Quantitative Relationships Between Potassium-43 Imaging and Left Ventricular Cineangiography Following Myocardial Infarction in Man

By Barry L. Zaret, M.D., Stephen C. Vlay, M.D., Gerald S. Freedman, M.D., Steven Wolfson, M.D., and Lawrence S. Cohen, M.D.

SUMMARY
To evaluate the quantitative relationships between resting potassium-43 (\(^{43}\)K) myocardial imaging and left ventricular segmental contraction abnormalities, 15 patients were studied by both radionuclide and contrast angiographic techniques at least two months following transmural myocardial infarction. The ECG location of infarction involved the anterior wall alone in six patients, inferior wall alone in three patients, both anterior and inferior walls in five patients, and in one patient ECG-anatomic correlation was obscured by newly developed left bundle branch block. \(^{43}\)K defects were noted in all patients. Anterior wall \(^{43}\)K defects were noted in all patients with previous anterior infarction and seven of nine inferior infarcts. These \(^{43}\)K defects were associated with a quantifiable decrease in regional radioactivity of at least 20% of normal appearing zones, and their location correlated with the angiographic site of akinesis or dyskinesis. The extent of the \(^{43}\)K defect (% \(^{43}\)K HP [% potassium 43 hypoperfusion]) was measured by planimetry and averaged 49% of the anterior view image (range 25–66%), 43% of the left anterior oblique image (range 0–58%), with the mean of both views being 47% (range 17–62%). The mean total area of the anterior image was 58 cm\(^2\) (range 40–101 cm\(^2\)). The extent of the \(^{43}\)K defect (% \(^{43}\)K HP) was related to the extent of segmental contraction abnormality (% ACS). Correlations between % ACS and anterior view % \(^{43}\)K HP (\(r = 0.67\)), left anterior oblique view % \(^{43}\)K HP (\(r = 0.54\)), and mean % \(^{43}\)K HP (\(r = 0.77\)) were found. The total size of the anterior view image correlated with left ventricular end-diastolic volume (\(r = 0.79\)). Thus, in this initial group of patients following transmural infarction, potassium-43 imaging can be accurately and quantitatively correlated with the site and extent of regional ventricular dysfunction as it is assessed by quantitative left ventricular angiography.

The recent introduction of radionuclide techniques has led to several new approaches of evaluation and investigation of the patient with coronary heart disease. One such approach has involved myocardial imaging following the intravenous injection of a radioactive intracellular cation such as potassium-43 (\(^{43}\)K). This radionuclide is rapidly extracted by cardiac muscle and is distributed in the myocardium in proportion to regional blood flow, intact cell membrane function, and size of the regional intracellular cation pool. In our experience, when obtained under basal conditions in the resting state, myocardial images containing defects or visualized regions of relatively decreased \(^{43}\)K radioactivity have been associated with transmural myocardial infarction and resultant scar.\(^1\), \(^2\) Comparable abnormalities due to noninfarcted transiently ischemic myocardial regions have best been visualized when the radionuclide has been administered during exercise stress.\(^3\), \(^4\) Previous studies from our laboratory have involved qualitative visual assessment of the radionuclide image and correlation with gross angiographic or clinical features. Although the approach to date has been primarily qualitative, the potential of these techniques to quantify infarct size and ischemic myocardial regions is apparent. This report describes our initial attempts at quantification of resting \(^{43}\)K myocardial images in patients with prior transmural myocardial infarction and relates the size and location of the regional defect in \(^{43}\)K accumulation to the site and extent of local left ventricular contraction abnormality demonstrable by quantitative contrast cineangiography.

Methods

Patient Selection
The 15 patients included in this study were referred for angiographic and radionuclide evaluation at least two months following a documented transmural myocardial infarction. Clinical referral had been prompted by either

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postinfarction congestive heart failure, angina, or both. The initial diagnosis of acute myocardial infarction had been made clinically on the basis of at least two of the following: typical chest pain, characteristic evolutionary serum enzyme elevations, and the development of new Q waves of at least 0.04 seconds duration in at least two standard ECG leads. The only criteria for inclusion in this study were the clearcut documentation of previous myocardial infarction, and technically acceptable angiographic and radionuclide studies. Four additional patients were evaluated, were considered for inclusion, and not included because of either ^43K images or contrast angiograms technically unsuitable for quantitative analysis.

