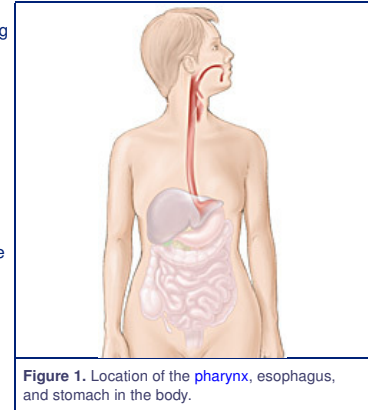


Swallowing Disorders: Introduction

Swallowing is a complex function that affects the physical and mental health of all human beings. Not only does eating provide nutrients, but it also serves as an important role in social interaction.

The mechanism of swallowing is a coordinated operation of the mouth, **pharynx**, and esophagus. Human beings swallow 600 times per day. Under normal circumstances, swallowing is performed without thought or effort. Only the oral phase requires conscious effort. Once the **bolus** passes into the **pharynx**, involuntary reflexes serve to successfully pass it to the stomach.

Dysphagia (from the Greek, "difficulty swallowing") refers to two related, but distinct, clinical problems. On the one hand, it refers to a patient's awareness of impaired transit of swallowed oral contents. On the other, it refers more generally to any swallowing disorder — disorders that are often, but not always, associated with **dysphagia**. Swallowing disorders can present with a variety of symptoms other than **dysphagia**. The most common symptoms are swallow-related coughing and regurgitation of previously swallowed food or liquid (see Symptoms). Disorders of swallowing may result from problems with neural control, muscular coordination, inflammation, or neoplasia.



Clinical Specialties

A large (and somewhat bewildering) number of clinical specialties are involved in the evaluation and management of swallowing disorders.

Dentistry	Neurosurgery	Neurology
Otolaryngology	Nursing	Pulmonary Medicine
Gastroenterology	Radiology	Oral Surgery
Pediatrics	Rehabilitation	Speech-Language Pathology
General Surgery	Nutrition	Oncology
Physical Therapy	Rheumatology	
Thoracic Surgery	Occupational Therapy	

Table 1. Clinical Specialties

Each of these specialties has its own unique perspective on swallowing disorders. However, there is considerable overlap in the types of disorders each specialty treats. As a result, it is often difficult to determine which specialty should be involved in the care of a particular patient. In more difficult or complicated situations, a multidisciplinary "swallowing center" is often best equipped to coordinate a patient's evaluation and treatment.

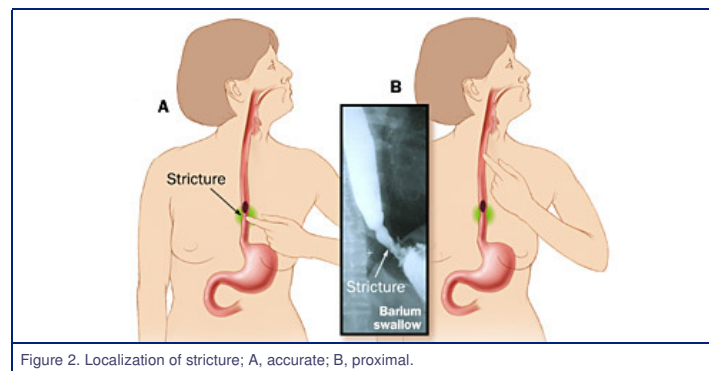
Symptoms

Swallowing disorders result in a variety of symptoms, which may occur in different combinations. In many cases, the temporal association of symptoms with swallowing, or with meals, is obvious. In other cases, the association between swallowing and symptoms may not be clear.

Dysphagia

Dysphagia refers to a patient's perception of difficulty in the passage of a swallowed bolus from mouth to stomach. Patients typically describe this as a sensation of food "sticking" in the throat or chest. They may also use the term "choking" (see below) to describe the same feeling.

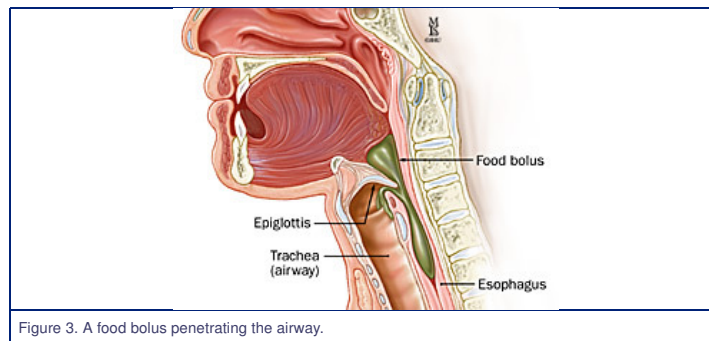
As previously mentioned, the term dysphagia can also be used to describe any swallowing disorder, whether the symptom of dysphagia is present or not. When used in this manner, the word refers to an entire category of disorders, rather than a single symptom. Although it is commonly assumed that patients can accurately localize dysphagia to the level of actual obstruction, this is not always the case (Figure 2). About one-third of patients identify a site well above the level of obstruction documented by radiographic studies. Distal localization (localization well below the level of obstruction), however, is rare.



Coughing

Coughing is a nonspecific response to a variety of stimuli usually originating in the pharynx, larynx, or lungs. When coughing occurs during, or immediately after swallowing, the symptom strongly suggests a swallowing problem.

However, because humans swallow throughout the day, patients may not recognize the temporal relationship of coughing to swallowing. Also clouding this association is the fact that coughing may be due to premature leaking of oral contents into the pharynx, incomplete clearance of the bolus from the pharynx, or regurgitation of esophageal contents back to the pharynx (Figure 3). The term "choking" is frequently used to refer to coughing, or to a sensation of food sticking, rather than to coughing.

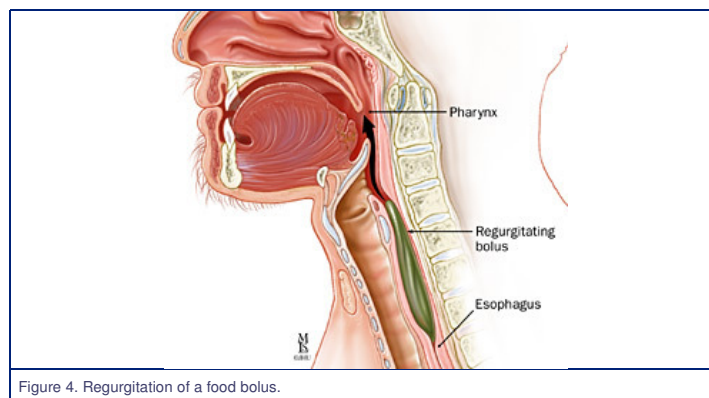


Choking

As mentioned above (see Dysphagia and Coughing), "choking" is a term frequently used by patients (and occasionally by physicians) to describe the feeling of food sticking in the esophagus or coughing. Although both symptoms may occur in patients with swallowing disorders, they imply somewhat different mechanisms of dysfunction. In analyzing symptoms, it is therefore important to determine exactly what the patient means by the term.

Regurgitation

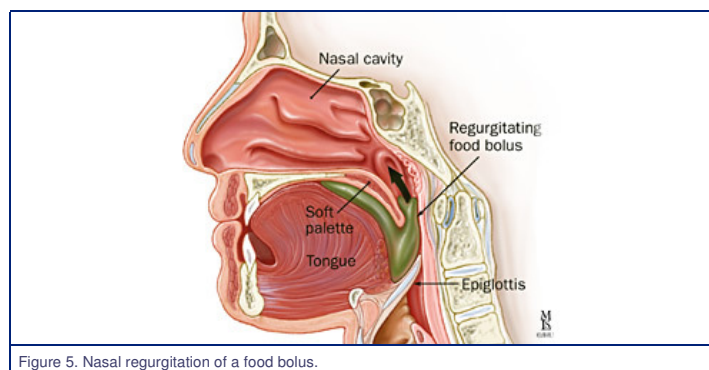
The swallowing mechanism is designed to assure unidirectional movement of the swallowed bolus. "Regurgitation" refers to the return of food or liquid back to the mouth or pharynx, after it has apparently successfully passed out of either region (Figure 4).



With regurgitation, material returns effortlessly to the mouth or throat. This contrasts with vomiting, during which nausea and retching are frequently present and contraction of the abdominal muscles and diaphragm play an important role. When patients suggest that the regurgitated material tastes like ingested food, a swallowing disorder is usually present. Regurgitation of sour or bitter-tasting food or liquid suggests that at least some of the regurgitated material had reached the stomach. When sour or bitter regurgitation is present, the problem may fall within the spectrum of gastroesophageal reflux disease (GERD), rather than a swallowing disorder.

Nasal Regurgitation

The nasopharynx closes through a combination of soft palate elevation and contraction of the upper pharyngeal constrictor muscles (the superior pharyngeal constrictors). Failure of this closure mechanism, pharyngeal retention, or esophagopharyngeal regurgitation can result in nasal regurgitation (Figure 5).



Other Symptoms

Depending on the type of swallowing disorder, patients may present with a sore throat, hoarseness, shortness of breath, and chest discomfort or pain. The relationship between swallowing and these symptoms may not be obvious. All of these symptoms may arise from a variety of other sources and none are specific to swallowing disorders.

Swallowing Disorders: Anatomy

Physiology of Swallowing

Normal swallowing requires the coordinated activity of the oral cavity, pharynx, and esophagus. A properly functioning swallowing mechanism provides efficient, unidirectional flow of the ingested bolus, while avoiding undesired diversion into the nasal cavity or respiratory tree.

