Chapter 77: Esophageal Emergencies

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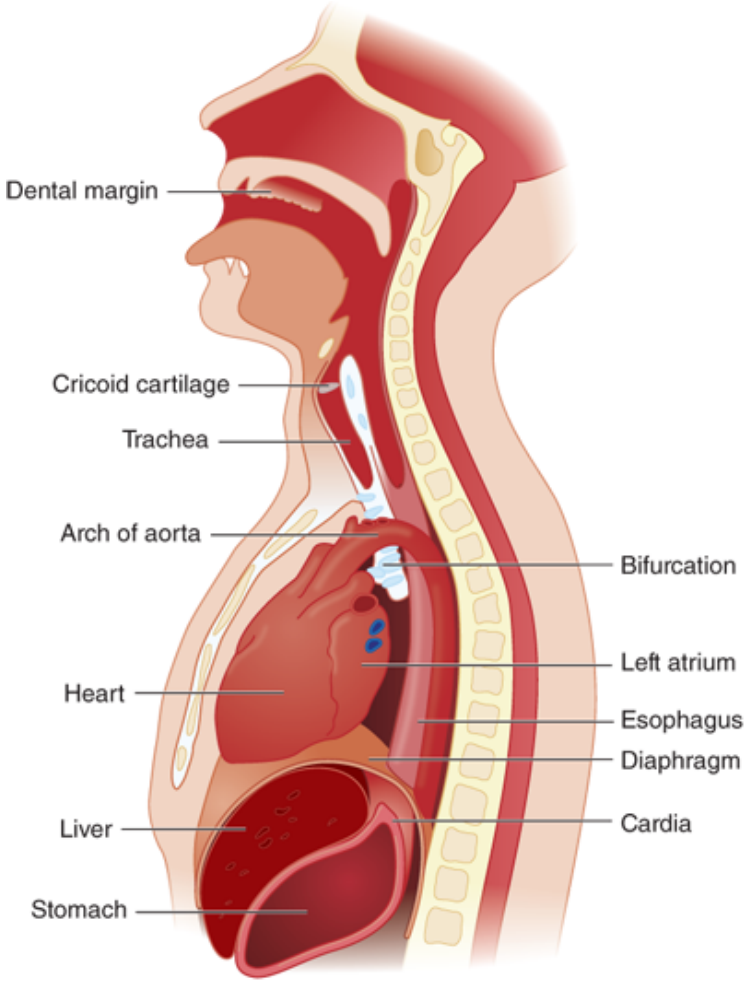
ESOPHAGEAL EMERGENCIES

The complaints of dysphagia, odynophagia, and ingested foreign body immediately implicate the esophagus. The esophagus also is often the site of pathology in patients who present with chest pain, upper GI bleeding (see chapter 75, "Upper Gastrointestinal Bleeding"), malignancy, and mediastinitis. Many diseases of the esophagus can be evaluated over time in an outpatient setting, but several, such as esophageal foreign body and esophageal perforation, require emergent intervention.

ANATOMY AND PATHOPHYSIOLOGY

The esophagus is a muscular tube approximately 20 to 25 cm long, primarily located in the mediastinum, posterior and slightly lateral to the trachea, with smaller cervical and abdominal components, as shown in **Figure 77–1**. There is an outer longitudinal muscle layer and an inner circular muscle layer. The upper third of the esophagus is striated muscle, while the lower half is all smooth muscle (including the lower esophageal sphincter). The esophagus is lined with stratified squamous epithelial cells that have no secretory function.

FIGURE 77–1.
Anatomic relations of the esophagus (seen from the left side). The distance from the upper incisor teeth to the beginning of the esophagus (cricoid cartilage) is about 15 cm (6 in); from the upper incisors to the level of the bronchi, 22 to 23 cm (9 in); and to the cardia, 40 cm (16 in). Structures contiguous to the esophagus that affect esophageal function are shown.



Two sphincters regulate the passage of material into and out of the esophagus. The upper esophageal sphincter prevents air from entering the esophagus and food from refluxing into the pharynx. The lower esophageal sphincter regulates the passage of food into the stomach and prevents stomach contents from refluxing into the esophagus. The upper sphincter is composed primarily of the cricopharyngeus muscle, with a resting pressure of around 100 mm Hg. The lower sphincter is not anatomically discrete. The smooth muscle of the lower 1 to 2 cm of the esophagus, in combination with the skeletal muscle of the diaphragmatic hiatus, functions as the sphincter, with a lower resting pressure around 25 mm Hg. An empty esophagus collapses, but three anatomic constrictions affect the adult esophagus:

1. At the cricopharyngeus muscle (C6)
2. At the level of the aortic arch (T4)
3. At the gastroesophageal junction (T10 to T11)

The pediatric esophagus gets two additional areas of constriction:

1. At the thoracic inlet (T1)
2. At the tracheal bifurcation (T6)

The innervation of the heart mirrors that of the esophagus, with visceral and somatic stimuli converging within the sympathetic system. This anatomy makes pain of esophageal and cardiac origin similar. The esophageal venous circulation includes a submucosal plexus of veins that drains into a separate plexus of veins surrounding the esophagus. Blood flows from this outer plexus in part to the gastric venous system, an important link between portal and systemic circulation. Variceal dilatation of the submucosal system can lead to massive upper GI bleeding.

