Assessment of myocardial viability by $^{31}$P-MR-spectroscopy and $^{23}$Na-MR imaging

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An exact differentiation between viable (hibernating or stunned) and non-viable (scar) tissue is crucial for the decision whether revascularisation is required after myocardial infarction [1]. Former studies demonstrated altered energy metabolism in ischemic myocardium [2]. $^{31}$P-MR-Spectroscopy offers the unique possibility for non-invasive study of cardiac energy metabolism. Aim of the present work was to analyze whether a reliable detection of myocardial viability is possible by $^{31}$P-MR-Spectroscopy. All examinations were performed on a 1.5 Tesla clinical MR system (Magnetom VISION, Siemens, Erlangen). $^{31}$P-spectra were acquired using a double-resonant $^{31}$P/$^1$H-surface coil and a double-angled 3D-CSI-technique (voxel size 25 cm). Due to the limited sensitivity for the deeper parts of the heart only patients with anterior wall infarction were included ($n = 20$). For each patient $^{31}$P-spectra from the infarcted area were compared to $^{31}$P-spectra from non-infarcted septal myocardium (internal reference). Additionally, left ventricular function was analyzed by short axis cine-MRI breath-hold sequences (slice thickness 8 mm). Both examinations were performed at study entry (3 weeks after acute myocardial infarction) and 3 months after revascularization. Improvement of regional function in MRI was used as gold standard for viability [3]. Aged-matched healthy volunteers ($n = 10$) served as control group. Using the AMARES software [4] the phosphocreatine to adenosine triphosphate (PCr/ATP)-ratio was $1.72 \pm 0.31$ in the septal myocardium and $1.76 \pm 0.38$ in the anterior myocardium for the control group, standing in close agreement with former publications [5]. In the non-infarcted septal myocardium a slight, however, non-significant reduction of the PCr:ATP-ratio was detected for all patients. Patients who showed recovery of regional function were classified as having viable myocardium. Here, no difference was detected between $^{31}$P-spectra of infarcted and non-infarcted tissue. Missing recovery of regional function was classified as non-viable myocardium. Here, no PCr peaks were detected in $^{31}$P-spectra. Therefore, PCr:ATP-ratios could not be calculated. As an indirect measurement of ATP content, the signal to noise ratio (SNR) for ATP was determined for these patients, demonstrating a significant reduction for these patients (Fig. 1). After acute myocardial infarction viable myocardium shows no significant alterations of the PCr/ATP ratio compared to healthy controls. A reduction of the ATP-SNR may correlate with non-viable myocardium. $^{31}$P-Spectroscopy allows a non-invasive insight into energy metabolism in (post-) ischemic myocardium in humans.

A different approach for the non-invasive measurement of the precise extent of myocardial infarction (MI) and for the prediction of myocardial viability is the assessment of electrolyte alterations, i.e. of sodium hemostasis. Therefore, imaging of myocardial sodium content by sodium MRI might offer a solution [6] to this. In acute subacute MI, loss of cell membrane in-
Integrity and ionic hemostasis lead to an increase of intracellular sodium and, accompanied by extracellular edema formation, also of total sodium content of the infarcted tissue. In chronic MI, the extracellular space with up to 10-fold higher $^{23}\text{Na}$ concentration increases vs the intracellular space during scar formation as

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**Fig. 1.** $^{31}\text{P}$-MR-Spectroscopy; 3 weeks after anterior myocardial infarction; voxel size 25 ccm each; depletion of PCr in non-infarcted septal myocardium (right); depletion of PCr and ATP in the infarcted anterior myocardium (left).

**Fig. 2.** $^{23}\text{Na}$-MR imaging of the entire heart in the short axis view.