Failed Antireflux Surgery

What Have We Learned From Reoperations?

Santiago Horgan, MD; Dieter Pohl, MD; Diego Bogetti, MD; Thomas Eubanks, DO; Carlos Pellegrini, MD

Hypothesis: Factors that lead to failures in antireflux procedures can be identified, and dealing with them at the initial operation may decrease the number of such failures.

Design: Analysis of symptoms, 24-hour esophageal pH monitoring, manometry, upper gastrointestinal tract radiographs, and correlation with operative anatomic findings.

Setting: University referral center.

Patients: Forty-eight patients who previously underwent antireflux surgery (Nissen fundoplication, 29; Hill fundoplication, 7; Angelchik prosthesis, 1; multiple, 5; unknown, 6) and had symptoms of foregut disease.

Main Outcome Measures: Determination of the cause of failure of previous operations and identification of factors that may prevent recurrence.

Results: Fourteen patients (29%) presented with symptoms of an incompetent cardia (heartburn and regurgitation), 15 patients (31%) presented with symptoms of defective esophageal emptying (dysphagia), 13 (27%) had symptoms of both, and 6 (13%) had other symptoms. All patients were initially treated medically and/or with dilation. A reoperation was performed in 31 patients (65%) whose symptoms persisted. Reoperation was completed laparoscopically in 28 patients (90%). At reoperation we identified 3 main types of failure: type I, patients in whom the gastroesophageal junction was herniated through the hiatus, either with the wrap (IA) or without it (IB). There were 13 patients (43%) classified as having type IA, and 5 patients (16%) classified as having type IB. Type II failure involved a paraesophageal component resulting from a redundant wrap in 5 patients (16%), and type III involved a malformation (defective position or construction) of the wrap in 2 patients (6%). The remainder had a failed Hill fundoplication (3 patients), a herniated Angelchik prosthesis (1 patient), and normal postoperative anatomy (2 patients).

Conclusions: Failure of the crural closure and malformation of the wrap are the main reasons for failure of antireflux procedures. Use of proper surgical techniques including meticulous closure of the crura and appropriate construction and fixation of the wrap at the first operation will help prevent recurrence.

Arch Surg. 1999;134:809-817

Several studies have now shown that antireflux operations done with minimally invasive techniques1 are effective in the treatment of gastroesophageal reflux disease, resulting in a better than 90% rate of patient satisfaction,2 short hospital stays, and rapid return to work or previous activities.3 Thus, the minimally invasive approach to antireflux surgery has emerged as one of the best alternatives for treatment of gastroesophageal reflux disease, especially in those patients who need lifelong therapy.4-6 As with other procedures, the operation has its pitfalls, occasional complications, and failures. Furthermore, even minor variations in surgical technique can alter its clinical outcome7,8 which is complicated by the fact that the procedure has a well-defined learning curve.9

Since 1994 we have seen a steady stream of patients who presented to our swallowing center with foregut symptoms after an antireflux procedure. This provided us with the opportunity to diagnose and manage (medically or surgically) a substantial number of these complex cases. With this experience, we thought it would be useful to analyze the causes that led to failure of the first operation. We postulated that, if properly identified, many of the factors that led to failure might be appropriately addressed initially so as to decrease the number of operative failures. During the study we also developed diagnostic and management strategies that should help

From the Department of Surgery, University of Washington Medical Center, Seattle.
PATIENTS AND METHODS

This study includes 48 patients who had a previous anti-reflux operation and were evaluated between January 1994 and December 1998 for foregut symptoms, comprising 3 patients from our own series of 430 and 43 patients referred to us from other centers. There were 32 men and 16 women ranging in age from 26 to 77 years (mean age, 50 years). All of these patients had symptoms that were recurrent, severe, complex, or unclear, or that persisted in time and required a specific therapy.

PREOPERATIVE EVALUATIONS

All patients underwent esophagogastroduodenoscopy, barium swallow test, manometry, and 24-hour esophageal pH monitoring. Manometry was performed with a water-perfused catheter connected to a computer (Gastrosoft Program; Medtronic-Synectics, Shoreview, Minn.). Lower esophageal sphincter location, pressure, relaxation, and body peristalsis were determined. A pH probe with 2 sensors 10 cm apart was used for esophageal pH monitoring. The lower sensor was positioned 5 cm over the lower esophageal sphincter and measured distal reflux. The proximal sensor, located 15 cm above the gastroesophageal (GE) junction, provided an estimate of proximal acid exposure. Patients recorded occurrences of heartburn, chest pain, cough, and other symptoms during the study. Results were analyzed using the same software and a DeMeester score was calculated.

