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Current Controversies in Paraesophageal Hernia Repair

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The management of paraesophageal hernia (PEH) has become one of the most widely debated and controversial areas in surgery. PEH's are relatively uncommon, often presenting in patients entering their seventh or eighth decades of life. These patients often bear complicating medical comorbidities making them potentially poor operative candidates. Taking this into account makes surgical management of these patients all the more complex. Many considerations must be taken into account in formulating a management strategy for patients with PEH, and these considerations have led surgeons into ongoing debates in recent decades (Box 1).

Common to all these discussions are the agreed-on tenets of PEH repair: reduction of the stomach to an intra-abdominal location, achieving adequate intra-abdominal esophageal length, excision of the hernia sac, and sound closure of the esophageal hiatus. This article reviews in detail a few of the major controversies in the evolution of the surgical approach to PEHs. Given the profile of the patients in whom these hernias occur, the indication for operation is perhaps the most important. Also discussed is the choice of surgical approach. Often the case in controversies of this sort, this debate typically blurs lines of subspecialty. Recently, the optimal technique for hiatal closure has been the most discussed topic, spurred by the revolution in hernia reinforcement by prosthetic and biologic materials. This progression was predictable as the use of mesh has spread from inguinal hernias to include repair of all manner of abdominal wall and even thoracic defects. This article does not pursue the discussion of the importance of "short esophagus" or preoperative evaluation, as these are discussed in detail in a previous issue in this series [1,2].

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Box 1. Current controversies in surgical management of paraesophageal hernias

Indications for surgery
Approach to surgery
- Transthoracic versus transabdominal
- Open versus Laparoscopic

Need for esophageal lengthening procedure
Closure of hiatus
- Simple suture
- Pledget reinforced
- Mesh-reinforced onlay or interpositiion tension-free
  - Prosthetic mesh
  - Biologic mesh

Need for antireflux procedure
Need for gastropexy
- Fundoplication
- Gastrostomy
- Anterior gastropexy

Classification of paraesophageal hernias

Perhaps one of the most agreed-on aspects within the PEH literature is their classification, as they generally are classified into four types ranging in extent and probably existing over a spectrum [3].

Type I: sliding hiatal hernias

Sliding hiatal hernias are the most common, accounting for 90% to 95% of cases. They are characterized by the migration of the gastroesophageal junction above the diaphragm with associated laxity of the phrenoesophageal ligament. These typically have a small, herniated peritoneal sac that can be reduced with gentle caudal retraction on the stomach. Patients typically suffer from typical symptoms of gastroesophageal reflux disease (GERD) resulting from a distortion of the anatomic characteristics of a normal hiatus, namely the absence of adequate intra-abdominal esophagus, and an altered angle of His. These hernias most likely are the consequence of increased intra-abdominal pressure and thus are seen more frequently in obese or pregnant patients, among others.

Type II: true paraesophageal hernias

Type II is ''true'' PEHs, characterized by herniation of the fundus of the stomach with an intact location of the gastroesophageal junction below the

diaphragm. They are considered relatively uncommon and probably account for less than 5% of patients diagnosed with PEH. They probably occur as a weakening of the pleuroperitoneal membrane, which allows the anterior wall the stomach to herniate into the potential space between the esophagus and the phrenoesophageal ligament. They typically are seen originating in the 12- or 4-o'clock position viewed transabdominally and in a minority of cases can become quite large. These hernias classically are associated with symptoms of dysphagia owing to compression of the lower esophagus by the herniated fundus. The gastroesophageal junction itself is considered intact so GERD symptoms frequently are not absent.

Type III: combined sliding and paraesophageal hernias

These generally are believed to account for the majority of PEHs. They are characterized by a combination of characteristics of types I and II, with the gastroesophageal junction located above the diaphragmatic crus and the upper portion of the stomach herniated into the posterior mediastinum. Like type II hernias, these can become large and likely represent the majority of large or "massive" PEHs. The upper portion of the stomach typically rolls into the posterior mediastinum, also originating at the 12- and 4-o'clock positions. Patients suffer the typical symptoms or GERD often, owing to the derangement of typical hiatal anatomy and the intrathoracic location of the gastroesophageal junction. In addition, atypical symptoms, such as regurgitation, chest pain, dysphagia, and respiratory symptoms, frequently are present due to the mechanical effects of the herniated stomach in the mediastinal space.

Type IV: complex paraesophageal hernias

Type IV is the rarest of the PEHs and defined by the intrathoracic migration of other intra-abdominal organs. This results from a combination of increased intra-abdominal pressure and a large hiatal defect. These are extreme examples of type II or type III hernias and manifest from the same process. They may or may not be associated with an abnormal location of the gastroesophageal junction; thus, patients may present with any of the typical or atypical symptoms seen in the other three hernia types, including symptoms attributable to organs herniated other than the stomach, often colon.

Indications for surgical treatment

The recommendations for surgery for patients were significantly affected by reports by Hill [4] and Skinner and Belsey [5] almost 4 decades ago. These articles raised concerns of significant complication rates suffered by patients left untreated. Skinner described 21 asymptomatic patients,

six (29%) of whom developed potentially catastrophic complications of bleeding, perforation, and strangulation when left untreated. In addition, operative mortality for emergent operations was estimated as high as 17%. The combination of these two observations led to surgical teaching that any patient who was medically fit for surgery should have the hernia repaired [6–10]. This dictum is based on small series largely from a different era, although little currently is known about the true natural history of PEHs. With careful history, some investigators suggest that nearly all patients who have PEHs are symptomatic [11]. Maziak and colleagues [11] described 94 patients undergoing thorough preoperative evaluation and found 94% had some degree of symptoms and further that 36% of the patients had erosive esophagitis. Modern pharmacotherapy may be sufficient for symptom control in a segment of patients, and these have been termed ''minimally symptomatic.''

