Angiographic Findings 1 Month After Myocardial Infarction: A Prospective Study of 259 Survivors

AMADEO BETRIU, M.D., ANGEL CASTAÑER, M.D., GINES A. SANZ, M.D., J. CARLOS PARE, M.D., EULALIA ROIG, M.D., SANTIAGO COLL, M.D., JORGE MAGRÍÑA, M.D., AND FRANCISCO NAVARRO-LOPEZ, M.D.

SUMMARY Coronary anatomy as it relates to left ventricular function was assessed prospectively in patients who survived acute myocardial infarction. The study population included 259 consecutive male patients age 60 years or younger who underwent catheterization 30 days after the acute event. Coronary artery obstructive lesions (> 50% reduction in luminal diameter) were found in 241 patients (93%), 118 (45%) of whom had total and 76 (29%) subtotal (> 90%) stenosis) occlusion of at least one coronary artery. Normal coronary vessels were seen in eight patients (3%) and nonobstructive lesions in 10 (4%). One-, two- and three-vessel disease were present in 89, 86 and 66 patients, respectively. Patients with normal coronary arteries or nonobstructive lesions had higher ejection fractions than those with obstructive lesions in one, two or three vessels (p < 0.05). Ejection fraction was lower (p < 0.001) and the percentage of akinetic segments higher (p < 0.001) in patients with total or subtotal lesions and no collaterals. Adequate collaterals, seen in 29 patients (11%), significantly improved regional wall motion (p < 0.05) and decreased the percentage of akinetic segments (p < 0.001). Thus, in a substantial number of patients (32% in our series), the infarced area is spontaneously reperfused by collaterals or through the involved artery. Both mechanisms ameliorate wall motion in corresponding areas.

CORONARY ARTERIOGRAPHY is not routinely performed after myocardial infarction. Most series, being retrospective, deal with selected patients catheterized at variable time intervals after the acute event.1-4 As a result, information from such series may not represent the overall spectrum of lesions associated with myocardial infarction.

Prospective studies are scarce,5-10 and because they exclude a significant number of potential candidates (30-70%), their results might be subjected to bias. In 1975, we designed a prospective protocol that includes cardiac catheterization within 1 month after infarction. Our study entered 91% of all male patients age 60 years or younger who survived myocardial infarction. The results from the first 259 patients form the basis of the present report. The purposes of our investigation were to determine the prevalence of coronary artery lesions of different degrees, to analyze left ventricular function as it relates to the extent and severity of coronary artery lesions, and to assess the influence of collaterals on regional wall motion.

Methods

Patients

From January 1975 to March 1979, 300 male patients age 60 years or younger were admitted to the coronary care unit at the Hospital Clinico, University of Barcelona, with a definite myocardial infarction. There were 284 survivors, 25 of whom were excluded because they either refused cardiac catheterization (21 patients) or had life-threatening conditions (cancer in two patients and severe cor pulmonale in two others). The remaining 259 patients entered the study. At the time of this study, neither heparin nor lidocaine was routinely used.

Criteria for Myocardial Infarction

Acute myocardial infarction was diagnosed when at least two of the following were present: ischemic chest pain lasting more than 20 minutes, typical rise and fall of enzymes, and evolving Q-wave abnormalities with acute ST-segment and T-wave changes on the ECG. Previous infarction was diagnosed by a history of a hospital admission for documented myocardial infarc-
tion or ECG Q-wave abnormalities diagnostic of old infarction.

Catheterization and Angiographic Procedures

Four weeks after infarction, right- and left-heart catheterization and selective coronary arteriography by the percutaneous femoral approach were performed. Pressures were recorded on a Hewlett Packard multichannel photographic recorder using Statham P23Db transducers and fluid-filled catheters. Cardiac output was measured by the Fick method. Single-plane left ventriculography was performed in the 30° right anterior oblique position. Coronary arteriograms were obtained in multiple projections, including angulated views in the sagittal plane. All angiographic images were recorded on 35-mm film at 50 frames/sec and reviewed on a Tagarno projector. Left ventricular volumes and ejection fraction were determined using the area-length method modified for single-plane calculations.11 The extent of abnormal segmental contraction was estimated according to the method of Feild et al.12 Briefly, the length of the akinetic or dyskinetic segment was measured and expressed as a percentage of the total end-diastolic circumference. To assess regional wall motion, five areas of interest — anterobasal, anterolateral, apical, diaphragmatic and posterobasal — were constructed by drawing a longitudinal axis from the midpoint of the aortic valve to the apex and bisecting it by two equidistant chords. The two apical areas were combined and treated as a single region. The systolic change for each region was measured as the segmental ejection fraction.13

