MASSIVE HIATUS HERNIA: EVALUATION AND SURGICAL MANAGEMENT

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Objective: **Paraesophageal hernias represent advanced degrees of sliding hiatus hernia with intrathoracic displacement of the intraesophageal junction. Gastroesophageal reflux disease occurs in most cases, resulting in acquired short esophagus, which should influence the type of repair selected.** ***Methods:*** **Between 1960 and 1996, 94 patients with massive, incarcerated paraesophageal hiatus hernia were operated on at the Toronto General Hospital. The mean age was 64 years (39 to 85 years), with a female to male ratio of 1.8:1. Organoaxial volvulus was present in 50% of cases. Clinical presentation in these patients included postprandial pain in 56%, dysphagia in 48%, chronic iron deficiency anemia in 38%, and aspiration in 29%. Symptomatic reflux, either present or remote, was recorded in 83% of cases. All patients underwent endoscopy by the operating surgeon. In 91 of 94 patients, the esophagogastric junction was found to be above the diaphragmatic hiatus, denoting a sliding type of hiatus hernia. Gross, endoscopic peptic esophagitis was observed in 36% of patients: ulcerative esophagitis in 22% and peptic esophagitis with stricture in 14%. A complete preoperative esophageal motility study was obtained for 41 patients. The lower sphincter was hypotensive in 21 patients (51%), and the amplitude of peristalsis in the distal esophagus was diminished in 24 patients (59%). These abnormalities are both features of significant gastroesophageal reflux disease. In 13 recent, consecutive patients with paraesophageal hernia, the distance between the upper and lower esophageal sphincters was measured during manometry. The average distance was 15.4 ± 2.33 cm (11 to 20 cm), which is consistent with acquired short esophagus. The normal distance is 20.4 cm ± 1.9 ($p < 0.0001$).** ***Results:*** **All 94 patients were treated surgically: 97% had a transthoracic repair with fundoplication. A gastroplasty was added in 75 cases (80%) because of clearly defined or presumed short esophagus. There were two operative deaths, and two patients were never followed up. Among the 90 available patients, the mean follow-up was 94 months; median follow-up was 72 months. Seventy-two patients (80%) are free of symptoms (excellent result); 13 (13%) have inconsequential symptoms requiring no therapy (good result); and three patients (4%) are improved but have symptoms requiring medical therapy or interval dilatation (fair result). Two patients had poor results because of recurrent hernia and severe reflux. Both were successfully treated by reoperation with the addition of gastroplasty because of acquired shortening, which was not recognized at the first operation.** ***Conclusions:*** **Most of these 94 patients had symptoms or endoscopic, manometric, and operative findings that were consistent with a sliding hiatus hernia. There was a high incidence of endoscopic reflux esophagitis and of acquired short esophagus.**

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True paraesophageal hernia, with the esophagogastric junction in a normal abdominal location, appears rare. Our observations were supported by measurements obtained at preoperative endoscopy and manometry, and by findings at the time of surgical repair. These observations support the choice of a transthoracic approach for repair in most patients. (J Thorac Cardiovasc Surg 1998;115:53-62)

The evolution of large paraesophageal hiatus hernias remains confusing. A pure paraesophageal hernia, commonly labeled a *type II hernia,* is one in which the esophagogastric junction remains in a normal intraabdominal location. These type II hernias feature symptoms of incarceration of the stomach in the mediastinum and are infrequently, if ever, associated with symptoms reflecting pathologic gastroesophageal reflux (GERD). In a sliding hiatal hernia, as the hiatus enlarges and an increasing amount of stomach herniates, the fundus of the stomach "rolls" upward and lies at a higher level than the displaced esophagogastric junction. This rolling variant is a stage in progression from a small to a large sliding hernia. If herniation progresses, the entire fundus and proximal antrum may migrate into the posterior mediastinum, and at this late stage organoaxial volvulus inevitably occurs. This anatomic configuration, a combination of sliding and paraesophageal hernias, has been labeled a *type III hernia.* When other intraabdominal contents, such as bowel and omentum, herniate with the stomach, the condition is labeled a *type IV hernia.* As noted in an earlier publication,[1] and contrary to popular concept, our observations indicate that almost all of these cases represent advanced degrees of sliding hiatal hernia with an intrathoracic displacement of the esophagogastric junction. This implies a need for an adequate antireflux reconstruction in all such patients undergoing operation, as well as an awareness that unanticipated cicatricial changes may be present in the distal esophagus (due to reflux esophagitis) and may prejudice the success of some of the standard hiatal repairs (because of acquired short esophagus).

