

## DE-MRI for Identifying the Ventricular Arrhythmia Substrate in Ischemic and Non-Ischemic Cardiomyopathy

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### Introduction:

Scar tissue in the myocardium is present in patients with ischemic cardiomyopathy (ICM) and often noted in patients with non-ischemic cardiomyopathy (NICM). Delayed-enhanced magnetic resonance imaging (DE-MRI) can precisely define the extension and distribution of this scar tissue. Such scar tissue can act as arrhythmogenic substrate and lead to ventricular arrhythmia. Patients with ICM and NICM who present with ventricular arrhythmia can undergo ablation therapy to eliminate these arrhythmia.

### Methods:

DE-MRI was performed in 42 consecutive patients (mean age  $55 \pm 14$  years) with ICM or NICM (mean ejection fraction  $36 \pm 10\%$ ) referred for catheter ablation of ventricular tachycardia (VT) or premature ventricular complexes (PVCs). If scar tissue was found on the DE-MRI, the myocardial contours and scar distribution was semi-automatically extracted, in order to generate 3-D maps of scar distribution. These maps were then integrated with the electroanatomic map in either a 2D polar display (Figure 1) or a 3D display using the CARTO Merge function (Figure 2). This integration involved initial matching of fiducial markers (LV apex, center of mitral valve, aortic outflow tract) in both modalities, and then a surface based optimized registration within the CARTO Merge software. Mapping data were correlated with respect to the localization of scar tissue (right ventricular vs left ventricular and endocardial vs epicardial vs intramural).

### Results:

Scar tissue was identified by DE-MRI in 13 out of 28 patients with NICM and in all (n=14) patients with ICM. Characteristics of these 27 patients were as follow. Patients had either myocardial infarct (n=14), sarcoidosis (n=3) or dilated cardiomyopathy (n=10). They either had a single focus on DE-MRI (n=19) or multifocal disease (n=8). The ventricular arrhythmia were VT (n=20) or PVC (n=8). The distribution was predominantly endocardial (n=12), midmyocardial (n=4), epicardial (n=2) and transmural (n=9). On the electroanatomic map, there was always low voltage present and matching the endocardial or epicardial surface displaying DE on MRI. The size of the endocardial scar on DE-MRI correlated well with the size of the endocardial scar defined by voltage mapping for patients with NICM ( $45 \pm 14 \text{ cm}^2$ ,  $R=0.94$ ,  $p<0.0001$  with cutoff of  $1.5 \text{ mV}$ ) and patients with ICM ( $45 \pm 25 \text{ cm}^2$ ,  $R=0.78$ ,  $p=0.002$  with cutoff of  $1.5 \text{ mV}$ ). All patients with inducible VT or sustained VT had evidence of DE on MRI. In all patients with DE on MRI where a critical site for the arrhythmia could be identified, this critical site was confined to the scar tissue.

### Conclusions:

DEMRI in patients with ICM or NICM can help to identify the arrhythmogenic substrate; furthermore it helps to plan an appropriate mapping and ablation strategy.

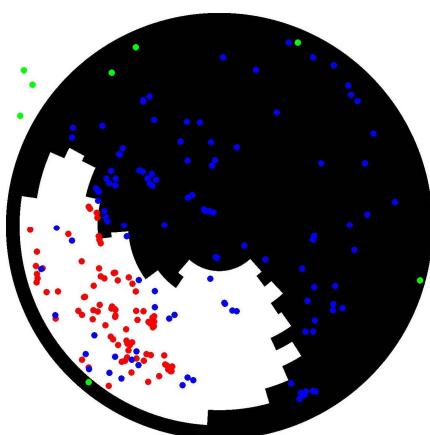


Figure 1: 2D polar map registration of scar (areas with delayed enhancement in white and normal areas in black) and points obtained from the bipolar electroanatomic map (red tags: sites with voltage  $\leq 1.0 \text{ mV}$ ; blue tags: areas with voltage  $> 1.0 \text{ mV}$ ; green tags: aortic points and mitral annular points).

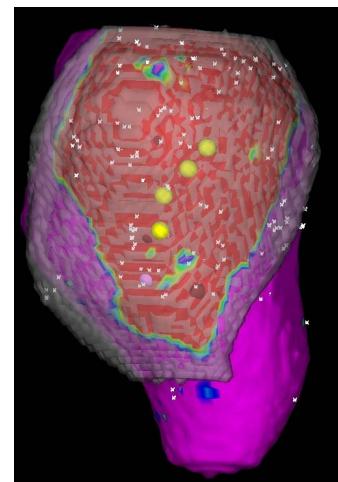


Figure 2: 3D fused DE-MRI and endocardial maps. The endocardial map is projected on the endocardial surface as determined by MRI. Areas with low voltages are illustrated in red, and areas of normal voltages are in purple.