# Chapter 13
# Paraesophageal Hernias: Indications and Surgical Treatment

**Ezra N. Teitelbaum and Nathaniel J. Soper**

**Abstract** Laparoscopic paraesophageal hiatal hernia repair is a complex operation that requires experience with advanced minimally invasive surgical techniques, as well as an expertise in both the anatomy and physiology of the esophagus and stomach. When performed correctly the operation should result in a high rate of symptomatic resolution with a low complication profile, despite often being performed in patients who are elderly with multiple medical comorbidities. However, if the principles of a proper repair are not followed, patients can be left with persistent dysphagia and/or gastroesophageal reflux, resulting in a worse quality of life than they had preoperatively and possibly necessitating reoperation. This chapter outlines the preoperative assessment, evaluation, and indications for surgery in patients presenting with paraesophageal hernia. The key steps and components of a laparoscopic repair are detailed, with an emphasis on adherence to the fundamentals of creating a functional repair. These include creating a setup and port placement that allows for efficient and effective operating, complete dissection and reduction of the hernia sac, mobilization of the distal esophagus, performing a tension-free crural repair, and creation of an effective antireflux fundoplication. The decisions of when to perform an esophageal lengthening procedure and/or reinforce the crural repair with a mesh are also addressed. While surgeons must tailor their technique to their own operating style and individual patient anatomy, if these basic principles and steps are adhered to, the operation should lead to successful and durable outcomes on a consistent basis.

**Keywords** Paraesophageal hernia • Hiatal hernia • Foregut surgery • Laparoscopy • Gastroesophageal reflux • Nissen fundoplication • Crural repair • Esophageal lengthening procedure • Esophageal physiology

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**Table 13.1** The four types of hiatal hernias

| Hiatal hernia type | Anatomy |
|---|---|
| I | The EGJ herniates above the diaphragmatic crura, often moving transiently from the abdomen into the mediastinum |
| II | A portion of the stomach is herniated into the mediastinum alongside the esophagus, with the EGJ in normal (i.e., intra-abdominal) position |
| III | The EGJ is above the hiatus and a portion, or the entirety, of the stomach is folded alongside the esophagus |
| IV | An intra-abdominal organ other than the stomach is additionally herniated through the hiatus |

## Introduction and Hernia Classification

Hiatal hernias result from a widening of the diaphragmatic crura and a weakening of the phrenoesophageal membrane [1]. This results in a protrusion of a hernia sac containing intra-abdominal organs through the diaphragmatic hiatus and into the mediastinum. This displacement can result in a wide range of symptoms and potentially lead to gastric incarceration and strangulation, a life-threatening emergency. For this reason, hernia repair is generally indicated for patients with symptomatic hernias. The technical aspects of such operations have undergone significant evolution in the last century [2], and laparoscopy is now considered the preferred approach, offering reductions in pain, convalescence, hospital length of stay, and morbidity, when compared with laparotomy or thoracotomy [3, 4]. However, many controversies still remain, including whether to reinforce the crural closure with mesh, how frequently an esophageal lengthening procedure is necessary, and the role of a concomitant antireflux procedure [5]. This chapter will address the work-up and preoperative evaluation of patients with paraesophageal hernia, describe the technical aspects of a laparoscopic repair as we perform it, and review the literature regarding the unresolved debates over optimal technique.

Hiatal hernias are subclassified into four types (Table 13.1). In a type I hiatal hernia, the esophagogastric junction (EGJ) migrates cephalad to the crura, resulting in a portion of intrathoracic stomach. As the EGJ forms the lead point of herniation between the abdomen and mediastinum, type I hiatal hernias are also termed "sliding hernias." Type I hernias are by far the most common form of hiatal hernia, making up 95 % of the total prevalence. Type II, III, and IV hernias are together termed paraesophageal hernias (PEH) and combined account for the remaining 5 % of hiatal hernias. Type II anatomy consists of a hernia in which a portion of the stomach (usually the fundus) has migrated through the hiatus and into mediastinum but with an EGJ that remains below the diaphragm. In a type III hernia, the EGJ is above the diaphragm and a portion of the stomach is additionally present within the chest and alongside the esophagus. Type III hernias are typically caused by a large crural separation which can result in a large portion, or the entirety, of the stomach lying intrathoracically. For this reason, type III hernias are often referred to as "giant

paraesophageal hernias." Type IV is defined as any hiatal hernia in which an intra-abdominal organ other than the stomach has also migrated through the crura. Common examples are the omentum, small bowel, transverse colon, spleen, and/or pancreas.

## Presenting Symptoms

Patients with PEHs commonly present with symptoms due to either intermittent obstruction or gastroesophageal reflux (GER). Obstruction is caused by a kinking of the esophagus and/or stomach and results in episodes of dysphagia, early satiety, regurgitation, nausea, vomiting, and/or chest pain. The anatomic distortion of PEHs often leads to an incompetence of normal EGJ function [6]. This in turn causes GER, with its characteristic symptom of intermittent retrosternal heartburn, which is often postprandial and exacerbated when supine. PEHs can also result in erosions of the gastric mucosa, termed "Cameron ulcers." These ulcers can cause anemia from chronic bleeding, and their exact etiology has not been conclusively determined [7]. Friction from repeated passage of the stomach through the hiatus, increased acid exposure from stasis of gastric juices, and ischemia have all been proposed as causal mechanisms [7, 8]. Larger type III and IV hernias can additionally cause respiratory and cardiac impairment via direct compression of the lungs and heart [9].

