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Better outcome of ablation for sustained outflow-tract ventricular tachycardia when tachycardia is inducible

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Aims In patients presenting with spontaneous sustained ventricular tachycardia (VT) from the outflow-tract region without overt structural heart disease ablation may target premature ventricular contractions (PVCs) when VT is not inducible. We aimed to determine whether inducibility of VT affects ablation outcome.

Methods and results Data from 54 patients (31 men; age, 52 ± 13 years) without overt structural heart disease who underwent catheter ablation for symptomatic sustained VT originating from the right- or left-ventricular outflow region, including the great vessels. A single morphology of sustained VT was inducible in 18 (33%, SM group) patients, and 11 (20%) had multiple VT morphologies (MM group). VT was not inducible in 25 (46%) patients (VTni group). After ablation, VT was inducible in none of the SM group and in two (17%) patients in the MM group. In the VTni group, ablation targeted PVCs and 12 (48%) patients had some remaining PVCs after ablation. During follow-up (21 ± 19 months), VT recurred in 46% of VTni group, 40% of MM inducible group, and 6% of the SM inducible group ($P = 0.004$). Analysis of PVC morphology in the VTi group further supported the limitations of targeting PVCs in this population.

Conclusion Absence of inducible VT and multiple VT morphologies are not uncommon in patients with documented sustained outflow-tract VT without overt structural heart disease. Inducible VT is associated with better outcomes, suggesting that attempts to induce VT to guide ablation are important in this population.

Keywords Ventricular tachycardia • Inducibility • Catheter ablation

Introduction

Sustained ventricular tachycardia (VT) with a presumptive outflow-tract (OT) origin that occurs in patients without overt structural heart disease is commonly considered for catheter ablation. Although efficacy is generally felt to be high, there are often challenges related to anatomy, inducibility, and procedure endpoints. Activation mapping during the arrhythmia is generally felt to be the most reliable means of identifying the ablation target, but requires the arrhythmia to be provokable.[1] When VT is not inducible, provokable or spontaneous premature ventricular contractions (PVCs) or non-sustained VT are often sought, sometimes combined with pace-mapping. There is a paucity of information as to how well targeting these spontaneous or inducible PVCs works for guiding ablation. Cyclic AMP-mediated triggered activity is thought to be the most common mechanism of idiopathic OT VT.[2,3] However, some evidence of focal structural abnormalities have been reported with MR imaging,[4] which suggests the potential for other mechanisms related to damaged tissue or scar that could even give rise to including small reentry circuit. Recently, we reported a small series of patients with arrhythmias from the periaortic region that seemed to be due to scar-related reentry despite absence of overt structural heart

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disease. More than one QRS morphology of arrhythmia was often seen in these patients and catheter ablation was often difficult.[5]

The aims of this study are to characterize VT inducibility in patients with documented sustained VT originating from OT region without overt structural heart disease, and to determine (i) whether ablation targeting PVCs when inducible sustained VT is absent has a similar outcome to ablation guided by mapping during inducible VT and (ii) to assess whether more than one morphology of OT arrhythmia is associated with worse outcome.

Methods

Patient characteristics

Between March 2003 and October 2012, 911 consecutive patients underwent 1231 catheter ablation procedures for ventricular arrhythmias (VAs) at the Brigham and Women's hospital. Of these, 54 patients without overt structural heart disease who underwent ablation at the OT region met the following criteria: (i) spontaneously occurring sustained monomorphic VT suspected to originate from the OT region had been documented and (ii) history, physical examination, 12-lead ECG, chest X-ray, echocardiography, exercise testing, and coronary angiography (when performed) were normal. We excluded patients where cardiac imaging had shown LV ejection fraction less than 45% or in whom an endocardial voltage maps showed an area of low voltage <1.5 mV remote from the pulmonary or aortic valve annulus indicating possible arrhythmogenic cardiomyopathy. Patients with idiopathic ventricular fibrillation or polymorphic VT, or incomplete data for analysis were excluded. We also excluded patients who had general anaesthesia during the electrophysiological study due to the potential effect of anaesthesia on inducibility.

ECG during sinus rhythm was analysed by one electrophysiologist who was blinded to clinical data. ECG scoring algorithm was applied to distinguish arrhythmogenic right-ventricular cardiomyopathy from idiopathic VT.[6,7] Data collection was performed according to protocols approved by the Human Research Committee of Brigham and Women's hospital. Of the 54 patients, 24 were previously included in a prior report.[5]

Electrophysiology study and ablation procedures

All anti-arrhythmic drugs were discontinued for at least five half-lives before ablation. The procedure was initiated with no or minimal sedation when possible, but all patients subsequently had conscious sedation using midazolam, fentanyl, or anaesthesia with propofol after the arrhythmia was identified and initial ablation performed. Programmed electrical stimulation was performed with up to four extrastimuli and burst pacing from the RV apex and/or OT to a cycle length of <250 ms. If sustained VT was not induced, stimulation was repeated under isoproterenol infusion at a rate up to 16 μg/min or after bolus injections (up to 30 μg). If isoproterenol administration was not successful, epinephrine bolus injections of 20–100 μg were performed to induce sustained VT in part of patients. If VT could not be induced, PVCs or non-sustained VT that were suspected to be potentially arising from the VT focus were targeted for ablation guided by pace-mapping and activation mapping. If sustained VT was inducible, the target for ablation was sought based on activation and in some cases entrainment mapping (concealed fusion, short post-pacing interval <30 ms of the VT cycle length).[8,9]

