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Substrate-guided ablation of haemodynamically tolerated and untolerated ventricular tachycardia in patients with structural heart disease: effect of cardiomyopathy type and acute success on long-term outcome

Riccardo Proietti[1,2], Vidal Essebag[1,3], Jessica Beardsall[4], Philip Hache[4], Alfredo Pantano[4], Zaev Wulffhart[4], Rasna Juta[4], Bernice Tsang[4], Jacqueline Joza[1], Thais Nascimento[1], Vagner Pegoraro[1], Yaariv Khaykin[4], and Atul Verma[1,4]*

[1]McGill University Health Centre, Montreal, Quebec, Canada; [2]Cardiology Department, Luigi Sacco Hospital, Milan, Italy; [3]Hôpital Sacré-Cœur de Montreal, Quebec, Canada; and [4]Southlake Regional Health Centre, Heart Rhythm Program, Suite 602, 581 Davis Drive, Newmarket, Ontario, Canada L3Y 2P6

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Aims The purpose of this study was to evaluate the outcomes of purely substrate-guided ventricular tachycardia (VT) ablation in patients with non-ischaemic dilated cardiomyopathy (NIDCM) and ischaemic cardiomyopathy (ICM) and the impact of acute procedural success on long-term outcome.

Methods and results One hundred and forty-two patients (65 ± 12 years old, 72% male) with ICM ($n = 87$) and with NIDCM ($n = 55$) underwent substrate-guided VT ablation. The ablation approach involved eliminating all LP regions and ablating all scar border zone regions with 10 or more out of 12 pace-matching. All patients were followed with regular implantable defibrillator interrogations for mean 641 ± 301 days. Complete acute success (no inducible VT) was achieved in 60 patients with ICM (69%) and in 29 patients with NIDCM (53%) ($P = 0.03$). Partial success (elimination of clinical VT only) was obtained in nine patients with ICM (10%) and in four patients with NIDCM (7%) ($P = 0.14$). Procedural failure (clinical VT still inducible) occurred in 18 patients within the ICM group (21%) and in 22 patients of the NIDCM (40%) ($P = 0.04$). Overall, 51 patients presented with recurrence of ventricular arrhythmias: 23 in the group with ICM (26%) and 28 in the group with NIDCM (51%) ($P = 0.03$). Long-term success was related to acute procedural outcome.

Conclusions Substrate-guided ablation is an effective approach in the treatment of VT with long-term outcome directly related to acute procedural success. Success rates are significantly lower in patients with NIDCM compared with those with ICM.

Keywords Catheter ablation • Cardiomyopathy • Ventricular tachycardia • Outcome

Introduction

Traditionally, catheter ablation of ventricular tachycardia (VT) involved inducing the arrhythmia, mapping the circuit during VT, and using pacing manoeuvers to localize critical portions of the circuit.[1–3] Substrate-guided ablation of VT, however, involves identifying potentially arrhythmogenic regions of myocardium in sinus rhythm and avoids the need for mapping during VT.[2,3] This can substantially reduce the risk associated with VT ablation.[4] Targets of substrate-guided ablation include (i) areas of late potentials in scar regions;[5,6] (ii) regions of good pace-match to the clinical VT in scar border zones;[7] and (iii) complete elimination of all signals within scar regions (scar 'homogenization').[8] Although first described in the context of haemodynamically unstable VT's,[9] substrate-guided ablation evolves as a method of choice for all VT ablations in cardiomyopathy. In patients with electrical storm, extensive substrate-guided

* Corresponding author. Tel: +1 905 953 7917; fax: +1 905 953 0046. *E-mail address*: atul.verma@utoronto.ca

What's new?

