Location and Size of Acute Transmural Myocardial Infarction Estimated from Thallium-201 Scintiscans

A Clinicopathological Study

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SUMMARY A clinicopathological study was performed in 23 patients who died of acute transmural myocardial infarction and who had been studied with thallium-201 during the acute phase. Twenty patients died within five days and three later than five days after scintigraphy. The scintigraphic location and the estimated size of infarction in vivo were correlated with postmortem findings. There was good agreement in 91% between scintigraphic and postmortem location of infarction and in 70% between the ECG and postmortem findings. The size of infarction as determined from computer-processed schematic drawings of postmortem slices of the heart correlated well with the size determined from processed schematic drawings of the scintiscans (r = 0.91 for anterior infarction, r = 0.97 for inferior infarction, r = 0.86 for anterior-inferior infarction). It is concluded that thallium-201 scintigraphy provided more precise location of infarction than the ECG and that the size of the scintigraphically abnormal area reflected the extent of necrotic myocardium.

SCINTIGRAPHIC VISUALIZATION OF MYOCARDIAL PERFUSION DEFECTS with thallium-201 has recently been shown to be a highly sensitive and reliable technique for the detection of acute myocardial infarction (AMI), especially in the first six hours after onset of symptoms. A good correlation was found between electrocardiographic and scintigraphic location of infarction. No correlation between data from thallium-201 scintigraphy in vivo and findings at postmortem examination have yet been described in patients with AMI. The purpose of this study was to compare location and size of infarction obtained from scintiscans in vivo with the actual postmortem findings. In addition, myocardial distribution studies were performed in ten patients who died within five days after injection of thallium-201.

Material and Methods

Of 550 patients with AMI, who were consecutively studied with thallium-201, 33 patients died from complications of their infarction; autopsies were performed in 23. The data from these 23 patients form the basis for this study. Clinical data are shown in table 1. All patients had AMI with typical history of chest pain and typical serial enzyme rises. Twenty patients had diagnostic ECG changes, one patient (14) had left bundle branch block. In two patients (9, 10) no diagnostic ECG changes were observed. In patient 9 this was probably caused by an old infarction. In patient 10 only ST-segment depression occurred in all leads. Electrocardiographic location of infarction was determined according to the criteria of the New York Heart Association. Ten patients died from cardiac rupture, 12 from pump failure, and one patient had sudden death on the tenth day after AMI. Nine patients died within 24 hours, 11 patients died between one and five days, two patients died on the tenth day and one patient died four weeks after thallium-201 scintigraphy. None of the patients in this study had β-blocking treatment at the time of scintigraphy. Three of the patients who died from pump failure (1, 13, 18) received digitalis at the time of scintigraphy.

Scintigraphy with thallium-201 (1.7–2.7 mCi i.v.) was performed as soon as possible after admission. In two of the 23 patients (22, 23) thallium-201 was injected, but scintigraphy was not performed because of the deteriorating condition of the patient. Analog images were obtained with an Ohio Nuclear ON-100 scintillation camera, using a low-energy high-resolution parallel-hole collimator. The energy window (20%) of the scintillation camera was set symmetrically over 75 keV. The scintiscans were obtained in three different views (anterior, left anterior oblique 45°, and left lateral). For each view 300k counts were accumulated. Unprocessed scintiscans were read independently and without knowledge of clinical data by at least two investigators as part of the daily routine. The location of perfusion defects was determined according to the diagram in figure 1. To determine the size of the perfusion defects schematic drawings of the scintigraphic images (21 patients) were made by one of the authors (F.W.). Areas with definite diminished thallium-201 activity, as compared to adjacent regions, were outlined. This was done retrospectively in patients 1–14, without knowledge of the patient’s name, cause of death, and postmortem findings. In patients 15–21 the delineation of the malperfused regions was done immediately after the scintigraphic study, without knowledge of clinical data.

