Residual Flow to the Infarct Zone as a Determinant of Infarct Size
After Direct Angioplasty

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Background. In acute myocardial infarction, residual flow to the infarct zone either through antegrade flow in the infarct-related coronary artery or collateral flow from the non–infarct-related arteries is often present before reperfusion therapy. The purpose of this study was to assess the influence of antegrade flow in the infarct-related artery and/or collateral flow to the infarct zone before successful direct angioplasty on infarct size and myocardial salvage in patients with acute evolving myocardial infarction.

Methods and Results. Sixty patients with acute evolving myocardial infarction underwent direct successful angioplasty without prior thrombolytic therapy. The myocardium at risk of infarction, the final infarct size, and myocardial salvage were measured by tomographic perfusion imaging with 99mTc sestamibi. Antegrade flow in the infarct-related artery before intervention was graded according to the Thrombolysis in Myocardial Infarction (TIMI) study group classification. Collateral flow to the infarct zone before angioplasty was also graded (0 through 3, 0 being no collateral flow). The presence of even minimal antegrade flow before angioplasty (TIMI grade 1) in the infarct-related artery compared with absent flow was associated with a significant reduction in final infarct size (9 ± 17% versus 23 ± 19% of left ventricle, P = .02) and a significant increase in myocardial salvage (23 ± 16% versus 14 ± 13% of left ventricle, P = .05) after angioplasty. When antegrade flow in the infarct-related artery was absent before angioplasty, the presence of collateral flow before angioplasty resulted in a significantly smaller final infarct size (P = .01) and more myocardial salvage (P = .05) after angioplasty. Both antegrade infarct-related artery flow and collateral flow to the infarct zone had significant independent ability to predict infarct size after angioplasty. When collateral grade and TIMI grade were added to provide an estimate of residual flow, a model including residual flow, myocardium at risk, and the interaction of residual flow and infarct site explained 83% of the variability in infarct size after angioplasty.

Conclusions. The presence of antegrade flow in the infarct-related artery and/or collateral flow to the infarct zone before direct angioplasty in acute evolving infarction results in a smaller infarct size after direct successful angioplasty. (Circulation. 1993;88[part 1]:1527-1533.)

KEY WORDS • blood flow • collateral circulation • angioplasty • myocardial infarction • sestamibi

The work of DeWood and colleagues emphasized the etiological importance of thrombosis in the development of acute myocardial infarction and encouraged the development of thrombolysis to limit infarct size and reduce mortality after myocardial infarction. Several large studies confirmed that thrombosis did reduce mortality in acute infarction. Furthermore, perfusion imaging has shown that infarct size was limited after thrombolysis and primary angioplasty in acute infarction in the clinical setting. Additional clinical studies have shown that myocardium at risk of infarction, delay from onset of myocardial infarction, and coronary collateral flow were important determinants of final infarct size after reperfusion. The amount of variability in final infarct size explained by these three variables was less in the clinical than in the experimental model of reperfusion, suggesting that other variables in clinical infarction contribute to final infarct size after reperfusion. One of these additional variables may be the presence of antegrade flow in the infarct-related artery before an intervention. Angiographic studies have indicated that the infarct-related artery was not totally occluded in up to one third of patients with an acute infarction before any intervention such as thrombolysis or angioplasty. The present study was designed to test the hypothesis that the presence of a patent but stenosed infarct-related coronary artery and/or collaterals to the infarct zone from the non–infarct-related arteries results in a smaller final infarct size in patients who receive successful primary angioplasty for revascularization in acute evolving myocardial infarction.

