Oesophageal pain

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ALTHOUGH Morgagni's account of a man with a 'vehement cardalgia' due to a hiatus hernia is now about two centuries old, I do not think anyone has established with any accuracy what proportion of otherwise normal people experience oesophageal pain. My own enquiries have made it clear that I am one of the fortunate but small minority who have never had heartburn. So one of the two most important things about oesophageal pain is its ubiquity, the other is that it may exactly simulate cardiac pain. This second aspect was the starting point of an investigation of oesophageal pain which Dr Atkinson and I carried out in Worcester. As a prospective study we looked carefully at all patients admitted to the unit with anterior chest pain over a 3-year period. There was an *unselected* group of 124, accepted on a fixed emergency rota, and seventy-six patients specifically referred to us for study of their pain. I have used only the *unselected* group in the analysis of incidence, but include all patients in the consideration of symptoms and signs.

Now of the emergency admission 74% had ischaemic heart disease causing their pain, though 12% had gastro-intestinal disease too. Twenty-four per cent had only alimentary tract disease, mostly oesophagitis. We showed that neither the referring practitioners nor the patients themselves made the right diagnosis in more than two-thirds, and of course we ourselves only reached a certain diagnosis in many instances after a good deal of investigation. Indeed in nine of the 200 we never made a final diagnosis.

The reasons for the confusion are not difficult to find (Bennett & Atkinson, 1966).

The age distribution is similar.

The site of the two pains and their radiation can be identical.

The type of pain cannot be relied upon for differentiation, nor can the factors which perpetuate it.

So although in most instances a physician aware of the problem will have no difficulty in establishing the cause of precordial pain, a proportion of patients will remain whose pain

cannot be categorized with any certainty. Here is a dilemma, for no-one wishes to pass off as trivial a complaint which may all too soon reveal its true origin to the Coroner's pathologist, yet it is cruel and bad medicine to suggest to a patient that he harbours a mortal illness within his breast, if, in fact, he only has a touch of indigestion. A middle-aged overweight patient with a raised blood pressure complaining of a gripping chest pain which may radiate to arm, jaw or back, and which is made worse by emotion, may well have reflux oesophagitis and not ischaemic heart disease.

Methods

Having assessed the use of acid perfusion of the oesophagus as a provocative test for oesophageal pain, we believe it is a useful aid. The basis is a simple one—all that is required is a Ryle's tube, a drip-set and a bottle each of saline and decinormal hydrochloric acid—but we took the opportunity of doing some manometric studies at the same time.

For the tests the patient sat in a comfortable high-backed chair and the tube was positioned under manometric control so that fluids entered the oesophagus 8-9 cm above the cardia. Pressures were recorded 5 cm above and below that point. Saline was infused for 15 min, then, without the patient's knowledge, the infusion solution was changed to decinormal hydrochloric acid at 10 ml/min. Unless symptoms occurred, this was continued for 15 min after which the rate of infusion was doubled for a further $\frac{1}{4}$ hr. If pain occurred, we usually tried the effect of decinormal sodium bicarbonate.

Results

Of fifteen normal subjects, mostly hospital staff and students, who did not suffer from dyspepsia, eight experienced central chest burning from 3 to 29 min after beginning the acid perfusion. Six of these eight had experienced similar heartburn on a few occasions in the past, usually after dietary or alcoholic excess. The induced discomfort was never severe and was rapidly relieved

by the infusion of sodium bicarbonate in the five in whom it was tried. We did not think it justifiable to subject these volunteers to oesophagoscopy or to X-ray studies.

Eleven patients with undoubted and typical angina were tested, eight of whom had abnormal ECGs. Four of them experienced pain during acid perfusion. Two were certain that it was different from their angina, and, since it was relieved by bicarbonate, this pain was thought oesophageal. The other two had pain identical with their angina during the control infusion of saline, as well as with acid, and one of them had a change in his ECG during the induced pain.

Thirdly we tested twenty-nine patients whom we considered to have oesophagitis. This diagnosis was made by Dr Atkinson in ignorance of the results of the acid-perfusion test, and was confirmed endoscopically in twenty-four.

Twenty-eight of the twenty-nine patients experienced their characteristic pain during the acid perfusion. The one who did not had suffered no spontaneous pain for several months. Three of the twenty-eight also experienced pain during the saline perfusion. In each case the pain was rapidly made worse by the acid, and in two was relieved by bicarbonate. Thus in twenty-five of the twenty-nine patients with oesophagitis this test was conclusively positive, whilst in three others it was probably so.

The duration of acid perfusion needed to produce the pain varied. In most instances pain appears early, but sometimes it takes almost 30 min.

The pain was relieved by sodium bicarbonate in twenty-one of the twenty-two in whom it was tried. The one whose pain was not relieved also had saline-induced pain.

These results in patients where the diagnosis was not in doubt seemed to indicate that the test was a valid one for oesophageal pain, so we applied it to twenty-two patients whose chest pain was not easy to diagnose on clinical grounds alone. Nine of them had abnormal ECGs.

An undoubtedly positive response to acid perfusion was obtained in fourteen, their characteristic pain being reproduced, while saline perfusion was pain-free. Sodium bicarbonate relieved the pain in five of the six in whom it was tried. Changes of oesophagitis were present in ten of the fourteen who were oesophagoscoped.

Of the eight with a negative response, three almost certainly have angina pectoris; two have ECG evidence of ischaemia, although one also shows endoscopic evidence of oesophagitis. Two others have peptic ulcers and another one gallstones, their pain being attributable to these

causes. The remaining two both have endoscopic oesophagitis, one of them also having an ischaemic ECG, though in neither does the pain conform to the usual pattern of oesophageal or cardiac pain.

