Dilated cardiomyopathy (DCM) accounts for one fifth of patients undergoing catheter ablation for drug-refractory ventricular tachycardia (VT) because of structural heart disease, with half recurring at 1 to 2 years.\(^1\)\(^-\)\(^4\) Discrete arrhythmogenic scar patterns have been described in nonischemic cardiomyopathy (NICM).\(^5\)\(^-\)\(^6\) Isolated septal substrate was reported as technically challenging for ablation because of intramural substrate leading to high redo procedure rates.\(^3\) Based on MRI, 2 discrete scar patterns, anteroseptal and inferolateral, were recently described in 19 patients with NICM undergoing VT ablation.\(^6\) The arrhythmogenic substrate is frequently intramural or epicardial in NICM as evidenced by both electroanatomic and cardiac MRI studies.\(^3\)\(^-\)\(^7\) High VT recurrence rates are partly because of the intramural disease, making scar identification and late potential (LP) mapping more challenging.\(^1\)\(^-\)\(^4\) Unipolar voltage mapping, because of the wider field of view, is thus an essential tool in this setting.\(^9\)\(^-\)\(^10\)

**Background**—The aim was to relate distinct scar distributions found in nonischemic cardiomyopathy with ventricular tachycardia (VT) morphology, late potential distribution, ablation strategy, and outcome.

**Methods and Results**—Eighty-seven patients underwent catheter ablation for drug-refractory VT. Based on endocardial unipolar voltage, 44 were classified as predominantly anteroseptal and 43 as inferolateral. Anteroseptal patients more frequently fulfilled diagnostic criteria for dilated cardiomyopathy (64% versus 36%), associated with more extensive endocardial unipolar scar (41 [22–83] versus 9 [1–29] cm\(^2\); \(P<0.001\)). Left inferior VT axis was predictive of anteroseptal scar (positive predictive value, 100%) and right superior axis for inferolateral (positive predictive value, 89%). Late potentials were infrequent in the anteroseptal group (11% versus 74%; \(P<0.001\)). Epicardial late potentials were common in the inferolateral group (81% versus 4%; \(P<0.001\)) and correlated with VT termination sites (\(k=0.667\); \(P=0.014\)), whereas no anteroseptal patient had an epicardial VT termination (\(P<0.001\)). VT recurred in 44 patients (51%) during a median follow-up of 1.5 years. Anteroseptal scar was associated with higher VT recurrence (74% versus 25%; log-rank \(P<0.001\)) and redo procedure rates (59% versus 7%; log-rank \(P<0.001\)). After multivariable analysis, clinical predictors of VT recurrence were electrical storm (hazard ratio, 3.211; \(P=0.001\)) and New York Heart Association class (hazard ratio, 1.608; \(P=0.018\)); the only procedural predictor of VT recurrence was anteroseptal scar pattern (hazard ratio, 5.547; \(P<0.001\)).

**Conclusions**—Unipolar low-voltage distribution in nonischemic cardiomyopathy allows categorization of scar pattern as inferolateral, often requiring epicardial ablation mainly based on late potentials, and anteroseptal, which frequently involves an intramural septal substrate, leading to a higher VT recurrence. (Circ Arrhythm Electrophysiol. 2014;7:414-423.)

**Key Words:** cardiomyopathies \(\sqcap\) tachycardia, ventricular
Methods
This is a retrospective single-center study. Three hundred and forty-five consecutive patients with structural heart disease and drug-refractory VT underwent catheter mapping and ablation at the Ventricular Tachycardia Unit of San Raffaele University Hospital, Milan, between January 2010 and December 2012. We sought to examine patients with DCM using electroanatomic mapping (EAM) data obtained by the Carto 3 system (Biosense-Webster, Diamond Bar, CA) because of the uniform availability of both contact bipolar and unipolar electrogram voltage data. Patients with coronary artery disease (≥75% stenosis) or congenital, hypertensive, or valvular heart disease were excluded (n=187). Of 158 patients with a primary cardiomyopathy, patients were categorized according to the European Society of Cardiology position statement on cardiomyopathy classification. An additional 42 patients were excluded because of arrhythmogenic right ventricular cardiomyopathy, restrictive cardiomyopathy, sarcoidosis, hypertrophic cardiomyopathy, and left ventricular (LV) noncompaction. Of the remaining 116 patients, 87 underwent a Carto 3 procedure and were included. Within this study group, 59 patients fulfilled conventional diagnostic criteria for DCM (ejection fraction <45% and moderate or severe LV dilatation). The remaining 28 patients were termed early cardiomyopathy (ECM) with well-defined scar and normal or mildly impaired LV function and dimensions.

