Quantification of myocardial infarct size by thallium-201 single-photon emission computed tomography: experimental validation in the dog

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ABSTRACT  To evaluate the potential advantages of thallium-201 (201T1) single-photon emission computerized tomography (SPECT) to assess myocardial infarct size in the experimental animal, six normal dogs and 14 dogs with 6 to 8 hr closed-chest coronary occlusion (eight left anterior descending and six left circumflex) were studied. Ten minutes after intravenous administration of 2 mCi of 201T1, 30 projections were obtained over 180 degrees. The dogs were killed and their hearts sliced and stained by triphenyl tetrAZolium chloride (TTC). Pathologic infarct size was calculated for each slice and for the entire left ventricular myocardium as percent weight. Tomograms were quantified by automatically generating maximum-count circumferential profiles, which were compared with normal limit profiles derived from the six normal dogs. Tomographic infarct size was defined as the percentage of circumferential points falling below normal for each tomogram. SPECT and TTC infarct size on 71 slices correlated highly (mean ± SD 27.9 ± 23.4% and 26.7 ± 25.3%, respectively; r = .93, p < .001, SEE = 9.4%). To determine SPECT infarct size as percent total left ventricular myocardial weight, infarct sizes from each slice were added to one another after each was multiplied by a coefficient that reflected the contribution of that slice to the total left ventricular weight. SPECT and TTC infarct size for the entire left ventricle correlated closely (mean ± SD 20.5 ± 7.6% and 19.3 ± 8.3%, respectively; r = .86, p < .001, SEE = 4.5%). It is concluded that 201T1 SPECT is a valid method for the noninvasive assessment of experimental myocardial infarct size.

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PROGNOSIS in acute myocardial infarction has been shown to be related to the extent of the necrotic myocardium.1-5 Several noninvasive, nonimaging,6-8 and imaging5, 8-21 methods have been developed for measurement of infarct size. With planar imaging or limited-angle tomography,8-13 thallium-201 (201T1) myocardial perfusion and technetium-99m–pyrophosphate infarct-avid scintigraphy are suboptimal for quantitation of infarct size because of overlap of normal and abnormal regions. Single-photon emission computerized rotational tomography (SPECT) has a theoretical advantage over planar imaging and limited-angle tomography for the accurate assessment of infarct size because of 180 or 360 degree angular sampling of the myocardium, which results in reduced overlap and improved contrast of the images.22, 23 Although quantification of infarct size with SPECT using 201T1 has been shown to be feasible,14-18 these previous studies used subjective measurements or computerized planimetric approaches that were dependent on detection of myocardial edges of the normal and nonvisualized regions (with severe perfusion defects).

In this study we sought (1) to develop an objective, computerized method for quantification of infarct size using maximum counts circumferential profile analysis, independent of edge detection, and (2) to validate the method in a living canine closed-chest preparation.
Methods

Experimental protocol. Twenty adult mongrel dogs (15.5 to 32 kg) were studied, six controls and 14 with induced acute myocardial infarction. In all dogs, anesthesia was induced with intravenous administration of sodium pentobarbital (25 mg/kg) and was maintained with supplemental intravenous doses thereafter. No intubation or artificial ventilation was needed except in two dogs that had large infarctions. In the 14 dogs assigned to the infarction study, the protocol was as follows: Femoral carotid artery was exposed and a modified No. 7F Judkins angiographic catheter with an indwelling inflatable balloon-tipped No. 2F Fogarty catheter was introduced into the carotid artery. The angiographic catheter was advanced under fluoroscopic control into the ostium of the left anterior descending coronary artery (LAD) in eight dogs or the left circumflex artery (LCX) in six dogs. The balloon-tipped catheter was then passed through the angiographic catheter and positioned in the coronary artery at different levels from the ostium. The coronary artery was then occluded by inflating the balloon with contrast material. The occlusion was maintained until the dog was killed. Imaging started 6 to 8 hr after the initiation of occlusion. The imaging protocol was the same for the control dogs and the dogs with infarction: The animals were put on the imaging table in the right anterior oblique position. Two milli- curies of $^{201}$TI was injected intravenously and tomography was performed in vivo 10 min after injection. A large-field-of-view camera (Siemens Rota for 18 dogs and Siemens Orbiter for two dogs) equipped with 75 photomultiplier tubes, a $\frac{1}{4}$ inch thick NaI (TI) crystal, and an all-purpose parallel-hole collimator was used. Twenty percent and 10% energy windows were used, positioned on the 68 to 80 keV and 160 keV photopeaks, respectively. The collimator rotated 180 degrees around the dog’s chest from the left lateral to the right lateral position, obtaining 30 projections spaced by 6 degrees. Each projection took 30 sec to acquire. Three-hour delayed images were also acquired in five of six controls and in 10 of 14 dogs with infarction. The data were stored in a $64 \times 64 \times 16$ bit matrix. The extrinsic full width at half maximum measured with a line source at 8 inches was 18 mm for $^{201}$TI. The pixel size was 0.434 \times 0.434 cm.

