Usefulness and Limitations of Thallium-201 Myocardial Scintigraphy in Delineating Location and Size of Prior Myocardial Infarction

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SUMMARY In order to evaluate the usefulness of thallium-201 (201Tl) myocardial scintigraphy in delineating the location and size of prior myocardial infarction, 32 patients were evaluated at a mean of 7 ± 2 months after infarction with a 12-lead ECG, resting 201Tl myocardial scintigram, biplane left ventriculogram and coronary angiograms. From the left ventriculogram, asynergy was quantified as percent abnormally contracting segment (% ACS), the percent of end-diastolic circumference which was either akinetic or dyskinetic. Using a computerized planimetry system, we expressed 201Tl perfusion defects as a percentage of total potential thallium uptake.

Of 21 patients with ECG evidence of prior transmural infarction, a 201Tl defect was present in 20 (95%), and angiographic asynergy was present in all 21 (100%). The site of prior infarction by ECG agreed with the 201Tl defect location in 24 of 32 patients (75%) and with site of angiographic asynergy in 23 of 32 patients (72%). Scintigraphic defects were present in only four of 10 patients (40%) with ACS ≤ 6%, but scintigraphic defects were found in 20 of 22 patients (91%) with ACS > 6% (p < 0.01). Thallium defect size correlated marginally with angiographic left ventricular ejection fraction (r = -0.60) but correlated closely with angiographic % ACS (r = 0.80). Thallium defect size was similar among patients with one-, two-, or three-vessel coronary artery disease (≥70% stenosis), but thallium defect size was larger in patients with electrocardiographic evidence of transmural infarction (p < 0.01) or pulmonary capillary wedge pressure > 12 mm Hg (p < 0.001). Thus, resting 201Tl myocardial scintigraphy is useful in localizing and quantifying the extent of prior myocardial infarction, but is insensitive to small infarcts (ACS < 6%).

ASSESSMENT OF THE EXTENT OF non-contractile left ventricle after myocardial infarction is important for correlating the extent of hemodynamic derangement and the subsequent clinical course in patients.1-3 The evaluation of left ventricular (LV) wall motion has been limited to invasive angiography because of the unreliability of noninvasive techniques in ischemic heart disease. Many techniques in nuclear cardiology are being examined for their usefulness in delineating LV wall motion.4-6

The application of resting potassium-analogue myocardial scintigraphy in the evaluation of ischemic heart disease has broadened rapidly over the last 5 years, and use of the potassium analogue, thallium-201 (201Tl), is now practical at a reasonable cost. Good qualitative relationships between location of 201Tl myocardial perfusion defects and ECG,7-10 ventriculography,11 cardiac enzymes,12 coronary arteriography,9-11 and postmortem studies12 have been reported. Significant quantitative relationships have been demonstrated between 42K myocardial perfusion defects and left ventriculographic wall motion abnormalities in the late post-infarction period.14 The magnitude of 201Tl perfusion defects correlates semi-quantitatively with the extent of left ventricular asynergy in man15 and quantitatively with pathological evidence of infarction in animals.16-17

However, no study has evaluated the usefulness and limitations of 201Tl scintigraphy in delineating quantitatively the extent of left ventricular asynergy in patients several weeks to months after infarction. Thus, we sought to establish, in such patients, a technique for sizing 201Tl scintigraphic defects and to compare the location and size of those defects with the location and extent of angiographic left ventricular asynergy.

Methods

Population

The study population consisted of all patients evaluated at the University of Alabama Medical Center over a 12-month period who satisfied all of the following criteria: 1) prior myocardial infarction documented by a typical enzyme pattern, and electrocardiographic (ECG) evidence of anterior, lateral or inferior infarction manifest by poor anterior lead R wave progression, QS complexes or Q waves ≥ 0.04 msec, as described by Friedman;18 2) technically satisfactory biplane left ventriculogram; and 3) 201Tl myocardial scintigraphy in three views (anterior, left lateral and 45° left anterior oblique (LAO)). Patients
with valvular heart disease were not included in this study.