Arrhythmia presentation was classified as either electrical storm, incessant, or paroxysmal VT. Subxiphoid epicardial access was standard unless clinical or prior procedural data were indicative of an endocardial substrate and double LV endocardial access (retrograde aortic and transseptal) routine. Procedures were performed under general anesthesia with inotropic support if needed. The study was approved by an institutional review committee. Written informed consent was obtained for all subjects.

VT Ablation Strategy and Procedure
Patients underwent high-density EAM (defined as a 5-mm EAM interpolation fill threshold in areas with reduced unipolar voltage and 10 mm elsewhere). Bipolar scar and low-voltage area were defined as areas with a bipolar voltage <0.5 and <1.5 mV, and unipolar scar and low voltage area as areas with a unipolar voltage <5 and <8 mV, respectively. Border zone was defined as the area compressed between 0.5 and 1.5 mV for bipolar and between 5 and 8 mV for unipolar voltage. The LP definition included either continuous fragmented activity or isolated potentials recorded after the QRS. Abnormal early potentials (EPs) were defined as fragmented or isolated electrograms inscribed within the QRS, typically recorded at the scar border; pacing was systematically attempted at these sites looking for morphology match with any induced VT and for latency between the stimulus and the QRS (>40 ms). LPs were a primary target, and EPs secondary or adjunctive. Remaps were undertaken post-LP ablation to assess for complete LP abolition with repeat ablation if necessary. EPs were manually tagged as points on the 3-dimensional EAM.

After substrate mapping, programmed ventricular stimulation with ≤4 extrastimuli from the right ventricular apex and multiple LV sites was performed. In patients with hemodynamically tolerated VT, ablation was performed using activation and entrainment mapping; it continued in sinus rhythm, aiming at the complete abolition of LPs when present or EPs when absent. All induced VTs were characterized according to bundle branch block pattern and axis using 12-lead recordings. The substrate modification strategy was also performed in noninducible patients. Radiofrequency (RF) current was delivered with a 3.5-mm open-irrigated catheter with power settings of 30 to 60 W and a temperature limit of 43°C. We aimed to achieve both VT noninducibility and LP abolition in all cases.

EAM Analysis and Scar Categorization
The endocardial LV EAM was initially segmented using the standardized 17-segment model with the aorta, mitral valve annulus, and apex as references. The aorta was defined by a separate aortic geometry and low voltage area as areas with a unipolar voltage <5 and <8 mV, excluding the apex. In patients with a normal endocardial unipolar map (n=6) or only epicardial map (n=5), the same system was then applied epicardially, using 3 anteroseptal and 8 inferolateral segments, excluding the apex.

Follow-Up
Regular outpatient follow-up was scheduled at 3 months and then 6-monthly intervals or whenever any symptoms recurred. In cases of drug-refractory VT recurrence, catheter ablation was repeated. End points were recurrence of sustained ventricular arrhythmia, appropriate implantable cardioverter defibrillator intervention, redo procedures, and overall mortality.

Statistical Analysis
Continuous variables are presented as means±SD or medians (25th and 75th percentiles), and categorical variables as numbers and percentages. Comparisons between groups were undertaken using the t test or nonparametric tests for continuous variables and Fisher exact or χ² tests for proportions as indicated. Agreement between LP and VT termination segments and imaging versus EAM data was assessed using Cohen κ coefficient. Event-free survival was estimated by the Kaplan–Meier method using the log-rank test. Univariable Cox proportional hazards analyses assessed the relationship between background (Table 1) and procedural characteristics (Table 2) with respect to time to VT recurrence and death. All baseline and procedural variables that were significantly (P<0.05) related to VT recurrence were entered into a forward-stepwise multivariable analysis performed separately for the clinical and procedural data. Differences were considered statistically significant at the 2-sided P<0.05 level. All analyses were undertaken using IBM SPSS Statistics version 21 (IBM Corporation).