- This study reports the results of a purely substrate-guided ablation in patients with ventricular tachycardia (VT). Prior studies have reported on the use of substrate ablation as adjunctive therapy; however, they also used entrainment manoeuvres and activation mapping in patients with haemodynamically stable tachycardia along with a substrate approach. We applied a purely substrate approach irrespective of whether the VT was haemodynamically tolerated, thereby avoiding the risks associated with repeated inductions and maintenance of VT.
- This approach was applied in a wide population of patients with ischaemic and non-ischaemic cardiomyopathy. Differences in the outcomes between the two types of cardiomyopathies are discussed.
- The acute success rate and long-term outcomes, including mortality and rate of hospitalizations, are provided.

ablation was shown to be more effective than more limited ablation.[8] Another study demonstrated no additional long-term benefit of activation mapping during VT to substrate-guided ablation alone.[10]

The efficacy of substrate-guided ablation may be less effective in patients with non-ischaemic dilated cardiomyopathy (NIDCM) compared with those with ischaemic cardiomyopathy (ICM).[11] This is because scar regions in NIDCM may be less extensive, patchier, and may have more epicardial involvement.[12–14] Consequently, the combined approach of activation map and substrate ablation during VT ablation in patients with NIDCM has shown unsatisfactory results in terms of arrhythmias recurrence after ablation.[15–19] Only very limited data exist for outcomes with VT ablation in NIDCM vs. ICM with an exclusively substrate-guided approach.[11,20] For the past several years, at our centre, we have been employing a substrate-guided approach for all ICM and NIDCM VT ablation to reduce the difficulties introduced to the procedure with repeated inductions and maintenance of ventricular arrhythmias. The purpose of this study was to evaluate the outcomes of a purely substrate-guided VT ablation approach in patients with ICM and NIDCM as well as the impact of acute procedural success on long-term outcome.

Methods

Patient population

All patients undergoing VT ablation at Southlake Regional Health Centre, Newmarket, ONT, Canada, have been followed in a prospective database since July 2005. For this study, only patients with a diagnosis of NIDCM or ICM who underwent the first ablation procedure from July 2005 until March 2013 were included ($n = 142$). No redo-procedures were included in the study. The definitions of ICM and NIDCM were in keeping with consensus guidelines.[21,22] Ischaemic cardiomyopathy is defined as impaired left ventricular ejection fraction (LVEF) with angiographically confirmed stenosis of $>70\%$ in at least one of the epicardial coronary arteries and/or history of myocardial infarction or coronary revascularization. Non-ischaemic dilated cardiomyopathy was defined as impaired LVEF in the absence of any significant epicardial coronary stenosis as defined above and in the absence of any systemic or endocrine diseases associated with cardiomyopathy. All patients had implantable defibrillators (ICDs) and underwent ablation for one or more ICD shock from their device or a high burden of recurrent anti-tachycardia pacing (ATP) therapy. Electrical storm was defined as three or more ICD shocks within 24 h.

All procedures were performed with patients' written informed consent. Collection of data was approved by the institution's research ethics board.

Ventricular tachycardia mapping and ablation protocol

All procedures were performed under conscious sedation or general anaesthesia. Transthoracic echocardiography was performed in all patients prior to the procedure to rule out the presence of intra-ventricular thrombus. A quadripolar electrode catheter was placed in the right ventricular apex and an intracardiac echocardiography (ICE) catheter was placed in the right ventricular cavity for imaging of the LV. All patients were administered intravenous unfractionated heparin to maintain an activated clotting time of ≥ 250 s prior to LV access. Endocardial access to the LV was obtained via the retrograde aortic approach in all patients. At discretion of the operator, an additional trans-septal access was performed to facilitate mapping and ablation of the arrhythmias.

Epicardial access was only obtained (i) if the 12-lead ECG of the VT was suggestive of an epicardial source; (ii) if prior ablation had failed to eliminate the VT; or (iii) if endocardial mapping did not reveal any evidence of LV scar. Epicardial access was obtained using the standard subxyphoid approach described elsewhere.[23] Epicardial access was obtained prior to systemic anticoagulation where possible. If epicardial access had to be obtained during the procedure, heparin was reversed with protamine until pericardial access was secured followed by re-initiation of heparin anticoagulation.

All data were recorded using a standard recording system (Prucka Cardiolab, GE Healthcare). Initially, programmed ventricular stimulation was performed from the right ventricular apex and outflow tract to check for inducibility of VT. Up to three ventricular extrastimuli were delivered at two different cycle lengths (600 and 400 ms) to a minimum coupling interval of 200 ms. Isoprotenerol infusion was not used during the induction protocol. The induced VT was defined as 'clinical' if the 12-lead ECG of the induced VT matched that of spontaneous VT. In cases where a 12-lead ECG of the VT was not available, 'clinical' VT was defined as an induced VT with the same cycle length and same morphology of the spontaneous VT ICD electrogram at visual inspection. The VT was considered haemodynamically intolerable if the patient became symptomatic (chest pain or pre-syncope or syncope), or if the systolic blood pressure dropped less than 80 mmHg systolic at one minute post-induction and lasting for at least one minute.

