CARDIAC INFARCTION WITH PAIN CONFINED TO EFFORT

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Occlusion of a coronary artery has long been regarded as a possible basis for angina of effort, but it was not realized that it might be the usual cause until the pathological work of Blumgart, et al. (1940). In angina of effort (without cardiac hypertrophy, aortic syphilis, or anæmia) they demonstrated by post-mortem injection studies the invariable presence of occlusion of at least two main branches of the coronary arteries. In some of these cases myocardial infarction had followed the occlusion without the clinical counterpart of prolonged rest pain: this conforms with clinical experience for it is not uncommon to find electrocardiographic evidence of myocardial infarction when cardiac pain is confined to effort. So far as we know, the first reported case of this type is that of Bourne (1939). The object of this paper is to determine how often this is so and to compare our clinical observations with the electrocardiographic evidence.

METHODS

Selection of cases. Between January, 1946 and April, 1949, 291 patients were seen who complained of pain considered to be of cardiac origin, and in 97 cases the pain was apparently confined to effort. At the time of this investigation, 82 of these patients were available, 8 having died without the opportunity for necropsy and 7 being untraced. These 82 patients were personally re-interviewed and 20 were rejected on the grounds given below:

<table>
<thead>
<tr>
<th>Description</th>
<th>Cases</th>
</tr>
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<tbody>
<tr>
<td>Pain on effort but lasting on occasions more than 15 minutes</td>
<td>9</td>
</tr>
<tr>
<td>Pain of short duration but sometimes occurring at rest</td>
<td>7</td>
</tr>
<tr>
<td>Pain so atypical as to be of uncertain origin</td>
<td>3</td>
</tr>
<tr>
<td>Positive Wassermann reaction</td>
<td>1</td>
</tr>
</tbody>
</table>

The 62 selected patients complied with the following criteria.

1. They complained of pain described as constricting, choking, pressing, aching, or burning, either retrosternal or radiating from some part of the chest to one or both arms or to the neck or jaw.
2. The pain was constantly related to effort, had never occurred at rest, and was invariably rapidly relieved by rest, never persisting for more than 15 minutes.
3. There was no clinical or radiological evidence of a valvular lesion. (With the exception of one case in which the combination of a basal systolic murmur, slight left ventricular enlargement and a normal blood pressure made it impossible to exclude aortic valve disease).
4. The blood count was normal and the Wassermann reaction negative.
5. (2) and (4) were introduced to exclude certain cases in which there might be some other cause for relative myocardial ischaemia.

In selecting these cases re-interviewing was found to be essential, for an initial prolonged attack of cardiac pain was not always mentioned at the first interview, as illustrated by the following cases.

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A man of 45 first attended the clinic in October, 1947, complaining of a mid-sternal crushing pain occurring when walking rapidly and immediately relieved by rest, which had been present for six weeks. He denied having any prolonged attack of pain. On re-interview he admitted that the initial attack, which had occurred when hurrying with a heavy bag, had lasted half an hour and had been so severe that he voluntarily spent three days in bed. Effort pain had been present since resuming activity. The electrocardiogram showed changes diagnostic of posterior myocardial infarction.

A man of 50 attended the clinic in August, 1948, complaining solely of retrosternal burning pain on effort relieved by rest. On re-interview he recollected being awakened one night in June, 1948, by severe epigastric pain lasting some hours and associated with vomiting. The following day he first noticed the effort pain. He regarded the initial attack as gastric in origin and therefore of no importance. The electrocardiogram showed changes diagnostic of posterior myocardial infarction.

**Electrocardiographic Technique.** A routine cardiogram had already been taken on the occasion of the patient's initial visit to the clinic. Unless this showed unequivocal evidence of infarction, we made what we considered to be an adequate electrocardiographic investigation. In all cases this included the standard limb leads, the unipolar limb leads, 6 precordial leads in the positions 1 to 6, and 6 high precordial leads taken one* interspace above the corresponding normal position.

In recording the precordial leads the exploring electrode was paired with the central terminal of Wilson (1943) using 5000 ohm resistances. The unipolar limb leads were recorded by Goldberger's (1942) technique but 5000 ohm resistances were inserted in the limb connections to the indifferent electrode as recommended by Wilson (1943) and Bryant, Johnston, and Wilson (1949).

