IS THERE A GOLD STANDARD TREATMENT FOR GASTROESOPHAGEAL REFLUX DISEASE?

A gold standard is a model of highest excellence or perfection; in medicine, it refers to an ideal treatment or procedure that is definitive and curative, with perfect short- and long-term outcome. Medical therapy with proton pump inhibitors (PPIs) is 80% to 90% effective in long-term control of symptoms of gastroesophageal reflux disease (GERD). However, 10% to 20% of patients are refractory to this extended and expensive therapy.1,2 Current surgical therapy for GERD creates an obligate gastric volvulus, which also may be 80% to 90% effective in controlling symptoms,1,3,4 but can produce new, and not always transient, symptoms.

As with all hernia surgery, GERD surgery has a tendency to disrupt, fostered by the transitional environment between thorax and abdomen, resulting in breakdown of the surgical repair, return of the stomach to its herniated state, and reappearance of GERD. Thereafter, reoperation produces exponentially poorer outcomes with each successive surgery, culminating in an irreparable problem. Furthermore, surgery for GERD is irreversible, and if incorrectly prescribed or performed, it becomes a disease itself. Therefore, neither medical nor surgical therapies are definitive, perfect, or appropriate for all patients; rather, they are standard therapies for comparison but not gold standards.

Laparoscopic surgery for GERD has been incorrectly characterized as gold standard therapy.5–16 After first being reported in 1991,17 it was rapidly adopted and
indiscriminately used in a broad spectrum of patients who were believed, correctly or incorrectly, to have GERD. Lessons previously learned from open surgery were ignored, forgotten, or unrecognized, only to be painfully relearned at patient expense, physicians’ professional reputations, and the status of GERD surgery. Declining use of surgery for GERD is evidence of an end to the laparoscopic frenzy and a return to appropriate use of both open and laparoscopic surgery in treating GERD.

Recognizing that no current therapy is a gold standard, an orderly exploration of definition, pathophysiology, natural history, and diagnosis and investigation of GERD, plus a review of operative techniques, postoperative care, special considerations, and surgical results, are necessary to elucidate the indications for surgical management of GERD.

DEFINITION

GERD is defined as either symptoms or mucosal damage produced by abnormal reflux of gastric contents into the esophagus. This definition broadly and imprecisely characterizes the disease, setting the stage for inappropriate or unsuccessful therapy: neither presence of symptoms nor mucosal damage are rigorously classified or measured. Causation by reflux of gastric contents is not quantified beyond being abnormal, and means of assessment are not stated. This unusual and vague definition allows a wide spectrum of disorders to fall under one diagnosis and fosters misdiagnosis. The authors propose a more rigorous definition when surgery is considered for managing GERD: GERD is documented abnormal reflux of gastric contents into the esophagus, producing proven reflux-related symptoms or confirmed mucosal damage.

PATHOPHYSIOLOGY

GERD results from failure of the reflux barrier. This barrier has three components: (1) an intra-abdominal esophagus of adequate length, (2) an extrinsic sphincter, the esophageal hiatus, and (3) an intrinsic sphincter, the lower esophageal sphincter. Loss or breakdown of any of these elements, but typically all three, results in GERD. Esophageal and gastric dysfunction independent of the reflux barrier may rarely cause GERD, but more commonly increases esophageal injury caused by reflux barrier failure. Upstream from the reflux barrier, esophageal dysmotility, which is sometimes secondary to GERD, inadequately clears gastric refluxate, heightening esophageal injury. Downstream from the reflux barrier, abnormal or delayed gastric emptying may overcome a marginal reflux barrier.

Epithelial and extramucosal mechanisms protect the esophageal mucosa from physiologic gastroesophageal reflux. Pathologic refluxate, either in amount or composition, overcomes these protective mechanisms, producing a characteristic but nonspecific injury of the esophageal mucosa. Microscopic review of reflux-induced esophagitis shows squamous hyperplasia and inflammatory infiltration of the mucosa by neutrophils, eosinophils, and lymphocytes. As injury progresses, mucosal erosion and eventual ulceration occurs. The reparative process may produce fibrous strictures. Metaplastic response to injury replaces esophageal squamous epithelium with specialized columnar epithelium (Barrett esophagus).

