



Colorized SEM of the interior surface of the small intestine showing villi.

C H A P T E R

24

Digestive System

Every cell of the body needs nourishment, yet most cells cannot leave their position in the body and travel to a food source, so the food must be converted to a usable form and delivered.

The digestive system, with the help of the circulatory system, acts like a gigantic “meals on wheels,” providing nourishment to over a hundred trillion “customer” cells in the body. It also has its own quality control and waste disposal system.

The digestive system provides the body with water, electrolytes, and other nutrients. To do this, the digestive system is specialized to ingest food, propel it through the digestive tract, digest it, and absorb water, electrolytes, and other nutrients from the lumen of the gastrointestinal tract. Once these useful substances are absorbed, they are transported through the circulatory system to cells, where they are used. The undigested portion of the food is moved through the digestive tract and eliminated through the anus.

This chapter presents the general *anatomy of the digestive system* (860), followed by descriptions of the *functions of the digestive system* (860), the *histology of the digestive tract* (862), the *regulation of the digestive system* (863) and the *peritoneum* (864). The anatomy and physiology of each section of the digestive tract and its accessory structures are then presented: the *oral cavity* (866), *pharynx* (870), *esophagus* (870), along with a section on *swallowing* (872), *stomach* (872), *small intestine* (881), *liver* (884), *gallbladder* (889), *pancreas* (890), and *large intestine* (891). *Digestion, absorption, and transport* (896) of nutrients are then discussed, along with the *effects of aging on the digestive system* (901).

Anatomy of the Digestive System

Objective

- Describe the general regions of the digestive tract.

The **digestive system** (figure 24.1) consists of the **digestive tract**, a tube extending from the mouth to the anus, and its associated **accessory organs**, primarily glands, which secrete fluids into the digestive tract. The digestive tract is also called the **alimentary tract**, or **alimentary canal**. The term **gastrointestinal** (gas'trō-in-tes'tin-āl; **GI**) **tract** technically only refers to the stomach and intestines but is often used as a synonym for the digestive tract.

The regions of the digestive tract include

1. the *mouth* or *oral cavity*, which has salivary glands and tonsils as accessory organs;
2. the *pharynx*, or throat, with tubular mucous glands;
3. the *esophagus*, with tubular mucous glands;
4. the *stomach*, which contains many tubelike glands;
5. the *small intestine*, consisting of the duodenum, jejunum, and ileum, with the liver, gallbladder, and pancreas as major accessory organs;

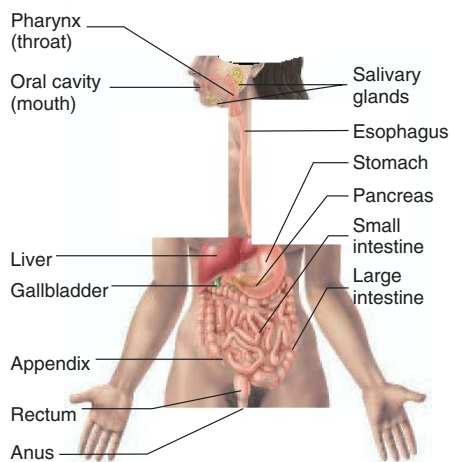


Figure 24.1 The Digestive System

6. the *large intestine*, including the cecum, colon, rectum, and anal canal, with mucous glands;
7. the *anus*.

- 1. List the major regions of the digestive tract.

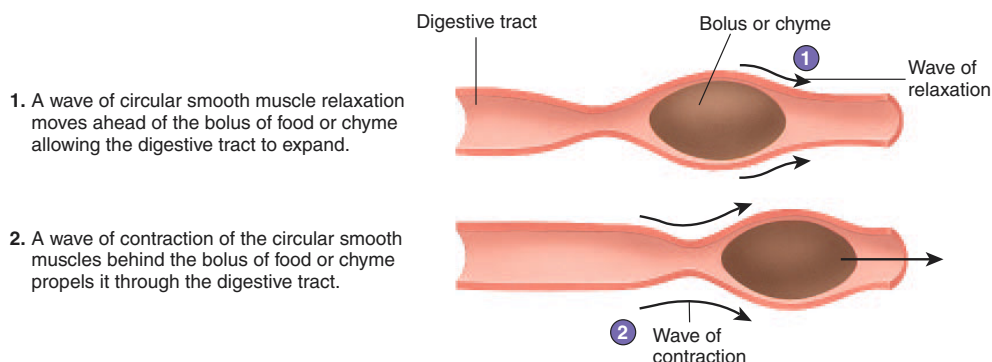
Functions of the Digestive System

Objective

- Describe the processes involved in the functioning of the digestive system.

The major functions of the digestive system are outlined as follows (table 24.1):

1. **Ingestion** is the introduction of solid or liquid food into the stomach. The normal route of ingestion is through the oral cavity, but food can be introduced directly into the stomach by a nasogastric, or stomach, tube.
2. **Mastication** is the process by which food taken into the mouth is chewed by the teeth. Digestive enzymes cannot easily penetrate solid food particles and can only work effectively on the surfaces of the particles. It's vital, therefore, to normal digestive function that solid foods be mechanically broken down into small particles. Mastication breaks large food particles into many smaller particles, which have a much larger total surface area than do a few large particles.
3. **Propulsion** in the digestive tract is the movement of food from one end of the digestive tract to the other. The total time that 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. **Deglutition** (dē'gloo-tish'ūn), or swallowing, moves food and liquids, called a **bolus**, from the oral cavity into the esophagus. **Peristalsis** (per-i-stal'sis; figure 24.2) is responsible for moving material through most of the digestive tract. Muscular contractions occur in **peristaltic** (per-i-stal'tik) **waves**, consisting of a wave of relaxation of the circular muscles, which forms a leading wave of distention in front of the bolus, followed by a wave of strong contraction of the



Process Figure 24.2 Peristalsis

Table 24.1 Functions of the Digestive Tract

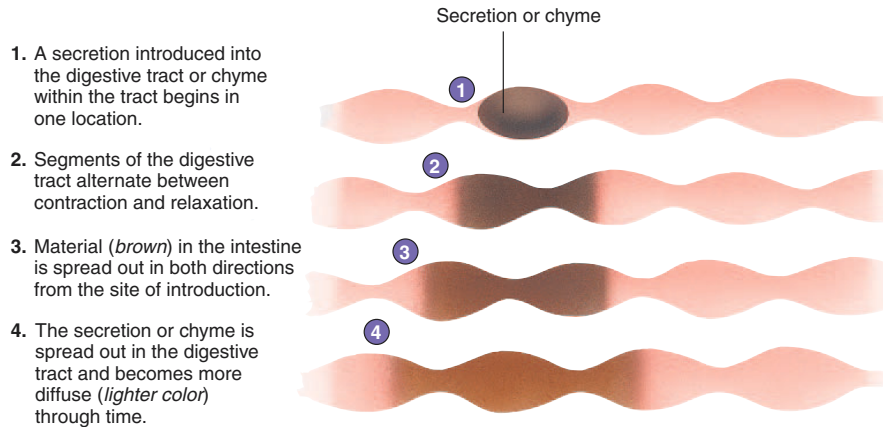
Organ	Functions
Oral cavity	<p><i>Ingestion.</i> Solid food and fluids are taken into the digestive tract through the oral cavity.</p> <p><i>Taste.</i> Tastants dissolved in saliva stimulate taste buds in the tongue.</p> <p><i>Mastication.</i> Movement of the mandible by the muscles of mastication cause the teeth to break food down into smaller pieces. The tongue and cheeks help to place the food between the teeth.</p> <p><i>Digestion.</i> Amylase in saliva begins carbohydrate (starch) digestion.</p> <p><i>Swallowing.</i> The tongue forms food into a bolus and pushes the bolus into the pharynx.</p> <p><i>Communication.</i> The lips, cheeks, teeth, and tongue are involved in speech. The lips change shape as part of facial expressions.</p> <p><i>Protection.</i> Mucin and water in saliva provides lubrication, and lysozyme kills microorganisms.</p>
Pharynx	<p><i>Swallowing.</i> 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 from entering the lower respiratory tract by the epiglottis and vestibular folds.</p> <p><i>Breathing.</i> Air passes from the nasal or oral cavity through the pharynx to the lower respiratory tract.</p> <p><i>Protection.</i> Mucus provides lubrication.</p>
Esophagus	<p><i>Propulsion.</i> Peristaltic contractions move the bolus from the pharynx to the stomach. The lower esophageal sphincter limits reflux of the stomach contents into the esophagus.</p> <p><i>Protection.</i> Glands produce mucus that provides lubrication and protects the inferior esophagus from stomach acid.</p>
Stomach	<p><i>Storage.</i> Rugae allow the stomach to expand and hold food until it can be digested.</p> <p><i>Digestion.</i> Protein digestion begins as a result of the actions of hydrochloric acid and pepsin. Intrinsic factor prevents the breakdown of vitamin B₁₂ by stomach acid.</p> <p><i>Absorption.</i> Except for a few substances (e.g., water, alcohol, aspirin) little absorption takes place in the stomach.</p> <p><i>Mixing and propulsion.</i> Mixing waves churn ingested materials and stomach secretions into chyme. Peristaltic waves move the chyme into the small intestine.</p> <p><i>Protection.</i> Mucus provides lubrication and prevents digestion of the stomach wall. Stomach acid kills most microorganisms.</p>
Small intestine	<p><i>Neutralization.</i> Bicarbonate ions from the pancreas and bile from the liver neutralize stomach acid to form a pH environment suitable for pancreatic and intestinal enzymes.</p> <p><i>Digestion.</i> Enzymes from the pancreas and the lining of the small intestine complete the breakdown of food molecules. Bile salts from the liver emulsify fats.</p> <p><i>Absorption.</i> 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.</p> <p><i>Mixing and propulsion.</i> Segmental contractions mix the chyme, and peristaltic contractions move the chyme into the large intestine.</p> <p><i>Excretion.</i> Bile from the liver contains bilirubin, cholesterol, fats, and fat-soluble hormones.</p> <p><i>Protection.</i> Mucus provides lubrication, prevents the digestion of the intestinal wall, and protects the small intestine from stomach acid. Peyer's patches protect against microorganisms.</p>
Large intestine	<p><i>Absorption.</i> The proximal half of the colon absorbs salts (e.g., sodium chloride), water, and vitamins (e.g., K) produced by bacteria.</p> <p><i>Storage.</i> The distal half of the colon holds feces until it is eliminated.</p> <p><i>Mixing and propulsion.</i> Slight segmental mixing occurs. Mass movements propel feces toward the anus and defecation eliminates the feces.</p> <p><i>Protection.</i> Mucus and bicarbonate ions protect against acids produced by bacteria.</p>

circular muscles behind the bolus, which forces the bolus along the digestive tube. Each peristaltic wave travels the length of the esophagus in about 10 seconds. Peristaltic waves in the small intestine usually only travel for short distances. In some parts of the large intestine, material is moved by **mass movements**, which are contractions that extend over much larger parts of the digestive tract than peristaltic movements.

4. *Mixing.* Some contractions don't propel food (chyme) from one end of the digestive tract to the other but rather move

the food back and forth within the digestive tract to **mix** it with digestive secretions and to help break it into smaller pieces. **Segmental contractions** (figure 24.3) are mixing contractions that occur in the small intestine.

5. *Secretion.* As food moves through the digestive tract, **secretions** are added to lubricate, liquefy, and digest the food. **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, from the damaging effect of acid



Process Figure 24.3 Segmental Contractions

in the stomach, and from the digestive enzymes of the digestive tract. The secretions also contain large amounts of **water**, which liquefies the food, thereby making it easier to digest and absorb. Water also moves into the intestine by osmosis. Liver secretions break large fat droplets into much smaller droplets, which makes possible the digestion and absorption of fats. **Enzymes** secreted by the oral cavity, stomach, intestine, and pancreas break large food molecules down into smaller molecules that can be absorbed by the intestinal wall.

6. **Digestion** is the breakdown of large organic molecules into their component parts: carbohydrates into monosaccharides, proteins into amino acids, and triglycerides into fatty acids and glycerol. Digestion consists of **mechanical digestion**, which involves mastication and mixing of food, and **chemical digestion**, which is accomplished by digestive enzymes that are secreted along the digestive tract. Digestion of large molecules into their component parts must be accomplished before they can be absorbed by the digestive tract. Minerals and water are not broken down before being absorbed. Vitamins are also absorbed without digestion and lose their function if their structure is altered by digestion.
7. **Absorption** is the movement of molecules out of the digestive tract and into the circulation 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 simple diffusion, facilitated diffusion, active transport, or cotransport (see chapter 3).
8. **Elimination** is the process by which the waste products of digestion are removed from the body. During this process, occurring primarily in the large intestine, water and salts are absorbed and change the material in the digestive tract from a liquefied state to a semisolid state. These semisolid waste products, called **feces**, are then eliminated from the digestive tract by the process of **defecation**.

2. Describe each of the processes involved in the normal functions of the digestive system.

Histology of the Digestive Tract

Objective

- Outline the basic histologic characteristics of the digestive tract.

Figure 24.4 depicts a generalized view of the digestive tract histology. The digestive tube consists of four major layers, or tunics: an internal mucosa and an external serosa with a submucosa and muscularis in between. 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 intestinal 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), 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) an outer thin smooth muscle layer, the **muscularis mucosae**.

Submucosa

The **submucosa** is a thick connective tissue layer containing nerves, blood vessels, and small glands that lies beneath the mucosa. The plexus of nerve cells in the submucosa form the **submucosal plexus** (plek'sūs; Meissner's plexus), a parasympathetic ganglionic plexus consisting of axons and many scattered cell bodies.

Muscularis

The next tunic is the **muscularis**, which 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 striated, and the stomach, which has three layers of smooth muscle. Another nerve plexus, the **myenteric plexus**

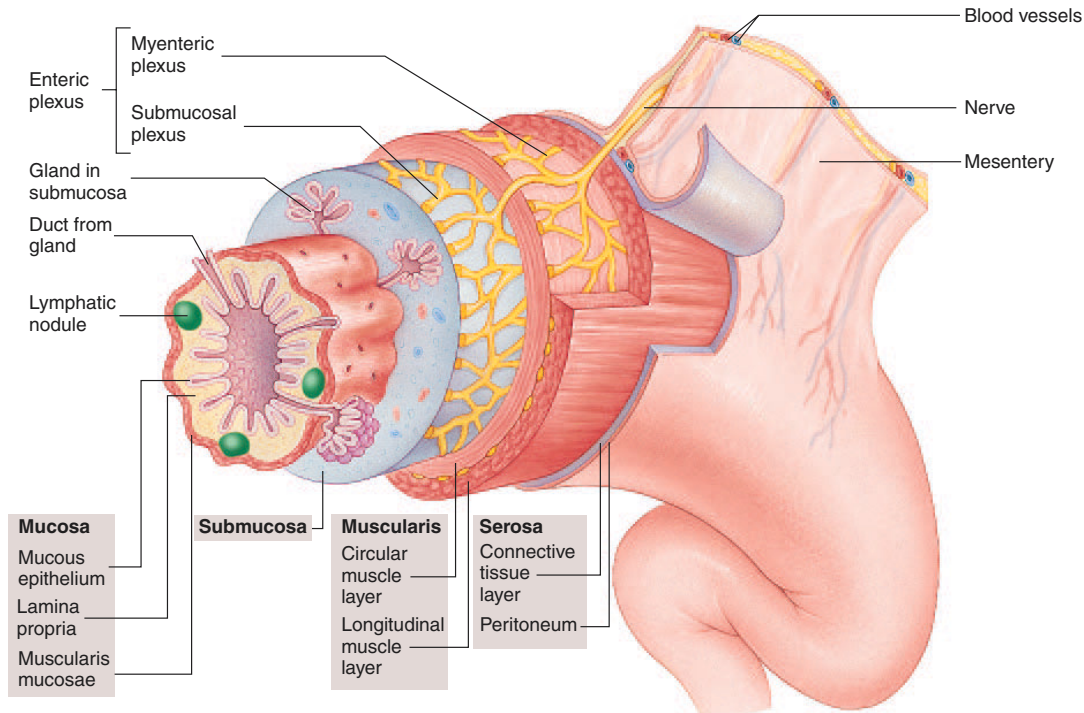


Figure 24.4 Digestive Tract Histology

The four tunics are the mucosa, submucosa, muscularis, and serosa or adventitia. Glands may exist along the digestive tract as part of the epithelium, within the submucosa, or as large glands that are outside the digestive tract.

(mī-en-ter'ik; Auerbach's plexus), which also consists of axons and many scattered neuron cell bodies, is between these two muscle layers (see figure 24.4).

Together, the submucosal and myenteric plexuses constitute the **enteric plexus** (en-tēr'ik; relating to the intestine) or **intramural** (in'trā-mū'rāl; within the walls) **plexus**. The enteric plexus is extremely important in the control of movement and secretion.

Serosa or Adventitia

The fourth layer of the digestive tract is a connective tissue layer called either the **serosa** or the **adventitia** (ad-ven-tish'ă; foreign or coming from outside), depending on the structure of the layer. Parts of the digestive tract that protrude into the peritoneal cavity have a serosa as the outermost layer. This serosa is called the visceral peritoneum. It consists of a thin layer of connective tissue and a simple squamous epithelium. 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 (discussed later in relation to the peritoneum, p. 864).

3. What are the major layers of the digestive tract? How do the serosa and adventitia differ?
4. Describe the enteric plexus. In what layers of the digestive tract are the submucosal and myenteric plexuses found?

Regulation of the Digestive System

Objective

- Outline the nervous and chemical mechanisms that regulate the digestive system.

Elaborate nervous and chemical mechanisms regulate the movement, secretion, absorption, and elimination processes.

Nervous Regulation of the Digestive System

Some of the nervous control is local, occurring as the result of local reflexes within the enteric plexus, and some is more general, mediated largely by the parasympathetic division of the ANS through the vagus nerve.

Local neuronal control of the digestive tract occurs within the **enteric nervous system (ENS)**. The ENS consists of the enteric plexus, made up of enteric neurons within the wall of the digestive tract (see figure 24.4). There are three major types of enteric neurons: (1) Enteric sensory neurons detect changes in the chemical composition of the 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. (3) Enteric interneurons connect enteric sensory and motor

neurons. The ENS coordinates peristalsis and regulates **local reflexes**, which control activities within specific, short regions of the digestive tract. Although the enteric neurons are capable of controlling the activities of the digestive tract independent of the CNS, normally the two systems work together. For example, autonomic innervation from the CNS influences the activity of the ENS neurons.

General control of the digestive system by the CNS occurs when reflexes are activated by stimuli originating in the digestive tract. Action potentials are carried by sensory neurons in the vagus nerves to the CNS, where the reflexes are integrated. In addition, reflexes within the CNS may be activated by the sight, smell, or taste of food, which stimulate the sensation of hunger. All of these reflexes influence parasympathetic neurons in 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

The digestive tract produces a number of hormones, such as gastrin, secretin, and others, which are secreted by endocrine cells of the digestive system and carried through the circulation to target organs of the digestive system or to target tissues in other systems. These hormones help regulate many gastrointestinal 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, which enter the circulation, other paracrine chemicals, such as histamine, are released locally within the digestive tract and 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.

5. What are the nervous and chemical mechanisms that regulate the digestive system?

Peritoneum

Objective

- Describe the serous membranes found in the abdominal cavity.

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

Peritonitis

Peritonitis is the inflammation of the peritoneal membranes. This inflammation may result from chemical irritation by substances such as bile that have escaped from a damaged digestive tract; or it may result from infection, again originating in the digestive tract, such as when the appendix ruptures. Peritonitis can be life-threatening. An accumulation of excess serous fluid in the peritoneal cavity is called **ascites** (ă-sī'tēz). Ascites may accompany peritonitis, starvation, alcoholism, or liver cancer.



Connective tissue sheets called **mesenteries** (mes'en-ter'ēz; middle intestine) hold many of the organs in place within the abdominal cavity. 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 body wall to the organs. Other abdominal organs lie against the abdominal wall, have no mesenteries, and are referred to as **retroperitoneal** (re'trō-per'i-tō-nē'āl; behind the peritoneum; see chapter 1). The retroperitoneal organs include the duodenum, the pancreas, the ascending colon, the descending colon, the rectum, the kidneys, the adrenal glands, and the urinary bladder.

Some mesenteries are given specific names. 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'tūm; 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** (see 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, or pocket, called the **omental bursa** (ber'să; pocket) is formed between the two layers of mesentery. A large amount of fat accumulates in the greater omentum, and it is sometimes referred to as the “fatty apron.” The greater omentum has considerable mobility in the abdomen.

P R E D I C T 1

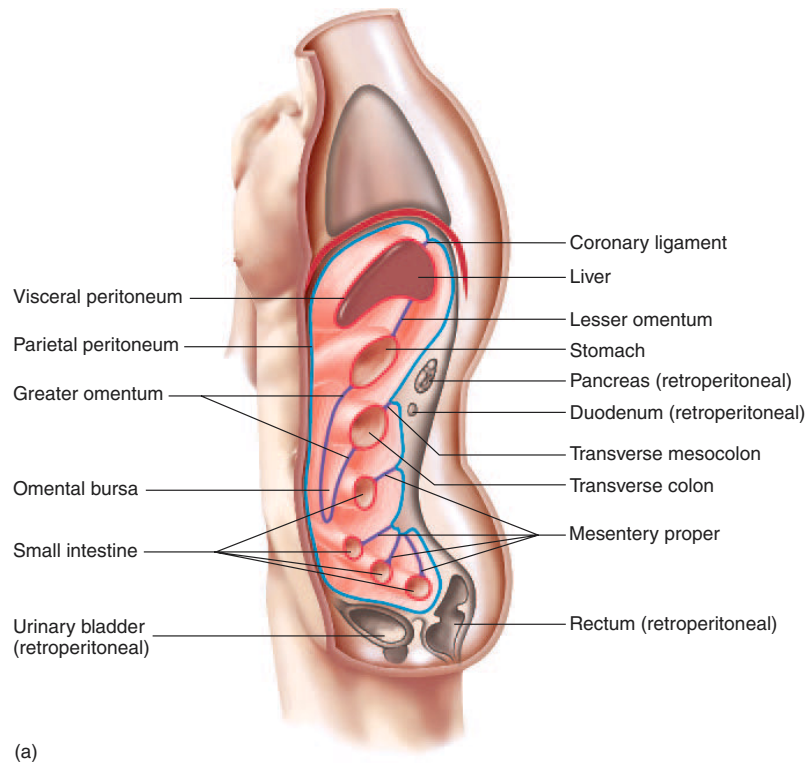
If you placed a pin through the greater omentum, through how many layers of simple squamous epithelium would the pin pass?

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 (see figure 24.5).

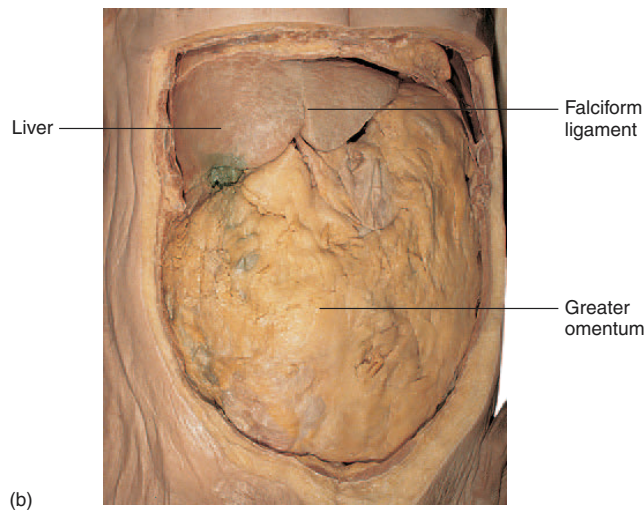
Although the term *mesentery* is a general term referring to the serous membranes attached to the abdominal organs, it is also used specifically to refer to the mesentery associated with the small intestine, sometimes called the **mesentery proper**. The mesenteries of parts of the colon are the **transverse mesocolon**, which extends from the transverse colon to the posterior body wall, and the **sigmoid mesocolon**. The vermiform appendix even has its own little mesentery called the **mesoappendix**.

