Paraesophageal Herniation

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Typically, a general surgeon sees only the occasional patient who has a paraesophageal hernia secondary, in part, to a low incidence of this entity. Some patients who have paraesophageal hernia are managed without surgical referral, however, because the potential danger of this diagnosis may be underappreciated by the primary physician. Additionally, there may be a hesitancy toward surgical referral because paraesophageal hernia tends to occur in older and potentially more frail patients. The risk of laparotomy or thoracotomy in this patient group may be judged, correctly or not, to exceed any potential benefit to herniorrhaphy. Since 1995, there has been an increasing number of reports of minimally invasive paraesophageal hernia repair. The feasibility of this approach may make surgical referral of the patient who has paraesophageal hernia a more attractive choice for the primary physician. In this chapter, we present our technique for minimally invasive paraesophageal hernia repair, which includes an optional prosthetic reinforcement of the diaphragmatic suture line.

Definition, Classification, and Incidence

A paraesophageal hernia is an enlargement of the esophageal hiatus of the diaphragm, through which the stomach and possibly other intraabdominal organs migrate into the left chest. Paraesophageal hernia is a form of hiatus hernia; the classification of hiatus hernia subtypes is shown in Figure 1. Although this classification system may not be entirely clinically relevant (e.g., hernia types II, III, and IV require similar management), it is still useful for the purpose of describing anatomic relationships. Normal anatomy (no hernia) is shown in Figure 1A, with the stomach intraabdominal and the gastroesophageal junction below the diaphragm. A type I hiatus hernia (also known as a sliding hiatal hernia) is illustrated in Figure 1B; the gastroesophageal junction has risen above the diaphragmatic hiatus, and the gastric wall forms the posterior portion of the hernia sac. This is the most common subtype of hiatus hernia, accounting for more than 90% of hernias of the esophageal hiatus. The true incidence in the general population is difficult to know. The presence of a sliding hiatus hernia has been demonstrated in approximately 5% of patients undergoing radiologic evaluation; such a demonstration, however, is often dependent on how vigorously it is pursued by the radiologist. Type I hiatus hernia is commonly associated with reflux, and is not associated with a risk of gastric ischemia or strangulation, or both; the management of this type of hiatus hernia and the associated gastroesophageal reflux is addressed in this chapter.

A type I (sliding hiatal) hernia is not classified as a paraesophageal hernia; hiatus hernia types II, III, and IV, however, are considered paraesophageal. As the prefix para suggests, these hernias involve abnormal migration of an organ alongside the esophagus. A type II (also known as a pure paraesophageal) hiatus hernia is shown in Figure 1C. In this type, the gastroesophageal junction is tethered by the phrenoesophageal ligament and remains below the diaphragm; a portion of the stomach, most commonly the fundus, herniates through the hiatus alongside the esophagus into the left chest. A true type II hiatus hernia is rare. The more commonly found (but still probably less than 5% of all hiatus hernias) paraesophageal hernia is the type III, or mixed hernia (Fig. 1D). In this entity, the gastroesophageal junction herniates into the chest (i.e., goes above the diaphragm) along with a paraesophageal component of the gastric corpus. A type III hiatus hernia, therefore, may be considered a combination of types I and II. A type IV (massive) hernia (Fig. 1E) is present when another intraabdominal organ (e.g., the spleen, colon, or small bowel) herniates into the chest along with the stomach.

Etiology and Pathophysiology

The immediate cause of a paraesophageal hernia is an enlarged esophageal hiatus of the diaphragm. The muscular structure that forms the margin of the esophageal hiatus is, in approximately 50% of subjects, the right and left bundles of the right diaphragmatic crus (Fig. 2). The remaining subjects may have a minor contribution to the hiatal musculature from the left crus, but the right crus is nearly always the dominant component. Whether one or another of these crucial arrangements increases the risk for hiatus hernia is not known. Overall, the causes for an enlarged hiatus are unclear. Factors such as chronic increases in intraabdominal pressure, obesity, lax or inadequate tissue, and congenital anatomic or metabolic defects have been considered. Some paraesophageal hernias may be iatrogenic, as after Nissen fundoplication with...
cavity—a condition known as *gastric volvulus* or *upside-down stomach*. There are two main types of gastric volvulus: organoaxial (Fig. 3A) and mesentericococaxial (Fig. 3B). In the former and more common type, the stomach twists about an axis that runs from the gastroesophageal junction to the pylorus. In the latter type, the volvulus axis is roughly in line with the right and left gastric arteries, and the antrum flips up anteriorly. Both types of volvulus can produce gastric ischemia, which can result in chest pain, upper gastrointestinal hemorrhage, and strangulation.

