Impending Myocardial Infarction
Recognition and Management

By R. E. Beamish, M.D., F.R.C.P. (C), and V. Marie Storrie, M.D.

It has long been known that attacks of myocardial infarction are frequently preceded by premonitory pain. The reported incidence of such warning symptoms usually varies from 30 to 50 per cent and the fatality rate in these series has ranged from 15 to 43 per cent.

Soon after introduction of anticoagulants in the treatment of acute myocardial infarction, several authors^1–3 conjectured that this therapy might well provide a means of preventing attacks. Stimulated by the results of Wood,^1 the present study was begun in 1949. A review of this experience suggests that impending infarction can be recognized with a high degree of accuracy and that the prompt administration of anticoagulants favorably influences the outcome.

Definition and Diagnostic Considerations
Since the earliest clinical studies on myocardial infarction it has been recognized that instances are frequent in which premonitory pain foreshadows the event.^4–6

Mounsey^6 placed the prodromal symptoms of myocardial infarction in the ill-defined borderline state between angina of effort and myocardial infarction. This state has been referred to as "coronary failure," (Blumgart et al.^7) spontaneous "acute coronary insufficiency" (Master),^8 "intermediate coronary syndrome" (Graybiel),^9 and "slight coronary attacks" (Smith and Papp).^10 The pathologic basis of the syndrome is no doubt variable; since it commonly develops rather abruptly, it is reasonable to attribute it to coronary thrombosis, but without accompanying myocardial infarction. The outcome of the state is also variable: the patient may return to normal health, may begin or continue to have angina of effort, or may go on to myocardial infarction. The relative incidence of these 3 modes of termination is not easily obtainable in a hospital series, since few patients who do not suffer from infarction are sent to the hospital. From observations of the present study carried out in the home, office, and hospital, it is apparent that these attacks are more significant and dangerous than has been commonly realized.

Mounsey's^6 found prodromal pain in 40 of 139 cases of myocardial infarction. This incidence of 29 per cent is lower than the 48 per cent and 50 per cent found by Sampson and Eliaser^11 and by Fell,^12 respectively, and higher than what Yater et al.^13 found (9.5 per cent) in men under the age of 40. Mounsey^6 suggested that the frequency is sufficient to make a recognizable clinical group. The pain was typical of cardiac ischemia in site, radiation, and quality, but differed from classical angina of effort in that there was a crescendo nature in the occurrence of symptoms, the attacks were more prolonged, and there was an inconstant relation of the pain to effort. The fatality rate in patients with prodromal symptoms was 15 per cent in Mounsey's series,^6 and 34 per cent and 43 per cent in Sampson and Eliaser's,^11 and Yater's series,^13 respectively.

Myocardial infarction may be manifest merely as angina pectoris of effort.^14 However, not infrequently myocardial infarction is preceded by an intensification of a previous angina of effort.^^1,6 The pathologic counterpart of a sudden increase in angina is frequently an occlusion of one or more major branches of the coronary arteries.
In a patient who has had a previous infarct and who has been subsequently pain-free, the recurrence of pain is particularly ominous. In such patients with known coronary disease, a more or less abrupt recurrence of pain indicates a sudden increase in the occlusive disease. In this situation it is reasonable to anticipate an increased incidence of infarction.

On the basis of these considerations and from the observations of the present series, it is suggested that impending myocardial infarction has 3 common presentations:

I. Onset of ischemic cardiac pain in a patient previously free of symptoms. Usually the patient experiences repeated bouts of spontaneously occurring pain lasting from 15 to 30 minutes or longer, without clinical, electrocardiographic, or laboratory evidence of infarction. Less commonly, it may take the form of a rapidly progressive angina of effort. Whether the pain be at rest or with exertion, it occurs oftener, lasts longer, and becomes more severe over the course of a few days or weeks.

II. Intensification of angina of effort in a patient with previous angina of several months' or years' duration. Usually there is an abrupt change in the pattern of the chronic symptoms: pain occurs oftener, occurs with less provocation, lasts longer, and is more severe.

III. Recurrence of pain, at rest or on slight provocation, in a patient who has been pain-free after a previous myocardial infarction.