Angiograms were assessed by two independent observers, who coded the degree and location of coronary artery lesions. Discrepancies were resolved by caliper measurements of the percent reduction in luminal diameter in the projection showing maximal stenosis. Total coronary occlusion was defined as the absence of forward flow of contrast material in the involved artery. Subtotal occlusion was characterized by more than 90% reduction in luminal diameter. Lesions of less than 50% were considered nonobstructive. Each patient was classified as having one-, two- or three-vessel coronary artery disease. Obstructions of diagonal and marginal branches were considered lesions of the left anterior descending and circumflex coronary arteries, respectively. Collaterals were judged well developed when the diameter of the receiving vessel measured more than 1 mm and the vessel of origin showed nonobstructive lesions. Complete revascularization by collaterals was assumed when all vessels with total or subtotal occlusion were filled by well-developed collaterals.

Statistical Analysis

Continuous variables were analyzed by unpaired t test. Comparisons between multiple groups of data were carried out by analysis of variance followed by the appropriate multiple range test. Discontinuous variables were evaluated by contingency tables with the chi-square statistic. A p value less than 0.05 was considered significant.

Results

Clinical Findings

Acute-phase mortality for all patients with acute myocardial infarction admitted to our coronary care unit between January 1975 and March 1979 was 13%. However, only 16 male patients age 60 years or younger (the study population) died before coronary angiography. A higher prevalence of congestive heart failure in the acute phase (68%, p < 0.001) was found in this group. No differences in mean age (57.3 ± 5.2 years), previous infarction, infarct location or prevalence of risk factors were found between survivors and nonsurvivors in this series.

There were no differences between participants (n = 259) and nonparticipants (n = 25) regarding age, previous infarct, peak CK, congestive heart failure or prevalence of risk factors. Inferior myocardial infarction was more frequently found among nonparticipants (42% vs 75%, p < 0.001).

In patients entering the study, myocardial infarction was anterior in 107 (41%), inferior in 110 (42%) and combined anterior and inferior in 30 (12%). In 12 patients (5%), the infarct was nontransmural. Previous myocardial infarction was present in 24 patients (9%).

Coronary Artery Lesions

One hundred eighteen patients (45%) had complete occlusion of at least one coronary artery. Thirty-two patients also had coronary artery stenoses greater than 90% in other vessels. Seventy-six patients (29%) had maximal lesions of 90–99% and 34 patients (13%) had lesions of 75–89% narrowing. Thus, 88% of patients had greater than 75% stenoses of one major vessel. Only 13 patients (5%) had 50–74% lesions and the remaining 18 (7%) had nonobstructive lesions. Eight patients (3%) had normal coronary arteries (fig. 1).

Lesions greater than 50% were confined to one major artery in 89 patients (34%), whereas two- and three-vessel disease were found in 86 (33%) and 66 (26%) patients, respectively. Only three patients had obstructive lesions of the left main coronary artery. Two percent of the patients with one-vessel, 9% with two-vessel and 21% with three-vessel coronary artery disease had a history of myocardial infarction (fig. 2). Patients with normal coronary arteries were younger than those who had diseased vessels (p < 0.001). Five of 11 patients younger than age 35 years had normal coronary angiograms; the four patients age 30 years or younger had normal coronary vessels. In patients younger than 40 years old, there was a high prevalence (p < 0.001) of nonobstructive (eight patients, 32%) or one-vessel (11 patients, 44%) coronary artery disease.