**Thallium Scintigraphy**

Each patient received 1.5 mCi $^{201}$TI (New England Nuclear, North Billerica, Massachusetts) intravenously and was imaged approximately 10 minutes later with a Series 110 Ohio Nuclear mobile scintillation camera (Ohio Nuclear, Inc, Solon, Ohio) equipped with a high resolution collimator. Anterior, 45° LAO and lateral scintigrams were recorded at a uniform image intensity gain setting on Polaroid 107,084 emulsion film at f-8, acquiring 300,000 counts for each view.

Location of the scintigraphic defects was designated as inferior, lateral, apical, anterior (including septal) or posterior, as shown in figure 1. For purposes of comparing scintigraphic and angiographic defect sites with ECG infarct site, the apical segment was combined with the involved, adjacent myocardial segment (anterior, lateral or inferior). The size of the $^{201}$TI defect was determined by a computerized planimetry system using a digitized sonic pen as described in figure 2. The defect size was computed in the anterior (% $^{201}$TIA), lateral (% $^{201}$TIL) and LAO (% $^{201}$TILA0) views. The average of the % $^{201}$TIA and % $^{201}$TIL was determined (% $^{201}$TII).

Each scintigram was evaluated independently by two observers who recorded the location and boundaries of the $^{201}$TI defects. Inter-observer agreement regarding location of $^{201}$TI scintigraphic defects was 100%. The defect boundaries were compared and minimal disagreement was settled by arbitration between the two observers. In five of 96 scintigraphic views (5.2%), disagreement in defect boundary recognition could not be arbitrated, and in those five cases, the mean of the two determinations of % $^{201}$TII was used.

**Coronary Arteriography**

Selective coronary arteriography was performed on all patients, and the arteriograms were interpreted by consensus of three observers who graded the percent stenosis of vessel diameter.

We determined the coronary stenosis score for each patient using the technique devised by Freisinger, which assigns a score of 0–5 to each of the three coronary arteries in order of increasing stenosis. The total of the three scores can range from no disease (0) to total obstruction of all three vessels (15).

**LV Angiography**

Biplane large film or cine left ventriculography was performed on all patients. Wall motion abnormality was quantitated as percent abnormally contracting segment (% ACS) by the method of Feild et al. using fixed external radiographic markers as references for superimposing the end-systolic and end-diastolic silhouettes as shown in figure 3. Two independent observers determined % ACS, and we used the average of these two determinations for subsequent data analysis.

**Statistics**

We found linear correlations by the method of least squares analysis, and used the unpaired $t$ test and chi square test to assess differences between groups of unpaired data. Data are expressed as the mean ± SEM.

**Results**

**Patient Population**

Thirty-two patients satisfied the study criteria; the 30 men and two women were 33–71 years old with a mean age of 51 ± 2 years. The time between infarction and scintigraphy ranged from 6 days–69 months.

**Figure 1. Location of ventriculographic and scintigraphic abnormalities. The segments of the left ventricle are designated for the ventriculographic silhouettes (left) and the scintigraphic images (right). Note the different nomenclature for the mediasternal portions of the left ventricle on the anterior view. INF = inferior; LAT = lateral; ANT = anterior; POST = posterior.**
SCINTIGRAM

TRACING

AREA OF DEFECT

21%

27%

% THALLIUM DEFECT = \frac{\text{AREA OF ABNORMAL TL UPTAKE}}{\text{AREA OF POTENTIAL TL UPTAKE}} \times 100

Figure 2. Quantitation of the size of the thallium (TL) defect. Polaroid images of the $^{201}$TI myocardial scintigrams in anterior and lateral projection (left column) were traced (right column). Using computerized planimetry, the area of decreased thallium uptake (shaded area) and the total area of potential thallium uptake (shaded plus solid area) were measured. The ratio of the area of abnormal thallium uptake to the total area of potential uptake was expressed as the percent thallium defect and averaged for the two views.

% ACS = \frac{\text{ASYNERGIC DIASTOLIC SEGMENT (cm)}}{\text{TOTAL END DIASTOLIC CIRCUMFERENCE (cm)}} \times 100

Figure 3. Quantitation of asynergy. End-diastolic and end-systolic silhouettes were superimposed using fixed x-ray beam markers. The segment of the end-diastolic circumference which was akinetic or dyskinetic (shown between the brackets) was expressed as a percentage of the total end-diastolic circumference. This percentage was averaged for the anteroposterior ($\% \text{ACS}_a$) and lateral ($\% \text{ACS}_l$) projections and designated the percent abnormally contracting segment ($\% \text{ACS}$).