Twelve of the 15 patients were male and three were female. The average age was 51 years with a range from 31 to 64 years. At the time of study, ECG localization of prior infarction, and hence residual transmural scar, involved the anterior wall alone in six (anteroseptal in four, anterolateral in two), inferior wall alone in three, both anterior and inferior walls in five patients who had sustained two distinct and separate infarctions, and in one patient ECG-anatomic correlation was obscured at the time of study by a newly developed left bundle branch block. Two of the three patients with ECG evidence of inferior wall infarction demonstrated, in addition, left ventricular hypertrophy (table 1). Twelve patients were receiving propranolol; ten patients, digoxin; seven, long acting nitrates; and five, a diuretic.

**Radionuclide Technique**

Potassium-43 myocardial imaging was performed following the intravenous injection of one mCi of the radionuclide. ^4K was administered following a 12-hour fast with the patient in an upright position. This procedure has been shown to reduce splanchnic blood flow and resultant hepatic and gastric ^4K accumulation.

Imaging was performed with a rectilinear scanner (Picker Magnascanner 500) with a 5-inch crystal and 31-hold model Picker 2112 collimator. In all studies the collimator was positioned just above the chest wall. A 0.34-0.70 MeV window was employed. Scanning was accomplished at an approximate count density of 800 to 1,000 with standard contrast enhancement of 40% count rate differential. This degree of contrast enhancement has been routinely employed in previous qualitative studies. Imaging at lesser degrees of contrast enhancement provides an increasing gray scale and much less clear definition of the borders of “cold” lesions. Increasing contrast enhancement can also lead to a slight increase in apparent lesion size. Scanning a heart phantom with a known defect at varying degrees of contrast enhancement (from 100% to 40% count rate differential) led to an increase in lesion size of 17%. Scan speed was 100

**Table 1**

*Relationships Between ECG, Angiography, and ^43K Imaging*

<table>
<thead>
<tr>
<th>Pt.</th>
<th>RCA</th>
<th>Significant coronary arterial stenosis</th>
<th>ECG infarct pattern</th>
<th>^43K defect</th>
<th>Ventriculographic site of dyskinesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+</td>
<td>+</td>
<td>ASMI</td>
<td>Antero-septal</td>
<td>Antero-apical akinesis</td>
</tr>
<tr>
<td>2</td>
<td>+</td>
<td></td>
<td>IWM!</td>
<td>Inferior</td>
<td>Inferior hypokinosis</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td></td>
<td>ALMI</td>
<td>Anterior</td>
<td>Antero-apical akinesis</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td></td>
<td>LBBB</td>
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<td>Apical dyskinesis</td>
</tr>
<tr>
<td>5</td>
<td>+</td>
<td></td>
<td>LVH</td>
<td>Apex</td>
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</tr>
<tr>
<td>6</td>
<td>+</td>
<td></td>
<td>SM!</td>
<td>Antero-septal</td>
<td>Apical dyskinesis</td>
</tr>
<tr>
<td>7</td>
<td>+</td>
<td></td>
<td>IAM!</td>
<td>Inferior</td>
<td>Inferior hypokinosis</td>
</tr>
<tr>
<td>8</td>
<td>+</td>
<td></td>
<td>AWMI</td>
<td>Antero-septal</td>
<td>Antero-apical akinesis</td>
</tr>
<tr>
<td>9</td>
<td>+</td>
<td></td>
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<td>Antero-septal</td>
<td>Antero-apical akinesis</td>
</tr>
<tr>
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<td>+</td>
<td></td>
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<td>Antero-apical akinesis</td>
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<tr>
<td>11</td>
<td>+</td>
<td></td>
<td>ASM!</td>
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</tr>
<tr>
<td>12</td>
<td>+</td>
<td></td>
<td>ASM!</td>
<td>Antero-septal</td>
<td>Antero-hypokinosis</td>
</tr>
<tr>
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<td>+</td>
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<tr>
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<td>Inferior akinesis</td>
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<tr>
<td>15</td>
<td>+</td>
<td></td>
<td>IAM!</td>
<td>Inferior</td>
<td>Inferior akinesis</td>
</tr>
</tbody>
</table>

