Rapid Quantification of Systolic and Diastolic Transverse T1ρ Relaxation Times in the Human Left Ventricle

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Introduction: Post infarction, left ventricular remodeling is responsible for almost a quarter of heart failure instances (1-2) and it is evident that infarct expansion and dilatation is an important factor for initiating and sustaining this process (3). Understanding the pathophysiology that occurs after an infarction is necessary for limiting infarct expansion and ventricular remodeling. Clinically, the most common methods for diagnosis of infarct expansion and remodeling are surface ECG and serum chemistry. Delayed contrast enhancement MRI has also shown to be a useful tool for assessing left ventricular remodeling. The purpose of this work was to develop a rapid pulse sequence for cardiac T1ρ (spin lattice relaxation in the rotating frame) imaging by k-space-weighted image contrast (KWIC) reconstruction and to measure ^{1}H relaxation times *in vivo*. We hypothesize that there are measurable transverse relaxation time changes during the cardiac cycle, which may be used as a surrogate measure of cardiac stress.

Materials and Methods: ***In Vivo MRI****:* Three volunteers (22-30 years of age) gave informed consent to participate in this study. Each volunteer was scanned 4 times at 1.5T (Tim Espree Model, Siemens Medical Solutions, Malvern, PA, USA) equipped with 18 mT/m nominal gradients. A posterior, spine matrix coil, together with an anterior body matrix coil, was used. A gradient echo localizer was used to obtain short axis cardiac views. ***Pulse Sequence Design:*** T1ρ-prepared, diametrical, k-space undersampled gradient echo acquisition was programmed in IDEA (Siemens). Two free-breathing, T1ρ maps were acquired; one each during systole and diastole by ECG triggering at 300 ms (systole) and 650 ms (diastole) from the R-wave. The parameters used for acquisition were as follows: TR = 2000 ms, 2.41 ms, bandwidth/pixel = 403 Hz, slice thickness = 5 mm, matrix = 128 x 128, FOV = 240 mm^2 or 280 mm^2, flip angle = 15^o, 2-4 shots, TSL = 6-48 ms in 6 ms increments, radial views = 40-60, scan time = 5.5 minutes. The spin lock pulse cluster consisted of five rectangular pulses, which made use of an integrated spin lock and spin echo approach to compensate for B1 and B0 homogeneity. ***KWIC Reconstruction****:* Raw data was reconstructed using kspace-weighted image contrast (4), accelerating acquisition by a factor of 8. A detailed explanation is shown in Figure 1. ***Data Analysis:*** T1ρ relaxation maps were generated by pixelwise, linear, least squares fitting to the linearized exponential T1ρ decay. Prior to fitting, each T1ρ-weighted image was smoothed using a Gaussian filter. Following mapping, each image was noise filtered on the basis of goodness of fit ($\chi^2 \geq 1$), maximum and minimum signal intensity, and the appropriateness of T1ρ relaxation times for the cardiac tissues ($0 \leq T1\rho \leq 250$ ms). In vivo images were overlaid on T1ρ-weighted images (TSL = 48) and the left ventricle was segmented with the ROI placed in the medial portion of the interventricular septum. Statistical analysis was performed by studentized, two-tailed t-test.

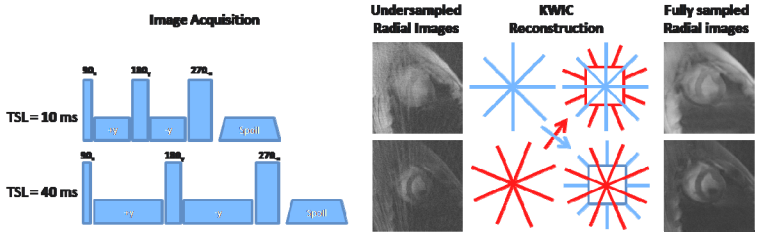

Figure 1: Method for KWIC reconstruction of T1ρ data. Undersampled radial images were acquired during which the spin lock duration (TSL) was varied in each (Columns 1 and 2). During KWIC reconstruction, high spatial frequency components with a minimum kspace radius k_{min} were combined to reconstruct fully, angularly sampled, radial data (Column 3), from which full resolution images were reconstructed (Column 4). Note that the experiment combines not two, but N sets of T1ρ data, accelerating acquisition by a factor N.

