Physiological Basis for Potassium ($^{39}$K) Magnetic Resonance Imaging of the Heart

David S. Fieno, Raymond J. Kim, Wolfgang G. Rehwald, Robert M. Judd

Abstract—The potassium cation (K$^+$) is fundamentally involved in myocyte metabolism. To explore the potential utility of direct MRI of the most abundant natural isotope of potassium, $^{39}$K, we compared $^{39}$K magnetic resonance (MR) image intensity with regional myocardial K$^+$ concentrations after irreversible injury. Rabbits were subjected either to 40 minutes of in situ coronary artery occlusion and 1 hour of reperfusion (n=26) or to 24 hours of permanent occlusion (n=4). The hearts were then isolated and imaged by $^{39}$K MRI (n=10), or tissue samples were analyzed for regional $^{39}$K content by MR spectroscopy (n=9), K$^+$ and Na$^+$ concentrations by atomic emission spectroscopy (inductively coupled plasma atomic emission spectroscopy; n=5), or intracellular K$^+$ content by electron probe x-ray microanalysis (n=6). Three-dimensional $^{39}$K MR images of the isolated hearts were acquired in 44 minutes with $3\times3\times3$–mm resolution. $^{39}$K MR image intensity was reduced in infarcted regions (51.7±4.8% of remote; $P<0.001$). The circumferential extent and location of regions of reduced $^{39}$K image intensity were correlated with those of infarcted regions defined histologically ($r=0.97$ and $r=0.98$, respectively). Compared with remote regions, tissue analysis revealed that infarcted regions had reduced $^{39}$K concentration (by MR spectroscopy, 40.5±9.3% of remote; $P<0.001$), reduced potassium-to-sodium ratio (by inductively coupled plasma atomic emission spectroscopy, 20.7±2.1% of remote; $P<0.01$), and reduced intracellular potassium (by electron probe x-ray microanalysis, K$^+$ peak-to-background ratio 0.95±0.32 versus 2.86±1.10, respectively; $P<0.01$). We acquired the first $^{39}$K MR images of hearts subjected to infarction. In the pathophysiologies examined, potassium ($^{39}$K) MR image intensity primarily reflects regional intracellular K$^+$ concentrations. (Circ Res. 1999;84:913-920.)

Key Words: K$^+$ ■ MRI ■ infarction ■ viability ■ myocyte

Previous studies have demonstrated the fundamental role of the potassium cation (K$^+$) to myocardial physiology. During ischemia, potassium efflux from myocytes has a time course similar to that of the loss of contractile function, and the resulting increase in extracellular K$^+$ concentration may underlie ventricular arrhythmia. Irreversible injury to myocytes is characterized by a profound decrease in tissue K$^+$, and the magnitude of intracellular K$^+$ loss after irreversible injury is closely related to the increase in serum creatine kinase enzyme. Therefore, it has been established that cellular level K$^+$ distributions closely relate to myocardial metabolism.

Because of the fundamental role of K$^+$ in myocardial physiology, radiopharmaceuticals of potassium ($^{39}$K, $^{41}$K, and $^{42}$K) and its analogues ($^{81}$Rb, $^{82}$Rb, $^{86}$Rb, and $^{201}$Tl) have been developed and used to obtain diagnostic images. Indeed, $^{201}$Tl single-photon-emission CT is currently in widespread clinical use. Systematic study of the myocardial kinetics of these radiopharmaceuticals has revealed that image intensities are strongly influenced by both active K$^+$ transport and regional delivery of the tracer by myocardial perfusion. Because active transport and perfusion both measurably contribute to image intensities, a number of injection and imaging protocols have developed depending on whether the clinical question is regional perfusion or viability.

