Scintigraphic Characteristics of Experimental Myocardial Infarct Extension

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SUMMARY
Technetium-99m-stannous pyrophosphate scintiphotos were evaluated for diagnosing and quantitating myocardial infarct (MI) extension in sedated dogs. Infarction and extension were produced by serial left anterior descending coronary artery ligations at 0 and 48 hours. We compared serial scintiphoto data with regional myocardial blood flow (MBF) (microsphere technique) and infarct histopathology. In eight control dogs, the scintigraphic MI area was stable at 24, 48, and 72 hours. In each of 11 dogs undergoing extension, the MI area increased after the 48-hour occlusion, averaging a 48.9% increase (p < 0.001). Grossly, most extensions were mixtures of confluent necrosis and moderate (patchy) necrosis. MBF to confluent infarct tissue decreased significantly, allowing the documentation of extension by totaling the grams of newly flow-deprived tissue, but patchy infarct tissue had little flow deprivation, making it difficult to quantitate this type of extension accurately by flow criteria alone. Rarely, extension could be diagnosed using conventional histologic criteria. We concluded that the scintiphoto MI area was related quantitatively to infarct weight in both control and extension. However, it was not possible to determine that an increase in the MI scintiphoto area was an accurate predictor of the degree of extension using independent flow or pathologic criteria.

MYOCARDIAL INFARCT (MI) size is an important determinant of morbidity and mortality during acute infarction.1,2 Increase in MI size by extension is believed to be frequent, based on ECG studies;3,4 however, there is little information available for estimating the mass of myocardium involved.5 Infarct scintigraphy may be a useful method, but extension has been shown in only one report.5

We examined the technetium-99m-stannous pyrophosphate (99mTc-PYP) scintigraphic method for detecting and quantitating infarct extension. We evaluated the relationships among serial 99mTc-PYP infarct scintiphotos, regional myocardial blood flow (RMBF) and cardiac histopathology in uncomplicated experimental myocardial infarction and in myocardial infarction complicated by induced infarct extension.

Materials and Methods

Anterior MIs were produced by left anterior descending (LAD) snare ligation, and “extension” was produced by a second, more proximal snare ligation (fig. 1).

Surgical Procedure

At thoracotomy, snares of 1-0 silk were placed around the LAD proximal and distal to its first diagonal branch and around the first diagonal branch. These snares were brought to subcutaneous locations through polyethylene tubing together with left atrial, carotid, and jugular venous silastic catheters. Antibiotic treatment was given with left atrial, carotid, and jugular venous silastic catheters. Antibiotic treatment was given for 2 days, and the dogs were studied 4–7 days postoperatively.

Experimental Protocol

Studies were conducted using morphine and diazepam for sedation. Control dogs underwent a single, distal LAD snare ligation, and the protocol outlined below was followed for 72 hours. Extension dogs underwent the identical protocol until 48 hours. Then, the proximal LAD snare and, frequently, the diagonal artery snare were pulled.

99mTc-PYP scintiphotos were obtained 24, 48 and 72 hours after coronary occlusion, each done 90–120 minutes after intravenous injection of 15–20 mCi of 99mTc-PYP (Mallinkrodt). Scintiphotos (750,000 counts) were obtained (Searle PhoGamma IV) using the 140 keV 99mTc peak, a 25% symmetrical window, and a 250-keV, high-resolution, parallel-hole collimator. Data were computer stored as 64 × 64 digital matrices (PDP 11/40). Anterior, 30° left anterior oblique (LAO), 40° LAO, left lateral and 40° right anterior oblique images were recorded.

RMBF was determined before and after coronary occlusion by the method of Rudolph and Heymann7 using 141Ce, 51Cr, 85Sr and 90Nb 15-μ radioactive microspheres (3M Company). Left atrial microsphere injections were made during the control period, 1 hour after ligation, at 24 or 48 hours, and 72 hours after ligation. For each determination, the spheres were dispersed using ultrasonic and mechanical agitators and drawn into a syringe to achieve a fivefold dilution in normal saline. Approximately 2 million spheres were injected into the left atrium over 30 seconds and flushed with saline. An arterial reference sample was
collected using a motorized pump withdrawing at 18.0 ml/min for 3 minutes (Harvard Apparatus). A 12-lead ECG and the arterial pressure were recorded during the control period and at each microsphere injection. The dogs were sacrificed after a final image and MBF at 72 hours. In three control dogs, MBF was determined at control, 1, 24 and 48 hours.