This review updates our experience with these large paraesophageal hernias and provides additional information to support the premise that most such cases are of the sliding type and are commonly associated with significant acquired shortening of the esophagus as a result of reflux esophagitis. These observations contrast sharply with the high incidence of type II, pure esophageal hernias reported in several recent publications.[2-4]

Patients and methods

Data were collected for 94 consecutive patients with large paraesophageal hiatus hernias who were seen at the Toronto General Hospital between 1960 and 1996. All patients were treated with a surgical repair. Data collected included demographics, symptoms (past and present), duration of symptoms, radiographic and endoscopic findings, manometric data, operative record, postoperative complications, and surgical results with long-term follow-up.

Preoperative evaluation included history, plain chest radiograph, upper gastrointestinal series, and esophagogastroscopy. Mucosal injury of the esophagus was classified according to Skinner and Belsey's classification[5]: *grade I*, mucosal reddening; *grade II,* linear erosions; *grade III,* confluent superficial erosions, becoming circumferential; and *grade IV,* peptic stricture, acquired columnar-lined esophagus.

Esophageal motility can be difficult to assess in these patients because it may be difficult to advance the catheter beyond the esophagogastric junction into the stomach. A complete manometric study was achieved in only 41 of the 94 cases. Lower esophageal sphincter (LES) pressures are measured with an 8 lumen perfusion catheter and station pull through at 0.5 cm intervals. Normal values range between 16 and 30 mm Hg. The LES was classified as hypotensive if the pressure was less than 16 mm Hg and hypertensive if the tone was greater than 30 mm Hg. Peristalsis in the body of the esophagus was characterized as normal, diminished in amplitude, or absent. The pressure profile of the upper esophageal sphincter (UES) was included in the evaluation.

Since 1990 we have measured the esophageal length, determined at the time of manometry. Esophageal length is the distance in centimeters between the proximal border of the LES and the distal border of the UES. Normal values were calculated on patients referred to our motility laboratory for chest pain who had no pathologic motility results, with normal results of barium esophagograms and endoscopy. Normal esophageal length in our laboratory is 20.4 ± 1.9 cm (male = 20.8 ± 1.9 cm; female = 20.0 ± 1.8 cm).

Follow-up examination was done with personal interview by a staff surgeon or resident, yearly for the first 5 years and at 2-year intervals thereafter. A standard questionnaire was used to record data regarding reflux, dys-

Table I. *Signs and symptoms in 94 patients*

Signs and symptoms	*Patients*		*Duration*
	No.	*%*	*(mo)*
Heartburn	29	31	13.6
Reflux	75	80	11.2
Nausea	29	31	9.8
Vomiting	11	12	4.8
Hematemesis	7	7	3.7
Postprandial fullness	14	15	11
Postprandial dyspnea	14	15	9.9
Cough	5	5	9.6
Aspiration	27	29	9.5
SOB: continual	3	3	3.3
SOB: postprandial	3	3	10.5
Dyspnea	1	1	10
Halitosis	0	0	0
Regurgitation	3	3	8.0
Burping	15	16	13.4
Dysphagia	45	48	7.9
Odynophagia	0	0	0
Anemia	36	38	2.9
Melena	15	16	1
Waterbrash	11	12	11.5
Pain—postprandial	53	56	8.4
Pain—spontaneous	14	15	5.3
Pain—pseudoangina	4	4	13.0
Weight loss	5	5	1.5
GI bleed	11	12	0
None	0	0	0

SOB, Shortness of breath; *GI,* gastrointestinal.

phagia, functional side effects such as the inability to burp or vomit, and incisional pain. Barium esophagograms were done at 1, 5, and 10 years. Additional investigations, such as motility studies, endoscopy, and 24-hour pH studies, were done only for the further evaluation of patients with symptoms.

The results of surgery were categorized as *excellent* if the patient was free of symptoms. Results were defined as *good* if there were inconsequential symptoms of reflux, dysphagia, or functional derangements, none of which required therapy or additional investigations. Results were defined as *fair* if the patient had improvement in condition but had symptoms of reflux, dysphagia, or functional side effects that required treatment such as medication or dilatation. Results were categorized as *poor* in patients whose conditions were unimproved or worse.

Continuous data were analyzed by two-tailed t tests and expressed as mean ± standard deviation. Statistically significant differences were defined as a p value of 0.05 or less.