Between swallows, the pharynx and esophagus are at rest. The nasal cavity and larynx are in open communication with the pharynx permitting the individual to breathe freely. The entrance to the esophagus remains closed by the upper esophageal sphincter (UES). This muscle serves to prevent the esophagus from filling with air during inspiration. At the lower end of the esophagus, the lower esophageal sphincter (LES) separates the esophagus from the stomach. The LES prevents reflux of irritating gastric contents into the esophagus.

The mouth prepares the bolus for swallowing. The lips prevent drooling. The tongue positions all or part of the oral contents on its upper surface. The back of the tongue, palatine arch, and soft palate prevent premature spillage of the bolus into the pharynx. At the beginning of a swallow, as the tongue forces the bolus up against the hard palate and then back into the pharynx, the soft palate elevates and the posterior wall of the nasopharynx contracts to prevent nasal regurgitation.

Penetration of the airway is prevented through a combination of events: inhibition of respiration, elevation of the larynx, approximation of vocal cords, and deflection of the epiglottis over the larynx. The presence of multiple mechanisms to prevent airway blockage suggests the importance of this function to the organism's well being. The failure of any one mechanism, such as the surgical removal of the epiglottis (epiglottectomy), usually does not result in aspiration of the swallowed bolus.

Propulsion of the swallowed bolus is accomplished through a combination of a piston-like compression of the back of the tongue against the posterior pharyngeal wall, and a sequential wave of pharyngeal muscles (pharyngeal peristalsis) (Figures 6 and 7).

As the swallowed bolus enters the pharynx, inhibition of neural stimulation results in the relaxation of the UES. This permits the swallowed bolus to enter the esophagus unimpeded. The peristaltic wave continues the length of the esophagus (esophageal peristalsis). Before the bolus arrives in the distal esophagus, cessation of firing of excitatory nerves and activation of inhibitory nerves relax the LES. This permits easy passage of the bolus into the stomach.

The initiation of swallowing by the oral cavity is under voluntary control, whereas control of the pharynx and esophagus are involuntary. This means that once the initial signal is received from the brain, the pharyngeal and esophageal phases of swallowing are carried out automatically. Initiation of swallowing is directed by the brainstem, which integrates sensory information from the swallowing channel with information from the other areas of the brain. Integration signals are then sent back to the swallowing channel to initiate the act of swallowing (Figure 8).

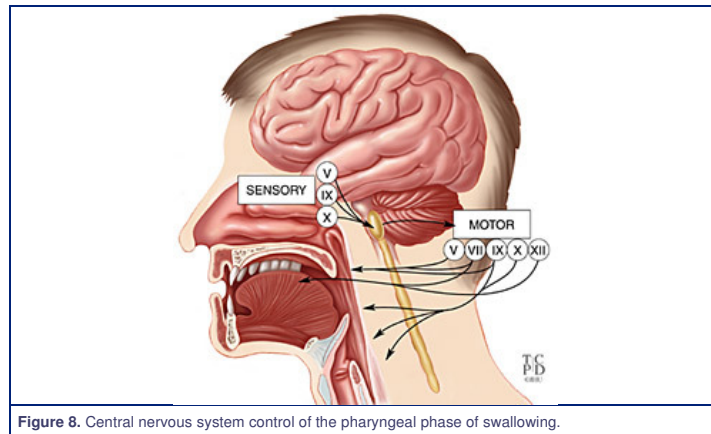


Figure 8. Central nervous system control of the pharyngeal phase of swallowing.

Once initiated, the esophageal phase of swallowing can continue without central nervous system involvement, with the brain serving to modify esophageal function.

Swallowing Disorders: Causes

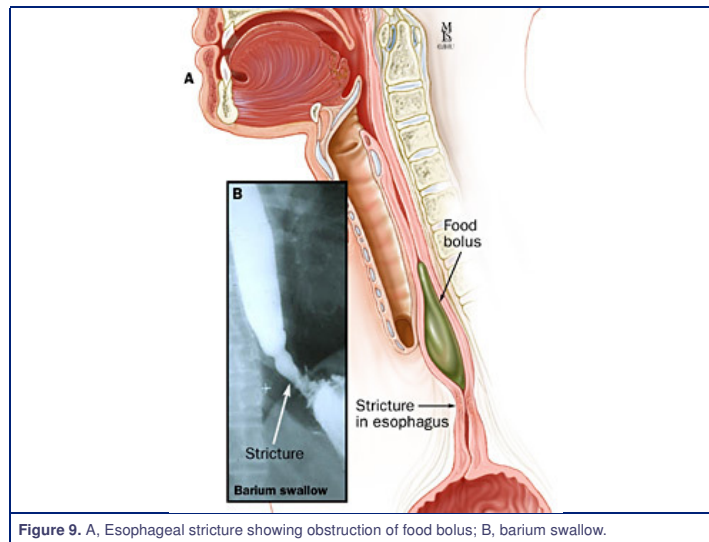
Overview

The mechanism of swallowing disorders may be either structural or motor. Structural disorders include luminal stenosis and diverticula. Motor disorders include paresis (muscle weakness), sphincteric dysfunction, and spastic disorders.

Structural Disorders

Luminal Stenosis

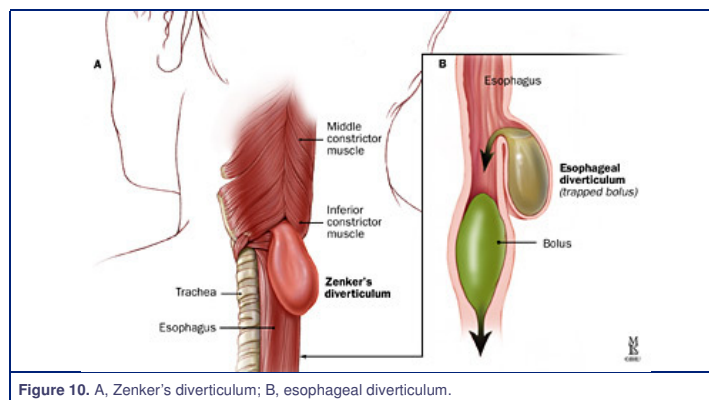
Luminal stenosis occurs as a result of mechanical narrowing of the esophageal lumen in patients with esophageal strictures (Figure 9). Symptoms arise when the swallowed food is too large to pass. The typical symptom in a patient with an esophageal stricture is dysphagia for solid food, often followed by regurgitation of undigested material.



In general, patients can swallow with little difficulty. This remains true until the swallowing channel is narrowed by about 50%. Even beyond this point, frequency and severity of symptoms may vary substantially, depending on the degree of narrowing, the presence of any associated motor dysfunction, and the choice and preparation of food.

Diverticulum

A diverticulum is a pouch extending out from the normal wall of the swallowing channel. Diverticula (the plural of diverticulum) can develop in either the pharynx or esophagus (Figure 10). Although small diverticula may not cause symptoms, larger diverticula can cause dysphagia for liquids and solids. Regurgitation of undigested food, often hours after ingestion is a characteristic symptom of patients with diverticula.



Motor Disorders

Paresis

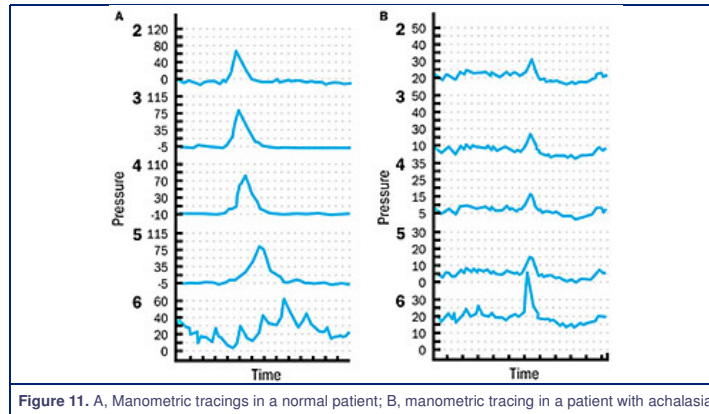
Pharyngeal weakness is commonly associated with neurological conditions, such as a cerebral vascular accident, amyotrophic lateral sclerosis (ALS), head and neck trauma, or brain surgery. Severe esophageal weakness is relatively rare. When it does exist, it is characteristically found in patients with certain types of collagen vascular disorders, especially scleroderma and mixed connective tissue disease. It is also found in patients with certain neurological and muscle conditions that can affect the function of the smooth muscle of the GI tract.

This condition may be asymptomatic. However, when the patient is symptomatic, dysphagia with liquids and solids usually occurs, often associated with regurgitation. Coughing during swallowing is common with pharyngeal weakness, because of aspiration due to pharyngeal retention or impaired laryngeal closure. Mild degrees of weakness are not uncommon in the elderly, in patients with neurological conditions, and in patients with gastroesophageal reflux disease.

