Background: Collis gastroplasty is indicated when tension-free fundoplication is not possible. Few studies have described the physiological results of this procedure, and no studies have evaluated outcomes of the endoscopic approach.

Objective: To assess the long-term outcomes of patients treated with laparoscopic Collis gastroplasty and fundoplication.

Design: Case series.

Setting: Tertiary care teaching hospital and esophageal physiology laboratory.

Patients: Fifteen consecutive patients with refractory esophageal shortening diagnosed at operation. Complicated gastroesophageal reflux disease or type III paraesophageal hernia (or both) was preoperatively diagnosed with esophagogastroduodenoscopy, 24-hour pH monitoring, esophageal motility, and barium esophagram. Fourteen (93%) of the 15 patients were available for long-term objective follow-up.

Interventions: Laparoscopic Collis gastroplasty with fundoplication and esophageal physiological testing.

Outcome Measures: Preoperative and postoperative symptoms, operative times, and complications were prospectively recorded on standardized data forms. Late follow-up at 14 months included manometry, 24-hour pH monitoring, and esophagogastroduodenoscopy with endoscopic Congo red testing and biopsy.

Results: Presenting symptoms included heartburn (13 patients [87%]), dysphagia (11 patients [73%]), regurgitation (7 patients [47%]), and chest pain (7 patients). An endoscopic Collis gastroplasty was performed, followed by fundoplication (12 Nissen and 3 Toupet). There were no conversions to celiotomy and no deaths. Long-term follow-up occurred at 14 months. Esophagogastroduodenoscopy revealed that all wraps were intact with no mediastinal herniations. Manometry demonstrated an intact distal high-pressure zone with a 93% increase in resting pressure over the preoperative values. Two (14%) of these patients reported heartburn, and 7 (50%) patients had abnormal results on postoperative 24-hour pH studies (mean DeMeester score, 100). Biopsy of the neoesophagus revealed gastric oxyntic mucosa in all patients. Endoscopic Congo red testing showed acid secretion in only those patients with abnormal DeMeester scores. Of these 7 patients, 5 (36%) had persistent esophagitis and 6 (43%) had manometric evidence of distal esophageal body aperistalsis that was not present preoperatively.

Conclusions: Collis gastroplasty allows a tension-free fundoplication to be performed to correct a shortened esophagus. It results in an effective antireflux mechanism but can be complicated by the presence of acid-secreting gastric mucosa proximal to the intact fundoplication and a loss of distal esophageal motility. These patients require close objective follow-up and maintenance acid-suppression therapy.

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A complicating factor in antireflux surgery is a shortened esophagus, which is thought to be the result of a vicious cycle of repetitive esophagitis, submucosal fibrosis, and subsequent axial shortening. Although the true incidence of this problem is controversial, most esophageal surgeons have little doubt that this clinical disorder exists. The decreased morbidity of the laparoscopic approach has increased the overall number of antireflux procedures performed and has thereby made the identification and treatment of this condition more relevant.

The success of an antireflux surgical procedure depends on the creation of a tension-free fundoplication. A fundoplication done around an intrinsically shortened esophagus will have a high failure rate due to mediastinal wrap herniation, disruption, or misplacement (“slipped Nissen”). An esophageal lengthening procedure is, therefore, indicated when, after extensive mediastinal esophageal mobilization, it is not possible to deliver the gastroesophageal junction 2.5 cm below the hiatus. The combination of a Collis gastroplasty and fundoplication has long been considered the treatment of choice for esophageal shortening due to complicated gastroesophageal reflux (GER), paraesophageal hernia, or both. Several studies have established the short- and
PATIENTS AND METHODS

STUDY DESIGN

A retrospective analysis of a prospectively recorded database of patients undergoing antireflux surgical treatment was performed between October 18, 1991, and June 1, 1997.

PATIENTS AND PREOPERATIVE EVALUATION

Fifteen consecutive patients with refractory esophageal shortening diagnosed at the time of the surgical procedure were identified. This represents a 2.6% (15/580) incidence for the period stated. The average age was 62 years (range, 42-69 years), and 7 (47%) patients were female. All patients had medically refractory GER disease, type III paraesophageal hernia, or both, and were preoperatively evaluated with esophagogastroduodenoscopy (15 of 15), esophageal manometry (15 of 15), barium esophagram (15 of 15), and 24-hour pH monitoring (12 of 15). Radionuclide esophageal and gastric emptying studies were performed when indicated. All patients completed a standardized preoperative symptom assessment form, and information was stored in a computerized database.

