Esophageal Motility Testing: Shadow and Substance

During the last decade, study of esophageal motility disorders has been transformed from the quaint, esoteric interest of an occasional odd physiologist to a thriving, dynamic arena of research with hundreds of acolytes thronging to the altar. Much of this change, that has resulted in a proliferation of esophageal manometry laboratories all over the world, owes to the observation that 10–30% of patients undergoing cardiac catheterization for chest pain have normal coronary or trivial lesions that do not explain the chest pain1,2 and their subsequent referral for exclusion of the esophagus as a cause of pain. Apart from these patients with chest pain, manometry laboratories receive referrals for evaluation of symptoms of heartburn and dysphagia as well as of suspected disorders like achalasia cardia or progressive systemic sclerosis.

Esophageal Manometry: What does it tell?

Motility disorders identified in patients undergoing esophageal manometry include well established clinicomanometric entities like achalasia cardia where one finds aperistalsis in the body of the esophagus, hypertensive lower esophageal sphincter (LES) with incomplete LES relaxation, and the scleroderma esophagus with a hypertensive or undetectable LES and no peristalsis in the body except within 3–5 cm of the upper esophageal sphincter.3,5 Entities like diffuse esophageal spasm (DES), nutcracker esophagus or supersqueeze, symptomatic esophageal peristalsis, hypertensive LES and nonspecific esophageal motility disorder have been differentiated mainly on the basis of manometric criteria and may have overlapping clinical features. These conditions merely represent the spectrum of manometric abnormalities identified in a population with chest pain. Correlation between symptoms and dysmotility, or a cause and effect relationship between the two, is unclear. An impressive list of manometric diagnoses may be derived at and, while several workers have described large series of selected patients, few have cared to examine their data of consecutive patients referred to a manometry lab,6,10,11 with a view to ascertaining the yield of manometry in identified subgroups. This lacuna has been filled by Shoemct and Sharma in their paper in the current issue of this Journal.12

Analysing consecutive studies performed over a five year period, Shoemct and Sharma find that of 661 procedures, two-thirds were for dysphagia, a third for chest pain and a fifth for heartburn. While 30% of studies were normal, abnormal motility was recorded in 75%, 62% and 58% respectively of the studies for each of the above indications. Only 10% of all studies identified achalasia and 1% sclerodermia. DES was diagnosed only once (0.15%) in this study while major or minor nonspecific motility disorder was found in 58% of cases. However, one must first examine the widely accepted manometric criteria described by Richter and Castell* for diagnosing DES. These are: simultaneous contractions in >10% of wet swallows; intermittent normal peristalsis; with or without associated findings like repetitive, prolonged, high amplitude or spontaneous contractions; with or without LES abnormalities. If these criteria are applied to Shoemct and Sharma's study, a large proportion of nonspecific motility disorders perhaps would be reclassified as DES. The authors have also not noted any supersqueezer, defined as high amplitude (>190 mm) and/or prolonged duration (>7 sec) contractions in the body of the esophagus, or patients with hypertensive LES, defined as LES basal pressure >50 mmHg, normal peristalsis in the body and normal deglutitive LES relaxation. However, their finding that manometry is more rewarding in the dysphagia patient is corroborated by others.13

Esophageal Manometry: Symptom-Dysmotility Correlation

How does abnormal motility correlate with the patient's symptoms?

Dysphagia correlates best with an elevated LES pressure and incomplete LES relaxation. In about a third of patients with DES, who may not have LES abnormalities, the severity of symptoms (dysphagia and chest pain) correlates best with simultaneous or long duration contractions.4

Chest pain, unlike dysphagia, is more difficult to correlate with motility patterns. There are many pitfalls in the interpretation of manometric data of patients with chest pain. Esophageal motility disorders are demonstrated in only 18–45% of such patients.5 Almost all these patients are asymptomatic at the time of the motility test; thus, demonstration of a dysmotility pattern at this time does not necessarily mean that this pattern would also be present during episodes of chest pain. Conversely, a normal study does not preclude the possibility of dysmotility at the time of symptoms. This problem has been tackled by attempts at provocation of symptoms at the time of testing, using ice water swallows, acid infusion, balloon distension in the esophagus, or injections of ergometrine, pentagastrin, bethanechol or edrophonium.15 Edrophonium injections have been the most accepted form of provocation but identify only an additional 20% of patients with chest pain and dysmotility. Ambulatory 2 hour pH and motility monitoring has been undertaken in an attempt to elucidate the symptom-motility correlation. In a recent study using portable, miniaturized, solid...
state systems for simultaneous esophageal pH and pressure monitoring,14 pH abnormalities (20%) were more commonly associated with chest pain than motility abnormalities (12%); the majority (64%) of episodes of chest pain did not correlate with either abnormality and may have been due to an "irritable esophagus," a concept first proposed in 1986.15 Thus, while esophageal dysmotility does result in chest pain, it is probably not a major cause of this symptom.

Gastroesophageal Reflux Disease

What is the place of motility studies in the patient with gastroesophageal reflux? Manometric characteristics of the LES, like basal pressure, sphincter length, and spasm length below the respiratory inversion point do not clearly discriminate between refuxers and non-refluxers.16 In fact, a substantial proportion of patients with reflux disease have a normal resting LES pressure.17 A significant proportion of refluxers also have associated esophageal motility disorders including DES.18 However, it is not clear whether esophageal dysmotility in reflux disease is the result of acid injury, is the cause of reflux or is another manifestation of a putative defect in vagal innervation of the LES and the esophagus. The following evidences favour the hypothesis of vagal dysfunction:

(1) Inappropriate transient LES relaxation preceding reflux occurs in 82% of reflux episodes and is probably due to defective LES innervation.19

(2) Inappropriate LES relaxation can be triggered by spontaneous non-propagating pressure activity in the esophagus which, in turn, may be due to neural mechanisms.20 Thus, esophageal motility studies have recently shed light on the pathogenesis of reflux disease but have little role in the diagnosis of reflux disease. Indeed, today reflux disease appears to the most common and important esophageal motility disorder.

Esophageal Manometry: The Bottom Line

What really is the practical utility of esophageal motility testing? In several areas esophageal motility testing is invaluable in therapeutic decision-making. In a patient with achalasia, for instance, manometry is valuable in making the diagnosis, especially when clinical and radiologic features are inconclusive. Following balloon dilation or surgical myotomy, completeness of the procedure and need for repeat dilatation can be assessed manometrically. Esophageal dysmotility in scleroderma, CREST syndrome, mixed connective tissue disorder and other collagen vascular diseases can be reliably assessed manometrically and one often identifies these patients by manometry before systemic clinical manifestations are apparent. In patients with DES, manometry can guide choice of treatment options that include medical therapy (nitrates, calcium channel blockers), pneumatic balloon dilatation and, occasionally, long esophageal myotomy. In patients with non-cardiac chest pain, establishing the esophagus as the source of pain and clearly communicating this information to the patient is important. Long term follow-up of these patients has shown that, although they continue to experience recurrent episodes of pain, patients who realise that the esophagus was the source of their pain feel significantly less disabled and require continued physician evaluation less often.21 Finally, as suggested by Sonnenat and Sharma, motility studies performed in refluxers, who are candidates for antireflux surgery, may identify those who are likely to fare poorly after such operations.

References