Sphincteric Dysfunction

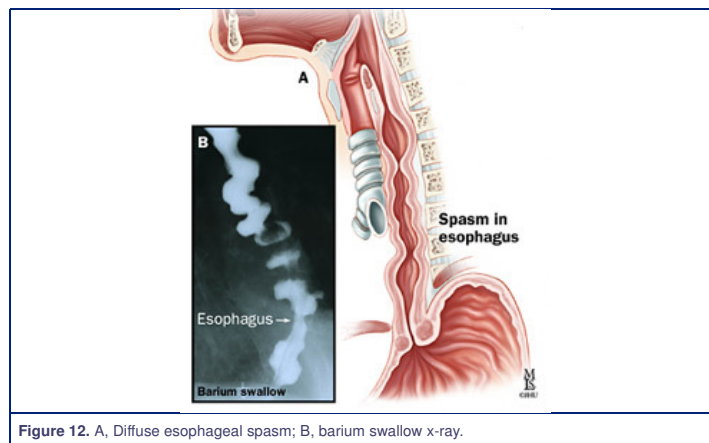
The upper esophageal sphincter (UES) and lower esophageal sphincter (LES) assure unidirectional flow of the swallowed bolus. However, they must relax during swallowing so as not to pose a barrier to flow. The best-described disorder of sphincteric function is achalasia. This is a condition in which the failure of LES

relaxation, associated with severe esophageal weakness, combines to retain food in the esophagus. Patients with sphincteric dysfunction typically complain of dysphagia for liquids and solids (Figure 11).

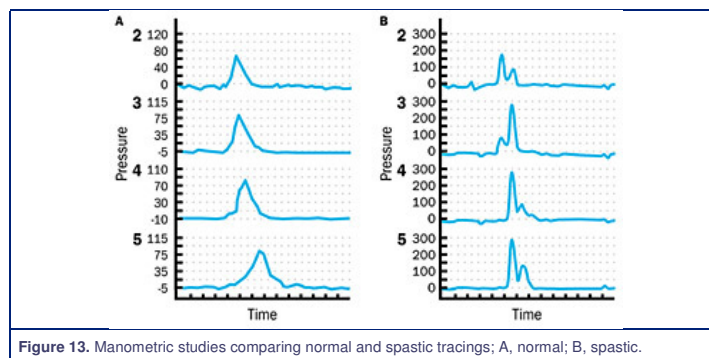


Spastic Disorders

Whereas spastic disorders of the pharynx are rare, those of the esophagus are common. A certain amount of motor dysfunction may be seen in normal individuals. Esophageal dysmotility may range from mild, infrequent events that are within normal limits, to profound incoordination associated with every swallow.



Diffuse esophageal spasm (Figure 12) represents one extreme of esophageal dysmotility. However, the term is often used loosely to refer to any degree of dysmotility noted by barium or manometric studies (Figure 13).



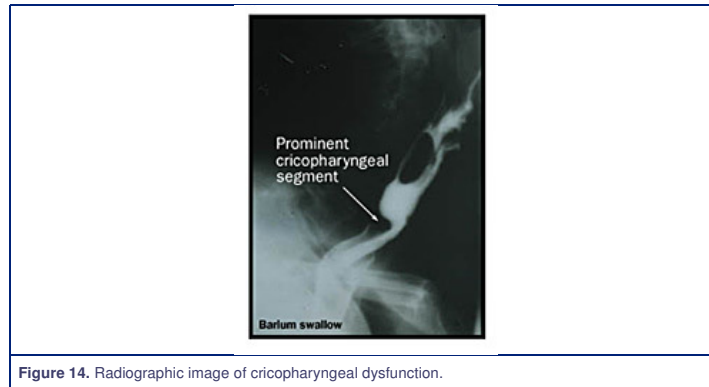
Neurogenic Dysphagia

Neurogenic dysphagia is a general term used to describe any neurological or muscular condition that affects oral or pharyngeal motor function. Common causes of neurogenic dysphagia include stroke (cerebral vascular accidents), amyotrophic lateral sclerosis (ALS), brain injury due to trauma or previous surgery, and local soft tissue and nerve damage due to head and neck surgery. However, many disorders of the nervous system and muscles can cause neurogenic dysphagia. In some patients, neurogenic-type radiographic abnormalities occur even when no diagnosable neurological or muscular disorder is present.

Symptoms of neurogenic dysphagia generally include dysphagia and swallow-induced coughing. Nasal regurgitation, if present, strongly suggests a neurogenic process. Some patients fail to recognize the presence or severity of aspiration, possibly because of associated cognitive problems or impaired sensation. Patients may experience recurrent bouts of pneumonia due to swallowed saliva, liquid, and food entering the airway. The prognosis depends on the severity of the swallowing impairment and the treatability of the underlying condition. Among the more treatable causes of neurogenic dysphagia are myasthenia gravis, polymyositis, and both hyper- and hypothyroidism.

Isolated UES Dysfunction

Upper esophageal sphincter (UES) dysfunction refers to an impaired opening of the cricopharyngeal segment, which is also called the pharyngoesophageal segment (PE). This is a 1–2 cm region where the UES is located. Incomplete opening of the cricopharyngeal segment is a common radiographic finding on videoradiography (Figure 14).

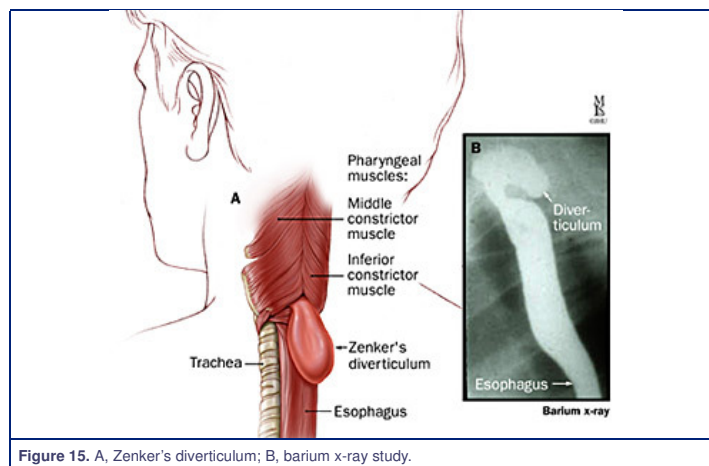


Because the UES protects against esophagopharyngeal regurgitation, an incomplete opening may be a sign of the body's attempt to prevent regurgitation in the face of downstream obstruction due to esophageal disorders. Alternatively, impairment of the PE opening may reflect inadequate force of bolus propulsion due to pharyngeal weakness in neurogenic dysphagia. Therefore, radiographic evidence of a "tight" PE, may point to pharyngeal or esophageal dysfunction. UES dysfunction must be interpreted with caution.

Isolated UES dysfunction (referred to as cricopharyngeal spasm or cricopharyngeal achalasia) refers to failure of the PE opening without evidence of pharyngeal paresis or evidence of esophageal disease. In patients with this condition, dysphagia is caused by obstruction at the level of the PE, due to either fibrosis (scarring) of the PE or UES dysfunction. Failure of UES relaxation may be documented by manometry.

Zenker's and Esophageal Diverticulum

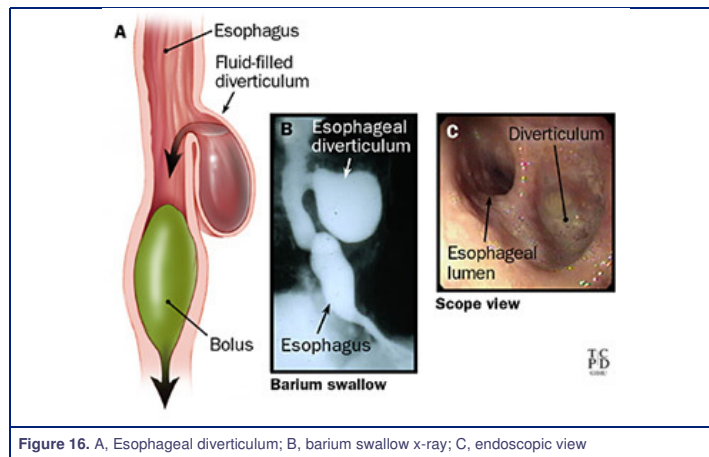
A diverticulum is a sac-like protrusion found in the normal swallowing channel. Diverticula may result from inflammatory or neoplastic processes outside the channel. As these processes evolve, they pull on the adherent wall of the channel, forming the diverticulum. This type of diverticulum is referred to as a traction diverticulum. Traction diverticula most often occur in the midesophagus as a result of inflammation in the mediastinum. Today, most diverticula are due to a different pathogenesis. They appear to result from gradual weakening of the wall of the swallowing channel as a result of abnormally increased intraluminal pressure (pulsion diverticula).



Zenker's (hypopharyngeal) diverticulum is an abnormal, sac-like protrusion on the wall of the lower pharynx (the hypopharynx) (Figure 15). It develops along the posterior pharyngeal wall, just above the upper esophageal sphincter. This area of the pharynx is a region of relative weakness because of the absence of any covering muscle layer. However, this area of weakness is a normal feature of human anatomy and does not in and of itself account for the development of the diverticulum. The primary cause of a Zenker's diverticulum appears to be abnormal relaxation of the upper esophageal sphincter. As a result, the pharyngeal pressure generated during swallowing is increased. Over time, the pharyngeal wall begins to bulge with each swallow until a diverticulum develops and persists between swallows.

Pulsion diverticula may be asymptomatic when small. When they grow, however, they retain liquid, food, and regurgitated matter. A patient may wake up coughing, only to find food, recognizable as portions of the meal eaten hours before, in the mouth or on the pillow.

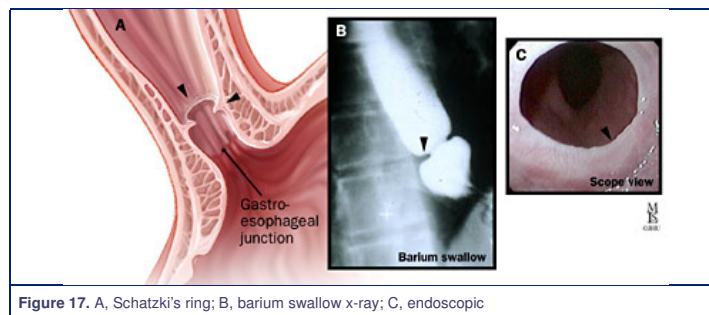
Diverticula may occur in the esophagus as well as the pharynx (Figure 16). Most esophageal diverticula result from abnormal intraluminal pressure as a consequence of increased downstream resistance. This may be due to a stricture, esophageal spasm, or abnormal lower esophageal sphincter function.



