Leading Article

Oesophageal chest pain: a point of view

Between 10–30% of patients referred to cardiology clinics, or admitted to coronary care units appear to be free of ischaemic heart disease.^{1 2} Furthermore, up to 20% of coronary angiograms carried out in some centres are either normal, or show trivial lesions considered unlikely to account for the patient's symptoms.³ A very few patients give a characteristic history with ECG changes at the time of pain, indicative of coronary artery spasm which may be shown at angiography by provocation with ergometrine.⁴

WHAT HAPPENS TO PATIENTS WITH NO APPARENT CARDIAC CAUSE FOR CHEST PAIN?

Some may be reassured by the assertion that their heart appears sound, but continuing symptoms without apparent alternative explanation from their physician usually results in continued concern and doubt.⁵ Rarely, a cause may become apparent as the history, or signs of a musculoskeletal disorder such as costochondritis, evolve. An occasional patient has cholelithiasis, or other intra-abdominal disease. Endoscopy will show oesophagitis in some, or the symptoms may be provoked by a Bernstein oesophageal acid perfusion test: in selected cases it may be necessary to monitor distal oesophageal pH with an oesophageal pH probe in order to confirm suspected gastro-oesophageal reflux. Yet in most gastroenterologists' experience the large majority of these patients cannot be shown to have any of these disorders.

There is at present a renewed wave of awareness that oesophageal dysmotility may cause chest pain. It should come as no surprise that the gullet might cause pain perceived in the chest wall, nor that the pain may closely mimic ischaemic heart disease, as the heart and oesophagus share a common innervation. A history of dysphagia is a strong pointer to oesophageal pathology and this symptom should be specifically sought, because intermittent mild difficulty with swallowing is often not volunteered when overshadowed by chest pain and concern about a possible cardiac cause. In the United States 'non-cardiac chest pain' has surpassed all other causes of referral for oesophageal manometry and gastroenterologists are asked to provide the answers that account for the patients' symptoms.

We are becoming painfully aware that we often do not have the answers and find ourselves groping for various diagnostic techniques. We arrived at our present position from the observation that patients with oesophageal 'spasm' may experience chest pain indistinguishable from cardiac pain. This, coupled with the blossoming of oesophageal manometry laboratories throughout the United States has led to an explosion in the use, and possibly abuse, of these tests. In the United Kingdom generally,

recognition of the clinical need for oesophageal manometry has been slow and perhaps cardiologists here have been less forward in referring their 'albatross' patients. One hopes that the 'albatross' patient may not be encountering the 'ostrich' gastroenterologist! We should perhaps, take a hard look at some of the questions that presently haunt us in approaching these patients, for in all fairness, they may present as many questions as there are answers.

WHAT ARE THE TYPES OF OESOPHAGEAL MOTILITY DISORDERS SEEN IN PATIENTS WITH CHEST PAIN SYNDROMES?

A number of published,⁶⁻¹⁸ or about to be published, series provide ample data at least to begin to answer this question. Diffuse oesophageal spasm, 'nutcracker oesophagus', 'hypertensive lower oesophageal sphincter', non-specific oesophageal motility disorder and achalasia, have all been shown. There may well be other disorders implicated and a new syndrome termed the 'tender' oesophagus has recently been described.¹⁹ The identification of some of these abnormalities has resulted from our attempts to quantitatively define normal values for oesophageal pressures in various laboratories. When this is carefully done, it becomes apparent that there are a number of patients who have raised peristaltic contraction pressures, usually of excessively long duration: 'the nutcracker oesophagus',^{13 14} or high resting lower oesophageal sphincter pressures: 'the hypertensive lower oesophageal sphincter'.^{15 16}

While expert upper gastrointestinal radiology may show some of these abnormalities, this is only the case in a minority of instances. More sensitive than routine radiology, the radionuclide oesophageal transit test²⁰⁻²² has a high degree of concordance with the results of manometry. Recent experience with this technique indicates that it will show abnormalities in almost all patients with clearly abnormal manometry, especially when there are non-propagated oesophageal contractions. In our experience this test gives normal results when the only abnormality is high amplitude, or prolonged duration peristalsis, or a normally relaxing lower oesophageal sphincter with a high resting pressure. For departments without manometric equipment, the radionuclide transit test may prove a satisfactory screening test as long as its limitations are recognised.²³

WHAT IS THE INCIDENCE OF OESOPHAGEAL MOTOR DISORDERS IN PATIENTS WITH NON-CARDIAC CHEST PAIN?

There are, again, some data available to answer this question.⁶⁻¹⁸ Incidence figures for oesophageal causes for non-cardiac chest pain depend on whether one attempts to exclude patients with abnormal gastro-oesophageal reflux. In one study of patients discharged from a coronary care unit in whom cardiac disease had been reasonably excluded, oesophageal disease was felt to be the cause of chest pain in one-half of the patients.¹¹ There were, however, a large number of patients with gastro-oesophageal reflux in this series. When patients with reflux are excluded, the prevalence of oesophageal abnormality as a cause of non-cardiac chest pain varies from 18-58%. In our own study of such patients, oesophageal dysmotility was shown in 45% of patients.²⁴ These data indicate that there is a high prevalence of abnormal oesophageal motility in patients with chest pain believed to be of non-cardiac origin.

DOES THE OESOPHAGEAL MOTILITY DISORDER CAUSE THE CHEST PAIN?

