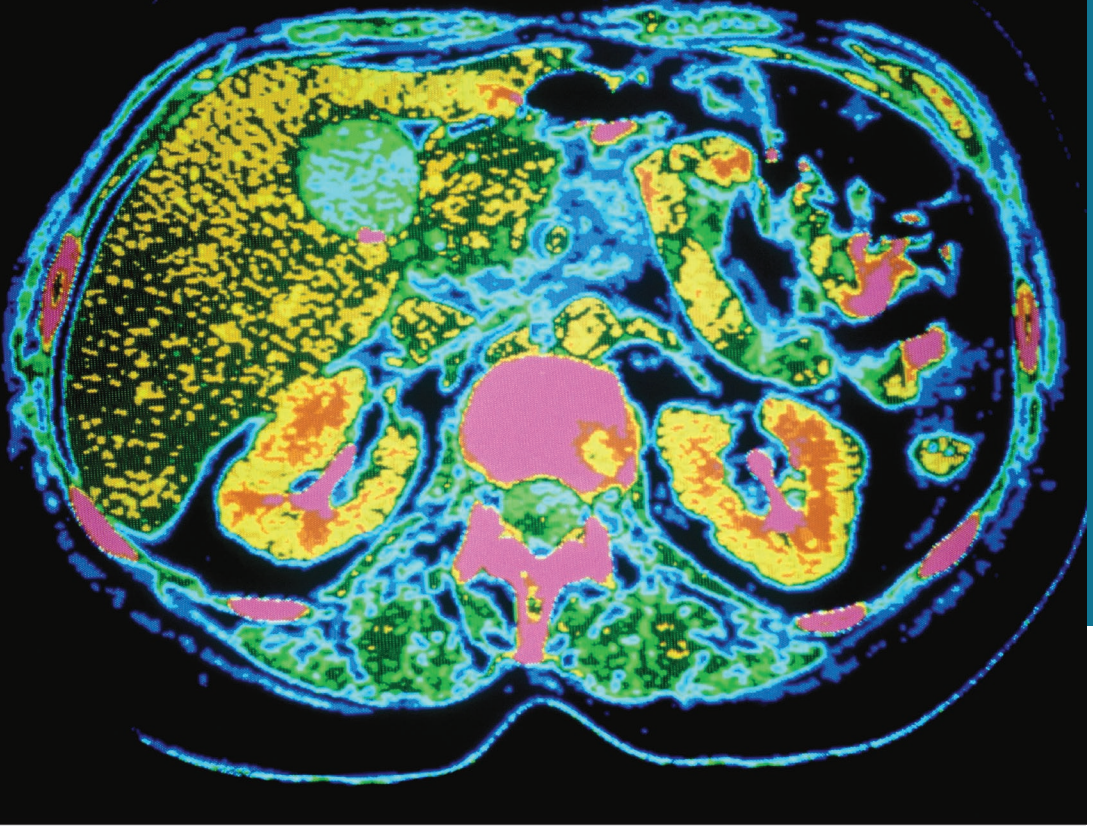


Photo: Colorized computed tomographic (CT) scan of an axial section through the abdomen. The image shows a gallstone (*pink*) obstructing the cystic duct where it leaves the gallbladder (*green circle*). The *large yellow* region around the gallbladder is the liver. A vertebra (*pink*) is visible in bottom center, with C-shaped kidneys (*yellow*) on either side. ©GJLP/Science Source

24

Digestive System

Almost everyone loves to eat, and we all must eat to stay alive. Throughout history, food and drink have provided not only nourishment but also the foundation for many social gatherings. Although it's not something we often think about while enjoying our pizza and favorite beverage, the body has an amazing digestive system that includes its own quality control and waste disposal methods.

Every cell of the body needs nourishment, yet most cells cannot leave their position in the body and travel to a food source. Therefore, the food must be converted to a usable form and delivered. To do this, the digestive system is specialized to ingest food, propel it through the digestive tract, digest it, and absorb water, electrolytes, and nutrients. The digestive process involves a choreographed mixing of food with digestive juices that include strong acids, detergent-like bile salts, and activated enzymes. The body then maximizes absorption of digested nutrients. Once these useful substances are absorbed, they are transported through the blood to cells, which use them for energy or as new molecules for building and maintaining tissues and organs. Indeed, the digestive system is the body's "meals on wheels."

Learn to Predict

Rebecca kept attributing her recurring abdominal pain to "something I ate"—and she was partly right about that. Several times during the past year, eating high-fat meals had led to episodes of serious abdominal pain. During the most recent attack, the discomfort became so intense that Rebecca went to the emergency room, where she was given medication to relieve the pain. Still, over the next few hours, her skin took on a yellowish tint, and the next morning she had diarrhea and clay-colored feces. Following lab tests and ultrasonography, a physician diagnosed gallstones and recommended the removal of Rebecca's gallbladder.

Explain how gallstones led to Rebecca's pain and other symptoms.

24.1 Anatomy of the Digestive System

LEARNING OUTCOME

After reading this section, you should be able to

A. **List the regions of the digestive tract.**

The **digestive system** consists of the digestive tract and accessory organs (figure 24.1). The **digestive tract** is a tube extending from the mouth to the anus. It is also called the **gastrointestinal** (gas′trō-in-tes′tin-ăl; **GI**) **tract** and the *alimentary canal.* The associated **accessory organs** include the salivary glands, liver, gallbladder, and pancreas. These organs are primarily glands that secrete fluids into the digestive tract.

The digestive tract and associated accessory organs include the following:

1. *Oral cavity,* including the tongue and teeth, with the salivary glands as accessory organs
2. *Pharynx*
3. *Esophagus*
4. *Stomach*
5. *Small intestine,* consisting of the duodenum, jejunum, and ileum, with the liver, gallbladder, and pancreas as accessory organs
6. *Large intestine,* including the cecum, colon, rectum, anal canal, and anus

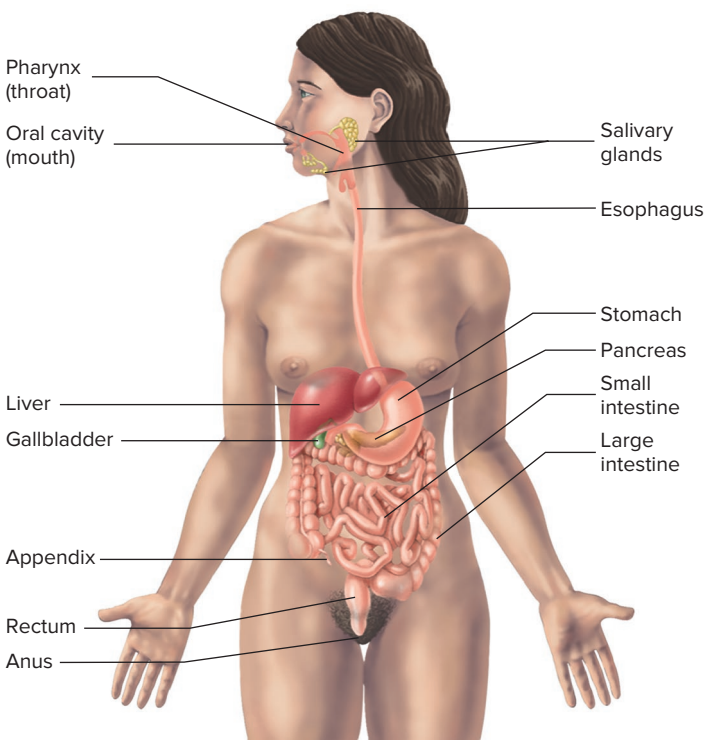


FIGURE 24.1 Digestive System
The digestive system consists of the digestive tract from the oral cavity to the anus, and the associated accessory organs: salivary glands, liver, gallbladder, and pancreas.

ASSESS YOUR PROGRESS

Answers to these questions are found in the section you have just completed. Re-read the section if you need help in answering these questions.

1. *List the regions of the digestive tract, from beginning to end.*

24.2 Functions of the Digestive System

LEARNING OUTCOMES

After reading this section, you should be able to

A. **Describe the major functions of the digestive system.**

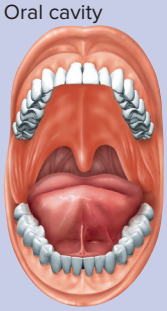
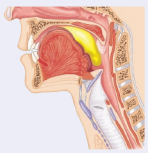
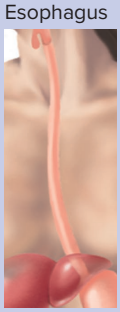
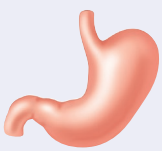
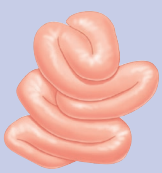
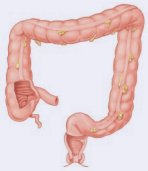
B. **State which digestive functions occur in the different regions of the digestive tract.**

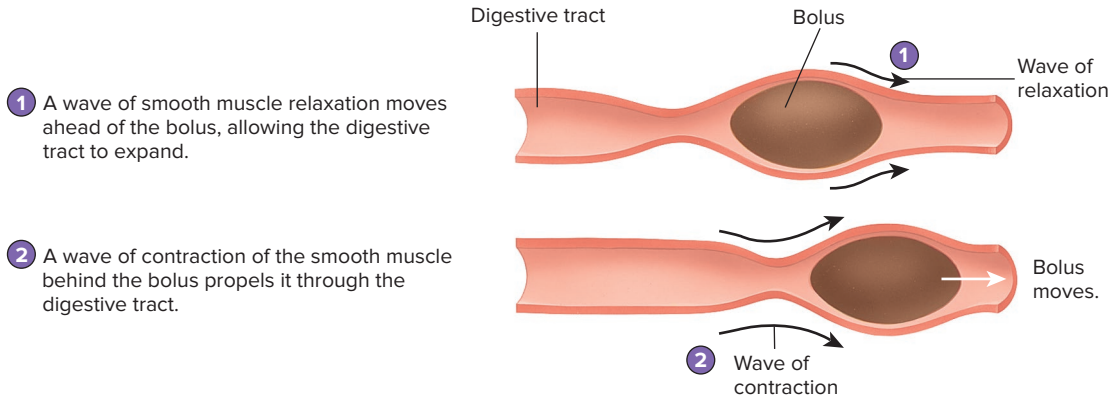
The six major functions of the digestive system are (1) ingestion and mastication, (2) propulsion and mixing, (3) secretion, (4) digestion, (5) absorption, and (6) elimination. These are listed in table 24.1 and described here:

1. *Ingestion and Mastication.* Ingestion is the intake of solid or liquid food into the stomach. The normal route of ingestion is through the oral cavity. Mastication is the process by which the teeth chew food in the mouth to begin the process of digestion. Digestive enzymes cannot easily penetrate solid food particles and can work effectively only on particle surfaces. It is vital, therefore, that solid foods be mechanically broken down by mastication into smaller particles to increase the total surface area of food for digestion.
2. *Propulsion and Mixing.* Propulsion is the movement of food from one end of the digestive tract to the other. Mixing is the movement of food back and forth in the digestive tract, without forward movement. The total time it takes food to travel the length of the digestive tract is usually about 24–36 hours. Each segment of the digestive tract is specialized to assist in moving its contents from the oral end to the anal end. The propulsive movements begin with swallowing, followed by peristalsis, and finally mass movements. Characteristics of these propulsive movements are:
   a. **Swallowing,** or *deglutition* (dē′gloo-tish′ŭn), moves liquids or a soft mass of food and liquid, called a **bolus** (bō′lŭs), from the oral cavity into the esophagus.
   b. **Peristalsis** (per-i-stal′sis; figure 24.2) propels material through most of the digestive tract. **Peristaltic** (per-i-stal′tik) **waves** are muscular contractions consisting of a wave of relaxation of the circular muscles in front of the bolus, followed by a wave of strong contraction of the circular muscles behind the bolus, which force the bolus along the digestive tract. Each peristaltic wave travels the length of the esophagus in about 10 seconds. Peristaltic waves in the small and large intestines usually travel only short distances.
   c. **Mass movements** are contractions that move material in the distal parts of the large intestine to the anus.

   Mixing contractions blend food with digestive fluids in the stomach and small intestine. These contractions aid

TABLE 24.1 Functions of the Digestive System

Organ	Functions
Oral cavity 	*Ingestion and Mastication.* Solid food and fluids are taken into the digestive tract through the oral cavity. Movement of the mandible by the muscles of mastication causes the teeth to break food into smaller pieces. The tongue and cheeks help place the food between the teeth. The food is mixed with saliva, which plays important protective roles in the oral cavity and allows the food to be tasted. *Propulsion and Mixing.* The tongue forms food into a bolus and pushes the bolus into the pharynx to begin the swallowing reflex. *Secretion.* Saliva contains mucin, bicarbonate, and water, which provide lubrication and protection of the oral cavity, and lysozyme (an enzyme that lyses cells) kills microorganisms. Amylase and lingual lipase are also released in saliva. *Digestion.* Mastication begins mechanical digestion of food. Amylase in saliva begins carbohydrate (starch) digestion. *Absorption.* There is no absorption of nutrients in the mouth, although some drugs can be absorbed across the oral mucosa.
Pharynx 	*Propulsion and Mixing.* The involuntary phase of swallowing moves the bolus from the oral cavity to the esophagus. Materials are prevented from entering the nasal cavity by the soft palate and kept out of the lower respiratory tract by the epiglottis and vestibular folds. *Secretion.* Mucus provides lubrication.
Esophagus 	*Propulsion and Mixing.* Peristaltic contractions move the bolus from the pharynx to the stomach. The lower esophageal sphincter limits reflux of the stomach contents into the esophagus. *Secretion.* Mucus provides lubrication and protects the inferior esophagus from stomach acid.
Stomach 	*Propulsion and Mixing.* Mixing waves churn ingested materials and stomach secretions into chyme. Rugae allow the stomach to expand and store food. This allows further mixing in the stomach prior to propulsion of small amounts of chyme into the small intestine. Peristaltic waves move the chyme into the small intestine. *Secretion.* Release of hydrochloric acid creates the acidic stomach environment. The acid kills most microorganisms and activates the precursor of the proteolytic enzyme pepsin. Gastric lipase and intrinsic factor are secreted. Mucus provides lubrication and prevents digestion of the stomach wall. *Digestion.* Mechanical digestion occurs as food is churned in the stomach by mixing waves. Protein digestion begins as a result of the actions of hydrochloric acid and pepsin. Gastric lipase and lingual lipase digest a small amount of lipids. *Absorption.* Absorption of a few substances (e.g., water, alcohol, aspirin) takes place in the stomach.
Small intestine 	*Propulsion and Mixing.* Segmental contractions mix the chyme, and peristaltic contractions move the chyme into the large intestine. *Secretion.* Bicarbonate ions from the pancreas and bile from the liver neutralize stomach acid to form a pH environment suitable for pancreatic and intestinal enzymes. Mucus provides lubrication, prevents digestion of the intestinal wall, and protects the small intestine from stomach acid. Bile from the liver contains bilirubin and excess cholesterol that will be eliminated in the feces. *Digestion.* Segmental contractions aid mechanical digestion. Enzymes from the pancreas and the lining of the small intestine complete the breakdown of food molecules. Bile salts from the liver emulsify lipids to allow lipid digestion. *Absorption.* The circular folds, villi, and microvilli increase surface area. Most nutrients are actively or passively absorbed. Most of the ingested water or the water in digestive tract secretions is absorbed.
Large intestine 	*Propulsion and Mixing.* Slight segmental mixing occurs. Mass movements propel feces toward the anus, and defecation eliminates the feces. *Secretion.* Mucus provides lubrication; mucus and bicarbonate ions protect against acids produced by bacteria. *Absorption.* The proximal half of the colon absorbs salts (e.g., NaCl), water, and vitamins (e.g., K) produced by bacteria. *Elimination.* The distal half of the colon holds feces until they are eliminated.



PROCESS FIGURE 24.2 Peristalsis and Segmental Contractions

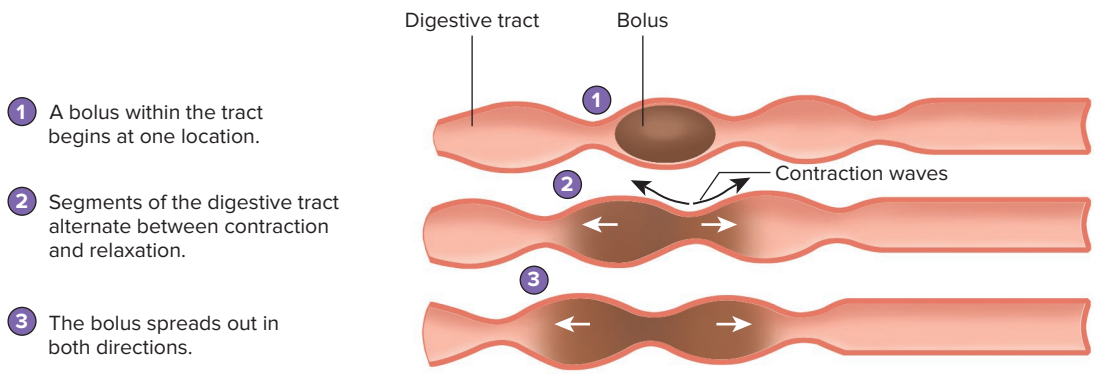
Waves of smooth muscle contraction push food and waste through the digestive tract.

? *Some agents that cause diarrhea increase peristalsis of the intestine. Explain how medications that slow intestinal motility relieve symptoms of diarrhea (see Systems Pathology 24.1).*

with mechanical digestion. There are two major types of mixing contractions:

a. **Mixing waves** in the stomach are gentle contractions that churn the food with gastric secretions. Ingested food is stored and mixed in the stomach, from where it is slowly released into the small intestine.
b. **Segmental contractions** (figure 24.3) mix food particles with digestive secretions in the small intestine.

3. *Secretion.* Secretions are added to lubricate, liquefy, buffer, and digest the food as it moves through the digestive tract. **Mucus,** secreted along the entire digestive tract, lubricates the food and the lining of the tract. The mucus coats and protects the epithelial cells of the digestive tract from mechanical abrasion, stomach acid, and digestive enzymes. The secretions also contain large amounts of **water,** which liquefies the food, making it easier to digest and absorb. Water also moves into the intestine by osmosis. Liver secretions break large lipid droplets into much smaller droplets, which makes the digestion and absorption of lipids possible. **Enzymes** secreted by the oral cavity, stomach, small intestine, and pancreas break down large food molecules into smaller molecules that can be absorbed by the intestinal wall.
4. *Digestion.* Digestion is the breakdown of large organic molecules into their component parts. Digestion consists of **mechanical digestion,** which involves the mastication and mixing of food, and **chemical digestion,** which is accomplished by digestive enzymes secreted along the digestive tract. Large organic molecules must be digested into their component parts before they can be absorbed by the digestive tract. Carbohydrates are broken into monosaccharides. Proteins are broken into amino acids, and triglycerides into fatty acids and glycerol. Minerals and water are not broken down before being absorbed. Vitamins are also absorbed without digestion; in fact, they lose their function if their structure is altered by digestion.
5. *Absorption.* Absorption is the movement of molecules out of the digestive tract and into the blood or into the lymphatic system. The mechanism by which absorption occurs depends on the type of molecule involved. Molecules pass out of the digestive tract by diffusion, facilitated diffusion, active transport, symport, or endocytosis (see chapter 3).
6. *Elimination.* Elimination is the process by which the waste products of digestion are removed from the body. During this process, which occurs primarily in the large intestine, water



PROCESS FIGURE 24.3 Segmental Contractions

Smooth muscle contractions in the wall of the small intestine disperse digesting food throughout its lumen.

? *How might reduced segmental contractions affect the digestive process?*

and salts are absorbed, changing the material in the digestive tract from liquefied to semisolid. These semisolid waste products, called **feces,** are stored in the distal large intestine, and then eliminated by the process of **defecation.**

ASSESS YOUR PROGRESS

2. *Describe each of the functions involved in the normal functions of the digestive system.*
3. *Explain the three types of propulsion through the digestive tract.*
4. *What is the difference between mechanical digestion and chemical digestion?*
5. *What digestive functions occur in the stomach? In the small intestine?*

24.3 Histology of the Digestive Tract

LEARNING OUTCOMES

After reading this section, you should be able to

A. **Describe the histology of the digestive tract.**

B. **List the types of glands associated with the digestive tract.**

The digestive tract consists of four major tunics, or layers (figure 24.4). From the inside of the tube, going outward, the four tunics are (1) mucosa, (2) submucosa, (3) muscularis, and (4) serosa or adventia. These four tunics are present in all areas of the digestive tract, from the esophagus to the anus.

Three major types of glands are associated with the digestive tract: (1) unicellular mucous glands in the mucosa, (2) multicellular glands in the mucosa and submucosa, and (3) multicellular glands (accessory glands) outside the digestive tract.

Mucosa

The innermost tunic, the **mucosa** (mū-kō′să), or *mucous membrane,* consists of three layers:

1. the inner **mucous epithelium,** which is moist stratified squamous epithelium in the mouth, oropharynx, esophagus, and anal canal and simple columnar epithelium in the remainder of the digestive tract;
2. a loose connective tissue called the **lamina propria** (lam′i-nă prō′prē-ă); and
3. a thin outer layer of smooth muscle called the **muscularis mucosae.**

The epithelium extends deep into the lamina propria in many places to form **intestinal glands** and **crypts.** Two types of

FUNDAMENTAL **Figure**

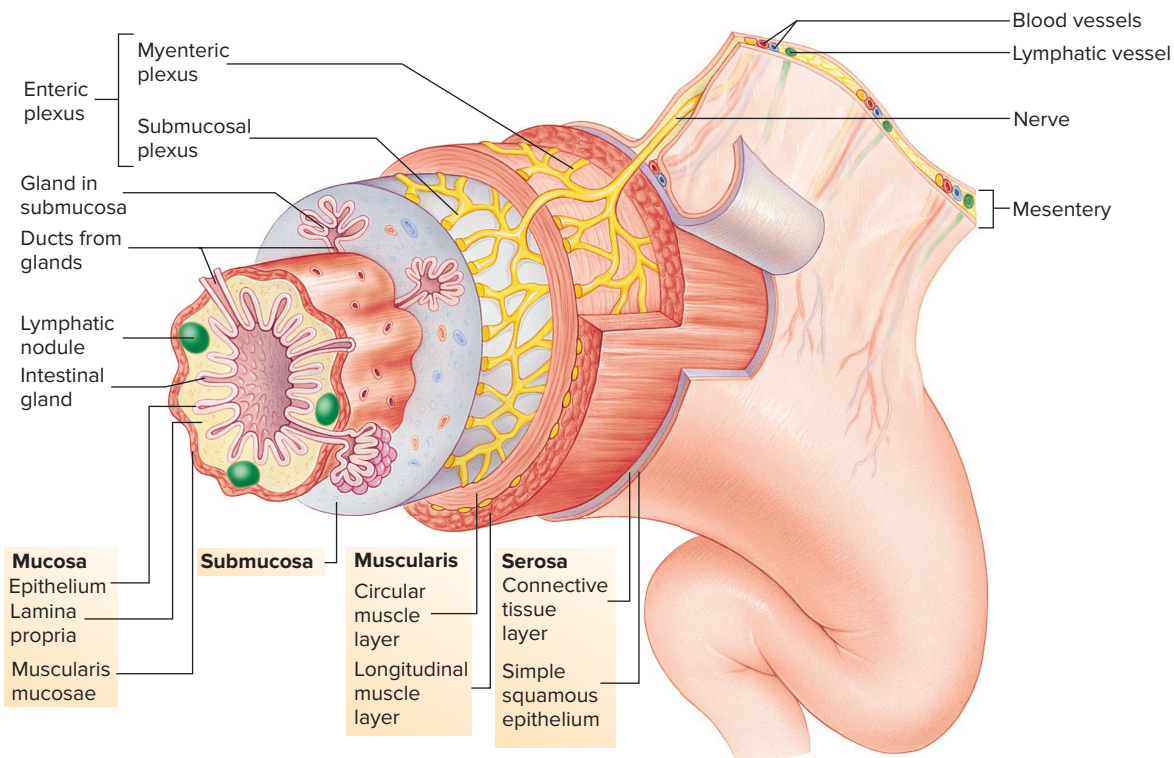


FIGURE 24.4 Digestive Tract Histology

The four tunics are the mucosa, the submucosa, the muscularis, and a serosa or an adventitia. In this image, the serosa is also called the visceral peritoneum, which forms part of the mesentery. Glands may exist along the digestive tract as part of the epithelium, as glands within the submucosa, or as large glands outside the digestive tract.

specialized cells in the mucosa are mechanoreceptors involved in peristaltic reflexes and chemoreceptors that detect the chemical composition of food.

Submucosa

Beneath the mucosa lies the **submucosa,** a thick connective tissue layer. This tunic contains nerves, blood vessels, lymphatic vessels, and small glands. A network of nerve cells in the submucosa forms the **submucosal plexus** (plek′sŭs), or *Meissner plexus,* consisting of axons, many scattered neuron cell bodies, and glial cells. Axons from the submucosal plexus extend to cells in epithelial intestinal glands, stimulating their secretion. The esophagus and stomach lack a submucosal plexus, but the plexus is extensive throughout the rest of the digestive tract.

Muscularis

The next tunic is the **muscularis,** a muscular layer. The muscularis consists of an inner layer of circular smooth muscle and an outer layer of longitudinal smooth muscle. Two exceptions are the upper esophagus, where the muscles are skeletal, and the stomach, which has three layers of smooth muscle. Between the two muscle layers is the **myenteric** (mī-en-ter′ik) **plexus,** or *Auerbach plexus* (figure 24.4). The myenteric plexus controls the motility of the intestinal tract. This function is in contrast with the submucosal plexus, which controls secretions. Both the myenteric plexus and the submucosal plexus consist of axons, many scattered neuron cell bodies, and glial cells, although the myenteric plexus is much more extensive than the submucosal plexus.

Within the myenteric plexus, specialized **interstitial cells** form a network of pacemakers, which promote rhythmic contractions of smooth muscle along the digestive tract. These cells also help transmit signals from neurons to muscles to regulate movement. Dysfunction of these pacemakers decreases motility in the digestive tract.

Together, the submucosal and myenteric plexuses constitute the **enteric nervous system (ENS),** or the *enteric* (en-tĕr′ik) *plexus,* which is extremely important in controlling secretion and movement (see section 24.4).

Serosa or Adventitia

The fourth layer of the digestive tract is either a **serosa** or an **adventitia** (ad-ven-tish′ă; foreign or coming from outside), depending on the structure of the layer. Parts of the digestive tract located within the peritoneal cavity have a serosa as the outermost layer. This serosa, or serous membrane, is called the visceral peritoneum. It consists of a thin layer of connective tissue and a simple squamous epithelium (see figure 24.4). When the outer layer of the digestive tract is derived from adjacent connective tissue, the tunic is called the adventitia and consists of a connective tissue covering that blends with the surrounding connective tissue. These areas include the esophagus and the retroperitoneal organs (see section 24.5).

ASSESS YOUR PROGRESS

6. *What are the major tunics of the digestive tract wall, listed from inside to outside?*
7. *What types of tissue are found in each tunic?*
8. *In what tunics of the digestive tract are the submucosal and myenteric plexuses found? What are their functions?*
9. *How do the serosa and adventitia differ?*

24.4 Regulation of the Digestive System

LEARNING OUTCOME

After reading this section, you should be able to

A. **Describe the overall neural and chemical regulation of the digestive system.**

The digestive system is controlled by elaborate nervous and chemical mechanisms that regulate the movement, secretion, absorption, and elimination processes.

Nervous Regulation of the Digestive System

Most of the nervous regulation of the digestive tract is under local control by the enteric nervous system. The **enteric nervous system (ENS)** is an extensive network of the submucosal and myenteric plexuses within the walls of the digestive tract (figure 24.4). This network of neurons and associated glial cells is a division of the autonomic nervous system (see chapter 16). The ENS contains more neurons than the spinal cord. In addition to local reflexes within the ENS, there is also control mediated by autonomic innervation from the CNS. This innervation is largely by the parasympathetic division of the ANS through the vagus nerves and to a lesser extent by sympathetic nerves (see chapter 16).

There are three major types of enteric neurons: (1) Enteric sensory neurons detect changes in the chemical composition of digestive tract contents or detect mechanical changes, such as stretch of the digestive tract wall; (2) enteric motor neurons stimulate or inhibit smooth muscle contraction and glandular secretion in the digestive system; and (3) enteric interneurons connect enteric sensory and motor neurons. The ENS functions through **local reflexes** to control activities within specific, short regions of the digestive tract. The ENS is capable of controlling the complex peristaltic and mixing movements, as well as blood flow to the digestive tract, without any outside influences. The importance of the ENS is highlighted by the poor intestinal motility observed in patients with **Hirschprung disease,** or *megacolon,* who lack a subset of enteric neurons (see Clinical Impact 24.1).

Although the ENS can control the activities of the digestive tract independent of the CNS, the two systems normally work together. Autonomic innervation from the CNS can increase or decrease ENS activity.

Control of the digestive system by the CNS occurs when reflexes are activated by stimuli originating either in the digestive tract or in the CNS. From within the digestive system, action potentials are carried by sensory neurons in the vagus and sympathetic nerves to the CNS, where the reflexes are integrated. Reflexes within the CNS can be activated by the sight, smell, or taste of food. An example is increased salivation and pancreatic secretions when food

is seen or smelled. All of these reflexes influence activity in parasympathetic neurons of the CNS. Parasympathetic neurons extend to the digestive tract through the vagus nerves to control responses or alter the activity of the ENS and local reflexes. Some sympathetic neurons inhibit muscle contraction and secretion in the digestive system and decrease blood flow to the digestive system.

Chemical Regulation of the Digestive System

Over 30 neurotransmitters are associated with the ENS. Two major ENS neurotransmitters are acetylcholine and norepinephrine. In general, acetylcholine *stimulates* and norepinephrine *inhibits* digestive tract motility and secretions.

Another major ENS neurotransmitter is serotonin, which stimulates digestive tract motility. In addition to neural release, serotonin is also produced by endocrine cells within the digestive tract wall. Over 95% of the serotonin in the body is found in the digestive tract, so drugs that increase serotonin levels and function, such as antidepressants (see chapter 11) and chemotherapeutics used for cancer treatment, can also affect digestive tract activity. An unintended consequence of many cancer therapies is nausea, due to increased serotonin release from endocrine cells in the digestive tract. Serotonin binds to a subset of serotonin receptors on sensory terminals of the vagus nerve, which stimulates the vomiting center in the brain. This results in the nausea and vomiting associated with chemotherapy and radiotherapy. Serotonin receptor blockers, such as ondansetron (ōn-dan′sē-tron), are commonly used to alleviate nausea.

A number of hormones, such as gastrin and secretin, are secreted by endocrine cells in the digestive system and are carried through the blood to target organs of the digestive system or to target tissues in other systems. These hormones help regulate many digestive tract functions, as well as the secretions of associated glands, such as the liver and pancreas.

In addition to the hormones produced by the digestive system that enter the blood, other paracrine chemicals, such as histamine, are released locally within the digestive tract, where they influence the activity of nearby cells. These localized chemical regulators help local reflexes within the ENS control local digestive tract environments, such as pH levels.

ASSESS YOUR PROGRESS

10. *Describe the roles of the ENS, CNS, and ANS in controlling the digestive system.*
11. *What chemical mechanisms regulate the digestive system?*

24.5 Peritoneum

LEARNING OUTCOME

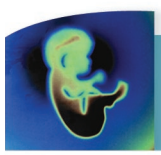
After reading this section, you should be able to

A. **Describe the peritoneum and its function.**

The walls and organs of the abdominal cavity are lined with **serous membranes.** These membranes are called the **peritoneum** (per′i-tō-nē′um; to stretch over; figure 24.5). Serous membranes are very smooth and secrete a serous fluid, which provides a lubricating film between the layers of membranes. The membranes and fluid reduce friction as organs move within the abdominal cavity. The serous membrane that covers the organs is the **visceral peritoneum,** and the one that covers the interior surface of the wall of the abdominal cavity is the **parietal peritoneum** (figure 24.5). Serous membranes also line other organs of the body.

Peritonitis is a potentially life-threatening inflammation of the peritoneal membranes. The inflammation can result from chemical irritation by substances, such as bile, that have escaped from a damaged digestive tract or from infection originating in the digestive tract, as when the appendix ruptures. The main symptoms of peritonitis are acute abdominal pain and tenderness that are worsened by movement. An accumulation of excess serous fluid in the peritoneal cavity, called **ascites** (ă-sī′tēz), can occur in peritonitis. Ascites can also accompany starvation, alcoholism, or liver cancer.

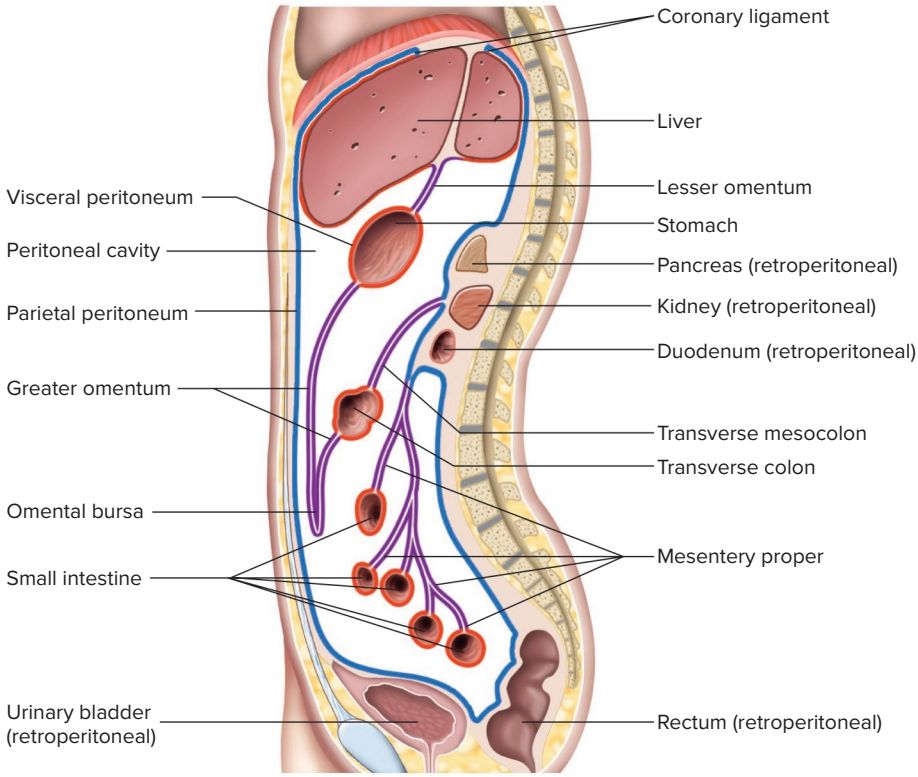
Many organs within the abdominal cavity are held in place by connective tissue sheets called **mesenteries** (mes′en-ter′ēz; middle intestine). The mesenteries consist of two layers of serous membranes with a thin layer of loose connective tissue between them. They provide a route by which vessels and nerves can pass from the abdominal wall to the organs. Although *mesentery* is a general term referring to the serous membranes attached to the abdominal organs, it is also applied specifically to the mesentery associated with the small intestine, sometimes called the **mesentery proper.** The mesenteries of parts of the colon are the **transverse mesocolon,** which extends from the transverse colon to the



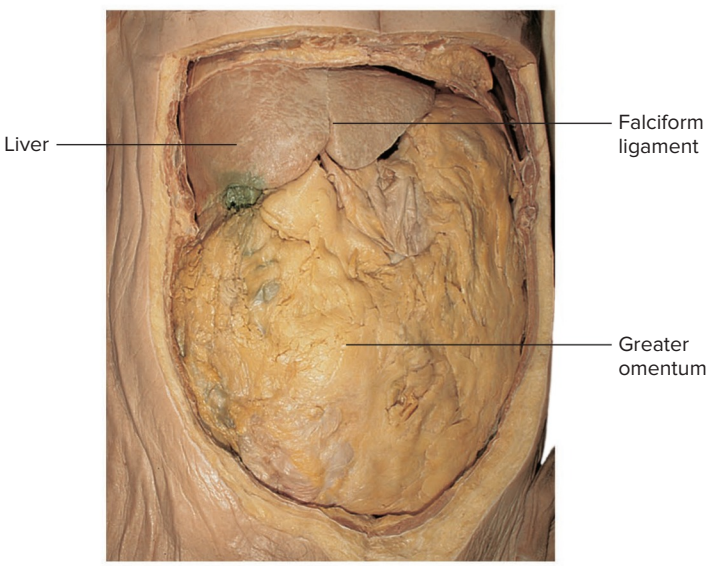
Clinical IMPACT 24.1

Enteric Neurons

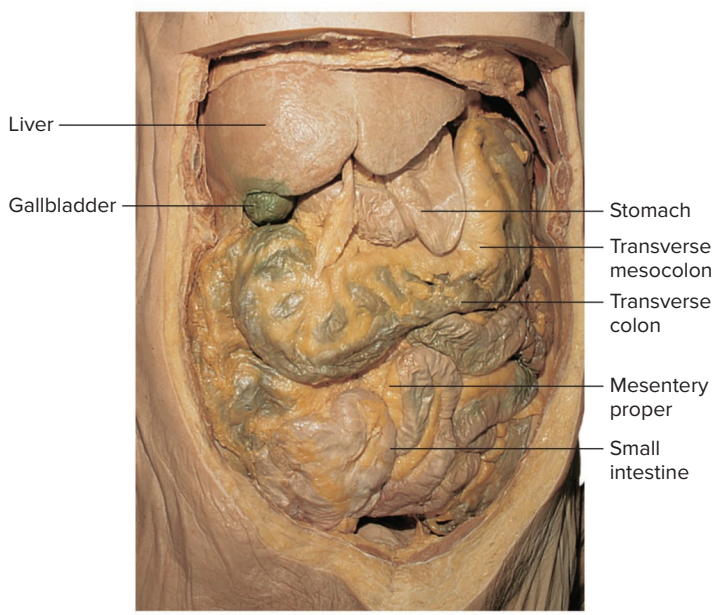
Hirschprung disease, also called *megacolon,* is a painful developmental disorder caused by the absence of enteric neurons in the distal large intestine. Mutations in the *RET* gene have been identified in patients with Hirschprung disease. The *RET* gene encodes a receptor that is normally activated by the growth factors required for the survival and differentiation of a subset of enteric neurons. The mutations in *RET* that lead to loss of receptor function result in loss of enteric neurons, which results in poor intestinal motility and severe constipation. Conversely, a different set of mutations in the *RET* gene is linked to an inherited cancer called **multiple endocrine neoplasia type 2 (MEN2).** In contrast to the loss of function due to Hirschprung mutations, the MEN2 mutations cause a gain of RET receptor function, so that it is active even in the absence of growth factors. Hence, two types of mutations in the same gene result in two very different syndromes. Rapid DNA tests are used to screen patients and family members for suspected Hirschprung and MEN2 mutations.



