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ORIGINAL ARTICLE

Revisional Surgery in Patients with Recurrent Dysphagia after Heller Myotomy

Kaylee E. Smith, BS[1] · Adham R. Saad, MD[1,2] · John P. Hanna, MD[3] · Thanh Tran, MPH[3] · John Jacobs, MD[4,2] · Joel E. Richter, MD[4,2] · Vic Velanovich, MD[1,2]

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Abstract

Background Recurrent/persistent symptoms of achalasia occur in 10–20% of individuals after Heller myotomy. The causes and treatment outcomes are ambiguous. Our aim is to assess the causes and outcomes of a multidisciplinary approach to this patient population.

Methods All patients undergoing revisional operations after a Heller myotomy were reviewed retrospectively. Data collected: demographics, date of initial Heller myotomy, preoperative evaluation, etiology of recurrent symptoms, date of revisional operation, and surgical outcomes.

Results A total of 34 patients underwent 37 revisional operations. Operations were tailored based on preoperative multidisciplinary evaluation. Causes of symptoms: periesophageal/perihiatal fibrosis 11 (27%), obstructing fundoplication 11 (27%), incomplete myotomy 8 (20%), progression of disease 9 (22%), and epiphrenic diverticulum 1 (2%). Operations performed: reversal/no creation of fundoplication with or without re-do myotomy 22 (59%), revision/creation of fundoplication with or without myotomy 6 (16%), and esophagectomy 9 (24%). Ten patients in the 37 operations (27%) developed postoperative complications. Of 33 patients for 36 operations with follow-up, 25 patient-operations (69%) resulted in resolution or improved dysphagia. Although there was variation in symptomatic improvement by cause and operation type, none reached statistical significance.

Conclusion There are several causes of dysphagia after Heller myotomy and a thoughtful evaluation is required. Complication rates are higher than first-time operations. Symptomatic improvement occurs in the majority of cases, but a significant minority will have persistent dysphagia. Although an individualized approach to dysphagia after Heller myotomy may improve symptoms and passage of food, the perception of dysphagia may persist in patients.

Keywords Achalasia · Heller myotomy · Dor fundoplication · Esophagectomy · Dysphagia

Introduction

Achalasia is a primary motility disorder of the esophagus characterized by lack of esophageal peristalsis and failure of the lower esophageal sphincter (LES) to relax. Patients commonly present with symptoms of dysphagia to solids and liquids, regurgitation, weight loss, aspiration, or heartburn.[1] Symptoms are generally nonspecific and can often lead to misdiagnosis or delay in diagnosis.[2] Although the exact etiology is unknown, the pathophysiology is due to loss of inhibitory neurons within the myenteric plexus of the esophageal body.[3] Achalasia is a relatively rare disease, with an incidence of 1 in 100,000.[4]

As there is no cure for achalasia, treatment is focused on symptom alleviation. It is assumed that the etiology of

✉ Vic Velanovich, MD
vvelanov@health.usf.edu

[1] Division of General Surgery, Department of Surgery, University of South Florida, 5 Tampa General Circle, Suite 740, Tampa, FL 33606, USA

[2] The Joy McCann Culverhouse Center for Swallowing Disorders, University of South Florida, Tampa, FL, USA

[3] Division of Surgical Research, Department of Surgery, University of South Florida, Tampa, FL, USA

[4] Division of Gastroenterology, Department of Medicine, University of South Florida, Tampa, FL, USA

achalasia symptoms is due to a relative outflow obstruction at the gastroesophageal junction from failure of the lower esophageal sphincter to relax and lack of esophageal peristalsis. Curiously, however, there does not seem to be a good correlation between objective, physiologic assessment of achalasia, and symptom severity.[5] This being said, treatments have been directed at reducing the resistance of food and liquid passing through the gastroesophageal junction, including dietary manipulation, medications (calcium channel blockers or nitrates), endoscopic treatments (pneumatic dilation, Botox injections), and surgical interventions (Heller myotomy, per-oral endoscopic myotomy (POEM), esophagectomy). It is important to note that there are no treatments designed to improve esophageal peristalsis. Due to the progressive nature of the disease and transient effect of pharmacologic therapies, the effects of noninvasive therapeutic modalities achieve only short-term relief,[6] often necessitating surgical or endoscopic procedures. Laparoscopic Heller myotomy is the most commonly performed surgical procedure, which involves at least a 6-cm anterior myotomy of the longitudinal and circular muscle layers of the distal esophagus and extending at least 2 to 3 cm onto the gastric cardia, with the primary aim to reduce esophagogastric outflow resistance. This has led to overall improvement in symptoms and quality of life.[7] A partial fundoplication is usually added to mitigate gastroesophageal reflux. An anterior 180° Dor or posterior 270° Toupet fundoplications are the most common options used.[6,7]