A 360° fundoplication was used unless the patient had significant esophageal dysmotility as defined by low-amplitude contractions (average, <35 mm Hg) or greater than 35% interrupted peristaltes.9 These patients underwent a 270° posterior fundoplication.

SURGICAL PROCEDURE

Patients are placed in a supine, split-leg position with a roll beneath the right scapula. The right side of the chest and abdomen is cleansed with an iodine solution and draped. Trocar placement is illustrated in Figure 1. Exposure of the esophageal hiatus includes a complete dissection of the right and left crura. All hernia contents are reduced by gentle traction, and the mediastinal sac is excised in patients with paraesophageal hernias. The gastric fundus is mobilized by dividing the short gastric vessels and posterior fundal attachments. If the location of the gastroesophageal junction cannot be determined, intraoperative upper endoscopy is performed to identify it. Once it is determined that, despite extensive transthoracic mediastinal dissection (up to the carina), the gastroesophageal junction is unable to be delivered at least 2.5 cm below the hiatus without undue tension, an esophageal Collis gastroplasty is performed.

A 12-mm sealed thoracic port is placed into the right side of the chest at the fourth intercostal space in the anterior axillary line, and a pneumothorax using 10 mm Hg side of the chest at the fourth intercostal space in the anterior axillary line, and a pneumothorax using 10 mm Hg.

The fundoplication is then created around the new angle of His (Figure 2). The stapler is fired, creating a 3-cm neoesophagus with a 56F lumen and a new angle of His. Additional firings can be done if required. A chest tube is not routinely placed at the completion of the operation; the thoracic port is vented as the patient is given positive-pressure ventilation. Any residual thoracic carbon dioxide is rapidly reabsorbed.

The fundoplication is then created around the neoesophagus. The fundus is passed through the posterior window and used to retract the esophagus to the left. This provides exposure for a loose hiatal closure with nonabsorbable sutures (Figure 3).

Once the appropriate placement of the wrap is determined, a 2- to 2.5-cm Nissen fundoplication is created around a 56F bougie. The standard 2 to 3 wrap sutures are augmented by additional sutures to the posterior hiatus and anterior diaphragm and 2 sutures from the lateral neo-esophagus to the insides of the fundoplication. All sutures are tied intracorporeally to minimize tissue trauma. The Toupet fundoplication is performed similarly except for the final sutures. These are placed from the fundus to the esophagus at the 10- and 2-o’clock positions.

FOLLOW-UP

The intraoperative and perioperative data collected included operative time, amount of blood loss, complications, and length of hospital stay. A diatrizoate meglumine (gastrografin) swallow study was done and an amylase level of the drain specimen was measured on postoperative day 1.

Early follow-up was at an average of 1 month and included the completion of a symptom assessment form (15 of 15 patients). Late follow-up was at 14 months and consisted of the completion of a symptom assessment form (14 of 15 patients), esophagogastroduodenoscopy with endoscopic Congo red testing and distal esophageal biopsies (14 of 15 patients), esophageal manometry (14 of 15 patients), and 24-hour pH testing (14 of 15 patients). Esophagogastroduodenoscopy included retroflexion views to assess the integrity and position of the fundoplication. In addition, the flap valve was objectively graded using the algorithm described by Hill et al.16 Four quadrant distal esophageal biopsy specimens were obtained and examined for the presence of oxyntic cells (parietal and chief cells) or intestinal metaplasia.

Endoscopic Congo red testing was modified to assess for neoesophageal acid production.11 The patient was given subcutaneous pentagastrin (6 µg/kg of body weight) and the distal esophageal mucosa was irrigated with sodium bicarbonate solution. The fluid was then aspirated and a bicarbonate—Congo red solution was applied. Acid secretion was indicated by the presence of blackened neoesophageal mucosa. A color change in the stomach was used as a control.

Stationary esophageal manometry was performed using a water-perfused capillary system along with a 4-port, radial manometry catheter (Arm drfer Medical, Greendale, Wis.). The data were interpreted using a computerized software package (Medtronic-Synetics Corp, Irving, Tex). All motility and endoscopy studies were performed by 1 investigator (B.A.J.).