(mean 7 ± 2 months); only two patients were scanned within 2 weeks of infarction. The median time between scan and angiography was 2 days (mean 6 ± 2 days, range 0–37 days), and only one patient had scintigraphy and angiography more than 1 month apart.

Electrocardiography

Eleven patients, including two with LV conduction abnormalities, had no residual ECG evidence of prior transmural infarction. Of the remaining 21 patients with ECG criteria for prior transmural myocardial infarction, there were 14 anterior, five lateral and eight inferior patterns. Of the 14 anterior infarctions, 12 had Q or QS patterns and two had diminished R waves or poor R wave progression. Six patients had infarction patterns involving more than one major area.

An ECG pattern of infarction, seen in 21 patients, correctly predicted the presence of a $^{201}$TI defect in 20 patients (95%) and the presence of an ACS in 21 patients (100%). In 20 of 21 patients (95%) with an ECG scar, the angiogram demonstrated an ACS involving that site.

The agreement between the location of the ECG scar site and the site of $^{201}$TI defect and angiographic
ACR is shown in figure 4. Overall, both scintigraphy and ventriculography agreed with ECG location of the scar site in about 75% of the cases. However, there was a difference in the sensitivity of the three techniques in detecting small residual scars. All patients had documented infarctions. The 11 patients with no residual ECG scar are therefore “falsely negative” for infarction. Of these “falsely negative” ECGs, seven patients (64%) had “falsely negative” scintigrams, which represents a 36% improvement in detection of old infarctions by scintigraphy. Similarly, of these 11 “falsely negative” ECGs, only three (27%) had angiograms which were “falsely negative” for evidence of infarction, an improvement of 73% in detection of old infarctions. The interval between infarction and scintigraphy was not significantly different in patients with (7 ± 2 months) or without (5 ± 3 months) 201TI defects.

Thus, of these 32 patients with myocardial infarction, 21 (66%) were recognized by ECG, 24 (75%) were recognized by scintigraphy and 27 (84%) were recognized by angiographic ACS. Statistical analysis (Cochran’s Q test for correlated tables22) showed that angiographic ACS was significantly more sensitive than ECG (p < 0.005), but only marginally more sensitive than scintigraphy (p = 0.06) in detecting prior infarction. Detection of infarction by scintigraphy and ECG was not significantly different (p > 0.10).

The size of the % 201TI defect was not significantly different in different ECG infarction sites (fig. 5).
although the size of the $^{201}$TI defect was clearly smaller ($p < 0.01$) in patients who had no ECG evidence of prior transmural myocardial infarction.

Coronary Arteriography

All patients had $\geq 70\%$ narrowing of at least one coronary artery. There were 10 patients with one-vessel, nine patients with two-vessel and 13 patients with three-vessel coronary artery disease (two of whom had left main coronary artery stenosis). There were no significant differences in % $^{201}$TI in patients with single-, two- or three-vessel coronary artery stenosis (fig. 5). The mean Freisinger score for patients with $^{201}$TI defects (9.7 $\pm$ 0.6) did not differ significantly from that of patients without $^{201}$TI defects (9.1 $\pm$ 1.0), and there was no correlation ($r = 0.04$) between the Freisinger score and % $^{201}$TI.

Hemodynamics

Patients with abnormal pulmonary capillary wedge pressure (>$12$ mm Hg) before angiography had significantly larger $^{201}$TI defects than those with normal wedge pressures (fig. 5; $p < 0.001$). A $^{201}$TI defect $>20\%$ was present in six patients, including all four patients who had elevated pulmonary capillary wedge pressure. The pulmonary capillary wedge pressure showed a poor linear correlation with $^{201}$TI defect size ($r = 0.51$). There was no significant correlation between $^{201}$TI defect size and cardiac index ($r = -0.31$).