(a) **Medial view**



(b) **Anterior view**



(c) **Anterior view**

FIGURE 24.5 Peritoneum and Mesenteries

(*a*) Sagittal section through the trunk, showing the peritoneum and mesenteries associated with some abdominal organs. (*b*) Photograph of the abdomen of a cadaver, with the greater omentum in place. (*c*) Photograph of the abdomen of a cadaver, with the greater omentum removed to reveal the underlying viscera. (b and c) ©McGraw-Hill Education/Rebecca Gray, photographer AP|R

posterior body wall, and the **sigmoid mesocolon,** which extends from the sigmoid colon to the posterior body wall. The vermiform appendix has its own little mesentery, called the **mesoappendix.**

The mesentery connecting the lesser curvature of the stomach and the proximal end of the duodenum to the liver and diaphragm is called the **lesser omentum** (ō-men′tŭm; membrane of the bowels), and the mesentery extending as a fold from the greater curvature and then to the transverse colon is called the **greater omentum** (figure 24.5). The greater omentum forms a long, double fold of mesentery that extends inferiorly from the stomach over the surface of the small intestine. Because of this folding, a cavity called the **omental bursa** (ber′să; pocket) forms between the two layers of mesentery. A large amount of adipose tissue accumulates in the greater omentum, and it is sometimes referred to as the "fatty apron." The greater omentum has considerable mobility in the abdomen.

Predict 1

If you placed a pin through the greater omentum, through how many layers of simple squamous epithelium would the pin pass?

The **coronary ligament** attaches the liver to the diaphragm. Unlike other mesenteries, the coronary ligament has a wide space in the center, the bare area of the liver, where no peritoneum exists. The **falciform ligament** attaches the liver to the anterior abdominal wall (figure 24.5*b*).

Other abdominal organs that have no mesenteries are referred to as **retroperitoneal** (re′trō-per′i-tō-nē′ăl; behind the peritoneum; see chapter 1). The retroperitoneal organs lie along the abdominal wall and include the duodenum, pancreas, ascending colon, descending colon, rectum, kidneys, adrenal glands, and urinary bladder.

ASSESS YOUR PROGRESS

12. *Where are the visceral peritoneum and parietal peritoneum found? Define and give examples of retroperitoneal organs.*
13. *What is the function of the peritoneum?*
14. *What are the mesenteries? Name and describe the location of the mesenteries in the abdominal cavity.*

24.6 Oral Cavity

LEARNING OUTCOMES

After reading this section, you should be able to

A. **Describe the oral cavity and the structure and function of the lips, cheeks, palate, and tongue.**
B. **Outline the structure and types of adult teeth and describe the process of mastication.**
C. **Compare the structures and locations of the major salivary glands and describe the composition and functions of saliva and the control of its release.**

The **oral cavity** (figure 24.6), or *mouth,* is divided into two regions: (1) The **vestibule** (ves′ti-bool; entry) is the space between the lips or cheeks and the teeth, and (2) the **oral cavity proper** lies medial to the teeth. The oral cavity is lined with moist stratified squamous epithelium, which protects against abrasion.

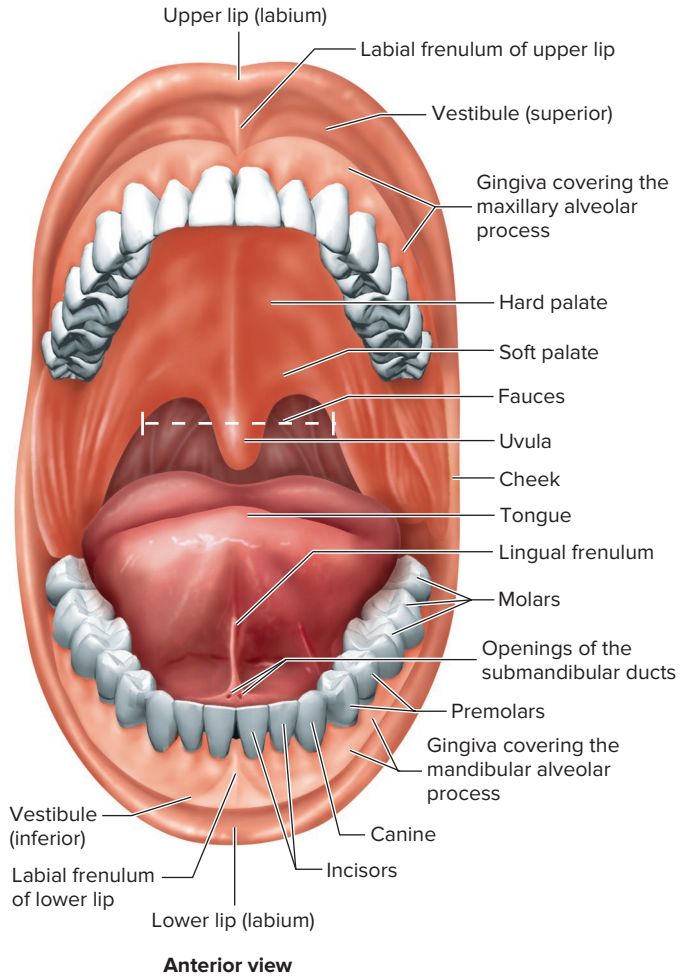


FIGURE 24.6 Oral Cavity
The oral cavity is the normal location for ingestion of liquid and solid food. AP|R

Lips, Cheeks, and Palate

The **lips,** or *labia* (lā′bē-ă; figure 24.6), are muscular structures formed mostly by the **orbicularis oris** (ōr-bik′ū-lā′ris ōr′is) **muscle** (see figure 10.9) and connective tissue. The outer surfaces of the lips are covered by skin. The keratinized stratified epithelium of the skin is thin at the margin of the lips and is not as highly keratinized as the epithelium of the surrounding skin (see chapter 5); consequently, it is more transparent than the epithelium over the rest of the body. The color from the underlying blood vessels shows through the relatively transparent epithelium, giving the lips a reddish-pink to dark red appearance, depending on the overlying pigment. At the internal margin of the lips, the epithelium is continuous with the moist stratified squamous epithelium of the mucosa in the oral cavity.

One or more **labial frenula** (fren′ū-lă; sing. *frenulum*), which are mucosal folds, extend from the alveolar process of the maxilla to the upper lip and from the alveolar process of the mandible to the lower lip.

The **cheeks** form the lateral walls of the oral cavity. They consist of an interior lining of moist stratified squamous epithelium and an exterior covering of skin. The substance of the cheek includes the **buccinator muscle** (see chapter 10), which flattens the cheek against the teeth, and the **buccal fat pad,** which rounds out the profile on the side of the face.

The lips and cheeks are important in mastication and speech. They help manipulate food within the oral cavity and hold it in place while the teeth crush or tear it. They also help form words when we speak. A large number of the muscles of facial expression are involved in moving the cheeks and lips (see chapter 10).

The roof of the oral cavity is called the **palate.** The palate separates the oral and nasal cavities and prevents food from passing into the nasal cavity during chewing and swallowing. The palate consists of two parts (figure 24.6). The anterior, bony part is the **hard palate** (see chapter 7). The posterior, nonbony part is the **soft palate,** which consists of skeletal muscle and connective tissue. The **uvula** (ū′vū-lă; a grape) is a posterior projection from the soft palate. The posterior boundary of the oral cavity is the **fauces** (faw′sēz), which is the opening into the pharynx, or *throat*. The **palatine tonsils** are in the lateral wall of the fauces (see chapter 22).

Tongue

The **tongue** is a large, muscular organ that occupies most of the oral cavity proper when the mouth is closed. Its major attachment in the oral cavity is through its posterior part. The anterior part of the tongue is relatively free, except for attachment to the floor of the mouth by a thin fold of tissue called the **lingual** (tongue) **frenulum.** The muscles associated with the tongue are divided into two categories: **Intrinsic muscles** are within the tongue itself, and **extrinsic muscles** are outside the tongue but attached to it. The intrinsic muscles are largely responsible for changing the shape of the tongue, such as flattening and elevating it during drinking and swallowing. The extrinsic tongue muscles protrude and retract the tongue, move it from side to side, and change its shape (see chapter 10).

A groove called the **terminal sulcus** divides the tongue into two parts. The part anterior to the terminal sulcus accounts for about two-thirds of the surface area and is covered by papillae, some of which contain taste buds (see chapter 15). The posterior one-third of the tongue is devoid of papillae and has only a few scattered taste buds. Instead, it has a few small glands and a large amount of lymphatic tissue, which form the **lingual tonsil** (see chapter 22). Moist stratified squamous epithelium covers the tongue.

The tongue moves food in the mouth and, in cooperation with the lips and gums, holds the food in place during mastication. It also plays a major role in swallowing. In addition, the tongue is a major sensory organ for taste (see chapter 15) and one of the primary organs of speech. Patients with cancer of the tongue often have part or all of their tongue removed. These patients can learn to speak fairly well but have difficulty chewing and swallowing.

Teeth

The teeth play an important role in mastication and assist in speech. Adults normally have 32 **teeth,** which are distributed in two **dental arches:** the maxillary arch and the mandibular arch. The teeth in the right and left halves of each dental arch are roughly mirror images of each other. As a result, the teeth are apportioned into four quadrants: right-upper, left-upper, right-lower, and left-lower. The teeth in each quadrant include one central and one lateral **incisor;** one **canine;** first and second **premolars;** and first, second, and third **molars** (figure 24.7*a*). The third molars are often called *wisdom teeth* because they usually appear in the late teens or early twenties, when a person is old enough to have acquired some wisdom. In people with small dental arches, the third molars may not have room to erupt into the oral cavity and remain embedded within the jaw. Embedded wisdom teeth are referred to as impacted and may cause pain or irritation. Usually, the impacted wisdom teeth are surgically removed.

The teeth of the adult mouth are called **permanent teeth,** or *secondary teeth*. Most of them are replacements for **deciduous** (dē-sid′ū-ŭs) **teeth,** or *primary teeth,* also called *milk teeth,* which are lost during childhood (figure 24.7*b*). The deciduous teeth erupt (the crowns appear within the oral cavity) between about 6 months and 24 months of age (figure 24.7*b*). The permanent teeth begin replacing the deciduous teeth at about 5 years, and the process is completed by about 11 years.

Each tooth consists of (1) a crown, (2) a neck, and (3) a root (figure 24.8). The **crown** is the entire enamel-covered part of the tooth. It is also called the *anatomical crown* to distinguish it from the *clinical crown,* which only includes the part of the tooth exposed in the oral cavity. The crown can have one or more **cusps** (points). The **neck** is the small region between the crown and root. The **root** is the largest region of the tooth. It anchors the tooth in the jawbone.

Within the center of the tooth, in the neck and root, is a **pulp cavity,** which is filled with blood vessels, nerves, and connective tissue called **pulp.** The portion of the pulp cavity within the root is called the **root canal.** The nerves and blood vessels of the tooth enter and exit the pulp through a hole at the point of each root called the **apical foramen.** The pulp cavity is surrounded by living, cellular, calcified tissue called **dentin.** The dentin of the tooth crown is covered by an extremely hard, nonliving, acellular substance called **enamel,** which protects the tooth against abrasion and acids produced by bacteria in the mouth. The surface of the dentin in the root is covered with a bonelike substance called **cementum,** which helps anchor the tooth to the periodontal ligament in the jaw.

The teeth are set in **alveoli** (al-vē′ō-lī; sockets) along the alveolar processes of the mandible and maxilla. Dense fibrous connective tissue and stratified squamous epithelium, referred to as the **gingiva** (jin′ji-vă; gums), cover the alveolar processes (see figure 24.6). **Periodontal** (per′ē-ō-don′tăl; around a tooth) **ligaments** secure the teeth in the alveoli.

Several conditions can affect the health of the tooth and the structures associated with it, such as the periodontal ligament and gingiva. **Dental caries,** or tooth decay, is a breakdown of enamel by bacterial acids on the tooth surface. Because the enamel is nonliving and cannot repair itself, a dental filling is necessary to prevent further damage. If the decay reaches the pulp cavity, with its rich supply of nerves, a toothache may result. Sometimes, when decay has reached the pulp cavity, a dentist must perform a procedure called a root canal, which consists of removing the pulp

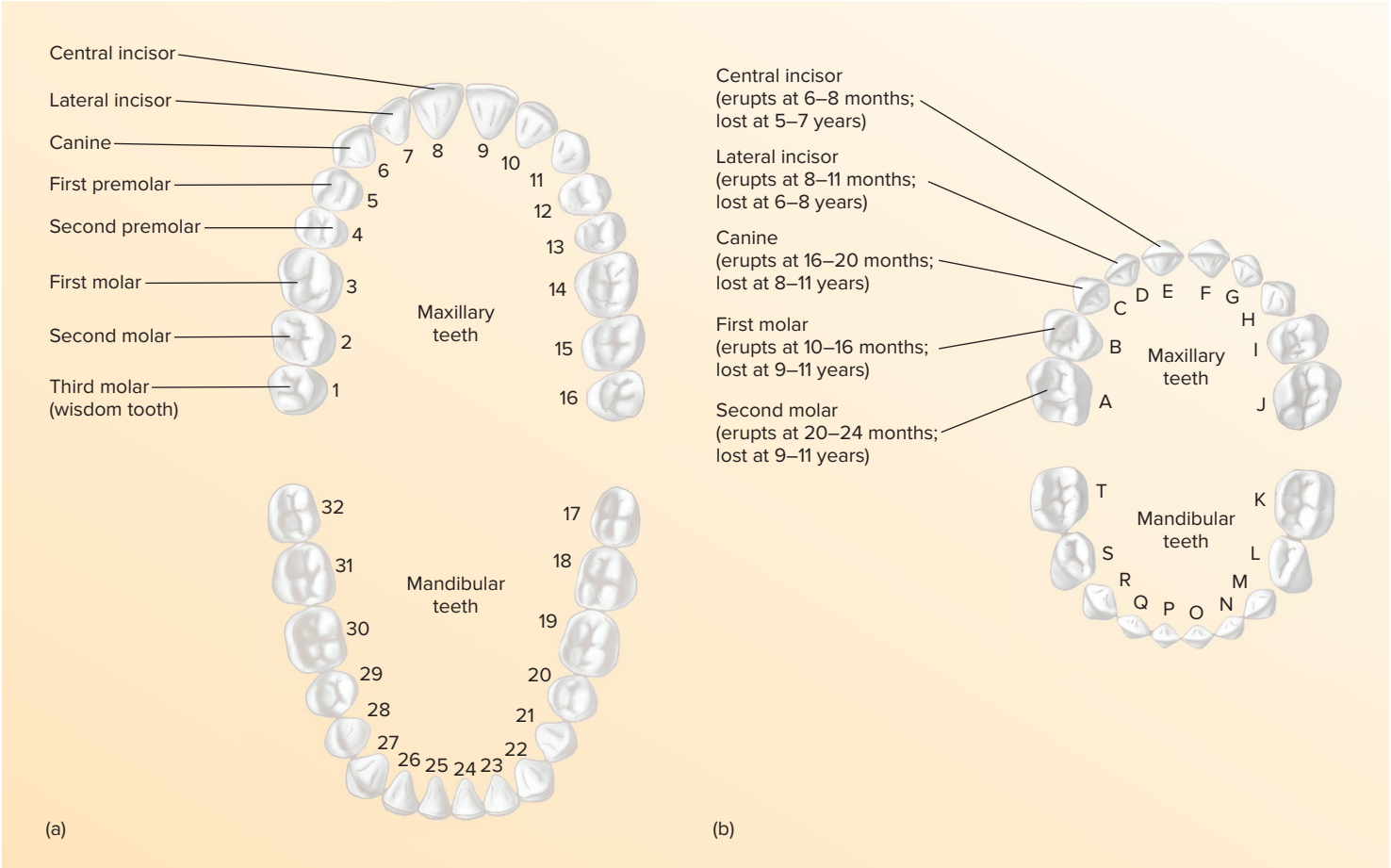


FIGURE 24.7 Teeth

(*a*) Permanent teeth. (*b*) Deciduous teeth. Dental professionals have developed a "universal" numbering and lettering system for convenience in identifying individual teeth.

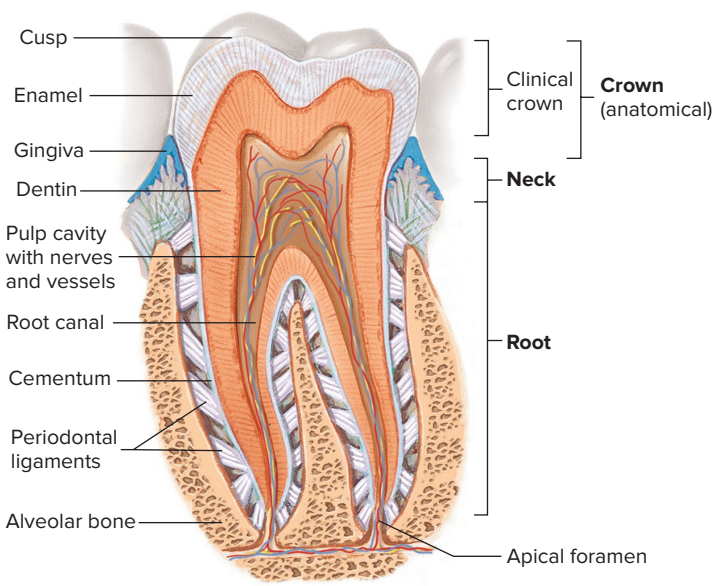


FIGURE 24.8 Molar Tooth in Place in the Alveolar Bone

A tooth consists of a crown (anatomical and clinical), a neck, and a root. The root is covered with cementum, and the tooth is held in the socket by periodontal ligaments. Nerves and vessels enter and exit the tooth through the apical foramen.

from the tooth. **Periodontal disease** is the inflammation and degradation of the periodontal ligaments, gingiva, and alveolar bone. This disease is the most common cause of tooth loss in adults. **Gingivitis** (jin-ji-vī′tis) is an inflammation of the gingiva, often caused by food deposited in gingival crevices and not promptly removed by brushing and flossing. Gingivitis may eventually lead to periodontal disease. **Halitosis** (hal-i-tō′sis), or bad breath, often occurs with periodontal disease.

ASSESS YOUR PROGRESS

15. *What is the difference between the vestibule and the oral cavity proper?*
16. *What are the functions of the lips and cheeks? What muscle forms the substance of the lips? The cheeks?*
17. *What are the hard and soft palates? Where is the uvula located?*
18. *List the functions of the tongue. Distinguish between intrinsic and extrinsic tongue muscles.*
19. *What are permanent and deciduous teeth? Name the types of teeth.*
20. *List the three parts of a tooth. What are dentin, enamel, cementum, and pulp?*

Mastication

Food taken into the mouth is **masticated,** or *chewed,* by the teeth. The anterior teeth (the incisors and the canines) primarily cut and tear food, whereas the premolars and molars primarily crush and grind it. Mastication breaks large food particles into smaller ones, creating a much larger total surface area. Because digestive enzymes digest food molecules only at the surface of the particles, mastication increases the efficiency of digestion.

Four pairs of muscles move the mandible during mastication: (1) **temporalis,** (2) **masseter,** (3) **medial pterygoid,** and (4) **lateral pterygoid** muscles (see chapter 10). All four close the jaw, while the lateral pterygoid muscle opens it. The medial and lateral pterygoids and the masseter muscles accomplish protraction and lateral and medial excursion of the jaw. The temporalis retracts the jaw. All these movements are involved in tearing, crushing, and grinding food.

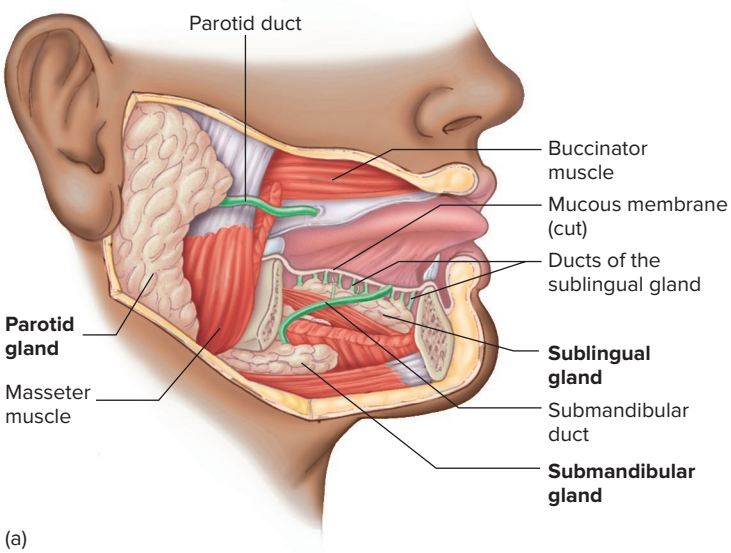
The **mastication reflex,** or *chewing reflex,* is integrated in the medulla oblongata and controls the basic movements of chewing. The presence of food in the mouth stimulates sensory receptors, which activate a reflex that relaxes the muscles of mastication. As the mandible is lowered, the muscles stretch and activate a reflex that causes the muscles of mastication to contract. Once the mouth is closed, the food again stimulates the muscles of mastication to relax, and the cycle repeats. Descending pathways from the cerebrum strongly influence the mastication reflex, so that chewing can be consciously initiated or stopped. The rate and intensity of chewing movements can also be influenced by the cerebrum.

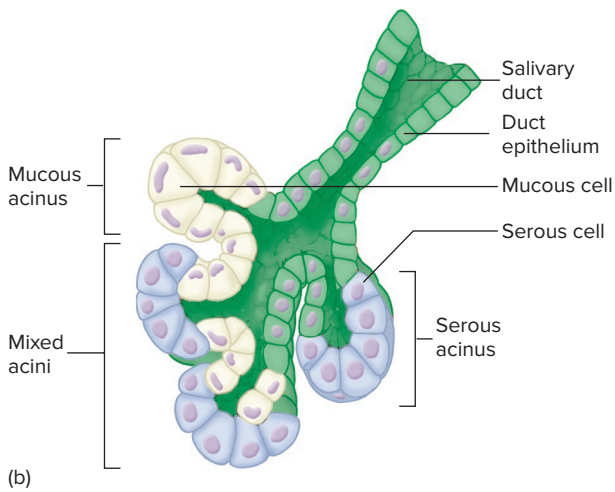
Salivary Glands

A considerable number of **salivary glands** are scattered throughout the oral cavity. There are three pairs of large, multicellular salivary glands: (1) parotid glands, (2) submandibular glands, and (3) sublingual glands (figure 24.9). In addition to these large salivary glands, numerous small, coiled, tubular salivary glands are located: (1) deep to the epithelium of the tongue (lingual glands), (2) in the palate (palatine glands), (3) in the cheeks (buccal glands), and (4) in the lips (labial glands).

All of the major large salivary glands are compound **acinar glands,** which are branching glands with clusters of acini resembling grapes (see chapter 4). They produce thin serous secretions or thicker mucous secretions. **Saliva** is a combination of serous and mucous secretions from the various salivary glands.

The largest salivary glands, the **parotid** (pă-rot′id; beside the ear) **glands,** are serous glands, which produce mostly watery saliva;





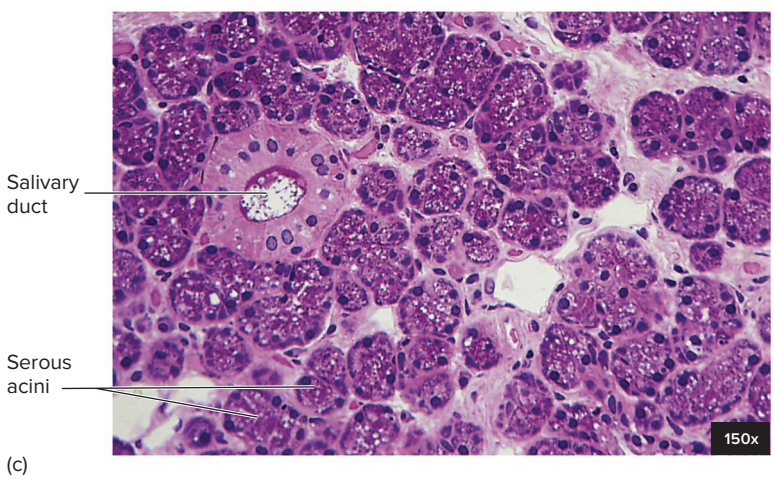


FIGURE 24.9 Salivary Glands

(*a*) The large salivary glands are the parotid glands, the submandibular glands, and the sublingual glands. The parotid duct extends anteriorly from the parotid gland. (*b*) An idealized schematic illustrates the histology of the large salivary glands. The figure is representative of all the glands and does not depict any specific salivary gland. (*c*) Photomicrograph of the parotid gland.

they are located just anterior to the ear on each side of the head. Each **parotid duct** exits the gland on its anterior margin, crosses the lateral surface of the masseter muscle, pierces the buccinator muscle, and enters the oral cavity adjacent to the second upper molar (figure 24.9*a*). A viral infection can cause the parotid glands to become inflamed and swollen, making the cheeks quite large. Before the measles/mumps/rubella (MMR) vaccination program was begun in the United States, **mumps** was a common childhood disease caused by the mumps virus. Now mumps is very rare in the United States. The virus causing mumps can also infect other tissues, including the testes, which can result in sterility in an adult male.

The **submandibular** (below the mandible) **glands** are mixed glands with more serous than mucous acini. Each gland can be felt as a soft lump along the inferior border of the posterior half of the mandible. A submandibular duct exits each gland, passes anteriorly deep to the mucous membrane on the floor of the oral cavity, and opens into the oral cavity beside the frenulum of the tongue (see figure 24.6).

The **sublingual** (below the tongue) **glands,** the smallest of the three large, paired salivary glands, are mixed glands containing some serous acini but consisting primarily of mucous acini. They lie immediately below the mucous membrane in the floor of the oral cavity. These glands do not have single, well-defined ducts like those of the submandibular and parotid glands. Instead, each sublingual gland opens into the floor of the oral cavity through 10–12 small ducts.

Saliva is composed of fluid and proteins and has three main roles (table 24.2): (1) it helps keep the oral cavity moist, which is needed for normal speech and for the suspension of food molecules in solution so they can be tasted; (2) it has protective functions; and (3) it begins the process of digestion.

The moistening function of saliva is aided by the large volume of serous saliva, 1–1.5 L/day, secreted primarily by the parotid and submandibular glands. In addition, the mucous secretions of the submandibular and sublingual glands contain a large amount of **mucin** (mū′sin), a proteoglycan that gives a lubricating quality to the secretions of the salivary glands.

There are several protective functions of saliva.

1. The large volume of saliva helps prevent bacterial infection in the mouth by continually washing the oral surface.
2. The bicarbonate ions in saliva act as a buffer to neutralize the acids produced by oral bacteria. This reduces the harmful effects of bacterial acids on tooth enamel.
3. Saliva contains the proteins **lysozyme,** an enzyme that has a weak antibacterial action, and immunoglobulin A, which helps prevent bacterial infection.
4. The mucous in saliva helps protect the digestive tract from physical irritation and enzymatic digestion. Any lack of salivary gland secretion increases the risk for ulceration and infection of the oral mucosa and for caries (cavities) in the teeth.

The digestive functions of saliva are relatively minor compared with digestion later in the tract. The serous part of saliva contains a digestive enzyme called **salivary amylase** (am′il-ās; starch-splitting enzyme), which breaks the covalent bonds between glucose molecules in starch and other polysaccharides to produce the disaccharides maltose and isomaltose (table 24.2). These sugars can give starches a sweet taste. However, food spends very little time in the mouth, so only about 3–5% of the total carbohydrates are digested there. In addition, most starchy foods come from plants and are therefore covered by cellulose, making them inaccessible to salivary amylase. Cooking and thoroughly chewing food destroy the cellulose covering and increase the efficiency of the digestive process. In addition to carbohydrate digestion, there is a small amount of lipid digestion initiated by **lingual lipase** in saliva.

Salivary gland secretion is stimulated by both the parasympathetic and the sympathetic nervous systems, but the parasympathetic system is more important. Salivary nuclei in the brainstem increase salivary secretions by sending action potentials through parasympathetic fibers of the facial (VII) and glossopharyngeal (IX) cranial nerves in response to a variety of stimuli, such as tactile stimulation in the oral cavity or certain tastes, especially sour. Higher centers of the brain also affect salivary gland activity. Odors that trigger thoughts of food or the sensation of hunger can increase saliva secretion as well.

ASSESS YOUR PROGRESS

21. *List the muscles of mastication and the actions they produce. Describe the mastication reflex.*

22. *Name and give the location of the three largest salivary glands. What are the other types of salivary glands called?*

23. *What are the functions of saliva? What substances are contained in saliva?*

24. *What is the difference between serous and mucous saliva?*

25. *Describe the stimuli that stimulate the release of saliva. What nerves are involved?*

24.7 Swallowing

LEARNING OUTCOMES

After reading this section, you should be able to

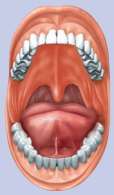
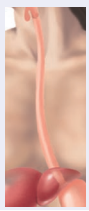
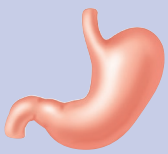
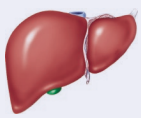
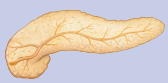
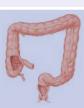
A. **List the parts of the pharynx involved with digestion.**
B. **Describe the structure of the esophagus.**
C. **Explain the three phases of swallowing.**

Swallowing involves distinct phases in the pharynx and esophagus (figure 24.10).

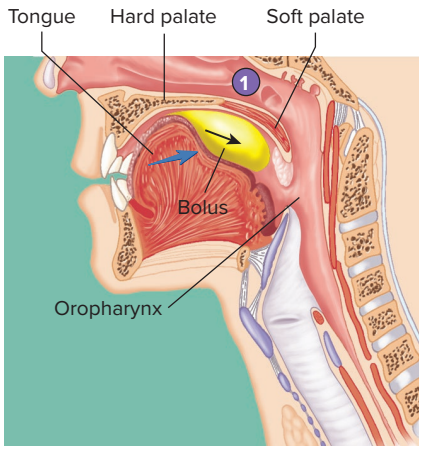
Pharynx

The **pharynx,** described in detail in chapter 23, consists of three parts: (1) nasopharynx, (2) oropharynx, and (3) laryngopharynx. Normally, only the oropharynx and laryngopharynx transmit food. The **oropharynx** communicates with the nasopharynx superiorly, with the larynx and **laryngopharynx** inferiorly, and with the mouth anteriorly. The laryngopharynx extends from the oropharynx to the esophagus and is posterior to the larynx. The epiglottis covers the opening of the larynx and keeps food and drink from entering the larynx. The posterior walls of the oropharynx and laryngopharynx consist of three muscles: the superior, middle, and inferior **pharyngeal constrictors,** which are arranged like three stacked flowerpots, one inside the other. The oropharynx and the laryngopharynx are lined with moist stratified squamous epithelium, and the nasopharynx is lined with ciliated pseudostratified columnar epithelium.

