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Laparoscopic Nissen Fundoplication Is an Effective Treatment for Gastroesophageal Reflux Disease

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Objective

The open Nissen fundoplication is effective therapy for gastroesophageal reflux disease. In this study, the outcomes in 198 patients treated with the laparoscopic Nissen fundoplication was evaluated for up to 32 months after surgery to ascertain whether similar positive results could be obtained.

Summary Background Data

To ensure surgical success, patients were required to have mechanically defective sphincters on manometry and increased esophageal acid exposure on 24-hour pH monitoring. The patients either had severe complications of gastroesophageal reflux disease or had failed medical therapy. These requirements have been found to be necessary to ensure a successful surgical outcome.

Methods

The disease was complicated by ulceration (46), stricture (25) and Barrett's esophagus (33). Patients underwent standard Nissen fundoplications identical in every detail to open procedures except that the procedures were carried out by the laparoscopic route.

Results

Perioperative complications included gastric or esophageal perforation (3), pneumothorax (2), bleeding (2), breakdown of crural repair (2) and periesophageal abscess (1). The only mortality occurred from a duodenal perforation. Six patients required conversion to the open procedure. The median hospital stay was 3 days. One hundred patients were observed for follow-up for 6 to 32 months (median 12 months), with outcomes similar to the open Nissen fundoplication. Further surgery was required for two patients who had recurrent gastroesophageal reflux and one who developed an esophageal stricture. Ninety-seven percent are satisfied with their decision to have the operation.

Conclusions

The laparoscopic Nissen fundoplication can be carried out safely and effectively with similar positive results to the open procedure and with all of the advantages of the minimally invasive approach.

Gastroesophageal reflux disease is a common condition in which the gastric contents gain access to the esophageal lumen or respiratory tract. Symptoms such as heartburn, regurgitation, dysphagia, and respiratory difficulty may occur. Damage to the esophageal mucosa may result in severe esophagitis, ulceration, strictures, or Barrett's esophagus. The lower esophageal sphincter often is mechanically incompetent in severe gastroesophageal reflux disease unresponsive to medical therapy.[1] Under these circumstances, medical therapy aimed at decreasing gastric acid output is ineffective, and surgical therapy, which improves the lower esophageal sphincter barrier, is a more logical and effective form of therapy.[2] Various surgical procedures have been devised, including the Nissen fundoplication, in which the fundus of the stomach is wrapped totally around the intra-abdominal esophagus, thereby increasing the intra-abdominal length of the sphincter, narrowing the sphincter and accentuating the angle of His.[3,4] Other procedures include the Toupet procedure, in which a 270-degree posterior partial fundoplication is performed; the Hill repair, in which the plicated proximal lesser curvature of the stomach is fixed to the preaortic fascia, and the Belsey procedure, which is performed via a thoracotomy, during which the cardia of the stomach is wrapped partially around the lower esophagus.[5-8] The Nissen fundoplication has proven itself to be a very effective form of therapy with excellent long-term results. Previously, we have shown that there is a 91% success rate in 100 patients observed for follow-up for 10 years after the open Nissen fundoplication.[9]

After the successful application of laparoscopy for biliary disease, other procedures that could be carried out by the laparoscopic approach were identified. It became clear that the abdominal esophagus and esophageal hiatus could be approached easily laparoscopically and that it was possible to carry out identical antireflux procedures to those that had been carried out previously by the open technique.[10] The open technique requires a hospital stay of up to 9 days and has significant morbidity, including wound sepsis, splenectomy, deep venous thrombosis, pulmonary complications, more pain, and a prolonged recovery compared with that of the laparoscopic approach.[11] Preliminary results with the laparoscopic Nissen fundoplication indicated that this procedure could be performed safely and effectively.[10] This led to experience with 198 patients who have been followed for 1 to 32 months.

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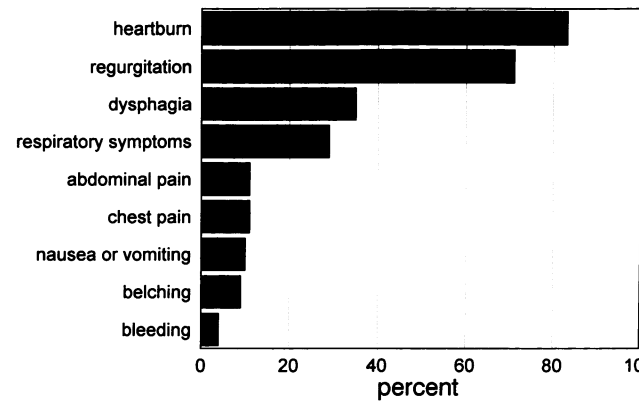


Figure 1. Incidence of presenting symptoms expressed as a percent of all patients.

PATIENTS AND METHODS

Between July 1991 and February 1994, 204 patients were referred to the Creighton University School of Medicine for surgical therapy of gastroesophageal reflux disease. Two patients—each with a hiatal hernia exceeding 5 cm in length on fluoroscopic examination in which there was suspected esophageal shortening—were excluded from this study, and a transthoracic approach was advised. Four patients with poor esophageal body motor function also were excluded and were continued on medical therapy or were subjected to a partial fundoplication of the Toupet type. The remaining 198 patients had laparoscopic Nissen fundoplications; they comprise the patients of this study. There were 122 men and 76 women. The median age of the patients was 48 years (range 11–80 years). The median weight was 184 pounds (range 67–320 pounds). The presenting symptoms included heartburn, regurgitation, dysphagia, respiratory symptoms, abdominal pain, chest pain, nausea or vomiting, belching, or evidence of gastrointestinal bleeding (Fig. 1). Symptoms were present for a median of 72 months (range 3–600 months). All of the patients underwent upper gastrointestinal endoscopies and biopsies, upper gastrointestinal roentgenographies, esophageal manometries using the station pull-through perfused catheter technique and 24-hour ambulatory esophageal pH measurement. The technique of manometry and 24-hour pH measurement have been described previously.[12,13] Thirty-three patients were found to have Barrett's esophagus, 46 had evidence of esophageal mucosal ulceration, and 25 had esophageal strictures that were dilated before surgery. The grade of esophagitis, scaled according to the Savary-Miller classification, is shown in Figure 2.[14] Thirty-three patients had radiologic evidence of a hiatal hernia 3 to 5 cm in length and considered to be reducible. Ten patients with histories of peptic ulcer disease or abnormally high esophageal acid

