Laparoscopic Heller Myotomy With Toupet Fundoplication

Outcomes Predictors in 121 Consecutive Patients

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Hypothesis: This study was performed to assess the intermediate-term outcomes after laparoscopic Heller myotomy and posterior Toupet fundoplication in a single-surgeon series with the expectation of identifying patient and disease factors associated with poor outcomes.

Design: Retrospective analysis of prospectively collected data.

Setting: Tertiary care teaching hospital with a comprehensive esophageal physiology laboratory.

Patients: A total of 121 patients undergoing laparoscopic Heller myotomy with Toupet fundoplication (between December 1, 1996, and December 31, 2004) for achalasia were included.

Interventions: All patients had preoperative objective documentation of achalasia. A 5- to 6-cm-long myotomy was performed on the distal esophagus. The myotomy incision was extended 2 cm onto the stomach. A partial (270°) posterior Toupet fundoplication was performed as an antireflux mechanism in all patients.

Main Outcome Measures: Data on preoperative and postoperative symptoms, manometry, and 24-hour ambulatory pH were prospectively collected. Symptoms were recorded with a standardized assessment tool. Patients with postoperative dysphagia scores of 2 or greater were considered treatment failure. Logistic regression modeling was performed to identify variables significant for poor outcomes.

Results: Preoperatively, 89 patients (73.6%) had severe dysphagia (dysphagia score, 3 or 4) and 32 patients (26.4%) had mild or moderate dysphagia (dysphagia score, 1 or 2). After a median follow-up period of 9 months, 102 patients (84.3%) (P<.001) had excellent relief of dysphagia (dysphagia score, 0 or 1). Eight additional patients (6.6%) demonstrated a significant (25%–75% [P=.01]) improvement in dysphagia scores. Only 11 patients (9.0%) had either no change or worse dysphagia. Postoperatively, all patients with manometry had a normal lower esophageal sphincter pressure (mean±SD, 14.7±6.6 mm Hg; P<.001) and good lower esophageal sphincter relaxation. Odds of failure were greatest for patients with severe preoperative dysphagia, male patients, and patients with classic amotile achalasia. Of the 60 patients having heartburnlike symptoms preoperatively (mean±SD score, 2.52±1.00), 19 (31.7%) continued to have similar symptoms after surgery. Sixteen (33.3%) of the 48 patients having postoperative pH studies demonstrated objective reflux (DeMeester score, >14.7). Five (31.2%) of these patients had symptoms of their reflux.

Conclusions: Dysphagia improves in most patients after laparoscopic Heller myotomy with partial fundoplication. Patients with severe preoperative dysphagia, esophageal dilation, or amotile achalasia may have greater chances of a poor outcome.

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Chalasia is an idiopathic primary motility disorder characterized manometrically by a poorly relaxing lower esophageal sphincter (LES) and complete loss of primary peristalsis, which leads to a compromise of the primary function of the esophagus. It untreated, it leads to an extremely poor quality of life because of progressive dysphagia, esophageal dilation, and stasis. All current treatments are palliative, as they are directed toward relieving dysphagia and preventing stasis-related complications, but do not restore normal esophageal motility. Surgical myotomy, endoscopic pneumatic dilation, and botulinum toxin injections of the LES are the most commonly used techniques for the treatment of achalasia at present, all of which seem to provide excellent symptomatic relief at least initially. Although most studies indicate that surgical myotomy is superior to endoscopic approaches in providing long-term benefits, it is associated with increased morbidity compared with endoscopic approaches. The long-term outcomes of surgical myotomy are not as good as those of endoscopic approaches in terms of dysphagia relief, but the latter approaches are associated with a high rate of re-treatment due to recurrence of symptoms. Therefore, surgical myotomy remains an important option in the treatment of achalasia.
relief of dysphagia.\textsuperscript{5,9} many physicians still favor endoscopic treatment because of the relatively higher morbidity associated with laparotomy or thoracotomy.\textsuperscript{10-13} Surgical myotomy, however, has regained primacy since the introduction of a minimally invasive laparoscopic approach in 1991.\textsuperscript{14} This is because of the obvious advantages associated with this approach, including less postoperative pain, a shorter hospital stay, and an earlier return to work.\textsuperscript{15} Current data suggest that, in experienced hands, results of laparoscopic Heller myotomy (LHM) are excellent and are comparable with those of open surgery.\textsuperscript{8,16-20} Even so, the symptoms of achalasia may recur in 6% to 23% of patients, and little is known, at present, about the factors that may lead to failure after myotomy. The aim of this study was to evaluate intermediate-term outcomes after LHM with a posterior 270° Toupet fundoplication and to identify patient, technical, and disease factors associated with or predictive of poor outcomes.

