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Clinical Pathology

Dysphagia: Clinical Evaluation, Assessment and Management

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Abstract **Review Article**

Dysphagia is a common problem affecting all ages. It's divided into oropharyngeal dysphagia and esophageal dysphagia. Oropharyngeal dysphagia manifests as difficulty initiating swallowing, coughing, choking, or aspiration, and it is most commonly caused by chronic neurologic conditions such as stroke, Parkinson disease, or dementia, and Obstructive (oropharyngeal) symptoms that seem to originate in the throat or neck may actually be caused by distal esophageal lesions. Patients with esophageal dysphagia may report a sensation of food getting stuck after swallowing. This condition is most caused by gastro esophageal reflux disease and functional esophageal disorders. Eosinophilic esophagitis is triggered by food allergens and is increasingly prevalent. Careful history taking remains the first and most important step in evaluating dysphagia, and it is especially important to distinguish an oropharyngeal versus esophageal origin, which helps to guide further investigation and therapy. The three main investigations for dysphagia remain endoscopy, barium study and manometry, with endoscopy also offering therapeutic potential. Management is largely determined according th the eventual diagnosis, often in a multi-disciplinary setting.

Keywords: Dysphagia, clinical evaluation, management.

INTRODUCTION

Dysphagia, defined as difficulty swallowing, is a prevalent symptom. Often encountered by family and otolaryngology physicians [1]. Many people occasionally experience difficult or impaired swallowing, but they often adapt their eating patterns to their symptoms and do not seek medical attention [1]. Dysphagia not only has the potential to cause severe complications such as malnutrition and risk of aspiration but also carries a significant social and psychological burden on the individual [2]. In the past two decades, there has been an increase in the incidence of eosinophilic esophagitis and achalasia, in part due to greater recognition of these conditions and the development of more advanced diagnostic techniques [3, 4]. In particular, the incidence of eosinophilic esophagitis has increased so rapidly that it is now thought to be the most frequent eosinophilic gastro-intestinal disorder as well as the second most common cause of chronic esophagitis and dysphagia after gastroesophageal reflux disease (GERD) [5]. In this perspective, we will present a systematic approach to dysphagia, including the selection and interpretation of investigations, as well as an overview of contemporary treatment strategies.

Pathophysiology

Swallowing (deglutition) is a complex process involving voluntary and involuntary neuromuscular contractions coordinated to permit breathing and swallowing through the same anatomic pathway [6]. Deglutition is into oropharyngeal and esophageal stages. In the oropharyngeal stage, food is chewed and mixed with saliva to form a bolus of appropriate consistency in the mouth. With the initiation of the swallow, the bolus is propelled into the oropharynx by the tongue. Other structures simultaneously seal the nasopharynx and larynx to prevent regurgitation or aspiration, and the lower esophageal sphincter begins to relax. In the esophageal stage, the food bolus passes the upper esophageal sphincter and enters the esophageal body, where it is propelled by peristalsis through the midthoracic and distal esophagus and into the stomach through the now fully relaxed lower esophageal sphincter [7, 8].

Oropharyngeal Pathology

Oropharyngeal dysphagia is most related to chronic neurologic conditions, particularly Parkinson disease, stroke, and dementia; it is not part of normal aging [9]. It may be the first symptom of a neuromuscular disorder, such as amyotrophic lateral sclerosis or myasthenia gravis [10, 11]. Some chronic conditions, such as poor dentition, dentures, dry mouth (xerostomia), or medication adverse effects, may be poorly tolerated in patients who also have progressive oropharyngeal dysfunction [11].

Structural abnormalities (e.g. Zenker diverticulum, cricopharyngeal bars or tumors, chronic infections with Candida or herpes virus) and extrinsic compression from cervical osteophytes or goiter can also interfere with normal swallowing (Table 1) [9].

