



# Approach to the Patient with Dysphagia

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## ABSTRACT

Dysphagia is a fascinating symptom. It is ostensibly simple when defined by trouble swallowing, yet its subtleties in deciphering and its variations in pathophysiology almost mandate a thorough knowledge of medicine itself. With patience and careful questioning, a multitude of various disorders may be suggested before an objective test is performed. Indeed, the ability to diligently and comprehensively explore the symptom of dysphagia is not only rewarding but also a real test for a physician who prides himself or herself on good history taking.

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## DEFINITION

### Objective

Dysphagia is defined objectively as an abnormal delay in transit of a liquid or solid bolus during the oropharyngeal or esophageal stages of swallowing. This delay can be transient, lasting seconds, or at its most severe manifest as a fixed delay, as in a food impaction. The periodicity also may vary widely, occurring yearly or with every swallow attempt. Nevertheless, if a test of esophageal transit such as barium radiography, nuclear scintigraphy, or multichannel impedance was performed, there should be clear evidence of abnormally slow bolus transport in a point between the mouth and the stomach. Likewise, a test of anatomic or motility assessment of the oropharynx and esophagus would demonstrate a finding that is associated clearly with objective dysphagia.

### Subjective

Dysphagia as defined subjectively is the sensation of a delay in transit of a liquid or solid bolus during the

oropharyngeal or esophageal stages of swallowing (oropharyngeal and esophageal dysphagia, respectively). This could be distinctly different from the objective measurement of dysphagia because various mechanisms of esophageal sensory function may account for the sensation of dysphagia without apparent delay in bolus transit. For example, in patients with functional dysphagia, symptoms may reflect a feeling of the passage of a bolus through the esophagus, even with normal transit. Likewise, a symptom that is generated from a true delay in bolus transit may be potentiated or attenuated through sensory neural dysfunction to seem out of proportion or outlast, respectively, a short-lived delay in bolus transit.<sup>1</sup> Conversely, other disorders may manifest with hyposensitive function of the esophagus, such that patients feel neither the severity nor the duration of bolus impaction as occurs in the late stages of achalasia.<sup>2</sup>

### Questionnaires

Several research groups have devised questionnaires to aid in the measurement and interpretation of dysphagia in a patient.<sup>3</sup> These questionnaires serve several purposes. The first is a standardization of questions, which may be applied to research as a measurable tool. The second is to complete a thorough characterization of dysphagia. For example, these questionnaires commonly seek additional information about the dysphagia, such as chronicity, frequency, severity, and associated symptoms. The third is to tailor the dysphagia to the clinical situation in which it is being applied. For

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example, some of these questionnaires,<sup>4</sup> such as the Mayo Dysphagia Questionnaire<sup>5,6</sup> and Northwestern Questionnaire,<sup>7</sup> have been developed for the general population. Others are disease specific, such as those developed for patients who have eosinophilic esophagitis or sustained strokes.<sup>8</sup>

## CATEGORIES

### Motility Versus Anatomic

Anatomic causes of dysphagia are usually those that compromise the esophageal lumen. These processes may be benign or malignant, oropharyngeal or distal esophageal, mucosal, intramural, or extrinsic to the esophagus. Common mechanical causes of dysphagia may include reflux-induced esophageal strictures, head and neck or esophageal cancer, and extrinsic compression from mediastinal lymph nodes or lung cancer. The general clinical principal related to anatomic causes of dysphagia is that symptoms primarily occur with ingestion of solids and not liquids and are more likely with larger solids and those of denser consistency (Figure 1).

In contrast, motility causes of dysphagia are more likely to occur with both solids and liquids as the neuromuscular forces required to propel the bolus affect both liquid and solid transport. Some motility disorders may start with solids, which require greater esophageal contractile amplitude, thus unmasking an esophageal muscular disorder earlier in the pathogenesis of the neuromyopathy, but with progression will involve liquids. In motility disorders of the oropharynx, liquids may be more problematic than solids because of easier entry to the airway with failure of the epiglottis to cover and of elevation of the laryngeal structures to protect the laryngeal vestibule. Typical disorders of foregut motility are achalasia or oropharyngeal dysfunction secondary to a cerebrovascular accident.

