ESOPHAGEAL OBSTRUCTION

Perspective

Patients with ingestion of foreign objects and esophageal food boluses commonly are seen in the emergency department (ED). Although most objects pass spontaneously, approximately 10 to 20% require a nonoperative intervention, and fewer than 1% require surgical removal. Death as a result of foreign body ingestion or impaction is rare. Patients with esophageal foreign bodies can be classified into four major categories: (1) pediatric patients, (2) psychiatric patients and prisoners, (3) patients with underlying esophageal disease, and (4) edentulous adults. Pediatric patients account for more than 75% of ingestions, with the peak incidence between the ages of 18 and 48 months.1 Coins account for the majority of pediatric ingestions, whereas most adult impactions involve pieces of food, particularly meat and bones.2 Patients with structural abnormalities of the esophagus, such as strictures, rings, webs, diverticuli, or malignancies, are at greater risk for foreign body impaction. Edentulous adults are also at increased risk because of impaired oral sensation and have a risk of accidental ingestion of their dental prosthesis.3

Principles of Disease

The adult esophagus is approximately 25 to 30 cm in length. Superi- orly, it begins in the hypopharynx as a transverse slit posterior to the larynx and approximately at the level of the cricoid cartilage. On either side of this cephalad slit are the piriform recesses, which are blind pouches that may occasionally harbor a foreign body. The esophagus is distensible; an adult can usually pass an object up to 20 mm in size without difficulty. Throughout its course, the esophagus has four natural areas of narrowing where the majority of foreign bodies become entrapped: at the cricopharyngeus muscle (the upper esophageal sphincter [UES]), the aortic arch, the left mainstem bronchus, and the lower esophageal sphincter (LES) at the diaphragmatic hiatus. Pediatric entrapment occurs primarily at the level of the cricopharyngeus, whereas adult entrapment occurs mainly at the LES. Large, proximal impactions can impinge on the trachea, leading to airway compromise that manifests as choking, stridor, or cough.

The esophagus comprises two main bands of muscle: an inner circular layer and an outer longitudinal layer. The resting tone of these muscles causes the inner epithelium to fold in on itself, effectively obliterating the lumen. Elastic fibers enable the esophageal lumen to expand and allow passage of a food bolus. The upper third of the esophagus, including the cricopharyngeus muscle, contains striated muscle to allow the voluntary initiation of swallow- ing. The middle portion of the esophagus is a mixture of skeletal and smooth muscle, and the distal third is composed only of smooth muscle.

Although it is relatively fixed at its origin, the esophagus becomes mobile as it traverses the mediastinum. Thus it can be easily displaced by adjacent structures such as an enlarged left atrium or ventricle, a goiter, or a mediastinal tumor. Displacement of the esophagus may alter its shape enough to impede the passage of a food bolus or foreign body.

Clinical Features

Patients with an esophageal obstruction have a wide range of symptoms. Most adults are able to describe the precipitating event and commonly complain of dysphagia (difficulty swallowing), odynophagia (painful swallowing), and neck or chest pain. Entrapment at the UES can generally be localized by the patient because of somatic nerve endings in the upper esophagus. In contrast, entrapment in the lower esophagus causes more visceral-type chest and epigastric discomfort.1 The obstruction may be partial or complete. The patient with complete obstruction is unable to swallow oral secretions and may be violently retching in an attempt to regurgitate the obstructing bolus. Patients should be evaluated for the presence of stridor or signs of perforation or peritonitis.

Pediatric patients are often brought to the ED after a witnessed ingestion. A high degree of suspicion is needed to diagnose foreign body ingestion when the event was unwitnessed, because 7 to 35% of children with proven esophageal foreign body impactions are asymptomatic at the time of presentation. Symptoms that should prompt consideration of unwitnessed foreign body ingestion include fever, wheezing, stridor, rhonchi, or poor feeding.8

A proximal obstruction may arise as a “café coronary,” characterized by sudden cyanosis and collapse caused by food (usually an unchewed piece of meat) lodging in the upper esophagus or oropharynx leading to airway obstruction. Similarly, “steakhouse syndrome” results when a large piece of food, usually improperly chewed, is swallowed and causes esophageal obstruction in the distal esophagus. The obstruction may be transient with spontaneous passage of the bolus and may be complete or partial. Intense discomfort develops shortly after swallowing a large piece of meat, and the patient is usually unable to swallow anything else. Ingestion of alcohol and absence of teeth are predisposing factors. Although obstruction may occur in a patient with a normal esophagus, preexisting structural abnormalities such as carcinoma, peptic stricture, or Schatzki’s ring are identified in almost 90% of patients with an esophageal obstruction. Schatzki’s ring is a fibrous, diaphragm-like stricture near the gastroesophageal junction present in up to 15% of the population.
Aside from naturally occurring areas of anatomic narrowing, there are other pathologic causes of esophageal stenosis that may lead to symptoms of obstruction. Intrinsic causes of luminal narrowing include carcinoma and webs. An esophageal web is a thin structure composed of mucosa and submucosa most commonly found in the middle or proximal esophagus. Although webs can occur in isolation, they are also seen in the Plummer-Vinson syndrome, which is characterized by anterior webs, dysphagia, iron deficiency anemia, cheilosis, spooning of the nails, glossitis, and thin friable mucosa in the mouth, pharynx, and upper esophagus. Most patients with this syndrome are women 30 to 50 years of age. Patients usually report dysphagia that is initially intermittent and worse with solids. If untreated, it may progress and become constant. Surgical changes after a gastric bypass can also predispose a patient to esophageal obstruction.

Extrinsic compression of the esophagus can occur in a variety of conditions. In the neck, thyroid enlargement from goiter or carcinoma may cause dysphagia. Symptoms may also be seen with a pharyngoesophageal or Zenker’s diverticulum, a progressive outpouching of the pharyngeal mucosa as a result of increased pressure generated by failure of proper relaxation of the cricopharyngeus muscle. Noisy deglutition, dysphagia, halitosis, and a palpable compressible mass in the neck may be present. Laryngotracheal aspiration when the patient is supine results from the emptying of contents from the diverticulum.

Diagnostic Strategies

Anteroposterior (AP) and lateral radiographs of the neck, chest, and/or abdomen can be obtained based on symptoms. Flat objects in the esophagus such as coins or button batteries orient in the coronal plane and appear as a circular object on an AP projection. Button batteries can be differentiated from coins by a characteristic radiographic “double-density” appearance. Small bones or radiopaque objects may occasionally be visualized. Air in the tissues may be present if perforation has occurred. However, failure to demonstrate a foreign body on radiographs does not rule out its presence. Contrast studies with barium or Gastrografin are rarely performed in this setting because they present a significant risk for aspiration and can obscure visualization if subsequent endoscopy is necessary. Computed tomography (CT) can be used in equivocal cases to identify and localize foreign bodies before endoscopy. CT is more sensitive than radiography at identifying foreign bodies including chicken or fish bones and other nonorganic objects. CT scans have the additional value of visualizing changes in the surrounding tissues associated with perforation.

Hand-held metal detectors have been reported to be useful screening devices for locating metallic foreign bodies in children without exposing them to radiation. They may also be of use in finding radiolucent metallic foreign bodies such as aluminum pull tabs. They do not, however, pinpoint the location of the object. Caution should be used in interpreting negative metal detector tests in obese children because esophageal coins have been missed in this scenario.

Persistent or concerning symptoms in a patient without evidence of a radiographic foreign body can be further evaluated by endoscopy.

Differential Considerations

Esophageal foreign bodies should be distinguished from foreign bodies in the airway. This distinction can be especially difficult in small children. Radiographically, esophageal foreign bodies usually lie in the frontal plain and are best visualized in AP views. Tracheal foreign bodies tend to lie sagittally.

Patients with esophageal obstruction may have retrosternal pain that can appear similar to that of an acute ischemic cardiac syndrome. The presence of odynophagia suggests an underlying mucosal lesion.

Management

Both flexible and rigid endoscopy are effective in removing esophageal foreign bodies. Flexible endoscopy is recommended in most cases as the first line in managing esophageal foreign bodies because it is better tolerated by patients and can usually be completed with use of procedural sedation. In contrast, rigid endoscopy requires general anesthesia, has a higher complication rate, and more commonly results in postinterventional dysphagia.

Upper Esophagus

Oropharyngeal foreign bodies can usually be removed with a Kelly clamp or Magill forceps under direct visualization. Smooth upper esophageal foreign bodies can often be removed with a Foley catheter. This procedure requires an experienced clinician, a cooperative patient, and fluoroscopic guidance. The patient is placed in a prone position, and the catheter is passed into the esophagus past the point of the foreign body impaction. The balloon is then inflated and the catheter withdrawn, pulling the foreign body with it. In a large study of children undergoing Foley balloon extraction with fluoroscopic guidance, 80% of foreign bodies were successfully removed, and an additional 8% were advanced into the stomach. Failure rates were highest with infants younger than 1 year of age. Controversy exists regarding the safety of this technique because there is no direct control of the foreign body. However, several large studies have shown complication rates to be less than 1% when patients are carefully chosen. Another technique is bougienage, which has been shown to be both safe and effective in coin removal. In this technique, an esophageal dilator is passed through the mouth into the esophagus to advance the coin into the stomach; the dilator is then quickly removed. In a large study, this procedure took less than 5 seconds to perform and was successful in 95% of cases with no serious complications. When these maneuvers fail to dislodge the esophageal foreign body, consultation with a qualified endoscopist is indicated.

Lower Esophagus

Lower esophageal obstruction is usually the result of an impacted food bolus. Anecdotally, administration of 1 mg of glucagon intravenously (IV) (up to a total of 2 mg) can cause enough relaxation of the esophageal smooth muscle to allow passage of a food bolus in the lower esophagus. However, no randomized controlled trials have shown a statistically significant benefit of using glucagon compared with placebo. In a small double-blind placebo-controlled study in children with esophageal coin impaction, glucagon was shown to be ineffective. In addition to lack of demonstrated efficacy, glucagon has multiple side effects such as vomiting, which can increase the risk of aspiration or esophageal perforation.

Effervescent agents are sometimes effective in accelerating the passage of an obstructing food bolus. Although the mechanism of action is unclear, it is hypothesized that the carbon dioxide released from bubbles escaping the fluid acts to disrupt the impacted food bolus and to distend the distal esophagus. There are case reports and case series in which administration of carbonated beverages...
(including soft drinks) has resulted in the passage of the obstructing food bolus in 60 to 80% of patients treated. However, there is only low-level evidence to support this practice, and the studies showing this benefit had multiple confounding factors. It has been recommended that effervescent agents be avoided in cases of complete obstruction and in cases in which an obstruction has been present for over 24 hours because of the theoretic potential of inducing perforation of a possibly ischemic distal esophagus. The use of meat tenderizer (papain) to soften a food bolus is not recommended. Although intact mucosa is resistant to papain's effects, an inflamed mucosa becomes much more inflamed when exposed to this proteolytic enzyme, and esophageal digestion or perforation may occur.