Table-1: Causes of Oropharyngeal Dysphagia

Causes of Oropharyngeal Dysphagia		
Progrisive chronic disease (geriatric syndrome) Stroke Parkinson disease Alzheimer and other dementias Sarcopenia	**Structural causes** Head and neck cancers Recent surgery or radiation for head and neck cancers (altered anatomy) Chemoradiation-induced mucositis and edema (short term) Zenker diverticulum Cervical osteophytes Lymphadenopathy Goiter Cricopharyngeal bar	**Oral causes** Poor dentition or dentures Dry moth (i.e., xerostomia) Medications causing dry moth (e.g., alpha and beta blockers, angiotensin-converting enzyme inhibitors, anticholinergics, antihistamines, anxiolytics, calcium chanel blockers, diuretics, muscle relaxants, tricyclic antidepressants) Antipsychotic medications
Neuromuscular disease Amyotrophic lateral sclerosis Myasthenia gravis Multiple sclerosis Dermatomyositis/ polymyositis (myopathies) Antipsychotic medications		

Esophageal Pathology

Gastroesophageal reflux disease (GERD), functional esophageal disorders, and eosinophilic esophagitis are the most common causes of esophageal dysphagia (Table 2) [1, 9, 12]. Less common causes include medications, obstructive lesions, and esophageal motility disorders [9].

Table-2: Causes of Esophageal Dysphagia

Causes of Esophageal Dysphagia		
Gastro-esophageal reflux disease and esophagitis (30% to 40%) Eosinophilic esophagitis Functional dysphagia **Functional esophageal disorders (20% to 30%)** Functional heartburn Gastro-esophageal reflux disease (nonerosive) Globus pharyngeus Reflux hypersensitivity **Structural or mechanical conditions (5%)** Esophageal or peptic stricture (caused by erosive esophagitis) Foreign body or food impaction (acute-onset dysphagia) Malignancy (esophageal or gastric cancer, mediastinal mass with extrinsic compression) Schatzki ring	**Medications (5%)** Pill esophagitis (direct irritation associated with ascorbic acid, bisphosphonates, ferrous sulfate, non-steroidal anti-inflammatory drugs, potassium chloride, quinidine, and tetracyclines) Reflux caused by decreased tone of lower esophageal sphincter (associated with alcohol, anticholinergics, benzodiazepines, caffeine, calcium channel blockers, nitrates, and tricyclic antidepressants)	**Esophageal motility disorders (< 5%)** Absent contractility achalasia Distal esophageal spasm Esophagogastric junction outflow obstruction Hypercontractile (jackhammer) esophagus Hypercontractile motility disorders Opioid-induced esophageal dysfunction **Rheumatologic conditions (< 5%)** Systemic sclerosis (scleroderma)

Clinical Evaluation

History

The first and most important step in evaluating dysphagia remains a careful history to distinguish between oropharyngeal or esophageal. Patients will report a sensation that food or drink sticks, holds up or stops, but uncommonly can have other presenting complaints such as regurgitation, vomiting or retrosternal discomfort. The three core aims on history are to establish firstly the presence of true dysphagia, secondly to determine whether the site of the problem is pharyngeal or esophageal, and finally to determine if there is a structural or motility problem [6]. Reports of 'difficulty swallowing' may not necessarily reflect true

dysphagia. Globus is the feeling of a lump or foreign body in the throat. A distinction from dysphagia is that globus is present between meals and is unrelated to swallowing, so much so that the symptom often disappears entirely during meals [7]. Xerostomia (oral dryness) can give rise to a sense of dysphagia, although in more severe cases true dysphagia can develop consequent to the loss of the lubricating qualities of saliva leading to delayed bolus transit. Finally, odynophagia, or painful swallowing, most commonly reflects an inflammatory process of the esophageal mucosa, and is generally transient, lasting only during the time taken for a bolus to traverse the esophagus [13].

Once the presence of true dysphagia has been established, a careful history to distinguish between an oropharyngeal and esophageal location of the problem not only guides subsequent investigation but can also be diagnostic on its own merits [13, 14]. The site of the sensation of the holdup can sometimes localize the site of the problem, with a suprasternal sensation suggestive oropharyngeal problem and a lower retrosternal sensation suggestive of an esophageal problem, but this can lack specificity and in particular, symptoms felt at the sternal notch may reflect obstruction lower down in the esophagus. Additional symptoms such as coughing, and spluttering may also help pointing to an upper cause [13, 14]. The age and comorbidity profile of the patient can also guide the differential diagnosis. The elderly are more likely to have a malignant or oropharyngeal cause (particularly if there is a concomitant neurological disorder), younger patients are more likely to have an inflammatory cause such as eosinophilic esophagitis, and those with connective tissue disorders may have underlying esophageal dysmotility or complications arising from chronic reflux [13, 14].