Clinical features. The mean age at presentation was 64.0 years (range 39 to 85 years), with a female to male ratio of 1.8 to 1. The nature and incidence of the most frequent clinical presentations are summarized in Table I. The most common clinical features were symptomatic reflux, 83%; postprandial fullness, discomfort, or pain, 56%; dysphagia, 48%; chronic iron-deficiency anemia, 38%; and aspiration, 29%. Although evidence of GERD was recorded in 83% of patients, symptomatic reflux was frequently reported only in the history, often decades earlier. Many patients had minimal or absent reflux symptoms at presentation with a paraesophageal hernia.

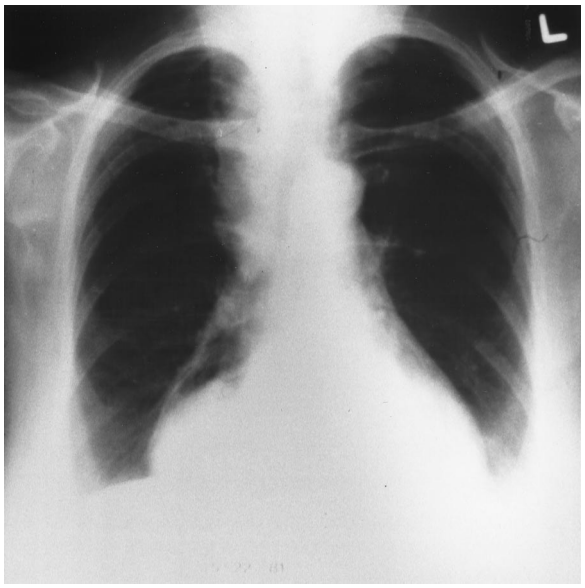

Fig. 1. Chest radiograph demonstrating an intrathoracic stomach.

A preoperative chest radiograph was available for review in 57 patients. The hiatus hernia was visible as a shadow in the posterior mediastinum in 54 of these 57 patients (Fig. 1). A preoperative contrast barium radiograph was available for review in 87 of 94 cases. A large paraesophageal hernia was demonstrated in all 87 cases, and organoaxial volvulus was present in 43 (50%) instances (Fig. 2). Three patients had complete obstruction, two at the esophagogastric junction and one at the pylorus (Fig. 3).

Esophagogastroscopy was performed in all cases by the operating surgeon. The esophagogastric junction was seen at a level above the diaphragmatic hiatus, denoting a sliding type of hiatus hernia, in 91 of these 94 patients. Gross endoscopic reflux esophagitis was found in 34 patients (36%). The degree of esophagitis was categorized as grade II in 7 patients (7%), grade III in 14 patients (15%), and grade IV in 13 patients (14%). Gastritis was recorded in 23 patients (24%) and pyloric obstruction was recorded in 13 patients (14%).

Esophageal motility studies were attempted in almost every case. In only 41 cases, however, was it possible to advance the manometry catheter beyond the esophagogastric junction and into the stomach. In these 41 patients, the LES was found to be hypotensive in 21 instances. The LES pressure was normal in 14 patients and mildly hypertensive in three. The amplitude of peristaltic waves in the distal thoracic esophagus was diminished in 24 of the 41 patients.

Since 1991 a measurement of esophageal length has been determined from the motility study. Manometric

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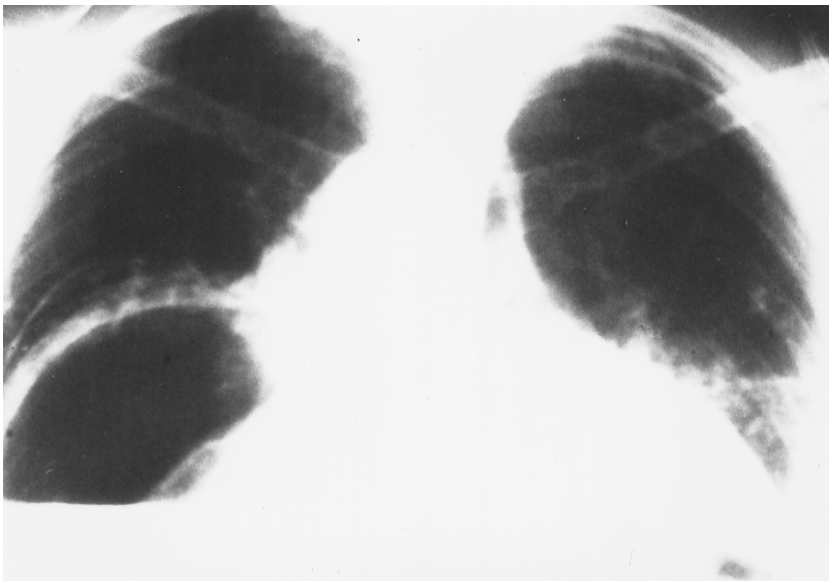

