The Effect of Transient Ischemia with Reperfusion on Thallium Clearance from the Myocardium

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SUMMARY Serial myocardial imaging after a single dose of thallium is often used in evaluating patients with coronary artery disease. Because the appearance of the image is related to myocardial thallium content, it is important to know if transient ischemia occurring after tracer administration will influence the rate of myocardial thallium clearance. To answer this question 13 mongrel dogs (eight study dogs and five controls) were studied as follows. The left anterior descending (LAD) coronary artery and the anterior interventricular vein (AIV) were isolated. A catheter was inserted in the AIV to collect venous blood draining from the myocardium in the LAD distribution. Next, a snare was positioned around the LAD proximal to the tip of the AIV catheter. Thallium was given intravenously and paired samples of arterial and AIV blood were obtained every 10 minutes for 1 hour to determine thallium activity and potassium and hydrogen ion concentration. The LAD was then occluded for 10 minutes and paired blood samples were obtained. Next, the snare was released and paired blood samples were obtained beginning 10 seconds, 4, 10 and 20 minutes after reperfusion. Regional myocardial blood flow at the time of thallium administration and during the coronary occlusion was determined by the microsphere technique. After 20 minutes of reperfusion, the dog was sacrificed. The hearts were sectioned and counted for thallium and microsphere activity. During the coronary occlusion, the rate of thallium clearance (µCi/min × 10^-2) from myocardium drained by the AIV (-1.1 ± 0.3, mean ± SEM) was similar to the rate just before occlusion (-0.68 ± 0.21, p = NS), as well as to the rate during the first minute of reperfusion (-0.77 ± 0.55, p = NS). In contrast, compared with the rate (µCi/min × 10^-3) just before occlusion (-0.55 ± 0.61) a significant (p < 0.01) increase in output of potassium (-7.15 ± 1.04) by myocardium in the AIV distribution was observed during the coronary occlusion. Likewise, hydrogen ion output (mEq/min × 10^-11) also increased significantly (p < 0.05) during the occlusion (-12.26 ± 0.94) compared with the rate just before occlusion (-6.98 ± 0.78). At postmortem examination the ratio of thallium activity in the ischemic zone to that of the normal zone (1.01 ± 0.01) did not differ significantly from the ratio of thallium activity of the LAD to that of the circumflex region (1.04 ± 0.05) in control dogs in which no occlusion was performed.

We conclude that transient ischemia occurring after thallium administration does not influence the normal rate of thallium clearance from the myocardium and thus may not be detected on serial scans.

SERIAL MYOCARDIAL IMAGING after a single dose of thallium has been used in evaluating patients with coronary artery disease in a variety of clinical circumstances, including acute myocardial infarction,1,2 variant and unstable angina,3,4 after exercise-induced myocardial ischemia,5,6 and at rest in the absence of symptoms or signs of acute ischemia.7 Extension or recurrence of myocardial ischemia after tracer administration could occur in any of the first three mentioned clinical conditions. In a previous study8 it was demonstrated that once thallium was taken up by the myocardium, 2 hours of subsequent, sustained ischemia failed to affect the normal rate of clearance of the isotope from the infarct zone. Clinically, the results of that study imply that extension of infarction into previously normal myocardium may not be associated with a new or enlarged defect on serial images unless there is an associated change in the geometry of the left ventricle.8 We do not know if the same conclusion applies when transient ischemia is followed by reperfusion. Under such circumstances thallium could leak from ischemic cells and be washed out of the ischemic area during reperfusion. The following study was performed in order to study this question in more detail.

Materials and Methods

Experimental Preparation (fig. 1)