NATURAL HISTORY

GERD is chronic and relapsing. Because of its broad definition, it includes a wide spectrum of disease, from mildly symptomatic with no mucosal injury to irreversible esophageal damage regardless of symptoms, produced by scarring or malignant
degeneration. In the United States, a 10,000-fold spread is present between the approximately 100 million patients who have at least weekly GERD symptoms to the tens of thousands of patients who have end-stage mucosal damage.

Although the natural history of the disease is poorly investigated and understood, it tends to persist and worsen with time. Unfortunately, which patients will develop unrelenting or uncontrollable symptoms or complications is unknown. Mortality is negligible for uncomplicated GERD, and small but significant for complicated GERD. Surgery for GERD remains controversial because the natural history of the disease is extremely variable, although progressive and relapsing.

**DIAGNOSIS AND INVESTIGATION**

**Diagnosis**

**Symptoms**

GERD produces specific symptoms. A detailed history must be obtained from any patient who has suspected GERD, particularly one being considered for surgery. Its importance in decision making cannot be underestimated or overlooked. Typical GERD symptoms—heartburn and regurgitation—reflect dysfunction of the reflux barrier. Heartburn responding to aggressive PPI therapy is considered diagnostic of GERD. It is necessary to differentiate (1) heartburn (acid reflux) from regurgitation of stagnant, fermented food and saliva from an obstructed esophagus, such as occurs with achalasia, and (2) regurgitation (passive) from vomiting (active). Dysphagia is a third, less-specific, GERD symptom. It may be caused by GERD itself or stricture complicating GERD. GERD-related dysphagia must be differentiated from functional and mechanical dysphagia resulting from multiple other diseases that cause symptomatic esophageal obstruction.

Atypical GERD symptoms are cough, asthma, laryngitis, sore throat, chest pain, abdominal pain, and bloating. These symptoms in the absence of typical GERD symptoms point to diseases other than GERD. Careful investigation of alternative causes of atypical symptoms is necessary. Sophisticated testing, including impedance/pH monitoring, must be performed if GERD is believed to be the cause of atypical symptoms and surgery is being considered.

**Mucosal injury**

GERD may also produce esophageal injury. Esophagogastroduodenoscopy (EGD) with biopsy has replaced the upright air-contrast phase of the barium esophagram for mucosa evaluation. EGD and biopsy both diagnose and assess esophageal injury by visual and histopathologic mucosal examination. Visual assessment of esophageal injury is graded using the Los Angeles classification. Histopathologic findings, although nonspecific, are confirmatory in the clinical setting of GERD. The finding of specialized columnar epithelium (Barrett esophagus) in the tubular esophagus is secondary to GERD. In the absence of dysplasia, surveillance esophagoscopy and biopsy are required in patients who have Barrett esophagus, regardless of therapy.

EGD should be performed by the surgeon before surgery. The finding of a hiatal hernia identifies failure of two elements of the reflux barrier: loss of intra-abdominal esophagus and extrinsic sphincter. The following must be noted:

- Measurements from incisor teeth to squamocolumnar junction, gastric rugal folds, and diaphragmatic hiatus
- Length of hiatal hernia
- Length of esophagus
- Type of hiatal hernia
Presence of volvulus of the intrathoracic stomach
Mucosal abnormalities (strictures or rings)
Mural abnormalities (eg, submucosal tumors, leiomyoma)

Gastroesophageal reflux
The definition of GERD requires causation of symptoms or complications by abnormal reflux of gastric contents into the esophagus. Ambulatory pH monitoring performed off-medication both quantifies acid reflux and relates symptoms to acid exposure. It has evolved from an in-hospital test to an ambulatory wireless 48-hour study. Once reserved for diagnostic dilemmas, it is now essential before any proposed operation. It is invaluable in diagnosing GERD and documenting the preoperative state for later comparison.

Excessive acid exposure on pH testing is a surrogate for reflux of gastric contents into the esophagus, and in most patients it is adequate to diagnose GERD. In uncommon patients in whom duodenal reflux must be confirmed and quantified, ambulatory bilirubin monitoring is required. Similarly, in patients in whom non–acid reflux must be assessed, combined impedance and pH monitoring is necessary.