In principle, image intensity in a $^{39}$K magnetic resonance (MR) image would be fundamentally different from that obtained using radiopharmaceuticals. $^{39}$K is a naturally occurring, nonradioactive isotope that constitutes 93% of whole-body potassium and can be directly detected by nuclear MR (NMR). Because no exogenous tracer is injected, $^{39}$K MR image intensity would not be dependent on tracer delivery by perfusion but rather would reflect “static” potassium distributions. In practice, potassium MRI is difficult, because the $^{39}$K MR signal is very small, and, to date, no $^{39}$K MR images of the heart have been reported. For example, compared with the proton ($^1$H) signal routinely used for MRI, the $^{39}$K signal is $\approx2$ million times lower given that in vivo concentrations of $^{39}$K are $\approx1000$ times lower and the NMR sensitivity is $\approx2000$ times lower. On the other hand, recent studies suggest that the application of fast imaging techniques originally developed for $^1$H MRI are well suited to imaging of...
nuclei such as $^{23}$Na and $^{39}$K because of the much shorter NMR relaxation times of these nuclei.22 When the advantages of fast imaging techniques are considered, we previously reported that the quality of in vivo $^{23}$Na MR images is significantly improved.23 More recently, we found that 3-dimensional $^{23}$Na images of the heart can be acquired in a few minutes in rabbits and dogs24 and in humans at 1.5 T.22 On the basis of the $^{23}$Na results and theoretical considerations,25 it is conceivable that some combination of larger voxel sizes, longer imaging times, specialized radio frequency (RF) receiver coils,25,26 and higher magnetic field strengths27 may also make $^{39}$K MRI of the heart feasible.

A definitive statement regarding the achievable quality of $^{39}$K MR images would require extensive study and, even then, the answer would likely evolve as MRI technology advances. The ultimate utility of $^{39}$K MRI, however, will be determined by the relationship of $^{39}$K MR image intensity to the underlying physiology, which will not be influenced by technological developments. To examine this relationship, we explored $^{39}$K MRI of isolated, nonbeating hearts previously subjected to infarction. We then compared image intensities to total (tissue-level) K$^+$ concentrations, intracellular K$^+$ levels, and histological changes. The goal of this study was to test the hypothesis that $^{39}$K MR image intensities reflect electrolyte imbalances secondary to cellular injury.

Materials and Methods

Thirty male New Zealand White rabbits (1.9 to 3.1 kg) were studied. All animals were cared for in accordance with the Position of the American Heart Association on Research Animal Use, adopted November 15, 1984. For each animal, the heart was studied by $^{39}$K MRI and 2,3,5-triphenyltetrazolium chloride (TTC) staining ($n=10$), $^{39}$K MR spectroscopy (MRS; $n=9$), inductively coupled plasma atomic emission spectroscopy (IC-PAES; $n=5$), or electron probe x-ray microanalysis (EPXMA). The following parameters were used for the rabbit hearts: voxel size $=3 \times 3 \times 3$ mm ($0$-filled from 6 mm in phase and slice directions), repetition time (TR) $=40$ ms, echo time (TE) $=3$ ms, $N_{TR}=128$, pulse width (pw) $=200$ µs, matrix size $=64 \times 32 \times 16$, digital sampling interval $=100$ µs, and echo offset $=20$ data points. $^{39}$K MRI times were $\approx 44$ minutes. For the dog heart, imaging parameters were similar, except that voxel size was $5 \times 5 \times 5$ mm and imaging time was 60 minutes.

After $^{39}$K MRI data were collected, the hearts were sliced into 3-mm-thick short-axis sections and histologically stained with TTC to reveal viable and nonviable regions. TTC forms a red precipitate in the presence of intact dehydrogenase enzyme systems and therefore causes viable areas of myocardium to stain brick red, whereas nonviable areas do not stain and appear yellowish-white.32 For each heart, 4 slices of TTC-stained myocardium were photographed and the location of the incision marking the orientation of the heart was recorded. The photographs were scanned into a computer and analyzed using the software package NIH Image.

