Significance of Inducible Nonsustained Ventricular Tachycardias After Catheter Ablation for Ventricular Tachycardia in Ischemic Cardiomyopathy

BACKGROUND: Noninducibility of sustained monomorphic ventricular tachycardia (SMVT) postablation does not insure absence of later recurrence in patients with ischemic cardiomyopathy. This study aims to determine the relation between inducible nonsustained VT postablation and VT recurrences.

METHODS AND RESULTS: One hundred sixty-five consecutive patients (156 male; age 68±9 years) underwent ablation for SMVT because of ischemic cardiomyopathy; 44 patients who did not have induction testing or in whom only ventricular fibrillation was induced after ablation were excluded. In 38 patients (23%), SMVT was inducible (group C). Of the 83 patients without inducible SMVT after ablation, nonsustained VT defined as ≥5 beats lasting for <30 s, was induced in 34 patients (group B, 21%), whereas the remaining 49 patients had no VT induced by the induction test (group A, 30%). Over a median follow-up of 18.7 months, freedom from recurrent VT at 24 months was 60% in group A, 45% in group B (P=0.017 versus group A), and 38% in group C (P=0.005 versus group A). In patients without inducible SMVT, inducible nonsustained VT and left ventricular ejection fraction was independently associated with VT recurrence (hazard ratio, 3.66 and 1.07; 95% CI, 1.3–11.1 and 1.01–1.14).

CONCLUSIONS: Inducible nonsustained VT postablation suggests the continued presence of functional arrhythmia substrate. Further trials are needed to assess whether additional ablation would improve outcome in this group.

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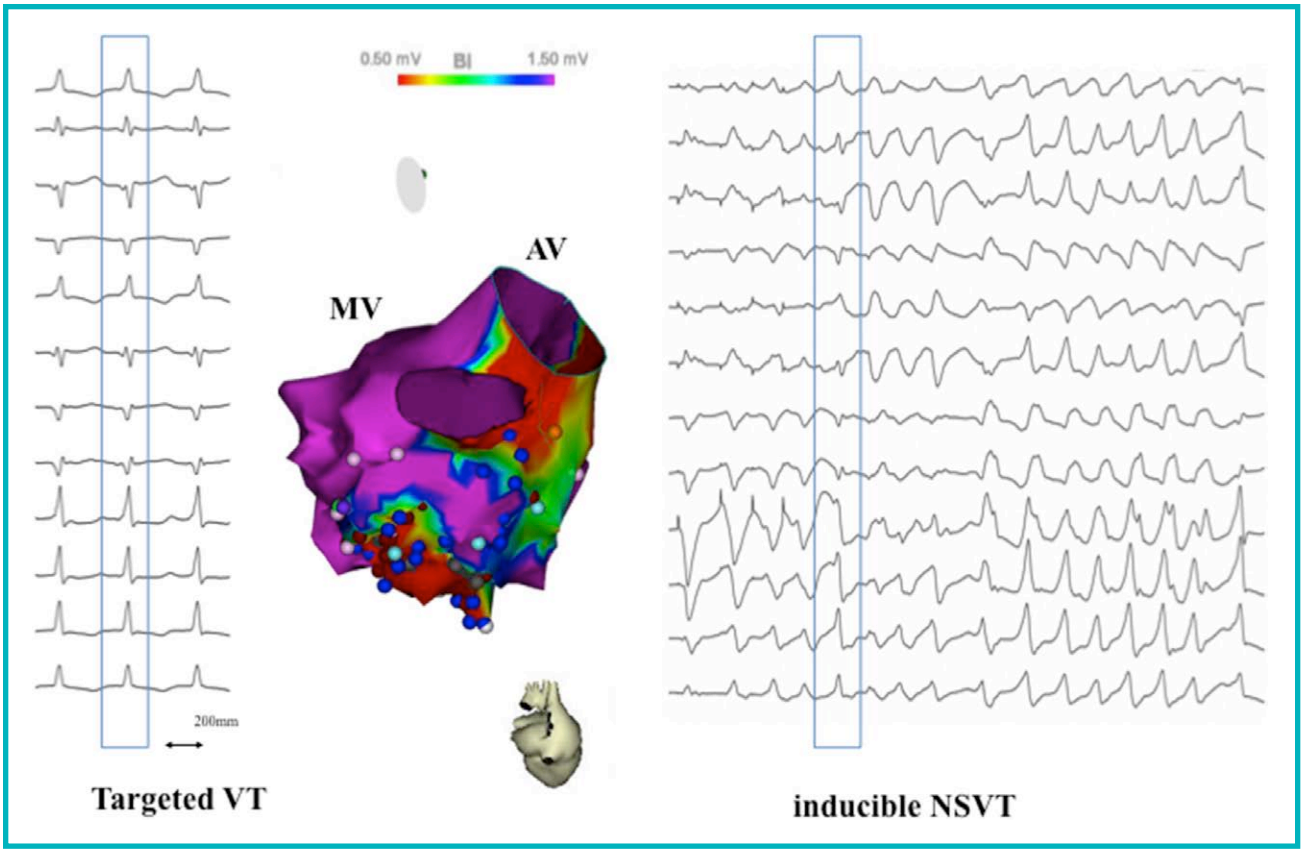

