Myocardial BOLD imaging at 3T using quantitative T2: Application in a myocardial infarct model

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Introduction: Coronary vasodilator dysfunction has been demonstrated in infarcted as well as remote myocardium in patients with acute coronary syndrome (1). Recently, blood-oxygen-level-dependent (BOLD) approaches have been employed to probe myocardial perfusion reserve/deficits in humans using T2* (2), in normal pigs using T2 (3) and in canine model of stenosis using SSFP signal contrast (4). We extended the T2-based BOLD approach to a myocardial infarct model, exploring the advantages of 3T to evaluate regional vasodilatory function using a stress agent. The aim of the study was two-fold: 1) to evaluate oxygen-sensitive T2 changes in normal myocardium at 3T; and 2) to apply this BOLD effect in assessing serial changes in vasodilatory reserve in infarct and remote zones after myocardial infarction (MI).

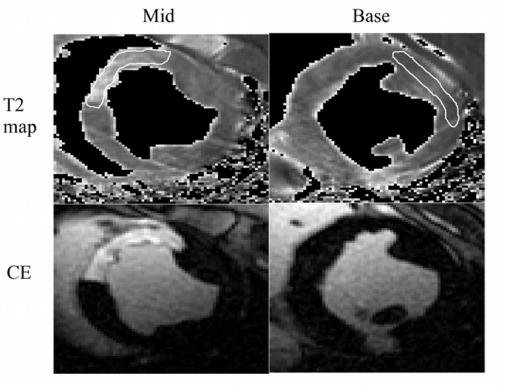

Figure 1: Representative short axis slices of porcine myocardium. T2 analysis was performed in two ROI's, as drawn above: 1) mid-ventricular anterio-septal infarct region (bright, edema) and 2) basal remote (non-infarcted) region.

Methods: 7 pigs underwent MRI before LAD occlusion (control) with subgroups studied at 2, 7, 14, 28 and 42 days post-infarction. Histology was performed upon sacrifice at either Day 14 (N=3) or 42 (N=4). Imaging was performed on a 3T MRI clinical scanner (MR 750, GE Healthcare). T2 measurements were performed using a previously validated T2-prepared spiral imaging sequence with the following parameters (5): sixteen 12.3 ms spirals (3072 points), 5 TE's (2.9-184.2 ms). An interecho spacing, τ=6 ms, was chosen due to it's relative insensitivity to field inhomogeneities. The sequences were repeated after coronary vasodilation with intravenous injection of Dipyridamole (0.56 mg/kg over 4 min). A contrast-enhanced (CE) IR-GRE sequence was used for infarct delineation using Gadolinium-DTPA (0.2 mmol/kg; Magnevist). For comparison purposes, 3 control animals were scanned on the GE 1.5T MRI scanner, of which 1 was also scanned on the 3T; both τ=6 and 12 ms were considered. In order to examine the effect of field strength on oxygen-sensitive T2 changes, a two-compartment model of tissue microcirculation (intra- and extra-vascular) was adopted (6); resting myocardial blood volume was assumed to be 5%, which increased by 30% in the vasodilated (stress) state.

Results: Tables 1 and 2 compare the theoretical and experimental values of oxygen-sensitive T2 changes in control pigs. Both τ's demonstrated greater sensitivity at 3T than 1.5T although the effect of increasing τ was less apparent at 3T. Figure 1 demonstrates T2 maps and CE images of mid-ventricular (infarct-slice) and basal regions (remote-slice) of representative pig myocardium at 2 weeks post-MI. Figure 2 shows the evolution of T2 in infarct and remote regions under rest and stress states. In remote regions, stress-induced T2 elevations were statistically significant at all time points ($p<0.04$) except at weeks 1 and 2. We also noted a subtle but significant T2 elevation in the rest state (42.4 vs 40.3ms control, $p<0.03$) at week 1. In the infarct territory, rest and stress T2's were both elevated compared to remote tissue, particularly after week 1 (indicating edema); however differences between the two states were not significant.

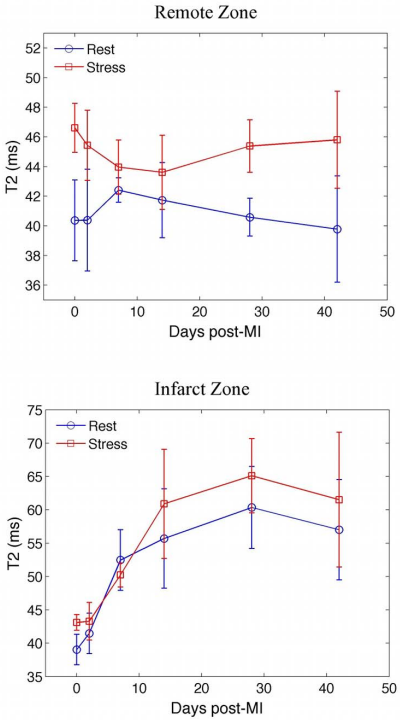

Figure 2: Plots demonstrate evolution of T2 after MI in remote and infarct zones under rest and stress states. Error bars indicate standard deviation over all animals.

Discussion: The benefit of 3T over 1.5T systems with respect to observing T2-based BOLD contrast has been previously demonstrated in blood (7). We explored the utility of the T2-based BOLD effect in probing regional myocardial oxygenation after MI on a 3T system. In control animals, T2 changes with vasodilation matched well with theoretical predictions, demonstrating the advantage of higher field strength. Suppressed stress response in the remote region between day 2 and week 4 could be suggestive of an already-vasodilated state resulting from a systemic inflammatory response to the infarction, which eventually resolves. T2 changes with stress seen in infarct zones of some animals might be attributed to a small area of salvageable myocardium. T2 at 3T appears to be a sensitive indicator of vasodilatory alterations in damaged and remote myocardium following MI.

Table 1: T2 (ms) at 1.5T

τ = 6 ms	*Rest*	*Stress*	*% change*
Theory	44.1	45.1	2.3
Exp	41.8	42.5	1.7
τ = 12 ms			
Theory	41.4	45.3	9.4
Exp	43.2	47.7	10.4*

* $p<0.01$

Table 2: T2 (ms) at 3T

τ = 6 ms	*Rest*	*Stress*	*% change*
Theory	40.2	46.3	15.2
Exp	40.4	46.6	15.3*
τ = 12 ms			
Theory	38.4	45.0	17.2
Exp	38.3	43.8	14.3**

* $p<0.0001$, ** $p<0.01$

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