Impact of Changing Activation Sequence on Bipolar Electrogram Amplitude for Voltage Mapping of Left Ventricular Infarcts Causing Ventricular Tachycardia

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Abstract. Introduction: Wavefront direction is a determinant of bipolar electrogram amplitude that could influence identification of low amplitude regions indicating infarction or scar.

Methods: To assess the importance of activation sequence on electrogram amplitude 11 patients with prior infarction and ventricular tachycardia were studied. At 819 left ventricular sites bipolar electrograms were recorded during atrial pacing and ventricular pacing, followed by unipolar pacing with a stimulus of 10 mA at 2 ms. Sites with a pacing threshold >10 mA were designated electrically unexcitable scar.

Results: Areas of low voltage (≤1.5 mV) were present in all patients. Atrial paced and ventricular paced electrogram amplitudes were strongly correlated (r = 0.77; p < 0.0001). Changing the activation sequence (from atrial pacing to ventricular pacing) produced a >50% change in electrogram amplitude at 28% of sites and a >100% change at 10% of sites, but only 8% of sites had an electrogram amplitude classified as abnormal (≤1.5 mV) with one activation sequence and normal (>1.5 mV) with the other activation sequence. Electrically unexcitable scar (6% of sites) was associated with lower electrogram amplitude but could not be reliably identified based on electrogram amplitude alone for either activation sequence.

Conclusion: Voltage maps created with bipolar recordings using these methods should be relatively robust depictions of abnormal ventricular regions despite variable catheter orientation and activation sequences that might be produced by different rhythms.

Methods

Patients

Left ventricular mapping was performed in 11 male patients (mean age 68 ± 9 years) who had at

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least one remote (>2 months) myocardial infarction with a mean left ventricular ejection fraction of 25 ± 9% and a range of 15 to 40%, who were referred for catheter ablation of recurrent sustained monomorphic VT (≥2 episodes in the preceding 6 months).

**Mapping**

Catheter mapping and ablation and data collection were performed prospectively according to protocols approved by the institutional review board of Brigham and Women’s Hospital, Boston, MA after obtaining written informed consent. Left ventricular (LV) access was achieved with a retrograde aortic or transseptal approach. Systemic anticoagulation was maintained with heparin.

Mapping used the non-fluoroscopic electroanatomic mapping system CARTO® (Biosense Webster, Diamond Bar, CA) [3]. Electrograms recorded from the catheter are stored in combination with their exact anatomic location with a spatial accuracy of ±0.8 mm [4]. The 7F deflectable, quadripolar catheter has a 4 mm ablation tip and a spacing of 1.7 and 4 mm between the distal, middle and proximal electrodes, respectively. Bipolar electrograms were recorded from the distal electrode pair and filtered at 10–400 Hz. At each stable ventricular site electrograms were recorded during atrial pacing, then during right ventricular pacing. Pacing was then performed from the mapping catheter (pace mapping) at one consistent paced cycle length for each patient, between 500–700 ms. Pace mapping utilized unipolar stimuli with a strength of 10 mA and pulse width of 2 ms [5]. This fixed stimulus strength was chosen due to time constraints of ventricular mapping and because we have extensive experience pacing at this stimulus strength for mapping purposes and have observed reliable capture in normal and abnormal hearts. Catheter stability was assessed from fluoroscopy, the CARTO mapping system, electrogram stability, and consistent capture or lack of capture with pacing. If pacing did not capture the position of the catheter was reviewed with biplane fluoroscopy and assessed for possible poor contact. Sites where pacing capture was intermittent or where motion of the catheter or gentle additional pressure moved the catheter suggesting that it was not in physical contact with the endocardium were excluded from analysis.

Custom software allowed the intracardiac electrograms and 12 surface ECG leads for each site to be digitized and stored during atrial pacing, right ventricular pacing, and pace mapping before moving the catheter to the next mapping site. The maximum electrogram amplitude (peak to peak) from the distal electrode pair of the mapping catheter was automatically measured and displayed on corresponding maps. Sites were manually reviewed to ensure exclusion of pacing stimuli from amplitude measurements.

**Statistical Analysis**

All values are expressed as mean ± standard deviation. Continuous variables are compared using the two-tail unpaired t-test. Correlation coefficients were calculated with the Pearson test. Discrete variables were compared using the Fisher exact test. Generalized estimating equations were used to adjust for multiple observations in individual patients. [6]. A two-sided probability value of <0.05 was considered significant. Calculations were performed using SAS Statistical Software (Version 6.12, Cary NC).

**Results**

All patients had large regions of low (≤1.5 mV) voltage electrograms consistent with infarcts. Infarct locations were inferior or posterior in 8, anterior in 1, and in two discrete regions in two patients (posterobasal and inferoseptal in one, and anteropapical and inferoseptal in the other).

A total of 819 sites (mean per patient 75 ± 24) met criteria for analysis, of which 59% had an amplitude ≤1.5 mV during atrial pacing and 59% during right ventricular pacing. Changing the ventricular activation sequence by pacing from the atrium as compared to the right ventricle had relatively little effect on maximal peak-to-peak electrogram amplitudes, particularly at sites with very low amplitude electrograms (Table 1 and Figs. 1–3). There was a strong correlation of electrogram amplitude between atrial pacing and right ventricular pacing (r = 0.77; p < 0.0001; Fig. 3). Considering >1.5 mV as normal, 8% of sites had an abnormal electrogram amplitude (≤1.5 mV) with one activation sequence and normal (>1.5 mV) with the other activation sequence. Although substantial changes in amplitude did occur, these were more frequent in normal than abnormal regions. Changing the activation sequence changed electrogram amplitude by >50% at a similar number of low amplitude and normal amplitude sites (Table 1), with a change >1.5 mV much more often at the normal amplitude sites.

With pacing from the ablation catheter capture was present (pacing threshold ≤10 mA) at 771 (94%) sites. The 48 (6%) sites where pacing did not capture were classified as electrically unexcitable scar [5]. Electrically unexcitable sites had lower electrogram amplitude than excitable sites during atrial pacing (0.9 ± 0.8 vs. 2.1 ± 2.5 mV; p = 0.004) and during right ventricular pacing (0.8 ± 1.0 vs. 2.2 ± 2.6 mV; p = 0.002). A clearly definable amplitude threshold for identifying electrically