Twenty-four-hour esophageal pH measurements were obtained by placing a single probe 5 cm proximal to the upper border of the distal high-pressure zone (DHPZ). Patients kept a detailed diary of events and symptoms during the testing period.
long-term clinical success of this procedure using an open surgical technique.

Although these studies have established excellent clinical results using the Collis gastroplasty, few investigations have included all aspects of postoperative objective testing. This study evaluates the clinical outcomes of an endoscopic approach to Collis gastroplasty. We present the results of comprehensive physiological testing of a prospective cohort of patients undergoing a lengthening procedure.

RESULTS

Presenting symptoms included heartburn (13 patients [87%]), dysphagia (11 patients [73%]), regurgitation (7 patients [47%]), reflux sensation (7 patients), and chest pain (7 patients). For 5 (33%) of the 15 patients, a previous antireflux operation had failed (2 misplaced fundoplications, 3 wrap herniations), probably because of a short esophagus. The average duration of symptoms before Collis gastroplasty was 15 years (range, 5-40 years). The average presurgical lower esophageal sphincter (LES) pressure and total length were 7.7 mm Hg (range, 0-30 mm Hg) and 1.1 cm (range, 0-1.8 cm), respectively. No patients had an intrabdominal LES length. Two (15%) of 13 patients had a nonspecific motility disorder, as evidenced by low-amplitude contractions. The results of ambulatory 24-hour pH monitoring were abnormal in 100% of patients, and the average DeMeester score was 100 (range, 19-197). The median DeMeester score was 126. Seven (47%) and 9 (60%) of 15 patients had a symptomatic preoperative stricture and stage III or IV esophagitis, respectively. Seven patients (47%) had Barrett esophagus. On review of the initial barium esophagram reports, the size of the hiatal hernia was measured in only 3 of the 15 patients, and there was no mention of hiatal hernia reducibility.

The operative time averaged 4.2 hours (range, 3.0-6.5 hours), with a mean blood loss of 198 mL (range, 20-1000 mL). There were no intraoperative complications or deaths. Twelve Nissen and 3 Toupet fundoplications were performed. Seven (47%) of the 15 patients required repair of a type III paraesophageal hernia. No procedures were
converted to a celiotomy. The results of all postoperative diatrizoate meglumine (gastrografin) swallow studies were normal, as were the drain amylase levels. There were 3 early postoperative complications. In 1 patient, a postoperative ileus developed that resolved spontaneously. Two patients returned to the emergency department soon after discharge with pleuritic chest pain. These episodes resolved with the use of nonsteroidal anti-inflammatory agents and rest. The length of hospital stay averaged 2 days (range, 1-3 days), and patients returned to work within 8 days (range, 4-10 days) of the operation. The average short- and long-term follow-up was at 1 and 14 months, respectively. One patient was unavailable for late follow-up because of death unrelated to the surgical procedure or esophageal disease. One (7%) of 15 patients and 2 (14%) of 14 patients had heartburn at early and late follow-up, respectively. Dysphagia, which was defined as any difficulty swallowing liquids or solid foods, was found in 2 (13%) of 15 patients at 1 month and 2 (14%) of 14 patients at 14 months (the same 2 patients). One of these patients had dysphagia due to recurrent stricture formation that responded to dilatation. Five (36%) patients had hyperflatulence and early satiety at late follow-up, but 14 (100%) patients surveyed considered their operation a success. Table 1 lists the preoperative and postoperative symptoms.

Table 1. Symptoms Before and After Laparoscopic Collis Gastroplasty*

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Preoperative (n = 15)</th>
<th>Early Postoperative (n = 14)</th>
<th>Late Postoperative (n = 14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heartburn</td>
<td>13</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>7</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Water brash</td>
<td>7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>11</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Chest pain</td>
<td>7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary artifact</td>
<td>7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Gas bloat</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Early satiety</td>
<td>0</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Hyperflatulence</td>
<td>0</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>3</td>
<td>1</td>
<td>5</td>
</tr>
</tbody>
</table>

*There were no interventions for fundoplication-related adverse effects.

Of the 15 patients, 14 (93%) were available for late postoperative objective testing, as described above. Endoscopy confirmed an intact and properly positioned fundoplication in all 14 patients. In addition, all patients were noted to have either a grade 1 or grade 2 flap valve appearance. Five patients (36%) had esophagitis, however, with 1 patient requiring dilatation for recurrent peptic stricture above the wrap. Biopsy of the esophagus directly proximal to the fundoplication revealed oxyntic mucosa (parietal and chief cells) in 11 (100%) of 11 patients. Differentiating grossly between the neoesophagus and Barrett esophageal changes was difficult and often depended on biopsy results. There was no dysplastic progression of Barrett esophagus. A positive result on endoscopic Congo red testing, as evidenced by black staining proximal to the DHPZ, was present in 7 patients.