In all patients the configuration of the entire left ventricle could be recognized on the unprocessed images. The size of the malperfused area was determined from the drawings in two ways: a naked eye estimation as a percentage of the total left ventricle (estimated scintigraphic size: ESC%) and by processing the drawings with a Quantimet 720 D TV

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Received October 28, 1976; revision accepted February 15, 1977.
scanner* and a Cyber 73 CDC computer.† The size of the scintigraphically abnormal area was expressed as the arithmetic mean of the percentages scintigraphically abnormal area of the left ventricle in three views (calculated scintigraphic size: CSC%).

At autopsy the site and extent of infarction was established after cutting the heart transversely into one centimeter thick slices. In eight cases (pts 14, 15, 17–22) Nitro Blue Tetrazolium (NBT) dehydrogenase staining was used for macroscopic demonstration of the infarcted myocardium in the slices. When considered necessary macroscopic inspection was supplemented by microscopic study. The extent of the infarction in the transverse slices was determined by three investigators (F.W., A.B., and A.V.) without knowledge of scintigraphic results. Schematic drawings were made of the infarction (including old scars, when present). They were processed by a Quantimet 720 D TV scanner and a Cyber 73 CDC computer. The infarcted region was expressed as a percentage of the total left ventricle (postmortem size: PM%).

Linear Pearson correlation coefficients and regression lines were determined for ESC%, CSC%, and PM%.

In five patients (14, 15, 19, 22, 23), who died within two days after injection of thallium-201, transverse slices of the heart were studied by scintigraphy for comparison with in vivo images. In ten patients (5, 10, 14–16, 18–20, 22, 23) who died within five days after injection of thallium-201, distribution of the radiopharmaceutical in the myocardium was measured and specific radioactivity of thallium-201 was determined as a fraction of administered thallium-201. Differences in mean values were analyzed by the Student’s t-test.

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14, with left bundle branch block on the ECG, the area of infarction was clearly visualized. In one of the two patients without diagnostic ECG changes (9) a clear anteroseptal defect was seen. In patient 10, who had only ST-segment changes on the ECG, a large area of diminished activity in the anteroseptal and lateral wall was observed.

At autopsy a recent infarction was macroscopically present or could be demonstrated by NBT staining in 22 patients. In two patients (4, 9), the microscopic study of the heart revealed a much larger infarction than was suspected from macroscopic inspection. No NBT staining was applied in these cases. The autopsy findings in patient 10 revealed a large circumferential infarction. This patient had concomitant severe aortic stenosis. In patient 20 definite infarction was present at the inferoposterior wall with subendocardial extension into the anterior part of the septum (fig. 7). Thallium-201 concentration studies (see below) offered evidence that the extension toward the septum represented a secondary enlargement of infarction after scintigraphy was performed. In seven patients (6–9, 12, 13, 17) scars of previous infarction were present. In two patients (6, 8) these old scars were not visualized on the scintiscans.

Site of Infarction

Correlation of the site of infarction as determined in vivo by ECG and thallium-201 scintigraphy and the site of infarction as determined at postmortem examination is shown in tables 2 and 3. In 91% of the cases there was good agreement between scintigraphic and postmortem location of infarction. Minor disagreement concerned the extent of the infarction. In one patient (13) there was complete disagreement. In this patient the scintiscans showed an extensive inferoposterior perfusion defect, which, at autopsy, was found to represent a previous infarction. A fresh infarction, involving one third of the septum, was not observed on the scintiscans. The location of infarction was similar in both the ECG and the postmortem findings in only 70% of the patients.

Size of Infarction

The correlation between size of scintigraphic perfusion defect and size of infarction at postmortem is shown for 19 patients in table 4 and figures 2 and 3. The infarct size from postmortem measurements includes fresh infarctions and old scars, since the total loss of viable myocardial tissue is of clinical relevance and no differentiation can be made by thallium-201 scintigraphy. Patients 4 and 9 were excluded from the correlation coefficient calculations. In both patients extensive infarction was present microscopically, but patchy necrosis was equally intermingled with normal myocardial fibers. Therefore definite delineation of the extent of infarction was impossible. The linear Pearson correlation coefficient between ESC% and CSC% was 0.74; between ESC% and PM% 0.67; and between CSC% and PM% 0.72. The linear regression lines (figs. 2 and 3) show that when the scintiscans were judged by naked eye (ESC%) the infarct size

