What, then, are our duties towards insulin dependent diabetic patients in relation to home blood glucose monitoring? Doctors' attitudes will vary, but all patients should be made aware of the technique and, if interested, be given or loaned equipment for a trial period. Those choosing to continue will need further guidance. The choice between a reflectance meter and strips suitable for direct visual reading is an individual one, but whichever method is used the patient's competence should be assessed. There are sources of potential error,²¹ and repeat checking of technique and accuracy is necessary. Patients will tire quickly of producing large numbers of results if they do not understand or are not told how to use them, so arrangements are needed for individual follow up by a physician. The results may point towards the need for radical changes in insulin treatment, and if these are not undertaken monitoring becomes no more than a record of poor control. All this means that if more patients are to use self monitoring effectively, demands on staff time will increase and efficient schemes will be needed for the education of patients. The emergence of the specialist nursing teacher is a step forward, but all those concerned with diabetic patient care, including family practitioners, can play a part by becoming familiar with self monitoring techniques.

What, finally, are the financial implications for patients and the National Health Service? Reflectance meters cost around £80 and strips retail at about 26p each (NHS cost about 15p each). Blood monitoring equipment is not available on prescription, and though some patients receive supplies through hospital pharmacies others bear the full cost. If we believe that near normal blood glucose concentrations are desirable, it seems illogical to provide on prescription urine testing apparatus which, though cheaper, can detect only frank hyperglycaemia and to penalise those who prefer to use blood testing. And, in balancing the costs with the advantages of blood glucose monitoring, we must be aware of patient preferences and needs, which are not reflected in objective indicators of control.

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Chest pain: heart or gullet?

Compared with the two centuries since Heberden's elegantly clear description of cardiac pain, recognition of the oesophagus as the origin of recurrent chest pain is relatively recent. Walter Verdon's quaint monograph¹ of 1920 was, perhaps, the first milestone. Public consciousness of the fact that chest pain may be a harbinger of death tends to concentrate both lay and medical attention on the heart in any patient with discomfort in the chest, and the patient may have extensive cardiological investigations before alternative explanations are considered. This is understandable, for the clear identification of cardiac pain is not always easy, and no one wishes to pass off as trivial a complaint which may all too soon disclose its true origin to the coroner's pathologists and which might be treatable by drugs or coronary bypass surgery.

Yet hazards exist in an entirely cardiological approach to chest pain, for inevitably this suggests to the patient that he may harbour a mortal illness within his breast. If in fact he has a benign oesophageal condition this may amount to a cruel deception, with a potential for cardiac neurosis. A recent study in Cardiff² showed that only two thirds of patients admitted to hospital because of anterior chest pain turned out to have ischaemic heart disease, similar to our figure in Worcester 17 years ago³ and that of William Evans⁴ in London before that. In as many as a fifth of patients the pain may have originated in the oesophagus.

The reason that so many patients with non-cardiac chest pain reach hospital with a diagnosis of heart disease is easy to understand. General practitioners and doctors in accident departments are likely to expect the hospital to resolve any doubts lest blame results. Such an attitude is reasonable provided that a firm diagnosis is quickly reached, for a patient may react adversely to medical doubt, and even a hint of a cardiac disability may have grave repercussions on his future health (mental and physical) and employment.

Many disorders may produce chest pain simulating that of coronary artery disease. These include spondylosis, root pains, chest wall pains, zoster, and hyperventilation, but some lesion of the oesophagus is the most common alternative. The most important step in differentiating cardiac from oesophageal pain is a careful history with an emphasis on discriminatory features. The Cardiff study² showed that reliance on Heberden's⁵ original features of angina—retrosternal location, strangling quality, relation to exertion, and accompaniment

by mental anxiety—is insufficient, for of 77 patients with pain of this character, a fifth had an oesophageal origin for their pain. Nevertheless, of 23 patients with non-anginal pain, only one had a myocardial infarct. A few patients may describe the oesophageal pain as gripping, or radiating down their arm, or precipitated mainly by exercise; some patients with ischaemic heart disease may complain of burning discomfort or experience most of their pain in bed; but most patients will cause no great diagnostic problem if the history is carefully sought.

