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Scar Dechanneling: A New Method for Scar-Related Left Ventricular Tachycardia Substrate Ablation

Running title: *Berruezo et al.; Scar Dechanneling for Scar-Related VT*

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Abstract:

Background - Ventricular tachycardia (VT) substrate ablation usually requires extensive ablation. Scar dechanneling technique may limit the extent of ablation needed.

Methods and Results - The study included 101 consecutive patients with left ventricular (LV) scar-related VT (75 ischemic, LV ejection fraction 36±13%). Procedural endpoint was the elimination of all identified conducting channels (CCs) by ablation at the CC-entrance followed by abolition of residual inducible VTs. By itself, scar dechanneling rendered non-inducible 54.5% of patients; ablation of residual inducible VT increased non-inducibility to 78.2%. Patients needing only scar dechanneling had a shorter procedure (213±64 vs 244±71 min, *P*=0.027) and fewer RF applications (19±11% vs 27±18%, *P*=0.01) and external cardioversion/defibrillation shocks (20% vs 65.2%, P<0.001). At 2 years, patients needing scar dechanneling alone had better event-free survival (80% vs. 62%) and lower mortality (5% vs. 11%). Incomplete CC-electrogram elimination was the only independent predictor [HR 2.54 (1.06-6.10)] for the primary endpoint. Higher endpoint-free survival rates were observed in patients non-inducible after scar dechanneling (log-rank *P*=0.013) and those with complete CC-electrogram elimination (log-rank *P*=0.013). The complications rate was 6.9%, with no deaths.

Conclusions - Scar dechanneling alone results in low recurrence and mortality rates in more than half of patients despite the limited ablation extent required. Residual inducible-VT ablation improves acute results but patients who require it have worse outcome. Recurrences are mainly related to incomplete CC-electrogram elimination.

Key words: ventricular tachycardia, ablation, outcome, conducting channels, scar dechanneling, substrate ablation

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Introduction

Acute results and long-term outcomes of conventional VT ablation in patients with LV scar-related VTs are suboptimal. The reported non-inducibility rate of sustained monomorphic VT after ablation ranges from 38% to 73% of patients, and this translates into a high recurrence rate, i.e., 50% to 88% during a follow-up of 12 months or more, despite the use of anti-arrhythmic drugs[1]. Acute prevention of VT inducibility after catheter ablation is related to a lower, though still significant, VT recurrence rate during follow-up[2]. Therefore, non-inducibility seems a desirable but insufficient acute endpoint.

A further step in reducing recurrences after ablation could be the complete elimination of all the substrate responsible for VT occurrence, a strategy based on the assumption that the substrate not participating in VTs at the time of ablation could activate and become part of a VT isthmus during follow-up. Recent reports have shown that substrate elimination, independent of participation or not as a VT circuit during the procedure, can be used as an acute endpoint[3-5]; however, extensive ablation is still required[4], the best technique for its elimination has not yet been described, and recurrences remain high[5]. In addition, the results obtained by substrate ablation as the first step during the ablation procedure for scar-related VTs are not yet known; in previous studies, the protocol started with VT induction and attempted ablation of sustained and tolerated VTs[4,5].

Substrate mapping during sinus or paced rhythm permits characterization of scar areas supporting possible re-entry circuits of VTs, called conducting channels (CCs) [6]. These CCs can be identified by voltage mapping and/or electrogram analysis[7-9] and are considered the ablation target[1]. More specifically, in small series of patients with arrhythmogenic cardiomyopathy and ischemic cardiomyopathy, it has been suggested that RF applications at the CC entrance (the

"scar dechanneling" technique) can homogenize the scar without extensive ablation and could possibly increase ablation efficiency[7-10].

We hypothesized that scar dechanneling is a feasible first step (before VT induction and ablation attempt) for patients with LV scar-related VT and for many patients can result in less extensive ablation and no ventricular arrhythmia during the procedure. We report the procedure details and outcomes of this strategy.

Methods

Patient Sample

All consecutive patients (n=101) undergoing LV scar-related VT ablation at a single center from November 2009 to April 2013 were prospectively included in the study and followed up. Inclusion criteria were the presence of structural heart disease –prior myocardial infarction, LV dilatation/systolic dysfunction or normal LV diameters/systolic function with evidence of ventricular scar on contrast enhanced-cardiac magnetic resonance (ce-CMR) or electroanatomic map (EAM)– and sustained monomorphic VT documented by 12-lead ECG or implantable cardioverter defibrillator (ICD) electrograms. Patients with ventricular arrhythmias due to reversible causes were excluded. All patients provided written informed consent to participate. The study was approved by the Local Ethics Committee.

Pre-procedural Evaluation

Each patient received a complete clinical evaluation. Before the indexed ablation procedure, all patients underwent transthorathic and/or transesophageal echocardiogram to rule out atrial or LV thrombus and evaluate LV function. Whenever possible, 3-Tesla ce-CMR was acquired to identify scar presence and merge the images with those obtained by EAM. In the absence of ce-CMR or when epicardial ablation was anticipated, a computed tomography (CT) scan was

acquired to merge images[11].

VT Ablation Procedure

Procedures were performed under conscious-sedation anesthesia (midazolam and fentanyl), or general anesthesia when epicardial access was anticipated. Invasive arterial pressure was obtained by radial artery cannulation. After femoral venous access, a multipolar diagnostic catheter was positioned at the RV apex. Endocardial access to the LV was obtained by single transseptal puncture (BRK needle: Medtronic Inc., Minneapolis, MN, USA), after which heparin was intravenously administered to maintain activated clotting time >250 seconds. Epicardial mapping and ablation was performed when pre-procedural ce-CMR showed epicardial scar, endocardial mapping did not identify subendocardial scars, ECG of clinical or induced VT suggested epicardial origin[12,13], or endocardial ablation was unsuccessful. Any pericardial adherences were removed via subxiphoid access by a cardiac surgeon in the electrophysiology laboratory or using a steerable sheath (Agilis St Jude Medical, St Paul, MN, USA). The procedure followed a 4-step protocol (Figure 1).

First step: Substrate mapping and CC identification/classification

Transseptal LV access was achieved and manipulation of the ablation catheter was assisted by a steerable sheath (Agilis St Jude Medical, St Paul, MN, USA). Retroaortic approach was used for endocardial LV mapping in 10 (9.9%) patients. High-density EAM was obtained of LV during stable sinus rhythm using the CARTO system (Biosense Webster, Diamond Bar, CA, USA) in 96 patients and Ensite Navx (St Jude Medical, St Paul, MN, USA) in 5 patients. Bipolar electrograms were filtered from 30 to 250 Hz. Scar areas were identified using standard voltage cut-off values for dense scar (<0.5 mV) and border zone (BZ) (<1.5 mV).

Electrograms with delayed components (E-DCs) were tagged and dichotomously

classified as entrance or inner CC points, depending on delayed-component precocity during sinus rhythm. The CC entrance was defined as the E-DC with the shortest delay between the far-field component of healthy/BZ muscle (low frequency, usually high voltage) and local component (delayed, high frequency, usually fractionated and low voltage) corresponding to the local activation of myocardial fibers in the scar (Figure 2). To avoid targeting healthy tissue beyond the scar area, CC entrances were tagged in a zone with 0.5-1.5 mV voltage (Figure 2).

Image integration between pre-acquired CT and/or ce-CMR, when available, was performed with CartoMerge software (CartoMergeR® Image Integration Software Module, Biosense Webster, Inc., Diamond Bar, CA, USA).

Second step: CC Elimination

Procedural endpoint at this step was RF elimination of all identified CCs by RFA at the CC entrance during sinus rhythm ("scar dechanneling"), using an externally irrigated 3.5-mm tip ablation catheter (Thermocool, Navistar, Biosense Webster) with 45ºC temperature control, 50W power limit, and 26-30 ml/min irrigation rate (40W and 17 ml/min at epicardium). Each RF application lasted 30-60 seconds, depending on elimination of the delayed component, and abolition/persistence of CC electrograms was checked with the ablation catheter. If needed, a new application was performed at the closest site having CC-entrance characteristics on E-DC until the entrance was blocked. CC-entrance conduction block was considered in cases of a) CC-inner points disappearance or automaticity with exit block or b) delayed activation of CC inner points, usually with the activation sequence in reversed order (Figure 3). Although the second criterion can not exclude conduction delay in the targeted entrance, RF was moved to another entrance when observed, in order to limit the RF delivery and coming back to the same entrance if the channel isolation was not achieved. Backup RF applications were delivered inside the core

scar area when RF lesions at the CC entrance did not eliminate internal E-DCs. If BZ tissue with E-DCs between scar/mitral annulus was identified, a short ablation line was used to achieve the endpoint.

Third step: Re-mapping and residual-CC ablation

Re-mapping was used to confirm the elimination of all CCs and check for residual E-DCs, focusing on the scar area and attempting to match the mapping density of the initial EAM. Residual E-DCs identified by re-mapping were targeted with the same approach (target and RFA setup) used during initial mapping and ablation (Figure 4).

Fourth step: Inducibility and residual-VT ablation

Programmed RV stimulation from the RV apex, with three basal cycle lengths (600-500-430 ms), up to 3 extraventricular stimuli decremented until refractoriness or 200 ms, and burst pacing up to 200 ms was used for VT induction, without isoproterenol infusion. The protocol was stopped when completed or VT/VF was induced. Overdrive pacing was used before electrical cardioversion to stop VT. Induced VT was considered the clinical VT, comparing 12-lead electrocardiographic morphologies, cycle lengths, and/or intracardiac electrogram morphologies.