An electroanatomical mapping system (CARTO, Biosense Webster, Diamond Bar, CA, USA), was used as previously described.[5] A voltage map was created during sinus rhythm. Dense scar was defined as peak-to-peak bipolar electrograms amplitude <0.5 mV, and scar border zone as voltage ≥ 0.5 and <1.5 mV. Radiofrequency (RF) ablation energy was delivered at a power setting between 25 and 50 W targeting to drop impedance more than 10 Ω as described in previous paper.[5] Also, RF ablation energy was limited below 35 W when targeting above aortic valve. After ablation, induction testing was repeated including administration of any beta-agonists that had been required for arrhythmia initiation before ablation. Acute failure was defined as inducible targeted VT present after the final ablation application. Also, we analysed whether any residual targeted PVCs were observed after the final ablation lesion.

We analysed the characteristics of inducible VT or PVCs from simultaneous 12-lead ECGs recorded during the procedure. Morphological similarity between VT and PVCs was assessed retrospectively by two independent electrophysiologists who were blinded to ablation data and outcome data. Differences were resolved by mutual agreement. Two QRS complexes were defined as having the same morphology if V1 was similar, the QRS axis was within 20° and the transition in the precordial leads was the same. PVCs for analysis were acquired before catheters were placed in or near the RVOT, and for PVCs acquired after catheter insertion, we reviewed the intracardiac electrograms of PVCs to exclude the possibility of catheter-induced PVCs. We also excluded PVCs suspected to originate from non-OT region based on morphology.

Follow-up

After ablation, patients were followed at our institution or by their referring physician. Recurrence data were collected by reviewing the electronic medical chart or contacting the physician's office by phone. Recurrence was defined as documented sustained VT, or repeat electrophysiology study or ablation for the same symptoms as before ablation or documented non-sustained VT. Three patients were lost to follow-up (95% follow-up rate).

Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD). Student's *t*-test or Mann–Whitney *U*-test was used to compare continuous variables with Bonferroni correction. χ^2 test or Fisher's exact test was used for categorical variables. Event-free survival curves were created using the Kaplan–Meier method and significance between groups was tested with the log-rank test. A *P*-value <0.05 was considered statistically significant comparing two groups. Comparing among three groups, *P*-value <0.017 (0.05/3) was considered statistically significant adjusted with Bonferroni correction. A Cox proportional hazard model was used to assess correlation between VT inducibility and recurrence adjusting for the presence of scar on MRI or by low-voltage area, and the presence of residual PVCs after ablation. SPSS software version 19.0 (IBM Corporation, NY, USA) was used for all statistical analyses.

Results

Patients characteristics

In the laboratory, sustained VT was induced (28 patients) or occurred spontaneously (one patient) in 29 patients (VTi group), whereas sustained VT was not inducible in 25 patients (VTni group). In the VTi group, 18 (62%) patients showed a single morphology of inducible VT (SM group), whereas 11 (38%) patients showed multiple morphologies of inducible VT (MM group). Baseline characteristics of patients are summarized in *Table 1*. Overall, 29 (53%) patients had a history of failing therapy with anti-arrhythmic drugs, and 14 (26%) patients had used more than two anti-arrhythmic drugs. Half of the

patients ($n = 27$) had more than one VT episodes per month. Twelve (22%) patients had an ICD implantation before procedure. Twenty-three (43%) patients had undergone a prior electrophysiology study and 22 (41%) had at least one attempt at catheter ablation (range 1–7 procedures) prior to referral.

Five patients (three patients in VTni group and two patients in SM group) showed T wave inversion in V1–V3, but with no other ECG markers or other findings of arrhythmogenic right-ventricular cardiomyopathy.

Patients with non-inducible VT

Table 2 summarizes the VT induction protocol for the 25 VTni patients. All received isoproterenol and/or epinephrine. The average maximal dose of isoproterenol infusion was slightly higher in the VTni group than the VTi group although not statistically different [5.3 ± 3.9 μg/min (range 1–16 μg/min) vs. 3.7 ± 2.4 μg/min (range 1–10 μg/min); $P = 0.189$]. Seven patients (two patients in VTi group and five patients in VTni group) received boluses of isoproterenol (range 10–30 μg bolus). Epinephrine (20–100 μg) boluses were administered in addition to an isoproterenol infusion or bolus to five patients in the VTni group.

