Value of Early Thallium-201 Scintigraphy for Predicting Mortality in Patients With Acute Myocardial Infarction


SUMMARY To determine whether the severity of thallium-201 scintigraphic defects present within hours of acute myocardial infarction (MI) could be used to predict subsequent mortality, thallium-201 imaging was performed within 15 hours of the onset of symptoms in 42 patients with acute MI. Patients with pulmonary edema or shock were excluded. The extent of perfusion defect was determined in three views (anterior and 40° and 60° left anterior oblique) by both objective computer-assisted and subjective methods, and expressed as a summed defect score. Mortality for the patient group as a whole was 17% in hospital, 24% at 6 months, and 33% at last follow-up (average 9 months). Using the objective method, a high thallium defect score (7.0 or greater, corresponding to at least a moderate reduction of activity involving 40% of the left ventricle in two views) identified a subgroup of 13 patients in which mortality was 46% in hospital, 62% at 6 months, and 92% at last follow-up. Corresponding values for the 29 patients with lower objective defect scores were 3%, 7% and 7%, respectively (all p < 0.001). Similar results were obtained with the subjective scoring method. Certain clinical variables, including a history of prior myocardial infarction, anterior location of the current infarct, peak CK > 1000 IU/l and moderate (vs none or mild) left ventricular failure were also associated with mortality. However, a high thallium defect score was significantly more predictive than any of these variables. Stepwise, multivariate analysis showed that the thallium score alone was a better predictor than the best combination of these clinical variables, and no variable added to the predictiveness of the high defect score. These results suggest that thallium-201 scintigraphy may provide an accurate, rapid, noninvasive method for separating high-risk and low-risk subgroups of hemodynamically stable patients admitted with acute MI.

CONSIDERABLE EFFORT has been directed toward identifying interventions that might preserve myocardium and diminish morbidity and mortality after acute myocardial infarction (MI).1-3 Because of the marked variations in infarct size, extent of underlying coronary artery disease and degree of left ventricular (LV) dysfunction among patients with acute MI,4-5 evaluation of potential life-saving therapeutic agents requires that patients be stratified before treatment according to their expected risk of death. Patients at low risk do well without special therapy, and very large numbers of patients are required to show that therapy improved survival. In contrast, patients at high risk may form a more suitable group for testing such treatment interventions, because smaller numbers are needed to demonstrate therapeutic efficacy. However, these patients must be identified early, before completion of necrosis and before the onset of hemodynamic deterioration, after which the results of aggressive therapy are disappointing.6

Clinical classifications based on the severity of LV failure7 have been widely used for early identification of high-risk patients. Patients with acute pulmonary edema or shock (Killip class III-IV) are considered to be at high risk (30-90% in-hospital mortality), while those with no or at most moderate failure (Killip class I-II) are considered to be at low risk, with an inhospital mortality of 10-20%.7-11 Because most patients fall into the Killip class I-II category, and the majority of deaths actually comes from the low-risk group, it would be useful to identify the high-risk subset of this overall low-risk patient group. The use of invasive hemodynamic measurements has improved the identification of high-risk patients in clinically stable subgroups.12 However, it is not clear that invasive hemodynamic indexes provide sufficient sensitivity or specificity for predictive purposes, and there is some associated morbidity.

Thallium-201 perfusion imaging performed within the first 24 hours of acute MI has been found useful for detecting, localizing and roughly sizing areas of infarction and surrounding ischemia.13-17 The observation by Wackers and associates18 that thallium-201 perfusion defects tend to diminish in size over time after acute MI, as well as our own finding that large thallium-201 defects may be associated with small infarcts at postmortem examination,17 supports the notion that areas of reduced thallium-201 uptake represent both ischemia and necrosis in the first few hours of infarction. The sum of old damage, new necrosis and ischemic, jeopardized myocardium should be an important determinant of short-term prognosis. The purpose of this study was to determine whether thallium-201 myocardial imaging within the first few hours in clinically stable patients with acute MI could be used to identify a high-risk subgroup.
Materials and Methods

Patients

The study population consisted of 42 consecutive patients with acute MI presenting within 12 hours of the onset of symptoms and judged to be Killip class I or II on admission. The diagnosis of acute MI was based on a typical history of chest pain, characteristic electrocardiographic changes and a rise in serum creatine kinase (CK). Thallium-201 myocardial perfusion scintigraphy was performed in the coronary care unit within 15 hours of the onset of infarction (range 3–15 hours; mean ± sd 8 ± 4 hours). Imaging was performed 10 minutes after intravenous injection of 1.5–2.0 mCi of thallium-201 in the anterior and 40° and 60° left anterior oblique views using an Ohio Nuclear Series 420 mobile scintillation camera with a low-energy, all-purpose, parallel-hole collimator. Images contained 400,000 counts full field and were acquired on magnetic disk in a 128 × 128 matrix (Medical Data Systems Simultaneity System).