The symptoms discussed so far are usually subacute, and patients can suffer for prolonged periods of time while being evaluated and are often incorrectly treated for more common conditions such as non-hernia-related GER, peptic ulcer disease, angina, and biliary colic. This scenario of clinical manifestation is distinct from patients who present acutely with an incarcerated PEH. Acute PEH incarceration is a life-threatening surgical emergency, as it can lead to gastric ischemia and, if not alleviated, necrosis. The classic presenting symptoms and signs of an acute incarceration are "Borchardt's triad" of chest pain, the urge but inability to vomit, and failure of nasogastric tube passage below the diaphragm. Immediate reduction of the hernia is required to restore blood flow to the stomach, and a laparotomy or thoracotomy is often necessary to achieve this. The remainder of this chapter will address only the evaluation and management of patients with PEH in an elective setting.

## Indications for Surgery

Based on the potential for gastric incarceration, it was a long accepted surgical principle that PEHs should be repaired on an elective basis when discovered, regardless of the patient's symptoms [10, 11]. This traditional assumption was challenged by a landmark study by Stylopoulos and colleagues in 2002 [12]. The authors

constructed a Markov Monte Carlo analytic model using pooled outcomes data to estimate quality of life years for patients with asymptomatic PEH, treated with either laparoscopic repair or watchful waiting. This analysis showed that watchful waiting resulted in a yearly acute incarceration rate of only 1.1 %, and was superior to surgery for 83 % of patients. Based on these findings, expectant management is now considered a reasonable option in patients with truly asymptomatic PEH. On the other hand, the presence of any symptoms related to PEH, whether due to obstruction or GER, is considered an indication for laparoscopic repair, as long as the patient is of reasonable operative risk.

## Preoperative Evaluation

In addition to a thorough history and physical examination, several tests are indicated preoperatively in order to secure the diagnosis of PEH and help define the anatomy and physiology of the esophagus and stomach. Contrast esophagram, or an "upper GI study," forms the basis for diagnosis of PEH and description of its anatomy. The location of the esophagus, EGJ, stomach, and pylorus can all be assessed. This secures the diagnosis and subclassification within hiatal hernia type and allows the surgeon to approximate the size of the hernia sac and width of the crural defect. The distance between the EGJ and hiatus can also be measured, which if >5 cm, serves as a predictor that a esophageal lengthening procedure may be required [13, 14]. The use of fluoroscopy to obtain multiple images over time allows for an assessment of esophageal function. Pooling of a contrast column within the esophagus and a delay in contrast transit through the EGJ indicate a functional obstruction as a result of the hernia. Conversely, reflux of contrast material from the stomach back into the esophagus is indicative of an incompetent EGJ resulting in GER.

Upper endoscopy is mandatory in the preoperative evaluation of patients prior to planned PEH repair. The primary purpose is to rule out a malignancy near the EGJ, which can present with the same obstructive symptoms as PEH. It is also important to check for the presence of esophagitis or gastritis, Barrett esophagus, Cameron ulcers, and/or peptic ulcer disease. It should be noted that upper endoscopy can be extremely challenging in these patients, especially those with type III PEHs, and the risk of esophageal perforation can be increased if not performed by a skilled endoscopist.

Although not universally adopted, we routinely perform an esophageal manometry study on patients being evaluated for PEH. This study is often technically difficult to perform in these patients [15], and it is often easiest to place the manometry catheter during endoscopy. The advance to high-resolution manometry is particularly useful in the setting of PEH, as the catheter does not have to be moved once it is positioned across the EGJ. Despite these challenges, it is useful to assess the peristaltic function of the esophagus preoperatively. Patients with PEH often have abnormal esophageal motility, and these impairments can improve after surgery [16]. However, in patients with complete aperistalsis on preoperative manometry or

those who have weak peristalsis and dysphagia that cannot be explained by the anatomy seen on esophagram, we will tailor our operation to include a partial, rather than complete 360°, fundoplication. Additionally, high-resolution manometry can be used to measure the distance between the EGJ and diaphragmatic hiatus (i.e., distance between high-pressure zone and respiratory inversion point), which can help stratify the risk of requiring an esophageal lengthening procedure.

Although PEH can result in pathologic GER, obtaining a 24-h pH monitoring study does not add any useful information preoperatively. This is because the dissection required to perform an effective repair will likely alter the physiology of the EGJ, and patients with PEH and heartburn (i.e., who are symptomatic) should undergo surgical repair regardless of the findings of pH monitoring.

## Operative Technique

### *Patient Positioning and Setup*

Laparoscopic PEH repair is performed under general anesthesia with endotracheal intubation and full paralysis. Patients are positioned supine with legs abducted. We tuck the right arm and abduct the left arm and use a vacuum beanbag mattress to support the patient's sides and perineum. This positioning provides stability when the table is shifted into a steep reverse Trendelenburg position and helps to prevent neuropathy during what may be a lengthy operation. Pneumatic compression stockings and a urinary catheter are placed, and patients receive appropriate antibiotic prophylaxis prior to the initial incision.

