GENERAL THORACIC SURGERY

CRICOPHARYNGEAL MYOTOMY FOR NEUROGENIC OROPHARYNGEAL DYSPHAGIA

Nancy Claire Poirier, MD[a]
Luigi Bonavina, MD[b]
Raymond Taillefer, MD[a]
Attilio Nosadini, MD[b]
Alberto Peracchia, MD[b]
André Duranceau, MD[a]

Background: **Forty patients (18 women, 22 men) with incapacitating oropharyngeal dysphagia of neurologic origin underwent cricopharyngeal myotomy. The subjective and objective response to myotomy was analyzed retrospectively with a mean postoperative follow-up of 48 months (range 1 to 255 months).** *Results:* **Radiologic evidence of functional obstruction caused by incoordination and incomplete relaxation of the upper esophageal sphincter was significantly reduced. Manometric recordings of resting and closing pressures of the upper esophageal sphincter were also significantly altered by the myotomy. Resting pressures decreased from 65 to 18 mm Hg and closing pressures dropped from 69 to 22 mm Hg. The relaxation time and poor coordination at the level of the upper esophageal sphincter, observed in the preoperative period, persisted after the operation. Radionuclide emptying studies in which a single liquid bolus was used showed persistent hypopharyngeal stasis with a 20% retention of radioactive material at 120 seconds. Subjectively, 33 patients initially had frequent aspiration episodes. Twenty became free of symptoms after myotomy ($p < 0.01$) and in six others the symptoms were improved. Overall, seven patients claimed to be free of symptoms of dysphagia and no longer had pharyngo-oral or pharyngonasal regurgitations and aspirations after their operation. Twenty-three other patients had improvement in symptoms. Ten patients reported no change in symptoms. All of them either were unable to swallow voluntarily or had dysarthria when assessed before the operation. One retropharyngeal hematoma is the only postoperative complication recorded. The operative mortality was 2.5% (1/40).** *Conclusion:* **Cricopharyngeal myotomy palliates neurogenic oropharyngeal dysphagia in patients with intact oral-phase deglutition. (J Thorac Cardiovasc Surg 1997;113: 233-41)**

From the Department of Surgery and Division of Thoracic Surgery, Université de Montréal, Hôtel-Dieu de Montréal, Montreal, Quebec, Canada,[a] and the Department of General and Oncological Surgery, University of Milan, Milan, Italy.[b]

Supported by the Department of Surgery Research and Development fund, Hôtel-Dieu de Montréal.

Read at the Seventy-sixth Annual Meeting of The American Association for Thoracic Surgery, San Diego, Calif., April 28–May 1, 1996.

Received for publication May 6, 1996; revisions requested June 5, 1996; revisions received Sept. 13, 1996; accepted for publication Sept. 18, 1996.

Address for reprints: Dr. André Duranceau, Department of Surgery, Hôtel-Dieu de Montréal, 3840, St-Urbain Montréal, Québec, Canada H2W 1T8.

0022-5223/97 $5.00 + 0 **12/6/78042**

Neurogenic oropharyngeal dysphagia results from disruption of the well-integrated mechanism of swallowing. Central nervous system diseases or interruption of either peripheral sensory or motor pathways are responsible for the variable clinical presentation, which is determined by location and extent of neurologic lesions. The oral phase is regulated by the cerebral cortex and brain stem. Its disruption causes dysfunction of the tongue, soft palate, or suprahyoid muscles, translating into the inability to initiate swallowing or to move food or liquid from the mouth to the pharyngeal cavity. Poor tongue propulsion delays reflex initiation of a swallow and reduces pharyngeal peristalsis.[1] The brain stem regulates the involuntary pharyngeal phase. Lesions located at this level produce pharyngeal