Circumferential extent and circumferential location of infarction were determined by manually tracing TTC negative areas. In the case of nontransmural infarction, a region was considered “infarcted” if $>50\%$ of wall thickness was TTC negative. Circumferential extent and circumferential location of reduced $^{39}$K MR image intensity were determined as follows. First, the mean and SD of $^{39}$K image intensity were measured in a remote (normal) region. Next, the $^{39}$K images were thresholded at a level defined as 2 SDs below the mean of the remote region. Myocardial regions with $^{39}$K image intensities below this threshold were defined as having reduced $^{39}$K image intensity. The circumferential extent of the infarct was measured as the circumferential angle of the pie slice–shaped region of reduced $^{39}$K image intensity. The circumferential location was defined as the angular location of the center of the pie slice–shaped region where 0 degrees was defined to the right. Also measured were the mean $^{39}$K MR signal intensities in remote and reduced intensity regions and the SD of the background signal. Myocardial signal-to-noise ratios were determined using Henkelman’s33 method for magnitude images.

MRS

Potassium concentrations of individual tissue samples were determined with $^{39}$K MRS to examine the difference in potassium content in remote and infarcted tissue samples using a previously described technique.23 Tissue sections $\approx 0.75$ g in weight were removed and weighed in labeled test tubes. Each sample was then placed in the same RF coil used for imaging next to 1 mL of a 47.7 mM/l KCl standard solution containing 43.5 mM/l dysprosium triethylene-tetraamine-hexacetic acid, which caused the precession frequency of the standard solution to shift such that 2 peaks appeared in the $^{39}$K MR spectrum, 1 from the tissue sample and 1 from the test tube containing the known amount of K$^+$. Spectra were obtained using the following MR parameters: $N_{TR}=4096$ and 8192 (remote and infarcted samples, respectively), pulse width $=140$ µs, digital sampling were not perfused with a hyperkalemic solution, to test whether the results would be similar in the absence of hyperkalemic arrest.
interval=200 μs, block size=1024 samples, and TR=250 ms. The free induction decays were Fourier transformed, phased, and analyzed to determine areas under the sample and standard peaks. From these data, the millimolar potassium concentration in each tissue sample was calculated. The accuracy of this technique was verified by measuring [K⁺] of 6 solutions with known potassium concentrations. The correlation coefficient relating the known to measured [K⁺] was 0.99.

ICPAES

Tissue Na⁺/K⁺ ratios were measured by using ICPAES (Jarrell Ash Autoscan, model 25). Tissue samples weighing ~200 mg were dissolved in 1 mL of 3N HCl at 80°C for 4 hours. The resulting solution was then filtered, diluted volumetrically to 10 mL, and analyzed for potassium and sodium content.

EPXMA and Morphometric Analysis

Intracellular sodium and potassium contents were examined using EPXMA. The techniques used for tissue preparation and analysis were similar to those previously described and validated in detail by other investigators.34–36 In brief, 1- to 2-mm-thick remote and infarcted myocardial tissue samples were obtained within 3 minutes of heart excision and "slam" frozen by clamping the tissue between copper blocks precooled to the liquid nitrogen temperature (77 K). Samples were transported to a cryomicrotome in a polystyrene container surrounded by dry ice and kept frozen during sectioning (-20°C). Frozen 1- to 3-μm-thick sections were transferred in the microtome to precooled aluminum stubs previously covered by double-adhesive carbon tape (to eliminate the aluminum x-ray peak). The frozen sections were then placed in an airtight chamber surrounded by dry ice (-70°C) and freeze-dried by evacuating the chamber to a pressure of <20×10⁻³ mm Hg for ~4 hours.

