Combined Endocardial and Epicardial Catheter Ablation in Arrhythmogenic Right Ventricular Dysplasia Incorporating Scar Dechanneling Technique

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Background—Ventricular tachycardia (VT) ablation in patients with arrhythmogenic right ventricular dysplasia/cardio-myopathy (ARVD/C) has a low success rate. A more extensive epicardial (Epi) arrhythmogenic substrate could explain the low efficacy. We report the results of combined endocardial (Endo) and Epi VT ablation and conducting channel (CC) elimination.

Methods and Results—Eleven consecutive patients with ARVD/C were included in the study. A high-density 3D Endo (321 sites mapped) and Epi (302 sites mapped) electroanatomical voltage map was obtained during sinus rhythm to define scar areas (<1.5 mV) and CCs inside the scars, between scars, or between the tricuspid annulus and a scar. The end point of the ablation procedure was the elimination of all identified CCs (scar dechanneling) and the abolition of all inducible VTs. The mean procedure and fluoroscopy time were 177±63 minutes and 20±8 minutes, respectively. Epi scar area was larger in all cases (26±18 versus 94±45 cm², P<0.01). The combined Endo and Epi VT ablation eliminated all clinical and induced VTs, and the addition of scar dechanneling resulted in noninducibility in all cases. Seven patients continued on sotalol. During a median follow-up of 11 months (6–24 months), only 1 (9%) patient had a VT recurrence. There was a single major bleeding event that did not preclude a successful procedure.

Conclusions—Combined Endo and Epi mapping reveals a wider Epi VT substrate in patients with ARVD/C with clinical VTs. As a first-line therapy, combined Endo and Epi VT ablation incorporating scar dechanneling achieves a very good short- and midterm success rate. (Circ Arrhythm Electrophysiol. 2012;5:111-121.)

Key Words: arrhythmogenic right ventricular dysplasia § ventricular tachycardia § ablation § pericardium

Arrhythmogenic right ventricular dysplasia/Cardiomyopathy (ARVD/C) is a genetically determined myocardial disease characterized by right ventricular (RV) atrophy and fibrofatty replacement. This replacement starts at the epicardium or midmyocardium and extends until becoming transmural,1 producing a patchy scar pattern predominantly in the apex, subtricuspid, and outflow tract regions.2 Left ventricular involvement usually is also confined to the posterolateral subepicardium and is present in more than one half of all cases.3,4 Therefore, pathophysiology suggests the epicardium as the most probable location of the ventricular tachycardia (VT) reentry circuits in ARVD/C.

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On the other hand, endocardial (Endo) catheter ablation of VT in ARVD/C has a low acute success rate, with noninducibility ranging from 46% to 75%5-7 and frequent relapses. A VT recurrence rate up to 91% during a 3-year follow-up has been reported.5,8,9 The progressive nature of the disease has been assumed to be the cause of the high recurrence rate.

We hypothesized that a larger epicardial (Epi) substrate could explain the low efficacy of Endo VT ablation in the general population of patients with ARVD/C with clinical VTs and that a first-line, combined Endo and Epi substrate-based ablation would increase the short- and long-term success rate of VT ablation. Complete scar conducting channel (CC) elimination was the substrate-based ablation strategy used.

Methods

Patients

The objective of this single-center prospective study was to analyze the short- and long-term results of a combined Endo and Epi catheter ablation incorporating the scar dechanneling technique as a first-line
therapy in patients with ARVD/C. For this purpose, all patients with ARVD/C diagnosed on the basis of 2010 task force criteria and presenting with a clinically sustained VT episode were included in the study. All patients provided written informed consent to participate.

Electrophysiology Study

The electrophysiology study was performed under intravenous sedation with midazolam and fentanyl. A multipolar diagnostic catheter was positioned at the RV apex, and electrophysiology testing was performed at the beginning of the procedure. A programmed RV stimulation, with up to 3 extraventricular stimuli and burst pacing, was used for VT induction together with isoproterenol infusion, when necessary. The same protocol was repeated after scar dechanneling to test for acute results. The induced VT was considered a clinical VT on the basis of a comparison of 12-lead electrocardiogram morphologies of induced and spontaneous VT in 4 patients (Table 1).

