Oesophageal motility, luminal pH, and electrocardiographic-ST segment analysis during spontaneous episodes of angina like chest pain

D G Hick, J F B Morrison, J F Casey, W Al-Ashhab, G J Williams, G A Davies

Abstract

The relation between oesophageal motility, luminal pH, and spontaneous pain events in 47 patients with recurrent angina like pain and normal coronary arteries was investigated. Preliminary investigation by conventional station pull through manometry (SPTM), was followed by a 24 hour period of ambulatory oesophageal motility and luminal pH monitoring. Computerised analysis of motility and pH data recorded during chest pain was then compared with pre-elected control samples taken before and after symptoms. Concurrent real time electrocardiographic (ECG)-ST segment analysis was performed to catalogue any ECG-STEP wave changes indicative of myocardial ischaemia. SPTM showed a high group percentage incidence of simultaneous contractions (mean (SD) 11-1 (2-3)% ) and a raised lower oesophageal sphincter tone (57-4 (15-2) mm Hg). During ambulatory monitoring, 35 patients experienced one or more episode of angina, providing a total of 59 pain events, although no significant change in group motility and reflux parameters peculiar to episodes of chest pain were found. Ischaemic ECG changes were detected in 10 (21%) patients, but were accompanied by pain in only two. Independent analysis of the ECG traces corresponding to these purported ischaemic ECG events determined them unequivocal in three patients and probable in a further two. No apparent correlation was noted, however, between these ECG events and corresponding patterns of motility or reflux.

The differential diagnosis of chest pain is notoriously difficult, compounded largely by an overlap in the distribution and perception of pain arising from many of the intrathoracic structures and viscera. The ability of the oesophagus to generate the angina like symptoms of myocardial ischaemia has long been appreciated and considered to arise through segmental overlay of the sensory afferent fibre entering the spinal cord from the sympathetic limb of the autonomic nervous system (oesophagus C8–T10; heart T1–T4). Accordingly, sensation may be perceived over the dermatomes or myotomes supplied by somatic fibres of the segments common to both organs.1-4

True angina like pain, however, is generally regarded as an atypical symptom of oesophageal dysfunction and is more likely to alert the physician to the possibility of cardiac disease. A symptomatology which exhibits the classic attributes of Heberden's angina is almost pathognomonic of ischaemic heart disease. Where the patient’s history includes a number of atypical components, however, the diagnostic sensitivity will decline considerably. Some clinicians consider that anginal pain of oesophageal origin is characterised by certain atypical elements that enable it to be differentiated from cardiac pain.5 In view of the many variants of angina, together with the diagnostic urgency which attends this type of pain, the physician will invariably elect to investigate the patient’s cardiac status first. While coronary angiography is generally regarded to be the definitive method of assessing atheromatous coronary disease, previous reports have shown that well over a quarter of the angiographic procedures performed for assessment of chest pain fail to show any significant coronary disease.6-7 With increasing attention being focused upon the oesophagus as the potential seat of angina like pain, a greater proportion of the oesophagologist’s work is given over to its investigation.

The incidence of oesophageal dysfunction among patients with non-cardiac angina like pain has been reported to be as high as 74%.8 As both disordered motility and gastro-oesophageal reflux are suggested causes of this type of pain, a variety of diagnostic tests have been developed to identify an oesophageal aetiology, though not all achieve the desired diagnostic sensitivity.9 While an overlap in the segmental distribution of the cardiac and oesophageal innervation might offer a possible explanation for some of the cardiac qualities often attributed to oesophageal pain, there seems to be a lack of objective evidence relating experimentally provoked oesophageal pain to the patient’s spontaneous symptoms. Furthermore, it must be appreciated that selective coronary angiography is only capable of imaging vessels of greater than 0-4 mm and therefore would normally be limited to visualising the epicardial and first septal vessels only. Consequently, a negative report does not necessarily exclude myocardial ischaemia. The term ‘microvascular angina’ has been attributed to diffuse small vessel disease of the myocardium. These patients may exhibit classic ST segment depression during exercise testing yet have no angiographic evidence of coronary disease.10 Furthermore, variant (Prinzmetal’s) angina, associated with spasm of the coronary vessels, may occur in the absence of appreciable atheromatous disease. As this is an intermittent phenomenon, it is best documented by protracted ECG where ‘spasms’ are characterised by transient elevation of the ECG-STEP segment.