Symptomatic gastroesophageal reflux variably is present in patients who have paraesophageal hernia. It might be presumed that the patient who has a type II hernia (gastroesophageal junction located intraabdominally; Fig. 1C) should have an intact antireflux mechanism and therefore not have heartburn, yet some type II patients do experience reflux. Conversely, the patient who has a type III hernia (gastroesophageal junction located in the chest; Fig. 1D) might be presumed to have a dysfunctional lower esophageal sphincter and therefore experience heartburn, yet many type III patients do not have reflux symptoms. These circumstances outline a controversy over whether an antireflux procedure should be performed routinely or selectively (for symptoms) during the paraesophageal hernia repair. Our tendency is to perform a concomitant antireflux procedure routinely (see Nissen Fundoplication section later in this chap-

Most patients who have paraesophageal hernia have symptoms, most commonly chest pain, vomiting, dysphagia, heartburn, and weight loss. Anemia and occult fecal blood may be present. An occasional patient may have severe erosive esophagitis. Gastric strangulation with perforation into the chest, although uncommon, usually makes its presence known with rapidly evolving and frequently fatal sepsis.

A major reason why paraesophageal hernia should not be managed nonoperatively is, as alluded to previously, the risk of gastric volvulus and strangulation. If, for example, the gastric body and antrum herniate along with the fundus, then the entire stomach can flip inverted into the thoracic cavity.

![Fig. 1. Types of hiatus hernia. A: Normal anatomy. B: Type I, or sliding, hernia. C: Type II, or pure paraesophageal, hernia. D: Type III, or mixed paraesophageal, hernia. E: Type IV, or massive paraesophageal, hernia. (From Duranceau A, Janieson GC. Hiatal hernia and gastroesophageal reflux. In: Sabiston DC Jr, Lyerly HK, eds. Textbook of surgery: the biological basis of modern surgical practice, 15th ed. Philadelphia: WB Saunders, 1997:775, with permission.)](image1)

![Fig. 2. Anatomy of the esophageal hiatus that is present in approximately 50% of subjects. (From Gray SW, Rove JE Jr, Skandalakis JE. Surgical anatomy of the gastroesophageal junction. Am Surg 1979;45:575-587, with permission.)](image2)
ter), but we acknowledge that selective performance also is acceptable.

**Operative Indications**

The presence of a paraesophageal hernia in a patient who can tolerate a general anesthetic is indication for repair of the hernia. Contraindications are relative and include limited life span or severe cardiopulmonary disease. In the 1960s, asymptomatic or minimally symptomatic patients who had paraesophageal hernia were managed expectantly. As the natural history of this entity was documented, however, it was noted that a minority of patients were dying from gastric strangulation that occurred unpredictably. Operative management recommendations began to appear. Generally, the current consensus is that non-operative management is inappropriate and that any paraesophageal hernia should be repaired in a patient who has reasonable operative risk. Some argue that observation is still safe, but the preponderance of data does not concur with this.

A minimally invasive approach to elective paraesophageal hernia repair is presented in the following paragraphs. If a patient has signs of strangulation, perforation, or both, however, then an open approach probably is most prudent. Gastric volvulus with strangulation may require an emergency subtotal gastrectomy. The minimally invasive approach is unproven in this circumstance.