OPERATIVE TECHNIQUE

A thorough description of the operative technique is impossible because each operation is different depending on the original approach (open vs laparoscopic), the amount and character of adhesions, the technique used in the first operation to reconstruct the cardia, and the magnitude of the anatomic malformation that led to recurrent symptoms. Patients who had undergone an open operation were generally more difficult, as they had more numerous and denser adhesions. Trocar positioning varied according to the site of previous incision and expected area of adhesions. Additional ports were used as needed. As adhesions to the abdominal wall were taken down, additional ports were positioned. The most difficult part of the procedure was the dissection of the cardia and the separation of the diaphragmatic crura from the esophagogastric junction. This part of the operation must be done slowly and meticulously as adhesions, previous sutures, clips, and occasionally the presence of a herniated fundus make it quite difficult. Once the esophagus and the cardia had been dissected from the crura, the previous repair was completely taken down in all patients. This required dividing sutures to the preaortic fascia in cases of Hill fundoplication and undoing the wrap in patients with a previous fundoplication. When the repair had been taken down, we proceeded with a reconstruction of the cardia. The essentials of our technique for laparoscopic reconstruction of the cardia have been previously described. It includes systematic crural closure and the creation of the antireflux barrier that best fits the patient (either a partial or a total fundoplication). Esophageal lengthening was performed using the Collis-Nissen technique if the esophagogastric junction remained above the hiatus after a thorough esophageal mobilization.

CLASSIFICATION OF FAILURES

As suggested in a previous publication by Skinner et al, patients were divided according to the presenting symptoms into those with symptoms related to an incompetent cardia, those with symptoms related to defective esophageal emptying, those with combined symptoms (incompetent cardia and defective emptying), and those with other manifestations. Each symptom was graded using a scale from 0 to 4, whereby 0 meant that the patient did not suffer from that symptom; 1, the symptom was felt once a month; 2, at least once per week; 3, once per day; and 4, the symptom was present all of the time.

In addition, we analyzed the anatomy in each patient based on endoscopic, radiologic, and operative data. We then classified all patients who had an anatomic defect according to the nature of the defect. In these patients we traced, whenever possible, the specific anatomic defect to the step or steps in the initial operation that were likely to have caused the problem. Further, we related these anatomic problems to the presenting symptoms that brought them to our attention.

physicians and surgeons dealing with this difficult subset of patients.

RESULTS

PRESENTATION AND EVALUATION

Twenty-one of the 48 patients had prior laparoscopic antireflux surgery (Nissen fundoplication, 19; Hill fundoplication, 2), and 27 patients had had open surgery (Nissen fundoplication, 10; Hill fundoplication, 5; Angelchik prosthesis, 1; multiple procedures, 5; other procedures, 6). Symptoms had recurred between 1 week and 12 years after surgery (mean recurrence, 24 months). Fourteen patients (29%) had symptoms of an incompetent cardia and presented primarily with heartburn and/or regurgitation, 15 (31%) had symptoms of inadequate clearance of the esophagus and presented primarily with dysphagia, 13 (27%) had symptoms of both incompetent cardia and defective emptying, and 6 (13%) had other foregut symptoms (ie, bloating, diarrhea, and abdominal pain). The mean symptom scores were 3.4 for heartburn, 3.0 for dysphagia, and 2.8 for other foregut symptoms. Patients who presented primarily with heartburn (incompetent cardia) had a mean DeMeester score of 71 (range, 12.8-240). Patients who presented with symptoms of defective emptying had a mean DeMeester score of 19 (range, 0.2-83) (Table 1). Although the means are not statistically significant because of the large variation in scores between patients, all patients in the group with defective cardia had abnormal reflux, whereas acid exposure was abnormal in 8 of 15 patients with dysphagia. The upper gastrointestinal tract study was abnormal in 43 patients (90%) and failed to show any anatomical defects in 5 patients (10%).

©1999 American Medical Association. All rights reserved.
MANAGEMENT

All patients were initially treated conservatively. Treatment was based on proton pump inhibitors for patients with incompetent cardia, on repeated dilatations for patients with defective esophageal emptying, and on both therapies for those with combined manifestations. Patients with other foregut symptoms were treated accordingly to provide symptomatic relief. Eventually, surgical treatment was carried out in 31 of these 48 patients. The remaining 17 patients were successfully treated or are still being treated with medical therapy.