Fig. 2. Chest radiograph depicting organoaxial volvulus of the intrathoracic stomach.

esophageal length is the distance between the proximal border of the LES and the distal border of the UES. The normal esophageal length in our laboratory is 20.4 ± 1.9 cm, (male = 20.8 ± 1.9 cm, female = 20.0 ± 1.8 cm). Esophageal length was determined in 13 recent consecutive patients in this series in whom it was possible to pass the catheter beyond the esophagogastric junction into the stomach. The average esophageal length in these 13 patients was 15.4 ± 2.3 cm. The difference in average esophageal length between these 13 patients and the normal population is highly significant ($p = 0.0001$).

Operative procedure. Ninety-one of the 94 patients were operated on through a left posterolateral thoracotomy through the sixth intercostal space. Between 1960 and 1970, most of the patients in this series were treated with a standard Belsey Mark IV repair. A transabdominal Nissen repair was used in three patients with associated intraabdominal disease necessitating concomitant treatment.

A modified Collis gastroplasty was combined with a partial Belsey-type fundoplication in 75 patients who were judged to have acquired short esophagus. All patients had reduction of the hernia contents with excision of the hernia sac.

Only two patients required an emergency operation. Both patients had complete obstruction as a complication of organoaxial volvulus. In one patient the obstruction was present at the esophagogastric junction, and in the other it occurred at the level of the pylorus. In neither instance could the obstruction be relieved by passage of a nasogastric tube.

When a modified Collis gastroplasty was added to the repair, the gastroplasty tube was created over an indwelling 48F Maloney bougie. Since stapling instruments have become available, the gastrointestinal stapler has been used to create the gastric tube, usually for a length of about 5 cm. A Belsey-like 270-degree fundoplication was then created with three tiers of fundoplicating sutures, placed between the gastroplasty tube and the fundus of the stomach. Precise details of this operative technique were recently described and illustrated elsewhere.[6]

Results

Operative mortality and morbidity. There were two operative deaths (2%). One patient died 38 days after the operation of complications from a free esophageal leak, which resulted in a subphrenic abscess, septic shock and renal failure. A second elderly woman died on the sixth postoperative day after severe aspiration pneumonitis and respiratory failure.

Significant postoperative complications occurred in 18 patients (19%) and are summarized in Table II. Significant dysphagia occurred in 10 patients and required one or more dilations for relief. In two of these 10 patients, early dysphagia was caused by a small, unsuspected leak that was contained within the fundoplication and only seen on a contrast esophagogram. Significant dysphagia resolved satisfactorily in all 10 cases.

In four patients, a leak developed in the region of the gastroplasty and fundoplication. In two of the four cases, a free extravasation of contrast material was seen in the diagnostic radiograph. The extravasation extended into the abdomen and subphrenic space in one patient; despite drainage, it resulted in

Table II. *Postoperative complication*

	Patients	
Complication	*No.*	*%*
Dysphagia	10	11
Leak	4	4
Atelectasis	5	5
Pneumonia	2	2
Myocardial infarction	2	2
Arrhythmias	2	2
Empyema	1	1
Reintubation	1	1
Wound infection	1	1
Urinary tract infection	1	1
Deep venous thrombosis	1	1

generalized sepsis, renal failure, and death. In the other patient with free extravasation, the leak originated at the upper end of the gastroplasty incision and communicated with the pericardial space through a small defect in the pericardium. This complication was identified within 48 hours, and reoperation was performed to close the small fistula; subsequent recovery was uneventful. The other two leaks were recognized in postoperative contrast esophagograms that were done because of marked postoperative dysphagia. In each case, a small amount of contrast material was seen in the fold between the fundoplication and the distal esophagus or gastric tube. Both leaks resolved spontaneously without further complication and had no adverse effect on the quality of the long-term result.

Follow-up and results. Two patients never returned for a follow-up visit after discharge from hospital. Eleven patients died of unrelated disease between 6 and 347 months from the time of operation, and at the time of last follow-up all 11 of these patients had had good or excellent results. One additional patient had adenocarcinoma of the distal esophagus that was diagnosed 25 months after his hiatus hernia repair. The tumor was resected, but the patient died of metastatic cancer 13 months after esophagectomy. It is assumed that the adenocarcinoma developed in dysplastic, columnar-lined esophagus.

