

Detection of Infarcted and Arrhythmogenic Myocardium with DTI Tractography and Electroanatomical Voltage Mapping

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Target Audience: Scientists/clinicians interested in MRI of the myocardium.

Purpose: The tractographic propagation angle (PA) is a topographic measure of fiber architecture in the myocardium. We have previously shown in animal models that a PA > 4° can differentiate normal and infarcted myocardium.¹ We aimed here: 1) To determine whether tract PA can be used to delineate the extent of injury in patients with acute myocardial infarction, 2) to correlate PA maps of the left ventricle (LV) after myocardial infarction with electroanatomical voltage maps, and 3) to determine whether PA can facilitate the identification of arrhythmogenic foci in infarcted hearts.

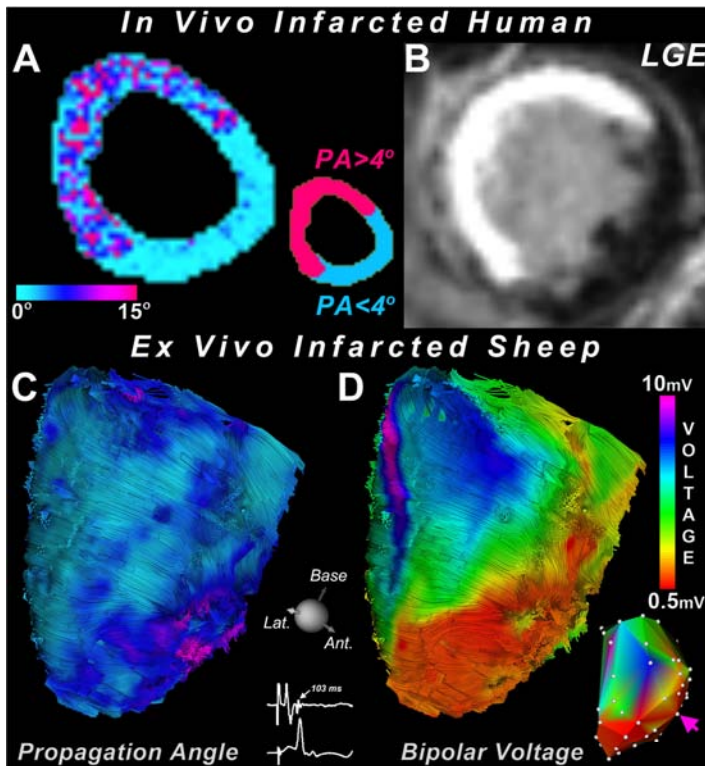


Figure 1: (A) Short axis PA map in a patient with a large anteroapical infarct. A PA threshold > 4° has been used to segment normal and injured myocardium (inset). (B) Late gadolinium enhancement (LGE) image at the same slice location. (C, D) Sheep heart with a large anteroapical infarct. In panel C, the tracts are color coded by PA, while in panel D they are color-coded by bipolar voltage. Strong agreement is seen between the area of the heart with increased PA and reduced voltage. The microstructural and electrical transitions between normal myocardium, border zone, and infarct can be seen. The inset on the right displays the original bipolar voltage map and the measurement locations. The arrow points to a location on the anterior wall where late presystolic potentials (left inset) were recorded.

linear and could be approximated by a rational polynomial function.

Discussion: The detection of myocardial infarction in patients using a PA threshold of 4° is accurate and does not require the injection of exogenous contrast agents. This could be of major value in patients with reduced renal function, in whom iodinated or gadolinium-based contrast agents may be contraindicated. PA can also be used to delineate regions of heterogeneous scar (PA 4-10°) from regions of dense scar (PA >10°) and help define the substrate for reentrant ventricular arrhythmias.

Conclusion: PA could become a valuable tool in cardiovascular imaging, in particular in patients with renal dysfunction and those at risk of sudden cardiac death.

References: 1) Mekkaoui C *et al.* ISMRM 2013. 2) Mekkaoui *et al.*, JCMR 2012.

