Upper-Esophageal Sphincter Dysfunction
Pathogenesis and Treatment

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Despite recognition of the upper-esophageal sphincter (UES) as a distinct anatomic and physiological entity (1–6), little is known regarding derangement of UES function (7). This lack of understanding is unfortunate, since incapacitation symptoms may result from minimal UES dysfunction.

NORMAL ANATOMY AND PHYSIOLOGY

The UES is described classically as being the cricopharyngeus muscle. It connects anteriorly to the cricoid cartilage and encircles the proximal end of the esophagus by spreading in a fan-shaped manner without any central raphe. In most of Zaino's (4) autopsy specimens (46 of 75), the cricopharyngeus was not incorporated in the musculature of the esophagus. In 24 specimens it fused with the longitudinal layer of the esophagus. In five specimens, no distinction could be made between the esophagus and the cricopharyngeus. Therefore, in most cases, the cricopharyngeus acts as an extrinsic sling for the pharyngoesophageal junction. Radiographically it appears as a posterior indentation on the barium column at the lower level of the sixth cervical vertebra (8).

On perfusion manometry, the UES has a resting tone of 30–100 mm Hg, relaxes when innervated, and contracts after its relaxation phase. Its greatest resting tone (100 mm Hg) is exerted in an anterior–posterior direction, while its lowest resting tone (30 mm Hg) is exerted at a right–left direction (9). Thus, like the LES (10), the UES has an axially determined pressure profile.

Manometrically, UES length exceeds the anatomic diameter of the cricopharyngeus by 2–3 cm. This is because a portion of the hypopharynx superiorly and the circular muscles of the esophagus inferiorly weakly contribute to this high-pressure zone (3).

The innervation of the UES is probably via pharyngeal branches of the vagus nerve, although the final neurogenic pathways have not been fully determined. Whereas bilateral section of the nerves to the UES in the dog leads to UES dysfunction (11), no such nerve has been isolated in the human. Henderson, however, described abnormalities in UES function in selective recurrent laryngeal nerve paralysis (12).

Upon swallowing, the UES relaxes to cervical esophageal baseline pressure; its relaxation phase totally encompasses pharyngeal contraction and is precisely coordinated with the contraction of the pharynx (Figure 1). Thus, the UES barrier function totally disappears with swallows, permitting ready passage of the oral bolus into the cervical esophagus (1, 8, 13).

OROPHARYNGEAL DYSPHAGIA

Oropharyngeal dysphagia (preesophageal or transfer dysphagia) is a symptom complex characterized by hesitation in initation of swallows, food or liquid sticking in the throat, nasal or oral regurgitation, and postdeglutitive cough. Some patients may have associated nasal speech and dysarthria. Weight loss and recurrent pulmonary soilage may occur (5, 14).

Oropharyngeal dysphagia is usually caused by neuromuscular disease at any location along the neuraxis (8, 14, 15). Thus, diseases of the central

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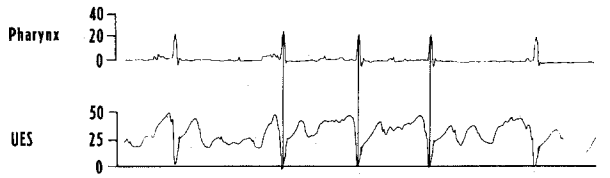

Fig 1. Normal perfusion manometry study of the pharyngoesophageal junction. UES relaxes completely to cervical esophageal baseline pressure and is coordinated with pharyngeal contraction. On this and other tracings "zero" = cervical esophageal baseline pressure. Paper speed = 5 mm/sec. [From Hurwitz et al (19).]

nervous system, peripheral nerve, motor endplate, and skeletal muscle may produce the clinical symptoms of this disorder. More rarely, local structural lesions may produce dysphagia by mechanical means. (Table 1).

A unique group of patients with oropharyngeal dysphagia are those with prior resective neck surgery or prior tracheostomy. These patients may develop cicatrix postoperatively which precludes elevation and anterior rotation of the larynx during swallowing; this impairment may lead to weak pharyngeal contraction and inadequate UES relaxation (16). Laryngectomy patients may show marked defects of UES relaxation and coordination as a result of cicatrix and neuromuscular disruption (17, 18).

