Quantification of Infarct Size by \textsuperscript{201}TI Single-Photon Emission Computed Tomography During Acute Myocardial Infarction in Humans

Comparison With Enzymatic Estimates

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We prospectively investigated whether \textsuperscript{201}TI single-photon emission computed tomography (SPECT) could accurately diagnose the presence and quantify the extent of acute myocardial infarction when compared with infarct size assessed by plasma MB-creatine kinase activity. Thirty patients with enzymatic evidence of infarction were imaged within 12–36 hours of chest pain (mean, 23.4 hours). No patient had a previous infarction, and none underwent intervention seeking to restore coronary patency. Infarct size was quantified with computer-generated polar maps of the myocardial radioactivity and expressed as a percentage of the total left ventricular volume. To assess left and right ventricular performance, blood-pool gated radionuclide angiography was performed immediately after SPECT. All 30 patients had perfusion defects consistent with myocardial infarction. Scintigraphic and enzymatic estimates of infarct size correlated well for the group as a whole ($r=0.78$, $p<0.001$, SEE = 9.1) but especially for those patients with anterior infarction ($r=0.91$, $p<0.001$, SEE = 7.9). The poor correlation observed in patients with inferior infarction ($r=0.50$, $p<0.05$, SEE = 10.0) was believed to be related to the frequent occurrence of right ventricular involvement because SPECT assessed only left ventricular damage, whereas the enzymatic method estimated the myocardial injury in both ventricles. A quantitative index of right ventricular infarct size, derived from the relation between the scintigraphic and enzymatic estimates, had a strong inverse correlation with right ventricular ejection fraction ($r = -0.89$, $p<0.001$, SEE = 3.6). Therefore, \textsuperscript{201}TI SPECT is highly sensitive for detecting the myocardial perfusion deficit during acute infarction in humans and can accurately quantify left ventricular infarct size. The comparison of infarct size by SPECT to the enzymatic estimate may provide a means to assess the extent of right ventricular involvement in patients with inferior infarction. (Circulation 1988;78:831–839)

The extent of myocardial damage after acute infarction is an important prognostic indicator of subsequent morbidity and long-term mortality.$^{1-7}$ The enzymatic estimate of infarct size determined by plasma MB-creatine kinase (CK) activity is accurate for assessing the degree of myocardial necrosis$^{8,9}$ but cannot depict the three-dimensional geometry of the infarcted myocardium or discern the infarct-related coronary artery. Radionuclide perfusion scintigraphy is an alternative noninvasive method that may provide this important anatomic information. In addition, this technique may be useful for assessing infarct size and the extent of myocardial salvage after coronary thrombolysis in acute infarction, which may not be accurately estimated by the plasma MBCK activity.$^{99m}$Tc pyrophosphate and \textsuperscript{201}TI scintigraphy have been used extensively over the past 2 decades in the diagnosis$^{10,11}$ and prognostic stratification$^{1-4,12,13}$ of patients with acute infarction. The former, however,
is of limited value for assessing infarct size during coronary reperfusion. When coronary patency is not restored, maximal $^{99m}$Tc pyrophosphate uptake occurs between 2 and 3 days after the acute event and thus is too late to be of diagnostic relevance in the majority of patients. $^{201}$TI planar scintigraphy, when performed within 24 hours of onset of symptoms, is a highly sensitive technique for diagnosing myocardial infarction, but the sensitivity rapidly diminishes with time, particularly in patients who have minimal injury or non–Q wave infarction.\textsuperscript{10} Planar imaging with either radionuclide is suboptimal for diagnosing or accurately quantifying infarct size attributable to the inevitable overlap of abnormal with normal myocardium and with other tissues located in the chest.\textsuperscript{14} Thallium scintigraphy with single-photon emission computed tomography (SPECT), an inherently three-dimensional technique, appears promising for quantifying infarct size because it minimizes myocardial overlap.

Previous reports in animals and in humans have incorporated semiquantitative planimetric methods to estimate the amount of infarcted myocardium in serial tomographic slices.\textsuperscript{15,16} An alternative computerized method has recently been validated with a circumferential profile technique.\textsuperscript{17–19} This technique is highly reproducible and does not rely on subjective edge detection of the outer myocardial border or that of the perfusion defect.

