Diagnosis of slight and subacute coronary attacks in the community

DAVID SHORT

From the Cardiac Department, Aberdeen Royal Infirmary, Aberdeen

SUMMARY A series of 456 episodes of spontaneous chest pain not considered by the primary medical attendant to be sufficiently severe or suggestive of coronary disease to warrant admission to hospital has been studied prospectively in an attempt to provide guidelines for diagnosis and management. A final diagnosis of acute myocardial infarction was made in 40 per cent, spontaneous (or unstable) angina in 15 per cent, doubtful coronary attack in 12 per cent, and non-coronary chest pain in 33 per cent of the episodes.

A diagnosis of myocardial infarction or spontaneous angina could never be made on the basis of a single feature, but demanded careful evaluation of the total evidence provided by the site and character of the chest pain, the associated symptoms, the clinical findings, and the electrocardiogram.

A guide to diagnosis based on the findings of this study is set out in Tables 7 and 8.

Attacks of chest pain which are not very severe or prolonged and yet are distressing enough to make a patient consult his general practitioner present a difficult problem in diagnosis and management. Severe and prolonged chest pain calls for immediate action whatever its cause. But a relatively mild attack of cardiac pain, once it has subsided, usually leaves the patient feeling perfectly well; indeed he often calls himself “a fraud”. Nevertheless, such a symptom cannot be ignored, for even slight coronary pain may herald major infarction or sudden death. Indeed, attacks of spontaneous angina are almost as serious as episodes of frank infarction. On the other hand, most chest pains are non-cardiac and unimportant. That is the problem. If the patient is reassured inadvisedly the result may be disastrous, but if unnecessary precautions are taken he may be greatly inconvenienced and even left with an ineradicable anxiety regarding his heart. There is an urgent need for guidelines to help general practitioners decide whether an attack of chest pain is cardiac or not.

The term “coronary attack” is used here to denote either a myocardial infarct or a prolonged episode of myocardial ischaemia. It includes what used to be called “acute coronary insufficiency” and is now usually referred to as spontaneous, or unstable, angina; in short all that Proger embraced in his term “acute coronary heart disease”.

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The difficulty in trying to offer advice in this field is that it is frequently impossible to reach a firm diagnosis. This is so even when all the facilities of a coronary care unit are available. The problem is obviously greater when the attack is slight and the patient treated at home.

Episodes studied

This is a report of a prospective study started 10 years ago of 456 consultations on patients who presented to their general practitioner with one or more attacks of spontaneous chest pain and who were referred for specialist cardiological opinion. Each attack was considered by the general practitioner to raise a suspicion of coronary disease, though in none was the diagnosis regarded as definite or the patient ill enough to require admission to hospital, apart from a very few who insisted on remaining at home. Half the patients were seen within three days of the onset of pain and the remainder between three and 14 days after the onset. Patients seen more than 14 days after the attack have not been included.

The 456 episodes occurred in 383 patients, each examined by the author. Fifty-seven were seen in two attacks and seven in three or more attacks. Thirty-six of the patients had had one episode and 12 had had two episodes studied by the author before the start of the 10-year period, and these have
been included in the series. The ages of the patients ranged from 24 to 84 years with a mean of 62 years. The male/female ratio was 1:3:1.

In one-third of the episodes the pain started while the patient was in bed, in two-thirds during the ordinary duties of the day, and in only four during exceptional exertion. In 311 episodes there was a single attack of pain, in 72 two attacks, and in 64 three or more attacks of pain. In nine episodes the number of attacks was uncertain.

**Previous History**

In 90 episodes (20%) there was a history of previous myocardial infarction, in 89 (20%) a history of chronic angina of effort, and in 17 (4%) a history of angina starting during the preceding month. In 68 episodes (15%) the patient was known to have been hypertensive, in 19 (4%) there was a history of paroxysmal or persistent arrhythmia, and in 13 (3%) a history of valvular heart disease.

**Method of study**

In each case, a history was taken with particular reference to the site, distribution, character, and duration of chest pain (or discomfort). The site of the pain was coded on the basis of a numbered plot of the torso as previously described. Inquiry was made about previous attacks of chest pain or discomfort either at rest or on effort, but the patient was not questioned routinely regarding a family history of heart disease to avoid increasing his anxiety.

