AGA Technical Review on Management of Oropharyngeal Dysphagia

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Oropharyngeal dysphagia has high morbidity, mortality, and cost. Although epidemiological data are scant, estimates of the prevalence of dysphagia among individuals older than 50 years range from 16% to 22%. Within health care institutions, it is estimated that 12%–13% of patients in short-term care hospitals and up to 60% of nursing home occupants have feeding difficulties. Of these, a substantial proportion are troubled by oral or pharyngeal as opposed to esophageal dysphagia. Similarly, special populations, such as those with head injuries, cerebrovascular accidents, or Parkinson's disease, have a 20%–40% prevalence of oropharyngeal dysphagia. The consequences of oropharyngeal dysphagia can be severe: dehydration, malnutrition, aspiration, choking, pneumonia, and death. Nursing home occupants with oropharyngeal dysphagia and aspiration have a 45% 12-month mortality. Thus, faced with the prevalence of oropharyngeal dysphagia among the elderly and the aging of the U.S. population, busy clinicians frequently confront the option of swallowing evaluation and rehabilitation. The alternative to swallowing rehabilitation is either enteral or parenteral alimentation, the costs of which can be enormous.

Several attributes of oropharyngeal dysphagia contribute to its clinical complexity: (1) because of accompanying neurological impairment, patients often have a limited ability either to communicate their subjective distress or to cooperate with their evaluation and therapy; (2) because oropharyngeal dysphagia is usually a manifestation of a systemic disease rather than a disease specific to the oropharynx, the clinician must be cognizant both of the diverse etiological possibilities (Table 1) and of the appropriate level of requisite investigation for the individual patient; (3) because oropharyngeal dysphagia is often the result of a functional rather than mucosal or structural aberration (as is often the case with esophageal dysphagia), a thorough understanding of normal oropharyngeal swallowing physiology is a prerequisite for understanding of dysfunctional swallowing; and (4) deglutition is a rapid, anatomically complex neuromuscular response that requires unique methodologies specifically tailored to its clinical evaluation. Together, these attributes define oropharyngeal dysphagia as a multidimensional syndrome often residing at the "fringe" of several professional domains: radiology, speech language pathology, neurology, otolaryngology, gastroenterology, oncology, and physiatry, to name the most commonly involved specialties. For a given patient, the input of one clinician may rightfully dominate, but for consideration of the overall diversity of potential patients and therapies, the most common institutional strategy has been to organize a dysphagia team to facilitate communication and cooperation among contributing clinicians with an otherwise diverse focus. Inevitably, however, each involved specialty is prone to develop its own biases regarding the appropriate methods for evaluation and treatment. Recognizing the extreme sensitivity of these issues in today's health care environment, it is not the intent of this paper to define who should perform which evaluations; rather, we will focus on which evaluations and treatments should be rendered to optimize patient treatment. The specifics of how that care is rendered necessarily vary among institutions.

In seeking to formulate recommendations for a patient-oriented approach to the evaluation and management of oropharyngeal dysphagia, we were immediately confronted with the magnitude and diversity of the medical literature. Searching MEDLINE under the subject headings deglutition and deglutition disorders, as well as the key words deglutition, dysphagia, dysphagic, swallow, and choking, the number of citations identified exceeds 600 for each year since 1990. Thus, it was necessary to adopt a selective, as opposed to inclusive, approach to this literature. First, this was accomplished by outlining a systematic clinical approach to the dysphagic patient and then selecting key illustrative references to either highlight the logic of that approach or substantiate advocated interventions. Second, editorial emphasis was placed on critical analysis of more current and controversial concepts requiring a broader perspective of the literature.

Throughout this technical review, we qualify our recommendations according to the strength of supporting data. Available data on the diagnostic and therapeutic choices for management of oropharyngeal dysphagia are
Table 1. Representative Causes of Oropharyngeal Dysphagia

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
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<tbody>
<tr>
<td>Iatrogenic</td>
<td>Medication side effects (chemotherapy, neuroleptics, etc.)</td>
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<tr>
<td>Postsurgical</td>
<td>muscular or neurogenic</td>
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<td>Radiation</td>
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<tr>
<td>Corrosive</td>
<td>(pill injury, intentional)</td>
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<td>Infectious</td>
<td>Diptheria</td>
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<td></td>
<td>Botulism</td>
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<td>Lyme disease</td>
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<td>Syphilis</td>
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<td></td>
<td>Mucositis (herpes, cytomegalovirus, candida, etc.)</td>
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<td>Metabolic</td>
<td>Amyloidosis</td>
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<td></td>
<td>Cushing’s syndrome</td>
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<td>Thyrotoxicosis</td>
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<td>Wilson’s disease</td>
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<td>Myopathic</td>
<td>Connective tissue disease (overlap syndrome)</td>
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<td></td>
<td>Dermatomyositis</td>
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<td></td>
<td>Myasthenia gravis</td>
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<td>Myotonic dystrophy</td>
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<td>Oculopharyngeal dystrophy</td>
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<td>Polymyositis</td>
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<td>Sarcoïdosis</td>
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<td>Paraneoplastic syndromes</td>
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<td>Neurological</td>
<td>Brainstem tumors</td>
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<td>Head trauma</td>
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<td>Stroke</td>
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<td>Cerebral palsy</td>
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<td>Guillain-Barré syndrome</td>
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<td>Huntington’s disease</td>
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<td>Multiple sclerosis</td>
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<td>Polio</td>
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<td>Postpolio syndrome</td>
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<td>Tardive dyskinesia</td>
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<td>Metabolic encephalopathies</td>
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<td>Amyotrophic lateral sclerosis</td>
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<td>Parkinson’s disease</td>
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<td>Dementia</td>
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<td>Structural</td>
<td>Cricopharyngeal bar</td>
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<td></td>
<td>Zenker’s diverticulum</td>
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<td>Cervical webs</td>
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<td>Oropharyngeal tumors</td>
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<td></td>
<td>Osteophytes and skeletal abnormalities</td>
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<td>Congenital (cleft palate, diverticula, pouches, etc.)</td>
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Oropharyngeal Dysphagia

Current knowledge of the swallowing mechanism is derived mainly from radiographic studies, which have been in use since the early 1900s. Plain films of the pharynx were replaced in the 1930s by cineradiography, which was subsequently replaced by videofluoroscopy in the 1970s. Videofluoroscopy is less cumbersome than cineradiography, involves less radiation exposure, and permits instant analysis of bolus transport, aspiration, and pharyngeal function. Using this descriptive method, deglutition can be divided into four phases: (1) oral preparatory phase, (2) oral voluntary phase, (3) pharyngeal phase, and (4) esophageal phase. The oral stages of deglutition entail mastication, bolus formation, and bolus transfer. Typically, as the bolus passes the anterior faucial arches, pharyngeal swallowing begins. The pharyngeal swallowing response is a rapid, highly coordinated activity that results in velopharyngeal closure, laryngeal elevation and closure, opening of the upper esophageal sphincter (UES), tongue loading, tongue pulsion, and pharyngeal clearance. The esophageal phase is made up of peristaltic action through the esophagus. This treatise focuses only on the oral and pharyngeal phases of swallowing.

Oropharyngeal dysphagia results from either oropharyngeal swallowing dysfunction or perceived difficulty in published evidence, when available. However, faced with the limitations of existing data, some of the ensuing treatise is based on expert opinion. In these instances, recommendations were based on combined weighing of evidence from the most rigorous studies available along with available indirect evidence such as (1) biological plausibility based on observations from physiological studies; (2) extrapolation from relevant studies even though they were not specifically designed to address the question at hand (e.g., uncontrolled studies, case series, natural history studies); and (3) authors’ opinions and opinions of experts in the field, which usually reflect current best practice. However, it is also important to acknowledge at the outset that this is not an unusual circumstance in clinical medicine; of necessity, clinicians frequently practice according to a pattern based largely on what has worked in the past, rather than according to the best available clinically relevant research. The lack of high-level evidence supporting efficacy is not sufficient reason to abandon current best clinical practice. The experienced clinician uses a combination of clinical expertise and the best available evidence–based medicine because neither alone is sufficient to dictate decisions on treatment of individual patients.
the process of swallowing. Major categories of dysfunction are (1) an inability or excessive delay in initiation of pharyngeal swallowing, (2) aspiration of ingestate, (3) nasopharyngeal regurgitation, and (4) residue of ingestate within the pharyngeal cavity after swallowing. Each of these categories of dysfunction can be mechanistically subcategorized using fluoroscopic and/or manometric data. Because our understanding of the perception of dysphagia is minimal, the current state of the art with oropharyngeal dysphagia management aims at detecting, quantifying, and attempting to correct these functional manifestations of dysphagia. A prerequisite for this is an understanding of normal oropharyngeal swallowing.

Normal Oropharyngeal Swallowing

The sensory cues that elicit pharyngeal swallowing are not precisely known, but based on experimental evidence, entry of fluid or food into the hypopharynx in the sensory receptive field of the superior laryngeal nerve seems to be the crucial stimulus. On the other hand, swallowing can also be initiated solely by volitional effort if there is food or fluid within the oral cavity. Therefore, the required afferent signal for initiation of the swallowing response is a mixture of both peripheral sensory input from oropharyngeal afferents and superimposed control from higher nervous system centers. Neither element of the afferent signal is capable of initiating swallowing without some contribution from the complementary element, as evidenced by the inability to swallow during sleep in which the higher centers are disconnected or with deep anesthesia to the oral cavity, in which peripheral afferents are deactivated. However, the relative contribution of the two afferent elements responsible for initiation of swallowing varies with circumstances. Cerebrovascular accident or head injury in which one or the other neural substrate of the afferent signal has been damaged can result in a relative inability to initiate swallowing.

