Quantitative ^{23}Na MRI in Chronic Human Myocardial Infarction

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Introduction: Sodium (^{23}Na) Magnetic Resonance Imaging (MRI) with a surface coil at 1.5 T shows elevated tissue sodium signal in myocardial infarction (MI) (1,2). The signal changes may reflect loss of cellular integrity, inhibition of the Na^+/K^+ pump function due to energy depletion, changes in the ratio of intra and extra cellular volumes (3), changes in the T_1 or T_2 of tissue sodium, or a combination of these factors. Although there is currently no reliable and safe way to entirely separate the signals of extra- and intra-cellular sodium in humans, the potential role played by relaxation changes can be eliminated. Thus, we used a fully-relaxed, ultra-short TE twisted projection imaging (TPI) sequence combined with surface-coil adiabatic excitation, to measure tissue sodium concentrations (TSC) in human chronic MI eliminating the confounding effects of T_1 and T_2 relaxation.

Methods: Six patients (ages 49-73, five males) with chronic MI ($\geq$ 95 days post MI; infarct territories 9±8 year old) and ten controls (male; 24-49 yrs) were studied. Informed consent was obtained from all subjects. A TPI sequence with a 0.4 ms tanh/tan amplitude/frequency-modulated adiabatic pulse was used to obtain fully-relaxed 3D ^{23}Na images (TE/TR 0.4/100ms) with 2ml isotropic voxel size. Images were recorded in 12 min (1240-projections, 6 averages) at 1.5T with a 25 cm surface coil tuned at 16.9 MHz. Concentrations were calculated from the ratio of the signals in the regions of interest from each subject and the signals from the same regions in a registered image of a 150 mM [Na] concentration reference phantom, recorded separately. The signal from three tubes with 100 mM NaCl in agarose gel embedded in the coil was used to correct for coil loading differences and as feducial markers for image registration. ^{23}Na MR images were registered with gated ^{1}H images recorded in the same exam with FSE (TR=1*R-R, TE 17 ms ETL=4), or burst gated FGRE (TR/TE = 9.1/4.2, 90º flip) sequences using the ^{1}H body coil. Delayed Contrast Enhancement (DCE) imaging was performed on the patients separately with a ^{1}H phased array coil to identify MI regions.

Results: Sample ^{1}H and ^{23}Na MR images are shown in figure1, revealing an increase in the sodium signal to almost the LV blood level in the inferior, septal LV region. In controls (see table 1), TSC was consistent with prior invasive and non-invasive measurements in canine hearts (4). In patients, TSC was significantly elevated by about 40% to 64±9 µmol/g in infarcted territories versus LV regions identified as uninvolved (paired t-test) and versus LV of controls. No significant difference was found for TSC in blood, uninvolved LV, or adipose tissues between patients and controls (p>0.05).

Table1	Patients (N=6)		Controls (N=10)	
tissue	mean	SD	mean	SD
LV wall[umol.g ww]	46*	11	45†	8
adipose[umol.g ww]	19	5	19	5
lesion[umol.g ww]	64†*	9	-	-
LV blood [mmol/l]	76	5	-	-
RVblood [mmol/l]	76	7	76	15

† t-Test: control LV vs. lesions Two-sample Unequal Variances p<0.001. * paired t-Test non-involved vs. lesions p< 0.01

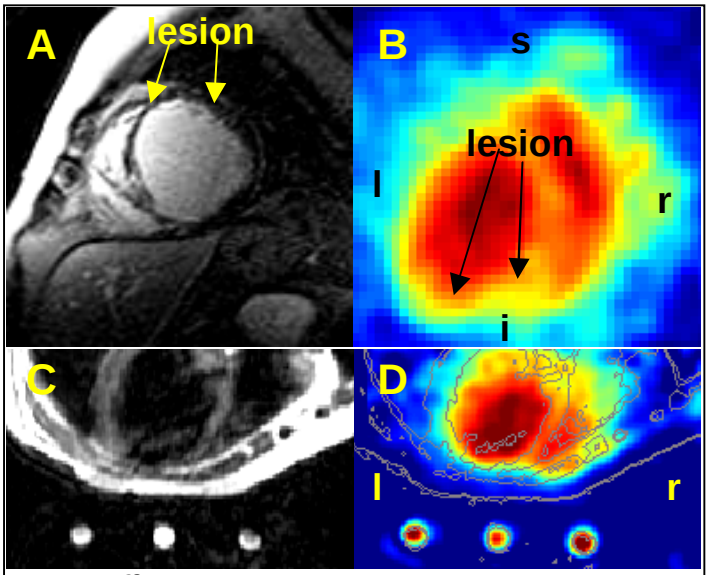

Figure 1. ^{23}Na images (jet color-map) and 1H images of a patient with a thirteen year old, anteroseptal, infarct. Short axis DCE image (A), a coronal view of the ^{23}Na images (B), FSE image (C) and matching axial ^{23}Na MR image (D), with ^{1}H level contours.

Conclusions: Absolute myocardial TSC is elevated in patients with chronic MI. Blood and adipose TSC in patients are consistent with prior values in controls. The results suggest that prior observations of elevated ^{23}Na MRI signal intensity in chronic MI are in fact due to TSC increases and not just relaxation changes**.**

References:

1. Sandstede JJ, et al. Magn Reson Med 2004; 52: p545-551.
2. Ouwerkerk R. 7th Ann. SCMR. Barcelona, Spain, 2004; pp435.
3. Jansen MA, Nederhoff MGJ, van Echteld CJA. 7th Ann. SCMR. Barcelona, Spain, 2004; pp439.
4. Constantinides CD.et al. Magn Reson Med 2001;46:1144-1151.

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