Influence of surgery on deglutitive upper oesophageal sphincter mechanics in Zenker's diverticulum

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Abstract

Background/Aims—To evaluate the role of upper oesophageal sphincter (UOS) compliance in dysphagia, the functional consequences of surgery were evaluated in eight patients with pharyngeal diverticula. The study examined the hypotheses that hypopharyngeal intrabolus pressure is an indicator of UOS compliance and that UOS opening and intrabolus pressure are normalised by surgery.

Methods—In eight patients and nine healthy controls, we measured the timing of swallow events, UOS relaxation, maximal UOS dimensions, intrabolus pressure, and trans-sphincteric bolus flow rates by simultaneous videoradiography and pharyngeal manometry.

Results—Bolus flow rates were not changed by surgery. Surgery significantly increased UOS opening ($p=0.0001$) and reduced hypopharyngeal intrabolus pressure ($p=0.0001$). The slope of the relation between sphincter area and intrabolus pressure was steeper in patients than controls and was normalised by surgery. Surgery had minor effects on basal UOS tone and timing of swallow events.

Conclusions—Upper oesophageal sphincter compliance is poor in Zenker's diverticulum and is normalised by surgery. Hypopharyngeal intrabolus pressure, which correlates with resistance to trans-sphincteric bolus flow, is a useful indicator of UOS compliance. Intrabolus pressure may be a predictor of outcome after myotomy in pharyngeal dysphagia. Cricopharyngeal myotomy is a mandatory component of surgery for Zenker's diverticulum.

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In 1878, Zenker proposed that pouch formation was caused by increased hypopharyngeal pressure generated during deglutition in the region of Killian's dehiscence.¹ Recently, the existence of increased hypopharyngeal intrabolus pressure during swallowing has been confirmed in these patients.² That study found raised intrabolus pressure was secondary to restricted upper oesophageal sphincter (UOS) opening during bolus flow while pharyngo-sphincteric coordination was normal. The

cricopharyngeus muscle from such patients demonstrates prominent fibroadipose tissue replacement and muscle fibre degeneration supporting the contention that the primary defect leading to pouch herniation is impaired UOS compliance.³

In this study we examined the hypothesis that intrabolus pressure is a valid indicator of poor UOS compliance. Specifically we hypothesised that cricopharyngeal myotomy in Zenker's diverticulum would normalise UOS opening; normalise hypopharyngeal intrabolus pressure, and the relation between these two variables; and increase trans-sphincteric bolus flow rates.

Methods

Patients and controls

We studied eight consecutive patients requiring surgery for a pharyngeal pouch causing dysphagia (5 M:3 F; mean age 62 years; range 48-85). They were studied before and eight weeks after surgery. Seven patients underwent complete cricopharyngeal myotomy by one surgeon (GGJ) of whom five had concurrent diverticulectomy, one diverticulopexy, and one the pouch was left in situ. The eighth patient was excluded from the group data analysis as he underwent only 'partial myotomy' by a different surgeon at another institution. The findings from this particular case are of some interest and are briefly discussed. Radiographic and manometric measures during swallowing in patients were compared with nine healthy controls without dysphagia of comparable age (mean age 76 years; range 52-85). Ethical approval for the study was granted by the Royal Adelaide Hospital Ethics Review Committee and all patients gave written informed consent.

Videoradiography

Patients were studied before and after surgery using simultaneous videoradiography and manometry as previously described.^{4,5} Patients and controls were studied in the upright seated position. Images of barium swallows were recorded in the lateral and anteroposterior projections using a 9" Phillips image intensifier (Phillips, Eindhoven, Holland). Fluoroscopic images of swallows in anteroposterior and lateral projections were recorded on video tape at 25 frames per second by a VHS video recorder (Panasonic, AG6500, Osaka, Japan) for later analysis. Magnification correction was

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established before the commencement of each study by placing two metallic markers set 3 cm apart in the field of the image intensifier, above the subject's head but in the plane of the UOS. Duplicate swallows of 2, 5, 10, and 20 ml boluses of high density liquid barium suspension (250% (wt/vol), E-Z-HD, E-Z EM, Westbury, NY) were measured and delivered to the mouth by syringe.

