PRACTICE OBSERVED

Practice Research

Is it a coronary?

JAMES K FINLAYSON, DAVID SHORT

The patient who has an attack of pain in the front of the chest that is not very severe, and may indeed have subsided, presents the general practitioner with a difficult problem. The patient frequently describes the episode as an attack of "acute indigestion," although he may be secretly worried about the possibility of a heart attack. He does not appear to be ill and there may be no abnormal cardiovascular signs. At this point the patient often describes himself as "a fraud." Yet the doctor may be slightly uneasy, for it may be an attack of spontaneous angina or even a small myocardial infarct. What should he do? He may be over-reacting if he arranges immediate admission to a coronary care unit. Should he try to obtain an urgent opinion from a cardiologist? Or should he attempt to reach a diagnosis himself?

A cardiologist (DS) has recently studied patients at home and as outpatients who had had a total of more than 400 such attacks.[1] The purpose was to find out for the assistance of general practitioners which features of the history, of the examination, and of the electrocardiographic (ECG) findings correlated best with the diagnosis of an acute coronary attack. (Acute infarction and spontaneous angina were grouped together under the heading "coronary attack" because the prognosis of the two conditions is similar.[2-4]) From the results of the study it was concluded that a coronary attack was unlikely if the pain was affected by breathing, twisting, or bending, if it was felt at more than one site, or if it had a different epicentre from that of a previous definite attack of myocardial infarction or angina, or if the ECG was absolutely normal. On the other hand, if the patient had a history of sweating, or had signs of dyspnoea, shock, or tachycardia in association with chest pain this suggested a coronary attack. Changes in blood pressure were unhelpful in this type of case. ST elevation, T wave inversion, or ST depression of ischaemic pattern strongly indicated a coronary attack when there was no previous history of cardiac pain. In patients with a previous history of infarction or angina such findings were of less value; though deterioration of the ECG by comparison with the latest previous tracing strongly suggested a fresh coronary attack.

Our study was designed to test the conclusions of the first study by critically analysing the findings in a further group of patients who presented over the past three years. The first study covered 10 years and during much of that time there was no coronary care unit (CCU). Throughout the three years of our study, however, a coronary care unit was available for all patients under 65 years of age who presented with suspected coronary attacks. Thus none of the patients in our series were thought by their general practitioners to have symptoms of myocardial ischaemia that warranted immediate admission to hospital.

Cardiac Department, Aberdeen Royal Infirmary, Aberdeen AB9 2ZB
JAMES K FINLAYSON, BSC, FRCP, consultant physician
DAVID SHORT, MD, FRCP, consultant physician

Patients and methods

Ninety-eight patients were seen at home for 100 episodes of acute anterior chest pain that suggested to the general practitioner a possible coronary attack. None was considered ill enough to be admitted urgently to hospital. In 57 episodes of chest pain the patients were seen within 24 hours of the latest or most severe attack, in 24 episodes they were seen between 24 and 72 hours, and in 19 episodes between three and seven days after the onset of the attack. Twenty-two patients gave a history of previous infarction and 22 a history of angina of effort without myocardial infarction; 56 gave no history of previous cardiac pain. Patients in 57 episodes had had an earlier ECG examination. The ages of the patients ranged from 29 to 96 years with a mean of 62 years; 52 were men and 46 women.

The history, physical examination, and ECG recording were undertaken as described.[1] A note was made of the site and distribution of the pain, and its relation to pain of any previous angina or myocardial infarction, and also whether the pain was affected by breathing, twisting, or bending. Associated symptoms were also noted, particularly sweating and nausea. A heart rate of less than 60 beats per minute was classified as bradycardia and a rate of 100 beats or more as tachycardia. The serum aspartate aminotransferase concentration was measured in patients in half the episodes, though the results

could not be used for immediate diagnosis because they could rarely be obtained in under 12 hours.

A working diagnosis was made at the consultation using any previous ECG recording that was available, and the patient was managed by (*a*) immediate admission to hospital; (*b*) continued observation at home with serum enzyme estimation, a further ECG recording, and other investigations as indicated; or (*c*) reassurance.

