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Gastrointestinal disorders

Introduction

The GI tract, also known as the *alimentary canal,* is a long, hollow, musculomembranous tube consisting of glands and accessory organs (salivary glands, liver, gallbladder, and pancreas). (See *Reviewing GI anatomy and physiology.* See also *Histology of the GI tract,* page 236.) The GI tract breaks down food—carbohydrates, fats, and proteins—into molecules small enough to permeate cell membranes, thus providing cells with the necessary energy to function properly; it prepares food for cellular absorption by altering its physical and chemical composition. (See *Primary source of digestive hormones*, page 237.) Consequently, a malfunction along the GI tract can produce far-reaching metabolic effects, eventually threatening life itself. The GI tract is an unsterile system filled with bacteria and other flora; these organisms can cause superinfection from antibiotic therapy or they can infect other systems when a GI organ ruptures. A common indication of GI problems is referred pain, which makes diagnosis especially difficult.

Accurate assessment vital

Your assessment of the patient with suspected GI disease must begin with a careful history that includes occupation, family history, and recent travel. The medical history should include previous hospital admissions; surgical procedures (including recent tooth extraction); family history of ulcers, colitis, or cancer; and current medications, whether prescribed, over-the-counter, or herbal remedies, with

particular attention to aspirin, steroids, or anticoagulants. In addition, assess for food or drug allergies.

Have the patient describe his chief complaint in his own words. Does he have abdominal pain, indigestion, heartburn, or rectal bleeding? How long has he had it? What relieves these symptoms or makes them worse? Has he experienced nosebleeds or difficulty in swallowing recently? Has he had recent weight loss or gain? Is he on a special diet? Does he drink alcoholic beverages or smoke? If yes to either, how much and how often? Ask about bowel habits. Does he regularly use laxatives or enemas? If he experiences nausea and vomiting, what does the vomitus look like? Does changing his position relieve nausea?

Next, try to define and locate any pain. Ask the patient to describe the pain. Is it dull, sharp, burning, aching, spasmodic, or intermittent? Where is it located? Does it radiate? How long does it last? When does it occur? What triggers it? What relieves it?

Visual assessment

Observe how the patient looks, and note appropriateness of behavior. Changes in fluid and electrolyte balance, severe infection, drug toxicity, and hepatic disease may cause abnormal behavior. Your visual examination should check:

- *Skin*—loss of turgor, jaundice, cyanosis, pallor, diaphoresis, petechiae, bruises, edema, and texture (dry or oily)
- *Head*—color of sclerae, sunken eyes, dentures, caries, lesions, tongue (color, swelling, dryness), and breath odor
- *Chest*—shape (asymmetrical, barrel, or sunken)
- *Lungs*—rate, rhythm, and quality of respirations
- *Abdomen*—size and shape (distention, contour, visible masses, and protrusions), abdominal scars or fistulae, excessive skin folds (may indicate wasting), and abnormal respiratory movements (inflammation of diaphragm).

Auscultation, palpation, and percussion

Auscultation provides helpful clues to GI abnormalities and should always be performed before palpation and percussion to avoid altering the assessment. For example, absence of bowel sounds over the area to the lower right of the umbilicus may indicate peritonitis. High-pitched sounds that coincide with colicky pain may indicate small bowel obstruction. Less intense, lowpitched rumbling noises may accompany minor irritation.

Palpating the abdomen after auscultation helps detect tenderness, muscle guarding, and abdominal masses. Watch for muscle tone (boardlike rigidity points to peritonitis or hemorrhage; transient rigidity suggests severe pain) and tenderness (rebound

tenderness may indicate peritoneal inflammation).

REVIEWING GI ANATOMY AND PHYSIOLOGY

The GI tract includes the mouth, pharynx, esophagus, stomach (fundus, body, and antrum), small intestine (duodenum, jejunum, and ileum), and large intestine (cecum, colon, rectum, and anal canal).

Digestion begins in the mouth through chewing and through the action of an enzyme secreted in saliva—ptyalin (amylase)—which breaks down starch. Digestion continues in the stomach, where the lining secretes gastric juice that contains hydrochloric acid and the enzymes pepsin (begins protein digestion), lipase (speeds hydrolysis of emulsified fats) and, in infants, rennin (curdles milk).

Through a churning motion, the stomach breaks food into tiny particles, mixes them with gastric juice, and pushes the mass toward the pylorus. The liquid portion (chyme) enters the duodenum in small amounts; any solid material remains in the stomach until it liquefies (usually from 1 to 6 hours). The stomach also produces an intrinsic factor necessary for the absorption of vitamin B12. Although limited amounts of water, alcohol, and

some drugs are absorbed in the stomach, chyme passes unabsorbed into the duodenum.

Small intestine's role

Most digestion and absorption occur in the small intestine, where the surface area is increased by millions of villi in the mucous membrane lining. For digestion, the small intestine relies on a vast array of enzymes produced by the pancreas or by the intestinal lining itself. Pancreatic enzymes include trypsin, which digests protein to amino acids; lipase, which digests fat to fatty acids and glycerol; and amylase, which digests starches to sugars. Intestinal enzymes include erepsin, which digests protein to amino acids; lactase, maltase, and sucrase, which digest complex sugars like glucose, fructose, and galactose; and enterokinase, which activates trypsin.

In addition, bile, secreted by the liver and stored in the gallbladder, helps neutralize stomach acid and aids the small intestine to emulsify and absorb fats and fat-soluble vitamins.

Final stages

By the time ingested material reaches the ileocecal valve (where the small intestine joins the large intestine), all its nutritional value has been absorbed through the villi of the small intestine into the bloodstream.

The large intestine, so named because it's larger in diameter than the small intestine, absorbs water from the digestive material before passing it on for elimination. Rectal distention by feces stimulates the defecation reflex, which, when assisted by voluntary sphincter relaxation, permits defecation.

Throughout the GI tract, peristalsis (a coordinated, rhythmic contraction of smooth muscle) propels ingested material along; sphincters prevent its reflux.

Percussion helps detect air, fluid, and solid matter in the abdominal region.

Diagnostic tests

After physical assessment, several tests can identify GI malfunction.

- A barium or gastrografin swallow is used primarily to examine the esophagus. Gastrografin may be used instead of barium. Like barium, gastrografin facilitates X-ray imaging. However, if gastrografin escapes from the GI tract, it's absorbed by the surrounding tissue, whereas escaped barium isn't absorbed and can cause complications.

HISTOLOGY OF THE GI TRACT

The GI tract consists of four tissue layers whose structure varies in different organs:

- *Mucous membrane*—innermost layer; secretes gastric juice, protects the tract, and absorbs nutrients
- *Submucosa*—connective tissue that contains the major blood vessels and nerves
- *External muscle coat (muscularis externa)*—double layer of smooth-muscle fibers; inner circular and outer longitudinal layers propel gastric contents downward by peristalsis
- *Fibroserous coat (serosa)*—outermost protective layer of connective tissue; forms the peritoneum, which is the largest serous membrane of the body. The peritoneum's parietal layer covers the walls of the abdominal cavity. An extension of the parietal peritoneum, called the mesentery, anchors the small intestine to the abdominal wall. The visceral layer drapes most of the abdominal organs, covering the upper surface of the pelvic organs.

- In an upper GI series, swallowed barium sulfate travels through the esophagus, stomach, and duodenum to reveal abnormalities. The barium outlines stomach walls and delineates ulcer craters and defects.
- A small-bowel series, an extension of the upper GI series, visualizes barium flowing through the small intestine to the ileocecal valve.
- A barium enema (lower GI series) allows X-ray visualization of the colon.
- A stool specimen is useful to detect suspected GI bleeding, infection, or malabsorption as well as the presence of parasites. Guaiac test for occult blood, microscopic stool examination for ova and parasites, and tests for fat require several specimens.
- In esophagogastroduodenoscopy, insertion of a fiber-optic scope allows direct visual inspection of the esophagus, stomach, and duodenum. These structures are examined for varices, tumors, inflammation, hernias, polyps, ulcers, and obstruction.
- Proctosigmoidoscopy permits inspection of the rectum and distal sigmoid colon; colonoscopy is used for inspection of the descending, transverse, and ascending colon. These tests help visualize tumors, polyps, hemorrhoids, or ulcers.
- Gastric analysis examines gastric secretions for the presence of high levels of gastrin and the amount of acid produced.
- Endoscopic retrograde cholangiopancreatography directly visualizes the esophagus, stomach, proximal duodenum, and fluoroscopic visualization of the pancreatic, hepatic, and biliary ducts. This test can help visualize duct obstruction, benign structures, cysts, anatomic variations, and malignant tumors.

Intubation

Certain GI disorders require nasogastric (NG) intubation to empty the stomach and intestine, to aid diagnosis and treatment, to decompress obstructed areas, to detect and treat GI bleeding, and to administer medications or feedings. Tubes generally inserted through the nose are the short NG tubes (the Levin, the Salem Sump, and the specialized

Sengstaken-Blakemore) and the long intestinal tubes (Cantor and Miller-Abbott). The larger Ewald tube is usually inserted orally.

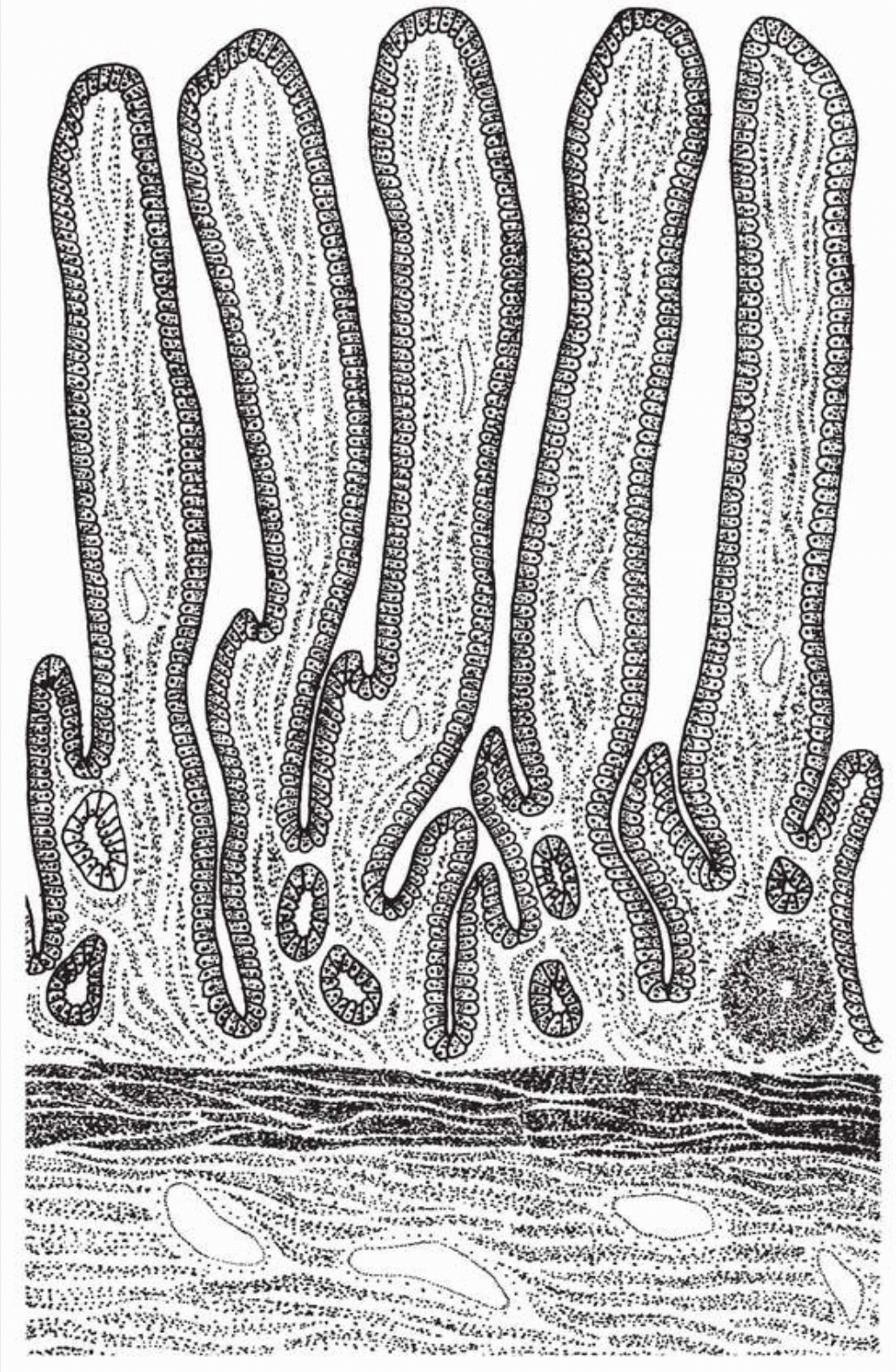
When caring for the patient with a tube:

- Explain the procedure before intubation.
- Maintain accurate intake and output records. Measure gastric drainage every 8 hours; record the amount, color, odor, and consistency. When irrigating the tube, note the amount of saline solution instilled and aspirated. Check for fluid and electrolyte imbalances.
- Provide good oral and nasal care. Brush the patient's teeth frequently and provide mouthwash. Make sure that the tube is secure, but isn't causing pressure on the nostrils. Change the tape to the nose every 24 hours. Gently wash the area around the tube, and apply a water-soluble lubricant to soften crusts. These measures help prevent sore throat and nose, dry lips, nasal excoriation, and parotitis.

PRIMARY SOURCE OF DIGESTIVE HORMONES

Pyloric mucosa

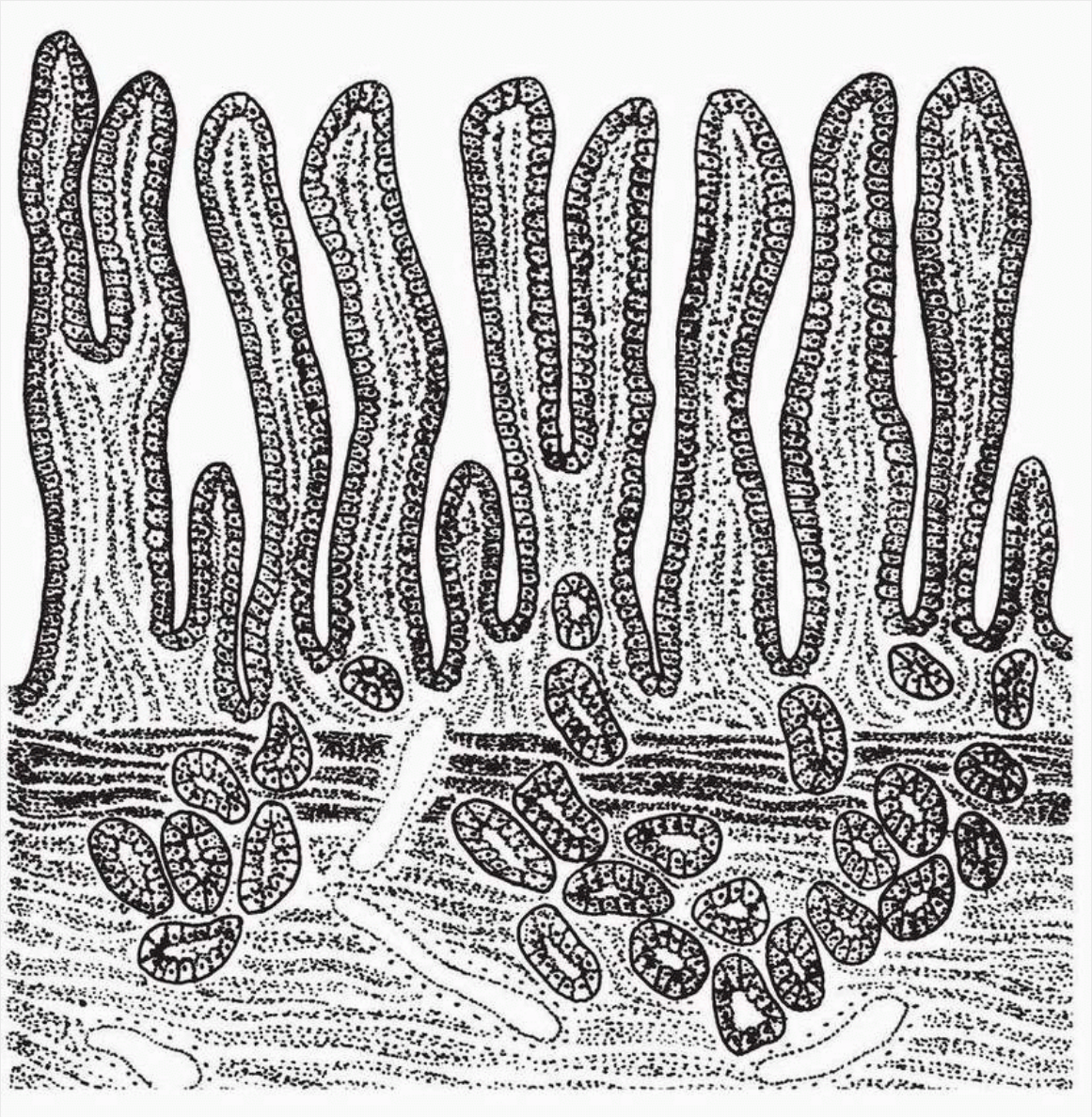
Gastrin, which originates in the G cells of the pyloric antral mucosa, stimulates secretion of hydrochloric acid by parietal cells and pepsinogen by chief cells.



Duodenal mucosa

Secretin, which originates in the duodenal mucosa, stimulates the pancreas to secrete alkaline fluid (water and bicarbonate) into the duodenum, which neutralizes acid from the stomach.

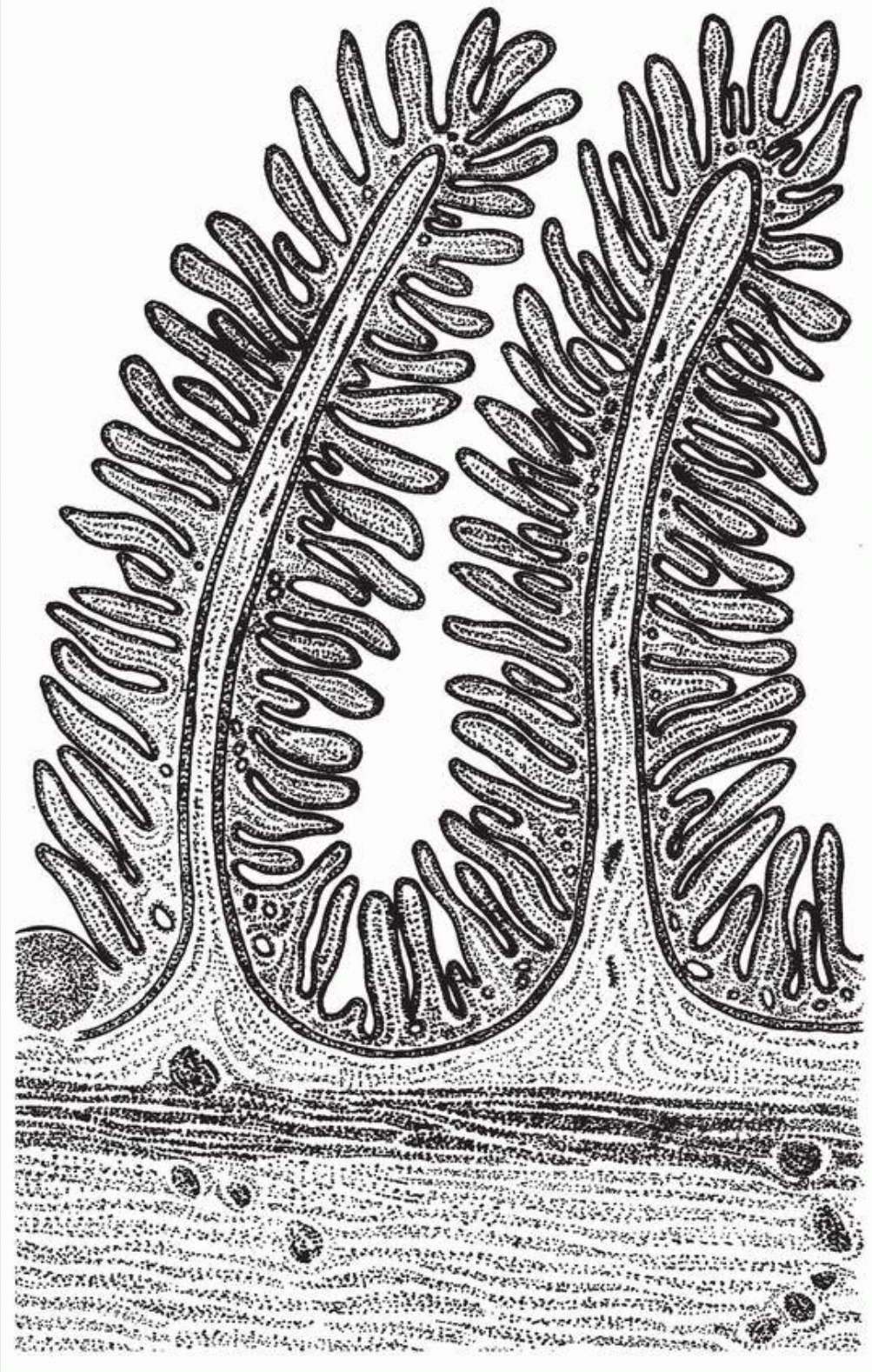
Cholecystokinin-pancreozymin, which originates in the duodenal mucosa, stimulates pancreatic enzyme secretion and contraction and evacuation of the gallbladder.



Motilin, which originates in the duodenal mucosa, slows gastric emptying and stimulates gastric acid and pepsin secretion by controlling the pattern of smooth-muscle contractions

Jejunal mucosa

Gastric inhibitory peptide (GIP), which originates in the jejunal mucosa (also in the duodenal mucosa), stimulates secretion of intestinal juice and insulin and inhibits gastric acid secretion and motility.



Other digestive hormones

Three other digestive hormonelike substances are thought to originate in the hypothalamus, GI tract, and neurons of the brain. These substances include substance P, which increases small bowel motility; bombesin, which increases gastrin secretion and small bowel motility; and somatostatin, which inhibits secretion of gastrin, vasoactive intestinal polypeptide, GIP, secretin, and motilin. Other possible digestive hormones include enterogastrone, enteroglucagon, and somatostatin. More research is needed to confirm and clarify the existence and function of these hormones.

- Ensure maximum patient comfort. After insertion of a long intestinal tube, instruct the patient to turn from side to side to facilitate its passage through the GI tract. Note the tube's progress. Never attach an intestinal tube to a patient's gown, bed linens, side rails of the bed, and so forth.
- With both types of tubes, tell the patient to expect a feeling of dryness or a lump in the throat; if he's allowed, suggest that he chew gum or eat hard candy to relieve discomfort.
- Always keep scissors taped to the wall near the bed when the patient has a Sengstaken-Blakemore tube in place. If the tube should dislodge and obstruct the bronchus, cut the lumen to the balloons immediately. Sometimes the tube is taped to the face piece of a football helmet worn by the patient to prevent the tube from dislodging and to put traction on the tube.
- After removing the tube from a patient with GI bleeding, watch for signs and symptoms of recurrent bleeding, such as hematemesis, decreased hemoglobin level, pallor, chills, diaphoresis, hypotension, and rapid pulse.
- Provide emotional support because the patient may panic at the sight of a tube. A calm, reassuring manner can help minimize his fear.

MOUTH AND ESOPHAGUS

Stomatitis and other oral infections

Stomatitis is an inflammation of the oral mucosa that may extend to the buccal mucosa, lips, and palate. It's a common infection that may occur alone or as part of a systemic disease. There are two main types: acute herpetic stomatitis and aphthous stomatitis. Acute herpetic stomatitis is usually self-limiting; however, it may be severe and, in neonates, may be generalized and potentially fatal. Aphthous stomatitis usually heals spontaneously, without a scar, in 10 to 14 days. Other oral infections include gingivitis, candidiasis, glossitis, periodontitis, and Vincent's angina. (See *Types of oral infections*.)

Causes and incidence

Acute herpetic stomatitis results from the herpes simplex virus. It's common in children ages 1 to 3. The cause of aphthous stomatitis is unknown, but predisposing factors include stress, fatigue, anxiety, febrile states, trauma, and solar overexposure. This type is common in young and teenage girls.

Complications

- Nutritional deficiencies
- Esophagitis
- Sepsis

Signs and symptoms

Acute herpetic stomatitis begins suddenly with mouth pain, malaise, lethargy, anorexia, irritability, and fever, which may persist for 1 to 2 weeks. Gums are swollen and bleed easily, and the mucous membrane is extremely tender.

Papulovesicular ulcers appear in the mouth and throat and eventually become punched-out lesions with reddened areolae. Submaxillary lymphadenitis is common. Pain usually disappears 2 to 4 days before healing of ulcers is complete. If the child with stomatitis sucks his thumb, these lesions spread to the hand.

A patient with aphthous stomatitis typically reports burning, tingling, and slight swelling of the mucous membrane. Single or multiple shallow ulcers with whitish centers and red borders appear and heal at one site and then reappear at another. (See *Looking at aphthous stomatitis*, page 240.)

Diagnosis

Diagnosis is based on the physical examination; in Vincent's angina, a smear of ulcer exudate allows for identification of the causative organism.

TYPES OF ORAL INFECTIONS

Disease and causes	Signs and symptoms	Treatment
Candidiasis *(infection of the oropharyngeal mucosa)*		
▪ Fungal infection caused by Candida albicans or related species ▪ High-risk patients include premature neonates, older adults, the immunosuppressed, and those taking antibiotics or long-term steroids	▪ cream-colored or bluish white patches of exudate on the tongue, mouth, or pharynx ▪ painful fissures at the corners of the mouth	▪ Antifungals for infection ▪ May benefit from eating active-culture yogurt or other live lactobacillus ▪ Topical anesthetic to relieve discomfort ▪ Nonirritating mouthwash to loosen tenacious secretions
Gingivitis *(inflammation of the gingiva)*		
▪ Early sign of hypovitaminosis, diabetes, blood dyscrasias ▪ Occasionally related to use of hormonal contraceptives	▪ Inflammation with painless swelling, redness, change of normal contours, bleeding, and periodontal pocket (gum detachment from teeth)	▪ Removal of irritating factors (calculus, faulty dentures)

		▪ Good oral hygiene, regular dental checkups, vigorous chewing ▪ Oral or topical corticosteroids
Glossitis *(inflammation of the tongue)*		
▪ Streptococcal infection ▪ Irritation or injury; jagged teeth; ill-fitting dentures; biting during seizures; alcohol; spicy foods; smoking; sensitivity to toothpaste or mouthwash ▪ Vitamin B deficiency; anemia ▪ Skin conditions: lichen planus, erythema multiforme, pemphigus vulgaris	▪ Reddened, ulcerated, or swollen tongue (may obstruct airway) ▪ Painful chewing and swallowing ▪ Speech difficulty ▪ Painful tongue without inflammation	▪ Treatment of underlying cause ▪ Topical anesthetic mouthwash or systemic analgesics (acetaminophen) for painful lesions ▪ Good oral hygiene, regular dental checkups, vigorous chewing ▪ Avoidance of hot, cold, or spicy foods and alcohol
Periodontitis *(gingival infection and recession, loosening of teeth)*		
▪ Early sign of hypovitaminosis, diabetes, blood dyscrasias ▪ Occasionally related to use of hormonal contraceptives ▪ Dental factors: calculus, poor oral hygiene, malocclusion; major cause of tooth loss after middle age	▪ Acute onset of bright red gum inflammation, painless swelling of interdental papillae, easy bleeding ▪ Loosening of teeth, typically without inflammatory symptoms, progressing to loss of teeth and alveolar bone ▪ Acute systemic infection (fever, chills)	▪ Scaling, root planing, and curettage for infection control ▪ Periodontal surgery to prevent recurrence ▪ Good oral hygiene, regular dental checkups, vigorous chewing
Vincent's angina *(trench mouth, necrotizing ulcerative gingivitis)*		
▪ Fusiform bacillus or spirochete	▪ Sudden onset: painful,	▪ Removal of

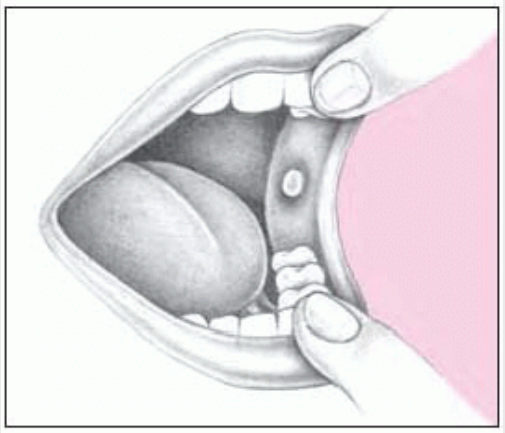
infection ▪ Predisposing factors: stress, poor oral hygiene, insufficient rest, nutritional deficiency, smoking	superficial bleeding; gingival ulcers (rarely, on buccal mucosa) covered with a gray-white membrane ▪ Ulcers become punched-out lesions after slight pressure or irritation ▪ Malaise, mild fever, excessive salivation, bad breath, pain on swallowing or talking, enlarged submaxillary lymph nodes	devitalized tissue with ultrasonic cavitron ▪ Antibiotics for infection ▪ Analgesics, as needed ▪ Hourly mouth rinses (with equal amounts of hydrogen peroxide and warm water) ▪ Soft, nonirritating diet; rest; no smoking ▪ With treatment, improvement common within 24 hours

Treatment

For acute herpetic stomatitis, treatment is conservative. For local symptoms, supportive measures include warm salt-water mouth rinses (antiseptic mouthwashes are contraindicated because they are irritating) and a topical anesthetic to relieve mouth ulcer pain. Topical antihistamines, antacids, or corticosteroids may also be recommended. Supplementary treatment includes a bland or liquid diet and, in severe cases, I.V. fluids and bed rest.