Although there is significant symptomatic relief achieved with initial treatment, up to 10–20% of patients experience some continuation of dysphagia symptoms after initial surgery.[8,9] The cause of early recurrence of symptoms after Heller myotomy has been thought to be an incomplete myotomy. The causes of recurrent symptoms after a presumably complete myotomy have not been clear. Possible causes of late symptom recurrence include scarring or cicatrix at the site of the myotomy or esophageal hiatus; failure or distortion of the fundoplication, including a herniated fundoplication; acid reflux with esophagitis/stricture; esophageal stasis due to aperistalsis, dilation, tortuosity, or "sigmoidization" of the esophagus, as well as late presentation of an incomplete myotomy. It is important to note that except for failure of esophageal clearance due to aperistalsis or the shape of the esophagus, the causes are presumed to be related to resistance of esophagogastric outflow. Additionally, despite appropriate surgical and non-surgical treatment, about 10–15% of patients will progress to end-stage achalasia, defined by dilated, tortuous, or megaesophagus, with about 5% of patients eventually requiring an esophagectomy.[10] Although repeat Heller myotomy has been considered a safe approach,[11] data on its success is limited.[10–13] The aim of this study is to examine causes of recurrent symptoms after Heller myotomy and outcomes of a tailored approach for revisional surgery.

Methods

This study has been approved by the Institutional Review Boards of the University of South Florida and Tampa General Hospital. We identified all patients who underwent revisional surgery for persistent symptoms after a Heller myotomy for achalasia between May 2012 and January 2019, inclusive. Inclusion criteria included age > 18, a preoperative diagnosis of achalasia, and persistent or recurrent symptoms after initial Heller myotomy. Other interventions for symptoms of dysphagia, such as endoscopic dilation or botulinum toxin injections, were also included.

A total of 34 patients who underwent 37 revisional operations after Heller myotomy were included. Patients were evaluated by members of the University of South Florida Joy McCann Culverhouse Center for Swallowing Disorders, consisting of gastroenterologists, surgeons, and speech pathologists. Every effort was made to obtain prior records, including operative reports. However, many of the prior operations were done many years or decades ago at other institutions; therefore, we frequently did not have these records. After an initial history and physical examination, timed barium esophagogram (TBE),[14,15] high-resolution esophageal manometry (HRM) and upper gastrointestinal endoscopy were performed as deemed necessary. With respect to TBE, emptying at 1 min, 5 min, and 10 min was evaluated, as well as passage of a 13-mm barium tablet at 10 min. Note was made of esophageal dilation and shape, as well as location, shape, and narrowing at the gastroesophageal junction. With respect to HRM, we collected data on the type of motility disorder, esophageal body peristalsis and pressurization, lower esophageal sphincter pressure (LESP) and integrative relaxation pressures (IRP). On a selective basis, endoscopic ultrasound examination was used to assess for pseudo-achalasia.

Preoperative Determination of the Cause of Recurrent Symptoms Each patient was presented and discussed at our interdisciplinary swallowing disorders conference. Clinical and objective data were discussed. A preliminary determination as to the cause of the recurrent symptoms was made based on the criteria defined in Table 1. If a patient had persistent symptoms after the Heller myotomy with no interval improvement, the integrative relaxation pressure (IRP) was elevated > 15 mmHg (Fig. 1a), timed barium esophagogram demonstrated a bird's beak deformity of the gastroesophageal junction with failure of a 13-mm barium table to pass (Fig. 1b), and/or upper endoscopy demonstrated "puckering" of the gastroesophageal junction without signs of mucosal injury, then the dysphagia was attributed to an incomplete myotomy. If there were several months to years when recurrent dysphagia occurred after the initial myotomy, IRP was < 15 mmHg, there was a narrowing at the gastroesophageal junction with failure of the barium tablet to pass, and/or there was a narrowing

Table 1 Criteria determining cause of persistent/recurrent dysphagia after Heller myotomy with or without partial fundoplication

Cause	Time from index Heller myotomy	Esophageal manometry	Time barium esophagogram	Upper gastrointestinal endoscopy
Incomplete myotomy	Immediate to weeks	IRP > 15 mmHg	"Bird's beak," Failure of 13-mm barium tablet to pass	"Puckering" at GE junction, Normal esophageal mucosa
Periesophageal/hiatal scarring/cicatrix	Months to years	IRP < 15 mmHg	Narrowing at GE junction, Failure of 13-mm barium tablet to pass	"Fishmouth" appearance of GE junction, Normal esophageal mucosa
Acid reflux–induced stricture	Months to years	IRP < 15 mmHg or unable to assess	Narrowing at GE junction, Failure of 13-mm barium tablet to pass	Stricture with esophagitis/ulceration in distal esophagus
Obstructing fundoplication	Immediate to years	IRP < 15 mmHg or unable to assess	Narrowing at GE junction, Failure of 13-mm barium table to pass at level of fundoplication	Extrinsic narrowing at GE junction with normal esophageal mucosa, possible visualization of "fishmouth" GE junction, "Tight" or "abnormal" fundoplication on retroflexion
Functional Dysphagia	Immediate to years	IRP < 15 mmHg	Normal TBS emptying, 13-mm barium tablet passes	"Fishmouth" appearance of GE junction, normal esophageal mucosa
End-stage achalasia	Years to decades	IRP < 15 mmHg or unable to assess	Dilated, tortuous esophagus with or without narrowing at GE junction	Dilated, tortuous esophagus with or without retained food