We were in contact with either the general practitioner or the patient over the subsequent three to seven days. In 47 episodes a further ECG recording was made, and in 47 episodes the patients were followed up for over a month or until death. When all the evidence that was judged necessary to establish the diagnosis was available from the general practitioner—from hospital admission or outpatient follow-up, and from previous hospital records—the episode was finally diagnosed in one of four categories.

A final diagnosis of *acute myocardial infarction* (30 episodes) was made if there was a history of chest pain consistent with ischaemic heart disease as reported in a review of the earliest symptoms of coronary heart disease,[4] together with either a changing ECG recording consistent with infarction or a rise in the aspartate aminotransferase concentration to at least twice the upper limit of normal 24 to 48 hours after the onset of chest pain. A diagnosis of *spontaneous angina* (25 episodes) was made if there had been an attack of chest pain at rest similar in character to previous or subsequent attacks of angina of effort or myocardial infarction, together with current or subsequent ECG evidence of ischaemic heart disease but without the characteristic changes of acute infarction and without an appreciable rise in the aspartate aminotransferase concentration during the episode. A diagnosis of *non-coronary disease* (31 episodes) was made if the pain was inconsistent with ischaemic heart disease or there was evidence of an alternative explanation for the pain—provided that the ECG recording was normal (or if abnormal the abnormality could be explained on other grounds, such as previous myocardial infarction, left ventricular hypertrophy, or acute pericarditis) and provided there was no appreciable rise in the concentration of aspartate aminotransferase. A diagnosis of *doubtful coronary attack* (14 episodes) was made if the pain was consistent with ischaemic heart disease but there was no ECG or other evidence to support it and no other explanation for it.

Results

The history, physical examination, ECG recorded at the time of examination, and evidence of deterioration by comparison with any earlier ECG recording available at the consultation was correlated with the final diagnosis. Episodes in patients who had a history of previous infarction or angina were analysed separately from those in which there was no such history.

HISTORY OF PREVIOUS MYOCARDIAL INFARCTION OR ANGINA

There were 44 episodes in the patients with a history of myocardial infarction or angina pectoris (fig 1). In two the history of angina had not been noted by the general practitioner; in one the symptom was slight, and in the other very recent. In 14 episodes the final diagnosis was acute infarction, in 22 episodes spontaneous angina, in six episodes non-coronary disease, and in two episodes the diagnosis was doubtful.

Episodes in which the pain had a different epicentre from that of previous infarction or angina or was pleuritic in type—In five episodes the pain had a different epicentre from that of previous infarction or angina and in one it was pleuritic. In these six episodes there was, on the basis of the previous study, prima facie evidence that the pain was not coronary in origin. In five of the six episodes the final diagnosis was of non-coronary pain; in the sixth the diagnosis was doubtful. None of these patients had any associated symptoms such as sweating or nausea. On examination one was breathless at rest and another had a bradycardia (he was not taking drugs). In two of the episodes the ECG showed a raised ST, but an earlier ECG recording showed that this was due to a cardiac aneurysm.

Episodes in which the pain had a similar epicentre to that of previous infarction or angina—In 38 of the 44 episodes the pain had a similar epicentre to that of the previous infarction or angina and was not pleuritic, thus providing prima facie evidence of fresh infarction or spontaneous angina. In 14 episodes the final diagnosis was acute infarction, in 21 spontaneous angina, in one non-coronary disease, and in two doubtful. There was a history of sweating in two cases, and a finding of dyspnoea in five, tachycardia in one, and bradycardia

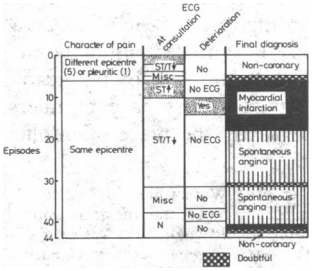

FIG 1—Relation between the history, the electrocardiographic (ECG) recording, and the final diagnosis in patients with a history of myocardial infarction or angina pectoris. ECG: stippling = a raised ST. N = Normal. ST/T↓ = ST depression or T inversion of ischaemic pattern. Misc = Miscellaneous abnormality. Deterioration: No ECG = no previous ECG recording.

in one. In only four of the 14 episodes that were finally diagnosed as infarction did the ECG show a raised ST. In one episode it was normal at the time of consultation and only 72 hours later showed abnormalities characteristic of a subendocardial infarction.