All 31 patients were approached laparoscopically, and the operation was completed this way in 28 (91%) of them. In 1 patient, the operation was converted when we realized he had a very short esophagus, and we opted for an esophagectomy. He had had 2 previous repairs. In 2 patients, conversion was done because we could not adequately identify structures and make progress using the laparoscopic approach. Twenty-three (75%) of these 31 patients had a reconstruction with a total fundoplication, 5 (15%) had a partial fundoplication, and the 3 remaining patients (10%) underwent other procedures (esophagectomy, esophageal lengthening, and others). Mean operative time was 307 minutes (range, 226-477 minutes). Mean length of stay was 4.1 days. Ten patients had postoperative complications that prolonged the hospital stay or required readmission. Eight of these were directly related to the operation (postoperative ileus, a small leak from a suture line, and pneumothorax complicated with pneumonia) and 2 patients had cardiovascular complications (atrial fibrillation in one patient and hypertension in another). The mortality rate was 0. At a mean follow-up of 25 months, all but 4 patients remained asymptomatic.

ANATOMIC CLASSIFICATION OF FAILURES

Based on a detailed analysis of endoscopic, radiologic, and most importantly, operative findings in the 31 patients who underwent surgery, we found that most of the patients could be classified into 3 categories:

Type I: Eighteen (56%) of the 31 patients had the esophagogastric junction above the hiatus. The anatomic problem in these patients would be the equivalent of a “sliding hiatal hernia” if they had not had a previous operative repair. In 13 of the 18 patients, the GE junction along with the fundoplication were herniated above the hiatus (type IA) (Figure 1). In the remaining 5 of the 18 patients with a type I defect, the GE junction had migrated above the hiatus but the fundoplication remained below (type IB), almost as if it had “slipped” or had been performed below the GE junction (Figure 2).

Type II: In 5 (16%) of the 31 patients, only a portion of the stomach, which was recognized as coming in all cases from the wrap itself, had migrated up through the crura. These patients’ upper gastrointestinal tract study showed a defect similar to a typical paraesophageal hernia, with the GE junction in the normal position (Figure 3). This defect was related in all patients to an exuberant wrap as if the fundoplication itself had been constructed to be very floppy.

Type III: Two patients had a normal position of the GE junction and no evident hernia on x-ray film or intraoperatively. In these patients the wrap itself had been constructed using the midbody of the greater curvature, near the antrum (Figure 4). In the remaining 6 patients we found the following: 1 patient had an Angelchik prosthesis that had migrated to the mid portion of the stomach, 3 had a pre-
vious Hill fundoplication, 2 presented with an incompetent cardia, and 1 had severe persistent dysphagia despite what seemed to be normal anatomy for that procedure. The last 2 patients had an incompetent cardia and no obvious anatomic explanation for it.

**Table 2** summarizes the findings in each group of patients according to their presenting symptoms. As can be seen, patients with type IA, in whom both the GE junction and the wrap had herniated through the hiatus, presented with dysphagia, heartburn, or both, and all patients with a type IB failure presented with dysphagia, probably caused by the “slipped” wrap. Four (80%) of the 5 patients with type II failure had a defective emptying of their esophagus secondary to the bulk occupied by the herniated fundus or by its defective emptying. Both patients with a malconstruction of the wrap (type III failures) presented with defective esophageal clearance. Thus, it is not possible from the presenting symptoms to predict the type of associated anatomical defect. On the other hand, certain anatomical defects clearly explained the presenting symptoms. The upper gastrointestinal tract study predicted the intraoperative findings correctly in 19 patients (62%). It seemed particularly insensitive to detect type IB failures (migration of the GE junction with a wrap on the stomach) so that this defect was recognized preoperatively in only 40% of patients who had it.

**COMMENT**

As surgical therapy is used with increasing frequency to treat patients with abnormal GE reflux, gastroenterologists and surgeons will inevitably have to face failures of this form of therapy. Our study provides some clues to the causes of failure and, through their analysis, it provides several suggestions on how to handle, at the initial operation, most of the common factors that lead to postoperative failures. In addition, we thought it might be important to discuss what we learned, as we dealt with these patients, about diagnostic approach and management.