LOOKING AT APHTHOUS STOMATITIS

In aphthous stomatitis, numerous small, round vesicles appear. They soon break and leave shallow ulcers with red areolae.



For aphthous stomatitis, primary treatment is application of a topical anesthetic. Effective long-term treatment requires alleviation or prevention of precipitating factors.

Gastroesophageal reflux

Gastroesophageal reflux, also called *gastroesophageal reflux disease* (GERD), is the backflow of gastric or duodenal contents, or both, into the esophagus and past the lower esophageal sphincter (LES) without associated belching or vomiting. Reflux may cause symptoms or pathologic changes. Persistent reflux may cause reflux esophagitis (inflammation of the esophageal mucosa). Prognosis varies with the underlying cause.

Causes and incidence

The function of the LES—a high-pressure area in the lower esophagus, just above the stomach—is to prevent gastric contents from backing up into the esophagus. Normally, the LES creates pressure, closing the lower end of the esophagus, but relaxes after each swallow to allow food into the

stomach. Reflux occurs when LES pressure is deficient or when pressure within the stomach exceeds LES pressure. (See *Influences on LES pressure*.)

Studies have shown that a patient with symptom-producing reflux can't swallow often enough to create sufficient peristaltic amplitude to clear

gastric acid from the lower esophagus. This results in prolonged periods of acidity in the esophagus when reflux occurs.

Predisposing factors include:

- pyloric surgery (alteration or removal of the pylorus), which allows reflux of bile or pancreatic juice
- long-term nasogastric (NG) intubation (more than 4 days)
- any agent that lowers LES pressure, such as food, alcohol, cigarettes; anticholinergics (atropine, belladonna, and propantheline); or other drugs (morphine, diazepam, calcium channel blockers, and meperidine)
- hiatal hernia with an incompetent sphincter
- any condition or position that increases intra-abdominal pressure, such as straining, bending, coughing, pregnancy, obesity, and recurrent or persistent vomiting.

About 25% to 40% of Americans experience symptomatic GERD at some point in their lives, while 7% to 10% of Americans experience symptoms on a daily basis. True incidence figures may be even higher because many people with GERD take over-the-counter remedies without reporting their symptoms.

Studies show that GERD is common and may be overlooked in infants and children. It can cause repeated vomiting, coughing, and other respiratory problems. An immature digestive sytem is usually responsible, and most infants grow out of GERD by the time they are age 1.

Complications

- Esophageal ulcer
- Esophageal stricture
- Barrett's esophagus
- Hoarseness
- Reflux esophagitis

INFLUENCES ON LES PRESSURE

Several factors can influence lower esophageal sphincter (LES) pressure, thereby affecting reflux, as noted here.


Factors that increase LES pressure
Carbohydrates
Low-dose ethanol
Nonfat milk
Protein
Factors that decrease LES pressure
Antiflatulents (simethicone)
Chocolate
Cigarette smoking
Fat
High-dose ethanol
Lying on right or left side
Orange juice
Peppermint
Sitting
Tomatoes
Whole milk

Signs and symptoms

GERD doesn't always cause symptoms, and in patients showing clinical effects, it isn't always possible to confirm physiologic reflux. The most common feature of GERD is heartburn, which may become more severe with vigorous exercise, bending, or lying down, and may be relieved by antacids or sitting upright. The pain of esophageal spasm resulting from reflux esophagitis tends to be chronic and may mimic angina pectoris, radiating to the neck, jaws, and arms.

Other symptoms include odynophagia, which may be followed by a dull substernal ache from severe, long-term reflux; dysphagia from esophageal spasm, stricture, or esophagitis; and bleeding (bright red or dark brown). Rarely, nocturnal regurgitation wakens the patient with coughing, choking, and a mouthful of saliva. Reflux may be associated with hiatal hernia. Direct hiatal hernia becomes clinically significant only when reflux is confirmed.

Pulmonary symptoms result from reflux of gastric contents into the throat and subsequent aspiration; they include chronic pulmonary disease or nocturnal wheezing, bronchitis, asthma, morning hoarseness, and cough. In children, other signs consist of failure to thrive and forceful vomiting from esophageal irritation. Such vomiting sometimes causes aspiration pneumonia.

Diagnosis

CONFIRMING DIAGNOSIS

After a careful history and physical examination, tests to confirm GERD include barium swallow fluoroscopy, esophageal pH probe, esophageal manometry, and esophagoscopy. In children, barium esophagography under fluoroscopic control can show reflux.

Recurrent reflux after age 6 weeks is abnormal. An acid perfusion (Bernstein) test can show that reflux is the cause of symptoms. Finally, endoscopy and biopsy allow visualization and confirmation of any pathologic changes in the mucosa.

Treatment

Promotility agents help increase LES sphincter tone and stimulate upper GI motility. Proton pump inhibitors and histamine-2 (H_2) receptor antagonists help reduce gastric acidity. If possible, NG intubation shouldn't be continued for more than 5 days because the tube interferes with sphincter integrity and allows reflux, especially when the patient lies flat.

PEDIATRIC TIP

Positional therapy is especially useful in infants and children who experience GERD without complications. Strategies, such as burping the infant several times during feeding, keeping the infant in an upright position for 30 minutes after feeding, and avoiding feeding 2 to 3 hours before bedtime, may help.

Surgery may be necessary to control severe and refractory symptoms, such as pulmonary aspiration, hemorrhage, obstruction, severe pain, perforation, an incompetent LES, or associated hiatal hernia. Surgical procedures that create an artificial closure at the gastroesophageal junction may be needed in some patients. These include a procedure that invaginates the esophagus into the stomach and procedures that create a gastric wraparound with or without fixation. The fundoplication procedure can be performed endoscopically. Also, vagotomy or pyloroplasty may be combined with an antireflux regimen to modify gastric contents.

Special considerations

Teach the patient what causes reflux, how to avoid reflux with an antireflux regimen (medication, diet, and positional therapy), and what symptoms to watch for and report.

- Instruct the patient to avoid circumstances that increase intra-abdominal pressure (such as bending, coughing, vigorous exercise, tight clothing, constipation, and obesity) as well as substances that

reduce sphincter control (cigarettes, alcohol, fatty foods, and caffeine).

- Advise the patient to sit upright, particularly after meals, and to eat small, frequent meals. Tell him to avoid highly seasoned food, acidic juices, alcoholic drinks, bedtime snacks, and foods high in fat or carbohydrates, which reduce LES pressure. He should eat meals at least 2 to 3 hours before lying down.

- Tell the patient to take antacids, as ordered (usually 1 hour before or 3 hours after meals and at bedtime).
- Teach the patient correct preparation for diagnostic testing. For example, he shouldn't eat for 6 to 8 hours before a barium swallow or endoscopy.
- After surgery using a thoracic approach, carefully watch and record chest tube drainage and the patient's respiratory status. If needed, give chest physiotherapy and oxygen. Position the patient with an NG tube in semi-Fowler's position to help prevent reflux. Offer reassurance and emotional support. (See *Preventing GI reflux*.)

PREVENTION

PREVENTING GI REFLUX

Changing his lifestyle will help the patient to prevent GI reflux.

Diet and eating habits

Foods, such as caffeine, chocolate, spicy food, carbonated beverages, orange juice, liquor, wine, and tomato sauce, stimulate the production of acid. Large meals expand the stomach and put pressure on the lower esophageal sphincter (LES). Gravity helps keep the stomach juices from backing up into the esophagus: Don't allow the patient to lie down for 2 hours after eating. If nighttime heartburn is a concern, raise the head of the bed 6 to 8 inches because a flat position places pressure on the LES.

Weight

Being overweight increases abdominal pressure, which can then push stomach contents up into the esophagus.

Smoking

Nicotine relaxes the esophageal sphincter and stimulates the production of stomach acid. Smoking also may injure the esophagus by causing irritation making it more susceptible to damage from acid reflux. Smoking can decrease gastric motility and reduce the effectiveness of digestion because the stomach takes longer to empty.

Stress

Although stress itself doesn't cause heartburn, the anxiety that comes along with stress can lead to behaviors that increase the risk of heartburn, such as overeating, drinking, and smoking.

Alcohol

Alcohol can increase the production of stomach acid and can also lower the esophageal sphincter, which allows stomach acids to move up into the esophagus. Alcohol also makes the esophagus more sensitive to stomach acid.

Tracheoesophageal fistula and esophageal atresia

Tracheoesophageal fistula is a developmental anomaly characterized by an abnormal connection between the trachea and the esophagus. It usually accompanies esophageal atresia, in which the esophagus is closed off at some point. Although these malformations have numerous anatomic variations, the most common, by far, is esophageal atresia with fistula to the distal segment. (See *Types of tracheoesophageal anomalies*, pages 244 and 245.)

These disorders, two of the most serious surgical emergencies in neonates, require immediate diagnosis and correction. They may coexist

with other serious anomalies, such as congenital heart disease, imperforate

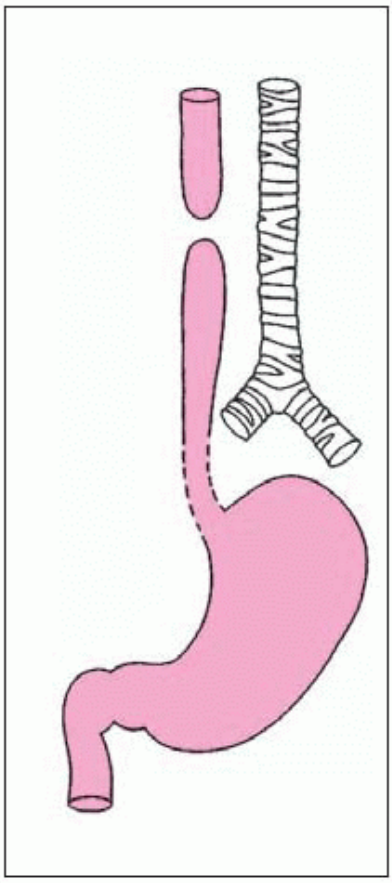
anus, genitourinary abnormalities, and intestinal atresia.

TYPES OF TRACHEOESOPHAGEAL ANOMALIES

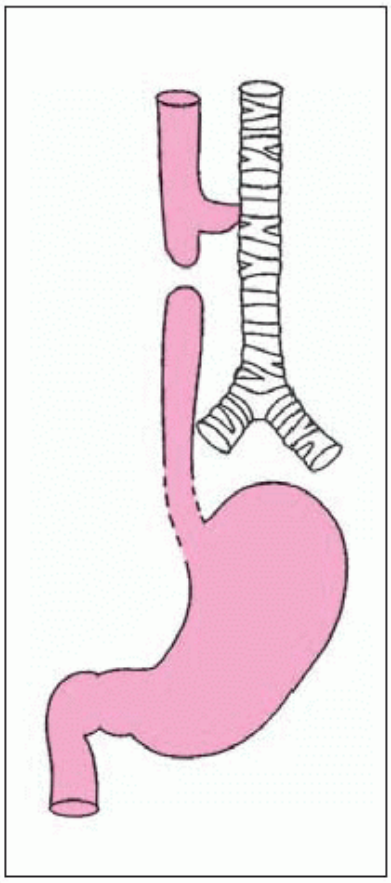
Congenital malformations of the esophagus occur in about 1 in 4,000 live births. The American Academy of Pediatrics classifies the anatomic variations of tracheoesophageal anomalies as follows.

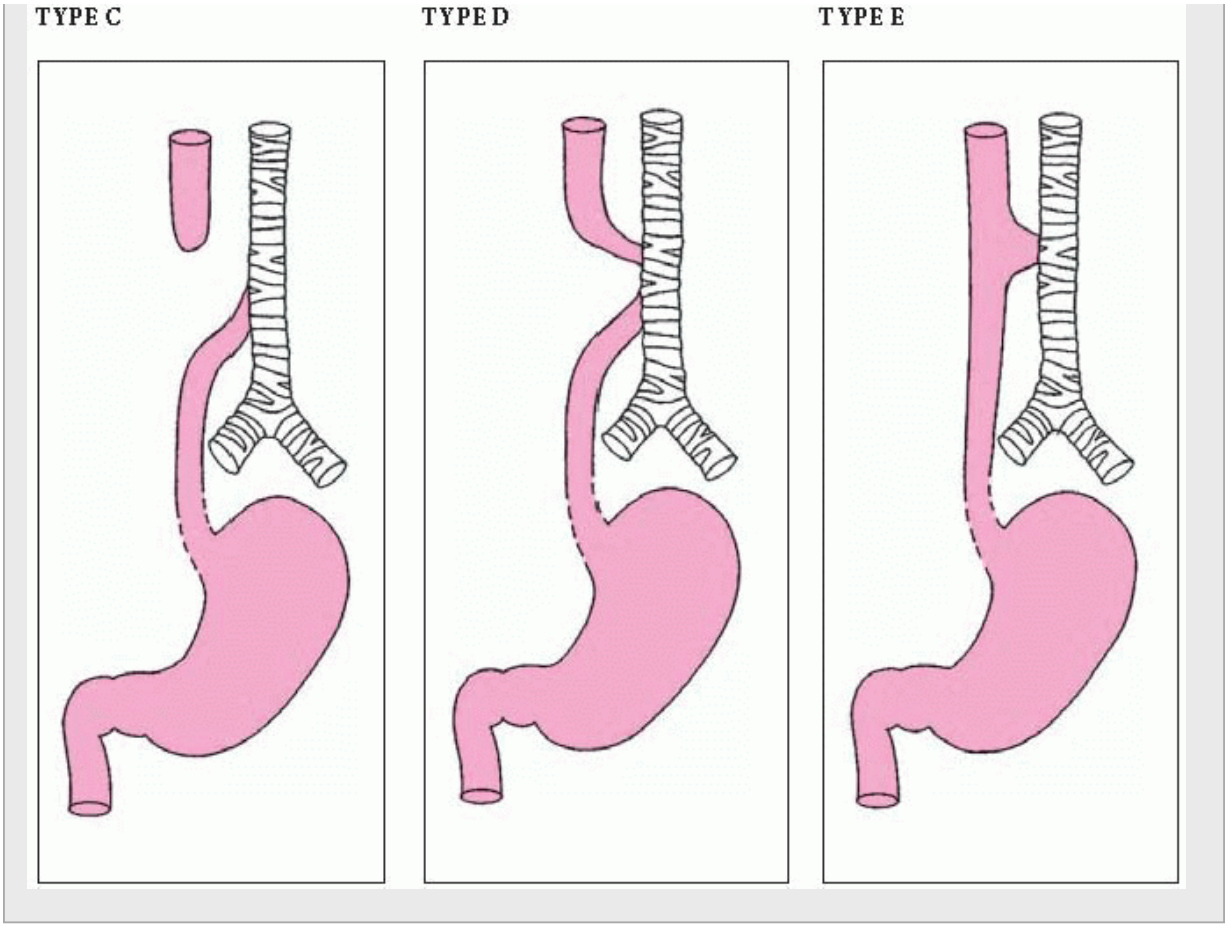
- ▪ Type A (7.7%): esophageal atresia without fistula
- ▪ Type B (0.8%): esophageal atresia with tracheoesophageal fistula to the proximal segment
- ▪ Type C (86.5%): esophageal atresia with fistula to the distal segment
- ▪ Type D (0.7%): esophageal atresia with fistula to both segments
- ▪ Type E (or H-Type) (4.2%): tracheoesophageal fistula without atresia

TYPE A



TYPE B





Causes and incidence

Tracheoesophageal fistula and esophageal atresia result from failure of the embryonic esophagus and trachea to develop and separate correctly. Respiratory system development begins at about day 26 of gestation. Abnormal development of the septum during this time can lead to tracheoesophageal fistula. The most common abnormality is type C tracheoesophageal fistula with esophageal atresia, in which the upper section of the esophagus terminates in a blind pouch, and the lower section ascends from the stomach and connects with the trachea by a short fistulous tract.

In type A atresia, both esophageal segments are blind pouches, and neither is connected to the airway. In type E (or H-type), tracheoesophageal fistula without atresia, the fistula may occur anywhere between the level of the cricoid cartilage and the midesophagus, but is usually higher in the trachea than in the esophagus. Such a fistula may be as small as a pinpoint. In types B and

D, the upper portion of the esophagus opens into the trachea; neonates with this anomaly may experience life-threatening aspiration of saliva or food.

Esophageal atresia occurs in about 1 of every 1,500 to 3,000 live births; about one-third of these neonates are born prematurely.

Complications

- Recurrent fistulas
- Abnormal esphageal motility
- Pneumothorax
- Esophageal stricture

Signs and symptoms

A neonate with type C tracheoesophageal fistula with esophageal atresia appears to swallow normally but soon after swallowing

coughs, struggles, becomes cyanotic, and stops breathing as he aspirates fluids returning from the blind pouch of the esophagus through his nose and mouth. Stomach distention may cause respiratory distress; air and gastric contents (bile and gastric secretions) may reflux through the fistula into the trachea, resulting in chemical pneumonitis.

An infant with type A esophageal atresia appears normal at birth. The infant swallows normally, but as secretions fill the esophageal sac and overflow into the oropharynx, he develops mucus in the oropharynx and drools excessively. When the infant is fed, regurgitation and respiratory distress follow aspiration. Suctioning the mucus and secretions temporarily relieves these symptoms. Excessive secretions and drooling in the neonate strongly suggest esophageal atresia.

Repeated episodes of pneumonitis, pulmonary infection, and abdominal distention may signal type E (or H-type) tracheoesophageal fistula. When a child with this disorder drinks, he coughs, chokes, and becomes cyanotic. Excessive mucus builds up in the oropharynx. Crying forces air from the trachea into the esophagus, producing abdominal distention. Because such a child may appear normal at birth, this type of

tracheoesophageal fistula may be overlooked, and diagnosis may be delayed as long as 1 year.

Type B (proximal fistula) and type D (fistula to both segments) cause immediate aspiration of saliva into the airway and bacterial pneumonitis.

Diagnosis

Respiratory distress and drooling in a neonate suggest tracheoesophageal fistula and esophageal atresia. The following procedures confirm the diagnosis:

- A size 10 or 12 French catheter passed through the nose meets an obstruction (esophageal atresia) approximately 4″ to 5″ (10 to 12.5 cm) distal from the nostrils. Aspirate of gastric contents is less acidic than normal.
- Chest X-ray demonstrates the position of the catheter and can also show a dilated, air-filled upper esophageal pouch, pneumonia in the right upper lobe, or bilateral pneumonitis. Both pneumonia and pneumonitis suggest aspiration.
- Abdominal X-ray shows gas in the bowel in a distal fistula (type C) but none in a proximal fistula (type B) or in atresia without fistula (type A).
- Cinefluorography allows visualization on a fluoroscopic screen. After a size 10 or 12 French catheter is passed through the patient's nostril into the esophagus, a small amount of contrast medium is instilled to define the tip of the upper pouch and to differentiate between overflow aspiration from a blind end (atresia) and aspiration due to passage of liquid through a tracheoesophageal fistula.

Treatment

Tracheoesophageal fistula and esophageal atresia require surgical correction and are usually surgical emergencies. The type and timing of surgical procedure depend on the nature of the anomaly, the patient's general condition, and the presence of coexisting congenital defects. In premature neonates (nearly 33% of neonates with this anomaly are born

prematurely) who are poor surgical risks, correction of combined tracheoesophageal fistula and esophageal atresia is done in two stages: first, gastrostomy (for gastric decompression, prevention of reflux, and feeding) and closure of the fistula; then, 1 to 2 months later, anastomosis of the esophagus.

Before and after surgery, positioning varies with the physician's philosophy and the infant's anatomy: the infant may be placed supine, with his head low to facilitate drainage, or with his head elevated to prevent aspiration.

The infant should receive I.V. fluids, as necessary, and appropriate antibiotics for superimposed infection.

Postoperative complications after correction of tracheoesophageal fistula include recurrent fistulas, esophageal motility dysfunction, esophageal stricture, recurrent bronchitis, pneumothorax, and failure to thrive. Esophageal motility dysfunction or hiatal hernia may develop after surgery to correct esophageal atresia.

Correction of esophageal atresia alone requires anastomosis of the proximal and distal esophageal segments in one or two stages. End-to-end anastomosis commonly produces postoperative stricture; end-toside anastomosis is less likely to do so. If the esophageal ends are widely separated, treatment may include a colonic interposition (grafting a piece of the colon) or elongation of the proximal segment of the esophagus by bougienage. About 10 days after surgery, and again 1 and 3 months later, X-rays are required to evaluate the effectiveness of surgical repair.

Postoperative treatment includes placement of a suction catheter in the upper esophageal pouch to control secretions and prevent aspiration, maintaining the infant in an upright position to avoid reflux of gastric juices into the trachea, I.V. fluids (nothing by mouth), gastrostomy to prevent reflux and allow feeding, and appropriate antibiotics for pneumonia.

Postoperative complications may include impaired esophageal motility (in one-third of patients), hiatal hernia, and reflux esophagitis.

Special considerations

Postoperative care should include the following:

- Monitor the infant's respiratory status. Administer oxygen and perform pulmonary physiotherapy and suctioning, as needed. Provide a humid environment.
- Administer antibiotics and parenteral fluids, as ordered. Keep accurate intake and output records.
- If the infant has chest tubes postoperatively, check them frequently for patency. Maintain proper suction; measure and mark drainage periodically.
- Observe carefully for signs of complications.
- Maintain gastrostomy tube feedings, as ordered. Such feedings initially consist of dextrose and water (not more than 5% solution); later, add a proprietary formula (first diluted and then full strength). If the infant develops gastric atony, use an isoosmolar formula. Oral feedings can usually resume 8 to 10 days postoperatively. If gastrostomy feedings and oral feedings are impossible because of intolerance to them or decreased intestinal motility, the infant requires total parenteral nutrition.
- If the infant can safely handle secretions, he may be given a pacifier to satisfy his sucking needs; however, this is done *only* when he can safely handle secretions because sucking stimulates saliva secretion.
- Offer the parents support and guidance in dealing with their infant's acute illness. Encourage them to participate in the infant's care and to hold and touch him as much as possible to facilitate bonding.

Corrosive esophagitis and stricture

Corrosive esophagitis is inflammation and damage to the esophagus after ingestion of a caustic chemical. Similar to a burn, this injury may be temporary or may lead to permanent stricture (narrowing or stenosis) of the esophagus that's correctable only through surgery. Severe injury can quickly lead to esophageal perforation, mediastinitis, and death from infection, shock, and massive hemorrhage (due to aortic perforation).

Causes and incidence

The most common chemical injury to the esophagus follows the ingestion of lye or other strong alkali; ingestion of strong acids is less common. The type and amount of chemical ingested determine the severity and location of the damage. In children, household chemical ingestion is accidental; in adults, it's usually a suicide attempt or gesture. The chemical may damage only the mucosa or submucosa or it may damage all layers of the esophagus.

Esophageal tissue damage occurs in three phases: the acute phase, consisting of edema and inflammation; the latent phase, with ulceration, exudation, and tissue sloughing; and the chronic phase, in which there is diffuse scarring.

Gastroesophageal reflux disease accounts for 70% to 80% of all cases of esophageal stricture. Postoperative strictures account for 10% of all cases, and corrosive strictures account for less than 5% of all cases. Peptic strictures are 10 times more common in Whites than in Blacks and Asians and two to three times more common in men than in women.

Complications

- Esophageal perforation
- Mediastinitis
- Infection, massive hemorrhage, shock

Signs and symptoms

Effects vary from none at all to intense pain and edema n the mouth, anterior chest pain, marked salivation, inability to swallow, and tachypnea. Bloody vomitus containing pieces of esophageal tissue signals severe damage. Signs of esophageal perforation and mediastinitis, especially crepitation, indicate destruction of the entire esophagus. Inability to speak implies laryngeal damage.

The acute phase subsides in 3 to 4 days, enabling the patient to eat again. Fever suggests secondary infection. Symptoms of dysphagia return

if stricture develops, usually within weeks; rarely, stricture is delayed and develops several years after the injury.

Diagnosis

CONFIRMING DIAGNOSIS

A history of chemical ingestion and physical examination revealing oropharyngeal burns (including white membranes and edema of the soft palate and uvula) usually confirm the diagnosis.

The type and amount of the chemical ingested must be identified; this may be done by examining the container of the ingested material or by calling the poison control center.

Two procedures are helpful in evaluating the severity of the injury:

- Endoscopy (in the first 24 hours after ingestion) delineates the extent and location of the esophageal injury and assesses the depth of the burn. This procedure may also be performed a week after ingestion to assess stricture development.
- Barium swallow (1 week after ingestion and every 3 weeks thereafter) may identify

  segmental spasm or fistula, but doesn't always show mucosal injury.

Treatment

Conservative treatment of corrosive esophagitis and stricture includes monitoring the patient's condition; early endoscopy; administering corticosteroids, such as prednisone and hydrocortisone, to control inflammation and inhibit fibrosis; and using a broad-spectrum antibiotic, such as ampicillin, to protect the corticosteroidimmunosuppressed patient against infection by his own mouth flora.

Treatment may also include bougienage, a procedure in which a slender, flexible, cylindrical instrument called a *bougie* is passed into the esophagus to dilate it and minimize stricture. Some physicians begin bougienage immediately and continue it regularly to maintain a patent

lumen and prevent stricture; others delay it for a week to avoid the risk of esophageal perforation.

Surgery is needed immediately for esophageal perforation or later to correct stricture untreatable with bougienage. Corrective surgery may involve transplanting a piece of the colon to the damaged esophagus. However, even after surgery, stricture may recur at the site of the anastomosis.

Supportive treatment includes I.V. therapy to replace fluids or total parenteral nutrition while the patient can't swallow, gradually progressing to clear liquids and a soft diet.

