Quantitative Thallium-201 Redistribution with a Fixed Coronary Stenosis in Dogs

JEFFREY LÉPPO, M.D., JOEL ROSENKRANTZ, B.S., ROBERT ROSENTHAL, M.D., ROBERT BONTEMPS, B.S., AND TADA YIPINTSOI, M.B., PH.D.

SUMMARY The redistribution of thallium-201 (201Ti) after coronary vasodilation was studied in 14 dogs with a proximal stenosis of the left circumflex coronary artery that did not reduce basal flow but attenuated reactive hyperemia. During an 8–10-minute i.v. infusion of adenosine, radioactive microspheres and 201Ti were injected into the left atrium. Sequential cardiac scintiscans and microsphere injections permitted subsequent determination of coronary blood flow during the redistribution of 201Ti. After 15–220 minutes of observation, the dogs were killed and the hearts removed for the measurement of the activity of 201Ti and the radioactive microspheres in the normal (left anterior descending coronary artery [LAD]) and flow-restricted (left circumflex coronary artery [LCX]) regions. The ratio of the activity in LAD/LCX for microspheres (TRmic) and for 201Ti (RT1) were compared with the activity ratio determined from the scintiscan (Rscan). RT1 for the microsphere simultaneously injected with 201Ti can be compared with the initial Rscan, which showed a significant hourly decrease from an initial value of 1.26 ± 0.12 to a mean final value of 1.02 ± 0.09 by 3–4 hours. The final Rscan (mean 1.07 ± 0.10) in each experiment also correlated significantly (r = 0.854) with the final true myocardial RT1 (mean 1.26 ± 0.25). Rscan underestimated RT1 (average 2.25 ± 0.48) when both 201Ti and microspheres were simultaneously injected; Rscan also underestimated RT1, but the directional changes were similar. A further analysis of the Rscan, Rmic, and RT1 in two groups of dogs with either relatively high or low coronary flow during adenosine infusion suggests that the net loss of cellular 201Ti from the normal scintigraphic area is the mechanism underlying the resolution of these initial defects.

IF A CARDIAC SCINTISCAN using thallium-201 (201Ti) during exercise shows a defect that does not persist on serial views, it is generally accepted1–10 that myocardial ischemia is the cause. The disappearance of these transient defects has been studied in experimental models that used a temporary or permanent occlusion of a coronary artery to produce an initial defect on scintiscans.11–13 These studies have shown that 201Ti redistributes into an ischemic region over time. However, a 201Ti scan defect has been associated with a disparity in coronary flow in the absence of ischemia.14

Some investigators15–17 created myocardial regions with different flows by producing a fixed stenosis in one of the major coronary arteries and injecting 201Ti during transient i.v. infusion of vasodilators. This experimental design allows observation of the initial scintigraphic defect in the myocardial region in which flow could not increase with the vasodilator. However, the redistribution of 201Ti in such a model was not studied. Accordingly, we have studied Ti distribution in a similar animal preparation using adenosine.18–20

By extending our studies over a variable time course, 201Ti redistribution could be observed and its mechanism investigated. In addition, sequential microsphere injections were performed permitting an examination of: (1) the relationship between the scintigraphic defect and regional myocardial blood flow as determined by the microspheres; (2) the relationship of the scintigraphic defect to the actual 201Ti content in the region when the flow disparity no longer existed; and (3) how the duration of redistribution and other factors affect the disappearance of a defect on 201Ti scintiscan.

Methods

Adult male mongrel dogs (average body weight 21.4 kg) were premedicated with intramuscular morphine sulfate (2 mg/kg) and anesthetized with i.v. sodium pentobarbital (15 mg/kg), which was supplemented whenever necessary. After tracheostomy, ventilatory assistance was maintained by means of a Harvard respirator. Arterial blood gas was maintained within physiologic values by appropriate adjustment of ventilation, oxygen administration or sodium bicarbonate infusion. After a left lateral thoracotomy, the proximal left circumflex coronary artery was dissected for placement of an electromagnetic flowmeter transducer (Zepeda or Biotronex) and a distal balloon occluder for zero flow. A #7 pigtail catheter was introduced into the right atrium for injection of indocyanine green dye and a #8 short polyethylene catheter was placed in the right femoral artery and connected in series to a densitometer (Gilford 103 IR) and to a Harvard withdrawal pump for determination of cardiac output and collection of arterial reference blood during the microsphere injections. A #7 catheter was placed in the right femoral vein for infusion of fluid or adenosine and another #7 catheter was positioned in the ascending aorta for monitoring pressure and for collecting another arterial reference blood during microsphere injections. Finally, a #8 catheter was placed in the left atrium for pressure monitoring and injection of microspheres and 201Ti. The ascending aorta and left atrial catheters were connected to Statham P23Db pressure transducers and all signals,
including ECG, were recorded on a VR6 Electronics for Medicine recorder.

