J Gastrointest Surg (2008) 12:1586–1591
DOI 10.1007/s11605-008-0475-1

Laparoscopic Heller Myotomy: Technical Aspects and Operative Pitfalls

Khashayar Vaziri · Nathaniel J. Soper

Received: 15 November 2007 / Accepted: 7 January 2008 / Published online: 23 January 2008

Abstract Achalasia is a rare motor disorder of the esophagus characterized by aperistalsis and impaired relaxation of the lower esophageal sphincter (LES). The etiology of this disease remains unknown. The current treatment is palliative and relies upon surgical disruption of the fibers of the LES. The technical aspects and operative pitfalls of laparoscopic Heller myotomy are described in this article.

Keywords Achalasia · Heller myotomy · Laparoscopic surgery · Esophagus

Introduction

Achalasia is a rare motor disorder of the esophagus characterized by the absence of peristalsis and impaired relaxation of the lower esophageal sphincter (LES). First recognized 300 years ago as "cardiospasm", it was then described as a functional esophageal obstruction at the cardiac sphincter. The understanding of this disease has evolved over time and is currently termed "achalasia", derived from the Greek term "chalasis" or relaxing.[1]. Achalasia is rare, affecting approximately one per 100,000 individuals in the United States. The etiology remains unknown, but multiple theories involving viral, inflammatory, and autoimmune processes targeting esophageal ganglion cells have been proposed. Most physiologic studies support the theory of dysfunction or loss of the esophageal myenteric plexus.

K. Vaziri · N. J. Soper (✉)
Department of Surgery,
Northwestern University Feinberg School of Medicine,
201 E. Huron Street, Galter 10-105,
Chicago, IL 60611, USA
e-mail: nsoper@nmh.org

Clinical Evaluation

Common symptoms include dysphagia, chest pain, regurgitation, and heartburn. The most common symptom is dysphagia, but chest pain and heartburn lead many physicians to an erroneous diagnosis of gastroesophageal reflux disease (GERD) and a delay in diagnosis. Antireflux medications are unsuccessful in relieving symptoms and over time esophageal dilatation results. The diagnosis is confirmed by functional studies. Esophageal fluoroscopy and manometry are the best diagnostic tests. The two key manometric findings are absence of esophageal body peristalsis and failure of the LES to relax in response to swallowing. The resting pressure of the LES may be normal or elevated. Barium swallow usually demonstrates a dilated esophagus with a "bird's beak" narrowing at the level of the gastroesophageal (GE) junction. Endoscopy should also be performed to exclude causes of pseudoachalasia such as peptic strictures and carcinoma.

Treatment

Current treatments of achalasia do not address the underlying neuropathology and are aimed at relaxation or disruption of the dysfunctional LES. Medications that reduce LES pressure such as isosorbide dinitrate have been used in the past with transient results. Other more successful methods involve intersphincteric injection of botulinum toxin (botox), forceful endoscopic balloon

dilatation (pneumatic dilatation), and surgical myotomy. Although endoscopic therapy offers a less invasive approach, results are generally not as durable as myotomy and repeat treatments are often necessary. Multiple controlled trials between these three treatment modalities have been performed. Botox was found to be less successful than balloon dilation with 12-month success rates of 32% and 70%, respectively.[2] Similarly, the probability of remaining asymptomatic at a 2-year follow-up favors surgical myotomy over botox, 87.5% versus 34%.[3] A prospective randomized trial comparing forceful balloon dilatation and open surgical myotomy reported symptom resolution in 51% of endoscopic patients and 95% in the surgical group after 5 years.[4] First line treatment of achalasia has traditionally been pneumatic dilatation, but the introduction of laparoscopic Heller myotomy with its reduced surgical morbidity has led to a paradigm shift. Laparoscopic Heller myotomy offers the most effective and durable treatment of achalasia. Nonsurgical candidates can benefit from repeated balloon dilatation, which carries a low but finite risk of esophageal perforation.

Surgical management requires a delicate balance of relieving esophageal outflow obstruction while maintaining a protective antireflux mechanism. Myotomy performed without an antireflux procedure is associated with increased esophageal acid exposure and esophagitis.[5] Heller myotomy with partial fundoplication significantly reduces esophageal acid exposure and the overall relative risk of postoperative GERD when compared to myotomy alone.[6] Studies have shown that both the Dor and Toupet fundoplications are effective with low morbidity and short-term failure rates.[7–11] We describe in this paper the technical aspects of laparoscopic Heller myotomy with partial fundoplication, and common operative pitfalls are discussed. As we preferentially perform a Toupet fundoplication after the myotomy, this will be the focus of the technical description.