Special considerations

If you're the first health care professional to see the patient who has ingested a corrosive chemical, the quality of your emergency care will be critical. To meet this challenge, follow these important guidelines:

- *Don't* induce vomiting or lavage because this will expose the esophagus and oropharynx to additional injury.
- *Don't* perform gastric lavage because the corrosive chemical may cause further damage to the mucous membrane of the GI lining.
- Provide vigorous support of vital functions, as needed, such as oxygen, mechanical ventilation, administration of I.V. fluids, and treatment for shock, depending on the severity of the injury.
- Carefully observe and record intake and output.
- Before X-rays and endoscopy, explain the procedure to the patient to lessen anxiety during the tests and to obtain cooperation.
- Because the adult who has ingested a corrosive agent has usually done so with suicidal intent, assist him and his family in seeking psychological counseling. Monitor the patient according to facility protocol if the attempt was a suicide.
- Provide emotional support for parents whose child has ingested a chemical. They'll be distraught and may feel guilty about the accident.

- Encourage long-term follow-up because of the increased risk of squamous cell carcinoma.

PREVENTION

Tell parents to lock accessible cabinets and keep all corrosive agents out of a child's reach without emphasizing blame.

Mallory-Weiss syndrome

Mallory-Weiss syndrome is mild to massive, usually painless bleeding due to a tear in the mucosa or submucosa of the cardia or lower esophagus. Such a tear, usually singular and longitudinal, results from prolonged or forceful vomiting. Sixty percent of these tears involve the cardia; 15%, the terminal esophagus; and 25%, the region across the esophagogastric junction.

Causes and incidence

Forceful or prolonged vomiting can cause esophageal tearing when the upper esophageal sphincter fails to relax during vomiting; this lack of sphincter coordination seems more common after excessive alcohol intake. Other factors that can increase intra-abdominal pressure and predispose a person to this type of tear include coughing, straining during bowel movements, traumatic injury, seizures, childbirth, hiatal hernia, esophagitis, gastritis, and atrophic gastric mucosa.

Mallory-Weiss syndrome accounts for 1% to 15% of all cases of upper GI bleeding. It's two to four times more common in men than in women. There's no racial predilection. Patients usually present with symptoms during their 40s and 50s, but it can affect people of all ages.

Complications

- Hypovolemia (if bleeding is excessive)
- Fatal shock

Signs and symptoms

Mallory-Weiss syndrome typically begins with vomiting of blood or passing large amounts of blood rectally a few hours to several days after forceful vomiting. The bleeding, which may be accompanied by epigastric or back pain, may range from mild to massive, but is usually more profuse than in esophageal rupture. In Mallory-Weiss syndrome, the blood vessels are only partially severed, preventing retraction and closure of the lumen.

ALERT

Massive bleeding—most likely when the tear is on the gastric side, near the cardia—may quickly lead to fatal shock.

Diagnosis

CONFIRMING DIAGNOSIS

Fiberoptic endoscopy (esophagogastroduodenoscopy) confirms Mallory-Weiss syndrome by identifying esophageal tears. Recent tears appear as erythematous longitudinal cracks in the mucosa; older tears appear as raised white streaks surrounded by erythema.

Treatment

Treatment varies with the severity of bleeding. GI bleeding usually stops spontaneously, thereafter requiring supportive measures and careful observation but no definitive treatment. However, if bleeding continues, treatment may include:

- proton pump inhibitors or histamine-2 receptor antagonists to help decrease acidity
- blood transfusions if blood loss is great
- endoscopy with electrocoagulation or heater probe for hemostasis

- transcatheter embolization or thrombus formation with an autologous blood clot or other hemostatic material (such as a shredded adsorbable gelatin sponge)
- surgery to suture each esophageal laceration.

Special considerations

Observation is necessary to determine whether bleeding is transitory or ongoing.

- Evaluate the patient's respiratory status, monitor arterial blood gas values, and administer oxygen as necessary.
- Assess the amount of blood lost and record the color, amount, consistency, and frequency of hematemesis and melena.
- Draw blood for coagulation studies (prothrombin time, partial thromboplastin time, and platelet count), and type and crossmatch.
- Try to keep 3 units of blood available at all times. Insert a 14G to 18G I.V. line, and start an infusion of I.V. solution, as ordered. (If the I.V. infusion is for blood transfusion, use normal saline solution; if the infusion is for fluid replacement, use lactated Ringer's solution or another appropirate solution, depending on the results of laboratory tests.)
- Monitor the patient's vital signs, central venous pressure, urine output, neurologic status, and overall clinical status.
- Explain diagnostic tests to the patient.
- Keep the patient warm and maintain a safe environment.
- Obtain a detailed history of recent medications taken, dietary habits, and alcohol ingestion.
- Administer antiemetics, as ordered, to prevent postoperative retching and vomiting.
- Advise the patient to avoid aspirin, alcohol, and other irritating substances.

Esophageal diverticula

Esophageal diverticula are hollow outpouchings of one or more layers of the esophageal wall. They occur in three main areas: just above the upper esophageal sphincter (Zenker's, or pulsion, diverticulum, the most common type); near the midpoint of the esophagus (traction); and just above the lower esophageal sphincter (epiphrenic). Generally, esophageal diverticula occur later in life—although they can affect infants and children—and are

three times more common in men than in women. Epiphrenic diverticula usually occur in middle-aged men, whereas Zenker's diverticula typically affect men older than age 60.

Causes and incidence

Esophageal diverticula are due to primary muscular abnormalities that may be congenital or to inflammatory processes adjacent to the esophagus. Zenker's diverticulum occurs when the pouch results from increased intraesophageal pressure; traction diverticulum occurs when the pouch is pulled out by adjacent inflamed tissue or lymph nodes. Some authorities classify all diverticula as traction diverticula.

Zenker's diverticulum results from developmental muscular weakness of the posterior pharynx above the border of the cricopharyngeal muscle. The pressure of swallowing aggravates this weakness, as does contraction of the pharynx before relaxation of the sphincter. A midesophageal (traction) diverticulum is a response to scarring and pulling on esophageal walls by an external inflammatory process such as tuberculosis. An epiphrenic diverticulum (rare) is generally right-sided and usually accompanies an esophageal motor disturbance, such as esophageal spasm or achalasia. It's thought to be caused by traction and pulsation.

Most diverticula occur in middle-aged and elderly patients. Zenker's diverticula most commonly in patients older than age 50 and are especially prevalent in patients in their 70s and 80s.

Complications

- Malnutrition

- Dehydration

Signs and symptoms

Midesophageal and epiphrenic diverticula with an associated motor disturbance (achalasia or spasm) seldom produce symptoms, although the patient may experience dysphagia and heartburn. Zenker's diverticulum, however, produces distinctly staged symptoms, beginning with initial throat irritation followed by dysphagia and near-complete obstruction. In early stages, regurgitation occurs soon after eating; in later stages, regurgitation after eating is delayed and may even occur during sleep, leading to food aspiration and pulmonary infection.

ELDER TIP

Hoarseness, asthma, and pneumonitis may be the only signs of esophageal diverticula in elderly patients.

Other signs and symptoms include noise when liquids are swallowed, chronic cough, hoarseness, a bad taste in the mouth or foul breath and, rarely, bleeding.

Diagnosis

CONFIRMING DIAGNOSIS

X-rays taken following a barium swallow usually confirm the diagnosis by showing characteristic outpouching.

Esophagoscopy can rule out another lesion; however, the procedure risks rupturing the diverticulum by passing the scope into it rather than into the lumen of the esophagus, a special danger with Zenker's diverticulum.

Treatment

Treatment of Zenker's diverticulum is usually palliative and includes a bland diet, thorough chewing, and drinking water after eating to flush out the sac. However, severe symptoms or a large diverticulum necessitates surgery to remove the sac or facilitate drainage. An esophagomyotomy may be necessary to prevent recurrence. Endscopic

stapling or laser surgery using CO_2 microscopy are commonly performed in patients who can't tolerate traditional surgeries.

A midesophageal diverticulum seldom requires therapy except when esophagitis aggravates the risk of rupture, in which case treatment includes antacids and an antireflux regimen: keeping the head elevated, maintaining an upright position for 2 hours after eating, eating small meals, controlling chronic coughing, and avoiding constrictive clothing.

Epiphrenic diverticulum requires treatment of accompanying motor disorders. Achalasia is treated by repeated dilations of the esophagus; acute spasm is controlled by anticholinergic administration and diverticulum excision; and dysphagia or severe pain are relieved by surgical excision or

suspending the diverticulum to promote drainage. Treatment may also include parenteral feeding to improve the patient's nutritional status.

Special considerations

Care includes documenting the patient's symptoms and nutritional status and educating him about the disorder.

- Regularly assess the patient's nutritional status (weight, calorie intake, and appearance).
- If the patient regurgitates food and mucus, protect against aspiration by positioning him carefully (head elevated or turned to one side). To prevent aspiration, tell the patient to empty any visible outpouching in the neck by massage or postural drainage before retiring.
- If the patient has dysphagia, record welltolerated foods and what circumstances ease swallowing. Provide a pureed diet, with vitamin or protein supplements, and encourage thorough chewing.
- Teach the patient about this disorder. Explain treatment instructions and diagnostic procedures.

Hiatal hernia

Hiatal hernia, also called *hiatus hernia,* is a defect in the diaphragm that permits a portion of the stomach to pass through the diaphragmatic

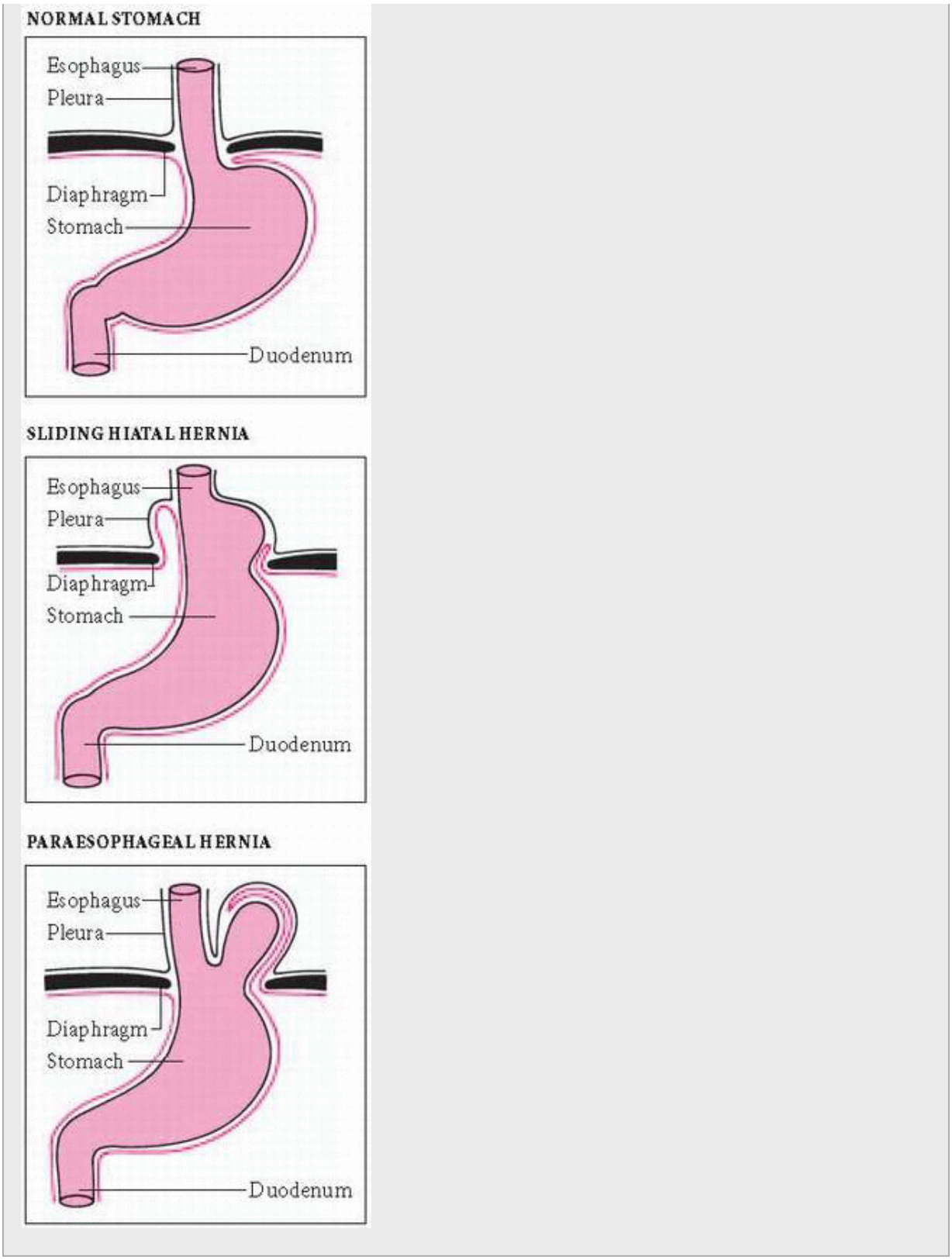
opening into the chest. Hiatal hernia is the most common problem of the diaphragm affecting the alimentary canal. Two types of hiatal hernia can occur: sliding hernia and paraesophageal hernia. (See *Types of hiatal hernia*.) In a sliding hernia, the stomach and the gastroesophageal junction slip up into the chest, so the gastroesophageal junction is above the diaphragmatic hiatus. In paraesophageal hernia, a part of the greater curvature of the stomach rolls through the diaphragmatic defect. Treatment can prevent complications such as strangulation of the herniated intrathoracic portion of the stomach.

Causes and incidence

Hiatal hernia typically results from muscle weakening that's common with aging and may be secondary to esophageal carcinoma, kyphoscoliosis, trauma, or certain surgical procedures. It may also result from certain diaphragmatic malformations that may cause congenital weakness. Obesity and smoking are common risk factors.

TYPES OF HIATAL HERNIA

A hiatal hernia is a displacement of the normal anatomy, as shown in the illustrations below.



In hiatal hernia, the muscular collar around the esophageal and diaphragmatic junction loosens, permitting the lower portion of the

esophagus and the stomach to rise into the chest when intra-abdominal pressure increases (possibly causing gastroesophageal reflux). Such increased intra-abdominal pressure may result from ascites, pregnancy, obesity, constrictive clothing, bending, straining, coughing, Valsalva's maneuver, or extreme physical exertion.

Sliding hernias are more common than paraesophageal hernias. The incidence of hiatal hernia increases with age (most occur in people older than age 40), and prevalence is higher in women than in men (especially the paraesophageal type). Contributing factors include obesity and trauma. No racial predilection exists.

Complications

- Dysphagia
- Gastroesophageal reflux
- Barrett's esophagus
- Esophageal adenocarcinoma

Signs and symptoms

Typically, a paraesophageal hernia produces no symptoms; it's usually an incidental finding during a barium swallow or when testing for occult blood. Because this type of hernia leaves the closing mechanism of the cardiac sphincter unchanged, it rarely causes acid reflux or reflux esophagitis. Symptoms result from displacement or stretching of the stomach and may include a feeling of fullness in the chest or pain resembling angina pectoris. Even if it produces no symptoms, this type of hernia needs surgical treatment because of the high risk of strangulation that can occur when a large portion of stomach becomes caught above the diaphragm.

A sliding hernia without an incompetent sphincter produces no reflux or symptoms and, consequently, doesn't require treatment. When a sliding hernia causes symptoms, they are typical of gastric reflux, resulting from the incompetent lower esophageal sphincter (LES), and may include the following:

- Pyrosis (heartburn) occurs 1 to 4 hours after eating (especially overeating) and is aggravated by reclining, belching, and increased intra-abdominal pressure. It may be accompanied by regurgitation or vomiting.
- Retrosternal or substernal chest pain results from reflux of gastric contents, stomach distention, and spasm or altered motor activity. Chest pain usually occurs after meals or at bedtime and is aggravated by reclining, belching, and increased intra-abdominal pressure.

Other common symptoms reflect possible complications:

- Dysphagia occurs when the hernia produces esophagitis, esophageal ulceration, or stricture, especially with ingestion of very hot or cold foods, alcoholic beverages, or a large amount of food.
- Bleeding may be mild or massive, frank or occult; the source may be esophagitis or erosions of the gastric pouch.
- Severe pain and shock result from incarceration, in which a large portion of the stomach is caught above the diaphragm (usually occurs with paraesophageal hernia). Incarceration may lead to perforation of the gastric ulcer and strangulation and gangrene of the herniated portion of the stomach. It requires immediate surgery.

Diagnosis

Diagnosis of hiatal hernia is based on typical clinical features and on the results of these laboratory studies and procedures:

- In barium study, hernia may appear as an outpouching containing barium at the lower end of the esophagus. Small hernias, however, are difficult to recognize. This study also shows diaphragmatic abnormalities.
- Endoscopy (esophagogastroduodenoscopy) and biopsy differentiate among hiatal hernia, varices, and other small gastroesophageal lesions; identify the mucosal junction and the edge of the diaphragm indenting the esophagus; and can rule out malignancy that otherwise may be difficult to detect.

- Esophageal motility studies assess the presence of esophageal motor abnormalities before surgical repair of the hernia.
- pH studies assess for reflux of gastric contents.

Treatment

The primary goals of treatment are to relieve symptoms by minimizing or correcting the incompetent cardia and to manage and prevent complications. Medical therapy is used first because symptoms usually respond to it and because hiatal hernia tends to recur after surgery. Such therapy attempts to modify or reduce reflux by changing the quantity or quality of refluxed gastric contents, by strengthening the LES muscle pharmacologically, or by decreasing the amount of reflux through gravity. These measures include restricting any activity that raises intra-abdominal pressure (coughing, straining, or bending), giving antiemetics, avoiding constrictive clothing, modifying diet, giving stool softeners or laxatives to prevent straining at stool, and discouraging smoking because it stimulates gastric acid production.

Modifying the diet means eating small, frequent, bland meals at least 2 hours before lying down (no bedtime snack), eating slowly, and avoiding spicy foods, fruit juices, alcoholic beverages, and coffee. Antacids also modify the fluid refluxed into the esophagus and are probably the best treatment for intermittent reflux.

To reduce the amount of reflux, the overweight patient should lose weight to decrease intra-abdominal pressure. Elevating the head of the bed 6″ (15 cm) reduces gastric reflux by gravity.

Drug therapy to strengthen cardiac sphincter tone may include a cholinergic agent or a GI stimulant to enhance smooth-muscle contraction, increase cardiac sphincter tone, and decrease reflux after eating.

Surgical repair is necessary when symptoms can't be controlled medically or with the onset of complications, such as stricture, bleeding, pulmonary aspiration, strangulation, or incarceration. Surgery typically involves creating an artificial closing mechanism at the gastroesophageal junction to strengthen the LES's barrier function. The surgeon may use an abdominal or a thoracic approach or he may repair the hernia by

laparoscopic surgery, which allows for less dependence on a nasogastric (NG) tube and a shorter hospital stay.

Special considerations

To enhance compliance with treatment, teach the patient about this disorder. Explain treatments, diagnostic tests, and significant symptoms.

- Prepare the patient for diagnostic tests as needed. After endoscopy, watch for signs of perforation (falling blood pressure, rapid pulse, shock, and sudden pain).
- If surgery is scheduled, review preoperative and postoperative considerations with the patient.
- After surgery, carefully record intake and output, including NG tube and wound drainage.
- While the NG tube is in place, provide meticulous mouth and nose care, but don't manipulate the tube. Give ice chips, if permitted, to moisten oral mucous membranes.
- If the surgeon used a thoracic approach, the patient may have chest tubes in place. Carefully observe chest tube drainage and the patient's respiratory status, and perform pulmonary physiotherapy.
- Before discharge, tell the patient what foods he can eat, and recommend small, frequent meals. Warn against activities that cause increased intra-abdominal pressure, and advise a slow return to normal functions (within 6 to 8 weeks).

STOMACH, INTESTINE, AND PANCREAS

Gastritis

Gastritis, an inflammation of the gastric mucosa, may be acute or chronic. Acute gastritis produces mucosal reddening, edema, hemorrhage, and erosion. Chronic gastritis is common among elderly people and people with pernicious anemia. It typically occurs as chronic atrophic gastritis, in which all stomach mucosal layers are inflamed, with reduced numbers of chief and parietal cells. Acute or chronic

gastritis can occur at any age.

Causes and incidence

Acute gastritis has numerous causes, including:

- chronic ingestion of (or an allergic reaction to) irritating foods or beverages, such as hot peppers or alcohol
- drugs, such as aspirin and other nonsteroidal anti-inflammatory agents (in large doses), cytotoxic agents, corticosteroids, antimetabolites, phenylbutazone, and indomethacin
- ingestion of poisons, especially DDT, ammonia, mercury, carbon tetrachloride, and corrosive substances
- endotoxins released from infecting bacteria, such as staphylococci, *Escherichia coli,* or salmonella.

Acute gastritis leading to stress ulcers also may develop in acute illnesses, especially when the patient has had major traumatic injuries; burns; severe infection; hepatic, renal, or respiratory failure; or major surgery.

Chronic gastritis may be associated with peptic ulcer disease or gastrostomy, both of which cause chronic reflux of pancreatic secretions, bile, and bile acids from the duodenum into the stomach. Recurring exposure to irritating substances, such as drugs, alcohol, cigarette smoke, or environmental agents, may also lead to chronic gastritis. Chronic gastritis may occur with pernicious anemia, renal disease, or diabetes mellitus. Pernicious anemia is commonly associated with atrophic gastritis, a chronic inflammation of the stomach resulting from degeneration of the gastric mucosa. In pernicious anemia, the stomach can no longer secrete intrinsic factor, which is needed for vitamin B_{12} absorption.

Bacterial infection with *Helicobacter pylori* is a common cause of nonerosive chronic gastritis. About 35% of adults are infected with *H. pylori,* but the prevalence of *H. pylori* infection in minority groups and in immigrants is much higher. Children ages 2 to 8 in developing nations acquire the infection at a rate of 10% per year; in the United States, the rate of yearly infection is less than 1%.

Complications

- Hemorrhage
- Shock
- Obstruction
- Perforation
- Peritonitis
- Gastric cancer

Signs and symptoms

After exposure to the offending substance, the patient with acute gastritis typically reports a rapid onset of symptoms, such as epigastric discomfort, indigestion, cramping, anorexia, nausea, vomiting, and hematemesis. The symptoms last from a few hours to a few days.

The patient with chronic gastritis may describe similar symptoms or may have only mild epigastric discomfort, or his complaints may be vague, such as an intolerance for spicy or fatty foods or slight pain relieved by eating. The patient with chronic atrophic gastritis may be asymptomatic.

Diagnosis

CONFIRMING DIAGNOSIS

Esophagogastroduodenoscopy or gastroscopy (with biopsy) confirms gastritis when done before lesions heal (usually within 24 hours). This test is contraindicated after ingestion of a corrosive agent.

Laboratory analyses can detect occult blood in vomitus or stool (or both) if the patient has gastric bleeding. Hemoglobin level and hematocrit are decreased if the patient has developed anemia from bleeding.

Treatment

Treatment for gastritis focuses on eliminating the cause; for example, bacterial gastritis is treated with antibiotics, whereas gastritis caused by ingested poison is treated by neutralizing the poison with the appropriate antidote. Histamine-2 (H_2) receptor antagonists may block gastric secretions. Many over-the-counter preparations are available. Antacids may be used as buffers.

For critically ill patients, antacids administered hourly, with or without H_2-receptor

antagonists, may reduce the frequency of gastritis attacks. Some patients also require analgesics. Until healing occurs, patients' oxygen needs, blood volume, and fluid and electrolyte balance must be monitored.

When gastritis causes massive bleeding, treatment includes blood replacement; iced saline lavage, possibly with norepinephrine; angiography with vasopressin infused in normal saline solution; and, sometimes, surgery.

Vagotomy and pyloroplasty achieve limited success when conservative treatments fail. Rarely, partial or total gastrectomy may be required.

Simply avoiding aspirin and spicy foods may prevent exacerbations of chronic gastritis. If symptoms develop or persist, antacids may be taken. If pernicious anemia is the cause, vitamin B_{12} may be administered parenterally. A combination of bismuth and an antibiotic, such as amoxicillin, may relieve *H. pylori* infection, but eradication is difficult.

Special considerations

Patient care includes education and attention to various aspects of nutritional status to control symptoms and prevent their recurrence.

- For vomiting, give antiemetics and I.V. fluids, as ordered. Monitor fluid intake and output and electrolyte levels.
- Monitor the patient for recurrent symptoms as food is reintroduced; provide a bland diet.
- Offer smaller, more frequent meals to reduce irritating gastric secretions. Eliminate foods that cause gastric upset.

- Administer antacids and other prescribed medications, as ordered.
- If pain or nausea interferes with the patient's appetite, give analgesics or antiemetics 1 hour before meals.
- Tell the patient to avoid alcohol, caffeine, and irritating foods such as spicy or highly seasoned foods.
- If the patient smokes, refer him to a smoking-cessation program.
- Urge the patient to seek immediate attention for recurring symptoms, such as hematemesis, nausea, or vomiting.
- To prevent exacerbation, urge the patient to take prophylactic medications, as ordered.

PREVENTION

Advise the patient to take steroids with milk, food, or antacids. Instruct him to take antacids between meals and at bedtime and to avoid aspirin-containing compounds.

Gastroenteritis

A self-limiting disorder, gastroenteritis is characterized by diarrhea, nausea, vomiting, and acute or chronic abdominal cramping. Also called *intestinal flu, traveler's diarrhea, viral enteritis,* or *food poisoning,* it occurs in persons of all ages and is a major cause of morbidity and mortality in underdeveloped nations. In the United States, gastroenteritis ranks second to the common cold as a leading cause of lost work time and fifth as the leading cause of death among young children. It also can be life-threatening in elderly or debilitated people.

Causes and incidence

Gastroenteritis has many possible causes, including:

- bacteria (responsible for acute food poisoning), such as *Staphylococcus aureus, Salmonella, Shigella, Clostridium botulinum, C. perfringens,* and *Escherichia coli*
- amebae, especially *Entamoeba histolytica*

- parasites, such as *Ascaris, Enterobius,* and *Trichinella spiralis*
- viruses (may be responsible for traveler's diarrhea) such as adenoviruses, echoviruses, or coxsackieviruses
- ingestion of toxins, including plants or toadstools
- drug reactions; for example, to antibiotics
- enzyme deficiencies
- food allergens.

The bowel reacts to any of these enterotoxins with hypermotility, producing severe diarrhea and secondary depletion of intracellular fluid. Chronic gastroenteritis is usually the result of another GI disorder such as ulcerative colitis.

Diarrhea accounts for as many as 3% of pediatric office visits and 10% of hospitalizations for patients younger than age 5. Each year, gastroenteritis affects many adults and accounts for 8 million physician visits and 250,000 hospitalizations. Traveler's diarrhea affects 20% to 25% of people traveling from industrialized countries to developing countries.

Complications

- Severe dehydration
- Electrolyte loss
- Shock
- Vascular collapse
- Renal failure

Signs and symptoms

Signs and symptoms vary depending on the pathologic organism and on the level of GI tract involved. However, gastroenteritis in adults is usually an acute, self-limiting, nonfatal disease producing diarrhea, abdominal discomfort (ranging from cramping to pain), nausea, and vomiting. Other possible signs and symptoms include fever, malaise, and

borborygmi. In children, the elderly, and the debilitated, gastroenteritis produces the same symptoms, but these patients' intolerance to electrolyte and fluid losses leads to a higher mortality.

Diagnosis

Patient history can aid in the diagnosis of gastroenteritis. Stool culture (by direct rectal swab) or blood culture identifies the causative bacteria or parasites.

Treatment

Treatment is usually supportive and consists of bed rest, nutritional support, and increased fluid intake. When gastroenteritis is severe or affects a young child or an elderly or debilitated person, treatment may necessitate hospitalization, specific antimicrobials, I.V. fluid and electrolyte replacement and, possibly, antiemetics (given orally, I.M., or by rectal suppository).

Special considerations

Patient care includes education, administering medications, and assessing symptoms for signs of improvement or worsening.

- Administer medications as ordered; correlate dosages, routes, and times appropriately with the patient's meals and activities (for example, give antiemetics 30 to 60 minutes before meals).
- If the patient can eat, replace lost fluids and electrolytes with broth, ginger ale, and lemonade, as tolerated. Vary the diet to make it more enjoyable, and allow some choice of foods. Warn the patient to avoid milk and milk products, which may provoke recurrence.
- Record intake and output carefully and obtain serial weight measurements. Watch for signs of dehydration, such as dry skin and mucous membranes, fever, and sunken eyes.
- Wash your hands thoroughly after giving care to avoid spreading infection.
- To ease anal irritation, provide warm sitz baths or apply witch hazel compresses.