In the past several years, investigators have questioned past surgical dictum and revisited defining the natural history of PEHs. Few studies exist that describe outcomes in unoperated patients. Allen and colleagues [12] described nonoperative management in 23 patients, who were followed for a mean of 78 months, with none developing acute complications requiring emergent operation. In this cohort, four patients did proceed to develop symptoms requiring repair, and there was one death from aspiration. They also included a cohort of 124 patients managed operatively, five of whom had surgery for emergent indications. Despite performing five emergent operations in that subset, they concluded from the nonoperative cohort that gastric strangulation was rare. Treacy and colleagues [13] evaluated progression of symptoms in 29 patients followed nonoperatively and found that 13 (45%) went on to require elective operations for progressive symptoms, although none was emergent.

Given the relative paucity of available data describing natural history, Stylopoulos and colleagues [14] designed a population-based decision analysis model used to determine if elderly asymptomatic, or minimally symptomatic, patients benefit from elective PEH repair. The model examined existing literature regarding mortality and hernia progression rates and modeled 5 million patients who were 65 years old and asymptomatic. They concluded that the mortality rate of emergency repair of PEHS likely was overestimated by early studies, likely only 5.4% versus the 17% quoted previously. Based on current literature, they also estimated that the mortality rate of elective PEH repair likely was 1.4% whereas the annual likelihood of developing gastric complications from the hernias was only 1.1%. This analysis indicated that fewer than one in five asymptomatic patients 65 years and older benefit from elective PEH repair and fewer than 1 in 10 patients at age of 85 and older.

These studies have led some to redefine their indications for repair [15]. The original observations inherent to the previous surgical dictum have changed. Although elective surgery clearly has improved mortality rates

over emergency surgery, outcomes have improved for emergent operations. This is likely because of better understanding and care of elderly, comorbid patients. Improved pharmacotherapy also offers the opportunity for improved symptom control for minimally symptomatic patients. Lastly, symptom progression is slow and less likely to evolve to emergent operation than previously believed. Many investigators now support conservative management for selected patients, agreeing that it is reasonable to follow asymptomatic, or minimally symptomatic, patients who are elderly and possess comorbidities.

Surgical approach

After the initial description of the PEH repair by Akerlund in 1926, patients who had PEH were managed for many decades via thoracotomy or laparotomy, with morbidity approximating 20% and mortality 2% [12,16–18]. Before the current era of minimally invasive surgery, debate was waged as to whether or not these repairs were approached best through the abdomen or thorax. The advent of minimally invasive techniques has revolutionized the approach to many surgical diseases and redefined gold standards of care. These techniques nearly universally improve morbidity and mortality significantly without degradation of efficacy, addressing a concern surgeons had with open approaches.

Transthoracic versus transabdominal

In general, excellent results have been reported with both open approaches (Table 1). As expected, the debate often follows the lines of subspecialty. Published series in the thoracic surgery literature are published predominantly by thoracic groups, whereas general surgeons publish the majority of transabdominal series.

Reviewing these series, proponents of the thoracic approach tout superior ability to mobilize the esophagus and ease of access to perform a Collis-Nissen gastroplasty as the main advantages to the thoracic approach [11,17,18]. They typically note a high incidence of short esophagus and report using an esophageal lengthening procedure in 69% to 97% of cases [11,12,25]. This is in contradistinction to investigators describing the transabdominal approach, who seem to find shortened esophagus rarely, citing that extensive mobilization of the esophagus allows for adequate intra-abdominal esophageal length and the need for Collis-Nissen to be minimal [20–22]. These data suggest that there is perhaps some aspect of the transthoracic exposure that lends itself to the appearance of a short esophagus, if not different sets of beliefs between thoracic and general surgeons. Recurrence rates typically are lowest in the transthoracic series, ranging from 0.8% to 2.6% [12,25]. Only one group critically studied patients postoperatively who had radiography or endoscopy, with most groups reporting

Table 1
Selected published series of open paraesophageal hernia repairs

Author	No. of patients	Mean follow-up (mo)	Mean length of stay (d)	Morbidity (%)	Symptomatic recurrence (%)	Radiographic recurrence (%)	Notes
Abdominal							
Ellis, et al [17]	55	52	9.5	24	9.1	NS	—
Williamson, et al [19]	124	61.5	NS	12	10.1	NS	Rarely need antireflux
Rakic, et al [20]	40	30	NS	5	2.5	NS	—
Geha, et al [21]	82	NS	NS	6	2	NS	—
Low and Unger [22]	72	30	4.5	23.6	NS	18	No Collis needed
Thoracic							
Allen, et al [12]	119	42	9	27	0.8	NS	Collis 69%
Maziak, et al [11]	94	72	NS	19	2.1	NS	Collis 80%
Altorki, et al [23]	47	45	NS	42	NS	NS	Collis 0%
Rogers, et al [24]	60	19	9	8	1.6	NS	—
Patel, et al [25]	240	42	7	8	2.6	7.9	Collis 97%

symptomatic recurrence as the principal outcome analyzed. In the lone study with objective follow-up, Patel and colleagues [25] reported 240 patients repaired transthoracically, with 97% requiring Collis gastroplasty. These patients were evaluated at 1-, 3-, and 5-year intervals with barium radiographs and of the 153 who had available reports, there were 19 recurrences (7.9%), considerably higher than the symptomatic recurrence rates reported other studies. Overall morbidity rates are highest in the transthoracic series, ranging from 8% to 42%, and mortality ranging from 0% to 2% [11,23,24,26]. Most concerning were reported leaks from the esophageal staple line at the site of the Collis gastroplasty, which occurred in as many as 4% of cases in these series [27]. Lengths of stay (LOS) usually are in excess of 7 days [11,24].