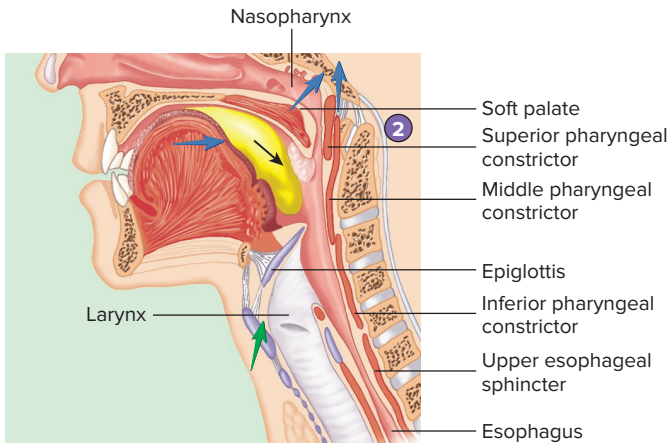
TABLE 24.2 Functions of Major Digestive System Secretions

	Secretions	Function
	**Oral Cavity**	
	Serous saliva (mostly water, bicarbonate ions)	Moistens food and mucous membrane; neutralizes bacterial acids; flushes bacteria from oral cavity; has weak antibacterial activity
	Salivary amylase	Digests carbohydrates
	Mucus	Lubricates food; protects digestive tract from digestion
	Lingual lipase	Digests a minor amount of lipids
	**Esophagus**	
	Mucus	Lubricates esophagus; protects lining of esophagus from abrasion and allows food to move more smoothly through esophagus
	**Gastric**	
	Hydrochloric acid	Antibacterial; decreases stomach pH to activate pepsinogen to pepsin
	Pepsin*	Digests protein into smaller peptide chains; activates pepsinogen
	Mucus	Protects stomach lining from acid and digestion
	Intrinsic factor	Binds to vitamin B_{12} and aids in its absorption in the small intestine
	Gastric lipase	Digests a minor amount of lipids
	**Liver**	
	Bile	Bile salts in bile emulsify lipids, making them available to lipases, and help make end products soluble and available for absorption by the intestinal mucosa; many of the other bile contents are waste products, such as bile pigments, that are transported to the intestines for disposal
	**Pancreas**	
	Trypsin*	Digests proteins (cleaves at arginine or lysine amino acids); activates trypsinogen and other digestive enzymes
	Chymotrypsin*	Digests proteins (cleaves at hydrophobic amino acids)
	Carboxypeptidase*	Digests proteins (removes amino acids from the carboxyl end of proteins)
	Pancreatic amylase	Digests carbohydrates (hydrolyzes starches and glycogen to form maltose and isomaltose)
	Pancreatic lipase	Digests lipids (breaks down triglycerides into monoglycerides and free fatty acids)
	Cholesterol esterase	Digests cholesteryl esters (breaks down into cholesterol and free fatty acid)
	Ribonuclease	Digests ribonucleic acid (hydrolyzes phosphodiester bonds)
	Deoxyribonuclease	Digests deoxyribonucleic acid (hydrolyzes phosphodiester bonds)
	Bicarbonate ions	Neutralize acid from stomach; provide appropriate pH for pancreatic enzymes
	**Small Intestine**	
	Mucus	Protects duodenum from stomach acid, and intestinal wall from digestive enzymes
	Peptidases†	Split amino acids from polypeptides
	Enterokinase†	Activates trypsin from trypsinogen
	Sucrase†	Splits sucrose into glucose and fructose
	Maltase†	Splits maltose into two glucose molecules
	Isomaltase†	Splits isomaltose into two glucose molecules
	Lactase†	Splits lactose into glucose and galactose
	**Large Intestine**	
	Mucus	Provides adhesion for fecal matter; protects intestinal wall from bacterial acids and actions

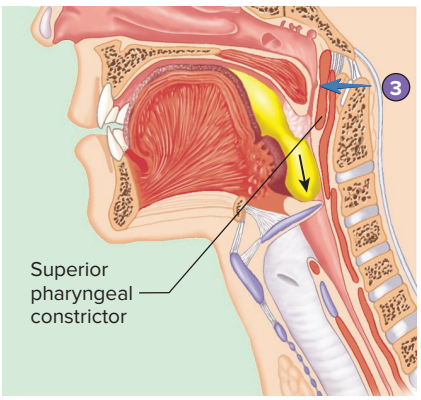
*These enzymes are secreted as inactive forms and then activated.
†These enzymes remain in the microvilli.

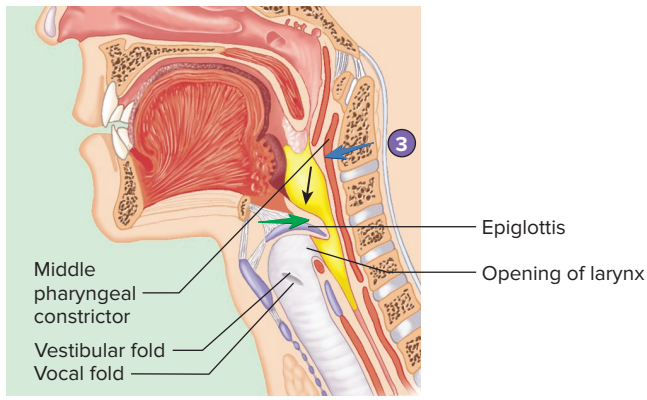


1 During the **voluntary phase**, a bolus of food (*yellow*) is pushed by the tongue against the hard and soft palates and posteriorly toward the oropharynx (*blue arrow* indicates tongue movement; *black arrow* indicates movement of the bolus). *Tan:* bone; *purple:* cartilage; *red:* muscle.

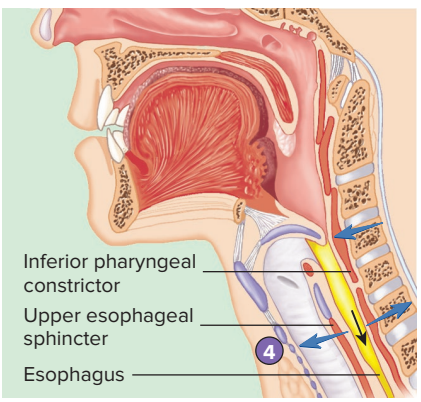


2 During the **pharyngeal phase**, the soft palate is elevated, closing off the nasopharynx. The pharynx and larynx are elevated (*blue arrows* indicate muscle movement; *green arrow* indicates elevation of the larynx).

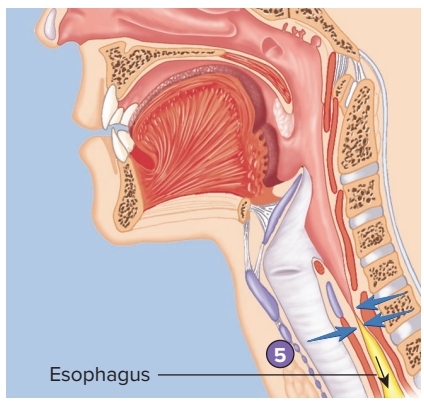




3 Successive constriction of the pharyngeal constrictors from superior to inferior (*blue arrows*) forces the bolus through the pharynx and into the esophagus. As this occurs, the vestibular and vocal folds expand medially to close the passage of the larynx. The epiglottis (*green arrow*) is bent down over the opening of the larynx largely by the force of the bolus pressing against it.



4 As the inferior pharyngeal constrictor contracts, the upper esophageal sphincter relaxes (outwardly directed *blue arrows*), allowing the bolus to enter the esophagus.



5 During the **esophageal phase**, the bolus is moved by peristaltic contractions of the esophagus toward the stomach (inwardly directed *blue arrows*).

PROCESS FIGURE 24.10 Phases of Swallowing

The three phases of swallowing are voluntary, pharyngeal, and esophageal.

? *During which part of the swallowing reflex is it not possible to talk or even breathe? Why?*

Esophagus

The **esophagus** is the part of the digestive tract that extends between the pharynx and the stomach. It is about 25 cm long and lies in the mediastinum, anterior to the vertebrae and posterior to the trachea. It passes through the esophageal hiatus (opening) of the diaphragm and ends at the stomach. The esophagus transports food from the pharynx to the stomach.

The esophagus has thick walls consisting of the four tunics common to the digestive tract: (1) mucosa, (2) submucosa, (3) muscularis, and (4) adventitia. The muscularis has an outer longitudinal layer and an inner circular layer, as is true of most parts of the digestive tract. However, it differs from other regions by having skeletal muscle in the superior part of the esophagus and smooth muscle in the inferior part. An **upper esophageal sphincter** and a **lower esophageal sphincter,** at the upper and lower ends of the esophagus, respectively, regulate the movement of materials into and out of the esophagus. The mucosal lining of the esophagus is moist stratified squamous epithelium. Numerous mucous glands in the submucosal layer produce a thick, lubricating mucus, which passes through ducts to the surface of the esophageal mucosa.

Swallowing Phases

Swallowing, or *deglutition,* is divided into three phases: (1) voluntary, (2) pharyngeal, and (3) esophageal. During the **voluntary phase** (figure 24.10, *step 1*), a bolus of food is formed in the mouth and pushed by the tongue against the hard palate, until it is forced toward the posterior part of the mouth and into the oropharynx.

The **pharyngeal phase** of swallowing (figure 24.10, *steps 2–4*) is a reflex initiated by the stimulation of tactile receptors in the area of the oropharynx. Afferent action potentials travel through the trigeminal (V) and glossopharyngeal (IX) nerves to the **swallowing center** in the medulla oblongata. There, they initiate action potentials in motor neurons, which pass through the trigeminal (V), glossopharyngeal (IX), vagus (X), and accessory (XI) nerves to the soft palate and pharynx. This phase of swallowing begins with the elevation of the soft palate, which closes the passage between the nasopharynx and oropharynx. The pharynx elevates to receive the bolus of food from the mouth and moves the bolus down the pharynx into the esophagus. The superior, middle, and inferior pharyngeal constrictor muscles contract in succession, forcing the food through the pharynx. At the same time, the upper esophageal sphincter relaxes, the elevated pharynx opens the esophagus, and food is pushed into the esophagus. This phase of swallowing is unconscious and is controlled automatically, even though the muscles involved are skeletal. The pharyngeal phase of swallowing lasts about 1–2 seconds.

During the pharyngeal phase, the vestibular folds and vocal cords close, and the **epiglottis** (ep-i-glot′is; on the glottis) is tipped posteriorly, so that the epiglottic cartilage covers the opening into the larynx, and the larynx is elevated. These movements prevent food from passing into the larynx.

Predict 2

Why is it important to close the opening between the nasopharynx and the oropharynx during swallowing? What may happen if a person emits an explosive burst of laughter while trying to swallow a liquid? Predict the consequences of trying to swallow and speak at the same time.

The **esophageal phase** of swallowing (figure 24.10, *step 5*), which takes about 5–8 seconds, is responsible for moving food from the pharynx to the stomach. Muscular contractions in the wall of the esophagus occur in peristaltic waves. Gravity helps move liquids and watery food through the esophagus. However, the peristaltic contractions in the esophagus are forceful enough to allow a person to swallow even while doing a headstand or floating in the zero-gravity environment of space.

As the peristaltic waves and the food bolus approach the stomach, the lower esophageal sphincter in the esophagus relaxes. This sphincter is not anatomically distinct from the rest of the esophagus, but it can be identified physiologically because it remains tonically constricted to prevent the reflux of stomach contents into the lower part of the esophagus.

The presence of food in the esophagus stimulates the myenteric plexus, which controls the peristaltic waves. Food in the esophagus also stimulates tactile receptors, which send afferent impulses to the medulla oblongata through the vagus nerves. Motor impulses, in turn, pass along the vagal efferent fibers to the skeletal and smooth muscles within the esophagus, thereby stimulating their contractions and reinforcing the peristaltic contractions.

ASSESS YOUR PROGRESS

26. *Name the parts of the pharynx involved with digestion. What are pharyngeal constrictors?*

27. *Where is the esophagus located? Describe the tunics of the esophageal wall and the esophageal sphincters.*

28. *What are the three phases of swallowing? Sequentially list the processes involved in the last two phases and describe how they are regulated.*

24.8 Stomach

LEARNING OUTCOMES

After reading this section, you should be able to

A. **Outline the anatomical and histological characteristics of the stomach.**

B. **Describe stomach secretions, their function, and their regulation.**

C. **Describe gastric movements and their regulation.**

The **stomach** is an enlarged segment of the digestive tract that primarily functions as a storage and mixing chamber. It is located in the left superior part of the abdomen (see figure 24.1). Its shape and size vary from person to person, even within the same individual from time to time, depending on food content and body posture. Nonetheless, several general anatomical features can be described.

Anatomy of the Stomach

The stomach is divided into four regions: (1) cardiac part, (2) fundus, (3) body, and (4) pyloric part (figure 24.11). The esophagus opens into the **cardiac part** of the stomach at the gastro-esophageal opening. The **lower esophageal sphincter,** also called the *cardiac*

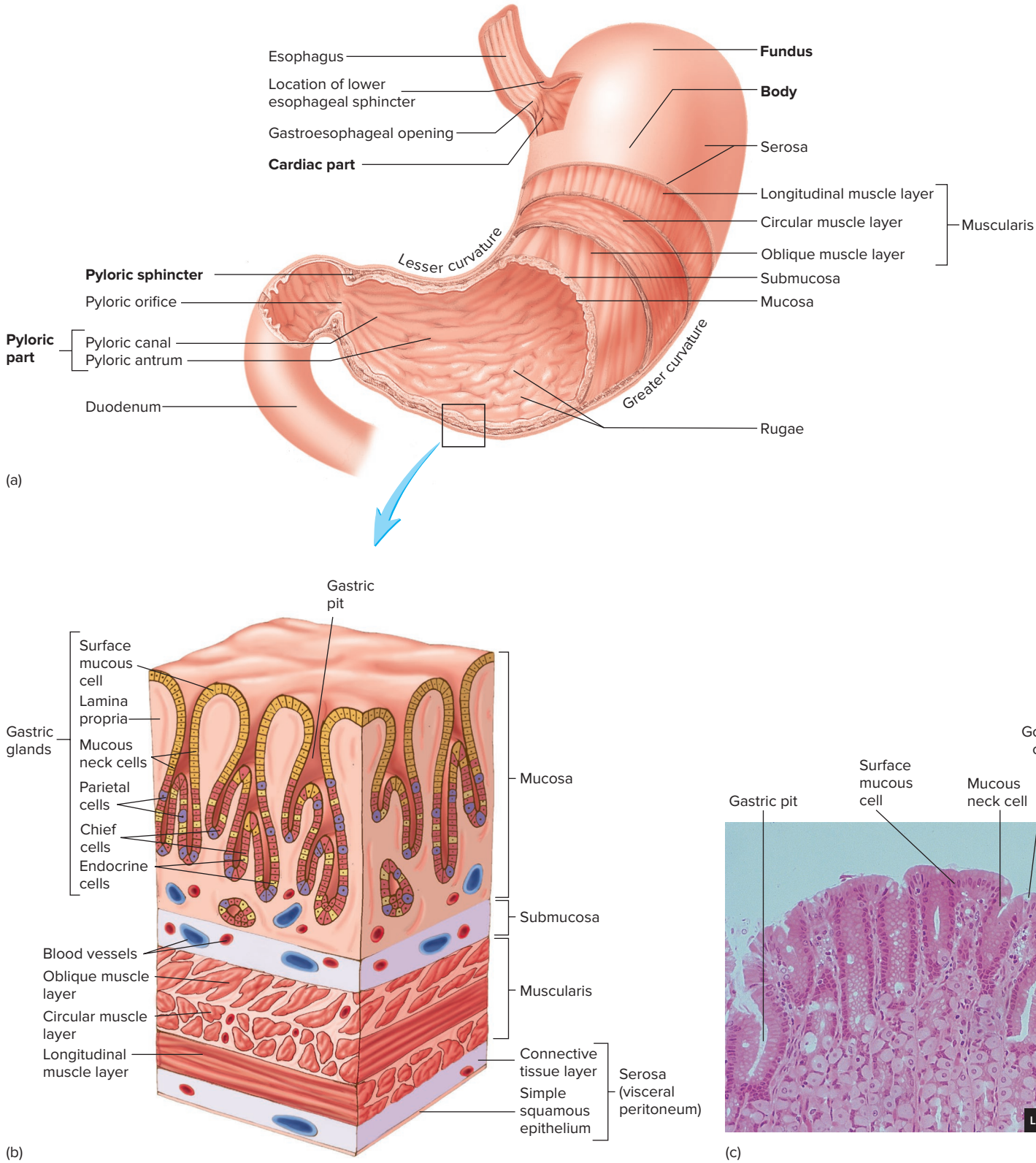


FIGURE 24.11 Anatomy and Histology of the Stomach

(*a*) Cutaway section reveals the muscular layers and internal anatomy of the stomach. (*b*) A section of the stomach wall illustrates its histology, including several gastric pits and glands. (c) Photomicrograph of gastric glands. (c) ©Victor Eroschenko APR

sphincter, surrounds the opening. Recall that, although this is an important structure in the normal function of the stomach, it is a physiological constrictor only and cannot be seen anatomically. The part of the stomach to the left of the cardiac part, the **fundus** (fŭn′dŭs), is actually superior to the cardiac opening. The largest part of the stomach is the **body,** which turns to the right, creating a *greater curvature* and a *lesser curvature.* The body narrows to form the funnel-shaped **pyloric** (pī-lōr′ik; gatekeeper) **part** of the stomach. The wider part of the funnel, toward the body of the stomach, is the **pyloric antrum.** The narrow part of the funnel is the **pyloric canal.** The pyloric canal opens through the pyloric orifice into the small intestine. The pyloric orifice is surrounded by the **pyloric sphincter,** or *pylorus,* a relatively thick ring of smooth muscle, which helps regulate the movement of gastric contents into the small intestine. **Hypertrophic pyloric stenosis** is a common defect of the stomach in infants, in which the pyloric sphincter is greatly thickened and thus interferes with normal stomach emptying.

Histology of the Stomach

The serosa, or *visceral peritoneum,* is the outermost tunic of the stomach. It consists of an outer layer of simple squamous epithelium and an inner layer of connective tissue. The muscularis of the stomach consists of three layers: (1) an outer longitudinal layer, (2) a middle circular layer, and (3) an inner oblique layer (figure 24.11*a*). The inner oblique layer is unique to the stomach wall. This layer helps generate the strong stomach contractions that physically break down ingested food into smaller particles. In some areas of the stomach, such as the fundus, the three layers blend with one another and cannot be separated. Deep to the muscularis are the submucosa and the mucosa, which are thrown into large folds called **rugae** (roo′gē; wrinkles) when the stomach is empty. These folds allow the mucosa and submucosa to stretch, and the folds disappear as the stomach volume increases as it is filled.

The mucous lining of the stomach is simple columnar epithelium. The epithelium forms numerous, tubelike invaginations called **gastric pits.** Gastric pits are the openings for the **gastric glands** that secrete acid and other substances (figure 24.11*b*). There are five types of epithelial cells of the stomach: surface mucous cells, mucous neck cells, parietal cells, chief cells, and endocrine cells. All but the surface mucous cells are found in the gastric glands.

1. **Surface mucous cells** are found on the surface around the gastric pit. These cells protect the stomach wall from being damaged by acid and digestive enzymes. The cells produce an alkaline mucus on their surface that neutralizes the acid and is a barrier to the digestive enzymes. The surface mucous cells are connected by tight junctions, which provide an additional barrier that prevents acids and enzymes from reaching deeper tissues. In addition, when surface mucous cells are damaged, they are rapidly replaced.
2. **Mucous neck cells** are located near the openings of the glands and produce mucus.
3. **Parietal cells** produce hydrochloric acid and intrinsic factor.
4. **Chief cells** produce the enzyme pepsinogen. They also produce the enzyme gastric lipase, which can digest lipids in the stomach.
5. **Endocrine cells** produce regulatory hormones and paracrine factors. There are several types of endocrine cells. Enterochromaffin-like cells produce histamine, which stimulates acid secretion by parietal cells. Gastrin-containing cells secrete gastrin, and somatostatin-containing cells secrete somatostatin, which inhibits gastrin and insulin secretion.

Secretions of the Stomach

Once food enters the stomach, it is mixed with stomach secretions to form a semifluid material called **chyme** (kīm; juice). The primary function of the stomach is to store and mix the chyme. Although some digestion and absorption occur in the stomach, they are not its major functions.

Stomach secretions include (1) hydrochloric acid, (2) intrinsic factor, (3) mucus, (4) digestive enzymes (pepsinogen and gastric lipase). The functions of these gastric secretions are summarized in table 24.2.

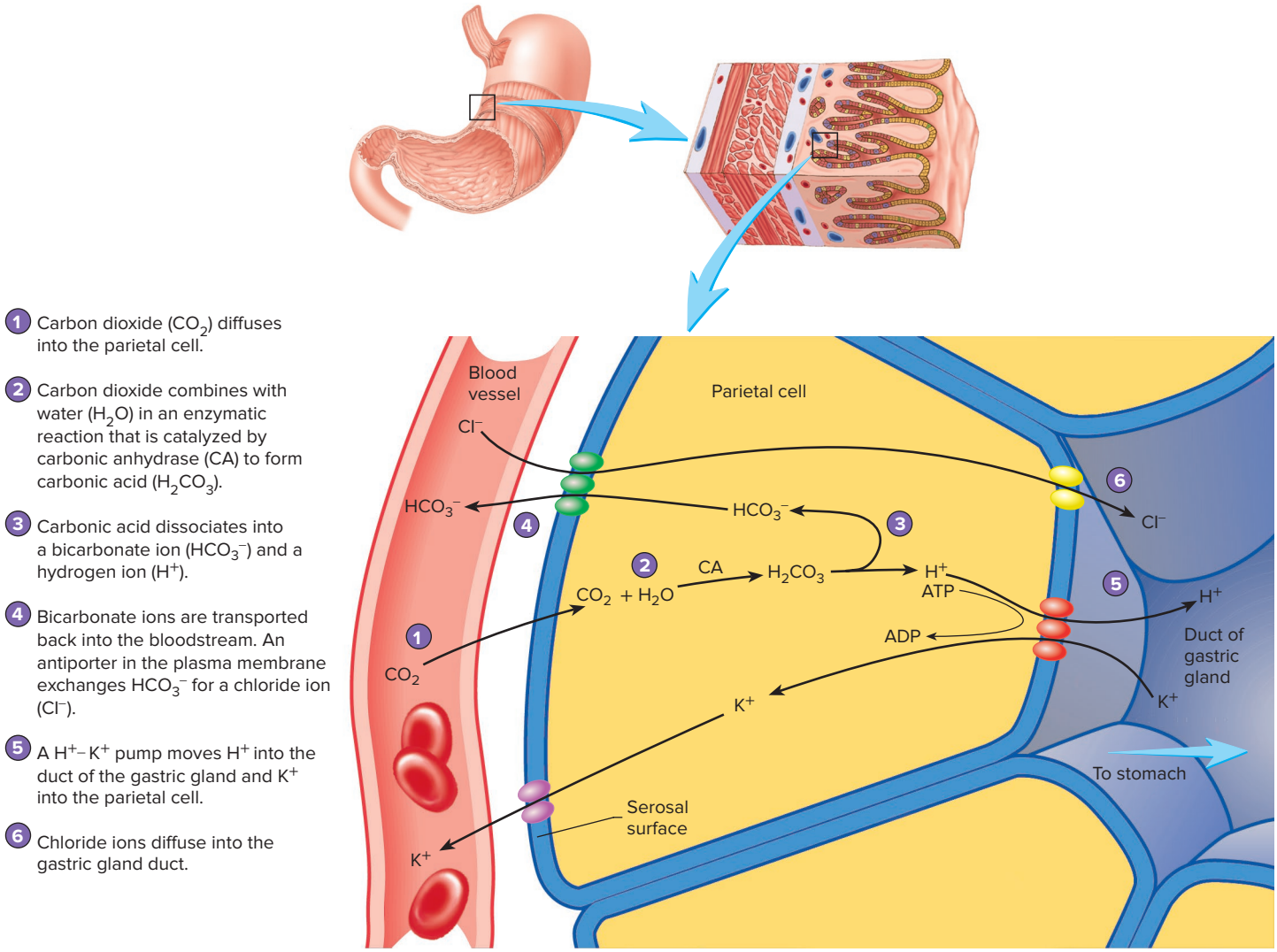
Parietal cells in the gastric glands of the pyloric region secrete a concentrated solution of hydrochloric acid. **Hydrochloric acid** produces the low pH of the stomach's contents, which is normally between 1 and 3. The key player in the formation of gastric acid is a H^+– K^+ exchange pump that is commonly called the **proton pump.** The proton pump actively transports hydrogen ions across the mucosal surface of the parietal cell into the lumen of the stomach (figure 24.12). The process begins with H^+ derived from CO_2 and water, which enter the parietal cell from its serosal surface (the side opposite the lumen of the gastric pit). Inside the cell the enzyme carbonic anhydrase catalyzes the reaction between CO_2 and water to form carbonic acid. Some of the carbonic acid molecules then dissociate to form H^+ and HCO_3^- (bicarbonate). Drugs that block the proton pump are used to lower gastric acid levels. The pump moves H^+ by active transport against a steep concentration gradient, and Cl^- diffuses from the cell through ion channels in the plasma membrane. Diffusion of Cl^- into the gastric gland duct balances the positively charged H^+ to reduce the amount of energy needed to transport the H^+ against both a concentration gradient and an electrical gradient.

While H^+ is pumped into the stomach lumen, bicarbonate ions move down their concentration gradient from the parietal cell into the extracellular fluid. During this process, HCO_3^- is exchanged for Cl^- through an antiporter, which is located in the plasma membrane, and the Cl^- subsequently moves into the cell. This results in an elevated blood pH in the veins that carry blood away from the stomach, called the **alkaline tide.** An alkaline tide normally occurs after eating a meal.

A major function of hydrochloric acid is to kill bacteria that are ingested with essentially everything humans put into their mouths. However, some pathogenic bacteria have an outer coat that resists stomach acids and one type of bacteria (*H. pylori*) is normally present in many human stomachs (see Clinical Impact 24.2).

The low pH of the stomach's contents has additional functions. Stomach acid denatures many proteins, so that proteolytic enzymes can reach internal peptide bonds. The acid environment provides the proper pH for the activation and function of pepsin. The acid also stops carbohydrate digestion by inactivating salivary amylase.

In addition to hydrochloric acid, parietal cells secrete intrinsic factor. **Intrinsic factor** is a glycoprotein that binds with vitamin B_{12}, making the vitamin more readily absorbed in the



PROCESS FIGURE 24.12 Hydrochloric Acid Production by Parietal Cells in the Gastric Glands of the Stomach

A series of steps involving carbonic anhydrase, a proton pump, and a HCO_3^-–Cl^- antiporter produce HCl in the gastric gland. AP|R

? *Predict what happens to the pH of blood in the veins leaving the stomach if someone has repeated episodes of vomiting.*

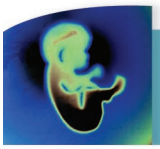
ileum of the small intestine. Vitamin B_{12} is important in deoxyribonucleic acid (DNA) synthesis, which is especially important for continual red blood cell production. A lack of vitamin B_{12} absorption leads to pernicious anemia (see chapter 19). Deficiency of vitamin B_{12} also leads to neurological symptoms, including peripheral neuropathy since vitamin B_{12} is required for maintaining myelin in the peripheral nervous system.

Chief cells within the gastric glands secrete **pepsinogen** (pep-sin′ō-jen). Pepsinogen is packaged in **zymogen** (zī-mō-jen) **granules,** which are released by exocytosis when pepsinogen secretion is stimulated. *Zymogen* is the term for an inactive enzyme. Once pepsinogen enters the lumen of the stomach, hydrochloric acid and previously formed pepsin molecules convert it to **pepsin.** Pepsin exhibits optimal enzymatic activity at a pH of 3 or less. Pepsin catalyzes the cleavage of some covalent bonds in proteins, thus breaking them into smaller peptide chains. Chief cells also secrete the enzyme gastric lipase, which can digest lipids even in an acidic environment.

The surface mucous cells and mucous neck cells secrete a viscous, alkaline mucus that covers the surface of the epithelial cells, forming a layer 1–1.5 mm thick. The thick layer of mucus lubricates and protects the epithelial cells of the stomach wall from the damaging effect of the acidic chyme and pepsin. Irritation of the stomach mucosa stimulates the secretion of a greater volume of mucus.

Regulation of Stomach Secretion

Approximately 2–3 L of gastric secretions (gastric juice) are produced each day. The amount and type of food entering the stomach and small intestine dramatically affect the quantity of gastric secretions, but up to 700 mL are secreted as a result of a typical meal. Both nervous and hormonal mechanisms regulate gastric secretions. The neural mechanisms involve reflexes integrated within the medulla oblongata and local reflexes integrated within the ENS. In addition, higher brain centers influence the reflexes. The chemical messengers that regulate



Clinical IMPACT 24.2 Peptic Ulcer

Approximately 10% of people in the United States will develop a **peptic ulcer** during their lifetime. Peptic ulcers are caused when the gastric juices (acid and pepsin) digest the mucosal lining of the digestive tract. Approximately 80% of peptic ulcers occur on the duodenal side of the pyloric sphincter, but peptic ulcers can also occur in the stomach (gastric ulcers) or esophagus (esophageal ulcers).

Nearly all peptic ulcers are due to infection by a specific bacterium, *Helicobacter pylori,* which is also linked to gastritis and gastric cancer. Because stress, diet, smoking, and alcohol cause excess acid secretion in the stomach, these lifestyle patterns were deemed responsible for ulcers for many years. Although these factors can contribute to ulcers, it is now clear that the root cause is *H. pylori.*

The presence of bacteria in the stomach mucosa was first discovered in 1892, but the finding was met with severe skepticism. In 1982, an Australian doctor, Barry Marshall, was finally able to culture an unusual bacterium, *H. pylori,* from stomach biopsies. To prove his belief that this bacterium can cause gastritis and ulcers, Marshall did something that no one should do at home (or even in a lab). He drank a solution of *H. pylori* and subsequently developed gastric inflammation. Luckily, antibiotic treatment was able to cure him. In 2005, along with his colleague, Dr. Robin Warren, he received the Nobel Prize in Physiology or Medicine for his discovery.

Antibiotic treatment to eradicate *H. pylori* is the best therapy for ulcers. A combination of antibiotics and antacids cures 95% of gastric and 74% of duodenal ulcers within 2 months, with less than a 10% recurrence rate. By contrast, the previous conventional treatment using antacids yields only temporary relief, with about 90% recurrence within a year. Other treatments involve drugs that prevent histamine-stimulated acid secretion or that directly inhibit the proton pumps that secrete the acid. Such treatments are effective only for short-term relief, not for long-term treatment.

Most bacteria cannot survive in the stomach. Hence, *H. pylori* is one of the most pervasive of human pathogens because it inhabits a niche without competition. Estimates suggest that well over half of the world's population is infected with *H. pylori.* The infection rate in the United States is about 1% per year of age—for example, 30% of all 30-year-olds are infected. In developing countries, nearly all people over age 25 are infected. This may contribute to the high rates of stomach cancer in some of those countries.

Analyses of the *H. pylori* DNA sequences from various ethnic and geographic populations suggest that *H. pylori* infection has been present in humans for over 150,000 years, yet only about 15–20% exhibit gastric problems attributed to *H. pylori.* What triggers the development of ulcers is a major unanswered question. It seems likely that both *H. pylori* infection and conditions that elevate acid secretion or damage the stomach wall, such as stress or the excessive ingestion of alcohol or aspirin, contribute to the development of an ulcer. For example, if a person is highly stressed, elevated sympathetic activity may inhibit duodenal gland secretion and increase the person's susceptibility to ulcers in the duodenum by reducing the protective coating of mucus on the duodenal wall.

stomach secretions include the hormones gastrin, secretin, and cholecystokinin (table 24.3), as well as the paracrine chemical messenger histamine.

The regulation of stomach secretion is divided into three phases: cephalic, gastric, and intestinal. The cephalic phase can be viewed as the "get started" phase, when stomach secretions are increased in anticipation of incoming food. This is followed by the "go for it" gastric phase, when most of the stimulation of secretion occurs. Finally, the intestinal phase is the "slow down" phase, during which stomach secretion decreases.

1. *Cephalic phase.* "Get started!" The cephalic phase is the brain phase of stomach secretion. It is controlled by the CNS. It begins even before the bolus of food enters the stomach. Several types of stimuli act on the centers within the medulla oblongata to influence gastric secretions (figure 24.13).

TABLE 24.3 Functions of the Major Gastrointestinal Hormones

Site of Production	Method of Stimulation	Secretory Effects	Motility Effects
Gastrin			
Stomach	Distension; partially digested proteins, autonomic stimulation, ingestion of alcohol or caffeine	Increases gastric secretion	Causes a minor increase in gastric motility
Secretin			
Duodenum	Acidity of chyme	Decreases gastric secretion; stimulates pancreatic and bile secretions high in bicarbonate ions	Decreases gastric motility
Cholecystokinin			
Duodenum	Fatty acids and peptides	Slightly decreases gastric secretion; stimulates pancreatic secretions high in digestive enzymes; causes contraction of the gallbladder and relaxation of the hepatopancreatic ampullar sphincter	Strongly decreases gastric motility

Cephalic Phase

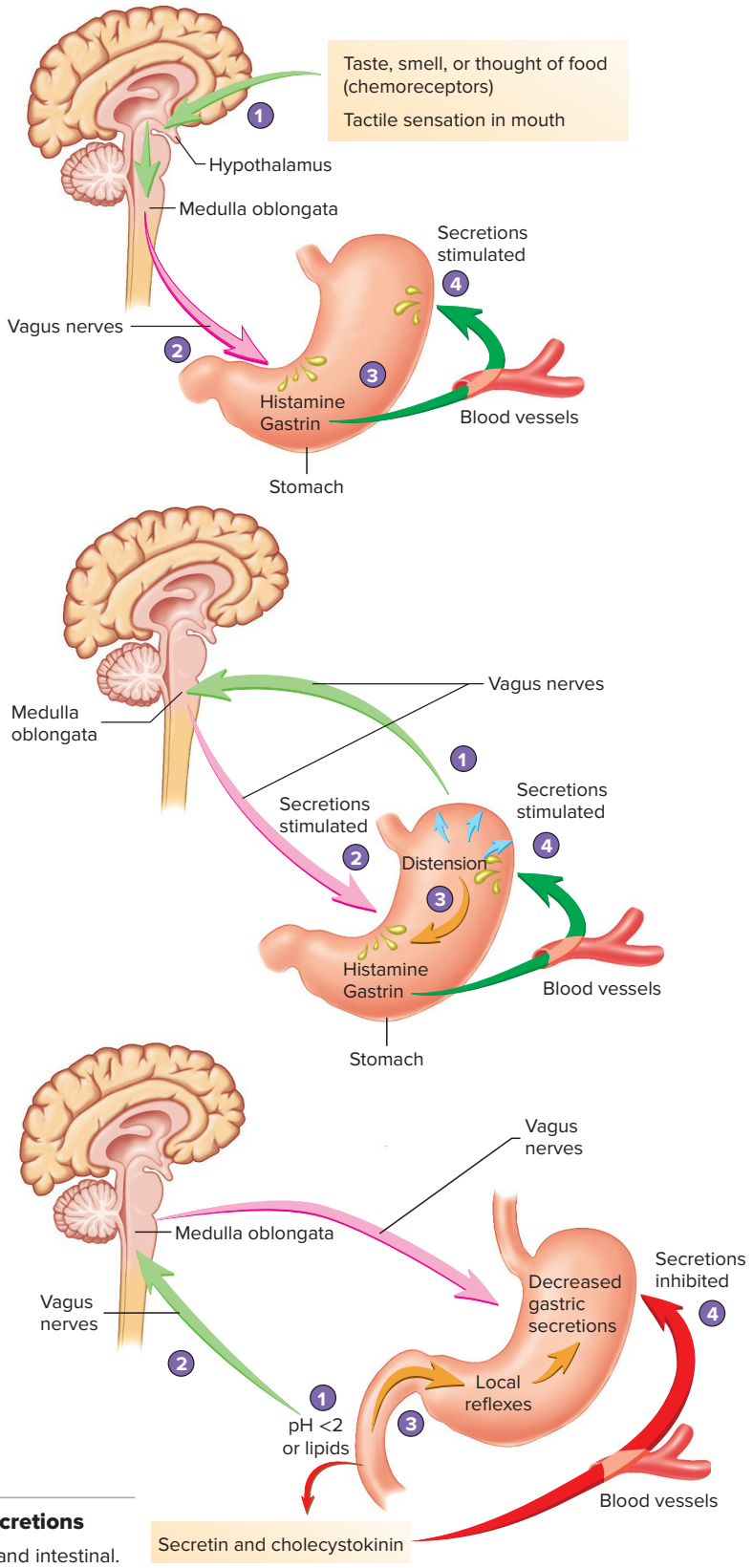
1. The taste, smell, or thought of food or tactile sensations of food in the mouth stimulate the medulla oblongata (*light green arrows*).
2. Vagus nerves carry parasympathetic action potentials to the stomach (*pink arrow*), where enteric plexus neurons are activated.
3. Postganglionic neurons stimulate secretion by parietal and chief cells and stimulate gastrin and histamine secretion by endocrine cells.
4. Gastrin is carried through the blood back to the stomach (*dark green arrow*), where, along with histamine, it stimulates secretion.