Methods

Patients

Between March 18, 1988, and April 21, 1991, 73 patients treated with direct angioplasty for acute myo-
Cardiac infarction underwent sequential \(^{99m}\text{Tc}\) sestamibi perfusion imaging to quantify myocardium at risk and final infarct size. Of these patients, 41\(^{\circ}\) received direct angioplasty rather than thrombolytic therapy because it was felt to be clinically appropriate by the patient’s attending physician, and the remaining patients (59\%) received direct angioplasty as part of a randomized trial comparing the efficacy of direct angioplasty and thrombolysis. No patients received thrombolyis before angioplasty. The patients had given informed consent and agreed to participate in studies approved by the Institutional Review Board of the Mayo Clinic. All patients had chest discomfort for \(\geq 30\) minutes and ST elevation of \(\geq 0.1\) mV in two contiguous leads of the standard 12-lead ECG.

The infarct location was classified as anterior if ST elevation criteria occurred in leads V\(_2\) through V\(_6\) and inferior if criteria were present in leads II, III, and aVF. If the infarct location was equivocal by ECG, the infarct-related artery defined infarct location. The infarct location was anterior if the left anterior descending coronary artery was involved and inferior if the right or circumflex coronary artery was the infarct-related artery. Thirteen patients were subsequently excluded: 3 because angioplasty was unsuccessful or not performed, 3 because of a past history of myocardial infarction, 6 because reocclusion of the infarct-related artery occurred before hospital dismissal, and 1 because the preangioplasty infarct-related artery flow could not be graded. Thus, 60 patients who underwent successful angioplasty formed the study group. Of these 60 patients, 50 were included in a previous study from this laboratory\(^5\) that analyzed the role of collateral flow to the infarct zone as a determinant of infarct size when infarct-related artery flow was either absent or markedly limited (Thrombolysis in Myocardial Infarction [TIMI] grade 0 or 1).

\(^{99m}\text{Tc}\) Sestamibi Imaging

Two \(^{99m}\text{Tc}\) sestamibi tomographic perfusion studies were obtained in each patient. The first was early after the onset of infarction, and the second was just before hospital discharge. After informed consent was obtained, 30 mCi of \(^{99m}\text{Tc}\) sestamibi were injected intravenously, usually in the emergency room. The patients then underwent coronary angiography and balloon angioplasty. Once the patient’s clinical condition was stable, he or she was transferred to the nuclear cardiology department for imaging. These early images reflect myocardial blood flow at the time of the injection (before angioplasty); the acute perfusion defect was a measure of the myocardium at risk of infarction. The perfusion defect on the discharge study measured the extent of the completed infarction. The difference between these two studies is a measure of myocardial salvage, also expressed as a proportion of the myocardium at risk of infarction (salvage index). Animal studies\(^8\) of reperfusion have demonstrated that anatomic area at risk for infarction measured by postmortem angiography correlated well \((r=0.94)\) with the perfusion defect measured after injection of \(^{99m}\text{Tc}\) sestamibi during the coronary occlusion. In models of permanent occlusion, infarct size measured by the size of perfusion defect after \(^{99m}\text{Tc}\) sestamibi injection was similar to the size of completed infarct estimated by vital staining of the myocardium.\(^9\)

Myocardial perfusion defects were detected and quantified by a previously described method.\(^10-12\) Tomographic imaging of the heart was performed with a gamma camera (Elscint, Haifa, Israel) with a low-energy all-purpose collimator. Images (40-second acquisition) were obtained at 6\(^{\circ}\) increments commencing at 45\(^{\circ}\) in the right anterior oblique projection and proceeding through 180\(^{\circ}\). The images were reconstructed with back projection and a Ramp-Hanning filter into a series of short-axis images (6 mm in width) from apex to base of the left ventricle at right angles to the long axis of the left ventricle. Five representative short-axis slices were chosen: one at the apex, one at the base, a middle slice halfway between the apex and base, a fourth slice halfway between the apex and middle slice, and a fifth slice halfway between the base and the middle slice. At 6\(^{\circ}\) intervals, from all of the five slices, the pixel with the maximal activity was identified and considered to show 100\% activity, and each pixel in a slice was given a value relative to this pixel. Pixels in a slice that showed activity \(\leq 60\%\) of the maximal pixel were considered to show severely reduced perfusion. A circumferential count profile was generated for each of the five short-axis slices, and by standard geometric formulas, the volume of myocardium that showed severely reduced perfusion was calculated from the five slices and expressed as a percentage of the total left ventricular myocardium.

