Sinus rhythm detection of conducting channels and ventricular tachycardia isthmus in arrhythmogenic right ventricular cardiomyopathy

Juan Fernández-Armenta, MD,[*†] David Andreu, Msc, PhD,[*†] Diego Penela, MD,[*†] Emilce Trucco, MD,[*†] Laura Cipolletta, MD,[*†] Elena Arbelo, MD, PhD,[*†] Paola Berne, MD,[*†] Jose María Tolosana, MD,[*†] Alonso Pedrote, MD, PhD,[‡] Josep Brugada, MD, PhD,[*†] Lluis Mont, MD, PhD,[*†] Antonio Berruezo, MD, PhD[*†]

From the [*]Arrhythmia Section, Cardiology Department, Thorax Institute, Hospital Clínic, Universitat de Barcelona, Barcelona, Spain, [†]IDIBAPS (Institut d'Investigació Agustí Pi i Sunyer), Barcelona, Spain, and [‡]Arrhythmia Unit, Cardiology Department, Hospital Universitario Virgen del Rocío, Seville, Spain.

BACKGROUND The identification of conducting channels (CCs) based on its relative high voltage or the presence of electrograms with delayed components has been proposed for substrate-guided scar-related ventricular tachycardia (VT) ablation. The relationship of these channels with the VT isthmuses remains unclear.

OBJECTIVE To assess the link between CCs identified during sinus rhythm (SR) and VT isthmuses in patients with arrhythmogenic right ventricular cardiomyopathy (ARVC).

METHODS Twenty-two consecutive patients with ARVC undergoing substrate-guided VT ablation (scar dechanneling technique) were analyzed. High-density endocardial and epicardial electroanatomic maps were obtained during SR. Standard bipolar cutoff values (0.5–1.5 and $<$0.5 mV) were used to define border zone and dense scar. The CCs were identified by voltage threshold adjustment (voltage channels) or by tagging the electrograms with delayed components that are sequentially activated (late potential channels).

RESULTS A total of 87 CCs were identified; 65 (74.7%) of them on the epicardial surface. Twenty-four (27.6%) CCs were voltage channels, and compared with late potential CCs, these had a higher bipolar voltage (0.96 [0.48–1.29] mV vs 0.39 [0.26–0.50] mV; $P < .001$] and required more radiofrequency applications (5 [4–7] vs 3 [2–5]; $P = .048$]. Eighteen (90%) of 20 identified VT isthmuses were located on the epicardium. Only 8 (40%) VT isthmuses were related to a voltage CC. The remaining 12 (60%) VT isthmuses were linked to a late potential CC.

CONCLUSION Late potential CCs more frequently act as the VT substrate in ARVC and therefore should also be considered to guide SR substrate-guided ablation.

KEYWORDS Arrhythmogenic right ventricular cardiomyopathy; Ventricular tachycardia; Radiofrequency ablation; Epicardial ablation; Substrate-guided ablation; Conducting channels

ABBREVIATIONS **ARVC** = arrhythmogenic right ventricular cardiomyopathy; **CC** = conducting channel; **EAM** = electroanatomic map; **E-DC** = electrogram with delayed component; **RF** = radiofrequency; **RV** = right ventricle/ventricular; **SR** = sinus rhythm; **VT** = ventricular tachycardia

(Heart Rhythm 2014;11:747–754)

Introduction

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an inherited myocardial disease characterized by a fibrofatty replacement of the right ventricle (RV). A more extensive epicardial arrhythmogenic substrate has been found in histological and small clinical studies.[1,2] This anatomic profile can mostly explain the low acute success rate and the high rate of recurrences after conventional endocardial ventricular tachycardia (VT) ablation.[3,4] Some studies have shown that a combined endo-epicardial substrate-guided ablation in patients with ARVC could improve the procedure results and mid-term outcomes.[2,5,6]

In contrast, multiple targets have been proposed for ablation during substrate mapping.[7] The use of bipolar voltage modification of the upper and lower thresholds to identify zones with higher voltage inside the scar is one of the methods described to identify the conducting channels (CCs) that can promote reentrant tachycardia. These CCs have been proposed as the target for ablation.[8,9] The relationship of the CC identified by this method with the VT isthmus has recently been questioned.[10] In contrast, CC

This study was supported in part by a grant (PI11/02049) from Instituto de Salud Carlos III, Ministry of Science and Innovation, Madrid, Spain, and from Fondo Europeo de Desarrollo Regional, European Union. Dr Andreu is an employee of Biosense Webster. **Address reprint requests and correspondence:** Dr Antonio Berruezo, Arrhythmia Section, Cardiology Department, Thorax Institute, Hospital Clinic, C/ Villarroel 170, 08036 Barcelona, Spain. E-mail address: berruezo@clinic.ub.es.

