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Utility of Ripple Mapping for Identification of Slow Conduction Channels During Ventricular Tachycardia Ablation in the Setting of Arrhythmogenic Right Ventricular Cardiomyopathy

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Abstract

BACKGROUND—Ripple mapping displays every deflection of a bipolar electrogram and enables the visualization of conduction channels (RMCC) within post-infarction ventricular scar to guide ventricular tachycardia (VT) ablation. The utility of RMCC identification for facilitation of VT ablation in the setting of arrhythmogenic right ventricular cardiomyopathy (ARVC) has not been described.

OBJECTIVE—We sought to a) identify the slow conduction channels in the endocardial/epicardial scar by ripple mapping, and b) retrospectively analyze whether elimination of RMCC is associated with improved VT free survival, in ARVC patients.

METHODS—High-density right ventricular endocardial and epicardial electrograms were collected using CARTO 3 system in sinus rhythm or ventricular pacing and reviewed for RMCC. Low voltage zones and abnormal myocardium in the epicardium were identified by using standardized late gadolinium enhanced (LGE)-MRI SI z-scores.

RESULTS—A cohort of 20 ARVC patients that had undergone simultaneous high-density right ventricular endocardial and epicardial electrogram mapping was identified (age 44±13 years). Epicardial scar defined as bipolar voltage <1.0 mV occupied 47.6% (IQR, 30.9–63.7) of the total epicardial surface area, and was larger than endocardial scar defined as bipolar voltage <1.5 mV, which occupied 11.2% (IQR, 4.2±17.8) of the endocardium ($P<0.01$). A median 1.5 RMCC, defined as continuous corridors of sequential late activation within scar were identified per patient

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(IQR, 1–3), most of which were epicardial. The median ratio of RMCC ablated was 1 (IQR 0.6–1). During a median follow-up of 44 months (IQR 11–49), the ratio of RMCC ablated was associated with freedom from recurrent VT (HR 0.01, P=0.049). Among 9 patients with adequate MRI, 73% of RMCCs were localized in LGE regions, 24% were adjacent to an area with LGE, and 3% were in regions without LGE.

CONCLUSION—Slow conduction channels within endocardial or epicardial ARVC scar were delineated clearly by ripple mapping and corresponded to critical isthmus sites during entrainment. Complete elimination of RMCC was associated with freedom from VT.

Keywords

Arrhythmogenic right ventricular cardiomyopathy; Ventricular tachycardia; Late gadolinium delayed enhancement; Magnetic resonance imaging; Ripple mapping; Catheter ablation

Introduction

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) is a cardiomyopathy characterized by fibro-fatty replacement of myocardium that primarily involves the right ventricle (RV).^{1, 2} Ripple mapping (RM) is a novel visualization method, which displays each electrogram component as a dynamic bar moving out from the surface relative to a fiduciary time point on a 3-dimensional (3D) map surface.³ The RM methodology has been demonstrated to be effective for identification of conduction channels and for functional substrate ablation within post-infarction scar.^{4, 5} In this study, we sought to a) study the utility of RM for visualization of continuous corridors of sequential late activation within scar, termed conduction channels (RMCC), b) define the association between complete RMCC ablation and VT recurrence, and c) examine the association of RMCCs with image characteristics on late gadolinium enhancement (LGE) MRI in a cohort of patients with ARVC.

Methods

Patient Population

The study protocol was reviewed and approved by the Institutional Review Board of the Hospital of the University of Pennsylvania. Patients with a diagnosis of ARVC that had undergone VT ablation with simultaneous high-density endocardial and epicardial electroanatomic mapping of the RV using the CARTO 3 system (Biosense Webster Inc. Diamond Bar, CA) during the period from 04/2010 to 12/2016 at our institution composed the retrospective study cohort.

Electrophysiological Study

Access to the RV endocardium via the femoral vein and to the epicardium via a subxiphoid approach was obtained as previously described.^{6, 7} Electrograms were sampled using a 4-mm standard tip, 3.5-mm open irrigated tip (Thermacool, Biosense Webster, Diamond Bar) or PentaRay NAV (Biosense Webster, Inc) catheter and displayed on an electroanatomic mapping system (CARTO, Biosense Webster, Inc). Bipolar signals were filtered at 30 to 300

Hz and displayed at 100-mm/s speed. Bipolar voltage amplitude measurements 1.5 mV (in endocardium) or 1.0 mV (in epicardium) were categorized as normal, whereas those <0.5 mV were defined as dense scar. For ripple mapping, bipolar signals were collected during sinus rhythm or ventricular pacing, using a window of interest to include the surface QRS and the diastolic period to the T wave. The peak-to-peak signal amplitude of the bipolar or late potential (LP) electrogram was measured automatically and confirmed during manual review. The bars were clipped at >1.5 mV to allow low-voltage bars to be seen without being obscured by large bars from areas of healthy tissue. The height of each Ripple bar was proportional to the voltage amplitude of the electrogram signal at that time point.