Coronary Arteriography and Angioplasty

Each patient received a bolus of 5000 U heparin in the emergency room before arteriography. Based on the initial ECG, a judgment was made as to which coronary artery was likely to be the infarct-related artery, and the contralateral coronary artery was visualized in several views by manual contrast injections (8 to 10 mL). The infarct-related coronary artery was then visualized in a similar manner. Angioplasty of the infarct-related artery was then performed with repeated balloon inflations until judged successful by the operating angiographer. The infarct-related artery was ultimately determined as the artery that showed features of thrombotic occlusion and was concordant with the distribution of acute injury on the initial ECG.

From the initial contrast injection of the infarct-related coronary artery, antegrade flow was described according to the TIMI study group classification,\(^7\) grade 0 being no antegrade flow beyond the point of occlusion, grade 1 being penetration of contrast material beyond the obstruction with failure to entirely opacify the distal coronary bed, grade 2 being passage of contrast beyond the obstruction into the distal coronary bed but with slow filling of the vessel or reduced clearance of the distal bed compared with regions not perfused by the infarct-related artery, and grade 3 being passage of contrast beyond the obstruction with prompt filling of the vessel and normal rapid clearance from the distal bed. Collateral flow to the infarct-related artery was classified according to the initial arteriogram before angioplasty by the method of Rentrop and coworkers,\(^13\) in which 0 indicated no filling of any collateral channels leading to the infarct-related artery, 1 indicated filling of branches without contrast reaching the epicardial
segment of the infarct-related artery, 2 indicated filling of branches and a portion of the epicardial artery, and 3 indicated filling of branches and the entire epicardial vessel. Residual flow grade was defined as the simple sum of TIMI grade and collateral grade.

The degree of luminal narrowing of the infarct-related artery at the site of the thrombotic occlusion was also estimated by the angiographer before and after angioplasty from multiple views of the infarct-related artery and expressed as a percent diameter narrowing compared with the adjacent uninvolved coronary artery. Angioplasty was performed if the infarct-related artery luminal diameter narrowing was ≥70%. Successful angioplasty was defined as an increase of ≥30% luminal diameter narrowing after angioplasty.

**Statistical Analysis**

Group data are presented as mean±SD, and comparison between group means was performed by one-way ANOVA or the unpaired Student’s t test where appropriate.

To assess the independent ability of TIMI and collateral grade before angioplasty to predict infarct size after angioplasty, ANCOVA was performed with infarct size as the dependent variable and myocardium at risk, TIMI grade, collateral grade, delay to angioplasty, and residual infarct-related artery stenosis after angioplasty as independent variables. Because previously the interaction between collateral grade before angioplasty and infarct size had been shown to have significant independent ability to predict final infarct size after angioplasty, this term was also included as a variable in the model.

In addition, a further ANCOVA was performed so that the independent ability of residual flow grade before angioplasty to predict final infarct size after angioplasty was assessed. In this analysis, infarct size was the dependent variable, and myocardium at risk, residual flow grade, delay to angioplasty, residual infarct-related artery stenosis after angioplasty, and the interaction of residual flow grade and infarct site were the independent variables.

