The development of laparoscopic surgery over the past 20 years has caused a significant shift in the treatment of benign esophageal diseases.

In the first part of the 1990s, it became clear that treatment of benign esophageal disorders with minimally invasive procedures yielded results comparable to those of treatment with traditional operations while causing minimal postoperative discomfort, reducing the duration of hospitalization, shortening recovery time, and permitting earlier return to work. Consequently, minimally invasive surgery was increasingly considered as first-line treatment for achalasia, and laparoscopic fundoplication was considered more readily and at an earlier stage in the management of gastroesophageal reflux disease (GERD).

Since then, minimally invasive esophageal procedures have continued to evolve, thanks to better instrumentation and improved surgical expertise. In addition, with greater experience and longer follow-up, it has become possible to analyze techniques and their results more rigorously. For instance, whereas a few years ago a left thoracoscopic Heller myotomy was considered the procedure of choice for achalasia, and laparoscopic fundoplication was considered more readily and at an earlier stage in the management of gastroesophageal reflux disease (GERD).

In this chapter, we focus on minimally invasive approaches to the treatment of GERD and esophageal motility disorders. The standard open counterparts of these operations are described elsewhere [see 4.7 Open Esophageal Procedures].

**Laparoscopic Nissen Fundoplication**

**PREOPERATIVE EVALUATION**

All patients who are candidates for a laparoscopic fundoplication should undergo a preoperative evaluation that includes the following: (1) symptomatic evaluation, (2) an upper gastrointestinal (GI) series, (3) endoscopy, (4) esophageal manometry, and (5) ambulatory pH monitoring.

**Symptomatic Evaluation**

The presence of both typical symptoms (heartburn, regurgitation, and dysphagia) and atypical symptoms of GERD (cough, asthma, chest pain, dental erosions, and hoarseness) should be investigated, and symptoms should be graded with respect to their intensity both before and after the operation. Nonetheless, a diagnosis of GERD should never be based solely on symptomatic evaluation. Some assert that the diagnosis can be made reliably from the clinical history, so that a complaint of heartburn should lead to the presumption that acid reflux is present; however, testing of this diagnostic strategy demonstrates that symptoms are far less sensitive and specific than is usually believed. For instance, a study from the University of California, San Francisco (UCSF), found that of 822 consecutive patients referred for esophageal function tests with a clinical diagnosis of GERD (based on symptoms and endoscopic findings), only 70% had abnormal reflux on pH monitoring. Heartburn and regurgitation were no more frequent in patients who had genuine reflux than in those who did not; thus, symptomatic evaluation, by itself, could not distinguish between the two groups.

The response to proton pump inhibitors (PPIs) is a better predictor of abnormal reflux. For example, in the UCSF study just cited, 75% of patients with GERD reported a good or excellent response to PPIs, compared with only 26% of patients without GERD. Similarly, a study involving multivariate analysis of factors predicting outcome after laparoscopic fundoplication identified a clinical response to acid suppression therapy as one of three factors predictive of a successful outcome, the other two being an abnormal 24-hour pH score and the presence of a typical primary symptom (e.g., heartburn). In addition, a 2007 report found that patients with a body mass index (BMI) of 35 kg/m^2 or greater experienced higher failure rates after laparoscopic Nissen fundoplication. These data support the theory that GERD in morbidly obese patients has a different pathophysiology and may warrant a different therapeutic approach (e.g., laparoscopic Roux-en-Y gastric bypass [see 5.7 Surgical Treatment of Morbid Obesity]).

**Upper Gastrointestinal Series**

An upper GI series is useful for diagnosing and characterizing an existing hiatal hernia. The size of the hiatal hernia helps predict how difficult it will be to reduce the esophagogastric junction below the diaphragm. In addition, large hiatal hernias are associated with more severe disturbances of esophageal peristalsis and esophageal acid clearance. Esophagograms are also useful for determining the location, shape, and size of a stricture and for suggesting the presence of a short esophagus.

**Endoscopy**

Endoscopy is typically the first test performed to confirm a symptom-based diagnosis of GERD. This approach has two...
pitfalls. First, even though the goal of endoscopy is to assess the mucosal damage caused by reflux, mucosal changes are absent in about 50% of GERD patients.11 Second, major interobserver variations have been reported with esophageal endoscopy, particularly for low-grade esophagitis.16 In one study, for instance, 60 (24%) of 247 patients with negative results on pH monitoring had been diagnosed as having grade I or II esophagitis.11 Endoscopy is also valuable for excluding gastric and duodenal pathologic conditions and detecting the presence of Barrett’s esophagus.

**Esophageal Manometry**

Esophageal manometry provides useful information about the motor function of the esophagus by determining the length and resting pressure of the lower esophageal sphincter (LES) and assessing the quality (i.e., the amplitude and propagation) of esophageal peristalsis. In addition, it allows proper placement of the pH probe for ambulatory pH monitoring (5 cm above the upper border of the LES).

**Ambulatory pH Monitoring**

Ambulatory pH monitoring is the most reliable test for the diagnosis of GERD, with a sensitivity and specificity of about 92%.15 It is of key importance in the workup for the following four reasons.

1. It determines whether abnormal reflux is present. In the UCSF study mentioned earlier,11 pH monitoring yielded normal results in 30% of patients with a clinical diagnosis of GERD, thereby obviating the continuation of inappropriate and expensive drugs (e.g., PPIs) or the performance of a fundoplication. In addition, pH monitoring prompted further investigation that in a number of cases pointed to other diseases (e.g., cholelithiasis and irritable bowel syndrome).

2. It establishes a temporal correlation between symptoms and episodes of reflux. Such a correlation is particularly important when atypical GERD symptoms (e.g., cough and chest pain) are present because 50% of these patients experience no heartburn and 50% do not have esophagitis on endoscopy.18

3. It allows staging on the basis of disease severity. Specifically, esophageal manometry and pH monitoring identify a subgroup of patients characterized by worse esophageal motor function (manifested by a defective LES or by abnormal esophageal peristalsis), more acid reflux in the distal and proximal esophagus, and slower acid clearance. These patients more frequently have Barrett metaplasia and experience respiratory symptoms; thus, they might benefit from early antireflux surgery.19

4. It provides baseline data that may prove useful postoperatively if symptoms do not respond to the procedure.

**Multichannel Intraluminal Impedance and pH** Combined multichannel intraluminal impedance and pH testing (MII-pH) has the ability to detect episodes of reflux, regardless of the pH of the refluxate, by identifying changes induced by the presence of a bolus in the esophagus; the episodes are then simply classified as acid or nonacid on the basis of concomitantly recorded pH values. Studies of healthy persons have demonstrated that MII-pH possesses increased sensitivity and specificity in detecting and characterizing gastroesophageal reflux, and the results have been shown to be highly reproducible.20

Although this technology is still not widely available, it has already been demonstrated to be useful in the workup of patients with GERD refractory to PPIs and patients with respiratory symptoms of unknown origin.21

**Operative Planning**

The patient is placed under general anesthesia and intubated with a single-lumen endotracheal tube. Abdominal wall relaxation is ensured by the administration of a nondepolarizing muscle relaxant, the action of which is rapidly reversed at the end of the operation. Adequate muscle relaxation is essential because increased abdominal wall compliance allows increased pneumoperitoneum, which yields better exposure. An orogastric tube is inserted at the beginning of the operation to keep the stomach decompressed; it is removed at the end of the procedure.