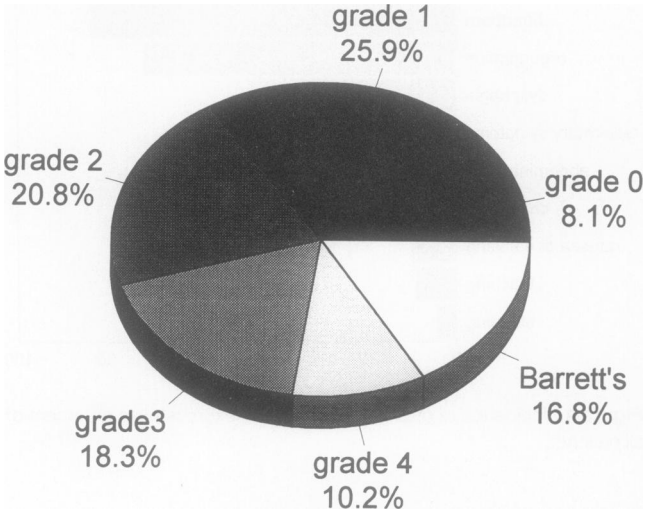


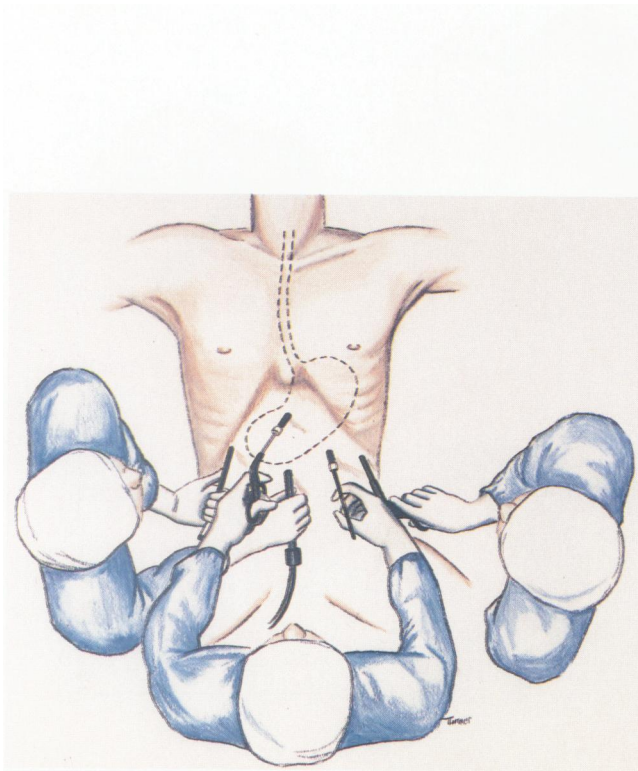
Figure 2. Preoperative grade of esophagitis in the patients, according to the Savary-Miller classification.[14]

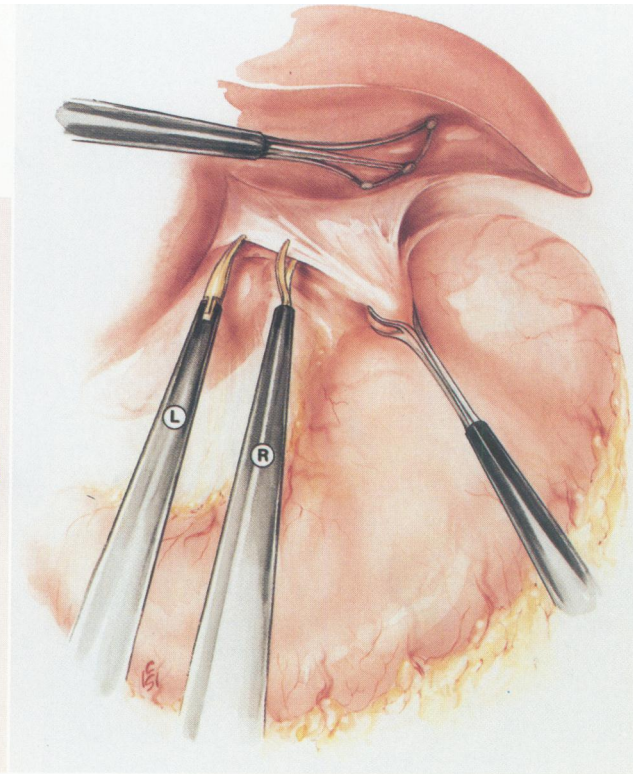
exposures in the presence of normal lower esophageal sphincter function also had gastric acid analyses. In six of these patients shown to have a high gastric acid output, a concomitant highly selective vagotomy was carried out together with the antireflux procedure. Otherwise, the indication for surgery was chronic gastroesophageal reflux disease unresponsive to medical therapy, with a poorly functioning lower esophageal sphincter and abnormal exposure of the esophagus to pH <4. Fifty-four patients had had previous abdominal surgery including cholecystectomies, appendectomies, colectomies, and gynecologic procedures.

