Surg Endosc (2004) 18: 31–34
DOI: 10.1007/s00464-002-8611-6

SURGICAL ENDOSCOPY
and Other Interventional Techniques

The myth of the short esophagus

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Received: 2 April 2003/Accepted: 29 April 2003/Online publication: 21 November 2003

Abstract
Background: The advent of laparoscopic surgery has increased the number of fundoplications performed today. With the increase in laparoscopic fundoplications, the reports of short esophagus continue to increase. This investigation was undertaken to review our data regarding the entity described as "short esophagus."
Method: All charts of patients who had laparoscopic fundoplications performed from 1991 to 2000 were reviewed. Patients with laparoscopic fundoplications received esophagrams at 3 months postoperatively and then at 6 months.
Results: A total of 628 fundoplications were performed, with 351 requiring hiatal hernia repair. After appropriate esophageal mobilization was performed, no further esophageal lengthening procedure was needed. There were 4 conversions, 16 recurrences, and 7 complications, and no deaths. Recurrences were due to "slipped fundoplications" ($n = 3$), ineffective valves ($n = 5$), and hiatal hernia disruptions ($n = 8$).
Conclusions: In our series of fundoplications and hiatal hernia repairs, no short esophagus was noted. With proper esophageal mobilization, clinically the entity described as "short esophagus" may not exist.

Key words: Gastroesophageal reflux disease — Laparoscopy — Fundoplication — Short esophagus — Esophagus — Nissen fundoplication

Gastroesophageal reflux disease (GERD) affects many people around the world and causes up to 75% of esophageal disorders [7]. With such a large prevalence, surgeons are constantly trying to improve the techniques and procedures used to treat this disease. Most authors agree that antireflux surgery is a better option than medical therapy for severe or complicated GERD [20, 37]. The ability to perform laparoscopic antireflux procedures has led to the resurgence of surgical treatment of GERD.

One of the highly debated issues within GERD surrounds the existence of the short esophagus. The short esophagus was first described as an entity in 1957 by Lortat-Jacob [24]. While many esophageal surgeons currently contend that short esophagus exists as a complication of long-standing GERD [9, 21, 22, 30, 31, 39], other surgeons argue against the existence of such a disease process [3, 6, 16, 22, 40]. Esophageal shortening is thought to be found in disease processes including type III (mixed) hiatal hernias, sarcoidosis, Barrett's metaplasia, caustic ingestion, scleroderma, and Crohn's disease [18, 28]. The incidence of short esophagus ranges from 0 to 60% [6, 16, 32]. We examined our experience in laparoscopic fundoplications and the entity described as "short esophagus." In addition, we reviewed all failures for any evidence or suggestion of association with short esophagus.

Methods

All charts of patients who had laparoscopic fundoplications performed from 1991 to 2000 were reviewed. Any patient undergoing a fundoplication with or without hiatal hernia repair was included in this study. This investigation also included patients with large hiatal hernias (greater than 8-cm wide defect). All fundoplications were performed by C.T.F. All patients received esophagrams and upper endoscopy. Only patients with atypical symptomology and abnormal barium studies received manometry and pH assessment.

The preoperative evaluation and technique of the fundoplication has been discussed elsewhere [13]. The technique for mobilization of the esophagus first involves dissection of the phrenoesophageal ligament. Further mobilization occurs when reducing any hiatal hernia contents and sac if present. Blunt dissection is used to separate any adhesions between the mediastinum and the esophagus. During high dissection, varying degrees of laparoscopes may be necessary for proper visualization. A lighted bougie helps avoid inadvertent esophageal injury. For each case, a minimum of 3 to 5 cm of intraabdominal esophagus was mobilized. A short esophagus was defined if it was not possible to mobilize the esophagus into the abdomen. All patients even without a hiatal hernia had a posterior cruroplasty.

Presented at the Society of American Gastrointestinal Endoscopic Surgeons (SAGES) meeting, New York, NY, USA, March 2002
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Table 1. Summary of patient population

Total patients	628
Hiatal herniorrhaphy	351 (56%)
Large hiatal hernia defect (>‰8 cm)	72 (11%)
Preoperative evidence of Barrett's	58 (9%)
Preoperative peptic stricture	13 (2%)
Mean follow-up (years)	4.3
Range of follow-up (years)	0.5 to 9
Conversions	4 (0.6%)
Short esophagus noted	0 (0%)
Esophageal lengthening procedures	0 (0%)

Patients with hiatal hernia >8 cm had a posterior cruroplasty reinforcement with PTFE [14].

Patients with laparoscopic fundoplications received esophagrams at 3 months postoperatively and then at 6 months to help document recurrences. Any subjective symptoms prompted an immediate esophagram at any time.