The average length of follow-up for the 90 available patients was 93.6 months ± 77.4. The median length of follow-up was 72 months, with a range between 6 and 347 months. The distribution of follow-up is divided into 5-year periods and graphically portrayed in Fig. 4. Fifty-three patients have been followed up for longer then 5 years, and 24 patients for longer than 10 years.

Fig. 3. Contrast barium radiograph demonstrating an intrathoracic stomach with complete obstruction at the pylorus.

An excellent result was maintained throughout follow-up for 72 of the 90 available patients (80%). Good results were recorded for 13 patients (14%), and fair results for 4 (4%). Two patients had unimproved or worse conditions. Both had recurrent herniation with severe symptoms of recurrent reflux. These patients were operated on during the first decade of this reported experience and were initially treated with a Mark IV Belsey repair. Both underwent reoperation, at which acquired short esophagus was recognized and a gastroplasty was added to the second repair. Both subsequently had good long-term results. Thus 87 of the 94 patients ultimately obtained good to excellent results in long-term follow-up.

Only five of the 94 patients have undergone reoperation. Both patients with a free postoperative leak underwent emergency reoperation, as described previously. Two patients initially treated with a Belsey Mark IV repair underwent reopera-

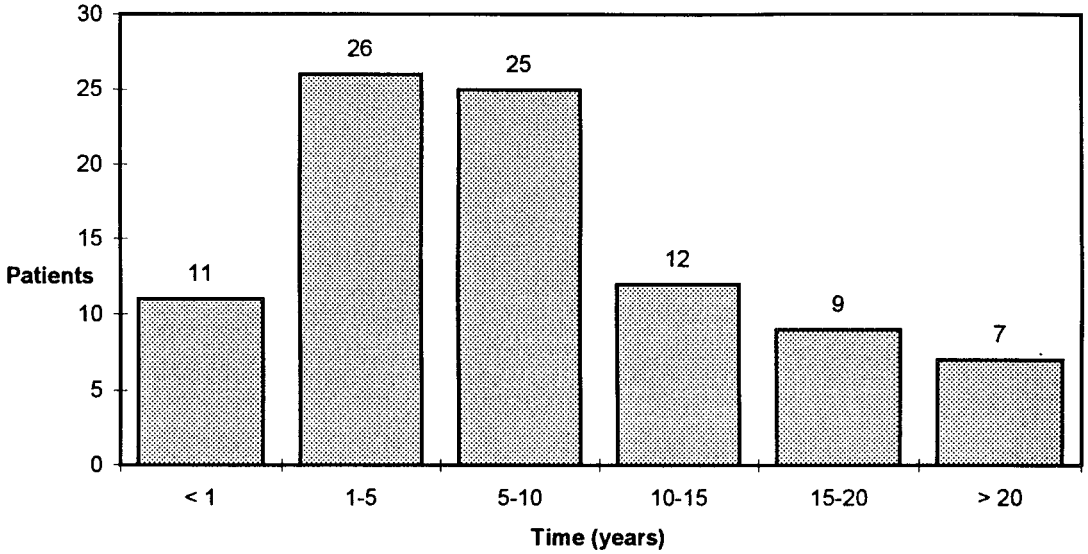

Fig. 4. Distribution of follow-up.

tion because of severe recurrent reflux, with good results in both cases. The fifth patient, previously described, underwent esophagectomy for adenocarcinoma of the distal esophagus 25 months after repair.

Discussion

Some clinical features recorded in this series of patients are characteristic of incarceration of the herniated stomach, which was frequently complicated by organoaxial volvulus. Common presentations of large incarcerated hernias are postprandial fullness or pain, dysphagia, iron-deficiency anemia related to chronic blood loss from the incarcerated gastric pouch, and regurgitation with aspiration. Our observations are similar to those documented in other reports of paraesophageal hiatus hernia.[4, 7-12] The incidence of chronic iron-deficiency anemia in our series (38%), however, is higher than that recorded in any other reports.[4, 9-12] It is emphasized that most patients with iron-deficiency anemia report no history of bleeding such as hematemesis or melena. These patients have such symptoms as pallor, palpitations, and exertional dyspnea but have no awareness of gastrointestinal bleeding.