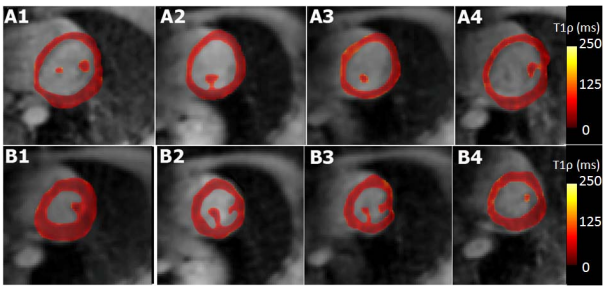

Figure 2: T1ρ maps of four short-axis cardiac slices imaged during diastole (A1-A4) and in systole (B1-B4). All images were acquired from a single healthy subject scanned four separate times.

Results: *In vivo* relaxation times measured in the interventricular septum were significantly different between systole (T1ρ = 63.5 ± 7.4 ms) and diastole (T1ρ = 72.0 ± 7.4 ms) ($p < 0.05$) (Table 1). No spatial variations in relaxation times were observed as a function of wall radial dimension or between the papillary muscles and the ventricle, although it is likely that such differences may exist. 4 sets of data from one volunteer during systole and diastole are displayed in Figure 2. Data was of moderate reproducibility, and coefficients of variation were between 8 and 13%. No significant differences were observed between right and left ventricular relaxation times. It is also possible to measure the relaxation times of blood in both ventricles, although it must be assumed that some saturation effects occur because of the shorter delay between triggered R-R intervals and blood T1. The use of undersampled, radial acquisition followed by KWIC reconstruct enabled scan times to be reduced 8-fold, from 28 to less than 5 minutes.

SUBJECT	Septum Mean Systole	Mean Systole Std Dev	Septum Mean Diastole	Mean Diastole Std Dev	Coefficient Variation Systole	Coefficient Variation Diastole
1	69.3	8.1	79.7	6.8	0.117	0.086
2	60.0	5.7	70.0	6.4	0.096	0.091
3	61.3	8.3	66.1	8.8	0.136	0.134

Table 1: Measurement of ^{1}H T1ρ in 3 subjects, 4 times each. All of the relaxation times represented are in milliseconds. Each measurement was performed from an ROI in the interventricular septum. The mean T1ρ for diastolic and systolic relaxation times and coefficient of variation is displayed.

Discussion: We suspect that the observed increase in ^{1}H transverse relaxation times between systole and diastole is related to the change in the rotational correlation time of the nucleus during the cardiac cycle. Normal contraction and relaxation exert a time-dependent stress/strain on the extracellular matrix and myocytes, which modulates the rate of rotation of water molecules. It is likely that spin locking suppresses competing mechanisms of relaxation such as susceptibility, chemical exchange and diffusion, such that these differences become observable. T1 is probably insensitive to changes in the rotational correlation time because the water ^{1}H T1 in heart muscle is typically longer than the cardiac cycle, giving way to a time-averaged relaxation time throughout. Partial volume effects and motion can shift the heart location, such that between a set of TSL times, the heart is no longer stationary. We are working to develop techniques that use self-navigation or acquire single slice data in a single breathhold, so that these artifacts are reduced. T1ρ MRI may be used for the diagnosis of acute or chronic MI to distinguish or follow the area of infarct after ischemia. Early increases in T1ρ may be because of edematous changes to the myocardium, which again modulate the rotational correlation time of the water protons. Later deposition of collagenous scar, during chronic ischemia can also increase T1ρ. This latter study is the focus of Abstract 1493. The method of KWIC reconstruction is quite general and may be used for the acquisition of T2 or diffusion-weighted data in the myocardium following a similar fashion.

Conclusion: T1ρ imaging and relaxation mapping was performed in the human myocardium using a T1ρ-prepared, multiecho, radial acquisition, from which KWIC reconstruction was performed to accelerate image acquisition by a factor of 8-fold. These techniques may be used to study infarct expansion and dilatation in patients undergoing left ventricular remodeling. A significant advantage of T1ρ imaging is for the use of patients with kidney disease, for which a delayed contrast enhanced MR scan is not possible.

References: (1) Kaul P, et al. *Circulation* (2004). (2) Gaudron, et al. *Circulation* (1993). (3) Jackson, et al. *JACR* (2002). (4) Song HK, et al. *Magn. Reson. Med.* (2000). **Acknowledgements:** This work was supported by Award P41RR002305 from the National Center for Research Resources. We also thank Niels Oesingmann of Siemens Medical Solutions for his assistance.