The Journal of Thoracic and
Cardiovascular Surgery
February 1997

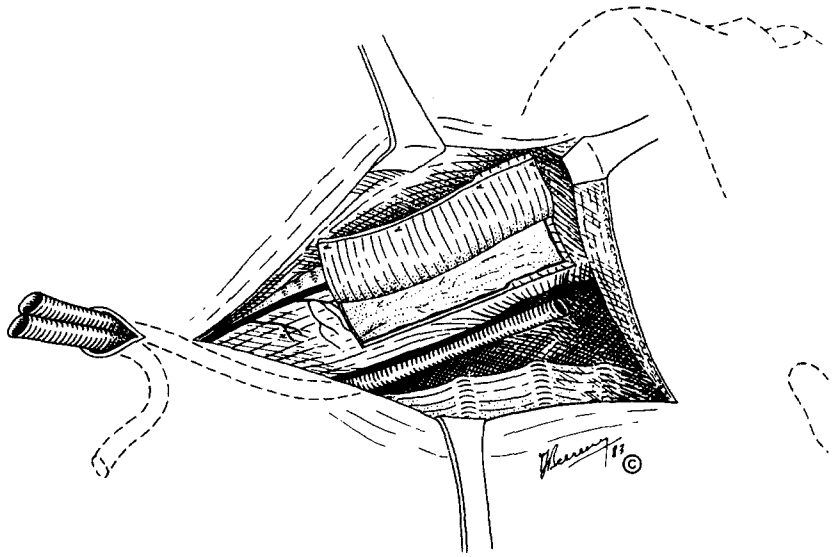

Fig. 1. A 6 cm myotomy is performed on the posterolateral aspect of the pharyngoesophageal junction through a left cervical incision. The myotomy is completed with a proximal and distal transection, creating a muscle flap that may be everted or resected for histologic analysis.

paralysis and upper esophageal sphincter (UES) achalasia, inhibiting free passage from the pharynx to the esophagus. The resulting misorientation of the food bolus causes pharyngo-oral regurgitation, pharyngonasal regurgitation, or laryngeal penetration and tracheobronchial aspiration.

Cricopharyngeal myotomy is thought to reduce difficulties in transport from pharynx to esophagus in refractory oropharyngeal dysphagia from neurologic causes. This report aims at identifying the subjective and objective responses to cricopharyngeal myotomy in patients with dysphagia caused by neurologic disease. Prognostic factors for improvement are sought from the observation.

Patients and methods

Between 1976 and 1994, 40 patients (18 women and 22 men) with an average age of 64 years (25 to 85 years) underwent cricopharyngeal myotomy for persistent oropharyngeal dysphagia resulting from a neurologic disease. Twenty-six (65%) patients had sequelae of a cerebrovascular accident (CVA). Seven patients with pseudobulbar palsy are included in this first group. Isolated pseudobulbar syndromes of undetermined origin without radiologic evidence of a stroke were seen in three additional patients. Three patients had radiologic evidence of cerebral atrophy and clinical signs of palsy of the ninth, tenth, and eleventh cranial nerves. The remainder of the patient population consisted of two patients with cerebral or upper cervical spine trauma (or both), two patients with amyotrophic lateral sclerosis (ALS), one patient with multiple sclerosis, one with Arnold Chiari syndrome, and another with Parkinson's disease. Oropharyngeal dysphagia was seen in one patient after posterior fossa surgery for an acoustic schwannoma. Symptoms were present for an average of 27 months varying from 1 to 96 months. Follow-up ranged from 1 to 255 months for a mean follow-up of 48 months.

Operation. All patients underwent cricopharyngeal myotomy by means of a standardized technique.[2] A left cervical oblique incision is used along the anteromedial border of the sternomastoid muscle (Fig. 1). A 36F Mercury bougie is introduced in the esophagus and used as a stent. In the posterolateral aspect of the pharyngoesophageal junction a 6 cm myotomy is performed. The muscularis along the myotomy is dissected free from the submucosa. A flap of muscle is created by proximal and distal transverse transection and resected for histologic analysis. A nasogastric tube is left in place for gastric decompression until peristalsis has resumed. This is used first to decompress the stomach and avoid aspiration. Second, it prevents the blind passage of a nasogastric tube through a freshly myotomized area if postoperative ileus and gastric distention were to occur. Penrose drains are left at the thoracic inlet level and behind the myotomized zone for 24 hours.

Thirty-five operations were performed with the patients under general anesthesia. Local anesthesia was used for the remaining five patients. A liquid diet was begun on the first postoperative day and patients were discharged on the second or third postoperative day.

Radiology. Preoperative and postoperative cineesophagograms and video-esophagograms or standard 6 frame/second barium esophagograms under fluoroscopic control were obtained in all patients at long-term follow-up. Voluntary deglutition, swallowing hesitation, pharyngeal stasis, functional obstruction, and incoordination at the

Table I. *Preoperative and postoperative symptoms*

Symptoms	*Preoperative (n =40)*	*Postoperative (n = 39*)*		
		Asymptomatic	*Improved*	*No response*
Dysphagia	40	7†	23	10
Aspiration	33	20‡	7	6
Regurgitation				
Pharyngo-oral	15	7	5	3
Pharyngonasal	12	5	4	3
Dysarthria	6	0	0	6

*One postoperative death.
†$p = 0.03$.
‡$p = 0.001$.

UES level were noted. Epiglottic dysfunction and tracheal aspirations were recorded.

