episode of chest pain during the monitored period as occurred in 22 of the patients in the present study. In this situation the reflux status of the patient can be determined, but no conclusion can be drawn concerning the relationship of the chest pain to a reflux episode other than the probability that it is unrelated if the patient does not have reflux. The authors have found that monitoring for a more prolonged period or during exercise can be helpful in this situation. Recently, the development of a portable esophageal pH monitoring device has made the monitoring of outpatients possible. It is hoped that such equipment that can monitor the patient during activities of daily living will help to clarify the etiology of effort precipitated chest pain.

The clinical situation becomes more difficult to sort out in patients with both conditions, *i.e.*, sufficient coronary artery pathology to account for ischemic chest pain and abnormal reflux on 24-hour esophageal pH monitoring. In this situation the chest pain may be caused by reflux, myocardial ischemia, or both. Treatment of the angina with long-acting nitrates can reduce the distal esophageal sphincter pressure and make the reflux worse. If this is not recognized, the reported increased incidence of chest pain, or a worsening in the severity of the chest pain, can be interpreted as a medical failure to control the angina and initiate a recommendation for coronary bypass surgery. The subsequent failure of surgery to give complete symptomatic improvement under these conditions is often accredited to inadequate revascularization until it is recognized that the patient has another disease. Esophageal evaluation should be performed in any patient who does not have a satisfactory explanation for persistent postoperative symptoms after coronary artery bypass. If possible, both abnormalities should be recognized before operation and a combined procedure performed.

This study suggests that determining the reflux status of the patient with chest pain and normal coronary angiography can uncover a treatable etiology about 50% of the time. In the present study fifteen such patients had total abolition of their chest pain and esophageal symptoms by either surgical or medical therapy. Of the remaining eight patients, seven were treated medically, and their response to antacid therapy was variable. Whether this represents a true failure of medical therapy to control reflux, or excludes an esophageal origin for the pain, cannot be determined until the response to antireflux surgery has been evaluated in these patients. It is probable that medical therapy, since it is administered intermittently and designed to alleviate symptoms rather than correct the underlying abnormality, may not be as effective as surgery in providing constant protection against reflux and its consequences during the activities of daily living.

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Discussion

DR. HIRAM C. POLK, JR. (Louisville, Kentucky): Those of us who see patients with complicated esophageal problems are seeing a steady trickle of patients who are precisely characterized by the signs and symptoms that Dr. DeMeester has talked about. Very often, they will have been worked up in detail, and have all the usual tests indicating

coronary ischemia. On occasion you will see patients who have had coronary abnormalities, undergone operative repair, and persist with their chest pain.

I think this is an important kind of differentiation. Indeed, it would be best, perhaps, if this material could be presented before our colleagues in medical cardiology, so that they might at least consider this option for more of their patients.

Really, I do believe Tom has made an important point about the 24-hour pH monitoring. We have cared for 11 of these patients, and it is my conviction, working on clinical grounds alone, that we have probably done the wrong thing for two of them. This is a difficult kind of discrimination. You simply cannot do it with a clinical test, or by clinical assessment alone.

On the other hand, I think this kind of objective measure can be extremely helpful, and think that we would not tackle this kind of case again without the objective data that Dr. DeMeester showed today.

I suspect I know the answer, but I would like to ask a single question. Does the monitoring in a relatively inactive state—which is, by definition, what you usually do with the 24-hour pH monitoring business—produce a lower incidence of positive tests, certainly, than you might see if you were able to study these patients while they were under conditions of exercise?

DR. HAROLD C. URSCHEL, JR. (Dallas, Texas): I would like to compliment Dr. DeMeester and his associates on emphasizing the importance of persisting in the search for a diagnosis in these patients with chest pain and normal coronary arteries.

In 1973 we presented a group of 45 patients with normal coronaries, but "angina-like" pain that we termed pseudoangina, and who had cervical ribs, or evidence of vascular compression at the thoracic outlet. Eighty per cent of these were improved by medical or surgical therapy.

We also noted in that paper that 20 other patients had significant gastroesophageal reflux and pseudoangina, as well as one patient whose pseudoangina was secondary to a superior sulcus tumor.

The mechanism that we proposed for the pain in the chest with thoracic outlet compression was that, instead of just the usual $C8T^1$ nerve route compression that we usually observe, the deep nerve fibers that travel the sympathetic plexus were also compressed in a certain percentage of cases, and the pain was appreciated as being in the chest because of the passage into the sympathetic ganglia of T-2 through T-5.

