Esophageal Manometrics in Patients with Angina-Like Chest Pain

DOUGLAS L. BRAND, MD, DORIS MARTIN, and CHARLES E. POPE, II, MD

Fifty-eight patients with angina-like chest pain had esophageal manometric testing. Forty-three had no evidence of coronary artery disease at the time of referral or at subsequent contact; 15 patients were proven to have coronary artery disease. High-amplitude contraction waves were the most frequently found manometric abnormality (15 patients). Less frequent were increased duration of contractions, achalasia, and diffuse esophageal spasm; the latter was present in only 3 patients. An approach to the interpretation of information obtained during manometry is presented. Using this approach, the esophagus was strongly implicated as the cause of the pain in 20 patients and was suspect in 18 others. Seven patients had results that exonerated the esophagus, and in the 13 remaining individuals, the esophagus was probably not the offending organ.

Chest pain clinically indistinguishable from angina pectoris of coronary artery disease (CAD) can arise from the esophagus. With the increased use of coronary arteriography, it has been recognized that some patients clinically thought to have CAD have perfectly normal coronary arteries demonstrated angiographically. This has led to an increased number of referrals to the esophageal motility laboratory in an attempt to ascertain the cause of chest pain. It is suggested in the literature that patients with chest pain of esophageal origin will have either diffuse spasm (1) or reflux (2). This paper will present the manometric findings in a series of patients referred to the esophageal laboratory for evaluation of chest pain mimicking that of CAD.

MATERIALS AND METHODS

Patient Selection

Between March 1972 and September 1976, 686 patients were referred for esophageal evaluation. Chest discomfort of varying types had been experienced by 109 individuals. Of these, 58 had chest pain which had the clinical characteristics of angina pectoris, defined as substernal chest pain that was exertional and/or radiated to the neck, jaw, or arms. These patients, subdivided into three groups, form the population under study.

Group I, consisting of 43 patients, was felt not to have significant CAD on the basis of normal coronary arteriograms (17 patients), normal exercise tolerance tests (16 patients), and/or at least 20 months of follow-up after esophageal manometric testing with no clinical or EKG evidence of coronary artery disease (6 patients). Of the four remaining patients, one died of non-CAD causes, two had a short follow-up period without evidence of CAD, and one was lost to follow-up.

Group II consists of 9 patients known to have CAD at the time of study because of previous myocardial infarcts (2 patients) and/or positive coronary arteriograms (8 patients) showing more than a 50% lesion. These patients were referred because of onset of an additional type of chest pain or worsening of their angina.

Group III consists of 6 patients who were clinically indistinguishable from patients in Group I at the time of manometric evaluation but who subsequently developed unequivocal signs of CAD (myocardial infarction in one patient, positive arteriograms in five).

Clinical details are given in Table 1.

Methods

Early in this study, patients were evaluated by use of a catheter assembly which measured peristaltic aboral force ("pull") and force of closure of the lumen

From the Medical Service, Veterans Administration Hospital, and the Department of Medicine, Division of Gastroenterology, University of Washington, School of Medicine, Seattle, Washington.

Address for reprint requests: Dr. Douglas L. Brand, Medical Service, Veterans Administration Hospital, 4435 Beacon Avenue South, Seattle, Washington 98108.

TABLE 1. CLINICAL CHARACTERISTICS OF 58 PATIENTS WITH ANGINA-LIKE CHEST PAIN

	Age (yr)	*Sex M/F*	*Dysphagia*	*Heartburn*	*Response to antacids*	*Response to nitroglycerin*	*Exercise tolerance test (Pos/Neg)*	*Coronary angiography (Pos/Neg)*	*Outcome*
Group I 43	51.8 (26–71)	28/15	23/43	18/43	11/35	14/30	3*/25	0/17	Better 14 Same 9 Worse 6 No data 14
Group II 9	53.3 (48–63)	5/4	3/9	2/9	0/9	3/9	4/0	8/0	Better 5 Same 3 Worse 0 No data 1
Group III 6	52.3 (36–63)	5/1	2/6	3/6	1/4	5/5	0/3	5/0	Better 2 Same 1 Worse 2 No data 1

*The 3 patients in group I with positive exercise tolerance tests had negative coronary angiograms.