Those favoring the transabdominal approach cite less morbidity because of avoidance of the thoracotomy incision [21,28]. Post-thoracotomy pain syndrome is described in as many as 8% of patients undergoing thoracotomy [29]. Because far fewer patients undergo Collis gastroplasty, the risk for staple line leak can be avoided. Pulmonary complications also are believed fewer with the abdominal approach, as there in no need for single lung ventilation and easier pulmonary toilet postoperatively. Large series describing the transabdominal approach are available for review [17,20–22]. Symptomatic recurrence rates generally are excellent, although perhaps slightly higher than those reported in the thoracic literature, ranging from 2.5% to 13% [19,20]. It is again notable that in only one published series were these patients evaluated critically for recurrences with postoperative barium studies. Low and Unger recently described 72 patients approached transabdominally, with none requiring Collis gastroplasty [22]. After a mean follow-up of 29.8 months, 88% of patients underwent objective assessment with endoscopy or radiography. They found recurrences in 10 patients (18%) and emphasized that eight of them were small sliding hernias, which were asymptomatic. Reported morbidity rates for transabdominal approaches range from 5% to 24% [17,20]. Reported LOS ranges from 4.5 to 9.5 days [17,22].

These series contain many factors that confound interpretation. Simple analysis of recurrence rates, morbidity, and mortality is difficult because of the varied nuances of technique. The primary outcome in most early series was symptom recurrence, not critical analysis with objective studies. In more recent literature, spurred by intense investigation into the efficacy of minimally invasive techniques, recurrences typically are sought by radiographic follow-up. There is only one series published for each open approach bearing this type of data, and in both series, objective follow-up revealed significantly higher recurrence rates than previously published. It generally is accepted that morbidity rates for transabdominal approaches are less than those for trans-thoracic and that hospital LOS are shorter. It seems this also is true of mortality rates; however, this in not borne out in the literature.

Open versus laparoscopic

The first laparoscopic PEH repair was described by Cuschieri and colleagues [30] in 1992. Since that time this approach has been widely adopted, driven by patients and physicians desiring a less morbid repair. Recent years have seen the transabdominal laparoscopic approach gain rapid acceptance as standard, principally because of several studies that demonstrate significant improvement in morbidity rates versus the open approaches [31,32]. This notion is consistent with findings comparing outcomes of open and laparoscopic approaches for the management of other surgical diseases. Given this, there would be no debate if outcomes were measured solely in the early postoperative period. The initial concerns raised by advocates of open approaches cited the alarmingly high recurrence rates reported in laparoscopic series (Table 2) [33,37]. Unfortunately, there are surprisingly few series comparing the results of open and laparoscopic approaches directly, and there are no randomized controlled series [31–33]. These studies are difficult to interpret because they often report on heterogenous patient populations with varying lengths of follow-up and severity of disease and presentation [32]. Operative techniques (ie, use of gastropexy, fundoplication, and esophageal lengthening procedures) vary between series.

The most commonly cited series by proponents of open approaches was put forth by Hashemi and colleagues [33]. This is one of the few that directly compares open and laparoscopic approaches. They compared 54 patients undergoing PEH repair, 27 by laparoscopy and 27 by open approaches (13 thoracotomy and 14 laparotomy), with mean follow-up of 35 months in the open group and 17 months in the laparoscopic group. This series was one of the first to include symptomatic questionnaires and objective radiologic follow-up. They found 12 out of 41 patients had recurred and of these nine were in the laparoscopic group, yielding a radiographic recurrence rate of 42% versus 15% in the open group. They noted that patients repaired laparoscopically reported good symptomatic outcomes equivalent to those repaired open, although they expressed concern for high radiographic recurrence rates. Later, Khaitan and colleagues [37] reported on 25 patients treated laparoscopically, with follow-up in 15 patients at a mean of 25 months. These patients also underwent barium esophagram in addition to symptom analysis. They reported one patient having a recurrent PEH and five patients having migration of the wrap above the diaphragm for a total recurrence rate of 40%. Only two of the patients who had wrap migration were symptomatic and these were managed with proton pump inhibitors, with one patient requiring reoperation.

Early literature describing the feasibility of minimally invasive repair used symptomatic questionnaires to evaluate for recurrence and reported rates ranging from 5% to 21%, significantly lower than rates reported when anatomic recurrence is sought by barium esophagram, which range

Table 2
Selected published series of laparoscopic paraesophageal hernia repairs

Author	N	Follow-up (m)	Morbidity (%)	Mortality (%)	OR time (min)	Lenth of stay (d)	Anatomic recurrence (%)	Symptomatic recurrence (%)	Reoperation (%)
Gantert, et al [6]	55	11	14.4	1.8	219	2.4	NR	5.4	4
Hashemi, et al [33]	27	17	11	0	184	3	42	NR	NR
Luketich, et al [34]	100	12	28	1	220	2	NR	NR	1
Dahlberg, et al [35]	37	15	28	5.4	179	4	14	NR	5.4
Wiechmann, et al [10]	54	13	NR	1.8	202	NR	5.5	5.5	5.5
Jobe, et al [36]	56	37	19	0	186	2.6	32	19	4.2
Khaitan, et al [37]	25	25	16	0	268	5.5	40	12	4
Mattar, et al [38]	136	136	10.2	2.2	218	40	33	4.4	2
Diaz, et al [39]	116	116	4.3	1.7	162	NR	22	11	2.6
Targarona, et al [40]	37	37	11	2.1	196	4	20	21	NR
Andujar, et al [41]	166	15	8.4	0	160	3.8	28	NS	6
Aly, et al [42]	100	48	14	0	87	3.6	30	20	7

from 5.5% to 42% [6,10,33,40]. A recent review by Mehta and colleagues [43] analyzed studies reporting on laparoscopic PEH repairs with radiographic follow-up. They found an average recurrence rate of 27%, again largely assessed objectively. They noted that only 30% of these reported cases were true PEHs, with the remainder sliding hernias or wrap migration. These data compare with only two studies, reviewed previously in this article, with radiographic follow-up in patients repaired using open techniques [22,25]. These reported 7.9% and 18% recurrence rates.