Manometry

Pharyngeal pressures were measured with a transnasally placed 9 lumen (OD 6 mm; ID each lumen 0.51 mm) silastic/PVC perfusion catheter incorporating six perfused sideholes spaced at 1.5 cm intervals and a 6 cm sleeve sensor of oval cross section (5×3 mm) (Dentsleeve, Belair, South Australia). The sleeve was positioned to straddle the UOS to accommodate the axial mobility of the sphincter and oriented to record posteriorly. The most distal of the four sideholes in the pharynx was sited at the level of the proximal sleeve margin. A sidehole located 3 cm distal to the proximal sleeve margin, in mid-sleeve position, aided positioning of the sleeve such that its midpoint was in the centre of the UOS high pressure zone at rest. All sideholes and sleeve assembly were perfused with de-gassed water by a low compliance pneumohydraulic perfusion system at 0.6 ml/min. The side holes were only perfused while swallows were being recorded to avoid fluid accumulation in the pharynx. Pressures were registered by external transducers (Deseret Medical, Park Davis, UT) and recorded on a 12 channel polygraph (Grass Instrument, Quincy, MA, USA), at a paper speed of 100 mm/sec. All pressures were referenced to basal hypopharyngeal pressure. A purpose built, video digital timer unit (Practel Sales International, Holden Hill, South Australia) imprinted simultaneously the elapsed swallow time on the video images in hundredths of seconds and a signal on the pressure tracing each whole second, to permit synchronisation of video images with pharyngeal and UOS pressures.

Surgery

In the seven cases in whom data are presented, the surgery was performed by one surgeon (GGJ). The tracings from the patient who had a partial myotomy and pouch excision at another institution was analysed separately and these data were not included in group mean data calculations. All patients underwent cricopharyngeal myotomy via a left sided cervical incision.⁶ A 3×0.5 cm window centred on the cricopharyngeus was excised with the excision extending superiorly to involve the distal segment of the inferior pharyngeal constrictor and distally to involve the proximal 1 cm of the cervical oesophagus. The incision was made as close to the cricopharyngeal posterior midline as possible. Five of seven patients had concurrent diverticulectomy and one had a diverticulopexy.

Data analysis

The timings of all swallow events were referenced to swallow onset defined as the initial movement of the tongue tip against the posterior surface of the maxillary incisors, and have been defined previously.⁴ UOS opening, closure, and duration of UOS flow were identified radiographically. Also measured were the onset of superior and anterior motion of the hyoid and larynx as well as timing of peak anterosuperior motion of these structures. Maximal UOS opening dimensions during transphincteric bolus flow were measured fluoroscopically in sagittal and transverse planes and maximal UOS area was derived by applying an elliptical model of sphincter profile.⁷ UOS flow rates were calculated by dividing swallowed bolus volume by the duration of sphincter opening for that bolus.

Basal UOS pressure was determined over a one minute interval after a 10 minute adaptation period after positioning of the catheter. Nadir deglutitive UOS pressure was determined in response to dry swallows. Hypopharyngeal intrabolus pressure was measured at the distal pharyngeal sidehole recording site immediately proximal to the opened UOS and was defined, on the basis of correlation of manometry and fluoroscopy, as the pressure at the time point midway between the timings of arrival of the bolus head and the departure of the bolus tail at that site.⁴ UOS relaxation onset, recorded by the sleeve, was defined as the time point when basal UOS pressure began to fall abruptly. Maximum UOS relaxation was defined as the point in time when the UOS relaxation profile ceased to fall rapidly and plateaued off. Because the proximal sleeve margin projects into the hypopharynx, the sleeve registers prematurely the apparent termination of UOS relaxation.⁸ Accordingly, termination of UOS relaxation was measured from the tracing recorded by the sidehole 1.5 cm distal to the proximal sleeve margin, which was seen fluoroscopically to lie within the UOS at the time of sphincter closure.