Results
Eighty-seven patients constituted the study population. Based on unipolar EAM data, 44 had a predominant anteroseptal, and 43 inferolateral scar distribution. Of these, 59 patients fulfilled DCM diagnostic criteria, 38 with predominant anteroseptal (AS-DCM) and 21 with inferolateral scar (IL-DCM). Of the remaining 28, defined as ECM, 6 had predominant anteroseptal scar (AS-ECM), and 22 inferolateral scar (IL-ECM). The background data are shown in Table 1. The IL-ECM versus AS-ECM group had a higher prevalence of previous clinically suspected myocarditis (73% versus 0%; P=0.002), younger age (53 [39–62] versus 72 [69–73] years; P=0.004), and more frequent New York Heart Association class I status (86% versus 33%; P=0.021). Previous suspected myocarditis was also more prevalent with IL-DCM (24%) versus AS-DCM (0%; P=0.004). Left bundle branch block sinus rhythm pattern (50% versus 13%; P=0.003) and prior cardiac resynchronization therapy device implantation (61% versus 19%; P=0.002) were more frequent in AS-DCM as compared with IL-DCM (Figure 1). The follow-up time was 1.5 (1.0–2.3) years with no patients lost to follow-up.

Thirty-five patients underwent delayed-enhanced preprocedural imaging (18 computed tomography; 17 MRI). The agreement for scar location with EAM was high (κ=0.706; P<0.001), entirely concordant with MRI, whereas in 4 patients,
an anteroseptal scar was missed with computed tomography, readily detectable with EAM.

**Segmental EAM Analysis**

A total of 1272/1394 endocardial and 575/768 epicardial segments fulfilled threshold criteria and were analyzed. The unipolar scar distribution was highly localized for inferolateral and anteroseptal scar patterns in patients with ECM and more extended for the DCM groups (Figure 2; Table 2): the endocardial area of unipolar scar (<5 mV) in the 8 anteroseptal segments was 11 (4–30) cm² for AS-ECM versus 0 (0–0) cm² for IL-ECM, and 42 (25–65) cm² for AS-DCM versus 4 (0–13) cm² for IL-DCM (both P<0.001), and in the 8 inferolateral segments was 1 (0–9) cm² for IL-ECM versus 0 (0–2) cm² for AS-ECM and 23 (9–33) cm² for IL-DCM versus 6 (0–18) cm² (P=0.01 and 0.014, respectively).

In all groups, the endocardial scar had a basal predominance when comparing the 3 basal segments with the 5 mid and apical segments in each group. In anteroseptal patients, 91% of anteroseptal basal segments displayed unipolar low-voltage area versus 52% of mid and apical segments (P<0.001).

Among inferolateral patients, 55% of the basal inferolateral
segments versus 26% of the mid and apical segments showed unipolar low-voltage area ($P<$0.001). Full perimital involvement with all 6 basal segments displaying unipolar low voltage was present in 18 patients (21%), 14 anteroseptal versus 4 inferolateral patients ($P=0.010$).

**VT Mapping Procedure**

The overall epicardial procedure rate was 74% higher in the inferolateral group (84% versus 64%; $P=0.034$). Five patients had a failed percutaneous epicardial access attempt with 2 converted to a surgical approach: 1 lateral minithoracotomy and 1 subxiphoid surgical window.

Bipolar scar was absent in both ECM groups (AS-ECM $0 \ [0–3]$ and IL-ECM $0 \ [0–0]$ cm²; $P=0.32$). It was present in 23 patients corresponding to AS-DCM ($5 \ [2–12]$ cm²) and 9 to IL-DCM ($3 \ [2–13]$ cm²; $P=0.010$). Endocardial unipolar scar ($<5$ mV) was significantly greater in the epicardium (AS-ECM $38 \ [28–46]$ cm² versus IL-ECM $0 \ [0–0]$ cm²; $P<0.001$) and in AS-DCM ($45 \ [27–85]$ versus IL-DCM $28 \ [9–49]$ cm²; $P=0.022$). Conversely, the epicardial unipolar scar was greater in IL-ECM ($4 \ [4–30]$ cm²) versus AS-ECM ($0 \ [0–0]$ cm²; $P<0.001$) and in IL-DCM ($36 \ [21–97]$ cm²) versus AS-DCM ($15 \ [5–23]$ cm²; $P=0.003$), also significant for bipolar epicardial scar ($13 \ [2–31]$ versus $1 \ [0–6]$ cm²; $P=0.004$).