The present investigation was undertaken to determine whether quantitative $^{201}$TI SPECT, with computer-generated polar maps of the myocardial radioactivity, could accurately estimate infarct size in patients during acute myocardial infarction when compared with a standard enzymatic technique.\textsuperscript{8} Furthermore, because many patients with inferior infarction have right ventricular involvement, we attempted to derive a quantitative index of right ventricular injury hypothesizing that the total measured plasma MBCK activity has variable contributions from the left and right ventricles, whereas infarct size assessed by SPECT would be restricted to the left ventricle.

**Patients and Methods**

**Patient Population**

The research protocol was approved by the Institutional Review Boards for Human Research of Baylor College of Medicine and The Methodist Hospital. All patients signed informed consent.

The study population consisted of 30 patients with their initial acute myocardial infarction (18 men, 12 women; mean age, 63.6 years; range, 38–87 years), 13 of whom had anterior infarctions and 17 of whom had inferior infarctions. All patients were imaged within 12–36 (mean, 23.4) hours after the onset of chest pain. No patient had previous myocardial infarction by electrocardiographic criteria or clinical history. Patients undergoing interventional treatment with thrombolytic drugs and/or percutaneous transluminal coronary angioplasty were excluded from the trial. All patients received routine acute coronary care unit treatment, consisting of opiates as required for chest pain and oxygen therapy. Nitrates, calcium channel antagonists, and $\beta$-blockers were only given if considered clinically indicated in individual cases.

Myocardial infarction was diagnosed if patients had chest pain lasting over 30 minutes, typical electrocardiographic changes of infarction, and a characteristic rise and fall in plasma MBCK activity. Electrocardiographic criteria for acute Q wave infarction required ST segment elevation of at least 0.1 mV in two or more of the six precordial leads for anterior infarction, and in leads II, III, and aVF for inferior infarction, with development of pathological Q waves. Non–Q wave infarction required new and persistent ST segment depression of at least 0.1 mV in the appropriate leads.

**Scintigraphic Protocol**

Patients enrolled in the study were transported by stretcher under constant electrocardiographic surveillance to the Nuclear Cardiology Laboratory where they were injected with 3 mCi $^{201}$TI. SPECT was performed with a large field of view rotating single-crystal gamma camera (ADAC 3000) equipped with a low-energy, high-resolution collimator and interfaced to a computer. The camera’s energy discriminator was set on the 71- and 167-keV photopeaks of $^{201}$TI with a 20% window. Image acquisition commenced in the 45° left posterior oblique projection approximately 5 minutes after the injection of $^{201}$TI. Thirty-two sequential images separated by 6° intervals were acquired over a 180° arc at 40 sec/image. The data were stored on a $64 \times 64 \times 8$ byte matrix for subsequent analysis.

Transaxial image reconstruction used a standard back projection technique with a Butterworth (order, 5) high-pass filter–low-pass window at a 50% cutoff. The reconstructed transaxial tomographic slices (6-mm thickness) were then obliquely reoriented and displayed in the short, horizontal long, and vertical long axes on a large (10.5×7.5 in.) color oscilloscope for visual interpretation.

Blood-pool gated radionuclide angiography (RNA) was performed in each patient immediately after completion of $^{201}$TI SPECT for determination of right ventricular ejection fraction (RVEF) and left ventricular ejection fraction (LVEF). Patients were injected through a peripheral vein with stannous pyrophosphate followed by 25–30 mCi $^{99m}$Tc pertechnetate for in vivo red cell labeling. The camera’s energy discriminator was set at 140 keV with a 20% window, and all images were processed with the dedicated computer. Images were obtained in the standard anterior, 70° left anterior oblique, and left anterior oblique projection with the best septal delineation. A total of 250 counts/pixel were acquired over the left ventricle at 16 frames/cardiac cycle. LVEF and RVEF were calculated with variable
regions of interest. This method for determining LVEF and RVEF has been previously validated and reported and exhibits small intraobserver variability.\textsuperscript{20\textendash}23 RVEF was calculated in all patients except for one patient who had a study of poor technical quality. Global LVEF and RVEF in our laboratory are considered to be abnormal if less than 50\% and 39\%, respectively.\textsuperscript{24} This strict radionuclide criterion for right ventricular dysfunction has been used by other investigators to diagnose right ventricular infarction.\textsuperscript{25\textendash}28 In the present study, however, we used the RVEF only as a clinical indicator of right ventricular dysfunction because not all patients with depressed right ventricular function necessarily have right ventricular infarction.

