the tip of the coccyx, where it becomes the anal canal (fig. 17.45).

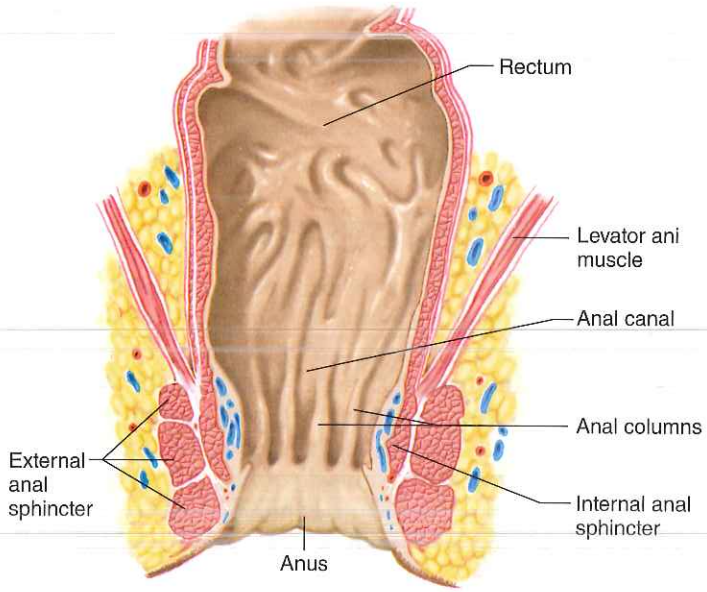


FIGURE 17.45 The rectum and the anal canal are at the distal end of the alimentary canal.

The **anal canal** is formed by the last 2.5 to 4.0 centimeters of the large intestine. The mucous membrane in the canal is folded into a series of six to eight longitudinal *anal columns.* At its distal end, the canal opens to the outside as the **anus.** Two sphincter muscles guard the anus—an *internal anal sphincter muscle,* composed of smooth muscle under involuntary control, and an *external anal sphincter muscle,* composed of skeletal muscle under voluntary control.

Hemorrhoids are enlarged and inflamed branches of the rectal vein in the anal columns that cause intense itching, sharp pain, and sometimes bright red bleeding. The hemorrhoids may be internal or bulge out of the anus. Causes of hemorrhoids include anything that puts prolonged pressure on the delicate rectal tissue, including obesity, pregnancy, constipation, diarrhea, and liver disease.

PRACTICE

51 What is the general function of the large intestine?

52 Describe the parts of the large intestine.

53 Distinguish between the internal sphincter muscle and the external sphincter muscle of the anus.

Structure of the Large Intestinal Wall

The wall of the large intestine includes the same types of tissues found in other parts of the alimentary canal but also has some unique features (fig. 17.46). The large intestinal wall lacks the villi and plicae circularis characteristic of the small intestine. The layer of longitudinal muscle fibers is not

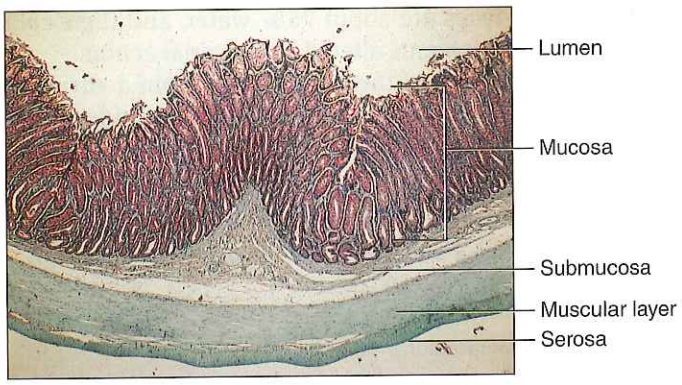


FIGURE 17.46 AP|R Light micrograph of the large intestinal wall (64×).

uniformly distributed throughout the large intestinal wall. Instead the fibers are in three distinct bands (teniae coli) that extend the entire length of the colon. These bands exert tension lengthwise on the wall, creating a series of pouches (haustra). The large intestinal wall also has small collections of fat (epiploic appendages) in the serosa on its outer surface (see fig. 17.43).