In the VTni group, PVCs were targeted for ablation (*Table 3*). One morphology of PVC was observed in 19 (76%) patients, whereas six (24%) patients showed more than one morphology of PVCs. During RF application, seven (28%) patients showed increased PVCs or non-sustained VT consistent with automaticity. During RF application,

Table 1 Baseline characteristics

Parameters	VT inducible with SM ($n = 18$)	VT inducible with MM ($n = 11$)	VT non-inducible ($n = 25$)
Age, years	47.2 ± 9.6	65.1 ± 9.3*,**	50.4 ± 13.0
Male	9 (50.0)	10 (90.9)	12 (48.0)
BMI, kg/m^2	30.2 ± 6.5	29.4 ± 6.4	26.8 ± 5.4
Exercise-related VT	7 (38.9)	4 (36.4)	11 (44.0)
Syncope	5 (27.8)	2 (18.2)	6 (24.0)
Hypertension	2 (11.1)	10 (90.9)*	8 (32.0)
Hypercholesterolaemia	3 (16.7)	7 (63.6)*,**	5 (20.0)
Diabetes mellitus	2 (11.1)	1 (9.1)	3 (12.0)
ICD implantation	4 (22.2)	5 (45.5) **	2 (8.0)
History of cardioversion	4 (22.2)	8 (72.7)*,**	3 (12.0)
Prior VT ablation	8 (44.4)	5 (45.5)	9 (36.0)
Number of prior VT ablation	0.8 ± 1.1	1.1 ± 1.8	0.5 ± 0.8
Number of failed anti-arrhythmic agents	0.9 ± 1.2	1.4 ± 0.9**	0.6 ± 0.8
Medication			
Amiodarone	4 (22.2)	9 (81.8)*,**	1 (4.0)
Sotalol	4 (22.2)	3 (27.3)	2 (8.0)
Class I anti-arrhythmic agents	4 (22.2)	0 (0)	7 (28.0)
Beta-blocker	13 (72.2)	11 (100)**	16 (64.0)
Calcium channel blocker	5 (27.8)	4 (36.4)	9 (36.0)
LV ejection fraction, %	62.1 ± 5.0	59.8 ± 9.2	59.1 ± 7.1
LGE in Cardiac MRI	0/4 (0)	4/5 (80.0)**	0/8 (0)

ICD, implantable cardioverter defibrillator; LGE, late-gadolinium enhancement; SM, single morphology; MM, multiple morphology.
*$P < 0.017$ compared with SM group.
**$P < 0.017$ compared with VT non-inducible.

Table 2 VT induction protocol

Protocol, *n* (%)	VT inducible with SM ($n = 18$)	VT inducible with MM ($n = 11$)	VT non-inducible ($n = 25$)
Spontaneous VT	1 (5.6)	0 (0)	0 (0)
PES only	5 (27.8)	8 (72.7)*	0 (0)
PES and isoproterenol	12 (66.7)	3 (27.3)**	19 (76.0)
PES, isoproterenol, and epinephrine	0 (0)	0 (0)	6 (24.0)

PES, programmed electrical stimulation; SM, single morphology; MM, multiple morphology.
*$P < 0.017$ compared with SM group.
**$P < 0.017$ compared with VT non-inducible.

non-sustained VT or PVCs became quiescent in six (24%) patients (Supplementary material online, *Figure S1*). After the final ablation, 13 (52%) patients showed complete abolition of targeted PVCs, whereas 12 (48%) patients showed a few residual PVCs.

Ablation sites are shown in *Table 4* and *Figure 1*. The most common ablation site was the RVOT (57%), with fewer at the great cardiac vein, below the aortomitral continuity, sinus of Valsalva, and LVOT. There was no significant difference in distribution of ablation target sites comparing the VTni and VTi groups.

Inducible VT and comparison between MM and SM group

Of the 29 VTi patients, 18 (62%) had a single morphology of VT (SM group), whereas 11 (38%) patients had multiple VT morphologies. The MM group had characteristics suggesting scar-related arrhythmias, as previously reported.[5] The MM group had more VTs inducible by PES without catecholamine stimulation compared with the SM group (73 vs. 28%, $P = 0.014$). In the MM group, different QRS morphologies were observed prior to any ablation in nine patients, and only after initial ablation in two patients, raising the possibility that ablation altered the arrhythmia exit. Detailed procedure of these these patients are described in the Supplementary material online.

The MM group were more frequently found to have abnormal electrograms (fractionated, late, or double potentials) at targeted sites than SM group (82 vs. 22%, $P<0.001$). A low-voltage area (< 1.5 mV) was identified in the ablation region in 91% of the MM group vs. 18% of the SM group and 8% of patients without inducible sustained VT ($P < 0.001$). Half (seven of 13) of the patients who had low-voltage area had had prior ablation procedures with lesions likely placed in the same area. The MM group had significantly longer

