

# Laparoscopic Surgery of the Gastroesophageal Junction

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Abstract. Incompetence of the lower esophageal sphincter mechanism leads to gastroesophageal reflux (GER), which is the most common indication for surgery of the gastroesophageal junction. Evaluation, diagnosis, and the modern surgical treatment of GER are discussed. Evaluation of patients with severe heartburn include upper endoscopy to evaluate the general condition of the esophagus, stomach, and duodenum; an upper gastrointestinal contrast study for a complete anatomic view of the esophagus and stomach; esophageal manometry to evaluate the function of the esophagus; 24-hour pH monitoring to determine esophageal acid exposure; and a gastric emptying study selectively to determine the presence of a motility disorder. These studies most often prove the diagnosis of gastroesophageal reflux, hiatal hernia, Barrett's esophagus, peptic esophageal stricture, paraesophageal hernia, or achalasia. The laparoscopic approach to treatments for these include Nissen fundoplication, Toupet fundoplication, Collis gastroplasty with fundoplication, modified Heller myotomy, esophageal diverticulectomy, and revisional operations. These procedures are described in detail. The results of these operations indicate that they are safe and effective and should be considered the new gold standard for correction of gastroesophageal pathology. Laparoscopic surgery has revolutionized many procedures traditionally performed through a laparotomy. Although they are technically more difficult and require a significant amount of time and practice for the surgeon to become proficient, it is becoming apparent that for functional surgery of the gastroesophageal junction laparoscopy is the access of choice.

The gastroesophageal junction (GEJ) is bounded anatomically by two diaphragmatic crura, which insert into the anterolateral plane of the first three or four lumbar vertebrae and ribs. Surrounding the GEJ like a wide, strong collar is the phrenoesophageal membrane, a fibroelastic layer that consists of subdiaphragmatic and diaphragmatic endothoracic aponeuroses. Under normal conditions this arrangement shapes the hiatus and encircles the GEJ at a position just at the border of the thorax and abdomen. A thin, loose connection by underlying areolar connective tissue allows the GEJ to move freely in relation to the diaphragm and slip through the hiatus as in a tendon sheath [1]. As a result of a continuous process of wear and tear, slacking of these ligaments and membranous gastric attachments can result in protrusion of the GEJ or stomach into the chest and formation of a hiatal hernia.

Although the correct anatomic position of the GEJ is important to the function of the GEJ, the critical element is the lower

esophageal sphincter (LES). Unlike a true anatomic sphincter, the LES is a circular or annular muscle that surrounds a lumen. It has a unique response to neurologic or hormonal stimulus, which makes it a true functional sphincter. The two main roles of the LES are to (1) relax during swallowing, allowing passage of food and liquid into the stomach, and (2) maintain a resting tone that prevents gross free reflux of gastric contents into the lower esophagus [2]. Malfunction or anatomic changes of the LES leads to a variety of characteristic symptoms. Heartburn, regurgitation, and dysphagia are the classic symptoms of gastroesophageal reflux. Chest pain, hoarseness, cough, asthma, and choking are considered atypical symptoms, although one or more of these symptoms are usually found in patients with gastroesophageal reflux (GER). Evaluation, diagnosis, and laparoscopic treatment of these conditions share some common key elements and are discussed in this paper.

#### **Evaluating the Patient with Esophageal Disease**

During the evaluation process, studies assessing the anatomy and physiology of the esophagus and GEJ should be completed. In addition to confirming the working diagnosis, the studies enable the surgeon to establish objective criteria and tailor the surgical procedure to the individual patient. Therefore it is recommended that one or all of the following tests be performed to evaluate the patient with esophageal symptoms.

# Upper Endoscopy

Endoscopy is the single most important examination when evaluating the esophagus and GEJ [3]. A direct, magnified view of the mucosa and flexible biopsy forceps enable the endoscopist to collect a tremendous amount of information. A patient must not eat 12 hours prior to this procedure or drink 8 hours prior to it. Laryngeal anesthesia is achieved by giving a spoonful of lidocaine and administering Xylocaine spray as needed. Sedation is achieve by a combination of a benzodiazepine and meperidine (Demerol), titrated for each person individually. Vital signs and oxygen saturation are always monitored. The endoscopist records the normal and diseased appearance of the esophagus, GEJ, stomach, and duodenum. The distance to the GEJ from the incisors is measured to determine the presence of a short esophagus.

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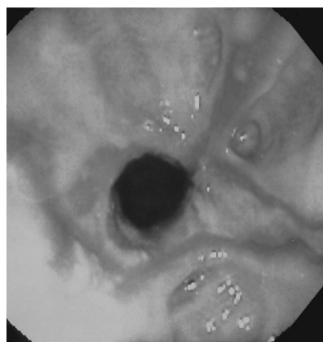


Fig. 1. Endoscopic view of the gastroesophageal junction shows linear erosions and a peptic stricture.

Stricture or rings must be identified prior to surgery to prevent injury from an esophageal dilator (Fig. 1). Any mucosal abnormality, esophagitis, ulcers, or tongue-like salmon-colored mucosal invaginations, which may represent Barrett's esophagus, indicate the need for biopsies in numerous locations. Occasionally, the endoscopist encounters unexpected pathology during the procedure (gastritis, peptic ulcers, or pyloric stenosis) that often manifest with symptoms similar to those of GEJ disease and should be addressed appropriately. If the surgeon is not involved in the endoscopic procedure, pictures of all abnormal findings should be obtained by the endoscopist and sent to the surgeon together with a written report. Otherwise, the surgeon may need to repeat the procedure because this information is crucial to the success of the operation and the management of the patient.

## Upper Gastrointestinal Contrast Study

Although the impact of flexible endoscopy has clearly reduced the significance of the barium swallow it still has a major role in the evaluation of patients with GEJ disease and is commonly used in clinical practice. A barium swallow study gives a complete anatomic view of the esophagus and stomach and is helpful for evaluating upper gastrointestinal abnormalities. The caliber of an esophageal stricture and resultant esophageal damage are better assessed with barium swallow than with endoscopy. The study can be performed with single or double contrast. The latter gives a detailed view of the esophageal mucosa, and minor changes can be detected. To study the GEJ the patient is placed in a horizontal prone position with the left side slightly elevated while drinking the contrast material through a straw. This is the only way to demonstrate the delicate Z-line of the GEJ [4], although the ability of barium swallow to detect damage to the esophageal mucosa has been questioned [5]. A single-contrast study points out small hiatal hernias (however small), estimates esophageal

Table 1. Normal values for esophageal manometry.

Parameter	Normal value
Upper esophageal sphincter (UES)	> 40 mmHg
UES relaxation	> 75%
Coordination with pharynx	> 75%
Hypopharyngeal pressure	> 30  mmHg
Lower esophageal sphincter (LES)	10-34 mmHg
LES relaxation	> 75%
Normal peristaltic progression	> 70%
Velocity of contraction	2-8 cm/s
Amplitude of contraction	35–150 mmHg (distal lead)
Duration of contraction	2–8 seconds

length, and may show esophageal or gastric anatomic changes. Although some series have shown that contrast radiology is a reasonably sensitive and specific test for detecting symptomatic GE reflux [6], there are conflicting data and the role of barium swallow by itself for quantifying reflux is not recommended [7]. Video-esophagography may provide an assessment of esophageal motility and swallowing function that complements esophageal manometry.

#### Esophageal Manometry

Esophageal manometry was first performed by Kronecker and Meltzer in 1883 [8] using air-filled balloons and an external pressure transducer. The LES was first identified manometrically by Fyke et al. in 1956 [9]. Today, modern assessment of the esophagus should always include a manometry study, especially for evaluating the LES and esophageal body function. The two systems of evaluation are the water infusion system and the solid-state catheter. The water infusion system consists of a catheter composed of several small capillary tubes that are continuously perfused with distilled water at a constant rate (0.5 ml/min) by a low-compliance pneumohydraulic capillary-infusion pump. The capillary tubes are connected to external transducers and a physiograph. The solid-state esophageal manometry catheter has microtransducers contained within the catheter that directly measure esophageal contractions [10]. Although there are some advantages to the use of a solid-state catheter system, the two systems give comparable results. In addition to its role in the diagnosis of esophageal and GEJ disorders, esophageal manometry enables the surgeon to choose the appropriate fundoplication. An inability of the LES to relax fully, weak esophageal body peristalsis, or incomplete peristaltic progression are common indicators of esophageal motor difficulties (Table 1).

