Dysphagia may be defined as difficulty in transferring a food bolus from the mouth to the stomach. It may be associated with abnormalities in the oral, the pharyngeal, or the esophageal phase of swallowing. Unlike the term globus, which describes a painless sensation of fullness in the neck or throat, the term dysphagia implies actual interference with the swallowing mechanism [see Sidebar The Swallowing Mechanism]. Dysphagia may be classified as oropharyngeal or esophageal. Whereas oropharyngeal dysphagia (i.e., dysphagia resulting from abnormalities in the oral or the pharyngeal phase of swallowing) usually implies a functional disturbance in the swallowing mechanism, esophageal dysphagia may result from a discrete mechanical obstruction or from an esophageal motility disorder.

The exact prevalence of dysphagia is difficult to determine, but it is estimated that 35% of persons older than 50 years complain of dysphagia at least once a week. It is important to keep in mind, however, that the changes in swallowing physiology associated with aging rarely lead to true dysphagia. Esophageal dysphagia arises primarily from intrinsic diseases of the esophagus; oropharyngeal dysphagia frequently occurs as part of a neurologic, metabolic, myopathic, or infectious syndrome.1

In what follows, we review the diagnostic and therapeutic decision-making approaches employed in assessing patients with dysphagia. Although it is important that surgeons have a working knowledge of all causes of dysphagia, our focus here is on evaluation of conditions that give rise to esophageal dysphagia. We mention surgical management options but do not address them in detail; the operative procedures performed to treat the various clinical entities that cause dysphagia are described more fully elsewhere [see 4:7 Open Esophageal Procedures, 4:8 Minimally Invasive Esophageal Procedures, and 4:10 Video-Assisted Thoracic Surgery].

Clinical Evaluation

Evaluation of a patient with dysphagia must be performed in a systematic manner. Assessment begins with obtaining a detailed history, followed by physical examination. Except in the case of acute caustic ingestion, for which direct flexible esophagoscopy is the first line of assessment, the barium swallow, a readily available and noninvasive test, should be the first investigative tool. The barium swallow is a cost-effective, rapid, and easily available test that provides a “road map” of the esophagus and the lesion and yields a tremendous amount of information before endoscopic assessment. Additional diagnostic information can be obtained by means of fiberoptic esophagoscopy, manometry, 24-hour pH study, and, occasionally, bronchoscopy and endoscopic ultrasonography (EUS). Further diagnostic imaging, in the form of computed tomography and positron emission tomography (PET), is particularly valuable in assessing patients with esophageal cancer.

OROPHARYNGEAL DYSPHAGIA

Oropharyngeal dysphagia is usually associated with symptoms that originate in the oropharynx, including inability to chew food, drooling, coughing during a meal, and nasal regurgitation of solids or liquids. In general, if a patient experiences dysphagia within 1 second of swallowing, an oropharyngeal origin is likely. A variety of different conditions are capable of causing oropharyngeal dysphagia. The common causes can be grouped into three broad categories: (1) generalized (systemic) conditions, (2) intrinsic functional disturbances, and (3) conditions that give rise to fixed mechanical obstruction [see Table 1]. Overall, the most common cause of oropharyngeal dysphagia is a cerebrovascular accident (CVA).

ESOPHAGEAL DYSPHAGIA

Esophageal dysphagia causes symptoms that are referable to the chest or the abdomen. In approximately 75% of cases, the patient’s perception of the location of the obstructive site corresponds to the actual anatomic site of the lesion.2 In addition to dysphagia, patients may experience associated symptoms, such as chest pain (with a character and a radiation pattern resembling those of coronary artery disease [CAD]), retrosternal burning, and regurgitation of undigested food.3

As a general rule, if the swallowing difficulty gradually progresses from solids to liquids, the dysphagia probably has a mechanical cause. Patients who have mechanical obstruction usually complain of dysphagia without pain and can relieve their symptoms only by regurgitating or by altering their diet. If significant weight loss and
Evaluation of Dysphagia

**Dysphagia secondary to systemic condition**
Focus on underlying cause (e.g., scleroderma, diabetes mellitus, alcoholism, amyloidosis, Parkinson disease, Crohn disease, or myxedema).

**Clinical findings and barium swallow are consistent with primary motor disorder**
Assess patient with manometry and endoscopy.