Table 3. Localization of Infarction (N = 23 pts)

<table>
<thead>
<tr>
<th>Scintigraphy</th>
<th>ECG*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postmortem</td>
<td></td>
</tr>
<tr>
<td>AS</td>
<td>11</td>
</tr>
<tr>
<td>A</td>
<td>2</td>
</tr>
<tr>
<td>I</td>
<td>1</td>
</tr>
<tr>
<td>IP</td>
<td>1</td>
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<td>AS</td>
<td>7</td>
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<td>A</td>
<td>1</td>
</tr>
<tr>
<td>I</td>
<td>1</td>
</tr>
<tr>
<td>IP</td>
<td>8</td>
</tr>
</tbody>
</table>

*In 3 pts ECG localization was not possible.

Abbreviations: A = anterior; AS = anteroseptal; I = inferior; IP = inferoposterior.

Figure 2. Relationship between size of infarction determined on the basis of postmortem findings and size of scintigraphically abnormal area as estimated from thallium-201 scintiscans (ESC%) and the resulting regression line. The dotted lines indicate a shift of the regression line by 10% of the total left ventricle. N = 19; y = (1.04 ± 0.26)x – (9.11 ± 10.66); r = 0.69. Abbreviations: CHF = congestive heart failure; LV = left ventricle.

Figure 3. Relationship between size of infarction determined on the basis of postmortem findings and size of scintigraphically abnormal area as calculated by computer processing of schematic drawing of thallium-201 scintiscans (CSC%). The dotted lines indicate a shift of the regression line by 10% of the total left ventricle. N = 19; y = (0.88 ± 0.21)x – (0.36 ± 7.89); r = 0.72 for the whole group; r = 0.91 for anterior infarcts; r = 0.97 for inferior infarcts; r = 0.86 for anterior-inferior infarcts. Abbreviations: CHF = congestive heart failure; LV = left ventricle.
size was constantly overestimated by approximately 10% of the total left ventricle. For the whole group the size of infarction was estimated correctly within ± 10% of total left ventricle in three quarters of the patients. For the different infarction locations the correlation coefficient between CSC% and PM% was 0.91 for the six patients with anterior wall infarction, 0.97 for the six patients with inferior wall infarction and 0.86 for the seven patients with anterior-inferior infarction.

**Thallium-201 Distribution**

Figure 4 shows myocardial scintiscans of patient 15 obtained during life and of a transverse slice of the heart at postmortem. The site of absent activity of thallium-201 in the postmortem slice correlates well with the interpretation of the in vivo images. Also the other four cases in which postmortem scintigraphy of slices of the heart was performed showed excellent agreement with in vivo scintiscans. Figure 5 illustrates the distribution of thallium-201 in the same transverse slice as in figure 4. A significant difference (P < 0.05) exists between mean values of normal and infarcted myocardium. Table 5 gives the thallium-201 distribution in the myocardium of ten patients. The data are expressed as fraction of administered radioactivity per gram tissue × 10⁻⁶ (average values) at the time of death. In all patients (except patient 10 who had a large circumferential infarction) there was significant difference (P < 0.05) between thallium-201 activity in normal and in infarcted myocardium. The ratios of mean values for normal and infarcted myocardium ranged from 1.5–6.6:1.

Figure 6 shows a NBT-stained slice of the heart of patient 19 and the scintigraphic image of the adjacent slice. Measurements of thallium-201 distribution revealed a definite transitional zone (values 3.26–7.75) between normal and macroscopically infarcted myocardium. Without NBT staining this zone could not be recognized as infarcted, but microscopically there was evidence for infarction. The differences in mean values for the three zones were statistically significant (P < 0.05). Figure 7 shows a NBT-stained slice of the heart of patient 20. When scintigraphy was performed five days before death an inferoposterior defect was observed. On the third day after AMI the patient’s condition deteriorated, probably due to an extension of the infarction, as indicated by a new rise of enzyme levels. The ECG was not conclusive. At postmortem (six days after AMI) the NBT-stained slice demonstrated a posterolateral infarction with subendocardial extension into the anteroseptal region. A significant difference (P < 0.05) existed not only between the mean values of thallium-201 concentration in the posterolateral infarction and normal myocardium but also between the posterolateral infarction and the anteroseptal infarction. No significant differences in mean values of thallium-201 concentration were found between the anteroseptal infarction and the normal myocardium.