Immediately after imaging, all 20 dogs were killed by intravenous injection of potassium chloride and the hearts were excised and cut perpendicular to the long axis of the left ventricle. The hearts were cut into 16 mm thick slices that were placed into 16 ml tubes with 5 ml of 2% buffered formalin. Two slices were taken from each ventricle, one from the left ventricular apex and the other around the midventricular level. All slices were then incubated in triphenyl tetrazolium chloride (TTC). TTC was injected as a milliliter/kg solution and left in place for 30 min. The pixel size was 0.208 \times 0.208 mm.

Tomographic data processing. Each of the 30 projections was corrected for nonuniformity with a 30 million count image of a cobalt-57 source and adjusted for center of rotation. Projections were filtered-back-projected by a low-resolution Hanning filter to obtain transaxial tomograms. These were nine-point smoothed (4-2-1 weighting) and reoriented into vertical long-axis and short-axis tomograms, which were respectively parallel to and perpendicular to the long-axis of the left ventricle. No attenuation or scatter correction was used. All tomograms were one pixel (0.43 cm) thick.

The tomograms were analyzed by a method described previously. Briefly, the operator selected the short-axis and vertical long-axis cuts that encompassed the left ventricular cavity. After determination of the center of the left ventricle, circumferential profiles were generated automatically by performing a radial search for maximum value on 60 equidistant points over the entire circumference of each tomographic cut (figure 1). The maximal values were then plotted for each angle as a percentage of the maximum value of the profile. For proper comparison of the different profile to the normal limit profile, an anatomic landmark was defined at the inferior junction of the two ventricles for the short-axis cuts and at the midapical point for the vertical long-axis cuts. The myocardial activity was analyzed from the short-axis circumferential profiles for all regions, except the apex, which was analyzed from the apical portions of the vertical long-axis profiles.

Quantification of infarct size on individual tomograms

Generation of a normal data base. A reference normal data base was established for initial distribution of $^{201}$TI based on the data from the six normal dogs. The short-axis normal profiles

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**FIGURE 1.** Diagram showing various steps of quantitative analysis of myocardial activity in a short-axis tomogram: The center of the left ventricle is manually assigned (A) and 60 radii 6 degrees apart are generated to a predetermined length. The computer searches the maximum count value along each radius (black dots) (B) until 360 degrees are covered (C). The maximum count values are then plotted (D) against angular location on the myocardial periphery as a percent maximum count value in a given tomogram. An anatomic landmark is determined at the inferior junction of the two ventricles (X in C) for proper matching of each profile to the normal limit profile.
were grouped into five regions extending from apex to base and the mean myocardial $^{201}$Tl distribution was determined for each region. Similarly, five mean normal profiles were generated in the vertical long-axis orientation.