Following induction, VT was promptly interrupted either by overdrive pacing or cardioversion.

Electroanatomical mapping of the LV was performed using a 3.5 mm open irrigated tip catheter (Navistar Thermocool, Biosense Webster) and a three-dimensional electroanatomical mapping system (CARTO, Biosense Webster, Johnson & Johnson), during sinus rhythm and/or pacing from the right ventricle. Scar regions were defined as areas with bipolar electrogram voltage ≤ 0.5 mV and scar border zone areas as those with a low bipolar voltage between 0.5 and 1.5 mV.[9] The Normal myocardium was defined as bipolar voltage ≥ 1.5 mV. The CARTO system tools for perimeter tracking and area calculation was used. The area of electroanatomical scar and low voltage area was provided in percentage compared with the total ventricular area.

During creation of the electro-anatomical map, scar regions with late potentials (LP) were tagged. A LP was defined as a low voltage, sharp,

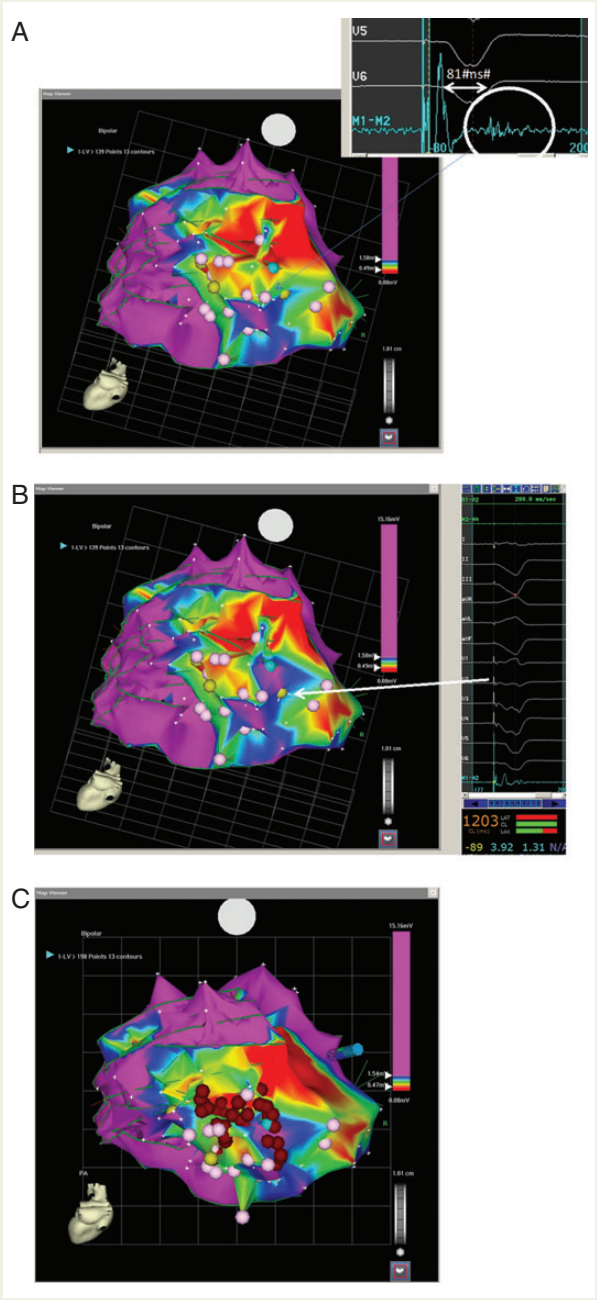

Figure 1 (*A*, *B* and *C*) The panels demonstrate an example of the mapping and ablation strategy used for substrate-guided ablation of VT. In (*A*) is a site showing a sample of a late potential. As can be seen, there is at least 20 ms (81 ms) from the far-field ventricular electrogram to the late-potential. However, the late potential is still contained within the surface QRS (see lead V6). In (*B*), there is an example of a site with a 12/12 pace-match to the induced VT. (*C*) shows the ablation strategy in which areas of scar or scar border zone with sites of late potentials and good pace-match sites are targeted. Red dots represent the ablation lesions.

