Early postinfarction ischemia: clinical, angiographic, and prognostic significance

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ABSTRACT Early ischemia, defined as angina with transient ST-T changes during hospitalization, 24 hr or more after an acute myocardial infarction (MI), was observed in 79 (18%) of a consecutive series of 449 patients surviving an MI and catheterized a mean of 10 ± 3 days after admission. Three clinical factors present 24 hr after admission could identify patients at low, medium, and high risk of subsequent ischemia: the 32 patients with a non–Q wave MI, previous angina, and two or more risk factors had a risk greater than 50% and the 118 patients with Q wave MI, no previous angina, and absence of risk factors had a risk of less than 8%. The angiographic correlates of early ischemia were number of vessels with 70% or more stenosis (2.1 ± 0.8 vs 1.7 ± 0.8/patient, p < .0001), number of diseased coronary artery segments (2.8 ± 1.4 vs 2.1 ± 1.2, p < .0001), left anterior descending coronary involvement (77% vs 62% of patients, p = .01), number of normally contractile segments at jeopardy because of a coronary stenosis (1.9 ± 1.3 vs 1.3 ± 1.1/patient, p < .0002), collateral circulation at jeopardy (24% vs 15% of patients, p < .005), and fewer collateral vessels distal to a tight stenosis (59 vs 72% of patients, p = .04). The stepwise logistic regression retained one angiographic and two clinical independent predictors of early ischemia: number of diseased vessels (p = .0008), presence of a non–Q wave MI (p = .0027), and previous angina (p = .017). Extension of the infarction during hospitalization was diagnosed in 31 patients (7% of the total population) and could be independently predicted only by the presence of early ischemia: it occurred in 22 of the 79 patients with ischemia (28%) vs nine of 370 without (2.4%, p < .0001). During a mean follow-up of 14 ± 8 months (2 to 28), cumulative survival was 83% in patients with early ischemia and 92% in those without (p = .01); survival without MI was 67% vs 81%, respectively. Thus, early ischemia after MI is a frequent finding that is clinically predictable. It is associated with more severe coronary artery disease and identifies, independently of non–Q wave MI and of extent of coronary artery disease, a group at high risk for MI extension in hospital and for cardiac events during follow-up.


RISK STRATIFICATION after myocardial infarction is generally based on the early clinical features associated with infarction1–10 and on the predischarge investigation.11–20 The early predictors are mainly clinical markers of infarct size1–6 and of infarct extension,7–10 whereas the late investigation is oriented to characterization of left ventricular function14, 15, 18–20 and detection of residual ischemia16–20 and electrical instability.11–13 The interim period of in-hospital early mobilization and rehabilitation receives relatively little attention. However, angina during that period is a frequent finding.21–27 A few studies have also reported a high incidence of subsequent cardiac events in affected patients22–28 and have suggested various pathophysiological mechanisms.22–29

The present investigation was designed to evaluate prospectively in a large series of consecutive patients admitted for acute myocardial infarction the prevalence of early ischemia, the clinical and angiographic correlates of this syndrome, and its prognostic implications.

Materials and methods

Between April 1982 and May 1984, 631 patients less than 70 years old and without previous coronary artery bypass surgery were admitted to the Coronary Care Unit of the Montreal Heart Institute with a confirmed diagnosis of acute myocardial infarction. Sixty-nine patients died in hospital, 37 within 24 hr of admission. Among the 32 patients who died after 24 hr, spontaneous ischemia had been present in 16. Coronary arteriography was performed before hospital discharge in 449 survivors; excluded were 23 patients with severe congestive heart failure, 23 with noncardiac contraindications to cardiac catheterization,
and 67 who were reluctant to accept early angiography. These 449 patients represent 80% of the survivors and form the study population. None of the patients included in this series received thrombolytic therapy nor had coronary angioplasty performed for the acute myocardial infarction.

**Diagnosis of myocardial infarction.** The diagnosis of an acute myocardial infarction was based on the Beta-Blocker Heart Attack Trial criteria and required two of the following three criteria: chest pain lasting 30 min or more, elevation of the serum level of creatine kinase to twice the upper limit of normal or presence of its MB fraction, and Minnesota code electrocardiographic criteria for an evolving acute myocardial infarction. In the absence of a new Q wave, both the chest pain and the enzyme criteria were required for diagnosis. Cardiac enzymes were measured every 6 hr during the first 24 hr, every 12 hr during the following 24 hr, and then daily until return to normal values. This procedure was repeated if the patient experienced recurrence of chest pain. A Q wave myocardial infarction was diagnosed in the presence of new Q waves (Minnesota code 1-1-1 through 1-3-1) and non-Q wave infarction when transient ST-T changes were present without new Q waves.