**Preoperative Evaluation**

The history should document the presence or absence of reflux symptoms, pain, hematemesis, dysphagia, stool changes, and previous upper gastrointestinal surgery or endoscopy. Physical signs of a paraesophageal hernia in the nonseptic patient usually are not evident. Most paraesophageal hernias can be identified with a chest roentgenogram. Subsequent evaluation should include a barium esophagogram to delineate the hernia type and look for gross esophageal motility disturbance or shortened esophagus. If there is evidence of esophageal dysmotility by history, barium esophagogram, or both, then it can be evaluated with esophageal manometry. The presence of esophageal dysmotility usually leads to the performance of a partial fundoplication or no fundoplication at all. The presence of a shortened esophagus, which makes reduction of the stomach into the abdomen difficult, is an indication for an esophageal lengthening procedure, which is beyond the scope of this chapter.

We routinely obtain an esophagostroduodenoscopy to evaluate for esophagitis, stricture, and dysplasia. In the patient who has Barrett’s epithelium with no to moderate dysplasia, we perform a fundoplication. If severe dysplasia (which can be considered carcinoma in situ) is present, consideration must be given for esophagectomy. If the diagnosis of gastroesophageal reflux disease is in question, then ambulatory pH monitoring can be performed.

A thorough cardiopulmonary evaluation should be completed, with emphasis on the history and physical examination. Objective tests of cardiac function (e.g., stress echocardiography) may be more pertinent and less invasive for defining surgical risk than tests of anatomy (e.g., coronary angiography).

**Surgical Technique**

**Patient Positioning**

If mesh implantation is probable, then cefazolin (2 g intravenously) is administered with induction of anesthesia. The abdomen is shaved after induction of anesthesia. Sequential compression devices are placed on the thighs and legs. The patient is placed in a modified lithotomy position (supine, thighs flexed 45 degrees and abducted 45 degrees, legs flexed 30 degrees, calves supported by cushioned led holders), so that the buttocks are at the end of the operating table. A buttress at the end of the bed against the buttocks is helpful to prevent the patient from sliding down when the head of the bed is raised. The left arm is tucked against the patient’s side, and the right arm is abducted 90 degrees on an armboard for anesthesia access. The abdomen is prepared with a chlorhexidine solution and draped.
the progress of the cutting tip through the layers of the abdominal wall is displayed on the monitor. The monitor provides visual feedback to the surgeon, and manual pressure on the trocar can be adjusted continuously to avoid visceral injury.

Once intraperitoneal placement of the 12-mm optical trocar is evident on the monitor, the laparoscope is withdrawn and the carbon dioxide is connected to the trocar sleeve. A 15-mm Hg pneumoperitoneum is established, the 0-degree laparoscope is reinserted, and inspection of the peritoneal cavity is performed. Before placement of each of the other four trocars, the abdominal wall is transilluminated with the intraabdominal laparoscope to identify and avoid abdominal wall vessels at the port sites. Subsequent entrance of each trocar into the peritoneal cavity is observed with the laparoscope. Four 10- to 11-mm trocars are inserted through 1-cm transverse skin incisions at sites 1 (supraumbilical in the midline), 2 (subxiphoid), 3 (right subcostal in the midclavicular line), and 5 (left subcostal in the anterior axillary line). After all the trocars are in position, the laparoscope is transferred to port 1 (supraumbilical), where it remains for the rest of the procedure. If the patient has a long torso, or if extensive thoracic dissection is anticipated, it is advisable to locate port 1 more superiority, such as at the midpoint between the xiphoid process and the umbilicus.

**Surgeon Positioning and Port Placement**

The surgeon stands between the patient’s legs. The first assistant stands on the patient’s left side, the camera person stands on the patient’s right side, and the operating room technician or scrub nurse stands on the dominant-handed side of the surgeon. Five trocars are placed (Fig. 4). A 12-mm optical trocar with a plastic cutting tip (Endopath, Ethicon Endo-Surgery, Cincinnati, OH) is inserted through a 1-cm transverse skin incision at site 4 (Fig. 4), which is just below the left costal margin in the midclavicular line. The laparoscope is inserted into the transparent trocar, and

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**Fig. 5.** Exposure of the enlarged gastroesophageal hiatus using the balloon retractor.
Exposure of the Esophageal Hiatus

The head of the operating table is tilted 15 to 20 degrees upward so that the viscera fall away from the hiatus. An inflatable balloon retractor (Soft-Wand, Circon Cabot, Santa Barbara, CA) is inserted into port 2 (subxiphoid), and the left lobe of the liver is retracted superomedially (Fig. 5). This maneuver exposes the enlarged gastroesophageal hiatus, providing a view into the thorax. The balloon retractor is kept in this position for the duration of the procedure.

