In-vivo right-ventricular myocardial T1 mapping at 3.0 Tesla

Nadja M Meßner[1], Lothar R Schad[1], and Frank G Zöllner[1]
[1]*Computer Assisted Clinical Medicine, Medical Faculty Mannheim, Heidelberg University, Mannheim, Germany*

Target audience: MR scientists, cardiologists and cardiac radiologists.

Purpose: Myocardial T1 mapping can quantitatively measure changes in myocardial collagen content (diffuse fibrotic changes) associated with various cardiovascular diseases. Certain diseases, such as pulmonary artery hypertension (PAH) [1] and arrhythmogenic right-ventricular dysplasia (ARVD) [2] predominantly affect the myocardium of the right ventricle (RV). The revised task force criteria for ARVD newly include RV MRI, so an inclusion of RV T1 mapping could be a next step. So far, T1 quantification in the RV has been mostly neglected. Until now, only two other studies reported T1 mapping in the RV, one with a 3(3)5 MOLLI strategy [3] and one with the ANGIE strategy [4], both at 1.5 T. However, as the free wall of the RV is commonly only 2 to 3 mm thick, a spatial resolution < 2 mm in-plane and a slice thickness ≤ 6 mm is necessary. In addition, adjacent blood T1 values are very different. This, heavy trabeculation and a high variability in structure and shape of the RV turn T1 mapping into a difficult task. Our goal was to investigate the feasibility of robust T1 mapping in the RV at 3.0 T with a higher spatial resolution and smaller slice thickness to reduce partial volume effects.

Methods: 6 healthy subjects (26±3 y/o, 3 males, 3 females) underwent CMRI in a 48-channel 3.0 T MR system (MAGNETOM Skyra; Siemens, Erlangen, Germany) with an 18-element body matrix receive coil and an inbuilt 32-element spine matrix coil.
A modified Look-Locker inversion (MOLLI) [5] sequence was employed. Images were acquired in the short-axis (SAX) view at mid-ventricle. A MOLLI scheme of 5(3)3 (5 acquisition heart beats (HB), 3 recovery HB, 3 acquisition HB), which acquires 8 images in 11 HB has proven to counteract heart rate sensitivity by increasing the spacing between the two inversions [6] and was therefore chosen instead of a 3(3)5 strategy. Breath-hold and a motion correction algorithm were used to suppress breathing motion artifacts. The cardiac phase was prospectively ECG triggered and, in contrast to the common procedure, images were acquired at systole, were the RV is most expanded. This corresponded to a mean trigger time of 384±55 ms. Other acquisition parameters were: b-SSFP readout, TE/TR=1.22/344.86 ms, flip angle 35°, partial Fourier=7/8, parallel imaging=2, start inversion time (TI)=132 ms, TI increment=80 ms, spatial resolution=1.17x1.17x5.00 mm³, matrix= 256 x 168, pixel bandwidth=1085 Hz. The mapping was feasible in all healthy volunteers, repeated 8 times in each subject and in 5 different slices.
T1 maps were reconstructed with the Siemens Quantitative Cardiac Parameter Mapping WIP package. Data and statistical analysis were performed with MATLAB (R2014a, The MathWorks Inc., Natick, USA) by a blinded observer who manually placed ROIs in the RV and LV myocardium in all T1 maps. In each subject, 2 repeated measurements in the same slice and 2 measurements in 2 different slices were compared. Precision was measured as the coefficient of variation (CV), which is defined by the standard deviation across the ROI divided by the mean value of the ROI. Reproducibility was measured as the reproducibility coefficient (RPC, 1.96*standard deviation) of the average T1 within a ROI in repeated measurements. For the paired t-test, the 5% limit was chosen for statistical significance.

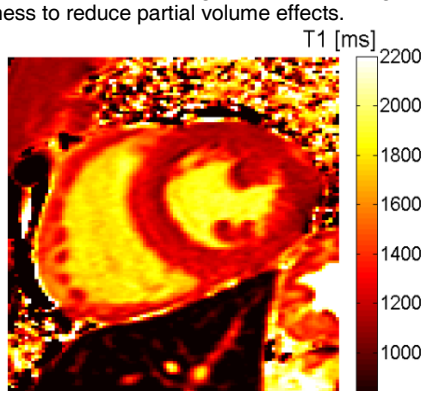

Fig. 1: T1 map acquired with MOLLI with a spatial resolution of 1.17x1.17x5.00 mm³. The position of the SAX slice and the moment in the cardiac cycle (systole) were chosen such that RV is most expanded and only the border zone is affected by partial volume effects.

Results: T1 maps showed homogeneously distributed T1 values inside the RV and the LV myocardium (Fig. 1), as well as amongst different slices of the heart. The average RV myocardial T1 time was 1,354±20 ms, average LV myocardial T1 time was 1,223±37 ms. A paired t-test proved the significance of this difference (p<0.0001). Acquisition during systole significantly increased the size of the ROIs that could be placed inside the RV wall, so the average size of a RV ROI was 113±24 mm². The CV of the average RV and LV myocardial T1 values of all subjects were 5.5% and 6.4%, respectively. A Bland-Altman plot of repeated measurements in the same slice and subject showed good agreement of repetitions in all 6 subjects, with a mean bias of -3.8, a RPC of 1.4% and a CV of 0.74% (Fig. 2). A RV T1 time analysis between repeated measurements in the same subject, but different imaging planes resulted in a mean bias of 4, a RPC of 2.3% and a CV of 1.2%.

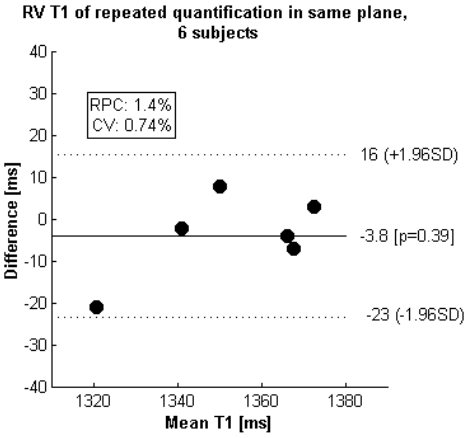

Fig. 2: Bland-Altman plot of the difference in RV T1 values between repeated measurements in the same imaging plane for the 6 subjects shows good agreement. RPC: Reproducibility coefficient (1.96*SD); CV: Coefficient of variation

Discussion: The tendency towards significantly higher T1 times of the RV compared to the LV agrees with the findings in [3,4], which can be explained by a higher collagen content of the RV. The higher field strength of 3.0 T yields a higher SNR. With a better isotropic in-plane resolution of 1.17x1.17 mm² and a slice thickness of 5.00 mm³, the known partial volume effects of MOLLI, where adjacent blood is included in the measured voxels, could be reduced. The higher precision in the RV values compared to LV values can be explained by the fact that measurements were optimized for RV T1 quantification in terms of cardiac phase and slice positioning. Reproducibility is high in both repeated measurements in the same subject and between different subjects.

Conclusion: RV myocardial T1 mapping in healthy volunteers with increased spatial resolution is feasible at 3.0 T. Acquiring images at systole shows improved visualization of RV mass and might reduce partial volume effects. Reproducibility analysis shows that the T1 values found and the measurement technique are robust. As T1 mapping of the LV is employed more commonly in clinical routine and sequence packages are commercially available, the inclusion of RV T1 mapping would not add logistic costs and would be feasible within clinically acceptable scan time. Future work will be directed towards the application in patients with RV hypertrophy to demonstrate clinical outcome.

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