NO HISTORY OF PREVIOUS INFARCTION OR ANGINA

There were 56 episodes in patients with no history of infarction or angina (fig 2). In 16 the final diagnosis was acute infarction, in three spontaneous angina, in 25 non-coronary pain, and in 12 doubtful.

Episodes in which the pain was pleuritic or multifocal—In 11 episodes the pain was pleuritic and in another it occurred in more than one site. In these 12 cases there was, on the basis of the previous study, prima facie evidence that the pain was not coronary in origin. In 11 episodes the final diagnosis was indeed non-coronary pain (three had pericarditis), but one patient proved to have acute infarction. This

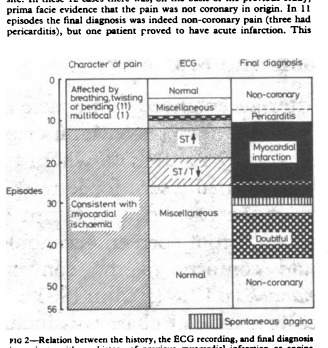

FIG 2—Relation between the history, the ECG recording, and final diagnosis in patients with no history of previous myocardial infarction or angina pectoris.

patient had no symptoms other than chest pain and no abnormal physical signs, but her ECG recording showed a raised ST segment with subsequent development of abnormal Q waves and an appreciable rise in enzyme concentration. One of the patients diagnosed as non-coronary complained of sweating and nausea, two had dyspnoea, and one tachycardia. Of the three patients with pericarditis, one had a raised ST, another showed major ST/T abnormalities of ischaemic type, and the third had only minor ST/T depression. Of the remaining eight non-coronary patients, three had ECGs showing minor ST/T abnormalities and in five the tracing was normal.

Episodes in which the pain was consistent with myocardial ischaemia—In 44 of the 56 episodes the pain was consistent with myocardial ischaemia. In 15 the final diagnosis was acute infarction, in three spontaneous angina, in 14 non-coronary, and in 12 doubtful. Three patients gave a history of sweating and nausea: in two the final diagnosis was acute infarction and in one it was doubtful. Nine gave a history of sweating alone: in four the final diagnosis was acute infarction, in four it was non-coronary, and in one doubtful. Eight gave a history of nausea alone: in four the final diagnosis was acute infarction, in three non-coronary, and in one doubtful.

In three episodes the patient seemed to be dyspnoeic: in two the final diagnosis was acute infarction and in one non-coronary. Three patients had a tachycardia: in one the final diagnosis was acute infarction, in one spontaneous angina, and in one doubtful. Three patients had a bradycardia: in one the final diagnosis was infarction, and in two doubtful. The one patient who was in shock had an acute infarction.

In seven episodes the ECG recording showed a raised ST, and the final diagnosis in each case was acute infarction. A further seven showed major ST/T abnormalities of coronary type. In five the final diagnosis was acute infarction, in one spontaneous angina, and in the fifth it was doubtful. In 14 episodes the ECG recording showed miscellaneous abnormalities, such as minor ST/T changes or left ventricular hypertrophy or bundle branch block: in three the final diagnosis was acute infarction, in two spontaneous angina, in two non-coronary pain, and in seven it remained doubtful. In 16 episodes the ECG recording was normal. In 12 the final diagnosis was non-coronary disease and in four it was doubtful.

Discussion

Attacks of chest pain are common, and only a few are due to coronary arterial disease. These, however, are potentially fatal so that attacks that raise a mere suspicion of coronary disease present a considerable problem to the general practitioner. Is it possible to offer guidelines for diagnosis and action? Unfortunately, it is often impossible to reach a firm diagnosis in such cases, even when the facilities of a coronary care unit are available. The difficulties are obviously greater if the patient is investigated outside hospital. We do not claim that our diagnoses are beyond dispute. Nevertheless, we believe that they are as accurate as can be achieved with the techniques that are available for a study in the community. Most of the patients were seen on more than one occasion, and over 80% had two or more ECG recordings.

The salient conclusions are the importance of the history, particularly in patients with previous infarction or angina (fig 1), and the importance of the ECG recording in patients with no history of cardiovascular disease (fig 2). Symptoms in addition to the pain and the findings on examination were relatively unhelpful.

