

Short-Term Outcomes Using Magnetic Sphincter Augmentation Versus Nissen Fundoplication for Medically Resistant Gastroesophageal Reflux Disease

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Background. In 2012 the United States Food and Drug Administration approved implantation of a magnetic sphincter to augment the native reflux barrier based on single-series data. We sought to compare our initial experience with magnetic sphincter augmentation (MSA) with laparoscopic Nissen fundoplication (LNF).

Methods. A retrospective case-control study was performed of consecutive patients undergoing either procedure who had chronic gastrointestinal esophageal disease (GERD) and a hiatal hernia of less than 3 cm.

Results. Sixty-six patients underwent operations (34 MSA and 32 LNF). The groups were similar in reflux characteristics and hernia size. Operative time was longer for LNF (118 vs 73 min) and resulted in 1 return to the operating room and 1 readmission. Preoperative symptoms were abolished in both groups. At 6 months or longer postoperatively, scores on the Gastroesophageal Reflux Disease Health Related Quality of Life scale

improved from 20.6 to 5.0 for MSA vs 22.8 to 5.1 for LNF. Postoperative DeMeester scores (14.2 vs 5.1, $p = 0.0001$) and the percentage of time pH was less than 4 (4.6 vs 1.1; $p = 0.0001$) were normalized in both groups but statistically different. MSA resulted in improved gassy and bloated feelings (1.32 vs 2.36; $p = 0.59$) and enabled belching in 67% compared with none of the LNFs.

Conclusions. MSA results in similar objective control of GERD, symptom resolution, and improved quality of life compared with LNF. MSA seems to restore a more physiologic sphincter that allows physiologic reflux, facilitates belching, and creates less bloating and flatulence. This device has the potential to allow individualized treatment of patients with GERD and increase the surgical treatment of GERD.

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Chronic gastroesophageal reflux disease (GERD) occurs in patients as a spectrum of disease that ranges from the endoscopically normal esophagus to erosive esophagitis and to Barrett's esophagus [1]. This is influenced by a hiatal hernia of varying sizes, a stricture of varying degrees (rings to fibrotic), and the potential for a foreshortened esophagus. Despite the wide variation, only two dominant therapies have been used to treat the entire spectrum of GERD during the past 70 years: Nissen fundoplication and proton pump inhibitors (PPIs). Both treatments are effective at controlling GERD, with a slight advantage toward operative treatment based on two randomized control trials [2, 3].

Despite similar outcomes, there is a large gap in the use of both treatments. Using current rates of antireflux operations, it is estimated that surgical repair is used in less

than 1% of patients [4]. Even though PPIs are the dominant therapy, only 60% of patients are satisfied with their treatment [5]. This leaves a therapy gap of at least 40% of patients who are taking PPIs with ongoing GERD symptoms. These patients are either not being referred for an equally effective therapy or have chosen not to undergo surgical treatment. The reasons for this include concerns about the ability to belch or vomit and the development of hyperflatulence or bloating [2, 6]. Furthermore, there are concerns about the perceived invasiveness and durability of the surgical outcomes because upwards of 25% of repairs will deteriorate over time [2]. This creates an opportunity for the development of new treatments.

In March 2012, the United States Food and Drug Administration approved a novel device to control GERD composed of a series of magnets set in a titanium casing and connected by titanium wires interconnected with a hollow housing in the configuration of a Roman arch

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(Fig 1). This “sphincter,” by virtue of the magnets, is potentially durable for the life of the patient and would augment the lower esophageal sphincter by limiting lower esophageal shortening and relaxations during gastric distension but open to gastric pressure to allow belching, and thus prevent hyperflatulence and bloating. One small pilot trial [7] and two single-series trials [8, 9] comprising 244 patients have demonstrated its initial efficacy. However, no comparison with standard treatments has been performed and is necessary. We evaluated our experience with magnetic sphincter augmentation (MSA) and compared it with laparoscopic Nissen fundoplication (LNF) at 6 months.

Material and Methods

We retrospectively reviewed prospectively collected data on consecutive patients who underwent laparoscopic implantation of a magnetic sphincter at Swedish Medical Center from September 2012 to December 2013. The Institutional Review Board of Swedish Medical Center approved this study and waived the need to implant the devices under a research protocol. Magnetic sphincters were placed as part of clinical care, and patient consent was provided for implantation; however, individual patient consent for this study was waived because of the study’s retrospective nature.

For comparison, we reviewed 427 primary antireflux repairs from a prospectively maintained benign esophageal surgical database from January 2010 to July 2013 to identify consecutive patients undergoing LNF. We excluded patients based on the following criteria: age younger than 18 years, body mass index exceeding 36 kg/m^2 , hiatal hernia exceeding 3 cm in axial length without a paraesophageal component, Barrett’s esophagus exceeding 1 cm, and named motility disorders.

