Evaluation and Treatment of Primary Esophageal Motility Disorders

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Achalasia, diffuse esophageal spasm, and nutcracker esophagus constitute the main primary esophageal motility disorders. During the past decade major progress has been made in understanding their pathophysiology and in the ability to establish a precise diagnosis. In addition, minimally invasive surgical intervention has radically changed the therapeutic approach, and thoracoscopic or laparoscopic myotomy is probably the best treatment for most patients.

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Achalasia, diffuse esophageal spasm, and nutcracker esophagus are the principal named primary esophageal motility disorders. They are characterized by esophageal dysmotility, which is responsible for the symptoms, in the absence of other diseases such as gastroesophageal reflux, connective tissue disorders, and cancer. Because of differences in clinical presentation, diagnosis, and treatment, achalasia will be discussed separately from diffuse esophageal spasm and nutcracker esophagus.*

Achalasia

Esophageal achalasia is characterized by the absence of primary esophageal peristalsis and a hypertensive lower esophageal sphincter (LES) that fails to relax appropriately in response to swallowing (Table 1).

Clinical Presentation

Because the pump function of the esophageal body is gone and the outflow resistance is increased by a hypertensive, nonrelaxing LES, 90% of patients with achalasia seek medical attention because of dysphagia and regurgitation.[1] Chest pain and heartburn are the presenting symptoms in the remaining 10%.[1] Weight loss may be present, but it often is not.

Diagnosis

Radiology. A barium esophagogram is indicated in all cases. It helps differentiate between achalasia ("bird beak" appearance) and other diseases, such as diffuse esophageal spasm ("corkscrew" esophagus), and esophageal cancer (irregular narrowing, with ulceration or intraluminal defects or both) (Figure 1). When cancer of the gastroesophageal junction is suspected, a computed tomographic (CT) scan sometimes provides better definition of a lesion of this area.

Endoscopy. Endoscopy is an essential part of the workup of dysphagia to rule out esophageal cancer. Endoscopic ultrasonography can be done when an intramural tumor is thought to be present at the gastroesophageal junction. In patients with achalasia, the esophageal mucosa is usually normal, but it can be inflamed as a result of the stasis of food or gastroesophageal reflux. No fixed stricture is found by endoscopy, but resistance to the passage of the endoscope into the stomach may be slightly increased.

Esophageal manometry. Esophageal manometry is essential to confirm a presumptive diagnosis of achalasia (see Table 1). Because stasis of food is frequently present in the distal esophagus, it is better to have the patient on a liquid diet for a couple of days before the test, which helps avoid aspiration and allows a better pressure recording to be made during the study. Dilatation or tortuosity of the esophagus is not a contraindication to the test, for the manometric catheter can be easily passed into the stomach under fluoroscopic guidance in all patients.

pH monitoring. Traditionally, pH monitoring has not been considered part of the routine workup of patients with esophageal achalasia on the assumption that gastroesophageal reflux does not occur in the presence of a hypertensive LES. Recent studies have shown, however, that gastroesophageal reflux is present in as many as 20% of untreated patients with achalasia,[2] and it is even more common following medical or surgical treatment. Furthermore, reflux is asymptomatic in many patients,[3,4] even though it may be producing changes that eventually result in esophageal stricture.[5] Thus, routine pH monitoring before and after treatment is indicated to detect abnormal

*See also the editorial by G. Triadafilopoulos, MD, "Primary Esophageal Motility Disorders: Incisive Decisions," on pages 289–290 of this issue.

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ABBREVIATIONS USED IN TEXT

CT = computed tomography
LES = lower esophageal sphincter
UCSF = University of California, San Francisco

reflux, and the results may affect the choice of subsequent treatment. For example, a laparoscopic myotomy with an antireflux procedure is the only treatment that simultaneously addresses concomitant dysphagia and gastroesophageal reflux, and esophageal balloon dilatation should not be performed in the face of gastroesophageal reflux, for the outcome can only be worse reflux.

Treatment

Because the cause of esophageal achalasia is unknown, treatment is directed toward relieving dysphagia by decreasing the outflow resistance caused by the dysfunctional LES. Medical treatment, endoscopic treatment (dilatation, the administration of botulinum toxin), and surgical intervention (open and minimally invasive surgery) are the available modalities.