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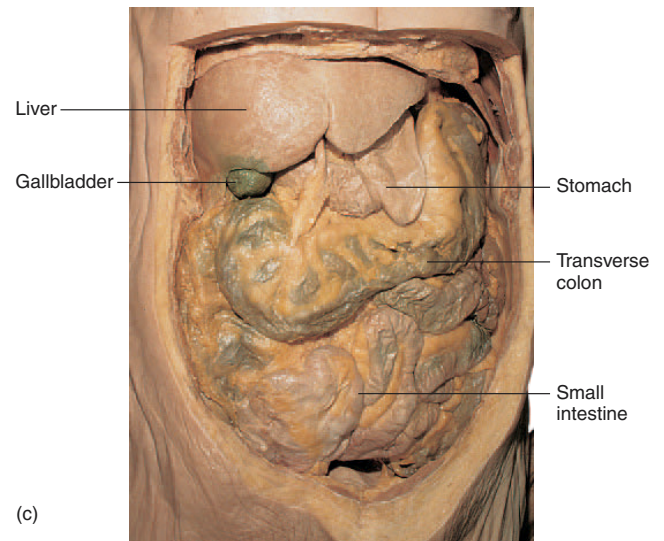
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(a)



(b)



(c)

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.

6. Where are visceral peritoneum and parietal peritoneum found? What is a retroperitoneal organ?
7. Define the term mesentery. Name and describe the location of the mesenteries found in the abdominal cavity.

Oral Cavity

Objective

- List and describe the major structures and secretions of the oral cavity.

The **oral cavity** (figure 24.6), or mouth, is that part of the digestive tract bounded by the lips anteriorly, the **fauces** (faw'sēz; throat; opening into the pharynx) posteriorly, the cheeks laterally, the palate superiorly, and a muscular floor inferiorly. The oral cavity is divided into two regions: (1) the **vestibule** (ves'ti-bool; entry), which is the space between the lips or cheeks and the alveolar processes, which contain the teeth; and (2) the **oral cavity proper**, which lies medial to the alveolar processes. The oral cavity is lined with moist stratified squamous epithelium, which provides protection against abrasion.

Lips and Cheeks

The **lips**, or **labia** (lā'bē-ă) (see figure 24.6), are muscular structures formed mostly by the **orbicularis oris** (ōr-bik'ū-lā'ris ōr'is) **muscle** (see figure 10.9a), as well as 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 can be seen 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 **frenula** (fren'ū-lā; bridle), which are mucosal folds, extend from the alveolar processes 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 the processes of mastication and speech. They help manipulate food within the mouth and hold it in place while the teeth crush or tear it. They also help form words during the speech process. A large number of the muscles of facial expression are involved in movement of the lips. They are listed in chapter 10.

Palate and Palatine Tonsils

The **palate** (see figure 24.6) consists of two parts, an anterior bony part, the **hard palate** (see chapter 7), and a posterior, non-bony part, the **soft palate**, which consists of skeletal muscle and

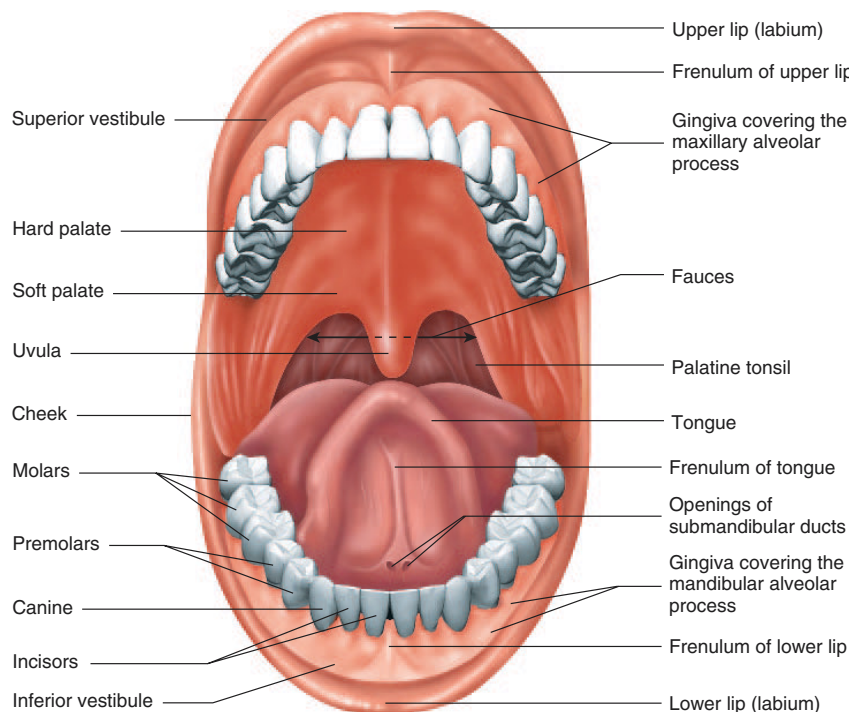


Figure 24.6 Oral Cavity

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connective tissue. The **uvula** (ū'vū-lă; a grape) is the projection from the posterior edge of the soft palate. The palate is important in the swallowing process; it prevents food from passing into the nasal cavity.

Palatine tonsils are located 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 and is attached to the floor of the mouth by a thin fold of tissue called the **frenulum**. The muscles associated with the tongue are divided into two categories: **intrinsic muscles**, which are within the tongue itself; and **extrinsic muscles**, which 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 the tongue 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).



Tongue-Tied

A person is “tongue-tied” in a more literal sense if the frenulum extends too far toward the tip of the tongue, thereby inhibiting normal movement of the tongue and interfering with normal speech. Surgically cutting the frenulum can correct the condition.

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. It has, instead, a few small glands and a large amount of lymphoid tissue, the **lingual tonsil** (see chapter 22). Moist stratified squamous epithelium covers the tongue.



Lipid-Soluble Drugs

Certain drugs that are lipid-soluble and can diffuse through the plasma membranes of the oral cavity can be quickly absorbed into the circulation. An example is nitroglycerin, which is a vasodilator used to treat cases of angina pectoris. The drug is placed under the tongue, where, in less than 1 minute, it dissolves and passes through the very thin oral mucosa into the lingual veins.

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 the mechanism of swallowing (discussed on p. 872). It is a major sensory organ for taste (see chapter 15) and one of the primary organs of speech.



Glossectomy and Speech

Patients who have undergone glossectomies (tongue removal) as a result of glossal carcinoma can compensate for loss of the tongue's function in speech, and they can learn to speak fairly well. These patients, however, have substantial difficulty chewing and swallowing food.

Teeth

Normal adults have 32 **teeth**, which are distributed in two **dental arches**. One is called the maxillary arch and the other is called 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 divided 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 24.7a). The third molars are called **wisdom teeth** because they usually appear in a person's late teens or early twenties, when the person is old enough to have acquired some wisdom.



Impacted Wisdom Teeth

In some 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 their surgical removal is often necessary.

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

Each tooth consists of a **crown** with one or more **cusps** (points), a **neck**, and a **root** (figure 24.8). The **clinical crown** is that part of the tooth exposed in the oral cavity. The **anatomical crown** is the entire enamel-covered part of the tooth. The center of the tooth is a **pulp cavity**, which is filled with blood vessels, nerves, and connective tissue called **pulp**. 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 a living, cellular, and 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 cellular, bonelike substance, called **cementum**, which helps anchor the tooth 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'tāl; around a tooth) **ligaments** secure the teeth in the alveoli.

The teeth play an important role in mastication and a role in speech.

8. Distinguish between the vestibule and the oral cavity proper.
9. What are the functions of the lips and cheeks? What muscle forms the substance of the cheek?
10. What are the hard and soft palate? Where is the uvula found?

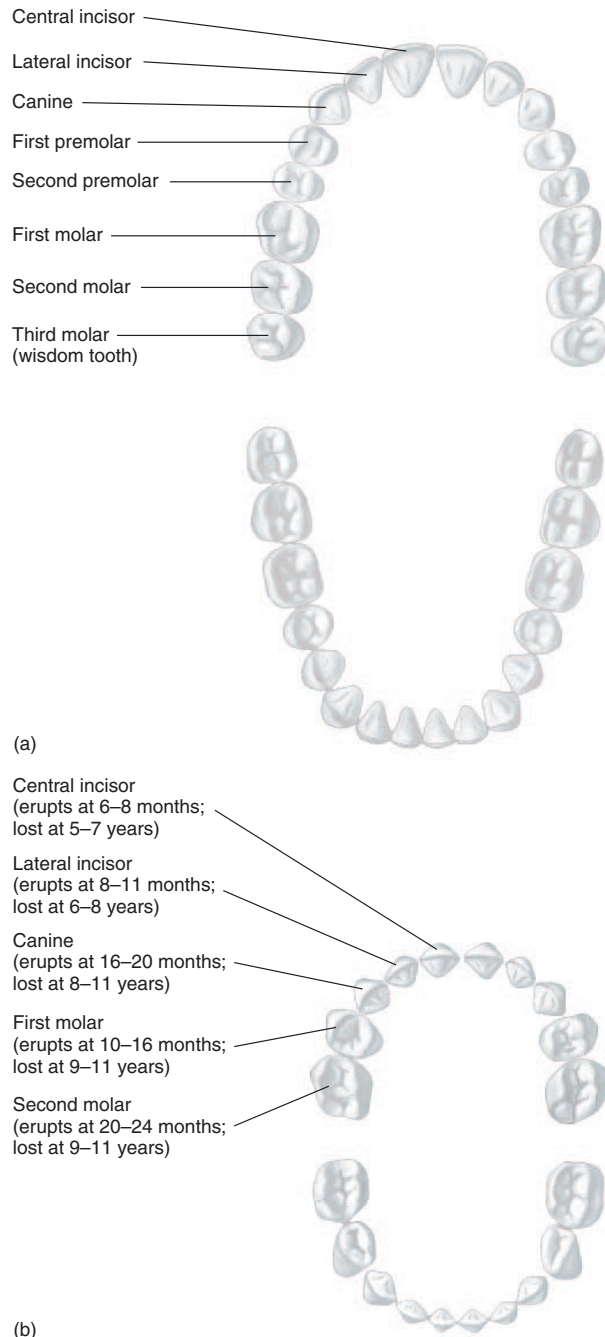


Figure 24.7 Teeth

(a) Permanent teeth. (b) Deciduous teeth.

11. List the functions of the tongue. Distinguish between intrinsic and extrinsic tongue muscles.
12. What are deciduous and permanent teeth? Name the different kinds of teeth.
13. Describe the parts of a tooth. What are dentin, enamel, cementum, and pulp?

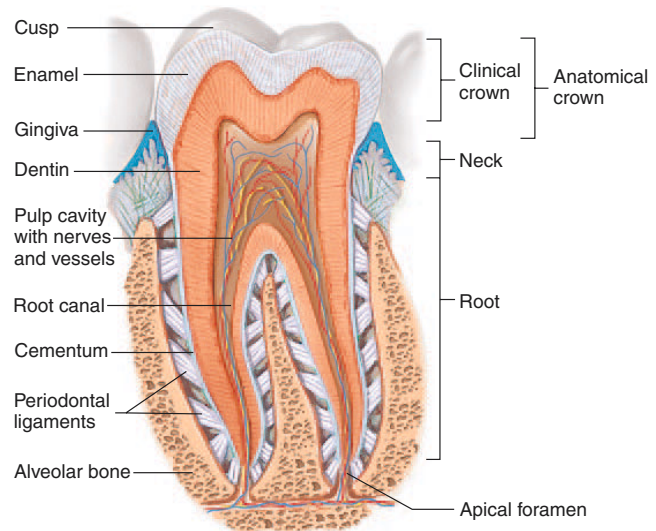


Figure 24.8 Molar Tooth in Place in the Alveolar Bone

The tooth consists of a crown and 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.

Dental Diseases



Dental caries, or tooth decay, is caused by a breakdown of enamel by acids produced by bacteria 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, toothache pain may result. In some cases in which decay has reached the pulp cavity, it may be necessary to perform a dental procedure called a “root canal,” which consists of removing the pulp 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-jī-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. **Pyorrhea** (pī-ō-rē’ă) is a condition in which pus occurs with periodontal disease. **Halitosis** (hal-i-tō’sis), or bad breath, often occurs with periodontal disease and pyorrhea.

Mastication

Food taken into the mouth is **chewed**, or **masticated**, 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, which have 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: the **temporalis**, **masseter**, **medial pterygoid**, and **lateral pterygoid** (see chapter 10 and figure 10.9). The temporalis, masseter, and medial pterygoid muscles close the jaw; and the lateral pterygoid muscle opens it. The medial and lateral pterygoids and the masseter muscles accomplish protraction and lateral and medial excursion of

Chapter 24 Digestive System

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the jaw. The temporalis retracts the jaw. All these movements are involved in tearing, crushing, and grinding food.

The **chewing**, or **mastication, reflex**, which is integrated in the medulla oblongata, controls the basic movements involved in chewing. The presence of food in the mouth stimulates sensory receptors, which activate a reflex that causes the muscles of mastication to relax. The muscles are stretched as the mandible is lowered, and stretch of the muscles activates a reflex that causes contraction of the muscles of mastication. Once the mouth is closed, the food again stimulates the muscles of mastication to relax, and the cycle is repeated. Descending pathways from the cerebrum strongly influence the activity of the mastication reflex so that chewing can be initiated or stopped consciously. 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. Three pairs of large multicellular glands exist: the parotid, the submandibular, and the sublingual glands (figure 24.9). In addition to these large consolidations of glandular tissue, numerous small, coiled tubular glands are located deep to the ep-

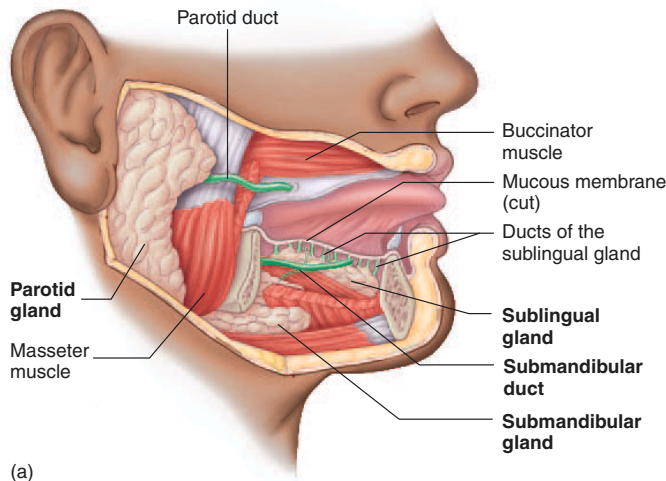
ithelium of the tongue (lingual glands), palate (palatine glands), cheeks (buccal glands), and lips (labial glands). The secretions from these glands help keep the oral cavity moist and begin the process of digestion.

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

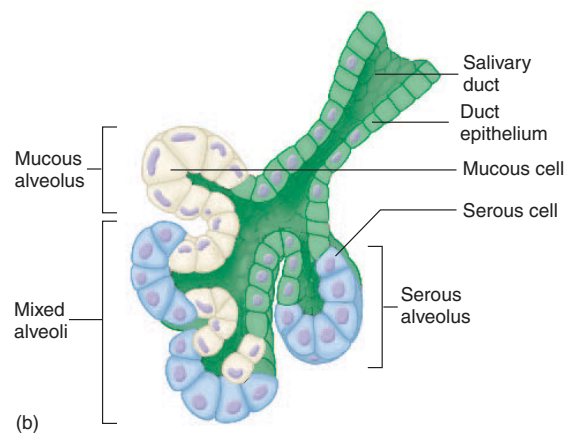
The largest salivary glands, the **parotid** (pă-rot'id; beside the ear) **glands**, are serous glands, which produce mostly watery saliva, and are located just anterior to the ear on each side of the head. Each **parotid duct** exits the gland on its anterior margin, crosses the lateral surface of the masseter muscle, pierces the buccinator muscle, and enters the oral cavity adjacent to the second upper molar (see figure 24.9).

Saliva and the Second Molar

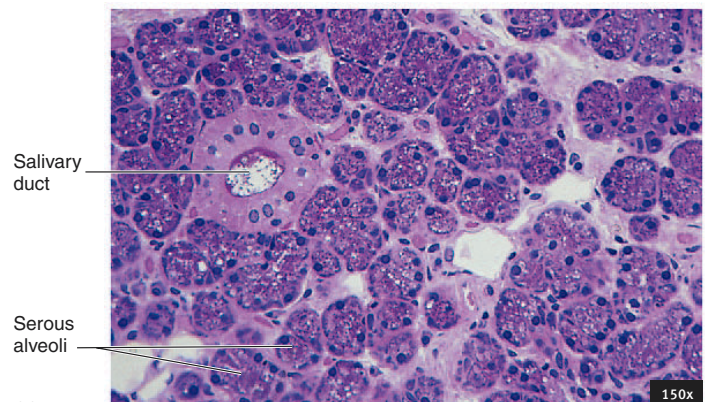
Because the parotid secretions are released directly onto the surface of the second upper molar, it tends to have a considerable accumulation of mineral, secreted from the gland, on its surface.



(a)



(b)



(c)

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 drawing of the histology of the large salivary glands. The figure is representative of all the glands and does not depict any one salivary gland. (c) Photomicrograph of the parotid gland.

Mumps

Inflammation of the parotid gland is called parotiditis. **Mumps**, which is caused by a virus, is the most common type of parotiditis.

The **submandibular** (below the mandible) **glands** are mixed glands with more serous than mucous alveoli. 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). In certain people, if the mouth is opened and the tip of the tongue is elevated, the submandibular ducts are compressed and saliva may squirt out of the mouth from the openings of these ducts.

The **sublingual** (below the tongue) **glands**, the smallest of the three large, paired salivary glands, are mixed glands containing some serous alveoli but consisting primarily of mucous alveoli. They lie immediately below the mucous membrane in the floor of the mouth. 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 secreted at the rate of about 1–1.5 L/day. The serous part of saliva, produced mainly by the parotid and submandibular glands, 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 (tables 24.2 and 24.4). The release of maltose and isomaltose gives starches a sweet taste in the mouth. Food spends very little time in the mouth, however; therefore, only about 3%–5% of the total carbohydrates are digested in the mouth. Most of the starches are covered by cellulose in plant tissues and are inaccessible to salivary amylase. Cooking and thorough chewing of food destroy the cellulose covering and increase the efficiency of the digestive process.

Saliva prevents bacterial infection in the mouth by washing the oral cavity. Saliva also contains substances, such as **lysozyme**, which has a weak antibacterial action, and immunoglobulin A, which helps prevent bacterial infection. Any lack of salivary gland secretion increases the chance of ulceration and infection of the oral mucosa and of caries in the teeth.

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.

Salivary gland secretion is stimulated by the parasympathetic and sympathetic nervous systems, with the parasympathetic system being 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 the activity of the salivary glands. Odors that trigger thoughts of food or the sensation of hunger can increase salivary secretions.

14. List the muscles of mastication and the actions they produce. Describe the mastication reflex.
15. Name and give the location of the three largest salivary glands. Name the other kinds of salivary glands.
16. What substances are contained in saliva?
17. What is the difference between serous and mucous saliva?

Pharynx

Objective

- Describe the anatomy of the pharynx and esophagus.

The **pharynx** was described in detail in chapter 23; thus, only a brief description is provided here. The pharynx consists of three parts: the nasopharynx, the oropharynx, and the laryngopharynx. Normally, only the oropharynx and laryngopharynx transmit food. The **oropharynx** communicates with the nasopharynx superiorly, the larynx and **laryngopharynx** inferiorly, and the mouth anteriorly. The laryngopharynx extends from the oropharynx to the esophagus and is posterior to 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.

18. Name the three parts of the pharynx. What are the pharyngeal constrictors?

P R E D I C T 2

Explain the functional significance of the differences in epithelial types among the three pharyngeal regions.

Esophagus

Objective

- Describe the esophagus, its layers and sphincters.

The **esophagus** is that part of the digestive tube 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.

Hiatal Hernia

A **hiatal hernia** is a widening of the esophageal hiatus. Hiatal hernias occur most commonly in adults and allow part of the stomach to extend through the opening into the thorax. The hernia can decrease the resting pressure in the lower esophageal sphincter, allowing gastroesophageal reflux and subsequent esophagitis to occur. Hiatal herniation can also compress the blood vessels in the stomach mucosa, which can lead to gastritis or ulcer formation. Esophagitis, gastritis, and ulceration can be very painful.

Table 24.2 Functions of Major Digestive Secretions

Fluid or Enzyme	Function
Saliva	
Serous (watery)	Moistens food and mucous membrane; lysozyme kills bacteria
Salivary amylase	Starch digestion (conversion to maltose and isomaltose)
Mucus	Lubricates food; protects gastrointestinal tract from digestion by enzymes
Esophagus	
Mucus	Lubricates the esophagus; protects the esophagus lining from abrasion and allows food to move more smoothly through the esophagus
Gastric Secretions	
Hydrochloric acid	Decreases stomach pH to activate pepsinogen
Pepsinogen	Pepsin, the active form of pepsinogen, digests protein into smaller peptide chains
Mucus	Protects stomach lining from digestion
Liver	
Bile	Bile salts emulsify fats, making them available to intestinal lipases; help make end products soluble and available for absorption by the intestinal mucosa; aid peristalsis. Many of the other bile contents are waste products transported to the intestine for disposal.
Sodium glycocholate (bile salt)	
Sodium taurocholate (bile salt)	
Cholesterol	
Biliverdin	
Bilirubin	
Mucus	
Fat	
Lecithin	
Cells and cell debris	
Pancreas	
Trypsin	Digests proteins (breaks polypeptide chains at arginine or lysine residues)
Chymotrypsin	Digests proteins (cleaves carboxyl links of hydrophobic amino acids)
Carboxypeptidase	Digests proteins (removes amino acids from the carboxyl end of peptide chains)
Pancreatic amylase	Digests carbohydrates (hydrolyzes starches and glycogen to form maltose and isomaltose)
Pancreatic lipase	Digests fat (hydrolyzes fats—mostly triacylglycerols—into glycerol and fatty acids)
Ribonuclease	Digests ribonucleic acid
Deoxyribonuclease	Digests deoxyribonucleic acid (hydrolyzes phosphodiester bonds)
Cholesterol esterase	Hydrolyzes cholesterol esters to form cholesterol and free fatty acids
Bicarbonate ions	Provides appropriate pH for pancreatic enzymes
Small Intestine Secretions	
Mucus	Protects duodenum from stomach acid, gastric enzymes, and intestinal enzymes; provides adhesion for fecal matter; protects intestinal wall from bacterial action and acid produced in the feces
Aminopeptidase	Splits polypeptides into amino acids (from amino end of chain)
Peptidase	Splits amino acids from polypeptides
Enterokinase	Activates trypsin from trypsinogen
Amylase	Digests carbohydrates
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
Lipase	Splits fats into glycerol and fatty acids

The esophagus has thick walls consisting of the four tunics common to the digestive tract: mucosa, submucosa, muscularis, and adventitia. The muscular tunic has an outer longitudinal layer and an inner circular layer, as is true of most parts of the digestive tract, but it's different because it consists of 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 that passes through ducts to the surface of the esophageal mucosa.

19. Where is the esophagus located? Describe the layers of the esophageal wall and the esophageal sphincters.

Swallowing

Objective

- Describe the process of swallowing.

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

The **pharyngeal phase** (figure 24.10b–d) of swallowing is a reflex that is initiated by 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.

P R E D I C T 3

Why is it important to close the opening between the nasopharynx and oropharynx during swallowing? What may happen if a person has an explosive burst of laughter while trying to swallow a liquid?

During the pharyngeal phase, the vestibular folds are moved medially, the **epiglottis** (ep-i-glōt'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 of the larynx prevent food from passing through the opening into the larynx.

P R E D I C T 4

What happens if you try to swallow and speak at the same time?

The **esophageal phase** (figure 24.10e) of swallowing takes about 5–8 seconds and is responsible for moving food from the pharynx to the stomach. Muscular contractions in the wall of the esophagus occur in peristaltic waves.

The peristaltic waves associated with swallowing cause relaxation of the lower esophageal sphincter in the esophagus as the peristaltic waves, and bolus of food, approach the stomach. 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 enteric plexus, which controls the peristaltic waves. The presence of 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 striated and smooth muscles within the esophagus, thereby stimulating their contractions and reinforcing the peristaltic contractions.

Swallowing and Gravity



Gravity assists the movement of material through the esophagus, especially when liquids are swallowed. The peristaltic contractions that move material through the esophagus are sufficiently forceful, however, to allow a person to swallow, even while doing a headstand or floating in the zero-gravity environment of space.