We identified 98 patients, with 50 excluded because they were part of another clinical trial and a further 16 patients excluded when preoperative video esophagograms and endoscopic photos showed the hiatal hernias were too large. Ten of the 32 Nissen patients were considered for MSA before the Nissen but chose not to proceed with MSA, were denied by insurance, or were

excluded due to the need for magnetic resonance imaging. Additional exclusion criteria included allergy to metal, delayed gastric emptying, prior esophageal or gastric operations, and an esophageal stricture.

All patients underwent preoperative evaluation, including video esophagogram, esophagogastroduodenoscopy, pH analysis with a 48-hour wireless probe or a 24-hour impedance-pH catheter, and high-resolution manometry. Patients underwent postoperative clinical follow-up at approximately 2 weeks, 6 weeks, and 6 months. Quality of life and symptom severity were assessed with the Gastroesophageal Reflux Disease Health Related Quality of Life (GERD-HRQL) scale, Quality of Life in Reflux and Dyspepsia (QOLRAD), and a modified Dakkak Dysphagia Severity Score preoperatively and at each clinic follow-up appointment. At 6 months, patients were requested and encouraged to repeat preoperative studies.

At endoscopy, the gastroesophageal junction was evaluated using the Hill Classification, and the presence or absence of esophagitis was graded according to the Los Angeles (LA) Classification system. The presence or absence of a hiatal hernia was noted and the size determined by the distance from the top of the rugal folds and the diaphragmatic impression. The patient’s use of PPIs was stopped 7 days before pH analysis. The highest score during a 48-hour wireless probe evaluation was used for the DeMeester score and the percentage of time the pH was less than 4.

Operative Techniques

LNF was performed using 5 ports. The esophageal hiatus was completely dissected and mediastinal dissection carried to the level of the inferior pulmonary veins. The upper gastric fundus was mobilized by dividing the proximal short gastric vessels and the retrogastric pancreatic attachments. Once 3 cm of intraabdominal esophagus was established, the esophageal hiatus was closed with single “0” polyester sutures (Ethicon, Cincinnati, OH) and a Ti-Knot (LSI Solutions, Victor, NY) suture-securing device.

After hiatal closure, a 2-0 silk marking suture was placed on the posterior fundus 6 cm down the greater

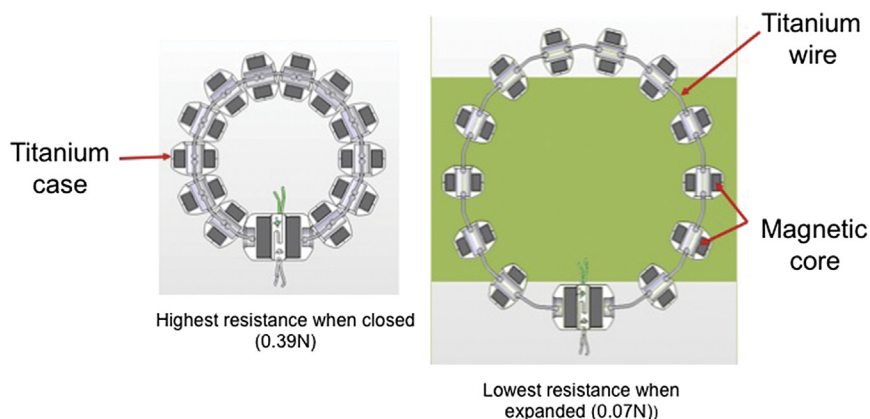


Fig 1. Magnetic sphincter device (left) closed and (right) open. (Images courtesy of Torax Medical Inc, Shoreview, Minnesota.)