Freeze-dried samples were then analyzed in a scanning electron microscope (either Hitachi S-4500-II or Hitachi S-570 ) equipped with an energy-dispersive x-ray spectrometer (Voyager, Noran Instruments, Inc). On each tissue section, individual cells were clearly visible at a magnification of ×500, and the x-ray detector was engaged. Individual myocytes lying <100 μm from the freezing surface were selected at random for x-ray analysis. Intracellular x-ray spectra were recorded for 300 seconds from a 5×5-μm area positioned within the myocyte using an accelerating voltage of 10 keV, an emission current of 10 to 100 μA, a dead time of 20% to 30%, and a working distance of ~18 mm. For each tissue sample, 3 to 10 spectra from within myocytes were acquired. As is commonly done for EPXMA,35 for each spectra the magnitudes of the sodium and potassium peaks were expressed as the ratio of the area under the characteristic peak divided by the area under the continuum (peak-to-background [P/B] ratio). The results from multiple spectra in the same area (remote or infarcted) and from the same animal were pooled.

In addition to taking 1- to 3-μm sections for analysis with EPXMA, sections of the frozen remote and infarcted tissue samples were used to measure extracellular volumes. In the same animals studied with EPXMA, adjacent slices from the microtome were adhered to room-temperature glass slides and fixed for ~3 minutes in ethanol. The slides were then stained with H&E, examined under a light microscope using ×400 magnification, and photographed at 5 random locations per slide, where 1 infarct and 1 remote slide were taken for each animal (n=6). The photographs were then scanned into a computer for threshold analysis to measure extracellular area. In Adobe Photoshop, a binary image was generated in which extracellular regions were white (gray level 255) and all other areas were black (gray level 0). The average pixel intensity over the entire microscopic field was then measured, and extracellular volume, expressed as percentage volume, was calculated as extracellular space=(average pixel value/255)×100.

Statistical Analysis

Results are expressed as mean±SEM. Circumferential location and extent of TTC negative regions were compared with those of reduced 39K image intensity by linear regression and by the method of Bland and Altman.37 Image intensities, potassium contents, P/B ratios, and

Figure 1. Sequential TTC-stained short-axis slices and corresponding 39K MR images from a 3-dimensional data set of a rabbit heart subjected to reperfused infarction. Histological sections and images are arranged as follows (left to right on figure): basal side up, short-axis view, base to apex from left to right, and left ventricular lateral wall on the right. Viable areas of myocardium stain brick red, whereas nonviable areas appear yellowish-white. Note the similarity of reduced 39K image intensity and nonviable areas of myocardium.

Figure 2. Results similar to those in Figure 1 in a rabbit heart 24 hours after permanent coronary artery occlusion.
sodium-to-potassium ratios were compared using 2-tailed paired \( t \) tests. Values of \( P<0.05 \) were considered significant.

**Results**

In all animals, visualization of necrosis by irreversible injury was confirmed by lack of TTC staining and characteristic features such as contraction band necrosis in H&E–stained sections.

**MRI and Spectroscopy**

Figure 1 shows 4 short-axis \( ^{39} \)K MR images extracted from the 3-dimensional image data with corresponding histological short-axis slices from a heart subjected to reperfused infarction. A direct spatial concordance seemed to exist between infarcted areas of myocardium, identified histologically, and areas of decreased pixel intensity in the MR images. Figures 2 and 3 show similar results in a rabbit 24 hours after nonreperfused infarction and in a dog after reperfused infarction, respectively. For all animals, circumferential extent and center of infarct using the TTC and the MR image data are shown in Figure 4. The correlation coefficients relating circumferential extent and location of infarction to regions of reduced \( ^{39} \)K image intensity were \( r=0.97 \) and \( r=0.98 \), respectively.

Figure 5 shows \( ^{39} \)K MR image intensities and \( K^+ \) concentrations determined by MRS. Infarcted areas had 51.7±4.8% of the MR image intensity in remote areas (\( P<0.001, n=11 \)). Figure 5 also reveals that the potassium concentration, determined by \( ^{39} \)K MRS, of infarcted myocardium was 40.5±9.3% of the concentration in remote regions (\( P<0.001, n=9 \)).