Manometry. The pharyngoesophageal junction was assessed with a manometer in 35 patients. Preoperative and postoperative pressures were available in 24 patients. Standard recordings of the pharyngoesophageal junction were completed with the use of a perfused system and a four-lumen catheter with each recording port oriented at 90 degrees and at 5 cm from each other. During recording, one port is located in the pharynx, one in the high-pressure zone between the pharynx and esophagus, and the last two are in the cervical esophagus. The system is perfused by a pneumohydraulic pump generating a pressure of 15 psi. Resting and closing pressures are recorded. Manometric data were interpreted by means of criteria outlined in an earlier publication.[3] Relaxation of the UES is termed complete when the UES resting pressure drops to within 5 mm Hg of the cervical esophageal baseline pressure. The UES is interpreted as being coordinated when the UES relaxation period completely encompasses the pharyngeal contraction duration and when the peak pharyngeal contraction concurs with the nadir of UES relaxation. Mean values recorded during ten voluntary deglutitions were calculated. These values and their standard deviations were compared before and after the operation.

Radionuclide pharyngeal emptying study. Routine pharyngeal emptying scintiscans were performed before and after the operation in eight patients in whom this investigation was possible. A single bolus of 0.1 mCi of technetium 99m diluted in 10 ml of water was used. The percentage of radioactivity retained in the hypopharynx was calculated at 2, 5, 15, 30, 45, 60, 90, and 120 seconds.

Statistical analysis. A two-tailed Student's t test for paired values and χ^2 analysis for discontinuous values were used when appropriate. A p value of less than 0.05 was considered significant.

Results

Symptoms. Clinical findings are outlined in Table I. Twenty-nine of 40 patients who initially had dysphagia became completely or partially free of symptoms after myotomy. Pharyngo-oral or pharyngonasal regurgitation (or both), although improved, persisted in one third of the patients after the operation. Aspiration was significantly less prevalent ($p = 0.001$), with 27 of 33 patients claiming to have no symptoms or to have fewer episodes of aspiration. Nine patients with tracheobronchial aspiration during meals were considered to have been unhelped by the operation. *Two* of these patients subsequently underwent laryngeal exclusion. Thirty-nine of the 40 patients showed intact voluntary deglutition before the operation, but two of them were unable to swallow voluntarily after the operation. These two patients, along with the one who could not swallow before the operation, were not helped by myotomy. A gastrostomy and a tracheostomy were performed in two other patients. The mortality in this series was 2.5% (1/40). Uncontrolled sepsis from aspiration pneumonia was the cause of the one death. One retropharyngeal hematoma necessitated evacuation and represents the sole surgical complication recorded.

Radiology. The radiologic observations are summarized in Table II. Functional obstruction resulting from UES incoordination and incomplete relaxation was observed in 27 patients in the preoperative period and could still be identified in five patients after the operation. This was the only statistically significant radiologic change documented after myotomy. Radiologic evidence of swallowing apraxia, pharyngeal stasis, and aspiration was recorded with the same frequency before and after myotomy. None of the patients had a pharyngoesophageal diverticulum.

Manometry. Table III details manometric information from normal subjects and from patients with neurogenic dysphagia before and after myotomy. Peak pharyngeal contraction pressures are unaffected by the myotomy. UES resting and contraction pressures are both significantly decreased by the operation. Incoordination between pharyngeal con-

Table II. *Radiologic findings*

Observations	*Preoperative (n = 40)*	*Postoperative (n = 39*)*	*p Value*
Swallowing apraxia	12	11	0.94
Pharyngeal stasis	21	17	0.57
UES functional obstruction	27	5	0.0001
Epiglottic incoordination	10	11	0.94
Aspirations	17	14	0.71

*One postoperative death.

Table III. *Manometric data*

Parameters	*Normal**	*p Value*	*Preoperative (n = 24)*	*Postoperative (n = 24)*	*p Value*
Pharyngeal					
Contraction pressures (mm Hg)	33.0 ± 7.8	NS	31.4 ± 28.4	34.6 ± 32.4	0.72
UES					
Resting pressure (mm Hg)	50.0 ± 18.0	0.02	64.9 ± 33.0	18.1 ± 14.5	0.001
Contraction pressure (mm Hg)	96.0 ± 38.0	0.008	69.1 ± 37.4	22.3 ± 26.8	0.001
Relaxation time (sec)	1.3 ± 0.37	0.0001	0.73 ± 0.35	0.84 ± 0.31	0.40
Coordination	0.99 ± 0.03	0.0001	0.35 ± 0.40	0.48 ± 0.45	0.33

NS, Not significant.
*From Duranceau and associates[3] (1983).