**Coronary angiography.** Coronary arteriography and left ventriculography were performed before hospital discharge a mean of 10 ± 3 days after myocardial infarction. A percutaneous transfemoral approach and preformed catheters were used. Each artery was filmed in four to six projections, including special angulated views in the sagittal plane. All angiographic images were recorded on 35 mm film at 50 frames/sec, reviewed on a Tagarno projector, and interpreted by a consensus of three observers unaware of the clinical status of the patients. The coronary tree was divided in 15 segments and the percent reduction in the internal vessel diameter in each segment was coded according to the criteria of the American Heart Association. Stenoses of 70% or more luminal diameter reduction of the right, left anterior descending, or circumflex branches of the left coronary artery and stenoses of 50% or more of the left main coronary artery were considered as significant. Stenoses of large diagonal or marginal branches were considered lesions of the left anterior descending and circumflex coronary arteries, respectively. The collateral circulation was judged present or absent by the consensus of observers. When present, it was classified as "jeopardized" when originating distal to a 70% or more narrowing and nonjeopardized when originating from a nonstenosed artery or proximal to a stenosis. The degree of residual coronary stenosis in the vessel supplying the infarcted zone was also determined. When both the left circumflex and the right coronary artery were involved in patients with inferior myocardial infarction, the artery showing the highest degree of stenosis was considered the vessel responsible for the infarct.

Left ventriculograms were obtained in the 30° RAO projection and analyzed by dividing the contour in five ventricular segments. Based on the known distribution of the coronary arteries, the anterobasal, anterolateral, and apical segments were assumed to receive their arterial blood supply from the left main and left anterior descending coronary arteries and the posterobasal and diaphragmatic segments were assumed to be supplied by right coronary artery or, in case of left dominant anatomy, by the left main or proximal circumflex vessels. The contraction pattern of the five ventricular segments was coded as 0 for normal or mild hypokinesis, 1 for more severe hypokinesis, 2 for akinesia, and 3 for dyskinesia. The contraction score of the infarct area was defined as the highest of the three individual scores of the three anterior segments in the case of anterior infarction and of the two inferior segments in the case of inferior infarction. This convention was used to avoid errors in selecting the infarcted segment, our interest being in the severity of wall motion involvement rather than in the extent of infarc-

**Results**

The study population consisted of 449 patients, 379 men and 70 women, with a mean age of 54 ± 9 years. One hundred thirty-six patients had a past history of hypertension, 158 that of angina, and 103 that of a myocardial infarction. The site of the acute infarct was anterior in 187 patients and inferior in 262. A Q wave infarction was present in 291 patients and a non-Q wave infarction was noted in 158. During the acute stage 318 patients were in Killip class I, 101 were in class II, 23 were in class III, and seven were in class IV.
TABLE 1
Clinical correlates of early ischemia

<table>
<thead>
<tr>
<th></th>
<th>Early ischemia</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present (n = 79)</td>
<td>Absent (n = 370)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>56 ± 8</td>
<td>54 ± 9</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>64</td>
<td>315</td>
</tr>
<tr>
<td>Female</td>
<td>15 (19)</td>
<td>55 (15)</td>
</tr>
<tr>
<td>Cholesterol levels (mg/dl)</td>
<td>225 ± 42</td>
<td>209 ± 49</td>
</tr>
<tr>
<td>Previous hypertension</td>
<td>27 (34)</td>
<td>109 (29)</td>
</tr>
<tr>
<td>Smokers</td>
<td>64 (81)</td>
<td>278 (75)</td>
</tr>
<tr>
<td>No. risk factors per patient</td>
<td>1.6 ± 0.7</td>
<td>1.4 ± 0.7</td>
</tr>
<tr>
<td>Duration heart disease (mo)</td>
<td>38 ± 50</td>
<td>24 ± 36</td>
</tr>
<tr>
<td>Previous angina</td>
<td>42 (53)</td>
<td>116 (31)</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>27 (34)</td>
<td>76 (21)</td>
</tr>
<tr>
<td>Site of acute MI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior wall</td>
<td>38 (48)</td>
<td>149 (40)</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>41 (52)</td>
<td>221 (60)</td>
</tr>
<tr>
<td>Non-Q wave MI</td>
<td>41 (52)</td>
<td>117 (32)</td>
</tr>
<tr>
<td>Peak CK (IU/l)</td>
<td>1280 ± 1121</td>
<td>1818 ± 1581</td>
</tr>
<tr>
<td>Peak MB-CK (IU/l)</td>
<td>188 ± 226</td>
<td>299 ± 323</td>
</tr>
<tr>
<td>Killip class</td>
<td>1.47 ± 0.7</td>
<td>1.35 ± 0.6</td>
</tr>
</tbody>
</table>

Values in parentheses are percentages; “plus or minus” values are mean ± SD.