### *Trocar Placement*

Five trocars are utilized: one for the laparoscope, two for the operating surgeon, one for the assistant, and one for a liver retractor (Fig. 13.1). We begin by placing a 10-mm trocar slightly to the left of midline and superior to the umbilicus, approximately 12–15 cm from the xiphoid process. This is typically done using a Veress technique in patients without prior upper abdominal surgery, but an open Hasson technique may be used as well. Once this trocar is inserted and the abdomen insufflated, a 30- or 45° laparoscope is inserted and an initial diagnostic laparoscopy is performed. Use of an angled laparoscope during PEH repair is essential so that unobstructed views can be obtained when working in the confined space of the hiatus and mediastinum.

A 5-mm trocar for the liver retractor is then placed just below the right costal margin, approximately 15 cm from the xiphoid. We use a self-retaining retractor to elevate the left lateral segment of the liver and expose the hiatus. A 5-mm port for

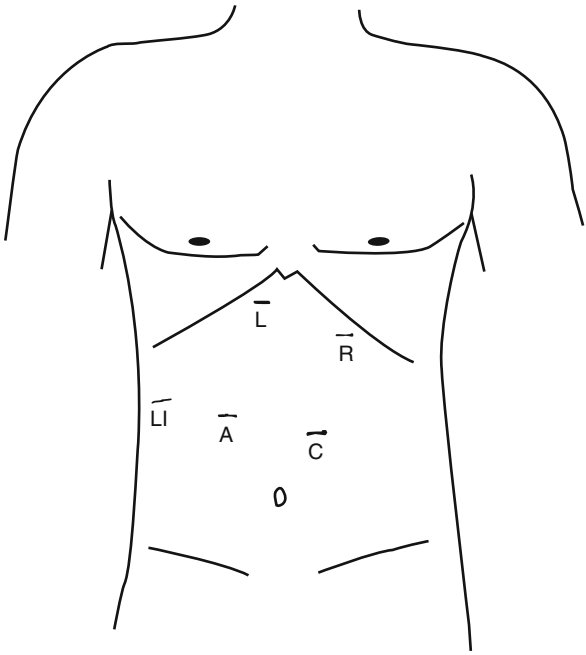


**Fig. 13.1** Trocar positioning for laparoscopic PEH repair: *R* surgeon's right hand instrument, *L* surgeon's left hand instrument, *A* assistant's instrument, *LI* liver retractor, *C* camera port (Adapted from Vaziri and Soper [17])

the assistant's instrument is then placed in the right upper abdomen, approximately midway between the liver retractor and laparoscope ports. A common alternative is to place the assistant's trocar in a lateral position below the left costal margin [18].

The two trocars for the operating surgeon's instruments are then placed. The positioning of these ports is intended to create a triangulation effect, in which the two instruments enter the operative field at a 30–60° angle from either side of the laparoscopic image. The esophagus enters the abdomen through the hiatus at a right-to-left angle, so the surgeon's two working trocars are also arranged "off center" towards the patient's left side. For the surgeon's right hand, a 10-mm trocar (to accommodate a curved needle) is inserted just inferior to the left costal margin, approximately 10 cm from the xiphoid process. We lastly place the surgeon's left hand 5-mm trocar, slightly inferior and to the right of the xiphoid process. Depending on the size and anatomy of the liver, this trocar may need to be placed more inferiorly on the abdominal wall. For this reason, once the liver retractor has been secured, we test potential locations for this trocar by first passing a Veress needle through the abdominal wall to ensure that the working instrument will have a clear path to the hiatus.

Once the trocars have been placed, the patient is tilted to a steep reverse Trendelenburg position in order to shift the abdominal contents inferiorly, away from the hiatus, and to bring the patient's upper abdomen closer to the surgeon, thereby improving ergonomics. This should be done slowly, and in coordination with the anesthesiologist, as this maneuver can significantly reduce venous return. The operating surgeon then moves to a position between the patient's legs with the

laparoscopic monitor placed directly over the head of the patient. The assistant stands to the patient's right and the camera operator is seated in a stool to the patient's left.

## *Dissection and Reduction of the Hernia Sac*

A thorough diagnostic laparoscopy is then performed, focusing on delineating the hernia anatomy. This can be difficult on initial inspection, as a significant portion of the stomach may be lying in the mediastinum. Of importance to note at the onset of the operation are the positions of the pylorus, left gastric artery, spleen, and short gastric vessels, as well as the width of the crural defect.

After initial anatomic identification, an attempt is made to reduce the stomach from the hernia sac and into the abdominal cavity (Fig. 13.2). This helps to facilitate the remainder of the operation by creating additional working space in the mediastinum. A hand-over-hand technique is used to gently pull the stomach inferiorly using atraumatic graspers. However, excessive force should never be applied to the stomach during this initial maneuver. Significant adhesions can exist between the stomach and the hernia sac, and traction under these conditions can result in gastric injury and even perforation. If the stomach does not reduce easily, this step should be abandoned and the operation proceeds with dissection of the hernia sac.