Contrary to many previous reports, our observations indicate that most large paraesophageal hernias are an advanced stage of the common sliding type. Indeed, 91 of our 94 consecutive patients were judged to have a large sliding hernia, with a rolling or paraesophageal component and organoaxial volvulus in 50% of cases. The distinction between a massive sliding paraesophageal hernia and a true paraesophageal hernia (in which the esophagogastric junction remains in a normal intraabdominal location) is obviously important for the selection of operative management. The evidence that supports or confirms a diagnosis of sliding hernia in our patients was obtained from the history of symptomatic reflux in 78 cases (85%), an intrathoracic location of the esophagogastric junction at endoscopy in 91 patients, evidence of short esophagus from manometric measurements, and the intraoperative evaluation. We believe that acquired short esophagus is related to reflux esophagitis with scarring caused by inflammatory injury in the muscular wall of the distal thoracic esophagus.

The most compelling evidence for the diagnosis of sliding paraesophageal hernia is the endoscopic location of the esophagogastric junction at a level above the diaphragmatic hiatus. With the flexible gastroscope, it is possible for the experienced operator to easily locate the level of the esophagogastric junction and the level of the diaphragmatic hiatus. The diaphragmatic hiatus can be located by movement and contraction of the crural muscles during respiration in the awake patient. All of our patients underwent esophagoscopy by the operating surgeon before their repair. In all 91 of the patients with sliding paraesophageal hernia, the esophagogastric junction was located in the mediastinum above the diaphragmatic hiatus. This information is not re-

corded as a routine part of the preoperative evaluation in any of the other reported series.[2-5, 8-16]

In 1993, Williamson and colleagues[2] reported observations on 119 patients with paraesophageal hiatal hernia. Although they judged that 91 of these 119 patients had a pure paraesophageal hernia, there is no confirmatory evidence from their endoscopic records. Indeed, preoperative endoscopy was done in only 56% of patients. Although they state that contrast upper gastrointestinal radiography was performed in 96% of their patients and "demonstrated the typical radiologic appearance of a paraesophageal hiatal hernia," they make no note of the position of the esophagogastric junction in relation to the diaphragmatic hiatus or abdominal cavity. Indeed, it is our experience that the contrast radiograph is rarely helpful, nor has any report in the radiology literature indicated that the position of the esophagogastric junction can be regularly and accurately identified by radiographic means in patients with large paraesophageal hernias.

Walther and associates[3] reported on 18 patients with pure paraesophageal hernia. The only support for this diagnosis, however, is the statement that "under strict radiographic criteria as to the location of the gastroesophageal junction, 18 had a pure paraesophageal hernia with the gastroesophageal junction located well within the abdomen and a portion of the stomach displaced in the chest." They cite a reference in a 1979 textbook of radiology edited by Sutton.[17]

In a more recent publication by Allen and coworkers,[4] the type of paraesophageal hernia was determined "after operation." They judged 51 of 124 cases (41.1%) to be true paraesophageal hernias. In our opinion, it is difficult to judge the location of the esophagogastric junction during mobilization of the esophagus at the time of surgical repair. Furthermore, only 78% of patients underwent preoperative endoscopy, and there is no mention of the endoscopic location of the esophagogastric junction relative to the diaphragmatic hiatus in this report.

It is logical to assume that GERD is a common accompaniment of a large sliding paraesophageal hernia and is a rare feature in patients with a true paraesophageal hernia. GERD was recorded in 78 of our 91 patients with sliding paraesophageal hernias (85%). Symptomatic reflux was not recorded in any of our three patients with a true paraesophageal hernia. Although Williamson and colleagues[2] state that 91 of 119 cases had a true paraesophageal hernia, they record heartburn in 39 patients (33%). Among the 67 patients who underwent preoperative endoscopy, gross endoscopic esophagitis was identified in 16 (24%) cases. The reported incidences of GERD in patients with paraesophageal hernia vary between 5% and 85% and are tabulated in Table III.*

Table III. *Paraesophageal hernia: Reported incidence of GERD*

Year	*Authors*	*Incidence of GERD (%)*
1967	Skinner and Belsey[5]	Rare
1972	Orringer, Skinner, and Belsey[18]	—
1973	Ozdemir, Burke, and Ikins[13]	—
1979	Wichterman et al.[1]	5
1983	Pearson et al.[1]	85
1984	Walther et al.[3]	60
1987	Treacy and Jamisson[11]	72
1988	Menguy[9]	7
1993	Williamson et al.[2]	33
1993	Allen et al.[4]	16
1996	Fuller et al.[15]	27
1997	Maziak, Todd, and Pearson (current report)	83

Measurement of esophageal length at the time of esophageal manometry is a new and additional parameter that we have used since 1991. Patients with massive paraesophageal hernia have almost always had abnormally short esophagi. Indeed, in 13 of 13 consecutive patients with paraesophageal hiatus hernia in whom a complete manometric study was possible, the intersphincteric length was significantly shorter than that recorded in the normal population ($p = 0.0001$).