TABLE 1. ETIOLOGIC CLASSIFICATION OF OROPHARYNGEAL DYSPHAGIA [ADAPTED FROM HURWITZ ET AL (19)]

Neuromuscular diseases
Central nervous system
Cerebrovascular accident with pseudobulbar palsy
Parkinson's disease
Huntington's chorea
Multiple sclerosis
Amyotrophic lateral sclerosis
Brain-stem tumors (primary or metastatic)
Tabes dorsalis
Miscellaneous congenital and degenerative disorders of the central nervous system
Peripheral nervous system
Bulbar poliomyelitis
Peripheral neuropathy
Disease of the motor endplate
Myasthenia gravis
Skeletal myopathy
Inflammatory muscle disease (polymyositis, dermatomyositis)
Muscular dystrophy (myotonic, oculopharyngeal)
Metabolic myopathy (thyrotoxicosis, hypothyroidism)
Unclassified
Pharyngoesophageal (Zenker's) diverticulum
Postsurgical resection of oropharynx, hypopharynx, and larynx
Local structural lesions
Oropharyngeal carcinoma
Inflammatory disorders (pharyngitis, pharyngeal abscess)
Congenital webs of proximal esophagus
Plummer-Vinson syndrome
Extrinsic compression (thyromegaly, senile ankylosing hyperostosis of the cervical spine, cervical lymphadenopathy)

With the exception of the less-common mechanical obstructing lesions, oropharyngeal dysphagia is caused by defects of UES function. These defects may be of two types: (1) failure of the UES to relax to cervical esophageal baseline pressure, and (2) failure of the UES relaxation phase to be coordinated with pharyngeal contraction. These two abnormalities may occur alone or in combination. They may be readily observed on perfusion manometry and usually, although not invariably, distinguish patients with oropharyngeal dysphagia from controls without such symptoms (Figures 2 and 3, Table 2) (19). Thus, despite a wide etiologic variety of neuromuscular diseases, the pathophysiology of UES dysfunction in this entity is highly circumscribed.

Since axially oriented manometry (9) has not been performed in a series of patients with oropharyngeal dysphagia, no comment can be made about absolute pressure measurements in these patients as compared to controls. Some of these technical difficulties may be overcome with more sophisticated recording devices.

Radiographic findings are determined by cine esophagram (30–60 frames/sec), since events at the pharyngoesophageal junction occur too rapidly for

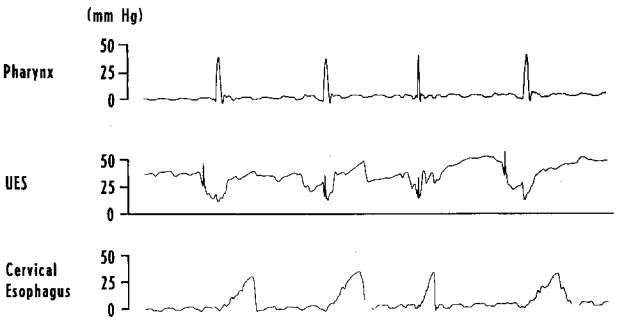

Fig 2. Perfusion manometry study of patient with Parkinson's disease and dysphagia. UES relaxation is incomplete. Paper speed = 5 mm/sec. [From Hurwitz et al (19).]

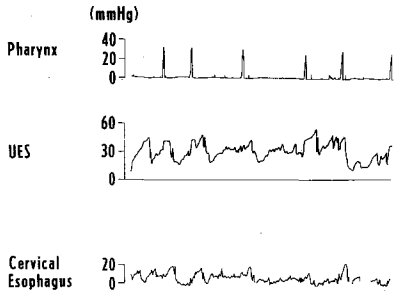

Fig 3. Perfusion manometry study of patient with dysphagia and Zenker's diverticulum. There is no coordination between pharyngeal contraction and UES relaxation; UES relaxation is also incomplete. Paper speed = 2 mm/sec. [From Hurwitz et al (19).]

detection by standard barium swallow (1, 8). Cine features may include abnormal tongue and palatal movements, asymmetric or nonpropulsive pharyngeal contractions, prominent posterior cricopharyngeus impression on the barium column, poor relaxation and coordination of the UES with pharyngeal contraction, and presence of pulmonary aspiration (8, 19).