MI = myocardial infarction; CK = creatine kinase.

Coronary angiography showed the absence of a significant stenosis in 14 patients, one-vessel disease in 178 patients, two-vessel disease in 148 patients, and three-vessel in disease in 109 patients. Fourteen patients had significant left main stenoses. Mean ejection fraction was 49 ± 13%.

Chest pain during hospitalization 24 hr or more after admission occurred in 164 patients and in 79 patients it was associated with transient ST-T changes. The prevalence of early spontaneous ischemia in this study was thus 18% (79/449). The ST-T changes were located in the infarct zone in 51 patients and at a distance in 28. The diagnosis of early spontaneous ischemia was made 4.5 ± 3.2 days after admission; 62% of the cases were diagnosed within the first 4 days.

Clinical correlates. The clinical characteristics of the 79 patients with early ischemia and of the 370 without ischemia are compared in table 1. There were no differences in age or in sex distribution. The serum cholesterol values obtained at admission to hospital were higher in the patients with early ischemia and they had more risk factors. The majority of patients in both groups were smokers. Patients with early ischemia also had a longer past history of heart disease and had more often experienced angina or a myocardial infarction. A non-Q wave myocardial infarction was encountered more frequently in these patients. Peak rise in creatine kinase was also less.

Angiographic correlates. The angiographic findings are listed in table 2. The left anterior descending artery, its proximal segment, and the circumflex coronary artery were all involved more frequently in patients with early ischemia (p = .01). The number of stenosed vessels per patient and of stenosed segments was also higher (p < .00001). Multivessel disease was more frequent in those with early ischemia (p < .005)

TABLE 2
Angiographic correlates of early ischemia

<table>
<thead>
<tr>
<th></th>
<th>Early ischemia</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present (n = 79)</td>
<td>Absent (n = 370)</td>
</tr>
<tr>
<td>No. of patients with ≥70% stenosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 VD</td>
<td>0 (0)</td>
<td>14 (4)</td>
</tr>
<tr>
<td>1 VD</td>
<td>20 (25)</td>
<td>158 (43)</td>
</tr>
<tr>
<td>2 VD</td>
<td>28 (35)</td>
<td>120 (32)</td>
</tr>
<tr>
<td>3 VD</td>
<td>31 (39)</td>
<td>78 (21)</td>
</tr>
<tr>
<td>LAD</td>
<td>61 (77)</td>
<td>230 (62)</td>
</tr>
<tr>
<td>Prox LAD</td>
<td>28 (35)</td>
<td>81 (22)</td>
</tr>
<tr>
<td>CCX</td>
<td>50 (63)</td>
<td>176 (48)</td>
</tr>
<tr>
<td>RCA</td>
<td>52 (66)</td>
<td>218 (59)</td>
</tr>
<tr>
<td>No. with 100% occluded infarct-related vessels</td>
<td>44 (56)</td>
<td>234 (63)</td>
</tr>
<tr>
<td>No. with collaterals to infarct-related vessels</td>
<td>42 (53)</td>
<td>234 (63)</td>
</tr>
<tr>
<td>No. with jeopardized collaterals</td>
<td>19 (24)</td>
<td>57 (15)</td>
</tr>
<tr>
<td>No. with collaters to ≥90% infarct-related vessel</td>
<td>41/69</td>
<td>232/322 (72)</td>
</tr>
<tr>
<td>No. of stenosed vessels per patient</td>
<td>2.1 ± 0.8</td>
<td>1.7 ± 0.8</td>
</tr>
<tr>
<td>No. of stenosed segments per patient</td>
<td>2.8 ± 1.4</td>
<td>2.1 ± 1.2</td>
</tr>
<tr>
<td>% stenosis infarct-related vessel</td>
<td>92 ± 18</td>
<td>93 ± 16</td>
</tr>
<tr>
<td>Jeopardized myocardium score</td>
<td>1.9 ± 1.3</td>
<td>1.3 ± 1.1</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>49 ± 13</td>
<td>49 ± 13</td>
</tr>
<tr>
<td>Contraction score</td>
<td></td>
<td></td>
</tr>
<tr>
<td>infarct area</td>
<td>1.5 ± 1</td>
<td>1.7 ± 1</td>
</tr>
</tbody>
</table>

Values in parentheses are percentages; “plus or minus” values are mean ± SD.