Functions of the Large Intestine

The large intestine has little or no digestive function. This is in contrast to the small intestine, which secretes digestive enzymes and absorbs the products of digestion. However, the mucous membrane that forms the inner lining of the large intestine includes many tubular glands. Structurally, these glands are similar to those of the small intestine, but they are composed almost entirely of goblet cells (fig. 17.47). Consequently, mucus is the large intestine's only significant secretion.

Mechanical stimulation from chyme and parasympathetic impulses control the rate of mucus secretion. In both cases, the goblet cells respond by increasing mucus production, which, in turn, protects the intestinal wall against the abrasive action of materials passing through it. Mucus also holds particles of fecal matter together, and, because it is alkaline, mucus helps control the pH of the large intestinal contents. This is important because acids are sometimes released from the feces as a result of bacterial activity.

Chyme entering the large intestine usually has few nutrients remaining in it and mostly consists of materials not digested or absorbed in the small intestine. It also contains water, electrolytes, mucus, and bacteria.

Absorption in the large intestine is normally limited to water and electrolytes, and this usually occurs in the proximal half of the tube. Electrolytes such as sodium ions can be absorbed by active transport, while water follows passively, crossing the mucosal layer by osmosis. About 90% of the water that enters the large intestine is absorbed, and little sodium or water is lost in the feces.

The many bacteria that normally inhabit the large intestine, called *intestinal flora*, break down some of the molecules that escape the actions of human digestive enzymes. For instance, cellulose, a complex carbohydrate in food of plant origin, passes through the alimentary canal almost unchanged, but colon bacteria can break down cellulose and use it as an energy source. These bacteria, in turn, synthesize vitamins, such as K, B_{12}, thiamine, and riboflavin, which the intestinal mucosa absorbs. Bacterial actions in the large intestine may produce intestinal gas (flatus).

Intestinal gas contains nitrogen and oxygen taken in while breathing and eating, plus variable amounts of methane (CH_4), carbon dioxide (CO_2), and hydrogen contributed from the bacterial fermentation of undigested food. The characteristic odor comes from bacterial action on the nitrogen and sulfur in proteins, which yields pungent-smelling ammonia (NH_3) and foul hydrogen sulfide (H_2S). Most people release a half liter of intestinal gas a day. Foods rich in sulfur-containing amino acids make intestinal gas more foul. These include beans, broccoli, bran, brussels sprouts, cabbage, cauliflower, and onions.

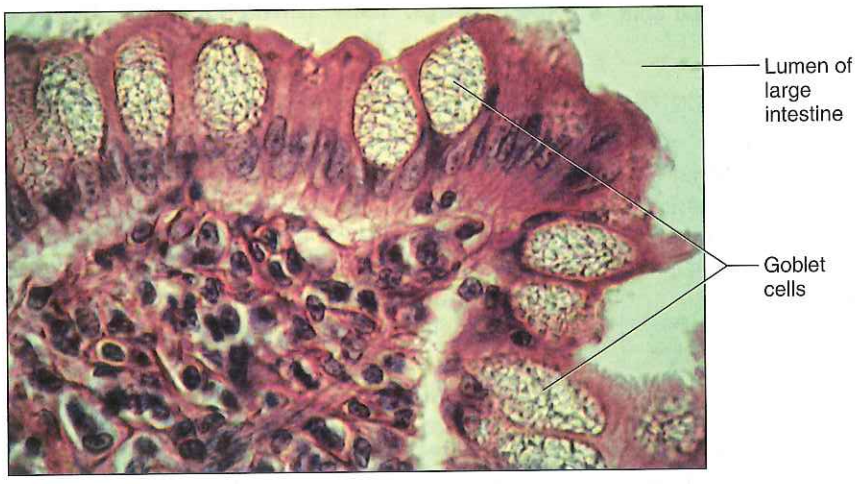


FIGURE 17.47 Light micrograph of the large intestinal mucosa (560×).

Q: *Note the many goblet cells in the mucosa of the large intestine. Why might it be important that there are so many more of these cells in the large intestinal wall than in the small intestinal wall?*

Answer can be found in Appendix G on page 938.

PRACTICE

54 How does the structure of the large intestine differ from that of the small intestine?

55 Which substances does the large intestine absorb?

56 Which useful substances do bacteria inhabiting the large intestine produce?

Movements of the Large Intestine

The movements of the large intestine—mixing and peristalsis—are similar to those of the small intestine, although usually slower. The mixing movements break the fecal matter into segments and turn it so that all portions are exposed to the intestinal mucosa. This helps absorb water and electrolytes.

