Achalasia Treatment

Improved Outcome of Laparoscopic Myotomy With Operative Manometry

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Hypothesis: Operative manometry detects residual esophagogastric junction (EGJ) high pressure, ensuring complete myotomy.

Design: Consecutive patients undergoing laparoscopic myotomy.

Setting: Tertiary care academic medical center.

Patients: From 1997 to 2003, 139 patients with achalasia underwent laparoscopic myotomy.

Interventions: We assessed myotomy completeness by operative endoscopy and performed operative manometry to measure pressure across the EGJ myotomy. Residual high pressure was isolated and intact muscle divided.

Main Outcome Measures: Esophageal manometry, quality of life, and dysphagia severity score.

Results: Median lower esophageal sphincter pressure was 27 mm Hg preoperatively; 10 patients had sigmoid esophagus and 57 had previous dilation and/or toxin injection. There were 136 laparoscopic myotomies and 3 conversions to open procedures (2%). Operative endoscopy was performed in all patients. Operative manometry, completed in 132 patients (95%), identified residual EGJ high pressure leading to myotomy revision in 45 patients (31 in the first 70 treated). Small perforations occurred in 19 patients, associated with previous dilation and/or toxin injection in 12 patients. One-month follow-up was available in 136 patients (98%); 126 patients had minimal symptoms (93%), whereas 1 had recurrent EGJ high pressure, 5 esophagitis, 3 sigmoid esophagus, and 1 para-esophageal hernia. In 60 patients with complete 1-year follow-up, quality of life and dysphagia improved (P<.05); mean lower esophageal sphincter pressure decreased to 7.6 mm Hg (P<.05).

Conclusions: Operative manometry identifies residual EGJ high pressure and reduces the incidence of incomplete myotomy. Laparoscopic myotomy improves quality of life and dysphagia symptoms and may be the treatment of choice in most patients with achalasia.

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Achalasia is a rare esophageal disorder characterized by impaired lower esophageal sphincter (LES) relaxation with swallowing and aperistalsis of the esophagus. The physiological alterations in achalasia result from damaged esophageal intramuscular neurons. The cause of ganglion cell degeneration and denervation is unknown.

All treatments of achalasia aim to alleviate symptoms, although no treatment reverses the underlying neuropathological changes or associated impaired LES relaxation and aperistalsis. Many nonsurgical therapies have been used to treat this disease, including esophageal dilation, oral nitrates, calcium channel blockers, nitroglycerin, and the injection of botulinum toxin directly into the nonrelaxing LES. These treatments range in mean±SD overall efficacy from 32%±19% (botulinum toxin) to 72%±26% (pneumatic dilation).1 Surgical myotomy has up to a 90% success rate for achieving complete symptom resolution, making it the most effective therapeutic modality for the treatment of achalasia.1

The esophageal myotomy was first described by Heller2 in 1913 and involved both an anterior and a posterior longitudinal esophageal myotomy via thoracotomy. Since that time, many modifications of the original procedure have been added, including laparoscopic approach as well as modifying the length and location of the myotomy. The development of significant gastroesophageal reflux in 60% of patients treated with the thoracoscopic
Heller myotomy has led to the inclusion of an antireflux procedure in most cases. However, one group of surgeons does not recommend an adjunctive antireflux procedure after a myotomy is performed. Failure of the Heller myotomy to relieve symptoms of achalasia has been most commonly attributed to an incomplete myotomy at the time of surgery. The adequacy of myotomy in the operating room has been assessed with operative endoscopy in numerous reports of laparoscopic Heller myotomy. Moreover, endoscopy allows for identification of the squamocolumnar junction. Operative esophageal manometry provides a functional assessment of the myotomy by measuring the pressures at the LES after myotomy and by looking for residual "high-pressure zones" (HPZs). Although manometry is imperative in the diagnosis of achalasia, operative manometry is not widely used in assessing the adequacy of a myotomy during laparoscopic laparoscopic procedures.

This report details a series of 139 patients with achalasia for whom both operative endoscopy and esophageal manometry were used to evaluate completeness of the myotomy during the performance of the Heller procedure.

