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Esophageal Etiologies of Dysphagia: A Guide for SLPs

Joel H. Rubenstein
Division of Gastroenterology
University of Michigan Medical School and the Ann Arbor
Veterans Affairs Medical Center
Ann Arbor, MI

Introduction

Patients referred to speech-language pathologists (SLPs) for dysphagia often have a primary esophageal etiology for their dysphagia. This is likely in part due to referring providers’ limited experience in distinguishing esophageal dysphagia from oropharyngeal dysphagia, but even expert otolaryngologists, gastroenterologists, and SLPs need assistance from each other in complicated cases for excluding etiologies from each other’s domain of expertise. Furthermore, SLPs may discover hesitancy in the initiation of the swallow or discover repetitive swallows, behaviors that may be due to poor esophageal clearance of the bolus. The aim of this overview is to familiarize SLPs with the esophageal physiology of the swallowing mechanism, the common and classic esophageal etiologies of dysphagia, the diagnostic modalities, and indications for referral.

Esophageal Physiology

The proximal third of the esophagus is made of striated muscle like the oropharyngeal musculature. The distal third of the esophagus is made of smooth muscle, as is the remainder of the gut. After the relaxation of the upper esophageal sphincter, and pharyngeal clearance of the bolus into the proximal esophagus, a reflexive, peristaltic “primary stripping wave” is initiated that propels the bolus distally. The amplitude of this peristaltic wave is normally higher in the distal esophagus (50–150 mmHg) than the proximal esophagus (20–120 mmHg), and it travels at approximately 3–5 cm per second. The lower esophageal sphincter (LES) is likewise made of smooth muscle, and, unlike the upper esophageal sphincter, it is not composed of a distinct set of muscle fibers that can be identified pathologically, nor can it be precisely identified endoscopically. Rather, it is a functional sphincter that can be identified manometrically in the living subject. The LES is tonically contracted and is aided by the impegement of the diaphragm to produce a pressure of approximately 10–40 mmHg in order to prevent reflux of gastric contents from the abdomen (which is under negative pressure), into the chest (which is under negative pressure, keeping the lungs inflated). As soon as the primary stripping wave is initiated by the oropharyngeal swallow, the LES relaxes to approximately 0 mmHg, and certainly less than 10 mmHg, and remains open until the stripping wave has arrived at the LES and the bolus has passed into the stomach. Secondary stripping waves, which are coordinated peristaltic waves that originate within the proximal esophagus, rather than from an oropharyngeal swallow, may propel any residual bolus into the stomach.

In general terms, dysphagia occurs when the amplitude of the stripping wave is too low, when the velocity of the wave is too slow or too fast (particularly when the wave is so fast that proximal and distal segments contract simultaneously), when the LES fails to relax, or when there is a mechanical obstruction that cannot be overcome by the stripping wave.

Clues to Etiology

Most esophageal causes of dysphagia are associated with few, if any, findings on a general physical examination, other than perhaps cachexia. Rarely, there may be findings that are suggestive of a systemic disease or metastases from esophageal cancer. In contrast, a careful history is imperative in order to minimize the number of tests the patient is subjected to and in order to expeditiously diagnose the patient.

First, esophageal etiologies should be distinguished from oropharyngeal etiologies (Table 1). Patients should be asked to point with one finger to where they feel the “hang up” of the bolus. In contrast to patients with oropharyngeal dysphagia, patients with esophageal etiologies usually localize the “hang up” distal to the larynx, less frequently report aspiration events or recurrent pneumonias, and almost never report nasopharyngeal regurgitation. Food impactions (where patients have prolonged stasis of the bolus to the point that they develop chest discomfort and cannot swallow their saliva, often resulting in visits to emergency rooms) are reported much more commonly in patients with mechanical causes of dysphagia than motility disturbances. Also, patients with mechanical obstruction typically can still swallow liquids with little or no difficulty, but have particular difficulty with cohesive foods like bread or poorly fractured foods like meat, poultry (the proverbial “chicken caught in the goose”), and, less frequently,

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**Table 1. Symptoms of Oropharyngeal Dysphagia, Esophageal Mechanical Dysphagia, and Esophageal Dysmotilities**

<table>
<thead>
<tr>
<th>Subjective localization</th>
<th>Oropharyngeal</th>
<th>Esophageal Mechanical</th>
<th>Esophageal Dysmotility</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bolus type</td>
<td>Varies by etiology</td>
<td>Solid &gt;&gt; liquid</td>
<td>Liquid &amp; solid</td>
</tr>
<tr>
<td>Aspiration</td>
<td>+++</td>
<td>Rare</td>
<td>+</td>
</tr>
<tr>
<td>Impaction</td>
<td>Rare</td>
<td>++</td>
<td>Rare</td>
</tr>
<tr>
<td>Nasopharyngeal regurgitation</td>
<td>+</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Dysphonia or dysarthria</td>
<td>++</td>
<td>–</td>
<td>Rare</td>
</tr>
</tbody>
</table>
roughage. A rapidly progressive course over a few months or weeks suggests a malignant cause.

In the setting of mechanical obstruction of the esophagus, the objective location of the stenosis is either near the point that the patient subjectively localizes it or distal (Wilcox, Alexander, & Clark, 1995). It is rarely more than 4 cm proximal to the subjective localization. Note that this rule is limited to patients who localize the stenosis distal to the level of the larynx. Those who localize it to the level of the larynx or proximal almost always have oropharyngeal dysphagia.

Diagnostic Modalities

The common modalities used to diagnose esophageal etiologies of dysphagia include esophagoduodenoscopy (EGD), also known as “upper endoscopy” (not to be confused with “upper GI” which is a barium radiograph exam of the stomach), barium esophagram, and esophageal manometry. EGD employs a flexible videoendoscope, typically 9–10 mm in diameter, and allows for the ability to obtain biopsies, and to perform dilations of strictures (usually via an inflatable balloon placed through the scope, or with tapered dilators placed over a guidewire that is introduced through the scope). EGD is routinely performed with the use of conscious sedation in the United States, but not in Europe or Asia. With sedation, complication rates are approximately 1/1,000 (1/100 if dilation is performed). Because EGD allows for pathologic diagnosis and therapeutic dilation, it is often preferred as the initial procedure in suspected mechanical esophageal causes of dysphagia. However, the monocular vision provided by the endoscope is less sensitive for detecting subtle strictures than barium esophagram (Ott, Chen, Wu, Gelfand, & Munitz, 1986). Furthermore, EGD cannot provide any assessment of motility.

Subtle strictures are best found using barium esophagram with a full column technique in the prone position and can be elucidated by the use of a barium impregnated pill (typically 13 mm). In addition to its sensitivity for identifying subtle strictures, barium esophagram is useful for providing a functional assessment of the stripping wave and relaxation of the LES. This assessment can be aided by the use of a marshmallow bolus.

Esophageal manometry is typically performed using a water-perfused, multiple lumen catheter that is introduced either nasally or orally. The esophageal contraction produces a pressure on the lumen port, which is transmitted through the water-filled lumen to an external transducer and is then transmitted to a recording device (typically a computer). The ports of each lumen are located at intervals along the catheter so that multiple portions of the esophagus can be measured simultaneously during the same swallow. Solid-state probes are also available and are more precise, but more uncomfortable. This test is unsedated, and swallows are recorded with boluses of sips of water.

A trained technician is required who can recognize the manometric landmarks and artifacts in order to produce a high-quality tracing. At least 10 swallows are recorded, as many dysmotilities have variable penetrance from one swallow to the next. Manometry might indirectly indicate a possible mechanical obstruction, but it is not as accurate for detecting such abnormalities as EGD or esophagram.

A newer modality, impedance plethysmography, which measures the conductance of the surrounding medium (which distinguishes air, boluses, and esophageal wall from each other) may gain acceptance as a supplementary or alternative test since it assesses the movement of the bolus directly and can also detect gastroesophageal reflux, rumination, and other disorders.

