Effect of glucose-insulin-potassium infusion on thallium myocardial clearance

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ABSTRACT Factors influencing the rate of 201TI clearance from the myocardium have not been clearly defined. This study determined the effect of an intravenous infusion of glucose-insulin-potassium (GIK) on the net 201TI clearance rates from myocardium with and without initial 201TI loading. Anesthetized open-chest dogs underwent 5 min of left anterior descending coronary artery occlusion and intravenous 201TI was injected and the occlusion released 5 min later. Thirty minutes after 201TI injection, 30 ml of either GIK (nine dogs) or saline (five dogs) was infused intravenously. The clearance rates of 201TI from the anterior wall (without initial 201TI loading) and from the posterior wall (with initial 201TI loading) were monitored with miniaturized cadmium telluride detectors placed on the myocardium. Calculation of net myocardial clearance rates was performed by linear regression analysis from serial 1 min counts. Compared with saline infusion, GIK increased the net clearance of 201TI from both myocardial regions with and without initial loading. The most marked change induced by GIK infusion was in the myocardial region without initial 201TI loading; a net increase in 201TI activity (72 ± 42 cpm/30 min) was converted into a net loss (−594 ± 228 cpm/30 min). There was no significant change in 201TI clearance after the saline infusion. Heart rate, aortic and left atrial pressure, somatometer-measured transmural myocardial wall thickness, microsphere-determined myocardial blood flow, and blood glucose and potassium concentrations did not change significantly during GIK or saline infusions. Thus, GIK infusion appears to increase net 201TI clearance from myocardial zones with and without initial 201TI loading.


ALTHOUGH previous studies have defined the relationship of initial 201TI distribution in the myocardium to regional myocardial blood flow,1 relatively little information is available regarding the factors influencing the rate of 201TI loss in normal and ischemic myocardium. Results of recent studies2–4 suggest that the rate of 201TI loss from the myocardium can be used as a criterion for the detection of myocardial ischemia. Approximately 20% of 201TI should be lost by the myocardium over a minimum 4 hr period in normal individuals.2 Some patients have an unusually rapid loss of 201TI from the myocardium, with a decrease of over 60% in 201TI activity in normal myocardium in the time between the recording of initial and delayed images (recorded 4 hr later). Preliminary investigations have suggested that rapid loss of 201TI is associated with the ingestion of a high-carbohydrate meal between initial and delayed imaging sessions. Since many investigators believe the kinetics of 201TI in the myocardium are similar to those of potassium,5,6 we investigated the possibility that the myocardial 201TI clearance time is influenced in part by insulin. Results of previous studies by Weich et al.7 suggested that the initial extraction of 201TI by the myocardium was not influenced by insulin. However, that study did not determine whether the myocardial release rate of 201TI was influenced by the hormone. Accordingly, our purpose was to define the impact of an intravenous infusion of glucose-insulin-potassium (GIK), with and without initial 201TI loading, on the clearance of 201TI from myocardium in dogs.

Methods

Fourteen adult mongrel dogs (mean weight 22 kg, range 18 to 27) were anesthetized with chloralose (140 mg/kg) and urethane (1400 mg/kg iv), intubated, and ventilated with a Harvard respirator apparatus with 5 cm of positive end-expiratory pressure. The heart was exposed by a left thoracotomy and was suspended in a pericardial cradle. Supplemental oxygen was used to maintain an arterial Po2 greater than 70 mm Hg. Miniature cadmium