Gastric Phase

1. Distension of the stomach stimulates mechanoreceptors (stretch receptors) and activates a parasympathetic reflex. Action potentials generated by the mechanoreceptors are carried by the vagus nerves to the medulla oblongata (*light green arrow*).
2. The medulla oblongata increases action potentials in the vagus nerves that stimulate secretions by parietal and chief cells and stimulate gastrin and histamine secretion by endocrine cells (*pink arrow*).
3. Distension of the stomach also activates local reflexes that increase stomach secretions (*orange arrow*).
4. Gastrin is carried through the blood back to the stomach (*dark green arrow*), where, along with histamine, it stimulates secretion.

Intestinal Phase

1. Chyme in the duodenum with a pH less than 2 or containing lipids inhibits gastric secretions by three mechanisms (2–4).
2. Chemoreceptors in the duodenum are stimulated by H^+ (low pH) or lipids. Action potentials generated by the chemoreceptors are carried by the vagus nerves to the medulla oblongata (*light green arrow*), where they inhibit parasympathetic action potentials (*pink arrow*), thereby decreasing gastric secretions.
3. Local reflexes activated by H^+ or lipids also inhibit gastric secretion (*orange arrows*).
4. Secretin and cholecystokinin produced by the duodenum (*dark red arrows*) decrease gastric secretions in the stomach.



PROCESS FIGURE 24.13 Regulation of Stomach Secretions

There are three phases of stomach secretions: cephalic, gastric, and intestinal.

? *Why is it advantageous to the overall digestive process for secretin and cholecystokinin to slow stomach emptying upon initiation of the intestinal phase?*

These stimuli include the taste and smell of food, the stimulation of tactile receptors during the process of chewing and swallowing, and pleasant thoughts of food. Action potentials are sent from the medulla oblongata along parasympathetic neurons within the vagus (X) nerves to the stomach. Within the stomach wall, the preganglionic neurons stimulate the postganglionic neurons in the ENS. The postganglionic neurons, which are primarily cholinergic, stimulate secretory activity in the cells of the stomach mucosa.

Parasympathetic stimulation of the stomach mucosa results in the release of the neurotransmitter acetylcholine, which increases the secretory activity of both the parietal and the chief cells and stimulates the secretion of **gastrin** (gas′trin) and **histamine** from endocrine cells. The gastrin released into the circulation travels to the parietal cells, where it stimulates additional hydrochloric acid and pepsinogen secretion. In addition, gastrin stimulates enterochromaffin-like cells to release histamine, which stimulates parietal cells to secrete hydrochloric acid. Histamine acts as both a local paracrine chemical messenger and a hormone in the blood to stimulate gastric gland secretory activity. Acetylcholine, histamine, and gastrin working together cause a greater secretion of hydrochloric acid than any of them does separately. Of the three, histamine has the greatest stimulatory effect. Drugs that block the actions of histamine are used to lower acid levels.

2. *Gastric phase.* "Go for it!" The gastric phase of stomach secretion produces the greatest volume of gastric secretions. The presence of food in the stomach initiates the gastric phase (figure 24.13). The primary stimuli are distension of the stomach and the presence of amino acids and peptides in the stomach.

   Distension of the stomach wall, especially in the body or fundus, stimulates mechanoreceptors. Action potentials generated by these receptors initiate reflexes that involve both the CNS and the ENS. These reflexes result in acetylcholine release and the cascade of events that increase secretion, as in the cephalic phase. The presence of partially digested proteins or moderate amounts of alcohol or caffeine in the stomach also stimulates gastrin secretion.

   When the pH of the stomach contents falls below 2, increased gastric secretion produced by distension of the stomach is blocked. This negative-feedback mechanism limits the secretion of gastric juice.

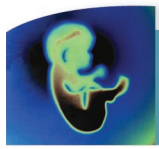
3. *Intestinal phase.* "Slow down!" The intestinal phase of gastric secretion inhibits gastric secretions (figure 24.13). It is initiated by acidic chyme entering the duodenum of the small intestine, which activates both neural and hormonal mechanisms. Secretions are inhibited when the pH of the chyme entering the duodenum drops to 2 or below. In addition, when the chyme contains lipid digestion products, gastric secretions are inhibited. There are two hormones involved in the intestinal phase: (1) secretin and (2) cholecystokinin.

   Secretin (se-krē′tin) is released in response to acidic solutions in the duodenum. Secretin inhibits gastric secretions by inhibiting both parietal and chief cells.

   Cholecystokinin (kō′lē-sis-tō-kī′nin) is released in response to fatty acids, other lipids, and to a lesser degree protein digestion products in the duodenum and the proximal jejunum. Cholecystokinin inhibits gastric secretions.

   The inhibition of gastric secretion is also under nervous control. The **enterogastric reflex** consists of a local reflex and a reflex integrated within the medulla oblongata that reduce gastric secretion. Distension of the duodenal wall, the presence of irritating substances in the duodenum, reduced pH, and hypertonic or hypotonic solutions in the duodenum activate the enterogastric reflex.

To summarize, gastric acid secretion is controlled by negative-feedback loops involving nerves and hormones. During the gastric phase, high acid levels in the stomach trigger a decrease in additional acid secretion. Then, during the intestinal phase, acidic chyme entering the duodenum triggers a decrease in stomach acid secretion. These negative-feedback loops ensure that the acidic chyme entering the duodenum is neutralized, which is required for the digestion of food by pancreatic and brush border enzymes in the intestine, and for the prevention of peptic ulcer formation.



Clinical IMPACT 24.3

Gastroesophageal Reflux

Gastroesophageal reflux is the reflux of acidic chyme from the stomach into the esophagus. Gastroesophageal reflux is commonly called *heartburn* because the refluxed acid causes a painful, burning sensation in the chest. The pain is usually short-lived but may be confused with the pain of an ulcer or a heart attack. The lower esophageal sphincter normally prevents acid reflux. Overeating (especially fatty and fried foods); lying down immediately after a meal; consuming too much alcohol or caffeine; overuse of nonsteroidal anti-inflammatory drugs, such as aspirin and ibuprofen; and smoking can all cause gastroesophageal reflux. Gastroesophageal reflux commonly occurs in infants, but they usually outgrow it by their first birthday.

Chronic reflux more than twice a week in infants or adults is more serious and is called **gastroesophageal reflux disease (GERD).** GERD in young infants can be difficult to diagnose. Women commonly experience GERD during pregnancy because of increased abdominal pressure from the fetus and higher levels of the hormone progesterone, which relaxes the lower esophageal sphincter.

For most adults, lifestyle changes and medications that decrease gastric acid secretion are sufficient to relieve the symptoms of GERD. Antacids that buffer gastric acid can also alleviate minor discomfort. One class of drugs acts by blocking the H_2 histamine receptors on parietal cells. H_2 receptors are different from the H_1 receptors involved in allergic reactions. Drugs that block allergic reactions do not affect histamine-mediated stomach acid secretion, and vice versa. The most effective inhibitors of gastric acid secretion are the proton pump inhibitors, such as omeprazole. These drugs inhibit the proton pumps on parietal cells, thus preventing acid secretion into the stomach. If not treated, GERD can lead to serious complications, including esophageal ulcers, scarring that constricts the esophagus, and esophageal cancer.

Predict 3

Alice, age 85, reported periods of laryngitis that began when she awakened in the morning and lasted a few days. Alice's physician used a laryngoscope to examine her vocal folds and upper trachea, which appeared inflamed. He prescribed an antacid and a drug to decrease H^+ secretion and told her to take the medications prior to going to bed at night. He also advised Alice to avoid eating just before bedtime. Explain the cause of her laryngitis and why the medications should relieve the symptoms.

Movements of the Stomach

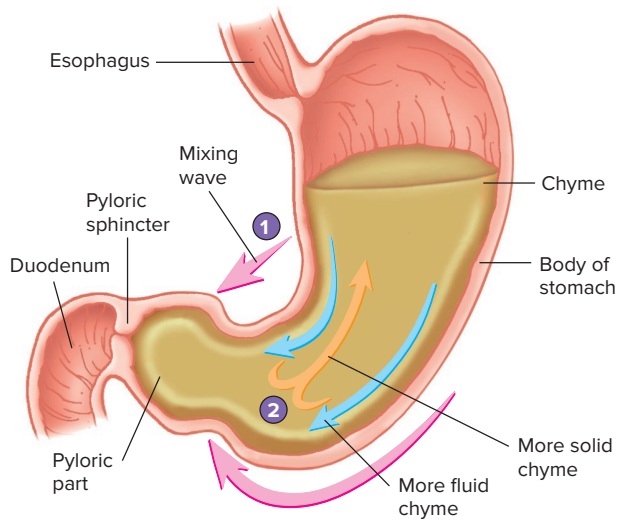
Stomach Filling

As food enters the stomach, the rugae flatten and the stomach volume increases up to 20-fold. This expansion allows the stomach to accommodate a large amount of food with very little increased pressure, until the stomach nears maximum capacity. Relaxation of the rugae is mediated by a reflex integrated within the medulla oblongata that inhibits muscle tone and pressure is further minimized by the ability of smooth muscle to stretch without an increase in tension (see chapter 9).

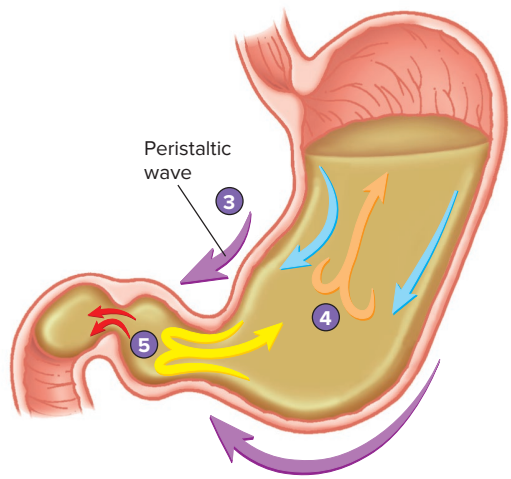
Mixing of Stomach Contents

Ingested food is thoroughly mixed with stomach gland secretions to form chyme. This mixing is accomplished by gentle **mixing waves,** which are contractions that occur about every 20 seconds. They proceed from the body of the stomach toward the pyloric sphincter. **Peristaltic waves** occur less frequently, are significantly more powerful than mixing waves, and force the chyme near the periphery of the stomach toward the pyloric sphincter. The more solid material near the center of the stomach is pushed superiorly toward the cardiac part for further digestion (figure 24.14). Roughly 80% of the contractions are mixing waves, and 20% are peristaltic waves. The back-and-forth movement of the chyme effectively mixes the ingested food with gastric juice.

1. A mixing wave initiated in the body of the stomach progresses toward the pyloric sphincter (*pink arrows directed inward*).
2. The more fluid part of the chyme is pushed toward the pyloric sphincter (*blue arrows*), whereas the more solid center of the chyme squeezes past the peristaltic constriction back toward the body of the stomach (*orange arrow*).



3. Peristaltic waves (*purple arrows*) move in the same direction and in the same way as the mixing waves but are stronger.
4. Again, the more fluid part of the chyme is pushed toward the pyloric region (*blue arrows*), whereas the more solid center of the chyme squeezes past the peristaltic constriction back toward the body of the stomach (*orange arrow*).
5. Peristaltic contractions force a few milliliters of the mostly fluid chyme through the pyloric opening into the duodenum (*small red arrows*). Most of the chyme, including the more solid portion, is forced back toward the body of the stomach for further mixing (*yellow arrow*).



PROCESS FIGURE 24.14 Movements in the Stomach

Food is mixed with hydrochloric acid and other secretions in the stomach to create chyme.

? *In a person suffering from heartburn, the gastroesophageal opening is weakened and stomach acid can be forced from the stomach back into the esophagus. Explain why medications that slow stomach movements and emptying may worsen heartburn symptoms.*

Stomach Emptying

The amount of time food remains in the stomach depends on a number of factors, including the type and volume of food. Liquids rapidly exit the stomach within minutes and are fully gone 1½–2½ hours after ingestion. After a typical meal, the stomach is usually empty within 3–4 hours. The pyloric sphincter normally remains partially closed because of mild tonic contraction. Each peristaltic contraction is strong enough to force a small amount of chyme through the pyloric opening and into the duodenum. The peristaltic contractions responsible for moving chyme through the partially closed pyloric opening are called the **pyloric pump.** In general, increased motility leads to increased emptying. In an empty stomach, peristaltic contractions that approach tetanic contractions can occur for about 2–3 minutes. The contractions are increased by low blood glucose levels and are strong enough to create uncomfortable sensations called **hunger pangs.** Hunger pangs usually begin 12–24 hours after a meal, in less time for some people. If nothing is ingested, hunger pangs reach their maximum intensity within 3–4 days and then become progressively weaker.

Regulation of Stomach Emptying

If the stomach empties too fast, the efficiency of digestion and absorption is reduced, and acidic gastric contents dumped into the duodenum may damage its lining. However, if the rate of emptying is too slow, then the slow delivery to the small intestine will reduce the rate at which nutrients are digested and absorbed. In addition, the highly acidic contents of the stomach may damage the stomach wall. To prevent these two extremes, stomach emptying is regulated.

The neural mechanisms that stimulate stomach secretions are also involved with increasing stomach motility. The major stimulus for both motility and secretion is distension of the stomach wall. Increased stomach motility increases stomach emptying. Conversely, the hormonal and neural mechanisms associated with the duodenum that decrease gastric secretions also decrease gastric motility and increase constriction of the pyloric sphincter. The enterogastric reflex and the hormone cholecystokinin are major inhibitors of gastric motility. The result is a decrease in the rate of stomach emptying.

A meal of polysaccharide carbohydrates (starch and glycogen) has the fastest clearance time from the stomach, typically 1 hour. For comparison, a meal heavy with dietary fats and proteins takes up to 6 hours to clear from the stomach. A major reason for this difference is that a fatty meal increases the release of cholecystokinin, which is a major inhibitor of stomach emptying.

Vomiting is usually a protective mechanism against the ingestion of toxic or harmful substances. Vomiting can result from irritation (e.g., overdistension or overexcitation) anywhere along the digestive tract. Action potentials travel through the vagus nerve and spinal visceral afferent nerves to the vomiting center in the medulla oblongata. Once the vomiting center is stimulated and the reflex is initiated, the following events occur: (1) A deep breath is taken; (2) the hyoid bone and larynx are elevated, opening the upper esophageal sphincter; (3) the opening of the larynx is closed; (4) the soft palate is elevated, closing the connection between the oropharynx and the nasopharynx; (5) the diaphragm and abdominal muscles are forcefully contracted, strongly compressing the stomach and increasing the intragastric pressure; (6) the lower esophageal sphincter is relaxed; and (7) the gastric contents are forced out of the stomach, through the esophagus and oral cavity, to the outside.

ASSESS YOUR PROGRESS

29. *Describe the parts of the stomach. List the tunics of the stomach wall. How is the stomach wall different from the esophagus wall?*
30. *What are gastric pits and gastric glands?*
31. *Name the types of cells in the stomach and the secretions they produce. What are the functions of the secretions?*
32. *Describe the three phases of regulation of stomach secretion.*
33. *How are gastric secretions inhibited? Why is this inhibition necessary?*
34. *As the stomach fills, why does the pressure not greatly increase until maximum volume is reached?*
35. *Name the two kinds of stomach movements. How are stomach movements regulated by hormones and nervous control?*

24.9 Small Intestine

LEARNING OUTCOMES

After reading this section, you should be able to

A. **List the sections of the small intestine and describe the characteristics that account for its large surface area.**
B. **Name the four major cell types of the duodenal mucosa and describe their functions.**
C. **Describe the secretions and movements of the small intestine.**

The **small intestine** consists of three parts: (1) duodenum, (2) jejunum, and (3) ileum (figure 24.15). The entire small intestine is about 6 m long (range: 4.6–9 m). The duodenum is about 25 cm long

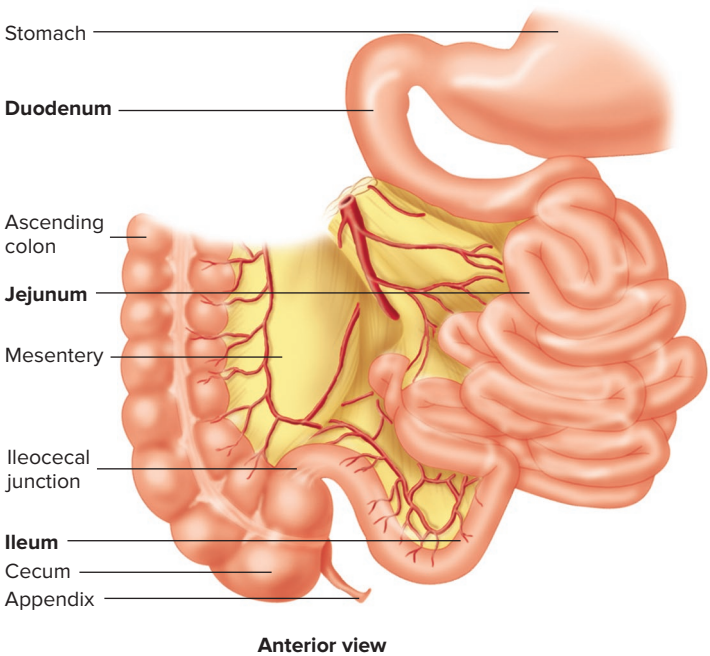


FIGURE 24.15 Small Intestine
The small intestine consists of three segments: the duodenum, jejunum, and ileum. AP|R

(*duodenum* means 12, suggesting that it is 12 inches long). The jejunum, constituting about two-fifths of the total length of the small intestine, is about 2.5 m long. The ileum, constituting three-fifths of the small intestine, is about 3.5 m long. Two major accessory glands, the liver and the pancreas, are associated with the duodenum.

The small intestine is where the greatest amount of digestion and absorption of nutrients and water occurs. Within the small intestine, the duodenum and jejunum are the major sites of nutrient absorption, although some absorption occurs in the ileum. Most of the water is absorbed by osmosis, along with the absorbed nutrients in the duodenum and jejunum. Over 90% of the water is absorbed before the colon.

Anatomy and Histology of the Small Intestine

Duodenum

The **duodenum** (doo-ō-dē′nŭm, doo-od′ĕ-nŭm) is the shortest part of the small intestine. The structure of the duodenum begins with a nearly 180-degree arc from where it leaves the stomach and curves around the head of the pancreas within the abdominal cavity (figure 24.16*a*). This short, superior part ends in a sharp bend, where it joins the jejunum. Within the duodenum, about two-thirds of the way down the descending part, are two small mounds: the **major duodenal papilla** and the **minor duodenal papilla.** Ducts from the liver and/or pancreas open at these papillae.

The surface of the duodenum has three modifications that increase its area about 600-fold to allow for more efficient digestion and absorption of food. All three of these modifications increase the surface area of the small intestine, which greatly enhances absorption.

1. The mucosa and submucosa form a series of folds called the **circular folds,** or *plicae* (plī′sē) *circulares* (figure 24.16*a*), which run perpendicular to the long axis of the digestive tract.
2. Tiny, fingerlike projections of the mucosa form numerous **villi** (vil′ī), which are 0.5–1.5 mm in length (figure 24.16*c*). Each villus is covered by simple columnar epithelium and contains a blood capillary network and a lymphatic capillary called a **lacteal** (lak′tē-ăl; figure 24.16*d*).
3. Most of the cells that make up the surface of the villi have numerous cytoplasmic extensions (about 1 μm long) called **microvilli,** which further increase the surface area (figure 24.16*e*). The combined microvilli on the entire epithelial surface form the **brush border.**

The mucosa of the duodenum is simple columnar epithelium with four major cell types: (1) absorptive cells, (2) goblet cells, (3) granular cells, and (4) endocrine cells. **Absorptive cells** are cells with microvilli that produce digestive enzymes and absorb digested food. **Goblet cells** produce a protective mucus. **Granular cells,** or *Paneth cells,* may help protect the intestinal epithelium from bacteria. **Endocrine cells** produce regulatory hormones. The hormones secretin and cholecystokinin stimulate hepatic and pancreatic secretions (see table 24.3, figures 24.21 and 24.24).

The epithelial cells are produced within tubular invaginations of the mucosa, called **intestinal glands,** or *crypts of Lieberkühn,* at the base of the villi. The absorptive and goblet cells migrate from the intestinal glands to cover the surface of the villi and are eventually shed from its tip. The granular and endocrine cells remain in the bottom of the glands. The submucosa of the duodenum contains coiled, tubular mucous glands called **duodenal glands,** or *Brunner glands,* which open into the base of the intestinal glands.

Jejunum and Ileum

The **jejunum** (jĕ-joo′nŭm) and **ileum** (il′ē-ŭm) are similar in structure to the duodenum (see figure 24.15). However, progressing from the duodenum through the ileum, there are gradual decreases in the diameter of the small intestine, the thickness of the intestinal wall, the number of circular folds, and the number of villi.

Lymphatic nodules called **Peyer patches** are numerous in the mucosa and submucosa of the ileum. Peyer patches and other mucosa-associated lymphoid tissue in the digestive tract initiate immune responses against microorganisms that enter the mucosa from ingested food (see chapter 22).

The site where the ileum connects to the cecum of the large intestine is the **ileocecal junction.** The junction is a ring of smooth muscle, the **ileocecal sphincter,** and a one-way **ileocecal valve.** Together, the sphincter and valve allow intestinal contents to move from the ileum to the large intestine, but not in the opposite direction (see figures 24.15 and 24.25).

Secretions of the Small Intestine

The mucosa of the small intestine produces secretions that contain primarily mucus, electrolytes, and water that lubricate and protect the intestinal wall.

1. Mucus is secreted from the duodenal glands, intestinal glands, and goblet cells. It protects the wall of the intestine from the irritating effects of acidic chyme and from the digestive enzymes that enter the duodenum from the pancreas. Secretions from the duodenal glands are stimulated by the vagus nerve, secretin, and chemical or tactile irritation of the duodenal mucosa. Chemical and tactile irritation of the mucosa also stimulate goblet cells to produce mucus.
2. Secretion of electrolytes and water from the intestinal epithelium helps keep the chyme in a liquid form to facilitate the digestive process by pancreatic enzymes and brush border enzymes (see table 24.2).

Enzymes of the intestinal mucosa are not actually secreted, but are bound to the membranes of the absorptive cell microvilli. These surface-bound enzymes include **disaccharidases,** which break down disaccharides to monosaccharides, and **peptidases,** which hydrolyze the peptide bonds between small amino acid chains (see table 24.2). Although these enzymes are not secreted into the intestine, they influence the digestive process significantly, and the large surface area of the intestinal epithelium brings these enzymes into contact with the intestinal contents. Small molecules, which are breakdown products of digestion, are absorbed through the microvilli and enter the circulatory or lymphatic system.

Movement in the Small Intestine

Movement in the small intestine involves mixing of the chyme and slow propulsion down the tract. Segmental contractions (see figure 24.3) mix the intestinal contents, and peristaltic contractions (see figure 24.4) primarily propel the intestinal contents along the

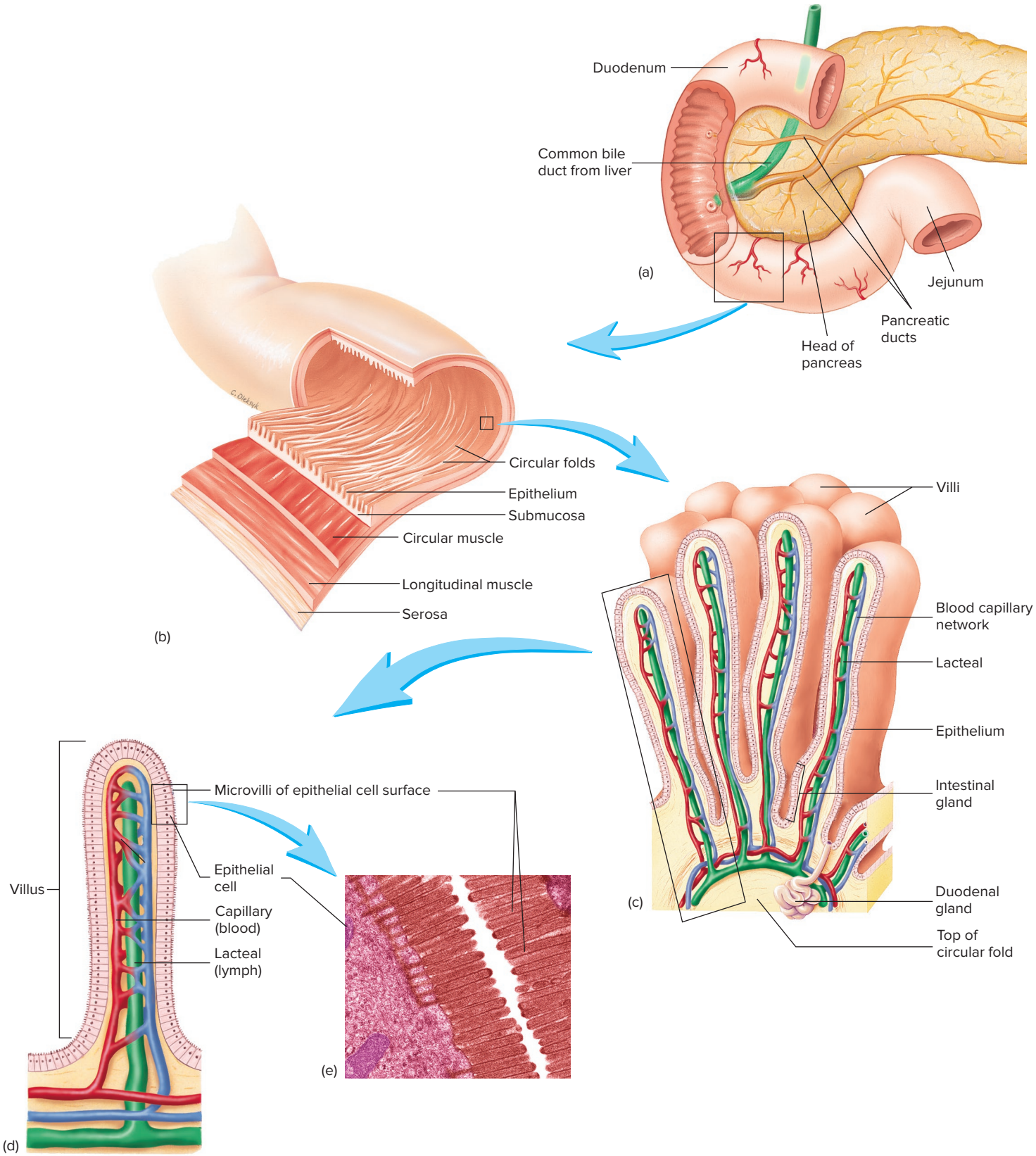


FIGURE 24.16 Anatomy and Histology of the Duodenum

(*a*) Ducts from the liver and pancreas empty into the duodenum. (*b*) Wall of the duodenum, showing the circular folds. (*c*) The villi on a circular fold. (*d*) A single villus, showing the lacteal and capillary network. (*e*) Transmission electron micrograph of microvilli on the surface of a villus. (e) ©Science Photo Library RF/Getty Images

digestive tract. The peristaltic contractions are generally propagated for only short distances, but a few may proceed the entire length of the intestine. Frequently, intestinal peristaltic contractions are continuations of peristaltic contractions that begin in the stomach. These contractions both mix and propel substances through the small intestine as the wave of contraction proceeds. The contractions move at a rate of about 1 cm/min. It usually takes 3–5 hours for chyme to move from the pyloric region to the ileocecal junction.

Local mechanical and chemical stimuli are especially important in regulating the motility of the small intestine. Smooth muscle contraction increases in response to distension of the intestinal wall. Solutions that are either hypertonic or hypotonic, solutions with a low pH, and certain products of digestion, such as amino acids and peptides, also stimulate contractions of the small intestine. Local reflexes, which are integrated within the ENS of the small intestine, mediate the intestine's response to these mechanical and chemical stimuli. Stimulation through parasympathetic nerve fibers may also increase the intestine's motility, but the parasympathetic influences in the intestine are not as important as those in the stomach.

The ileocecal sphincter at the juncture between the ileum and the large intestine remains mildly contracted most of the time, but peristaltic waves reaching it from the small intestine cause it to relax and allow the chyme to move from the small intestine into the cecum. Cecal distension, however, initiates a local reflex that causes more intense constriction of the ileocecal sphincter. Closure of the sphincter facilitates digestion and absorption in the small intestine by slowing the rate of chyme movement from the small intestine into the large intestine and prevents material from returning to the ileum from the cecum.

Predict 4

Amos suffers from intermittent pain in the epigastric area that begins about 2 or 3 hours after eating. The pain is relieved by taking an antacid. An endoscopic exam identified duodenal ulcers and Amos's physician recommended antacids and an antibiotic. Amos wondered why he could not control the condition with antacids alone, but his physician was worried about perforation of the duodenum. Explain why Amos's physician prescribed both antacids and antibiotics. How could the lack of antibiotics lead to perforation of the duodenum?

ASSESS YOUR PROGRESS

36. *Name and describe the three parts of the small intestine.*

37. *What are the circular folds, villi, and microvilli in the small intestine? What are their functions?*

38. *Name the four types of cells found in the duodenal mucosa, and state their functions.*

39. *What are the functions of the intestinal glands and duodenal glands? State the factors that stimulate secretion from the duodenal glands and from goblet cells.*

40. *List the enzymes of the small intestine wall, and give their functions.*

41. *What are the two kinds of movement of the small intestine? How are they regulated?*

42. *What is the function of the ileocecal sphincter and valve?*

24.10 Liver

LEARNING OUTCOMES

After reading this section, you should be able to

A. **Describe the anatomy, histology, and ducts of the liver.**

B. **Describe the major functions of the liver and explain how they are regulated.**

Anatomy of the Liver

The **liver** is the largest internal organ of the body, weighing about 1.36 kg (3 pounds). It is in the right-upper quadrant of the abdomen, tucked against the inferior surface of the diaphragm (figure 24.17; see figure 24.1). The liver consists of two major lobes, (1) the **right lobe** and (2) the **left lobe.** The right and left lobes are separated by a connective tissue septum, the falciform ligament. Two minor lobes, (1) the **caudate lobe** and (2) the **quadrate lobe,** can be seen from an inferior view, along with the porta.

The **porta** (gate) is on the inferior surface of the liver, where blood vessels and nerves enter and bile ducts and lymphatic vessels leave the liver (figure 24.17*b*). Blood flows into the liver via the **hepatic** (he-pat′ik) **portal vein** and the **hepatic artery.** Bile flows out of the liver via two hepatic ducts, one each from the right and left lobes, exit the liver at the porta.

The duct system that conveys bile from the liver also receives ducts from the gallbladder and pancreas before connecting to the small intestine. The right and left hepatic ducts first unite to form a single **common hepatic duct** (figure 24.18). The **cystic duct** from the gallbladder joins the common hepatic duct to form the **common bile duct.** The common bile duct joins the pancreatic duct at the **hepatopancreatic ampulla** (hĕ-pat′ō-pan-crē-at′ik am-pul′lă), which is an enlargement where the two ducts merge. The hepatopancreatic ampulla empties into the duodenum at the major duodenal papilla (figure 24.18). A smooth muscle sphincter surrounds the common bile duct where it enters the hepatopancreatic ampulla.

The gallbladder is a small sac on the inferior surface of the liver that stores bile. Bile can flow from the gallbladder through the cystic duct into the common bile duct, or it can flow back up the cystic duct into the gallbladder.

Histology of the Liver

A connective tissue capsule and visceral peritoneum cover the liver, except for the **bare area,** a small area on the diaphragmatic surface that lacks a visceral peritoneum and is surrounded by the coronary ligament (see figure 24.17*c*). At the porta, the connective tissue capsule sends a branching network of septa (walls) into the substance of the liver to provide its main support. Vessels, nerves, and ducts follow the connective tissue branches throughout the liver.

The liver is divided into **hepatic lobules.** Hepatic lobules are hexagon-shaped regions surrounded by connective tissue septa and defined by a **portal triad** at each corner and a **central vein** in the center of the lobule (figure 24.19). The portal triads are so named because three structures—the (1) hepatic portal vein, (2) hepatic artery, and (3) hepatic duct—are located in them. Hepatic nerves and lymphatic vessels, often too small to be seen easily in light

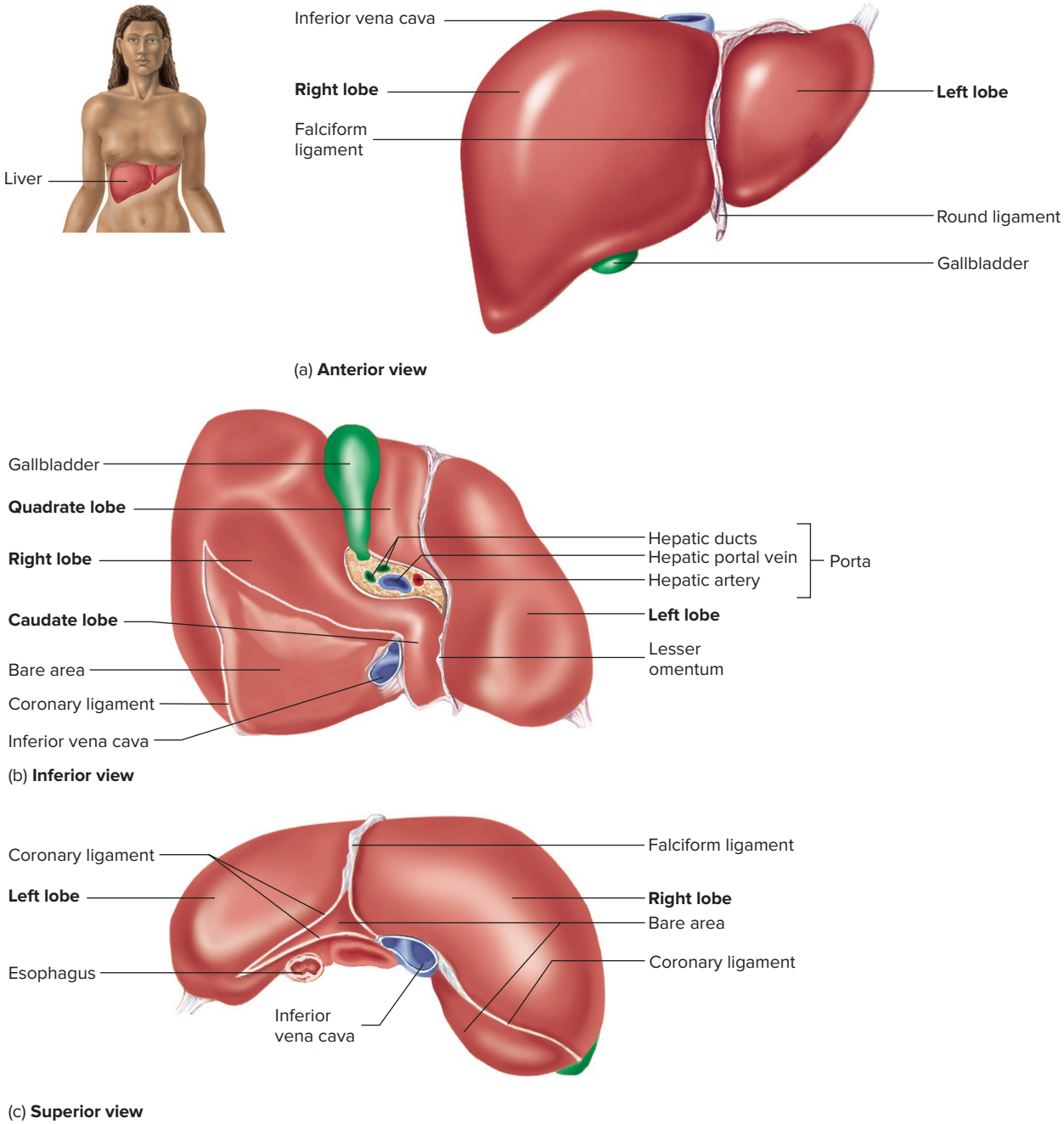


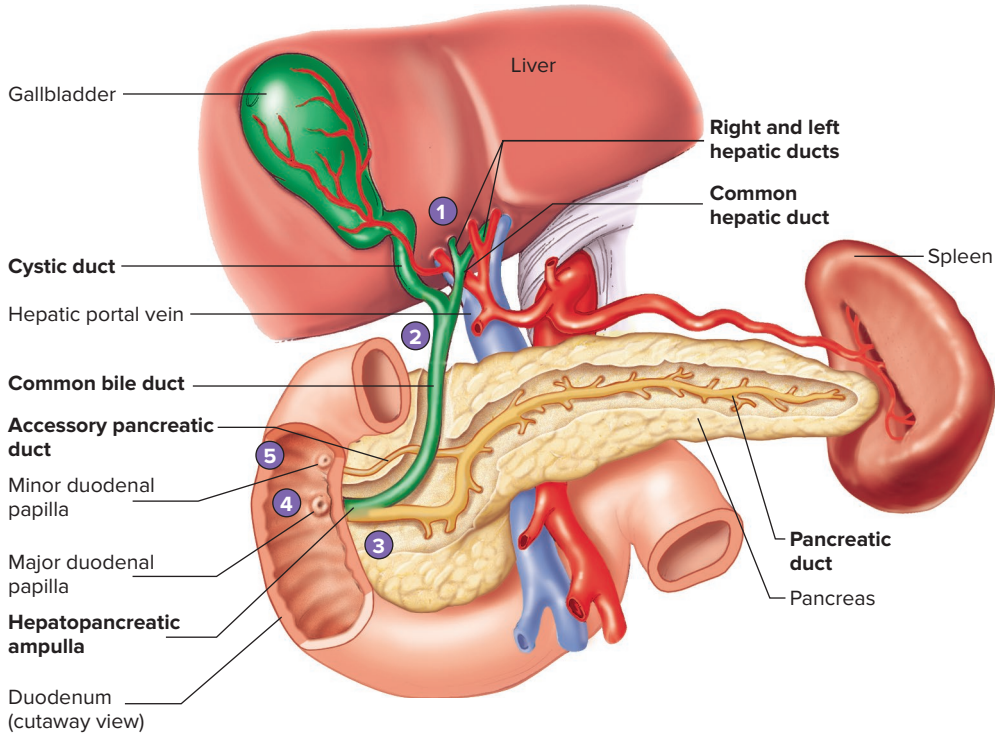
FIGURE 24.17 Liver
(*a*) Anterior view. (*b*) Inferior view. (*c*) Superior view. AP|R

micrographs, are also located in these areas. The central vein collects the blood as it leaves the lobule. Central veins of the lobules unite to form **hepatic veins,** which exit the liver on its posterior and superior surfaces and empty into the inferior vena cava (figure 24.19).