Once initiated, pharyngeal swallowing results in a transient reconfiguration of the pharynx, illustrated at rest in Figure 1. Mechanistically, several closely coordinated actions are involved: (1) elevation and retraction of the soft palate with closure of the nasopharynx, (2) UES opening, (3) laryngeal closure at the level of the laryngeal vestibule, (4) tongue loading (ramping), (5) tongue pulsion, and (6) pharyngeal clearance. These events occur in close synchrony, as shown in the sequence of radiographs and computer reconstructions in Figure 2. A fundamental aspect of deglutitive pharyngeal reconfiguration is in transforming the oropharynx from a respiratory to a swallowing pathway by opening the inlet to the esophagus and sealing the inlet to the larynx. Laryngeal vestibule closure, and hence airway protection during swallowing, is achieved by laryngeal elevation and anterior tilting of the arytenoid cartilages against the base of the epiglottis. Laryngeal elevation is quantifiable fluoroscopically by vertical movement of the tracheal air column and its persistence above the critical height necessary to achieve closure of the laryngeal vestibule. Videofluoroscopic studies done concurrently with manometry have shown that UES relaxation occurs during swallowing-associated laryngeal elevation and that relaxation of the sphincter precedes opening by approximately 0.1 seconds. After relaxation, UES opening results from anterior traction caused by contraction of the suprahyoid and infrahyoid musculature, evident fluoroscopically by anterior hyoid movement. Clinically, this is a significant point in that impaired UES opening can result from either impaired traction on the sphincter or impaired sphincter relaxation; instances of impaired traction should be evident fluoroscopically by diminished anterior hyoid displacement, whereas impaired relaxation is only detectable manometrically.

The functional elements of pharyngeal swallowing responsible for bolus transport are the propulsive phase of tongue action and of propagated contraction of the pharyngeal constrictors. Bolus propulsion relies heavily on deglutitive tongue action, transpiring between times 0.00 and 0.13 in Figure 2. The geometric complexity of the tongue requires biplane or even three-dimensional imaging to visualize its function, which consists largely
Figure 2. The oropharyngeal swallow as imaged by videofluoroscopy and reconstructed in three dimensions with computer graphics. From left to right, each horizontally arranged group of images contains the three-dimensional reconstruction of the pharyngeal cavity and surrounding structures, the lateral radiographic appearance of the pharynx during a 10-mL barium swallow, the corresponding posterior-anterior radiographic appearance, and a magnified view of the hypopharynx at the time indicated at the left. Time 0.00 is the instant of UES opening; the entire sequence of events transpires within 1 second. The metal sphere under the chin is used to correlate among images. In the magnified hypopharyngeal reconstructions, 1 is the epiglottis, 2 is the laryngeal vestibule, 3 is the arytenoid cartilage, 4 is the esophagus, and 4' is the pyriform sinus after closure of the UES. Note the importance of laryngeal elevation during the pharyngeal reconfiguration and synchrony of UES opening with laryngeal vestibule closure.
of deforming the central groove to create a bolus cavity while maintaining a seal peripherally and then rapidly expelling the contents of that bolus cavity into the opened esophageal inlet.\textsuperscript{24–27} In coordination with the expulsion phase of tongue activity, the propagated pharyngeal contraction progresses from the superior to middle to inferior pharyngeal constrictors at approximately 15 cm/s, stripping almost all residue from the pharynx and hypopharynx except for trace amounts that may be left in the valleculæ or pyriform sinuses, thereby minimizing the chance that aspiration will occur with resumed respiration.\textsuperscript{28} The overall pattern of pharyngeal swallowing is similar among bolus volumes, but the period of reconfiguration into an alimentary conduit is prolonged while a larger bolus chamber is formed by accentuated tongue deformation (Figure 3).\textsuperscript{27} After bolus clearance, the process of pharyngeal reconfiguration is reversed, allowing resumption of respiration; the entire pharyngeal swallow occurs in approximately 1 second.

### Approach to the Dysphagic Patient

Clinical assessment of patients with suspected oropharyngeal dysphagia should address five fundamental questions that then permit the clinician to establish investigational priorities:

1. Does the patient describe dysphagia as opposed to globus sensation or hyposalivation?
2. Is the dysphagia oropharyngeal or esophageal in origin?
3. Is the dysphagia caused by a structural or functional disorder?
4. How severe is the dysphagia and are complications present?
5. Is there an underlying related or causative disease?

An accurate distinction between esophageal dysphagia, pharyngeal dysphagia, globus sensation, and xerostomia can usually be made on the basis of a careful history. In some cases, the underlying cause of oropharyngeal dysphagia can also be diagnosed reliably on the basis of history and examination.

### Patient History

Dysphagia is defined as difficulty in swallowing. The purely sensory symptom of globus is sometimes erroneously equated with dysphagia. Globus is a common, nonpainful sensation of a lump or fullness in the throat, of unknown etiology, in which deglutitive food bolus transport is unimpaired.\textsuperscript{29,30} Indeed, globus sensation is usually most apparent to the patient between meals, is not necessarily related to the act of swallowing, and is usually alleviated by eating. When associated with dysphagia, chest pain, or heartburn, globus may be related to esophageal dysmotility.\textsuperscript{31} In the absence of an accompanying specific diagnosis after careful physical examination and laryngoscopy, the best treatment for patients with globus sensation is explanation and reassurance.

Xerostomia is frequently accompanied by dysphagia...
and is common in the elderly, present in 16% of men and 25% of women. Dysphagia is attributed to loss of the moistening and lubricating qualities of saliva. Dry mouth may be accompanied by dry eyes, inflammatory arthropathy, a history of head and neck radiotherapy, or concurrent medications with anticholinergic side effects. A detailed drug history is also important because a number of centrally acting drugs can impair oropharyngeal function and can cause tardive dyskinesia with masticatory and swallowing difficulties, as well as toxic or inflammatory myopathy or dysfunctional neuromuscular transmission (see below).

In cases of pharyngeal dysphagia, bolus holdup is reported in the cervical area. However, distal esophageal obstruction can give rise to a sensation of the bolus catching in either the retrosternal region or the cervical region in 15%-30% of cases. Hence a perception by the patient of apparent bolus holdup in the neck has low diagnostic specificity, and cervical localization per se does not help the clinician distinguish pharyngeal from esophageal causes of dysphagia. If dysfunction relates to the oral or pharyngeal region, the patient can localize accurately the site of such dysfunction, which correlates well with radiographic localization of the problem. Dysphagia solely for solids is indicative of a structural lesion such as a stricture, ring, web, or tumor. However, the distinction between dysphagia for liquids and solids is of little diagnostic value in distinguishing oropharyngeal dysphagia from esophageal dysphagia; the specific type of pharyngeal dysfunction, rather than the presence or absence of pharyngeal dysfunction, dictates which bolus type generates most symptoms. The ability to expectorate (rather than regurgitate or vomit) the offending bolus is more indicative of pharyngeal than esophageal dysphagia.

Patients with oropharyngeal dysphagia may have oral dysfunction, pharyngeal dysfunction, or both. Typical symptoms of oral dysfunction might include drooling from the mouth or spillage of food because of poor labial and facial muscle function, inability to chew or propel the bolus from the mouth, siaorrhea or xerostomia, difficulty initiating swallowing, piecemeal swallowing, and dysarthria. Typical symptoms of pharyngeal dysfunction include an immediate sense of bolus holdup localized to the neck; nasal regurgitation; the need to swallow repeatedly to clear food or fluid from the pharynx; coughing or choking during meals; suggesting aspiration; gurgly voice; and dysphonia. Pain on swallowing or persistent sore throat may indicate malignancy, infection, or inflammation from corrosive agents or ionizing radiation. Immediate expectoration of an offending bolus is indicative of bolus retention in the hypopharyngeal or cricopharyngeal region. Delayed regurgitation of old food is typical of a large pharyngeal diverticulum.

Oropharyngeal dysphagia usually has a neuromuscular basis. A history of stroke is often obtained. Symptoms of bulbar muscle dysfunction or other brainstem symptoms, such as vertigo, nausea, vomiting, hiccuping, tinnitus, diplopia, or drop attacks, should be sought. More widespread neuromuscular symptoms such as dysarthria, diplopia, limb weakness, or fatigability are variably present in motor neuron disease, myasthenia, and myopathy. The patient may report tremor, ataxia, or unsteadiness, which might indicate an underlying movement disorder such as Parkinson's disease, or dysphagia may be the first symptom of a neurological disease.

The circumstances of symptom onset, duration, and progression of dysphagia also provide useful diagnostic information. The duration of dysphagia is often an important clue to whether the underlying cause is benign or malignant. Malignant dysphagia usually presents with a relatively short progressive history and is frequently associated with weight loss. A sudden onset of dysphagia, often in association with other neurological symptoms or signs, usually indicates a cerebrovascular cause such as stroke. A subacute or more insidious onset is more consistent with disorders such as inflammatory myopathy, myasthenia, and amyotrophic lateral sclerosis.

Physical Examination

Aims of physical examination in dysphagic patients are to (1) identify features of underlying systemic or metabolic disease when present; (2) localize the neuroanatomic level and severity of a causative neurological lesion when present; and (3) detect adverse sequelae such as aspiration pneumonia or nutritional deficiency. The patient's general nutritional state and body weight are important indicators of disease severity. Examination of the chest may reveal signs of respiratory infection or increased secretions, suggesting significant aspiration, which is common in patients with pharyngeal dysphagia. Eye signs, sweating, tremor, and tachycardia may be present in patients with thyrotoxic myopathy. Dysphagia may be the first symptom of a neurological disease. Examination of the neck may reveal signs of respiratory infection or increased secretions, suggesting significant aspiration, which is common in patients with pharyngeal dysphagia. Eye signs, sweating, tremor, and tachycardia may be present in patients with thyrotoxic myopathy. In addition to the dysphagia assessment, evaluation by a speech language pathologist will provide further information about language, cognitive and behavioral dysfunction, and strength and range of movement of the muscles involved in speech and swallowing. This information will directly influence decisions about patients' suitability for swallowing therapy and the type of therapy adopted.