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telluride (CdTe) radiation detection probes were placed through the left ventricular apex and positioned against the endocardial surface of the anterior and posterior walls to measure regional myocardial \(^{201}\)TI activity. In the anterior wall the detector was positioned to face toward the open thoracotomy, thus markedly reducing the detection of background activity. A lead sheet was placed between the posterior wall and pericardium to reduce the background activity detected by the posterior wall probe. Previous validation studies have shown that the lead shielding around the crystal effectively reduces contamination from blood pool activity. The probes were connected via a preamplifier to a multichannel analyzer (Canberra Corp., Meriden, CT) to continuously monitor and display \(^{201}\)TI activity in the anterior and posterior walls. Probe background activities were determined before \(^{201}\)TI injection as the average of three 1 min collections. The \(^{201}\)TI activity data were acquired as serial 1 min collections. These count vs time data were displayed on the multichannel analyzer screen and recorded on paper tape. The CdTe radiation probe consisted of a 2 mm² CdTe crystal surrounded by lead foil that was housed in a steel (4 mm od) cylinder attached to an arterial clamp. In addition, sonomicrometer crystals (3 mm in diameter) were attached to both arms of the arterial clamp and connected to a sonomicrometer (Norland Instruments model NL-202-4R, Fort Atkinson, WI). These ultrasonic crystals permitted frequent determination of anterior and posterior left ventricular wall thickness. The monitoring of wall thickness changes was performed to exclude changes in wall thickness as a cause of changes in \(^{201}\)TI activity recorded by the CdTe probe. 

Care was taken not to position the probe over a coronary artery branch. To induce myocardial ischemia at the time of \(^{201}\)TI injection, the left anterior descending coronary artery was dissected free and a ligature was loosely placed around the vessel. Aortic and left atrial pressures were monitored by 20 cm vinyl catheters attached to Statham P23Db pressure transducers and were recorded on paper with a Sanborn 7700 series recorder. To sample coronary sinus \(^{201}\)TI activity, a coronary sinus catheter (No. 7F modified NIH catheter) was placed through the right atrial appendage. 

The experimental protocol is illustrated in figure 1. Baseline blood gas, aortic and left atrial pressure, heart rate, and wall thickness measurements were obtained. The left anterior descending coronary artery was then ligated. After 5 min of occlusion, \(^{201}\)TI (1.2 to 1.4 mCi) and approximately 4.5 million \(^{113}\)Sn-labeled microspheres (15 μm diameter, 30 μCi total activity, New England Nuclear Corp., North Billerica, MA) were simultaneously injected intravenously and into the left atrium, respectively. Aortic blood was drawn with a Holter pump to obtain reference samples for microsphere determination of regional myocardial blood flow. Five minutes after \(^{201}\)TI administration, the coronary artery ligation was released. Serial 1 min regional counts were acquired with the CdTe detectors from the time of \(^{201}\)TI injection and this was continued for the duration of the study (2 hr). In all dogs, the probe-recorded \(^{201}\)TI activity was at least 1000 cpm (mean ± SEM = 4929 ± 466). Thirty minutes after \(^{201}\)TI injection (25 min after ligation release) the intravenous infusion of 30 ml GIK (glucose [10% dextrose in water], regular insulin [12 U], and potassium [16 mEq]) was begun in nine dogs at a rate of 1 ml/min for 30 min. Five additional control dogs were given an intravenous infusion of normal saline instead of GIK. Just before, during (after 20 to 30 min of the infusion), and 1 hr after the infusion of GIK (or saline), venous blood samples for glucose and potassium and arterial blood samples for pH, PO\(_2\), and PCO\(_2\) were obtained. In addition, serial aortic and coronary sinus blood samples (1 ml) for \(^{201}\)TI were obtained simultaneously at specific times before, during, and after the GIK or saline infusion (2, 4, 6, 8, 10, 15, 20, 25, 28, 29, 30, 31, 32, 33, 35, 40, 45, 50, 55, 60, 70, 80, 90, 100, 110, and 120 min after \(^{201}\)TI injection, figure 1). \(^{103}\)Ru- and \(^{46}\)Sc-labeled microspheres were injected during GIK or saline infusion, respectively, and at the end of the study. The total number, mean size, and total radioactivity of the \(^{103}\)Ru- and \(^{46}\)Sc-microspheres were the same as for the \(^{113}\)Sn-microspheres. No changes in probe counts were detected during microsphere administration. During the 120 min of the experiment, no bicarbonate, heparin, or fluid boluses were administered. The dogs were killed 120 min after \(^{201}\)TI injection.

