Esophageal Motility Disorders

11

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Overview

Esophageal motility disorders are broad and present at various times in their natural course. This can make an exact diagnosis challenging. These various pathologies unfortunately have no definitive cure. All treatments, medical or surgical, are based on palliation and symptom relief [1].

The goal of this chapter is to provide an overview of the latest recommendations in the diagnosis, workup, and management in a spectrum of esophageal dysmotility syndromes.

History

The first account of surgical disease of the esophagus dates from 3600 BC to 2500 BC to the famed ancient Egyptian "Edwin Smith Papyrus," in which there is a description of "a gaping wound of the throat penetrating the gullet," and the repair of a cervical esophagus with assumingly a muscle flap, "Thou shouldst bind it with fresh meat the first day. Thou shouldst treat it afterwards with grease, honey, (and) lint every day, until he recovers" [2, 3].

Later, circa AD 0 Chinese scripts detail patients with esophageal cancer and associated dysphagia and dysmotility [4]. The first documented treatment of esophageal dysphagia, thought to be achalasia, was recorded in 1679 by Thomas Willis in which he described using a sponge-tipped whale bone to assist in passage of food

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bolus lodged in the esophagus [5, 6]. Later, in 1913, Heyrovsky published the first open surgical approach to "idiopathic dilation of the esophagus" in which he described a series of patients in which he performed an anastomosis of the distal esophagus to the gastric fundus [7]. Shortly afterward, Heller, De Bruine Groeneveldt, and Zaaijer described the esophagocardiomyotomy [8], which has since been modernized to what we refer to as the Heller myotomy. Since that time, minimally invasive techniques using endoscopy and robotics have emerged, which we will explore in this chapter. Presently, treatment for esophageal dysmotility syndromes ranges from behavioral, to pharmacologic, to endoscopic and surgical.

Initial Workup and Diagnosis

History and Physical Exam

As with all ailments, proper diagnosis begins with the careful history and physical examination of the patient. Most patients will complain of chest pain, and thus it is important to rule out acute coronary syndrome while proceeding with a workup. Particular attention should be paid to habits pertaining to diet and associated symptoms including chest pain and weight loss. Points to question in detail are any symptoms of dysphagia, retrosternal chest pain, immediate postprandial regurgitation, and halitosis [9, 10]. With achalasia, patients may complain of retrosternal pains when ingesting cold liquids and cold substances such as ice cream, which sit statically in the distal esophagus. If dysphagia is present, what is its quality? Has the dysphagia been progressive and does it favor solids or liquids? If gastric bloating, distension, and delayed postprandial emesis are endorsed, gastroparesis may be present. Upper respiratory tract complaints may be present as well, which are similar to those with gastrointestinal (GI) reflux disease such as cough, asthma, and even pulmonary fibrosis [11].

The Eckardt scoring system (Fig. 11.1) is traditionally used for patients with dysphagia and is a good and validated subjective marker for the need for treatment and can be followed postoperatively [12–15].

Physical examination, while important, is likely to be unremarkable. With the exception of signs of weight loss such as cachexia, temporal wasting, and thinning

Score				
Symptom	0	1	2	3
Dysphagia	None	Occasional	Daily	Every meal
Regurgitation	None	Occasional	Daily	Every meal
Chest pain	None	Occasional	Daily	Several times per day
Weight loss (kg)	0	<5	5–10	>10

Fig. 11.1 Eckardt score graded 0–12 for subjective measurement of severity of dysphagia

of the thenar eminences, an examination is likely to be negative. It is of utmost importance to examine nodal basins, as esophageal and gastric cancers should be a part of the initial differential diagnosis. The findings of enlarged cervical, supraclavicular or periumbilical lymphadenopathy will drastically change the further workup and management.

Initial Testing

Upper GI Fluoroscopy

Fluoroscopic evaluation is the first test of choice and should be obtained on all patients being assessed for upper GI motility disorders. Contrast-enhanced video fluoroscopy should be performed prior to endoscopy to evaluate for diverticulum, since endoscopy in this setting can possibly result in perforation. Video fluoroscopy allows for visualization of esophageal dilation, length, the presence of diverticula or a hiatal hernia, as well as gastroesophageal reflux. Several pathognomonic signs can be present on an esophagram, most famously the "bird's beak" (Fig. 11.2) appearance of the esophagus at the lower esophageal sphincter [16].

The Rezende classification (Fig. 11.3), sorted between I and IV, has typically been used to communicate the extent of esophageal dilation and tortuosity [17].

EGD

Esophagogastroduodenoscopy (EGD) should be performed on all patients for all suspected esophageal motility disorders and most other pathologies of the foregut. There are multiple utilities for EGD including, importantly, its assessment for carcinoma. Additional pertinent findings on EGD are for the caliber and mucosal quality of the esophagus, if a hiatal hernia is present and the concomitant presence of *Helicobacter pylori* [11]. Biopsies should always be taken of any suspicious esophageal, gastric, or duodenal lesions.

Endoscopic findings particularly indicative of achalasia are numerous. In 2012, the Japan Esophageal Society established several typical findings including the dilation of the esophageal lumen, retained food bolus in the distal esophagus after their midnight fast, whitish thickening along the mucosa—a combination of adhesive debris from food and candida—functional stenosis of the gastroesophageal junction, and abnormal contractions of the esophageal body [18] (Fig. 11.4). An additional finding, a so-called "Pinstripe pattern" can also been seen in up to 60% of patients and is characterized by the longitudinal wrinkling of esophageal [19] (Fig. 11.5). On passage of the endoscope of the gastroesophageal junction, a typically popping sensation may be felt as the endoscope overcomes the pressure of the lower esophageal sphincter.