**Determination of Infarct Size by Plasma MBCK Activity**

Blood samples for total CK and MBCK analysis were drawn every 4 hours for the first 24 hours, every 6 hours for the next 24 hours, and every 12 hours thereafter until normalization. All samples were mixed with ethyleneglycoltetraacetic acid and centrifuged for 10 minutes at 2,500g to remove the cellular components. Samples were then stored at \(-20\)° C after addition of \(\beta\)-mercaptoethanol. MBCK was quantified after separation from MMCK with the glass-bead batch absorption assay.\textsuperscript{29} Total plasma CK and MBCK activities were determined spectrophotometrically by the method of Rosalki.\textsuperscript{30} Myocardial infarct size was calculated from the plasma MBCK time activity curve for each patient with a previously validated technique.\textsuperscript{8,31}

**Quantification of Infarct Size by Tomography**

Infarct size was quantified by tomography with computer-generated two-dimensional polar maps of the three-dimensional \(^{203}\text{Tl}\) activity in the heart.\textsuperscript{19} The distribution of myocardial radioactivity for each slice was determined with a circumferential profile technique whereby each individual short-axis slice was normalized to the maximal count activity within the slice. The polar maps were generated as follows (Figure 1). The center of the left ventricle, the maximal search radius, the right ventricular junction, and the boundaries of the left ventricular cavity were determined from the midcavity short-axis slice by a single investigator for all patients. The maximal search radius was constructed around the outer border of the left ventricle, thus effectively excluding right ventricular activity. The center and the boundaries of the left ventricular cavity, as well as the position of the apex, were also marked in the midcavity vertical long-axis slice. These boundaries were visually selected at the scintigraphic endocardial edges. The apex of the heart was represented within the center of the polar map and derived from the vertical long-axis slices. The sequential short-axis slices from apex to base were represented as concentric circumferential rings from the center to the periphery of the polar map.

The polar map of each patient was then compared with that of a mixed-gender normal data bank consisting of 50 patients (23 men and 27 women; mean age, 50; range, 26\textendash}69 years), who either had normal coronary arteriograms or were at low risk for coronary artery disease in that they had non-
anginal chest pain, achieved more than 85% of their predicted heart rate on exercise stress testing without development of ischemic electrocardiographic changes, and had no more than two cardiac risk factors. Each patient’s polar map was also compared with gender-specific data banks, consisting of 30 men and 35 women. Although these data banks were obtained from exercise images, we believed ourselves to be justified in using them as the normal standard of relative regional ${}^{201}$TI uptake for analyzing our resting studies because the distribution of ${}^{201}$TI in normal myocardium is homogeneous and similar during rest or exercise. A pixel count was considered abnormal if it fell 2.5 standard deviations below its corresponding mean normal pixel count. Infarct size was then automatically expressed as the percentage of abnormal pixels in the total polar map (the total left ventricular volume) (Figure 2).

**Assessment of Right Ventricular Infarct Size**

Right ventricular damage is common with inferior but rare with anterior infarction. We therefore hypothesized that the linear regression equation obtained by comparing enzymatic and scintigraphic estimates of infarct size in our patients with anterior infarction (with the scintigraphic infarct size as the independent variable) would describe a relation expressing solely left ventricular damage. Using this equation, we calculated the predicted enzymatic estimate of left ventricular infarct size from the scintigraphic estimate in patients with inferior infarction. We then derived a quantitative index for right ventricular infarct size in this subgroup of patients with inferior infarction by subtracting the predicted left ventricular enzymatic infarct size from the observed total enzymatic infarct size. This index assumes that enzymatic estimates reflect release of MBCK from the left and right ventricles. Right ventricular infarction was considered present if the index had a positive value.

**Statistical Analysis**

Comparisons of LVEF, RVEF, and infarct size in the anterior versus inferior infarction groups were performed with unpaired two-tailed $t$ tests. Paired two-tailed $t$ tests were used when comparing infarct size as assessed by SPECT and plasma MBCK
activity. Linear regression analysis was used to compare scintigraphic with enzymatic estimates of infarct size, RVEF and LVEF with infarct size, and RVEF with the right ventricular infarct size index. Correlation coefficients (r) and SEE were calculated, p<0.05 was considered significant. Data are expressed as the mean ± SD.