Most patients received intravenous heparin to maintain a clotting time of 20–30 minutes and continuous lidocaine infusion at an initial dose of 20 μg/kg/min, adjusted as necessary to suppress ventricular ectopic activity. By random selection, 19 of the 42 patients received nitroglycerin after perfusion scintigraphy was completed as part of an ongoing clinical trial.

The location of the acute MI was determined electrocardiographically. Anterior location was defined by primary ST-segment or T-wave changes or by Q-wave development in leads I, aV_L, or V_1 to V_6. Inferior location was defined by primary changes in leads 2, 3 or aV_R or by abnormalities suggesting “true posterior” wall involvement. Transmural extent of acute infarction was defined by evolution of abnormal Q waves with a duration ≥0.04 second and a depth ≥ 25% of the R wave in the same lead. Nontransmural infarction meant that ST-T changes were observed without Q-wave development. All patients had measurements of total serum CK activity on admission and every 4 hours thereafter using the Rosalki technique.18

Patients were classified (Killip criteria) according to the severity of clinical LV failure on admission.1 Two of the authors reviewed the recorded physical examination and the admission chest radiographs. Class I was defined as the absence of any signs of LV failure. Class IIA included patients with basilar rales or ventricular gallop on auscultation or an increase in upper lobe vascularity or interstitial edema on chest x-ray. Class IIB consisted of patients with alveolar infiltrates due to pulmonary edema, but without the clinical syndrome of acute pulmonary edema.

Analysis of Thallium-201 Scintigrams

Thallium scintigrams were scored objectively by a technologist using a computer-assisted technique called “circumferential profiles” (fig. 1).19 Each view was independently analyzed after a single, nine-point, weighted smoothing without background subtraction. An ellipse was used to isolate the left ventricle from the rest of the image, and a computer-generated circumference was constructed around the outer edge of the left ventricle using an isocount criterion (usually about 50% of average activity in the ellipse) to provide a visual “best fit” to the outer edge of the left ventricle. Radii were then constructed by the computer from the image center (automatically determined) to each point on the circumference (usually 75–125 radii). The average activity per pixel was calculated along each radius and normalized to the highest value. A curve was displayed of normalized thallium activity vs angular location, starting from the radius oriented upward and proceeding counterclockwise. An objective “defect score” was then determined by the computer by comparing the patient’s profile curve with normal limits obtained by averaging the curves of 13 normal volunteers. The patient curve and normal curves were aligned about the radius corresponding to the apex. The lower limit of normal was defined as 2 standard deviations below the normal mean curve. An objective defect score was found for each view by integrating the area of the patient’s curve below normal (percentage of radii with reduced activity multiplied by the average reduction in activity for these radii). In figure 1, 54%
of the radii had activity reduced more than 2 standard deviations below normal. The score for this view was calculated by multiplying 0.54 by 0.19 by 100, resulting in a score of 10.26 (a very high score). The objective score for each view can also be considered to represent the total amount of reduction in activity below normal divided by the total number of radii, or the average reduction in activity per radius. This method of computer scoring has high intra- and interobserver reproducibility.\textsuperscript{19} Objective defect scores were summed for the three views to obtain a total defect score for each patient.

Perfusion scintigrams were also interpreted subjectively by two of the authors for comparison with the computer-assisted method. The left ventricle in each image was divided into five segments, the apical segments being somewhat smaller than the others (fig. 2). Each segment was scored for intensity of defect based on the worst area within the segment (0 = normal, 1 = slightly but definitely abnormal, 2 = moderately abnormal, and 3 = severely abnormal). After consensus agreement an image score was obtained by summing the individual segment scores, and a total subjective score was obtained for each patient by adding the scores for these views. In figure 1, the lower septal and apical segments each received a score of 2, and the distal lateral wall segment received a score of 3, resulting in a subjective score of 7 for this view. The other two views in this patient received scores of 9 and 6, for a total score of 22. Reproducibility of the consensus score was good. Ten patients with scores ranging from 0–26 (mean 6.7) were analyzed on two occasions; the mean difference in absolute score was 1.0 ± 0.9 (SD). Both objective and subjective analyses were done without knowledge of patient identity or outcome.