DYSPHAGIA

Dysphagia is difficulty with swallowing. Most patients with dysphagia have an identifiable, organic cause.

Oropharyngeal (transfer) and esophageal (transport) dysphagia describe two broad pathophysiologic types. **Transfer dysphagia** occurs very early in swallowing (as the food bolus moves from the oropharynx through the upper sphincter) and is often reported as difficulty in initiating a swallow. **Transport dysphagia** is impaired movement of the bolus down the esophagus and through the lower sphincter. Transport dysphagia is perceived later in the swallowing process, usually 2 to 4 seconds or longer after swallowing is initiated, and most commonly results in the feeling of the food "getting stuck." A history geared toward discerning transfer from transport dysphagia provides a differential of the likely underlying pathology (Table 77–1). Another useful classification scheme divides dysphagia into that due to obstructive disease and that due to motor dysfunction. Functional or motility disorders usually cause dysphagia that is intermittent and variable. Mechanical or obstructive disease is usually progressive (difficulty swallowing solids, then liquids).[1]

Table 77–1

Dysphagia

Transfer Dysphagia (Oropharyngeal)	Transport Dysphagia (Esophageal)
Discoordination in transferring bolus from pharynx to esophagus	Improper transfer of the bolus from the upper esophagus into the stomach
Swallowing symptoms—gagging, coughing, nasal regurgitation, inability to initiate swallow, need for repeated swallows	Swallowing symptoms—food "sticking," retrosternal fullness with solids (and eventually liquids), possibly odynophagia
Risk of aspiration present	Risk of aspiration present, generally less pronounced than in transfer dysphagia
Long term—weight loss, malnutrition, chronic bronchitis, asthma, multiple episodes of pneumonia	Long term—malnutrition, dehydration, weight loss, systemic effects of cancer
Neuromuscular disease (80%)—cerebrovascular accident, polymyositis and dermatomyositis, scleroderma, myasthenia gravis, tetanus, Parkinson's disease, botulism, lead poisoning, thyroid disease	Obstructive disease (85%)—foreign body, carcinoma, webs, strictures, thyroid enlargement, diverticulum, congenital or acquired large-vessel abnormalities
Localized disease—pharyngitis; aphthous ulcers; candidal infection; peritonsillar and retropharyngeal abscesses; carcinoma of tongue, pharynx, larynx; Zenker's diverticulum; cricopharyngeal bar; cervical osteophytes	Motor disorder—achalasia, peristaltic dysfunction (nutcracker esophagus), diffuse esophageal spasm, scleroderma
Inadequate lubrication—scleroderma	Inflammatory disease

CLINICAL FEATURES

Although often an independent symptom, dysphagia can be associated with odynophagia, which is painful swallowing (suggesting an inflammatory process), or with chest pain that is esophageal in nature, suggesting gastroesophageal reflux disease (GERD) or a motility disorder. Question the patient regarding the course of symptoms (acute, subacute, or chronic, intermittent, progressive); food patterns (solids, liquids); location; and previous disease. Transport dysphagia that is present for solids only generally suggests a mechanical or obstructive process. Motility disorders typically cause transport dysphagia for solids and liquids.

Impaction of a poorly chewed meat bolus in the esophagus is a well-recognized complication of esophageal disease. A history of dysphagia may or may not be present. An impacted food bolus can be the presenting complaint for a variety of underlying esophageal pathologies. Patients are generally accurate in identifying location if the bolus is in the upper

third of the esophagus. Esophageal filling proximal to the impacted bolus can cause inability to swallow secretions and can present an airway/aspiration risk.

Focus the physical examination on the head and neck and perform the neurologic examination. Assess for signs of previous stroke, muscle disease, or Parkinson's disease. Cachexia and cervical or supraclavicular nodes can be observed in patients with cancer of the esophagus. The physical findings are often normal in patients with dysphagia.[2] Watch the patient take a small sip of water. Inability to swallow water generally confirms at least a partial obstruction.

DIAGNOSIS

The diagnosis of the underlying pathology is most often made outside of the ED. Initial evaluation of dysphagia in the ED can include anteroposterior and lateral neck radiographs, which can be helpful in transfer dysphagia and cases in which the transport dysfunction seems proximal. Obtain a chest x-ray if considering transport dysphagia. Direct laryngoscopy can be used to identify proximal lesions.

Diagnosis of oropharyngeal dysphagia uses a variety of tools. Traditional barium swallow is often recommended as a first test. Video esophagography is a specialized form of barium swallow study in which videotaped images are played at low speed to allow detailed analysis. Manometry and esophagoscopy are also used, depending on the clinical picture.[2] If a foreign body is suspected, the diagnostic evaluation takes yet another path (see later section, "Swallowed Foreign Bodies and Food Impaction").