**Patient has not ingested a caustic chemical**
Perform barium swallow.

**Patient has other primary motor disorder (DES, hypertensive LES, nutcracker esophagus)**
Perform laparoscopic esophagomyotomy with modified (i.e., anterior or posterior partial) fundoplication.

**Patient has achalasia**
Perform laparoscopic esophagomyotomy with modified (i.e., anterior or posterior partial) fundoplication.

**Patient has pharyngoesophageal (Zenker's) diverticulum**
If diverticulum is $\geq 2$ cm, treat with cricopharyngeal myotomy and diverticulectomy or, alternatively, with cricopharyngeal myotomy and diverticulopexy.
If diverticulum is $< 2$ cm, treat with cricopharyngeal myotomy alone.

**Patient has pharyngoesophageal web**
Treat with endoscopic dilatation.

**Patient has Barrett's esophagus**
Rule out dysplasia. Perform surveillance endoscopy. Treat GERD symptoms medically or surgically as appropriate.

**Patient has normal or inflamed esophagus**
Focus on underlying cause (e.g., scleroderma, diabetes mellitus, alcoholism, amyloidosis, Parkinson disease, Crohn disease, or myxedema).

Treat medically. In rare circumstances, consider myotomy.
Patient presents with difficulty in swallowing

Obtain complete history.
Perform thorough examination.

Barium swallow reveals esophageal diverticular disease

Treat according to anatomic level of diverticulum.

Barium swallow suggests fixed mechanical obstruction

Assess patient with endoscopy.

Patient has ingested a caustic chemical

Perform direct flexible esophagoscopy.
Ensure adequate airway, breathing, and circulation.
Place patient on NPO regimen.
Give I.V. antibiotics.
Initiate total parenteral nutrition.
Perform barium swallow in 2–3 wk.

Patient has midesophageal diverticulum

Lesions result from periesophageal inflammation and are frequently asymptomatic; dysphagia is rare.
If there are no significant symptoms, they need not be treated, and therapy focuses on underlying inflammatory condition.

Patient has epiphrenic diverticulum

Assess patient with manometry and endoscopy.
If symptoms are absent or mild, manage conservatively.
If significant symptoms are present, manage surgically with myotomy, diverticulectomy, and partial fundoplication.

Patient has peptic stricture

Treat with endoscopic dilatation, and perform brush biopsy to rule out malignancy.
Give PPIs.
Consider manometry and 24-hr pH study.
Consider antireflux surgery.

Patient has Schatzki’s ring

If lesion is asymptomatic, no treatment is required.
If lesion is symptomatic, treat with endoscopic dilatation and medical (or, if necessary, surgical) therapy for GERD.

Patient has esophageal cancer

Perform esophagoscopy for pathologic diagnosis.
Rule out distant metastatic disease with CT or PET. EUS may aid in locoregional staging.
Treat surgically according to stage of disease.
If cancer is localized and patient is medically fit, perform esophagectomy.
anorexia develop and symptoms are progressing rapidly, esophageal cancer is the likely cause of the dysphagia.

If, however, the patient has dysphagia for both liquids and solids, an underlying esophageal motor disorder is probably responsible. Patients who have motor disorders that cause dysphagia often develop certain unusual maneuvers to relieve their difficulty, including repeated swallowing, raising the arms over the head or assuming different positions during swallowing, and the Valsalva maneuver. Such patients also frequently complain of other associated symptoms (e.g., chest pain).

Physical examination is not as helpful as a detailed history. Clues that may help identify malignant conditions include the development of head and neck lymphadenopathy, the presence of an oropharyngeal mass, the appearance of subcutaneous lumps (suggestive of cutaneous metastasis), and the occurrence of clinical features associated with abdominal organ metastasis (jaundice, ascites, hepatomegaly). Muscle weakness, fatigability, and other neurologic deficits detected on physical examination may suggest a CVA or myasthenia gravis as the cause of dysphagia. Like oropharyngeal dysphagia, esophageal dysphagia may be caused by a number of different generalized conditions, intrinsic functional disturbances, or conditions that give rise to fixed mechanical obstruction [see Table 2].4

**Workup and Management of Specific Causes of Dysphagia**

### SECONDARY MOTOR DISORDERS RESULTING FROM SYSTEMIC CONDITIONS

In patients with secondary motility disorders, the esophageal motor disturbance is a manifestation of a systemic condition; thus, organs other than the esophagus are also involved. The classic examples of systemic conditions that give rise to esophageal motility disorders are scleroderma, diabetes mellitus, and alcoholism. Patients who have one or more of these conditions may present with varying degrees of dysphagia, and their evaluation usually entails functional and structural evaluation of the esophagus.