Finally, there is some experimental evidence of a probable viscerocardiac reflex whereby
oesophageal events may influence myocardial performance. This further indicates the need to consider myocardial ischaemia in a proportion of patients with normal angiographic investigations.

We elected to investigate a representative group of patients referred to our oesophageal laboratories for assessment of recurrent angina like pain and normal coronary arteries. Our principal aim was to ascertain whether, irrespective of any baseline oesophageal anomalies, there was any reason to suppose a direct relation between spontaneous chest pain and oesophageal dysfunction. We chose to address this by simultaneously recording oesophageal motility and luminal pH over a 24 hour period while the patients remained normally active. In doing so, motility and pH events corresponding to spontaneous episodes of chest pain could be compared with time matched samples taken before and after pain to ascertain whether they exhibited significant variation. Throughout the period of oesophageal monitoring, we also employed a portable real time ECG analyser to identify ECG-ST segment shifts which would be indicative of myocardial ischaemia. In this way, any appreciable change in one or more of our recorded parameters which was unique to the period of symptomatic monitoring would suggest a probable cause and effect relation. The continuous 24 hour record might also disclose any diurnal trends peculiar to this group of patients.

Methods
Forty seven patients (21 men, 26 women) with a mean age of 50 years (range 34–63) referred to our laboratory for oesophageal manometric and pH assessment were studied. These patients had a six month or greater history of recurrent angina which was considered by the cardiologist to be sufficiently indicative of ischaemic heart disease to warrant full cardiological assessment. After treadmill ECG exercise testing, each patient underwent cardiac catheterisation and was subsequently reported to have normal intracardiac pressures and angiographically normal coronary arteries.

Before referral, barium radiology was performed to eliminate the likelihood of a neoplasia or other structural abnormality of the oesophagus, stomach, and duodenum as the cause of pain. As a prelude to ambulatory oesophageal studies, a manometric pressure profile was established for each patient using conventional station pull through manometry (SPTM). Within one week of their SPTM, each patient returned to be fitted with an ambulatory oesophageal motility and pH monitoring system together with a portable ECG-ST segment analyser. All patients were fasted overnight and those medications known or suspected to influence oesophageal performance were stopped 24 hours before investigation (48 hours for those taking β blockers). Each patient was monitored for a 24 hour period during which time they were requested to maintain an accurate diary, noting the time of onset and duration of any symptoms together with the nature and location of their discomfort and any associated activities. Their dietary intake was also documented together with the time they retired to and awoke from bed. They were advised to remain normally active throughout the period of monitoring and, within reason, encouraged to pursue those activities known by them to provoke symptoms. No restrictions were imposed upon dietary intake, which was documented in the patient diary, and each patient was permitted their normal intake of cigarettes or alcohol, or both, where appropriate.

SPTM
SPTM was performed using a triple lumen polyvinyl catheter with 1 mm (ID) side holes positioned at 5 cm intervals from the tip and radially orientated by 120° (Ormed UK, Arndorfer ESX3). This was continuously perfused with distilled water at 0.6 ml/minute using a low compliance pneumatic hydraulic system.[1] The catheter was connected via strain gauge pressure transducers (SE Laboratories, SEM 4-88) to a bank of biomedical amplifiers (SEM 4001). The output signals from the pressure amplifiers were digitised at 5 Hz per channel using a microcomputer (BBC Master Series, Acorn Computers Ltd).

Motility was recorded after a standard 4 ml wet swallow performed at 1 cm increments during stepwise catheter withdrawal from the stomach through to the cricopharyngeus. The manometric profile of the lower oesophageal sphincter was calculated from the mean pressure excursion at the point of end expiration and end inspiration and is quoted in respect of the mean fundal pressure. The amplitude and duration of pressure waves were calculated for the proximal, middle, and distal third oesophageal segments by taking the mean of 18 to 24 (six to eight in each of three channels) wet swallows, depending upon the oesophageal length. The percentage incidence of non-propagated pressure waves was determined from the temporal relation of pressure responses from adjacent catheter ports. Similarly, the incidence of double peaked pressure waves, defined as additional peaks greater than 10% of the primary wave, were also determined for each patient.