Cine and manometric correlation is generally good, although manometry is more sensitive in determining the presence of UES dysfunction. Cine esophagography obviously complements the motility studies by delineating structural defects in the swallowing mechanism. Therefore, patients with oropharyngeal dysphagia should be evaluated initially with both esophageal motility and cine esophagraphic studies (19).

TREATMENT

Treatment of UES dysfunction has been empiric and uncontrolled up to this time. Three approaches are possible.

TABLE 2. UES FUNCTION IN PATIENTS WITH OROPHARYNGEAL DYSPHAGIA AND IN CONTROLS [ADAPTED FROM HURWITZ ET AL (19)]

	% swallows with complete UES relaxation (mean ± SEM)	*% swallows with normal UES coordination (mean ± SEM)*
Patients	50 ± 9.6	55 ± 8.6
Controls	95 ± 2.1	98 ± 1.3
P	< 0.001	< 0.001

The first approach is to treat the underlying medical illness to see if reversal of dysphagia occurs. In Parkinson's disease, hesitancy in forming a food bolus, aspiration, and abnormalities in UES function may be observed (8, 19). Cotzias and colleagues noted amelioration of dysphagia in selected patients with parkinsonism treated with L-dopa (20). Others have not observed significant dysphagia in Parksinson's disease, suggesting that patient selection and extent of disease may be important factors in determining symptoms (21).

Patients with myasthenia gravis may have symptoms of oropharyngeal dysphagia. Treatment with cholinesterase inhibitors may improve swallowing function (22).

Certain forms of myopathy may respond to medical management. The "pharyngeal" dysphagia of myotonic dystrophy may respond dramatically to quinine (23) or procaine amide (24). The myopathy of thyrotoxicosis (23) or hypothyroidism (25) may rarely present with oropharyngeal dysphagia, and management with appropriate agents has resulted in correction of the dysphagia. Finally, improvement in the handling of oropharyngeal secretions and prevention of dysphagia have been observed in patients with polymyositis treated with corticosteriods (26).

Despite the relative success of medical management in cases of neuromuscular disease, reports are too scanty and uncontrolled to make a definitive judgment about the results of medical treatment for oropharyngeal dysphagia.

The second approach involves bougienage to alleviate obstruction created by the malfunctioning UES. Simple bougienage of the UES with tapered mercury dilators is efficacious in some patients (27). It may be useful in those patients with previous neck surgery which results in local cicatricial scarring. However, dilatation may not have a long-term effect. It is contraindicated in patients with pharyngoesophageal diverticula because of the risk of perforation. The effects of bougienage on oropharyngeal dysphagia in patients with neuromuscular disease needs to be further assessed.

The third approach is surgical. Cricopharyngeal myotomy represents a major therapeutic advance in the treatment of oropharyngeal dysphagia (28). The rationale of this procedure is to weaken or abolish the proximal high-pressure zone by sectioning the cricopharyngeus muscle as well as 3–4 cm of the adjacent cervical esophagus. The procedure is extramucosal which simplifies the operation and the

postoperative course. The surgery can be performed under local anesthesia which enhances its application to a wide variety of patients (29). A large number of case reports attest to the relatively high surgical success rate when cricopharyngeal mytomy is performed for oropharyngeal dysphagia (19, 27, 29–43). Dramatic improvement may occur in terms of dysphagia, weight gain, and pulmonary symptoms. However, despite the ease with which the procedure is performed and its low morbidity, strict criteria for operative intervention are necessary. Myotomy should only be performed in patients having significant morbidity attributable to oropharyngeal dysphagia (i.e., local discomfort, weight loss, or pulmonary soilage). In addition, confirmation of pharyngoesophageal motor dysfunction by cine esophagography or manometry is a necessary requirement for operation. More than 230 cricopharyngeal myotomies for oropharyngeal dysphagia have been reported by 17 authors (Table 3). Variable results have occurred in patients with preexisting vascular, degenerative, or demyelinating disease of the central nervous system (33, 40–43). Patients in this category tend to have better results if voluntary tongue and pharyngeal movement is intact and if sensation in the oropharynx is present. Patients with peripheral neuropathy also have an unpredictable response to myotomy.