Hepatic cords are strings of cells that radiate out from the central vein of each lobule like the spokes of a wheel. The hepatic cords are composed of **hepatocytes,** the functional cells of the liver. Hepatocytes take up nutrients from the portal blood. The nutrients are stored, detoxified, or used to synthesize new compounds before being released into the hepatic sinusoids or into the bile canaliculi. **Hepatic sinusoids** are blood channels in the spaces between the hepatic cords. The sinusoids are lined with a very thin, irregular squamous endothelium consisting of two cell populations: (1) extremely thin, sparse **endothelial cells** and (2) **hepatic phagocytic cells** called Kupffer cells. The **bile canaliculus** (kan-ă-lik′ū-lŭs; little canal) is a cleftlike lumen that lies between the cells within each cord (figure 24.19).

Blood from the digestive tract first flows into the liver. Nutrient-rich, deoxygenated blood from the viscera enters the hepatic sinusoids from branches of the hepatic portal vein and mixes with nutrient-depleted, oxygenated blood from the hepatic arteries (figure 24.20). Mixed blood in the hepatic sinusoids flows

1. The hepatic ducts, which carry bile from the liver lobes, combine to form the common hepatic duct.
2. The common hepatic duct combines with the cystic duct from the gallbladder to form the common bile duct.
3. The common bile duct and the pancreatic duct combine to form the hepatopancreatic ampulla.
4. The hepatopancreatic ampulla empties bile and pancreatic secretions into the duodenum at the major duodenal papilla.
5. The accessory pancreatic duct empties pancreatic secretions into the duodenum at the minor duodenal papilla.



PROCESS FIGURE 24.18 Flow of Bile and Pancreatic Secretions Through the Duct System of the Liver, Gallbladder, and Pancreas

The liver produces bile, some of which is stored in the gallbladder. The bile enters the duodenum through the common bile duct.

? *If gallstones have formed in the gallbladder, what consequence could they have on release of bile from the gallbladder?*

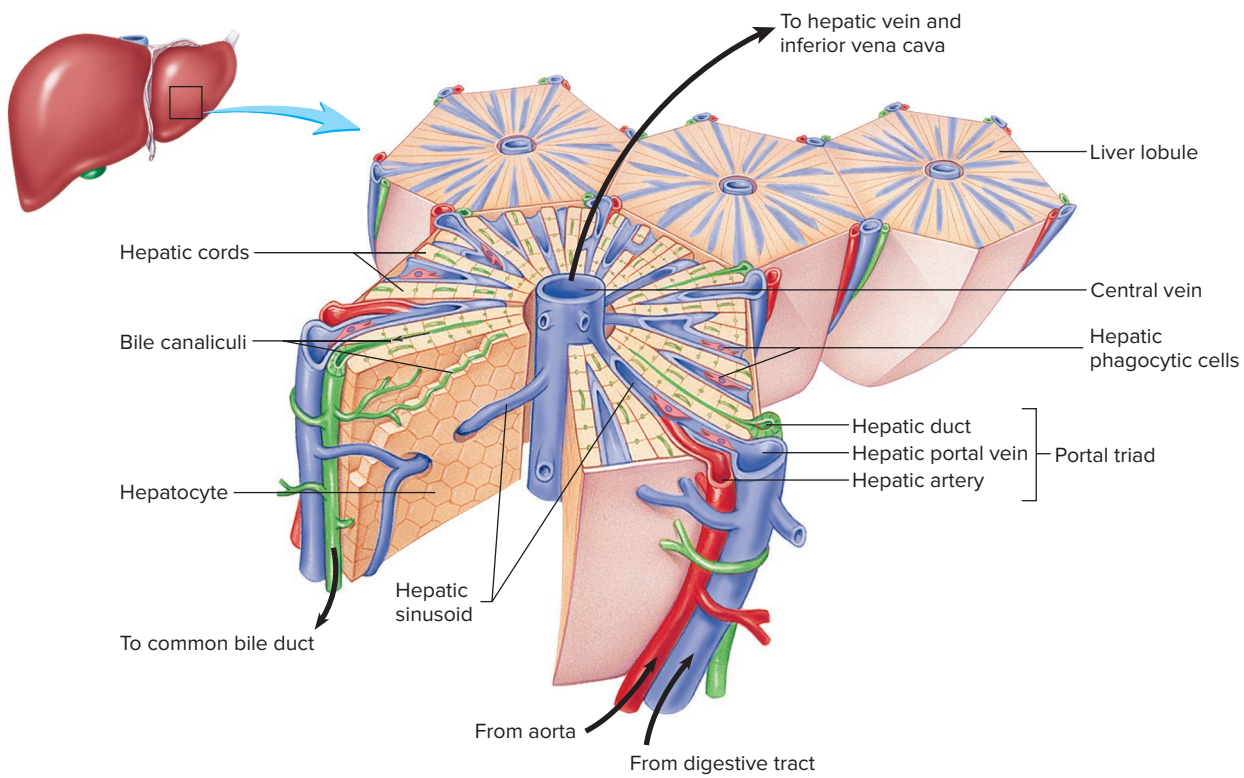
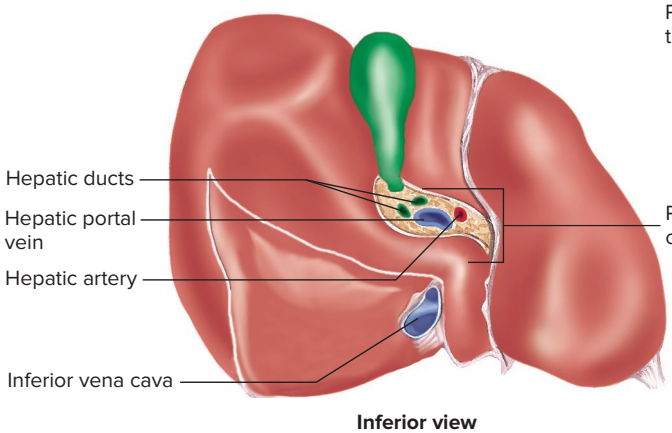


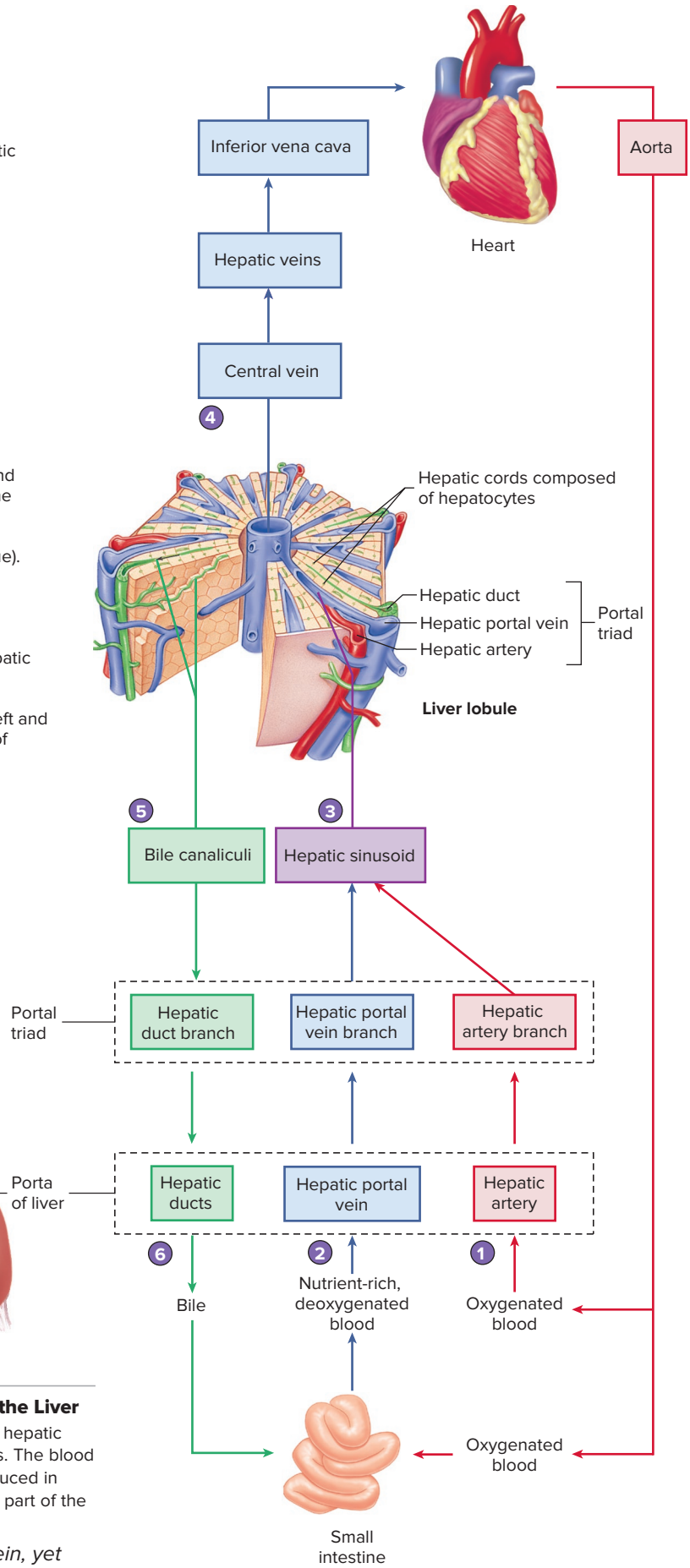
FIGURE 24.19 Histology of Hepatic Lobules in the Liver

The liver consists of hexagon-shaped lobules with a portal triad at each corner. A central vein is located in the center of each lobule. AP|R

1. The hepatic artery (red) carries oxygenated blood from the aorta through the porta of the liver. Hepatic artery branches become part of the portal triads. Blood from the hepatic artery branches enters the hepatic sinusoids and supplies hepatocytes in the hepatic cords with oxygen.
2. The hepatic portal vein (blue) carries nutrient-rich, deoxygenated blood from the intestines through the porta of the liver. Hepatic portal vein branches become part of the portal triads. Blood from the hepatic portal vein branches enters the hepatic sinusoids and supplies hepatocytes in the hepatic cords with nutrients.
3. Blood in the hepatic sinusoids that comes from the hepatic artery and hepatic portal vein (purple) picks up plasma proteins, processed molecules, and waste products produced by the hepatocytes of the hepatic cords.
4. The hepatic sinusoids empty into central veins (blue). The central veins connect to hepatic veins, which connect to the inferior vena cava.
5. Bile produced by hepatocytes in the hepatic cords enters bile canaliculi (green), which connect to hepatic duct branches that are part of the portal triads.
6. The hepatic duct branches converge to form the left and right hepatic ducts, which carry bile out the porta of the liver.



Inferior view



PROCESS FIGURE 24.20 Blood and Bile Flow Through the Liver

Blood enters the porta of the liver through the hepatic portal vein and hepatic artery, which branch to become part of the portal triads of liver lobules. The blood then empties into the central vein and is sent to the heart. Bile is produced in the liver and leaves the lobules via the hepatic duct branches that are part of the portal triad.

? *Explain why the portal triad has both an artery and a vein, yet both have blood flowing in the same direction into the liver.*

to the central vein, where it exits the lobule and then exits the liver through the hepatic veins. **Bile** produced by the hepatocytes flows through the bile canaliculi toward the hepatic triad and exits the liver through the hepatic ducts. Blood, therefore, flows from the triad toward the center of each lobule, whereas bile flows away from the center of the lobule toward the triad.

In the fetus, special blood vessels bypass the liver sinusoids. The remnants of fetal blood vessels can be seen in the adult as the round ligament (ligamentum teres) and the ligamentum venosum (see chapter 29).

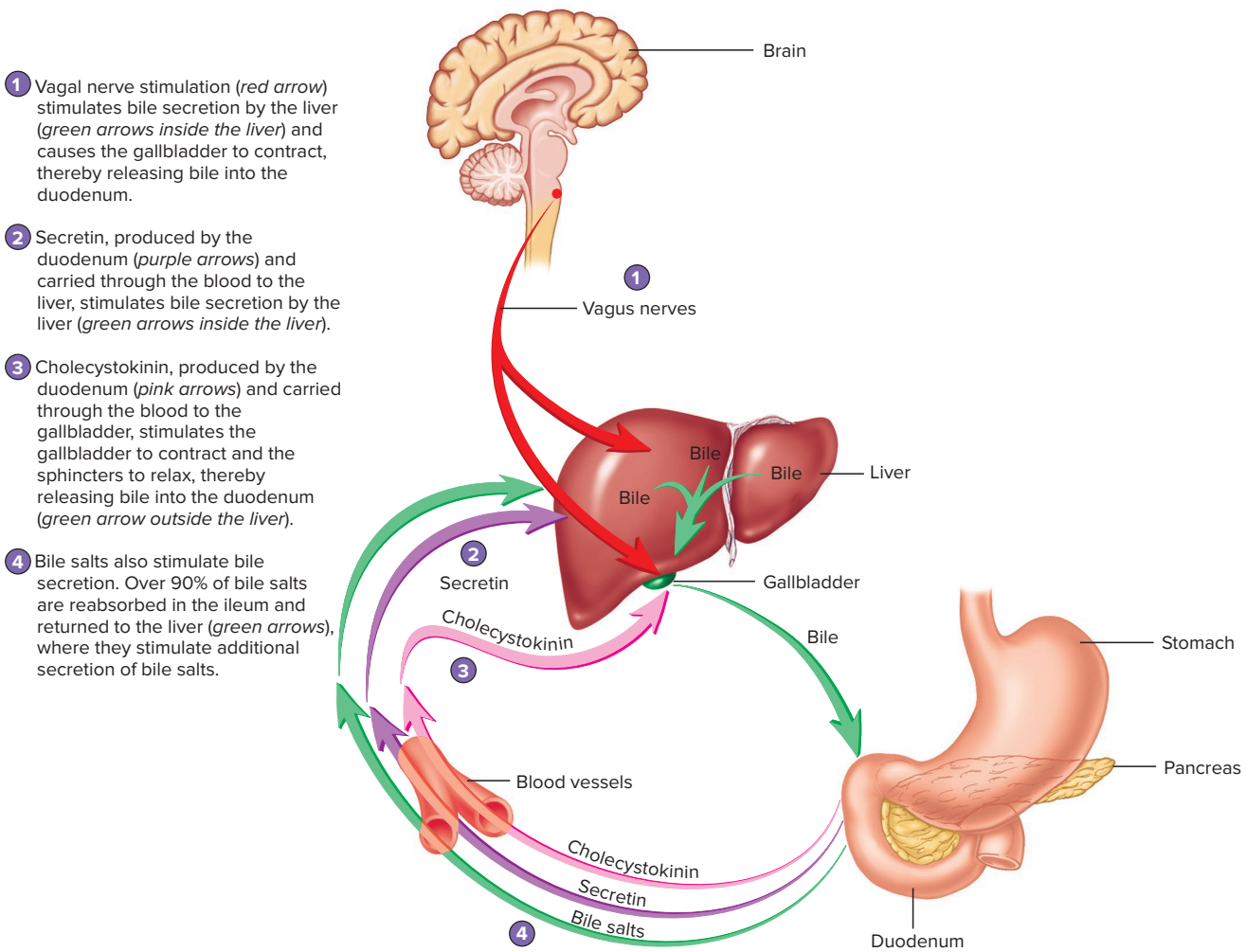
Functions of the Liver

The hepatocytes of the liver perform five important functions: (1) bile production for digestion and excretion, (2) storage of nutrients, (3) processing of nutrients, (4) detoxification, and (5) synthesis of new molecules.

Bile Production for Digestion and Excretion

The liver produces and secretes about 600–1000 mL of bile each day (see table 24.2). Bile is a complex solution that contains (1) bile salts, (2) bile pigments, (3) cholesterol, (4) lipids, (5) lipid-soluble hormones, and (6) lecithin (a mixture of phospholipids). Bile contains no digestive enzymes, but it plays a role in digestion because it neutralizes stomach acid and emulsifies lipids. The relative alkaline pH of bile helps neutralize the acidic chyme entering the duodenum. This is important because the pancreatic enzymes cannot function at the acidic pH of the chyme leaving the stomach. **Bile salts** emulsify lipids, which is necessary for subsequent digestion by lipase. Bile pigments are not required for any digestive function but rather are excretory products. Bile pigments have greenish-yellow to brown colors and give feces their characteristic color. One common bile pigment is bilirubin, which results from the breakdown of hemoglobin (see chapter 19).

Neural and hormonal signals stimulate the secretion and release of bile (figure 24.21). Parasympathetic stimulation through the vagus nerve increases bile secretion from the liver. Two hormones released from the duodenum increase bile in the digestive tract: (1) secretin and (2) cholecystokinin. Secretin stimulates bile secretion from the liver, primarily by increasing the water and bicarbonate ion content of bile. Cholecystokinin stimulates gallbladder contractions to release bile into the duodenum.



PROCESS FIGURE 24.21 Control of Bile Secretion and Release

Parasympathetic nerve impulses, cholecystokinin, and secretin all stimulate bile secretion and gallbladder contractions.

? *What effect, if any, would cholecystokinin have on bile secretion in an individual whose gallbladder has been removed?*

Bile salts increase bile secretion through a positive-feedback system (figure 24.21). Over 90% of bile salts are reabsorbed in the ileum and carried in the blood by the hepatic portal circulation. Upon their return to the liver, the bile salts stimulate further bile secretion and are once again secreted into the bile. This recycling process reduces the loss of bile salts in the feces. Bile secretion into the duodenum continues until the duodenum empties.

Storage of Nutrients

Hepatocytes can remove sugar from the blood and store it in the form of **glycogen.** They can also store lipids, vitamins (A, B_{12}, D, E, and K), copper, and iron. This storage function is usually short-term, and the amount of stored material in the hepatocytes—hence their size—fluctuates during the day.

Hepatocytes help maintain blood glucose levels within very narrow limits. If a large amount of sugar enters the general circulation after a meal, the blood osmolality will increase, resulting in hyperglycemia. Under normal conditions, this is prevented because the blood from the small intestine passes through the hepatic portal vein to the liver, where hepatocytes remove glucose and other substances from the blood, store them, and then secrete them back into the circulation when needed.

Processing of Nutrients

The processing, or interconversion, of nutrients is another important function of the liver. Ingested nutrients are not always present in the proportion needed by the tissues. In this case, the liver can convert some nutrients into others. For example, if a person is on a diet that is excessively high in protein, an oversupply of amino acids and an undersupply of lipids and carbohydrates may be delivered to the liver. The hepatocytes break down the amino acids and cycle many of them through metabolic pathways, so that they can be used to produce adenosine triphosphate, lipids, and glucose (see chapter 25).

Hepatocytes also transform substances that cannot be used by most cells into more readily usable substances. For example, they combine ingested dietary fats with choline and phosphorus in the liver to produce phospholipids, which are essential components of plasma membranes. In addition, vitamin D is hydroxylated in the liver hepatocytes. The hydroxylated form of vitamin D, which is the major circulating form of vitamin D, is transported through the blood to the kidneys, where it is again hydroxylated. The double-hydroxylated vitamin D is the active form of the vitamin, which functions in calcium maintenance.

Detoxification

Many ingested substances are harmful to body cells. In addition, the body itself produces many by-products of metabolism that, if accumulated, are toxic. The liver forms a major line of defense by altering the structure of many of these harmful substances to make them less toxic or to make their elimination easier. Ammonia, for example, a by-product of amino acid metabolism, is toxic and not readily removed from the blood by the kidneys. Hepatocytes remove ammonia from the blood and convert it to urea, which is less toxic than ammonia. Urea is then secreted into the blood and eliminated by the kidneys in the urine. The liver hepatocytes also remove other substances from the blood and excrete them into the bile.

Hepatic phagocytic cells (Kupffer cells), which lie along the sinusoid walls of the liver, phagocytize "worn-out" and dying red and white blood cells, some bacteria, and other debris that enters the liver through the blood vessels.

Synthesis of New Molecules

The liver can produce its own new compounds, including plasma proteins such as albumins, fibrinogen, globulins, heparin, and clotting factors, which are released into the blood (see chapter 19). In addition, the liver is the major site of cholesterol synthesis. Cholesterol is used throughout the body.

ASSESS YOUR PROGRESS

43. *Describe the lobes of the liver. What is the porta?*
44. *Diagram the duct system from the liver, gallbladder, and pancreas that empties into the major duodenal papilla.*
45. *Describe the flow of blood to and through the liver. Describe the flow of bile away from the liver.*
46. *Explain and give examples of the major functions of the liver.*
47. *What stimulates bile secretion from the liver?*

24.11 Gallbladder

LEARNING OUTCOMES

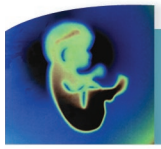
After reading this section, you should be able to

A. **Discuss the structure and function of the gallbladder.**
B. **State what stimulates the release of bile.**

The **gallbladder** is a saclike structure for bile storage. It is on the inferior surface of the liver; it is about 8 cm long and 4 cm wide (see figure 24.18). Three tunics form the gallbladder wall: (1) an inner mucosa folded into rugae which allow the gallbladder to expand; (2) a muscularis, which is a layer of smooth muscle that allows the gallbladder to contract; and (3) an outer covering of serosa. The cystic duct connects the gallbladder to the common bile duct.

The function of the gallbladder is to store and concentrate bile. The liver continually secretes bile, which flows to the gallbladder, where 40–70 mL of bile is stored. While the bile is in the gallbladder, water and electrolytes are absorbed. Thus, bile salts and pigments become as much as 5–10 times more concentrated than when secreted by the liver. Bile is released from the gallbladder by contractions stimulated by cholecystokinin and, to a lesser degree, by vagal stimulation. In this way, large amounts of concentrated bile are dumped into the small intestine shortly after a meal (figure 24.21).

Gallstones are insoluble aggregates formed in the gallbladder. They are often caused by precipitation of excess cholesterol, which can be the result of a high-cholesterol diet or other causes. Cholesterol is not soluble in water and is ordinarily kept in solution by bile salts. Occasionally, a gallstone passes out of the gallbladder and enters the cystic duct, blocking the release of bile. This condition interferes with normal digestion, and often the gallstone must be surgically removed. If the gallstone moves far enough down the duct, it can also block the pancreatic duct, resulting in pancreatitis.



Clinical IMPACT 24.4

Cystic Fibrosis

Cystic fibrosis is a hereditary disorder that occurs in 1 of every 2000 births and affects 33,000 people in the United States; it is the most common lethal genetic disorder among Caucasians. The most critical effects of the disease, accounting for 90% of the deaths, are on the respiratory system. Several other problems occur, however, in affected people. Because the disease is a disorder in a Cl^- transport channel protein—which affects chloride transport and, as a result, the movement of water—all exocrine glands are affected. The buildup of thick mucus in the pancreatic and hepatic ducts causes blockage of the ducts, so that bile salts and pancreatic digestive enzymes are prevented from reaching the duodenum. As a result, digestion is reduced, and fat-soluble vitamins are poorly absorbed due to the lack of bile to form micelles. The person suffers from vitamin A, D, E, and K deficiencies, which result in conditions such as night blindness, skin disorders, rickets, and excessive bleeding. Therapy includes administering the missing vitamins to the person and reducing dietary fat intake.

ASSESS YOUR PROGRESS

48. *Describe the three tunics of the gallbladder wall.*

49. *What is the function of the gallbladder? What stimulates the release of bile from the gallbladder?*

24.12 Pancreas

LEARNING OUTCOMES

After reading this section, you should be able to

A. **Describe the anatomy, histology, and ducts of the pancreas.**

B. **List the secretions of the pancreas, their functions, and their regulation.**

Anatomy of the Pancreas

The **pancreas** is a complex organ composed of both endocrine and exocrine tissues that perform several functions. The pancreas is located behind the stomach. The head of the pancreas is nestled within the curvature of the duodenum (figure 24.22*a*). The body and tail extend to the spleen.

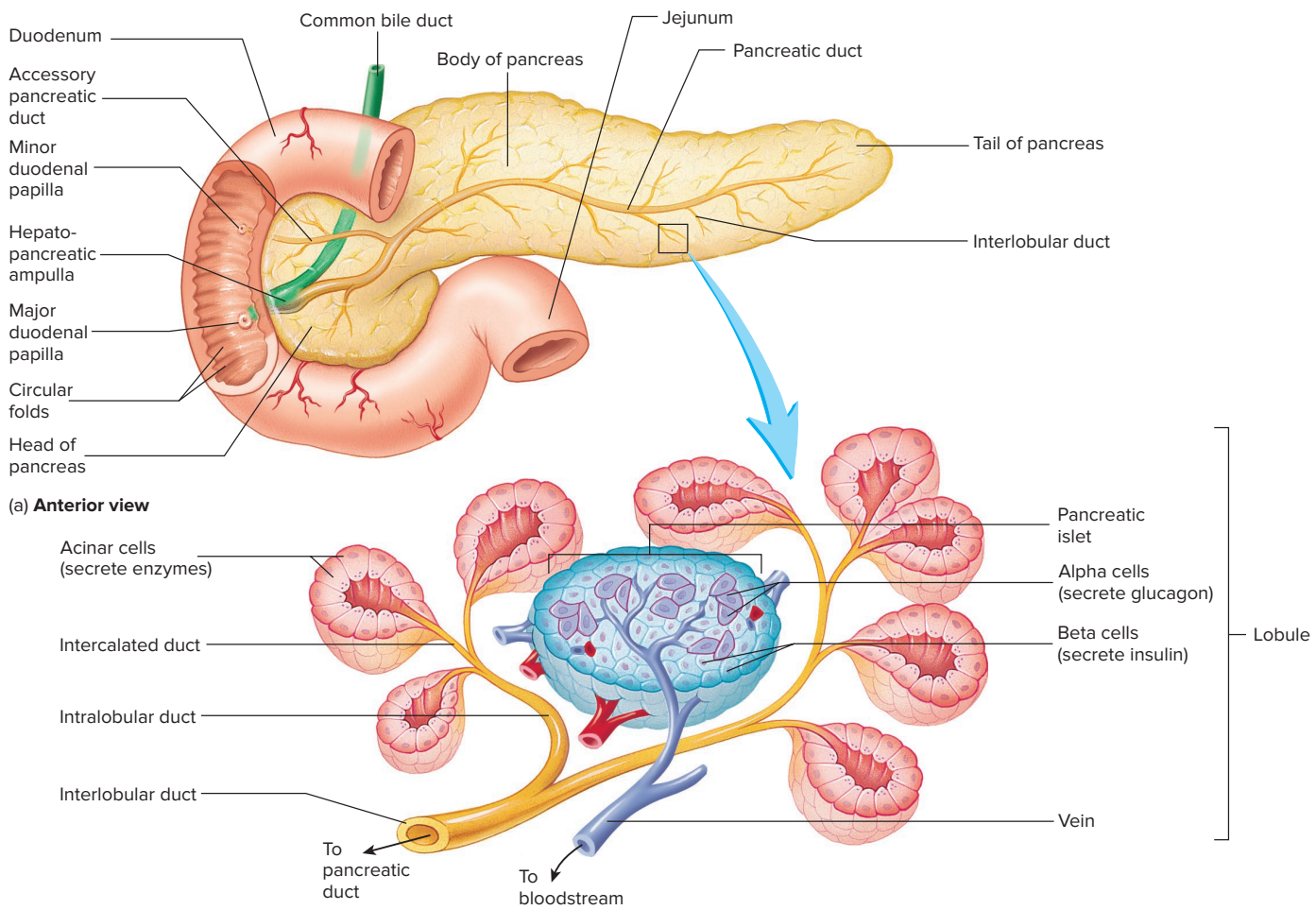


FIGURE 24.22 Anatomy and Histology of the Duodenum and Pancreas

(*a*) The head of the pancreas lies within the duodenal curvature, with the pancreatic duct emptying into the duodenum. (*b*) Histology of the pancreas, showing both the acinar cells and the pancreatic duct system. AP|R

The endocrine part of the pancreas consists of **pancreatic islets,** or *islets of Langerhans* (figure 24.22*b*). The islet cells produce the hormones insulin and glucagon, which are very important in controlling the blood levels of nutrients, such as glucose and amino acids, and somatostatin, which regulates insulin and glucagon secretion and may inhibit growth hormone secretion (see chapter 18).

The exocrine part of the pancreas is a compound acinar gland (see chapter 4). Acinar cells within the **acini** (as′i-nī; figure 24.22*b*) produce digestive enzymes. Clusters of acini form lobules that are separated by thin septa.

Exocrine secretions flow from the pancreas to the small intestine via a series of ducts (figure 24.22*b*). Secretions from the acini first flow into small **intercalated ducts,** then into **intralobular ducts,** which leave the lobules to join **interlobular ducts** between the lobules. The interlobular ducts attach to the main **pancreatic duct,** which joins the common bile duct at the hepatopancreatic ampulla, or *Vater's ampulla* (figure 24.22*a*; see figure 24.18). The hepatopancreatic ampulla empties into the duodenum at the major duodenal papilla. A smooth muscle sphincter, the **hepatopancreatic ampullar sphincter,** or *sphincter of Oddi,* regulates the opening of the ampulla. In most people, an accessory pancreatic duct opens at the minor duodenal papilla. The ducts are lined with simple cuboidal epithelium, and the epithelial cells of the acini are pyramid-shaped. A smooth muscle sphincter surrounds the pancreatic duct where it enters the hepatopancreatic ampulla.

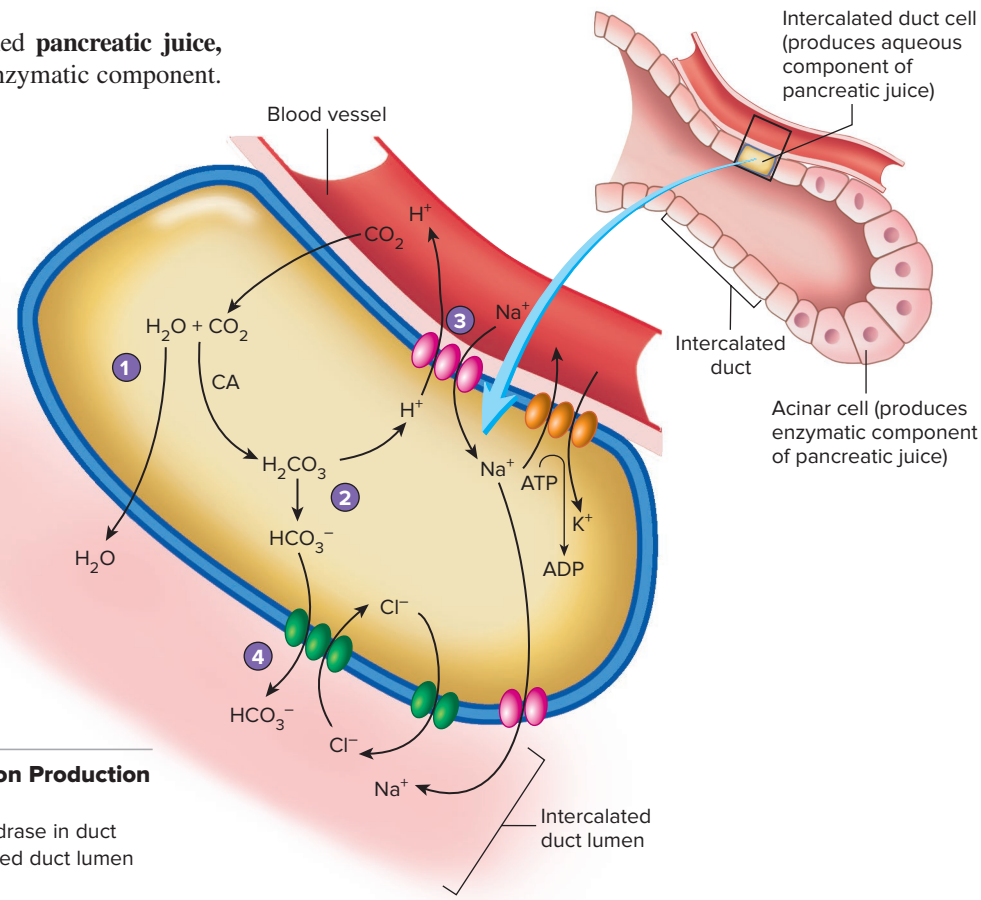
Pancreatic Secretions

The exocrine secretions of the pancreas, called **pancreatic juice,** have (1) an aqueous component and (2) an enzymatic component. Pancreatic juice is delivered to the small intestine through the pancreatic ducts, where it functions in digestion.

The **aqueous pancreatic juice** is bicarbonate-rich. Bicarbonate ions (HCO_3^-) are a major part of the aqueous pancreatic juice. They are actively secreted by columnar epithelial cells that line the smaller ducts of the pancreas. The aqueous juice contains Na^+ and K^+ in about the same concentration found in extracellular fluid, and water follows passively to make the pancreatic juice isotonic. The HCO_3^- ions of the aqueous pancreatic juice neutralize the acidic chyme that enters the small intestine from the stomach. The increased pH caused by pancreatic secretions in the duodenum stops pepsin digestion. Importantly, the neutralized pH is required for the functions of pancreatic and brush-border enzymes. The neutralized pH also prevents damage to the duodenum by the acid from the stomach.