Collaterals

There were 194 patients with total or subtotal stenoses in one (142 patients), two (40 patients) or three (12 patients) vessels. Collaterals were identified in 81 patients with one-vessel disease, 26 with two-vessel disease and eight with three-vessel disease. The collaterals were judged well developed in 40, 17 and two patients, respectively. Two patients with lesions greater than 90% in two vessels had well-developed collaterals to both. Complete revascularization, such
as that offered by well-developed collaterals to all obstructed vessels, was seen in 29 patients (11%): 28 with critical lesions in one vessel and one patient with two-vessel disease.

Left Ventricular Function

Hemodynamic and angiographic data are listed in table 1. No differences in cardiac index were found, but the left ventricular end-diastolic pressure was lower (p < 0.05) in patients with normal coronary arteries or nonobstructive lesions than in those with three-vessel disease. Stroke work index was higher in the former group of patients than in subjects with two- and three-vessel coronary artery disease (p < 0.05). Patients with three-vessel disease showed a larger end-diastolic volume than those with normal coronary arteries or nonobstructive lesions (p < 0.05). Patients with normal arteries or nonobstructive lesions had higher ejection fractions (p < 0.05) than patients with obstructive lesions in one, two or three vessels. The ejection fraction in patients with two-vessel coronary artery disease was not different from that in patients with one- or three-vessel disease, but the ejection fraction was significantly lower in patients with three-vessel disease than in those with one-vessel disease (p < 0.05).

Figure 3 shows the frequency distribution of ejection fraction in the present series. Severe left ventricular dysfunction (ejection fraction less than 30%) was found in 44 patients (17%), while moderate impairment of left ventricular function (ejection fraction 30–50%) was seen in 122 patients (47%). The remaining 93 patients (36%) had a normal ejection fraction. Patients with previous myocardial infarction accounted for 23% of those with severely depressed ejection fraction; only 4% had normal left ventricular function (p < 0.001).

Ejection fraction was lower and the percentage of akinetic segment higher in patients with a total or subtotal occlusion in corresponding arteries (p < 0.001) (fig. 4). Well-developed collaterals improved ejection fraction and significantly decreased the size of the akinetic segment (p < 0.001). Excluding the 27 patients with a previous infarct from the analysis did not modify our results.

Regional wall motion was analyzed in the 238 patients with a first myocardial infarction. In anterior infarctions, anterolateral (p < 0.02) and apical (p < 0.01) wall motion was significantly depressed and was more pronounced in patients with total or subtotal occlusion of the left anterior descending artery and no collaterals. Similarly, in inferior infarcts, apical and diaphragmatic wall motion was significantly depressed (p < 0.05 and p < 0.02, respectively) when the corresponding artery had at least 90% stenosis and no collaterals (fig. 5).

Discussion

Most of our knowledge of coronary artery lesions associated with acute myocardial infarction is based on pathologic data. Because autopsy series involve a very selected sample of the population, they do not represent overall anatomic findings after infarction. Although coronary artery lesions can be assessed by selective coronary arteriography, there has been a reluctance to perform invasive procedures early after infarction. Thus, angiographic studies soon after infarction are scarce.

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

**Figure 1.** Severity of coronary artery disease as assessed by the degree of maximal lesions 1 month after infarction. The shaded area represents the number of patients with subtotal lesions in other vessels. nl = normal.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Number of diseased coronary arteries in the study population. The shaded areas in the last three bars represent the number of patients with previous myocardial infarction. nl = normal.
Table 1. Hemodynamic Findings After Myocardial Infarction

<table>
<thead>
<tr>
<th></th>
<th>Nonobstructive lesions (n = 10)</th>
<th>1-vessel (n = 89)</th>
<th>2-vessel (n = 86)</th>
<th>3-vessel (n = 66)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CI (l/min/m²)</td>
<td>3.5 ± 0.8</td>
<td>3.5 ± 0.9</td>
<td>3.2 ± 0.8</td>
<td>3.3 ± 0.9</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>11 ± 4</td>
<td>14 ± 6</td>
<td>15 ± 7</td>
<td>17 ± 9</td>
</tr>
<tr>
<td>LVSWI (g·m/m²)</td>
<td>75 ± 29</td>
<td>57 ± 19</td>
<td>43 ± 16</td>
<td>45 ± 12</td>
</tr>
<tr>
<td>LVEDVI (ml/m²)</td>
<td>64 ± 14</td>
<td>72 ± 25</td>
<td>77 ± 26</td>
<td>82 ± 21</td>
</tr>
<tr>
<td>EF (%)</td>
<td>57 ± 10</td>
<td>49 ± 14</td>
<td>42 ± 13</td>
<td>38 ± 14</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

Abbreviations: CI = cardiac index; LVEDP = left ventricular end-diastolic pressure; LVSWI = left ventricular stroke work index; LVEDVI = left ventricular end-diastolic volume index; EF = ejection fraction.