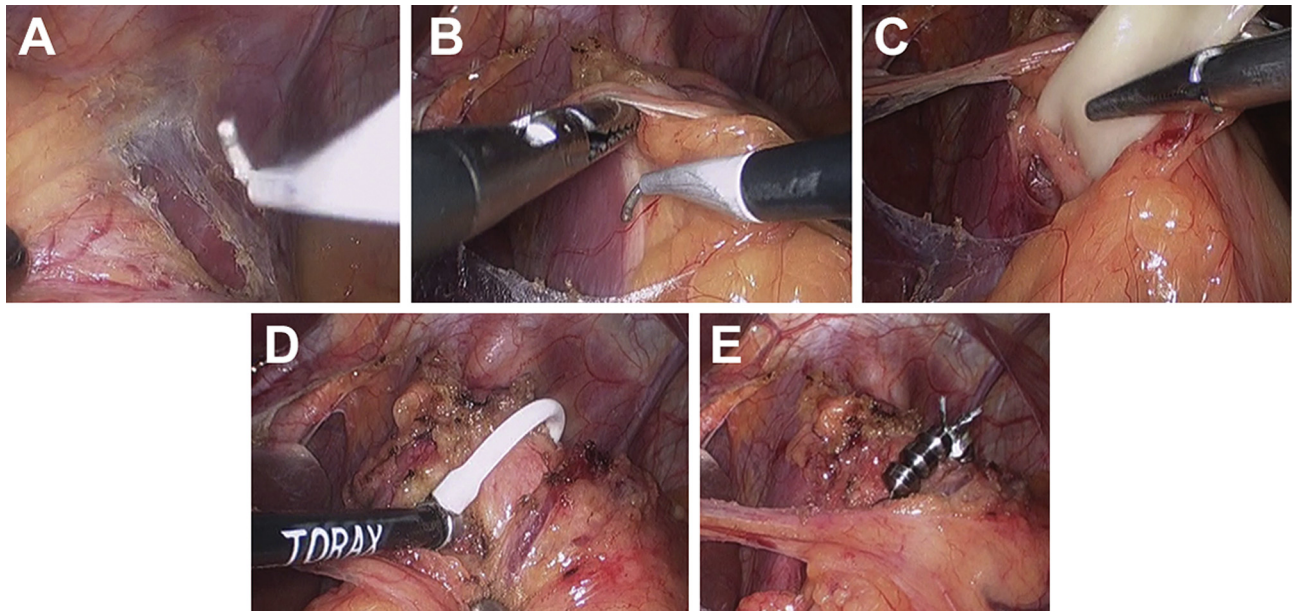


Fig 2. Implantation of magnetic sphincter augmentation (MSA) device: (A) Landing zone on left crus. (B) Site of MSA placement under the hepatic branch of the vagal nerve. (C) Creation of tunnel with isolation of posterior vagal nerve. (D) Determination of MSA size. (E) Implanted MSA device in anterior trench.

curve and one-third of the distance perpendicular to the lesser curve. The posterior fundus was brought through the retroesophageal window, and a “shoe-shine” maneuver was performed to ensure a 1:1 relationship between anterior and posterior fundus. A 58F or 60F bougie was advanced and the shoe-shine maneuver repeated. The fundoplication was created by placing a 2-0 polypropylene suture with a 1-cm pledget in a horizontal mattress formation through the anterior fundus—right lateral wall of esophagus—posterior fundus, followed by the second pledget. The fundoplication was lengthened by placing 2-0 silk sutures from anterior to posterior fundus above and below the pledget to create a 2-cm to 2.5-cm length wrap.

The magnetic sphincter was implanted laparoscopically using 5 ports in a similar configuration to fundoplication (Fig 2). The location of device placement was identified on the patient’s right side, opening the gastrohepatic ligament above and below the hepatic branch of the anterior vagus nerve. Directly opposite to this on the left crus, a “landing zone” was created by incising the peritoneum between the lateral left crus and the posterior fundus. A tunnel was created behind the esophagus from the edge of the right crus under the vagal branch and directed toward the landing zone. A 0.25-inch Penrose drain was placed through the tunnel behind the esophagus. The posterior vagal nerve was identified and isolated by placing the Penrose inside of the nerve and around the gastroesophageal junction.

On the anterior surface, the peritoneum and fat were cleared with monopolar cautery, taking care to preserve the anterior vagal nerve and the phrenoesophageal ligament; thus, creating a “trench” for the device to sit in on

the surface of the esophagus. If the posterior esophageal hiatus showed a “gap,” it was closed with 1 or 2 “0” polyester sutures.

To determine the correct size of device, the outer circumference of the esophagus is measured using a sizing device provided by the manufacturer. The device is placed in the tunnel between the posterior vagus and the esophagus and wrapped around the esophagus. The device is tightened till it approximates the circumference of the esophagus without indenting the tissue. At this point, the device identifies the recommended size. The appropriately sized device was situated in the tunnel and around the esophagus, and the sutures were secured using a suture-securing device.

Postoperatively, patients who underwent LNF were kept nothing by mouth with the nasogastric tube to low intermittent suction. After an overnight stay, the nasogastric was removed, and a barium swallow was obtained. If satisfactory, the patient was initiated on clear to full liquids and discharged after tolerating oral intake. Patient-controlled analgesia was used for all LNFs.

Patients who received a magnetic sphincter were initiated on clear liquids immediately after the procedure. A barium swallow was obtained the next day and a regular diet started the morning after the procedure. For most patients, only oral analgesics were used. The most recent 5 patients were discharge home the same day.