### METHODS

#### PATIENTS

Patients were selected from a prospective database of patients undergoing esophageal procedures at our institution. Patients were included only if they had a preoperative diagnosis of achalasia, underwent LHM with 270° posterior Toupet fundoplication, and had a postoperative follow-up of at least 6 months’ duration. Patients undergoing LHM with no antireflux procedure and those undergoing Dor fundoplication were excluded from the study, as were patients who had surgery before 1996, when a different symptom assessment tool was in use. A total of 158 LHMs were performed by one of us (L.L.S.) between December 1, 1996, and December 31, 2004. One hundred forty patients with achalasia satisfied the selection criteria for this study. Nineteen of these patients were eliminated because of incomplete follow-up, resulting in a study population of 121 patients. Forty-five of the patients had had previous endoscopic interventions (bougienage, 35; balloon dilation, 10; and/or botulinum toxin injections, 8), and 7 patients had a previous myotomy.

#### OUTCOME MEASURES

Baseline demographics and data on preoperative and postoperative symptoms, upper gastrointestinal tract studies, manometric data, and 24-hour ambulatory pH were prospectively collected on standardized data collection forms, which were maintained in an electronic database system (Microsoft Access 97; Microsoft Corp, Redmond, Wash). Symptoms were recorded with a symptom assessment tool using a scale of 0 to 4, with higher ordinal values representing greater frequency of symptoms (Table 1). Baseline demographics and preoperative clinical data were obtained at the time of the first office visit. Achalasia was defined manometrically by the complete absence of primary peristalsis of the esophageal body. Other manometric characteristics of achalasia, such as failure of the LES to relax to gastric baseline with swallowing or the presence of a hypertensive LES, were also recorded. Esophageal dilation was determined by the maximum esophageal diameter on review of upright barium swallow radiographs. Minimal dilation was described as less than 2.5 cm, moderate as 2.5 to 5 cm, and severe as greater than 5 cm. Data on operative time and intraoperative complications were acquired at the time of surgery. The primary symptom outcomes for this study were dysphagia and sensation of gastroesophageal reflux.

Patients were followed up at 1 to 2 weeks after surgery, and again at 6 weeks if indicated. At 3 months, patients were asked to complete a symptom assessment form. After a postoperative period of 6 months, all of the patients were recalled and encouraged to undergo esophageal manometry and 24-hour ambulatory pH testing at no charge. Symptom assessment forms were administered at each visit. Long-term follow-up of all patients was done by telephone interview every year, and symptomatic patients were brought back for further testing. Patients with a postoperative dysphagia score of 2 or higher were considered to represent treatment failures.

### INTERVENTIONS

All surgical procedures were performed under the supervision of 1 of us (L.L.S.). Surgery residents or fellows were involved in most of the cases. A posterior 270° wrap (Toupet fundoplication) was the primary antireflux surgery used after an LHM. Dor fundoplications or no wrap were used occasionally for specific anatomic reasons or because of a failed Toupet procedure; these patients were excluded from the present study to achieve uniform data.

Five trocars were placed in the upper part of the abdomen. The gastroesophageal junction and lower mediastinal esophagus were widely mobilized while both vagus nerves were preserved. The short gastric vessels were routinely divided. A 54F to 56F bougie was used in all cases. A myotomy to the level of submucosa was started on the anterior gastric cardia, 2 cm below the gastroesophageal junction, and was extended 5 to 6 cm proximal to it on the anterior esophageal wall. Adequacy of the myotomy was assessed by noting mucosal bulging without any visible crossing fibers and by performing endoscopy if indicated. Both crura were loosely approximated posterior to the esophagus, and a 270° posterior Toupet fundoplication was completed by suturing the wrapped fundus to the edges of the myotomy as well as to the right and left crura. A closed suction drain was placed adjacent to the myotomy, and a watersoluble contrast study was performed before the drain was removed and oral feeding was started the following day.

