Esophageal Pain

Dear Sir:

The article by Lacey Smith et al. (1) once more brings to the surface the problem of where, how, and when the symptoms associated with gastroesophageal reflux arise. The surface or epithelium of the lower esophagus, mainly in its lower 10 cm, is where the pain or heartburn arises. Smith et al have added to our knowledge of how this epithelium is stimulated experimentally in relation to the amount and pH of different HCl solutions. They have correctly questioned when the natural pain and symptoms occur in relation to 24-h pH recordings when reflux is defined as a drop in intraesophageal pH to 4 or below and they have shown that only about two thirds of these pain episodes are associated with a fall in pH below 4.

Pain reproduction tests in ulcer disease and esophageal symptoms surface every 10 yr or so as a fashionable research project. The original experiments both in the United States (2) and in the United Kingdom (3) demonstrated some correlation with pain but not reliably. In 1958, Bernstein et al.'s paper (4) was a landmark for esophageal pain; they used 200–600 ml HCl infused through a nasogastric tube but it was not completely reliable. Because epigastric pain was also reproduced in this classical paper, the author embarked on a series of experiments in the 1970s that demonstrated that if a solution of hydrochloric acid with a pH of 1 was perfused into the lower esophagus at a rate of 8–10 ml/min under accurate manometric control and attention was paid to recent pain, i.e., nocturnal waking in the last 1 mo, the test was always positive with a mean quantity of 37 ml (5, 6). Nocturnal pain the night before the test left an even more sensitive mucosa, which was stimulated to produce pain with a mean quantity of 22 ml. On the basis of this epigastric pain reproduction test, the symptoms of patients with a duodenal ulcer were divided into those with epigastric pain, ie those without. Those with epigastric pain had more reflux, a sensitive lower esophagus, and more heartburn. Those with nonepigastric pain had less reflux, a stronger more competent gastroesophageal sphincter (7), an insensitive lower esophagus, and infrequent heartburn. Further research suggested that the radiological and manometric findings bore no reliable correlation with symptoms (8,9). Similarly, in the 25 cases of Lacey Smith et al. (1) gastroesophageal reflux and esophagitis were not always found together.

The best comparison for the site of the gastroesophageal junction pain mechanism is with the ano-rectal junction. Both are junctional sites of the autonomic and somatic nervous systems. Injection of hemorrhoids into the rectal mucosa produces tension but not pain; similarly acid into the stomach does not cause pain. Mistaken injection into the squamous epithelium of the anus is painful. Perfusion of the normal lower esophageal mucosa is usually painless but if it has been damaged by reflux then it becomes sensitive, hence the Bernstein test and the epigastric pain reproduction test. There is no convincing evidence that acid can cause pain by stimulation of the gastric mucosa, for which we all have cause to be grateful.

Lacey Smith et al. have quantitated the Bernstein test by paying attention to where (manometric control), how (pH 1 or 2), and when (the amount, 8–10 ml/min) in a manner similar to the epigastric pain reproduction test. But the test must be made more reliable by paying attention to who. It is the author's experience that many of the patients had not read the textbooks or the results of previous experiments and reacted surprisingly. Occasionally, lower esophageal acid reproduced pain in the left shoulder and down the outer arm. There was often discrepancy between symptoms, their recent exacerbation, and the perfusion test. Then there were the rarities who had a positive test before duodenal ulcer surgery, then had successful surgery with vagotomy and pyloroplasty with total elimination of symptoms but who had a positive test 1 yr later, presumably demonstrating a very sensitive mucosa. The other interesting group consisted of those who had a positive test before a partial gastrectomy, were positive for 3 to 4 wk afterwards in spite of having had their ulcer removed, and who never had another positive test. That phenomenon could be explained by recovery of the esophageal mucosa from previous attack, but could not be explained by pain arising from the ulcer itself.

It must be hoped that this article persuades people to use the Bernstein test further and to examine symptoms more accurately because the search for a duodenal ulcer or hiatus hernia by radiology, endoscopy, or manometry is a search for the wrong end point. The patient is relatively uninterested in the hole in his duodenal mucosa or his displaced gastric mucosa. He has symptoms that need analyzing, understanding, and treating. Now that the pain reproduction test has been refined, the anomalies can perhaps be explained by individual variations in pain threshold. We must learn more.

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