# 24-Hour pH Monitoring

In 1964 Miller was first to describe the use of an indwelling pH probe to evaluate acid-peptic diathesis [11]. The 24-hour pH test has rapidly evolved into an accurate diagnostic test GE reflux. Since 1985 portable, solid-state digital recorders have been utilized for ambulatory pH studies. The small, lightweight recorder is carried by the patient at the waist, and at the completion of the monitoring period the stored information is analyzed to determine the amount of esophageal acid exposure. Total esophageal acid exposure time is a simple, accurate parameter to determine the existence of abnormal acid exposure in the distal esophagus. A drop in pH to below 4 at the distal esophagus for more than 4%

Table 2. Indications for 24-hour intraesophageal pH monitoring.

Incomplete response of typical reflux symptoms to medical therapy Evaluation of typical symptoms of reflux in patients without endoscopic evidence of esophagitis

Recurrence of reflux symptoms following fundoplication

Evaluation of atypical reflux symptoms such as asthma, chronic cough, hoarseness, noncardiac chest pain

of time is considered abnormal. A composite score (Johnson and DeMeester score) that weighs the number of reflux episodes, duration of reflux episodes, and several other parameters may also be used to quantify GER [12, 13].

When preparing for this test, one should consider the many variables that could influence the results. Alcohol consumption, smoking, and exercise have been shown to increase GE reflux; but strict dietary and activity restrictions during pH monitoring make it a somewhat artificial sampling, bearing less resemblance to the normal daily diet and activity of the symptomatic patient [12]. On the other hand, we recommend stopping medication such as antacids,  $H_2$ -receptor blockers, and prokinetics for at least 2 days before the study. Proton pump inhibitors should be stopped 10 days before the study.

Indications for 24-hour pH monitoring have been summarized in Table 2. Whenever possible, it is important to correlate the symptoms that occurred during the study period with the precise time of documented esophageal acidity. The severity of symptoms, however, cannot always be correlated with the results of pH monitoring [14]. Most reports of 24-hour pH monitoring in patients with GER describe the method's sensitivity as 76% to 100% and the specificity as 82% to 100% [14-18]. In earlier studies, 23% to 29% of patients with erosive esophagitis at endoscopy had normal pH monitoring results [14], but we have found otherwise-that 100% of patients with erosive esophagitis and typical symptoms of GER had an abnormal 24-hour pH test [19]. On the other hand, patients with typical reflux symptoms who have no esophagitis on endoscopy and have normal 24-hour pH monitoring are usually found to have an alternate diagnosis as the cause of their symptoms [20]. pH monitoring is not routinely performed for patients with paraesophageal hernias or achalasia because the results of this study would not influence the surgical procedure or the type of repair.

#### Gastric Emptying Study

Gastroesophageal reflux may be influenced by inadequate gastric emptying. A gastric emptying study is used predominantly in patients with gastric symptoms, a history of retained food, diabetes, bezoar formation, and gastric ulcers. The patient is given a radioisotope meal, and the progression of the chyme to the duodenum is followed. If delayed emptying (half-time more than twice the normal time) is detected, it may indicate a gastric cause of the GER. These patients usually require addition of pyloroplasty or pyloromyotomy to the fundoplication to facilitate postoperative emptying of the stomach. Delayed gastric emptying in some cases is an indicator of a diffuse gut motility disorder. In cases of suspected diffuse gut motility disorders, patients should have a small-intestinal motility study prior to undergoing antireflux surgery. Generalized gut dysmotility is considered a contraindication to antireflux surgery.

# General Principles of the Laparoscopic Approach to the GEJ

Many of the laparoscopic procedures of the GEJ share technical aspects and have similar principles. After the induction of general anesthesia and endotracheal intubation, the patient is placed in the lithotomy position or on a special fracture table so the surgeon can operate from between the patient's legs (Fig. 2) [21]. The first assistant stands at the patient's left side. An orogastric tube, Foley catheter, and compression boots are always placed. In patients with a large paraesophageal hernia the stomach often must be straightened by the surgeon before an orogastric tube can be inserted. After the pneumoperitoneum is achieved with a pressure of 15 mmHg, five trocars in a diamond-shaped configuration are inserted [22] (Fig. 3). The left lobe of the liver is elevated by a liver retractor positioned underneath the right costal margin through the right lateral 5-mm port. The surgeon operates using a two-handed technique from the two highest ports, and the first assistant uses the left lateral 5-mm port. The first step is to expose the GEJ by dissecting the right phrenoesophageal membrane (for large paraesophageal hernias this step may be delayed until the hernia contents are reduced and part of the sac is removed). An atraumatic grasper is held in the surgeon's left hand and curved scissors in the right hand. The membrane is dissected off the esophagus from the right crus to the left; then the plane between the crura and the esophagus is found and bluntly dissected (Fig. 4). Care is taken not to injure the vagus nerves. The short gastric vessels are divided starting at the level of the lower tip of the spleen. Dissection is greatly facilitated by the use of laparoscopic coagulating shears (LCS, Ultracision, Smithfield, RI, USA). The short gastric vessels, including the posterior gastric vessels, are dissected completely from this level. With further dissection the posterior esophagus is freed, and a Penrose drain is placed behind the esophagus to include the posterior vagus and anterior vagus nerves (Fig. 5). A clip is placed on the drain, and the first assistant retracts it to expose the GEJ. In every case the hiatal defect is closed by approximating the left and right crura posterior to the esophagus with interrupted O-neurolon suture tied intracorporeally. To avoid tension on this closure the pneumoperitoneum is decreased, for large hernias, to a pressure of 8 mmHg; 1-cm<sup>2</sup> pledgets may be used to decrease the likelihood of suture "cheese wiring" through the crura. At this point the surgeon continues with full or partial fundoplication or other specific procedure as planned. Some patients require deviation from this stepwise approach, especially those with achalasia, large paraesophageal hernias, or large esophageal diverticula, but for most GEJ operations the above-described steps are identical.

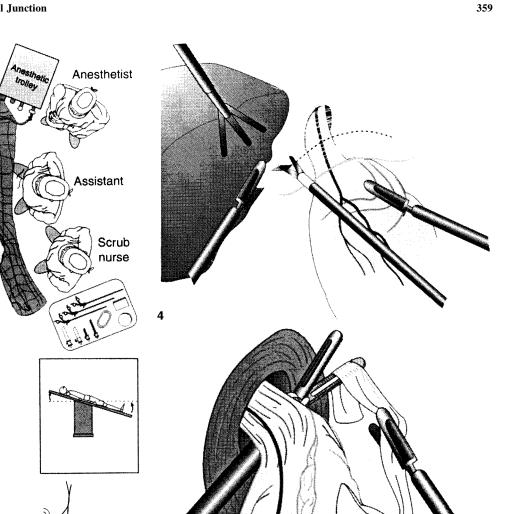
#### **Specific Indications**

#### Gastroesophageal Reflux

Symptoms of GER are the most common symptoms related to the GEJ. About 7% of U.S. adults have daily heartburn, 14% experience it weekly, and 40% have it monthly [23]. Generally, these symptoms respond well to over-the-counter antacids. Mild symptomatic GER can usually be managed empirically with life style and dietary modifications along with nonprescription hista-mine receptor antagonists. This approach is perfectly appropriate for the occasional symptom, especially when they occur as a result of a spicy or fatty meal or other similar digestive stress inducers.

Monitor

Surgeon



2

3

Camera operator

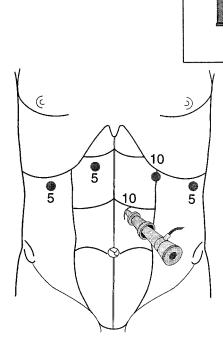


Fig. 2. Operating room set up for laparoscopic surgery of the gastroesophageal junction. (From Toouli et al. [21], with permission of Churchill Livingstone.)

Fig. 3. Trocar position for laparoscopic surgery of the gastroesophageal junction. (From Toouli et al. [21], with permission of Churchill Livingstone.)

Nevertheless, if the symptoms persist, become more frequent or severe, or if dysphagia develops, the patient should consult a physician. Although the diagnosis of GER is often suggested by the patient's history, one should be careful to exclude motility disorders and neoplastic causes of dysphagia. The most frequent indication for antireflux surgery is persistence of GER symptoms

**Fig. 4.** Dissection of the gastroesophageal junction (GEJ) starts by elevating the left lobe of the liver and dividing the phrenoesophageal ligament above the hepatic branch of the vagus. Except with very large livers, a 45-degree telescope optimizes exposure for GEJ procedures. (From Toouli et al. [21], with permission of Churchill Livingstone.)

