

# Navigator-gated T2 and T2\*-weighted imaging of myocardial edema and hemorrhage following primary coronary intervention.

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## Introduction

Occlusion of a coronary artery leads to myocardial tissue edema in the vascular bed downstream of the vessel. This develops after an hour or so of occlusion and may persist for several months. The increase in mobile water content within ischemic myocardium causes a prolongation of T2-relaxation. Therefore the extent of hyperintense edema on T2-weighted images allows the area at risk (AAR) from ischemic injury to be retrospectively determined. However, reperfusion of severely ischemic myocardium also leads to interstitial hemorrhage and this may be an important marker for irreversible microvascular damage. The effects of hemorrhage on T2 signal are complex but between 1 to 7 days post-reperfusion paramagnetic effects appear to predominate. Cardiac T2\* mapping has the potential to quantify the extent of myocardial hemorrhage and compare it to other indices of ischemic injury. Furthermore, the paramagnetic effects of hemorrhage would also be expected to cause signal loss on T2-weighted spin echo images which may lead to an important underestimation of the ischemic AAR using signal threshold criteria.

## Purpose

We assessed the feasibility of using T2\* mapping to quantify regions of myocardial hemorrhage following percutaneous primary coronary intervention (PPCI) for acute myocardial infarction. We also assessed myocardial edema imaging using a T2-weighted asymmetric turbo spin echo with spectrally-selective inversion recovery (SPIR) fat suppression. To reduce respiratory motion artifact and ghosting from the blood pool we used navigator gating and a black blood prepulse in both sequences.

## Methods

Fifteen patients who had recently undergone PPCI within the previous 7 days were imaged. Left ventricular function was assessed with conventional cine sequences. Myocardial edema was imaged with the T2-weighted SPIR sequence (voxel size 1.9 x 2.6 x 10 mm, flip angle 90°, slice thickness 10mm, bandwidth 467 Hz/pixel, TE 100 ms, TR 2 R-R intervals). Myocardial hemorrhage was imaged with a black-blood multiecho T2\* sequence using navigator respiratory-gating (voxel size 1.7 x 2.8 x 10 mm, flip angle 20°, slice thickness 10mm, bandwidth 1387 Hz/pixel, TR 17 ms, 7 echoes, TE 2.3 – 16.1 ms,  $\Delta$ TE 2.3ms). Microvascular obstruction (MVO) and late gadolinium enhancement were imaged at 1 minute and 15 minute delays respectively using a 3 dimensional inversion-recovery sequence (1.4 x 1.4 x 8 mm, flip angle 15°, slice thickness 8mm, bandwidth 266 Hz/pixel, TE 1.4 ms, TR 4.3 ms). The area of myocardial edema on the T2 SPIR images was measured with a level-set boundary detection algorithm. This was compared to a conventional signal intensity threshold method using 2, 3 and 5 standard deviations (sd) above the mean of remote normal myocardium. A salvage index was calculated as the proportion of the AAR that did not show late enhancement. T2\*-mapping of the left ventricle was performed using a threshold of 20ms to define the presence of hemorrhage.

## Results

The mean area of hemorrhage was 5.0% at the level of the infarct. There was a close correlation between hemorrhage and the MVO ( $r^2=0.75$ ,  $p<0.01$ ) and infarct volumes ( $r^2=0.76$ ,  $p<0.01$ ) (Figure 1). When  $\geq 5\%$  hemorrhage is present the AAR was underestimated by 50% at a 5 standard deviation threshold compared to a boundary detection tool (21.8% vs 44.0%,  $p<0.05$ ). Estimation of myocardial salvage at 3sd and 5sd signal thresholds becomes unreliable in hemorrhagic infarcts as the apparent AAR becomes smaller than the actual infarct size (Figure 2).

## Discussion

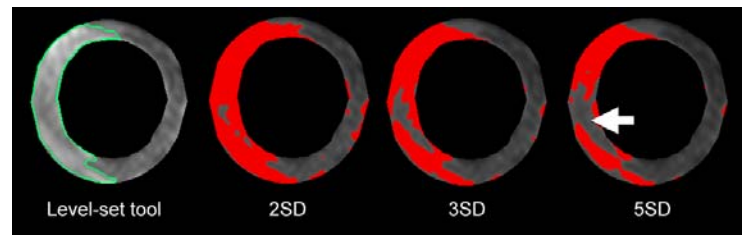
Our findings demonstrate the feasibility of using T2\* mapping to quantify myocardial hemorrhage following infarct reperfusion. Hemorrhage is frequently observed and is associated with large infarcts where MVO is present and is an indicator of poor myocardial salvage. Hemorrhage in the core of the infarct causes signal loss on T2-weighted imaging and boundary-detection is required to reliably assess the AAR. T2-weighted asymmetric turbo spin echo with SPIR fat suppression and navigator gating shows promise as a technique for imaging the edematous myocardium and reduces unsuppressed blood signal adjacent to stunned myocardium.

## Conclusion:

Studies using CMR to determine the AAR and myocardial salvage should use boundary detection methods for quantification as arbitrary signal thresholds are unreliable when hemorrhage is present. Post-reperfusion hemorrhage can be assessed with T2\*-mapping and may provide an imaging marker of poor myocardial salvage.



**Figure 1** – A patient with a reperfused anterior myocardial infarction. A) T2\* map of the left ventricle. Pixels in red show hemorrhagic myocardium with a T2\* $<20$ ms (arrow). Localised susceptibility artefact is present (arrowhead) B) Boundary of MVO, and C) late enhancing infarct.



**Figure 2** – Masked T2-weighted images from the same patient. Boundary segmentation has been used to define the edematous AAR. At increasing signal intensity thresholds there is a progressive decrease in apparent AAR due to hypointensity in the hemorrhagic infarct core (arrow).