Endoscopy should be performed immediately for patients experiencing significant distress and for children with impaction of an alkaline button battery. Button batteries lodged in the esophagus can cause severe tissue damage in just 2 hours. Damage is primarily related to localized corrosive effects and occurs by three main mechanisms: leakage of an alkaline electrolyte, pressure necrosis, and generation of an external current that causes electrolysis of tissue fluids and generates hydroxide at the battery's negative pole. Larger batteries carry a greater risk of impaction and leakage. Delayed complications include esophageal perforation, tracheoesophageal fistula, esophageal abscess, and esophageal strictures. In a review of over 8000 battery ingestions that were reported to the National Battery Ingestion Hotline, outcomes have significantly worsened over the past decade. This is primarily attributable to newer 20 mm–diameter lithium cell batteries that now account for 92% of fatal ingestions. Batteries that pass into the stomach should be followed radiographically and clinically to ensure passage. Assistance with the management of a patient with button battery ingestion can be obtained through the National Battery Ingestion Hotline at 1-202-625-3333 or at www.poison.org/prevent/battery.asp.

Urgent intervention is also indicated for sharp objects, coins in the proximal esophagus, and impactions that impair the handling of secretions. It is unclear whether patients with mild to moderate symptoms of esophageal obstruction from a suspected food bolus require immediate endoscopy. In such cases, some experts believe that emergent intervention is unnecessary if the patient is still able to handle secretions because the bolus often passes on its own. Others believe that the softened bolus makes endoscopic removal more difficult and predisposes to complications such as ulcers, lacerations, erosions, and perforations. In a recent retrospective review, factors associated with a risk of complications included a longer duration of impaction, bone foreign bodies, and larger-size foreign bodies. Although it may be acceptable to delay endoscopy in stable patients without high-grade obstruction to allow possible spontaneous passage, a foreign body or food bolus impaction should not be allowed to remain in the esophagus for longer than 24 hours. Any object remaining in the esophagus for more than 24 hours carries a higher risk of complications, including perforation, aortoenteric fistula, tracheoesophageal fistula, or abscess. These complications may occur up to years after the ingestion. Many experts advocate follow-up endoscopic evaluation after an esophageal obstruction to rule out underlying pathologic conditions.

Stomach

Conservative outpatient management is appropriate for the vast majority of foreign bodies that have entered the stomach. However, certain foreign bodies that pass into the stomach still require endoscopic retrieval. Objects longer than 5 cm or wider than 2.5 cm in diameter (e.g., toothbrushes, spoons) rarely pass the duodenum. All sharp and pointed foreign bodies (e.g., toothpicks, bones) should be removed before they pass into the stomach because up to 35% may cause intestinal perforation. Smaller objects that pass into the stomach can be followed with stool inspections and with serial radiographs if necessary to confirm passage. Surgical removal should be considered for objects that remain in the stomach for more than 3 to 4 weeks or that remain in the same intestinal location for more than 1 week.

ESOPHAGEAL PERFORATION

Perspective

Esophageal perforation is a potentially life-threatening condition that is critical to identify and treat early to minimize morbidity and mortality. Boerhaave’s syndrome was first described in the early 1700s as a result of a rapid increase in intraesophageal pressure related to forceful vomiting. It can also result from any Valsalva-like maneuver, including childbirth, coughing, or heavy lifting. Iatrogenic esophageal perforation has become increasingly common in the past two decades, with endoscopy being the most common cause. Perforation has also been reported as a complication of both nasogastric tube placement and endotracheal intubation. Other causes of perforation include foreign body ingestion, caustic substance ingestion, severe esophagitis, carcinoma, and direct injury related to blunt or penetrating trauma.

Principles of Disease

Spontaneous rupture occurs because of a rapid increase in intraluminal esophageal pressure through a patent LES. The cricopharyngeal muscle fails to relax, leading to transmural rupture of the esophagus. More than 90% of spontaneous esophageal ruptures occur in the distal esophagus. In contrast, rupture resulting from blunt trauma to the neck or thorax usually occurs in the proximal and middle third of the esophagus. Most iatrogenic injuries occur at the pharyngoesophageal junction because the wall in this area is thin and there is no serosal layer to reinforce it, and force is frequently used to pass the tube beyond the level of the cricopharyngeus. Another site of frequent iatrogenic injury is the esophagogastric junction. In this area the esophagus curves anteriorly and to the left as it enters the abdomen, and an esophageal perforation has a greater likelihood of perforating the posterior wall. This usually occurs during therapeutic dilation for strictures or achalasia. Other factors predisposing to iatrogenic perforation include anterior cervical osteophytes, Zenker’s diverticulum, esophageal strictures, and malignancy.

Perforation has been reported as a complication of most endoscopic procedures, including transesophageal echocardiography, sclerotherapy, and inflation of the gastric balloon of a Sengstaken-Blakemore tube to control bleeding esophageal varices. When a perforation occurs, saliva and gastric contents can enter the mediastinum. Rapid spread of an infectious or inflammatory response to the surrounding tissues and organs occurs because of the thinness of the esophageal wall. Changes in intrathoracic pressure during respiration draw contaminants deeper into the mediastinum. The presence of gastric enzymes and other foreign material in the mediastinum induces an intense inflammatory response that may result in enough fluid buildup to displace adjacent structures.

Clinical Features

Clinical presentations vary and can depend on the cause, location, size, degree of contamination, and site of injury. Patients with an upper esophageal perforation usually have neck or chest pain, dysphagia, respiratory distress, and fever. Odynophagia, nausea, vomiting, hoarseness, or aphonia may also result. Mackler’s triad
of subcutaneous emphysema, chest pain, and vomiting is considered pathognomonic for spontaneous esophageal rupture. However, the complete triad is seen in less than half of cases. Patients with perforation of the lower esophagus may have abdominal pain, pneumothorax, hydro pneumothorax, and pneumomediastinum. The pain often radiates into the back, to the left side of the chest, and to the left or both shoulders. Early physical examination findings include epigastric or generalized abdominal tenderness, often with involuntary guarding and rigidity. Up to 30% of patients develop mediastinal or cervical emphysema, which may be noted by crepitus on palpation or by the pathognomonic Hamman’s sign with a “crunching” sound heard during auscultation. Patients with severe mediastinitis may be in full-nant shock.

Pain, fever, dyspnea, or crepitus after esophageal instrumentation should be considered an indication of perforation until proven otherwise. Symptoms related to iatrogenic perforation may not appear until several hours after the procedure.

**Diagnostic Strategies**

Radiographic studies are used to establish the diagnosis of an esophageal perforation. A chest and an upright abdominal radiograph are usually obtained first. Soft tissue lateral neck radiographs should be considered if a proximal perforation is suspected and may reveal air in the prevertebral fascial planes. Radiographic abnormalities may be detected in up to 90% of patients with esophageal perforation. Patients with upper esophageal injuries commonly have chest radiographs that show pneumomediastinum alone or a right-sided pleural effusion, whereas patients with distal esophageal perforations typically have a left-sided effusion. Other radiographic abnormalities include subcutaneous emphysema, mediastinal widening, or pulmonary infiltrates. These classic radiographic changes are often not present in the first few hours after perforation, so a normal radiograph should not be used early to exclude the possibility of esophageal perforation.

Patients with possible perforation should undergo contrast radiographic studies. Controversy exists regarding the contrast agent of choice. Barium sulfate is superior in identifying small perforations; however, it may incite an inflammatory response in tissues. For this reason, some experts advocate the use of water-soluble agents (e.g., Gastrografin). However, the water-soluble agents are less dense and may not demonstrate the abnormality. In addition, pneumonitis may result if these agents are aspirated because of their hypertonicity. A prudent approach would be to attempt the study with a water-soluble agent first in patients who are not at risk for aspiration. If a clinically suggested perforation is not identified, the examination should be repeated with barium.

CT of the chest may be considered if a contrast study does not demonstrate a clinically suggested perforation. It can also be used in patients who are intubated or cannot complete an esophagram. Findings such as mediastinal air, extraluminal contrast material, or fluid collections or abscesses adjacent to the esophagus confirm a perforation. CT also allows evaluation of other adjacent areas that may suggest an alternative diagnosis. Flexible esophageal endoscopy may be useful to directly visualize the perforation, especially in cases of penetrating external trauma, where this has a sensitivity of 100% and a specificity of 83%. This technique is not recommended for other situations because insufflation could potentially enlarge a minimal transmural opening. Laboratory studies are not usually helpful soon after a perforation, although leukocytosis may be noted.

**Differential Considerations**

Misdiagnosis occurs in more than half of patients with esophageal perforation or rupture because of the broad differential diagnosis of chest and abdominal pain. This includes pulmonary embolism, acute myocardial infarction, aortic dissection, perforated ulcer, pneumothorax, lung abscess, pericarditis, or pancreatitis. Esophageal perforations should be diagnosed as soon as possible because the morbidity and mortality associated with unrecognized perforations dramatically increase with time.

**Management**

Clinically unstable patients with esophageal perforation require rapid resuscitation and treatment. Broad-spectrum intravenous antibiotics should be initiated early. Patients should receive nothing by mouth (NPO), and a nasogastric tube should be considered to eliminate oral and gastric secretions. Early surgical consultation is warranted. A recent study compared survival of patients with esophageal perforation who were treated within 24 hours of perforation and those treated after 24 hours. This study found that aggressive treatment within the first 24 hours resulted in a 97% survival versus 89% survival in those treated after 24 hours.

There is growing evidence that some iatrogenic perforations in certain patients at low risk can be managed conservatively. These include clinically stable patients with minimal symptoms or fever, those whose perforation is contained, and those who are seen long after their procedure and have demonstrated no ill effects. In a recent retrospective study, patients who had a contained leak without respiratory compromise had worse outcomes when managed operatively compared with nonoperative management. Patients should be kept NPO and treated with broad-spectrum antibiotics and parenteral nutrition. These patients require diligent observation and assessment for failure of nonoperative therapy. In addition to true “nonoperative” management with close observation and intravenous antibiotics, other “palliative interventions,” including endoscopy, stent placement, drainage gastrostomy, feeding jejunostomy, and tube thoracostomy, have become more common.

**ESOPHAGITIS**

**Perspective**

*Esophagitis* is defined as inflammation of the esophagus. The most common cause of esophagitis is gastroesophageal reflux disease (GERD). Other important causes include infectious esophagitis, pill esophagitis, and injuries from the effects of caustic ingestion, radiation, or sclerotherapy. In addition, eosinophilic esophagitis is being diagnosed with increasing frequency.