To initiate this dissection, the hepatogastric ligament is divided in order to gain access to the lesser sac and mobilize the lesser curvature of the stomach. In the case of a large type III PEH, a significant portion of the lesser curvature may lie intrathoracically. In operations involving this severe an anatomic distortion, the surgeon

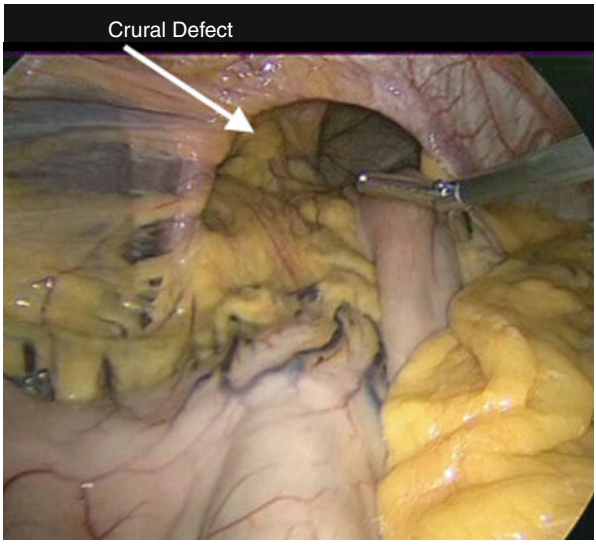


**Fig. 13.2** A large PEH with a significant portion of intrathoracic stomach is seen after liver retractor placement. Gentle traction is applied to reduce as much of the stomach as possible into the abdomen prior to beginning dissection of the hernia sac

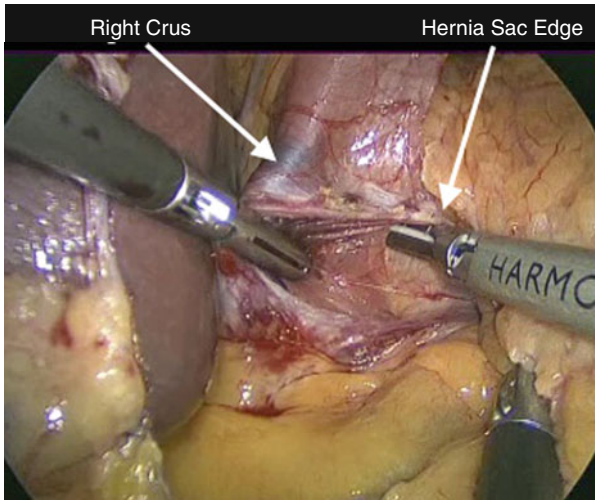


**Fig. 13.3** Dissection of the hernia sac begins at the medial border of the right crus. The hernia sac and sac contents are swept to the right of the laparoscopic image, and the right crus is swept to the left in order to enter the mediastinum on the outside of the sac. The assistant provides retraction inferiorly on the hernia sac

must be extremely careful to identify the location of the left gastric artery, right gastric artery, and even porta hepatis, prior to dividing the hepatogastric ligament, as these structures can be shifted towards the hiatus. The hepatic branch of the vagus nerve, on the other hand, can be divided without physiologic consequence.

Once the lesser sac is entered, division of the lesser omentum continues superiorly to the level of the right crus. We use an ultrasonic dissector to accomplish this, although bipolar or monopolar energy devices can also be employed. The next step is to enter the mediastinum and develop a plane on the outside of the hernia sac. The importance of this maneuver cannot be overemphasized, and the relative ease or difficulty of the remainder of the operation often hinges upon it. To achieve this, the surgeon grasps the right crus with a blunt grasper and then incises the peritoneal layer at its medial aspect (Fig. 13.3). The hernia sac is an extension of this peritoneal membrane and therefore if it is divided at the medial edge of the right crus, the mediastinum can be entered external to the sac. Once this entry is made, blunt dissection is used to sweep the sac and its contents medially and inferiorly, separating them from the rest of the mediastinal structures. The assistant forcibly retracts the hernia sac inferiorly in order to continuously reduce the hernia contents as the dissection proceeds. It should be noted that neither the surgeon nor assistant should grasp the esophagus directly, as it can be injured easily. During this portion of the procedure, the use of cautery should also be limited so as to not inadvertently cause a tear in the hernia sac or thermal injury to the esophagus or vagus nerves.

If the correct plane has been entered, the hernia sac should separate relatively easily, revealing the right-sided mediastinal pleura laterally, pericardium anteriorly, and vertebrae and aorta posteriorly. The anterior and posterior vagus nerves should be identified as well and kept alongside the esophagus. As this mediastinal working space is enlarged, the edge of the hernia sac is sequentially divided at its junction with the crura. This is done in a clockwise direction, starting at the point of mediastinal entry and proceeding towards the left crus. Blunt dissection of the hernia sac