After the placement of the flowmeter and the intravascular catheters, a 15-second coronary artery occlusion was performed and the reactive hyperemic response was recorded. Peak response, defined as peak flow during release divided by mean flow before occlusion, was 413 ± 159% (mean ± SD) for 14 dogs. Adenosine (Sigma Chemical Company) at a concentration of 0.5–1.0 mg/ml in normal saline was then infused intravenously at a rate that doubled or tripled resting circumflex flow. This rate was subsequently used during 201Tl injection. A silk ligature and external probe were used to create a constriction around the circumflex artery distal to the balloon occluder. The constriction was judged to be satisfactory if resting flow was not reduced but the peak reactive hyperemic response was curtailed to not greater than 200% (average 138 ± 35%). The chest was closed in layers and the intrathoracic air evacuated.

Radioactive microspheres 15 µ in diameter, labeled with 46Sc, 46Sr, 113Sn, 141Ce or 128I, were obtained from New England Nuclear or 3M Company. Two to eight million microspheres (50–100 µCi) were resuspended in 10% Dextran with Tween and ultrasonicated for 20 minutes before injection. When 201Tl (New England Nuclear) was to be simultaneously injected with the microspheres, 1.5 mCi were added to the microsphere suspension before the ultrasonication. Dextran (20 ml) was infused to replace blood loss after the collection of reference arterial blood samples.

Protocol

Control hemodynamic recordings were made in all 14 dogs before the adenosine infusion, and in four dogs microspheres were also injected. This time period is called the pre-adenosine period. Adenosine was then infused at the predetermined rate in each dog and after hemodynamic recordings demonstrated stable heart rate and aortic pressure, the mixture of microspheres and 201Tl was injected into the left atrium. The adenosine infusion was continued for 1–2 minutes after the blood collections (total duration of infusion was 8–10 minutes) and then myocardial scintigraphic recordings were started. This period of observation is called the adenosine or initial period. Hemodynamic measurements and scintiscans were repeated every 10–30 minutes for the duration of each experiment. In another four of these 14 dogs, microspheres were injected at hourly intervals to ensure that with cessation of adenosine, myocardial flow distal to the stenosis remained similar to the flow in regions supplied by nonconstricted coronary vessels. Before the end of a study (final time), differently labeled microspheres were injected. This final time for each experiment varied from 15–220 minutes after the 201Tl injection.

Thallium Scintigraphy

A standard gamma camera (Dymax, Elscint) with an intermediate collimator and a 21% window centered at 71 keV was used to collect 400,000–500,000 counts per image. A Polaroid analogue scintigram was made and the digital image on a 128 × 128 matrix was stored in a dedicated computer (Elscint). The relative position of the camera and the heart was maintained by the superposition of the previously recorded cobalt-57 markers placed in a triangle on the chest wall.

The digitized images were analyzed by selecting several regions of interest. An area encompassing the defect (D), an area judged to be normal (N), an area comprising the entire left ventricle (LV) and multiple areas of background (Bkg) were selected from the initial scintiscan as shown in figure 1. These areas could be exactly retracted on subsequent scans as each image was recorded in the same matrix position. A scan ratio (Rscan) was determined from the average counts/pixel in these areas.

\[
R_{scan} = \frac{(N-Bkg)/(LV-Bkg)}{(D-Bkg)/(LV-Bkg)}
\]

Hence, this activity ratio denotes the degree of disparate distribution of 201Tl and can be followed up at different intervals after stopping adenosine infusion. The term (LV–Bkg) was used to normalize each region to the total LV so that activity in each zone could be more easily compared on both intra- and interexperimental bases.

