World Gastroenterology Organisation Practice Guidelines:

Dysphagia

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Contents

1 Definition

Dysphagia either refers to the difficulty someone may have with initiating a swallow (usually referred to as oropharyngeal dysphagia) or it refers to the sensation that foods and or liquids are somehow hindered in their passage from the mouth to the stomach (usually referred to as esophageal dysphagia).

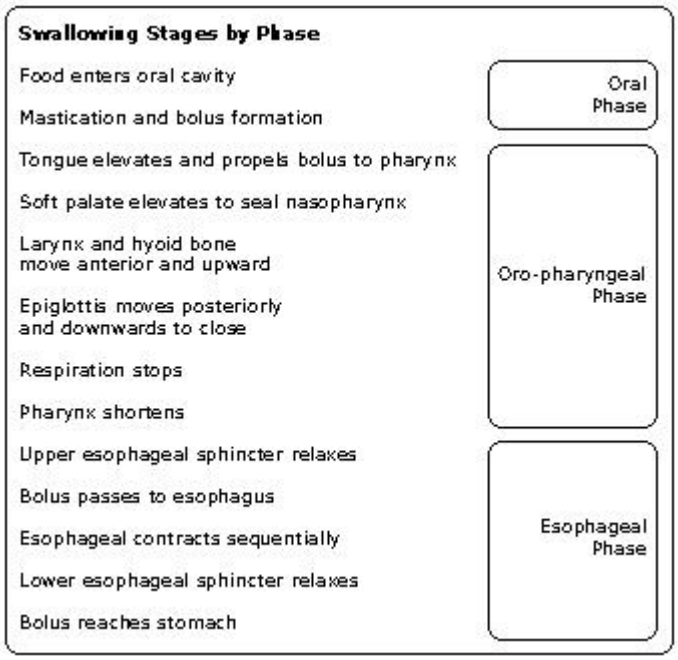
Dysphagia therefore is the “perception” that there is an impediment to the normal passage of swallowed material.

2 Introduction and key points

Swallowing is a process governed by the swallowing center in the medulla, and in the mid-esophagus and distal esophagus by a largely autonomous peristaltic reflex

coordinated by the enteric nervous system. Figure **1** indicates the physiological mechanisms involved in these various phases.

Fig. **1** Swallowing stages by phase



A decision has to be made about the location of the dysphagia as described by the patient; the lesion will be at or below this perceived location. Similarly, it is important to establish whether the dysphagia is for solids, liquids, or both, and whether it is progressive or intermittent. It is also important to establish symptom duration.

Although the conditions can frequently occur together, it is also important to exclude odynophagia (painful swallowing). Finally, a symptom-based differential diagnosis should exclude globus pharyngeus (lump-in-the-throat sensation), chest pressure, dyspnea, and phagophobia (fear of swallowing).

Key features to consider in the medical history are:

- Location
- Types of foods and or liquids
- Progressive or intermittent
- Duration of symptoms

Key decision: is the dysphagia oropharyngeal or esophageal? This distinction may be confidently made on the basis of a very careful history, which provides an accurate assessment of the type of dysphagia (oropharyngeal vs. esophageal in about 80–85% of cases).

2.1 Oropharyngeal dysphagia: chief manifestations

Oropharyngeal dysphagia can also be called "high" dysphagia, referring to an oral or pharyngeal location. Patients have difficulty initiating a swallow, and they usually identify the cervical area as the area presenting a problem.

Frequent accompanying symptoms:

- Difficulty initiating swallow
- Nasal regurgitation
- Coughing
- Nasal speech
- Diminished cough reflex
- Choking (note that laryngeal penetration and aspiration may occur without concurrent choking or coughing).
- Dysarthria and diplopia (may accompany neurologic conditions that cause oropharyngeal dysphagia).
- Halitosis may be present in patients with a large residue-containing Zenker's diverticulum, also with advanced achalasia or long-term obstruction with luminal accumulation of decomposing residue.

A precise diagnosis can be obtained when there is a defined neurological condition accompanying the oropharyngeal dysphagia, such as:

- Hemiparesis following an earlier cardiovascular accident
- Ptosis of the eyelids
- Indications of myasthenia gravis (end-of-the-day weakness)
- Parkinson's disease
- Other neurological diseases including cervical dystonia, cervical hyperostosis and Arnold–Chiari deformity (hindbrain herniations)
- Specific deficits of the cranial nerves involved in swallowing can also help in pinpointing the origin of the oropharyngeal disturbance establishing a diagnosis.