The cardiovascular system, lungs, and abdomen were examined routinely, and other systems as indicated. The diastolic blood pressure was always recorded at the fourth phase (zone or muffling) and frequently also at the fifth phase (cessation of sound). All electrocardiograms were recorded on a Cambridge Transrite Mark IV machine and analysed on the basis of the Minnesota code. Abnormal Q waves, T wave inversion, and displacement of the ST segment by 0·5 mm or more were classed as major abnormalities. Definite ST displacement less than 0·5 mm and an upright T less than one-tenth the height of the preceding R were classed as minor abnormalities. Left ventricular hypertrophy and bundle-branch block were classed as miscellaneous abnormalities.

A working diagnosis was made at the time of consultation with the help of any previous electrocardiogram available, and a decision was reached with regard to management—either (a) immediate admission to hospital, (b) continued observation at home with a further electrocardiogram and other investigations, or (c) immediate reassurance.

A previous electrocardiogram was available in 234 episodes and a later electrocardiogram was recorded in 301 episodes. Either an earlier or a later electrocardiogram was available in 405 episodes (89%).

Enzyme studies were performed in 148 episodes. They were not done as a routine, because the results could rarely be obtained in less than 12 hours and so could be of no help in immediate diagnosis. Nevertheless, enzyme estimations were valuable in helping to establish the final diagnosis. At the start of the study, both aspartate and alanine aminotransferase (AAT and GPT) estimations were freely available; later GPT was discontinued as a routine estimation.

In 436 episodes (96%) the patient was followed for at least a week. In 205 episodes (45%) the patient was followed for at least a year and in 99 (22%) for five years or more. In 15 episodes (3%) the patient was dead within a week, in 25 (5%) within a month, and in 99 (22%) by the end of the follow-up period.

A final diagnosis of acute myocardial infarction was based on a history of chest pain consistent with ischaemic heart disease as established in a previous review of the earliest symptoms of coronary heart disease, together with either an evolving electrocardiogram consistent with infarction (153 episodes), or a rise of AAT to at least twice the upper limit of normal 24 to 48 hours after the onset (29 episodes), or a single electrocardiogram consistent with infarction followed by death from arrhythmia within 24 hours (two episodes). In 19 of the 184 episodes of acute infarction the patient was dead within four weeks.

A diagnosis of spontaneous angina was made on the basis of 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 electrocardiographic evidence of ischaemic heart disease but without the characteristic evolution of acute infarction and without a significant rise in AAT level at the time of the episode. In five of the 69 episodes of spontaneous angina the patient was dead from ischaemic heart disease within four weeks.

A diagnosis of non-coronary disease was made if the pain was inconsistent with ischaemic heart disease or there was evidence of an alternative explanation for it—provided the electrocardiogram was normal (or if abnormal with an abnormality that could be explained on other grounds, such as previous myocardial infarction, left ventricular hypertrophy, or acute pericarditis) and provided there was no significant rise in AAT.

A diagnosis of doubtful coronary attack was made if the pain was consistent with ischaemic heart disease but there was no electrocardiographic or other evidence to support it and no alternative explanation for it.
Diagnosis of slight coronary attacks

Results

DIAGNOSIS
The final diagnosis of the 456 episodes, taking into account all the evidence, including later electrocardiograms and serum enzyme levels, was as follows: acute myocardial infarction, 184 (40%); spontaneous angina (acute coronary insufficiency), 69 (15%); doubtful coronary attack, 54 (12%); non-cardiac chest pain, 149 (33%).

There were several instances in which it was impossible to categorise the episode with certainty. This is particularly true of the distinction between acute infarction and spontaneous angina—a differentiation which may in any case be artificial. Some of the episodes which were classified as doubtful coronary attack were almost certainly attacks of spontaneous angina. For example, one patient had two identical attacks of praeordial pain. In the first, the electrocardiogram was normal, and the nature of the attack therefore remained doubtful; but in the second it showed characteristic ischaemic abnormalities.

The proportion of episodes finally diagnosed as infarction increased with age, from seven out of 38 (18%) of those under 40 to 59 out of 127 (46%) of those aged 70 or over. There was no appreciable difference in the incidence of infarction between the sexes.

The proportion of episodes ultimately diagnosed as infarction was highest in patients seen within 24 hours of the onset (62 out of 128, that is 48%) and lowest in those seen between seven and 14 days after the onset (31 out of 104, that is 30%). The proportion in which no diagnosis could be made was also highest in those seen within 24 hours, 20 out of 128 (16%) compared with 34 out of 328 (10%) in the case of those seen later. Non-ischaemic heart disease, with or without ischaemic heart disease, was diagnosed in 115 of the 456 episodes; hypertensive heart disease in 70 (15%), valvular disease in 13 (3%), an important arrhythmia (atrial fibrillation, sinusual disease, complete heart block, paroxysmal supraventricular tachycardia) in 24 (5%), and pericarditis in eight (2%). Of the 184 patients with acute infarction, 31 (17%) had associated hypertensive disease. Non-coronary chest pain occurred in 149 of the 456 episodes (33%). The commonest alternative diagnoses were skeletal pain in 31, pleuro-pulmonary pain in 13, biliary pain in 10, and pericarditis in eight episodes. In 57 of the episodes, no diagnosis could be made, though in several the pain was believed to be oesophageal in origin.

