Laparoscopic Nissen Fundoplication and Heller Myotomy

Michael E. Friscia and Jo Buyske

LAPAROSCOPIC NISSEN FUNDOPLICATION

Case Study

A 38-year-old female is referred for management of gastroesophageal reflux. She reports a 10-year history of severe substernal postprandial chest pain. Calcium carbonate tablets and diet modification do little to improve these symptoms. She is currently taking a proton pump inhibitor, which provides moderate control of her symptoms. She denies dysphagia or weight loss. On recent esophagoscopy, orange patches interspersed between normal-appearing mucosa were noted in the distal esophagus. Biopsy confirmed the diagnosis of Barrett’s esophagus without dysplasia.

BACKGROUND

Some degree of reflux from the stomach into the esophagus is normal, particularly after meals, and is easily cleared by esophageal peristalsis. The high-pressure zone created by the lower esophageal sphincter (LES), diaphragmatic contraction at the esophageal hiatus during periods of increased abdominal pressure (e.g., coughing and bending), and the intra-abdominal positive pressure exerted on the distal esophagus serve to impede reflux. In contrast, migration of the distal esophagus and LES into the chest, as in a hiatal hernia, transient relaxation of the LES, and inhibitors of LES contraction, including caffeine, smoking, and alcohol, may exacerbate reflux. Repetitive injury of the esophageal mucosa by gastric acid results in gastroesophageal reflux disease (GERD) characterized by chronic symptoms (e.g., substernal chest pain and regurgitation) and, sometimes, complications such as Barrett’s esophagus and stricture formation.

Surgical therapy for the treatment of GERD is a relatively recent innovation. Indeed, the gastric fundoplication operations still performed today were introduced in the 1950s by Nissen, Belsey, and others. The application of minimally invasive techniques has led to more rapid recovery and reduced morbidity after antireflux surgery. As a result, the indications for surgery have broadened despite the availability of relatively effective medical therapies. In most cases, the antireflux operation of choice is the Nissen fundoplication, which involves a 360-degree wrap of the fundus of the stomach around the distal esophagus. In selected circumstances (e.g., when esophageal motility is markedly abnormal), a partial wrap may be preferable. Laparoscopic Nissen fundoplication is the focus of the first part of this chapter.

INDICATIONS FOR SURGERY

There are few absolute indications for antireflux surgery. A number of relative indications have emerged and must be weighed against associated risks.

I. Intractability of symptoms despite maximal medical therapy with proton pump inhibitors and H$_2$ receptor blockers is the most common indication for surgery. Although the presence of typical primary symptoms (e.g., heartburn) is generally
predictive of good outcomes after antireflux surgery, patients with pulmonary manifestations attributable to GERD (e.g., cough, pharyngitis, and recurrent pneumonia) may benefit from surgery as well, assuming other etiologies have been excluded. Patient age is an important factor in identifying appropriate surgical candidates; the benefits of surgery over lifelong proton pump inhibitor therapy are greatest in younger patients with severe disease. When patients receive no benefit from proton pump inhibition, diagnoses other than GERD should be considered. In general, such patients benefit little from antireflux surgery.

II. Peptic strictures are areas of fibrotic narrowing in the distal esophagus. Patients typically present with dysphagia and obstruction. The presence of a stricture suggests severe, repetitive esophageal injury from acid exposure. After evaluation, including endoscopy and biopsy to exclude malignancy, consideration should be given to antireflux surgery to reduce esophageal acid exposure and promote healing. Strictures are sometimes accompanied by a shortened esophagus. In such cases, fundoplication should be performed with an esophageal lengthening procedure (e.g., Collis gastroplasty).

III. Barrett’s esophagus, columnar metaplasia of the distal esophagus, is a premalignant condition that predisposes to the development of esophageal adenocarcinoma. Delayed progression or even regression of Barrett’s esophagus has been documented after antireflux surgery. However, whether surgery influences the subsequent risk of esophageal cancer remains controversial. Barrett’s esophagus with high-grade dysplasia is associated with undiagnosed esophageal adenocarcinoma in 38% to 72% of cases and should be treated with a resection (i.e., esophagectomy) rather than antireflux surgery.