high-frequency signal with a distinct onset at least 20 ms from the local ventricular electrogram on the ablation catheter[24] (*Figure 1A*). Scar regions in the epicardial space were defined using similar voltage criteria, but abnormal scar was distinguished from epicardial fat by looking for fractionated or wide duration (>80 ms) electrograms. Intracardiac echocardiography was also used to examine for wall motion abnormalities and echodense or thinned myocardium to confirm the presence of scar where possible. Pace-mapping was also performed in scar border zone regions (*Figure 1B*). All areas with a pace-mapping that match 10 or more of the 12 lead electrocardiogram of the clinical VT were also tagged (*Figure 1C*).

Ablation was performed with the same catheter used for mapping. Ablation was performed at 50 w with a maximum temperature of 45°C and an irrigation rate of 30 mL/min. The ablation approach involved eliminating all LP regions and ablating all scar border zone regions with 10 or more out of 12 pace-matching. Regions of good pace-match in the scar border zone were also connected to lesions within the scar region. When ablating in the epicardial space, a simultaneous pigtail drain was placed in the pericardial space along with the catheter introducer sheath to avoid fluid overload in the pericardium resulting in tamponade.

After all targeted regions were ablated ventricular programmed stimulation was repeated as described earlier to evaluate acute success of the procedure. Acute success was defined as the inability to induce any VT. Partial success was defined as the inability to induce the clinical VT but other VT(s) remain inducible. Unsuccessful ablation was defined as the ability to re-induce the clinical VT at the end of the procedure.

Patient follow-up

After discharge, all patients were followed up in our regional device clinic a minimum of every 6 months. At each follow-up, all device data and episodes were recorded and downloaded. Prior to 2008, our clinic used standard 'Pain-Free' programming parameters in which a monitor zone was programmed at 150 b.p.m., VT at 172–174 b.p.m., and VF at >200 b.p.m. At least three trains of ATP were programmed in the VT zone prior to shock. After 2008, the VT zone was programmed at 182–188 b.p.m. and the VF zone at >207–220 b.p.m. and the number of intervals needed to detect arrhythmia was increased to 24 based on newly published data. Post-ablation setting of the ICD therapy was adjusted to detect and treat all the slowest inducible VT.

The primary endpoint of the study was the recurrence of sustained ventricular arrhythmia >30 s documented by the ICD and/or ventricular arrhythmia requiring ICD therapy. Secondary endpoints of the study included cardiovascular hospitalization and cardiac mortality. Mean follow-up for patients was 641 ± 301 days.

Statistical analysis

Continuous variables are expressed as mean ± standard deviation and categorical variables as numbers and percentages. Comparisons between groups were performed by independent *t* test for continuous variables and by Fisher's exact test or χ^2 test for proportions. Survival curves were created by the Kaplan–Meier method and comparisons between groups were based on the log-rank test. Univariable and multivariable Cox proportional hazard analyses were used to assess the predictors of study endpoints. Hazard ratios with corresponding 95% confidence intervals (CIs) are presented. Statistical significance was defined by *P* value <0.05. Statistical analysis was performed with SPSS version 14.0 (PASW Statistics, IBM).

Results

Patient characteristics

Of the 142 patients, 87 (61%) had ICM, while 55 had NIDCM (39%). Patients were predominantly male ($n = 102$, 72%), with a mean age of 65 ± 12 years. Patients underwent ablation for one or more

Table 1 Baseline characteristics

	ICM ($n = 87$)	NIDCM ($n = 55$)	*P* value
Age (years)	66 ± 12	63 ± 13	0.07
Male gender (%)	63 (72%)	39 (71%)	0.54
Clinical presentation (%)			
One or more ICD shocks	66 (76%)	46 (84%)	0.14
Electrical storm	34 (39%)	24 (44%)	0.25
Recurrent/high-burden ATP	23 (26%)	7 (13%)	0.06
AAD at presentation (%)			
Beta-blocker	84 (97%)	53 (96%)	0.42
Sotalol	18 (21%)	10 (18%)	0.30
Amiodarone	49 (56%)	35 (64%)	0.38
Mexilitine	2 (2%)	1 (2%)	0.51
Prior open heart surgery (%)	34 (39%)	0 (0%)	<0.001
NYHA class (%)			
I	17 (20%)	10 (18%)	0.61
II	37 (43%)	29 (53%)	0.11
III	23 (26%)	12 (22%)	0.34
IV	10 (11%)	4 (7%)	0.22
LV ejection fraction (%)	29 ± 10%	30 ± 10%	0.53

ICD, implantable cardioverter defibrillator; ATP, antitachycardia pacing; NYHA, New York Heart Association; LV, left ventricular.