CORRELATION BETWEEN SYMPTOMS AND FINAL DIAGNOSIS

Site of pain
In 107 episodes (23%) the patient complained of pain in the sternal region or like a band round the chest with classical radiation either to both arms, or to the throat, jaw, across the shoulders, or between the scapulae. In 221 episodes (48%) the pain was sternal or band-like without classical radiation. In 64 episodes, the pain was in the left chest with or without radiation to other areas, and in 49 episodes it was in some other site. In 15 episodes, the patient described pain in multiple sites.

A final diagnosis of myocardial infarction or spontaneous angina correlated most strongly with those episodes in which the pain was sternal or band-like and had a classical radiation (Table 1). Nevertheless, in 22 of 107 such episodes (21%) the ultimate diagnosis was non-coronary pain and in a further tenth the diagnosis remained uncertain.

Table 1 Characteristics of chest pain

<table>
<thead>
<tr>
<th>No previous history of infarction or angina</th>
<th>Previous history of infarction or angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Episodes Infarction Spon- Non- Doubtful</td>
<td>Episodes Infarction Spon- Non- Doubtful</td>
</tr>
<tr>
<td>Sternal or band-like with classical radiation</td>
<td>59 22 8 18 11</td>
</tr>
<tr>
<td>Sternal or band-like without classical radiation</td>
<td>129 46 9 53 21</td>
</tr>
<tr>
<td>Other site</td>
<td>78 27 1 38 12</td>
</tr>
<tr>
<td>Multiple sites</td>
<td>12 1 0 10 1</td>
</tr>
<tr>
<td>Affected by breathing, twisting, or bending</td>
<td>22 0 0 19 3</td>
</tr>
<tr>
<td>Relation to site of previous infarction/angina: Same or below</td>
<td>140 79 46 8 7</td>
</tr>
<tr>
<td>Elsewhere</td>
<td>24 2 0 22 0</td>
</tr>
<tr>
<td>All cases</td>
<td>278 96 18 119 45</td>
</tr>
</tbody>
</table>
(Two of these patients were shown to have normal coronary arteriograms. One had evidence of an acute Coxsackie infection and in the other no firm diagnosis was made.) The lowest incidence of infarction or spontaneous angina was found in those episodes in which the pain was in multiple sites.

Relation to site of previous cardiac pain
In 178 episodes, the patient gave a history of previous infarction or angina and in 164 of these he was able to recall the site of the pain. (A history of recent onset angina was frequently overlooked by the general practitioner.) In 127 of these 164 episodes, the patient indicated that the pain in the present attack had the same epicentre as before (though in 23 the present pain was more widespread); in 13 it was immediately below the site of the previous ischaemic pain, and in 24 it was in another site. In 125 (89%) of the 140 episodes in which the pain had the same epicentre as before or was immediately below that of the preceding angina, the final diagnosis was myocardial infarction or spontaneous angina (Table 1). In only two of the 24 episodes in which the pain was in a different site was the final diagnosis myocardial infarction or spontaneous angina; in one of these, myocardial infarction was complicated by pulmonary infarction and in the other by cervical spondylosis.

Duration of pain
In 37 episodes, the patient said that the pain lasted less than 15 minutes, in 79 between 15 and 60 minutes, in 101 one to three hours, in 109 three to 12 hours, in 113 over 12 hours, and in 17 episodes the duration was uncertain. Of the 37 episodes lasting less than 15 minutes, seven were diagnosed as myocardial infarction and four as spontaneous angina. In the case of attacks lasting over 15 minutes, the proportion diagnosed as myocardial infarction and spontaneous angina was similar whether the duration was half an hour, eight hours, or over 24 hours.

Severity of pain
In 187 (41%) episodes, the patient described the pain as severe, and in 34 (7%) as slight. The remainder were classified as moderate. Of the 187 episodes in which the patient described the pain as severe, 90 (48%) were diagnosed as acute infarction, and 21 (11%) as spontaneous angina. Of the 34 episodes in which the patient described the pain as slight, 11 were diagnosed as myocardial infarction and four as spontaneous angina. It is perhaps worth mentioning that a district nurse carried on her duties for a whole day with the pain of acute infarction, and a man of 41 actually played badminton in spite of the pain of infarction.