Manometry revealed an intact DHPZ reconstruction for all patients. There was an average LES resting pressure of 16 mm Hg (range, 10-20 mm Hg), a total LES length of 3 cm (range, 2.0-4.5 cm), an intra-abdominal length of 1.3 cm (range, 0-2.5 cm), and 71% LES receptive relaxation to 0 mm Hg. This conferred a 93% increase in the resting pressure and a 196% increase in the total LES length after Collis gastroplasty and fundoplication. Distal esophageal body function (levels IV and V) was absent in 6 patients (43%) (all 6 patients had normal motility preoperatively); 1 patient had low postoperative contraction amplitudes. Seven patients had abnormal results on a postoperative 24-hour pH study as defined by an elevated DeMeester score (average, 100). The average and median postoperative DeMeester scores were 52 (range, 0.3-232) and 8.4, respectively. Two of these patients were symptomatic (DeMeester scores, 44.3 and 75). All patients with abnormal 24-hr pH studies are currently being treated with antacid therapy. There was a 48% overall decrease in the DeMeester score (100-52), but patients with abnormal results on 24-hour pH testing exhibited a 20% increase in the average single longest reflux episode (43 minutes) (Table 2).

Table 2. Postoperative Characteristics of Patients With Abnormal Results on Ambulatory 24-Hour pH Testing*

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Prior Antireflux Surgery</th>
<th>Antireflux Procedure Performed</th>
<th>Postop Heartburn</th>
<th>Postop Esophagitis</th>
<th>ECRT</th>
<th>Flap Valve Grade</th>
<th>Postop Resting LES Pressure, mm Hg</th>
<th>Longest Reflux Episode, min/24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Yes</td>
<td>Nissen</td>
<td>No</td>
<td>No</td>
<td>+</td>
<td>1</td>
<td>Normal/amotile</td>
<td>20</td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>Nissen</td>
<td>No</td>
<td>No</td>
<td>+</td>
<td>1</td>
<td>Normal/amotile</td>
<td>18</td>
</tr>
<tr>
<td>3</td>
<td>No</td>
<td>Toupet</td>
<td>Yes</td>
<td>Yes</td>
<td>+</td>
<td>1</td>
<td>Low amp/amotile</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>No</td>
<td>Nissen</td>
<td>Yes</td>
<td>Yes</td>
<td>+</td>
<td>1</td>
<td>Normal/amotile</td>
<td>15</td>
</tr>
<tr>
<td>5</td>
<td>No</td>
<td>Nissen</td>
<td>No</td>
<td>Yes</td>
<td>+</td>
<td>1</td>
<td>Normal/amotile</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>No</td>
<td>Nissen</td>
<td>No</td>
<td>Yes</td>
<td>+</td>
<td>1</td>
<td>Normal/low amp</td>
<td>12</td>
</tr>
<tr>
<td>7</td>
<td>No</td>
<td>Nissen</td>
<td>No</td>
<td>Yes</td>
<td>+</td>
<td>1</td>
<td>Normal/amotile</td>
<td>15</td>
</tr>
</tbody>
</table>

*Postop indicates postoperative; ECRT, endoscopic Congo red test; Preop, preoperative; LES, lower esophageal sphincter; and plus sign, positive result (acid secreting).

†Normal indicates normal contraction amplitude and peristalsis; amotile, amotile esophageal body at segments IV and V; and low amp, low-amplitude contractions.

Bremner et al. have shown that as the complications related to GER disease become more severe, the esophageal length progressively decreases. For patients with true
esophageal shortening who undergo Nissen fundoplication without a lengthening procedure, the procedure is doomed to fail because of excessive tension. The clinical outcomes described in the literature support the use of Collis gastroplasty as an effective treatment of intrinsic esophageal shortening without the need for an esophagectomy. This series describes the physiological outcomes of the Collis gastroplasty performed laparoscopically.