The peristaltic waves of the large intestine are different from those of the small intestine. Instead of occurring frequently, they happen only two or three times each day. These waves produce *mass movements* in which a large section of the intestinal wall constricts vigorously, forcing the intestinal contents to move toward the rectum. Typically, mass movements follow a meal, as a result of the gastrocolic reflex initiated in the small intestine. Irritation of the intestinal mucosa can also trigger such movements. For instance, a person suffering from an inflamed colon (colitis) may experience frequent mass movements. Clinical Application 17.5 examines conditions affecting the large intestine.

When it is appropriate to defecate, a person usually can initiate a *defecation reflex* by holding a deep breath and contracting the abdominal wall muscles. This action increases the internal abdominal pressure and forces feces into the rectum. As the rectum fills, its wall distends, triggering the defecation reflex, which stimulates peristaltic waves in the descending colon. The internal anal sphincter relaxes. At the same time, other reflexes involving the sacral region of the spinal cord strengthen the peristaltic waves, lower the diaphragm, close the glottis, and contract the abdominal wall muscles. These actions further increase the internal abdominal pressure and squeeze the rectum. The external anal sphincter is signaled to relax, and the feces are forced to the outside. A person can voluntarily inhibit defecation by contracting the external anal sphincter.

Hirschsprung disease causes extreme, chronic constipation and abdominal distension. In this condition, the part of the large intestine distal to the distension lacks innervation, so the person does not feel the urge to defecate. The problem begins in the embryo, when a mutant gene prevents neurons from migrating to this portion of the gastrointestinal tract. Surgery may be used to treat the disease.

Feces

Feces (fe'sēz) are composed of materials not digested or absorbed, along with water, electrolytes, mucus, and bacteria. Usually the feces are about 75% water, and their color derives from bile pigments altered by bacterial action.

The pungent odor of the feces results from a variety of compounds that bacteria produce. These compounds include phenol, hydrogen sulfide, indole, skatole, and ammonia.

PRACTICE

57 How does peristalsis in the large intestine differ from peristalsis in the small intestine?

58 List the major events of defecation.

59 Describe the composition of feces.

17.11 LIFE-SPAN CHANGES

Changes to the digestive system associated with the passing years are slow and slight, so most people can enjoy eating a variety of foods as they grow older. Maintaining healthy teeth is vital to obtaining adequate nutrition. This requires frequent dental checkups, cleanings, and plaque removal, plus care of the gums. Tooth loss due to periodontal disease becomes more likely after age thirty-five.

Despite regular dental care, some signs of aging may affect the teeth. The enamel often thins from years of brushing, teeth grinding, and eating acidic foods. Thinning enamel may make the teeth more sensitive to hot and cold foods. At the same time, the cementum may thicken. The dentin heals more slowly and enlarges as the pulp shrinks. Loss of neurons in the pulp may make it more difficult to notice tooth decay. The gums recede, creating more pockets to harbor the bacteria whose activity contributes to periodontal disease. The teeth may loosen as the bones of the jaw weaken. On a functional level, older people sometimes do not chew their food thoroughly, swallowing larger chunks of food that may present a choking hazard.

A common complaint of older individuals is "dry mouth," or xerostomia. This condition is not a normal part of aging—studies have shown that the oldest healthy people make just as much saliva as healthy younger people. Dry mouth is common, however, because it is a side effect of more than 400 medications, many of which are more likely to be taken by older persons. These include antidepressants, antihistamines, and drugs that treat cancer or hypertension. In addition, radiation and chemotherapy used to treat cancer can cause mouth sores and tooth decay.

The gastrointestinal tract gradually becomes less efficient with age. Slowing peristalsis may cause frequent heartburn as food backs up into the esophagus. The stomach lining thins with age, and secretion of hydrochloric acid, pepsin, and intrinsic factor decline. Exit of chyme from the stomach slows. Overall, these changes may affect the rate at which certain medications are absorbed.

The small intestine is the site of absorption of nutrients, so it is here that noticeable signs of aging on digestion arise. Subtle shifts in the microbial species that inhabit the small intestine alter the rates of absorption of particular nutrients.

