Magnetic Field Dependence of Myocardial R2* Values and Segmental Myocardial Field Inhomogeneity Assessed at 1.5 T, 3.0 T and 7.0 T.

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Introduction. Parametric T2* is of proven clinical value for noninvasive quantification of cardiac iron overload [1,2]. A segmental approach can detect heterogeneous iron distribution, but could be affected by the presence of geometric and susceptibility artifacts, that may affect myocardial segments in a different way [3] and can be pronounced at high and ultrahigh magnetic fields. Moreover, R2* (1000/T2*) in normal myocardium is expected to increase with static magnetic field, due to the faster relaxation of tissue. For all these reasons this study examines the relationship between myocardial R2* values and magnetic field strength with the ultimate goal to develop a segmental field inhomogeneity map of the human heart at 1.5T, 3T and 7T.

Materials and methods. For 1.5T and 3T data , healthy subjects involved in previous studies were considered [4,5]. Ten healthy subjects (4 males, 27.8±2.6 years) underwent MRI exams at 7T (Magnetom, Siemens, Erlangen, Germany) using a dedicated 16-element TX/RX cardiac coil array. Three parallel short-axes of the left ventricle (LV) were obtained using a T2* weighted GRE multi-echo technique. For each single short-axis view (thickness 4 mm; pixel size 1.6X1.6 mm) a set of 9 echo times (1.56–9.72 ms, ΔTE=1.02 ms) was acquired in a single end-expiratory breath-hold. An MR-stethoscope (MRI.TOOLS, Berlin, Germany) was used for cardiac gating. Macroscopic B_0 inhomogeneities were reduced by applying volume selective shimming. Image analysis was performed using a validated software (HIPPO MIOT®) [2,4]. The LV was divided into 16 segments according to a standardized model [6]. he For each segment T2* was calculated as well as the global and the mid-septal T2* values. The map was developed in the R2* domain [3,4]. The artifactual factor for each segment was defined as the sum of the averaged deviations of the R2* in the segment with respect to the global value plus a constant factor representing the global drift in R2* measurement.

Results. Figure 1 shows the he dependence of global heart (left) and mid-septum (right) R2* values on the static magnetic field strength. The mean R2* values increased approximately linearly with the field strength. For the global R2*, the regression line had a slope of 6.1 and an intercept of 17.6. The R-squared value was 0.992. For the mid-septum R2*, the regression line had a slope of 5.1 and an intercept of 20.0. The R-squared value was 0.995.

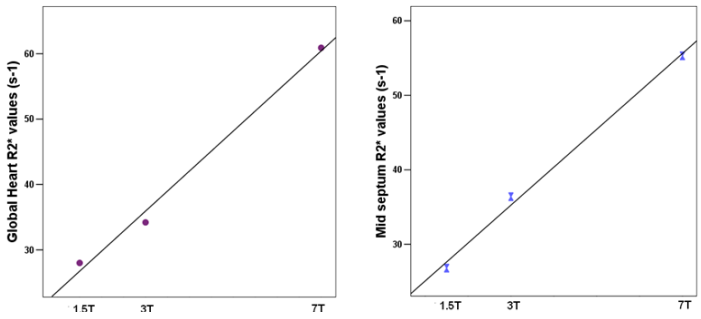

Figure 1

Figure 2 shows the segmental artifactual factors derived from T2* mapping at 1.5T, 3T and 7T. A closer examination showed that the global artifactual factor was significantly enlarged at 7T versus 1.5T (P=0.010). At 7T the most severe susceptibility artifacts were detected in the inferior lateral wall (segments 5, 11 and 16). There was no significant difference between the global artifactual factor observed at 3T and 7T. The analysis also revealed that the artifactual factor observed at 7.0 T was almost 0 for the anteroseptal and inferoseptal segments. This observation is in alignment with the results obtained at 1.5T and 3T.

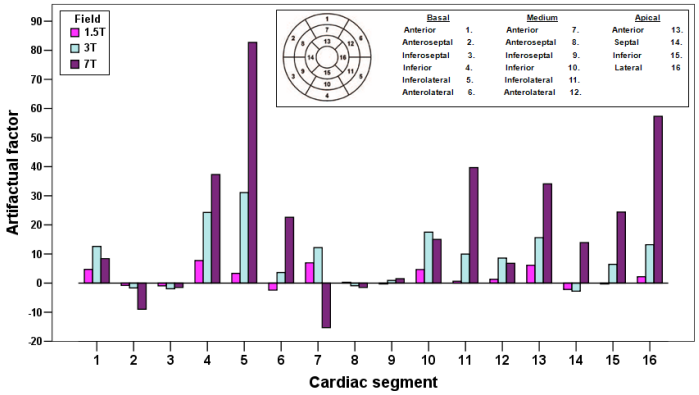

Figure 2

Conclusion. Although R2* relaxation is a complicated process, with possible contributions non-linear with the field strength, our results suggest a nearly linear field dependence of cardiac R2*. Similar results have been reported for the brain R2* [7]. This suggests that the dramatic susceptibility contrast available with 7T MRI could be exploited to quantitatively study iron accumulations in different organs with high sensitivity and resolution. Admittedly, at 7T the pronounced propensity to macroscopic susceptibility artefacts is challenging and it seemed to spare only the septal regions. The most severe artifact source was the heart-lung interface. A recognized limitation of this study is that different groups of healthy subjects were considered for different magnetic field strengths. However, the mean R2* values of healthy subjects are not expected to change in between volunteer cohorts assuming that the T2* mapping technique is applied.

References: [1] Wood JC et al. Blood 2005;106:1460-1465. [2] Pepe A et al. JMRI 2006;23:662-668. [3] Reeder SB et al. Magn Res Med 1998; 39: 988–998. [4] Positano V et al. NMR Biomed 2007;20:578-590. [5] Meloni A et al. MRM 2011, in press. DOI 10.1002/mrm.23236. [6] Cerquiera MD et al. Circulation 2002;105:539-542. [7] Yao B et al. NeuroImage 2009;44:1259–1266.