The cellular mechanism responsible for the secretion of HCO_3^- is diagrammed in figure 24.23. The enzyme carbonic anhydrase in duct epithelial cells forms carbonic acid, which dissociates into H^+ and HCO_3^-. The H^+ are exchanged for Na^+, which are removed from the cell by the **Na^+–K^+ pump.** The Na^+–K^+ pump is an ATPase that moves Na^+ out and K^+ into the cell. It generates a Na^+ gradient that is important in many cellular processes, including intestinal nutrient and water absorption described later in this chapter. The HCO_3^- are then transported into the intercalated duct lumen in exchange for Cl^-. The result is

1. Water (H_2O) and carbon dioxide (CO_2) combine under the influence of carbonic anhydrase (CA) to form carbonic acid (H_2CO_3).
2. Carbonic acid dissociates to form hydrogen ions (H^+) and bicarbonate ions (HCO_3^-).
3. The H^+ is exchanged for sodium ions (Na^+) by an antiporter. Sodium ions are removed by the Na^+–K^+ pump.
4. The HCO_3^- is transported into the intercalated ducts in exchange for Cl^-, which returns to the lumen by a channel. Sodium ions and H_2O follow the HCO_3^- into the ducts.



PROCESS FIGURE 24.23 Bicarbonate Ion Production in the Pancreas

Bicarbonate (HCO_3^-) is produced by carbonic anhydrase in duct epithelial cells. It is then secreted into the intercalated duct lumen in exchange for Cl^-.

? *How is the production of HCO_3^- in the pancreas similar to production of H^+ in the gastric gland?*

a bicarbonate-rich aqueous pancreatic juice. The production of HCO_3^- in the pancreas is similar to the production of H^+ in the gastric gland (see figure 24.12). Both processes require carbonic anhydrase and the exchange of HCO_3^- and Cl^-. The major difference is that an alkaline solution is produced in the pancreas, whereas an acidic solution is produced in the stomach.

The **enzyme-rich pancreatic juice** contains enzymes that digest all major classes of food. This enzyme-rich secretion is produced by the acinar cells of the pancreas. Without the enzymes produced by the pancreas, lipids, proteins, and carbohydrates cannot be adequately digested (see tables 24.1 and 24.2).

The three major proteolytic enzymes are (1) **trypsin,** (2) **chymotrypsin,** and (3) **carboxypeptidase.** These enzymes, which digest proteins, are secreted in inactive forms, whereas many of the other enzymes are secreted in active form. The inactive forms are called trypsinogen, chymotrypsinogen, and procarboxypeptidase. They are activated by proteolytic removal of certain peptides from the precursor proteins. The proteolytic enzyme **enterokinase** (en′tĕr-ō-kī′nās), which is attached to the brush border of the small intestine, activates trypsinogen. Trypsin then activates more trypsinogen, as well as chymotrypsinogen and procarboxypeptidase. This process of releasing inactive enzymes is necessary because if the enzymes were produced in their active forms, they would start to digest the pancreas itself. Inappropriate activation causes **pancreatitis,** which is a painful inflammation of the pancreas. Pancreatitis can result from alcoholism, the use of certain drugs, pancreatic duct blockage, cystic fibrosis, viral infection, or pancreatic cancer. Symptoms can range from mild abdominal pain to systemic shock and coma.

Enzyme-rich pancreatic juice also contains other enzymes that digest carbohydrate, lipids, and nucleic acids. The enzyme **pancreatic amylase** continues the polysaccharide digestion initiated in the oral cavity. The lipid-digesting enzyme **pancreatic lipase** breaks down lipids into monoglycerides and free fatty acids. In addition to lipase, the pancreas also secretes the enzyme cholesterol esterase, which digests cholesteryl esters (dietary form of cholesterol) into cholesterol and free fatty acid. Deoxyribonucleases and ribonucleases are enzymes that degrade DNA and RNA, respectively.

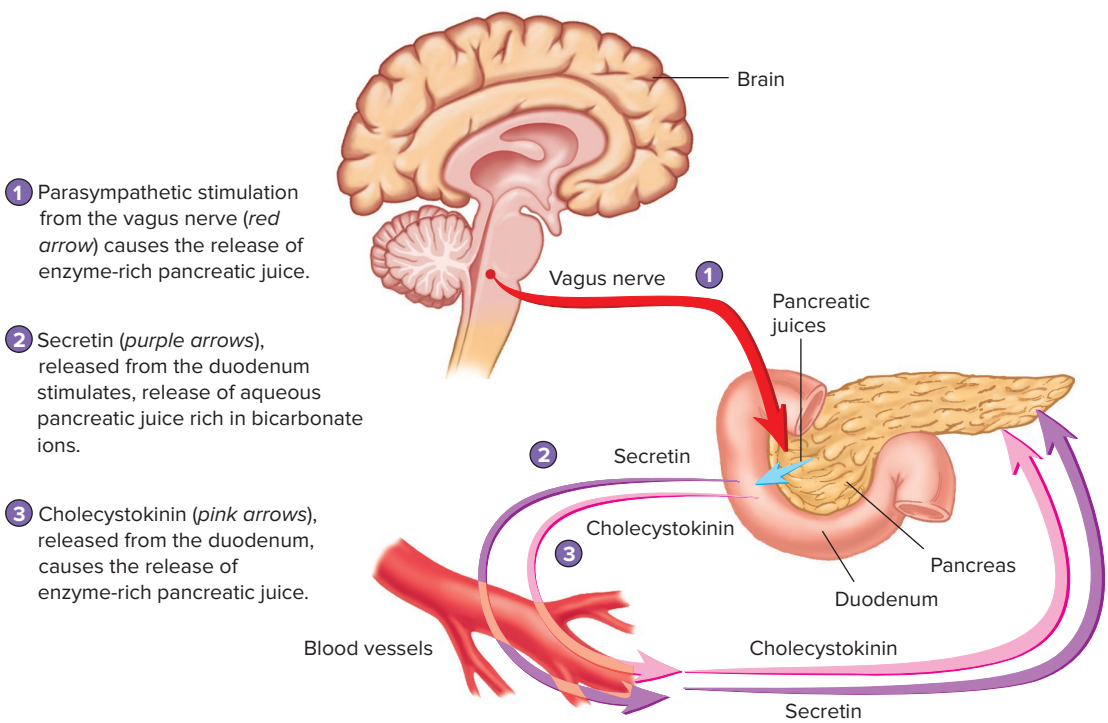
Regulation of Pancreatic Secretion

Both hormonal and neural mechanisms control secretion of pancreatic juice (figure 24.24). The hormones secretin and cholecystokinin are released from the duodenum in response to specific stimuli. Each hormone stimulates secretion of a specific type of pancreatic juice. Secretin stimulates secretion of the bicarbonate-rich aqueous juice. An acidic chyme in the duodenum stimulates the release of secretin.

Predict 5

Explain why secretin production in response to acidic chyme and its stimulation of bicarbonate ion secretion constitute a negative-feedback mechanism.

Cholecystokinin stimulates the secretion of the enzyme-rich pancreatic juice. Recall that cholecystokinin also stimulates the release of bile from the gallbladder, which aids in the digestion



PROCESS FIGURE 24.24 Control of Pancreatic Secretion

Cholecystokinin and parasympathetic impulses stimulate pancreatic enzyme secretion. Secretin stimulates secretion of bicarbonate from the pancreas.

? *Blockage of the pancreatic duct prevents pancreatic enzymes from being delivered to the small intestine. What effect would you expect this to have on cholecystokinin and secretin levels in the blood?*

of lipids. The major stimulus for the release of cholecystokinin is the presence of fatty acids and other lipids in the duodenum.

Parasympathetic stimulation through the vagus (X) nerves also stimulates the secretion of enzyme-rich pancreatic juices. Sympathetic impulses inhibit secretion. The effect of vagal stimulation on pancreatic juice secretion is greatest during the cephalic and gastric phases of stomach secretion.

ASSESS YOUR PROGRESS

50. *Describe the parts of the pancreas responsible for endocrine and exocrine secretions. Diagram the duct system of the pancreas.*

51. *Name the two kinds of exocrine secretions produced by the pancreas. What stimulates their production, and what is their function?*

52. *What enzymes are present in pancreatic juice? Explain the function of each.*

24.13 Large Intestine

LEARNING OUTCOMES

After reading this section, you should be able to

A. **List the parts of the large intestine and describe its anatomy and histology.**

B. **Describe the major functions of the large intestine and explain how defecation is regulated.**

The **large intestine** is the portion of the digestive tract extending from the ileocecal junction to the anus. It consists of four parts: (1) cecum, (2) colon, (3) rectum, and (4) anal canal. Normally, 18–24 hours are required for material to pass through the large intestine, in contrast to the 3–5 hours required for chyme to move through the small intestine. Thus, the movements of the colon are more sluggish than those of the small intestine. While in the colon, chyme is converted to feces. The formation of feces involves the absorption of water and salts, secretion of mucus, and extensive action of microorganisms. The colon stores the feces until they are eliminated by defecation. About 1500 mL of chyme enter the cecum each day, but more than 90% of the volume is reabsorbed, so that only 80–150 mL of feces are normally eliminated by defecation.

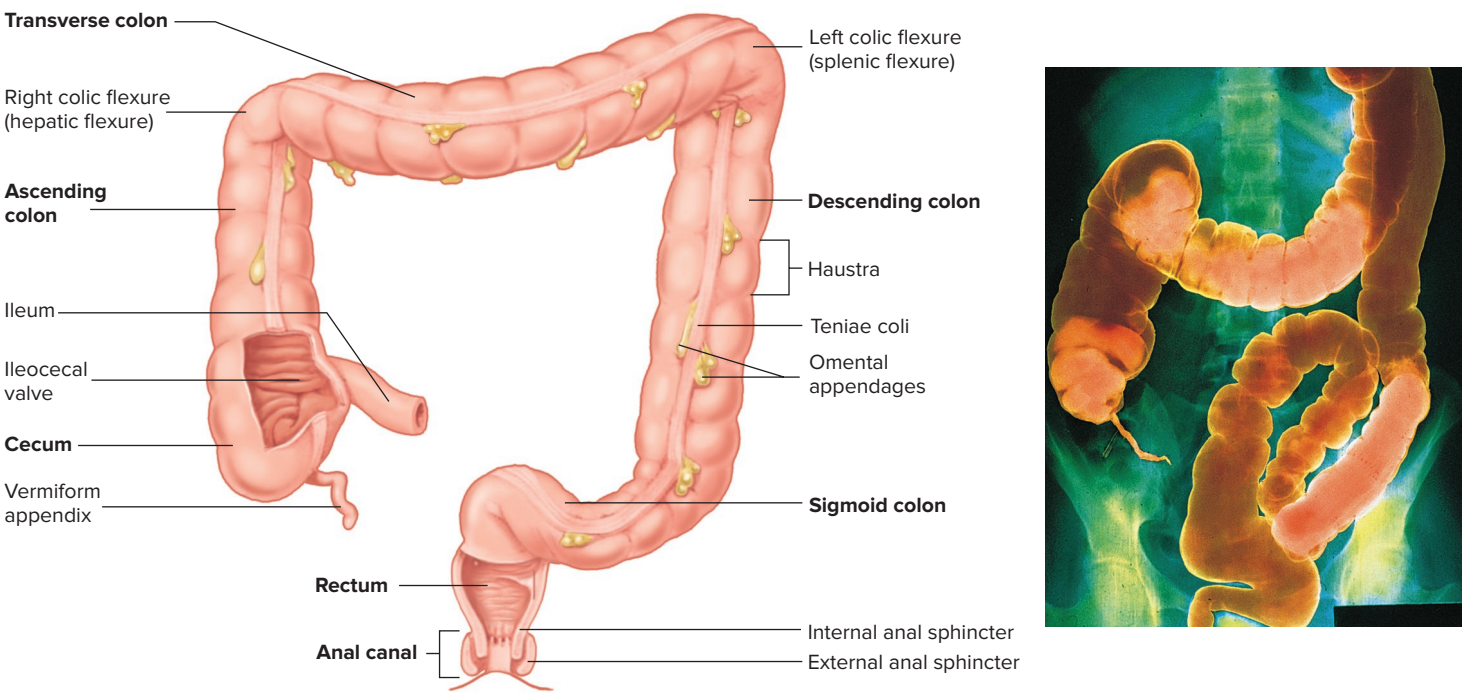
Anatomy of the Large Intestine

Cecum

The **cecum** (sē′kŭm) is the proximal end of the large intestine, where it meets the small intestine at the ileocecal junction. The cecum extends inferiorly about 6 cm past the ileocecal junction in the form of a blind sac (figure 24.25). The **vermiform** (ver′mi-fōrm; worm-shaped) **appendix** is a smaller, blind tube about 9 cm long attached to the cecum. The walls of the appendix contain many lymphatic nodules, which contribute to immune functions.

Colon

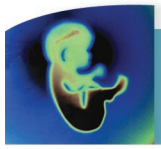
The **colon** (kō′lon), about 1.5–1.8 m long, consists of four parts: (1) ascending colon, (2) transverse colon, (3) descending colon,



(a) **Anterior view** (b) **Anterior view**

FIGURE 24.25 Large Intestine

(*a*) The large intestine consists of the cecum, colon, rectum, and anal canal. The teniae coli and omental appendages are along the length of the colon. (*b*) Radiograph of the large intestine following a barium enema. (b) ©CNRI/Science Source AP|R



Clinical IMPACT 24.5

Appendicitis

Appendicitis is an inflammation of the vermiform appendix that usually occurs because of an obstruction of the appendix. Secretions from the appendix cannot pass the obstruction and accumulate, resulting in enlargement and pain. Bacteria in the area can cause infection of the appendix. Symptoms include sudden abdominal pain, particularly in the right-lower portion of the abdomen; slight fever; loss of appetite; constipation or diarrhea; nausea; and vomiting. In the right-lower quadrant of the abdomen, about one-third the distance along a line from the right anterior superior iliac spine to the umbilicus, is an area called the McBurney point. This area of the body surface becomes very tender in patients with acute appendicitis because of pain referred from the inflamed appendix. Each year, 500,000 people in the United States experience appendicitis. The usual treatment is surgical removal of the appendix, called an appendectomy. If the appendix bursts, the infection can spread throughout the peritoneal cavity, causing peritonitis, with life-threatening results.

and (4) sigmoid colon (figure 24.25). The **ascending colon** extends superiorly from the cecum and ends at the right colic flexure (hepatic flexure) near the right inferior margin of the liver. The **transverse colon** extends from the right colic flexure to the left colic flexure (splenic flexure), and the **descending colon** extends from the left colic flexure to the superior opening of the true pelvis, where it becomes the sigmoid colon. The **sigmoid colon** forms an S-shaped tube that extends into the pelvis and ends at the rectum.

The muscularis of the colon differs from the small intestine. While the circular muscle layer of the colon is complete, the longitudinal muscle layer is incomplete. Rather than completely enveloping the intestinal wall, the longitudinal layer forms three bands, called the **teniae coli** (tē′nē-ē kō′lī). The teniae coli run the length of the colon (figure 24.26*a*; see figure 24.25). Contractions of the teniae coli cause pouches called **haustra** (haw′stră; to draw up) to form along the length of the colon, giving it a puckered appearance. Small, lipid-filled connective tissue pouches called **omental appendages** are attached to the outer surface of the colon along its length.

The mucosal lining of the large intestine consists of simple columnar epithelium. This epithelium is not formed into folds or villi like that of the small intestine but has numerous, straight, tubular glands called **crypts** (figure 24.26*b–d*). The crypts,

MICROBES In Your Body 24.1 — Fecal Transplants

Would you be shocked if your doctor said the one thing that could save your life is feces? Unfortunately, we are in the midst of a global, hospital-acquired diarrhea epidemic. The cause of this epidemic is a bacterium called *Clostridium difficile* (commonly referred to as *C. diff*), a pathogen that is normally found in the colon, but is controlled by the normal microbiota. One of the most critical functions of the normal gut microbiota is prevention of infections through competition with pathogens. As a consequence, when a patient takes antibiotics, *C. diff* can flourish and cause life-threatening diarrhea. Treatment of *C. diff* infections with specific antibiotics will often stop the diarrhea initially. However, *C. diff* are spore-forming bacteria. Spores are very stable structures that allow bacteria to withstand harsh conditions until favorable conditions return and the bacteria can regrow. Thus, antibiotics kill only the *C. diff* cells, not the spores. Hence, it is very common for patients to suffer multiple recurrences of diarrhea for months, which can lead to death in some patients. Additionally, a more virulent, resistant strain of *C. diff* has emerged. This strain is resistant to certain antibiotics and makes a greater number of spores and more of the toxins responsible for the diarrhea.

So, where do feces come into play? Because antibiotic treatments are not effective (65% infection recurrence), physicians are considering an old treatment: fecal transplants. The first documented case of transplanting feces from a healthy donor into a diseased recipient was in 1958. Fecal transplantation has since been used successfully in veterinary medicine for decades. However, due to the unappealing nature of this treatment, it has only recently been considered an option in humans. Now more commonly known as intestinal fecal transplantation (IFT), it has been shown to effectively treat diarrhea in over 90% of *C. diff.* infections. The idea is that a healthy donor—usually a close family household member such as a spouse or significant partner—donates their feces. The feces are mixed with physiological saline, filtered, and then introduced into the recipient's gastrointestinal (GI) tract by one of two ways: the upper GI tract route or the lower GI tract route. The upper GI tract route uses either a gastroscope or nasogastric tube to transfer the material to the recipient's intestine. Of the two, this one is easier and costs less. However, there is the possibility the donor microbiota may not reach the end of the colon or that the patient may vomit the fecal material. The lower GI tract route uses a colonoscope or enema and is sometimes the preferred approach, but does run the risk of perforating the colon. Thus, as yet, there is no standardized method for transferring the donor feces. But, research is showing that more and more patients may overcome their initial reluctance when presented with a predictable success rate and greater reliability than other protocols. In addition, the recent "RePOOPulating" study shows promise that doctors may soon be able to treat *C. diff.* infections simply by prescribing a pill that contains normal microbiota.

Predict 6

Predict the mechanism by which ingestion of a capsule filled with dried normal microbiota could treat a *C. diff* infection. Would the pill be able to be swallowed, or would it have to be administered directly into the intestine? Explain.

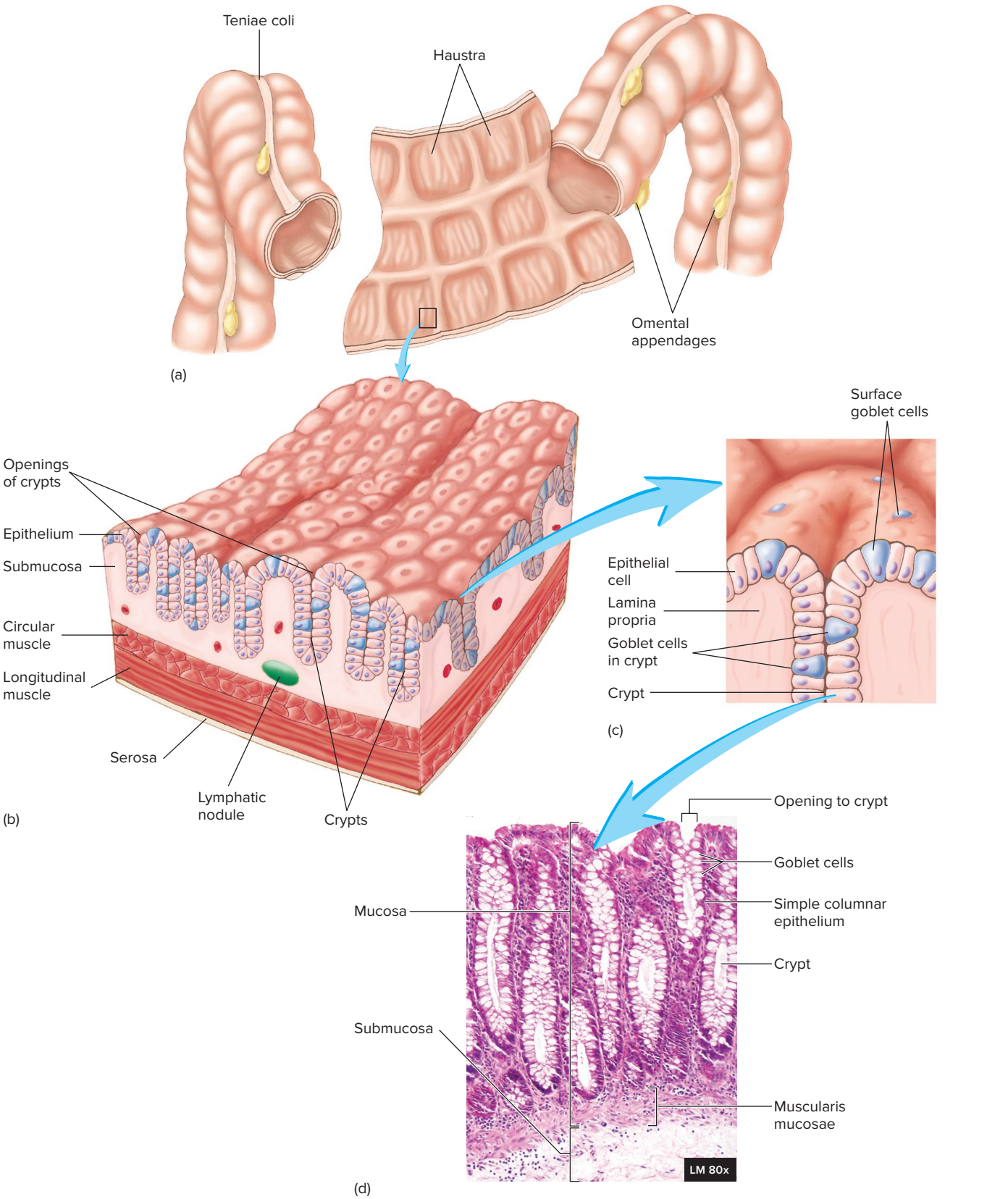


FIGURE 24.26 Histology of the Large Intestine

(*a*) Section of the transverse colon cut open to show the inner surface. (*b*) Enlargement of the inner surface, showing openings of the crypts. (c) Higher magnification of a single crypt. (*d*) Photomicrograph showing the histology of the large intestine wall. (d) ©McGraw-Hill Education/Al Telser, photographer

which are somewhat similar to the intestinal glands of the small intestine, are composed of three cell types: (1) absorptive, (2) goblet, and (3) granular. The major difference is that, in the large intestine, goblet cells predominate, and the other two cell types are greatly reduced in number.

Rectum

The **rectum** is a straight, muscular tube that begins at the distal end of the sigmoid colon and ends at the anal canal (see figure 24.25). The mucosal lining of the rectum is simple columnar epithelium, and the muscular tunic is relatively thick, compared with the rest of the digestive tract.

Anal Canal

The last 2–3 cm of the digestive tract is the **anal canal** (see figure 24.25). It begins at the inferior end of the rectum and ends at the **anus** (external digestive tract opening). The smooth muscle layer of the anal canal is even thicker than that of the rectum and forms the **internal anal sphincter** at its superior end. Skeletal muscle forms the **external anal sphincter** at the inferior end of the canal. The epithelium of the superior part of the anal canal is simple columnar, and that of the inferior part is stratified squamous. Rectal veins that supply the anal canal can become enlarged or inflamed, a condition known as **hemorrhoids.** Hemorrhoids cause pain, itching, and bleeding around the anus. They can usually be treated by changes in diet or medications.

Secretions of the Large Intestine

The major secretion product of the large intestine is mucus (see tables 24.1 and 24.2). Mucus is secreted from the numerous goblet cells scattered along in the length of the mucosa of the colon. In addition, there are numerous crypts lined almost entirely with goblet cells. Little enzymatic activity is associated with secretions of the colon. Mucus lubricates the wall of the colon and helps the fecal matter stick together. Tactile stimuli and irritation of the colon wall trigger local enteric reflexes that increase mucous secretion. Parasympathetic stimulation also increases the secretory rate of the goblet cells.

The feces that leave the digestive tract consist of water, solid substances (e.g., undigested food), microorganisms, and sloughed-off epithelial cells. An abnormally frequent discharge of watery feces is called **diarrhea** (see Systems Pathology 24.1).

Numerous microorganisms inhabit the colon. They reproduce rapidly and ultimately constitute about 30% of the dry weight of the feces. An important function of colonic bacteria is to synthesize vitamin K, which is passively absorbed in the colon. Acids are secreted by colonic bacteria as metabolic by-products. An antiporter exchanges HCO_3^- for Cl^- in epithelial cells of the colon in response to acid produced by colic bacteria. Another antiporter exchanges Na^+ for H^+. Movement of Na^+ into the epithelial cells via this exchanger and other Na^+ channels is driven by the Na^+ gradient established by the Na^+–K^+ pump. Water leaves the lumen of the colon through osmosis as Na^+ and Cl^- move into the epithelial cells. Colonic bacteria also break down a small amount of cellulose to glucose. However, the glucose cannot be absorbed in the large intestine. Bacterial actions in the colon produce gases called **flatus** (flā′tŭs; blowing). The amount of flatus depends partly on the bacterial population in the colon and partly on the type of food consumed. For example, beans, which contain certain complex carbohydrates, are well known for their flatus-producing effect.

Movement in the Large Intestine

Segmental mixing movements occur in the colon much less often than in the small intestine. Peristaltic waves are largely responsible for moving chyme along the ascending colon. At widely spaced intervals (normally three or four times each day), large parts of the transverse and descending colon undergo several strong contractions, called **mass movements.** Each mass movement contraction extends over a much longer part of the digestive tract (≥20 cm) than does a peristaltic contraction and propels the colon contents a considerable distance toward the anus (figure 24.27). Mass movements are very common after meals because they are initiated by the presence of food in the stomach or duodenum. Mass movements are most common about 15 minutes after breakfast. They usually persist for 10–30 minutes and then stop for perhaps half a day.

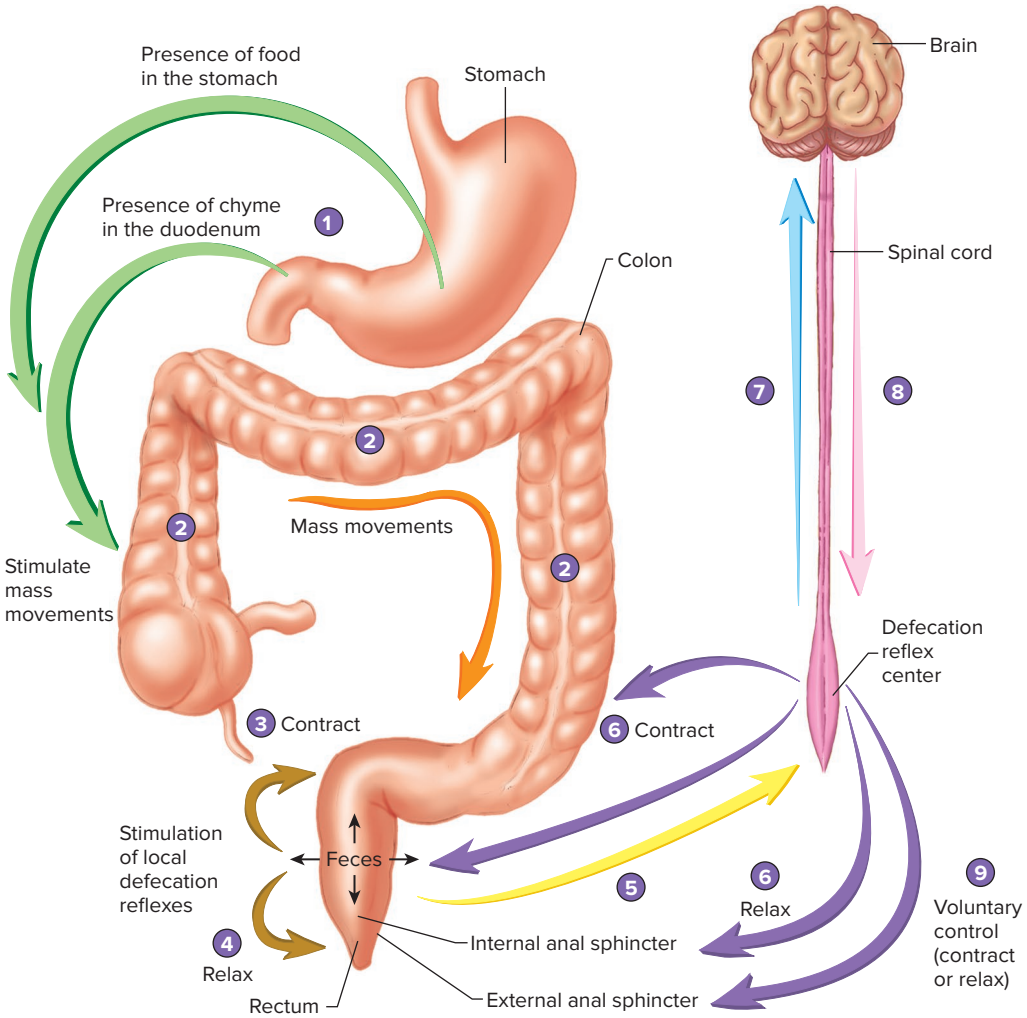
Mass movements are coordinated by two reflexes in the ENS: (1) gastrocolic and (2) duodenocolic. **Gastrocolic reflexes** are initiated by distension in the stomach, and **duodenocolic reflexes** are initiated by distension in the duodenum. The gastrocolic and duodenocolic reflexes promote peristalsis of the small and large intestines, including mass movements. These reflexes are mediated by parasympathetic reflexes, local reflexes, and hormones, such as cholecystokinin and gastrin. The thought or smell of food, distension of the stomach, and the movement of chyme into the duodenum can stimulate them.

During defecation, the contractions that move feces toward the anus must be coordinated with the relaxation of the internal and external anal sphincters. The movement of feces from the colon into the rectum distends the rectal wall, which stimulates the defecation reflex. The **defecation reflex** consists of local and parasympathetic reflexes (figure 24.27). Local reflexes cause weak contractions of the distal colon and rectum and relaxation of the internal anal sphincter. Parasympathetic reflexes are responsible for most of the defecation reflex. Action potentials produced in response to distension of the rectal wall travel along afferent nerve fibers to the defecation reflex center (S2–S4) in the conus medullaris of the spinal cord. Then efferent action potentials are initiated that return through nerves to the colon and rectum, reinforcing peristaltic contractions and relaxation of the internal anal sphincter.

The internal and external **anal sphincters** prevent defecation. Resting sphincter pressure results from tonic muscle contractions, mostly of the internal anal sphincter. In response to increased abdominal pressure, reflexes mediated through the spinal cord cause contractions of the external anal sphincter. Thus, the untimely expulsion of feces during coughing or exertion is avoided.

There is also conscious control of the defecation reflex. Action potentials from the sacral spinal cord ascend to the brain,

1. Distension of the stomach and chyme in the duodenum can stimulate the gastrocolic and duodenocolic reflexes (*green arrows*).
2. Mass movements occur in the colon, which propel the contents toward the rectum (*orange arrow*).
3. Distension of the rectum by feces stimulates local defecation reflexes that cause contractions of the colon and rectum (*brown arrows*), which move feces toward the anus.
4. Local reflexes cause relaxation of the internal anal sphincter (*brown arrows*).
5. Distension of the rectum by feces stimulates parasympathetic reflexes. Action potentials are propagated to the defecation reflex center located in the spinal cord (*yellow arrow*).
6. Action potentials stimulate contraction of the colon and rectum and relaxation of the internal anal sphincter (*purple arrows*).
7. Action potentials are propagated through ascending nerve tracts to the brain (*blue arrow*).
8. Descending nerve tracts from the brain regulate the defecation reflex center (*pink arrow*).
9. Action potentials from the brain control the external anal sphincter (*purple arrow*).



PROCESS FIGURE 24.27 Control of Defecation

Defecation involves both reflex and voluntary neural signals that are triggered by distension of the rectum.

What would be the consequence of a spinal cord injury above the defecation reflex center?

where parts of the brainstem and hypothalamus inhibit or facilitate reflex activity in the spinal cord. In addition, action potentials ascend to the cerebrum, where awareness of the need to defecate is realized. The external anal sphincter is composed of skeletal muscle and is under conscious cerebral control. If this sphincter is relaxed voluntarily, feces are expelled. On the other hand, increased contraction of the external anal sphincter prevents defecation. The defecation reflex persists for only a few minutes and quickly declines. Generally, the reflex is reinitiated after a period that may be as long as several hours. Mass movements in the colon are usually the reason for reinitiation of the defecation reflex.

Defecation can be initiated by voluntary actions that stimulate a defecation reflex. This "straining" includes a large inspiration of air, followed by closure of the larynx and forceful contraction of the abdominal muscles. As a consequence, the pressure in the abdominal cavity increases and forces feces into the rectum. Stretch of the rectum initiates a defecation reflex, and input from the brain overrides the reflexive contraction of the external anal sphincter stimulated by increased abdominal pressure. The increased abdominal pressure also helps push feces through the rectum.

ASSESS YOUR PROGRESS

53. *Describe the parts of the large intestine. What are teniae coli, haustra, and crypts?*

54. *Explain the difference in structure between the internal anal sphincter and the external anal sphincter.*

55. *Name the substances secreted and absorbed in the large intestine.*

56. *What is the role of microorganisms in the colon?*

57. *What kinds of movements occur in the colon? Describe the defecation reflex.*

24.14 Digestion and Absorption

LEARNING OUTCOMES

After reading this section, you should be able to

A. **Describe the chemical digestion and absorption of carbohydrates, lipids, and proteins.**

B. **Explain the transport of water and ions through the intestinal wall.**

Digestion is the breakdown of food to molecules small enough to be absorbed into the blood. **Mechanical digestion** breaks large food particles into smaller ones. **Chemical digestion** is the breaking of covalent chemical bonds in organic molecules by digestive enzymes. Carbohydrates break down into monosaccharides, lipids break down into fatty acids and monoglycerides, and proteins break down into amino acids. However, some molecules (e.g., vitamins, minerals, and water) are not broken down. A relatively small amount of digestion begins in the oral cavity and some occurs in the stomach, but the vast majority of digestion occurs in the proximal end of the small intestine, especially in the duodenum.

Absorption is the means by which molecules are moved out of the digestive tract into the blood for distribution throughout the body. Nearly all absorption of nutrients occurs in the duodenum and jejunum of the small intestine. Some absorption also occurs in the ileum. A few chemicals, such as nitroglycerin, can be absorbed through the thin mucosa of the oral cavity below the tongue. Some small molecules (e.g., alcohol and aspirin) can diffuse through the stomach epithelium into the blood.

Some molecules can be absorbed by diffusion, whereas others must be transported across the intestinal wall. Transport requires transport proteins, which work by facilitated diffusion, active-transport, or secondary-transport mechanisms, such as symport and antiport. The epithelial cells that form the intestinal wall have two distinct sides with different transport proteins on each side. The side that faces the digestive tract lumen is called the **apical membrane,** and the side that faces the blood vessels is called the **basolateral membrane.** The transport proteins in these membranes are responsible for the one-way movement of molecules from the digestive tract to the rest of the body.

Once the digestive products have been absorbed, they are transported to other parts of the body by two routes. Water, ions, and water-soluble digestion products, such as glucose and amino acids, enter the hepatic portal system and travel to the liver. The products of lipid metabolism are coated with proteins and transported into lymphatic capillaries called lacteals (see figure 24.16*c,d*). The lacteals are connected by lymphatic vessels to the thoracic duct (see chapter 22), which empties into the left subclavian vein. The protein-coated lipid products then travel in the blood to adipose tissue or to the liver.

Carbohydrates

Ingested **carbohydrates** consist primarily of polysaccharides, such as starches; disaccharides, such as sucrose (table sugar) and lactose (milk sugar); and monosaccharides, such as glucose and fructose (the sugar found in many fruits). During digestion, polysaccharides break down first into smaller chains and some disaccharides and monosaccharides. Disaccharides then break down into monosaccharides.