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Fig. 5. After the crura are cleaned off posterior to their union, a grasper is passed directly behind the esophagus and a Penrose drain is pulled around the esophagus. (From Toouli et al. [21], with permission of Churchill Livingstone.)

despite intensive medical therapy. With modern proton pump inhibitor (PPI) treatment, failure of GER symptoms to respond is unusual, yet partial response or diminished effectiveness with time is not unusual. Furthermore, heartburn may be alleviated in many patients but significant regurgitation remains and continues to have a major life style impact. Atypical symptoms such as asthma

Study	No.	Conver- sion (%)	Hospital stay (days)	Incisional hernia (%)	Perforated viscus (%)	Acute fundopli- cation herniation (%)	Acute repair disruption (%)	Bleeding requiring transfusion or laparotomy (%)	Pneumo- thorax (%)	Persistent solid food dysphagia (%)	Gas bloat (%)	Recurrent heartburn (%)	Mortality (%)
Hinder [34]	198	2.0	3	-	2	1	1	1	1	6	13	1	0.5
Hunter [22]	252	1	2	0.3	2.8	0.3	-	0	1	4	11	3	0
Weertz [35]	132	3.3	2.8	1.5	2.3	_	0.8	0	_	5.4	-	0.8	0
Gotley [36]	200	4.5	3	_	1.5	1	0.5	2.5	1	2	_	7	0
Watson [37]	230	10	3	-	0.9	2.2	-	0.4	1.7	15	-	1.3	0.4
Total	1012	4.2	2.8	0.9	1.9	1.1	0.8	0.8	1.2	6.5	12	2.6	0.2

Table 3. Comparative results of laparoscopic Nissen fundoplication series.

and cough require large doses of PPI for control, so patients with atypical symptoms may need to take antireflux medications several times a day to notice symptom improvement. Individuals who show no response to medical therapy are less likely to have an optimal surgical result than those who respond well. The ideal candidate for surgery is a person who achieves heartburn control with PPIs and diet modification but has continued regurgitation and develops recurrent heartburn if he or she misses a day of medical therapy. Lastly, surgery is generally indicated to manage complicated GER, specifically Barrett's esophagus, esophageal stricture, and recurrent aspiration pneumonia.

Although many antireflux procedures have been described, most surgeons perform a variation of Nissen fundoplication. For patients with poor esophageal motility a Toupet (partial) fundoplication is recommended, and for patients with a short esophagus a Collis gastroplasty is added.

Nissen Fundoplication. The pressure gradient between the abdomen and thorax causes continuous stress on the GEJ and the antireflux mechanism. Routine daily activities such as lifting, running, coughing, and bending over may also aggravate this pressure gradient and facilitate reflux even in healthy, asymptomatic individuals. To prevent constant reflux, a number of factors are required for a competent GE barrier, including the LES, crural diaphragm, phrenoesophageal membrane, cardioesophageal angle, and intraabdominal location of the GEJ. It was not until the middle of this century that all these factors were known to influence reflux suppression. In fact, for many years hiatal hernia was thought to be the sole factor associated with the development of esophagitis and surgical reduction of the hernia was believed to be sufficient to prevent reflux and esophagitis [24]. In 1952 Ronald Belsey described a new method for correcting the GE barrier mechanism. Using a transthoracic approach, he described attaching the GEJ inside a collar of stomach and then attaching both structures to the anterior arch of the hiatus, creating the first flap-valve mechanism [25]. The concept of fundoplication was incidentally introduced 20 years earlier when in 1936 Rudolph Nissen wrapped gastric fundus around a GE anastomosis in patients who required distal esophagectomy for penetrating ulcer and severe esophagitis. Sixteen years later when he examined the area by endoscopy he found no evidence of esophagitis. In 1955 he applied this technique for GE reflux disease and performed a 360-degree 6-cm gastric fundoplication around the distal esophagus [26]. The use of a nipple valve to create a high pressure zone around the GEJ has been the mainstay of surgical treatment for GE reflux ever since. A modification of Nissen's technique, the floppy Nissen fundoplication, which reduces dysphagia and gas bloat, was proposed in 1976 by Donahue [27] as an improved technique to prevent gas bloat syndrome. In 1986 DeMeester demonstrated improved results using the technique of short, floppy Nissen fundoplication.

During the last 10 years there has been substantial progress in both medical and surgical treatment of GER disease. The introduction of PPIs signaled a revolution in the medical management of most gastric acid-related diseases, and surgically the laparoscopic fundoplication offers excellent results with minimal morbidity. The first laparoscopic Nissen fundoplication was performed by Geagea in 1991 [29], and since then the procedure has gained popularity. In 1996 Hunter et al. [22] reported 300 laparoscopic fundoplications performed over a 4-year period at a single institution. One year after surgery 93% of the patients reported no heartburn, and an additional 4% had only infrequent symptoms. Typical reflux symptoms responded best to surgery, and patients who responded to medical therapy had the best surgical outcome. The 24-hour pH tests performed 1 year postoperatively were normal in 94% of patients tested. Long-term results are beginning to be reported and appear similar to short-term results. These results as well as others [30-33] prove that laparoscopic fundoplication is safe, effective, and should be considered the new gold standard for treatment of GER (Table 3).

Toupet Fundoplication. A partial 270-degree fundoplication was first described in 1963 [38]. This operation provides less reflux protection than a Nissen fundoplication and is primarily indicated for patients with reflux who have poor esophageal motility. A complete 360-degree wrap often results in severe postoperative dysphagia because low amplitude peristalsis (<30 mmHg) may be unable to propel a bolus of food through the fundoplication. With the Toupet modification a 270-degree fundoplication is performed using three sutures on each side of the esophagus, with full-thickness bites of stomach and partial-thickness bites of the esophagus. Some authors recommend additional stitches between the posterior fundus and the crura [33]. The other steps of the procedure are similar to the steps of a Nissen fundoplication including intraoperative placement of an esophageal dilator. Despite incomplete reflux protections, early results of Toupet fundoplication are similar to those of the Nissen procedure [30, 33, 39].

Collis Gastroplasty and Fundoplication. In 1957 Collis introduced an operation for hiatal hernia repair in patients with a short

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esophagus [40]. Today it is estimated that 3% to 5% of patients who are candidates for antireflux surgery are found to have a short esophagus. For these patients a simple fundoplication is not adequate. A short esophagus might be suspected preoperatively in patients with a large diaphragmatic hernia, longstanding esophagitis, or esophageal stricture [41–43]. An upper gastrointestinal contrast study may also detect a short esophagus preoperatively. Nevertheless, the most reliable test is the one that is performed during the operation. After complete mobilization and dissection of the distal esophagus the GEJ is positioned under the diaphragm with no tension. If it remains comfortably in this position, a fundoplication alone suffices. A tendency of the GEJ to return to its former position in the chest is a sign that the repair may have a tendency to slip or herniate postoperatively. By continuing the dissection further into the mediastinum the surgeon can mobilize up to one-third of the esophagus to facilitate tension-free positioning of the GEJ. If high dissection and mobilization of the distal esophagus fails to bring the GEJ comfortably below the diaphragm, we use the Collis-Nissen technique to elongate the esophagus. An airtight circular stapler is introduced through a small subcostal incision and is fired at a point 3 inches from the angle of His next to a large Maloney dilator (usually 48F). A linear stapler is introduced through the periumbilical (laparoscope) port and passed through the new circular gastric opening; it is then fired toward the angle of His, creating a tube of stomach as a continuation of the distal esophagus. This "neoesophagus" now comfortably lies below the diaphragm with no tension. Fundoplication is performed in the usual way over the neoesophagus. Results of the laparoscopic Collis-Nissen approach are promising.