VD = vessel disease; LAD = left anterior descending coronary artery; CCX = circumflex coronary artery; RCA = right coronary artery.
and three-vessel disease was almost twice as frequent. Severity of the stenosis of the infarct-related vessel and presence of visible collaterals to this vessel were similar in patients with and without ischemia. However, the collateral circulation was jeopardized by a stenosis of 70% or more in significantly more patients with early ischemia and collaterals to an infarct-related vessel with a subtotal occlusion (≥ 90% stenosis) were less often observed in these patients (p = .04). Although the global contraction score and the ejection fraction were similar in both groups, normal or mildly hypokinetic left ventricular segments at jeopardy were more often found in patients with early ischemia (p < .0002). Ischemia at a distance compared with ischemia in the infarct zone was more frequent in those with Q wave (64% vs 39% of patients) and inferior infarction (79% vs 37%, p < .05). It was also associated with more frequent two- and three-vessel disease (93% vs 65% of patients, p < .003), three-vessel disease (68% vs 24%, p < .003), and complete occlusion of the infarct-related vessel (71% vs 47%, p < .04).

Independent predictors. Among the clinical variables that differed significantly between patients with and those without ischemia by the univariate analysis, the stepwise logistic regression retained three independent predictors: previous angina (p = .001), a non–Q wave myocardial infarction (p = .003), and the number of risk factors (p = .02). To evaluate the fit of the logistic model to actual presence of early ischemia, the prob-

### Table 3
Summary of stepwise logistic regression of variables associated with early spontaneous ischemia

<table>
<thead>
<tr>
<th></th>
<th>χ²</th>
<th>p value</th>
<th>βi</th>
<th>Odds ratio (95% confidence limits)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of vessels with ≥70% stenosis</td>
<td>11.2</td>
<td>.0008</td>
<td>.518</td>
<td>2.8 (2.1–3.8)*</td>
</tr>
<tr>
<td>Non–Q wave myocardial infarction</td>
<td>9</td>
<td>.0027</td>
<td>.39</td>
<td>2.2 (1.7–2.8)</td>
</tr>
<tr>
<td>History of angina</td>
<td>5.7</td>
<td>.017</td>
<td>.317</td>
<td>1.9 (1.5–2.4)</td>
</tr>
</tbody>
</table>

χ² = chi-square values based on maximum likelihood ratio test, 1 degree of freedom chi-square distribution; βi = correlation coefficient of the variables retained by the regression; odds ratio = ratio of the probabilities of a right and a wrong prediction.

*Three- vs one-vessel disease.

ability of early ischemia was computed for each patient and the total group was divided into classes defined by a calculated low, medium, and high probability of occurrence of ischemia. The results shown in figure 1 confirm the accuracy of the predicted probability of early ischemia ( Hosmer's statistic, p = .43 [NS]).

When the angiographic characteristics were also entered in the stepwise logistic regression, three independent predictors were retained: the number of vessels with 70% or greater stenosis (p = .0008), non–Q wave infarction (p = .0027), and a history of previous angina (p = .017). The relative risk for early ischemia in the presence of these factors is shown in table 3.

In-hospital prognosis. Extension of infarction during hospitalization, diagnosed by the same criteria as for the initial infarct, occurred in 31 of the entire population of 449 patients (7%). The clinical and angiographic factors associated with its occurrence are listed in table 4. They were older age, number of risk factors, previous infarction or angina, non–Q wave myocardial infarction, lower peak creatine kinase value, early ischemia, and lower ejection fraction. By stepwise logistic regression only early ischemia was retained as a significant predictor (p < .0001, odds ratio 15.4, 95% confidence limits 10 to 23). Twenty-two of the 31 patients with extension had experienced early ischemia and nine had not (p < .0001). The sensitivity of early ischemia for predicting infarct extension was thus 71% and the specificity 86%. Sixteen of the 51 patients with ischemia in the infarct zone had extension vs six of the 28 with ischemia at a distance (31% and 21%, NS). Coronary artery dilatation or bypass surgery was performed because of uncontrolled pain despite optimal medical treatment in 34 of the 164 patients with postinfarction angina. Early ischemia had been present in 15 of these patients.