A gastroplasty was added to the fundoplication in 75 of our 94 patients. In many of these cases, the presence of acquired shortening was obvious or strongly suspected before the operation. At the operation, it is possible to make an additional judgment about esophageal length after mobilization of the intrathoracic esophagus from its mediastinal bed. In some cases, it is obvious that the esophagogastric junction rests clearly above the superior margin of the diaphragmatic hiatus and that any attempt at a standard antireflux repair would result in undue tension. These intraoperative observations are subjective and are not currently amenable to any meaningful or reproducible measurement. We have had the experience of complet-

*References 1-5, 9-11, 13, 15, and 18.

ing a standard Belsey Mark IV repair in some of these patients, only to find that the intrathoracic esophagus was tense and "bowstring" in the mediastinum because of unrecognized acquired shortening. It is easier to recognize tension from the thoracic side of the diaphragmatic hiatus.

The presence or absence of acquired short esophagus has an important bearing on the selection of antireflux reconstruction. We believe that the frequent addition of gastroplasty in our series of patients is responsible for the high proportion of good to excellent results and the low incidence of anatomic recurrence of the hernia. Indeed, the only anatomic recurrences of hiatus hernia in this series were in two of the 17 patients treated with a standard Belsey Mark IV repair. In the report by Williamson and colleagues[2] in which the results of 115 repairs were analyzed, they record that "13 symptomatic paraesophageal hernias recurred in 12 patients, an incidence of 11%. Recurrent hernias developed between 4 days and 12 years after the original operation, with a median of 2 years. Eight of these patients have undergone reoperation." This recurrence rate is exceptionally high, perhaps as a result of failure to recognize the complication of acquired esophageal shortening. All of these patients with recurrent paraesophageal hernia had undergone a transabdominal operation with closure of the diaphragmatic hiatus, with or without a gastropexy. This group did not consider an antireflux procedure necessary in patients with a presumed pure paraesophageal hernia.

The burgeoning enthusiasm for laparoscopic antireflux surgery has led to an increasing number of reports describing laparoscopic repair for large paraesophageal hernias.[19-24] Follow-up is necessarily short in these cases, and only one group has noted the specific problem of acquired shortening as a complication. Swanstrom, Marcus, and Galloway[23] reported on 35 patients with short esophagus who were treated with laparoscopic repair. Twenty-four of these 35 patients had paraesophageal hernias. There was anatomic recurrence in five of these 24 cases after laparoscopic fundoplication. Accordingly, in the next three patients with paraesophageal hernia and short esophagus, Swanstrom, Marcus, and Galloway[23] added a gastroplasty. They describe an ingenious technique with a right thoracoscopic approach for placement of an endostapler. If a standard fundoplication is used, one may anticipate a high incidence of recurrent herniation and reflux after laparoscopic repair of these giant hernias.

In conclusion, most paraesophageal hernias represent advanced degrees of sliding hiatus hernia with intrathoracic displacement of the esophagogastric junction. GERD occurs in 83% of these cases and frequently results in acquired short esophagus. Before the operation, the evaluation of acquired short esophagus is best determined from measurements of esophageal length during endoscopy and manometry. The position of the esophagogastric junction is difficult to locate in a traditional contrast esophagogram.

The presence of acquired short esophagus should influence the type of repair selected. Acquired short esophagus may be confirmed at operation and is most easily evaluated during the operation through a transthoracic approach.

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Discussion

Dr. Mark S. Allen *(Rochester, Minn.).* I compliment you on an excellent presentation. The management of an intrathoracic stomach continues to be an area of controversy, not only in the surgical literature but in the medical literature as well. I agree that there is a great deal of confusion about the classification of these defects and applaud your efforts to clarify the issue. I also believe that most patients with a paraesophageal hernia have some component of a sliding hernia and therefore some degree of GERD.

I am intrigued by your method of repair in these patients and am curious as to why you used an esophageal lengthening procedure in almost all cases. About 5 years ago at this meeting, we reported our experience from the Mayo Clinic and found that we achieved good or excellent results in 93% of patients after surgical repair in 115 patients with an intrathoracic stomach without any esophageal lengthening procedures. Our operative mortality rate was zero. In your series, you had two postoperative deaths, at least one of which was directly attributable to a leak from the staple line, and you had significant postoperative dysphagia in 11% of your patients. Do you think the increased risk is justified when similar results can be obtained without adding an esophageal lengthening procedure?