Operating Room Setup

Patient positioning, operating room setup, communication, and an experienced operating room team are key elements in achieving successful and reproducible results. A general anesthetic and good muscle relaxation is required to ensure an adequate intraabdominal working space. Rapid sequence intubation is preferred as many achalasia patients will have retained food or secretions in their esophagus. The patient is placed supine on the operating room table with legs abducted on flat padded leg boards to minimize the likelihood of lower extremity neurovascular injury. The right arm is tucked at the patient's side and the left arm remains on an arm board. The patient should be well secured, as steep reverse Trendelenberg is needed for the majority of the operation, displacing the intraabdominal organs from the subdiaphragmatic area, and bringing the operative site closer to the surgeon. This is achieved with the use of a vacuum beanbag mattress. The surgeon stands between the abducted legs allowing easy access to the upper abdomen and minimizing muscle strain and fatigue. The first assistant stands to the right of the patient and the scrub nurse to the left. The camera operator assumes a seated position to the surgeon's right allowing for a comfortable camera operation throughout the procedure. A laparoscopic monitor is placed directly above the patient's head for easy and ergonomically neutral visualization by the operative team. An endoscopic monitor is positioned above the patient's right shoulder to have a side-by-side view with the laparoscope during endoscopy. Two 11-mm and three 5-mm ports are used in a laparoscopic Heller–Toupet operation. Instrumentation includes a 10-mm 30-degree laparoscope, atraumatic graspers, a Babcock grasper, a liver retractor, a needle driver, hook cautery, and ultrasonic shears.

Surgical Procedure

Access to the abdominal cavity is attained approximately 12 cm inferior to the xiphoid process and slightly to the left of midline with a Verress needle. A pneumoperitoneum is established, an 11-mm port is placed and the laparoscope is introduced. This camera port is almost always superior to the umbilicus and care must be taken to ensure that this port is not placed too low, making visualization of the hiatus difficult. A 5-mm port is placed at least 15 cm from the xiphoid process and 3–4 cm below the right costal margin for the liver retractor. The assistant's 5-mm port is placed midway between the camera and liver retractor ports. The surgeon's right hand port is placed approximately 10 cm from the xiphoid process and 3–4 cm below the left costal margin. An 11-mm port is used in this location to facilitate laparoscopic suturing with curved needles. The left lateral segment of the liver is lifted and fixed anteriorly using a self-retaining retractor before placing the final 5-mm port. This port, for the surgeon's left hand, varies depending on the edge of the retracted liver and the location of the esophageal hiatus. Optimally, this 5-mm port is placed in the right subxiphoid area allowing for the camera to look between the surgeon's left and right hands for optimal instrument manipulation (Fig. 1).

Dissection begins by dividing the gastrohepatic ligament just superior to the hepatic branch of the vagus nerve using the ultrasonic shears. This dissection is carried up to the level of the right crus of the diaphragm. The surgeon must be aware of the possibility of an aberrant left hepatic artery,

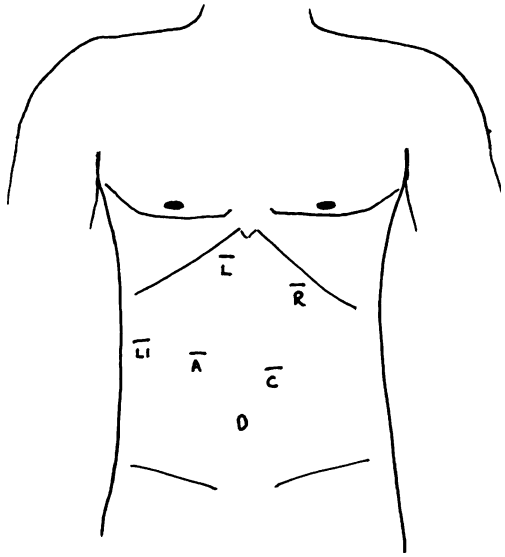

Figure 1 Port placement: *LI*—liver retractor port, *A*—assistant's port, *C*—camera port, *L*—surgeon's left hand port, *R*—surgeon's right hand port.

as incidental transaction can compromise arterial blood flow to the left lateral segment of the liver. Once the right crus is reached, the phrenoesophageal ligament is divided transversely. Only the superficial layers of the phrenoesophageal ligament are divided to avoid injury to the underlying anterior wall of the esophagus and anterior vagus nerve. The assistant provides adequate countertraction by grasping the GE fat pad and retracting caudally. As dissection continues transversely toward the left crus, the fundus and GE fat pad are retracted inferiorly and to the patient's right facilitating mobilization of the cardia.