The tests used for statistical comparisons were the *t* test for continuous variables and the Pearson χ^2 test for categorical variables. Symptom improvement was assessed using the McNemar paired change test. All *p* values were two-tailed, and no adjustments were made for multiple comparisons.

Table 1. Patient Baseline Demographics and Gastroesophageal Reflux Disease Characteristics

Characteristic	MSA (n = 34)	Nissen (n = 32)	p Value
Age, mean ± SD y	54 ± 11.8	47 ± 12.2	0.007
Gender, No.			0.32
Female	16	19	
Male	18	13	
BMI, kg/m ² ± SD	27 ± 5.1	30 ± 4.4	0.03
GERD duration, y ± SD	12 ± 11.6	12 ± 8.5	0.88
Hernia size, cm ± SD	1.4 ± 1.1	1.5 ± 0.8	0.62
Hill grade, No.			
I	4	2	0.44
II	11	8	
III	14	18	
IV	5	5	
Esophagitis grade, No.			
No esophagitis	14	15	0.78
A	14	10	
B	4	6	
C	1	1	

BMI = body mass index; GERD = gastroesophageal reflux disease.

Results

Of the 34 patients who underwent MSA, 24 completed the 6-month follow-up. For comparison, 32 patients underwent LNF. The baseline demographic and GERD characteristics of both groups were similar, except MSA patients were older, and LNF patients had a higher body mass index (Table 1).

The operative time for MSA was 73 minutes compared with 118 minutes for LNF ($p = 0.001$). There were no operative deaths. In the MSA group, there were no major morbidities. Minor morbidities included symptomatic bradycardia in 1 patient and corneal abrasion in 1 patient. Two major morbidities occurred in the Nissen group. One patient was readmitted 5 days after discharge with dehydration and nausea, and 1 patient had symptoms of esophageal obstruction, which was confirmed on barium swallow. The patient returned to the operating room so a suture could be removed from the hiatal closure and was discharged without further sequelae. Two minor morbidities in the Nissen group included a postoperative seizure and a urinary tract infection.

At a mean follow-up of 6 months for MSA patients and 10 months for LNF patients, the symptoms of heartburn, regurgitation, cough, aspiration, chest pain, and ear, nose, and throat symptoms, such as throat clearing and hoarseness, were significantly improved compared with baseline (Table 2). The quality of life improved in both MSA ($n = 23$) and Nissen ($n = 17$) from baseline to 6 weeks postoperatively and to 6 months, and there was no difference between the groups for the QOLRAD (4.4, 6.0, and 6.6 vs 4.3, 5.8, and 6.6; $p = 0.77, 0.57, \text{ and } 0.91$, respectively) or for the GERD-HRQL (20.6, 8.8, and 5.0 vs 22.8, 10.0, and 5.1; $p = 0.51, 0.43, \text{ and } 0.93$, respectively). Swallowing ability worsened in both groups at 6 weeks (37.7 to 33.2 vs 37.1 to 26.3) but was significantly worse in the Nissen group ($p = 0.023$). Swallowing returned to baseline at 6 months in both groups (40.2 vs 36.9; $p = 0.24$; Fig 3). One component of the GERD-HRQL evaluates bloating and gassy feelings and showed a trend (1.32 vs 2.36; $p = 0.059$) in favor of MSA patients. Similarly, 16 of 24 MSA patients (67%) reported the

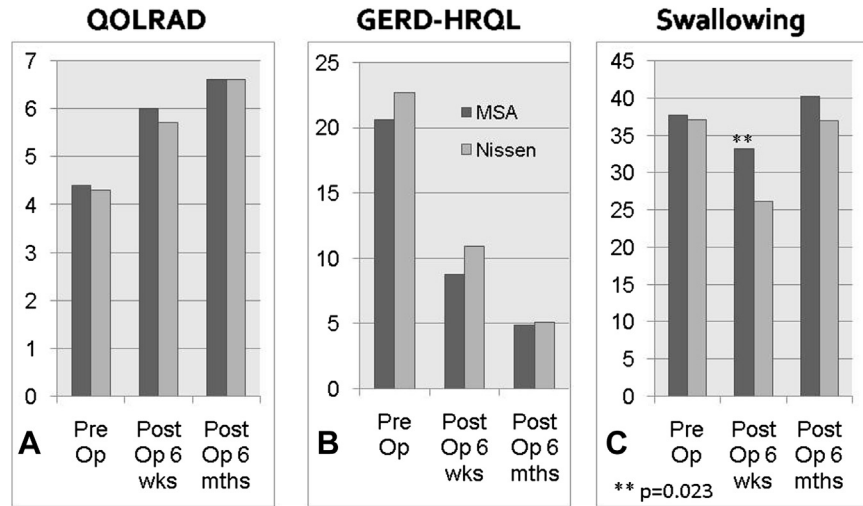
Table 2. Symptom Resolution by Magnetic Sphincter Augmentation and Nissen Procedure

