Ultrarapid identification of activation channels within the scar using high-density mapping from a basket catheter

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Introduction

Most patients presenting for catheter ablation of scar-related ventricular tachycardia (VT) have unstable arrhythmias that prevent an accurate definition of the critical areas of the reentrant circuit with activation or entrainment mapping. In these patients, a detailed definition of the intracardiac activation sequence or analysis of response to entrainment mapping is not possible, most often because of rapid hemodynamic decompensation that requires immediate VT termination but also because of spontaneous or pacing-induced termination or transition to different morphologies. In this article, we describe the benefit of multielectrode high-density mapping to define late potential activation “channels” within a large infarct-related scar.

Image description

A 80-year-old male patient with severe ischemic cardiomyopathy (left ventricular [LV] ejection fraction 25%), large inferior myocardial infarction, and complete heart block with biventricular implantable cardioverter defibrillator (ICD) presented for catheter ablation of recurrent VT that led to syncope and multiple ICD shocks despite treatment with amiodarone and mexiletine. During the procedure, 5 distinct VT morphologies were induced (Supplemental Figure 1). None of the VTs could be mapped, because of changing morphology with attempts at entrainment (VT1 to VT2) and rapid hemodynamic deterioration (VT3 to VT5). Substrate mapping was performed during right ventricular (RV) apical pacing (QRS duration 250 ms—15,168 points displayed in the map within 2 mm of the shell, scar-mapping time 40 minutes, total LV time 72 minutes) using a high-density Orion basket catheter (Boston Scientific, Marlborough, MA); contact between tissue and catheter was validated with intracardiac echocardiography. Substrate mapping (Supplemental Material) showed a large inferior and lateral scar extending from the LV base to the apex, with a high density of late potentials (scar size 120 cm², corresponding to 60% of total LV surface area; Fig. 1) and matched regions of akinesia noted on an intracardiac echocardiogram and segments with fixed perfusion defects shown in the results of a preprocedural single-photon emission computed tomography scan. To limit the number of ablation lesions necessary to target the large abnormal substrate harboring late potentials, a “scar dechanneling” approach was attempted by identifying the conductive channels within the abnormal substrate, as previously described by Berruezo et al.¹ Ultrarapid activation mapping of conductive channels within the abnormal substrate was performed by manually adjusting the activation time reference to the end of the surface QRS complex (Supplemental Material). Activation mapping revealed 3 focal entrance sites at the apical aspect of the large inferior scar, with a discrete line of block and slow conduction turnaround elbow at the most lateral aspect of the abnormal substrate (Figure 1). Because activation mapping was performed only with RV apical pacing at a fixed rate, we could not distinguish a functional versus fixed line of activation block. Focal ablation at the identified “entrance sites” within the scar (proceeding from the periphery of the scar to more central regions) resulted in complete disappearance of abnormal and late electrograms (including the perimital region), as validated by scar remapping (5282 points, mapping time 18 minutes, gray color represents any electrogram with peak-to-peak voltage of <0.03 mV; Supplemental Figure 2). At the end of the procedure, programmed stimulation was not repeated, because of labile hemodynamic status and fluid overload. After 3 days post procedure, a repeat noninvasive programmed stimulation (from the ICD lead) was performed, and results confirmed the elimination of the 5 VTs induced at the time of the procedure. With triple extrastimulation,
we induced a further VT, for which no further ablation attempt was made.

**KEY TEACHING POINTS**

- Most patients with scar-related ventricular tachycardia present with unstable arrhythmias that are not amenable to interrogation from multiple sites to define the ventricular tachycardia circuit based on the intracardiac activation sequence and the response to entrainment mapping.

- Different substrate-based ablation strategies have been described, with the aim of targeting the abnormal substrate defined with mapping in sinus or paced rhythm.

- The “scar dechanneling” technique targets the entrance site(s) of activation channels of late potentials within the scar, and it has been shown effective to achieve the elimination of late potentials with potentially limited ablation.

- Ultrahigh-density mapping via a multipolar basket catheter with small electrodes and tight interelectrode distance may improve the detection of discrete activation patterns within the scar, possibly improving the efficacy of the scar dechanneling ablation technique.
Acknowledgments
The authors would like to thank Lauren Walsh, BSME, and George J. Winton, BS, for their technical expertise.

Appendix
Supplementary data
Supplementary data are available in the online version of this article at http://dx.doi.org/10.1016/j.hrcr.2016.06.009

Reference
1. Berruezo A, Fernandez-Armenta J, Andreu D, et al. Scar dechanneling: new method for scar-related left ventricular tachycardia substrate ablation. Circ Arrhythm Electrophysiol 2015;8(2):326–336.