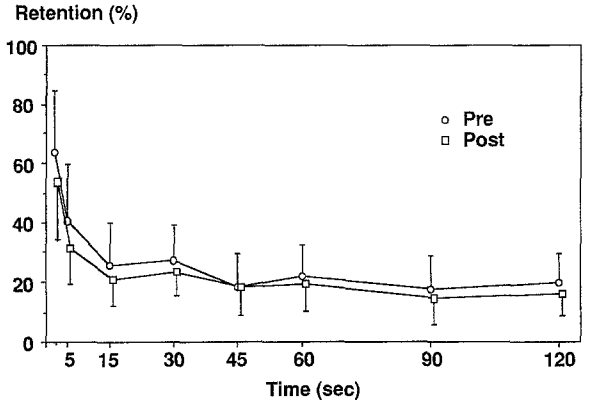

Fig. 2. Hypopharyngeal stasis persists after myotomy according to a single liquid bolus emptying scintiscan of the pharyngoesophageal junction. The hypopharyngeal retention of radioactive material over a 2-minute period is plotted here.

traction and UES relaxation is the most frequent observation in the patient with neurologic disease. Cricopharyngeal myotomy does not modify this dysfunction.

Radionuclide hypopharyngeal emptying study. In eight patients preoperative and postoperative emptying scintiscans were performed with a single liquid bolus. The hypopharyngeal retention of radioactive material over a 2-minute period is plotted in Fig. 2. No statistically significant difference was observed between these two emptying curves for the swallowing of liquids. Hypopharyngeal stasis persisted after myotomy.

Discussion

In 1951, Kaplan[4] reported the first successful cricopharyngeal myotomy to treat a patient with significant dysphagia from bulbar poliomyelitis. To our knowledge, more than 262 cricopharyngeal myotomies have been reported since then in the English literature to treat neurogenic oropharyngeal dysphagia (Table IV). Satisfactory to excellent subjective results are observed in approximately 50% of the documented cases. Extension of disease, location of lesions, and severity of the neurologic abnormalities affect the results. Very little objective information is reported to document the functional outcome of myotomy in these patients.

Analysis of this experience revealed that the functional obstruction resulting from an uncoordinated and spastic UES is mitigated by cricopharyngeal myotomy. This is seen radiologically when comparing preoperative and postoperative observations. The corresponding decrease in resting and closing pressures of the pharyngoesophageal junction suggests that a decrease in resistance to pharyngoesophageal transit is a possible mechanism to explain the clinical response. Persistent incoordination abnormalities and hypopharyngeal stasis with

Table IV. *Neurogenic dysphagia as reported in the English literature*

Etiology	*No. of patients*	*Results*		
		Excellent	*Moderate*	*Poor*
Cerebrovascular disease	86	22	35	12
ALS	55	25	14	11
Bulbar and pseudobulbar	22	1	8	5
Miscellaneous central nervous system	66	6	—	4
Trauma	12	3	1	5
Peripheral nervous system	21	7	3	—
	262			

Adapted from Duranceau[7] with added series of Van Overbeek[24] and Poirier, Taillefer, and Duranceau.[26]

liquids favor this explanation. Videoradiology techniques would certainly permit more specific muscle group abnormalities to be observed. Similarly, the sophisticated coupling of videoradiology and motor function recording, as used by Cook and associates[5] in patients with Zenker's diverticulum and by Kahrilas and colleagues[6] in normal subjects, would have allowed correlation between radiologic abnormalities and dysfunction. We did not have access to both methods simultaneously, thus allowing more "static" observations. Similarly, the visual functional obstruction of the UES cannot be translated in a compliance concept or into a restrictive condition, because our method of evaluation did not allow exact surface measurements. The stasis observed during barium esophagograms and quantified by radionuclide emptying studies were obtained with the use of a liquid medium only. Part of the liquid bolus pools mostly in the valleculae and piriform sinuses. Video techniques with boluses of various consistencies would allow added information on the anatomic and functional anomalies. Furthermore, the addition of a semisolid and solid bolus to the liquid scintiscan evaluation that we used would help to quantify the end result of deglutition in the patient with neurologic disease. Subjective improvement occurs in the bolus misorientation. Twenty-seven (65%) of patients with dysphagia from neurologic damage were either relieved of symptoms or had fewer aspiration episodes. This improvement in symptoms, however, does not always correlate with the radiologic observations, for a significant number of patients still have radiologic soilage of the tracheobronchial tree despite the myotomy. Dysphagia, present in all patients before the operation, was improved in 75% of the group (30/40) after myotomy. Pharyngo-oral and pharyngonasal regurgitations were less prevalent as well, possibly because of a lower resistance to pharyngoesophageal transit.