Neoplasm

Neoplasms are a common cause of both transfer and transport dysphagia. The esophagus or surrounding structures can be the primary site. A large majority of esophageal neoplasms are squamous cell; the remaining are adenocarcinomas. Risk factors for squamous cell disease include alcohol, smoking, achalasia, and previous ingestion of caustic material with lye. Barrett's esophagus predisposes to adenocarcinoma. Surgery and radiation therapy for head and neck cancer are also important associations.[3]

There is usually a fairly rapid progression of dysphagia from solids to liquids (6 months). Bleeding is another sign suggesting neoplasm. Assume neoplasia in patients >40 years old with new-onset dysphagia. Definitive diagnosis is made by endoscopy with biopsy.

Anatomic Causes

Esophageal stricture develops as a result of scarring from GERD or other chronic inflammation. Generally strictures occur in the distal esophagus proximal to the gastroesophageal junction and may interfere with lower sphincter function. Symptoms may build over years and are often noted solely with solids. Stricture can serve as a barrier to reflux, so heartburn may decrease as dysphagia increases. Evaluation involves ruling out malignancy, and treatment is dilatation.[4]

Schatzki ring is the most common cause of intermittent dysphagia with solids. This stricture near the gastroesophageal junction is present in up to 15% of the population, and most are asymptomatic. A ring may form over time in response to GERD. Food impaction in the esophagus is a frequent presenting event with a Schatzki ring. The treatment is dilatation.[4]

Esophageal webs are thin structures of mucosa and submucosa found most often in the middle or proximal esophagus. They can be congenital or acquired. Esophageal webs also occur as a component of Plummer-Vinson syndrome (along with iron deficiency anemia) and can be seen in patients with pemphigoid and epidermolysis bullosa. Treatment is dilatation.

Diverticula can be found throughout the esophagus. Pharyngoesophageal or Zenker's diverticulum is a progressive out-pouching of pharyngeal mucosa, just above the upper sphincter, caused by increased pressures during the hypopharyngeal phase of swallowing. Symptom onset is often after age 50, as most diverticula are acquired rather than congenital. Patients complain of typical transfer dysfunction or halitosis and the feeling of a neck mass. Diverticula can also be seen in the mid or distal esophagus, the latter usually in association with a motility disorder.[5]

Neuromuscular and Motility Disorders

Neuromuscular disorders typically result in misdirection of food boluses with repeated swallowing attempts. Liquids, especially at the extremes of temperature, are generally more difficult to handle than solids, and symptoms are often intermittent. Stroke is the most common cause of this type of dysphagia. Oropharyngeal muscle weakness is often the mechanism, although upper sphincter dysfunction can also contribute. Polymyositis and dermatomyositis are also common causes of transfer dysphagia.

Achalasia is a dysmotility disorder of unknown cause and the most common motility disorder producing dysphagia. Impaired swallowing-induced relaxation of the lower sphincter is noted, along with the absence of esophageal peristalsis. Symptom onset is usually between 20 and 40 years of age. Achalasia may be associated with esophageal spasm and chest pain and with odynophagia. Associated symptoms can include regurgitation and weight loss. Dilation of the esophagus can be massive enough to impinge on the trachea and cause airway symptoms.[6] Therapy includes reduction of lower sphincter pressure by oral medications, endoscopic injection of botulinum toxin into the muscle of the sphincter, dilatations, or surgical myotomy.

Diffuse esophageal spasm is the intermittent interruption of normal peristalsis by nonperistaltic contraction. Dysphagia is intermittent and does not progress over time. Chest pain is a common symptom. Therapy involves control of acid reflux and consideration of smooth muscle relaxants and/or antidepressants, although effectiveness is unclear.[6]

Esophageal dysmotility is the excessive, uncoordinated contraction of esophageal smooth muscle. Clinically, chest pain is the usual presenting symptom of esophageal dysmotility. The onset is usually in the fifth decade. Pain often occurs at rest and is dull or colicky, and stress or ingestion of very hot or very cold liquids may serve as triggers. Acute pain may be followed by hours of dull, residual discomfort. Many patients also have dysphagia, usually intermittent. Pain from spasm may respond to nitroglycerin. Calcium channel blockers and anticholinergic agents can also be used. The other motility disorders commonly recognized include ineffective esophageal motility, hypertensive lower esophageal sphincter, and nutcracker esophagus. Nutcracker esophagus is a motility disorder in which there are high-amplitude, long-duration peristaltic contractions in the distal body of the esophagus or the lower sphincter. The cause is unknown.[6]

CHEST PAIN OF ESOPHAGEAL ORIGIN

Differentiating esophageal pain from ischemic chest pain is difficult at best and may be impossible in the ED. Patients with esophageal disease often report spontaneous onset of pain or pain at night, regurgitation, odynophagia, dysphagia, or meal-induced heartburn, but these symptoms are also found in patients with coronary artery disease, and there are no historical features with enough predictive value to allow accurate differentiation.