**DIAGNOSTIC CONSIDERATIONS**

Some degree of dysphagia, bloating, occasional nausea, and abdominal discomfort is common after antireflux operations. These manifestations disappear without treatment in approximately 6 to 8 weeks. Thus, so long as the manifestations are tolerable, we simply reassure patients that they will subside. On the other hand, when manifestations are severe, recurrent, or complex, or when they do not seem to get better within a few weeks, we recommend a full diagnostic workup. This should include at least a barium swallow test, manometry, 24-hour esophageal pH monitoring, and esophagogastroduodenoscopy.
We found that the information provided by the barium swallow test was of great value, certainly of much greater value than we usually assign to this study when doing a workup on patients who have not had an operation. The main reason is that the study provides an excellent overview of the anatomy and the relationship of the GE junction to the hiatus. The study allowed us to determine if there was a gross anatomic defect that would explain the symptoms. When this was the case we indicated an operation early and were satisfied with the results.

As usual, functional studies were critical to characterize the degree of competency of the cardia, and in some cases, to eliminate reflux as the underlying cause of symptoms. Endoscopy was particularly useful in patients with dysphagia, not necessarily as a diagnostic tool but as a way to initiate therapy. The importance of a thorough evaluation before undertaking operative therapy cannot be overstated because the chance of success of reoperation is totally dependent on an appropriate characterization of the problem.

**MANAGEMENT CONSIDERATIONS**

We have learned that not all of the patients who complain of symptoms after an antireflux operation are candidates for reoperation. For example, we operated on only 31 (65%) of the 48 patients in this series. We offered some form of medical management to almost every patient initially. Symptoms of GE reflux were treated with proton pump inhibitors and about 10% of the patients who initially presented with symptoms of an incompetent cardia are still being treated with this form of therapy. Dysphagia was treated with dilation in 8 patients and we were successful in 3 patients. However, we soon found out that those patients in whom dysphagia was associated with a clear anatomic defect such as a slipped wrap or a paraesophageal herniation (type IB and type II failures) did not respond well to dilation and eventually required an operation. We are now more liberal in recommending an earlier operation for a patient who has significant dysphagia and a clear abnormality of the type described. On the other hand, patients with other manifestations (not reflux and not dysphagia) are rarely candidates for operation. We speculate that the problems in these patients are usually associated with a temporary or permanent vagal injury or other functional problem and redoing the operation is unlikely to help.

When an operation is indicated, we choose the laparoscopic approach. The details on how to carry out this procedure are beyond the scope of this article; however, certain essential elements should be considered. First, the operation is demanding, whether done by laparoscopy or by open approach, and should be undertaken only by an experienced team. To underscore this point, our average operative time is still about 5 hours and our study did not show a substantial reduction in operative time in the course of our series. Second, the biggest challenges were the detachment of the stomach, the GE junction, and the distal esophagus from the liver and the crura, and the takedown of the wrap. In 2 instances we opened the esophagus. In 1 patient, the opening was closed laparoscopically. The other patient was found to have a short esophagus, he had had 2 previous repairs and we thought the problem would be better addressed by an esophagectomy. In 3 instances, the fundus was lacerated and had to be repaired. As difficult as this is, we believe the laparoscopic approach offers some advantages of its own over the open procedures: for example, the view is better because of magnification and the ability to change the angle of view, and the constant flow of carbon dioxide opens tissue planes and stretches adhesions. Finally, we recommend a takedown of the previous repair in all patients. This is not easy, but the repair is not functioning, regardless of how it looks, and once it has been undone the surgeon can perform a fresh one following the indications provided by the preoperative workup.

**Table 2. Type of Failure in Relation to Patient’s Presenting Symptoms**

<table>
<thead>
<tr>
<th>Type</th>
<th>IA</th>
<th>IB</th>
<th>II</th>
<th>III</th>
<th>Normal</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heartburn</td>
<td>7</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>2</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>Combination</td>
<td>3</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>13</strong></td>
<td><strong>5</strong></td>
<td><strong>5</strong></td>
<td><strong>2</strong></td>
<td><strong>1</strong></td>
<td><strong>5</strong></td>
<td><strong>31</strong></td>
</tr>
</tbody>
</table>
CAUSES OF FAILURE

Skinner et al \textsuperscript{11} provided a physiologic classification that we and others have used extensively. He identified patients in whom treatment failed primarily because of an incompetent cardia, a defective clearance, a combination of these problems, and alkaline reflux. We did not routinely measure alkaline reflux, but found that our patients were almost equally divided among the first 3 categories. We also found that functional characterizations through a detailed analysis of 24-hour esophageal pH monitoring was essential, as symptoms were not always reliable. For instance, 6% of patients whose main manifestation was reflux did not have an abnormal 24-hour esophageal pH study.