**Determination of infarct size.** The profiles of the 14 dogs with infarction were compared with the corresponding mean normal profiles (figure 2). Since the boundary between the normally and abnormally perfused myocardium was not sharply demarcated on the $^{201}$Tl tomograms, definition of defect edge as the points at which the experimental profile fell below the mean normal profile resulted in overestimation of defect size (figure 2). To circumvent this problem, different thresholds below the mean normal profile were evaluated in 15 short-axis tomograms ("calibration tomograms") to determine the best definition for the edge of the abnormal zone. These calibration tomograms were selected so that among the slices there were five normal slices (four from the normals dogs and one from the dogs with infarction) and 10 slices with different-sized regions with transmural necrosis. The extent of perfusion defect on these tomograms using various thresholds below the mean normal profile was compared with the TTC pathologic infarct size. Similarly, different thresholds were applied to the vertical long-axis profiles of three dogs (two with different-sized infarcts in the apex and one normal dog). Since the optimal normal limit thresholds were determined in the calibration tomograms, the remaining 94 tomograms were prospectively quantified with the adopted threshold. For each heart, all short-axis tomograms were matched to the pathologic slices corresponding to the same depth along the long axis of the left ventricle.

**Determination of infarct size for the entire left ventricular myocardium.** Two methods were used to determine total left ventricular myocardial infarct size from infarct size on individual tomograms. In method 1, it was assumed that each slice contributed equally to the total left ventricular mass, and thus the number of abnormal profile points of all slices were added up and then were divided by the total number of profile points, according to the formula:

$$\% IS = \frac{AP_{\text{apex}} + AP_1 + AP_2 + \ldots}{TP_{\text{apex}} + 60 \times nsa}$$

where IS = infarct size, AP = number of abnormal points for the apical profile ($AP_{\text{apex}}$) and short-axis profiles from apex to base ($AP_1$, $AP_2$, ...), $TP_{\text{apex}}$ = total number of apical profile points, and nsa = number of short-axis slices, and 60 = total number of profile points in the short-axis slices. This method will be referred to as the incorrect method.

In method 2, the difference in mass of the myocardial slices from apex to base was taken into consideration. As shown in figure 3, when the left ventricle is sliced at equidistant intervals perpendicular to its long axis, slice mass differs from apex to base because of varying slice radius and myocardial wall thickness. Because the extent of infarction is given as a percentage with the maximum counts circumferential profile analysis, it is necessary to account for myocardial slice mass before summation of individual slice infarct size. Thus it was postulated that each myocardial slice would represent a certain fraction of the left ventricular mass according to its fractional distance along the long axis of the left ventricle. Therefore the number of abnormal profile points on each tomogram ($AP_{\text{apex}}$, $AP_1$, $AP_2$, ...) was multiplied by a coefficient, K, reflecting the contribution of each myocardial slice to the total left ventricular mass and then summed. This sum was then divided by the total number of left ventricular profile points corrected for K according to the formula:

$$\% IS = \frac{AP_{\text{apex}}(K_{\text{apex}}) + AP_1(K_1) + AP_2(K_2) + \ldots}{TP_{\text{apex}}(K_{\text{apex}}) + 60(K_1 + K_2 + \ldots)}$$

where AP = number of abnormal points in the apex ($AP_{\text{apex}}$) and short-axis cuts numbered from the most apical from the

**FIGURE 2.** Comparison of pathologic (A and B) and tomographic (C and D) infarct size in a myocardial slice with anterior wall infarction. The pale area on the TTC-stained slice (A) represents the region with myocardial necrosis. By planimetry, the infarct region was shown to occupy 18% of the slice area (B). On the tomographic slice (C), the border of the infarcted region was not discrete (arrows). When the circumferential profile generated from the tomogram (D) was compared with a proper threshold, the tomographic infarct size was measured to be 22%.
The comparison of the infarct size linear regressions for the uncorrected and corrected methods was made with F tests as described by Smith and Choi. All values were expressed as the mean ± SD. The alpha level of significance was .05. All p values were two sided.

