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T2-based BOLD effect in myocardial infarction: a study at 3 T

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Introduction

Coronary vasodilator dysfunction has been demonstrated in infarcted as well as remote myocardium in patients with acute coronary syndrome. Recently, blood-oxygen-level-dependent (BOLD) approaches have been employed to probe myocardial perfusion reserve using T2, T2* and characterize stenosis using SSFP signal contrast. We extended the T2-based BOLD approach to a myocardial infarct model, exploring the advantages of 3 T to evaluate regional vasodilatory function using a stress agent.

Purpose

The aim of the study was two-fold, 1) to evaluate oxygensensitive T2 changes in normal myocardium at 3 T, 2) to apply this BOLD effect in assessing serial changes in vasodilatory reserve in infarct and remote zones after myocardial infarction (MI).

Methods

7 pigs underwent MRI before LAD occlusion (control) with subgroups studied at 2,7,14 and 30-42 days post-inf-arction. Histology was performed upon sacrifice at either Day 14 (N = 3) or 30-42 (N = 4). Imaging was performed on a 3 T MRI scanner (MR 750, GE Healthcare). T2 quantification was performed using a previously validated T2-prepared spiral sequence (interecho-spacing, 6 ms). The sequences were repeated after coronary vasodilation with intravenous injection of Dipyridamole. A contrast-

enhanced (CE) IR-GRE sequence was used for infarct delineation.

Results

Table 1 compares the theoretical and experimental values of oxygen-sensitive T2, demonstrating the advantage of higher field strength with respect to changes observed (15% vs 2%). 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 cumulative evolution of T2 in infarct and remote regions under rest and stress state. In remote regions, stress-induced T2 elevations were statistically significant at all time points (P < 0.04) except at week-1,2. We also noted a subtle but significant T2 ele-

Table 1: Oxygen-sensitive T2 changes

Bo = 1.5 T	T2 (ms, rest)	T2 (ms, stress)	% change
Theory	44.1	45. I	2.3
Experiment	43.2	44 . I	2.1
Bo = 3 T	T2 (ms, rest)	T2 (ms, stress)	% change
Theory	40.2	46.3	15.2
Experiment	40.4	46.6	15.3*

^{* -} P < 0.0001

Resting blood volume was assumed to be 5%, which increases by 30% in stress state $\,$

Theory - Two-compartment tissue model

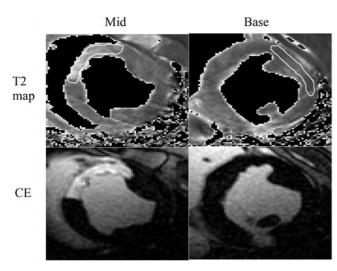
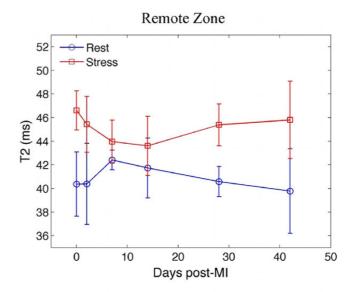


Figure I
Row I shows T2 maps while 2 shows CE images of
representative porcine myocardium in infarcy (mid)
and remote (base) slices. ROI in anterio-septal infarct
region indicated edema as reflected by eleveated T2 (bright).
(Note that this animal also developed an inferio0lateral infarct). Indicated ROIs were chosen for T2 analysis.

vation in the rest state (42.4 vs 40.3 ms 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, however differences between the two were not significant.

Conclusion

We have demonstrated the utility of the T2-based BOLD effect in probing regional myocardial oxygenation after MI on a 3 T system. Suppressed stress response in remote region between day-2 and week-4 could be suggestive of an already-vasodilated state resulting from a systemic acute inflammatory response, which eventually resolves. T2 changes with stress seen in infarct zones of some animals could be attributed to salvageable myocardium. T2 at 3 T appears to be a sensitive indicator of vasodilatory alterations in remote myocardium following MI.



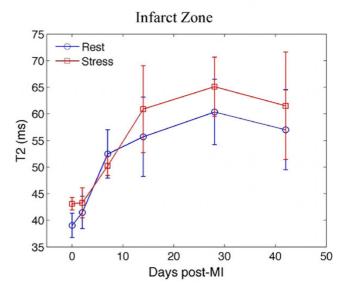


Figure 2
Plots demonstrate evolution of T2 after MI in remote and infarct zones under rest and stress states.

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