2.2 Esophageal dysphagia: chief manifestations

Esophageal dysphagia can also be called "low" dysphagia, referring to a likely location in the distal esophagus, although it should be noted that some patients with esophageal dysphagia, such as achalasia, may describe it in the cervical region mimicking oropharyngeal dysphagia.

- Dysphagia that occurs equally with solids and liquids, often involves an esophageal motility problem. This suspicion is reinforced when intermittent dysphagia for solids and liquids is associated with chest pain.
- Dysphagia that occurs only with solids but never with liquids suggests the possibility of mechanical obstruction with luminal stenosis to diameter < 15 mm. If progressive, consider particularly peptic stricture or carcinoma. Furthermore, it is worth noting that patients with peptic strictures usually have a long history of heartburn and regurgitation, but no weight loss. Conversely, patients with esophageal cancer tend to be older men with marked weight loss.

The physical examination of patients with esophageal dysphagia is usually of limited value, although cervical/supraclavicular lymphadenopathy may be palpable in patients with esophageal cancer. Furthermore, some patients with scleroderma and secondary peptic strictures may present with the syndrome of calcinosis, Raynaud phenomenon, esophageal involvement, sclerodactyly, and telangiectasia (CREST).

Halitosis may suggest advanced achalasia or long-term obstruction, with accumulation of slowly decomposing residue in the esophageal lumen.

3 Disease burden and epidemiology

Dysphagia is a common problem. For example, the incidence of dysphagia in acute care has been put as high as 33%, and studies in nursing homes have shown that 30–40% of patients have swallowing disturbances, resulting in a high incidence of aspiration complications.

On the other hand, epidemiological data cannot be provided on a global basis, since the base rate of most diseases that may cause dysphagia tends to differ between western Europe and North America and south Asia, the Middle East, or Africa. Also, base rates will vary depending on the age of the patient, and it should also be remembered that the spectrum of disorders in childhood dysphagia is different from that of older age. Therefore, only approximations are possible on a global scale. Generally, dysphagia occurs in all age groups but its prevalence increases with age.

In younger patients, dysphagia often involves accident-related head and neck injuries, as well as cancers of the throat and mouth. Tumor prevalence differs among various countries. For instance, whereas in the USA adenocarcinoma is the most common type of esophageal cancer, in India and China it is squamous-cell carcinoma. Similarly, corrosive strictures of the esophagus (individuals consuming corrosive agents with suicidal intent) and tuberculosis can also be important aspects in a non-Western setting.

4 Causes of dysphagia

In trying to establish the etiology of dysphagia, it is useful to follow the same classification adopted for symptom assessment — that is, to make a distinction between those causes that mostly affect the pharynx and proximal esophagus (oropharyngeal or “high” dysphagia) and causes that mostly affect the esophageal body and esophagogastric junction (esophageal or “low” dysphagia). However, it is true that many disorders overlap, and they can produce both oropharyngeal and esophageal dysphagia. A thorough history-taking including medication use is very important, since drugs may be involved in the pathogenesis of dysphagia.

4.1 Oropharyngeal dysphagia

In young patients, oropharyngeal dysphagia is most often caused by inflammatory muscle diseases, webs, and rings. In older people, it is usually caused by central

nervous system disorders, including stroke, Parkinson's disease, and dementia. Generally, it is useful to try to make a distinction between mechanical problems and neuromuscular motility disturbances, as shown below.

4.1.1 Mechanical and obstructive causes

- Infections (e.g., retroperitoneal abscesses)
- Thyromegaly
- Lymphadenopathy
- Zenker's diverticulum (with small diverticula, the cause may be upper esophageal sphincter dysfunction)
- Reduced muscle compliance (myositis, fibrosis)
- Head and neck malignancies
- Cervical osteophytes (rare)
- Oropharyngeal malignancy and neoplasms (rare)

4.1.2 Neuromuscular disturbances

- Central nervous system diseases such as stroke, Parkinson's disease, cranial nerve, or bulbar palsy (e.g., multiple sclerosis, motor neurone disease), amyotrophic lateral sclerosis.
- Contractile disturbances such as cricopharyngeal spasm (upper esophageal sphincter dysfunction) or myasthenia gravis, oculopharyngeal muscular dystrophy, and others.