Results

Pathologic infarct size. The mean heart weight was 82 ± 9 g (range 68 to 95) in the normal dogs and 93 ± 27 g in the dogs with infarction. The infarct weights ranged from 7 to 40 g, representing 8% to 31% of the left ventricle. The mean infarct weight was 21 ± 10 g in dogs with LAD occlusion and 16 ± 7 g in those with LCX occlusions. There were 107 slices in the dogs with infarction, 83 of which were found to contain infarcted tissue by TTC staining. Necrosis was transmural in 51 of 83 slices (61%), involved more than 50% of the myocardial wall thickness in 24 of 83 slices (29%), and involved less than 50% of the wall thickness in the remaining eight of 83 slices (10%).

In all 14 dogs, the infarction involved at least 50% of the myocardial wall thickness in at least two-thirds of the infarcted slices and at least one slice per heart had a totally transmural infarct.

Mean normal profile. Figure 4 illustrates the short-axis mean normal profiles for the five different anatomic regions along the long axis of the left ventricle. The pattern of initial myocardial distribution of $^{201}$TI was similar for the first three regions; the region extending from 90 to 270 degrees, corresponding to the interventricular septum, showed relatively lower activity than the remaining portions of the left ventricular circumference, presumably because of photon attenuation by the right ventricle. In region IV, which was closer to the left ventricular base, the arc between 45 and 180 degrees had relatively lower counts, presumably because of the increased distance of this region from the detector in frontal views and increased attenuation from the left ventricle. In the basal region (region V), the reduction of $^{201}$TI activity in the 45 to 180 degree arc became even more pronounced.

Comparison of different thresholds for infarct sizing in the pilot calibration sample. Different thresholds below the normal profile (40%, 45%, 55%, 60%, and 65%) were tested to calculate infarct size in the short-axis and vertical long-axis tomograms. The best thresholds were 60% of the mean for the short-axis tomograms with LAD necrosis, 45% of the mean for the short-axis tomograms with LCX necrosis, and 40% of the mean normal profile for the apical portion of the vertical long-axis profiles for both LAD and LCX infarction. The LAD territory was considered to include the septum and anterior wall on all the short-axis slices. How-

FIGURE 3. Varying masses of myocardial slices from apex to base of the left ventricle. Each slice mass is dependent on the mean radius (R) and thickness (t) of the slice.
ever, the proportion of the anterior wall vascularized by the LAD progressively narrowed from apex to base, representing 33% of the short-axis periphery in region I, 25% in region II, 23% in region III, and 22% in regions IV and V. The circumflex territory was considered to include the rest of the left ventricular myocardium (inferolateral region) on the short-axis slices. With these thresholds, the correlation coefficient was .95 (p < .001) for the 18 calibration slices and .91 (p < .001) when only the 12 calibration slices with infarction were considered.

Figure 2 illustrates an example of correlation between pathologic and tomographic infarct size in an individual slice with LAD necrosis. On the pathologic slice (A), the infarct size (pale area) was measured by planimetry to be 18% (B). On the circumferential profile (D) generated from the tomogram (C), the infarct size was measured to be 22% when the adopted threshold of 60% was used.

**Relationship between TTC and SPECT estimates of infarct size in individual slices.** The optimum thresholds derived from the 18 calibration tomograms were applied prospectively to the remaining 94 tomograms. When all 94 prospective slices (whether or not they contained infarction) of the dogs with infarction were considered together, the correlation coefficient was .94 (p < .001) and linear regression yielded an SEE of 8.2%. When only the 71 prospective slices with infarction were considered, the correlation coefficient was .93 (p < .001) and the SEE was 9.4% (figure 5). The regression line (slice tomographic infarct size vs pathologic infarct size) did not differ significantly from the line of identity. All 24 normal slices (no infarct by TTC) of the dogs with infarction as well as all slices of the control dogs were also normal by quantitative analysis.

