

Single-breathhold Myocardial T2 and T2* Quantification in Normal Volunteer Subjects at 3T

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INTRODUCTION

With SNR increase and a number of other technical improvements, 3T MRI scanners have become increasingly available for clinical investigations. Despite their advantages, increased B0 and B1 inhomogeneity effects at 3T present challenges for cardiac imaging and quantitation [1]. Adversely affected by physiological motion and flow artifacts, free-breathing multi-echo spin echo (MESE) sequences cannot provide reliable myocardial T2 measurement at 3T for monitoring iron overload in patients with thalassaemia major and other iron overload disorders. Earlier work has demonstrated the utility of a breathhold MESE sequence for myocardial T2 mapping with significantly reduced respiratory motion and blood flow artifacts at 1.5T [2]. The current study aimed to quantify the myocardium T2 in normal subjects at 3T using a single-breathhold black-blood hybrid TSE/MESE T2 measurement protocol.

METHOD

3T Protocol and Analysis: Combining partial Fourier and SENSE acquisition, a hybrid TSE/MESE sequence [3] was implemented on a 3T Philips Achieva scanner with 6-channel cardiac coil. In brief, 2 k-space lines are acquired per TR (i.e., turbo factor = 2) so that the single-slice multi-echo T2 mapping can be obtained within a single breathhold (~15 cardiac cycles). The TE of the 1st echo was 5 ms. The effective echo spacing was 10 ms with FOV=370-400 mm, TR=1 cardiac cycle (750-1200ms), acquisition matrix=128x96, SENSE factor=2, partial Fourier factor=0.6, slice thickness = 10 mm for 90° excitation (and 30 mm thickness for 180° excitations to minimize stimulated echo effects), crushers along all 3 directions, and ECG trigger delay set to late diastole (~400ms). A double-inversion black blood technique was used for flow artifact reduction and better LV wall delineation [2,4]. The slice was positioned to cover the short-axis LV view at the mid-ventricular level. The total echo numbers ranged from 10 to 12 (with 5-6 echo images accordingly), which were determined by the heart rate and 3T SAR limit. The single-breathhold acquisition was repeated three times during each scan for T2 averaging. For T2* measurement with a multi-echo gradient echo (MEGE) sequence, echo number was 25, turbo field echo factor was 4, and one breathhold with 9 cardiac cycles was used. The first TE and echo spacing were set to 3 ms and 2 ms, respectively. All other parameters were the same as in the T2 measurement. For analysis, ROIs were drawn in the mid-ventricular septum. T2 and T2* values were calculated by fitting the ROI signals to a mono-exponential model. For heart T2 measurements, an identical ROI was used in analyzing the 3 single-breathhold acquisitions but with slight position adjustments to account for the shifts between the 3 breathholds, and the mean T2 value calculated.

Subjects: Eight healthy volunteers (23-31 yrs with mean age 25.8 ± 2.9 yrs) were scanned for T2. To minimize inter-subject variation, all were trained in the breathhold procedure before MR acquisitions. To estimate T2 measurement reproducibility, T2 values were determined in two subjects on three different days. To examine the effect of B1 inhomogeneity on myocardial T2 quantitation at 3T, one subject was scanned with 6 different flip angle sets by using 6 different RF calibration scaling factors (0.7, 0.8, 0.9, 1.0, 1.1 and 1.2 assuming 1.0 by the direct system calibration using the total MRI signal within imaging slice). Myocardial T2* was measured in 2 subjects. In addition, we implemented and examined a free-breathing black blood ECG-triggered MESE sequence with 11 echoes (and 1st TE and echo spacing of 5 ms and 5 ms, respectively).