Manometry was performed with an 8-channel water-perfused catheter. The LES was located by means of the stationary pull-through technique, and the resting LES pressure (LESP), LES relaxation, and esophageal body contractility were determined for a minimum of 10 wet swallows. A commercial software program (Medtronic, Inc, Stockholm, Sweden) was used for the interpretation of manometry tracings and data analysis.

### Table 1. Scale for Assessment of GER and Dysphagia Symptoms

<table>
<thead>
<tr>
<th>Score</th>
<th>GER Symptoms</th>
<th>Dysphagia</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Never</td>
<td>Never</td>
</tr>
<tr>
<td>1</td>
<td>Occurs once or twice a month but not weekly</td>
<td>Rarely (once or twice a month but not weekly) to bread or meat</td>
</tr>
<tr>
<td>2</td>
<td>Occurs once or twice a week but not daily</td>
<td>Occasionally (once or twice a week but not daily) to thick food</td>
</tr>
<tr>
<td>3</td>
<td>Occurs daily but not continuously</td>
<td>To thin liquids</td>
</tr>
<tr>
<td>4</td>
<td>Occurs daily and continuously (all the time)</td>
<td>Cannot swallow oral secretions</td>
</tr>
</tbody>
</table>

Abbreviation: GER, gastroesophageal reflux.
The means of all continuous variables were compared by appropriate parametric or nonparametric tests. Categorical variables and proportions were compared with the χ² test or the Fisher exact test. Logistic regression modeling was performed to identify variables significant for the prediction of a poor outcome. Factors included in the model were age, sex, weight, severity of preoperative dysphagia (mild to moderate [grade 1 or 2] vs severe [grade 3 or 4]), amiotile vs vigorous achalasia, resting LESP (hypertensive vs normotensive), preoperative endoscopic interventions, previous myotomy, preoperative esophageal dilation, and postoperative resting LESP (>20 mm Hg vs ≤20 mm Hg). P<.05 was considered to be significant. All data are reported as proportions, mean±SD, or median (range).

RESULTS

Of the 121 patients, 48 (39.7%) were male and 73 (60.3%) were female. The mean age of the patients was 46.4±14.1 years; mean weight was 76.9±20.0 kg. Preoperatively, 89 patients (73.6%) had severe dysphagia (dysphagia score, 3 or 4) and 32 patients (26.4%) had mild or moderate dysphagia (dysphagia score, 1 or 2). Sixty patients (49.6%) had heartburnlike symptoms preoperatively. The mean preoperative score for these reflux symptoms was 2.52±1.00.

All patients included in this study had preoperative documentation of complete absence of primary peristalsis on manometry. In 51 patients who underwent preoperative manometry outside of our facility, either the original tracings were obtained or the physician who performed the study was contacted to ensure the diagnosis of achalasia; otherwise, the study was repeated in our laboratory. Immediate preoperative manometry was performed in our esophageal laboratory in 70 of the 121 patients. All manometry and radiographic studies were interpreted by one of us (L.L.S.). The diagnosis of achalasia was based on manometric criteria (completely aperistaltic esophageal body and a poorly relaxing LES [<50% relaxation, or relaxation pressure >6 mm Hg]) and barium swallow study findings showing delayed transit and/or dilation of the esophageal body. Sixty-five patients (53.7%) demonstrated an amiotile variety of achalasia. Mean preoperative resting LESP was 38.3±23.9 mm Hg and mean LES relaxation was 62.9±28.7%. Sixteen patients (13.2%) demonstrated nearly normal LES relaxation (>90%) but had radiographs typical of achalasia and classic symptoms, and 46 (38.0%) had a hypertensive LES (mean resting LESP, 58.6±24.2 mm Hg). After a median postoperative 9-month follow-up (range, 6–48 months), 102 patients (84.3%; P<.001) had relief of dysphagia after surgery (dysphagia score, 0 or 1), 8 patients (6.6%) demonstrated a 25% to 50% improvement in their dysphagia (P=.01), and 11 patients (9.0%) had either no change or a worsening of their dysphagia (Figure 1).

