# Cyclic variation of T1 in the myocardium at 3T

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## Introduction

Myocardial blood volume (MBV) contributes to about 6% to 15% of left ventricular (LV) mass<sup>1</sup>. Knowledge of MBV could help understand the microvascular potency as well as intra-myocardial compressive forces. While tissue T1 is virtually independent of blood oxygenation<sup>2</sup>, T1 of myocardium may increase as MBV increases. It is also well known that arterial inflow occurs at end diastole (ED) and that overall vascular space decreases at end systole (ES). These changes can be readily depicted by cine T1 maps of the LV.

### Method

The ideal steady state signal Mss, of the RF spoiled gradient echo sequence (FLASH, SPGR) is a function of T1 and flip angle a, given by

 $Mss/sin(\alpha) = e1 Mss/tan(\alpha) + M0(1-e1)$ Where e1 = exp(-TR/T1) is the slope, from which the T1 can be derived by measuring Mss at two values of  $\alpha$  at constant TR. However, this relationship is no longer valid given a non-perfect slice profile, a long T1 and a very short TR. To estimate the error in T1, the Bloch equation was simulated for a segmented FLASH sequence. Following parameters were used; excitation pulse shape = Gaussian, slice thickness = 5mm, TR/TE = 6.4/3.7ms, number of segments = 9,  $\alpha = 3^{\circ}$ , 15°, T1 = [400:1700]ms, T2\* = [15:200]ms. T1 measurements were also performed on Gd doped phantoms, using the same imaging parameters with a simulated ECG wave and also with conventional inversion recovery method. Seven healthy volunteers were scanned in the short axis plane at 3-4 slice locations. For each slice, two cine sequences with  $\alpha=3^{\circ}$  and 15° were performed sequentially, in a single breath hold. Matrix size=256x108, number of frames = 17-20, scan time = 25xR-R, other parameters were same as previously described. A phased array cardiac coil was used. All imaging were performed on a 3.0T, (Trio) Siemens scanner.



Figure 2. Normalized T1 maps of the LV in short axis taken at different time points of the heart cycle.



Figure 1. Regression of calculated vs actual  $\Delta$ T1/T1. Flip angle errors of 0%,  $\pm$ 10%,  $\pm$ 20%,  $\pm$ 30% were simulated for both  $\alpha$ 1 and  $\alpha$ 2 and are depicted by various symbols. No significant difference in calculated and actual  $\Delta$ T1/T1 was found for T1 between 400 ms to 1300 ms (arrows).

#### Results

Bloch simulation shows a significant error in T1 calculated using Eq[1] for the given experimental conditions. However, T1 was shown to be linearly related to the measured T1 (T1<sup> $^{-}$ </sup>) with the shape depending on the T2\*, a fact that was confirmed by the phantom studies. For a range of short T2\* values (15-80ms), typical for the myocardium tissue, this relationship was highly correlated; *T1* = 3.49T1<sup> $^{-}$ </sup> - 460 ( $r^2 = 0.95$ ), thus was used to correct the T1 measured in human studies. However, a significant inter-subject variation in T1 was observed, probably due to error in flip angle. Remarkably, Bloch simulation also showed that quotient  $\Delta$ T1/T1 was relatively unaffected by the variations in the flip angle as shown in Fig[1]. Therefore, by normalizing the measured T1 to that of a consistent tissue type one is able to accurately compare the variation in T1 between subjects. The T1 maps presented here are normalized to the T1 of liver of the subject. Fig[2], shows several normalized T1 maps of a subject during the heart cycle. The general T1 decrease in ES and the heterogeneity of T1 distribution was well depicted in all subjects. The average T1 drop from ED to ES in the septum was 43±8% (p<0.01), while in the lateral wall it was 28±4% (p<0.003).

# Conclusion

Although it appears MBV to be the major contributory to the changes in T1, cyclic changes of morphology need to be addressed. Errors in flip angles caused by dielectric mismatch are common for high fields but can be well compensated as described here. This method is non-invasive and does not involve administration of contrast agents, therefore can be easily accommodated in a routine clinical protocol.

#### References

1. Marcus ML, *The coronary circulation in health and disease*, New York, NY:McGraw-Hill 1983:17 2. Atalay, MK et al, *Magn Reson Med*. 1995, 34(4):623-7