Our classification of recurrences complements and expands on those of Skinner et al and it not only helped us better understand the cause of the problem and plan an appropriate repair, but also has important implications for preventive measures at the initial operation. For example, type I recurrences involved a herniation of the GE junction through the hiatus. This defect can be caused by 1 or more of the following: an inadequate closure of the hiatus, an inadequate fixation of the repair, or a short esophagus that has pulled the GE junction with or without its repair up into the mediastinum. Although a short esophagus has been invoked by many as a common cause of this type of failure, we found it to be the cause in only the minority of the patients. Indeed, we were able to dissect out all of the structures, undo the previous repair, and bring the GE junction into the abdomen in most of the patients. Furthermore, once the initial dissection and detachment from the hiatus had been done, we found that in most patients the distal esophagus was surrounded by normal tissue that had not been dissected in the previous operation and that was easily dealt with, allowing the esophagus to come into the abdomen. If our assessment of this situation were wrong, we would have expected to see frequent repeated failures of our operation, something we have not observed. Thus, the findings of our study suggest that either inadequate closure of the hiatus or inadequate fixation of the GE junction and the wrap in the abdomen, or both, are the main causes of this problem. Thus, to prevent this type of failure, we recommend closing the crura in all patients posterior to the GE junction rather tightly with nonabsorbable strong sutures 0.5 cm apart, leaving enough space only to accommodate the esophagus with a 60 F bougie. In addition, we recommend fixing the wrap to the esophagus and to the undersurface of the crura by “coronal” sutures to maximize anchorage of the repair in the infradiaphragmatic position. Using this technique, we have had only 5 of our 430 patients present with a type IA postoperative hernia. Of course, if at the time of reoperation the GE junction cannot reach the abdomen, then a lengthening procedure such as that described by Collis,\textsuperscript{12} with an added fundoplication, should be performed. In patients with multiple previous operations and a short esophagus, an esophagectomy may also be an appropriate way to deal with this problem.

Type II failure, the paraesophageal herniation of the top of the wrap, was a much more common finding than we expected. This defect, we believe, occurs as a result of at least 2 factors acting concomitantly: a redundant fundoplication and a defective diaphragmatic closure. We have only seen this defect in patients whose first operation was done laparoscopically. It is, indeed, possible that gauging the appropriate circumference of the fundoplication, in other words, the amount of gastric fundus needed to perform it, may be more difficult laparoscopically because of the magnification and because of the bi-dimensional representation of the image (loss of depth of field perception). It may also represent a purposeful effort on the part of the surgeon to create a very floppy wrap to decrease the incidence of postoperative dysphagia. To minimize the occurrence of this problem, we now carefully measure the amount of tissue involved before bringing the fundus behind the stomach. Then, once we have selected what we believe is the appropriate amount, and are ready to place the stitch from one side of the wrap to the other, we bring the wrap back behind the esophagus (undoing it) while still holding both sides (the right side is being held by a grasper passing behind the esophagus). This allows the surgeon to examine and measure the length of the wrap on the left side of the GE junction without interposition of any other structures. This “shoe shine” maneuver described by Peters et al\textsuperscript{13} is also very useful to place the wrap appropriately and to eliminate any excess fundus. If this is not done correctly, an excess of stomach will remain behind the GE junction and will eventually herniate back into the mediastinum. In both type I and type II failures, crural closure may be a crucial component. In several of our patients, the operative report from the initial operation related that the crura had not been closed. Furthermore, we did not have a single patient in whom treatment failed because of a closure that was too tight. That suggests to us that in most instances the defective closure of the crura may have been deliberate in an effort to prevent postoperative dysphagia. Our study suggests that a tighter closure may be preferable to a looser one.

Type III failure simply represents a malconstruction of the wrap. Ultimately, it is an exaggeration of a type II failure in the sense that the wrap is very redundant, but it differs from a type II failure in that there is no hernia through the hiatus. In these patients, a point in the greater curvature near the antrum has been brought up to become the left and anterior side of the fundoplication. The fact that we tend to do a greater mobilization of the fundus when using laparoscopic techniques may predispose to this type of error. Patti et al\textsuperscript{14} have previously recognized this potential pitfall of the operation and have given excellent suggestions on how to determine the 2 critical points in the construction of the fundoplication.

Lastly, we reoperated on 3 patients with a previous Hill fundoplication and with what seemed to be normal anatomic findings for this type of procedure, as well as 2 patients with a previous Nissen fundoplication who also had no specific defects but had documented reflux and bothersome regurgitation that was not responding to medical therapy. Presumably these repairs were too loose and failed to create an appropriate barrier to reflux.