When the slices were divided into three groups with regard to the type of necrosis — transmural (group 1), subendocardial involving more than 50% of the myocardial wall thickness (group 2), and subendocardial involving less than 50% of the myocardial wall thickness (group 3) (table 1) — the correlation between tomographic and pathologic infarct size was excellent for group 1 (mean ± SD 38.1 ± 27.5% and 35.2 ± 28.3% for pathology [P] and tomography [T], respectively; T = −1.6 + .97P, r = .94, p < .001, SEE = 9.6%, ratio SEE/mean slice infarct size = .27); the correlation was good in group 2 (mean ± SD 20.7 ± 11.0% for P and 22.4 ± 15.8% for T; T = −3.2 + 1.24P, r = .86, p < .001, SEE = 8.3%, ratio SEE/mean infarct size = .37), and there was no correlation in group 3 (mean ± SD 5.7 ± 3.1% for P and 3.4 ± 5.7% for T; r = .44, p = .14, SEE = 5.4%, ratio SEE/mean infarct size = 1.6).

In 65 of 71 slices (92%), the absolute error between pathologic and tomographic infarct size was 0.5 g or
FIGURE 5. Relationship between pathologic and tomographic infarct size in the 71 prospective slices with infarction.

less, regardless of the slice infarct weight. In the remaining six (8%), this absolute error was greater than 0.5 g and ranged from 0.6 to 1.2 g.

Dependence of myocardial slice mass on its distance from the apex. A relationship was observed between slice fractional distance and slice mass in all five normal dogs in that the mass of each slice increased with increasing distance from the apex with a plateau or reduction at the region of the base (figure 6). A second-degree equation best fit the data for all five dogs. The

FIGURE 6. Dependence of myocardial slice weight on its distance from the left ventricular apex in the five control dogs. A second-degree equation best described this relationship.

TABLE 1
Pathologic (P) and tomographic (T) infarct size in 107 slices divided into four groups according to the type of myocardial infarction

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Group 2: Infarction involving >50% of myocardial wall thickness (n = 24)

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Group 3: Infarction involving <50% of myocardial wall thickness

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*aAll 24 slices containing no infarction by pathology (group 4) were also normal by tomography.

individual equations for each dog were then compared to determine whether one equation would suffice to describe the relationship between slice fractional distance and slice mass for all five dogs. The five equations did not differ significantly so a pooled second-degree polynomial equation was fit to all of the data as

\[ K = -1.2 + 2.9x - .023x^2 \]  

(figure 6).

Total myocardial infarct size in vivo: tomography vs pathology. There was a high correlation between the pathologic and tomographic infarct size assessed by the uncorrected method (r = .82, p < .001, SEE = 6.3%)
(NO CORRECTION FOR MYOCARDIAL SLICE WEIGHT)

![Graph showing the relationship between the tomographic and pathologic infarct size expressed as a percentage of the entire left ventricular weight by the uncorrected method.]

(CORRECTION FOR MYOCARDIAL SLICE WEIGHT)

![Graph showing the relationship between the tomographic and pathologic infarct size expressed as a percentage of the entire left ventricular weight by the corrected method.]

FIGURE 7. Relationship between the tomographic and pathologic infarct size expressed as a percentage of the entire left ventricular weight by the uncorrected method.

FIGURE 8. Relationship between the tomographic and pathologic infarct size expressed as a percentage of the entire left ventricular weight by the corrected method.

(figure 7). With this method, however, all tomographic infarct sizes in the eight dogs with LAD occlusion were above the line of identity, signifying overestimation by tomography, and four of six with LCX necrosis were underestimated by tomography. The regression line had a slope greater (1.16) but insignificantly different from the line of identity (p = .29).

When correction for differing myocardial slice mass was done, the correlation between the pathologic infarct size and tomographic infarct size was improved (r = .86, p < .001, SEE = 4.5%) and the slope was closer to unity (.94) (figure 8). Figure 9 shows the effect of slice mass correction on the relationship between pathologic and tomographic infarct size. In all eight dogs with LAD necrosis, the correction reduced the tomographic infarct size and in six of eight this correction resulted in a closer one-to-one relationship between TTC and SPECT infarct size. The LCX infarct size increased slightly in five of six dogs with LCX infarction and in four of these five, this correction partially compensated for the underestimation by SPECT. Overall, with correction, 11 of 14 data points came closer to the line of identity.