Eighteen (94.7%) of 19 patients with postoperative failure had severe preoperative dysphagia, while 71 (69.7%) of 102 patients with a favorable outcome had grade 3 or 4 dysphagia before surgery (odds ratio, 11.31; 95% confidence interval, 1.45-88.22; P=.01).

A total of 43 patients underwent postoperative manometry. Mean postoperative resting LESP was 14.7±6.6 mm Hg. This was significantly (P<.001) lower than preoperative values (Figure 2). All patients had improved apparent LES relaxation after LHM (average nadir, 90.2%±27.6%; interquartile range, 70.1%-108.8%), and/or with a relaxation pressure less than 6 mm Hg. There was, however, no restoration of primary esophageal peristalsis in any patient.

Preoperative upper gastrointestinal tract studies with contrast were available for 94 patients (77.7%). Of 15 patients who continued to have postoperative dysphagia, 8 (53.4%) had severe (maximum diameter, >5 cm) esophageal dilation as compared with 26 (32.9%) of the 79 patients who had a favorable outcome. Odds of having postoperative esophageal dilation were 2.3 (95% confidence interval, 0.7-7.1; P=.22) in patients having postoperative failure as compared with patients with no postoperative dysphagia.

Of the 60 patients who had heartburnlike symptoms preoperatively, 19 (31.7%) continued to complain of heartburn after surgery (mean score, 2.2±1.0). Twelve patients (19.7%) demonstrated new-onset reflux symptoms after surgery (mean score, 2.0±0.9). A total of 48 patients had postoperative pH studies; of these, 12 (25.0%) were complaining of reflux symptoms. Of the 73 patients who did not undergo postoperative objective testing, 18 (24.7%) complained of heartburn (P=.86). Sixteen (33.3%) of the 48 patients having postoperative pH studies demonstrated objective reflux (DeMeester score >14.7). Only 5 (31.2%) of these 16 patients, however, had any symptoms of reflux.

Results of regression modeling are summarized in Table 2. None of the factors included in the regression modeling was identified as being a statistically significant predictor of a poor outcome. However, the odds of failure were higher among patients with severe preoperative dysphagia, in male patients, in patients with the amiotile variety of achalasia, and in patients having preoperative esophageal dilation.

Intraoperatively there were no conversions to open procedures in the series. Mean±SD operative time was 2 hours 18 minutes±2 hours. Mean blood loss was 65.0±54.5 mL. Intraoperative and immediate postoperative complications are summarized in Table 3. Of the 8 esophageal mucosal tears that occurred, 7 were identified and repaired at the time of surgery and patients had no sequelae. In 1 patient an esophageal leak was identified at the time of routine postoperative upper gastrointestinal tract study. The perforation was successfully repaired thoracoscopically; however, the patient had an extended hospital stay of 3 weeks because of a postoperative myocardial infarction. An additional patient had a postoperative myocardial infarction and was discharged on postoperative day 12. Mean hospital stay for the remaining patients was 1.7±1.3 days. One patient (0.8%) in this series died on postoperative day 11 of aspiration pneumonia.

COMMENT

Laparoscopic Heller myotomy has become the standard surgical treatment for patients with achalasia during the...
past decade. Most patients have good to excellent relief of dysphagia with this treatment; however, about 6% to 23% of patients may continue to have dysphagia.8,16-20 Patients who fail to have good outcomes after cardiomyotomy generally have a poor quality of life10 and may require invasive endoscopic interventions,21 revision of myotomy,22 or even esophagectomy23 to relieve their symptoms. In addition to determining the outcomes of LHM with 270° posterior Toupet fundoplication, one of the primary goals of the present study was to analyze the influence of various preoperative and postoperative factors on outcomes, particularly dysphagia and reflux symptoms.

Technical factors such as inadequate myotomy and tight fundoplication are thought to be the most important cause of persistent dysphagia after myotomy.24,25 In a review of 100 consecutive Heller myotomies without fundoplication, Sharp et al17 reported that an inadequate myotomy, as judged by a residual LESP greater than 18 mm Hg, was associated with a significantly higher incidence of postoperative dysphagia. In the present series, we had 11 patients with postoperative LESPs greater than 20 mm Hg but less than 30 mm Hg. None of these patients had postoperative dysphagia. This is most likely because of the interesting phenomenon we noted of an apparent return of receptive relaxation after myotomy. All patients undergoing postoperative manometry and no dysphagia had a decrease in resting pressure to less than 6 mm Hg in response to an induced swallow. It is not clear whether this
response is a true neurologically mediated one or merely a physiologic reaction to the presence of a swallowed bolus. Regardless, this basal pressure may be a very important factor in minimizing postoperative dysphagia. One patient did have a high postoperative LESP of 33.4 mm Hg and also had grade 2 dysphagia.