RESULTS AND DISCUSSIONS

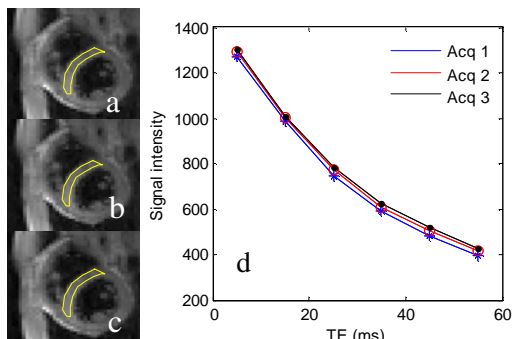


Fig. 1 (a), (b), and (c) are the typical 1st echo MESE images from 3 single-breathhold acquisitions during one exam, with ROI signal decays shown in (d).

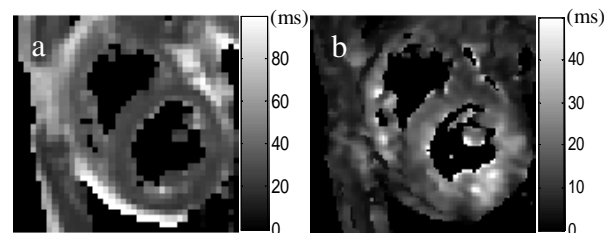


Fig. 2 (a) T2 and (b) T2* maps at 3T.

T2 and T2* Measurements: Fig. 1a-c illustrates the representative MESE images from three consecutive acquisitions (i.e., three different breathholds) in one subject. Fig. 1d shows the corresponding signal decay curves in the septal ROI. Note that ROIs are the same but for slight position adjustments to account for the shifts between the 3 breathholds. In contrast, the traditional free-breathing MESE sequence could not provide decay curves and often exhibited odd/even echo zigzag patterns that were largely caused by increased stimulated echoes in presence of B1 inhomogeneity (data not shown). Fig. 2 shows typical T2 and T2* maps from one subject. Note that the T2 map was more homogeneous than the corresponding T2* map. The average myocardial T2 was found to be 39.6±7.4ms among the 8 normal subjects studied, which was ~30% less than that reported for 1.5T (56.9±8.4ms [2]). The average myocardial T2* was 32.7±3.6ms in the 2 normal subjects studied, which was similar to that (33.3ms) reported previously [5].

Reproducibility Estimate: The peak-to-peak variations of the measured T2 values were found to be 3.5% and 2.2%, respectively, in the two subjects who underwent MRI during 3 different days, demonstrating an excellent reproducibility of the T2 measurement protocol employed.

Effect of B1 Reduction on T2: Fig. 3 plots the T2 measurements in the same patient when varying the B1 calibration scale factor (i.e., changing all excitation flip angles proportionally). For scale factor range of 0.9-1.2, the peak-to-peak variation of T2 measurements was within 5%. This preliminary result indicated that the B1 reduction in myocardium at 3T as previously reported [1,6,7] may not affect the T2 quantitation greatly in normal subjects.

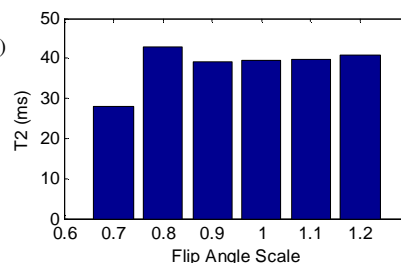


Fig. 3 T2 values with different flip angle scales.

CONCLUSION

We demonstrated the feasibility of myocardial T2 quantitation at 3T. Septal myocardium T2 was observed to be 39.6±7.4ms in normal subjects. Furthermore, this T2 measurement protocol yielded reproducible and robust measurements despite the increased motion artifacts and B1 inhomogeneity at 3T.

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References: [1] Cunnigham CH et al. MRM 2006;55:1326. [2] He T et al. JMRI 2006;24:580. [3] Kim D et al. 2009 ISMRM Proceedings (submitted). [4] Greenman RL et al. JMRI 2003;17:648. [5] Storey P et al. JMRI 2007;25:540. [6] Saekho S et al. MRM 2005;53 :479. [7] Sung K et al. JMRI;2008;27:643.