My next question relates to laparoscopic repair. I think I know the answer to this question, but because the minimally invasive repair is here to stay, whether or not we agree with it, I think it is important that we discuss it. My question is what advice do you have for those of us who do a laparoscopic repair when we see a patient with this type of lesion? The patient and the gastroenterologist both want a minimally invasive approach. What should we tell them?

Finally, I would like to know how you treat a patient with an intrathoracic stomach who is elderly, perhaps 80 years old, and has no symptoms. Should this be repaired electively, or just observed closely?

Dr. Maziak. In response to the first question, with regard to the gastroplasty we believe that because most of these were sliding hernias and, as the data I showed reveal, most of the patients have shortened esophagus according to investigations and manometry, the addition of a gastroplasty is required. We believe that this is essential for very low recurrence rate in the long-term follow-up.

With regard to laparoscopic hernias, Swanstrom, who has done quite a few repairs in cases of sliding hernias as well as paraesophageal hernias, reported that in 24 of 35 patients with paraesophageal hernias, five recurred. He now routinely adds a gastroplasty with an approach through the right side of the chest. His follow-up is still short, so it will be interesting to see his long-term results.

With respect to the older patient without symptoms, first, most of these patients are not free of symptoms; with a proper history, there is always some sort of symptom, whether postprandial fullness, early satiety, or bloating. However, assuming that this hypothetic patient truly is free of symptoms, we would not operate. In the article you published, you have, I think, followed up 21 patients for about 78 months and have had no complications or untoward sequelae. We also advocate following up the patient without symptoms.

Dr. F. Griffith Pearson *(Toronto, Ontario, Canada).* I do not want to discuss the article, but I had a question to ask Dr. Allen. If I remember correctly, you did an uncut gastroplasty in a large number of those 115 patients you mentioned, and an uncut gastroplasty is in fact a lengthening procedure because you do not have to envelop the distal esophagus with the same distance. Many of the patients in whom we have done a gastroplasty have the whole gastric tube below the diaphragm, which is like a cut version of what you are doing. Without doubt you have good results, and in my opinion what you have added is necessary to avoid the problem of undue tension on the repair. I cannot remember the number of patients in whom you performed it, but it was fairly high.

Dr. Maziak. It was 79 of the 115 patients.

Dr. David B. Skinner *(New York, N.Y.).* Dr. Altorki and I will be publishing a similar experience shortly. In fact, we tried to get it on this program, but your abstract beat us out. Our results are almost identical in terms of the mix of sliding and paraesophageal and so on. The big difference, though, is that there is a difference of having a shortened esophagus because the stomach is pushing it up as opposed to the esophagus being scarred from reflux and pulling the stomach up. The old kind, the short esophagus that we all used to see 30 years ago, is essentially gone these days. Those were mostly Barrett's esophagus cases that had a lot of stricture and scarring and shortening. So we believe that the shortened esophagus that you are talking about is shortened because the stomach pushed it

up there. If you put the stomach back down, as we have done routinely in all of our cases, you will get the same results from a standard antireflux repair that you do without a paraesophageal hernia. So we totally agree with the Mayo point of view that you do not need to do an esophageal lengthening procedure for these kinds of cases. You simply have to put the stomach where it belongs, and the esophagus will stretch back down again.

Dr. Mark B. Orringer *(Ann Arbor, Mich.)* Dave and I agree about the best method of esophagectomy for carcinoma, so we will agree about this also. I completely support your article. I have also found, Griff, ever since you presented your initial work on this topic years ago, that when we go to reduce the esophagogastric junction below the diaphragm and assess the degree of tension, it is extremely difficult to determine objectively whether there is shortening. The concept of "relative esophageal shortening" is quite real. It is a subjective sense that the esophagogastric junction just will not go down quite the way you would like it to without tension. I guess those of us who have become more comfortable with the Collis gastroplasty add it to the operation just as one would a relaxing incision in the groin. I believe that there is a lot of merit to this approach, which reduces the amount of postoperative morbidity by getting a tension-free repair. So I support your approach and hope to provide some additional corroborative data in the not too distant future.

Dr. Victor F. Trastek *(Rochester, Minn.).* You had 38% of patients who had chronic anemia. Could you tell us in how many of those the anemia resolved after repair of this hernia?

Dr. Maziak. All patients had resolution of chronic iron-deficiency anemia with the repair.