Medical treatment. The use of calcium channel blockers (verapamil hydrochloride, nifedipine) may be considered with the goal of improving the outflow resistance by a direct effect on the LES. Two double-blind, placebo-controlled studies have shown, however, that symptoms persist in most patients, probably because these drugs have such a modest effect on the resting LES pressure and do not improve LES relaxation in response to swallowing.[6,7]

Pharmacologic treatment is of marginal clinical value, and then only in patients with mild symptoms.

TABLE 1.—*Manometric Criteria of Primary Esophageal Motility Disorders*

Disorder	*Diagnostic Findings*
Achalasia	Absent primary esophageal peristalsis; hypertensive lower esophageal sphincter (LES) resting pressure; partial or absent or, more often, absent LES relaxation in response to swallowing
Diffuse esophageal spasm	Simultaneous waves in >20% of wet swallows; normal primary peristalsis; LES*
Nutcracker esophagus	Peristaltic waves with high distal amplitude (>180 mm of mercury) and prolonged duration (>6 sec); LES*

*In either diffuse esophageal spasm or nutcracker esophagus, the LES can be normal or have high pressure, partial relaxation, or both

Endoscopic treatment. Pneumatic dilatation involves inflating a balloon within the gastroesophageal junction, which ruptures sphincter muscle fibers. This treatment was initially popularized by Vantrappen and Hellemans, who reported the relief of dysphagia in 77% of patients and a perforation rate of 2.6%.[8] These good results, however, have not been regularly duplicated by others. For instance, among 79 patients with achalasia who underwent pneumatic dilatation at the University of California, San Francisco (UCSF), Medical Center between 1977 and 1988, good to excellent results were obtained initially in 80% of patients, but there was a 12% rate of esophageal perforation.[9] In addition, 48 months after treatment, only 50% of treated patients had good to excellent results, 30% had symptomatic gas-