Reduction of the Hernia

The surgeon operates primarily through ports 3 and 4; the first assistant uses ports 2 and 5. Nontraumatic tissue graspers with plastic inserts (Atraugrip, Pilling Weck Surgical, Research Triangle Park, NC; hereafter referred to as graspers) are inserted through ports 3 and 4. The herniated viscera are grasped just below the esophageal hiatus, and gentle traction is applied (Fig. 6). In the majority of cases, the viscera and the sac reduce into the abdomen without much difficulty. Occasionally, though, the viscera and hernia sac have formed extensive adhesions in the chest, particularly to the pleura. This sets up a potentially hazardous situation, in that a pleural tear during the dissection provides a communication between the pleural cavity and the pneumoperitoneum, which can result in a tension pneumothorax.

If confronted with a stomach and a hernia sac that are stuck in the mediastinum, the surgeon should use meticulous sharp dissection to mobilize these structures, starting with the viscera. Such a dissection usually proceeds slowly. Exposure is maintained with graspers through ports 3 and 5, and hook cautery or scissors is applied through port 4. Dissection generally is taken from anterior to posterior, developing a plane between the stomach and sac. The hernia sac should be anterior and the esophagus posterior. In one sense, it is advantageous to approach such a case laparoscopically, because of the scope’s ability to enter the mediastinum and provide a magnified view. At times, it may be beneficial to use the 30-degree laparoscope to improve the operative view. As the dissection progresses, the location of the gastroesophageal junction can be identified with the aid of a fiberoptically lit bougie passed into the esophagus by the anesthesiologist. After the viscera have been reduced, the sac needs to be dissected out of the mediastinum. To facilitate this, the sac may be entered anteriorly so that a plane can be developed sharply between the sac and the pleura. Once the sac has been mobilized into the abdomen, it is excised and removed from the abdomen. If not excised, the sac generally is a nuisance and obstructs view during the rest of the operation.

Dissection of the Crus

With the viscera reduced and the hernia sac excised, preparation for the hernia repair is performed. The gastrohepatic ligament is grasped through port 3 and the gastric corpus grasped through port 5, placing the ligament on stretch. This is incised with hook cautery (through port 4) up to the gastroesophageal junction (Fig. 7), taking care to clip the occasional accessory artery that can be within this ligament. The potential space posterior to the gastroesophageal junction is developed bluntly using the graspers (jaws closed) and an palpation probe. This dissection is aided by gently elevating the esophagus with a closed grasper (through port 3) placed posterior to the gastroesophageal junction (Fig. 8). This is a key maneuver, providing exposure for the crural dissection and subsequent hernia repair. To get optimal elevation of the esophagus, the lighted bougie should be backed out of stomach into the proximal esophagus. The
posterior (right) vagus should be identified and elevated with the esophagus. The right and left bundles of the right crus posterior to the esophagus then are delineated with blunt dissection. It is possible at this time that a window of dissection may open posterior to the stomach, such that the tip of the spleen may be visible if the surgeon views posterior to the stomach from the lesser curvature (Fig. 8). This window is essential for mesh placement and Nissen fundoplication. If not made here, it is made with the next step.