A minor amount of carbohydrate digestion begins in the oral cavity with the partial digestion of starches by **salivary amylase** (am′il-ās). Digestion continues in the stomach until the food is well mixed with acid, which inactivates salivary amylase. Carbohydrate digestion is resumed in the small intestine by **pancreatic amylase** (figure 24.28). Pancreatic lipase in the small intestine is responsible for the majority of carbohydrate digestion. However, many of the digested carbohydrates at this point are disaccharides, which can not be absorbed. The final step in carbohydrate digestion is performed by a series of **disaccharidases** that are bound to the microvilli of the intestinal epithelium. These enzymes digest disaccharides into three monosaccharides: (1) glucose, (2) galactose, and (3) fructose. The major monosaccharide is glucose.

The monosaccharides glucose and galactose are taken up into intestinal epithelial cells by symport, powered by a Na^+ gradient (figure 24.29). The Na^+ gradient is generated by the **Na^+–K^+ pump** located on the basolateral membrane. Diffusion of Na^+ down its concentration gradient provides the energy to transport glucose or galactose across the plasma membrane. In contrast to glucose and galactose, the monosaccharide fructose is taken up by

Case STUDY 24.1 Spinal Cord Injury and Defecation

Dan, a 17-year-old male, was driving home late at night after a ski trip when he missed a sharp curve and crashed. He suffered traumatic injury at the T11 level of the spinal cord, with complete paralysis of both lower limbs. As a result, Dan became incontinent and unable to control his bowel movements.

Approximately 10,000 new spinal cord injuries occur per year in the United States. About 80% of those injuries involve men, usually in their late teens or twenties. The most common cause is motor vehicle accidents, followed by violence, falls, and sports.

Loss of the ability to control defecation affects the quality of life of most spinal cord injury patients, at least temporarily. The spinal cord is required for a normal defecation reflex and for voluntary control of the external anal sphincter (figure 24.27). In terms of their effect on defecation, spinal cord injuries can be divided into two groups: injuries that occur above the conus medullaris and those that damage the conus medullaris where the defecation reflex center is located. Immediately following a spinal cord injury, loss of reflexes below the level of the injury, called **spinal shock,** occurs. However, the reflexes usually become functional again, and the defecation reflex may be depressed for a few weeks but eventually returns.

Predict 7

Explain how an enema involving the injection of fluid into the rectum can stimulate defecation.

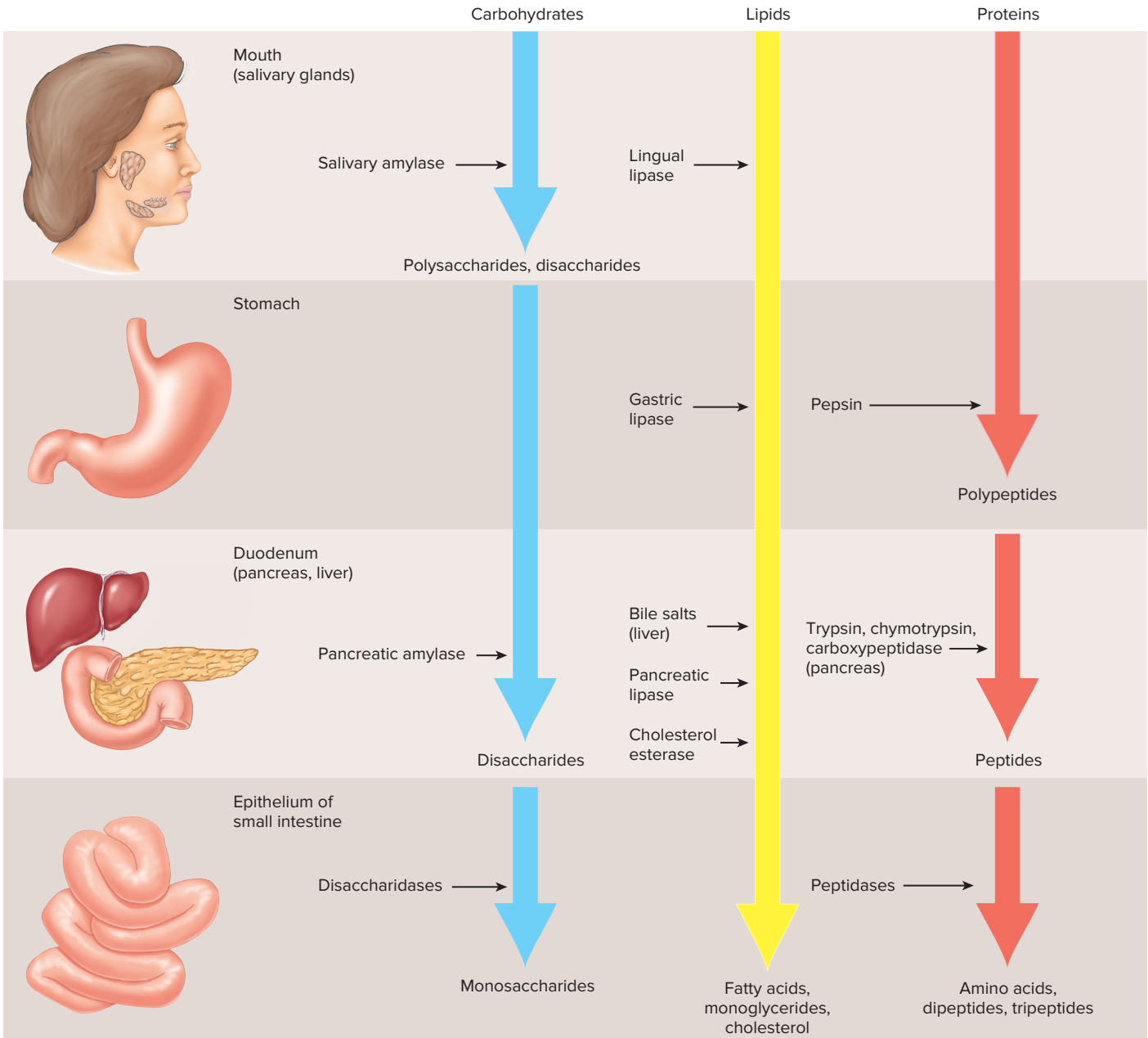


FIGURE 24.28 Digestion of the Three Major Food Types

The enzymes involved in digesting carbohydrates, lipids, and proteins are depicted in relation to the region of the digestive tract where each functions. AP|R

facilitated diffusion. Once inside the intestinal epithelial cell, monosaccharides are transported by facilitated diffusion to the capillaries of the intestinal villi and carried by the hepatic portal system to the liver, where the nonglucose monosaccharides are converted to glucose. Glucose enters the cells through facilitated diffusion. The rate of glucose transport into most types of cells is greatly influenced by **insulin** and may increase 10-fold in its presence (see chapter 18).

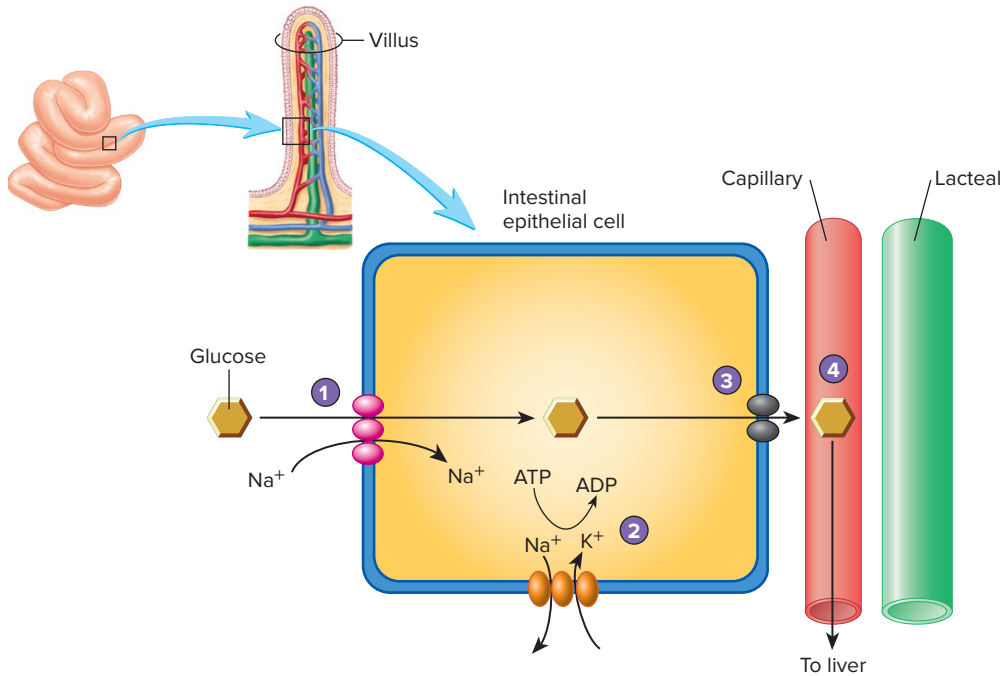
Lipids

Lipids are molecules that are insoluble or only slightly soluble in water. They include triglycerides, phospholipids, cholesterol, steroids, and fat-soluble vitamins. **Triglycerides** (trī-glis′er-īdz) are the most common type of lipid and are often referred to as fats. They consist of three fatty acids bound to glycerol.

Lipase (lip′ās) enzymes digest lipid molecules (see figure 24.28). The primary products of lipase digestion are free fatty acids and monoglycerides. There are three lipases released into the digestive tract: (1) pancreatic lipase, (2) lingual lipase, and (3) gastric lipase. The vast majority of lipase is **pancreatic lipase,** which is secreted by the pancreas and digests lipids in the small intestine. A minor amount of **lingual lipase** is secreted in the oral cavity and swallowed with food. It digests a small amount (<10%) of lipid in the stomach. The stomach also produces very small amounts of **gastric lipase.** Lingual and gastric lipase are most

Monosaccharide (glucose) transport

1. Glucose is absorbed by symport with Na^+ into intestinal epithelial cells.
2. Symport is driven by a sodium gradient established by a Na^+–K^+ pump.
3. Glucose moves out of the intestinal epithelial cells by facilitated diffusion.
4. Glucose enters the capillaries of the intestinal villi and is carried through the hepatic portal vein to the liver.



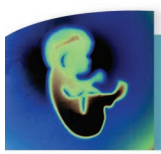
PROCESS FIGURE 24.29 Transport of Glucose Across the Intestinal Epithelium

Glucose absorption occurs by symport powered by a Na^+ gradient.

? *If a drug is given that specifically inhibits the Na^+–K^+ pump in intestinal epithelial cells, how will glucose absorption be affected?*

important in neonatal infants, while pancreatic lipase is the major enzyme in adults.

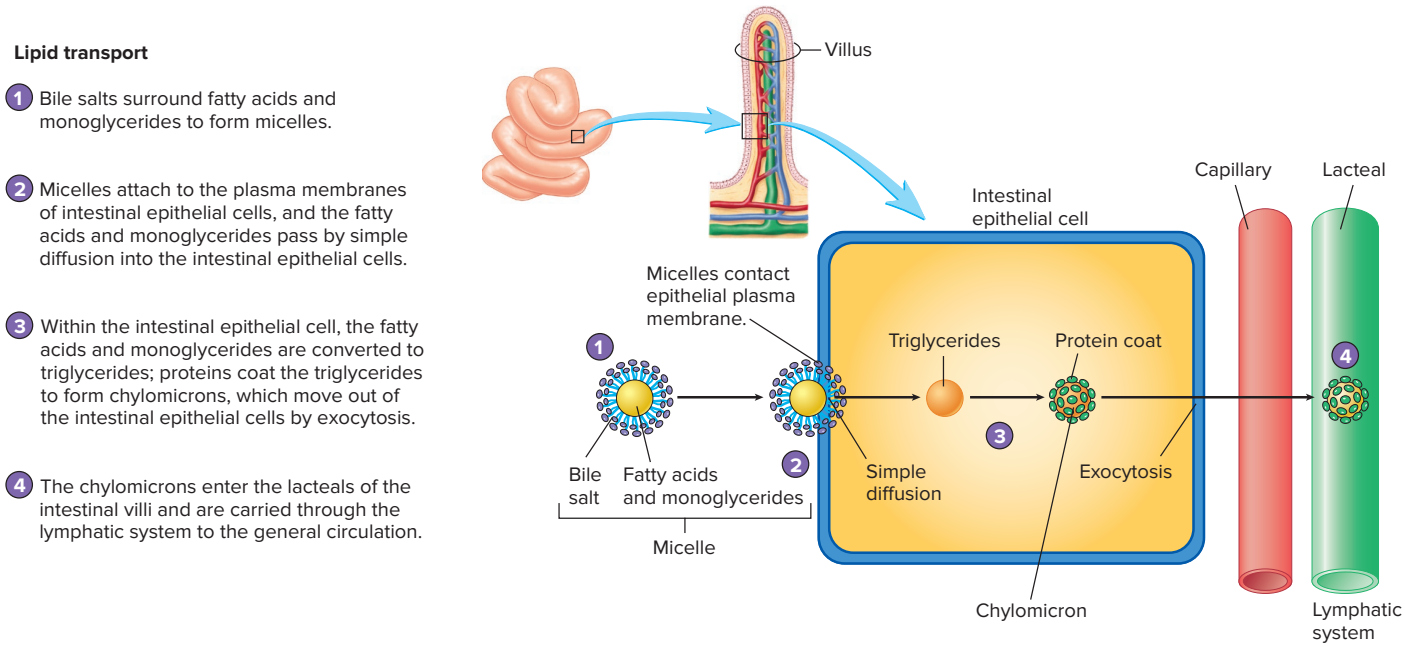
Pancreatic lipase alone cannot efficiently digest lipids. A key step in lipid digestion is **emulsification** (ē-mŭl′si-fi-kā′shŭn), by which bile salts transform large lipid droplets into much smaller droplets. Bile salts mix with lipids and act as detergents to disrupt lipid droplets. By decreasing the droplet size, emulsification increases the surface area of the lipid exposed to lipase and other digestive enzymes. This is necessary because lipase is water-soluble and can digest lipids only at the surface of the droplets. The bile salts are secreted by the liver and stored in the gallbladder until needed in the duodenum. Lingual and gastric lipase, which work in the acidic environment of the stomach, do not require bile salts.



Clinical IMPACT 24.6 Lactose Intolerance

Lactose intolerance is the inability to digest the lactose in milk and other dairy products. The majority of adults in most regions of the world are lactose intolerant, although infants are not. Why can infants digest milk, whereas their parents cannot? The reason is that many adults lack the enzyme lactase. Lactase, present on the surface of absorptive cells in the intestinal mucosa, digests the disaccharide lactose down to two monosaccharides. Lactase is made at birth but is no longer synthesized after about age 6 in 5–15% of Europeans and 80–90% of Africans and Asians. Therefore, these people can no longer digest lactose. The major exceptions are people of northern European ancestry and some pastoral nomadic tribes in Africa and the Middle East. In these populations, a mutation in the promoter (see chapter 3) of the lactase gene permits the continued expression of lactase into adulthood. Normally, lactase production stops because the promoter is "turned off" as the infant ages, but the mutation allows the promoter to ignore this developmental switch.

Researchers believe that the dietary reliance on milk and milk products in some societies provided a selective advantage for lactase persistence. In the United States, most people are lactose tolerant, but intolerance is still one of the most common digestive tract disorders seen by primary care physicians. The main symptom of lactose intolerance is diarrhea due to fluid loss as water follows lactose through the digestive tract. In addition, a considerable amount of gas is generated from lactose metabolism by bacteria in the large intestine. Even though these colonic bacteria metabolize lactose to monosaccharides, it is too late for the monosaccharides to be absorbed. Gene therapy has proven successful in animal models of lactose intolerance, although at present the best treatment is simply to avoid foods containing lactose.



PROCESS FIGURE 24.30 Transport of Lipids Across the Intestinal Epithelium

Lipid absorption occurs when micelles enter intestinal epithelial cells and exit by exocytosis.

? *Why must digested lipids be moved out of the intestinal epithelial cells into lacteals of the lymphatic system by exocytosis?*

Once lipids are digested in the intestine, bile salts aggregate around the small droplets. Lipid droplets surrounded by bile salts are called **micelles** (mī-selz′; small morsels; figure 24.30). The hydrophobic ends of the bile salts are directed toward the free fatty acids, cholesterol, and monoglycerides at the center of the micelle; the hydrophilic ends are directed outward toward the water environment. When a micelle comes in contact with the epithelial cells of the small intestine, the lipid contents of the micelle pass by simple diffusion through the plasma membrane of the epithelial cells. The bile salts are not absorbed until they reach the epithelium of the distal ileum.

Lipid Transport

Within the smooth endoplasmic reticulum of the intestinal epithelial cells, free fatty acids are combined with monoglyceride molecules to form triglycerides. Proteins synthesized in the epithelial cells attach to droplets of triglycerides, phospholipids, and cholesterol called **chylomicrons** (kī-lō-mi′kronz). Chylomicrons contain about 90% triglyceride, 5% cholesterol, 4% phospholipid, and 1% protein (figure 24.31).

The chylomicrons leave the epithelial cells and enter the lacteals of the lymphatic system within the villi. Chylomicrons

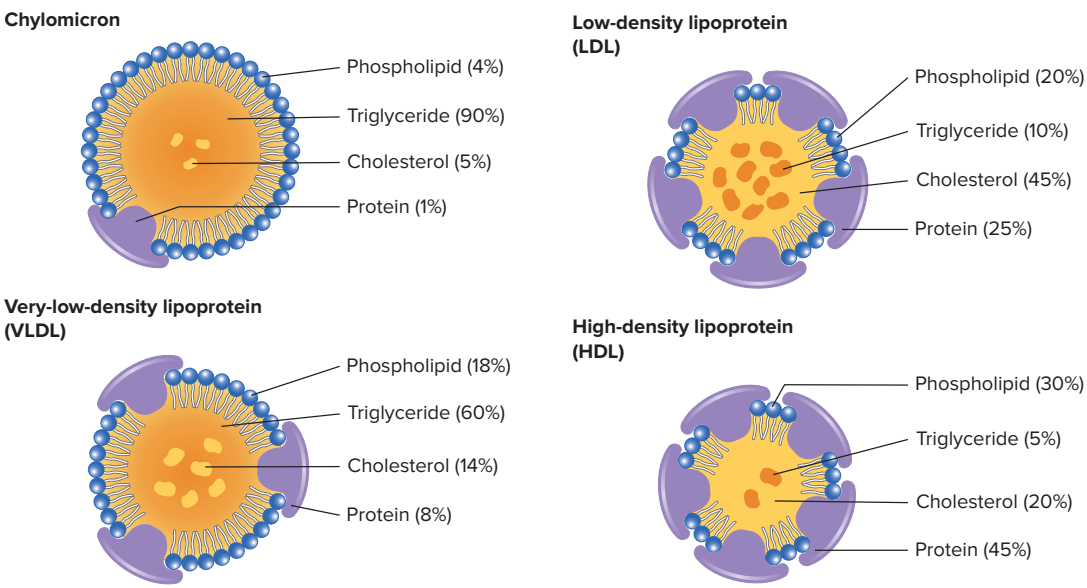
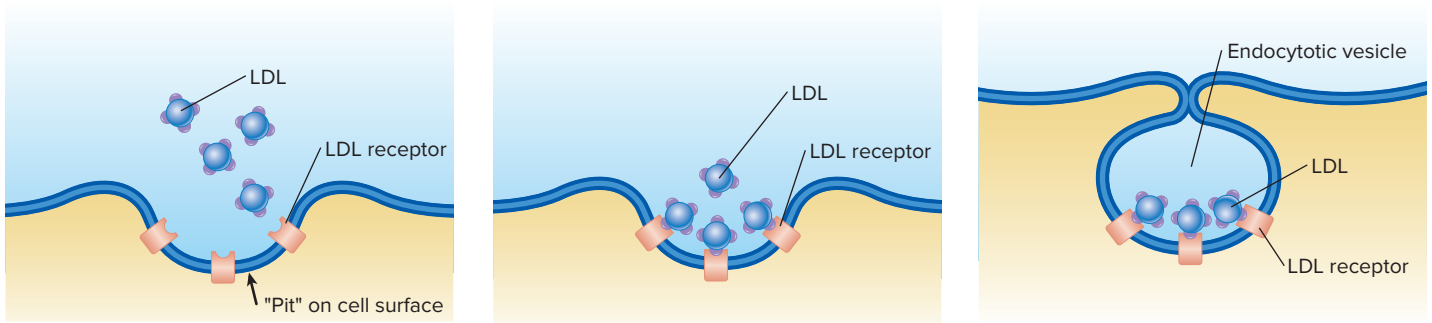


FIGURE 24.31 Lipoproteins

Cholesterol and lipids are transported through the blood by lipoproteins that are classified based on their density. A low protein content results in a low density. Chylomicrons are lipoproteins with extremely low density.



1 Cells have pits on the surface, which contain LDL receptors.

2 LDL binds to the LDL receptors in the pits.

3 The LDL, bound to LDL receptors, is taken into the cell by endocytosis.

PROCESS FIGURE 24.32 Transport of LDL into Cells

The LDL receptor transports LDL into cells by endocytosis.

? *Predict the consequence of a mutation that prevents endocytosis of LDL receptors.*

enter the lymphatic capillaries rather than the blood capillaries because the lymphatic capillaries lack a basement membrane and are more permeable to large particles, such as chylomicrons, which are about 0.3 mm in diameter. They travel through the lymphatic system via the thoracic duct to the bloodstream and then by the blood to adipose tissue. Before entering the adipose cells, triglycerides break back down into fatty acids and glycerol, which enter the adipocytes and are once more converted to triglycerides. Triglycerides are stored in adipose tissue until an energy source is needed elsewhere in the body. In the liver, the chylomicron lipids are stored, converted into other molecules, or used as energy. The chylomicron remnant, minus the triglyceride, is conveyed through the blood to the liver, where it breaks up.

Because lipids are either insoluble or only slightly soluble in water, they are transported through the blood in combination with proteins, which are water-soluble. Lipids combined with proteins are called **lipoproteins** and are categorized as high- or low-density (figure 24.31). *Density* describes the compactness of a substance and is the ratio of mass to volume. Lipids are less dense than water and tend to float in water. Proteins, which are denser than water, tend to sink in water. A lipoprotein with a high lipid content has a very low density, whereas a lipoprotein with a high protein content has a relatively high density. Chylomicrons, which are made up of 99% lipid and only 1% protein, are lipoproteins with an extremely low density. The other major transport lipoproteins are **very low-density lipoprotein (VLDL),** which is 92% lipid and 8% protein; **low-density lipoprotein (LDL),** which is 75% lipid and 25% protein; and **high-density lipoprotein (HDL),** which is 55% lipid and 45% protein (figure 24.31).

About 15% of the cholesterol in the body is ingested in the food we eat. Eating foods containing saturated fatty acids can raise plasma cholesterol levels by stimulating LDL production and inhibiting LDL receptor production. Conversely, ingesting unsaturated fatty acids lowers plasma cholesterol. Replacing fats with carbohydrates in the diet can also reduce blood cholesterol. The remaining 85% is manufactured in body cells, mostly in the liver and intestinal mucosa. Most of the cholesterol and other lipids taken into or manufactured in the liver leave the liver in the form of VLDL. Most of the triglycerides are removed from the VLDL to be stored in adipose tissue; as a result, VLDL becomes LDL.

The cholesterol in LDL is critical for the production of steroid hormones and bile salts in the liver. It is also an important component of plasma membranes. Abnormally low cholesterol levels may lead to weakened blood vessel walls and an increased risk for cerebral hemorrhage.

LDL is delivered to cells of various tissues through the blood. Cells have **LDL receptors** in "pits" on their surfaces, which bind the LDL. Once LDL is bound to the receptors, the pits on the cell surface become endocytotic vesicles, and the cell takes in LDL by receptor-mediated endocytosis (figure 24.32). For example, each fibroblast has 20,000–50,000 LDL receptors on the surface. However, those receptors are confined to cell surface pits, which occupy only 2% of the cell surface. Once inside the cell, the endocytotic vesicle combines with a lysosome, and the LDL components are separated for use in the cell.

Cells not only take in cholesterol and other lipids from LDLs but also make their own cholesterol. When the combined intake and manufacture of cholesterol exceeds a cell's needs, a negative-feedback system reduces the amount of LDL receptors and cholesterol manufactured by the cell. Excess lipids are also packaged into HDLs by the cells. These are transported back to the liver for recycling or excretion in bile.

LDL is commonly considered "bad" because, when in excess, it deposits cholesterol in arterial walls. On the other hand, HDL is considered "good" because it transports cholesterol from the tissues via blood to the liver for removal from the body in the bile. A high HDL/LDL ratio in the blood is related to a lower risk for heart disease. Low HDL levels are linked to obesity, and weight reduction increases HDL levels. Aerobic exercise can decrease LDL levels and increase HDL levels.

Proteins

Proteins are taken into the body from a number of dietary sources. Digestion of proteins begins in the stomach. **Pepsin** secreted by the stomach catalyzes the cleavage of covalent bonds in proteins to produce smaller polypeptide chains. Pepsin digests as much as 10–20% of the total ingested protein. Once the proteins and

Clinical GENETICS 24.1 Familial Hypercholesterolemia

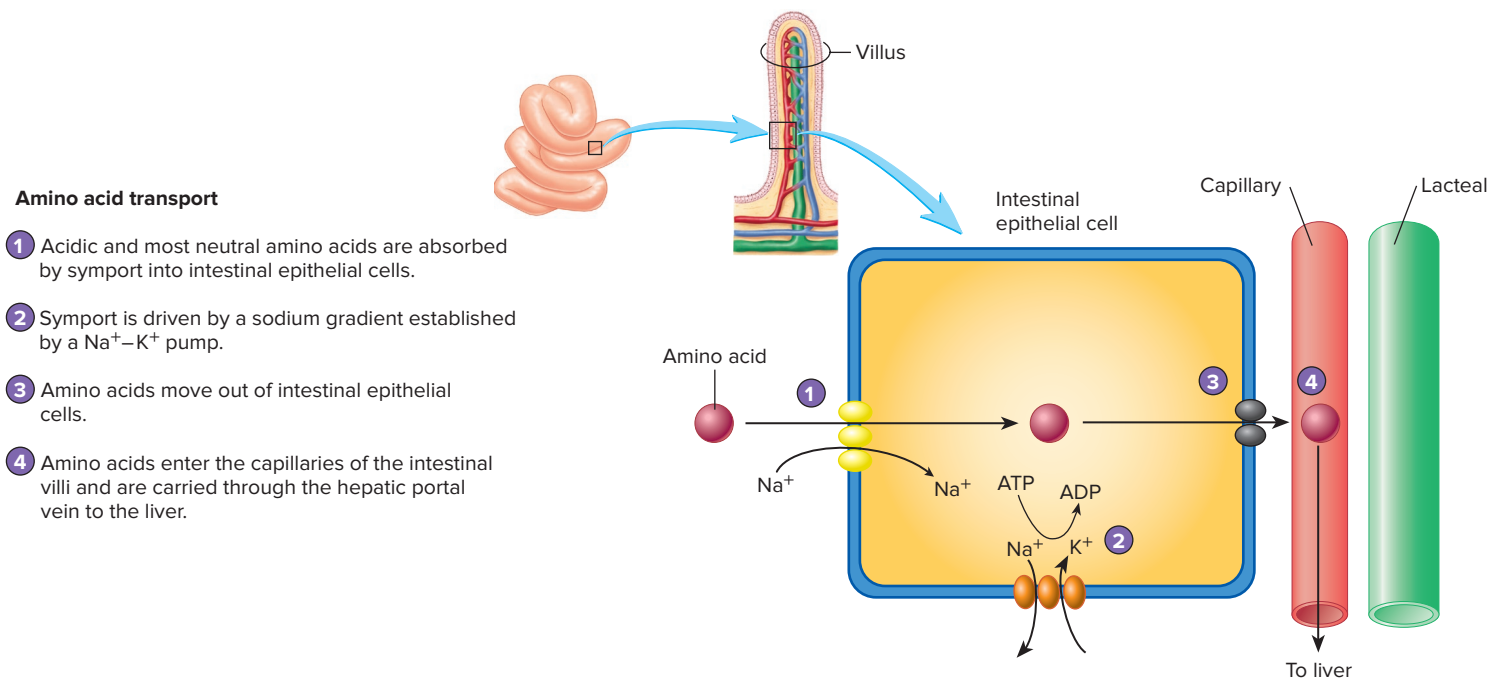
Familial hypercholesterolemia (FH) is a common genetic disorder in Europe and North America that affects 1 out of 500 people. The clinical sign of the disease is increased blood levels of LDL cholesterol. The elevated cholesterol levels accelerate the development of atherosclerosis, which often leads to coronary artery disease and heart attacks among people in their forties and fifties. Another common feature of FH is the presence of **xanthomas** (zan-thō′măs), which are nodules of cholesterol and other lipids just under the skin, especially at joints.

FH is caused by mutations in the LDL receptor gene that result in defective LDL receptors. The LDL receptor normally removes cholesterol from the blood by transporting LDL cholesterol into cells. Once inside the cell, LDL cholesterol is metabolized, and cholesterol synthesis is inhibited by a negative-feedback mechanism. When less LDL cholesterol is transported into cells, blood LDL cholesterol rises for two reasons: (1) The normal removal of LDL cholesterol does not occur, and (2) there is less inhibition of cholesterol synthesis. The usual treatment for FH is statin drugs, which lower blood LDL levels by inhibiting the synthesis of cholesterol.

The severity of FH depends on whether a person is homozygous or heterozygous. Homozygous individuals have two mutant alleles and completely lack LDL receptors. These patients have the most severe form of FH, with very high blood LDL cholesterol levels, and they often have heart attacks in their teens. Most FH patients are heterozygous, with one normal and one mutant allele. These patients have half the number of normal LDL receptors and are at increased risk for heart attacks at midlife. Although genetic testing is not yet standard, automated assays are available for the more common mutations in the LDL receptor, and they can allow early diagnosis and treatment before life-threatening symptoms develop. In the future, FH will be an excellent candidate for gene therapy because it is a severe but well-studied single-gene defect.

polypeptide chains leave the stomach, **pancreatic proteases,** continue the digestive process to produce small peptide chains (see figure 24.28). There are three major pancreatic proteases: (1) trypsin, (2) chymotrypsin, and (3) carboxypeptidase. The protein digestion products are finally broken down into tripeptides, dipeptides, and amino acids by **peptidases** bound to the microvilli of the small intestine. Each peptidase is specific for a certain peptide chain length or for a certain amino acid sequence.

Basic, acidic, and neutral amino acids are transported into the epithelial cells by separate carrier molecules. Acidic and most neutral amino acids enter by symport with a Na^+ gradient (figure 24.33), similar to the mechanism used for glucose transport. Basic amino acids enter the epithelial cells by facilitated diffusion. Dipeptides and tripeptides enter intestinal epithelial cells by a H^+ symport mechanism analogous to Na^+ symport. The total amount of each amino acid that enters the intestinal epithelial cells as dipeptides or



PROCESS FIGURE 24.33 Amino Acid Transport Across the Intestinal Epithelium

Most amino acid absorption occurs by symport powered by a Na^+ gradient.

How is transport of acidic and neutral amino acids across the intestinal epithelia similar to transport of glucose and galactose?

tripeptides is considerably more than the amount that enters as single amino acids. Once inside the cells, dipeptidases and tripeptidases split the dipeptides and tripeptides into their component amino acids. Individual amino acids then leave the epithelial cells and enter the hepatic portal system, which transports them to the liver (figure 24.33). The amino acids may be modified in the liver or released into the bloodstream and distributed throughout the body.

Amino acids are actively transported into the various cells of the body. This transport is stimulated by growth hormone and insulin. Most amino acids serve as building blocks to form new proteins (see chapter 2), but some amino acids may be used for energy.

Water

About 9 L of **water** enter the digestive tract each day as a combination of ingested and secreted fluids. Of this 9 L, about 92% is absorbed in the small intestine, and another 6–7% is absorbed in the large intestine (figure 24.34). Water moves in either direction across the wall of the small intestine by osmosis. Osmotic gradients across the epithelium determine the direction of this diffusion. When the chyme is dilute, water is absorbed by osmosis across the intestinal wall into the blood. When the chyme is very concentrated and contains very little water, water moves by osmosis into the lumen of the small intestine. As nutrients are absorbed in the small intestine, its osmotic pressure decreases; as a consequence, water moves from the small intestine into the surrounding extracellular fluid. Water in the extracellular fluid can then enter the blood. Because of the osmotic gradient produced as nutrients are absorbed in the small intestine, nearly all the water that enters the small intestine by way of the oral cavity, stomach, or intestinal secretions is reabsorbed.

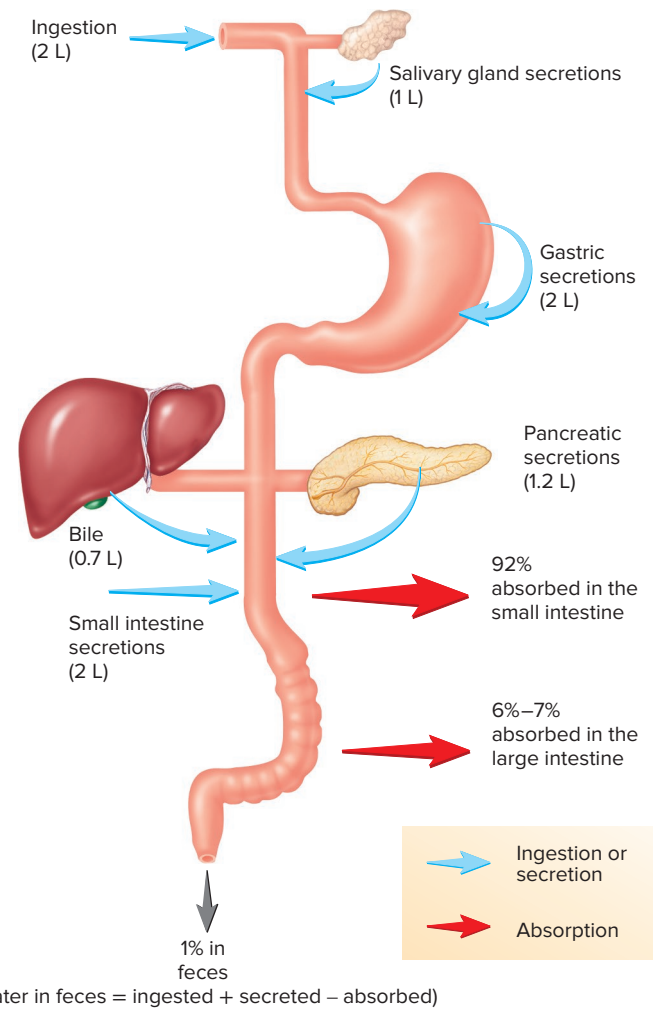


FIGURE 24.34 Fluid Volumes in the Digestive Tract
Fluid movement across the digestive tract varies depending on the particular segment.

An effective rehydration strategy is to drink water containing sodium and glucose. As sodium and glucose are absorbed by symport across the intestinal epithelium, water follows by osmosis. As an added value, this strategy also replaces ions and provides an immediate energy source. Most sports drinks contain sodium and glucose, which efficiently rehydrate the athlete. The same principle is used in **oral rehydration therapy** for severe diarrhea. This simple, cheap treatment is especially valuable in Third World countries where people often die from diarrhea caused by intestinal infections.

Ions

Ions are predominantly absorbed by active transport in the small intestine. Active transport mechanisms drive the absorption of (1) **Na^+**, (2) **K^+**, (3) **Ca^{2+}**, (4) **Mg^{2+}**, (5) **PO_4^-**. **Chloride** ions move passively through the intestinal wall of the duodenum and the jejunum following the positively charged Na^+, but Cl^- is actively transported from the ileum. Although Ca^{2+} is actively transported along the entire length of the small intestine, vitamin D is required for that transport process. The absorption of Ca^{2+} is under hormonal control, as are its excretion and storage. Parathyroid hormones, calcitonin, and vitamin D all play a role in regulating blood levels of Ca^{2+} (see chapters 6, 18, and 27).