Oropharyngeal dysphagia

Four symptoms suggestive of oropharyngeal dysfunction are [14]:

1. Delayed/absent swallow initiation.
2. Deglutitive cough
3. Postnasal regurgitation during swallowing
4. Need for repetitive swallows to clear a bolus from the pharynx.

Oropharyngeal dysphagia, particularly in the elderly, is most commonly associated with stroke, neurodegenerative causes and dementia (Table 1) [15].

Esophageal Dysphagia

Causes of esophageal dysphagia can be broadly categorized into structural problems and motility disorders (Table 2) [1, 9, 12]. Dysphagia to solids only is suggestive of a mechanical obstruction, whereas motility disorders tend to cause problems with both solid and liquid boluses (although solids are generally affected more). The time- course of dysphagia is helpful for structural causes, with slowly progressive symptoms being a feature of benign pathologies such as a peptic stricture, whereas malignant pathology such as esophageal carcinoma tends to present more rapidly, often with associated weight loss [16]. Intermittent dysphagia with generally normal function is a feature of eosinophilic esophagitis, spasm and minor esophageal mucosal strictures. Given that GERD is the most common disease associated with dysphagia, eliciting typical reflux symptoms such as heartburn and/or regurgitation is helpful [16]. The presence of atopy is a useful clue, as there is a strong association between eosinophilic esophagitis and atopic diseases such as asthma, food allergy, eczema and chronic rhinitis [6].

Certain esophageal conditions, for example, achalasia and spasm, can also manifest as chest pain independent of swallowing, and should be considered in the right clinical context. Finally, it is important to note on history-taking risk factors for esophageal cancer, namely, smoking, alcohol use and longstanding reflux [12].

Physical Examination

Oropharyngeal dysphagia may be just one of many manifestations of various neurological conditions such as stroke and Parkinson disease, and therefore a complete neurological examination is important when a neurological disorder is suspected [13, 15]. Examination of the neck is also important to exclude any mass lesion that might cause obstruction, or tenderness including lymphadenopathy which might point to an infective or inflammatory process [13, 15]. In contrast, the physical examination is generally non-contributory in esophageal dysphagia; however, it is important to examine the skin and joints for features of connective tissue disorders such as systemic sclerosis, given the association of these conditions with esophageal hypomotility [13, 15]. The oral cavity should be inspected for dentition, evidence of xerostomia and of infective conditions such as candidiasis [13, 15]. Noting major chest and spine deformities may provide clues for under- lying syndromes [13, 15]. Complications of dysphagia such as malnutrition, weight loss and pulmonary complications may also be evident on physical examination [13, 15].

Barium Swallow

The standard barium swallow involves ingestion of high- density barium in an upright position for double-contrast views of the esophagus, followed by discrete swallows of low-density barium in a prone position to evaluate esophageal motility [18]. It is a non-invasive, inexpensive, widely available test that also allows for the assessment of aspiration risk [17, 18]. A barium swallow can be used as the first investigation particularly when the suspected etiology is oropharyngeal rather than esophageal, or if there is concern about patient's fitness for endoscopy or

regarding endoscopic intubation of the esophagus such as in suspected laryngeal malignancy, Zenker's diverticulum or upper esophageal stricture [17, 18]. Cervical osteophytes and cricopharyngeal bar, although not uncommonly found particularly in the elderly, are rarely the sole pathology causing dysphagia unless obstruction to passage of bolus can be objectively demonstrated [17, 18]. Although particularly useful in cases of suspected oropharyngeal dysphagia, barium swallow can also evaluate for obstructive esophageal lesions or esophageal dysmotility [17, 18].