Character of pain
The great majority of patients described their pain as being unaffected by breathing, twisting, or bending. But in 21 episodes aggravation with breathing was noted and in six the pain was affected by twisting or bending. In only one of these 27 episodes was a diagnosis of myocardial infarction established and in this case it was complicated by pulmonary infarction (Table 1).

Some other observations regarding the character of the pain may be mentioned here. In two cases of pericarditis the pain was not affected by respiration, and in one it was relieved by lying rather than sitting. Conversely, at least one patient with acute infarction found relief on sitting. In two patients with pulmonary infarction, the pain was not pleuritic at first, though it became so later. Three patients with severe chest pain sought relief by walking around the room; none of these proved to have acute infarction.

<table>
<thead>
<tr>
<th>Table 2: Associated symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No previous history of infarction or angina</strong></td>
</tr>
<tr>
<td><strong>Episodes</strong></td>
</tr>
<tr>
<td>Nausea + vomiting</td>
</tr>
<tr>
<td>Dyspnoea</td>
</tr>
<tr>
<td>Palpitation</td>
</tr>
<tr>
<td>Faintness/syncope</td>
</tr>
<tr>
<td>Sweating</td>
</tr>
<tr>
<td>Headache</td>
</tr>
<tr>
<td>Cough</td>
</tr>
<tr>
<td>Miscellaneous</td>
</tr>
<tr>
<td>All cases</td>
</tr>
</tbody>
</table>
Diagnosis of slight coronary attacks

Table 3  Clinical examination in patients seen within 72 hours of onset

<table>
<thead>
<tr>
<th></th>
<th>No previous history of infarction or angina</th>
<th>Previous history of infarction or angina</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Episodes</td>
<td>Infarction</td>
</tr>
<tr>
<td>Breathlessness</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Shock</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Atriopeptic beats</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>14</td>
<td>8</td>
</tr>
<tr>
<td>Tachycardia (&lt;60/min)</td>
<td>15</td>
<td>6</td>
</tr>
<tr>
<td>All cases</td>
<td>140</td>
<td>57</td>
</tr>
</tbody>
</table>

Associated symptoms
In 185 episodes (41%) the patient described some symptom in addition to his chest pain. The commonest was nausea (71 episodes) and the next commonest dyspnœa (29 episodes). Others included palpitation (19), faintness or syncope (18), sweating (14), headache (5), and cough (4). The highest incidences of acute infarction were seen in association with sweating (9 out of 14) and nausea (36 out of 71) and the lowest with cough (one out of four) and headache (one out of five) (Table 2).

Correlation between signs and final diagnosis

General appearance
In 235 episodes, the patient was seen within 72 hours of the onset of chest pain. In 199 of these he did not appear in any way ill or distressed; yet in 93 the final diagnosis was acute infarction. In nine episodes, the patient appeared breathless and in a further nine he appeared cold and sweating. Of these 18 episodes, 11 were diagnosed as myocardial infarction and five as spontaneous angina (Table 3).

Pulse
In 166 of the 235 episodes in which the patient was seen within 72 hours of the onset of chest pain, the pulse was regular throughout the examination with a rate within the range 60 to 99 per minute. In 17 episodes extrasystoles were noted, in 20 there was atrial fibrillation, in 20 bradycardia (two of these patients were on a beta-blocking drug and two had received an opiate), and in 21 tachycardia (including one patient who had received atropine). In nine episodes, a combination of two abnormalities was noted, for example atrial fibrillation and tachycardia. The only group in which the proportion of cases of acute infarction or spontaneous angina was much different from that of the series as a whole was in the case of tachycardia; 11 of the 21 episodes being diagnosed as myocardial infarction and six as spontaneous angina.

Blood pressure
In 14 episodes a systolic blood pressure below 110 mmHg was recorded. In nine of these, the final diagnosis was myocardial infarction. At all other levels of systolic and diastolic blood pressure, the proportion with acute infarction and spontaneous angina was similar to that of the series as a whole.