In the case of residual sustained monomorphic VT induction, this VT was targeted for ablation by mapping/pacing techniques during VT (if tolerated) or during sinus rhythm (if untolerated/unsustained). Inducibility was checked after each induced VT was ablated.

Procedural Success

Induction protocol results were used to evaluate procedural success. Acute success was reported both for patients needing scar dechanneling alone (no further VT mapping needed because they became noninducible) and for those needing the complete procedure, including residual-VT ablation. Non-inducibility was considered when sustained monomorphic VT was not inducible.

The absence/persistence of E-DCs that could not be eliminated was also reported.

Follow-up

The endpoint was the occurrence of any sustained ventricular arrhythmia episode or sudden cardiac death. Each follow-up visit, at 1 month and every 6 months thereafter, included ICD interrogation and clinical status evaluation. Two investigators (and a third in cases of discrepancy) evaluated each stored arrhythmia episode. Ventricular arrhythmia episodes in non-ICD patients were collected from all types of ECG registry/monitoring.

Statistical Analysis

Values are expressed as percentage, mean±SD or median [interquartile range (IQR)]. T-test was used to compare continuous paired (endo vs. epicardial and map vs. remap electroanatomical data) and unpaired data (clinical and electrophysiological variables between ischemic vs. non-ischemic patients and with complete vs. incomplete CC ablation), and Wilcoxon signed rank test and Mann-Whitney U test for non-normally distributed data. We have performed the comparison of the characteristics of the bipolar electrograms with nested ANOVA methodology. Kaplan-Meier analysis was used to estimate event-free survival probabilities and log-rank test for between-group comparison. Variables selected from the univariate analyses ($P \leq 0.10$) were entered into multivariable Cox proportional hazards regression models to estimate predictors of VT recurrence. $P<0.05$ was considered significant. Statistical analysis was performed using PASW Statistics 18.0 software (SPSS Inc, Chicago, IL, USA).

Results

Patient Sample

The sample included 101(41%) of 246 patients that underwent any ventricular arrhythmia ablation during the study period. Baseline patient characteristics are summarized in Table 1.

An ICD was implanted in 53 (52.5%) patients before their referral for ablation, in 29 (28.7%) post-ablation, and 19 (18.8%) patients did not receive an ICD. For image fusion purposes, 39 (38.6%) patients underwent pre-procedure ce-CMR and 24 (23.8%) patients a CT scan.

Ablation Procedure

The mean procedure duration and fluoroscopy time were 227±69 min (range 90-480 minutes) and 18±8 min (range 2-42 minutes), respectively.

First step: Substrate mapping and CC identification/classification

Substrate mapping during sinus rhythm as a first step was precluded in 6 (5.9%) patients with incessant VT or frequent repetitive VT episodes, which required starting with conventional VT activation/entrainment mapping and ablation. In all other patients (94.1%), the procedure began with substrate mapping (data summarized in Table 2). The mean number of points acquired to build the endocardial and epicardial EAMs were 481±187 and 486±218, respectively.

Epicardial mapping was attempted in 31 (30.7%) patients and succeeded in 27 (26.7%), with epicardial access obtained at the beginning of the procedure in 18 (17.8%) patients and after failed endocardial VT ablation in 9 (8.9%) patients. In 2 of these patients, only the epicardium was mapped because of no endocardial scarring on the voltage map from a previous VT ablation attempt (another institution) or ce-CMR.

Electrogram characteristics of the endocardial and epicardial CCs are summarized in Table 3. CC-entrance electrograms on the lateral scar edge showed significantly later delayed-component lateness with respect to the beginning of the QRS complex, compared with those located at the septal scar border (see example, Figure 5). The amplitude of the delayed component of the CC electrograms tended to be greater in epicardial scars, without differences in lateness compared to the QRS-complex onset.

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Second step: CC elimination

Complete scar dechanneling could be achieved in 85(84.2%) patients. The median endocardial and epicardial CC-entrance electrograms targeted for ablation was 8(4-13) and 6(3-10), respectively, per patient. More CC-entrance electrograms were targeted for ablation on the endocardium in ischemic as compared with nonischemic patients (10(5-10) vs 3(1-7), $P<0.001$); there were no differences between them on the epicardium (5(3-8) vs 8(2-11), $P=0.611$). A median of 18(9-28) endocardial and 8(4-14) epicardial RF applications per patient were necessary for elimination of all the E-DCs identified. Short linear ablation lesions were created in 21(20.8%) patients, most of them (66.6%) between a scar and the mitral annulus; none were created intra-scar. Table 2 summarizes RF ablation data.

Third step: Re-mapping and residual-CC ablation

A mean of 567±327 points was acquired during endocardial remapping. Mean scar area identified during remapping (75±45 cm^2) was larger than that of the first map (52±39 cm^2), $P<0.001$. The epicardial remap (524±417 sites) also had a larger scar area than the first map (71±52 vs 44±41 cm2, $P=0.009$). The mean number of endocardial and epicardial CC electrograms per patient decreased from 42 (25-72) and 52 (26-69) in the map to 10 (3-18) and 13 (0-26) in the remap ($P<0.001$ and $P=0.001$), respectively. Eliminating these residual-CC electrograms required a median of 7(4-15) endocardial and 3(0-9) epicardial RF applications.

Complete scar dechanneling could not be achieved in 16 (15.8%) patients due to the proximity of coronary arteries (1 patient), phrenic nerve capture (1 patient), or conduction system (His bundle, left bundle branch) electrogram recording at the target site for RF application (5 patients) –or simply because E-DCs could not be eliminated despite repeated RF delivery (9 patients).

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Fourth step: Inducibility and residual-VT ablation

After complete scar dechanneling, 55 (54.5%) patients were non-inducible; in the 46 inducible patients, 71 of 75 residual sustained monomorphic VTs were targeted for ablation. Ablation was guided by mapping/pacing during 40 VTs and by pace-mapping in 31 . The mean cycle length of residual VTs was 335±88 msec.

Endocardial ablation was successful in 38 (50.7%) residual VTs, epicardial ablation in 13 (17.3%), and ablation failed in 20 (26.7%) VTs. Therefore, the non-inducibility of sustained monomorphic VTs increased from 54.5% after scar dechanneling, to 78.2% after residual-VT ablation. The cycle length of epicardially-ablated VTs did not differ significantly from endocardially-ablated VTs (339±86 ms vs 349 ±102 ms, respectively; P=0.284).

Procedure-Related Complications

Acute complications potentially related to the procedure occurred in 7 (6.9%) patients; there were no procedure-related deaths. Fewer complications occurred in patients rendered non-inducible by scar dechanneling alone than in patients requiring residual-VT ablation (1.8% vs 13%, respectively; P=0.027). Direct-current cardioversion was necessary in 11 (20%) patients needing only scar dechanneling vs 30 (65.2%) requiring residual-VT ablation (P<0.001). Two patients had cardiac tamponade requiring pericardiocentesis after endocardial ablation. Two patients presented with complete AV block; one of them (with previous LBBB) underwent CRT-D implantation, the other had permanent atrial fibrillation and a previously implanted single-chamber ICD. In the first weeks of follow-up, 2 patients with epicardial ablation had post-procedural pericarditis with pericardial effusion, one of them requiring pericardiocentesis. One patient had transient ischemic attack without sequelae, another with non-ischemic cardiomyopathy had cardiogenic shock, and symptomatic phrenic nerve palsy was detected

during follow-up in 1 patient who underwent epicardial RF ablation.

Follow-up

After a median (IQR) follow-up of 21 (11-29) months, recurrences of sustained ventricular arrhythmia episode or sudden cardiac death were observed in 27(26.7%) patients. Oral amiodarone was prescribed at some point in 28(27%) patients. There were no significant differences in amiodarone usage before ablation in inducible vs noninducible patients after scar dechanneling (34.8% vs 21.8%, P=0.147), in amiodarone administration after the procedure in patients with or without VT recurrence (34.6% versus 25.3%, P=0.362), nor in patients who died vs survivors (22.3% versus 28.3%, P=0.699). ICD interrogation showed that 20 (19.8%) patients had appropriate therapies. Of these, 10(9.9%) received ICD shocks and 10 (9.9%) had VT episodes that were terminated by anti-tachycardia pacing. Cycle length of the recurrent VTs during follow-up exceeded that of pre-procedure clinical VTs (399±78 ms vs 342±86 ms, respectively; P=0.049). Four (3.9%) patients experienced VTs that were monitored in the slow VT detection zone and did not receive therapy. Two patients without ICD implantation, due to comorbidities and low life expectancy, had VT recurrence; one of them required re-ablation.

Nine (8.9%) patients died during follow-up, of various causes: 4 of advanced heart failure (4%); 1 of sudden cardiac death (severe comorbidities, low life-expectancy); 1 of arrhythmic storm (despite ICD); 2 of non-cardiac death (acute renal failure with heart failure decompensation and nosocomial pneumonia, respectively); and cause of death is not available for 1 patient.

