Outcomes of Laparoscopic Nissen Fundoplication in Patients With the “Hypercontractile Esophagus”

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Hypothesis: To determine if a hypercontractile esophagus, manifested by high-amplitude peristaltic contractions (HAPCs) or hypertensive lower esophageal sphincter (HLES), affects the outcome of antireflux surgery.

Design: Case series. Prospectively maintained database. Direct contact with patients. Mean follow-up 28.7 months.

Setting: University hospital.

Patients: Of 643 patients who had antireflux surgery for uncomplicated gastroesophageal reflux disease (GERD), 15 had HAPCs (≥150 mm Hg) and 4 HLES (≥45 mm Hg).

Intervention: Laparoscopic Nissen fundoplication in all patients.

Main Outcome Measures: (1) Frequency of hypercontractile esophagus in patients considered for antireflux procedure. (2) Effect of fundoplication on esophageal acid exposure and symptoms. (3) Establish whether dysphagia or chest pain develop after fundoplication.

Results: The typical GERD symptoms of heartburn and/or regurgitation occurred in 15 (79%) and 13 (69%) of 19 patients. Dysphagia was present in 5 of 15 patients with HAPCs and in 0 of 4 with HLES; chest pain was found in 5 of 15 patients with HAPCs. After fundoplication acid exposure was improved in all (92%, 16/17) but 1 and was totally normal in 10 patients (83%). Heartburn improved in 11 (78%) of 14 and resolved in 8 patients (57%) of the 14. Chest pain improved in 4 (80%) of the 5 patients who had it, and developed in 3 (23%) who did not have it preoperatively. In patients with HAPCs, dysphagia improved in 4 (80%) of 5 patients with complete resolution in 3 (60%). New dysphagia developed in 2 (11%) of the 19 patients, 1 in each group. No patient with HLES developed chest pain.

Conclusions: High-amplitude peristaltic contractions or HLES may be associated with GERD in a subset of patients with dysphagia or chest pain. In such patients, a Nissen fundoplication, by effectively controlling GERD, relieves these symptoms in most patients. A hypercontractile esophagus in patients with GERD should not be considered a contraindication to a total fundoplication. The surgeon and the patient should be aware of the risk of developing chest pain after the operation.

Arch Surg. 2002;137:724-729

A HYPOCONTRACTILE esophagus, manifested by ineffective esophageal motility and/or a hypotensive lower esophageal sphincter (LES) is commonly seen in patients with severe abnormal gastroesophageal reflux.1,2 A hypotensive LES has traditionally been associated with loss of gastroesophageal competence,3 and ineffective peristalsis has been thought to either result from or contribute to severe gastroesophageal reflux disease (GERD).4-6 Because ineffective esophageal motility has the potential to affect the result of treatment, it has been extensively investigated.7,8

However, a hypercontractile esophagus manifested by high-amplitude peristaltic contractions (HAPCs)9 or a hypertensive LES (HLES)10 is encountered frequently as primary motility disorders in patients evaluated for chest pain or dysphagia.11,12 In the absence of GERD, these primary motility disorders are treated with smooth muscle relaxants,1,3,14 balloon dilation,15 or esophageal myotomy.16-18 In patients with associated GERD these remedies are ineffective and often detrimental19,20 because of their effect on the LES. Medical therapy, in the form of acid suppression, has proven effective in patients with GERD and a hypercontractile esophagus,19,22 but surgery may be needed for those who do not respond to medical therapy or for those who wish to explore it as an alternative modality. Yet, the frequency with which a hypercontractile esophagus is diagnosed in patients with GERD and the potential effects of a hypercontractile esophagus on the results of operative treatment for
PATIENTS AND METHODS