Our results indicate that the laparoscopic Collis gastroplasty with fundoplication provides excellent symptomatic relief from GER disease with minimal operative morbidity and a rapid return to the presurgical standard of living. This repair provides long-term protection from GER by establishing a tension-free, intra-abdominal DHPZ reconstruction. This is supported by the fact that all patients in our series were free of symptoms of reflux and had an intact fundoplication at upper endoscopy. In addition, the LES resting pressure and length were within normal ranges for all patients at late follow-up. Several series have documented similar excellent long-term outcomes with respect to symptom relief and competency of an intra-abdominal DHPZ.

Despite these favorable symptomatic results, many patients having Collis gastroplasty continue to have abnormal distal esophageal pH measurements. At late follow-up, Martin et al documented abnormal results on 24-hour pH studies in 30% of patients, with poor symptom correlation. Anselmino et al described a 54% continued dependence on medical therapy after Collis gastroplasty and fundoplication despite objective documentation of a physiologic, normal-pressure DHPZ. The present series demonstrates that at late follow-up, 7 patients (50%) had abnormal results on 24-hour pH studies and 5 (36%) had evidence of esophagitis. Although there was a substantial overall improvement in the DeMeester score and a dramatic resolution of symptoms, these results were obviously cause for concern.

We think that neoesophageal acid production proximal to an intact fundoplication, coupled with poor distal esophageal clearance, is the cause of these abnormal postoperative findings. This is supported by the fact that all distal esophageal biopsy specimens revealed oxyntic mucosa proximal to the DHPZ, and all 7 patients with abnormal results on 24-hour pH studies exhibited positive Congo red staining, indicating the presence of actively secreting gastric mucosa. Acid damage to esophageal squamous mucosa is compounded by a 20% increase in the average longest reflux episode in this group. This delayed acid clearance is explained by the finding that there was an amotile or poorly functioning distal esophagus in all 7 patients who had abnormal results on a 24-hour pH study. Only 1 patient with a normal 24-hour pH had a positive result on the Congo red test, and that patient had normal esophageal motility. Regarding the cause of neoesophageal acid production, several possible explanations exist: First, all patients had oxyntic mucosa proximal to their fundoplications and irrespective of the results of postoperative testing. This indicates that in the patients with normal results on 24-hour pH studies and negative Congo red tests, the parietal cells no longer functioned. Whether this is the result of a regional vagotomy during esophageal mobilization or due to the placement of a staple line across the fundus at the angle of His is unknown. Second, the amount of functional gastric mucosa above the midpoint of the DHPZ logically relates to the amount of distal esophageal acid exposure. Several patients with normal results on 24-hour pH monitoring studies had black staining only inside the wrap. We would, therefore, recommend that the esophagus be mobilized as much as possible before Collis gastroplasty in an attempt to make the squamocolumnar junction as low as possible. Finally, all patients with abnormal results on 24-hour pH studies had an amotile or poorly functioning distal esophagus with a resultant delay in esophageal clearance. The cause of this is not entirely clear, but we think that there is a gradual dilatation of the neoesophagus above the DHPZ, which leads to poor contractility of the gastric tube. Two patients in this series were noted to have enlargement of the distal esophagus on late follow-up upper gastrointestinal series. There was poor correlation between the manometrically determined length of an amotile esophagus and the neoesophageal length. This indicates that other factors, such as distal esophageal dysmotility, may result from the creation of a neoesophagus.

Although potential complications from neoesophageal acid production are cause for concern, the laparoscopic Collis gastroplasty with fundoplication provides excellent long-term protection from duodenogastroesophageal reflux, a condition that is thought to play a primary role in the progression of Barrett esophagus to esophageal adenocarcinoma. We have shown that the Collis gastroplasty with fundoplication abolishes GER. We think this is the reason that most patients are asymptomatic postoperatively. Only 2 (29%) of 7 patients with abnormal results on 24-hour pH studies had heartburn at 14 months. Because of this, we recommend rigorous objective follow-up that includes esophageal manometry, upper endoscopy, and 24-hour pH monitoring. Medical treatment of patients with abnormal results on a 24-hour pH study should be initiated regardless of symptoms.