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Comparison between number of patients with ischemia observed vs number predicted with the use of clinical variables available 24 hr after admission. Patients are separated into three categories of estimated risks. The number of patients in each risk group is indicated at the top of the bar; patients with and without early ischemia were counted in each estimate risk group, providing an observed rate of occurrence of ischemia (hatched sections of the bars). Low-risk group includes patients with Q wave myocardial infarction without prior angina and none or one risk factor. Patients with non–Q wave myocardial infarction and prior angina and two or three risk factors make up the high-risk group. The medium-risk group is defined by any combination of two of the three clinical factors associated with early spontaneous ischemia.
The results were analyzed by different groups and differences in survival and in survival without myocardial infarction developed mainly during the follow-up. The analyses revealed that the differences in survival and in survival without myocardial infarction were statistically significant (p < .001). Inspection of the survival curves showed that patients without early ischemia had a better prognosis compared to patients with early ischemia. The overall results were consistent with previous studies that demonstrated a better prognosis for patients without early ischemia. The Kaplan-Meier survival curves for patients with and without early ischemia are shown in Figure 2. The top and middle panels show, respectively, survival and survival without myocardial infarction in the entire study population. In the bottom panel, patients were considered lost to follow-up at the time of aortocoronary bypass surgery or coronary angioplasty. Cardiac events considered were mortality and myocardial infarction.
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the medical staff, instructions to the patients to report all episodes of chest pain, and the recording of 12-lead electrocardiogram before nitroglycerin administration whenever a patient complained of a chest pain. The special organization of our coronary care unit, where patients remain hospitalized until discharge under the care of the same medical staff, could certainly also have increased the detection rate. However, most of the anginal episodes occurred within the first days after the infarct so that a similar incidence would be expected in coronary care units in which patients spend shorter stays. In fact, the average time for detection of early spontaneous ischemia in this study was 4.5 days after admission and the diagnosis was made within the first 4 days in 62% of the patients.

The population of the study included patients with well-documented infarction diagnosed by the usual criteria who were catheterized an average of 1 week after admission. It did not include patients suffering early deaths or those who refused or had contraindications to angiography. However, 80% of all patients admitted during that period were included and only 23 patients were not catheterized for cardiac reasons. The study population thus accurately reflects the episodes of ischemia occurring during the hospital stay after an acute myocardial infarction, as assessed by clinical and electrocardiographic criteria. It does not, however, reflect all ischemic events because no attempt was made to systematically detect episodes of silent ischemia by Holter recordings or other means.

Pathophyslogic mechanisms. In this study, all of the indexes of severity and extent of coronary artery disease that were studied showed significantly more advanced disease in patients with early spontaneous ischemia. The multivariate analysis retained the number of vessels with 70% or more stenosis as the strongest correlate: 2.1 per patient with ischemia compared with 1.7 per patient without ischemia. This difference is accounted for by more frequent three-vessel disease in patients with early ischemia (39% vs 21%) and also more frequent multivessel disease (74% vs 53%). Thus, early ischemia after myocardial infarction appears to be a clinical indicator of the presence of multiple disease, with a specificity of 90%, and of three-vessel disease, with a specificity of 86%.

The other independent predictors of early ischemia in the multivariate analysis were a non-Q wave myocardial infarction and the presence of previous angina. Many studies have reported that the pathophyslogic mechanisms involved in non-Q wave myocardial infarction differ from those in Q wave infarction, with less frequent coronary artery thrombosis40,42 and less extensive injury but a higher propensity for postinfarction angina and reinfarction in patients with the former.4-10 Thus, despite an initial favorable prognosis, long-term survival after non-Q wave myocardial infarction is similar to, or even less favorable, than that after Q wave infarction.7, 9, 43, 44 A previous history of angina pectoris or of myocardial infarction has also been shown to be associated with more extensive coronary artery disease and with a poor prognosis.4, 18, 45 When only the clinical characteristics were entered in the logistic regression, the number of risk factors also became an independent predictor. Combining the three clinical characteristics it was possible 24 hr after admission to accurately predict subgroups at high, medium, and low risk of occurrence of early ischemia (figure 1), with a non-Q wave myocardial infarction being one of the major determinants.