#### "Redo" Operations

Reoperation is one of the most challenging laparoscopic procedures. The most common reasons for redo procedures are recurrent reflux, postoperative dysphagia, or both [44, 45]. Postoperative dysphagia occurs for three main reasons: tight closure of the wrap or diaphragm, postoperative herniation of the GEJ into the chest, or a fundoplication that was created or slipped down around the stomach. Symptoms of heartburn and regurgitation may occur in patients who have disruption of the fundic wrap or the crural closure. One should remember that a fundoplication that is too floppy is not a reason for recurrent reflux symptoms. Occasionally, reflux occurs following partial fundoplication even if the fundoplication is intact. A study of redo fundoplication after laparoscopic fundoplication has shown that operative failure was mainly due to suture line disruption (4%), slipped or misplaced fundoplication (37%), herniation of the fundoplication (48%), or fundoplication that was too tight or too long (4%). The repair could usually be performed laparoscopically, but the conversion rate was 12% (Hunter, unpublished). The trocar position and initial approach are similar to those described above. Usually there are dense adhesions between the left lobe of the liver and the fundoplication itself. Meticulous sharp dissection is undertaken to reach the GEJ and dissect the crural ring under direct vision. Occasionally it is possible to reduce a herniated fundoplication, but most commonly the entire repair must be taken down and redone.

#### Barrett's Esophagus

The normal esophagus is lined by squamous epithelium. The junction of squamous epithelium with the columnar epithelium of the gastric cardia occurs within the LES and is normally seen endoscopically as the "Z-line." The LES situated at the distal esophagus may be up to 2.5 cm long, and the Z-line can be situated anywhere within the sphincter. Barrett's esophagus is present when intestinal (metaplastic) columnar epithelium is found in the tubular esophagus. It is recognized endoscopically as salmon-colored mucosa distinctly redder than the adjacent pale squamous esophageal mucosa. Infrequently, Barrett's epithelium extends into the middle and upper esophagus. Discovery of adenocarcinoma in Barrett's esophagus is discovered in about 10% of patients at initial endoscopy [46, 47]. The number of new cases of cancer in patients with Barrett's is estimated to range from one case in 48 patient-years to one case in 441 patient-years of follow-up [48]. Extrapolation of the incidence data has yielded a calculated incidence of 500 cancers per 100,000 patients with Barrett's esophagus per year. This incidence of 0.5% per annum suggests that some patients have a lifetime risk of developing cancer that approaches 15% to 25% [49]. Because of this risk, evidence of intestinal metaplasia at the GEJ is an indication for aggressive intervention. Dysplasia of Barrett's epithelium is defined as noninvasive neoplastic cellular proliferation. The detection of adenocarcinoma in Barrett's esophagus is preceded by the detection of dysplasia of increasing grades of severity with time [50]. Currently, intestinal metaplasia or low-grade dysplasia is an indication for an antireflux procedure. High-grade dysplasia or adenocarcinoma requires partial or total esophagectomy.

It has been shown that not only gastric acid but bile has an important role in the pathogenesis of Barrett's esophagus [51]. Although PPIs are strong acid suppressors, they have only a partial impact on the volume of reflux and do not prevent bile from reaching the GEJ. Therefore antireflux procedures have an advantage over PPIs for the treatment of Barrett's esophagus. Nevertheless, it should be remembered that patients who have undergone fundoplication should have careful routine endoscopic surveillance because the prevention of reflux by a successful fundoplication does not reverse intestinal metaplasia [52, 53].

## Peptic Esophageal Stricture

Peptic stricture is one of the most devastating complications of untreated or partially treated GER and usually occurs late in the course of the disease. The incidence of peptic stricture ranges from 1% to 5% of patients who have esophagitis and 0.01% of the total population [54]. The spectrum of peptic stricture ranges from a thin membrane like Schatzki's ring to a long fusiform esophageal narrowing of several centimeters. Repeated exposure of the distal esophagus to low pH gastric refluxate irritates the mucosa and may ultimately lead to the development of a constricted mucosal band. Diffuse circumferential peptic stricture may ensue after healing of deep untreated esophageal erosions or ulcers. It has been suggested that optimal medical therapy with PPIs decreases stricture recurrence and the need for repeat esophageal dilations. Nevertheless, even with this intensive treatment, 30% of patients require dilation within 1 year of successful initial dilation [55]. Generally, patients have had a few sessions of esophageal dilation and some form of medical treatment before they are referred for surgery. We believe that a single course of

Treatment	Series	Year	No.	Follow-up (years)	Effectiveness <sup>a</sup> (%)	Morbidity (%)	Mortality (%)
H <sub>2</sub> blockers with	Hands [56]	1989	195	4.8	54	3.6 (perforation)	None
dilations	Smith [55]	1994	185	1.0	54	N/A	None
PPI with	Smith [55]	1994	180	1.0	70	N/A	None
dilations	Angew $[57]^b$	1996	58	5.5	84	N/A	N/A
Open antireflux	Mercer [58]	1986	160	4.0	45-90	10 (splenectomy)	2.5
operation	Little [59]	1989	34	4.3	82	N/A	None
1	Vollan [60]	1992	43	0.2 - 12.0	53	9 (splenectomy)	None
	Bonavina [61]	1993	46	2.0	75	2 (leakage)	2.0
Laparoscopic fundoplication	Current series		40	1.5	88	2.5 (pneumothorax)	None

Table 4. Results of laparoscopic fundoplication compared to other treatments for peptic esophageal strictures.

PPI: proton pump inhibitors; N/A: not available.

"No further dilations after initial treatment.

<sup>b</sup>Mostly PPI.

dilatations, in addition to maximal pharmacologic therapy, is a reasonable initial approach to management of an esophageal stricture. When there is a need for frequent repetitive dilations, the patient should be referred for surgery. It appears, but has not been proved, that stricture patients benefit from aggressive preoperative dilation.

The surgical approach is similar to that previously described. The largest dilator possible (up to 60 F) based on previous dilation history, is introduced carefully into the esophagus before the fundoplication is constructed. In cases of esophageal stricture this step is important for dilating the stricture and ensuring the floppiness of the wrap. Thus the Maloney dilates the stricture and the fundoplication prevents future reflux from irritating the mucosa. Needless to say, this should be done after a careful esophageal workup including multiple biopsies of the stricture and other suspicious areas. Results of surgery in this group of patients are almost as good as those in patients without stricture and superior to other forms of therapy (Table 4). Nevertheless, 10% to 15% of patients who had dysphagia preoperatively continue to experience some form of disturbing dysphagia and still require esophageal dilations postoperatively [43].

#### Paraesophageal Hernia

Paraesophageal hernia is a defect of the diaphragmatic hiatus and was first described in 1926 by Akerlund as an uncommon form of hiatal hernia [62]. Most hiatal hernias (> 90%) are type I or sliding hiatal hernias where the GEJ is most cephalad. Paraesophageal hernias (types II and III) account for only 3% to 10% of hiatal hernias. In type II the GEJ is in the abdomen, and the fundus herniates. In type III both the GEJ and the gastric fundus herniate (Fig. 6). Type III is more common than type II especially for large hernias. Rarely, a hiatal hernia is found where the GEJ is in its anatomic position and the fundus herniates through a separate diaphragmatic defect (parahiatal hernia). Symptoms of paraesophageal hernias include dysphagia, chest pain, postprandial pain, nausea, vomiting, and excessive belching. Patients with paraesophageal hernia are older and have more co-morbid conditions than other groups of patients who have disorders of the GEJ [63, 64].

Some issues are debated regarding management of paraesophageal hernias. First, is the presence of paraesophageal hernia in asymptomatic patients an indication for elective surgical repair?

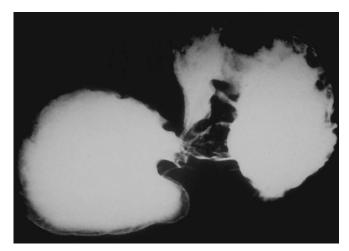


Fig. 6. Type III paraesophageal hernia.

Second, should the hiatal defect be closed primarily in all cases, or should a prosthetic mesh be added for large defects? Finally, controversy remains about whether a concomitant antireflux procedure should be included as part of the operation in all patients with paraesophageal hernia. Traditionally, all paraesophageal hernias, symptomatic and asymptomatic, are repaired as soon as they are found. This approach is based on evidence that although many patients are asymptomatic paraesophageal hernias are associated with significant complications. Emergency admission for obstruction, bleeding, chest pain, and perforation occur in up to one-third of patients. Elective repair of all paraesophageal hernias was further supported during the laparoscopic era as laparoscopic repair has been shown to be associated with less morbidity than open repair [65-68]. Nevertheless, a more selective and conservative approach has been advocated [69]. We currently repair all symptomatic paraesophageal hernias; elderly patients with asymptomatic hernias and normal endoscopy are followed closely and are not routinely referred for surgery.