Esophageal Stenosis

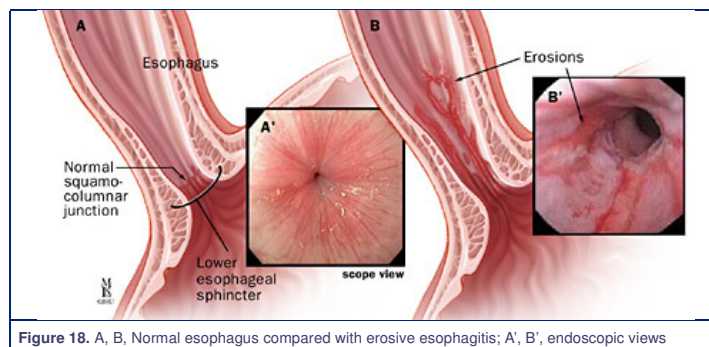
A stenosis refers to any point of narrowing of a channel that results from a structural abnormality, as opposed to a motor disorder. Although the term stricture is often used to describe the same thing, stricture more specifically refers to a narrowing that results from a thickening of the wall of the swallowing channel. Rings, webs, inflammatory strictures, or tumors may also narrow the lumen. Stenosis tends to cause more difficulty in swallowing solids than liquids, but dysphagia for solids only is strongly suggestive of a stenosis. The major difference among the different types of stenotic lesions is the rate of progression of symptoms.

Esophageal rings or webs refer to a short, ring-like band protruding into the lumen. These rings or webs are typically composed of a fold of the lining of the esophagus without any muscle tissue. They may be congenital or acquired, and may occur anywhere in the pharynx or esophagus. The most common is the Schatzki's ring (Figure 17), which by definition occurs at the junction of the esophagus and stomach. Named for the radiologist who described it, Schatzki's ring is very common and only causes symptoms when it narrows the channel by about 50%. A ring-like band located elsewhere in the pharynx or esophagus is often called a web; the distinction is relatively arbitrary. Webs and rings typically cause intermittent dysphagia for solids, which may remain stable or progress slowly over time.



Typically the patient describes intermittent dysphagia of sudden onset, separated by symptom-free periods (without swallowing difficulty). Symptoms are often greatest with tough and difficult-to-chew foods. Patients may describe particular problems with foods generally considered to be soft, such as pasta or bread. Symptoms are more likely to occur when the patient is eating hurriedly, is offered a limited choice of foods (e.g., when dining out), or is distracted by conversation.

Gastroesophageal reflux disease (GERD) is the most common cause of inflammatory strictures. Inflammation resulting from prolonged contact with irritating gastric contents causes scarring. Thickening of the esophageal wall narrows the channel, producing the dysphagia for solids characteristic of an esophageal stenosis. Esophageal strictures tend to be longer than rings or webs (Figure 18). Unless the cause of inflammation has been removed, the surface of the stricture may appear inflamed and even ulcerated in some cases.



Unlike webs and rings, dysphagia from an inflammatory stricture tends to grow steadily worse over time. The intervals between symptomatic episodes become shorter and the variety of foods that cause symptoms increases over a period that can often be measured in months rather than years. Symptoms may improve when the cause of inflammation is removed (e.g., by effective treatment of reflux disease) and as the swelling associated with inflammation decreases. However, even with effective treatment, symptoms may continue to progress as the scarring increases. With complete eradication of inflammation, scarring gradually stops and the size of the channel stabilizes.

A final cause of esophageal stricture is infiltration of the esophageal wall by a tumor. A tumor may also grow into the channel, further narrowing the lumen. Compared with an inflammatory stricture, malignant strictures tend to progress rapidly, often causing dysphagia for liquids as well as solids within a few months of onset of

symptoms (Figure 19). Rapidly progressive dysphagia for solids is a cause of concern and requires early evaluation.

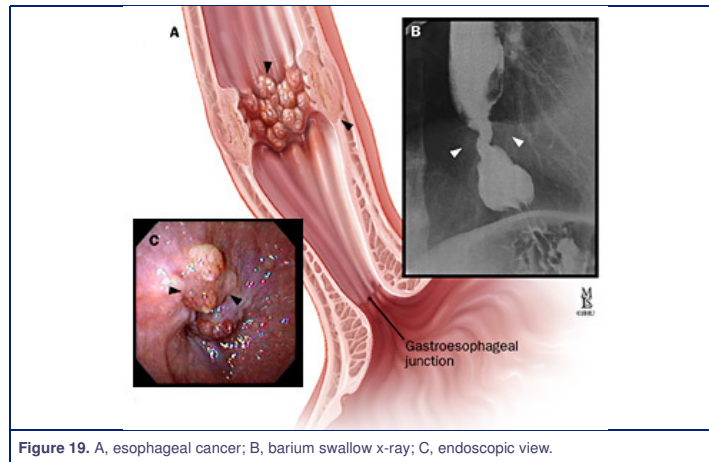


Figure 19. A, esophageal cancer; B, barium swallow x-ray; C, endoscopic view.

Diffuse Esophageal Spasm

Esophageal dysmotility refers to any abnormality in the coordination or strength of esophageal contractions. Diffuse esophageal spasm (Figure 20) refers to severe degrees of esophageal dysmotility that may produce symptoms of dysphagia and chest pain.

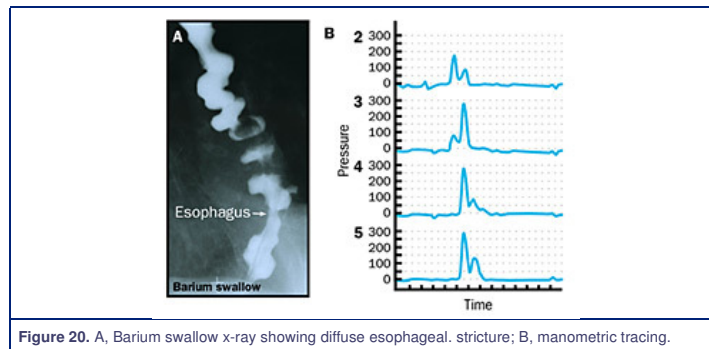


Figure 20. A, Barium swallow x-ray showing diffuse esophageal stricture; B, manometric tracing.

Dysphagia typically occurs with both liquids and solids. Chest pain — which may simulate cardiac pain — may be swallow-induced, but may also be unrelated to swallowing. Even when severe, esophageal dysmotility, does not always produce symptoms.

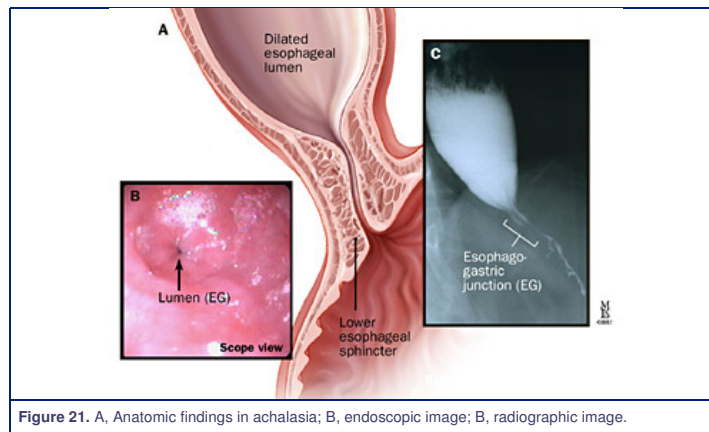
Esophageal spasm is often diagnosed on the basis of clinical symptoms alone. Documentation of abnormal motility may be difficult to obtain because esophageal spasm can be intermittent and therefore may not be detected on short duration studies such as barium x-ray or esophageal manometry. Some degree of abnormal motility occurs in normal asymptomatic volunteers, and the line between esophageal spasm and abnormal contractions that can occur in normal individuals is poorly defined.

Esophageal dysmotility (including spasm) may result from a variety of causes including downstream obstruction, esophageal irritation, or neurological diseases affecting the autonomic nervous system, which controls much of the gastrointestinal function. When no cause has been determined, the condition is referred to as idiopathic esophageal spasm, but this is a relatively rare phenomenon. The most common cause of esophageal spasm is gastroesophageal reflux disease (GERD).

It is important to look for reflux and a stenotic lesion in anyone with symptoms suggestive of esophageal spasm because the treatment of these secondary spastic conditions is different from that of idiopathic spasm. Benign esophageal stenosis is usually treated by esophageal dilation. Reflux is treated with dietary measures designed to avoid stimulation of gastric acid and to decrease the volume of gastroesophageal regurgitation. These measures are usually combined with drugs that decrease acid secretion and improve gastric emptying. Idiopathic spasm, however, may be treated with drugs (smooth muscle relaxants) that decrease the strength of esophageal contractions. Unfortunately, smooth muscle relaxants are often ineffective for the treatment of esophageal spasm.

Achalasia

Achalasia is a motor disorder of the esophagus without an obvious etiology. In achalasia, nerve cells (myenteric plexus) located between the esophageal muscle layers are damaged (degenerate). The result is a complete loss of coordinated esophageal contractions (peristalsis) and failure of lower esophageal sphincter (LES) relaxation (Figure 21). This combination produces obstruction at the esophagogastric junction and loss of effective propulsion.