LPs were more frequent in the inferolateral group (74% versus 11%; $P<0.001$), especially in the epicardium (81% [29/36 inferolateral] versus 4% [1/28 anteroseptal]; $P<0.001$) and more commonly identified in segments with reduced unipolar voltage (51% unipolar scar, 28% border zone, and 21% normal unipolar voltage) than within bipolar scar (11% bipolar scar, 48% border zone, 41% normal bipolar voltage). In the anteroseptal group, late activity was rare (11%), located endocardially (80%) and of lower amplitude (87% bipolar scar, 13% border zone; $P=0.005$; Figure 3). We observed significantly greater areas of bipolar scar in anteroseptal patients with LPS than in those without (16 [8–23] versus 0 [0–4] cm²; $P=0.007$).

**Acute Ablation Results**

Within the anteroseptal group, 20 patients (45%) underwent VT mapping and ablation, 19 patients underwent VT mapping and ablation and electrogram-guided ablation, and in the remaining 5 patients who were either noninducible ($n=3$) or had nontolerated VT ($n=2$), a substrate ablation was performed (4 LP, 20 EP). Of 104 induced VTs, 55 were terminated during RF delivery (Figure 4). Thirty-one (56%) were terminated in the LV basal anterior and basal anteroseptal segments, 3 (5%) in inferolateral segments (in patients with perimital...
extension), 2 (4%) in the aortic cusps, and 5 (9%) in the right ventricular septum. Epicardial LPs were not completely abolished in the only patient with epicardial LPs because of left anterior descending artery proximity. Thirteen patients (30%) remained inducible at the end of the procedure (10 nonclinical monomorphic VT, 3 clinical VT).

Within the inferolateral group, 7 patients (16%) underwent VT mapping and ablation, 21 patients underwent VT mapping and ablation and electrogram-guided ablation, and in 15 patients who were noninducible at baseline, the modification of the substrate was performed in sinus rhythm (33 LP and 3 EP). Of 53 induced VTs, 27 were terminated during LV ablation (14 endocardial and 13 epicardial; Figure 4). Epicardial LPs were successfully abolished in 15 inferolateral patients, whereas in 6 patients, phrenic nerve proximity limited ablation, and in 9 patients, coronary artery proximity prevented complete LP abolition. In 3 patients (7%), a nonclinical monomorphic VT was inducible postablation.

Overall, VT was induced in 69 patients (79%) at baseline versus 16 (19%) after ablation (2 not assessed because of complications). Final VT inducibility provided a high positive predictive value (14/16 patients; positive predictive value, 88%) but not negative predictive value for VT recurrence (30/51 patients; negative predictive value, 59%).

**VT Characteristics and Termination Sites**

The anteroseptal group was more easily inducible at baseline (93% versus 65%; \( P<0.001 \)) with a greater number of VTs induced per patient (2 [1–3] versus 1 [0–2]; \( P<0.001 \)) and a significantly higher proportion of left bundle branch block VT configuration (73% versus 12%; \( P<0.001 \)). A left inferior VT axis was only seen in anteroseptal patients

---

**Figure 1. A.** Characteristic surface ECG findings in patients with an anteroseptal scar pattern (frequent first-degree heart block, wide QRS, and left bundle branch block pattern with rS or QS in V5/6). **B.** The more subtle ECG findings seen with inferolateral scar, showing altered ventricular repolarization in the inferolateral leads and low voltage in the limb leads.
LOW UNIPOLAR VOLTAGE (<8 mV) AREAS

ANTEROSEPTAL \( N=6 \)

ENDOCARDIUM \( N=6 \)

EPICARDIUM \( N=2 \)

EARLY CARDIOMYOPATHY

ANTEROSEPTAL \( N=38 \)

ENDOCARDIUM \( N=38 \)

EPICARDIUM \( N=26 \)

DILATED CARDIOMYOPATHY

ANTEROSEPTAL \( N=22 \)

ENDOCARDIUM \( N=20 \)

EPICARDIUM \( N=21 \)

INFEROLATERAL \( N=22 \)

ENDOCARDIUM \( N=20 \)

EPICARDIUM \( N=21 \)

There were significant differences in the endocardial and epicardial termination sites. In the anteroseptal group, 80% of patients had at least 1 endocardial VT terminated during ablation versus 23% of inferolateral patients \( (P<0.001) \). Conversely, no anteroseptal patients had an epicardial VT termination versus 30% of patients in the inferolateral group \( (P<0.001) \), colocating with epicardial LPs \( (\kappa=0.667; P=0.014) \); Figure 4).