The preoperative identification of patients who will require an esophageal lengthening procedure is difficult. Patients with complicated GER disease for a long
duration and a nonreducing hiatal hernia should raise concerns of intrinsic esophageal shortening. Consent should be obtained, and these patients should be prepared for an esophageal lengthening procedure. Swanson et al\(^2\) showed that 14% of patients presenting for antireflux surgical treatment will have some degree of esophageal shortening, and 20% to 70% of this group will require an esophageal lengthening procedure to minimize the chance of wrap failure. In the present series, previous laparoscopic antireflux surgery had failed in a third of patients, and all had intact wraps at the time of reoperation (2 misplaced or “slipped” and 3 intrathoracic herniated fundoplications). This implies a failure to recognize and manage esophageal shortening at the time of the initial operation. It has been shown\(^2\) that patients for whom an initial antireflux operation failed before Collis gastroplasty have worse outcomes than patients who have not had a failure.

**CONCLUSIONS**

Patients treated with endoscopic Collis gastroplasty and fundoplication for esophageal shortening have excellent long-term outcomes with minimal morbidity. The neo-esophagus may contain functional gastric mucosa 50% of the time, resulting in esophagitis but with poor symptom correlation. This mandates rigorous postoperative objective testing and the use of medical acid suppression when necessary. Endoscopic testing and the use of medical acid suppression when indicated. This mandates rigorous postoperative objective testing and the use of medical acid suppression when necessary.

Presented at the 69th Annual Session of the Pacific Coast Surgical Association, Maui, Hawaii, February 18, 1998.

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**REFERENCES**


**DISCUSSION**

David W. McFadden, MD, Los Angeles, Calif: Dr Horvath has described a group of 15 patients subjected to a laparoscopic Collis-Nissen gastroplasty that was performed safely. This group of patients represents only 3% of the total of 380 patients approached laparoscopically by Dr Swanstrom’s group. They all underwent extensive esophageal localization to the level of the carina before being subjected to this rare procedure.

The operation was performed with minimal morbidity, no mortality, and with an 86% reflux-symptom-free result. In fact, all patients postoperatively viewed their operations as successful and would choose to do it again. This is an incredible result in a selected group of subjects with end-stage reflux. Somehow, Dr Swanstrom’s group persuaded all of these patients to undergo extensive postoperative testing that included endoscopy, manometry, and 24-hour pH testing. These results showed that 30% of patients had abnormal results on pH testing postoperatively, 36% had esophagitis, and nearly half had distal esophageal aperistalsis or hyperperistalsis that was not previously present.

Now, we all know that subjective impressions of an operation’s success often poorly correlate with physiological indexes. But the results Dr Horvath has revealed here are so incongruous that they lead us to ask several questions.

First, thanks to your group, we now know that the laparoscopic Collis-Nissen gastroplasty can be performed safely. But when should it be performed? Does your group now perform it in the presence of stricture, Barrett esophagus, or in patients with previously documented peristaltic disorders? When should a pure endoscopic approach make a pure thoracic approach or an esophageal resection be entertaining?

Second, you have described active partial cell mucosa in the neo-esophagus. Have you considered the addition of a selective vagotomy to your operation to decrease the risk of this described sequela?

Third, how did you get such thorough follow-up testing? Where did you get such compliant patients, and who paid for the testing?

Fourth, because of the difficulty in differentiating normal mucosa in your neo-esophagus vs Barrett esophagus, are you not subjecting many of your patients to serial surveillance that would otherwise be unnecessary?

Ralph W. Aye, MD, Seattle, Wash: Dr Swanstrom has been a pioneer in minimally invasive surgery, and I think this is a great
testimony to his outstanding technical and innovative skills. I would first question the validity of the concept of the short esophagus. It is certainly a long-accepted concept in some circles, but perhaps it is less relevant today with the availability of proton pump inhibitors. For example, it is rare today to need to perform esophageal resection for peptic stricture. Most strictures can resolve preoperatively with aggressive medical therapy.

I have had the privilege to work with Luke Hill during the past 10 years, and during that time we have a combined experience of about 800 antireflux procedures. In no case have we had difficulty reducing the gastroesophageal junction below the diaphragm and doing a standard repair. In fact, Luke asked me to repeat his standing offer that if you have a short esophagus and cannot reduce the gastroesophageal junction below the diaphragm, send it to Seattle, and we will do the case free of charge.

I have 2 questions. The first has to do with whether there is an adequate comparison with a similar group of patients with perceived esophageal shortening who have had a standard repair, without gastroplasty. In other words, is this procedure really necessary? Second, what options do we have, having done a gastroplasty, if the operation fails? Can it be redone, or is resection required?