Myocardial regions around the CdTe probes in the areas with and without \(^{201}\)TI loading were excised and later divided into 48 transmural sections (of approximately 1 g each). Microsphere and \(^{201}\)TI activities were determined in the different sections of myocardium by well counting (Auto-Gamma Scintillation Spectrometer, Packard Instruments Company, Downers Grove, IL) for 5 min to collect at least 10,000 counts for each isotope. The myocardial and blood microsphere reference samples were counted after \(^{201}\)TI activity had decayed one to two half-lives. Serial aortic and coronary sinus blood samples were analyzed for \(^{201}\)TI in the well counter within 24 hr. The \(^{113}\)Sn was counted within a 350 to 435 keV window, the \(^{103}\)Ru within a 460 to 760 keV window, the \(^{46}\)Sc within a 800 to 1200 keV window, and the \(^{201}\)TI within a 60 to 110 keV window. Appropriate corrections were made for higher energy contamination of the \(^{201}\)TI and microsphere energy windows. Regional myocardial blood flow was calculated by a computer program from the sample activity and the activity in the reference blood samples was determined simultaneously with the administration of each isotope. Regional myocardial blood flows determined during left anterior descending coronary artery ligation \((^{113}\)Sn), during GIK or saline infusion \((^{103}\)Ru), and immediately before the dogs were killed \((^{46}\)Sc) were expressed as milliliters per minute per gram. 

Blood and myocardial \(^{201}\)TI activities were expressed as counts per minute per milliliter and counts per minute per gram, respectively. To correct for differences in sensitivity between the two CdTe probes, the serial background-corrected 1 min counts from the CdTe probes were normalized to the average \(^{201}\)TI activity, as determined by well counting in their respective myocardial regions at the end of the study. The \(^{201}\)TI clearance curves were also normalized to the highest \(^{201}\)TI activity recorded for each CdTe probe.

Statistics. The rates of \(^{201}\)TI clearance before, during, and after GIK or saline infusion were calculated by linear regression analysis from the serial 1 min counts and expressed as counts per minute per 30 minutes. Group values were expressed as a mean ± SEM. Difference in clearance rates and transmural wall thickness parameters were assessed by one-way analysis of variance. Differences between coronary sinus and aortic blood \(^{201}\)TI activities before and during infusion were determined by paired t test.

Results

Plots of \(^{201}\)TI activity in myocardium with and without \(^{201}\)TI loading in a representative control dog and in a dog that received a GIK infusion are illustrated in

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**FIGURE 1.** Experimental protocol. LAD = left anterior descending coronary artery; LA = left atrium; mic = microspheres.
FIGURE 2. Representative example of myocardial 201TI time-activity curves in myocardium with and without 201TI loading during infusion of saline. Serial 1 min counts were corrected for background activity and differences in CdTe probe sensitivities and normalized to maximal 201TI activity.

FIGURE 3. Representative examples of myocardial 201TI time-activity curves in myocardium with and without initial 201TI loading during infusion of GIK.
Slope of Thallium Clearance in Normally Perfused Myocardium

![Graph](image)

**FIGURE 4.** The slopes of the $^{201}$Tl time-activity curves before, during, and after infusion in myocardium with initial $^{201}$Tl loading (posterior wall). Saline infusion caused no change in $^{201}$Tl clearance. GIK, however, caused a significant increase in myocardial $^{201}$Tl clearance.

The infusion of either GIK or saline was not associated with significant changes in heart rate, mean aortic or left atrial pressure, sonomicrometer-measured transmural myocardial wall thickness, or anterior/posterior myocardial blood flow ratio (table 1). Serum glucose and potassium concentrations and arterial pH, $\text{PCO}_2$, and $\text{PO}_2$ did not change significantly during the experiment (table 2).

To further evaluate changes in myocardial $^{201}$Tl during GIK infusion, coronary sinus blood $^{201}$Tl activity was measured serially before and during GIK or saline infusion (figure 6). GIK infusion was associated with a

Slope of Thallium Clearance in Transiently Ischemic Myocardium

![Graph](image)

**FIGURE 5.** The slopes of the $^{201}$Tl time-activity curves in myocardium without initial $^{201}$Tl loading (anterior wall). The dogs that received infusions of GIK had a decrease in myocardial $^{201}$Tl activity (negative change in slope) while the control dogs (saline infusion) had an increase in activity (positive change in slope) from before to during infusion. With cessation of the infusion, the GIK-infused regions manifested a positive change in slope, reflecting a decrease in the rate of $^{201}$Tl clearance, while the saline-infused regions showed only a slight negative change in slope.
TABLE 1