Esophageal Dysmotilities

Achalasia

Achalasia is the classic primary esophageal dysmotility. It is characterized by incomplete relaxation of the LES. It often is associated with hypertensive resting LES pressure and aperistalsis of the esophageal body. The etiology of achalasia is unknown, but it has been associated with loss of the ganglion cells within the myenteric plexus. It presents in a wide range of ages of adulthood and affects both genders equally. Patients can develop retention of food in the esophagus with resulting regurgitation up to days after the food was ingested. Many patients report heartburn (despite the inability of the LES to relax); this is due to the poor clearance of ingested acidic substances or the bacterial fermentation of retained food. EGD might identify a dilated esophagus with retained food and increased resistance at the LES. Barium esophagram is much more sensitive, identifying aperistalsis and the classic “bird’s beak” appearance of the poorly relaxing LES. However, manometry is the gold standard for diagnosis.

Treatments for achalasia include courses of pneumatic dilations (which are performed at much higher pressures and carry a higher complication rate than the typical dilations performed for other indications), endoscopic injection of botulinum toxin into the LES to paralyze the LES for months to years, and surgical myotomy of the LES.

Pseudoachalasia has identical manometric findings as achalasia, but is due to a tumor at the gastroesophageal junction, including esophageal or gastric adenocarcinomas identifiable by EGD, as well as tumors infiltrating from outside the wall of the gut. Chagas disease also results in the same manometric findings; it is caused by Trypanosome cruzi, a protozoan infection transmitted by the bite of the reduvid bug. It is endemic to South America, but has also been reported in Texas, Florida, and the Caribbean. Chagas also typically presents with hypomotility of the duodenum, colon, or urinary tract, and dilated cardiomyopathy with cardiac conduction abnormalities.

Scleroderma

Scleroderma is the best defined secondary cause of esophageal dysmotility. This is a systemic disease resulting in atrophy and fibrosis of smooth muscle. It most commonly presents in women between the 4th and 6th decades of life. The esophageal involvement manifests as dysphagia, reflux, and heartburn due to a hypotensive LES and low amplitude distal esophagus (but normal contractions in the proximal
Nutcracker Esophagus

Nutcracker esophagus is a manometric finding characterized by elevated distal esophageal pressures (“strong enough to crack a nut”), but it also has poor temporal correlation with symptoms and has normal pathology. Patients rarely have dysphagia, but rather are evaluated for chest pain. As with other non-specific esophageal dysmotilities, the manometric findings of nutcracker are associated with gastroesophageal reflux. Awareness of this nonspecific manometric finding is helpful because patients with non-cardiac chest pain often get labeled with nutcracker by cardiologists, emergency room physicians, or other health care providers without performing esophageal manometry. Treatment should primarily be aimed at coexistent gastroesophageal reflux, if present. Calcium channel antagonists or nitrates might be beneficial in some patients, but the data is sparse.

Gastroesophageal Reflux Disease

Gastroesophageal reflux disease or GERD, which primarily manifests with heartburn (a retrosternal burning sensation) and/or reflux (effortless regurgitation of stomach contents), can also co-manifest with dysphagia. The dysphagia can be due to a mechanical obstruction which is the sequella of long-standing GERD (see below), or due to dysmotility.

As mentioned above, GERD is associated with a number of non-specific manometric abnormalities. In addition to nutcracker, GERD may be associated with low amplitude distal contractions or aperistalsis. In contrast to achalasia, patients with GERD tend to have a hypotensive LES, particularly if there is severe erosive esophagitis. Treatment is aimed primarily at GERD, but the dysphagia may persist even after adequate control of GERD.

Mechanical Causes

The differential diagnosis of mechanical causes of dysphagia are broad (Table 2 on page 5). The most common and significant causes are discussed here.

Schatzki’s Rings

A Schatzki’s rings are a benign, thin, solitary, circumferential mucosal shelf located just proximal to the gastroesophageal junction. They are usually associated with a hiatal hernia and symptoms of gastroesophageal reflux. They account for approximately 20% of esophageal etiologies of dysphagia. Dilation, usually after EGD while under conscious sedation, is effective, but the rings recur in the majority of patients within 5 years. Treatment of GERD with proton pump inhibitors, even if the GERD symptoms are only mild, may prevent this relapse.

Esophageal Cancer

This is the most feared cause of dysphagia, as the long-term survival of esophageal cancer is poor. Squamous cell carcinoma, which has the same risk factors as squamous cell carcinoma of the head and neck (male gender, increased age, tobacco, alcohol, and low socioeconomic status), is usually located in the proximal or mid-esophagus. Esophageal adenocarcinoma is rapidly increasing in incidence and is now more common than squamous cell in the United States. It is typically located in the distal esophagus and is associated with gastroesophageal reflux and obesity, as well as male gender, white race, and increasing age.
The accepted premalignant lesion of esophageal adenocarcinoma is Barrett’s esophagus, a change in the lining of the distal esophagus from the normal squamous mucosa to a specialized columnar mucosa similar to that found in the small intestine. Barrett’s esophagus does not cause symptoms and cannot be identified by barium esophagram. Endoscopic screening for Barrett’s esophagus with the use of EGD is controversial due to the rarity of the cancer from a population perspective, and the unproven ability to reduce mortality with the use of screening (Sharma et al., 2004).

**Eosinophilic Esophagitis**

Eosinophilic esophagitis presents most commonly in boys or young men, often with a personal or family history of asthma, atopic dermatitis, or seasonal allergies. Adult patients usually present with intermittent dysphagia for solids and sometimes with unexplained chest pain. Children commonly present with vomiting. The diagnosis is rapidly becoming more common (Cherian, Smith, & Forbes, 2006), but this is at least partly due to increased recognition by gastroenterologists and pathologists. Barium esophagram shows a narrowed, corrugated esophagus. EGD usually discloses multiple subtle rings throughout the esophagus, often with long, linear furrows, but the EGD might be entirely normal. Mucosal biopsies show intense inflammation with eosinophils.

The etiology is unknown, but it may be due to ingested allergens, airborne allergens, or an atypical inflammatory response to acid reflux, as nearly 50% of adult patients may have documented reflux by ambulatory pH probe (Remedios, Camp-bell, Jones, & Kerlin, 2006). A recent randomized, controlled trial in children found that swallowed, high-dose topical steroids administered from inhalers typically used for asthma resulted in histologic remission in 50% of subjects compared to 9% receiving placebo (Konikoff et al., 2006).

**Webs**

Webs are subtle, wispy, non-circumferential mucosal abnormalities, typically found in the cervical esophagus. Diagnosis is aided with the ingestion of a barium tablet on esophagram. They can be missed on EGD or may be incidentally ruptured with the initial intubation of the endoscope beyond the upper esophageal sphincter. Historically, cervical webs had been anecdotally noted among women with iron deficiency anemia (Plummer-Vinson syndrome), but no such association was found in a well designed study (Elwood, Jacobs, Pitman, & Entwistle, 1964). Treatment is dilation.

**Peptic Strictures**

Before the advent of potent acid suppressing therapies, peptic strictures were a common presentation of GERD. Now they are most common among patients with barriers to access to medical care (primarily the uninsured) or medically noncompliant patients (particularly patients with psychiatric disease). Peptic strictures are most common in the distal esophagus. Treatment is with dilation and high dose proton pump inhibitors. Multiple dilations may be required. EGD with biopsies is required to exclude malignancy.

**Conclusion**

Esophageal causes of dysphagia are many and varied. Patient localization inferior to the level of the larynx is the most important clue in distinguishing from oropharyngeal dysphagia. Occasional patients can present with multiple etiologies (i.e., residual stroke causing oropharyngeal dysphagia in addition to a new esophageal cancer). Patients evaluated by SLPs whose findings do not fully explain the type or severity of symptoms should be referred to a gastroenterologist for further evaluation.

Joel Rubenstein is a clinical lecturer in gastroenterology at the University of Michigan, and a Staff Physician at the Ann Arbor Veterans Affairs Medical Center. His clinical focus is in diseases of the esophagus, and his research focuses primarily on Barrett’s esophagus and esophageal adenocarcinoma using outcomes and translational methods. He may be contacted at jhr@med.umich.edu.