Post-stroke dysphagia has been identified in around 50% of cases. The severity of dysphagia tends to be associated to the severity of stroke. Up to 50% of Parkinson patients manifest some symptoms consistent with oropharyngeal dysphagia, and up to 95% show abnormalities on video esophagography. Clinically significant dysphagia may occur early in Parkinson's disease, but it is more usual in the later stages.

4.1.3 Other

- Poor dentition
- Oral ulcers
- Xerostomia
- Long-term penicillamine use

4.2 Esophageal dysphagia

Three types of condition are most likely to cause dysphagia (Table **1**):

- Mucosal (intrinsic) diseases, which narrow the lumen through inflammation, fibrosis, or neoplasia
- Mediastinal (extrinsic) diseases, which obstruct the esophagus by direct invasion or through lymph-node enlargement

- Neuromuscular diseases affecting the esophageal smooth muscle and its innervation, disrupting peristalsis or lower esophageal sphincter relation, or both.

Table **1** Most common causes of esophageal dysphagia

Intraluminal foreign bodies (usually a cause of acute dysphagia)
Mucosal diseases: • Peptic stricture secondary to gastroesophageal reflux disease • Esophageal rings and webs (sideropenic dysphagia or Plummer–Vinson syndrome) [1] • Esophageal tumors • Chemical injury (e.g., caustic ingestion, pill esophagitis, sclerotherapy for varices) • Radiation injury • Infectious esophagitis • Eosinophilic esophagitis
Mediastinal diseases • Tumors (e.g., lung cancer, lymphoma) • Infections (e.g., tuberculosis, histoplasmosis) • Cardiovascular (dilated auricula, vascular compression)
Diseases affecting smooth muscle and its innervation • Achalasia (both idiopathic and associated with Chagas disease) • Scleroderma • Other motility disorders • Postsurgical (i.e., after fundoplication, antireflux devices)

5 Clinical diagnosis

5.1 Introduction

An accurate history covering the key diagnostic elements is important and can often establish a diagnosis with certainty. It is important to establish carefully the location of the perceived swallow problem (oropharyngeal vs. esophageal dysphagia).

5.2 Diagnosis and management of oropharyngeal dysphagia

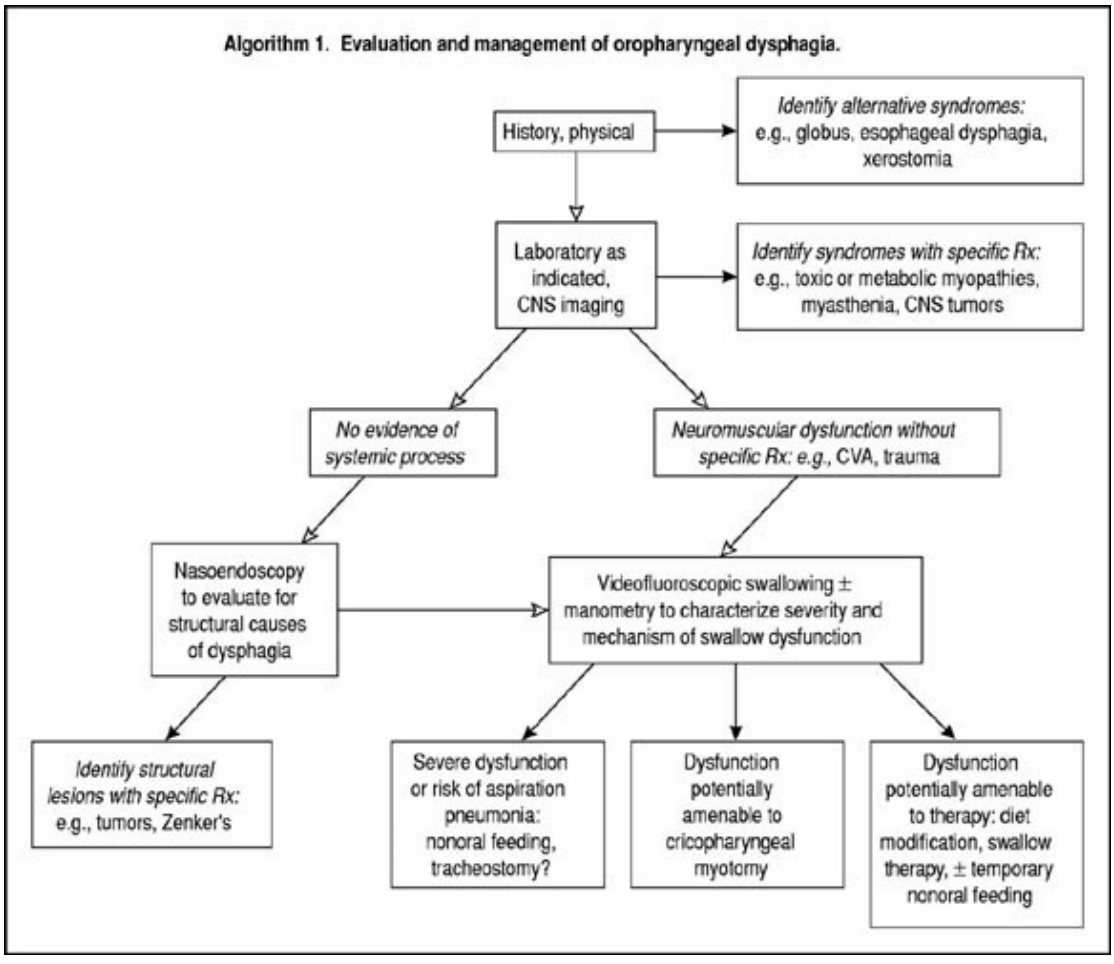
The timed water-swallow test is an inexpensive, potentially useful basic screening test to complement the evidence obtained by clinical history and physical examination. The test consists of the patient drinking 150 mL water from a glass as quickly as possible, with the examiner recording time taken and number of swallows. From these data, the speed of swallowing and the average volume per swallow can be calculated. This test is reported to have a predictive sensitivity of > 95% for identifying the presence of dysphagia. This test may be complemented by a "food test" using a small amount of pudding placed on the dorsum of the tongue [2].