Substrate Mapping

An Endo high-density substrate voltage map of the RV was obtained during stable sinus rhythm using the CARTO system. Image integration between a preacquired contrast-enhanced cardiac CT or contrast-enhanced cardiac magnetic resonance scan was performed using CartoMerge software.

The next step was a percutaneous subxyphoid access to the pericardial space to acquire an high-density substrate voltage map of the RV epicardium. The Epi boundaries of the RV were defined as being opposite to the electroanatomical RV Endo shell. Image integration between contrast-enhanced cardiac CT (9 patients [82%]) or contrast-enhanced cardiac magnetic resonance (2 patients [18%]) and the Endo electroanatomical map permitted further delineation by identifying the course of the right and anterior descending coronary arteries and avoiding radiofrequency (RF) applications over these sites. No attempt was made to identify small branch coronary vessels over the RV. The fill frequency (RF) applications over these sites. No irrigation was used for Epi mapping. Pericardiocent

### Table 1. Clinical Characteristics of the Patient Population

<table>
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<tr>
<th>Patient No.</th>
<th>Age, y</th>
<th>Sex</th>
<th>LVEF</th>
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<th>VT Episodes*</th>
<th>Appropriate ATP/Shock</th>
<th>ECG Morphology</th>
<th>ECG Epi Criteria</th>
<th>AAD</th>
<th>Preprocedure</th>
<th>LV Involvement</th>
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<td>Yes</td>
<td>Sotalol</td>
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<td></td>
</tr>
</tbody>
</table>

ECG criteria from Bazan et al.29

LVEF indicates left ventricular ejection fraction; VT, ventricular tachycardia; ATP, antitachycardia pacing; Epi, epicardial; AAD, antiarrhythmic drug; LV, left ventricular; SMVT, sustained monomorphic ventricular tachycardia; LBBB, left bundle branch block.

*Number of VT episodes in the 6 months before VT ablation.

†Patients without an implanted cardioverter-defibrillator before the ablation procedure.

Conducting Channels

The CCs have been defined as pathways of orthodromically activated sites inside the scar.16,17 In the present study, CCs were identified during stable sinus rhythm. Those CCs between 2 confluent scar areas or between a scar and the tricuspid annulus were considered in addition to intrascar CCs. Scar CCs were identified by (1) a color-coded voltage map adjustment of the lower and upper thresholds (voltage channels) and (2) the presence of ≥2 tagged recordings of E-IDCs, with the delayed component showing sequential orthodromic activation (late potential [LP] channels) (Figures 1–4). After CC identification, the entrance of each CC in the scar was tagged during mapping in the scar area, E-IDCs were tagged.14,15

Ablation

The RF catheter ablation of clinical VT was the first step. Activation mapping and entrainment mapping were used for sustained, well-tolerated VT, and substrate-based ablation guided by pace mapping was used for nonsustained or not-well-tolerated VT.

The RF was delivered with an externally irrigated 3.5-mm tip ablation catheter (Thermocool). The catheter was manipulated through a deflectable sheet (Agilis) for both Endo and Epi mapping and ablation. A 45°C temperature control and 40-W power limit was used at both the endocardium and the epicardium. The irrigation rate was 26 mL/min for Endo RF applications and 17 mL/min for Epi RF applications. No irrigation was used for Epi mapping. Pericardioce-
esis was performed through the deflectable sheath in the event of a significant intraarterial pressure decrease or >200 mL intrapericar-dial saline infusion.

**Scar Dechanneling**

In addition to the clinical VT ablation, elimination of all CCs identified during substrate mapping was attempted by means of combined Endo and Epi scar dechanneling. It has been reported that the higher the scar heterogeneity in patients with ischemia, the higher the arrhythmogenic potential of scars.18,19 Scar heterogeneity can be recognized during substrate voltage mapping and permits identifying CCs of consecutive electrograms with higher voltage amplitude than the surrounding area,16 which have been demonstrated to be responsible for VT reentrant circuits; their elimination renders VTs noninducible.15,20 Therefore, to reduce possible VT relapses using different CCs than those participating in the clinical VT, the substrate-based ablation strategy was an Endo and Epi scar CC elimination on top of clinical VT ablation.

