



Management & Evaluation of Dysphagia

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D*ysphagia* is defined as a sensation of impaired passage of food from the mouth to the esophagus and the stomach. *Oropharyngeal dysphagia* usually is defined as the inability to *initiate* the act of swallowing (ie, to pass food and secretions from the mouth to the esophagus); and *esophageal dysphagia* (transfer dysphagia) is the sensation of a “holdup” in passing solids or liquids from the throat to the stomach. Normally such transit is not perceived so that any sense of blockage must be investigated promptly. The incidence of dysphagia has been estimated to be as great as 60% in nursing home residents¹ and can result from dysfunction or obstruction anywhere from the mouth to the stomach. Dysphagia should be distinguished from *odynophagia* (painful swallowing) usually associated with inflammation, although the two conditions may coexist.

Swallowing consists of a series of coordinated events involving many muscles, several cranial nerves, as well as a cortical swallowing center that propels the passage of food and secretions into the esophagus. Importantly, this transit of material from the mouth into the esophagus also must protect and not compromise the airway.

Physiology and Pathophysiology

Oropharyngeal transfer of a bolus in the mouth requires intact lingual peristalsis, the glossopalato sphincter mechanism, pharyngeal peristalsis, and coordinated and adequate opening of the upper esophageal sphincter (UES). During the oral phase, contact of the tongue with the hard and soft palate induces a peristaltic pressure wave that propels the bolus into the pharynx. Patients with cerebrovascular accidents and other cerebral neurological conditions have difficulty initiating this phase or controlling lingual functions. Such patients also may have sensory defects in the pharynx and the protective airway reflexes may be altered predisposing to aspiration. These sequential events have been beautifully described in sev-

eral publications.^{2,3} Sensory afferent signals from the oropharyngeal cavity are carried by the glossopharyngeal and vagus nerves to a central organizing center in the medulla, which has been termed the “swallowing center,” that processes efferent signals and programs motor swallowing sequences. Motor efferent signals are carried by the trigeminal, facial, and hypoglossal nerves. As food passes from the pharynx into the esophagus, contractions of the upper esophageal skeletal muscles propel the bolus into the mid-esophagus. At the same time the lower esophageal sphincter (LES) relaxes until the food bolus passes into the stomach. A number of changes in the physiology of this process have been described in healthy elderly

TABLE I Medications Causing Dysphagia

BY DIRECT MUCOSAL INJURY

Antibiotics
 Nonsteroidal anti-inflammatory drugs
 Alendronate
 Potassium chloride
 Quinidine
 Ferrous sulphate
 Clinitest tablets

DRUGS LOWERING LOWER ESOPHAGEAL SPHINCTER PRESSURE

Theophylline
 Calcium channel antagonists
 Nitrates
 Butylscopolamine
 Progesterone

DRUGS INDUCING XEROSTOMIA

Anticholinergics
 Angiotensin-converting enzyme inhibitors
 Antihistamines
 Diuretics
 Antipsychotic drugs
 Opiates

individuals, which are thought to represent the “normal aging process.”^{4,5}

Clinical Features

It generally is believed that a careful history and physical examination can provide sufficient information to make a diagnosis of the cause of dysphagia in most cases.⁶ Thus, patients with oropharyngeal dysphagia classically present with complaints of trouble initiating a swallow, multiple swallows, regurgitation through the nose, choking, coughing, nasal speech, postprandial dysarthria, and aspiration.⁷ Recurrent pneumonia may suggest frequent aspiration,⁸ avoidance of social dining may suggest spillage of food from the mouth, a careful history of medication usage may indicate an agent that contributes to difficulty in swallowing (Table I), and hoarseness can be caused either by unilateral paralysis of the vocal cords or inflammation of the glottis because of frequent aspiration. The correct diagnosis of such problems requires laryngeal examination.