Both approaches seem to have excellent symptomatic results [6,10,41]. The literature shows that quality-of-life measures are improved with patients undergoing laparoscopic repair [40,44]. Velanovich and Karmy-Jones analyzed quality-of-life data in 44 patients undergoing repair, three emergently for ischemia or gastric necrosis. In this series 31 patients were repaired laparoscopically with five conversions to open. They found higher quality-of-life scores in patients repaired laparoscopically, but these data were difficult to interpret given the emergent nature of many of the open operations and that five operations were converted. Targarona and colleagues evaluated 37 patients with Short Form 36 (SF-36) Health Survey and the Gastrointestinal Quality of Life Index and found improved to normal population with both instruments. Similar data exist for open procedures, as described by Low and Unger [21]. They noted in their series that SF-36 scores were better than age-matched population controls in eight of eight categories.

Advocates of minimally invasive approaches cite many other benefits, including improved visualization of the hiatus, easier esophageal mobilization high into the mediastinum, and decreased pulmonary complications. They also suggest that the impact of surgery is lessened in these elderly patients who have frequent medical comorbidities. Draaisma and colleagues [45] reviewed 32 published studies and found a significantly shorter LOS (3 days laparoscopic versus 10 days open). Postoperative complication rates were significantly lower for laparoscopic repairs (0% to 14% versus 5.3% to 25%) and lower median mortality rate (0.3% versus 1.7%), although the ranges for these values overlapped significantly. It seems generally accepted that recovery and complication rates immediately related to surgery are lower when minimally invasive techniques are used.

Techniques for hiatal closure

Critical analysis of recurrence rates led much of recent debate to focus on technique of hiatal closure. Historically, simple sutures, occasionally with the use of Teflon pledgets, were used to reapproximate the hiatal defect. Similar to the argument regarding surgical approach, investigators sought to decrease recurrence rates, knowing that the most common cause of failure for these procedures was intrathoracic wrap migration [46,47]. In the past decades, this discussion has evolved to include the use of synthetic or biologic prostheses for reinforcing or even bridging the crural closure. Standard

hernia repairs were anatomic based and used available autologous tissues to close and reinforce defects. The concept of tension-free repair, originally described by Lichtenstein and colleagues [48] for the repair of inguinal hernias, was popularized and applied more broadly to all other hernias. Prosthetic materials bridging defects in a tension-free manner were shown to lower recurrence rates and improve recurrence rates of inguinal hernia repairs [49–51]. Later this was applied widely to incisional hernia repair, becoming standard [52–54]. Because all PEHs were considered an indication for surgery in fit patients, it was only natural that the use of these materials at the hiatus would be investigated.

Prosthetic mesh

The first report of mesh at the hiatus was put forth by Kuster and Gilroy [55] in 1993. They described placing a patch of polyester mesh anterior to the esophagus in six patients whose crura were unable to be closed primarily during laparoscopic PEH repair. In short-term follow-up of 8 to 22 months, there were no mesh-related complications although two had herniation of the posterior portion of the fundus on radiographic studies that were asymptomatic. They concluded that this was not only feasible but safe in selected patients. Subsequently, many series were published reporting the use of a variety of materials and surgical techniques (Table 3). Early series were characterized by small numbers of patients who had large hiatal defects that were unable to be closed primarily. Only two prospective randomized trials have been done evaluating mesh hiatoplasty for PEHs and both demonstrated markedly lower recurrence rates [56,63]. Carlson and colleagues [56] repaired the hiatus with a keyhole-shaped polytetraflouroethylene (PTFE) patch in 15 patients and found no recurrences compared with three recurrences (18.8%) in the control group, although this did not reach statistical significance. Later, Frantzides and colleagues [27] randomized 72 patients who had hiatal defects larger than 8 cm to primary suture repair or keyhole PTFE graft. They studied all patients at 6-month intervals with esophagogastroduodenoscopy (EGD) or barium swallow, with a mean follow-up of 3.3 years. They found eight recurrences (22%) in the primary repair group versus none (0%) in the mesh hiatoplasty group and reported no mesh-related complications. In a third randomized study of fundoplications done for GERD, Granderath and colleagues [64] used a polypropylene onlay to the crural closure and found a significantly reduced wrap migration rate (8% versus 26%). These trials form the basis of the compelling argument made by several investigators that mesh hiatoplasty not only markedly decreases recurrence rates but also is safe with minimal mesh-related complications.

There are several limitations in interpreting the literature with regards to the use of mesh at the hiatus. Foremost is the lack of level 1 data, in particular regarding long-term outcomes. Most data come from small series with

Table 3
Selected published series of prosthetic mesh hiatoplasty

		No mesh		Mesh used				
Author	Follow-up	No. of patients	Recurrence (%)	No. of patients	Recurrence (%)	Material	Placement	Recurrence
Carlson, et al [56]	24	16	18.7	44	0	Polypropylene	Keyhole	NA
Champion and Rock [57]	25	—	—	52	1.9	Polypropylene	Onlay	Symptomatic
Hui [57]	37	12	0	12	8.3	PTFE	Onlay	Symptomatic
Frantzides [27]	39.6	36	22.7	36	0	PTFE	Keyhole	Radiologic
Basso [59]	22.5	65	13.8	70	0	Polypropylene	Interposition	Symptomatic
Leeder [60]	51	37	2.7	14	14.3	Polypropylene	Anterior	Symptomatic
Keidar [61]	58	23	17.3	10	10	Composite	Keyhole	Symptomatic
Horstmann [62]	14	—	—	16	0	Polypropylene	Onlay	Radiologic

short follow-up and reflects not only a variety of different prostheses but also a wide range of surgical techniques, making comparison difficult if not impossible. This was highlighted in a review by Targarona and colleagues [39], who illustrated at least nine different techniques for hiatal closure described. Many prosthetic materials have been investigated, including polypropylene, polyester, PTFE, and composite materials [57,58,61,65].

Some reports indicate a higher rate of dysphagia associated with mesh repair of the hiatus. In one nonrandomized series there was a significantly higher rate of dysphagia 3 months after surgery; however, this decreased at 1 year to a rate similar to that for nonmesh repairs. Granderath and colleagues [66] later investigated this finding in a randomized controlled trial looking at 40 patients undergoing laparoscopic Nissen fundoplication for GERD. The mesh repair performed was a posterior buttress of the crural closure with a 1 × 3 cm only a polypropylene mesh. Objective studies were performed at 3 months and 1 year after surgery. They found that resting lower esophageal sphincter (LES) pressure was higher in the mesh group at 1 year (13.9 versus 8.9 mm Hg); however, there was neither evidence for esophageal dysmotility nor any differences in LES length or relaxation. They again showed that at 1 year reported dysphagia rates were 5% in both groups, although early dysphagia rates again were higher (15%). They concluded that the earlier increased rate of dysphagia reported in mesh repairs resolves in longer-term follow-up with no evidence for induced esophageal dysmotility.