Several important conclusions may be drawn from our study. First, an appropriate amount of distal esophagus needs to be dissected at the initial operation to bring...
the GE junction well into the abdomen without tension. Second, construction of the fundoplication is a critical part of the operation and appropriate steps need to be taken to determine the right amount of fundus to be used. The tendency is to use too much; magnification and lack of depth of field perception may be misleading. Third, the crura must be closed appropriately in all patients. The tendency is to leave it too loose. Fourth, the repair needs to be anchored well to the esophagus and to the infra-diaphragmatic tissues to prevent its migration. Finally, if all fails and the patient returns with recurrent severe or complex symptoms, a thorough characterization of the problem is crucial. Medical therapy is effective in many patients, particularly those with normal postoperative anatomy. For those with defective anatomy, an operation, performed by an experienced and dedicated team, has a very good chance of success.

Presented at the 70th Annual Session of the Pacific Coast Surgical Association, San Jose del Cabo, Baja California Sur, February 16, 1999.

Corresponding author: Carlos A Pellegrini, MD, Department of Surgery, University of Washington, 1959 NE Pacific St, Box 356410, Seattle, WA 98191

REFERENCES


DISCUSSION

Jeffrey H. Peters, MD, Los Angeles, Calif: The authors have reminded us that many of the lessons of surgery come not from prospective randomized trials but from a careful analysis of our failures. This is particularly true in antireflux surgery where prospective randomized trials are relatively uncommon. In this respect, I think this excellent study presents us with several important lessons. Reoperative antireflux surgery is a challenge. The more I do, the more I recognize this simple fact. It’s a challenge to identify the reasons for failure in the first place. It’s a technical challenge at the time of remedial surgery. It’s also a challenge to obtain successful results in this population of patients who have already selected themselves out as being difficult.

The authors have shown us the reasons for failure in 48 patients, roughly half of which had their first procedure done laparoscopically and the other half open. Symptoms returned in an average of 2 years postoperatively, which I think is an important finding. One third of the patients had recurrent heartburn, one third had difficulty swallowing, and roughly one third had both, although the procedure failed in 10% of the patients for nonreflux symptomatic reasons. Three types of failures are recognized, 2 of which were recurrent herniation. The first and the most common was recurrent herniation of an intact fundoplication through the hiatus, the second being the so-called slipped Nissen, which perhaps is a misnomer and should be more accurately termed a misplaced Nissen. The third and final reason for repair was inadequate construction of the geometry of the fundoplication. Despite the fact that nonsurgical therapy was initiated in all patients, 31 of the 48 eventually chose remedial surgery. Remember that number 31 because it becomes important in a minute. All were approached laparoscopically. I remain ambivalent about the wisdom of laparoscopic reoperative antireflux surgery. It’s certainly technically possible as has been shown by this and other studies. Key judgments depend upon the reason for failure in the first place. If that reason had anything to do with the laparoscope, then it doesn’t make sense to do it with a laparoscope the second time around, whether one believes that there are technical compromises that creep into the laparoscopic approach (a conclusion that I am increasingly coming to), such as our ability to close the crura as well as we can with our hands. Furthermore, given the presence of one failure, one would think that it is important to maximize the chances of success the second time. We know full well that the success of a third time antireflux procedure approaches 50-50 and raises the significant possibility of an esophagectomy.

My only criticism of this study is that it includes no outcome data. It does, however, point us to several very important lessons of the technique of primary antireflux surgery. I have a few questions.

The first is that the technical challenge as I alluded to is exemplified by the intraoperative difficulties encountered by the authors. This data is in the article but is not presented here. Of the 31 patients who were operated on, 2 of them suffered esophageal perforations, 1 required an esophagectomy, and 3 had fundic lacerations. That certainly would make me sleep poorly at night. In retrospect, do the authors continue to advocate the laparoscopic approach in this difficult population of patients?

Second, the authors have perhaps consciously minimized the importance of the short esophagus around which considerable controversy continues to ensue. Do you believe that esophageal shortening contributes to reherniation, or perhaps even more importantly, a slipped fundoplication? Shouldn’t we perhaps define the presence of a short esophagus as a failure in the first place and not necessarily by what we perceive to be true at the time of surgery? If so, what can be done to identify and prevent it?

Finally, do you think that the mechanisms of failure have changed in the era of laparoscopic fundoplication? I believe they have. In our experience, recurrent herniation is the most common reason for failure. If this is true, then what steps can we take to minimize its occurrence?