Following this initial mobilization, a careful hiatal dissection is performed. The assistant provides traction on the esophagus by retracting the GE fat pad caudally. The hiatal dissection begins at the medial border of the right crus. With appropriate tension on the distal esophagus provided by the assistant, the plane between the esophagus and the medial border of the right crus is entered with a blunt instrument. The right crus is then grasped with the surgeon's left hand and retracted to the patient's right. The esophagus is gently swept away in the opposite direction by the surgeon's right hand instrument. The esophagus is gradually and bluntly mobilized in this fashion and the posterior vagus nerve is identified. The posterior vagus nerve is kept with the esophagus and swept away from the periesophageal tissues until the base of the right crus is seen. Tissue between the base of the right crus and esophagus is divided to visualize the origin of the left crus. This blunt dissection is then continued anteriorly along the medial border of the right crus generously mobilizing the mediastinal esophagus (Fig. 2). As the apex of the hiatus is reached, the surgeon's left hand instrument is slipped into this plane and elevates the anterior crural fibers while the esophagus is gently swept away in a blunt fashion with the right-hand instrument. The left-hand instrument continues to retract the crural fibers moving in a clockwise fashion along the hiatus, effectively dissecting ahead of the right hand. The anterior vagus is identified and swept away from the hiatus and toward the esophagus. As the dissection is carried around to the left of the esophagus, the surgeon's left hand instrument is used to bluntly retract the esophagus as the right hand instrument sweeps the hiatus away toward the base of the left crus. Once the base of the left crus is visualized, attention is turned to the short gastric vessels.

The fundus is mobilized by retracting the gastrosplenic ligament to the left and the lateral border of the fundus to the right. The short gastric vessels and all fundal attachments are divided starting approximately 10–15 cm inferior to the angle of His. Adequate mobilization of the fundus is important to ensure a tension-free fundoplication. The ultrasonic shears are used to divide the short gastric vessels up to the angle of His. This dissection plane joins the previous hiatal dissection at the base of the left crus. This allows visualization of the retrogastric space and facilitates the creation of a retroesophageal window. After the fundic mobilization, the hiatal dissection is re-inspected and adequate esophageal dissection is ensured. The GE fat pad is then divided with the ultrasonic shears to expose the GE junction anteriorly, and the anterior vagus nerve is mobilized to avoid injury during the myotomy.

A 6–7 cm esophageal myotomy is planned along the anterior aspect of the esophagus extending onto the gastric

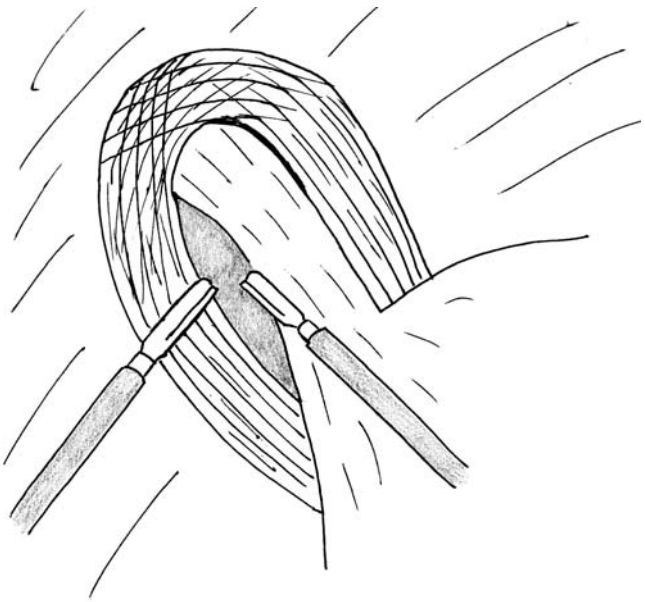

Figure 2 Hiatal Dissection—The hiatal dissection begins at the medial border of the right crus. This dissection can be performed in a blunt fashion.