A neurological examination is mandatory in the evaluation of oropharyngeal dysphagia of unknown etiology. Physical findings might indicate cranial nerve dysfunction, neuromuscular disease, cerebellar dysfunc-
Disease duration, severity, and specific parkinsonian features did not correlate with the severity or mechanism of dysphagia. Muscle fasciculation, ptosis, wasting, and weakness or fatigability should be sought to detect underlying motor neuron disease, myopathy, or myasthenia. Dysphagia is a frequent (sometimes presenting) symptom of inflammatory myopathy, occurring in 30%-60% of cases. Pharyngeal dysphagia can also occur in up to 20% of cases of mixed connective tissue disease overlap syndrome, which may include clinical and serological features of Sjögren’s syndrome, lupus, or scleroderma.

Myasthenia gravis is a relatively common neuromuscular disease with an estimated prevalence as high as 1 in 20,000. Facial and pharyngeal weakness is present in 70% and significant dysphagia affects 30%-60% of myasthenics. Dysphagia is noted at diagnosis in approximately 20% of cases and may dominate the clinical picture; dysphagia is the sole presenting symptom in 6%-17% of affected individuals. Because myasthenic patients presenting with dysphagia do not always have the typical ocular signs, this treatable disorder must always be considered. Tensilon (edrophonium) stimulation test results may still be positive in such instances. The diagnosis may also be made electromyographically using either repetitive nerve stimulation or single-fiber EMG recordings, the most sensitive diagnostic test. A number of pharmacological agents, including botulinum toxin, penicillamine, large doses of aminoglycosides, and procainamide, can produce a reversible myasthenic syndrome; in some such cases, AChR antibodies are also detectable.

Diagnosis of inflammatory myopathies is made by determination of abnormalities of one or more serological muscle enzymes, by EMG, or by muscle biopsy. Abnormalities in serum creatinine phosphokinase (CPK) levels are the most sensitive enzymatic indicator, but elevations in alanine aminotransferase, aspartate aminotransferase, lactate dehydrogenase, and aldolase levels are also sensitive indicators of muscle injury. At the time of presentation, CPK levels are elevated in approximately 70% of cases; CPK levels are elevated at some point in the course of the disease in 95% of cases. However, CPK levels can be normal in patients with active disease. Antinuclear antibody is detectable in only approximately 25% of patients but is much more likely to be present (85%-
myopathy may present as dysphagia,72 and the cholesterol—considered. For example, amiodarone-induced thyroid toxic or inflammatory myopathy should also be confined to the pharyngeal musculature.69 If cricopharyngeal inflammatory process may be quite focal and is occasion-ation of the site for muscle biopsy is important because the referral confirmation of the diagnosis by the above techniques, muscle biopsies should be performed on the cricopharyngeus and other neck muscles (e.g., omohyoid, sternomastoid) for histological examination.

Toxic and metabolic myopathies should be considered as a potential cause of oropharyngeal dysphagia. Hypothyroidism should always be considered, particularly in elderly patients in whom the more classical thyrotoxic features may be absent, because it is a reversible cause of dysphagia.35,70 In contrast to myxedema myopathy, in which weakness is uncommon but CPK levels are usually increased, serum CPK levels are not increased with thyrotoxicosis.71 A number of drugs capable of causing reversible toxic or inflammatory myopathy should also be considered. For example, ami-darone-induced thyroid myopathy may present as dysphagia,72 and the cholesterol-lowering HMG-CoA reductase inhibitors are a well-known cause of toxic myopathy.73

Investigations to Characterize Oropharyngeal Dysphagia

As mentioned earlier, categories of oropharyngeal swallowing dysfunction are (1) an inability or excessive delay in initiation of pharyngeal swallowing, (2) aspiration of ingestate, (3) nasopharyngeal regurgitation, and (4) residue of ingestate within the pharyngeal cavity after swallowing. The consequences of swallowing dysfunction parallel its severity, ranging from subjective dysphagia to wasting, aspiration pneumonia, and even death. Because clinical evaluation of suspected oropharyngeal dysphagia aims to identify and quantify these parameters of dysfunction, we assess the utility of the available clinical tools by their ability to achieve this objective. The brief ensuing discussion is limited to methodologies that have been widely applied clinically.

Videofluoroscopic evaluation of oral and pharyngeal function. A protocol for a videofluoroscopic swallowing evaluation, frequently referred to as a modified barium swallow, has been described by Logemann.13 The key distinction between this type of evaluation and a standard radiographic evaluation of the oropharynx is that the videofluoroscopic evaluation is structured to detect and analyze functional impairment of the swallowing mechanism. A series of swallows of varied volumes and consistencies of contrast material are imaged in a lateral projection, framed to include the oropharynx, palate, proximal esophagus, and proximal airway. Studies are recorded on videotape to permit instant replay, in slow motion if necessary, and examination of both the presence and mechanism of swallowing dysfunction. As such, the videofluoroscopic study provides evidence of all four categories of oropharyngeal swallowing dysfunction alluded to above: (1) inability or excessive delay in initiation of pharyngeal swallowing, (2) aspiration of ingestate, (3) nasopharyngeal regurgitation, and (4) residue of ingestate within the pharyngeal cavity after swallowing. Furthermore, the procedure allows for testing of the efficacy of compensatory dietary modifications, postures, swallowing maneuvers, and facilitatory techniques in correction or observed dysfunction. On the other hand, videofluoroscopy does not permit quantification of pharyngeal contractile forces, detection of incomplete UES relaxation (which can occur despite normal opening), or quantification of the magnitude of intrabolus pressure during swallowing.

Nasoendoscopy. Nasoendoscopy is performed by passing a small fiberoptic or videendoscope transnasally, permitting direct visualization of all mucosal surfaces of the oral cavity, nasopharynx, pharynx, and larynx. This examination is the optimal method for identification of intracavitary structural lesions and for both identification and biopsy of mucosal abnormalities. Swallowing assessment can also be done as an extension of a standard endoscopic examination of the mucosal surfaces.74 Liquids and foods colored with food dye are swallowed. Feasibility studies suggest that nasoendoscopy can identify (1) an inability or excessive delay in initiation of pharyngeal swallowing and (2) residue of ingestate within the pharyngeal cavity after swallowing. Although aspiration is not directly imaged, accumulated oropharyngeal secretions and dye staining of the subglottic airway offer indirect evidence.74,75 The endoscope is positioned (1) at the level of the soft palate to view the tongue base, epiglottis, and superior pharyngeal wall and (2) below the epiglottis to view the laryngeal vestibule. Recently,
nasoendoscopy has been adapted to also assess the integrity of pharyngeal sensation using discrete air pulse stimuli to evoke glottic adduction. Reduced sensation in the hypopharynx of stroke victims can be demonstrated using this technique.

Manometry. Intraluminal manometry, performed using a transnasally positioned manometric assembly, can quantify the strength of pharyngeal contraction, the completeness of UES relaxation, and the relative timing of these two events. These may be useful ancillary findings in some pathological conditions such as the quantification of pharyngeal weakness in oculopharyngeal muscular dystrophy. Technical considerations make pharyngeal manometry more complex than esophageal manometry because of transducer requirements for high-fidelity recording, extreme longitudinal and radial asymmetry of intraluminal pressures recorded from within the pharynx during swallowing, and unpredictable structural movements during the pharyngeal swallowing response that have the effect of displacing the pressure sensor from its preswallowing position. When manometry is performed as a stand-alone test, it offers only indirect evidence of the key swallowing outcomes alluded to above.

Manofluorography. Manometry performed concurrently with videofluoroscopy permits the integration of manometric data with fluoroscopic observations; this technique has been called manofluorography. The technique requires an accurate method of synchronizing manometric and fluoroscopic recordings for manofluorography to realize its full potential. When suitable synchronization is accomplished, manofluorography overcomes the key weaknesses of manometry performed in a stand-alone manner: (1) inability to be certain of sensor location with respect to pharyngeal structures at a given instant in the recording and (2) inability to distinguish between recordings of intrabolus pressure as opposed to recordings from within a closed lumen. Manofluorography can detect all of the key swallowing outcome measures alluded to above (as can videofluoroscopy) and correlate fluoroscopic events with manometric data, thereby providing the method with the potential to identify subcategories of swallowing dysfunction. In particular, impaired UES opening can be distinguished from impaired relaxation, and weak propulsive pharyngeal forces can be distinguished from increased outflow resistance as manifest by high intrabolus pressure.

Selection Among Management Options

Clinical, functional, and other laboratory evaluations are selected to provide the clinician with a diagnosis of the underlying cause of dysphagia in many cases and a clear idea of the specific mechanisms of oropharyngeal dysfunction in all cases. In most instances, this evaluation can be accomplished by the combination of history, physical examination, direct or indirect laryngoscopy, and a videofluoroscopic swallowing study, which must be viewed as the primary evaluation modalities for oropharyngeal dysphagia. The video barium swallow also provides information about aspiration risk for a variety of bolus volumes and consistencies. Further testing is then applied selectively, depending on the specific issues raised by this evaluation. How does this information influence management decision making and ultimately clinical outcome? Management decisions potentially influenced by swallowing evaluation are broadly categorized in Table 2. Each of these will be considered in the following sections along with available efficacy data. However, influencing management and improving clinical outcome are not synonymous. In some instances, the goal of the clinical evaluation is to institute the accepted best practice of management, irrespective of whether this has been demonstrated to improve outcome.

Dysphagic syndromes amenable to surgery or dilation. Identification of a functionally significant structural lesion will lead to specific management in most cases. For example, pharyngeal or cricopharyngeal strictures, oropharyngeal tumors, posterior pharyngeal diverticulum, and cervical web are indications for either surgery, dilatation, or antineoplastic therapy, or a combination of these treatments. These local lesions are generally readily detected radiographically and/or endoscopically. Identification of one of these lesions will dictate surgery or dilation. In the setting of benign stenoses or webs, simple dilatation is very safe and effective. Although no controlled trials exist, dilatation of benign stenoses or webs is recommended on the basis of consistent level C evidence (Table 3) combined with obvious biological plausibility and strong opinions of the authors and other experts in the field that this reflects current best practice.