WHAT IS KNOWN?

- Acute prevention of ventricular tachycardia (VT) inducibility after catheter ablation seems a desirable but insufficient acute end point.

WHAT THE STUDY ADDS?

- Five beats or more of nonsustained VT was induced in 41% of patients who did not have sustained VT induced after catheter ablation.
- Inducible nonsustained VT was associated with a risk for VT recurrence similar to that of patients who had sustained monomorphic VT induced after VT ablation.

Radiofrequency catheter ablation is a useful therapy to reduce episodes of ventricular tachycardia (VT) in patients with ischemic cardiomyopathy. The first goal of the procedure is usually to abolish inducible clinical VTs, defined as those that have occurred spontaneously. Many laboratories also aim to abolish all inducible sustained monomorphic VTs (SMVTs), particularly because the morphologies of spontaneous VTs terminated by implanted defibrillators are often not known. This goal has been associated with a lower risk of spontaneous VT recurrence..[1,2] Unfortunately, recurrences of VT are not uncommon despite acutely successful ablation.[3,4]

Nonsustained VT (NSVT) is felt to be a nonspecific response to programmed stimulation, but inducible NSVT postablation has not been extensively evaluated in relation to outcomes of ablation for SMVT.[5] We hypothesized that the induction of NSVTs may also indicate the presence of some arrhythmogenic substrate and may be associated with a risk of spontaneous recurrent sustained VT.

A second consideration in using VT inducibility as an end point for some patients is the concern that inducing sustained VT at the end of an ablation procedure may unnecessarily subject the patient to additional hemodynamic stress. If inducibility is being used to determine if further ablation lesions will be placed, and the stimulation protocol could be terminated when NSVT is encountered, some patients may be spared induction of sustained VT/ventricular fibrillation (VF).

The aim of this analysis is to determine whether there is a relation between inducible NSVT and sustained VT recurrences and to assess the characteristics of inducible NSVT as a predictor for VT recurrence.

METHODS

Patient Characteristics

From January 2011 to June 2015, 165 consecutive patients (156 male; age 68±9 years) who underwent their first radiofrequency ablation at our institution for SMVT associated with ischemic cardiomyopathy were included. Patients who

did not have programmed stimulation reinduction test after VT ablation (35 patients) or who had only inducible VF at reinduction testing (9 patients) were excluded (Figure 1; Table I in the Data Supplement). Only the first ablation procedure at our institution was analyzed. Each patient gave written informed consent for the procedure. Studies and data collection were performed according to protocols approved by the Human Research Committee of Brigham and Women's Hospital.

Electrophysiological Study and Ablation

Procedures were performed either under conscious sedation or general anesthesia (Table 1). Multipolar electrode catheters were positioned in the right ventricular (RV) apex and the His bundle region through femoral venous and arterial access. Programmed ventricular stimulation for initiation of VT was performed with upto 3 extrastimuli scanned to refractoriness or a minimum coupling interval of 180 ms, applied after 2 basic drives (600 ms and then 400 ms) and burst pacing from a minimum of 2 RV or an RV and an left ventricular (LV) site. In 9 cases without VT induced by the above protocol, programmed RV or LV stimulation with upto 4 extrastimuli after basic drive of 350 ms was performed.