Eric Fonkalsrud, MD, Los Angeles: I would like to congratulate Drs Horgan and Pellegrini for this excellent review of an important subject. Because so many patients are under-
going fundoplications today, it is important to look at some of the complications that occur. Two years ago we had an opportunity to review our experience at UCLA with more than 900 children combined with the experiences from 6 other children’s hospitals, for a total of approximately 7,000 children to review long-term. There was a 4% recurrence of children who were neurologically normal, whereas there was more than a 12% recurrence rate in those who were neurologically impaired. How many adult patients in your series were neurologically impaired? In our combined children’s hospital series, neurologically impaired children tended to have retching episodes and were more likely to have chronic lung disease, both of which increased strain on the fundoplication. In addition, these children tend to have gastric parietal and nondestructive stasis, which lead to gas bloat. In that setting, if the wrap is too tight or too long so that the patient can’t burp or bring up air, there is a much greater likelihood of developing breakdown of the fundoplication. Could you tell us if you see these problems in the adult patients who you deal with and whether you feel that the fundoplication should be made very loose and short, as we have tried to do with the children?

Another problem we have seen in the recurrent fundoplications is that the more dissecting that we do around the hiatus, the greater the likelihood of injuring the vagus nerves, which may cause more stasis; therefore, we routinely do a gastric emptying study on these patients to determine whether there is excessive stasis and whether some type of emptying procedure should be performed.

Lawrence Way, MD, San Francisco, Calif: This report contributes substantially to an understanding of the causes, prevention, and treatment of failed antireflux operations. The findings parallel those of Stein et al and Dallemagne et al (Am J Surg, 1996;171:36) (Surg Endosc, 1996;10:305), who in previous papers identified structural problems of the reconstruction to be the principle causes of failure. The good news here is that the causes are understandable in simple mechanical or architectural terms, and the standard methods of performing antireflux operations can and should be modified to include deliberate steps to prevent them. Namely, reducing the size of the hiatus, securing the wrap to the esophagus, fixing the wrap within the abdomen, carefully gauging the tightness of the wrap, and so forth. For the literature to make sense, articles on the results of antireflux operations must include technical details such as these (Arch Surg, 1998;133:600).

Short esophagus was rare in this series as has also been true in our experience. Furthermore, we have found no correlation between herniation of the wrap and the preoperative size of the hernia, and none of our patients with this complication had a large sliding hernia preoperatively (Arch Surg, 1998;133:600).

We endorse the authors recommendation to attempt the second operation laparoscopically because the evidence suggests that if the dissection proceeds smoothly, the cause of the failure can be detected and corrected. The cause of failure does not seem to be an inscrutable adverse effect of the laparoscopic approach.

While the authors noted that most of the abnormalities causing failure can be demonstrated with barium x-rays, it should be added that when the findings seem normal and reflux persists, the problem is usually due to the wrap being too loose.

The question of hiatus closure deserves the emphasis given by the authors. There are several reports in the literature which claim that this step is unimportant and optional. Evidence from many sources, however, including this study, demonstrates persuasively that downsizing an enlarged hiatus is essential to minimize the frequency of herniation of the wrap.

Edward Phillips, MD, Los Angeles: I have 2 questions for the authors. One is in part what Dr Fonkalsrud brought up, whether patients who recur should all have gastric motility studi...ies or whether it should be restricted for patients with certain symptoms that possibly are related to gastric motility. Second is whether any “successful outcome patient or asymptomatic patients postoperatively have had these anatomic studies” because we have found that a certain number of “normals or successful patients” have these same anatomic abnormalities that we are now indicting as the etiology of their recurrent symptoms.

Thomas Demeester, MD, Los Angeles: I would like to make a couple of comments about Dr Pellegrini’s paper, and thank him for giving me the opportunity to read it prior to its presentation. I am motivated to do so because of what I heard Larry Way just say. Perhaps my comments will bring the discussion regarding the short esophagus back to being reconsidered as a serious complication of gastroesophageal reflux disease that affects surgical outcome. Dr Pellegrini, as I see your data there are 2 conditions that contribute to recurrences: a short esophagus or a large crural separation commonly associated with a large hiatal hernia. I would emphasize that just because the esophagus doesn’t seem short, it still can be short. It only has to be short by about 2 cm to cause too much tension on the repair because the diaphragm goes up and down 30,000 times a day with breathing and the esophagus goes up and down 1000 times a day with swallowing. Just like the ocean meets the shore, in time, there is erosion. For a repair to last a lifetime it must be free of tension or breakdown is the rule. Surgeons, when they first start doing antireflux surgery by the laparoscopic approach, deny that there is a short esophagus, but it is there and if they do careful follow-up, their recurrences will convince them of its existence. It’s a good example of the statement that adults never learn anything until it hurts not to know. I would say, Dr Pellegrini, most of your patients with a type-I recurrence had a short esophagus. Patients with a type-I recurrent had a short esophagus, and as a consequence the repair retracted up into the chest. Patients with type-IB recurrence had a short esophagus and as a consequence the surgeon made the fundoplication too low; ie, around the pulled up stomach.