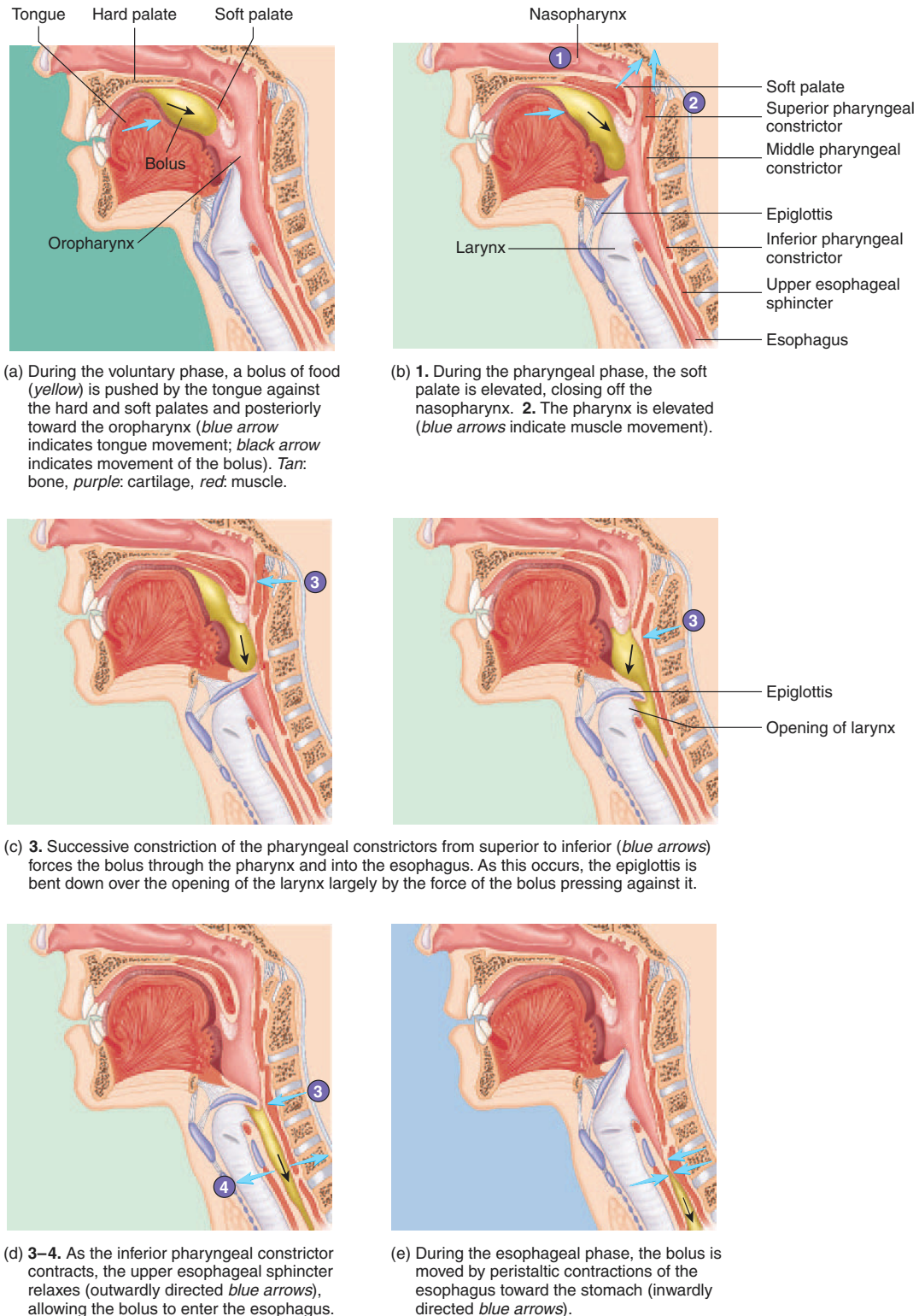
20. What are the three phases of deglutition? List sequentially the processes involved in the last two phases, and describe how they are regulated.

Stomach

Objectives

- List the anatomic and histologic characteristics of the stomach that are most important to its function.
- Describe the stomach secretions and their functions during the cephalic, gastric, and intestinal phases of stomach secretion regulation.
- Describe gastric filling, mixing, and emptying, and explain their regulation.

The **stomach** is an enlarged segment of the digestive tract 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 its size and shape change from time to time, depending on its food content and the posture of the body. Nonetheless, several general anatomic features can be described.



Process Figure 24.10 Three Phases of Swallowing (Deglutition)

Anatomy of the Stomach

The opening from the esophagus into the stomach is the **gastro-esophageal**, or **cardiac** (located near the heart), **opening**, and the region of the stomach around the cardiac opening is the **cardiac region** (figure 24.11). The lower esophageal sphincter, also called the **cardiac sphincter**, surrounds the cardiac opening. Recall that although this is an important structure in the normal function of the stomach, it is a physiologic constrictor only and cannot be seen anatomically. A part of the stomach to the left of the cardiac region, the **fundus** (fūn'dūs; the bottom of a round-bottomed leather bottle), is actually superior to the cardiac opening. The largest part of the stomach is the **body**, which turns to the right, thus creating a **greater curvature** and a **lesser curvature**. The body narrows to form the **pyloric** (pī-lōr'ik; gatekeeper) **region**, which joins the small intestine. The opening between the stomach and the small intestine is the **pyloric opening**, which is surrounded by a relatively thick ring of smooth muscle called the **pyloric sphincter**.

Hypertrophic Pyloric Stenosis

Hypertrophic pyloric stenosis is a common defect of the stomach in infants, occurring in 1 in 150 males and 1 in 750 females, in which the pylorus is greatly thickened, resulting in interference with normal stomach emptying. Infants with this defect exhibit projectile vomiting. Because the pylorus is blocked, little food enters the intestine, and the infant fails to gain weight. Constipation is also a frequent complication.

Histology of the Stomach

The **serosa**, or visceral peritoneum, is the outermost layer of the stomach. It consists of an inner layer of connective tissue and an outer layer of simple squamous epithelium. The **muscularis** of the stomach consists of three layers: an outer longitudinal layer, a middle circular layer, and an inner oblique layer (figure 24.11a). In some areas of the stomach, such as in the fundus, the three layers blend with one another and cannot be separated. Deep to the muscular layer 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 stomach is lined with simple columnar epithelium. The epithelium forms numerous tubelike **gastric pits**, which are the openings for the **gastric glands** (figure 24.11b). The epithelial cells of the stomach are of five types. The first type, **surface mucous cells**, which produce mucus, is on the surface and lines the gastric pit. The remaining four cell types are in the gastric glands. They are **mucous neck cells**, which produce mucus; **parietal** (oxyntic) **cells**, which produce hydrochloric acid and intrinsic factor; **chief** (zymogenic) **cells**, which produce pepsinogen; and **endocrine cells**, which produce regulatory hormones. The mucous neck cells are located near the openings of the glands; whereas the parietal, chief, and endocrine cells are interspersed in the deeper parts of the glands.

Secretions of the Stomach

Ingested food and stomach secretions, mixed together, form a semifluid material called **chyme** (kīm; juice). The stomach functions primarily as a storage and mixing chamber for the chyme. Although some digestion and absorption occur in the stomach, they are not its major functions.

Stomach secretions include mucus, hydrochloric acid, gastrin, histamine, intrinsic factor, and pepsinogen. Pepsinogen is the inactive form of the protein-digesting enzyme pepsin.

The surface mucous cells and mucous neck cells secrete a viscous and alkaline **mucus** that covers the surface of the epithelial cells and forms 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 results in stimulation of the secretion of a greater volume of mucus.

Parietal cells in the gastric glands of the pyloric region secrete intrinsic factor and a concentrated solution of hydrochloric acid. **Intrinsic factor** is a glycoprotein that binds with vitamin B₁₂ and makes the vitamin more readily absorbed in the ileum. Vitamin B₁₂ is important in deoxyribonucleic acid (DNA) synthesis.

Hydrochloric acid produces the low pH of the stomach, which is normally between 1 and 3. Although the hydrochloric acid secreted into the stomach has a minor digestive effect on ingested food, one of its main functions is to kill bacteria that are ingested with essentially everything humans put into their mouths. Some pathogenic bacteria may avoid digestion in the stomach, however, because they have an outer coat that resists stomach acids.

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

Hydrogen ions are derived from carbon dioxide and water, which enter the parietal cell from its serosal surface, which is the side opposite the lumen of the gastric pit (figure 24.12). Once inside the cell, carbonic anhydrase catalyzes the reaction between carbon dioxide and water to form carbonic acid. Some of the carbonic acid molecules then dissociate to form hydrogen ions and bicarbonate ions. The hydrogen ions are actively transported across the mucosal surface of the parietal cell into the lumen of the stomach; some potassium ions are moved into the cell in exchange for the hydrogen ions. Although hydrogen ions are actively transported against a steep concentration gradient, chloride ions diffuse with the hydrogen ions from the cell through the plasma membrane. Diffusion of chloride ions with the positively charged hydrogen ions reduces the amount of energy needed to transport the hydrogen ions against both a concentration gradient and an electrical gradient. Bicarbonate ions move down their concentration gradient from the parietal cell into the extracellular fluid. During this process, bicarbonate ions are exchanged for chloride ions through an anion exchange molecule, which is located in the plasma membrane, and the chloride ions subsequently move into the cell.

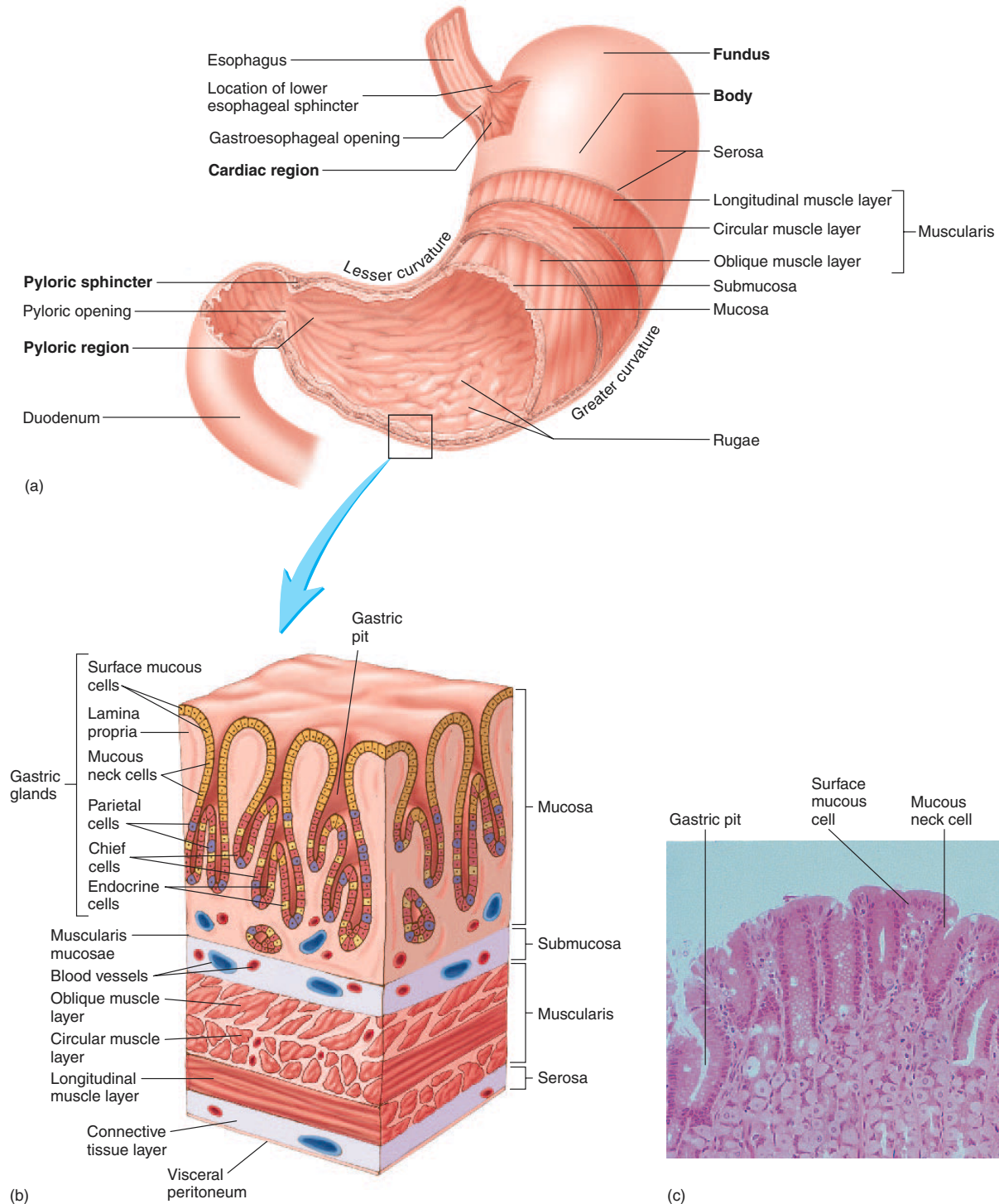
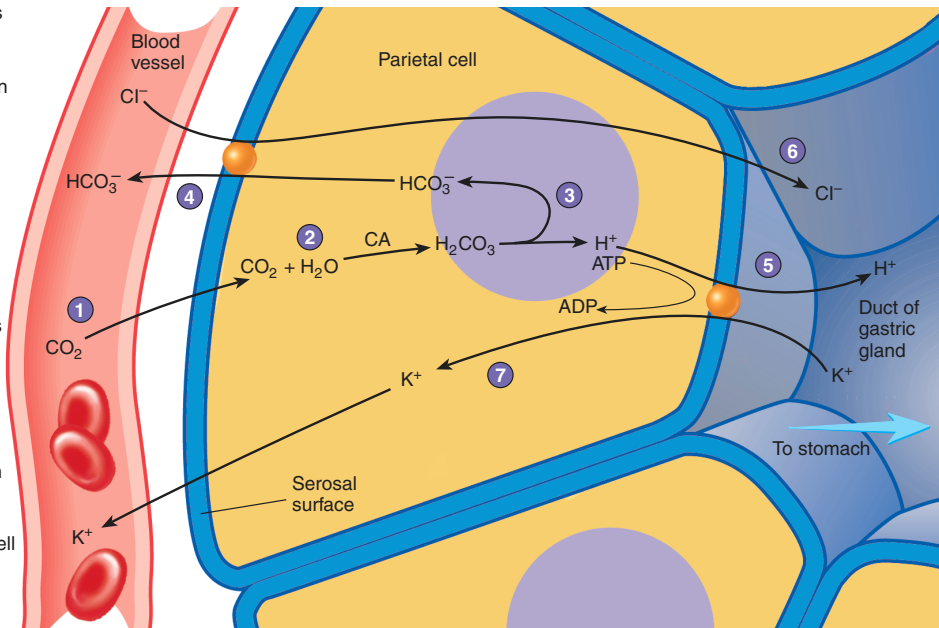


Figure 24.11 Anatomy and Histology of the Stomach

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

1. Carbon dioxide (CO_2) diffuses into the cell.
2. CO_2 is combined with water (H_2O) in an enzymatic reaction that is catalyzed by carbonic anhydrase (CA) to form carbonic acid (H_2CO_3).
3. Carbonic acid dissociates into a bicarbonate ion (HCO_3^-) and a hydrogen ion (H^+).
4. HCO_3^- is transported back into the bloodstream. An anion exchange molecule in the plasma membrane exchanges HCO_3^- for a chloride ion (Cl^-) (counter transport).
5. The hydrogen ion (H^+) is actively transported into the duct of the gastric gland.
6. Chloride ions (Cl^-) diffuse with the charged hydrogen ions.
7. Some potassium ions (K^+) are counter transported into the cell in exchange for the hydrogen ions.



Process Figure 24.12 Hydrochloric Acid Production by Parietal Cells in the Gastric Glands of the Stomach

PREDICT 5

Explain why a slight increase in the blood pH may occur following a heavy meal. The elevated pH of blood, especially in the veins that carry blood away from the stomach, is called “the postenteric alkaline tide.”

Chief cells within the gastric glands secrete **pepsinogen** (pep-sin’ō-jen). Pepsinogen is packaged in **zymogen** (zī-mō-jen; related to enzymes) **granules**, which are released by exocytosis when pepsinogen secretion is stimulated. Once pepsinogen enters the lumen of the stomach, hydrochloric acid and previously formed pepsin molecules convert it to **pepsin**. Pepsin exhibits optimum 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.

Heartburn

Heartburn, or **pyrosis** (pī-rō’sis), is a painful or burning sensation in the chest usually associated with reflux of acidic chyme into the esophagus. The pain is usually short-lived but may be confused with the pain of an ulcer or a heart attack. Overeating, eating fatty foods, lying down immediately after a meal, consuming too much alcohol or caffeine, smoking, or wearing extremely tight clothing can all cause heartburn. A hiatal hernia can also cause heartburn, especially in older people.

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 dramatically affects the secretion amount, but up to 700 mL is 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 enteric plexus of the GI tract. In

addition, higher brain centers influence the reflexes. Chemical signals that regulate stomach secretions include the hormones gastrin, secretin, gastric-inhibitory polypeptide, and cholecystokinin, as well as the paracrine chemical signal histamine (table 24.3).

Regulation of stomach secretion is divided into three phases: cephalic, gastric, and intestinal.

1. **Cephalic phase.** In the cephalic phase of gastric regulation, the sensations of the taste and smell of food, stimulation of tactile receptors during the process of chewing and swallowing, and pleasant thoughts of food stimulate centers within the medulla oblongata that influence gastric secretions (figure 24.13a). Action potentials are sent from the medulla along parasympathetic neurons within the vagus (X) nerves to the stomach. Within the stomach wall, the preganglionic neurons stimulate postganglionic neurons in the enteric plexus. 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 chief cells and stimulates the secretion of **gastrin** (gas’trin) and histamine from endocrine cells. Gastrin is released into the circulation and travels to the parietal cells, where it stimulates additional hydrochloric acid and pepsinogen secretion. In addition, gastrin stimulates endocrine cells to release histamine, which stimulates parietal cells to secrete hydrochloric acid. The histamine receptors on the parietal cells are called H_2 receptors, and 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. Acetylcholine, histamine, and

Table 24.3 Functions of the Gastrointestinal Hormones

Site of Production	Method of Stimulation	Secretory Effects	Motility Effects
Gastrin Stomach and duodenum	Distention; partially digested proteins, autonomic stimulation, ingestion of alcohol or caffeine	Increases gastric secretion	Increases gastric emptying by increasing stomach motility and relaxing the pyloric sphincter
Secretin Duodenum	Acidity of chyme	Inhibits gastric secretion; stimulates pancreatic secretions high in bicarbonate ions; increases the rate of bile and increases intestinal secretion; mucus secretion	Decreases gastric motility
Cholecystokinin Intestine	Fatty acids and other lipids	Slightly inhibits gastric secretion; stimulates pancreatic secretions high in digestive enzymes; and causes contraction of the gallbladder and relaxation of the hepatopancreatic ampullar sphincter	Decreases gastric motility
Gastric Inhibitory Polypeptide Duodenum and proximal jejunum	Fatty acids and other lipids	Inhibits gastric secretions	Decreases gastric motility

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.

Inhibitors of Gastric Acid Secretion

Cimetidine (Tagamet) and ranitidine (Zantac) are synthetic analogs of histamine that can bind to H₂ histamine receptors on parietal cells, and prevent histamine binding, without stimulating the cell. These chemicals are called histamine blockers and are extremely effective inhibitors of gastric acid secretion. Cimetidine, one of the most commonly prescribed drugs, is used to treat cases of gastric acid hypersecretion associated with gastritis and gastric ulcers.



- Gastric phase.** The greatest volume of gastric secretions is produced during the gastric phase of gastric regulation. The presence of food in the stomach initiates the gastric phase (figure 24.13*b*). The primary stimuli are distention of the stomach and the presence of amino acids and peptides in the stomach.

Distention of the stomach wall, especially in the body or fundus, results in the stimulation of mechanoreceptors. Action potentials generated by these receptors initiate reflexes that involve both the CNS and enteric reflexes, resulting in secretion of mucus, hydrochloric acid, pepsinogen, intrinsic factor, and gastrin. 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 distention of the stomach is blocked. This negative-feedback mechanism limits the secretion of gastric juice.

Amino acids and peptides released by the digestive action of pepsin on proteins directly stimulate parietal cells of the stomach to secrete hydrochloric acid. The mechanism by which this response is mediated is not clearly understood. It doesn't involve known neurotransmitters, and, when the pH drops below 2, the response is inhibited. Histamine also stimulates the secretory activity of parietal cells.

- Intestinal phase.** The entrance of acidic stomach contents into the duodenum of the small intestine controls the intestinal phase of gastric regulation (figure 24.13*c*). The presence of chyme in the duodenum activates both neural and hormonal mechanisms. When the pH of the chyme entering the duodenum drops to 2 or below, or if the chyme contains fat digestion products, gastric secretions are inhibited.

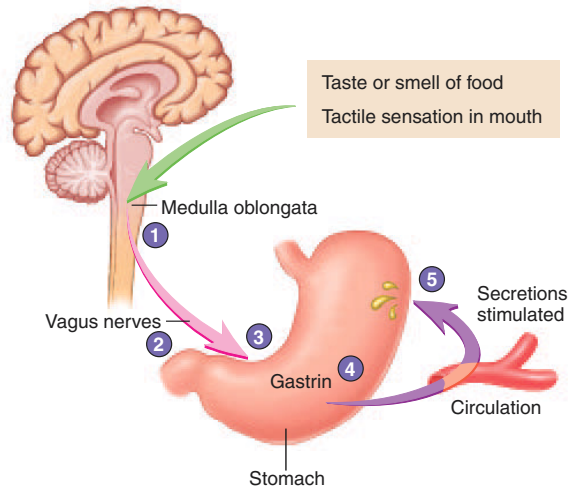
Acidic solutions in the duodenum cause the release of the hormone **secretin** (se-krē'tin) into the circulatory system. Secretin inhibits gastric secretion by inhibiting both parietal and chief cells. Acidic solutions also initiate a local enteric reflex, which inhibits gastric secretions.

Fatty acids and certain other lipids in the duodenum and the proximal jejunum initiate the release of two hormones: **gastric inhibitory polypeptide** and **cholecystokinin** (kō'lē-sis-tō-kī'nin). Gastric inhibitory polypeptide strongly inhibits gastric secretion, and cholecystokinin inhibits gastric secretions to a lesser degree. Hypertonic solutions in the duodenum and jejunum also inhibit gastric secretions. The mechanism appears to involve the secretion of a hormone referred to as **enterogastrone** (en'ter-ō-gas'trōn), but the actual existence of this hormone has never been established.

Cephalic Phase

1. The taste or smell of food, tactile sensations of food in the mouth, or even thoughts of food stimulate the medulla oblongata (*green arrow*).
2. Parasympathetic action potentials are carried by the vagus nerves to the stomach (*pink arrow*).
3. Preganglionic parasympathetic vagus nerve fibers stimulate postganglionic neurons in the enteric plexus of the stomach.
4. Postganglionic neurons stimulate secretion by parietal and chief cells and stimulate gastrin secretion by endocrine cells.
5. Gastrin is carried through the circulation back to the stomach (*purple arrow*), where it stimulates secretion by parietal and chief cells.

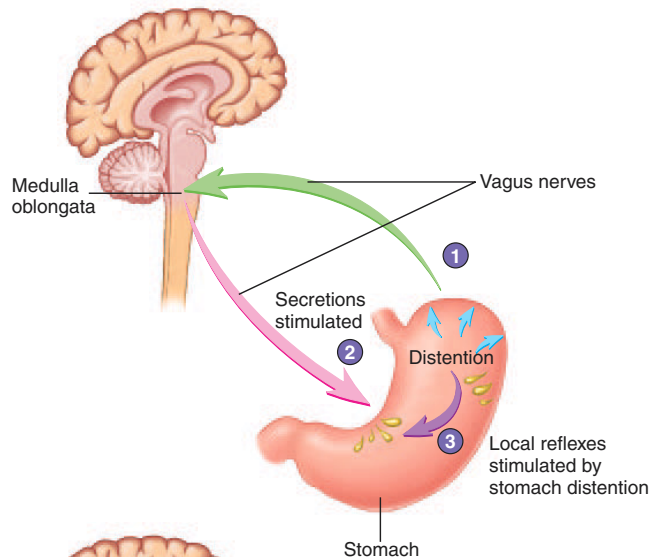
(a)



Gastric Phase

1. Distention of the stomach activates a parasympathetic reflex. Action potentials are carried by the vagus nerves to the medulla oblongata (*green arrow*).
2. The medulla oblongata stimulates stomach secretions (*pink arrow*).
3. Distention of the stomach also activates local reflexes that increase stomach secretions (*purple arrow*).