Investigations
The preoperative barium esophagram has been neglected, misused, and in some cases abandoned, with the advent of modern investigations of GERD. However, it provides valuable information about the mucosa, esophageal complications, reflux of gastric contents, reflux barrier, and esophageal function. It should, whenever possible, be ordered by the surgeon and performed by a radiologist who is experienced in preoperative assessment of GERD and is a member of the multidisciplinary treatment team.

If dysphagia is the predominant symptom and the diagnosis is in question, the examination should start as a timed barium esophagram. If not, the first phase of the barium esophagram is the upright air-contrast examination. Intended to evaluate esophageal mucosa for esophagitis, it is more importantly an excellent assessment of esophageal length and thus useful in diagnosing short esophagus. The second phase is a motility examination, performed with five sequential swallows separated by at least 30 seconds and video recorded. The third phase is the distended single-contrast examination, which identifies small reducible hiatal hernias, subtle strictures, rings, and other abnormalities of esophageal contour. The fourth phase involves provocative measures to identify reflux. The fifth phase is a solid-food examination using, for example, a barium tablet or barium-coated marshmallow to identify areas of obstruction. The final phase is the oropharyngeal examination, but this is not critical in patients who have GERD without cervical dysphagia. The barium esophagram report should describe each element of the examination:

1. Emptying assessment in patients who have dysphagia (timed barium esophagram)
2. Mucosal assessment
3. Assessment of esophageal length
4. Assessment of esophageal motility
5. Presence and type of hiatal hernia
6. Reducibility of hiatal hernia in upright position
7. Identification of reflux, including maneuver, height of reflux, and time to clear reflux
8. Presence of stricture or ring

Esophageal manometry excludes unsuspected motility disorders or motility disorders masquerading as GERD, confirms adequate esophageal peristalsis for GERD.
surgery, and quantifies preoperative resting pressure and relaxation of the lower esophageal sphincter for later comparison. High-resolution manometry is replacing conventional manometry because it provides a spatially enhanced dynamic representation of the esophageal body and reflux barrier. It isolates the esophageal hiatus from the lower esophageal sphincter (LES), increasing understanding of GERD and facilitating treatment decision-making. For modern GERD surgery, high-resolution manometry is invaluable and highly recommended.

If gastric emptying abnormalities are suspected by history or investigations, radio-nucleotide gastric emptying studies are warranted.

**SURGICAL TECHNIQUE**

Surgery for GERD rebuilds the three components of the reflux barrier. This requires three steps to address each main component.

**Restoration of Intra-Abdominal Esophagus**

Regardless of approach, the first step commences with dissection of the esophageal hiatus, which indirectly mobilizes the distal esophagus, esophagogastric junction, and gastric fundus. Hiatal dissection then permits intrathoracic mobilization of the esophagus into the posterior mediastinum, at least to the level of the inferior pulmonary veins. In many patients, this is sufficient to restore an adequate length of intra-abdominal esophagus. However, in certain patients, such as those who have the much-disputed diagnosis of short esophagus, this dissection alone is inadequate.

Short esophagus should be diagnosed preoperatively. It is suspected in patients who have a history of peptic stricture or repeated esophageal dilatation, long-segment Barrett esophagus, type I hiatal hernia (sliding) more than 4 cm long, type III hiatal hernia (rolling, giant paraesophageal), or nonreducible hiatal hernia on upright air-contrast barium esophagram (Fig. 1). In these patients, adequate intra-abdominal length is obtained

![Fig. 1. Short esophagus. (A) Preoperative single-contrast phase of barium esophagram in an 83-year-old woman who has typical GERD refractory to escalating PPI therapy and a symptomatic type III hiatal hernia with organoaxial volvulus (not discussed in this article). (B and C) Emergency esophagram 2 days after laparoscopic GERD surgery. Extensive esophageal dissection and excessive intraoperative traction applied to the esophagus provided adequate intra-abdominal length to her short esophagus. However, this was obtained by unrecognized degloving injury to the muscularis propria in the lower thoracic esophagus. The resultant leak (arrows) from the denuded esophageal submucosa necessitated emergency esophagectomy. The patient, who was deemed too old and ill for open antireflux surgery, is alive and well 3 years postoperatively.](image)
by adding a Collis gastroplasty. This procedure begins with dissection of the esophagastric fat pad, which selectively vagotomizes the gastroplasty segment. A tube of stomach 3 to 6 cm in length is constructed along its lesser curve using surgical staplers. With adoption of laparoscopy, esophageal lengthening has evolved into a simple wedge gastroplasty, because of technical difficulties presented by laparoscopy and misunderstanding of the principles of constructing a Collis gastroplasty. Predictably, acid production in this unprepared gastric segment perpetuates GERD.

