GENERAL THORACIC SURGERY

MASSIVE HIATAL HERNIAS: THE ANATOMIC BASIS OF REPAIR

Nasser K. Altorki, MD^a
David Yankelevitz, MD^b
David B. Skinner, MD^a

Objectives: In the repair of giant hiatal hernias, controversy persists as to whether an antireflux repair is required and whether a Collis gastroplasty is necessary. This study was undertaken to determine the location of the gastroesophageal junction in giant hiatal hernias with an intrathoracic stomach, as well as the outcome after repair without a Collis gastroplasty. **Methods:** Fifty-two patients were evaluated for a giant hiatal hernia, of whom 47 underwent surgical correction. Preoperative evaluation included esophagoscopy ($n = 45$), gastrointestinal series ($n = 40$), esophageal manometry ($n = 20$), and 24-hour pH testing ($n = 13$). The dominant clinical features were acute chest or abdominal pain (72%), heartburn (53%), and gastrointestinal bleeding (49%). The gastroesophageal junction was located in the mediastinum in 77% of patients, in the abdomen in 17%, and was not determined in 6%. Twenty-eight patients (59%) had clinical or objective evidence of reflux. Reduction with an antireflux repair without a gastroplasty was done in 47 (Belsey, $n = 28$; Nissen, $n = 19$). An excellent or good result was achieved in 38 patients (90%) with a median follow-up of 45 months. **Conclusions:** These results, obtained without a Collis gastroplasty, are equivalent to those obtained by an antireflux repair with an esophageal lengthening procedure. The frequent location of the gastroesophageal junction in the mediastinum suggests that these massive hernias often are the result of progressive enlargement of a sliding component. An antireflux repair is therefore necessary in the majority of patients. (J Thorac Cardiovasc Surg 1998;115:828-35)

Although most surgeons concur on the need for surgical intervention in patients with symptomatic giant hiatal hernias, several basic issues remain controversial.^{1,2} Some of the unresolved questions include the choice of the surgical approach (trans-thoracic or transabdominal), the need for an esophageal lengthening procedure, and the need for an antireflux repair.³⁻⁵ Feeding the controversy is the lack of consensus on a precise anatomic definition of these massive hernias that have been variously and

often synonymously called paraesophageal hernias, total intrathoracic stomachs, or simply giant hiatal hernias. Our basic definition of the various types of hiatal herniation is therefore described. In brief, a type I hernia or a sliding hiatal hernia is considered present when the gastric cardia is displaced cephalad while the remainder of the gastric pouch remains within the abdominal cavity and the phrenoesophageal membrane, though stretched, has no anatomic defects. A type II or rolling hernia usually occurs as progressive enlargement of a sliding hernia allows the adjacent gastric fundus to "roll" up into the mediastinum alongside the esophagus, usually through a defective phrenoesophageal membrane. The pressure gradient between the abdomen and the mediastinum favors progressive enlargement of this type of hernia until the entire stomach migrates into the thorax, the so-called type IIA hernia or total intrathoracic stomach. We reserve the term *paraesophageal hernia* to describe what we believe to be the uncommon situation of a type II or IIA hernia in

From the Department of Cardiothoracic Surgery^b and Radiology,^a The New York Hospital–Cornell Medical Center, New York, N.Y.

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Address for reprints: Nasser K. Altorki, MD, The New York Hospital–Cornell Medical Center, 525 East 68th St., New York, NY 10021.

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which the esophagogastric junction remains securely anchored in the abdomen by the endoabdominal fascia. The current report is a retrospective analysis of our experience with the surgical repair of massive hiatal hernia (45 patients with an intrathoracic stomach and two with >50% of the stomach within the mediastinum) undertaken to answer two questions. First, in giant hiatal hernias, does the gastroesophageal (GE) junction reside within the abdomen or within the mediastinum? Second, what are the intermediate-term results when surgical repair of these hernias is accomplished without an esophageal lengthening procedure?