**METHODS**

Between July 1, 1997, and June 30, 2003, we attempted a laparoscopic Heller myotomy in 139 patients (71 male and 68 female) with esophageal achalasia at Northwestern Memorial Hospital, Chicago, Ill, and Veterans Affairs Chicago Health Care System–Lakeside Division, Chicago. Patient age was 46.3±17.9 years (mean±SD), and the median duration of their symptoms, including dysphagia and regurgitation, was 40 months. Forty-one percent of the patients had received previous therapy for achalasia, including esophageal dilation (n=45) and/or botulinum toxin injection (n=45) and/or botulinum toxin injection (n=45) and/or botulinum toxin injection (n=45) and/or botulinum toxin injection (n=45). Six patients had sigmoid esophagus.

Preoperatively, each patient underwent upper endoscopy, and esophageal manometry was performed with an 8-channel esophageal manometry catheter incorporating a perfused sleeve assembly for accurate measurement of LES pressure (Dentsleeve Pty Ltd, Parkside, South Australia, Australia).

In the first 3 years of this study, patients also completed questionnaires regarding their disease-specific quality of life and severity of dysphagia. For quality of life, they assigned a score of 0 (low) to 6 (high) for each of the following qualities: ability to eat foods they like; ability to eat as much food as they like; ability to enjoy meals with family or friends; ability to sleep comfortably lying flat; and ability to enjoy social activities. Scores for the 5 qualities were tallied to determine the disease-specific quality-of-life score. For the dysphagia severity score, patients were asked to determine their degree of swallowing difficulty for 8 varieties of medication, food, or liquid, with scores of 0 (no difficulty), 1 (mild difficulty), 2 (moderate difficulty), and 3 (unable to swallow). The categories were (1) pills or capsules; (2) meat (steak, pork, chicken breast); (3) apple, raw carrot, or celery; (4) bread, baked fish, or baked potato; (5) mashed potatoes or scrambled eggs; (6) pudding, gelatin, or jelly; (7) water, juice, or milk; and (8) cream or milk shake. Each patient’s scores were pooled for a total dysphagia symptom score (potential range, 0-24).

On the day of surgery, immediately before the administration of general anesthesia, which included paralytic agents, a 4.2-mm–outer diameter 16-lumen esophageal manometry catheter (Dentsleeve Pty Ltd) was placed transnasally and positioned with one half of the recording sites in the stomach. Recording sites on the catheter were arranged in a spiral radial configuration, starting 5 cm from the distal tip and continuing proximally at 1-cm intervals. The catheter was connected to a low-compliance pneumohydraulic perfusion pump (Dentsleeve Pty Ltd) and a computer polygraph reading system (Neomedix Systems Pty Ltd, Warrnambool, New South Wales, Australia).

In the operating room, the patient was placed in a low lithotomy position. After pneumoperitoneum was established, 5 laparoscopic ports were inserted across the upper abdomen. The proximal gastric fundus was pulled down and mobilized by division of the short gastric vessels with the use of 5-mm ultrasonic shears. The phrenoesophageal ligament was incised to expose the anterior gastric cardia and distal esophagus, and limited dissection behind the esophagogastric junction (EGJ) was performed to place a Penrose drain around the distal esophagus. Care was taken to identify and preserve the vagus nerves. Then, upper endoscopy was performed (Pentax Model 2730; Pentax Corp, Orangeburg, NY) and the squamocolumnar junction was identified. Traction was placed in a caudal direction on the EGJ. A longitudinal myotomy was begun on the gastric cardia 1 to 2 cm distal to the squamocolumnar junction and extended proximally into esophageal muscle, to the left of the anterior vagus nerve, for 6 to 8 cm proximal to the squamocolumnar junction. Longitudinal and circular muscle fibers were divided, exposing submucosa. If penetration or perforation of the esophageal or gastric mucosa occurred, these defects were closed with interrupted 3-0 silk sutures.