**Figure 1.** Endocardial (left) and epicardial (right) bipolar right ventricular voltage maps of a 34-year-old patient (#9) submitted for evaluation because of a syncopal ventricular tachycardia and diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy based on task force criteria. The electroanatomical map was merged with a preacquired contrast enhanced cardiac magnetic resonance. A subtricuspid inferolateral low-voltage area was observed in the endocardial and epicardial map. The epicardial scar is widest, with higher density of isolated, delayed components (blue dots). Two other small, nonconfluent scar areas were identified epicardially: 1 at the right ventricular outflow tract and 1 at the anterior wall. Therefore, no interscar potential ventricular tachycardia isthmuses were present, and no interscar radiofrequency ablation lines were deployed. In this case, 3 conducting channels were identified on the basis of tagged isolated, delayed components, 2 of which in the epicardial interolateral scar and another in the epicardial right ventricular outflow tract scar.

**Figure 2.** Epicardial color-coded activation maps during sinus rhythm from patient #9 (see Figure 1). Local activation time on normal tissue (>1.5 mV) was considered as the peak of the higher positive voltage deflection. Local activation time on late potential channels (blue dots) was considered as the peak of the latest isolated, delayed component. Electrograms with <0.5 mV and without delayed components were considered as scar tis-sue (gray) with no activation time. Late potential conducting channel entrance (black dots) can be observed in the 3 channels identified in this patient. Channels have an orthodromic sequential activation from the edge of the scar (A). An electrogram with an isolated, delayed component recorded at the entrance (black dot) of the late potential channel located at the right ventricular outflow tract (B) is shown. A comparison of the latest activated zones before (C) and after (D) epicardial scar dechanneling is shown.
After each CC entrance was identified, RF applications (30–60 s) were delivered at these locations. After RF application at the CC entrance, the presence and absence of E-IDC and CC was checked. Backup RF applications inside the scar core area were also performed when the RF lesions at the CC entrance were not able to eliminate internal E-IDCs. After finishing the scar dechanneling, a new high-density postablation remap was performed to document the elimination of all the CCs (Figure 4) and to eliminate new E-IDCs, if necessary.

Linear Ablation Lines

Short linear ablation lines were created to connect scars and to connect a scar to the tricuspid annulus if separated by a BZ region with E-IDCs, which in fact can be considered a CC. E-IDC elimination was also the end point. Normal myocardium was not targeted. Therefore, the distance from the scar to the tricuspid valve or to another scar was determined by the width of the BZ between these structures. The line between the scar and the tricuspid valve was first deployed at the endocardium.

Procedural Success

After finishing the scar dechanneling, programmed RV stimulation with up to 3 extraventricular stimuli and burst pacing was used in all cases. Isoproterenol infusion and stimulation with triple extrastimuli and burst pacing was used if needed for VT induction at the beginning of the procedure. The RF ablation was considered successful when both monomorphic and polymorphic VTs were noninducible. When the clinical VT was successfully ablated but the patient continued to be inducible, partial success was reported. When the clinical VT could not be ablated, ablation failure was reported.

Follow-Up

Patients included in the study were followed for 1, 3, 6, 9, and 12 months and every 6 months thereafter. Each visit included implanted cardioverter-defibrillator (ICD) interrogation and clinical status evaluation. Sotalol was the antiarrhythmic medication of choice and was maintained in patients who were receiving it before the ablation procedure.

Statistical Analysis

Descriptive results are expressed as frequencies and percentages and as mean±SD for qualitative and quantitative variables, respectively. The Wilcoxon signed rank test was used to compare continuous paired data. To compare LP channels to voltage channels, generalized estimating equation methodology was used to account for within-patient correlation. A 2-sided type I error of 5% was used for all tests. SPSS version 18.0 for Windows was used for all statistical analyses.