This classification is somewhat similar to that of Denham based on study of the acute phase of myocardial infarction in 920 patients.

Patients with symptoms of impending infarction are to be distinguished from those with potential recurrent infarction as described by Nichol and Fassett and others. In this latter group, patients with previous infarction are placed on long-term anticoagulant therapy to prevent a recurrence of infarction. In group III of the present series the indication for anticoagulants was the recurrence of pain rather than the mere fact of previous infarction.

Whenever a patient is suspected of impending myocardial infarction because of ischemic pain, the electrocardiogram may provide helpful confirmatory evidence. The changes consist of primary T-wave abnormalities or S-T depressions. Such alterations are particularly helpful if electrocardiograms before the onset of pain are available for comparison. It must be stressed, however, that the clinical diagnosis must be acted upon even if the electrocardiogram is normal. Unfortunately it has been an all too common experience to have patients report to a physician because of pain, have an electrocardiogram done, be told that it is normal, be reassured, and then die suddenly a short time later. We have frequently observed that serial electrocardiograms, taken over a period of several days at the onset of treatment for impending infarction, may show transitory but conclusive evidence of ischemia. To wait for incontrovertible electrocardiographic changes is to waste precious time and may permit preventable infarction to occur.

Patients and Methods

From early 1949, all patients with symptoms of any of the 3 categories of threatened infarction were diagnosed as impending myocardial infarction and immediate hospitalization for anticoagulant treatment and rest was advised. By December, 1957, this diagnosis and recommendation had been made in 100 patients. In 85 of the 100 patients this recommendation was carried out; in the remaining 15 patients it was not. The latter patients therefore serve as "control" subjects.

All patients were seen by the same observers and the diagnoses were based on the same criteria. In some additional instances there was doubt at the onset of symptoms but treatment was started in any case. After a period of observation had enabled a diagnosis other than impending infarction to be established, such cases were eliminated from the series. Also, after some days of observation, it became apparent in a few cases that infarction had occurred at the time of presentation. These cases also were eliminated from the study. It must be constantly remembered that accurate diagnosis of impending infarction can sometimes be made only in retrospect but treatment must be initiated at the outset of a suspicious assembly of symptoms.

In the early years of the study patients were admitted to the hospital as soon as possible after diagnosis for treatment with anticoagulants. More recently, in a few cases, anticoagulants have been
administered in the office or home, pending admission to hospital. In addition to oral anticoagulants, in patients with rapid progression of symptoms, heparin was administered in a dose of 50 mg. intravenously every 4 hours until prothrombin activity reached a therapeutic level. A total of 21 patients received heparin, the remainder received only oral preparations. From 1949 to 1951 the oral anticoagulant used wasbishydroxycoumarin (Dicumarol); subsequently the usual preparation was phenylindanedione (Danilone). Later, ethyl bisoumaacetate (Tronexan) and acenocoumarin (Sintrom) were also used. Among the 85 cases that received anticoagulants, Dicumarol was given to 8 patients, Sintrom to 4 patients, Tromexan to 1, and the remaining 72 were given Danilone.

After admission to the hospital, patients received a complete physical examination, a 12-lead electrocardiogram, and roentgen examination of the chest. Routine laboratory investigation included complete blood count, Kahn test, estimation of sedimentation rate, fasting blood sugar, and serum cholesterol. Electrocardiograms and sedimentation rates were repeated several times during the first few days, and subsequently as indicated by symptoms; they were again performed prior to discharge from hospital, usually 2 to 4 weeks later. Serum transaminase and other enzymes were measured in more recent cases.

Patients were encouraged to remain in bed, usually with bathroom privileges, until therapeutic anticoagulation was achieved, and until they were no longer subject to pain. They then became ambulant. When levels of prothrombin activity were stabilized in the therapeutic range patients were allowed to go home. Reexaminations were carried out at intervals of 1 to 3 months, or as clinically required.

