Gastric and Esophageal Emergencies

Alessandro Mangili, MD

KEYWORDS
- Gastric
- Esophageal
- Emergency
- Dysphagia
- GERD

ESOPHAGEAL ANATOMY AND PHYSIOLOGY

The esophagus is a hollow muscular tube extending from the pharynx to the stomach. Its primary role is to carry solids and liquids from the mouth to the stomach. In the adult the esophagus measures 23 to 25 cm and runs in the mediastinum posterior and lateral to the trachea. Along its course 3 areas of luminal narrowing exist: (1) proximally at the level of the cricoid cartilage; (2) at the level of the aortic arch and left main stem bronchus; and (3) distally at the gastroesophageal junction, where the esophagus penetrates the diaphragm.

The walls of the esophagus are composed of different layers. From internal to external they include a mucosa, submucosa, muscularis propria, and adventitia. The mucosa is composed of nonkeratinizing, stratified squamous epithelial cells. Melanocytes, endocrine cells, and Langerhans cells are present in small numbers in the deeper epithelial layer. The proximal 5% of the esophagus contains striated muscle alone. The remaining 30% to 40% of the upper esophagus contains both smooth and striated muscle. The remaining distal portion of the esophagus is composed of smooth muscle only.

The esophagus has 2 high-pressure areas known as sphincters that help regulate flow. The upper esophageal sphincter (UES) consists of a 3-cm segment composed primarily of the cricopharyngeus muscle. It functions primarily to prevent aspiration and swallowing of large amounts of air. The lower esophageal sphincter (LES) is a 2-cm to 4-cm segment located just proximal to the gastroesophageal junction at the level of the diaphragm, and its primary function is to prevent retrograde flow of gastric contents into the esophagus (reflux).

Swallowing occurs in 3 stages: oral, pharyngeal, and esophageal. In the oral stage food is chewed, formed into a bolus, and moved to the posterior pharynx by the tongue. During the pharyngeal phase the bolus is moved across the UES from the pharynx into the proximal esophagus. In the final stage the food moves from the proximal esophagus into the stomach across the relaxed LES via peristalsis.
DYSPHAGIA

Dysphagia is the sensation of impaired passage of food from the mouth to the stomach that occurs after swallowing. In contrast, globus refers to the constant sensation of a lump or fullness in the throat regardless of swallowing. In individuals older than 50 years the prevalence of dysphagia is estimated to be 16% to 22%. Dysphagia occurs more commonly in older adults and in patients who have had cerebrovascular accidents, head injuries, esophageal cancers, and neuromuscular disorders. In addition, 60% to 87% of residents of nursing homes experience some form of feeding difficulty, and of those residents with oropharyngeal dysphagia and aspiration, 12-month mortality is estimated at 45%.

Dysphagia is generally divided into 2 types based on the stage of swallowing during which it occurs: oropharyngeal, or transfer, dysphagia and esophageal dysphagia. Oropharyngeal dysphagia occurs during the oral and pharyngeal phases of swallowing when food is transferred from the mouth to the upper esophagus, hence symptoms generally occur within 1 second of swallowing. Esophageal dysphagia occurs during passage of a bolus from the esophagus to the stomach. Dysphagia is further characterized by the mechanism that causes it. Functional causes refer to disordered motor function, whereas mechanical causes include obstruction or narrowing of the esophageal lumen.

Oropharyngeal Dysphagia

Causes of oropharyngeal dysphagia include inflammatory, rheumatologic, neuromuscular, and infectious disorders. Symptoms of oropharyngeal dysphagia may include drooling, difficulty in initiating swallowing, need to swallow repetitively to clear food from the mouth and pharynx, coughing, gagging, nasal or oral regurgitation, dysarthria, dysphonia, and aspiration. Chronic oropharyngeal dysphagia may also lead to weight loss, failure to thrive, malnutrition, and recurrent pneumonia.

Esophageal Dysphagia

Esophageal dysphagia results from an obstructive process secondary to either a structural lesion, neuromuscular disorder, or inflammatory process. In contrast to oropharyngeal dysphagia, symptoms typically occur 10 to 15 seconds after swallowing, and patients describe a sensation of fullness or pressure located in the substernal or epigastric region. Studies indicate that in approximately 75% of cases patients are able to localize the site of the obstruction based on the location of their symptoms. Symptoms associated with esophageal dysphagia may include chest pain, late regurgitation, and odynophagia. Clinical history can be helpful in differentiating between neuromuscular and mechanical causes of esophageal dysphagia. In general, dysphagia that is present equally with solids and liquids suggests a neuromuscular disorder, whereas dysphagia that is present only with solids, or that has progressed from solids to liquids, is more consistent with a mechanical cause. Onset, progression, relief of symptoms, and sensitivity to food temperature also help to distinguish neuromuscular from mechanical causes.

Neuromuscular Causes of Dysphagia

Achalasia

Achalasia is a primary motility disorder that affects men and women equally, most commonly between the ages of 25 and 60 years. The estimated prevalence in the United States is 10 cases per 100,000. The disease results from the degeneration of neurons in the myenteric plexus enervating the esophagus secondary to an
inflammatory process. This loss of enervation leads to impaired LES relaxation, decreased esophageal peristalsis, and esophageal dilatation. In addition, there is decreased synthesis of important mediators affecting LES relaxation, nitric oxide, and vasoactive intestinal polypeptide.\(^\text{10}\)

The most common presenting symptom of patients with achalasia is a progressive dysphagia to both solids and liquids. Sixty percent of patients also report regurgitation, which typically occurs after meals and can occur nocturnally and lead to cough and aspiration, resulting in significant bronchopulmonary complications in 10% of patients.\(^\text{11}\) Chest pain is also reported in 20% to 60% of patients. Other symptoms include difficulty belching and heartburn. Weight loss is a rare finding and is generally associated with end-stage disease.