("squeeze") (3, 4). Experience showed that no abnormalities in "pull" were found without associated abnormalities in squeeze, so most patients were studied with an assembly consisting of a pH probe and three infused catheters, two at the same level, and one 5 cm distal. A few were studied with an assembly consisting of four infused catheters, one proximal, two 5 cm distal, and one 10 cm distal. Infusion was performed with a pump delivering 2.4 ml/min, until 1975, when a pressurized system which delivers small quantities of fluid per unit time was substituted for the pump (5).

The recording catheters in use were initially placed in the stomach, and the results of three pull-throughs of the lower esophageal sphincter were averaged to estimate LES pressure. Then five swallows were recorded at 2-cm intervals from the lower esophageal sphincter to the upper sphincter. Each swallow was measured for its amplitude (squeeze), duration (time from rise from baseline pressure to return to baseline pressure), and velocity of peristalsis (time from peak of wave from the proximal catheter to the peak of the wave from the distal catheter, divided into the distance between catheter tips). Mean values for each patient at each level were compared to mean values from 11 control subjects (ages 23–70, $\bar{X} = 41$) without gastrointestinal symptoms or evidence of diabetic or alcoholic neuropathy. The mean value from a patient had to exceed two standard deviations from the mean of the control subjects in order to be considered abnormal. Diffuse spasm was defined as an elevation of the resting baseline more than 5 mm with simultaneous contractions during the period of baseline elevation.

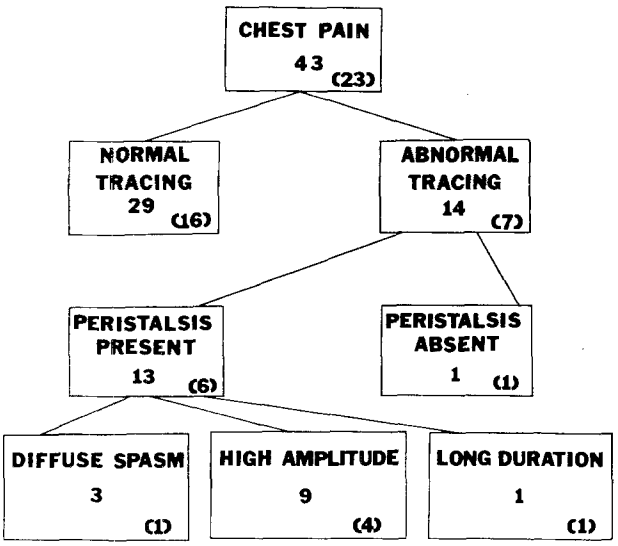

Fig 1. Manometry results in 43 patients with no evidence of coronary artery disease (group I). Number of patients with dysphagia shown in parentheses.

In the patients in which a pH probe was used, spontaneous reflux was sought before the peristaltic waves were measured. If no reflux was observed, 300 ml of 0.1 N HCl was placed in the stomach and reflux was again sought. Any relation between documented episodes of reflux and the patient's pain was noted.

In November 1976, the patients were contacted to ascertain the subsequent clinical course of their chest pain. Of the 58 patients, 41 were successfully contacted, 2 had died, and 15 could not be traced.

RESULTS

Figure 1 is a flow chart of the manometric results in group I (no evidence for CAD). It can be seen that 29 patients had perfectly normal manometric tracings. Six of these had their typical chest pain during the study, thus making a motor abnormality unlikely as a cause of their chest pain. Of the 29 patients with normal tracings, 16 gave a history of dysphagia.