Although it is clear that strictures or webs compromising the proximal esophageal lumen cause dysphagia and are amenable to dilatation, the clinician should be wary of other “abnormalities” identified by the radiologist for...

Table 2. Potential Findings From Clinical Evaluation of Oropharyngeal Dysphagia That Can Direct Management

| Characterization of dysphagic syndromes amenable to surgery or dilation |
| Severe aspiration necessitating the institution of nonoral feeding |
| Identification of an underlying systemic disease |
| Identification of a specific pattern of oropharyngeal dysfunction that has specific diagnostic, therapeutic, or prognostic implications |
which the evidence linking the radiographic finding with dysphagia is less convincing. These radiographic anomalies include cricopharyngeal bars, prominent cervical osteophytes or skeletal hyperostoses, and lateral pharyngeal diverticula.

A cricopharyngeal bar is a common radiographic finding, the significance of which is controversial. Cricopharyngeal bars have been reported in 5%-19% of patients undergoing dynamic pharyngeal radiography, making it somewhat inevitable that cricopharyngeal bars will be identified in many patients with dysphagia, regardless of whether the bar causes the dysphagia. In a series of 124 patients, cricopharyngeal bars were identified in 24 (19%), but esophageal abnormalities is likely to exceed that of the cricopharyngeus, making it somewhat inevitable that cricopharyngeal bars can relieve dysphagia.101 Acknowledging the above reservations, it is the opinion of the authors that a cricopharyngeal bar can assume pathophysiological and functional significance when either (1) alternative esophageal or pharyngeal abnormalities have been ruled out by extensive radiographic, manometric, and endoscopic evaluation or (2) coexistent pharyngeal neuromuscular dysfunction is identified, in which case the functional significance of the combined abnormalities is likely to exceed that of the cricopharyngeal bar in isolation.

Cervical osteophytes are a common incidental finding in the dysphagic patient, occurring in 6%-30% of elderly patients.103,104 Only 0.7% of patients with cervical disc disease report dysphagia.105 Nonetheless, if prominent, osteophytes can cause dysphagia that is correctable by surgical removal of the bony spur.106-108 The major mechanism of dysphagia is mechanical compression of the posterior pharyngeal wall, but associated periesophageal inflammation induced by pharyngoesophageal motion over the cervical exostoses (predominantly over the C3–C6 vertebrae) may contribute as well.109,110 Development of osteophytes may be related to DISH (diffuse idiopathic skeletal hyperostosis ankylosing spondylitis), infectious spondylitis, previous

Table 3. Critique of Efficacy Data on Use of Cricopharyngeal Myotomy (or Dilation) for Structural Cricopharyngeal Disorders

<table>
<thead>
<tr>
<th>Population (intervention)</th>
<th>Design (level of evidence)</th>
<th>Outcome measures</th>
<th>Excellent or good responses</th>
<th>Efficacy? (grade of recommendation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zenker’s (myotomy)102</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Not stated</td>
<td>8/8 (100%)</td>
<td>Yes (C); morbidity not stated</td>
</tr>
<tr>
<td>Zenker’s (myotomy)106</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>10/10 (100%)</td>
<td>Yes (C); 0% morbidity</td>
</tr>
<tr>
<td>Cervical webs (dilation)133</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Not stated</td>
<td>7/14 (7)</td>
<td>Yes (C); 1 perforation (died), 49 dilations required in 14 patients</td>
</tr>
<tr>
<td>Zenker’s (myotomy)107</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>4/4 (100%)</td>
<td>Yes (C); 0% morbidity</td>
</tr>
<tr>
<td>Zenker’s (myotomy)108</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>92/100 (92%)</td>
<td>Yes (C); 24% operative morbidity; mean follow-up, 4 yr</td>
</tr>
<tr>
<td>Zenker’s, n = 37; web/stenosis, n = 10 (myotomy)90</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Standardized questionnaire</td>
<td>32/39 (82%)</td>
<td>Yes (C); 9% operative morbidity</td>
</tr>
<tr>
<td>Zenker’s, n = 37; web/stenosis, n = 10 (myotomy)90</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Global assessment</td>
<td>35/37 (95%)</td>
<td>Yes (C); 23% operative morbidity; recurrent dysphagia in 2 (1.5 and 3 yr), 14/60 mixed structural/neurogenic</td>
</tr>
<tr>
<td>Cervical webs (dilation, n = 45; myotomy, n = 12)104</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Dietary modification</td>
<td>Dilatation 50% Myotomy 75%</td>
<td>Yes (C); 0% morbidity; diverticulum in 4, precise number with dysphagia unclear</td>
</tr>
<tr>
<td>Zenker’s (myotomy)103</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>17/18 (94%)</td>
<td>Yes (C); morbidity not stated; mean follow-up, 30 mo, 44% have residual diverticula (no correlation with symptoms)</td>
</tr>
<tr>
<td>Zenker’s (myotomy)102</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>7/7 (100%)</td>
<td>Yes (C); 0% morbidity</td>
</tr>
</tbody>
</table>

*Postoperative morbidity included wound infection, salivary fistula, recurrent laryngeal nerve damage, and pneumonia.
surgical fusion of the cervical vertebrae, or local trauma or be of idiopathic origin.\textsuperscript{111,112} Surgical treatment of cervical osteophytes remains controversial, and appropriate objective criteria have not been consistently used to assess results. Complications of surgical excision include vocal fold paralysis or paresis, vertebral disc prolapse, fistula, hematoma, infection, aspiration, and Horner’s syndrome.\textsuperscript{113} In view of these limitations, most believe surgery should be performed only for those with severe dysphagia in whom conservative treatment has failed.\textsuperscript{111} Small lateral pharyngeal diverticula are also a commonly encountered incidental radiographic anomaly to which symptoms may be erroneously attributed. Lateral diverticula are found on the wall of the midpharynx, usually at the level of the vallecula, and should not be confused with the typical posterior, hypopharyngeal diverticulum (Zenker’s), which arises at the proximal margin of the cricopharyngeus. Lateral diverticula may be congenital or, more commonly, acquired. Congenital lateral pouches are true branchial cleft cysts, representing an embryological remnant of the third pharyngeal pouch corresponding to the thyrohyoid membrane.\textsuperscript{114} A acquired lateral pharyngeal pouches or pharyngoceles are frequently bilateral, are more common in the elderly, and protrude through an area of relative weakness in the thyrohyoid membrane at a site that is relatively poorly supported by cartilage or muscle. The area of relative weakness is bounded by the hyoid bone superiorly where there is incomplete overlap of the thyrohyoid muscle anteriorly and the inferior constrictor muscle inferiorly.\textsuperscript{115,116} Lateral diverticula are such frequent incidental findings that it is controversial whether they cause symptoms; one study reported lateral diverticula in 51% of asymptomatic individuals.\textsuperscript{117} In another report examining the relationship between lateral diverticula and dysphagia, an alternative cause for dysphagia could usually be identified, frequently in the esophagus.\textsuperscript{98} Nonetheless, there are sporadic case reports of successful alleviation of dysphagia after surgical ligation or removal of the lateral diverticulum.\textsuperscript{118,119} Cricopharyngeal myotomy. Cricopharyngeal myotomy, with the potential to reduce both resting sphincter tone and resistance to flow across the UES, is the most common surgical treatment for oropharyngeal dysphagia.\textsuperscript{86,92,120} Actually, resting UES tone is not abolished by myotomy, but it is reduced by roughly 50%, suggesting that the derived benefit is probably from increased sphincter opening and decreased resistance to transsphincteric flow.\textsuperscript{92,121} Myotomy is most efficacious when applied to patients with structural disorders that limit opening of the cricopharyngeus in association with preserved pharyngeal contractility.\textsuperscript{90,122} These conditions are met with postcricoid stenosis, webs, and Zenker’s diverticulum. Simple dilatation can afford benefit of variable duration in Zenker’s diverticulum,\textsuperscript{123–125} but myotomy is the essential element for successful long-term relief of dysphagia.\textsuperscript{122,126,127} Good or excellent responses can be expected in 80%–100% of Zenker’s patients treated by transcervical myotomy combined with diverticulectomy or diverticulopexy (Table 3). Myotomy via the endoscopic route is used less commonly but is an effective alternative treatment for Zenker’s diverticulum. Endoscopic therapy and can be performed with a rigid esophagoscope under general anesthesia\textsuperscript{128–130} or under conscious sedation using a flexible endoscope.\textsuperscript{131} The endoscopic technique has traditionally been reserved for elderly patients with significant comorbidity; however, mediastinitis, hemorrhage, or restenosis complicates approximately 6% in the largest series.\textsuperscript{129} Data supporting efficacy of either dilatation or myotomy for cervical esophageal webs and postcricoid stenosis are also consistently favorable, albeit less clear-cut and, again, uncontrolled (Table 3). Repeated dilations over many years seem to be required in at least 40% of such patients, and in one study, 20% eventually required myotomy.\textsuperscript{83,84} Based on the consistency of available (albeit level C) efficacy data (Table 3), the profound benefit of myotomy in treatment of structural cricopharyngeal disorders, the measurable improvement in sphincter opening and reduction in resistance to bolus flow in such cases,\textsuperscript{92,121} and the clinical impression of those adopting this treatment, there are sufficient grounds to presume that cricopharyngeal myotomy is beneficial in cases of dysphagia caused by structural lesions.