**References**


Management of Esophageal Dysphagia: The Otolaryngologist's Perspective Updated

Janet A Wilson
Newcastle University
Newcastle upon Tyne, United Kingdom

It is well known that any patient who presents with cervical level dysphagia may in fact turn out to have a lesion at a lower level. Thus, every otolaryngologist assessing an obstructive type dysphagia will bear esophageal diseases in mind. The aim of this article is to review the latest thinking on the more commonly encountered scenarios and the relevant evidence base.

Emergency Presentations
Foreign bodies: Beyond the sword swallowers. Over 80% of esophageal foreign bodies are affected in the cervical esophagus and present, therefore, little difficulty to the otolaryngologist. The management of pediatric esophageal impactions will often be determined by local strategy worked out in conjunction with the pediatric thoracic department. What is the current thinking on non-instrumental therapy?

A recent survey of ENT surgeons suggested that 95% undertake a preliminary trial of antispasmodic drugs such as hyoscine, but there is no supporting evidence for this consensus (Price, Jones, & Montgomery, 2007). Recent in vitro evidence, on the other hand, offers some support for the use of pineapple juice (Thomas et al., 2004), and glucagon may offer a reasonable alternative (Nguyen & Holloway, 2005). Increasingly, otolaryngologists propose flexible endoscope approaches (whether pull back or push through) performed either by themselves or by a gastrointestinal colleague for suspected lower third bolus impactions.

Esophageal rupture. At least half of esophageal perforations are iatrogenic, and so the diagnosis may be suspected from the outset. Spontaneous post-emetic rupture is likely to be a delayed diagnosis and the resulting mediastinitis has a 30% mortality even when treated. Suspicion should be aroused by post-emetic pain and fever. Water soluble contrast esophagography establishes the diagnosis.

Endoscopy: Opportunities for the Otolaryngologist
The big picture: Esophageal manifestations of GERD. In the confusion and side-taking of the debates on the importance of supra- or extra-esophageal manifestations of gastroesophageal reflux disease (GERD), it is easy for the otolaryngologist to forget that typical GERD remains incontrovertibly one of the most common Western diseases—the so called disorder of the third millennium, affecting at least 10% of the population of the USA, and with much higher prevalence in older age groups. The European incidence of reflux esophagitis has almost doubled in the last 10 years (Loffeld, 2006). Similarly, the fashion for attribution of a huge gamut of symptoms to GERD may overlook the fact that a degree of GER is physiological, not least in the postprandial phase. There are three principal GERD dysphagia mechanisms: esophagitis, stricture, and acid induced dysmotility.

Dysphagia is often the sole symptom of severe esophagitis: It is easy to forget that those with the most troublesome heartburn have less severe esophagitis. The optimum strategies for long term maintenance therapy have massive financial implications for the West, but very few guidelines (Loffeld, 2006). Eosinophilic esophagitis is a steroid responsive variant with a dysphagia incidence >90%. PPI therapy is the most effective therapy in esophagitis but H2Receptor antagonist therapy is also superior to placebo (Khan, Santana, Donnellan, Preston, & Moayyedi, 2007).

Stricture is both the rarest and the hardest to discriminate clinically from neoplastic obstruction. Fewer than 5% of those with severe esophagitis will develop stricture (Hoang, Koh, & Maddaus, 2005). Typically, those with pathologic reflux have a high body mass index, in contrast to the weight loss of the stricture patient. Some strictures are amenable to balloon dilatation, including those rare variants due to bullous dermatoses (Okada et al., 2006).

With regard to acid induced dysmotility, as well as dysphagia, impaired motor function can lead to nocturnal choking. The tonic upper esophageal sphincter tone normally drops during sleep, and any distension of the esophagus by refluxate should cause a reflex increase in this tone. Where the barrier is insufficient, however, the patient may abruptly awake from sleep. The differential diagnosis is obstructive sleep apnoea or nocturnal panic disorder.

Before treating GERD, it is useful to consider various treatment strategies.

Endoscopy. For squamous cancer, the risk factors in countries such as the USA are very similar to those of head and neck cancer—namely smoking and alcohol. The occurrence of adenocarcinoma relates to gene expression profiles (Siersema, 2007)—perhaps explaining why Asian countries such as China have such very different incidence patterns from the West (i.e., squamous cancer is still very much more common than adenocarcinoma). United Kingdom Department of Health dyspepsia guidelines for family practitioners advise empiric treatment with anti-secretory medications and referral for endoscopy only in the presence of alarm symptoms. The efficacy of proton pump inhibition makes the treatment a useful therapeutic trial.

Conversely, however, review of 4,018 subjects who underwent open access gastroscopy from 1990 to 1998 identified esophagogastric carcinoma in 123 (3%). Of these 123 patients, 104 (85%) with esophageo-gastric cancer had "alarm" symptoms (anemia, mass, dysphagia, weight loss, vomiting) and would
The reported increased risk of patients with dyspepsia may be treated without investigation. Twice daily National guidelines now suggest that new presentations of dyspepsia and other problems, but good evidence of therapeutic response to lifestyle change is remarkably scant. U.K. medical intervention for dyspepsia.

**H. Pylori screening.** Helicobacter pylori “test and treat” has been recommended for the management of young dyspeptic patients without alarm symptoms. A meta-analysis of five trials including 1924 patients compared endoscopy with H. pylori “test and treat.” Prompt endoscopy was found to confer a small benefit in curing dyspepsia but was more expensive than “test and treat” and thus not a cost-effective strategy (Ford et al., 2005). Antimicrobial resistance has decreased eradication rates for Helicobacter pylori infection worldwide. A 10-day sequence—40 mg of pantoprazole and 1 g of amoxicillin for 5 days, followed by 40 mg of pantoprazole, 500 mg of clarithromycin, and 500 mg of tinidazole, each twice daily for 5 days—had a significantly greater eradication than standard treatment, particularly for clarithro-mycin-resistant strains (Vaira et al., 2007). Conversely, in H. pylori-positive patients with non-ulcer dyspepsia, there appears little added benefit of eradication of the bacterium over proton pump inhibitor therapy alone (Mazzoleni et al., 2006). Early investigation by endoscopy or H. pylori testing may benefit some patients with dyspepsia but is not overall a cost-effective strategy (Delaney, Ford, Forman, Moayyedi, & Qume, 2005).

**Lower esophageal manometry.** Achalasia, although rare (1: 100,000), remains an important diagnosis to be excluded in patients under 50 years old presenting with heartburn and dysphagia. The dysphagia is typically associated with a degree of pain and weight loss, may be exacerbated by stress, and alleviated by carbonated beverages (Woltman, Pellegrini, & Oelschla-ger, 2005). Surgical myotomy, typically botulinum toxin injection, pneumatic dilatation, and surgery, gave the shortest lived, least durable response followed by achalasia management showed that pharmacologic therapy was a once-in-a-lifetime approach. (Despite the long recognition of nocturnal acid breakthrough, many patients are still offered primary once daily, morning therapy.)

**Polysonomography.** This might be valuable not only in order to differentiate GER choking episodes from those of sleep apnea, but also because the two conditions share an association, although which is the primary issue and which secondary is unclear. Swallow symptoms may also arise due to morning swelling of the uvula, exacerbated by the dry mouth of sleep breathing disturbance.

**Danger Scenarios**

**Barrett’s esophagus.** Making the diagnosis and mapping the extent of Barrett’s intestinal metaplasia of the squamous epithelium is relevant to a small minority of GERD patients. The risk of adenocarcinoma is increased in Barrett’s metaplasia, but the size of the risk and hence the optimum intensity of cancer screening are unknown (Garside et al., 2006). The least aggressive adequate strategy may be a once-in-a-lifetime screen of males with GERD for cancer, followed by further surveillance only of those with dysplasia. Gene microarray analysis appears able to distinguish adenocarcinoma from Barrett’s phenotype—the presence of p53 abnormalities in Barrett’s mucosa. DNA abnormalities on flow cytometry may also be useful in identifying those patients with Barrett’s who are at the highest risk for cancer development (Kwong, 2006).