Electroanatomical mapping was performed using a 3.5-mm-tip open irrigated catheter (NaviStar ThermoCool, Biosense Webster, Diamond Bat, CA) or a multipolar, multispline diagnostic catheter (Pentaray NAV, Biosense Webster, Diamond Bat, CA) with the CARTO mapping system (CARTO3, Biosense Webster). In the electroanatomical mapping system, bipolar electrograms were high-pass filtered at 20 to 30 Hz and low-pass filtered at 400 Hz. Bipolar electrograms were also band-pass filtered from 30 to 500 Hz and digitally recorded along with a 12-lead surface ECG using the Cardiolab EP system (General Electric Healthcare, Buckinghamshire, United Kingdom). Voltage maps were created during sinus or paced rhythm. Peak-to-peak bipolar electrogram amplitude <0.5 mV was defined as dense scar, voltage ≥0.5 and <1.5 mV as scar border zone.[6] Maps included higher density points around areas of scar, focusing on the scar border and electrograms within the scar. Normal voltage regions were less densely mapped.

Ablation targeted all inducible SMVTs, initially focusing on clinical VTs or presumptive clinical VTs (if 12-lead ECGs were not available), and including VTs with a similar or slower cycle length to clinical VTs. Remaining VTs with shorter cycle lengths than the clinical or presumptive clinical VTs were then targeted depending on whether potential substrate areas for these VTs were present, and the tolerance of the patient for continuing the procedure as assessed by the treating physicians. If VT was hemodynamically tolerated and reproducibly induced, mapping and ablation was performed during VT. Sites where pacing entrained the VT with concealed fusion and a postpacing interval within 30 ms of the VTCL or an isolated mid-diastolic potential or presystolic potential was present in the absence of entrainment were targeted for ablation. If VTs were unmappable because of hemodynamic intolerance or poor reproducibility, ablation was guided by substrate mapping during sinus rhythm with limited or no assessment during VT, targeting presumptive channels and exits within low-voltage (<1.5 mV) areas identified from a paced QRS morphology similar to the VT QRS morphology, abnormal fractionated potentials, double potentials or late potentials during sinus or paced rhythm at sites where pacing captured, particularly with stimulus-QRS intervals of >40 ms. Pace mapping and entrainment mapping used unipolar stimuli at 10 mA and pulse width of 2 ms.[7]

Radiofrequency energy was delivered with the irrigated catheter at a power of 25 to 50 W targeting an impedance drop of 10 to 20 Ω. Applications at target regions were usually repeated until unipolar pacing at 10 mA at 2 ms stimulus strength failed to capture, although this was not always achieved at the border zone areas.[7]

Epicardial mapping was considered if a subepicardial VT origin was suspected based on endocardial mapping and was performed either at the same or a subsequent session. Percutaneous subxiphoid epicardial access was obtained as previously described, either before administration of systemic anticoagulation or after anticoagulation was reversed to achieve an activated clotting time of <200 s.[8]

At the end of the procedure, the same stimulation protocol was repeated. In 86 patients, programmed stimulation was also performed from a LV site after ablation. Acute complete success was defined as the absence of any inducible SMVT. These patients were further divided into 2 groups;

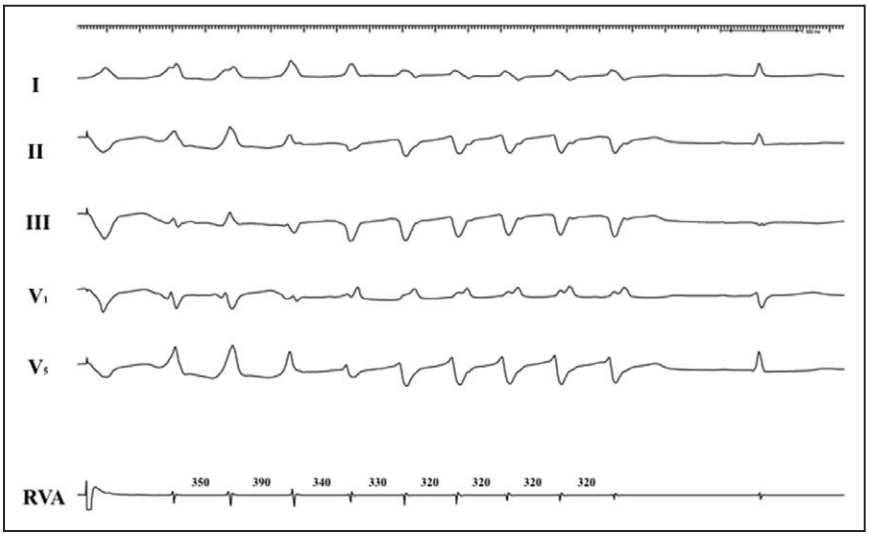

Figure 1. Induced nonsustained ventricular tachycardia. Monomorphic nonsustained ventricular tachycardia having 5 consecutive beats with uniform QRS configuration after 4 polymorphic beats. RVA indicates right ventricular apex.