Diagnosis is typically delayed because symptoms are often attributed to gastro-esophageal reflux disease (GERD) or other disorders. Secondary causes of achalasia include Chagas disease secondary to infection with *Trypanosoma cruzi*, esophageal or gastric cancer, paraneoplastic syndrome from small-cell lung cancer, eosinophilic esophagitis (EE), and postfundoplication achalasia.

Numerous imaging modalities exist to aid in the evaluation of dysphagia. The barium esophagram is typically the initial study performed. Esophagram findings consistent with achalasia are a tapering of the distal esophagus with contrast in the distal esophageal lumen, classically described as a bird’s beak, as well as dilation of the distal esophagus (Fig. 1). Esophageal manometry has the highest sensitivity for diagnosing

### Table 1

<table>
<thead>
<tr>
<th>Structural Lesions</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Pharyngeal diverticula</td>
<td>Zenker diverticulum, lateral pharyngeal pouch or diverticula</td>
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<tr>
<td>Intrinsic lesions</td>
<td>Oropharyngeal or laryngeal carcinoma, surgical resection, cricopharyngeal achalasia, cricopharyngeal bar and rings, proximal esophageal webs (Plummer-Vinson), radiation injury</td>
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<tr>
<td>Extrinsic compression</td>
<td>Osteophytes, skeletal abnormalities, thyromegaly</td>
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<tr>
<th>Neuromuscular Diseases</th>
<th>Examples</th>
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<tr>
<td>Central nervous system</td>
<td>Cerebrovascular accidents, head injury, neoplasm, Parkinson disease, multiple sclerosis, amyotrophic lateral sclerosis, Huntington chorea</td>
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<tr>
<td>Peripheral nervous system</td>
<td>Poliomyelitis, amyotrophic lateral sclerosis, tabes dorsalis, glossitis, pharyngitis, thrush (sensory)</td>
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<td>Neuromuscular transmission myopathies</td>
<td>Myasthenia gravis, polymyositis, dermatomyositis, muscular dystrophies, alcoholic myopathy, thyrotoxicosis, hypothyroidism, Amyloidosis, Cushing syndrome</td>
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achalasia; however, patients presenting with dysphagia typically first undergo upper endoscopy, which may reveal retained food and secretions. However, up to 44% of patients have a normal upper endoscopy. Upper endoscopy is especially useful in evaluating for other mechanical causes of dysphagia. Diagnostic findings of esophageal manometry include aperistalsis of the distal esophagus and decreased or absent LES relaxation. Vigorous achalasia is a manometric variant of achalasia in which aperistalsis of the distal esophagus is replaced by normal or high-amplitude esophageal body contractions. High-resolution esophageal manometry (HRM) combined with

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<th>Common causes of esophageal dysphagia</th>
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<td><strong>Structural Lesions</strong></td>
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<tr>
<td>Intrinsic lesions</td>
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<tr>
<td>Peptic stricture, Schatzki ring</td>
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<tr>
<td>Esophageal carcinoma</td>
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<tr>
<td>Leiomyoma, lymphoma</td>
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<tr>
<td>Hiatal hernia</td>
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<td>Extrinsic compression</td>
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<td>Mediastinal tumors (lung cancer, lymphoma)</td>
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<td>Vascular structures (dysphagia lusoria)</td>
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<td>Surgical changes (fundoplication)</td>
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<td><strong>Motor Disorders</strong></td>
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<td>Primary motor disorders</td>
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<tr>
<td>Achalasia</td>
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<tr>
<td>DES</td>
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<tr>
<td>Hypertensive LES</td>
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<tr>
<td>Nutcracker esophagus</td>
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<tr>
<td>Ineffective esophageal motility</td>
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<tr>
<td>Secondary motor disorders</td>
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<tr>
<td>Collagen vascular diseases or scleroderma, CREST</td>
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<td>Diabetes mellitus</td>
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<td>Alcoholism</td>
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<td><strong>Mucosal Diseases</strong></td>
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<td>Esophagitis</td>
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<tr>
<td>GI reflux diseases</td>
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<tr>
<td>Infectious esophagitis</td>
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<td>Pill induced</td>
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<td>Radiation injury</td>
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<td>Caustic ingestion</td>
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Table 3

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<th>Esophageal dysphagia: mechanical versus motor disorders</th>
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<td>History</td>
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<tr>
<td>Mechanical Disorder</td>
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<tr>
<td>Motor Disorder</td>
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<tr>
<td>Onset</td>
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<td>Gradual or sudden</td>
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<tr>
<td>Usually gradual</td>
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<tr>
<td>Progression</td>
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<tr>
<td>Often</td>
</tr>
<tr>
<td>Usually not</td>
</tr>
<tr>
<td>Type of bolus</td>
</tr>
<tr>
<td>Solid (unless high-grade obstruction)</td>
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<tr>
<td>Solids or liquids</td>
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<tr>
<td>Response to bolus</td>
</tr>
<tr>
<td>Often must be regurgitated</td>
</tr>
<tr>
<td>Usually passes with repeated swallowing or drinking liquids</td>
</tr>
<tr>
<td>Temperature dependent</td>
</tr>
<tr>
<td>No</td>
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<td>Worse with cold liquids; may improve with warm liquids</td>
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contour plot topographic analysis enhances the sensitivity of conventional manometry in the diagnosis of achalasia.12