Table 2 Procedural details

	ICM ($n = 87$)	NIDCM ($n = 55$)	*P* value
Clinical VT CL (ms)	340 ± 90	348 ± 92	0.18
Conscious sedation (%)	30 (34%)	21 (38%)	0.36
General anaesthesia (%)	57 (66%)	34 (62%)	0.38
# Inducible VTs at the start of ablation	1.9 ± 1.1	1.9 ± 1.4	0.75
Retrograde aortic access (%)	87 (100%)	55 (100%)	0.99
Transseptal access (%)	35 (40%)	27 (49%)	0.12
Epicardial access (%)	1 (1%)	14 (25%)	<0.001
Number of points mapped per patient	297 ± 181	282 ± 168	0.18
Total low voltage area <1.5 mV (cm^2)	96 ± 34	42 ± 36	0.01
Total scar area <0.5 mV (cm^2)	59 ± 20	20 ± 11	0.008
Procedure time (min)	212 ± 40	221 ± 49	0.05
Fluoroscopy time (min)	28 ± 18	31 ± 19	0.07
Radiofrequency energy time (min)	96 ± 51	84 ± 54	0.07
AAD post-procedure (%)	51 (58%)	37 (67%)	0.17
Acute procedural outcome			
No inducible VTs	60 (69%)	29 (53%)	0.03
VT inducible; no more clinical VT	9 (10%)	4 (7%)	0.14
Clinical VT still inducible	18 (21%)	22 (40%)	0.04

CL, cycle length; VTs, ventricular tachycardias; AAD, antiarrhythmic drugs.

ICD shock from their device ($n = 112$, 79%) or a high burden of recurrent ATP therapy ($n = 30$, 21%). Of the patients with ICD shock, 58 (41%) presented with electrical storm. The mean ejection fraction was 29 ± 10%. Additional baseline details of the patients are listed in *Table 1*.

Procedural characteristics and acute outcomes

Procedural details for both groups are listed in *Table 2*. In all 142 patients, VT was induced during the EP study. Overall 38% of induced VTs were haemodinamically unstable, specifically 35% in ICM group and 44% in NIDCM group.

The mean number of induced VTs at the beginning of the procedure was similar between patients with ICD vs. NIDCM (1.9 ± 1.1 vs. 1.9 ± 1.4, $P = 0.75$).

When more than one VT was induced, only 53% of the additionally induced VTs had a morphology that matched the initial VT by ≥10 leads. The rest had <10 lead match.

The mean tachycardia cycle length was shorter in patients with ICD than in patients with NIDCM, although the difference was not statistically significant (340 ± 90 vs. 348 ± 92, $P = 0.18$).

Access to the LV was obtained via the retrograde aortic approach in all patients with additional trans-septal access in 35 patients with ICM (40%) and in 27 patients with NIDCM (49%). Nearly all patients with ICM were ablated endocardially; only one patient required epicardial access and ablation. Of the group of patients with NIDCM, an epicardial ablation was performed in 14 patients (15%) of the 55 ($P < 0.001$ compared with ICM).

A mean of 291 ± 174 points was obtained per map for all patients. Specifically, the average number of points per map was 297 ± 181 in the ischaemics and 282 ± 168 in the non-ischaemics ($P = 0.18$). Total low voltage area (<1.5 mV) was significantly greater for ICM (96 ± 34 cm^2) compared with NIDCM (42 ± 36 cm^2) ($P = 0.01$). Deep scar areas (<0.5 mV) were also significantly larger for ICM (59 ± 20 cm^2) compared with NIDCM (20 ± 11 cm^2) ($P < 0.01$). In the NIDCM patients, scar regions were predominantly localized to the basal inferolateral (49%), basal septal (36%), and basal anterior (20%) regions. In the ICM patients, scar was predominantly distributed in the inferior (40%), anterior (29%), anterolateral (20%) regions.