The rationale for the addition of a fundoplication in all patients with paraesophageal hernia is based on three premises: First, abnormal reflux is present in many patients and the absence of symptoms of reflux does not always correlate well with the physiologic status of the LES mechanism or with pathologic reflux episodes. Second, a proper repair requires significant dissection of

Table 5. Results of laparoscopic and open repair of paraesophageal hernia.

Study	Year	No.	Procedure	Mesh	Crural closure (no.)	Antireflux procedure (no.)	Collis Nissen	Hospital stay (days)	Major complication
Myers [67]	1995	37	Laparotomy	No	15	No	N/A		1 Splenic injury, 1 esophageal perforation, 1 CVA, 1 recurrence
Pitcher [68]	1995	12	Laparoscopy	Selectively	8	No	Usually	2.5	1 Gastric atony
Casabella [65]	1996	15	Laparoscopy	No	15	No		3	1 Atrial fibrilation
Fuller [70]	1996	14	Laparoscopy, thoracotomy, laparoscopy	No	14	No		3.6 (lap) 7.7 (open)	N/A
Perdikis [69]	1997	65	Laparoscopy	No	65	No		2	3 Reoperations
Trus [63]	1997	76	Laparoscopy	No	71	6		4.2	2 Deaths, 11 gastric or esophageal perforations
Willekes [66]	1997	30	Laparoscopy	Selectively	23	No	Usually	2–3	1 Dysphagia, 1 pulmonary embolus, 3 GE reflux

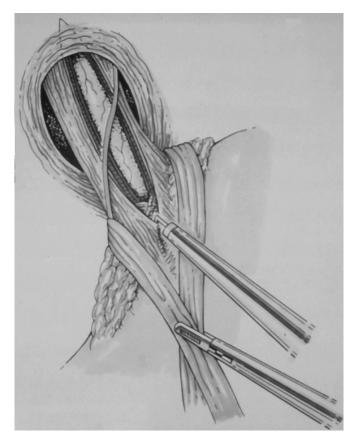
CVA: cerebral vascular accident (stroke); GE: gastroesophageal; lap: laparoscopy; N/A: not available.

the phrenoesophageal membrane and complete mobilization of the GEJ attachments such that even if reflux was not present preoperatively it is likely to occur after the operation. Recent studies have shown that many patients with type III hernias had significant heartburn before the gastric fundus herniated (i.e., before the type I hernia became a type III hernia) [64]. Realigning the stomach below the GEJ may allow resumption of reflux. Third, a bulky fundoplication can be helpful for anchoring the GEJ in the subdiaphragmatic position, making the recurrence of the hernia less likely. Support for the selective use of an antireflux procedure is based on the observation that most patients with type II hernias have normal position and attachments of GEJ, and 80% of these patients do not have reflux preoperatively and would therefore undergo an "unnecessary" antireflux procedure if routine use were advocated [66, 69]. Unfortunately, true type II hernias are rare. Following the initial experience and in agreement with most other authors we recommend adding an antireflux procedure in all cases of paraesophageal hernia repair (Table 5). Finally, although some authors add Prolene mesh to the repair in large paraesophageal hernias [65, 68], it has been our practice to repair the hernias primarily with interrupted nonabsorbable sutures enforced with pledgets [63, 71]. Failed procedures that involve prosthetic mesh may require partial gastrectomy or esophagogastrectomy.

The principles of the laparoscopic operative approach are similar to those described for an open technique. Reduction of the hernia contents, removal of the peritoneal sac, closure of the diaphragm, and fundoplication are necessary steps in the repair [63, 71]. Reduction of the entire hernia initially would not always be feasible because of mediastinal adhesions. In these cases it is better to dissect the sac circumferentially first, starting from the lower aspect of the left crus and continuing counterclockwise. If the sac cannot be dissected entirely, it should be transected and the cut portion removed. Detachment of the GEJ from the mediastinum is greatly facilitated by these first steps. Results have been satisfactory (Table 5), although complications of this procedure are more frequent than those associated with Nissen or Toupet fundoplication [63, 66].

#### Laparoscopic Modified Heller Myotomy

Achalasia of the esophagus is a primary motor disorder characterized by intermittent and progressive dysphagia produced by denervation of the esophagus with absence of neurons in the myenteric plexus. The main pathophysiologic consequences are aperistalsis of the esophageal body, hypertensive LES, and incomplete relaxation of the LES. These changes cause impairment of esophageal emptying, regurgitation, and dysphagia. Food accumulates, and the esophagus becomes dilated and elongated. Unfortunately, the role of pharmacotherapy is limited, and almost all patients eventually require forceful dilations, injections of butolinium toxin, or surgery. In 1914 Heller [72] introduced a surgical procedure aimed at correcting achalasia. He performed both anterior and posterior myotomy extending from the esophagus down over the cardia to the stomach. A modified Heller myotomy was carried out by Groenevelt in 1918 [73] in which a single anterior myotomy was performed. The modified Heller procedure has since been the technique of choice for the surgical treatment of achalasia. Generally, the initial treatment for patients with achalasia is some type of forceful dilation. Nevertheless, pneumatic or hydrostatic dilations and more recently injections of botulinum toxin give only temporary relief and generally last only a few months [74, 75]. Perforation of the esophagus occurs in 3.5% of patients undergoing balloon dilation and GER is the most common late complication, its incidence ranging from 4.2% to 8.8% [76]. It has been suggested that myotomy offers a better long-term outcome than pneumatic dilatation for patients with achalasia [77, 78]. Results of laparoscopic Heller myotomy are equivalent or better than those of open myotomy because the myotomy can be performed under magnification, allowing precise division of the muscle fibers [79, 80]. Trocar placement for this procedure is similar to that for other GEJ operations [80]. After complete mobilization of the distal esophagus and fundus, blunt dissection using scissors is employed to spread the longitudinal esophageal muscles just above the GEJ. Once the longitudinal fibers are separated, the closed scissors are slipped below the circular muscle and opened again, lifting the muscularis off the mucosa. The shears are opened and rotated 90 degrees, and the circular muscle is cut. Dissection continues with the hook, blunt rods, and blunt graspers. The mucosa can usually be seen after 1 to 2 cm of dissection and is bluntly pushed away from the circular muscular layer (at this point there is no dilator inside the lumen of the esophagus). The myotomy is carried proximally 6 cm from the GEJ exposing the mucosa of 180 degrees at the anterior esophagus. Distally, the dissection continues 1 cm over the muscular layers of the stomach to ensure complete division of the LES. This latter dissection should be done carefully because the



**Fig. 7.** Posterior hemifundoplication anchors the well mobilized fundus to the cut edges of the myotomy.

tissue planes are not always apparent, and it is relatively easy to injure the mucosa. When the dissection is completed the mucosal integrity is ensured by injecting methylene blue solution, and fundoplication (Dor or Toupet) is performed. This is sutured to the cut muscle edges to prevent reflux and to keep the muscles from reapproximating (Fig. 7).

#### **Esophageal Diverticulectomy**

Epiphrenic or distal esophageal diverticula are pulsion diverticula and are associated with esophageal motility disorders. With a mechanism similar to that of pharyngoesophageal diverticula, abnormally elevated intraluminal pressure is responsible for the protrusion of esophageal mucosa and submucosa through the muscle layers. The diverticula can reach remarkable size, and when distended with food they can cause dysphagia, regurgitation, and retrosternal chest pain. Asymptomatic, small diverticula (< 3 cm) require no treatment, whereas those with progressively severe dysphagia and chest pain, and an anatomically dependent enlarging pouch, require operation. The principles of surgery are identical to those for Zencker's diverticula, which are also pulsion diverticula that result from motility disorders of the cricopharyngeal muscle.

A liberal myotomy of the distal esophageal muscle that creates a constricting muscle ring in addition to resection of the diverticula is the procedure of choice. This procedure is achieved adequately laparoscopically. The long esophagomyotomy is performed using scissors and hook electrocautery, which elevates the circular muscle away from the mucosa. The diverticular sac is resected at its neck with an endostapler taking care not to constrict the esophagus. A 60F dilator placed at the distal esophagus is helpful for preventing the stapler from narrowing the lumen. Myotomy alone is adequate treatment for most small diverticula. Those that cannot be reached from the abdomen are treated by thoracoscopic myotomy and diverticulectomy *or* laparoscopic myotomy followed by staged thoracoscopic diverticulectomy for the few patients who remain symptomatic after myotomy alone.