### Upper Esophageal Versus Mid- and Lower Esophageal

Determining the location of esophageal dysfunction relative to where the patient feels the symptoms is not reliable. This has been found not only clinically but also through experimentation in which balloon distention of the esophagus or duplication of symptoms by radiographic impaction of a marshmallow in the distal esophagus<sup>9-11</sup> leads to a highly variable symptom location among individuals. One factor that may help determine location is detecting a perturbation of other oropharyngeal functions accompanying dysphagia. For example, because cranial nerve deficits commonly affect

other functions in addition to swallowing, patients with oropharyngeal dysphagia may note voice changes, nasal regurgitation (failure to seal off the nasopharynx), or prandial coughing (failure to protect the airway). If dysphagia leads to regurgitation, the volume of regurgitant also may be helpful. For example, the esophageal body is capable of holding a larger volume of food with obstruction than the oropharynx.

## CLINICAL SIGNIFICANCE

- To perform a well-done dysphagia evaluation, it is necessary to recognize alarm symptoms.
- Order barium esophagram early if you suspect oropharyngeal or motility disorder.
- If dysphagia persists, or the diagnosis remains unclear despite initial work up, consider referring to the appropriate specialist (GI, ENT, Neurology) at an early stage.
- Eosinophilic esophagitis is an emerging cause of dysphagia, especially to solids, with food impaction in advanced cases.

### Organ-Specific Versus Involvement by Another Disease

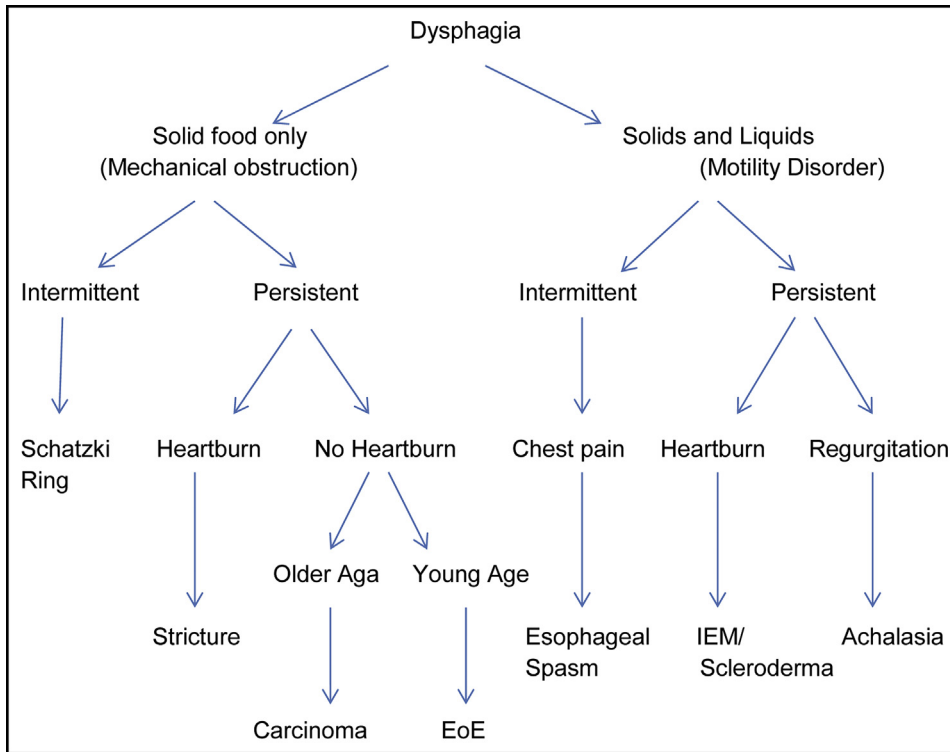
In a patient with dysphagia, one also has to determine whether the symptom derives from an esophageal specific disorder or a more generalized disorder affecting the esophagus. For example, systemic neuromuscular diseases, such as scleroderma, may cause dysphagia of the lower esophagus, whereas amyotrophic lateral sclerosis or myasthenia gravis may affect oropharyngeal function. Other diseases may lead to stricture formation, such as lichen planus or

Crohn's disease, whereas others may lead to extrinsic compression, such as dysphagia aortica or dysphagia lusoria. Furthermore, diseases such as breast or lung cancer may cause an achalasia-like syndrome without clear evidence of the cancer on initial evaluation. As a result, detection of an esophageal disorder leading to dysphagia may not just stop at the esophagus. This is emphasized further by the fact that many of the disorders described and other systemic disorders may present with esophageal symptoms.