AMBULATORY MOTILITY AND pH MONITORING
Combined 24 hour ambulatory monitoring was performed using a lightweight belt mounted monitoring system of our own design. This system utilises a multilumen pH electrode (MI-508, Microelectrodes Inc) attached alongside a triple sensor transducer tipped pressure probe (16CT/S/3, Gaeltec Ltd). The combined pressure/pH probe assembly was introduced per nares into the oesophagus to monitor pressures at 5, 10, and 15 cm above the proximal margin of the lower oesophageal sphincter. The pH electrode was attached to the pressure probe so that it recorded pH midway between the middle and distal pressure elements (lower oesophageal sphincter +7–5 cm). This irregular positioning of our pH probe in the oesophagus was considered very necessary in order to avoid potential problems associated with prolonged contact between the pH glass bulb and
the metal shroud surrounding each pressure sensing element of the transducer tipped probe. Correct positioning of the pressure/pH probe assembly in respect of the lower oesophageal sphincter was achieved according to previous SPTM.

The pressure probe connects via a transducer control unit (modified S11, Gaeltec Ltd) to a Medilog four channel cassette recorder (4:24, Oxford Medical Systems). The miniature pH electrode uses a disposable ECG type skin reference electrode fixed to the patient’s chest. The mv potential developed by the pH probe is amplified by a small belt mounted adjustable pH meter then output to the fourth channel of the Medilog recorder. Before recording the signals on magnetic tape, the analogue pressure/pH signals are pulse width modulated (PWM) by modulator cards fitted within the recorder (AM-3, Oxford Medical Systems). The pressure and pH data were then replayed and demodulated at 60 times the recording velocity using a tape transport deck with demodulation amplifiers (PB2/PM3, Reynolds Medical). The analogue output signals from the replay deck were digitised at 240 Hz per channel using an eight bit analogue to digital converter (Greyhound and Micropower) before being stored to two 3½ inch floppy disks under control of a computer (BBC Master Series, Acorn Computers Ltd).

DATA ANALYSIS
The ambulatory data were analysed by microcomputer using our own software. This software permitted the 24 hour motility and pH record to be analysed either in entirety or as discrete epochs corresponding to different patient or monitoring circumstances. After keyboard entry of the desired sample time and duration, the software quantified the pressure data in terms of: the mean and maximum pressure wave amplitude and duration; the frequency of propagated and non-propagated (synchronous) contractions; the mean pressure baseline at two levels; and the sum integrated pressure wave area divided by the sample period – a useful index of contractile activity. Similarly, the corresponding pH data were also quantified according to the frequency and duration of pH excursion below selected pH values in the range of pH 1–pH 8. During analysis, those motility and pH variables influenced by sample duration were normalised.

Using the patient’s diary as a reference, the motility and pH events during reported episodes of spontaneous chest pain were compared with time matched samples taken before and after chest pain events. These ‘control’ samples were selected, where possible, 30 minutes either side of the pain event and corresponded to pain free periods of monitoring. Statistical comparison was then performed on these data to ascertain whether any of our recorded pressure or pH parameters, or both, differed significantly from their baseline values at the time of chest pain.

STATISTICAL METHODS
Comparison of manometric data obtained by SPTM was based on the large sample test for differences between means. Two way analysis of variance was used with ambulatory oesophageal data to establish significant differences between painful and non-painful control samples. The incidence of reflux disease within our study was determined by separate analysis using the six discriminant reflux variables of the DeMeester scoring method. 13

ECG-ST SEGMENT ANALYSIS
Real time analysis of the patient’s ECG-ST segment was performed using the Star Monitor One (Q-MED) which employed a modified V3V5 lead configuration. Transient ischaemic events were defined according to the instrument’s microprocessor based algorithm which identifies ischaemia as: 1 mm or greater horizontal or down sloping depression of the ST segment 80 ms after the J point and lasting at least 40 seconds. Documented examples of the ECG signal (five second duration) are stored in the instrument’s memory upon detection of an ischaemic event, or alternatively, when the patient event button is used. A detailed disclosure provides a hard copy of the ECG trace corresponding to ischaemic events and annotated with the time of onset, duration, and heart rate. Similarly, where the patient event button is used, the ECG trace provides confirmation of the presence or absence of ischaemia.

Results

SPTM
The group mean pressure wave amplitude and duration together with the pressure profile of the lower oesophageal sphincter are summarised in Table 1, where they are contrasted with those values of normal healthy subjects obtained using comparable instrumentation. 14 The mean oesophageal pressure wave amplitude was seen to be systematically lower in our patients compared with normal subjects, though exhibiting a greater duration throughout the distal segment.