Table 1. Patient Characteristics According to Inducible VT at the End of the Ablation Procedure

	Group A: No VT (n=49)	Group B: NSVT (n=34)	Group C: Sustained VT (n=38)
Age, y	68±11	68±11	68±13
Male	47 (99)	33 (97)	36 (95)
Body mass index, kg/m^2	28.8±5.5	27.2±3.4	27.8±4.0
NYHA≥III	16 (39)	10 (31)	14 (39)
Time since MI, mo	180 (84–300)	174 (93–258)	216 (132–273)
No. of vessels	2.2±0.8	2.1±0.9	2.2±0.8
PCI	30 (61)	17 (50)	55 (59)
CABG	29 (64)	15 (48)	27 (71)
Prior ablation	14 (29)	11 (32)	13 (34)
VT storm	20 (44)	14 (41)	17 (45)
ICD	49 (100)	32 (94)	35 (92)
CRT	23 (47)	6 (18)*	13 (34)
Failed amiodarone	37 (76)	26 (76)	31 (84)
LVEF, %	29.4±8.8	34.9±11.5*†	27.1±9.3
Amiodarone at discharge	22 (45)	15 (44)	24 (63)
General anesthesia	46 (94)	32 (94)	34 (89)
No. of induced VTs	2 (1–3)‡	2 (1.8–3)†	4 (2.8–5)
Epicardial ablation	3 (6)	2 (6)	3 (8)
Use of multipolar catheter	7 (14)	6 (18)	4 (11)
No. of extrastimuli for final induction test	2.9±0.4‡	2.7±0.4	2.4±1.1
RF ablation time, min	33±14	37±19	42±19
Fluoroscopy time, min	27±12	29±14	32±14
Procedure time, min	214±48§	209±60	259±82

Values are mean±SD, median (25th–75th interquartile range), or n (%). CABG indicates coronary artery bypass graft; CRT, cardiac resynchronization therapy; ICD, implantable cardiac defibrillator; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSVT, nonsustained VT; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; RF, radiofrequency; and VT, ventricular tachycardia.

*Bonferroni-adjusted P<0.05 no VT vs NSVT.

†Bonferroni-adjusted P<0.01 NSVT vs sustained VT.

‡Bonferroni-adjusted P<0.01 no VT vs sustained VT.

§Bonferroni-adjusted P<0.05 no VT vs sustained VT.

those without NSVT (defined below; group A) and those with NSVT induced (group B). The patients with any SMVT inducible were classified as group C.

Analysis of Induced NSVT

In patients without inducible sustained VT, inducibility of NSVT was assessed. NSVT was defined as 5 beats to 30 s of VT that did not require termination within that period for hemodynamic compromise. The first episode of NSVT encountered was analyzed, noting the point in the stimulation protocol that induced the NSVT. The characteristics of the first episode of NSVT analyzed included the coupling interval from the stimulus artifact to the onset of the NSVT in either the intracardiac or surface ECG recordings (whichever was earlier) and the QRS morphology. NSVT was defined as monomorphic when there were ≥3 consecutive ventricular beats with the same monomorphic QRS configure (Figure 1). Polymorphic NSVT was defined as having a changing QRS morphology with <3 monomorphic ventricular beats. The QRS morphology of the first beat and any monomorphic beats were compared with that of sustained VTs observed during the procedure. Additionally, we compared these morphologies to that of the paced QRS during reinduction testing. Finally, the potential origin (exit) of the first NSVT relative to low-voltage (<1.5 mV bipolar) areas was estimated based on the QRS morphology as follows:

- VT with left bundle branch block in V1 was considered to indicate a LV septal origin or RV origin.
- A frontal plane axis directed inferiorly, with dominant R wave in II, III, and aVF, indicated an exit in the anterior wall of the ventricle. A superiorly directed frontal plane axis indicated origin on the inferior wall of the ventricle.
- A rightward frontal plane axis suggested a left lateral wall or apical position. In the midprecordial leads (V_3 and V_4), dominant R waves indicate a location close to the base of heart; dominant S waves indicated an apical location.