Hyoid excursion was determined by measuring the percentage reduction in geniohyoid muscle length as previously described.⁵ Laryngeal excursion was measured similarly except the laryngeal reference point used was the posteroinferior margin of the true vocal cords.

Duplicate values of radiographic and manometric measurements were averaged before calculation of group mean data. Statistical inferences were made regarding the bolus volume effect, surgical effect, and volume/surgery interaction using a two way, mixed design ANOVA for repeated measures.⁹ Parametric statistics were used having established that the data were normally distributed by applying the Shapiro-Wilks test to each variable. All values are represented as mean (SEM) unless stated otherwise.

Results

Basal UOS pressure fell from a pre-operative

mean of 55 (10) mm Hg to 43 (22) mm Hg after surgery; a statistically non-significant change ($p=0.2$). Pre and postoperatively, sphincter relaxation in all patients was complete. Mean UOS nadir pressures were 2 (2) mm Hg, 2 (1) mm Hg, and 1 (1) mm Hg in pre, and postoperative patients and controls respectively. Deglutitive UOS flow rates were unaltered by surgery but did increase significantly as a function of swallowed bolus volume in both states. For example, as bolus volume increased from 2 ml to 20 ml, corresponding pre-operative flow rates increased from 3.9 (0.1 ml/sec to 32.8 (1.2) ml/sec ($p=0.01$).

Sphincter opening was increased by surgery. Maximal sagittal UOS diameter increased significantly after surgery ($p=0.0001$) and was observed with all bolus volumes (Fig 1). Transverse sphincter dimensions also increased postoperatively but this did not reach statistical significance (Fig 1). The significant volume dependent increase in sagittal ($p=0.0008$) and transverse ($p=0.01$) diameters was preserved after surgery. Maximal UOS cross sectional area increased significantly after surgery ($p=0.0001$), and mean sphincter area for each swallowed bolus volume postoperatively did not differ significantly to those values seen in controls. The volume dependent modulation of the extent of sphincter opening was preserved after surgery ($p=0.006$).

As previously described, a prominent intrabolus pressure ramp was seen to precede the major hypopharyngeal pressure upstroke. This intrabolus ramp disappeared postoperatively (Fig 2). The postoperative manometric tracings

(Fig 2A) corresponds to the radiograph (Fig 2B). Note that the intrabolus pressure ramp diminished in a fashion comparable to other patients in whom pouches were resected despite the fact that the pouch in this case was left in situ. Figure 3 shows group mean intrabolus pressures, which fell significantly ($p=0.0001$) after surgery, to values comparable to those seen in controls. The relative gradients of the pre and postoperative curves differ. The intrabolus pressure increment in response to increasing swallowed bolus volume from 2–20 ml was greater pre-operatively (27.7 (7.6) mm Hg) than the corresponding increment seen postoperatively (13.2 (3.4) mm Hg) although this difference failed to reach statistical significance ($p=0.08$).

The relation between intrabolus pressure and the extent of UOS opening was examined by plotting corresponding mean values for each measure for each swallowed bolus volume before and after surgery (Fig 4). The gradient of this relation is considerably steeper pre-operatively indicating a considerable rise in intrabolus pressure for only a small increase in UOS opening. Postoperatively the slope of the curve approximates that of controls.

The timing of swallow events was not influenced by surgery although there was a tendency for earlier complete relaxation ($p=0.03$) and a shorter pharyngeal clearance time ($p=0.03$) postoperatively. The timing laryngohyoid motion was not influenced by surgery. The swallowed bolus volume dependent change in timing of UOS relaxation onset and opening ($p=0.01$) were not affected by surgery.

Maximal anterosuperior hyoid excursion was not influenced by surgery. Peak anterosuperior laryngeal excursion was significantly reduced after surgery particularly in response to larger swallowed bolus volumes ($p=0.02$). Because filling of the diverticulum itself might have displaced the larynx anteriorly we measured the larynx to cervical spine distance before and after surgery but these measures did not differ.