- If food poisoning is the likely cause of gastroenteritis, contact public health authorities so they can interview patients and food handlers, and take samples of the suspected contaminated food.

PREVENTION

Teach good hygiene to prevent recurrence. Instruct patients to cook foods—especially pork—thoroughly; to refrigerate perishable foods, such as milk, mayonnaise, potato salad, and cream-filled pastry; to always wash hands with warm water and soap before handling food, especially after using the bathroom; to clean utensils thoroughly; to avoid drinking water or eating raw fruit or vegetables when visiting a foreign country; and to eliminate flies and roaches in the home.

Peptic ulcers

Peptic ulcers—circumscribed lesions in the mucosal membrane—can develop in the lower esophagus, stomach, pylorus, duodenum, or jejunum. About 80% of all peptic ulcers are duodenal ulcers, which affect the proximal part of the small intestine.

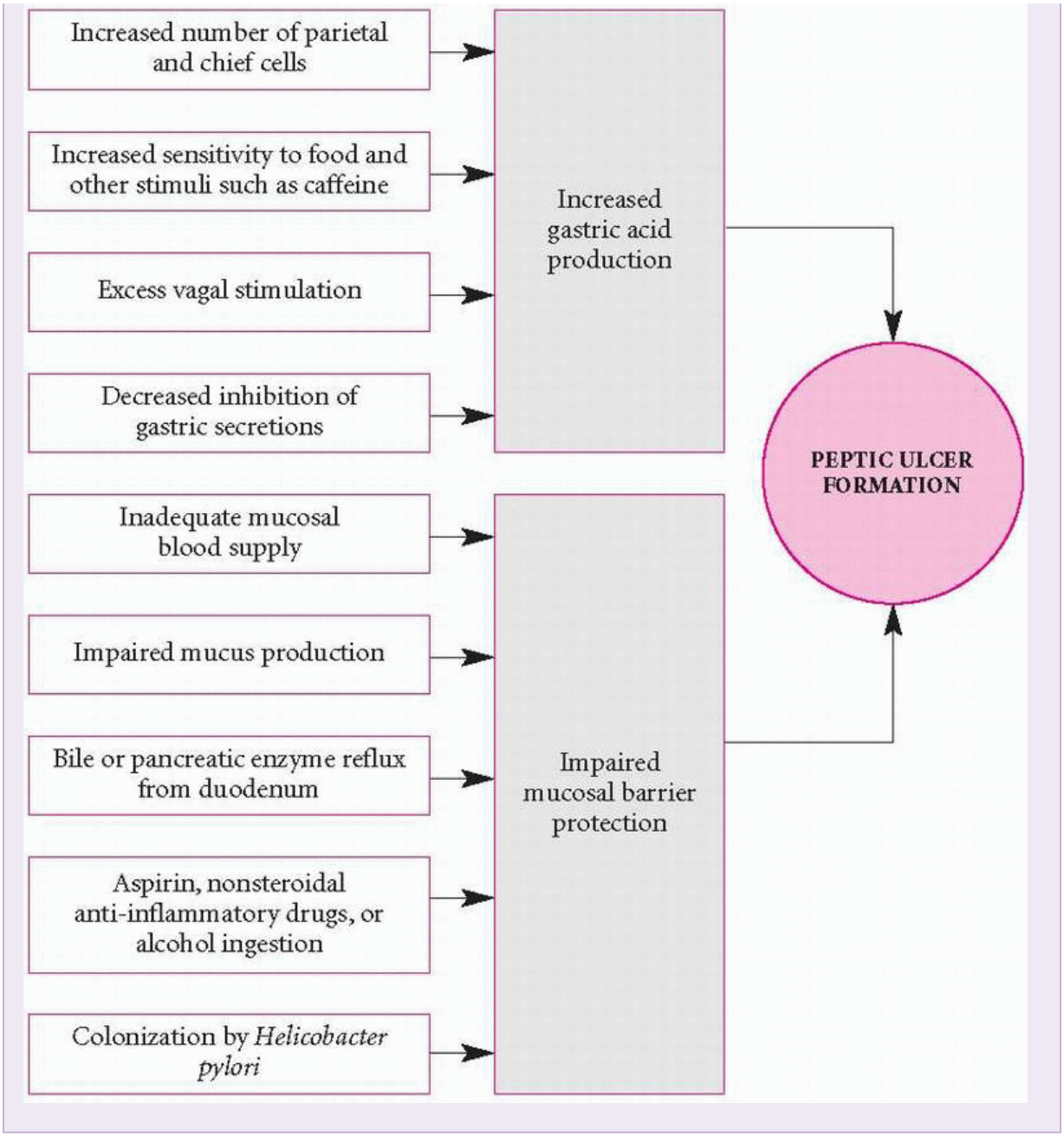
Gastric ulcers, which affect the stomach mucosa, are most common in middle-aged and elderly men, especially in chronic users of nonsteroidal anti-inflammatory drugs

(NSAIDs), alcohol, or tobacco. Duodenal ulcers usually follow a chronic course, with remissions and exacerbations; 5% to 10% of patients develop complications that necessitate surgery.

PATHOPHYSIOLOGY

HOW PEPTIC ULCERS DEVELOP

Peptic ulcers can result from factors that increase gastric acid production or from factors that impair mucosal barrier protection.



Causes and incidence

Researchers recognize three major causes of peptic ulcer disease: infection with *Helicobacter pylori* (formerly known as *Campylobacter pylori*), use of NSAIDs, and pathologic hypersecretory disorders such as Zollinger-Ellison syndrome. (See *How peptic ulcers develop*.)

How *H. pylori* produces an ulcer isn't clear. Gastric acid, which was considered a primary cause, now appears mainly to contribute to the

consequences of infection. Ongoing studies should soon unveil the full mechanism of ulcer formation.

Salicylates and other NSAIDs encourage ulcer formation by inhibiting the secretion

of prostaglandins (the substances that suppress ulceration). Certain illnesses, such as pancreatitis, hepatic disease, Crohn's disease, preexisting gastritis, and Zollinger-Ellison syndrome, are also known causes.

Besides peptic ulcer's main causes, several predisposing factors are acknowledged. They include blood type (gastric ulcers tend to strike people with type A blood; duodenal ulcers tend to afflict people with type O blood) and other genetic factors. Exposure to irritants, such as alcohol, coffee, and tobacco, may contribute by accelerating gastric acid emptying and promoting mucosal breakdown. Ulceration occurs when the acid secretion exceeds the buffering factors. Physical trauma, emotional stress, and normal aging are additional predisposing conditions.

In the United States, about 1.6 million people develop peptic ulcers yearly. Men and women are affected equally, and incidence increases with age. A higher percentage of *H. pylori* infection occurs in people older than age 50.

Complications

- GI hemorrhage
- Hypovolemic shock
- Perforation
- Obstruction
- Extension of ulcer into adjacent structures
- Dumping syndrome
- Vitamin deficiency

Signs and symptoms

Heartburn and indigestion usually signal the beginning of a gastric ulcer attack. Eating stretches the gastric wall and may cause or, in some cases, relieve pain and feelings of fullness and distention. Other typical effects include weight loss and repeated episodes of massive GI bleeding.

Duodenal ulcers produce heartburn, well-localized midepigastric pain (relieved by food), weight gain (because the patient eats to relieve discomfort), and a peculiar sensation of hot water bubbling in the back of the throat. Attacks usually occur about 2 hours after meals, whenever the stomach is empty, or after consumption of orange juice, coffee, aspirin, or alcohol. Exacerbations tend to recur several times per year and then fade into remission. Vomiting and other digestive disturbances are rare.

Ulcers may penetrate the pancreas and cause severe back pain. Other complications of peptic ulcers include perforation, hemorrhage, and pyloric obstruction. Ulcers may, on occasion, produce no symptoms.

Diagnosis

CONFIRMING DIAGNOSIS

Esophagogastroduodenoscopy confirms the presence of an ulcer and permits cytologic studies and biopsy to rule out H. pylori *or cancer.*

Diagnosis may be confirmed by the following tests:

- Barium swallow or upper GI and small-bowel series may reveal the presence of the ulcer. This is the initial test performed on a patient whose symptoms aren't severe.
- Laboratory analysis may detect occult blood in stools.
- Serologic testing may disclose clinical signs of infection such as an elevated white blood cell count.
- Carbon 13 (^{13}C) urea breath test results reflect activity of *H. pylori*.

Treatment

Experts recommend treating the patient with two antibiotics and an acid suppressor or stomach-lining protector to eradicate *H. pylori*. The patient taking NSAIDs may take a prostaglandin analog (misoprostol) to suppress ulceration (or the patient may take the analog with NSAIDs to prevent ulceration). Histamine-2 (H_2) receptor antagonists or proton pump inhibitors may reduce acid secretion. A coating agent or bismuth may be administered to the patient with a duodenal ulcer to protect the lining.

If GI bleeding occurs, emergency treatment begins with passage of a nasogastric (NG) tube to allow for iced saline lavage, possibly containing norepinephrine. Gastroscopy allows visualization of the bleeding site and coagulation by laser or cautery to control bleeding. This type of therapy allows postponement of surgery until the patient's condition stabilizes. Surgery is indicated for perforation, unresponsiveness to conservative treatment, and suspected

malignancy. Surgery for peptic ulcers may include:

- vagotomy and pyloroplasty: severing one or more branches of the vagus nerve to reduce hydrochloric acid secretion and refashioning the pylorus to create a larger lumen and facilitate gastric emptying
- distal subtotal gastrectomy (with or without vagotomy): excising the antrum of the stomach, thereby removing the hormonal stimulus of the parietal cells, followed by anastomosis of the rest of the stomach to the duodenum or the jejunum
- pyloroplasty: surgical enlargement of the pylorus to provide drainage of gastric secretions.

Special considerations

Management of peptic ulcers requires careful administration of medications, thorough patient teaching, and skillful postoperative care.

- Watch for adverse reactions to H_2-receptor antagonists and omeprazole (dizziness, fatigue, rash, and mild diarrhea).
- Advise any patient who uses antacids, who has a history of cardiac disease, or who follows a sodium-restricted diet to take only those

antacids that contain low amounts of sodium.

- Warn the patient to avoid NSAIDs because they irritate the gastric mucosa. For the same reason, advise the patient to stop smoking and to avoid stressful situations, excessive intake of coffee, and drinking alcoholic beverages during exacerbations of peptic ulcer disease.

After gastric surgery:

- Keep the NG tube patent. If the tube isn't functioning, don't reposition it; you might damage the suture line or anastomosis. Notify the surgeon promptly.
- Monitor intake and output, including NG tube drainage. Check for bowel sounds, and allow the patient nothing by mouth until peristalsis resumes and the NG tube is removed or clamped.
- Replace fluids and electrolytes. Assess the patient for signs of dehydration, sodium deficiency, and metabolic alkalosis, which may occur secondary to gastric suction.
- Monitor the patient for possible dumping syndrome (a rapid gastric emptying, causing distention of the duodenum or jejunum produced by a bolus of food). Signs and symptoms of dumping syndrome include diaphoresis, weakness, nausea, flatulence, explosive diarrhea, distention, and palpitations within 30 minutes after a meal.
- To avoid dumping syndrome, advise the patient to lie down after meals, to drink fluids *between* meals rather than with meals, to avoid eating large amounts of carbohydrates, and to eat four to six small, high-protein, low-carbohydrate meals during the day.

Ulcerative colitis

Ulcerative colitis is an inflammatory, usually chronic disease that affects the mucosa of the colon. It invariably begins in the rectum and sigmoid colon and commonly extends upward into the entire colon; it rarely affects the small intestine, except for the terminal ileum. Ulcerative colitis produces edema (leading to mucosal friability) and ulcerations. Severity ranges from a mild, localized disorder to a fulminant disease that may cause a perforated colon, progressing to potentially fatal peritonitis and toxemia.

Causes and incidence

Although the etiology of ulcerative colitis is unknown, it's thought to be related to abnormal immune response in the GI tract, possibly associated with food or bacteria such as *Escherichia coli*. Stress was once thought to be a cause of ulcerative colitis, but studies show that although it isn't a cause, it does increase the severity of the attack.

Ulcerative colitis occurs primarily in young adults, especially in women. It's also more prevalent among those of Jewish ancestry, indicating a possible familial tendency. The incidence of the disease is unknown; however, some studies indicate as many as 10 to 15 out of 100,000 persons have the disease. Onset of symptoms seems to peak between ages 15 and 30; another peak occurs between ages 50 and 70.

Complications

- Nutritional deficiency
- Perineal sepsis
- Anal fissure or fisulta
- Perirectal abscess
- Hemorrhage
- Toxic megacolon
- Coagulation deficit

Signs and symptoms

The hallmark of ulcerative colitis is recurrent attacks of bloody diarrhea, in many cases containing pus and mucus, interspersed with asymptomatic remissions. The intensity of these attacks varies with the extent of inflammation. It isn't uncommon for a patient with ulcerative colitis to have as many as 15 to 20 liquid, bloody stools daily. Other symptoms include spastic rectum and anus, abdominal pain, irritability, weight loss, weakness, anorexia, nausea, and vomiting.

Ulcerative colitis may lead to complications, such as hemorrhage, stricture, or perforation of the colon. Other complications include joint inflammation, ankylosing spondylitis, eye lesions, mouth ulcers, liver disease, and pyoderma gangrenosum. Scientists think that these complications occur when the immune system triggers inflammation in other parts of the body. These disorders are usually mild and disappear when the colitis is treated.

Patients with ulcerative colitis have an increased risk of developing colorectal cancer; children with ulcerative colitis may experience impaired growth and sexual development.

Diagnosis

CONFIRMING DIAGNOSIS

Sigmoidoscopy showing increased mucosal friability, decreased mucosal detail, pinpoint hemorrhages, and thick inflammatory exudate suggests this diagnosis. Biopsy can help confirm it.

Colonoscopy may be required to determine the extent of the disease and to evaluate strictured areas and pseudopolyps. (Biopsy would then be done during colonoscopy.) Barium enema can assess the extent of the disease and detect complications, such as strictures and carcinoma.

A stool sample should be cultured and analyzed for leukocytes, ova, and parasites. Other supportive laboratory values include decreased serum levels of potassium, magnesium, hemoglobin, and albumin as well as leukocytosis and increased prothrombin time. An elevated erythrocyte sedimentation rate correlates with the severity of the attack.

Treatment

The goals of treatment are to control inflammation, replace nutritional losses and blood volume, and prevent complications. Supportive treatment includes bed rest, I.V. fluid replacement, and a clear-liquid diet. For patients awaiting surgery or showing signs of dehydration and debilitation from excessive diarrhea, total parenteral nutrition (TPN) rests the intestinal tract, decreases stool volume, and restores positive

nitrogen balance. Blood transfusions or iron supplements may be needed to correct anemia.

Sulfasalazine (Azulfidine), the drug of choice, is used for its anti-inflammatory and anti-microbial action. Immunomodulators or 5-aminosalicylates may be used to decrease the frequency of attacks. Drug therapy to control inflammation includes steroids. Antispasmodics and antidiarrheals are used only in patients whose ulcerative colitis is under control but who have frequent, loose stools.

ALERT

Antispasmodics and antidiarrheals may lead to massive dilation of the colon (toxic megacolon) and are generally contraindicated.

Surgery is the last resort if the patient has toxic megacolon, fails to respond to drugs and supportive measures, or finds symptoms unbearable. A common surgical technique is proctocolectomy with ileostomy. Another procedure, the ileoanal pull-through, is being performed in more cases. This procedure entails performing a total proctocolectomy and mucosal stripping, creating a pouch from the terminal ileum, and anastomosing the pouch to the anal canal. A temporary ileostomy is created to divert stool and allow the rectal anastomosis to heal. The ileostomy is closed in 2 to 3 months, and the patient can then evacuate stool rectally. This procedure removes all the potentially malignant epithelia of the rectum and colon. Total colectomy and ileorectal anastomosis isn't as common because of its mortality rate (2% to 5%). This procedure removes the entire colon and anastomoses the terminal ileum to the rectum; it requires observation of the remaining rectal stump for any signs of cancer or colitis.

Pouch ileostomy (Kock pouch or continent ileostomy), in which the surgeon creates a pouch from a small loop of the terminal ileum and a nipple valve from the distal ileum, may be an option. The resulting stoma opens just above the pubic hairline and the pouch is emptied periodically through a catheter inserted in the stoma. In ulcerative colitis, a colectomy may be performed after 10 years of active disease because of the increased incidence of colon cancer in these cases. Performing a partial colectomy to prevent colon cancer is controversial.

Special considerations

Patient care includes close monitoring for changes in status.

- Accurately record intake and output, particularly the frequency and volume of stools. Watch for signs of dehydration and electrolyte imbalances, especially signs and symptoms of hypokalemia (muscle weakness and paresthesia) and hypernatremia (tachycardia, flushed skin, fever, and dry tongue). Monitor hemoglobin level and hematocrit, and give blood transfusions as ordered. Provide good mouth care for the patient who's allowed nothing by mouth.
- After each bowel movement, thoroughly clean the skin around the rectum. Provide an air mattress or sheepskin to help prevent skin breakdown.
- Administer medications, as ordered. Watch for adverse effects of prolonged corticosteroid therapy (moon face, hirsutism, edema, and gastric irritation). Be aware that corticosteroid therapy may mask infection.
- If the patient needs TPN, change dressings as ordered, assess for inflammation at the insertion site, and check capillary blood glucose levels every 4 to 6 hours.
- Take precautionary measures if the patient is prone to bleeding. Watch closely for signs of complications, such as a perforated colon and peritonitis (fever, severe abdominal pain, abdominal rigidity and tenderness, and cool, clammy skin) and toxic megacolon (abdominal distention and decreased bowel sounds).

For the patient requiring surgery:

- Carefully prepare the patient for surgery, and inform him about ileostomy.
- Do a bowel preparation, as ordered.
- After surgery, provide meticulous supportive care and continue teaching correct stoma care.
- Keep the nasogastric tube patent. After removal of the tube, provide a clear-liquid diet and gradually advance to a low-residue diet, as

tolerated.

- After a proctocolectomy and ileostomy, teach good stoma care. Wash the skin around the stoma with soapy water and dry it thoroughly. Apply karaya gum around the stoma's base to avoid irritation, and make a watertight seal. Attach the pouch over the karaya ring. Cut an opening in the ring to fit over the stoma, and secure the pouch to the skin. Empty the pouch when it's one-third full. Encourage the patient to report to physician stoma color changes or purulent drainage.
- After a pouch ileostomy, uncork the catheter every hour to allow contents to drain. After 10 to 14 days, gradually increase the length of time the catheter is left corked until it can be opened every 3 hours. Then remove the catheter and reinsert it every 3 to 4 hours for drainage. Teach the patient how to insert the catheter and how to take care of the stoma.
- Encourage the patient to have regular physical examinations.

Necrotizing enterocolitis

Necrotizing enterocolitis (NEC) is characterized by diffuse or patchy intestinal necrosis, accompanied by sepsis in about one-third of cases. Sepsis usually involves *Escherichia coli, Clostridia, Salmonella, Pseudomonas,* or *Klebsiella.* Initially, necrosis is localized, occurring anywhere along the intestine, but usually in the ileum, ascending colon, or rectosigmoid. If diffuse bleeding occurs, NEC usually results in disseminated

intravascular coagulation (DIC).

Causes and incidence

The exact cause of NEC is unknown. Suggested predisposing factors include birth asphyxia, postnatal hypotension, respiratory distress, hypothermia, umbilical vessel catheterization, exchange transfusion, or patent ductus arteriosus. NEC may also be a response to significant prenatal stress, such as premature rupture of membranes, placenta previa, maternal sepsis, toxemia of pregnancy, or breech or cesarean birth.

NEC may develop when the infant suffers perinatal hypoxemia due to shunting of blood from the gut to more vital organs. Subsequent mucosal ischemia provides an ideal medium for bacterial growth. Hypertonic formula may increase bacterial activity because—unlike maternal breast milk—it doesn't provide protective immunologic activity and it contributes to the production of hydrogen gas. As the bowel swells and breaks down, gas-forming bacteria invade damaged areas, producing free air in the intestinal wall. This may result in fatal perforation and peritonitis.

NEC usually occurs in premature infants (less than 34 weeks' gestation) and those of low birth weight (less than 1,500 grams). NEC is more common in some geographic areas, possibly due to the higher incidence and survival of premature neonates and neonates who have low birth weights in these areas. NEC also may occur in infants in a nursery where an outbreak has occurred, suggesting an infectious cause. Although the outcome is improved with aggressive, early treatment, it's a serious disease with a death rate of 25%.

Complications

- Perforation
- Mechanical and functional abnormalities of the intestine

Signs and symptoms

Neonates who have suffered from perinatal hypoxemia have the potential for developing NEC. A distended (especially tense or rigid) abdomen with gastric retention is the earliest and most common sign of oncoming NEC, which usually appears 1 to 10 days after birth. Other clinical features are increasing residual gastric contents (which may contain bile), bile-stained vomitus, and occult blood in the stool. About 25% of patients have bloody diarrhea. A red or shiny, taut abdomen may indicate peritonitis.

Nonspecific signs and symptoms include thermal instability, lethargy, metabolic acidosis, jaundice, and DIC. The major complication is perforation, which requires surgery. Recurrence of NEC and mechanical and functional abnormalities of the intestine, especially stricture, are

the usual cause of residual intestinal malfunction in any infant who survives acute NEC; this complication may develop as late as 3 months postoperatively.

Diagnosis

Successful treatment of NEC relies on early recognition.

CONFIRMING DIAGNOSIS

Abdominal X-rays confirm the diagnosis, showing nonspecific intestinal dilation and, in later stages of NEC, pneumatosis cystoides intestinalis (gas or air in the intestinal wall).

Blood studies show several abnormalities. Platelet count may fall below 50,000/µl. Serum sodium levels are decreased, and arterial blood gas levels indicate metabolic acidosis (a result of sepsis). Infection-induced red blood cell breakdown elevates bilirubin levels. Blood and stool cultures identify the infecting organism, clotting studies and the hemoglobin level reveal associated DIC, and the guaiac test detects occult blood in the stool.

Treatment

The first signs of NEC necessitate removal of the umbilical catheter (arterial or venous) and discontinuation of oral intake for 7 to 10 days to rest the injured bowel. I.V. fluids, including total parenteral nutrition, maintain fluid and electrolyte balance and nutrition during this time; passage of a nasogastric (NG) tube aids bowel decompression. If coagulation studies indicate a need for transfusion, the infant usually receives dextran to promote hemodilution, increase mesenteric blood flow, and reduce platelet aggregation. Antibiotic therapy consists of parenteral agents—administered through an NG tube, if necessary—to suppress bacterial flora and prevent bowel perforation. Anteroposterior and lateral X-rays are repeated to monitor disease progression.

Surgery is indicated if the patient shows any of these signs or symptoms: signs of perforation (free intraperitoneal air on X-ray or symptoms of

peritonitis), respiratory insufficiency (caused by severe abdominal distention), progressive and intractable acidosis, or DIC. Surgery removes all necrotic and acutely inflamed bowel and creates a temporary colostomy or ileostomy. At least 12" (30.5 cm) of bowel must remain or the infant may suffer from malabsorption or chronic vitamin B_{12} deficiency.

Special considerations

ALERT

Be alert for signs or symptoms of gastric distention and perforation: apnea, cardiovascular shock, sudden drop in temperature, bradycardia, sudden listlessness, rag-doll limpness, increasing abdominal tenderness, edema, erythema, or involuntary abdominal rigidity.

- To avoid perforating the bowel, *don't* take rectal temperatures.
- Prevent cross-contamination by disposing of soiled diapers properly and washing your hands after diaper changes.
- After surgery, the infant needs mechanical ventilation. Gently suction secretions, and assess his breathing often.
- Replace fluids lost through NG tube and stoma drainage. Include drainage losses in output records. Weigh the infant daily. A daily weight gain of 10 to 20 g indicates a good response to therapy.
- An infant with a temporary colostomy or ileostomy needs special care. Explain to the parents what a colostomy or ileostomy is and why it's necessary. Encourage them to participate in their infant's physical care after his condition is no longer critical.
- Because of the infant's small abdomen, the suture line is near the stoma; as a result, keeping the suture line clean can be a problem. Good skin care is essential because the immature infant's skin is fragile and vulnerable to excoriation and the active enzymes in bowel secretions are corrosive. Improvise premature-sized colostomy bags from urine collection bags, medicine cups, or condoms. Karaya gum is helpful in making a seal. Watch for wound disruption, infection, and excoriation—potential dangers because of severe catabolism.

- Watch for intestinal malfunction from stricture or short-gut syndrome. Such complications usually develop 1 month after the infant resumes normal feedings.

PREVENTION

Encourage mothers to breast-feed because breast milk contains live macrophages that fight infection and has a low pH that inhibits the growth of many organisms. Also, colostrum—fluid secreted before the milk—contains high concentrations of immunoglobulin A, which directly protects the gut from infection and which the neonate lacks for several days postpartum. Tell mothers that they may refrigerate their milk for 48 hours but shouldn't freeze or heat it because this destroys antibodies. Tell them to use plastic—not glass—containers because leukocytes adhere to glass.

Crohn's disease

Crohn's disease, also known as *regional enteritis* and *granulomatous colitis,* is an inflammation of any part of the GI tract (usually the proximal portion of the colon or, less commonly, the terminal ileum) that extends through all layers of the intestinal wall. It may also involve regional lymph nodes and the mesentery. Granulomas are usually surrounded by normal mucosa; when these lesions are present in multiples, they're commonly referred to as *skip lesions.* The surface of the inflamed GI tract usually has a cobblestone appearance, which is different from alternating areas of inflammation and fissure crevices.

Causes and incidence

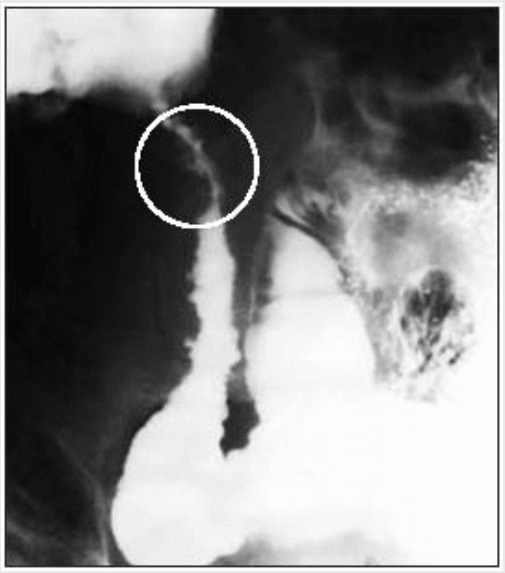
In Crohn's disease, lacteal blockage in the intestinal wall leads to edema and, eventually, inflammation, ulceration, and stenosis. Abscesses and fistulas may also occur.

Although the exact cause of Crohn's disease is unknown, autoimmune and genetic factors are thought to play a role. Up to 5% of those with the disease have one or

more affected relatives; Jewish ancestry is also a risk factor. However, a pattern of Mendelian inheritance hasn't been identified.

THE "STRING SIGN"

The characteristic "string sign" (marked narrowing of the bowel), resulting from inflammatory disease and scarring, strengthens the diagnosis of Crohn's disease.



The incidence of Crohn's disease has risen steadily over the past 50 years; it now affects 7 out of every 100,000 people. Crohn's disease is most prevalent in adults ages 20 to 40. It's two to three times more common in those of Jewish ancestry and least common in blacks.

Complications

- Anal fistula
- Perineal abscess
- Intestinal obstruction
- Nutritional deficiency
- Peritonitis (rare)

Signs and symptoms

Clinical effects may be mild and nonspecific initially; they vary according to the location and extent of the lesion. Acute inflammatory signs and symptoms mimic appendicitis and include steady, colicky pain in the right lower quadrant, cramping, tenderness, flatulence, nausea, fever, and diarrhea. Bleeding may occur and, although usually mild, may be massive. Bloody stools may also occur.