Table 3 Electrophysiological characteristics

Parameters	VT inducible with SM ($n = 18$)	VT inducible with MM ($n = 11$)	VT non-inducible ($n = 25$)
Inducible VT			
TCL, ms	320.6 ± 97.1	334.0 ± 47.4	
Haemodynamic unstable VT	1 (5.9)	4 (36.4)	
Number of inducible VT	1.0	2.6 ± 0.5*	
Number of spontaneous or induced PVCs	0.9 ± 0.4	1.0 ± 0.6	
Ablation procedures			
Abnormal electrograms at targeted site	4 (22.2)	9 (81.8)*,**	6 (24.0)
VT termination during ablation	7/12 (58.3)	5/8 (62.5)	
Presence of low-voltage area in voltage mapping	1 (5.6)	9 (81.8)*,**	3 (12.0)
S-QRS interval at best pace map site, ms	35.8 ± 9.5	63.1 ± 15.8*,**	46.1 ± 24.0
Activation time at the best ablation site, ms	−35.4 ± 5.0	−33.3 ± 10.4	−31.4 ± 6.3
Number of ablation applications	8.0 ± 6.2	15.0 ± 7.9	11.4 ± 8.7
Ablation time, min	6.8 ± 5.1	16.8 ± 7.4*	10.5 ± 6.4
Fluoroscopy time, min	30.9 ± 30.3	46.6 ± 17.7**	26.6 ± 14.4
Both endocardial and epicardial approach	3 (16.7)	4 (36.4)	0 (0)

Abnormal electrograms were defined as fractionation, late potentials, or double potentials.
TCL, tachycardia cycle length; S-QRS, interval between stimulation to QRS onset; SM, single morphology; MM, multiple morphology.
*$P < 0.017$ compared with SM group.

Table 4 Targeted sites of ablations

Sites, number (%)	VT inducible with SM (18 sites)	VT inducible with MM (18 sites)	VT non-ndducible (27 sites)
RVOT	15 (83.3)	3 (16.7)*	18 (66.7)
LVOT	0 (0)	6 (33.3)	3 (11.1)
Sinus of Valsalva	1 (5.6)	7 (38.9)	4 (14.8)
Aortomitral continuity	0 (0)	2 (11.1)	1 (3.7)
Great cardiac vein	2 (11.1)	0 (0)	1 (3.7)

RVOT, right-ventricular outflow tract; LVOT, left-ventricular outflow tract; SM, single morphology; MM, multiple morphology.
*$P < 0.017$ compared with SM group.

stimulation to QRS during pace mapping than SM and VTni groups (63.1 ± 15.8 vs. 35.8 ± 9.5 vs. 46.1 ± 24.0 min, all $P < 0.001$). Activation time at the best ablation site tended to be earlier in SM group than VTni group, but this did not reach statistical significance (−35.4 ± 5.0 vs. −31.4 ± 6.3 ms, $P = 0.026$).

Cardiac MR imaging was available in 17 patients (eight VTni, four SM, and five MM) patients. Late-gadolinium enhancement was seen in the periaortic region in four of five MM patients, but in none of the SM or VTni patients. Ablation time was longer in the MM than in the SM group (16.8 ± 7.4 vs. 6.8 ± 5.1 min, $P = 0.009$). Further details of ablation procedures are described in supplement.

Acute results and recurrence after ablation

Figure 2 summarizes the acute results of ablation and recurrence during follow-up according to VT inducibility and morphology. After the final ablation lesion, no VT was inducible in any of the 18 SM group, but VT was still inducible in two patients in MM group (acute failure). In one an epicardial origin of VT was suspected, but

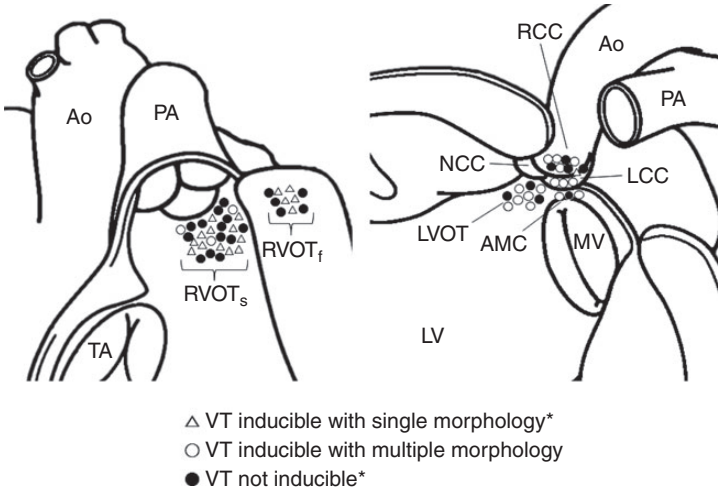

Figure 1 Ablation targeted sites. *Three ablation sites (two from VT inducible with single morphology and one from VT not inducible group) in great cardiac vein were not marked in this figure. AMC, aortomitral continuity; Ao, ascending aorta; LCC, left coronary cusp; LV, left ventricle; LVOT, left-ventricular outflow tract; MV, mitral valve; NCC, non-coronary cusp; PA, pulmonary artery; TA, tricuspid annulus; RCC, right coronary cusp; RVOT$_s$, right-ventricular outflow tract septal side; RVOT$_f$, right-ventricular outflow tract free wall side. (Modified from Das MK, Zipes DP. Electrocardiography of Arrhythmias, reprinted permission from Elsevier).