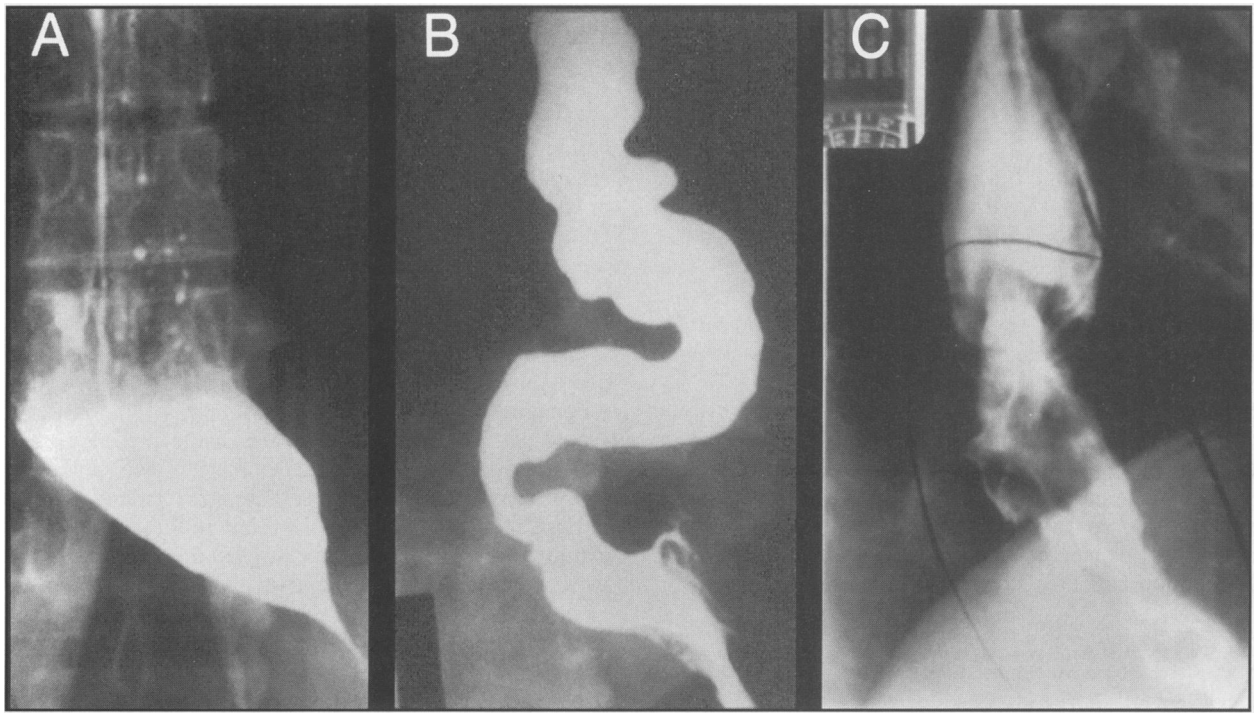

Figure 1.—A barium swallow study shows the radiologic appearance of **(A)** achalasia, **(B)** diffuse esophageal spasm, and **(C)** adenocarcinoma.

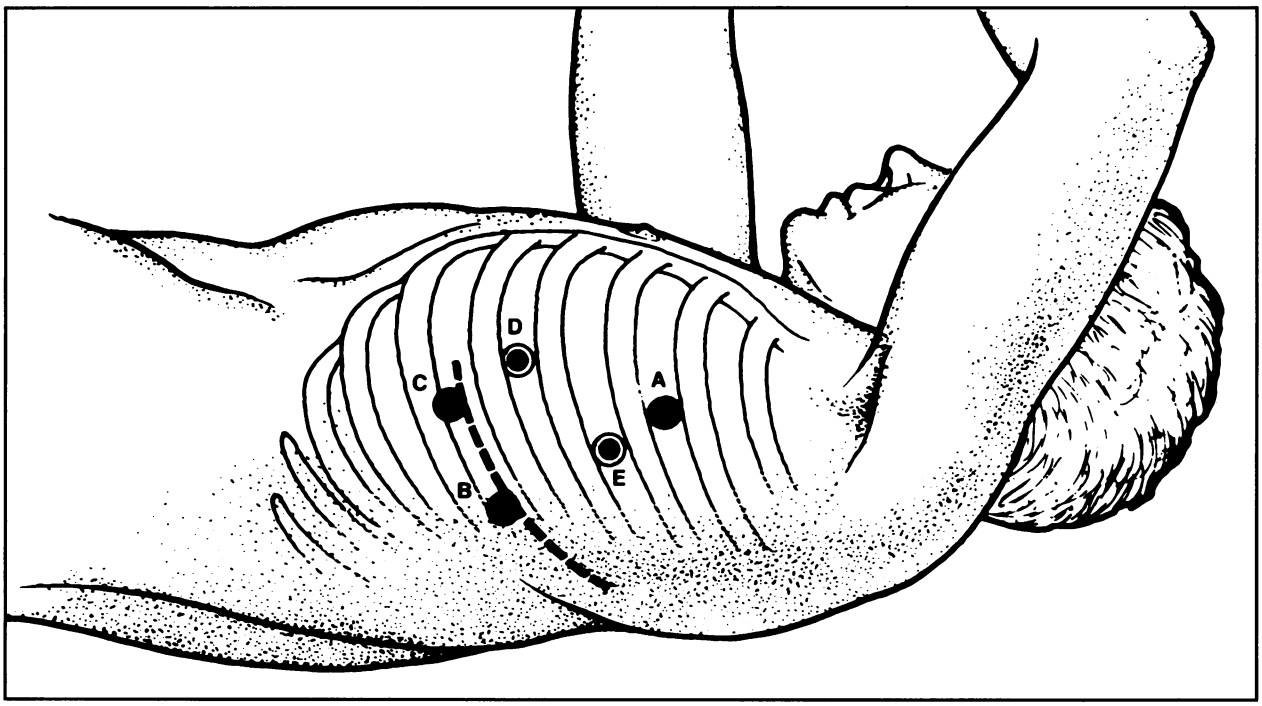

Figure 2.—The position of the trocars for a left thoracoscopic myotomy is shown. The dotted line indicates a posterolateral thoracotomy that was the incision traditionally used for an open myotomy. Incisions **A** and **C** are used for retraction of the lung **(A)** and the diaphragm **(C)**; incision **B** is used for the camera; incisions **D** and **E** are used by the surgeon for the dissection and the myotomy.

troesophageal reflux, and 20% had persistent dysphagia.[9] With the use of the Rigiflex balloon dilator, dysphagia can be relieved in about 85% of patients, but the perforation rate remains around 8%.[10] Perforation caused by balloon dilatation usually requires an emergent thoracotomy to close the hole and to perform a myotomy on the opposite side of the esophagus.