The patient is placed in a steep reverse Trendelenburg position, with the legs extended on stirrups. The surgeon stands between the patient’s legs. To keep the patient from sliding as a result of the steep position used during the operation, a bean bag is inflated under the patient, and the knees are flexed only 20° to 30°. A Foley catheter is inserted at the beginning of the procedure and usually is removed at the end. Because increased abdominal pressure from pneumoperitoneum and the steep reverse Trendelenburg position decrease venous return, pneumatic compression stockings are always used as prophylaxis against deep vein thrombosis.

The equipment required for a laparoscopic Nissen fundoplication includes five 10 mm trocars, a 30° laparoscope, a hook cautery, and various other instruments [see Table I]. In addition, we use a three-chip camera system that is separate from the laparoscope.

**Operative Technique**

A total fundoplication is the procedure of choice. A partial fundoplication [see Laparoscopic Partial (Guarner) Fundoplication, below] is performed only when peristalsis is absent.22,23 The operation may be divided into nine key steps as follows.

<table>
<thead>
<tr>
<th>Table 1 Instrumentation for Laparoscopic Nissen Fundoplication</th>
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<tbody>
<tr>
<td>Five 10 mm trocars</td>
</tr>
<tr>
<td>30° scope</td>
</tr>
<tr>
<td>Graspers and needle holder</td>
</tr>
<tr>
<td>Babcock clamp</td>
</tr>
<tr>
<td>L-shaped hook cautery with suction-irrigation capacity</td>
</tr>
<tr>
<td>Scissors</td>
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<tr>
<td>Laparoscopic clip applier</td>
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<tr>
<td>Ligasure™ Vessel Sealing System*</td>
</tr>
<tr>
<td>Fan retractor</td>
</tr>
<tr>
<td>Endo Stitch device†</td>
</tr>
<tr>
<td>Penrose drain</td>
</tr>
<tr>
<td>2-0 silk sutures</td>
</tr>
<tr>
<td>56 French esophageal bougie</td>
</tr>
</tbody>
</table>

* (Valleylab, Boulder, CO)  † (Autosuture, Norwalk, CT)
Step 1: Placement of Trocars

Five 10 mm trocars are used for the operation [see Figure 1]. Port A is placed about 14 cm below the xiphoid process; it can also be placed slightly (2 to 3 cm) to the left of the midline to be in line with the hiatus. This port is used for insertion of the scope. Port B is placed at the same level as port A but in the left midclavicular line. It is used for insertion of the Babcock clamp; insertion of a grasper to hold the Penrose drain once it is in place surrounding the esophagus; or insertion of an instrument to take down the short gastric vessels. Port C is placed at the same level as the previous two ports but in the right midclavicular line. It is used for insertion of the fan retractor, the purpose of which is to lift the lateral segment of the left hemiliver and expose the esophagogastric junction. We do not divide the left triangular ligament. The fan retractor can be held in place by a self-retaining system fixed to the operating table. Ports D and E are placed as high as possible under the costal margin and about 5 to 6 cm to the right and the left of the midline so that they are about 15 cm from the esophageal hiatus; in addition, they should be placed so that their axes form an angle of 60° to 120°. These ports are used for insertion of the graspers, the electrocautery, and the suturing instruments.

Troubleshooting  If the ports are placed too low in the abdomen, the operation is made more difficult. If port C is too low, the fan retractor will not retract the left lateral section of the liver well, and the esophagogastric junction will not be properly exposed. If port B is too low, the Babcock clamp will not reach the esophagogastric junction, and when the instrument used to take down the short gastric vessels is placed through the same port, it will not reach the upper short gastric vessels. If ports D and E are too low, the dissection at the beginning of the procedure and the suturing at the end are problematic.

Other mistakes of positioning must be avoided as well. Port C must not be placed too medially, because the fan retractor may clash with the left-hand instrument; the gallbladder fossa is a good landmark for positioning this port. Port A must be placed with extreme caution in the supraumbilical area: its insertion site is just above the aorta, close to its bifurcation. Accordingly, we initially inflate the abdomen to a pressure of 18 mm Hg just for placement of port A; increasing the distance between the abdominal wall and the aorta reduces the risk of aortic injury. We also recommend directing the port toward the coccyx. Once port A is in place, the intraperitoneal pressure is reduced to 15 mm Hg. A Hasson cannula can be used in this location, particularly if the patient has already had one or more midline incisions. Maintaining the proper angle (60° to 120°) between the axes of the two suturing instruments inserted through ports D and E is also important: if the angle is smaller, the instruments will cover part of the operating field, whereas if it is larger, depth perception may be impaired. Finally, if a trocar is not in the ideal position, it is better to insert another one than to operate through an inconveniently placed port.

If the surgeon spears the epigastric vessels with a trocar, bleeding will occur, in which case we prefer to ligate the vessel under laparoscopic guidance. We favor the Carter-Thomason CloseSure System (Inlet Medical, Inc., Eden Prairie, MN) to ligate the vessel securely with 0 absorbable suture. Once hemostasis is obtained, the surgeon simply repositions the trocar away from the vessels.

Step 2: Division of Gastrohepatic Ligament; Identification of Right Crus of Diaphragm and Posterior Vagus Nerve

Once the ports are in place, the gastrohepatic ligament is divided. Dissection begins above the caudate lobe (segments 1 and 9) of the liver, where this ligament usually is very thin, and continues toward the diaphragm until the right crus is identified. The crus is then separated from the right side of the esophagus by blunt dissection, and the posterior vagus nerve is identified. The right crus is dissected inferiorly toward the junction with the left crus.

Troubleshooting  An accessory left hepatic artery originating from the left gastric artery is frequently encountered in the gastrohepatic ligament. If this vessel creates problems of exposure, it may be divided; in our experience, doing so has not caused problems. In dissecting the right crus from the esophagus, the electrocautery should be used with particular caution. Because the monopolar current tends to spread laterally, the posterior vagus nerve may sustain damage simply from being in proximity to the device, even when there is no direct contact. A better alternative is to use a bipolar instrument.

Step 3: Division of Peritoneum and Phrenoesophageal Membrane above Esophagus; Identification of Left Crus of Diaphragm and Anterior Vagus Nerve

The peritoneum and the phrenoesophageal membrane above the esophagus are divided with the electrocautery, and the anterior vagus nerve is identified. The left crus of the diaphragm is dissected downward toward the junction with the right crus.
**Troubleshooting** Care must be taken not to damage the anterior vagus nerve or the esophageal wall. To this end, the nerve should be left attached to the esophageal wall, and the peritoneum and the phrenoesophageal membrane should be lifted from the wall by blunt dissection before they are divided.

*Step 4: Division of Short Gastric Vessels*

The 5 mm laparoscopic Ligasure Vessel Sealing System (Valleylab, Boulder, CO) is introduced through port B. A grasper is introduced by the surgeon through port D, and an assistant applies traction on the greater curvature of the stomach through port E. Dissection begins at the level of the middle portion of the gastric body and continues upward until the most proximal short gastric vessel is divided.

**Troubleshooting** There are two problems to watch for during this part of the procedure: (1) bleeding, either from the short gastric vessels or from the spleen, and (2) damage to the gastric wall.