From November 1, 1993, through November 30, 2000, 728 patients underwent laparoscopic Nissen fundoplication at the University of Washington Medical Center, Seattle, to treat GERD or as part of a repair of a paraesophageal hernia. Eighty-five patients with a paraesophageal hernia were excluded from the present study, both to standardize the population and because these hernias often cause dysphagia or chest pain. Thus, there were a total of 643 patients with GERD treated during the study period. We analyzed preoperative and postoperative clinical finding from all patients in whom stationary esophageal manometry revealed a mean distal esophageal amplitude of peristaltic contractions of 150 mm Hg or more (HAPCs group) or a mean LES pressure of 45 mm Hg or higher (HLES group). These values represent the upper limit of the 95% confidence interval for our control population and constitute a distinct deviation from normal in our laboratory. Patients with peptic strictures or abnormal LES relaxation were also excluded from the study. Normal relaxation of the LES was defined as a decrease in the sphincter pressure to below 8 mm Hg in response to a wet swallow. Nineteen (2.9%) of the 643 patients, 15 with HAPCs and 4 with HLES, met the inclusion criteria and are the subject of the present study.

SYMPTOMS

All patients were asked about the frequency of symptoms during the initial visit and after the operation. A physician administered a standardized questionnaire containing 2 groups of 11 questions to each patient on the day of the first visit in our esophageal function laboratory. This questionnaire has been used in our center to quantify the frequency of symptoms for the last 10 years and has proved to be reliable and reproducible. The first set of questions concerned esophageal and other gastrointestinal tract symptoms. Patients were asked about frequency of symptoms and, in particular, about heartburn, regurgitation, dysphagia for both solids or liquids, abdominal or chest pain, belching, bloating, nausea, odynophagia, and globus sensation. The second group of questions related to frequency of extraesophageal symptoms such as hoarseness, aspiration, wheezing, choking, coughing, dyspnea, sore throat, asthma attacks, bronchitis, and pneumonia. Symptoms were scored 0 to 4 according to frequency (0, never; 1, once a month; 2, once a week; 3, once a day; and 4, several times daily).

STATIONARY ESOPHAGEAL MANOMETRY

A water-perfused 8-channel catheter (4 radial ports at the same level and 4 separated by 5-cm intervals) was used to assess esophageal pressures with the patient in the supine position. The LES was examined with the 4 radial ports. A station pull-through measurement of the LES pressure determined the characteristics of the sphincter. The LES pressure was averaged over a series of 3 respiratory cycles. The esophageal body was assessed over a minimum of 10 episodes of deglutition with 5-mL aliquots of water.

24-HOUR ESOPHAGEAL pH MONITORING

Ambulatory 24-hour pH monitoring was performed using a dual probe catheter. The distal probe was located 5 cm above the manometrically determined LES. The proximal probe was located 10 cm above the distal probe. A portable digital data logger (Synectics Medical Inc, Shoreview, Minn) was used to record pH fluctuations, while the patient recorded symptoms in an event diary. All data were downloaded and analyzed by a computer program. Abnormal acid exposure was defined as a pH of less than 4 more than 1% of the total time in the proximal channel and more than 4% in the distal channel, or a DeMeester composite score greater than 14.7.

STATISTICAL ANALYSIS

All data were recorded in a prospectively collected database (FileMaker Pro4; FileMaker Inc, Santa Clara, Calif) and subsequently imported in a statistical package (STATA7; Stata Corp, College Station, Tex) for analysis.

GERD have to be defined. In this study we sought to determine (1) the frequency with which hypercontractile esophagus was seen in a population of patients being considered for an antireflux procedure; (2) the effects of total fundoplication on the control of abnormal esophageal acid exposure and the major presenting symptoms; and (3) whether new dysphagia, chest pain, or other problems developed after Nissen fundoplication in patients with GERD and a hypercontractile esophagus.

RESULTS

HAPCs GROUP

There were 8 men and 7 women with a mean age of 47.6 years (age range, 14-82 years). Patients’ clinical presentations are given in Table 1. Heartburn and/or regurgitation were significant symptoms in 12 (80%) of 15 patients. Five patients (33%) reported dysphagia; 5 (33%) reported chest pain.

Preoperatively, manometry showed a distal esophageal amplitude (DEA) between 150 and 180 mm Hg in 10 patients (67%) and more than 180 mm Hg in the remaining 5 patients (33%). Twenty-four-hour pH monitoring was abnormal in all patients (100%) (Table 2). All patients underwent laparoscopic Nissen fundoplication. Fourteen (93%) of the 15 patients were contacted at an average clinical follow-up of 27.5 months (range, 1-69 months).