Add to this careful history a clinical examination which may find cardiac abnormalities and a standard electrocardiogram and only a few patients will remain undiagnosed. For some of these, in whom the pain is infrequent and whose initial investigation shows no hint of cardiac disease, a label of "chest pain ?cause" may be the most practical solution.⁶ If the doctor or patient finds that unsatisfactory further investigation is desirable.

If ischaemic heart disease is uppermost in the doctor's mind he will first turn to exercise electrocardiography, though he should bear in mind the interpretative difficulties. If still in doubt some would proceed to coronary angiography, but as oesophageal pain is a likely alternative an equally logical—and certainly safer—response would be to seek abnormalities in the gullet. Pain-producing oesophageal disorders range from the common reflux oesophagitis to rarities such as achalasia and diffuse spasm; other motor abnormalities may be discovered but these may not always be identified as the definite cause of the patient's pain.

A barium swallow and meal examination may display gross lesions such as achalasia or classical diffuse oesophageal spasm, suggest lesser degrees of abnormal oesophageal motility in the form of tertiary contractions, or may sometimes show an unexpected ulcer; the examination is less satisfactory for recognising gastro-oesophageal reflux. Fibreoptic endoscopy and oesophageal biopsy will find oesophagitis and ulcers, but endoscopy is a poor guide to problems of motility.

Perfusion of the oesophagus with acid, a simple inexpensive test, will usually precipitate the patient's pain if it is caused by reflux oesophagitis, and any doubts about cardiac problems may be resolved by simultaneous electrocardiography.^{7 8} Acid gastro-oesophageal reflux may be accurately delineated by intraoesophageal pH monitoring.⁹⁻¹¹

Oesophageal manometry will uncloak some motility disorders causing chest pain, but the technique is not available widely enough in Britain and anyway poses problems of interpretation. Now that gammacameras are more generally accessible isotopic oesophageal transit studies may come into more use as a screening test for oesophageal motor abnormalities,¹² though any suspected abnormality will need further investigation by manometry. Davies and his colleagues have shown that some oesophageal motor abnormalities causing pain may be provoked by injecting ergometrine during manometric studies,¹³ but the potential dangers of this drug in provoking coronary artery spasm makes its routine use unwise. Edrophonium has similar benefits and disadvantages.¹⁴ Safer provocative agents are needed, for even when pain indisputably arises from oesophageal motor abnormalities these are transient

and elusive, readily escaping detection by tests done between episodes of pain.

When the whole gamut of cardiac and oesophageal investigation has been run there will remain two groups of patients still posing problems—those for whom there is no answer, and those who have both oesophageal and cardiac pain.

The "insoluble group" may frustrate their doctors, but this should not make them victims of overenthusiastic diagnostic endeavours. A decision to halt tests, assurance that they have neither oesophageal nor cardiac disease of major importance, and perhaps symptomatic relief with analgesics should be sufficient. The unfortunate patients with both oesophageal and cardiac pains are potentially more difficult unless they (or their doctor) can learn to discriminate between them. All too often the fear of heart disease leads to multiple unnecessary hospital admissions for successive episodes of pain. More than anything else these patients need an understanding general practitioner who is available to calm their anxieties and to treat the pains appropriately.

As William Heberden wrote¹⁵ after clearly delineating angina pectoris, ". . . the breast is often the seat of pains which are distressing sometimes even from their vehemence, oftener from their duration, as they have continued to tease the patient for six, for eight, for nine, and for 14 years. There have been several examples of their returning periodically every night. . . . There has appeared no reason to judge that they proceed from any cause of much importance to health . . . or that they lead to any dangerous consequences."

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