**Discussion**

The present study shows a correlation between the scintigraphic and postmortem findings with regard to both location and size of infarction. We considered this correlation to be reliable because 20 out of 23 patients in this study died within five days after onset of AMI and the scintigraphic

<table>
<thead>
<tr>
<th>Pt</th>
<th>ESC%</th>
<th>CSC%</th>
<th>PM%</th>
<th>Time of SC after AMI</th>
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<tbody>
<tr>
<td>1</td>
<td>40</td>
<td>36</td>
<td>29</td>
<td>10 days</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>33</td>
<td>29</td>
<td>2½ days</td>
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<td>3</td>
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<td>28</td>
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<td>3½ hr</td>
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<td>6 hr</td>
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<td>5 hr</td>
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<td>3½ hr</td>
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<td>28 hr</td>
</tr>
<tr>
<td>21</td>
<td>30</td>
<td>28</td>
<td>22</td>
<td>3 days</td>
</tr>
</tbody>
</table>

*Abbreviations: ESC% = estimated size of scintigraphic abnormal area as percentage of left ventricle; CSC% = computer calculated size of scintigraphic abnormal area as percentage of left ventricle; PM% = computer calculated size of infarction on basis of postmortem slices of the heart; SC = scintigraphy; AMI = acute myocardial infarction; t = delineation of infarction not possible; *only inferoposterolateral infarction measured.*

**Figure 4. Scintiscans with thallium-201 obtained in vivo and of a postmortem transverse slice of the heart of patient 15. In vivo diminished activity was noted at the anterior wall and probably small part of the septum (arrows). The image at postmortem shows close agreement in distribution of thallium-201. An evident region of absent activity is present at the anterior wall. Abbreviations: Ant = anterior; LAO = left anterior oblique 45°; LL = left lateral; a = anterior; p = posterior; r = right; l = left; PM = postmortem.**
study so that the heart damage at autopsy probably did not differ much from that present at the time of scintigraphy.

The site of infarction as predicted by scintigraphy showed only minor disagreement with the postmortem findings. In five of 12 patients with antero(septal) infarction the scintiscans indicated also lateral involvement, which was not observed at postmortem examination. In our experience the lateral wall is sometimes difficult to judge when only our three routine views are made. This is likely to be due to the individual variation in the position of the heart and differences in visualization of the mitral orifice.

The site of infarction is of prognostic significance. Thallium-201 scintigraphy provides an additional diagnostic aid in recognizing patients with AMI at high risk, especially in patients with abnormal ventricular activation pattern (i.e., left bundle branch block, pacemaker rhythm, WPW-syndrome), where ECG diagnosis and localization of infarction is difficult. Although the number of patients in this study is relatively small, our data suggest that thallium-201 scintigraphy might provide more precise localization than the ECG. In a previous study comparing electrocardiographic and scintigraphic localization of infarction, we found that three quarters of electrocardiographically located inferior infarctions also extended into the posterior wall on the scintigraphic images.

Estimation of infarct size during the acute phase of AMI is of crucial importance when therapeutic efforts to reduce the extent of jeopardized myocardium are being considered. Initially we doubted that accurate estimations of the size of malperfused or infarcted myocardium could be made reliably from thallium-201 scintiscans. Our skepticism was based on the following considerations.

1) It seemed difficult to relate two-dimensional projections with a three-dimensional shape. Moreover, the heart cannot be viewed from all angles due to technical difficulties such as patient positioning and photon absorption.

2) The static images represent the integrated radioactivity distribution of a moving organ.

3) Changes in size of scintigraphic abnormalities occur during the first 24 hours after AMI. The situation is usually stabilized only after 24 hours.

4) The schematic drawings were subjective interpretations of the outlines of scintigraphic abnormal areas. Usually there was no sharp demarcation.

5) No differentiation was made between areas of absent activity and areas of diminished activity, whereas they most probably represent different anatomical situations.