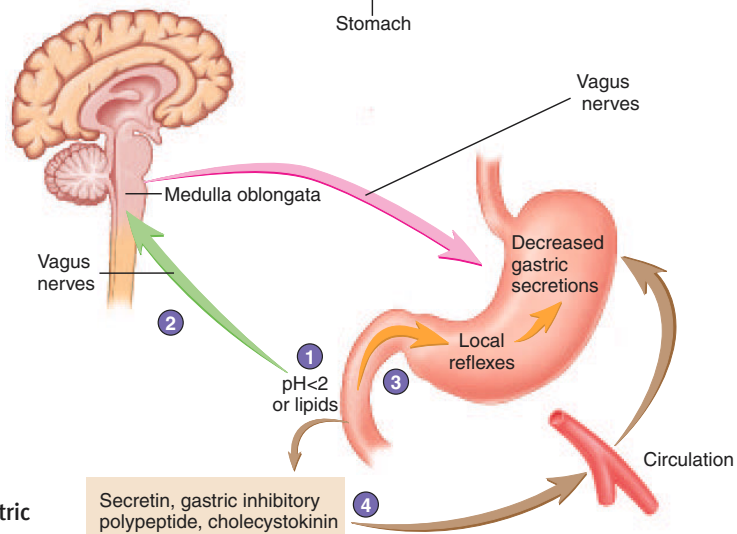
(b)



Intestinal Phase

1. Chyme in the duodenum with a pH less than 2 or containing fat digestion products (lipids) inhibits gastric secretions by three mechanisms (2–4).
2. Sensory vagal action potentials to the medulla oblongata (*green arrow*) inhibit motor action potentials from the medulla oblongata (*pink arrow*).
3. Local reflexes inhibit gastric secretion (*orange arrows*).
4. Secretin, gastric inhibitory polypeptide, and cholecystokinin produced by the duodenum (*brown arrows*) inhibit gastric secretions in the stomach.

(c)



Process Figure 24.13 The Three Phases of Gastric Secretion

(a) Cephalic phase. (b) Gastric phase. (c) Intestinal phase.

Clinical Focus Peptic Ulcer

Approximately 10% of the U.S. population will develop peptic ulcers during their life-time. Most cases of peptic ulcer are apparently due to the infection of a specific bacterium, *Helicobacter pylori*. It's also thought that the bacterium is involved in many cases of gastritis and gastric cancer. Conventional wisdom has focused for years on the notion that stress, diet, smoking, or alcohol cause excess acid secretion in the stomach, resulting in ulcers.

Antacids remain very popular in treating ulcers, as well as for the relief of temporary stomach problems. Close to \$1 billion is spent on antacids in the United States annually. Antacid therapy does relieve the ulcer in most cases. A 50% incidence of relapse occurs within 6 months with antacid treatment, and a 95% incidence of relapse occurs after 2 years. On the other hand, studies using antibiotic therapy in addition to bismuth and ranitidine have demonstrated a 95% eradication of gastric ulcers and 74% healing of duodenal ulcers within 2 months. Dramatically reduced relapse rates have also been obtained. One such study reported a recurrence rate of 8% following antibiotic therapy, compared with a recurrence rate of 86% in controls.

Other treatments include H_2 receptor antagonists, which bind histamine receptors and prevent histamine-stimulated HCl secretion. Proton pump inhibitors directly inhibit HCl secretion. Prostaglandins are naturally produced by the mucosa of the GI tract and help the mucosa resist injury. Synthetic prostaglandins can supplement this resistance as well as inhibit HCl secretion.

the infection rate from *h. Pylori* in the united states population is about 1% per

year of age: 30% of people that are 30 years old have the bacterium, and 80% of those age 80 are infected. In Third World countries, as many as 100% of people age 25 or older are infected. This may relate to the high rates of stomach cancer in some of those countries. We still have much to learn before we can understand this bacterium. Very little is known concerning how people become infected. Also, with such high rates of infection, it's not known why only a small fraction of those infected actually develop ulcers. It may be that factors such as diet and stress predispose a person who is infected by the bacterium to actually develop an ulcer.

Peptic ulcer is classically viewed as a condition in which the stomach acids and pepsin digest the mucosal lining of the GI tract itself. The most common site of a peptic ulcer is near the pylorus, usually on the duodenal side (i.e., a duodenal ulcer; 80% of peptic ulcers are duodenal). Ulcers occur less frequently along the lesser curvature of the stomach or at the point at which the esophagus enters the stomach. The most common presumed cause of peptic ulcers is the oversecretion of gastric juice relative to the degree of mucous and alkaline protection of the small intestine. One reason that bacterial involvement in ulcers was dismissed for such a long time is that it was assumed that the extreme acid environment killed all bacteria. Apparently not only can *H. pylori* survive in such an environment, but it may even thrive there.

People experiencing severe anxiety for a long time are the most prone to develop duodenal ulcers. They often have a high rate of gastric secretion (as much as 15 times the normal amount) between meals.

This secretion results in highly acidic chyme entering the duodenum. The duodenum is usually protected by sodium bicarbonate (secreted mainly by the pancreas), which neutralizes the chyme. When large amounts of acid enter the duodenum, however, the sodium bicarbonate is not adequate to neutralize it. The acid tends to reduce the mucous protection of the duodenum, perhaps leaving that part of the digestive tract open to the action of *H. pylori*, which may further destroy the mucous lining.

In one study, it was determined that ulcer patients prefer their hot drinks extra hot, 62°C compared with 56°C for a control group without ulcers. The high temperatures of the drinks may cause thinning of the mucous lining of the stomach, thus making these people more susceptible to ulcers, again perhaps by increasing their sensitivity to *H. pylori* invasion.

In some patients with gastric ulcers, often normal or even low levels of gastric hydrochloric acid secretion exist. The stomach has a reduced resistance to its own acid, however. Such inhibited resistance can result from excessive ingestion of alcohol or aspirin.

Reflux of duodenal contents into the pylorus can also cause gastric ulcers. In this case, bile, which is present in the reflux, has a detergent effect that reduces gastric mucosal resistance to acid and bacteria.

An ulcer may become perforated (a hole in the stomach or duodenum), causing peritonitis. The perforation must be corrected surgically. Selective vagotomy, cutting branches of the vagus (X) nerve going to the stomach, is sometimes performed at the time of surgery to reduce acid production in the stomach.

Inhibition of gastric secretions is also under nervous control. Distention 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. The **enterogastric reflex** consists of a local reflex and a reflex integrated within the medulla oblongata. It reduces gastric secretions.

Movements of the Stomach

Stomach Filling

As food enters the stomach, the rugae flatten, and the stomach volume increases. Despite the increase in volume, the pressure within the stomach doesn't increase until the volume nears maximum capacity because smooth muscle can stretch without an increase in tension (see chapter 9) and because of a reflex integrated within

the medulla oblongata. This reflex inhibits muscle tone in the body of the stomach.

Mixing of Stomach Contents

Ingested food is thoroughly mixed with the secretions of the stomach glands to form chyme. This mixing is accomplished by gentle **mixing waves**, which are peristaltic-like contractions that occur about every 20 seconds and proceed from the body toward the pyloric sphincter to mix the ingested material with the secretions of the stomach. **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 region for further digestion (figure

24.14). Roughly 80% of the contractions are mixing waves, and 20% are peristaltic waves.

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 exit the stomach within $1\frac{1}{2}$ – $2\frac{1}{2}$ hours after ingestion. After a typical meal, the stomach is usually empty within 3–4 hours. The pyloric sphincter usually remains partially closed because of mild tonic contraction. Each peristaltic contraction is sufficiently strong to force a small amount of chyme through the pyloric opening and into the duodenum. The peristaltic contractions responsible for movement of chyme through the partially closed pyloric opening are called the **pyloric pump**.

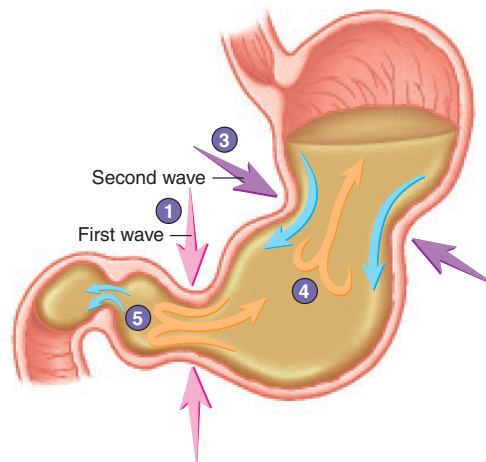
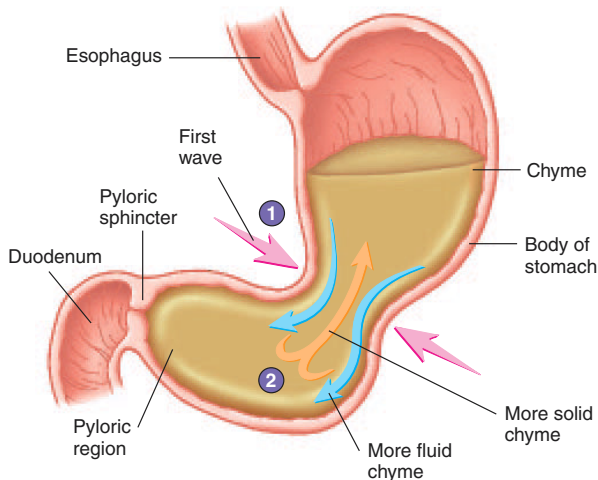
1. Mixing waves initiated in the body of the stomach progress toward the pyloric region (pink arrows directed inward).

2. 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).

3. Additional mixing waves (purple arrows) move in the same direction and in the same way as the earlier waves (1) that reach the pyloric region.

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 toward the body of the stomach (orange arrow).

5. Some of the most fluid chyme is squeezed through the pyloric opening into the duodenum (small blue arrows), whereas most of the chyme is forced back toward the body of the stomach for further mixing (orange arrows).



Process Figure 24.14 Movements in the Stomach

Hunger Contractions

Hunger contractions are peristaltic contractions that approach tetanic contractions for periods of about 2–3 minutes. Low blood glucose levels cause the contractions to increase and become sufficiently strong to create uncomfortable sensations called “hunger pangs.” Hunger pangs usually begin 12–24 hours after the previous meal or in less time for some people. If nothing is ingested, they 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. If the rate of emptying is too slow, the highly acidic contents of the stomach may damage the stomach wall and reduce the rate at which nutrients are digested and absorbed. Stomach emptying is regulated to prevent these two extremes. Some of the hormonal and neural mechanisms that stimulate stomach secretions also are involved with increasing stomach motility. For example, during the gastric phase of stomach secretion, distention of the stomach stimulates local reflexes, CNS reflexes, and the release of gastrin, all of which increase stomach motility and cause relaxation of the pyloric sphincter. The result is an increase in stomach emptying. Conversely, some of the hormonal and neural mechanisms that decrease gastric secretions also inhibit gastric motility, increase constriction of the pyloric sphincter, and decrease the rate of stomach emptying.

Vomiting

Vomiting can result from irritation (e.g., overdistention or overexcitation) anywhere along the GI 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 posterior nares; (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.

21. Describe the parts of the stomach. List the layers of the stomach wall. How is the stomach different from the esophagus?
22. What are gastric pits and gastric glands? Name the different cell types in the stomach and the secretions they produce.
23. Describe the three phases of regulation of stomach secretion, and discuss the cause and result of each phase.
24. How are gastric secretions inhibited? Why is this inhibition necessary?
25. Why does pressure in the stomach not greatly increase as the stomach fills?
26. What are two kinds of stomach movements? How are stomach movements regulated by hormones and nervous control?

Small Intestine

Objectives

- Describe the anatomy of the small intestine.
- List the secretions of the small intestine, and explain how secretion and movement are regulated.

The **small intestine** consists of three parts: the duodenum, the jejunum, and the 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 (the term *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; and 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 the site at which the greatest amount of digestion and absorption occur. Each day, about 9 L of water enters the digestive system. It comes from water that is ingested and from fluid secretions produced by glands along the length of the digestive tract. Most of the water, 8–8.5 L, moves by osmosis, with the absorbed solutes, out of the small intestine. A small part, 0.5–1 L, enters the colon.

Anatomy of the Small Intestine

Duodenum

The **duodenum** nearly completes a 180-degree arc as it curves within the abdominal cavity (figure 24.16), and the head of the pancreas lies within this arc. The duodenum begins with a short superior part, which is where it exits the pylorus of the stomach, and ends in a sharp bend, which is where it joins the jejunum.

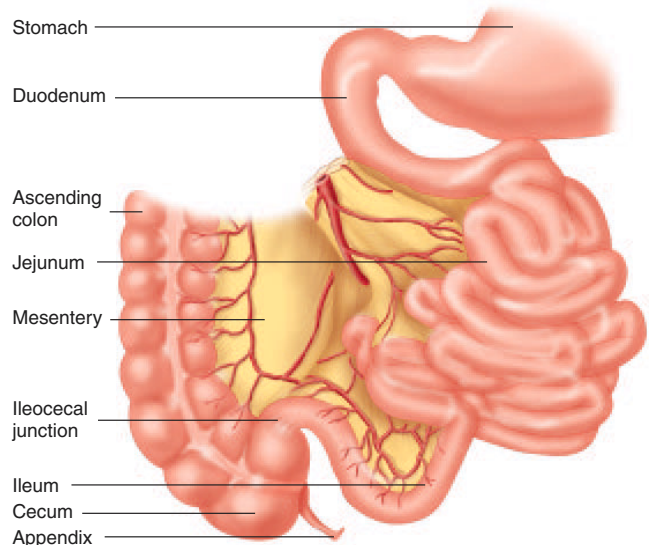


Figure 24.15 The Small Intestine

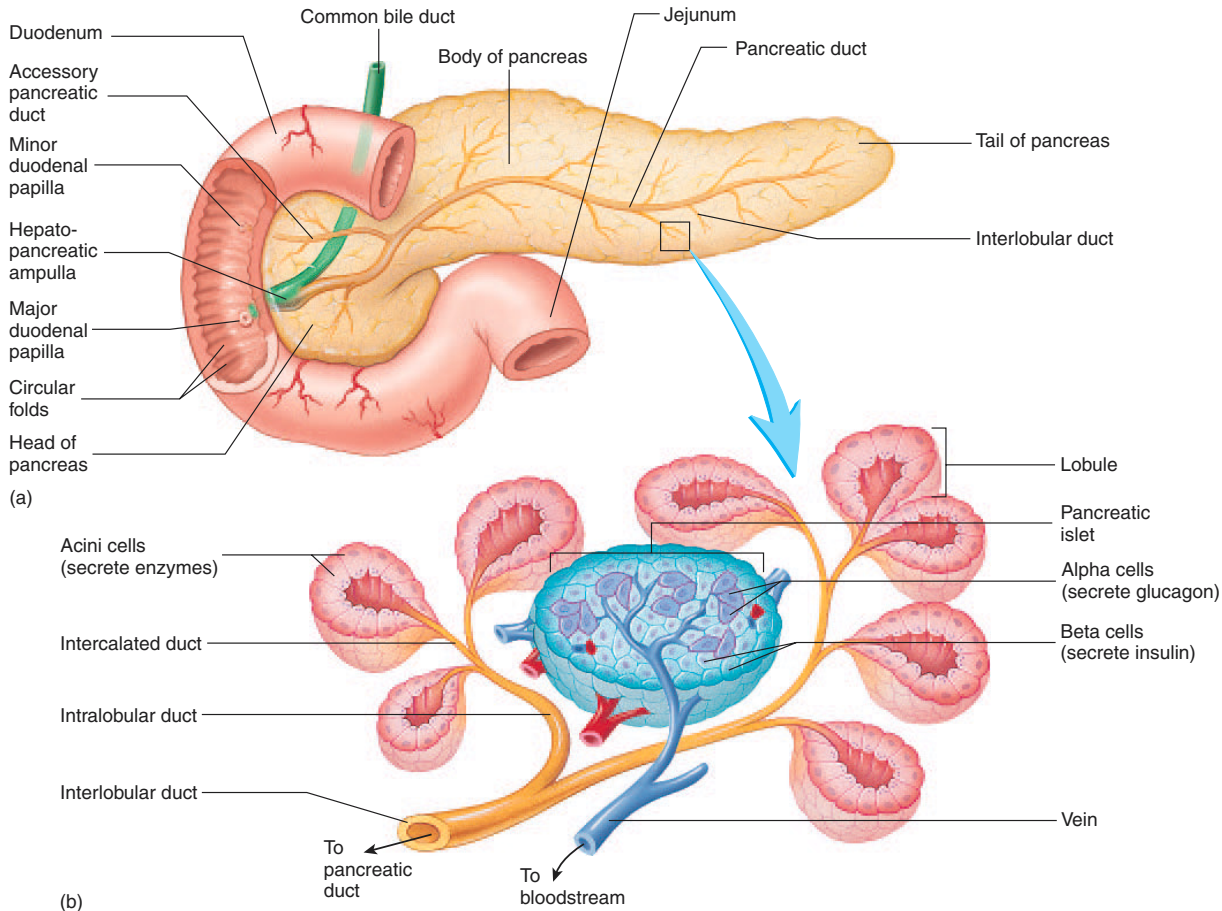


Figure 24.16 Anatomy and Histology of the Duodenum and Pancreas

(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 acini and the pancreatic duct system.

Two small mounds are within the duodenum about two-thirds of the way down the descending part: the **major duodenal papilla** and the **lesser duodenal papilla**. At the major papilla, the **common bile duct** and **pancreatic duct** join to form the **hepatopancreatic ampulla** (Vater's ampulla), which empties into the duodenum. A smooth muscle sphincter, the **hepatopancreatic ampullar sphincter** (sphincter of Oddi) regulates the opening of the ampulla. An accessory pancreatic duct, present in most people, opens at the tip of the lesser duodenal papilla.

The surface of the duodenum has several modifications that increase its surface area about 600-fold to allow for more efficient digestion and absorption of food. The mucosa and submucosa form a series of folds called the **circular folds**, or **plicae** (pli'sē; folds) **circulares** (figure 24.17a), which run perpendicular to the long axis of the digestive tract. Tiny fingerlike projections of the mucosa form numerous **villi** (vil'i; shaggy hair), which are 0.5–1.5 mm in length (figure 24.17b). Each villus is covered by simple columnar epithelium and contains a blood capillary network and a lymphatic capillary called a **lacteal** (lak'tē-āl) (figure 24.17c). Most of the cells that make up the surface of the villi have numerous cy-

toplasmic extensions (about 1 μ m long) called **microvilli**, which further increase the surface area (figure 24.17d). The combined microvilli on the entire epithelial surface form the **brush border**. These various modifications greatly increase the surface area of the small intestine and, as a result, greatly enhance absorption.

The mucosa of the duodenum is simple columnar epithelium with four major cell types: (1) **absorptive cells** are cells with microvilli, which produce digestive enzymes and absorb digested food; (2) **goblet cells**, which produce a protective mucus; (3) **granular cells** (Paneth's cells), which may help protect the intestinal epithelium from bacteria; and (4) **endocrine cells**, which produce regulatory hormones. The epithelial cells are produced within tubular invaginations of the mucosa, called **intestinal glands** (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 eventually are 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** (Brunner's glands), which open into the base of the intestinal glands.

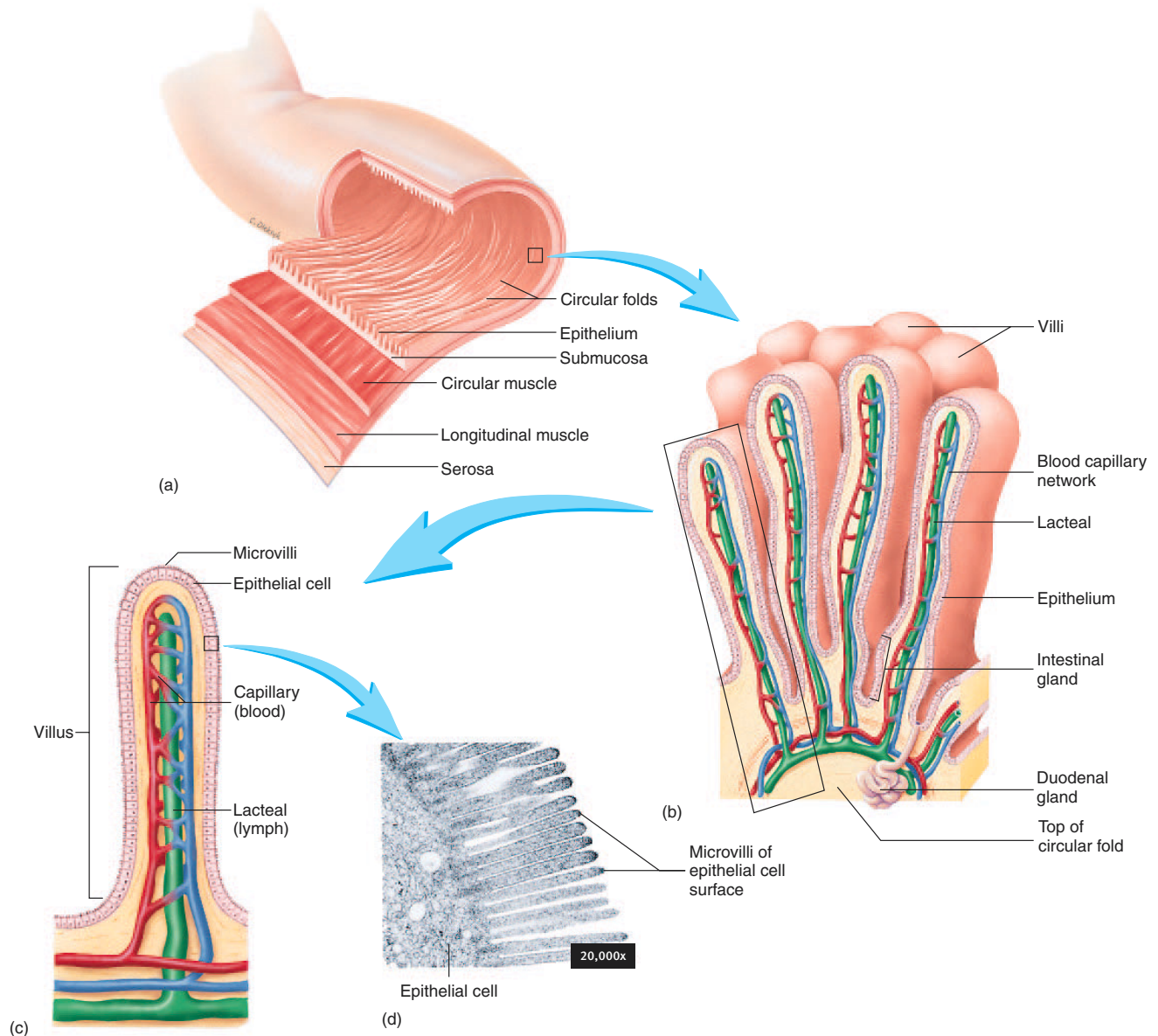


Figure 24.17 Anatomy and Histology of the Duodenum

(a) Wall of the duodenum, showing the circular folds. (b) The villi on a circular fold. (c) A single villus, showing the lacteal and capillary network. (d) Transmission electron micrograph of microvilli on the surface of a villus.

Jejunum and Ileum

The **jejunum** and **ileum** are similar in structure to the duodenum (see figure 24.15), except that a gradual decrease occurs in the diameter of the small intestine, the thickness of the intestinal wall, the number of circular folds, and the number of villi as one progresses through the small intestine. The duodenum and jejunum are the major sites of nutrient absorption, although some absorption occurs in the ileum. Lymph nodules called **Peyer's patches** are numerous in the mucosa and submucosa of the ileum.

The junction between the ileum and the large intestine is the **ileocecal junction**. It has a ring of smooth muscle, the **ileocecal sphincter**, and a one-way **ileocecal valve** (see figure 24.24).

Secretions of the Small Intestine

The mucosa of the small intestine produces secretions that primarily contain mucus, electrolytes, and water. Intestinal secretions lubricate and protect the intestinal wall from the acidic chyme and the action of digestive enzymes. They also keep the chyme in the

small intestine in a liquid form to facilitate the digestive process (see table 24.2). The intestinal mucosa produces most of the secretions that enter the small intestine, but the secretions of the liver and the pancreas also enter the small intestine and play essential roles in the process of digestion. Most of the digestive enzymes entering the small intestine are secreted by the pancreas. The intestinal mucosa also produces enzymes, but these remain associated with the intestinal epithelial surface.

The duodenal glands, intestinal glands, and goblet cells secrete large amounts of mucus. This mucus provides the wall of the intestine with protection against the irritating effects of acidic chyme and against the digestive enzymes that enter the duodenum from the pancreas. Secretin and cholecystokinin are released from the intestinal mucosa and stimulate hepatic and pancreatic secretions (see figures 24.21 and 24.23).