Table 4 shows univariate and multivariable Cox regression analysis results for the study endpoint. Univariate analysis showed four predictors of the primary endpoint: VT inducibility after scar dechanneling (HR 2.58 [1.18-5.67], *P*=0.018), VT inducibility after residual-VT

ablation (HR 2.59 [1.18-5.68], *P*=0.018), epicardial mapping (HR 0.16 [0.05-0.57], *P*=0.004), and incomplete CC-electrogram elimination (HR 3.50 [1.55-7.87], *P*=0.003). Under multivariable analysis, the only independent predictor of any sustained ventricular arrhythmia episode or sudden cardiac death was incomplete CC-electrogram elimination (HR 2.54 [1.06-6.10], *P*=0.037).

Survival curve analysis (Figure 6) showed higher endpoint-free survival for patients with complete CC-electrogram elimination (log-rank test P=0.001).

Scar Dechanneling as the First Step

Scar dechanneling was the first step and the only ablation strategy needed for obtaining non-inducibility in 54.5% of patients; in consequence, the procedure duration was shorter than that of patients requiring residual inducible-VT ablation (213±64 vs 244±71 minutes, *P*=0.027).

Baseline characteristics and procedure-related data of these patients and those needing residual inducible-VT ablation are summarized in Table 5. Despite having no significant differences in LVEF and myocardial scar area, patients needing only scar dechanneling had fewer endocardial E-DCs (36±29 vs 50±32, *P*=0.04), required fewer RF applications (19±11 vs 27±18, *P*=0.01), and less frequently had incomplete CC-electrogram elimination (7.3% vs 26.1%, P=0.01). Most importantly, they had higher 2-year probability of overall (95% vs. 89%) and event-free survival (80% vs. 62%) (Figure 6).

Re-do procedures

A re-do procedure using the same ablation protocol was performed in 11 of the 27 patients who reached the primary endpoint (8 ischemic, 3 non-ischemic cardiomyopathies) at a median 11(3-18) months after the index ablation. The indication for the re-do procedure was appropriate ICD therapy in 6 patients, slow VT with no therapy in 4 patients, and VT requiring external

cardioversion in 1 patient without ICD. The mean VT cycle length was 401±96ms. Nine (82%) of these 11 patients required epicardial ablation, 5 needed a combined endocardial/epicardial substrate ablation, and ablation was exclusively epicardial in 4 patients. In 2 patients, surgical subxiphoid access was necessary. In addition to substrate ablation, 7 more VTs were targeted and ablated during the re-do procedures, 6 of them from the epicardium.

A third procedure was necessary in 2 patients, both in the first month after re-ablation. Both patients had incessant VT that required high RF-energy delivery, in the LV septum in an ischemic patient with anterior aneurysm and in epicardial LV summit in a patient with dilated cardiomyopathy. During a median follow-up of 19 (11-28) months, 15 (14.9%) patients reached the endpoint of any sustained ventricular arrhythmia episode or sudden cardiac death after 1.13 procedures per patient.

Discussion

Main Findings

The present study shows that substrate ablation with the scar dechanneling technique as the first step of the ablation protocol (before any VT induction/ablation attempt) renders more than half of the patients with LV scar-related VTs non-inducible. These patients can benefit from starting and finishing the procedure in sinus rhythm, with lower procedural requirements (procedure duration, RF delivery), fewer complications, no hemodynamic instability, and a much lower chance of direct current cardioversion/defibrillation. In the long term, a patient subgroup (ie, non-inducible after scar dechanneling) is identified that, despite a similar clinical profile (age, VT cycle length, LVEF, scar size), have a lower probability of VT recurrences or sudden death (16.4% vs 37%, P=0.018) and lower mortality (3.6% vs 15.2%, P=0.042), compared to patients needing residual inducible-VT ablation. The differences in procedure requirements and better

outcomes likely reflect that patients needing scar dechanneling alone have a more accessible substrate.

Although other substrate-guided VT strategies using different ablation targets have been described, they systematically included an induction protocol, and ablation of sustained VT, if well tolerated, before substrate ablation[3-5]. The present findings suggest substrate ablation as the preferred first step, regardless of the technique used.

CC Identification and Ablation

CCs have been reliably identified in right ventricular dysplasia[7, 9], ischemic[6, 14] and also in non-ischemic[8] cardiomyopathy. Therefore, it could be assumed that they are present in every scar-related VT. In the present study, although differences in scar distribution depending on cardiomyopathy type have been noted, the technique has been equally applicable in the different substrates.

The study definition of a CC-entrance electrogram was the same at that used for scar dechanneling in ARVD[7,9] as it does not depend on activation time or the voltage of the delayed component. The definition only takes into account the relative position of the local electrogram in the CC activation sequence. The CC-entrance electrograms were mostly located at the scar edge, as in the case of ARVD. Dichotomization of E-DCs into entrance and inner points, without the need for tracking along every single CC path, facilitated mapping, because CC are three-dimensional and a planar construct on a single endo/epicardial surface is an oversimplification and sometimes not possible[14].

Various definitions have been used to identify the target electrogram in substrate ablation[3-5, 15-16], mainly based on the local electrogram delay. As shown in the present study, the delay of the local EG with respect to the beginning of the QRS is not critical to be qualified as a

CC-entrance, as this delay mostly depends on the septal vs lateral position at the scar edge. With respect to the lateness or separation between the delayed component and the far-field electrogram, the definition used for the CC entrance makes it non-quantifiable because fusion is usually present. This supports a recent study showing that local electrogram lateness is affected by its location with respect to the septum, other early-to-activate regions, and the scar margin[17]. No significant differences between endocardial and epicardial CCs were observed with respect to activation time. However, a higher voltage of the local electrogram was found on the epicardium, which could be explained by the higher scar heterogeneity (border zone percentage of scar) at the subepicardial LV wall in ischemic patients[14], as the bipolar voltage of the border zone areas is higher than that of the scar core areas[18].

With respect to the RF energy delivered, although populations are not fully comparable between different studies, 28±16 minutes of RF application with scar dechanneling favorably compare with the requirements of other substrate ablation strategies, such as "linear ablation"[19] or "scar homogenization"[4] and approximate "LAVA" ablation[5]. It also has been recently confirmed that targeting the earlier late potentials contained in the conducting channels (CC-entrance) can eliminate inner CC-electrograms and homogenize the scar without extensive ablation, increasing the efficiency of the ablation methodology used[10].

Outcomes

The 26.7% event rate for the study endpoint of any sustained ventricular arrhythmia or sudden cardiac death is in the lower range of previously published studies in which the endpoint was complete substrate elimination using different ablation targets[4,5,20], and is better than previous studies on standard VT ablation[21]. However, the most attractive finding is the possibility of identifying a large subgroup of patients with a very low event rate when complete substrate

ablation is the first step taken, before any VT induction/ablation attempt. These patients maintain sinus rhythm during the whole procedure because they become non-inducible after scar dechanneling. This subgroup cannot be distinguished by clinical data (except for the lower proportion of VT storm before the procedure), echocardiographic characteristics, or scar dimensions from those who will remain inducible. However, they have fewer E-DCs and require fewer RF applications to obtain complete CC-electrogram elimination, which in turn is the only independent predictor for VT recurrence or SCD.

Interestingly, 82% of patients undergoing a re-do procedure required epicardial ablation. If epicardial mapping/ablation is considered for the first ablation procedure in appropriate patients (eg, non-ischemic patients with evidence of epicardial scar or ischemic patients with transmural scars), there is a potential margin for improved outcomes[22]. In fact, in the present study, epicardial mapping was significantly associated with a lower event rate for the primary endpoint in univariate analysis.

The LVEF did not differ between patients needing scar dechanneling alone vs those requiring residual inducible-VT ablation, nor was it a predictor of clinical outcomes during follow-up. Primary prevention studies have shown that the presence, quantity, and characteristics of the arrhythmogenic substrate (i.e., the scar and its heretogeneity) provide the strongest predictor of arrhythmic events[23]. Therefore, it should not be surprising that the only independent predictor of clinical outcomes after ablation is the variable most closely related with incomplete substrate elimination (i.e., incomplete CC-electrogram elimination). LVEF was not a predictor of recurrence in other studies on VT substrate ablation that have attempted complete elimination of the arrhythmic substrate[4,5].

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Limitations

This study could not determine whether some of the benefits observed –lower procedure requirements, fewer complications, and better outcomes for patients needing only the scar dechanneling technique– are exclusively due to substrate ablation as the first step during the procedure or involve other conditions that permit the use of this strategy (ie, the absence of incessant VT) or uncontrolled determinant factors not taken into account. Improved outcomes observed in patients for whom scar dechanneling alone achieved non-inducibility suggests that this subgroup had a more accessible substrate. A small proportion of patients with clinical VTs could be noninducible before ablation; the lack of a basal induction protocol limits the evaluation of the acute effect of substrate ablation. However, taking into account that scar dechanneling seeks the complete elimination of all the substrate, the induction or not at the beginning of the procedure will not change the ablation set. Therefore, testing only the acute effect of scar dechanneling might not provide sufficient justification to perform the initial VT induction protocol, which increases the procedure duration and is not free of complications. Finally, because substrate mapping was performed during intrinsic rhythm in the vast majority of patients, we did not analyze the effect of different activation wavefronts (i.e. intrinsic rhythm vs paced rhythm) on CC entrance location and characteristics.