Patients and methods

Fifty-two patients were evaluated for a giant hiatal hernia, of whom 47 underwent surgical correction at The New York Hospital–Cornell Medical Center between January 1988 and March 1997. These 47 patients are the subject of this report. The office and hospital records of all 47 patients were reviewed. Reports of upper gastrointestinal tract series were reviewed to determine the size of the hernia and the location of the GE junction. If these data points were not stated, then the radiographs were retrieved and evaluated by one of us (D.Y.) for these parameters. The GE junction was considered to lie within the mediastinum if it was so stated in the radiology report or if on review of the barium swallow the junction was clearly seen above the level of the hiatus. Similarly, reports of upper gastrointestinal endoscopy, often performed by the operating surgeon, were reviewed to determine the distance between the incisor teeth and the GE junction, as well as the impression created by the crus of the diaphragm on the herniated gastric pouch. The presence of mucosal abnormalities within the esophagus or the gastric pouch were noted, as well as the presence of a volvulus of the herniated stomach. Finally, the operative reports were carefully examined to determine the operative approach and the type of antireflux repair.

Follow-up information was obtained by direct patient contact. Patients were carefully queried about the presence or absence of symptoms, such as dysphagia, heartburn, regurgitation, chest pain, gas bloat syndrome, postprandial fullness, and any new symptoms that may have developed after the surgical procedure. Patients were considered to have had an excellent result if they were entirely free of symptoms, a good result if mild symptoms occurred occasionally and were not considered important by the patient, and poor if symptoms were frequent or considered significant by the patient.

Results

Clinical presentation. The patients comprised 28 women and 19 men with an age range of 39 to 93 years (median 70 years). The symptoms at referral are shown in Table I. Acute pain syndromes dominated the clinical picture, occurring in 34 patients,

Table I. *Clinical presentation*

	No.	%
Heartburn	25	53
Present	13	
Past	12	
Regurgitation	8	17
Dysphagia	13	27.6
Weight loss	9	19
Vomiting	9	19
Pain	34	72
Epigastric	14	
Precordial	18	
Back	1	
Shoulder	1	
Bleeding	23	48.9
Frank	10	
Occult	13	
Respiratory	6	12.7
Dyspnea	4	
Aspiration	1	
Asthma	1	

nine of whom were admitted to the emergency room to rule out coronary insufficiency. Heartburn was present in 13 patients at presentation or previously in 12. Obstructive symptoms occurred in 22 patients, manifested by dysphagia in 13 patients and vomiting in 9. Gastrointestinal bleeding, occult or frank, was present in almost half of the patients.

Preoperative evaluation. Preoperative evaluation included an upper gastrointestinal tract series in 40 patients and an esophagogastroduodenoscopy in 45. The location of the GE junction was determined at endoscopy in 26 patients, by barium swallow in 15 patients, intraoperatively in 3 (all in the chest), and could not be determined in 3 patients. The data points retrieved from the endoscopic reports of 26 patients in whom endoscopic determination of the location of the gastroesophageal junction was possible are shown in Table II. These include the distance from the incisor teeth to the GE junction and to the crus of the diaphragm in 14 patients. In 13 of these 14 patients the junction was located in the mediastinum an average of 7.25 cm above the hiatus (range 4 to 12 cm). In another 12 patients the esophagoscope could not be passed through the diaphragmatic hiatus because of a severe twist in the herniated gastric pouch. Other findings noted at endoscopy are shown in Table III. On the barium swallow the GE junction was considered to lie in the mediastinum in nine patients and within the abdomen in six patients. Overall, the GE junction was located within the mediastinum in 36 of 44 patients who could be evaluated (82%, 77% of all patients)

Table II. Endoscopic findings: Site of GE junction and crus in 26 patients

No.	Distance in cm from:		Location of GE junction	No.	Distance in cm from:		Location of GE junction
	GE junction	Crus			GE junction	Crus	
1	36	41	M	14	33	38	M
2	35	40	M	15	35	*	M
3	30	38	M	16	30	*	M
4	35	40	M	17	30	*	M
5	28	40	M	18	30	*	M
6	35	41	M	19	38	*	A
7	32	42	M	20	35	*	M
8	35	39	M	21	35	*	M
9	32	42	M	22	35	*	M
10	35	42	M	23	24	*	M
11	35	40	M	24	32	*	M
12	40	42	A	25	34	*	M
13	35	40	M	26	34	*	M

A, Abdomen; M, mediastinum.

*Scope could not be passed across hiatus.

Table III. Endoscopic findings

	No.
Volvulus	13
Esophagitis	8
Barrett's esophagus	2
Gastric ulceration	12
Discrete ulcers	8
Pouch gastritis	1
Erosions	3

and in the abdomen in eight (18%). The location of the GE junction could not be determined in three patients.

