A Study of 362 Consecutive Laparoscopic Nissen Fundoplications

C. T. Frantzides, MD, PhD, FACS
Professor of Surgery, University of Chicago
Chairman, Division of Surgery,
Louis A. Weiss Memorial Hospital
(773) 564-5926

Gastroesophageal reflux disease (GERD) affects approximately 40 million people in the United States.[1] Prior to the advent of laparoscopic surgery, the only patients referred for operative correction of GERD were those who had severe intractable reflux, usually with complication (e.g., stricture, aspiration, or epithelial dysplasia).[2] However, now that antireflux surgery may be performed with minimally invasive methods,[3-6] more patients are being referred earlier for operative correction of their reflux. This article reviews a series of 362 consecutive patients who have undergone laparoscopic Nissen fundoplication for GERD over a period of six years.

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Methods

A total of 362 patients (173 men, 189 women; average age, 48 years; age range, 11 to 78 years) were selected to undergo a laparoscopic Nissen fundoplication. Preoperative evaluation included esophagogastroduodenoscopy (EGD) and esophagogram. Individuals without evidence of esophagitis underwent 24-hour ambulatory pH monitoring. Patients with evidence of dysmotility on esophagogram or who reported symptoms of dysphagia or odynophagia, also underwent manometry. Fifty-eight patients had preoperative manometry, and 39 had postoperative manometry. All patients had GERD, 128 (35.3 percent) had associated hiatal hernias, and 41 (11.3 percent) underwent another laparoscopic procedure at the same time (e.g., cholecystectomy, adhesiolysis, inguinal herniorrhaphy, liver biopsy, gastrostomy, or lymph node biopsy). A total of 114 patients (31.5 percent) had previous abdominal surgery.

The surgical technique used, though previously described,[3] is presented here in brief because of the introduction of newer instrumentation.

Technique

The patient is placed in a modified lithotomy position and the abdomen is prepped and draped in the usual sterile manner. A 1 cm incision is made below the left costal margin at the mid-clavicular line and the Optiview trocar (Ethicon Endosurgery, Cincinnati, Ohio) is used to enter the abdominal cavity. Carbon dioxide pneumoperitoneum is then established and maintained at 15 mm Hg. Four additional 10 to 11 mm trocars are placed: one in the right upper quadrant; one on the subcostal left anterior axillary line; one about 3 cm above the umbilicus in the mid-line; and one in the subxiphoid area. All trocars are placed under direct view and by transillumination of the abdominal wall. The laparoscope is introduced through the supraumbilical port. The left lobe of the liver is retracted cephalad and to the right with an inflatable balloon retractor introduced through the subxiphoid port (Soft Wand Retractor, Circon, Santa Barbara, California). Babcock forceps with atraumatic inserts (Pilling Weck, Inc., Research Triangle Park, North Carolina) is used through the left lateral port to retract the stomach caudad and to the left. The gastrohepatic ligament is then opened above the caudad lobe of the liver and the lesser sac is entered. The gastrohepatic ligament is separated up to the area of the esophageal hiatus. The phrenoesophageal ligament is divided, exposing the anterior wall of the esophagus.

Accurate identification of the esophagus is facilitated, especially in obese patients, with the introduction of a lighted bougie (Bioentrics Corp., Carpinteria, California) into the esophagus by the anesthesiologist. The esophagus is then safely mobilized primarily by blunt dissection using a palpation probe. During this dissection, the esophageal hiatus is exposed, and the right crus and posterior vagus are identified. We prefer to leave the posterior vagus attached to the esophagus so that injury or entrapment of the nerve can be avoided during cruroplasty. The next step in the procedure is to ligate the short

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Dr. Frantzides and his team perform a laparoscopic procedure.

gastric vessels. In order to facilitate division of these vessels, the stomach is retracted to the right by atraumatic Babcock forceps. The Harmonic scalpel (Ultracision Ethicon Endosurgery, Cincinnati, Ohio) is used for the division of the short gastric vessels beginning at a point high on the greater curvature and extending up to the gastroesophageal junction. A 30-degree laparoscope is used when dividing the short gastrics at the upper pole of the spleen.