## DIAGNOSTIC APPROACH

### Initial Evaluation

**Further History.** In addition to the routine characterizations of a symptom, such as chronicity, frequency, and severity, further history needs to be pursued to determine the category of dysphagia under which it falls (as described earlier) and, if possible, the specific cause or disease in that category that is causing the dysphagia (Figures 1 and 2). Because specific diseases and locations commonly are associated with symptoms in addition to dysphagia, it is important to elicit these symptoms in the history. For example, because individual cranial nerves innervate muscles pertinent to multiple aspects of normal oropharyngeal function, such as mouth closing, sealing off of the nasal cavities, and protection of the airway and voice, cranial neuropathies may cause drooling, nasal



**Figure 1** Symptom differential of common causes of esophageal dysphagia. Overlap exists among the features. Persistent dysphagia represents more severe disease that requires earlier medical attention. EoE = eosinophilic esophagitis; IEM = ineffective esophageal motility.

regurgitation, pulmonary aspiration, and voice dysfunction, respectively, in addition to dysphagia.

It is important to appreciate that patients with chronic diseases commonly learn techniques and strategies to adapt to their symptom and thus ease the difficulty that attends this problem. This is particularly true in patients with dysphagia. As a result, it is important in collecting further history to

inquire about compensatory mechanisms that reduce the frequency and severity of dysphagia. Such adaptations include eating slowly, manifest by being the last to finish a meal in company. Another adaptation is avoiding problematic foods. Indeed, some patients state they do not have dysphagia, but a careful dietary history may reveal the avoidance of hard solids such as meat or bread in these patients.

Patients will also learn techniques to facilitate bolus passage. For example, patients with solid food dysphagia may drink fluids with every bite to facilitate bolus passage. When patients sustain episodes of complete bolus obstruction, they may also learn to regurgitate as a method of clearing the bolus. As a result, patients may avoid social situations in which they fear occurrence of and embarrassment at their dysphagia, such as meals at restaurants and business affairs. Finally, another important aspect in eliciting a history of dysphagia is asking an accompanying family member about the symptom. Often, they will note the slow eating and struggle at mealtimes better than the patient who has both consciously and subconsciously adapted to the disorder.

Anatomic	Neurologic	Muscular
Zenker's	Cerebrovascular	Polymyositis
Diverticulum	Accident	Myasthenia Gravis
Tumor	Post-Polio Syndrome	Muscular dystrophy
Enlarged Thyroid	Radiation Injury	Radiation Injury
Osteophyte	Parkinsonism	Thyroid Disease
Head/Neck Surgery	Head/Neck Surgery	Head/Neck surgery
Web	Multiple Sclerosis	
Abscess	CNS tumor	
	Botulism	
	Supranuclear Palsy	
	Myotonic Dystrophy	
	Amyotrophic Lateral Sclerosis	

**Figure 2** Disorders causing oropharyngeal dysphagia. Several disorders may manifest with more than 1 cause of dysphagia, such as head and neck surgery or radiation injury. Zenker's diverticulum arises from dysmotility but then gives rise to further dysphagia by esophageal compression. CNS = central nervous system.

**Physical Examination.** Examination of the patient may play a strong role in determining the cause of dysphagia. This is particularly true in patients with a suspected neuromuscular cause of difficulty swallowing. As a result, a simple examination of cranial nerves should be performed,

which can easily be accomplished in a brief time. Likewise, more generalized involvement of these disorders should be sought, such as proximal or asymmetric extremity weakness, dysarthria, fasciculations, tremor, and cognitive dysfunction.

An oral examination is also necessary. Clues to the cause of dysphagia might include poor dentition, buccal lesions such as lichen planus, tongue fasciculations, asymmetric palate elevation, and labial droop. A neck examination may be helpful if a mass or adenopathy is detected, such as a Virchow's node associated with esophageal cancer.