**Results**

Table 1 indicates the relevant demographic data on the 60 study patients. Roughly equal numbers of patients had either anterior or inferior infarction. 99mTc sestamibi was injected on average just <4 hours after the onset of symptoms of infarction in the study group, and angioplasty was performed approximately 100 minutes later. Myocardium at risk averaged just over one third of the left ventricle, with an ultimate mean salvage of more than one half of the myocardium at risk. Patients were divided into two groups (Table 2) depending on whether antegrade flow in the infarct-related artery was absent (TIMI grade 0, 37 patients) or present (TIMI grade ≥1, 23 patients) before angioplasty. Patients with anterior infarction were more likely than inferior to have a totally occluded infarct-related artery, but this trend was not significantly different. In the 23 patients with antegrade flow in the infarct-related artery, 14, 7, and 2 patients had TIMI grade flow of 1, 2, and 3, respectively. The extent of collateral flow to the infarct-related artery did not differ between patients with and without antegrade flow in the infarct-related artery (Table 2). Patients with absent infarct-related flow tended to be injected later (P=.06) with 99mTc sestamibi, and the intervals from onset of symptoms to arteriography and angioplasty were significantly longer (P<.05) than in those patients with flow present in the infarct-related artery.

Table 3 indicates myocardium at risk before angioplasty, final infarct size, myocardial salvage, and salvage index after angioplasty in patients with TIMI grade 0, 1, and ≥2. Since only two patients had TIMI grade 3 flow, patients with TIMI grade 2 and 3 flow were combined for purposes of analysis. The figure presents graphically the comparisons between TIMI grade and final infarct size, myocardial salvage, and salvage index. The amount of myocardium at risk was not significantly different between TIMI grades. However, the final infarct size was significantly less (P=.002) and the salvage index was significantly greater (P=.001) as TIMI grade increased. Importantly, the benefit of antegrade infarct-related artery flow before angioplasty on outcome after angioplasty for final infarct size and salvage index was observed even in those patients with TIMI 1 antegrade flow.

Before angioplasty, collateral flow was present (grade ≥1) in 40 (66%) of the patients and absent (grade 0) in 20 patients. Collateral flow before angioplasty influenced outcome after angioplasty. In the 37 patients with no antegrade infarct-related artery flow (TIMI grade 0) before angioplasty, final infarct size was significantly smaller (P=.01, ANOVA) and myocardial salvage was significantly greater (P=.02) after angioplasty for a higher collateral grade before angioplasty; after angioplasty, final infarct size was 29±14%, 34±16%, 14±12%, and 10±8% and myocar-
TABLE 2. Demographic Data of Patients in Whom Infarct-Related Artery Flow Was Absent or Present

<table>
<thead>
<tr>
<th>Infarct-Related Artery Flow</th>
<th>Absent</th>
<th>Present</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (% of all patients)</td>
<td>37 (62)</td>
<td>23 (38)</td>
</tr>
<tr>
<td>Anterior infarction, n (%)</td>
<td>22 (37)</td>
<td>10 (17)</td>
</tr>
<tr>
<td>Inferior infarction, n (%)</td>
<td>15 (25)</td>
<td>13 (22)</td>
</tr>
<tr>
<td>TIMI grade, n (%)</td>
<td>37 (62)</td>
<td>. . .</td>
</tr>
<tr>
<td>. . .</td>
<td>14 (23)</td>
<td></td>
</tr>
<tr>
<td>. . .</td>
<td>7 (12)</td>
<td></td>
</tr>
<tr>
<td>. . .</td>
<td>2 (3)</td>
<td></td>
</tr>
<tr>
<td>Collateral grade, n (%)</td>
<td>11 (18)</td>
<td>9 (15)</td>
</tr>
<tr>
<td>1</td>
<td>9 (15)</td>
<td>3 (5)</td>
</tr>
<tr>
<td>2</td>
<td>9 (15)</td>
<td>9 (15)</td>
</tr>
<tr>
<td>3</td>
<td>8 (13)</td>
<td>2 (3)</td>
</tr>
<tr>
<td>Onset to $^{99m}$Tc sestamibi injection, min</td>
<td>265±228</td>
<td>168±119*</td>
</tr>
<tr>
<td>Onset to coronary arteriogram, min</td>
<td>343±271</td>
<td>212±126†</td>
</tr>
<tr>
<td>Onset to angioplasty, min</td>
<td>377±274</td>
<td>247±128†</td>
</tr>
</tbody>
</table>

*P=.06, †P<.05 absent vs present infarct-related artery flow.

dial salvage was 11±7%, 9±10%, 24±14%, and 15±15% with collateral grade before angioplasty of 0, 1, 2, and 3, respectively.