In view of the belief that a fall in blood pressure is an indication of acute infarction, particular attention was paid to any change in systolic pressure by comparison with a previous reading when the patient was in his usual health. A valid comparison was possible in 129 episodes. In 25 episodes there was a fall of 20 mmHg or more, in 13 a fall of 40 mmHg or more, in 23 a rise of 20 mmHg or more, and in 12 a rise of 40 mmHg or more. No group showed an incidence of acute infarction

Table 4  Change in blood pressure

<table>
<thead>
<tr>
<th>Change of blood pressure (mmHg)</th>
<th>Episodes</th>
<th>Final diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Infarction</td>
</tr>
<tr>
<td>Fall of 20</td>
<td>25</td>
<td>9</td>
</tr>
<tr>
<td>40</td>
<td>13</td>
<td>6</td>
</tr>
<tr>
<td>Rise of 20</td>
<td>23</td>
<td>9</td>
</tr>
<tr>
<td>40</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td>No rise or fall of 20</td>
<td>56</td>
<td>22</td>
</tr>
<tr>
<td>All cases</td>
<td>129</td>
<td>47</td>
</tr>
</tbody>
</table>

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which differed significantly from that of the series as a whole apart from that showing a rise of 40 mmHg or more, only one of whom proved to have acute infarction (Table 4). If consideration is limited to episodes in which the patient was seen within 24 hours of the onset, the findings were similar.

OTHER OBSERVATIONS

Basal crepitations
Nine patients showed this sign. Five were ultimately diagnosed as acute infarction and three as spontaneous angina.

Tenderness of chest wall
Five patients showed this sign of whom one was diagnosed as acute infarction.

Raised jugular venous pressure
Four patients had this sign and all were diagnosed as acute infarction.

Angina induced by excitement
This was recorded in three patients, all of whom were diagnosed as spontaneous angina.

Pericardial rub
This was noted in three patients each of whom was diagnosed as acute non-ischaemic pericarditis.

Murphy's sign
Three patients had inspiratory right subcostal tenderness, one of whom was found to have acute infarction.

Epigastric tenderness
This was noted in two patients, both of whom were ultimately diagnosed as acute infarction.

Extracardiac cause of chest pain
This was found in 96 (21%) episodes. In 32 of these, the final diagnosis was acute infarction. Out of 184 episodes ultimately diagnosed as acute infarction, an associated extracardiac cause of pain was present in 17 per cent, most commonly pleuritic, peptic, skeletal, biliary, or oesophageal.

Many patients offered a plausible explanation for their pain, such as unwonted exercise, an exceptional meal, a heavy cold, or a previous diagnosis of hiatus hernia. These were sometimes the cause of the chest pain; but in many instances the patient was found to have sustained acute infarction.

CORRELATION BETWEEN ELECTROCARDIOGRAM AND FINAL DIAGNOSIS

One hundred and ninety-five (43%) of the 456 electrocardiograms recorded at the time of the initial examination showed a major abnormality consistent with coronary disease; a further 118 (26%) showed minor or borderline ischaemic abnormalities, 53 (12%) showed miscellaneous abnormalities, and 90 (one-fifth) were strictly normal (Table 5).

The diagnostic significance of electrocardiographic abnormalities was naturally greater in the absence of a history of previous infarction or angina. Of the 278 episodes in patients with no previous history of infarction or angina, 78 showed major electrocardiographic abnormalities of coronary type. Of these, 62 (79%) were diagnosed as acute infarction and a further two as spontaneous angina. (Major electrocardiographic abnormalities not resulting from acute infarction were mostly the result of left ventricular hypertrophy or acute pericarditis.) Of the 178 episodes in patients with a previous history of infarction or angina, 117 showed major

<table>
<thead>
<tr>
<th>Table 5 Electrocardiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>
|                          | Episodes Infarction Spon- |aneous angina Non- coronary Doubtful | Episodes Infarction Spon-
|                          | 96  | 96  | 18  | 119 | 45  | 117 | 70  | 31  | 13  | 3  |
| Major abnormality        | 78  | 62  | 56  | 9   | 11  | 53  | 43  | 1    | 0   |
| Minor abnormality        | 60  | 19  | 7   | 23  | 11  | 37  | 74  | 2    | 0   |
| Borderline abnormality   | 30  | 7   | 3   | 14  | 6   | 22  | 85  | 1    | 0   |
| Miscellaneous            | 41  | 7   | 3   | 18  | 13  | 11  | 78  | 4    | 0   |
| Normal                   | 69  | 1   | 2   | 23  | 11  | 21  | 76  | 2    | 0   |
| All cases                | 278 | 96  | 18  | 119 | 45  | 178 | 88  | 51  | 30  | 9  |

ST elevation
All cases
Cases seen within 72 h of onset

32 27 2 2 1
19 16 0 2 1
Diagnosis of slight coronary attacks

Electrocardiographic abnormalities. Of these, 70 (60%) were diagnosed as acute infarction and a further 31 (26%) as spontaneous angina.