Results

Patients

The baseline characteristics of each included patient are summarized in Table 1. The mean age of the patient population was 42±13 years (82% men) with a mean LV ejection fraction of 55±7%; only 2 patients had depressed LV ejection fraction in whom LV involvement was observed on the contrast-enhanced cardiac magnetic resonance. The diagnosis of ARVD/C was confirmed in all patients according to 2010 task force criteria, with only 3 (27%) patients having familial forms of the disease. All patients had an ICD, 6 before and 5 after the procedure. Seven patients were taking antiarrhythmic drugs before the procedure (all of whom treated with sotalol) and continued with the same medication afterward under the original study protocol.

The indication for VT ablation was frequent ICD therapies (electrical storm), documented syncopal VT, or tolerated VT in 5, 1, and 5 patients, respectively. All clinical VTs with an
available 12-lead surface ECG had left bundle branch block configuration (n = 7), 4 (57%) of which were inferior axis; the transition in precordial leads was present in > V3 in all except 1 (86%) case. All 7 clinical VTs required an Epi ablation, and ECG criteria suggesting an Epi RV origin were present in 5 (71%) of these epicardially ablated VTs. Ablation was guided by mapping during VT in 4 events and by pace mapping in 3 events.

Electrophysiology Study
The mean procedure and fluoroscopy times were 177 ± 63 and 20 ± 8 minutes, respectively. Because the study protocol aimed to ablate the clinical VT first and perform scar dechanneling thereafter, a VT different from the clinical one was induced in only 3 of 11 patients. In 3 patients, isoproterenol was required to induce the VT. Fourteen VTs were induced and ablated (a mean of 1.3 ± 0.4 VTs per patient). Of these, 2 (14%) were ablated from the endocardium, and 12 (86%) required an Epi ablation. The mean VT cycle length of ablated VTs was 373 ± 132 ms. Ablation was guided by mapping during VT in 7 VTs and by pace mapping in 7 VTs. The characteristics of the epicardially ablated VTs in which ablation was guided by mapping and pacing techniques during VT (n = 7) were (1) a lack of Endo diastolic electrograms during VT in 3 (43%) cases, (2) a focal Endo activation pattern in 1 (14%) case, and (3) a late VT termination with persistent inducibility or no termination in 3 (43%) cases. A lack of concordance between paced QRS morphology and VT morphology was the main reason to opt for Epi ablation when mapping during VT was not possible.
Substrate Mapping and Ablation

Mapping data are summarized in Tables 2 and 3. Endo and Epi voltage maps were obtained in all 11 patients, with a mean of 321 ± 11006 Endo sites and 302 ± 158 Epi sites mapped. Endo scars were identified in 10 (91%) patients, whereas Epi scars were identified in all patients. However, the mean surface scar area was 3 times lower at the endocardium than at the epicardium (26 ± 18 versus 95 ± 45 cm², P < 0.01).

Individually, Endo surface scar area was never larger than Epi scar area (Figure 5). With respect to the electrogram characteristics of the Epi scars (Table 4), the percentage of pathological electrograms (≥ 70 ms width, presence of E-IDC, or both) in Epi BZ areas between 1 and 1.5 mV was 86%, supporting the use of 1.5 mV as the cutoff value for both Endo and Epi scars in patients with ARVD/C (Figure 6).

Conducting Channels

The CCs (Table 2) were identified more frequently by annotating E-IDCs (32 LP channels) than with voltage scanning (10 voltage channels). Intrascar CCs represented 66% of all CCs, followed by 24% being interscar CCs and 10% related to the tricuspid annulus. The mean number of CCs per patient was 3 ± 2, with 78% being Epi. The mean CCs per Epi scar was 1.25 ± 0.85. Table 2 summarizes the differential characteristics of the 2 types of CCs identified. The mean CC width was measured only for voltage channels. Identified channels were measured by means of voltage adjustment, with an upper limit < 1 mV. LP channel width could not be measured because these channels cannot be identified by voltage scanning. To estimate this width, points taken all along and directly next to the LP channel were checked, and 92% did not have E-IDCs, suggesting that LP channels are narrower. The mean distance of the parallel lines of points at each side of the channel was 28 mm. The maximum length of a linear lesion was 37 mm (between 2 confluent scars). The mean length of the linear lesions was 28 ± 10 mm. The location and number of RF ablation lesions are summarized in Table 2.