**Abbreviations:** Pt. = patient; + = greater than 70% reduction of luminal diameter; ASMI = antero-septal myocardial infarction; IWM! = inferior wall myocardial infarction; ALMI = anterolateral myocardial infarction; LBBB = left bundle branch block; LVH = left ventricular hypertrophy; RCA = right coronary artery; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery.

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cm/min. Line spacing was 0.5 cm. Scans were obtained in anterior and 40-50° left anterior oblique positions. The average duration of each scan was about ten minutes. Data were simultaneously recorded in two ways: as the familiar radionuclide scan recorded on photographic film, and as a "dot image" on teledeltos paper (fig. 1). The dot image is generated electrostatically by a stylus attached to the moveable carriage on which the crystal detector is mounted. The stylus records a dot on the sensitive teledeltos paper when a preset amount of activity (128 or 256 cpm) is encountered. As the detector moves back and forth across the precordium, the dot image, which can be readily quantified, is described simultaneously with the photographic image. The spatial orientation of the dot image is exactly the same as the photographic scan; and by employing constant fixed spatial references, the two presentations of myocardial radionuclide distribution can be superimposed. A translucent grid of 10 × 12 mm rectangular spaces was overlayed on the dot image and the number of dots and hence radioactivity in each rectangle was counted. This allowed a simple quantification of the visualized regional 43K left ventricular myocardial distribution (fig. 1).

To relate data obtained with this approach to actual regional count rate reductions, phantom studies were performed in which defects were created by placing lead discs of varying heights in an enclosed fluid-filled volume containing 0.1 mCi of 43K. The height of the discs in relation to the height of the 43K bath determines the signal-to-noise ratio. For example, discs half the height of the 43K would be associated with 50% reduction in regional activity. Phantom photographic and dot scans were obtained as in patient studies.

43K data was analyzed by two of the authors, independent of knowledge of angiographic or clinical findings. A qualitative assessment of the scans was made as to whether 43K myocardial activity was normal (homogeneous) or whether "cold" regions of relatively decreased radionuclide concentration existed (abnormal). Because of the potential geometric problem of increased left ventricular cavity size alone producing an abnormal image pattern, zones of decreased activity were not considered abnormal as a result of decreased regional myocardial uptake unless the abnormality extended to an outer border of the myocardial image.

Only images in which cardiac and hepatic activities were separable were considered adequate for evaluation. Outlines of the left ventricular images and the cold spot defects in anterior and LAO views were drawn. Outlines of the left ventricular image were drawn from the margins of sharpest demarcation between myocardial activity and background. When a defect extended to the periphery of the image, the outline was completed by connecting the adjacent portions of the drawn silhouette circumference. In individual cases

Figure 1

Representation of the two types of display of 43K myocardial distribution. The familiar photographic scan is shown on the left in the anterior (ANT) and left anterior oblique (LAO) views. A region of decreased 43K uptake in the anterolateral zone is seen in the ANT view, with zones of decreased uptake involving the inferior wall and high septal region seen in the LAO view. Quantification of the simultaneously obtained dot image is shown on the right. Each small box represents a 10 × 12 mm rectangle on the translucent grid which was superimposed on the original dot scan. The double hatched regions represent normal zones, the single hatched regions represent zones of at least 20% reduction in count rates. Background areas are shown in white.
superimposition of the quantified dot image aided in more precisely defining outer margins. In each instance the margin of a 43K defect was taken as the point of sharpest demarcation between “hot” and “cold” areas on the photographic scan. Both the total area of the myocardial image and the defect in each view were measured by planimetry and the size of the defect was expressed as a percentage of the total image area. This percentage was designated % 43K HP (percent potassium-43 hypoperfusion). The average of this fractional defect size in each view was obtained. The traced outlines of the myocardial image were compared to the grid distribution of the dot image in each patient.