associated with a "fishmouth" appearance of the gastroesophageal junction which describes the ovoid endoscopic appearance of the gastroesophageal junction after a complete Heller myotomy (Fig. 2), but no mucosal inflammation, then the dysphagia was attributed to periesophageal/hiatal scarring/cicatrix. If there were several months to years after the initial myotomy, IRP was < 15 mmHg, there was narrowing at the gastroesophageal junction with failure of the barium tablet to pass, and/or there was stricture associated with esophagitis, then the dysphagia was attributed to acid-reflux-induced stricture. If the symptoms occurred immediately to months/years after the myotomy, IRP < 15 mmHg (Fig. 3a), there was narrowing at the gastroesophageal junction and failure of the barium tablet to pass at the level of the fundoplication (Fig. 3b), and/or the gastroesophageal junction had a fishmouth appearance with no mucosal inflammation, then it was determined that the cause was related to the fundoplication. This category includes fundoplications which were too tight, as well as those which may have herniated through the hiatus. If at any time after the myotomy, the IRB < 15 mmHg, there was good emptying of contrast by timed barium esophagogram and passage of the barium tablet, and/or the upper endoscopy showed an open, fishmouth gastroesophageal junction, then the dysphagia was attributed to aperistalsis

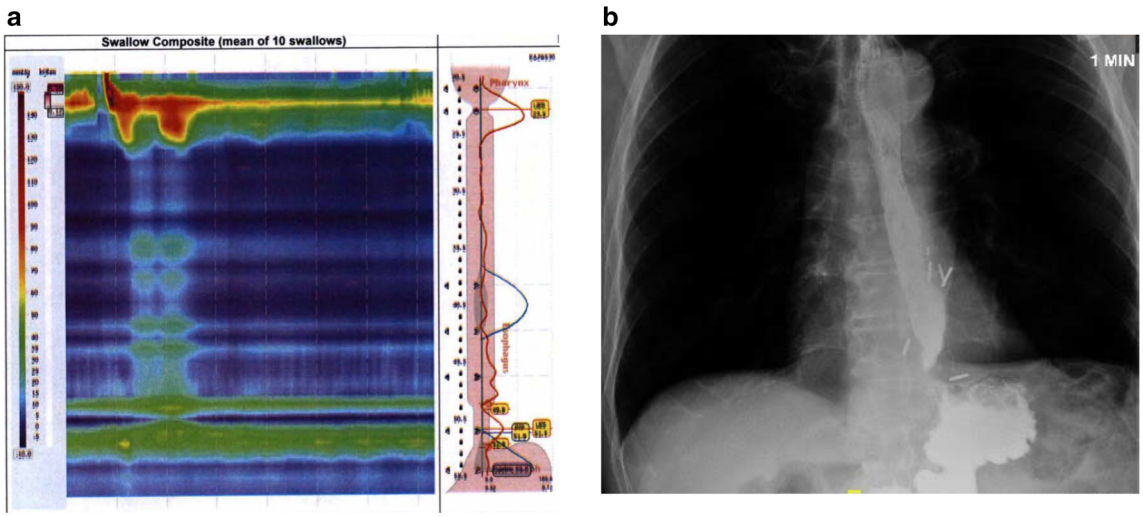

Fig. 1 **a** High-resolution manometry of the patient, showing no relaxation of the lower esophageal sphincter, pan-pressurization, and an IRP of 26, diagnostic of type II achalasia. **b** Timed barium esophagogram of patient who underwent a transthoracic Heller myotomy showing poor emptying at 1 min and "bird's beaking" of the distal esophagus suggestive of an incomplete myotomy

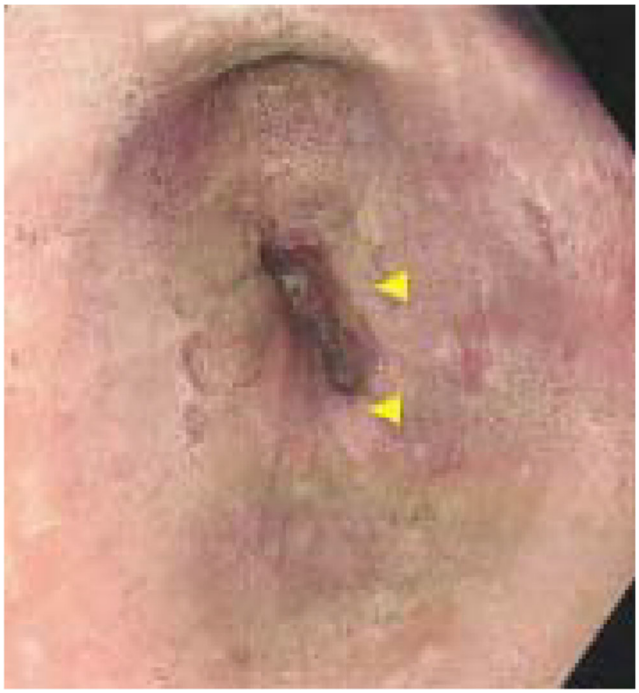

Fig. 2 Endoscopic appearance of the gastroesophageal junction after a complete myotomy. We have described this as a "fishmouth" appearance

or a functional, non-anatomic cause. Lastly, if there was severe dilation or tortuosity of the esophagus after many years of disease, usually after several endoscopic and operative procedures (Fig. 4), then it was determined that the cause of the dysphagia was progression of disease to end-stage achalasia.