All patients who underwent complete myotomy had an excellent response with none reporting significant dysphagia or regurgitation postoperatively. The 45 year old male patient who had undergone partial myotomy and diverticulectomy elsewhere complained of incomplete resolution of dysphagia postoperatively. The postoperative radiograph in this patient shows a partial constriction persisting in the post-cricoid region during UOS opening, which was associated with persistence of a prominent intrabolus pressure ramp postoperatively (Fig 5).

Discussion

In 1878, Zenker and Ziemssen hypothesised that herniation of the posterior pharyngeal pouch proximal to the cricopharyngeus muscle was caused by high hypopharyngeal pressures, with the actual site of herniation being determined by a zone of weakness in the posterior hypopharyngeal wall; Killian's dehiscence.¹

Figure 1: Maximal sagittal diameter during UOS opening increased after surgery ($p=0.0001$). The increase in maximal transverse diameter postoperatively did not reach statistical significance ($p=0.16$). Swallowed bolus volume modulation of UOS opening is also preserved after surgery ($p=0.008$).

Figure 2: (A) Manometric tracings from a patient with Zenker's diverticulum before and after surgery. The stippled segment of the pressure trace represents the hypopharyngeal intrabolus pressure domain, which is very prominent pre-operatively and which normalises after surgery. (B) Postoperative radiograph from the same patient demonstrating persistence of the pouch after surgery despite abolition of the intrabolus pressure ramp.

Initially it was believed that UOS incoordination was the cause of the proposed increased hypopharyngeal pressure.¹⁰⁻¹² The primary cricopharyngeal abnormality resulting in Zenker's diverticulum has recently been shown to be incomplete UOS opening while neuromuscular pharyngosphincteric coordination and sphincter relaxation are normal.² Inadequate UOS opening considerably increases hypopharyngeal intrabolus pressure during trans-sphincteric bolus flow, which may cause pouch herniation. The probable cause of inadequate sphincter opening is muscle fibre degeneration and fibroadipose tissue replacement.³ These combined findings suggest that Zenker's diverticulum results from poor UOS compliance. We hypothesised that if UOS compliance were decreased in these patients, then cricopharyngeal myotomy should normalise both UOS opening and intrabolus pressure and might also increase trans-sphincteric bolus flow rate during swallowing.

Figure 3: Intrabolus pressure fell significantly after surgery ($p=0.0001$) with postoperative group mean values approximating those seen in healthy aged controls.

The major findings in this study were that UOS opening was normalised by surgery, and that this was accompanied by normalisation of hypopharyngeal intrabolus pressure while trans-sphincteric flow rate remained unchanged. The fact that myotomy did not increase UOS bolus flow rates indicates that liquid flow through the non-compliant sphincter is maintained by a secondary increase in hypopharyngeal intrabolus pressure. Cricopharyngeal myotomy had no effect on the timing of sphincter relaxation onset nor on the completeness of relaxation during swallowing and only caused a marginal reduction in basal UOS pressure. Neither sphincter spasm nor failed relaxation are factors in the pathogenesis of Zenker's diverticulum,² and the lack of effect of surgery on UOS pressures confirms the notion that this disorder is one of inadequate sphincter opening and decreased compliance and not one of defective UOS relaxation or excessive basal tone.

The implications of this study extend beyond the treatment of Zenker's diverticulum; a disorder in which cricopharyngeal myotomy is of confirmed efficacy. There have

Figure 4: Shown is the relation between UOS opening and intrabolus pressure before and after surgery. Note that the curve is much steeper and is shifted to the left pre-operatively. After surgery the location and gradient of this curve approximates that of controls. The steepness of the curve suggests substantially reduced sphincter compliance in patients and an increase in compliance after surgery.