Fourteen patients had some manometric abnormality. One patient, known to have active achalasia and a previous myotomy, demonstrated aperistalsis. Of the remaining 13, three had at least one episode of diffuse spasm, each episode being associated with the patient's characteristic chest pain (Figure 2). There were 9 patients with increased amplitude of the force of contraction, always found in the lower third of the esophagus and occasionally present proximally as well. In three of these patients who experienced their typical chest pain during the manometric examination, the amplitude of the peristaltic wave bore a direct relationship to the intensity of the chest pain. In addition, a decreased velocity and increased duration were frequently found in the high-amplitude patients, although not always to a statistically significant degree (Figure 3). One patient had a significantly prolonged duration of his peristaltic waves as his only abnormality.

Response to pharmacologic agents was variable. Of the 58 patients, 12 reported help from antacids, and 22 from nitroglycerin, and 5 were helped by both. Two patients with diffuse spasm tried nitroglycerin; neither benefitted. The patients with abnormalities in "squeeze" had an inconsistent response to both nitroglycerin (3/6) and antacids (2/9).

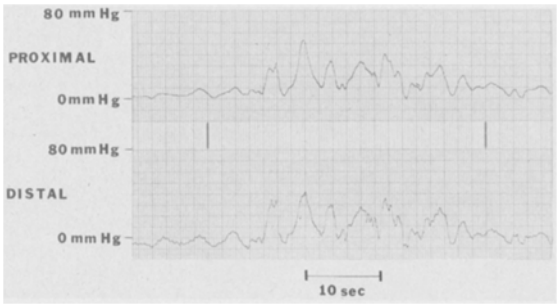

Fig 2. Manometry record of patient with diffuse esophageal spasm taken during episode of pain. The baseline pressure is elevated in both proximal and distal catheters and simultaneous contractions occur. Peristaltic waves preceded and followed the segment of record illustrated.

Among the 9 patients in group II (known CAD at the time of manometry), four had high amplitude; two of these had pain during manometry. In group III (subsequently proven to have CAD), two had high amplitude with no pain during manometry, and one with achalasia had no peristalsis. Group II patients had an inconsistent response to nitroglycerin; conversely, all 5 patients in group III on whom information was available responded to this medication.

Mean lower esophageal sphincter pressure of the 54 patients tested was 17.5 ± 11.1 mm Hg. Of the 17 Bernstein tests performed, six were positive. Reflux as shown by a pH probe was present in 17 of 28 patients tested. None of these test results was associated with manometric abnormalities.

Of the 58 patients in the study, follow-up was available on 29/43 in group I, 8/9 in group II, and 4/6 in group III; the mean follow-up period was 19.8 months. Among those with no evidence for CAD (group I), 14 reported their pain to be significantly better. Four of these patients had undergone surgery (one myotomy in a patient with diffuse spasm, one myotomy with a Belsey anti-reflux procedure in a patient with high wave amplitude, and two anti-reflux procedures in patients with reflux but normal manometries) to which they attributed their improvement. Nine others reported no change in their pain, and the remaining six said their pain had worsened. There was no relation between pain outcome and initial response to antacids or nitroglycerin. Among the 15 patients with CAD (groups II and III), follow-up in 13 patients yielded seven who had improved; one was a patient with high amplitude

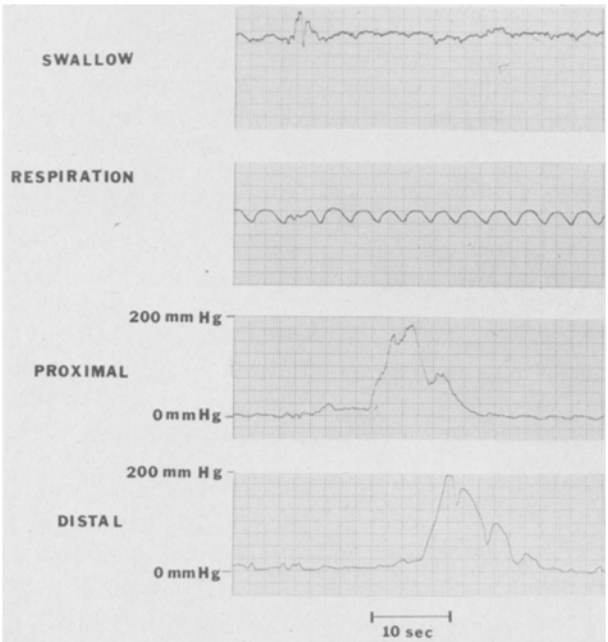

Fig 3. Manometry record of patient with high-amplitude contraction waves (200 mm Hg). Decreased velocity (0.7 cm/sec) and increased wave duration (12 sec) are also present.