**Principles of Disease**

**Infectious Esophagitis**

Esophageal infections primarily occur in immunocompromised hosts. When they occur in healthy patients, there is usually an underlying esophageal abnormality or local area of immune compromise, as might occur with the use of inhaled steroids. Iatrogenic alterations in host defenses through the use of immunosuppressive agents, potent chemotherapeutic agents, and broad-spectrum antibiotics can predispose an individual to the development of an esophageal infection. Human immunodeficiency virus (HIV) is a significant risk factor for infectious esophagitis, but rates have decreased since the advent of highly active antiretroviral therapy (HAART). Esophageal candidiasis is one of the most common acquired immunodeficiency syndrome (AIDS)-defining illnesses, but the incidence decreased by over 90% from 1994 to 2004. Patients with acute HIV seroconversion syndrome that occurs 2 to 3 weeks after primary
exposure to HIV can develop esophageal ulcerations and severe odynophagia.  

In addition to iatrogenic immunosuppression, diseases that weaken immunologic defenses in otherwise normal hosts can predispose the esophagus to infections. These conditions include diabetes mellitus, alcoholism, underlying malignancy, use of corticosteroids, and advanced age. Changes that occur in the mucosal barrier of the esophagus as a result of these conditions lead to an increased susceptibility to infection. The Candida species (primarily Candida albicans) are the most common esophageal pathogens. 

As empirical antifungal prophylaxis in immunosuppressive states has become more common, viral esophagitis has become more prominent. Herpes simplex virus 1 (HSV) and cytomegalovirus (CMV) are the most common viral pathogens. Human papillomavirus has been implicated as well. Bacteria, mycobacteria, other fungi, and parasitic organisms such as Trypanosoma cruzi, Cryptosporidium, and Pneumocystis are uncommon causes of infectious esophagitis and are usually diagnosed by culture or biopsy. 

Pill Esophagitis 

More than 1000 cases of pill-induced esophageal injury have been reported in the literature from nearly 100 different types of pills. The exact incidence of pill esophagitis is unknown because most cases are unrecognized and therefore unreported. The condition results when a pill or capsule fails to pass into the stomach and remains in contact with the esophageal mucosa for a prolonged period. This results in inflammation and injury of the esophageal mucosa. Pill esophagitis has been reported in all age groups. Pre-disposing factors include advanced age, decreased esophageal motility, and extrinsic compression. Large pills are more likely to be retained, as are those coated with gelatin. Pills can stick to a normal esophagus, especially when taken without water or by a patient in the supine position. Any area of the esophagus can be affected, although sites of natural compression may be more susceptible. Sustained-release compounds may be more damaging than standard preparations. Injury can range from minor irritation to frank ulceration, hemorrhage, and ultimately stricture formation. Some of the more common offending medications include antibiotics (especially the tetracycline family) and antivirals, aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs), potassium chloride, quinidine, ferrous sulfate, alendronate, and pamidronate.  

Eosinophilic Esophagitis 

Eosinophilic esophagitis was first described in 1978 and is defined by the presence of eosinophils within the esophageal mucosa or deeper tissues. Initially thought to be a disease of children, it is being diagnosed in adults with increasing frequency. Although diagnostic guidelines vary, recent reports have suggested use of the following criteria: clinical symptoms of esophageal dysfunction, more than 15 eosinophils in one high-power field on esophageal biopsy, and lack of responsiveness to high-dose proton pump inhibitors (PPIs) or normal pH monitoring of the distal esophagus. 

The cause is unknown, although there is an association with food allergens, especially in the younger age group. More than 50% of patients have associated atopic disorders, such as asthma or eczema. 

Caustic and Radiation-Induced Esophagitis 

Esophagitis from caustic substance ingestion occurs most commonly in children, although adults may intentionally ingest a large amount of a caustic substance in a suicide attempt. The most corrosive agents are strongly acidic with a pH less than 2 or alkaline with a pH greater than 12. The degree of injury depends on the concentration of the substance, the volume ingested, and the duration of mucosal contact. Strong acids produce coagulation necrosis, which results in eschar formation that usually limits the damage. In contrast, alkalis produce liquefaction necrosis, which continues to cause injury as long as the substance or its active breakdown products are in contact with tissue.  

Patients undergoing radiation treatment for underlying malignancy may develop esophagitis. The degree of injury is related to the total dose of radiation received. The mucosa becomes inflamed and friable. Agents used during sclerotherapy can also cause esophagitis. 

Clinical Features 

Esophagitis, regardless of cause, most commonly manifests with dysphagia or odynophagia. Chest pain is frequently present, and esophageal bleeding ranging from localized oozing as a result of inflammation to frank hemorrhage can occur. Ulceration and perforation can result in mediastinitis. 

Infectious Esophagitis 

Most commonly, infectious esophagitis causes severe odynophagia. Dysphagia of both solids and liquids may be present. Pain may be so severe that the patient refuses to eat or drink. Chest pain may also be present and may be described as acute in onset, constant, and not improved by antacid therapy. Heartburn and nausea may be presenting symptoms. Some immunocompromised patients may have fever or bleeding without dysphagia or odynophagia. 

Pill Esophagitis 

Patients with pill esophagitis have odynophagia. Most patients have no prior history of esophageal disease and experience sudden onset of pain worsened by swallowing. Dysphagia may be present. Although some patients complain that a pill has become “stuck,” the history of pill ingestion can be difficult to obtain because symptoms may begin hours after the offending pill was taken. Atypical presentations include a burning type of pain suggesting GERD as the cause. 

Eosinophilic Esophagitis 

Patients usually have dysphagia, nausea and vomiting, food impaction, or heartburn. This diagnosis should be considered in patients who have severe GERD symptoms despite the use of acid suppression medications and in patients with chronic unexplained dysphagia or recurrent esophageal food impaction. The diagnosis is confirmed by biopsy during endoscopy. 

Caustic and Radiation-Induced Esophagitis 

Patients with caustic injuries may have pain in the mouth, chest, or epigastrium. Dysphagia and vomiting can also occur. Drooling and airway compromise may be present because of direct tissue injury or resulting edema. Later, perforation may occur, and strictures are a common long-term complication. Radiation-induced esophagitis usually causes odynophagia and dysphagia. Strictures may ultimately develop. 

Diagnostic Strategies 

Endoscopy is the best method of diagnosing pill-induced, eosinophilic, and infectious esophagitis. With infectious esophagitis,
direct visualization may reveal characteristic signs of infection, such as white plaques of Candida or herpetic vesicles. Definitive diagnosis can be made through brushings and biopsies. Radiographic studies are usually not helpful because the findings are nonspecific. A strong clinical suspicion is necessary to diagnose pill esophagitis. The other causes of esophagitis are usually clinically apparent.

**Differential Considerations**

Other causes of esophageal pain include GERD, esophageal motility disorder, foreign body, and perforation. Chest pain may also be a component, and therefore an acute coronary syndrome should be considered. Esophageal pain is more likely to be positional and related to swallowing.

**Management**

**Infectious Esophagitis**

For infectious esophagitis, therapy should be directed at the causative organism. Patients with normal immune systems and mild cases of oropharyngeal candidiasis can be treated with clotrimazole troches (10 mg dissolved in the mouth five times a day for 1 week) or nystatin (400,000-600,000 million units orally [PO] four to five times per day for 2 weeks). Patients with true esophageal candidiasis should be treated with fluconazole (400 mg as a loading dose and then 100-400 mg daily for 14 to 21 days). In patients unable to tolerate taking oral medication, fluconazole can be given IV.34

Herpes esophagitis is generally a self-limited process that resolves over about 7 days. Immunocompromised patients should be treated with antivirals, such as acyclovir (400 mg PO five times per day for 7-14 days or 5-10 mg/kg IV every 8 hours for 7-14 days), famciclovir (500 mg PO three times a day for 7-14 days), or valacyclovir (1 g three times a day for 7-14 days).35 For CMV, initial treatment can begin with ganciclovir (5 mg/kg IV every 12 hours for 2-3 weeks) or foscarnet (60 mg/kg IV every 8 hours or 90 mg/kg IV every 12 hours for 2-3 weeks).

If the causative organism cannot be adequately identified or if the patient is severely debilitated, admission to the hospital may be required. Patients discharged from the ED should receive appropriate follow-up with the relevant specialist (e.g., gastroenterology, infectious disease). In addition to therapy directed at the infecting organism, treatment with antacids, topical anesthetics, or sucralfate may provide symptomatic relief.

**Pill Esophagitis**

If a patient with suspected pill esophagitis has persistent symptoms, endoscopy may be necessary. It also helps to determine alternative causes. No data exist supporting any specific treatment, although, intuitively, antacid medication may prevent further erosion of damaged mucosa. Symptoms may take up to 6 weeks to resolve.

The best treatment for pill esophagitis is prevention. Patients should be instructed to drink at least 4 ounces of liquid with any pill. All medications should be taken when the patient is in an upright position, and the patient should remain upright for several minutes after medication ingestion. Patients with underlying esophageal abnormalities or those who are bedridden should avoid the use of pills whenever practical.

**Eosinophilic Esophagitis**

These patients usually are seen after standard antireflux measures have failed or after they have developed a food impaction. The treating physician should consider the possibility of food impaction, ensure that appropriate antacid therapy is used, and refer the patient to a gastroenterologist for further treatment. Untreated eosinophilic esophagitis can lead to esophageal remodeling and stricture formation in up to 40% of adult patients. Although consensus has not yet been reached regarding an optimal treatment regimen, success has been reported with the use of topical (e.g., swallowed) corticosteroids. Recent pediatric studies have also shown efficacy with oral viscous budesonide.36

**Caustic and Radiation-Induced Esophagitis**

Management of caustic injuries includes evaluation and treatment of possible airway injury, followed by assessment of the extent of esophageal involvement. Although the use of mild diluents like water or milk to limit the extent of chemical injury has been advocated by some authorities, others warn against the possibility of inducing emesis, which reexposes the esophagus to the caustic substance. In general, it is probably best to avoid having patients ingest anything by mouth while undergoing evaluation. Likewise, gastric lavage and the administration of charcoal are not indicated. Symptomatic patients should be admitted to a monitored setting for observation, further evaluation with endoscopy, and treatment of potential complications, such as perforation. There is a high morbidity associated with caustic ingestions, with stricture formation in 26 to 55% of patients and possible later malignant transformation.37 As a result, multiple other treatments have been tried in an effort to improve long-term outcomes. This includes the use of intravenous corticosteroids and antibiotics, although the data on these have been mixed and they are not currently recommended.38 Asymptomatic patients who give a reliable history of a low-volume, accidental ingestion of a low concentration of an acidic or alkaline substance can be discharged after a period of observation and followed as outpatients.