Further mobilization of the esophagus is carried out and a window posterior to the esophagus is created. If necessary, depending on the presence of hiatal hernia, a posterior cruroplasty is carried out. Before the crural sutures are placed, a 50 French bougie is passed into the stomach. The sutures are placed from caudad to cephalad direction with generous bites of the left and right bundle of the right crus. A Babcock forceps is then passed posterior to the esophagus and the fundus is brought around the esophagus to form the wrap. A 3 cm long, 360-degree fundoplication is created loosely around the 50 French bougie with three interrupted #2-0 nonabsorbable sutures taking bites of the fundus. To prevent slippage of the wrap, the anterior arch of the crus is incorporated in the upper stitch. Before evacuation of the pneumoperitoneum, the fascial defects at the ports are closed using a fascial closer (Carter-Thomason, Eden Prairie, Minnesota). All operations were performed by the same surgeon.

Patients were discharged from the hospital with written instructions to avoid red meat for one week; carbonated beverages and gas-producing foods (e.g., beans, peas, broccoli, and onions) were to be avoided for two months. Individuals were also instructed to chew their food well, eat small meals the first few weeks, and avoid alcohol, citrus juices, and spicy foods. Postoperative pain was managed with acetaminophen/oxycodone and/or ketorolac.

Postoperative evaluation included an esophagogram and endoscopy at two to three months, with an esophagogram yearly thereafter. Fifty-one (14 percent) patients who had been found to have Barrett's esophagus underwent yearly endoscopy and biopsy. One of the patients with Barrett's esophagus progressed to severe dysplasia despite the absence of reflux and was treated with ablation by photodynamic therapy.

Results

With greater experience and improved instrumentation, the mean time of surgery decreased from 2.7±0.4 hours (during the period from 1991 to 1994) to 1.8±0.3 hours (from 1994 to 1997). During those same periods, days of hospitalization decreased from a mean of 2.2 days to a mean of 1.5 days.

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The conversion rate was 0.8 percent (n=3), with one of these conversions due to gastric perforation by Babcock forceps; the other two conversions were due to delayed gastric perforations. The complication rate was 1.9 percent (n=7) and included the three conversions, two pneumothoraces, one patient with postoperative bleeding who was managed expectantly, and one patient with a large abdominal wall hematoma at a trocar site, which resolved spontaneously.

There were five failures of the procedure (1.2 percent). Three of the failures were in patients with large hiatal hernia defects, one in a patient with persistent dysphagia, and one in a patient with a Nissen repair that "slipped," necessitating laparoscopic reconstruction.

Postoperative gastrointestinal symptoms were broken into two categories. The first category consisted of symptoms experienced only within the first two months. These included early satiety, bloating and flatulence, dysphagia, odynophagia, constipation, diarrhea, dry heaves, and nausea. Table 1 outlines the relative frequency of these postoperative symptoms.

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Table 1. Early Postoperative Gastrointestinal Symptoms

Symptoms	No. of Patients (%)	
Early Satiety	263	(72.6)
Bloating and Flatulence	147	(40.6)
Dysphagia	75	(20.7)
Constipation	28	(7.7)
Diarrhea	15	(4.1)
Odynophagia	13	(3.5)
Nausea	7	(1.9)
Dry Heaves	6	(1.6)

Table 2. Persistent Gastrointestinal Symptoms

Symptoms	No. of Patients (%)	
Bloating and Flatulence	8	(2.2)
Dysphagia	2	(0.5)
Diarrhea	3	(0.8)

The second category consisted of symptoms that persisted beyond two months and included bloating and flatulence, dysphagia, and diarrhea (Table 2). In the two patients with persistent dysphagia, one patient had a cruroplasty that was too tight; the cause in the other patient could not be determined. Extensive evaluation of the patients with diarrhea revealed no identifiable cause. One of these individuals had complaints of preoperative diarrhea that became worse after surgery.

The Figure demonstrates lower esophageal sphincter pressures measured in patients both pre- and postoperatively. Preoperative pressures were 4±1.2 mm Hg (n=58), compared with postoperative values of 14.6±1.8 mm Hg (n=39).

Discussion

This series of 362 patients presents some of the potential complications inherent in laparoscopic Nissen fundoplication. Experience has led to the development of various strategies to minimize the occurrence of these complications. Three patients developed perforations that required re-operation. One was recognized at the time of operation and was due to perforation by the Babcock forceps. This complication occurred early in the course of this series (ninth patient). The operation was converted to open and the defect sutured. It is likely that this perforation would now be repaired laparoscopically. Since that time, atraumatic Babcocks have been developed to prevent future perforations.

Two perforations had a delayed presentation (fourth postoperative day). One was repaired laparoscopically, the other through open surgery. Potential causes of delayed presentation include cautery injury with subsequent necrosis of the gastric wall, or stitches that were tied overly tight, resulting in necrosis.

