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# Surgical intervention for esophageal dysmotility

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ABSTRACT

Esophageal motility disorders encompass a number of distinct disease processes that share the common end result of peristaltic derangement and impaired relaxation of the lower esophageal sphincter. This article outlines the surgical and endoscopic interventions appropriate for individuals with achalasia and other motor disorders of the esophagus. Conceptualizing the esophageal motility disorders as primarily nonspastic (types I and II achalasia and esophagogastric junction outflow obstruction) or spastic (type III achalasia, jackhammer esophagus, and distal esophageal spasm) can help inform the choice of initial and subsequent treatments. Pneumatic dilatation and laparoscopic Heller myotomy have roughly equivalent success in relieving symptoms of esophageal outflow obstruction, with a more durable effect seen with the surgical approach as well as decreased incidence of gastroesophageal reflux with the addition of a partial fundoplication. In the management of spastic motility disorders, surgical myotomy has provided significantly better outcomes than pneumatic dilation, potentially attributable to division of spastic musculature proximal to the esophagogastric junction. Similarly, BoTox delivered to the esophageal body has recently been shown to relieve symptoms in distal esophageal spasm. Peroral endoscopic myotomy (POEM) is a novel therapy that allows for the creation of a surgical myotomy, without the need for skin incisions. The antegrade, endolumenal approach used in POEM has also allowed for personalized tailoring of the proximal myotomy of the esophageal body in the treatment of spastic motility disorders. POEM has a growing body of longer-term follow-up that suggest initial concerns surrounding increased incidence of gastroesophageal reflux may not be realized.



## 1. Introduction

Esophageal motility disorders (EMDs) include a heterogeneous group of diagnoses attributed to an imbalance of inhibitory and excitatory signaling in the esophageal body and lower esophageal sphincter (LES) [1]. Decreased delivery of nitric oxide and vasoactive intestinal peptide by the inhibitory ganglions at the level of the esophagogastric junction (EGJ) causes the hallmark failure of LES relaxation seen in EMD. The resulting esophageal outflow obstruction is responsible for the predominant symptoms of dysphagia, regurgitation, and weight loss. The etiology of chest pain, another common presenting symptom, is less well defined and likely multifactorial. Unopposed or upregulated excitatory innervation of esophageal smooth muscle, mediated by acetylcholine, is implicated in spastic disorders of the esophagus where chest pain is more commonly encountered [2].

The introduction of high-resolution manometry (HRM) in the evaluation of esophageal symptoms allowed the development, and subsequent refinement, of a classification system based on parameters of peristaltic vigor and timing as well as the function of the LES [3,4]. The Chicago classification has subsequently been shown to have prognostic value for the 3 subtypes of achalasia in determining response to endoscopic and surgical intervention [5,6].

In the absence of curative treatment, the goals of interventions in EMD are symptom palliation and prevention of disease progression. Therapeutic approaches to the individual motility disorders can be broadly considered to be addressing either the functional obstruction at the level of the EGJ, the spastic smooth muscle of the esophageal body, or both.

## 2. Nonspastic motility disorders

The nonspastic motility disorders include type I and type II achalasia as well as EGJ outflow obstruction and are defined by the loss of deglutitive inhibition of the LES, seen on HRM as an elevated mean 4-second integrated relaxation pressure (Figure 1A-C).

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A common underlying pathophysiology, the loss of inhibitory ganglions within the myenteric plexus of the esophagus, results in the hallmark failure of LES relaxation accompanied by varying degrees of peristaltic dysfunction [1]. The dilation of the esophagus frequently seen in patients with type I achalasia, and the defining absence of peristalsis, may represent a later stage in a disease progression that includes EGJ outflow obstruction with preserved peristalsis and type II achalasia with panesophageal pressurization. Untreated, esophageal outflow obstruction causes progressive dilation, eventually leading to development of sigmoid megaesophagus, also known as end-stage achalasia. With the goal of relieving the esophageal outflow obstruction, surgical and endoscopic treatments are targeted at reducing the pathologically elevated pressure at the LES.

### 2.1. Surgical myotomy

There has been extensive debate regarding how best to balance relief of dysphagia with mitigation of unopposed gastroesophageal reflux (GER) following destruction of the anatomical antireflux barrier at the LES [7-11]. Extensive modifications have been applied to the surgical approach and technical details of the operation first described by Heller [12] in 1914. The original procedure, featuring both a longitudinal anterior and posterior myotomy performed through the abdomen, has undergone continuous evolution in search of a technique that ensures a myotomy adequate for symptomatic relief while minimizing morbidity. Ellis et al [7] summarized the arguments for a transthoracic approach with a minimal gastric myotomy, but with the advent of laparoscopy, the procedure returned to an abdominal approach and the debate turned to prevention of iatrogenic GER. A series of large randomized trials have guided the progression from the ubiquitous reflux that accompanied the original esophagogastric myotomy, to the dysphagia-inducing, 360° Nissen fundoplication to the current practice of combining a longer gastric myotomy (2.5-3 cm) with a 270°, posterior (Toupet) fundoplication [8-10]. Wright et al [10] showed that the current practice, when performed with the standard 6-cm proximal esophagomyotomy, results in lower final pressures at the LES than a shorter gastric myotomy with an anterior (Dor) fundoplication (9.5 vs 15.8 mm Hg) as well as significantly decreased rates of recurrent dysphagia. Further benefits of the posterior partial fundoplication were suggested by a multicenter randomized trial of laparoscopic Heller myotomy (LHM) with Dor vs Toupet fundoplication that resulted in