As mentioned, gastroesophageal reflux is another unwanted complication of pneumatic dilatation, and postdilatation gastroesophageal reflux is in fact common, albeit asymptomatic in most patients.[3] Among 22 patients referred to the UCSF Swallowing Center for the evaluation of persistent dysphagia after balloon dilatation, ambulatory pH monitoring identified abnormal reflux in 7 patients (32%), 2 of whom had heartburn.[11] In another study abnormal gastroesophageal reflux was found by pH monitoring in 5 (23%) of 22 patients who underwent technically successful pneumatic dilatation.[12] Esophagitis developed in two of these five patients within six months of the dilatation. Asymptomatic reflux may be dangerous, for the first manifestation may be dysphagia due to stricture formation.

Intrasphincteric injection of botulinum toxin. In patients with achalasia, an imbalance exists between excitatory (acetylcholine and substance P) and inhibitory neurotransmitters (nitric oxide and vasoactive intestinal peptide), which results in the high pressure and lack of relaxation of the LES.[13,14] The rationale for the intrasphincteric injection of botulinum toxin is to restore balance by decreasing the amount of acetylcholine available to affect the sphincter.[15] The botulinum toxin acts by decreasing the release of acetylcholine by nerve endings of the myenteric plexus. The results of a double-blind trial of botulinum toxin injection and placebo showed, however, that 2.4 years after botulinum toxin treatment, only 30% of patients had satisfactory relief of dysphagia despite several botulinum toxin injections being given to those for whom treatment failed.[16] The response rate was better in patients with vigorous achalasia and those who were older than 50 years. Even though botulinum toxin therapy is scientifically attractive in the sense that it addresses the pathophysiology and is simple, its usefulness is limited.[17] There is another drawback that has not previously been reported. We have seen during surgical procedures in patients previously treated with botulinum toxin a moderately severe fibrosis of the muscle, which makes esophagomyotomy more difficult.

Intrasphincteric administration of botulinum toxin should probably be reserved for patients with mild symptoms and concomitant diseases severe enough to contraindicate an operation under general anesthesia.

Surgical treatment. A Heller myotomy involves the longitudinal division of the muscle fibers of the distal esophagus and gastroesophageal junction—that is, the LES. Until a few years ago, esophagomyotomy could be done only through a left thoracotomy or laparotomy. It was sometimes accompanied by the antireflux procedure.[18-24] Now minimally invasive surgery has replaced these open approaches. The operation can be done through a thoracoscopic or laparoscopic approach. The advantages include a short hospital stay, minimal postoperative discomfort, and a return to normal activities with-