Eight (73%) of 11 patients with heartburn preoperatively reported either no heartburn (46%) or improvement in symptom frequency (27%) after the operation. Three patients’ (27%) conditions did not improve after fundoplication (Figure 1). Dysphagia resolved in 4 (80%) of the 5 patients and improved in the other patient (20%). Only 1 patient developed new dysphagia (Figure 2). Four (80%) of the 5 patients who had chest pain preoperatively had resolution of the symptom postoperatively, whereas 1 patient (20%) reported an increase in its frequency (Figure 3). Three patients developed new chest pain after the operation.
Table 1. Patient Symptom Frequency Characteristics

<table>
<thead>
<tr>
<th>Symptom Grade</th>
<th>Patients With HAPCs (n = 19)</th>
<th>Patients With HLES (n = 6)</th>
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<tbody>
<tr>
<td></td>
<td>Heartburn</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>3 (20)</td>
<td>1 (25)</td>
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<tr>
<td>1</td>
<td>0</td>
<td>0</td>
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<tr>
<td>2</td>
<td>0</td>
<td>0</td>
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<tr>
<td>3</td>
<td>6 (40)</td>
<td>1 (25)</td>
</tr>
<tr>
<td>4</td>
<td>6 (40)</td>
<td>2 (50)</td>
</tr>
<tr>
<td></td>
<td>Dysphagia</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>9 (60)</td>
<td>4 (100)</td>
</tr>
<tr>
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<td>2 (13)</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Chest pain</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>10 (67)</td>
<td>4 (100)</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
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<tr>
<td>2</td>
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<td>3</td>
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<tr>
<td>4</td>
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</tbody>
</table>

*Data are given as the number (percentage) of patients with each frequency of symptom grade. HAPCs indicates high-amplitude peristaltic contractions; HLES, hypertensive lower esophageal sphincter;
0, asymptomatic; 1, symptom occurs once a month; 2, symptom occurs once a week; 3, symptom occurs once a day; and 4, symptom occurs several times daily.

All patients were asked to return for manometry and 24-hour pH monitoring postoperatively. Eight patients (57%) agreed to a second manometry. Mean DEA returned to within normal limits (DEA, <140 mm Hg) in 4 (80%) of the 5 patients who had a mean DEA between 140 and 180 mm Hg before surgery. It remained above 180 mm Hg in the 3 patients (100%) with a DEA higher than 180 mm Hg preoperatively. Twenty-four-hour pH monitoring was normal in 8 (80%) of the 10 patients who agreed to undergo a second manometry, improved in 1 patient (10%), and remained unchanged in 1 patient (10%). Two of the 3 patients with persistent heartburn had abnormal acid exposure on 24-hour pH monitoring. In the 3 patients with new chest pain, 24-hour pH recording revealed normal esophageal acid exposure whereas persistent HAPCs higher than 180 mm Hg were observed in the 2 patients who underwent a second stationary esophageal manometry.

**HLES GROUP**

There were 2 men and 2 women in the HLES group with a mean age of 43 years (age range, 32-55 years). Heartburn and/or regurgitation were present in all patients (100%), but none reported dysphagia or chest pain (Table 2).

Mean LES pressure was 48.7 mm Hg (range, 43-53 mm Hg). Preoperative 24-hour pH monitoring was performed in all patients and confirmed the presence of abnormal GERD in all of them. All patients underwent laparoscopic Nissen fundoplication. All patients (100%) were contacted at an average clinical follow-up of 58 months (range, 1-120 months).

All patients reported complete resolution of heartburn at clinical follow-up. One patient developed dysphagia postoperatively. No patient developed chest pain after the operation. Two (50%) of 4 patients underwent second 24-hour pH monitoring and stationary esophageal manometry postoperatively. The LES pressure returned to normal limits in 1 patient. In the patient who developed dysphagia, the LES pressure remained elevated after fundoplication. Twenty-four-hour pH monitoring was normal in both patients.