**Cancer.** Adenocarcinoma of the gastric cardia has one of the fastest growing incidences of any western tumor (Garside et al., 2006) and since 1994 has exceeded that of squamous esophageal cancer. Recurrent disease post resection is very common such that the 3 year survival is between 6 and 40%. Improved survival is, however, noted in patients with a near complete response to induction chemotherapy and may be predicted by fluoro-deoxyglucose PET scanning (Siersema, 2007). CT-PET performed after neoadjuvant therapy in patients with potentially resectable esophageal carcinoma is important for detecting interval metastases that preclude surgical resection (Komaki, Fukami, & Erasmus, 2007).

**Achalasia.** The reported increased risk of patients with achalasia developing a squamous cell carcinoma varies from 0 to 140 times that of the normal population. In addition, achalasia may predispose to Barrett’s metaplasia. Surveillance endoscopy can be problematic due to food obscuring the epithelium and hyperplastic changes of the mucosa. The optimum surveillance technique and time interval remain to be determined (Leeuwenburgh et al., 2006). A 40-year review of achalasia management showed that pharmacologic therapy gave the shortest lived, least durable response followed by botulinum toxin injection, pneumatic dilatation, and surgery, respectively (Bonavina, 2006; Lake & Wong, 2006; Leyden, Moss, & MacMathuna, 2006). Surgical myotomy, typically laparo-scopic, perhaps under intra-operative manometric control, requires extension of the incision 3 cm onto the stomach, with a partial fundoplication to reduce the incidence of postoperative reflux.
**Surgical Causes**

**Fundoplication.** Dysphagia is a common long-term symptom following fundoplication (Papasavas, 2005), and recent laparoscopic variants thus attempt a partial fundal wrap to reduce the hiatal obstruction (Hayden & Jamieson, 2006). The most common cause of failure is transdiaphragmatic prolapse of the wrap. Although endoscopic procedures are less effective than surgical approaches, the avoidance of general anaesthesia and the reduced complication rate make them a realistic intermediate option for some patients (Yeh & Triadafil-lo-poulos, 2005). There are three types of endoscopic antireflux procedures: endoscopic suturing, radiofrequency, or bulking. Suturing represents the most technically demanding and its precise method of action is unknown, but it does appear to increase lower esophageal sphincter pressure. The Stretta radiofrequency system delivers up to 5W energy down each of 4 probes. Bulking agents include biopolymer. All endoscopic reports suffer from relatively small numbers and high dropout rates.

**Resection.** Esophagogastrectomy remains a “formidable” operation (Atkins et al., 2004), with > 5% mortality and a 64% complication rate. The quantification of dysphagia following esophageal resection is very important, as postoperative pneumonia appears the only postoperative complication significantly to affect prognosis. Acceptable pharyngeal function and airway protection should thus be established before oral intake is resumed. Minimally invasive esophageal resection has the theoretical advantage of reduced postoperative complications compared with open resection. However, in one small series, postoperative events included laryngeal penetration, vocal cord paralysis, and/or aspiration in three patients, two of whom experienced severe respiratory complications. These problems are probably due to transient denervation of the pharynx and laryngeal structures. Thus, postoperative aggressive pulmonary toilet and aspiration precautions and serial evaluation of swallow safety are required (Atkins, Fortes, & Watkins, 2007). Self expanding metal stents are used for palliation (Ferrante, Feliziani, Imperatori, Ferraris, & Bernasconi, 2006; Zanca et al., 2006), but may be less effective than chemotherapy.

**Laryngeal innervation.** The recurrent laryngeal nerve may be bruised or severed during the course of esophagectomy. Isolated lateralization of the vocal cord does not of itself cause dysphagia, but older patients and those with other swallow impairments may demonstrate aspiration. Where the integrity of the nerve is not known to have been surgically disrupted, the decision whether to wait a full 12 months postoperatively or to offer earlier medialization or temporary medialization, must be patient lead and informed by the symptom severity and the day-to-day vocal demands.

**Watch This Space**

Weight loss and head of bed elevation appear to be effective lifestyle interventions for GERD. There is no evidence supporting improvement after cessation of tobacco, alcohol, or other dietary interventions (Kaltenbach, Crockett, & Gerson, 2006).

Gastroesophageal reflux disease (GERD) is said to be the causative factor in up to 41% of adults with chronic cough. However, cough and GERD are common ailments, and their co-existence by chance is high. Also, cough can induce reflux episodes. A recent systematic review of the role of proton pump inhibitors for cough associated with GERD in adults showed a small beneficial effect only in sub-analysis. The authors advise clinicians to be aware of the natural resolution with time and the placebo effect in studies using cough as an outcome measure (Chang, Lasser-son, Gaffney, Connor, & Garske, 2006). In terms of laryngeal signs, a recent study found no evidence of a therapeutic benefit of twice daily esomeprazole 40 mg for 16 weeks compared with placebo for signs or symptoms of posterior laryngitis (Vaezi et al., 2006). Acid reflux is a common problem, and is thought to occur in 4–10% of patients presenting to ENT clinics. There is, however, no evidence to support the custom of empirical use of anti-reflux therapy for hoarseness (Hopkins, Yousaf, & Pedersen, 2006). Nonetheless, GERD is among the most common causes of dysphagia, and appropriate enquiry should always be made when dysphagia is a major symptom.

Janet Wilson is professor of Otolaryngology, Head & Neck Surgery at Newcastle University. She has published numerous peer-reviewed publications in the area of dysphagia. Her current work centers on the extra-oesophageal manifestations of gastroesophageal reflux, the mechanisms underlying recurrent acute tonsillitis and the role of cognitive behaviour therapy in medically unexplained symptoms such as globus, dysphonia and catarrh.

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Fluoroscopic Assessment of Dysphagia: Which Radiological Procedure is Best for Your Patient?

Jo Puntit Sheltman
Dixie Regional Medical Center
St. George UT

Dysphagia is defined as the subjective awareness of swallowing difficulty during passage of a solid or liquid bolus from the mouth to the stomach. This symptom can be caused by functional or structural abnormalities of the oral cavity, pharynx, esophagus, or even the gastric cardia. A barium study may be performed with videofluoroscopy to assess oral-pharyngeal function and esophageal motility as well as a series of double and single contrast static images to assess structural abnormalities such as rings, strictures and tumors (Levine & Rubesin, 1990). The best procedure for any patient with dysphagia is a tailored examination for the purpose of defining the problem and subsequent treatment regime. Speech-language pathologists (SLPs) need to understand that swallowing is a dynamic process from the lips to the stomach and all phases are interrelated. If visualization is stopped at the upper esophageal sphincter, how does one know if the food or fluid reached the stomach?

The choice of which test or combination of tests to use is key to helping patients with dysphagia obtain answers to their discomfort or localization of dysphagia. These patients can subjectively localize a sensation of blockage or discomfort to the throat or substernal region. Patients with pharyngeal dysphagia typically complain of food sticking in the throat or of a globus sensation with a lump in the throat. Other symptoms of oropharyngeal dysfunction include coughing or choking during swallowing. When oropharyngeal dysphagia has an attributable cause (e.g., recent stroke), a modified barium swallow (MBS) may be the appropriate test to assess the patient’s swallowing status and initiate treatment by a SLP. For patients with unexplained oropharyngeal dysphagia, however, a more detailed barium study may be needed to determine the cause.

It also is important to recognize that abnormalities of mid or distal esophagus or even the gastric cardia may cause referred dysphagia to the upper chest or pharynx, whereas abnormalities of the pharynx rarely cause referred dysphagia to the lower chest. “The esophagus and cardia should therefore be evaluated in patients with pharyngeal symptoms, particularly if no abnormalities are found in the pharynx to explain these symptoms. Thus, a combined radiologic examination of the pharynx, esophagus, and gastric cardia is appropriate for patients with unexplained dysphagia” (Levine & Rubesin, 1990, p. 35). Other studies show that simultaneous disorders of the pharynx and esophagus are so frequent that the complete swallowing chain should be examined in all patients with dysphagia (Jones, 1985).