Failure to restore adequate intra-abdominal esophageal length produces a repair under tension that will eventually fail. In patients for whom GERD surgery failed, review of the operative report may identify inadequate restoration of the intra-abdominal esophagus as a reason for failure of the initial surgery.

**Reconstruction of Extrinsic Sphincter**

The esophageal hiatus, which serves as the extrinsic sphincter, is composed of a right and left crus arising from the right crus of the diaphragm (Fig. 2). The right crus of the esophageal hiatus is nearly vertical and lies over the vertebrae, whereas the left crus of the esophageal hiatus is slightly more semicircular, slightly longer, and has no underlying support. Progressive herniation of the stomach into the posterior mediastinum results in minimal change of the right crus but bowing and further elongation of the left crus (Fig. 3).

Suture closure of the esophageal hiatus to approximate its normal size is the essence of hiatal reconstruction. Deep suture bites into each crus, with slightly wider spacing on the left crus, constitutes standard reconstruction (Fig. 4). For severely disrupted hiatus, a complex reconstruction is required. The left crus is plicated to normalize crural length, permitting a standard hiatal reconstruction (Fig. 5).

The importance of hiatal reconstruction was not appreciated or stressed in the early laparoscopic experience (Fig. 6). Recurrent hiatal hernia was initially the most common cause of failure with laparoscopic GERD surgery. Attempts to solve this problem led to reinforcing of marginal hiatal reconstructions or primary reconstruction of the esophageal hiatus with prosthetic mesh (Fig. 7). This strategy presents three problems: (1) it treats the hiatus as a hole to be covered, ignoring its function in the reflux barrier; (2) it disregards the dynamic nature of the reflux barrier, resulting in the disastrous complication of mesh erosion into the gastrointestinal tract (see Fig. 7); and (3) it obstructs if applied aggressively (see Fig. 7). Oelschlager and colleagues reported that using biosynthetic mesh to reinforce hiatal reconstruction reduced recurrence in repair of type III

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**Fig. 2.** Esophageal hiatus. The esophageal hiatus lies to the left of the patient’s inferior vena cava and above and to the right of the aorta. It arises from the right diaphragmatic crus and is composed of a right and left crus. The right crus of the esophageal hiatus is nearly vertical and lies over the vertebrae, and the left crus is slightly more semicircular and longer, and has no underlying support. (Courtesy of the Cleveland Clinic Foundation, Cleveland, Ohio.)
Reinforcement of Intrinsic Sphincter

The most problematic, variably conducted, and operator-dependent step in GERD surgery is fundoplication for reinforcement of the lower esophageal sphincter. The prior steps attempt to restore and reconstruct; however, reinforcing the lower esophageal sphincter produces an unnatural and potentially problematic volvulus of the gastric fundus. Typically in North America, this involves division of short gastric vessels and a 360° total fundoplication (Nissen) (Fig. 8). Although controversial and cited as a cause of postoperative dysphagia,33 for accomplished surgeons, constructing a fundoplication without division of short gastric vessels (Rossetti modification) can produce results similar to those reported with division of these vessels.34

Similarly, agreement on the extent of fundoplication has not been reached. Some surgeons, mostly outside the United States, have favored 270° partial fundoplication, citing less dysphagia, fewer postprandial fundoplication symptoms, and similar reflux control compared with total fundoplication. In the United States, the general feeling is that total fundoplication provides better reflux control with more but transient symptoms. Experts also disagree on the practice of matching esophageal motility to extent