The group mean propagation velocities, measured at 2 cm increments throughout the oesophageal body, are plotted in Figure 1. While the mean propagation velocity from Richter’s study group 14 are given for two levels only (prox-
of pain, at these levels they do not differ significantly from our patients. The shape of this velocity graph is also characteristic of that previously reported by Humphries and Castell in their earlier study of normal subjects. 13

In Table II the mean percentage incidence of pressure wave anomalies observed in our patient group at SPTM are listed and again contrasted with their reported incidence among Richter’s normal subjects. The only notable anomaly observed in our patients was a significantly higher than normal incidence of simultaneous contractions (p<0.001). This apart, our patients where found to have noticeably fewer double peaked contractions and were free of other signs of obvious dysmotility.

AMBULATORY MOTILITY AND pH MONITORING
Each patient was monitored over a 24 hour period which began between 9.00 and 10.30 am. From our 47 patients, 46 complete sets of data were successfully obtained. The remaining patient was excluded from the study because of partial data loss associated with instrument failure. Thirty-five of the 46 patients experienced at least one episode of chest pain during their monitoring session which was described as characteristic of their usual symptoms. A number of our patients experienced more frequent chest pain (range 1–5 episodes) providing a total of 59 pain events.

Only 37% of these 59 pain events showed any direct relation to reported physical effort. A similar proportion (33%) occurred while the patient was at rest – typically while watching evening television. A further 14% of symptoms occurred either during or immediately after the patient’s main meal and 7% were reported to be precipitated by minor emotional upset or agitation. The remaining 9% occurred while the patient was in bed.

To determine any causal relation between recorded oesophageal events and symptoms, individual motility and pH parameters, corresponding to episodes of chest pain, were compared with those of our elected time matched control samples. Table III shows the group mean (SD) corresponding to the motility parameters. Though there was a considerable amount of interpatient variation noted in both the control and chest pain samples, we were unable to identify any motility parameter which differed significantly at the time of symptoms from the control samples. Though a small number of our patients showed some moderate change in one or more motility parameters at the time of pain, these changes were diminutive compared with the natural variations seen throughout the monitoring session and which were not associated with any reported symptoms. Table IV lists the group mean (SD) for the percentage sample time where the pH remained below a series of threshold values in the range of pH 2.0–pH 5.0 together with the integrated area under the pH curve at each value. Once again, analysis of variance did not show any significant difference in reflux exposure during chest pain compared with our asymptomatic control samples.

Each pH record was also individually scored to determine the incidence of gastro-oesophageal reflux disease within our study group. Almost 70% (32 of 46) of our patients were shown to have a pathological degree of reflux (group mean (SEM) De Meester score 135.59 (21.61)), which was associated with reflux occurring predominately during the supine monitoring position.
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(mean (SEM) percentage supine time=35.04 (14.95); percentage erect time 9.59 (1.31)).

In Figure 2 each patient’s composite reflux score is plotted against their end expiratory lower oesophageal sphincter pressure. This showed a poor correlation ($r=0.126$).

**AMBULATORY ECG-ST SEGMENT ANALYSIS**

The occurrence of ischaemic ECG changes in our patient group throughout the 24 hour monitoring period was determined according to the algorithm of the portable real time analyser. Ischaemic events (ST $\downarrow$ $>1$ mm) were detected in 10 of our 47 patients and were accompanied by chest pain in only two. Independent cardiological assessment of the ECG traces corresponding to these purported ischaemic events showed that in four patients ischaemia was detected at a time when the ECG signal was severely distorted by artefact, and in another patient the pre-existence of left bundle branch block (LBBB) excluded such analysis.

While the five second ECG trace which accompanies a detected ischaemic even would not normally be considered sufficient for reliable interpretation, it was the cardiologist’s opinion that of the remaining five patients, the trace was unequivocally ischaemic in three, one of whom exhibited T-wave inversion on effort (Fig 3 (A)) and regarded probable in the remaining two. On no occasion were we able to show concomitant dysmotility or gastro-oesophageal reflux during either the confirmed or dubious ischaemic events.

**Discussion**

Through a protracted period of ambulatory monitoring, we have shown that parameters of oesophageal motility and luminal pH, recorded in a group of patients with non-cardiac angina like pain, showed no significant change at the time of spontaneous symptoms when compared with time matched asymptomatic data samples.