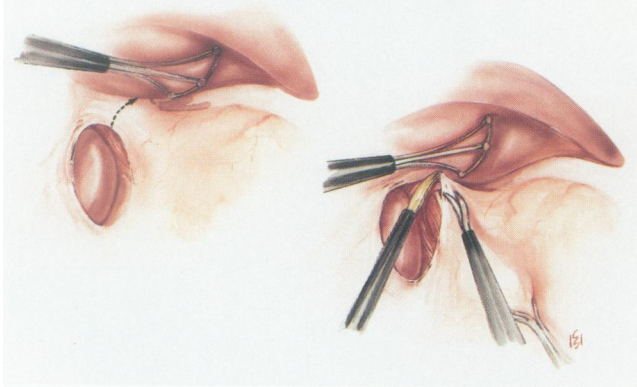
OPERATIVE PROCEDURE

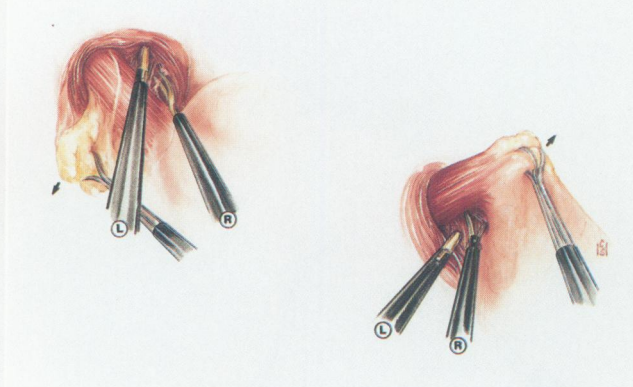
The procedure is carried out using general anesthesia, with the patient in the lithotomy position and in the steep reverse Trendelenburg position. The operating team is positioned as shown in Figure 3. The best position for the surgeon was found to be between the legs of the patient, to allow comfortable access to the hiatal area without having to twist the body or lean across the table. Ten-millimeter ports are placed as shown in Figure 3 after a pneumoperitoneum is produced using a Verres needle introduced through a small supraumbilical incision. The Hasson cannula was used in three cases in which adhesions from previous surgeries were expected in the upper abdomen. The assistant on the right-hand side of the patient maneuvers the camera and retracts the liver. The assistant on the left-hand side of the patient retracts the stomach and elevates the esophagus, allowing the surgeon to gain access to the area behind the esophagus.

The first step of the procedure is to divide the gastrohepatic omentum along the upper lesser curvature of the stomach (Fig. 4). This should be done over a short distance to avoid damage to the hepatic branches of the vagus nerve. In 12% of patients, an aberrent left hepatic artery was discovered and was preserved intact in 21 of 24 patients. In the remaining three patients, the vessels obstructed the view to such an extent that they required ligations with silk loop ties and were divided. The next step is to retract the stomach to the left and expose the right crus of the diaphragm. The anterior edge of the right crus can be identified easily by incising the overlying peritoneum. The crus can be peeled gently off the esophagus, allowing access to the mediastinum around the esophagus (Fig. 5). The stomach is retracted to the right-hand side, and the left crus is identified similarly and dissected and removed from the esophagus (Fig. 6). At this time, it is important to dissect the free edge of the left crus well behind the esophagus to prepare for the establishment of the window behind the esophagus. The esophagus is elevated, and the posterior vagus nerve can be identified easily behind the esophagus. This is dissected gently and removed from the esophagus, and a window is created anterior to the nerve and posterior to the esophagus. The window is made in the connective tissue immediately inferior to the left crus of the diaphragm and allows access to the peritoneal cavity superior to the fundus of the stomach. Great care should be exercised in making this opening under clear vision without damaging the posterior wall of the stomach or esophagus. If the dissection is performed too high, the left pleural cavity may be entered, and a pneumothorax may be created. Extensive dissection into the mediastinum may result in air tracking into the neck, but this usually is of little consequence and quickly resorbs. This occurred in two cases. Once the window posterior to the esophagus is created, it can be widened by further dissecting the posterior vagus nerve and removing it from the lower esophagus. A small branch of the posterior nerve passing onto the back of the esophagus may have to be divided to facilitate this maneuver. The left and right crura should be stripped of their surface connective tissue in preparation for the crural closure behind the esophagus and in front of the posterior vagus nerve (Fig. 7). This usually is accomplished using one to three 2-O Prolene sutures passed through the muscle bundles of the crura. The first stitch is placed posteriorly as far as possible. The stitches are placed to snugly close the hiatus about the esophagus, which contains a size 58 to 60 French Maloney bougie. The bougie then is retracted into the thoracic esophagus, and the upper 10 cm of the greater curvature of the stomach is mobilized by clipping and dividing short gastric vessels and peritoneal attachments to the spleen and diaphragm (Fig. 8). It usually is not necessary to carry out this dissection all the way to the angle of His because the