Treatment is aimed at the underlying cause. Patients with scleroderma exhibit atrophy and sclerosis of distal esophageal smooth muscle with fragmentation of connective tissue. Consequently, primary peristalsis is absent, and the lower esophageal sphincter (LES) is hypotensive or virtually absent. Patients usually present with gastroesophageal reflux and heartburn. Because they also lack secondary peristalsis, there is no mechanism for clearing the refluxed acid back to the stomach, and as a result, patients are predisposed to ulcerative esophagitis and peptic stricture [see Disorders Producing Fixed Mechanical Obstruction, Peptic Stricture, below].

Other causes of secondary esophageal motility disturbance resulting in dysphagia are amyloidosis, Parkinson disease, Crohn disease, and myxedema.

### INTRINSIC FUNCTIONAL DISTURBANCES

**Primary Motor Disorders**

**Achalasia** The majority of patients with achalasia present with dysphagia for liquids and solids. Most describe a long-standing history of swallowing difficulty, with or without associated weight loss. Other presenting symptoms include regurgitation, chest pain, heartburn, and coughing or choking spells. Typically, patients will have developed coping mechanisms to deal with the problem (e.g., changing position during eating, drinking liquids to “wash down” the food, and practicing repetitive swallowing and chewing).

A barium x-ray with fluoroscopy will show absent peristalsis and a dilated esophagus. Other findings include a tapered narrowing in the distal esophagus (a so-called bird’s beak [see Figure I]) and, occasionally, an epiphrenic diverticulum [see Esophageal Diverticula, below]. Because the study is dynamic, failure of LES relaxation must be watched for as it is performed. Over time, the esophagus can dilate significantly, to the point where it takes on a sigmoid shape.

Although some radiologists will declare a diagnosis of achalasia solely on the basis of barium x-rays and fluoroscopy, upper GI endoscopy is essential to rule out fixed mechanical obstruction (long-standing achalasia is a risk factor for squamous cell cancer of the esophagus because of chronic stasis and retention esophagitis) or so-called pseudoachalasia (a motility disorder resulting from a carcinoma on the underside of the cardia that extends proximally within the wall of the esophagus). The key diagnostic test in the setting of suspected achalasia is esophageal manometry. Characteristic manometric features of achalasia include aperistalsis and incomplete relaxation of the LES; the LES pressure may be either high or normal.

A detailed discussion of the management of achalasia is beyond the scope of this chapter. Briefly, medical management offers virtually no benefit. Pneumatic dilatation offers subjective improve-

### Table 2 Common Causes of Esophageal Dysphagia

<table>
<thead>
<tr>
<th>Conditions producing fixed mechanical obstruction</th>
<th>Neoplasm</th>
<th>Web</th>
<th>Previous surgical treatment</th>
<th>Previous radiation therapy</th>
</tr>
</thead>
</table>

### Table 1 Common Causes of Oropharyngeal Dysphagia

<table>
<thead>
<tr>
<th>Generalized conditions</th>
<th>CVA</th>
<th>Myasthenia gravis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intrinsic functional disturbances</td>
<td>Cricopharyngeal achalasia</td>
<td>Zenker’s diverticulum</td>
</tr>
<tr>
<td>Conditions producing fixed mechanical obstruction</td>
<td>Neoplasm</td>
<td>Web</td>
</tr>
</tbody>
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Dysphagia — 5

Laparoscopic esophagomyotomy with anterior (Dor) fundoplication (or, as some surgeons prefer, posterior partial fundoplication) probably represents the current standard of care.

Diffuse esophageal spasm

Diffuse esophageal spasm (DES) is a motility disorder of unknown etiology that gives rise to dysphagia and chest pain. The dysphagia is nonprogressive and is encountered with both liquids and solids. The chest pain is nonexertional but may respond to nitroglycerin.