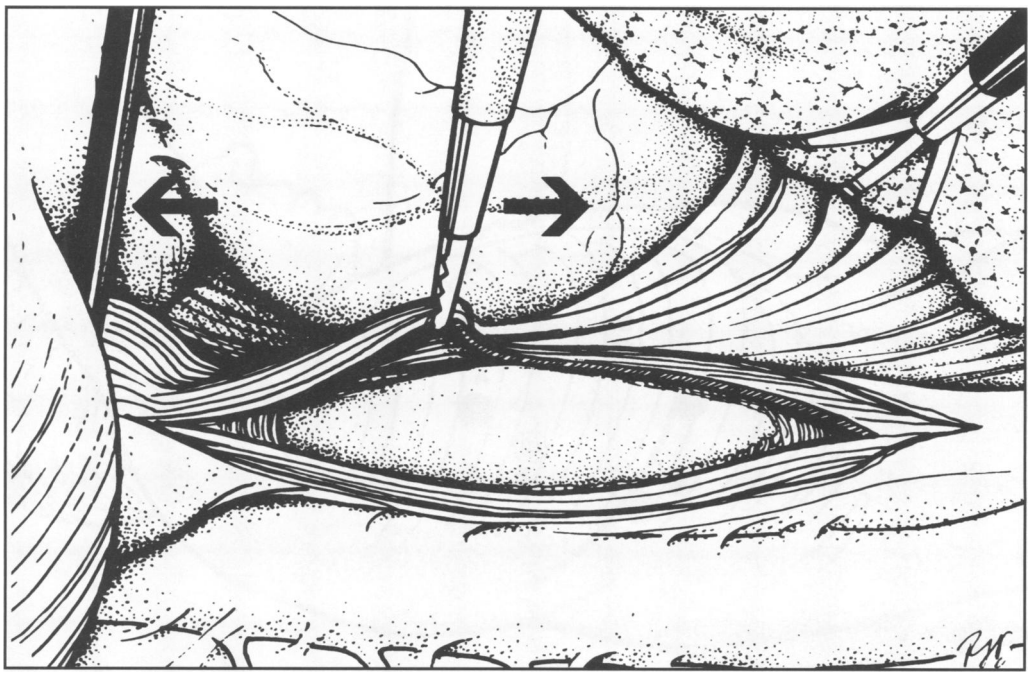

Figure 3.—A Heller myotomy is performed through a left thoracoscopic approach. The myotomy extends for 6 cm of the distal esophagus and includes the proximal 5 mm of the gastric wall.

in a few days instead of weeks.[4,25-27]

A Heller myotomy produces excellent or good results in 90% of patients, with little morbidity and a low mortality rate.[4,20] Long-term follow-up shows that the good results persist over time.[18,20] In a prospective randomized trial comparing the results of esophageal dilatation and open esophagomyotomy, good to excellent results were seen in 65% of patients after pneumatic dilatation and 95% of patients after myotomy and anterior fundoplication.[22] Similar results were reported in two large retrospective studies.[23,24]

Although myotomy gives better results, pneumatic dilatation has usually been the first treatment of achalasia because of concerns about postoperative pain, a long hospital stay, and the duration of postoperative disability. Surgical treatment was principally reserved for patients who had a perforation during dilatation, residual dysphagia after several dilatations, or contraindications to dilatation, such as a previous perforation or an epiphrenic diverticulum. The pendulum is now swinging toward esophagomyotomy as the first choice, for it combines the precision and results of open surgery with the lesser morbidity previously reserved for balloon dilatation.

We performed our first thoracoscopic myotomy for achalasia in January 1991.[25] The myotomy spanned the distal 6 cm of the esophagus, extending 5 mm onto the gastric wall (Figures 2 and 3). The operation was designed to match the procedure recommended by Ellis[18] and co-workers, which for many years had been our approach for open techniques. Between January 1991 and October 1993, we did thoracoscopic Heller myotomies in 30 patients with achalasia.[4] The patients were discharged from the hospital an average of 3.5 days after the operation, required only oral analgesics for pain, and returned to normal activity in 10 to 14 days. Follow-up interviews showed that 26 patients (87%) had good to excellent relief of dysphagia. Postoperative esophageal manometry

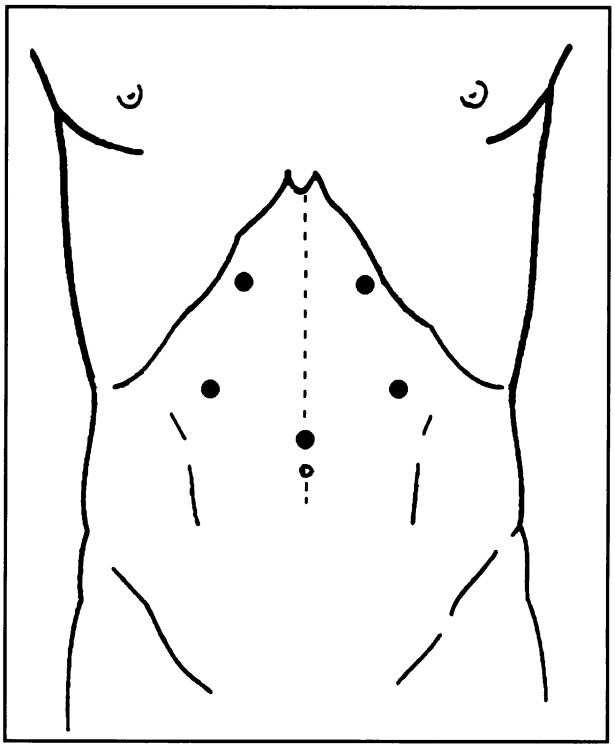

Figure 4.—The position of the trocars for a laparoscopic myotomy is shown. The dotted line shows the incision traditionally used for an open myotomy.