Whereas a water-swallow test can be carried out to establish dysphagia, it fails to identify aspiration in 20–40% of cases when followed up by video fluoroscopy, because of the absent cough reflex, as described earlier.

More specific and reliable tests for evaluation of dysphagia must be considered depending on characteristics of patient and significance of his/her problem. In this regard, it should be noted that the video-fluoroscopic swallowing study (also known as the "modified barium swallow") is the gold standard for diagnosing oropharyngeal dysphagia and that nasoendoscopy is the gold standard for the evaluation of structural causes of dysphagia [3–5]. Video-fluoroscopic techniques can also be transmitted via the Internet, facilitating interpretative readings at remote sites [6]. Video-fluoroscopic evaluation may also help predict the risk of aspiration pneumonia [7].

The algorithm shown in Fig. **2** gives an indication of more sophisticated tests and procedures necessary to pursue a diagnostic investigation leading to specific therapies.

Fig. **2** The evaluation and management of oropharyngeal dysphagia.



Jejunal tube feeding should be used in the acute setting, and percutaneous gastrostomy or jejunostomy tube feeding in the chronic setting.

5.3 Diagnosis and management of esophageal dysphagia

5.3.1 Clinical history

The clinical history should be considered first. The main concern with esophageal dysphagia is to exclude malignancy. The patient's history may provide clues; a malignancy is likely if there is:

- Short duration (< 4 months)
- Disease progression
- Dysphagia more for solids than for liquids
- Weight loss

Achalasia is more likely if:

- There is dysphagia for both solids and liquids.
- The problem has existed for several months or years.
- There is no weight loss.

With regard to diagnostic tests, there is some debate as to whether endoscopy or barium swallow should be the initial test employed.