**Mobilization of the Fundus**

A mobilized fundus is important to improve the ease of polytetrafluoroethylene placement and to facilitate the construction of a loose Nissen wrap. The gastroepiploic ligament is grasped at its inferior portion through port 5, and the gastric corpus is grasped through port 3. Gentle stretch is placed on the ligament between the two graspers, and the ligament is incised with the hook cautery (port 4) at an avascular point until the lesser sac is entered. The 5- or 10-mm ultrasonic shears (Harmonic Scalpel, Ethicon Endo-Surgery) then is inserted through port 4, and the gastroepiploic ligament (containing the short gastric vessels) is transected, proceeding from the ligament incision site superiorly (Fig. 9). As the superior pole of the spleen is approached, it usually is helpful to switch to the 30-degree laparoscope and place the patient temporarily in a steeper head-up position. Dissection of the splenic pole can be the most difficult part of the procedure, so it is advisable to be extra cautious here. After the gastroepiploic ligament has been taken down, the fundus should have ample medial mobility.

The exposure illustrated in Figure 8 is obtained again (i.e., port 5 grasper, posterior to the gastroesophageal junction, anterior retraction), and the retrogastric dissection is connected with the fundal dissection. The 30-degree laparoscope usually provides a better view for this maneuver. This action completes the “window” that was described in the previous paragraph. The gastroesophageal junction now should be free of attachments.

**Cruroplasty**

Before repairing the hiatal hernia, the surgeon should ensure that there is adequate length of tension-free intraabdominal esophagus (in general, 3 to 4 cm of esophagus below the diaphragm). If the distal esophagus needs to be pulled down into the abdomen under tension, the integrity of the hernia repair is compromised. If the patient had the preoperative workup that made the intraoperative finding of shortened esophagus unlikely, then transhiatal mobilization of the esophagus should yield an adequate length of intraabdominal esophagus.

Maintaining the exposure of Figure 8, the right and left bundles of the right crus (Fig. 2) are cleaned sufficiently so that a cruroplasty can be performed. Before the crural repair, a No. 50 French dilator is passed orally and well into the stomach. The right and left bundles are approximated with interrupted sutures of 2-0 polyester (Fig. 10). Our preference is to use an Endo Stitch suturing device (U.S. Surgical Corp., Norwalk, CT). Large bites (1 cm or larger) of each crural bundle, including the peritoneum, should be taken; the suture interval should be 7 to 8 mm. The surgeon should remain aware of the close medial location of the aorta during the crural repair. After completion of the cruroplasty, a 10-mm grasper should be able to pass into the hiatus alongside the esophagus with the dilator in place.

**Mesh Reinforcement of Cruroplasty**

It should be emphasized that mesh reinforcement is optional. We have chosen empirically a hiatal defect of 8 cm or larger as our threshold for mesh placement. We have used a laparoscopic hernia patch spreader (now discontinued) to measure the defect intraoperatively.

Fig. 9. Transection of the gastroepiploic ligament with the ultrasonic shears.
A 15 x 10 x 0.1-cm fenestrated sheet of polytetrafluoroethylene (Gore-Tex, W. L. Gore and Associates, Flagstaff, AZ) is brought on the field, and a radial slot with a 3-cm central circular defect (keyhole) is cut into the sheet. The superior edge of the mesh is marked with the placement of a stitch to aid with orientation once the mesh is intraabdominal. The mesh then is pushed through the 12-mm port (site 4) into the abdomen. The mesh is applied to the undersurface of the diaphragm, with the esophagus passing through the keyhole and the keyhole slot positioned anteriorly. The mesh then is stapled to the diaphragm (No. 50 French dilator in position) with a straight hernia stapler, as shown in Figure 11. Staples are placed around the mesh perimeter, the two leaves of the keyhole are stapled together, and a circle of staples is placed around the keyhole defect, taking care to avoid the esophagus. During staple placement, the surgeon should remain aware of the heart beating against the other side of the diaphragm.

**Nissen Fundoplication**

We believe that our dissection around the gastroesophageal junction destroys the function of the phrenoesophageal liga-
ment and weakens the natural antireflux valve. To compensate for this, we perform a short, loose 360-degree wrap (floppy Nissen fundoplication) around the esoph-

gus, except in patients who have esophageal dysmotility. Dysphagia and bloating have not been long-term postoperative problems.