A barium x-ray may show the classic corkscrew appearance [see Figure 2], but this finding is nondiagnostic. The diagnosis is established by manometry; the key finding is the periodic occurrence of simultaneous high-amplitude contractions with intervening periods of normal peristalsis. The presence of these intervals of normal peristalsis is important for distinguishing DES from nutcracker esophagus [see Nutcracker Esophagus, below]. An elaborate workup is often necessary to clarify the basis of the chest pain and rule out the possibility of CAD.

Once CAD has been ruled out, management is primarily medical and consists of reassurance, nitrates, and calcium channel blockers. Botulinum toxin injection and extended esophagomyotomy have been used to treat DES, with some success, but in general, surgery does not have an established role in this setting.

Hypertensive lower esophageal sphincter

Hypertensive LES is a rare motility disorder of unknown etiology. Although it can occur in isolation, it is usually seen in association with achalasia, nutcracker esophagus, or DES. Patients present with dysphagia for liquids and solids. Manometric evaluation shows a mean LES resting pressure higher than 45 mm Hg in midrespiration. Treatment is primarily medical, but balloon dilatation has been employed to relieve persistent dysphagia.

Nutcracker esophagus

Nutcracker esophagus is an esophageal motility disorder of unknown etiology that affects women more often than men. Patients usually present with chest pain but may have associated dysphagia as well. Manometry typically shows peristaltic waves with significantly elevated amplitude (> 180 mm Hg). Barium x-rays are usually normal. Treatment is similar to that of DES and is primarily medical.

Esophageal Diverticula

Esophageal diverticula account for fewer than 5% of all cases of dysphagia. They may be classified into two broad categories: true and false. True diverticula include all layers of the esophageal wall, whereas false diverticula include only the mucosal layer. True diverticula develop as a result of a periesophageal inflammatory process that places traction on the esophageal wall (and thus are also referred to as traction diverticula), whereas false diverticula are manifestations of an underlying motor dysfunction (and thus are also referred to as pulsion diverticula). Esophageal diverticula may also be classified into three categories on the basis of the anatomic level at which they occur: pharyngoesophageal (Zenker’s), midesophageal, and epiphrenic. A few patients will exhibit...
diffuse intramural diverticulosis, a rare condition characterized by the development of multiple 1 to 5 mm outpouchings in association with esophageal inflammation and fibrosis. These outpouchings are believed to be dilated esophageal mucous glands resulting from chronic inflammation. Dysphagia is the most common presenting complaint for this condition, though one third of patients complain of gastroesophageal reflux.

**Pharyngoesophageal diverticula** Zenker’s diverticula are the most common esophageal diverticula. These pulsion diverticula result from pharyngocricopharyngeal incoordination that leads to herniation of the mucosa in Killian’s triangle (the posterior midline of the lower pharynx, between the oblique muscle fibers of the inferior pharyngeal constrictor and the transverse fibers of the cricopharyngeus). Dysphagia is the most common symptom, but patients may also complain of halitosis, regurgitation of undigested food, throat discomfort, a palpable neck mass, a gurgling noise during swallowing, and recurrent aspiration pneumonia.

The best initial diagnostic tool is a barium swallow [see Figure 3], which will establish the diagnosis and also may help diagnose any associated problems (e.g., gastroesophageal reflux disease [GERD] and hiatal hernia). Performing upper GI endoscopy without first obtaining a barium study is a potentially disastrous maneuver: because the endoscope will preferentially enter the pouch rather than the true esophageal lumen, there is a significant risk of inadvertent esophageal perforation. Endoscopy does play a role in assessing the esophageal mucosa, but it is best performed at the time of operation.

The fundamental component of surgical treatment is relief of the functional obstruction at the cricopharyngeus (via cricopharyngeal myotomy) [see 4:7 Open Esophageal Procedures]. Once this is done, diverticula larger than 2 cm should be excised; smaller diverticula can generally be managed with myotomy alone. Diverticulopexy is another option for dealing with the pouch.

**Midesophageal diverticula** Midesophageal diverticula are true (i.e., traction) diverticula that are caused by periesophageal inflammation. The usual cause is granulomatous inflammation of the subcarinal lymph nodes resulting from tuberculosis or fungal infection (typically, histoplasmosis). These diverticula are frequently asymptomatic and are often found incidentally during evaluation for some other disorder. Dysphagia does occur but is a rare symptom; the clinical manifestations are usually related to the underlying inflammatory disease or to associated complications (e.g., infection, bleeding, or fistulization to the airway). If the diverticula are not symptomatic, they need not be treated, and the therapeutic focus is on the underlying problem that prompted the evaluation.