showed a reduction of the LES pressure from an average of 30 mm of mercury preoperatively to about 10 mm of mercury postoperatively, demonstrating that the results were similar to what was accomplished through a thoracotomy. Postoperative pH monitoring, however, identified abnormal gastroesophageal reflux in six of ten patients, only one of whom complained of heartburn. Because of this unexpected finding, we subsequently switched to a laparoscopic approach coupled with an antireflux procedure.[11,26,27] Moreover, the operation was simpler because the patient was supine and did not require a double-lumen endotracheal tube during it and a chest tube postoperatively. Access involves five 1-cm abdominal incisions, similar to laparoscopic antireflux surgery (Figure 4).

Follow-up examinations of the first 24 patients showed good to excellent relief of dysphagia in 22 patients (92%) and only 1 patient (4%) with marginally abnormal reflux (patient's score 19; normal score ≤15).[11] The patients are given liquids the night of the operation and solid food thereafter. They usually leave the hospital the next morning, require no more than oral analgesics for pain, and return to work in 10 to 14 days.

Conclusion

Minimally invasive surgery is now becoming the primary treatment of esophageal achalasia. Pharmacologic treatment and botulinum toxin use have marginal efficacy, and their use is reserved for elderly patients. Dilatation is indicated for patients who wish to avoid an operation and those with previous abdominal and thoracic operations that rule out the use of minimally invasive surgery.

Diffuse Esophageal Spasm and Nutcracker Esophagus

Diffuse esophageal spasm and nutcracker esophagus are characterized by abnormalities in the structure or propagation of primary peristaltic waves and by normal or abnormal function of the lower esophageal sphincter (see Table 1).

Clinical Presentation

Patients with diffuse spasm or nutcracker esophagus seek medical attention because of dysphagia, chest pain, or both.[1] The diagnosis and treatment of these conditions are more difficult than of achalasia. Although a complaint of dysphagia implicates the esophagus, the character of esophageal pain is not different enough from angina pectoris to allow a distinction to be made from the history alone. Consequently, patients with esophageal chest pain often undergo an extensive cardiac workup, including coronary angiography, before being referred for gastroenterologic evaluation.[28]

Diagnosis

Radiology. Barium esophagogram is useful to rule out mechanical obstruction and to demonstrate tertiary contractions, which give the characteristic "corkscrew" esophagus (see Figure 1-B).

Endoscopy. Endoscopy is always indicated, even if the barium study is normal, because gastroesophageal reflux is the most common cause of noncardiac chest pain, and endoscopy may show esophagitis in this situation.[29,30] Obviously, if the patient actually has gastroesophageal reflux, therapy should be directed toward this problem.