Bleeding from the short gastric vessels is usually caused by excessive traction or by division of a vessel that is not completely coagulated. Vessels up to 5 mm in diameter can be taken down with the Ligasure device. Damage to the gastric wall can be caused by a burn from the electrocautery used to dissect between vessels or by traction applied with the grasper or the Babcock clamp.

*Step 5: Creation of Window between Gastric Fundus, Esophagus, and Diaphragmatic Crura; Placement of Penrose Drain around Esophagus*

The esophagus is retracted upward with a Babcock clamp applied at the level of the esophagogastric junction. By means of blunt and sharp dissection, a window is created under the esophagus between the gastric fundus, the esophagus, and the left pillar of the crus. The window is enlarged with the LigaSure device. Damage to the gastric wall can be avoided by properly dissecting and identifying the left pillar without having done enough dissection. Care must be taken not to damage the gastric wall.

**Troubleshooting** The two main problems to watch for during this part of the procedure are (1) creation of a left pneumothorax and (2) perforation of the gastric fundus.

A left pneumothorax is usually caused by dissection done above the left pillar of the crus in the mediastinum rather than between the crus and the gastric fundus. This problem can be avoided by properly dissecting and identifying the left pillar of the crus.

Perforation of the gastric fundus is usually caused by pushing a blunt instrument under the esophagus and below the left pillar without having done enough dissection. Care must be exercised in taking down small vessels from the fundus when the area behind the esophagus is approached from the right: the anatomy is not as clear from this viewpoint, and perforation can easily occur. Sometimes, perforation is caused by the use of a monopolar electrocautery for dissection. An electrocautery burn can go unrecognized during dissection and manifest itself in the form of a leak during the first 48 hours after operation.

*Step 6: Closure of Crura*

The diaphragmatic crura are closed with interrupted 2-0 silk sutures on an Endo Stitch device (Autosuture, Norwalk, CT); the sutures are tied intracorporeally. Exposure is provided by retracting the esophagus upward and toward the patient’s left with the Penrose drain. The lens of the 30° laparoscope is angled slightly to the left by moving the light cable of the scope to the patient’s right. The first stitch should be placed just above the junction of the two pillars. Additional stitches are placed 1 cm apart, and a space of about 1 cm is left between the uppermost stitch and the esophagus.

**Troubleshooting** Care must be taken not to spear esophageal wall with the needle. So as not to limit the space available for suturing, the bougie is not placed inside the esophagus during this part of the procedure.

*Step 7: Insertion of Bougie into Esophagus and through Esophagogastric Junction*

The esophageal stethoscope and the orogastric tube are removed, and a 56 French bougie is inserted by the anesthesiologist and passed through the esophagogastric junction under laparoscopic vision. The crura must be snug around the esophagus but not too tight: a closed grasper should slide easily between the esophagus and the crura.

**Troubleshooting** The most worrisome complication during this step is perforation of the esophagus. This can be prevented by lubricating the bougie and instructing the anesthesiologist to advance the bougie slowly and to stop if any resistance is encountered. In addition, it is essential to remove any instruments from the esophagogastric junction and to open the Penrose drain; these measures prevent the creation of an angle between the stomach and the esophagus, which can increase the likelihood of perforation. The position of the bougie can be confirmed by pressing with a grasper over the esophagus, which will feel full when the bougie is in place.

*Step 8: Wrapping of Gastric Fundus around Lower Esophagus*

The gastric fundus is gently pulled under the esophagus with the grasper. The left and right sides of the fundus are wrapped above the fat pad (which lies above the esophagogastric junction) and held together in place with a Babcock clamp introduced through port B. (The Penrose drain should be removed at this point because it is in the way.) Usually, three 2-0 silk sutures are used to secure the two ends of the wrap to each other. The stitches do not include the esophagus. Two coronal stitches are then placed between the top of the wrap and the esophagus, one on the right and one on the left. Finally, one additional suture is placed between the right side of the wrap and the closed crura.

To avoid the risk of injuring the inferior vena cava at the beginning of the dissection, some surgeons use a different method—the so-called left crus approach. In this approach, the operation begins with identification of the left crus of the diaphragm and division of the peritoneum and the phrenoesophageal membrane overlying it. The next step is division of the short gastric vessels, starting midway along the greater curvature of the stomach and continuing upward to join the area of the previous dissection. When the fundus has been thoroughly mobilized, the peritoneum is divided from the left to the right crus, and the right crus is dissected downward to expose the junction of the right and left crus. With this technique, the vena cava is never at risk. In addition, the branches of the anterior vagus nerve and...
the left gastric artery are less exposed to danger. This technique can be very useful, particularly for management of very large paraesophageal hernias and for second antireflux operations [see Reoperation for GERD, below].

Troubleshooting To determine whether the wrap is going to be floppy, the surgeon must deliver the fundus under the esophagus, making sure that the origins of the short gastric vessels have been transected are visible. Essentially, the posterior wall of the fundus is being used for the wrap. If the wrap remains to the right of the esophagus without retracting back to the left, then it is floppy, and suturing can proceed. If not, the surgeon must make sure that the upper short gastric vessels have been transected and the posterior dissection completed. If tension is still present after these maneuvers, it is probably best to perform a partial wrap [see Laparoscopic Partial (Guarner) Fundoplication, below].

Damage to the gastric wall may occur during the delivery of the fundus. Atraumatic graspers must be used, and the gastric fundus must be pulled gently and passed from one grasper to the other. Sometimes, it is helpful to push the gastric fundus under the esophagus from the left. The wrap should measure no more than 2 to 2.5 cm in length and, as noted, should be done with no more than three sutures. The first stitch is usually the lowest one; it must be placed just above the fat pad where the esophagogastric junction is thought to be.

If the anesthesiologist observes that peak airway pressure has increased (because of a pneumothorax) or that neck emphysema is present (because of pneumomediastinum), the pneumoperitoneum should be reduced from 15 mm Hg to 8 or 10 mm Hg until the end of the procedure. Pneumomediastinum tends to resolve without intervention within a few hours of the end of the procedure. Small pneumothoraces (usually on the left side) tend to resolve spontaneously, rendering insertion of a chest tube unnecessary. Larger pneumothoraces (> 20%), however, call for drainage. If the anesthesiologist observes that peak airway pressure has increased (because of tension from intact short gastric vessels or because the fundus is small), a corkscrew effect is created.

Step 9: Final Inspection, Removal of Instruments and Ports from Abdomen, and Closure of Port Sites

After hemostasis is obtained, the instruments and the ports are removed from the abdomen under direct vision. We usually close all port sites with 0 absorbable suture material using the Carter-Thomason CloseSure System.

Troubleshooting If any areas of ooze were observed, they should be irrigated and dried with sponges rolled into a cigaretlike shape before the ports are removed. In addition, if some grounds for concern remain, the oozing areas should be examined after the pneumoperitoneum is decreased to 7 to 8 mm Hg to abolish the tamponading effect exerted by the high intra-abdominal pressure.

All the ports should be removed from the abdomen under direct vision so that any bleeding from the abdominal wall can be readily detected. Such bleeding is easily controlled, either from inside or from outside.

Complications

A feared complication of laparoscopic Nissen fundoplication is esophageal or gastric perforation, which may result either from traction applied with the Babcock clamp or a grasper to the esophagus or the stomach (particularly when the stomach is

Almost every patient experiences some degree of dysphagia postoperatively. This problem usually resolves after 4 to 6 weeks, during which period patients receive pain medications in an elixir form and are advised to avoid eating meat and bread. If, however, dysphagia persists beyond this period, one or more of the following causes is responsible.