Swallowing initiates a primary peristaltic sequence that traverses the pharynx and esophagus. The act of swallowing is often divided into three phases. The preparatory phase involves the ingestion of food or fluid, chewing, and lubrication with saliva. The tongue then forms the food or fluid into a discrete bolus, propelling it to the posterior oral cavity. The pharyngeal phase is a reflexive, rapid, and highly coordinated event in which the bolus is propelled from the tongue to the esophagus. Very little barium is needed to view the function of the pharynx. The propulsion of food from the mouth to the stomach is the essential function of the esophagus.

The esophagus is a collapsed tube that needs to be filled with barium to view the peristaltic activity. The esophageal phase involves the transit of ingested materials from the upper esophageal sphincter into the stomach. The peristaltic sequence consists of a rapid wave of neurologic inhibition, followed by a slower wave of muscular contraction, which is propagated down the length of the esophagus. The peristaltic wave moves rapidly through the pharynx at 10-25 cm/sec and slows in the esophagus to a velocity of 2-4 cm/sec. The peristaltic wave propagates through the esophageal body in 6-10 secs. Lower esophageal sphincter (LES) relaxation terminates when the peristaltic contraction reaches the lower sphincter (Goyal, Prasad, & Chang, 2004).

It is essential to view the esophagus in the upright and supine positions to assess motility with and without gravity. There are discreet differences for each barium procedure, and it is necessary to understand in each order to ascertain which study is best for the patient with dysphagia.

**Modified Barium Swallow**

Indications for use of the modified barium swallow are dysphagia, suspected aspiration, odyno-phagia, and trauma. Primary evaluators are both the SLP and Radiologist, or Radiology PA.

Most SLPs are aware of the examination for dysphagia developed by Dr. Jeri Logemann in the 1970s. This procedure has evolved over time from the delivery of 1/3 teaspoon of liquid and solid foods to assessing functional amounts of food and fluid and providing compensatory strategies to reduce or eliminate aspiration to aid in the assessment. Normally the examination includes the use of a variety of thin barium, more viscous barium, and an assortment of foods mixed with barium paste. The purpose is to ensure that the patient transfers the bolus through the oral, pharyngeal and esophageal phases in a safe, timely manner. Transport of the bolus through the reflexive phases (pharyngeal and esophageal) should not take more than 10 seconds.

The examination procedure follows.

1. Record the entire exam for later review.
2. Position the patient in the upright lateral position. The patient can either stand or use a special chair for comfort.
if unable to stand.

3. The fluoroscope is positioned initially at the mouth and pharynx. Deliver the initial amount of thin liquids in controlled amounts for safety and to visualize the safety of the swallow.

4. Observe the quality of oral transfer, any nasopharyngeal reflux, possible pooling in the hypopharynx, laryngeal penetration and or aspiration.

5. If there is any pooling in the valleculae or pyriform sinuses one may want to visualize the patient in the anterior posterior view for symmetry of the swallow.

6. If the patient is able to tolerate controlled small amounts without aspiration, allow him or her to (independently if possible) drink more functional bolus sizes as well as sequential swallows (with and without a straw).

7. Visualize the oral pharyngeal phases of the swallow for transfer throughout both areas for safety.

8. Visualize the bolus through the esophagus (with gravity assist) to make sure the bolus passes through the LES in a timely manner.

9. Subsequent swallows follow the same sequence, except that different food substances are used. Commonly presented foods are a puree and chewable consistencies mixed with dense barium.

10. It is important to visualize the bolus transfer from the mouth through the pharynx and into to esophagus for safety and timing of the bolus.

11. It is common to watch the variety of boluses through the entire esophageal phase to make sure the bolus passes through the LES (with gravity assist). In some institutions this is called an esophageal screen to watch the transport of the bolus from the mouth through the LES.

12. If there are motility problems in the esophagus, an esophagram may be needed to assess the lumen.

**Combination of the MBS and Esophagram**

The evaluators are the SLP and Radiologist or Radiology PA. This is an evaluation of patients with dysphagia in whom a combination of oral/pharyngeal and esophageal motor disorder is of concern. This test requires the examination from the mouth to the gastric cardia. Emphasis is placed on the tubular esophagus, but depending on specific symptoms, the examination should ideally be tailored appropriately. A combination of the double contrast esophagram and the MBS is the most comprehensive procedure. Overnight fasting is preferable to optimize mucosal coating, which is compromised by fluid and secretions.

1. With the patient in erect lateral position a single mouthful of high-density barium is swallowed and video recordings are obtained.

2. If there is no significant abnormality seen in the oro- or hypopharynx, the patient is then turned to a standing left posterior oblique (LPO) position and is asked to drink the remainder of the cup of high-density barium rapidly. Double contrast views of the esophagus from the thoracic inlet to the gastroesophageal junction are obtained for morphologic assessment of the esophageal mucosa.

3. An A/P viewing of the oro-pharynx should be obtained to view symmetry of the swallow.

4. A barium pill may be given followed by water or liquid barium to view motility of the bolus throughout the entire oral, pharyngeal, esophageal phases of the swallow to check for strictures or obstruction.

5. The fluoroscopic table is brought to a horizontal position and the patient position in the right anterior oblique (RAO) position. The patient is then asked to sip barium through a straw. Wait for an entire bolus to pass through the LES.

6. Esophageal motility is assessed from the initiation of the swallow through the gastroesophageal junction. The dynamic aspect of the video esophagram is used to evaluate peristalsis, functional abnormalities, and gastroesophageal reflux. The presence or absence of normal peristalsis, delay of transit, and tertiary contraction, and retention of contrast are specifically evaluated for each swallow.

7. Then have the patient drink sequential swallows and obtain spot films of the entire distended esophagus and open gastroesophageal junction.

8. Turn the patient supine to coat the fundus then back to right posterior oblique to evaluate for GER and obtain spot films of the gastric cardia and fundus.

9. Barium pill/marshmallow or semisolid food may be given at this point (if not provided earlier; Goyal et al., 2004).

**Double Contrast Esophagram: DCE**

Radiological examination of esophageal motility should include a systematic evaluation of motor function in the pharynx, both esophageal sphincters, and the esophageal body. In motility studies, both esophageal sphincters are evaluated for evidence of abnormal opening or incompetency and esophageal body is studied for peristaltic and nonperistaltic activity, emptying and caliber. The patient needs to have an empty stomach for this procedure.

Indications are dysphagia, globus sensation, early mucosal disease (erosions, ulcers, polyps, tumors, inflammation, infection). Primary evaluators are the Radiologist or Radiology PA.

The basis of double-contrast esophagram is the distention of the esophagus with gas and coating it with high-density barium (approximately 200–250% W/V) that has excellent mucosal-coating properties. The gas used is carbon dioxide formed from commercially available effervescent agents as well as any swallowed air.

1. The fluoroscopic table starts in the upright position, with the patient being placed in the LPO. This is done to
move the esophagus off the thoracic spine. The patient takes 1-2 swallows to ascertain tolerance of the dense barium.

2. The radiologist places the gas-forming granules mixed with 15ml of water into the patient’s mouth and then asks the patient to drink the fluid immediately while it effervesces.

3. Immediately thereafter, the patient drinks high-density barium taking multiple rapid sequential swallows. Due to the gaseous distention of the esophagus, peristalsis will be inhibited and allow the esophagus to be coated with the barium mixture.

4. Pictures are obtained of the proximal, thoracic and distal esophagus.

5. Then the patient participates in a single contrast esophagram.

**Single Contrast Esophagram**

Indications are dysphagia, odynophagia, globus, retrosternal discomfort, evaluation of masses, vascular rings/slings, webs, strictures or aberrant anatomy and evaluation of esophageal motility. The primary evaluator is the Radiologist or Radiology PA.

Examination of the esophagus with barium only can be done as the sole study or combined with the DCE. The use of single contrast or double contrast esophagram or a combination of the two examinations is considered to be the best method to evaluate for gastroesophageal reflux. The barium used is of a lower density (approximately 35–85% W/V) than that used for DCE. Again, the patient needs to have an empty stomach for this procedure.

1. With the patient placed upright in the LPO position, fluoroscopy of the esophagus is obtained following sequential swallows of barium.