Esophageal manometry was not performed in 14 patients who required urgent or semielective operation within 48 hours after emergency admission to the hospital. Twenty patients of the remaining 33 underwent esophageal manometry. These results are shown in Table IV. Notably, the motility catheter could not be passed beyond the sphincter in a third of the patients. Additionally, although the esophageal body showed evidence of primary peristalsis in 18 patients, 15 patients had either low-amplitude peristalsis or frequent simultaneous contractions. Two patients had no evidence of primary peristalsis, both of whom underwent a Belsey antireflux repair.

We have not routinely obtained 24-hour pH monitoring in patients with massive hernias because we routinely perform an antireflux repair. Thirteen patients, however, underwent a pH study and six had abnormal scores, including five of 11 patients with the GE junction in the mediastinum and one of two with the GE junction in the abdomen. Overall,

Table IV. Results of esophageal manometry (n = 20)

	No.
Lower esophageal sphincter	
Normal	10
Not entered	6
Abnormal	4
Short	3
Hypotensive	1
Peristalsis	
Present	18
Low amplitude	4
Simultaneous contractions	11
Absent	2

objective evidence of reflux as indicated by an abnormal 24-hour pH score or evidence of esophageal mucosal injury such as esophagitis or Barrett's esophagus was present in 13 of 47 patients (25%). Twenty-eight patients (59%) had heartburn, esophagitis, Barrett's esophagus, or an abnormal 24-hour pH study.

Surgical procedures. Surgical repair was carried out electively in 33 patients and on an urgent or semielective basis (within 48 hours of hospital admission) in 14. All 14 patients were admitted to the hospital on an emergency basis for the reasons outlined in Table V. Forty-six patients underwent repair of their hernia through a left thoracotomy approach, and in one a laparotomy was used after a failed attempt at laparoscopic reduction. At thoracotomy the esophagus was extensively mobilized from its mediastinal bed up to the level of the aortic arch. This always required division of the middle

Table V. *Indications for urgent operation*

	No.
Acute chest pain	2
Persistent vomiting	6
Acute esophageal obstruction	1
Hematemesis	4
Acute respiratory distress	1

esophageal artery and occasionally the esophageal branches of the left inferior bronchial artery. The hernial sac usually had both an anterior sliding component and a large posterior sac that occasionally extended well into the right side of the chest. Both components of the sac were excised with particular care to avoid injury to both vagus nerves. Once the dissection was completed, the two pillars of the crus were approximated posterior to the esophagus and an antireflux repair was performed. If any tension was still appreciated on the repair, then the pulmonary branches of the left vagus nerve were dissected to provide further mobility. The types of antireflux repairs performed are shown in Table VI. In each case sufficient length of the esophagus was available to accomplish the repair without the need for a Collis gastroplasty. A postoperative barium swallow was obtained on the fifth postoperative day before oral intake was begun.

There was one hospital death (2%) in an 89-year-old patient who had become acutely dyspneic after a sudden increase in the size of the hernia. After successful surgical repair she was treated by antibiotics for a urinary tract infection for several days before severe pseudomembranous colitis with peritonitis developed. Complications occurred in 19 patients (42%) and are shown in Table VII.

Follow-up. Follow-up information was available in 42 of 46 operative survivors (91%), with a median follow-up of 45 months (range 6 to 108 months). Results are shown in Table VIII. Overall, 90% of patients were either free of symptoms or had occasional minimal symptoms. In one symptom-free patient who was included among those with an excellent result after hernia repair, dysphagia developed 3 years after the repair as a result of a carcinoma developing within the Nissen fundoplication. Four patients were considered to have a poor result. Two patients had persistent dysphagia. One had a small anatomic recurrence after reduction of an intrathoracic stomach and a Nissen fundoplication. The other patient underwent repair of a massive hernia with a contained perforation treated by a

Table VI. *Surgical procedures*

	No.
Belsey repair	27
Transthoracic Nissen	18
Transabdominal Nissen	1
Belsey and diverticulectomy	1

Belsey antireflux repair that was complicated by a leak and an empyema. This latter patient also had a significant nonspecific esophageal motility disorder. A small asymptomatic recurrence occurred in a third patient, a mentally retarded 36-year-old man with spina bifida whose postoperative course was marked by acute psychosis. All three anatomic recurrences occurred within 12 months of follow-up. A fourth patient, who was relieved of all preoperative symptoms, was considered to have had a poor result because of severe postthoracotomy pain.