6) Macroscopic recognition of infarction from postmortem specimen demands a time interval of approximately 24 hours between onset of AMI and death. However, NBT staining permitted earlier recognition.

**Table 5. Distribution* of Thallium-201 in Myocardium (N = 10)**

<table>
<thead>
<tr>
<th>Death after inj. Tl-201</th>
<th>#58</th>
<th>#19</th>
<th>#14</th>
<th>#23</th>
<th>#10</th>
<th>#16</th>
<th>#22</th>
<th>#15</th>
<th>#18</th>
<th>#20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death after inj. Tl-201</td>
<td>13/2 h</td>
<td>13/2 h</td>
<td>3 h</td>
<td>31/2 h</td>
<td>81/2 h</td>
<td>14 h</td>
<td>2 d</td>
<td>2 d</td>
<td>4 d</td>
<td>5 d</td>
</tr>
<tr>
<td>Normal myocardium</td>
<td>8.05</td>
<td>12.07</td>
<td>15.6</td>
<td>1.0</td>
<td>7.7</td>
<td>6.1</td>
<td>3.1</td>
<td>0.64</td>
<td>0.43</td>
<td>0.66</td>
</tr>
<tr>
<td>Infarction</td>
<td>5.5</td>
<td>1.82</td>
<td>9.9</td>
<td>0.63</td>
<td>6.8</td>
<td>3.2</td>
<td>1.8</td>
<td>0.30</td>
<td>0.25</td>
<td>0.43</td>
</tr>
<tr>
<td>Normal: infarction ratio</td>
<td>1.5</td>
<td>6.6</td>
<td>1.6</td>
<td>1.6</td>
<td>1.1</td>
<td>1.9</td>
<td>1.7</td>
<td>2.1</td>
<td>1.7</td>
<td>1.5</td>
</tr>
</tbody>
</table>

*Data in fraction of administered activity per gram tissue x 10^4 (average values) at the time of death.

†Patient numbers.

‡Large circumferential infarction.

§only relative measurements.
7) Delineation of the extent of infarcted regions from postmortem specimen was an approximation. Here again NBT staining was helpful.

In spite of these limitations, scintigraphically estimated size of infarction and postmortem findings correlated well and the relationship was stronger when the location of the infarction was included. A constant overestimation, by approximately 10%, was found in evaluation of the scintiscans with the naked eye. When the schematic drawings were processed by the computer, the regression line approached the line of identity.

In this study the correlation was not influenced by time of scintigraphy after infarction or time interval between scintigraphy and death. This seems in contradiction with our previous observations that, especially during the first hours after onset of AMI, thallium-201 scintiscans overestimate the actual size of infarction by visualizing surrounding ischemia. These observations of decreasing size of defects were most impressive in small infarctions. In the present study all patients had moderate to large transmural infarctions and all patients died from complications of their infarctions. Therefore, the results in this study do not necessarily apply to the general population of patients with AMI. Although a correlation was found between scintigraphic and postmortem studies, the scintigraphic abnormal area observed in vivo may not represent only infarcted tissue at the time of scintigraphy. The infarction could have extended between scintigraphy and death. This is less likely in patients who died from cardiac rupture than in patients who died from pump failure, in whom secondary extension of infarction may occur. Indeed two of the patients with cardiac rupture and two of the patients with pump failure in our study showed overestimation (indicating ischemia) and underestimation (indicating extension), respectively.

We believe that estimation of the extent of ischemic or infarcted myocardium, without differentiation between the two, can be done correctly from analog thallium-201 scintiscans. The differentiation between ischemia and actual necrosis in the setting of AMI remains of direct clinical importance because the jeopardized myocardium may then be identified and quantified. Recently Pohost et al. suggested that serial images after a single dose of thallium-201 could distinguish between transient ischemia and myocardial infarction. Further studies are needed to evaluate the usefulness of this procedure in the setting of AMI.