Treatment of radiation esophagitis is supportive. Patients who cannot eat or drink because of radiation injury to the esophagus should be admitted for intravenous fluid therapy.

**GASTROESOPHAGEAL REFLUX DISEASE**

**Perspective**

Asymptomatic reflux of gastric contents from the stomach into the esophagus occurs in most people several times a day as a normal physiologic phenomenon. GERD occurs when reflux becomes symptomatic or causes histopathologic alterations in the upper gastrointestinal (GI) or respiratory tracts. In the United States, symptomatic reflux in the form of heartburn occurs daily in 7% of adults, weekly in 14%, and monthly in 40%.

**Principles of Disease**

The primary mechanism that enables reflux of gastric contents into the esophagus is inappropriate relaxation of the LES. This can occur because of general hypotension of the LES, increased intrabdominal pressure, or transient LES relaxations. Multiple risk factors can decrease LES pressure and lead to reflux, including medications (nitrates, calcium channel blockers, anticholinergics, albuterol), fatty meals, and chocolate. Other mechanisms that may contribute to GERD include esophageal motility abnormalities, increased intragastric pressure (e.g., obesity, pregnancy), acid hypersecretion, gastric outlet obstruction, and conditions that cause delayed gastric emptying (e.g., gastroparesis, neuromuscular disease).39
Clinical Features

Symptoms

The most common clinical manifestation of GERD is heartburn, defined as a burning sensation that begins in the subxiphoid area and radiates toward the neck. Reflux may also cause a dull discomfort, localized pressure, or severe squeezing pain across the middle of the chest. The patient may appear comfortable or may have associated diaphoresis, pallor, nausea, and vomiting, leading to the consideration of an ischemic cardiac syndrome. A detailed history is often helpful in differentiating cardiac chest pain from reflux, although the distinction may not be possible in the ED.

Other symptoms of GERD include regurgitation (the spontaneous appearance of acid or bitter material in the mouth or pharynx) and water brash (a vagally mediated hypersalivation response that may produce as much as 10 mL of saliva in 1 minute). Dysphagia and odynophagia may also be presenting complaints and may be associated with more serious complications.

Any condition or agent that decreases LES pressure, decreases esophageal motility, or prolongs gastric emptying predisposes patients to reflux (Box 89-1). Positions that place the esophagus in a position that is dependent to the stomach or increase intra-abdominal pressure tend to precipitate reflux. Stooping, bending, leaning forward, performing Valsalva-type maneuvers, and assuming a supine position are common precipitants.

GERD can manifest itself in extragastroesophageal locations. Reflux-induced asthma may result from either microaspiration of gastric contents into the lung or activation of a vagal reflex arc from the gut to the lung. GERD has been identified in up to 80% of asthmatic patients based on pH probe monitoring, but up to half of these patients have no reflux symptoms. However, studies have shown no benefit in treating poorly controlled asthmatic patients with proton pump inhibitors (PPIs) in the absence of GERD symptoms.

If the refluxate reaches the proximal esophagus, otolaryngologic manifestations may result, even in the absence of esophageal symptoms. Reflux can cause hoarseness, chronic laryngitis, refractory sore throat, and globus sensation. Refluxate that enters the oropharynx may lead to gingivitis, halitosis, or dental problems such as erosion of the lingual sides of the teeth as a result of acid exposure. Otalgia and hiccups can also result from reflux.

Complications

Repetitive exposure to acid can lead to changes in the esophageal mucosa. Continued reflux can lead to thinning of the normal stratified squamous epithelial layer. With the development of esophagitis, an inflammatory response occurs within the mucosa and submucosa with infiltration of polymorphonuclear leukocytes. The inflammatory response is the result of chemical irritation of the esophageal mucosa from reflux of gastric acid, pepsin, and bile acids. Both acid and alkaline refluxes produce the same pathologic changes. Continued exposure can lead to further endoscopically visible changes of erosion, ulceration, and scarring. Ultimately, stricture formation may result. The most severe histologic consequence of GERD is replacement of the normal stratified squamous epithelium with metaplastic columnar epithelium in a condition known as Barrett’s metaplasia. In patients with reflux undergoing endoscopy, approximately 10 to 15% are found to have Barrett’s esophagus. There is a strong correlation between the development of Barrett’s metaplasia and adenocarcinoma of the esophagus.

Differential Considerations

Acute cardiac ischemia should be considered as a possible cause of chest pain in adults. Radiation of pain can be a feature in both esophageal and cardiac chest pain. The pain seen with reflux may radiate into the neck, jaw, shoulders, back, arms, and abdomen. Radiation into the back is more often ascribed to the esophagus. Radiation of pain into one arm or into the neck or jaw is not helpful in distinguishing ischemic cardiac pain from esophageal pain. Radiation of pain into the abdomen is present approximately three times more often in reflux than in ischemic heart disease. Radiation into both arms is rarely seen in reflux, whereas it may be present in approximately one quarter of patients with ischemic heart disease. Precipitation of pain by exercise and relief by rest may occur in pain from reflux as well as in ischemic cardiac disease. Emotional precipitation of pain occurs in reflux, although it is also seen with coronary artery disease. The occurrence of reflux after meals is another important feature in the history. A feeling of fullness after meals occurs commonly in reflux and is helpful in differentiating it from coronary artery disease.

Relief of chest pain from reflux by antacids is a key point in the history; however, one should not place too much weight on this point as evidence against a cardiac cause. The relief is often short-lived, and pain may recur in a short time. Esophageal pain may be provoked by swallowing. The physical examination in patients with esophageal reflux is not usually helpful in diagnosis. Thus the history is by far the most valuable aid. It is important to maintain an acute awareness of the diverse presentations of ischemic heart disease and to be cautious in attributing chest pain to esophageal causes solely on the basis of historical elements. Other GI disorders such as gastritis, esophagitis, peptic ulcer disease (PUD), and biliary tract disease should be considered in the differential diagnosis.
Management

Earlier treatment guidelines for GERD recommended lifestyle modification solely as an initial approach; however, this has been shown to have little therapeutic benefit without concomitant medical management. Lifestyle modifications to reduce GERD symptoms include avoidance of foods that can precipitate reflux (caffeine, alcohol, chocolate, fatty foods) and avoidance of acidic foods that can cause heartburn (citrus, spicy foods). In addition to these dietary changes, other behavioral modifications include weight loss, smoking cessation, elevation of the head of the bed, and avoidance of a recumbent position for several hours after eating. The only lifestyle recommendations that have evidence-based support are weight loss and head-of-bed elevation; however, others can be useful adjuncts in selected patients. The pharmacologic therapy of GERD includes agents that neutralize acids, decrease acid production, act on the LES or affect motility, and protect the mucosa. The most effective treatment for GERD is reduction of acid production. Many patients initially self-medicate with antacids or over-the-counter-strength type 2 histamine receptor (H2)–receptor antagonists or PPIs, both of which have been demonstrated to relieve and prevent symptoms. A recent Cochrane review revealed that PPIs are more effective than H2 blockers in eliminating symptoms and healing mucosal damage.44 However, H2 blockers are often effective in patients with mild-to-moderate GERD. These agents do not stop the reflux but rather reduce the potency of the refluxate. Choices of H2 blockers and PPIs are listed in Tables 89-1 and 89-2. All of these agents are generally regarded as safe and effective. PPIs should be avoided in patients with acute coronary syndrome (ACS) who are taking clopidogrel, as multiple studies have demonstrated an increased risk of reinfarction and rehospitalization.45,46

Prokinetic agents treat GERD by increasing LES pressure. They may also be used for patients whose symptoms suggest a superimposed motility disturbance (e.g., regurgitation, choking, abdominal distention). In addition to improving propulsive activity of the stomach and small and large intestine, the increase in esophageal peristalsis and LES tone make an effective therapy for reflux by improving the clearance of refluxate. Cisapride (Propulsid) was formerly used for this purpose but was withdrawn from the marketplace by the manufacturer because of adverse cardiac effects. Metoclopramide, a dopamine antagonist, may be used for these patients, but its efficacy has not been conclusively demonstrated and it can cause significant irreversible extrapyramidal side effects such as tardive dyskinesia. Baclofen has been used in conjunction with PPI therapy with some success in selected patients.48 Candidates for this type of therapy are probably best chosen by a gastroenterologist.

Another agent that may be of benefit in refractory cases of symptomatic esophageal reflux is sucralfate, which is a mucosal protectant that binds to inflamed tissue to create a protective barrier. It blocks the diffusion of gastric acid and pepsin across esophageal mucosa and can limit the erosive action of pepsin and bile. It has limited side effects and can be safely used in pregnant women.49

Although the emergency physician can initiate antireflux therapy, the patient with clinically suggested GERD who does not improve with empiric therapy or those who are at high risk for complications should be referred to a gastroenterologist for confirmation of the diagnosis and follow-up care. In these cases, further diagnostic evaluation may be necessary. Patients who are intolerant of acid suppressive medications may be candidates for surgical therapy with laparoscopic fundoplication. Less invasive endoscopic therapies include thermal ablation to narrow the esophagus at the LES, suturing to create a plication at the LES, and injection or implantation techniques to bulk up the LES.50

GASTRITIS

Perspective

Strictly speaking, gastritis is a histologic diagnosis denoting inflammation of the gastric mucosa. Hence the diagnosis of gastritis can be made only by endoscopy and biopsy. However, it is common practice for clinicians to use the term gastritis to refer to symptoms of dyspepsia. To confuse the picture further, gastroenterologists frequently use the term to refer to the endoscopic finding of an edematous, friable mucosa. However, without accompanying inflammation, this is more appropriately termed gastropathy rather than gastritis. Controversy exists regarding how best to classify the entities that cause gastritis or gastropathy. This section considers gastritis and gastropathy together as one entity because the distinction makes little difference in the ED setting. Regardless of the cause, up to half of the population has endoscopic evidence of gastritis or gastropathy by age 50.

Principles of Disease

The most common cause of gastritis is infection with Helicobacter pylori. Although most patients are asymptomatic at the time of initial exposure, acute infection with H. pylori can cause severe gastritis and upper GI symptoms. Suppurative gastritis (also known as acute phlegmonous gastritis) can result from a bacterial infection of the stomach wall, usually from gram-positive cocci or gram-negative rods. Patients usually have an underlying mucosal

Table 89-1 Summary of Histamine Receptor Antagonists

<table>
<thead>
<tr>
<th></th>
<th>GERD</th>
<th>PUD*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cimetidine</td>
<td>800 mg bid or 400 mg qid</td>
<td>800 mg qhs or 400 mg bid</td>
</tr>
<tr>
<td>Famotidine</td>
<td>20 or 40 mg bid</td>
<td>40 mg or 20 mg bid</td>
</tr>
<tr>
<td>Nizatidine</td>
<td>150 mg bid</td>
<td>300 mg qhs or 150 mg bid</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>150 mg bid</td>
<td>300 mg qhs or 150 mg bid</td>
</tr>
</tbody>
</table>

GERD, gastroesophageal reflux disease; PUD, peptic ulcer disease.
*Maintenance dose for PUD is half the qhs dose.