Data Collection and Follow-Up

Data were collected from a centralized system containing records of all patients treated and followed at Brigham and Women's Hospital and all associated Partners Healthcare sites. These records include emergency department visits, outpatient clinic visits, and data recorded during inpatient care as well as follow-up progress notes from referring physicians monitoring out-of-area patients. In addition, referring cardiologists and primary care physicians were contacted for clinical follow-up of their patients if necessary. For the purpose of this study, follow-up ended at the time of first recurrence of SMVT and all-cause death.

Statistical Analysis

Continuous variables were expressed as mean±SD values or median and the 25th and 75th percentiles are shown in parentheses, as appropriate. Student t test or Mann–Whitney U test was used to compare continuous variables, depending on whether the values were normally distributed, and the χ^2 test was used to compare dichotomous variables unless the expected values in any cells were <5, in which case Fisher exact test was used. Survival curves were created using the Kaplan–Meier method, and comparisons between groups performed using the Gehan generalized Wilcoxon test which gives more weight to early events because we theorized that if inducible NSVT reflected the continued presence of arrhythmia substrate, VT recurrences would be expected to occur early. P<0.05 was considered to be statistically significant. Cox proportional hazards analyses were used to assess the relationship between the inducibility of NSVT and VT recurrence. In comparison among groups A to C, a P value of 0.017 (equivalent to a Bonferroni-adjusted [3 test] P value of 0.05) was considered significant. All statistical analyses were performed with JMP 11 software (SAS Institute, Cary, NC).

RESULTS

Baseline Characteristics

Of the 165 patients with SMVT, 35 patients and 9 patients were excluded because no VT induction test was performed after ablation, and polymorphic VT/VF was induced, respectively (Figure 2). At the end of the procedure, no SMVT was inducible (acute complete success) in 83 patients (53%); NSVT was induced in 34 patients (group B, 41% of the complete success group); and the remaining 49 patients had no NSVT or sustained VT induced (group A, 59% of the acute complete success group). SMVT was inducible in 38 patients (group C, 23%). Baseline characteristics and procedure details are shown in Table 1. LV ejection fraction (LVEF) in patients with NSVT (group B) was higher than in those without NSVT (group A). Fewer patients had cardiac resynchronization therapy devices in group B compared with group A. The number of sustained VT morphologies induced at any time during the procedure increased from group A to group B, and then to group C (median, 2 [1–3] versus 2 [1.8–3] versus 4 [2.8–5]; $P<0.01$ for group B versus C; $P<0.01$ for group A versus group C). The proportion of epicardial ablation was similar among all groups. The maximal number of extrastimuli for final induction test was higher in group A than in group C, but similar between group A and group B.

VT Recurrence and All-Cause Mortality

During a median of 18.7 months of follow-up, VT recurred in 43 patients (36%; Figure 3); the median time to recurrent VT in these patients was 3.2 (0.1–13) months. At 24 months after VT ablation, 60% of patients in group A were free of recurrent VT compared with 45% in group B ($P=0.013$ by Wilcoxon test for group A versus B) and 38% in group C ($P=0.005$ for group A versus C); there was no statistical difference between groups B and C ($P=0.69$). For all-cause mortality, there were no significant differences among the 3 groups (Figure I in the Data Supplement).

In 83 patients with acute complete success (group A and B), a multivariable model, incorporating LVEF, prior ablation, the number of inducible sustained VTs during the procedure and the inducibility of NSVT after ablation, demonstrated that inducibility of NSVT and LVEF was independently associated with VT recurrence. (hazard ratio, 3.66 and 1.07; 95% CI, 1.3–11.1 and 1.01–1.14).

During follow-up, VT storm occurred in 2% (1/49), 15% (5/34), and 16% (6/38) of group A, B, and C, respectively. In group C, 80% of patients with VT recurrence had recurrent monomorphic VTs (Table II in the Data Supplement).

