

Substrate-Guided Ablation of Ischemic Ventricular Tachycardia: Is it Really Necessary to Induce Ventricular Tachycardia to be Successful?

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Abstract

Traditionally, catheter ablation of ventricular tachycardia (VT) consists of mapping the circuit during VT, and using pacing maneuvers to localize critical isthmus of the circuit. However, substrate-guided ablation of VT is a relatively new technique and involves identifying potentially arrhythmogenic regions of myocardium in sinus rhythm and avoids the need for mapping during VT. In this short editorial article, we aimed to discuss substrate mapping of VT.

Keywords: Ablation; Tachycardia, Heart failure, Cardiomyopathy

Introduction

Catheter ablation of ventricular tachycardia (VT) currently has an important role in the treatment of ischemic ventricular tachycardia. Conventional activation mapping techniques require ongoing tachycardia and haemodynamic stability during the procedure [1]. However, mapping during ongoing tachycardia is not always possible due to haemodynamic deterioration. Furthermore, non-inducibility of clinical tachycardia, poor induction reproducibility, and multiple ventricular tachycardias with frequent spontaneous changes of morphology complicate tachycardia activation mapping. To overcome all these limitations, substrate mapping strategies have been developed. The potential advantage of this method is that it allows mapping and ablation in sinus rhythm. In this short communication we aimed to discuss substrate mapping approach in patients with ischemic ventricular tachycardia.

The Birth of Substrate Mapping

In 2000, Marchlinski et al. [2] firstly used substrate mapping in the patients with unmappable ventricular tachycardia. They evaluated 16 patients with drug refractory, monomorphic, unmappable VT. Nine of these patients had ischemic cardiomyopathy (ICM). Patients underwent bipolar catheter mapping during baseline rhythm. Radiofrequency point lesions extended linearly from the "dense scar," which had a voltage amplitude <0.5 mV, to anatomic boundaries or normal endocardium. Twelve patients (75%) have been free of VT during 3 to 36 months of follow-up.

Arenal et al. [3] studied 24 patients with documented unmappable monomorphic VT. Twenty-one patients had ischemic cardiomyopathy. The reason of failed conventional activation mapping was non-inducibility in 12 patients and haemodynamic instability in 6 patients. Endocardial electroanatomic activation maps (Carto System) during sinus rhythm (SR) and right ventricular apex (RVA) pacing were obtained to define areas for which an electrogram displayed isolated, delayed components (E-IDC). These electrograms were characterized by double or multiple components separated by ≥ 50 ms. Ablation guided by E-IDC suppressed all but one clinical VT whose inducibility suppression was tested. During a follow-up period of 9 ± 4 months, three patients had recurrences of the ablated VT and two of a different VT. Nakahara et al. [4] compared the characteristics and prevalence of late potentials (LP) which commonly seen in post-infarction scars, reflect areas of myocardium where conduction is slowed and interrupted by fibrosis in patients with non-ischemic cardiomyopathy (NICM) and ICM etiologies and evaluated their value as targets for catheter ablation. Seventeen of 33 patients had ischemic VT. The LP were defined as low voltage electrograms (<1.5 mV) with onset after the

QRS interval. Very late potentials (vLP) were defined as electrograms with onset >100 ms after the QRS. An LP-targeted ablation strategy was effective in 82% of ICM patients at a 12 ± 10 months of follow-up.

Comparison of Results of Substrate Mapping Between Ischemic and Non-Ischemic Cardiomyopathy

The efficacy of substrate-guided ablation may be less effective in patients with non-ischaemic dilated cardiomyopathy compared with those with ICM [4]. As a potential explanation, scar areas in non-ischaemic dilated cardiomyopathy may be less extensive, patchier, and may have more epicardially located involvement [5-7]. Consequently, the combined approach of activation map and substrate ablation during VT ablation in patients with non-ischemic etiology has demonstrated limited success in case of arrhythmias recurrence [8,9]. However, on the basis of substrate mapping, only a few comparison data exist for outcomes with VT ablation [4,10]. Recently, Proietti et al. [10] evaluated the outcomes of purely substrate guided VT ablation in patients with NICM and ICM and the impact of acute procedural success on long-term outcome. They found that success rates are significantly lower in patients with non-ischaemic compared with those with ischemic.