**Criteria for Diagnosis of Infarction.** Principles governing the electrocardiographic diagnosis of infarction have been laid down by Wilson and his associates (1933, 1934, 1935). The application of these principles to the patterns occurring in the precordial leads in association with infarction of the anterior free wall of the left ventricle and the septum has been clearly described (Wilson, 1944), and further electrocardiographic and pathological correlation has been carried out by Myers et al. (1948a, 1948b, 1949a, 1949b). We have followed their criteria, which may be summarized as the combination of two of the following three types of change in leads facing ventricular muscle.

(1) Characteristic QRS changes.
   (a) Q waves of more than 0.03 seconds duration from onset to lowest point.
   (b) Absent R waves from complexes normally of RS type.

(2) Elevation of S–T segment by more than 0.4 mV.

(3) T wave changes of a form and distribution not explicable by ventricular enlargement.

The changes in form of the ventricular complex to be accepted as diagnostic of infarction of the posterior free wall of the left ventricle are, however, much less certain, for the form of the complex in the foot lead is profoundly affected by changes in the electrical position of the heart. Myers and Oran (1945) studying the form of lead VF in 25 cases of posterior infarction proven by autopsy or oesophageal leads, concluded that, although a Q of more than 25 per cent of the amplitude of the succeeding R was suggestive of infarction and absence of a Q wave was strongly against infarction, diagnostic evidence was difficult to obtain. In more recent articles Myers et al. (1949c, 1949d) have studied the form of lead VF in different electrical positions of the heart and have shown that posterior infarction is often not recognizable inVF when the heart is electrically horizontal or semi-horizontal. On the basis of their work, we have regarded the foot lead as diagnostic only when both the following changes were present—(1) A Q wave of at least 0.03 seconds duration from onset to lowest point followed by a small R and deep S or a tall late R: or a QS deflection in an electrically intermediate to vertical heart (left bundle branch block must be excluded for this may give rise to a broad slurred QS or QR complex), and (2) Characteristic changes in the ST–T complex.

Two examples of posterior infarction, from the present series of cases, are shown in Fig. 1. The upper record shows the standard and unipolar limb leads from a man of 51 who had suffered anginal pain on effort for five years. The Q wave in lead VF is of almost 0.04 sec. duration from onset to lowest point and the corresponding T wave is deeply and symmetrically inverted. The

*We now usually take high precordial leads two spaces above the usual positions.
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The electrical position is intermediate. The lower record shows the standard limb, unipolar limb and præcordial leads from a man of 50 who had had angina of effort for almost three years. Here the Q wave in VF is 0.03 sec. in duration and the T wave inversion is less striking than in the upper record. Since the electrical position of the heart is semi-vertical, we regard these changes as diagnostic of posterior infarction but they are the minimum changes that we would accept.

**Classification of Electrocardiograms.** If two of the three types of change in the ventricular complex already enumerated were present we classed the electrocardiogram as diagnostic of infarction. If only one type of change was present we classed it as abnormal, and suggestive but not
diagnostic of infarction. Two typical cardiograms from this group are shown in Fig. 2 and 3. Fig. 2, from a 65-year-old woman who had suffered from classic effort angina for eight years shows symmetrical T wave inversion from the second to the sixth position in both the normal and high præcordial leads. The QRS complexes are within normal limits and there is no S–T displacement. Fig. 3 is from a 54-year-old woman who had suffered from angina of effort for only five weeks. Here the T wave abnormalities are localized to V2, V3, and V4 and the corresponding high leads. The QRS complexes are within normal limits. In neither case do we think the T wave changes are explicable by ventricular enlargement. The remaining records were classified as showing other abnormalities or as being within normal limits.

**RESULTS**

The selected group comprised 53 men and 9 women whose ages ranged from 31 to 74 years. Forty-eight had normal blood pressure and 14 had a persistent diastolic pressure of 105 mm. of Hg, or greater. Six of the group with normal blood pressure showed radiological evidence of enlarge-
FIG. 2.—Example of an electrocardiogram regarded as abnormal and suggestive of infarction. There are widespread T wave changes extending from V2 to V6 which are not entirely explicable by ventricular enlargement.