Chronic symptoms, which are more typical of the disease, are more persistent and less severe; they include diarrhea (four to six stools per day) with pain in the right lower abdominal quadrant, steatorrhea (excess fat in feces), marked weight loss and, rarely, clubbing of fingers. The patient may complain of weakness and fatigue. Complications include intestinal obstruction, fistula formation between the small bowel and the bladder, perianal and perirectal abscesses and fistulas, intra-abdominal abscesses, and perforation.

Diagnosis

CONFIRMING DIAGNOSIS

Barium enema showing the string sign (segments of stricture separated by normal bowel) supports a diagnosis of Crohn's disease. (See The "string sign.")

Sigmoidoscopy and colonoscopy may show patchy areas of inflammation, thus helping to rule out ulcerative colitis. However, biopsy is required for a definitive diagnosis.

Laboratory findings commonly indicate increased white blood cell count and erythrocyte sedimentation rate, hypokalemia, hypocalcemia, hypomagnesemia, and a decreased hemoglobin level.

Treatment

To control the inflammatory process, medications, such as 5-aminosalicylate and sulfasalazine, may be prescribed. Corticosteroids and immunomodulators, such as azathioprine (Imuran) and mercaptopurine (Purinethol), may be prescribed if 5-aminosalicylate isn't effective or in patients with severe Crohn's disease. In debilitated

patients, therapy includes total parenteral nutrition to maintain nutritional status while resting the bowel. If abscesses or fistulas occur, antibiotics may be prescribed. Infliximab (Remicade), an antibody to tumor necrosis factor-alpha (an immune chemical that promotes inflammation), may also be prescribed.

Effective treatment requires important changes in lifestyle: physical rest, restricted diet (specific foods vary from person to person), vitamin B_{12} injections and supplements, and elimination of dairy products for lactose intolerance.

Surgery may be necessary to correct bowel perforation, massive hemorrhage, fistulas, or acute intestinal obstruction. Colectomy with ileostomy is necessary in many patients with extensive disease of the large intestine and rectum.

Special considerations

Although treatment is based largely on symptoms, you should monitor the patient's status carefully for signs of worsening.

- Record fluid intake and output (including the amount of stool), and weigh the patient daily. Watch for dehydration and maintain fluid and electrolyte balance. Be alert for signs of intestinal bleeding (bloody stools); check stools daily for occult blood.
- Check hemoglobin level and hematocrit regularly. Give iron supplements and blood transfusions, as ordered.
- Provide good patient hygiene and mouth care if the patient is restricted to nothing by mouth. After each bowel movement, give good skin care. Always keep a clean, covered bedpan within the patient's reach. Ventilate the room to eliminate odors.
- Observe the patient for fever and pain or pneumaturia, which may signal bladder fistula. Abdominal pain and distention and fever may indicate intestinal obstruction. Watch for stools from the vagina and an enterovaginal fistula.
- Before ileostomy, arrange for a visit by an enterostomal therapist.

- After surgery, frequently check the patient's I.V. line and NG tube for proper functioning. Monitor his vital signs and fluid intake and output. Watch for wound infection. Provide meticulous stoma care, and teach it to the patient and his family. Realize that ileostomy changes the patient's body image, so offer reassurance and emotional support.
- Stress the need for a severely restricted diet and bed rest, which may be trying, particularly for the young patient. Encourage him to try to reduce tension. If stress is clearly an aggravating factor, refer him for counseling.
- Refer the patient to a support group such as the Crohn's and Colitis Foundation of America.

Pseudomembranous enterocolitis

Pseudomembranous enterocolitis is an acute inflammation and necrosis of the small and large intestines, which usually affects the mucosa but may extend into submucosa and, rarely, other layers. Marked by severe diarrhea, this rare condition is generally fatal in 1 to 7 days due to severe dehydration and toxicity, peritonitis, or perforation.

Causes and incidence

The exact cause of pseudomembranous enterocolitis is unknown; however, *Clostridium difficile* is thought to produce a toxin that may play a role in its development. Pseudomembranous enterocolitis has occurred postoperatively in debilitated patients who undergo abdominal surgery and in patients treated with broad-spectrum antibiotics. Whatever the cause, necrotic mucosa is replaced by a pseudomembrane filled with staphylococci, leukocytes, mucus, fibrin, and inflammatory cells.

Incidence of antibiotic-related diarrhea varies from 5% to 39%, depending on the antibiotic. Pseudomembranous enterocolitis complicates 10% of these cases.

PEDIATRIC TIP

Ampicillin is the most common antibiotic associated with pseudomembranous enterocolitis in children.

Complications

- Severe dehydration
- Electrolyte imbalance
- Hypotension
- Shock
- Colonic perforation
- Peritonitis

Signs and symptoms

Pseudomembranous enterocolitis begins suddenly with copious watery or bloody

diarrhea that may contain pus or mucus, abdominal pain, and fever. Serious complications, including severe dehydration, electrolyte imbalance, hypotension, shock, and colonic perforation, may occur in this disorder.

Diagnosis

Diagnosis is difficult in many cases because of the abrupt onset of enterocolitis and the emergency situation it creates, so consideration of patient history is essential. A rectal biopsy through sigmoidoscopy confirms pseudomembranous enterocolitis. Stool cultures can identify *C. difficile*.

Treatment

A patient receiving broad-spectrum antibiotic therapy must discontinue antibiotics at once. Effective treatment usually includes metronidazole. Oral vancomycin is usually given for severe or resistant cases. A patient with mild pseudomembranous enterocolitis may receive anion exchange resins, such as cholestyramine, to bind the toxin produced by *C. difficile*. Supportive treatment must maintain fluid and electrolyte balance and combat hypotension and shock with pressors, such as dopamine and levarterenol.

Special considerations

Careful observation for signs of worsening is essential.

- Monitor the patient's vital signs, skin color, and level of consciousness. Immediately report signs of shock.
- Record fluid intake and output, including fluid lost in stools. Watch for dehydration (poor skin turgor, sunken eyes, and decreased urine output).
- Check serum electrolyte levels daily, and watch for clinical signs of hypokalemia, especially malaise, and a weak, rapid, irregular pulse.

PREVENTION

Antibiotics should be used only as prescribed by physicians. In addition, studies show the use of probiotics along with antibiotics may help prevent pseudomembranous colitis.

Irritable bowel syndrome

Irritable bowel syndrome (IBS), also called *spastic colon* and *spastic colitis,* is a common condition marked by chronic or periodic diarrhea, alternating with constipation, and accompanied by straining and abdominal cramps. The prognosis is good. Supportive treatment or avoidance of a known irritant usually relieves symptoms.

Causes and incidence

This functional disorder is generally associated with psychological stress; however, it may result from physical factors, such as diverticular disease, hormonal changes during the menstrual cycle, ingestion of irritants (coffee, raw fruits or vegetables), lactose intolerance, laxative abuse, food poisoning, or colon cancer. Some patients may experience a disturbance in the movement of the intestine or a lower tolerance for stretching and movement of the intestine.

IBS affects 10% to 20% of U.S. residents and has a yearly incidence rate of 1% to 2%. The condition occurs most commonly in women ages 20 to

30.

Complications

- Diverticulitis
- Colon cancer (higher incidence)

Signs and symptoms

IBS characteristically produces lower abdominal pain (usually relieved by defecation or passage of gas) and diarrhea that typically occurs during the day. These symptoms alternate with constipation or normal bowel function. Stools are commonly small and contain visible mucus. Dyspepsia and abdominal distention may occur.

Symptoms of IBS are two to three times more common in women than in men, with women comprising 80% of patients with a more severe form of the disorder.

Diagnosis

Diagnosis of IBS requires a careful history to determine contributing psychological factors such as a recent stressful life change. Diagnosis must also rule out other disorders,

such as amebiasis, diverticulitis, colon cancer, and lactose intolerance. Appropriate diagnostic procedures include sigmoidoscopy, colonoscopy, barium enema, rectal biopsy, and stool examination for blood, parasites, and bacteria.

Treatment

Therapy aims to relieve symptoms and includes counseling to help the patient understand the relationship between stress and his illness. Strict dietary restrictions aren't beneficial, but food irritants should be investigated and the patient should be instructed to avoid them. Rest and heat applied to the abdomen are helpful, as is judicious use of sedatives and antispasmodics. However, with chronic use, the patient may become dependent on these drugs. If the cause of IBS is chronic

laxative abuse, bowel training may help correct the condition. Tegaserod maleate (Zelnorm) may be prescribed for the patient with constipationpredominant IBS and no heart problems.

Special considerations

Because the patient with IBS isn't hospitalized, focus your care on patient teaching.

- Tell the patient to avoid irritating foods, and encourage him to develop regular bowel habits.
- Help the patient deal with stress, and warn against dependence on sedatives or antispasmodics.
- Encourage regular checkups because IBS is associated with a higher-than-normal incidence of diverticulitis and colon cancer. For patients older than age 40, emphasize the need for an annual sigmoidoscopy and rectal examination.

Celiac disease

Celiac disease (also known as *idiopathic steatorrhea, nontropical sprue, gluten enteropathy,* and *celiac sprue*) is characterized by poor food absorption and intolerance of gluten, a protein in wheat and wheat products. Malabsorption in the small bowel results from atrophy of the villi and a decrease in the activity and amount of enzymes in the surface epithelium. The prognosis is good with treatment (eliminating gluten from the patient's diet), but residual bowel changes may persist in adults.

Causes and incidence

In celiac disease, an intramucosal enzyme defect produces an inability to digest gluten. Resulting tissue toxicity produces rapid cell turnover, increases epithelial lymphocytes, and damages surface epithelium of the small bowel.

Celiac disease affects 1 of every 133 people in the United States and results from environmental factors and a genetic predisposition, but the exact mechanism is unknown. A strong association exists between the

disease and two human leukocyte antigen haplotypes, DR3 and DQw2. It may also be autoimmune in nature. It affects twice as many women and girls as men and boys and occurs more commonly among relatives, especially siblings. This disease primarily affects whites and those of European ancestry.

Many diseases and conditions are associated with celiac disease, including:

- anemia
- lactose intolerance
- skin disorders such as dermatitis herpetiformis (a burning, itching, blistering rash)
- type 1 diabetes mellitus
- thyroid disease
- Down syndrome
- unexplained infertility or miscarriage
- osteoporosis or osteopenia
- autoimmune disorders, such as rheumatoid arthritis and systemic lupus erythematosus.

Complications

- Anemia from malabsorption
- Syncope, heart failure, and angina from anemia
- Bleeding disorders from vitamin K deficiency
- Intestinal lymphoma (higher incidence)

Signs and symptoms

Celiac disease produces clinical effects on many body systems:

- GI symptoms include recurrent attacks of diarrhea, steatorrhea, abdominal distention due to flatulence, stomach cramps, weakness,

anorexia and, occasionally, increased appetite without weight gain. Atrophy of intestinal villi leads to malabsorption of fat, carbohydrates, and protein as well as loss of calories, fat-soluble vitamins (A, D, and K), calcium, and essential minerals and electrolytes. In adults, celiac disease produces multiple nonspecific ulcers in the small bowel, which may perforate or bleed.

- Hematologic effects include normochromic, hypochromic, or macrocytic anemia due to poor absorption of folate, iron, and vitamin B12 and to hypoprothrombinemia from jejunal loss of vitamin K.
- Osteomalacia, osteoporosis, tetany, and bone pain (especially in the lower back, rib cage, and pelvis) are some of the musculoskeletal symptoms of celiac disease. These signs and symptoms are due to calcium loss and vitamin D deficiency, which weakens the skeleton, causing rickets in children and compression fractures in adults.
- Neurologic effects may include peripheral neuropathy, seizures, or paresthesia.
- Dry skin, eczema, psoriasis, dermatitis herpetiformis, and acne rosacea are some of the dermatologic effects of celiac disease. Deficiency of sulfur-containing amino acids may cause generalized fine, sparse, prematurely gray hair; brittle nails; and localized hyperpigmentation on the face, lips, or mucosa.
- Endocrine symptoms include amenorrhea, hypometabolism and, possibly, with severe malabsorption, adrenocortical insufficiency.
- Psychosocial effects include mood changes and irritability.

Symptoms may develop during the first year of life, when gluten is introduced into the child's diet as cereal. Clinical effects may disappear during adolescence and reappear in adulthood. One theory proposes that the age at which symptoms first appear depends on the strength of the genetic factor: A strong factor produces symptoms during the child's first 4 years; a weak factor, in late childhood or adulthood.

Diagnosis

CONFIRMING DIAGNOSIS

Histologic changes seen on small-bowel biopsy specimens obtained with an esophagogastroduodenoscopy confirm the diagnosis: a mosaic pattern of alternating flat and bumpy areas on the bowel surface due to an almost total absence of villi and an irregular, blunt, and disorganized network of blood vessels. These changes appear most prominently in the jejunum.

An elevated alkaline phosphatase level may indicate bone loss, which is commonly experienced before diagnosis. Low cholesterol and albumin levels may reflect malabsorption and malnutrition. Mildly elevated liver enzymes and abnormal blood clotting may also be noted as well as anemia.

Antibody blood tests useful in screening for celiac disease include IgA antiendomysial antibody (AEA), anti-tissue transglutaminase (+TGA), antigliadin (IgA and IgG), and total serum IgA. Combined, these antibodies provide a sensitive and specific indicator for the presence of celiac disease.

Treatment

Treatment requires elimination of gluten from the patient's diet for life. Even with this exclusion, a full return to normal absorption and bowel histology may not occur for months or may never occur.

Supportive treatment may include supplemental iron, vitamin B12, and folic acid; reversal of electrolyte imbalance (by I.V. infusion, if necessary); I.V. fluid replacement for dehydration; corticosteroids to treat accompanying adrenal insufficiency; and vitamin K for hypoprothrombinemia.

Special considerations

Explain the necessity of a gluten-free diet to the patient (and to his parents, if the patient is a child). Advise eliminating wheat, barley, rye, and oats as well as foods made from these grains, such as breads and baked goods; suggest substituting corn or rice. Consult a dietitian for nutritional instruction on a gluten-free diet. Depending on individual

tolerance, the diet initially consists of proteins and gradually expands to include other foods. Assess the patient's

acceptance and understanding of the disease, and encourage regular reevaluation.

ALERT

Because many foods contain hidden sources of gluten, food labels must be read carefully.

- Observe the patient's nutritional status and progress by daily calorie counts and weight checks. Also, evaluate his tolerance to new foods. In the early stages, offer small, frequent meals to counteract anorexia.
- Assess the patient's fluid status: record intake, urine output, and number of stools (may exceed 10 per day). Watch for signs of dehydration, such as dry skin and mucous membranes, and poor skin turgor.
- Check serum electrolyte levels. Watch for signs of hypokalemia (weakness, lethargy, rapid pulse, nausea, and diarrhea) and low calcium levels (impaired blood clotting, muscle twitching, and tetany).
- Monitor prothrombin time, hemoglobin level, and hematocrit. Protect the patient from bleeding and bruising. Use the Z-track method to give iron I.M. If the patient can tolerate oral iron, give it between meals, when absorption is best. Dilute oral iron preparations, and give them through a straw to prevent staining teeth.
- Protect the patient with osteomalacia from injury by keeping the bed side rails up and assisting with ambulation, as necessary.
- Advise the patient to contact the Gluten Intolerance Group or the Associated Celiac Disease Foundation for information and support.

Diverticular disease

In diverticular disease, bulging pouches (diverticula) in the GI wall push the mucosal lining through the surrounding muscle. The most common site for diverticula is in the sigmoid colon, but they may develop

anywhere, from the proximal end of the pharynx to the anus. Other typical sites are the duodenum, near the pancreatic border or the ampulla of Vater, and the jejunum. Diverticular disease of the stomach is rare and is usually a precursor of peptic or neoplastic disease. Diverticular disease of the ileum (Meckel's diverticulum) is the most common congenital anomaly of the GI tract. (See *Meckel's diverticulum*.)

MECKEL'S DIVERTICULUM

In Meckel's diverticulum, a congenital abnormality—a blind tube, like the appendix—opens into the distal ileum near the ileocecal valve. This disorder results from failure of the intra-abdominal portion of the yolk sac to close completely during fetal development. It occurs in 2% of the population, mostly in males.

Uncomplicated Meckel's diverticulum produces no symptoms, but complications cause abdominal pain, especially around the umbilicus, and dark red melena or hematochezia. The lining of the diverticulum is gastric mucosa. This disorder may lead to peptic ulceration, perforation, and peritonitis and may resemble acute appendicitis.

Meckel's diverticulum also may cause bowel obstruction when a fibrous band that connects the diverticulum to the abdominal wall, the mesentery, or other structures snares a loop of the intestine. This may cause intussusception into the diverticulum or volvulus near the diverticular attachment to the back of the umbilicus or another intra-abdominal structure. Meckel's diverticulum should be considered in cases of GI obstruction or hemorrhage, especially when routine GI X-rays are negative.

Treatment consists of surgical resection of the inflamed bowel and antibiotic therapy if infection is present.

Diverticular disease has two clinical forms. In *diverticulosis,* diverticula are present but don't cause symptoms. In *diverticulitis,* diverticula are inflamed and may cause potentially fatal obstruction, infection, or hemorrhage.

Causes and incidence

In diverticulitis, retained undigested food mixed with bacteria accumulates in the diverticular sac, forming a hard mass (fecalith). This substance cuts off the blood supply to the thin walls of the sac, making them more susceptible to attack by colonic bacteria. Inflammation follows, possibly leading to perforation, abscess, peritonitis, obstruction, or hemorrhage. Occasionally, the inflamed colon segment may produce a fistula by adhering to the bladder or other organs.

Diverticula probably result from high intraluminal pressure on areas of weakness in the GI wall, where blood vessels enter. Diet may also be a contributing factor because insufficient fiber reduces fecal residue, narrows the bowel lumen, and leads to higher intra-abdominal pressure during defecation. The prevalence of diverticulosis in Western industrialized nations, where processing removes much of the roughage from foods, supports this theory. Diverticular disease is most prevalent in those older than age 40.

The incidence of diverticular disease increases with age, but 20% of patients are younger than age 50. Right-sided diverticulitis is most common in Asians, accounting for 75% of cases in that ethnic group. Left-sided diverticulitis is more common in Western countries, where it accounts for 70% of cases.

ELDER TIP

About 50% of older adults develop diverticulosis. In elderly patients, a rare complication of diverticulosis (without diverticulitis) is hemorrhage from colonic diverticula. Such hemorrhage is usually mild to moderate and easily controlled, but may occasionally be massive and life-threatening.

Complications

- Rectal hemorrhage
- Portal pyremia
- Fistula

Signs and symptoms

Diverticulosis usually produces no symptoms but may cause recurrent left lower quadrant pain, which is commonly accompanied by alternating constipation and diarrhea and is relieved by defecation or the passage of flatus. Symptoms resemble irritable bowel syndrome (IBS) and suggest that both disorders may coexist.

Mild diverticulitis produces moderate left lower abdominal pain, mild nausea, gas, irregular bowel habits, low-grade fever, and leukocytosis. In severe diverticulitis, the diverticula can rupture and produce abscesses or peritonitis, which occurs in up to 20% of such patients. Symptoms of rupture include abdominal rigidity and left lower quadrant pain. Peritonitis follows release of fecal material from the rupture site and causes signs of sepsis and shock (high fever, chills, and hypotension). Rupture of the diverticulum near a vessel may cause microscopic or massive hemorrhage, depending on the vessel's size.

Chronic diverticulitis may cause fibrosis and adhesions that narrow the bowel's lumen and lead to bowel obstruction. Symptoms of incomplete obstruction are constipation, ribbonlike stools, intermittent diarrhea, and abdominal distention. Increasing obstruction causes abdominal rigidity and pain, diminishing or absent bowel sounds, nausea, and vomiting.

Diagnosis

In many cases, diverticular disease produces no symptoms and is found during an upper GI series performed as part of a differential diagnosis.

Tests showing diverticular disease include computed tomography (reveals areas of inflammation), colonoscopy, sigmoidoscopy, and barium enema.

CONFIRMING DIAGNOSIS

A barium study confirms the diagnosis. An upper GI series confirms or rules out diverticulosis of the esophagus and upper bowel; a barium enema confirms or rules out diverticulosis of the lower bowel.

Barium-filled diverticula can be single, multiple, or clustered and may have a wide or narrow mouth. Barium outlines—but doesn't fill—diverticula blocked by impacted feces. In patients with acute diverticulitis, a barium enema may rupture the bowel, so this procedure requires caution. If IBS accompanies diverticular disease, X-rays may reveal colonic spasm.

Biopsy rules out cancer; however, a colonoscopic biopsy isn't recommended during acute diverticular disease because of the strenuous bowel preparation it requires. Blood studies may show an elevated erythrocyte sedimentation rate in diverticulitis, especially if the diverticula are infected.

Treatment

Diverticulosis that doesn't produce symptoms generally doesn't necessitate treatment. Intestinal diverticulosis with pain, mild GI distress, constipation, or difficult defecation may respond to a liquid or bland diet, stool softeners, and occasional doses of mineral oil. These measures relieve symptoms, minimize irritation, and lessen the risk of progression to diverticulitis. After pain subsides, patients also benefit from a high-residue diet and bulk medication such as psyllium.

Treatment of mild diverticulitis without signs of perforation must prevent constipation and combat infection. It may include bed rest, a liquid diet, stool softeners, and a broad-spectrum antibiotic.

If diverticulitis is refractory to medical treatment, a colon resection is necessary to remove the involved segment. Perforation, peritonitis, obstruction, or fistula that accompanies diverticulitis may require a temporary colostomy to drain abscesses and rest the colon, followed by later reanastomosis 6 weeks to 3 months after initial surgery.

Special considerations

Management of uncomplicated diverticulosis chiefly involves thorough patient education about fiber and dietary habits.

- Make sure that the patient understands the importance of dietary fiber and the harmful effects of constipation and straining during defecation. Encourage increased intake of foods high in indigestible fiber, including fresh fruits and vegetables, whole grain bread, and wheat or bran cereals. Warn that a high-fiber diet may temporarily cause flatulence and discomfort. Advise the patient to relieve constipation with stool softeners or bulk-forming cathartics. However, caution the patient against taking bulk-forming cathartics without plenty of water; if swallowed dry, they may absorb enough moisture in the mouth and throat to swell and obstruct the esophagus or trachea.
- If the patient with diverticular disease is hospitalized, observe his stools carefully for frequency, color, and consistency, and keep accurate pulse and temperature charts because changes may signal developing inflammation or complications.

After surgery to resect the colon:

- Watch for signs of infection.
- Provide meticulous wound care because perforation may already have infected the area.
- Check drain sites frequently for signs of infection (purulent drainage or foul odor) or fecal drainage.
- Change dressings as necessary.
- Encourage coughing and deep breathing to prevent atelectasis.
- Watch for signs of postoperative bleeding (hypotension and decreased hemoglobin level and hematocrit).
- Record intake and output accurately.
- Keep the nasogastric tube patent.
- Teach ostomy care as needed.
- Arrange for a visit by an enterostomal therapist.

Appendicitis

Appendicitis is inflammation of the vermiform appendix due to an obstruction.

Causes and incidence

Appendicitis probably results from an obstruction of the appendiceal lumen caused by a fecal mass, stricture, barium ingestion, or viral infection. This obstruction sets off an inflammatory process that can lead to infection, thrombosis, necrosis, and perforation. If the appendix ruptures or perforates, the infected contents spill into the abdominal cavity, causing peritonitis, the most common and most perilous complication of appendicitis.

Appendicitis occurs more commonly in men than in women, with a peak incidence in the late teens and early 20s. About 250,000 cases are reported annually. It's a

common cause of surgical emergency in children, with 4 appendectomies per 1,000 admissions for appendicitis annually in the United States.

Signs and symptoms

Typically, appendicitis begins with generalized or localized abdominal pain in the right upper abdomen, followed by anorexia, nausea, and vomiting (rarely profuse). Pain eventually localizes in the right lower abdomen (McBurney's point) with abdominal “boardlike” rigidity, retractive respirations, increasing tenderness, increasingly severe abdominal spasms and, almost invariably, rebound tenderness. (Rebound tenderness on the opposite side of the abdomen suggests peritoneal inflammation.)

Later signs and symptoms include constipation or diarrhea, slight fever, and tachycardia. The patient may walk bent over or lie with his right knee flexed to reduce pain.

ALERT

Sudden cessation of abdominal pain indicates perforation or infarction of the appendix.

Complications

- Perforation
- Peritonitis
- Appendiceal abscess
- Pyelophlebitis

Diagnosis

Diagnosis of appendicitis is based on physical findings and characteristic clinical symptoms. Supportive findings include a temperature of 99° to 102° F (37.2° to 38.9° C) and a moderately elevated white blood cell count (12,000 to 15,000/mcl), with increased immature cells.

Diagnosis must rule out illnesses with similar symptoms: gastritis, gastroenteritis, ileitis, colitis, diverticulitis, pancreatitis, renal colic, bladder infection, ovarian cyst, and uterine disease. It may be strongly suspected based on abdominal sonography or computed tomography scan. Appendicitis can be confirmed by exploratory laparoscopy.

Treatment

Appendectomy is the only effective treatment. Laparoscopic appendectomies decrease the recovery time and thus the hospital stay. If peritonitis develops, treatment involves GI intubation, parenteral replacement of fluids and electrolytes, and administration of antibiotics.

Special considerations

If appendicitis is suspected, or during preparation for appendectomy:

- Administer I.V. fluids to prevent dehydration. *Never* administer cathartics or enemas, which may rupture the appendix. Give the patient nothing by mouth, and administer analgesics judiciously because they may mask symptoms.
- To lessen pain, place the patient in Fowler's position.

ALERT

Never apply heat to the right lower abdomen; this may cause the appendix to rupture.

An ice bag may be used for pain relief.

After appendectomy:

- Monitor the patient's vital signs and intake and output. Give analgesics, as ordered.
- Encourage the patient to cough, breathe deeply, and turn frequently to prevent pulmonary complications.
- Document bowel sounds, passing of flatus, and bowel movements. In a patient whose nausea and abdominal rigidity have subsided, these signs indicate readiness to resume oral fluids.
- Watch closely for possible surgical complications. Continuing pain and fever may signal an abscess. The complaint that “something gave way” may mean wound dehiscence. If an abscess or peritonitis develops, incision and drainage may be necessary. Frequently assess the dressing for wound drainage.
- Help the patient ambulate as soon as possible after surgery.
- In appendicitis complicated by peritonitis, a nasogastric tube may be needed to decompress the stomach and reduce nausea and vomiting. If so, record drainage and provide mouth and nose care.

Peritonitis

Peritonitis is an acute or chronic inflammation of the peritoneum, the membrane that lines the abdominal cavity and covers the visceral organs. Inflammation may extend throughout the peritoneum or may be localized as an abscess. Peritonitis commonly decreases intestinal motility and causes intestinal distention with gas. Mortality is 10%, with death usually a result of bowel obstruction; mortality was much higher before the introduction of antibiotics.

Causes and incidence

Although the GI tract normally contains bacteria, the peritoneum is sterile. When bacteria invade the peritoneum due to inflammation and perforation of the GI tract, peritonitis results. Bacterial invasion of the peritoneum typically results from appendicitis, diverticulitis, peptic ulcer, ulcerative colitis, volvulus, strangulated obstruction, abdominal neoplasm, or a stab wound. Peritonitis may also occur following chemical inflammation, as in the rupture of a fallopian or ovarian tube or the bladder, perforation of a gastric ulcer, or released pancreatic enzymes. It may also be associated with peritoneal dialysis.

In chemical and bacterial inflammation, accumulated fluids containing protein and electrolytes make the transparent peritoneum opaque, red, inflamed, and edematous. Because the peritoneal cavity is so resistant to contamination, infection is commonly localized as an abscess instead of disseminated as a generalized infection.