<table>
<thead>
<tr>
<th>Table 2. Conducting Channel Characteristics</th>
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<td>Total (n=32)</td>
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</tr>
<tr>
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<td>Epicardial CCs</td>
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<td>Location</td>
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<tr>
<td>Interscar</td>
</tr>
<tr>
<td>Scar-tricuspid annulus</td>
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<tr>
<td>Location</td>
</tr>
<tr>
<td>Subtricuspid area</td>
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<tr>
<td>RF applications in CCs</td>
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<td>Entrance</td>
</tr>
<tr>
<td>Entrance + scar core</td>
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<tr>
<td>Linear ablation</td>
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</table>

Data are presented as estimated means (95% CI) (quantitative variables) or n (%) (observed frequencies of qualitative variables). P values were obtained by generalized estimating equation methodology.

LP indicates late potential; CC, conducting channel; RVOT, right ventricular outflow tract; RF, radiofrequency.

*Mean CC width could be measured only for voltage channels (see text).

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Scar Dechanneling

A total of 280 (98 Endo, 182 Epi) RF ablation lesions (Endo, 8.9 ± 7.2; Epi, 16.5 ± 5.4) were created in the whole patient population. Of these, 177 RF ablation lesions (16.1 ± 9.8) were necessary for complete scar dechanneling after induced VT ablation. The location and number of RF applications according to channel type are shown in Table 2. Additional RF applications far away from the edge of the scar were necessary in a total of 5 (23%) LP channels, with only 2 of these cases during the remap. Linear ablation lesions between scars were created in 2 (19%) patients at the epicardium and in 0 patients at the endocardium because Endo scars were never confluent. Endo linear ablation lesions between a scar and the tricuspid valve were created in 3 (27%) patients. Only 1 Epi short linear ablation lesion was created between a scar and the atrioventricular groove. The maximum distance between the core of confluent scars that marked a channel was 28 mm. The maximum length of a linear lesion was 37 mm (between 2 confluent scars). The mean length of the linear lesions was 28 ± 10 mm. The location and number of RF ablation lesions are summarized in Table 2.
Although all tagged E-IDCs of CCs were checked after ablation in all patients, documentation with remapping (231±35 points sampled per map) after scar dechanneling was obtained in the last 5 patients (Table 3). Interestingly, the Epi scar area increased in all cases in which the Epi remap was obtained (from 106±34 to 147±49 cm², P=0.04) (Figures 5 and 7). Linear ablations were performed in all these cases; however, normal myocardium (>1.5 mV) was not targeted in any case. Together with the scar area increase, a trend toward a reduction of the BZ percentage (with respect to the total Epi scar area) from 36.9±12.3% to 13.3±8.5% (P=0.07) was observed.

Follow-Up

During scheduled visits, ICD interrogation showed no ICD shocks delivered during a median follow-up of 11 months (interquartile range, 6–24 months). Only 1 (9%) episode of antitachycardia pacing that terminated the VT was registered in the ICD memory at 3 months after the ablation procedure. This VT episode had a longer cycle length than the ablated one, suggesting a different VT. Sotalol was maintained in the 7 patients who were receiving it before ablation.

Complications

A small quantity of intrapericardial bleeding was observed in the majority of patients. In 1 case, a major bleeding event occurred after RV puncture; 300 mL of blood was extracted and reinfused through the femoral vein without consequences. After bleeding stopped, Epi mapping and ablation were performed without any further complication.

Discussion

Main Findings

The combined Endo and Epi mapping confirms a wider Epi than Endo scar area in all patients with ARVD/C presenting with clinical VTs. As a consequence, the majority of scar CCs are also localized epicardially. These observations are in agreement with pathological findings in ARVD/C hearts, in which fatty or fibrofatty infiltration is much more extensive on the Epi side.4

The present study also highlights that a combined Endo and Epi catheter ablation incorporating the described scar dechanneling technique obtains both a high acute success rate for clinical VT ablation and a low incidence of recurrences during follow-up. The latter suggests that the major cause of recurrences is more related to the ablation technique than to the progressive nature of the disease, at least during a midterm follow-up.

