

# Repeated, Pixel-By-Pixel T1-Mapping in Canine Myocardial Infarction, Using an Infarct-Avid, Persistent Contrast Agent, Gd(ABE-DTTA)

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## Introduction

Contrast enhanced magnetic resonance imaging (ceMRI) has the potential to detect viability following myocardial infarction (MI)<sup>1</sup>. Contrary to signal intensity (SI), the longitudinal relaxation time (T1) is an intrinsic parameter. Determining T1 after the administration of an infarct-avid CA, therefore, allows the visualization of infarcted tissue in a manner that eliminates many confounders of SI (field inhomogeneity, T2-effects, time of imaging, differences in equipments and pulse sequences used)<sup>2</sup>. Pixel-by-pixel T1 maps in six dogs were generated repeatedly, 24 through 96 hours following the administration of an infarct-avid, persistent contrast-agent (PCA), Gd(ABE-DTTA)<sup>3</sup>. Distribution of infarcted (PCA accumulating), versus viable (non-accumulating) tissue was detected with high-resolution. In this work we demonstrate the feasibility of repeated in-vivo T1-mapping in an in vivo closed-chest canine model, using a single administration of PCA.

## Methods

Following the administration of 0.05mmol/kg Gd(ABE-DTTA to LAD occluded (180min) and reperfused dogs, high spatial resolution inversion-recovery images were generated with varying inversion times (TI). The delayed enhancement sequence of a 1.5T GE Signa CV scanner was used with the following imaging parameters: FOV=300mm, matrix=256x256, slice=10mm, flip angle=25°, echo time=3.32ms, VPS=16, TR~1200-2400 ms (2-3 cardiac cycles, depending on heart rate of individual dogs, but constant throughout an individual T1 mapping experiment). Six to nine TIs were used in the range of 200-1000ms. T1 was calculated from the TI dependence of the signal intensity (SI), using a non-linear, three-parameter, least-squares curve-fitting routine, in a pixel-by-pixel manner<sup>4</sup>. A single short-axis (SAX) slice through the center of infarction was imaged in four dogs. Infarcted areas with decreased T1 due to PCA accumulation were measured in each T1 map and results of the 72h time point were validated with the postmortem gold-standard, TTC staining. In two dogs, the left ventricle (LV) was tomographically divided into six SAX slices and the three-chamber, long-axis (LAX) view was used to assess the apex. Thus a 3D reconstruction of the LV was obtained.

## Results

Figure 1. shows average T1 results in infarct core versus in remote, viable tissue following a single administration of PCA (n=6). From 24 hours through 96 hours following PCA administration, average T1 values in remote areas were 733, 810, 850 and 892ms. Corresponding T1 values in the core of the infarct were 476, 489, 400 and 528ms. At all four time points there was significant (p<0.01) difference between infarcted and non-infarcted T1 values. Peak PCA accumulation in the infarct was detected 72 hours following administration. Figure 2. shows the 3D reconstruction of an LV 24 hours following PCA administration. Figure 3 shows the correspondence between in vivo T1-mapping and postmortem TTC staining. There was significant correlation (P<0.01, R=0.97) between infarct area measurements in T1 maps and area measurements in TTC staining photos (n=4).

## Conclusions

We have shown that using a single administration of infarct-specific PCA, high-resolution T1-maps can be generated repeatedly for the entire LV, based on inversion-recovery images with multiple inversion times. In this manner, PCA accumulation can be visualized while eliminating many confounders existent in SI measurements. This method is more sensitive to the presence of viable tissue than traditional threshold-planimetry using SI. Inhomogeneities in infarcted areas are revealed, suggesting the presence of patches of viable tissue in these areas. Repeated T1 mapping could be a useful tool in clinical monitoring of infarct expansion and reinfarction, as well as in predicting long-term functional recovery. Multi-center studies of viability, using a PCA with T1-mapping, could be standardized more effectively, improving comparability and reproducibility.

## References

1. Kim RJ, Wu E, Rafael A, et al. *N Engl J Med.* 2000;343:1445-53.
2. Messroghli DR, Niendorf T, Schulz-Menger J, et al. *J Cardiovasc Magn Reson.* 2003;5:353-9.
3. Saab-Ismail NH, Simor T, Gaszner B, et al. *J Med Chem.* 1999;42:2852-2861.
4. Kaldoudi E, Williams SCR. *Concepts in Magnetic Resonance.* 1993;5:217-242.

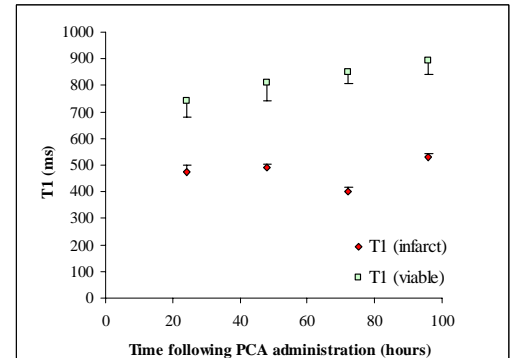


Figure 1.

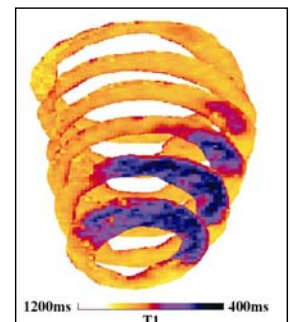


Figure 2. 3D reconstruction of multi-slice color-coded T1maps of a left ventricle 24 hours following PCA administration. T1 is decreased in the region supplied by LAD due to the accumulation of PCA in infarcted tissue.

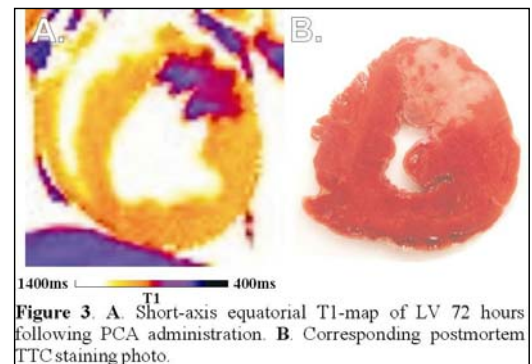


Figure 3. A. Short-axis equatorial T1-map of LV 72 hours following PCA administration. B. Corresponding postmortem TTC staining photo.