Left Ventriculography

The % ACS, ranged from 0-40% (mean 14 $\pm$ 2%). The % $^{201}$TI, defect ranged from 0-40% (mean 11.4 $\pm$ 1.8%). The presence of a $^{201}$TI defect predicted the presence of an ACS in 22 of 23 patients (96%). In the one patient with a $^{201}$TI defect but without LV asynergy, the $^{201}$TI defect was inferoapical. In the eight patients with normal $^{201}$TI scans, six patients had an ACS, $<6\%$ (mean 5 $\pm$ 2%).

When a $^{201}$TI defect was present in a segment (in the anteroposterior or lateral view), an ACS was seen in that segment in 42 of 57 patients (74%). Conversely, given the presence of an ACS in a segment, a $^{201}$TI defect was seen in that segment in 42 of 60 patients (70%). $^{201}$TI defect was most accurately predictive of an ACS in the anterior segment; all 10 segments involved with an anterior $^{201}$TI defect were involved with an anterior ACS. Scintigraphy was less accurately predictive of an ACS in the inferior (13 of 20, 65%) and apical (19 of 24, 79%) segments. Only three scintigrams showed perfusion defects in the lateral segment and in these three patients the lateral defect was not confirmed by a lateral ACS.

For all five LV segments in the 32 patients, agreement between scintigraphy and angiography regarding the presence or absence of a defect occurred in 78% of segments (fig. 6). The lateral wall segment showed the poorest agreement (66%). The high agreement in the posterior segment was caused by the absence of defects on all scintigrams and angiograms in the posterior segment.

There was an inverse correlation (fig. 7) between the % $^{201}$TI, and LV ejection fraction ($r = -0.60$). Figure 8 shows the relationship between the size of the ACS and the presence or absence of a $^{201}$TI defect. A critical minimum scar size (an ACS $>6\%$) had to be present before the scintigram reliably showed a defect. In this series 20 of 22 patients (91%) with % ACS $>6\%$ had $^{201}$TI defects, whereas six of 10 patients (60%) with % ACS $\leq 6\%$ had no $^{201}$TI defect ($p < 0.01$). The linear correlation between size of ACS, and size of $^{201}$TI defect is shown in figure 9. A wide range of defect sizes by both techniques is plotted. From 0-40% the linear correlation between angiographic % ACS and scintigraphic defect size is 0.80.

The correlation between angiographic and scintigraphic defects in the separate lateral and anterior views is shown in figure 10. The % $^{201}$TI, defect, when compared with the % ACS, (fig. 11), correlates the least well of all the scintigraphic views ($r = 0.39$).

Discussion

This study demonstrates that resting $^{201}$TI myocardial scintigraphy is useful in the identification and
quantification of abnormal segments of left ventricle after myocardial infarction if the abnormal segments are ≥ 6% of left ventricular circumference.

Technical Considerations

Quantification of $^{201}$TI scintigraphic perfusion defects in survivors of acute myocardial infarction has not previously been reported. Difficulties arise in devising a technique for assessing the size of a $^{201}$TI scintigraphic defect. The technique used in this study required only tracing and measuring the area of the $^{201}$TI defect from the Polaroid prints. Computer enhancement and color displays of $^{201}$TI scintigrams$^{23, 24}$ may eventually be able to detail more precisely and objectively the magnitude and extent of regional decreased radionuclide count density. This degree of sophistication, however, is not widely available.

Because of the known effect of acute infarction$^{25, 26}$ and transient ischemia$^{27}$ on both the size of the $^{201}$TI defect and the degree of LV asynergy, our patients were studied at rest long after acute infarction. By this technique we hoped to measure the fixed myocardial scar. The good overall correlation in our study between angiography and scintigraphy suggests that these mitigating factors do not play a large role.

Locating the Infarction Site by Scintigraphy

In this study the electrocardiogram was used as a standard for localizing infarction, against which the scintigram and LV angiogram were compared. The electrocardiogram may not be an excellent tool for localizing infarction$^{19}$ because the number of possible responses is limited. Both the $^{201}$TI scintigram and LV angiogram, however, represent more of a continuum with unlimited patterns of response.