1. A wrap that is too tight or too long (i.e., > 2.5 cm).
2. Lateral torsion with corkscrew effect. If the wrap rotates to the right (because of tension from intact short gastric vessels or because the fundus is small), a corkscrew effect is created.
3. A wrap made with the body of the stomach rather than the fundus. The relaxation of the LES and the gastric fundus is controlled by vasoactive intestinal polypeptide and nitric oxide; after fundoplication, the two structures relax simultaneously with swallowing. If part of the body of the stomach rather than the fundus is used for the wrap, it will not relax as the LES does on arrival of the food bolus.
4. Choice of the wrong procedure. In patients who have severely abnormal esophageal peristalsis (as in end-stage connective tissue disorders), a partial wrap is preferable. A 360° wrap may cause postoperative dysphagia and gas bloat syndrome.

If the wrap slips into the chest, the patient may experience dysphagia and regurgitation. The diagnosis is confirmed by means of a barium swallow. This problem can be prevented by using coronal sutures and by ensuring that the crura are closed securely.

Paraesophageal hernia may occur if the crura have not been closed or if the closure is too loose. We believe that closure of the crura not only is essential for preventing paraesophageal hernia but also is important from a physiologic point of view, in that it acts synergistically with the LES against stress reflux. Sometimes, it is possible to reduce the stomach and close the crura laparoscopically. More often, however, because the crural opening is very tight and the gastric wall is edematous, laparoscopic repair is impossible and laparotomy is preferable.

Postoperative Care and Outcome Evaluation

Postoperative care and outcome evaluation of laparoscopic Nissen fundoplication are considered elsewhere in conjunction with the discussion of partial fundoplication [see Laparoscopic Partial (Guarner) Fundoplication, Postoperative Care and Outcome Evaluation, below].

Laparoscopic Partial (Guarner) Fundoplication

Preoperative Evaluation and Operative Planning

Preoperative evaluation and operative planning are essentially the same for partial (Guarner) fundoplication as for Nissen
Fundoplication. This operation should be performed only in patients with the most severe abnormalities of esophageal peristalsis: it is less effective than a 360° wrap for long-term control of reflux. In addition, laparoscopic partial fundoplication may be performed after laparoscopic Heller myotomy for achalasia [see Laparoscopic Heller Myotomy with Partial Fundoplication, below].

Operative Technique

The first seven steps in a Guarner fundoplication are identical to the first seven in a Nissen fundoplication. The wrap, however, differs in that it extends around only 240° to 280° of the esophageal circumference. Once the gastric fundus is delivered under the esophagus, the two sides are not approximated over the esophagus. Instead, 80° to 120° of the anterior esophagus is left uncovered, and each of the two sides of the wrap (right and left) is separately affixed to the esophagus with three 2-0 silk sutures, with each stitch including the muscle layer of the esophageal wall. The remaining stitches (i.e., the coronal stitches and the stitch between the right side of the wrap and the closed crura) are identical to those placed in a Nissen fundoplication.

Postoperative Care

Currently, our average operating time for a laparoscopic fundoplication is approximately 2 hours. We start patients on a soft mechanical diet on the morning of postoperative day 1 and usually discharge them after 23 to 48 hours. The recovery time typically ranges from 10 to 14 days.

Outcome Evaluation

The initial results of laparoscopic fundoplication obtained in the early 1990s indicated that the operation was effective in controlling reflux but that postoperative dysphagia occurred more often than had been anticipated. Many experts thought that this problem could be avoided by tailoring the fundoplication to the strength of esophageal peristalsis as measured by esophageal manometry. Accordingly, partial fundoplication (240°) was recommended for patients with impaired peristalsis, and total fundoplication (360°) was recommended for those with normal peristalsis. The short-term results of this tailored approach were promising. Gradually, however, it became evident that a partial fundoplication was not as durable as a total fundoplication and that a total fundoplication was not associated with a higher incidence of postoperative dysphagia even in patients with weak peristalsis.

These findings suggest that the initial problems with postoperative dysphagia were primarily attributable to unknown technical factors that were largely eliminated from the procedure as surgeons garnered more experience with it. As a result, total fundoplication is currently considered the procedure of choice for patients with GERD, regardless of the strength of their esophageal peristalsis.

In a 2007 study, pre- and postoperative manometric results were reviewed in 71 patients who underwent laparoscopic fundoplication. Not only did LES pressure increase after the procedure, but distal esophageal amplitude also increased in patients with abnormal preoperative motility, with normalization of peristalsis occurring in the majority of patients. A 2001 report found that 62% of fundoplication patients were using antireflux medications 10 years after the operation, a finding that raised concerns about long-term durability of the procedure. This study has been criticized on the grounds that many of the patients were taking PPIs for reasons other than reflux symptoms and that the reflux status was not assessed by pH monitoring. A 2006 study aimed at critically assessing 10-year outcomes reported results from 100 consecutive patients after complete and partial fundoplication. In this series, the rate of symptomatic control of reflux symptoms at 5 and 10 years was 90%, with fewer than 10% of patients using antacid medications at 10 years; only one patient required reintervention for persistent dysphagia. Similar results were documented in subsequent studies confirming laparoscopic Nissen fundoplication as an effective long-term treatment for GERD.

Laparoscopic Heller Myotomy with Partial Fundoplication

Minimally invasive surgical procedures for primary esophageal motility disorders (achalasia, diffuse esophageal spasm [DES], nutcracker esophagus [NE] and hypertensive LES [H-LES]) yield results that are comparable to those of open procedures but are associated with less postoperative pain and with a shorter recovery time. Today, laparoscopic Heller myotomy with partial fundoplication has supplanted left thoracoscopic myotomy as the procedure of choice for esophageal achalasia. Long-term studies demonstrated that even though left thoracoscopic myotomy led to resolution of dysphagia in about 85% to 90% of patients, it had the following four drawbacks.

1. Gastroesophageal reflux developed postoperatively in about 60% of patients because no fundoplication was performed in conjunction with the myotomy. With the laparoscopic approach, in contrast, a partial fundoplication can easily be performed, which prevents reflux in the majority of patients and corrects many instances of preexisting reflux arising from pneumatic dilatation. A prospective, randomized, double-blind clinical trial that compared Heller myotomy alone with Heller myotomy and Dor fundoplication clearly demonstrated that the addition of a fundoplication is essential: the incidence of postoperative reflux (as measured by pH monitoring) was 48% in patients who underwent myotomy alone but only 9% in those who underwent myotomy and Dor fundoplication.

2. The extension of the myotomy onto the gastric wall (clearly the most critical and challenging part of the operation) proved difficult because of poor exposure, with the consequent risk of a short myotomy and persistent dysphagia. With the laparoscopic approach, in contrast, excellent exposure of the esophagogastric junction is easily achieved, and the myotomy can be extended onto the gastric wall for about 2 to 2.5 cm.