The vagus nerve, secretin, and chemical or tactile irritation of the duodenal mucosa stimulate secretion from the duodenal glands. Goblet cells produce mucus in response to the tactile and chemical stimulation of the mucosa.

Duodenal Ulcer



Sympathetic nerve stimulation inhibits duodenal gland secretion, thus reducing the coating of mucus on the duodenal wall, which protects it against acid and gastric enzymes. If a person is highly stressed, elevated sympathetic activity may therefore inhibit duodenal gland secretion and increase the person's susceptibility to **duodenal ulcers**.

Enzymes of the intestinal mucosa are bound to the membranes of the absorptive cell microvilli. These surface-bound enzymes include **disaccharidases**, which break disaccharides down to monosaccharides; **peptidases**, which hydrolyze the peptide bonds between small amino acid chains; and **nucleases**, which break down nucleic acids (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 systems.

Movement in the Small Intestine

Mixing and propulsion of chyme are the primary mechanical events that occur in the small intestine. These functions are the result of segmental or peristaltic contractions, which are accomplished by the smooth muscle in the wall of the small intestine and which are only propagated for short distances. **Segmental contractions** (see figure 24.3) mix the intestinal contents, and **peristaltic contractions** propel the intestinal contents along the digestive tract. A few peristaltic contractions 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 contrac-

tion proceeds. The contractions move at a rate of about 1 cm/min. The movements are slightly faster at the proximal end of the small intestine and slightly slower at the distal end. 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 distention of the intestinal wall. Solutions that are either hypertonic or hypotonic, solutions with a low pH, and certain products of digestion like amino acids and peptides also stimulate contractions of the small intestine. Local reflexes, which are integrated within the enteric plexus of the small intestine, mediate the response of the small intestine to these mechanical and chemical stimuli. Stimulation through parasympathetic nerve fibers may also increase the motility of the small intestine, but the parasympathetic influences in the small 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 movement of chyme from the small intestine into the cecum. Cecal distention, 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.

27. Name and describe the three parts of the small intestine. What are the major and lesser duodenal papilla?
28. What are the circular folds, villi, and microvilli in the small intestine? What are their functions?
29. Name the four types of cells found in the duodenal mucosa, and state their functions.
30. 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.
31. List the enzymes of the small intestine wall and give their functions.
32. What are two kinds of movement of the small intestine? How are they regulated?
33. What is the function of the ileocecal sphincter?

Liver

Objective

- Describe the structure and function of the liver.

Anatomy of the Liver

The **liver** is the largest internal organ of the body, weighing about 1.36 kg (3 pounds), and it is in the right-upper quadrant of the abdomen, tucked against the inferior surface of the diaphragm (see figures 24.1 and 24.18). The liver consists of two major **lobes**, **left** and **right**, and two minor lobes, **caudate** and **quadrate**.

Chapter 24 Digestive System

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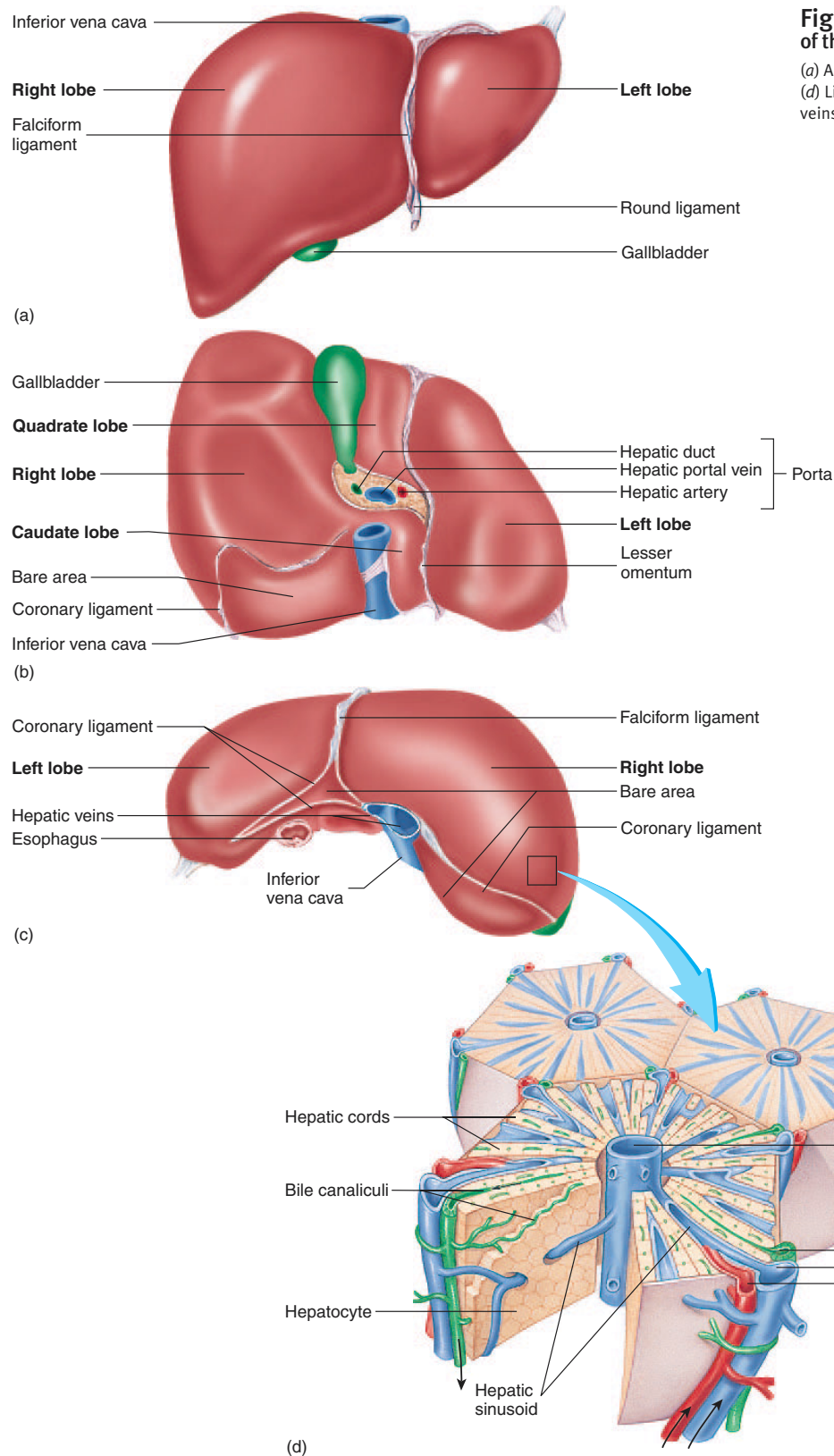
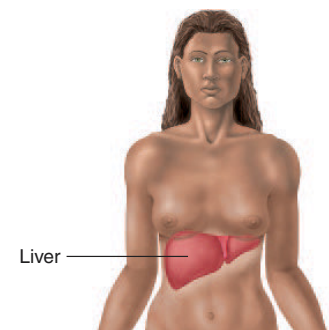


Figure 24.18 Anatomy and Histology of the Liver

(a) Anterior view. (b) Inferior view. (c) Superior view. (d) Liver lobules with triads at the corners and central veins in the center of the lobules.



A **porta** (gate) is on the inferior surface of the liver, where the various vessels, ducts, and nerves enter and exit the liver. The **hepatic** (he-pat'ik; associated with the liver) **portal vein**, the **hepatic artery**, and a small hepatic nerve plexus enter the liver through the porta (figure 24.19). Lymphatic vessels and two hepatic ducts, one each from the right and left lobes, exit the liver at the porta. The hepatic ducts transport bile out of the liver. The right and left hepatic ducts unite to form a single **common hepatic duct**. The **cystic duct** from the gallbladder joins the common hepatic duct to form the **common bile duct**, which joins the pancreatic duct at the **hepatopancreatic ampulla** (hē-pat'ō-pan-crē-at'ik am-pul'lā), an enlargement where the hepatic and pancreatic ducts come together. The hepatopancreatic ampulla empties into the duodenum at the major duodenal papilla (see figures 24.16a and 24.20). 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**, which is a small area on the diaphragmatic surface surrounded by the coronary ligament (see figure 24.18c). 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 connective tissue septa divide the liver into hexagon-shaped **lobules** with a **portal triad** at each corner. The triads are so named because three vessels—the hepatic portal vein, hepatic artery, and hepatic duct—are commonly located in them (see figure 24.18d). Hepatic nerves and lymphatic vessels, often too small to be easily seen in light micrographs, are also located in these areas. A **central vein** is in the center of each lobule. Central veins unite to form **hepatic veins**, which exit the liver on its posterior and superior surfaces and empty into the inferior vena cava (see figure 24.19).

Hepatic cords 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. The spaces between the hepatic cords are blood channels called **hepatic sinusoids**. 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** (Kupffer cells). A cleftlike lumen, the **bile canaliculus** (kan-ă-lik'ū-lūs; little canal), lies between the cells within each cord (see figure 24.18d).

Hepatocytes have six major functions (described in more detail starting on the next page): (1) bile production, (2) storage, (3) interconversion of nutrients, (4) detoxification, (5) phagocytosis, and (6) synthesis of blood components. Nutrient-rich, oxygen-poor blood from the viscera enters the hepatic sinusoids from branches of the hepatic portal vein and mixes with oxygen-rich, nutrient-depleted blood from the hepatic arteries. From the blood, the hepatocytes can take up the oxygen and nutrients, which are stored, detoxified, used for energy, or used to synthesize new molecules. Molecules produced by or modified in the hepatocytes are released into the hepatic sinusoids or into the bile canaliculi.

Mixed blood in the hepatic sinusoids flows to the central vein, where it exits the lobule and then exits the liver through the hepatic veins. **Bile**, produced by the hepatocytes and consisting primarily of metabolic by-products, 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).

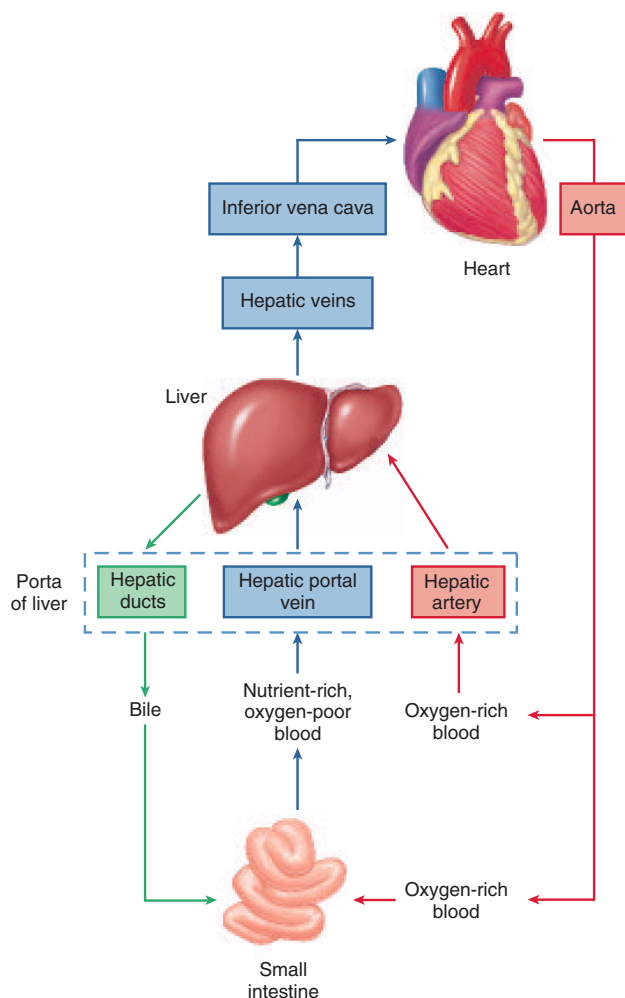


Figure 24.19 Blood and Bile Flow Through the Liver

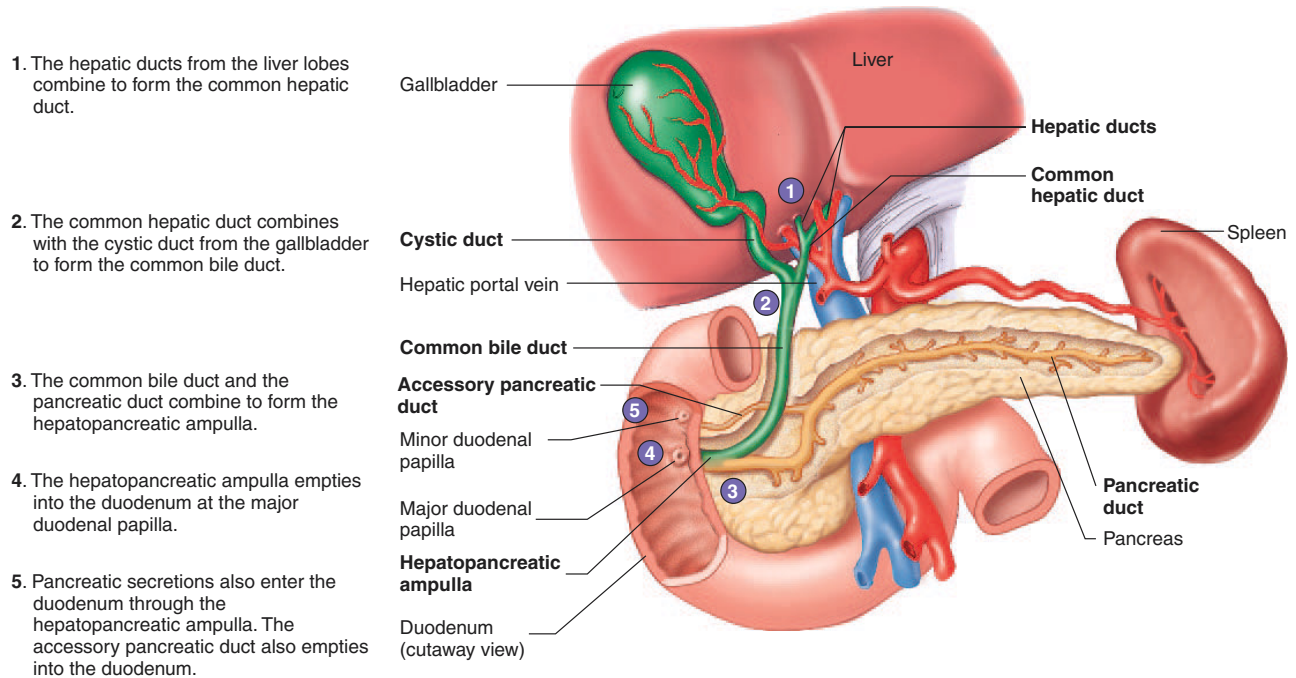


Figure 24.20 The Liver, Gallbladder, Pancreas, and Duct System

Liver Rupture or Enlargement

The liver is easily ruptured because it is large, fixed in position, and fragile, or it can be lacerated by a broken rib. Liver rupture or laceration results in severe internal bleeding.

The liver may become enlarged as a result of heart failure, hepatic cancer, cirrhosis, or Hodgkin's disease (a lymphatic cancer).

Functions of the Liver

The liver performs important digestive and excretory functions, stores and processes nutrients, synthesizes new molecules, and detoxifies harmful chemicals.

Bile Production

The liver produces and secretes about 600–1000 mL of bile each day (see table 24.2). Bile contains no digestive enzymes, but it plays a role in digestion because it neutralizes and dilutes stomach acid and emulsifies fats. The pH of chyme as it leaves the stomach is too low for the normal function of pancreatic enzymes. Bile helps to neutralize the acidic chyme and to bring the pH up to a level at which pancreatic enzymes can function. Bile salts **emulsify** fats (described in more detail on p. 896). Bile also contains excretory products like bile pigments. Bilirubin is a bile pigment that results from the breakdown of hemoglobin. Bile also contains cholesterol, fats, fat-soluble hormones, and lecithin.

Secretin stimulates bile secretion, primarily by increasing the water and bicarbonate ion content of bile (figure 24.21). Bile salts

also increase bile secretion through a positive-feedback system. Most bile salts are reabsorbed in the ileum and carried in the blood back to the liver, where they contribute to further bile secretion. The loss of bile salts in the feces is reduced by this recycling process. Bile secretion into the duodenum continues until the duodenum empties.

Storage

Hepatocytes can remove sugar from the blood and store it in the form of **glycogen**. They can also store fat, vitamins (A, B₁₂, D, E, and K), copper, and iron. This storage function is usually short term, and the amount of stored material in the hepatocytes and, thus, the cell size fluctuate during a given day.

Hepatocytes help control blood sugar levels within very narrow limits. If a large amount of sugar enters the general circulation after a meal, it will increase the osmolality of the blood and produce hyperglycemia. This is prevented because the blood from the intestine passes through the hepatic portal vein to the liver, where glucose and other substances are removed from the blood by hepatocytes, stored, and secreted back into the circulation when needed.

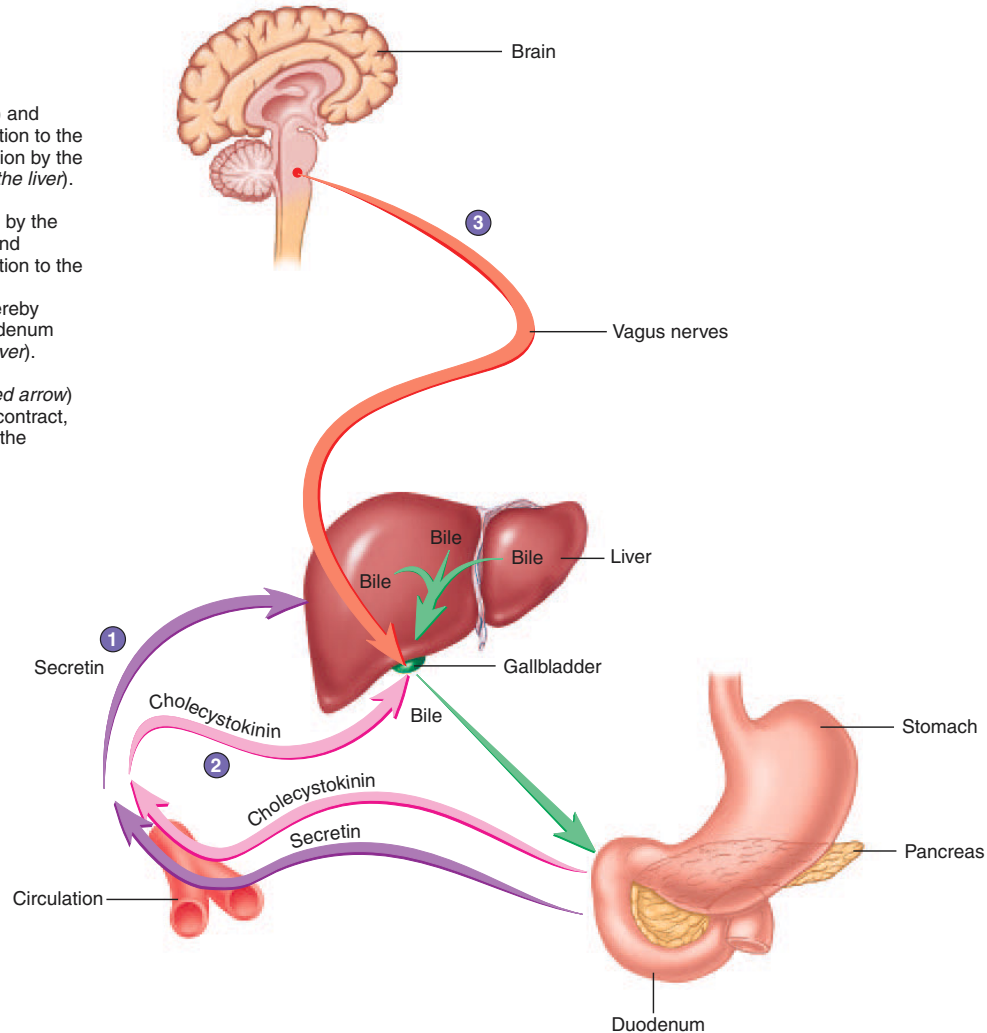
Nutrient Interconversion

Interconversion of nutrients is another important function of the liver. Ingested nutrients are not always in the proportion needed by the tissues. If this is the case, the liver can convert some nutrients into others. If, for example, a person is on a diet that is excessively

1. Secretin, produced by the duodenum (purple arrows) and carried through the circulation to the liver, stimulates bile secretion by the liver (green arrows inside the liver).

2. Cholecystokinin, produced by the duodenum (pink arrows) and carried through the circulation to the gallbladder, stimulates the gallbladder to contract, thereby releasing bile into the duodenum (green arrow outside the liver).

3. Vagal nerve stimulation (red arrow) causes the gallbladder to contract, thereby releasing bile into the duodenum.



Process Figure 24.21 Control of Bile Secretion and Release

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 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, ingested fats are combined with choline and phosphorus in the liver to produce phospholipids, which are essential components of plasma membranes. Vitamin D is hydroxylated in the liver. The hydroxylated form of vitamin D is the major circulating form of vitamin D, which is transported through the circulation to the kidney, where it's 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 the cells of the body. In addition, the body itself produces many by-products of metabolism that, if accumulated, are toxic. The liver forms a major line of defense against many of these harmful substances. It detoxifies many substances by altering their structure to make them less toxic or make their elimination easier. Ammonia, for example, a by-product of amino acid metabolism, is toxic and is not readily removed from the circulation by the kidneys. Hepatocytes remove ammonia from the circulation and convert it to urea, which is less toxic than ammonia and is secreted into the circulation and then eliminated by the kidneys in the urine. Other substances are removed from the circulation and excreted by the hepatocytes into the bile.

Phagocytosis

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 circulation.

Synthesis

The liver can also produce its own unique new compounds. It produces many blood proteins, such as albumins, fibrinogen, globulins, heparin, and clotting factors, which are released into the circulation (see chapter 19).



Hepatitis, Cirrhosis, and Liver Damage

Strictly defined, **hepatitis** is an inflammation of the liver and does not necessarily result from an infection. Hepatitis can be caused by alcohol consumption or infection. Infectious hepatitis is caused by viral infections. Hepatitis A, also called infectious hepatitis, is responsible for about 30% of hepatitis cases in the U.S. Hepatitis B, also called serum hepatitis, is a more chronic infection responsible for half the hepatitis cases in the U.S. Hepatitis C, also called non-A and non-B hepatitis, causes 20% of the U.S. hepatitis cases. It's caused by one or more virus types that cannot be identified in blood tests. It's spread by blood transfusions or sexual intercourse. If hepatitis is not treated, liver cells die and are replaced by scar tissue, resulting in loss of liver function. Death caused by liver failure can occur.

Cirrhosis (sir-rō'sis) of the liver involves the death of hepatocytes and their replacement by fibrous connective tissue. The liver becomes pale in color (the term *cirrhosis* means a tawny or orange condition) because of the presence of excess white connective tissue. It also becomes firmer, and the surface becomes nodular. The loss of hepatocytes eliminates the function of the liver, often resulting in jaundice, and the buildup of connective tissue can impede blood flow through the liver. Cirrhosis frequently develops in alcoholics and may develop as a result of biliary obstruction, hepatitis, or nutritional deficiencies.

Under most conditions, mature hepatocytes can proliferate and replace lost parts of the liver. If the liver is severely damaged, however, the hepatocytes may not have enough regenerative power to replace the lost parts. In this case, a liver transplant may be necessary. Recent evidence suggests that the liver also maintains an undifferentiated stem cell population, called “oval” cells, which gives rise to two cell lines, one forming bile duct epithelium and the other producing hepatocytes. It is hoped that these stem cells can be used to reconstitute a severely damaged liver. It may even be possible at some time in the future to remove stem cells from a person with hemophilia, genetically engineer the cells to produce the missing clotting factors, and then reintroduce the altered stem cells into the person's liver.

- 34. Describe the lobes of the liver. What is the porta?
- 35. Diagram the duct system from the liver, gallbladder, and pancreas that empties into the major duodenal papilla.
- 36. What are the hepatic cords and the sinusoids?
- 37. Describe the flow of blood to and through the liver. Describe the flow of bile away from the liver.
- 38. Explain and give examples of the major functions of the liver.
- 39. What stimulates bile secretion from the liver?

Gallbladder

Objective

- Describe the structure and function of the gallbladder.

The **gallbladder** is a saclike structure on the inferior surface of the liver that is about 8 cm long and 4 cm wide (see figure 24.20). Three tunics form the gallbladder wall: (1) an inner mucosa folded into rugae that 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.