The most concerning issue related to the use of prosthetics near the esophagus is safety. History teaches consequences of previous attempts to place devices near the gastroesophageal junction [67]. The use of Teflon pledgets is associated with erosions into the viscera, foreign body reaction, and gastroesophageal fistula [68–70]. Thus far, reports of esophageal erosion of prosthetic mesh are few but nonetheless increasing [68,71–75]. This event can be catastrophic for these patients for whom in most cases an esophagogastrectomy is the only solution. In others, significant fibrosis of the hiatus leading to progressive dysphagia requiring esophagectomy is reported [9,47,76]. Animal studies confirm that these suspicions are warranted. Jansen and colleagues [77] studied the effect of two prosthetic mesh materials placed using a keyhole technique in rabbits. They found migration into the esophageal wall at 1 year in 5 of 6 animals using polypropylene and 5 of 9 animals using polypropylene-polyglecaprone composite material. It is of great concern that despite the marked improvement in recurrence rates reported in mesh hiatoplasty series, there is vast underreporting of the associated complications, leading to an illusion of safety.

Biologic mesh

The introduction of biologic mesh materials may provide a superior alternative to prosthetic for hiatal reinforcement. Surgeons agree that the ideal material would form minimal adhesions, incorporate into the hiatal closure

without inducing significant fibrosis, and provide sound closure, with no mesh-related complications. To date, porcine small intestinal submucosa (SIS) and acellular human dermis (AHD) have been used to repair or reinforce the hiatal closure (Table 4) [78–83]. Ample scientific evidence exists to support the increasingly popular notion that these materials might be ideal for PEH repair. Investigators have shown that SIS initially degrades but then is replaced over time by a remodeling process, which leads to tissue strength which exceeds that of native tissue over time [85,86]. Similar findings are demonstrated with AHD [87]. In addition, these materials are shown to be resistant to infection and to have strength similar to that of PTFE at the time of placement in surgery [87].

The most critical issue driving the use of biologics at the hiatus is whether or not esophageal erosion, or fibrosis leading to progressive dysphagia, can be avoided. This likely would eliminate the potential for patients to require esophagogastrectomy as a consequence of implant-related complications. Animal studies again support that biologics might prove useful. Desai and colleagues [88] performed laparoscopic PEH repair using porcine SIS on six canines with histologic analysis at 1 year. They found no clinical evidence for dysphagia or weight loss and histologically noted good reinforcement of the hiatus and circumferential ingrowth of connective tissue and skeletal muscle into the graft, concluding the graft may act as a scaffold for tissue ingrowth and does not lead to erosion.

The initial clinical report of PEH repair supplemented with a biologic mesh was by Oelschlager and colleagues [78], who described the use of SIS in nine patients as a feasibility study. That group later followed this case series with a multicenter, prospective randomized trial in which 108 patients were randomized to repair with or without onlay crural reinforcement with the biologic graft, placed in a keyhole fashion [82]. Although follow-up was short (6 months), 90% of patients were available for objective assessment with upper gastrointestinal (UGI) series. They found a significant decrease in recurrence rates (9% mesh versus 24% nonmesh), and moreover, reported no mesh-related complications. In addition, the patients had no difference in symptom severity scores, in particular for dysphagia, which previously was shown to be an issue in the short-term with prosthetic mesh closures. In reviewing other series, reported recurrence rates have ranged from 0% to 11.8%, with studies characterized by small numbers and short-term follow-up. These series, however, include objective follow-up with EGD or UGI in the majority of patients, allowing for complete early assessment of results.

Do recurrences matter?

Many causal factors are proposed, including tension on the crural repair, large hiatal defects, attenuated muscle fibers at the hiatus, and inappropriate diaphragmatic stressors in the early postoperative period [89]. In a review,

Table 4
Selected published series of biologic mesh hiatoplasty

Author	n	Follow-up (m)	Material	Placement	Morbidity (%)	Mortality (%)	Radiographic recurrence (%)	Symptomatic recurrence (%)
Oelschlager, et al [78]	9	8	SIS	Keyhole	11	11	11	0
Strange [79]	12	6	SIS	Keyhole	0	0	0	0
Wisbach, et al [80]	11	12	Alloderm	Onlay-Y	9	0	9	0
Ringley, et al [81]	22	6.7	Alloderm	Onlay-U	4	0	0	0
Oelschlager, et al [82]	51	6	SIS	Onlay- Keyhole	24	0	9	NR
Lee, et al [83]	17	14.4	Alloderm	Onlay-V	6	0	11.8	6
St. Peter, et al [84]	12	36	SIS	Onlay-Keyhole	0	0	0	0

Puri and colleagues [90] found that surgeon inexperience, retention of the hernia sac, early postoperative vomiting, and heavy lifting were the only factors supported by the literature to lead to increased recurrence rates. They also suggest that some of these problems might be augmented by the quicker recovery seen after minimally invasive approaches. It is known that recurrences occur usually soon after surgery and usually are the result of intrathoracic migration of the fundoplication [91–93]. This also is the most likely reason for patients requiring revisional surgery.

The significance of these recurrences must be addressed. The short answer to the question is that, yes, anatomic recurrence must be considered a technical failure. Despite this, data support that many radiographic recurrences are not clinically significant. Many patients who have anatomic recurrences are asymptomatic and require no further therapy [33,38]. Mehta and colleagues [43] reviewed the available literature and found that the majority of recurrences represented intrathoracic wrap migration or wrap disruption but indicated that only 3% of patients who had radiographic recurrence required revisional procedures. With this data in mind, it also seems that most of those patients who are symptomatic can be easily managed with conservative therapy, averting need for further operation. This notion also is supported by the generally excellent results reported in patients by quality-of-life questionnaires and GERD-specific questionnaires, despite many of whom having known anatomic recurrences. The reoperation rates for laparoscopic and open series are less than 5%, indicating that many of the identified anatomic recurrences are unlikely to be clinically significant. Long-term data as to the natural history of these anatomic recurrences are lacking.