Conclusions

Scar dechanneling alone results in low recurrence and mortality rates in more than half of patients despite the limited ablation extent required. Residual inducible-VT ablation improves acute results but patients who require it have worse outcome. Recurrences are mainly related to incomplete CC-electrogram elimination.

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Conflict of Interest Disclosures: David Andreu is an employee of Biosense Webster.

References:

1. Aliot EM, Stevenson WG, Almendral-Garrote JM, Bogun F, Calkins CH, Delacretaz E, Della Bella P, Hindricks G, Jaïs P, Josephson ME, Kautzner J, Kay GN, Kuck KH, Lerman BB, Marchlinski F, Reddy V, Schalij MJ, Schilling R, Soejima K, Wilber D; European Heart Rhythm Association (EHRA); Registered Branch of the European Society of Cardiology (ESC); Heart Rhythm Society (HRS); American College of Cardiology (ACC); American Heart Association (AHA). EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). *Heart Rhythm.* 2009;6:886-933.

2. Carbucicchio C, Santamaria M, Trevisi N, Maccabelli G, Giraldi F, Fassini G, Riva S, Moltrasio M, Cireddu M, Veglia F, Della Bella P. Catheter ablation for the treatment of electrical storm in patients with implantable cardioverter-defibrillators: short- and long-term outcomes in a prospective single-center study. *Circulation.* 2008;117:462-469.

3. Vergara P, Trevisi N, Ricco A, Petracca F, Baratto F, Cireddu M, Bisceglia C, Maccabelli G, Della Bella P. Late potentials abolition as an additional technique for reduction of arrhythmia recurrence in scar related ventricular tachycardia ablation. *J Cardiovasc Electrophysiol*. 2012;23:621-627.

4. Di Biase L, Santaangeli P, Burkhrdt DJ, Bai R, Mohanty P, Carbucicchio C, Dello Russo A, Casella M, Mohanty S, PumpA, Hongo R, Beheiry S, Pelargonio G, Santarelli P, Zucchetti M, Horton R, Sanchez JE, Elayi CS, Lackkireddy D, Tondo C, Natale A. Endo-epicardial homogenization of the scar versus limited substrate ablation for the treatment of electrical storms in patients with ischemic cardiomyopathy. *J Am Coll Cardiol.* 2012;60:132-141.

5. Jaïs P, Maury P, Khairy P, Sacher F, Nault I, Komatsu Y, Hocini M, Forclaz A, Jadidi AS, WeeraSooryia R, Shah A, Derval N, Cochet H, Knecht S, Miyazaki S, Linton N, Rivard L, Wright M, Wilton S, Scherr D, Pascale P, Roten L, Pedersen M, Bordachar P, Laurent F, Kim SJ, Ritter P, Clémenty J, Haïssaguerre M. Elimination of Local Abnormal Ventricular Activities: A New End Point for Substrate Modification in Patients with Scar-Related Ventricular Tachycardia. *Circulation.* 2012;125:2184-2196.

6. Arenal A, del Castillo S, Gonzalez-Torrecilla E, Atienza F, Ortiz M, Jimenez J, Puchol A, García J, Almendral J. Tachycardia-related channel in the scar tissue in patients with sustained monomorphic ventricular tachycardias: influence of the voltage scar definition. *Circulation.* 2004;110:2568-2574.

7. Berruezo A, Fernández-Armenta J, Mont L, Zeljko H, Andreu D, Herczku C, Boussy T, Tolosana JM, Arbelo E, Brugada J. Combined endocardial and epicardial catheter ablation in arrhythmogenic right ventricular dysplasia incorporating scar dechanneling technique. *Circ Arrhythm Electrophysiol.* 2012;5:111-121.

8. Fernández-Armenta J, Berruezo A, Ortiz-Pérez JT, Mont L, Andreu D, Herczku C, Boussy T, Brugada J. Improving safety of epicardial ventricular tachycardia ablation using the scar dechanneling technique and the integration of anatomy, scar components, and coronary arteries into the navigation system. *Circulation.* 2012;125:e466-e468.

9. Fernández-Armenta J, Andreu D, Penela D, Trucco E, Cipolletta L, Arbelo E, Berne P, Tolosana JM, Pedrote A, Brugada J, Mont L, Berruezo A. Sinus rhythm detection of conducting channels and ventricular tachycardia isthmus in arrhythmogenic right ventricular cardiomyopathy. *Heart Rhythm.* 2014;11:747-754.

10. Tung R, Mathuria NS, Nagel R, Mandapati R, Buch EF, Bradfield JS, Vaseghi M, Boyle NG, Shivkumar K. Impact of local ablation on interconnected channels within ventricular scar: mechanistic implications for substrate modification. *Circ Arrhythm Electrophysiol.* 2013;6:1131-1138.

11. Zeppenfeld K, Tops LF, Bax JJ, Schalij MJ. Epicardial radiofrequency catheter ablation of ventricular tachycardia in the vicinity of coronary arteries is facilitated by fusion of 3-dimensional electroanatomical mapping with multislice computed tomography. *Circulation.* 2006;114:e-511-e512.

12. Berruezo A, Mont L, Nava S, Chueca E, Bartholomay E, Brugada J. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. *Circulation.* 2004;109:1842-1847.

13. Vallès E, Bazan V, Marchlinski FE. ECG criteria to identify epicardial ventricular tachycardia in nonischemic cardiomyopathy. *Circ Arrhythm Electrophysiol.* 2010;3:63-71.

14. Fernández-Armenta J, Berruezo A, Andreu D, Cámara O, Silva E, Serra L, Barbarito V, Carotenutto L, Evertz R, Ortiz-Pérez JT, De Caralt TM, Perea RJ, Sitges M, Mont L, Frangi A, Brugada J. Three-dimensional architecture of scar and conducting channels based on high resolution CE-CMR. Insights for ventricular tachycardia ablation. *Circ Arrhythm Electrophysiol.* 2013;6:528-537.

15. Arenal A, Glez-Torrecilla E, Ortiz M, Villacastin J, Fdez-Portales J, Sousa E, del Castillo S, Perez de Isla L, Jimenez J, Almendral J. Ablation of electrograms with an isolated, delayed

component as treatment of unmappable monomorphic ventricular tachycardias in patients with structural heart disease. *J Am Coll Cardiol.* 2003;41:81-92.

16. Bogun F, Good E, Reich S, Elmouchi D, Igic P, Lemola K, Tschopp D, Jongnarangsin K, Oral H, Chugh A, Pelosi F, Morady F. Isolated potentials during sinus rhythm and pace-mapping within scars as guides for ablation of post-infarction ventricular tachycardia. *J Am Coll Cardiol.* 2006;47:2013-2019.

17. Komatsu Y, Daly M, Sacher F, Derval N, Pascale P, Roten L, Scherr D, Jadidi A, Ramoul K, Denis A, Jesel L, Zellerhoff S, Lim HS, Shah A, Cochet H, Hocini M, Haïssaguerre M, Jaïs P. Electrophysiologic characterization of local abnormal ventricular activities in postinfarction ventricular tachycardia with respect to their anatomic location. *Heart Rhythm*. 2013;10:1630-1637.

18. Andreu D, Berruezo A, Ortiz-Pérez JT, Silva E, Mont L, Borràs R, de Caralt TM, Perea RJ, Fernández-Armenta J, Zeljko H, Brugada J. Integration of 3D electroanatomic maps and magnetic resonance scar characterization into the navigation system to guide ventricular tachycardia ablation. *Circ Arrhythm Electrophysiol.* 2011;5:674-683.

19. Marchlinski FE, Callans DJ, Gottlieb CD, Zado E. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. *Circulation.* 2000;101:1288-1296.

20. Carbuciccio C, Ahmad Raja N, Di Biase L, Volpe V, Dello Russo A, Trivedi C, Bartoletti S, Zucchetti M, Casella M, Russo E, Santangeli P, Moltrasio M, Tundo F, Fassini G, Natale A, Tondo C. High-density substrate-guided ventricular tachycardia ablation: role of activation mapping in an attempt to improve procedural effectiveness. *Heart Rhythm*. 2013; 10:1850-1858.

21. Reddy VY, Neuzil P, Taborsky M, Ruskin JN. Short-term results of substrate mapping and radiofrequency ablation of ischemic ventricular tachycardia using a saline-irrigated catheter. *J Am Coll Cardiol*. 2003;41:2228-2236.

22. Andreu D, Ortiz-Pérez JT, Boussy T, Fernández-Armenta J, de Caralt TM, Perea RJ, Prat-González S, Mont L, Brugada J, Berruezo A. Usefulness of contrast-enhanced cardiac magnetic resonance in identifying the ventricular arrhythmia substrate and the approach needed for ablation. *Eur Heart J.* 2014;35:1316-1326.

23. Klem I, Weinsaft JW, Bahnson TD, Hegland D, Kim HW, Hayes B, Parker MA, Judd RM, Kim RJ. Assessment of myocardial scarring improves risk stratification in patients evaluated for cardiac defibrillator implantation. *J Am Coll Cardiol*. 2012;60:408-420.