For postmortem analysis, the atria and fat were excised, and the combined ventricles were sliced into serial 4–5-mm transverse slices using an electric meat slicer. Thirty-five-millimeter photographs of the whole heart and the slices were made. Two weighed, representative slices of the infarct — one apical and one basal — were fixed in formalin for subsequent histopathologic studies. Three or more representative infarct slices adjacent to the surfaces of the histologic slices were weighed, sketched and divided into multiple sections for determining their \(^{99m}\)Tc and microsphere activities. The remaining myocardium was apportioned into additional counting vials according to its gross appearance — yellow MI, red ("hemorrhagic") MI, pale and patchy MI, normal and peri-infarct tissue (a normal-appearing zone 3–5 mm wide outside the gross MI borders). Sections with red, red-yellow, and yellow necrosis were designated as "confluent infarct" tissues.

The histologic slices were stained with hematoxylin and eosin and by the PAS method and examined microscopically in detail to define the extent of the 24-hour vs the 72-hour infarct. \(^{99m}\)Tc activity in the sections was measured in 14 dogs in duplicate using a two-channel gamma counter (Searle PhoGamma HP, Model 1185). Microsphere activity was measured in each tissue sample for 500 seconds using a multichannel analyzer with counting windows centered over the principal energy peak of each isotope (Packard AutoGamma Scintillation Spectrometer, Model 5986).

Data Analysis

The dogs with a minimum of two sets of scintiphotos and flows were included in the analysis.

Tissue Data

There was an average of 65 representative slice sections for each heart (range 24–96) weighing 0.625 ± 0.388 g (mean ± SD). All samples were coded according to their gross appearance, location in the ventricle (anterior, lateral, posterior and septal), and position in the ventricular wall (endocardial, epicardial, transmural, left interventricular septum, mid-septum and right septum).

MI weight was defined as the sum of the weights of all infarct samples. The weights of infarct and normal tissue in the slices used for histologic study were calculated using the formula \(MI_M = \frac{MI_{ADJ}}{WT_{ADJ}}\), where \(MI_{ADJ}\) is the average MI weight in two adjacent slices, \(WT_{ADJ}\) is the average weight of the adjacent slices, \(WT_H\) is the weight of the histologic slice, and \(MI_H\) is the MI weight of the histologic slice.

\(^{99m}\)Tc Activity

The differences between duplicate counts in tissue sections were quite small, ranging from 0.009%
(n = 61) to 0.5% (n = 94) in two dogs chosen at random.

**Myocardial Blood Flow**

The radioactivity of individual spheres was identified using a computer program modeled after the matrix method of Rudolph and Heymann. This resolved in vitro mixtures of the spheres we used to within 3% of their individual count rates. Flow to each section was determined in ml/min/g wet weight and was also calculated as relative MBF, the flow to each section as a fraction of the weighted mean flow to all normal tissue in that heart at each flow determination.

The precision of our MBF technique after myocardial infarction was determined as follows. Microspheres (\(^{14}\)Ce, \(^{51}\)Cr and \(^{85}\)Sr) were injected during the control period and 1 and 24 hours after infarction, respectively. The resulting 108 myocardial sections were counted five times sequentially. The standard deviation of the mean flow to the sections was 2.2, 3.4 and 1.6% for the control, 1- and 24-hour flows, respectively. Total myocardial flow was also quite stable. The standard deviation was 0.5, 1.9 and 0.5% of the mean total flow at control, 1 and 24 hours, respectively.

**Scintiphotos**

The computer-stored images were processed to define the MI area. The best view for demonstrating the infarct over the 3-day study was chosen. We used constant, measured background subtraction and manually outlined the \(^{99}\)Tc-PYP-positive image using an electronic cursor system to mark the MI border. The MI area was expressed in numbers of matrix cells. Frequent reference to analog Polaroid images facilitated the analysis.

Serial MBF and scintiphot data were analyzed using the CLINFO Data Management System (Division of Research Resources, NIH, Bethesda, Maryland). Flow parameters were compared with changes in the scintigraphic infarct area. Data were analyzed using paired and unpaired t tests, chi-square and linear regression analyses as appropriate. Statistical significance was defined as \(p < 0.05\).