Ten patients had no improvement in their symptoms: two patients with ALS, one with pseudobulbar palsy, one with Parkinson's disease, and one who had had cerebral and cervical spine trauma; the other five patients had sequelae of strokes: lacunar bulbar CVAs in two, Wallenburg syndrome in one, cerebellar CVA in one, and left hemispheric CVA in one. All 10 of these patients had dysarthria or absence of voluntary deglutition (or both). Because the effect of the cricopharyngeal myotomy is primarily directed to the pharyngeal phase of deglutition, patients with predominant oral-phase dysfunction do not consistently respond to operative treatment. This observation is supported by a recent review on the results of surgery in the patient with neurologic disease.[7] Numerous authors from this review report that intact voluntary deglutition remains the essential factor to improve pharyngoesophageal transit in the patient with neurologic dysphagia. Absent bolus transport from mouth to pharynx and absent voluntary activity of the tongue explains the poor chances of improving the situation in these patients despite a patulous pharyngoesophageal junction created by the myotomy. Velopharyngeal muscle dysfunction causes malocclusion of the nasopharynx and consequent pharyngonasal regurgitations.

CVAs are the most common neurologic cause of oropharyngeal dysphagia.[8] Partial or complete spontaneous remission of related symptoms is frequent, with one study recording normal swallowing in 86% of patients 2 weeks after an acute unilateral stroke.[9] Severe unrelentless dysphagia appears especially after diffuse, bihemispheric, and brain stem lesions.[9, 10] Fifty-four cricopharyngeal myotomies performed on patients who have had a stroke have been

reported in the surgical literature.[11-17] Lacunar lesions of the brain stem are associated with better results. Overall, 35 of 54 (65%) patients were completely or partially relieved of symptoms. This figure corresponds with our observation that 20 of the 26 (77%) patients with a CVA were helped by their operation. The most common problem in these patients was pharyngeal dysfunction. Of patients with bulbar palsy or pseudobulbar palsy,[4, 16, 18-21] who also mainly have pharyngeal phase dysfunction, 87% report subjective improvement after cricopharyngeal myotomy.

ALS is characterized by the degeneration of motor neurons in the brain, brain stem, and spinal cord. Muscles of the tongue and hypopharynx show progressive signs of denervation leading to complete loss of voluntary deglutition. The swallowing reflex is delayed, the pharyngeal phase is uncoordinated, and the UES does not relax. Because the oral-phase dysfunction predominates, cricopharyngeal myotomy for ALS does not consistently help these patients. The two patients with ALS in our series saw no improvement of their oropharyngeal dysphagia. One died after the operation as a result of continued aspirations and secondary pulmonary infection. Similar observations are reported in larger groups of surgically treated patients with ALS. Whereas Lebo and associates[21, 22] reported a 50% improvement 6 months after surgical therapy, Loizou, Small, and Dalton[17] recorded a 20% mortality in this category of patients, mostly as a result of the impaired capacity to control aspiration.

The deglutition abnormalities seen in Parkinson's disease are also caused by oral-phase dysfunction. Hesitancy and poor control of the bolus result in symptoms similar to those of patients with ALS.[23] Good clinical response was however obtained after six of seven cricopharyngeal myotomies reported in the literature.[15, 24] Patients with oropharyngeal dysphagia resulting from trauma and peripheral nerve lesions respond unpredictably to early cricopharyngeal myotomy.[12, 25] Stabilization of the neurologic lesions is in order because spontaneous recuperation of a normal swallowing reflex is seen in a significant proportion of patients. Persistent dysfunction more than 6 months after the neurologic lesion, in a patient with well-preserved voluntary deglutition, stands an excellent chance of being improved by myotomy.[26]

Patients with neurogenic oropharyngeal dysphagia best suited for cricopharyngeal myotomy can be selected on the basis of precise assessment of symptoms along with radiologic, manometric, and radionuclide transit observations.

The subjective assessment of symptoms is the most important aspect of the initial evaluation in the patient with oropharyngeal dysphagia. Precise description and localization of the dysphagia, symptoms of bolus misorientation (pharyngo-oral and nasal regurgitations, aspirations), the effects of symptoms on the patient's daily life, and the consequences of these swallowing difficulties on nutrition are what orient the patient to further evaluation. Videoradiology is at present the most precise description of the functional abnormalities present. Anatomic distortions, such as a prominent unrelaxing cricopharyngeus, asymmetry of various muscle group contractions, incoordination, apraxia, poor bolus preparation and manipulation, misorientation of the bolus in the nasopharynx, oropharynx, or larynx are undeniable abnormalities that may influence selection for treatment. The correlation of symptoms with the abnormalities seen on radiologic examinations represent the most useful assessments for the therapeutic orientation of the patients.

In some patients with oropharyngeal dysphagia, symptoms and radiologic evidence of dysfunction may be more discrete. In these patients, we give importance to radionuclide transit studies. Our observation did not record changes in the emptying capacity of the hypopharynx. However, the normal hypopharynx should be empty after 3 seconds of a single bolus swallow. Although no changes occurred after cricopharyngeal myotomy in this group of patients having neurologic damage, radionuclide transit shows a 20% to 25% retention at 15 seconds. This parallels but adds quantification to the hypopharyngeal stasis seen on radiologic studies. To our knowledge, no information is available to assess hypopharyngeal emptying in these patients using liquid semiliquid and solid boluses. Although there is little evidence that scintigraphic studies change the selection of patients for cricopharyngeal myotomy, they can be used to better quantify retention in patients in whom poor pharyngeal emptying is suspected.