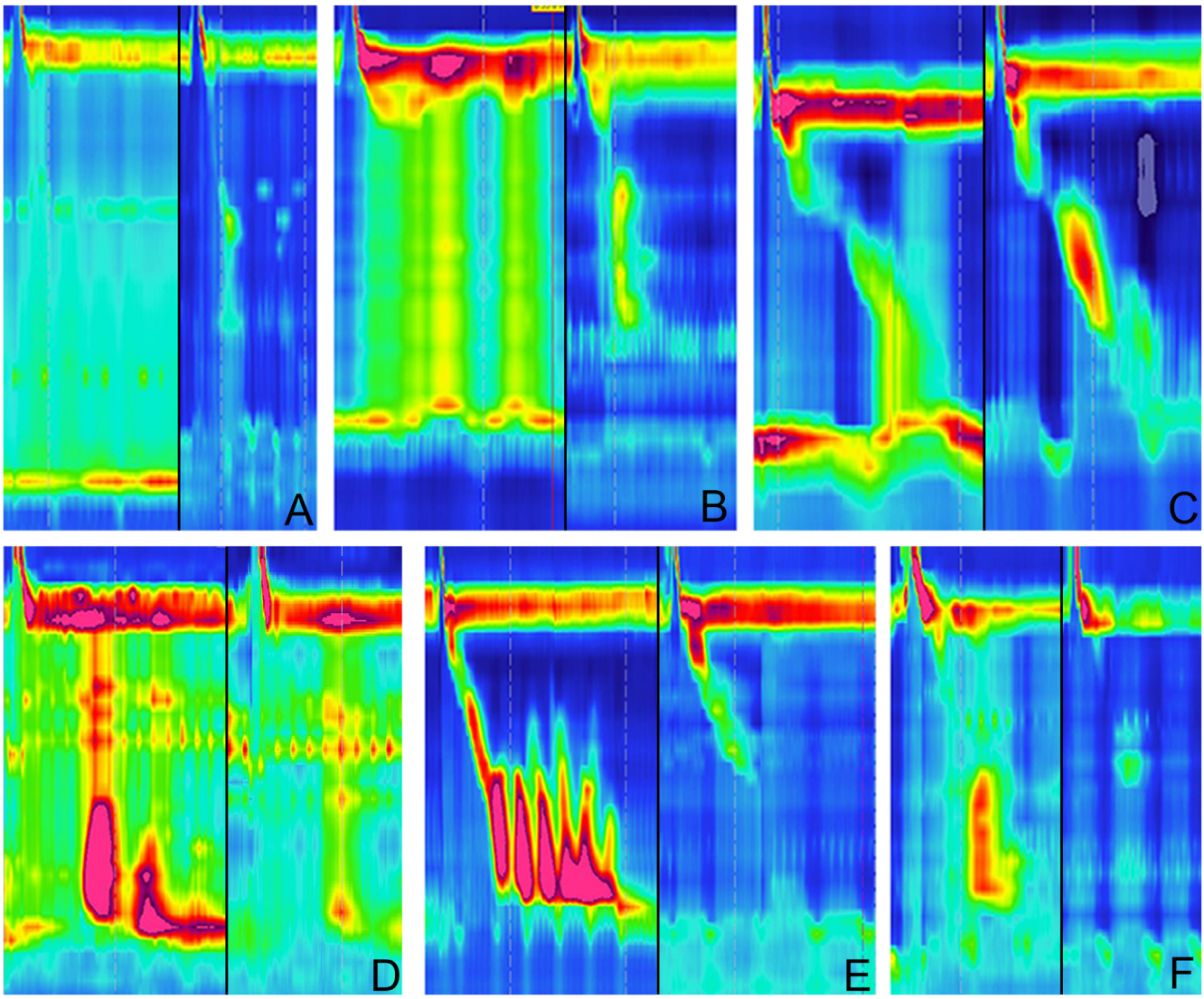


**Fig. 1.** Esophageal pressure topography plots before and after peroral endoscopic myotomy. The top row includes examples of nonspastic achalasia: (A) type I achalasia, (B) type II achalasia, and (C) esophagogastric junction outflow obstruction. The bottom row shows examples of spastic esophageal motor disorders: (D) type III achalasia, (E) jackhammer esophagus, and (F) distal esophageal spasm.