Table 4A indicates a multivariate model to predict infarct size using ANCOVA. In this model, in decreasing order of significance, myocardium at risk, collateral grade, the interaction between collateral grade and infarct site, and TIMI grade show significant independent ability to predict infarct size after angioplasty; delay to angioplasty and residual stenosis after angioplasty were not independently significant. This model accounted for 77% of the variability in infarct size.

TABLE 3. Myocardium at Risk Before Angioplasty and Final Infarct Size and Myocardial Salvage After Angioplasty in Patients With TIMI Grades 0, 1, and ≥2 Antegrade Flow in the Infarct-Related Artery Before Angioplasty

<table>
<thead>
<tr>
<th>TIMI Grade</th>
<th>N</th>
<th>Myocardium at Risk*</th>
<th>Final Infarct*</th>
<th>Myocardial Salvage*</th>
<th>Salvage Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>37</td>
<td>37±20</td>
<td>23±19</td>
<td>14±13</td>
<td>0.45±0.36</td>
</tr>
<tr>
<td>1</td>
<td>14</td>
<td>32±18</td>
<td>9±17†</td>
<td>23±16†</td>
<td>0.76±0.33§</td>
</tr>
<tr>
<td>≥2</td>
<td>9</td>
<td>19±19</td>
<td>2±4</td>
<td>17±19</td>
<td>0.86±0.3</td>
</tr>
<tr>
<td>P</td>
<td>. . .</td>
<td>.06†</td>
<td>.02</td>
<td>.18</td>
<td>.001</td>
</tr>
</tbody>
</table>

*Percent of left ventricle.
†P=.02, †P=.05, †P=.006 by unpaired t test, TIMI grade 0 vs TIMI grade 1. §Means compared by one-factor ANOVA.

Table 4B indicates a multivariate model to predict infarct size that incorporates residual flow grade (TIMI grade plus collateral grade). In this model, myocardium at risk, residual flow grade, and the interaction of residual flow grade and infarct site showed significant independent ability to predict infarct size after angioplasty; delay to angioplasty and residual stenosis after angioplasty did not. This model explained 83% of the variability in infarct size.

Nine patients were excluded from analysis who could have been included on an "intention-to-treat" basis. These patients were excluded because angioplasty was unsuccessful or not performed or because reocclusion occurred. When these nine patients were included with the 60 study patients, infarct size and salvage index after angioplasty continued to be significantly related to TIMI flow before angioplasty ($P=.02$ and $P=.04$, respectively). The multivariate models (Table 4A and 4B) did not change significantly.

Discussion

A major finding of this study was that the presence of antegrade infarct-related artery flow before successful balloon angioplasty was associated with a lesser degree of infarction and enhanced salvage of jeopardized myocardium compared with absence of flow in the infarct-related artery. Patients with even minimal antegrade flow (TIMI grade 1) before angioplasty demonstrated a considerably smaller final infarct size after angioplasty compared with patients without any antegrade flow in the infarct-related artery before angioplasty. This study confirmed an earlier finding from this laboratory that patients without antegrade infarct-related artery flow before angioplasty had a smaller final infarct size and more myocardial salvage after angioplasty if collateral flow to the infarct zone was present before angioplasty. An additional specific finding of this study was that both antegrade infarct-related artery flow and collateral flow to the infarct zone before angioplasty were significant independent determinants of final infarct size and myocardial salvage after successful angioplasty.

Although the statistical analysis suggests that the beneficial effects of antegrade and collateral flow are independent, antegrade flow and collateral flow may not be physiologically independent of each other, since antegrade flow in the infarct-related artery may minimize collateral visualization with angiography. However, the collateral flow profile was similar in patients with and without antegrade infarct-related artery flow.