The results in prospective studies may vary, depending on the population, the age and sex of the subjects and the selection criteria. Our study included a higher percentage of patients who survived infarction than did other series (table 2). Taylor et al.⁷ studied 109 survivors of transmural and nontransmural infarcts, but only 39% of the possible candidates underwent catheterization. Turner et al.⁸ evaluated 92 patients within 1 month of infarction; these patients represented 64% of those deemed candidates for angio- graphy. De Wood et al.⁹ performed coronary angiography in 322 of the 1210 patients (26.6%) admitted within 24 hours after the onset of symptoms. Bertrand et al.⁸ specified neither criteria for exclusion nor the number of rejected patients.

There are two reasons for the higher rate of inclusions in our study: Few patients refused catheterization and no patient was excluded because of a poor cardiac condition. Although we analyzed a limited age group of men, our series included 70% of all patients admitted to our coronary unit with myocardial infarction. We chose an age limit of 60 years to increase the likelihood of a 10-year follow-up.

Females were excluded from the study to increase the uniformity of the sample, as infarction in women is still uncommon in our environment. Only 11 women younger than age 60 years were admitted with acute myocardial infarction during the time of the study. (male:female ratio 27.2:1). In addition, the incidence of symptomatic artery disease in women parallels that of men, with a time lag of 8–10 years.¹⁰ Thus, angiographic findings documented in the present study should not be seriously distorted by exclusion of females.

Figure 4. Influence of degree of lesions on ejection fraction (EF) and percentage of akinetic segments (AK). The ejection fraction is lower and the percentage of akinetic segments higher in patients with total or subtotal occlusion. Well-developed collaterals (shaded bars) improved ejection fraction and significantly decreased the size of the akinetic segment.
Coronary angiography can be safely performed within the first hours of infarction. However, when the study was started, we considered 30 days a reasonable compromise between time and safety that would allow assessment of existing chronic lesions.

In our patients, one-, two- and three-vessel disease were evenly distributed. The prevalence of left main coronary artery obstructive lesions was significantly lower than that reported by Taylor et al. (1.1% vs 11%). The small number of potential candidates for angiography and the high percentage of three-vessel disease (53%) in the series of Taylor et al. suggest that their results were biased by patient selection. Turner et al. reported similar prevalence (10%) of left main lesions, even though they used stricter criteria for obstructive lesions (> 70% occlusion).

De Wood et al. showed that the incidence of total coronary occlusion is high during the first 4 hours after the onset of symptoms, but decreases significantly (65%) after 12-24 hours. These results suggest that progression from total to subtotal occlusion occurs in some patients. Although to prove reversal of total coronary occlusion would require serial studies in a given patient, comparison of the data of De Wood et al. with those of Bertrand et al. and our own series (53% and 45% prevalence of total occlusions at 2 weeks and 1 month, respectively) suggests a trend of decreasing frequency with time. This indicates that recanalization is probably an ongoing process in the early stages of myocardial infarction. When catheterization is performed at a longer interval, 1 year or more, prevalence of complete occlusion has been reported to be higher (79% in the series of Fuster et al.). This finding can be explained on the basis of selection criteria. The prevalence of normal coronary arteries found in our study was low (3%), which indicates that complete recanalization is uncommon in unselected patients. The patients with normal coronary arteries were younger than those with obstructive lesions. Five of 11 patients younger than age 35 years and the four patients age 30 years or younger had normal coronary angiograms.