The ED working assumption is often that the pain is cardiac in nature. However, the incidence of esophageal disease in patients with chest pain and normal coronary arteries has been reported as up to 80%.[7] The use of ED observation units can help sort patients out with protocol-driven, rapid rule-out of infarction, followed by risk stratification for underlying acute coronary syndrome via a variety of modalities (see chapters 48, "Chest Pain" and 49, "Acute Coronary Syndrome" for further discussion). If a patient's presentation can be determined to be noncardiac in nature, treatment for reflux is often initiated empirically.[7]

GASTROESOPHAGEAL REFLUX DISEASE

Reflux of gastric contents into the esophagus causes a wide array of symptoms and long-term effects. Classically, a weak lower esophageal sphincter has been the mechanism held responsible for reflux, and this is seen in some patients. However, transient relaxation of the lower esophageal sphincter complex (with normal tone in between periods of relaxation) is the primary mechanism causing reflux. Hiatal hernia, prolonged gastric emptying, agents that decrease lower sphincter pressure, and impaired esophageal motility predispose to reflux.[8] **Table 77–2** highlights some common contributors.

Table 77–2

Causes of Gastroesophageal Reflux Disease

Decreased Pressure of Lower Esophageal Sphincter	Decreased Esophageal Motility	Prolonged Gastric Emptying
High-fat food	Achalasia	Medicines (anticholinergics)
Nicotine	Scleroderma	Outlet obstruction
Ethanol	Presbyesophagus	Diabetic gastroparesis
Caffeine	Diabetes mellitus	High-fat food
Medicines (nitrates, calcium channel blockers, anticholinergics, progesterone, estrogen)		
Pregnancy		

Heartburn is the classic symptom of GERD, and chest discomfort may be the only symptom of the disease. The burning nature of the discomfort is probably due to localized lower esophageal mucosal inflammation. Many GERD patients report other associated GI symptoms, such as odynophagia, dysphagia, acid regurgitation, and hypersalivation. Pain and discomfort with meals point to GERD. Reflux symptoms can sometimes be dramatically exacerbated with a head-down position or an increase in intra-abdominal pressure. Symptoms are often relieved by antacids, but pain can return after the transient antacid effect wears off. Some patients with symptoms due to ischemic disease also report improvement with the same therapy. Unfortunately, like cardiac pain, GERD pain may be squeezing and pressure-like, and the history may include pain onset with exertion and offset with rest. Both types of pain may be accompanied by diaphoresis, pallor, and nausea and vomiting. Radiation of esophageal pain can occur to one or both arms, the neck, the shoulders, or the back. Given the serious outcome of unrecognized ischemic disease, a cautious approach is warranted.

Over time, GERD can cause complications including strictures, dysphagia, and inflammatory esophagitis. A severe consequence of GERD, Barrett's esophagus, is present in up to 10% of patients with GERD.[9,10] Less obvious presentations of GERD also occur such as asthma exacerbations, sore throat, and other ear, nose, and throat symptoms. GERD has also been implicated in dental erosion, vocal cord ulcers and granulomas, laryngitis with hoarseness, chronic sinusitis, and chronic cough.

Treatment of reflux disease involves decreasing acid production in the stomach, enhancing upper tract motility, and eliminating risk factors. Mild disease is often treated empirically. Histamine-2 blockers (histamine-2 antagonists) or proton pump inhibitors are mainstays of therapy. A prokinetic drug may help. ED discharge instructions given to patients thought to be experiencing reflux-related symptoms should include: avoid agents that exacerbate GERD (ethanol, caffeine, nicotine, chocolate, fatty foods), sleep with the head of the bed elevated (30 degrees), and avoid eating within 3 hours of going to bed at night. The need for additional testing can be determined at outpatient follow-up care.

ESOPHAGITIS

Esophagitis can cause prolonged periods of chest pain and almost always causes odynophagia as well. Diagnosis of more established esophagitis is by endoscopy. Low-grade disease can be seen by histopathologic examination.

INFLAMMATORY ESOPHAGITIS

GERD may induce an inflammatory response in the lower esophageal mucosa. Over time, esophageal ulcerations, scarring, and stricture formation can develop. The presence of reflux-induced esophagitis warrants aggressive pharmacologic therapy with acid-suppressive medications. If this treatment regimen is not sufficient, surgical options are considered.[8] Ingested medications can also be a source of inflammatory esophagitis, usually from prolonged contact of the medication with the esophageal mucosa. Ulcerations can form. Multiple medications have been implicated. Common offenders include nonsteroidal anti-inflammatory drugs and other anti-inflammatory drugs, potassium chloride, and some antibiotics (e.g., doxycycline, tetracycline, and clindamycin). Risk factors for pill-induced esophageal injury include swallowing position, fluid intake, capsule size, and age. Withdrawal of the offending agent is generally curative. Eosinophilic esophagitis is a chronic allergic-inflammatory condition in which eosinophils and other immune system cells infiltrate the esophagus and induce an inflammatory response.[10] Diagnosis is by endoscopy. Treatment is avoidance of allergens and swallowed liquid corticosteroids. Dilatation is necessary if strictures form.