**Results**

Forty-three dogs were operated. Twenty-four dogs were excluded from analysis, because 12 died before completing the study, nine had incomplete flow or

**Table 1. Scintigraphic Area, Myocardial Infarct Weight, and New Flow Deprivation in Control and Infarct Extension**

<table>
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<tr>
<th>Dog</th>
<th>Scintiphoto area*</th>
<th>MI (g)</th>
<th>NFD (g)</th>
<th>Change in scintiphoto area*</th>
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<tr>
<td></td>
<td>24 hr</td>
<td>48 hr</td>
<td>72 hr</td>
<td>24-48 hr</td>
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<td>Control group</td>
<td></td>
<td></td>
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<tr>
<td>10</td>
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<td>190</td>
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<td>101</td>
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Mean change in MI area

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<tr>
<td>Extension</td>
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<td>+47.7</td>
<td>+53.1</td>
<td>+53.1</td>
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*As number of matrix cells (pixels).

Abbreviations: MI = weight of myocardial infarct; NFD = weight of newly flow-deprived tissue.
scintiphoto data, and three had no MI after LAD ligation. Nineteen had infarction, survived the study protocol and had sufficient paired flow and scintiphoto data for analysis. There were eight control and 11 extension dogs. A mean of 3.2 flows per dog was obtained. Three scintiphotos were obtained in all extension dogs and a mean of 2.25 per control dog.

Scintiphotos

The optimum projection was selected and used for serial views in each dog. The 30° or 40° LAO views were the most commonly used images (16 of 19 dogs), as they were the most easily followed and had the least bony interference. The scintiphotos were outlined after background erasure, which averaged 30% of infarct counts. The data reported (table 1) are the results obtained by one trained observer who applied the criteria objectively, but not in a “blinded” fashion. The same serial images of seven randomly selected dogs (20 scintiphotos) were reanalyzed in a blinded fashion 18 months later by the same observer and by a second blinded observer. The results were highly reproducible. For the two trials of observer 1, \( r = 0.97 \), and for each trial of observer 1, the results of observer 2 were closely related (\( r = 0.94 \) and 0.96, respectively; for each, \( n = 20, p < 0.001 \)). For the first trial, the mean difference between the observers was 3.0 ± 1.41 pixels (SD), an average difference of 2.6%. The difference was ≥15 pixels in only three images. There was no consistent difference between the observers.

Serial scintiphotos of a control “stable” infarct are displayed in figure 2. For the control series, the infarct areas were stable. The MI areas of the first scintiphotos in control dogs averaged 109.9 ± 56.9 cells (SD) and increased by a mean of 0.6 cells over 72 hours (range −10 to +13 cells) (SD = 15%). The serial scintiphotos in extension dogs were equally stable until 48 hours, when all showed a pronounced increase in the infarct area (fig. 3). For extension dogs, the infarct area increased by 48.9%, from 85.9 ± 29.5 to 139 ± 40.8 cells (SD), representing an average increase of 53 cells (range 26–81; \( p < 0.001 \)). When first and last images were compared, there were no changes in the control dogs that approached the magnitude of the changes in the dogs with extension. All dogs with extension were detected by the scintigraphic method. When spontaneous changes before 48 hours were compared with induced changes after 48 hours, the largest spontaneous change (+20 cells) and the smallest induced change (+18 cells) overlapped in only one case.

We graded the intensity of the 99mTc-PYP uptake of the in vivo scintiphotos on a 0–4+ scale according to the method of Berman et al.10 Of the three dogs without MI, two had no evidence of uptake, and one had 1+ diffuse uptake (negative). All dogs with infarction had definite localized 99mTc-PYP uptake, and only one had <3+ uptake. In 15 paired 24–72-hour scintiphotos, four of eight control and 11 of 11 extension dogs, there was a gradual increase in 99mTc-PYP intensity from 24 hours (2.77) to 72 hours (3.40) (\( p = 0.004 \)).

Histopathology

All infarcts were anteroseptal or anterolateral and two-thirds to full-thickness in extent. The degree of necrosis (patchy, confluent, etc.) varied. The hearts were divided into an average of 14 transverse slices, of

**FIGURE 2.** Serial technetium-99m-stannous pyrophosphate scintiphotos in a control dog. On the left are serial Polaroid images after infarction; on the right, the myocardial infarct area is shown on digitized background-erased images. There is little change over the 72-hour period. Forty-degree left anterior oblique view.
FIGURE 3. Serial technetium-99m-stannous pyrophosphate scintiphotos in infarct extension. Format as in figure 2. A second, more proximal left anterior descending artery occlusion was done after the 48-hour scintiphotograph, and the myocardial infarct area increased significantly. Forty-degree left anterior oblique view.