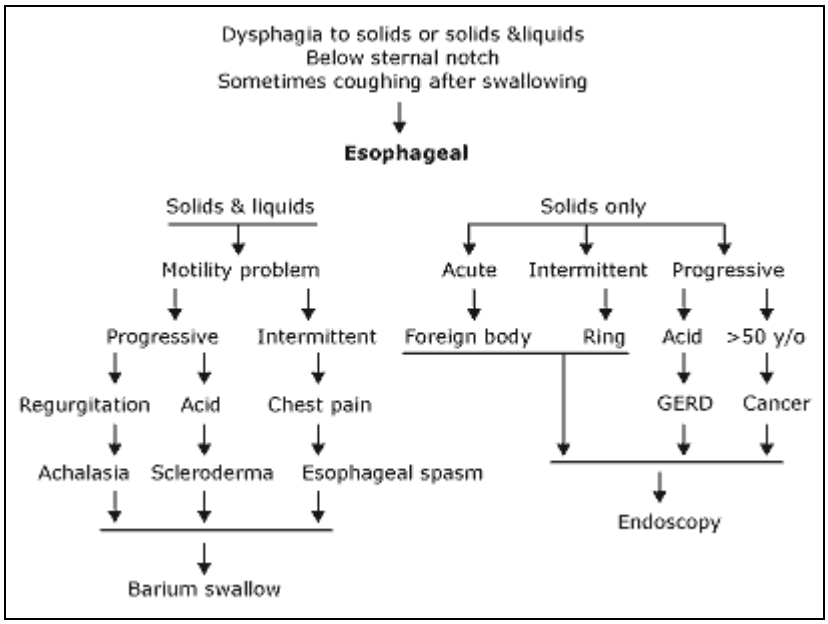
5.3.2 Barium-contrast esophagogram (barium swallow)

The barium esophagogram, taken with the patient supine and upright, outlines irregularities in the esophageal lumen and identifies most cases of obstruction, webs, and rings. The barium examination of the oropharynx and esophagus during swallowing is the most useful initial test; it can also be helpful in the detection of achalasia and diffuse esophageal spasm, although these conditions are more definitively diagnosed by manometry. It may also be useful to include a barium tablet to identify subtle strictures. A barium swallow may also be helpful in dysphagic patients with a negative endoscopy if the tablet is added.

5.3.3 Endoscopy

Endoscopy uses a fiberoptic endoscope passed through the mouth into the stomach, with detailed visualization of the upper gastrointestinal tract. The introduction of the scope into the gastric cavity is very important to exclude pseudoachalasia due to a tumor of the esophagogastric junction. The algorithm shown in Fig. **3** outlines management decision-making.

Fig. **3** Evaluation and management of esophageal dysphagia



5.3.4 Other diagnostic tests

- *Esophageal manometry.* This diagnostic method is less commonly available than barium swallow and endoscopy, but can be very useful in selected cases. It is based on recording the esophageal lumen pressure using either solid-state or perfusion techniques. Manometry is indicated when an esophageal cause of dysphagia is suspected following an inconclusive barium swallow and endoscopy and following adequate antireflux therapy (with healing of esophagitis shown endoscopically). The three main causes of dysphagia that can be diagnosed using esophageal manometry are achalasia, scleroderma (ineffective esophageal peristalsis), and esophageal spasm.
- *Radionuclide esophageal transit scintigraphy.* The patient swallows a radiolabeled liquid (for example, water mixed with ^{99m}technetium sulfur colloid), and the radioactivity within the esophagus is measured. Patients with esophageal motility disorders typically have a delayed disappearance of the radiolabel from the esophagus. The technique is primarily used for research purposes, but it is now beginning to be used for clinical purposes in some specialized institutions.

6 Treatment options

6.1 Oropharyngeal dysphagia

There are few treatment options for oropharyngeal dysphagia, because the neurological and neuromuscular disturbances that produce it can rarely be corrected through pharmacological or surgical therapy. Notable exceptions are the medical treatment of Parkinson's disease and myasthenia. The management of complications is of paramount importance. In this regard, identifying the risks of aspiration is a key element when considering treatment options.

Nutrition and diet. Diet change, with softer foods, and postural measures are helpful. Oral feeding is best whenever possible. Modifying the food consistency to thicken fluids and the soft foods can make an important difference [8]. Care must be taken to monitor fluid and nutritional needs (dehydration risk). Addition of citric acid to feedings improves swallowing reflexes, possibly on account of increased gustatory and trigeminal stimulation of acid [9]. Adjuvant treatment with an angiotensin-converting enzyme inhibitor to facilitate cough reflex may also be helpful [10].

If there is a high risk of aspiration, or when oral intake does not provide adequate nutritional status, alternative nutritional support should be considered. A fine-bore soft feeding tube can be passed down under radiological guidance. Gastrostomy feeding post-stroke reduces the mortality and improves nutritional status in comparison with nasogastric feeding. Percutaneous endoscopic gastrostomy involves passing a gastrostomy tube into the stomach via a percutaneous abdominal route under guidance from an endoscopist, and if available is usually preferable to surgical gastrostomy. The probability that feeding tubes may be eventually removed is lower in patients who are elderly, suffer a bilateral stroke, or aspirate during the initial video-fluoroscopic study [11].