Additional factors may also be operative in early ischemia. Although no differences were found in the total number of collaterals, these were less developed in patients with early ischemia when the residual coronary stenosis was 90% or more and were more often at jeopardy because of a coronary artery stenosis. Patients with early ischemia also had a higher jeopardy score of normal or mildly hypokinetic left ventricular segments subtended by a coronary artery with a 70% or greater stenosis.

Reoclusion of a patent but severely stenosed coronary artery can also be a factor, particularly when ischemia is located in the infarct zone; in these patients the incidence of patent infarct-related vessel and of non-Q wave myocardial infarction is higher. Another cause could be severe multivessel disease, with the actual infarction precipitating an unstable state possibly in relation to abrupt changes in the collateral circulation39; this can be a mechanism for ischemia at a distance, which is associated with more extensive coronary artery disease. Observations made during coronary angioplasty in patients with single-vessel disease and ischemia, which is sometimes observed in areas remote from the primary zone, also suggest that occlusion of an unusually extensive coronary artery can explain ischemia at a distance in some patients.46

Prognosis. The diagnosis of infarct extension was made in this study based on the usual clinical criteria. The incidence in the hospital was 7%, significantly lower than the incidence that has been reported when serial electrocardiographic or MB–creatine kinase analyses were obtained.25, 47-49 Despite this relatively low incidence, the presence of early ischemia was clearly predictive of infarct extension: extension occurred in 28% of patients with ischemia and 2% of
patients without it. This difference was present despite more intensive medical treatment and more frequent coronary angioplasty and bypass surgery in patients with early ischemia. Indeed, early ischemia was the only independent predictor of infarct extension retained by the multivariate analysis. Thus, although extent of coronary artery disease and non-Q wave myocardial infarction could predict occurrence of early ischemia, their influence on prognosis appears indirect and related to the presence of early ischemia. Prognosis may be better determined by examination of dynamic factors than by fixed clinical entities. This could explain why prognosis was relatively independent of the site of ischemia in this study.

The prognostic value of early ischemia extended to follow-up and its presence was associated with a twofold increase in the rates of mortality and of myocardial infarction during a mean follow-up of 14 ± 8 months. On the other hand, when early ischemia was absent, the prognosis was favorable. Schuster and Bulkley reported a 6 month mortality of 56% with early ischemia. Figueras et al. showed no worsening of prognosis during a follow-up of 26 months. The populations of these two series were selected; the former included patients referred from other hospitals specifically because of postinfarction complications and the latter excluded patients with non-Q wave myocardial infarction. Other reports have included postinfarction angina and other variables in the multivariate analysis: in one series, postinfarction angina was an independent predictor of prognosis along with hypertension, three-vessel disease, and previous myocardial infarction. In another series, peak treadmill workload achieved, the change in ejection fraction during exercise, and recurrent ischemic pains were predictors of cardiac events. Angina after discharge from the coronary care unit, in another report, identified patients at high risk of rehospitalization and recurrent myocardial infarction in the following year.

The difference in the incidence of cardiac events was especially marked during the first 3 months of follow-up, suggesting that the unstable state present during hospitalization extends to the first few months after infarction. Late mortality may be relatively less because of the early attrition of the high-risk patients. It is also possible that different pathophysiologic mechanisms are operative at different times during follow-up and thus that management should be different also. In a 5 year follow-up of another series of patients, we found that the clinical indexes related to ischemia were mainly predictors of cardiac events during the first year, whereas factors related to left ventricular function were predictors of events occurring after 1 year.

This study does not represent, strictly speaking, a natural history study, since the results of the coronary angiographic examinations performed in all patients were available to the patient's physicians. This information could have had an independent influence on patient treatment and prognosis.

To avoid a possible confounding effects of surgery or angioplasty on prognosis, an analysis was performed in which patients were considered lost to follow-up from the time they underwent these interventions. This procedure did not affect the overall results. This observation does not imply that revascularization is either good or bad since this study was not designed to define its role. Moreover, revascularization was not performed prophylactically, but when it was clinically indicated for uncontrolled symptoms in the presence of suitable coronary anatomy.

This study confirms the high prevalence of early ischemia after myocardial infarction in a consecutive series of unselected patients and shows that it constitutes an unstable state. A high degree of vigilance to detect its presence is thus indicated, considering that ischemia is one of the postinfarction complications that is most amenable to medical and surgical treatment. Aggressive investigation and medical and surgical treatment are indicated in these patients.

We thank Ms. Luce Bégin for her excellent secretarial work.

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