**COMMENT**

Hypertensive LES and HAPCs are relatively rare among patients with GERD being considered for an operation. In fact, these findings were present in only 2.9% (19/643) of our patients. This is not surprising since the hypercontractile esophagus is more frequently reported in patients evaluated for noncardiac chest pain or dysphagia than in those studied for typical reflux symptoms. While chest pain and dysphagia may be characteristic of all patients in this broad category, those with HAPC are more likely to have chest pain than those with an HLES are more likely to have dysphagia. Despite differences in the individual characteristic, therapy with smooth-muscle relaxants is effective treatment for these primary esophageal motility disorders in most instances. Even so, the association of functional symptoms such as dysphagia or chest pain with manometric findings of a hypercontractile esophagus does not always predict the response to such treatment. This may be due to the coexistence of GERD, which may play a role in the initiation of the symptoms perceived by these patients. The incidence of reflux in patients with HAPCs has been reported to range from 35% to 58%, and in those with an HLES it is approximately 5%. Therefore, there are patients with a hypercontractile esophagus and no reflux who may be effectively treated with smooth-muscle relaxants, but there is a subset of patients with a hypercontractile esophagus and associated reflux who need reflux-specific therapy. We advocate the use of 24-hour pH monitoring in patients with esophageal symptoms and a “hypercontractile esophagus,” particularly when they have failed traditional forms of medical therapy directed to smooth-muscle relaxation.

Achem et al and Adamek et al reported that patients with HAPCs rarely, if ever, experience normalization of peristalsis with medical antireflux treatment. This suggested that the hypermotility disorder may coexist with, but is not due to, reflux. We, too, noted that patients with classic, nutcracker esophagus (HAPCs > 180 mm Hg) did not experience a fall in the amplitude of contractions after surgical correction of reflux. However, manometric patterns returned to within normal limits (< 140 mm Hg) after successful control of...
esophageal acid exposure in 80% (4/13) of our patients whose
preoperative amplitude of contractions was between 140
and 180 mm Hg, suggesting that in these patients abnor-
mal motility may be associated to the existence of reflux
and perhaps not a primary motility disorder.

Our study also suggests that laparoscopic Nissen fun-
doplication effectively controls esophageal acid expo-
sure in patients with GERD and associated hypercon-
tractile esophagus. Indeed, objective measurement showed
complete normalization of pH-metric parameters in 83%
(15/17) of patients 3 to 6 months after surgery. These
results are similar to those we reported for patients with
GERD and normal esophageal motility. 25 In most cases
this was associated with complete relief of the primary
symptoms, ie, heartburn, dysphagia, and chest pain.

Heartburn, the most common symptom in this popu-
lation, was eliminated in almost 60% (8/14) of the cases
and was improved in approximately 80% (11/14) of pa-
tients. Two of the 3 patients with postoperative heart-
burn had abnormal 24-hour acid exposure.

Dysphagia improved in all patients after successful sur-
gical control of reflux, and it persisted in 1 patient with a
positive result after 24-hour pH monitoring, thus suggesting that, in patients with hypertensive lower esophageal sphincter and GERD, dysphagia may likely be due to the irritant effect of acid on the esophageal mucosa rather than to the hypertonic sphincter or the aberrant contractions of the esophageal musculature. It also emphasizes the value of ruling out abnormal reflux in all patients with hypertensive esophageus, especially before initiation of therapy with a smooth-muscle relaxant. New dysphagia, as a result of fundoplication, is a rare event that occurs in less than 10% of the patients.

The effect of surgery on chest pain is, in our opinion, more problematic. In fact, while 85% (5/6) of our patients reported complete resolution of the symptom, the one fifth without chest pain preoperatively developed chest pain after the operation. All of these patients had normal esophageal acid exposure during 24-hour pH recording performed postoperatively, while DEA of contractions was greater than 180 mm Hg in the 2 patients who underwent second stationary manometry postoperatively.