Eight mongrel dogs (mean weight 28 kg, range 24-33 kg) were anesthetized with sodium pentobarbital (30 mg/kg i.v.), intubated, and ventilated on a volume respirator using 2 l/min of oxygen mixed with room air and 3 cm of positive end-expiratory pressure. Sodium bicarbonate was given intravenously and the ventilator was adjusted to maintain the pH of arterial blood at 7.35-7.45, and the Pco2 at 25-35 mm Hg. The partial pressure of oxygen was maintained above 100 mm Hg. A large-bore cannula (#10F) was placed in the right femoral artery and connected by means of plastic tubing (12.5 mm i.d.) to a closed collection bag. A valve was placed in the line and opened as needed to release blood from the dog to maintain mean arterial pressure at 95-110 mm Hg. It was sometimes necessary to do this early in the procedure when spontaneous mean arterial pressures exceeded these limits. The right femoral vein was cannulated with a large-bore catheter (#18F) and connected by plastic tubing.
FIGURE 1. Schematic diagram of the experimental preparation. A catheter inserted in the anterior interventricular vein (AIV) is used to collect blood draining from the myocardium in the distribution of the left anterior descending (LAD) coronary artery. Between sampling periods the AIV blood is collected in a basin (placed at the level of the right atrium) and returned to the dog at frequent intervals via the open funnel. Blood can be released as needed from the femoral artery into the reservoir bag to maintain mean arterial pressure in the desired range. The transient LAD occlusion is made at a level 1.5–2.5 cm proximal to the distal end of the AIV catheter.

(12.5 mm i.d.) to an open reservoir. Whole blood from donor animals and/or normal saline was transinfused into the dog from the reservoir after first passing through a blood filter and heat exchange system. Fluids were given to replace losses and to ensure that mean arterial pressure did not fall below 95 mm Hg. Additional 20-cm vinyl catheters were placed in the left femoral and right brachial veins for administration of thallium and medications. Another catheter was introduced into the left femoral artery to monitor arterial pressure. A 23-cm vinyl catheter (1.8 mm i.d.) placed in the right brachial artery was attached to a Holter pump in order to obtain reference samples for microsphere determination of regional myocardial blood flow.10 After all catheters were in place, the chest was opened by a left thoracotomy and the heart suspended in a pericardial cradle. A #8 Sones catheter was placed in the left atrium and held in place by a pursestring suture. Next, the anterior interventricular vein (AIV) was dissected free and the distal end of a shortened (10 cm) #7 Sones catheter inserted into it. The catheter tip was advanced distally approximately 1.5–2.5 cm until it was positioned at a level between the second and third diagonal branches of the left anterior descending (LAD) coronary artery. The LAD was then dissected free just above the second diagonal branch and an umbilical tape positioned at that point. The open end of the Sones catheter was connected to a 100-cm plastic tube (3.0 mm i.d.) that fit snugly over the end of the catheter. The length of tubing with an estimated dead space of 7 ml was required to facilitate collection of blood samples from the open end of the AIV catheter. Before occlusion and after reperfusion of the LAD, blood flow in the line always exceeded 7 ml/min. Because representative AIV samples were collected no more frequently than every 5–10 minutes, the dead space was always completely replaced before each new collection. Furthermore, 5 minutes were allowed to elapse before beginning the blood collections during occlusion. Thus, blood that collected in the tubing before the occlusion was not mixed with samples obtained during occlusion. Blood dripped freely through the tubing and was collected continuously in a basin placed level with the right atrium. The basin was emptied at frequent intervals and the blood returned to the dog via the overflow reservoir. ECG lead II and systemic arterial and left atrial pressures (Statham P23Db transducers) were monitored continuously throughout the experiment and recorded on paper with a Hewlett Packard recorder (Model 7788A).