**ICPAES and EPXMA**

ICPAES revealed that the potassium-to-sodium concentration ratio of infarcted myocardial tissue was 20.7±2.1% of the remote tissue (\( P<0.01, n=5 \)). Potassium and sodium contents within individual myocytes were determined using EPXMA. The top panels of Figure 6 show typical scanning electron micrographs of an unfixed, unstained, freeze-dried cryosec-
tion of control myocardium at both high and low magnifications. The immediately adjacent section showing the same myocytes was stained with H&E and is displayed below the electron micrographs for comparison. Although scanning electron microscopy showed general structural features of myocardium such as individual myocytes, many intracellular features were not readily identified. Although the use of transmission rather than scanning electron microscopy of freeze-dried cryosections may have improved image contrast and resolution, this scanning electron micrograph is shown to demonstrate that image quality was sufficient to identify myocytes and acquire intracellular EPXMA spectra. Figure 7 shows typical intracellular spectra from remote and infarcted myocytes. Compared with the remote region, the spectrum from the infarcted region exhibited a lower potassium peak and a higher sodium peak. Figure 8 summarizes the EPXMA data for all animals. Each point represents the average of 3 to 10 spectra acquired for each animal for a total of 72 spectra analyzed. From Figure 8, the K⁺ P/B ratio of infarcted myocytes was 0.95±0.32 compared with the value inside remote myocytes, which was 2.86±1.10 (P<0.001, n=6). EPXMA further revealed that the potassium-to-sodium ratio, in which both quantities were expressed as P/B, of infarcted myocytes was 0.40±0.17 compared with the value for remote myocytes, which was 3.07±0.97 (P<0.002, n=6), also shown in Figure 8. These data demonstrate that the regions of reduced 39K MR image intensity were associated with a loss of intracellular K⁺.

Extracellular Volume
The extracellular volumes in remote and infarcted tissues determined histologically were 14.3±5.3% and 17.2±7.6%, respectively (P<0.02, n=6).

Discussion
To our knowledge, this is the first study in which 39K MRI of the heart has been described. We found that 39K MR image...
intensities were significantly reduced in regions subjected to infarction compared with remote regions. The results of tissue analysis by MRS, ICPAES, and EPXMA provide insight into the mechanisms that govern $^{39}$K MR image intensities.

**Mechanisms Governing Image Intensity**

MR image intensities in our experiments were primarily dependent on 3 parameters: spin density (concentration), $T_1$, and $T_2$. Direct information regarding $^{39}$K spin densities can be found in the MRS data of Figure 5. The magnitudes of the differences in tissue-level $^{39}$K concentrations in remote and infarcted regions, $24.3 \pm 2.8$ versus $9.8 \pm 4.5$ mmol/L, respectively, were similar to the differences in $^{39}$K image intensities (Figure 5), which suggests that image intensities are primarily determined by tissue-level K$^+$ concentrations. Although in principle regional differences in longitudinal relaxation time constant ($T_1$) and transverse relaxation time constant ($T_2$) in remote versus infarcted regions could affect image intensities, our experiments differences in $T_1$ would not be expected to influence the results because the repetition time for our imaging pulse sequence (TR = 40 ms) was ~4 times longer than the $^{39}$K $T_1$ ($\approx 10$ ms). Regional differences in $T_2$ (or $T_2^*$), however, may have influenced our results, and in principle this issue could have been addressed by directly measuring $T_2$ values in tissue samples. In practice, we were unable to perform these experiments because the $^{39}$K spin densities in infarcted regions were so low that MRS signal-to-noise ratios were not sufficient to accurately measure multiple echo amplitudes with increasing echo times. Even without the $T_2$ data, however, our results (Figure 5) strongly suggest that differences in $^{39}$K image intensities were primarily determined by regional differences in spin density.

