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Pneumatic dilation and laparoscopic Heller's myotomy equally effective for achalasia.

Stuart Jon Spechler, M.D.

VA North Texas Healthcare System and, The University of Texas Southwestern Medical Center, Dallas, Texas

In primary (idiopathic) achalasia, there is degeneration of neurons in the wall of the esophagus, especially inhibitory neurons that produce nitric oxide.[1] Without inhibitory innervation, the lower esophageal sphincter (LES) cannot relax completely with swallowing and the body of the esophagus cannot contract in a peristaltic fashion. The demonstration of these two abnormalities by esophageal manometry establishes the diagnosis of achalasia. The unrelenting LES contraction poses a barrier that impedes the passage of any swallowed material into the stomach. This results in the typical achalasia symptoms of dysphagia for both liquids and solids, and regurgitation. In addition, the abnormal esophageal motility frequently is associated with chest pain.

No therapy is currently available that can reverse the neuronal degeneration of achalasia and, consequently, there is no way to restore esophageal peristalsis and normalize LES relaxation. Available treatments aim at decreasing pressure in the LES to the point that it no longer poses a barrier to the passage of ingested material. Medications that relax LES smooth muscle (e.g. nitrates, calcium channel blockers) have been used to treat achalasia, but this pharmacotherapy is inconvenient, often ineffective, and frequently associated with unpleasant side effects and tachyphylaxis.[2] LES pressure also can be reduced by the perendoscopic injection of botulinum toxin, which inhibits the release of acetylcholine from excitatory neurons in the esophagus. Approximately two-thirds of patients report improvement in achalasia symptoms after botulinum toxin injection, but most relapse within 6 months and the efficacy of repeated injections may decrease over time.[3] Today, pharmacotherapy and botulinum toxin injection generally are reserved for patients who are deemed too old or infirm to tolerate invasive, mechanical therapies that disrupt the LES.[2]

The mechanical therapies used most frequently for achalasia are pneumatic dilation (PD) and surgical myotomy. For PD, a large-diameter balloon is passed through the mouth, positioned at the LES, and inflated abruptly with the intent of tearing the LES muscle.

Address correspondence to: Stuart Jon Spechler, M.D., Chief, Division of Gastroenterology, VA North Texas Healthcare System, 4500 South Lancaster Road, Dallas, Texas 75216, Telephone: 214-374-7799, Fax: 214-857-1571, SJSpechler@AOL.com.

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Substantial differences in methods for performing PD and criteria for therapeutic success confound direct comparisons among reports, but studies published in the last dozen years or so using Rigiflex balloon dilators describe good to excellent results for PD in 70% to 80% of patients.[2–4] For myotomy, the surgeon weakens the LES by cutting its muscle fibers using a modification of an operation described by Ernst Heller in 1913. Heller myotomy is now performed laparoscopically and, since LES disruption can cause reflux esophagitis, the myotomy is often combined with an antireflux procedure like Dor fundoplication. Reports of current, single-center series on laparoscopic Heller myotomy (LHM) with short-term success rates that approach 100% have fueled growing interest in the surgical approach.[5] Nevertheless, there has been a dearth of prospective trials comparing the safety and efficacy of PD and surgical myotomy.[6,7]

In this issue of the *Journal*, Boeckxstaens *et al.* report the results of a European, multicenter, randomized trial of PD *vs.* LHM with Dor fundoplication for 201 patients with achalasia.[8] Therapeutic success, the primary outcome, was defined as a drop in the Eckardt score (a composite symptom score ranging from 0 to 12 based on the severity of dysphagia, regurgitation, chest pain and weight loss) to 3 or less. Patients were followed for a mean of 43 months, and there were no statistically significant differences in success rates between the treatment groups (90% and 86% for PD vs. 93% and 90% for LHM at 1 and 2 years, respectively). Complications included esophageal perforation in 4.3% of the PD group, and mucosal tears in 12% of the LHM group; there were no procedure-related deaths. At one year, there were no significant differences between the groups in the frequency of abnormal acid exposure by esophageal pH monitoring or in the frequency of reflux esophagitis by endoscopy.

This well done, randomized trial declares no clear winner - PD and LHM have similar safety and efficacy, at least for several years. In deciding between these two procedures, clinicians and patients should consider a number of factors. First, the PD protocol used in this study was rigorous, involving up to three sets of balloon dilations over a period of >2 years, with individual sets including as many as three separate dilation sessions; each such session generally means a day lost from work. In contrast, the inconvenience of LHM is primarily “up front,” involving the operation itself and the postoperative recovery. Another consideration is that the esophageal perforations that complicate PD in >4% of cases result in hospitalization, which may be lengthy, and may require emergency surgical repair with an open (rather than laparoscopic) procedure. The mucosal tears that complicate LHM in 12% of cases, in contrast, usually have no clinical sequelae because they are detected and repaired immediately during the operation. Next, one must consider that PD and LHM in this study were performed by very experienced gastroenterologists and surgeons. It is not clear that their excellent results can be reproduced in community practices, and the local availability of an expert practitioner should weigh in the therapeutic decision. Finally, the mean follow up for this study was only 43 months. The beneficial effects of both PD and LHM may deteriorate over time, and the long-term outcomes of this study remain to be determined.

Recent studies suggest that it may be possible to predict therapeutic outcomes in achalasia based on esophageal motility patterns demonstrable by high-resolution manometry.[9] In the future, those patterns might be used to guide the decision between PD and LHM. For now,

this study by Boeckxstaens *et al.* shows that either option is reasonable, and that the choice between the two should be based on patient preferences and local expertise. For those who remain undecided after a detailed discussion of potential risks and benefits, my preference is for PD, primarily because it is clear what to do if PD eventually fails - that is the time for surgery. Data on what to do if LHM fails are very limited.[10] The safety and efficacy of rescue PD in this setting are not clear, and esophageal re-operations are notoriously difficult. Perhaps the road to Heller myotomy should be paved with good distensions.

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