The mean age for the onset of achalasia ranges between 30–60 years, with a peak in the 40s. The prevalence appears to be about 8–12 per 100,000 population. The disorder is more prevalent in men than women with a ratio of about 2:1.

The initial clinical presentation of achalasia is characterized by solid food dysphagia. Patients complain of fullness in the chest while eating, with a filling or overflow sensation as the meal progresses. Food is unable to pass effectively into the stomach and piles up above the LES, resulting in the gradual dilation of the esophagus. This causes dysphagia for liquids and solids and regurgitation of recognizable food, often many hours after ingestion. This form of regurgitation is almost always found in patients with hypopharyngeal or esophageal diverticulum or achalasia. Patients may also complain of substernal chest pain and weight loss.

In most instances, the esophagus becomes dilated, and sometimes tortuous probably as a result of neuropathic changes in the esophagus and the pressure exerted on the esophageal wall by retained food. endoscopic ultrasound (EUS) studies reveal a thickened lower esophageal sphincter (31 mm as compared 22 mm) and thickened muscularis propria.

Swallowing Disorders: Diagnosis

Objective Tests

The clinical examination, particularly the patient's description of the nature and history of their symptoms, should always be the initial step in the evaluation of swallowing disorders. The patient's self-report often suggests both the type of disorder most likely responsible for the complaints and the objective tests required to determine, or verify, the specific cause. Minor abnormalities of the swallowing mechanism are frequent, especially in older patients. They must not be over-interpreted. The clinical examination offers a check against objective findings and allows for comparison and interpretation.

The following objective tests are commonly used in the evaluation of patients with symptoms of swallowing disorders: Barium Radiography, Modified Barium Swallow, Radionuclide Clearance Studies, Pharyngoscopy, Esophagoscopy, Manometric Studies, and Continuous (24 Hour) pH Monitor.

Barium Radiography

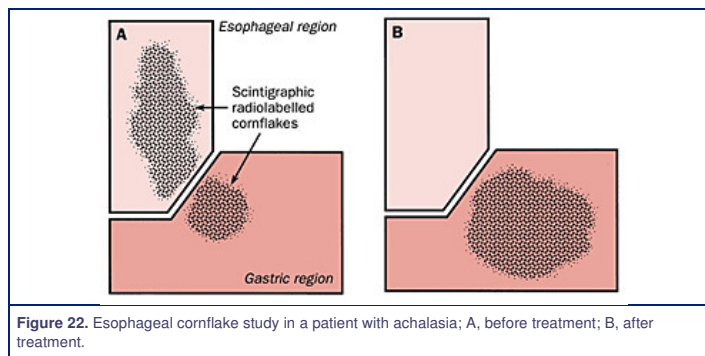
Except when re-evaluating a patient with an established diagnosis, barium x-ray studies are usually the first step in the evaluation of swallowing disorders. These studies permit evaluation of the entire swallowing channel (mouth, pharynx, and esophagus). Even when the location of the abnormality is suggested by the clinical history, barium radiography allows assessment of both the structure and function of the swallowing mechanism. When symptoms suggest an esophageal condition, a standard barium swallow (often performed as part of an upper GI [UGI] series) is generally adequate. However, the flow of barium through the pharynx is too rapid for the radiologist to record important details of swallowing. Therefore, when a pharyngeal condition is suspected, a video-barium study (videopharyngoesophagram, during which the flow of barium through the pharynx and esophagus is recorded on a videotape recorder) is essential

Modified Barium Swallow

A modified barium swallow is a variant of the video-barium study in which the effect of various maneuvers on the efficiency of swallowing is evaluated. Maneuvers may include modifications in the type of swallowed bolus, in head or body position, and in the timing of swallowing in relationship to respiration. Primarily designed to evaluate the effect of therapeutic maneuvers on patients with established neurological or post surgical disorders of oral and pharyngeal function, the modified barium swallow is sometimes used as the first test in patients in whom these disorders are strongly suspected. However, those who specialize in this procedure (most often speech language pathologists with special training in the field of swallowing) may have limited familiarity with structural disorders of the pharynx and even less with radiological abnormalities of the esophagus. In general, the modified barium swallow should be used only after a detailed diagnostic video-barium study has been performed under the supervision of an experienced radiologist, or as a combined study in which the swallowing therapist and radiologist collaborate.

Radionuclide Clearance Studies

During esophageal clearance studies a small amount of radioactive material is mixed with liquid or solids. These studies provide important information about the physiology of swallowing and reflux disease; however, their use is limited in clinical practice. The major clinical application of radionuclide clearance studies is for achalasia, in which esophageal weakness combines with failure of the lower esophageal sphincter relaxation, causing retention of food in the esophagus. The esophageal clearance study is used to quantify the severity of retention and to determine the extent of improvement after treatment (Figure 22).



Pharyngoscopy

Pharyngoscopy is a standard component of the otolaryngologist's (head and neck physician) swallowing evaluation and is usually performed without sedation during a clinic visit. Pharyngoscopy involves the visual examination of pharynx and larynx (but not the esophagus), using either a specially designed mirror (indirect pharyngoscopy) or a flexible telescope (flexible pharyngoscopy). Pharyngoscopy can detect inflammation, tumors, or abnormal function of the pharynx or larynx (Figure 23).

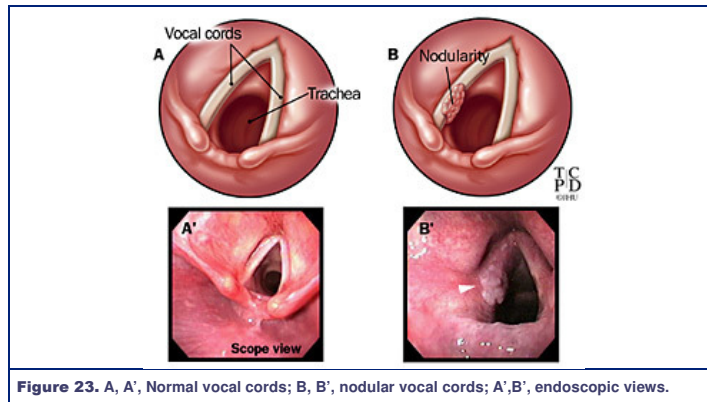


Figure 23. A, A', Normal vocal cords; B, B', nodular vocal cords; A',B', endoscopic views.

Esophagoscopy

Esophagoscopy involves visual examination of the esophagus, using either a rigid or flexible endoscope. Because rigid esophagoscopy requires anesthesia and involves more discomfort and risk, flexible endoscopes are generally used for swallowing evaluations except in specific instances. Flexible endoscopes allow examination of the stomach and first part of the small intestines (duodenum) as well as the esophagus. Endoscopy is the best test for the evaluation of esophageal mucosal abnormalities, such as esophagitis (Figure 24). It is also a good way to evaluate conditions that narrow the esophagus (esophageal strictures), although mild to moderate strictures may be missed. Endoscopy is a poor means of evaluating esophageal function, but it does play a supplemental role in assessing specific conditions that may cause such motor abnormalities.

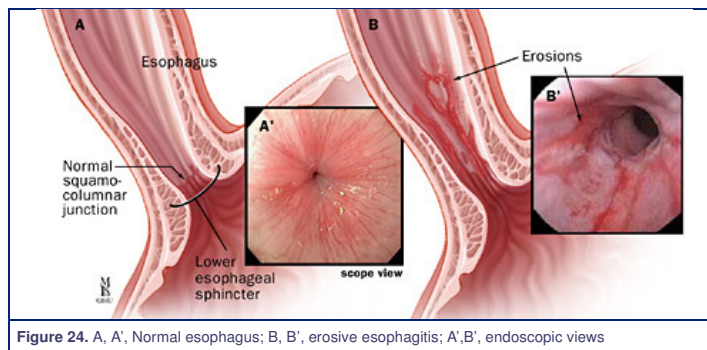


Figure 24. A, A', Normal esophagus; B, B', erosive esophagitis; A',B', endoscopic views

Endoscopy is the primary means of obtaining tissue for microscopic analysis. Although the endoscopist may examine the pharynx during intubation, endoscopes used for upper endoscopy are somewhat thicker and less flexible than those developed for the pharynx. Additionally, gastroenterologists perform most upper endoscopies; generally their training and experience with pharyngeal disease is limited. Therefore, upper endoscopy does not substitute for pharyngoscopy when the latter is indicated.

Manometric Studies

Manometric studies evaluate pressure changes that occur during swallowing. The test involves use of a thin, flexible catheter that is passed through the nose or mouth into the esophagus. The tube has a series of pressure sensors attached to, or imbedded into, specific locations throughout. These pressure sensors permit assessment of the strength and coordination of the pharyngeal and esophageal peristalsis wave and the strength and relaxation function of the upper and lower esophageal sphincters (Figure 25).