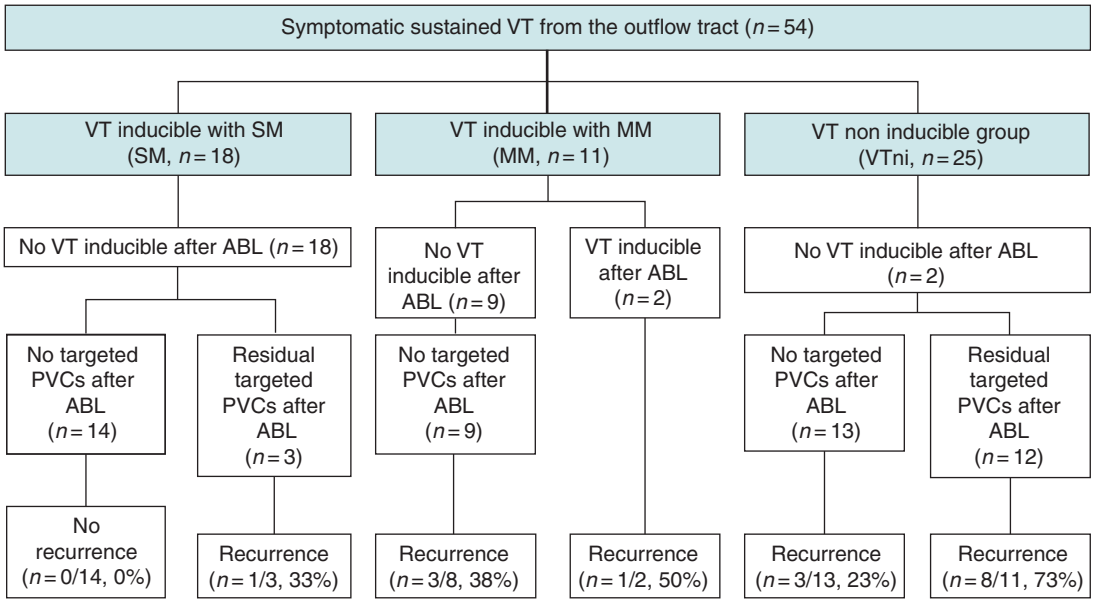

Figure 2 Summary of recurrence according to acute result of ablation and Inducibility of VT during electrophysiology study.

attempted pericardial puncture failed due to pericardial adhesions. In the second an intramural origin near the aortic annulus was suspected.

During a mean waiting time after the final ablation of 30.7 ± 13.5 min, PVCs with a morphology consistent with the targeted VT or PVCs were seen in 15 (27%) patients. PVCs were more often present in the VTni group (52%) than in the VTi group (7%, $P < 0.001$). There were no significant complications related to ablation.

During follow-up of 21 ± 19 months, 16 of 51 (31%) patients had arrhythmia recurrences. These were sustained VT in 13 patients (one patient in SM group, four patients in MM group, and eight patients in VTni group), and non-sustained VT or similar symptoms as before ablation which subsequently led to a second procedure in three patients, all of whom were in the VTni group.

In the VTni group, patients who had a residual targeted PVC present after the final ablation lesion had more frequent recurrences than those who had no residual targeted PVC had recurrences (73 vs. 23%, $P = 0.001$ by log-rank test). Thus, the VTni group had a higher recurrence rate than the VTi group (46 vs. 18%; $P = 0.002$ by log-rank test; *Figure 3A*). Within the VTi group, more MM patients recurred compared with the SM patients (36 vs. 18%, $P = 0.044$ by log-rank test; *Figure 3B*).

In a Cox proportional hazards model adjusting for the presence of scar and the presence of residual PVCs after ablation, VT inducibility was an independent predictor of recurrence [hazard ratio 4.45 (95% confidence interval 1.38–14.35), $P = 0.012$].

QRS morphology of VT and PVCs during electrophysiology study

Of the 29 patients with inducible VT, 25 also showed PVCs during the procedure. One morphology of PVCs was seen in 22 patients and three patients showed two different morphologies of PVCs.

In the SM group ($n = 18$), 17 PVCs were recorded in 16 patients during the procedure with one PVC morphology in 15 and two morphologies in one patient, respectively. The QRS morphology of 14 (82%) PVCs matched that of induced VT, whereas three (18%) differed from the induced VT (*Figure 4*).

In the MM group ($n = 11$), 11 PVCs were observed in nine patients. Two of nine patients had two different morphologies of PVCs. In comparing the morphology of the PVCs and VTs, seven (64%) PVCs in six patients were the same as a VT, whereas four (36%) PVCs in four patients were different from the induced VT. In the SM group, 13 of 18 VTs had PVCs that were the same as the VT, whereas only seven of 29 VTs in the MM group had an observed similar PVC (72 vs. 24%, $P = 0.002$).