PREOPERATIVE EVALUATION

I. History: The timing and chronicity of symptoms, modifying factors, and the effect of reflux symptoms on the patient’s lifestyle should be elicited. Response to acid suppression medications should be determined and is predictive of outcome after fundoplication.

II. Upper gastrointestinal fluoroscopy with barium allows for the diagnosis of a shortened esophagus, hiatal hernia, or mass, which may influence the surgical approach (e.g., the addition of an esophageal lengthening procedure).

III. Esophagogastroduodenoscopy allows for an assessment of the degree of esophageal injury. Specifically, esophagitis can be graded, and the presence of Barrett’s esophagus, dysplasia, and neoplasm can be identified.

IV. Twenty-four-hour pH monitoring is the gold standard for documentation of acid reflux into the distal esophagus. After the patient undergoes a 1-week hiatus from acid suppression therapy, pH probes are passed through the nose into the stomach, the distal esophagus above the LES, and the upper esophagus. Symptoms that correlate with episodes of acidification of the distal esophagus are more likely to improve with antireflux surgery.

V. Esophageal manometry provides information about the function of the LES and contractile properties of the esophageal body. Patients with abnormal peristalsis of the esophageal body will likely experience dysphagia when a 360-degree fundoplication is used and are better served by a partial fundoplication.

COMPONENTS OF THE PROCEDURE AND APPLIED ANATOMY

See Figure 7-1.

I. Preoperative Considerations
   A. Prophylactic antibiotics to cover gram-positive and enteric organisms are administered within 1 hour before skin incision.
   B. An orogastric tube is inserted to decompress the stomach.
   C. A urinary catheter is inserted to decompress the bladder and to facilitate intraoperative assessment of volume status.
II  Patient Position and Preparation
   A. The patient is placed in the modified lithotomy or split-leg position, allowing the operating surgeon to stand between the patient's legs. The table is placed in the reverse Trendelenburg position (i.e., the bottom of the table tilted down) so that intra-abdominal fat falls away from the esophageal hiatus. The video monitor is positioned at the patient's head so that the surgeon's body, the instruments, and the video screen form a straight line, to optimize the surgeon's spatial orientation.

   B. The sterile preparation is applied and should extend to the nipples superiorly, the pubis inferiorly, and the midaxillary lines laterally.

III. Port Placement
   A. A 10-mm port is placed 15 cm inferior to the xiphoid process (Fig. 7-2), just to the left of the midline. Either an open technique or a Veress needle technique may be used to access the peritoneal cavity; however, the former should always be used if the patient has had previous abdominal surgery. After port insertion, pneumoperitoneum is established with insufflation of CO₂.

   B. Four additional ports (see Fig. 7-2) are placed under laparoscopic visualization. The left and right subcostal ports, placed in the midclavicular lines, function as the primary operative ports and correspond to the surgeon's left and right hands, respectively. A lateral right subcostal port is placed to allow for the insertion of a self-retaining liver retractor, and a lateral left subcostal port provides access through which an assistant can retract.

IV. Exposure
   A. A liver retractor is inserted through the right lateral port and positioned to elevate the left lateral segment of the liver, exposing the esophageal hiatus.
B. The avascular portion of the gastrohepatic ligament (pars flaccida) is divided, exposing the right crus of the diaphragm. Care is taken to avoid injury to the hepatic branch of the vagus nerve or a replaced left hepatic artery arising from the left gastric artery.

C. The phrenoesophageal ligament is incised and attachments to the esophagus are swept down toward the gastroesophageal (GE) junction to allow for visualization of the left and right crura (Fig. 7-3). The anterior vagus nerve is identified and preserved.

D. Posterior attachments to the esophagus are dissected bluntly. The posterior vagus nerve is identified behind the esophagus and is preserved. Once the distal esophagus has been fully mobilized, it is encircled with a rubber drain (Penrose drain).