Table 2 summarizes the variables of the regression line for the two methods. The mean percent infarct size was (mean ± SD) 20.5 ± 7.6% for pathology, 23.1 ± 10.7% for the uncorrected method, and 19.3 ± 8.3% for the corrected method. The correlation coefficient was better for the corrected than for the uncorrected method (.86 vs .82). Even though none of the regression lines significantly differed from the line of identity, the slope was slightly closer to the line of identity with correction (slope = .94, intercept = .1, and p = .56 for comparison of the regression line with the line of identity) than without correction (slope = 1.16, intercept = .8, p = .29). The SEE, the relative bias, and the relative precision were smaller with correction (4.5%, −1.2%, and 4.3%, respectively) than without correction (6.3%, 2.6%, and 6.2%).

Discussion

In this study, we attempted to quantify myocardial infarct size by 201Tl tomography in a living canine preparation with 180 degree acquisition without scatter or attenuation correction. Quantification of myocardial infarct size with SPECT and 99mTc-pyrophosphate has been shown to be feasible.5, 14, 19 Although this approach is useful in the setting of acute myocardial infarction, there is a delay of several hours to days before 99mTc-pyrophosphate is optimally taken up by the acutely necrotic myocardium and the study usually becomes negative several days to weeks after the event. On the contrary, 201Tl scintigraphy can be applied to estimate the infarct size at the onset of acute myocardial infarction, and the study usually remains positive thereafter. Thus the two techniques are com-
FIGURE 9. Effect of tomographic data correction on the relationship between the pathologic and tomographic infarct size. Correction consistently reduced the infarct size in the eight dogs with LAD occlusion and slightly increased the infarct size in five of six dogs with LCX infarction. With correction, the infarct size was better estimated in 11 of 14 dogs (values closer to the line of identity).

to human subjects and showed a good correlation between the tomographic infarct size and the enzyme-derived estimate. Caldwell et al. measured the relative myocardial perfusion defect size in dogs, using 180 degree acquisition without scatter or attenuation correction, and found a good correlation with the measurement in vitro. In all of these studies, endocardial and epicardial myocardial borders as well as the edge of infarcted area were assigned manually or by a computerized edge-detection algorithm.

In this study, a previously described quantitative technique using maximum counts circumferential profile analysis was applied. The technique requires the intervention of the operator only for the selection of tomograms and for the assignment of the center of the left ventricle, apex, and the interventricular junction and has been shown to be reproducible. We chose to acquire 30 projections over 180 degrees rather than 360 degrees to obtain higher spatial and contrast resolution. The tomograms were reconstructed without correction for scatter or attenuation because methods for correction of the variable attenuation of the ⁴®Tl activity in the thorax have not been optimized. Although scatter and attenuation may be partly responsible for the lack of definition of the edges of the perfusion defect on tomographic images and on the profiles, establishment of normal ⁴®Tl distribution profiles in various regions of the left ventricle and comparison of the experimental profiles to these normal profiles partly compensated for this problem. This compensation is evidenced by the fact that despite the appearance in normal tomograms of reduced counts in the more basal portions of the septal and inferior walls, none of the tomograms showing noninfarcted tissue was considered abnormal after their circumferential profiles were compared with the respective lower limits of normal.

To avoid partial volume effect, only the short-axis slices containing the left ventricular cavity were analyzed, and the apex was analyzed from the vertical long-axis cuts. This aspect of our approach to assess the left ventricular apex is particularly important in cases with necrosis in the LAD distribution, which frequently involves the apex, and may go undetected if apical partial volume correction is not done.