17.5 CLINICAL APPLICATION

Disorders of the Large Intestine

The large intestine (colon) is the source of familiar digestive discomforts as well as more serious disorders.

Diverticulosis and Inflammatory Bowel Disease

In diverticulosis, parts of the intestinal wall weaken, and the inner mucous membrane protrudes through. If chyme accumulates in the outpouching and becomes infected (diverticulitis), antibiotics or surgical removal of the area may become necessary. Lack of dietary fiber may increase the risk of developing diverticulosis.

Inflammatory bowel disease is a group of disorders that includes ulcerative colitis and Crohn's disease. The disorders differ by the site and extent of inflammation and ulceration of the intestines. In the United States, about 100,000 people suffer the abdominal cramps and diarrhea of ulcerative colitis, and 500,000 individuals have similar symptoms of Crohn's disease.

Ulcerative colitis affects the mucosa and submucosa of the distal large intestine and the rectum. In about 25% of cases, the disease extends no farther than the rectum. Bloody diarrhea and cramps may last for days or weeks and may recur frequently or only rarely. The severe diarrhea leads to weight loss and electrolyte imbalances and may develop into colon cancer or affect other organs, including the skin, eyes, or liver. The inflamed and ulcerous tissue is continuous.

Crohn's disease is more extensive than ulcerative colitis, infiltrating the small and large intestines and penetrating all tissue layers. In contrast to the uniformity of ulcerative colitis, affected portions of intestine in Crohn's disease are interspersed with unaffected areas, producing a "cobblestone" effect after many years. The ileum and cecum are affected in about 40% of individuals, only the small intestine is involved in 30% of cases, and only the large intestine is involved in 25% of cases. Rarely, the disease affects more proximal structures of the gastrointestinal tract. The diarrhea is often not bloody, and complications such as cancer are rare.

Overall, about 20% of people with inflammatory bowel disease have symptoms that fall between the descriptions of ulcerative colitis and Crohn's disease, and are considered to have "indeterminate colitis." Autoimmunity, infection, or a genetic predisposition may contribute to causing inflammatory bowel disease. Surgery and drugs that suppress the immune response are used to treat some cases of inflammatory bowel disease.

Colorectal Cancer

Cancer of the large intestine or rectum, known as *colorectal cancer*, is the fourth most prevalent cancer in the United States and the second most common cause of cancer death. More than 30,000 new cases are diagnosed each year, and more than 56,000 people die of the condition. It tends to run in families. Symptoms of colorectal cancer include

- a change in frequency or consistency of bowel movements
- blood in the feces
- a narrowing of feces
- abdominal discomfort or pain
- weight loss
- fatigue
- unexplained vomiting

Diagnostic tests, described in table 17A, may detect colorectal cancer. These tests are of two general types—the fecal occult blood test performed on a stool sample and imaging the large intestinal wall. An experimental test screens the DNA from cells in feces for mutations associated with colorectal cancer.

Fiberoptic colonoscopy is a test commonly performed on people over age 50, when the risk of colorectal cancer increases. Under sedation, a flexible lit tube is inserted into the rectum, and polyps and tumors are identified and removed. People with a family history of colon cancer should be screened at an earlier age. Fiberoptic colonoscopy takes less than an hour. A newer procedure, computed tomographic colonography (virtual colonoscopy), requires the same preparatory bowel cleansing, but does not require sedation and is faster. However, if a lesion is detected, the more invasive approach must be used to remove the suspicious tissue.

Treatment for colorectal cancer removes the affected tissue. If a large portion of the intestine is removed, surgery is used to construct a new opening to release feces. The free end of the intestine is attached to an opening created through the skin of the abdomen, and a bag is attached to the opening to collect the fecal matter. This procedure is called a colostomy.

For people who have a certain inherited form of colon cancer (familial adenomatous polyposis), nonsteroidal anti-inflammatory drugs (NSAIDs) called Cox-2 inhibitors are used to treat the disease and may even help to prevent it in those identified by a genetic test to be at high risk. However, these drugs increase the risk of heart disease in certain individuals. ■

TABLE 17A | Diagnostic Tests for Colorectal Cancer

Diagnostic Test	Description
Digital rectal exam	Physician palpates large intestine and rectum
Double-contrast barium enema	X-ray exam following ingestion of contrast agent highlights blockages in large intestine
Fecal occult blood test	Blood detected in feces sample
Colorectal cancer gene test (experimental)	Mutations associated with colorectal cancer detected in DNA of cells shed with feces
Sigmoidoscopy	Endoscope views rectum and lower colon
Colonoscopy	Endoscope views rectum and entire colon

With age, the small intestine becomes less efficient at absorbing vitamins A, D, and K and the mineral zinc. This raises the risk of deficiency symptoms—effects on skin and vision due to a lack of vitamin A; weakened bones from inadequate vitamin D; impaired blood clotting seen in vitamin K deficiency; and slowed healing, decreased immunity, and altered taste evidenced in zinc deficiency.