2. Then the patient is placed in the recumbent prone RAO position.

3. During continuous drinking of the barium so that the esophagus is distended obtain images of the proximal, thoracic, and distal esophagus. Views of the distal esophagus may be taken in suspended respiration in order to accentuate the presence of an issue at the LES (Valsalva maneuver to look for a hiatal hernia or Schatzki’s ring).

4. After the bolus has passed through the esophagus, collapsed views of the esophageal mucosa may be obtained for evaluation of the esophageal fold pattern.

5. The procedure at this point can vary. Have the patient roll to his or her back and on to his or her left side and back again. Views of the LES are performed to monitor for reflux. At times water will be provided to ascertain reflux.

6. If an esophageal stricture is identified, having the patient swallow a 13mm barium pill in the upright position can perform further characterization. Document the position of the pill if it cannot be passed beyond the stricture, making sure to capture the image when barium can be seen on both sides of the stricture. A retained pill will dissolve and pass through the gastroesophageal junction in 20-30 minutes.

**Summary**

The esophagrams, both single and double contrasts, are completed when the patient has an empty stomach. The radiologist is primarily looking at the lumen of the esophagus and ascertaining if there are any structural or motility disorders. This procedure is best visualized when the patient ingests gas crystals to dilate the esophagus and to use dense barium to cling to the lumen. A MBS is completed to ascertain oral and pharyngeal safety and esophageal motility issues. The SLP should observe the bolus transport from the oral cavity through the LES. The bolus should transport through these phases in a timely, safe manner with gravity assist and without aspiration. If the patient demonstrates disorders in the oral and or pharyngeal cavity, the SLP must decide which postures, positions, or compensatory strategies are needed to reduce or eliminate the difficulties. The purpose of the MBS is to try and emulate what a patient eats during a meal in functional measures if possible.

Jo Puntil Sheltman has worked in the field of dysphagia for 25 years and has lectured extensively regarding critical care issues for 20 years. She is currently on the Board for Specialty Recognition in Swallowing and Swallowing Disorders. Ms. Puntil Sheltman currently works in southern Utah in the adult and neonatal intensive care units. She may be contacted at jo.puntit@ihc.com.

**References**


### Treatment of Dysphagic Symptoms Resulting From Esophageal Disorders

**Benson T. Massey**  
GI Manometry Laboratory, Division of Gastroenterology and Hepatology  
Medical College of Wisconsin  
Milwaukee, WI

**Introduction**

The speech-language pathologist (SLP) needs to remain aware of the dysphagic symptoms that result from esophageal disorders for several reasons:

- Often, it is not possible to determine on the basis of history and physical examination whether the symptoms result from an oropharyngeal or an esophageal disorder.
- Oropharyngeal and esophageal disorders can coexist, further confounding management.
- During the course of evaluating a patient the SLP may uncover evidence of an esophageal disorder and be in a position to recommend appropriate referral.
- Certain treatments for oropharyngeal conditions can exacerbate problems of esophageal dysphagia; the converse is also possible.
- Finally, recognition of the presence of an esophageal problem may help lead to the diagnosis of a previously unsuspected systemic disease, some of which may have implications for oropharyngeal function.

The most challenging aspect of treating dysphagia from esophageal disorders is often establishing the correct diagnosis in the first place. Although diagnostic testing is not the focus of this article, the SLP needs to be cognizant of the fact that radiographic and endoscopic evaluations individually are often insufficient to detect subtle structural and motor disorders, and that more advanced testing with manometry, pH monitoring, and/or esophageal impedance testing may be necessary. Moreover, these tests are very operator-dependent in their diagnostic accuracy.

Esophageal disorders resulting in swallowing difficulty are often classified as falling into one of two major categories: structural or motor disorders. This dichotomy is not perfect, as some diseases can have elements of both disorders. A typical example is severe peptic esophagitis with both a stricture and failure of peristaltic function. Classically, it is taught that the two categories can be distinguished by the fact that structural disorders have dysphagia for solids, whereas motor disorders have dysphagia for both solids and liquids. However, patients with a severe structural stenosis can also have difficulties with fluids, and patients with early esophageal achalasia often report more difficulties with solids than liquids.

**Dietary Modifications**

Until the esophageal disorder can be effectively treated, patients will require dietary modifications. Essentially all disorders benefit from the patient remaining in an upright position during eating and drinking, as well as for some time after ingestion, to allow the action of gravity to facilitate esophageal emptying. Patients with structural disorders need to chew their food carefully and use liquids liberally to help wash these down. Patients with esophageal dysmotility and significant esophageal retention may require more frequent and smaller meals. Patients with spastic disorders often report more difficulty with cold beverages and may have improvement in symptoms with hot (not scalding) beverages (Triadafilopoulos, Tsang, & Segall, 1998).

Patients with dilated, flaccid esophageal bodies should avoid gummy, glutinous foods that adhere to the esophageal wall. They may benefit from the use of carbonated beverages to help “flush out” the lumen, so long as there is not esophageal outflow obstruction. If the patient has a coexisting oropharyngeal disorder, some of these recommendations may run counter to what the SLP might recommend for that disorder, and reconciliation between the different specialties will be needed to optimize care for the patient.

### Table 1. Intrinsic Structural Esophageal Disorders

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Example</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammatory</td>
<td>Peptic structure from GERD</td>
<td>Acid suppression, anti-reflux surgery</td>
</tr>
<tr>
<td></td>
<td>Eosinophilic esophagitis</td>
<td>Elimination diet, topical steroids</td>
</tr>
<tr>
<td></td>
<td>Candida esophagitis +/- pseudo-diverticulosis</td>
<td>Treat infection, dilation, esophageal replacement</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Esophageal cancer (primary/ metastatic)</td>
<td>Resection, esophageal replacement, chemoradiation, dilation, tumor debulking, esophageal stent</td>
</tr>
<tr>
<td></td>
<td>Leiomyoma</td>
<td>Resection</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Pill injury</td>
<td>Stop/change medication, dilation</td>
</tr>
<tr>
<td></td>
<td>Radiation</td>
<td>Dilation, steroid injection</td>
</tr>
<tr>
<td></td>
<td>Caustic injection</td>
<td>Dilation, steroid injection, esophageal replacement</td>
</tr>
<tr>
<td></td>
<td>Anastomotic stricture</td>
<td>Dilation, steroid injection</td>
</tr>
<tr>
<td>Congenital</td>
<td>Congenital webs/rings</td>
<td>Dilation</td>
</tr>
<tr>
<td></td>
<td>Congenital esophageal stenosis/ atresia</td>
<td>Dilation, surgical repair, surgical esophageal reconstruction</td>
</tr>
<tr>
<td></td>
<td>Tracheoesophageal fistula</td>
<td>Surgical repair, surgical esophageal reconstruction</td>
</tr>
</tbody>
</table>
Esophageal Dysphagia: Diagnosis and Treatment Options

### Table 2. Extrinsic Structural Esophageal Disorders

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Example</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammatory</td>
<td>Pancreatitis with pseudocyst compression</td>
<td>Drain pseudocyst</td>
</tr>
<tr>
<td></td>
<td>Sarcoidosis with inflammatory adenopathy in mediastinum</td>
<td>Systemic corticosteroids</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Lung cancer</td>
<td>Chemoradiation</td>
</tr>
<tr>
<td></td>
<td>Lymphoma</td>
<td>Chemoradiation</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Tight, slipped, or herniated duplication</td>
<td>Dilation, surgical repair</td>
</tr>
<tr>
<td>Congenital</td>
<td>Vascular compression (dysphagia lusoria)</td>
<td>Surgical correction</td>
</tr>
</tbody>
</table>

Some patients may have a significant delay in achieving satisfactory treatment for their disorder or have a disorder that cannot be effectively treated, either due to the severity of the disorder or because co-morbid illness makes the treatment impossible. These patients may require temporary or permanent tube feedings, via nasogastric, gastrostomy, or jejunostomy tubes.