INFECTIOUS ESOPHAGITIS

Patients with immunosuppression can develop infectious esophagitis. The diagnosis of infectious esophagitis in an otherwise seemingly healthy host should prompt an evaluation of immune status. Candidal species are the most common pathogens, often associated with dysphagia as a primary symptom. Herpes simplex or cytomegalovirus infection and aphthous ulceration are also seen and may be more frequently associated with odynophagia. Other causative agents are rare and include fungi, mycobacteria, and other viral pathogens such as varicella-zoster virus and Epstein-Barr virus. Endoscopy with biopsy and specimen culture is used to establish this diagnosis.[11]

ESOPHAGEAL PERFORATION

Perforation of the esophagus can occur secondary to a number of disparate processes (Table 77–3). Iatrogenic perforation is the most frequent.[12]

Table 77–3

Causes of Esophageal Perforation

Cause of Perforation	Description
Iatrogenic	Intraluminal procedures Endoscopy Dilatation Variceal therapy Gastric intubation Intraoperative injury
Boerhaave's syndrome	"Spontaneous," usually associated with transient increase in intraesophageal pressure
Trauma	Penetrating Blunt (rare) Caustic ingestion
Foreign body	Includes pill-related injury
Infection	Rare
Tumor	May be intrinsic or extrinsic cancer
Aortic pathology	Aneurysm Aberrant right subclavian artery
Miscellaneous	Barrett's esophagus Zollinger-Ellison syndrome

Boerhaave's syndrome is full-thickness perforation of the esophagus after a sudden rise in intraesophageal pressure. The mechanism is sudden, forceful emesis in about three fourths of the cases; coughing, straining, seizures, and childbirth have been reported as causing perforations as well. Alcohol consumption is frequently an antecedent to this syndrome. The perforation is usually in the distal esophagus on the left side.

Blunt or penetrating **neck trauma** can cause esophageal perforation. Rupture from blunt injury is rare. Penetrating esophageal wounds are often masked by injury to the airway and major vessels. A combination of esophagography and esophagoscopy is used to assess patients for potential esophageal injury.

Foreign-body ingestion or food impaction may result in perforation of the esophagus as well (see below).

The perforation rate from endoscopy is lower in an esophagus free of disease than in a diseased esophagus. Dilation of strictures increases the risk of perforation greatly. Other intraluminal procedures, such as variceal therapy and

palliative laser treatment for cancer, are also associated with perforation. **Boerhaave's syndrome** is responsible for roughly 10% to 15% of esophageal perforations and is discussed above.

PATHOPHYSIOLOGY

Perforation causes a dramatic presentation if esophageal contents leak into the mediastinal, pleural, or peritoneal space. Fulminant, necrotizing mediastinitis, pneumonitis, or peritonitis can rapidly lead to shock. If the perforation is small and leakage is contained by contiguous structures, the course may be significantly more indolent. Most spontaneous perforations occur through the left posterolateral wall of the distal esophagus.[12] Proximal perforation, seen mostly with instrumentation, tends to be less severe than distal perforation and can form a periesophageal abscess with minimal systemic toxicity.

CLINICAL FEATURES

The pain of rupture is classically described as acute, severe, unrelenting, and diffuse, and is reported in the chest, neck, and abdomen. Pain can radiate to the back and shoulders. Back pain may be a very predominant symptom. The pain is often exacerbated by swallowing. Dysphagia, dyspnea, hematemesis, and cyanosis can be present as well.

Physical examination varies with the severity of the rupture and the elapsed time to presentation. Abdominal rigidity with hypotension and fever often occur early. Tachycardia and tachypnea are common. Cervical subcutaneous emphysema is common in cervical esophageal perforations. **Mediastinal emphysema takes time to develop. It is less commonly detected by examination or radiography in lower esophageal perforation, and its absence does not rule out perforation**. Hamman's crunch, caused by air in the mediastinum that is being moved by the beating heart, can sometimes be auscultated. Pleural effusion develops in half of patients with intrathoracic perforations but is uncommon in those with cervical perforations. Pleural fluid can be due to either direct contamination of the pleural space or a sympathetic serous effusion from mediastinitis.

DIAGNOSIS

Timely diagnosis in an ill patient with esophageal perforation requires suspicion on the clinician's part. Mistaking perforation for acute myocardial infarction, pulmonary embolism, or an acute abdomen can lead to delays in therapy. Chest radiographs can suggest the diagnosis. CT of the chest or emergency endoscopy is most often used to confirm the diagnosis. Perforation of the esophagus is associated with a high mortality rate regardless of the underlying cause. The pace of care and the location and etiology of the perforation all affect outcome. In the ED, resuscitate the patient from shock, administer broad-spectrum parenteral antibiotics, and obtain emergency surgical consultation as soon as the diagnosis is seriously entertained. Patients with systemic symptoms and signs after perforation need operative management.[12]

SWALLOWED FOREIGN BODIES AND FOOD IMPACTION

PATHOPHYSIOLOGY

Children 18 to 48 months of age and those with mental illness account for most cases of ingested foreign bodies. Small objects, such as coins, toys, and crayons, typically lodge in the anatomically narrow proximal esophagus. In adults, dentures are sometimes swallowed, because diminished palatal sensitivity leads to unintentional ingestion. Adult candidates for swallowed foreign bodies are those with esophageal disease, prisoners, and psychiatric patients. In

adults, most impactions are distal. In children and adults, once an object has traversed the pylorus, it usually continues through the GI tract and is passed without issue. If, however, the object has irregular or sharp edges or is particularly wide (>2.5 cm) or long (>6 cm), it may become lodged distal to the pylorous.[13] Esophageal impaction can result in airway obstruction, stricture, or perforation, the latter being the result of direct mechanical erosion (e.g., ingested bones) or chemical corrosion (e.g., ingested button batteries). Esophageal mucosal irritation (often mechanical from a swallowed bone, for example) can be perceived as a foreign body by the patient as well.