Prothrombin activity was estimated at the onset of treatment and daily thereafter while patients were in the hospital. After discharge it was estimated at weekly, and later at 2 weekly intervals. The prothrombin activity was estimated by the 1-stage method of Quick, the normal control value being 12 to 15 seconds. The upper and lower limits of the therapeutic range were considered to be 25 to 50 seconds (35 and 17 per cent, respectively).

It soon became apparent that there was considerable advantage in securing prompt control of prothrombin time with oral anticoagulants, thus permitting earlier discontinuance of intravenous injections of heparin. With the usually recommended doses of phenylindanedione, several days were often necessary before the therapeutic range was reached. Accordingly a study\textsuperscript{20} of optimum dosage to achieve rapid effect was undertaken.

\textsuperscript{*}For simplicity, the trade-names of these compounds are used hereafter.

\textit{Circulation, Volume XXI, June 1960}

<table>
<thead>
<tr>
<th>Age in years</th>
<th>Male</th>
<th>Female</th>
<th>Anticoagulant Male</th>
<th>Female</th>
<th>&quot;Control&quot; Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
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<td>30—39</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>1</td>
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<tr>
<td>40—49</td>
<td>16</td>
<td>14</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>50—59</td>
<td>38</td>
<td>31</td>
<td>7</td>
<td>7</td>
<td>1</td>
<td>1</td>
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<tr>
<td>60—69</td>
<td>19</td>
<td>12</td>
<td>11</td>
<td>11</td>
<td>3</td>
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</tr>
<tr>
<td>70 and over</td>
<td>6</td>
<td>1</td>
<td>5</td>
<td>5</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Totals</td>
<td>82</td>
<td>69</td>
<td>16</td>
<td>16</td>
<td>13</td>
<td>2</td>
</tr>
</tbody>
</table>

With larger doses (e.g., 500 to 600 mg. in the first 24 hours), a more rapid control could be safely effected. These larger doses were not used in patients over the age of 65 or in patients with any evidence of congestive heart failure. In the case of Tromexan, Dicumarol, and Sintrom, standard initial and maintenance doses were used.

**Clinical Features of Patients**

The age and sex of all patients included in this series are shown in table 1: 82 were male and 18 female. Age ranged from 31 years to 77 years and averaged 57 years. The group exhibited the distribution in sex and age characteristic of coronary disease in general. The 15 "control" patients were similar in average age and in sex distribution.

Presenting symptoms at the time of diagnosis in the 100 cases are shown in table 2. Patients in the "control" group were comparable to the treated group. The largest group consists of 45 patients who described the occurrence or onset of characteristic cardiac ischemic pain, which they had never had before. In this group a few patients began as angina of effort, which progressed rapidly, but in all cases one or more episodes of pain at rest had occurred before the diagnosis was made. Duration of symptoms prior to inclusion in the series varied from 1 day to 5 months, with an average of 16 days. Significantly, in 15 patients the duration of symptoms was 1 day or less.

The second most common mode of presentation was that of a rapidly progressive intensification of preexisting angina of effort. This occurred in 30 patients. In these patients angina had been present from 2 months to 15
years, with an average duration of 23 months. The period of intensification of symptoms varied from 1 day to 5 months and averaged 13 days.

The third group consisted of 25 patients with previous myocardial infarction in whom cardiac pain occurred after a pain-free interval of weeks, months, or years. The interval elapsing between the old infarction and the recurrence of fresh ischemic pain ranged from 1 month to 10 years and averaged 28 months. The duration of the new premonitory symptoms of an additional impending infarction ranged from 1 day to 1 month and averaged 7 days. It is apparent that these patients, having previously experienced an episode of infarction, reported the recurrence of pain much sooner than did the other 2 groups.

Duration of symptoms in the treated and "control" groups is compared in Table I. In the treated group, the duration of symptoms before diagnosis and administration of anticoagulants averaged 13 days. In the "control" group, duration of symptoms prior to diagnosis averaged 10 days. The interval from onset of symptoms to initiation of treatment in the treated cases, and the interval from onset of symptoms to the occurrence of infarction in the "controls," may be considered to be the numbers of days of "impending." It is seen that this interval was 13 days for the treated patients and 15 days for the "controls." In the "control" patients there thus existed a period averaging 5 days during which anticoagulants could have been administered.