Myocardial Blood Flow

We evaluated MBF as a method for quantitating the original infarction and extension. There were distinct, contrasting flow patterns in control and extension hearts. The MBF for a representative slice in a typical “control” heart is shown in figure 4. The MBF to infarcted lateral and anterior subendocardial sections diminished by 1 hour after occlusion and usually remained low. Flow to epicardial sections in other slices dropped but often increased later to nearly normal levels.

Figure 5 shows flow changes in extension dogs. MBF was more complex, because infarct tissue could be originally flow deprived or newly flow deprived (NFD) (i.e., after extension). Tissue originally flow-deprived was defined as tissue in which flow decreased after the first occlusion, in contrast to NFD tissue, where flow was within normal limits until after the second occlusion.

Figure 6 summarizes the flow data. For both control and extension, normal and MI flow varied considerably. In the control period, absolute MBF appeared to be greater in control than in extension dogs, but this trend was not statistically significant.

Infarct MBF in control dogs decreased markedly by 1 hour. Flow to confluent infarct tissue decreased further by 24 hours and then remained stable. Flow to pale-patchy infarct tissue rose slightly by 72 hours, but not significantly.

Infarct MBF in extension dogs showed
characteristics that suggested a mixture of normal and infarct tissue. At 72 hours, infarct flows in extension dogs were equal to those in the control dogs. However, before then, the higher flow level and mid-course fluctuations similar to those in normal tissue are believed due to a mixture of normal and infarct tissue, all of which became infarcted after the second occlusion. Several variables might affect the mid-course MBF. Mid-course flow would be highest in dogs with small initial infarcts and large amounts of extension. Also, the mid-course data could have been altered if dogs with the largest initial infarcts had died after extension was induced. However, only one dog died after extension.

To define changes due to extension, we examined MBF in terms of relative MBF, with normal mean flow defined as 1.0 in order to reduce the influence of serial fluctuations in normal flow (see Data Analysis). The relative flows to infarct and normal tissues were ranked in ascending order by gross tissue morphology to define the best flow discriminator among them and to estimate the degree of new flow deprivation (NFD) necessary for patchy and confluent infarction. At 72 hours, the relative flow value of 0.66 ± 0.14 (sp) most accurately distinguished normal from confluent infarct tissue, with the fewest misclassified sections (average 2.2/heart). Patchy infarct tissue had higher flow, and it was impossible to accurately separate this tissue type from normal by flow criteria alone.

Based on the above analysis, we designed several criteria for defining NFD. The strictest criterion was maintenance of normal flow (>0.66) until after 48 hours, then a decrease of at least 25% in MBF to that tissue section. Tissue originally flow deprived had a decrease in flow to <0.66 at 1, 24, or 48 hours. Results are listed in Table 1. For NFD, sensitivity and specificity are high on a section-by-section basis but are somewhat lower on a dog-by-dog basis. Three dogs in the extension series had little or no NFD tissue despite large increases in the scintigraphic MI area. Two of seven control dogs had small amounts of NFD tissue (<1.4 g). More lenient flow criteria showed greater sensitivity but less specificity for extension.

The NFD algorithm was also applied to normal tissue in the extension dogs. Only nine of 303 normal tissue sections met the criteria for NFD. By definition, no normal tissue was included in the NFD calculations.

The accuracy of the representative slice method for defining the fraction of NFD tissue was evaluated in 4 hearts. The NFD fraction was estimated using the flow data from three representative slices in each heart and compared to the actual NFD mass, which was obtained by evaluating the flow to all sections of the in-
farct in every slice. The mean difference between the estimated and actual NFD fraction was 5.8% (actual differences -6.6%, -3.0%, 0%, +13.8%).

Scintigraphic Estimation of Infarct Weight

We compared the final scintigraphic infarct area with morphologic infarct weight (fig. 7). The correlation was significant \( r = 0.70, p < 0.001 \) but somewhat weaker than previously described for \(^{99m}\)Tc-PYP images.\(^{12,13} \) For control dogs, \( r = 0.81 \), and for extension dogs, \( r = 0.67 \). By including only the 10 extension hearts with at least 3+ uptake, the correlation coefficient in extension dogs improved to 0.96, and to 0.75 for the total series.

Flow vs \(^{99m}\)Tc-PYP Activity

MBF and \(^{99m}\)Tc-PYP uptake subdivided by tissue type were available in 14 hearts at 72 hours (fig. 8).