History—Of the 12 episodes in which the patient described the pain as being affected by breathing, twisting, or bending, 11 were finally diagnosed as non-coronary (though three patients had pericarditis). In one patient whose symptoms presented like pericarditis—pain affected by respiration and a raised ST segment—acute infarction was eventually diagnosed.

None of the five patients who gave a history of chest pain having a different epicentre from their previous cardiac pain showed evidence of acute infarction or spontaneous angina. Conversely, of the 38 patients whose pain had the same epicentre as that of their previous cardiac pain, 35 were finally diagnosed as having an acute infarction or spontaneous angina. This shows the importance of recording the precise site of the pain (or discomfort) when a patient is diagnosed as having myocardial infarction or angina. A vague description such as "pain across the chest" or "pain in the chest referred to the left arm" is inadequate. One of us has for many years used a code to record the site of pain.[5] In this series of patients (in contrast to the earlier study[1]) a history of sweating or nausea was too equivocal to be of value in diagnosis.

Examination—Symptoms of dyspnoea or tachycardia suggested a coronary attack but were too infrequent to be of much help. Most patients with small myocardial infarcts or attacks of spontaneous angina had no abnormal physical signs.

ECG recording—In patients with no previous history of cardiac pain a raised ST, T inversion, or ST depression of ischaemic pattern was virtually diagnostic of acute infarction. Sometimes the raised ST was very slight, so it is important to be able to recognise the earliest appearance of this important abnormality.[6] In patients with a history of previous infarction or angina the ECG recording was inconclusive. At the other end of the scale a completely normal recording virtually excluded infarction, though it did not entirely exclude an attack of spontaneous angina. Other ECG patterns, such as minor ST/T depression, bundle branch block, or left ventricular hypertrophy, were unhelpful. When the ECG recording deteriorated from the previous tracing this strongly suggested a fresh infarction, but if there was no deterioration this did not exclude a coronary attack.

Action—The diagnosis frequently remained in doubt after the first examination. If the pain was still present or had only recently subsided and if the suspicion of a coronary attack was strong we usually advised admission to hospital. If, on the other hand, there had been no pain for 24 hours we usually left the patient at home with instructions to call the doctor if the pain returned. In such cases we usually arranged to have the serum enzyme concentration estimated at the appropriate time and repeated the ECG examination in 24 to 48 hours.

Conclusions

Slight or brief attacks of chest pain present the general practitioner with a difficult problem in diagnosis and management. We have analysed 100 such episodes, none of which was considered by the general practitioner to be sufficiently severe or suggestive of myocardial ischaemia for immediate admission to hospital. We tried to determine which features of the history, clinical examination, and electrocardiographic (ECG) findings correlated best with the final diagnosis. In 44 episodes the patient gave a history of previous angina or infarction. The final diagnosis was myocardial infarction in 30 episodes, spontaneous angina in 25, non-coronary pain in 31, and uncertain in 14. In patients who had suffered from previous cardiac pain the history was crucial, but a single ECG recording was rarely conclusive. In patients without previous cardiac pain the history sometimes indicated an alternative diagnosis, but was usually equivocal. The ECG recording, on the other hand, was frequently decisive. Clinical examination was usually unhelpful.

References

1 Short D. Diagnosis of slight or subacute coronary attacks in the community. *Br Heart J* 1981;45:299-310.
2 Pedoe HT, Clayton D, Morris JN, Brigden W, McDonald L. Coronary heart-attacks in East London. *Lancet* 1975;ii:833-8.
3 Schroeder JS, Lamb IH, Harrison DC. Patients admitted to the coronary care unit for chest pain; high risk sub-group for subsequent cardiovascular death. *Am J Cardiol* 1977;39:829-32.
4 Norland R, Nyquist O. Patients treated in a coronary care unit without acute myocardial infarction. Identification of high risk sub-group for subsequent myocardial infarction and/or cardiovascular death. *Br Heart J* 1979;41:647-53.
5 Short D, Stowers M. Earliest symptoms of coronary heart disease and their recognition. *Br Med J* 1972;ii:387-91.
6 Short D. The earliest electrocardiographic evidence of myocardial infarction. *Br Heart J* 1970;32:6-15.