Endo Versus Epi Clinical VTs

Although in some cases electrocardiographic VT characteristics could and Endo mapping could help to identify when a given VT is related to an Epi scar, the high prevalence of Epi VTs in ARVD/C (86% in the present nonselected series) is probably reason enough to support a first-line therapy of combined Endo and Epi mapping and ablation.

In addition, using Endo mapping during VT identified some differentiating characteristics between Endo and Epi scar-related VTs that can be useful in deciding to take an Epi
approach to clinical VT ablation. These criteria merit prospective testing in the present and other substrates, such as LV scar-related VTs.

**Mapping and Ablation**

The Epi mapping and ablation technique has been previously described and used in patients with ARVD/C. In agreement with the present results, it has been reported that patients with a previously failed ablation in whom Epi mapping and ablation has been performed have wider scar areas at the epicardium, with an Endo/Epi scar area proportion that nearly coincides with that of the present study. However, to our knowledge, the present study is the first to report that the Epi scar area is always wider in patients with ARVD/C with or without a previously failed Endo ablation. This information has important clinical value because it strongly supports a change from the standard approach to a combined Endo and Epi ablation strategy as a first-line therapy for these patients.

**CC Identification**

It has been demonstrated that scanning the lower and upper bipolar voltage thresholds is key to CC identification in the setting of ischemic cardiomyopathy. On the other hand, E-IDCs are present in locations where CCs are identified on the basis of voltage scanning. An interesting observation in the present study was the fact that the majority of CCs were identified on the basis of a visual electrogram analysis that tagged consecutively activated late potentials and could not be identified by voltage scanning. These LP channels have several characteristics that differentiate them from the voltage channels: (1) a lower voltage, (2) a predominant location within the scar core, and (3) a tendency to have longer activation times. The fact that E-IDCs in LP channels could only be detected at a single point all along its course and that fewer RF applications were required at its entrance for complete elimination also suggests that these channels are narrower than the voltage channels, more superficially located on the epicardium, or both. From a practical point of view, the present study suggests that LP channels are an integral part of the substrate for recurrent VTs and that a combined Endo/Epi ablation strategy may be effective in these patients.

![Figure 5. Comparison of endocardial and epicardial scar areas. Shown are the size of scar before the ablation procedure for each individual patient (A), the increase of the epicardial scar area before and after the ablation (B), and the decreased heterogeneity of the scars after ablation (C).](image)
Table 4. Electrogram Analysis of the Epicardial Scar in Each Patient

<table>
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<tr>
<th>Patient No.</th>
<th>E-IDC in Core (&lt;0.5 mV)</th>
<th>E-IDC in BZ (1–1.5 mV)</th>
<th>BZ (1–1.5 mV) Electrogram Width, ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Yes</td>
<td>No</td>
<td>94±25</td>
</tr>
<tr>
<td>2</td>
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<td>No</td>
<td>88±24</td>
</tr>
<tr>
<td>3</td>
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<td>Yes</td>
<td>92±16</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>Yes</td>
<td>84±19</td>
</tr>
<tr>
<td>5</td>
<td>Yes</td>
<td>No</td>
<td>81±24</td>
</tr>
<tr>
<td>6</td>
<td>Yes</td>
<td>No</td>
<td>90±18</td>
</tr>
<tr>
<td>7</td>
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<td>Yes</td>
<td>81±13</td>
</tr>
<tr>
<td>8</td>
<td>Yes</td>
<td>No</td>
<td>85±18</td>
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<tr>
<td>9</td>
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<td>Yes</td>
<td>102±35</td>
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<td>10</td>
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<tr>
<td>Mean±SD</td>
<td></td>
<td></td>
<td>86±8</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD. The presence of abnormal electrograms (those with E-IDC, >70 ms width, or both) was checked in the core and in the BZ area between 1 and 1.5 mV.

E-IDC indicates electrogram with isolated, delayed components; BZ, border zone.