Patchy infarct tissue had a marked \(^{99m}\)Tc-PYP uptake (4.8 times normal) but only a 19% flow deficit compared with the large mass of "average-normal" tissue. \(^{99m}\)Tc-PYP uptake in confluent infarct tissue was 9.4 times normal and was associated with a much greater flow deficit (63% decrease).

Estimation of Extension

Figure 9 shows the change in the scintigraphic infarct area compared with the percent of the infarct newly flow deprived. Scintigraphic change was computed using the initial and final scintiphotos. The percent of the infarct newly flow deprived was calculated for all 14 dogs with four flows. Despite the significant relationship between infarct weight and infarct area, there was no significant relationship between the flow and the scintigraphic parameters (fig. 9A).

This poor relationship can probably be explained by
the presence of patchy infarct tissue. All infarcts were easily visible in scintiphotos, including predominantly patchy infarcts. This patchy tissue was ischemic, but its flow was relatively high due to a mixture of normal and infarct tissue. Thus, little patchy tissue met the definition for NFD, although scintigraphy showed extension. When all dogs that had infarcts with >40% patchy necrosis were excluded from analysis (fig. 9B), there was a significant relationship between the change in the scintiphotos and the percentage of the heart newly flow deprived \( r = 0.69 \). The percent patchy infarct weight in control dogs was 23% (range 0–90%), and only two dogs had >20% patchy infarction (46% and 90%). Extension dogs had an average of 39% patchy necrosis (range 10–88%), but only two had <25% patchy tissue (10% and 14%).

**Discussion**

MI size is a powerful determinant of prognosis and complications in acute myocardial infarction.14-18

**Figure 7.** Relationship of scintigraphic myocardial infarct area and infarct weight. The relationships are significant for the entire series and the subsets of control and extension dogs. TcPYP = technetium-99m-stannous pyrophosphate.

**Figure 8.** Myocardial blood flow (MBF) compared with tissue technetium-99m-stannous pyrophosphate (TcPYP) content. Each point represents the mean ± SEM of all available samples. Infarcted tissue had high TcPYP uptake. Tissue with confluent necrosis had much lower MBF than pale or patchy infarct tissue. AVG = average; NL = normal; POST = posterior; cpm = counts per minute.
Thus, infarct extension could contribute to the development of heart failure, cardiogenic shock and possibly death. Extension is reported to be frequent in fatal cases of cardiogenic shock and in nonfatal infarction. Considering the probable frequency of extension, therapies aimed at salvaging ischemic myocardium may also have an important effect on preventing extension. There are pronounced time restraints on preserving the originally ischemic tissue.

Clinical, electrocardiographic, biochemical, scintigraphic, pathologic, and MBF parameters may be useful for detecting and quantitating infarct extension. Clinical recognition is difficult. Electrocardiographic criteria have been the most frequently investigated, and ST-segment mapping of anterior and anterolateral infarction suggests that there is at least a 50% incidence of infarct extension during the acute stage of infarction, usually associated with new release of creatine kinase (CK). However, the validity of these ECG observations on extension has been questioned by others.

Increases in serum CK concentration occur during extension in both experimental animals and man. However, quantitation of infarct extension has not been adequately investigated. Mathey et al. found that extension was frequent in their series of patients and accounted for a 24% average increase in infarct weight as judged by serum CK time-activity curves. Reperfusion may also release excess CK into the blood, and thus it may be difficult to define the cause of new or prolonged CK elevations.

This study was designed to evaluate the use of scintigraphy to determine the presence and degree of infarct extension. Little information is available on this topic. Willerson and associates showed extension in a single case report, but we are aware of no experimental studies to evaluate this phenomenon.

We found that serial ligation of the LAD was a reliable method for producing extension. In the control dogs, the scintigraphic MI area remained relatively constant over the 3-day study. In dogs with induced extension, the increase in scintigraphic MI...
area was easily detectable and outside the expected range of variation when compared with that of the control series. Comparing first and last images, there was no overlap between the control and extension series. The spontaneous variability in the scintiphotos before 48 hours was greater than the smallest induced change from extension after 48 hours in only one dog.

The scintiphotos showed a quantitative relationship to gross infarct weight. The statistical relationship was good for the entire series as well as for the control group and reasonably strong in the extension group. The dog that had the most poorly defined infarct scintiphoto fell outside reasonable statistical variability.