Of the total series of 100 patients, 34 were obese, 20 had persistent diastolic hypertension, and 5 were diabetic. Incidence of these conditions was not significantly different in the treated and "control" groups.

Electrocardiograms were taken on all patients at the time diagnosis was established. In the 25 patients with known previous infarction, the old infarct was recognizable in 24, the remaining record showed marked left ventricular hypertrophy with S-T depression. Comparison with previous electrocardiograms indicated the presence of recent ischemia in 13 of the 24 patients (52 per cent).

In 75 patients without known previous infarctions, 13 patients (17 per cent) had normal records, 58 patients (77 per cent) had evidence of ischemia, and the remaining 4 showed left ventricular hypertrophy. Eleven of the 13 normal records remained normal and 35 of the 58 ischemic records improved.

Of the 15 "control" patients, 4 had normal tracings, 8 showed ischemia, 2 revealed old infarcts with recent ischemia, and 1 indicated left ventricular hypertrophy.

### Results of Treatment

It is difficult to assess the effect of treatment in impending infarction without taking into account the duration of treatment and its relationship to the time at which symptoms of "impending" are first manifest. Accordingly the first 6 weeks after diagnosis have been arbitrarily considered to be the "acute phase," the period from 7 weeks to 6 months the "chronic phase," and from 6 months onward the "late phase." Table 4 shows the outcome of the 85 treated patients compared to the 15 "control" patients in each of these phases.

In the 15 "control" patients it is almost

### Table 2

<table>
<thead>
<tr>
<th>Manifestation</th>
<th>Onset of new progressive cardiac pain</th>
<th>Intensification of chronic angina</th>
<th>Recurrence of pain after previous infarction</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration</td>
<td>Average (days)</td>
<td>16</td>
<td>13</td>
<td>7</td>
</tr>
<tr>
<td>Range</td>
<td>1 day to 5 months</td>
<td>1 day to 5 months</td>
<td>1 day to 1 month</td>
<td></td>
</tr>
<tr>
<td>Total cases</td>
<td>45</td>
<td>30</td>
<td>25</td>
<td>100</td>
</tr>
<tr>
<td>Anticoagulant</td>
<td>38 (45%)</td>
<td>24 (28%)</td>
<td>23 (27%)</td>
<td>85</td>
</tr>
<tr>
<td>&quot;Control&quot;</td>
<td>7 (47%)</td>
<td>6 (40%)</td>
<td>2 (13%)</td>
<td>15</td>
</tr>
</tbody>
</table>

*Note: Table 2 presents data on the incidence of symptoms and treatment outcomes.*
incredible to find that 14 infarcted. Of these, 11 were fatal and 3 were nonfatal. One of the patients with nonfatal infarction died of congestive heart failure 18 months later. One patient, a man of 66 years at the time of diagnosis, rested at home but remained ambulant for 2 months, and subsequently remained well except for mild angina of effort. The 3 nonfatal infarcts occurred from 3 to 4 days after diagnosis of impending infarction had been made. The 11 fatal infarctions occurred from 3 to 92 days (average 20 days) after diagnosis. However, if 2 patients who survived 56 days and 92 days respectively are excluded, the remaining 9 fatalities occurred on an average of 8 days after diagnosis, (range 3 to 19 days). Thus 12 patients infarcted, 9 of them fatally, during the "acute phase." Two of the 3 patients who reached the "chronic phase" then infarcted fatally.

Unfortunately no autopsies were performed in this group, as 8 patients died outside hospital and in the other 3, permission for necropsy was not granted. The manner of death was observed by a physician in 7 patients, and there seemed no doubt of infarction in 5, and that infarction was probable in 2. In 3 patients, death was sudden and unobserved. Three patients lived long enough to reach the hospital but died shortly after admission. In these patients, electrocardiograms confirmed acute myocardial infarction.