Cardiac Catheterization and Angiography

Hemodynamic and angiographic evaluation included right and retrograde left heart catheterization and selective right and left coronary cineangiography on 35 mm film in multiple projections. Single plane left ventricular cineangiography was performed in a 30 degree right anterior oblique position following injection of 45-54 cc of Renografin-76. In four patients, left ventricular cineangiography was repeated following either epinephrine infusion (two patients) or administration of nitrates (two patients).

Coronary arterial stenosis was considered significant if greater than 70% of the luminal diameter was compromised. Left ventriculograms were evaluated for the presence of dysynchrony by both qualitative assessment and superimposition of traced outlines of the end-diastolic and end-systolic cavitary silhouettes. Akinetic segments (segments sharing a common line in systole and diastole) or dyskinetic segments (those showing paradoxic systolic expansion) were identified from the superimposed outlines and their length measured with a map measurer. This length was expressed as a percentage of the total end-diastolic circumference, excluding the aortic valve plane (fig. 2). This percentage was designated as percent abnormally contracting segments (%ACS). Ventricular volumes were determined by the method of Greene.

Results

All 15 patients demonstrated angiographically significant coronary atherosclerosis with an average of 2.4 major coronary arteries stenosed (range 1–3) (table 1). In each instance the location of the stenosed or obstructed coronary vessels provided a suitable anatomic explanation for the site of infarction. Contrast left ventriculography revealed abnormally contractile akinetic or dyskinetic segments in every patient (table 1). The site of angiographic abnormality corresponded to the electrocardiographic location of infarction with only two exceptions. In patients #4 and #7, both of whom presented ECG pictures of inferior wall infarction with associated left ventricular hypertrophy, significant anterior wall dysfunction consistent with anterior infarction was also present angiographically. In patient #4 inferior wall contraction was normal. In patient #3, with left bundle branch block, the site of contraction abnormality was related to the electrocardiogram obtained following infarction but prior to the development of the subsequent conduction disturbance. The pre-bundle branch block ECG demonstrated evidence of anteroseptal and inferior infarctions in this patient with two distinct clinical infarctions.

Six of the abnormally contracting segments were dyskinetic, the remaining abnormalities represented segmental akinesis. The percent abnormally contracting segments (%ACS) averaged 43% with a range

![Figure 2](https://example.com/figure2.png)

Selected end-diastolic and end-systolic left ventricular cineangiographic frames and superimposed outlines of the cavitary silhouettes. The %ACS was calculated at 42% in this study, demonstrating inferior and apical akinesis.
from 10% to 58%. In four patients further evaluated with intervention left ventriculography, there was no change in either the site or extent of segmental contraction abnormality following the infusion of epinephrine or administration of nitrates. Ejection fraction averaged 44% (range 12–69). This correlated with the %ACS (r = −0.76). Left ventricular end-diastolic volume averaged 144 cc (range 87–399 cc). Cardiac output for the entire group averaged 3.8 L/min with a range from 1.9–6.9 L/min and left ventricular end-diastolic pressure (LVEDP) averaged 19 mm Hg (range 10–35 mm Hg).

On visual inspection, the $^{43}$K myocardial images in all 15 patients demonstrated significant areas of relatively decreased $^{43}$K uptake (figs. 3–5). The anatomic sites of decreased uptake in the two views were: anterior wall (eight patients), anterior and inferior wall (six patients) and inferior wall (one patient). The defect size (% $^{43}$K HP) averaged 49% of the anterior view image (range 25–66%), 43% of the LAO view image (range 0–58%), with a mean defect percentage of 47% in both views (17–62%). The mean total area of the anterior image in the 15 patients was 58 cm$^2$ (range 40–101 cm$^2$) (table 2).