Table 89-2 Summary of Proton Pump Inhibitors*

<table>
<thead>
<tr>
<th></th>
<th>GERD</th>
<th>PUD OR NSAID-INDUCED ULCERS*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esomeprazole</td>
<td>20 mg qd or 40 mg qd</td>
<td>40 mg PO qd</td>
</tr>
<tr>
<td>Lansoprazole</td>
<td>30 mg qd or 30 mg qd</td>
<td>30 mg PO qd</td>
</tr>
<tr>
<td>Omeprazole</td>
<td>20 mg qd or 20 bid</td>
<td>20 mg PO qd</td>
</tr>
<tr>
<td>Pantoprazole</td>
<td>40 mg qd or 40 bid</td>
<td>40 mg PO qd</td>
</tr>
<tr>
<td>Rabeprazole</td>
<td>20 mg qd or 20 bid</td>
<td>20 mg PO qd</td>
</tr>
</tbody>
</table>


GERD, gastroesophageal reflux disease; NSAID, nonsteroidal anti-inflammatory drug; PO, orally; PUD, peptic ulcer disease.
*All doses should be administered before breakfast; second doses (when necessary) should be administered before the evening meal.
†Patients with duodenal ulcer should be treated for 4 weeks; patients with gastric ulcer should be treated for 8 weeks.
abnormality such as cancer, ulcer, or preexisting gastritis. Less common infectious causes of gastritis include mycobacterial, viral, parasitic, and fungal organisms.

Gastritis can also result from exposure to drugs. Aspirin or other NSAIDs are the most common offending agents. Inflammation occurs as a result of prostaglandin inhibition both locally and systemically and is probably a precursor to gastric ulcer formation. Other drugs implicated in causing gastritis are potassium preparations and iron supplements. Gastritis can result from both short- and long-term exposure to ethanol, although some authors feel that the long-term effects are more likely a result of *H. pylori* than the ethanol itself.

The presence of corrosive agents in the stomach can induce gastritis. Intrinsic substances such as bile or ingested substances such as acids, alkali, and corrosive agents can induce an inflammatory response and subsequent gastritis.

Any condition that causes hypovolemia or hypotension can lead to gastritis. Ulcer formation may ultimately result. This may be a major causative factor in the development of gastritis and upper GI bleeding in intensive care unit patients. Other causes of gastritis include radiation, autoimmune reactions, Crohn’s disease, and sarcoidosis. These disorders can only be diagnosed by biopsy.

**Clinical Features**

No particular symptoms are characteristic of gastritis. Acute gastritis may cause abdominal pain, nausea, and vomiting, although most patients are asymptomatic unless ulcers or other complications develop. By definition, it is not possible to diagnose gastritis or gastropathy on the basis of clinical features alone. However, a good clinical history such as recent NSAID use or alcohol ingestion in the setting of the classic symptoms supports a presumptive clinical diagnosis of gastritis.

Acute infection with *H. pylori* may cause epigastric abdominal pain, nausea, and vomiting. Systemic signs such as fever are usually absent. Symptoms may last days to weeks. If the infection goes untreated, chronic gastritis may result. Patients with phlegmonous gastritis usually appear toxic. Patients with gastritis as a result of decreased mucosal blood flow may have symptoms of abdominal pain and upper GI bleeding in addition to those of their underlying disease. Complications of gastritis include perforation and gastric outlet obstruction.

**Diagnostic Strategies**

Because the presumptive diagnosis of gastritis is made empirically, no specific diagnostic tests are necessary. Ancillary tests should be ordered as clinically indicated to rule out other possible diagnoses or to assess for complications of gastritis, such as bleeding, obstruction, and perforation.

**Differential Considerations**

Before the diagnosis of gastritis is made, other diseases that cause nausea, vomiting, and upper abdominal pain, such as pancreatitis, biliary tract disease, and small bowel obstruction, should be excluded. The possibility of an acute coronary syndrome should also be considered, particularly in elderly patients.

**Management**

Therapy of presumptive gastritis should be directed toward treating the suggested underlying cause. Acid suppression may improve symptoms of dyspepsia in patients taking NSAIDs. Patients with persistent symptoms should be referred to a gastroenterologist for further diagnostic evaluation.

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**Box 89-2 Substances and Conditions That Damage the Gastric Mucosal Barrier**

- Bile
- Cigarette smoking
- Ethanol
- Glucocorticoids
- *Helicobacter pylori*
- Nonsteroidal anti-inflammatory drugs
- Pancreatic secretions
- Shock conditions
- Stress

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**PEPTIC ULCER DISEASE**

**Perspective**

Gastric and duodenal ulcers are usually grouped together as PUD because of the similarity in their pathogenesis and treatment. Approximately 500,000 people in the United States are diagnosed with PUD each year. The annual cost to the health care system is estimated to be over $10 billion. PUD is now considered to have two main causes: *H. pylori* infection and NSAID use. Approximately 1% of PUD is caused by increased levels of circulating gastrin from gastrin-secreting tumors (Zollinger-Ellison syndrome). These patients have increased parietal cell mass and hypersecretion of acid leading to ulcer formation. A small minority of patients have no identifiable cause for their ulcers.

**Principles of Disease**

Histologically, the stomach is composed of different types of cells with varying secretory functions. Mucous cells secrete acidic mucus, parietal cells secrete hydrochloric acid and intrinsic factor, chief cells secrete pepsinogens, and enterochromaffin-like cells release substances such as histamine and gastrin. There are over 1 billion parietal cells, which can generate a hydrogen ion concentration gradient of greater than 1 million to 1 within the lumen of the stomach.

Many mechanisms exist to protect the gastric mucosa from the digestive effects of the hydrochloric acid, proteolytic enzymes, bile, and other deleterious substances to which it is exposed. Normally a gastric mucosal barrier to intraluminal gastric acid is present and prevents the back-diffusion of hydrogen ions from the gastric lumen. Sodium ions are barred from moving in the opposite direction. This ionic impermeability protects the gastric mucosa from damage in a hostile environment. Damage to the gastric mucosal barrier from any cause (Box 89-2) allows hydrogen ions and digestive enzymes to make contact with the gastric mucosa, leading to inflammation, bleeding, and potential ulceration.

The identification of *H. pylori* has dramatically shifted our notion of PUD from an acid-related to an infectious disease-mediated process. *H. pylori* is a spiral, flagellated, gram-negative rod whose natural habitat is the human stomach between the epithelial cell surface and the overlying mucus. Infection with *H. pylori* is a primary risk factor for development of PUD. It is estimated that 70 to 80% of patients with duodenal ulcer and 60 to 70% of patients with gastric ulcer are infected with *H. pylori*. It is more prevalent in lower socioeconomic groups and in developing countries. It is probably spread by the fecal-oral route, although oral-to-oral and iatrogenic transmissions have also been suggested. It is estimated that 30 to 40% of the U.S. population is infected with *H. pylori*. It is found in people of all age groups, although infection is typically acquired during childhood. Its presence is believed to cause mucosal inflammation that disrupts the normal defense mechanisms and leads to ulceration. It also
increases the risk of gastric carcinoma and, less often, lymphoma. Although there is a strong association between *H. pylori* and PUD, only 5 to 10% of infected patients develop ulcers. It is unclear what role environmental and host factors (such as diet) play. It is now accepted that almost all non–NSAID-related ulcers are caused by *H. pylori*. Eradication of infection with *H. pylori* results in more rapid healing of ulcers, prevents relapse, and diminishes the rate of ulcer complications. It is also more cost-effective than chronic antisecretory therapy. Patients can be tested for *H. pylori* by both invasive and noninvasive methods. These include a urea breath test, serum antibody testing, stool antigen testing, and direct mucosal biopsy during endoscopy. None of these methods are currently practical to use in the ED. There are multiple treatment regimens that are more than 80% effective at eradication of *H. pylori*.

The second most common cause of peptic ulcer formation is the use of NSAIDs. Up to 25% of chronic NSAID users develop ulcer disease, and 2 to 4% of patients have serious complications including perforation or bleeding. The cause of NSAID-related ulcers was traditionally linked to “ion trapping” and reduction of mucosal hydrophobicity. However, it is now believed that the primary mechanism is related to suppression of gastric prostaglandin synthesis. Prostaglandins promote mucosal integrity by maintaining mucosal blood flow, promoting mucosal mucus and bicarbonate formation, and reducing mucosal acid secretion. It is believed that the inhibition of cyclooxygenase by NSAIDs leads to a diminished level of protective prostaglandins in the stomach. In addition, the antiplatelet aggregation effect of NSAIDs may increase the amount of bleeding associated with the development of NSAID-induced ulcers. NSAIDs differ in their ulcerogenic potential. Studies have shown a higher risk of upper GI bleeding with ketorolac and piroxicam. The cyclooxygenase-2 specific inhibitors (celecoxib, rofecoxib, and valdecoxib) were initially felt to have a better GI safety profile than traditional NSAIDs. However, further studies refuted this belief and noted an increased risk of cardiovascular side effects, including myocardial infarction and stroke. As a result, rofecoxib (Vioxx) and valdecoxib (Bextra) were withdrawn from the market. Celecoxib (Celebrex) is still available in the United States as a treatment for arthritis and familial polyposis but has a black box warning regarding both an increased incidence of GI side effects and an increased cardiovascular risk. Certain patients are at higher risk for NSAID-induced gastroduodenal toxicity. These include patients older than 60, those with a prior history of an ulcer or hemorrhage, those receiving higher doses of NSAIDS, and patients concurrently taking glucocorticoids or anticoagulants. These patients should be considered for ulcer prophylaxis with a PPI or misoprostol. Other drugs with ulcerogenic potential include 5-fluorouracil, mycophenolate mofetil, and the bisphosphonates.

PUD also occurs in infants and children. Infants with PUD usually demonstrate poor feeding, vomiting, or failure to thrive. Up to a quarter of children have isolated hematemesis or melena as presenting signs. Toddlers and preschool children may have abdominal pain, vomiting, and bleeding. Eighty percent of ulcer locations in this age group are stress ulcers associated with systemic illness such as sepsis, head trauma, burns, or sickle cell disease. Older children and adolescents usually have primary PUD, with presentations similar to those of adults.