Among the various preoperative factors, LESP\(^{16}\) and esophageal dilation\(^{26,27}\) have been shown to have some correlation with postoperative outcomes. Arain et al\(^{16}\) reported that a higher preoperative resting LESP was a strong predictor of resolution of dysphagia; however, in another study, preoperative LESP was not found to have any influence on outcomes.\(^{28}\) Our data seem to support the findings of the latter study, as results of both univariate and multiple regression modeling did not find increased odds of failure for patients having normotensive preoperative LES. We did find higher odds of failure in patients with marked preoperative dilation of the esophagus; however, it was not statistically significant.

In the present study we also analyzed the influence of several other important preoperative factors, including the severity of dysphagia, amotile vs vigorous forms of achalasia, a history of endoscopic intervention, and previous myotomy. Univariate analysis of these factors showed that severe preoperative dysphagia is the only factor that is significantly associated with poor outcomes (odds ratio, 11.3; \(P = .01\)). Patients with preoperative esophageal dilation, previous myotomy, or endoscopic intervention had marginally higher odds of failure after surgery. When adjustment for confounding variables was performed by multiple logistic regression, none of the factors included in our model was found to be a statistically significant predictor of poor outcome. The odds of failure were still greatest among patients with severe preoperative dysphagia and those with the amotile variety of achalasia (Table 2).

### Table 2. Results of Regression Modeling

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Adjusted Odds Ratio</th>
<th>95% Confidence Interval</th>
<th>(\beta) Coefficient</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative dysphagia severity</td>
<td>3.54</td>
<td>0.26-46.82</td>
<td>1.266</td>
<td>.33</td>
</tr>
<tr>
<td>Sex</td>
<td>2.90</td>
<td>0.16-51.88</td>
<td>1.065</td>
<td>.46</td>
</tr>
<tr>
<td>Preoperative distal esophageal amplitude</td>
<td>2.24</td>
<td>0.23-21.67</td>
<td>0.808</td>
<td>.48</td>
</tr>
<tr>
<td>Preoperative esophageal dilation</td>
<td>1.79</td>
<td>0.06-45.41</td>
<td>0.547</td>
<td>.76</td>
</tr>
<tr>
<td>Postoperative resting LES pressure</td>
<td>1.18</td>
<td>0.08-17.85</td>
<td>0.230</td>
<td>.86</td>
</tr>
<tr>
<td>Weight</td>
<td>1.00</td>
<td>0.96-1.03</td>
<td>0.032</td>
<td>.93</td>
</tr>
<tr>
<td>Age</td>
<td>0.99</td>
<td>0.92-1.06</td>
<td>-0.007</td>
<td>.86</td>
</tr>
<tr>
<td>Preoperative hypertensive LES</td>
<td>0.72</td>
<td>0.06-7.60</td>
<td>-0.325</td>
<td>.78</td>
</tr>
<tr>
<td>Preoperative endoscopic intervention</td>
<td>0.65</td>
<td>0.21-2.05</td>
<td>-0.419</td>
<td>.47</td>
</tr>
<tr>
<td>Previous myotomy</td>
<td>0.25</td>
<td>0.20-3.32</td>
<td>-1.36</td>
<td>.29</td>
</tr>
</tbody>
</table>

Abbreviation: LES, lower esophageal sphincter.