#### **Intraoperative and Postoperative Complications**

Massive bleeding from the stomach, spleen, or liver that requires conversion or transfusion is rare. Most intraoperative bleeding is minor and can usually be dealt with laparoscopically. Trocar wound infections are uncommon or resolve quickly. Intraabdominal abscesses are rare and usually indicate occult visceral perforation. Several types of perforation are readily identified. The esophagus may be injured by the Maloney pushed too quickly or too aggressively or that turns back on itself at the level of a stricture. To prevent esophageal injury graspers are never placed on the esophagus. The stomach can be injured from vigorous pulling and manipulation especially if inappropriate instrumentation is used. Small serosal tears can be left alone. Deep laceration should be repaired with laparoscopic suturing. The omentum can be used to buttress gastric repairs, and the fundoplication itself can be used to seal an esophageal repair. When repairing a large paraesophageal hernia that requires mediastinal dissection, the surgeon should keep in mind the possibility of cardiac arrhythmia or pneumothorax. Pneumothorax commonly occurs during esophageal mobilization when the surgeon dissects the left mediastinal esophagus from the parietal pleura. If a grasper is allowed to pass behind the esophagus above the left crus of the diaphragm a pneumothorax occurs. If unrecognized during the dissection, a left pneumothorax first manifests after eversion of the left diaphragm. Pneumothorax is usually well tolerated because of positive-pressure ventilation unless a small pleural laceration allows tension pneumothorax physiology. To prevent a tension pneumothorax, any pleural laceration is opened widely and the pneumoperitoneum is lowered to 8 to 10 mmHg. A small red rubber catheter may be inserted laparoscopically through the defect in the pleura until the procedure is completed. At the end of the procedure the catheter is brought out a trocar site, and the end is placed in a basin of water. The anesthetist "blows out" the pneumothorax through this homemade water seal. Chest tubes are unnecessary. Because there is usually no injury to the lung itself, if a small pneumothorax remains after the catheter is removed the  $CO_2$  is absorbed rapidly.

The most common postoperative complication is dysphagia [81]. During the immediate postoperative period it is usually the result of swelling at the operative site and generally resolves within a few weeks. In our experience long-term dysphagia is not a significant problem except with the Rosetti-Nissen procedure [22] or with a disrupted (slipped or herniated) fundoplication. For patients with immediate postoperative dysphagia we recommend continuing a soft or liquid diet for up to a month. A barium swallow is performed 1 month after the procedure in patients with persistent dysphagia. Endoscopic esophageal dilation may help patients with postoperative dysphagia and no evidence of wrap disruption, sliding of the fundoplication, or paraesophageal her-

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niation. In the latter cases, which occur 2% to 8% of the time, a plan for reoperation should be made.

Self-limiting postoperative bloating or explosive diarrhea occurs in about 15% of patients. With appropriate diet instruction, addition of fiber to the diet, and time those symptoms usually resolve. The physiologic cause of these symptoms is unknown, although neuropraxis of the vagal nerve may be the cause of most. In rare cases the symptoms persist, indicating an underlying intestinal motility disorder, permanent vagal injury, gastroparesis, or irritable bowel syndrome.

Laparoscopic surgery has revolutionized many of the traditional concepts of surgery. However, advanced laparoscopic procedures are technically complicated and more difficult than the equivalent operation performed through a laparotomy. The benefits of laparoscopic access makes this approach popular with patients, primary physicians, and gastroenterologists. The amount of time required to learn advanced technical skills is extensive, and there must be a commitment from the surgeon to spend these frustrating hours learning the maneuvers that can be performed in just few seconds with the traditional approach. Nevertheless, after mastering the laparoscopic techniques the laparoscopist can offer more advanced surgery. Although many laparoscopic procedures are under close scrutiny, it is becoming apparent that for functional surgery of the GEJ laparoscopy is the access of choice.

#### Résumé

Le reflux gastro-oesophagien (RGO), secondaire à l'incompétence du sphincter inférieur de l'œsophage, est l'indication la plus fréquente de chirurgie de la jonction gastro-oesophagienne. L'évaluation, le diagnostic et le traitement chirurgical moderne du RGO sont discutés. L'évaluation des patients ayant un pyrosis sévère comprend une fibroscopie du tube digestif supérieur pour apprécier l'état de l'œsophage, de l'estomac et du duodénum, un transit oesophagogastrique pour étudier l'anatomie complète de l'œsophage et l'estomac, une pH-métrie des 24 heures pour déterminer l'exposition de l'œsophage à l'acidité et une étude de la vidange gastrique pour étudier la motilité gastrique. Ces études sont le plus souvent suffisantes pour faire le diagnostic de RGO, de hernie hiatale, de l'oesphagite de Barrett, de la sténose peptique, de la hernie para-oesophagienne ou de l'achalasie. La panoplie du traitement laparoscopique comprend la fundoplicature de Nissen, l'opération de Toupet, la gastroplastie de Collis, la myotomie de Heller modifiée, la diverticulotomie de l'œsophage et les différentes techniques de reprise. Ces procédés sont décrits en détails. Les résultats de ces opérations indiquent qu'elles sont sûres, efficientes et devraient être considérées comme le nouveau ≪gold standard≫ pour la correction des troubles de la jonction gastro-oesophagienne. La chirurgie laparoscopique a beaucoup changé par rapport aux procédés pratiqués traditionnellement par laparotomie. Bien que plus difficile techniquement, nécessitant du temps et de l'adresse pour être performante, il devient évident que pour la chirurgie fonctionnelle de la jonction oesophagogastrique, la laparoscopie représente un procédé de choix.

# Resumen

La incompetencia del mecanismo del esfínter esofágico inferior, que resulta en reflujo gastroesofágico (RGE), constituye la indicación más común para practicar cirugía de la unión gastroesofágica. En el presente artículo se discute la evaluación, el diag-

nóstico y el tratamiento moderno del RGE. La evaluación de los pacientes con pirosis severa incluye endoscopia superior para determinar la condición general del esófago, el estómago y el duodeno; un estudio con medio de contraste del tracto gastrointestinal superior para una visión anatómica del esófago y el estómago; manometría esofágica para evaluar la función del esófago; monitoría de pH de 24 horas para determinar la exposición esofágica al ácido; y un estudio selectivo del vaciamiento gástrico para determinar la presencia de alteraciones de la motilidad. Tales estudios comprueban el diagnóstico de RGE, hernia hiatal, esófago de Barrett, estrechez péptica del esófago, hernia parahiatal o acalasia. El abordaje laparoscópico incluye la fundoplicación de Nissen, la fundoplicación de Toupet, la gastroplastia de Collis con fundoplicación, la miotomía de Heller modificada, la diverculectomía esofagiana y las operaciones de revisión, procedimientos que se describen detalladamente. Los resultados de estas operaciones señalan su seguridad y efectividad, por lo cual deben ser consideradas como el nuevo patrón oro en la corrección de la patología gastroesofágica. La cirugía laparoscópica ha revolucionado muchos de los procedimientos tradicionales que se practican mediante laparotomía. Aunque técnicamente son más difíciles de ejecutar y requieren un tiempo y una práctica significativos para que el cirujano llegue a dominarlos, se hace cada día más evidente que el abordaje laparoscópico es el de escogencia para realizar cirugía funcional de la unión gastroesofágica.