### Clinical Features

#### Presenting Symptoms

The classic presenting symptom of PUD is epigastric pain that is described as “burning” or “gnawing.” However, up to 2% of patients with endoscopically proven PUD are asymptomatic. Patients may also have atypical symptoms, including pain in other areas of the abdomen, chest, or back, and may describe the pain as vague or crampy. Associated symptoms include fullness, nausea, early satiety, and bloating. Pain usually occurs 2 to 5 hours after a meal or at night. Symptoms that awaken a patient from sleep between midnight and 3 AM is a classic indicator of ulcer disease, because in most people gastric acid output is highest at about 2 AM. Ulcer pain is usually not present on awakening in the morning because gastric acid output is at its lowest at this time. Colicky pain is rarely gastric or duodenal in origin. Well-defined periods of exacerbation and remission are usually present with duodenal ulcer and aid in the diagnosis. A constant pain lasting from weeks to months is uncommonly caused by ulcer disease. Relief of pain after eating is another feature of gastric or duodenal ulcer. The pain from a duodenal ulcer is usually worse immediately before a meal, and the complex of pain—eating—relief is typical of duodenal ulcer.

Although some patients with ulcers may vomit, alternative diagnoses such as gastric volvulus, gastric outlet obstruction, small-bowel obstruction, pancreatitis, or biliary tract disease should be considered in patients with epigastric pain and vomiting. Relief of abdominal pain with antacids is an important aspect of the history. Antacids usually afford relief of pain in both PUD and gastritis. Ninety percent of patients with PUD have pain relief with antacids, and 75% with gastritis have relief. Patients with duodenal ulcer usually experience pain relief within 5 minutes after taking an antacid.

Physical findings in patients with PUD are usually minimal. Mild epigastric tenderness may be elicited. A positive stool guaiac test may be evidence of a bleeding ulcer, but other causes of occult bleeding should also be considered.

### Complications

The most serious complications of PUD include hemorrhage, perforation, penetration, and gastric outlet obstruction. Hemorrhage is the most common complication, occurring in 15 to 20% of patients. Ulceration into an artery can lead to life-threatening hemorrhage. Patients older than age 60 are at greater risk. Approximately 2 to 10% of patients experience perforation, which occurs when an ulcer erodes through the wall and leaks air and digestive contents into the peritoneal cavity. Perforation most commonly involves the anterior wall of the duodenum. Penetration is pathologically similar to perforation, except that the ulcer erodes into another organ such as the liver (usually from a gastric ulcer) or the pancreas (usually from a duodenal ulcer) instead of into the peritoneal cavity. Gastric outlet obstruction occurs in 2% of ulcer patients as a result of edema and scarring near the gastroduodenal junction. Symptoms may manifest as gastrsophageal reflux, early satiety, weight loss, abdominal pain, and vomiting.

Pain patterns may be helpful in diagnosing some of the complications of PUD. Pain from a perforated duodenal ulcer is usually appreciated first in the epigastrium but becomes generalized within a short time. Vomiting is present in 50% of patients, and peritoneal findings usually result. Pneumoperitoneum commonly occurs after duodenal ulcer perforation, and the accumulated air under the diaphragm may cause referred pain to the shoulder. One or both shoulders may be involved, depending on the location of the free air.

A history of ulcer-like anterior abdominal pain that begins to radiate into the back suggests penetration of a duodenal ulcer. The pain is usually described as steady and is perceived at the level of the lower thoracic and upper lumbar vertebrae. The pain becomes refractory to treatment with antacids and food. Also, the pain may radiate to the chest, right upper quadrant, and left upper quadrant in up to 20% of patients. The sudden onset of pain, especially if unrelated to eating, suggests either ulcer perforation or gastric volvulus.
Diagnostic Strategies

The initial diagnosis of PUD is usually made clinically. Upper endoscopy is the procedure of choice for confirming the diagnosis. Ancillary tests may be of benefit in evaluating possible complications of PUD in patients in distress. They may also be of benefit in providing indirect evidence of another disease. A complete blood count may diagnose anemia, and liver enzyme values may help elucidate a hepatic or biliary tree cause. Electrolytes may provide indirect evidence of disease, and a lipase level should be considered to rule out pancreatitis and may provide indirect evidence of a posterior penetrating ulcer.

Abdominal and chest radiographs should be ordered if obstruction, perforation, or penetration is suggested or if a pulmonary cause is being considered, although negative radiographs do not definitively rule out these diagnoses. Electrocardiography should be performed in any patient thought to have a cardiac cause for the pain. A pregnancy test should be performed on any woman of childbearing age.

As noted earlier, several methods exist for diagnosing infection with H. pylori, although at this time none is of practical application in the ED.

Differential Considerations

Fifty percent of patients with symptoms of dyspepsia have no identifiable cause. These patients are classified as having functional or nonulcer dyspepsia (NUD). The most recent definition by the Rome III consensus is the presence of epigastric pain or burning, postprandial fullness, and early satiety in the absence of underlying organic disease.50 NUD may be caused by peptic ulcers that are not yet large enough to appear endoscopically. Gastritis related to hypersecretion of gastric acid, H. pylori infection, bile reflux, or viral infection may cause NUD, although these cases should be identifiable endoscopically or pathologically. Maldigestion or malabsorption of carbohydrates can arise as NUD in patients with lactase deficiency or in patients who consume large quantities of nonabsorbable sugars, such as sorbitol, mannitol, and fructose. Intestinal parasites such as Giardia intestinalis or Strongyloides stercoralis may cause NUD, as can chronic pancreatitis. NUD may also be caused by gastric motility disorders, which have been reported in 25 to 60% of patients with NUD. Abnormalities in the biliary tract, such as increased resting pressure of the sphincter of Oddi, or incomplete relaxation of the sphincter on gallbladder contraction, may lead to bile duct distention and pain.

Many other disorders can produce epigastric pain that mimics the pain of an ulcer. It can be difficult to distinguish between gastritis and PUD. The discomfort associated with gastritis is often mild to moderate in severity and described as a hot, burning pain or bloating. In particular, burning pain is twice as common in gastritis as in PUD. Esophageal disorders such as GERD, esophagitis, or esophageal spasm can arise with abdominal symptoms. Mesenteric ischemia (“abdominal angina”) should be considered, especially in elderly patients and those with underlying vascular disease or atrial fibrillation. Aortic dissection, other intrathoracic processes such as biliary tract and pancreatic disease, and atypical presentation of an acute cardiac syndrome or other intrathoracic process should be considered in the differential diagnosis. Finally, abdominal pain may be the presenting symptom in psychiatric patients with somatoform disorders. These patients have an altered perception of visceral pain and have an increased sensation of pain when the stomach or small intestine is dilated.

MANAGEMENT

It is now well recognized that PUD is a result of either infection with H. pylori or NSAID use, so initial treatment should target the presumed underlying cause. For NSAID-related ulcers, treatment should begin with discontinuation of the offending agent and initiation of a PPI. A Cochrane review recently examined methods of preventing NSAID-associated gastroduodenal ulcers. There is evidence that misoprostol, PPIs, and double doses of H₂ blockers reduce the risk of both endoscopic gastric and duodenal NSAID-induced ulcers.54 If NSAIDs are not being used by a patient with suggested PUD, it is currently recommended to treat for H. pylori infection. Dyspeptic symptoms without proven ulcer may also be an indication for treatment, but that decision may best be left to a gastroenterologist. Antacid therapy may be started with a PPI or H₂ blocker. Nonendoscopic testing for H. pylori is available in the form of antibody detection, urea breath test, and fecal antigen tests; however, their role in the evaluation of ED patients is not yet defined.

Some recommended regimens combine antibiotics with acid-suppressing agents for treatment of H. pylori infection (Box 89-3). Commercially available combination products may also be prescribed that may assist in compliance (Prevpac, which contains bismuth subsalicylate, metronidazole, and tetracycline). Most gastroenterologists recommend continued therapy with antisecretory agents after the antibiotic-containing regimens.

GASTRIC VOLVULUS

Perspective

Gastric volvulus is a rare cause of severe abdominal pain that occurs when the stomach rotates on itself more than 180 degrees, creating a closed-loop obstruction. Only 400 cases have been reported in the literature, although its true incidence is unknown because some types of volvulus are intermittent and resolve spontaneously. It most commonly occurs in persons 40 to 50 years of age and is usually associated with the presence of a paraesophageal hernia. Approximately 20% of cases occur in infants younger than 1 year, often associated with a congenital diaphragmatic defect. If an acute volvulus is not identified and corrected early, it may lead to gastric ischemia, perforation, and death. The mortality rate from acute gastric volvulus has been reported to be 30 to 50%.51
Principles of Disease

The stomach is fixed at only two points: the esophagocardiac junction and the pylorus. The remainder of the organ is relatively distensible and mobile and can occupy various positions within the abdomen. When a person is supine, the stomach lies entirely above the umbilicus, whereas it descends below the umbilicus in the erect position. Regardless of its position, the stomach maintains its familiar morphology because of ligamentous attachments to the surrounding organs. A primary (or subdiaphragmatic) volvulus occurs when the stabilizing ligaments are too lax or are congenitally abnormal in such a way that the stomach is able to twist on itself. Approximately one third of cases are of this type.

Secondary (or supradiaphragmatic) volvulus occurs in patients with diaphragmatic defects such as a paraesophageal hiatal hernia, an elevated diaphragm, gastric ulcer or carcinoma, diaphragmatic paralysis, extrinsic pressure on the stomach from other organs, or abdominal adhesions. The combination of one of these factors and ligamentous laxity makes a volvulus more likely.

Gastric volvulus can be classified on the basis of its axis of rotation. The most common form is organoaxial volvulus, which occurs when the stomach twists on its long axis. Less commonly, the stomach folds on its short axis from the lesser to greater curvature and is classified as a mesenteroaxial volvulus. Approximately one third of cases of gastric volvulus are of this type.

Clinical Features

Presenting Symptoms

The presenting features of a gastric volvulus vary depending on the type. Primary volvulus may arise with the sudden onset of severe abdominal pain. The upper abdomen may demonstrate marked distention. Patients with secondary volvulus may experience predominant symptoms in the chest, with pain radiating to the back and shoulders along with accompanying dyspnea. The abdominal examination may be unremarkable. Nonbilious vomiting is usually present and may be persistent and severe. The combination of severe epigastric pain and distention, vomiting followed by violent nonproductive retching, and inability to pass a nasogastric tube (Borchardt’s triad) increases the likelihood of a gastric volvulus. Up to a quarter of children with acute gastric volvulus are presented with life-threatening events that necessitate resuscitation, including apnea, cyanosis, and acute respiratory distress. A volvulus may be chronic if the rotation is minimal and there is no vascular compromise. Symptoms usually consist of mild intermittent upper abdominal pain. Early satiety, dyspnea, bloating, eructation, and upper abdominal fullness may be present. It is unknown how often a chronic volvulus can lead to an acute volvulus.