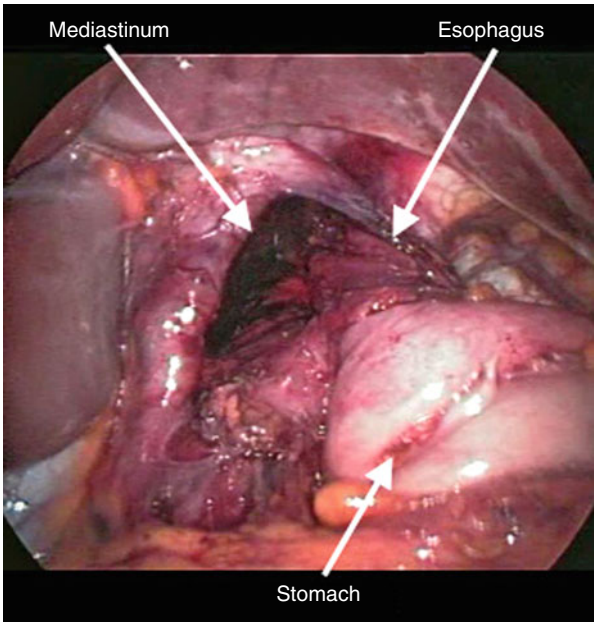


**Fig. 13.4** The anatomy seen after completion of hernia sac dissection and esophageal mobilization. The entire stomach and EGJ lie intra-abdominally, and the esophagus is mobilized off of the crura circumferentially

then proceeds to the patient's left, and the left pleura is exposed. During this step in the operation, tears in the pleura on either side can occur. This usually does not result in adverse physiologic consequences, but the anesthesiologist should be immediately informed. In the case of capnothorax that results in hypotension or increased airway pressures, a reduction in insufflation pressure, or complete deinsufflation of the abdomen, will almost always correct these abnormalities. Insertion of a chest tube is rarely, if ever, required.

Once the dissection reaches the left crus, we next divide the short gastric vessels and gastrosplenic ligament. This mobilization will be required eventually in order to perform the fundoplication, and when performed at this point in the operation, it allows for easier access to the posterior aspect of the hiatus and hernia sac. We prefer to mobilize the entire fundus, starting at the point at which the vessels begin to run perpendicularly to the greater curve (i.e., the short gastric vessels). The assistant retracts the stomach medially, while the surgeon uses his or her left hand to retract the omentum laterally. This aligns the short gastric vessels horizontally in the laparoscopic view. Division with an ultrasonic dissector, or other energy device, then proceeds proximally up the greater curvature until the stomach is separated completely from the left crus and posterior hiatal attachments. The posterior hernia sac, arising from the lesser peritoneal sac, is divided at the base of the crura.

At this point the esophagus should be circumferentially mobilized away from the crura. Blunt dissection of any remaining hernia sac off of the mediastinal structures and into the abdomen continues until the sac is completely freed and reduced (Fig. 13.4). At this point we prefer to excise as much of the hernia sac as possible. This allows for accurate identification of the EGJ and prevents incorporation of

remaining sac tissue into the eventual fundoplication. Care must be taken to identify and trace both vagal trunks prior to sac excision, as there can be dense adhesions between the vagi, sac, and stomach.

## *Esophageal Mobilization and Lengthening*

Once the sac is excised and removed though a trocar, the intra-abdominal length of the esophagus is measured. We prefer to have an esophageal segment of at least 2.5 cm below the diaphragm, with no axial traction exerted, so that a 2-cm-long Nissen fundoplication can be comfortably constructed around it. Failure to achieve this length will predispose to re-herniation of the wrap into the chest, which can cause obstructive symptoms, and may necessitate reoperation. In order to measure this length accurately, we use the distance between the open jaws of an atraumatic grasper (2.5 cm in our instrument set) and, if any question exists, a sterile tape measure. It is critical that no caudad traction is placed on the stomach while obtaining these measurements, as this can falsely lengthen the intra-abdominal distance.

If there is less than 2.5 cm of esophagus below the crura, the mediastinal esophagus is mobilized further cephalad in order to gain additional length. This circumferential dissection can be taken to the level of the inferior pulmonary veins and is successful in achieving the desired intra-abdominal segment in the majority of cases. However, even after meticulous dissection, in 3–14 % [19–21] of cases, the EGJ remains close to or above the crura, resulting in a "short esophagus." Preoperative risk factors that predispose to the occurrence of short esophagus include long-standing GER or reoperation, an EGJ that is greater than 5 cm above the hiatus on esophagram or manometry, or the presence of peptic strictures or Barrett esophagus on endoscopy [14, 22]. However, even when taken in combination, these risk factors do a poor job of predicting which patients will ultimately require esophageal lengthening, and the final diagnosis is always made intraoperatively after a complete esophageal mobilization has been performed.

If a short esophagus still exists after the previously described maneuvers, an esophageal lengthening procedure should be performed so that a completely intra-abdominal fundoplication can be created. We prefer a stapled-wedge gastroplasty technique that creates a length of "neo-esophagus" out of the gastric cardia and lesser curve. This is performed using a standard laparoscopic linear cutting-stapler that is capable of articulation. First, a 40- or 50-French bougie is passed into the stomach along the lesser curve. A marking stitch is placed on the left edge of the bougie at a distance approximately 3 cm inferior to the hiatus, at the point that will become the new "EGJ." The stapler is then used to divide the fundus from the greater curvature to this marked point. The stapler is then articulated to the right and fired alongside the left lateral aspect of the bougie to create a length of neo-esophagus and resect a small wedge of fundus. Other techniques for accomplishing a similar gastroplasty have been described, including introduction of the stapler through a right-sided thoracoscopy port, which eliminates the need to resect a portion of fundus [20].