Reimer and coworkers identified angiographic area at risk, collateral flow to the infarct zone, and myocardial metabolic demand as independent determinants of infarct size after reperfusion in an animal model controlled for infarct location and duration of occlusion. Recently, Christian et al measured myocardium at risk and final infarct size in clinical myocardial infarction with the $^{99m}$Tc sestamibi technique and found that final infarct size after reperfusion was determined in large degree by the extent of myocardium at risk, angiographic collateral grade, an interaction term between collaterals and infarct location, and time to reperfusion. In this present study, a model using myocardium at risk, TIMI grade and collateral grade before angioplasty, and the interaction of collateral grade and infarct site explained 77% of the variability in infarct size after...
angioplasty. Furthermore, when residual flow to the infarct zone before angioplasty was defined as the sum of collateral and TIMI grade, residual flow, in combination with myocardium at risk and an interaction term with infarct size, generated a very powerful model, which explained 83% of the variability in infarct size after angioplasty. This was similar to the ability of myocardium at risk, collateral flow, and rate-pressure product to predict infarct size after reperfusion in the animal model.6

The delay to angioplasty in the present study was long, a mean of >5 hours. The major portion of delay to angioplasty occurred before the 99mTc sestamibi injection (just <4 hours). Since patients were entered into this study, other studies14 have shown that considerable delays exist and that these delays can be minimized with patient and physician education. There were significant differences in the delay to angioplasty between the patients with infarct-related artery flow and those without, although multivariate analysis suggested that these differences in delay to angioplasty did not account for the differences in outcome.

The clinical model of evolving myocardial infarction is very different from the animal model. In clinical myocardial infarction, the precise time of onset of myocardial infarction may be uncertain, and often, symptoms suggest a stuttering onset. In the animal model, onset is abrupt and determined by the time of mechanical occlusion of the coronary artery. In addition, angiographic studies in acute myocardial infarction in patients show that even in the early hours after infarction, one third of infarct-related coronary arteries demonstrate antegrade flow despite subsequent evidence of the myocardial necrosis.17 In the present study, 40% of patients had at least minimal antegrade infarct-related artery flow. Studies with animal models of infarction have not assessed the possibility that residual infarct-related artery flow may be another variable determining final infarct size, independent of myocardium at risk, collaterals, and time to reperfusion.

A number of factors may have contributed to the presence of persistent antegrade flow in the infarct-related artery in patients at the time of arteriography. Patency of the infarct-related artery may have been present throughout or be a consequence of spontaneous reperfusion of a previously occluded vessel. The process of coronary occlusion in acute myocardial infarction in humans may well be dynamic and involve several processes, including clot lysis, rethrombosis, coronary spasm, and coronary vasodilatation.15 All patients received heparin intravenously soon after diagnosis, but it is unclear whether heparin by itself could have restored patency of the infarct-related artery. Injection of radiographic contrast into infarct-related coronary arteries may have caused disruption of an occluding thrombus in a certain proportion of patients. Leiboff and coworkers16 found that, over a 90-minute period of angiographic observation, spontaneous reperfusion occurred in 17% of patients.

Whatever the mechanism for preserved antegrade infarct-related artery flow, its presence, in addition to collateral flow, may maintain viability of myocardium at risk and may lead to enhanced survival of jeopardized myocardium after angioplasty or thrombolysis. These data confirm the findings of Rogers et al.,17 who showed that global ejection fraction and ejection fraction of the infarct zone were improved after successful revascularization only in those patients with either visible collaterals or limited flow in the infarct-related artery before revascularization.

Patients with unsuccessful angioplasty and clinical evidence of reocclusion were excluded from this study. However, inclusion of these patients on an “intention-to-treat” basis did not substantially alter the study.