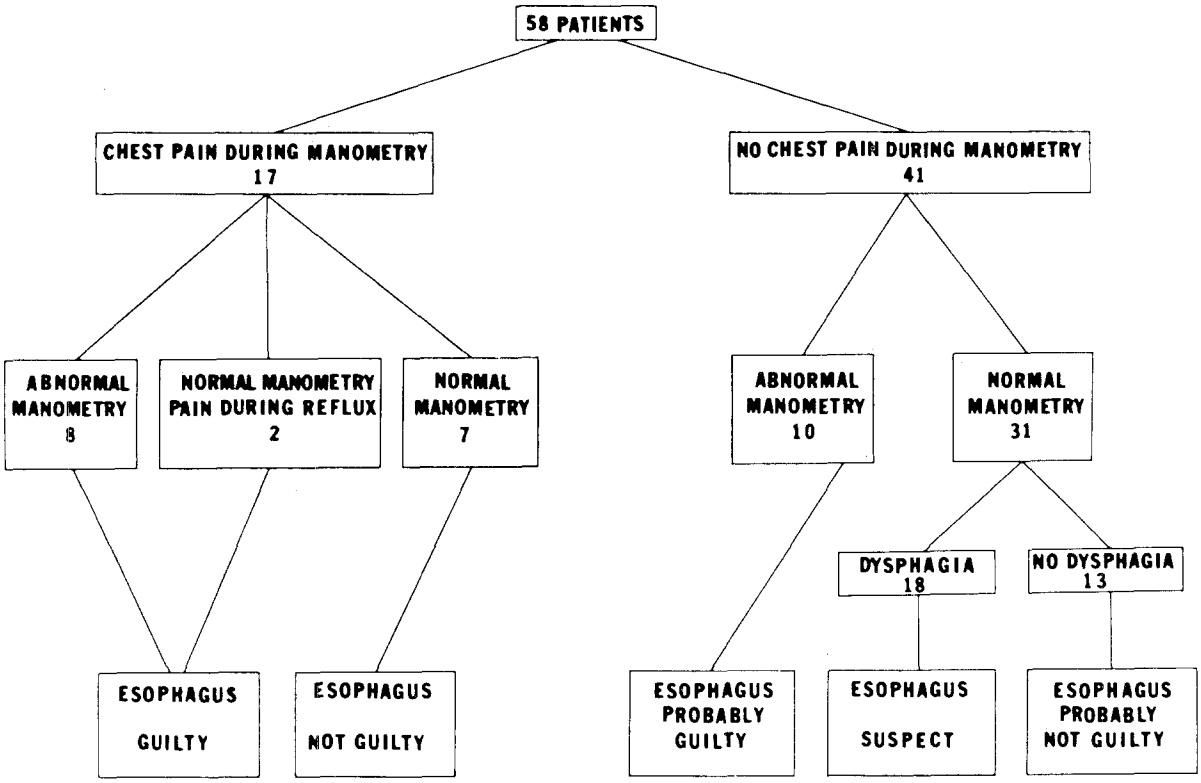

Fig 4. Flow diagram for interpretation of manometry results in patients with chest pain.

who improved after myotomy. Four patients were the same, and two had worse pain. There was no association of symptoms, manometric findings, or length of follow-up with pain outcome.