Summary

PEHs remain a complex and challenging disorder to treat in a patient population that has many confounding medical factors. Excellent outcomes are reported using a variety of different approaches and a variety of nuances to those approaches. At the author's institution, laparoscopic approach is believed to offer recovery advantages to patients that are difficult to ignore. With this in mind, maximization of the efficacy of this technique has the potential for superior outcomes generally. The literature regarding this topic is replete with small studies and few randomized data. Given the infrequency of this disease, it is likely this characteristic will not change. The use of mesh at the hiatus to supplement the use of minimally invasive techniques is encouraging and likely represents the best choice for patients. That said, the use of prosthetics introduces a potentially catastrophic outcome for patients, which, the author believes, is greatly underreported. This notion likely will bear out in time. Biologic materials offer a safer and equally effective alternative for bolstering the crural repair that eliminates this risk and likely will become the standard of care.

References

[1] Hoang CD, Koh PS, Maddaus MA. Short esophagus and esophageal stricture. Surg Clin North Am 2005;85:433.

[2] Landreneau RJ, Del Pino M, Santos R. Management of paraesophageal hernias. Surg Clin North Am 2005;85:411.

[3] Landreneau RJ, Johnson JA, Marshall JB, et al. Clinical spectrum of paraesophageal herniation. Dig Dis Sci 1992;37:537.

[4] Hill LD. Incarcerated paraesophageal hernia. A surgical emergency. Am J Surg 1973; 126:286.

[5] Skinner DB, Belsey RH. Surgical management of esophageal reflux and hiatus hernia. Long-term results with 1,030 patients. J Thorac Cardiovasc Surg 1967;53:33.

[6] Gantert WA, Patti MG, Arcerito M, et al. Laparoscopic repair of paraesophageal hiatal hernias. J Am Coll Surg 1998;186:428.

[7] Halpin VJ, Soper NJ. Paraesophageal hernia. Curr Treat Options Gastroenterol 2001;4:83.

[8] Hawasli A, Zonca S. Laparoscopic repair of paraesophageal hiatal hernia. Am Surg 1998; 64:703.

[9] van der Peet DL, Klinkenberg-Knol EC, Alonso Poza A, et al. Laparoscopic treatment of large paraesophageal hernias: both excision of the sac and gastropexy are imperative for adequate surgical treatment. Surg Endosc 2000;14:1015.

[10] Wiechmann RJ, Ferguson MK, Naunheim KS, et al. Laparoscopic management of giant paraesophageal herniation. Ann Thorac Surg 2001;71:1080.

[11] Maziak DE, Todd TR, Pearson FG. Massive hiatus hernia: evaluation and surgical management. J Thorac Cardiovasc Surg 1998;115:53.

[12] Allen MS, Trastek VF, Deschamps C, et al. Intrathoracic stomach. Presentation and results of operation. J Thorac Cardiovasc Surg 1993;105:253.

[13] Treacy PJ, Jamieson GG. An approach to the management of para-oesophageal hiatus hernias. Aust N Z J Surg 1987;57:813–7.

[14] Stylopoulos N, Gazelle GS, Rattner DW. Paraesophageal hernias: operation or observation? Ann Surg 2002;236:492.

[15] Floch NR. Paraesophageal hernias: current concepts. J Clin Gastroenterol 1999;29:6.

[16] Akerlund A, Ohnell A, Key E. Hernia diaphragmatica hiatus oesophagei vom anatomischen und rontgenologischen gesichtspunkt. Acta Radiol 1926;6:3.

[17] Ellis FH Jr, Crozier RE, Shea JA. Paraesophageal hiatus hernia. Arch Surg 1986;121:416.

[18] Pearson FG, Cooper JD, Ilves R, et al. Massive hiatal hernia with incarceration: a report of 53 cases. Ann Thorac Surg 1983;35:45.

[19] Williamson WA, Ellis FH Jr, Streitz JM Jr, et al. Paraesophageal hiatal hernia: is an antireflux procedure necessary? Ann Thorac Surg 1993;56:447.

[20] Rakic S, Pesko P, Dunjic MS, et al. Paraoesophageal hernia repair with and without concomitant fundoplication. Br J Surg 1994;81:1162.

[21] Geha AS, Massad MG, Snow NJ, et al. A 32-year experience in 100 patients with giant paraesophageal hernia: the case for abdominal approach and selective antireflux repair. Surgery 2000;128:623.

[22] Low DE, Unger T. Open repair of paraesophageal hernia: reassessment of subjective and objective outcomes. Ann Thorac Surg 2005;80:287.

[23] Altorki NK, Yankelevitz D, Skinner DB. Massive hiatal hernias: the anatomic basis of repair. J Thorac Cardiovasc Surg 1998;115:828.

[24] Rogers ML, Duffy JP, Beggs FD, et al. Surgical treatment of para-oesophageal hiatal hernia. Ann R Coll Surg Engl 2001;83:394.

[25] Patel HJ, Tan BB, Yee J, et al. A 25-year experience with open primary transthoracic repair of paraesophageal hiatal hernia. J Thorac Cardiovasc Surg 2004;127:843.

[26] Myers GA, Harms BA, Starling JR. Management of paraesophageal hernia with a selective approach to antireflux surgery. Am J Surg 1995;170:375.