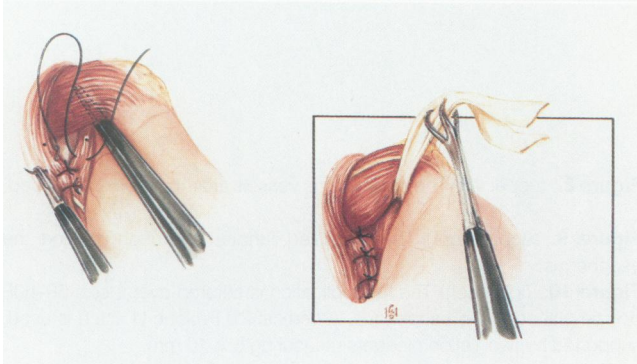


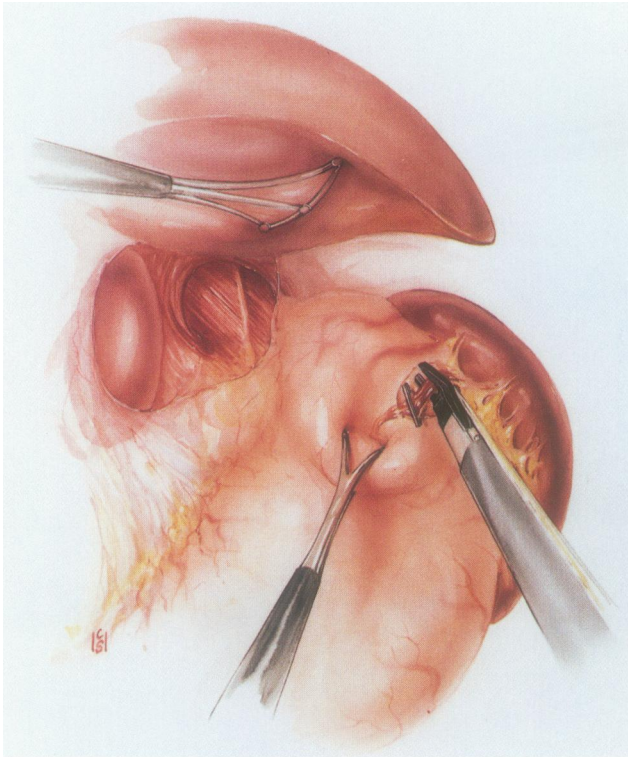
Figure 3. (upper left) Position of the surgical team with the patient in the lithotomy position in steep reverse Trendelenburg. Position of the five 10-mm ports is shown.

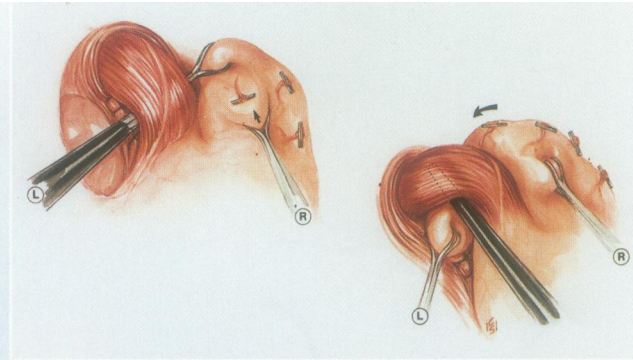
Figure 4. (upper right) The gastrohepatic omentum is divided.

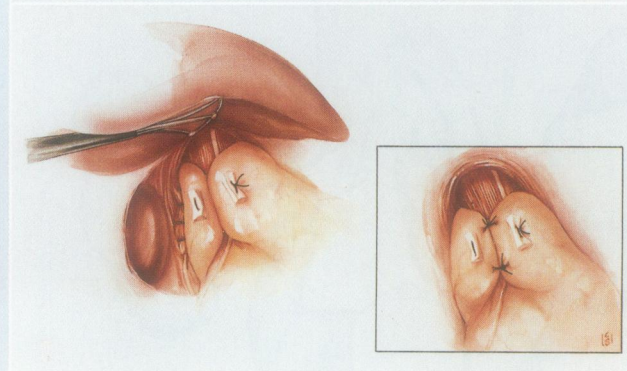
Figure 5. (middle left) The gastrohepatic omentum is further incised to expose the right crus of the diaphragm and the front of the esophagus.

Figure 6. (middle right) The stomach is retracted to the patient's right-hand side and the left crus of the diaphragm is dissected off the esophagus as far posteriorly as possible. A window is then created behind the esophagus, and the esophagus can be elevated to allow further dissection of the posterior vagus nerve and the left crus of the diaphragm.

Figure 7. (lower left) The crura are approximated posterior to the esophagus and anterior to the posterior vagus nerve. A penrose drain may be used to retract the esophagus.







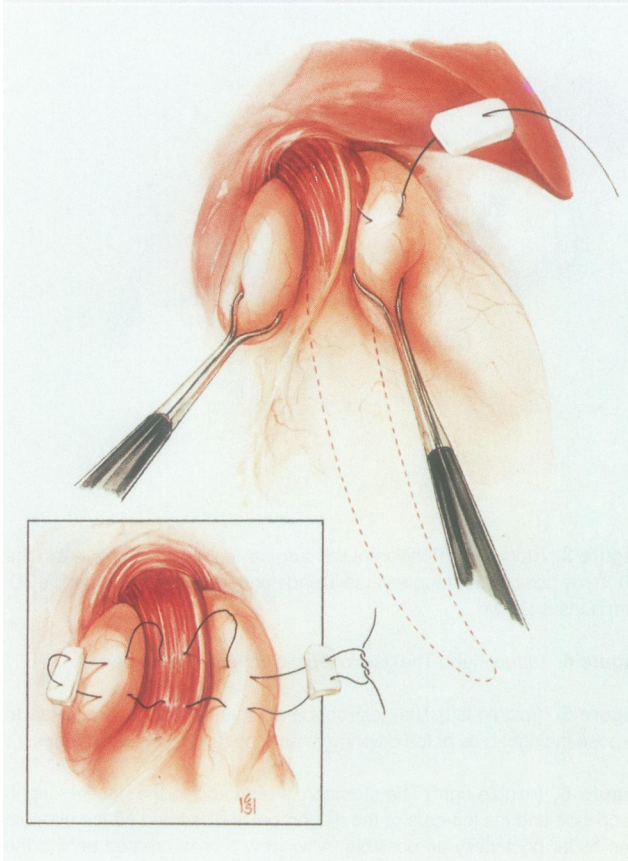


Figure 8. (upper left) The short gastric vessels are mobilized and divided.