Many people who have inherited lactose intolerance begin to notice the telltale cramping after eating dairy foods in the middle years. They must be careful that by avoiding dairy products, they do not also lower their calcium intake. Less hydrochloric acid also adversely affects the absorption of calcium, as well as iron. Too little intrinsic factor may lead to vitamin B_{12} deficiency anemia.

The lining of the large intestine changes too, thinning and containing less smooth muscle and mucus. A dampening of the responsiveness of the smooth muscle to neural stimulation slows peristalsis, ultimately causing constipation. Compounding this common problem is a loss of elasticity in the walls of the rectum and declining strength and responsiveness of the internal and external sphincters.

The accessory organs to digestion age too, but not necessarily in ways that affect health. Both the pancreas and the liver are large organs with cells to spare, so a decline in their secretion abilities does not usually hamper digestion. Only 10% of the pancreas and 20% of the liver are required to digest foods. However, the liver may not be able to detoxify certain medications as quickly as it once did. The gallbladder becomes less sensitive to cholecystokinin, but in a classic feedback response, cells of the intestinal mucosa secrete more of it into the bloodstream, and the gallbladder continues to be able to contract. The bile ducts widen in some areas, but the end of the bile duct narrows as it approaches the small intestine. As long as gallstones do not become entrapped in the ducts, the gallbladder generally functions well into the later years.

PRACTICE

60 Describe the effects of aging on the teeth.

61 Which conditions might be caused by the slowing of peristalsis in the digestive tract that occurs with aging?

CHAPTER SUMMARY

17.1 INTRODUCTION (PAGE 649)

Digestion is the process of mechanically and chemically breaking down foods so that they can be absorbed. The digestive system consists of an alimentary canal and several accessory organs that carry out the processes of ingestion, propulsion, digestion, absorption, and defecation.

17.2 GENERAL CHARACTERISTICS OF THE ALIMENTARY CANAL (PAGE 649)

Regions of the alimentary canal perform specific functions.

1. Structure of the wall
   a. The wall consists of four layers.
   b. These layers include the mucosa, submucosa, muscular layer, and serosa.
2. Movements of the tube
   a. Motor functions include mixing and propelling movements.
   b. Peristalsis is responsible for propelling movements.
   c. The wall of the tube undergoes receptive relaxation just ahead of a peristaltic wave.
3. Innervation of the tube
   a. The tube is innervated by branches of the sympathetic and parasympathetic divisions of the autonomic nervous system.
   b. Parasympathetic impulses generally increase digestive activities; sympathetic impulses generally inhibit digestive activities.
   c. Sympathetic impulses contract certain sphincter muscles, controlling movement of digesting food through the alimentary canal.

17.3 MOUTH (PAGE 653)

The mouth is adapted to receive food and begin digestion by mechanically breaking up solid particles (mastication). It also serves as an organ of speech and sensory perception.