In marked contrast to the high efficacy of myotomy for structural cricopharyngeal disorders, the efficacy of myotomy in neurogenic dysphagia is variable. Our MEDLINE search retrieved 71 reports on the efficacy of cricopharyngeal myotomy from 1981 to June 1997. After exclusion of reports focused on structural lesions, postsurgical dysfunction, head and neck cancer resections, or velopharyngeal incompetence, there were 5 review articles and 15 original reports examining the efficacy of myotomy in neurogenic dysphagia; these studies are summarized in Table 4.\textsuperscript{85,87,90,120,132–142} As shown in Table 4, none of these are controlled trials, and many did not specify outcome measures, instead relying on subjective assessment of dysphagia severity and raising the issue of reporting bias. For example, a study reporting an excellent response to myotomy in 50% of patients found no significant change in postoperative pharyngeal isotope clearance.\textsuperscript{132} Combining of data from these 15 studies yields an overall favorable response rate of 63% and average operative mortality of 1.8%.
Several reports attempt to define specific measures or observations predictive of response to cricopharyngeal myotomy. Although radiographic studies readily detect impaired deglutitive UES opening, they are inherently limited by their inability to distinguish between failed UES relaxation and compromise of the sphincter opening mechanism. Manometric measures could conceivably make this distinction. Intuitively, manometrically defined failed UES relaxation or incoordination in conjunction with preservation of the pharyngeal contraction might infer a favorable response to myotomy. However, the data supporting this contention are uncontrolled and methodologically weak, using only qualitative descriptions of incomplete UES relaxation, UES incoordination, and "intact" pharyngeal contraction.\textsuperscript{87,133,136} Furthermore, conflicting data suggest that if pharyngeal propulsive activity is normal or near normal, bolus transport is unimpaired despite failure of UES relaxation.\textsuperscript{46} Conversely, the virtual absence of pharyngeal contractile activity may still be compatible with maintenance of oral feeding without a myotomy.\textsuperscript{144} Seeking to improve on these limitations, most current investigations have sought to combine the strengths of manometry and radiography, using these methods concurrently to quantify deglutitive hypopharyngeal intrabolus pressure as an indirect indicator of resistance to flow across the UES.\textsuperscript{100,102} Such studies suggest that preoperative hypopharyngeal intrabolus pressure is significantly greater in individuals responding to cricopharyngeal myotomy than in nonresponders.\textsuperscript{137} Nonetheless, precisely which manometric or radiographic measures are optimal predictors of benefit from cricopharyngeal myotomy remains a focus of investigation.

In summary, there are no absolute indications for cricopharyngeal myotomy, and, ultimately, clinical decisions need to be made on a case-by-case basis. The strongest indication exists in patients with a combination of dysphagia and stenosis of the cricopharyngeus, especially with a coexistent hypopharyngeal diverticulum. Currently available clinical data do not strongly support treatment with cricopharyngeal myotomy for neuro-

### Table 4. Critique of Efficacy Data on the Use of Cricopharyngeal Myotomy in Neuropathic and Myopathic Dysphagia

<table>
<thead>
<tr>
<th>Population</th>
<th>Design (level of evidence)</th>
<th>Outcome measures</th>
<th>Excellent or good responses</th>
<th>Efficacy? (grade of recommendation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurogenic\textsuperscript{132}</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Global assessment, aspiration</td>
<td>20/ 40 (50%)</td>
<td>Yes (C); operative mortality 2.5%</td>
</tr>
<tr>
<td>Neuromyogenic\textsuperscript{133,134}</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Dietary consistency</td>
<td>21/ 38 (55%)</td>
<td>Yes (C); subjective assessment, operative mortality 0%</td>
</tr>
<tr>
<td>Neurogenic\textsuperscript{135}</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>17/ 23 (74%)</td>
<td>Yes (C)</td>
</tr>
<tr>
<td>Neurogenic\textsuperscript{136}</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Global assessment</td>
<td>12/ 20 (60%)</td>
<td>Yes (C); operative mortality 3%</td>
</tr>
<tr>
<td>Oculopharyngeal dystrophy\textsuperscript{120}</td>
<td>Uncontrolled case series (V)</td>
<td>Dysphagia frequency; aspiration; scintigraphy</td>
<td>7/ 15 (7%)</td>
<td>Yes (C); significantly reduced dysphagia; improved isotope clearance (% responders not stated); operative mortality 0%</td>
</tr>
<tr>
<td>Neurogenic\textsuperscript{137}</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>13/ 20 (65%)</td>
<td>Yes (C); CP disruption by either myotomy (n = 8) or dilatation (n = 12); operative mortality 0%</td>
</tr>
<tr>
<td>Neurogenic\textsuperscript{138}</td>
<td>Uncontrolled retrospective case series (V)</td>
<td>Global assessment</td>
<td>11/ 13 (85%)</td>
<td>Yes (C); operative mortality 0%</td>
</tr>
<tr>
<td>Neurogenic\textsuperscript{97}</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>8/ 12 (67%)</td>
<td>Yes (C); operative mortality 9% (1/11)</td>
</tr>
<tr>
<td>Parkinson’s\textsuperscript{143}</td>
<td>Uncontrolled case series (V)</td>
<td>Global assessment</td>
<td>4/ 4 (100%)</td>
<td>Yes (C); 2/ 4 also had Zenker’s; operative mortality 0%</td>
</tr>
</tbody>
</table>

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pathic or myopathic causes of oropharyngeal dysphagia. Although it is acknowledged that cricopharyngeal myotomy benefits roughly 50% of such patients, precisely which manometric or radiographic measures predict benefit in such cases remains unknown. Hence, there are currently no clear guidelines for preoperative selection of patients for myotomy.

Detection of severe aspiration necessitating nonoral feeding. Fundamental objectives of the videofluoroscopic swallowing study are to ascertain whether aspiration is occurring, to estimate the severity of aspiration, to determine the mechanism by which aspiration occurs, and to ascertain whether this is rectifiable by posture or compensatory strategy. Although there is currently no grading system for documentation of the severity of oropharyngeal dysphagia, there is universal agreement that aspiration represents severe dysfunction. Videofluoroscopy is believed to be the most sensitive test for detection of aspiration, but because it is the gold standard for this determination, this strong clinical impression is difficult to prove. Videofluoroscopy also allows the clinician to make a reliable qualitative assessment of aspiration severity based on features such as the presence or absence of a pharyngeal swallowing response, bolus consistency and volume susceptible to aspiration, and extent of bolus clearance from the pharyngeal and oral cavities.

Oropharyngeal dysphagia associated with aspiration and aspiration pneumonia is a frequent sequelae of stroke, occurring in one third of stroke patients overall and 67% of those with brainstem stroke. Because substantial spontaneous recovery is observed during the first 2 weeks after a stroke, it is prudent to delay long-term management decisions for that period. Among the subset of patients with poststroke dysphagia, aspiration pneumonia occurs in 43%–50% during the first year and has a mortality of up to 45%. Radiography detects aspiration not evident at the time of bedside assessment in 42%–60% of patients. Additionally, a poor gag reflex does not have prognostic value for aspiration because only 60% of aspirators have impaired gag reflexes. Similarly, dysphonia had a 91% sensitivity for aspirators but a positive predictive value of only 58%. Radiographic demonstration of delayed or absent pharyngeal swallowing response combined with poor pharyngeal contraction carries the highest risk of aspiration. These studies provide strong evidence that videofluoroscopy is the only way to be certain whether aspiration is occurring in the dysphagic patient. If fluoroscopy shows gross pharyngeal dysfunction with severe aspiration, immediate introduction of nonoral feeding is indicated.

Despite the logical association between deglutitive aspiration and the subsequent development of pneumonia, this sequence is not inevitable (Table 5). For example, in a case-control study, the incidence of pneumonia and death was 19% among 26 poststroke dysphagic patients with radiographically demonstrated aspiration compared with 3% in 33 case-matched controls without aspiration, yielding odds ratios of 7.6 for development of pneumonia in patients aspirating bolus of any consistency (P < 0.05) and 9.2 for death in those aspirating thickened liquids or more solid consistencies (P < 0.01). Other reports summarized in Table 5 are

<table>
<thead>
<tr>
<th>Population</th>
<th>Study design (level of evidence)</th>
<th>Pneumonia incidence (follow-up duration)</th>
<th>Positive predictive value for pneumonia</th>
<th>Negative predictive value for pneumonia</th>
<th>Significant findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke, n = 60</td>
<td>Uncontrolled, retrospective (V)</td>
<td>30% (12 mo)</td>
<td>68%</td>
<td>69%</td>
<td>Predictive of sooner development of pneumonia but not pneumonia incidence</td>
</tr>
<tr>
<td>Mixed neurogenic, n = 40</td>
<td>Uncontrolled, retrospective (V)</td>
<td>43% (12 mo)</td>
<td>50%</td>
<td>55%</td>
<td>Predictive for rehospitalization (82% positive predictive value) but not pneumonia</td>
</tr>
<tr>
<td>Stroke, n = 26</td>
<td>Retrospective case-control (III)</td>
<td>19% (18 mo)</td>
<td>19%</td>
<td>97%</td>
<td>Predictive for pneumonia; odds ratio for pneumonia, 7.6; odds ratio for death, 9.2</td>
</tr>
<tr>
<td>Stroke, n = 115</td>
<td>Randomized control trial (II)</td>
<td>7% (12 mo)</td>
<td>?</td>
<td>?</td>
<td>Not predictive of pneumonia; low incidence of pneumonia because of selection criteria; low statistical power</td>
</tr>
<tr>
<td>Stroke, n = 121</td>
<td>Uncontrolled, prospective (V)</td>
<td>25% (1 wk)</td>
<td>35%</td>
<td>84%</td>
<td>Not predictive of pneumonia or mortality</td>
</tr>
</tbody>
</table>

*p < 0.05; **p < 0.01; †not significant (our calculation contradicts that of authors).
less conclusive. For example, in the study of Johnson et al., although 29 of the 60 dysphagic post-cardiovascular accident patients developed pneumonia within 12 months, this was not correlated with either radiographic aspiration or pharyngeal pooling; the positive and negative predictive values of radiographic aspiration for subsequent pneumonia were only 68% and 69%, respectively (both nonsignificant). Similarly, Smithard et al. found that dysphagia after stroke significantly increased the risks of death, chest infections, poor nutritional status, institutionalization, and disability; however, videofluoroscopic detection of aspiration did not predict pneumonia risk. In another report, although radiographic aspiration was not a significant predictor of subsequent pneumonia, it did have predictive value for rehospitalization (82%).