![Figure 3](http://circ.ahajournals.org/lookup/figure/00000000)

Figure 3

$^{43}$K photographic images and processed dot images in anterior (ANT) and left anterior oblique (LAO) views (patient #3). Note anteroseptal and inferior defects. Again, as in figure 1, in the quantified dot image, single hatched zones indicate regions of at least 20% decrease in regional count rates.

<table>
<thead>
<tr>
<th>Pt,</th>
<th>EF</th>
<th>%ACS</th>
<th>ANT</th>
<th>LAO</th>
<th>Mean</th>
<th>Area (cm$^2$)</th>
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<td>47</td>
</tr>
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<td>10%</td>
<td>33%</td>
<td>0%</td>
<td>17%</td>
<td>40</td>
</tr>
</tbody>
</table>

$^{43}$K = ejection fraction; %ACS = percent abnormally contracting segments; ANT = anterior view; LAO = left anterior oblique view; Area = planimetered area of $^{43}$K image in anterior view.

rates were within ±10% of the mean, with most areas being within ±5%. The lowest count rates in apparently normal regions were usually noted at the left ventricular apex. This is consistent with similar studies done in seven normal patients with the same technique, where quantified regional $^{43}$K activity over the myocardial image was within ±10% of mean activity, and only occasionally as low as 15% less than
Anterior (left) and LAO (right $^{43}$K images with superimposed end-diastolic and end-systolic cavitary outlines obtained from the left ventricular cineangiogram. (patient #12) Note the large $^{43}$K defect associated with an extensive local contraction abnormality.

Figure 4

On the other hand, count rates in regions corresponding to zones of decreased visualized activity were at least 20% less than that in normal regions, with a range from 20–40% reduction. Phantom studies demonstrated that an actual 70% reduction in regional count rates resulted in an observed 20% reduction measured from the dot image, while 100% reduction (lead

Figure 5

Left panel) Display of anterior and LAO $^{43}$K photographic scans and end-diastolic and end-systolic left ventricular cineangiographic frames (patient #7). Note the large $^{43}$K defect extending to the periphery of the myocardial image in both views. Right panel) Superimposed end-diastolic and end-systolic angiographic cavitary silhouettes demonstrating the extent of akinesis.
discs the total height of $^{43}$K volume) was associated with 50% reduction as measured from the dot image.

The site of the $^{43}$K imaging defect qualitatively related well with few exceptions to both the ECG location of infarction and the contrast ventriculographic site of abnormal segmental contraction (table 1). Two of nine patients with angiographic inferior wall dysfunction ($\#6$, $\#13$) had apparent normal $^{43}$K inferior wall uptake. One of these patients ($\#6$) had no ECG evidence of inferior infarction. In two patients ($\#4$, $\#7$) anterior wall dysfunction probably secondary to infarction was not associated with classic electrocardiographic evidence of anterior infarction (possibly because of associated LVH) but was associated with abnormal anterior wall $^{43}$K uptake. Therefore, in this series, angiographically demonstrable anterior wall dysfunction secondary to infarction was associated with $^{43}$K imaging abnormalities in all patients, while inferior wall defects were seen in seven of nine (78%) patients.

The extent of the $^{43}$K defect was related to the extent of the segmental contraction abnormality (%ACS). The mean % $^{43}$K HP of both views and that for the anterior and left anterior oblique views alone correlated well with %ACS. The correlation between mean % $^{43}$K HP and %ACS was: % $^{43}$K HP vs %ACS: $r = 0.77$ (fig. 6); anterior view: % $^{43}$K HP vs %ACS: $r = 0.67$; left anterior oblique view: % $^{43}$K HP vs %ACS: $r = 0.54$. The extent of $^{43}$K HP correlated less well with ejection fraction ($r = 0.48$), and not at all with cardiac output ($r = 0.04$) or LVEDP ($r = 0.09$). For these comparisons akinetic and dyskinetic segments were considered together. There was no significant difference in mean % $^{43}$K HP in those patients with akinesis (% $^{43}$K HP = 46%) and those with dyskinesis (% $^{43}$K HP = 47%).

