Ventricular Tachycardia Ablation in a Patient With Arrhythmogenic Right Ventricular Cardiomyopathy

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ABSTRACT

Tachycardias in the setting of arrhythmogenic right ventricular cardiomyopathy (ARVC) are inducible with programmed stimulation and can be entrained. Most sites of origin of ventricular tachycardia (VT) cluster within the low-voltage peritricuspid and/or peripulmonic regions, usually within 2–3 cm of the valve’s orifice. In this case, unstable VTs were induced by ventricular extrastimulus, and substrate mapping and pace-mapping were used to delineate the region of low-voltage electrogram (EGM). It was also used to identify putative components of the reentry circuits at the anterior right ventricular (RV) free-wall. With pace-mapping, we were able to identify sites with good pace-maps around the tricuspid valve (TV) annulus. Radiofrequency application was delivered around these sites. Eventually, the VTs could no longer be induced by any pacing maneuvers.

Key words: pace-mapping tachycardia

Introduction

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an inherited myocardial disease characterized by replacement of the myocardium by fibrous and fatty tissue that predisposes one to ventricular arrhythmias and sudden cardiac death. Ventricular tachycardia (VT), occurring in up to 64% of ARVC patients usually originates in the right ventricle (RV) and exhibits left bundle branch block morphology.1 Reentry is the predominant mechanism, as suggested by the fact that tachycardias can be initiated by programmed stimulation and that they can be entrained in patients with stable VT. In this case, substrate mapping and pace-mapping were used to identify reentry circuits because the patient’s vital signs were unstable during clinical VT.2

Case reports

We report the case of a 65-year-old man with a history of implantable cardioverter–defibrillator...
implantation (ICD) due to several episodes of sustained VT with syncope that required termination by cardioversion. Two-dimensional echocardiography and ventriculography revealed diffuse and severe RV enlargement, slightly reduced RV systolic function, and normal left ventricular dimension and function. Cardiac magnetic resonance imaging (MRI) showed a dilated RV with mild RV dysfunction and DE of the anterior RV free wall. These findings were consistent with a diagnosis of ARVC based on published criteria. The patient did well on sotalol with good functional status. However, he began to have palpitations with a syncopal history. Recurrent VTs were documented, which were terminated by pacing several times and by shock on one occasion. The clinical VT showed the same morphology: left bundle left-inferior (LBLI) axis, V5 transition, and a 330 msec cycle length with presyncopal attack during VT (Figure 1). The baseline heart rhythm was sinus rhythm. During sinus rhythm, the 12-lead ECG exhibited a localized prolongation (110 msec) of the QRS complex and inverted T waves with epsilon waves in leads V1 and V2.

First, electroanatomic mapping of the RV was performed using a 7-French, 4-mm tip ablation catheter (CARTO® 3, Biosense Webster, Inc., Diamond Bar, USA) during sinus rhythm in order to identify the VT substrate on the voltage map. The voltage map revealed a low voltage area (<1.5 mV) in the free wall of the RV outflow tract, pericruspid area, and apex. Next, a clinical sustained VT1 with a left bundle branch block and superior axis QRS morphology (cycle length = 340 msec) was induced by double extrastimuli from the RV outflow tract. During VT1, vital signs were unstable, and therefore, endocardial bipolar voltage mapping was performed during sinus rhythm. Endocardial voltage mapping in the RV revealed anterolateral wall scarring near the tricuspid annulus (TA) (Figure 2). A good pace-map was identified near the annulus at the superior border of the scar. Slightly lower down, there was a long stimulus-to-QRS with the same good pace-map morphology (Figure 3). There were early signals at the distal tip of the

Figure 1. Surface ECG of clinically sustained VT1.
Figure 2. Endocardial bipolar voltage mapping in RV.

Figure 3. Good RV pace-map site with long stimulus-to-QRS.
ablation catheter, over 100 msec, during clinical premature ventricular contraction (PVC) at the site of previous pace-mapping (Figure 4A). The activation map, at this earliest site, revealed isolated potentials, which were separated from the ventricular EGM by an isoelectric line (Figure 4B). During the clinical VT1 induction study, a non-clinical, non-sustained VT2 (cycle length = 340
msec) was induced by 600 msec triple extrastimuli with a 250 msec cycle length (Figure 5). The excellent pace-map for VT2 was slightly lower down on the annulus than that of VT1. Our ablation strategy was to make several lines between the scar borders to the TA annulus linearly with a transverse line from the lateral scar border to the anterior (Figure 6).

Radiofrequency (RF) applications of 60 s each with a target temperature of 50°C and maximum power output of 50 W were delivered around these sites. After RF ablation, 400 triple extrastimuli on 3 μg/kg/min isoproterenol only obtained the non-sustained VT2 whereas the clinical VT1 was non-inducible. Excellent pace-maps for VT2 were identified on the annulus slightly lower down from the site of the previous perfect pace-map of clinical VT1. Several RF ablations were delivered

Figure 6. Ablation strategy of making lines between scar borders to the TA annulus linearly, with a transverse line from the lateral scar border to the anterior.
around these good pace–map sites. Eventually, the VTs could no longer be induced by any pacing maneuver. No additional epicardial procedure was performed since both the clinical and non-clinical VTs were not inducible after the successful endocardial RF ablations.

**Discussion**

Tachycardias in the setting of ARVC are inducible with programmed stimulation and can be entrained. Reentry involving regions of abnormal EGMs is the most likely mechanism.

Therefore, the same mapping principles discussed for idiopathic dilated cardiomyopathy (IDCM) can be applied in patients with ARVC.

Most VT sites of origin cluster within the low-voltage peritricuspid and/or peripulmonic region, usually within 2~3 cm of the valve’s orifice. In patients with larger scars, the VT can exit toward the apical extent of the scar, but still within the region of abnormal EGM voltage. Therefore, these are the regions initially targeted by activation, entrainment, and pace–mapping. Activation and entrainment mapping can be used in hemodynamically stable VT.

Presystolic activity at the earliest activated site usually precedes the QRS by at least 30~50 msec. Its participation in the reentry circuit should be confirmed by entrainment. In patients with noninducible or untolerated VTs, substrate mapping and pace–mapping can be used to delineate the region of low–voltage electrogram and then to identify putative components of the reentry circuits. The acute success rate of ablation ranges between 50% and 90% in different studies. The variable reported outcomes can be attributed to differences in mapping techniques, endpoints, and operator experience. Overall, substrate–based approaches are associated with better acute and long–term success rates, a finding that has been attributed to the patchy distribution of the scar, harboring multiple regions of slow conduction.

In cases where there are more extensive epicardial than endocardial substrates, a more aggressive ablation approach targeting both the epicardium and endocardium, is often required. This study clearly indicates that combined endocardial and epicardial ablation is associated with better long–term results in terms of freedom from arrhythmia recurrence.

**References**