Choice of Operation Based on Cause (Table 2) If the cause was determined to be an incomplete myotomy, then primary treatment options included pneumatic dilation, botulinum toxin

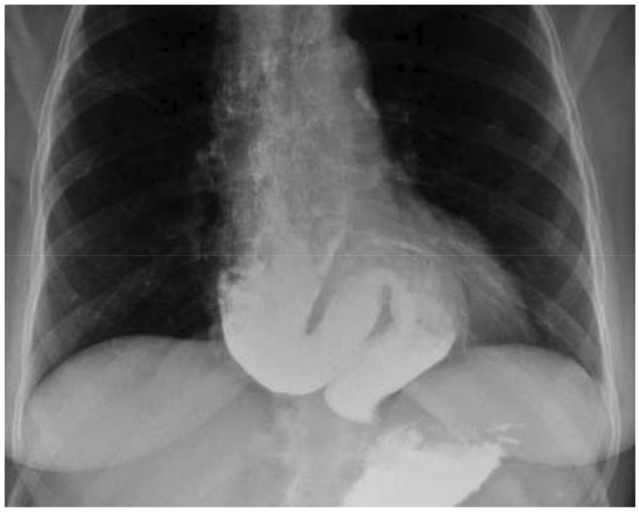

Fig. 4 Timed barium esophagogram of a patient with end-stage achalasia showing severe tortuosity of the distal esophagus in a "sink drain" pattern

intramuscular injection, re-do laparoscopic or open Heller myotomy with or without Dor fundoplication, or a POEM. If endoscopic therapies failed, then a re-do Heller myotomy or POEM was offered. If the cause was determined to be periesophageal/hiatal scarring, then primary therapy would be pneumatic dilation; which if failed, a laparoscopic or open lysis of adhesions with re-do Heller myotomy and reversal of the existing fundoplication was offered. If the cause was determined to be the obstructing fundoplication, then primary therapy would be pneumatic dilation; which if failed, a laparoscopic reversal of the fundoplication with or without a re-do Heller myotomy was offered. If the cause was determined to be a reflux-related stricture, then dilation with acid-reducing medication was offered (this will not be discussed in this

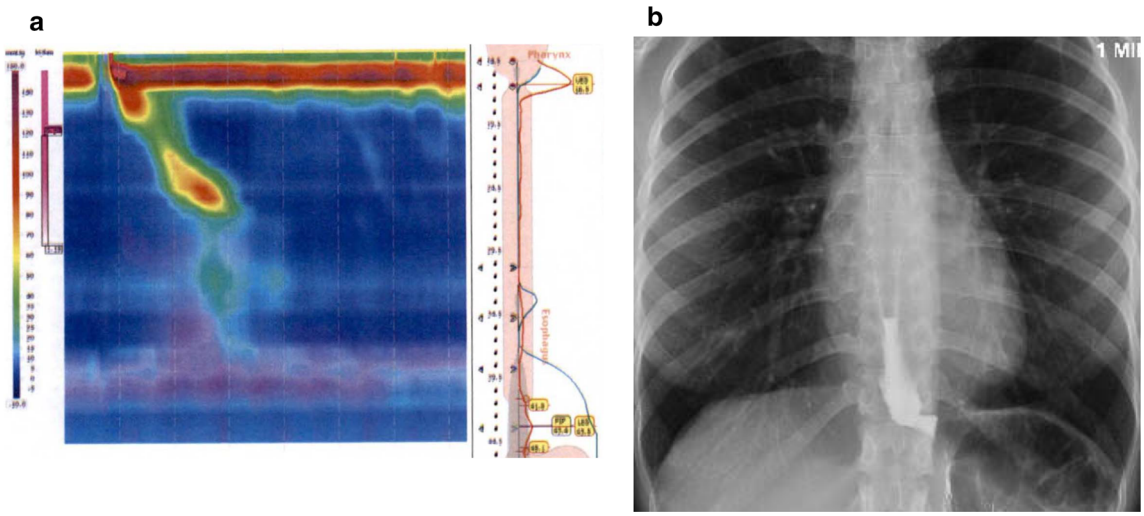

Fig. 3 **a** High-resolution manometry of the patient in Fig. 2, showing some return of peristalsis and relaxation of the lower esophageal sphincter, with an IRP of 7 confirming a complete myotomy. And, therefore, obstruction by the fundoplication as the cause of her recurrent symptoms. **b** Timed barium esophagogram of a patient with recurrence dysphagia, showing an abrupt cut-off of contrast at the level of the Dor fundoplication (arrow)

Table 2 Therapeutic approaches based on cause

Cause	Primary treatment	Secondary treatment
Incomplete myotomy	Pneumatic dilation, Botox, POEM, re-do Heller myotomy	POEM, re-Do Heller myotomy with/without reversal fundoplication
Periesophageal/hiatal scarring/cicatrix	Dilation (savory, balloon, pneumatic)	Adhesiolysis with/without re-do Heller myotomy and reversal fundoplication
Acid reflux induced stricture	Dilation (savory or balloon) with acid-reducing medication	Revision of fundoplication
Obstructing fundoplication	Dilation (savory or balloon)	Reversal of fundoplication with/without re-do Heller myotomy
Functional dysphagia	Reassurance	Promotility medication
End-stage achalasia	Conservative management with/without dilation	Esophageal resection based on esophageal morphology

study). If a functional cause was determined, then patient reassurance was the primary therapy, with consideration of a promotility agent if the patient insisted on some type of therapy (this will not be discussed in this study). If the cause was determined to be end-stage achalasia, then a trans-abdominal or Ivor Lewis esophagectomy was selected with the extent of the resection based on the morphology of the esophagus with the aim to be both straightening the esophagus and providing a wide anastomosis to minimize esophagogastric outflow resistance (Table 2).