Fig. 11.2 Barium
swallow. Dilated
esophagus with retained
column of barium and a
"bird's beak sign"
suggestive of achalasia.
(Reproduced from Farrokhi
and Vaezi [106])
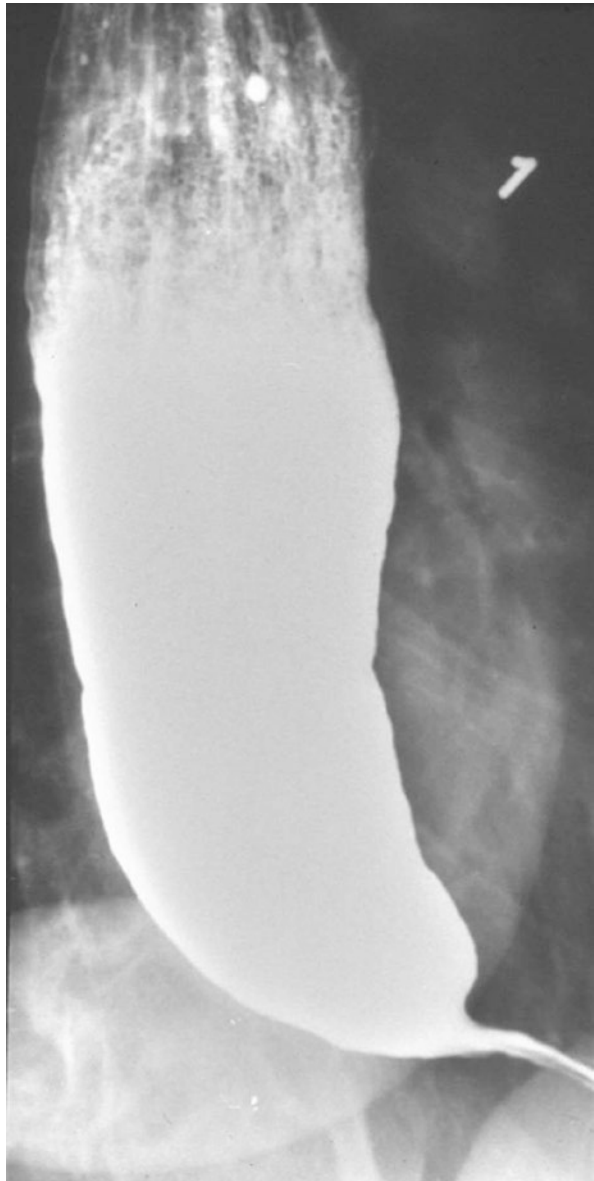
Manometry

Esophageal manometry for diagnosis of dysmotility syndromes is not only gold
standard but also helps classify dysmotility syndromes into several subclasses. The
advent and incorporation of high-resolution manometry (HRM) in the early 2000s
have been of invaluable help. Previous manometric studies limited recordings of
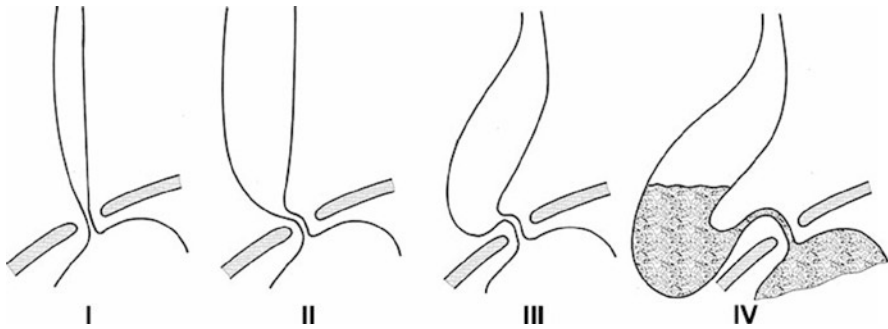
Fig. 11.3 Progression of esophageal dilation and contrast retention according to Rezende's classification of Chagastic megaesophagus. (Reproduced with permission from Griffiths et al. [102])
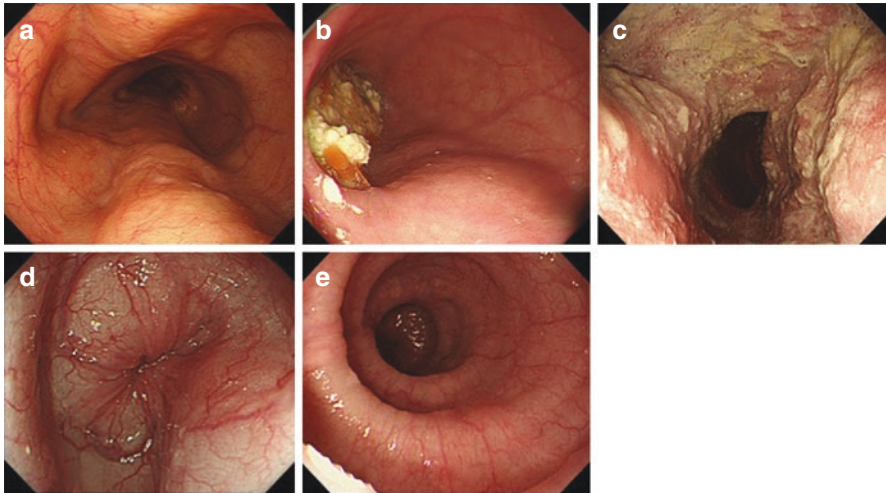

Fig. 11.4 Typical findings of achalasia on EGD (**a**) dilated esophagus, (**b**) retained food, (**c**) increased debris and bacterial overgrowth, (**d**) hypertrophic lower esophageal sphincter, (**e**) spastic paralysis. (Reproduced from Minami et al. [19])

esophageal pressure every 5–8 cm, whereas present HRM systems record pressures every 1 cm apart [20]. They are more accurate in providing a proper diagnosis and they generate colored output graphs that are easier to interpret and conceptualize in comparison to older linear plots [20].

The Chicago Classification v3.0 system [21], modified in 2015, gives the latest subdivisions of esophageal motility disorders based on HRM findings (Fig. 11.6). HRM has resulted in better diagnosis and differentiation of various esophageal motility disorders than traditional manometry [20].