Results

Correlation Between Scintigraphic and Enzymatic Estimates of Infarct Size

All 30 patients had perfusion defects by SPECT consistent with the enzymatic, electrocardiographic, and clinical evidence of acute myocardial infarction. Scintigraphic and enzymatic results are shown in Table 1. Infarct size assessed by SPECT with the mixed gender data bank was similar for both inferior and anterior infarction (28.8±11.2% vs. 27.2±18.0%, respectively; p=NS), whereas infarct size assessed by plasma MBCK activity tended to be larger in inferior infarction (40.7±16.4 vs. 33.3±31.6 CK/geq), although this difference did not reach statistical significance.

When all patients were reanalyzed with the gender-specific data banks and compared with our original mixed-gender analysis, the mean scintigraphic infarct size was not significantly different (29.1±14.6% vs. 28.1±14.3%, respectively; p=NS). Regression analysis yielded a high correlation coefficient (r=0.99, p<0.001, SEE=2.3) with the regression equation having a y intercept and slope of 0.03 and 0.97, respectively. Likewise, infarct size assessed by the original data bank was similar to that assessed by gender-specific data banks in men (33.0±13.6% vs. 32.9±14.3%, respectively; r=0.99, p<0.001, SEE=1.8) as well as in women (20.8±12.5% vs. 23.3±13.7%, respectively; r=0.99, p<0.001, SEE=1.5). Thus, using separate data banks for men and women did not alter the infarct size analysis.

The correlation coefficient between the scintigraphic and enzymatic estimates of infarct size in the group as a whole was r=0.78, p<0.001, and SEE=9.1 (Figure 3). Subgroup analysis showed a high correlation in patients with anterior infarction (r=0.91, p<0.001, SEE=7.9), with the following regression equation: SPECT infarct size = 10.0 + 0.52 × enzymatic infarct size. In contrast, the correlation in patients with inferior infarction was poor (r=0.50, p<0.05, SEE=10.0). The respective slopes and intercepts of the regression equations for anterior and inferior infarction patients, however, were not statistically different, probably because of the small numbers in each subgroup. Right ventricular dysfunction, as assessed by RNA, was common in inferior infarction (11 of 16 patients) but rare in anterior infarction (one of 13 patients). When the patients with anterior infarction and those with inferior infarction without right ventricular dysfunction were analyzed together, the resultant regression equation was virtually identical (SPECT infarct size = 11.8 + 0.53 × enzymatic infarct size; r=0.88, p<0.001, SEE=8.1) to that observed in patients with anterior infarction.

Table 1. Enzymatic and Scintigraphic Characteristics

<table>
<thead>
<tr>
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<th>Anterior myocardial infarction (n = 13)</th>
<th>Inferior myocardial infarction (n = 17)</th>
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<tr>
<td></td>
<td>Mean (± SD)</td>
<td>Range</td>
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<tr>
<td>LVEF (%)</td>
<td>37.5±11.7</td>
<td>17–63</td>
</tr>
<tr>
<td>RVEF (%)</td>
<td>48.1±8.0</td>
<td>34–61</td>
</tr>
<tr>
<td>SPECT IS (%)</td>
<td>27.2±18.0</td>
<td>2–68</td>
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<tr>
<td>MBCK IS (geq)</td>
<td>33.3±31.6</td>
<td>3–99.5</td>
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</tbody>
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*Anterior versus inferior infarction.

IS, infarct size; LVEF, left ventricular ejection fraction; RVEF, right ventricular ejection fraction; SPECT, single-photon emission computed tomography.
alone. This connotes that the major difference between the two techniques for sizing infarcts is not the site of infarction but rather the presence or absence of right ventricular involvement.

**Right Ventricular Infarct Size Index**

A positive right ventricular infarct size index was present in 10 of the 11 patients with inferior infarction who had right ventricular dysfunction but in none of the five patients with a normal RVEF. Likewise, enzymatic estimates of infarct size in patients with inferior infarction who had documented right ventricular dysfunction by RNA were significantly larger than scintigraphic estimates (43.7 ± 17.3 CK/geq vs. 24.4 ± 9.8%, respectively; \( p<0.001 \)). In the five patients with inferior infarction who had normal right ventricular function, however, the enzymatic and scintigraphic estimates of infarct size were virtually identical (38.0 ± 13.8 CK/geq vs. 38.4 ± 9.8%, respectively, \( p=NS \)). The correlation between the derived quantitative index of right ventricular infarct size and the RVEF was good \(( r = -0.89, \ p<0.001, \ SEE = 3.6) \) (Figure 4).