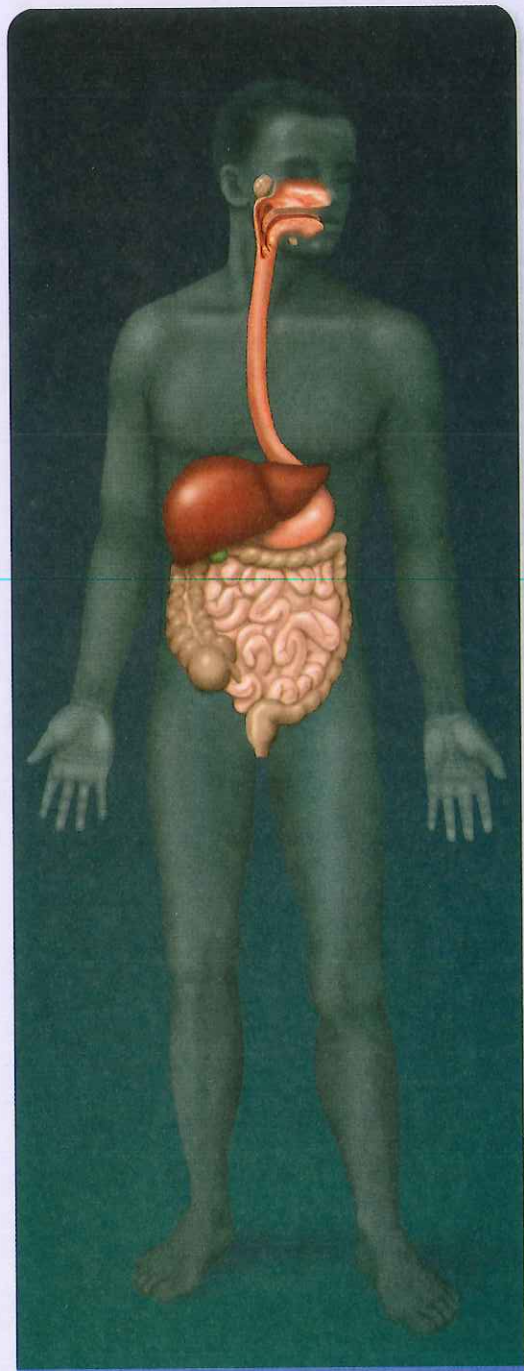
1. Cheeks and lips
   a. Cheeks form the lateral walls of the mouth.
   b. Lips are highly mobile and have a variety of sensory receptors useful in judging the characteristics of food.
2. Tongue
   a. The tongue is a thick, muscular organ that mixes food with saliva and moves it toward the pharynx.
   b. The rough surface of the tongue handles food and has taste buds.
   c. Lingual tonsils are located on the root of the tongue.
3. Palate
   a. The palate comprises the roof of the mouth and includes hard and soft portions.
   b. The soft palate, including the uvula, closes the opening to the nasal cavity during swallowing.
   c. Palatine tonsils are located on either side of the tongue in the back of the mouth.
   d. Tonsils consist of lymphatic tissues.
4. Teeth
   a. Two sets of teeth develop in sockets of the mandibular and maxillary bones.
   b. There are twenty primary and thirty-two secondary teeth.
   c. Teeth mechanically break food into smaller pieces, increasing the surface area exposed to digestive actions.
   d. Different types of teeth are adapted to handle foods in different ways, such as biting, grasping, or grinding.
   e. Each tooth consists of a crown and root and is composed of enamel, dentin, pulp, nerves, and blood vessels.
   f. A tooth is attached to the alveolar process by the periodontal ligament.

17.4 SALIVARY GLANDS (PAGE 657)

Salivary glands secrete saliva, which moistens food, helps bind food particles, begins chemical digestion of carbohydrates, makes taste possible, helps cleanse the mouth, and regulates pH in the mouth.

1. Salivary secretions
   a. Salivary glands include serous cells that secrete digestive enzymes and mucous cells that secrete mucus.
   b. Parasympathetic impulses stimulate the secretion of a large volume of watery saliva.

INNERCONNECTIONS ●●● Digestive System



Digestive System

The digestive system ingests, digests, and absorbs nutrients for use by all body cells.

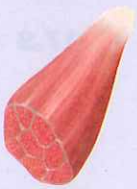
Integumentary System

Vitamin D activated in the skin plays a role in absorption of calcium from the digestive tract.

Skeletal System

Bones are important in mastication. Calcium absorption is necessary to maintain bone matrix.

Muscular System



Muscles are important in mastication, swallowing, and the mixing and moving of digestion products through the gastrointestinal tract.

Nervous System

The nervous system can influence digestive system activity.

Endocrine System

Hormones can influence digestive system activity.

Cardiovascular System

The bloodstream carries absorbed nutrients to all body cells.

Lymphatic System

The lymphatic system plays a major role in the absorption of fats.

Respiratory System

The digestive system and the respiratory system share common anatomical structures.

Urinary System

The kidneys and liver work together to activate vitamin D.

Reproductive System

In a woman, nutrition is essential for conception and normal development of an embryo and fetus.