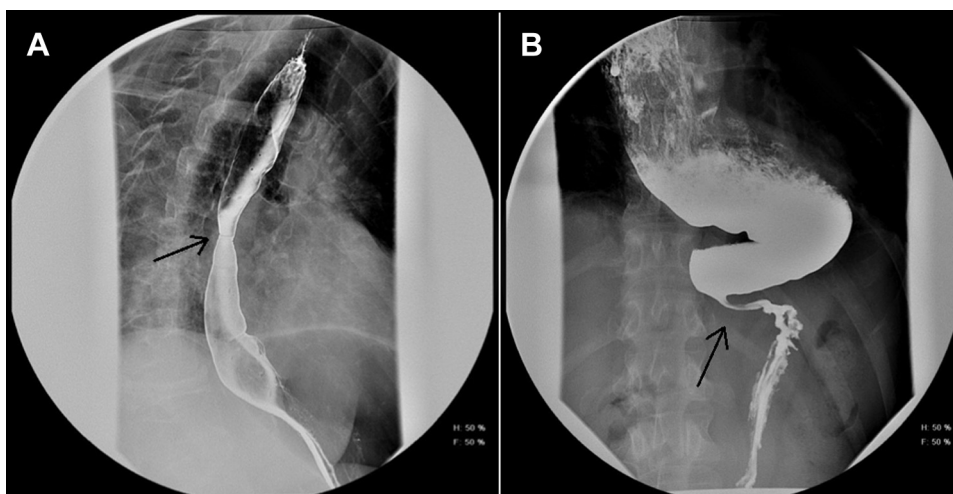
**Bedside Testing.** One of the easiest and potentially important parts of the physical examination is watching the patient swallow in the office. Observation of deglutition of a simple glass of water or bite of a solid food can give a plethora of information on the patient's dysphagia. Patients commonly underestimate their degree of difficulty, but it may be revealed by observations such as multiple swallows required for a single bolus, the use of small sips of fluid or small bites of a solid, post-deglutitive throat clearing, and a general fear of swallowing.

**Barium Swallow Versus Endoscopy.** The first important consideration of these 2 tests is in realizing they are complementary and not duplicative (Figure 3).<sup>12</sup> The decision on which test to order initially depends in large part on which is most likely to yield the diagnosis. Because endoscopy gives accurate information on esophageal anatomy, it is used commonly as the first test to evaluate solid food dysphagia. It allows for more precise mucosal inspection and the ability to biopsy. Endoscopy also serves as a potential therapeutic tool because dilation may be performed if needed at the time of the diagnostic study.

On the other hand, barium esophagography gives far greater information for motility disorders. As a result, for

oropharyngeal dysphagia (which is mostly attributable to dysmotility), a video modified barium swallow is the initial diagnostic test of choice.<sup>13-15</sup> This approach not only allows for direct visualization of muscular strength and coordination but also usually includes different food consistencies to test or duplicate the patient's symptoms. Furthermore, this test is performed commonly with a speech and swallowing therapist, who not only has expertise in swallowing disorders but also may start to implement effective therapy with key compensatory maneuvers based on the study. For suspected distal esophageal motility disorders such as achalasia<sup>16</sup> or scleroderma<sup>17</sup> involving the esophagus, barium evaluation has been shown to be superior to endoscopy. Barium esophagography is more sensitive than endoscopy for detection of esophageal strictures.<sup>18-20</sup> Another advantage of an initial esophagogram is the ability to plan the endoscopy. For example, identification of a tight esophageal stricture may help determine the type of dilation needed and the potential need for fluoroscopy to aid in the dilation. Likewise, an extant diagnosis of achalasia by barium may allow for pneumatic dilation or injection of intrasphincteric botulinum toxin during endoscopy. Finally, for the purposes of finding an esophageal fistula or perforation in a patient with dysphagia, radiography is the safest and most accurate approach. It is our belief that the diagnostic approach to all patients with dysphagia should begin with a barium study, although performing endoscopy, particularly for patients with suspected mechanical causes of dysphagia, is reasonable.

**High-Resolution Impedance Manometry.** High-resolution impedance manometry is a modern adaptation of traditional esophageal manometry but with greater accuracy and visualization based on having 32 pressure transducers that span the esophagus—as opposed to the limitations of only a few transducers in the past.<sup>21,22</sup> As a result, a clear



**Figure 3** Barium esophagogram of (A) mid-esophageal stricture (arrow) and (B) advanced Achalasia (characterized by dilation and sigmoidization of esophagus with arrow pointing to classic bird's beak sign).



panesophageal pressure tracing can be generated and then described through a colorimetric graphic presentation (the Clouse plot) (Figure 4).<sup>23</sup> This gives a far clearer reading on peristaltic and sphincter function. Impedance measurement also has been added to this technique such that catheter-based electrodes measure the conductivity of a substance based on the characteristics and speed of the bolus.<sup>24</sup> A swallow of a liquid-based bolus will conduct electricity better and therefore generate a low impedance signal concordant with esophageal peristalsis. Because direction of flow is measured easily, reflux of content from the stomach into the esophagus also may be easily seen. Ideally, this combination of manometry and impedance measurement is suited for assessing esophageal motility disorders. There is recent evidence that abnormal bolus transit detected by impedance during test swallows is the most sensitive indicator of abnormal esophageal motility. Ordering the appropriate diagnostic test by the referring physician helps in eliciting the next step in management of the patient's dysphagia.