Bile is continually secreted by the liver and flows to the gallbladder, where 40–70 mL of bile can be stored. While the bile is in the gallbladder, water and electrolytes are absorbed, and bile salts and pigments become as much as 5–10 times more concentrated than they were when secreted by the liver. Shortly after a meal, the gallbladder contracts in response to stimulation by cholecystokinin and, to a lesser degree, in response to vagal stimulation, thereby dumping large amounts of concentrated bile into the small intestine (see figure 24.21).



Gallstones

Cholesterol, secreted by the liver, may precipitate in the gallbladder to produce **gallstones** (figure A). Occasionally, a gallstone can pass out of the gallbladder and enter the cystic duct, blocking release of bile. Such a condition interferes with normal digestion, and the gallstone often must be removed surgically. If the gallstone moves far enough down the duct, it can also block the pancreatic duct, resulting in pancreatitis.



Figure A Gallstones

Drastic dieting with rapid weight loss may lead to gallstone production. In one study, 25% of obese people participating in an 8-week, quick-weight-loss program developed gallstones. Six percent required surgical removal of the stones. No gallstones developed in nondieting obese controls.

- 40. Describe the three tunics of the gallbladder wall.
- 41. What is the function of the gallbladder? What stimulates the release of bile from the gallbladder?

Pancreas

Objective

- Explain the structure and function of the pancreas.

Anatomy of the Pancreas

The **pancreas** is a complex organ composed of both endocrine and exocrine tissues that perform several functions. The pancreas consists of a **head**, located within the curvature of the duodenum (see figure 24.16a), a **body**, and a **tail**, which extends to the spleen.

The endocrine part of the pancreas consists of **pancreatic islets** (islets of Langerhans; see figure 24.16b). The islet cells produce insulin and glucagon, which are very important in controlling 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 discussion of glands in chapter 4). The **acini** (as'i-nī; grapes; see figure 24.16b) produce digestive enzymes. Clusters of acini form lobules that are separated by thin septa. Lobules are connected by small **intercalated ducts** to **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 (see figures 24.16a and 24.20). 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 are called **pancreatic juice** and have two major components: an aqueous component and an enzymatic component. Pancreatic juice is produced in the pancreas and is then delivered through the pancreatic ducts to the small intestine, where it functions in digestion. The **aqueous component** is produced principally by columnar epithelial cells that line the smaller ducts of the pancreas. It contains Na^+ and K^+ ions in about the same concentration found in extracellular fluid. Bicarbonate ions are a major part of the aqueous component, and they 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 but provides the proper environment for the function of pancreatic enzymes. Bicarbonate ions are actively secreted by the duct epithelium, and water follows passively to make the pancreatic juice isotonic. The cellular mechanism that is responsible for the secretion of bicarbonate ions is diagrammed in figure 24.22.

The enzymes of the pancreatic juice are produced by the acinar cells of the pancreas and are important for the digestion of all major classes of food. Without the enzymes produced by the pancreas, lipids, proteins, and carbohydrates are not adequately digested (see tables 24.1 and 24.2).

The proteolytic pancreatic enzymes, which digest proteins, are secreted in inactive forms, whereas many of the other enzymes are secreted in active form. The major proteolytic enzymes are **trypsin**, **chymotrypsin**, and **carboxypeptidase**. They are secreted in their inactive forms as trypsinogen, chymotrypsinogen, and procar-

boxypeptidase and are activated by the removal of certain peptides from the larger precursor proteins. If these were produced in their active forms, they would digest the tissues producing them. The proteolytic enzyme **enterokinase** (en'tēr-ō-kī'nās; intestinal enzyme), which is an enzyme attached to the brush border of the small intestine, activates trypsinogen. Trypsin then activates more trypsinogen, as well as chymotrypsinogen and procarboxypeptidase.

Pancreatic juice also contains **pancreatic amylase**, which continues the polysaccharide digestion that was initiated in the oral cavity. In addition, pancreatic juice contains a group of lipid-digesting enzymes called **pancreatic lipases**, which break down lipids into free fatty acids, glycerides, cholesterol, and other components.

Enzymes that reduce DNA and ribonucleic acid to their component nucleotides, **deoxyribonucleases** and **ribonucleases**, respectively, are also present in pancreatic juice.



Pancreatitis and Pancreatic Cancer

Pancreatitis is an inflammation of the pancreas and occurs quite commonly. Pancreatitis involves the release of pancreatic enzymes within the pancreas itself, which digest pancreatic tissue. It may result from alcoholism, 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.

Cancer of the pancreas can obstruct the pancreatic and the common hepatic ducts, resulting in jaundice. Pancreatic cancer may not be detected until the mass has become fairly large and may become so large as to block off the pyloric region of the stomach.

Regulation of Pancreatic Secretion

Both hormonal and neural mechanisms (figure 24.23) control the exocrine secretions of the pancreas. Secretin stimulates the secretion of a watery solution that contains a large amount of bicarbonate ions from the pancreas. The primary stimulus for secretin release is the presence of acidic chyme in the duodenum.

P R E D I C T 6

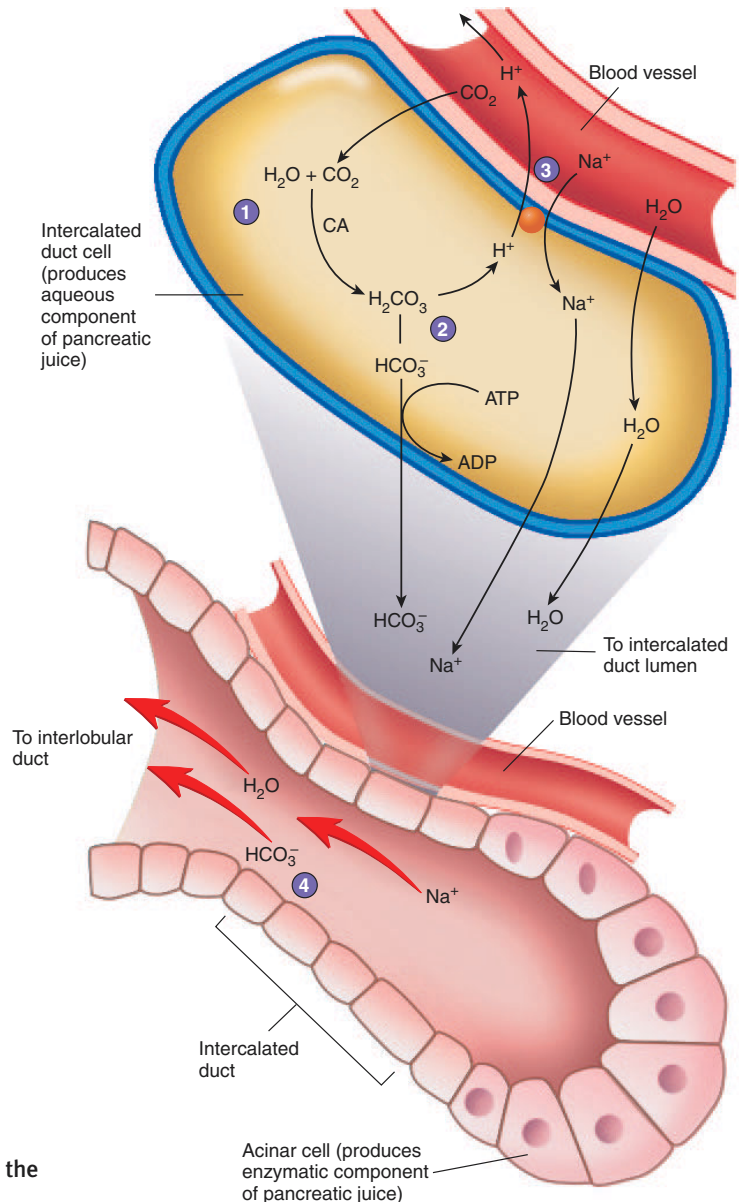
Explain why secretin production in response to acidic chyme and its stimulation of bicarbonate ion secretion constitute a negative-feedback mechanism.

Cholecystokinin stimulates the release of bile from the gallbladder and the secretion of pancreatic juice rich in digestive enzymes. 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 pancreatic juices rich in pancreatic enzymes, and sympathetic impulses inhibit secretion. The effect of vagal stimulation on pancreatic juice secretion is greatest during the cephalic and gastric phases of stomach secretion.

- 42. Describe the parts of the pancreas responsible for endocrine and exocrine secretions. Diagram the duct system of the pancreas.
- 43. Name the two kinds of exocrine secretions produced by the pancreas. What stimulates their production and what is their function?
- 44. What are the enzymes present in pancreatic juice? Explain the function of each.

1. Water (H_2O) and carbon dioxide (CO_2) combine under the influence of carbon anhydrase (CA) to form carbonic acid.
2. Carbonic acid (H_2CO_3) dissociates to form hydrogen ions (H^+) and bicarbonate ions (HCO_3^-).
3. The H^+ are exchanged for sodium ions (Na^+) and are removed in the bloodstream.
4. The HCO_3^- are actively transported into the intercalated ducts. Na^+ and water follow the HCO_3^- ions into the ducts.



Process Figure 24.22 Bicarbonate Ion Production in the Pancreas

Large Intestine

Objective

- Describe the anatomy and functions of the large intestine.

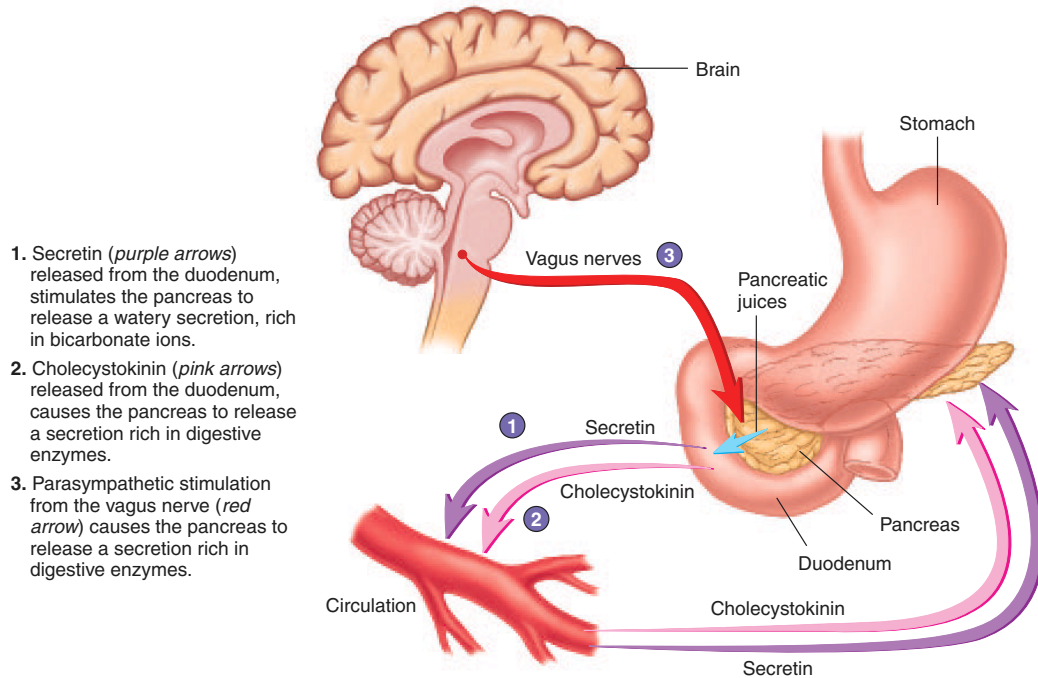
The **large intestine** is the portion of the digestive tract extending from the ileocecal junction to the anus. It consists of the cecum, colon, rectum, and anal canal. Normally 18–24 hours are required for material to pass through the large intestine, in contrast to the 3–5 hours required for movement of chyme 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. Absorption of water and salts, the secretion of mu-

cus, and extensive action of microorganisms are involved in the formation of feces, which the colon stores until the feces are eliminated by the process of defecation. About 1500 mL of chyme enters the cecum each day, but more than 90% of the volume is reabsorbed so that only 80–150 mL of feces is normally eliminated by defecation.

Anatomy of the Large Intestine

Cecum

The **cecum** (sē'kūm; blind) is the proximal end of the **large intestine**. It's where the large and small intestines meet at the ileocecal junction. The cecum extends inferiorly about 6 cm past the ileocecal junction in the form of a blind sac (figure 24.24). Attached to



Process Figure 24.23 Control of Pancreatic Secretion

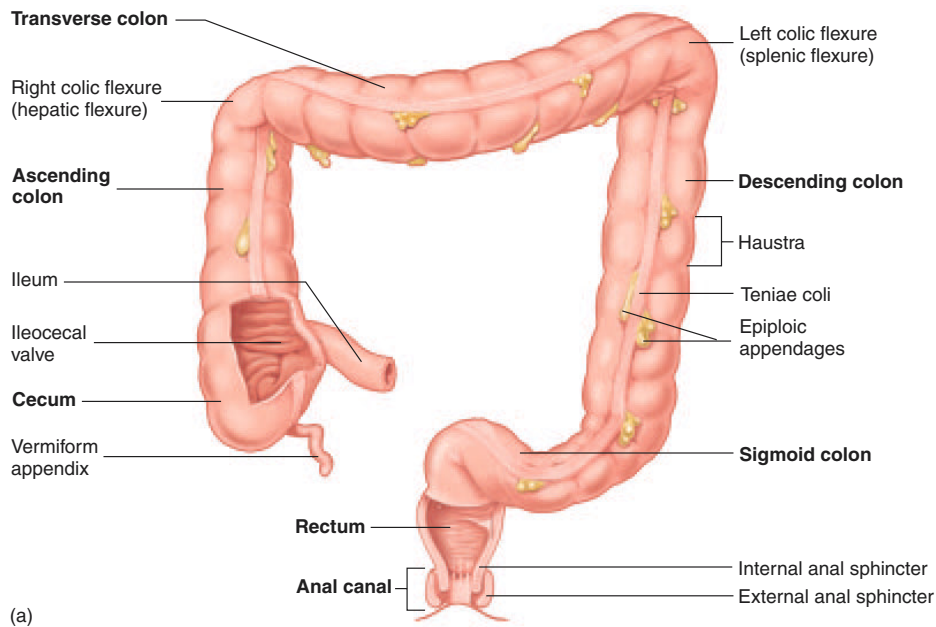


Figure 24.24 Large Intestine

(a) Large intestine (i.e., cecum, colon, and rectum) and anal canal. The teniae coli and epiploic appendages are along the length of the colon. (b) A radiograph of the large intestine following a barium enema.

the cecum is a small blind tube about 9 cm long called the **vermiform** (ver'mi-fōrm; worm-shaped) **appendix**. The walls of the appendix contain many lymphatic nodules.



Appendicitis

Appendicitis is an inflammation of the vermiform appendix and usually occurs because of obstruction of the appendix. Secretions from the appendix cannot pass the obstruction and accumulate, causing 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, along with a slight fever, loss of appetite, constipation or diarrhea, nausea, and vomiting. In the right-lower quadrant of the abdomen, about midway along a line between the umbilicus and the right anterior superior iliac spine, is an area on the body's surface called McBurney's point. This area becomes very tender in patients with acute appendicitis because of pain referred from the inflamed appendix to the body's surface. Each year, 500,000 people in the United States suffer an appendicitis. An appendectomy is removal of the appendix. If the appendix bursts, the infection can spread throughout the peritoneal cavity, causing peritonitis, with life-threatening results.

Colon

The **colon** (kō'lon) is about 1.5–1.8 m long and consists of four parts: the ascending colon, transverse colon, descending colon, and sigmoid colon (see figure 24.24). 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 circular muscle layer of the colon is complete, but the longitudinal muscle layer is incomplete. The longitudinal layer doesn't completely envelop the intestinal wall but forms three bands, called the **teniae coli** (tē'nē-ē kō'li; a band or tape along the colon), that run the length of the colon (see figures 24.24 and 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, fat-filled connective tissue pouches called **epiploic** (ep'i-plō'ik; related to the omentum) **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** (see figure 24.25). The crypts are somewhat similar to the intestinal glands of the small intestine, with three cell types that include absorptive, goblet, and granular cells. 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 termination of the sigmoid colon and ends at the anal canal (see figure 24.24). The mucosal lining of the rectum is simple columnar epithelium, and the muscular tunic is relatively thick compared to the rest of the digestive tract.

Anal Canal

The last 2–3 cm of the digestive tract is the **anal canal** (see figure 24.24). It begins at the inferior end of the rectum and ends at the **anus** (external GI 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 the superior end of the anal canal. 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.



Hemorrhoids

Hemorrhoids are the enlargement, or inflammation, of the hemorrhoidal veins, which supply the anal canal. The condition is also called varicose hemorrhoidal veins. Hemorrhoids cause pain, itching, and bleeding around the anus. Treatments include increasing the bulk (indigestible fiber) in the diet, taking sitz baths, and using hydrocortizone suppositories. Surgery may be necessary if the condition is extreme and doesn't respond to other treatments.

Secretions of the Large Intestine

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



Diarrhea

When the large intestine is irritated and inflamed, such as in patients with bacterial enteritis (inflamed intestine resulting from bacterial infection of the bowel), the intestinal mucosa secretes large amounts of mucus and electrolytes, and water moves by osmosis into the colon. An abnormally frequent discharge of fluid feces is called **diarrhea**. Although such discharge increases fluid and electrolyte loss, it also moves the infected feces out of the intestine more rapidly and speeds recovery from the disease.

A molecular pump exchanges bicarbonate ions for chloride ions in epithelial cells of the colon in response to acid produced by colic bacteria. Another pump exchanges sodium ions for hydrogen ions. Water crosses the wall of the colon through osmosis with the sodium chloride gradient.

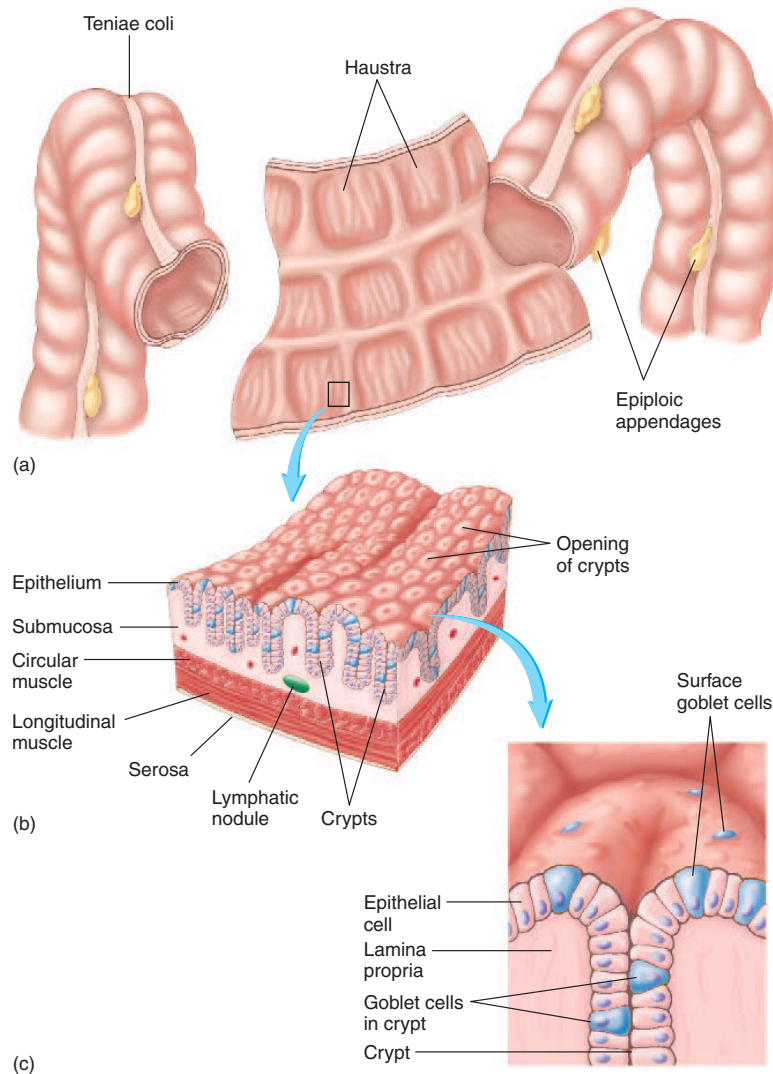


Figure 24.25 Histology of the Large Intestine

(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.

The feces that leave the digestive tract consist of water, solid substances (e.g., undigested food), microorganisms, and sloughed-off epithelial cells.

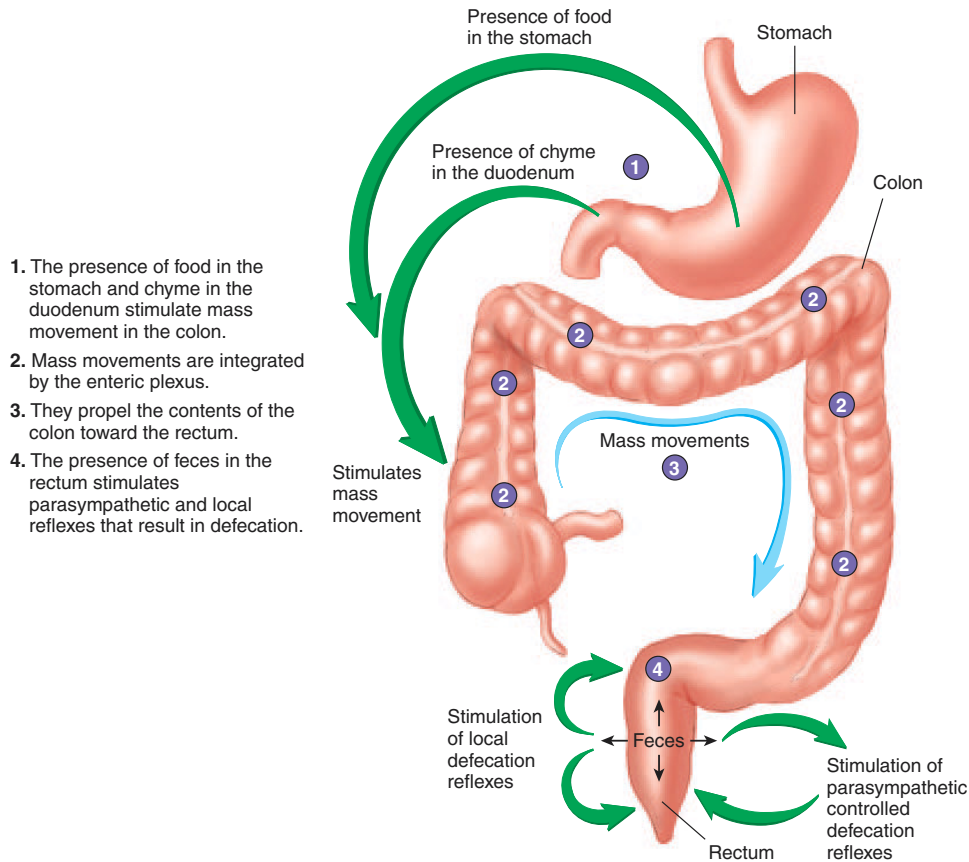
Numerous microorganisms inhabit the colon. They reproduce rapidly and ultimately constitute about 30% of the dry weight of the feces. Some bacteria in the intestine synthesize vitamin K, which is passively absorbed in the colon, and break down a small amount of cellulose to glucose.

Bacterial actions in the colon produce gases called **flatus** (flā'tūs; blowing). The amount of flatus depends partly on the bacterial population present 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 peristaltic contractions, called **mass movements**. Each mass movement contraction extends over a much longer part of the digestive tract (≥ 20 cm) than does a



Process Figure 24.26 Reflexes in the Colon and Rectum

peristaltic contraction and propels the colon contents a considerable distance toward the anus (figure 24.26). Mass movements are very common after meals because the presence of food in the stomach or duodenum initiates them. 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. Local reflexes in the enteric plexus, which are called **gastrocolic reflexes** if initiated by the stomach or **duodenocolic reflexes** if initiated by the duodenum, integrate mass movements.

Distention of the rectal wall by feces acts as a stimulus that initiates the **defecation reflex**. Local reflexes cause weak contractions of the rectum and relaxation of the internal anal sphincter. Parasympathetic reflexes cause strong contractions of the rectum and are normally responsible for most of the defecation reflex. Action potentials produced in response to the distention travel along afferent nerve fibers to the sacral region of the spinal cord, where efferent action potentials are initiated that reinforce peristaltic contractions in the lower colon and rectum. The defecation reflex reduces action potentials to the internal anal sphincter, causing it to relax. The external anal sphincter, which is composed of skeletal muscle and is under conscious cerebral control, prevents the movement of feces out of the rec-

tum and through the anal opening. If this sphincter is relaxed voluntarily, feces are expelled. 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 the reinitiation of the defecation reflex.