3. Double-lumen endotracheal intubation and single-lung ventilation were required, with the patient in the right lateral decubitus position. In contrast, the setting for a laparoscopic myotomy (the same as that for a laparoscopic fundoplication) is much easier for the patient, the anesthesiologist, and the operating room personnel. In addition, most surgeons have by now acquired substantial experience with laparoscopic antireflux procedures and thus are more familiar and comfortable with laparoscopic exposure of the distal esophagus and the esophagogastric junction.
4. The average postoperative hospital stay was about 3 days because of the chest tube left in place at the time of the operation and the discomfort arising from the thoracic incisions. After a laparoscopic Heller myotomy, the hospital stay is only 1 or 2 days; there is no need for a chest tube, and patients are more comfortable.

Although there is now a consensus on the preferred treatment of achalasia, there is no such general agreement on the treatment of the remaining primary esophageal motility disorders (i.e., DES, NE, and H-LES). We reported our experience comparing thorascopic and laparoscopic approaches to these disorders and found that laparoscopic myotomy was superior to thorascopic myotomy in relieving dysphagia in patients with DES and H-LES, and that the two approaches yielded equally disappointing results with respect to relieving chest pain in patients with NE, the surgical treatment of which remains elusive. These results support the view that laparoscopic Heller myotomy should be the standard surgical treatment for achalasia, DES, and H-LES. Accordingly, we would consider surgical intervention (laparoscopic Heller myotomy) in an NE patient only in an attempt at relieving severe dysphagia.

We no longer perform a long myotomy via a right thorascopic approach.

**PREOPERATIVE EVALUATION**

All candidates for a laparoscopic Heller myotomy should undergo a thorough and careful evaluation to establish the diagnosis and characterize the disease.

An upper GI series is useful. A characteristic so-called bird’s beak is usually seen in patients with achalasia. A dilated, sigmoid esophagus may be present in patients with long-standing achalasia. A corkscrew esophagus is often seen in patients with diffuse esophageal spasm. Endoscopy is performed to rule out a tumor of the esophagogastric junction and gastroesophageal pathologic conditions.

Esophageal manometry is the key test for establishing the diagnosis of esophageal achalasia. The classic manometric findings are (1) absence of esophageal peristalsis and (2) an LES that fails to relax appropriately in response to swallowing.

Ambulatory pH monitoring should always be done in patients who have undergone pneumatic dilatation to rule out abnormal gastroesophageal reflux. In addition, pH monitoring should be performed postoperatively to detect abnormal reflux, which, if present, should be treated with acid-reducing medications.

In patients older than 60 years who have experienced the recent onset of dysphagia and excessive weight loss, secondary achalasia or pseudoachalasia from cancer of the esophagogastric junction should be ruled out. Endoscopic ultrasonography or computed tomography can help establish the diagnosis.

**OPERATIVE PLANNING**

Patient preparation (i.e., anesthesia, positioning, and instrumentation) is identical to that for laparoscopic fundoplication.

**OPERATIVE TECHNIQUE**

Many of the steps in a laparoscopic Heller myotomy are the same as the corresponding steps in a laparoscopic fundoplication. The ensuing description focuses on those steps that differ significantly.

Either a Dor or a Guaraner fundoplication [see Laparoscopic Partial (Guaraner) Fundoplication, above] may be performed in conjunction with a Heller myotomy. The Dor fundoplication is an anterior 180° wrap. Its advantages are that (1) it does not require posterior dissection and the creation of a window between the esophagus, the stomach, and the left pillar of the crus; (2) it covers the exposed esophageal mucosa after completion of the myotomy; and (3) it is effective even in patients with GERD. Its main disadvantage is that achieving the proper geometry can be difficult, and a wrong configuration can lead to dysphagia even after a properly performed myotomy. The advantages of the Guaraner fundoplication are that (1) it is easier to perform; (2) it keeps the edges of the myotomy well separated; and (3) it might be more effective than a Dor procedure in preventing refl ux. Its main disadvantages are that (1) it requires more dissection for the creation of a posterior window, and (2) it leaves the esophageal mucosa exposed.

**Steps 1 through 6**

Steps 1, 2, 3, 4, 5, and 6 of a laparoscopic Heller myotomy are essentially identical to the first six steps of a laparoscopic fundoplication. Steps 5 and 6, however, are necessary only if a posterior partial fundoplication is to be performed. Care must be taken not to narrow the esophageal hiatus too much and push the esophagus anteriorly.

**Step 7: Intraoperative Endoscopy**

At the beginning of a surgeon’s experience with laparoscopic Heller myotomy, intraoperative endoscopy is an important and helpful step; however, once the surgeon has gained adequate experience with this procedure and has become familiar with the relevant anatomy from a laparoscopic perspective, it may be omitted.

**Troubleshooting** The most worrisome complication during intraoperative endoscopy is perforation of the esophagus. This complication can be prevented by having the procedure done by an experienced endoscopist who is familiar with achalasia.

**Step 8: Initiation of Myotomy and Entry into Submucosal Plane at Single Point**

The fat pad is removed with the LigaSure device to provide clear exposure of the esophagogastric junction. A Babcock clamp is then applied over the junction, and the esophagus is pulled downward and to the left to expose the right side of the esophagus. The myotomy is performed at the 11 o’clock position. It is helpful to mark the surface of the esophagus along the line through which the myotomy will be carried out [see Figure 2]. The myotomy is started about 3 cm above the esophagogastric junction. Before it is extended upward and downward, the proper submucosal plane should be reached at a single point; in this way, the likelihood of subsequent mucosal perforation can be reduced.

**Troubleshooting** The myotomy should not be started close to the esophagogastric junction, because at this level the layers often are poorly defined, particularly if multiple dilatations or injections of botulinum toxin have been performed. At the preferred starting point, about 3 cm above the esophagogastric junction, the esophageal wall is usually normal. As a rule, we do
not open the entire longitudinal layer first and then the circular
layer; we find it easier and safer to try to reach the submucosal
plane at a single point and then move upward and downward
from there. In the course of the myotomy, there is always some
bleeding from the cut muscle fibers, particularly if the esophagus
is dilated and the wall is very thick. After the source of the bleed-
ing is identified, the electrocautery must be used with caution.
The most troublesome bleeding comes from the submucosal
veins encountered at the esophagogastric junction (which are
usually large). In most instances, gentle compression is prefer-
able to electrocautery. A sponge introduced through one of the
ports facilitates the application of direct pressure.

**Step 9: Proximal and Distal Extension of Myotomy**

Once the mucosa has been exposed, the myotomy can safely
be extended [see Figure 3]. Distally, it is extended for about 2 to
2.5 cm onto the gastric wall; proximally, it is extended for about
6 cm above the esophagogastric junction. Thus, the total length
of the myotomy is typically about 8 to 8.5 cm [see Figure 4]. We
find it useful to alternate between a dolphin-nose grasper and
using a curved micrograsper during extension of the myotomy;
this makes the dissection safer, in that it allows the muscle fibers
to be separated from the mucosa before being transected.

**Troubleshooting** The course of the anterior vagus nerve
must be identified before the myotomy is started. If this nerve
crosses the line of the myotomy, it must be lifted away from the
esophageal wall, and the muscle layers must then be cut under
it. In addition, care must be taken not to injure the anterior
vagus nerve while removing the fat pad. Treatment with botuli-
um toxin occasionally results in fibrosis with scarring and loss
of the normal anatomic planes; this occurs more frequently at
the level of the esophagogastric junction.

If a perforation seems possible or likely, it should be sought
as described earlier [see Step 7: Intraoperative Endoscopy,
above]. Any perforation found should be repaired with 5-0
absorbable suture material, with interrupted sutures employed
for a small perforation and a continuous suture for a larger one.
When a perforation has occurred, an anterior fundoplication is
usually chosen in preference to a posterior one because the
stomach will offer further protection against a leak.