Insight into the relationship of tissue-level spin densities to cellular-level distributions of K$^+$ can be obtained by consideration of the ICPAES and EPXMA data. By ICPAES, we found that the ratio of K$^+$/Na$^+$ was much lower in infarced regions demonstrating an imbalance of tissue electrolytes. Because K$^+$ concentrations are normally far higher in the intracellular compared with extracellular space, one would expect that tissue K$^+$ would be greatly affected by changes in intracellular K$^+$. The EPXMA data (Figures 7 and 8) confirmed that the loss of tissue-level K$^+$ demonstrated by MRS and ICPAES was associated with large losses of intracellular K$^+$. In view of these data, we conclude that $^{39}$K MR image intensities are reduced in regions subjected to irreversible injury primarily because of a loss of intracellular K$^+$.

**Effects of Myocardial Injury on K$^+$ Content**

After irreversible injury, we found that tissue K$^+$ content in infarcted myocardium constituted $40.5 \pm 9.3\%$ of the remote region by MRS (Figure 5). This value is similar to the finding of Jennings et al, who reported K$^+$ concentrations of 41.5 versus 21.0 mmol per 100 g fat-free dry weight for remote versus infarcted myocardium (infarcted myocardium was 51% of remote) in a canine model with 40 minutes of occlusion followed by 50 minutes of reperfusion. In another study by this same group, Whalen et al demonstrated that K$^+$ concentration of infarcted canine myocardium decreased from 40.6 to 31.3 mmol per 100 g fat free dry weight (infarcted myocardium was 77% of remote), corresponding to 155 versus 661.6 mmol/L tissue water (infarcted myocardium was 58% of remote) after 40 minutes of occlusion and 10 minutes of reperfusion. Jennings et al also reported that, 1580 minutes ($\approx$ 1 day) after permanent occlusion, the K$^+$ concentration of infarcted tissue decreased from 38.3 to

![Figure 7. Representative intracellular x-ray spectra from 1 rabbit in remote (left panel) and infarcted (right panel) myocytes. Potassium P/B ratio is clearly lower in the spectrum acquired from a myocyte in the infarcted region compared with remote. Sodium P/B ratio is higher in the spectrum acquired from a myocyte in the infarcted region. Notice the appearance of a calcium peak in the infarcted myocyte spectrum at 3.7 keV.](http://circres.ahajournals.org/)
5.4 mmol per 100 g fat-free dry weight (infarcted was 14% of remote). In our study, \(^{39}\text{K}\) image intensity in infarcted regions was \(61.7 \pm 3.5\%\) of that of remote regions 1 hour after the infarct had been reperfused (Figure 5, ○) and 33.9 \(\pm 7.6\%\) of that of remote regions 1 day after permanent occlusion (Figure 5, ○; \(P < 0.001\) compared with ○). The similarity between the data of Jennings et al\(^{1}\) concerning K\(^{+}\) concentrations and our data concerning \(^{39}\text{K}\) MRI is consistent with our conclusion that \(^{39}\text{K}\) MR image intensity primarily reflects regional K\(^{+}\) concentrations.

In previous studies of \(^{23}\text{Na}\) MRI by our group,\(^{23,24}\) we found in the same rabbit model (40 minutes of occlusion, 60 minutes of reperfusion) that \(^{23}\text{Na}\) image intensity in areas of infarction were \(142\%\) of that of remote regions and that the Na\(^{+}\) concentration of these infarcted tissues were \(163\%\) of that of remote regions. Similarly, others have found that irreversible injury is characterized by an increase in Na\(^{+}\) and a decrease in K\(^{+}\), and the time course of these changes has been studied.\(^{6,7}\) These results suggest that, for infarcted myocardium, the information reflected by \(^{23}\text{Na}\) MRI may be comparable with that reflected by \(^{39}\text{K}\) MRI.