Complications

Of the 87 patients, 3 died in hospital (3%). Causes were operated cardiac tamponade with late infectious sequelae and low cardiac output after surgery \( (n=1) \), refractory heart failure \( (n=1) \), and intractable electrical storm \( (n=1) \), despite 2 emergency ablation procedures with invasive circulatory support and subsequent sympathectomy. The overall complication rate was 11%. Two patients developed tamponade, 1 needing surgery. One patient developed a pulmonary embolus needing anticoagulation. Three patients in the anteroseptal group developed heart block, subsequently needing device implant or upgrade to cardiac resynchronization therapy. Two patients had vascular complications requiring surgical intervention. One patient developed left phrenic nerve palsy after epicardial ablation, despite high-output phrenic nerve pace mapping, without clinical sequelae.

VT Recurrence

In the overall population, 44 patients (51%) had VT recurrences, either early in hospital or during follow-up (Table 3). Anteroseptal scar location was associated with significantly higher VT recurrence \( (74\% \text{ versus } 25\%; \text{log-rank } P<0.001) \) and higher first redo procedure rates \( (59\% \text{ versus } 7\%; \text{log-rank } P<0.001) \). Additionally, in the anteroseptal group, another 7 patients underwent 2, and 1 patient 3 redo procedures. The recurrence rate was not significantly different comparing ECM and DCM in the inferolateral group \( (18\% \text{ IL-ECM versus } 33\% \text{ IL-DCM}; \text{log-rank } P=0.27) \) and in the anteroseptal group \( (67\% \text{ AS-ECM versus } 76\% \text{ AS-DCM}; \text{log-rank } P=0.85; \text{Figure 5}) \).

Clinical predictors of VT recurrence included age \( (\text{hazard ratio } [HR], 1.040; P=0.007) \), advanced New York Heart Association class \( (HR, 1.898; P=0.001) \), electrical storm presentation \( (HR, 4.356; P<0.001) \), lower LV ejection fraction \( (10\%; HR, 0.0742; P=0.016) \), and broader baseline QRS duration \( (10 \text{ ms}; HR, 1.101; P=0.011) \). Procedural predictors of VT recurrence were larger endocardial unipolar scar area \( (10 \text{ cm}^2; HR, 1.103; P=0.001) \), anteroseptal scar type \( (HR, 4.369; P<0.001) \), number of induced VTs \( (HR, 1.210; P=0.001) \).
and final VT inducibility (HR, 2.786; P=0.002). The presence of epicardial LPs (HR, 0.299; P=0.003) was associated with lower VT recurrence. At multivariable analysis of the clinical data, presentation with electrical storm (HR, 3.211 [1.656–6.224]; P=0.001) and New York Heart Association class (HR, 1.608 [1.085–2.383]; P=0.018) predicted VT recurrence. The only procedural predictor of VT recurrence at multivariate analysis was anteroseptal scar type (HR, 5.547 [2.369–12.985]; P<0.001).

Mortality
There were 13 deaths (15%) during the follow-up period (9 AS-DCM and 5 IL-DCM patients). Of these, 12 patients had a cardiac death (5 because of electrical storm and 7 because of decompensated heart failure). A further 2 patients underwent cardiac transplantation, and 2 ventricular assist device implantation, 1 respectively in each scar distribution group (Table 3).

Univariable mortality predictors included age (HR, 1.074; P=0.026), advanced New York Heart Association class (HR, 5.999; P<0.001), lower LV ejection fraction (10%; HR, 0.473; P=0.011), greater LV end-diastolic volume (10 mL; HR, 1.076; P=0.002), chronic renal impairment (<30 mL/min; HR, 4.247; P=0.016), larger endocardial bipolar scar (10 cm²; HR, 2.528; P=0.001), larger endocardial unipolar scar (10 cm²; HR, 1.196; P<0.001), final VT inducibility (HR, 3.278; P=0.038), and VT recurrence (3.548; P=0.029).