**Step 10 (Dor Procedure): Anterior Partial Fundoplication**

Two rows of sutures are placed. The first row (on the left
side) comprises three stitches: the uppermost stitch incorporates
the gastric fundus, the esophageal wall, and the left pillar of the
crus [see Figure 5], and the other two incorporate only the gastric
fundus and the left side of the esophageal wall [see Figure 6]. The
gastric fundus is then folded over the myotomy, and the second
row (also comprising three stitches) is placed on the right side
between the fundus and the right side of the esophageal wall,
with only the uppermost stitch incorporating the right pillar
of the crus [see Figure 7 and Figure 8]. Finally, two additional
stitches are placed between the anterior rim of the hiatus and the
superior aspect of the fundoplication [see Figure 9]. These stitches
remove any tension from the second row of sutures.
Figure 4  Laparoscopic Heller myotomy with partial fundoplication. The myotomy is approximately 8 cm long, extending distally for about 2 to 2.5 cm onto the gastric wall and proximally for about 6 cm above the esophagogastric junction.

Figure 5  Laparoscopic Heller myotomy with anterior partial fundoplication (Dor procedure). The uppermost stitch in the first row incorporates the fundus, the esophageal wall, and the left pillar of the crus.

Figure 6  Laparoscopic Heller myotomy with anterior partial fundoplication (Dor procedure). The second and third stitches in the first row incorporate only the fundus and the left side of the esophageal wall.

Figure 7  Laparoscopic Heller myotomy with anterior partial fundoplication (Dor procedure). The uppermost stitch in the second row incorporates the fundus, the esophageal wall, and the right crus.
Troubleshooting  Efforts must be made to ensure that the fundoplication does not become a cause of postoperative dysphagia. Accordingly, we always take down the short gastric vessels, even though some authorities suggest that this step can be omitted. In addition, the gastric fundus rather than the body of the stomach should be used for the wrap, and only the uppermost stitch of the right row of sutures should incorporate the right pillar of the crus.

Step 10 (Guarner Procedure): Posterior Partial Fundoplication

Alternatively, a posterior 220° fundoplication may be performed. The gastric fundus is delivered under the esophagus, and each side of the wrap (right and left) is attached to the esophageal wall, lateral to the myotomy, with three sutures [see Figure 10].

Step 11: Final Inspection and Removal of Instruments and Ports from Abdomen

Step 11 of a laparoscopic Heller myotomy is identical to step 9 of a laparoscopic Nissen fundoplication.

Complications

Delayed esophageal leakage, usually resulting from an electrocautery burn to the esophageal mucosa, may occur during the first 24 to 36 hours after operation. The characteristic signs and symptoms are chest pain, fever, and a pleural effusion on the chest x-ray. The diagnosis is confirmed by an esophagogram. Treatment options depend on the time of diagnosis and on the size and location of the leak. Early, small leaks can be repaired directly. If the site of the leak is high in the chest, a thoracotomy...
is recommended; if the site is at the level of the esophagogastric junction, a laparotomy is preferable, and the stomach can be used to reinforce the repair. If the damage to the esophagus is too extensive to permit repair, an esophagectomy [see 4:7 Open Esophageal Procedures] is indicated.

Dysphagia may either persist after the operation or recur after a symptom-free interval. In either case, a complete workup is necessary, and treatment is individualized on the basis of the specific cause of dysphagia. Reoperation may be indicated [see Reoperation for Esophageal Achalasia, below].

Abnormal gastroesophageal reflux occurs in 7 to 20% of patients after operation.43 Because most patients are asymptomatic, it is essential to try to evaluate all patients postoperatively with manometry and prolonged pH monitoring. Reflux should be treated with acid-reducing medications.

**POSTOPERATIVE CARE**

We do not routinely obtain an esophagogram before initiating feeding. Patients are started on a soft mechanical diet on the morning of postoperative day 1, and this diet is continued for the rest of the first week. Patients are discharged after 24 to 48 hours and are able to resume regular activities in 7 to 14 days.

**OUTCOME EVALUATION**

The results obtained to date with laparoscopic Heller myotomy and partial fundoplication are excellent and are generally comparable to those obtained with the corresponding open surgical procedure: dysphagia is alleviated or eliminated in more than 90% of patients.3,6,42 These results have been confirmed in long-term follow-up outcome studies. One report from the University of Padua found that at a minimum of 6 years after surgical intervention, 85% of myotomy patients were satisfied.43

In a 2008 report describing our experience with 113 patients treated for achalasia with laparoscopic Heller myotomy and Dor fundoplication, we found that at a median follow-up of 45 months (range, 7 months to 12.5 years), 80% experienced complete relief of dysphagia after the operation.44 The remaining 20% required endoscopic dilatation, which was successful in 50% of the cases. We have observed a significant change in the treatment algorithm for this condition: gastroenterologists now typically refer these patients for surgical treatment before any other therapy has been instituted, reserving endoscopic dilatation for cases where operative treatment has failed.45 Overall, combined treatment yields a success rate of about 90%.

Historically, the presence of a sigmoid-shaped esophagus has been considered a contraindication to performing a myotomy to treat longstanding achalasia, making esophagectomy the only option. In the study just mentioned,44 we subdivided the patients according to the degree of esophageal dilatation and observed the responses to surgery in the various groups. Even in those cases where the esophageal diameter exceeded 6 cm and the esophagus was sigmoid, the outcomes of laparoscopic myotomy were not significantly different.44

**Left Thoracoscopic Myotomy**

Currently, we would consider a left thoracoscopic myotomy for patients in whom multiple previous abdominal procedures (done to treat other diseases) would preclude a laparoscopic approach.

**PREOPERATIVE EVALUATION**

Preoperative evaluation is essentially the same as that for laparoscopic Heller myotomy.

**OPERATIVE PLANNING**

The patient is placed under general anesthesia and intubated with a double-lumen endotracheal tube so that the left lung can be deflated during the procedure. As for a left thoracotomy, the patient is placed in the right lateral decubitus position over an inflated bean bag. The instrumentation is similar to that for a laparoscopic myotomy and partial fundoplication. Instead of conventional trocars, four or five thoracports with blunt obturators are employed, because insufflation of the thoracic cavity is not required. The myotomy can be performed with a monopolar hook cautery, bipolar scissors, or an ultrasonic scalpel. A 30° scope and a 45° scope are essential for thoracoscopic procedures. In addition, an endoscope is used for intraoperative endoscopy.

**OPERATIVE TECHNIQUE**

**Step 1: Placement of Thoracoports**

Five ports are usually placed [see Figure 11]. Port A, used for the 30° scope, is inserted in the sixth intercostal space about 3.5 to 5 cm behind the posterior axillary line. Port B, used for the lung retractor, is placed in the third intercostal space about 1.25 to 2.5 cm anterior to the posterior axillary line. Port C, used for insertion of a grasper, is placed in the sixth intercostal space in the anterior axillary line. Port D, used for the 45° scope, is inserted in the sixth intercostal space about 2.5 cm anterior to the posterior axillary line. Port E is placed in the seventh intercostal space in the midaxillary line. This port is optional: it is needed in about 30% of cases to allow the surgeon to obtain further exposure of the esophagogastric junction through retraction of the diaphragm.