Concurrent real time ECG-ST segment analysis showed classic ECG-ST segment depression in a small though significant proportion of our patients, with equivocal ischaemic ECG events detected in a number of others. With the exception of two patients, these ECG events were not accompanied by pain and were frequently noted in patients who, at other times during the ambulatory period, reported symptoms in the absence of ECG events. There was no apparent correlation between these detected ECG events and recorded patterns of reflux and motility.

We consider that the absence of any appreciable change in motility parameters at the time of spontaneous chest pain questions the clinical relevance of specific motility anomalies that are often demonstrable in these patients by conventional ‘in clinic’ manometrics, and which have previously been supposed to be the cause of their pain. The absence of any convincing relation between exposure to reflux and episodes of chest pain suggests that their pain is not directly associated with episodes of oesophageal acidification. Individual analysis of each patient’s 24 hour pH record, however, showed a notably high incidence of pathological gastro-oesophageal reflux within this study group. Moreover, and almost without exception, the most severe episodes of reflux occurred during the supine monitoring period, whereas pain events occurred mostly by day. In view of this, we believe that gastro-oesophageal reflux should not be discounted as a contributory agent of pain in these patients without further consideration of the daytime effects of this prolonged exposure to supine reflux.

Our choice of control samples taken before and after pain were arbitrary, but consistent, aiming to reference pain data against time matched non-pain data. We appreciate that such an approach might overlook immediate or distant events possibly pertinent to the onset of symp-
Illustrations of increments in amplitude

Amplitude variation not were on period, however, at variation during patients. In line patients, comparable. In line asymmetry some the data, comparison may as

Manual to provide ambulatory pressure/pH data, however, the presence of a number of observations is associated with inappropriate data selection.

A change in attitudes regarding the normal range of pressure wave parameters recorded by conventional SPTM has required us to reconsider the incidence of motor disorders such as nutcracker oesophagus which have previously been associated with this type of chest pain. Therefore, for the purpose of comparison only, our SPTM data have been compared with the normal values reported in the study of Richter et al.

As shown in Figure 4, however, we have noted considerable variability in the peristaltic wave amplitude when measured at different points throughout the oesophagus. In Richter's study, specific points above the lower oesophageal sphincter were chosen, irrespective of oesophageal length, whereas the values presented here represent the mean value corresponding to division of the oesophagus into thirds. Furthermore, our lower oesophageal sphincter pressure profile was obtained using a catheter with pressure ports having the same radial orientation, and may not therefore account for tonic radial asymmetry within this segment. While such comparison may provide some perspective for our data, in view of the positional differences between the two experiments, we accept that some of these parameters may not be directly comparable.

Our initial observation at SPTM of a significantly greater than normal incidence of synchronous contractions might have pointed towards dysmotility as the probable seat of pain in our patients. In line with other motility parameters recorded during the ambulatory monitoring period, however, these showed no significant variation at the time of pain.

The lower oesophageal sphincter pressures were not recorded during the period of ambulatory monitoring, and we are therefore unable to comment on the clinical relevance of the higher than normal pressure profile noted in many of our patients at SPTM. Hypertonicity of the lower oesophageal sphincter has previously been documented in patients with chest pain, however, and may present an area for future research. The absence of any correlation between our patients' individual end expiratory lower oesophageal sphincter pressure and composite reflux score (Fig 2) is not a new finding. Considerable overlap has previously been reported between reflux and non-reflux patients, especially at lower oesophageal sphincter pressures greater than 6 mm Hg. This supports the hypothesis that in many patients reflux occurs through inappropriate or transient sphincter relaxation rather than an inability to generate an adequate resting pressure.

Naturally, patient selection is a major consideration when attempting to relate oesophageal disorder to the development of this type of chest pain. Possibly most fundamental to this is the element of subjectivity in both the patient's description and the clinician's interpretation of chest pain, as these are most likely to result in the misclassification of angina.

As angina is a symptom complex rather than a disease, the diagnosis would be based upon clinical features such as the quality, distribution, and precipitating agents of the patient's pain together with predisposing risk factors such as age, gender, and family history. The extent to which objective evidence of ischaemic heart disease is then sought would depend upon a number of considerations, not least of which are the cost, availability, and attendant risks to the patient from authoritative cardiological investigation such as coronary angiography. Consequently, the patient complaining of chest pain would not normally be subjected to unpleasant and costly invasive procedures without there being strong clinical grounds to suspect cardiac disease. The nature of symptoms experienced by patients in our study were sufficiently characteristic of angina to necessitate invasive angiographic investigation before ischaemic heart disease could satisfactorily be eliminated.