Myocardial Isotope Determinations

After the dogs were killed, the hearts were removed and Evans blue was injected into the left circumflex artery just distal to the stenosis. Each heart was cut into five or six concentric rings in a plane parallel to the atrioventricular groove and each ring was then cut into transmural radial segments. The left circumflex (LCX) region was carefully separated from the left anterior descending (LAD) region and each piece was weighed and placed in a test tube. All tissue and reference arterial blood samples were counted in a Nuclear Chicago 1185 gamma counter. In general, 201Tl was counted within 2–5 days after the experiment; the isotopically labeled microspheres were counted about 30–40 days later to allow 201Tl to decay by 10 half-lives. The net activity of each isotope was obtained by conventional correction for background, energy overlap and decay due to counting. In the present study, data analysis was limited to myocardial pieces obtained from the left ventricle and septum. The LCX region stained by Evans blue and the unstained LAD region were selected by an interactive computer program and expressed as an average isotopic activity (counts per minute per gram [cpm/g]). Isotopic activity for the dual arterial reference blood deviated by an average of 3.0 ± 2.5% from the mean of the two samples. This mean value was used to calculate blood flow in ml/g/min for each of the regions (LAD and LCX) for every microsphere. A ratio (Rmic) of the microsphere concentrations in the LAD and LCX regions was calculated. The final myocardial ratio (Rmc) of the 201Tl counts/g in the LAD and LCX regions was similarly determined in each heart.
An additional computer analysis was used to examine the possibility that $^{201}$TI distribution at the final time might be related to the blood flow during $^{201}$TI introduction, i.e., during the period of unequal coronary flow response to adenosine infusion. We made this correlation between adenosine regional myocardial blood flow and final $^{201}$TI content in large ventricular pieces. A typical example is shown in figure 2. Beginning with the region that comprised 15–20% of the left ventricular weight and also had the lowest absolute blood flow during the adenosine infusion, the blood flows in the remaining left ventricular regions were converted into multiples of this lowest flow. The number of steps for each normalization varied from five to eight; the greater number of steps allows a better description of hearts that have a wider range of regional flows. The $^{201}$TI content (cpm/g) in each of these flow-determined steps was also converted into a multiple of the $^{201}$TI activity in the region with the lowest microsphere flow. A linear regression fit between the normalized flow and the normalized $^{201}$TI content was performed using the left ventricular mass for each step as a weighting function. This process (normalized isotope distribution) yields a significant correlation if the initial high and normal flow regions as determined by the adenosine microspheres still have the highest and lowest $^{201}$TI content, respectively, at the final time.

**Validation**

The ability of the method of analysis using $R_{mic}$, $R_T$, and $R_{scan}$ to detect large regions of disparate or homogeneous cardiac perfusion was confirmed in an initial study involving a separate group of four dogs. An infarction was produced in two dogs and multiple determinations of $R_{mic}$ and $R_{scan}$ over 130 minutes showed a persistent regional difference in flow and $^{201}$TI content. $R_{mic}$ averaged 1.96 ± 0.08 (SD) and 3.16 ± 0.19 (n = 3 for each). $R_{scan}$ averaged...
1.47 ± 0.08 and 1.36 ± 0.05 (n = 4) for each dog), while R_{TI} was 1.80 and 2.16, respectively. In two other dogs, studies were performed using the experimental protocol without an adenosine infusion. In these, R_{mic} averaged 0.99 ± 0.03 (n = 8) and R_{TI} was 0.99 and 0.90 at 60 and 180 minutes, respectively. Repeated scans in each dog did not show a random difference of more than 8% between the anterior and posterior walls.

**Results**

In the 14 experimental dogs, the mean ± SD for the determinations from the arterial blood showed pH of 7.35 ± 0.04, P_{O_2} of 108 ± 33, P_{CO_2} of 36 ± 7 mm Hg and hematocrit of 37 ± 7%. The left ventricular region supplied by the LAD weighed 49.4 ± 9.0 g and that supplied by the LCX weighed 30 ± 5.5 g. The amount of adenosine infused was 0.32 ± 0.28 mg/kg/min.

**Hemodynamics**

The heart rate, mean aortic and left atrial pressures as well as the cardiac output and coronary blood flow were determined during two control periods (before and after adenosine) and also during the adenosine infusion. Adenosine significantly increased the heart rate and cardiac output, but decreased the mean aortic pressure. In 10 of 14 dogs, the pre-adenosine heart rate of 138 ± 35 beats/min increased to 150 ± 37 beats/min with adenosine. The mean aortic pressure in all 14 dogs decreased from 102 ± 16 mm Hg during the pre-adenosine period to 89 ± 19 mm Hg with adenosine and returned to 107 ± 17 after adenosine. The mean left atrial pressure remained constant for all periods and averaged 6 ± 2 mm Hg. Cardiac output increased in 12 of 14 dogs, from an average of 2.26 ± 0.58 l/min before adenosine to 2.90 ± 0.78 during adenosine and returned to 2.32 ± 0.68 after adenosine. In each experiment the coronary blood flow increased during the adenosine infusion with a mean flow that approximately tripled, but there were marked variations between dogs. Coronary blood flow calculated from all the heart samples was 0.76 ± 0.19 ml/g/min before adenosine, which increased to 2.56 ± 1.18 ml/g/min with adenosine and then returned to 0.96 ± 0.34 ml/g/min.