Completeness of the myotomy was assessed by visualizing a bulge of esophageal and gastric submucosa between the divided muscle fibers in response to endoscopic air insufflation. Remaining circular muscle fibers, identified at this time, were cut. Next, the position of the manometry catheter in the stomach was ensured and/or confirmed endoscopically. The manometric intragastric pressure was set to zero. The catheter was then withdrawn incrementally, 1 cm at a time, to optimize recording of the entire length of the EGJ. With the catheter straddling the EGJ, a positive pressure at any recording site between the stomach and the dilated portion of the esophagus was interpreted to represent a residual HPZ. We probed along the myotomy site with a blunt laparoscopic instrument and simultaneously observed the response on the manometry tracing (Figure 1). This maneuver permits localization of a residual HPZ. Residual intact muscle fibers identified by this method were divided or the myotomy was extended until the HPZ was 3 mm Hg or less. At completion of the myotomy, a Dor anterior fundoplication was created by folding the fundus over the abdominal side of the myotomy and sutured it to the crural diaphragm with 2-0 nonabsorbable suture.

After recovery from general anesthesia, patients were admitted to a surgical inpatient floor. Most patients were offered liquids to drink on the day of surgery and advanced to a soft diet by the next day. For patients who had perforation through the submucosa during the myotomy, an esophagogram x-ray study was performed on the first postoperative day. If extraluminal contrast material was noted, the patient was treated with appropriate antibiotics and not allowed to drink or eat for 3 to 7 days, then the esophagogram radiographic study was repeated. All patients were prescribed a once-daily dose of a proton pump inhibitor until the first follow-up visit.

Patients were routinely examined 1 month postoperatively. After 1 year, we recruited patients to have manometric pressure measured across the myotomy and, for those who had completed preoperative questionnaires, to have an assessment of postoperative disease-specific quality of life and dysphagia severity, using the same questionnaires as were used preoperatively. Data are listed as mean±SD, and statistical significance was determined with the paired t test.
In these 139 patients, there were 136 laparoscopic myotomies performed, with 3 conversions to an open procedure (2%). The mean operating time for the myotomy procedure as well as operative endoscopy and manometry was $2.3 \pm 0.6$ hours (median, 2.0 hours; range, 1.25-4.5 hours). Operative endoscopy was performed in all patients. Operative manometry was successfully performed in 132 of 139 patients. Inability to position the manometry catheter across the EGJ, primarily because of a tortuous sigmoid esophagus and tight LES, resulted in failure to perform operative manometry in 7 patients. An HPZ, ranging from 8 to 20 mm Hg above the baseline intragastric pressure, was identified by operative manometry after the initial myotomy in 45 (34%) of the 132 patients (31 in the first 70 treated). This finding indicated extension of the myotomy or division of residual circular muscle fibers that were not seen initially in each of these 45 patients. In the majority of these patients, the residual HPZ was located at the proximal extent of the myotomy. On the basis of the operative manometric findings, the myotomy was extended up to 1 cm. The range of postmyotomy LES pressure, measured at myotomy completion, was 0 to 3 mm Hg.

Esophageal or gastric mucosal perforations occurred during laparoscopic myotomy in 19 (14%) of 139 patients, 12 of whom had had previous dilation and/or toxin injection; 15 perforations occurred in the first 70 myotomies and 4 in the last 69. Five of these perforations occurred during extension of the myotomy in patients for whom a residual HPZ was identified by manometry. All were repaired at the time of the operation. Eighteen of these patients experienced no adverse consequences as a result of the perforation. However, persistent but asymptomatic perforation was noted in 1 patient who required antibiotics and total parenteral nutrition for 3 weeks (1 week in the hospital) until the perforation healed. Fortunately, this patient had minimal postoperative dysphagia.

One 80-year-old man, who had stable though moderate cardiac disease, died 6 days postoperatively of *Escherichia coli* sepsis. Marked postoperative dysphagia persisted or recurred in 10 patients (7%), because of persistent high LES pressure in 1 (who responded to esophageal dilation), esophagitis in 5 (who were effectively treated with proton pump inhibitors), sigmoid esophagus in 3 (1 required esophagectomy for complete esophageal obstruction and 1 developed esophageal carcinoma 3 years postoperatively), and a postoperative paraseophageal hernia in 1 (it was reduced and the hiatus repaired during remedial surgery).