2. Major salivary glands
   a. The parotid glands are the largest, and they secrete saliva rich in amylase.
   b. The submandibular glands in the floor of the mouth produce viscous saliva containing amylase.
   c. The sublingual glands in the floor of the mouth primarily secrete mucus.

17.5 PHARYNX AND ESOPHAGUS (PAGE 659)

The pharynx and esophagus serve as passageways.

1. Structure of the pharynx
   a. The pharynx is divided into a nasopharynx, oropharynx, and laryngopharynx.
   b. The muscular walls of the pharynx contain fibers in circular and longitudinal groups.
2. Swallowing mechanism
   a. Swallowing (deglutition) occurs in three stages.
      (1) Food is mixed with saliva and forced into the pharynx.
      (2) Involuntary reflex actions move the food into the esophagus.
   b. Swallowing reflexes momentarily inhibit breathing.
   c. Peristalsis transports food in the esophagus to the stomach.
3. Esophagus
   a. The esophagus passes through the mediastinum and penetrates the diaphragm.
   b. Circular muscle fibers at the distal end of the esophagus help prevent regurgitation of food from the stomach.

17.6 STOMACH (PAGE 663)

The stomach receives food, mixes it with gastric juice, carries on a limited amount of absorption, and moves food into the small intestine.

1. Parts of the stomach
   a. The stomach is divided into the cardia, fundus, body, and pylorus.
   b. The lower esophageal sphincter serves as a valve between the esophagus and the stomach.
   c. The pyloric sphincter serves as a valve between the stomach and the small intestine.
2. Gastric secretions
   a. Gastric glands secrete gastric juice.
   b. Gastric juice contains pepsin(begins digestion of proteins), hydrochloric acid, lipase, and intrinsic factor.
3. Regulation of gastric secretions
   a. Parasympathetic impulses and the hormone gastrin enhance gastric secretion.
   b. The three stages of gastric secretion are the cephalic, gastric, and intestinal phases.
   c. The presence of food in the small intestine reflexly inhibits gastric secretions.
4. Gastric absorption
   a. The stomach is not well adapted for absorption.
   b. A few substances such as water and other small molecules are absorbed through the stomach wall.
5. Mixing and emptying actions
   a. As the stomach fills, its wall stretches, but its internal pressure remains unchanged.
   b. Mixing movements aid in producing chyme; peristaltic waves move chyme into the pylorus.
   c. The muscular wall of the pylorus regulates chyme movement into the small intestine.
   d. The rate of emptying depends on the fluidity of the chyme and the type of food present.
   e. The upper part of the small intestine fills, and an enterogastric reflex inhibits peristalsis in the stomach.
   f. Vomiting results from a complex reflex that has many stimuli.

17.7 PANCREAS (PAGE 668)

The pancreas is closely associated with the duodenum.

1. Structure of the pancreas
   a. It produces pancreatic juice secreted into a pancreatic duct.
   b. The pancreatic duct leads to the duodenum.
2. Pancreatic juice
   a. Pancreatic juice contains enzymes that can split carbohydrates, proteins, fats, and nucleic acids.
   b. Pancreatic juice has a high bicarbonate ion concentration that helps neutralize chyme and causes the intestinal contents to be alkaline.
3. Regulation of pancreatic secretion
   a. Secretin from the duodenum stimulates the release of pancreatic juice that contains few digestive enzymes but has a high bicarbonate ion concentration.
   b. Cholecystokinin from the intestinal wall stimulates the release of pancreatic juice that has a high concentration of digestive enzymes.

17.8 LIVER (PAGE 670)

The liver is located in the upper-right quadrant of the abdominal cavity.

1. Liver structure
   a. The liver is a highly vascular organ, enclosed in a fibrous capsule, and divided into lobes.
   b. Each lobe consists of hepatic lobules, the functional units of the liver.
   c. Bile from the lobules is carried by bile ductules to hepatic ducts that unite to form the common hepatic duct.
2. Liver functions
   a. The liver has many functions. It metabolizes carbohydrates, lipids, and proteins; stores some substances; filters blood; destroys toxins; and secretes bile.
   b. Bile is the only liver secretion that directly affects digestion.
3. Composition of bile
   a. Bile contains bile salts, bile pigments, cholesterol, and electrolytes.
   b. Only the bile salts have digestive functions.
   c. Bile pigments are products of red blood cell breakdown.