E. The short gastric vessels are divided from the mid-greater curvature to the angle of His to allow for mobilization of the fundus of the stomach and fundoplication (Fig. 7-4).

V. Crural Closure and Fundoplication
A. The right and left crura are approximated through placement of interrupted sutures to recreate a snug hiatus around the distal esophagus (Fig. 7-5).
B. The posterior fundus is passed posterior to the esophagus and retracted toward the patient’s right. A bougie (a flexible tube used to dilate the esophagus) is placed in the esophagus and a loose, 360-degree wrap is created around 2 to 3 cm of distal esophagus and secured with several interrupted permanent sutures (Fig. 7-6).

C. The bougie is removed and a nasogastric tube is inserted under laparoscopic visualization.

D. Fascial defects from ports greater than 5 mm in diameter are closed. All skin incisions are closed.

**POSTOPERATIVE COURSE**

A nasogastric tube is left in place overnight. Antiemetics are used liberally to prevent retching or vomiting, which can disrupt the wrap or crural repair. On the first postoperative day, the nasogastric tube is removed and a liquid diet is initiated. Patients may be advanced to a soft solid diet as tolerated but are instructed to avoid bread, meat, and raw
vegetables until after the first postoperative visit. In the absence of dysphagia, the diet can then be liberalized.

**COMPLICATIONS**

I. **Immediate complications** include esophageal or gastric perforation, pneumothorax, and splenic or liver trauma. Hollow viscus perforations are repaired intraoperatively if recognized. Delayed recognition of such injuries often results in significant morbidity. Early postoperative fevers, tachycardia, tachypnea, or abdominal tenderness should prompt evaluation with a gastrograffin swallow, and surgical exploration should be considered. Likewise, splenic or liver injuries are addressed intraoperatively when recognized and often require surgical exploration when diagnosed postoperatively. Pneumothorax usually results from passage of insufflated CO₂ into the pleural space and resolves without intervention.

II. **Early complications** include bloating (30%) and dysphagia (20%). Bloating results from an inability to reflux gas past the wrap or gastric dysfunction from vagal irritation and can be exacerbated by ileus. Dysphagia in the early postoperative period is generally due to edema of the distal esophagus and, in most cases, is self-limited. Persistent dysphagia, however, may reflect an overly tight wrap and sometimes necessitates surgical revision.

III. The most important **late complication** of fundoplication is recurrent symptomatic reflux. Although this may occur in the absence of a technical failure, the wrap should be evaluated with upper gastrointestinal fluoroscopy with barium. Reasons for failure include recurrent hiatal herniation resulting in an intrathoracic wrap, breakdown of the fundoplication, and herniation of the stomach cephalad to the wrap. All of these are best addressed with surgery, particularly when associated with symptoms.
LAPAROSCOPIC HELLER MYOTOMY

Case Study

A 45-year-old male with a history of a 10-pound weight loss over the previous 6 months presents for evaluation. He describes progressive difficulty swallowing over several years and intermittent regurgitation of foul-smelling undigested food. Additionally, he notes that he has always been a “slow eater,” and he often walks after meals to “help the food go down.” He appears thin, but otherwise, findings on physical examination are unremarkable. A barium swallow shows a dilated esophagus that tapers distally at the GE junction. Subsequent esophageal manometry reveals aperistalsis and an elevated LES resting pressure.

BACKGROUND

Achalasia is a rare disorder characterized by esophageal aperistalsis, high resting LES pressures, and failure of LES relaxation. Although the etiology of achalasia is unknown and is likely multifactorial, the pathogenesis involves loss of inhibitory ganglion cells in the esophageal body and LES. Typical symptoms include dysphagia and regurgitation; however, the onset of symptoms is often insidious. Early in the disease course, dysphagia is more pronounced with liquids than with solids. Patients with achalasia often eat slowly and contort their bodies to propel food through the esophagus. Ultimately, as esophageal dysfunction progresses, food cannot transit through the LES and is regurgitated. Malnutrition, weight loss, and recurrent pulmonary complications, such as pneumonia, bronchiectasis, and lung abscess, may ensue.