The interpretation of minor and borderline electrocardiographic abnormalities presented considerable difficulty. In 60 (22%) of the 278 episodes in those with no previous history of infarction or angina, the initial electrocardiographic abnormalities were too slight to be classified under the Minnesota code and in a further 30 (11%) there were borderline abnormalities. The proportion ultimately diagnosed as acute infarction or spontaneous angina was 43 per cent in the case of those with minor abnormalities and 33 per cent for those with borderline abnormalities. Three patients who presented with electrocardiograms showing only minor ST/T abnormalities were dead within a month.

ST elevation of 1 mm or more was noted in 43 of the 456 episodes, and minor or borderline elevation in a further 35. Sixty-three of the 78 cases with ST elevation of any degree proved to have acute infarction (Table 5), five were diagnosed as acute pericarditis, four as ventricular aneurysm, and four as a physiological pattern. Confining consideration to episodes in which the patient was seen within three days of the onset, the incidence of ST elevation was higher but the significance was similar.

Intraventricular conduction defect: right or left bundle-branch block was found in 15 episodes; 10 were diagnosed as acute infarction and three as spontaneous angina. Left axis deviation (left anterior hemiblock) was found as the sole abnormality in 35 episodes: six of these proved to be acute infarction and eight spontaneous angina.

A normal initial electrocardiogram was recorded in 90 episodes. Three of these were ultimately diagnosed as acute infarction and 10 as spontaneous angina.

Comparison with previous electrocardiogram
An evolving electrocardiogram consistent with myocardial infarction was accepted from the start as one of the most important pieces of evidence in making a diagnosis of acute infarction. It is therefore no surprise that deterioration of the electrocardiogram showed a strong positive correlation with a diagnosis of infarction whereas lack of deterioration showed an equally strong negative correlation with that diagnosis. The correlation was not, however, absolute. There were 234 episodes in which a previous electrocardiogram was available for comparison. Definite deterioration was noted in 102. In 92 of these, the final diagnosis was acute infarction and in three spontaneous angina. Absence of deterioration (or improvement) was noted in 100 and of these only three were diagnosed as acute infarction and 21 as spontaneous angina. In 32 episodes there was doubtful deterioration. There was one case ultimately diagnosed as acute infarction in which the electrocardiogram would have been accepted without question as being within normal limits had not a previous "more normal" tracing been available for comparison.

Accuracy of bed-side diagnosis
Of the 184 episodes ultimately diagnosed as acute infarction, 174 were so diagnosed at the bedside on the basis of the history, examination, and available electrocardiograms. One was misdiagnosed as a non-coronary episode, three as spontaneous angina, and in six the bedside diagnosis was uncertain.

In six episodes, an incorrect diagnosis of myocardial infarction was made at the bedside. Two proved to be non-coronary episodes, two were attacks of spontaneous angina, and two remained doubtful.

Often, the diagnosis had to be made by piecing together a number of clues. For example, one patient was cold and clammy, suggesting a large infarct; but the electrocardiogram showed only borderline abnormalities. Another complained of dyspnoea and nausea in association with central chest pain lasting three hours. If the symptoms had been the result of myocardial infarction, it would

<table>
<thead>
<tr>
<th>AAT</th>
<th>Episodes</th>
<th>Infarction</th>
<th>Spontaneous angina</th>
<th>Non-coronary</th>
<th>Doubtful</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>86</td>
<td>34</td>
<td>19</td>
<td>26</td>
<td>7</td>
</tr>
<tr>
<td>Raised, but less than twice normal; GPT not done</td>
<td>14</td>
<td>13</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Raised, but less than twice normal; GPT normal</td>
<td>11</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Raised, but less than twice normal; GPT raised</td>
<td>5</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Twice normal or more; GPT not done</td>
<td>25</td>
<td>25</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Twice normal or more; GPT normal</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Twice normal or more; GPT raised</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>
have had to be a large one. In fact the electrocardiogram was normal.

ENZYMES
At least one estimation of AAT was made in 148 episodes, and in one-third of these GPT was also estimated. In 62 episodes, raised AAT was reported and in 53 of these the final diagnosis was myocardial infarction (Table 6). Of 13 episodes in which AAT was raised and GPT normal, 12 were diagnosed as myocardial infarction; whereas of 10 episodes in which both AAT and GPT were raised only three were diagnosed as myocardial infarction. In 86 episodes a normal AAT was reported; 34 of these were diagnosed as myocardial infarction and 19 as spontaneous angina.

