An accurate and quantitative T2 Mapping technique to detect myocardial edema in acute coronary syndrome

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Objective: To develop a rapid, quantitative and accurate myocardial T2 Mapping technique to detect edema associated with acute myocardial infarction and demonstrate its ability to address some of the problems associated with T2 Weighted (T2W) imaging; to test this new method in porcine model and patients.

Background: T2W imaging sequences can detect myocardial edema associated with acute inflammation, infarction, and the area at risk, but these techniques suffer from several drawbacks(1). In this work, we describe a rapid technique for quantitative myocardial T2 mapping. This method is expected to quantitatively differentiate edema from normal tissue, to be insensitive to tissue motion, to easily distinguish edema from stagnant blood, and to be immune to surface coil sensitivity variations. The proposed T2-mapping method can be performed with either a short breath-hold or respiratory navigator gating.

Methods:

Sequence: A single-shot T2-prepared SSFP (T2P-SSFP) acquisition was used to generate images with three T2-prep echo times: 0 (i.e., no T2 prep), 24, and 55 ms with parameters listed in Table 1. The technique is relatively motion insensitive due to the fast SSFP readout and the non-selective T2-preparation pulse. T2 maps were produced by fitting pixel intensities to a two-parameter mono-exponential model (Signal = $M_0 * \exp(-TE/T2)$), and any pixel with T2 > 120 ms was set to zero.

Healthy subjects: T2 Maps were acquired in 6 healthy subjects to determine the normal range of T2 values. Three short-axis (SAX) and two long-axis views were imaged during breath hold (duration ~ 7 HB) and in free breathing using navigator gating. In 5 subjects, four averages were acquired with navigator gating to test the benefits of increased SNR. Average T2 values were calculated in 16 myocardial segments using both methods and compared using a paired t-test.

Signal intensity variability: In 6 healthy subjects, T2W images using conventional dark-blood STIR turbo spin echo (DB-STIR-TSE) were acquired and compared to the T2 maps generated using the proposed method. Only anterior coil elements were used to investigate signal variability due to surface coil intensity variation, as well as motion induced signal loss. Parameters are listed in Table 1. In each subject, average signal was computed and normalized to the maximum segment. The standard deviation (SD) of this normalized mean was used as a measure of variability.

Animal studies: Three pigs underwent 90 minute LAD occlusion and were imaged within six hours of reperfusion.

Patient data: Two patients with Acute Coronary Syndrome were imaged. Clinical end-points were established from coronary angiography and cardiac biomarkers. The new T2 Mapping technique was compared to DB-STIR-TSE(2). Viability imaging was performed using delayed enhancement to determine the infracted region.

Results:

Healthy subjects: The T2 Values in 6 subjects showed statistically significant variation (p-value<0.001, ANOVA test for 6 subjects using 16 segments). Accordingly, we report a range of values: 40.5 to 54.8 ms. There was no significant difference between breath-hold and free-breathing technique (p-value = 0.76, paired t-test).

Signal intensity variability: Signal in T2W DB-STIR-TSE showed high variability (36.7%) while T2 maps showed no such variation (3%) as seen in Figure 1.

Animal Study: Results from the pig study are shown in Figure 2 and Table 2. In each animal, the occluded-reperfused segment showed a T2 value > 2SD of remote segment.

Patients: T2W imaging had high static blood pool signal and making it difficult to detect edema. In one case, the high blood signal obscured edema (Fig 3). T2 Maps were able to detect edema in the infarct-related artery territories from remote myocardium (Figure 3 for Patient 1; Figure 4 and Table 3 for Patient 2)

Conclusion:

We have demonstrated a rapid method of T2-mapping for quantitative detection of myocardial edema in porcine model and two patients. Direct quantification of T2 eliminates many unwanted sources of signal variation, and removes the subjectivity of observer interpretation of bright regions. Further studies with more patients are required to assess sensitivity and specificity.

Table 1: Imaging Parameters

Parameter	T2Prep SSFP	STIR
TE (ms)	0, 24, 55	62
TR	3xRR	2xRR
Total Imaging time	7xRR	10xRR
Avg. FOV	350 x 400	350 x 400
Image Matrix	96 x 160	144 x 192
Flip angle	40	90

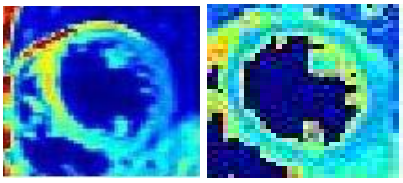

Figure 1: Surface coil effects in T2W(l) and T2Maps (r)

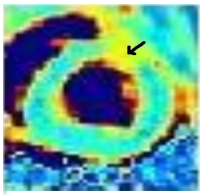

Table 2: Pig study results

	T2 (ms) in segment	
pig	infarct	remote
1	83.5±11	58.6±4.5
2	80.4±6.8	51.1±5.6
3	83.6±10	58.4±7.2

Fig 2: T2 Maps from one of the pigs showing T2 Enhancement (arrow) in T2 Map. Table 2 gives the quantitative values in all 3 pigs

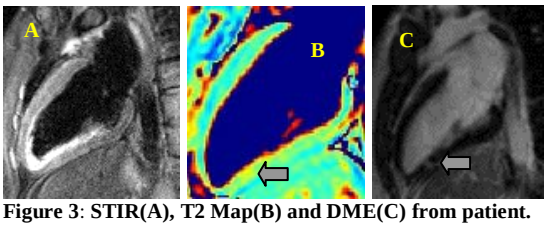

Figure 3: STIR(A), T2 Map(B) and DME(C) from patient. Note how the static blood signal is obscuring the apical edema. T2 Map was able to detect this edema (T2=85±7 ms vs 60±7 ms in remote segment)

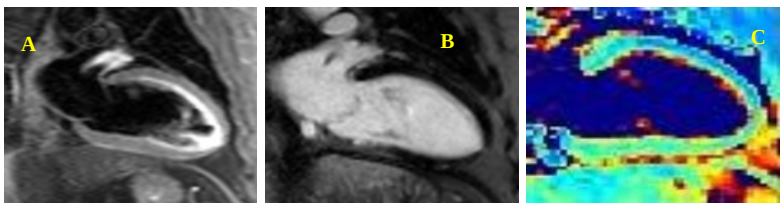

Figure 4: STIR (A), ACUTE (B), DME (C) and T2Maps (D) from a patient with STEMI. Note the high blood pool signal in the apical regions of STIR; T2 Maps address this concern. The quantitative values for this patient from VLA and SAX are given in tables 3 and 4

Table 3: T2 (ms) in myocardial segments from VLA and SAX. Segments with high T2 are marked in red.

View	Segment	T2 (mean±SD)
VLA	**Basal inferior**	**63.9±6.6**
	Mid inferior	**63.3±3.5**
	Apical inferior	59.5±6.1
	Apical anterior	46.6±8.1
	Mid anterior	46±5.7
	Basal anterior	47.4±6.6
Base SAX	Anterior	46.5±5.5
	Anteroseptal	50.9±6.7
	Inferoseptal	51.3±6
	Inferior	**65.7±6.7**
	Inferolateral	57.9±9.1
	Anerolateral	48.9±9.2

References:

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2. Simonetti OP, et al. Radiology. 1996; 199(1):49-57.