**Ejection Fraction and Infarction Site**

Patients with anterior infarction had more depressed left ventricular function than those with inferior infarction (mean LVEF, 37.5 ± 11.7% vs. 56.7 ± 9.8%, respectively; \( p<0.001 \)), whereas patients with inferior infarction had more depressed right ventricular function compared with those with anterior infarction (mean RVEF, 34.4 ± 7.9% vs. 48.1 ± 8.0%, respectively; \( p<0.001 \)) (Table 1). Seven of 13 patients (54%) with anterior infarction had an LVEF of less than 40% compared with only two of 17 patients (12%) with inferior infarction. The right ventricular function was depressed in 11 of 16 patients with inferior infarction (69%) but in only one patient with anterior infarction (8%). The RVEF was only mildly depressed (34%) in this latter patient, whereas eight of the 11 patients with inferior infarction had an RVEF of less than 32%.

**Correlation Between Estimates of Infarct Size and Ejection Fraction**

In patients with anterior infarction, a good correlation was found between infarct size assessed by SPECT and LVEF \(( r = -0.74, \ p<0.004, \ SEE = 8.2) \). In patients with inferior infarction, the correlation between scintigraphic infarct size and LVEF was poor \(( r = -0.28, \ p = NS, \ SEE = 9.7) \). In these patients, a poor correlation was also observed when left ventricular scintigraphic and enzymatic estimates of infarct size were compared with RVEF \(( r = -0.41, \ p = NS, \ SEE = 7.40; \ r = -0.31, \ p = NS, \ SEE = 7.7, \) respectively). Despite a similar infarct size by SPECT in patients with anterior or inferior infarction, the latter had only mild left ventricular dysfunction (mean LVEF, 56.7 ± 9.8%).

**Discussion**

The results of the present study show that \(^{201}\text{TI SPECT can be used to accurately diagnose and quantify the extent of myocardial infarction in humans with computer-generated polar maps of the three-dimensional myocardial radioactivity. Because }^{201}\text{TI perfusion scintigraphy cannot distinguish previous infarction from acute myocardial necrosis, only patients without a history of infarction were evaluated. Furthermore, patients undergoing acute coronary reperfusion were excluded because of present controversy as to whether the enzymatic method, using plasma MBCK activity, is valid for measuring infarct size in such a population. A unique feature of our investigation was to combine }^{201}\text{TI SPECT with RNA imaging in all patients during the acute stage of myocardial infarction. Although it is well recognized that pathology remains the definitive method for confirming the diagnosis of right ventricular infarction, the blood-pool RNA technique provides reasonable indirect evidence of right ventricular involvement by documenting the presence of right ventricular dysfunction.}^{20,21,23–28,34,36}

Previous investigators have used \(^{201}\text{TI planar scintigraphy to diagnose acute myocardial infarction. Wackers et al}^{19}\) clearly showed that patients imaged within 24 hours of chest pain invariably had \(^{201}\text{TI perfusion defects. The defects tended to diminish, however, after the first 24 hours, especially when non-Q wave or biochemically small infarctions were present. Recently, several investigators have demonstrated an improved sensitivity of SPECT over planar imaging for detecting infarction.}^{37,38}\) Ritchie et al.\(^{37}\) reported a sensitivity of 87% with tomography but only 63% with planar imaging in 38 patients who had remote myocardial infarctions. Tomography was especially helpful for diagnosing inferior and non-Q wave infarctions. In the present study, where most patients were imaged an average of 1 day after the onset of chest pain, all demonstrated perfusion defects consistent with infarction, including the five patients with non-Q wave infarctions. These data further substantiate the high sensitivity of \(^{201}\text{TI SPECT imaging for diagnosing small infarctions.}

Infarct size estimated by tomographic imaging in the animal model compares well with morphological estimates of myocardial damage. Prigent et al.\(^{18}\) reported an excellent correlation between infarct size delineated by triphenyltetrazolium chloride myocardial staining and that assessed by SPECT in dogs. Their tomographic technique was similar to ours, with acquisition over a 180° rotational arc and without correction for attenuation or photon scatter. Likewise, Prigent et al used a circumferential profile technique that does not depend on myocardial edge detection of perfused and nonperfused areas.

Tamaki et al.\(^{16}\) studied 18 patients in the late (4 weeks) postinfarction period in what is the only
clinical trial comparing $^{201}$TI scintigraphy and enzymatic estimates of infarct size. Perfusion defects were measured through manual and computer-aided planimetry of the perfused and nonperfused myocardial regions. The studies were acquired over 360° with attenuation correction and background subtraction to aid with myocardial edge detection. Quantification of infarct size with planar scintigraphy fared less well than tomography when compared with enzymatic estimates of infarct size ($r=0.69$ vs. 0.89, respectively). Tamaki et al deliberately excluded patients with inferior infarction who had clinical, hemodynamic, or scintigraphic evidence of right ventricular damage, which may explain why their overall correlation is better than ours.