Hemodynamic variables for two groups of dogs

<table>
<thead>
<tr>
<th></th>
<th>Before infusion</th>
<th>During infusion</th>
<th>After infusion</th>
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<tbody>
<tr>
<td>Heart rate (bpm)</td>
<td></td>
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<td></td>
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<tr>
<td>GIK (n = 9)</td>
<td>154 ± 6.4</td>
<td>149 ± 6.8</td>
<td>151 ± 7.0</td>
</tr>
<tr>
<td>Saline (n = 5)</td>
<td>176 ± 3.7</td>
<td>166 ± 6.8</td>
<td>161 ± 10.5</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GIK</td>
<td>88 ± 7.6</td>
<td>86 ± 7.7</td>
<td>86 ± 4.1</td>
</tr>
<tr>
<td>Saline</td>
<td>110 ± 2.0</td>
<td>105 ± 2.2</td>
<td>103 ± 3.7</td>
</tr>
<tr>
<td>Mean left atrial pressure (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GIK</td>
<td>4.4 ± 0.8</td>
<td>3.9 ± 0.9</td>
<td>4.5 ± 0.8</td>
</tr>
<tr>
<td>Saline</td>
<td>5.8 ± 1.1</td>
<td>4.6 ± 0.9</td>
<td>3.8 ± 0.9</td>
</tr>
<tr>
<td>Anterior wall transmural diastolic thickness (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GIK</td>
<td>8.0 ± 0.5</td>
<td>8.1 ± 0.5</td>
<td>8.1 ± 0.5</td>
</tr>
<tr>
<td>Saline</td>
<td>7.7 ± 0.7</td>
<td>7.7 ± 0.6</td>
<td>7.7 ± 0.6</td>
</tr>
<tr>
<td>Posterior wall transmural diastolic thickness (mm)</td>
<td></td>
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</tr>
<tr>
<td>GIK</td>
<td>9.4 ± 0.8</td>
<td>9.5 ± 0.8</td>
<td>9.3 ± 0.8</td>
</tr>
<tr>
<td>Saline</td>
<td>8.4 ± 0.6</td>
<td>8.5 ± 0.7</td>
<td>8.5 ± 0.8</td>
</tr>
<tr>
<td>Anterior/posterior wall myocardial blood flow ratio&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>GIK</td>
<td>0.06 ± 0.02&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.85 ± 0.15</td>
<td>0.82 ± 0.37</td>
</tr>
<tr>
<td>Saline</td>
<td>0.07 ± 0.03&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.79 ± 0.14</td>
<td>0.85 ± 0.10</td>
</tr>
</tbody>
</table>

<sup>a</sup>Preinfusion values are those for the period during left descending coronary artery occlusion. During and postinfusion values are those for the period after coronary artery reperfusion.

<sup>b</sup>p < .05 compared with "during infusion" value.

29% rise in coronary sinus blood <sup>201</sup>Tl activity (p < .01), while no significant change occurred in coronary sinus blood <sup>201</sup>Tl activity during saline infusion. After the GIK infusion was stopped, coronary sinus blood <sup>201</sup>Tl activity decreased abruptly. Serial aortic blood samples were also obtained before and during GIK or saline infusion. A 27% increase in aortic blood <sup>201</sup>Tl activity was found with GIK infusion, but not with saline infusion, suggesting that in addition to loss from the myocardium, <sup>201</sup>Tl loss from other sites occurred.