Figure 9. (upper right) The mobilized fundus is brought behind the esophagus.

Figure 10. (lower left) The fundoplication is sutured over a size 58–60F bougie placed in the esophagus. A vertical 2'0 Prolene U stitch is used, supported by two Teflon pledgets measuring 5 × 10 mm.

Figure 11. (lower right) The completed fundoplication. One or two additional simple sutures may be placed as shown.

Table 1. COMPLICATIONS OF LAPAROSCOPIC NISSEN FUNDOPLICATION

Complication	No. (%)
Gastric or esophageal perforation	3 (1.5)
Pneumothorax	2 (1)
Bleeding	2 (1)
Breakdown of crural repair	2 (1)
Urinary tract infection	2 (1)
Duodenal perforation day 4 with ARDS	1 (0.5)
Periesophageal subphrenic abscess	1 (0.5)
Pneumonia	1 (0.5)
Bacteremia with jaundice	1 (0.5)

ARDS = adult respiratory distress syndrome.

most proximal 1 to 2 cm of the fundus does not require displacement very far behind the esophagus in performing the fundoplication. A Babcock grasper is passed from the right subcostal port behind the esophagus to grasp the mobilized fundus, which is then pulled through the window behind the esophagus (Fig. 9). The bougie is replaced in the lower esophagus, and the wrap is fixed snugly around the bougie contained in the esophagus. It is important that a contiguous part of the fundus of the stomach is used for the fundoplication to avoid an excessively loose wrap (Fig. 10). A U-shaped stitch of 2-0 Prolene with two Teflon pledgets on the outer surface of each limb of the wrap is used to secure the fundoplication. An additional one or two simple Prolene sutures may be placed above or below the U-stitch, as required (Fig. 11). The bougie then is replaced into the stomach to finally check the tightness of the wrap. The pneumoperitoneum is released, and the ports are removed. The median operative time was 2.5 hours (range 1.4–6 hours) and the median blood loss was 50 mL (range 10–2000 mL).

OPERATIVE COMPLICATIONS

The operative complications are listed in Table 1. In the early experience, a small gastric perforation in two cases and an esophageal perforation in another case were repaired immediately after conversion to open procedures. All three patients made uneventful recoveries. Pneumothoraxes occurred in two cases and did not interfere with the laparoscopic procedures. No chest drains were required. Bleeding from a short gastric vessel required conversion to the open procedure in one patient. Another patient had a decrease in hemoglobin percentage from 13 g down to 7 g during the first 24 hours after surgery and was the only patient who required a blood transfusion. There were no splenic injuries. The need for conversion to open procedures occurred in 4 of the first 30 patients and in only 2 of the subsequent 168 patients. This was done because of esophageal or gastric perforations, bleeding from a short gastric vessel, bleeding from the cystic artery during cholecystectomy, and difficulty in dissecting the esophageal hiatus. This indicates the prolonged learning curve of the procedure. However, once mastered, the procedure can be accomplished without undue difficulty, even in the most obese patients. Previous abdominal surgery was not found to be a contraindication for the laparoscopic approach.

CONCOMITANT LAPAROSCOPIC PROCEDURES

Other concomitant laparoscopic procedures were done for 25 patients (Table 2). Three of the first 100 patients returned with acute attacks of cholecystitis or biliary pancreatitis within the first year after surgery. This prompted us to do routine preoperative ultrasonographies of the gallbladder in the subsequent cases and to perform laparoscopic cholecystectomies at the same time as the laparoscopic Nissen fundoplications in those patients in whom gallstones were found to be present. Fourteen patients had gallstones shown on ultrasound examination of the abdomen and had a concomitant laparoscopic cholecystectomy. Six had a highly selective vagotomy, three had a laparoscopic repair of an inguinal hernia and one had a laparoscopic repair of an incidentally found Morgagni's hernia using Prolene mesh. In one patient who was mentally impaired, a gastrostomy tube was placed.

EARLY POSTOPERATIVE CARE

In the first 41 cases, nasogastric tubes were left in the stomach until the next morning. This was omitted in the next 157 cases and led to no untoward problems. In the early experience and in patients in whom there were any surgical difficulties, a gastrografin swallow was done the morning after surgery to confirm that there was no injury to the esophagus or stomach and that the fundoplication was in good position. This was done in 86 cases, and no

Table 2. OTHER SIMULTANEOUS LAPAROSCOPIC PROCEDURES

Procedure	No.
Cholecystectomy	14
Highly selective vagotomy	6
Inguinal herniorrhaphy	3
Gastrostomy	1
Morgagni hernia repair	1

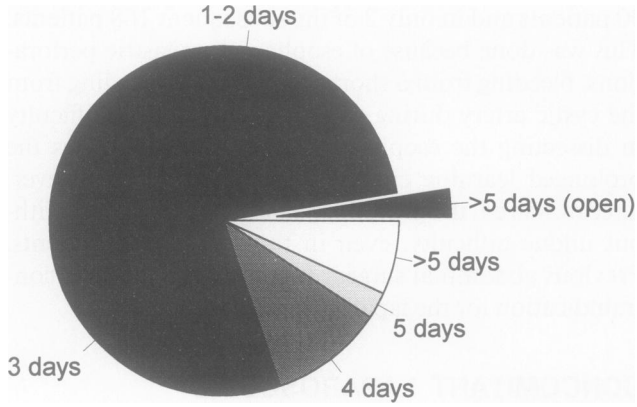


Figure 12. The length of hospital stay for all patients. All patients converted to the open procedure were in the hospital for > 5 days.

leaks or other problems were detected. Patients were given small amounts of clear liquid on the same evening of surgery and were offered a light breakfast of scrambled egg and cereal on the morning after surgery. The median hospital stay was 3 days (range 1–26 days) (Fig. 12). In the later experience most patients left hospital after 2 days. Patients were encouraged to eat a soft diet for the first 2–3 weeks after surgery and were told to avoid dry bread and chunky food such as steak for this period. The median time for return to work was 3 weeks.

