techniques. But exercise testing also serves as the doorway to these and other more powerful studies for coronary disease, including cardiac catheterization. It is very important for informing about prognosis for medical and psychosocial purposes, and it provides a reasonable and objective means of follow-up, particularly in patients who don't provide an excellent history or don't know how far or how long they're walking.

In summary, exercise testing is not the most sensitive noninvasive test for the diagnosis of chest pain, but its reliability and cost-effectiveness for monitoring the progression of disease and determining therapeutic efficacy are unmatched by other diagnostic means. In the United States, the volume of exercise tests being performed is increasing. The Health Care Financing Administration reports a 22% increase in the number of exercise tests interpreted and reported from 1986 to 1988. This represents an estimated increased annual cost of about \$30 million. With the test's increasing use and its role as a means of access to more expensive noninvasive and invasive procedures, improvements in the recognition of its indications for performance can have a huge impact on health care.

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SUGGESTED READING

Chaitman BR. The changing role of the exercise electrocardiogram as a diagnostic and prognostic test for chronic ischemic heart disease. J Am Coll Cardiol 1986; 8(5):1195–1210.

Froelicher V, Marcondes G. A manual of exercise testing. Chicago: Year Book Medical Publishers, 1989.

Schlant RC, Blomqvist CG, Brandenburg RO, et al. Guidelines for exercise testing. A report of the Joint American College of Cardiology/American Heart Association Task Force on Assessment of Cardiovascular Procedures. Circulation 1986; **74**:653A–667A.

Sox JH, Littenberg B, Garber AM. The role of exercise testing in screening for coronary artery disease. Ann Intern Med 1989; 110(6):456–469.

TRENDS IN NONCARDIAC CHEST PAIN

Up to 30% of patients referred for cardiac catheterization every year are found to have normal coronary arteries. Of these, up to 50% may have esophageal abnormalities. Although the prognosis for these patients is excellent, their functional outcome is generally poor. Even with appropriate treatment, many of these patients continue to have chest pain which

they believe has a cardiac source. The result is costly in terms of quality of life and lost work.

ESOPHAGEAL CAUSES RECONSIDERED

Until recently it was believed that most esophageal chest pain resulted from motor disorders. However, the association has been difficult to prove in controlled settings; patients rarely have chest pain at the time of manometry, even if a motor abnormality is present. Even 24-hour manometry has failed to show a clear association between pain and motor abnormalities. Increasingly, both the "nutcracker" esophagus (characterized by high-amplitude, long-duration peristaltic contractions) and nonspecific motor disorders are viewed as manometric epiphenomena.

In the 1990s, the pendulum has swung to gastroesophageal reflux disease (GERD) as the predominant esophageal cause of chest pain. Studies have demonstrated that pain is reproduced more frequently in the setting of lowered pH (15% to 33%) than with abnormal motor events.

DIAGNOSTIC STRATEGY

The American Gastroenterology Association has issued guidelines for the management of esophageal chest pain. The first, crucial step is to exclude heart disease with stress testing, cardiac catheterization, or both. Musculoskeletal causes can be excluded on physical examination.

Second, structural gastrointestinal disease should be ruled out with a barium esophagram or upper endoscopy. If these are negative, then diagnosis of GERD should be attempted, preferably with 24-hour pH monitoring. Although expensive, 24-hour pH monitoring will determine the extent of reflux in upright, supine, and combined positions, and it will correlate reflux with chest pain.

A less expensive option is the Bernstein test. This relatively simple procedure involves reproducing heartburn or chest pain by perfusing the esophagus with hydrochloric acid. However, the yield is low, ranging in various studies from 6% to 36%. The Bernstein test is helpful if the results are positive, but not if they are negative.

If reflux cannot be identified, then specialized testing is indicated to rule out motor disorders and altered pain perception. This includes esophageal manometry,

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edrophonium provocation, balloon distension, and psychological evaluation. An esophageal cause is "definite" if symptoms can be reproduced during provocative studies; an esophageal cause is "probable" if only a manometric abnormality is seen. Although still investigational, 24-hour manometry may be useful in the future. At present the criteria for abnormal studies with this technique are not yet well defined. The test is also expensive and the diagnostic yield is low.

The prevalence of psychiatric disorders is high in this patient population, and psychiatric referral is helpful if the patient can be convinced to cooperate. Indeed, the only controlled data available that support pharmacologic therapy in noncardiac chest pain conclude that antidepressants are helpful.

MANAGEMENT OF GERD

Even if a relationship can be established between acid and chest pain, many questions remain unresolved. Does treatment resolve the pain? What percentage of chest pain episodes should correlate with reflux to be diagnostic? What is a positive symptom index?

The mechanism of acid-related pain is also not clearly understood.

Only one study has evaluated whether treatment of reflux has a positive effect on noncardiac chest pain. Of 50 patients with noncardiac chest pain who underwent 24-hour pH monitoring, nearly half were shown to have abnormal amounts of gastroesophageal reflux. In 13 patients who had chest pain during the 24-hour monitoring period, 12 had symptoms that correlated with reflux drops of 92%. Medical or surgical antireflux treatment resolved these patients' symptoms.

When GERD has been established in a patient with noncardiac chest pain, it is critical to reassure the patient of the absence of cardiac disease. GERD warrants aggressive treatment with either an H-2 receptor antagonist or omeprazole. Omeprazole is probably the best antireflux agent available today, although controlled studies documenting its effect on chest pain have not yet been published. A study is now under way at the Cleveland Clinic to determine whether treatment of GERD with omeprazole resolves chest pain and whether 24-hour pH monitoring predicts a response to therapy.

For patients whose chest pain does not resolve with antireflux therapy, antidepressant therapy with trazodone has been shown to be helpful, although the difference between placebo and trazodone was not statistically significant. This patient population has a high placebo response rate.

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SUGGESTED READING

Browning TH. Diagnosis of chest pain of esophageal origin. Dig Dis Sci 1990; 35;289–293.

Clouse RE, Lustman PJ, Eckert TC, Ferney DM, Griffith LS. Low dose trazodone for symptomatic patients with esophageal contraction abnormalities. Gastroenterology 1987; 92:1027–1036.

Demeester TR, O'Sullivan GC, Bermudez G, et al. Esophageal function in patients with angina-type chest pain and normal coronary angiograms. Ann Surg 1982; 196:488–498.

Hewson EG, Sinclair JW, Dalton CB, Richter JE. Twenty-four-hour esophageal pH monitoring: the most useful test for evaluating noncardiac chest pain. Am J Med 1991; 90:576–583.

Richter JE, Bradley LA, Castell DO. Esophageal chest pain: current controversies in pathogenesis, diagnosis, and therapy. Ann Int Med 1989; 110:66–78.