Upper Endoscopy

Upper endoscopy is mandated in most patients and serves both diagnostic and therapeutic purposes. It allows for direct visualization of the pharynx, esophagus, stomach and duodenum, which is particularly useful in cases of intraluminal masses, strictures and mucosal inflammation. The procurement of biopsies via endoscopy to confirm a histological diagnosis is also important [19]. Although eosinophilic esophagitis has characteristic endoscopic features of longitudinal rings, circular furrows, white plaques and submucosal edema, biopsies are required for diagnosis and the esophagus may indeed be macroscopically normal in 10–25% of patients [19]. In motility disorders, such as achalasia, upper endoscopy may provide some clues such as a dilated esophagus, retention of food and a tight gastroesophageal junction (GEJ), but is also compulsory to exclude pseudoachalasia due to a mechanical obstruction at the GEJ [20]. Upper endoscopy also allows the application of therapy, such as dilatation via a bougie- or balloon-based technique, in many structural causes of dysphagia, particularly benign pathologies, such as peptic strictures, Schatzki ring , radiation- related stricturing and eosinophilic esophagitis [22].

Other investigations for oropharyngeal dysphagia videofluorographic

The videofluorographic swallowing study (or modified barium swallow), involves a speech pathologist assessing barium contrast-impregnated food and liquid traversing the oral cavity, pharyngeal cavity and esophagus in real time, and is the gold standard for assessment of aspiration [15]. Further workup in the setting of oropharyngeal dysphagia may necessitate the involvement of an otolaryngologist to perform flexible nasoendoscopy for direct visualization of the oropharynx and larynx [20].

High-resolution manometry

High-resolution manometry (HRM) is performed utilizing a catheter with closely spaced pressure sensors positioned 1 cm apart along the length of the catheter, and the subsequent pressure variations being displayed topographically as Clouse plots [17]. The manometry catheter is placed trans-nasally after the application of a topical local anesthetic agent such that the distal tip of the catheter is 2–3 cm below the diaphragm. The patient, while lying recumbent, then performs 10 swallows of 5 mL water boluses [17]. Compared to the previous generation perfused manometry, the modern solid-state electronic sensored HRM is easier to interpret and provides superior diagnostic yield [18]. The Chicago classification of esophageal motility disorders, now in its fourth edition, uses an algorithm for analysis of HRM and is accepted worldwide [17].

Intraluminal esophageal impedance

IEI is a Measuring the movement of a swallowed bolus using impedance in real time, in conjunction with measuring pressure waves recorded by manometry, allows further in-depth analysis of a swallow and correlation with symptoms. The functional lumen imaging probe is a newer method of using impedance planimetry for assessing esophageal motility and distensibility. This involves endoscopic placement of a probe with a cylindrical fluid-filled bag within which there are pressure sensors and paired electrodes. This technique, however, currently still remains outside of routine clinical use [23].

Management

Our approach to the initial workup and management of dysphagia is outlined in Figure 1.