Experimental Protocol

The experimental protocol is illustrated in figure 2. Blood draining from the AIV was collected for 1 minute in a graduated cylinder. At the same time heart rate and mean arterial and left atrial pressures were recorded. Samples of arterial and AIV blood were obtained for baseline determinations of background radioactivity, pH, PCO₂ and potassium concentration. Background radioactivity and potassium concentration were determined 1–2 minutes later in arterial and AIV blood. Then, approximately 4 million commercially available (New England Nuclear, Billerica, Massachusetts), ruthenium-103-labeled microspheres (15 ± 3 μCi; total activity approximately 85 μCi) were given via the left atrium to determine regional myocardial blood flow. Studies by other investigators11, 12 have shown that microspheres of this size do not impair myocardial function when given in comparable numbers. The spheres were suspended in a solution of 10% Dextran and 0.01% Tween-80 and sonically dispersed for 15 minutes before injection. Immediately after completion of the 2-minute reference collection of arterial blood, approximately 600 μCi of thallium was given intravenously. At 10, 20, 30, 40, 50 and 60 minutes after thallium administration, paired samples of arterial and AIV blood were obtained for determination of thallium activity and potassium concentration. Blood flow in the AIV line was determined at each time period by collecting the effluent in a graduated cylinder for 1 minute. Arterial and AIV pH and PCO₂ were measured again 60 minutes after thallium was given. The dog then was given 3–5 mg/kg of bretyllium intravenously and after arterial pressure had returned to control levels an atrumatic vascular clamp was placed on the LAD just proximal to its second diagonal branch. One minute later, 4 million scandium-46-labeled microspheres (15 ± 3 μCi; total activity approximately 95 μCi) were injected into the left atrium. At 5 and 10 minutes after occlusion, AIV flow was again measured and samples of arterial and AIV blood were obtained for determination of
thallium activity and potassium concentration. The pH and PCO₂ of arterial and AIV blood were measured 7 minutes after occlusion. After 10 minutes of LAD occlusion the clamp was released. Flow in the AIV line was determined again beginning at 10 seconds, 4, 10 and 20 minutes after reperfusion. At the start of reperfusion, 10 seconds were allowed to pass before beginning the AIV blood collection to ensure that blood that had collected in the tubing during the occlusion was washed out and not mixed with the initial reperfusion blood sample. Arterial and AIV blood samples were also obtained at each of these time periods for determination of thallium activity and potassium concentration. A final sample of arterial and AIV blood was obtained 20 minutes after reperfusion for determination of pH and PCO₂. Heart rate and mean arterial and left atrial pressures were recorded continuously throughout the experiment. Immediately after the final collection period (i.e., 20 minutes after reperfusion) the dog was sacrificed by rapidly excising the beating heart.

Five additional dogs were prepared and studied in the same fashion as described above except that the LAD, although dissected free, was not occluded. This control group served to determine the natural time course of changes in thallium activity in arterial and AIV blood.

**Determination of Thallium Activity**

**Blood Samples**

Precisely measured 1-ml aliquots of arterial and AIV blood were placed in plastic vials and counted for 5 minutes in a gamma well counter (Packard Instruments, Downers Grove, Illinois) using a 27-280 keV window-width setting. Thallium activity in the blood first was expressed as counts per minute per milliliter (CPM/ml) corrected for background. To convert thallium activity from CPM/ml to μCi/ml, 1-ml samples of known activity (μCi) were placed in the well counter and CPM at the above-noted energy settings recorded. A conversion factor (μCi/CPM) was derived from these data, which permitted measured CPM/ml in each sample to be expressed in terms of μCi/ml. Once thallium activity (μCi/ml) in the blood was known, net rate of uptake or loss of the tracer (NETup/to [μCi/min]) by the myocardium could be calculated as:

\[ \text{NET}_{\text{up/to}} = T_{\text{lin}} - T_{\text{out}} \]

where \( T_{\text{out}} \) is thallium output by the myocardium (AIV flow [ml/min] × AIV Tl activity [μCi/ml]), \( T_{\text{in}} \) is thallium input to the myocardium (arterial flow [ml/min] × arterial Tl activity [μCi/ml]), and arterial flow is assumed equal to AIV flow.

**Tissue Samples**

The heart was thoroughly washed in tap water and blotted dry. After removing the atria and the free wall of the right ventricle and trimming off epicardial fat and blood vessels, the left ventricle was sectioned as follows. A tissue block approximately 6 × 3 cm at the base of the heart in the distribution of the circumflex coronary artery was removed and divided into cubes of approximately equal size (0.6–2.0 g). Each cube was subdivided into endocardial and epicardial halves. The samples were placed in preweighed plastic vials that were then reweighed to determine the precise weight of each sample. Each vial was counted in a well counter for 5 minutes using a 27-280 keV window setting. A computer program was used to correct for ruthenium and scandium activity spilling into the thallium window. The sum total of activity (CPM/g) in all sections from the normal zone was divided by the total weight of all the sections to obtain a weighted mean value for thallium activity in the entire section. The free wall of the left ventricle in the distribution of the transiently occluded LAD was separated from the remainder of the left ventricle and then sectioned and counted in the same fashion as the normal zone. Samples were included in the ischemic zone, however, only if they met specific regional myocardial blood flow criteria.
Regional Myocardial Blood Flow