Symptoms	Magnetic Sphincter Augmentation				Laparoscopic Nissen Fundoplication			
	Preoperative	Symptom Present Postoperatively?		p Value ^a	Preoperative	Symptom Present Postoperatively?		p-Value ^a
		No (No.)	Yes (No.)			No (No.)	Yes (No.)	
Heartburn	No	31	3	0.000	No	28	1	0.001
	Yes	31	2		Yes	31	4	
Regurgitation	No	34	6	0.000	No	30	3	0.001
	Yes	28	0		Yes	29	2	
Cough	No	31	27	0.000	No	31	22	0.001
	Yes	7	2		Yes	10	1	
Aspiration	No	32	28	0.000	No	31	29	0.001
	Yes	6	0		Yes	3	1	
Chest pain	No	32	25	0.000	No	32	26	0.001
	Yes	9	2		Yes	6	0	
ENT symptoms	No	32	15	0.000	No	31	24	0.001
	Yes	19	1		Yes	8	1	

^a Related samples McNemar's change test.

ENT = ear, nose, and throat.

Fig 3. Quality of life scores are shown for (A) Quality of Life in Reflux and Dyspepsia questionnaire, (B) Gastroesophageal Reflux Disease-Health Related Quality of Life scale, and (C) swallowing for 23 of 24 MSA patients (mean follow-up, 6 months), and 17 of 32 Nissen patients (mean follow-up, 10 months).



MSA: N=23/34; mean follow up 6 m
Nissen: N=17/32; mean follow up 10 months

ability to belch, whereas none of the Nissen patients could belch ($p = 0.0001$).

Postoperative pH testing was performed in 18 of 34 MSA patients and in 22 of 32 Nissen patients between 6 and 10 months postoperatively. Both procedures normalized the DeMeester score, with the MSA group dropping from 49.5 to 14.2 and the Nissen group from 49.0 to 5.1. Similarly, the percentage of time pH was less than 4 was normalized, with the MSA group going from 14.8% to 4.6% and the Nissen group going from 13.5% to 1.1%. Despite normalization, there was still a significant difference between the postoperative DeMeester scores ($p = 0.0001$) and the percentage of time the pH was less than 4 ($p = 0.0001$) when MSA and LNF were compared.

In absolute terms, 10 of 18 MSA patients (56%) had a DeMeester score below the 14.7 threshold and 10 of 18 had a percentage of time the pH was below the 4.9 threshold. Comparatively, only 1 Nissen patient had a DeMeester score above 14.7. Furthermore, when two components of the DeMeester score were reviewed—total number of refluxes and the number of postprandial refluxes—the total number of refluxes were below the normal of 104, with MSA having 60.1 refluxes compared with 21.5 for Nissen ($p = 0.002$). The number of postprandial refluxes was 35.1 for MSA and 8.4 for Nissen ($p = 0.001$).

At their respective mean follow-up assessments, all of the MSA patients (0 of 24) remained off PPI therapy whereas 1 of the 32 Nissen patients was on a PPI despite having normal postoperative testing. In the MSA group, 1 patient had an episode of a food bolus impaction requiring evaluation, but no invasive treatment, and 1 patient underwent endoscopic balloon dilation for dysphagia early in our experience. Comparatively, gas bloat occurred in 2 Nissen patients, symptomatic esophageal spasms occurred in 2 requiring medical therapy, and 1 patient had new-onset diarrhea related to fundoplication. Endoscopically, esophagitis occurred in 4 MSA patients (LA class A in 3; LA class B in 1), with each having

an elevated DeMeester score. Comparatively, 1 Nissen patient had LA class A esophagitis with a normal DeMeester score. There were no identified erosions, device migrations, or removals in the MSA group. A recurrent hiatal hernia developed in 1 Nissen patient at 1 year, but the Nissen was intact.

Comment

The main finding in this study is that patients with GERD, with or without a hiatal hernia smaller than 3 cm, undergoing MSA with the LINX device (Torax Medical Inc, Shoreview, MN) have equivalent outcomes compared with patients with similar characteristics undergoing LNF. MSA alleviates typical and atypical symptoms of GERD, improves quality of life, and normalizes distal esophageal acid exposure. Our MSA results are similar compared with previous published studies and add to the growing experience with this device [7–9].