[27] Frantzides CT, Madan AK, Carlson MA, et al. A prospective, randomized trial of laparoscopic polytetrafluoroethylene (PTFE) patch repair vs simple cruroplasty for large hiatal hernia. Arch Surg 2002;137:649.
[28] Menguy R. Surgical management of large paraesophageal hernia with complete intrathoracic stomach. World J Surg 1988;12:415.
[29] Stirling MC, Orringer MB. Continued assessment of the combined Collis-Nissen operation. Ann Thorac Surg 1989;47:224.
[30] Cuschieri A, Shimi S, Nathanson LK. Laparoscopic reduction, crural repair, and fundoplication of large hiatal hernia. Am J Surg 1992;163:425.
[31] Ferri LE, Feldman LS, Stanbridge D, et al. Should laparoscopic paraesophageal hernia repair be abandoned in favor of the open approach? Surg Endosc 2005;19:4.
[32] Schauer PR, Ikramuddin S, McLaughlin RH, et al. Comparison of laparoscopic versus open repair of paraesophageal hernia. Am J Surg 1998;176:659.
[33] Hashemi M, Peters JH, DeMeester TR, et al. Laparoscopic repair of large type III hiatal hernia: objective followup reveals high recurrence rate. J Am Coll Surg 2000;190:553.
[34] Luketich JD, Raja S, Fernando HC, et al. Laparoscopic repair of giant paraesophageal hernia: 100 consecutive cases. Ann Surg 2000;232:608.
[35] Dahlberg PS, Deschamps C, Miller DL, et al. Laparoscopic repair of large paraesophageal hiatal hernia. Ann Thorac Surg 2001;72:1125.
[36] Jobe BA, Aye RW, Deveney CW, et al. Laparoscopic management of giant type III hiatal hernia and short esophagus. Objective follow-up at three years. J Gastrointest Surg 2002; 6:181.
[37] Khaitan L, Houston H, Sharp K, et al. Laparoscopic paraesophageal hernia repair has an acceptable recurrence rate. Am Surg 2002;68:546.
[38] Mattar SG, Bowers SP, Galloway KD, et al. Long-term outcome of laparoscopic repair of paraesophageal hernia. Surg Endosc 2002;16:745.
[39] Diaz S, Brunt LM, Klingensmith ME, et al. Laparoscopic paraesophageal hernia repair, a challenging operation: medium-term outcome of 116 patients. J Gastrointest Surg 2003; 7:59.
[40] Targarona EM, Novell J, Vela S, et al. Mid term analysis of safety and quality of life after the laparoscopic repair of paraesophageal hiatal hernia. Surg Endosc 2004;18:1045.
[41] Andujar JJ, Papasavas PK, Birdas T, et al. Laparoscopic repair of large paraesophageal hernia is associated with a low incidence of recurrence and reoperation. Surg Endosc 2004;18:444.
[42] Aly A, Munt J, Jamieson GG, et al. Laparoscopic repair of large hiatal hernias. Br J Surg 2005;92:648.
[43] Mehta S, Boddy A, Rhodes M. Review of outcome after laparoscopic paraesophageal hiatal hernia repair. Surg Laparosc Endosc Percutan Tech 2006;16:301.
[44] Velanovich V, Karmy-Jones R. Surgical management of paraesophageal hernias: outcome and quality of life analysis. Dig Surg 2001;18:432.
[45] Draaisma WA, Gooszen HG, Tournoij E, et al. Controversies in paraesophageal hernia repair: a review of literature. Surg Endosc 2005;19:1300.
[46] Carlson MA, Frantzides CT. Complications and results of primary minimally invasive antireflux procedures: a review of 10,735 reported cases. J Am Coll Surg 2001;193:428.
[47] Trus TL, Bax T, Richardson WS, et al. Complications of laparoscopic paraesophageal hernia repair. J Gastrointest Surg 1997;1:221.
[48] Lichtenstein IL, Shulman AG, Amid PK, et al. The tension-free hernioplasty. Am J Surg 1989;157:188.
[49] Brown JH, Khaira HS. Early results with the Lichtenstein tension-free hernia repair. Br J Surg 1995;82:419.
[50] Davies N, Thomas M, McIlroy B, et al. Early results with the Lichtenstein tension-free hernia repair. Br J Surg 1994;81:1478.
[51] Hinson EL. Early results with Lichtenstein tension-free hernia repair. Br J Surg 1995;82:418.