Discussion

Inability to induce the target VA is a major factor contributing to failure of catheter ablation in patients with OT arrhythmias. This study emphasizes that clinical perception and further shows that targeting spontaneous PVCs in lieu of inducible VT is associated with a less successful outcome compared with ablation guided by inducible VT. Furthermore, the study defines three groups of patients who present with sustained VT felt to originate in the right- or left-ventricular OT regions, who have normal ventricular function, but whose outcome with ablation are different. Also, the patients who had multiple morphologies of sustained VT have a higher recurrence rate and more often evidence of possible ventricular scar, as we have previously reported.[5]

The absence of inducible VT is likely dependent on the vigour of attempts to induce VT. Programmed stimulation with burst pacing and extrastimuli combined with isoproterenol infusion is routinely employed by many laboratories. The effect of beta-adrenergic stimulation is often difficult to predict and in many cases, the arrhythmia emerges during the washout phase of isoproterenol administration suggesting that the heart-rate acceleration may overdrive suppress the arrhythmia in some cases. We also routinely employ boluses of

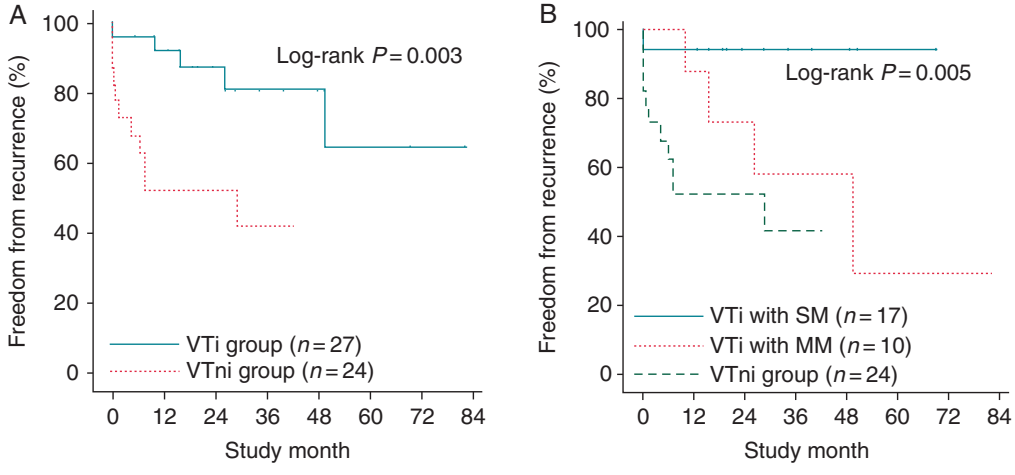

Figure 3 Kaplan–Meier curves showing the recurrence of sustained VT and second ablation procedure due to non-sustained VT or symptoms. (*A*) Recurrence in patients with VT inducible (VTi) and non-inducible (VTni). (*B*) Recurrence in patients with VTi with single morphology, VTi with multiple morphology, and VTni.