In contrast to patients whose neurological disease is proximal to the UES, patients with primary dysfunction of the sphincter respond more favorably to cricopharyngeal myotomy. Examples of this latter group are patients with symptomatic pharyngoesophageal (Zenker's) diverticula and patients with oculopharyngeal dystrophy. Zenker's diverticula may be associated with motility derangements of the UES (34), although these abnormalities may not always be present. Following myotomy, an approximate 50% reduction in UES pressure occurs, although the underlying motility disturbance persists (34). When UES myotomy is performed in these patients, a nearly uniform favorable clinical response can be anticipated (29, 30, 32, 34, 37, 39, 40). Diverticulectomy of diverticulopexy may be performed, if the diverticulum is greater than 2 cm in diameter or if additional symptoms directly attributable to the diverticulum are reported. Smaller diverticula may disappear spontaneously after cricopharyngeal myotomy.

Progressive oculopharyngeal dystrophy is a disease characterized by ptosis and dysphagia, often affecting families of French-Canadian descent (27, 44). Inability to propel the food or liquid bolus through the pharyngoesophageal junction, oral and nasal regurgitation, and frequent aspiration occur as the disease progresses in older patients. Low pharyngeal pressures, UES defects in coordination and relaxation, and abnormalities in peristalsis have been described (45). While early and intermediate stages of the disease may respond to bougienage, the advanced cases require cricopharyngeal myotomy to alleviate symptoms. Reports of myotomy have been very favorable in this disorder (27, 33, 36, 45).

As noted above, resective neck surgery (usually for malignancy) may lead to oropharyngeal dysphagia for a variety of reasons (16–18). On occasion, cricopharyngeal myotomy done at the time of resection, or at a later date, may alleviate symptoms of dysphagia (35). Results of myotomy are variable and depend on the extent of the underlying pathology, the level of the resection, the amount of tissue resected, and the adverse tissue effects of radiation therapy.

In patients with oropharyngeal dysphagia of unknown cause, it is not possible to determine whether the patient will benefit from cricopharyngeal myotomy, since the underlying pathophysiology is not known. Nonetheless, surgical reports in the literature are highly favorable in this relatively large group of patients (30, 33, 42).

While serious complications are rare, deaths from aspiration following cricopharyngeal myotomy have been reported (33, 39–41) (Table 3). One of us (A.H.) observed this complication in a patient with decreased LES pressure preoperatively. This patient had no preoperative history of gastroesophageal reflux or heartburn, and yet died of massive tracheobronchial aspiration several weeks after surgery. Clearly, great caution should be exercised in patients with LES hypotension. Significant documented gastroesophageal reflux or gastroesophageal regurgitation should be considered absolute contraindications to cricopharyngeal myotomy.

Bleeding at the operative site has been reported as a complication but was readily controlled in the one patient in whom it was reported (35). Other theoretical complications such as esophageal perforation, damage to the recurrent laryngeal nerve, mediastinitis, air swallowing, esophageal breathing, and incomplete sectioning of the cricopharyngeus have not been reported.

To summarize, the overall response to cricopharyngeal myotomy for oropharyngeal dysphagia