Methods: Patients with acute myocardial infarction (MI, n=3) were scanned prior to discharge on a clinical 1.5T scanner with a diffusion-encoded stimulated echo EPI sequence and the following parameters: resolution 1.75x1.75x8 mm³, b-value of 500 s/mm², 6 diffusion encoding directions, and 8 averages. Imaging was performed in the diastolic sweet spot of the cardiac cycle to mitigate the effect of strain. Late gadolinium enhancement (LGE) was performed after the injection of 0.2 mmol/kg of Gd-DTPA using an inversion recovery gradient echo sequence. Infarcted sheep (n=5) were also studied. Large anteroapical infarcts were created in the sheep by balloon occlusion of the left anterior descending coronary artery. Bipolar voltage mapping of the LV was performed at 3 months using the CARTO® 3D electroanatomic mapping system. The hearts were then perfusion-fixed and excised. Cardiac diffusion tensor MR imaging (DTI) was performed on a clinical 3T scanner, as previously described,² with a resolution of 2x2x2 mm³, b-value of 2000 s/mm², and 6 diffusion-encoding directions. PA values for each heart were computed along myofiber tracts using an adaptive 5th order adaptive Runge-Kutta method and co-registered with the LGE and bipolar voltage data. Infarcted myocardium by LGE was defined by a signal intensity > 2 standard deviations (SD) above background. Bipolar voltage (V) was used to segment the sheep hearts into normal regions (> 1.5 mV), heterogeneous scar (0.5-1.5 mV), and dense scar (< 0.5 mV). PA in the sheep hearts was more densely sampled than voltage and each voltage point was thus associated with a distribution of PA values. Results are reported on a per slice basis.

Results: PA was markedly increased in segments of myocardium showing LGE and robustly delineated the area of infarction in all patients imaged (Fig. 1A, B). A strong correlation was also seen between elevated PA and reduced myocardial voltage in the infarcted sheep hearts (Fig. 1C, D). Coregistration of the voltage and PA maps allowed the fibers in the heart to be color coded by either PA (Fig. 1C) or the local bipolar voltage (Fig. 1D). PA in the remote zone of infarcted patients and sheep averaged 4.65±0.45° and 2.9±0.65°, respectively (Fig. 2A). An excellent correlation (R² = 0.97) was seen between infarct size by LGE and PA in the patients with MI (Fig. 2B). In the sheep hearts, PA was < 4° in electrically-normal myocardium, between 4° and 10° in areas with heterogeneous scar, and > 10° in regions of dense scar (Fig. 2C). The relationship between voltage and PA was non-

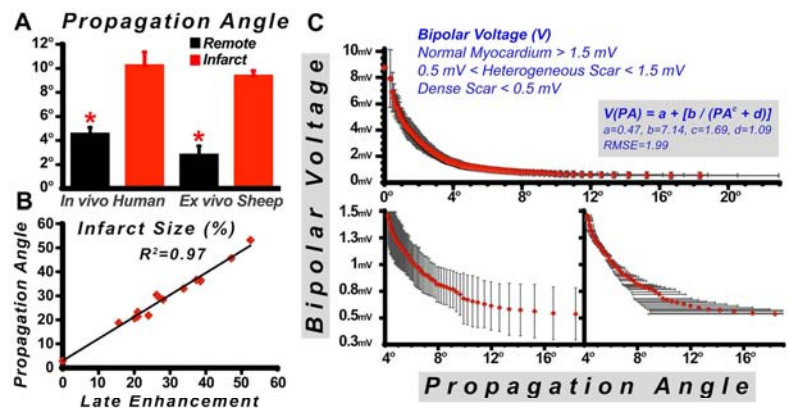


Figure 2: (A) PA is significantly elevated (p<0.05) in regions of infarction consistent with myofiber disorganization. Similar values are seen in patients with recent infarcts and infarcted sheep hearts. (B) An excellent correlation is seen between infarct size by PA (threshold 4°) and LGE in patients with recent infarcts. (C) The correlation between myocardial voltage and PA can be described by a rational polynomial curve. Regions of normal myocardium are characterized by voltage > 1.5 mV and PA < 4°. Regions with heterogeneous scar have a voltage between 0.5-1.5 mV and PA between 4-10°. Dense scar is characterized by PA >10° and a very low voltage.