The rationale for subsequent oesophageal investigation of these patients was based upon the pervasive appreciation among physicians that symptoms of oesophageal dysfunction, especially dysmotility and gastro-oesophageal reflux, may mimic those of ischaemic heart disease. However, an oesophageal aetiology is frequently inferred from either conventional manometric findings, typically obtained while the patient is asymptomatic, or experimental provocation of like symptoms after oesophageal distension, acid instillation, or the use of pharmacological agents.

It has often been argued that experimental provocation techniques are non-physiological and, therefore, possibly not representative of the oesophageal event accompanying spontaneous symptoms. These criticisms have been addressed, to some extent, by the use of long term ambulatory intraoesophageal pH monitoring in the assessment of chest pain, and, more recently, combined oesophageal motility and pH monitoring. There seems to be some disparity, however, in the proportion of patients in

Distance above lower oesophageal sphincter (cm)

![Graph illustrating the variation in group mean pressure wave amplitude when measured at 2 cm increments throughout the oesophageal body.](http://gut.bmj.com/Downloaded from http://gut.bmj.com/ on November 28, 2017 - Published by group.bmj.com)
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whom either acid reflux or dysmotility could be attributed to the occurrence of spontaneous chest pain. A possible explanation may lie in the criteria used by different studies to show a causative association.

We consider it unreasonable to consider oesophageal events recorded during pain as directly causative unless they show a clear temporal relationship with the onset and duration of each pain event and are repeatedly seen to deviate significantly from their baseline state. That is to say, motility and reflux anomalies that are noted at the time of symptoms should be considered in terms of their baseline activity, irrespective of whether this falls outside the currently accepted normal range.

We feel our experimental design has given appropriate consideration to the clinical relevance of baseline motility anomalies in these patients by using each patient as his or her own control. In this way, an abnormal incidence of, say, non-propagated activity or abnormally high amplitude contractions at the time of pain is not considered causative where a similar occurrence is noted in the control samples taken before and after an episode of pain when the patient was reported to be pain free. Therefore, in view of the absence of any appreciable change in motor activity at the time of spontaneous chest pain, it is unlikely that dysmotility is directly implicated as the agent of pain in our patients.

As myocardial ischaemia, and indeed infarction, may occur in the absence of angiographically evident coronary disease, there continues to be a risk of exonerating the heart on the grounds of a normal coronary angiogram when, through an as yet unspecified mechanism, the patient may in fact suffer ischaemic pain from the heart.

Ambulatory ECG-ST segment analysis showed evidence of myocardial ischaemia in at least 10% of our patients. These purported ischaemic ECG events were generally not accompanied by chest pain, though in all but one patient they were associated with moderate physical exertion (heart rate >110 bpm). They were characterised by depression of the ECG-ST segment, supportive of epicardial or microvascular disease, rather than the appreciable ST segment rise that is more characteristic of coronary artery spasm.

In the two patients who did experience pain at the time of detected ECG changes, it is probable, though not diagnostic, that their chest pain is of cardiac origin. In the others, however, the clinical importance of predominantly ‘silent’ ischaemic ECG events in the absence of angiographic evidence of coronary disease remains unclear.

Finally, we should like to acknowledge the comments made by Valori. He suggests that oesophageal motility as a cause of chest pain is probably overstated and also highlights some of the inadequacies of previous investigative approaches in the patient with non-cardiac chest pain.

It is unfortunate that of the plethora of techniques which aim to identify the oesophagus as the seat of angina like chest pain, few provide convincing evidence of a direct cause and effect relation. Clearly, motor anomalies should not be suggested as the cause of pain unless they are unique to, and repeatedly correlate with spontaneous pain of the type the patient presented with.

With regard to the taxonomy of chest pain, we feel it appropriate, until proved otherwise, to assume that chest discomfort which is constricting in quality and predictable on effort to be termed angina. The patient who describes a persistent retrosternal ache, a brief sharp sub-mammary stabbing pain, or recurrent epigastric or substernal burning might less reasonably be termed angina. In view of the numerous angina equivalents, however, it is possible (though accepted less typical) that almost any type of pain, from the epigastrium to the crown, may be of cardiac origin and therefore, according to current reports, may aptly be termed angina.

DGH thanks the National Heart Research Fund (NHRF) for their generous support of this clinical study.


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*Gut* 1992 33: 79-86
doi: 10.1136/gut.33.1.79

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