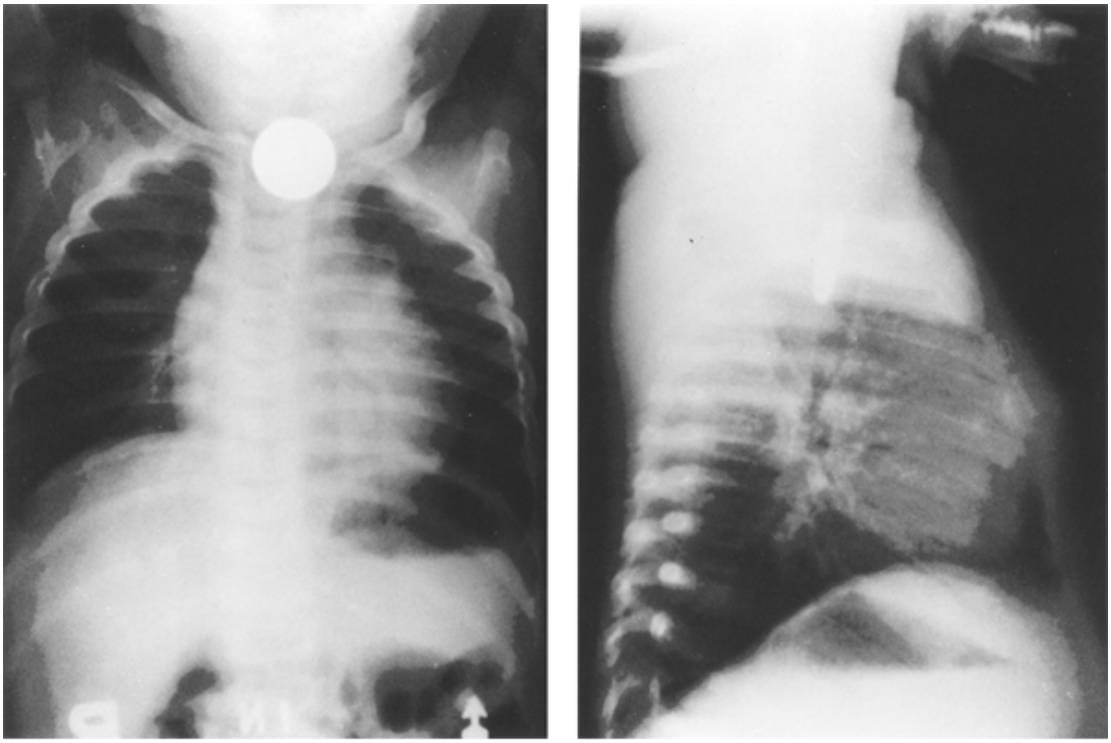
CLINICAL FEATURES

Adults with an esophageal foreign body generally provide unequivocal history. Patients often complain of retrosternal pain and may localize the object (often accurately in the upper third of the esophagus). Patients may have dysphagia, vomiting, and choking. If the patient attempts to wash down the object with liquid or if swallowed secretions pool proximal to the obstruction, coughing or aspiration can occur. **In children, the history can be unclear. Signs and symptoms can include refusal or inability to eat, vomiting, gagging and choking, stridor, neck or throat pain, and drooling. A high degree of suspicion is necessary for unwitnessed ingestions in children, especially in those <2 years of age**. Physical examination starts with an assessment of the airway. The nasopharynx, oropharynx, neck, and chest should also be examined but are often unremarkable. Occasionally, a foreign body can be directly visualized in the oropharynx.

DIAGNOSIS

Plain films are used to screen for radiopaque objects. Coins in the esophagus generally present their circular face on anteroposterior films (coronal alignment), as opposed to coins in the trachea, which show that face on lateral films (Figure 77–2). Obtaining plain films in patients with food impaction is rarely helpful and can generally be omitted. CT scanning is a very high-yield test for esophageal foreign body and has generally replaced the barium swallow test to evaluate ingestion of nonradiopaque objects. CT also delivers excellent information regarding perforation and subsequent infection.

FIGURE 77–2.
A coin lodged in a child's esophagus is visible on an anteroposterior radiograph. [Reproduced with permission from Effron D (ed): *Pediatric Photo and X-Ray Stimuli for Emergency Medicine*, vol II. Columbus, OH, Ohio Chapter of the American College of Emergency Physicians, 1997, case 27.]



TREATMENT

Endoscopy

Patients in extremis or with pending airway compromise are resuscitated and often require active airway management. Complete obstruction of the esophagus (often distal esophageal food impactions) can lead to proximal pooling of secretions and aspiration. Emergent endoscopy is needed. Table 77–4 lists other situations that require urgent endoscopy, even if patients are clinically stable. Some of these ingestions are discussed in more detail in the sections "Food Impaction," "Coin Ingestion," "Button Battery Ingestion," "Ingestion of Sharp Objects," and "Narcotics Ingestion" later in the chapter. In general, if endoscopy is clearly indicated, performing advanced imaging studies delays definitive intervention while adding little value to the care of the patient. In the vast majority of cases, the foreign body can be removed relatively easily with endoscopy without complication. Hospital admission is generally not needed.