**Structural Disorders**

For structural esophageal disorders, a useful sub-classification is whether the disorder in intrinsic to the esophageal wall or arises from a pathophysiologic process extrinsic to the esophagus that acts to compress or encase the esophagus and prevent adequate opening of the esophageal lumen. Intrinsic processes are far more common and are more readily identified by the standard diagnostic modalities of endoscopy and/or barium esophagrams. Because of their rarity, extrinsic lesions are often overlooked, especially since they produce no mucosal abnormalities that can be recognized by the endoscope. Radiology will also fail to pick up what may be a subtle stenosis, if the patient is not given sufficiently large barium volume or solid bolus challenges. Imaging modalities that look extrinsically, such as CT/MRI scans or endosonography, are usually required to detect these disorders.

Structural esophageal disorders can also be classified by the underlying pathogenesis, such as whether the disorder is inflammatory, neoplastic, iatrogenic, or congenital. Examples of extrinsic and intrinsic structural esophageal disorders are given in Tables 1 and 2.

These categories are not mutually exclusive for the individual patient. Moreover, the presence of some disorders predisposes the patient to develop others, the prime example being the development of esophageal adenocarcinoma in a patient who already has a reflux induced stricture associated with Barrett’s esophagus. All patients with structural disorders are predisposed to developing further injury by medications that can damage the esophagus when they lodge and dissolve in the region of stenosis. Treatment of some structural disorders can give rise to others. For example, topical steroid treatment of eosinophilic esophagitis can result in Candida infection of the esophagus. These patients can also develop oral thrush, so that the treatment of the esophageal disorder can also have consequences for oropharyngeal deglutition.

Among the large number of structural disorders, two warrant particular mention because they are frequently unrecognized or underappreciated. Eosinophilic esophagitis is an allergic disorder that was uncommonly recognized before 1990, but is likely increasing in incidence (Straumann & Simon, 2005). The presence of eosinophilic esophagitis is often not recognized because the endoscopist and radiologist fail to recognize the classic (corrugations, mucosal white spots) or subtle (narrow caliber lumen) features of the disease (Potter et al., 2004; Vasilopoulos et al., 2002), the endoscopist fails to obtain mucosal biopsies, and/or the pathologist fails to recognize that the number of eosinophils in the pathology specimens is more than is typical for GERD. These patients are most commonly misdiagnosed as having peptic strictures from GERD and subsequently undergo repeat dilations with lack of durable benefit or, worse, suffer esophageal perforations from these dilations (Kaplan et al., 2003).

The risk for esophageal injury by retained medication is under-appreciated. A variety of medications have been reported to cause injury to the esophagus (Abid et al., 2005; Kirkendall, 1991), with the most common class being the non-steroidal anti-inflammatory agents. Other frequently reported and commonly used agents include the tetracycline class of antibiotics; bisphosphonates for treatment of osteoporosis; vitamin C, iron, and potassium supplements. The possibility for swallowed capsules to be retained in the esophagus in healthy subjects, even with a fluid chaser (Stein et al., 1992), is under-appreciated.

In general, all patients should swallow potentially injurious medications in an upright position with a glass of water. This admonition can be a particular problem for bed-bound patients with oropharyngeal dysphagia who are placed on a regimen of thickened liquids swallowed in small aliquots. Such patients should be considered for change to alternative medications or non-pill formulations.

There are several general principles for treatment of structural disorders, once the etiology has been identified. When possible, treatment should be directed towards the underlying disease. As an example, effective acid reduction regimens for GERD reduce the need for repeat dilation of peptic strictures (Marks et al., 1994). The patient’s medication regimen should be reviewed to avoid compounding the initial problem with an additional pill injury. When possible, luminal patency should be achieved at a diameter that allows the patient to maintain their usual diet. Most patients with a luminal diameter sufficient to allow passage of a 12 mm barium tablet will not have dysphagia with solid foods (Saeed et al., 1997). Once adequate patency has been maintained, the patient needs to remain in follow-up, as most stenoses have the potential to reform and require re-treatment.

Several techniques are available for dilating strictures (American Society for Gastrointestinal Endoscopy [ASGE], 2006). Different models of tapered bougies or dilators can be passed through the stenosis to split and stretch the luminal wall into a more open caliper. Some have a central lumen,
allowing them to be advanced over a guidewire that has previously been placed through the stricture. They are also radio-opaque, which allows the use of fluoroscopy to help guide their passage (McClave, Brady, Wright, Goldschmid, & Minocha, 1996). These come in sets with about 3 mm increments in circumference.

The clinical rule during a dilation session is to pass the bougies sequentially, starting at a diameter close to that of the narrowest part of the stenosis, until three have been passed with appreciable resistance during the passage. Depending on the nature of the stricture, several dilation sessions may be necessary to achieve a dilation diameter of 13-15 mm. The exception to this rule of 3 is with dilation of isolated thin mucosal webs or rings. In this situation, a single large caliber bougie (typically 16-20 mm diameter) is passed briskly through the lesion to try to split or shear it. Dilation can also be performed using catheters with hydrostatic plastic balloons, which can be passed through an endoscope and inflated in the stenosis, exerting a radial force.

While some strictures respond well to a single dilation treatment, others have a high rate of re-stenosis, requiring frequent repeat dilations. Such defiant strictures can be injected with corticosteroids using an injection catheter passed through the endoscope. Such treatment has been shown to decrease the need for subsequent dilations (Ramage et al., 2005). Defiant rings can also be incised with a needle-knife, passed through an endoscope (Burdick, Venu, & Hogan, 1993). Complications of dilation include aspiration, bleeding, and perforation, which may require surgical repair. The perforation rate is less than 1%, but may be increased with complex strictures and the passage of dilators blindly (Hernandez, Jacobson, & Harris, 2000).

In patients with esophageal cancer, the hope is to be able to resect the lesion for cure. This usually requires replacement of the thoracic esophagus with a gastric pull-up or a colonic interposition. These surgical procedures can be complicated by anastomotic strictures, and many patients also develop transient oropharyngeal swallowing problems (Easterling et al., 2007). Patients undergoing resection for cure often receive adjuvant chemoradiation therapy. Such therapy can also be used to try to debulk incurable cancers, but the response can be somewhat delayed, and patients with significant esophageal obstruction from the tumor frequently require additional treatments. While balloon or bougie dilators can often open the lumen acutely, the response is not durable. That part of the tumor that protrudes into the lumen can be reduced, for example by endoscopically delivered injections of alcohol or thermal cautery. Plastic and expandable metal stents can also be placed in the lumen.

### Table 3. Treatment options for dysphagia in esophageal motor disorders

<table>
<thead>
<tr>
<th>Disordered Mechanism</th>
<th>Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outflow obstruction from abnormal LES relaxation</td>
<td>Smooth muscle inhibitors (anti-cholinergics, calcium channel blockers, nitrates), botulinum toxin injection, pneumatic dilation, myotomy of LES</td>
</tr>
<tr>
<td>Inadequately propulsive esophageal body contractions</td>
<td>Smooth muscle contractor (cholinergics), esophageal replacement</td>
</tr>
<tr>
<td>Uncoordinated esophageal body contractions</td>
<td>Smooth muscle inhibitors, botulinum toxin injection, long myotomy of esophageal body</td>
</tr>
</tbody>
</table>

### Motor Disorders

Primary esophageal motor disorders are relatively rare and this can make them difficult to diagnose. Treatments are palliative, as the loss of normal neuromuscular control cannot be restored. While different classification criteria have been proposed, a simple scheme for deciding on treatment options can be devised by determining the presence of these dysfunctions:

- Inadequately propulsive esophageal body contractions that fail to propel the bolus into the stomach, and/or
- Uncoordinated, spastic contractions in the esophageal body that are as likely to retropel the bolus into the oropharynx as they are to propel the bolus into the stomach.

Treatments are tailored toward the combination of disturbances present (Table 3). Systemic medication treatments tend to work poorly when the LES dysfunction dominates concomitant esophageal body dysfunction as the cause for dysphagic symptoms.

Esophageal motor disorders can be complicated by the acquisition of structural disorders. These patients are all subject to pill injury. Patients with achalasia have an increased risk of developing esophageal cancer (Sandler et al., 1995). Patients with a myotomy can develop reflux and a peptic stricture (Parkman, Ogorek, Harris, & Cohen, 1994). Patients requiring esophageal replacement can develop an anastomotic stricture.