POSTOPERATIVE COMPLICATIONS

Two patients required antibiotics for urinary tract infections and one required an emergency transurethral prostatectomy 6 days after the Nissen fundoplication. Another patient required nasogastric suction for 6 days for a prolonged ileus, and another had a bacteremia associated with bilirubinemia, which resolved with antibiotic therapy. One patient developed postoperative pneumonia. There was one death—i.e., a 68-year-old woman had a duodenal perforation on the fourth postoperative day, with subsequent adult respiratory distress syndrome. A 67-year-old patient on steroids developed a small periesophageal subphrenic abscess—not associated with bowel perforation—which was drained successfully. She made a good recovery. Two patients developed acute paraesophageal hernias of the fundus of the stomach through the esophageal hiatus on the third and fifth days after surgery. One of these was repaired laparoscopically and the other was associated with a small bowel obstruction at a port site and was repaired at laparotomy.

POSTOPERATIVE FOLLOW-UP

Patients were seen in the clinic at 1, 3 and 5 weeks after surgery. Dysphagia was the most common early symptom after surgery and occurred in 23% of patients. This resulted in the need for esophageal dilatation in 2 patients on one occasion, 3 patients on two occasions, 3 patients on three occasions, 1 patient on four occasions and 1 patient on five occasions. Postoperative foregut symptoms required upper gastrointestinal endoscopy in 16 patients. Nine were found to be normal, 6 had evidence of gastritis and one had a prepyloric gastric ulcer which healed on medical therapy.

Three patients have required subsequent reoperation. One of the early patients had recurrent reflux symptoms and was found at laparotomy to have had the wrap incorrectly placed. One patient who in retrospect had a short esophagus preoperatively, required a thoracotomy and Collis-Nissen repair 4 months after surgery. One patient with persistent dysphagia underwent open reoperation in another state without having attempted dilatation.

The first one hundred patients were surveyed 6–32 months after surgery and answered the questions shown in Table 3. Ninety-seven of the 100 reported that they were satisfied with their decision to have surgery and none of the patients require to take medication for symptoms of reflux disease.

FOLLOW-UP ESOPHAGEAL MANOMETRY AND 24-HOUR pH STUDIES

Fifty-one patients volunteered to have repeat esophageal manometry, and 24 patients, 2 of whom had recurrent reflux symptoms, agreed to 24-hour esophageal pH studies 3 to 12 months after surgery. The results of these studies are shown in Figures 13 through 16. There was a significant increase in resting pressure of the lower esophageal sphincter, with a significant decrease in its intrathoracic length and increase in its intra-abdominal length. The postoperative lower esophageal sphincter pressure was in the normal range in 49 of 51 patients

Table 3. RESULTS 6 TO 32 MONTHS AFTER SURGERY (N = 100)

	Absent	Mild	Moderate	Severe
Dysphagia	69%	25%	6%	0%
Nausea	92%	3%	5%	0%
Bloating	64%	23%	12%	1%
Reflux symptoms	92%	7%	1%	0%
Diarrhea	80%	11%	9%	0%
(preoperative)	(85%)		(15%)	
Early satiety	51%	0%	49%	0%
Dietary restriction	98%	2%	0%	0%

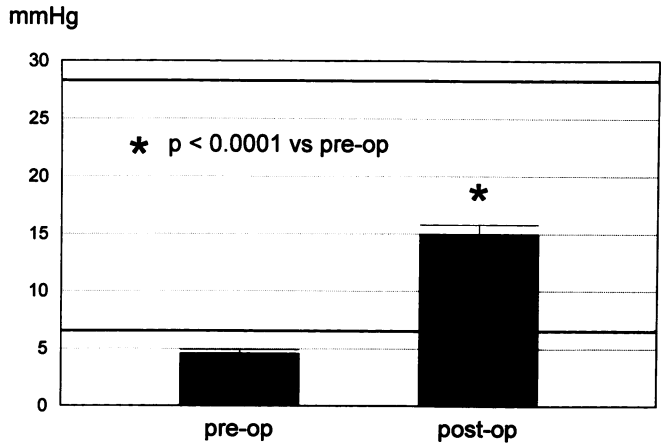


Figure 13. Lower esophageal sphincter manometry (mm Hg ± SEM) before and after laparoscopic Nissen fundoplication, showing a significant increase in resting sphincter pressure after the procedure. The 2.5th–97.5th percentile for normals lies between 6–28 mm Hg.

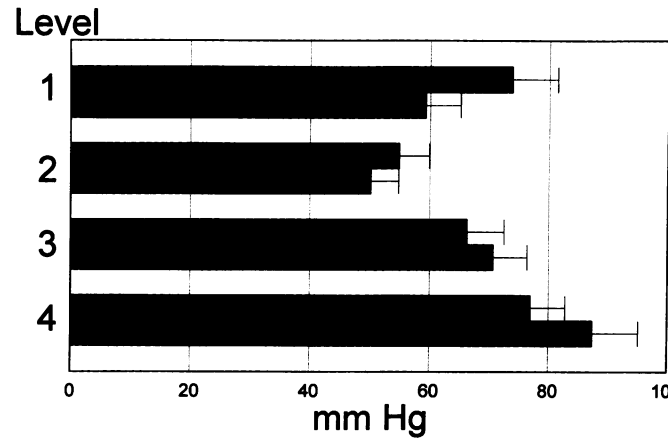


Figure 15. No significant change in esophageal body pressure during swallowing in the upper level down to the lower level of the esophagus comparing preoperative to postoperative results. There also was no difference in the incidence of simultaneous or abnormal waves.

tested. There were no significant changes in the manometric characteristics of the esophageal body (Fig. 15). Twenty-one of 24 patients had no evidence of abnormal acid exposure in the esophagus after surgery (Fig. 16). Two of the three who tested positive were operated on again for recurrent reflux disease.