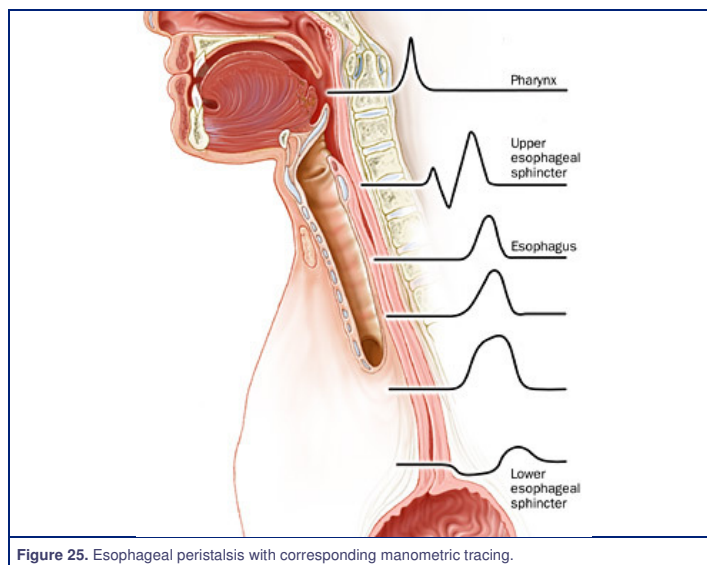
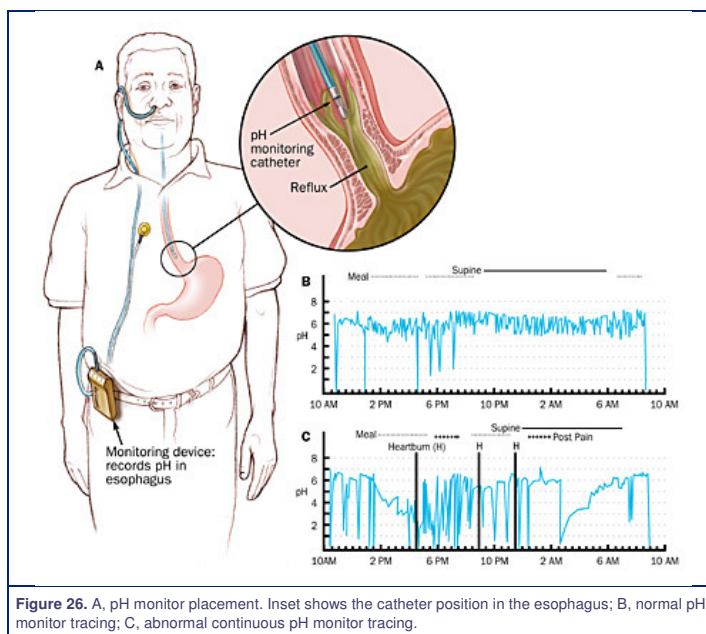


Figure 25. Esophageal peristalsis with corresponding manometric tracing.

The test has been used extensively for the evaluation of esophageal motor disorders, particularly when barium studies are unremarkable or when x-ray findings raise questions about motor dysfunction. Manometry is also used to properly position pH probes for the evaluation of gastroesophageal reflux disease (see below). Finally, it is used preoperatively in patients for whom antireflux surgery is planned to help determine the appropriateness and approach to surgery. A special variant of the test, in which the pressure catheter is attached to a small computer or to record data, permits continuous recording of esophageal pressure for an extended period (up to 24 hours). This is occasionally used for patients with suspected esophageal spasm in whom standard manometric studies have proven unrevealing. Pharyngeal manometry can be used for the evaluation of pharyngeal and UES function. This requires special equipment because the speed of pharyngeal pressure changes during swallowing exceeds the capacity of the usual equipment used for esophageal manometry. The equipment used for pharyngeal manometry is not available in all esophageal manometry laboratories. The role of manometry in the evaluation of pharyngeal dysfunction is not as well established as that for esophageal motor disorders.

Continuous (24 Hour) pH Monitor

pH monitoring permits evaluation of gastroesophageal reflux disease (GERD). pH monitoring involves the placement of a thin, flexible catheter with an acid-sensitive tip through the nose into the esophagus (Figure 26A). A short version of this test (20–30 minutes) has been available for decades. The procedure was later adapted for the evaluation of reflux prolonging the test over extended periods of time—up to 24 hours (24-hour, or continuous pH monitor study) (Figure 26 B,C). Continuous pH monitoring has played a critical role in advancing our understanding of GERD. It is considered the best test for the evaluation of GERD and permits determination of the presence and severity of reflux.



Continuous (24-hour) pH monitoring has demonstrated that most normal individuals (nonrefluxers) actually reflux on a daily basis. Therefore, gastroesophageal reflux disease implies not just the presence of reflux, but reflux in excess of that experienced by nonrefluxers.

Swallowing Disorders: Therapy

Overview

The treatment of patients with swallowing disorders should be individualized. The symptoms along with the physical and emotional impact upon quality of life should be assessed and considered in the development of a treatment strategy. To date, relatively small numbers of well-controlled clinical trials have been conducted; therefore, experience, rather than analysis of data, is often the basis for treatment.

Reassurance / Psychological Interventions

Although swallowing disorders are almost always caused by structural abnormalities or motor dysfunction, the severity of symptoms occasionally appear to exceed what would be expected. Swallowing disorders are rarely a result of psychological factors alone; anxiety may play a role in exaggerating the severity of symptoms, especially if the patient is concerned about the possibility of cancer or heart disease (if chest pain is a prominent associated symptom). Clinical reassurance alone is generally not effective and is often ill advised if it takes the place of an appropriate evaluation. However, after the evaluation is performed, symptom severity may improve with reassurance. Formal psychological intervention, including counseling and biofeedback, may help patients who fail to respond adequately to reassurance alone.

Medications

Most causes of neurogenic dysphagia have no specific therapy. However, it is important to detect those for which drug therapy is available (e.g., myasthenia gravis, polymyositis, Parkinson's disease, and both hyper- and hypothyroidism).

A number of different drug therapies, administered for varying lengths of time, for patients with non-achalasia motility disorders have been utilized with differing degrees of success. However, very few of the studies have been well-controlled. Nor have they included large numbers of patients examined over time. Despite these shortcomings, several drugs have shown utility in well-controlled studies.

Short-acting nitrates have been shown to be effective in the relief of chest pain and dysphagia within minutes. The duration of action is approximately 30 minutes. Short-acting nitrates have been used in patients with diffuse esophageal spasm. These drugs are often used for rapid treatment of predictable patterns of symptoms.

Anticholinergic drugs such as hyoscyamine, atropine, cimetropium bromide, and propantheline have been associated with a decrease in peristalsis. These drugs, when compared to placebo, have demonstrated a reduction in symptoms in patients with non-achalasia motility disorders. Further studies of this class of drugs are warranted.

Calcium channel blockers have demonstrated utility in reducing lower esophageal sphincter pressure and the amplitude of esophageal contractions. Nifedipine, in doses of 10–30 mg 3 times per day, has been used in esophageal motility disorders and has shown to decrease the frequency, amplitude, and duration of chest pain attacks when compared to placebo. Diltiazem has also been shown to decrease the amplitude of pain, lower esophageal sphincter pressure, and impact the duration of esophageal contractions in patients in placebo-controlled trials.

Experimentally induced stress can result in considerable changes in esophageal peristalsis. Sedatives and tranquilizers have been shown to be helpful in patients with non-achalasia esophageal motility disorders accompanied by anxiety disorders and depression. Sedatives and tranquilizers such as trazodone, imipramine, and alprazolam may be useful in this group of patients with symptoms of chest pain and/or dysphagia.

Swallowing Retraining

Swallowing therapy is commonly recommended for neurogenic dysphagia and postoperative pharyngeal dysfunction and is most often performed by a speech-language pathologist with expertise in this area. Swallowing therapy employs modifications of bolus volume and consistency, head and neck position, and swallowing related maneuvers to improve swallowing efficiency. There are also exercises that have been developed to help selected patients with these types of disorders. The modified barium swallow is often used to determine the effect of potential interventions on the bolus transit and airway penetration. The information gathered from this the modified barium swallow may serve as the basis of treatment recommendations.

Botulinum Toxin

Botulinum toxin has been shown to improve symptoms in patients with achalasia. Success in this group has prompted clinical trials in patients with non-achalasia esophageal motility disorders who were unresponsive to drug therapy. The clinical trial group demonstrated short-term relief with intrasphincteric injection of botulinum toxin.

Dilation

Dilation utilizing any number of available dilator systems is used to increase the diameter of the swallowing channel. This procedure, usually performed at the time of an endoscopy, is very successful for webs, rings, and benign strictures. Re-stenosis is relatively common, especially in inflammatory strictures. However, effective treatment of the underlying inflammation will decrease the need for repeat dilations (Figure 27, 28, 29).

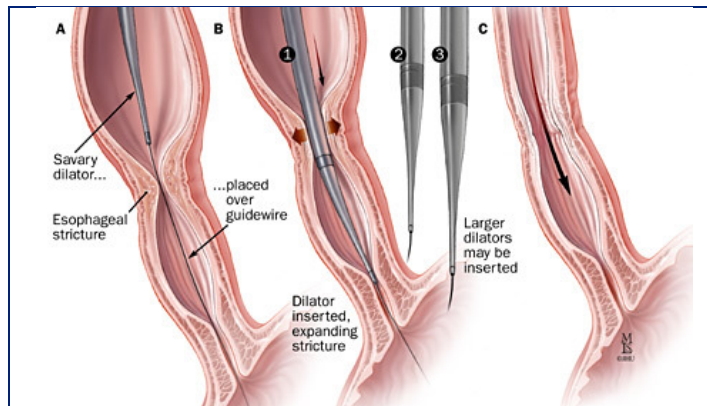


Figure 27. Endoscopic dilation. Technique for Savary dilation of an esophageal stricture.