## *Crural Closure and Options for Mesh Reinforcement*

Once an adequate intra-abdominal esophageal length has been established (via dissection or lengthening procedure), the crura are then closed in order to repair the hernia defect. Interrupted 0 or 2–0 nonabsorbable braided sutures are placed at 1 cm intervals, beginning at the posterior crural junction and working anteriorly (Fig. 13.5). The use of pledgeted sutures has been described [23], but we prefer not to leave any synthetic material in this closure, as it may come in contact with the esophagus. It is important to incorporate intact crural fascia, along with muscle, into these bites so they do not pull through. This relies on meticulous preservation of this fascia throughout the prior hernia sac dissection. Often only posterior sutures are necessary, but if this configuration creates an abnormal anterior angulation at the EGJ, then one or more anterior sutures may be needed.

As the role of synthetic mesh in reinforcing inguinal and ventral hernia repairs became firmly established, their use in hiatal hernia repair gained considerable attention. Several early series, and even randomized controlled trials [24, 25], appeared to indicate that routine reinforcement of hiatal hernia repairs with synthetic mesh resulted in lower recurrence rates when compared with primary closure alone. However, a number of serious, and potentially life-threatening, complications have been described as a result of mesh erosion into the esophagus and even aorta and bronchi [26–29]. For this reason, the use of synthetic mesh for PEH repair has largely been abandoned.

Biologic meshes used in this context have the potential to provide structural support with less theoretical risk for erosion, as they result in a less severe inflammatory response and are eventually incorporated and absorbed. A trial by Oelschlager and colleagues randomized patients undergoing PEH repair to crural reinforcement with a biologic mesh (porcine intestinal submucosa) or primary closure only. While rates of recurrent hiatal hernia at 6 months were lower in the mesh group (9 vs. 23 %)

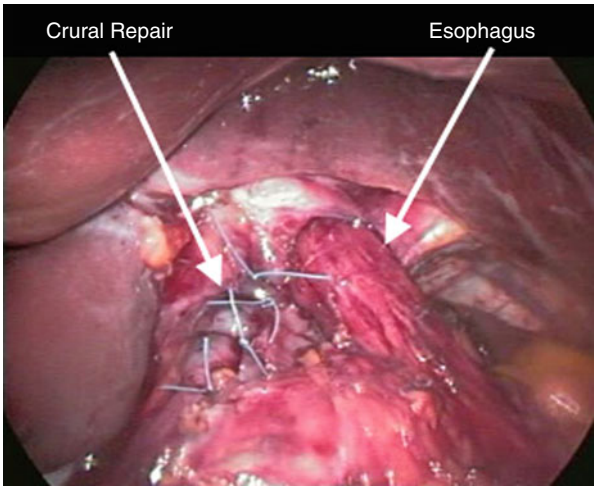


**Fig. 13.5** After completing a posterior crural repair with interrupted sutures, the esophagus has been sufficiently mobilized so that a segment longer than 2.5 cm lies intra-abdominally

[30], this advantage was no longer present at 5-year follow-up (54 vs. 59 %) [31]. However, despite the fact that both groups had high radiologic recurrence rates, they had relatively minor symptoms and improvements in quality of life, and reoperation was rarely needed. Based on these results, there is insufficient evidence currently to support the routine use of biologic mesh during PEH repair.

If there is considerable tension placed on the closure, and a primary repair is therefore not possible, our current approach is to create a "relaxing incision" in the right hemidiaphragm. The diaphragm is incised just lateral to the right crus, to mobilize the crus to the patient's left and allow the two crura to come together without undue tension. We then sew a biologic, or nonbiologic absorbable, mesh patch over the resulting diaphragmatic defect. This provides the advantage of not having any mesh in direct contact with the esophagus, although the long-term results of such a repair have not been established.

## *Fundoplication*

Once the hiatus has been closed, a functional antireflux barrier is constructed. We perform a 360° Nissen fundoplication regardless of the presence of preoperative heartburn or objective evidence of GER (e.g., esophagitis on upper endoscopy). However, we modify this to a partial fundoplication if preoperative manometry shows complete aperistalsis or severely impaired peristalsis that is associated with dysphagia. Other authors have contended that these markers are poor predictors of postoperative function and advocate for use of a complete fundoplication in all cases [16].

To create the fundoplication, the surgeon first passes his or her left hand instrument posterior to the esophagus and grasps the most superior aspect of the fundus along the greater curvature. The instrument is then pulled back behind the esophagus in order to wrap the fundus around the esophagus posteriorly. With the right hand, the surgeon then grasps the anterior fundus that remains to the left of the esophagus and performs a "shoe-shine" maneuver, sliding the fundus back and forth with both hands, in order to check for twists in the wrap and abnormal angulation of the esophagus. It is essential that the wrap be situated entirely around the esophagus, rather than the stomach. This is because a low-lying fundoplication at the level of the gastric body can cause pooling of acidic secretions proximal to the wrap, which can then reflux into the esophagus. Additionally, this anatomy recreates that of a "slipped wrap," which is generally associated with significant dysphagia.