Disorders can be simply subdivided into three main categories: (i) major disorders or peristalsis, (ii) minor disorders of peristalsis, and (iii) disorders with esophagogastric junction (EGJ) outflow obstruction [21].
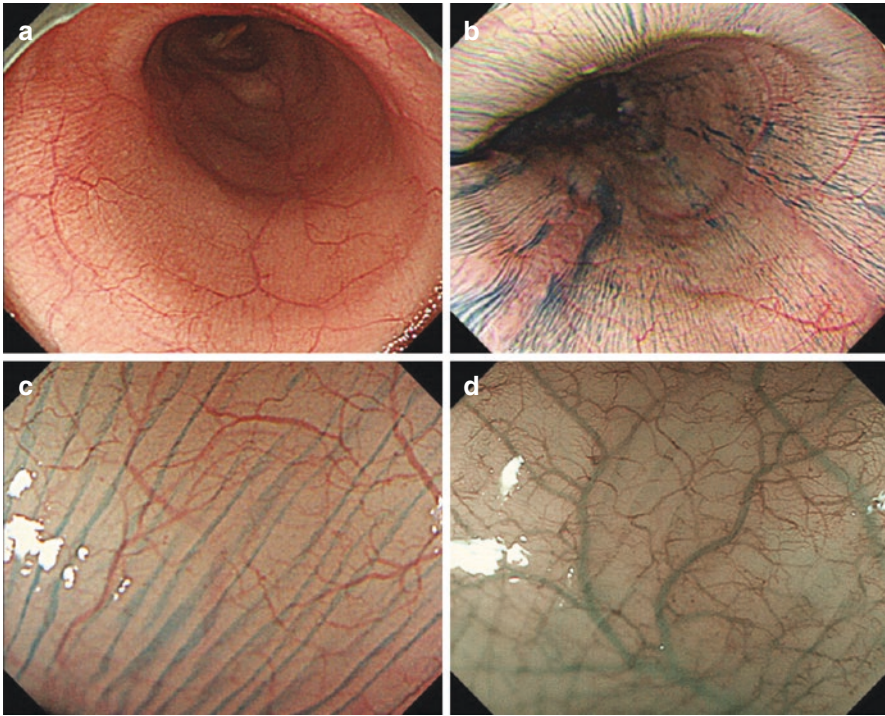
Fig. 11.5 Pinstripe pattern (**a**) minute superficial wrinkle on mucosal surface, (**b**) indigo carmine spray making the superficial structure clearer, (**c**) magnification after indigo carmine spraying, (**d**) narrow band imaging with magnification. (Reproduced from Minami et al. [19])

Some authors have started to use HRM intraoperatively to identify exact areas of esophageal hypertension to tailor the location of performed myotomy [22, 23]. However, multiple studies performed show varying results.

Several patient factors such as obesity, previous bariatric surgeries, diabetes, and possibly eosinophilic esophagitis can effect manometry studies and should be taken into account when evaluating a patient [24–28].

Differential of Dysmotility Syndromes

Achalasia

Achalasia has an incidence of approximately 1 in 100,000 per year. It does not discriminate against gender and has an increasing incidence later in life, but it can be present in the pediatric and young adult populations as well [29, 30].

Esophageal achalasia is defined by features from the Chicago Classification v3.0 system, and is a subdivision of EGJ outflow obstruction. Achalasia falls within a major disorder of peristalsis. It is defined by an elevation of the median integrated
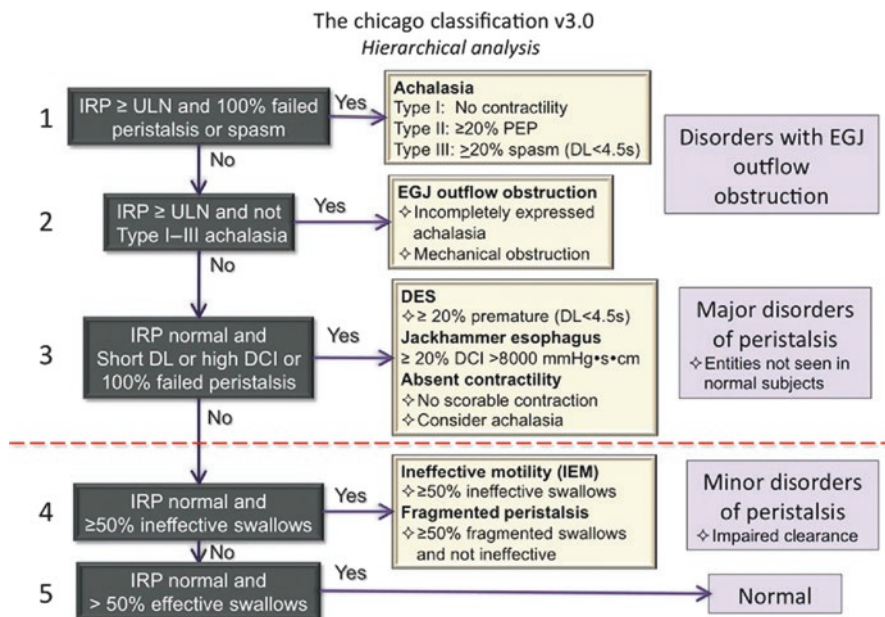
The chicago classification v3.0
Hierarchical analysis

Fig. 11.6 Hierarchical algorithm for classification of motility disorders by the Chicago Classification. (Reproduced with permission from Kahrilas et al. [21])

relaxation pressure (IRP) with associated failed peristalsis or spasm. It can be broken down into three subtypes: type 1 achalasia is classical achalasia—100% failed peristalsis (distal contractile integral [DCI] <100 mmHg/s/cm) with failure of relaxation of the lower esophageal sphincter on swallowing (IRP >15 mmHg); type 2 has the added feature of increased panesophageal pressures on greater than 20% of swallows; and type 3 has the further added feature of esophageal spastic contractions (DCI >450 mmHg/s/cm) in greater than 20% of swallows. The differentiation of the subtypes is important as different surgical options are more efficacious than others depending on subtype [31].