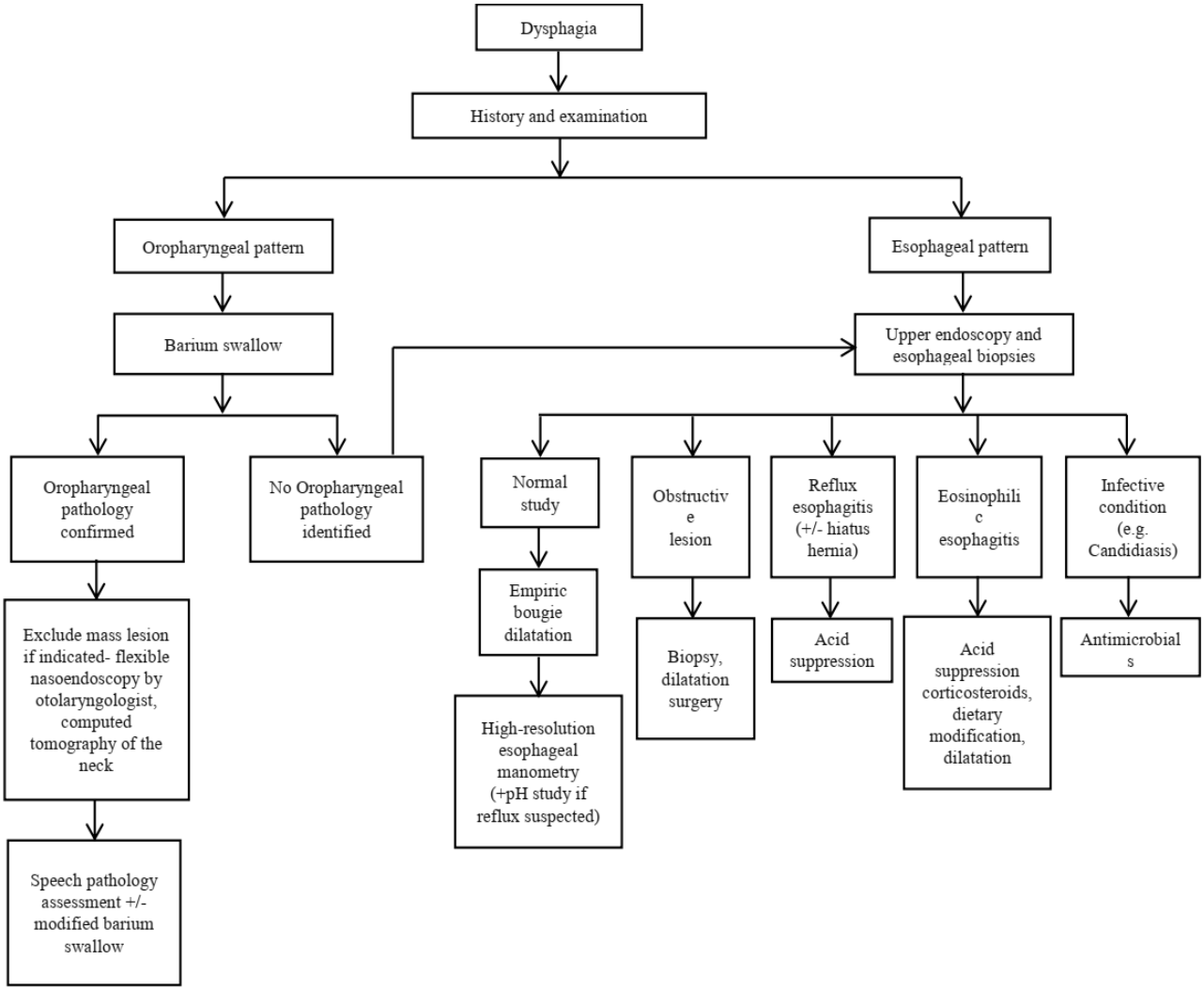

Figure 1: The initial workup and management of dysphagia

Although management is largely targeted at a specific disorder once identified, in cases where a cause is not apparent there is a role for empiric bougie dilatation and a trial of proton-pump inhibitor therapy, given that these can potentially treat an unrecognized underlying pathology [23].

Oropharyngeal Dysphagia

The treatment of oropharyngeal dysphagia largely is in the domain of a multidisciplinary team, which may involve gastroenterologists, neurologists, Otorhinolaryngologist, dieticians and speech pathologists. The most important step is to identify and treat an underlying primary disease. Stenotic conditions such as cervical web or cricopharyngeal stenosis may require dilatation or myotomy. In the event of a non-modifiable underlying primary pathology, then it is important to establish the risk of aspiration via modified barium swallow, to determine if non-oral feeding is required, for example percutaneous gastrostomy tube. Finally, dietary modification and swallow therapy, led by speech pathology, is usually required to allow for adequate nutrition while mitigating aspiration risk [14].

Mechanical obstruction

Mechanical obstruction due to strictures, webs or rings are generally adequately managed by endoscopic dilatation using a bougie over guide wire technique (considered safer compared to blind), or balloon dilatation. Patients can be treated with repeated dilatation at appropriate frequency of up to 1–2 weekly [22]. Recurrent or recalcitrant strictures may require endoluminal stenting. Steroid injection to the stenosis has been shown to reduce recurrence and can be considered in select cases. Surgical intervention may be required for intraluminal neoplastic masses and in cases of extrinsic compression [22, 24].

Dysmotility

The three main dysmotility conditions that can give rise to dysphagia include achalasia, hypercontractile conditions of the esophagus and esophageal hypomotility.

Achalasia

Define as failure of relaxation of the lower esophageal sphincter (LES) coupled with loss of normal esophageal body peristalsis. The former has been the

main therapeutic target of therapies used today, namely botulinum toxin injection, pneumatic dilatation, laparoscopic Heller myotomy and peroral endoscopic myotomy (POEM). Newly diagnosis of achalasia is divided into three subtypes, based on manometric characteristics. Types 1 and 2 rep resent esophageal hypomotility and generally carry good prognosis to treatment. Type 3 represents spastic achalasia, and although traditionally responds poorly to treatment, there is now good evidence that a long myotomy, whether it be surgical or endoscopic, will substantially improve prognosis [24].