TABLE 2

Laboratory values for the two groups of dogs

<table>
<thead>
<tr>
<th></th>
<th>Before infusion</th>
<th>During infusion</th>
<th>After infusion</th>
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</thead>
<tbody>
<tr>
<td>Serum glucose (mg/dl)</td>
<td></td>
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<tr>
<td>GIK (n = 9)</td>
<td>144 ± 26</td>
<td>152 ± 34</td>
<td>107 ± 21</td>
</tr>
<tr>
<td>Saline (n = 5)</td>
<td>108 ± 14</td>
<td>105 ± 15</td>
<td>98 ± 10</td>
</tr>
<tr>
<td>Serum potassium (mEq/l)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GIK</td>
<td>2.8 ± 0.4</td>
<td>3.8 ± 0.5</td>
<td>3.1 ± 0.4</td>
</tr>
<tr>
<td>Saline</td>
<td>2.8 ± 0.1</td>
<td>2.7 ± 0.1</td>
<td>2.6 ± 0.3</td>
</tr>
<tr>
<td>pH</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GIK</td>
<td>7.36 ± 0.03</td>
<td>7.32 ± 0.02</td>
<td>7.27 ± 0.01</td>
</tr>
<tr>
<td>Saline</td>
<td>7.37 ± 0.03</td>
<td>7.37 ± 0.03</td>
<td>7.39 ± 0.03</td>
</tr>
<tr>
<td>Pco&lt;sub&gt;2&lt;/sub&gt; (mm Hg)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>GIK</td>
<td>42.6 ± 2.4</td>
<td>42.1 ± 2.6</td>
<td>45.8 ± 3.5</td>
</tr>
<tr>
<td>Saline</td>
<td>37.5 ± 3.1</td>
<td>36.8 ± 2.9</td>
<td>41.0 ± 2.5</td>
</tr>
<tr>
<td>Po&lt;sub&gt;2&lt;/sub&gt; (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GIK</td>
<td>101.3 ± 11.2</td>
<td>92.6 ± 8.8</td>
<td>86.3 ± 2.4</td>
</tr>
<tr>
<td>Saline</td>
<td>110.0 ± 9.1</td>
<td>101.7 ± 8.4</td>
<td>98.0 ± 3.5</td>
</tr>
</tbody>
</table>

Discussion

The results of this study suggest that an infusion of GIK is associated with an increase in net myocardial <sup>201</sup>Tl clearance in myocardium with and without initial <sup>201</sup>Tl loading. In addition, GIK infusion not only decreased net <sup>201</sup>Tl uptake in a region without <sup>201</sup>Tl loading, but also caused a net washout of <sup>201</sup>Tl for the duration of the infusion. Although our study was performed in anesthetized open-chest dogs and therefore the results may not be directly applicable to the conscious patient, the results suggest that a GIK infusion might affect the rate at which such regions "fill-in" in a serial <sup>201</sup>Tl imaging study. If this effect occurs in man, food ingestion (especially foods rich in carbohydrates and potassium) may cause similar changes in <sup>201</sup>Tl kinetics and redistribution rates. A single 550 g (1/4 lb) hamburger with a bun would contain more carbohydrate (approximately 23 vs 30) and more potassium (23 vs 16 mEq) than the GIK infusion used in this study. Thus, <sup>201</sup>Tl clearance rates from myocardium with and without <sup>201</sup>Tl loading could initially be significantly increased if patients are allowed to eat in...
the time between the recording of initial and delayed images. In addition, the value of quantitative 201TI myocardial clearance rates as a criterion for abnormality on 201TI exercise stress tests3-4 could be enhanced by eliminating the possible GIK effect on 201TI washout. This could be accomplished by requiring patients to fast throughout the imaging period, although this is not current practice in most medical centers. Furthermore, other factors that could alter myocardial 201TI clearance rates must be examined before clearance rate criteria can be applied to the interpretation of data from clinical studies in which 201TI is used.

The myocardial kinetics of 201TI have been studied in dog models. The initial myocardial uptake of 201TI demonstrates a linear relationship to regional myocardial blood flow12 and the myocardial extraction fraction of the agent is about 88%.7 The myocardial uptake rate of 201TI in a flow-independent model is described by a single exponential13 and the uptake rate appears to be unrelated to the extent of reversible injury.14 Some investigators have demonstrated a sodium-potassium—like pump mechanism for myocardial 201TI uptake,15 whereas others have described a passive transport mechanism.5 After peak concentration, normal myocardial 201TI clearance is monoexponential and is related to the rate of clearance from the blood.9 When the peak myocardial 201TI concentration is reduced by decreasing coronary blood flow during 201TI administration, the subsequent clearance of the agent is reduced.16 During the myocardial clearance phase, there is continuous uptake and release of 201TI from the blood pool as the net cellular concentration falls. This dynamic process continues throughout the experimental period.