A good correlation was noted between the tomographic and pathologic infarct size in individual slices. Although the SEE was 8.2%, the absolute error of the measurement was small (<0.5 g) regardless of the size of the infarction. We observed that subendocardial infarction that involved less than 50% of the myocardial wall thickness and less than 10% of the myocardial periphery were either missed or underestimated by to-

<table>
<thead>
<tr>
<th>Variable</th>
<th>Uncorrected</th>
<th>Corrected</th>
<th>Pathology (TTC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean IS ± SD (%)</td>
<td>23.1 ± 10.7</td>
<td>19.3 ± 8.3</td>
<td>20.5 ± 7.6</td>
</tr>
<tr>
<td>r</td>
<td>.82</td>
<td>.86</td>
<td></td>
</tr>
<tr>
<td>P₁ (&lt;)</td>
<td>.001</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>- .8</td>
<td>.1</td>
<td></td>
</tr>
<tr>
<td>Slope</td>
<td>1.16</td>
<td>.94</td>
<td></td>
</tr>
<tr>
<td>Comparison with line of identity (P₂)</td>
<td>.29</td>
<td>.56</td>
<td></td>
</tr>
<tr>
<td>SEE (%)</td>
<td>6.3</td>
<td>4.5</td>
<td></td>
</tr>
<tr>
<td>Relative bias (%)</td>
<td>2.6</td>
<td>-1.2</td>
<td></td>
</tr>
<tr>
<td>Relative precision (%)</td>
<td>6.2</td>
<td>4.3</td>
<td></td>
</tr>
</tbody>
</table>

r = correlation coefficient, P₁ = value in testing that the true correlation coefficient is equal to 0; P₂ = p value for the comparison of the regression line with the line of identity; IS = infarct size.
tomography (table 1, group 3). This is believed to be mainly caused by the system spatial resolution, since there is a significant reduction in the reconstructed voxel value for objects smaller than twice the full width at half maximum. It is likely that this dependence on the object size would tend to mask nontransmural perfusion or small perfusion defects.

We observed that when the uncorrected method was used to determine left ventricular infarct size, the LAD infarcts were generally overestimated while LCX infarcts were underestimated. This was due to the fact that this method assumes that all slices contribute to the same degree to the total left ventricular mass and overestimates the relative contribution of the apical part of the left ventricle. Thus infarction involving the apex, such as LAD infarction, will be overestimated. Conversely, infarction involving the mid and basal portion of the left ventricle, such as LCX infarction, will be underestimated. As expected, correction for slice mass consistently decreased the LAD infarct size by decreasing the relative contribution of the apical slices to the total left ventricular mass and slightly increased the LCX infarct size in five of six dogs by increasing the relative contribution of the mid and basal slices to the total left ventricular mass. In the remaining dog with LCX infarction, the infarct size decreased by 1% with correction; in that experiment, the infarct was larger in the apical part of the left ventricle than in its basal part. Considering all dogs, correction for slice mass was beneficial in 11 of 14 cases by bringing their data points closer to the line of identity. When comparing the two methods, the regression line was closer to the line of identity with the corrected method; the correlation coefficient was greater and the SEE, the relative bias, and the relative precision were smaller with correction.

In a separate study, we have developed and validated a scintigraphic-based algorithm in normal dogs, which expresses the relationship between slice mass and fractional distance from the apex. This approach was then applied to eight normal subjects and a relationship between slice mass and fractional distance from the apex in human beings, close to the relationship in dogs, was found. The utility of the approach outlined in the manuscript for infarct sizing in human subjects by thallium tomography requires further validation, which is currently underway in our institution.

In conclusion, a method for quantifying myocardial infarct size by thallium SPECT and circumferential profile analysis has been described. Analysis of maximum-count circumferential profiles appears to be a suitable method for quantification of infarct size in individual tomograms. Assessment of total left ventricular infarct size requires further correction accounting for differing myocardial slice mass. Extension of this method for quantifying the extent of myocardial infarction in human beings requires further validation.

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