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Table 1: Baseline clinical characteristics of the patient population

	All (n=101)	CAD (n=75)	Non-ischemic (n=26)	*P*
Age, years	65±12	67±11	61 ±13	0.037
Men, n (%)	92 (91.1%)	67 (89.3%)	25 (96.2%)	0.440
Hypertension, n (%)	75 (74.3%)	62 (83.8%)	13 (50.0%)	0.001
Diabetes, n (%)	23 (22.8%)	21 (28.4%)	2 (7.7%)	0.033
LVEF, %	36±13	35±13	41±13	0.032
ICD, n (%)	82 (81.2%)	64 (85.3%)	18 (69.2%)	0.085
AAD, n (%)				
β-blockers	81 (80.2%)	64 (85.3%)	17 (65.4%)	<0.001
Class I	8 (7.9%)	5 (6.7%)	3 (11.5%)	0.482
Class III	55 (54.5%)	42 (56.0%)	13 (50.0%)	0.597
Number of VT episodes/patient	4 (1-19)	5 (1-25)	3 (1-6)	0.339
Arrhythmic Storm, n (%)	28 (27.7%)	19 (25.3%)	9 (34.6%)	0.362
Clinical VT CL, ms	358±80	365±82	341±73	0.233

AAD indicates anti-arrhythmic drugs; CL, cycle length; ICD, implantable cardioverter defibrillator; LVEF, left ventricular ejection fraction; VT, ventricular tachycardia

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Table 2: Baseline clinical characteristics and substrate mapping and ablation data depending on the achievement of complete scar dechanneling

	All (n=101)	Complete SD (n=85)	Incomplete SD (n=16)	*P*
Age, years	65±12	66±11	63 ±13	0.348
Hypertension, n (%)	75 (74.3%)	64 (75.3%)	11 (73.3%)	1.000
LVEF, %	36±13	37±13	36±11	0.941
ICD, n (%)	82 (81.2%)	68 (80.0%)	14 (87.5%)	0.730
AAD, n (%)				
β-blockers	81 (80.2%)	67 (78.8%)	14 (87.5%)	0.424
Class I	8 (7.9%)	5 (5.9%)	3 (18.8%)	0.080
Class III	55 (54.5%)	46 (54.1%)	9 (56.2%)	0.875
Number of VT episodes/patient	4 (1-19)	3.5 (1-16)	4 (2-35)	0.335
Arrhythmic Storm, n(%)	28 (27.7%)	23 (27.1%)	5 (31.2%)	0.477
Clinical VT CL, ms	358±80	359±82	355±65	0.907
Procedure time, min	227±69	224±20	239±62	0.467
Fluoroscopy time, min	18±8	18±9	18±7	0.778
Endo Scar area <1.5 mV, cm2	52±39	51±37	52±51	0.948
Epicardial mapping, n(%)	27(26.7%)	23(27.1%)	4(25.0%)	1.000
Number of CC-EG/patient*	52(29-86)	55(30-84)	43(21-86)	0.798
Number of CC entrances-EG /patient*	10(5-15)	10(5-15)	11(4-16)	0.841
Radiofrequency ablation time, min	28±16	26±11	43±28	0.126
SMVT inducible after scar dechanneling, n (%)	46(45.5%)	34(40.0%)	12(75.0%)	0.013
Residual VT mean CL, ms	340±85	335±84	356±92	0.540
Acute success†, n (%)	79(78.2%)	72(84.7%)	7(43.8%)	0.001

* Sum of endo and epicardial CC-EG
Non-normally distributed variables are presented as median and interquartile range (25th, 75th percentile)
† Non-inducibility of any SMVT at the end of the procedure.
CC-EG indicates conducting channel electrogram; CC entrance-EG indicates conducting channel entrance electrogram (the CC-EG with the shortest local activation time of the near-field); Endo, endocardial; Epi, epicardial; RF, radiofrequency; SMVT, sustained monomorphic ventricular tachycardia; VT, ventricular tachycardia.

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Table 3: Conducting channel electrogram characteristics, depending on the endocardial vs epicardial location and septal vs lateral position at the scar edge. Voltage and delay values shown refer to local (near-field) electrogram.

	Endocardial	Epicardial	*P*	Septal*	Lateral*	*P*
Entrance CC-EG amplitude (mV)	0.38 (0.16-0.44)	0.74 (0.38-1.52)	0.028	0.54 (0.25-0.97)	0.48 (0.29-0.71)	0.289
QRS onset to Entrance CC-EG delay (ms)	87 (71-117)	107 (80-116)	0.612	87 (71-106)	108 (98-145)	0.004
Inner CC-EG amplitude (mV)	0.23 (0.13-0.48)	0.49 (0.23-0.87)	0.085			
QRS onset to latest Inner CC-EG delay (ms)	156 (113-183)	140 (97-177)	0.707			

* Endocardial EGMs
CC-EG indicates conducting channel electrogram

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Table 4: Univariate and multivariable Cox regression analysis for the association between clinical and procedure-related variables to the study endpoint (VT recurrence or sudden cardiac death)

	Univariate HR (95%CI)	***P***	**Multivariable HR (95%CI)**	
Age	1.02(0.99-1.06)	0.177		
CAD	0.59(0.27-1.29)	0.190		
LVEF	0.99(0.97-1.03)	0.910		
Heart failure	1.66(0.72-3.79)	0.23		
ICD	1.39(0.48-4.04)	0.541		
AAD				
Class I	1.83(0.63-5.30)	0.267		
Class III	1.24(0.57-2.67)	0.590		
Arrhythmic Storm/Incessant VT	1.59(0.73-3.47)	0.290		
Clinical VT CL	0.99(0.99-1.00)	0.788		
VT morphologies induced	1.14(0.94-1.39)	0.193		
Induced VT CL	1.00(0.99-1.00)	0.870		
VT inducibility after scar dechanneling	**2.58(1.18-5.67)**	**0.018**	2.17(0.92-5.12)	0.076
End-procedure VT inducibility	**2.59(1.18-5.68)**	**0.018**	1.29(0.47-3.34)	0.615
Endo Scar area <1,5 mV (cm^2)	1.00(0.99-1.01)	0.533		
Epicardial mapping	**0.16(0.05-0.57)**	**0.004**	0.56(0.21-1.50)	0.251
Number of CC-EG	1.00(1.00-1.01)	0.405		
Number of CC entrance-EG	1.02(0.98-1.07)	0.277		
Radiofrequency ablation time	1.00(1.00-1.00)	0.397		
Number of CC-EG (remap)	1.00(0.97-1.02)	0.698		
Incomplete CC-EG elimination	**3.50(1.55-7.87)**	**0.003**	**2.54(1.06-6.10)**	**0.037**

AAD indicates anti-arrhythmic drugs; CAD, coronary artery disease; CC-EG, conducting channel electrogram; CC entrance-EG, conducting channel entrance electrogram (short local activation time of the near-field); CL, cycle length; ICD, implantable cardioverter defibrillator; LVEF, left ventricular ejection fraction; VT, ventricular tachycardia.

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Table 5: Comparison of baseline clinical variables and procedure-related data between patients needing only scar dechanneling to become non-inducible and patients needing residual inducible VT ablation.

	Scar dechanneling (n=55)	**Scar dechanneling +VT ablation (n=46)**	***P***
Age, years	65±12	65±12	0.846
CAD, n (%)	42(76.4%)	33(71.7%)	0.597
LVEF, %	38±13	35±13	0.198
Number of VT episodes/patient	3(1-6)	6(1-25)	0.082
Arrhythmic Storm, n(%)	10(18.2%)	18(39.1%)	**0.019**
Clinical VT CL, ms	354±78	363±83	0.640
Epicardial approach, n (%)	11(20.0%)	16(34.8%)	0.095
Fluoroscopy time (minutes)	16.8±8.5	19±8.1	0.208
Procedure time	213±64	244±71	**0.027**
Direct current cardioversion/defibrillation	11(20%)	30(65.2%)	**<0.001**
Number of CC-EG	53±35	74±50	**0.025**
Scar area endo (cm2)	48±34	56±45	0.377
RF time (minutes)	24±10	31±18	0.086
RF applications	26±14	33±20	**0.006**
RF applications (remap)	7±6	15±9	**<0.001**
Incomplete CC-EG elimination	4(7.3%)	12(26.1%)	**0.01**
Primary endpoint	10(18.2%)	17(37.0%)	**0.013**
Mortality	2(3.6%)	7(15.2%)	**0.013**

CAD indicates coronary artery disease; LVEF, left ventricular ejection fraction; VT CL, ventricular tachycardia cycle length; CC-EG, conducting channel electrogram; RF, radiofrequency

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Figure Legends:

Figure 1: Ventricular tachycardia catheter ablation procedure workflow (left panel) and schematic representation of scar, conducting channels (CC) and characteristic electrograms with delayed components marking the CC entrance (red dots) and inner part.

Figure 2: Substrate characterization for scar dechanneling. Posterior view of the epicardial bipolar voltage electroanatomic map from a patient with non-ischemic cardiomyopathy. Normal myocardium (voltage >1.5 mV) is coded in purple, dense scar (<0.5 mV) in red, and border zone (0.5-1.5 mV) in blue-yellow-green. Electrograms with delayed components were classified as entrance (black dots, 1 and 5) or inner (blue dots, 2, 3, 4) conducting channel points, depending on delayed-component lateness during sinus rhythm. Electrograms within a submitral conducting channel are shown (1-5). Only electrograms 1 and 5 were targeted for ablation (red dots). MA, mitral annulus; RV, magnetic-resonance-imaging-derived right ventricle.