During ischemia, however, the information reflected by \(^{23}\text{Na}\) with \(^{39}\text{K}\) MR image intensities may be different. Hill and Gettes\(^{1}\) report that K\(^{+}\) concentrations change within the first 15 to 30 seconds of ischemia, whereas Pike et al\(^{39}\) report that changes in Na\(^{+}\) occur over the time scale of minutes to tens of minutes. Although K\(^{+}\) and Na\(^{+}\) are cotransported by the Na\(^{+}\)-K\(^{+}\) ATPase, each ion also has unique transport channels and, in certain situations, Na\(^{+}\) and K\(^{+}\) MRI may reflect different pathophysiological information.

**Relationship of Image Intensity to Absolute [K\(^{+}\)]**

Although image intensity appears to primarily reflect regional intracellular K\(^{+}\) concentrations, direct comparisons of \(^{39}\text{K}\) image intensity with absolute tissue concentration may be more complex. One can compare our results with estimates of tissue-level K\(^{+}\) concentrations. Assuming the intracellular space is \(75\%\) of the water space, that the water space is \(80\%\) of the total, that intracellular K\(^{+}\) concentration in normal myocardium is 140 mmol/L, and that extracellular K\(^{+}\) concentration is 4 mmol/L, tissue K\(^{+}\) would be 
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\approx 85 \text{ mmol/L} = 0.8 \times (0.75 \times 140 + 0.25 \times 4)\]

Experimental, however, we found that tissue K\(^{+}\) concentrations in normal myocardium were only 24.3 \(\pm 1.4\) mmol/L by MRS (Figure 5). Isolated hearts are often subject to mild injury, which may decrease intracellular K\(^{+}\) concentrations below in vivo values.\(^{40}\) Additionally, however, this apparent discrepancy might be partially explained by the results of other investigators, who have suggested that a portion of the intracellular K\(^{+}\) pool in the heart may be “invisible” to NMR.\(^{41}\) This “invisibility” may be due to different magnetic environments within myocardial tissue.

To investigate whether the tissue environment may have influenced our MRS results, we performed the following experiment. First, we measured \(^{39}\text{K}\) spin density by MRS as previously described and found, as reported in Figure 5, that K\(^{+}\) concentration in normal myocardium was 24 mmol/L. Next, the tissue sample was removed from the test tube, placed in a clean metal bowl, and freeze-thawed several times with liquid nitrogen. The frozen sample was also crushed with a mortar while bathed in the liquid nitrogen. Then, the sample was transferred back to the test tube and dissolved by adding 1 mL of either 4 mol/L NaOH or 3N HCl. Seven days later, we repeated the identical MRS experiment on each tissue sample. Interestingly, we found that measured K\(^{+}\) concentrations increased from 24 to 37 mmol/L (\(n=4, P < 0.001\)). This result provides strong evidence that the magnetic environment experienced by K\(^{+}\) ions is in some way affected by the structure of the tissue itself, resulting in a net reduction in the detected \(^{39}\text{K}\) MR signal. Although this effect appears to measurably affect the magnitude of the myocardial \(^{39}\text{K}\) signal, regions containing lower K\(^{+}\) concentrations will still be reflected as regions of reduced image intensity compared with normal areas.

**Study Limitations**

The \(^{39}\text{K}\) MR images of this study were acquired at high field (4.7 T) and were of isolated, nonbeating hearts. To date, the ability to acquire similar images in living humans has not been demonstrated. The data from this study, however, strongly suggest that useful physiological information is reflected by regional \(^{39}\text{K}\) MR image intensities.

**Summary**

In irreversibly injured regions, \(^{39}\text{K}\) MR image intensities were reduced, the spatial extent and locations of regions of reduced image intensity correlated with infarct size histologically, and regions of reduced \(^{39}\text{K}\) image intensity were characterized by electrolyte imbalances associated with a loss of intracellular K\(^{+}\). We conclude that, in the pathophysiology investigated, \(^{39}\text{K}\) MR image intensity primarily reflects regional intracellular K\(^{+}\) concentrations.

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**References**

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