Ironically, the strongest evidence supporting the contention that radiographic aspiration predicts the development of pneumonia comes from a randomized controlled trial of swallowing therapy that found no differences in incidence of pneumonia between radiographically defined aspirators and nonaspirators. However, aspirators developed pneumonia significantly sooner than nonaspirators, as did those who aspirated thick liquids (compared with thin liquids) and those who had aspiration after swallowing (compared with aspiration before or during swallowing). However, the selection bias in that investigation greatly diminished its statistical power for detection of differences in pneumonia incidence. After exclusion of patients who aspirated more than 50% of all consistencies or continued to aspirate more than 50% of all consistencies after use of compensatory swallowing strategies, the overall pneumonia incidence was only 7%, a small fraction of the value observed without that selection bias.

Although detection of dysphagia-related aspiration is viewed as an indication for nonoral feeding, tube feeding does not necessarily eliminate the risk of aspiration pneumonia. A provocative finding by Croghan et al. was that of the 22 patients with radiographic aspiration, the 15 who had feeding tubes placed had a significantly higher rate of pneumonia and pneumonia-related death than did the 7 who did not receive feeding tubes. This finding, along with the need to eliminate aspiration of oral secretions, prompts consideration of surgery in some cases. Surgical procedures aim to minimize or eliminate aspiration and enhance swallowed bolus clearance from the pharynx. Relatively conservative, speech-preserving surgical approaches include cricopharyngeal myotomy, epiglottoplasty, partial or total cricoid excision, laryngeal suspension, and vocal fold medialization. Vocal fold augmentation is advocated for a symptomatic open glottis caused by mechanical impairment or neuromuscular disability that prevents contralateral fold adduction. If vocal fold medialization is necessary for a relatively short period, as with recovery from neurological insult, an absorbable item such as Gelfoam, collagen, fat, or glycerine may be injected directly into the vocal fold. If there is no hope of functional improvement, Teflon, which is not absorbed and not easily removed from the larynx, may be injected into the vocal fold via direct laryngoscopy under local anesthesia. More radical approaches that eliminate continuity between airway and digestive tracts include cuffed tracheostomy, medialization of the vocal folds, glottic closure, laryngotracheal diversion, and laryngectomy. Placement of a cuffed tracheostomy can reduce aspiration but is generally insufficient to permit oral feeding in cases of severe aspiration.

In summary, accurate detection of aspiration requires videofluoroscopy. Although not indisputable, the balance of evidence suggests that detection of aspiration is a predictor of pneumonia risk and/or probability of rehospitalization (Table 5). Furthermore, based on the likelihood that radiographic findings predict pneumonia risk, the radiographic detection of aspiration dictates introduction of compensatory swallowing strategies, nonoral feeding, and in some instances corrective surgery. It is a usual practice to recommend nonoral feeding if the patient demonstrates radiographic aspiration on all food consistencies despite introduction of all appropriate treatment strategies (see below). However, there is little evidence that nonoral feeding reduces or eliminates the risk of aspiration. Further studies are needed to resolve existing contradictory findings and to clarify the other determinants of pneumonia risk, which probably include the level of patient alertness, pharyngolaryngeal sensory function, host defenses, and preexisting respiratory dysfunction.

Identifying underlying disease. An important potential outcome of swallowing evaluation is the identification of an underlying neuromuscular, neoplastic, or metabolic disorder that will dictate specific treatment or management of that disorder (Table 2). For example, dysphagia is commonly the presenting symptom in patients with myopathies and can be the presenting symptom in myasthenia, thyrotoxicosis, motor neuron disease, and Parkinson’s disease. In each instance, identification of the underlying condition results in a specific therapy. However, the response of the underlying disease in general, and of dysphagia in particular, is extremely variable. Whether identification of the underlying disorder influences outcome with respect to swallowing function depends on a number of factors, including: (1) whether clinical and laboratory findings are predictive of
severity, complications, comorbidity, or prognosis; (2) the natural history of the untreated underlying condition and its potential for recovery or progression; and (3) whether effective treatment is available and whether such treatment influences swallowing per se. Brief consideration of these issues in relation to potentially correctable underlying diseases is outlined below. Regardless of the responsiveness of the dysphagia to these treatments, the underlying condition itself is likely to warrant treatment for the other disease manifestations. It is also a firmly held conviction among practitioners, and an expectation among patients, that accurate diagnosis is an important medical goal, irrespective of whether an effective therapy exists.

In view of the 25% prevalence of failed UES relaxation with Parkinson’s disease, cricopharyngeal myotomy would seem to be a logical treatment. However, failure of UES relaxation was not correlated with either severity of the associated motor disorder or severity of dysphagia. Nonetheless, a favorable response to myotomy has been reported in one small series of 4 patients, many of whom had coexistent structural abnormalities (Table 4). However, more data are needed before surgery is recommended for this condition. Similarly, there has been little systematic evaluation of the responsiveness of dysphagia to anti-parkinsonian medication. The short-term effect of central dopaminergic stimulation on pharyngeal mechanics is controversial. An early cineradiographic study reported no difference in swallowing function after short-term administration of oral levodopa compared with placebo. A subsequent videofluoroscopic study found that central dopaminergic stimulation by apomorphine caused short-term improvement in pharyngeal bolus clearance in 50% of patients with mild disease. Bushmann et al. found that 15% of patients with Parkinson’s disease who had abnormal results on videofluoroscopic swallowing studies had short-term radiographic improvement and clinical symptom ratings after oral levodopa administration but that general improvement in parkinsonian motor features did not parallel changes in swallowing function. Persistent effects of pharmacological therapy for Parkinson’s on swallowing function also remain unclear. One study found that a combination of levodopa and swallowing therapy improved swallowing function, but it is uncertain to what extent improvement could be attributed to pharmacotherapy per se. It has been the experience of the authors that dysphagia is relatively unresponsive to anti-parkinsonian drug therapy. Nevertheless, evidence for a good clinical response to drug therapy exists in isolated case reports. A appropriate timing of medication, 1 hour before meals, would seem logical and was found to be beneficial in at least one case report.

Improvement of oropharyngeal dysphagia in response to medical therapy for myasthenia gravis (acetylcholinesterase inhibitor and/or immunosuppressive drugs) is variable and often less satisfactory than the response of other disease manifestations. Some have reported dramatic improvement in hypopharyngeal bolus clearance in response to edrophonium, but this has not been observed consistently. For example, in a report of 8 elderly men whose primary symptoms of myasthenia gravis were dysarthria and dysphagia, dysphagia improved satisfactorily in only 3; the other 5 required feeding tubes. Even in patients with myasthenia gravis who have a satisfactory clinical response, the videofluoroscopic improvement after therapy may be marginal, casting doubt on the use of videofluoroscopy in assessment of clinical response. Notwithstanding the frequently disappointing response to medical therapy, diagnosis of myasthenia does influence management because it always warrants specific pharmacological therapy, initiates a search for thymoma, and prompts avoidance of risk factors for myasthenic crisis to which the dysphagic patient is frequently exposed, e.g., respiratory tract infections, anesthesia, and surgery.

The mainstay of treatment for inflammatory myopathies is immunosuppressive therapy, initially with steroids, and subsequently with azathioprine or methotrexate as steroid-sparing agents. Despite the relative lack of controlled efficacy trials, a number of patients respond favorably to these agents. Inclusion body myositis is generally resistant to these therapies, but high-dose intravenous immunoglobulin may be helpful. Reports of improved swallowing function in response to pharmacological therapy are anecdotal but generally favorable. Similarly, although cricopharyngeal myotomy might have a role in treatment, there has been no systematic evaluation of this. In the case of thyrotoxicosis, the response of limb muscle function to specific treatment of thyrotoxicosis is usually excellent. Although published data are limited, there are also case reports of resolution of pharyngeal dysphagia with return to the euthyroid state. This has also been the anecdotal experience of the authors.

Specific patterns of dysphagia amenable to specific swallowing therapy. Current strategies of swallowing therapy involve modification of either eating behavior or swallowing technique. Modifications of swallowing technique are intended to strengthen weak oropharyngeal muscle groups, thereby improving their speed and range of movement, and/or to selectively modify the mechanics of swallowing to facilitate bolus flow and minimize aspiration. However, not all functional elements of oropharyngeal swallowing are amenable to
volitional modulation; to some degree, plausible techniques of swallowing therapy are limited by the neural pattern generator in the medulla, which orchestrates the sequencing of events within the swallow. Nonetheless, compensatory strategies can modify the geometry of the upper aerodigestive tract through changes in head position or exploit gravitational forces with postural changes to prevent aspiration and facilitate bolus clearance. Alternatively, corrective strategies aim to improve the function of selected elements within the swallow that are at least partially under volitional control (e.g., tongue action, hyoid motion). Commonly advocated strategies and the rationale for their use are listed in Table 6.13,162,177 An important caveat to the application of swallowing therapies is that other than dietary or postural modifications that can be instituted by the caregiver, their use requires adequately preserved cognitive function so that the patient can understand and comply with the maneuvers; in practice, this excludes a significant proportion of patients with neurogenic dysphagia.36

Application of specific swallowing therapies to patients with specific patterns of oropharyngeal dysphagia depends on accurate definition of the relevant mechanism of dysfunction. Of the available diagnostic modalities, videofluoroscopy is most applicable in this regard. A methodical videofluoroscopic swallowing study (1) defines the anatomy of the oropharynx; (2) detects dysfunction as evident by aspiration, poor clearance, or poor control of the bolus; (3) determines the mechanism responsible for that dysfunction; and (4) examines the short-term effects of therapeutic strategies designed to eliminate or compensate for that dysfunction.162 Although other investigations such as videoendoscopy, ultrasonography, manometry, electromyography, or combinations thereof may subsequently permit more focused analysis of a particular element within the overall swallowing mechanism, none of these investigations can substitute for videofluoroscopy. As a practical note emphasizing this point, in most instances the range of therapeutic possibilities has been defined by videofluoroscopy, as has the assessment of their efficacy. This becomes evident in the ensuing critiques of the literature on the biological