Defecation is usually accompanied by voluntary movements that support the expulsion of feces. These voluntary movements include 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, thereby helping force the contents of the colon through the anal canal and out of the anus.

- 45. Describe the parts of the large intestine. What are *teniae coli*, *haustra*, and *crypts*?
- 46. Explain the difference in structure between the internal anal sphincter and the external anal sphincter.
- 47. Name the substances secreted and absorbed by the large intestine. What is the role of microorganisms in the colon?
- 48. What kind of movements occur in the colon? Describe the defecation reflex.

On Being “Regular”

The importance of regularity of defecation has been greatly overestimated. Many people have the misleading notion that a daily bowel movement is critical for good health. As with many other body functions, what is “normal” differs from person to person. Whereas many people defecate one or more times per day, some normal, healthy adults defecate on the average only every other day. A defecation rate of only twice per week, however, is usually described as constipation. Habitually postponing defecation when the defecation reflex occurs can lead to constipation and may eventually result in desensitization of the rectum so that the defecation reflex is greatly diminished.

Digestion, Absorption, and Transport

Objectives

- Describe the process of digestion, absorption, and transport for carbohydrates, lipids, and proteins.
- Describe the movement of water and ions through the intestinal wall.

Digestion is the breakdown of food to molecules that are small enough to be absorbed into the circulation. **Mechanical digestion** breaks large food particles down into smaller ones. **Chemical digestion** involves the breaking of covalent chemical bonds in organic molecules by digestive enzymes. Carbohydrates are broken down into monosaccharides, proteins are broken down into amino acids, and fats are broken down into fatty acids and glycerol. **Absorption** and **transport** are the means by which molecules are moved out of the digestive tract and into the circulation for distribution throughout the body. Not all molecules (e.g., vitamins, minerals, and water) are broken down before being absorbed. Digestion begins in the oral cavity and continues in the stomach, but most digestion occurs in the proximal end of the small intestine, especially in the duodenum.

Absorption of certain molecules can occur all along the digestive tract. 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 circulation. Most absorption, however, occurs in the duodenum and jejunum, although some absorption occurs in the ileum.

Once the digestive products have been absorbed, they are transported to other parts of the body by two different routes. Water, ions, and water-soluble digestion products, such as glucose and amino acids, enter the hepatic portal system and are transported to the liver. The products of lipid metabolism are coated with proteins and transported into lymphatic capillaries called lacteals (see figure 24.17c). The lacteals are connected by lymphatic vessels to the thoracic duct (see chapter 21), which empties into the left subclavian vein. The protein-coated lipid products then travel in the circulation to adipose tissue or to the liver.

Carbohydrates

Ingested **carbohydrates** consist primarily of polysaccharides, such as starches and glycogen; disaccharides, such as sucrose (table sugar) and lactose (milk sugar); and monosaccharides, such as glucose and fructose (found in many fruits). During the digestion process, polysaccharides are broken down into smaller chains and finally into disaccharides and monosaccharides. Disaccharides are broken down into monosaccharides. Carbohydrate digestion begins in the oral cavity with the partial digestion of starches by **salivary amylase** (am'il-ās). A minor amount of digestion occurs in the stomach through the action of gastric amylase and gelatinase. Carbohydrate digestion is continued in the intestine by **pancreatic amylase** (table 24.4). A series of **disaccharidases** that are bound to the microvilli of the intestinal epithelium digest disaccharides into monosaccharides.

Lactose Intolerance

Lactase deficiency results in **lactose intolerance**, which is an inability to digest milk products. This disorder is primarily hereditary, affecting 5%–15% of Europeans and 80%–90% of Africans and Asians. Symptoms include cramps, bloating, and diarrhea.

Monosaccharides such as glucose and galactose are taken up into intestinal epithelial cells by cotransport, powered by a sodium ion gradient (figure 24.27). Monosaccharides such as fructose are taken up by facilitated diffusion. The monosaccharides are transferred by facilitated diffusion to the capillaries of the intestinal villi and are carried by the hepatic portal system to the liver, where the nonglucose sugars 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.

Type I Diabetes Mellitus

In patients with type I diabetes mellitus, insulin is lacking, and insufficient glucose is transported into the cells of the body. As a result, the cells do not have enough energy for normal function, blood glucose levels become significantly elevated, and abnormal amounts of glucose are released into the urine. This condition is discussed more fully in 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-glīs'er-īdz), also called **triacylglycerol** (trī-as'il-glīs'er-ol), consist of three fatty acids and one glycerol molecule covalently bound together. The first step in lipid digestion is **emulsification** (ē-mūl'si-fi-kā'shūn), which is the transformation of large lipid droplets into much smaller droplets. The enzymes that digest lipids are water-soluble and can digest the lipids only by acting at the surface of the droplets. The emulsification process increases the surface area of the lipid exposed to the digestive

enzymes by decreasing the droplet size. Emulsification is accomplished by **bile salts** secreted by the liver and stored in the gallbladder.

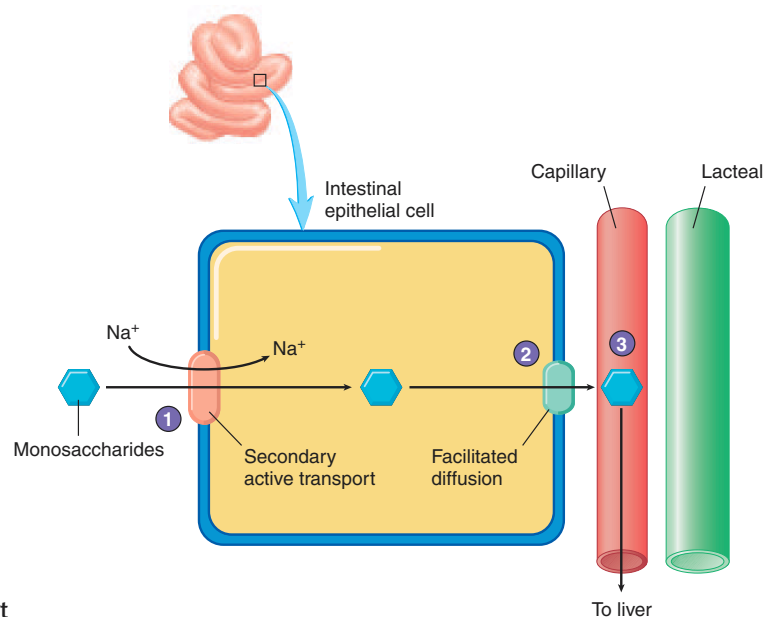
Lipase (lip'ās) digests lipid molecules (see table 24.4). The vast majority of lipase is secreted by the pancreas. A minor amount of lingual lipase is secreted in the oral cavity, is swallowed with the

food, and digests a small amount (<10%) of lipid in the stomach. The stomach also produces very small amounts of gastric lipase. The primary products of lipase digestion are free fatty acids and glycerol. Cholesterol and phospholipids also constitute part of the lipid digestion products.

Table 24.4 Digestion of the Three Major Food Types

	Carbohydrates	Proteins	Lipids
<i>Mouth (Salivary Glands)</i>	Salivary amylase → Polysaccharides Disaccharides		
<i>Stomach</i>	Gastric amylase and gelatinase →	Pepsin → Dipeptides Polypeptides	Lingual lipase → Gastric lipase →
<i>Duodenum (Pancreas)</i>	Pancreatic amylase → Disaccharides	Trypsin → Chymotrypsin → Carboxypeptidase →	Lipase → Esterase →
<i>Lining of Small Intestine</i>	Lactase → Sucrase → Maltase → Isomaltase → Monosaccharides	Aminopeptidase → Peptidase → Amino acids Dipeptides Tripeptides	Lipase → Glycerol Fatty acids

1. Monosaccharides are absorbed by secondary active transport into intestinal epithelial cells.
2. Monosaccharides move out of intestinal epithelial cells by facilitated diffusion.
3. They enter the capillaries of the intestinal villi and are carried through the hepatic portal vein to the liver.



Process Figure 24.27 Monosaccharide Transport

Once lipids are digested in the intestine, bile salts aggregate around the small droplets to form **micelles** (mī-selz', mī-selz'; a small morsel; figure 24.28). The hydrophobic ends of the bile salts are directed toward the free fatty acids, cholesterol, and glycerides at the center of the micelle; and the hydrophilic ends are directed outward toward the water environment. When a micelle comes into contact with the epithelial cells of the small intestine, the contents of the micelle pass by means of simple diffusion through the plasma membrane of the epithelial cells.

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's 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 chloride ion transport channel proteins, which affects chloride transport and, as a result, 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, fats and fat-soluble vitamins, which require bile salts to form micelles and which cannot be adequately digested without pancreatic enzymes, are not well digested and absorbed. The patient suffers from vitamin A, D, E, and K deficiencies, which result in conditions like night blindness, skin disorders, rickets, and excessive bleeding. Therapy includes administering the missing vitamins to the patient and reducing dietary fat intake.

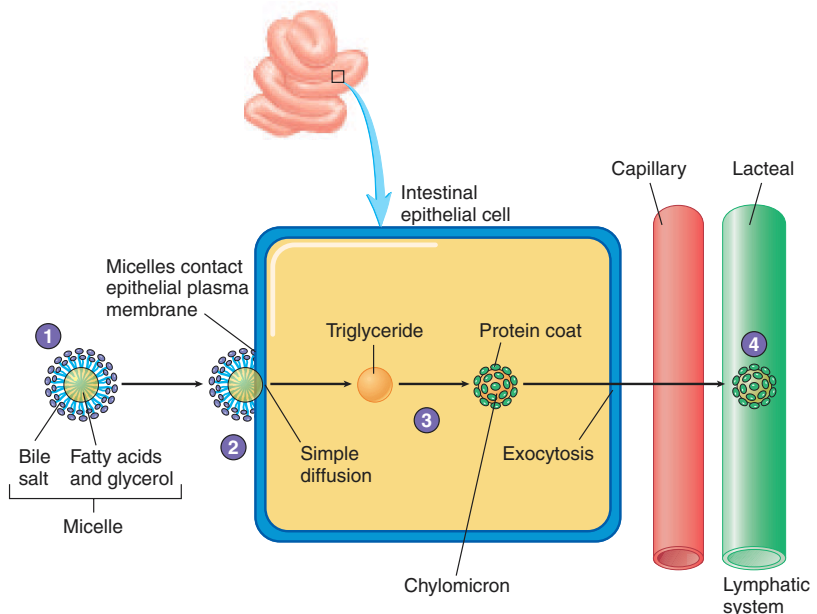
Lipid Transport

Within the smooth endoplasmic reticulum of the intestinal epithelial cells, free fatty acids are combined with glycerol molecules to form triglycerides. Proteins synthesized in the epithelial cells attach

to droplets of triglycerides, phospholipids, and cholesterol to form **chylomicrons** (kī-lō-mī'kronz; small particles in the chyle, or fat-filled lymph). The chylomicrons leave the epithelial cells and enter the lacteals of the lymphatic system within the villi. Chylomicrons enter the lymphatic capillaries rather than the blood capillaries because the lymphatic capillaries lack a basement membrane and are more permeable to large particles like chylomicrons (about 0.3 mm in diameter). Chylomicrons are about 90% triglyceride, 5% cholesterol, 4% phospholipid, and 1% protein (figure 24.29). They are carried through the lymphatic system to the bloodstream and then by the blood to adipose tissue. Before entering the adipose cells, triglyceride is broken back down into fatty acids and glycerol, which enter the fat cells and are once more converted to triglyceride. 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 circulation to the liver, where it is broken 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**. Chylomicrons are one type of lipoprotein. Other lipoproteins are referred to as high- or low-density lipoproteins. 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, have 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 (see figure 24.29).

1. Bile salts surround fatty acids and glycerol to form micelles.
2. Micelles attach to the plasma membranes of intestinal epithelial cells, and the fatty acids and glycerol pass by simple diffusion into the intestinal epithelial cells.
3. Within the intestinal epithelial cell, the fatty acids and glycerol are converted to triglyceride; proteins coat the triglyceride to form chylomicrons, which move out of the intestinal epithelial cells by exocytosis.
4. The chylomicrons enter the lacteals of the intestinal villi and are carried through the lymphatic system to the general circulation.



Process Figure 24.28 Lipid Transport

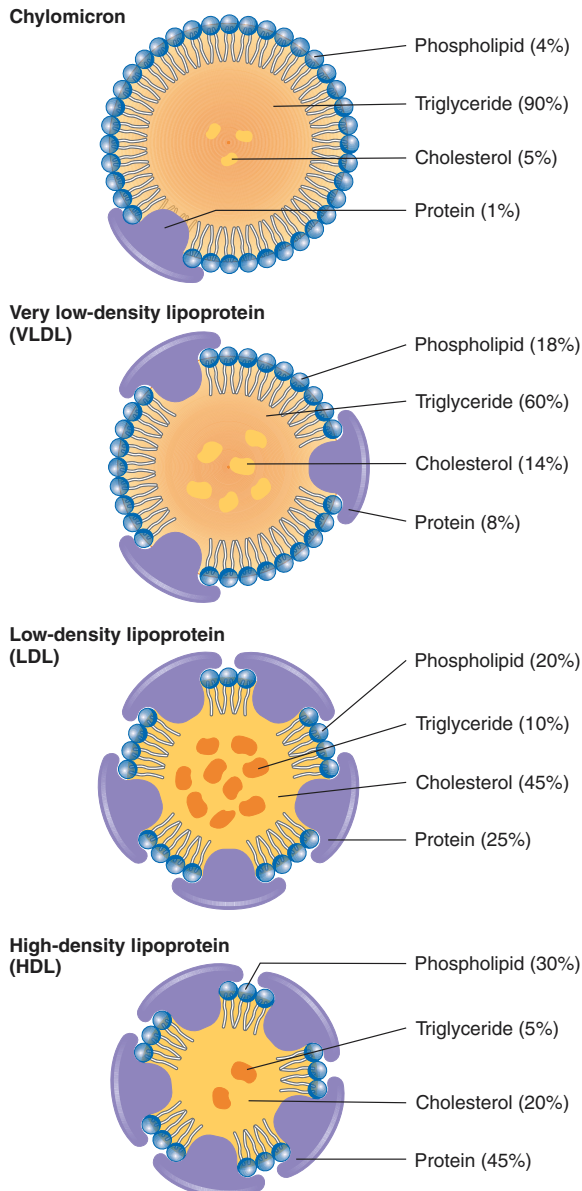


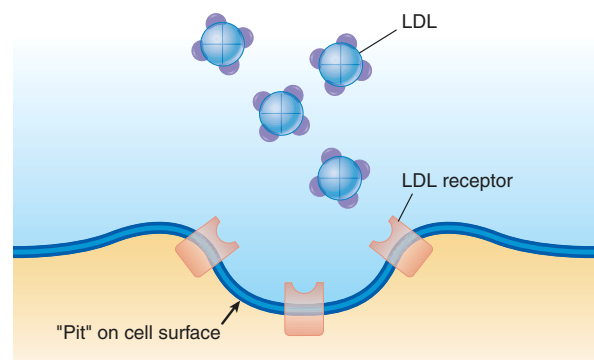
Figure 24.29 Lipoproteins

About 15% of the cholesterol in the body is ingested in the food we eat, and the remaining 85% is manufactured in the cells of the body, mostly in the liver and intestinal mucosa. Most of the lipid taken into or manufactured in the liver leaves the liver in the form of VLDL. Most of the triglycerides are removed from the VLDL to be stored in adipose tissue and, as a result, VLDL becomes LDL.

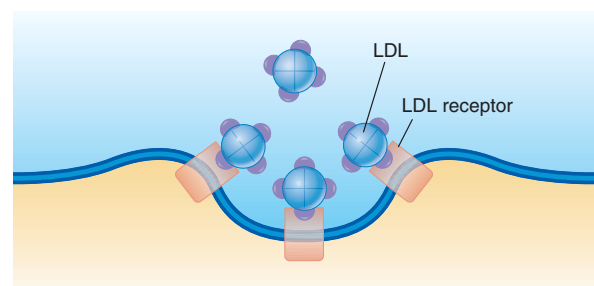
The cholesterol in LDL is critical for the production of steroid hormones in the adrenal cortex and the production of bile acids in the liver. It's also an important component of plasma membranes. LDL is delivered to cells of various tissues through the circulation. Cells have **LDL receptors** in "pits" 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 LDL is taken into the cell by receptor-mediated endocytosis (figure 24.30). Each fibroblast, as an example of a tissue cell, has 20,000–50,000 LDL receptors on the surface. Those receptors are confined to cell surface pits, however, 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.

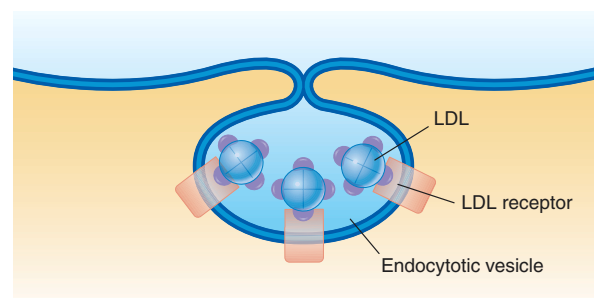
Cells not only take in cholesterol and other lipids from LDLs, but they also make their own cholesterol. When the combined intake and manufacture of cholesterol exceeds a cell's needs, a negative-feedback system functions, which reduces the amount of LDL receptors and cholesterol manufactured by the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



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



LDL binds to the LDL receptors in the pits.



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

Figure 24.30 Transport of LDL into Cells

Cholesterol and Coronary Heart Disease

Cholesterol is a major component of atherosclerotic plaques. The level of plasma cholesterol is positively linked to coronary heart disease (CHD). Cholesterol levels of over 200 mg/100 mL increase the risk of CHD. Other risk factors, which are additive to high cholesterol levels, are hypertension, diabetes mellitus, cigarette smoking, and low plasma HDL levels. Low HDL levels are linked to obesity, and weight reduction increases HDL levels. Aerobic exercise can decrease LDL levels and increase HDL levels. Ingestion of saturated fatty acids raises plasma cholesterol levels by stimulating LDL production and inhibiting LDL receptor production, which would enhance HDL production and cholesterol clearance. Ingestion of unsaturated fatty acids lowers plasma cholesterol levels. Replacing fats by carbohydrates in the diet can also reduce blood cholesterol levels. The American Heart Association recommends that no more than 30% of an adult's total caloric intake should be from fats and that only 10% be from saturated fats. Our total cholesterol intake should be no more than 300 mg/day. People should eat no more than 7 ounces of meat per day, and that should be chicken, fish, or lean meat. We should eat only two eggs per week and drink milk with 1% or less butter fat. Young children, however, require more fats in their diet to stimulate normal brain development, and whole milk is recommended in their diets. Some evidence also exists that severely reducing plasma cholesterol levels, below about 180 mg/100 mL may be harmful in adults. Cholesterol is required for normal membrane structure in cells. Abnormally low cholesterol levels, may lead to weakened blood vessel walls and an increased risk for cerebral hemorrhage.

A small number of people have a genetic disorder in the production or function of LDL receptors, resulting in poor LDL clearing, and, as a result, have what is called **familial hypercholesterolemia**. These people commonly develop premature atherosclerosis and are prone to die at an early age of a heart attack. In some of these disorders, the LDL receptor is not produced. In other cases, the receptor is produced, but it has a lower-than-normal affinity for LDL. In yet other cases, the receptor binds to LDL but the receptor–LDL complex is not taken into the cell by endocytosis.

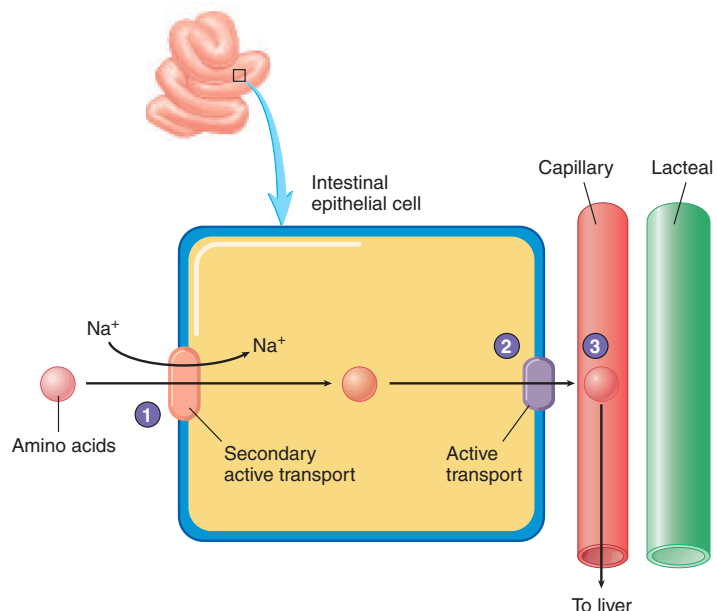
Proteins

Proteins are taken into the body from a number of dietary sources. **Pepsin** secreted by the stomach (see table 24.3) catalyzes the cleavage of covalent bonds in proteins to produce smaller polypeptide chains. Gastric pepsin digests as much as 10%–20% of the total ingested protein. Once the proteins and polypeptide chains leave the stomach, proteolytic enzymes produced in the pancreas continue the digestive process and produce small peptide chains. These are broken down into dipeptides, tripeptides, 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 peptide bond.

Dipeptides and tripeptides enter intestinal epithelial cells through a group of related carrier molecules, by a cotransport mechanism, powered by a sodium ion concentration gradient similar to that described for glucose. Separate molecules transport basic, acidic, and neutral amino acids into the epithelial cells. Acidic and most neutral amino acids are cotransported with a sodium ion gradient, whereas basic amino acids enter the epithelial cells by facilitated diffusion. The total amount of each amino acid that enters the intestinal epithelial cells as dipeptides or 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 24.31). 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 are used as building blocks to form new proteins (see chapter 2), but some amino acids may be used for energy.

1. Amino acids are absorbed by secondary active transport into intestinal epithelial cells.
2. Amino acids move out of intestinal epithelial cells by active transport.
3. They enter the capillaries of the intestinal villi and are carried through the hepatic portal vein to the liver.



Process Figure 24.31 Amino Acid Transport

Water

About 9 L of **water** enters the digestive tract each day, of which about 92% is absorbed in the small intestine, and another 6%–7% is absorbed in the large intestine (figure 24.32). Water can move in either direction across the wall of the small intestine. Osmotic gradients across the epithelium determine the direction of its 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 intestine into the surrounding extracellular fluid. Water in the extracellular fluid can then enter the circulation. Because of the osmotic gradient produced as nutrients are absorbed in the small intestine, 92% of the water that enters the small intestine by way of the oral cavity, stomach, or intestinal secretions is reabsorbed.

Ions

Active transport mechanisms for **sodium** ions are present within the epithelial cells of the small intestine. **Potassium**, **calcium**, **magnesium**, and **phosphate** are also actively transported. **Chlo-**

ride ions move passively through the intestinal wall of the duodenum and the jejunum following the positively charged sodium ions, but chloride ions are actively transported from the ileum. Although calcium ions are actively transported along the entire length of the small intestine, vitamin D is required for that transport process. The absorption of calcium is under hormonal control, as is its excretion and storage. Parathyroid hormones, calcitonin, and vitamin D all play a role in regulating blood levels of calcium in the circulatory system (see chapters 6, 18, and 27).

- 49. Describe the mechanism of absorption and the route of transport for water-soluble and lipid-soluble molecules.
- 50. Describe the enzymatic digestion of carbohydrates, lipids, and proteins, and list the breakdown products of each.
- 51. Explain how fats are emulsified. Describe the role of micelles, chylomicrons, VLDLs, LDLs, and HDLs in the absorption and transport of lipids in the body.
- 52. Explain how dipeptides and tripeptides enter intestinal epithelial cells.
- 53. Describe the movement of water through the intestinal wall.
- 54. When and where are various ions absorbed?

Effects of Aging on the Digestive System

Objective

- Describe the effects of aging on the digestive tract.

As a person ages, gradual changes occur throughout the entire 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. There is also a decrease in the number of smooth muscle cells in the muscularis, resulting in decreased motility in the digestive tract. In addition, goblet cells within the mucosa secrete less mucus. Glands along the digestive tract, such as the gastric pits, the liver, and the pancreas, also tend to secrete less with age. These changes by themselves don't appreciably decrease the function of the digestive system.

Through the years, however, 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 may be toxic to the tissues. Because the connective tissue of the digestive tract becomes thin with age and because the protective mucus covering is reduced, the digestive tract of elderly people becomes less and less protected from these outside influences. In addition, the mucosa of elderly people tends to heal more slowly following injury. The liver's ability to detoxify certain chemicals tends to decline, the ability of the hepatic phagocytic cells to remove particulate contaminants decreases, and the liver's ability to store glycogen decreases. These problems are increased in people who smoke.