1547-5271/$-see front matter http://dx.doi.org/10.1016/j.hrthm.2014.02.016

identification based on electrogram analysis, that is, analyzing the activation sequence of the delayed local component of complex electrograms, has also been proposed.[5,11]

We aimed to assess the presence and percentage of CCs identified by means of voltage adjustment or electrogram analysis during sinus rhythm (SR) and the link between them and the VT isthmuses in patients with ARVC.

Methods

Patient population

Twenty-two consecutive patients with a diagnosis of ARVC who underwent VT ablation in our hospital between May 2008 and April 2013 were included in the study. High-density endocardial and epicardial electroanatomic maps (EAMs) were obtained from all patients and were analyzed. All patients provided written informed consent to participate in the study.

Electrophysiological procedure

A tetrapolar catheter was positioned at the RV apex. A 3.5-mm externally irrigated tip ablation catheter (ThermoCool, NaviStar, Biosense Webster, Inc, Diamond Bar, CA) was used for mapping and ablation. After endocardial mapping, a percutaneous subxyphoid access was obtained in all patients for mapping and ablation. Endocardial and epicardial mapping was facilitated with a steerable sheath (Agilis, St Jude Medical, St Paul, MN). In the first 15 (68%) patients, a programmed RV stimulation with a train of 600-, 500-, and 430-ms cycle length and up to 3 extra ventricular stimuli and burst pacing was used for VT induction at the beginning of the procedure, using isoproterenol when necessary. The protocol was then revised, eliminating initial VT induction. The same protocol was repeated after substrate-based ablation using the scar dechanneling technique to test for acute results in all patients.[5]

Substrate mapping

Image merging with contrast-enhanced cardiac tomography or contrast-enhanced cardiac magnetic resonance was performed by using the CartoMerge software (Cartomerge, Biosense Webster). High-density endocardial and epicardial bipolar voltage map of the RV was obtained during SR by using the CARTO system (Biosense Webster). Standard voltage cutoff values were used for dense scar ($<$0.5 mV) and border zone (0.5–1.5 mV) identification.

Electrograms with delayed components (E-DCs) were defined as electrograms with low-voltage and high-frequency component (local component) with variable delay with respect to a distinguishable far-field ventricular component recorded during SR. In ambiguous cases, RV pacing (600-ms cycle length) was performed to confirm the local component and distinguish between far-field and near-field components. Neither isoelectric interval nor predefined minimum local electrogram delay was required. The voltage or the voltage/duration ratio between both components was also not taken into account. E-DCs were classified as being an entrance or an inner part of a given CC, depending on the local activation time of the near-field component. E-DCs with the shortest delay between the far-field component and the delayed local component were considered the CC entrance after comparison with the neighboring E-DCs belonging to the CC.

VT ablation

Clinically and nonclinically induced VTs were targeted for ablation. VT isthmus and exit sites were defined through entrainment mapping if the VT was tolerated (using the standard criteria: concealed fusion, postpacing interval measurements, and stimulus to QRS delay) and by pace mapping if not tolerated. The procedure started with radiofrequency (RF) catheter ablation of the induced VTs in patients who underwent induction protocol at baseline. In the remaining patients, substrate mapping and ablation was the first step. Inducible VTs after substrate guided ablation (residual VTs) were also mapped by conventional techniques and targeted for ablation.

RF ablation was controlled by a temperature limit of 45°C with a power limit of 40 W at the endocardium and the epicardium. The catheter irrigation rate during RF application was 26 mL/min in the endocardium and 17 mL/min in the epicardium. During mapping, the irrigation rate was decreased to 2 mL/min at the endocardium and 0 mL/min at the epicardium. Coronary arteries were localized through the integration of cardiac imaging into the navigation system. Phrenic nerve course was obtained by high-output epicardial pacing.

Substrate-guided ablation

RF energy was delivered at the previously identified CC entrances (Figure 1). For that purpose, the ablation catheter was positioned and oriented over and along the CC path whenever possible in order to check for the CC entrance block during the RF application. A change in the CC activation sequence of the delayed components, or their disappearance during RF application, indicated a CC entrance block. These changes were detected by continuously recording the local activation in the CC with the proximal dipoles of the ablation catheter (Figure 1). RF was delivered during a minimum of 30 seconds, until CC entrance block, or a maximum of 60 seconds when the CC entrance block could not be checked during RF application. The presence and activation sequence of E-DCs along the CC was always checked after RF delivery. In addition, RF was delivered at the inner part of the CC when ablation at the CC entrance could not eliminate inner E-DCs. A postablation remap was always performed to document the elimination of all the CCs and to eliminate the remaining E-DCs, if necessary. Short linear ablation lines were created in some patients to connect dense scars or a dense scar to the tricuspid annulus, if separated by a voltage CC.