#### References

- Mittal, K.R., Balaban, D.H.: The esophagogastric junction. N. Engl. J. Med. 336:924, 1997
- Castell, D.O.: The lower esophagus sphincter: physiologic and clinical aspects. An. Intern. Med. 83:390, 1975
- Misumi, A., Murakami, A., Honmyo, U., Donahue, P.E.: Endoscopic examination including dye endoscopy. In Surgery of the Esophagus Stomach and Small Intestine, C. Watsell, L.M. Nyhus, P.E. Donahue, editors. Boston, Little Brown, 1995
- Freeman, A.H.: Diagnostic radiography of the esophagus. In: Surgery of the Esophagus Stomach and Small Intestine, C. Watsell, L.M. Nyhus, P.E. Donahue, editors. Boston, Little Brown, 1995
- Neumann, C.H., Forster, C.F.: Gastroesophageal reflux: reassessment of the value of fluoroscopy based on manometric evaluation of the lower esophageal segment. Am. J. Gastroenterol. 78:776, 1983
- Sellar, R.I., Decaestecker, J.S., Heading, P.B.: Barium radiology: a sensitive test for gastroesophageal reflux. Clin. Radiol. 38:303, 1987
- Bombeck, C.T.: Gastro-oesophageal reflux. In: Surgery of the Stomach and Duodenum (4<sup>th</sup> ed.), L.M. Nyhus, C. Westell, editors. Boston, Little Brown, 1986
- Kronecker, H., Meltzer, S.J.: Der Schluckmechanismus, sein Erregung and seine Hemmung. Arch. Ges. Anat. Physiol. 7(suppl):328, 1883
- Fyke, F.E., Code, C.F., Schlegel, J.: The gastroesophageal sphincter in healthy human beings. Gastroenterologia (Basel) 86:135, 1956
- Castell, J.A., Boag-Dalton, C.: The esophageal motility laboratory: material and equipment. In: Esophageal Motility Testing (2<sup>nd</sup> ed.), D.O. Castell and J.A. Castell, editors. Norwalk, CT, Appleton & Lange, 1994, pp. 27–34
- Miller, F.A.: Utilization of inlying pH probe for evaluation of acidpeptic diathesis. Arch. Surg. 89:199, 1964
- Jamieson, J.R., Stein, H.J., DeMeester, T.R., Bonavina, L., Schwizer, W., Hinder, R.A., Albertucci, M.: Ambulatory 24-h esophageal pH monitoring: normal values, optimal thresholds, specificity, sensitivity and reproducibility. Am. J. Gastroenterol. 87:1102, 1992
- Johnson, L.F., DeMeester, T.R.: Development of the 24-hour intraesophageal pH monitoring composite scoring system. J. Clin. Gastroenterol. 1(suppl):52, 1986
- Vitale, G.C., Cheadle, W.G., Sadek, S., Michel, M.E., Cuschieri, A.: Computerized 24-hour ambulatory esophageal pH monitoring and

esophagogastroduodenoscopy in the reflux patient: a comparative study. Ann. Surg. 200:724, 1984

- Fink, S.M., McCallum, R.W.: The role of prolonged esophageal pH monitoring in the diagnosis of gastroesophageal reflux. J.A.M.A. 252:1160, 1984
- Grande, L., Pujol, A., Ros, E., Garcia-Valdecasas, J.C., Fuster, J., Visa, J., Pera, C.: Intraesophageal pH monitoring after breakfast + lunch in gastroesophageal reflux. J. Clin. Gastroenterol. 10:373, 1988
- Johnson, F., Joelsson, B., Isberg, P.F.: Ambulatory 24-hour intraesophageal pH monitoring in the diagnosis of gastroesophageal reflux disease. Gut 28:1145, 1987
- Porro, G.B., Pace, F.: Complication of the three methods of intraesophageal pH recording in the diagnosis of GER. Scand. J. Gastroenterol. 23:743, 1988
- Waring, J.P., Hunter, J.G., Oddsdottir, M., Wo, J., Katz, E.: The preoperative evaluation of patients considered for laparoscopic antireflux surgery. Am. J. Gastroenterol. 90:35, 1995
- DeMeester, T.R., Wang, C.I., Wernly, J.A., Pellegrini, C.A., Little, A.G., Klementschitsch, P., Bermudez, G., Johnson, L.F., Skinner, D.B.: Technique, indications and clinical use of 24 hour esophageal pH monitoring. J. Thorac. Cardiovasc. Surg. 79:656, 1980
- Hunter, J.G., Champion, J.K.: Laparoscopic Nissen fundoplication. In Endosurgery, J. Toouli, D. Gossot, J.G. Hunter, editors. London, Churchill Livingstone, 1996, pp. 305–313
- Hunter, J.G., Trus, T.L., Branum, G.D., Waring, J.P., Wood, W.C.: A physiologic approach to laparoscopic fundoplication for gastroesophageal reflux disease. Ann. Surg. 223:673, 1996
- Nebel, O.T., Fornes, M.F., Castell, D.O.: Symptomatic gastroesophageal reflux: incidence and precipitating factors. Am. J. Dig. Dis. 21:953, 1976
- 24. Alison, P.O.: Reflux esophagitis, sliding hiatal hernia and the anatomy of repair. Surg. Gynecol. Obstet. *92*:419, 1951
- Hiebert, C.A., Belsey, R.: Incompetency of the gastric cardia without radiologic evidence of hiatal hernia. J. Thorac. Cardiovasc. Surg. 53:33, 1961
- Nissen, R.: Eine einfache operation Zur beeinflussung der refluxoesophagitis. Schweiz. Med. Wochenschr. 86:590, 1956
- Donahue, P.E., Samuelson, S., Nyhus, L.M., Bombeck, C.T.: The floppy Nissen fundoplication: effective long term control of pathologic reflux. Arch. Surg. 120:663, 1987
- DeMeester, T.R., Bonovina, L., Albertucci, M.: Nissen fundoplication for gastroesophageal reflux disease: evaluation of primary repair in 100 consecutive patients. Ann. Surg. 204:9, 1986
- Geagea, T.: Laparoscopic Nissen fundoplication: preliminary report on ten cases. Surg. Endosc. 8:1417, 1991
- Mosnier, H., Leport, J., Aubert, A., Kianmanesh, R., Sbai Idrissi, M.S., Guivarch, M.: A 270 degree laparoscopic posterior fundoplication in the treatment of gastroesophageal reflux. J. Am. Coll. Surg. 181:220, 1995
- Anvari, M., Allen, C., Borm, A.: Laparoscopic Nissen fundoplication is a satisfactory alternative to long-term omeprazole therapy. Br. J. Surg. 81:938, 1995
- McAnena, O.J., Wilson, P.D., Evans, D.F., Kadirkamanathan, S.S., Mannur, K.R., Wingate, D.L.: Physiological and symptomatic outcome after laparoscopic gastric fundoplication. Br. J. Surg. 82:795, 1995
- Laws, H.L., Clements, R.H., Swillie, C.M.: A randomized, prospective comparison of the Nissen fundoplication versus the Toupet fundoplication for gastroesophageal reflux disease. Ann. Surg. 225:647, 1997
- Hinder, R.A., Filipi, C.J., Wetscher, G., Neary, P., DeMeester, T.R., Perdikis, G.: Laparoscopic Nissen fundoplication is an effective treatment for gastroesophageal reflux disease. Ann. Surg. 220:472, 1994
- 35. Weerts, J.M., Dallemagne, B., Hamoir, E., Demarche, M., Markiewicz, S., Hehaes, C., Lombard, R., Demoulin, J.C., Etienne, M., Ferron, P.E., Fontaine, F., Gillard, V., Delforge, M.: Laparoscopic Nissen fundoplication: detailed analysis of 132 patients. Surg. Laparosc. Endosc. 3:359, 1993
- Gotley, D.C., Smithers, B.M., Rhodes, M., Menzies, B., Branicki, F.J., Nathanson, L.: Laparoscopic Nissen fundoplication: 220 consecutive cases. Gut 38:487, 1996
- 37. Watson, D.I., Jamieson, G.G., Devitt, P.G., Matthews, G., Britten-Jones, R.E., Game, P.A., Williams, R.S.: Changing strategies in the performance of laparoscopic Nissen fundoplication as a result of experience with 230 operations. Surg. Endosc. 9:961, 1995
- 38. Toupet, A.: Technique de oesophagogastropastie a el phrenogas-

tropexie appliquee dans la cure radicale des hernia hiatal es et comme complement de operation de Heller dans les cardiospamsus. Mem. Acad. Clin. 89:374, 1963