**Regional Blood Flow and 201TI Scintigraphy**

Table 1 lists the average regional myocardial blood flow, R_{mic} and R_{scan} for the 14 experimental dogs. During the adenosine infusion, flow to the non-constricted LAD region averaged 3.47 ± 1.67 ml/g/min, and LCX blood flow was consistently less due to the coronary stenosis. Compared with flows obtained before or after the infusion, LCX flow during adenosine increased in nine of 14 dogs by an average of 61 ± 53%. There were no alterations of LCX flow in four dogs. In one dog there was only a single microsphere injection. R_{mic} during adenosine averaged 2.25 but ranged around unity before and after adenosine, showing that the effect of adenosine was transient and that the stenosis did not affect resting LCX flow. During the adenosine infusion, all R_{scan} values were greater than 1.10, but the average was only half that of R_{mic}. This is further illustrated in figure 3, where each adenosine R_{mic} was plotted against the initial R_{scan}. The value of R_{scan} underestimated that of R_{mic} by an average of 43 ± 10%.

**R_{scan} as a Function of Time**

The R_{scan} decreased significantly during each successive time period, reaching a mean of 1.02 ± 0.09 after 180-220 minutes (table 1). However, our quantitation of R_{scan} is highly dependent on the background subtraction. Regional thallium kinetics were therefore analyzed here from microsphere and in vitro counting of thallium in tissue samples.

**Final R_{scan} vs R_{TI}**

The R_{TI} expresses the final myocardial LAD/LCX 201TI ratio and, as experimental time increased, its value tended to reach unity.

**Table 1. Adenosine Effect on Regional Flow and Serial Thallium-201 Scintigraphy**

<table>
<thead>
<tr>
<th></th>
<th>Before adenosine</th>
<th>Adenosine</th>
<th>10-60 minutes</th>
<th>61-125 minutes</th>
<th>180-220 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD flow (ml/g/min)</td>
<td>0.88 ± 0.24</td>
<td>3.47 ± 1.64</td>
<td>1.01 ± 0.16</td>
<td>1.14 ± 0.39</td>
<td>1.16 ± 0.52</td>
</tr>
<tr>
<td>LCX flow (ml/g/min)</td>
<td>0.87 ± 0.20</td>
<td>1.48 ± 0.53</td>
<td>0.97 ± 0.18</td>
<td>1.12 ± 0.40</td>
<td>1.11 ± 0.45</td>
</tr>
<tr>
<td>R_{mic}</td>
<td>1.01 ± 0.06</td>
<td>2.25 ± 0.48†</td>
<td>1.05 ± 0.08</td>
<td>1.02 ± 0.07</td>
<td>1.04 ± 0.10</td>
</tr>
<tr>
<td>n</td>
<td>(n = 4)</td>
<td>(n = 14)</td>
<td>(n = 6)</td>
<td>(n = 7)</td>
<td>(n = 6)</td>
</tr>
<tr>
<td>R_{scan}</td>
<td>1.26 ± 0.12</td>
<td>1.17 ± 0.09</td>
<td>1.08 ± 0.10</td>
<td>1.02 ± 0.09</td>
<td>1.13 ± 0.09</td>
</tr>
<tr>
<td>n</td>
<td>(n = 13)</td>
<td>(n = 13)</td>
<td>(n = 9)</td>
<td>(n = 6)</td>
<td>(n = 6)</td>
</tr>
</tbody>
</table>

Values are mean ± SD; n = number of observations.