The pathophysiology and mechanism of achalasia are still debated with several theories. There is some evidence that achalasia may be a consequence of infectious disease with an antecedent viral infection [31, 32] or due to the parasite *Trypanosoma cruzi*, the so-called Chagas disease, which will be highlighted later in this chapter. Otherwise, much of its mechanism is still idiopathic and under investigation. Histologically, there is proliferation of cytotoxic T-lymphocytes [33] and mast cell into the myenteric (Auerbach) plexus with neural loss and inflammation [34]. The aperistaltic segment of esophagus is likely due to failure of normal vagal motor function and dysfunctional cholinergic mechanisms. These alterations take time to develop, accounting for the various subtle symptoms of this disease and sometimes discrepancies in objective test results may explain for its delay in diagnosis [35, 36].

Long-term achalasia without treatment can progress to malnutrition, chronic aspiration, or eventual esophageal carcinoma. The malignant transformation, unlike

in Barrett's esophagus with gastrointestinal reflux disease, has a higher prevalence of degeneration into squamous cell carcinoma. The prevalence for squamous cell carcinoma is 26 per 1000 patients—nearly 300-fold absolute risk increase when compared to the general population [37]. The risk for adenocarcinoma, while still elevated, is not nearly as outstanding as squamous cell. Its prevalence is nearly 4 per 1000 cases of achalasia and has an 18-fold increased absolute risk with its predilection potentially from nitrate concentration due to bacterial overgrowth [37]. Patients with at least 10-year history of disease should undergo endoscopic surveillance [37].

It is important to take history from a patient to rule out pseudoachalasia, usually hallmarked by extrinsic compression of the esophagus. Such causes can be a previous gastric fundoplication performed too tightly, a gastric band for weight loss [38], an overtightened LINX reflux management system [39], or dysphagia lusoria—extrinsic compression of an aberrant right subclavian artery [40]. It is not uncommon for those with achalasia to be diagnosed initially as having pseudoachalasia with reflux to and for those patients to undergo fundoplication.

Treatment of achalasia is vast—from pharmacologic management with calcium channel blockers to nitrates to relax the lower esophageal sphincter to endoscopic means to surgical. Treatment should best be individualized to the patient and degree of achalasia with consideration to potential comorbid conditions.

Chagas Disease

Chagas disease is caused by the parasite *Trypanosoma cruzi* and is mostly endemic to South America. The World Health Organization classifies it as a neglected tropic disease and in 2010, nearly six million people were thought to be infected [41]. The effects of Chagas disease is syndromic, causing dysfunction of the heart, viscera, brain, and other organs [41–43]. The exact mechanism of virulence is still under investigation, but is thought to involve over-excitatory effects of T lymphocytes, interferon gamma, tumor necrosis factor, and other cytokines [43]. Elevated levels of M2 acetylcholine muscarinic receptor autoantibodies have been identified at a higher rate compared to idiopathic achalasia—84% vs. 28%—which may play a role in the development of megaesophagus and the loss of Auerbach's and Meissner's plexuses [43].

Diagnosis involves a high index of suspicion when interviewing patients from endemic areas and confirmation using histology, polymerase chain reaction (PCR), and serology antigen assay [41, 42]. All patients with a confirmed diagnosis should have a cardiology consultation, with a minimum of a 12-lead echocardiogram (EKG). Workup of esophageal involvement is the same as other dysmotility disorders and is highlighted above but with the additional cavoite that manometry should be performed even in the setting of a normal esophagram [41]. Exact patterns on high-resolution manometry are currently being investigated and show mixed results. One study of symptomatic patients shows a decrease in esophageal body and lower esophageal sphincter pressure when compared to idiopathic achalasia, which may reflect more the degree of esophageal dilation, which is inversely related to transduced pressures on

HRM [44]. While, on the other hand, additional findings suggest that in the chronic phase of the disease (which can be relatively asymptomatic), there is a relatively hypotonic lower esophageal sphincter and hypertonic upper esophageal sphincter and may not correlate to a patient's degree of symptoms [45].

The risk for carcinoma of the esophagus is significantly higher in areas of the world where Chagas disease is endemic. Prevalence is as high has 56 per 1000 cases of achalasia patients. Mutations of the Fragile Histidine Triad Diadenosine Triphosphatase (FHIT) and tumor protein 53 (TP53) genes as well as abnormalities in chromosomes 7, 11, and 17 may be associated with degeneration to carcinoma [37].

The mainstay of treatment for Chagas disease remains medical with antiparasitic agents, namely benznidazole and nifurtimox [42]. It is paramount to work closely with infectious disease physicians who specialize in tropical diseases. Initial surgical treatment with Chagastic esophagus can be similar to that of idiopathic achalasia. One must be cautious however because some patients with Chagas disease can have an entity of gastroparesis, causing drastic worsening of reflux disease and gastric distension. In such cases, other surgical options such as the Serra-Dória procedure—cardioplasty with partial gastrectomy and Roux-en-Y reconstruction—should be entertained [46, 47]. In cases of megaesophagus an esophagocardioplasty (Fig. 11.7)—Grondhal's cardioplasty [46], or with a gastric patch—modified Thal procedure may be needed [48]. In other cases of end-stage disease, endoscopic mucosal resection [32] or a total esophagectomy may be necessary [47].