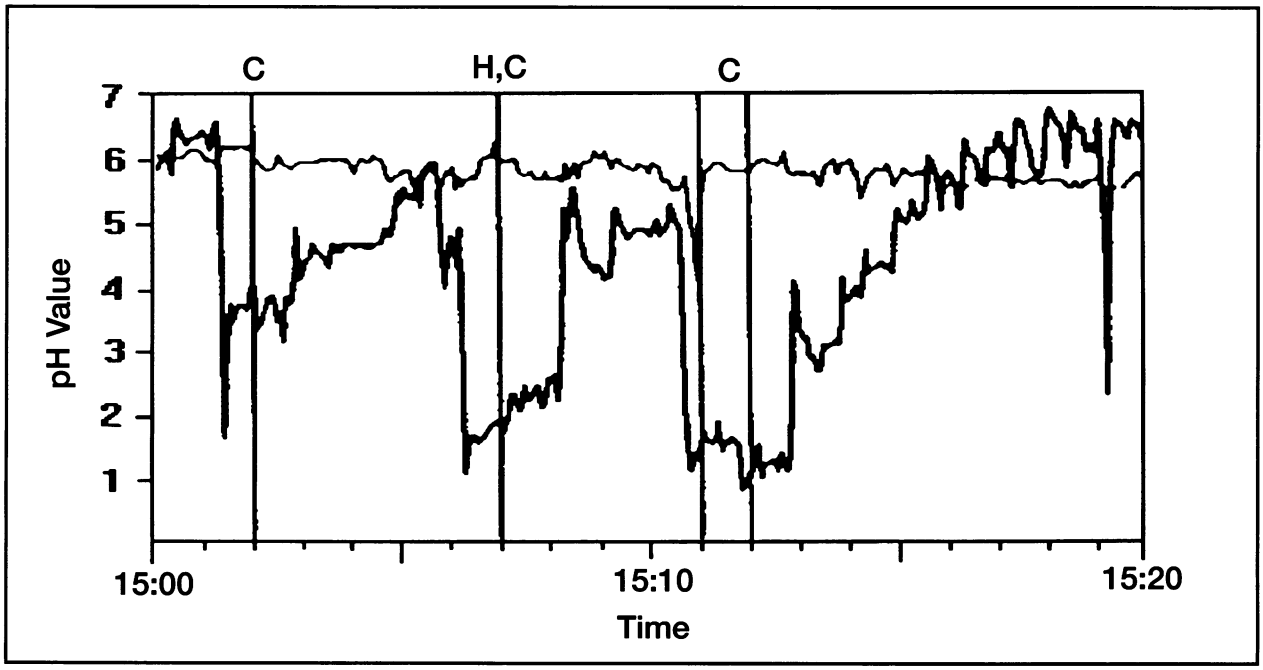

Figure 5.—Ambulatory pH monitoring in a patient with noncardiac chest pain (**C**) shows a temporal correlation between episodes of gastroesophageal reflux and chest pain. H = heartburn