VTs were induced by programmed ventricular stimulation with up to triple extrastimuli from 2 ventricular sites and 2 drive cycle lengths. Isthmus sites for VT were defined as previously described,⁷ and using the following criteria: entrainment with concealed fusion, post pacing interval 30 ms, stimulus to QRS > 30% and <70% VT cycle length, and a stimulus to QRS and electrogram to QRS interval within 30 ms. Ablation points on the VT isthmus and the site of VT termination within 10–15s of RF application were tagged. Acute success was defined as non-inducibility of VT at the end of the ablation procedure with the described stimulation protocol and at the time of repeat programmed stimulation typically with the implanted defibrillator before hospital discharge.⁸

Analysis of Ripple Maps and Electroanatomic Maps

Ripple map conduction channels (RMCC) were identified as previously described.⁴ Briefly, multiple electrograms within the scar are reviewed by CARTO Ripple mapping viewer: the initial wave front is presumed to represent far field activation, and the delayed wave front is presumed to represent local slow activation. RMCCs were defined as channels with adjacent sequential bars representing local linear activation moving through scar. Design lines were drawn on the CARTO mapping shell to encircle the boundaries of the local activation and highlight the pathway. A minimum of 3 bars (3 consistent electrograms) was necessary to identify late propagation in a channel. Abnormal electrograms included LPs (discrete late electrograms in sinus rhythm/RV pacing in the low-voltage region) and fractionated potentials (low amplitude, high frequency, multicomponent signals of prolonged duration). Ablation lesion locations were registered onto the maps to examine the relationship between ablation lesions and identified RMCCs. The relationship between the location of entrainment data and VT termination during ablation, where available, was compared with RMCC locations. All RMCCs were identified retrospectively upon post-procedure analysis of the CARTO maps.

Cardiovascular Magnetic Resonance Image Acquisition and Analysis

A subgroup of 13 of 20 patients had undergone electrocardiogram (ECG)-triggered cardiac magnetic resonance (CMR) using a 1.5 T scanner (Sonata or Avanto, Siemens, Erlangen, Germany) and previously described standardized imaging protocol for the RV.⁷ Of these 13 patients, 9 had images with adequate visualization of RV, spared from ICD artifact, to enable image analysis. The LGE images were acquired after intravenous injection of 0.1 mmol/kg gadolinium-DTPA gadolinium-DTPA (Schering, Berlin, Germany) using a short-axis multi-slice 3D segmented inversion-recovery gradient-echo pulse sequence with effective slice

thickness 5 mm. A 3D RV reconstruction was obtained by using Qmass (Version 7.2, Leiden University Medical Center, Leiden, Netherlands). RV endocardial and epicardial contours were manually defined on contiguous short-axis slices. Based on previously described methodology,⁹ sampled points on the EAM were co-registered to the LGE image based on the procedural registration coordinates of EAM merge with the RV CMR image planes. RV LGE was defined using signal intensity z-score thresholds of >0.05 and <-0.16 corresponding to bipolar voltage measures <0.5 and >1.0 mV, respectively.¹⁰

Follow-Up

Patients were followed every 6 months or sooner if they experienced device therapy. All device therapy data were logged. When longitudinal office follow-up visits were not performed after the 1-year visit, telephone interviews were performed with patients, referring physicians and family members at 6- and 12-month interval to confirm the absence of arrhythmias with review of stored device electrogram information whenever reported therapy was indicated.

Statistical Analysis

Continuous variables are expressed as mean $\pm$ standard deviation (SD) for normally distributed data, or median and interquartile range for non-normal data. Categorical variables are expressed as percentages. Comparisons were made using the Wilcoxon signed-rank test. The association of ratio of RMCC ablated with time to VT recurrence was examined using a Cox proportional hazard model. The proportional-hazards assumption was tested using Schoenfeld residuals. A 2-sided *P* value <0.05 was considered significant. Statistical analyses were performed by using Stata (version 12, StataCorp, College Station, TX).