FIG. 3.—T wave changes confined to V2, V3 and V4 and the corresponding high leads. This record was regarded as abnormal and suggestive of infarction.
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ment of the left ventricle suggesting the possibility of past hypertension. The electrocardiographic evidence is given below.

Electrocardiograms diagnostic of infarction ... ... ... ... 17 cases
Electrocardiograms abnormal and suggestive of infarction ... ... ... ... 14 cases
Electrocardiograms within normal limits ... ... ... ... 23 cases
Electrocardiograms showing other abnormalities ... ... ... ... 8 cases

Twelve infarctions were situated posteriorly and therefore diagnosed from the limb leads. There were five anterior infarcts and in two of them the limb leads were within normal limits.

<table>
<thead>
<tr>
<th>Site of Infarct</th>
<th>No.</th>
<th>Limb leads diagnostic</th>
<th>Limb leads abnormal</th>
<th>Limb leads normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior</td>
<td>12</td>
<td>12</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Anterior</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>2</td>
</tr>
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</table>

Fig. 4 shows two records from one of these two cases, a man of 57. When the first electrocardiogram (A) was taken angina of effort had been present for only six weeks. V2 and V4 show diagnostic changes with absent initial R waves, S–T elevation, and terminal inversion of the T waves. The standard limb leads are within normal limits though TI is of low voltage. Two months later (B) TI has become negative because the T wave changes have spread far enough to the left to modify the left arm lead. Changes confined to the centre of the praecordium will rarely be transmitted to the limb leads.

In 14 cases the electrocardiograms were abnormal and suggestive of infarction. The abnormalities were confined to negative T waves in the praecordial leads not explicable by ventricular enlargement in 11 cases; in the remaining 3 they were confined to the QRS complexes.

![Fig. 4.—Two electrocardiograms from the same case. (A) six weeks after the onset of angina of effort, shows normal limb leads but changes diagnostic of infarction in V2 and V4. (B) three months later, shows that T wave changes have extended to V6 and therefore appear in lead I.](image)

The proportion of definite infarcts was lower in the cases with hypertension or radiological evidence of cardiac enlargement than in those with a normal blood pressure and without radiological cardiac enlargement; on the other hand, the proportion of doubtful infarcts was higher in the group with cardiac enlargement.

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Definite infarcts</th>
<th>Doubtful infarcts</th>
<th>No infarcts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases without cardiac enlargement</td>
<td>42</td>
<td>15</td>
<td>7</td>
<td>20</td>
</tr>
<tr>
<td>Cases with enlarged hearts</td>
<td>20</td>
<td>2</td>
<td>7</td>
<td>11</td>
</tr>
</tbody>
</table>

It may be that when the heart is enlarged, angina of effort occurs more readily in the absence of
infarction, but the number of cases is not sufficient to justify any firm conclusion on this point. The high precordial leads were recorded in 44 cases but we did not derive any additional information from them.

**DISCUSSION**

We believe that our diagnostic criteria were sufficiently strict to ensure that myocardial infarction was not diagnosed unless it had certainly occurred. On the other hand there are reasons why the clinical data should underestimate the incidence of infarction. In the first place, infarction may occur in a site inaccessible to ordinary electrocardiographic examination and this is often so when the posterior free wall of the left ventricle is involved. Myers et al. (1949c, 1949d) found changes diagnostic of posterior infarction in only 11 of 35 cases in which the heart was electrically horizontal, despite extensive old posterior infarction at necropsy. When the heart was electrically intermediate to vertical the proportion of healed infarctions diagnosed cardiographically increased to 42 out of 75. Secondly, diagnostic changes in the cardiogram may disappear in time; changes in the ST–T complex are usually transient and even changes in the QRS complex may not be permanent. Wilson (1943) has reported a case in which R waves, absent shortly after clinical coronary occlusion, returned later and a similar case (not included in the present series) is illustrated in Fig. 5. The patient, a man of 51, had a clinical coronary occlusion on October 29. The electrocardiogram taken on October 30 (Fig. 5A) showed absent R waves in V2 and V3, a Q wave of abnormal duration (0-03 sec.) in V4 and inversion of the T waves in V4 and V5. Three months later (Fig. 5B) the record was within normal limits.