Endoscopy-guided botulinum toxin injection reduces LES pressure by inhibiting acetylcholine release from nerve endings and is usually a safe procedure with the predominant drawback of a response time limited to months, although subjective symptomatic benefits can last longer [24, 25].

Pneumatic dilatation utilizes a pressurized balloon with endoscopic and fluoroscopic guidance to disrupt the fibres of the LOS. It conventionally starts with a 30 or 35 mm balloon, progressing up to 40 mm in a graded manner to provide a response [24, 25].

Laparoscopic Heller myotomy, which involves a laparoscopic myotomy with a partial fundoplication, provides an improvement in dysphagia in up to 90% of patients and has been the gold-standard for therapy in the past many decades. POEM was introduced in 2008 and involves creation of a submucosal tunnel via electro cautery in the mid-esophagus through which the endo-scope is advanced to the LOS, followed by electro cautery of its circular fibres [24, 25].

Hypercontractile disorders

Major hypermotility disorders of peristalsis such as hypercontractile (jackhammer) esophagus and distal esophageal spasm cause episodic symptoms and can be difficult to diagnose. Treatment is aimed at reducing the hyper contractility via medical or surgical means. Pharmacological relaxation of the smooth muscle with calcium channel blockers, long-acting nitrates and phosphodiesterase inhibitors are reasonable options albeit lacking good evidence, and therapeutic response is highly variable and often modest. Surgical myotomy or POEM targeting the esophageal body are potential options in severe cases [25].

Hypomotility

Therapeutic options to improve esophageal peristalsis are limited and therefore the management of severe hypomotility is focused on dietary lifestyle and nutritional supplementation [25, 26].

Eosinophilic esophagitis

The overall goals of treatment are to reduce symptoms, prevent disease progression (fibrosis at late stage), improve quality of life and reverse existing complications [26]. The three main treatment options are: topical corticosteroids, empirical elimination diet and proton-pump inhibitors [27]. The six-food elimination diet that removes milk, wheat, soy, egg, nuts and seafood has response rates between 50 and 75%, with the four-food elimination diet (wheat, soy, egg, milk) being a less restrictive option that also mildly compromises efficacy [27]. Consultation with a clinical allergist may also be helpful, particularly when there is the presence of comorbid atopic conditions. In patients who select for a pharmacologic approach, initial treatment is with 8 weeks of twice-daily proton-pump inhibitor or swallowed steroids including fluticasone or budesonide. Those with esophageal strictures may require dilatation as an adjunct to pharmacologic and dietary therapy [27]. Treatment efficacy is gauged by performing follow-up endoscopy and obtain esophageal biopsies to demonstrate resolution of esophageal eosinophilia [28]. The natural history of eosinophilic esophagitis is incompletely understood, but appears to be a chronic disease with persistence of symptoms and long-term complications including esophageal fibrosis if left untreated [28].

Functional/idiopathic dysphagia

In the setting of functional/idiopathic dysphagia, it is important to attempt to reduce the use of drugs that impact motility, in particular opioids. Given functional dysphagia is not associated with a detrimental outcome, and the natural history is generally one of improvement over time, simple measures, such as eating upright and careful chewing, may suffice. Other approaches that can be trialed include acid suppression, antidepressants and empiric bougie dilatation [29].

CONCLUSION

A detailed history is the first step in evaluating dysphagia, main three investigation tests consist of upper endoscopy, barium swallow and HRM. Upper endoscopy is the initial, and generally mandated test in suspected esophageal dysphagia, and is particularly important to exclude malignant obstructive pathologies and to obtain tissue biopsies for the diagnosis of the increasingly prevalent eosinophilic esophagitis. Barium swallow is useful in the setting of oropharyngeal dysphagia or proximal esophageal pathology. For assessment of dysmotility, HRM in conjunction with color-contour topography is the current gold standard and allows for diagnosis based on the Chicago classification. Management of dysphagia largely depends on the underlying pathology, which can include dietary, pharmacologic, endoscopic, and less commonly, surgical approaches.

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