Regional myocardial blood flow was measured by determining the ruthenium and scandium activity in each tissue sample. The ruthenium window was set at 450-551 keV and the scandium window at 832-1172 keV. A computer program was used to calculate regional myocardial blood flow based on measured microsphere activity in the tissue sample and in a known volume of reference arterial blood.\(^\text{10}\) The mean transmural flow in the normal zone was calculated in the same fashion as the mean transmural thallium activity. Ischemic zone “A” was defined as the set of tissue samples in which 1) control (i.e., ruthenium) flow did not differ from that of the normal zone by more than 12% and 2) occlusion (i.e., scandium) flow was ≤ 45% of that in the normal zone. Ischemic zone “B” was defined in the same fashion except that occlusion flow had to be ≤ 25% of that in the normal zone. A weighted mean value for flow in each ischemic zone was obtained for each set of samples and used for comparison with that of the normal zone.

Determination of Blood Gases and Potassium Concentration

The pH, \(\text{PO}_2\) and \(\text{PCO}_2\) in arterial and AIV blood was determined with a standard blood gas analyzer (Instrumentation Laboratory, Inc., Model 213, Boston, Massachusetts). The potassium concentration of arterial and AIV serum was determined with a flame photometer (Instrumentation Laboratory, Inc., Model 143). To ensure that the AIV blood collected during the temporary coronary occlusion originated from predominantly ischemic myocardium, two checks were used. First, the pH of arterial and AIV blood samples was converted to actual hydrogen ion (\(\text{H}^+\)) concentrations (mEq/l). Net output of hydrogen ion during preocclusion, occlusion and postocclusion phases of the study was calculated in the same fashion as described above for thallium. Second, net uptake or loss of potassium by the myocardium was also determined from paired samples of arterial and AIV blood in the same fashion as described for thallium and hydrogen ion.

Statistical Methods

All results are expressed as mean ± SEM. The significance of differences between means was assessed using a two-way analysis of variance and the Newman-Keuls procedure for multiple comparisons.\(^\text{13}\) In addition, Dunnett’s test was used to perform multiple comparisons with a single control.\(^\text{14}\) The significance of correlations between various parameters was determined by using linear regression analysis.

Results

Hemodynamic and Regional Myocardial Blood Flow Data (table 1)

There was no significant change in heart rate, mean arterial pressure or mean left atrial pressure during the study. Nor was there a significant change in AIV blood flow during the first hour of the study. Flow through the AIV catheter decreased by an average of 55% (\(p < 0.001\) vs control) during the coronary occlusion, then increased twofold (\(p < 0.01\) vs control) in the immediate reperfusion period, and then returned to control 10 minutes after release of the coronary occlusion.

Regional myocardial blood flow determined by the microsphere technique showed that control (ruthenium-103) flow (\(\text{ml} \cdot \text{min}^{-1} \cdot \text{g}^{-1}\)) was nearly identical in myocardium supplied by the circumflex (1.19 ± 0.16) and LAD (1.16 ± 0.17) coronary arteries (\(r = 0.99, p < 0.001\)). One minute after occlusion of the LAD, flow did not change significantly in the circumflex region (1.23 ± 0.06) but did decline considerably in myocardium distal to the LAD occlusion (0.37 ± 0.05 in ischemic zone “A” \([p < 0.001]\) and 0.15 ± 0.02 in ischemic zone “B” \([p < 0.001]\)). The ratio of flow in ischemic myocardium to that in normal myocardium was 0.30 ± 0.03 for zone “A” and was 0.13 ± 0.02 for zone “B”.

Metabolic Data

Net myocardial output of hydrogen ion (mEq/min \(\times 10^1\)) increased significantly (\(p < 0.05\)) during the coronary occlusion (12.26 ± 1.94) compared with values before thallium (6.79 ± 0.27), 60 minutes after thallium (6.98 ± 0.78) and 20 minutes after reperfusion (7.11 ± 1.46). During the first hour after thallium administration, net myocardial uptake/loss of potassium did not differ significantly from zero at any time (fig. 3). Because the values of AIV flow and arterial and AIV potassium concentration were not

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<td><strong>AIV flow</strong></td>
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**FIGURE 3.** Bar graph depicting the net rate of myocardial uptake/loss of potassium. There is no significant difference in any of the rates during the first hour after thallium administration. Transient ischemia caused significant (p < 0.001 vs value 30 minutes after thallium) efflux of potassium from the myocardium. Reperfusion is associated with a definite trend in the direction of re-uptake of potassium by the myocardium. The "1 minute" reperfusion bar represents the value for a blood collection starting 10 seconds after release of the left anterior descending coronary artery occlusion.