DISCUSSION

The medical management of gastroesophageal reflux disease often is ineffective; most patients who respond to initial medical therapy relapse after one year.[2,15] The reason for this is that medical therapy directed against gastric acid secretion does not address the major underlying problem, which is a mechanically ineffective antireflux barrier at the lower esophagus.[1] A recent comparison of medical and surgical therapy indicated the superiority of surgical therapy in controlling this disease.[2] The current study of laparoscopic Nissen fundoplication has shown the effectiveness of surgery in controlling this disease in carefully selected cases. It is not surprising that the results after the laparoscopic approach were very similar to the outcome after the open procedure because exactly the same procedure was carried out in both. The operative mortality of 0.5% is very similar to many studies of the open Nissen fundoplication, in which mortality rates of 0 to 2% are reported.[3,4,7–9,11] However, there were some remarkable differences. This study and the experience of others with the laparoscopic Nissen fundoplication have shown an absence of the need for splenectomy.[16] With the open procedure, splenectomy is required in about 2% of cases.[11] Other advantages are the

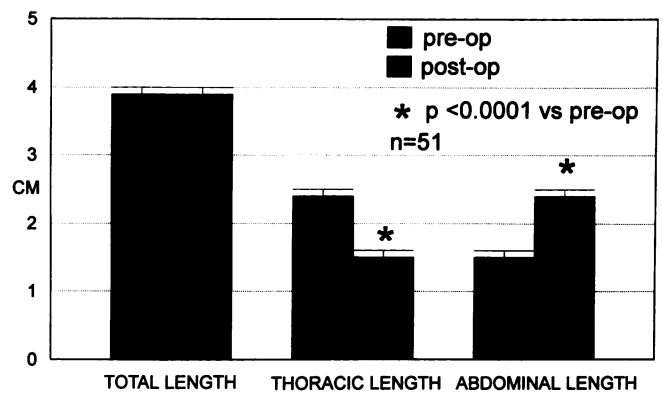


Figure 14. Length of the lower esophageal sphincter before and after laparoscopic Nissen fundoplication, showing a significant decrease in the intrathoracic length, with a significant increase in the intra-abdominal length after surgery.

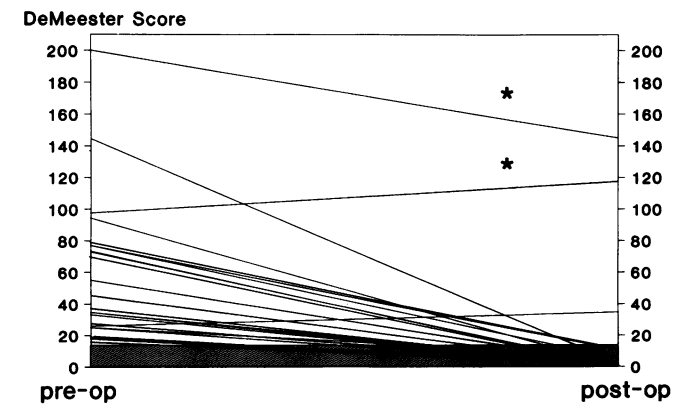


Figure 16. The 24-hour esophageal pH composite score[13] before and after laparoscopic Nissen fundoplication. Normal score <14.8. Twenty-one of 24 patients had a normal score after surgery. Two of those with abnormal results after surgery had recurrent disease (*).

virtual absence of wound sepsis, pulmonary complications, deep venous thrombosis, and incisional hernias, which occur in up to 10% of open cases and may require further surgery. As proficiency is acquired with the laparoscopic approach, even the most obese patients can undergo operations with relative ease. The greatest risk of the laparoscopic procedure is an unsuspected perforation of the esophagus or stomach. An incidence of unsuspected bowel perforation in approximately 2% of cases is reported with the open procedure and has a high mortality.[11] It is necessary to be particularly aware of this complication and respond promptly to early postoperative signs or symptoms suggestive of peritoneal sepsis.

This series has again shown that a good result from surgical therapy can be anticipated for patients who are unresponsive to medical therapy and who have a mechanically incompetent lower esophageal sphincter, good esophageal body function, and evidence on pH testing of abnormal acid exposure in the esophagus. The mere presence of a hiatal hernia and a history suggestive of gastroesophageal reflux disease is not sufficient evidence to recommend surgery. Conditions such as scleroderma, esophageal body motility disorders, disorders of gastric motility, and pill-induced esophagitis, may have similar symptoms to those of gastroesophageal reflux disease, but these patients would be poorly served by an antireflux procedure. Excellent results can be achieved by insisting on strict standards of investigation. Should there be evidence of esophageal shortening, difficulty may be experienced in returning the lower esophageal sphincter to the abdomen. There may be axial tension on the stomach within the wrap and a "slipped Nissen" may result. To avoid this complication, these patients should be offered a transthoracic approach with extensive mobilization of the esophagus and perhaps esophageal lengthening in the form of a Collis gastroplasty.