We lack a clear explanation for the onset of postoperative chest pain in this group of patients. Using balloon distention, Mujica et al26 could reproduce chest pain in patients with nutcracker esophagus, hypothesizing that these patients might have a hypersensitive and unyielding esophagus, and that visceral hyperalgesia may be relevant to the pathogenesis of their pain. Stein et al27 demonstrated that Nissen fundoplication creates a relative outflow obstruction in the distal esophagus. We have hypothesized that by this mechanism, a fundoplication may lead to dilation and stretching of the distal esophagus, thus causing chest pain postoperatively. This remains a speculative hypothesis; even so, based on our results, we recommend that surgeons be aware of the potential for the development of this problem and appropriately discuss the issue with their patients before doing surgery.

From a clinical perspective there are 2 scenarios related to the hypertensive lower esophageus: (1) those patients who primarily report dysphagia and/or chest pain and (2) those patients who also have associated symptoms suggesting the presence of abnormal gastroesophageal reflux. We believe that the investigation of the first group should include the performance of a 24-hour pH monitoring as reflux may be present without heartburn or regurgitation and the medical and/or surgical correction may positively affect the outcome of those symptoms. When reflux is absent, their condition should be managed with therapy directed to the smooth-muscle function. For those (group 2) in whom reflux is already suspected, the objective demonstration of its presence should be followed by the institution of appropriate (medical and or surgical) antireflux therapy. Since reflux is always easier to deal with than modifications of smooth-muscle function and since its correction leads to the resolution of symptoms in many patients, reflux should always be investigated and corrected.

Gastroesophageal reflux disease is occasionally associated with a hypertensive esophageus. It may contribute to symptoms such as chest pain and dysphagia; nevertheless, these symptoms can also be caused by the primary esophageal motility disorder. An effective Nissen fundoplication improves esophageal acid exposure and relieves most of the symptoms in these patients. Therefore, the presence of a hypertensive esophageus should not dissuade the surgeon from performing a total fundoplication. The development of chest pain postoperatively is more likely in this subset of patients and should be discussed when considering fundoplication.

This paper was presented at the 109th Scientific Session of the Western Surgical Association, San Antonio, Tex, November 14, 2001.

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REFERENCES

I have a concern that as lots of people are coming through and getting evaluated, there is a temptation to have patients on the periphery of normality suddenly have GERD. This is the first paper that I have heard presented on reflux with no mention of esophagitis. This is interesting. It is a sign of the times. The presentation did not describe anything about esophagitis. Instead, we are treating numbers. I am concerned about that.

Dr Pellegrini: As Dr Deschamps implied, this study was retrospective, but it was based on the analysis of data prospectively collected in our database. Therefore, what we did is look at all patients who eventually underwent an operation for GERD—the 728. From those patients, we identified those who had the abnormality in question and examined the outcome at the end of 2 years. That was the idea. We do not, therefore, have a control group. It would have been important to have one and I think it would give us, if we could do a prospective randomized control in this population, further information.

When we say 3 patients had chest pain postoperatively—and we emphasized that on the study—we mean 3 of 18 patients who had hypercontractile esophagus, not 3 of 728. This hopefully answers Dr Donahue’s question as well.

Do we pay attention to x-ray films? Yes. This was not described herein because we pay attention to x-ray films in as much as they provide us an anatomical landmark for the operation. We do not get any functional information from those x-ray films, other than occasionally the hypercontractile esophagus may manifest itself on the x-ray films with tertiary contractions.

Why did we exclude patients who had incomplete LES relaxation? We did that because many of those patients end up having achalasia and we did not want to put anybody who did not have, in our opinion, bona fide reflux.

Do we do myotomies? We have treated with myotomy patients who have chest pain and hypercontractile esophagus but not when they have reflux. We have abstained from doing myotomy in patients with reflux.

Dr Donahue made reference to a “5% away from the mean.” Let me clarify that in this study the patients who were considered to have abnormal peristalsis were outside the 95th percentile, so the values that you are seeing here are outside the bell-shaped curve and, therefore, would be abnormal in any laboratory. This is not 5%; it is outside the 95th percentile for healthy subjects.