This overall decline in the defenses of the digestive tract with advancing age leaves elderly people more susceptible to infections and to the effects of toxic agents. Elderly people are 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.

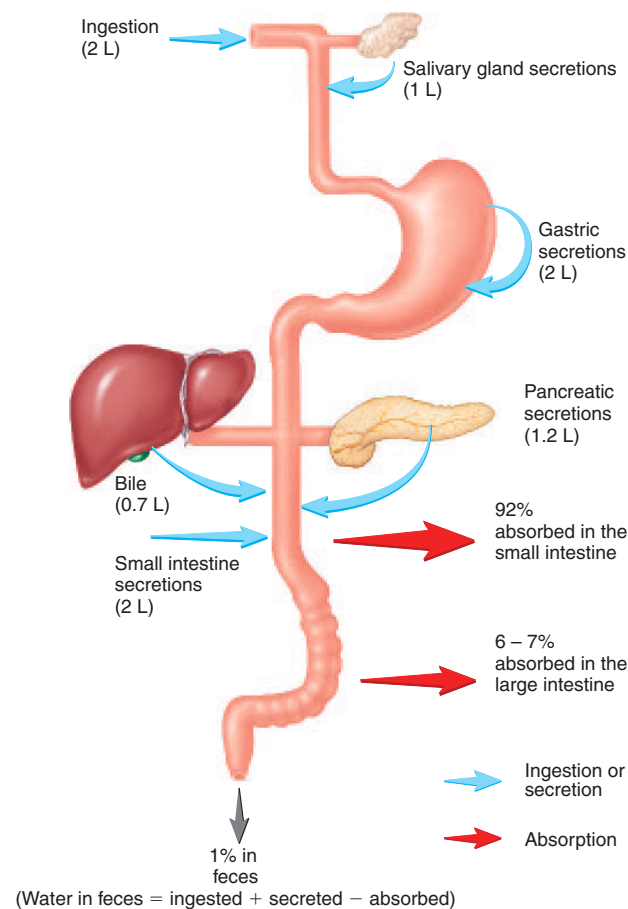


Figure 24.32 Fluid Volumes in the Digestive Tract

Clinical Focus Intestinal Disorders

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is the general name given to either Crohn's disease or ulcerative colitis. IBD occurs at a rate in Europe and North America of approximately 4 to 8 new cases per 100,000 people per year, which is much higher than in Asia and Africa. Males and females are affected about equally. IBD is of unknown cause, but infectious, autoimmune, and hereditary factors have been implicated. **Crohn's disease** involves localized inflammatory degeneration that may occur anywhere along the digestive tract but most commonly involves the distal ileum and proximal colon. The degeneration involves the entire thickness of the digestive tract wall. The intestinal wall often becomes thickened, constricting the lumen, with ulcerations and fissures in the damaged areas. The disease causes diarrhea, abdominal pain, fever, and weight loss. Treatment centers around anti-inflammatory drugs, but other treatments, including avoiding foods that increase symptoms and even surgery, are employed. **Ulcerative colitis** is limited to the mucosa of the large intestine. The involved mucosa exhibits inflammation, including edema, vascular congestion, hemorrhage, and the accumulation of

plasma cells, lymphocytes, neutrophils, and eosinophils. Patients may experience abdominal pain, fever, malaise, fatigue, and weight loss, as well as diarrhea and hemorrhage. In rare cases, severe diarrhea and hemorrhage may require transfusions. Treatment includes the use of anti-inflammatory drugs and, in some cases, avoiding foods that increase symptoms.

Irritable Bowel Syndrome

Irritable bowel syndrome (IBS) is a disorder of unknown cause in which intestinal mobility is abnormal. The disorder accounts for over half of all referrals to gastroenterologists. Male and female children are affected equally, but adult females are affected twice as often as males. IBS patients experience abdominal pain mainly in the left lower quadrant, especially after eating. They also have alternating bouts of constipation and diarrhea. There is no specific histopathology in the digestive tracts of IBS patients. There are no anatomic abnormalities, no indication of infection, and no sign of metabolic causes. Patients with IBS appear to exhibit greater-than-normal levels of psychological stress or depression and show increased contractions of the esophagus

and small intestine during times of stress. There is a high familial incidence. Some patients might present with a history of traumatic events such as physical or sexual abuse. Treatments include psychiatric counseling and stress management, diets with increased fiber and limited gas-producing foods, and loose clothing. In some patients, drugs that reduce parasympathetic stimulation of the digestive system may be useful.

Malabsorption Syndrome

Malabsorption syndrome (sprue) is a spectrum of disorders of the small intestine that results in abnormal nutrient absorption. One type of malabsorption results from an immune response to gluten, which is present in certain types of grains and involves the destruction of newly formed epithelial cells in the intestinal glands. These cells fail to migrate to the villi surface, the villi become blunted, and the surface area decreases. As a result, the intestinal epithelium is less capable of absorbing nutrients. Another type of malabsorption (called tropical malabsorption) is apparently caused by bacteria, although no specific bacterium has been identified.

Gastroesophageal reflux disorder (GERD) increases with advancing age. It is probably the main reason that elderly people take antacids, H₂ antagonists, and proton pump inhibitors. Disorders that are not necessarily age-induced, such as hiatal hernia and irregular or inadequate esophageal motility, may be worsened by the effects of aging, because of a 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 artificial teeth are provided. The muscles of mastication tend to become weaker and, as a result, older people tend to chew their food less before swallowing.

Another complication of the age-related changes in the digestive system is 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 circulatory system. 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.

- 55. What is the general effect of aging on digestive tract secretions?
- 56. What are the effects of the overall decline in the defenses of the digestive tract with advancing age?

Enteritis

Enteritis is any inflammation of the intestines that can result in diarrhea, dehydration, fatigue, and weight loss. It may result from an infection, chemical irritation, or from some unknown cause. **Regional enteritis**, or Crohn's disease, is a local enteritis of unknown cause characterized by patchy, deep ulcers developing in the intestinal wall, usually in the distal end of the ileum. The disease results in overproliferation of connective tissue and invasion of lymphatic tissue into the involved area, with a subsequent thickening of the intestinal wall and narrowing of the lumen.

Colitis is an inflammation of the colon.

Colon Cancer

Colon cancer is the second leading cause of cancer-related deaths in the United States and accounts for 55,000 deaths a year. Susceptibility to colon cancer can be familial; however, a correlation exists between colon cancer and diets low in fiber and high in fat. People who eat beef, pork, or lamb daily have 2.5 times the risk of developing colon cancer compared to people who eat these meats less than once per month. Eating processed meats increases the risk by an additional 50%–100%. Ingesting calcium in the form of calcium carbonate antacid tablets at twice

the recommended daily allowances may prevent 75% of colon cancers. Greatly increased calcium levels may also cause constipation.

A gene for colon cancer may be present in as many as 1 in 200 people, making colon cancer one of the most common inherited diseases. Nine different genes have been found to be associated with colon cancer. Most of those genes are involved in cell regulation, that is, keeping cell growth in check, but one gene mutation results in a high degree of genetic instability. As a result of this mutation, the DNA is not copied accurately during cell division of the colon cancer cells, causing wholesale errors and mutations throughout the genome (all the genes). Such genetic instability has been identified in 13% of sporadic (not occurring in families) colon cancer. Screening for colon cancer includes testing the stool for blood content and performing a colonoscopy, which allows the physician to see into the colon.

Constipation

Constipation is the slow movement of feces through the large intestine. The feces often become dry and hard because of increased fluid absorption during the extended time they are retained in the large intestine. In the United States, 2.5 million doctor visits

occur each year from people complaining of constipation, and \$400 million dollars is spent each year on laxatives.

Constipation often results after a prolonged time of inhibiting normal defecation reflexes. A change in habits, such as travel, dehydration, depression, disease, metabolic disturbances, certain medications, pregnancy, or dependency on laxatives can all cause constipation. Irritable bowel syndrome, also called spastic colon, which is of unknown cause but is stress-related, can also cause constipation. Constipation can also occur with diabetes, kidney failure, colon nerve damage, or spinal cord injuries or as the result of an obstructed bowel; of greatest concern, the obstruction could be caused by colon cancer. Chronic constipation can result from the slow movement of feces through the entire colon, in just the distal part (descending colon and rectum), or in just the rectum. Interestingly, in one large study of people who claimed to be suffering from chronic constipation, one-third were found to have normal movement of feces through the large intestine. Defecation frequency was often normal. Many of those people were suffering from psychologic distress, anxiety, or depression and just thought they had abnormal defecation frequencies.

S U M M A R Y

Anatomy of the Digestive System (p. 860)

1. The digestive system consists of a digestive tube and its associated accessory organs.
2. The digestive system consists of the oral cavity, pharynx, esophagus, stomach, small intestine, large intestine, and anus.
3. Accessory organs such as the salivary glands, liver, gallbladder, and pancreas are located along the digestive tract.

Functions of the Digestive System (p. 860)

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

Histology of the Digestive Tract (p. 862)

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

Mucosa

The mucosa consists of a mucous epithelium, a lamina propria, and a muscularis mucosae.

Submucosa

The submucosa is a connective tissue layer containing the submucosal plexus (part of the enteric plexus), blood vessels, and small glands.

Systems Pathology

Diarrhea

While on vacation in Mexico, Mr. T was shopping with his wife when he started to experience sharp pains in his abdominal region (figure B). He also began to feel hot and sweaty and felt an extreme urge to defecate. His wife quickly looked up the word *toilet* in their handy Spanish–English pocket travel dictionary, and Mr. T anxiously inquired of a local resident where the nearest facility could be found. Once the immediate need was taken care of, Mr. and Mrs. T went back to their hotel room, where they remained while Mr. T recovered. Over the next 2 days, his stools were frequent and watery. He also vomited a couple of times. Because they were in a foreign country, Mr. T didn’t consult a physician. He rested, took plenty of fluids, and was feeling much better, although a little weak, in a couple of days.

Background Information

Diarrhea is one of the most common complaints in clinical medicine and affects more than half of the tourists in developing countries. **Diarrhea** is defined as any change in bowel habits in which stool frequency or volume is increased or in which stool fluidity is increased. Diarrhea is not itself a disease but is a symptom of a wide variety of disorders. Normally, about 600 mL of fluid enters the colon each day and all but 150 mL is reabsorbed. The loss of more than 200 mL of stool per day is considered abnormal.

Mucus secretion by the colon increases dramatically in response to diarrhea. This mucus contains large quantities of bicarbonate ions, which comes from the dissociation of carbonic acid into bicarbonate ions (HCO_3^-) and hydrogen (H^+) ions within the blood supply to the colon. The HCO_3^- enter the mucus secreted by the colon, whereas the H^+ remain in the circulation and, as a result, the blood pH decreases. Thus, a condition called metabolic acidosis can develop (see chapter 27).



Figure B Many Tourists Develop Diarrhea

Diarrhea in tourists usually results from the ingestion of food or water contaminated with bacteria or bacterial toxins. Acute diarrhea is defined as lasting less than 2–3 weeks, and diarrhea lasting longer than that 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 gut or an increase in fluid secretion. Some bacterial toxins and other chemicals can also cause an increase in bowel motor activity. As a result, chyme is moved more rapidly through the digestive tract, fewer nutrients and water are absorbed out of the small intestine, and more water enters the colon. Symptoms can occur in as little as 1–2 hours after bacterial toxins are ingested to as long as 24 hours or more for some strains of bacteria.

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.

Serosa or Adventitia

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

Regulation of the Digestive System (p. 863)

1. Nervous, hormonal, and local chemical mechanisms regulate digestion.
2. Nervous regulation involves the enteric nervous system and CNS reflexes.
3. The digestive tract produces hormones that regulate digestion.
4. Other chemicals are produced by the digestive tract that exercise local control of digestion.

Peritoneum (p. 864)

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.

Oral Cavity (p. 866)

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

System Interactions The Effects of Diarrhea on Other Systems

System	Interactions with Digestive System
Integumentary	Pallor occurs due to vasoconstriction of blood vessels in the skin, resulting from a decrease in blood fluid levels. Pallor and sweating increase in response to abdominal pain and anxiety.
Muscular	Muscular weakness may result due to electrolyte loss, metabolic acidosis, fever, and general malaise. The involuntary stimulus to defecate may become so strong as to overcome the voluntary control mechanisms.
Nervous	Local reflexes in the colon respond to increased colon fluid volume by stimulating mass movements and the defecation reflex. Abdominal pain, much of which is felt as referred pain, can occur as the result of inflammation and distention of the colon. Decreased function is due to electrolyte loss. Reduced blood fluid levels stimulate a sensation of thirst in the CNS.
Endocrine	A decrease in extracellular fluid volume, due to the loss of fluid in the feces, stimulates the release of hormones (antidiuretic hormone from the posterior pituitary and aldosterone from the adrenal cortex) that increase water retention and electrolyte reabsorption in the kidney. In addition, decreased extracellular fluid volume and anxiety result in increased release of epinephrine and norepinephrine from the adrenal medulla.
Cardiovascular	Movement of extracellular fluid into the colon results in a decreased blood volume. The reduced blood volume activates the baroreceptor reflex, antidiuretic hormone release, the renin-angiotensin-aldosterone mechanism, and the fluid shift mechanism, which all function to elevate blood volume or increase blood pressure.
Lymphatic and immune	White blood cells migrate to the colon in response to infection and inflammation. In the case of bacterial diarrhea, the immune response is initiated to begin production of antibodies against bacteria and bacterial toxins.
Respiratory	As the result of reduced blood pH, the rate of respiration increases to eliminate carbon dioxide, which helps eliminate excess H^+ .
Urinary	A decrease in urine volume and an increase in urine concentration results from activation of the baroreceptor reflex, which decreases blood flow to the kidney; antidiuretic hormone secretion, which increases water reabsorption in the kidney; and aldosterone secretion, which increases electrolyte and water reabsorption in the kidney. After a period of approximately 24 hours, the kidney is activated to compensate for metabolic acidosis by increasing hydrogen ion secretion and bicarbonate ion reabsorption.

In cases of short-term acute diarrhea, the infectious agent is seldom identified. Nearly any bacterial species is capable of causing diarrhea. Some types of bacterial diarrhea include severe vomiting, whereas others do not. Some bacterial toxins also induce fever. Some viruses and amebic parasites can also cause diarrhea. In most cases, laboratory analysis of food or stool is necessary to identify the causal organism. In cases of mild diarrhea away from home, laboratory evaluation is not practical, and empiric therapy is usually applied. Fluids and electrolytes must be replaced, and consumption of fluids with elec-

trolytes is important. The diet should be limited to clear fluids during at least the first day or so. Bismuth subsalicylate (Pepto-Bismol) or loperamide (Imodium; except in cases of fever) may also be used to help combat secretory diarrhea. Milk and milk products should be avoided. Breads, toast, rice, and baked fish or chicken can be added to the diet with improvement. A normal diet can be resumed after 2–3 days.

P R E D I C T 7

Predict the effects of prolonged diarrhea.

- Twenty deciduous teeth are replaced by 32 permanent teeth.
 - The types of teeth are incisors, canines, premolars, and molars.
 - A tooth consists of a crown, a neck, and a root.
 - 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.
 - Periodontal ligaments hold the teeth in the alveoli.
- The muscles of mastication are the masseter, the temporalis, the medial pterygoid, and the lateral pterygoid.
- Salivary glands produce serous and mucous secretions. The three pairs of large salivary glands are the parotid, submandibular, and sublingual.

Pharynx (p. 870)

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

Esophagus (p. 870)

- The esophagus connects the pharynx to the stomach. The upper and lower esophageal sphincters regulate movement.
- 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 (p. 872)

- During the voluntary phase of deglutition, a bolus of food is moved by the tongue from the oral cavity to the pharynx.
- The pharyngeal phase is a reflex caused by 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.

Stomach (p. 872)

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. Mucus protects the stomach lining.
2. Pepsinogen is converted to pepsin, which digests proteins.
3. Hydrochloric acid promotes pepsin activity and kills microorganisms.
4. Intrinsic factor is necessary for vitamin B₁₂ absorption.
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. Distention 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.

Small Intestine (p. 881)

1. The small intestine is divided into the duodenum, jejunum, and ileum.
2. The wall of the small intestine consists of an external serosa, muscles (longitudinal and circular), submucosa, and simple columnar epithelium.
3. Circular folds, villi, and microvilli greatly increase the surface area of the intestinal lining.
4. 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. Chemical or tactile irritation, vagal stimulation, and secretin 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. Distention of the cecum initiates a reflex that inhibits peristalsis.

Liver (p. 884)

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 that are 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 bring 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 fats. Secretin increases bile production.
2. The liver stores and processes nutrients, produces new molecules, and detoxifies molecules.
3. Hepatic phagocytic cells phagocytize red blood cells, bacteria, and other debris.
4. The liver produces blood components.

Gallbladder (p. 889)

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.

Pancreas (p. 890)

1. The pancreas is 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.
4. Secretin stimulates the release of a watery bicarbonate solution that neutralizes acidic chyme.
5. Cholecystokinin and the vagus nerve stimulate the release of digestive enzymes.

Large Intestine (p. 890)

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 provides protection to the intestinal lining.
2. Epithelial cells secrete bicarbonate ions. Sodium is absorbed by active transport, 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 to 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.

Digestion, Absorption, and Transport (p. 896)

1. Digestion is the breakdown of organic molecules into their component parts.
2. Absorption and transport are the means by which molecules are moved out of the digestive tract and are distributed throughout the body.
3. Transportation occurs by two different 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 active transport or by facilitated diffusion.
4. The monosaccharides are carried to the liver where the nonglucose sugars 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. Emulsification is the transformation of large lipid droplets into smaller droplets and is accomplished by bile salts.
3. Lipase digests lipid molecules to form free fatty acids and glycerol.
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 glycerol to form triglyceride.
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. Triglyceride is 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 smaller polypeptide chains.
2. Proteolytic enzymes from the pancreas produce small peptide chains.
3. Peptidases, bound to the microvilli of the small intestine, break down peptides.
4. Amino acids are absorbed by cotransport, which requires transport of sodium.
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 can move 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.

Effects of Aging on the Digestive System (p. 901)

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

R E V I E W A N D C O M P R E H E N S I O N

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 enteric plexus is found in the
 - a. submucosa layer.
 - b. muscularis layer.
 - c. serosa layer.
 - d. both a and b.
 - e. all of the above.
3. The tongue
 - a. holds food in place during mastication.
 - b. plays a major role in swallowing.
 - c. helps to form words during speech.
 - d. is a major sense organ for taste.
 - e. all of the above.
4. 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.

5. The number of premolar deciduous teeth is
 - a. 0.
 - b. 2.
 - c. 4.
 - d. 8.
 - e. 12.
6. Which of these glands does *not* secrete saliva into the oral cavity?
 - a. submandibular glands
 - b. goblet glands
 - c. sublingual glands
 - d. parotid glands
7. The portion of the digestive tract in which digestion begins is the
 - a. oral cavity.
 - b. esophagus.
 - c. stomach.
 - d. duodenum.
 - e. jejunum.
8. During deglutition (swallowing),
 - a. 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.
9. The stomach
 - a. has large folds in the submucosa and mucosa called rugae.
 - b. has two layers of smooth muscle in the muscularis layer.
 - c. opening from the esophagus is the pyloric opening.
 - d. has an area closest to the duodenum called the fundus.
 - e. all of the above.
10. 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
11. HCl
 - a. is an enzyme.
 - b. creates the acid condition necessary for pepsin to work.
 - c. is secreted by the small intestine.
 - d. activates salivary amylase.
 - e. all of the above.
12. Why doesn't the stomach digest itself?
 - a. The stomach wall is not composed of protein, so it's 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.
13. Which of these hormones stimulates stomach secretions?
 - a. cholecystokinin
 - b. gastric inhibitory peptide
 - c. gastrin
 - d. secretin
14. Which of these phases of stomach secretion is correctly matched?
 - a. Cephalic phase: the largest volume of secretion is produced.
 - b. Gastric phase: gastrin secretion is inhibited by distention of the stomach.
 - c. Gastric phase: initiated by chewing, swallowing, or thinking of food.
 - d. Intestinal phase: stomach secretions are inhibited.
15. Which of these structures function to increase the mucosal surface of the small intestine?
 - a. circular folds
 - b. villi
 - c. microvilli
 - d. length of the small intestine
 - e. all of the above
16. Given these parts of the small intestine:
 1. duodenum
 2. ileum
 3. jejunumChoose the arrangement that lists the parts in the order food encounters them as it passes from the stomach through the small intestine.
 - a. 1,2,3
 - b. 1,3,2
 - c. 2,1,3
 - d. 2,3,1
 - e. 3,1,2
17. Which structures release digestive enzymes in the small intestine?
 - a. duodenal glands
 - b. goblet cells
 - c. endocrine cells
 - d. absorptive cells
18. 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 the above.
19. Given these ducts:
 1. common bile duct
 2. common hepatic duct
 3. cystic duct
 4. hepatic ductsChoose the arrangement that lists the ducts in the order bile passes through them when moving from the bile canaliculi of the liver to the small intestine.
 - a. 3,4,2
 - b. 3,2,1
 - c. 3,4,1
 - d. 4,1,2
 - e. 4,2,1
20. Which of these might occur if a person suffers from a severe case of hepatitis that impairs liver function?
 - a. Fat 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 the above.
21. 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.
22. The aqueous component of pancreatic secretions
 - a. is secreted by the pancreatic islets.
 - b. contains bicarbonate ions.
 - c. is released primarily in response to cholecystokinin.
 - d. passes directly into the blood.
 - e. all of the above.

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23. Given these structures:
 1. ascending colon
 2. descending colon
 3. sigmoid colon
 4. transverse colonChoose the arrangement that lists the structures in the order that food encounters them as it passes between the small intestine and the rectum.
 - a. 1,2,3,4
 - b. 1,4,2,3
 - c. 2,3,1,4
 - d. 2,4,1,3
 - e. 3,4,1,2
24. Which of these is *not* a function of the large intestine?
 - a. absorption of fats
 - b. absorption of certain vitamins
 - c. absorption of water and salts
 - d. production of mucus
 - e. all of the above
25. 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 the above.
26. 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
 - e. all of the above
27. Bile
 - a. is an important enzyme for the digestion of fats.
 - b. is made by the gallbladder.
 - c. contains breakdown products from hemoglobin.
 - d. emulsifies fats.
 - e. both c and d.
28. 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.
29. If the thoracic duct were tied off, which of these classes of nutrients would *not* enter the circulatory system at their normal rate?
 - a. amino acids
 - b. glucose
 - c. lipids
 - d. fructose
 - e. nucleotides
30. 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 in Appendix F

C R I T I C A L T H I N K I N G

1. While anesthetized, patients sometimes vomit. Given that the anesthetic eliminates the swallowing reflex, explain why it's 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 Worrystudent experienced the pain of a duodenal ulcer during final examination week. Describe the possible reasons. Explain what habits could have caused the ulcer, and recommend a reasonable remedy.
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 of the spinal cord. How will this injury affect the patient's ability to defecate? What components of the defecation response are still present, and which are lost?
6. The bowel (colon) occasionally can become impacted. Given what you know about the functions of the colon and the factors that determine the movement of substances across the colon wall, predict the effect of the impaction on the contents of the colon above the point of impaction.

Answers in Appendix G

A N S W E R S T O P R E D I C T Q U E S T I O N S

1. A pin placed through the greater omentum passes through four layers of simple squamous epithelium. The greater omentum is actually a folded mesentery, with each part consisting of two layers of serous squamous epithelium.
2. The moist stratified squamous epithelium of the oropharynx and the laryngopharynx protects these regions from abrasive food when it is first swallowed. The ciliated pseudostratified epithelium of the nasopharynx helps move mucus produced in the nasal cavity and the nasopharynx into the oropharynx and esophagus. It's not as necessary to protect the nasopharynx from abrasion because food does not normally pass through this cavity.
3. It's important for the nasopharynx to be closed during swallowing so that food doesn't reflux into it or the nasal cavity. An explosive burst of laughter can relax the soft palate, open the nasopharynx, and cause the liquid to enter the nasal cavity.
4. Usually if a person tries to swallow and speak at the same time, the epiglottis is elevated, the laryngeal muscles closing the opening to the larynx are mostly relaxed, and food or liquid could enter the larynx, causing the person to choke.
5. After a heavy meal, blood pH may increase because, as bicarbonate ions pass from the cells of the stomach into the extracellular fluid, the pH of the extracellular fluid increases. As the extracellular fluid exchanges ions with the blood, the blood pH also increases.

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