Discussion

Controversy in the surgical literature regarding the surgical repair of giant hiatal hernias revolves around two basic issues, namely, the need for an antireflux repair and the need for an esophageal lengthening procedure to counteract an acquired short esophagus.^{3,4} Williamson and associates³ reported that in only 15% of 119 patients undergoing hernia operations was an antireflux repair necessary because of either symptoms of severe heartburn or objective evidence of GE reflux. In that report, the authors noted that in the majority of their patients with giant hiatal hernias the posterior attachments of the cardia at the hiatus remained intact, thus guarding against the development of GE reflux. However, the location of the GE junction was not documented either radiographically or endoscopically in any of these patients. In contrast, Pearson and associates⁵ believe that all giant hiatal hernias result from progressive enlargement of a sliding component and therefore require an antireflux repair. Others, still, have favored a selective approach, adding an antireflux repair to the procedure only in patients with objective evidence of GE reflux.⁴ In a small study of 15 patients with "massive hiatal hernias," Walter and coworkers⁶ reported that 60% had objective evidence of GE reflux on 24-hour pH studies and that in these patients the lower esophageal sphincter was hypotensive, short, or entirely within the negative pressure environment of the lower mediastinum. Although we were able to perform 24-hour pH studies in only 13 patients, six or

Table VII. Operative morbidity

	No.
Morbidity	19/47 (42%)
Empyema	1
Transient dysphagia	12
Atrial fibrillation	4
Gas bloat syndrome	5
Leak	1
Ileus	1
Dumping	1
Peritonitis	1*

*Hospital death.

46% had abnormal scores. Furthermore, 59% of our patients had heartburn, endoscopic evidence of esophagitis, Barrett's esophagus, or an abnormal pH study. Clearly some patients have no clinical, endoscopic, or pH evidence of reflux despite the clear presence of the GE junction within the mediastinum. It is possible that progressive gastric herniation into the thorax may result in a pathologically created fundoplication that partially compresses the lower esophagus, thus precluding reflux in this patient subset (Fig. 1). It is of interest in this regard that 50% of our patients with a history of heartburn had that symptom in their remote medical history with subsequent resolution.

In this report we have shown that the cardia is displaced into the mediastinum in almost 80% of patients. To make that determination, we used data obtained by endoscopy, barium swallow, or by intraoperative evaluation. Endoscopy, which is often performed by the operating surgeon, is our method of choice because the location of the GE junction is easily determined relative to the impression created by the crus of the diaphragm on the herniated gastric pouch (Fig. 2, *A* and *B*). Occasionally, however, one is unable to determine the exact location of the crural impression, as in cases of torsion of the supradiaphragmatic stomach. This has precluded us from passing the endoscope through the hiatus in 12 patients. The location of the GE junction can also occasionally be seen clearly on the barium swallow and is unlikely to vary significantly in location with the respiratory cycle, as it may in cases of small sliding hiatal hernias. The location of the GE junction within the mediastinum in the majority of patients has previously been reported by Pearson and colleagues.⁵ This cephalad migration almost certainly disrupts all hiatal attachments and sets the stage for GE reflux. If the absence of reflux symptoms or objective evidence of reflux leads to a

Table VIII. Results

	No.
Excellent	32 (76.2%)
Good	6 (14.3%)
Occasional dysphagia	4
Postprandial fullness	1
Occasional regurgitation	1
Poor	4 (9.5%)
Asymptomatic recurrence	1
Postthoracotomy pain	1
Dysphagia	2

reduction of the hernia without an antireflux repair, then GE reflux may result, because the repositioned cardia is now completely detached from its usual hiatal attachments, resulting in a type I hernia. Williamson and associates³ reported a poor result in 14 patients, two of whom had symptoms of severe GE reflux, and 12 patients (or 11%) had hernia recurrence necessitating reoperation. Because all of our patients underwent antireflux repair, none had reflux symptoms on follow-up, including those with a poor result. It is possible that if the GE junction is intraabdominal (17% in this report), a true paraesophageal hernia will be present, in which case reduction of the hernia by a transabdominal approach will probably suffice without incorporation of an antireflux repair into the procedure. We have not practiced this approach, because we believe that the necessary esophageal mobilization can be better accomplished via a thoracotomy.