As might be expected, from previous radiologic studies, the total area of the $^{43}$K image in the anterior view (which reflects left ventricular end-diastolic volume) could be related to left ventricular end-diastolic volume ($r = 0.79$) (fig. 7).

**Discussion**

Regional myocardial uptake of an intracellular radioactive cation such as radiopotassium is probably governed by several factors. As demonstrated by comparison with radioactive microspheres, the distribution is clearly blood flow-dependent. The role of cell membrane function affecting accumulation of intracellular radioactive cations is equally relevant. This was recently shown by Levenson et al. who produced cesium-129 imaging defects in experimental animals by direct coronary perfusion of hypoxic blood at normal flow rates. Following myocardial infarction where a portion of viable myocardium has been replaced by acellular scar tissue, regional cell membrane function and size of the regional intracellular cation pool available for exchange with the radioactive tracer may be of greater importance. Hence, the distribution of radiopotassium in the resting state should reflect regional viability and the extent of postinfarction scar formation, and possibly resting ischemia. The purpose of this study was twofold: 1) to examine, in the postinfarction patient, the quantitative relationships between site and extent of the defects obtained with $^{43}$K myocardial imaging, and the site and extent of ventricular dysfunction as assessed by segmental left ventricular contraction disturbance; and 2) to relate these visualized $^{43}$K perfusion defects to a preliminary quantification of regional radioactivity.

Each patient in this series demonstrated significant abnormality in segmental ventricular contraction.
This is comparable to the previous angiographic study of Feild et al. in which akinetic or dyskinetic segments were identified in 24 of 25 patients studied in the year following myocardial infarction. The relative size of the abnormality contracting segment could be quantitatively related to impairment of left ventricular function. Although no patient in this study came to postmortem examination, it is reasonable to assume that the segmental contraction abnormalities following transmural myocardial infarction in the 15 patients in this study were for the most part due to scar formation. In some patients abnormalities may have been due to a combination of scar in association with reversible ischemic change. A failure to improve segmental contraction during intervention, left ventricular cineangiography was noted in all four patients so evaluated. This would support the view that, at least in those patients in whom ventriculography was evaluated in two states, contraction abnormalities were most likely due to scar. In a recent report by Balaxe et al., abnormalities in segmental ventricular contraction were correlated with a histopathologic estimate of regional fibrosis in 21 patients studied at postmortem examination. In 51% of ventricular segments analyzed, the authors noted the regional dysfuction paralleled the presence or absence of fibrosis, while in 42% angiographic dysfuction was more severe than the extent of fibrosis. This was felt to be due to ischemia or "coronary steal." The number of patients who had sustained a previous transmural infarction was not, however, stated.

The correlation between the extent of angiographically determined contraction abnormalities and the extent of radionuclide perfusion defects represents an initial validating step in the development of a radionuclide approach for quantitatively assessing myocardial viability. Our results are in agreement with the data recently presented by Rigo et al. Using the technique of ECG gated blood pool imaging to determine regional ventricular dysfunction and scintillation camera 42K imaging the authors noted a similar correlation between extent of akinesia and the area of reduced 42K uptake (r = 0.74). As in our study, a much poorer correlation was noted between the area of decreased 42K uptake and ejection fraction (r = 0.54). Although these results are encouraging, they must be viewed as preliminary. The next steps in further refining the quantitative aspects of "cold spot" imaging will have to deal with gradations in abnormality and definition of the limits of detection of poorly perfused myocardium, the factors of cardiac and respiratory motion, the unusual geometry of the left ventricle leading to problems in detection of cold areas which overlap normal regions with high radioactivity, edge and cavity effects, intra- and extra-cardiac radiation scatter, irregular thickness of the ventricular muscle, and the need for multiple views in order to develop a three dimensional functional construct of left ventricular myocardium.

Although similar results might be expected in the instance of acute myocardial infarction, where the development of a precise means of assessing lesion size is even more relevant, this remains to be proven. In the acute stages of infarction a postulated heterogeneity of 42K uptake would most likely have a different primary mechanism, based upon flow dependency and regional cell membrane function, rather than replacement of cellular tissue by acellular scar.