Outcomes and Complications

The purpose of the stimulation protocol at the beginning of the study was to induce clinical VT. This explains the relatively low number of induced VTs. During this protocol, a VT different from the clinical one was induced in only 3 of the 11 patients. In addition to the clinical VT ablation, the scar dechanneling strategy renders a high proportion of patients noninducible (100% in the present study). The high rate of short-term success is accompanied by a lack of recurrences over a midterm follow-up. Previous reports showed no relationship between procedural success and freedom from recurrences in patients with ARVD/C. However, in the present series, short-term procedural success (ie, noninducibility) was concordant with the lack of recurrences.

The most probable explanation for the low recurrence rate is that the complete Endo and Epi scar dechanneling eliminated a high proportion (if not all) of potential scar-related VT circuits. The lower recurrence rate in the present study during a similar follow-up makes it improbable that the progressive nature of the disease is the only cause of the high recurrence rate in previous series.

Although larger studies should be performed to confirm the present results, given the lack of severe complications, this approach could be recommended as the first-line therapy for VT ablation in this subset of patients, at least in high-volume centers. Lower-volume centers might have more bleeding complications and perhaps not as high a success rate.

Limitations

Two strategies have been tested in this study: combined Endo and Epi mapping and ablation and scar dechanneling. The short-term efficacy of scar dechanneling cannot be calculated individually because a complete stimulation protocol was not repeated after clinical or induced VT ablation. However, given that the majority of the substrate for clinical and nonclinical VTs was present at the epicardium, the combined approach seems justified, and each strategy can be assumed to have contributed to the overall results of this study. Furthermore, previous studies on VT ablation in ARVD/C have not obtained a 100% noninducibility after scar dechanneling, suggesting a significant effect of the combined protocol not only in short-term, but also in midterm outcomes. Finally, the possibility exists that some CCs do not participate in VTs during the stimulation protocol but do activate during follow-up. Thus, the only possibility to determine the proportionate

Figure 6. Epicardial substrate voltage maps with the border zone area defined between 0.5 and 1.5 mV (left) and 0.5 and 1 mV (right). The border zone area between 1 and 1.5 mV contains electrograms with isolated, delayed components (blue dots), vessels, and probably fat surrounding them. The scar area becomes smaller by reducing the voltage threshold (ie, <1 mV); however, this reduction results in an underestimation of the actual scar area containing conducting channels, particularly in those areas not covered by fat.
advantage of scar dechanneling over a combined Endo and Epi strategy for clinical and inducible VT ablation would be to perform a larger, randomized, follow-up study.

Conclusions
Combined Endo and Epi mapping reveals a wider Epi VT substrate in all patients with ARVD/C with clinical VTs. As a first-line therapy, combined Endo and Epi VT ablation incorporating scar dechanneling achieves a very good short- and midterm success rate.

Acknowledgments
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Disclosures
None.

References
Patients with ventricular tachycardias due to arrhythmogenic right ventricular cardiomyopathy often have extensive abnormalities in the right ventricle, leading to multiple potential electrical conducting channels. This study reports the results of a first-line, combined, endocardial and epicardial catheter ablation approach in arrhythmogenic right ventricular dysplasia/cardio-myopathy, incorporating the scar dechanneling technique. The end point of the ablation procedure was the elimination of all identified conducting channels (scar dechanneling) and abolition of all inducible ventricular tachycardias. This approach obtained a 100% noninducibility and very low recurrence rate at midterm. In addition, it permitted recognition that the epicardial scar area is wider than the endocardial scar area and that the majority of conducting channels are present on the epicardial surface. Given the high efficacy and lack of severe complications, this approach could be recommended as the first-line therapy for ventricular tachycardia ablation in patients with arrhythmogenic right ventricular dysplasia/cardio-myopathy, at least in high-volume centers.
Combined Endocardial and Epicardial Catheter Ablation in Arrhythmogenic Right Ventricular Dysplasia Incorporating Scar Dechanneling Technique
Antonio Berruezo, Juan Fernández-Armenta, Lluís Mont, Hrvojka Zeljko, David Andreu, Csaba Herczku, Tim Boussy, Jose María Tolosana, Elena Arbelo and Josep Brugada

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