

Oxygen-enhanced T2* cardiac magnetic resonance imaging in non-ischemic cardiac diseases

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Synopsis: In this study, we analyzed T2* value in the mid-left ventricular septum amid both normoxia and hyperoxia among clinical cases with non-ischemic heart disease. The oxygen-enhanced T2* cardiac magnetic resonance (CMR) was promising to evaluate the myocardial blood-oxygen dependent (BOLD) response to hyperoxia. The development of quantitative evaluation technique for the oxygen metabolism in human myocardium in vivo has opened up new avenues for the study of the cardiac pathophysiology. To our knowledge, this is the first clinical study that has assessed the myocardial $\Delta T2^*$ response to hyperoxic respiratory challenge by BOLD-CMR. [92/100 words]

Target audience: Researchers, radiologist, and physicians who are interested in cardiac magnetic resonance imaging, especially with regard to myocardial oxygen metabolism in cases with cardiac disease.

Purpose: The aim of this study was to estimate the diagnostic potential of oxygen-enhanced T2* CMR as an alternative method of BOLD technique in non-ischemic cardiac disease.

Methods: Subjects: Consecutive 50 patients (30 men and 20 women; mean age of 55), retrospectively diagnosed as congestive heart failure (CHF) (n=18), Dilated cardiomyopathy (DCM) (n=12), arrhythmia (n=7), hypertrophic cardiomyopathy (n=3), and other disease (n=8) MRI: All patients underwent 3 Tesla MR imaging (Achieva 3.0 T Quasar Dual; Philips Healthcare, Best, The Netherlands) equipped with dual-source parallel radiofrequency transmission, 32-element cardiac phased-array coils used for radiofrequency reception and a 4-lead vectorcardiogram used for cardiac gating. Short-axis black-blood T2* CMR imaging was obtained within a single breath-hold using multi-echo gradient-echo sequence (TE=2.9 to 10.9msec, 6 point). A double inversion recovery pulse was applied on the R-wave during diastole. T2* measurement was performed in the mid-left ventricular septum to minimize the susceptibility artifact from lungs. An exponential function was fitted to the data, as follows; $S_n = S_0 e^{-TE_n/T2^*}$. Subsequently, LGE images were obtained with an inversion-recovery T1 turbo field-echo sequence (fast gradient-echo pulse sequence) performed 10 minutes after contrast injection and acquired in the same orientation as the short axis cine images. Each Myocardial T2*air, T2*oxy, and $\Delta T2^*$ ($=T2^*_{oxy} - T2^*_{air}$) was also calculated and compared to New York Heart Association (NYHA) functional classification, LV ejection fraction (LVEF), brain natriuretic peptide (BNP), and late-gadolinium enhancement (LGE) in the mid-LV septum.

Results: Myocardial T2*oxy was prolonged, comparing to T2*air in patients with NYHA II to IV (2.4msec), LVEF<35% (2.0msec), BNP>40 (2.7msec), and positive LGE (4.0msec) [Paired t-test]. No difference between T2*oxy and T2*air was observed in patients with NYHA I, LVEF>35%, BNP<40 and negative LGE. Myocardial $\Delta T2^*$ was significantly greater for patients with positive LGE (1.9msec) or NYHA II to IV (1.2msec) than those for patients without LGE or NYHA I [Wilcoxon rank-sum test] (Figure 2).

Discussion: Recent developments in T2* cardiac magnetic resonance (CMR) imaging techniques are enabling clinically-feasible rapid parametric mapping of magnetic relaxation properties that are further expanding the range of unique tissue parameters. Generally, T2*-weighted BOLD-MRI visualizes the perivascular signal change due to bulk susceptibility effect by the ratio of diamagnetic oxy-hemoglobin (oxy-Hb) and paramagnetic deoxy-hemoglobin (deoxy-Hb) in small vessels of the heart. The development of non-invasive evaluation technique for the oxygen metabolism in human myocardium has opened up new avenues for the study of the cardiac pathophysiology. Relative increase of diamagnetic property of Oxy-Hb in the peripheral myocardial vessels reflects lower oxygen consumption due to dysfunction of mitochondria or oxygen-binding iron, lacking the normal reaction for hypo-perfusion caused by hyperoxic respiratory challenge in myocardium.

Conclusion: Oxygen-enhanced T2* CMR provides a unique information about the myocardial signal change due to hyperoxia in various non-ischemic cardiac disease.

References: 1. Winklhofer S et al. NMR Biomed (2014), 2. Salerno M et al. JACC (2013), 3. Meloni, A et al. MRM(2013).

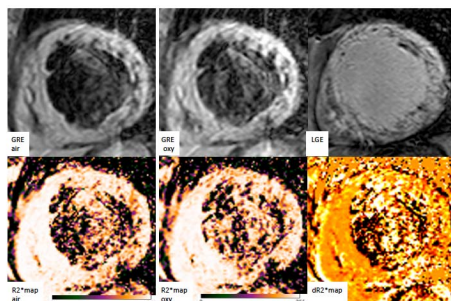


Figure1. A 67-year-old woman with DCM (NYHA III, LGE positive).

Upper column: GRE-air (TE=2.9msec), GRE-oxy, LGE,

Lower column: R2*map-air, R2*map-oxy, $\Delta R2^*_{map}(= \Delta 1/T2^*)$

Note the signal decrease in mid-LV septum at $\Delta R2^*_{map}$

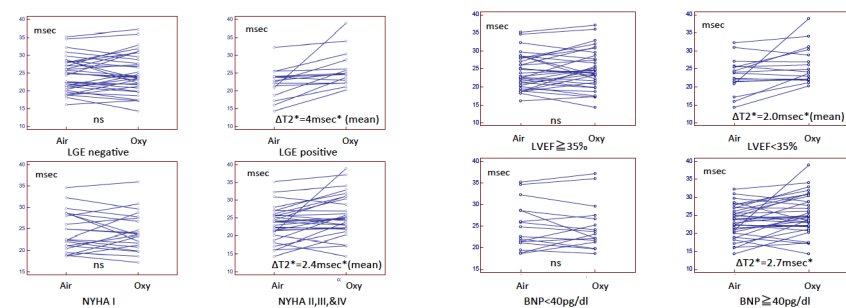


Figure2. Change of T2*air and T2*oxy

Upper column: LGE negative, LGE positive, LVEF ≥ 35%, LVEF < 35%.

Lower column: NYHA I, NYHA II to IV, BNP ≤ 40pg/dl, BNP > 40pg/dl.

Note the T2* increase in LGE positive, NYHA II to IV, LVEF<35%, and BNP ≥ 40pg/dl.