Jeffrey E. Doty, MD, San Jose, Calif: I think it is important again to note that this is a rarely required operation, and we can frequently get the gastroesophageal junction well below the hiatus. I am wondering if Dr Swanstrom ever resorts to transecting the anterior vagus nerve, which will frequently tether the gastroesophageal junction if it is extremely scarred, or if he thinks a Collis should be performed in that situation.

Also, I have questions regarding the changes in motility. Dr Swanstrom has previously reported a high incidence of failure with the Toupet fundoplication; yet, it seems that this procedure creates decreased peristalsis obviously in the neoesophagus, which is constructed of amotile stomach. I believe 3 of these patients had a Collis-Toupet procedure, and I was surprised with the low incidence of postoperative dysphagia. I wonder if we are not creating a situation where we will see more frequent dysphagia in the future.

Finally, how can we predict which patients will need this Collis fundoplication? The indications for this operation seem to most commonly fall into 2 groups of patients, those who respond dramatically to proton pump inhibitors—and we are operating on them primarily to get them off lifelong medications—and patients who have chronic heartburn that is resolved with proton pump inhibitors but who also have intractable mechanical regurgitation. Those patients do well and I think would merit a Collis fundoplication. But to do a Collis fundoplication on a patient who has solely heartburn controlled with proton pump inhibitors and not to tell them that they have a 50% chance of still needing to be on maintenance proton pump inhibitors is worrisome. So we would like to know how to predict which patients may require the Collis fundoplication so we can appropriately inform them preoperatively.

Mark A. Vierra, MD, Stanford, Calif: The physiological follow-up of these patients is exactly what we need. I commend the authors for doing that. I also want to know how you have been able to get all of these patients to come back to you for this amount of testing.

I have a concern that although this operation has been done with no morbidity and what looked like good symptomatic outcomes, it does not seem to me that it would stand up to the same standards we use for most of our other patients with fundoplications. It is particularly concerning that a third of the patients are on acid suppression therapy. In talking with Dr Patti, in our combined experience with the University of California, San Francisco, we would have probably done 11 or 12 Collis gastroplasties. In fact, between the 2 of us, we have done none. So I wonder how often this is truly necessary. This is a common preoperative discussion with patients that we may have to lengthen their esophagus. In fact, in the operating room, it usually turns out not to be necessary.

Marco G. Patti, MD, San Francisco, Calif: This presentation highlights a difficult, although rare, problem encountered by surgeons who treat GER disease. I would like to make 2 comments and ask 2 questions.

A lot of stress has been placed on the physiological aspect of this operation, and I think there are some misunderstandings. Postoperative manometry showed an LES pressure of 16 mm Hg. I think these readings overestimated the real LES pressure because about 30% to 40% of the esophageal wall was less pliable and probably rigid because of the staple line. Therefore, the real pressure is probably much lower than your manometric findings, thus explaining the presence of residual abnormal reflux in 30% of your patients. The second point regards the importance of the aperistalsis of the lower esophagus as a cause of delayed clearance. It was surprising to learn that there was a 70% incidence of dysphagia preoperatively, but only 7% postoperatively. It is surprising because the distal esophagus was aperistaltic and because 12 of your patients had a 360° fundoplication, which creates a higher outflow resistance.

My first question relates to the indications for the procedure. How can we recommend an operation that has a 30% failure rate? In particular, you showed that some of your patients had a postoperative DeMeester score of 52 (normal, <15), and the youngest was 42 years old. I think that the residual reflux may create later problems, such as Barrett esophagus or strictures.

My second question relates to the technique of laparoscopic Collis-Nissen fundoplication, as proposed by Dr John Hunter, vs the combined thoracoscopy-laparoscopic approach you use. What are the advantages and disadvantages of each technique?

Bruce M. Wolfe, MD, Sacramento, Calif: Our application of the Collis esophageal lengthening procedure has been limited to patients undergoing reoperation following an initial failed operation for GER. Common among these patients having reoperation is a finding of the fundoplication at least in part having been done around the proximal stomach. In the third of your patients who underwent reoperations, did this situation arise as the result of a shortened esophagus at the outset or of inadequate mobilization of the stomach and esophagus at the time of the first operation, thereby failing to get the esophageal wrap around the esophagus rather than the proximal stomach? As with other discussants, we have been almost uniformly able to get these gastroesophageal junctions below the diaphragm at the initial operation. Perhaps we are missing some patients with a short esophagus and should be doing Collis gastropexies as an initial procedure, but if so, we are not recognizing it at operation.