Results

Patient Characteristics

The study group included 20 patients (age 44 ± 14 , 13 males) who fulfilled the diagnosis of definite ARVC based on the 2010 Revised Task Force Criteria (Table 1) and had undergone RV endocardial and epicardial mapping during the same VT ablation procedure. Of these patients, 17 (85%) experienced palpitations, 7 (35%) had experienced pre-syncopal episodes, 1 (5%) had experienced syncope, and 1 (5%) had experienced cardiac arrest. Of all patients, 9 underwent genetic testing, 5 of whom (25%) had a pathogenic mutation identified. Another 7 (35%) had a direct relative with genetically diagnosed ARVC-associated mutations. All patients received an ICD, 17 of which were implanted before VT ablation.

Electrogram mapping

Electrogram mapping was performed during sinus rhythm in 18 patients and during ventricular pacing in the remaining 2. Right ventricular endocardial and epicardial maps were available in all 20 patients. Endocardial RMCC localization was precluded in 2 of 18 patients due to lack of identifiable endocardial delayed potentials. Median map density in the endocardium was 277 points (IQR, 214–352) with a scar area of 11.2% (IQR, 4.2–17.8) by bipolar voltage criteria <1.5 mV (Table 2). Median map density in the epicardium was 320

points (IQR, 210–426) with a scar area of 47.6% (IQR, 30.9–63.7) by bipolar voltage criteria 1.0 mV (Table 2). Due to inclusion of the RV septum on endocardial mapping, RV endocardial surface area 179.3 cm² (IQR, 152.45–220.4 cm²) was larger than the RV epicardial surface area of 136.2 cm² (114.1–158.2 cm²). However, the epicardial scar area of 59.8 cm² (IQR, 46.9–91 cm²) and the percent of epicardial scar 47.6% (IQR, 30.9–63.7%) to the complete surface area were significantly larger on the epicardium compared to the endocardial scar area of 20.3 cm² (9.85–30.05 cm²) and the percentage of endocardial scar of 11.2% (4.2–17.8%), respectively.

RM Analysis to Identify RMCC in Endocardial/Epicardial mapping

RMCCs were identified in 33.3% (6/18) of RV endocardial maps and 100% (20/20) of RV epicardial maps of all patients with ARVC. Figure 1 and Figure 2 illustrate the RMCCs identified in different patients (extracted from data shown in Movie I and Movie II of the data Supplement). Location of RMCCs were grouped into distinct anatomic segments based on a previously described model.^{11, 12}

In the endocardium, most RMCCs were distributed near the inferior tricuspid annulus of RV free wall (66.7%); however, the most common RMCC location in the epicardium was in the free wall of RVOT (82.4%). Compared with endocardial mapping, there were more RMCCs [2 (IQR, 1–3)] and a larger total area of RMCCs [8.3 cm²(IQR, 7.3–15.0)] on epicardial mapping (Table 3).

Comparison of EGM characteristics of RMCC with LGE-MRI

Among the subset of 9 patients with LGE-MRI, 73% of RMCCs were localized in RV LGE regions, 24% were adjacent to an area with LGE, and 3% were seen in regions without LGE. Figure 1H illustrates an example of an RMCC co-localization to an area with RV LGE.

Association of RMCC Elimination with Outcome

Mapping of VT circuits was performed using entrainment or pace mapping as described in the methods section. RMCC locations were not specifically sought or prospectively targeted. Acute non-inducibility was achieved in all but 2 patients in whom ablation was discontinued due to hemodynamic intolerance of anesthesia or volume overload. Ablations were delivered to a total of 33 RMCCs (median 1.5 per patient, IQR 1–3). The median ratio of RMCC ablated per patient was 1 (IQR 0.6–1). Figure 1I shows an example of ablation lesions, which covered all retrospectively identified RMCCs. In contrast, Figure 2C shows examples of retrospectively identified RMCC remnants following ablation. Isochronal late activation maps were also constructed to display ventricular activation during sinus rhythm over 8 isochrones¹³. The latest activation ripple is consistent with the latest isochrones of activation in these patients (Fig. 1D–F and Fig. 2D).

Forty-two distinct VT morphologies were identified in 20 patients; however, only 16.7% (7/42) were hemodynamically stable and allowed entrainment maneuvers. The VT circuit sites with concealed fusion demonstrated with entrainment mapping, collocated to RMCCs in all cases (Fig. 2E). However, 71.4% (5/7) of successful VT termination sites were not localized to the latest activation ripple, but were adjacent to an RMCC (Fig. 1I and Fig.

2C/D), representing the slow conduction “entrance” into the latest ripple area (Movie I and Movie II).

During a median follow-up of 44 (IQR 11–49) months, 14 patients (70%) remained free of any VT recurrence following the first procedure. The median time to VT recurrence was 58 months (IQR 48–∞, incidence rate 0.009/month). Two patients required an additional procedure and remained VT free following the second procedure. The remaining 4 patients did not undergo a repeat procedure because previously ineffective antiarrhythmic drug therapy led to VT quiescence following the first ablation. In the Cox proportional hazard model, the ratio of RMCC ablated was associated with freedom from recurrent VT after a single procedure (HR 0.01, $P=0.049$).