The finding of recanalization suggests that platelet aggregation or transient thrombus formation may play a role in the genesis of infarction. Coronary artery spasm may be responsible for transient occlusion in an occasional patient with acute infarction. The relative frequency of each of the above mechanisms remains to be elucidated. Why resolution of total occlusion occurs is uncertain, though its decreasing frequency with time suggests that release of spasm or lysis of the thrombus may occur even several days after the onset of symptoms. Comparison of our data with those of De Wood et al. (table 2) indicates...

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**Table 2. Prospective Studies Including Coronary Angiography Early After Infarction**

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>% entered into study</th>
<th>Time of angiography</th>
<th>Number of diseased vessels</th>
<th>Incidence of left main stenosis</th>
<th>Prevalence of total occlusion</th>
</tr>
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<td></td>
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<tr>
<td>Taylor et al.⁷</td>
<td>280</td>
<td>39%</td>
<td>12 days</td>
<td>0% 27% 20% 53%</td>
<td>11%</td>
<td></td>
</tr>
<tr>
<td>Turner et al.⁶</td>
<td>196</td>
<td>70%</td>
<td>1 month</td>
<td>2% 25% 32% 30%</td>
<td>11%</td>
<td></td>
</tr>
<tr>
<td>Bertrand et al.⁸</td>
<td>116</td>
<td>?</td>
<td>15 days</td>
<td>1% 23% 43% 32%</td>
<td>?</td>
<td>87% 65%</td>
</tr>
<tr>
<td>De Wood et al.¹⁰</td>
<td>1210</td>
<td>26.6%</td>
<td>4-24 hours</td>
<td>0% - - -</td>
<td>?</td>
<td>87% 65%</td>
</tr>
<tr>
<td>Present series</td>
<td>259</td>
<td>91%</td>
<td>1 month</td>
<td>7% 34% 33% 26%</td>
<td>1.1%</td>
<td></td>
</tr>
</tbody>
</table>
that in a substantial number of patients, recanalization occurs after the first 24 hours. In experimental animals, properly timed reperfusion has been shown to limit the extent of necrosis.\textsuperscript{29-30} Reperfusion of ischemic regions can be accomplished in man by recanalization of the occluded artery. Factors determining the effectiveness of this mechanism in decreasing infarct size are the timing of revascularization and the amount of blood delivered to the ischemic region. While our data show a remaining stenosis of less than 90\% to be effective, further investigation is required to determine whether wall motion improvement is exclusive of early recanalization or whether it also results from late reopening of the artery. Experimental and clinical data seem to indicate that reperfusion of the myocardium might not be beneficial unless accomplished early after the onset of ischemia.\textsuperscript{29, 27-30} However, animal studies and bypass grafting in acute myocardial infarction probably represent a different situation from spontaneous reopening of an occluded artery. In the latter situation, the possibility exists that increasing flow in the border zones would limit the extension of necrosis.\textsuperscript{31}

An explanation for reperfusion of ischemic regions is the development of collateral channels. The clinical role of the collateral circulation has been extensively investigated for many years.\textsuperscript{32-40} Conflicting results have been reported concerning its influence on wall motion. Some investigators have found a definitive beneficial effect,\textsuperscript{31-42} but others have not.\textsuperscript{44-46} One month after infarction, only 11\% of our patients had well-developed collaterals. However, a definite improvement in both regional wall motion and ejection fraction was documented, and the degree of amelioration was similar to that attained after recanalization. Our data suggest that 32\% of patients will increase blood flow to the ischemic area (11\% through collaterals and 21\% by reopening of the occluded artery), which may account for a reduction in infarct size. Documentation of the beneficial effects of spontaneous reperfusion to the infarct area would be particularly relevant because new techniques of intracoronary fibrinolytic therapy are being developed.\textsuperscript{47-50} Results from the present study suggest that for streptokinase therapy to be effective in reducing infarct size, the remaining stenosis in the occluded vessel should be less than 90\%.

We conclude that complete coronary artery occlusion 1 month after infarction is confined to less than half of survivors; distribution of one-, two- and three-vessel coronary artery disease is uniform; prevalence of normal coronary arteries is low (3\%), as is that of left main coronary artery lesions (1.1\%); and reperfusion by reopening of the occluded artery or by collateral circulation improves wall motion in 32\% of patients.

References

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