Acute success (non-inducibility of any arrhythmias) was achieved in 60 patients with ICM (69%) and in 29 patients with NIDCM (53%) ($P = 0.03$). Partial success, defined by elimination of the clinical VT but other VTs remain, was obtained in nine patients with ICM (10%) and in four patients with NIDCM (7%) ($P = 0.14$). Unsuccessful ablation (clinical VT was still inducible) occurred in 18 patients in the ICM group (21%) and in 22 patients with NIDCM (40%) ($P = 0.04$).

There was a trend towards longer procedure time in the NIDCM group (212 ± 40 vs. 221 ± 49 min, $P = 0.05$). There was also a trend towards greater fluoroscopy time in the NIDCM group (28 ± 18 vs. 31 ± 19 min, $P = 0.07$). The RF time was 96 ± 51 min in the ICM group and 84 ± 54 min in the NIDCM group ($P = 0.07$).

No intra-procedural death occurred in either group. Three patients in the NIDCM group had procedural cardiac tamponade that was successfully drained. Other complications, such as vascular access haematoma, pseudoaneurysm and fluid overload, were similar among

the two groups (*Table 3*). Post-procedural mortality occurred in two patients in each group. Refractory arrhythmic storm was the cause of death in the 2 patients with NIDCM, while the two patients with ICM died from cardiogenic shock.

Long-term outcomes

During a mean follow-up of 641 ± 301 days, 51 patients overall had recurrence of ventricular arrhythmias. The recurrence rate for both groups was directly related to acute procedural outcome (*Figure 2*). All patients with acute procedural failure experienced VT recurrence. In the ICM group, only 3% with acute procedural success and 44% of patients with partial success experience arrhythmia recurrence. In the NIDCM group, only 7% of patients with acute procedural success and 75% of patients with partial success had ventricular arrhythmia recurrence. Overall, freedom from ventricular arrhythmia recurrences was significantly higher in the ICM vs. NIDCM group (74 vs. 49%, $P = 0.03$, *Figure 3*).

During follow-up, cardiovascular hospitalization occurred in 30 of 142 (21%) patients, and more specifically in 13/87 (15%) of ICM patients compared with 17 of 55 (31%) NICM patients ($P = 0.03$). The most common causes for cardiovascular hospitalization were worsening heart failure (14/30, 47%), recurrent ventricular arrhythmia (11/30, 37%), and acute coronary syndrome (5/30, 17%).

Death from various causes occurred in 14 of 142 (10%) patients. There was a trend of increased all-causes mortality in patients with NIDCM, although not statistically significant ($P = 0.34$). Specifically, mortality for patients with ICM and NIDCM was 8 of 87 (9%) and 6 of 55 (11%), respectively.

Discussion

Main findings

A purely substrate-guided ablation strategy can be effective in obtaining acute procedural success (complete or partial) in a majority of patients (72%) with both ICM and NIDCM. At nearly 2 years' follow-up, 64% of patients were free of ventricular arrhythmias recurrence. However, both acute procedural success and long-term freedom from arrhythmias were higher in patients with ICM compared with NIDCM. This is the first large study to examine the effect of cardiomyopathy type on both acute and long-term outcome using an exclusively substrate-guided approach. Furthermore, at long-term follow-up, we showed that hospitalization rates were significantly higher for patients with NIDCM while long-term mortality was similar for both types of cardiomyopathy.

Table 3 Procedural complications

	ICM ($n = 87$)	NIDCM ($n = 55$)	*P* value
Vascular access haematoma (%)	4 (5%)	3 (5%)	0.81
Access pseudoaneurysm (%)	1 (1%)	0 (0%)	0.18
Cardiac tamponade (%)	0 (0%)	3 (5%)	0.08
Stroke/TIA (%)	0 (0%)	0 (0%)	–
Fluid overload/CHF (%)	8 (9%)	4 (7%)	0.71
In hospital death (%)	2 (2%)	2 (4%)	
Cardiogenic shock	2 (2%)	0 (0%)	
Refractory arrhythmia	0 (0%)	2 (4%)	0.12

TIA, transient ischaemic attack; CHF, congestive heart failure.