Surgical intervention should be considered for all patients with achalasia; however, two effective non-surgical therapies are available: pneumatic dilation and botulinum toxin injection. Pneumatic dilation involves passage of a balloon dilator to the level of the LES and disruption of the muscular fibers of the LES through balloon inflation under endoscopic guidance. The vast majority of patients experience symptomatic relief after balloon dilation, but it is frequently short-lived. Additionally, pneumatic dilation is associated with a 5% risk of esophageal perforation. Botulinum toxin inhibits acetylcholine release from the excitatory neurons in the LES, thereby reducing the resting pressure. Injection of botulinum toxin in the area of the LES results in symptomatic relief in the majority of patients, but like pneumatic dilation, this therapy is often transient. Generally, patients at lower surgical risk should be offered surgical myotomy or pneumatic dilation. Patients at high surgical risk should be offered botulinum toxin injection and medical therapies (e.g., calcium channel blockers) aimed at reducing LES pressure (Fig. 7-7).

INDICATIONS FOR SURGERY

The most durable method of reducing resting LES pressure in patients with achalasia is surgical esophagomyotomy. The procedure can be approached through the chest or abdomen. The transabdominal laparoscopic approach is most frequently used.

PREOPERATIVE EVALUATION

1. History and Physical Examination: Patients with achalasia typically report a history of progressive dysphagia to both solids and liquids as well as regurgitation. Weight loss is a late finding and indicates severe esophageal dysfunction. When pain is a prominent symptom, other diagnoses (e.g., esophageal spasm syndromes) should be considered. The presence of cervical adenopathy on physical examination suggests esophageal or GE junction neoplasia rather than achalasia in the patient with progressive dysphagia.
II. **Upper gastrointestinal fluoroscopy with barium** allows for the identification of mass lesions, dilation, tortuosity (*sigmoid esophagus*), air–fluid level, strictures, or tapering of the esophagus (Fig. 7-8). Esophageal aperistalsis, failure of LES relaxation, and abnormal progression of the food bolus may also be noted.

III. **Esophageal manometry** is required to confirm the diagnosis of achalasia and exclude other esophageal motility disorders associated with a similar constellation of symptoms, such as scleroderma and diffuse esophageal spasm. Manometric findings associated with achalasia include a resting LES pressure of greater than 45 mm Hg, aperistalsis, and failure of LES relaxation during swallowing (Fig. 7-9). The latter finding in particular distinguishes achalasia from other disorders that lead to aperistalsis of the esophageal body (e.g., scleroderma).

IV. **Upper endoscopy** is performed to exclude GE junction tumors, which may produce similar findings on manometry and barium studies to achalasia (*pseudo-achalasia*). Several other rare infiltrative processes can also result in pseudoachalasia and can be distinguished by histology. The presence of squamous cell carcinoma of the esophagus must also be excluded by endoscopy before esophagomyotomy, particularly in older patients with achalasia.

**COMPONENTS OF THE PROCEDURE AND APPLIED ANATOMY**

I. As noted in the “Indications for Surgery” section, the transabdominal laparoscopic approach to Heller myotomy is most frequently used today. Alternatively, Heller myotomy can be performed through a left thoracotomy. The latter approach has particular advantages in patients who have undergone extensive intra-abdominal surgery. Patients with achalasia are particularly vulnerable to reflux because the aperistaltic esophagus does not effectively clear gastric acid. Because Heller myotomy obliterates the antireflux barrier of the LES, most surgeons combine this operation with a fundoplication. A 360-degree fundoplication may result in functional obstruction of the esophagus in the patient with achalasia; a posterior 270-degree (Toupet) fundoplication is generally the preferred approach. Alternatively, an anterior 180-degree (Dor) fundoplication may be performed but provides less of a barrier against reflux. Preoperative considerations, patient positioning and preparation, port placement, and initial exposure proceed as described in the section on Nissen fundoplication.
Figure 7-8