The exact mechanism by which GIK alters 201TI myocardial clearance is not known, but it is known that insulin stimulates potassium transport into muscle cells.17 It is possible that stimulation of this transport system rapidly increases transport of potassium intracellularly and displaces 201TI from its intracellular location, resulting in a net loss of 201TI. Investigators have reported an insulin-induced increase in myocardial blood flow,18 and it could be questioned whether such an increased flow could have contributed to the increased myocardial 201TI clearance directly. We were unable to determine if insulin increased myocardial blood flow in the current study since the flows determined during GIK infusion and at the end of the experiment were separated by several hours. However, even if an increase in flow did occur after insulin, previous data from our laboratory indicate that it is unlikely that this affected myocardial 201TI kinetics. We have found that doubling myocardial blood flow after 201TI loading has no effect on its myocardial clearance.19

Since 201TI redistribution has been shown to be due to clearance in normal myocardium and to slow uptake in ischemic myocardium,16 the reversal of the ischemic myocardium uptake curve to a clearance curve might be expected to decrease the rate and extent of redistribution. In a preliminary study, 59 patients were prospectively randomized into two groups; 32 patients ate a high-carbohydrate meal between initial and delayed imaging sessions and 27 fasted.20 Myocardial 201TI clearance in the time between initial and delayed imaging was significantly faster for both anterior and posterior walls in the patients who ate. In addition, the patients who ate had a significantly lower incidence of isolated transient defects compared with those in the fasting group.

Since changes in the volume of myocardium (and apparent 201TI activity) under the CdTe radiation detector would be decreased if ventricular wall thinning occurred during the experiment, transmural wall thickness was measured to exclude wall thinning as a possible cause for the change in net 201TI clearance rate observed. The absence of a change in transmural wall thickness discernible by ultrasonography during GIK infusion confirmed that the apparent increase in net 201TI clearance was not due to wall thinning during GIK infusion. In addition, the increase in coronary sinus blood 201TI activity during GIK infusion also suggests that net clearance of the agent had increased in response to the GIK infusion. It is possible, however, that the increased coronary sinus blood 201TI activity was exclusively due to higher aortic blood 201TI activity from noncardiac sources.
The addition of potassium to the infusion mixture of glucose and insulin was believed to be necessary to prevent hypokalemia. The substantial amount of potassium (16 mEq) infused may have had a direct effect on \(^{201}\)TI kinetics independent of the effect of insulin. Recent work by Krivokapich and Shine\(^5\) suggests that potassium infusion does increase \(^{201}\)TI myocardial eflux in an isolated, arterially perfused rabbit interventricular septum preparation when the concentration of potassium in the perfusate is increased from 5 to 10 mm. This change in potassium in the perfusate is much higher than that induced by the intravenous GIK infusion in this study, which did not significantly change serum potassium concentration. Moreover, since potassium infusion itself may stimulate insulin secretion,\(^21,22\) it was not possible for us to distinguish between an insulin effect and a potassium effect on \(^{201}\)TI myocardial clearance. It should be noted that although the changes were not significant, there was a small increase in mean serum potassium level during the GIK infusion. A statistically significant change may have been demonstrated in a larger group of dogs.

In conclusion, the results of our study demonstrate that an intravenous infusion of GIK significantly increases the rate of net \(^{201}\)TI myocardial clearance in myocardium with and without initial \(^{201}\)TI loading. Although the data were obtained in anesthetized open-chest dogs and therefore may not be directly applicable to conscious patients, this possible interaction must be considered when interpreting clearance data derived from clinical serial \(^{201}\)TI imaging studies. Furthermore, GIK may significantly alter the rate of \(^{201}\)TI redistribution in myocardium without initial \(^{201}\)TI loading. Our study also provides a rationale for determining whether patients should fast between initial and delayed \(^{201}\)TI imaging sessions. Other factors that could affect myocardial \(^{201}\)TI clearance rates must be examined before such criteria can be applied to the interpretation of clinical \(^{201}\)TI study results.

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