**Troubleshooting** A common mistake is to insert port A too anteriorly. This port must be placed well beyond the...
posterior axillary line to provide the best angle for the 30° scope. Often, the other ports are placed one or two intercostal spaces too high. This mistake hampers the performance of the most delicate portion of the operation, the myotomy of the distal portion of the esophagus and the stomach.

Sometimes, chest wall bleeding occurs as a consequence of port insertion. This bleeding will obscure the operating field and therefore must be stopped before the intrathoracic portion of the procedure is begun. This is accomplished either by using the cautery from the inside or by applying a stitch from the outside if an intercostal vessel has been damaged.

**Step 2: Retraction of Left Lung and Division of Inferior Pulmonary Ligament**

Once the ports are in place, the deflated left lung is retracted cephalad with a fan retractor introduced through port B. This maneuver places tension on the inferior pulmonary ligament, which is then divided. After the ligament is divided, the fan retractor can be held in place by a self-retaining system fixed to the operating table.

**Troubleshooting** Before the inferior pulmonary ligament is divided, the inferior pulmonary vein must be identified to prevent a life-threatening injury to this vessel. If oxygen saturation decreases, particularly in patients with lung disease, the retractor should be removed and the lung inflated intermittently.

**Step 3: Division of Mediastinal Pleura and Dissection of Periesophageal Tissues**

The mediastinal pleura is divided, and the tissues overlying the esophageal wall are dissected until the wall of the esophagus is visible. This maneuver varies in difficulty depending on the width of the space between the aorta and the pericardium (which sometimes is very small) and on the size and shape of the esophagus. Large (sigmoid) esophagi tend to curve to the right, which makes identification of the wall difficult. If the esophagus is not immediately apparent, it can be easily identified in the groove between the heart and the aorta by means of transillumination provided by an endoscope [see Figure 12].

**Troubleshooting** The endoscope placed inside the esophagus at the beginning of the procedure plays an important role. In the early stages of the procedure, it allows identification of the esophagus via transillumination. When the light intensity of the 30° scope is turned down, the esophagus appears as a bright structure. In addition, tilting the tip of the endoscope brings the esophagus into view as it is lifted from the groove between the aorta and the heart.

**Step 4: Initiation of Myotomy and Entry into Submucosal Plane at Single Point**

As in a laparoscopic Heller myotomy, it is helpful to mark the surface of the esophagus along the line through which the myotomy will be carried out. The myotomy is started halfway between the diaphragm and the inferior pulmonary vein. Again, the proper submucosal plane should be reached at a single point before the myotomy is extended upward and downward.

**Troubleshooting** Troubleshooting for this step is essentially the same as that for step 8 of a laparoscopic Heller myotomy, with the exception that here the myotomy is started 4 to 5 cm (rather than 3 cm) above the esophagogastric junction.

**Step 5: Proximal and Distal Extension of Myotomy**

Once the mucosa has been exposed, the myotomy can safely be extended proximally and distally [see Figure 13]. We usually extend the myotomy for about 5 mm onto the gastric wall, without adding an antireflux procedure. Typically, the total length of the myotomy is about 6 cm for patients with achalasia.

**Troubleshooting** Proximally, the myotomy is extended all the way to the inferior pulmonary vein only in cases of vigorous achalasia (high-amplitude simultaneous contractions associated with chest pain in addition to dysphagia) or diffuse

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**Figure 12** Left thoracoscopic myotomy. The esophagus may be identified by means of transillumination from the endoscope.

**Figure 13** Left thoracoscopic myotomy. Shown are the distal and proximal extensions of the myotomy.
esophageal spasm; otherwise, it is limited to the distal 5 to 6 cm of the esophagus. If a longer myotomy is needed, the lung is displaced anteriorly and the myotomy extended to the aortic arch.

Distally, the myotomy is continued for 5 mm past the esophagogastric junction. The endoluminal view provided by the endoscope is useful for assessing the location of the esophagogastric junction. Often, the stomach is distended by the air insufflated by the endoscope and pushes the diaphragm upward, thereby limiting the view of the esophagogastric junction. If sucking air out of the stomach does not resolve this problem, an additional port (i.e., port E) may be placed in the eighth intercostal space, and a fan retractor may be introduced through this port to push the diaphragm down.

Because the myotomy of the gastric wall is the most challenging part of the operation, good exposure is essential. It is at this level that an esophageal perforation is most likely to occur. The risk is particularly high in patients who have undergone pneumatic dilatation or injection of botulinum toxin, both of which may lead to the replacement of muscle layers by scar tissue and the consequent loss of the regular planes. Perforations recognized in the operating room can be repaired by thoracoscopic intracorporeal suturing or, if this fails, by thoracotomy and open repair. The gastric fundus can be used to buttress the repair. If it is unclear whether a perforation has occurred, the esophagus should be covered with water and air insufflated through the endoscope; bubbling will be observed over the site of any perforation present.

Step 6: Insertion of Chest Tube and Removal of Thoracoports

A 24 French angled chest tube is inserted under direct vision through port D or port E. The ports are removed under direct vision, and the thoracic wall is inspected for bleeding.

Complications

As with laparoscopic Heller myotomy, delayed esophageal leakage is a common postoperative complication, and treatment options are similar.

If the myotomy is not extended far enough onto the gastric wall, residual dysphagia occurs. To prevent this problem, the distal extent of the myotomy should be assessed by means of endoscopy with the goal of including 5 mm of the gastric wall. Patients with residual dysphagia must be evaluated by means of esophageal manometry, which will document the extent of the residual high-pressure zone and the pressure within it. The myotomy can be easily extended by a laparoscopic approach, and a Dor fundoplication can be added.

If, on the other hand, the myotomy is extended too far onto the gastric wall, abnormal gastroesophageal reflux occurs. Some patients present with heartburn; others are asymptomatic. It is essential to evaluate patients postoperatively with manometry and prolonged pH monitoring. Mild reflux can be treated with acid-reducing medications, particularly in elderly patients. In younger patients, abnormal reflux should be corrected with a laparoscopic partial fundoplication (e.g., Dor fundoplication).

Postoperative Care

Patients are started on a liquid diet the morning of postoperative day 1; on postoperative day 2, they are started on a soft mechanical diet, which is continued for the rest of the first week. We do not routinely obtain an esophagogram before starting feedings. The chest tube is removed after 24 hours if the lung is fully expanded and there is no air leak. Patients are discharged after 48 to 72 hours and are able to resume regular activities in 7 to 10 days.

Outcome Evaluation

The results obtained with thoracoscopic myotomy are generally comparable to those obtained with corresponding open surgical procedures. In a 1999 study from UCSF, 26 (87%) of the first 30 patients with achalasia who were treated in this fashion experienced good or excellent results [see Table 2]. In present, however, this procedure is rarely used to treat esophageal achalasia: laparoscopic Heller myotomy and Dor fundoplication is now the treatment of choice.

Reoperation for GERD

Currently, an increasing number of patients are being seen for evaluation and treatment of foregut symptoms after laparoscopic antireflux surgery. These patients are treated as follows.

Preoperative Evaluation

Some degree of dysphagia, bloating, and abdominal discomfort is common during the first 6 to 8 weeks after a fundoplication. If these symptoms persist or heartburn and regurgitation occur, a thorough evaluation (with barium swallow, endoscopy, esophageal manometry, and pH monitoring) is carried out with the aim of answering the following three questions:

1. Are the symptoms attributable to persistent gastroesophageal reflux?
2. Are the symptoms attributable to the fundoplication itself?
3. Can the cause of the failure of the first operation be identified and corrected by a second operation?