Complications

- Abscess
- Septicemia
- Respiratory compromise
- Bowel obstruction
- Shock

Signs and symptoms

The key symptom of peritonitis is sudden, severe, and diffuse abdominal pain that tends to intensify and localize in the area of the underlying disorder. For instance, if appendicitis causes the rupture, pain eventually localizes in the right lower quadrant. Many patients display weakness, pallor, excessive sweating, and cold skin as a result of excessive loss of fluid, electrolytes, and protein into the abdominal cavity. Decreased intestinal motility and paralytic ileus result from the effect of bacterial toxins on the intestinal muscles. Intestinal obstruction causes nausea, vomiting, and abdominal rigidity.

Other clinical characteristics include hypotension, tachycardia, signs and symptoms of dehydration (oliguria, thirst, dry swollen tongue, and

pinched skin), an acutely tender abdomen associated with rebound tenderness, temperature of 103° F (39.4° C) or higher, and hypokalemia. Inflammation of the diaphragmatic peritoneum may cause shoulder pain and hiccups. Abdominal distention and resulting upward displacement of the diaphragm may decrease respiratory capacity. Typically, the patient with peritonitis tends to breathe shallowly and move as little as possible to minimize pain. He may lie on his back, with his knees flexed, to relax abdominal muscles.

Diagnosis

Severe abdominal pain in a person with direct or rebound tenderness suggests peritonitis. Abdominal X-rays or computed tomography scan showing edematous and gaseous distention of the small and large bowel support the diagnosis. In the case of perforation of a visceral organ, the X-ray shows air lying under the diaphragm in the abdominal cavity. Other appropriate tests include the following:

- Chest X-ray may show elevation of the diaphragm.
- Blood studies show leukocytosis (more than 20,000/µl).
- Paracentesis reveals bacteria, exudate, blood, pus, or urine.
- Laparotomy may be necessary to identify the underlying cause.

Treatment

Early treatment of GI inflammatory conditions and preoperative and postoperative antibiotic therapy help prevent peritonitis. After peritonitis develops, emergency treatment must combat infection, restore intestinal

motility, and replace fluids and electrolytes.

Antibiotic therapy depends on the infecting organisms. If peritonitis is associated with peritoneal dialysis, antibiotics may be infused through the dialysis catheter; however, if the infection is severe, the catheter must be removed. To decrease peristalsis and prevent perforation, the patient should receive nothing by mouth; he should receive supportive fluids and electrolytes parenterally.

Other supplementary treatment measures include preoperative and postoperative administration of an analgesic, nasogastric (NG) intubation to decompress the bowel and, possibly, using a rectal tube to facilitate passage of flatus. When peritonitis results from perforation, surgery is necessary as soon as possible. Surgery aims to eliminate the source of infection by evacuating the spilled contents and inserting drains.

Special considerations

Patient care includes monitoring and measures to prevent complications and the spread of infection.

- Monitor the patient's vital signs, fluid intake and output, and amount of NG drainage or vomitus.
- Place the patient in semi-Fowler's position to help him deep-breathe with less pain and thus prevent pulmonary complications and to help localize purulent exudate in his lower abdomen or pelvis.

After surgery to evacuate the peritoneum:

- Maintain parenteral fluid and electrolyte administration, as ordered. Accurately record fluid intake and output, including NG tube and any drain output.
- Place the patient in semi-Fowler's position to promote drainage (through drainage tube) by gravity. Move him carefully because the slightest movement will intensify the pain.
- Implement other safety measures if fever and pain disorient the patient.
- Encourage and assist ambulation, as ordered, usually on the first postoperative day.
- Watch for signs of dehiscence (the patient may complain that “something gave way”) and abscess formation (continued abdominal tenderness and fever).
- Frequently assess for peristaltic activity by listening for bowel sounds and checking for gas, bowel movements, and a soft abdomen.
- Gradually decrease parenteral fluids and increase oral fluids.

Intestinal obstruction

Intestinal obstruction is the partial or complete blockage of the lumen in the small or large bowel. Small-bowel obstruction is far more common (90% of patients) and usually more serious. Complete obstruction in any part of the bowel, if untreated, can cause death within hours from shock and vascular collapse. Intestinal obstruction usually occurs after abdominal surgery or in persons with congenital bowel deformities.

Causes and incidence

Adhesions and strangulated hernias usually cause small-bowel obstruction; large-bowel obstruction is typically due to carcinomas. Mechanical intestinal obstruction results from foreign bodies (fruit pits, gallstones, or worms) or compression of the bowel wall due to stenosis, intussusception, volvulus of the sigmoid or cecum, tumors, or atresia. Nonmechanical obstruction results from physiologic disturbances, such as paralytic ileus, electrolyte imbalances, toxicity (uremia or generalized infection), neurogenic abnormalities (spinal cord lesions), and thrombosis or embolism of mesenteric vessels. (See *Paralytic ileus*.)

Intestinal obstruction develops in three forms:

- *Simple:* Blockage prevents intestinal contents from passing, with no other complications or with a change in blood flow.
- *Strangulated:* The blood supply to part or all of the obstructed section is cut off, in addition to blockage of the lumen.
- *Close-looped:* Both ends of a bowel section are occluded, isolating it from the rest of the intestine.

The physiologic effects are similar in all three forms of obstruction: When intestinal obstruction occurs, fluid, air, and gas

collect near the site. Peristalsis increases temporarily as the bowel tries to force its contents through the obstruction, injuring intestinal mucosa and causing distention at and above the site of the obstruction. Distention blocks the flow of venous blood and halts normal absorptive processes; as a result, the bowel begins to secrete water, sodium, and potassium into the fluid pooled in the lumen.

PARALYTIC ILEUS

Paralytic ileus is a physiologic form of intestinal obstruction that usually develops in the small bowel after abdominal surgery. It causes decreased or absent intestinal motility that usually recovers spontaneously after 2 to 3 days. Clinical effects of paralytic ileus include severe abdominal distention, extreme distress and, possibly, vomiting. The patient may be severely constipated or may pass flatus and small, liquid stools.

Causes

This condition can develop as a response to trauma, toxemia, or peritonitis or as a result of electrolyte deficiencies (especially hypokalemia) and the use of certain drugs, such as ganglionic blocking agents and anticholinergics. It can also result from vascular causes, such as thrombosis or embolism. Excessive air swallowing may contribute to it, but paralytic ileus brought on by this factor alone seldom lasts more than 24 hours.

Treatment

Paralytic ileus lasting longer than 48 hours requires intubation for decompression and nasogastric suctioning. Because of the absence of peristaltic activity, a long, weighted intestinal tube—called a *Miller-Abbott tube*—may be necessary in the patient with extraordinary abdominal distention. However, such procedures must be used with extreme caution because additional trauma to the bowel can aggravate ileus. When paralytic ileus results from surgical manipulation of the bowel, treatment may also include cholinergic agents, such as neostigmine or bethanechol.

When caring for patients with paralytic ileus, warn those receiving cholinergic agents to expect certain paradoxical

adverse effects, such as intestinal cramps and diarrhea. Remember that neostigmine produces cardiovascular adverse effects, usually bradycardia and hypotension. Check frequently for returning bowel sounds.

Obstruction in the small intestine results in metabolic alkalosis from dehydration and loss of gastric hydrochloric acid; lower bowel obstruction causes slower dehydration and loss of intestinal alkaline fluids, resulting in metabolic acidosis. Ultimately, intestinal obstruction may lead to ischemia, necrosis, and death. (See *Symptom progression in intestinal obstruction*, pages 276 and 277.)

ELDER TIP

Watch for air-fluid lock syndrome in older adults who remain recumbent for extended periods. In this syndrome, fluid first collects in the dependent bowel loops. Then peristalsis is too weak to push fluid "uphill." The resulting obstruction primarily occurs in the large bowel.

Complications

- Perforation
- Peritonitis
- Septicemia
- Secondary infection
- Metabolic acidosis or alkalosis
- Hypovolemic or septic shock

Signs and symptoms

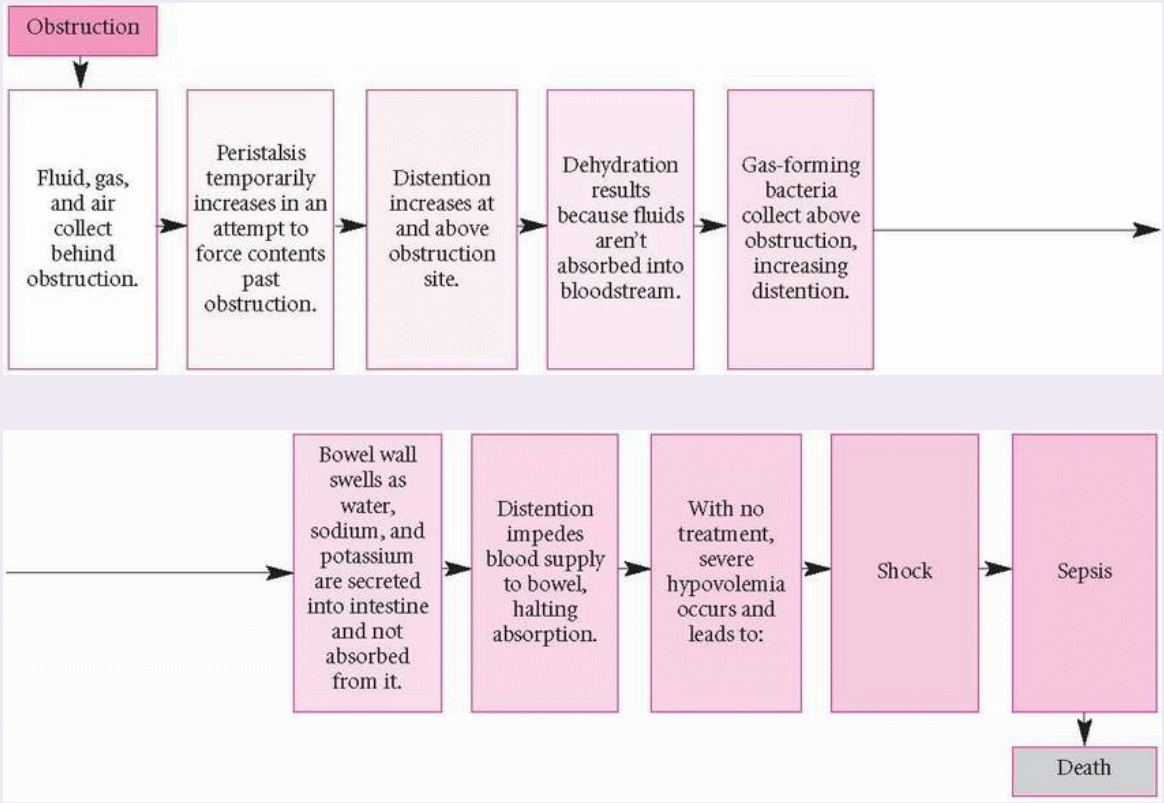
Colicky pain, nausea, vomiting, constipation, and abdominal distention characterize small-bowel obstruction. It may also cause drowsiness, intense thirst, malaise,

and aching and may dry up oral mucous membranes and the tongue. Auscultation reveals bowel sounds, borborygmi, and rushes; occasionally, these are loud enough to be heard without a stethoscope. Palpation elicits abdominal tenderness, with moderate distention; rebound tenderness occurs when obstruction has caused strangulation with ischemia. In late stages, signs of hypovolemic shock result from progressive dehydration and plasma loss.

PATHOPHYSIOLOGY

SYMPTOM PROGRESSION IN INTESTINAL OBSTRUCTION

A partial or complete blockage of the small or large intestine creates an obstruction with resultant symptoms.



In complete small-bowel obstruction, vigorous peristaltic waves propel bowel contents toward the mouth instead of the rectum. Spasms may occur every 3 to 5 minutes and last about 1 minute each, with persistent epigastric or periumbilical pain. Passage of small amounts of mucus and

blood may occur. The higher the obstruction, the earlier and more severe the vomiting. Vomitus initially contains gastric juice, then bile, and finally contents of the ileum.

Symptoms of large-bowel obstruction develop more slowly because the colon can absorb fluid from its contents and distend well beyond its normal size. Constipation may be the only clinical effect for days. Colicky abdominal pain may then appear suddenly, producing spasms that last less than 1 minute each and recur every few minutes. Continuous hypogastric pain and nausea may develop, but vomiting is usually absent at first. Large-bowel obstruction can cause dramatic abdominal distention; loops of the large bowel may become visible on the abdomen. Eventually, complete large-bowel obstruction may cause fecal vomiting, continuous pain, or localized peritonitis.

Patients with partial obstruction may display any of the previously discussed signs and symptoms in a milder form. However, leakage of liquid stool around the obstruction is common in partial obstruction.

Diagnosis

Progressive, colicky, abdominal pain and distention, with or without nausea and vomiting, suggest bowel obstruction.

CONFIRMING DIAGNOSIS

Tests that show obstruction include barium enema, abdominal computed tomography, upper GI and small-bowel series,

and abdominal films. Abdominal films show the presence and location of intestinal gas or fluid. In small-bowel obstruction, a typical "stepladder" pattern emerges, with alternating fluid and gas levels apparent in 3 to 4 hours. In large-bowel obstruction, barium enema reveals a distended, air-filled colon or a closed loop of sigmoid with extreme distention (in sigmoid volvulus).

Laboratory results supporting this diagnosis include:

- decreased sodium, chloride, and potassium levels (due to vomiting)
- a slightly elevated white blood cell count (with necrosis, peritonitis, or strangulation)
- an increased serum amylase level (possibly from irritation of the pancreas by bowel loop).

Treatment

Preoperative therapy consists of correction of fluid and electrolyte imbalances, decompression of the bowel to relieve vomiting and distention, and treatment of shock and peritonitis. Strangulated obstruction usually necessitates blood replacement as well as I.V. fluid administration. Decompression of the intestine may be accomplished with the use of a nasogastric (NG) tube inserted into the stomach or intestine to relieve distension and vomiting.

Close monitoring of the patient's condition determines the duration of treatment; if he fails to improve or if his condition deteriorates, surgery is necessary. In large-bowel obstruction, surgical resection with anastomosis, colostomy, or ileostomy commonly follows decompression with an NG tube.

Total parenteral nutrition may be appropriate if the patient suffers a protein deficit from chronic obstruction, postoperative or paralytic ileus, or infection. Drug therapy includes analgesics, sedatives, and antibiotics for peritonitis due to bowel strangulation or infarction.

Special considerations

Effective management of intestinal obstruction, a life-threatening condition that commonly causes overwhelming pain and distress, requires skillful supportive care and keen observation.

ALERT

Monitor the patient's vital signs frequently. A drop in blood pressure may indicate reduced circulating blood volume due to blood loss from a strangulated hernia.

Observe the patient closely for signs of shock (pallor, rapid pulse, and hypotension).

- Stay alert for signs and symptoms of metabolic alkalosis (changes in sensorium; slow, shallow respirations; hypertonic muscles; and tetany) or acidosis (shortness of breath on exertion, disorientation and, later, deep, rapid breathing, weakness, and malaise).
- Watch for signs and symptoms of secondary infection, such as fever and chills.
- Monitor the patient's urine output carefully to assess renal function, circulating blood volume, and possible urine retention caused by bladder compression by the distended intestine. If you suspect bladder compression, catheterize the patient for residual urine immediately after he has voided. Also, measure abdominal girth frequently to detect progressive distention.
- Provide mouth and nose care if the patient has vomited or undergone decompression by intubation. Look for signs of dehydration (thick, swollen tongue; dry, cracked lips; and dry oral mucous membranes).
- Record the amount and color of drainage from the decompression tube. Irrigate the tube with normal saline solution to maintain patency. If a weighted tube has been inserted, check periodically to make sure it's advancing. Help the patient turn from side to side (or walk around, if he can) to facilitate passage of the tube.
- Keep the patient in Fowler's position as much as possible to promote pulmonary ventilation and ease respiratory distress from abdominal distention. Listen for bowel sounds, and watch for signs of returning peristalsis (passage of flatus and mucus through the rectum).
- Explain all diagnostic and therapeutic procedures to the patient, and answer any questions he may have. Make sure that he understands that these procedures are necessary to relieve the obstruction and reduce pain. Tell him to lie on his left side for about a half hour before X-rays are taken.
- Prepare the patient and his family for the possibility of surgery, and provide emotional support and positive reinforcement afterward.

Arrange for an enterostomal therapist to visit the patient who has had an ostomy.

Inguinal hernia

A hernia occurs when part of an internal organ protrudes through an abnormal opening in the containing wall of its cavity. Hernias typically occur in the abdominal cavity. Although many kinds of abdominal hernias are possible, inguinal hernias (also called *ruptures*) are most common. (See *Common sites of hernia*.) In an inguinal hernia, the large or small intestine, omentum, or bladder protrudes into the inguinal canal. Hernias can be reduced (if the hernia can be manipulated back into place with relative ease), incarcerated (if the hernia can't be reduced because adhesions have formed, obstructing the intestinal flow), or strangulated (part of the herniated intestine becomes twisted or edematous, seriously interfering with normal blood flow and peristalsis, and possibly leading to intestinal obstruction and necrosis).

Causes and incidence

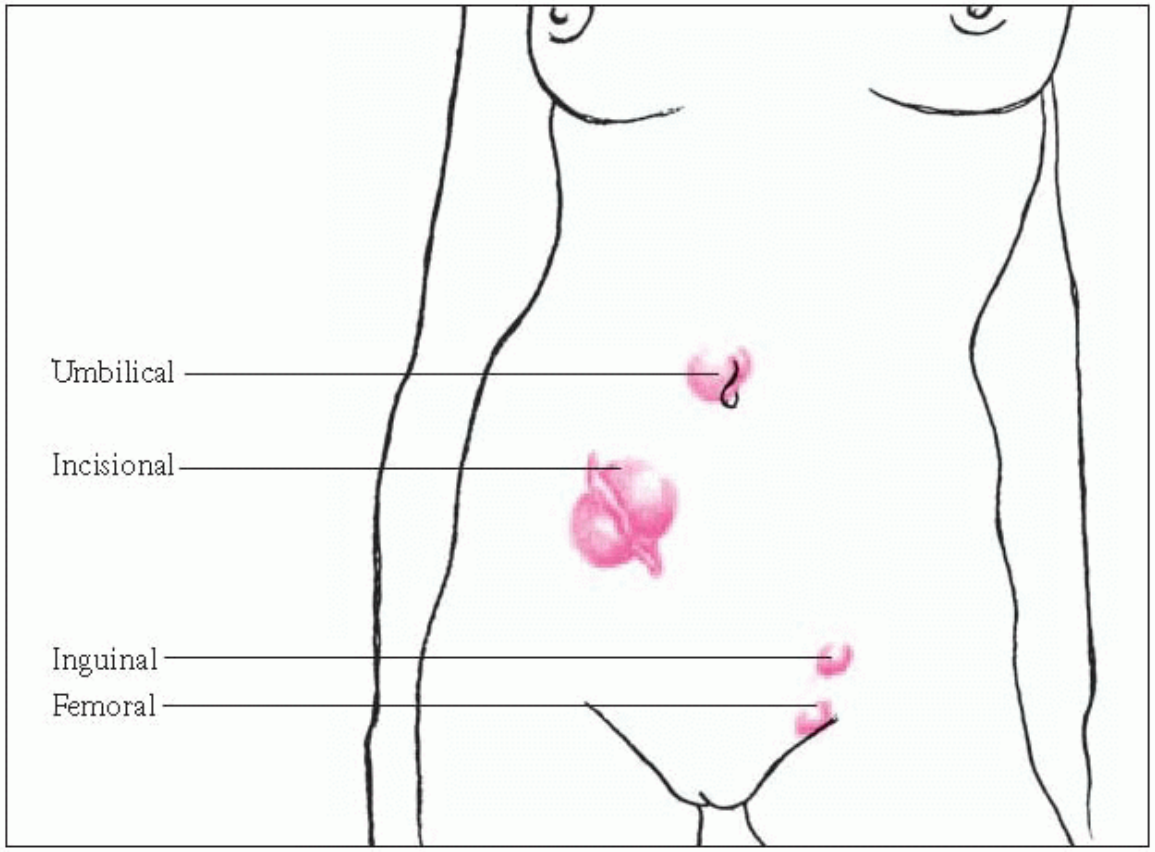
An inguinal hernia may be indirect or direct. An indirect inguinal hernia, the more common form, results from weakness in the fascial margin of the internal inguinal ring. In an indirect hernia, abdominal viscera leave the abdomen through the inguinal ring and follow the spermatic cord (in males) or round ligament (in females); they emerge at the external ring and extend down the inguinal canal, commonly into the scrotum or labia. An indirect inguinal hernia may develop at any age, is more common in males, and is especially prevalent in infants younger than age 1. According to the American Academy of Pediatrics, about 5 out of 100 children have inguinal hernias.

A direct inguinal hernia results from a weakness in the fascial floor of the inguinal canal. Instead of entering the canal through the internal ring, the hernia passes through the posterior inguinal wall, protrudes directly through the transverse fascia of the canal (in an area known as *Hesselbach's triangle*), and comes out at the external ring.

COMMON SITES OF HERNIA

Femoral hernia occurs where the femoral artery passes into the femoral canal. Typically, a fatty deposit within the femoral canal enlarges and eventually creates a hole big enough to accommodate part of the peritoneum and bladder. A femoral hernia appears as a swelling or bulge at the pulse point of the large femoral artery. It's usually a soft, pliable, reducible, nontender mass, but commonly becomes incarcerated or strangulated.

Umbilical hernia results from abnormal muscular structures around the umbilical cord. This hernia is quite common in neonates, but also occurs in women who are obese or who have had several pregnancies. Because most umbilical hernias in infants close spontaneously, surgery is warranted only if the hernia persists for more than 4 or 5 years. Taping or binding the affected area or supporting it with a truss may relieve symptoms until the hernia closes. Severe congenital umbilical hernia allows the abdominal viscera to protrude outside the body. This condition necessitates immediate repair.



Incisional (ventral) hernia develops at the site of previous surgery, usually along vertical incisions. This hernia may result from a weakness in the abdominal wall, perhaps as a result of an infection or impaired wound healing. Inadequate nutrition, extreme abdominal distention, or obesity also predispose the patient to incisional hernia. Palpation of an incisional hernia may reveal several defects in the surgical scar. Effective repair requires pulling the layers of the abdominal wall together without creating tension. If this isn't possible, surgical reconstruction uses Teflon, Marlex mesh, or tantalum mesh to close the opening.

Inguinal hernia can be direct or indirect. Indirect inguinal hernia causes the abdominal viscera to protrude through the inguinal ring and follow the spermatic cord (in males) or round ligament (in females). Direct inguinal hernia

results from a weakness in the fascial floor of the inguinal canal.

In males, during the seventh month of gestation, the testicle normally descends into the scrotum, preceded by the peritoneal sac. If the sac closes improperly, it leaves an opening through which the intestine can slip. In either sex, a hernia can result from weak abdominal muscles (caused by congenital malformation, trauma, or aging) or increased intra-abdominal pressure (due to heavy lifting, pregnancy, obesity, or straining).

About 10% of people develop some type of hernia during their lifetime, and more than 500,000 hernia operations are performed in the United States each year. Hernias are seven times more common in males than in females.

Complications

- Incarceration or strangulation of the bowel
- Intestinal obstruction
- Intestinal necrosis

Signs and symptoms

Inguinal hernia usually causes a lump to appear over the herniated area when the patient stands or strains. The lump disappears when the patient is supine. Tension on the herniated contents may cause a sharp, steady pain in the groin, which fades when the hernia is reduced. Strangulation produces severe pain and may lead to partial or complete bowel obstruction and even intestinal necrosis. Partial bowel obstruction may cause anorexia, vomiting, pain and tenderness in the groin, an irreducible mass, and diminished bowel sounds. Complete obstruction may cause shock, high fever, absent bowel sounds, and bloody stools.

PEDIATRIC TIP

In an infant, an inguinal hernia commonly coexists with an undescended testicle or a hydrocele.

Diagnosis

In a patient with a large hernia, physical examination reveals an obvious swelling or lump in the inguinal area. In the patient with a small hernia, the affected area may simply appear full. Palpation of the inguinal area while the patient is performing Valsalva's maneuver confirms the diagnosis. To detect a hernia in a male patient, the patient is asked to stand with his ipsilateral leg slightly flexed and his weight resting on the other leg. The examiner inserts an index finger into the lower part of the scrotum and invaginates the scrotal skin so the finger advances through the external inguinal ring to the internal ring (about 1½″ to 2″ [4 cm to 5 cm] through the inguinal canal). The patient is then told to cough. If the examiner feels pressure against the fingertip, an indirect hernia exists; if pressure is felt against the side of the finger, a direct hernia exists.

A patient history of sharp or “catching” pain when lifting or straining may help confirm the diagnosis. Suspected bowel obstruction requires X-rays and a white blood cell count (may be elevated).

Treatment

If the hernia is reducible, the pain may be temporarily relieved by pushing the hernia back into place. A truss may keep the abdominal contents from protruding into the hernial sac; however, this won't cure the hernia. This device is especially beneficial for an elderly or debilitated patient for whom surgery might be hazardous.

For infants, adults, and otherwise healthy elderly patients, herniorrhaphy is the treatment of choice. Herniorrhaphy replaces the contents of the hernial sac into the abdominal cavity and closes the opening. In many cases, this procedure is performed under local anesthesia in a short-term unit or as a single-day admission. Another effective surgical procedure for repairing hernia is hernioplasty, which reinforces the weakened area with steel mesh, fascia, or wire.

A strangulated or necrotic hernia necessitates bowel resection. Rarely, an extensive resection may require temporary colostomy. In either case, bowel resection lengthens postoperative recovery and requires antibiotics, parenteral fluids, and electrolyte replacement.

Special considerations

Care includes managing symptoms to increase patient comfort and prevent worsening.

- Apply a truss only after a hernia has been reduced. For best results, apply it in the morning, before the patient gets out of bed.
- To prevent skin irritation, tell the patient to bathe daily and apply cornstarch or baby powder. Warn against applying the truss over clothing because this reduces the effectiveness of the truss and may make it slip.

ALERT

If incarceration and strangulation occur, don't try to reduce the hernia because this may perforate the bowel. If severe intestinal obstruction develops because of hernial strangulation, inform the physician immediately. A nasogastric tube may be inserted promptly to empty the stomach and relieve pressure on the hernial sac.

- Before surgery for an incarcerated hernia, closely monitor the patient's vital signs. Administer I.V. fluids and analgesics, as ordered. Place the patient in Trendelenburg's position to reduce pressure on the hernia site.
- Give special reassurance and emotional support to a child and parents when hernia repair is scheduled. Encourage him to ask questions, and answer them as simply as possible. Offer appropriate diversions to distract him from the impending surgery.
- After outpatient surgery, make sure that the patient voids before he leaves the hospital. Teach him to check his incision and dressing for drainage, inflammation, or swelling and to watch for fever. If any of these occur, he should notify the physician.
- To reduce scrotal swelling, have the patient support the scrotum with a rolled towel and apply an ice bag.

- Instruct the patient to drink plenty of fluids to maintain hydration and prevent constipation.
- Before discharge, warn the patient against lifting heavy objects or straining during bowel movements. In addition, tell him to watch for signs and symptoms of infection (oozing, tenderness, warmth, and redness) at the incision site and to keep the incision clean and covered until the sutures are removed.
- Advise the patient not to resume normal activity or return to work without the surgeon's permission.

Intussusception

Intussusception is a telescoping (invagination) of a portion of the bowel into an adjacent distal portion. Intussusception may be fatal, especially if treatment is delayed for more than 24 hours.

Causes and incidence

When a bowel segment (the intussusceptum) invaginates, peristalsis propels it along the bowel, pulling more bowel along with it; the receiving segment is the intussuscipiens. This invagination produces edema, hemorrhage from venous engorgement, incarceration, and obstruction. If treatment is delayed for longer than 24 hours, strangulation of the intestine usually occurs, with gangrene, shock, and perforation. (See *Understanding intussusception*, page 282.)