wall 2–3 cm below the GE junction. Hook cautery is used to deliver low-wattage energy to "map out" and initiate the myotomy. Once the myotomy has been started on the distal esophagus, the edges are grasped with atraumatic graspers, elevated away from the underlying tissue and gently peeled away from the submucosa (Fig. 3). Hook cautery can also be used to lift and then divide/cauterize circular muscle fibers. The myotomy is extended in a cephalad direction and then caudad onto the gastric wall. Care must be taken during the myotomy to avoid injury to the anterior vagus nerve, esophageal perforation, and spiraling of the myotomy. After the myotomy is completed and all muscle fibers are divided, upper endoscopy is performed to ensure adequacy of the myotomy and to identify any mucosal injury. Completeness of the myotomy is confirmed by comparing laparoscopic and endoscopic views. Before constructing the fundoplication, the hiatus is inspected and reapproximated posteriorly with interrupted permanent sutures. The hiatal closure must not impinge on or angulate the esophagus. A 50 French bougie is then passed perorally into the stomach and remains in place during creation of the fundoplication. The leading edge of the lateral aspect of the fundus is then passed through the retroesophageal space. When mobilized properly, this portion of the fundus should sit comfortably to the right of the esophagus. The posterior aspect of the fundus is secured to the right crus. The leading edge of the wrapped fundus is then sutured to the right side of the myotomy over a length of 3 cm using interrupted 2–0 braided polyester suture. Similarly, the anterior fundus is secured to the left side of the myotomy over a length of 3–4 cm. The most cephalad sutures on each side also incorporate the crura. It is important to prevent redundancy in the fundoplication and to avoid snaring the anterior vagus nerve during suturing. A completed Heller–Toupet fundoplication is shown (Fig. 4).

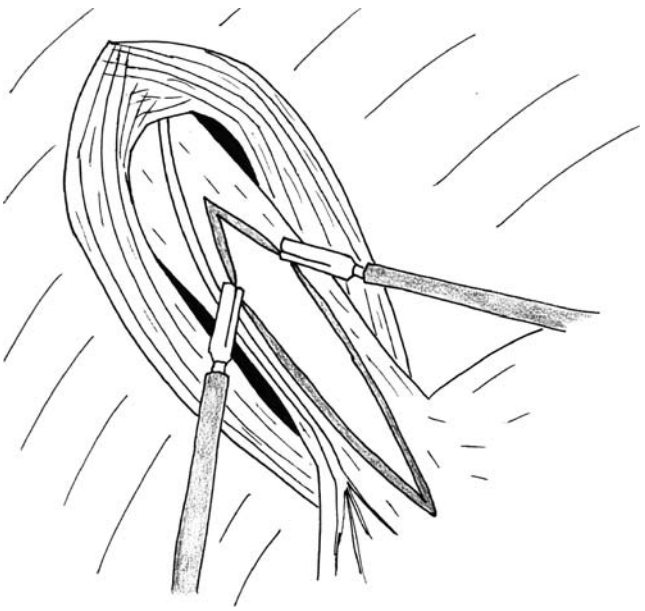

Figure 3 Laparoscopic Heller Myotomy—The edges of the myotomy are grasped and separated. Both longitudinal and circular muscle layers are divided revealing the submucosa. The anterior vagus nerve is seen coursing across the esophagus and not included in the myotomy. The myotomy is extended below the GE junction.

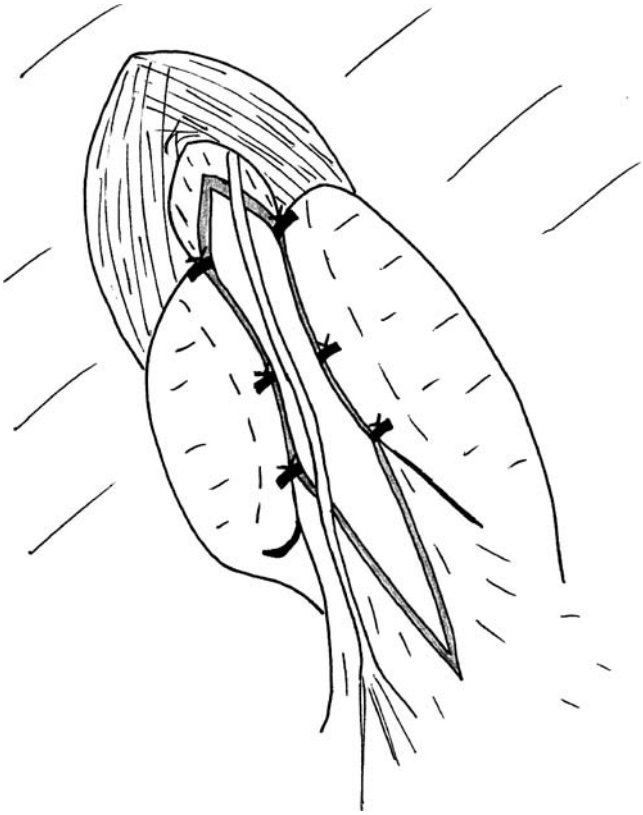

Figure 4 Completed Heller–Toupet Fundoplication—The superior sutures in the partial fundoplication incorporate the crus, myotomy, and stomach. The vagus nerve is seen lying anteriorly and not included in the fundoplication. The fundoplication is positioned above the GE junction.