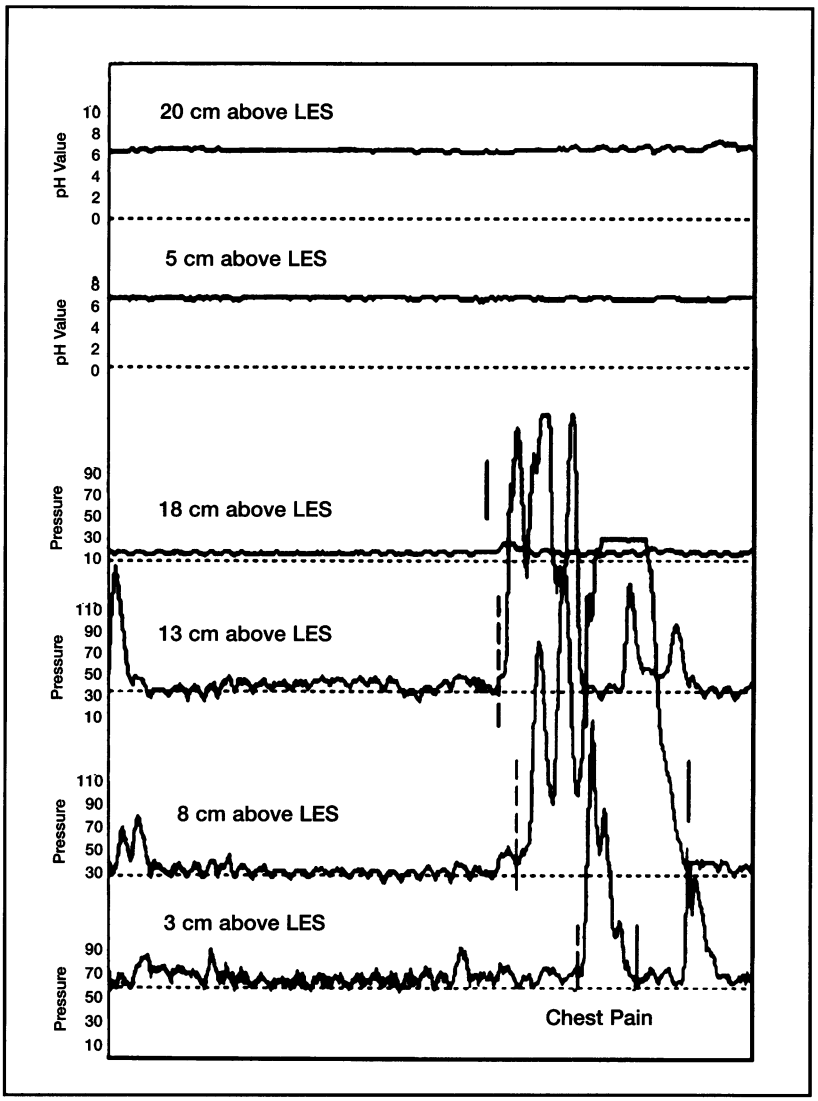

Figure 6.—Ambulatory esophageal manometry and pH monitoring in a patient with noncardiac chest pain shows a temporal correlation between chest pain and abnormal motility (high-amplitude, long-lasting, multipeaked peristaltic waves) of the esophageal body. LES = lower espophageal sphincter.

Stationary esophageal manometry. Table 1 lists the typical manometric findings in patients with diffuse spasm and nutcracker esophagus. Because gastroesophageal reflux can produce similar abnormalities in peristalsis, these findings should be considered diagnostic of a primary motility disorder only when reflux has been excluded by ambulatory pH monitoring. In diffuse spasm and nutcracker esophagus the proximal and distal extent of the muscular dysfunction may vary from one patient to another. The abnormality may affect only the distal esophagus, or it may extend all the way to the thoracic inlet; LES function may be normal or abnormal. The details of the motor disorder are of importance when designing surgical treatment, for the operation must be tailored to the pattern of dysmotility.[4]

pH monitoring. Because gastroesophageal reflux is the most common cause of noncardiac chest pain, ambulatory pH monitoring should be done in the workup of these patients.[29,30] pH monitoring will determine whether reflux is present and whether a temporal correlation exists between episodes of reflux and chest pain (Figure 5). If reflux coincides with pain, treatment of the reflux alone will usually control the pain.[31]

Ambulatory esophageal manometry. In patients with chest pain and no reflux on pH monitoring, a correlation should be sought between the abnormal motor events (that is, high-amplitude, simultaneous, or multipeaked waves) and the pain. Ambulatory esophageal motility can now be recorded for 24 hours or more using catheters that incorporate pH sensors. pH and motility are measured simultaneously (Figure 6).[1,4,28]

Treatment

Medical treatment. The use of calcium channel

blockers is usually the first treatment of diffuse spasm or nutcracker esophagus. Even if the amplitude of peristalsis drops in response to these drugs, however, changes in the frequency and severity of chest pain are no better than following the administration of a placebo.[32-34] In comparing the results of medical and minimally invasive surgical therapy for primary esophageal motility disorders, we found that 74% of patients treated medically had persistent symptoms,[4] and the medically treated patients required the attention of a variety of specialists, many office visits, and periodic hospital admissions.[4]

Surgical treatment. Surgical therapy controls symptoms in 80% of patients.[4,35,36] In one study 34 patients with diffuse esophageal spasm were treated with a myotomy and a short fundoplication through a left thoracotomy.[35] After an average follow-up of eight years, 30 patients (88%) were free of dysphagia, chest pain, or both. Stimulated by the excellent results obtained by the minimally invasive treatment of achalasia, we used similar techniques to treat these disorders. For diffuse esophageal spasm, a left thoracoscopic myotomy extending from the left inferior pulmonary vein to the proximal 5 mm of the gastric wall is the most common procedure because the dysmotility is most often confined to the distal esophagus and the LES. A long myotomy performed through a right thoracoscopic approach and sparing the LES is chosen when the dysmotility involves the entire esophagus, which is typical in nutcracker esophagus. About 90% of patients with diffuse esophageal spasm and 70% of patients with nutcracker esophagus have important benefits in their quality of life.[4]

Conclusion

Medical therapy is mostly ineffective for diffuse esophageal spasm and nutcracker esophagus, as it has little effect on symptoms. Minimally invasive surgery is effective for these conditions, for it relieves dysphagia or chest pain or both in most patients. The keys to success are an accurate diagnosis, a detailed pathophysiologic analysis, and an appropriate operation by a surgeon who has experience with this kind of surgery.

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