 Patients with esophageal motor disorders can have concomitant oropharyngeal disorders and vice versa (Triadafilopoulos, Hallstone, Nelson-Abbott, & Bedinger, 1992). Treatments of each can complicate the other. For instance, a cricopharyngeal myotomy can increase the risks for regurgitation in patients with untreated esophageal achalasia. Anti-cholinergic agents to decrease excessive salivation, can further impair already weak esophageal contractions, whereas cholinergic agents that may help ineffective esophageal motility (Agrawal, Hila, Tutuian, Mainie, & Castell, 2007) can increase sialorrhea.

### Conclusion

Esophageal conditions causing dysphagia are almost all chronic, frequently progressive, and occasionally fatal, especially if diagnosed late in the course. Ongoing management and follow-up are necessary for these patients. The SLP who remains alert to the possible presence of such a disorder in the patient being evaluated can be in position to avert considerable morbidity and potential mortality for that patient.
his undergraduate degree in biomedical engineering at Tulane University and his medical degree at Johns Hopkins University School of Medicine. He is president-elect of the Dysphagia Research Society. Dr. Massey is the author of over 100 published papers, editorials, reviews, and abstracts.

References


Food for Thought: What Happens in the Esophagus Doesn’t Stay in the Esophagus...So Who Cares?

Caryn S. Easterling
University of Wisconsin
Milwaukee, WI

The esophagus is an organ of transport. As speech language pathologists (SLP), we are rarely concerned with the function of the esophagus unless the bolus transport is slowed, halted, or redirected resulting in patient symptoms and signs unexplained by the oropharyngeal evaluation findings. Esophageal function appears deceptively simple and ignorable as we assess the efficient, brisk movement of the prepared, cohesive bolus as it moves through the pharynx, past the closed airway, into the relaxed, compliant, and maximally opened upper esophageal sphincter (UES) leaving no remnants behind to be inhaled or cleared by the next breath or swallow.

The patient is happy, you are happy, and there is no reason at all to look any further because the patient’s oropharyngeal deglutitive function appears normal. However, how does the SLP explain the patient’s clinical history that precipitated the evaluation? After all, the patient’s clinical history included reports of occasional regurgitation, coughing, and the feeling of solid food “sticking” in his or her throat. The clinician may be confident that the outcome of the patient’s oropharyngeal swallow evaluation was characteristic of functional, safe, efficient, and normal swallowing for all consistencies and volumes; the SLP attributes the patient’s complaints to another portion of the digestive tract, that is, the esophagus!

ASHA Guidelines for Speech-Language Pathologists Performing Videofluoroscopic Swallowing Studies (2004) state, “Clinicians should be aware that oropharyngeal swallowing function is often altered in patients with esophageal motility disorders and dysphagia. SLP’s have knowledge and skills to recognize patient signs and symptoms associated with esophageal phase dysphagia.” So, what would be the characteristic signs and symptoms of esophageal dysphagia?

Esophageal structural abnormalities and or motor disorders disrupt bolus flow from the pharynx to the stomach or allow retrograde bolus flow from the body of the esophagus to the pharynx (esophaga-gopharyngeal reflux), or from the stomach to the esophagus (gastroesophageal reflux). Despite all of the protective mechanisms like the upper and lower esophageal sphincters and esophageal peristalsis that moves the bolus toward the stomach for the antegrade or retrograde bolus transport, reflux of gastric contents into the esophagus occurs in varying degrees in all individuals, with 20-70% of the distal reflux episodes reaching the proximal esophagus (Shaker, Dodds, Helm, Kern, & Hogan, 1991).

The anatomical proximity of the inlets of the respiratory and digestive tracts, along with the dual function of the pharynx as both airway and gastrointestinal paths, can predispose the system to the risks of irritation and aspiration during suprathoracic distribution of gastric refluxate. The copious literature describing supraesophageal symptoms of retrograde gastric content transit to the level of the larynx and pharynx includes areas of particular concern for the SLP. Entrance of gastric contents to this level is commonly referred to as laryngopharyngeal reflux (LPR) with accompanying symptoms including hoarseness, chronic cough, laryngospasm, globus, post nasal drip, heartburn, throat clearing, chronic sinusitis, night-time heartburn, otalgia, and sore throat. Potential laryngopharyngeal signs associated with supraesophageal reflux are edema and hyperemia of the larynx, hyperemia and lymphoid hyperplasia of the posterior pharynx (cobblestoning), interarytenoid changes, granuloma, contact ulcers, laryngeal polyps, tumors, subglottic stenosis, posterior glottic stenosis, strictures, and apnea (Vaezi, 2003).

However, the results of several large systematic investigations challenge the diagnostic specificity of laryngopharyngeal findings attributed to gastroesophageal reflux (Hicks, Ours, Abelson, Vaezi, & Richter, 2002; Vavricka et al., 2007). Our ability to reliably identify the changes associated with LPR has also been called into question. Kelchner and colleagues (2007) found the level of inter-rater reliability in identifying the presence and severity of physical findings attributed to laryngopharyngeal reflux signs between multidisciplinary team members was not consistent while intra-rater reliability did not show significant difference between trials. In addition, there is poor correlation between the described signs and symptoms of LPR and positive patient therapeutic outcomes, because patients may be unresponsive to medications, indicating that there is another mechanism contributing to the patient signs and symptoms (Qadeer et al., 2005).

The results of the instrumental oropharyngeal swallow evaluation should include an esophageal screening. The observations of a structural or motor abnormality from the esophageal screening may suggest that a referral to a gastroenterologist for an upper GI series or double/air contrast esophagram may be needed to view the structure and function of the esophagus with optimal positioning. Abnormalities of the mid or distal esophagus or gastric cardia can cause referred dysphagia to the upper chest or pharynx. Therefore, a combined radiographic evaluation of the pharynx, esophagus and gastric cardia may be recommended in patients with unexplained pharyngeal dysphagia. In addition, diagnostic assessment of esophageal motor function should include manometry, impedance testing, and pH studies to assess esophageal motility and the frequency, duration and degree of reflux events. “SLP’s should have sufficient knowledge of esophageal structure and function to make an appropriate referral and plan cooperative management” (ASHA, Instrumental Diagnostic Procedures for Swallowing, 1991; Knowledge and Skills Needed by Speech-Language Pathologists Performing Videofluoroscopic Swallowing Studies, 2004).

Realizing that disrupted bolus transport may be caused by
an esophageal structural (for example; esophageal ring, web, stricture or eosinophillic esophagitis) or motor disorder (for example; achalasia) resulting in signs and symptoms dictates that the patient be referred to a gastroenterologist or an otolaryngologist for further evaluation. Referring to a medical specialty allows the SLP to establish relationships with physicians, improving future patient referrals, fostering research collaboration, as well as allowing comprehensive multidisciplinary care for their patient.

There are several mechanisms that play a major role in the delicate interaction between the upper gastrointestinal and airway tracts and help prevent invasion of retrograde aspiration. The specific mechanism responsible for volume clearance of the pharynx and esophagus is the pharyngeal swallow and secondary esophageal peristalsis. The esophago-UES and pharyngo-UES reflexes help in prevention of entry of gastric content into the esophagus and pharynx. While the contractile reflexes, specifically the esophagoglottal and the pharyngo-glottal reflexes accentuate the closure of the upper and lower esophageal sphincters and also induce closure of the vocal cords (Shaker & Hogan, 2003). Understanding the complicated reflexes that allow the unique upper aerodigestive system to operate so efficiently is the key to efficient assessment, management and care of patients with dysphagia.

Caryn Easterling is an assistant professor at the University of Wisconsin-Milwaukee. She has an appointment as a research scientist at the Veterans Administration Medical Center and is an assistant professor in the Department of Neurology, and Pediatric Gastroenterology and Nutrition at the Medical College of Wisconsin in Milwaukee, WI. She serves as treasurer of the Board for Specialty Recognition in Swallowing and Swallowing Disorders and has served as a member of the Division 13 Steering Committee.

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