Many patients report heartburn after a fundoplication. It is often assumed that this symptom must be the result of a failed operation and that acid-reducing medications should be restarted. In most cases, however, this assumption is mistaken:

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Results of Thoracoscopic Myotomy in 30 Patients with Achalasia</th>
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</thead>
<tbody>
<tr>
<td>Results</td>
<td>Patients (% of Total)</td>
</tr>
<tr>
<td>Excellent (no dysphagia)</td>
<td>21 (70)</td>
</tr>
<tr>
<td>Good (dysphagia &lt; once/wk)</td>
<td>5 (17)</td>
</tr>
<tr>
<td>Fair (dysphagia &gt; once/wk)</td>
<td>3 (10)</td>
</tr>
<tr>
<td>Poor (persistent dysphagia)</td>
<td>1 (3)</td>
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postoperative pH monitoring yields abnormal results in only about 20% of patients. The value of manometry lies in its ability to document the changes caused by the operation at the level of the LES and the esophageal body. The pH monitoring assesses the reflux status and determines whether there is a correlation between symptoms and actual episodes of reflux. If abnormal reflux is in fact present, the therapeutic choice is between medical therapy and a second operation.

Other patients complain of dysphagia arising de novo after the operation. This symptom is usually attributable to the operation itself and may occur in the absence of abnormal reflux. In addition to manometry and pH monitoring, a barium swallow is essential to define the anatomy of the esophagogastric junction. A study from the University of Washington found that the anatomic configurations observed could be divided into three main types: (1) type I hernia, in which the esophagogastric junction was above the diaphragm (subdivided into type IA, with both the esophagogastric junction and the wrap above the diaphragm, and type IB, with only the esophagogastric junction above the diaphragm); (2) type II hernia, a paraesophageal configuration; and (3) type III hernia, in which the esophagogastric junction was below the diaphragm and there was no evidence of hernia but in which the body of the stomach rather than the fundus was used for the wrap. In 10% of patients, however, the cause of the failure could not be identified preoperatively. Some patients present with a mix of postprandial bloating, nausea, and diarrhea. These symptoms may be the result of damage to the vagus nerves. Radionuclide evaluation of gastric emptying often helps quantify the problem.

**OPERATIVE PLANNING**

Patient preparation (i.e., anesthesia, positioning, and instrumentation) for a reoperation for reflux is identical to that for the initial laparoscopic fundoplication.

**OPERATIVE TECHNIQUE**

We do not routinely attempt a second antireflux operation laparoscopically. To provide a stepwise technical description that would be suitable for all reoperations for reflux is impossible, because the optimal procedure depends on the original approach (open versus laparoscopic), the severity of the adhesions, and the specific technique used for the first operation (total or partial fundoplication). The key goals of reoperation for reflux are as follows.

1. To dissect the wrap and the esophagus away from the crura. This is the most difficult part of the operation. The major complications seen during this part of the procedure are damage to the vagus nerves and perforation of the esophagus and the gastric fundus.
2. To take down the previous repair. The earlier repair must be completely undone and the gastric fundus returned to its natural position. If the short gastric vessels were not taken down during the first procedure, they must be taken down during the second.
3. To dissect the esophagus in the posterior mediastinum so as to have enough esophageal length below the diaphragm and avoid placing tension on the repair.
4. To reconstruct the cardia. The same steps are followed as for a first-time repair. If, after extensive esophageal mobilization, the esophagogastric junction remains above the diaphragm (short esophagus), esophageal lengthening can be accomplished by adding a thoracoscopic Collis gastroplasty to the fundoplication. To date, however, we have never found this step to be necessary.

**COMPLICATIONS**

Because the risk of gastric or esophageal perforation or damage to the vagus nerves is much higher during a second antireflux operation, the surgeon must proceed with extreme care, making sure to identify structures completely before dividing them. Most perforations are recognized and repaired intraoperatively. Leaks manifest themselves during the first 48 hours. Peritoneal signs are noted if the spillage is limited to the abdomen; shortness of breath and a pleural effusion are noted if spillage also occurs in the chest. The site of the leak should always be confirmed by means of a contrast study with barium or a watersoluble agent. Perforation is best handled with laparotomy and direct repair of the leak.

**OUTCOME EVALUATION**

Whereas the success rate is around 80 to 90% for a first antireflux operation, it falls to 70 to 80% for a second such operation. In our view, a second operation should be attempted by an expert team only if medical management fails to control heartburn or dilatation has not relieved dysphagia.

**Reoperation for Esophageal Achalasia**

Laparoscopic Heller myotomy improves swallowing in more than 90% of patients. What causes the relatively few failures reported is still incompletely understood. Typically, a failed Heller myotomy is signaled either by persistent dysphagia or by recurrent dysphagia that develops after a variable symptom-free interval following the original operation.

A complete workup (routinely including barium swallow, endoscopy, manometry, and pH monitoring) is required before treatment is planned. In addition, it is our practice to review the video of the first operation to search for technical errors that might have been responsible for the poor outcome. Such errors typically fall into one of the following three categories.

1. A myotomy that is too short either distally or proximally. If the myotomy is too short distally, a barium swallow shows persistent distal esophageal narrowing and manometry shows a residual high-pressure zone. If the myotomy is too short proximally, it will be apparent from the barium swallow.
2. A constricting Dor fundoplication. Often, manometry and pH monitoring yield normal results, but a barium swallow shows slow passage of contrast media from the esophagus into the stomach. In one study from UCSF, problems with Dor fundoplications occurred in four (4%) of 102 patients. Analysis of the video records of the first operations showed that in three of the four patients, all the stitches in the right suture row had incorporated the esophagus, the right pillar of the crus, and the stomach, thereby constricting the myotomy. In one patient, the short gastric vessels had not been taken down, and the body of the stomach rather than the fundus had been used for the fundoplication.
3. Transmural scarring caused by previous treatment. In patients treated with intraspincteric injection of botulinum toxin, transmural fibrosis can sometimes be found at the level of the esophagogastric junction. This
unwelcome finding makes the myotomy more difficult and the results less reliable.

There are two treatment options for persistent or recurrent dysphagia after Heller myotomy: (1) pneumatic dilatation and (2) a second operation tailored to the results of preoperative evaluation. In a 2002 study, pneumatic dilatation was successfully used to treat seven of 10 patients who experienced dysphagia postoperatively; of the remaining three patients, two required a second operation and one refused any treatment.

Reoperation for achalasia is technically challenging. It is of paramount importance to avoid perforating the exposed esophageal mucosa during the dissection. A small hole can be repaired, but a larger laceration might necessitate an esophagectomy. This option should always be discussed with the patient before the operation. It is our belief that the surgeon attempting a reoperation after a failed attempt at surgical treatment of achalasia should perform a laparotomy, even though several reports have stressed the feasibility of laparoscopic reoperation after a failed myotomy.

Overall, about 10 to 20% of patients experience some degree of dysphagia after a Heller myotomy. Pneumatic dilatation, a second myotomy, or both should always be tried before an esophagectomy is considered.

References


Acknowledgment

Figures 1 through 13 Tom Moore.