It should be emphasized, however, that often more than one cause may be responsible for the patient's symptoms. This is particularly true for periesophageal/perihiatal scarring/fibrosis in patients with IRP < 15. Most patients will have some postoperative scarring. In these cases, as it may be difficult to pinpoint a single, specific cause, adhesiolysis, fundoplication reversal, and re-do myotomy may all be done to insure the best chance for symptomatic improvement.

Data The data collected included demographics, date of initial Heller myotomy, date of revisional operation, pretreatment pre-revisional imaging and manometry, pretreatment and pre-revisional symptoms, postoperative complications, and etiology and description of recurrent symptoms.

Objective Symptomatic Response An attempt was made to contact patients to objectively assess their symptomatic response to revisional surgery using the Achalasia Symptom Questionnaire (ASQ).[16] This is a validated instrument assessing the symptoms of achalasia. The best possible score is 10, the worst possible score is 31.

Statistical Analysis Demographics and data measurements were summarized using descriptive statistics. Comparisons of categorical data were performed using chi-square and Fisher exact tests. The paired preoperative and postoperative ASQ scores were analyzed using the paired Student's *t* test. A *p* value of ≤ 0.05 was considered statistically significant.

Results

Demographics Of the 34 patients with recurrent or persistent dysphagia symptoms after Heller myotomy, 37 revisional surgeries were performed. Three patient underwent 2 revisional operations. During this same period of time, our group did 318 Heller myotomies and 56 POEM's. In comparison, of the 244 pneumatic dilations performed in the same timeframe, 32 were for patients with dysphagia after a Heller myotomy. Because most patients requiring revision had their initial operation at other institutions, it is impossible to determine what the rate of failure was. The mean age was 52.4 ± 15.5 years (range, 22–77 years), with 59% being female. The median time from initial Heller myotomy to revisional operation was 59 months. Of these patients, 8 had no relief of their symptoms after the initial operation. Prior to revisional surgery, 9 patients had no additional interventions; however, there were 11 pneumatic dilations, 10 balloon dilations, 6 bougie dilations, and 6 botulinum toxin injections. Five patients had two or more types of interventions. The median time from revisional operation to recurrent dysphagia of 4 months, and median time from revisional operation to last follow-up of 1.5 months (Table 3).

Table 3 Patient characteristics ($n = 34$)

Demographics	*n*	
Age (years) Mean ± SD (range)	34	52.4 ± 15.5 (22–77)
Gender (%): Male Female	14 20	41% 59%
Time to redo (in months)	37	59
Time from re-do to recurrent dysphagia (in months)	12	4
Time to last follow-up (in months)	33	1.5

Symptoms and Causes The distribution of primary pre-revisional recurrent symptoms was dysphagia 31 (84%), regurgitation 5 (14%), vomiting 3 (8%), and heartburn 2 (5%), including 4 patients with more than one cause. The determined causes of recurrent symptoms included: 11 (27%) patients with periesophageal/perihiatal scar/cicatrix, 11 (27%) with obstructive fundoplication, 8 (20%) with incomplete myotomy, 9 (22%) with progression of disease to end-stage achalasia, and 1 (2%) with an epiphrenic diverticulum, including 4 patients were determined to have more than one cause.

Revisional Operations Performed Revisional operations performed were based on determined etiology of symptoms and can be group into three basic types: (1) Reversal/no creation of fundoplication with or without additional myotomy (to completely eliminate a fundoplication as a cause of esophagogastric outflow resistance), including reversal Dor/Nissen/Toupet fundoplication with or without re-do Heller myotomy (15 total, 12 laparoscopic, including 3 reversal of fundoplication without re-do myotomy), re-do Heller myotomy without fundoplication (5 total, 4 laparoscopic), POEM (2), for a total 22 (59%) operations. (2) Revision/creation of Dor fundoplication with or without re-do Heller myotomy: laparoscopic re-do Heller myotomy with Dor fundoplication (2), revision of fundoplication (4 total, 3 laparoscopic), for a total of 6 (16%) of operations. (3) Esophagectomy: trans-abdominal (7), Ivor Lewis (2), for a total of 9 (24%) of operations. In addition, there were 3 pyloroplasties and 2 tube gastrostomies.

Perioperative Complications Ten patients (10 patients with 37 operations, 27%) developed postoperative complications. Seven experienced major complications (i.e., septic shock secondary to esophageal perforation, mediastinitis, pneumonia, incarcerated umbilical hernia, ventral hernia, postoperative leak, and pleural effusion). One of these patients with advanced chronic obstructive lung disease could not be weaned from the ventilator and it was determined by our pulmonary service that he may be ventilator-dependent indefinitely. With this information, the patient chose comfort measures only and was terminally extubated on postoperative day 24. Three patients experienced minor complications (i.e., *Clostridium difficile* colitis and subclinical leak).