In no instance was infarction associated with undue effort or emotion. Two of the nonfatal attacks occurred while the patients were in bed and 1 while the patient was sitting in a chair. Eight of the fatal attacks occurred while patients were in bed, the other 3 during the ordinary activity of a housewife, a physician, and a merchant. It therefore does not seem reasonable to attribute these deaths to undue exertion or to lack of rest alone. One can only speculate about the probable outcome in these patients had anticoagulants been given, but in the light of results in treated patients, now to be presented, it seems likely that more would have survived.

Of the 85 patients who received anticoagulants during the "acute phase," only 2 had infarcts and neither of these was fatal. One of them occurred when the prothrombin time was temporarily above the therapeutic range. In 5 patients, anticoagulants were discontinued during this phase. Of the 80 patients carried on treatment to the "chronic phase," 3 infarcted, 2 being fatal. In the "late phase," reached by 57 treated patients, 10 suffered infarctions and 7 of these were fatal.

The benefits of anticoagulants in the "acute" and "chronic" phases appears substantial. Although the data are not susceptible to statistical analysis, the striking difference in outcome between the treated and "control" patients strongly suggests that anticoagulants exert a protective influence in the state of impending infarction. The incidence of fatal infarction and sudden death in the "chronic phase" appears to be a matter of the influence of long-term anticoagulants in patients with chronic coronary disease, rather than in the more restricted state of "impending infarction."

Ideally, one would prefer to have a large series with alternate patients serving as controls. In the present series the "controls" were selected in part by the patients, and in part by the chance unavailability of hospital accommodation. However, there was striking similarity to the treated groups in respect to age, sex, mode of presentation, duration of symptoms, and electrocardiographic findings.

The outcome in the 29 patients in whom anticoagulants were eventually discontinued is of interest. In 13 patients who had been treated for an average of 47 weeks (range 3 to 244 weeks), infarction occurred at a variable period after anticoagulants were discontinued. Of these, 4 were fatal and 9 were non-

### Table 3

<table>
<thead>
<tr>
<th>Duration of Symptoms in Treated and &quot;Control&quot; Patients</th>
<th>Anticoagulant</th>
<th>&quot;Control&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Days of symptoms before diagnosis</td>
<td>13</td>
<td>10</td>
</tr>
<tr>
<td>Days &quot;impending&quot;</td>
<td>13</td>
<td>15</td>
</tr>
</tbody>
</table>
fatal. Seven infarcts (3 fatal) occurred in the first 6 weeks after withdrawal, 6 (1 fatal) occurred over 6 weeks after withdrawal. It is impossible to know how many of these might have occurred even with continuation of treatment. A further 16 patients who had been on anticoagulants for an average of 24 weeks (range 2 to 131 weeks) had no cardiac complication on withdrawal but 2 suffered non-fatal cerebral thromboses. In the 6 patients in whom anticoagulants were discontinued because of bleeding, 3 infarcted, and 2 of these were fatal.

Outcome was not significantly influenced by the presenting symptoms. As might be expected, more infarctions occurred during anticoagulant treatment in those patients with previous chronic angina and in those with previous infarctions, but the small numbers of patients do not permit of valid deductions.

Complications of Treatment

Two unusual complications were encountered. One patient with a history of a previous infarction was given Danilone when symptoms of impending infarction developed. Due to excessive hypoprothrombinemia, hematuria and large ecchymoses occurred. Emulsified Vitamin K₁ (Mephyton) was administered intravenously and was immediately followed by a severe hemolytic reaction. Fortunately, the patient recovered. This appears to be the first instance of a reaction to the antidote to therapeutic hypoprothrombinemia.²¹

Another patient, with chronic angina of effort, was treated with Danilone when symptoms abruptly intensified. A sensitivity reaction to Danilone,²² characterized by hepatitis, dermatitis, and pyrexia, developed at the fifteenth day. Prompt recovery from the reaction followed administration of prednisone.

The main complication in this series was hemorrhage. During a total of 6,484 patient-weeks of therapy, hemorrhage occurred on 37 occasions. In 15 patients the bleeding consisted of hematuria, and in 6 patients epistaxis occurred. In 27 patients the hemorrhage was of minor degree and did not necessitate more than simple local measures and the temporary withholding of further anticoagulants. In 10 patients the bleeding was considered of major proportions and necessitated hospital treatment; in 6 cases the source of this severe bleeding was from the gastrointestinal tract. In no case was death directly attributable to hemorrhage although in 2 instances hemorrhage necessitated the discontinuance of anticoagulants and this in turn was promptly followed by fatal infarction.