There are likely to be even more cases with coronary occlusion than the number of cardiac infarctions suggests, for Blumgart et al. (1941a and b) have shown that coronary occlusion may not be followed by cardiac infarction and possibly not even by permanent ischemia of sufficient extent to affect the electrocardiogram.

Making allowance for the inadequacy of the electrocardiogram as a means of detecting healed infarction, and for the fact that coronary occlusion may occur without consequent myocardial infarction, it seems to us that our results are not inconsistent with the conclusion of Blumgart et al. (1940) that in the absence of cardiac enlargement, syphilis, or anemia, occlusion of a major coronary vessel is the usual underlying cause of the ischemia associated with angina of effort. If we accept this conclusion, it leads us to regard angina of sudden onset as evidence of coronary occlusion (with or without infarction). If the onset of the angina is recent, then the management of the case will be similar to that usually adopted in recent cardiac infarction, though possibly rest in bed need not be so prolonged. Bourne (1939) reported a case in which the cardiogram taken 4 weeks after the sudden onset of angina of effort showed a negative T wave in lead IV which had reverted to normal 8 weeks later; he attributed the negative TIV to coronary thrombosis and concluded that a patient may present symptoms of angina of effort as a result of coronary thrombosis. With this conclusion we entirely agree. However, since coronary occlusion need not be followed by cardiac infarction, or even by myocardial ischemia sufficient to produce T wave changes on the cardiogram, we regard angina of sudden onset as evidence of coronary occlusion whether the electrocardiogram is normal or not. The wisdom of adopting this attitude in practice is illustrated by the following case.

A man of 45 consulted one of us (A.M.J.) because 2 weeks previously he had suddenly developed severe constricting pain in the chest whenever he walked 20 to 30 yards. This was rapidly relieved by rest or trinitrin and was in every way typical of severe angina of effort. In these circumstances cardiographic evidence of recent cardiac infarction might be expected, but in this case the cardiogram was entirely normal (Fig. 6). Nevertheless, it was assumed that a coronary occlusion had occurred two weeks previously and rest in bed was advised. Ten days later the patient insisted on getting up, however, and went into the garden where he collapsed and died within a few minutes.

It seems possible that had this man rested for a sufficient period to permit the establishment of a collateral circulation, death would not have occurred so soon.
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Fig. 5.—Two electrocardiograms from the same case. (A) Diagnostic changes of infarction in V3 to V6 on the day after the infarction. (B) Three months later, is entirely normal, with recovery of the R waves, in addition to the disappearance of T wave changes.

SUMMARY

Ninety-seven consecutive cases of angina of effort have been reviewed: 82 were available for re-interview; in 62 of these typical angina of effort was present and pain had never occurred at rest or persisted longer than 15 minutes.

If the routine cardiogram did not reveal diagnostic evidence of infarction an additional cardiogram was taken comprising standard and unipolar limb leads, 6 unipolar precordial leads in the positions 1–6 and 6 high unipolar precordial leads.

Diagnostic evidence of myocardial infarction was present in 17 cases, and in a further 14 cases the records were abnormal and suggestive of infarction or ischaemia.

The pathological evidence concerning the relationship between coronary occlusion and myocardial infarction is briefly discussed and related to the clinical data presented. Our clinical observations are not inconsistent with the view that in the absence of other causes of relative myocardial ischemia such as cardiac hypertrophy, angina of effort is usually due to coronary occlusion with or without subsequent myocardial infarction. It is suggested that the sudden appearance of angina of effort, or the sudden exacerbation of symptoms in established angina, should be regarded as an indication of coronary occlusion at that time whether the electrocardiogram shows evidence of infarction or not.

We wish to thank Professor Crighton Bramwell, in whose department these observations were made, for his advice and help, and Mr. T. Corless for his technical help in the preparation of the electrocardiograms.
Fig. 6.—Normal electrocardiogram from a case of recent angina of effort in which death occurred 10 days later.

REFERENCES

——— (1949a). Ibid., 37, 205.
——— (1949c). Ibid., 38, 547.