Figure 7-9
Manometric findings recorded from the lower esophageal sphincter (LES) and the esophagus 3, 8, and 13 cm proximal to the LES in two patients with achalasia. Tracings at the different locations within the esophageal body are largely identical suggesting aperistalsis. The LES does not relax appropriately in response to a wet swallow (WS). (From Feldman M, Friedman LS, Brandt LJ, [eds]: Sleisenger and Fordtran’s Gastrointestinal and Liver Disease, 8th ed. Philadelphia, Saunders, 2006.)
Laparoscopic Nissen Fundoplication and Heller Myotomy

II. Myotomy
A. After mobilization of the distal esophagus, a point just proximal to the GE junction, on the anterior surface of the esophagus, to the left of the anterior vagus nerve, is chosen for the myotomy. Fibers of the outer longitudinal muscle layer of muscle are dissected, exposing the circular muscle fibers. These inner fibers are gently lifted away from the underlying mucosa and divided. The myotomy is extended 5 to 6 cm proximal to the GE junction and then across the GE junction and onto the stomach for 1 to 2 cm.

B. The mucosa along the length of the myotomy is inspected for injuries that, if identified, are immediately repaired. Mucosal injuries that are not initially apparent can be identified by instilling dilute methylene blue dye through the gastric tube while gently occluding the gastric lumen with a dissector. In the presence of a perforation, the blue dye will extravasate into the peritoneal cavity.

III. Crural Repair and Fundoplication
A. The right and left crura are approximated through placement of interrupted sutures to recreate a snug hiatus around the distal esophagus.

B. Posterior (Toupet) fundoplication is performed by delivering the fundus of the stomach behind the esophagus from left to right. The fundus is secured to the diaphragmatic crura and the free edges of the myotomy on both sides with interrupted sutures. This buttresses the GE junction posteriorly and helps to maintain the myotomy.

C. Anterior (Dor) fundoplication is performed by wrapping the fundus around the anterior esophagus, directly over the myotomy and exposed mucosa. As in the Toupet fundoplication, the fundus is secured to the edges of the myotomy and crura. Dor fundoplication can be used to reinforce a mucosal repair if a perforation resulted from the dissection.

D. Fascial defects from ports larger than 5 mm in diameter are closed. All skin incisions are closed.

POSTOPERATIVE COURSE
The nasogastric tube is left in place overnight. On the first postoperative day, a barium swallow is performed to exclude perforation. If the findings are normal, the nasogastric tube is removed and the patient's diet is advanced slowly to thickened liquids. Patients are typically discharged on the second or third postoperative day. In the absence of dysphagia, the patient's diet may be liberalized after the first postoperative visit (typically 2 weeks after surgery). Routine endoscopic surveillance is recommended for all patients with achalasia because of the associated increased incidence of esophageal cancer.

COMPLICATIONS
I. Immediate complications include esophageal or gastric perforation, pneumothorax, and splenic or liver trauma (see discussion in the section on Nissen fundoplication). The incidence of esophageal perforation is higher during Heller myotomy.

II. Late complications of Heller myotomy include dysphagia and gastroesophageal reflux.

A. Dysphagia that persists beyond 2 weeks after surgery should prompt investigation with a barium swallow and endoscopy. Causes of persistent dysphagia include incomplete myotomy and an overly tight fundoplication. The former may be addressed with pneumatic dilation or reoperation, whereas the latter usually requires reoperation. A very small percentage of patients still have a poor quality of life despite myotomy. This is often a result of poor esophageal motility from long-standing, progressive disease. In these patients, an esophagectomy with gastric pull-up or colonic interposition may be considered as a last option. Onset of dysphagia after a symptom-free period should raise
concern for the development of an esophageal malignancy, peptic stricture, or paraesophageal hernia.

B. **Gastroesophageal reflux**, both symptomatic and asymptomatic, is common after myotomy. Symptoms are usually well controlled with pharmacologic acid suppression. Dietary modification and sleeping at a 45-degree angle to enhance nighttime emptying of the esophagus may also improve symptoms.

**SUGGESTED READINGS**