Intraoperative and In-Hospital Complications*:

<table>
<thead>
<tr>
<th>Complication</th>
<th>No. of Patients</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraoperative esophageal mucosal tears</td>
<td>7</td>
<td>All identified and repaired during surgery with no sequelae; 2 in reoperative cases, 2 in patients with previous endoscopic treatments</td>
</tr>
<tr>
<td>Intraoperative esophageal serosal tears</td>
<td>2</td>
<td>Both identified and repaired during surgery with no sequelae</td>
</tr>
<tr>
<td>Blood loss &gt;300 mL</td>
<td>2</td>
<td>One had liver retraction injury; other was reoperative case</td>
</tr>
<tr>
<td>Postoperative gastric leak</td>
<td>1</td>
<td>None</td>
</tr>
<tr>
<td>Postoperative esophageal leak repair</td>
<td>1</td>
<td>Also had myocardial infarction after repair of esophageal leak</td>
</tr>
<tr>
<td>Cardiac complications</td>
<td>2</td>
<td>None</td>
</tr>
<tr>
<td>Aspiration pneumonia</td>
<td>1</td>
<td>Died of aspiration pneumonia on postoperative day 11</td>
</tr>
<tr>
<td>Urinary retention</td>
<td>1</td>
<td>None</td>
</tr>
<tr>
<td>Cellulitis around trocar site</td>
<td>1</td>
<td>None</td>
</tr>
</tbody>
</table>

*There were 18 complications in 15 patients (12%).

Patients are also urged to return for a pH and motility study at no charge as part of routine follow-up. Patients with normal study results are asked to return if they develop recurrent symptoms or heartburn. Patients with abnormal study results are seen more regularly, and 5 patients in this series eventually had revision of their surgery during follow-up. It has been our experience that the vast majority of failures are apparent at 1 year, and, after a year, it is almost impossible to get asymptomatic patients back for studies.

The need for an antireflux procedure after myotomy remains one of the most controversial issues surrounding LHM. It is widely accepted that a complete 360° wrap should not be used in achalasia, but even the use of a partial wrap is controversial. Opponents of using an antireflux procedure argue that a good postoperative LESP (14 mm Hg) with low incidence of reflux symptoms can be achieved without partial fundoplication by avoiding excessive posterior dissection and any closure of the crura.\(^{17}\)
Such techniques, however, need careful long-term evaluation for potential paraesophageal herniation or other problems.

In our series, the mean postoperative resting LESP of 14.7 mm Hg (Figure 2) was similar to that observed by previous authors. Only a small proportion (20.1%) of patients had postoperative LESPs greater than 20 mm Hg, and this did not increase the chances of a poor outcome. Our observations suggest that a modest postoperative LESP can be achieved by performing a carefully fashioned Toupet repair in combination with an adequate extension of the myotomy onto the anterior gastric wall, and that this also achieves a secure closure of the hiatus. Most important, it does not lead to an increased barrier to the passage of food. Oelschläger et al18 also demonstrated that, with adequate extension of the myotomy onto the gastric cardia and with a Toupet fundoplication, a low LESP can be achieved without increasing the incidence of postoperative reflux.

The postoperative rate of reflux detected by pH testing was 33.3% in our study, which is somewhat higher than is frequently reported by other authors. This may be owing to our aggressive myotomy technique or an artifact from the aggressive use of postoperative pH studies. We advocate postoperative pH studies for all patients with achalasia irrespective of their symptom status. If a patient is found to have significant acid reflux after achalasia surgery, we prescribe acid suppression therapy regardless of whether the patient has heartburn symptoms. We do this in the hope of preventing recurrent dysphagia secondary to peptic stricture. In the current study, one might suspect that more patients would follow through with postoperative testing if they were having problems, which might negatively bias our results. However, the proportions of symptomatic patients among those undertaking pH studies and those who did not undergo postoperative pH studies were the same (25%). This would seem to reduce any sampling bias with regard to pH outcome. We showed as well that postoperative symptoms are an unreliable indicator of objective reflux, as only 6 of the 12 patients who developed new-onset reflux symptoms had an abnormal pH study. On the basis of these data and our previous experience with the poor correlation between postoperative symptoms and objective reflux in patients with gastroesophageal reflux disease,21 we believe that routine 24-hour pH monitoring is optimally performed to detect reflux in all patients undergoing Heller myotomy. Similar observations have been made by other authors.50

**CONCLUSIONS**

Our results indicate that in most patients with achalasia, LHM with 270° posterior Toupet fundoplication provides excellent relief of dysphagia with low intraoperative and perioperative morbidity and mortality. Postoperative reflux remains a problem in 33.3% of patients, and these patients should be closely monitored for esophageal mucosal damage and should probably be treated with antireflux medications. Nonetheless, we prefer good dysphagia relief to low reflux rates, as there is no medication for dysphagia, and progressive dysphagia and esophageal dilation often culminate in esophagectomy. Patients with severe preoperative dysphagia, the amotile variety of achalasia, and severe esophageal dilation should be warned that there may be a greater likelihood of a poor outcome.