When the posteriorly wrapped fundus appears to result in excessive anterior esophageal angulation, and in the rare instance of esophageal perforation during myotomy, we perform a Dor fundoplication instead of the Toupet. If a Dor fundoplication is planned preoperatively, the posterior esophageal dissection is unnecessary. The fundus is mobilized and the myotomy is performed as described above. The leading edge of the lateral aspect of the fundus is pulled anteriorly to the right side of the hiatus, effectively covering the myotomy. An inner row of interrupted sutures is placed to fix the inner medial aspect of the fundus to the left side of the myotomy over a 3-cm length as it is being folded over anteriorly. The leading edge of the fundus is then sutured to the right side of the myotomy over 4 cm. The fundus is also sutured to the left and right crura.

Once the myotomy and partial fundoplication is completed, the bougie is removed. The liver retractor is

removed under direct laparoscopic visualization, and hemostasis is assured. All laparoscopic ports are removed under direct vision and the pneumoperitoneum is released. The fascia of the camera port can be reapproximated in interrupted fashion, taking care not to incorporate any intraperitoneal structures in the closure. The fascia of the 11-mm subxiphoid port (surgeon's left hand) does not need to be reapproximated as this incision will commonly migrate above the costal margin when the pneumoperitoneum is released. All skin incisions are closed in subcuticular fashion.

Pitfalls

Previous Surgery or Endoscopic Treatments

Patients with previous upper abdominal or hiatal surgery should be approached with caution. As in any reoperative field, the risk of organ injury, bleeding, and poor outcomes are increased. Access to the peritoneal cavity can be performed using the Verress needle in an area away from the previous operative site, or an open Hasson approach can be used. The left lateral segment of the liver may be densely adherent to the distal esophagus and proximal stomach. Care must be taken to avoid excessive bleeding while mobilizing the liver. The risk of esophageal and gastric perforation is increased, as is the rate of conversion to an open procedure. These risks must be taken into consideration and select patients may benefit from a transthoracic approach. Many patients have undergone endoscopic treatments for achalasia before seeking surgical myotomy. Controversy exists as to whether previous endoscopic treatments increase operative complications and poor outcomes.[12, 13] It has been our experience that prior Botox treatment leads to a more difficult myotomy with longer operative times but otherwise equivalent outcomes to the untreated patient.[10]

Adequacy of Myotomy

The purpose of surgical myotomy is to disrupt the LES fibers and relieve symptoms of dysphagia. Adequacy of the myotomy has been a point of controversy. When the myotomy was performed via a transthoracic approach, it was extended just across the GE junction.[8] With the advent of laparoscopy, most surgeons extend the myotomy onto the gastric wall for 1–2 cm. A recent study advocates that an extended myotomy (>3 cm) provides superior symptomatic relief of dysphagia when compared to a standard myotomy of 2 cm.[9] Unfortunately, this study had sequential patient accrual and compared standard myotomy with Dor fundoplication to extended myotomy with Toupet fundoplication; it is unclear whether long-term symptomatic relief is caused by the myotomy or improved reflux protection. Until more definitive studies are performed, we believe the myotomy should extend onto the gastric wall for at least 2 cm and intraoperative endoscopy should be used to gauge its adequacy.

Esophageal Perforation

Hiatal dissection and mobilization of the mediastinal esophagus can result in an esophageal or gastric injury. At no time should the esophagus be grasped directly. A careful and meticulous dissection should be performed in all patients especially the elderly, immunosuppressed, and reoperative patients. If recognized, gastric perforation or serosal tears can generally be easily repaired at the time of surgery. Esophageal perforations can be handled in a similar fashion by suturing with fine absorbable sutures. If an anterior esophageal injury is created at the time of myotomy, the surgeon can elect to buttress the repair with a Dor fundoplication as opposed to a Toupet. The true danger lies in unrecognized injuries. Unrecognized injury can result in peritonitis and/or mediastinitis and may require diversion and gastrostomy tube placement or esophagectomy. For this reason, some surgeons advocate the routine use of a Dor fundoplication to cover the myotomy, and this approach obviates the need for the posterior esophageal dissection. Despite this theoretical advantage, the Heller–Toupet operation has been shown to be safe and effective in experienced hands.[9, 10, 14]