Discussion
The present study provides the first electroanatomic definition of 2 distinct patterns of myocardial scarring in patients with NICM with drug-refractory VT that relate directly to both the optimal ablation strategy and outcome. The relatively high proportion of patients manifesting an anteroseptal pattern in this study as compared with previous reports is because of our methodology of including all anteroseptal segments as opposed to only isolated septal substrate.3

At present, the precise cause of distinct scar distributions in NICM is unclear, possibly relating to a combination of excess basal mechanical stress and underlying cardiomyopathic processes. A high proportion of inferolateral patients with or without LV dysfunction had a clinically suspected myocarditis supporting the possible role of viral infection in this subgroup.13 In contrast, a previous MRI study in patients with the lamin A/C mutation and DCM described a predominance of basal and midseptal LV involvement, correlated with conduction abnormalities.16 The higher proportion of anteroseptal patients fulfilling diagnostic criteria for DCM in our data may relate to more extensive endocardial scarring.
and common intraventricular conduction delay, frequently as a left bundle branch block pattern, causing LV dyssynchrony and dysfunction, leading to a worse prognosis.

Unipolar Scar Distribution

Unipolar low-voltage distribution allows categorization of scar type as an anteroseptal or inferolateral pattern for NICM and shows, as previous reports describe, a basal predominance of scarring in those patients. It is probable from our data that a slight overestimation of low-voltage areas was obtained because of inclusion of perimital fibrous tissue. However, this potential confounder is overcome using a segmental analysis that includes only the predominant voltage type within the perimital segments.

Anteroseptal and inferolateral scar subtype can be observed even in patients without overt HF as evidenced by concordant but less severe scar patterns compared with the DCM cohort. Baseline characteristics can help to predict these patterns even in the absence of imaging of which VT morphology is especially useful. The VT axis alone is a powerful tool for dividing anteroseptal (left inferior axis) and inferolateral scar types (right superior axis).

Considerations for Procedural Approach Before Ablation

The findings of this study can also be used to restrict the need for an epicardial approach in NICM. In the setting of anteroseptal type, the epicardial approach is not useful because of septal substrate, complex local anatomy, proximal left anterior descending artery, and prominent epicardial fat (ablation was performed in only 14% of these patients; 4 of 28 epicardial maps). This finding sets the rationale to proceed with a first-line epicardial approach in cases where the ECG pattern or imaging suggests an inferolateral pattern, because half of