Symptom Change Excluding the one patient who died, 33 patients undergoing 36 operations were available for symptomatic follow-up. Of these, 25 patient-operations (69%) had resolution/improvement of symptoms, while 11 (31%) did not. Table 4 presents the rates of symptomatic improvement by cause and operation type. Note that 4 patients had more than one cause; therefore, although there were 36 operations, there were 40 patient-causes. There was no statistically significant difference between these categories. The average amount of time from initial operation to redo operation was 75 months. Although there was no statistical difference, individuals ($n = 26/36$ who had available data for time to redo and resolution of dysphagia reported) who eventually achieved relief of dysphagia had a longer interval of symptomatic relief prior to revision (128 months) versus patients who had no symptomatic improvement after revisional surgery (55 months). In patients who had recurrent dysphagia after revisional surgery, the average time to representation was 4 months (Table 3). We were able to contact 7 patients to administer the ASQ, with 6 of 7 patients reporting improvement in symptoms. There remainder either could be found or did not return our calls. The mean and standard deviation preoperative ASQ score was 21.9 ± 4.7 which improved postoperatively to 15.9 ± 5.9 ($p = 0.03$).

Discussion

Although initial Heller myotomy achieves symptomatic relief in a great number of patients with achalasia, upwards of 20% experience persistent or recurrent symptoms despite intervention. In this subset of patients, there is no consensus on the cause and therapeutic approach. There are also multiple opinions as to the treatment of patients with recurrent or persistent symptoms after Heller myotomy.[17] In addition, there are potentially a wide variety of causes for these symptoms.[17,18] Options for management include dietary and lifestyle modification; bougie, balloon, or pneumatic dilation; botulinum toxin injection; and revisional surgery. Because of the myriad of causes and variety of treatment options, a "one size fits all" approach is unwise. It should be remembered that the rationale of the cause of dysphagia in achalasia and after treatment is persistent resistance to esophagogastric outflow. As no treatment at present improve esophageal peristalsis, it is unclear to what extent peristalsis affects esophageal clearance and symptom perception.

We have also found that a variety of causes can lead to dysphagia after Heller myotomy. These include periesophageal/perihiatal fibrosis/scar, incomplete myotomy, obstructing fundoplication, progression of disease, as well as an epiphrenic diverticulum (Tables 1 and 4). Others have also found a range of similar causes.[17–21] Our group is a multidisciplinary team of gastroenterologists, surgeons, and speech pathologists. We are then able to determine, with reasonable degree of certainty, the cause (Table 1) and chose our treatment accordingly (Table 2). However, it should be emphasized that there may be overlapping causes, for example, periesophageal/perihiatal scarring may occur with other causes and it may be difficult to discern the proportion of the symptoms are due to one cause or another. We had 4 patients that we could contribute multiple causes, such as scarring, incomplete myotomy, and obstructing

Table 4 Number and frequency of patient-causes with symptomatic improvement by cause and types of surgery

Resolution of dysphagia ($n = 40$)*	Re-do myotomy without fundoplication**	Revision/creation fundoplication with or without re-do myotomy+	Esophagectomy++
Periesophageal/perihiatal Scarring/Cicatrix	6 of 11 (54%)		
Obstructing fundoplication	4 of 7 (57%)	2 of 4 (50%)	
Incomplete myotomy	4 of 6 (67%)	1 of 2 (50%)	
Progression of disease			7 of 9 (78%)
Epiphrenic diverticulum	1 of 1 (100%)		

*Excludes postoperative death patient and 4 patients with multiple causes

**Includes laparoscopic take down Dor/Nissen/Toupet fundoplication with re-do Heller myotomy, POEM, laparoscopic reversal Dor fundoplication, open re-do Heller myotomy, laparoscopic re-do Heller myotomy

+ Includes laparoscopic re-do Heller myotomy with Dor fundoplication, open re-do Dor fundoplication, laparoscopic revision Dor fundoplication

++ Includes transabdominal esophagectomy, Ivor Lewis esophagectomy

fundoplication. We do lyse adhesions as necessary to achieve an operative field to complete a re-do myotomy and/or revise/reverse a fundoplication, so we cannot be sure how much of the patient's symptomatic relief was due to the adhesiolysis as opposed to the other aspects of the operation. We will sometimes use endoscopic ultrasound and/or computed tomographic scanning to better determine anatomically features, but for the most part, operative exploration is both diagnostic and therapeutic.

For the most part, non-operative approaches are the first choice (Table 2). Pneumatic dilation is a reasonable first choice, and the overall efficacy is 50 to 60%.[22] If these non-operative approaches fail, revisional surgery should be considered. Revisional surgery has tended to focus on extending the myotomy and reversing or revising the fundoplication,[10–13,18–21,23] or esophagectomy.[24,25] Newer approaches, such as POEM, have also shown efficacy in selected patients.[26,27] In fact, a POEM would be the ideal revisional operation in a patient with an incomplete myotomy as the sole cause. Difficulty arises when it is unclear whether that is the sole cause or whether an obstructing fundoplication or periesophageal fibrosis may also contribute. In these more opaque cases, laparoscopic intervention is the surest way of achieving a reduction in esophagogastric outflow resistance. Another approach of performing a POEM first and operating only if there is no symptomatic improvement is an option; however, in our experience, most patients have opted for one procedure which would give them the best chance for symptomatic relieve with just one procedure. An innovative technique of stapled cardioplasty to achieve a wide gastroesophageal junction without esophagectomy has great theoretical appeal.[28] Our rate of improved symptoms is similar to the published literature cited. As an objective measure of symptomatic improvement, we prefer the ASQ as it has been more rigorously validated, and, we feel that the weight loss item in the Eckardt score lacks face validity for symptomatic improvement.[29] However, it should be noted that there is poor correlation between patient-perceived symptoms of achalasia and physiological measurements of achalasia.[5] However, it should be noted that these are challenging operations, with much higher complication rates than first-time laparoscopic Heller myotomies. Interestingly, the cause of the dysphagia may play a role in symptomatic improvement (Table 4).