**Table 4. Predictors of Infarct Size**

<table>
<thead>
<tr>
<th>A. With collateral and TIMI grade</th>
<th>F Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardium at risk</td>
<td>38.1</td>
<td>.0001</td>
</tr>
<tr>
<td>Collateral grade</td>
<td>5.47</td>
<td>.003</td>
</tr>
<tr>
<td>Collateral grade and infarct site</td>
<td>4.43</td>
<td>.004</td>
</tr>
<tr>
<td>TIMI grade</td>
<td>3.45</td>
<td>.04</td>
</tr>
<tr>
<td>Delay to angioplasty</td>
<td>1.86</td>
<td>.18</td>
</tr>
<tr>
<td>Residual stenosis after angioplasty</td>
<td>0.23</td>
<td>.63</td>
</tr>
<tr>
<td>R² of the model=0.77</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>B. With residual flow grade</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardium at risk</td>
<td>51.6</td>
<td>.0001</td>
</tr>
<tr>
<td>Residual flow grade</td>
<td>8.08</td>
<td>.0001</td>
</tr>
<tr>
<td>Residual flow grade and infarct site</td>
<td>7.54</td>
<td>.0001</td>
</tr>
<tr>
<td>Delay to angioplasty</td>
<td>2.22</td>
<td>.14</td>
</tr>
<tr>
<td>Residual stenosis after angioplasty</td>
<td>0.85</td>
<td>.36</td>
</tr>
<tr>
<td>R² of the model=0.83</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*By ANCOVA.
findings. Since predischARGE angiography was not rou-
tine in these patients, it is not possible to determine how
many patients underwent silent reocclusion.

\[^{99m}Tc\] sestamibi perfusion tomographic imaging is a
well-established method\(^{9-11}\) of quantifying myocardium
at risk and final infarct size in acute infarction. Myocardial
extraction of \[^{99m}Tc\] sestamibi was found to be
rapid,\(^{18}\) and the uptake was proportional to blood flow
in myocardial ischemia\(^{19}\) with minimal washout from
infarcted myocardium\(^{20}\) even after reperfusion.\(^{21}\) How-
ever, certain technical limitations of the method exist,\(^{11}\)
related to the selection of the short-axis slices, the
assumption of uniform thickness of the left ventricle,
gamma ray scatter, and the poor definition of subendocar-
dial ischemia.

Sinusas and coworkers\(^8\) showed an excellent ability of
\[^{99m}Tc\] sestamibi to measure myocardial at risk of infar-
cation in dogs. In the clinical setting,\(^{12}\) \[^{99m}Tc\] sestamibi
perfusion imaging and angiographic estimates of area at
risk yielded a good correlation \(r=0.89\), and a study
using radioactive microspheres\(^{22}\) demonstrated esti-
mates of myocardium at risk in inferior and anterior
infarction similar to the \[^{99m}Tc\] sestamibi method.\(^{5}\) The
\[^{99m}Tc\] sestamibi perfusion defect at hospital dismissal
was felt to be a reasonable estimate of final infarct size,
since this defect correlated with left ventricular ejection
fraction at discharge and 1 year later\(^{24-25}\) and with
kinetic enzyme release.\(^{26}\)

This is a descriptive study. Without having a control
group of patients with preserved infarct-related artery
flow who did not undergo angioplasty, it is not possible
to indicate the relative benefits of patent infarct-
related artery alone compared with angioplasty alone.
A control group was not available in this study, since
the clinicians involved with these patients decided to
perform angioplasty if \(\geq 70\%\) luminal diameter nar-
rowing was present in the infarct-related artery. In the
absence of angioplasty, several studies\(^{16,27}\) have
suggested that spontaneous infarct-related artery flow
protects myocardium. However, since the degree of
stenosis in those arteries with residual flow was con-
siderable and tight stenoses are more likely to oc-
clude,\(^{28}\) some patients probably benefited from angi-
oplasty. It is probably unlikely that such a control group
would become available, since most patients undergo
coronary arteriography in the early hours of myocardial
infarction because of ongoing myocardial isch-
emia, and coronary angioplasty is performed for ther-
apetic reasons.

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occlusion during the early hours of transmural myocardial
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