- Patti, M.G., DePinto, M., DeBellis, M.: Comparison of laparoscopic total and partial fundoplication for gastroesophageal reflux. J. Gastrointest. Surg. 1:309, 1997
- Collis, J.L.: An operation for hiatal hernia with short esophagus. J. Thorac. Cardiovasc. Surg. 34:768, 1957
- Pati, M.G., Goldberg, H.I., Arcerito, M., Bartolasi, L., Tong, J., Way, L.W.: Hiatal hernia size affects lower esophageal sphincter function esophageal acid exposure, and the degree of mucosal injury. Am. J. Surg. 171:182, 1996
- Swanstrom, L.L., Marcus, D.R., Galloway, G.O.: Laparoscopic Collis gastroplasty is the treatment of choice for the shortened esophagus. Am. J. Surg. 171:477, 1996
- Spivak, H., Trus, T.L., Branum, G.D., Waring, J.P., Hunter, J.G.: Laparoscopic fundoplication for peptic esophageal strictures and dysphagia. J. Gastrointest. Surg. 2:555, 1998
- 44. Stein, H.J., Feussner, H., Siewert, J.R.: Failure of antireflux surgery: causes and management strategies. Am. J. Surg. *171*:36, 1996
- Rieger, N.A., Jamieson, G.G., Britten-Jones, R., Tew, S.: Reoperation after failed antireflux surgery. Br. J. Surg. 81:1159, 1994
- 46. Dent, J., Bremner, C.G., Collen, M.J., Haggitt, R.C., Spechler, S.J.: Barrett's oesophagus [review]. J. Gastroenterol. Hepatol. *6*:1, 1991
- Spechler, S.J., Goyal, R.K.: Barrett's esophagus. N. Engl. J. Med. 315:3622, 1986
- Tytgat, G.N., Hameeteman, W.: The neoplastic potential of columnarlined (Barrett's) esophagus [review]. World J. Surg. 16:308, 1992
- Spechler, S.J.: Endoscopic surveillance for patients with Barrett's esophagus: does the cancer risk justify the practice? Ann. Intern. Med. 106:902, 1987
- Robertson, C.S., Mayberry, J.F., Nicholson, D.A., James, P.D., Atkinson, M.: Value of endoscopic surveillance in the detection of neoplastic change in Barrett's oesophagus. Br. J. Surg. 75:760, 1988
- Clark, G.W.B., Ireland, A.P., Peters, J.H., Chandrasoma, P., De-Meester, T.R., Bremner, C.G.: Short-segment Barrett's esophagus: a prevalent complication of gastroesophageal reflux disease with malignant potential. J. Gastrointest. Surg. *1*:1113, 1997
- McDonald, M.L., Trastek, V.F., Allen, M.S., Deschamps, C., Pairolero, P.C.: Barrett's esophagus: does an antireflux procedure reduce the need for endoscopic surveillance? J. Thorac. Cardiovasc. Surg. 111:1135, 1996
- Sagar, P.M., Ackroyd, R., Hosie, K.B., Patterson, J.E., Stoddard, C.J., Kingsnorth, A.N.: Regression and progression of Barrett's oesophagus after antireflux surgery. Br. J. Surg. 82:806, 1995
- Ferguson, M.K.: Medical and surgical management of peptic esophageal strictures. Chest Surg. Clin. North Am. 4:673, 1994
- 55. Smith, P.M., Kerr, G.D., Cockel, R., Ross, B.A., Bate, C.M., Brown, P., Dronfield, M.W., Green, J.R., Hislop, W.S., Theodossi, A.: A comparison of omeprazole and ranitidine in the prevention of recurrence of benign esophageal stricture. Gastroenterology 107:1312, 1994
- Hands, L.J., Papavramidis, S., Bishop, H., Dennison, A.R., McIntyre, R.L., Kettlewell, M.G.: The natural history of peptic oesophageal strictures treated by dilation and antireflux therapy alone. Ann. R. Coll. Surg. Engl. 71:306, 1989
- Angew, S.R., Pandya, S.P., Reynolds, R.P.E., Preiksaitis, H.G.: Predictors for frequent esophageal dilatations of benign peptic strictures. Dig. Dis. Sci. 41:931, 1996
- Mercer, C.D., Hill, L.D.: Surgical management of peptic esophageal stricture. J. Thorac. Cardiovasc. Surg. 91:3471, 1986
- Little, A.G., Naunheim, K.S., Ferguson, M.K., Skinner, D.B.: Surgical management of esophageal stricture. Ann. Thorac. Surg. 45:144, 1988
- Vollan, G., Stangeland, L., Soreide, J.A., Janssen, C.W., Svanes, K.: Long term results after Nissen fundoplication and Belsey Mark IV operation in patients with reflux oesophagitis and stricture. Eur. J. Surg. *158*:357, 1992
- Bonavina, L., Fontebasso, V., Bardini, R., Baessato, M., Peracchia, A.: Surgical treatment of reflux stricture of the esophagus. Br. J. Surg. 80:317, 1993
- Perdikis, G., Hinder, R.A.: Paraesophageal hiatal hernia. In: Hernia (4<sup>th</sup> ed.), L.M. Nyhus, R.E. Condon, editors. Phliadelphia, Lippincott, 1995, pp. 543–554.
- 63. Trus, T.L., Bax, T., Richardson, W.S., Branum, G.D., Mauren, S.J.,

- esophageal hernia repair. J. Gastrointest. Surg. 1:221, 1997
  64. Wo, J.M., Branum, G.D., Hunter, J.G., Trus, T.L., Mauren, S.J., Waring, J.P.: Clinical features of type III (mixed) paraesophageal hernia. Am. J. Gastroenterol. 91:914, 1996
- Casabella, F., Sinanan, M., Horgan, S., Pellegrini, C.A.: Systematic use of gastric fundoplication in laparoscopic repair of paraesophageal hernias. Am. J. Surg. 171:485, 1996
- Willekes, C.L., Edoga, J.K., Frezza, E.E.: Laparoscopic repair of paraesophageal hernia. Ann. Surg. 225:31, 1997
- Myers, G.A., Harms, B.A., Starling, J.B.: Management of para esophageal hernia with a selective approach to antireflux surgery. Am. J. Surg. 170:375, 1995
- Pitcher, D.E., Curet, M.J., Martin, D.T., Vogot, D.M., Mason, J., Zucker, K.A.: Successful laparoscopic repair of paraesophageal hernia. Arch. Surg. 130:590, 1995
- Perdikis, G., Hinder, R.A., Filipi, C.J., Walenz, T., McBride, P.J., Smith, S.L., Katada, N., Klingler, P.J.: Laparoscopic paraesophageal hernia repair. Arch. Surg. *132*:586, 1997
- Fuller, C.B., Hagen, J.A., DeMeester, T.R., Peters, J.H., Ritter, M., Bremner, C.G.: The role of fundoplication in the treatment of type II paraesophageal hernia. J. Thorac. Cardiovasc. Surg. 111:655, 1996
- Oddsdottir, M., Franco, A.L., Laycock, W.S., Waring, J.P., Hunter, J.G.: Laparoscopic repair of paraesophageal hernia: new access, old technique. Surg. Endosc. 9:164, 1995
- Heller, E.: Extanmukose karkioplastic beim chronisken kardiospasmus mit dilatation des oesophagus. Mitt. Grenzgeb. Med. Chir. 27:141, 1914

- Groeneveldt, F.R.B.: Overcardiplasticck. Ned. Tijdschr. Geneeskd. 62:1281, 1918
- Pasrich, P.J., Ravich, W.J., Hendrix, T.R., Sostre, S., Jones, B., Kalloo, A.N.: Intrasphincteric botulinum toxin for treatment of achalasia. Gastroenterology 111:1418, 1996
- Annese, V., Basciani, M., Perri, F., Lombardi, G., Frusciante, V., Simone, P., Andriulli, A., Vantrappen, G.: Controlled trial of botulinum toxin injection versus placebo and pneumatic dilation in achalasia. Gastroenterology *111*:1418, 1996
- Reynolds, J.C., Parkman, H.P.: Achalasia. Gastroenterol. Clin. North Am. 18:223, 1989
- Anselmino, M., Perdikis, G., Hinder, R.A., Polishuk, P.V., Wilson, P., Terry, J.D., Lanspa, S.J.: Heller myotomy is superior to dilatation for the treatment of early achalasia. Arch. Surg. *132*:233, 1997
- Raiiser, F., Perdikis, G., Hinder, R.A., Swanstrom, L.L., Filipi, C.J., McBride, P.J., Katada, N., Neary, P.J.: Heller myotomy via minimalaccess surgery: an evaluation of antireflux procedures. Arch. Surg. 131:593, 1996
- Ancona, E., Anselmino, M., Zaninotto, G., Costantini, M., Rossi, M., Bonavina, L., Boccu, C., Buin, F., Peracchia, A.: Esophageal achalasia: laparoscopic versus conventional oen Heller-Dor operation. Am. J. Surg. 170:265, 1995
- Hunter, J.G., Trus, T.L., Branum, G.D., Waring, J.P.: Laparoscopic Heller myotomy and fundoplication for achalasia. Ann. Surg. 225:655, 1997
- Hunter, J.G., Swanstrom, L.L., Waring, J.P.: Dysphagia after laparoscopic antireflux surgery: the impact of operative technique. Ann. Surg. 224:51, 1996