The mean postoperative hospital stay for all patients was $1.9 \pm 1.9$ days (median, 1 day; range, 1-13 days). One hundred thirty-six patients (98%) were examined 1 month postoperatively, and 60 (55%) of the 110 patients available for 1-year follow-up were recruited for completion of detailed postoperative outcomes (disease-specific quality of life, dysphagia severity score, and postmyotomy LES pressure). One hundred twenty-six (93%) of the 136 patients available for 1-month follow-up reported minimal dysphagia as well as improved swallowing ability. Among the 60 patients examined 1 year after myotomy, disease-specific quality-of-life scores for all 5 quality categories improved compared with preoperative scores (Figure 2). Dysphagia severity scores improved at 1-year follow-up ($12.7 \pm 4.0$ preoperatively vs $2.7 \pm 2.5$ postoperatively; $P<.05$) (Figure 3). The 2 patients with the least improvement in dysphagia severity scores had sigmoid esophagus.

We analyzed the number of patients with persistent HPZs and number of myotomy perforations for consecutive groups of 20 operations performed. With time and increasing operative experience, a declining proportion of patients with postmyotomy residual HPZs (identified by operative manometry) and with myotomy perforations in the distal esophagus or gastric cardio
confirmed by endoscopy) was observed. This observation demonstrates our learning curve for performing the Heller procedure (Figure 4). We also analyzed the amount of LES pressure change from preoperative manometry to 1-year postoperative manometry in the 60 patients for whom this was evaluable (Figure 5); the mean decrease in LES pressure was 21.0 ± 11.2 mm Hg (median, 22 mm Hg; range, 3-70 mm Hg). The mean preoperative LES pressure was 28.1 ± 12.5 mm Hg and the mean postoperative LES pressure, 7.6 ± 3.6 mm Hg (P < .05).

COMMENT

Our experience with the use of upper endoscopy as well as operative manometry to assess myotomy completeness during esophagogastric Heller myotomy demonstrates a high success rate, with only 10 of 136 patients developing recurrent or persistent dysphagia. After the primary cause of postmyotomy dysphagia—esophagitis, paraesophageal hernia, focal residual high pressure—was identified and treated, 7 patients had relief of dysphagia and only 3 patients (2%) with end-stage, sigmoid esophagus—the most difficult cause of achalasia to treat and palliate—remained with significant dysphagia.

Operative upper endoscopy has been used by many surgeons during both open and laparoscopic Heller myotomy. From outside the distal esophagus viewed by the laparoscope, the endoscope can be readily seen transilluminating the esophageal wall. Thus, combined laparoscopic and endoscopic views allow precise identification of the squamocolumnar junction, which determines and guides the proximal and distal extent, as well as the end points of the surgical myotomy. In this manner, the surgeon can be confident that 1 to 2 cm of the myotomy incision resides on the gastric cardia, while the remaining portion of the myotomy extends retrograde up the distal esophagus.

After the myotomy is completed, insufflation by the endoscope distends the region of exposed submucosa, so that residual intact circular muscle fibers in the middle of the myotomy can be seen. From within the lumen, the EGJ should open easily in response to insufflation, compared with the preoperative state in which the EGJ is abnormally tight. If the EGJ does not open easily, the myotomy may need to be extended. Small perforations in the area of the myotomy are also easily detected with endoscopy and repaired at this time.

Operative esophageal manometry was first used by Hill,10 both to measure LES pressure during the performance of antireflux procedures and to assess the adequacy of the myotomy during the Heller operation. Hill et al10 reported a 94% success rate at a mean follow-up of 34 months after using operative esophageal manometry during transthoracic Heller myotomy. Mattioli et al12 used operative manometry to demonstrate the significance of the gastric component of the LES in the HPZ of achalasic patients. In their study of 32 patients, mean LES pressure decreased by 20 mm Hg after myotomy above the EGJ, and it decreased an additional 10 mm Hg after the myotomy was continued below the EGJ onto the gastric cardia.12

These results are comparable with our observations in which the median change in LES pressure immediately after completion of the myotomy was 22 mm Hg.