Conversions, recurrences, complications, and mortality were noted. Operative reports were reviewed for any description of a short esophagus or any utilization of esophageal lengthening procedure. Failures were reviewed to determine if short esophagus may have contributed to the failure.

Results

A total of 628 fundoplications were performed during the period of this investigation as summarized in Table 1. Hiatal hernia repair was required in 351 (56%) patients; and 72 (11%) patients had large hiatal defects (>8 cm). Preoperatively, 52 (8%) patients had Barrett's esophagus and 13 (2%) patients had a benign stricture due to advanced reflux disease. After appropriate esophageal mobilization was performed, no short esophagus was noted. No further esophageal lengthening procedure was needed or utilized.

All patients received esophagrams at least once in the first year of their follow-up period. The average follow-up was 4.3 years (range of 0.5 to 9 years). There were four (0.6%) conversions, 16 (2.5%) recurrences, seven (1.1%) complications, and no deaths. The four conversions were not due to the inability to mobilize the esophagus but due to technical problems (bleeding, gastric perforation, etc.). No conversions were required because of a short esophagus. Recurrences were due to crural disruptions (n = 8; 1.3%), "slipped fimdoplications" (n = 3; 0.5%), and ineffective valves (n = 5; 0.8%). The patients with ineffective valves had a normal intraabdominal esophagus by both endoscopic and radiological evaluation. All of these patients had an intact, well-placed fundoplication by all evaluations but still had evidence of reflux. The possible theoretical etiologies for a failed but intact wrap include (1) that initial construction was too loose or (2) that one of the three stitches involved in the wrap may have either broken or torn through the fundus. One of the patients with a "slipped fundoplication" was reoperated upon. The failure was a technical failure, not due to short esophagus, as evidenced by successful reoperation without the need for any esophageal lengthening procedure. Follow-up data for the other two patients were not available. No complications were related to a short esophagus.

Discussion

The term "short esophagus" itself can be confusing. Because of the debate over its existence, a concrete and consistent definition has not been set. Horvath et al. categorize the short esophagus into three categories: (1) true, reducible; (2) true, nonreducible; and (3) apparent [18]. An apparent short esophagus is one of normal length that has accordioned within the chest and thus appears short before proper mobilization. A true, reducible short esophagus is shorter in length although reduction of the gastroesophageal junction to 2.5 cm below the hiatus is still possible. Only the true, nonreducible short esophagus requires a lengthening procedure [18].

These complex definitions are not necessary. For our study, we defined a short esophagus to be one requiring an esophageal lengthening procedure after mobilization (or true, nonreducible by the terminology proposed by Horvath et al. [18]). We did not differentiate between a true, reducible esophagus and an apparent esophagus since both have similar diagnostic and therapeutic implications. Our feeling is that many surgeons may encounter an "apparent" short esophagus and may not perform sufficient esophageal mobilization. On the other hand, we may have encountered "true–reducible" short esophagi rather than "apparent" short esophagi. More importantly, however, an esophageal lengthening procedure was not required for any patient in our series.

According to the advocates of short esophagus, esophageal shortening occurs with advanced GERD. In advanced cases, acid reflux produces transmural inflammation that results in fibrosis, scarring, and shortening of the outer longitudinal muscle layer with subsequent shortening of the esophagus [1, 15, 22]. Many surgeons feel that in some short esophagus cases, the esophagus is not long enough to reduce the gastroesophageal junction (GEJ) to its normal position (even with esophageal mobilization) to allow a tension-free fundoplication [27]. In order to correctly perform a wrap, it has been suggested that 2.5 to 3 cm of intraabdominal esophagus is necessary [4, 11, 17, 19, 34]. We agree that ≥2.5 cm intraabdominal esophagus is necessary. In fact, we mobilized ≥3 cm of intraabdominal esophagus in all our patients.

Since a shortened esophagus with an intrathoracic wrap can result in postoperative dysphagia or failure, surgeons have explored utilizing esophageal lengthening procedures. Authors have suggested that failures, including "slipped" or misplaced fundoplication and crural disruption (resulting in wrap herniation into the mediastinum), may be due to failure to recognize the presence of a short esophagus [15, 33]. It has been suggested that 20–33% of the surgical failures may be due to a short esophagus and that corrections of such failures (i.e., reoperations) result in more complications and less successful results [8, 10, 21, 36, 38]. Previous studies, however, of recurrent laparoscopic fimdoplications demonstrated that a short esophagus was not the cause of any of the failures [3, 5, 12]. We agree that inadequate intraabdominal mobilization of the esophagus (which can occur even without the presence of a

short esophagus) is a potential cause of failure after fundoplication.