Seasonal peaks—in the spring-summer, coinciding with peak incidence of enteritis, and in the midwinter, coinciding with peak incidence of respiratory tract infections—have been noted in the incidence of intussusception, suggesting a connection to viral infections. Other potential causes include a mass, such as a lymph node, polyp, or tumor that telescopes the gut and leads to intussusception.

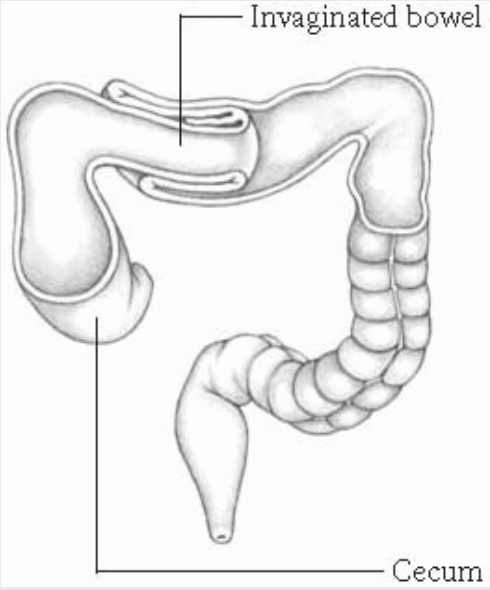
Although it can affect adults, intussusception is most common in infants ages 6 months to 1 year. It occurs twice as often in male infants as in female infants.

Complications

- Strangulation of intestine
- Gangrene
- Shock
- Perforation
- Peritonitis

UNDERSTANDING INTUSSUSCEPTION

In intussusception, a bowel section invaginates and is propelled along by peristalsis, pulling in more bowel. In this illustration, a portion of the cecum invaginates and is propelled into the large intestine. Intussusception typically produces edema, hemorrhage from venous engorgement, incarceration, and obstruction.



Signs and symptoms

PEDIATRIC TIP

In an infant or child, intussusception produces four cardinal clinical effects:

- *Intermittent attacks of colicky pain cause the child to scream, draw his legs up to his abdomen, turn pale and diaphoretic and, possibly, display grunting respirations.*
- *Vomiting of stomach contents may occur initially, followed by further vomiting of bilestained or fecal material.*
- *"Currant-jelly" stools, containing a mixture of blood and mucus, may be observed.*
- *The patient will have a tender, distended abdomen, with a palpable, sausage-shaped abdominal mass; the viscera are usually absent from the right lower quadrant.*

In adults, intussusception produces nonspecific, chronic, and intermittent symptoms, including colicky abdominal pain and tenderness, vomiting, diarrhea (occasionally constipation), bloody stools, and weight loss. Abdominal pain usually localizes in the right lower quadrant, radiates to the back, and increases with eating. Adults with severe intussusception may develop strangulation with excruciating pain, abdominal distention, and tachycardia.

Diagnosis

CONFIRMING DIAGNOSIS

Barium enema confirms colonic intussusception when it shows the characteristic coiled-spring sign; it also delineates the extent of intussusception.

Upright abdominal X-rays may show a soft-tissue mass and signs of complete or partial obstruction, with dilated loops of bowel. Signs of dehydration or shock support the diagnosis, as does the presence of a palpable mass in the abdomen.

Treatment

In children, therapy may include hydrostatic reduction or surgery. Surgery is indicated for children with recurrent intussusception, for

those who show signs of shock or peritonitis, and for those in whom symptoms have been present longer than 24 hours. In adults, surgery is always the treatment of choice.

During hydrostatic reduction, the radiologist drips a barium solution into the rectum from a height of no more than 3′ (1 m); fluoroscopy traces the progress of the barium. If the procedure is successful, the barium backwashes into the ileum, and the mass disappears. If not, the procedure is stopped, and the patient is prepared for surgery.

During surgery, manual reduction is attempted first. After compressing the bowel above the intussusception, the physician attempts to milk the intussusception back through the bowel. However, if manual reduction fails or if the bowel is gangrenous or strangulated, the physician will perform a resection of the affected bowel segment.

Special considerations

Care focuses on changes in the patient's condition that might indicate worsening.

ALERT

Monitor the patient's vital signs before and after surgery. A change in temperature may indicate sepsis; infants may become hypothermic at the onset of infection. Rising pulse rate and falling blood pressure may be signs of peritonitis.

- Check the patient's intake and output. Watch for signs of dehydration and bleeding.
- A nasogastric (NG) tube is inserted to decompress the intestine and minimize vomiting. Monitor tube drainage and replace volume lost, as ordered.
- Monitor the patient who has undergone hydrostatic reduction for passage of stools and barium, a sign that the reduction was successful. Keep in mind that the patient may have a recurrence of intussusception, usually within the first 24 to 36 hours after the reduction.

- After surgery, administer antibiotics, as ordered. Closely check the incision for inflammation, drainage, or suture separation.
- Encourage the patient to deep-breathe and cough productively. Be sure to splint the incision when he coughs, or teach him to do so himself.
- The NG tube may be removed when bowel sounds and peristalsis resume. The patient's diet can be advanced as tolerated.
- Check for abdominal distention after the patient resumes a normal diet and monitor his general condition.
- Offer special reassurance and emotional support to the child and parents. This condition is considered a pediatric emergency, and parents are generally unprepared for their child's hospitalization and possible surgery; they may feel guilty for not seeking medical aid sooner. Similarly, the child is unprepared for a separation from his parents and home.

To minimize the stress of hospitalization, encourage parents to participate in their child's care as much as possible.

Volvulus

Volvulus is a twisting of the intestine at least 180 degrees on its mesentery, which results in blood vessel compression and ischemia.

Causes and incidence

Twisting in volvulus may result from an anomaly of rotation, an ingested foreign body, or an adhesion; in some cases, however, the cause is unknown. Volvulus usually occurs in a bowel segment with a mesentery long enough to twist. The most common area, particularly in adults, is the sigmoid; the small bowel is a common site in children. Other common sites include the stomach and cecum. Volvulus secondary to meconium ileus may occur in patients with cystic fibrosis.

Acute gastric volvulus has a mortality rate of 42% to 56%. There's no racial predilection, and it affects males and females equally. Peak incidence occurs in people ages 40 to 50, but about 20% of cases occur in infants younger than age 1.

Complications

- Strangulation of the twisted intestinal loop
- Ischemia
- Infarction
- Perforation
- Fatal peritonitis

Signs and symptoms

Vomiting and rapid, marked abdominal distention follow sudden onset of severe abdominal pain. Nausea, vomiting, bloody stools, constipation, and shock may occur. Without immediate treatment, volvulus can lead to strangulation of the twisted bowel loop, ischemia, infarction, perforation, and fatal peritonitis.

Diagnosis

The sudden onset of severe abdominal pain and physical examination that may reveal a palpable mass suggest volvulus. Appropriate tests include the following:

- X-rays—Abdominal X-rays may show obstruction and abnormal air-fluid levels in the sigmoid and cecum; in midgut volvulus, abdominal X-rays may be normal.
- Computed tomography scan—may show evidence of intestinal obstruction.
- Barium enema—In cecal volvulus, barium fills the colon distal to the section of cecum; in sigmoid volvulus in children, barium may twist to a point and, in adults,

  it may take on an “ace of spades” configuration.
- Upper GI series (with small bowel follow-through)—In midgut volvulus, obstruction and possibly a twisted contour show in a narrow area near the duodenojejunal junction, where barium won't pass.

- White blood cell count—In strangulation, the count is greater than 15,000/µl; in bowel infarction, greater than 20,000/µl.

Treatment

Treatment varies according to the severity and location of the volvulus. For children with midgut volvulus, treatment is surgical. For adults with sigmoid volvulus, a proctoscopic examination is performed to check for infarction, and nonsurgical treatment includes reduction by careful insertion of a sigmoidoscope or a long rectal tube to deflate the bowel.

Success of nonsurgical reduction is indicated by expulsion of gas and immediate relief from abdominal pain. If the bowel is distended but viable, surgery consists of detorsion (untwisting); if the bowel is necrotic, surgery includes resection and anastomosis.

Special considerations

After surgical correction of volvulus:

- Monitor the patient's vital signs, watching for temperature changes (a sign of sepsis) and a rapid pulse rate and falling blood pressure (signs of shock and peritonitis). Carefully monitor fluid intake and output (including stools), electrolyte values, and complete blood count. Be sure to measure and record drainage from the nasogastric (NG) tube and drains.
- Encourage frequent coughing and deep breathing with splinting of the incision.
- Reposition the patient often and perform suction as needed.
- Record excessive or unusual drainage.
- Check for incisional inflammation and separation of sutures.
- When bowel sounds and peristalsis return, remove the NG tube and begin oral feedings with clear liquids, as ordered. When solid food can be tolerated, gradually expand the diet. Reassure the patient and his family appropriately, and explain all diagnostic procedures. If the patient is a child, encourage parents to participate in their child's care to minimize the stress of hospitalization.

Inactive colon

Inactive colon, also known as *lazy colon, colonic stasis,* or *atonic constipation,* is a state of chronic constipation that, if untreated, may lead to fecal impaction.

Causes and incidence

Inactive colon usually results from some deficiency in the three elements necessary for normal bowel activity: dietary bulk, fluid intake, and exercise. It's common in bedridden people because of their inactivity and is generally relieved with diet and exercise. Other possible causes can include habitual disregard of the impulse to defecate, emotional conflicts, chronic use of laxatives, or prolonged dependence on enemas, which dull rectal sensitivity to the presence of feces.

Signs and symptoms

The primary symptom of inactive colon is chronic constipation. The patient commonly strains to produce hard, dry stools accompanied by mild abdominal discomfort. Straining can aggravate other rectal conditions such as hemorrhoids.

Diagnosis

A patient history of dry, hard, infrequent stools suggests inactive colon. A digital rectal examination reveals stool in the lower portion of the rectum and a palpable colon. Proctoscopy may show an unusually small colon lumen, prominent veins, and an abnormal amount of mucus. Diagnostic tests to rule out other causes include upper GI series, barium enema, and examination of stool for occult blood from neoplasms.

Treatment

Treatment varies according to the patient's age and condition. A higher-bulk diet, sufficient exercise, and increased fluid intake commonly relieve constipation. Treatment for severe constipation may include bulk-forming laxatives, such as psyllium, or well-lubricated glycerin suppositories; for

fecal impaction, manual removal of feces is necessary. Administration of an oilretention enema usually precedes removal; an enema is also necessary afterward. For lasting relief from constipation, the patient with inactive colon must modify bowel habits.

PREVENTION

PREVENTING CONSTIPATION

Recommend these lifestyle changes to your patients to help them avoid constipation.

Increase fluids

- Advise the patient to drink at least 8 to 10 glasses (2 qt [2 L]) of liquid every day because fluids help keep the intestinal contents in a semisolid state for easier passage. This is particularly important for an older patient. Stimulate the bowel with a drink of hot coffee, warm lemonade, iced liquids—plain or with lemon—or prune juice before breakfast or in the evening.

Add fiber

- Adding fiber to the diet with foods such as whole grain cereals (rolled oats, bran, shredded wheat, brown rice, whole wheat bread, and oatmeal) to contribute bulk and induces peristalsis. However, too much bran can create an irritable bowel, so keep watch of the fiber content (low fiber—0.3 to 1 g; moderate fiber—1.1 to 2 g; high fiber—2.1 to 4.2 g). Include fresh fruits with skins as well as raw and coarse vegetables (broccoli, brussel sprouts, cabbage, cauliflower, cucumbers, lettuce, and turnips) in the diet for additional bulk.

Avoid fats

- Moderate the consumption of fatcontaining foods, such as bacon, butter, cream, and oil. Although these foods

will help to soften intestinal contents, they sometimes cause diarrhea.

- Instruct the patient to avoid highly refined foods, such as white rice, cream of wheat, farina, white pastries, pie or cake, macaroni, spaghetti, noodles, candy, cookies, and ice cream.

Exercise

- The patient should incorporate moderate exercise, such as walking, into his daily routine.

Take prescribed medications

- Bulk-forming laxatives, such as psyllium, should be taken with at least 8 oz (240 ml) of liquid. Juices, soft drinks, or other pleasant-tasting liquids help mask the gritty texture of these laxatives.

What to avoid

- Avoid overusing laxatives and maintain a regular time for bowel movements (usually after breakfast). Autosuggestion, relaxation, pleasant reading material, privacy, and use of a small footstool to promote thigh flexion while sitting on the toilet may be helpful. The patient should respond promptly to the urge to defecate. If he worries about constipation, explain that a 2- to 3-day interval between bowel movements can be normal.
- Advise the patient against overusing enemas. Frequent use of sodium biphosphate, in particular, should be avoided because its hypertonic solution can absorb as much as 10% of the colon's sodium content or draw intestinal fluids into the colon, thereby causing dehydration.

Special considerations

In many cases, patient education can help break the constipation habit. (See *Preventing constipation.*)

ELDER TIP

If an older patient with inactive colon is hospitalized, help him move to a bedside commode for bowel movements because using a bedpan causes additional strain. However, if he must use a bedpan, have him sit in Fowler's position or sit on the pan at the side of his bed to facilitate elimination. Occasional digital rectal stimulation or abdominal massage near the sigmoid area may help stimulate a bowel movement.

CHRONIC PANCREATITIS

Chronic pancreatitis is associated with alcoholism in over 50% of patients, but it also may follow hyperparathyroidism (causing hypercalcemia), hyperlipidemia or, infrequently, gallstones, trauma, or peptic ulcer. Inflammation and fibrosis cause progressive pancreatic insufficiency and eventually destroy the pancreas.

Symptoms of chronic pancreatitis include constant dull pain with occasional exacerbations, malabsorption, severe weight loss, and hyperglycemia (leading to diabetic symptoms). Diagnosis is based on the patient history, X-rays showing pancreatic calcification, an elevated erythrocyte sedimentation rate, and examination of stool for steatorrhea.

In many cases, the severe pain of chronic pancreatitis requires large doses of analgesics or opioids, making

addiction a serious problem. Treatment also includes a low-fat diet and oral administration of pancreatic enzymes, such as pancreatin or pancrelipase, to control steatorrhea; insulin or oral hypoglycemics to curb hyperglycemia; and, occasionally, surgical repair of biliary or pancreatic ducts or the sphincter of Oddi to reduce pressure and promote the flow of pancreatic juice. The prognosis is good if the patient can avoid alcohol, but poor if he can't.

ALERT

If the patient has a history of arteriosclerosis, heart failure, or hypertension, constipation and straining may induce Valsalva's maneuver, thereby causing a vagal effect, in which the heart rate slows or stops entirely.

Pancreatitis

Pancreatitis, inflammation of the pancreas, occurs in acute and chronic forms and may be due to edema, necrosis, or hemorrhage. In men, this disease is commonly associated with alcoholism, trauma, or peptic ulcer; in women, it's linked to biliary tract disease. The prognosis is good when pancreatitis follows biliary tract disease, but poor when it follows alcoholism. Mortality rises as high as 60% when pancreatitis is associated with necrosis and hemorrhage. (See *Chronic pancreatitis*.)

Causes and incidence

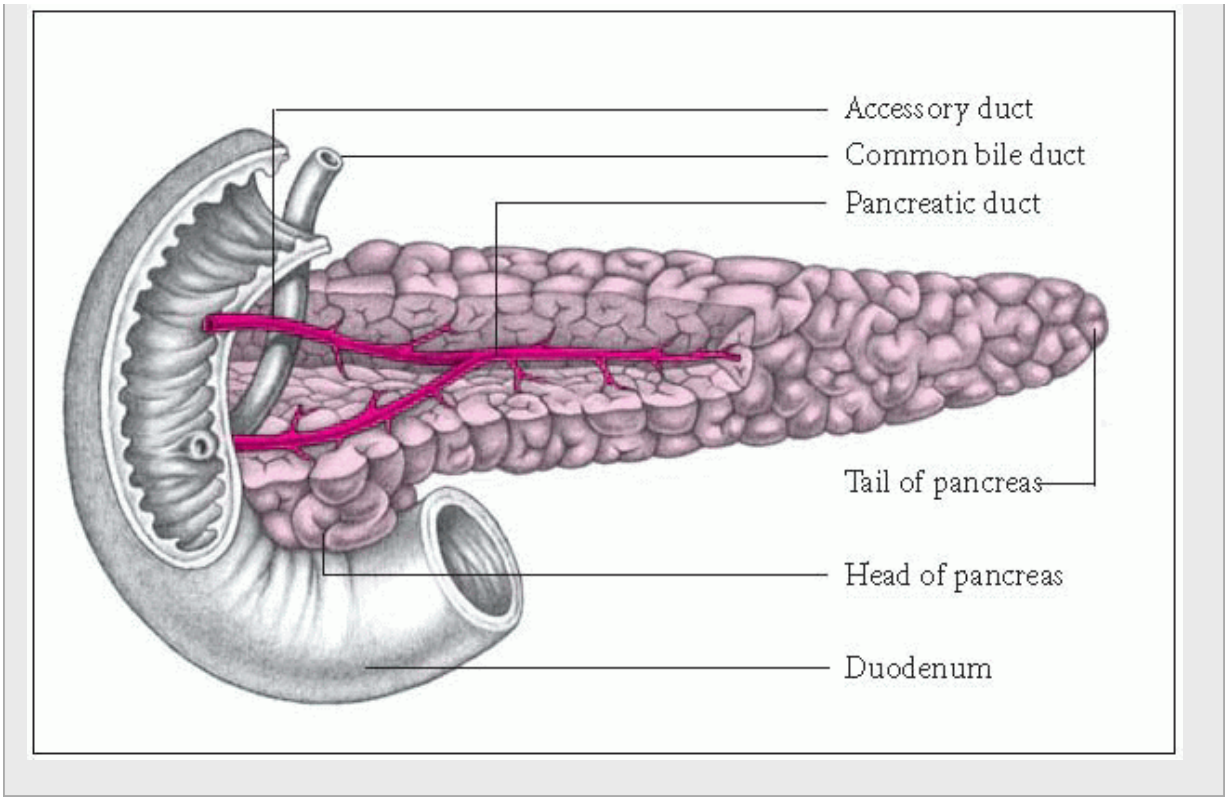
The most common causes of pancreatitis are biliary tract disease and alcoholism, but it can also result from pancreatic cancer, trauma, or use of certain drugs, such as glucocorticoids, sulfonamides, chlorothiazide, and azathioprine. This disease also may develop as a complication of peptic ulcer, mumps, or hypothermia. Rarer causes are stenosis or obstruction of the sphincter of Oddi, hyperlipidemia, metabolic endocrine disorders (hyperparathyroidism and hemochromatosis), vasculitis or vascular disease, viral infections, mycoplasmal pneumonia, and pregnancy.

Diabetes, pancreatic insufficiency, and calcification occur in young people, probably from malnutrition and alcoholism, and lead to pancreatic atrophy. Regardless of the cause, pancreatitis involves autodigestion: The enzymes normally excreted by the pancreas digest pancreatic tissue. (See *Anatomy of the pancreas*.)

The incidence of acute pancreatitis varies with age. In the United States, it affects 270 of every 100,000 people ages 15 to 44 and 540 of every 100,000 people age 65 and older. It's uncommon in children. Blacks have a higher incidence than Whites. Men and women are affected equally. Among people with acquired immunodeficiency syndrome, it affects 4 to 22 people out of every 100.

ANATOMY OF THE PANCREAS

Pancreatitis is an inflammation of the pancreas involving irritation and infection of the organ. Obstruction or overdistention of the pancreatic duct can start the inflammation that occurs with pancreatitis. Exposure to toxins and other underlying causes also may start it. The overall effect is a release of activated pancreatic destructive enzymes into the pancreas and surrounding tissue.



Complications

- Diabetes mellitus (if islets of Langerhans are damaged)
- Massive hemorrhage
- Destruction of pancreas
- Diabetes acidosis
- Shock
- Coma
- Adult respiratory distress syndrome
- Atelectasis
- Pulmonary effusion
- Pneumonia
- GI bleeding
- Pancreatic abscess

Signs and symptoms

In many patients, the first and only symptom of mild pancreatitis is steady epigastric pain centered close to the umbilicus, radiating between the tenth thoracic and sixth lumbar vertebrae, and unrelieved by vomiting. However, a severe attack causes extreme pain, persistent vomiting, abdominal rigidity, diminished bowel activity (suggesting peritonitis), crackles at lung bases, and left pleural effusion. Progression produces extreme malaise and restlessness, with mottled skin, tachycardia, low-grade fever (100° to 102° F [37.7° to 38.8° C]), and cold, sweaty extremities. The proximity of the inflamed pancreas to the bowel may cause ileus.

If pancreatitis damages the islets of Langerhans, complications may include diabetes mellitus. Fulminant pancreatitis causes massive hemorrhage and total destruction of the pancreas, resulting in diabetic acidosis, shock, or coma.

Diagnosis

A thorough patient history (especially for alcoholism) and physical examination are the first steps in diagnosis, but the retroperitoneal position of the pancreas makes physical assessment difficult.

CONFIRMING DIAGNOSIS

Dramatically elevated serum amylase levels—in many cases over 500 units/L—confirm pancreatitis and rule out perforated peptic ulcer, acute cholecystitis, appendicitis, and bowel infarction or obstruction. Similarly, dramatic elevations of amylase also occur in urine, ascites, or pleural fluid. Characteristically, amylase levels return to normal 48 hours after the onset of pancreatitis, despite continuing symptoms.

Supportive laboratory values include:

- increased serum lipase levels, which rise more slowly than serum amylase

- low serum calcium levels (hypocalcemia) from fat necrosis and formation of calcium soaps
- white blood cell counts ranging from 8,000 to 20,000/µl, with increased polymorphonuclear leukocytes
- elevated glucose levels—as high as 500 to 900 mg/dl, indicating hyperglycemia.

Tests used to diagnose pancreatitis may include the following:

- Abdominal X-rays or computed tomography (CT) scans show dilation of the small or large bowel or calcification of the pancreas.
- Ultrasound or CT scan reveals an increased pancreatic diameter and helps distinguish acute cholecystitis from acute pancreatitis.

Treatment

The goal of therapy is to maintain circulation and fluid volume. Treatment measures must also relieve pain and decrease pancreatic secretions.

Emergency treatment of shock (which is the most common cause of death in earlystage pancreatitis) consists of vigorous I.V. replacement of electrolytes and proteins. Metabolic acidosis that develops secondary to hypovolemia and impaired cellular perfusion requires vigorous fluid volume replacement.

Drug treatment may include meperidine for pain, diazepam for restlessness and agitation, and antibiotics for bacterial infections. Morphine and codeine are usually avoided as pain medications because of their effect on the sphincter of Oddi. If the patient has hypocalcemia, he'll need an infusion of 10% calcium gluconate; if he has elevated serum glucose levels, he may require insulin therapy.

After the emergency phase, continuing I.V. therapy should provide adequate electrolytes and protein solutions that don't stimulate the pancreas for 5 to 7 days. If the patient isn't ready to resume oral feedings by then, total parenteral nutrition (TPN) may be necessary. Nonstimulating elemental gavage feedings may be safer because of the decreased risk of infection and overinfusion. In extreme cases,

laparotomy to debride the pancreatic bed, partial pancreatectomy, or a combination of both and feeding jejunostomy may be necessary.

Special considerations

Acute pancreatitis is a life-threatening emergency, requiring meticulous supportive care and continuous monitoring of vital systems.

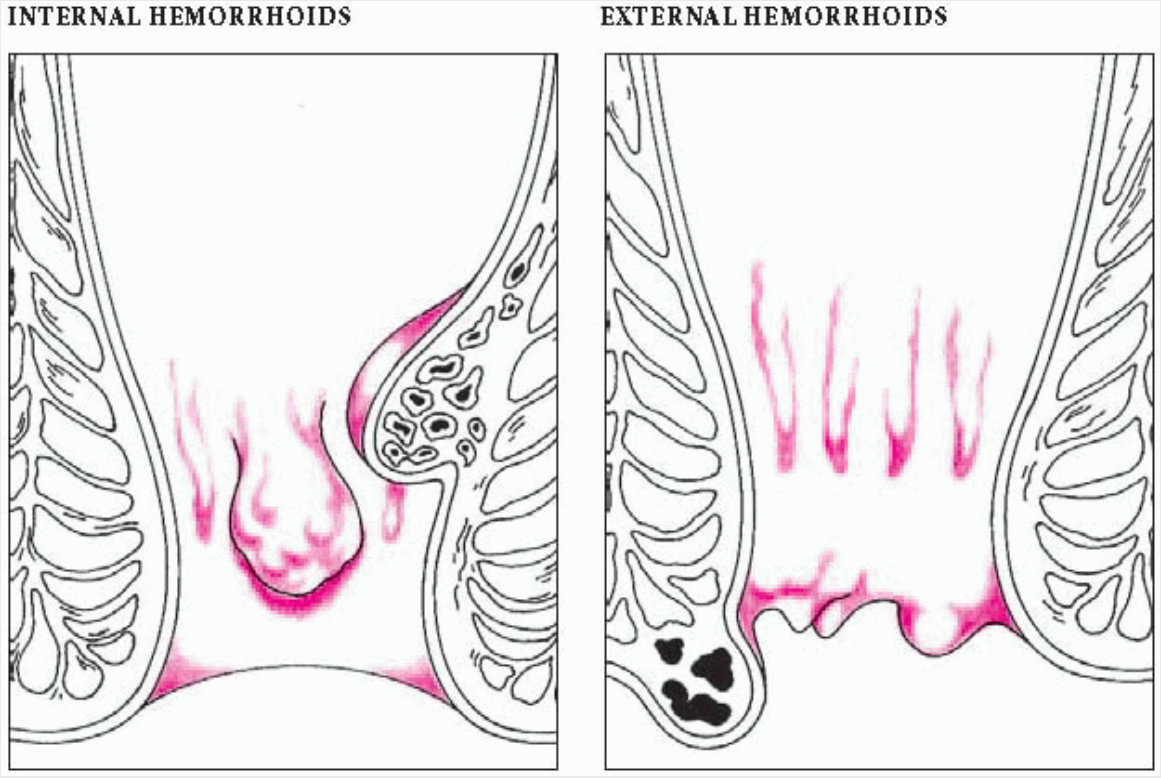
- Monitor the patient's vital signs and pulmonary artery pressure or central venous pressure closely. Give plasma or albumin, if ordered, to maintain blood pressure. Record fluid intake and output; check urine output hourly, and monitor electrolyte levels. Assess for crackles, rhonchi, or decreased breath sounds.
- For bowel decompression, maintain constant or intermittent nasogastric suctioning, and give nothing by mouth. Perform good mouth and nose care.
- Watch for signs and symptoms of calcium deficiency—tetany, cramps, carpopedal spasm, and seizures. If you suspect hypocalcemia, keep airway and suction apparatus handy and pad side rails.
- Administer analgesics as needed to relieve the patient's pain and anxiety. Remember that anticholinergics reduce salivary and sweat gland secretions. Warn the patient that he may experience dry mouth and facial flushing. *Caution:* Narrow-angle glaucoma contraindicates the use of atropine or its derivatives.
- Monitor glucose levels.
- Watch for complications due to TPN, such as sepsis, hypokalemia, overhydration,

  and metabolic acidosis. Watch for fever, cardiac irregularities, changes in arterial blood gas measurements, and deep respirations. Use strict aseptic technique when caring for the catheter insertion site.

TYPES OF HEMORRHOIDS

Covered by mucosa, *internal hemorrhoids* bulge into the rectal lumen and may prolapse during defecation. Covered by skin, *external hemorrhoids* protrude from the

rectum and are more likely to thrombose than internal hemorrhoids. The illustrations below show frontal and cross-sectional views.



ANORECTUM

Hemorrhoids

Hemorrhoids are varicosities in the superior or inferior hemorrhoidal venous plexus. Dilation and enlargement of the superior plexus produce internal hemorrhoids; dilation and enlargement of the inferior plexus produce external hemorrhoids that may protrude from the rectum. (See *Types of hemorrhoids*.) Hemorrhoids occur in both sexes; incidence is usually highest between ages 20 and 50.