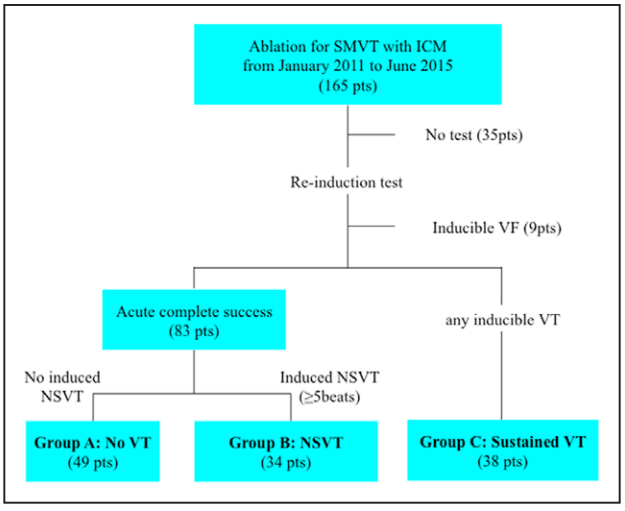

Figure 2. Flow chart of the protocol for patients' enrollment.
ICM indicates ischemic cardiomyopathy; NSVT, nonsustained VT; pts, patients; SMVT, sustained monomorphic VT; VF, ventricular fibrillation; and VT, ventricular tachycardia.

Number of Extrastimuli to Induce NSVT and Sustained VT

In 83 patients without inducible SMVT after VT ablation (acute complete success), no patient had NSVT induced by 1 extrastimulus; 16 (19%) patients had inducible NSVT induced by 2 extrastimuli, 7 of whom had recurrent VT. Three extrastimuli were used in 61 patients who did not have NSVT induced by 1 or 2 extrastimuli, and NSVT was induced in 15 of these patients (25%), 8 of whom had VT recurrence. Four extrastimuli was used in only 4 patients, and induced NSVT in 3 (Figure II in the Data Supplement), none of whom had a VT recurrence.

In the patients who had inducible SMVT after ablation (group C), all 5 patients who had SMVT induced by 1 extrastimuli had recurrent VT; 12 patients had SMVT induced by 2 extrastimuli, 7 of whom had recurrent VT; 13 patients had SMVT induced by 3 extrastimuli, 4 of whom had VT recurrence. Five patients had SMVT induced by 4 extrastimuli, 3 of whom had spontaneous recurrences.

NSVT Characteristics According to VT Recurrence

In group B, VT recurred in 15 (44%) patients. There was no significant difference between patients with and without VT recurrence in baseline characteristics or characteristics of mode of initiation of NSVT, although patients with recurrence tended to have worse ventricular function and functional class (Table 2).

A similar morphology of the first beat of inducible NSVT to that of a VT induced during procedure was more frequently observed in patients with VT recurrence compared with those without VT recurrence (40% versus 5%; $P=0.01$). Examples of the NSVT morphology in patients with VT recurrence and without VT