Table 3. Clinical End Points During Follow-Up

<table>
<thead>
<tr>
<th>Procedure to discharge, d</th>
<th>AS-ECM (n=6)</th>
<th>IL-ECM (n=22)</th>
<th>P Value</th>
<th>AS-DCM (n=38)</th>
<th>IL-DCM (n=21)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedure to discharge, d</td>
<td>9 (7–12)</td>
<td>6 (4–7)</td>
<td>0.009</td>
<td>9 (5–16)</td>
<td>6 (4–12)</td>
<td>0.086</td>
</tr>
<tr>
<td>VT recurrence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follow-up time, d</td>
<td>344 (287–474)</td>
<td>659 (393–886)</td>
<td>0.007</td>
<td>552 (241–836)</td>
<td>756 (436–819)</td>
<td>0.43</td>
</tr>
<tr>
<td>Time to VT recurrence, d</td>
<td>110 (1–320)</td>
<td>659 (387–886)</td>
<td>0.008</td>
<td>19 (3–381)</td>
<td>407 (71–780)</td>
<td>0.048</td>
</tr>
<tr>
<td>Redo in hospital (patients)</td>
<td>2 (33%)</td>
<td>1 (5%)</td>
<td>0.10</td>
<td>13 (34%)</td>
<td>1 (5%)</td>
<td>0.011</td>
</tr>
<tr>
<td>AAD discharge</td>
<td>2 (33%)</td>
<td>8 (36%)</td>
<td>1.00</td>
<td>18 (51%)</td>
<td>8 (38%)</td>
<td>0.33</td>
</tr>
<tr>
<td>VT recurrence overall</td>
<td>4 (67%)</td>
<td>4 (18%)</td>
<td>0.038</td>
<td>29 (76%)</td>
<td>7 (33%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Electrical storm</td>
<td>2 (33%)</td>
<td>2 (9%)</td>
<td>0.19</td>
<td>10 (26%)</td>
<td>3 (14%)</td>
<td>0.34</td>
</tr>
<tr>
<td>Incessant VT</td>
<td>1 (17%)</td>
<td>0 (0%)</td>
<td>0.21</td>
<td>1 (3%)</td>
<td>0 (0%)</td>
<td>1.00</td>
</tr>
<tr>
<td>Paroxysmal VT</td>
<td>1 (17%)</td>
<td>2 (9%)</td>
<td>0.53</td>
<td>18 (47%)</td>
<td>4 (19%)</td>
<td>0.031</td>
</tr>
<tr>
<td>Medical therapy</td>
<td>0</td>
<td>0</td>
<td>1.00</td>
<td>2</td>
<td>0</td>
<td>0.53</td>
</tr>
<tr>
<td>ATP termination</td>
<td>1</td>
<td>0</td>
<td>0.21</td>
<td>3</td>
<td>0</td>
<td>0.54</td>
</tr>
<tr>
<td>Shock termination</td>
<td>0</td>
<td>2</td>
<td>1.00</td>
<td>13</td>
<td>4</td>
<td>0.21</td>
</tr>
<tr>
<td>AAD follow-up</td>
<td>3 (50%)</td>
<td>11 (50%)</td>
<td>1.00</td>
<td>23 (66%)</td>
<td>9 (43%)</td>
<td>0.094</td>
</tr>
<tr>
<td>Total patients undergoing redo</td>
<td>3 (50%)</td>
<td>1 (5%)</td>
<td>0.022</td>
<td>23 (61%)</td>
<td>2 (10%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Death</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In-hospital death</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1.00</td>
<td>3 (8%)</td>
<td>0 (0%)</td>
<td>0.54</td>
</tr>
<tr>
<td>CD/VAD/transplant</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1.00</td>
<td>8/1/1 (26%)</td>
<td>4/1/1 (29%)</td>
<td>0.85</td>
</tr>
<tr>
<td>VT recurrence CD/VAD/transplant</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1.00</td>
<td>9 /10 (90%)</td>
<td>3/6 (50%)</td>
<td>0.20</td>
</tr>
<tr>
<td>Death</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1.00</td>
<td>9 (24%)</td>
<td>4 (19%)</td>
<td>0.75</td>
</tr>
</tbody>
</table>

AAD indicates antiarrhythmic drug; AS, anteroseptal; ATP, antitachycardia pacing therapy; CD, cardiac death; DCM, dilated cardiomyopathy; EAM, electroanatomic mapping; ECM, early cardiomyopathy; VAD, ventricular-assist device; and VT, ventricular tachycardia.
mappable VTs require epicardial ablation and three quarters of patients have epicardial LPs. Anteroseptal patients are best dealt with a first-line endocardial ablation as evidenced by the high proportion of VT termination sites in the endocardial basal anterior and anteroseptal segments and additionally in the aortic cusps and right ventricular septum. Late activity in the anteroseptal pattern is rare; when present, linked to bipolar scar in the basal anterior and basal anteroseptal segments implies a greater scar density and advanced disease, conferring a poor prognosis.

Specific Technical Challenges

Catheter ablation for both arrhythmogenic substrates poses unique technical challenges. Fifty percent of patients with inferolateral scars and LPs had overlying coronary arteries or phrenic nerve capture at target ablation sites, and hence coronary image integration is mandatory, and double epicardial access for balloon inflation lifting of the phrenic nerve may sometimes be needed. In the anteroseptal group, we observed an increased number of VTs, often with subtle changes in morphology during ablation and little late activity, highlighting a complex intramural substrate that at present is difficult to fully address with current techniques of unipolar RF delivery from either side of the septum necessitating high-energy RF ablation often with concomitant right ventricular septal ablation, leading to a relatively high incidence of complete heart block and subsequent device implantation or upgrade. This experience presents disappointing results of catheter ablation, with high rates of VT recurrence and cardiac death, mostly occurring in patients with a clinical picture of DCM and anteroseptal scar pattern. Despite frequent VT terminations during ablation, recurrences are frequent and not reliably predicted by postablation programmed stimulation. The intramural scarring process typical of DCM does not allow the identification of VT isthmuses in many instances and also prevents effective and long-lasting lesion formation because of the limited (<5 mm) penetration of the RF energy achieved even with irrigated catheters at high power. Although alternative energy delivery modalities have been described, including bipolar RF,17 high-intensity focused ultrasound,18 needle ablation,19 or intracoronary ethanol injection,20 none of them are currently available for routine clinical application. The data from this series should also stimulate technical and clinical research in this field.