**Epiphrenic diverticula**

Epiphrenic diverticula are acquired pulsion diverticula that arise in the distal 10 cm of the esophagus [see Figure 4]. Although these diverticula are usually associated with other esophageal motor disorders (e.g., achalasia, DES, and hypertensive LES), they occasionally occur in the absence of any underlying esophageal dysfunction. If symptoms are absent or mild, conservative management is appropriate. If significant symptoms (e.g., dysphagia) are present, however, surgical management—usually entailing myotomy, diverticulectomy, and modified fundoplication—is indicated. Before operation, patients should undergo a thorough functional assessment with manometry.

**Figure 3** Shown is a large pharyngoesophageal pouch (Zenker’s diverticulum) in an elderly patient with dysphagia, regurgitation of old retained food, and recurrent pneumonia.

**Figure 4** Shown is a giant epiphrenic diverticulum in an elderly woman with progressive dysphagia and weight loss, in whom cancer was initially suspected.
DISORDERS PRODUCING FIXED MECHANICAL OBSTRUCTION

Esophageal Webs

A web is a localized narrowing of the esophagus caused by intraluminal extension of the mucosa and part of the submucosa of the esophageal wall. Webs may be either congenital or, more commonly, acquired, usually secondary to conditions such as iron deficiency anemia, pemphigoid, and ulcerative colitis (among others). The main presenting symptom is dysphagia, the severity of which is proportional to the degree of obstruction. Treatment usually consists of simple endoscopic dilation [see 5:18 Gastrointestinal Endoscopy] after careful verification of the nature of the lesion.

Peptic Stricture

GERD is a very common problem. The majority of patients will present with heartburn and regurgitation. Although the associated hypomotility observed in some GERD patients can account for some of the dysphagia, it is important to consider (and, if possible, rule out) more significant complications, such as peptic stricture, Barrett’s esophagus, and carcinoma.

Peptic stricture represents the end stage of ulcerative esophagitis, in which the healing of circumferential ulceration results in annular fibrosis. Proton pump inhibitors (PPIs) have proved highly effective in controlling GERD symptoms and enhancing the healing of esophageal ulcers; as a result of their widespread use, peptic stricture is now an infrequent complication. Peptic strictures may occur at any age, and there is usually an antecedent history of GERD. The symptoms are progressive but unlike those of a malignant process, in that the dysphagia usually dates back several years and weight loss is usually absent. Typically, a patient with a peptic stricture describes gradually worsening dysphagia, initially for solids and eventually for liquids as well. If the patient has dysphagia for liquids before solids or has dysphagia for both liquids and solids, an associated motility disorder must be suspected. Strictures induced by reflux are located in the distal esophagus at the squamocolumnar junction [see Figure 5]; the presence of strictures in other parts of the esophagus raises the possibility of causes other than acid-peptic injury. The best initial diagnostic testing approach consists of a barium swallow [see Figure 5] followed by upper GI endoscopy, which shows ulcers and a concentric stenosis (which is usually short).

Management of GERD patients with peptic strictures is controversial. One approach consists of dilatation of the stricture [see 5:18 Gastrointestinal Endoscopy] in conjunction with high-dose PPI therapy. An important component of this approach is the performance of a brush biopsy at the time of dilatation to rule out a malignancy. If the stricture recurs, then dilatation with an antireflux procedure [see 4:7 Open Esophageal Procedures and 4:8 Minimally Invasive Esophageal Procedures] is indicated. In the rare cases in which the stricture cannot be dilated, esophageal resection [see 4:7 Open Esophageal Procedures and 4:10 Video-Assisted Thoracic Surgery] must be considered.

Schatzki’s Ring

Schatzki’s ring is a concentric, symmetrical narrowing at the squamocolumnar junction that arises from the development of submucosal annular fibrosis and is usually accompanied by a small hiatal hernia. The exact cause is unknown, though there is a strong correlation with GERD. Dysphagia is usually for solids and is proportional to the diameter of the ring. Barium swallow establishes the diagnosis [see Figure 6], and esophagoscopy is recommended to confirm it.