A sensation of food sticking in the throat often reflects inadequate clearance of the bolus from the pharynx. This sensation is caused by the presence of large amounts of residue in the pyriform sinus or valleculae, or occasionally an obstructive lesion of the proximal distal esophagus. Thus, patients with oropharyngeal dysphagia should have their entire esophagus evaluated. Direct visualization of the hypopharynx should rule out inflammation, abrasions, or tumors. Screening procedures have been advocated to help diagnostic accuracy and risk of aspiration.⁹

In patients who have primarily esophageal dysphagia, the crucial clinical questions are whether the “holdup” is with solids, liquids, or both, if the symptoms are intermittent or progressive, and if there are associated symptoms of pain, such as heartburn, or of weight loss. Motor disorders (motility disorders) usu-

ally present with dysphagia involving both solids and liquids from the onset, whereas obstructing anatomic lesions usually cause dysphagia with solids initially, which progresses to trouble with liquids with time.

Algorithms for a diagnostic approach to patients with dysphagia have been found to be helpful in clinical practice.⁶

Table II presents a list of the types of disorders that may be associated with oropharyngeal dysphagia. Neurologic disorders include cerebrovascular

TABLE II Causes of Oropharyngeal Dysphagia

NEUROLOGIC

Cerebrovascular accidents
Parkinsonism
Alzheimer’s disease
Multiple sclerosis
Tumors
Peripheral neuropathy
(Poliomyelitis and postpolio syndrome)

MUSCULAR/NEUROMUSCULAR

Polymyositis, dermatomyositis
Muscular dystrophy
Alcoholic myopathy
Myasthenia gravis

STRUCTURAL

Carcinoma
Cricopharyngeal achalasia
Zenker’s diverticulum
Hyperostosis of the cervical spine
Postcricoid web

PHARMACOLOGIC AGENTS

Anticholinergics
Phenothiazines

METABOLIC

Thyrotoxic-associated myopathy
Cushing’s disease

accidents and Alzheimer's disease as well as parkinsonism and diseases of the peripheral nervous system, including poliomyelitis and postpolio syndrome.

Patients with Parkinson's disease may present with problems initiating a swallow or with the pooling of saliva in the mouth. Nearly half of patients with Parkinson's disease have some dysphagia,¹⁰ and almost all show impairment of the oral and pharyngeal phases of swallowing during videofluorography. Many also show incomplete relaxation of the upper esophageal sphincter. In some hands, treatment using a combination of swallowing therapy and drug therapy has been shown to be effective in this disease.¹¹ Dysphagia also may be prominent in patients with Alzheimer's disease in whom no specific abnormality often can be detected.^{12,13}

Polymyositis and *dermatomyositis* are the important inflammatory disorder of striated muscles involving the pharynx and upper third of the esophagus. Such patients often present with oropharyngeal dysphagia or aspiration, and this may be the first symptom of these diseases. It is one of the few causes of oropharyngeal dysphagia that is readily susceptible to treatment with agents such as immunosuppressive drugs (corticosteroids or methotrexate). Such therapy is effective in relieving symptoms in most patients.¹³ Alcoholism and the consequent alcoholic myopathy, which can affect any striated muscle, often may be missed in elderly patients.

Myotonic dystrophy involves the striated muscles of the pharynx and the esophagus and is usually accompanied by incomplete UES relaxation. Impaired pharyngeal contraction amplitude and esophageal manometric abnormalities are often present. Cricopharyngeal myotomy usually improves dysphagia in these conditions.^{14,15}

It is important to recognize that dysphagia may result from a variety of conditions that cross many

disciplines. Thus, physical examination involves a detailed neurological evaluation that aims to define changes in striated muscles, signs of ptosis in myasthenia gravis, calcinosis and Raynaud's phenomenon in the CREST syndrome, and scleroderma. In addition, common disorders that may be responsible for oropharyngeal dysphagia and which may be diagnosed during the physical examination include thyromegaly and thyrotoxicosis, and previous radiation therapy of the head and neck.