**Figure 6.** Nitro Blue Tetrazoleum (NBT) stained transverse slice of the heart of patient 19 and a scintiscan of the same slice. Close agreement exists between thallium-201 distribution on the scintiscan and pattern of NBT staining. Measurements of thallium-201 distribution (expressed as fraction of administered activity per gram tissue × 10⁶) revealed three zones (values 1.08–2.73; 3.26–6.75, and 10.90–12.96). Significant differences (P < 0.05) existed between the mean value of each of the three zones. Values for thallium-201 distribution in the right ventricle are significantly (P < 0.05) lower than in the normal myocardium of the left ventricle. Abbreviation: A = anterior.

**Figure 7.** Thallium-201 distribution in a Nitro Blue Tetrazoleum stained transverse slice of the heart of patient 20. The normal myocardium is darkly stained; the infarcted myocardium remained unstained. Significant differences (P < 0.05) existed between mean values of thallium-201 distribution in the two sites of infarction. In view of the thallium-201 distribution it is conceivable that the septal infarction represents a secondary enlargement after scintigraphy was performed. Data are expressed as fraction of administered activity per gram tissue × 10⁶. Abbreviation: A = anterior.
Planimetric estimation of the size of infarction with $^{99m}$Tc-pyrophosphate in animal experiments has been reported to correlate exceedingly well with histological infarct size in some,12, 13 but not all studies.14, 15 The clinical reports on the estimation of the extent of infarction, using $^{99m}$Tc-pyrophosphate and other positive imaging agents, are still preliminary and the findings differ.16-20 It seems that the correlation is strong for anterior infarctions but is not as strong for inferior infarctions. Using negative imaging agents, $^{13}$Cs and $^{67}$Cs, Gustin et al.21 and Burguet et al.22 found a close correlation between the extent of the scintigraphic defect and the clinical course of patients with AMI. Zaret et al.23 demonstrated good correlation between $^{40}$K images of patients with AMI and the site and extent of regional left ventricular dysfunction.

In this study scintigraphic images obtained in vivo were compared to in vitro scintigraphy of transverse slices of the heart. Thallium-201 distribution reflected the extent of infarction as demonstrated with NBT staining. In one of the five patients a NBT-stained and low thallium-201 activity zone was present (fig. 6). This zone was partly faintly or partly normally NBT stained at the lateral wall and partly unstained or partly normally stained in the septum. Microscopically the unstained regions revealed fresh necrosis, but in the normally NBT stained areas patchy necrosis was intermingled with normal myocardial fibers. One could explain this finding by assuming that severely ischemic myocardium at the time of scintigraphy progressed into infarction. In view of the short time interval (1½ hours) between scintigraphy and death in this patient, the possibility exists that no sharp demarcation in thallium-201 uptake at the border of an infarction occurs. In experimental infarction in dogs Buja et al.24 observed a similar thallium-201 accumulation in the outer periphery of an infarcted area. Since thallium-201 concentrates in the myocardium primarily according to regional perfusion25 it was surprising that thallium-201 activity was measurable in the necrotic tissue. At autopsy the ratios of activity per gram tissue in normal myocardium versus infarcted myocardium ranged from 1.5-6.6:1. Due to the time interval between scintigraphy and death we suppose that these relatively low ratios may not reflect the actual ratios at the time of scintigraphy. Thallium-201 may have entered the necrotic tissue by passive diffusion or by subsequently developed collateral flow. It is also possible that passive diffusion after death accounted for this finding. Autopsy was in this study never performed earlier than 10 hours after death.

From this preliminary study we conclude that thallium-201 scintigraphy is a valuable tool for precise localization of malperfused or infarcted myocardium. Scintigraphy was found to be superior for this purpose to the ECG. Since in this study the size of the scintigraphically abnormal area reflected closely the extent of infarction, we speculate that in the clinical setting estimation of size of malperfused or infarcted myocardium can be done from analog scintiscans.

Further prospective clinical studies are needed to establish the clinical relevance of our findings.

Acknowledgment

We express our thanks to C. L. Alons, M.D. (Department of Pathology, Quantitative Pathology, Free University, Amsterdam, The Netherlands) for quantitating and processing our morphological data.

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Location and size of acute transmural myocardial infarction estimated from thallium-201 scintiscans. A clinicopathological study.

*Circulation*, 1977;56:72-78
doi: 10.1161/01.CIR.56.1.72

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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