Abbreviations: LAD flow = mean flow in normal left ventricle region; LCX flow = mean flow in left ventricle region distal to stenosis; R_{mic} = LAD flow/LCX flow; R_{scan} = normal/defect area on 201TI scans.
The relationship between the final $R_{scan}$ and the $R_{TI}$ appears linear (fig. 4). Over the entire range of experimental times, the mean final $R_{scan}$ was $1.07 \pm 0.10$ and $R_{TI}$ averaged $1.26 \pm 0.25$. When the study was terminated by 1 hour, the mean $R_{TI}$ and final $R_{scan}$ for four hearts were $1.50 \pm 0.30$ and $1.16 \pm 0.12$, respectively. The scintigraphic quantitation underestimates the actual myocardial $^{201}$TI distribution by an average of $22 \pm 8\%$. At the time when most clinical redistribution scans are performed (180–220-minute period), the mean $R_{TI}$ was $1.16 \pm 0.14$ ($n=6$) and the mean final $R_{scan}$ was $1.02 \pm 0.09$ ($p<0.05$).

### Regional Flow Differences During Adenosine Infusion

Although there was a wide individual variation, the flow in the LAD region for each experiment was always higher during the adenosine infusion than for any other time period. The flow to the LCX region with adenosine was less consistent because of varying severity of the stenoses. In eight dogs, the LCX flow during adenosine infusion was relatively high (> 1.6 ml/g/min) and these dogs will be referred to as the high-flow group. In the remaining six dogs, LCX flow was less than 1.07 ml/g/min during the adenosine administration, which was not significantly different from their basal levels. Table 2 shows the data for the high-flow and low-flow groups. The scintigraphic appearance of hearts in the high-flow group showed a rapid filling in of the initial defect. This observed faster disappearance in the high-flow group occurred despite a similarity of adenosine $R_{mic}$ and initial $R_{scan}$. In addition, $R_{TI}$ was lower and final $R_{scan}$ tended to be lower in experiments in the high-flow group that lasted longer than 80 minutes. Because of the similarity in observed adenosine $R_{mic}$ values, the high-flow group defined by higher LCX flow during adenosine also showed a significantly higher LAD flow.

### Normalized Isotope Distribution

Figure 2 is a typical example of how this normalized isotope distribution was determined in each experi-
In all but one experiment, there was a significant linear relationship between the initial myocardial distribution of microspheres and the final $^{201}$TI distribution of microspheres. The correlation coefficients ranged from 0.88-0.99 in 13 hearts and the experiments lasted 15-220 minutes. One exception ($r = 0.59$) was noted where at 184 minutes there was a relatively high final $^{201}$TI content in the region that initially showed low flow. The high correlation coefficients of the linear regression lines in the remaining hearts support the hypothesis that the regions of each heart with the highest and lowest flow values during the $^{201}$TI injection will also be the regions of highest and lowest contents of $^{201}$TI at the end of the experiment.

**Discussion**

The results of this present study should be interpreted only with regard to vasodilator infusion in conjunction with $^{201}$TI scintigraphy. We have observed that final myocardial $^{201}$TI distribution as represented by $R_{TI}$, tends to reach unity after 180-220 minutes, but $R_{TI}$ remains significantly higher than final $R_{scan}$ values. The analysis of the normalized isotope distribution suggests that the initial coronary flow to any region determines the final $^{201}$TI content at 15-220 minutes later. The observed difference in the time for the disappearance of the $^{201}$TI defect between the high-flow and low-flow groups can be explained by the relatively faster net loss of $^{201}$TI from the initially normal area on the scintiscans in the high-flow hearts.

**Gamma Camera Quantitation**

Standard scintigraphic imaging can detect the redistribution of $^{201}$TI after an initial vasodilator-induced disparity in regional coronary blood flow.

We used the $R_{scan}$ to describe the ratio of the relatively isotopic content in different scintigraphic regions of the heart as reported by other investigators using similar techniques. The initial $R_{scan}$ reflects the initial flow disparity, albeit only qualitatively, as the initial $R_{scan}$ underestimates the adenosine $R_{mic}$ by an average of 43 ± 10% (fig. 3). Strauss and Pitt reported that after an adenosine bolus, the initial $^{201}$TI myocardial content underestimated that of simultaneously injected microspheres. The scintigraphic underestimation of the initial flow disparity could be due to poor resolution of the imaging technique and/or the lack of proportionality of thallium content to blood flow in the high-flow regions, where diffusion limitation may play an important role. Poor resolution of the technique is suggested by the fact that final $R_{scan}$ underestimated the $R_{TI}$ by an average of 14 ± 10% for all 14 experiments, and this underestimation averaged 22 ± 8% in the four studies completed within 1 hour. Inappropriate quantitation of background activity also may contribute to this underestimation.