Even though we did not perform an esophageal lengthening procedure in any of our patients, almost 90% of patients considered themselves to have had an excellent or good result. These results are similar to those reported by Allen and associates⁷ after the almost routine use of an uncut Collis gastroplasty. Cut or uncut, a Collis gastroplasty is by definition a lengthening procedure because it allows the wrap to be placed further down on the foregut than it would have been otherwise. Using a transthoracic approach without gastroplasty, we were able to perform a tension-free repair in all patients.

Acquired esophageal shortening is known to occur in advanced cases of peptic esophagitis in which panmural fibrosis results in circumferential and axial cicatrization, as evidenced by the development of strictures and acquired shortening. Undoubtedly in such instances a simple antireflux repair is associated with a high failure rate approaching 25%, unless accompanied by a Collis gastroplasty or, alternatively, unless an esophageal resection is per-

Fig. 1. A, B, and C, Three patients with massive hiatal hernia demonstrating the intrathoracic location of the GE junction and the compression of the lower esophagus by the herniated stomach simulating a fundoplication.

formed.^{8,9} Only seven of our patients (16%) had esophagitis, which was grade I or II. None had severe erosive esophagitis or strictures. This may be the result of the more liberal use of proton pump inhibitors by gastroenterologists in recent years. In

contrast Maziak, Todd, and Pearson¹⁰ reported that 22% of their 94 patients with massive hiatal hernias seen between 1960 and 1996 had ulcerative esophagitis and 14% had peptic strictures. In that series 79% of patients had a gastropasty done. We have

Fig. 2. A retroflexed endoscopic view showing (A) the crural impression and the GE junction proximal to it. B, The GE junction in an intraabdominal location at the level of the diaphragmatic crus.

not appreciated any evidence of panmural fibrosis or significant periesophageal inflammation in any of our patients despite the fact that the hernia had usually been present for many years. We therefore find the proposition that true shortening of the esophagus is present in cases of massive hiatal hernias does not rest on a sound etiologic or pathogenetic basis. It is likely that cephalad migration of the GE junction with subluxation of its attachments allows the longitudinal muscle layer of the esophagus to shorten, resulting in "pseudo-shortening" of the organ. This shortening is easily remedied by extensively mobilizing the esophagus, as described, and securing it within the abdomen.

A transthoracic approach permits optimal esophageal mobilization to perform a tension-free and durable repair. Division of the middle esophageal artery and occasionally the left inferior bronchial artery is necessary. Additional mobility is obtained by bluntly dissecting the esophagus beneath the aortic arch and by mobilizing the pulmonary branches of the left vagus nerve. Undoubtedly, this extent of mobilization may not be safely or adequately achieved through a transabdominal approach. It is possible that laparoscopic techniques, by enhancing exposure within the mediastinum, may result in adequate and safe mobilization of the esophagus to permit a satisfactory repair. However, the short- and long-term outcomes of this mobiliza-

tion remain to be seen. Once the hernia has been reduced, the choice of the type of antireflux repair is determined by the surgeon's preference and expertise, as well as by the presence or absence of an associated primary motor disorder of the esophagus. Persistent postoperative dysphagia occurred in only two patients in this series; one underwent a Nissen fundoplication and the other, who has a motility disorder, received a Belsey antireflux repair.

Although we routinely perform preoperative esophageal function tests in all patients before operations for GE reflux, that has not been our standard of care in the patients with massive hiatal hernia for two reasons. First, a significant number of these patients require urgent or semielective surgical intervention (30% in this study). Second, symptoms other than GE reflux usually predominate at the time of referral for surgical repair. However, we were sufficiently concerned by the lack of evidence of peristaltic activity in two of our patients on preoperative manometry that we have now altered our strategy in favor of a more liberal use of preoperative esophageal function testing in this group of patients.

This is a retrospective analysis of our experience and is therefore subject to the shortcomings inherent in such studies. A prospective randomized trial would obviously be desirable with preoperative and postoperative 24-hour pH studies and periodic fol-

low-up barium swallows. However, these types of hiatal hernias are relatively uncommon. Our experience with 47 patients extends over a 10-year period, whereas Maziak's experience with 94 patients was accumulated over an interval of 33 years. That, coupled with the emergency nature of the operation in a sizable subset of patients, may render a randomized trial a difficult undertaking.

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