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**REFERENCES**

patients may not respond as well, would you recommend some- 
esophagi can't empty quite easily? And, now that you know these 
you define it? Are you including or excluding patients with the

Why is this the case? You included patients operated on over 
series. I noted with surprise that your follow-up is only 9 months.

What the authors have shown today is that in a selective group of patients with acha- 
lasia, Heller myotomy when combined with a Toupet fundo- 
llication completely reverses dysphagia in 85% of the patients 
and alleviates it in an additional 8%. They have also shown that, 
in those who underwent postoperative studies (about one third 
of the population), the operation reduced the lower esopha- 
geal sphincter pressure (LES pressure) in the lower esopha- 
geal sphincter (LES) pressure to approximately 15 mm Hg and 
that abnormal reflux occurs in 30% of those individuals. Fu- 
thermore, they suggested (although I don't think the numbers 
prove it) that among those patients who have severe dyspha-

Dr Pellegrini, thanks so much for your comments; you are 
the ideal discussant and certainly somebody whom we all look 
to as a leader in minimally invasive treatments of achalasia. You 
would be, are you doing anything differently with your patients in terms of 
PPI [proton pump inhibitor] therapy, especially in the asym-
tomatic patient, particularly since more and more of these pa-
tients are young and there is a little bit of concern about asym-
tomatic reflux. Finally, a couple of patients had over 90% LES 
relaxation. Tell us a little bit more about this because by clas-
sic criteria, that would not fall into achalasia. With that degree 
of relaxation and any degree of peristalsis, how would you de-
cide in that case to go ahead and do a myotomy?

Dr Swanström: Thank you, Mr President. I would very much 
like to thank the Association for the privilege of presenting our 
work. I have to also thank Yash Khajanchee, who has worked 
with me for several years and has really been a trailblazer in 
understanding this very interesting disease. He's done a great 
job, as you can all see from this presentation.

Dr Pellegrini, thanks so much for your comments; you are 
the ideal discussant and certainly somebody whom we all look 
to as a leader in minimally invasive treatments of achalasia. You 
asked us about the complete relief of dysphagia, and as we all 
know from our experience in antireflux surgery, the answer al-
ways depends on how you ask the patient and what you are 
asking. We use a numeric scoring system for dysphagia. For 
this study, any patient who scored grade 1 or 0 we put down 
as "complete relief" of dysphagia because we have found when 
establishing our "norms" for this that many asymptomatic vol-
teers will say on occasion that they have some dysphagia. 
Since achalasia patients will remain without motility after sur-
gery, it would be very rare to find one who didn't on rare oc-
casions have some dysphagia. I ascribe our good results to a 
very aggressive approach with our myotomies, extending them 
well onto the stomach, as you have advocated, and always mak-
ing a point of aggressively separating the fibers, peeling the 
myotomized walls back a long way to really open up the distal 
esophagus, and perhaps that partly explains our low dyspha-
gia rates.

A question about the 9-month follow-up in what is essen-
tially a 14-year experience: we did exclude patients from our 
early experience, 1996 and earlier. At that time we used a 
different symptom-scoring tool and probably were in our learn-

Carlos A. Pellegrini, MD, Seattle, Wash: What the authors have 
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geal sphincter pressure (LES pressure) to approximately 15 mm Hg and 
that abnormal reflux occurs in 30% of those individuals. Fur-
thermore, they suggested (although I don't think the numbers 
prove it) that among those patients who have severe dyspha-
gia or a dilated esophagus preoperatively, the outcome of the 
operation tends to be worse than among those who don't have 
those features preoperatively.