Treatment of achalasia centers on decreasing basal LES pressure. Medical and surgical treatments exist and include medications, botulinum toxin injection, pneumatic dilatation, and myotomy. Treatment is oriented toward symptom relief and improvement in dysphagia as well as in objective measures of esophageal and LES function. As a result of inefficacy and many side effects, medical therapy with calcium channel blockers such as nifedipine is generally considered a temporizing measure while awaiting definitive management with more invasive therapy. Botulinum toxin injection into the LES decreases basal pressure by targeting acetylcholine-releasing neurons and carries a low side effect profile. Several studies have shown good efficacy and improved symptoms with this treatment, although benefits tend to be short lived and additional injections are required.13,14 Balloon pneumatic dilatation has been used for many years with good success, and numerous long-term outcome studies have shown it to be an effective first-line treatment of achalasia.10,12 The most serious complication from pneumatic dilation is esophageal rupture, which occurs at a mean rate of 2.6%. Laparoscopic myotomy has been shown to have a 90% success rate. However, it can lead to significant GERD and its associated complications. Consequently, myotomy is often performed in conjunction with a GERD-reducing procedure such as fundoplication.

**Diffuse esophageal spasm**

Like achalasia diffuse esophageal spasm (DES) is a primary motor disorder; however, the true pathophysiology is poorly understood. In general DES is characterized by
simultaneous contractions of the distal esophageal smooth muscle, which result in dysphagia and chest pain. Decreased levels of nitric oxide in the myenteric plexus have also been implicated in the pathophysiology. The true prevalence of DES is not known because most of the epidemiologic data are derived from patients referred for a variety of complaints, including noncardiac chest pain and dysphagia. In these patients the prevalence was estimated at 4% to 4.5%.\textsuperscript{15,16} Furthermore, studies do not reveal a clear relationship between the prevalence of the disease and factors such as age, gender, or race, although data suggest an association with mitral valve prolapse, obesity, and psychiatric illness.\textsuperscript{17}

The diagnosis of DES is made by esophageal manometry criteria and specifically requires synchronous pressure waves (>8 cm/s propagation) with a minimum amplitude of 30 mm Hg.\textsuperscript{18–20} However, a clear relationship between these manometric criteria and symptoms has not been established. The use of high-resolution manometry may help in diagnosing DES.

Treatment of DES involves medical and surgical options, with varying degrees of success. Trials of acid suppression with proton pump inhibitors (PPIs), anticholinergic medications, calcium channel blockers, and long-acting nitrates can result in improvement of symptoms, although not with any consistency. Treatment with antidepressants, most commonly trazodone, has been shown to decrease the pain associated with DES in some patients. Botulinum toxin injection in the distal esophageal body has been effective, although there are no large trials to evaluate long-term outcomes. Balloon dilation, bougienage, and esophagomyotomy are reserved for patients who have failed medical therapy.

**Nutcracker esophagus**

Nutcracker esophagus is a neuromuscular disorder characterized by high-amplitude peristaltic contractions of prolonged duration that may result in dysphagia and chest pain.\textsuperscript{21} The diagnosis is frequently made in patients undergoing manometry for evaluation of noncardiac chest pain. Diagnosis is confirmed with manometry when distal esophageal body contractions reach an amplitude of more than 180 mm Hg. Some studies have suggested that changing the diagnostic criteria to require amplitudes as high as more than 260 mm Hg may increase the sensitivity for nutcracker esophagus.\textsuperscript{22} Much like in DES and other spastic esophageal disorders, manometric findings often do not correlate with symptoms, and the cause of associated pain is unclear. Treatment is similar to that for DES and includes treatment of anxiety and depression.

**Mechanical Causes of Dysphagia**

**Benign esophageal strictures**

Benign esophageal strictures form in the setting of chronic inflammation and as a consequence of mucosal and epithelial injury, leading to collagen deposition and fibrous tissue formation.\textsuperscript{23} Most benign strictures (70%–75%) result from chronic inflammation of the esophageal mucosa by gastric acid from peptic ulcer disease (PUD) and GERD.\textsuperscript{24} Esophageal strictures may also be caused by Schatzki rings, pill esophagitis, corrosive ingestions, reaction to chronic foreign bodies (nasogastric tubes), sclerotherapy, radiation therapy, and surgery. Benign strictures are further characterized as simple and complex based on their length, angulation, luminal narrowing, and ability to pass an endoscope.\textsuperscript{23}

Peptic esophageal strictures have a prevalence of 0.1%, and male gender, increased age, and white race are associated with higher rates.\textsuperscript{25,26} Peptic strictures typically occur in the distal esophagus, where acid reflux is most frequent.
Midesophageal to upper esophageal strictures should heighten suspicion of Barrett esophagus and malignancy. Schatzki rings are membranous mucosal rings formed in the distal esophagus at the gastroesophageal junction and represent a variant of peptic stricture disease. Patients often present with dysphagia to solids greater than liquids. Diagnosis can often be made by history alone but is supplemented by barium esophagram and upper endoscopy findings.

Treatment of benign strictures aims both to relieve the obstruction as well as to prevent recurrence. Dilation of the stricture and esophageal lumen is the mainstay of therapy and can be accomplished with balloon dilators, mechanical dilators, and stents, depending on the nature and location of the stricture. Complications include esophageal perforation (0.1%–0.4%), bleeding, and bacteremia. Patients often require repeat dilation for recurrence. Treatment of GERD and PUD with antihistamines and PPIs is essential to prevent formation of strictures.

**Diverticula**

Esophageal diverticula are a rare cause of mechanical dysphagia. The true prevalence of this disease is unknown, but an association with increased age and male gender has been noted. Diverticula are characterized by location, wall structure, origin, and mechanism of formation. Most adult diverticula are acquired rather than congenital and have false walls lacking a full muscularis layer. Although midesophageal diverticula occur, most diverticula occur in the distal 10 cm of the esophagus in the epiphrenic region and are formed by pulsion. Risk factors for diverticular formation include esophageal dysmotility, obstruction, and focal wall weakness.