Complications

If not recognized, volvulus can lead to bowel ischemia and necrosis of the stomach. Untreated, this may lead to shock and death. Fortunately, the frequency of gastric infarction is low (reportedly 5 to 28% for organoaxial volvulus) because of the redundant blood supply of the stomach. Other complications include ulceration, perforation, hemorrhage, pancreatic necrosis, and omental avulsion.

Diagnostic Strategies

A plain abdominal radiograph often demonstrates a large, gas-filled loop of bowel in the abdomen or chest. A barium swallow may help visualize the abnormality, and CT can be used for confirmation in equivocal cases. There are no laboratory findings specific for volvulus, although elevations in amylase and alkaline phosphatase have been reported.

Differential Considerations

The differential diagnosis of gastric volvulus includes any disease that can arise with sudden upper abdominal pain and vomiting. Perforated peptic ulcer, gastric outlet obstruction, biliary tract disease, and acute pancreatitis should be considered. Symptoms of a volvulus may suggest an acute cardiac syndrome.

Management

The goal of treatment of an acute gastric volvulus is reduction. Mortality rates increase with delayed diagnosis because of complications of ischemia. Acutely, one should attempt passage of a nasogastric tube, which may occasionally reduce the volvulus. Although somewhat controversial, patients without signs of gastric infarction may undergo an attempt at endoscopic reduction. Ultimately, treatment is a surgical emergency, with the goal of reducing the volvulus and preventing recurrence by fixing the stomach within the abdomen. Surgical repair of predisposing diaphragmatic defects is also recommended to prevent recurrence.

DYSPHAGIA

Perspective

Precise motor control of the act of swallowing is necessary to ensure that food is successfully transferred from the mouth through the esophagus into the stomach. This includes the muscles of the oropharynx, the UES, the body of the esophagus, and the LES. Failure at any one of these levels results in a motility disorder, the primary symptom of which is dysphagia, which literally means “difficulty swallowing.”

Principles of Disease

Normal Physiology

Swallowing is a complex phenomenon requiring both voluntary and involuntary skeletal muscle activity. Control of swallowing is coordinated by the swallowing center in the medulla. Afferent sensory input involves the trigeminal, glossopharyngeal, vagus, and spinal accessory cranial nerves; efferent motor activity travels through the trigeminal, facial, glossopharyngeal, vagus, and hypoglossal cranial nerves. The act of swallowing begins a process of both simultaneous and sequential activity in all three esophageal zones. A rapidly progressive pharyngeal contraction transfers the food bolus through a relaxed UES into the esophagus, where a moving ringlike contraction begins in the upper esophagus and propagates distally, making the transition from striated to smooth muscle, culminating with the propulsion of the bolus through a relaxed LES. Three mechanisms have been described that regulate the peristaltic wave, ensuring a smooth transition from the striated muscle in the upper esophagus to the smooth muscle of the middle and lower esophagus and coordination of UES and LES activity. These mechanisms are sequential firing of vagal afferents that begin in the brainstem, an intramural neural mechanism that responds to local stimuli, and myogenic propagation of the contraction through the myocytes themselves.

Physiologically, swallowing can be divided into oral, pharyngeal, and esophageal phases. The oral phase involves preparation of the food bolus by mastication and lubrication. The tongue then propels the bolus into the pharynx by progressive AP contractions. In the pharyngeal phase of swallowing, events are initiated by...
delivery of the food bolus to the oropharynx. Voluntary contraction of the pharyngeal muscles seals the nasopharynx by elevation of the soft palate. The oropharynx is sealed by the upward movement of the tongue against the palate. The larynx and hyoid bone are elevated to seal off the respiratory passage. The cricopharyngeus muscle, or UES, relaxes, and the bolus is swept into the esophagus by sequential peristaltic waves initiated in the upper pharynx. During the esophageal phase, the bolus is propelled toward the stomach by sequential peristaltic waves. Peristalsis can be initiated by swallowing or in response to luminal distention of the gut or changes in the pH or osmotic environment of the mucosa. The lower sphincter normally maintains a degree of tone sufficient to prevent reflux of gastric contents. When the food bolus reaches the lower sphincter, the sphincter relaxes to allow passage of the bolus and then regains its degree of resting tone.

Pathophysiology

Disturbances of the interactions between the components of the upper GI tract lead to a motility disorder. The motor disorders of the body of the esophagus are only now beginning to be understood. The major primary esophageal motility disorders are achalasia, diffuse esophageal spasm, hypercontractile esophagus (“nutcracker esophagus”), and nonspecific motor disorder. Of these, the only two that are well defined are achalasia and diffuse esophageal spasm. Controversy exists about whether the other entities are true disease states because symptoms are not always associated with manometric abnormalities and correction of the abnormalities does not always result in symptom improvement. Motor disorders may be the primary cause of other esophageal abnormalities such as GERD or esophageal diverticula.

Clinical Features

Dysphagia at any age is abnormal and requires evaluation. Dysphagia can be classified into one of two types. The first is oropharyngeal dysphagia (also known as transfer dysphagia), which involves difficulty transferring a food bolus from the oropharynx to the proximal esophagus. The second type, esophageal dysphagia, involves difficulty in transporting material down the esophagus. Although dysphagia has many causes, a thorough history reveals the diagnosis in most patients (Box 89-4). It is crucial to determine what types of food cause the symptoms (liquids, solids, or both) and whether the symptoms are intermittent or progressive. One should determine where the bolus sticks, whether it is associated with pain, and whether the patient has any previous gastroesophageal history (e.g., esophageal reflux). Any family history of neurologic disease should be obtained.

The examination should include a thorough evaluation of the head and neck and a detailed neurologic examination. The patient should be observed while swallowing. Difficulty in initiating the swallow, misdirection of the bolus with regurgitation or aspiration, and unusual posturing of the patient when swallowing should be noted. Many patients with neuromuscular disorders depend on gravity to swallow, and having the patient swallow in the prone position may be helpful in diagnosis.

Oropharyngeal Dysphagia

Oropharyngeal causes of dysphagia inhibit the initiation of swallowing. Patients complain that “food gets stuck” on swallowing, often pointing to the cervical region when describing their symptoms. Coughing, choking, or drooling may be associated. Neuromuscular diseases cause approximately 80% of oropharyngeal dysphagias, with most remaining causes being localized structural lesions. Most neuromuscular causes of dysphagia result in misdirection of the bolus, sticking, and the need for repeated swallowing attempts. Patients may drool and turn the head and neck to the side to facilitate swallowing. Liquids, especially of extreme temperatures, usually cause dysphagia more commonly than solids, and symptoms are more often intermittent. Progressive unremitting dysphagia is usually not caused by neuromuscular disorders of the oropharynx. Cerebrovascular accidents are probably the most common cause of neuromuscular dysphagia, especially those involving the vertebrobasilar system and posteroi

<table>
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<td>Cerebrovascular accident</td>
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<td>Magnesium deficiency</td>
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<td>Familial dysautonomia</td>
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<td>Muscular dystrophies</td>
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<td><strong>Obstructive</strong></td>
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<td>Aortic aneurysm</td>
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<td>Esophageal motility disorder (e.g., achalasia, diffuse esophageal spasm, hypertensive LES, nutcracker esophagus)</td>
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<td>Esophageal rings</td>
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<td>Esophageal stricture</td>
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<td>Esophageal webs</td>
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<td>Hypertrophic cervical spurs</td>
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<td>Neoplasm</td>
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<td>Thyroid enlargement</td>
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<td>Vascular anomalies (e.g., enlarged aorta, aberrant subclavian artery)</td>
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<td>Zenker’s diverticulum</td>
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<td>Alcoholism</td>
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<td>Decreased saliva production (Sjögren’s syndrome, postirradiation)</td>
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<td>Diabetes</td>
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<td>Gastroesophageal reflux disease</td>
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LES, lower esophageal sphincter.
Disorders. Dysphagia is a presenting symptom in 21% of patients with these degenerative changes in striated muscle that can produce dysphagia from weakness of the palate, pharynx, and upper esophagus. Dysphagia is a presenting symptom in 21% of patients with these disorders.63

A cause of oropharyngeal dysphagia that deserves particular mention is myasthenia gravis. At least 40% of patients with myasthenia gravis have dysphagia, and it is the presenting symptom in up to 15% of patients. The dysphagia becomes progressively worse with repeated swallowing attempts and is temporarily reversible with edrophonium.64

Disorders in the pharyngeal phase of swallowing may lead to misdirection of the food bolus, pain, sticking, or multiple swallowing attempts. Tongue weakness can result in oral regurgitation. Inability to seal the nasopharynx because of obstruction or muscular weakness can cause nasal regurgitation. Inefficient laryngeal elevation from muscular weakness or a fixed larynx can result in laryngotracheal aspiration. Delayed aspiration can occur with pharyngeal weakness and with pooling of food in the piriform recesses or in a diverticulum. Inability to contract the pharyngeal muscles is often composed by failure of the criocopharyngeus to relax. Failure of relaxation of the criocopharyngeus with or without pharyngeal weakness causes misdirection of the food bolus or necessitates repeated swallowing attempts. Inflammatory lesions of the tongue or oropharynx can result in odynophagia and even complete inability to swallow because of pain.

Congenital anomalies of the aortic arch may cause dysphagia in both children and adults. In children, respiratory symptoms are usually present and commonly predominate. In adults, an anomalous right subclavian artery is the most common vascular cause for dysphagia, often referred to as dysphagia lusoria. Patients often do not become symptomatic until the fourth decade of life. The most common symptoms in adults are dyspnea on exertion and dysphagia.65 Vascular compression of the esophagus with dysphagia may also occur with aneurysms of the aortic arch and great vessels. Bronchogenic carcinoma can cause dysphagia by direct involvement of the esophagus or by compression with nodes.

Esophageal Dysphagia

Dysphagia from upper esophageal lesions is usually perceived 2 to 4 seconds after the initiation of swallowing. Dysphagia that the patient localizes to the substernal or retrosternal area may be anatomically accurate, but localization to the neck may be referred from anywhere in the esophagus.

Esophageal dysphagia can be caused by mechanical lesions or a motility disorder. Mechanical lesions include strictures, webs, rings, tumors, esophagitis, or postsurgical changes. Pressure from extrinsic lesions such as osteophytes, mediastinal masses, or aortic aneurysms can also cause dysphagia.