Excluding esophagectomy, individuals who most benefitted from redo operation, i.e., those who had complete resolution of dysphagia had a longer symptom-free period between initial operation and reoperation than those who experienced continued dysphagia symptoms after reoperation. This trend suggests that individuals experience increased periods without symptoms after initial surgery may experience a better response from redo operation. Furthermore, in patients who experienced recurrent dysphagia after reoperation, symptoms appeared at 4 months, on average, which tells us people who fail repeat intervention likely do so within the first year. Although mechanical obstruction is still possible, this failure of symptomatic improvement may be related to underlying aperistalsis or patient-perceived symptoms without an anatomic or physiologic explanation. It is our opinion that aperistalsis may not be as important in esophageal clearance as previously thought. Many patients with aperistalsis after Heller myotomy will have no dysphagia whatsoever. On the other hand, there are patients who experience dysphagia, yet so complete clearance of liquids and solid by fluoroscopic esophagography. This once again shows that patient-perceived symptoms may not correlate with our ability to assess esophageal function.[5]

Esophagectomy has been reserved for patients with end-stage achalasia. Esophagectomy in this group has been found to be an overall safe operation in experienced hands.[30] Nine patients in the study had evidence of end-stage achalasia, i.e.,

dilated and tortuous esophagus on barium swallow or aperistalsis with 100% failed swallows on HRM. These patients required resection to both "straighten" the esophagus to allow gravity to promote esophageal clearance and provide a wide esophagogastric anastomosis to minimize resistance to the flow of solid and liquids from the esophagus to the stomach. These 9 patients underwent either trans-abdominal or Ivor Lewis esophagectomy. Choice of operation was based on the morphology of the esophagus. If the esophagus could be straightened, the diseased gastroesophageal junction resection and the esophagus was not massively dilated, then a trans-abdominal approach was used. If the esophagus was massively dilated or could not be straightened sufficiently through the abdomen, then an Ivor Lewis approach was used. Others have also advocated a tailored approach based on esophageal morphology.[17,24]

There are a number of limitations to our study. Firstly, it was conducted at a single institution with a relatively small cohort ($n = 34$). Although, it is one of the larger series of revisional surgery for achalasia over a 6-year period of time; still, due to the small size, we did not find statistical significance in many of the analyses. Increasing the cohort size would likely lead to some statistically significant findings as opposed to trends. Some have advocated using balloon distensibility testing (EndoFlip^{R}, Medtronic, Inc.)[31] but we have not found it particularly useful. Lastly, this was a retrospective study and, although, pre-revisional diagnostic evaluation was often conducted within our hospital network, the majority of patients presented to our institution having received their initial operation and evaluation elsewhere. Because of this, pre-treatment diagnostic manometry and TBE, as well as operative notes, were unavailable for most patients. Thus, we were unable to gather significant data regarding initial disease presentation, treatment, complications, etc., which may have had significant impact on disease recurrence.

Conclusion

In conclusion, a thoughtful, multidisciplinary approach to recurrent or persistent symptoms after Heller myotomy for achalasia is required. This has clearly demonstrated that there are multiple causes for these symptoms and a "one size fits all" approach is inappropriate. Our data suggests that regardless of age, gender, or etiology of presentation, a majority of the patients will achieve symptomatic improvement of dysphagia in achalasia patients who failed initial surgical treatment and underwent revisional surgery. The cause of the symptoms after initial Heller myotomy and the length of time to presentation may be predictors of symptomatic improvement, although these did not reach statistical significance due to sample size. Lastly, there is a subset of patients who will continue to have perceived dysphagia, despite no evidence of esophagogastric outflow obstruction and documented clearance of the masticated food bolus. Further endoscopic or surgical intervention may not help these patients.

Author Contribution Kaylee Smith, Adham Saad, John P. Hanna, Thanh Tran, John Jacobs, Joel E. Richter, and Vic Velanovich all had substantial contributions to the conception, design of the work and data acquisition, analysis, and interpretation of the data for the work; and drafting the work or revising it critically for important intellectual content; and final approval of the version to be published; and agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