Table 77–4

Circumstances Warranting Urgent Endoscopy for Esophageal Foreign Bodies

Ingestion of sharp or elongated objects (including toothpicks, aluminum soda can tabs)
Ingestion of multiple foreign bodies
Ingestion of button batteries
Evidence of perforation
Coin at the level of the cricopharyngeus muscle in a child
Airway compromise
Presence of a foreign body for >24 h

Laryngoscopy

In stable patients, indirect laryngoscopy or visualization of the oropharynx using a fiberoptic scope may allow removal of very proximal objects. For more distal objects, imaging studies are used to define the location and nature of the ingestion. Objects that persist over time or are in the upper half of the esophagus are less likely to pass, and consultation for endoscopy is prudent.

Expectant Treatment

If the object is distal to the pylorus, has a benign shape and nature, and the patient is comforTable and tolerating intake by mouth, treatment is expectant. For worrisome foreign bodies that are in the more distal GI tract, surgery consultation may be necessary.

Foley Catheter Removal

Alternatives to endoscopy have been advocated by some authors. These include use of a Foley catheter to remove coins (see section "Coin Ingestion" later in the chapter) and bougienage to advance the object from the esophagus into the stomach. Generally, the Foley catheter technique and bougienage should only be attempted in patients with blunt foreign bodies present for <24 hours and in patients without underlying esophageal disease. Provider comfort with the procedure likely influences the generally low complication rates in published series.[14]

Glucagon

For distal esophageal objects, **glucagon, 1 to 2 mg IV in adults,** has been used to relax the lower sphincter and allow passage of the object. **Success rates of glucagon therapy are generally reported as poor, however, and it may be no better than watchful waiting without other intervention.**[15]

SPECIAL CONSIDERATIONS

FOOD IMPACTION

Meat is the food most commonly identified in food impaction. Food impaction with complete esophageal obstruction or impaction of food containing bony fragments requires emergency endoscopy. Uncomplicated food impaction may be treated expectantly. Time and sedation often allow the bolus to pass into the stomach, but the bolus should not be allowed to remain impacted for >12 to 24 hours. **The use of proteolytic enzymes (e.g., Adolph's Meat Tenderizer®, which contains papain) to dissolve a meat bolus is *contraindicated*, because of the potential for severe mucosal damage and esophageal perforation, and the availability of superior alternatives**. If **glucagon** therapy is attempted, an initial dose of 1 to 2 milligrams IV is given (for adults). If the food bolus is not passed in 20 minutes, an additional dose can given.[15]

COIN INGESTION

Many centers use endoscopy to remove esophageal coins in children. Removal with a Foley catheter is done under fluoroscopy, and advanced airway management should be immediately available. The patient is placed in the Trendelenburg position to prevent aspiration of the object. The catheter is passed down the esophagus beyond the object, the balloon inflated, and the catheter slowly withdrawn, bringing the object with it. Complications can include airway compromise and mucosal injury, although in experienced hands, the rate of these events is low.[14]