Dysphagia associated with diverticula is generally felt to be secondary to the motility disorder and obstruction that led to the diverticula formation rather than to the diverticula itself. Most patients with diverticula are asymptomatic, and the size of the diverticulum does not correlate with the presence of symptoms. If present, symptoms are typically progressive and include dysphagia, regurgitation, vomiting, aspiration, halitosis, chest pain, dyspnea, and dysrhythmias. Furthermore, diverticular rupture can lead to significant complications. Diverticula can be diagnosed on chest radiographs but are often discovered on esophagrams and upper endoscopy in patients being evaluated for dysphagia. Endoscopy should be performed to evaluate for associated malignancy.

Treatment is reserved for symptomatic patients and requires diverticulectomy with or without myotomy. Complications include recurrence of diverticula, esophageal leaks, and postoperative GERD.

**Esophageal neoplasms**

Two types of esophageal cancer exist: esophageal squamous cell carcinoma (ESCC) and esophageal adenocarcinoma (EAC). Over the last 30 years there has been a significant change in the epidemiology of esophageal cancer; EAC has become more prevalent than ESCC in the United States and among white individuals in most of the world. ESCC remains the most prevalent esophageal cancer in nonwhites. The increased prevalence of EAC has been attributed to a concomitant increase in GERD related to higher rates of obesity and fat ingestion in white individuals. Risk factors for EAC include male gender, smoking, excess alcohol consumption, obesity, GERD, Barrett esophagus, and excess fat consumption.

Barrett esophagus refers to a metaplastic change in the esophageal lining from squamous epithelium to intestinal columnar epithelium. This change is preceded by destruction of the squamous epithelium, most commonly by chronic acid exposure. Barrett esophagus is considered a precancerous condition and predisposes
individuals to developing EAC; however, the precise absolute risk is unclear. Management of Barrett esophagus is guided by the degree of cellular atypia noted on biopsy results and includes antacid medications, antireflux surgical procedures, endoscopic surveillance, and esophageal resection.\textsuperscript{30,31} Much controversy still exists regarding the role of screening for EAC in patients with Barrett esophagus.

Patients with esophageal neoplasms generally present with dysphagia that starts with solids and rapidly progresses to liquids. Dysphagia is often associated with significant weight loss. Definitive diagnosis of esophageal cancer is made by pathologic analysis of endoscopic biopsy samples.

Treatment of esophageal neoplasms is determined by staging. Endoscopic ultrasound, computed tomography (CT), and fludeoxyglucose F 18 positron emission tomography (PET) can be used to assess local wall infiltration, lymph node involvement, and the presence of metastases.\textsuperscript{33} Treatment of local minimally invasive cancer including high-grade interepithelial neoplasia, Barrett esophagus, and cancer limited to the mucosa can be treated with ablative techniques such as argon plasma coagulation as well as with endoscopic mucosal resection. In contrast, surgical resection is still considered the treatment of choice for advanced esophageal cancers. Other treatment options include neoadjuvant chemotherapy, chemotherapy alone, and radiation therapy. However, the benefits and efficacy of these therapies are still unclear because of limited data, and their use must be carefully weighed against the detriment they can cause to the patient’s quality of life.\textsuperscript{33}

**EMERGENCY DEPARTMENT EVALUATION AND MANAGEMENT OF DYSPHAGIA**

Evaluation and treatment of dysphagia in the emergency department (ED) are limited. A thorough history can often allow the emergency physician to differentiate between oropharyngeal and esophageal dysphagia and subsequently between mechanical and neuromuscular causes. All patients older than 40 years presenting with rapidly progressive dysphagia and weight loss should be suspected of having esophageal cancer.

Physical examination should focus on general appearance, head, nose, mouth, oropharynx, neck, chest, and abdomen. Neck examination should include auscultation for stridor and palpation for lymphadenopathy and thyromegaly. Cranial nerve examination is essential in the evaluation of oropharyngeal dysphagia. If available, nasopharyngoscopy may be helpful in the evaluation of patients presenting with symptoms concerning for proximal obstruction. If they can tolerate it, patients should be observed swallowing.

Plain radiographs of the chest and soft tissues of the neck may reveal obstructive masses typically in the setting of esophageal dysphagia. Laboratory testing is of little usefulness in the emergent evaluation of dysphagia but should be considered in patients with signs of dehydration and significant malnutrition.

Although a barium esophagram should be the initial radiographic test performed in the evaluation of dysphagia, it is seldom performed in the ED and should be arranged on an outpatient basis by a primary care physician. Further outpatient evaluation can include upper endoscopy and manometry.

ED treatment of dysphagia is also limited and should be aimed at addressing acute issues such as dehydration, electrolyte abnormalities, and acute obstruction. Gastroenterology consultation should be obtained on all patients with signs and symptoms of acute esophageal obstruction and inability to tolerate oral intake. These patients often require emergent endoscopy to relieve the obstruction. A trial of nitrates can be
considered in patients in whom DES is suspected, whereas motility disorders can be treated initially with calcium channel blockers such as diltiazem or nifedipine if no contraindications exist.

All stable patients should be referred to a primary care physician for a complete workup.

GERD

GERD is the most common gastroesophageal disorder encountered in the ED. Nevertheless, clinicians have struggled to devise definite criteria for the diagnosis of GERD. Some degree of gastroesophageal reflux (GER) and heartburn is physiologic and alone is not felt to constitute a disease. GERD is now defined as the presence of GER in association with troublesome symptoms and/or complications. Diagnosis can be made based on clinical symptoms alone or by showing reflux of gastric contents as well as its associated injuries.