TABLE 3. RESULTS OF CRICOPHARYNGEAL MYOTOMY FOR OROPHARYNGEAL DYSPHAGIA

Author	*No. of cases*	*Diagnoses*	*Immed. results**	*Complications (immed. and long term)*
Sutherland (1962) (30)	8	Cricopharyngeal achalasia (two with Zenker's diverticula)	Good—7 Poor—1	None
Bingham (1962) (31)	1	Cricopharyngeal achalasia	Good—1	None
Peterman et al (1964) (27)	1	Oculopharyngeal dystrophy	Good—1	None
Belsey (1966) (32)	32	Zenker's diverticula	Good—32	None
Blakeley et al (1968) (33)	6	Oculopharyngeal dystrophy (2); S/P CVA (1); carcinomatous myopathy (1); idiopathic dysphagia (1)	Good—4 Fair—1 Poor—1	1. Sudden death in 1 pt (several mos. postop) 2. Aspiration (1 pt)
Ellis et al (1969) (34)	18	Zenker's diverticula	Good—13 Fair—4 Poor—1	None
Mladick et al (1971) (35)	22	Dysphagia following oral and pharyngoesophageal resections	Good—19 Fair—3	Bleeding at operative site
Montgomery et al (1971) (36)	8	Oculopharyngeal dystrophy	Good—8	Occasional "postcibal regurgitation"
Nogueira et al (1972) (37)	3	Zenker's diverticula	Good—3	None
Mills (1973) (38)	23	"Motor neurone disease" (17); S/P CVA (4); carcinomatous neuropathy (1); carcinomatous involvement, cranial nerves IX, X (1)	Good—12 Fair—10 Poor—1 (death)	None
Viard et al (1973) (39)	7	Zenker's diverticula	Good—6 Poor—1 (death)	Death due to aspiration
Akl et al (1974) (40)	19	CNS disease (2); peripheral neuropathy (3); skeletal myopathy (9); Zenker's diverticula (5)	Good—13 Fair—2 Poor—4 (1 death)	Death due to massive aspiration (1 pt)
Hurwitz et al (1975) (19)	6	CNS disease (1); peripheral neuropathy (1); skeletal myopathy (1); Zenker's diverticula (2); unclassified (1)	Good—2 Fair—3 Poor—1	None
Mitchell et al (1975) (41)	6	CNS disease (1); amyotrophic lateral sclerosis (1); bulbar palsy (1); oculopharyngeal dystrophy (1); recurrent Zenker's diverticulum (1); "cricopharyngeus spasm" (1)	Good—4 Fair—2	Aspiration (1 pt)
Hamman et al (1975) (42)	18	CNS disease (8); peripheral neuropathy (3); motor endplate (1); skeletal myopathy (2); idiopathic (4)	Good—11 Fair—2 Poor—5	No immediate complications; pneumonia or aspiration developed in 5 pts (resulting in 3 deaths)
Hiebert et al (1976) (29)	15	Zenker's diverticula (9); dysphagia after prior Zenker's diverticulectomy (2); "achalasia" of UES (4)	Good—13 Fair—2	Severe postop depression (1 pt)
Lebo et al (1976) (43)	38	Amyotrophic lateral sclerosis	Fair—27 Poor—11	1. Respiratory failure and death (2 pts) 2. Myocardial infarction and death (1 pt) 3. Atelectasis (1 pt)
Duranceau (unpublished)	5	Amyotrophic lateral sclerosis (1); oculopharyngeal dystrophy (1); Zenker's diverticula (3)	Good—4 Poor—1	None
Hurwitz (unpublished)	1	Oropharyngeal dysphagia, indeterminate cause	Poor—1 (death)	Death from massive aspiration

*Results: Good—marked improvement or disappearance of dysphagia; fair—improvement in dysphagia, but persistent symptoms; poor—no improvement in dysphagia or clinical deterioration.

is gratifying. Table 3 classifies the surgical result as "good," "fair," or "poor." A good result (marked improvement or disappearance of dysphagia) was noted in 64% of all reported myotomies. A fair result (improvement in dysphagia but with persistent bothersome symptoms) occurred in 24% of operated patients. A poor result (no improvement in dysphagia or deterioration in clinical status) was observed in only 12% of the patients. Thus, with appropriate patient selection, the clinician can anticipate a satisfactory response to cricopharyngeal myotomy. Surgical failures are most often seen in patients with central nervous system disease or peripheral neuropathy. However, even these patients may benefit from the procedure.

Strict follow-up of patients treated for oropharyngeal dysphagia should include assessment of swallowing symptoms, serial weights, repeat chest x-ray, and repeat cine esophagogram. Manometric studies are very helpful for initial diagnosis and can be useful to assess the effects of surgery on cricopharyngeal function; however, esophageal manometry is not critical for follow-up management (19).

AREAS FOR FUTURE RESEARCH

With the recent development of nonperfused pressure transducers incorporated directly in the manometry tubing, more accurate determinations of UES pressure and function may be possible (46). Dodds has commented on the difficulty in obtaining accurate measurements in the region of the UES (47, 48). Improved technique in perfused and nonperfused manometry systems should make evaluation of the normal and diseased UES an easier task. Use of spatially oriented manometry (9) will increase the accuracy of absolute pressure measurements in this area.

Much research needs to be done in the treatment of oropharyngeal dysphagia. Pharmacologic alteration of UES dysfunction remains a totally untried area of needed investigation. In addition, the present empiric medical and surgical approaches to oropharyngeal dysphagia need to be supplanted by more conclusive clinical trials. (46).

UES dysfunction is not as uncommon as once thought. The significant morbidity suffered by patients with this affliction dictates that more clinical and research interest be generated in this field.

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