Discussion
An episode of chest pain raising some degree of suspicion of a heart attack is a common experience. This series clearly represents a selected group. Most individuals who experience chest pain do not report to a doctor at all. Of those who do, the mildest episodes are dealt with by the general practitioner alone, the most severe are sent to hospital, and only the equivocal ones are referred for cardiological opinion. Nevertheless, these equivocal cases form an important and difficult group in which guidelines are badly needed, particularly by the doctor who first sees the patient. This study was designed to meet this need.

Unfortunately, this is a field in which certainty is unattainable. This is reflected in the large proportion of unsolved episodes in the present series. Even where a label has been more or less confidently applied, the diagnosis is by no means beyond dispute. Nevertheless, I believe that the diagnosis is as accurate as can be achieved with currently available techniques under the circumstances of a study in the community. It is hoped that the size of the series will prevent serious distortion from mistakes in diagnosis.

DIAGNOSIS
What conclusions can be drawn from this study? First, with regard to the history. In patients with no previous history of infarction or angina, the strongest correlations were negative ones (Fig. 1). Pain affected by breathing, twisting, or bending was never, and pain at multiple sites was rarely, associated with a final diagnosis of acute infarction or spontaneous angina. Surprisingly, perhaps, classical sternal pain radiating to both arms and the jaw did not show any outstanding correlation with infarction or spontaneous angina. Of symptoms associated with chest pain, the only two that were in any degree remarkable were nausea (with or without vomiting) and sweating, both of which showed a positive correlation with acute infarction.

In patients with a previous history of infarction or angina there was a valuable additional point (Fig. 2). The pain of acute infarction or spontaneous angina almost invariably had the same epicentre as before or was immediately below it. Pain in another part of the chest was rarely cardiac in origin. The commonest mistakes made by the referring doctor were overlooking recent onset cardiac angina and failing to elicit the fact that the pain was affected by breathing.

With regard to examination, the most important point to emphasise is that in most of the cases of infarction in this series there were no abnormal physical signs. This was so even in patients seen within 24 hours of the onset. Shock and tachycardia (heart rate 100 per minute or over) were strongly correlated with infarction, and breathlessness was positively associated with a diagnosis of either infarction or spontaneous angina (Fig. 1). Bradycardia (heart rate below 60 per minute) showed a positive correlation with infarction in patients with a previous history of infarction or angina (Fig. 2) but the numbers were small, and this association was not found in cases without a previous history. Changes in blood pressure, abnormalities of the cardiac impulse and heart sounds, and the presence of a murmur were all unhelpful. The presence of a pericardial rub (in the absence of evidence of major infarction) indicated non-ischaemic pericarditis. Angina induced by history-taking or examination was diagnostic of spontaneous angina.

The electrocardiogram was the most valuable aid to diagnosis, though no single piece of electrocardiographic evidence had a conclusive diagnostic value. ST elevation, however slight, showed a strongly positive correlation with acute infarction (Fig. 1 and 2). Nevertheless, almost one-fifth of the episodes in which ST elevation was recorded did not prove to have acute infarction; the abnormality being the result of ventricular aneurysm, acute pericarditis, or a physiological pattern. In patients with no previous history of infarction or angina, the finding of a major abnormality of coronary type was strongly correlated with acute infarction. An ST/T abnormality too slight to be codifiable under the Minnesota code was associated with evidence of acute infarction or spontaneous angina in over one-third of the instances in which it was found. A strictly normal electrocardiogram largely excluded infarction, though not the almost equally serious diagnosis of spontaneous angina.

In patients with a previous electrocardiogram, evidence of deterioration was strongly, though not
Fig. 1 Predictive value of various symptoms, signs, and electrographic features for diagnosis of myocardial infarction and spontaneous angina in patients presenting with chest pain but with no previous history of infarction or angina.

The vertical lines at 35 per cent and 41 per cent indicate the percentage of episodes with a final diagnosis of myocardial infarction and spontaneous angina in all patients with no previous history of infarction or angina. The section headed signs refers to patients seen within 72 hours of the onset. The symbols (● ○) on the right of the figure indicate the features with the strongest positive and negative associations with myocardial infarction (Mi) and spontaneous angina (SA).
Fig. 2  Predictive value of various symptoms, signs, and electrocardiographic features for diagnosis of myocardial infarction and spontaneous angina in patients presenting with chest pain who had a history of infarction or angina. The vertical lines at 49 per cent and 78 per cent indicate the percentage of episodes with a final diagnosis of myocardial infarction and spontaneous angina in all patients with a previous history of infarction or angina. For the significance of abbreviations and symbols see notes on Fig. 1.
Diagnosis of slight coronary attacks