### Table 6. Swallowing Therapy Techniques, Indications, and Rationale

<table>
<thead>
<tr>
<th>Technique</th>
<th>Execution (rationale)</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary modification</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thickened liquids</td>
<td>Reduced tendency to spill over tongue base</td>
<td>Disordered tongue function</td>
</tr>
<tr>
<td>Thin liquids</td>
<td>Offers less resistance to flow</td>
<td>Preswallow spill/aspiration</td>
</tr>
<tr>
<td>Maneuvers</td>
<td></td>
<td>Impaired laryngeal closure</td>
</tr>
<tr>
<td>Supraglottic swallow</td>
<td>Breath hold, double swallow, forceful expiration (closes vocal folds before and during swallowing)</td>
<td>Aspiration: reduced/late vocal fold closure</td>
</tr>
<tr>
<td>Supersupraglottic swallow</td>
<td>Effortful breath hold (closes vocal folds before and during swallow)</td>
<td>Aspiration (poor closure of laryngeal introitus)</td>
</tr>
<tr>
<td>Effortful swallow</td>
<td>Increased anterior tilting of arytenoids</td>
<td>Poor posterior tongue base motion</td>
</tr>
<tr>
<td>Mendelsohn maneuver</td>
<td>Prolong hyoid excursion guided by manual palpation (prolongs UES opening)</td>
<td>Poor pharyngeal clearance and laryngeal movement</td>
</tr>
<tr>
<td>Postural adjustments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head tilt</td>
<td>Tilt posteriorly at swallow initiation (gravity clears oral cavity)</td>
<td>Poor tongue control</td>
</tr>
<tr>
<td></td>
<td>Tilt laterally to unaffected side (directs bolus down stronger side)</td>
<td>Unilateral pharyngeal weakness</td>
</tr>
<tr>
<td>Chin tuck</td>
<td>Chin down (widens valleculae, displaces tongue base and epiglottis posteriorly)</td>
<td>Aspiration, delayed pharyngeal response, reduced posterior tongue base motion</td>
</tr>
<tr>
<td>Head rotation</td>
<td>Rotate head to affected side (isolates damaged side from bolus path, reduces LES pressure)</td>
<td>Unilateral pharyngeal weakness</td>
</tr>
<tr>
<td>Head rotation</td>
<td>Rotate head to affected side with extrinsic pressure on thyroid cartilage (increases adduction)</td>
<td>Unilateral laryngeal dysfunction</td>
</tr>
<tr>
<td>Lying on side, elevation</td>
<td>R or L lateral (bypass laryngeal introitus)</td>
<td>Unilateral laryngeal dysfunction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Aspiration, bilateral pharyngeal impairment or reduced laryngeal elevation</td>
</tr>
<tr>
<td>Facilitatory techniques</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strengthening exercises</td>
<td>Various</td>
<td>Nonprogressive disease</td>
</tr>
<tr>
<td>Biofeedback</td>
<td>Augment volitional component</td>
<td>Poor pharyngeal clearance</td>
</tr>
<tr>
<td>Thermal stimulation</td>
<td>Cold, tactile stimulation to anterior faucial pillar</td>
<td>Delayed/absent swallow response</td>
</tr>
<tr>
<td>Gustatory stimulation</td>
<td>Sour bolus (facilitates swallow response)</td>
<td>Huntington's chorea, stroke</td>
</tr>
</tbody>
</table>

Data from Logemann13,162 and Miller and Langmore.177
plausibility and efficacy of swallowing therapy, which revolve largely around the use of videofluoroscopy simply because this technique has been the foundation of the clinical field.

Biological plausibility of swallowing therapy strategies. Several of the compensatory swallowing strategies in Table 6 relate to head positioning. Head turning has been studied systematically using combined videoradiography and manometry in normal subjects and in patient with lateral medullary syndrome. In normal subjects, head turning increased UES opening by 2 mm, reduced basal UES pressure by 18 mm Hg (35%), and caused the bolus to lateralize away from the direction of head rotation. The proportion of the bolus swallowed and extent of UES opening was also increased by head rotation in 5 patients with lateral medullary syndrome who had unilateral pharyngeal weakness and thus were expected to respond to this therapy. Another systematic videofluoroscopic study evaluated the effect of 5 postures (chin down, chin up, head rotation, head tilt, and lying down) in 165 patients with radiographic aspiration. In normal subjects, head turning increased UES opening by 2 mm, reduced basal UES pressure by 18 mm Hg (35%), and caused the bolus to lateralize away from the direction of head rotation. The proportion of the bolus swallowed and extent of UES opening was also increased by head rotation in 5 patients with lateral medullary syndrome who had unilateral pharyngeal weakness and thus were expected to respond to this therapy. Another systematic videofluoroscopic study evaluated the effect of 5 postures (chin down, chin up, head rotation, head tilt, and lying down) in 165 patients with radiographic aspiration.

One or more of these compensatory strategies benefited 77% of the patients studied, evident by an increase in the bolus volume that they could swallow without aspiration. In 25% of patients, aspiration was eliminated for all test bolus volumes and during drinking from a cup.

Another strategy of swallowing therapy is to facilitate swallowing with the use of pharyngeal sensory stimulation. This concept evolved from an old physiological study, suggesting that stimulation of the anterior faucial pillars consistently induces swallowing in humans. Stimulation of this area with a cold probe before swallowing hastens the onset of the pharyngeal swallowing response and reduces the latency to spontaneous swallows. Similarly, pharyngeal stimulation with a cold probe of the mucosa reduces swallowing latency in response to electrical stimulation of the superior laryngeal nerve in the cat. These observations provide an experimental basis for the technique of applying cold tactile stimulation to the faucial pillars to facilitate an otherwise delayed or absent swallowing response in patients with neurogenic dysphagia. Taste stimuli have also been shown to reduce the delay in initiation of swallowing and hasten triggering of pharyngeal swallowing in patients with neurogenic dysphagia. A recent study tested the effect of a sour bolus (50% lemon juice and 50% liquid barium) in patients with neurogenic dysphagia and reported a reduced delay in triggering of pharyngeal swallowing and reduced frequency of radiographically observed aspiration, suggesting that afferents from oral-pharyngeal chemoreceptors can facilitate swallowing.

Corrective strategies of swallowing therapy use volitional control to augment impaired aspects of pharyngeal swallowing. Oropharyngeal dysphagia with impaired UES opening is a frequent clinical problem in brainstem stroke victims and in other neurologically impaired patients. Videofluoroscopic analyses, including semiquantitative measures of bolus clearance, found that the Mendelsohn maneuver (purposeful prolongation of the anterior-superior displacement of the larynx at midswallow) increased the extent and duration of UES opening. Mechanical analysis has shown that using the maneuver, normal subjects were able to maintain substantial traction on the anterior sphincter wall to prolong sphincter opening. Supporting this notion, a case report describes a stroke victim able to use the Mendelsohn maneuver to accentuate UES opening, thus transforming from tube feeding to oral feeding.

Another corrective swallowing therapy is forceful or effortful swallowing, which is used in individuals with diminished deglutitive tongue force. The plausibility of this concept was demonstrated with a manometric technique aimed at yielding data on tongue force exerted during the bolus propulsive phase of swallowing. Rather than using a small pressure sensor that yields data only at the time of luminal closure, force acting on a plastic bulb of 1–5 mL volume positioned either in the midpalate or above the valleculae is registered on a polygraph, thereby mimicking a bolus within the oropharyngeal chamber. Normal subjects were able to volitionally modulate deglutitive tongue force substantially, greatly exceeding the range of modulation evoked by alteration of bolus viscosity.

Efficacy of swallowing therapy techniques. A MEDLINE search from 1981 to June 1997 retrieved 12 original articles specifically addressing the question of the therapeutic efficacy of swallowing therapy for oropharyngeal dysphagia. As summarized in Table 7, only 5 of these are controlled trials and 2 randomized letters, case reports, single case studies, articles in foreign languages, and articles relating to children, dysphagia caused by head and neck cancer, or surgery are not included. Papers retrieved covered a heterogeneous group of patients with neuromuscular diseases of multiple etiologies. The largest body of literature relates to swallowing therapy after strokes.

Although they are quite dissimilar in design, both randomized controlled trials of swallowing therapy pertained to stroke rehabilitation. DePippo et al. studied 115 orally fed stroke patients with mild dysphagia and absent or minimal aspiration on videofluoroscopy. Patients were randomized to three graded levels of treatment, including dietary modification and swallowing...
therapy, but for ethical reasons a nontreatment control group was not included. Outcome measures included pneumonia, dehydration, calorie-nitrogen deficit, and death, but only 15% of the study population developed any of these complications within 12 months. Thus, although there were no differences in these outcomes among the three treatment groups, the strong possibility of a type II error related to the mild disease group studied, combined with the lack of a nontreatment control group, make it impossible to conclude that there is a lack of efficacy on the basis of this trial. The other randomized, controlled trial in Table 7 examined the effect of dietary modification alone on the incidence of pneumonia in 66 orally fed dysphagic stroke patients in a long-term care setting who also had a history of pneumonia. Patients were randomized to one of two dietary regimens: controls received the customary purees and nonaltered fluids, and the treatment group’s diet was modified to a soft mechanical diet with thickened fluids. After 6 months, there was an 80% reduction in pneumonia episodes in the group receiving dietary modification. Unfortunately, dropout bias may have influenced these results because data from the 15% of patients who either died or required gastrostomy feeding were not accounted for. Notwithstanding, the results do suggest benefit from simple dietary modification in respect to a complication with a high mortality.

The remaining efficacy studies summarized in Table 7 provide minimal data from which reliable inferences can be drawn. Among other problems, it is often impossible to distinguish a treatment effect from spontaneous recovery, various treatments and combinations of treatments are applied, variable outcome measures are used, and nonhomogeneous patient populations and controls are studied. For reasons summarized in Table 7, although almost all of these studies conclude that swallowing therapy is efficacious, it is with a low level of evidence and consequently a grade C recommendation. On the other hand, swallowing therapy has not been proven ineffective either, and it must be emphasized that the available data are inconclusive. Thus, in formulating the recommendations here we attempt to balance the weight of evidence with a risk-benefit assessment of swallowing therapy.