DISCUSSION

What evidence is necessary for the esophagus to be implicated as a cause of chest pain? A definitive statement can only be made when the patient has a typical attack of chest pain during a manometric examination. If a characteristic pattern appears (diffuse spasm) or the intensity of the pain can be predicted by watching the amplitude of the peristaltic waves, then a clear-cut case can be made for an esophageal origin of the pain. Only 8 patients of the 58 examined fulfill these criteria (Figure 4). In addition to these patients with manometric abnormalities, 2 patients were observed to have pH-probe-proven reflux at times of pain; no abnormal manometric pattern was seen.

Seven patients had an attack of their typical chest pain during a completely normal manometric test. It seems reasonable to exclude an esophageal origin of pain in those individuals. Medical attention might then shift to other areas of investigation.

Ten patients demonstrated abnormalities of amplitude or peristalsis during a pain-free period. These abnormalities often resemble the tracings obtained from other patients during pain attacks, so it seems reasonable to at least suspect the esophagus as a source of pain in these individuals, especially if they suffer from dysphagia.

There is also a group of 18 patients whose manometric tracings are completely normal during an asymptomatic period, but who also complain of dysphagia. It is possible that if these patients were studied during a period in which they suffered from pain, abnormalities might be detected. This is often technically impractical, especially if the attacks of chest pain are infrequent and of short duration. However, the sequence of a completely normal manometric tracing during an asymptomatic period followed by an abnormal tracing during an episode of chest pain has occurred in at least 3 patients, including one of the authors (CEP, II).

Finally, there were 13 patients with no pain during the manometry, normal manometric tracings, and no dysphagia. It seems unlikely that the esophagus is involved in causing chest pain in this group.

The type of abnormality seen manometrically is also of interest. Although diffuse spasm is considered the most commonly occurring manometric abnormality during attacks of angina-like chest pain (1), it was infrequently seen in this series, even when liberal criteria for the presence of spasm were employed. More often we found an increased amplitude of contraction, often accompanied by a slow velocity and a tendency towards an increased wave duration. Similar results have been found occasionally in other patients with chest pain (6–8). In a few patients, it was possible to predict the intensity and duration of the chest pain by monitoring the amplitude and duration of the accompanying peristaltic wave.

It was originally hoped that manometric classification would allow better selection of a pharmacologic agent to modify the pain. However, the response to nitroglycerin and to antacids was not predictable on the basis of the manometry tracing. Although nitroglycerin has been said to be helpful in relieving the pain of diffuse spasm (9, 10), our two patients did not respond to it. A response to nitroglycerin seemed to occur as often in patients with normal manometric tracings as in those with abnormal tracings.

Prior to this study, the authors held the opinion that most patients complaining of true angina-like pain were rarely, if ever, acid-sensitive or responsive to antacids. However, data reported here have forced a reappraisal of this opinion. One of the 7 patients with abnormal Bernstein tests had his chest pain, rather than heartburn, reproduced by the acid infusion; this has been reported previously (11, 12). Furthermore, 12 of our 58 patients noted improvement (usually not permanent) when vigorous antacid therapy was employed. Two patients were shown by pH monitoring to have their typical chest pain only when acid refluxed into the esophagus (even though no manometric abnormalities occurred at that moment). Yet one of these patients stated that intensive antacid therapy had not affected her pain at all. It is now our practice to perform Bernstein tests and to monitor pH during all manometric evaluations of chest pain, hoping to clarify the role of acid reflux in the production of esophageal pain.

If the definition of chest pain is less restrictive than that used in this study, many more patients with esophageal origin of their pain will be included (13). In this report, we include only those patients whose chest pain was indistinguishable from angina pectoris, in an effort to determine the usefulness of esophageal study in this clinically important group. In the absence of universal coronary arteriography, we cannot say with complete assurance that in all group I patients the heart is not responsible. Furthermore, in the patients with known CAD and manometric abnormalities (group II), it is particularly difficult to be certain which malfunction is responsible for the pain. However, we have found the approach to the data that is outlined in Figure 4 helpful in all of the subgroups included in this study.

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