Although MSA results in normalization of distal esophageal acid exposure overall, our mean DeMeester approaches the normal of 14.7, and only 56% have a normalized score. These findings are similar to the results of Ganz and colleagues [8], who reported postoperative DeMeester scores at 1 year of 13.5 and an absolute normalization in 58%. Comparatively, Bonavina and colleagues [9] reported a median composite DeMeester score of 11.2 and absolute normalization of 80% but had a longer median follow-up of 4.2 years.

There are several possibilities for these findings. First, it is possible that with time, there is “maturation” of the device with scarring around the gastroesophageal junction leading to continued improvement in GERD control. Second, endoscopy and pH testing are done at a point in time and the test results may reflect only what has occurred during a short period of time before testing. Lastly, and most likely, it may depend on the grade of esophagitis before MSA because the relative

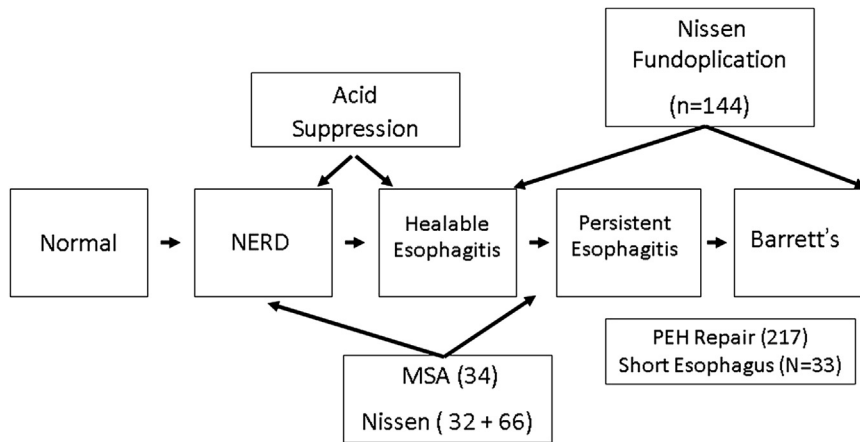


Fig 4. Individualized treatment for the spectrum of gastroesophageal reflux disease. (MSA = magnetic sphincter augmentation; NERD = nonerosive reflux disease; PEH = paraesophageal hernia). (Adapted from Lord and colleagues [1].)

proportions of no esophagitis vs LA class A vs LA class B were 60:20:20 in the Ganz trial, 83:10:6 in the Bonavina trial, and 41:41:12 in our study. This may reflect subtle differences in disease severity and lower esophageal sphincter dysfunction that may not be augmented as well with the device.

The outcome data presented here suggest that there are differences between MSA and Nissen that address patient and referring physician concerns about Nissen fundoplication. First, the ability to belch is substantially improved by MSA. Second, the trend toward less bloating and gassy feelings also favors MSA. Third, the side effects experienced by the Nissen patients, such as gas bloat, spasm, and diarrhea/dumping, which can last more than 5 years, did not occur after MSA [3]. We hypothesize that these differences may be explained by restoration of a more normal sphincter when MSA is used.

Asymptomatic normal patients experienced a mean number of total refluxes of 43.8 in 48 hours [10]. In MSA patients, 60 total refluxes occurred, whereas Nissen patients experienced only 21 refluxes. Furthermore, in the postprandial period, air that has been entrained during ingestion of food is often vented. But because Nissen allows for only 8 reflux episodes compared with 35 with MSA, the ability to vent is less effective with Nissen, giving way to the side effects of bloating and flatulence. Thus, MSA likely results in a more normal sphincter, whereas Nissen may be considered “super-normal” because little to no reflux is not physiologic.

Given the findings in this study, MSA may allow GERD treatment to be further individualized because it offers patients an option if PPIs are not effective (Fig 4). Of the 427 patients we screened, 217 were for type II to IV paraesophageal hernias, including 33 with a short esophagus requiring Collis gastroplasty. Another 144 Nissen procedures were performed for refractory esophagitis and hiatal hernias sized between 3 and 5 cm. In the spectrum of disease where MSA is indicated, there were only 98 patients representing the group of patients who previously would have remained on acid suppression with incomplete control but might never be referred for or considered surgical intervention.

This study has several limitations. First, it represents a small series with very short follow-up. As such, these results may not be indicative of future outcomes, although the longer-term data have shown durability out to 6 years [9].

Second, the short follow-up precludes definitive comments about the issues of erosion, migration, and removal. However, an analysis of the first 1,048 MSA implants showed no migrations and removal of 36 devices [11]. One erosion was reported, but recent reports have identified 4 erosions in nearly 1,600 implants, which is significantly less than Angelchik and lap bands, which were considerably larger and exerted pressure on the esophagus (Torax Medical Data).