Manometric studies give a good indication of the physiologic abnormalities at the pharyngoesophageal junction and of the physiologic impact of the operation on the area. When these recording are made simultaneously with the videoradiologic observations, they allow a meticulous assessment of the dysfunction. Manometric recordings, however, are not used as an indication for surgical treatment.

They are seen as one facet of the functional assessment to be looked at with the global perspective of the patient with oropharyngeal dysphagia. Some aspects of manometric recordings may eventually be retained as prognostic factors (vs strength of pharyngeal contractions). However, significant improvement in recording of the pharyngoesophageal junction are needed to add consistency to manometric reports of this specific area.

Improvement after cricopharyngeal myotomy can be expected if the following criteria are fulfilled, regardless of the nature of the neurologic lesions: (1) normal voluntary deglutition, (2) adequate tongue movement, (3) intact laryngeal function and phonation, and (4) absence of dysarthria. Laryngeal diversion or excision with permanent tracheostomy must be considered in association with the extensive cricopharyngeal myotomy in patients with severe laryngeal dysfunction in whom no improvement can be anticipated.[4] Many patients, namely those who have had a stroke, regain oral feedings without aspiration either spontaneously or through feeding reeducation. Cricopharyngeal myotomy therefore should be delayed for at least 6 months after the neurologic event to allow stabilization of damage.

The myotomy technique we used was initially described by Montgomery and Lynch[27] and performed on patients with dystrophy. The rationale of removing the whole posterior muscularis at the pharyngoesophageal junction is to minimize all resistance to bolus transit. At present, there is no evidence that this technique results in easier and better pharyngoesophageal transit when compared with a standard linear myotomy. This extensive myotomy and myectomy provides good muscle sampling for histologic examination.

In our experience, cricopharyngeal myotomy palliates oropharyngeal dysphagia in the patient with neurologic disease who retains proper voluntary deglutition. It does so with minimal morbidity and mortality despite the unchanged functional abnormalities that result from the disease.

REFERENCES

1. Veis S, Logemann J. The nature of swallowing disorders in CVA patients. Arch Physical Med Rehabil 1985;66:372-5.
2. Duranceau AC, Jamieson GG, Beauchamp G. The techniques of cricopharyngeal myotomy. Surg Clin North Am 1983;63:833-9.
3. Duranceau AC, Devroede G, Lafontaine E, Jamieson GG. Esophageal motility in asymptomatic volunteers. Surg Clin North Am 1983;63:777-86.
4. Kaplan S. Paralysis of deglutition, a post poliomyelitis complication treated by section of the cricopharyngeus muscle. Ann Surg 1951;133:572-6.
5. Cook IJ, Gabb M, Panagopoulos V. Pharyngeal (Zenker's) diverticulum is a disorder of upper esophageal sphincter opening. Gastroenterology 1992;103:1229.
6. Kahrilas PJ, Logemann JA, Krugler C, Flanagan E. Volitional augmentation of upper esophageal sphincter opening during swallowing. Am J Physiol 1991;260:G450-6.
7. Duranceau A. Pharyngeal and cricopharyngeal disorders. In: Pearson FG, et al., editor. Esophageal surgery. New York: Churchill Livingstone, 1995:389-415.
8. Gordon C, Langton Hewer R, Wade DT. Dysphagia in acute stroke. BMJ 1987;295:411-4.
9. Meadows JC. Dysphagia in unilateral cerebral lesions. J Neurol Neurosurg Psychiatr 1973;36:853-60.
10. Ellis FH, Crozier RE. Cervical esophageal dysphagia in indications for and results of cricopharyngeal myotomy. Ann Surg 1981;194:279-89.
11. Hirano M. Cricopharyngeal myotomy for paralytic dysphagia. J Fr Otorhinol Laryngol Audiopharmol Chir Maxillofaciale 1974;23:731-4.
12. Blakeley WR, Gerety EJ, Smith DE. Section of the cricopharyngeus muscle for dysphagia. Arch Surg 1968;96:745-62.
13. Butcher RB. Treatment of chronic aspirations as a complication of cerebrovascular accident. Laryngoscope 1982;92: 681-5.
14. Gay I, Chisin R, Elidan J. Myotomy of the cricopharyngeal muscle: a treatment for dysphagia and aspiration in neurological disorders. Rev Laryngol 1984;105:271-4.
15. Bonavena L, Khan NA, DeMeester TR. Pharyngoesophageal dysfunctions: the role of cricopharyngeal myotomy. Arch Surg 1985;120:541-9.
16. Orringer MB. Extended cervical esophagomyotomy for cricopharyngeal dysfunction. J Thorac Cardiovasc Surg 1980;80: 669-78.
17. Loizou LA, Small M, Dalton GA. Cricopharyngeal myotomy in motor neuron disease. J Neurol Neurosurg Psychiatr 1980;43:42-5.
18. Mills CP. Dysphagia in progressive bulbar palsy relieved by division of the cricopharyngeus. J Laryngol Otol 1964;78: 963-4.
19. Bofenkamp B. The surgical correction of aphagia following bulbar poliomyelitis. Arch Otolaryngol 1958;68:165-72.
20. Schneider MA, Nagourney J. Progressive supranuclear ophthalmoplegia: association with cricopharyngeal dysfunction and recurrent pneumonia. JAMA 1977;237:994-5.
21. Lebo CP, U KS, Norris FH. Cricopharyngeal myotomy in amyotrophic lateral sclerosis. Trans Pacific Coast Oto-Ophthal Soc 1975;56:125-33.
22. Lebo CP, U KS, Norris FH. Cricopharyngeal myotomy in amyotrophic lateral sclerosis. Laryngoscope 1976;86:862-8.
23. Calne DB, Shaw DG, Spiers ASD, Stern GM. Swallowing in parkinsonism. Br J Radiol 1970;43:456-7.
24. Van Overbeek JJM. Upper esophageal sphincterotomy in dysphagic patients with and without a diverticulum. Dysphagia 1991;6:228-34.
25. Henderson RD, Boszko A, Van Nostrand AWP, Pearson FG. Pharyngoesophageal dysphagia and recurrent laryngeal nerve palsy. J Thorac Cardiovasc Surg 1977;68:507-12.
26. Poirier NC, Taillefer R, Duranceau A. Cricopharyngeal myotomy for neurologic dysphagia. In: Bremner CG, DeMeester TR, Peracchia A, et al., editor. Modern approach to