For asymptomatic patients, no specific treatment is needed. For patients who present with food impaction, emergency treatment, involving rigid esophagoscopy and removal of the food bolus, is indicated. Definitive treatment entails dilatation of the ring in conjunction with medical therapy for GERD. If the ring proves refractory to this approach, dilatation plus antireflux surgery (fundoplication) [see 4:7 Open Esophageal Procedures and 4:8 Minimally Invasive Esophageal Procedures] may be indicated.

Chemical Ingestion

Alkali are commonly found in household cleaning agents, mostly in the form of sodium or potassium hydroxide (NaOH, KOH). The majority of alkali-related injuries occur accidentally in children; however, such injuries also occasionally occur in adults as part of a suicide attempt. The magnitude and site of the injury are directly relat-
ed to the length of the contact time between the offending substance and the esophageal mucosa. Injury can occur at any level, but the most common site is the distal esophagus; the proximal esophagus, where the transit time is very short, is frequently spared. The inflammation and injury eventually lead to submucosal scar formation, which in turn leads to stricture formation and dysphagia.

A careful endoscopic examination is an essential initial step. The scope should be advanced under direct vision to the proximal injury site; if severe injury is observed, the scope should not be advanced any further. A barium swallow should be done in the first month after injury to detect any stricture that may have formed and to determine its location, severity, and length. Serial barium swallows are helpful in following patients to monitor healing after caustic injury [see Figure 7].

At one time, it was common to administer steroids prophylactically to patients with caustic injuries to the esophagus as a strategy for preventing stricture formation. A 1990 study, however, found that this practice had no beneficial effect on healing and stricture formation rates in children, and thus, steroids currently are not widely used in this setting. Strictures are treated by endoscopic dilatation as necessary [see 5:18 Gastrointestinal Endoscopy]. The ultimate solution for a tight, nondilatable stricture [see Figure 7] is esophageal resection [see 4:7 Open Esophageal Procedures and 4:10 Video-Assisted Thoracic Surgery].

Esophageal Cancer

The incidence of esophageal adenocarcinoma is rising at an alarming rate. Dysphagia is the presenting symptom in more than 90% of esophageal cancer patients. Dysphagia caused by cancer is usually gradual in onset and starts with solids, then progresses to include liquids. Other nonspecific presenting symptoms of esophageal cancer are odynophagia, regurgitation, and pain in the

Figure 6  Shown is Schatzki’s ring in a middle-aged man with severe reflux symptoms and recent-onset dysphagia.

Figure 7  Barium swallow from a 22-year-old patient who ingested toilet cleaner shows long, stringlike lumen from midesophagus to stomach. Dilatation was impossible in this case, and thus, management included esophageal resection with colonic interposition.

Figure 8  Shown is classic appearance of midesophageal squamous cell carcinoma. Mucosal irregularity is apparent within lesion, along with proximal dilatation and shouldering at upper and lower borders. Bronchoscopy confirmed penetration of tumor into airway mucosa.
neck, abdomen, or back. Fistulization of an esophageal tumor into the airway will result in ongoing aspiration (the so-called swallow-cough sequence) and pulmonary sepsis. Weight loss occurring in association with dysphagia is strongly suggestive of esophageal cancer and should prompt appropriate investigation.

A complete history and physical examination should be carried out, followed by a barium swallow. Characteristic features of esophageal cancer on barium x-ray include narrowing, mucosal irregularity, the presence of a mass, and, occasionally, a so-called shouldered stricture [see Figure 8]. Esophagoscopy is essential for establishing a pathologic diagnosis by means of biopsy, brushings, and washings. Treatment is determined by the stage of the disease. In a medically fit patient with localized esophageal cancer, esophagectomy is indicated [see 4.7 Open Esophageal Procedures and 4.10 Video-Assisted Thoracic Surgery]. Traditionally, CT scanning of the neck, the chest, the abdomen, and the pelvis has been performed to rule out distant metastatic disease. More recently, PET scanning has also been used for this purpose. EUS, if available, may also aid in locoregional staging. For lesions in the upper third or the middle third of the esophagus, bronchoscopy is necessary to rule out direct tumor invasion of the airway.

References