Now the second problem is the crural opening. Sometimes the crura fail to develop properly and the space between them is just too wide to close effectively. It is a congenital defect, I think. Usually the space between the crura is ovoid, but in this condition it is more circular. Normally the distance between the crura is less than 4 cm, but in these patients it is usually greater than 4 cm. In this situation you will often find that the right crura is atretic and does not hold sutures. They tear through resulting in crural separation and reherniation. So it is basically a crural problem or a short esophagus problem that results in recurrence. You have to recognize these abnormalities to avoid the problem of recurrence.

One last comment, knowing all of this, requires that in some patients you must lengthen the esophagus and in others you must expose the crura in a way that will allow for an effective closure. So we perform a thoracic approach on these patients to really maximize esophageal mobilization and crura closure. Five to 7 days in the hospital really doesn’t make that much difference in a lifetime. Just because you have a laparoscope does not mean that every hernia needs to be treated laparoscopically.

I do think there is a role for the laparoscope in patients with a recurrence that on barium swallow or endoscopy doesn’t seem to have a short esophagus and the crura look fairly close together. To those patients you might want to say, “you know, I think we should take a look with the laparoscope and see if we can determine what went wrong.” Occasionally you will find something that you can attend to laparoscopically. But those who have a short esophagus or wide crural opening, I think you have to approach them by open surgery through the chest. If you attempt to approach these patients laparoscopically, you will experience the problems of correcting a second time recurrence.
Dr Pellegrini: I will answer the questions in the order they were posed. First, Dr Peters asked about the wisdom of laparoscopy as a technique to be used in these patients. I think that laparoscopy in these patients offers a great advantage. (1) The view is substantially better. (2) Adhesiolysis during laparoscopy is facilitated by the increased pressure in the peritoneal cavity and by the flow of carbon dioxide. A little bit of power irrigation once in a while allows adhesions to be identified and dealt with a lot easier than with open technique. I think the angles that one can look at the problem with laparoscopy also helps. Having said that, we have opened 3 of these patients and completed the operation open because we thought that that was safer in those patients. We have not opened any patient in the last 250 patients done primarily, so there is a sharp difference in between the 2 groups, and I think that one should open liberally when the circumstances call for it.

Dr Peters also asked us about outcomes. While this is important, because of relatively short follow-up (our experience was all acquired in a 4-year period), we decided to concentrate in trying to identify the causes that led to failure. This would alert surgeons of certain key elements that should be addressed at the primary operation and prevent the development of a recurrence. That was the purpose of our paper, not a report on outcomes, but I promise you that in the next two years we will bring our long-term outcomes to bear here. So far we know of one patient who has had a recurrence of her hernia, one patient that is symptomatic without any other evidence of an anatomic malformation, and the rest are doing fine.

Dr Peters asked if the pattern of failure has changed with laparoscopy. Is it different than it was with the open operation? I agree, Dr Peters, that it is clearly different. Most of the patients who have had the laparoscopic operation tended to be clustered in the type II failures. I think that the failure is the result of an almost deliberate effort on the part of the surgeon to construct the wrap too loose. Having been taught that we have to make it loose, the surgeon tries to put the wrap behind the esophagus and we have no view of that part of the stomach that is behind the esophagus and the esophagus can be mobilized very nicely laparoscopically, not routinely, but commonly, laparoscopically some and we did an esophagectomy in this group for precisely that reason, but I don't think that the fact that you are going to be confronted with a short esophagus necessarily tells you that you cannot start laparoscopically. Today we do esophagectomies, not routinely, but commonly, laparoscopically, and the esophagus can be mobilized very nicely laparoscopically in ways in which you cannot from the open abdominal approach unless you open the hiatus widely and put your hand all the way up, and that I think was the basis for the thoracic approach in the past. I feel very comfortable dissecting the esophagus to any length that is needed via a laparoscopic approach. Mobilization of the esophagus, which I think is the key of this operation, can be achieved that way.

One of the concerns that I have is, have we missed some patients' short esophagus? If that is the case, we are going to have a high recurrence rate in our patients, and that is why we wanted to wait about 3 more years or so to report our final outcomes.