## SPECIFIC DISORDERS

### Neuromuscular Diseases

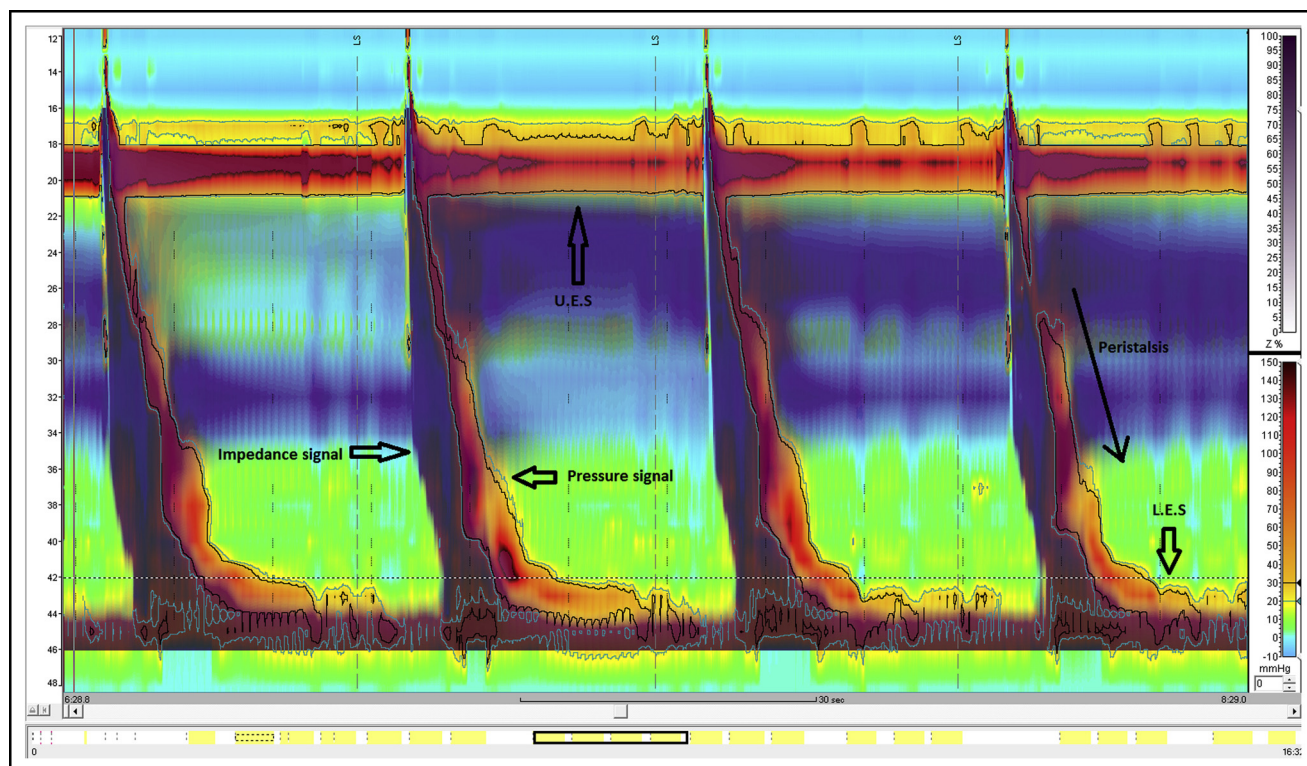
Control of oropharyngeal function is mediated by cerebral, brain stem, cranial nerve, and striated muscle function. Thus, numerous neural or muscular disorders may involve or even manifest with oropharyngeal dysphagia. For example, centrally mediated diseases that may cause

dysphagia include cerebrovascular accidents, Parkinson's disease, or progressive supranuclear palsy. Diseases that affect the brain stem and cranial nerve function include botulism, amyotrophic lateral sclerosis, and supranuclear palsy. Examples of primary striated muscle disease are inclusion body myositis, myasthenia gravis, and polymyositis. The important aspect of these diseases, as discussed earlier, is that multiple stages of the oropharyngeal portion of the swallow may be affected.