Limitations

Biopsy-proven diagnoses are not provided in this study. The dichotomous classification of patients in this study may be a limiting factor given the heterogeneity of patients. Another potential confounder is that we did not systematically classify scar patterns using imaging.

Conclusions

Patients with NICM may be categorized from scar predominance and consequent arrhythmia as either anteroseptal or inferolateral. Delayed-enhanced imaging and simple ECG analysis allow scar location to be predicted, guiding either a first-line biventricular endocardial approach in anteroseptal patients or left ventricular endoepicardial approach in inferolateral patients. VT recurrence and redo rates are particularly high in the setting of anteroseptal substrate. Further developments are required to improve outcomes in patients with NICM with drug-refractory VT focusing primarily on overcoming anatomic restrictions that are scar type-dependent.

Acknowledgments

We would like to thank all the staff at the Ventricular Tachycardia Unit of San Raffaele University Hospital for their tireless work and professionalism.

Disclosures

Dr. Della Bella is a consultant for St Jude Medical and has received honoraria for lectures from Biosense Webster, St Jude Medical,
References


9. Hutchinson MD, Gerstenfeld EP, Desjardins B, Bala R, Riley MP, Garcia FC, Dixit S, Lin D, Tzou WS, Cooper JM, Verdino RJ, Callans DJ, Marchlinski FE. Endocardial unipolar voltage mapping to detect epicardial and Biotronik. Drs Oloriz and Silberbauer are Advanced European Heart Rhythm Association Fellows with grants funded by Biosense Webster. The other authors report no conflicts.


CLINICAL PERSPECTIVE

Sustained monomorphic ventricular tachycardia in nonischemic left ventricular cardiomyopathy is usually because of reentry in regions of ventricular scar. In contrast to ischemic cardiomyopathy, the location of the arrhythmic substrate is variable in location and often located intramurally or epicardially, making catheter ablation a significant challenge. In this detailed study of 87 patients undergoing left ventricular mapping for ventricular tachycardia because of nonischemic cardiomyopathy, we show that these patients can be categorized into anteroseptal or inferolateral scar types based on electro-anatomic unipolar voltage maps. Patients with an inferolateral pattern usually have epicardial arrhythmia substrate with late potentials, although overlying coronary arteries or the left phrenic nerve are obstacles to epicardial ablation in up to half of these patients. In contrast, in the anteroseptal scar subtype, the arrhythmia substrate is often intramural in location, with-out late potentials. Anteroseptal scars are associated with higher rates of ventricular tachycardia recurrence after catheter ablation. This study provides a better understanding of the arrhythmogenic substrate in nonischemic cardiomyopathy and describes specific technical challenges that are scar type–dependent and have prognostic significance.
Catheter Ablation of Ventricular Arrhythmia in Nonischemic Cardiomyopathy:
Anteroseptal Versus Inferolateral Scar Sub-Types
Teresa Oloriz, John Silberbauer, Giuseppe Maccabelli, Hiroya Mizuno, Francesca Baratto, Senthil Kirubakaran, Pasquale Vergara, Caterina Bisceglia, Giulia Santagostino, Alessandra Marzi, Nicoleta Sora, Carla Roque, Fabrizio Guarracini, Dimitris Tsiachris, Andrea Radinovic, Manuela Cireddu, Simone Sala, Simone Gulletta, Gabriele Paglino, Patrizio Mazzone, Nicola Trevisi and Paolo Della Bella

*Circ Arrhythm Electrophysiol.* 2014;7:414-423; originally published online May 1, 2014;
doi: 10.1161/CIRCEP.114.001568

*Circulation: Arrhythmia and Electrophysiology* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2014 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/7/3/414

**Permissions:** Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation: Arrhythmia and Electrophysiology* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

**Reprints:** Information about reprints can be found online at:
http://www.lww.com/reprints

**Subscriptions:** Information about subscribing to *Circulation: Arrhythmia and Electrophysiology* is online at:
http://circep.ahajournals.org/subscriptions/