Lastly, the study is retrospective and thus subject to biases, but the patients in the comparison group were carefully evaluated and all would have qualified for MSA, thus making the conclusions perhaps more meaningful. A randomized controlled trial would be ideal to compare MSA with existing therapies; however, because there are objective data points, such as pH, to allow comparison, the need for such a trial may not be as great.

In conclusion, MSA in patients with chronic GERD and a hiatal hernia of less than 3 cm in size results in similar objective control of GERD, symptom resolution, and improved quality of life compared with Nissen fundoplication. MSA seems to restore a more physiologic sphincter that allows physiologic reflux in patients with earlier reflux disease that facilitates belching and creates less bloating and flatulence by allowing total reflux events to move toward the mean and maintaining postprandial reflux events. This device has the potential to allow individualized treatment of patients with GERD and increase the surgical treatment of GERD.

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DISCUSSION

DR STEVEN DeMEESTER (Los Angeles, CA): Great presentation. A lot of times with these new devices and so forth, we learn more from the failures than we do from the successes. We expect it to work, but the failures are where we can really dig in and understand the mechanism and where it might be best applicable. So in light of that, I didn't really see that you presented the absolute percent of normalization. You showed us mean or median data for pH.

Can you tell us what percentage of patients that had the LINX (Torax Medical Inc, Shoreview, MN) and the Nissen were normalized, and then tell us about the patients that weren't normalized with the LINX, particularly the 3 with esophagitis. What can we learn from them? Did they have esophagitis before the surgery and it persisted, or is this new esophagitis? Can you also describe how you managed these patients? Thanks.

DR LOUIE: Certainly. So the percent normalization for the Nissen group, 100% of those patients had normalization of their DeMeester scores all the way down below, which I think accounts for such a low number.

There, the percent normalization for the LINX device is about 60%, and those values are just above the 14.7 threshold. So we looked at those patients specifically. And you look at them, they are like 16, 18, 19, and almost all of those are postprandial reflux events. When we looked at the 3 with esophagitis, none of those patients had any symptoms. They had some esophagitis preoperatively. It was better postoperatively, but we never got rid of it. But they were 1-mm breaks, and we were very strict about our assessment of esophagitis. Whether those episodes or those findings endoscopically, at that one point in time, will persist, or they come and go is unclear. None were treated with proton pump inhibitors (PPIs).

You know, one of the anecdotal stories is on one of those patients, right before he was tested, he was an Air Force pilot. He was a very smart guy. He was interested in figuring out what the burst pressure for his LINX device was. So before surgery he could normally eat about 2 bowls of chili, and then he would have horrendous reflux disease. So he figured he would do the same, and he did it, unfortunately, right before we did his reflux testing. And he figured that at 2½ bowls of chili, he was good. When he got to the top of the third bowl, he had horrendous reflux disease. So he's, like: Doc, my burst pressure is this. This is when I get reflux disease. So I think, unfortunately, the

patients select themselves for LINX in this early period, and some of them are very interested in sort of understanding the physiology.

But I think it is correct. I think that you have a little bit of postprandial reflux, and if those patients continue their habits, which are big meals once or twice a day, they might get some reflux or esophagitis because of it.

DR THOMAS WATSON (Rochester, NY): Brian, I really enjoyed your talk. I agree with you; there is a place for LINX in the marketplace. The device seems to be as efficacious as a Nissen in controlling gastroesophageal reflux disease (GERD), and perhaps with a slightly better side effect profile. I think also that the reversibility aspect of it has appeal to patients.

We have been putting in the LINX device in Rochester, and I would say we have had three major barriers to utilizing it more frequently. I would be curious how you have dealt with these issues:

One is insurance reimbursement. We are having a hard time getting our insurers to pay for it. And while I hope that will get easier in time, I am curious if you have any insights into how you have been dealing with that problem in Seattle.

Number two, we still do not have good long-term data about potential complications, such as erosions, from these devices left in for decades or more. What are you telling patients about such possibilities?

And thirdly, the fact that having an internal magnet precludes the patient from ever having an magnetic resonance imaging (MRI) scan is a big turnoff, particularly for younger patients looking at long life ahead of them. Will this problem ever be overcome?

Thanks for your insights and a great presentation.

DR LOUIE: Sure. Let us talk about insurance. Insurance continues to be a battle. I think the company has been very good about that. Since Swedish is self-insured, we went to the medical director, and for our internal employees, we have made a deal with them and we are covered. We have used the two companies that the Torax Medical is engaged in. We have been reasonably successful, but still, we have some insurers that absolutely will not cover it, deeming it experimental. We have gone to peer-to-peer review, we have done the whole scheme of things. I think it is better. The company has statistics showing that they are getting closer to 30%, 40%, 50% approval over

the last year, with the process that they are using, and it still remains early.