The location of $^{201}$TI perfusion defects after infarction has been observed by others to correlate with
ECG infarct site with a frequency of 70–94%, the higher frequencies being observed when acute rather than chronic ECG changes were used for locating the ECG infarct site. Our data showing 75% agreement in “scar” localization between 201TI scintigraphy and chronic ECG changes (fig. 4) are compatible with postmortem data showing a 70% correlation between ECG scar site and anatomical infarct site.

Since our study used anteroposterior and lateral angiographic views rather than RAO and LAO views, the reliability of the LAO scintigram in predicting infarct location cannot be assessed with certainty. It has been suggested that the LAO view best delineates vascular beds for localization of coronary stenosis by 201TI scintigraphy, but this has not been proven for infarct localization. The standard LAO scintigraphic projection used in this study tends to foreshorten the LV image, making quantitative assessment of tangential perfusion defects less precise. A comparison of “long-axial” LAO LV angiograms and “long-axial” LAO 201TI scintigrams would be of interest in this regard.

**Angiographic and Hemodynamic Considerations**

The lack of correlation of 201TI defect size with number of stenotic coronary arteries (fig. 5) supports our clinical experience that massive infarctions are as common in patients with single-vessel coronary artery disease as small infarctions in patients with diffuse three-vessel disease.

The higher pulmonary capillary wedge pressures observed in patients with large 201TI defects (fig. 5) may be explained by reduced LV contractility and decreased LV compliance in the patients with larger infarcts. The linear fall in LV ejection fraction with increasing 201TI defect size (fig. 7) probably also reflects reduction in LV pump function as scar size increases.

**Quantitative Correlation of 201TI Scintigraphic Scar Size with Angiographic ACS**

Although our study showed a very good correlation between % ACS and 201TI defect size (fig. 9), we could argue that neither technique definitely assessed infarct size. For example, some 201TI perfusion defects might have represented hypoperfused, ischemic noninfarcted zones, and conversely, nontransmural infarctions might not have been represented as clear-cut 201TI defects. Similarly, angiographic wall motion abnormalities might have over- or underestimated the myocardial scar. In figure 9, for example, note that several patients had disproportionately large % ACS for their respective 201TI defect size. In these patients it could be postulated that asynergic, although perfused, myocardium accounted for the ACS, but failed to demonstrate a defect in 201TI uptake.

Another problem that influences the accuracy of comparing 201TI defects with angiographic wall motion abnormalities is that although both techniques optimally detect abnormalities in tangential views, the measurement of such abnormalities is perhaps in-
fluenced to different degrees by adjacent normal myocardium, which in the case of \(^{201}\)TI perfusion studies may "overlie" nonperfused zones on two-dimensional images, making recognition of the defect border difficult.

It is interesting to compare our findings in survivors of acute myocardial infarction to the findings of Wackers et al.\(^2\) in nonsurvivors of acute infarction (fig. 12). Wackers et al. showed a linear correlation between pathological infarct size and \(^{201}\)TI defect size in patients who died after large myocardial infarcts (\(^{201}\)TI, \(37 \pm 2\%\)), whereas our study showed correlation between angiographic ACS and \(^{201}\)TI defect size in patients surviving smaller myocardial infarcts (\(^{201}\)TI, \(11 \pm 2\%\)). The regression lines for the two studies are statistically inseparable,\(^3\) suggesting that over a wide range of infarct size (0-60\%), \(^{201}\)TI scintigraphy may quantify the extent of necrosis (fig. 12).

The well-documented problem of misidentifying apical \(^{201}\)TI defects caused by apical thinning\(^32,33\) provided only a 3\% incidence of false positive \(^{201}\)TI scans, i.e., an apical \(^{201}\)TI defect in the absence of angiographic asynergy. This agrees with reports of the frequency of false positive \(^{201}\)TI scintigrams.\(^11,13\) \(^{201}\)TI scintigraphy is insensitive (at least without computer enhancement) to scars \(\leq 6\%\). A similar insensitivity of \(^{201}\)TI scintigraphy to small defects has been noted in ischemic dogs.\(^34\)

Usefulness and Limitations of \(^{201}\)TI Myocardial Scintigraphy After Infarction

Resting \(^{201}\)TI myocardial scintigraphy complements the electrocardiogram in establishing the location of nonacute myocardial infarction, but is less sensitive than invasive angiographic studies. The weakness of

**Figure 11.** Comparison of size of total abnormally contracting segment (ACS) with size of the left anterior oblique (LAO) view thallium defect.