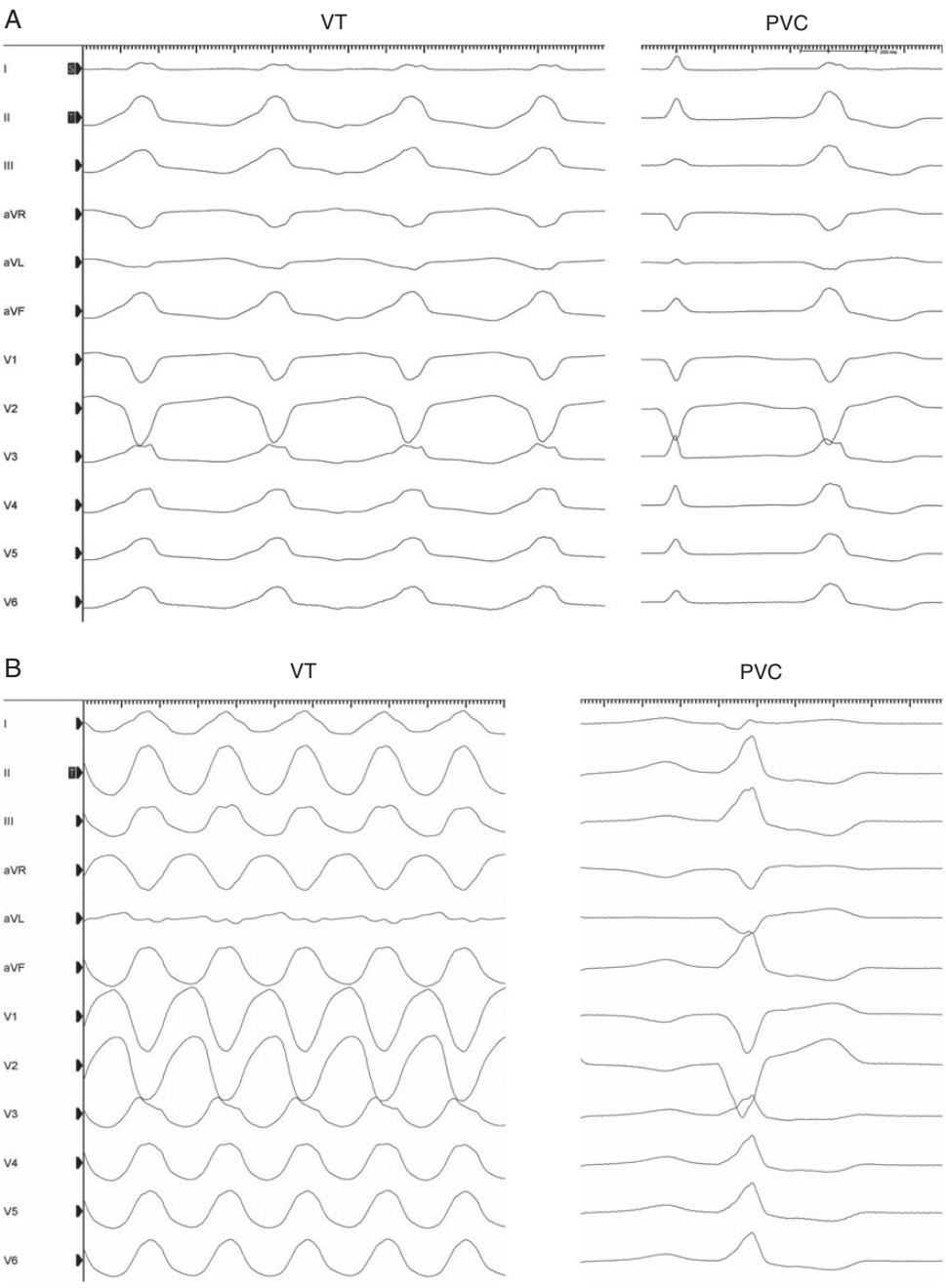

Figure 4 VT and PVC morphologies in the 12-lead ECG from two patients in the VTi group. (*A*) VT that matches the QRS morphology of the PVC, whereas (*B*) shows a different morphology of VT and PVC from the same patient during same procedure.

isoproterenol or epinephrine. Whereas isoproterenol often elicits hypotension, epinephrine increases blood pressure, such that a different magnitude of beta-adrenergic effect may be achievable. The increase in blood pressure may elicit an increase in vagal tone or withdrawal of intrinsic sympathetic tone. Anaesthesia and consious sedation also affect autonomic tone and likely the inducibility of these arrhythmias in some patients. We routinely started with no or minimal sedation, but greater degrees of sedation are required for some patients. Absorption of local anaesthesia can also potentially result in systemic levels (eg of lidocaine) that could potentially suppress arrhythmias.[10] Therefore, we do attempt to use the minimal sufficient amount of local anaesthetic needed for vascular access.

The endpoint of ablation was different according to the inducibility of VT. Although non-inducibility of VT is an appropriate endpoint for ablation in the VT inducible group, it is not feasible for the non-inducible group. Complete abolition of targeted PVCs is a potential surrogate endpoint when VT is not inducible. However, we found

that rare residual targeted PVCs were commonly observed during the waiting period after the final ablation, although the frequency had been decreased significantly and that these patients recurred more frequently than those without residual targeted PVCs. This observation suggests that complete abolition of the PVCs which resemble the clinical VT should be attempted, but whether this would improve the outcome in these pateints is only speculative.

There are several potential contributing factors to the lower success rate of ablation when VT is not inducible. Ablation targeting PVCs is done under the assumption that these originated from the same focus as the VT. Although more than one morphology of spontaneous VA is uncommon in patients without structural heart disease, it does occur,[5] and it is possible that in some cases the PVC targeted is not from the same focus as the VT. In our patients with inducible sustained VT, most of PVCs (75%) were morphologically identical with induced VT. However, we found that half of VTs (27 of 47 induced VTs) were different from the PVC. Furthermore, VT observed in MM group has more frequent non-matchable PVCs than those in SM group, suggesting that PVC-guided ablation strategy would be limited especially in patients who had multiple inducible VTs. Also, one-fourth of VTni group showed multiple morphologies of PVCs, which would be difficult to find the true target site. The activation time at the best ablation site in VTni group (of the targeted PVC) tended to be later than that for VT in the SM group, which further suggests that the PVC-based activation map may not be as likely to identify the origin of VT. A limited number of PVCs makes mapping difficult in some patients. It is also possible that the PVCs were more likely to originate from an intramural location that was difficult to reach with ablation in VTni group.

Activation mapping is generally accepted to be more accurate for selecting ablation sites than assessment of QRS morphology of the PVCs and pace-mapping. When PVCs were infrequent, pace-mapping was used to guide ablation, although we always tried to combine this with some limited activation mapping as allowed by the frequency of the PVCs. From the time the patient is connected to the EP laboratory recording equipment, all spontaneous arrhythmias were recorded. At some sites, pace-mapping can produce a similar QRS morphology over an area of 1–2 cm.[11] In the aortic sinuses, isolated potentials are a better marker of the successful ablation site; pace-mapping can produce a markedly different QRS than the VT and be misleading.[12] The high recurrence in patients with multiple morphologies of VT might be explained by more difficult ablation, consistent with longer ablation time, fluoroscopy time, and number of failed prior procedures. Also, the change of morphology of VT after ablation would suggest the possibility of an origin deep to the endocardium and the potential of ablation to modify the exit from the focus without abolishing the focus. Furthermore, the QRS morphology of VT, during repetitive ventricular activation, may be somewhat different than the morphology of PVCs from the same focus, further confounding morphology assessment for predicting target sites.