Surgical treatments aimed at relieving the spastic causes of dysphagia, such as cricopharyngeal myotomy, have been successful in up to 60% of cases, but their use remains controversial [12]. On the other hand, removal of a mechanical impediment such as a large, compressing Zenker's diverticulum often helps.

Swallowing re-education. Various swallowing therapy techniques have been developed to help facilitate impaired swallowing. These include strengthening exercises, biofeedback, and thermal and gustatory stimulation.

6.2 Esophageal dysphagia

Table **2** provides a list of management options for esophageal dysphagia that may be taken into consideration.

Table **2** Management options for esophageal dysphagia

Condition	Conservative treatment	Invasive treatment
Diffuse esophageal spasm	Nitrate, calcium-channel blockers	Serial dilations or longitudinal myotomy
Achalasia	Soft food, anticholinergics, calcium channel blockers	Dilation, botulinum toxin injections, Heller's myotomy
Scleroderma	Antireflux, systemic medical management of scleroderma	None
Peptic stricture	Anti-reflux drugs (H_2 blockers, proton-pump inhibitors)	Dilation; fundoplication
Infectious esophagitis	Antibiotics (nystatin, acyclovir)	None
Pharyngoesophageal	None	Endoscopic or external repair in addition to cricopharyngeal

(Zenker's) diverticulum		myotomy
Schatzki's ring	Soft food	Dilation

6.2.1 Peptic esophageal strictures

Peptic strictures are usually the result of gastroesophageal reflux disease (GERD)– but they can also be caused by medication. The differential diagnosis has to exclude:

- Caustic strictures after ingestion of corrosive chemicals
- Drug-induced strictures
- Postoperative strictures
- Fungal strictures

After confirmation by endoscopy, dilation is the treatment of choice, and the procedure is described below.

Esophageal strictures should be dilated in a progressive manner with flexible Savary bougies or balloons. The choice among dilator types should be based on the availability of the dilators in a given institution and on the operator's experience and comfort in using them, because published experience has not convincingly established the superiority of one dilator type over another.

If dilation is performed with bougies, the first bougie passed should have a diameter approximately equal to that estimated for the stricture. Bougies of progressively increasing diameter are introduced until resistance is first encountered, after which no more than two additional bougies are passed during any one session. If balloon dilators are used, the initial dilation usually should be limited to a diameter of no more than 45 Fr. The extent of initial stricture dilation does not seem to influence either stricture recurrence or the requirement for subsequent dilation, so there is little support for the concept that strictures should be dilated aggressively to prevent recurrence. The extent of dilation in an individual patient should be based on the symptomatic response to therapy and on the difficulties encountered during the dilation procedure. Most patients experience good relief of dysphagia with dilation to a diameter between 40 Fr and 54 Fr. Strictures generally should not be dilated to a diameter beyond 60 Fr.

Aggressive antireflux therapy with proton-pump inhibitors or fundoplication improves dysphagia and decreases the need for subsequent esophageal dilations in patients with peptic esophageal strictures. For patients whose dysphagia persists or returns after an initial trial of dilation and antireflux therapy, healing of reflux esophagitis should be confirmed endoscopically before dilation is repeated. When healing of reflux esophagitis has been effected, the need for subsequent dilations is determined empirically. Patients who experience only short-lived relief of dysphagia after dilation can be taught the technique of self-bougienage. For refractory strictures a trial of steroid injection of the stricture can be considered. Rarely, truly refractory strictures require esophageal resection and reconstruction. Exceptionally endoluminal prosthesis may be indicated in benign strictures [13].The risk of rupture is about 0.5%. Surgery is generally indicated if frank perforation occurs.