**Figure 12.** Comparison of size of thallium (TL) defect with angiographic (present study) and post-mortem (Wackers et al.\(^4\)) infarct size. A statistical comparison\(^9\) of the regression line from Wackers' data \((Y = (0.88 \pm 0.21)X + (-0.36 \pm 7.9)\)) and the line from the present data \((Y = (0.86 \pm 0.12)X + (4.41 \pm 1.8)\)) shows no significant difference between slopes or intercepts.
201Tl scintigraphy appears to be in delineating lateral infarctions and in interpreting apical-inferior defects. Very simple methods of measuring scintigraphic defect size from standard, noncomputerized images show a strong correlation with myocardial scar size as assessed independently by either angiography or postmortem studies. Although the correlation between 201Tl defect size and extent of angiographic asynergy is good (r = 0.80), the techniques of myocardial scintigraphy and contrast angiography are independent descriptors of cardiac physiology — one assesses myocardial perfusion, the other, LV wall motion. Thus, in individual patients there may be occasional discrepancies between the two techniques.

A limitation of the usefulness of 201Tl in detecting myocardial scars occurs in patients whose angiographic scar size is very small (ACS < 6%). Previous studies have shown that an ACS < 8% has no demonstrable effect on LV function. Thus, the inability of 201Tl scintigraphy to detect scars of this size is not a great disadvantage. A negative 201Tl myocardial scintigram does not exclude the possibility of a prior infarction, but makes the presence of a significant scar highly unlikely. Advances in computer technology that provide image enhancement by background subtraction and color displays may soon provide even greater accuracy in locating and sizing myocardial scars.

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Effect of Left Ventricular Akinesis on Cardiac Performance
Experimental Study Using a New Model

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SUMMARY The understanding of left ventricular failure and cardiogenic shock after myocardial infarction has been facilitated by a two-component model proposed by Swan et al. which views the left ventricle as consisting of a clearly defined infarcted portion and a normally functioning remainder. Although appealing, this model is difficult to substantiate experimentally. We describe a new experimental preparation in which the infarct is simulated by replacing part of the left ventricular wall with an inert patch of Dacron material.

In 18 dogs studied after replacing varying amounts of left ventricular contractile mass with noncontractile patches, the left ventricular end-diastolic pressure rose in proportion to the size of the patch. Contractility was unchanged, and overall left ventricular pump function was normal. These data support the conceptual model. The experimental preparation is applicable to other studies of left ventricular akinesis.

CLINICAL OBSERVATIONS of left ventricular pump function after myocardial infarction do not consistently support the concept of global left ventricular loss of function. Swan et al. proposed a more logical two-component model that makes the useful, though arbitrary, distinction between an area of abnormality and a remaining left ventricle that is functioning normally. The model recognizes that infarction alters function as a result of changes in both compliance and contractility, and has proved to be a valuable contribution to the understanding of left ventricular power failure over a wide range of clinical profiles. This model has been helpful despite recognition that clinical left ventricular abnormalities are usually poorly defined, nonhomogenous, and temporally variable.

Experimental support for this model has been scarce. Conventional methods of producing akinesis by infarction result in irregular areas of fibrous tissue interspersed with functioning myofibrils, which exhibit varying degrees of contractile function or distention during systole. These problems make accurate evaluation of the conceptual model extremely difficult.

To help clarify this problem, we developed an experimental preparation of left ventricular akinesis with a clearly delineated area that neither contracted nor expanded in ventricular systole. The results of these experiments show close correlation with the predictions of the conceptual model as applied to a noncompliant and noncontracting segment, and offer further insight into the effects of left ventricular akinesis after myocardial infarction. The model is readily applicable to more sophisticated studies of left ventricular function and diastolic compliance.

Materials and Methods
Experimental Model

Mongrel dogs weighing about 20 kg were anesthetized with sodium pentobarbital (Nembutal), 20–30 mg/kg body weight, and were intubated and ventilated. The femoral artery and vein were isolated, and the heart was exposed through a left thoracotomy.
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