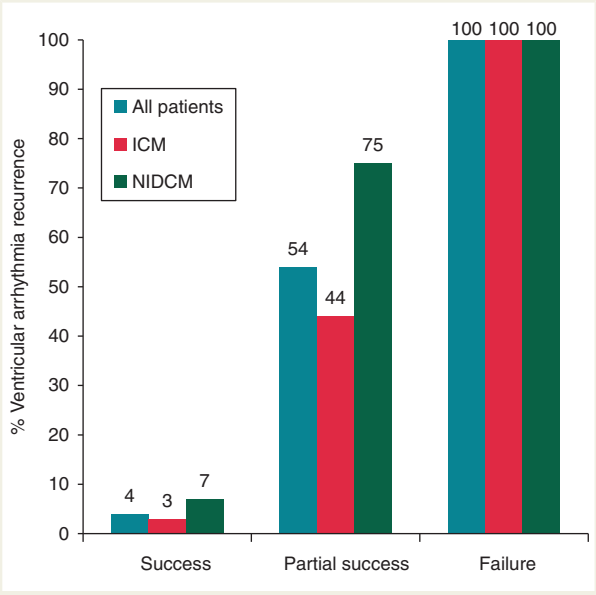

Figure 2 Recurrence rate of ventricular arrhythmia by acute procedural success.

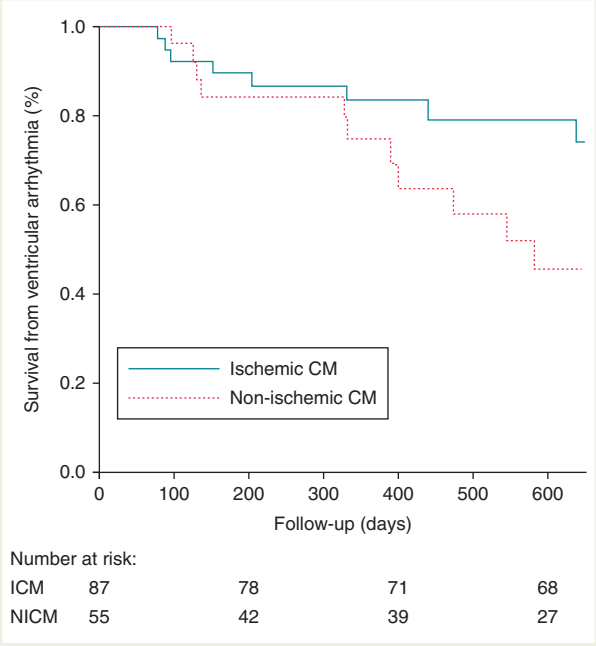

Figure 3 Freedom from recurrence of ventricular arrhythmia.

Substrate in ischaemic cardiomyopathy and non-ischaemic dilated cardiomyopathy

The substrate for both ICM and NIDCM based on voltage mapping have been well defined previously.[6,9] The scar region for ICM is predominantly endocardial[25] and follows the distribution of the coronary anatomy.[9] In contrast, NIDCM is characterized by a patchy distribution of myocardial fibrosis that is largely found in the perivalvular region with less dense scarring than that found in ICM.[6] Furthermore, there is more epicardial involvement of scarring compared with those with ICM.[12,16] All of these observations were largely confirmed by the findings of the electroanatomical maps in this study in which we showed significantly less low voltage surface area in NIDCM patients compared with those with ICM.

Recently, Nakahara *et al.*[11] further characterized the substrate of patients with NIDCM in comparison to ICM, with specific attention to potential targets for substrate-based ablation. In particular, they demonstrated that patients with NIDCM had significantly lower numbers of late potentials compared with patients with ICM, whether defined as moderately late potentials (occurring $<$100 ms after the QRS) or very late potentials (occurring $>$100 ms after the QRS). They also demonstrated a more epicardial distribution of myocardial fibrosis in patients with NIDCM, and there was no significant difference in the number of late potentials seen epicardially between NIDCM and ICM.