Discussion

Despite its potential importance, little attention has been paid to recognition of impending infarction. Perhaps this lack of interest has been due, until recently, to inability to alter the outcome significantly. Prior to the advent of anticoagulants the principal treatment advocated has been rest,²³ but it has been stated²⁴ that only a small number of infarctions are provoked by undue demands on the heart, and many occur at rest, even during sleep. Now with the demonstrated value of anticoagulants, particularly during the "acute phase" of impending infarction, early recognition is of great consequence.

Reference has been made to the incidence of

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**Table 4**

<table>
<thead>
<tr>
<th>Outcome in Treated and &quot;Control&quot; Groups</th>
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<tr>
<td></td>
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<tr>
<td>&quot;Controls&quot;</td>
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<td></td>
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<tr>
<td>Entire series</td>
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<tr>
<td>Acute phase</td>
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<tr>
<td>Chronic phase</td>
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<tr>
<td>Anticoagulant</td>
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<tr>
<td>Chronic phase</td>
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<tr>
<td>late phase</td>
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<tr>
<td>No. of patients</td>
</tr>
<tr>
<td>No. of infarcts</td>
</tr>
<tr>
<td>No. of deaths</td>
</tr>
</tbody>
</table>

*Includes those occurring after anticoagulants were discontinued.
†One with prothrombin time above therapeutic range.
premonitory symptoms preceding myocardial infarction. In a recent study, Maurice et al. pointed out that in an impressive minority of cases, (39 per cent) infarction is not an unforeseeable event but is heralded by symptoms varying from a few hours to a month or more. Clinical features of their patients were similar to the present series, but more of them presented as an intensification of preexisting angina of effort.

Although it has taken nearly 9 years to assemble the present series, a more or less gradual increase in frequency of this diagnosis has been observed over the years. Part of this increase is no doubt due to the increasing number of cardiac patients seen by the authors but a portion of the increase is also due to a program of education of patients, whereby all patients with coronary disease are instructed to report to the physician any appreciable change in their cardiac pain. As a result more patients come under medical observation before they have progressed to the stage of actual infarction. If the preventive measures of anticoagulation and rest are of value, this program of patient education is important. There is, of course, the disadvantage that the physician is frequently informed of insignificant pains but after a few discussions patients soon learn which pains to report and which to ignore. In any case the advantages of early reporting of pain are considered to outweigh by far the occasional annoyances. This procedure might be crudely compared to having patients report potentially premalignant lesions to the physician before established cancer has occurred.

As early as 1944, Bayley and LaDue described the electrocardiographic findings of impending infarction and stated, "if infarction can be prevented or minimized at its impending stage, the recognition of this stage would be important clinically." Although the electrocardiogram provides welcome confirmatory value when abnormal, it is emphasized that 17 per cent of the present series, and 29 per cent of the patients of Maurice et al. had normal records.

The pathologic basis of impending infarction is even less well understood than the mechanism of coronary occlusion itself. Parkinson and Bedford considered that the prodromal pain announced the beginning of coronary thrombosis while the subsequent myocardial infarction marked complete occlusion. When premonitory symptoms develop abruptly, it seems likely that an acute change has occurred in the coronary circulation, whether it be thrombosis, subintimal hemorrhage, or occlusion by a plaque. The incidence and role of subintimal hemorrhage in production of coronary occlusion is not agreed upon, but its possible presence is sometimes stated to be a contraindication to the use of anticoagulants. In view of the beneficial effects of anticoagulants noted in this and other series, it would appear that either subintimal hemorrhage is not aggravated by anticoagulants, that secondary thrombosis in relation to the subintimal hemorrhage is prevented, or that it is not a common event.