ASSESS YOUR PROGRESS

58. *Describe the mechanism of absorption and the route of transport for water-soluble and lipid-soluble molecules.*

59. *Describe the enzymatic digestion of carbohydrates, lipids, and proteins. List where each step of digestion occurs and the breakdown products of each step.*

60. *Explain how lipids are emulsified. Describe the role of micelles, chylomicrons, VLDLs, LDLs, and HDLs in the absorption and transport of lipids in the body.*

61. *Explain how tripeptides, dipeptides, and amino acids enter intestinal epithelial cells.*

62. *Describe the movement of water through the intestinal wall.*

63. *When and where are various ions absorbed?*

24.15 Effects of Aging on the Digestive System

LEARNING OUTCOME

After reading this section, you should be able to

A. **Discuss the effects of aging on the digestive system.**

Systems PATHOLOGY 24.1 Diarrhea

Background Information

Tom, a tourist visiting a developing country, started to experience sharp pains in his abdominal region. He also began to feel hot and sweaty and felt an extreme urge to defecate. Tom anxiously inquired about the nearest facility. Once the immediate crisis was taken care of, Tom and his wife went back to their hotel room, where they remained while Tom recovered. During the next 2 days, his stools were frequent and watery. He also vomited a couple of times. Tom was encouraged to rest and drink plenty of fluids. He was feeling much better, although a little weak, in a couple of days. Diarrhea is one of the most common complaints in clinical medicine. Diarrhea affects more than half the tourists in developing countries (figure 24.35), where it may result from eating food to which the digestive tract is not accustomed or from ingesting food or water contaminated with microorganisms.

Diarrhea is any change in bowel habits involving increased stool frequency or fluidity (figure 24.36). It is not a disease in itself, but it can be a symptom of a wide variety of disorders. Diarrhea that lasts less than 2–3 weeks is acute diarrhea; diarrhea lasting longer is considered chronic. Acute diarrhea is usually self-limiting, but some forms of diarrhea can be fatal if not treated. Diarrhea results from either a decrease in fluid absorption in the intestine or an increase in fluid secretion. It can also be caused by increased bowel motor activity that moves chyme rapidly through the small intestine, so that more water enters the colon. Normally, about 600 mL of fluid enter the colon each day, and all but 150 mL are reabsorbed. The loss of more than 200 mL of fluid per day in the stool is considered abnormal.

Secretion of mucus by the colon increases dramatically in response to diarrhea. This mucus contains large quantities of bicarbonate ions, which come from dissociation of carbonic acid into bicarbonate ions and hydrogen ions within the blood supply to the colon. The bicarbonate ions enter the mucus secreted by the colon, whereas the hydrogen ions remain in the circulation; as a result, the blood pH decreases. Thus, a condition called metabolic acidosis can develop (see chapter 27).

Diarrhea is usually caused by bacteria, viruses, amoebic parasites, or chemical toxins. Symptoms can begin from as little as 1–2 hours after bacterial toxins are ingested to as long as 24 hours or more for some strains of bacteria. Nearly any bacterial species is capable of causing diarrhea. Some types of bacterial diarrhea are associated with severe vomiting, whereas others are not. Some bacterial toxins also induce fever. Identifying the causal organism usually requires laboratory analysis of the food or stool but, in cases of acute diarrhea, the infectious agent is seldom identified.

Treatment of diarrhea involves replacing lost fluids and ions (figure 24.36). The diet should be limited to clear fluids during at least the first day or so. Medicines that may help combat diarrhea include bismuth subsalicylate (sŭb-să-lis′i-lāt), which increases mucus and HCO_3^- secretion and decreases pepsin activity, and loperamide (lō-per′ă-mīd), which slows intestinal motility. Patients should avoid milk and milk products. Breads, rice, and baked fish or chicken can be added to the diet as the person's condition improves. A normal diet can be resumed after 2–3 days.

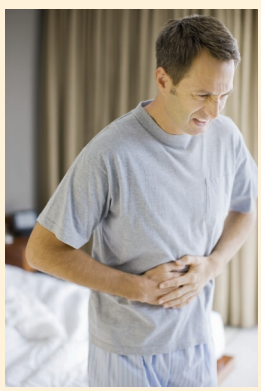


FIGURE 24.35 Symptoms of Diarrhea Often Begin with Sharp Stomach Cramps

Predict 8

Predict the effects of prolonged diarrhea.

There are a variety of diseases and disorders associated with the digestive system (table 24.4). As a person ages, gradual changes occur throughout the digestive tract. The connective tissue layers of the digestive tract—the submucosa and serosa—tend to thin. The blood supply to the digestive tract decreases. The number of smooth muscle cells in the muscularis also decreases, resulting in reduced motility in the digestive tract. In addition, goblet cells within the mucosa secrete less mucus. Glands along the digestive tract, such as the gastric glands, the liver, and the pancreas, also tend to secrete less with age. However, these changes by themselves do not appreciably decrease the function of the digestive system.

Through the years, the digestive tract, like the skin and lungs, is directly exposed to materials from the outside environment. Some of those substances can cause mechanical damage to the digestive tract, and others are toxic to the tissues. Because the connective tissue of the digestive tract becomes thin with age and because the protective mucous covering is reduced, an elderly person's digestive tract becomes less and less protected from these outside influences. In addition, the mucosa of elderly people tends to heal more slowly following injury. Declines also occur in the liver's ability to detoxify certain chemicals, the hepatic phagocytic cells' ability to remove particulate contaminants, and the liver's ability to store glycogen. These problems worsen in people who smoke.

The overall decline in the defenses of the digestive tract leaves elderly people more susceptible to infections and toxic agents. Elderly people are therefore more likely to develop ulcerations and cancers of the digestive tract. Colorectal cancers, for example, are the second-leading cause of cancer deaths in the United States, with an estimated 135,000 new cases and 57,000 deaths each year.

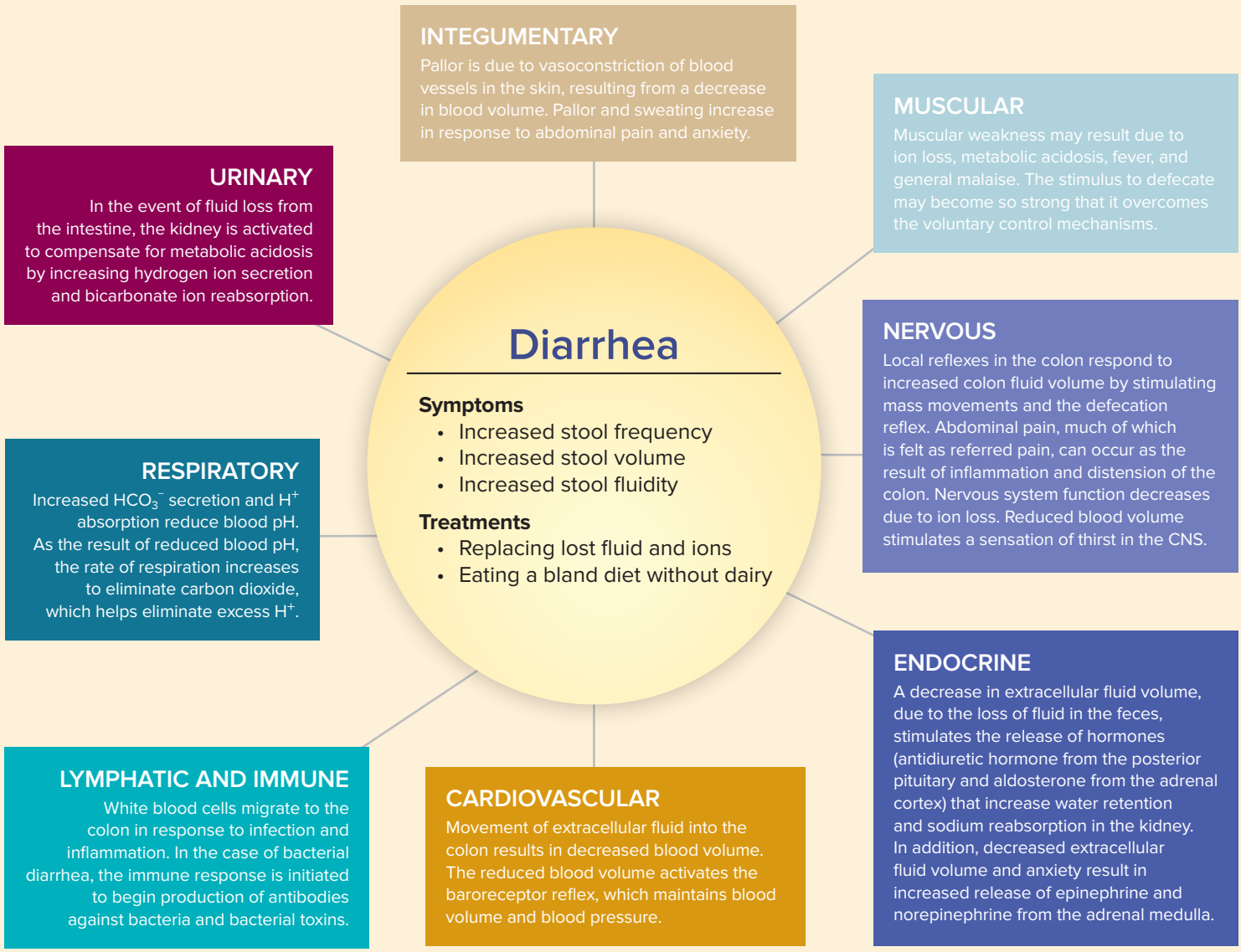


FIGURE 24.36 Symptoms and Treatments of Diarrhea

Multiple systems are affected by diarrhea.

Gastroesophageal reflux disorder increases with advancing age. It is probably the main reason that elderly people take antacids, H_2 antagonists, and proton pump inhibitors. Disorders that are not necessarily age-induced, such as hiatal hernia and irregular or inadequate esophageal motility, can be worsened by the effects of aging because of general decreased motility in the digestive tract.

The enamel on the surface of elderly people's teeth becomes thinner with age and may expose the underlying dentin. In addition, the gingiva covering the tooth root recedes, exposing additional dentin. Exposed dentin may become painful and change the person's eating habits. Many elderly people also lose teeth, which can have a marked effect on eating habits unless they are fitted with artificial teeth. The muscles of mastication tend to become weaker; as a result, older people tend to chew their food less before swallowing.

Another age-related complication in the digestive system involves the way medications and other chemicals are absorbed from the digestive tract. The decreased mucous covering and the thinned connective tissue layers allow chemicals to pass more readily from the digestive tract into the blood. However, a decline in the blood supply to the digestive tract hinders the absorption of such chemicals. Drugs administered to treat cancer, which occurs in many elderly people, may irritate the mucosa of the digestive tract, resulting in nausea and loss of appetite.

ASSESS YOUR PROGRESS

64. *List the effects of aging on the digestive system.*

65. *What are the effects of the overall decline in the defenses of the digestive tract with advancing age?*

66. *Explain why absorption of medication is a problem with aging.*

TABLE 24.4 Representative Diseases and Disorders of the Digestive System

Condition	Description
Stomach	
Vomiting	Contraction of the diaphragm and abdominal muscles and relaxation of the esophageal sphincters to forcefully expel gastric contents; vomiting reflex is initiated by irritation of the stomach or small intestine
Peptic ulcer	Lesions in the lining of the stomach or duodenum, usually due to infection by the bacterium *Helicobacter pylori;* stress, diet, smoking, or alcohol may be a predisposing factor; antibiotic therapy is the accepted treatment
Liver	
Cirrhosis (sir-ō′sis)	Characterized by damage and death of hepatic cells and replacement by connective tissue; results in loss of normal liver function and interference with blood flow through the liver; a common consequence of alcoholism
Hepatitis (hep-ă-ti′tis)	Inflammation of the liver that causes liver cell death and replacement by scar tissue; if not corrected, results in loss of liver function and eventually death; symptoms include nausea, abdominal pain, fever, chills, malaise, and jaundice; caused by any of seven distinct viruses
Hepatitis A	Infectious hepatitis; usually transmitted by poor sanitation practices or from mollusks living in contaminated waters
Hepatitis B	Serum hepatitis; usually transmitted through blood or other body fluids through either sexual contact or contaminated hypodermic needles
Hepatitis C	Often a chronic disease leading to cirrhosis and possibly cancer of the liver
Gallstones	Most often due to excess cholesterol in the bile; gallstones can enter the cystic duct, where they block the release of bile and/or pancreatic enzymes, which interferes with digestion
Intestine	
Inflammatory bowel disease (IBD)	Localized inflammatory degeneration that may occur anywhere along the digestive tract but most commonly involves the distal ileum and proximal colon; the intestinal wall often becomes thickened, constricting the lumen, with ulcers and fissures in the damaged areas; symptoms include diarrhea, abdominal pain, fever, fatigue, and weight loss; cause is unknown; treatments involve anti-inflammatory drugs, avoidance of foods that produce symptoms, and surgery in some cases; also called Crohn disease or ulcerative colitis
Irritable bowel syndrome (IBS)	Disorder of unknown cause marked by alternating bouts of constipation and diarrhea; may be linked to stress or depression; high familial incidence
Gluten enteropathy (celiac disease)	Malabsorption in the small intestine due to the effects of gluten, a protein in certain grains, especially wheat; the reaction can destroy newly formed epithelial cells, causing the intestinal villi to become blunted and decreasing the intestinal surface, which reduces absorption of nutrients
Constipation (kon-sti-pā′shŭn)	Slow movement of feces through the large intestine, causing the feces to become dry and hard because of increased fluid absorption while being retained; often results from inhibiting normal defecation reflexes; spasms of the sigmoid colon resulting from irritation can also result in slow feces movement and constipation; high-fiber diet can be preventive
Infections of the Digestive Tract	
Food poisoning	Caused by ingesting bacteria or toxins, such as *Staphylococcus aureus, Salmonella,* or *Escherichia coli;* symptoms include nausea, abdominal pain, vomiting, and diarrhea; in severe cases, death can occur
Typhoid (tī′foyd) fever	Caused by a virulent strain of the bacterium *Salmonella typhi,* which can cross the intestinal wall and invade other tissues; symptoms include severe fever, headaches, and diarrhea; usually transmitted through poor sanitation practices; leading cause of death in many developing countries
Cholera (kol′er-a)	Caused by a bacterium, *Vibrio cholerae,* in contaminated water; bacteria produce a toxin that stimulates the secretion of chloride, bicarbonate, and water into the large intestine, resulting in severe diarrhea; the loss of as much as 12–20 L of fluid per day causes shock and even death; still a major health problem in parts of Asia
Giardiasis (jē-ar-dī′a-sis)	Caused by a protozoan, *Giardia lamblia,* invading the large intestine; symptoms include nausea, abdominal cramps, weakness, weight loss, and malaise; the protozoans are transmitted in the feces of humans and other animals, often by drinking from contaminated wilderness streams
Intestinal parasites	Common under conditions of poor sanitation; parasites include tapeworms, pinworms, hookworms, and roundworms
Diarrhea (dī-ă-rē′ă)	Intestinal mucosa secretes large amounts of water and ions due to irritation, inflammation, or infection; diarrhea moves feces out of the large intestine more rapidly and speeds recovery
Dysentery (dis′en-tār-ē)	Severe form of diarrhea with blood or mucus in the feces; can be caused by bacteria, protozoa, or amoebae

Answer

Learn to Predict

In this chapter we learned that the gallbladder stores bile, a secretion of the liver that neutralizes stomach acids and emulsifies lipids. We also learned that gallstones may form when there is an abundance of cholesterol in the bile, such as from a high-cholesterol diet. Gallstones can block the cystic duct, blocking the flow of bile from the gallbladder to the duodenum. Eating food high in fat causes gallbladder contractions. Specifically, gallbladder contraction is stimulated hormonally by cholecystokinin from the duodenum and parasympathetic stimulation. If Rebecca's cystic duct were blocked by gallstones, the increased pressure in the contracting gallbladder would result in pain and inflammation.

But why did Rebecca's skin turn yellow? Recall that bile contains bilirubin, a yellow pigment produced from the breakdown of hemoglobin that when further processed in the intestine turns brown and contributes to the normal color of feces. The gallstones blocked Rebecca's common bile duct, preventing bile from passing from the liver to the duodenum and reducing the amount of bilirubin removed from the blood. Rebecca's skin turned yellow due to the accumulation of bile pigments in the blood. Also, the reduced volume of bile entering the duodenum resulted in poor emulsification of lipids. The lipids remained in the small intestine, causing distension of the intestine, and the undigested lipids passing through the small and large intestine were responsible for the diarrhea. The lack of bilirubin resulted in the clay-colored feces.

Answers to the odd-numbered Predict questions for this chapter appear in appendix E.

Summary

24.1 Anatomy of the Digestive System

1. The digestive system consists of the digestive tract and its associated accessory organs.
2. The digestive tract includes the oral cavity, pharynx and esophagus, stomach, small intestine, and large intestine.
3. Accessory organs include the salivary glands, tonsils, liver, gallbladder, and pancreas.

24.2 Functions of the Digestive System

The functions of the digestive system are ingestion, propulsion, and mixing, secretion, digestion, absorption, and storage and elimination.

24.3 Histology of the Digestive Tract

The digestive tract is composed of four tunics: mucosa, submucosa, muscularis, and serosa or adventitia.

Mucosa

1. The mucosa consists of a mucous epithelium, a lamina propria, and a muscularis mucosae.
2. The epithelium extends into the lamina propria to form intestinal glands.

Submucosa

The submucosa is a connective tissue layer containing the submucosal plexus, blood vessels, and small glands.

Muscularis

1. The muscularis consists of an inner layer of circular smooth muscle and an outer layer of longitudinal smooth muscle.
2. The myenteric plexus is between the two muscle layers.
3. Interstitial pacemaker cells are located throughout the myenteric plexus.

Serosa or Adventitia

The serosa or adventitia forms the outermost layer of the digestive tract.

24.4 Regulation of the Digestive System

Nervous, hormonal, and local chemical mechanisms regulate digestion.

Nervous Regulation of the Digestive System

Nervous regulation involves the ENS and CNS reflexes.

Chemical Regulation of the Digestive System

1. Over 30 neurotransmitters are associated with the ENS.
2. The digestive tract produces hormones that regulate digestion.
3. Other chemicals produced by the digestive tract exercise local control of digestion.

24.5 Peritoneum

1. The peritoneum is a serous membrane that lines the abdominal cavity and organs.
2. Mesenteries are peritoneum that extends from the body wall to many of the abdominal organs.
3. Retroperitoneal organs are located behind the peritoneum.

24.6 Oral Cavity

The oral cavity includes the vestibule and the oral cavity proper.

Lips, Cheeks, and Palate

1. The lips and cheeks are involved in facial expression, mastication, and speech.
2. The roof of the oral cavity is divided into the hard and soft palates.
3. The palatine tonsils are located in the lateral wall of the fauces.

Tongue

1. The tongue is involved in speech, taste, mastication, and swallowing.
2. The intrinsic tongue muscles change the shape of the tongue, and the extrinsic tongue muscles move the tongue.
3. The anterior two-thirds of the tongue is covered with papillae; the posterior one-third is devoid of papillae.

Teeth

1. Twenty deciduous teeth are replaced by 32 permanent teeth.
2. The types of teeth are incisors, canines, premolars, and molars.
3. A tooth consists of a crown, a neck, and a root.
4. The root is composed of dentin. Within the dentin of the root is the pulp cavity, which is filled with pulp, blood vessels, and nerves. The crown is dentin covered by enamel.
5. Periodontal ligaments hold the teeth in the alveoli.

Mastication

The muscles of mastication are the temporalis, masseter, medial pterygoid, and lateral pterygoid.

Salivary Glands

1. Salivary glands produce serous and mucous secretions.
2. The three pairs of large salivary glands are the parotid, submandibular, and sublingual.

24.7 Swallowing

Swallowing involves the pharynx and esophagus. It is divided into three phases.

Pharynx

The pharynx consists of the nasopharynx, oropharynx, and laryngopharynx.

Esophagus

1. The esophagus connects the pharynx to the stomach. The upper and lower esophageal sphincters regulate movement.
2. The esophagus consists of an outer adventitia, a muscular layer (longitudinal and circular), a submucosal layer (with mucous glands), and a stratified squamous epithelium.

Swallowing Phases

1. During the voluntary phase of swallowing, a bolus of food is moved by the tongue from the oral cavity to the pharynx.
2. The pharyngeal phase is a reflex caused by the stimulation of stretch receptors in the pharynx.
   - The soft palate closes the nasopharynx, and the epiglottis and vestibular folds close the opening into the larynx.
   - Pharyngeal muscles move the bolus to the esophagus.
3. The esophageal phase is a reflex initiated by the stimulation of stretch receptors in the esophagus. A wave of contraction (peristalsis) moves the food to the stomach.

24.8 Stomach

Anatomy of the Stomach

The openings of the stomach are the gastroesophageal (to the esophagus) and the pyloric (to the duodenum).

Histology of the Stomach

1. The wall of the stomach consists of an external serosa, a muscle layer (longitudinal, circular, and oblique), a submucosa, and simple columnar epithelium (surface mucous cells).
2. Rugae are the folds in the stomach when it is empty.
3. Gastric pits are the openings to the gastric glands, which contain mucous neck cells, parietal cells, chief cells, and endocrine cells.

Secretions of the Stomach

1. Hydrochloric acid promotes pepsin activity and kills microorganisms.
2. Intrinsic factor is necessary for vitamin B_{12} absorption in the small intestine.
3. Mucus protects the stomach lining.
4. Pepsinogen is converted to pepsin, which digests proteins. Gastric lipase digests lipids.
5. The sight, smell, taste, or thought of food initiates the cephalic phase. Nerve impulses from the medulla stimulate hydrochloric acid, pepsinogen, gastrin, and histamine secretion.
6. Distension of the stomach, which stimulates gastrin secretion and activates CNS and local reflexes that promote secretion, initiates the gastric phase.
7. Acidic chyme, which enters the duodenum and stimulates neuronal reflexes and the secretion of hormones that inhibit gastric secretions, initiates the intestinal phase.

Movements of the Stomach

1. The stomach stretches and relaxes to increase volume.
2. Mixing waves mix the stomach contents with stomach secretions to form chyme.
3. Peristaltic waves move the chyme into the duodenum.
4. Gastrin and stretching of the stomach stimulate stomach emptying.
5. Chyme entering the duodenum inhibits movement through neuronal reflexes and the release of hormones.

24.9 Small Intestine

The small intestine is divided into the duodenum, jejunum, and ileum.

Anatomy and Histology of the Small Intestine

1. Circular folds, villi, and microvilli greatly increase the surface area of the intestinal lining.
2. Absorptive, goblet, and endocrine cells are in intestinal glands. Duodenal glands produce mucus.

Secretions of the Small Intestine

1. Mucus protects against digestive enzymes and stomach acids.
2. Digestive enzymes (disaccharidases and peptidases) are bound to the intestinal wall.
3. The vagus nerve, secretin, and chemical or tactile irritation stimulate intestinal secretion.

Movement in the Small Intestine

1. Segmental contractions mix intestinal contents. Peristaltic contractions move materials distally.
2. Stretch of smooth muscles, local reflexes, and the parasympathetic nervous system stimulate contractions. Distension of the cecum initiates a reflex that inhibits peristalsis.

24.10 Liver

Anatomy of the Liver

1. The liver has four lobes: right, left, caudate, and quadrate.
2. The liver is divided into lobules.
   - The hepatic cords are composed of columns of hepatocytes separated by the bile canaliculi.
   - The sinusoids are enlarged spaces filled with blood and lined with endothelium and hepatic phagocytic cells.

Histology of the Liver

1. The portal triads supply the lobules.
   - The hepatic arteries and the hepatic portal veins take blood to the lobules and empty into the sinusoids.
   - The sinusoids empty into central veins, which join to form the hepatic veins, which leave the liver.
   - Bile canaliculi converge to form hepatic ducts, which leave the liver.
2. Bile leaves the liver through the hepatic duct system.
   - The hepatic ducts receive bile from the lobules.
   - The cystic duct from the gallbladder joins the hepatic duct to form the common bile duct.
   - The common bile duct joins the pancreatic duct at the point at which it empties into the duodenum.

Functions of the Liver

1. The liver produces bile, which contains bile salts that emulsify lipids.
2. The liver stores and processes nutrients, detoxifies harmful chemicals, and synthesizes new molecules.
3. Hepatic phagocytic cells phagocytize red blood cells, bacteria, and other debris.
4. The liver produces blood proteins.

24.11 Gallbladder

1. The gallbladder is a small sac on the inferior surface of the liver.
2. The gallbladder stores and concentrates bile.
3. Cholecystokinin stimulates gallbladder contraction.

24.12 Pancreas

Anatomy of the Pancreas

1. The pancreas is both an endocrine and an exocrine gland. Its exocrine function is the production of digestive enzymes.
2. The pancreas is divided into lobules that contain acini. The acini connect to a duct system that eventually forms the pancreatic duct, which empties into the duodenum.

Pancreatic Secretions

1. Digestive enzymes, including inactive proteolytic enzymes that are activated in the small intestine
2. A watery bicarbonate solution that neutralizes acidic chyme

Regulation of Pancreatic Secretion

Cholecystokinin and the vagus nerve stimulate the release of digestive enzymes. Secretin stimulates release of bicarbonate ions and water.

24.13 Large Intestine

Anatomy of the Large Intestine

1. The cecum forms a blind sac at the junction of the small and large intestines. The vermiform appendix is a blind tube off the cecum.
2. The ascending colon extends from the cecum superiorly to the right colic flexure. The transverse colon extends from the right to the left colic flexure. The descending colon extends inferiorly to join the sigmoid colon.
3. The sigmoid colon is an S-shaped tube that ends at the rectum.
4. Longitudinal smooth muscles of the large intestine wall are arranged into bands, called teniae coli, that contract to produce pouches called haustra.
5. The mucosal lining of the large intestine is simple columnar epithelium with mucus-producing crypts.
6. The rectum is a straight tube that ends at the anus.
7. An internal anal sphincter (smooth muscle) and an external anal sphincter (skeletal muscle) surround the anal canal.

Secretions of the Large Intestine

1. Mucus protects the intestinal lining.
2. Epithelial cells secrete HCO_3^-. Sodium is absorbed by active transport driven by the Na^+–K^+ ATPase pump, and water is absorbed by osmosis.
3. Microorganisms are responsible for vitamin K production, gas production, and much of the bulk of feces.

Movement in the Large Intestine

1. Segmental movements mix the colon's contents.
2. Mass movements are strong peristaltic contractions that occur three or four times a day.
3. Defecation is the elimination of feces. Reflex activity moves feces through the internal anal sphincter. Voluntary activity regulates movement through the external anal sphincter.

24.14 Digestion and Absorption

1. Digestion is the breakdown of organic molecules into their components.
2. Absorption is the means by which molecules are moved out of the digestive tract and distributed throughout the body.
3. Transport from the intestinal epithelium occurs by two routes.
   - Water, ions, and water-soluble products of digestion are transported to the liver through the hepatic portal system.
   - The products of lipid digestion are transported through the lymphatic system to the circulatory system.

Carbohydrates

1. Carbohydrates consist of starches, glycogen, sucrose, lactose, glucose, and fructose.
2. Polysaccharides are broken down into monosaccharides by a number of different enzymes.
3. Monosaccharides are taken up by intestinal epithelial cells by symport that is powered by a Na^+ gradient or by facilitated diffusion.
4. The monosaccharides are carried to the liver, where the nonglucose monosaccharides are converted to glucose.
5. Glucose is transported to the cells that require energy.
6. Glucose enters the cells through facilitated diffusion.
7. Insulin influences the rate of glucose transport.

Lipids

1. Lipids include triglycerides, phospholipids, steroids, and fat-soluble vitamins.

2. Lipase digests lipid molecules to form free fatty acids and monoglycerides.
3. Emulsification, the transformation of large lipid droplets into smaller droplets, is accomplished by bile salts.
4. Micelles form around lipid digestion products and move to epithelial cells of the small intestine, where the products pass into the cells by simple diffusion.
5. Within the epithelial cells, free fatty acids are combined with a monoglyceride to form triglycerides.
6. Proteins coat triglycerides, phospholipids, and cholesterol to form chylomicrons.
7. Chylomicrons enter lacteals within intestinal villi and are carried through the lymphatic system to the bloodstream.
8. Triglycerides are stored in adipose tissue, converted into other molecules, or used as energy.
9. Lipoproteins include chylomicrons, VLDL, LDL, and HDL.
10. LDL transports cholesterol to cells, and HDL transports it from cells to the liver.
11. LDLs are taken into cells by receptor-mediated endocytosis, which is controlled by a negative-feedback mechanism.

Proteins

1. Pepsin in the stomach breaks proteins into polypeptide chains.
2. Trypsin and other proteolytic enzymes from the pancreas produce smaller peptides.
3. Peptidases, bound to the microvilli of the small intestine, break down peptides.
4. Amino acids, dipeptides, and tripeptides are absorbed by symport that is powered by Na^+ or H^+ gradients or by facilitated diffusion.
5. Amino acids are transported to the liver, where the amino acids can be modified or released into the bloodstream.
6. Amino acids are actively transported into cells under the stimulation of growth hormone and insulin.
7. Amino acids are used as building blocks or for energy.

Water

Water moves in either direction across the wall of the small intestine, depending on the osmotic gradients across the epithelium.

Ions

1. Sodium, potassium, calcium, magnesium, and phosphate are actively transported.
2. Chloride ions move passively through the wall of the duodenum and jejunum but are actively transported from the ileum.
3. Calcium ions are actively transported, but vitamin D is required for transport, and the transport is under hormonal control.

24.15 Effects of Aging on the Digestive System

The mucous layer, the connective tissue, the muscles, and the secretions of the digestive tract all tend to decrease as a person ages. These changes make an older person more open to infections and toxic agents.

REVIEW AND COMPREHENSION

1. Which layer of the digestive tract is in direct contact with the food that is consumed?
   a. mucosa
   b. muscularis
   c. serosa
   d. submucosa
2. The ENS is found in
   a. the submucosa layer.
   b. the muscularis layer.
   c. the serosa layer.
   d. Both a and b are correct.
   e. All of these are correct.
3. Dentin
   a. forms the surface of the crown of the teeth.
   b. holds the teeth to the periodontal ligaments.
   c. is found in the pulp cavity.
   d. makes up most of the structure of the teeth.
   e. is harder than enamel.
4. The number of premolar deciduous teeth is
   a. 0.
   b. 2.
   c. 4.
   d. 8.
   e. 12.
5. Which of these glands does *not* secrete saliva into the oral cavity?
   a. submandibular gland
   b. pancreas
   c. sublingual gland
   d. parotid gland
6. The portion of the digestive tract in which digestion begins is the
   a. oral cavity.
   b. esophagus.
   c. stomach.
   d. duodenum.
   e. jejunum.
7. During swallowing
   a. the movement of food results primarily from gravity.
   b. the swallowing center in the medulla oblongata is activated.
   c. food is pushed into the oropharynx during the pharyngeal phase.
   d. the soft palate closes off the opening into the larynx.
8. The stomach
   a. has large folds in the submucosa and mucosa called rugae.
   b. has two layers of smooth muscle in the muscularis tunic.
   c. opening from the esophagus is the pyloric opening.
   d. has an area closest to the duodenum called the fundus.
   e. All of these are correct.
9. Which of these stomach cell types is *not* correctly matched with its function?
   a. surface mucous cells—produce mucus
   b. parietal cells—produce hydrochloric acid
   c. chief cells—produce intrinsic factor
   d. endocrine cells—produce regulatory hormones
10. Why doesn't the stomach digest itself?
    a. The stomach wall is not composed of protein, so it is not affected by proteolytic enzymes.
    b. The digestive enzymes of the stomach are not strong enough to digest the stomach wall.
    c. The lining of the stomach wall has a protective layer of epithelial cells.
    d. The stomach wall is protected by large amounts of mucus.
11. Which of these hormones stimulates stomach secretions?
    a. cholecystokinin
    b. insulin
    c. gastrin
    d. secretin

12. Which of these structures increase the mucosal surface of the small intestine?
    a. circular folds
    b. villi
    c. microvilli
    d. length of the small intestine
    e. All of these are correct.
13. Which cells in the small intestine have digestive enzymes attached to their surfaces?
    a. mucous cells
    b. goblet cells
    c. endocrine cells
    d. absorptive cells
14. The hepatic sinusoids
    a. receive blood from the hepatic artery.
    b. receive blood from the hepatic portal vein.
    c. empty into the central veins.
    d. All of these are correct.
15. Which of the following might occur if a person suffers from a severe case of hepatitis that impairs liver function?
    a. Lipid digestion is difficult.
    b. By-products of hemoglobin breakdown accumulate in the blood.
    c. Plasma proteins decrease in concentration.
    d. Toxins in the blood increase.
    e. All of these occur.
16. The gallbladder
    a. produces bile.
    b. stores bile.
    c. contracts and releases bile in response to secretin.
    d. contracts and releases bile in response to sympathetic stimulation.
    e. Both b and c are correct.
17. The aqueous pancreatic juice
    a. is secreted by the pancreatic islets.
    b. contains HCO_3^-.
    c. is released primarily in response to cholecystokinin.
    d. passes directly into the blood.
    e. All of these are correct.
18. Which of these is *not* a function of the large intestine?
    a. absorption of glucose
    b. absorption of certain vitamins
    c. absorption of water and salts
    d. production of mucus
19. Defecation
    a. can be initiated by stretch of the rectum.
    b. can occur as a result of mass movements.
    c. involves local reflexes.
    d. involves parasympathetic reflexes mediated by the spinal cord.
    e. All of these characteristics are true of defecation.
20. Which of these structures produces enzymes that digest carbohydrates?
    a. salivary glands
    b. pancreas
    c. lining of the small intestine
    d. Both a and b are correct.
    e. All of these are correct.
21. Bile
    a. is an important enzyme for the digestion of lipids.
    b. is made by the gallbladder.
    c. contains breakdown products from hemoglobin.
    d. emulsifies lipids.
    e. Both c and d are correct.
22. Micelles are
    a. lipids surrounded by bile salts.
    b. produced by the pancreas.
    c. released into lacteals.
    d. stored in the gallbladder.
    e. reabsorbed in the colon.
23. If the thoracic duct were tied off, which of these classes of nutrients would *not* enter the blood at their normal rate?
    a. amino acids
    b. glucose
    c. lipids
    d. fructose
    e. nucleotides
24. Which of these lipoprotein molecules transports excess lipids from cells back to the liver?
    a. high-density lipoprotein (HDL)
    b. low-density lipoprotein (LDL)
    c. very low-density lipoprotein (VLDL)

Answers appear in appendix F.

CRITICAL THINKING

1. While anesthetized, patients sometimes vomit. Given that the anesthetic eliminates the swallowing reflex, explain why it is dangerous for an anesthetized patient to vomit.
2. Achlorhydria is a condition in which the stomach stops producing hydrochloric acid and other secretions. What effect would achlorhydria have on the digestive process? On red blood cell count?
3. Victor experienced the pain of a duodenal ulcer during final examination week. Explain what habits caused the ulcer, and recommend possible remedies.
4. Gallstones sometimes obstruct the common bile duct. What are the consequences of such a blockage?
5. A patient has a spinal cord injury at level L2. How does this injury affect the patient's ability to defecate? What components of the defecation response are still present, and which are lost?
6. The bacterium *Vibrio cholerae* produces cholera toxin, which activates a chloride channel in the intestinal epithelium. In contrast, mutations that inactivate the same channel cause cystic fibrosis. Explain how increased chloride channel activity causes severe diarrhea, whereas decreased activity causes the intestinal symptoms of cystic fibrosis.
7. Discuss why the most effective oral rehydration therapy is water containing sodium and glucose instead of water alone or water with fructose.
8. Would a patient with familial hypercholesterolemia (FH) benefit from dietary changes?

Answers to odd-numbered questions appear in appendix G.