After the fundus is deemed to be in an acceptable location, a 60-French bougie is passed into the gastric body under direct laparoscopic vision. The wrap is secured in place with interrupted seromuscular bites of 2–0 nonabsorbable, braided suture (Fig. 13.6). Typically three sutures are required to create a wrap that is approximately 2 cm in length. We incorporate the most proximal suture into the muscle of the esophageal body in order to prevent wrap slippage. We do not anchor the fundoplication to the crura, although other authors have described doing so to prevent

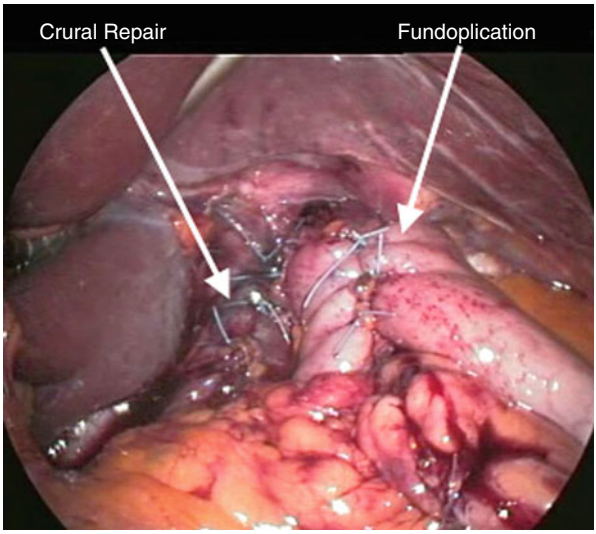


**Fig. 13.6** The final anatomy after completion of crural repair and Nissen fundoplication. The fundoplication is created around intra-abdominal esophagus, rather than the stomach body

wrap migration into the chest [32]. Some surgeons add a gastropexy to the anterior abdominal wall, although we have not found this to be routinely necessary [33].

After completion of the fundoplication, the abdomen is aspirated and checked for hemostasis. If any question exists regarding esophageal or gastric injury, or wrap malformation, an upper endoscopy and insufflation leak test are performed. The liver retractor and trocars are then removed under direct vision. The fascia of trocar sites >5 mm is closed and the skin is closed with absorbable suture.

## Postoperative Care

Patients are typically extubated immediately after surgery and a nasogastric tube is not needed. Patients are started on scheduled antiemetics and intravenous ketorolac, with intravenous narcotics as needed for breakthrough pain. Unless the mediastinal dissection was difficult and required extensive esophageal and gastric manipulation, patients are allowed sips of liquids on the day of surgery and then full liquids the following morning. A routine esophagram is not obtained, unless an esophageal lengthening procedure was performed. If advancing as expected, a soft diet is initiated for lunch and patients are discharged home in the afternoon of the first postoperative day. Retching occurs not infrequently in the early postoperative period and can cause wrap herniation above the crural repair. For this reason, any nausea should be treated aggressively with additional antiemetics, and an esophagram should be performed after any episode of vomiting to check for anatomic disruption. Any significant deviation from the normal postoperative course, such as severe nausea, significant abdominal or chest pain, fever, or tachycardia, should be assumed to be a leak from an esophageal or gastric perforation until proven otherwise. Such

patients should be investigated immediately with an esophagram using water-soluble contrast, with a low threshold for diagnostic laparoscopy if the results are inconclusive.

After hospital discharge, patients are maintained on a soft diet until their first postoperative visit at 2 weeks, and then slowly reintroduce solid foods as tolerated. We typically have patients then return to clinic on a yearly basis and obtain a routine esophagram at 6–12 months postoperatively. Symptoms that are potentially related to either obstruction or GER are first investigated with an esophagram to confirm the anatomy of the repair and fundoplication and then an upper endoscopy. HRM and 24-h pH studies are reserved for patients in whom these tests are nondiagnostic.

## Conclusion

Laparoscopic PEH repair is a complex operation that presents a unique challenge with each case due to the anatomic variation inherent to the disease. A detailed understanding of esophageal physiology and the ability to safely perform a thorough upper endoscopy in the context of distorted anatomy are essential in the preoperative work-up of these patients. Intraoperatively, patience and adaptability are required when formulating strategies to achieve adequate intra-abdominal esophagus length and a durable and functional crural repair. The optimal techniques for accomplishing these aspects of PEH repair have not been conclusively defined, and specifically, further research is required to determine if, and when, cruroplasty with biologic mesh is most effective.