The MRI issue, I think, is a concern for younger patients, although they did receive conditional Food and Drug Administration approval about 3 weeks ago for a 0.7 Tesla MRI, so the LINX can go in a 0.7 Tesla MRI. I have an e-mail out to the radiologist to find out what a 0.7 Tesla MRI can do, but as I understand it, that includes most bone and joint procedures, but will not allow you to have a sort of a spinal MRI.

And then the third point, long-term foreign body, I think that remains a concern when you implant anything around the esophagus. We have seen that with permanent mesh around the hiatus. We do know, and it is available online, that there have been 4 erosions in the world. These erosions, in total, about 1,550 cases worldwide, 3 of them in Europe, 1 of them in the United States. And I think, at least my interpretation of that data is that if you are having difficulty getting in that tunnel underneath, that is where most of the erosions are occurring. I think probably, at the time of surgery, maybe that is surgeon judgment. We are not putting them in properly or we are having some difficulty back there doing stuff, when you look at that data. So I think that is still a far cry from other implantable devices, but I think it is a concern.

We have told all our patients that, look, this is a concern. We have told them what the data are. We have also engaged them that if you are going to have LINX at this point in time, that your follow-up has to be complete, so they are getting an endoscopy once a year, or they are being surveyed so we have an idea what is going on, because it is such a new device. And most patients who are interested in the new device will continue to come back, because they are interested in the device, as well.

DR MARK B. ORRINGER (Ann Arbor, MI): As a "gray beard," I would like to add historical perspective to this discussion. Years ago, a thoracic surgeon named Mr Ronald Belsey developed the Belsey Mark IV hiatal hernia repair. And the Belsey Mark IV operation was named that because there had been a Mark I, a Mark II, and a Mark III before the Mark IV.

It took Dr David Skinner going over to Bristol, England, to convince Mr Belsey to allow him to review and report his series with 10-year follow-up, because Belsey felt that a minimum of 5 to 10 years of follow-up was necessary before one could authoritatively talk about the efficacy of an antireflux operation, which in his case he modified four times in search of better results. This principle of long-term follow-up has guided thoracic surgeons whose practice focuses upon esophageal disease.

So I would submit that a new antireflux operation with only a few months of follow-up has little proven efficacy justifying clinical adoption. This paper is more or less a "proof of concept"

discussion. The LINX device appears to control reflux in the short-term, but how it will fare in long-term follow-up is unknown.

Historically, we can draw upon the experience with the Angelchik prosthesis of a few decades ago. With this device, there was not any question of "tunneling too deep" or "getting too near" the esophageal wall as discussed with the LINX device today. One took this beautiful, soft, spongy silicone ring and secured it around the esophagus. What could be safer than that? Except that in long-term follow-up, the eventual migrations up through the hiatus and down onto the stomach, and erosions through the esophageal wall, at times with passage of the ring per rectum, were disasters which led to the ring being pulled off the market. So the concept of placing a semirigid ring around a part of the body that is constantly exposed to the motion of the diaphragm and moving up and down is not exactly new. The face may be a little different, but it remains to be seen if history will be repeated.

I would like to ask one question: We always show on the slides of the indications for antireflux surgery "failure of PPI therapy." The decision to undertake an antireflux operation should not be made lightly, as multiple failed repairs may ultimately result in an esophagectomy and the physical adjustments that have to be made to it afterward.

And the quality of life after an esophagectomy is generally not as good as it is with putting up with a little reflux and modifying lifestyle: getting on a weight reduction program, limiting carbohydrate intake, and walking 3 miles every day.

So I would submit that "failure of PPI therapy" alone is not a sufficient indication for antireflux surgery as it does not constitute "failure of medical management," but rather just one aspect of medical management. I would like to know how you counsel patients being considered for a LINX procedure. Do you make them lose weight? Or, do they come to you saying, "I heard about this new antireflux device you are putting in, and I would like one of these?"

DR LOUIE: Well, you can see that we chose people who were under a body mass index of 35, and the LINX people were actually much lower statistically. But yes, these patients, we counseled them. I counseled them extensively about diet, exercise, and weight, because we know that that is the biggest significant contributor to recurrent GERD after repair.

And I think your comments about the longer-term effects are key. I think this is clearly early, which we have labeled it early because we do not have those long-term data. And I think it is important that we are going to follow these people out so that we avoid potentially the issues with Angelchik.

DR BLACKMON: Thank you.