Fig. 3. The left crus. (A) Abdominal forces (large blue arrow) acting on the left crus. These forces stretch and elongate the left crus and displace it to the left and posterior (small blue arrows). (Courtesy of the Cleveland Clinic Foundation, Cleveland, Ohio.) (B) Intraoperative photograph of dissected esophageal hiatus. The esophagus is seen between the surgeon’s fingers. A black dot has been placed on the caudate lobe of the liver. The right crus (white arrow) is of normal length. The left crus (yellow arrow) is stretched and deformed. (C) The true length of this left crus is shown through light lateral traction. Left lung (white dot) is seen in this dissection.

hiatal hernia from 24% to 9% at 6 months postoperatively. The manner in which this study was conducted and the analysis performed raise the question of whether these recurrence rates are different.32 Regardless, at 6 months neither result is acceptable.
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Fig. 4. Standard reconstruction of esophageal hiatus. Nonabsorbable sutures are placed with deep bites into each crus, avoiding the inferior vena cava on the patient’s right and aorta on the patient’s left. Unequal spacing of sutures is required—normal distance between sutures in the left crus, slightly smaller distance between sutures in the right crus. This placement shortens the minimally elongated left crus. A sufficient number of sutures are placed to return the hiatus to its original, normal size. The sutures are tied, completing the reconstruction. (Courtesy of the Cleveland Clinic Foundation, Cleveland, Ohio.)

of fundoplication, reserving partial fundoplication for patients who have “poor” motility. However, in all but the aperistaltic esophagus, a correctly constructed total fundoplication is well tolerated. The debates continue, but these issues illustrate how surgeon dependent and subjective this portion of the operation can be.

Components of fundoplication (length, looseness, floppiness, position, and extent of fundoplication) must be meticulously, precisely, and repeatedly constructed despite variable gastric dimensions and anatomy. The potential for error and postfundoplication problems is enormous (Figs. 9 and 10).

SPECIAL FEATURES OF POSTOPERATIVE CARE

Vomiting in the early postoperative period and abdominal stressors, such as weight lifting, have been associated with failure of GERD surgery. Preemptive, preventive
antiemetic therapy and aggressive management of vomiting starting in the operating room are necessary to minimize early failure. In terms of abdominal stressors, laparoscopy is a double-edged sword. Desirably short postoperative recovery and early return to work increase the possibility of early abdominal stressors. Patients, particularly those who have jobs requiring heavy lifting, should be cautioned and perhaps return to work delayed if abdominal stressors cannot be avoided.

Postoperative diet is typically liquid and soft foods in the first weeks of recovery. Dysphagia is a common occurrence, and patients must be warned of this expected problem and instructed to eat slowly, chew thoroughly, and swallow carefully. This problem is usually transient, and early dilatation is unnecessary and can disrupt the repair.

Fig. 5. Complex reconstruction of the severely disrupted hiatus. The massively elongated left crus is plicated to normalize crural length. A standard hiatal reconstruction is then possible between the crura of equal length. (Courtesy of the Cleveland Clinic Foundation, Cleveland, Ohio.)
RESULTS

Outcome assessment of therapy for GERD should be driven by its definition and effect on lifestyle. Therefore, symptom control, reversal and prevention of mucosal injury, prevention of gastroesophageal reflux, and impact of treatment on lifestyle should be evaluated. To determine best evidence-based indications for surgery, a randomized trial is necessary to compare outcomes of similar patients treated medically and surgically.

Randomized Trials

A randomized controlled study reported by Spechler and colleagues may be dismissed today because it compared surgery with H-2 blocker medical therapy. However, it provides an excellent picture of the results of surgery. Follow-up at a mean of 10.6 years showed that 63% of patients treated surgically were taking antireflux medication, compared with 92% of patients treated medically. Symptom control on medication was no different between groups; however, symptom control off medication was significantly worse for surgical patients. No difference was seen between the groups in grade of esophagitis, frequency of treatment of esophageal stricture, subsequent surgery, quality of life (SF-36 survey), and overall satisfaction with therapy. For the surgical group, 16% required at least one reoperation and 14% treatment of esophageal stricture. The authors concluded that “antireflux surgery should not be advised with the expectation that the patient with GERD will no longer need to take antisecretory medication.” Since this study was conducted in the late 1990s, more effective medical therapy has become available; the same cannot be said for surgery.