The strongest evidence-based recommendation that can be made pertains to diet modification, for which the

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**Table 7. Critique of Swallowing Therapy Literature (Original Papers)**

<table>
<thead>
<tr>
<th>Population</th>
<th>Design (level of evidence)</th>
<th>Intervention (duration)</th>
<th>Outcome measures</th>
<th>Efficacy? (grade of recommendation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rehabilitation, 3–6 wk after stroke, n = 115152</td>
<td>Randomized controlled trial (II)</td>
<td>Swallow therapy, diet consistency (12 mo)</td>
<td>Pneumonia, dehydration, nutritional deficit, death</td>
<td>No (B); graded levels of therapy without a nontreatment group; low statistical power</td>
</tr>
<tr>
<td>Rehabilitation, 8–364 wk after stroke, prior pneumonia, n = 66149</td>
<td>Randomized controlled trial (II)</td>
<td>Diet consistency (6 mo)</td>
<td>Pneumonia</td>
<td>Yes (B); no intention-to-treat analysis (15% dropout)</td>
</tr>
<tr>
<td>Rehabilitation, 4–24 wk after multiple strokes, n = 7188</td>
<td>Controlled multiple crossover (II)</td>
<td>Thermal stimulation fauces (1 mo)</td>
<td>Radiographic (timing, aspiration, clearance)</td>
<td>No (C); subjective outcomes, no statistical comparisons</td>
</tr>
<tr>
<td>Stroke, neurogenic, postsurgical, n = 69189</td>
<td>Controlled, nonrandomized concurrent cohorts (III)</td>
<td>Swallow therapy (3-12 mo)</td>
<td>Pneumonia</td>
<td>Yes (C); retrospective chart review; cohorts not comparable</td>
</tr>
<tr>
<td>Mixed neurogenic, n = 31194</td>
<td>Controlled, nonrandomized historical cohorts (IV)</td>
<td>Swallow therapy (variable, mean 41 days)</td>
<td>Weight change, caloric intake, pneumonia</td>
<td>Yes (C); no statistical comparisons; subset of Neumann et al.193</td>
</tr>
<tr>
<td>Mixed neurogenic, n = 66193</td>
<td>Uncontrolled case series (V)</td>
<td>Various (?)</td>
<td>Radiographic aspiration, oral feeding, “ease” of feeding</td>
<td>Yes (C); therapy based only on bedside assessment; no controls</td>
</tr>
<tr>
<td>Stroke rehabilitation, n = 170195</td>
<td>Uncontrolled case series (V)</td>
<td>Swallow therapy (variable, mean 41 days)</td>
<td>Weight change, caloric intake, pneumonia</td>
<td>Yes (C); no objective outcome measures</td>
</tr>
<tr>
<td>Rehabilitation, 0.6–10 wk after stroke, n = 30196</td>
<td>Uncontrolled, retrospective case series (V)</td>
<td>Swallow therapy (3-12 mo)</td>
<td>Oral feeding, “ease” of feeding</td>
<td>Yes (C); chart review</td>
</tr>
<tr>
<td>Postpolio syndrome, n = 20197</td>
<td>Uncontrolled case series (V)</td>
<td>Various (2–52 wk)</td>
<td>Oral feeding</td>
<td>Yes (C)</td>
</tr>
</tbody>
</table>

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best published efficacy study found a reduction in risk of aspiration pneumonia.\textsuperscript{149} In addition, systematic videofluoroscopic examination of the effects of modification of bolus consistency has shown reduced bolus penetration of the airway, providing a logical basis for the concept of benefit from dietary modification. On these grounds, and considering the relatively low cost involved, routine introduction of dietary modifications in patients considered at risk for aspiration pneumonia is logical. However, this is done with the knowledge that reliable predictors of aspiration pneumonia are not currently available.\textsuperscript{10,147,151}

Among other modalities of swallowing therapy, only in the case of thermal stimulation of the fauces has efficacy been analyzed when the technique is used in isolation. In that case, there is good evidence of the biological plausibility of faecal thermal-tactile stimulation in that the stimulus results in short-term reduction in pharyngeal swallowing latency,\textsuperscript{181,182,188} but there is no evidence that the effects of thermal stimulation are long-lasting or that they are efficacious in improving overall oral intake.\textsuperscript{182,188} Thus, its use cannot be recommended. In assessing the merits of other specific swallowing therapies, we can only evaluate individual maneuvers according to the strength of data supporting their biological plausibility because no trials have addressed efficacy when any of these techniques are used in isolation. The short-term effects of manipulation of body or head posture and the supraglottic swallowing maneuver have been studied systematically using videofluoroscopy. These maneuvers result in short-term reduction of radiographic aspiration, but the evidence that pharyngeal bolus clearance is enhanced by one or more of these maneuvers is less convincing.\textsuperscript{178,179} The Mendelson maneuver can prolong UES opening and increase opening diameters,\textsuperscript{185,186} but it is unknown whether these changes translate into improved pharyngeal clearance, reduced aspiration risk, or greater ease of swallowing. Exercises to strengthen swallowing muscles in isolation have received little attention, but there is some evidence that the concept has biological plausibility.\textsuperscript{187} Hence, the probability of therapeutic benefit is greatest for head and body postures, the suprasupraglottic swallow, and the supraglottic swallow, slightly lower for the Mendelson maneuver, lower still for strengthening exercises, and lowest for thermal stimulation techniques.

In summary, the literature provides reasonable evidence of the plausibility of swallowing therapy but minimal evidence of efficacy. Nonetheless, although no hard evidence supports its efficacy, the available data are inconclusive and swallowing therapy has not been proven ineffective. Moreover, subsets of patients in whom therapy might be most effective have yet to be identified. Thus, the current weight of opinion (which is shared by the authors), combined with the convincing demonstration of biological plausibility for specific techniques and the consistency of low-grade evidence, is the basis for our recommendation that swallowing therapy should be used. Large-scale randomized, controlled trials are needed to clarify the current recommendations.

Summary

Management of oropharyngeal dysphagia often involves a polydisciplinary evaluation, the aims of which are to identify and characterize oropharyngeal dysphagia and identify the underlying cause whenever possible. Specific diagnosis of the underlying cause of neurogenic dysphagia is rarely made on the basis of videoradio- graphic or manometric observations because observed patterns of oral, laryngeal, pharyngeal, and cricopharyngeal dysfunction can exist in a range of neurogenic disorders. Finding the underlying cause of oropharyngeal dysphagia often requires that the clinical team think broadly because of the wide array of diagnostic possibilities. Special emphasis should be placed on detection of treatable underlying systemic conditions such as thyrotoxicosis, myopathy, myasthenia, and neoplasms. Similarly, when neuromuscular disease is suspected, appropriate imaging (computed tomography, magnetic resonance imaging), function tests (EMG, nerve conductance, Tensilon test), histological examinations (mucosal or muscle biopsy), or serological tests (CPK level, antinuclear antibody level, anticholinesterase antibody level, lumbar puncture) should be obtained. While seeking evidence for a systemic disorder, the second aim of clinical evaluation is to identify surgically (or endoscopically) treatable structural abnormalities. Careful radiographic and/or endoscopic examination of the oropharynx and proximal esophagus is aimed at detecting signs of neoplasm, infection, strictures, or diverticuli, each of which implies a specific therapy. Even when effective therapy does not exist for the underlying condition, it is a firmly held conviction among practitioners, and an expectation among patients, that accurate determination of diagnosis and prognosis is an important medical goal.

After important etiological abnormalities have been sought, functional abnormalities of oropharyngeal swallowing should be defined. In some instances, endoscopic examination of the oropharynx may suggest the main abnormality of the swallow, but characterization of the temporal disruption of swallowing coordination\textsuperscript{82} and identification of the underlying mechanism leading to that dysfunction require videofluoroscopic or cineradiographic examination. In some instances, especially with
suspected UES dysfunction, concurrent use of pharyngeal manometry with videofluoroscopy can allow further delineation of the underlying pathology and direct treatment.

From this point on, management decisions will be applicable to those patients in whom a structural, surgically treatable abnormality has been excluded. When considering further treatment strategies, the clinician must first establish whether institution of nonoral (e.g., gastrostomy) feeding is indicated. This will depend on establishing the likelihood that the patient will be able to sustain adequate nutrition safely via the oral route and on the unproven but reasonable premise that nonoral feeding is likely to reduce the risk of aspiration pneumonia. This decision is made in conjunction with the speech language pathologist who can, on the basis of videofluoroscopic analysis of therapeutic maneuvers, estimate the likelihood that such maneuvers will reduce the risks of oral feeding and enhance the efficiency of swallowing. The natural history and prognosis of the underlying cause of dysphagia, as well as the patient’s cognitive ability, will also influence the choice between oral and nonoral feeding. Introduction of appropriate dietary modification and specific swallowing therapy by the speech language pathologist is appropriate at this point. The choices among therapies will be directed by the videofluoroscopic findings and the individual patient’s ability to comprehend and cooperate with the various strategies. This process will frequently involve a subsequent videofluoroscopic examination to assess progress and the advisability of ongoing swallowing therapy and ascertain whether alternatives should be considered. The place of cricopharyngeal myotomy and other procedures such as laryngeal suspension in this class of patient remains controversial. The clinician can predict an overall response rate for myotomy of approximately 60% but at present cannot predict with certainty the likelihood of response in an individual patient. Until further well-designed studies in clearly defined subsets of patients are conducted, the decision about myotomy will remain empirical after the patient is informed of the risks and possible, but unproven, benefits.

Management of oropharyngeal dysphagia is currently an inexact science. The quality of evidence supporting much of what is generally accepted as current best practice is not high but is backed by reasonable evidence of biological plausibility and weight of clinical opinion. However, it is the responsibility of the professional groups involved in the care of these patients to undertake studies that examine rigorously the efficacy of current and future therapies, both medical and surgical, and the diagnostic and predictive utility of tools for the measurement of swallowing mechanics and dysfunction.

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