Prevalence in the Western world is estimated at 15% to 25%, with a steadily increasing incidence. In contrast, the prevalence in Asia is estimated at less than 5%. In the United States alone the annual cost of managing the disease is estimated at more than $14 billion. The true prevalence of reflux esophagitis is unknown, and studies indicate that up to one-third of patients diagnosed with reflux esophagitis are asymptomatic. The most recognized risk factor for GERD is obesity, although the precise causal mechanism is unclear. Although gender does not seem to be a risk factor for symptomatic GER, endoscopic studies indicate that males seem to have a higher incidence of esophagitis. In addition, the escalating incidence of GERD has also been attributed to a decline in Helicobacter pylori infection. It has been suggested that H pylori–associated gastritis reduces GERD by decreasing parietal cell mass and gastric acid secretion.

Heartburn and acid reflux are the typical symptoms reported by patients with GERD. Symptoms classically occur after large meals, and heartburn is exacerbated by intake of alcohol, fats, spicy food, and citrus. The supine position and bending over also tend to exacerbate both heartburn and acid regurgitation. Dysphagia, odynophagia, burping, water brash, and cough are additional symptoms associated with GERD. Complications of GERD include esophagitis, peptic strictures, and Barrett esophagus. It is also believed that GERD can exacerbate asthma, chronic cough, recurrent pneumonitis, and dental erosion.

Abundant diagnostic tests for GERD exist, and consequently efficient testing is paramount. Testing for GERD may involve evaluation for reflux, esophageal mucosal injury, and esophageal and LES function with a combination of endoscopy, esophageal pH monitoring, and manometry. In patients with symptomatic GER, improvement after a trial of PPIs is also diagnostic.

Dietary and lifestyle modifications, medications, and surgical procedures are all used in the spectrum of GERD treatment. Although smoking, alcohol intake, fats, chocolate, and citrus are believed to exacerbate GERD, studies have not shown that avoiding these substances leads to significant symptom improvement. Over-the-counter antacids and H2 receptor antagonists are effective in the treatment of mild disease, whereas PPIs have been shown to decrease gastric acid production for longer times and constitute the mainstay of therapy for moderate disease. PPIs have also been shown to reverse esophagitis and the changes associated with Barrett esophagus. Chronic use of PPIs has been associated with gastric polyp formation, recurrent pneumonias, and enteric infections. There are some data to suggest that baclofen may also help reduce esophageal acid reflux by decreasing LES relaxation. Various endoscopic techniques and surgery are typically reserved for severe and
intractable disease, although it is unclear whether these procedures are more effective than medical therapy.

ED evaluation and management of patients presenting with symptoms suggestive of GERD can be difficult. Initial evaluation requires exclusion of life-threatening cardiac and abdominal conditions. Acute coronary syndrome and angina can masquerade as GERD especially in diabetic and elderly patients. Consequently, heartburn should warrant a thorough evaluation in such patients. Physicians should be wary of excluding cardiac disease because of symptomatic improvement with antacids, H2 blockers, and PPIs. Gastrointestinal (GI) disorders such as pancreatitis, peptic ulcer disease, and biliary colic may also present with similar symptoms. Clinicians should rely on a thorough history and physical examination to guide their management. Laboratory testing and imaging are nondiagnostic and of little usefulness in the ED management of GERD but may be helpful in excluding other conditions. ED treatment of GERD typically includes use of antacids, H2 blockers, and PPIs. In cases in which GERD is deemed the most likely diagnosis, patients should be started on a trial of H2 blockers or PPIs and advised to follow up with a primary care physician for further evaluation.

ESOPHAGITIS

Inflammation of the esophagus, referred to in general as esophagitis, has many causes, including infection, radiation exposure, GERD, sclerotherapy, and medications; it can also be immune mediated. Infectious esophagitis can be caused by a variety of pathogens, including viruses, fungi, and bacteria, and is most commonly encountered in immunocompromised hosts. Candida, herpes simplex, and cytomegalovirus are the most common causative organisms in such patients. In addition, diabetic patients, patients on chronic steroids, and those undergoing treatment with broad-spectrum antibiotics are all at increased risk for candidal esophagitis.

Patients suffering from infectious esophagitis typically present with complaints of dysphagia, odynophagia, and chest pain. Only in the most severe cases do patients present with dehydration from inability to tolerate liquids. Accurate diagnosis requires endoscopy and culture.

Treatment of candidal esophagitis depends on the host. Diabetic patients and patients on chronic corticosteroids who are deemed not to be severely immunocompromised can be treated as outpatients with oral nystatin solution. In contrast, severely immunocompromised patients require admission for treatment with systemic antifungal medications such as ketoconazole and amphotericin B. Similarly, systemic antivirals such as acyclovir are indicated in the treatment of esophagitis secondary to herpes simplex.

Radiation-induced esophagitis is a common complication of patients undergoing localized radiation therapy for oral, neck, lung, esophageal, and mediastinal cancers. Severity is directly related to radiation exposure. Diagnosis is mostly clinical and should be suspected in patients undergoing radiation therapy who present with dysphagia and odynophagia, with the caveat that infectious causes should be ruled out. Early endoscopic findings include an erythematous and friable esophageal mucosa that can progress to more extensive fibrinous tissue deposition and frank strictures. Initial treatment is with topical anesthetics such as viscous lidocaine and xylocaine. Endoscopic dilation may be required in patients who develop significant strictures.