significantly different 5 and 10 minutes after occlusion, each was averaged and a single value used to represent the change during the occlusion. There was a net efflux of potassium (mEq/min × 10⁻⁵) from the myocardium drained by the AIV (-7.15 ± 1.01, p < 0.001 vs rate 30 minutes after thallium) during the coronary occlusion. In addition, during reperfusion there was a trend in the direction of net uptake of potassium by myocardium in the AIV distribution (fig. 3). Each value of potassium uptake during reperfusion differed significantly (p < 0.01) from the value during occlusion, though none differed significantly from the value 30 minutes after thallium administration. In the control dogs there was no significant change in net myocardial output of hydrogen ion at any time. Likewise, net myocardial uptake/loss of potassium did not differ significantly from zero at any time.

Thallium Clearance From the Myocardium

The value for net myocardial uptake/loss of thallium are shown in figure 4. Net uptake of the tracer ceased approximately 20-30 minutes after thallium administration. Thereafter, a net loss of tracer from the myocardium was observed at each time period. Thallium activity in arterial and AIV blood was not significantly different 5 and 10 minutes after occlusion. Accordingly these values were averaged and a single value was used to represent the value of each during the occlusion. At no time did the net clearance rate of thallium differ significantly from the value at 30 minutes. Similar results were obtained in the control dogs (fig. 4). Postmortem determination of thallium activity in the ischemic and normal zones supports these findings. At the end of the study the ratio of thallium activity (ischemic : normal) was 1.01 ± 0.01 in the tissue from ischemic zone “A” and 1.03 ± 0.01 in samples from ischemic zone “B,” despite a highly significant fall in blood flow during the coronary occlusion and marked reactive hyperemia after release of the occlusion. Finally, the ischemic : normal thallium ratio in the study dogs did not differ significantly from the LAD : circumflex region thallium ratio in the control dogs (1.04 ± 0.05).

**Discussion**

**Critique of Method**

Before discussing the results of the study it is important to consider potential limitations of the experimental technique used to collect venous blood draining from the myocardium in the distribution of the LAD. The principal limitation of this method relates to uncertainty over which regions of the myocardium contribute venous blood to the effluent draining from the AIV catheter. A related issue concerns how accurately true regional myocardial blood flow can be estimated from measurements of flow through the catheter-tubing collection system.

In five of eight dogs, the percent decrease (vs control) in regional myocardial blood flow during the
LAD occlusion determined by the venous collection method (table 2) was almost identical to that of the microsphere method ($r = 0.97, p < 0.01$). In the remaining three dogs, the venous collection technique overestimated regional flow compared with the microsphere technique (table 2, dogs 3, 5 and 6), probably because the AIV catheter received additional blood from border zone ischemic tissue (where flow was better preserved), whereas the myocardial sample finally used for the microsphere determination of flow represented a central zone of more intense ischemia. However, despite the lack of close agreement between the two techniques in three dogs, flow in the AIV catheter did decrease in each during the LAD occlusion. In addition, in the first minute of reperfusion, flow in the catheter increased by $102 \pm 8\%$ over control levels in these three dogs (range 85-128%) compared with $75 \pm 11\%$ in the other five (range 42-115%). Moreover, the net rate of potassium efflux from the myocardium recorded during the occlusion in these three dogs was comparable to that of the other five dogs (table 2). Further, in these three dogs, the final myocardial ischemic : normal zone thallium ratio was similar to that of the other five dogs (table 2). If ischemia had caused thallium to "leak" from the myocardium and if reperfusion then washed the isotope away, we would expect to find that transiently ischemic myocardium contained significantly less thallium at the end of the study than the reference zone, whether or not we were able to collect all or only some of it in the AIV catheter. In fact, no tissue deficit of thallium was observed. It is unlikely that thallium leaked during ischemia and then was taken up again by the myocardium during reperfusion because during reperfusion the AIV collections continued to show a net loss of thallium that did not differ significantly from control levels (fig. 4A). In contrast, potassium fluxes determined from the same blood samples indicated a clear trend in the direction of net accumulation of the ion during the reperfusion phase of the study (fig. 3). These observations indicate that directional changes in AIV flows reflect directional changes in microsphere determinations of regional myocardial blood flow, and that blood collected from the AIV catheter came predominately, although perhaps not exclusively, from ischemic myocardium. Accordingly, the use of timed samples of AIV blood to help calculate the net rate of thallium clearance from the myocardium probably provides a reasonable estimate of the true net clearance rate.