Figure 3: Recordings showing the 3 types of response observed during radiofrequency (RF) application (in sinus rhythm) at the conducting channel (CC) entrance when conduction block was obtained. Left panel (A) shows the surface ECG leads (I, II, III, aVR, aVL, aVF) and intracardiac bipolar signals from the ablation catheter from the distal (d-2, placed at a CC-entrance into the scar, see text) to the proximal (3-4) dipoles. When RF application started (RFon), CC activation sequence of the delayed electrograms went from the distal to the proximal dipole. After a few seconds, a reversed order of the activation sequence was observed due to a conduction block at the CC-entrance. Right upper panel (B) shows surface ECG leads (aVL and

aVF) and intracardiac bipolar signals from the right ventricular apex catheter (RV) and distal dipole of the ablation catheter (Map) placed inside the scar area over a CC path. The recording was obtained after RF application at the CC-entrance. Automaticity in the CC path with exit block to the normal myocardium is observed. Right lower panel (C) shows the surface ECG lead (aVF) and intracardiac bipolar signals from the ablation catheter from the distal (d-2, placed at the CC-entrance into the scar) to the proximal (3-4) dipoles. When RF application started (RFon), a CC activation sequence of the delayed electrograms from the distal to the proximal dipole was observed. Seconds after RF application started (RFon), the inner CC electrogram disappeared (*).

Figure 4: Inferior view of bipolar voltage electroanatomic substrate maps during sinus rhythm before (MAP) and after (reMAP) scar dechanneling in a patient with healed myocardial infarction. Electrograms recorded as conducting channel entrances are labeled with black dots, inner sites with blue dots. Examples of bipolar electrograms at entrances (1 and 5) and inner parts (2-4) are shown in the left panel. Delayed components of the electrograms are highlighted with arrows. Electrogram aspect after elimination of the delayed component (asterisks) in the same sites after scar dechanneling is shown in the left panel.

Figure 5: Example of a patient with an old inferior infarction with aneurysm. A high-density map was obtained, with mapping focused on the area of interest. Standard voltage thresholds were used to identify the scar area. Electrograms with delayed components were tagged in black when they had the appearance of a conducting channel entrance (delayed component with the shortest delay, usually fused with the far field electrogram) and in blue when they had the

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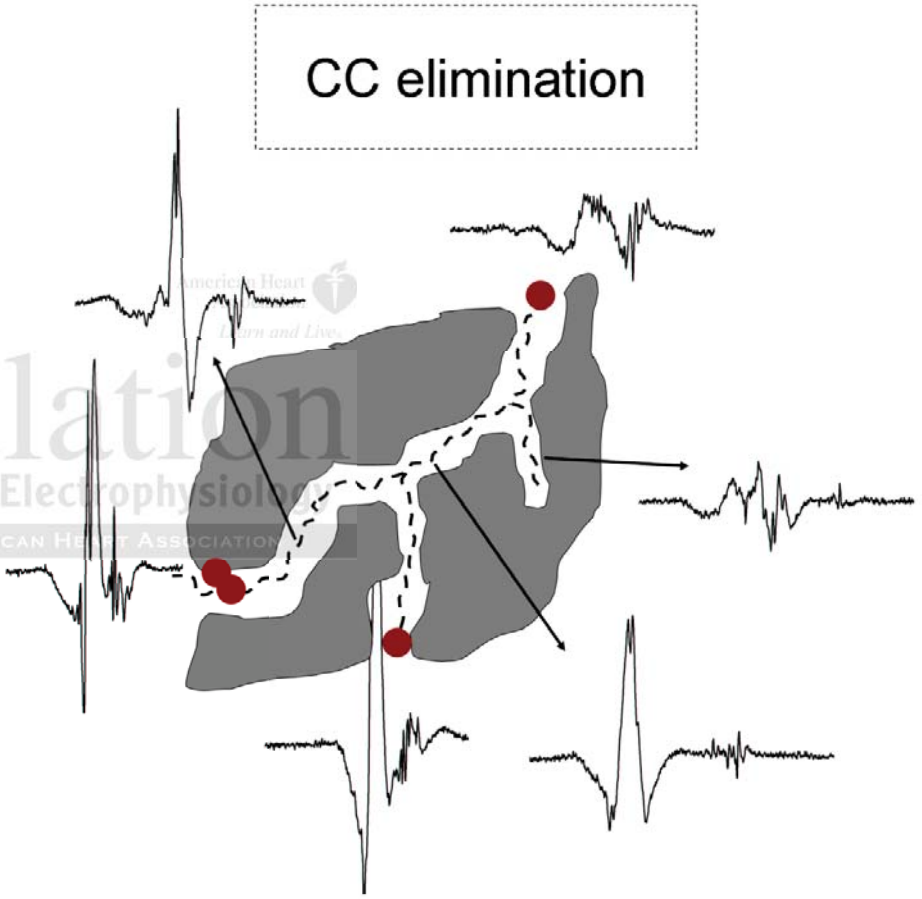
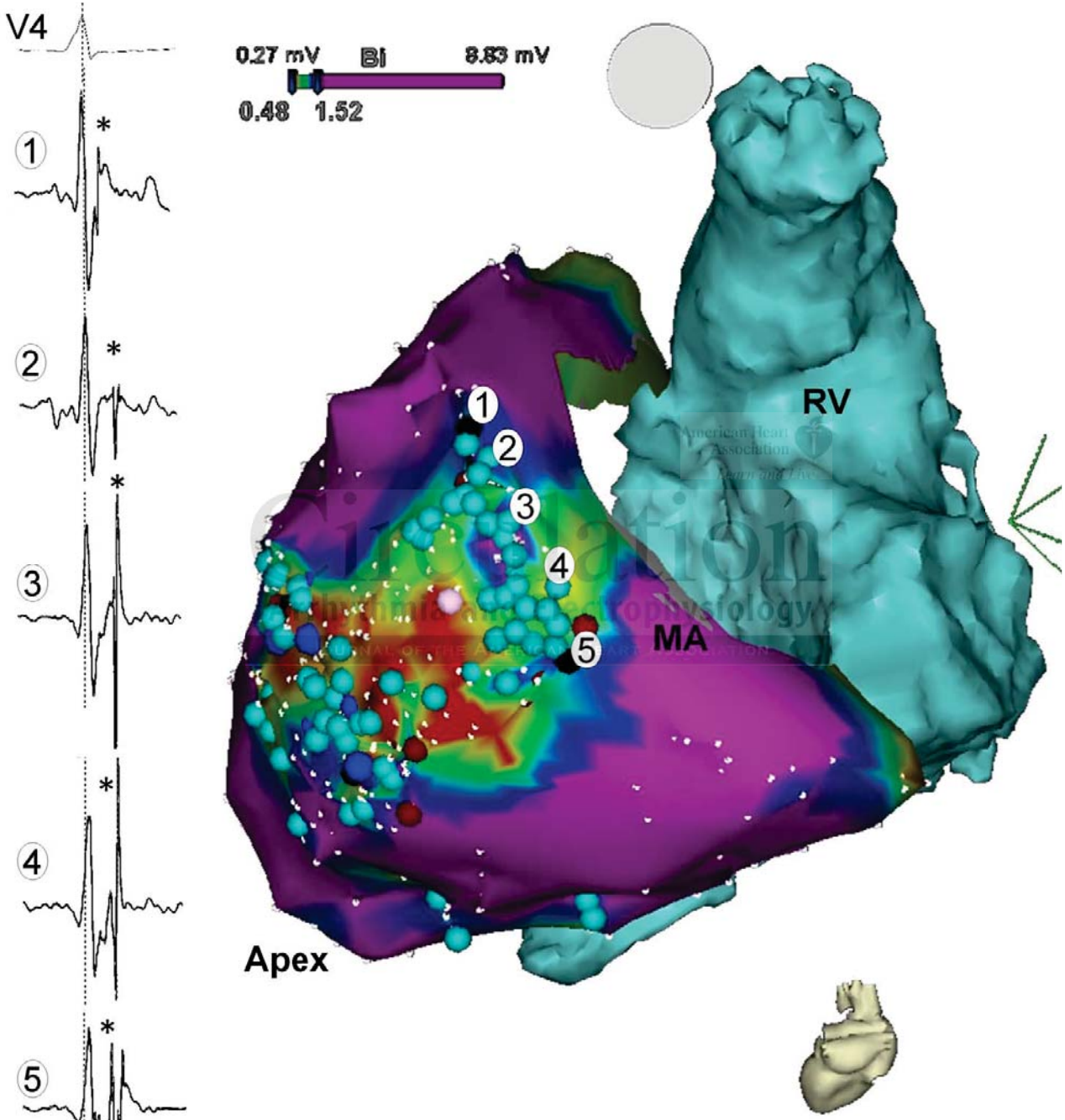
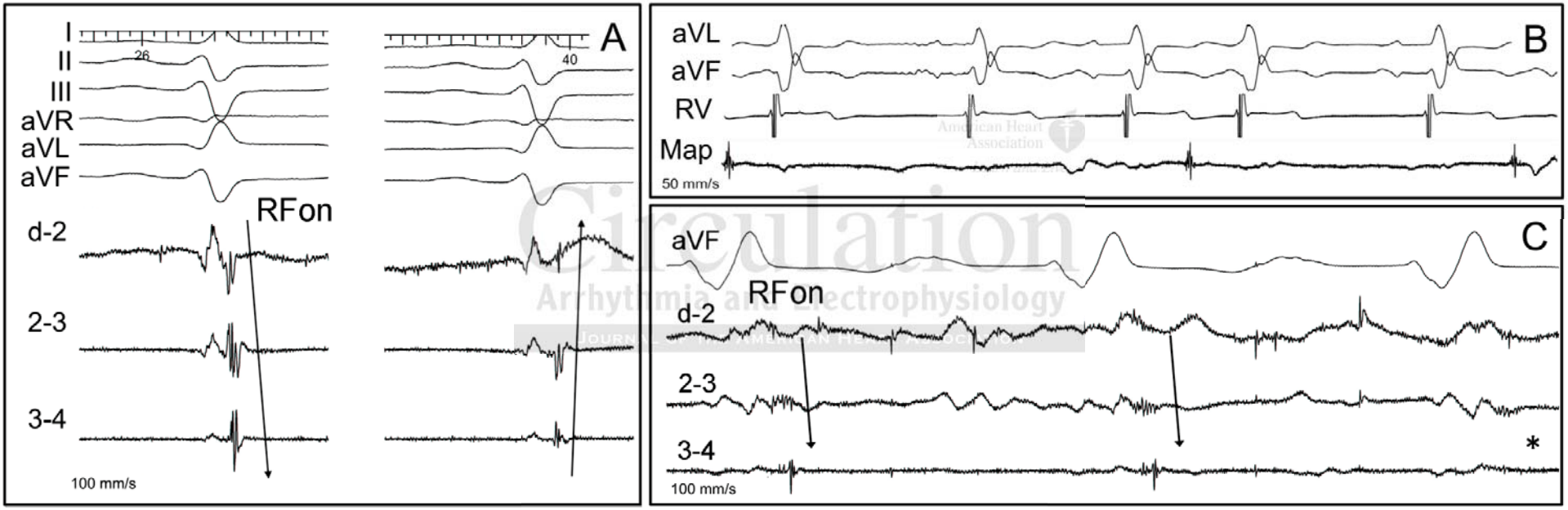
appearance of an inner conducting channel part (delayed component with longer delays with respect to the far-field electrogram). The relationship to the beginning/end of the QRS-complex is independent of identifying a conducting channel entrance vs inner point, which depends mainly on septal vs lateral location in the ventricle during sinus rhythm. In the example, local electrograms of lateral entrance (left) are inscribed at the end of the QRS while at septal entrances (right) local electrograms are inside the QRS. Inner conducting channel electrograms recorded inside the scar are shown at the bottom.