Discussion

On this evidence it is impossible to be certain that the perfusion test has led us to the correct diagnosis, though during the period of follow-up no patient has produced fresh evidence to suggest that the result was misleading.

The test undoubtedly has real value in the cases where it is *positive*, for by provoking the patient's original pain with a controlled stimulus one has strong evidence as to its source.

Sometimes one may produce heartburn in subjects who have never previously experienced it—what Bernstein & Baker (1958) called a 'positive unrelated response'. This will not cause confusion except in unco-operative or unintelligent patients unable to differentiate one pain from another.

This is not a test for oesophagitis, but for oesophageal pain. It should be emphasized that the degree of oesophagitis seen at endoscopy did not correlate closely with the amount of pain experienced.

What I have described so far is not very contentious—in the main it simply emphasizes or confirms what others have said before. However, the method of production of oesophageal pain remains a matter of dispute. The more favoured view—supported by Jones & Richardson (1926) and Siegel & Hendrix (1963): is that muscular contractions of the oesophagus cause the pain; Tuttle's group in Los Angeles believe that chemical irritation is the basis of heartburn (Tuttle, Bettarello & Grossman, 1961).

We made recordings of oesophageal motility, which were adequate for study in thirty-nine patients with oesophagitis and in fourteen normal volunteers.

In three subjects with oesophagitis cine radiographs were made at the same time as manometry, the infused solutions having been made radio-opaque with barium sulphate.

Secondary peristalsis, i.e. a progressive contraction not initiated by a swallow, was inevitably produced, the frequency depending on the rate of infusion, whatever the fluid. This has been excluded from a consideration of abnormal motility. Induced pain was associated with a change of motility in about two-thirds of the subjects—twenty-five of thirty-nine with oesophagitis, and five of eight normals.

The induced changes were of two types:

(1) 'Non-progressive' contractions occurring syn-

FIG. 1. Non-progressive contractions during pain. (a) No pain, (b) pain.

chronously in both pressure traces. Cine radiography combined with manometry showed these to represent 'tertiary contractions' or rippling. This was seen in all the thirty subjects who showed altered motility (Fig. 1). In ten cases synchronous waves had appeared during the control saline perfusion but increased in size, frequency or duration. More frequently synchronous contractions first appeared during pain.

(2) A rise in basal end expiratory pressure above that during the control saline infusion. This was seen in ten instances, the rise of pressure varying from 2 to 10 mmHg.

Sometimes it seemed that the motor changes might have contributed to the pain.

In four instances the onset of pain coincided with a striking alteration in the motility pattern, and in three patients spasms of more severe pain

coincided with synchronous oesophageal contractions.

However, identical increases in synchronous activity appeared without inducing pain in two normal subjects and in two patients with angina. Furthermore, during acid-induced pain fifteen patients with oesophagitis and three normal subjects showed motility patterns no different from those during the pain-free control infusion of saline.

Three patients with oesophagitis had pain precipitated by saline perfusion and accompanied by frequent synchronous contractions. On the other hand seven subjects with synchronous contractions during saline infusion had no pain until the acid drip was started.

Most important are the effects of propantheline on the motility and the pain.

In four subjects, reduction or abolition of

FIG. 2. Pain relieved by propantheline.

FIG. 3. Pain not relieved by propantheline. (Reproduced by courtesy of the Editor, *Amer. J. dig. Dis.*)

oesophageal motility in this way improved the pain considerably, though never caused it to disappear completely (Fig. 2).

But in eleven subjects whose motility was similarly affected, pain was not relieved (Fig. 3). Indeed in two the pain became worse after the injection.

Oesophageal pain was more closely associated with an increase in oesophageal acidity than it was with alterations of oesophageal motility.

A tenable hypothesis for the cause of pain of the type associated with gastro-oesophageal reflux is that it is produced by the effects of an irritant fluid on a sensitive inflamed mucosa. This irritation—or even the mechanical effect of fluid entering the oesophagus—may induce muscular contractions as a secondary phenomenon. These contractions sometimes aggravate and perpetuate the pain and lead to other symptoms such as dysphagia, but in the majority their abolition does not reduce the pain.

A small proviso is necessary here:

During these acid perfusion tests, we never produced very violent pains of any duration. It does seem likely that some of these *are* due to muscular spasm, but we are reasonably sure that the less dramatic, but much commoner attacks of mild pain or heartburn, are due to effects on the mucosa.

Conclusion

I have tried to point out that oesophageal pain is a very common condition. In one individual it may appear rarely, as the penalty of over-indulgence, while in another it can be a constant and disabling complaint. Usually it responds well to simple treatment, yet many suffer for years

because the cause of their pain is not recognized. The unfortunate victim may be labelled as having heart disease, peptic ulceration, gall-stones, psychoneurosis, or a multitude of other mistaken diagnoses. We can say, as Moersch & Miller did in 1943—'On a theoretical basis a careful history should enable one to distinguish between cardiac and oesophageal pain. But in spite of the most careful studies it is at times impossible to distinguish accurately between them'. Oesophagitis is truly the masquerader of the upper abdomen, but we have shown that acid perfusion of the oesophagus is a valuable aid in establishing the cause of precordial pain, and can be carried out simply, safely and inexpensively.

We have also shown that under the conditions of the test pain production correlated well with an increase in oesophageal acidity and in the majority was independent of any oesophageal muscular activity. In a minority, oesophageal contractions appeared to aggravate the pain without being a major cause in its production.

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