Blayne A. Standage, MD, Portland, Ore: My question is similar. I was interested in the patients who had a reoperation. Were these 5 patients referrals to you, representing a diverse mix of patients, or were these your patients who were early failures of the laparoscopic Nissen due to the shortened esophagus? If that is the case, in retrospect, is there anything you could have recognized at the time of the first operation to allow you to do the Collis gastropasty at that time?

John R. Benfield, MD, Sacramento: The reoperations that we have encountered after unsuccessful antireflux procedures have been among our more challenging thoracic operations. One lesson that I have learned is that if the thoracic dissection becomes tedious, it is usually good to open the diaphragm and to mobilize the stomach and esophagus with this adjunct to move the operation ahead. Those have been the circumstances under which we have most often employed the Collis gastroplasty to lengthen the esophagus. You said that 33% of the patients you operated on by endoscopic means had reoperations. Surely there were additional patients whose second
operations were not done endoscopically. How many patients did you encounter during this period who had reoperations by the open method?

Dr Swanstrom: The intent of our presenting these data was not so much to preach to people about what they should be doing but to raise some questions about what is currently being done. Many of us, at least at larger centers, are doing a lot of laparoscopic antireflux surgery, and I think we need to be asking some hard questions about that. Who are we doing these operations on? How are we doing them? Do we really understand what we are doing? And are we really following up these patients adequately to know that we are doing the best job?

Many of the questions that were asked deal with this, and I think I will take these and try to clump them together because there were so many. We have a policy, to answer Dr Benfield's question, of at least trying to treat all patients, even patients being reoperated on, endoscopically. We have done about 55 repeated fundoplications and, for the most part, have been successful. Only a small percentage of these patients needs a Collis gastroplasty.

There are some patients whose surgical procedure we would do open. These would include patients with strangulated peri-esophageal hernias, multiple repeated fundoplications, and perhaps patients with an extremely short esophagus. To answer Dr Wolfe's question, we have found that a large number of these were not properly performed. They were fundoplications done around the upper stomach, so-called slipped wraps, or they were done under tension and subsequently had herniation up into the chest. To answer Dr Standage's question, some of these cases were done originally by me, and some were done by others. I think that many surgeons have pushed the limits of adequate mobilization of the esophagus in these repairs. Some of our early repairs were under tension, and now, 6 or 7 years later, we are seeing some failures with those that were done under a bit of tension.

When to do a Collis gastroplasty? Or, as Ralph Aye has questioned, does this disease even exist? Several large series have been published of laparoscopic fundoplications that don't report any Collis gastroplasties. To some extent, I think proton pump inhibitors have minimized the need for lengthening procedures. We see fewer patients with end-stage strictures and fewer with chronic, severe esophagitis. I also think that patients are being referred earlier and that we will continue to see a drop-off in the number of these really complex patients—those with huge type III peri-esophageal hernias, high-grade strictures, or the columnar Barrett esophagus up to the carina. We need to educate our medical colleagues to refer these patients earlier.

How do you recognize these problems preoperatively? I don't think there is a single good test. It is also not a good thing in a Meckel diverticulum or wherever gastric rests occur. But how do you fix it? Well, fortunately, we have acid-suppressing medication while it's going to have a significant rate of needing long-term acid suppression. But it is better than an esophagectomy. Dr Patti, you had asked a technical question about how we do the acid suppression. But it is better than an esophagectomy. Dr Patti, you had asked a technical question about how we do the motility testing. I think your question about the compliance of the distal esophagus is a good one, but it is not a problem. Our motility tracings have a characteristic look. This is possibly because of the way the Collis wrap is formed, which results in a bit of asymmetry.

Another factor may be that the gastric wall that is used doesn't have the layers of muscle and thickness that may result in progressive dilatation, which may be disruptive to postoperative function.

Finally, the lesson to learn with this study is not so much whether it was a failure or a success as a procedure. After all, it has been a well-established procedure for 40 years. But we need to raise the question of how can we make it better? Perhaps there are ways we should be looking at in the laboratory to make this a better procedure. The problem we have identified is that the ectopic gastric mucosa of the Collis gastroplasty is not a good thing. It is also not a good thing in a Meckel diverticulum or where else gastric rests occur. But how do you fix it? Well, fortunately, we have acid-suppressing medication while we are looking for this answer. It was mentioned that perhaps we could do a highly selective vagotomy in these patients. In fact, that is what we are currently looking at in the laboratory.