Figure 5: Postoperative manometry tracing (A) and radiograph (B) from the patient who underwent 'partial' myotomy. This patient's dysphagia was only partially relieved by surgery. Note the persistent post-cricoid constriction and prominent intrabolus pressure ramp indicating persisting poor sphincter compliance resulting from inadequate disruption of the UOS zone.

been few studies of the functional consequences of cricopharyngeal myotomy on the mechanics of swallowing in pharyngeal dysphagia.¹³ In this study, the homogeneous patient population with pharyngeal dysphagia provided an ideal opportunity to study the relations among UOS opening, compliance, and intrabolus pressure. The major changes in these measures and in their relations suggest that these parameters, perhaps in combination, might be valuable predictors of outcome after cricopharyngeal myotomy for pharyngeal dysphagia from other causes.

Raised intrabolus pressure indicates a disorder of UOS opening, which can be caused by either intrinsic muscle disease, such as fibrosis,

or by failed UOS relaxation.¹⁴ The steep rise in intrabolus pressure in patients with a diverticulum induced by increasing swallowed bolus volume (Figs 3 and 4), in parallel with a plateau in the maximum UOS area for the same increase in bolus volume (Fig 4), both suggest the pressure gradient increases secondary to a fixed cricopharyngeal stenosis and not to lack of interruption of neural tone to the muscle during bolus flow. The latter situation is seen for example in cases of failed, manometric UOS relaxation. In these instances, intrabolus pressure is increased at low bolus volumes but does not increase appreciably as the swallowed bolus volume increases because the persistent sphincter tone applies a fixed resistance to flow regardless of the sphincter diameter achieved.¹⁵

It is possible that the pouch itself, by extrinsic compression, may reduce UOS opening but we believe this component is probably of minor importance in comparison to the intrinsic structural changes in the cricopharyngeus muscle³ because the magnitude of the pressure effects and diminution in UOS opening are not dependent upon pouch size.² It is well recognised that pouch resection is sometimes incomplete without compromising symptomatic outcome⁶ and the present study found that cricopharyngeal myotomy without pouch excision can normalise UOS opening and intrabolus pressure. The patient described in this report in whom the pouch was completely resected but in whom myotomy was incomplete, shows that neither UOS opening nor intrabolus pressure are normalised simply by removing the pouch (Fig 5). These findings all indicate that myotomy is the key element in treating this disorder and is the major determinant of the observed functional changes in this study. The early finding that cricopharyngeal dilatation by bougienage gave temporary relief of dysphagia¹⁶ prompted the introduction of cricopharyngeal myotomy.¹⁷ It has since been established that resection of the pouch alone is inadequate treatment and that myotomy is the essential element in treatment of this condition.¹⁸⁻²⁴ Indeed excellent symptomatic results can be achieved from myotomy alone.^{6, 22} Regression of the intact pouch can be seen after myotomy^{25, 26} and radiological recurrence of the pouch is reduced after myotomy.²⁷

The reason for the observed modest fall in UOS pressure in the context of a pronounced increase in sphincter opening postoperatively is unclear. However, our findings suggest that the active component of sphincter tone under resting conditions is minimally impaired while the passive component, which normally opposes dilatation of the relaxed sphincter during bolus transit, is considerably changed by myotomy. Presumably with healing, the cricopharyngeus muscular ring is reconstituted by interposition of connective tissue but the ultimate overall diameter of the relaxed ring is increased. As the sphincter is capable of developing similar basal tone from a greater resting length postoperatively it is probable that there is a compensatory increase in muscle fibre shortening when the sphincter is tonically

active. During deglutition, the muscle is inactive thus permitting it to expand with bolus pressure to its full capacity.

In summary this study shows that surgery reverses the UOS abnormality in Zenker's diverticulum indicating the underlying defect is one of poor cricopharyngeal muscle compliance. These findings underpin the current belief that cricopharyngeal myotomy is mandatory for the successful treatment of this disorder. Intrabolus pressures may be applicable to other forms of pharyngeal dysphagia as a measure of sphincter compliance and as a possible predictor of outcome after myotomy.

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