A good deal of experience has been accumulated with 42K imaging. It is clear, however, that this radionuclide presents several problems which limit its widespread use. Because of its physical characteristics, a large degree of Compton scatter is encountered. The relative high energy of the tracer limits imaging to the rectilinear scanner unless special collimation is employed with the scintillation camera. If scanner technique is not meticulous, images will be less than optimal. The relatively short physical half-life (22.4 hours), cyclotron or accelerator production, and high cost limit availability. These problems have led to the search for alternative radioactive tracers with more optimal physical properties and similar physiologic behavior. Rubidium-81, cesium-129, and more recently thallium-201 have been proposed. Thallium-201, because of a much lower energy spectrum, would allow routine imaging with the conventional scintillation camera. Nevertheless, a pathophyslogic demonstration with a prototype radioactive tracer such as 42K should allow application of the same principles to newer radionuclides with similar physiologic behavior.

This study demonstrates as well that visualized defects of apparent reduced regional radioactivity are associated with quantifiable decreases in regional count rates. The magnitude of the decrease in regional count rates of between 20 to 40% in abnormal zones does not, however, accurately reflect what is probably a far greater decrease in radionuclide uptake in previously infarcted myocardium. The externally detected defect represents a combination of true decrease in regional 42K uptake and superimposition of the effect of Compton scatter, isoresponse curve of collimators, and ventricular geometry. The effects of scatter were well illustrated in the phantom studies. Smaller magnitude decreases in count rates can be seen in normal hearts usually in the apical region. This most probably represents the effects of irregular muscle-thickness and over-all ventricular geometry. Defects can be artifically created or enlarged by
varying collimator distance from the patient source and hence affecting the relationship between target and optimal focal depth of the imaging system. For this study, collimator distance was held constant and was as close as possible to the chest wall. Although some quantitative statements can be made with regard to defect size, this must be interpreted in the light of rectilinear scanner resolution which is certainly not ideal. Advances in both instrumentation and collimator design, application of higher resolution scintillation cameras, tomography, and computer processing of data will undoubtedly lead to further improvement in resolution and definition.

In this relatively small series of patients with previous transmural myocardial infarction, qualitative diagnostic identification of regions of infarction was quite good. This is comparable to the previously reported studies of patients following infarction by Gorten et al. using 42K,24 and Romhilt et al.21 and Planiol et al.25 using radioactive cesium. Several conditions other than myocardial infarction have been associated with abnormal images simulating the finding of myocardial infarction and provide false-positive results. 42K imaging in patients with left bundle branch block and normal coronary anatomy has been associated with septal wall defects, thought to possibly represent a specific septal membrane defect.25 Selected patients with asymmetric septal hypertrophy (ASH) have been reported to have poorly localized defects.27 Finally, study of patients with congestive cardiomyopathy and enlarged left ventricular cavities and thin left ventricular walls may also yield an image similar to that of myocardial infarction.2 Because of the geometry of the left ventricle a markedly enlarged cavity can produce a large central defect. In this study, however, a defect was only considered significant in terms of infarction if it extended to the outermost margins of the radionuclide silhouette.

Limitations in the assessment of segmental contraction patterns in coronary artery disease from single plane left ventricular angiography should also be noted. Although motion of the anterior, apical, and inferior walls are visualized, contraction of the septal and posterior walls cannot be evaluated. For this purpose biplane angiography would be helpful.

Nevertheless, within the limitations currently imposed by imaging instrumentation and radionuclide availability, it does appear that following transmural myocardial infarction, myocardial imaging with radioactive intracavitary cations such as potassium-43 can accurately and quantitatively reflect the site and extent of myocardial dysfunction and scar as assessed by left ventricular angiography. Such an approach may have value in the diagnostic evaluation of the patient in whom previous infarction has not been documented. Combining this approach with exercise or stress imaging to assess reversible ischemia, and radionuclide functional studies of ejection fraction and regional myocardial performance, may provide an informative quantitative noninvasive evaluation of the patient with documented coronary heart disease.

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