## References

1. Curci JA, Melman LM, Thompson RW, et al. Elastic fiber depletion in the supporting ligaments of the gastroesophageal junction: a structural basis for the development of hiatal hernia. J Am Coll Surg. 2008;207(2):191–6.
2. Stylopoulos N, Rattner DW. The history of hiatal hernia surgery: from Bowditch to laparoscopy. Ann Surg. 2005;241(1):185–93.
3. Zehetner J, Demeester SR, Ayazi S, et al. Laparoscopic versus open repair of paraesophageal hernia: the second decade. J Am Coll Surg. 2011;212(5):813–20.
4. Nguyen NT, Christie C, Masoomi H, et al. Utilization and outcomes of laparoscopic versus open paraesophageal hernia repair. Am Surg. 2011;77(10):1353–7.
5. Draaisma WA, Gooszen HG, Tournoij E, et al. Controversies in paraesophageal hernia repair: a review of literature. Surg Endosc. 2005;19(10):1300–8.
6. Pandolfino JE, Shi G, Trueworthy B, et al. Esophagogastric junction opening during relaxation distinguishes nonhernia reflux patients, hernia patients, and normal subjects. Gastroenterology. 2003;125(4):1018–24.
7. Cameron AJ, Higgins JA. A lesion associated with large diaphragmatic hernia and chronic blood loss anemia. Gastroenterology. 1986;91(2):338–42.
8. Moskovitz M, Fadden R, Min T, et al. Large hiatal hernias, anemia, and linear gastric erosion: studies of etiology and medical therapy. Am J Gastroenterol. 1992;87(5):622–6.

9. Low DE, Simchuk EJ. Effect of paraesophageal hernia repair on pulmonary function. Ann Thorac Surg. 2002;74(2):333–7; discussion 337.
10. Hill LD. Incarcerated paraesophageal hernia. A surgical emergency. Am J Surg. 1973;126(2):286–91.
11. 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(1):33–54.
12. Stylopoulos N, Gazelle GS, Rattner DW. Paraesophageal hernias: operation or observation? Ann Surg. 2002;236(4):492–500; discussion 500–491.
13. Gastal OL, Hagen JA, Peters JH, et al. Short esophagus: analysis of predictors and clinical implications. Arch Surg. 1999;134(6):633–6; discussion 637–638.
14. Mittal SK, Awad ZT, Tasset M, et al. The preoperative predictability of the short esophagus in patients with stricture or paraesophageal hernia. Surg Endosc. 2000;14(5):464–8.
15. Roman S, Kahrilas PJ, Kia L, et al. Effects of large hiatal hernias on esophageal peristalsis. Arch Surg. 2012;147(4):352–7.
16. Swanstrom LL, Jobe BA, Kinzie LR, et al. Esophageal motility and outcomes following laparoscopic paraesophageal hernia repair and fundoplication. Am J Surg. 1999;177(5):359–63.
17. Vaziri K, Soper NJ. Laparoscopic Heller myotomy: technical aspects and operative pitfalls. J Gastrointest Surg. 2008;12(9):1586–91.
18. Nason KS, Luketich JD, Witteman BP, et al. The laparoscopic approach to paraesophageal hernia repair. J Gastrointest Surg. 2012;16(2):417–26.
19. Terry ML, Vernon A, Hunter JG. Stapled-wedge Collis gastroplasty for the shortened esophagus. Am J Surg. 2004;188(2):195–9.
20. Swanstrom LL, Marcus DR, Galloway GQ. Laparoscopic Collis gastroplasty is the treatment of choice for the shortened esophagus. Am J Surg. 1996;171(5):477–81.
21. Mattioli S, Lugaresi ML, Costantini M, et al. The short esophagus: intraoperative assessment of esophageal length. J Thorac Cardiovasc Surg. 2008;136(4):834–41.
22. Yano F, Stadlhuber RJ, Tsuboi K, et al. Preoperative predictability of the short esophagus: endoscopic criteria. Surg Endosc. 2009;23(6):1308–12.
23. Antonoff MB, D'Cunha J, Andrade RS, et al. Giant paraesophageal hernia repair: technical pearls. J Thorac Cardiovasc Surg. 2012;144(3):S67–70.
24. 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(6):649–52.
25. 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(1):40–8.
26. Hazebroek EJ, Leibman S, Smith GS. Erosion of a composite PTFE/ePTFE mesh after hiatal hernia repair. Surg Laparosc Endosc Percutan Tech. 2009;19(2):175–7.
27. Stadlhuber RJ, Sherif AE, Mittal SK, et al. Mesh complications after prosthetic reinforcement of hiatal closure: a 28-case series. Surg Endosc. 2009;23(6):1219–26.
28. Arroyo Q, Arguelles-Arias F, Jimenez-Saenz M, et al. Dysphagia caused by migrated mesh after paraesophageal hernia repair. Endoscopy. 2011;43(Suppl 2 UCTN):E257–8.
29. Zugel N, Lang RA, Kox M, et al. Severe complication of laparoscopic mesh hiatoplasty for paraesophageal hernia. Surg Endosc. 2009;23(11):2563–7.
30. 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(4):481–90.
31. Oelschlager BK, Pellegrini CA, Hunter JG, et al. Biologic prosthesis to prevent recurrence after laparoscopic paraesophageal hernia repair: long-term follow-up from a multicenter, prospective, randomized trial. J Am Coll Surg. 2011;213(4):461–8.
32. Auyang ED, Pellegrini CA. How I do it: laparoscopic paraesophageal hernia repair. J Gastrointest Surg. 2012;16(7):1406–11.
33. Ponsky J, Rosen M, Fanning A, et al. Anterior gastropexy may reduce the recurrence rate after laparoscopic paraesophageal hernia repair. Surg Endosc. 2003;17(7):1036–41.