Since then, we have assessed over 400 patients with normal coronary angiograms, and found a significant cause of psuedoangina outside of the heart in approximately 90%. Of these, two-thirds have been related to thoracic outlet, and one third to gastroesophageal reflux.

We have noted another interesting subset, and that is the postoperative coronary patients who develop angina-like symptoms who have a thoracic outlet because of the spread of the sternal retractor. Before the conduction studies were available, these patients often had ulnar nerve transfer at the elbow because they thought there was compression lying on the operating table. Almost all of these patients improved with only conservative treatment.

We feel that people with bona fide angina-like chest pain have a cause that can usually be diagnosed, and it is extremely important for the psychologic integrity of the patient that the physician persist in establishing an organic etiology.

DR. ROBERT E. CONDON (Milwaukee, Wisconsin): A couple of years ago, before another forum, we presented our experience with somewhat over 300 patients with reflux esophagitis. In presenting our material, we indicated it had been our experience that elegant, complicated, high-technology work-ups for patients with relatively straightforward symptomatic reflux confimed by esophagoscopy probably was not warranted.

But, we recognized then and we agree that there are subsets of patients with atypical presentations and atypical symptoms in whom all of this high technology diagnostic effort is not only indicated but certainly is of benefit in sorting out complicated problems. I think that 24-hour pH monitoring probably is essential in the sorts of patients Dr. DeMeester has been reporting to you today.

I certainly also second the thrust of Dr. Polk's and Dr. Urschel's remarks that perhaps this presentation has not been made to the most acute audience. I know that we have appreciated this report greatly, but I think the people we need to get the message to are our physician colleagues, particularly cardiologists.

Within the last few months, I have had three patients come to me to see if something could be done about their chest pain. The story in all of them was that they had had a complete cardiological work-up, stress test, and coronary angiography, without the demonstration of coronary disease. Nothing further had been done. Obviously, something further should have been done. All of these patients had demonstrable reflux and esophagitis, and have responded very, very well symptomatically to fundoplication.

DR. TOM R. DEMEESTER (Closing discussion): I wish to respond to Dr. Urschel first. We concur with him that in patients who do not have an explanation for their chest pain, thoracic outlet syndrome, skeletal deformities, and cervical osteoarthritis should be looked for, particularly if the patient does not demonstrate reflux on 24-hour esophageal pH monitoring. We think that reflux, however, accounts for the chest pain in most of these patients since each patient in the study had a skeletal survey to exclude these abnormalities.

In response to Dr. Condon's comments, we are becoming impressed—maybe it is because of the type of patients we see in our practice—that even in the presence of endoscopic esophagitis, one cannot always proceed to antireflux surgery expecting a good result. Esophageal surgery is different than most surgery performed. The goal of esophageal surgery is to restore function, and not to extirpate an organ. As a consequence, it is very important for the surgeon to have a full understanding of esophageal function. It is because we do not fully understand what we are trying to accomplish in the operating room that the result of esophageal surgery can be less than adequate. On the basis of this, we obtain esophageal function studies in almost all patients, and encourage surgeons to become physiologically oriented as well as procedure oriented. They should understand the abnormal physiology of the esophagus prior to the operative repair. We agree, however, that 24-hour esophageal pH monitoring is particularly useful for evaluating patients with reflux-induced respiratory symptoms or chest pain, and for patients with recurrent postoperative reflux symptoms.

In regard to Dr. Polk's statement, it is indeed true that it would be helpful to be able to study these patients during their daily activity and not while hospitalized. We reported about half of the patients that were studied did not experience chest pain during the monitoring period. This may be because they were at rest or partially immobile while the 24-hour pH test was being done. We have studied eight patients with EKG stress testing and simultaneous esophageal pH monitoring. In three we have observed the onset of chest pain occur during a reflux episode. Two of the three had a positive 24-hour esophageal pH monitoring test at rest, and in one the test was normal at rest. Recognizing the importance of what Dr. Polk has pointed out, we have developed a portable esophageal pH monitoring system. It contains a touch-tone keyboard of various symptoms, and the patient simply punches in the symptom he experiences, and it is recorded along with the pH. This makes the recording of symptoms easy for the patient and determining the relationship between the symptoms and a reflux episode, easy for the test reader. Since the system is completely portable, 24-hour esophageal pH monitoring can be performed as an outpatient from an office setting. The unit is returned the next day, and the stored record is read and analyzed by a computer.