Postoperative Management

A nasogastric tube is not used routinely, and patients are hospitalized overnight. A clear liquid diet is started on the afternoon of the operation and a soft diet the morning after. Adequate pain control is achieved using oral (liquid) analgesics. Intravenous anti-emetic medications are given as needed to prevent retching and vomiting to avoid stress on the newly created fundoplication. An esophagram is not routinely performed, and obtained only if there is clinical suspicion of a perforation, or to evaluate for herniation and disruption if the patient retches or vomits. Patients are discharged home on the first postoperative day on a diet of soft foods for 2–4 weeks. Bread, bread products, hard fruits and vegetables, and coarse meats should be avoided for this time period. Patients are seen on an outpatient basis at 2–4 weeks, 6 months, and yearly thereafter. We generally perform a timed barium swallow to evaluate gastric emptying and a 24-hour pH test to assess for silent GE reflux at 6–12 months postoperatively.

Conclusion

Surgical myotomy is the most effective and durable treatment for achalasia. The use of laparoscopy has decreased operative morbidity, which has led to the laparoscopic Heller myotomy becoming the first line treatment.[15] A partial fundoplication should be performed in conjunction with the myotomy to minimize postoperative GE reflux, a harmful occurrence in an esophagus with poor clearance capabilities. The ideal fundoplication awaits the performance of prospective randomized trials. We have found the combined laparoscopic Heller–Toupet procedure to be a safe and effective treatment of achalasia.

References

1. Lendrum F. Anatomic features of the cardiac orifice of the stomach with special reference to cardiospasm. Arch Intern Med 1937;59:474–476.
2. Vaezi MF, et al. Botulinum toxin versus pneumatic dilatation in the treatment of achalasia: a randomised trial. Gut 1999;44(2):231–239.
3. Zaninotto G, et al. Randomized controlled trial of botulinum toxin versus laparoscopic heller myotomy for esophageal achalasia (see comment). Ann Surg 2004;239(3):364–370.
4. Csendes A, et al. Late results of a prospective randomised study comparing forceful dilatation and oesophagomyotomy in patients with achalasia. Gut 1989;30(3):299–304.
5. Burpee SE, et al. Objective analysis of gastroesophageal reflux after laparoscopic heller myotomy: an anti-reflux procedure is required. Surg Endosc 2005;19(1):9–14.
6. Richards WO, et al. Heller myotomy versus Heller myotomy with Dor fundoplication for achalasia: a prospective randomized double-blind clinical trial.[see comment]. Ann Surg 2004;240 (3):405–412. discussion 412–5.
7. Bessell JR, et al. Laparoscopic cardiomyotomy for achalasia: long-term outcomes. AN Z J Surg 2006;76(7):558–562.
8. Csendes A, et al. Very late results of esophagomyotomy for patients with achalasia: clinical, endoscopic, histologic, manometric, and acid reflux studies in 67 patients for a mean follow-up of 190 months. Ann Surg 2006;243(2):196–203.
9. Jeansonne LO, et al. Ten-year follow-up of laparoscopic Heller myotomy for achalasia shows durability. Surg Endosc 2007;21 (9):1498–1502.
10. Perrone JM, et al. Results of laparoscopic Heller–Toupet operation for achalasia. Surg Endosc 2004;18(11):1565–1571.
11. Wright AS, et al. Long-term outcomes confirm the superior efficacy of extended Heller myotomy with Toupet fundoplication for achalasia. Surg Endosc 2007;21(5):713–718.
12. Deb S, et al. Laparoscopic esophageal myotomy for achalasia: factors affecting functional results. Ann Thorac Surg 2005;80 (4):1191–1194. discussion 1194–5.
13. Smith CD, et al. Endoscopic therapy for achalasia before Heller myotomy results in worse outcomes than heller myotomy alone. Ann Surg 2006;243(5):579–584. discussion 584–6.
14. Hunter JG, et al. Laparoscopic Heller myotomy and fundoplication for achalasia. Ann Surg 1997;225(6):655–664. discussion 664–5.
15. Spiess AE, Kahrilas PJ. Treating achalasia: from whalebone to laparoscope. JAMA 1998;280(7):638–642.