This is obviously one of the most crucial questions and one of the most difficult to answer. Most patients report that they have intermittent episodes of pain and are rarely obliging enough to experience the pain during the manometric motility study. When this does occur, the simultaneous occurrence of pain and oesophageal dysmotility enables a more certain diagnosis to be established. There is no way to be sure, however, that the chest pain is due to abnormal motility if the patient is asymptomatic at the time of testing. There is also no way of being certain that any motility disorder shown is the same as that which occurs when the patient has the pain. Studies are presently underway in which the intraoesophageal pressures are monitored during longer periods of time, in order to define what motility abnormality actually occurs when the patient has a spontaneous episode of chest pain. We must, however, deal with the observation that there is a high prevalence of oesophageal motor disorders in patients with such non-cardiac chest pain. Is this cause or effect; or merely guilt by association? There are at present no published data on manometry in patients with known ischaemic heart disease to make comparisons with that important control group. The critical manoeuvre is to provoke the pain and the oesophageal motor disorder, as most patients will be asymptomatic at the time of study.

HOW CAN WE PROVOKE THE PAIN AND THE MOTILITY DISORDER?

Several techniques have been suggested over the years to provide the oesophageal 'stress test'. These include iced-water swallows,²⁵ intraoesophageal instillation of acid,²⁶ injections of bethanechol,^{17 27} edrophonium,^{17 28} pentagastrin,^{17 29} or ergometrine.^{28 30} There has been no laboratory support for the clinical suggestion that swallowing cold liquids might provoke a diagnostic manometric response. Although intraoesophageal acid perfusion in the Bernstein test can be of great help in diagnosing the symptom of heartburn, or atypical symptoms secondary to acid reflux, there is no evidence that an abnormal oesophageal pressure response after acid reflux occurs in patients who are Bernstein test 'negative'. Although injections of bethanechol, or pentagastrin have been shown to amplify the motility abnormality in patients with a known diagnosis of diffuse oesophageal spasm, they have not been shown to be effective as provocative diagnostic agents. Infusion of ergometrine will produce chest pain and associated abnormal oesophageal contractions in a number of patients with unexplained chest pain syndromes. Unfortunately, the cardiac risks of this drug are sufficiently great to agree that it is generally ill-advised to use it as a diagnostic test in any setting other than the cardiac catheterisation laboratory during monitoring of the coronary arteries.³¹

There is, however, some encouragement to diagnostic testing. In one study²⁸ comparing the response of the oesophagus with ergometrine and to edrophonium, it was shown that most patients who responded to ergometrine also responded to edrophonium. In another recent observation¹⁷ from our laboratory we have shown that approximately 20% of patients with non-cardiac chest pain will have a change in their manometric tracing simultaneously with chest pain provoked by the injection of edrophonium 80 mcg/kg IV. There is also evidence that although the

amplitude and duration of peristaltic contractions increases in patients who do not experience edrophonium provoked pain, the degree of response in patients who do experience pain appears quantitatively greater.³² We would suggest that at present the routine use of edrophonium as a stress test provides the best available provocative agent in the diagnosis of oesophageal dysmotility causing chest pain.

WHAT IS THE APPROPRIATE THERAPY FOR THESE PATIENTS?

Many physicians have been discouraged by the often poor response of patients with oesophageal spasm to medical therapy. Again, matters have improved in recent times and a more encouraging outlook seems justified. Paradoxically, those drugs which reduce smooth muscle tone and have been developed for the symptomatic treatment of angina pectoris have been used for treatment. Such drugs also affect the distal oesophageal musculature. Although short³³ and longer acting nitrates³⁴ have proved beneficial in oesophageal spasm, some patients do not improve, or cannot tolerate the side effects. Recent studies with nifedipine,^{35 36} diltiazem,³⁷ and verapamil³⁸ have shown a potential use for these calcium channel blocking drugs in oesophageal motility disorders. The vasodilator hydalazine³⁹ has also shown some promise. Controlled trials are notable for their absence, in part because of the intermittent nature of oesophageal dysmotility, but clinicians should perhaps be encouraged to be more experimental with these drugs in the treatment of their patients.

Further optimism and justification for full investigation, may be gained from the observation of the natural history of motility disorders. Some patients with mild, yet psychologically disturbing symptoms, will be so reassured by an accurate diagnosis and a confident prognosis of continued survival based on positive grounds, that treatment becomes superfluous. Other patients experience intermittent symptoms, so that treatment is only needed for short spells. In our experience the patient with persistent and severe symptoms is uncommon and we have rarely had to resort to empiric pneumatic dilatation, or oesophageal myotomy. Repeated investigation, however, will be necessary to detect the occasional patient who appears to progress from diffuse oesophageal spasm to achalasia.

WHAT POINT HAVE WE REACHED AND WHERE ARE WE GOING?

It is clear that there is considerable confusion and controversy at present in the area of identifying the patient with a definite oesophageal cause of chest pain. By careful attempts to identify in quantitative terms what the normal manometric values should be in our laboratories, we can now begin to define more clearly what is abnormal. In doing this, we have recognised that there are variants of what was once called diffuse spasm. Diagnoses such as the 'nutcracker oesophagus', or oesophageal 'supersqueezers', or the 'hypertensive LOS' all become apparent as we begin to talk in quantitative terms. We must now begin to work on a system of communication, for clearly, at present one man's spasm is another man's 'nutcracker'. Despite criticisms⁴⁰ of a classification based principally on manometric features it is the best available to us at present. Research directed towards identification of a better provocative test, or a better monitoring system to show that the oesophagus is causing the pain, are to

be encouraged. Oesophageal motor abnormalities do cause chest pain. We must continue to attempt to define their importance.

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