Although visual inspection with both the endoscope and the laparoscope suggested that the myotomy was complete, a persistent HPZ was discovered in 34% of our patients when operative manometry was performed. In all instances when a residual HPZ was identified by manometry, it was subsequently eliminated by further dissection and/or extension of the myotomy. Clemente et al,11 in their study, found a persistent HPZ in 17 of their 38 patients who underwent operative manometry during a transabdominal Heller myotomy. Their patients went on to report excellent results in postoperative assessment. The results of the Clemente et al study
tomy without any antireflux procedure is as high as 23%. We believe the addition of both endoscopy and manometry during the operative procedure may have reduced the theoretically avoidable persistent dysphagia from technically incomplete myotomy. In a series of minimally invasive Heller myotomies performed for achalasia, Patti et al3 studied 35 patients undergoing left thoracoscopic myotomy and 133 undergoing laparoscopic myotomy. During their procedure, operative endoscopy was used in 89% of the thorascopic group and in 56% of the laparoscopic group. Operative manometry was not used. As a result, the incidence of theoretically avoidable persistent dysphagia from technically incomplete myotomy was as high as 23%. We believe the addition of both endoscopy and manometry during the operative procedure may have reduced the occurrence of postoperative dysphagia.

One area of debate concerns the need for and choice of an antireflux procedure at the time of Heller myotomy. The incidence of gastroesophageal reflux after Heller myotomy without any antireflux procedure is as high as 50%.5,8,13,14 If the myotomy is extended 2 cm onto the gastric cardia, there may be 100% incidence of postmyotomy gastroesophageal reflux.15 Patients with achalasia may be less sensitive to significant gastroesophageal reflux than the general population. Shoenut et al15 found that 38% of patients after treatment for achalasia (pneumatic dilation or Heller myotomy) had significant levels of gastroesophageal reflux; however, 67% were asymptomatic. We agree with Sharp et al7 that there is no need to mobilize the lateral and posterior esophageal attachments to the crural diaphragm. We believe that leaving these attachments intact is an important factor in preventing gastroesophageal reflux after myotomy. All of our patients started treatment with a proton pump inhibitor in the postoperative period to minimize any postmyotomy acid reflux that might occur despite the inclusion of the Dor fundoplasty in the procedure. Most patients subsequently discontinued the medication after several weeks.

In conclusion, laparoscopic Heller myotomy is a feasible and efficacious treatment for patients with achalasia. Moreover, the use of operative esophageal manometry and upper endoscopy during laparoscopic Heller myotomy quantitatively ensures obliteration of the nonrelaxing LES and HPZ. Patients have both short-term and long-term improvement of not only their dysphagia but also their quality of life after this procedure. We believe the addition of manometry to endoscopy to assess completeness of myotomy, especially during the initial experience of the laparoscopic surgeon, aids in identifying residual HPZs and results in the decreased incidence of recurrent or persistent dysphagia that we observed. While we found operative manometry, conducted by interested and skilled clinicians, to be helpful, it requires a level of expertise that may not be available in many institutions. Finally, we are thankful and grateful for our patients, especially the 60 patients who voluntarily returned 1 year after myotomy to complete follow-up questionnaires and to endure another esophageal manometry at a time when they were healthy. These patients with achalasia are very special and we were fortunate to be able to help them.

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REFERENCES

Jeffrey H. Peters, MD, Los Angeles, Calif: At the outset I thought it interesting that each of the last 4 papers involved the use of technology to try to improve the care and outcome of our patients.

Two fundamental abnormalities combine to form the disabilities associated with achalasia, ie, esophageal outflow obstruction caused by the nonrelaxing lower esophageal sphincter and the lack of functional propulsion of the esophagus caused by esophageal body failure. The latter cannot be improved much short of esophageal replacement, and thus the former forms the basis for the treatment of the symptoms associated with the disease. It is now well established and fair to say that the relief of dysphagia and regurgitation and, to a lesser extent, chest pain is dependent upon the degree of the relief of the outflow obstruction; that is, the resting and residual pressures of the lower esophageal sphincter must be lowered to or below 10 mm Hg in order to provide long-lasting symptomatic improvement. Achieving this physiologic outcome, however, can be difficult, particularly with nonsurgical methods, such as balloon dilatation.