Causes and incidence

Hemorrhoids probably result from increased venous pressure in the hemorrhoidal plexus. Predisposing factors include occupations that

require prolonged standing or sitting; straining due to constipation, diarrhea, coughing, sneezing, or vomiting; heart failure; hepatic disease, such as cirrhosis, amebic abscesses, or hepatitis; alcoholism; anorectal infections; loss of muscle tone due to old age, rectal surgery, or episiotomy; anal intercourse; and pregnancy.

Hemorrhoids are more common in whites, in persons of higher socioeconomic classes, and in persons who live in rural areas. However, actual incidence figures are

unknown because many patients with hemorrhoids self-medicate.

Complications

- Local infection
- Thrombosis

Signs and symptoms

Although hemorrhoids may be asymptomatic, they characteristically cause painless, intermittent bleeding, which occurs on defecation. Bright red blood appears on stool or on toilet paper due to injury of the fragile mucosa covering the hemorrhoid. These first-degree hemorrhoids may itch because of poor anal hygiene. When second-degree hemorrhoids prolapse, they're usually painless and spontaneously return to the anal canal following defecation. Third-degree hemorrhoids cause constant discomfort and prolapse in response to any increase in intra-abdominal pressure. They must be manually reduced. Thrombosis of external hemorrhoids produces sudden rectal pain and a subcutaneous, large, firm lump that the patient can feel. If hemorrhoids cause severe or recurrent bleeding, they may lead to secondary anemia with significant pallor, fatigue, and weakness; however, such systemic complications are rare.

Diagnosis

Physical examination confirms external hemorrhoids. Proctoscopy confirms internal hemorrhoids and rules out rectal polyps.

Treatment

Treatment depends on the type and severity of the hemorrhoid and on the patient's overall condition. Generally, treatment includes measures to ease pain, combat swelling and congestion, and regulate bowel habits. The patient can relieve constipation by increasing the amount of raw vegetables, fruit, and whole grain cereal in the diet or by using stool softeners. Venous congestion can be prevented by avoiding prolonged sitting; local swelling and pain can be decreased with local anesthetic agents (lotions, creams, or suppositories), astringents, or cold compresses, followed by warm sitz baths or thermal packs. Rarely, the patient with chronic, profuse bleeding may require a blood transfusion. Other nonsurgical treatments are injection of a sclerosing solution to produce scar tissue that decreases prolapse, manual reduction, and hemorrhoid ligation or laser ablation.

Hemorrhoidectomy, the most effective treatment, is necessary for patients with severe bleeding, intolerable pain and pruritus, and large prolapse. This procedure is contraindicated in patients with blood dyscrasias (acute leukemia, aplastic anemia, or hemophilia) or GI carcinoma and during the first trimester of pregnancy.

Special considerations

Patient care includes preoperative and postoperative support.

- To prepare the patient for hemorrhoidectomy, administer an enema as appropriate (usually 2 to 4 hours before surgery), and record results. Prepare the area as ordered.
- Postoperatively, check for signs of prolonged rectal bleeding, administer adequate analgesics, and provide sitz baths as ordered.
- As soon as the patient can resume oral feedings, administer a bulk medication, such as psyllium, about 1 hour after the evening meal, to ensure a daily stool. Warn against using stool-softening medications soon after hemorrhoidectomy because a firm stool acts as a natural dilator to prevent anal stricture from the scar tissue. (The patient may need repeated digital dilation to prevent such narrowing.)
- Keep the wound site clean to prevent infection and irritation.

- Before discharge, stress the importance of regular bowel habits and good anal hygiene. Warn against too-vigorous wiping with washcloths and using harsh soaps. Encourage the use of medicated astringent pads and white toilet paper (the fixative in colored paper can irritate the skin).

Anorectal abscess and fistula

Anorectal abscess is a localized collection of pus due to inflammation of the soft tissue near the rectum or anus. Inflammation

may produce an anal fistula—an abnormal opening in the anal skin—that may communicate with the rectum.

Causes and incidence

The inflammatory process that leads to abscess may begin with an abrasion or tear in the lining of the anal canal, rectum, or perianal skin and subsequent infection by *Escherichia coli,* staphylococci, or streptococci. Trauma may result from injections for treatment of internal hemorrhoids, enematip abrasions, puncture wounds from ingested eggshells or fish bones, or insertion of foreign objects. Other preexisting lesions include infected anal fissure, infections from the anal crypt through the anal gland, ruptured anal hematoma, prolapsed thrombosed internal hemorrhoids, and septic lesions in the pelvis, such as acute appendicitis, acute salpingitis, and diverticulitis. Systemic illnesses that may cause abscesses include ulcerative colitis and Crohn's disease. However, many abscesses develop without preexisting lesions.

As the abscess produces more pus, a fistula may form in the soft tissue beneath the muscle fibers of the sphincters (especially the external sphincter), usually extending into the perianal skin. The internal (primary) opening of the abscess or fistula is usually near the anal glands and crypts; the external (secondary) opening, in the perianal skin.

The peak incidence of anorectal abscess occurs in people in their 30s and 40s, but there's also a high occurrence in infants. Men are affected two to three times more often than women. About 30% of patients have a previous history of abscess.

Complications

- Anorectal fistula
- Perineal cellulitis
- Scar tissue formation
- Anal stricture

Signs and symptoms

Characteristics are throbbing pain and tenderness at the site of the abscess. A hard, painful lump develops on one side, preventing comfortable sitting. Discharge of pus may occur from the rectum, and there may be constipation or pain associated with bowel movements.

Diagnosis

Anorectal abscess is detectable on physical examination. If the abscess drains by forming a fistula, the pain usually subsides and the major signs become pruritic drainage and subsequent perianal irritation. The external opening of a fistula generally appears as a pink or red, elevated, discharging sinus or ulcer on the skin near the anus. Depending on the infection's severity, the patient may have chills, fever, nausea, vomiting, and malaise. Digital examination may reveal a palpable indurated tract and a drop or two of pus on palpation. The internal opening may be palpated as a depression or ulcer in the midline anteriorly or at the dentate line posteriorly. Examination with a probe may require an anesthetic. A proctosigmoidoscopy may be performed to exclude associated diseases.

Treatment

Anorectal abscesses require surgical incision under caudal anesthesia to promote drainage. Fistulas require a fistulotomy—removal of the fistula and associated granulation tissue—under caudal anesthesia. If the fistula tract is epithelialized, treatment requires fistulectomy—removal of the fistulous tract—followed by insertion of drains, which remain in place for

48 hours. Warm sitz baths are useful to relieve inflammation; however, pain medication and antibiotics may be needed.

Special considerations

After incision to drain anorectal abscess, follow these guidelines:

- Provide adequate medication for pain relief, as ordered.
- Examine the wound frequently to assess proper healing, which should progress from the inside out. Healing should be complete in 4 to 5 weeks for perianal fistulas; in 12 to 16 weeks for deeper wounds.
- Inform the patient that complete recovery takes time, and offer encouragement.
- Stress the importance of perianal cleanliness.
- Be alert for the first postoperative bowel movement. The patient may suppress the

  urge to defecate because of anticipated pain; the resulting constipation increases pressure at the wound site. Such a patient benefits from a stool-softening laxative.

Rectal polyps

Rectal polyps are masses of tissue that rise above the mucosal membrane and protrude into the GI tract. Types of polyps include common polypoid adenomas, villous adenomas, hereditary polyposis, focal polypoid hyperplasia, and juvenile polyps (hamartomas). Most rectal polyps are benign; however, villous and hereditary polyps show a marked inclination to become malignant. Indeed, a striking feature of familial polyposis is that it's commonly associated with rectosigmoid adenocarcinoma.

Causes and incidence

Formation of polyps results from unrestrained cell growth in the upper epithelium. Predisposing factors include heredity, age, infection, and diet.

Villous adenomas are most prevalent in men older than age 55; common polypoid adenomas, in white women between ages 45 and 60. Incidence in both sexes rises after age 70. Juvenile polyps usually occur among children younger than age 10 and are characterized by rectal bleeding.

Complications

- Anemia from slow bleeding
- Bowel obstruction
- Colorectal cancer

Signs and symptoms

Because rectal polyps don't generally cause symptoms, they're usually discovered incidentally during a digital examination or rectosigmoidoscopy. Rectal bleeding is a common sign; high rectal polyps leave a streak of blood on the stool, whereas low rectal polyps bleed freely.

Rectal polyps vary in appearance. Common polypoid adenomas are small, multiple lesions that are redder than normal mucosa. They're commonly pedunculated (attached to rectal mucosa by a long, thin stalk) and granular, with a red, lobular, or eroded surface.

Villous adenomas are usually sessile (attached to the mucosa by a wide base) and vary in size from 0.5 to 12 cm. They are soft, friable, and finely lobulated. They may grow large and cause painful defecation; however, because adenomas are soft, they rarely cause bowel obstruction. Sometimes adenomas prolapse outside the anus, expelling parts of the adenoma with feces. These polyps may cause diarrhea, bloody stools, and subsequent fluid and electrolyte depletion, with hypotension and oliguria.

In hereditary polyposis, rectal polyps resemble benign adenomas but occur as hundreds of small (0.5 cm) lesions carpeting the entire mucosal surface. Associated signs include diarrhea, bloody stools, and secondary anemia. In patients with hereditary polyposis, changes in bowel habits with abdominal pain usually signal rectosigmoid cancer.

Juvenile polyps are large, inflammatory lesions, commonly without an epithelial covering. Mucus-filled cysts cover their usually smooth surface.

Focal polypoid hyperplasia produces small (less than 3 mm), granular, sessile lesions, similar to the colon in color, or gray or translucent. They usually occur at the rectosigmoid junction.

Diagnosis

CONFIRMING DIAGNOSIS

Firm diagnosis of rectal polyps requires identification of the polyps through proctosigmoidoscopy or colonoscopy and rectal biopsy.

Barium enema can help identify polyps that are located high in the colon. Supportive laboratory findings include occult blood in the stools, low hemoglobin level and hematocrit (with anemia) and, possibly, serum electrolyte imbalances in patients with villous adenomas.

Treatment

Treatment varies according to the type and size of the polyps and their location in the colon. Common polypoid adenomas less than 1 cm require polypectomy, usually by fulguration (destruction by high-frequency electricity) during endoscopy. For common

polypoid adenomas over 4 cm and all invasive villous adenomas, treatment usually consists of abdominoperineal resection or low anterior resection.

Focal polypoid hyperplasia can be obliterated by biopsy. Depending on GI involvement, hereditary polyps necessitate total abdominoperineal resection with a permanent ileostomy, subtotal colectomy with ileoproctostomy, or ileoanal anastomosis. Juvenile polyps are prone to autoamputation; if this doesn't occur, snare removal during colonoscopy is the treatment of choice.

Special considerations

During diagnostic evaluation:

- Check sodium, potassium, and chloride levels daily in the patient with fluid imbalances; adjust fluid and electrolytes, as necessary. Administer normal saline solution with potassium I.V., as ordered. Weigh the patient daily, and record the amount of diarrhea. Watch for signs of dehydration (decreased urine and increased blood urea nitrogen levels).
- Tell the patient to watch for and report evidence of rectal bleeding.

After biopsy and fulguration:

- Check for signs of perforation and hemorrhage, such as sudden hypotension, a decrease in hemoglobin level or hematocrit, shock, abdominal pain, and passage of red blood through the rectum.
- Have the patient walk as soon as possible after the procedure.
- Watch for and record the first bowel movement, which may not occur for 2 to 3 days.
- Provide sitz baths for 3 days.
- If the patient has benign polyps, stress the need for routine follow-up studies to check the polypoid growth rate.
- Prepare the patient with precancerous or familial lesions for abdominoperineal resection. Provide emotional support and preoperative instruction.

After ileostomy or subtotal colectomy with ileoproctostomy:

- Properly care for abdominal dressings, I.V. lines, and indwelling urinary catheters. Record intake and output, and check the patient's vital signs for hypotension and surgical complications. Administer pain medication, as ordered.
- To prevent embolism, have the patient walk as soon as possible, and apply antiembolism stockings; encourage range-of-motion exercises.
- Provide enterostomal therapy and teach the patient stoma care.

Pilonidal disease

In pilonidal disease, a coccygeal cyst forms in the intergluteal cleft on the posterior surface of the lower sacrum. It usually contains hair and becomes infected, producing an abscess, a draining sinus, or a fistula. Incidence is highest among hirsute white men ages 18 to 30.

Causes and incidence

Pilonidal disease may develop congenitally from a tendency to hirsutism, or it may be acquired from stretching or irritation of the sacrococcygeal area (intergluteal fold) from prolonged rough exercise (such as horseback riding), heat, excessive perspiration, or constrictive clothing.

The incidence rate of pilonidal disease is 0.7%. It affects men two to four times more often than women, but the onset is earlier in females, possibly because they begin puberty at an earlier age.

Complications

- Impaired social interaction due to pain and discomfort
- Difficulty performing work-related activities

Signs and symptoms

Generally, a pilonidal cyst produces no symptoms until it becomes infected, causing local pain, tenderness, swelling, or heat. Other clinical features include continuous or intermittent purulent drainage, followed by development of an abscess, chills, fever, headache, and malaise.

Diagnosis

CONFIRMING DIAGNOSIS

Physical examination confirms the diagnosis and may reveal a series of openings along the midline, with thin, brown, foul-smelling drainage or a protruding tuft of hair.

Pressure on the sinus tract may produce purulent drainage. Passing a probe back through the sinus tract toward the sacrum shouldn't reveal a

perforation between the anterior sinus and anal canal. Cultures of discharge from the infected sinus may show staphylococci or skin bacteria, but don't usually contain bowel bacteria.

Treatment

Conservative treatment of pilonidal disease consists of incision and drainage of abscesses, regular extraction of protruding hairs, and sitz baths (four to six times daily). However, persistent infections may necessitate surgical excision of the entire affected area.

After excision of a pilonidal abscess, the patient requires regular follow-up care to monitor wound healing. The surgeon may periodically palpate the wound during healing with a cotton-tipped applicator, curette excess granulation tissue, and extract loose hairs to promote wound healing from the inside out and to prevent dead cells from collecting in the wound. Complete healing may take several months.

Special considerations

Care includes preoperative and postoperative support and patient education.

- Before incision and drainage of a pilonidal abscess, assure the patient that he'll receive adequate pain relief.
- After surgery, check the compression dressing for signs of excessive bleeding, and change the dressing as directed.
- Encourage the patient to walk as soon as possible after the procedure.
- Tell the patient to place a gauze pad over the wound site after the dressing is removed to allow ventilation and prevent friction from clothing. Advise him to continue taking sitz baths and to let the area air-dry instead of rubbing or patting it dry with a towel.
- After healing, the patient should briskly wash the area daily with a washcloth to remove loose hairs. Encourage the obese patient to establish a weight loss plan.

Rectal prolapse

Rectal prolapse is the circumferential protrusion of one or more layers of the mucous membrane through the anus. Prolapse may be complete (with displacement of the anal sphincter or bowel herniation) or partial (mucosal layer). (See *Types of rectal prolapse*.)

Causes and incidence

Rectal prolapse usually occurs in children younger than age 6 and in adults in their 60s and 70s. It's commonly associated with other conditions, such as pinworms (enterobiasis), whipworm infection (trichuriasis), cystic fibrosis, malnutrition and malabsorption (such as celiac disease), constipation, and previous trauma to the anus or pelvic area.

True incidence figures are unavailable because many cases go unreported. Females are affected more often than males, accounting for 80% to 90% of reported cases.

Complications

- Rectal ulceration
- Bleeding
- Incontinence

Signs and symptoms

In rectal prolapse, protrusion of tissue from the rectum may occur during defecation or walking. Other symptoms include a persistent sensation of rectal fullness, bloody diarrhea, pain in the lower abdomen due to ulceration, a feeling of incomplete evacuation, and rectal incontinence. Hemorrhoids or rectal polyps may coexist with a prolapse.

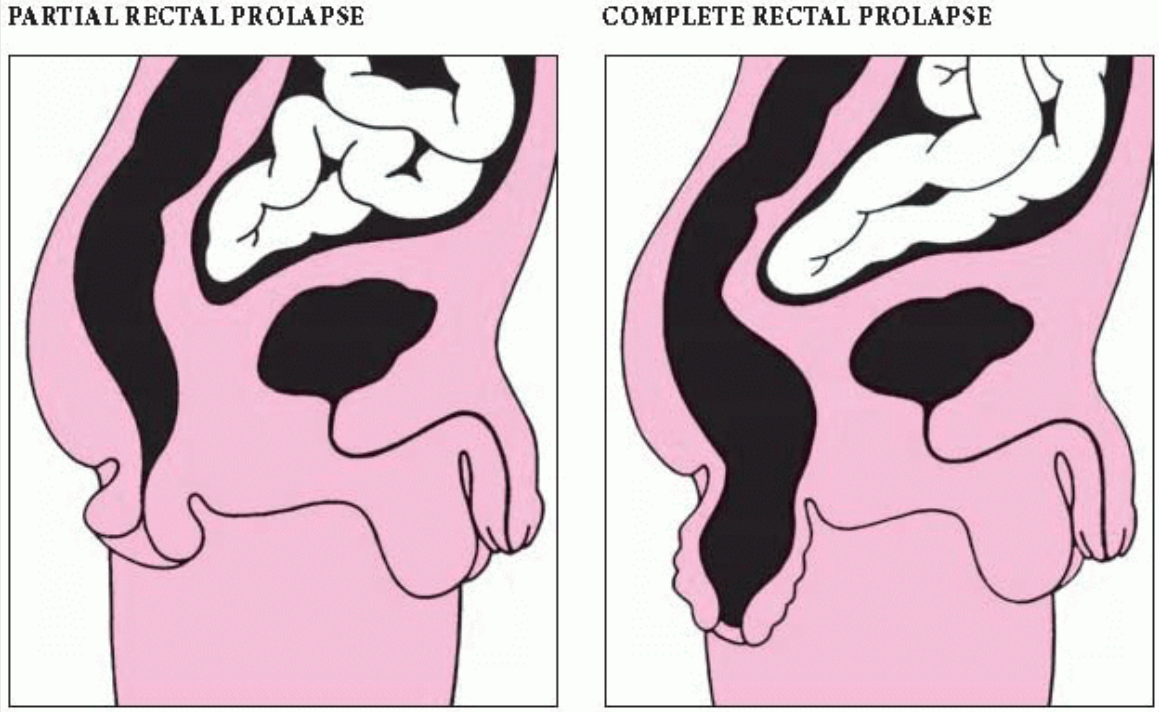
Diagnosis

Typical clinical features and visual examination confirm the diagnosis. In complete prolapse, examination reveals the full thickness of the bowel wall and, possibly, the sphincter muscle protruding and mucosa falling into bulky, concentric folds. In partial prolapse, examination reveals only partially protruding mucosa and a smaller mass of radial mucosal

folds. Straining during examination may disclose the full extent of prolapse.

TYPES OF RECTAL PROLAPSE

Partial rectal prolapse involves only the rectal mucosa and a small mass of radial mucosal folds. However, in complete rectal prolapse (also known as *procidentia*), the full rectal wall, sphincter muscle, and a large mass of concentric mucosal folds protrude. Ulceration is possible after complete prolapse.



Treatment

In some cases, eliminating the underlying cause is the only treatment necessary. The rectal mucosa can be returned to the rectum manually. While the patient is in a knee-chest position, a soft, warm, wet cloth may be used to apply gentle pressure to the mass to push it back through the anal opening, thereby allowing gravity to help return the prolapse into place. In a child, prolapsed tissue usually diminishes as the child grows. In an older patient, injection of a sclerosing agent to cause

a fibrotic reaction fixes the rectum in place. Severe or chronic prolapse requires surgical repair by strengthening or tightening the sphincters with wire or by anterior or rectal resection of prolapsed tissue.

Special considerations

Provide the patient with education regarding underlying causes and preoperative and postoperative support.

PREVENTION

Help the patient prevent constipation by teaching her the correct diet and stool-softening regimen.

- Advise the patient with severe prolapse and incontinence to wear a perineal pad.
- Before surgery, explain possible complications, including permanent rectal incontinence.
- After surgery, watch for immediate complications (hemorrhage) and later ones (pelvic abscess, fever, pus drainage, pain, rectal stenosis, constipation, or pain on defecation). Teach perineal strengthening exercises: Have the patient lie down, with her back flat on the mattress; then ask her to pull in her abdomen and squeeze while taking a deep breath; or have the patient repeatedly squeeze and relax her buttocks while sitting on a chair.

Anal fissure

Anal fissure is a laceration or crack in the lining of the anus that extends to the circular muscle. Most fissures heal on their own and don't require treatment, aside from good diaper hygiene. However, some fissures may require medical treatment. The prognosis is very good, especially with fissurectomy and good anal hygiene.

Causes and incidence

Anal fissure results from passage of large, hard stools that stretch the lining beyond its limits. It may also be due to prolonged diarrhea, strain

on the perineum during childbirth and, rarely, from scar stenosis. Occasionally, anal fissure is secondary to proctitis, anal tuberculosis, cancer, or Crohn's disease.

Anal fissures are common in young infants but may occur at any age, with incidence decreasing rapidly with age. Studies suggest that 80% of infants will have had an anal fissure by age 1. Fissures are less common among school-age children than infants. They affect males and females equally.

Complications

- Abscess, fistula, and septicemia (rare)
- Scar tissue
- Hampered bowel elimination

Signs and symptoms

Onset of an acute anal fissure is characterized by tearing, cutting, or burning pain during or immediately after a bowel movement. A few drops of blood may streak toilet paper or underclothes. Painful anal sphincter spasms result from ulceration of a “sentinel pile” (swelling at the lower end of the fissure). A fissure may heal spontaneously and completely or it may partially heal and break open again. Chronic fissure produces scar tissue that hampers normal bowel evacuation.

Diagnosis

Anoscopy showing a longitudinal tear and typical clinical features help establish the diagnosis. Digital examination that elicits pain and bleeding supports the diagnosis. Gentle traction on perianal skin can create sufficient eversion to visualize the fistula directly.

Treatment

Treatment varies according to the severity of the tear. Conservative treatment measures include stool softeners, dietary adjustment (addition of bulk to absorb water while in the intestinal tract), use of petroleum jelly and sitz baths, and cleaning more gently. Anesthetic

ointment may be useful if pain interferes with normal bowel movements. Topical muscle relaxants may also be soothing. These measures generally heal more than 90% of anal fissures. For fissures that don't heal with these treatments, injection of botulinum toxin into the anal sphincter will temporarily paralyze the anal sphincter muscle, thereby promoting healing. Another option for nonhealing fissures is a minor surgical procedure to relax the sphincter.

For superficial fissures without hemorrhoids, forcible digital dilatation of the anal sphincter under local anesthesia stretches the lower portion of the sphincter. For complicated fissures, treatment includes surgical excision of tissue, adjacent skin, and mucosal tags and division of hypertrophied internal sphincter muscle to release tension.

Special considerations

Care consists of patient education and support.

- Prepare the patient for rectal examination; explain the necessity for the procedure.
- Provide warm sitz baths, warm soaks, and local anesthetic ointment to relieve pain. A low-residue diet, adequate fluid intake, and stool softeners prevent straining during defecation.
- Give diphenoxylate or other antidiarrheals to control diarrhea.

Pruritus ani

Pruritus ani is perianal itching, irritation, or superficial burning. This disorder is more common in men and boys than in women and girls.

Causes and incidence

Factors that contribute to pruritus ani include overcleaning of the perianal area (harsh soap, vigorous rubbing with a washcloth or toilet paper); minor trauma caused by straining to defecate; poor hygiene; sensitivity to spicy foods, coffee, alcohol, food preservatives, perfumed or colored toilet paper, detergents, or certain fabrics; specific medications (antibiotics, antihypertensives, or antacids that cause

diarrhea); excessive sweating (in occupations associated with physical labor or high stress levels); anal skin tags; systemic disease, especially diabetes; skin disorders, such as psoriasis or eczema; certain skin lesions, such as those associated with squamous cell carcinoma, basal cell carcinoma, Bowen's disease, Paget's disease, melanoma, syphilis, and tuberculosis; fungal or parasitic infection; and local anorectal disease (fissure, hemorrhoids, and fistula).

Signs and symptoms

The key symptom of pruritus ani is perianal itching or burning after a bowel movement, during stress, or at night. In acute pruritus ani, scratching produces reddened skin, with weeping excoriations; in chronic pruritus ani, skin becomes thick and leathery, with excessive pigmentation.

Diagnosis

A detailed patient history is essential. Rectal examination rules out fissures and fistulas; biopsy rules out cancer. Allergy testing may also be helpful.

Treatment

After elimination of the underlying cause, treatment is symptomatic, such as advising the patient to avoid scratching or rubbing the itchy areas. Lukewarm baths and a skin-soothing oatmeal or cornstarch bath may be comforting. Temporary relief may be obtained with cold compresses. Topical over-the-counter ointments or creams containing hydrocortisone or zinc oxide are also useful.

Special considerations

- Make sure that the patient understands his condition and the causes.
- Recommend keeping fingernails short to avoid skin damage from inadvertent scratching. Suggest using cool, light, loose bedclothes and avoiding wearing rough clothing, particularly wool, over the irritated area.

- Advise the patient to avoid prolonged exposure to excessive heat and humidity.
- Advise the patient to avoid selfprescribed creams or powders, perfumed soaps, colored toilet paper, and moistened wipes because they may be irritating.
- Teach the patient to keep the perianal area clean and dry. Suggest witch hazel pads for wiping and cotton balls tucked between the buttocks to absorb moisture.

Proctitis

Proctitis is an acute or chronic inflammation of the rectal mucosa. It can result in discomfort, bleeding, and possibly a discharge of mucus or pus.

Causes and incidence

Proctitis caused by sexually transmitted disease (STD) occurs with high frequency among individuals who engage in anal intercourse. STDs that can cause proctitis include gonorrhea, herpes, chlamydia, and lymphogranuloma venereum. Amebiasis can also cause proctitis and can be transmitted by anal-oral sex.

In children, beta-hemolytic streptococcus may cause proctitis. Autoimmune proctitis is associated with such diseases as ulcerative colitis or Crohn's disease. Proctitis may also be caused by medications, radiation, or noxious agents such as chemicals inserted into the rectum.

Other contributing factors include chronic constipation, habitual laxative use, emotional upset, radiation (especially for cancer of the cervix or uterus), endocrine dysfunction, rectal injury, rectal medications, bacterial infections, allergies (especially to milk), vasomotor disturbance that interferes with normal muscle control, and food poisoning.

Signs and symptoms

Key symptoms include tenesmus, constipation, a feeling of rectal fullness, and abdominal

cramps on the left side. The patient feels an intense urge to defecate, which produces a small amount of stool that may contain blood and mucus.

Diagnosis

A detailed patient history is essential. In acute proctitis, sigmoidoscopy or proctoscopy reveal edematous, bright red or pink rectal mucosa that's thick, shiny, friable and, possibly, ulcerated. In chronic proctitis, sigmoidoscopy shows thickened mucosa, loss of vascular pattern, and stricture of the rectal lumen. Other supportive tests include biopsy to rule out cancer as well as rectal culture and examination of a stool sample.

Treatment

Primary treatment eliminates the underlying cause (fecal impaction, laxatives, or other medications). Proctitis caused by infection is treated with antibiotics specific for the causative organism. Corticosteroids or mesalamine suppositories may relieve symptoms in Crohn's disease or ulcerative colitis. Soothing enemas or steroid (hydrocortisone) suppositories or enemas may be helpful if proctitis is due to radiation. Tranquilizers may be appropriate for the patient with emotional stress.

Special considerations

- Tell the patient to watch for and report bleeding and other persistent symptoms.
- Fully explain proctitis and its treatment to the patient to help him understand the disorder and prevent its recurrence.
- Offer explanations, emotional support, and reassurance during rectal examinations and treatment.

Selected references

Baldi, F. “Key Points in the Management of Gastroesophageal Reflux Disease,” *Current Gastroenterology Reports* 9(1):1-2, February 2007.