Structural abnormalities include carcinoma of the pharynx and larynx and hyperostosis of the cervical spine. *Cricopharyngeal dysfunction*, also called cricopharyngeal achalasia, can be caused by abnormalities of the UES with premature closure or delayed relaxation. A radiologic feature is the cricopharyngeal bar. Since this finding occurs in asymptomatic individuals, whether it is a true cause of dysphagia is controversial.¹⁶ Cricopharyngeal myotomy, botulinum toxin injection,¹⁷ or alternatively, bouginage with large dilators should be restricted to those individuals with this radiologic abnormality when no other cause of esophageal or pharyngeal dysphagia can be found.

Zenker's diverticulum is formed by the protrusion of the hypopharyngeal mucosa posteriorly at the boundary of the transverse fibers of the cricopharyngeal muscle and the oblique oriented fibers of the inferior pharyngeal constrictor. It is thought that reduced muscle compliance increases pressures generated in the hypopharynx to push the swallowed bolus through a rigid UES. Diagnosis is made by barium swallow, and treatment consists of cricopharyngeal myotomy with or without diverticulectomy.

Pharmacologic agents occasionally may cause the disorder of the oropharyngeal region, including such drugs as anticholinergics and phenothiazines.¹⁸ Metabolic causes such as those associated with thyrotoxicosis and Cushing's disease are extremely rare.

Causes of esophageal dysphagia are summarized in Table III. Obstructive lesions such as carcinoma of the esophagus and strictures, webs, and foreign bodies are best diagnosed by esophagoscopy. Lower esophageal rings such as Schatzki's ring and esophageal webs occasionally may be diagnosed by barium esophagram, and foreign bodies may be removed if found at esophagoscopy. Mediastinal masses such as carcinoma of the lung and vascular structural lesions obstructing the esophagus are readily diagnosed by radiography.

Motor disorders including achalasia and scleroderma are usually diagnosed by barium esophagram since both of these conditions cause dilatation of the body of the esophagus. They may be distinguished by careful evaluation of the LES. Esophageal spastic disorders, which may present with severe dysphagia, must be distinguished from the common tertiary contractions seen so often in elderly patients, the

“cork screw esophagus.” This is not believed to cause dysphagia, although there is some evidence that swallowed pills may be held up in the esophageal body, fail to pass into the stomach, and may be the cause of pill-induced esophagitis.¹⁹

Inflammatory disorders that cause dysphagia include peptic esophagitis inflammation due to monilial infection (candidiasis) and more rarely are due to viral infections. Radiation also will produce an inflammatory esophagitis.

An important diagnostic component is the use of videofluoroscopic recording of a modified barium swallow.^{20,21} Real-time video recordings are often crucial with patients swallowing boluses of different volumes and consistencies. Such an evaluation not only can define the cause of the dysphagia but also provides information about failure of airway protection resulting in pulmonary aspiration. Newer therapeutic techniques including biofeedback and swallowing maneuvers can be evaluated in real time under direct vision.

There may be inadequate clearance of the barium bolus from the mouth resulting in residual barium and difficulty in initiating the swallowing. Fluoroscopy may define weak muscle activity, drooling, and premature spilling of oral contents into the pharynx before the pharyngeal phase is activated. The pharyngeal phase may be abnormal due to reduced upward and forward movement of the larynx and hyoid bone, an incompetent pharyngeal closure mechanism, and a cricopharyngeal bar—a finding which, however, may be seen in up to 5% of asymptomatic patients older than 40 years of age.