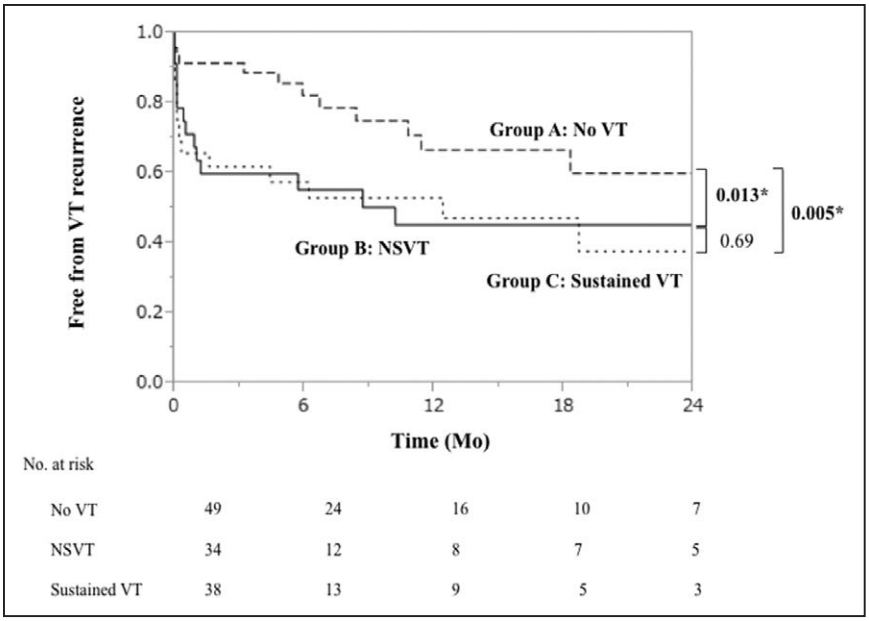

Figure 3. Freedom from ventricular tachycardia (VT). Kaplan–Meier curves showing the recurrence of VT among the 3 groups defined by the response to programmed stimulation after ablation; *P*=0.013 for group A vs B and *P*=0.005 for group A versus C. NSVT indicates nonsustained VT.

recurrence are shown in Figures 4 and 5. The number of beats of inducible NSVT was not significantly related to VT recurrence (median 5 [3–7] versus 6 [3–9]; *P*=0.29). The maximum duration, the rate and initial coupling interval of inducible NSVT was not different between both groups.

Inducible NSVT postablation was monomorphic in 21 patients (62%) and polymorphic in 13 (38%), and this characteristic was not different between those with and without recurrence (Table III in the Data Supplement). We analyzed the morphology of ≥3 consecutive ventricular beats with the same monomorphic QRS configuration within an induced run of NSVT. This QRS morphology was similar to that of a VT induced during the procedure, more frequently in patients with VT recurrence (7 of 15 patients, 47%) compared to those with inducible NSVT without VT recurrence (4 of 19 patients, 21%; *P*=0.11) but this did not reach statistical significance. The potential origin (exit) that was estimated based on the morphology of induced monomorphic NSVT was more frequently consistent with origin from a low-voltage (<1.5 mV bipolar) area in patients with VT recurrence (12 of 15 patients, 80%) compared to those without VT recurrence (9 of 19 patients, 47%; *P*=0.04; Table 2).

Inducible NSVTs Before Initiation of SMVT

Of 38 group C patients with SMVT induced after VT ablation, only 7 patients (18%) had NSVT induced at an earlier step in the stimulation protocol. VT recurred in 4 of the 7 patients, all of whom had inducible monomorphic NSVT. The 1 patient with polymorphic NSVT induced remained free from spontaneous VT.

Morphological Characteristics of Recurrent Sustained VT

In 4 patients with available ECGs of the recurrent sustained VT, the VT morphology was similar to that of monomorphic NSVT in only 1. In the remaining 3 patients, the recurrent VT was similar to an inducible sustained VT observed during procedure, but not the NSVT induced after ablation (Table IV in the Data Supplement).

DISCUSSION

The main findings of this study are as follows: (1) Five beats or more of NSVT was induced in 41% of patients who did not have sustained VT induced after ablation. (2) NSVT was associated with a risk for VT recurrence similar to that of patients who had inducible SMVT. (3) A QRS morphology of the first beat of the NSVT that was consistent with the location of the low-voltage scar was associated with spontaneous VT recurrence. These findings suggest that inducible NSVTs could be an indicator of surviving, clinically relevant VT substrate after ablation.

Recent meta-analysis demonstrated that noninducibility of VT at programmed stimulation after VT ablation is associated with improved arrhythmia-free survival.[2] A significant number of patients without inducible sustained VT postablation, however, still have VT recurrences. The association of inducible NSVT with recurrence of sustained VT suggests that inducible NSVT is an indicator of arrhythmogenic substrate. The nature of the relation of NSVT to the arrhythmia substrate is not certain. Radiofrequency ablation induced edema formation might prevent VT inducibility immediately after ablation, but then resolve after several days, allowing recovery of circuits capable of supporting sustained VT. This mechanism of recurrence is suggested by a previous report, that found that 15%

Table 2. Characteristics of Patients With Inducible NSVT After Ablation According to Subsequent VT Recurrence