Dr DeBord and his coauthors are to be congratulated on recognizing this fact in an excellent study and their attempt to provide the means to consistently achieve this desired result. As you just saw, intraoperative manometry resulted in 45 of 132 patients, or nearly a third, where they found unacceptably high intraabdominal pressures stimulating revision of their myotomy. I have 3 conceptual questions and 1 methodological question, all pertaining to the use of intraoperative manometry.

The first is that the use of manometry is hard enough to standardize, implement, and get surgeons and even gastroenterologists, for that matter, to apply routinely in the management of patients with esophageal disease in the laboratory setting, let alone in the operating room. I wonder if Dr Joehl has any insight and wisdom as to how we can get that accomplished inside the operating room. I suspect it is going to be even harder.

The second, would he agree that the need for intraoperative manometry is related to the surgical technique? Several large studies have now shown that as a good technique has been established, namely the adoption of the laparoscopic rather than the thoracoscopic approach, removing the gastroesophageal fat pad and extending the myotomy 2 to 2.5 cm onto the stomach, LES pressures are consistently reduced below 10 mm Hg, even without the use of intraoperative manometry.

Third, as he demonstrated in the final slide, there is clearly a learning curve, and nearly two thirds of the patients who benefited from intraoperative manometry were in his early experience. Perhaps this should be an adjunct recommended for the inexperienced.

And finally, a methodological question that has some bearing on the interpretation of the results. You have used a sleeve manometry sensor that averages the pressures around the clock face. This is an important nuance. You have done it in a patient on positive pressure ventilation and you have used any elevation in pressure as an indication of an inadequate myotomy, even as low as 6, 7, or 8 mm Hg, which could be caused by the ventilator. And finally, you found that the proximal extent of the myotomy was the usual failure site. I find all of those 4 coincidences to be interesting and suspect that the methodological peculiarities of the study contributed to what you found.

Dr Joehl: First, how do we get the GI people to come to the operating room and to help us? I was very fortunate for many years to work with so many interested esophagologists. So at our institution it was easy once Dr Kahrihas and colleagues came to the operating room. They saw a totally different relationship of the distal esophagus to the diaphragm through the laparoscope.

Second, the need for operative manometry may be related to surgical technique and especially at institutions with low volume.

Third, regarding a learning curve, this certainly involves a significant amount of time for us to learn how to use these different modalities in the operating room. Finally, you asked about the logistics and the mechanics of operative manometry in a setting that is very much nonphysiologic, ie, the patient is under general anesthesia, receiving assisted ventilation, positive pressure breathing, and with 12 to 15 mm Hg pressure of pneumoperitoneum, and we are placing a pressure-sensing catheter into the distal esophagus with its tip in the stomach. Suffice it to say that with the manometry catheter in place, great care was taken to zero all pressures along the catheter to the intraabdominal pressure. It was very easy to find where patients had an elevated pressure across the esophagogastric junction, or at the distal extent or proximal extent of the myotomy. Also, we had laparoscopic access and could easily probe along the various parts of the myotomy, whether it was above the crural diaphragm in the proximal extent of the myotomy or distally onto the cardia. Operative manometry typically took 10 minutes to perform, but sometimes as long as 40 minutes to complete.

James R. DeBord, MD, Peoria, Ill: (1) Do you use any other technical adjuncts like lighted bougies to help guide your myotomy? (2) You used the pressure readings to indicate when you had an incomplete myotomy, but was there a number that you used to determine when the myotomy was complete? And finally, how do you manage those perforations that do occur?

Dr Joehl: Thank you, Dr DeBord. Regarding lighted bougies, we have no experience with them. Certainly at the time of operative manometry, the tip of the endoscope transilluminates the wall of esophagus and certainly served almost the same role as a lighted bougie. Typically these perforations were a millimeter or two in size and we closed them with 3-0 silk suture, often with 1 or 2 sutures.

Regarding an end point for the myotomy, we stopped when the pressure across the myotomy was zero. Now there were many times where the catheter crossed the crural diaphragm, and the left half of the crus was causing increased pressure. By moving the left crus farther to the left, we could show a pressure drop to near zero.

Regarding reflux control, all patients had a Dor anterior fundoplayast.