Fig. 6. Failure to reconstruct the esophageal hiatus. (A) Posteroanterior view of barium upper gastrointestinal study 6 weeks after laparoscopic GERD surgery without reconstruction of the esophageal hiatus, showing herniation of the entire stomach into the mediastinum. (B) Oblique view of barium upper gastrointestinal study shows organoaxial volvulus of the entire intrathoracic stomach.
Lundell and colleagues\textsuperscript{1} conducted a randomized study comparing surgery with PPI therapy in GERD. Treatment failure was the outcome measure and was defined as (1) moderate-to-severe heartburn or acid regurgitation in the 7 days before assessment, (2) at least grade 2 esophagitis, (3) moderate or severe dysphagia or odynophagia in combination with mild heartburn or regurgitation more than 3 months after operation, or (4) reoperation or PPI required more than 8 weeks for symptom control or consideration or request for surgery by a physician or patient. Significantly more treatment failures occurred in the medical group; however, with dose escalation of PPI this difference became nonsignificant. Quality of life was similar and within normal range for both therapies.

\textbf{Fig. 7.} Complications of mesh reinforcement of esophageal reconstruction. (A) Synthetic mesh reinforcement of a laparoscopic hiatal reconstruction was complicated by redundant mesh being incorporated into the upper half of the stomach (arrows). At reoperation for failed GERD surgery and obstruction secondary to mesh, reconstruction was not possible and a distal esophagectomy and total gastrectomy were required. (B) Polytetrafluoroethylene (PTFE) strip reinforcement of hiatal reconstruction was complicated by erosion into the esophagogastric junction. PTFE strips, sutures, and titanium clips are seen in the esophageal lumen at esophagoscopy. (C) Biosynthetic mesh reinforcement of a laparoscopic hiatal reconstruction was complicated by obstruction. A barium tablet could not pass through the repair. Spiral tacks (arrows) used to affix the mesh to the hiatus are visible. At reoperation, resection (thoracic esophagectomy and proximal gastrectomy) was necessary.
In a study of laparoscopic Nissen fundoplication and PPI therapy, Mehta and colleagues randomized 91 patients to surgery and 92 to optimized (dose adjusted to abolish symptoms) PPI therapy. At 12 months, those randomized to PPI therapy were offered surgery; 54 had it (crossover group) and 38 did not. In the surgical group, 2 required early reoperation for failed surgery and 4 required dilatation for dysphagia in the first 3 months. Of patients who underwent PPI therapy, 10% experienced side effects of therapy (headache, diarrhea, vomiting, abdominal pain) requiring altering therapy, and 18% required dose escalation during the trial. All three groups had significant improvement in their symptom scores at 12 months. At long-term follow-up (median 6.9 years, range 4.3–8.3 years), patients crossing over to surgery experienced further symptomatic improvement; however, symptom scores were not different among the groups. The authors concluded that “both optimal PPI therapy and laparoscopic Nissen fundoplication are effective treatments for GERD, however, surgery offers additional benefits for those who have only partial symptomatic relief with PPI therapy.”

**Nonrandomized Trials**

An interesting query of the Veterans Administration database identified 5064 patients who had erosive esophagitis, 542 who had ulcers and strictures, and 605 who had no ulcers and strictures but had Nissen fundoplication. At a mean follow-up of 4.2 years in the patients who had ulcer or stricture, surgery showed better results than nonsurgical therapy, with esophagitis reported in 46% versus 56% ($P<.001$), ulcers in 33% versus 38% ($P<.05$), and strictures in 32% versus 43% ($P<.001$), respectively. The clinical significance of these statistically significant improvements is questionable. In patients who had nonerosive esophagitis who underwent surgery or nonsurgical

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**Fig. 8.** Nissen fundoplication. Dividing the short gastric vessels is advisable to facilitate fundoplication. The fundus is passed posteriorly to the esophagus to lie on the patient’s right. Maintaining the correct orientation of the stomach is crucial. Sutures are passed through the greater curve aspect of the rightward lying fundus, then through anterior muscularis propria of the esophagus, and finally through the greater curve aspect of the leftward-lying fundus. Three sutures are usually used to construct a fundoplication 2 cm in length (measured anteriorly). (Courtesy of the Cleveland Clinic Foundation, Cleveland, Ohio.)
therapy, no difference was seen in esophagitis (24% versus 25%). However, in the surgical group who had no strictures or ulcers, more dysphagia, 4.6% versus 2.6% ($P$ not given), outpatient visits (40 versus 34; $P < .05$), and outpatient procedures (4.3 versus 2.7; $P < .01$) were seen. No difference was seen in the patients who had strictures or ulcers. Repeat surgery was required in 2.3% of surgical patients who had no ulcers and strictures and in 5.1% of those who did.