Medication-induced esophagitis occurs when an ingested substance remains in contact with the esophageal mucosa for a prolonged period. Such prolonged contact
leads to localized mucosal irritation and inflammation. Antibiotics are most often implicated with tetracycline and its derivatives, accounting for more than half of cases. Other medications known to cause esophagitis include iron and potassium preparations, quinidine, nonsteroidal antiinflammatory drugs (NSAIDs), and alendronate. Capsule size, fluid intake, swallowing position (taking the medication when lying down or lying down immediately after ingestion), and age are additional risk factors. Treatment is conservative, with removal of the offending medication and instruction on proper medication ingestion.

EE refers to a syndrome in which the esophageal mucosa is infiltrated by eosinophils. The true prevalence in the United States is unknown although incidence is believed to be increasing. Male gender (3:1 male/female ratio) and younger age have been reported as risk factors for the disease. The pathophysiology of EE is also poorly understood but is believed to be secondary to both allergic and immunologic mechanisms. Both immunoglobulin E (IgE)-mediated and non–IgE-mediated allergic reactions have been implicated. Up to 60% of patients diagnosed with EE have been noted to have concomitant food, inhalant, and seasonal allergies. Furthermore, GERD and environmental allergens have been postulated to play a role in the pathophysiology of EE.

Patients typically present with complaints of dysphagia, more commonly to solids, and food impaction. Other symptoms may include GERD, diarrhea, weight loss, chest pain, and abdominal pain. Children may present with difficulty feeding, vomiting, and regurgitation. Diagnosis is made with a combination of endoscopy and biopsy. Suggestive endoscopic findings include esophageal rings, raised white specks, longitudinal furrows, whitish exudates, and extremely friable mucosa. These endoscopic findings are suggestive of EE but definitive diagnosis requires biopsy. Although definitive criteria have not been established, studies indicate that multiple samples should be obtained from different esophageal locations and should contain at least 15 eosinophils per high-power field. Additional diagnostic criteria include normal distal esophageal pH and no symptomatic improvement with PPIs. Given the association of EE with atopic conditions such as asthma and environmental allergies, some advocate further evaluation with skin prick testing, skin patch testing, and peripheral eosinophil counts.

Treatment of EE includes dietary modifications and avoidance of suspected allergens, swallowed and inhaled topical corticosteroids, systemic corticosteroids, and possibly endoscopic dilation. Leukotriene receptor antagonists have not been shown to be effective in the treatment of EE and are not recommended.

CAUSTIC INGESTIONS

Caustic injuries with strong acids and alkalis represent a true esophageal emergency, with the potential to cause severe morbidity and mortality. In 2004 the American Association of Poison Control Centers documented more than 200,000 exposures to caustic substances via household and industrial products. Several factors determine the extent of esophageal injury with caustic ingestions: amount and concentration of substance ingested, substance pH, tissue contact time, and the state of the substance (solid, liquid, or gas).

Caustic injury to esophageal tissue differs with acids and alkalis. Acid exposure leads to coagulation necrosis and eschar formation. This process is believed to limit the extent to which deeper tissues are affected. In contrast, alkali ingestions result in liquefactive necrosis and saponification, leading to increased penetration and injury to deeper tissues. These classic teachings may be correct in the setting of mild acids...
and alkalis; however, rapid penetration into esophageal tissues has been noted with both strong bases as well as acids. Initial esophageal injury occurs within minutes of exposure, may continue for hours, and leads to tissue necrosis. The initial injury is followed by a period of mucosal sloughing, bacterial invasion, and ulceration that can be complicated by esophageal perforation and can last up to 1 week. Esophageal repair begins approximately 10 days after the initial injury.

After a caustic ingestion, patients may be asymptomatic or present with a variety of GI and respiratory symptoms, including dyspnea, dysphagia, odynophagia, drooling, nausea, vomiting, abdominal pain, and chest pain. Laboratory studies, radiographs, and esophagogastroduodenoscopy (EGD) are essential to the initial evaluation. Studies that have attempted to correlate a variety of laboratory values with morbidity and mortality have had mixed results; however, laboratory tests are crucial to the diagnosis and management of severe acidosis, disseminated intravascular coagulation, hemolysis, renal failure, and liver failure. Patients presenting with abdominal pain and peritoneal findings require chest and abdominal radiographs to evaluate for viscus perforation both in the abdomen and the mediastinum. Management of esophageal injury is mostly guided by EGD findings. EGD is considered safe after caustic ingestion except in the setting of known or suspected viscus perforation, when it is contraindicated. Indications for EGD after minor caustic ingestions in asymptomatic patients are controversial; however, all patients noted to have posterior pharyngeal burns, dyspnea, stridor, vomiting, chest pain, and abdominal pain should undergo endoscopy. EGD findings are used to grade the severity of injury based on the extent and depth of the burn. Endoscopic ultrasound and technetium 99m sucralfaté–swallowing studies have shown potential in detecting deeper tissue injury as well as in documenting healing.

Initial treatment of caustic ingestions should focus on airway management and hemodynamic assessment. Airway compromise should be managed by nasotracheal or endotracheal intubation and possibly with a surgical airway. Laryngoscopy can also help determine the presence of upper airway edema. Adequate intravenous (IV) access should be obtained to enable aggressive resuscitation. Use of activated charcoal is contraindicated because it does not adsorb caustic agents. Patients with evidence of viscus perforation, whether abdominal or mediastinal, require emergent surgery.

Studies in animal models indicate that early pH neutralization therapy with weak acids and bases and dilution with water or milk ingestion can decrease esophageal injury; however, no human data exist and these interventions are not recommended at this time. There are also no data to support the use of H2 blockers and PPIs. Hence, initial ED treatment should focus on airway and hemodynamic management, pain control, and evaluation for esophageal and intestinal perforation. Once the patient has been stabilized, further management of esophageal injuries aims to prevent and treat esophageal strictures.