Systemic Sclerosis (Scleroderma)

Systemic sclerosis is an autoimmune syndrome with a predilection to females in the fourth through sixth decades of life. The esophagus is the most common gastrointestinal organ involved with the disease, and can occur in up to 90% of patients, 40–80% of whom are symptomatic [49]. At its root, there is fibroblastic and collagen proliferation in cellular tissue, leading to calcifications and sclerosis. A patient's
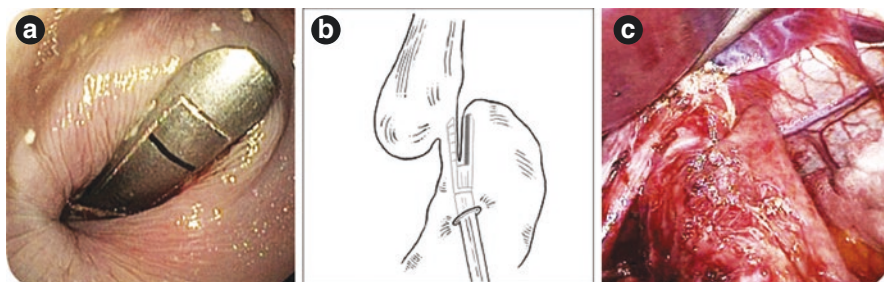

Fig. 11.7 Esophagocardioplasty. (**a**) endoscopic view of bottom jaw of the laparoscopic angled gun into the esophagus, (**b**) optimal positioning of the gastrotomy 4 cm below the gastroesophageal junction, (**c**) laparoscopic view of the stapler placement into the esophagus and fundus simultaneously. (Reproduced with permission from Griffiths et al. [102])

most typical complaint when there is esophageal involvement is gastrointestinal reflux disease and dysphagia [50].

Most diagnostic workup as detailed above reveals a hypotonic lower esophageal sphincter, aperistalsis, dilated esophagus, and acidic esophageal pH [51, 52].

Pharmacotherapy is first line of treatment, and should be managed primarily by a rheumatologist. The objective is to decrease symptoms of reflux and promote esophageal motility, done with a combination of proton pump inhibitors and prokinetic agents such as domperidone and buspirone [49].

Surgical management should be cautioned, especially in the treatment of reflux. Fundoplications alone should be contraindicated due to profound postoperative dysphagia. Other surgical choices such as the Roux-en-Y gastric bypass can be problematic as well if small bowel dysmotility is involved, but it may be the best option [49, 53]. As in Chagas disease, some patients with crippling quality of life may have indication for esophagectomy [54]. Regardless, surgical options should be reserved for patients who are refractory to medical management and for whom symptoms effect quality of life.

Other Spastic Disorders of the Esophagus

Distal esophageal spasm, hypercontractile esophagus, and aperistalsis are additional, but not a complete list of other esophageal motility disorders. High-resolution manometry is used to break down and subclassify this cohort and help to guide treatment [55] (Fig. 11.8).

Absent peristalsis characteristically has lack of esophageal motility with a normal lower esophageal sphincter. Patients with scleroderma will typically have this pattern of manometry and complain of regurgitation and reflux. Patients with long-standing severe gastroesophageal reflux disease (GERD) can also develop this pathology as a result of its ensuing fibrosis. Patients commonly have respiratory complaints such as cough, wheezing, and asthma; they can even progress to a state of pulmonary fibrosis [50]. The Chicago Classification subclassifies this cohort into four types of delayed peristalsis: absent, weak with large or small peristalsis defects, and frequent failed peristalsis. As one may imagine, fundoplication procedures may make this problem exceedingly worse and should be considered in a multidisciplinary fashion if chosen as part of therapy [56].

Hypertensive lower esophageal sphincter or esophagogastric junction outflow obstruction was most recently described by Code in 1960 [57]. Failure to previously identify this disease was probably due to failure to discriminate this pathology from achalasia [58]. Manometry shows an increased resting pressure of the lower esophageal sphincter greater than 15 mmHg but retains its ability to relax. Esophageal peristalsis is usually preserved, but can be diminished—but not to the extent as to meet criteria for achalasia.

Distal esophageal spasm is diagnosed in approximately 4–10% of patients on HRM and is more frequently seen in females and the elderly. This disorder was first proposed by Osgood in the 1880s in people who complained of chest and epigastric
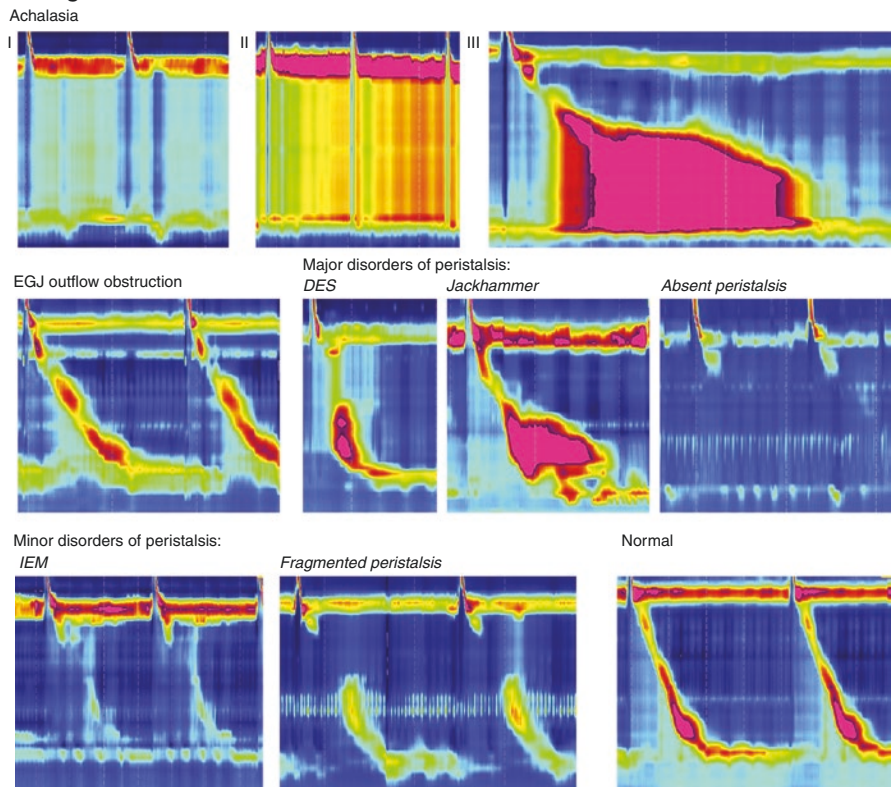
Chicago classification:

Achalasia

Fig. 11.8 Outputs of high-resolution manometry for various motility disorders, including subtypes of achalasia. Horizontal axis shows time, vertical axis shows length along the esophagus, colors from blue to red spectrum demonstrate increasing pressure. (Reproduced with permission from Rohof and Bredenoord [20])

pain with concomitant dysphagia [59]. Barium esophagram may show a "corkscrewing" pattern or pseudodiverticula. Manometry will show esophageal spasm in greater than 20% of swallows, usually in the distal esophagus, with a normal lower esophageal sphincter pressure [20, 60].