Table 7  Guide to immediate diagnosis of slight and subacute coronary attacks in patients with a previous history of infarction or angina

<table>
<thead>
<tr>
<th></th>
<th>Unlikely</th>
<th>Equivocal</th>
<th>Likely</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>Affected by breathing, twisting, or bending</td>
<td>Nausea, dyspnoea, palpitation</td>
<td>Sweating</td>
</tr>
<tr>
<td></td>
<td>Different epicentre from that of previous infarction or angina</td>
<td>Fall in BP</td>
<td>Dyspnoea, shock, bradycardia, tachycardia</td>
</tr>
<tr>
<td><strong>Other Signs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Absolute normality</td>
<td>Conduction defect</td>
<td>ST elevation (however slight)</td>
</tr>
<tr>
<td>Electrocardiogram</td>
<td>No deterioration by comparison</td>
<td>Minor ST/T depression</td>
<td></td>
</tr>
<tr>
<td></td>
<td>with previous electrocardiogram</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

...continued...

...continued...

...continued...

Absolutely, correlated with acute infarction—as assumed at the start of the study. Lack of deterioration showed a strong negative correlation with infarction. There were, however, several instances in which the electrocardiogram showed no deterioration and yet the total evidence pointed conclusively to acute infarction, and many more in which the final diagnosis was spontaneous angina.

Serum enzyme estimation was no help in immediate diagnosis because of the delay in obtaining the result. In some episodes with convincing electrocardiographic evidence of minor infarction no definite elevation was recorded.

MANAGEMENT

How should a patient with one or more spontaneous attacks of chest pain be managed? If he is known to have had angina or a confirmed myocardial infarct in the past (Table 7), the key question is: is the present pain in the same situation or does it have the same focus as previous cardiac pain? If it does not have the same epicentre nor is it immediately below the site of previous angina, it is most unlikely to be cardiac in origin. If the patient denies previous infarction or angina, he should be asked specifically whether he had had pain in the chest induced by effort and rapidly relieved by rest during the immediately preceding days. Even a very short history of classical angina culminating in similar pain at rest is diagnostic of myocardial infarction or spontaneous angina.

If there is no previous history of infarction or angina (Table 8), the patient should be asked if the pain is affected by taking a deep breath, or by bending or twisting. If the answer is in the affirmative, it is not cardiac pain. It may be pericardial, but it is more likely to be pleuritic.

If the patient indicates that the pain has moved from one site to another, a diagnosis of acute infarction is unlikely. An association with nausea or sweating is suggestive of acute infarction—though not diagnostic. It is important to recognise that an alternative cause for chest pain, whether skeletal or alimentary, does not exclude coincidental acute infarction.

On examination, breathlessness (in the absence of a history of chest disease) and evidence of shock or tachycardia are suggestive of acute infarction or...
spontaneous angina, but a complete absence of abnormal physical signs by no means excludes the diagnosis.

If the character of the chest pain is not inconsistent with a diagnosis of myocardial infarction, an electrocardiogram should be arranged. The degree of urgency is inversely related to the interval since the onset—the shorter the history the greater the urgency. If the electrocardiogram shows ST elevation of even the slightest degree in any lead except VR, V1, or V2, the likelihood of acute infarction is great. In a patient with no previous history of infarction or angina, the presence of a major abnormality of coronary type is almost equally suggestive. If a previous electrocardiogram is available, a finding of deterioration strongly suggests infarction and lack of deterioration makes it improbable. An absolutely normal electrocardiogram is strong evidence against a diagnosis of acute infarction, though not of spontaneous angina; but the finding of minor or even borderline changes is no ground for reassurance.

If the diagnosis remains doubtful after electrocardiography, the course of action depends on the length of interval since the last attack of suspicious chest pain and the availability of a coronary care unit. Any possibility of myocardial infarction must be treated seriously, and a diagnosis of spontaneous angina must be regarded almost as seriously as one of infarction. If the pain is still present or has subsided within the past 24 hours, and if a coronary care unit is within easy reach, it is probably safest to admit the patient for observation. If an interval of over 24 hours has elapsed since the pain subsided or if a coronary care unit is not available, the patient should be kept under "active observation" at home. This involves keeping him at rest, administering a beta-blocking drug, a nitrate, or diazepam as indicated; checking the serum enzyme level and repeating the electrocardiogram after an interval of 12 to 24 hours.

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References


Requests for reprints to Dr David Short, Cardiac Department, Aberdeen Royal Infirmary, Foresterhill, Aberdeen AB9 2ZB.
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D Short

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