**Experimental Results**

The results of the present study indicate that transient myocardial ischemia occurring after thallium administration does not affect the normal net rate of myocardial thallium clearance. These results are in accord with a previous study in which 2 hours of persistent ischemia failed to influence the normal net rate of myocardial thallium clearance. The explanation for this observation may be related to the rate at which thallium is released from myocardial cells. Clearly, thallium cannot be removed from the heart until it is released from the cells. If the amount of tracer (i.e., $\mu$Ci/min in excess of arterial input) released by the cells remains relatively constant, increasing or decreasing flow will have little effect on the resultant rate at which thallium is lost from the myocardium, provided of course that flow is not zero. Therefore, we would expect a significant change in the net rate of thallium clearance from the myocardium only if ischemia actually altered the rate at which thallium was released from the cells into the blood.

However, thallium release from myocardial cells was not influenced by ischemia in our study. Despite a substantial fall in blood flow in the ischemic region during the coronary occlusion, there was no significant change in the net clearance rate of thallium compared with preocclusion values. Likewise, in the first minute of reperfusion, although flow in the ischemic area increased almost fourfold over occlusion levels, the net clearance rate of thallium again remained unchanged. In contrast, the net clearance rate of both potassium and hydrogen ion increased during coronary occlusion. This result is in accordance with the data of other authors who have shown enhanced release of potassium and increased production of hydrogen ions in regions of myocardial ischemia.15-17 In addition,
during reperfusion there was a trend in the direction of net myocardial uptake of potassium but not of thallium, which is consistent with the concept that transient ischemia accelerated the rate of loss of potassium but not of thallium from the myocardium.18

The postmortem material also is in accord with this hypothesis. Thallium activity at the end of the experiment in samples from both ischemic areas was comparable to that of the normal zone despite a substantial fall in blood flow during occlusion and brisk reactive hyperemia during reperfusion.* Furthermore, the fact that the ischemic : normal thallium ratio was comparable to that of the control dogs in tissue with the greatest reductions in regional myocardial blood flow (i.e., ischemic zone "B") indicates that even very intense myocardial ischemia does not appear to augment loss of thallium from myocardial cells.

One additional point should be made concerning the interpretation of the results of this study. Previous work by other investigators19, 20 has shown that thallium clearance from ischemic myocardium is delayed in comparison with the rate of clearance from normal myocardium. Although it appears to, this observation in fact does not conflict with the results of the present study.

Different experimental designs were used to answer fundamentally different questions. In the experiments of other investigators, thallium was administered after rather than before the onset of myocardial ischemia, as in the present study. When thallium is given after the onset of ischemia, there is active but delayed uptake of the tracer by viable cells in the ischemic area, resulting in a decrease in the net rate at which thallium leaves the ischemic zone.19, 20 In contrast, we sought to determine if regions of myocardium that had already achieved maximal attainable levels of thallium would lose the tracer at an accelerated rate if they were subjected to ischemia followed by reperfusion. We found under these circumstances that they did not and that the net rate of loss of the tracer from the myocardium was unaltered compared with that of the normal zone. Thus, the two types of experiments are different and the results should not be interpreted as contradictory.

In conclusion, transient myocardial ischemia occurring after thallium administration did not influence the normal rate of thallium clearance from the myocardium. Therefore, myocardial thallium content is unlikely to decrease abruptly under such circumstances. Clinically, this implies that serial imaging in the setting of acute myocardial infarction, variant or unstable angina, or after exercise stress testing may not detect extension or recurrence of myocardial ischemia.

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References


*We can assume that thallium activity was essentially equal in the two zones at the start of the experiment because microsphere-determined flow was essentially equal in the two areas just before tracer administration. Our own data as well as those of others clearly indicate that the initial distribution of the isotope is largely dictated by the distribution of regional blood flow. Therefore, regions with comparable flows at the time of thallium administration also attain comparable myocardial concentrations of the isotope.
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