Hypercontractile esophagus, colloquially termed Jackhammer esophagus, is defined by greater than 20% of contractions being greater than 8000 mmHg/s/cm [21, 55]. The location of contraction along the esophagus is nonspecific, and can include the lower esophageal sphincter. It is present in 4.1% of patients who undergo manometry [61]. Most patients with this pathology will complain of chest pain and dysphagia.

Like achalasia, therapy is palliative and guided at treating dysphagia chest pain and reflux. Surgical therapy is explored after failure of initial medical and endoscopic management. Because of the multifocal nature of this nonspecific esophageal disorders, balloon dilation and Botox injection have limited roles. Medical

and endoscopic therapies are profoundly less efficacious than eventual surgical therapy [11].

There is limited prospective data on surgical options outside of achalasia, but similarly, authors agree on procedures involving myotomy and fundoplication. However, success rates in decreasing dysphagia and GERD are less successful than in achalasia. Furthermore, of considerable note is the length of esophageal myotomy needed in this patient cohort. The hypertrophic musculature and spasm occur along a greater length of the esophagus, and a transabdominal approach may result in a short myotomy. Thoracic approaches then are used to perform a long esophageal myotomy and gastric fundopexy [62–64]. This subset of patient may be benefit greatly from and endoscopic myotomy [65, 66], which will be discussed further later in this chapter.

Pharmacological Treatment

Esophageal motility disorders are not curative diseases. All treatments, whether medical, endoscopic, or surgical, are palliative with the goal of symptom alleviation [67, 68].

The mainstay of nonprocedural management is based on dietary habits and the use of calcium channel blockers and nitrates. The calcium channel blocker nifedipine is taken to relax the smooth muscle of the lower esophageal sphincter by inhibiting calcium reuptake. Resting pressure can be at times lowered by up to 60%. Side effects can occur in up to 30% of patients and could be cause for poor compliance. Mainly patients complain of dizziness, headache, and orthostatic hypotension [22]. These symptoms can be exacerbated if the patient is taking other antihypertensive agents. Nitrates behave similarly, resulting in increased relaxation of the lower esophageal sphincter and the increased passage of food bolus. The subjective patient improvement, however, was limited and full recommendations to clinic practice cannot be made.

Endoscopic Treatment for Achalasia

Endoscopic treatment for achalasia consists mainly of three procedures: Botox injection, pneumonic dilation, and per-oral endoscopic myotomy (POEM). Injection of botulinum toxin A into the lower esophageal sphincter is an attractive and logical treatment option. Unfortunately, its effects are short lived, lasting between 6 and 12 months [36]. Consequently, botulinum toxin causes scaring in the submucosal layers. This results in increased rate of perforation during later surgical myotomy [69, 70]. It is an increasingly controversial approach, and may only be appropriate to those who are unable to undergo general anesthesia and for whom life expectancy may be short [71].

Pneumatic dilation was introduced in the 1970s and 1980s and still is relevant in current treatment. Endoscopic treatment results in perforation with 1–3% of cases,

half of which require surgery [72]. This risk increases both with each subsequent dilation and with the use of larger balloon dilators greater than 30 mm on initial dilation [36]. Treatment, however, can be quite robust, with maintained relief of dysphagia of 84% at one month, but may decline to 58% by 3 years [73]. Results may be best in those with type 2 achalasia. Balloon dilation is favored over bougie due to the ability to have direct visualization of the pathology, rather than blindly or with fluoroscopic guidance [74].

Surgical Treatment for Achalasia

Ernest Heller first described his operation in 1914 (on a patient he operated on in 1913) and was in fact different from his namesake procedure. In his original approach, he performed a myotomy both on the anterior and on the posterior esophageal surfaces [8]. The named procedure "Heller myotomy" is more similar to De Bruine Groeneveldt's description in which a single longitudinal anterior esophagocardiomyotomy was performed, but without a gastric fundoplication. Presently, with the advent of laparoscopy and improvements in surgical technique, this has brought us to a single anterior myotomy with partial fundoplication, known today as a Heller's myotomy—today's gold standard in the United States [70, 75].

The application of a partial fundoplication has since reduced the rate of postoperative reflux from 32% to 8% [73]. Whether an anterior or posterior partial fundoplication is used has been a point of debate in the literature for some time. The majority of patients in literature-based searches has a Dor fundoplication, but this is likely favored based on surgeon preference rather than robust data [76].

Comparison meta-analysis of Heller myotomy and endoscopic balloon dilation favors surgical myotomy in reducing dysphagia, while having similar safety profiles and rates of reflux disease [72, 73]. The Heller myotomy has shown to decrease the rate of chest pain and dysphagia in up to 90% of patients, and depends somewhat on the stage of achalasia and subclassifications based on high-resolution manometry [73]. Perioperative complications and generally minimal and include perforation, wrap dysfunction, and dysphagia. Perforations are reported in approximately 1.6% of cases [70].

Since its advent, robotic surgery has been introduced to the foregut as well. While potentially having ergonomically advantages to the surgeon, outcomes data has not shown robotic-assisted Heller or transthoracic myotomy to be superior to laparoscopic [77, 78]. A recent publication, however, reported a decreased rate of complications with a robotic approached compared to traditional laparoscopic [79].