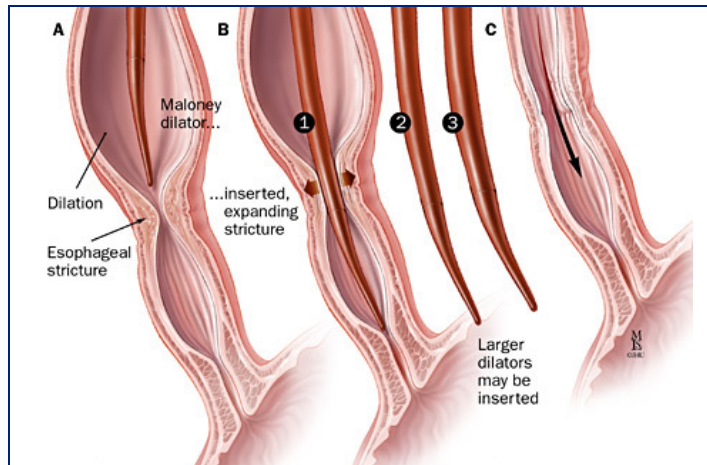


Figure 28. Endoscopic dilation. Technique for Maloney dilation of an esophageal stricture.

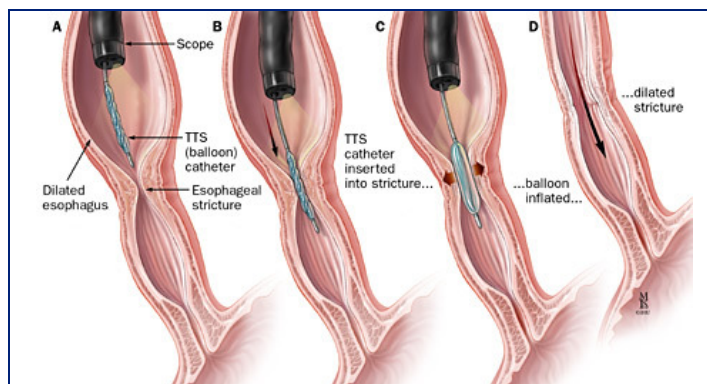


Figure 29. Endoscopic dilation. Technique for through-the-scope (TTS) dilation of an esophageal stricture.

In patients with achalasia, abrupt inflation of large diameter balloon dilators has been used for decades to weaken the lower esophageal sphincter and allow food to pass by gravity. Dilation of the upper esophageal sphincter has been successful in patients with isolated upper esophageal dysfunction, and in selected patients with neurogenic dysphagia.

Enteral Feeding

Some patients with swallowing disorders are so impaired that they are unable to take adequate nutrition safely by mouth. This is especially true in patients with neurogenic or postoperative dysphagia. Enteral feeding, most often provided by an endoscopically placed gastrostomy tube, provides a means of assuring adequate nutrition and decreasing the severity of aspiration. At the same time, measures are taken to correct the swallowing problem.

Enteral feeding should be considered for any patient who has lost substantial amounts of weight or has had recurrent aspiration pneumonia due to swallowing disorders, unless the initial evaluation indicates that the problem can be substantially improved in a short period of time. Patients are often reluctant to accept a gastrostomy. The physician must take the time to explain that malnutrition and excessively prolonged meals have a serious impact on the quality of a patient's life. It must also be explained that the gastrostomy tube does not prevent further efforts to treat the swallowing problem and it can be removed if and when the patient's ability to swallow, and consumption of an adequate number of calories, safely improves.

Structural Lesions

Structural lesions may be treated surgically and endoscopically. The goal of therapy is mechanical disruption. Myotomy is often used for sphincteric dysfunction. Myotomy reduces the resting sphincter tone by 50% without completely abolishing it by increasing sphincter opening capacity and reducing resistance to transsphincteric flow. Myotomy of the lower esophageal sphincter (Heller myotomy) is an effective alternative to dilation for achalasia. The major limitations are the discomfort associated with the operation itself and a relatively high risk of postoperative gastroesophageal reflux. Many surgeons add an anti-reflux operation to the myotomy to prevent reflux. Recent techniques for laparoscopic and thoracoscopic myotomy have been developed and have become the preferred approach to the surgical treatment of achalasia. Myotomy of the cricopharyngeal muscle (the major component of the upper esophageal sphincter) is commonly used as part of the surgical approach to diverticulectomy for Zenker's diverticula. It is also occasionally used for isolated upper esophageal sphincter dysfunction (a rare condition). Its use in patients with neurogenic dysphagia remains controversial.

Esophageal Stenosis

Avoiding offending foods, choosing foods prepared in creamy sauces, and eating slowly with careful attention to small bites and chewing may minimize the frequency of symptoms of esophageal stenosis.

Dysphagia from an inflammatory stricture requires the source of inflammation to be removed (e.g., by effective treatment of reflux disease) thereby decreasing associated swelling. With complete eradication of inflammation, scarring gradually stops and the size of the channel stabilizes.

Methods of mechanical disruption such as myotomy or dilation (refer to Figures 27–29), are efficacious in the treatment of esophageal webs and stenoses where there is preserved contractility. Almost half of the patients who undergo dilation will require repeat procedures over the course of many years.

Zenker's Diverticulum

Small Zenker's diverticulum do not require treatment intervention other than periodic esophagrams. In cases where treatment is required, surgical and endoscopic alternatives are available (Figure 31).

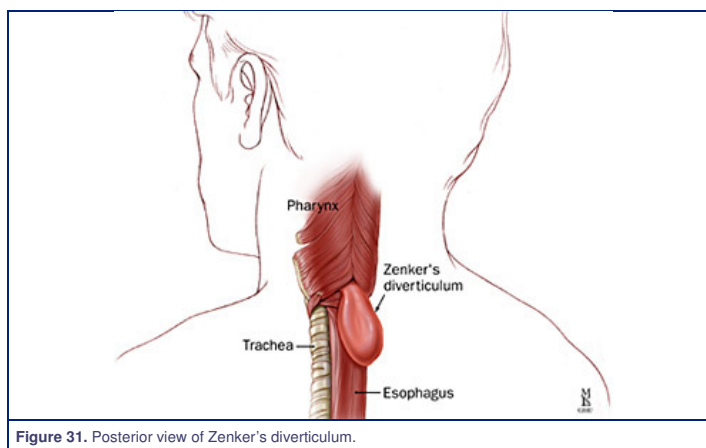


Figure 31. Posterior view of Zenker's diverticulum.

Treatment involves relieving the source of resistance by cutting the upper esophageal sphincter (cricopharyngeal myotomy) and removing the diverticulum (diverticulectomy). Diverticulectomy with (Figure 32) or without myotomy (Figure 33) is the traditional approach to the treatment of Zenker's diverticulum. A one-stage pharyngoesophageal diverticulectomy with or without myotomy has been shown to provide excellent results with low mortality (1.2%), morbidity (8%) and recurrence rates (3.6%).

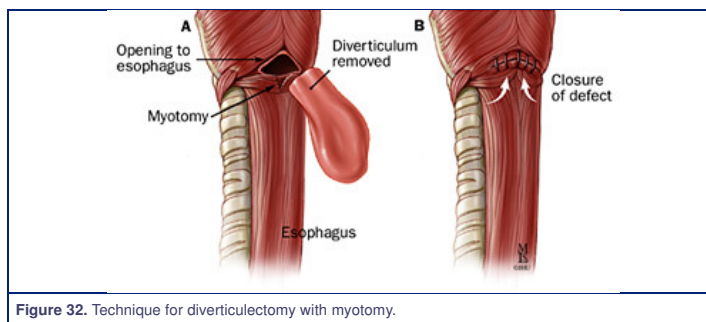


Figure 32. Technique for diverticulectomy with myotomy.

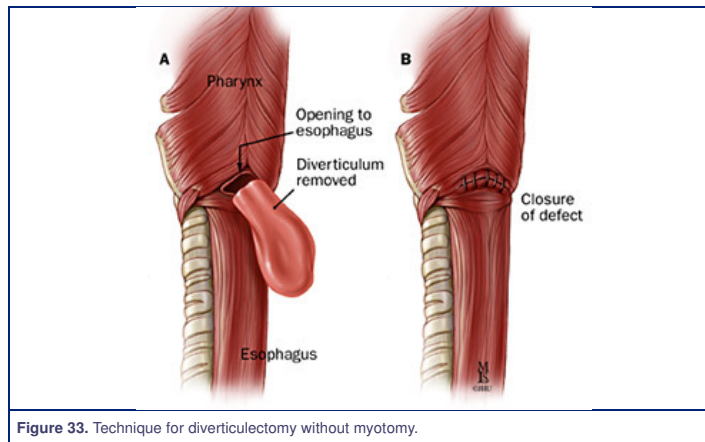


Figure 33. Technique for diverticulectomy without myotomy.

The surgery is performed through a left cervical incision. The diverticulum is retracted, dissected, and resected with sutured or stapled closure. Myotomy, dissection of the cricopharyngeal muscle fibers away from the mucosa, may be performed before or after diverticulectomy. Long-term follow-up studies are needed to assess physiologic consequences of these procedures. Complications such as infection, vocal cord paralysis, fistulas, and aspiration may result from surgical intervention.

Recently, techniques for the endoscopic approach to treatment of a Zenker's diverticulum have been developed. In these procedures, the wall between the diverticulum and the esophageal lumen is divided, thereby cutting the upper esophageal sphincter and allowing swallowed food and liquid to decant out of the unroofed pouch. Modifications of this technique using laser versus electrocoagulation have shown comparable results, though the laser procedure appeared to produce less pain and faster recovery. Whether this represents a more efficacious procedure than the traditional transcutaneous diverticulectomy is still uncertain.

Endoscopic management of Zenker's is not popular in the US. Most surgeons perform myotomy with diverticulectomy.

