Intracellular sodium MRI during acute regional myocardial ischemia and reperfusion

M. A. Jansen1, M. G. Nederhoff1,2, C. J. Van Echteld1

1CardioNMR-Laboratory, University Medical Center, Utrecht, Netherlands, 2Interuniversity Cardiology Institute of the Netherlands, Utrecht, Netherlands

Introduction
Due to the rapid changes of intracellular [Na+] ([Na+]i) during ischemia and reperfusion of viable myocardium, 23Na-MRI appears to be an ideal diagnostic modality for early detection of myocardial ischemia and viability. So far, data on cardiac 23Na-MRI are extremely limited and are mostly concerned with imaging of total Na+. For proper interpretation, imaging of both intra- and extracellular Na+ is essential. Previously, using 23Na-MR chemical shift imaging (CSI) we found a very good correlation between Na+ image intensity at the end of global low flow ischemia and recovery of rate pressure product at the end of reperfusion of isolated rat hearts. This shows that Na+ image intensity can predict the ability of myocardial tissue to recover after ischemia. In this study, the value of intracellular sodium imaging was assessed in a model of acute regional ischemia and reperfusion.

Methods
Data were acquired using a Bruker AVANCE 400 spectrometer. Rat hearts were perfused using a dual-perfusion cannula2, which allowed independent perfusion of both sides of the heart. To assess the area at risk, one side of the heart was perfused with a Gd-DTPA-BMA-containing perfusate and a T1-weighted 1H-image was acquired after 15 min. Next, the contrast agent was omitted and the shift reagent TmDOTP5 was included in the perfusate on both sides to separate the intra- and extracellular sodium resonance. Subsequently, acquisition-weighted 23Na-CSI (16×16, FOV 20×20 mm, slice thickness 5 mm, voxel size 7.8 µl, 5 min/scan) was performed during control perfusion, ischemia of only the left side of the heart for 40 min (flow to the other side remained unaltered) and reperfusion. At the end of the experiment, the right side of the heart was perfused with methylene blue to determine the area at risk for histology. After that, the whole heart was perfused with 1% triphenyltetrazolium chloride (TTC) to stain the viable tissue.

Results and Discussion
Figure A shows short axis 23Na images of a heart subjected to acute regional ischemia and reperfusion and the corresponding 1H- and TTC image. Na+ was already visible during control perfusion. Na+-image intensity increased significantly during ischemia of the left side to 345 ± 75 % while that of the right side remained unaltered. During reperfusion, Na+-image intensity returned to normal in 2 of the 5 hearts. Total 23Na image intensity remained unaltered during the entire protocol in both sides of the heart. The area on the Na+-image at the end of ischemia where Na+-intensity was above 4% (% of the buffer) correlated well with the unstained area on the TTC-image (R=0.73). However, further research is necessary to confirm this finding.

Figure B. Intracellular Na+ image intensities relative to the buffer intensity (%) of the area at risk (closed circles) and the right coronary perfusion bed (open circles) (n=5 hearts).

Conclusion
These data demonstrate that intracellular 23Na-CS-imaging is a promising tool for assessment of myocardial viability.

References