Peroral Endoscopic Myotomy

POEM is perhaps the champion of the nature orifice surgery movement and continues to gain traction. The procedure was first performed and described in 1980 by Ortega, Madureri, and Perez on a short series of treatments involving six dogs and
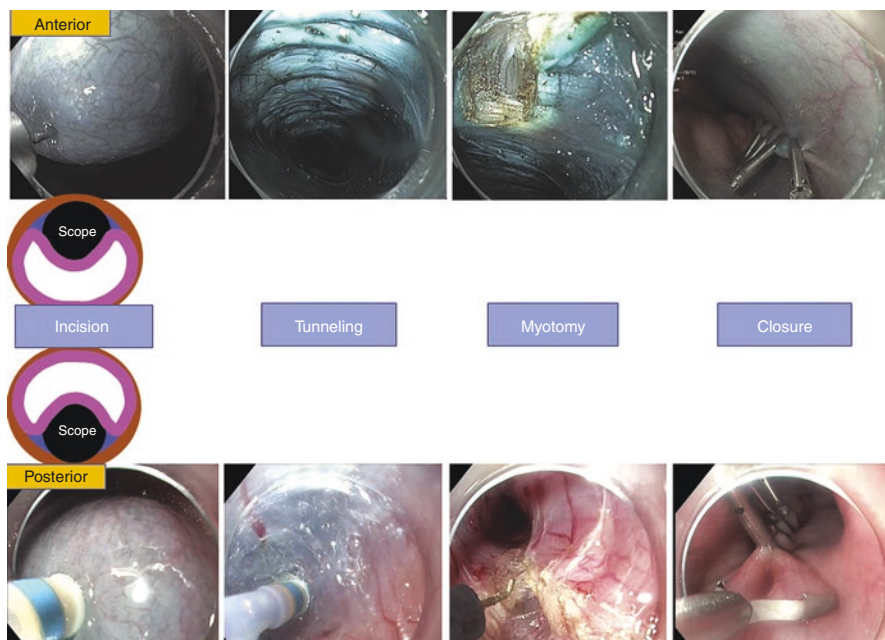
Fig. 11.9 Peroral endoscopic myotomy (POEM) procedure. Anterior POEM is performed in the 12 to 2 o'clock position, whereas posterior POEM is performed in the 5 to 6 o'clock position. Both approaches entail four stages: mucosotomy, submucosal tunneling, myotomy, and mucosal closure. (Reproduced with permission from Khashab et al. [107])

seventeen patients [80]. The technique, however, was abandoned for some time until it was reintroduced by Haruhiro Inoue in 2008 [81]. The procedure entails using a therapeutic gastroscope in which an esophageal submucosal tunnel is made approximately 4 cm proximal to the start of the myotomy and carried down 3–4 cm into the stomach distally. The circular fibers of the lower esophageal sphincter are identified and divided, and the mucosal defect is then closed endoscopically. Creation of the myotomy on the anterior or posterior surfaces of the esophagus seems to have equal efficacy [82] (Fig. 11.9).

Complications are minimal [83] in experienced hand and are limited to bleeding usually manageable with cautery and small perforations that can be controlled with endoclips. Rarely is conversion to laparoscopic/thoracoscopic or open procedures necessary. Obtaining an upper GI series on postoperative day 1 is not necessarily standard and may only be helpful if symptoms concerning for perforation are present such as tachycardia, shortness of breath, and chest or abdominal pain [84].

Inoue and his group's experience of 500 patients from 2008 to 2013 were successful in treating all variants of achalasia including sigmoid esophageal anatomy. They report significant improvement in Eckardt scores out to 3 years [81].

In comparing treatment-naive patients with achalasia, POEM had superior 2-year outcomes than endoscopic balloon dilation [60]. Specifically, 92% of patients who

had POEM experienced improvement in dysphagia, compared to only 54% in the balloon dilation group. However, rates of reflux were higher in the POEM group—41% compared to 7%.

The use of POEM is gaining traction as for nonspecific esophageal motility disorders. The principle of an extended or long myotomy is well suited for POEM, since a submucosal tunnel can be easily initiated anywhere on the esophageal body and tailored towards manometric results. The use of a long myotomy, as advocated for in type III achalasia, can be accomplished nicely with POEM as shown in a recent series to have better success in this achalasia subtype when compared to Heller myotomy—98% vs. 80.8% success rate [13]. The success of POEM for type III achalasia, diffuse esophageal spasm, and jackhammer esophagus may be on the order of 92%, 88%, and 72%, respectively [65]. The decreased rate of success in jackhammer esophagus may be of a technical error if the lower esophageal sphincter is not included in the myotomy [85].

Recent reports demonstrate that POEM is more effective than Heller myotomy in relieving short-term dysphagia [86]. However, this does come at a price of increased levels of gastrointestinal reflux disease and asymptomatic esophagitis, which also increases in rate the further into the postoperative period the patient is [81, 86–88]. This result is not surprising as POEM is done without performing fundoplication [35]. These findings of increased GERD in patients who undergo POEM as compared to Heller myotomy are also seen on postoperative pH monitoring [86]. Interestingly, body mass index may not be a risk factor in the development of postoperative reflux in the POEM patient [89]. However, this may suggest that POEM can predispose a patient to the Barrett's metaplasia-dysplasia-carcinoma pathway. But until longer follow-up is established with POEM, this is still speculation. POEM has not been showed to be a carcinogenic procedure at this time.

Presently, POEM is still a highly specialized procedure and is not offered at all specialty centers. Most general surgery residents and many fellows who train in minimally invasive surgery are not exposed to this procedure. Thus, many who do POEM, learned the technique after their formal graduate medical education. It takes approximately 15–20 procedures, with appropriate faculty supervision to become facile and independent in POEM [35, 90].

Recurrent Dysphagia

Success of surgery is usually defined as postoperative Eckardt scores of less than 3 [12, 65, 91]. Surgical intervention for dysphagia is typically a robust intervention with lasting long-term results. There is, however, a small subset of patients who fail surgical intervention and have recurrent dysphagia. Preoperative predictors of failure may include higher Eckardt scores (≥ 9) and achalasia subset type III [91, 92]. POEM has a potential dysphagia recurrence rate of 10%, while laparoscopic myotomy is reported between 3.5% and 15%.