Is there a causal relationship between reflux and the hypercontractile esophagus? The only thing I can say is that if you look at heartburn, we were able to cure heartburn in a large number of patients, namely, about 80% of the patients, 78.9% of the patients who had heartburn preoperatively did not have heartburn postoperatively. That is what it did. Did heartburn in these patients have anything to do with a hypercontractile esophagus? I do not know, but again the hypercontractility disappeared postoperatively (when the amplitude of peristalsis was between 140-180 mm Hg preoperatively). Perhaps there is a relationship. I cannot say anymore than that.

Chest pain is a problem. Chest pain was present in 4 and went away in 3, but in 3 patients who did not have it, it became a problem postoperatively, and I think that is something that goes back to what Dr Deschamps says. Maybe those patients would have been better served with a myotomy and some form of antireflux procedure. We did not do it and we cannot comment on that.

Dr Donahue said that there is no mention of esophagitis, and it is true. We did not mention esophagitis and we should include that in the manuscript. We have these data. Approximately 60% of the patients we operated on with abnormal gastroesophageal reflux on 24-hour pH monitoring do not have erosive esophagitis. Erosive esophagitis is something of the past. Most of the patients that are operated on today have an abnormal 24-hour pH monitoring, have abnormal acid exposure, have symptoms, but have not developed esophagitis.

DISCUSSION

Claude Deschamps, MD, Rochester, Minn: Dr Barreca and his colleagues have nicely and carefully characterized a small group of patients with GERD that we do not see very often in everyday clinical practice. These patients have the potential of generating anxiety postoperatively, both for the patient and the surgeon alike.

Dr Barreca’s group looked back at their laparoscopic reflux practice of the past 7 years and found a group of patients with HLES or HAPC. The bottom line is that, for the most part, functional results after laparoscopic Nissen fundoplication in those patients were good. Three patients in the HAPC group had de novo chest pain postoperatively and 1 patient in the HLES group had dysphagia postoperatively.

This is a retrospective study where the true denominator, the number of patients with GERD and chest pain coming to their clinic for evaluation, was not mentioned, and it is possibly a very hard number to get.

The authors included 5 patients with paraesophageal hernias and these patients probably have a different physiology and one could theorize that the manometric abnormalities are secondary to obstruction rather than an idiopathic motility abnormality. A control group, even historical, would have been better. Who can tell what the authors would have found? They study their patients so well, who could tell what they would have found in the match group when examined with their questionnaire looking at chest pain and dysphagia?

This is a very useful study because there is new information for all of us. The question is what to do with that information. These patients were treated like everybody else and still did okay. I certainly agree with the authors that it is probably not worth changing the indication or the type of antireflux procedure for what amounts to be 3 patients with chest pain postoperatively of 728 patients, about less than 0.4%.

My questions are the following: There was mention of the barium swallow examination, but we have not seen the data. Has its importance changed in your preoperative workup over the recent years? Do you pay any attention to that barium swallow test result anymore?

Why in that GERD population did you exclude patients with incomplete relaxation of the LES? Will you be tempted to offer thoroscopic myotomy to some of the patients with HAPC if they develop chest pain postoperatively without reflux?

Philip E. Donahue, MD, Chicago, Ill: I too enjoyed the paper and the manuscript, which I received just before the meeting. You know, I am not as enthusiastic about this report because I presented some of the concerns when he said deviations from normality. These represent the 5% at the end of the bell curve of the population and, it seems, they are considered “abnormal.” That strikes the wrong chord for me; because they represent the tail ends of the population distribution does not mean that they have a disease. I have 3 questions.

Could you verify that you are not suggesting any causal relationship between those hypertensive contractions and the symptoms in these patients? It has always been a big problem correlating LES pressures and peristaltic amplitude pressures with patient symptoms. Even in the nutcracker esophagus or patients with crushing pain, there is no definite correlation between amplitude of peristalsis and pain. Could you clarify that please?

The manuscript is more reserved in its conclusion about resolving symptoms compared with the abstract in the program booklet that talks about having clinical resolution of heartburn and more in the vast majority of patients. Yet, if we look at the numbers, there were 4 of 18 patients who preoperatively had chest pain; there were 4 of 18 patients postoperatively with heartburn who had chest pain; by my calculation, only 56% had resolution.