**Regional Changes in Myocardial $^{201}$TI from Imaging**

The significant decrease in $R_{scan}$ with time supports the visual interpretation of the disappearance of a defect. In other reports on the redistribution of $^{201}$TI, sequential analysis of myocardial tissue samples showed that true myocardial $^{201}$TI content always decreased as a function of time in regions that did not have a severe reduction in basal flow during the injection of $^{201}$TI. Therefore, our observed decrease in $R_{scan}$ with time strongly implies that the rate of net $^{201}$TI loss from the normal area has to be greater than that from the defect area, in which there was no decrease in coronary flow during each experiment. However, scintigraphic quantitation alone suggests the possibility that the $^{201}$TI content in the defect area could also decrease but at a slower rate than the normal area, or that it might not change or could even increase as a function of time.

**Initial Regional Flow and Final Myocardial $^{201}$TI Content**

The analysis of the normalized isotope distribution showed a significant linear relationship between the final (15-220-minute) distribution of $^{201}$TI to that of microspheres simultaneously injected with $^{201}$TI during the adenosine infusion. This implies that in any region of the heart the final (2-4-hour) $^{201}$TI content depends on the initial flow, which we assume should be related to the initial $^{201}$TI content. If regions with disparate blood flow had the same rate of net wash out, $R_{TI}$ would have remained constant as a function of time after injection of isotopes. With time, however,
R\textsubscript{T1} approached unity and, therefore, the net washout rate must have been higher for the region with the transient highest blood flow, which also had the higher 201Tl content.

Resolution of Scan Defects: Flow Independence and Regional Myocardial 201Tl Changes

Assuming that 201Tl has a potassium-like distribution\textsuperscript{29,30} the net release of cellular thallium is the result of both the loss of tracer from the cell, which is flow-independent, and its gain from the blood, which is flow- and extraction-dependent. The 201Tl redistribution process after vasodilators cannot be due to persistence of regional differences in blood flows, because coronary blood flow determined after the adenosine was discontinued remained equal in the LAD and LCX regions, despite a temporal reduction in the R\textsubscript{scan}. The independence of 201Tl redistribution from changes in blood flow has been suggested in earlier studies.\textsuperscript{27,31}

Calculated ratios such as R\textsubscript{T1} and R\textsubscript{scan}\textsuperscript{1,28,32,33} cannot provide adequate information for determinations of the magnitude and direction of the alteration in 201Tl content in the two myocardial regions that form these ratios. The determination of the absolute change of 201Tl in these regions would require knowledge of the actual activity of the tracer in the tissue as a function of time. Not knowing this 201Tl content, we would like to extrapolate the change in myocardial 201Tl concentration from our measurement of microsphere distribution at different phases of the experiment. We will review the observations made in the two groups of dogs with either high or low LCX flow during the adenosine infusion and analyze the data from experiments lasting longer than 80 minutes. Both groups had similar initial flow and scintigraphic disparities as well as homogeneous coronary perfusion during 201Tl redistribution. The high-flow group shows a higher LCX as well as LAD flow during the 201Tl injection and also shows a more rapid disappearance of the scintigraphic defect. Therefore, this group should also have a higher initial 201Tl content in both LAD and LCX regions. Regions with higher 201Tl content show a faster rate of net washout. For R\textsubscript{scan} and R\textsubscript{T1} to approach unity faster in the high-flow group, its LAD must wash out faster or its LCX must wash out slower than the corresponding region in the low-flow group. However, the LCX in the high-flow group has a higher 201Tl content than that in the low-flow group and would not, therefore, have a slower net washout. Therefore, we conclude that the disappearance of a defect is primarily caused by washout of 201Tl from the initially normal area, while the change in the defect area should be minimal. This final conclusion suggests that the “filling in” of a defect after vasodilator administration may be a misnomer because tracer content may remain relatively unaltered in the region with lower initial flow and the rapidity of redistribution can be accounted for by relatively faster changes that occur in the 201Tl content of the initially higher flow regions.

The current experiments were conducted in states where transiently elevated flow was produced in at least one portion of the myocardium and there were no regions of abnormally low flow. Similar conclusions should not be made when a transient 201Tl scan defect is noted at rest or with ischemia resulting from absolutely low regional coronary perfusion.

This study shows that 201Tl redistribution occurs after vasodilator administration and can be detected by standard gamma camera imaging. The observation of transient defects on serial 201Tl scans after a vasodilator infusion should be interpreted as viable myocardial regions associated with a critical arterial stenosis that limits the initial regional flow response to vasodilatation.

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