### Stricturing Diseases

Diseases that lead to fibrotic change and luminal narrowing of the esophagus are common entities that cause dysphagia. They all uniformly lead to solid food dysphagia. Esophageal strictures due to gastroesophageal reflux are most common, although some data suggest that with increasing use of proton pump inhibitors, these may be decreasing in incidence.<sup>25,26</sup> These strictures may manifest as mildly symptomatic focal distal annular narrowing to severely symptomatic long distal esophageal strictures involving a large portion of the esophagus. The treatment rests in adequate control of reflux and mechanical dilation of the strictures. Of note, as in location, the severity of solid food dysphagia does not always belie the severity of luminal narrowing because patients commonly adapt with avoiding certain foods, chewing carefully, and using fluids copiously with solid meals.

Another common stricturing disease is eosinophilic esophagitis.<sup>27</sup> Initially thought uncommon, this disease is



**Figure 4** Normal high-resolution impedance manometry. LES = lower esophageal sphincter; UES = upper esophageal sphincter.

estimated to occur in 0.4% of the population.<sup>28</sup> It typically affects children, adolescents, and young adults but is becoming increasingly recognized in middle-aged patients. Eosinophilic esophagitis manifests typically in adults with years of dysphagia, sometimes punctuated by food impactions. Its pathophysiology centers on a T-helper 2-mediated food allergy of the esophagus, which leads to chronic eosinophilic infiltration, inflammation, and fibrogenesis. Treatment is aimed at identification and elimination of causative foods or medical control of the allergic response by using topical or systemic steroids. The need for dilation is also common in this disease.

Other stricturing diseases include iatrogenic causes, such as radiation therapy or caustic medications (eg, bisphosphonates, doxycycline, or potassium supplements), skin diseases (eg, lichen planus or pemphigus syndromes), caustic ingestion, and Crohn's disease.

## Esophageal Cancer

Adenocarcinoma is one of the most rapidly increasing cancers in Western society, now far outnumbering the previously common squamous cell carcinoma.<sup>29</sup> Theories that might explain this increasing incidence include increasing gastroesophageal reflux disease and central obesity (which mechanically and chemically may contribute to increasing esophageal neoplasia),<sup>30</sup> decreasing *Helicobacter pylori* infection (which may allow for more patients with higher levels of gastric acid secretion),<sup>31,32</sup> and dietary changes.<sup>33,34</sup> Barrett's esophagus is the most dominant precursor to adenocarcinoma. Dysphagia symptoms may be insidious, starting intermittently with hard solids such as bread and meats and then progressing. Patients may commonly have advanced lesions with marked luminal narrowing yet mild symptoms. Whether this reflects accommodation or a generalized esophageal hyposensitivity to symptoms is unclear. Other alarm symptoms that may accompany progressive dysphagia include weight loss, anorexia, and hematemesis.

## Achalasia

Achalasia is the prototype esophageal motility disorder characterized manometrically by aperistalsis and an incompletely relaxing lower esophageal sphincter.<sup>35</sup> Although often considered typical, a hypertensive lower esophageal sphincter may not be present. A new manometric profile, the Chicago Classification,<sup>36</sup> based on high-resolution impedance manometry, has been proposed to better characterize these variants. On radiography, patients have a dilated esophagus with an incompletely opening lower esophageal sphincter. The underlying cause seems to be an autoimmune esophageal ganglionitis with relative loss of inhibitory input possibly initiated by a viral infection.<sup>37</sup> Although dysphagia to liquids and solids is the most common symptom, patients may have a wide variation in symptoms, including chest pain, unresponsive heartburn,

weight loss, and regurgitation.<sup>38</sup> Treatment is aimed at mechanical disruption of the lower esophageal sphincter through pneumatic dilation or laparoscopic, and more recently, endoscopic myotomy.

## Functional Dysphagia

At the beginning of this review, dysphagia was defined specifically as an abnormal delay in bolus transit. This objective definition is stated because patients may note a sense of dysphagia despite normal radiographic or manometric measures of normal transit. These patients are theorized to have a sensory disorder in which they sense the normally passing bolus due to augmented afferent esophageal sensation.<sup>39</sup> Whether this is a disorder or peripheral or central sensory processing is unclear. These patients will "feel" the bolus going down. Conversely, they may still sense food or fluid in their esophagus for prolonged periods after the meal, although they may still eat and drink without difficulty. Radiography is an excellent means of making this diagnosis in which ingestants, such as barium-coated foods or tablets, may reproduce symptoms yet demonstrate normal transport. Another clue to this disorder in younger patients is the association of dysphagia with other functional disorders of the gastrointestinal tract, including dyspepsia, bloating, early satiety, and belching.<sup>40</sup> Treatment may include pharmacologic, behavioral, or cognitive therapy.

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