Because the period of prodromal symptoms is often short, there is urgency in achieving anticoagulant effect. Thus the more quickly acting oral anticoagulants have an advantage. The necessity for heparin at the initial stage has not been established but its use seems rational. Maurice et al. used it in 46 of 71 patients. In the present series it was used in 21 of the 85 patients; in none of these did death or infarction occur during the "acute phase," but 1 patient, adequately controlled, died 20 weeks later.

The reported incidence of infarction (usually 30 to 50 per cent) and of fatality (15 to 43 per cent) in patients with prodromal symptoms has been mentioned. The reduction of both by use of anticoagulants in this series is impressive, both by comparison with a "control" series and with published studies. Further support is afforded by results of anticoagulant therapy of impending infarction reported by several authors. Wood treated 33 patients with "acute coronary insufficiency" with a result of 2 infarcts and no deaths; in 25 similar untreated cases there
were 12 infarcts and 5 deaths. Nichol et al.\textsuperscript{28} recently reported 6 per cent mortality in 96 treated patients and 18 per cent mortality in 32 patients abandoning treatment. Maurice et al.\textsuperscript{29} observed only 2 infarctions in 71 patients and were impressed with the fact that 64 per cent of patients became free of pain. The similarity in these reports is persuasive.

The mechanism causing death in patients with coronary atherosclerosis is often not clear, even after autopsy examination. Patients may die with no evidence of infarction, and patients with infarcts may live with little manifestation of the cardiac lesion. In assessing the effect of anticoagulants in patients with coronary disease, it is necessary to take into account not only the infarctions which may or may not be prevented, but also the reduction in "sudden deaths" by whatever mechanism.

**Summary**

Coronary occlusion with myocardial infarction is a common condition with a high mortality. The mechanism of coronary occlusion in such cases is not always clear, but thrombosis, either directly or indirectly, is a dominant process.

In many instances premonitory symptoms give warning that myocardial infarction is threatening. It is suggested that these symptoms form a recognizable pattern which permits recognition of impending myocardial infarction with a high degree of accuracy. Three common presentations are described.

Frequency of diagnosis of impending infarction may be increased by a program of patient education.

Prompt administration of anticoagulants appears to influence the outcome favorably, particularly in the first few days or weeks of symptoms.

**Summario in Interlingua**

Occlusion coronari con infarcimento myocardial es un condition common con alte cifras de mortalitate. Le mechanismo del occlusion coronari in tal casos non es semper clar, sed thrombose—directe o indirecte—es un proceso dominante.

In multe casos symptomas premonitori annuncia le imminente infarcimento myocardial. Es opinare que iste symptomas forma un recognoscibile ensemble que permette le recognition del veniente infarcimento myocardial con alte grados de accuratia. Tres situationes commun es describite.

Le frequentia del diagnose de imminentea de infarcimento myocardial pote esser augmentate per un programma de education del paciente.

Ti pare que le prompte administration de anticoagulantes exerce un influentia favorabile super le resultato final, specialmente durante le prime dies e septimanas del symptomas.

**References**

IMPEENDING MYOCARDIAL INFARCTION


Vesalius

“My study of anatomy,” says Vesalius, “would never have succeeded had I when working at medicine at Paris been willing that the viscera should be merely shewn to me and to my fellow students at one or another public dissection by wholly unskilled barbers, and that in the most superficial way. I had to put my own hand to the business.” . . .

Complete dissection was then well-nigh impossible, the most that could be gained was the hurried examination of some parts of the body of a patient who had succumbed to disease. One part of the human body, the foundation of all other parts, the skeleton, could however be freely used for study. In those rude times burial was rough and incomplete, and in the cemeteries bones lay scattered about uncovered. In the burial-ground attached to the church of the Innocents at Paris Vesalius spent many hours, studying the bones; and he also tells us how in another burial-ground, on what is now ‘Les Buttes Chaumont,’ he and a fellow student nearly left their own bones, being on one occasion attacked and in great risk of being devoured by savage, hungry dogs who too had come there in search of bones. By such a rough, perilous study Vesalius laid the foundation of his great work, a full and exact knowledge of the human skeleton.—Sir M. Foster. Lectures on the History of Physiology. London, Cambridge University Press, 1901.
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