References

1. Boeckxstaens GE, Zaninotto G, Richter JE. Achalasia. Lancet. 2014;383(9911):83–93.
2. Eckardt VF, Köhne U, Junginger T, Westermeier T. Risk factors for diagnostic delay in achalasia. Dig Dis Sci 1997;42:580–585.
3. Ates F, Vaezi MF. The pathogenesis and management of achalasia: current status and future directions. Gut Liver 2015;9:449–463.
4. Sadowski DC, Ackah F, Jiang B, Svenson LW. Achalasia: incidence, prevalence and survival. A population-based study. Neurogastroenterol Motil. 2010;22:256–61.
5. Ross D, Richter J, Velanovich V. Health-related quality of life and physiological measurements in achalasia. Dis Esophagus 2017;30: 1–5
6. Moonen A, Boeckxstaens G. Current diagnosis and management of achalasia. J Clin Gastroenterol 2014;48:484–490.
7. Katilius M, Velanovich V. Heller myotomy for achalasia: Quality of life comparison of laparoscopic and open technique. JSLS 2001;5: 227–231
8. Torres-Villalobos G, Coss-Adame E, Furuzawa-Carballeda J, et al. Dor vs Toupet fundoplication after laparoscopic Heller myotomy: Long-term randomized controlled trial evaluated by high-resolution manometry. J Gastrointest Surg 2018;22:13–22.
9. Ramirez M, Patti MG. Changes in the diagnosis and treatment of achalasia. Clin Transl Gastroenterol 2015 May 21;6:e87
10. Petersen RP, Pellegrini CA. Revisional surgery after Heller myotomy for esophageal achalasia. Surg Laparosc Endosc Percutan Tech 2010;20:321–325.
11. Duranceau A, Liberman M, Martin J, Ferraro P. End-stage achalasia. Dis Esophogus 2012; 25:319–30.
12. James DR, Pukayastha S, Aziz O, et al. The feasibility, safety and outcomes of laparoscopic re-operation for achalasia. Minim Invasive Ther Allied Technol. 2012; 21:161–7.
13. Wood TW, Ross SB, Ryan CE, et al. Reoperative Heller myotomy: More pain, less gain. Am Surg 2015;81:637–645
14. de Oliveira JM, Birgisson S, Doinoff C, et al . Timed-barium swallow: a simple technique for evaluating esophageal emptying in patients with achalasia. AJR Am J Roentgenol 1997;169:473–9.
15. Vaezi MF, Baker ME, Richter JE. Assessment of esophageal emptying post-pneumatic dilation: use of the timed-barium esophagram. Am J Gastroenterol 1999;94:1802–7.
16. Urbach DR, Tomlinson GA, Harnish JL, et al. A measure of disease-specific health-related quality of life for achalasia. Am J Gastroenterol 2005;100:1668–1676
17. Patti MC, Allaix ME. Recurrent symptoms after Heller myotomy for achalasia: Evaluation and treatment. World J Surg 2015;39: 1625–1630

18. Zaninotto G, Costantini M, Portale G, et al. Etiology, diagnosis and treatment of failures after laparoscopic Heller myotomy for achalasia. Ann Surg 2002;235:186–192
19. Loviscek MF, Wright AS, Hinojosa MW, et al. Recurrent dysphagia after Heller myotomy: Is esophagectomy always the answer? J Am Coll Surg 2013;216:736–743
20. Iqbal A, Tierney B, Haider M, et al. Laparoscopic re-operation for failed Heller myotomy. Dis Esophagus 2006;19:193–199
21. Veenstra BR, Goldberg RF, Bowers SP, et al. Revisional surgery after failed esophagogastric myotomy for achalasia: Successful esophageal preservation. Surg Endosc 2016;30:1754–1761
22. Saleh CM, Ponds FA, Schijven MP, et al. Efficacy of pneumodilation in achalasia after failed Heller mytomy. Neurogastroenterol Motil 2016;28:1741–1746
23. Mandovra P, Kalikar V, Patel A, Patankarlev. Re-do laparoscopic Heller's cardiomyotomy for recurrent achalasia: Is laparoscoic surgery feasible? J Laparoendosc Adv Surg Tech 2018;28:298–301
24. Watson TJ. Esophagectomy for end-stage achalasia. World J Surg 2015;39:1634–1641
25. Aiolfi A, Asti E, Bonitta G, Bonevina L. Esophagectomy for end-stage achalasia: Systematic review and meta-analysis. World J Surg 2018;42:1469–1476
26. Tyberg A, Sharaiha RZ, Familiari P, et al. Per-oral endoscopic myotomy as salvation technique post-Heller: International experience. Dig Endosc 2018;30:52–56
27. Vigneswaran Y, Yetasook AK, Zhao JC, et al. Per-oral endoscopic myotomy (POEM): Feasible as reoperation following Heller myotomy. J Gastrointest Surg 2014;18:1071–1076
28. Griffiths EA, Devitt PG, Jamieson CG, et al. Laparoscopic cardioplasty for end-stage achalasia. J Gastrointest Surg 2013;17:997–1001
29. Slone S, Kumar A, Jacobs J, et al. Achalasia quality of life score and Eckhardt score: A comparison for the assessment of clinical improvement post treatment for achalasia. Presented at the 2019 Annual Meeting of the American Gastroenterological Association, San Diego, CA, May 18-21, 2019
30. Molena D, Mungo B, Stern M, et al. Outcomes of esophagectomy for esophageal achalasia in the United States. J Gastrointest Surg 2014;18;310–317
31. DeHaan RK, Frelich MJ, Gould JC. Intraoperative assessment of esophagogastric junction distensibility during laparoscopic Heller myotomy. Surg Laparosc Endosc Percutan Tech 2016;26:137–140