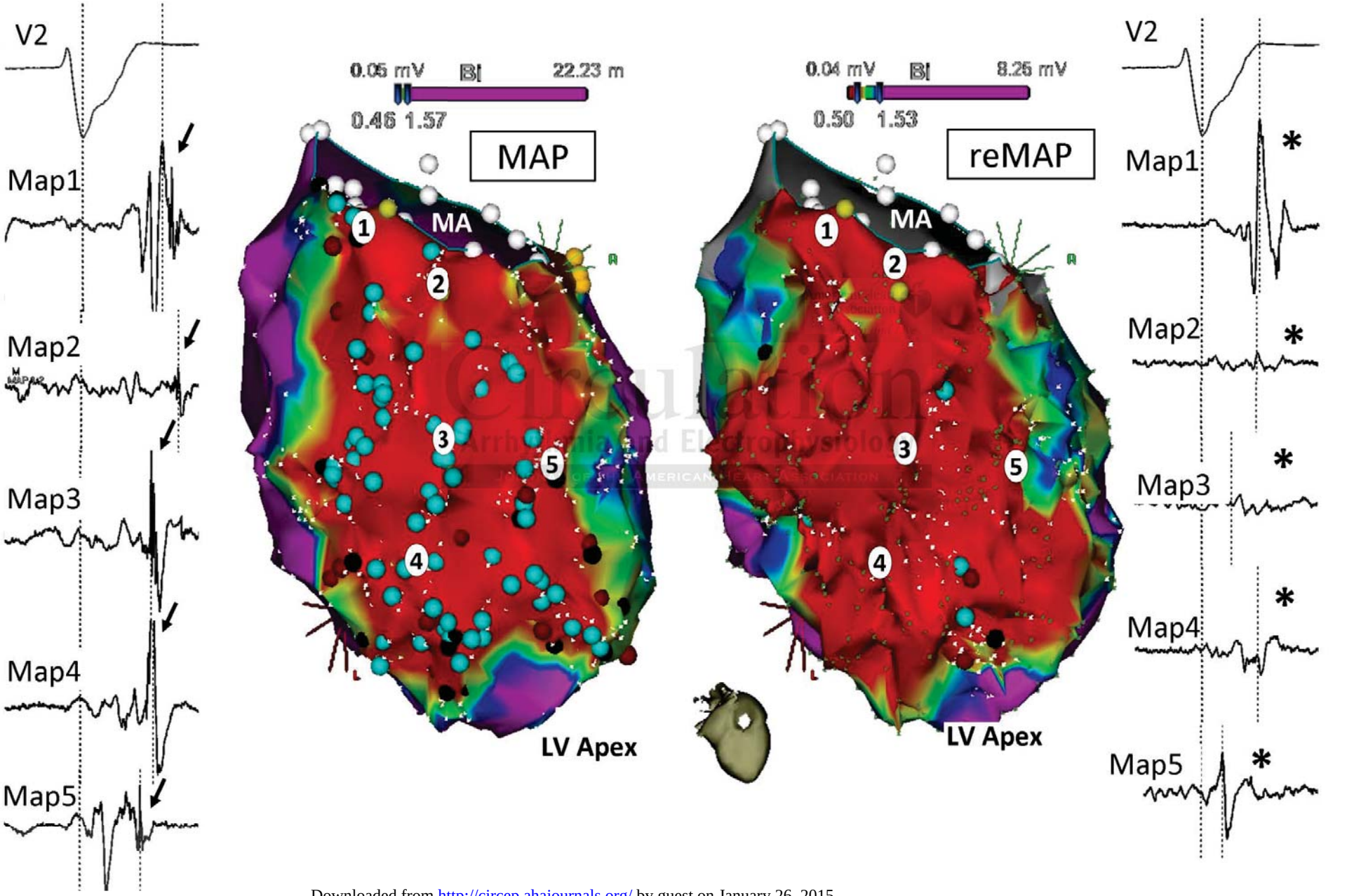
Figure 6: Kaplan-Meier Curve for the primary endpoint of any sustained ventricular arrhythmia or sudden cardiac death (superior panels) and all-cause mortality (inferior panels) according to the complete/incomplete elimination of conducting channel electrograms (left) and the need for residual inducible VT ablation (right). SD, scar dechanneling; VT, ventricular tachycardia.

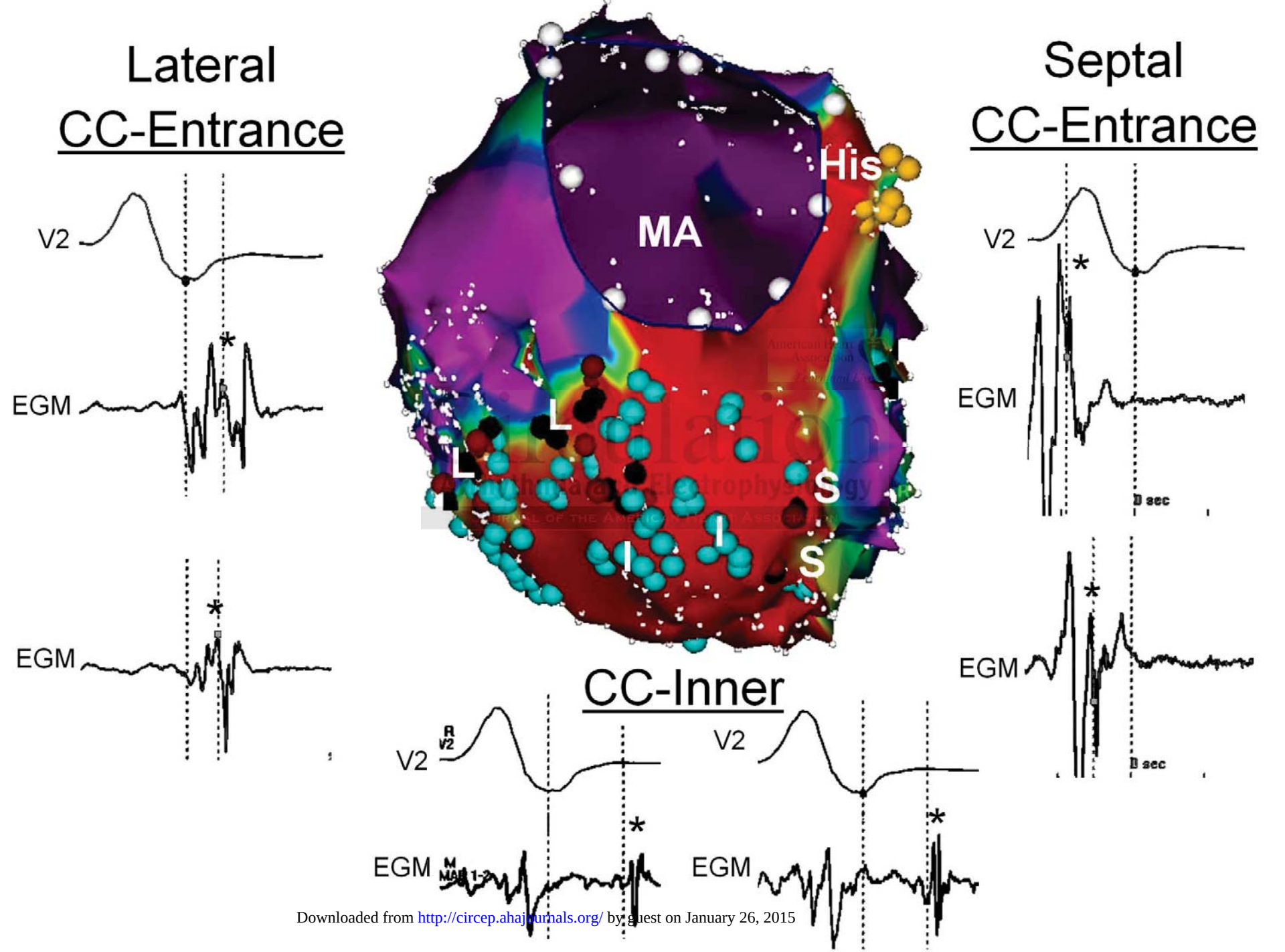
Steps	VT Ablation Procedure
1	Substrate mapping CC entrance identification
2	CC elimination
3	Re-mapping Residual CC ablation
4	Inducibility Residual VT ablation