In order to correct a short esophagus and allow the GEJ to be brought tension-free into its normal subdiaphragmatic position, numerous lengthening procedures have been suggested. Our contention is that esophageal mobilization, with or without extensive mediastinal (type II) dissection [39], is usually the only procedure needed, although many feel that the Collis gastroplasty is the most effective procedure for esophageal lengthening [29, 31]. In both open and laparoscopic investigations, an esophageal lengthening procedure is required in 8% to 10% of patients undergoing fundoplication for GERD [15, 23, 32, 35]. Open options include simple Collis procedure, Collis-Belsey procedure, transthoracic/transabdominal Collis-Nissen procedure, and transthoracic/transabdominal uncut Collis gastroplasty. These procedures can also be performed total laparoscopically or a combination of laparoscopic and thoracoscopic techniques. One study by Ellis et al. [9] compared the Collis-Belsey operation to the Collis-Nissen operation and found the Collis-Nissen procedure more successful in terms of preventing reflux. The drawback to any of these esophageal lengthening procedures is the complications (up to 10%) which include gastroplasty line leaks, fistulas, and acid secretion from the neoesophagus [25, 26].

Some authors have attempted to detect short esophagus preoperatively [2, 15, 29, 40]. Awad et al. [2] examined the following criteria for their predictive value for short esophagus: (1) endoscopic evidence of stricture or Barrett's esophagus; (2) an irreducible, ≥5 cm hernia on barium esophagram; or (3) esophageal shortening on manometric analysis. Only endoscopic evidence of a stricture or Barrett's esophagus was associated with a short esophagus. Gastal et al. [15] observed that only the presence of an esophageal stricture predicted a short esophagus. Yau et al. [40] found that manometric evidence of short esophagus was associated with paraesophageal herniation, which may suggest that many of the short esophagi are due to a large hiatal defect that causes an accordion effect on the esophagus.

Since short esophagus is a clinical diagnosis assessed only in the operating room, it is difficult to disprove its existence, although the fact that no consistent preoperative factor predicts the occurrence of short esophagus suggests that it may not be as common as reported. In fact, Yau et al. [40] initiated a study that showed that there is no correlation between esophageal length and the need for reoperation. These authors argued that patients with manometrically shorter esophagi do not necessarily need a lengthening procedure. Like us, Belsey [3] felt that if esophageal mobilization is done completely, there is no need for lengthening even in cases of esophageal stricture. Hill et al. [16] examined cases of Barrett's esophagus and also suggested that the short esophagus does not exist. Johnson et al. [22] stated that there is no evidence that postoperative dysphagia is due to short esophagus.

It has been our experience that the short esophagus phenomenon is overemphasized, overreported, and overtreated. Our investigation focused on patients with GERD (with and without Barrett's metaplasia), although a large number of our patients also had hiatal hernias. Since we had few patients with other possible causes of short esophagus (such as sarcoidosis, caustic ingestion, scleroderma, and Crohn's disease), no definite conclusion can be made about the association, existence, and/or incidence of short esophagus in other disease processes.

In this study, conversions, hiatal hernia recurrences, slipped fundoplications, and complications were noted and most can be attributed to something other than esophageal length. The hiatal hernia recurrences ($n = 8$; 1.3%) were all from hiatal hernias defects >8 cm. In these cases, mesh was not utilized. Our previous investigation has demonstrated a recurrence rate of 22% without the use of mesh and a recurrence rate of 0% with the use of mesh for large hiatal hernias [14]. In all of these eight recurrences, esophageal length was not an issue and the recurrence could have been prevented by using the mesh overlay. At least one of the "slipped" fundoplications was due to technical reasons not related to a short esophagus, since the patient had successful reoperation without the need for an esophageal lengthening procedure. The two other patients did not undergo reoperation, so it is not known if the true GEJ was not recognized or if the stitches from the most cephalad side of the fundoplication tore from the crura. However, even if we failed to diagnose a short esophagus that led to the slipped fundoplication in these two patients, the rate of short esophagus in our series would be very low at 0.3% (2 out of 628), which is much less than the reported incidence in the current literature.

The higher incidence of short esophagus in the literature may be due to its association with large hiatal hernia defects. These patients need appropriate esophageal mobilization before fundoplication. Since many of these patients may have recurrence due to their large hiatal defect, a short esophagus has been blamed in cases where a mesh reinforcement of cruroplasty may have prevented the failures [14]. It is hard to ignore the continuous mention of short esophagus in the literature. Based on the current literature, the short esophagus probably does exist, although the incidence of short esophagus reported in the literature is misleading. It may be that our patient population illustrates the earlier treatment of GERD with proton pump inhibitors and/or surgery and not what was typically seen before the era of laparoscopic surgery. Our data suggest that if short esophagus is encountered today, it is of low incidence. A formal esophageal lengthening procedure, with its associated complications, is rarely required when proper esophageal mobilization is carried out.

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