**ESOPHAGEAL FOREIGN BODIES**

Foreign bodies in the esophagus can result in dysphagia, odynophagia, and perforation; however, 80% to 90% of ingested foreign bodies traverse the GI tract spontaneously. The sites at which foreign body impaction is most likely to occur correspond to the areas of anatomic narrowing of the esophageal lumen: proximally at the level of the cricopharyngeal muscle (23%–36%), in the midesophagus at the level of the aortic arch and left mainstem bronchus, and distally at the LES (38%–52%). In adults, impaction is more likely to occur in the distal esophagus,
whereas in pediatric patients most impactions occur proximally. Of esophageal foreign body ingestions, 75% to 80% occur in children, typically aged 18 to 48 months. Other at-risk populations include edentulous adults, psychiatric patients, and prisoners.

The nature of the foreign bodies ingested varies significantly. Most impactions in adults result from food boluses (38%–59%) followed by bones (16%–18%), dental prostheses (2%–10%), pills (3%), coins (2%), and batteries (1%). In contrast, most impactions in children are a consequence of coin ingestions. Size and shape of the ingested material determine the likelihood that the foreign body passes through the GI tract without complication. Objects less than 20 mm typically pass through the esophagus and into the stomach and generally do not require acute retrieval. Once in the stomach these smaller foreign bodies are expected to traverse the rest of the GI tract. Objects larger than 20 mm in children and 6 cm in adults that pass through the esophagus should be removed because they can become impacted at the pylorus or in the duodenum. Sharp objects such as toothpicks, safety pins, bones, and tacks are more likely to cause perforations and should be removed before they pass beyond the pylorus. Overall, less than 1% of all ingested foreign bodies result in perforation. Dysphagia, odynophagia, and chest pain are the most common presenting symptoms in adults and can be accompanied by regurgitation, drooling, and inability to tolerate secretions in cases of complete obstruction. Proximal obstructions may lead to significant dyspnea, coughing, and stridor if the trachea is compressed. Abdominal pain can result when obstructions occur in the stomach or more distally. Up to 35% of pediatric patients with ingested foreign bodies are asymptomatic, and a high index of suspicion must be maintained, especially when the history is limited and unclear.

Initial evaluation should focus on determining the nature and location of the impacted foreign body. The ABCs of ED management are essential in these patients, and the history and physical examination must evaluate for signs and symptoms of airway compromise, hemodynamic instability, and perforation. Fiberoptic nasopharyngoscopy can be used to visualize suspected foreign bodies in the pharynx and proximal esophagus. Anteroposterior (AP) and lateral radiographs of the neck, chest, and abdomen are useful in detecting radiopaque foreign bodies but do not detect food boluses. Up to 70% of bones are not detected on radiographs because of poor calcification. Symptomatic patients with negative radiographs should undergo upper endoscopy for further evaluation. Endoscopy in asymptomatic patients with negative radiographs should also be considered on a case-by-case basis depending on the nature of the ingested foreign body.

Ingested foreign bodies can be managed medically, endoscopically, and surgically. Medical therapy with IV glucagon has been shown to have variable success rates, ranging from 12% to 50%, with higher rates for distal obstructions when glucagon acts to relax the smooth muscle. Typical doses range from 1 to 2 mg and can be repeated once. Nausea, vomiting, and hyperglycemia are typical side effects, and contraindications include pheochromocytoma, insulinoma, and Zollinger-Ellison syndrome. Some investigators advocate use of benzodiazepines in conjunction with glucagon because they are believed to result in striated muscle relaxation. Calcium channel blockers, nitrates, and anticholinergic medications have not been shown to be effective. Papain, meat tenderizer, and gas-forming pellets are also not recommended because they can lead to complications of esophageal digestion, perforation, and pulmonary edema.

Use of a Foley catheter to remove foreign bodies has been successful if performed within 72 hours and in cases in which a single smooth foreign object is involved. This
procedure requires sedation and should be performed under fluoroscopy in the presence of personnel with expert airway management skills. Some studies have shown the Foley catheter technique to be a more rapid and cost-effective alternative to endoscopy. Endoscopy with both flexible and rigid endoscopes has become the gold standard for foreign body retrieval and should be the modality of choice if available. Several different retrieval techniques are described, with success rates approaching 100%. Difficulties arise with small and sharp foreign objects, and surgery may be required in such cases. All sharp objects and button batteries lodged in the esophagus should be removed as soon as possible. Perforation rates with sharp objects can be as high as 35%, and button batteries contain alkalis that may cause esophageal corrosion, voltage burns, or pressure necrosis. Small batteries that have traversed the esophagus and passed into the stomach may be managed conservatively and given a chance to pass spontaneously.

**ESOPHAGEAL PERFORATION**

Esophageal perforation has many associated complications that can lead to severe morbidity. Esophageal injuries can range from superficial mucosal tears to full transmural perforations. Iatrogenic causes from endoscopic procedures or surgery account for most esophageal perforations. Other causes include Boerhaave syndrome, trauma, foreign bodies, pills, infections, and caustic ingestions. Complications of esophageal rupture are more likely to occur in the setting of complete transmural perforation. Esophageal perforations are further characterized by their onset as acute, subacute, and chronic.

Symptoms of esophageal perforation are largely dependent on the associated complications and vary based on the location of the perforation. Proximal perforations can lead to periesophageal abscess and can further spread into the posterior mediastinum through the retropharyngeal space. Patients can present with neck pain, dysphagia, odynophagia, and hoarseness, and the physical examination is often remarkable for subcutaneous air and crepitus. Thoracic perforations are more likely to lead to mediastinitis and pleural contamination, and patients often present with dysphagia, odynophagia, chest pain, back pain, dyspnea, and fever. Pneumomediastinum can often be detected on physical examination by auscultation of a crunching, rasping sound (Hamman sign). Distal esophageal perforations are more commonly associated with abdominal pain and peritoneal signs.