Patients with esophageal dysphagia who have no readily identifiable cause may have a motor disorder. The motor disorders include achalasia, diffuse esophageal spasm, nutcracker esophagus, and hypertensive LES. Achalasia is a disorder of unknown cause in which the resting pressure of the LES is markedly increased and peristalsis in the body of the esophagus is absent. The incidence increases with age, with the highest incidence in the seventh decade of life and a smaller peak incidence in patients aged 20 to 40 years.66 Dysphagia is the most common presenting symptom and usually begins insidiously with equal frequency for solids and liquids. Patients may report that maneuvers that increase esophageal pressure (raising arms above the head, standing erect with back straight) help pass the food. Odynophagia from esophageal spasm may also be seen early in the course of achalasia. The symptoms are often worse with rapid eating and during periods of stress. The patient may also report chest pain as a symptom. As dilation occurs above the sphincter, retention of undigested food in the esophagus occurs, and the patient may be aware of gurgling while eating. Regurgitation of the undigested material can occur after a meal (prompting consideration of the diagnosis of an eating disorder) or with changes in position or vigorous exercise. The regurgitated food usually has no acid taste, although bacterial contamination may lead to fermentation of the undigested food. Laryngotracheal aspiration may occur, especially at night, and may cause nocturnal coughing. Physical examination is usually unremarkable except for weight loss. Radiographically, a dilated esophagus is seen proximal to a narrowed gastroesophageal junction that has a beaklike appearance.67

The second type of intrinsic motor disorder of the esophagus is diffuse esophageal spasm. Manometrically, simultaneous prolonged strong esophageal contractions are noted to be interspersed over normal peristaltic waves. If a barium swallow is performed during a spasm, findings such as a “corkscrewing,” or curling, of the esophagus may be noted. Diffuse spasm may be precipitated by swallowing very hot or cold liquids. Symptoms include chest pain, dysphagia, or both.68

Nutcracker esophagus is the term used to describe prolonged, high-intensity peristaltic waves. Many experts feel that this represents a variant of diffuse esophageal spasm. Nonspecific motor disorders include repetitive esophageal contractions, nontransmitted esophageal contractions, or low-amplitude esophageal contractions.69

Diagnostic Strategies

Given the myriad causes of dysphagia, a careful history and physical examination are essential. Patients with oropharyngeal dysphagia should undergo laboratory studies and central nervous system imaging as indicated. Nasopharyngoscopy may also be performed to rule out obvious structural abnormalities. If these are nondiagnostic, patients may be referred for a swallowing study (videoesophagram). Patients with esophageal dysphagia in whom carcinoma, radiation or caustic injury, or achalasia is suggested should undergo a barium swallow. If a motor disorder is suggested, a swallowing study may prove helpful as well, but this may not detect intermittent dysfunction. In such cases, referral to a gastroenterologist for manometric examination may be required. At that time, additional provocative studies can be performed.

Differential Considerations

The differential diagnosis of lower esophageal dysphagia includes acute coronary syndromes. Substernal chest pain is the main symptom in 80 to 90% of patients with esophageal motility disorders. The chest pain can be similar to angina, described as crushing or squeezing with patterns of radiation similar to those of cardiac chest pain. Nitroglycerin may relieve the pain of spasm as well, further confusing the picture.

Symptoms that suggest an esophageal cause of chest pain are pain that is prolonged and nonexertional, pain that interrupts sleep, pain related to meals, relief with antacids, and presence of other symptoms of esophageal disease such as heartburn, dysphagia, or regurgitation. Because of considerable overlap in symptoms, the emergency physician must exclude a cardiac diagnosis before attributing chest pain to an esophageal cause.
Management

Appropriate management of dysphagia is based on the identified or suggested cause. Most patients with no readily identifiable cause can be evaluated as outpatients; however, it is prudent to admit patients who are at high risk for aspiration. Patients in whom an esophageal motility disorder is suggested should be referred to a gastroenterologist because the diagnosis is usually made manometrically. Achalasia is the only motility disorder for which reasonably good studies support specific treatment. Surgical management is the mainstay of treatment for the majority of patients with severe achalasia. Pharmacologic therapy is directed at decreasing the tone of the LES. Nitrates and calcium channel blockers have been used with some success; however, adverse effects limit their usefulness. Other therapies used with some degree of success have included botulinum toxin injection, pneumatic dilation, and surgical intervention.70

Medical therapy for esophageal motility disorders is rather limited, and clinical results are usually minimal. Anticholinergic drugs such as hyoscyamine sulfate or dicyclomine have been used because they decrease the amplitude of esophageal peristalsis and LES pressure. However, these drugs may also exacerbate reflux symptoms because they cause delayed gastric emptying and decreased esophageal peristalsis. Other therapies include calcium channel blockers, which decrease both LES pressure and the amplitude of esophageal contractions. Psychotropic medications such as trazodone have been used to treat some esophageal motility disorders. Although no study has demonstrated specific beneficial manometric effects, it is believed that these medications may reduce the discomfort experienced and the patient’s perception of the pain.71

PHARMACOLOGIC AGENTS FOR UPPER GASTROINTESTINAL DISORDERS

Antacids

By the time most patients seek treatment for upper GI complaints, they have already tried some form of antacid therapy because these agents are readily available as over-the-counter preparations. Antacids afford pain relief in most patients with PUD. Doses with low neutralizing capacity (as low as 30 mEq) promote ulcer healing. Antacids may also work by binding bile acids or inhibiting pepsin.

The choice of antacid should be individualized. The magnesium-containing antacids can produce diarrhea in up to 25% of patients. Magnesium-containing antacids can also lead to an increase in serum magnesium levels and should be avoided or used with caution in patients with impaired renal function. Aluminum-containing antacids may cause constipation, and prolonged use may lead to phosphate depletion. Calcium-containing antacids have been marketed both as acid neutralizers and as a means of calcium supplementation, especially for postmenopausal women. Calcium-containing antacids have been traditionally believed to cause the highest incidence of acid rebound, a paradoxical increase in gastrin secretion and acid production. Calcium antacids can also lead to constipation, and their excess consumption can lead to hypercalcemia, alkalosis, and renal insufficiency (the milk-alkali syndrome).

Antacids can also decrease the absorption of warfarin, digoxin, some anticonvulsants, and some antibiotics. It is recommended that antacids be administered 1 to 3 hours after meals and at bedtime. Antacids are the least expensive drugs available to treat PUD, but their use is somewhat limited by side effects and inconvenient administration schedules.

Histamine Blockers

Histamine is the primary stimulus to gastric acid secretion. It binds to the H2 receptor located on the basolateral portion of the parietal cell to stimulate the release of hydrochloric acid. The discovery of the ability of H2 blockers to inhibit gastric acid production was a major advance in antulcer therapy because ulcers cannot develop in the absence of acid. These drugs are highly selective competitive inhibitors of histamine for the H2 receptor on parietal cells and reduce both the volume of gastric juice and its hydrogen ion concentration. All of the currently available H2 blockers are rapidly absorbed after an oral dose, with peak levels reached within 1 to 2 hours. All have half-lives of approximately 2 to 3 hours, so the effects last for about 6 hours. Most are now available over the counter in lower dosage strength. H2 blockers are effective in treating duodenal ulcer and, to a lesser extent, gastric ulcer, although they are not as effective as the PPIs. They are widely prescribed for symptoms of dyspepsia and work well in patients with episodic heartburn. All H2 blockers are mainly heptatically and renally metabolized with the exception of nizatidine, which is almost exclusively renally metabolized. Dosages of all these agents should be reduced in patients with renal failure.

H2 blockers are safe and generally well tolerated. Side effects are rare, including central nervous system effects such as somnolence, dizziness, and confusion. Transient increases in liver enzyme levels may be noted. Some patients may exhibit abnormalities in cardiac conduction, as there are H2 receptors in the heart. Cimetidine has been shown to cause gynecomastia. Dosages of the various agents are summarized in Table 89-1.

Proton Pump Inhibitors

The H+, K+-ATPase (proton pump) is located on the apical portion of the parietal cell and is responsible for the production of hydrogen ions in gastric acid. PPIs are the most potent inhibitors of gastric acid secretion. They work by irreversibly binding to stimulated proton pumps to block secretion of hydrogen ions. Although they have no effect on the volume of gastric juice produced, production of acid can be reduced by up to 95%. Both basal and stimulated gastric acid secretions are reduced. The antisercretory effects last up to 72 hours. PPIs should be administered before the first meal of the day, as the number of proton pumps is maximized after a fasting state. At the cellular level, additional proton pumps are continually recruited to produce more acid in response to stimulation; therefore several doses of a PPI are necessary for maximal antacid effect to be achieved. The use of these medications on an as-needed basis would not be expected to provide a good clinical response. H2 blockers are more suitable for this purpose.73

PPIs are heptically metabolized, and dosage should be modified in patients with hepatic failure. Side effects are usually minimal, and the long-term safety of these drugs has been shown in multiple studies.72 PPIs may be used at significantly higher dosages in patients with Zollinger-Ellison syndrome. Dosages of the various agents are summarized in Table 89-2. Lansoprazole, pantoprazole, and esomeprazole are available as intravenous formulations.

Prostaglandins

Prostaglandins exert protective effects on the gastric mucosa by inhibiting acid secretion and decreasing the amount of cyclic adenosine monophosphate generated in response to histamine. Inhibition of gastric acid secretion, increased secretion of mucus
and bicarbonate, and stimulation of mucosal blood flow have all been demonstrated. Misoprostol is an analogue of prostaglandin E₁ with a longer duration of action and greater potency than endogenous prostaglandins. It should be used only for prevention of NSAID-induced gastric ulcers in patients at high risk. The dose is 200 µg four times a day with food, but crampy abdominal pain and diarrhea may necessitate the use of a somewhat less effective dose of 100 µg four times a day. Misoprostol is an abortifacient and therefore is contraindicated in any female patient of child-bearing age who is not using contraception.

**Other Agents**

Sucralfate binds to epithelial cells and especially to ulcerated surfaces, providing a protective layer that inhibits further acid damage. Its mechanism of action is not completely understood, although it has been shown to enhance epithelial growth, suppress acid secretion, and inhibit growth of *H. pylori*. The usual dose is 1 g four times a day given 30 to 60 minutes before meals.

Bismuth compounds such as bismuth subsalicylate decrease pepsin activity, increase mucus secretion, and form a barrier to further acid damage in ulcer craters. They also increase prostaglandin synthesis and retard hydrogen ion diffusion through the mucosal barrier. Bismuth may also help heal ulcers through its bactericidal action on *H. pylori*. Bismuth compounds are not approved for the treatment of peptic ulcers.

**KEY CONCEPTS**

- Radiographic contrast studies of patients with suggested perforation of the esophagus or stomach should first be performed with water-soluble agents such as Gastrografin.
- There is a growing role for nonsurgical management of esophageal perforation for selected low-risk patients.
- GERD treatment includes lifestyle modification and therapy with an antisecretory agent, usually a PPI.
- Peptic ulcer disease results primarily from NSAID use or infection with *H. pylori*.
- Proton pump inhibitors are the most effective means of suppressing gastric acid secretion.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.