The dilator is withdrawn into the proximal esophagus to facilitate the fundal wrapping. A grasper (port 5) picks up the superior fundus, and another grasper (port 3) is passed posterior to the esophagus from medial to lateral. The first grasper passes the fundus to the posterior grasper. The latter grasper pulls the fundus medial and posterior to the esophagus. The port 5 grasper then grabs the

lateral fundus, taking care not to grab lower down on the greater curve (i.e., the gastric body). The No. 50 French dilator is readvanced into the stomach. The two graspers pull the fundal folds together on the anterior surface of the esophagus. There should be no tension on this wrap; if there is, the surgeon should reposition the graspers. The fundal folds are sutured together with three interrupted stitches of 2-0 polyester; the proximal stitch takes a bite of the prosthetic to anchor the wrap. The wrap length should be approximately 2 cm. The completed wrap (Fig. 12) should be able to accommodate a 10-mm grasper alongside the esophagus with the No. 50 French dilator in place.

**Closure, Postoperative Care, and Follow-Up**

The pneumoperitoneum is evacuated. Port sites are closed with the aid of a fascial closing device (Arrow Medical, Libertyville, IL); the skin of all sites is closed with intracuticular 5-0 polyglactin suture and adhesive strips. If there is a chance that the pleural cavity was entered, then a chest roentgenogram is obtained in the recovery room. If the procedure was performed in the morning, the patient is allowed liquids in the evening. A modified diet (no bread, meat, or gas-producing foods or beverages) is started the following day. The patient generally stays in the hospital for 1 to 2 days. Follow-up visits in clinic are scheduled for 1 and 2 weeks, 1 and 3 months, and then yearly.

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**Fig. 10. Suture closure of the esophageal hiatus.**

**Fig. 11. Polytetrafluoroethylene reinforcement of the crural repair.**

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for 5 years. If there is recurrence of symptoms, then an esophagogastroduodenoscopy and barium esophagram are obtained.

Suggested Reading


EDITOR'S COMMENT

The minimally invasive approach to paraesophageal hernias may, as the authors claim, increase the referral of these patients, many of whom are elderly and frail. However, it is precisely this type of patient that should be repaired, because incarceration and strangulation are fairly common. Indeed, in one study, 22 of 28 patients were dead within two years, often of the complication of strangulation and incarceration after diagnosis. The question of whether a type II hiatus diaphragmatic hernia exists has been examined by some authorities. To be sure, there may be certain individuals who do not have a displaced phrenoesophageal membrane and whose gastroesophageal junction therefore remains within the abdomen. Others have claimed that type II hernias have a sliver of normal diaphragm between the esophagus and the herniated stomach. Most individuals who deal with this type of surgery have never seen that sliver of normal diaphragm; in my opinion, then, the existence of a type II hernia is questionable.

Technically, the authors are correct in categorizing a previous laparoscopic Nissen as an antecedent cause of the paraesophageal hernia, especially in the case of closing the two sides of the right crus under tension. As originally proposed by Dr. Frantzides, the addition of a tension-free hernioplasty as a reinforcement to a 360-degree laparoscopic fundoplication was recently utilized by Basso and colleagues (Surg Endosc 14:164, 2000). The addition of polypropylene mesh (3 cm × 4 cm) was placed on the hiatus behind the esophagus in their hands and helped what they believe to be cutting through of the crural sutures that are placed under tension with inspiration.

Horgan and co-workers (Am J Surg 177:354, 1999) stressed the importance of resecting the entire hernia sac, despite its dangers. They point out that in doing so, the esophagus is at risk and the anterior vagus may inadvertently be severed. Edye and colleagues (Ann Surg 228:528, 1998) have shown that the recurrence rate is 20% when the sac is not removed, and 0% when resection of the sac has been performed.

Swanson and colleagues (Am J Surg 177:359, 1999) reviewed 52 patients with a mean age of 63 who had sometimes large paraesophageal hernias. Follow-up consisted of 24-hour pH tests, symptom assessment form, and manometry. Of the 52 patients, 24-hour pH tests were abnormal in four patients, of which two were symptomatic. Lower esophageal pressures increased in 63% of patients and functioned well in 71% of patients. Of note is that 50% of preoperative motility disorders improved.

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