Esophageal Diverticulum

Small diverticula are often asymptomatic and should prompt further evaluation for an alternative cause of symptoms. However, when symptoms do arise from a diverticulum, the condition is often treated by surgical removal (diverticulectomy). Diverticula are usually a consequence of chronic obstruction downstream from the diverticulum. Unless the obstructive lesion is found and treated, symptoms may not improve with diverticulectomy.

Midesophageal diverticula are often asymptomatic and do not require treatment. However, if treatment does become necessary it is important to exclude esophageal motility disorders before recommending surgery. Though surgery is rarely indicated, diverticulectomy with or without myotomy is the procedure of choice.

Epiphrenic diverticula, arising near the diaphragm, usually do not require treatment. However, in symptomatic patients the goal of therapy should be aimed at removing the cause of abnormal resistance to avoid further enlargement of the diverticulum. Diverticulectomy alone or with myotomy has shown favorable results in symptom relief, weight gain, and absence of recurrence.

Neurogenic Dysphagia

A multidisciplinary approach to the treatment of neurogenic dysphagia is often needed for optimal patient management. Since neurogenic dysphagia may be the presenting or accompanying symptom of many systemic diseases, the strategy is to treat any underlying disease whenever possible. Structural, neuromyogenic mechanisms need to be identified by use of radiographic and endoscopic evaluation; treatment is dictated by these assessments. The risk of aspiration pneumonia also needs to be assessed. If this risk is high, gastrostomy feeding should be instituted. Local therapy should be considered after structural lesions, treatable underlying disease, and the safety of oral feeding have been established.

Modification of the diet, swallowing therapy, and surgery (singularly or in combination), constitute therapeutic options. The treatment for neurogenic dysphagia emphasizes measures to improve the efficiency of swallowing to minimize aspiration. Swallowing retraining, most often under the direction of a specially trained speech-language pathologist or occupational therapist, is an important method of intervention. Swallowing technique is modified improving the speed and range of movement of oropharyngeal muscles. The mechanics involved in swallowing are selectively modified to facilitate flow and minimize aspiration. In select patients, when objective testing suggests that the upper esophageal sphincter is contributing to the severity of swallowing problems, therapy directed at weakening the upper esophageal sphincter (myotomy) might be helpful. When adequate nutrition cannot be maintained safely, alternative methods of feeding—most often a feeding tube placed directly into the stomach—may be advisable.

Isolated UES Dysfunction

The severity of symptoms in isolated upper esophageal sphincter dysfunction often responds to treatment directed at weakening the lower esophageal sphincter (using, for example, dilation or myotomy). These measures work only occasionally in patients with neurogenic dysphagia, even when the cricopharyngeal segment appears to open poorly.

Diffuse Esophageal Spasm

Patients with symptoms suggestive of esophageal spasm require assessment to determine if reflux or stenotic lesions are playing a role. These secondary spastic conditions differ from idiopathic spasm. Benign esophageal stenosis is usually treated by esophageal dilation. Reflux is treated by dietary measures designed to avoid the stimulation of gastric acid and to decrease the volume of gastroesophageal regurgitation. These measures are usually combined with drugs that decrease acid secretion and improve gastric emptying. Idiopathic spasm is treated with drugs (smooth muscle relaxants) that decrease the strength of esophageal contractions. Often, however, smooth muscle relaxants are ineffective for the treatment of esophageal spasm. Recent studies suggest that patients with symptoms of esophageal spasm, especially chest pain, have a sensory disorder in which the threshold for perceiving esophageal stimulation as a painful phenomenon is decreased. The implication of this sensory disorder for treatment of spasm-like chest pain remains unclear.

Achalasia

Dilation of the obstructed distal esophagus is the focus of achalasia treatment. Treatment involves decreasing the pressure in, and thereby the resistance to, flow through the lower esophageal sphincter.

Dilation is probably the most common approach to treating the patient with achalasia. Pneumatic dilation is performed endoscopically (Figure 34). Patients are sedated with intravenous medications. Esophageal lavage and aspiration of gastric contents are performed prior to positioning the endoscope in the antrum. A stiff guide wire is advanced through the biopsy channel of the scope and positioned in the antrum (Figure 34A). The dilator is passed over the guide wire and advanced through the gastroesophageal junction. The balloon is slowly inflated and held in position. Gradual increase in the size of the dilator may be necessary (Figure 34B). The objective is to stretch the circular muscle of the lower esophageal sphincter to allow the passage of solids and liquids without subsequent reflux or perforation (Figure 34C,D).

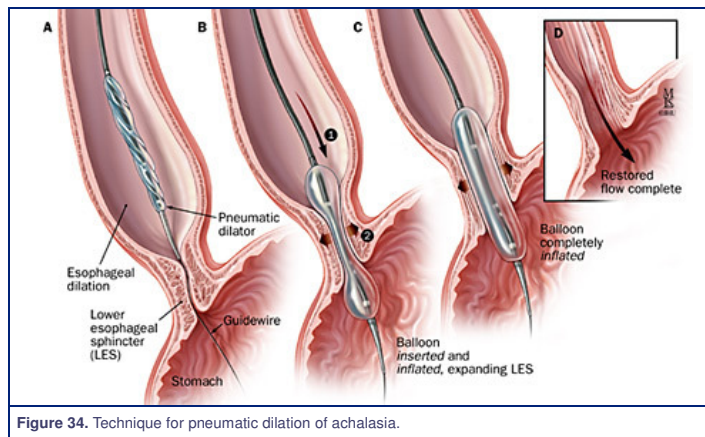


Figure 34. Technique for pneumatic dilation of achalasia.

Controversy exists regarding the efficacy of using rapid, maximal, short duration stretches compared with rapid, submaximal long duration stretches. Most patients (65%–80%) report a significant improvement in their symptoms of dysphagia. Physiologically lower esophageal pressures decrease immediately after pneumatic dilation, but increase over time with a concomitant increase in symptoms.

Recently, intrasphincteric injection of the potent neurotoxin botulinum toxin has been successfully used in patients with achalasia. Safe and effective in the majority of patients, it is particularly effective in the elderly and has earned a place in the management of patients considered poor candidates for dilation or myotomy. The procedure involves injection of the lower esophageal sphincter causing a chemical denervation of the sphincter. Twenty to 25 units of botulinum toxin are injected into each quadrant of the lower esophageal sphincter with a sclerotherapy needle passed through the endoscope (Figure 35). Although it is the safest of available techniques, botulinum toxin injection has a limited duration of effect, lasting on average one year. Repeat treatment is necessary to maintain the effect. Some patients may experience mild chest pain and there have been reports of skin rashes noted after treatment.

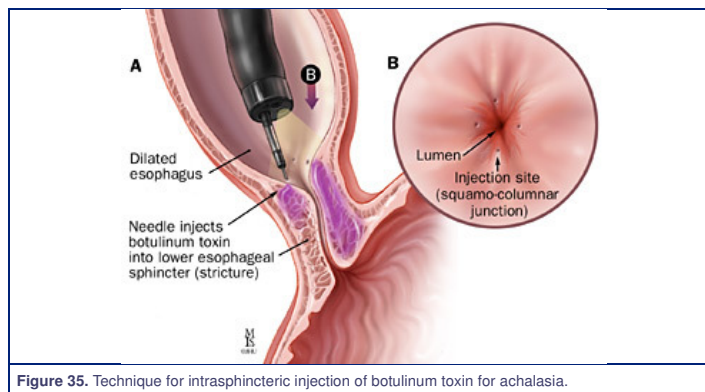


Figure 35. Technique for intrasphincteric injection of botulinum toxin for achalasia.

Minimally invasive surgery, using either a laparoscopic or a thorascopic technique has significantly decreased the morbidity associated with achalasia surgery. A single anterior lateral myotomy (Figure 36) or a modified Heller myotomy is typically the surgical procedure performed. A lower esophageal sphincter myotomy incises enough muscle to relieve symptoms but not enough to result in gastroesophageal reflux. In many cases, an antireflux procedure is performed at the same time.

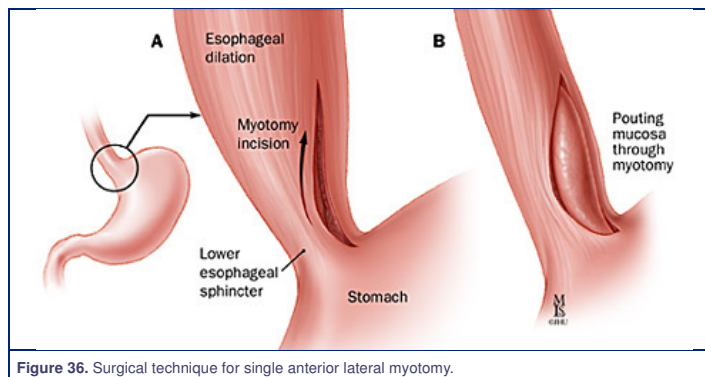


Figure 36. Surgical technique for single anterior lateral myotomy.

Overview

The inability to obtain adequate nutrition results in weight loss and easy fatigability. Difficulty swallowing can lead to a voluntary decrease in food intake and alterations in diet that can further impair nutrition. Aspiration can lead to pneumonia, bronchiectasis, and life-threatening pneumonia (aspiration pneumonia). These problems are correctable, either by treatment of swallowing disorders or by provision of adequate calories through an alternative route.

Treatment may have its own complications. All drugs have the potential for producing side effects—some allergic and others dose-related. Dilation may result in perforation or less often, gastrointestinal bleeding. These complications are relatively rare with dilation for benign strictures, but are more common if the dilation is performed for malignant strictures or achalasia. The risks of radiation therapy are discussed in the section on esophageal cancer. Surgery carries risks associated with anesthesia and the specific surgical procedure.