Three patients with large (>8 cm) sliding hiatal hernias had a recurrence of the hernia within six months after laparoscopic repair. In an effort to reduce these recurrences we initiated the use of polytetrafluoroethylene (PTFE) prosthesis to reinforce the cruroplasty in large hiatal hernias.[7]

Some patients (n=75) reported dysphagia following operation, though only two of those reported symptoms that persisted beyond two months. Initial dysphagia may be due to edema, technical errors, or necessitated changes in diet for patients with a newly constructed wrap. What previously was a wide open canal may now require dietary modifications, including more thorough chewing of food.

Two patients suffered continued dysphagia beyond two months. Despite extensive evaluation that included esophagogram and manometry, one patient's dysphagia had no identifiable cause. Evaluation of the other patient demonstrated stenosis at the gastroesophageal junction. Review of the intraoperative videotape revealed a cruroplasty that was too tight. Laparoscopic removal of one of the cruroplasty stitches resulted in resolution of symptoms. In order to avoid too tight of a wrap, it has become our practice to use several strategies. In addition to performing the wrap and cruroplasty around a 50 French lighted bougie, the middle fundoplication stitch is placed first. This stitch is then elevated anterior and cephalad using grasping forceps, thus allowing

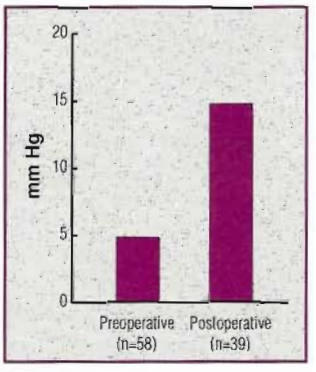

Figure. Mean lower esophageal sphincter pressures before and after laparoscopic Nissen fundoplication.

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evaluation of how tight the fundoplication is constructed; placement of subsequent stitches can be more exact, ensuring an optimal wrap. While this same method is used in patients with dysmotility for whom a fundopli-cation is indicated, we elect to perform a "floppy" Nissen around a 60 French bougie rather than employ a partial wrap, as advocated by other authors.[8]

In one instance, the failure of fundoplication was due to a "slipped" Nissen. In this case, a laparoscopic reconstruction was performed. While some authors advocate incorporating tissue from the gastroesopha-geal junction or esophagus into the wrap stitches,[9] studies in animal models at our institution have shown this to be ineffective (unpublished data). These anchoring esophageal stitches avulse the sutured esophageal muscle with potential for esophageal perforation. Alternatively, we incorporate the anterior arch of the crus into the uppermost stitch of the fundoplication to prevent slippage.

In conclusion, we have seen increasing experience and standardization of technique in the performance of laparoscopic Nissen fundoplication. These advances, coupled with the use of strict selection criteria, result in a surgical procedure that can provide both safe and effective results for patients with GERD.

For more information or to refer patients, call Dr. Frantzides at (773) 564-5926.

References

1. Landreneau RJ, Keenan RJ, Ferson PF. Gastroesophageal reflux disease. In: Cameron JL, ed. *Current Surgical Therapy.* St. Louis, MO: Mosby-Year Book, Inc.; 1995.
2. Rossetti ME, Liebermann-Meffert D. Nissen antireflux operation. In: Nyhus LH, Baker RJ, eds. *Mastery of Surgery.* Boston, MA: Little, Brown and Company; 1992.
3. Frantzides CT, Carlson MA. Laparoscopic versus conventional fundoplication. *J Laparoendosc Surg.*1995;5:137-143.
4. Hinder RA, Filipi CJ. Laparoscopic Nissen fundoplication. In: Frantzides CT, ed. *Laparoscopic and Thoracoscopic Surgery.* St. Louis, MO: Mosby-Year Book, Inc.; 1994.
5. Hinder RA, Filipi CJ, Wetscher G, et al. Laparoscopic Nissen fundoplication as an elective treatment for gastroesophageal reflux disease. *Ann Surg.* 1994;220:472-483.
6. Trus TL, Laycock WS, Branum G, et al. Intermediate follow-up of laparoscopic anti-reflux surgery. *Am J Surg.* 1996;171:32-35.
7. Frantzides CT, Carlson MA. Prosthetic reinforcement of posterior cruroplasty during paroscopic hiatal herniorrhaphy. *Surg Endosc.* 1997;11:769-771.
8. McKernan JB, Champion JK. Laparoscopic antireflux surgery. *Am Surgeon.* 1995;61:530-536.
9. Cuschieri A, Shimi S, Nathanson LK. Laparoscopic reduction, crural repair, and fundoplication of large hiatal hernia. *Am J Surg.* 1992;163:425-430.