Unipolar vs Bipolar Voltage Mapping for Unmappable Scar-Related Ventricular Tachycardia Ablation

Scars causing ventricular tachycardia can extend deep to and beyond bipolar low-voltage areas (LVAs) and they may be a reason for endocardial ablation failure. Analysis of endocardial unipolar voltage maps has been used to detect scar transmural and epicardial scar. Recently, we evaluated whether endocardial unipolar LVA around the overlying bipolar LVA may predict endocardial ablation recurrence in patients with structural heart disease undergoing substrate modification [11]. Twenty consecutive patients with structural heart disease (11 ICM and 9 NICM) and undergoing substrate modification due to unmappable VT were studied. Detailed endocardial mapping was performed in all patients by using the CARTO system. Total 6242 endocardial points obtained from the left ventricle during sinus

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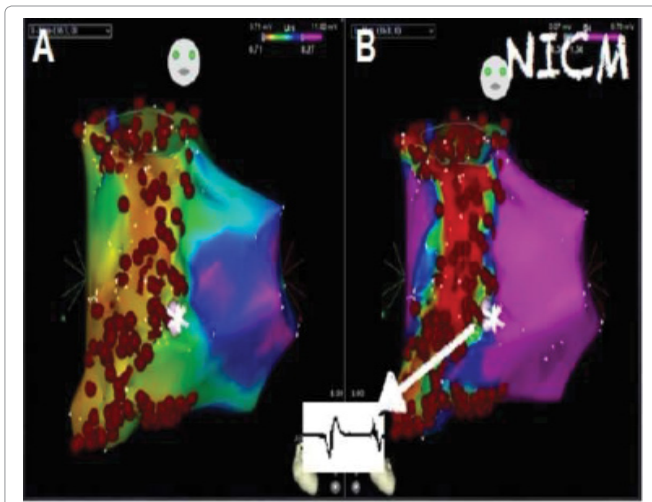


Figure 1: Voltage map of a patient with non-ischaemic cardiomyopathy. Red spheres show ablation sites. In bipolar endocardial recordings, low-voltage area and late potentials are demonstrated with green color. The arrow shows that peripheral unipolar LVA surrounding bipolar LVA may predict recurrence if it does not compass during ablation.

rhythm were reviewed. Bipolar LVA defined as <1.5 mV and unipolar LVA defined as <8.3 mV, respectively, on electro-anatomic mapping system. Peripheral unipolar LVA (pUni-LVA) surrounding bipolar LVA was measured and compared patients with and without VT recurrence at 6-month follow-up period. Mean unipolar voltage and mean bipolar voltage were 6.26 ± 4.99 and 1.90 ± 2.30 mV, respectively. Bipolar voltage and unipolar voltage in corresponding points were correlated ($r=0.652$, $P=0.0001$). In our study, in all patients with ICM, bipolar LVA was covered by unipolar LVA, all NICM patients had bipolar LVA, in majority of the cases bipolar LVA was surrounded by unipolar LVA (8 out of 9), in one patient with NICM, there was minimal bipolar LVA and surrounding unipolar LVA in inferior wall and a predominant unipolar LVA with healthy bipolar voltage in the septal wall. Unipolar LVA in the healthy bipolar voltage area (pUni-LVA) was larger in patients with NICM compared with ICM patients (55.9 ± 45 cm² vs. 21.7 ± 14.5 cm², $P=0.029$). Total bipolar LVA (91.1 ± 93.5 cm² vs. 87.5 ± 47.5 cm², $P=0.91$) and unipolar LVA (148.1 ± 96.3 cm² vs. 104.7 ± 44.2 cm², $P=0.21$) were similar in patients with and without VT recurrence, respectively. Total number of late potentials was similar in patients with and without recurrence (15.3 ± 8.2 vs. 17.8 ± 9.2 , $P=0.53$). As a unique result, pUni-LVA was significantly larger in patients with VT recurrence than without (57.0 ± 40.4 cm² vs. 17.2 ± 12.9 cm², $P=0.01$) (Figure 1). Furthermore, a 25 cm² area cut-off pUni-LVA had a sensitivity of 87% and a specificity of 70% to predict VT recurrence. So, we speculated that pUni-LVA surrounding bipolar scar may predict recurrence of VT ablation in patients with structural heart disease and unmappable VT.

Conclusion

Substrate-based ablation seems a good technique to treat VT in patients with structural heart disease, with good overall outcomes and a low rate of complications. The most important advantage of the technique is avoidance from VT induction. This is particularly true in the patients with ischemic cardiomyopathy. Thus, addition of further ablation strategies in non-ischemic patients, such as elucidation of focal tachyarrhythmias and epicardial substrates, may be very important in improving the outcomes in these patients.

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