benign esophageal disease. St. Louis: Ms Quality Medical Publishing, 1995:155-64.
27. Montgomery WW, Lynch JP. Oculopharyngeal muscular dystrophy treated by inferior constrictor myotomy. Trans Am Acad Ophthalmol Otolaryngol 1971;75:986-93.

Discussion

Dr. Mark B. Orringer *(Ann Arbor, Mich.).* The patient with cervical dysphasia, as you have said, is a diagnostic and a therapeutic challenge. There is a need in such a patient to exclude local causes for this complaint: extrinsic compression of the esophagus by the thyroid or parathyroid gland, or lymphadenopathy or cervical spine exostoses, or intrinsic compromise of the lumen by tumor, web, stricture, or abscess. As you have pointed out, oropharyngeal or cricopharyngeal dysfunction can occur in association with a number of abnormalities of the central and peripheral nervous systems, but also with metabolic and inflammatory myopathy and reflux.

Of very practical importance to every thoracic surgeon is the disordered swallowing that follows either a tracheostomy or recurrent laryngeal nerve injury, which is a disastrous complication in patients with marginal pulmonary function who have life-threatening aspiration from injury to the recurrent nerve during a pulmonary resection or an esophagectomy. After a tracheostomy, normal excursion of the larynx, the elevation and the anterior rotation, that occurs with swallowing may be impaired, resulting in intractable and serious aspiration.

Your series of 40 patients with oropharyngeal dysphasia resulting exclusively from neurologic causes and treated with a cricopharyngeal myotomy is unusually large. I presume that this represents the pooled experience of the Duranceau group from Montreal and the Peracchia team from Milan. Is this correct, what was the relative contribution of patients from each group? Were the preoperative evaluations, the manometric techniques and interpretations, and the operative technique standardized in these two groups of patients?

In these patients with neurologic disease (strokes, cerebral or spinal trauma), how many had undergone a prior tracheostomy that could have contributed to their dysphagia or diminished their response to a cervical esophagomyotomy? When the barium esophagogram in such patients shows the typical posterior cricopharyngeal bar that you showed in your example, the surgeon certainly feels more comfortable directing a cervical myotomy at that target. However, when there is total inability of the patient to initiate a swallow, when there is oral or pharyngonasal regurgitation on the esophagogram, I am more concerned that there is more global neurologic dysfunction than a cricopharyngeal myotomy might be able to relieve. In these types of patients, I am inclined to be more patient, particularly after a stroke, and to use the response to passage of a 58F or a 60F bougie, both as a predictor of the efficacy of cricopharyngeal myotomy as well as therapeutically, if it works. Dr. Poirier, I would be interested to know your specific indications and the timing of surgery in your patients with dysphagia after a stroke. Who are the best candidates for this operation, and was dilation therapy tried before the operation in any of this group?