**Follow-up and Analysis of Data**

Thirty-five of 42 patients survived the initial hospitalization. They were followed 6–20 months (mean 9 months) and were cared for by their private physicians, with none of the authors participating. Kaplan-Meier survival curves were used to describe survival.\textsuperscript{20} Differences in survival curves between patients with high and low thallium defect scores were evaluated by the Peto method\textsuperscript{21} at three intervals: at the end of the hospitalization, 6 months after infarction, and at last follow-up. This method was also used to confirm that survival-curve differences were not due to other prognostic variables by the adjustment for

**FIGURE 2.** For subjective scoring each scintigraphic view was divided into five segments as shown. ANT = anterior; LAO = left anterior oblique.
each in turn. Relative predictiveness of mortality by single prognostic variables and by groups of variables was evaluated at each time interval. Focusing on end-point mortality as opposed to a time-dependent process indicated use of the technique of discriminant function analysis. Thallium defect score, age and peak CK were analyzed as both continuous and discrete variables. Only the latter analysis is reported here because the results were more precise.

## Results

### Clinical Outcome

Fourteen of the 42 patients died, seven during hospitalization and seven after discharge 1.5-18 months after initial presentation. Although post-mortem examinations were performed on only five patients, all of the deaths appeared to be cardiac in origin based upon the clinical history. The clinical characteristics of these patients, along with the circumstances of death, are presented in Table 1. The mean age of the 14 nonsurvivors was 59.2 years and 50% were male. Five patients (36%) had a well-documented history of prior MI. Seven (50%) presented with an acute anterior transmural infarction, five (36%) had inferior transmural infarcts and two (14%) had nontransmural infarcts. Five patients (36%) were in clinical class IIB and 10 (72%) had peak serum CK greater than 1000 IU/l. In contrast, none of the 28 survivors had a history of prior infarction, only four (14%) had acute anterior transmural infarctions, two (7%) were in clinical class IIB on admission and 10 (36%) had peak CK greater than 1000 IU/l. The mean age (56.4 years) and male/female distribution (69% male) were similar to those of nonsurvivors.

### Thallium-201 Scintigrams and Mortality

The size of thallium perfusion defects was examined in survivors and nonsurvivors (fig. 3). Nonsurvivors had significantly larger defects and a mean objective defect score of 14.3 vs a mean defect score of 2.3 in survivors (p < 0.001). Only one of 28 patients alive at last follow-up had a total objective defect score of more than 7.0, while 12 of 14 patients who died had a score of 7.0 or more. An objective score of 7 generally corresponded to at least a moderate reduction of thallium uptake (2+ of 3+) involving 40% or more of the left ventricle in two or more views. A severe reduction of uptake involving 60% of the left ventricle in a single view at times resulted in a score of this magnitude.

On the basis of the data in figure 3, we defined thallium scintigraphic studies with a computer score of 7.0 or more as high risk and those with a computer score of less than 7.0 as low risk. Actuarial survival curves for patients with high-risk and low-risk scintigrams are shown in figure 4. The difference between the two curves was highly significant at 2 weeks (p < 0.001), 6 months (p < 0.001) and last follow-up (p < 0.001). Mortality for the overall patient population and for high-risk and low-risk subgroups is compared during hospitalization, at 6 months and at last follow-up in table 2.
than 12, while 11 of 14 patients who died had scores of 12 or more. A subjective defect score of 12 corresponded to a reduction in thallium uptake similar to that present with an objective score of 7.0. The difference in survival between patients with subjective defect scores greater than 12 and patients with subjective defect scores less than 12 was highly significant at all three time intervals studied ($p < 0.001$). There was no significant difference in the predictive value of the objective, computer-derived scoring system compared with the subjective scoring system. The two systems disagreed on only three patients; the computer-derived system correctly predicted the outcome in each case.

All patients were imaged less than 15 hours from the onset of symptoms. Because the size of a thallium perfusion defect may decrease spontaneously during the acute phase of infarction, we analyzed the results to determine if there was any relationship between the time of scintigraphy and defect size. No significant association was found between time of scintigraphy and the proportion of patients with high or low defect scores. Of 17 patients imaged within 6 hours of symptoms, seven (41%) had high defect scores. Of the 25 patients imaged between 6 and 15 hours, six (24%) had high defect scores ($p = NS$). We obtained 2- and 5-hour redistribution (delayed) images in 22 patients, and 19 had no or minimal change in the defect score. Two patients had a moderate increase in defect score and one a moderate decrease. No patient had a change in the thallium defect score classification from low-risk to high-risk or vice-versa.

