Feasibility of CINE Myocardial T_2^* Mapping Using Susceptibility Weighted Gradient-Echo Imaging at 7.0 T

F. Hezel[1], T. Frauenrath[1], W. Renz[2], J. Schulz-Menger[1,3], and T. Niendorf[1,4]

[1]Berlin Ultrahigh Field Facility, Max-Delbrueck Center for Molecular Medicine, Berlin, Germany, [2]Siemens Healthcare, Erlangen, Germany, [3]Helios Klinkum, Berlin, Germany, [4]Experimental and Clinical Research Center (ECRC), Charité Campus Buch, Humboldt-University, Berlin, Germany

Introduction

Emerging cardiovascular magnetic resonance (CVMR) imaging applications include T_2* relaxation sensitized techniques which are increasingly used in basic research and (pre)-clinical imaging (1-4). Myocardial T_2^* mapping is of proven value for the assessment of myocardial iron content, myocardial blood level oxygenation and myocardial perfusion reserve. Because of the super-linear relationship between magnetic field strength and microscopic B_0 inhomogeneities, access to susceptibility weighted myocardial imaging at 7.0 T (i) would extend the capabilities of quantification of myocardial iron content, (ii) would make it easier to differentiate healthy tissue from myocardial regions underlying perfusion deficits due to the multi-fold increase in BOLD sensitivity together with the enhanced differences in T_2*. For this reason it is conceptually appealing to pursue myocardial T_2* mapping at ultrahigh magnetic field strength (B_0>7.0 T). This pilot study demonstrates the feasibility of ultrahigh field susceptibility weighted myocardial imaging and examines its applicability to CINE myocardial T_2* mapping at 7.0 T.

Methods

Volunteer experiments were performed on a 7.0 T whole body MR system (Magnetom, Siemens, Erlangen, Germany) together with a dedicated 4-element TX/RX cardiac coil array (5) using retrospectively triggered 2D CINE FLASH (slice thickness=8 mm, acquisition matrix 128x80, FOV=(360x360) mm^2). An MR stethoscope, which is immune from interference with electromagnetic fields and to magneto-hydrodynamic effects was used for cardiac gating (6). A set of CINE images, each encompassing 25 cardiac phases to cover the entire cardiac cycle was acquired. For each set, susceptibility weighting was incremented in steps of 1.02 ms to receive in-phase images with TE ranging from 3.06 ms to 17.34 ms. TR was kept constant (TR=54.8 ms ± 3.56 ms) by changing the views per segment per R-R interval. Images were processed with MATLAB (Mathworks, Natick, MA, USA) routines including a rigid landmark-based co-registration, a segmentation of the left ventricle. Mono-exponential fitting using nonlinear least squares optimization implemented by Trust-Region algorithm was applied for pixel-by-pixel T_2* quantification.

Results:

The flip angle of the excitation pulse was adjusted to preserve myocardial signal by reducing T_1-saturation effects, which resulted in a low contrast between the blood pool and the surrounding myocardium as illustrated in Figure 1. No severe susceptibility artifacts were detected in the inferoseptal myocardium and in the anterior lateral wall for TE up to 10.20 ms. For posterior myocardial areas close to the main cardiac vein susceptibility related signal void was observed even for minimum TE=3.06 ms. T2* maps showed significant non-uniformity in T_2* across the myocardium. For example, the inferoseptal segment revealed a T_2^* value of (6.8±0.4) ms. In comparison, T_2*=(14.64±4.7) ms was obtained for the anterior region with a maximum of 24.2 ms at diastole. Posterior myocardial areas close to the main cardiac vein showed T_2*=(1.52±0.4) ms. It should be also noted, that T_2* varied across the cardiac cycle as illustrated in Figure 2 which shows an diastolic myocardial T_2* colour map superimposed to the corresponding 2D CINE FLASH image together with a whole R-R interval time series of one-dimensional projections of T_2* along the profile (dotted line) marked in the short axis view T_2* map. For example, T_2* was found to be approximately 15 ms for systolic cardiac phases and around 25 ms for diastolic cardiac phases for a region of interest located in the anterior myocardium.

Discussion and Conclusions:

This is the first report on CINE myocardial T_2^* Mapping at 7.0 T. The proposed approach offers the potential for the quantification of myocardial iron content, assessment of endothelial function, detection of stress induced angina pectoris, tracking of superparamagnetic iron-oxide labeled cells or devices and blood level oxygenation dependent mapping of myocardial perfusion deficits. In the latter case, the ability to monitore changes in tissue oxygenation using T_2* sensitized imaging/mapping offers the potential to address some of the spatial and temporal resolution constraints of conventional first pass perfusion imaging and holds the promise to obviate the need for exogenous contrast agents. In conclusion, we anticipate the extension of this work to a broader clinical study at 7.0 T to exploit the susceptibility sensitivity advantage at ultrahigh fields with the ultimate goal of moving towards 3D T_2* mapping of the heart.

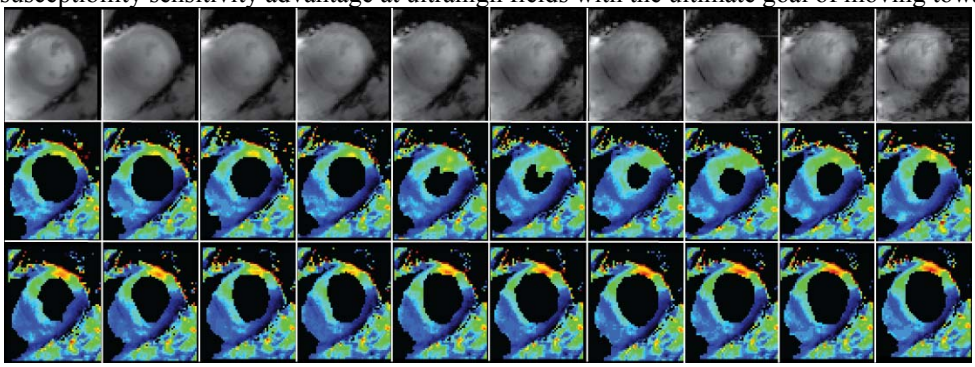

Figure 1: Top row: Short axis views of an end-diastolic phase of a mid-cavity slice of the heart. T_2* weighting was increased from left to right in increments of 1.02 ms with TE ranging from 3.06 to 12.24 ms. **Middle and bottom row:** CINE myocardial T_2^* maps including the first 20 cardiac phases of a whole R-R coverage CINE data set consisting of 25 cardiac phases.

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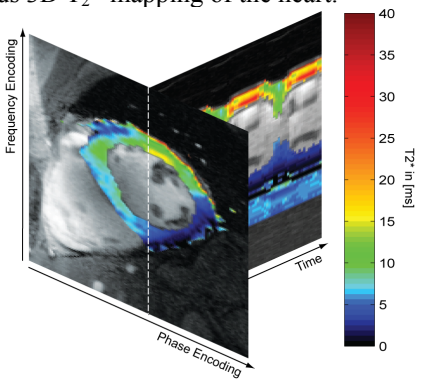

Figure 2: Myocardial T_2* colour map superimposed to the corresponding 2D CINE FLASH image together with whole R-R interval time series of 1D projections of T_2* along the profile (dotted line) marked in the short axis view.