Many question the necessity of a 6 cm myotomy when the UES is anatomically only 2 to 3 cm long. I support your concept that longer is better here, but perhaps you could review the rationale for this extended myotomy.

The true value of esophageal manometry is subject to debate in these patients, many of whom cannot even initiate a swallow. This is a tough group of patients. They do not all get better with a cricopharyngeal myotomy. Careful patient selection remains crucial.

Dr. Poirier. In answer to Dr. Orringer's questions, the studied population consisted of 40 patients. Twenty underwent their evaluation and operation in Montreal and the remaining 20 patients in Professor Peracchia's service, in Milan. The recording technique and the interpretation criteria for the manometric data were identical. The cricopharyngeal myotomy was performed by means of the same approach for both groups. A total of five patients had a tracheostomy after their neurologic event. Four of the five patients underwent their myotomy with the tracheostomy in place. The tracheostomies were subsequently allowed to close in two patients with improvement of their condition.

Stroke is the most frequent cause of neurogenic dysphagia. This type of oropharyngeal dysphagia usually resolves spontaneously or with rehabilitation in 90% of patients. Surgery is considered only if the dysphagia persists for more than 6 months after the CVA. Patients are considered for surgery if, with their oropharyngeal symptoms, they have evidence of abnormal transit between pharynx and cervical esophagus. These abnormalities must be present radiologically, and poor emptying must be evident on the scintiscan. Poor control of deglutition as such is not a contraindication for the operation so long as voluntary deglutition remains intact. Patients with a predominantly oral-phase dysfunction are unable to prepare the food bolus in their mouth and propel it toward the pharynx. They do not respond well to this operation because the oral phase is untouched by the myotomy. The patients with an intact oral phase, able to initiate voluntary deglutition and showing no dysarthria especially if they show isolated upper esophageal sphincter achalasia or incoordination are significantly improved by the myotomy. The role of dilatation in this class of patients has not yet been defined and was not considered in this work because none of the patients in our series were treated by dilatation before the operation. The timing for the operation is important. We emphasize the fact that all neurologic damage must be stabilized for at least 6 months the operation can be considered. Why a 6 cm myotomy? Although the high-pressure zone recorded at the pharyngoesophageal junction is narrow, it is not well defined anatomically. For that reason, the myotomy used covers 2 cm on the cervical esophagus, 2 cm at the pharyngoesophageal junction, as identified by palpating the cricoid cartilage, and 2 cm on the distal hypopharynx. This technique has resulted in a well-documented decrease of the resting pressures in the sphincter.

Dr. Rodney J. Landreneau *(Pittsburgh, Pa.).* I have two questions. The diagram that was provided of your myotomy is a bit different from the type of myotomy that we usually use for primary disorders of the cricopharyngeus muscle. It appears that a large flap of muscle is resected, leaving a large defect with submucosal exposure. Do you

think that this extensive myotomy is necessary in the management of this disease as opposed to the usual linear myotomy that many surgeons use?

I was also wondering about the use of preoperative speech therapy among your patients. Did they all receive this? Were those who responded to the myotomy the same ones who had some response to any up-front speech therapy? Were the responders to surgical therapy also given speech therapy as an adjunctive measure of management after their operation?

Dr. Poirier. This extensive myotomy technique has been described by Montgomery when operating on patients with dystrophy. The rationale of removing the whole posterior muscularis of the pharyngoesophageal junction is to minimize all resistance for bolus transit. We know of no evidence favoring a linear myotomy or a resectional myotomy. One advantage is the histologic assessment of the resected muscle.

In response to the second question, there was no planned rehabilitation therapy for this group. Thus no correlation can be established between this approach and the improvement noted by the patients. No specific rehabilitation was offered after the operation.

Dr. Nasser Altorki *(New York, N.Y.).* One of your motility tracings showed a patient with retained pharyngeal contraction. You have had occasion to analyze 40 motility tracings. Did you see some correlation between the presence and absence of pharyngeal contraction and the motor status of the UES?

Did you use your motility tracing to select out patients who would benefit from this operation?

Dr. Poirier. Globally, the patient with neurologic disease has poorer pharyngeal function. Those patients who showed nonrelaxation of the UES usually had very poor contractions in the pharynx.

We did not try to correlate pharyngeal function with UES function. Manometry as such is not used as an indication for the operation. However, the observations during the recording are important, because they clarify location and severity of the deglutition abnormality and how much of the voluntary deglutition mechanism is retained.

Symptoms coupled with radiologic evidence of dysfunction and scintiscanning documentation of pharyngeal retention remain the basis for selecting the operation and establishing the prognosis for a successful outcome.