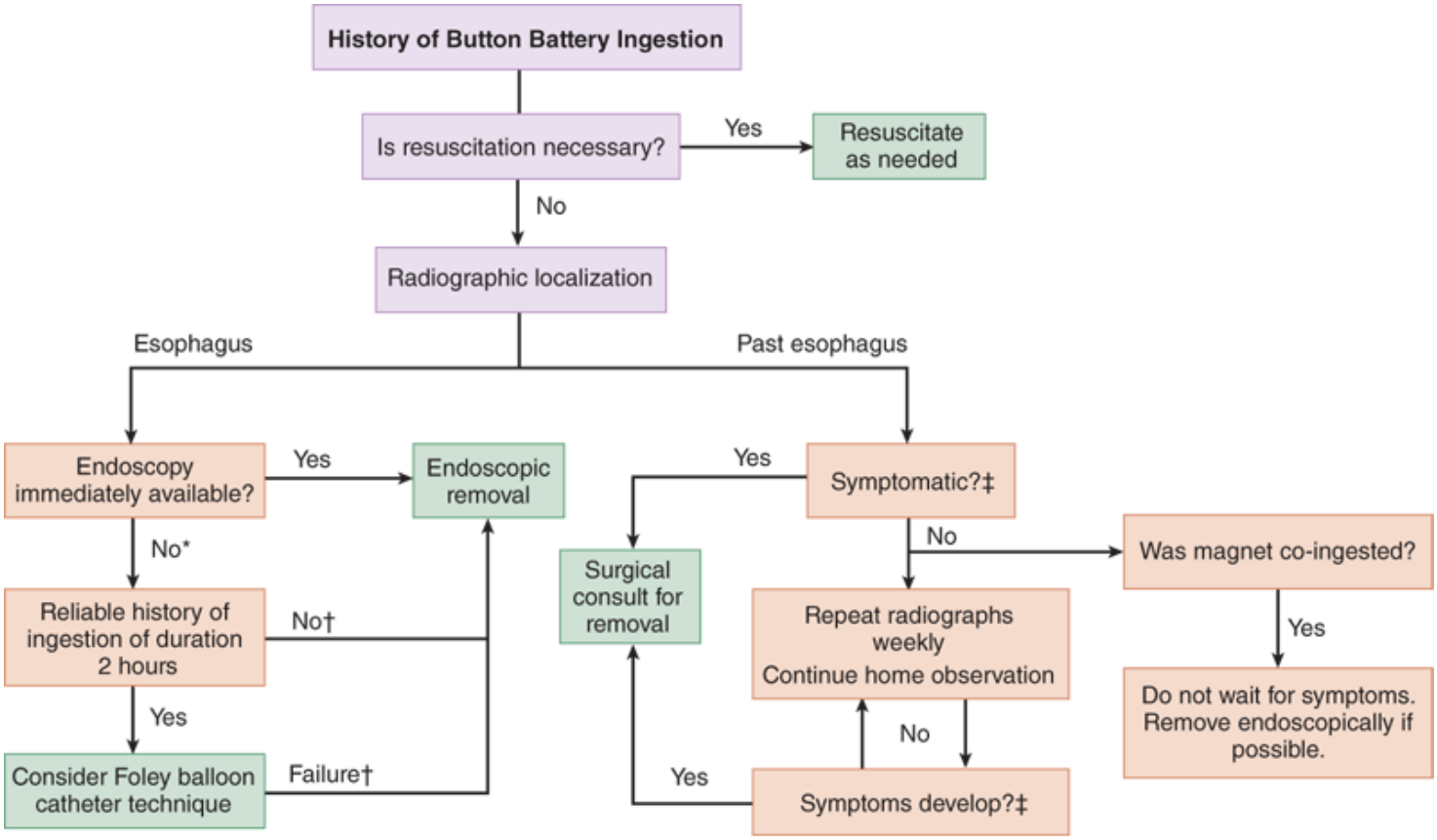
BUTTON BATTERY INGESTION

A button battery lodged in the esophagus is a true emergency requiring prompt removal, because the battery may quickly induce mucosal injury and necrosis. Perforation can occur within 6 hours of ingestion. Morbidity caused by the battery is likely related to the flow of electricity through a locally formed external circuit. Lithium cells are associated disproportionately with adverse outcome, probably due to higher voltage.[14]

Figure 77–3 outlines a management algorithm for button battery ingestion. Button batteries that have passed the esophagus can be managed expectantly, as long as follow-up in 24 hours can be assured. Repeat films should be obtained at 48 hours to ensure that the cell has passed through the pylorus (which may not occur if the battery is of large diameter and/or the patient is <6 years old). Most batteries pass completely through the body within 48 to 72 hours, although passage can take longer. Any patient with symptoms or signs of GI tract injury requires immediate surgical consultation. The National Button Battery Ingestion Hotline (National Capital Poison Center, Washington, DC) at 202-625-3333 is a 24-hour/7-day-a-week resource for help in management decisions.

FIGURE 77–3.

Algorithm for management of button battery ingestion. *Button batteries in the esophagus must be removed. Endoscopy should be used, if available. The balloon catheter technique can be used if the ingestion occurred ≤2 hours previously, but it should not be used after this period, because it may increase the amount of damage to the weakened esophagus. †When the Foley technique fails or is contraindicated because >2 hours have elapsed, the button battery should be removed endoscopically. This may require transfer of the patient. ‡Acute abdomen, tarry or bloody stools, fever,



Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
www.accessmedicine.com

INGESTION OF SHARP OBJECTS

Sharp objects in the esophagus need immediate removal. Sharp objects may pass into the stomach spontaneously. **Because intestinal perforation from ingested sharp objects that pass distal to the stomach is common, the American Society for Gastrointestinal Endoscopy guidelines recommend removal of sharp objects by endoscopy while they are in the stomach or duodenum.** If intestinal perforation occurs, it is usually at the ileocecal valve.

If the object is distal to the duodenum at presentation and the patient is asymptomatic, the object's passage should be documented with daily plain films. Surgical removal should be considered if 3 days elapse without passage. The development of symptoms or signs of intestinal injury (e.g., pain, emesis, fever, GI bleeding) requires immediate surgical consultation.[13]

NARCOTICS INGESTION

Narcotic couriers (body packers) ingest multiple small packets of a drug in order to conceal transport. A favored packet is the condom, which may hold up to 5 grams of narcotic. These packets are often visible on plain films. Rupture of even one such packet may be fatal, and endoscopy is contraindicated because of the risk of iatrogenic packet rupture. If the packet(s) appears to be passing intact through the intestinal tract, observation until the packet reaches the rectum is the favored treatment. Some authors advocate the use of whole-bowel irrigation to aid the process.

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USEFUL WEB RESOURCES

1a. National Capital Poison Center—http://www.poison.org/

2a. The National Button Battery Ingestion Hotline resource for help in management decisions—
http://www.poison.org/battery/index.asp