Most single-center surgical series reporting the results of laparoscopic surgery for GERD focus only on the subjective aspects of symptom control and ask the leading questions, “How pleased are you with the surgery?” and “Would you have this

Fig. 9. Some fundoplication mistakes. Twisted fundoplication occurs when the gastric fundus is allowed to rotate as it passes behind the esophagus (top left). Tight fundoplication occurs by placing sutures inside the greater curve boundaries of the fundus (top right). Slipped fundoplication occurs in two instances: (1) the anterior esophagogastric junction is not cleared of the esophagogastric fat pad and the fundoplication is inadvertently constructed on the proximal stomach, or (2) the fundoplication is not anchored to the anterior esophageal wall, allowing the fundoplication to “slip” onto the stomach (bottom left). Multiple errors can occur, such as a tight, slipped fundoplication (bottom right). (Courtesy of the Cleveland Clinic Foundation, Cleveland, Ohio.)
Fig. 10. Atypical symptoms. A 37-year-old woman who has cough and bronchitis not responsive to PPI therapy, no typical GERD symptoms, normal pH monitoring, and inducible reflux on barium esophagram underwent laparoscopic GERD surgery with total fundoplication. At 14 months postoperatively, her cough persists, she lost 40 pounds, and she complains of dysphagia and postprandial bloating. (A) A postoperative single-contrast phase of barium esophagram shows a relatively long, tight fundoplication that wraps the stomach. (B) An upright abdominal film the day of the barium study and immediately after a meal shows normal transit of the barium into the small bowel and a markedly distended, gas- and fluid-filled stomach. She was experiencing her disturbing postprandial symptoms during this examination.

operation again?” Most are small series with short follow-up. Despite millions of operations, outcome has been reported in only a fraction of 1% of these patients, usually retrospectively, from high-volume specialty centers and by the surgeons themselves, bringing into question the validity of the analysis and its worth in evaluating outcome.

Two reports have greater than 10-year follow-up. Reporting from a center that had performed 1528 operations, Dallemagne and colleagues3 followed up 100 patients who in 1993 underwent laparoscopic fundoplication performed by one experienced surgeon who had completed more than 100 operations. At 10 years and among 90 patients (7 patients died, 3 were lost to follow-up), 5 had heartburn requiring therapy, 15 had occasional regurgitation, 29 had occasional dysphagia, and 4 required reoperation. At 5-year follow-up, one third of the patients who underwent partial fundoplication and none of those who underwent total fundoplication experienced herniation of their fundoplication into the chest at barium esophagram.

In a 10-year or more follow-up of 226 of 250 patients (21 patients had died, 3 were lost to follow-up), Kelley and colleagues4 reported that 84% had good to excellent control of heartburn and 83% were highly satisfied with the outcome; however, 21% required antireflux medication and 17% underwent reoperation.

Two reports have been published of GERD surgery in community-based practices. In 45 patients undergoing open GERD followed up for a mean of 78 months, Rantanen and associates40 reported that 85% were free of or had mild reflux, 31% experienced dysphagia, 67% flatulence, and 46% bloating. Of 35 who had esophagoscopy, 37% had a defective fundoplication and 29% erosive esophagitis; 13% required antireflux medication and 13% were scheduled for reoperation. Two patients underwent reoperation in the follow-up period. The frequency of GERD surgery for six surgeons ranged from 0.08 to 1.8 operations per year.
In 80 patients undergoing laparoscopic GERD surgery followed up for a mean of 20 months, Vakil and colleagues\textsuperscript{41} reported that 61% were satisfied with their outcome, 32% required PPI medication, 11% experienced dilatation, 7% underwent reoperation, 67% had new symptoms, 48% experienced gas, 13% bloating, and 13% dysphagia.