Despite the fact that the crura were approximated behind the esophagus in all cases, there was evidence of breakdown of the crural repair with herniation of the upper stomach into the chest in the early postoperative period in two patients. Should the crura have not been closed, the incidence of this complication would have been even greater. The avoidance of gastric decompression in the early postoperative period may have contributed to this complication. The fundus of the stomach should be freed to allow it to lie comfortably behind the esophagus without tension. Should there be tension on the wrap, this could result in torsion on the esophagus, with possible dysphagia or disruption of the wrap. We have shown that a short wrap measuring less than 2 to 3 cm is sufficient to maintain the antireflux properties of the Nissen fundoplication. Previously, it has been shown that a longer wrap will result in a high incidence of dysphagia.[17] In patients with gastric acid hypersecretion who have prior histories of peptic ulceration or who have high acid exposure in the esophagus in the presence of normal lower esophageal sphincter pressure, a concomitant highly selective vagotomy should be performed. The steps of the laparoscopic vagotomy are essentially the same as for the open procedure, with dissection of the anterior and posterior vagal nerve trunks and the creation of a window into the lesser sac along the lesser curvature of the stomach.[18] Because the lesser curvature of the stomach has been denuded of its blood supply, we do not favor dividing short gastric vessels in these cases before performing the fundoplication. It may be more appropriate in this case to do a partial posterior fundoplication of the Toupet type if the fundus does not easily come around the esophagus in the form of a 360-degree Nissen fundoplication. We always were able to easily identify the posterior vagus nerve but did not deliberately dissect the anterior vagus nerve and remove it from the esophagus. The anterior nerve remains closely adherent to the esophagus and is enclosed within the wrap. Placing the fundoplication between the posterior vagus nerve and the esophagus aids in preventing later slippage of the fundoplication.

Others have reported the ability to perform different antireflux procedures via the laparoscopic route. It is possible to perform the Hill procedure or the Toupet procedure. This approach also has been used successfully for the laparoscopic Heller myotomy for achalasia of the esophagus.[19]

This study has shown the ability to perform a conventional Nissen fundoplication using the laparoscopic approach. The early results after this procedure are at least as good as those reported for the open procedure. There is a high acceptance rate of the laparoscopic approach by patients and gastroenterologists because of the shorter hospitalization, less pain, fewer side effects, and a more rapid return to normal activities. However, the procedure is technically challenging and is potentially dangerous for surgeons who have not undergone proper training and proctoring in advanced laparoscopy. The laparoscopic Nissen fundoplication is our preferred method for the surgical treatment of gastroesophageal reflux disease, in which medical therapy has failed and offers all of the advantages of the minimally invasive approach.

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Discussion

DR. LUCIUS D. HILL (Seattle, Washington): The authors state that their goal is to see if they can duplicate the long-term results of the open procedure, so I think it's worthwhile to take a look at this goal.

There's been a recent report in the New England Journal of Medicine concerned with the randomized trial done by the VA System across the United States. It was interesting that the gastroenterologists had to admit that surgery was superior to medicine but both treatments were effective.

But let's look at the surgical series because it's a good assessment of the long-term results of the Nissen procedure. The procedure that was used in that randomized trial was the two-stitch Nissen quite similar to what the authors have just reported.

The results of the Nissen in this study showed a 15% operative complication, an 18% immediate postoperative complication, and 81% over the long term who had more than one surgical symptom. Now, I would agree with the authors that this is an attainable goal, but I wonder if it is a desirable one.

Another assessment of the Nissen procedure recently was reported by Thor from Stockholm in which they did a randomized trial of the Nissen *versus* the Toupet. At the end of 5 years the good-to-excellent results of the Toupet procedure were 95% and the Nissen, 67%.

Meanwhile our group, in concert with Snow of Alabama, has done 141 patients laparoscopically. We did 33 pigs before we ever did a human, so that we were fairly comfortable when we started doing the laparoscopic procedure on humans.

And I would admonish anyone who is going to do this to either work with someone who has done it or do some pigs, because it's quite different from the laparoscopic cholecystectomy. There's a good deal more suturing and you're working in an area that's a little more difficult to expose than the gallbladder.

So far we have had no mortalities nor perforations. We have dilated six patients postoperatively; only one of these has persistent dysphagia and this one is a fairly recent one. We have minimal reflux in three patients; two of these were asymptomatic but one has symptoms of heartburn. We have had to redo no patients to date.

So in light of these long-term results with the Nissen, I wonder if the authors had considered doing some alternative procedure. I notice that they have done some Toupet procedures.

If they do not like our reconstruction of the gastroesophageal junction, perhaps a Toupet procedure would give the patient a little better opportunity to face the future with the prospect of doing better than the 81% with more than one surgical complication.

DR. TOM R. DEMEESTER (Los Angeles, California): I rise to place this paper in perspective from the standpoint of how the study started. The decision to start performing laparoscopic Nissen fundoplications at Creighton University took place in the latter part of 1990, when Dr. Hinder and Dr. Filipi discussed with me the reports they were hearing about Nissen fundoplication being done in Belgium by Dr. Dellamagne. They suggested that we should look into the possibility. I was resistant and reluctant because of my commitment to the open Nissen, but suggested that they go to Belgium to take a look. They went, and came back enthusiastic and advised that we should begin doing the procedure.

A decision was made that the same inclusion criteria used for the open procedure would be used for the laparoscopic approach and that the laparoscopic procedure would be done exactly as we were doing the open procedure. With that agreed to, they began, and I moved to Los Angeles.

We kept in contact and after Ron [Hinder] had done his 100th patient, I became convinced that they were having the same outcome that we had with the open procedure, both from the measurement of symptomatic and functional improvement. They were achieving the same augmentation in lower