Discussion

To the best of our knowledge, this is the first study to examine the utility of ripple mapping for VT ablation in the setting of ARVC. The major findings of the present study are that RMCCs are associated with critical isthmus sites during entrainment and able to delineate channel activation clearly. Complete ablation of continuous corridors of sequential late activation, identified as RMCC, may improve freedom from VT following ablation in the setting of ARVC. Additionally, in the subset of patients with adequate MRI, the majority of RMCC were localized in or adjacent to RV regions that displayed LGE.

Ripple mapping preserves all components of the electrogram, optimally displays delayed low-amplitude local activation within scar, and has been used to identify slow conduction channels within post-infarct scar during catheter ablation.^{4, 5} RMCCs streamline the process to understand the spatial and sequential activation of delayed potentials through scar and provide a representation of what experienced electrophysiologists have always performed in their mind when using conventional mapping. Like post-infarct scar, the fibro-fatty myocardial replacement in ARVC leads to extensive networks of delayed and fractioned potentials in both the endocardial and epicardial RV.^{14, 15} In our study, ripple mapping easily detected such abnormal potentials as RMCC in patients with ARVC. Autopsy studies in patients with ARVC have shown that fibro-fatty dysplasia progresses from the epicardium toward the endocardium and predominantly involves the RV free wall.^{1, 16} Consistent with prior observations, most RMCCs in our study were distributed in the RV free wall. Compared to the endocardium, more RMCCs were noted in the epicardium and the total area of RMCC was larger in the epicardium.

In the setting of post-infarct VT, sites of entrainment with concealed fusion have been associated with RMCCs.⁴ Similarly, our study showed a strong association between VT circuit sites and RMCCs. Interestingly, the latest activation ripple in sinus rhythm/ventricular pacing did not correspond to VT termination sites. These results are consistent with the previous understanding that central channel sites rather than the entrance or exit (corresponding to the latest isochrones of RMCC) comprise optimal ablation targets.^{13, 17–20}

Current techniques using endocardial and epicardial VT substrate ablation in ARVC provide 71% long-term VT-free survival.²¹ In the current study, which evaluated RMCC ablation

retrospectively, 90% of those with complete RMCC ablation remained VT free during long-term follow-up. Future prospective studies are necessary to refine these retrospective results. However, complete ablation of RMCC should reasonably be pursued as the endpoint in ARVC related VT ablation. Additionally, in patients with hemodynamically unstable VT, elimination of all RMCCs observed during sinus rhythm, may provide an alternative to other substrate ablation methodologies.

RMCC and LGE-MRI in the Setting of ARVC

We have previously demonstrated an association between increased RV gadolinium uptake in the setting of ARVC and lower epicardial bipolar and unipolar electrogram voltage amplitude. In that study, we also determined appropriate standardized signal intensity thresholds for identification of abnormal RV tissues. In the current study, 73% of RMCCs were localized in regions with LGE and an additional 25% were adjacent to an LGE region as defined by our criteria. Thus, fine mapping of RMCC can primarily be focused upon areas with or adjacent to RV LGE on CMR.

Limitations

This is a retrospective study and may thus be prone to inadvertent biases regarding patient selection, and unmeasured temporal effects due to changes in ablation methodology. Further prospective study is required to demonstrate whether the RMCC elimination strategy improves VT ablation outcome. The majority of patients in this study underwent point by point mapping; therefore, point density is relatively low compared to studies that use multipolar mapping catheters. On the other hand, we believe that catheter contact, signal quality, and ectopic beat rejection are substantially improved with point by point mapping. Due to the low prevalence of ARVC, the sample size is relatively small; therefore, multivariable analysis to examine confounding by other variables could not be performed. Some VT episodes demonstrated poor hemodynamic parameters and did not allow entrainment to fully characterize the circuit. Finally, CMR images were often affected by ICD susceptibility artifacts; therefore, not all images were utilized for RMCC and image comparisons.

Conclusion

RMCCs within endocardial or epicardial scar were associated with critical isthmus sites during entrainment and able to delineate channel activation clearly in the setting of ARVC. In this retrospective assessment, complete elimination of RMCC was associated with improved VT free survival.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1 (Movie I).