Mortality in high-risk and low-risk thallium scintigram groups was adjusted for age, sex, peak CK, history of previous MI, anterior location of the acute infarct, clinical class IIB on admission and treatment with nitroglycerin. Analysis showed that after adjustment for each variable, mortality in the high-risk scintigraphic group was significantly higher than that in the low-risk group during hospitalization, at 6 months and at last follow-up. Therefore, the greater mortality in the high-risk thallium group was not due to partial confounding by any of the variables examined.

**Predictivity of Clinical Variables and Scintigraphy**

Statistically significant predictors of mortality during hospitalization, at 6 months and at last follow-up are shown in table 3. The ability of a single clinical variable to predict mortality was indexed by the

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**Figure 3.** Thallium-201 defect scores for survivors and nonsurvivors. The dashed line is drawn at a score of 7.0. All but one of the survivors had a score less than 7.0, while all but two of the nonsurvivors had a score greater than 7.0.

**Figure 4.** Actuarial survival curves for patients with high-risk and low-risk scintigrams.

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**Table 2. Mortality in Overall Population and High-risk and Low-risk* Subgroups**

<table>
<thead>
<tr>
<th></th>
<th>In hospital</th>
<th>6 months</th>
<th>Last follow-up</th>
</tr>
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<tbody>
<tr>
<td>Overall (n = 42)</td>
<td>17%</td>
<td>24%</td>
<td>33%</td>
</tr>
<tr>
<td>*High-risk (n = 13) (Score $\geq 7$)</td>
<td>46%</td>
<td>62%</td>
<td>92%</td>
</tr>
<tr>
<td>*Low-risk (n = 29) (Score $&lt; 7$)</td>
<td>3%</td>
<td>7%</td>
<td>7%</td>
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</table>

*Determined by computer-derived scoring system.
percentage of patients whose mortality was correctly classified by the analysis. History of previous MI, anterior location of the acute infarction, alveolar infiltrates on admission (class IIB), and peak serum CK greater than 1000 IU/l were all significantly predictive at 6 months and at last follow-up, with values ranging from 66.7–78.6%. A high-risk thallium scintigram was significantly better at predicting mortality at these two time intervals (83.3% and 92.9%, respectively, using objective scoring). Anterior location of the acute infarct was the only clinical variable predictive of mortality during the initial hospitalization (78.6%, p < 0.05). Again, a high-risk thallium scintigram was significantly more predictive (83.3% by the objective method). The predictive value of a high-risk thallium scan determined by subjective scoring varied from 81% during the initial hospitalization to 85.7% at last follow-up and was not significantly different from that by the objective method. Age and sex were not significantly predictive at any of the three time intervals.

Table 4 lists the sensitivity and specificity of high-risk thallium scintigrams, determined both subjectively and objectively, and the single clinical variables examined above for predicting mortality at the three different times. Although many of the clinical variables were highly specific, sensitivity was poor. Thallium scintigraphy appeared to provide the best combination of sensitivity and specificity for predicting mortality, with the objective scoring system slightly superior to the subjective scoring system.

Stepwise, multivariate, discriminant-function analysis was performed to determine whether thallium scintigraphy was a better predictor of mortality than the best combination of clinical variables that were available on admission. Age, sex, history of previous infarct, location of the acute infarct and clinical class at admission were used for this analysis. At all three time intervals, the best combination of clinical variables included a history of infarction and an anterior location of the present infarct, which correctly classified 73.8% for the initial hospitalization, 78.6% at 6 months and 88.1% at last follow-up. The addition of peak CK, which was not available on admission in most patients, did not increase the predictiveness for initial hospitalization or last follow-up, but slightly increased predictiveness at 6 months, from 78.6% to 81.0%. Comparison of these values with those in Table 3 indicates that thallium scintigraphy was more predictive than the best combination of clinical variables. Further, when the thallium defect score was added to this best combination in the multivariate analysis, predictiveness increased significantly.