Limitations

The number of study patients is relatively small. Although non-sustained arrhythmias are more common than sustained VT in patients without structural heard disease, we only included those with documented sustained VT originating from the OT region and no overt structural heart disease to test our hypothesis that VT inducibility has clinical implications. The results of our study should not be extrapolated to those who only had idiopathic frequent PVC or non-sustained VT. The mechanims of VT was not clearly determined using all electrophysiological manoeuvres including entrainment mapping and pharmacological tests due to our focus on activation mapping and desire to avoid terminating the induced tachycardia. The characteristics of our study population differs from previous idiopathic VT studies.[12–14] Many of our patients had an ICD implanted or a history of syncope, and half of our patients already had failed ablation in other institutions.

Although there was no evidence of overt structural heart disease, a focal or limited cardiomypathic process cannot be excluded. Four of our patients with multiple VT morphologies showed late-gadolinium enhancement on cardiac MRI, suggesting this possibility. Low electrogram voltage areas were seen in 13 patients suggesting there might be subclinical structural heart disease. However, the specificity of low voltage adjacent to a valve annulus as an indicator of scar is not clear. Cardiac MR imaging can also have difficult distinguishing scar from fibrous annulus in this area. Patients with sustained VT may be more likely to have underlying structural heart disease than those with only non-sustained arrhythmias. We usually do perform RV bipolar endocardial voltage maps in patients with RVOT VT and excluded patients with large areas of low voltage ($<$1.5 mV), felt to indicate possible arrhythmogenic cardiomyopathies. We did not perform LV endocardial voltage maps in every patient.

Conclusion

Sustained VT originating from the OT region is not inducible in a substantial number of patients without overt structural heart disease despite documented spontaneous sustained VT. In the absence of sustained VT, PVCs can be targeted for ablation, but recurrences of VT are more common with this approach, compared with ablation guided by inducible sustained VT. Multiple morphologies of induced sustained VT are often associated with evidence of potential perivalvular scar and a higher likelihood of recurrences compared with those with SM of sustained VT. These findings suggest that aggressive attempts to induce VT to guide ablation seem reasonable in this population. When VT is not inducible, complete ablation of targeted OT PVCs seems a reasonable goal. Novel methods to increase inducibility would be of interest for future study.

Conflict of interest: W.G.S. is a coholder of a patent for the needle catheter, rights assigned to Brigham and Women's Hospital.

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Ventricular tachycardia originating from the right ventricular outflow tract in a patient with dextrocardia

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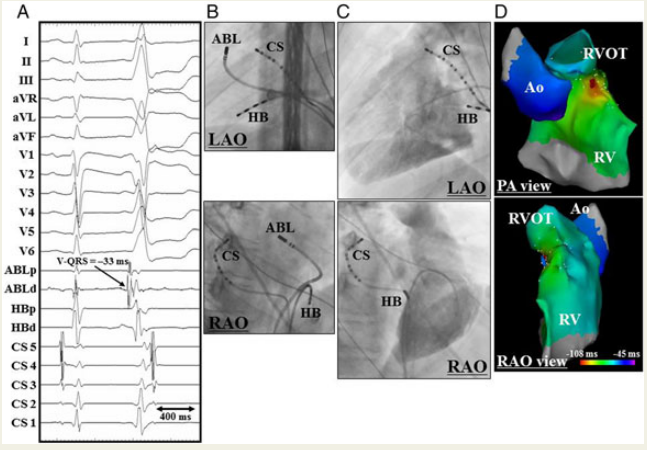

A 38-year-old woman with dextrocardia with situs inversus (D-SI) underwent catheter ablation (CA) of idiopathic premature ventricular contractions and non-sustained ventricular tachycardia (VT). Electrocardiograms were recorded with the electrodes placed in reversed positions and VT exhibited left bundle branch block (LBBB) and inferior axis QRS morphology with precordial transition between leads V3 and V4 (*Figure, panel A*). With a guidance of right ventriculogram and electroanatomical mapping, CA was successful at the right ventricular outflow tract (RVOT) free wall (*Figure, panels B–D*).

Catheter ablation is always challenging in patients with congenital heart disease not only because of unfamiliar anatomy but also because interpretation of the electrocardiogram is rendered challenging by abnormal anatomy. This report illustrated successful CA of RVOT VT in a patient with D-SI. In normal hearts, electrocardiographic algorithms such as LBBB with late precordial transitions, and low and narrow initial R waves in leads V1 and V2 can predict idiopathic RVOT VTs from left ventricular outflow tract VTs. R waves in lead I suggest VT origins in the RVOT free wall. Therefore, this report suggests that standard electrocardiographic algorithms may be valid for predicting RVOT VT origins in patients with a mirror image dextrocardia when the electrodes are placed in a mirror image (reversed) position.

The full-length version of this report can be viewed at: http://www.escardio.org/Guidelines-&-Education/E%E2%80%93learning/Clinical-cases/Electrophysiology/EP-Case-Reports.