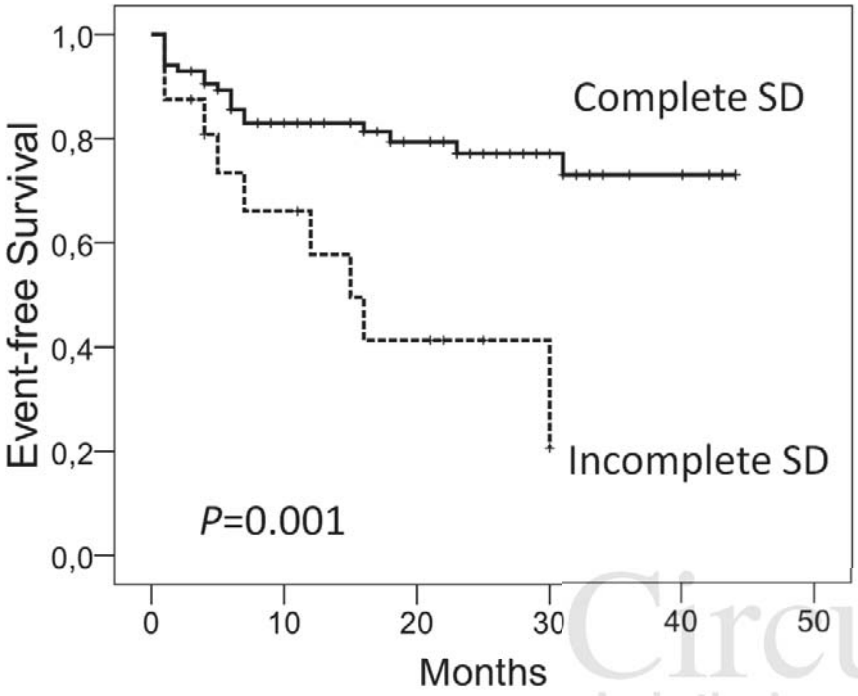


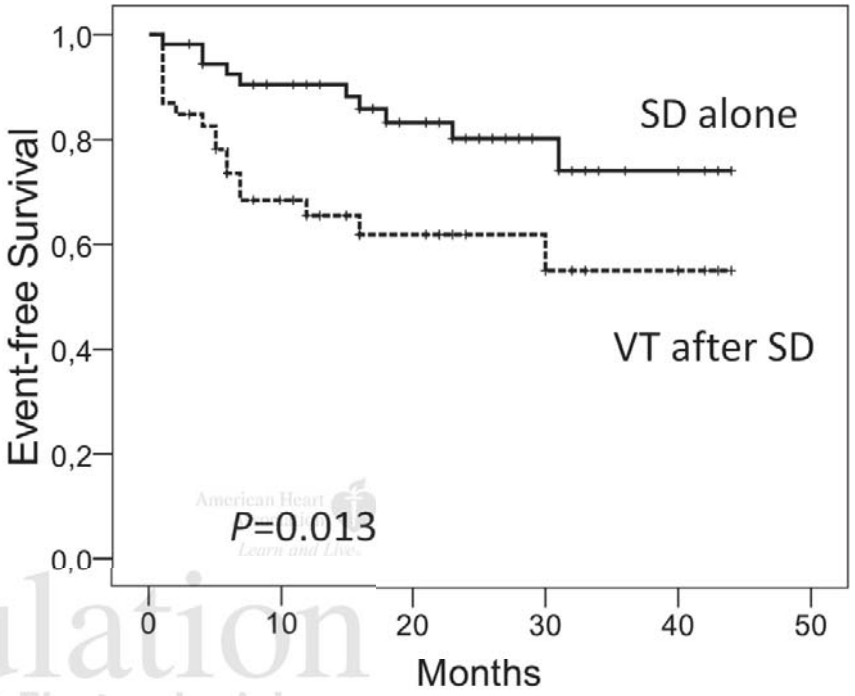


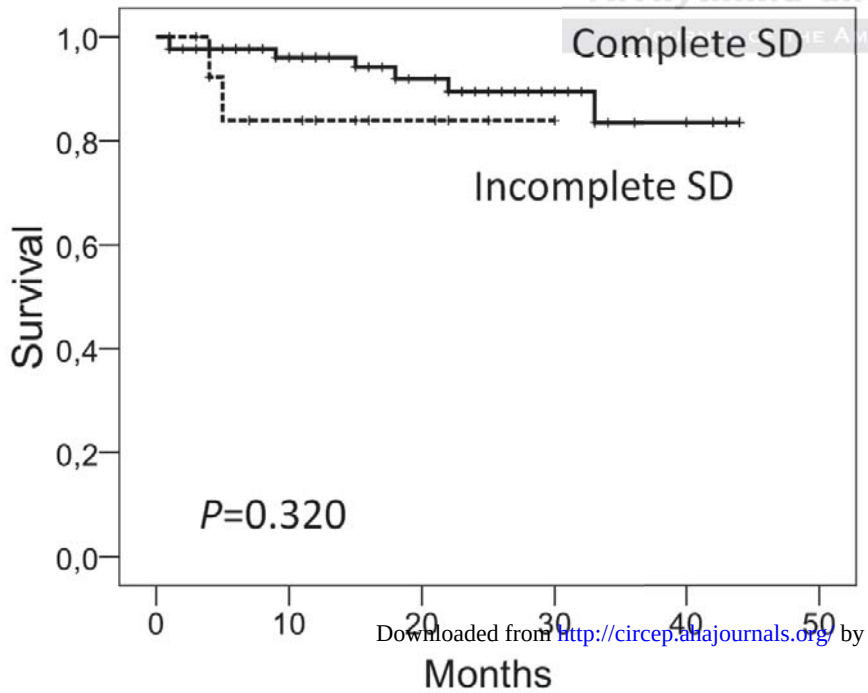


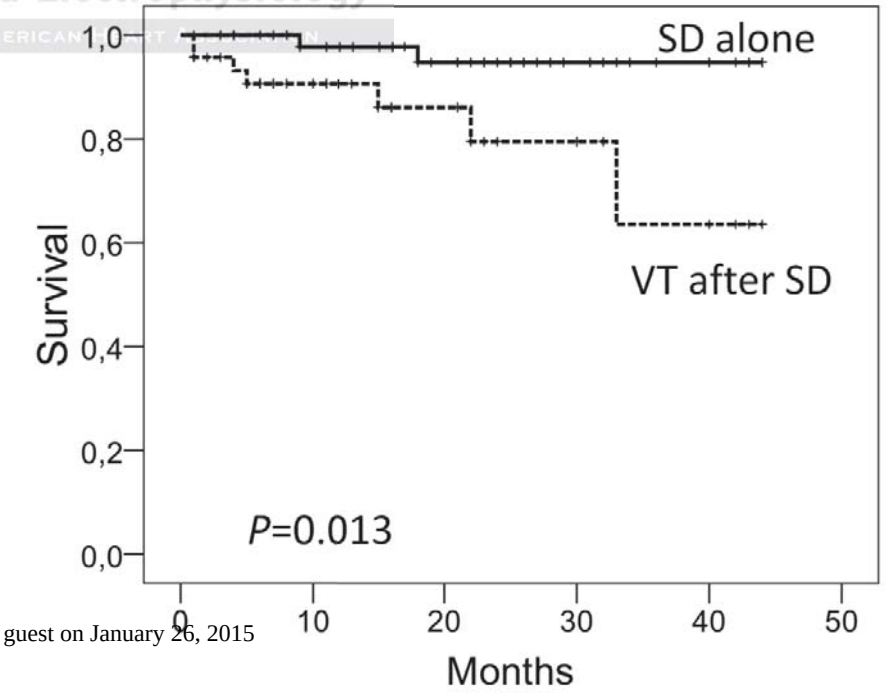












Scar Dechanneling: A New Method for Scar-Related Left Ventricular Tachycardia Substrate Ablation

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