Analysis of Ripple mapping with epicardial scar to guide VT ablation in the different setting. The mapping was collected in sinus rhythm. Design lines have outlined the path of RMCC activation. A-C, Ripple bar were seen to propagate from the apex to basal in the voltage map. Scar (<1.0 mv) was identified on the free wall of RV epicardium. A cluster of late activating bars from +50 ms to +90 ms post QRS onset is shown within basal scar and corresponds to late potentials traveling from apex to basal. When the activation propagates in to scar(“entrance”), the activation conducts in two different directions. D-F, Ripple bar

were shown in isochronal late activation map. The purple is the latest activate region, which is consistent with the latest ripple activation. G, The “channel” is exposed in the scar by adjust the upper and lower voltage threshold cutoff. In the RMCCs area, the purple region is exposed as channels in the voltage setting from 0.15–0.30 mV. H, The “channel” was exposed by CMR by using the criteria of SI z-score thresholds of >0.05 and <-0.16 corresponded to bipolar voltage measures <0.5 and >1.0 mV, respectively. Red region is the “dense scar”, blue region is the “health issue”, the green is the border of “dense scar”. RMCCs are located in the green region. I, Ablation overlapping in all RMCCs. In this patient, all RMMCs overlap areas with all of RMCCs. Blue point is the located near the propagation “entrance” of RMCCs, which can be recorded the mid diastolic potential. VT was terminated in the blue point after ablation for about 1 s. There has been no recurrence of VT during a follow-up period about 53 months.

Figure 2 (Movie II).

Ripple mapping conduction channel (RMCC) in another patient. The map was collected in sinus rhythm and the movie could show the direction of ripple activation, Design lines have outlined the path of RMCC activation. Scar ($<1.0\text{mV}$) was identified on the epicardium of ARVC/D patient. A, the terminal end of the first wavefront of Ripple bars (white and yellow arrows) is seen at +55ms post QRS onset. B, the terminal end of the first wavefront of Ripple bars (white and yellow arrows) is seen at +92ms post QRS onset. C and D show the ablation site in voltage map and in isochronal late activation maps separately. Ablation lesions are shown around the terminate site (blue point) and circumferentially outside the latest isochrones of activation. E, Entrainment at the terminate site (blue point). VT was successfully entrained (PPI-TCL=13 ms) and terminated outside RMCC and the latest

isochrones (the purple), however, there has been recurrence of VT during a follow-up period about 58 months.

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Table 1.

Baseline Characteristics of the ARVC Study Cohort

Viable	n=20
Age (y)	44±14
Male	13(65%)
Clinical symptom	
Palpitations	17 (85%)
Pre-syncope	7 (35%)
Syncope	1 (5%)
Cardiac arrest	1 (5%)
LVEF	59.1±9.2%
Task force criteria (%)	
Structural abnormalities *	13 (65%)/2 (10%)
Depolarization changes *	5 (25%)/15 (75%)
Repolarization changes *	13 (65%)/7 (35%)
Ventricular arrhythmias *	12 (60%)/8 (40%)
Family history *	7(35%)/10(50%)
ICD	20(100%)
Medication at discharge	
Drug free or β -blocker only	12 (60%)
Class I or III	8 (40%)

* According to the 2010 Revised Task Force criteria (Major/minor)

Table 2.

Electrogram mapping and ablation statistics

	Endocardial Mapping (n=18, median, IQR)	Epicardial Mapping (n=20, median, IQR)	<i>P</i>
Map density(n)	277 (214–352)	320 (210–426)	0.69
Surface area(cm2)	179.3 (152.45–220.4)	136.2 (114.1–158.2)	0.001
Scar area (cm2)	20.3 (9.85–30.05)	59.8(46.9–91)	<0.001
Ratio of scar area to whole surface area (%)	11.2 (4.2–17.8)	47.6(30.9–63.7)	<0.001
Map scar point density (cm-1)	2.7 (2.2–5.1)	3.3 (2.5–4.7)	0.52

Table 3.

RMCC Characteristics in RV endocardium and epicardium

	Endocardial Mapping(n=18)	Epicardial Mapping(n=20)	P
Total Patients of RMCC	6 (33.3%)	20 (100%)	<0.001
Number of RMCC	0 (IQR, 0–1)	2 (IQR, 1–3)	<0.001
Location of RMCC			
RVOT	2 (33.3%)	14 (82.4%)	
TVA Sup	3 (50%)	8 (42.1%)	
Basal Sup	0	5 (26.3%)	
TVA Inf	4 (66.7%)	8 (42.1%)	
Basal Inf	0	2 (10.5%)	
Area of RMCC (cm ²)	0 (IQR, 0–2.65)	8.3 (IQR, 7.3–15.0)	<0.001

RVOT = right ventricular outflow tract; TVA = tricuspid valve annulus; Sup=Superior; Inf= Inferior