Boerhaave syndrome refers to spontaneous transmural esophageal rupture resulting from increased esophageal pressure from forceful vomiting. It is most commonly associated with alcohol abuse and PUD but has also occurred with seizures, heavy lifting, and laughing, in which intraabdominal pressure can be significantly increased. Rupture typically occurs in the left posterolateral aspect of the distal esophagus, and patients present with severe sudden onset of abdominal pain, chest pain, dyspnea, and shock.

Initial evaluation of esophageal rupture includes AP and lateral radiographs of the neck, chest, and abdomen to look for free air. Pneumomediastinum, pneumothorax, and pleural effusions are common in thoracic perforations. Perivertebral and abdominal free air can be seen in cases of cervical and abdominal perforations, respectively. Stable patients can be further evaluated with contrast esophagography, CT scan, and endoscopy.

Unstable patients require emergent airway stabilization and aggressive resuscitation. Adequate IV access should be obtained and shock treated with volume replacement and possibly vasopressors. Broad-spectrum antibiotics aimed at covering
gram-positive, gram-negative, and anaerobic organisms should be initiated. Stable patients with contained ruptures may be managed nonoperatively with antibiotics, nil-by-mouth status, and drainage of pleural effusions. Unstable patients with large ruptures require emergent surgery to undergo primary closure, esophagectomy, exclusion and diversion, or drainage. Endoscopic esophageal clipping and stenting represent alternatives to surgical management, although their role is not clearly defined. Mortality remains high both with operative and nonoperative management.

PUD

PUD has a high lifetime prevalence, ranging from 8% to 14%, and is the most common cause of upper GI bleeding. Estimated annual costs attributed to PUD surpass $9 billion. Prevalence is slightly greater in men than women and in elderly patients.

Ulceration of the gastric mucosa occurs when the physiologic mechanisms meant to protect the gastric mucosa are overwhelmed by the corrosive action of gastric acid. The most established risk factors for development of PUD are *H pylori* infection and chronic NSAID use. Infection with *H pylori* leads to cytokine-mediated mucosal inflammation, hypergastrinemia, and increased acid production. NSAIDs contribute to mucosal breakdown by inhibiting the synthesis of prostaglandins that function to protect the gastric mucosa by stimulating mucus and bicarbonate secretion. Once ulcers are formed, NSAIDs impede healing by preventing hemostasis and platelet aggregation. In the past, smoking, alcohol use, and dietary factors have also been associated with increased risk of PUD; however, definitive evidence to support a direct relationship is limited. On the other hand, some studies have reported that psychological stress has been shown to increase gastric acid production. Inflammatory, infectious, neoplastic, and endocrine causes have also been implicated in the development of PUD.

PUD classically manifests with periodic epigastric pain that can last weeks to months and is then followed by a period of remittance. This pain is commonly referred to as dyspepsia and can be associated with nausea and vomiting. In contrast to duodenal ulcers, pain secondary to gastric ulcers is typically absent during fasting and worsens immediately after eating. Patients may also report symptoms associated with GI bleeding or anemia. Complications of PUD include GI bleeding, perforation, gastric outlet obstruction, and penetration into adjacent structures. Perforation carries the highest mortality and is most commonly seen in elderly patients with chronic NSAID use. Perforations cause sudden severe abdominal pain, and patients typically present with peritoneal signs, a rigid abdomen, absent bowel sounds, and hemodynamic instability.

Diagnosis of PUD is typically made with upper GI endoscopy; however, it is not performed routinely at the time of initial presentation unless severe disease or gastric cancer is suspected. Patients are initially tested for *H pylori* infection and if positive treated accordingly. Several different assays for *H pylori* can be performed and include serology, urea breath testing, urine and fecal antigens, and endoscopic biopsy.

Treatment of PUD aims to promote ulcer healing and prevent recurrence. PPIs are the mainstay of pharmacotherapy. *H pylori* infections should be treated with a combination of PPIs, antibiotics, and a bismuth; several different regimens exist, some of which are available in combination packs to ease patient compliance. In cases of NSAID-induced PUD, offending agents must be discontinued.

ED evaluation requires a thorough history and physical examination aimed at differentiating PUD from other causes of epigastric and abdominal pain. Physicians should
further investigate for signs and symptoms of GI bleeding and anemia. As with GERD, cardiac and respiratory causes should also be considered and evaluated appropriately. Laboratory testing can be helpful in excluding pancreatic and biliary causes of abdominal pain and a complete blood count, coagulation studies, and blood typing should be obtained in all patients with evidence of significant GI bleeding. Patients presenting with dyspepsia should be treated with oral or IV PPIs but may require opioid analgesics for initial pain relief. In addition, nausea should be treated with antiemetics. Patients presenting with complications of PUD such as GI bleeding or perforation require aggressive management with volume resuscitation, transfusion, and endoscopy or surgery. All stable patients can be started on a PPI and should be referred to a primary care physician for further evaluation including *H pylori* testing.

**SUMMARY**

Gastroesophageal disease is frequently encountered in the ED and often presents subtly with a variety of symptoms that can mimic cardiovascular and respiratory disorders. Consequently, diagnosis of gastroesophageal disorders is frequently delayed. Laboratory testing and imaging are often helpful only in critically ill patients, and emergency physicians should focus on the history to differentiate gastroesophageal disorders from cardiorespiratory disease and to guide management. Whereas unstable patients require aggressive measures and a multidisciplinary approach, ED treatment of stable patients is often limited to alleviation of symptoms. All patients should be referred for definitive evaluation and treatment.

**REFERENCES**