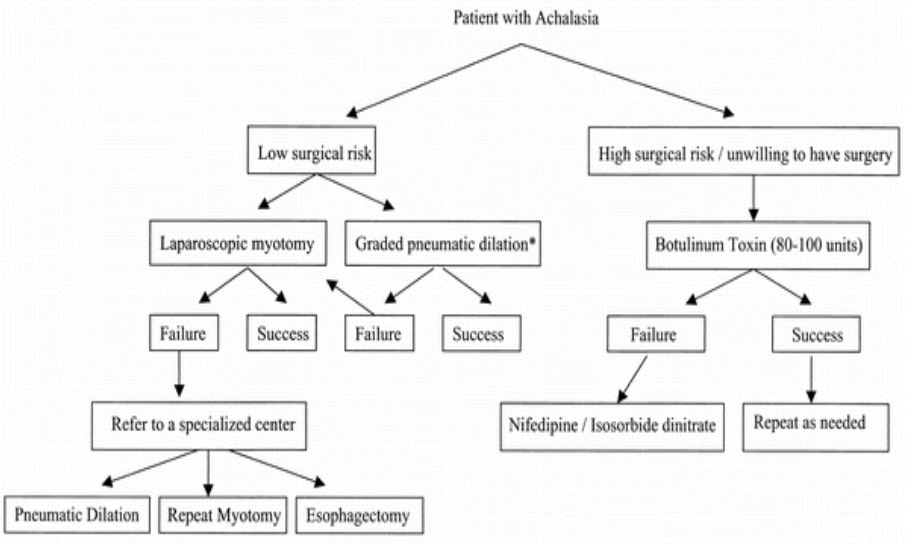
6.2.2 Treatment of lower esophageal mucosal rings (Schatzki's rings)

Dilation therapy for lower esophageal mucosal rings involves the passage of a single large bougie or balloon (45–60 Fr) aimed at fracturing (rather than merely stretching) the rings. After abrupt dilation, any associated reflux esophagitis is treated aggressively. The need for subsequent dilations is determined empirically. However, recurrence of dysphagia is likely, and patients should be advised that repeated dilation probably will be needed in the future. Esophageal manometry is recommended for patients whose dysphagia persists or returns quickly despite adequate dilation and antireflux therapy. For patients with a treatable motility disorder such as achalasia, therapy is directed at the motility problem. If no treatable motility disorder is found, endoscopy is repeated to confirm that esophagitis has healed and that the ring has been disrupted. For patients with persistent rings, another trial of abrupt dilation usually is warranted. Refractory rings that do not respond to abrupt dilation using standard balloons and bougies may respond to pneumatic dilation with large balloons (those used to treat achalasia), endoscopic electrosurgical incision, and surgical resection. These therapies should be required only rarely for patients with lower esophageal mucosal rings, and only after other causes of dysphagia have been excluded.

6.2.3 Achalasia

The management of achalasia depends largely on surgical risk. A low-risk endoscopic procedure such as botulinum toxin injection, often effective but with only temporary effects (usually 6 months or less), is reserved for patients totally excluded from surgery. For those in whom surgery is an option, most gastroenterologists will start with pneumatic dilation by endoscopy (about 6% risk of perforation) and opt for laparoscopic Heller type myotomy on those in whom two forced dilations fail. Some gastroenterologists prefer to opt directly for surgery without a prior trial of forced dilation. Figure **4** shows the algorithm for management options and courses.

Fig. **4** Management options in patients with achalasia



Medical therapy with nitrates or calcium channel blockers is often ineffective or poorly tolerated. Botulinum toxin injection may be used as initial therapy for patients

who represent poor surgical risks if the clinician judges that medications and bougienage would be poorly tolerated. Botulinum toxin injection appears to be a safe procedure that can induce a clinical remission for at least 6 months in approximately two-thirds of patients with achalasia. However, most patients will need repeated injections to maintain the remission; about two-thirds of patients in remission at 6 months will remain in remission at 1 year, despite repeated injections. When these treatments have failed, the physician and patient must decide whether the potential benefits of pneumatic dilation or myotomy outweigh the substantial risks that these procedures pose for elderly or infirm patients. A feeding gastrostomy is a safer alternative than pneumatic dilation or myotomy, but many neurologically intact patients find life with a gastrostomy unacceptable.

7 References

1 Atmatzidis K, Papaziogas B, Pavlidis T, Mirelis CH, Papaziogas T. Plummer–Vinson syndrome. Dis Esophagus 2003;16:154–7 (PMID: 12823219).

2 Chang YC, Chen SY, Lui LT, Wang TG, Wang TC, Hsiao TY, et al. Dysphagia in patients with nasopharyngeal cancer after radiation therapy: a videofluoroscopic swallowing study. Dysphagia 2003;18:135–43 (PMID: 12825907).