We have performed this operation in a manner similar to 
that reported, except that we prefer to extend our myotomy fur-
ther into the stomach, which presumably accounts for our lower 
LES pressure postoperatively. Using the Toupet procedure we 
have seen the same incidence of abnormal reflux but have noted 
higher pressure postoperatively. Using the Toupet procedure we 
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duplication on these patients or, at most, adding a little 90° re-
the fundoplication. I strongly advise not adding another fun-
and try a second myotomy, 180° from the first, and to take down
we had 2 in this series. It's been our philosophy to go ahead
socially patients who actually get worse with a myotomy, of which
What do you do
myotomies are a fascinating clinical puzzle. What do you do
with these unfortunate patients? It's a tough problem, espe-
cially patients who actually get worse with a myotomy, of which
anything less than 5 cm we called minimal dilation; any-
thing over 5 cm we listed as a large degree of dilation; and
I think our results support the intuitive sense that greater di-
lolation represents more end-stage organ failure and therefore
worse outcomes.
You question the use of the Toupet with perforations. You
are correct to assume that many of the Dor repairs that were
excluded from this study group were done to reinforce muco-
sal perforations. On the other hand, I have not found a small
plastic closure of the mucosa to be especially threatening; in
this series, none of the patients with postoperative problems
were those who had a repaired mucosal perforation. If I feel
that the Toupet repair is a better "fit" for a patient, I will sim-
ply do a nice repair, perhaps place some fibrin glue on it, and
watch the patient closely.
How about the role of the Dor? I personally favor the Tou-
pet, mostly because it holds the myotomy open. Once again,
our philosophy is to attempt to minimize dysphagia. I am not
that concerned about reflux. Almost everyone in the world is
on Prilosec or Nexium anyway. Our goal is a more aggressive
opening of the lower esophageal sphincter and a little better
antireflux repair, and I feel that the Toupet achieves this. That
being said, for the last 2½ years we have been involved with a
randomized prospective study comparing the Dor and Toupet
repairs, and those patients, of course, were excluded from this
study as well.
Dr Patti, thank you very much for your comments. Failed
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cially patients who actually get worse with a myotomy, of which
we had 2 in this series. It's been our philosophy to go ahead
and try a second myotomy, 180° from the first, and to take down
the fundoplication. I strongly advise not adding another fun-
doplication on these patients or, at most, adding a little 90° re-
creation of the angle of His. Results for reoperative myotomies
are not great. There is a 50% success rate, at the most. We don't
try a third myotomy. We would instead proceed to a vagal-
sparing laparoscopic esophagectomy in that group.
Dr Smith, it is great to see you, although you are a long way
from home. Duration of symptoms: no, we didn't specifically
look at that. Achalasia patients come with so many odd symp-
toms. Some of them present with chest pain; a lot of them pre-
sent as reflux patients. Because it was difficult to pinpoint, we
didn't record duration of symptoms. We use, and I think it is a
fair surrogate, dilation of the esophagus as an indicator of dis-
eease progression, and perhaps our results also support the pro-
gression of achalasia from vigorous (early) to amotile (late);
a long-standing theory.
Our 30% reflux rate: I think that it is a little bit higher than
most in the literature. I think we are very aggressive, once again,
at relieving dysphagia and will not compromise that in an ef-
fort to stop reflux. I think some more recent data have shown
reflux rates much higher if you don't do a fundoplication, up
to 60%, so I think we are doing something to control it. You
also refer to the growing awareness, supported again by our data,
that patients who complain of heartburn symptoms after sur-
gery most often don't have reflux as measured by 24-hour pH
testing. Should we then put all patients on PPIs after surgery?
It's a valid question because recurrent dysphagia following treat-
ment because of peptic stricture is almost an automatic esoph-
agectomy. Perhaps we should be treating all postoperative acha-
lasia patients with prophylactic medications.
Then, finally, you made a comment on the traditional defi-
nition of achalasia as an amotile esophagus. I think we all agree
with this as the key criteria. Of course, the definition used to
also require a hypertensive LES and then a nonrelaxing LES.
Dr Patti has produced data from his Swallowing Center that
the LES criteria is no longer necessary, finding as we did that
the LES can be hypertensive, normotensive, or even hypoten-
sive. It can also show a wide spectrum of relaxation, ranging
from no relaxation to complete. When we looked very care-
fully, however, at that group of patients with normal LES pres-
ures and what appeared to be relaxation, we noted that the
relaxation often is transient or poorly timed, and I believe Dr
Patti has found the same thing. I believe that the actual 2005
definition of achalasia is an aperistaltic esophagus that is pro-
gressively dilating.