**Table 3. Mortality: Significant Prognostic Variables**

<table>
<thead>
<tr>
<th></th>
<th>In hospital</th>
<th>6 months</th>
<th>Last follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thallium score: objective (83.3%)‡</td>
<td>Thallium score: objective (83.3%)‡</td>
<td>Thallium score: objective (92.9%)‡</td>
<td></td>
</tr>
<tr>
<td>Thallium score: subjective (81.0%)‡</td>
<td>Thallium score: subjective (81.0%)‡</td>
<td>Thallium score: subjective (85.7%)‡</td>
<td></td>
</tr>
<tr>
<td>Anterior location (78.6%)*</td>
<td>Anterior location (78.6%)†</td>
<td>Anterior location (78.6%)†</td>
<td></td>
</tr>
<tr>
<td>History previous MI (78.6%)*</td>
<td>History previous MI (78.6%)†</td>
<td>History previous MI (78.6%)†</td>
<td></td>
</tr>
<tr>
<td>Class IIB (78.6%)*</td>
<td>Class IIB (73.8%)*</td>
<td>Class IIB (73.8%)*</td>
<td></td>
</tr>
<tr>
<td>Peak CK &gt; 1000 (66.7%)*</td>
<td>Peak CK &gt; 1000 (66.7%)*</td>
<td>Peak CK &gt; 1000 (66.7%)*</td>
<td></td>
</tr>
<tr>
<td>Age &gt; 65 (78.6%)*</td>
<td>Age &gt; 65 (78.6%)*</td>
<td>Age &gt; 65 (78.6%)*</td>
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Numbers in parenthesis refer to "predictiveness" of each variable for mortality (see text for definition). Statistical significance of the predictiveness of each variable for mortality:

* p < 0.05.
† p < 0.01.
‡ p < 0.001.

**Table 4. Sensitivity and Specificity of Thallium-201 Scintigraphy and Clinical Variables for Predicting Mortality**

<table>
<thead>
<tr>
<th></th>
<th>In hospital</th>
<th>6 months</th>
<th>Last follow-up</th>
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<tbody>
<tr>
<td></td>
<td>Sensitivity</td>
<td>Specificity</td>
<td>Sensitivity</td>
</tr>
<tr>
<td>High-risk thallium-201 scintigraphy: objective</td>
<td>86%</td>
<td>80%</td>
<td>80%</td>
</tr>
<tr>
<td>High-risk thallium-201 scintigraphy: subjective</td>
<td>86%</td>
<td>80%</td>
<td>80%</td>
</tr>
<tr>
<td>History of previous MI</td>
<td>29%</td>
<td>91%</td>
<td>30%</td>
</tr>
<tr>
<td>Anterior transmural MI</td>
<td>57%</td>
<td>80%</td>
<td>60%</td>
</tr>
<tr>
<td>Class IIB on admission</td>
<td>29%</td>
<td>86%</td>
<td>40%</td>
</tr>
<tr>
<td>Peak CK &gt; 1000 IU/l</td>
<td>71%</td>
<td>57%</td>
<td>80%</td>
</tr>
</tbody>
</table>

Sensitivity for each time interval is defined as the percentage of dead patients who have the characteristic listed. Specificity is defined as the percentage of living patients without the characteristic. Abbreviation: MI = myocardial infarction.
cantly at each interval: in hospital ($p < 0.01$), 6 months ($p < 0.05$) and last follow-up ($p < 0.001$). Finally, analysis was performed to determine whether predictiveness by thallium scintigraphy could be significantly improved by the addition of any of the other clinical variables. No variable significantly increased the proportion of patients properly classified at any time interval.

**Discussion**

Our results indicate that in hemodynamically stable patients with acute MI, thallium-201 scintigraphy performed within 15 hours of the onset of symptoms can be used to predict subsequent in-hospital and post-discharge mortality. The overall mortality rate in our class I-II patients was 17% in hospital, 24% at 6 months and 33% at last follow-up (average 9 months), values comparable to those of other series.\(^7\)\(^-\)\(^11\)\(^,\)\(^22\) Goldberg et al.\(^a\) reported a 16.9% in-hospital mortality for acute infarct patients admitted without congestive heart failure and Norris et al.\(^a\) found an 18% hospital mortality in patients with at most moderate failure (admission chest radiographs described as normal or showing venous congestion or interstitial edema). More recently, Henning et al.\(^a\) reported a 13% hospital mortality for patients classified as class I-II on admission, although their class III appears to be more comparable to our class IIb; inclusion of class III patients increases their hospital mortality to 17%. In our study, the presence of large perfusion defects, as measured by an objective, computer-assisted scoring technique, identified a high-risk subset of class I-II patients with a mortality rate of 46% in hospital, 62% at 6 months and 92% at last follow-up. The predictiveness of a high thallium defect score could not be accounted for by any of a number of clinical variables previously associated with mortality. The presence of a high score was more predictive than the best combination of all other variables examined.