Reasons for recurrent dysphagia are multifactorial. Eventual failure of the Heller myotomy or recurrence of symptoms is due to incomplete myotomy (33%),

myotomy fibrosis (27%), fundoplication disruption (13%), tight fundoplication (7%), or some combination of two (20%) [93]. Other lesser, but still possible, reasons for failure include overtightening of crural closure (if performed), peptic stricture, carcinoma, and even incorrect index diagnosis [94]. It can be easy to confuse gastroparesis, either undiagnosed or iatrogenic, from vagal nerve injury, as a possible cause of recurrence, as these patients will present with emesis and bloating with or without dysphagia.

Eckardt scores can be followed in patients to track their subjective complaints. As with following patients with reflux, it is important to correlate their subjective complaints with objective data [95]. Workup of recurrent dysphagia should be meticulous and mirror that of primary dysphagia [75, 94]. Again it starts with an upper GI contrast study. Evidence of the classic "bird's beak" appearance can be seen. HRM can often be misleading and unhelpful for a previously operated patient. Endoscopy can be both diagnostic and therapeutic. Pneumatic dilation, at times up to 40 mm, is typically the first intervention performed and its rate of success has been reported between 50% and 70% [96, 97]. Cross-sectional imaging, while not mandatory, can be helpful in defining anatomy and may provide insight to the etiology of what is provoking the recurrent dysphagia.

Unfortunately, some patients do not respond to repeat dilations, and their pathology can be lifestyle crippling. Many patients undergo repeat surgical intervention, such as redo Heller myotomy or POEM and therapy is usually successful [98–100]. Procedure selection is challenging and should be tailored to the patient. Redo myotomy is typically more difficult, as shown by increased rates of esophageal perforation, reported between 13% and 33% [98–100]. Presently, with more and more centers and surgeons becoming proficient in POEM, success rates on patients needing redo procedures approach that of primary POEM [99]. Importantly, when planning redo interventions, a previous primary Heller myotomy does not preclude a patient from having a secondary POEM, and vice versa [12].

Repeated recurrence despite persistent therapy usually leads to esophagectomy. Several salvage procedures, while are seldomly performed, do exist. Some surgeons and centers have experience with advanced techniques such as the modified Thal procedure, Serra-Dória operation [78, 95, 101, 102]. A newly published limited series describes resection of the dysfunctional gastroesophageal junction with esophagojejunostomy, using a Roux limb taken 30 cm from the ligament of Trietz and anastomosis of the in situ gastric remnant with biliopancreatic limb 60 cm down the Roux limb [103]. (insert pictures of operative anatomy).

Total Esophagectomy

Esophagectomy, while controversial, may be necessary for patients with end-stage disease of achalasia and dysmotility disorders [68]. Up to 5% of patients with disease, particularly achalasia, will undergo esophagectomy, most of them having previous endoscopic and myotomy procedures [1, 104]. Indications for total esophagectomy involve symptomatic features of end-stage disease, which are

refractory to previous medical and surgical management. Such features include sigmoid esophagus >6 cm (the so-called dolicho-megaesophagus), disabling gastric reflux disease and dysphagia, malnutrition, recurrent aspiration pneumonia, airway compromise from extrinsic compression, recurrent bleeding, stricture, underlaying cancer, and if part of clinical trials [54, 67, 104].

Some authors report increased rates of bleeding, especially when a transhiatal approach is used, due to dense mediastinal adhesions from the chronic inflammatory state of the disease and previous procedures [105]. Laryngeal nerve anatomy and its displacement must be taken into account as well [68]. While most procedures are performed transthoracic as compared to transhiatal (73.9% vs. 26.1%) [1], both are acceptable and safe, especially since surgeons have become more experienced with minimally invasive and robotic approaches.

Esophagectomy has generally been tolerated well, especially at centers of excellence. Study of the Nationwide Inpatient Sample from 2000 to 2010 in comparing esophagectomy for achalasia compared to cancer showed decreased rates of mortality (3% vs. 8%) and postoperative complications were linked more to preoperative nutrition status rather than indication for surgery [67]. Pneumonia (15%) and anastomotic leak (7%) are the two most significant complications [1].

The choice of conduit for reconstruction is still of some debate. However, most surgeons and authors advocate for gastric interposition as first line conduit when available and is most used (95%), followed by colon and then small bowel [1, 54, 68, 104]. This is generally due to the robust vascular supply of the stomach and the need only for a single anastomosis when used.

Quality of life improves for the vast majority of discharged patients (75–100%) [1], with approximately 20–30% require further dilation due to anastomotic stricture and 20% complaining of dumping syndrome. Nearly all patients are able to normalize nutritional parameters and gain weight [104].

Conclusion

Esophageal motility disorders are a complicated pathology. Importantly, providers must be vigilant with a patient's preoperative workup. Obtaining all objective data possible and identifying the nuances are paramount, allowing a standardized, yet tailored approach to each patient. For achalasia, laparoscopic Heller myotomy remains the gold standard. However, for other pathologies such as nonspecific spastic disorder, achalasia type 3, and recurrent dysphagia after Heller myotomy, POEM continues to gain traction and more data on long-term outcomes are being published annually and is considered by some to be the procedure of choice.

Glossary
- Achalasia
- Adenocarcinoma
- Barrett's esophagus
- Chagas disease
- Chicago classification

- Distal esophageal spasm
- Dysphagia
- Dysphagia lusoria
- Eckardt score
- Edwin Smith Papyrus
- Ernst Heller
- Esophagectomy
- Esophagogastroduodenoscopy
- Gastroesophageal reflux disease
- Gastroparesis
- Haruhiro Inoue
- Heller myotomy
- High-resolution manometry
- Hypercontractile esophagus
- Jackhammer esophagus
- Modified Thal procedure
- Pinstripe pattern
- Pneumatic dilation
- Por oral endoscopy myotomy
- Pseudoachalasia
- Roux en Y gastric bypass
- Rezende classification
- Scleroderma
- Serra-Dória procedure
- Squamous cell carcinoma
- Thomas Willis
- Trypanosoma Cruzii
- Upper GI fluoroscopy

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