**Predictors of Outcome**

Reported predictors of outcome have been obtained for the most part from single-institution, retrospective data. There are too many of these studies to review in the space allowed. Predictably, there is considerable disagreement among these reports. However, in keeping with the surgical definition proposed, typical GERD symptoms accompanied by positive response of heartburn to aggressive medical management with PPI therapy has been reported to be an excellent predictor of successful antireflux surgery.\textsuperscript{42–44} Abnormal pH monitoring is predictive of successful outcome after surgery for GERD.\textsuperscript{42}

**INDICATIONS**

Based on these results, and to avoid pitfalls of the recent past, surgery for GERD must be used in highly selected patients to treat specific symptoms and reverse or halt documented, severe mucosal damage resulting from quantified abnormal reflux of gastric contents into the esophagus. Surgery for GERD should be considered only if (1) a trial of aggressive medical treatment using PPI with dose escalation and lifestyle modifications failed, (2) mucosal damage was identified and quantified, (3) abnormal gastroesophageal reflux was documented, and (4) a repairable problem in the reflux barrier was found.

**Symptom Control**

Heartburn is the main symptom indication for GERD surgery. Three typical clinical scenarios exist: (1) heartburn initially controlled with PPI therapy that has become refractory or is poorly controlled despite dose escalation, (2) heartburn is well controlled, but side effects of PPI therapy are intolerable, and (3) volume regurgitation despite effective heartburn control. GERD-related dysphagia in these scenarios may not be as effectively treated with surgery but is an indication nonetheless. Physicians should beware of patients who have typical symptoms that do not respond to medication or those demanding immediate surgery for relief of intolerable symptoms, and those who have scleroderma. Age itself should not change these indications; need for lifelong medical therapy in young patients who have well-controlled symptoms is an indication for GERD surgery only if its durability can be assured.

Atypical symptoms should be associated with typical GERD symptoms and responsive to PPI therapy for surgery to be considered. If symptoms are only atypical, they must be proven to be GERD-related and responsive to PPI therapy or shown to be the result of acid reflux before surgery is prescribed.

**Mucosal Injury**

Poorly controlled or recurrent ulcerative esophagitis after aggressive PPI therapy is an indication for surgery. Others causes for lack of healing, such as pill-induced injury, must be ruled out. Most strictures can be managed initially with medical therapy and dilatation. As with esophagitis, unsuccessful medical therapy or recurrent strictures despite effective medical therapy are indications for surgery. The ability of any therapy to completely reverse Barrett esophagus or prevent its progression to cancer...
has not been shown; therefore, GERD surgery is not indicated in patients who have GERD with Barrett esophagus for prevention of cancer.

**SPECIAL SITUATIONS**

**Obesity**

An ignored but essential part of physical examination is measuring and recording weight and height and calculating body mass index (BMI). Overweight (BMI 25–29) and obese (BMI 30–34) patients who have GERD should be counseled on weight loss and encouraged to reach their ideal weight before elective surgery. Because obesity and GERD are interrelated, successful and sustained weight loss may eliminate need for surgery. Although disagreement exists concerning impact of obesity on outcome of antireflux surgery, health benefits of weight loss in severely (BMI 35–39) and morbidly (BMI ≥ 40) obese patients who have GERD should make weight loss surgery the preferred operation in these patients.

**SUMMARY**

Managing GERD is difficult because it is a chronic relapsing disease. Surgical management is indicated only after medical management has failed to control symptoms or when mucosal injury persists (esophagitis, ulcer, and stricture). In patients who have the most advanced forms of GERD, surgical therapy is good but far from a gold standard. The patient must be aware that once operation has been performed, heartburn and regurgitation should be controlled but other symptoms may not; freedom from medical therapy of GERD is not assured; side effects such as dysphagia, bloating, and inability to belch are possible; and reoperation may be necessary. Further operations are less successful, and the path of multiple reoperations leads to resection (esophagectomy/gastrectomy). Care must be taken when patients are placed on this path, and it is best that an experienced surgeon at a specialty center participate in the patient’s lifelong care.

**REFERENCES**