The value of a reliable prognostic index for patients suffering acute MI is clear. Early, accurate identification of high-risk patients may permit improved and more selective medical care. High-risk patients can be closely monitored in the coronary care unit for a longer period of time, ambulated more slowly, and followed more closely after hospital discharge, whereas low-risk patients can be ambulated more quickly and discharged from the hospital early. As new, more aggressive interventions are planned, it will be essential to identify high-risk patients quickly and reliably. These patients are the best candidates for aggressive treatment, provided they can be identified early, before hemodynamic deterioration and presumably before a sizable amount of ischemic myocardium progresses to infarction. By selecting high-risk patients, fewer patients would be required to prove a beneficial effect of therapy, and low-risk patients would not be unnecessarily exposed to the possible increased risk of these interventions.

Other investigators have described prognostic indexes in patients with acute MI using a combination of clinical variables\(^a\)\(^-\)\(^11\)\(^,\)\(^22\) and invasive hemodynamic measurements.\(^22\) Although these indexes improved stratification of risk, all were derived from studies that include patients admitted with marked hemodynamic compromise, including acute pulmonary edema or cardiogenic shock (class III-IV). We specifically excluded these patients from our study because we felt that they were more likely to have already suffered irreversible myocardial injury and, therefore, would be poor candidates for infarct-limiting therapy.

The thallium defect score, as a single index, appeared to be better than a number of clinical variables believed to be prognostically important: history of prior MI, anterior location of the acute infarct, higher peak levels of serum CK, and moderate LV failure. This finding may be explained by the fact that the extent of the thallium defect reflects the total mass of LV myocardium with reduced perfusion, including 1) areas of previous infarction, 2) areas of fresh necrosis, and 3) areas of jeopardized myocardium that are ischemic but not yet irreversibly injured.\(^18\)\(^,\)\(^19\) The combined mass of these areas would be expected to be an important determinant of reduced LV function. Although we did not perform invasive hemodynamic measurements in our patients to allow comparison with thallium scintigraphy, earlier studies have suggested that hemodynamic measurements, while providing certain prognostic information, lack sensitivity and specificity.\(^12\) This is probably because hemodynamic methods do not directly measure the size of the ischemic region, but reflect instead the depression of LV function produced by ischemia. LV function may also be depressed by alterations in LV loading conditions independent of myocardial ischemia.

The thallium defect score is prognostically useful because it reflects both the extent of necrosis that has already occurred and the amount of myocardium still jeopardized.\(^17\) Most of the in-hospital deaths occurred suddenly with an acute deterioration in LV pump function (table 1).\(^24\) This may have been related to extensive areas of old and new necrosis or possibly to extension of the infarct process. Myocardial rupture was not found in any of the five patients autopsied. The majority of postdischarge deaths also occurred suddenly, but the history in most cases suggested a new ischemic event. In these patients, the large perfusion defect found initially may have identified extensive areas of myocardium at risk for future ischemia.

Thallium scintigraphy, as currently performed, however, reflects only the relative distribution of perfusion, and the area of myocardium with the highest uptake in each view is arbitrarily defined as having normal blood flow. "Defects" are determined by comparing each area of left ventricle with this "normal" area. Theoretically, the left ventricle could have markedly but homogeneously reduced perfusion throughout, such as with extensive subendocardial infarction, cardiogenic shock or very severe triple-vessel coronary artery disease. The image would then look homogeneous and lack segmental defects. However, one would expect other abnormal findings in this
situation, such as high lung activity and low myocardial-to-background ratio, a dilated left ventricle with thin walls, or a severely reduced ejection fraction by echocardiography or gated cardiac blood pool imaging. Although this was not a problem in any of our patients, it is an important theoretical limitation and may be seen as more patients are studied.

Because the separation between high and low defect score was empirically determined, the results must be interpreted cautiously. Thallium scintigraphy is a safe, noninvasive technique, with prognostic ability superior to all combinations of clinical variables in predicting mortality in this population. We are presently performing further studies to validate our findings in a larger group of consecutive patients admitted to the coronary care unit. If the predictivity of thallium scintigraphy is validated, this technique will facilitate more aggressive and selective medical care of patients with acute MI.

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