Our technique of extension after 48 hours frequently created larger infarcts than were seen in the surviving control group. Infarcts in extension dogs had more patchy necrosis than those in control dogs. This finding may be due to the development of collateral circulation or other compensatory mechanisms during the 48 hours before the second ligation. Support for this concept may be found in other studies. Paul et al. found angiographically demonstrable interarterial coronary anastomoses as early as 2 days after circumflex artery ligation in the pig. Also, Pasyk and associates found increases in peripheral circumflex artery pressure and xenon-133 clearance during circumflex artery occlusions lasting 10–31 hours in dogs. The causes of such increases in collateral indices are speculative, but it is reasonable to suspect increased coronary pressure and flow in occluded vessels in our model as well. Thus, a second occlusion in that region might cause a milder degree of ischemic necrosis under these conditions.

In the studies reported here, it was not possible to use histopathology or MBF as the standards to establish that the scintigraphic method can define infarct extension quantitatively. Standard histopathology did not distinguish reliably between the extension of the infarct (which was 24 hours old) and the original infarct (which was 72 hours old). The classic techniques for defining the age of infarcts require longer intervals to allow characteristic histologic changes to evolve. Such changes were seen in the studies reported by Page, Alonso, and their associates, who made observations on a prolonged basis.

Hutchins and Bulkley have shown in man that infarcts often “expand” within their original borders by intramyocardial disruption and “extend” peripherally as well. Most of their patients with clinically recognizable recurrent ischemic events had both expansion and extension. We found with scintigraphy that extension beyond original borders occurred in every case in our model. Our flow data (fig. 5) show that there is NFD peripherally as well as recurrent flow deprivation in the more central tissue that was originally flow deprived. Although unproven, this could occur in man as well and might be the mechanism of expansion and extension.

When there were large flow decreases (with confluent infarction), serial MBF data were useful in documenting extension. However, when extension caused patchy infarction, the flow changes were smaller and less conclusive. For extension dogs, flow to patchy infarct tissue decreased 11% after the second occlusion. This change was statistically significant (p < 0.02), but MBF remained within the normal range. By comparison, there was a 33% flow decrease to confluent infarction tissue after the second occlusion (p < 0.001), and flow dropped to definitely ischemic levels. Flow to confluent MI tissue in control dogs was stable, decreasing only 5.3% between the last two flows for each dog (p = NS).

We concluded that neither flow decreases nor absolute flow levels were sufficient to include patchy infarct extension in the category of tissue newly flow deprived, although patchy infarcts were visible on scintiphotos. The hypothesis that patchy necrosis is an important factor in the scintiphoto-flow comparison fits the data in figure 9A, which shows a greater increase in the scintiphoto area than in NFD in several dogs (upper left). The two extension dogs with the smallest percentage of patchy necrosis (0.142 and 0.245) had greater NFD and are points close to the regression line. However, the data are not sufficient for statistical analysis.

Geometric inaccuracy of conventional two-dimensional scintigraphy may be an additional factor in preventing a better correlation between flow and scintigraphic changes. Some extension probably occurred in planes perpendicular to the en face view of the infarct we sought to obtain. Our anatomic data showing septal and lateral wall extension by flow criteria support this possibility. To examine the importance of this variable, we used linear regression analysis for two groups to compare scintigraphic MI area with MI weight in the control and extension groups, after excluding the dog with the most poorly defined scintiphotos. There was no significant difference between the regression slopes and variances in control and extension dogs. It is possible that the lack of difference in the regression parameters is due to large infarcts in each series we tested, with portions nonparallel to the scintillation camera. Nonetheless, this does not diminish the validity of our conclusion that two-dimensional scintiphotos were sensitive and specific for extension and were quantitatively related to infarct weight in both control and extension dogs. Thus, the difference between the two would relate to the mass of tissue involved in extension. The minimum weight of extension needed for detection is undefined, but experimental infarcts as small as 1–5 g have been detected by conventional 99mTc-PYP scintigraphy. Further study of promising three-dimensional techniques for analyzing pyrophosphate MI scintiphotos may improve the quantitation of extension.

This study shows the difficulties in confirming or denying the hypothesis that scintiphotos are quantitatively related to infarct extension. The significant agreement between infarct weight and scintigraphic
infarct area makes it reasonable to believe that semi-quantitative scintigraphic estimation of extension should be possible, although the accuracy with which the quantity of extension can be judged remains uncertain.

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