3 Scharitzer M, Pokieser P, Schober E, Schima W, Eisenhuber E, Stadler A, et al. Morphological findings in dynamic swallowing studies of symptomatic patients. Eur Radiol 2002;12:1139–44 (PMID: 11976859).

4 Barkhausen J, Goyen M, von Winterfeld F, Lauenstein T, Arweiler-Harbeck D, Debatin JF. Visualization of swallowing using real-time TrueFISP MR fluoroscopy. Eur Radiol 2002;12:129–33.

5 Ramsey DJ, Smithard DG, Kalra L. Early assessments of dysphagia and aspiration risk in acute stroke patients. Stroke 2003;34:1252–7 (PMID: 12677020).

6 Perlman AL, Witthawaskul W. Real-time remote telefluoroscopic assessment of patients with dysphagia. Dysphagia 2002;17:162–7 (PMID: 11956842).

7 Pikus L, Levine MS, Yang YX, Rubesin SE, Katzka DA, Laufer I, et al. Videofluoroscopic studies of swallowing dysfunction and the relative risk of pneumonia. AJR Am J Roentgenol 2003;180:1613–6 (PMID: 12760930).

8 Wilkinson TJ, Thomas K, MacGregor S, Tillard G, Wyles C, Sainsbury R. Tolerance of early diet textures as indicators of recovery from dysphagia after stroke. Dysphagia 2002;17:227–32 (PMID: 12140651).

9 Pelletier CA, Lawless HT. Effect of citric acid and citric acid-sucrose mixtures on swallowing in neurogenic oropharyngeal dysphagia. Dysphagia 2003;18:231–41 (PMID: 14571326).

10 Marik PE, Kaplan D. Aspiration pneumonia and dysphagia in the elderly. Chest 2003;124:328–36 (PMID: 12853541).

11 Ickenstein GW, Kelly PJ, Furie KL, Ambrosi D, Rallis N, Goldstein R, et al. Predictors of feeding gastrostomy tube removal in stroke patients with dysphagia. J Stroke Cerebrovasc Dis 2003;12:169–74.

12 Gervais M, Dorion D. Quality of life following surgical treatment of oculopharyngeal syndrome. J Otolaryngol 2003;32:1–5 (PMID: 12779254).

13 Pouderoux P, Verdier E, Courtial P, Bapin C, Deixonne B, Balmes JL. Relapsing cardial stenosis after laparoscopic Nissen treated by esophageal stenting. Dysphagia 2003;18:218–22 (PMID: 14506988).

8 Useful web sites and guidelines

- Medical position statement on the management of oropharyngeal dysphagia. Gastroenterology 1999;116;452–78: http://www2.us.elsevierhealth.com/scripts/om.dll/serve?action=open&location=gast/aga-pps&id=gast&group=toc
- Diagnosis and management of achalasia. Practice guideline. Am J Gastroenterol 1999;94:3406–12: http://www-east.elsevier.com/ajg/issues/9412/ajg1639fla.htm
- ACR appropriateness criteria for imaging recommendations for patients with dysphagia. Radiology 2000;215(Suppl):225–30: http://www.guideline.gov/FRAMESETS/guideline_fs.asp?guideline=002485&sSearch_string=dysphagia
- Management of patients with stroke, III: identification and management of dysphagia. SIGN Guideline No. 20; pilot edition, November 1997: www.sign.ac.uk
- Diagnosis and treatment of swallowing disorders (dysphagia) in acute care stroke patients. (ACHPR-99-E023. Rockville: AHCPR, 1999): www.achpr.gov
- Louay Omran, dysphagia: http://www.healthandage.com/PHome/gm=20!gsq=dysphagia!gid2=478
- Clinical use of esophageal manometry. AGA medical position statement, reviewed 2001: http://www.guideline.gov/FRAMESETS/guideline_fs.asp?guideline=000533&sSearch_string=dysphagia
- Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration. American Society of Anesthesiologists. Anesthesiology 1999;90:896–905: http://www.guideline.gov/FRAMESETS/guideline_fs.asp?guideline=001080&sSearch_string=dysphagia

9 Queries and feedback

The Practice Guidelines Committee welcomes any comments and queries that readers may have. Do you feel we have neglected some aspects of the topic? Do you think that some procedures are associated with extra risk? Tell us about your own experience. You are welcome to click on the link below and let us know your views.

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