Gastroesophageal Reflux Disease

Dysphagia may be the presenting symptom of gastroesophageal reflux disease (GERD),²² which appears to be more common in the elderly than in

TABLE III Causes of Esophageal Dysphagia

OBSTRUCTIVE LESIONS

Tumors
 Strictures
 Lower esophageal (Schatzki's) ring
 Webs
 Foreign bodies
 Vascular structural lesions
 Mediastinal masses

MOTOR DISORDERS

Spastic disorders
 Achalasia
 Scleroderma

INFLAMMATORY DISORDERS

Peptic esophagitis
 Monilial esophagitis
 Radiation

the young.²³ Complications such as severe erosive esophagitis stricture and Barrett's esophagus clearly are much more common in advanced age.²⁴ An important reason why GERD appears to be more severe in the elderly is that pain often is absent or is severely muted²⁵⁻²⁷ or that partially incapacitated elderly patients tend to ignore reflux symptoms. GERD results from a lowering of LES pressure and more frequent transient LES relaxation, permitting more frequent and prolonged reflux of gastric contents into the esophagus. Importantly in the elderly, many medications reduce LES pressure, and the incidence of hiatus hernias, which can enhance symptoms, is more common than in the young.²⁸ Furthermore, clearance into the stomach of refluxed gastric contents that pass into the esophagus requires vigorous esophageal contractions. Such contractions may be lower in advanced age.²⁹

The clinical picture of GERD in addition to dysphagia may include bronchospasm, hoarseness, cough, and dyspnea as well as recurrent pneumonia. In addition, weight loss may be a presenting symptom in the elderly.³⁰ The diagnosis of esophagitis that accompanies GERD is best made by esophagoscopy. Esophagoscopy not only can detect the presence of esophagitis but importantly is the method that defines its severity and the presence of peptic strictures.

Therapy of peptic esophagitis that accompanies GERD depends on gastric acid suppression using proton pump inhibitors, which should be used in full dosages in the elderly, together with positional maneuvers to ensure that patients take food and medications in the upright position with adequate amounts of fluids. In most cases, medical treatment must be continued for several months, and in some cases indefinitely. When strictures are present, they respond to bouginage or balloon dilatation. It is important to evaluate whether medications taken by

older nursing home individuals may be contributing to reflux by reducing the lower esophageal sphincter pressure.³¹ Moniliasis is a frequent cause of esophagitis in the elderly,³² although it rarely causes dysphagia. Medications are an important cause for dysphagia. Medications can cause direct mucosal injury by lowering the pressure in the lower esophageal sphincter or through inducing xerostomia resulting in reduced production of saliva. Common medications causing such changes are shown in Table I.

Management of Dysphagia

The goals of treatment of dysphagia are to improve bolus transport and nutritional status, and to eliminate aspiration. Therefore, a search for structural lesions that are amenable to surgical or endoscopic treatment must first be pursued. In patients with oropharyngeal dysphagia, therapy often is quite limited. Specific therapy for patients with parkinsonism, substitute therapy for hypothyroidism, and immunomodulatory therapy for polymyositis and dermatomyositis may improve the dysphagia. Stroke victims show considerable recovery in swallowing, particularly with early nutritional and physiotherapy assistance.³³ Later, percutaneous endoscopic gastrostomy (PEG) is indicated despite the fact that long-term nutritional improvement is not achieved adequately.³⁴ In milder cases, care with regulating the character of foods (eg, semi-solids rather than liquids) or rotation of the head to the paralyzed side may improve swallowing function best with the help of speech therapists.³⁵

Upper esophageal sphincter or cricopharyngeal myotomy is the standard therapy for cricopharyngeal dysfunction syndromes and Zenker's diverticulum. Although endoscopic therapy has been performed and botulinum toxin is sometimes employed,¹⁵ the classical surgical approach still appears optimal.¹⁴ Finally, a pathophysiologic biofeedback and physio-

therapy approach to treatment of patients with oropharyngeal dysphagia, even when they are tube feeding-dependent, has allowed many patients to swallow independently.³⁶ Patients with inadequate glottis closure resulting in frequent aspiration may benefit from injection of a nonabsorbable material such as Teflon[®].

Many patients with oropharyngeal dysphagia require rehabilitation techniques utilizing the combined therapeutic wisdom of speech therapists, primary care nurses, and physicians, and several postural approaches may be beneficial.²⁹ The solid data available to determine the best management of elderly nursing home residents with dysphagia are sparse. Recent calls for more research point the way to better care of this common clinical problem.^{37,38}

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