Furthermore, Nakahara *et al.*[11] also showed that NIDCM patients have fewer good-to-excellent pace-match sites compared with ICM. This may be due to the lack of protected isthmuses and consistent exit points found in ischaemic causes of cardiomyopathy. There is also a known higher incidence of focal arrhythmia mechanisms in NIDCM which are not reliant on a typical scar-based circuit.[26]

Indeed, our study seems to support the findings of Nakahara *et al.* We showed that using an ablation approach based on targeting of late potentials and regions of good pace-match was not as successful (acutely or in the long-term) in NIDCM patients compared with those with ICM. Given the lower incidence of late potentials and good pace-match sites, it would make sense that such an empiric approach would not fare as well in NIDCM patients.

Comparison with other outcome studies

In spite of the differences in outcome for NIDCM and ICM patients, the overall success rate of the substrate-based approach was 64% overall after almost 2 years follow-up in the present study. Furthermore, acute procedural success was obtained in 63% of all patients. These results compare favourably with other experiences[10,11,17] and are actually better than those reported in the multicentre Thermocool study,[27] suggesting that a purely substrate-based approach may be appropriate for patients with structural heart disease and VT. In fact, a recent study suggested that addition of activation mapping provides no further long-term benefit over substrate-based ablation alone.[10]

Recently, Dinov *et al.*[17] presented a large single-centre experience on acute and long-term outcome after VT ablation in a large cohort of patients with ICM and NIDCM. In this study, predominantly activation mapping and ablation were performed in almost 50% of patients, and substrate-based ablation was not used in all patients. One of their principal findings was that patients with NIDCM had significantly worse outcome compared with those with ICM, which is very similar to our finding and occurs despite use of additional activation mapping/ablation. Furthermore, it is interesting that at $\sim$2 years, our success rates would appear to be higher than those reported in the Dinov study. At $\sim$2 years, arrhythmia-free survival was 30% in patients with NIDCM and 50% in ICM, compared with 49 and 74%, respectively, from our present study. The main explanation is likely the lower use of antiarrhythmics in the Dinov study (34%) compared with our study (62%). This is also in spite of the fact that the rate of epicardial ablation was double in the Dinov study (31%) compared with ours (15%).

Finally, at long-term follow-up Dinov's results and ours both show an increased trend for all-causes mortality in the group with NIDCM but do not reach a statistical significance in comparison with ICM. This could be due to the length of follow-up, the sample or the inclusion of all-cause mortality. However, we detected a statistically significant increase in cardiovascular morbidity in patients with NIDCM.

Clinical implications

Substrate-based ablation may be a good technique to treat VT in patients with structural heart disease, with good overall outcomes and a low rate of complications. Thus, some of the difficulties introduced into the procedure with repeated inductions and maintenance of ventricular arrhythmias may be avoided. However, the acute and long-term outcome is better in those with ICM compared with those with NIDCM owing to differences in the substrate. Thus, addition of further ablation strategies in NIDCM patients, such as elucidation of focal tachyarrhythmias and epicardial substrates, may be very important in improving the outcomes in these patients.

Study limitations

Our study sample size is relatively modest, although there are not many large-scale studies on VT ablation in the literature and our population size is one of the largest exclusively substrate-based VT ablation experiences published to date. When the 12-lead ECG was not available, the induced clinical VT was determined on the basis of VT cycle length and morphologies. This approach can be limited by the difference detected in the cycle length between clinical and induced clinical VTs reported by Yoshida *et al.*[28] However, the same authors report a good accuracy of visual inspection in the identification of clinical VT.

We also did not systematically add activation mapping and ablation to these patients in a randomized fashion, thus we cannot conclude that addition of activation mapping would not have further improved the outcome. However, as mentioned earlier, prior studies have suggested that additional activation mapping makes little difference to outcome.[10,29] Finally, we did not perform high-density mapping of the ventricular substrate as was done in a prior study[11] and thus, our characterization of the substrate may have been limited. However, point-by-point mapping is used for most VT ablation procedures and the average number of points per map in this study is certainly consistent with prior publications. Furthermore, our findings of scar areas are not dissimilar from those found with higher density mapping.

Conclusions

A purely substrate-guided ablation is an effective approach in the treatment of VT with long-term outcomes directly related to acute procedural success. Success rates are significantly lower in patients with NIDCM compared with those with ICM.

Conflict of interest: V.E. is a recipient of a Clinician Scientist Award from the Canadian Institutes of Health Research.

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