[52] Korenkov M, Sauerland S, Arndt M, et al. Randomized clinical trial of suture repair, polypropylene mesh or autodermal hernioplasty for incisional hernia. Br J Surg 2002;89:50.
[53] Millikan KW. Incisional hernia repair. Surg Clin North Am 2003;83:1223.
[54] Utrera Gonzalez A, de la Portilla de Juan F, Carranza Albarran G. Large incisional hernia repair using intraperitoneal placement of expanded polytetrafluoroethylene. Am J Surg 1999;177:291.
[55] Kuster GG, Gilroy S. Laparoscopic technique for repair of paraesophageal hiatal hernias. J Laparoendosc Surg 1993;3:331.
[56] Carlson MA, Richards CG, Frantzides CT. Laparoscopic prosthetic reinforcement of hiatal herniorrhaphy. Dig Surg 1999;16:407.
[57] Champion JK, Rock D. Laparoscopic mesh cruroplasty for large paraesophageal hernias. Surg Endosc 2003;17:551.
[58] Hui TT, Thoman DS, Spyrou M, et al. Mesh crural repair of large paraesophageal hiatal hernias. Am Surg 2001;67:1170.
[59] Basso N, De Leo A, Genco A, et al. 360 degrees laparoscopic fundoplication with tension-free hiatoplasty in the treatment of symptomatic gastroesophageal reflux disease. Surg Endosc 2000;14:164.
[60] Leeder PC, Smith G, Dehn TC. Laparoscopic management of large paraesophageal hiatal hernia. Surg Endosc 2003;17:1372.
[61] Keidar A, Szold A. Laparoscopic repair of paraesophageal hernia with selective use of mesh. Surg Laparosc Endosc Percutan Tech 2003;13:149.
[62] Horstmann R, Klotz A, Classen C, et al. Feasibility of surgical technique and evaluation of postoperative quality of life after laparoscopic treatment of intrathoracic stomach. Langenbecks Arch Surg 2004;389:23.
[63] Frantzides CT, Richards CG, Carlson MA. Laparoscopic repair of large hiatal hernia with polytetrafluoroethylene. Surg Endosc 1999;13:906.
[64] Granderath FA, Schweiger UM, Kamolz T, et al. Laparoscopic Nissen fundoplication with prosthetic hiatal closure reduces postoperative intrathoracic wrap herniation: preliminary results of a prospective randomized functional and clinical study. Arch Surg 2005; 140:40.
[65] Granderath FA, Schweiger UM, Pointner R. Laparoscopic antireflux surgery: tailoring the hiatal closure to the size of hiatal surface area. Surg Endosc 2007;21:542.
[66] Granderath FA, Kamolz T, Schweiger UM, et al. Impact of laparoscopic nissen fundoplication with prosthetic hiatal closure on esophageal body motility: results of a prospective randomized trial. Arch Surg 2006;141:625.
[67] Purkiss SF, Argano VA, Kuo J, et al. Oesophageal erosion of an Angelchik prosthesis: surgical management using fundoplication. Eur J Cardiothorac Surg 1992;6:517.
[68] Arendt T, Stuber E, Monig H, et al. Dysphagia due to transmural migration of surgical material into the esophagus nine years after Nissen fundoplication. Gastrointest Endosc 2000;51:607.
[69] Baladas HG, Smith GS, Richardson MA, et al. Esophagogastric fistula secondary to Teflon pledget: a rare complication following laparoscopic fundoplication. Dis Esophagus 2000; 13:72.
[70] Dally E, Falk GL. Teflon pledget reinforced fundoplication causes symptomatic gastric and esophageal lumenal penetration. Am J Surg 2004;187:226.
[71] Carlson MA, Condon RE, Ludwig KA, et al. Management of intrathoracic stomach with polypropylene mesh prosthesis reinforced transabdominal hiatus hernia repair. J Am Coll Surg 1998;187:227.
[72] Casabella F, Sinanan M, Horgan S, et al. Systematic use of gastric fundoplication in laparoscopic repair of paraesophageal hernias. Am J Surg 1996;171:485.
[73] Coluccio G, Ponzio S, Ambu V, et al. [Dislocation into the cardial lumen of a PTFE prosthesis used in the treatment of voluminous hiatal sliding hernia, a case report]. Minerva Chir 2000;55:341 [in Italian].

[74] Hergueta-Delgado P, Marin-Moreno M, Morales-Conde S, et al. Transmural migration of a prosthetic mesh after surgery of a paraesophageal hiatal hernia. Gastrointest Endosc 2006; 64:120 [discussion: 121].
[75] Tatum RP, Shalhub S, Oelschlager BK, et al. Complications of PTFE Mesh at the Diaphragmatic Hiatus. J Gastrointest Surg 2007;12:953–7.
[76] Edelman DS. Laparoscopic paraesophageal hernia repair with mesh. Surg Laparosc Endosc 1995;5:32.
[77] Jansen M, Otto J, Jansen PL, et al. Mesh migration into the esophageal wall after mesh hiatoplasty: comparison of two alloplastic materials. Surg Endosc 2007;21:2298.
[78] Oelschlager BK, Barreca M, Chang L, et al. The use of small intestine submucosa in the repair of paraesophageal hernias: initial observations of a new technique. Am J Surg 2003; 186:4.
[79] Strange PS. Small intestinal submucosa for laparoscopic repair of large paraesophageal hiatal hernias: a preliminary report. Surg Technol Int 2003;11:141.
[80] Wisbach G, Peterson T, Thoman D. Early results of the use of acellular dermal allograft in type III paraesophageal hernia repair. JSLS 2006;10:184.
[81] Ringley CD, Bochkarev V, Ahmed SI, et al. Laparoscopic hiatal hernia repair with human acellular dermal matrix patch: our initial experience. Am J Surg 2006;192:767.
[82] Oelschlager BK, Pellegrini CA, Hunter J, et al. Biologic prosthesis reduces recurrence after laparoscopic paraesophageal hernia repair: a multicenter, prospective, randomized trial. Ann Surg 2006;244:481.
[83] Lee E, Frisella MM, Matthews BD, et al. Evaluation of acellular human dermis reinforcement of the crural closure in patients with difficult hiatal hernias. Surg Endosc 2007;21:641.
[84] St Peter SD, Ostlie DJ, Holcomb GW III. The use of biosynthetic mesh to enhance hiatal repair at the time of redo Nissen fundoplication. J Pediatr Surg 2007;42:1298.
[85] Badylak S, Kokini K, Tullius B, et al. Strength over time of a resorbable biosc.affold for body wall repair in a dog model. J Surg Res 2001;99:282.
[86] Gloeckner DC, Sacks MS, Billiar KL, et al. Mechanical evaluation and design of a multilayered collagenous repair biomaterial. J Biomed Mater Res 2000;52:365.
[87] Choe JM, Kothandapani R, James L, et al. Autologous, cadaveric, and synthetic materials used in sling surgery: comparative biomechanical analysis. Urology 2001;58:482.
[88] Desai KM, Diaz S, Dorward IG, et al. Histologic results 1 year after bioprosthetic repair of paraesophageal hernia in a canine model. Surg Endosc 2006;20:1693.
[89] Kakarlapudi GV, Awad ZT, Haynatzki G, et al. The effect of diaphragmatic stressors on recurrent hiatal hernia. Hernia 2002;6:163.
[90] Puri V, Kakarlapudi GV, Awad ZT, et al. Hiatal hernia recurrence: 2004. Hernia 2004;8:311.
[91] Hatch KF, Daily MF, Christensen BJ, et al. Failed fundoplications. Am J Surg 2004; 188:786.
[92] Hunter JG, Smith CD, Branum GD, et al. Laparoscopic fundoplication failures: patterns of failure and response to fundoplication revision. Ann Surg 1999;230:595.
[93] Soper NJ, Dunnegan D. Anatomic fundoplication failure after laparoscopic antireflux surgery. Ann Surg 1999;229:669.