In the present study, we found a good overall correlation between the SPECT and enzymatic estimates of infarct size ($r=0.78$, $p<0.001$, SEE = 9.1), which was especially close in patients with anterior infarction ($r=0.91$, $p<0.001$, SEE = 7.9). The corresponding correlation in patients with inferior myocardial infarction, however, was poor ($r=0.50$, $p<0.05$, SEE = 10.0). In the latter patients, the enzymatic estimates were significantly higher than those determined by scintigraphy. Enzymatic estimates include an inseparable contribution of MBCK from both ventricles in patients who have inferior and concomitant right ventricular infarction, whereas scintigraphic estimates are restricted to left ventricular damage. Scintigraphic infarct size should, therefore, better approximate the enzymatic results in patients without right ventricular infarction. Indeed, when we combined patients with anterior infarction and inferior infarction with a normal RVEF, the correlation between enzymatic and scintigraphic estimates was very good ($r=0.88$, $p<0.001$, SEE = 8.1). Many of our patients with inferior infarction, however, had concomitant right ventricular dysfunction, albeit at a prevalence consistent with previous reports. Therefore, the disparity between left ventricular scintigraphic and enzymatic estimates of infarct size in patients with inferior infarction is, we believe, reconciled by the frequent association of left and right ventricular infarction in these patients.

In an attempt to derive a quantitative index of the extent of injury in the right ventricle, a regression equation was developed relating scintigraphic and enzymatic estimates of infarct size in patients with anterior infarction. With this equation, we then calculated the predicted enzymatic infarct size for the left ventricle from the corresponding scintigraphic value in each patient with inferior infarction. The extent of right ventricular involvement was calculated by subtracting this predicted value from the actual enzymatic measurement and represented a quantitative index of right ventricular infarct size. Linear regression analysis showed a close inverse correlation between this index and RVEF, similar to what we had observed between scintigraphic infarct size and LVEF in patients with anterior infarction.

The present study would be further strengthened if our quantitative findings on right ventricular damage could be confirmed by an additional independent technique. Although right precordial electrocardiography is both highly sensitive and specific for diagnosing right ventricular infarction when obtained early in patients with inferior infarction, the technique affords only a qualitative assessment of right ventricular damage. Likewise, hemodynamic criteria are useful for detecting the presence but not for quantifying the extent of right ventricular infarction. Further validation of our method would therefore require morphological confirmation of right ventricular injury because there is at present no noninvasive method to quantify the extent of right ventricular infarction.

Radionuclide angiography performed during the acute phase of infarction revealed greater left ventricular dysfunction in those patients with anterior infarction than in those with inferior infarction. There was a good correlation between the degree of left ventricular dysfunction and infarct size by SPECT in patients with anterior infarction ($r=−0.74$, $p<0.004$, SEE = 8.2) but not in those with inferior infarction. The latter patients generally had well-preserved left ventricular function despite a mean scintigraphic infarct size comparable to that seen in anterior infarction. Although patients with anterior infarction generally have more extensive damage than those with inferior infarction, this was not observed scintigraphically in the present study, probably due to the patient selection criteria. At our institution, patients with clinical evidence of extensive anterior infarction usually undergo acute coronary reperfusion and so were excluded from enrollment.

In conclusion, we have shown that $^{201}$TI SPECT is a highly sensitive technique for detecting the myocardial perfusion deficit during acute infarction in humans and can be used to accurately quantify the extent of myocardial damage. We have also documented that the apparent discrepancy between the estimates of infarct size by SPECT and plasma MBCK activity in patients with inferior myocardial infarction may be ascribed to concomitant right ventricular involvement. Furthermore, by comparing enzymatic and scintigraphic estimates of infarct size, we derived a quantitative index of the extent of right ventricular infarction, which correlated well with the severity of right ventricular dysfunction. A larger study will be required to confirm our findings in patients with acute infarction. With the advent of reperfusion techniques during acute myocardial infarction, assessment of infarct size by SPECT may prove to be an invaluable tool for determining the efficacy of acute coronary recanalization and the extent of myocardial salvage.

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