(Accepted 20 November 1981)

Pathology of Partnerships

All partnerships are brittle

DAVID METCALFE

Most partnerships are benign, but some are malignant. The prevailing ambience of most partnerships is of friendship, co-operation, and comradeship in which ideals are shared and mutually agreed standards of behaviour are accepted. Although this happy state of affairs may account for most partnerships there is an important minority where that ambience cannot be found, and the relationships run the gamut from barely veiled hostility, through armed neutrality, to studied indifference. Explicit fission, where the partnership actually breaks up, although increasing, is still uncommon. Disguised fission, where members leave, is much more common.

Why do some partnerships become hostile environments for their members? There are two different sets of reasons. Firstly, partnership is a concept pertaining to commercial endeavours and denotes a sharing of capital investment, of input of effort and various sorts of expertise, a sharing of risk, and, one hopes, a sharing of the profits. Nowadays in many practices there is no capital investment, and even in those with privately-owned premises the nature of that investment is essentially different from the "risk capital" put up by partners in business. The input of expertise is uniform rather than complementary. There is a poor correlation between effort and profit. Most of the forces that bind partners in commerce and industry are absent from general practice. The second category of reasons concern the nature of the "learned professions" in general and of medicine in particular. A professional person is responsible only to himself or herself. This is enshrined in the independent contractor status of the general practitioner. Registered medical practitioners instinctively shy away from any idea that they should monitor each other's standards of work. Differences in performance are seen only as differences of "style" that can be enshrined in the concept of clinical freedom. All these characteristics militate against the concept of partnership: each partner's performance is within the realm of his personal privacy.

For these reasons all practices are in fact brittle. (Of course, before the Health Service the much rarer partnerships between doctors did conform largely to the commercial ethic of partnership. It is also interesting to remember that until quite recently in many States of America partnership practice was actually illegal.) All partnerships carry within them the seeds of their own destruction. Germination of those seeds is almost always brought about by one of two stimuli: money or work load.

Department of General Practice, University of Manchester, Manchester M13 0FW
DAVID METCALFE, MB, FRCGP, professor and head of department

Money and work load

The profits of the practice are either equally divided or shared out with some sort of a differential. Seldom if ever does this sharing reflect, as it would in business, the individual's share of the equity. If there are differentials they are usually based on the concept of seniority or differentials in work load. Seniority is an elusive concept that enshrines an element of recognition of pioneering (if the senior partner did in fact start or build up the practice), or administrative responsibilities, or expertise gathered in years of experience. The junior partner, however, knows that his arrival has been instigated by the senior partner largely for his own benefit, would be prepared to take his share of the administrative chores—particularly if he thinks he could do them better than the senior—and may well feel that his knowledge of technical medicine outweighs the other's years of experience. Initiation by disadvantage (the "flagging syndrome") is a British tradition, but there is little evidence that people learn by this sort of suffering. The resentments that build up, however, can certainly be stored for a very long time. The claim to a differential on the basis of better patient care is rarely if ever substantiated for the simple reason that neither senior nor junior partner is prepared in the circumstances to allow the other to make a critical judgment of his work.

Work load as a bone of contention or a seed of destruction operates even when there is financial parity and certainly can exacerbate the situation where there isn't. Since doctors are not equipped with order books or tills the normal commercial monitors of work load are not available. Indeed, it is surprising how few practices actually keep log books to analyse work patterns. In any case, if there is no measure of quality, quantity of work load is a poor measure of the effort put in and therefore of the rewards deserved. Quantitative work loads do, as a matter of fact, vary widely—much more so than can be accounted for by the variation in the registered population. The brittle partnership is characterised by its members having dark suspicions that the others are not working as hard.

If the problems of partnership absorb, as they seem to, an appreciable amount of doctors' emotional and intellectual energy, and if cordial relations can be maintained only by studious care being taken not to compare notes on clinical activity, the game may not be worth the candle. The theoretical benefits of group practice might be more readily obtained without partnership. Group practice in which expenses are pooled but each doctor retains his own earnings, which accrue to him for dealing only with his own registered patients during normal working hours, might avoid these stresses. Arrangements to cover the early stages of an additional member's career with the group, or late stages of a senior winding down his commitment, would not be difficult to arrange. Similarly, arrangements could be made for coping with illness, holidays, and other absences.) Continuity of care would be restored. Meanwhile, interpersonal relationships would not be threatened by comparing notes, sharing ideas, asking for advice, and even performing audit within the group.