	Recurrence (n=15)	No Recurrence (n=19)	*P* Value
Baseline			
Age, y	65±3	70±2	0.22
Male	15 (100)	18 (95)	0.28
Body mass index, kg/m^2	27±4	27±3	0.94
NYHA≥III	6 (40)	4 (21)	0.32
No. of vessels	2 (1–3)	2.5 (2–3)	0.23
Time from MI, mo	138 (33–219)	210 (132–282)	0.16
Scar location			
Anterior	5	3	
Septal	11	14	
Inferior	9	13	
Lateral	7	6	
Apical	8	6	
VT storm	7 (47)	7 (37)	0.56
Failed amiodarone	12 (80)	14 (74)	0.66
LVEF, %	32±8	37±13	0.14
Amiodarone at discharge	6 (40)	9 (47)	0.66
Electrophysiological study and ablation			
No. of VT morphologies induced	2 (2–3)	2 (1–4)	0.90
Epicardial ablation	0 (0)	2 (11)	0.12
First induced NSVT			
LV stimulation	3 (20)	9 (47)	0.09
Baseline PCL≤400 ms	7 (47)	12 (63)	0.34
No. of beats	5 (3–7)	6(3–9)	0.29
Mean cycle length, ms	328±45	372±99	0.13
CI to the last stimulus, ms	374±18	393±99	0.54
The first beat of NSVT			
Similar to pacing QRS morphology	8 (53)	7 (37)	0.34
Similar to area to scar	10 (67)	7 (37)	0.08
Similar to VT induced during procedure	6 (40)	1 (5)	0.01
Monomorphic NSVT	11 (73)	10 (53)	0.21
The monomorphic beats			
Similar to pacing QRS morphology	4 (27)	3 (17)	0.48
Similar to area to scar	12 (80)	9 (47)	0.04
Similar to VT induced during procedure	7 (47)	4 (21)	0.11

Values are the mean±SD, median (25th–75th interquartile range), or n (%). CI indicates coupling interval; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSVT, nonsustained ventricular tachycardia; NYHA, New York Heart Association; and PCL, paced cycle length.

of patients who did not have an inducible VT early after ablation, again had inducible VT a few days later.[9] In our series, 50% of patients with inducible NSVT had a VT recurrence within 1 week after VT ablation. Interestingly, patients with inducible NSVT had similar recurrence rates to those with sustained VT, despite the patients with inducible NSVT appearing to have less severe disease (eg, higher LVEF, less extensive revascularization, lower number of induced VTs). Additionally, in 9 cases with inducible polymorphic VT/VF after ablation the recurrence rate also seemed similar to that for inducible NSVT (Figure III in the Data Supplement), suggesting this arrhythmia may also reflect an arrhythmogenic substrate, but this is only speculative, because of the small number of patients.

We hypothesized that if inducible NSVT reflects the presence of arrhythmia substrate, the morphology of the NSVT may suggest the location of the substrate, and would be more likely to resemble that of a sustained VT. In 40% of the NSVT patients with VT recurrence, the morphology of the first beat of inducible NSVT was similar to that of sustained VTs induced during the procedure. Interestingly, in 80% of patients with inducible monomorphic NSVT with VT recurrence, the morphology of NSVT was consistent with the low-voltage scar location. However, only half of these NSVTs had a morphology similar to that of an induced sustained VT observed during the procedure. We have ECG morphologies of recurrent VT in only a small number of patients, the recurrent VT appeared to often have a different exit than that of the NSVT induced at the end of the procedure. The discrepancies between QRS morphology of induced NSVT, induced SMVT, and recurrent VT do not exclude the possibility that the induced NSVT originates from the same arrhythmogenic part of the infarct as the sustained and recurrent VTs. An exit region for one VT may be an entrance region into the circuit for the other. It is possible that ablation transiently impaired a portion of the circuit that later recovered, allowing it to revolve more easily in the opposite direction. These speculations warrant further study.

Aggressive programmed stimulation, such as with 4 extrastimuli, seems more likely to induce nonspecific arrhythmias and the optimal stimulation protocol for postablation testing has not been defined. In our study, none of the patients with NSVT induced only with 4 extrastimuli had a spontaneous VT recurrence, although this was only a small number of patients. On the other hands, of the 38 group C patients, 5 had SMVT induced by 4 extrastimuli and 3 subsequently had spontaneous recurrences of VT. Further investigation was needed to identify whether additional programmed stimulation from other sites or with more aggressive stimulation protocols would be helpful to predict VT recurrence in patients with only NSVT by less aggressive stimulation.

In 27% of NSVT patients who had VT recurrence, inducible NSVT was polymorphic. Thus, even though polymorphic NSVT can be a nonspecific finding, it may

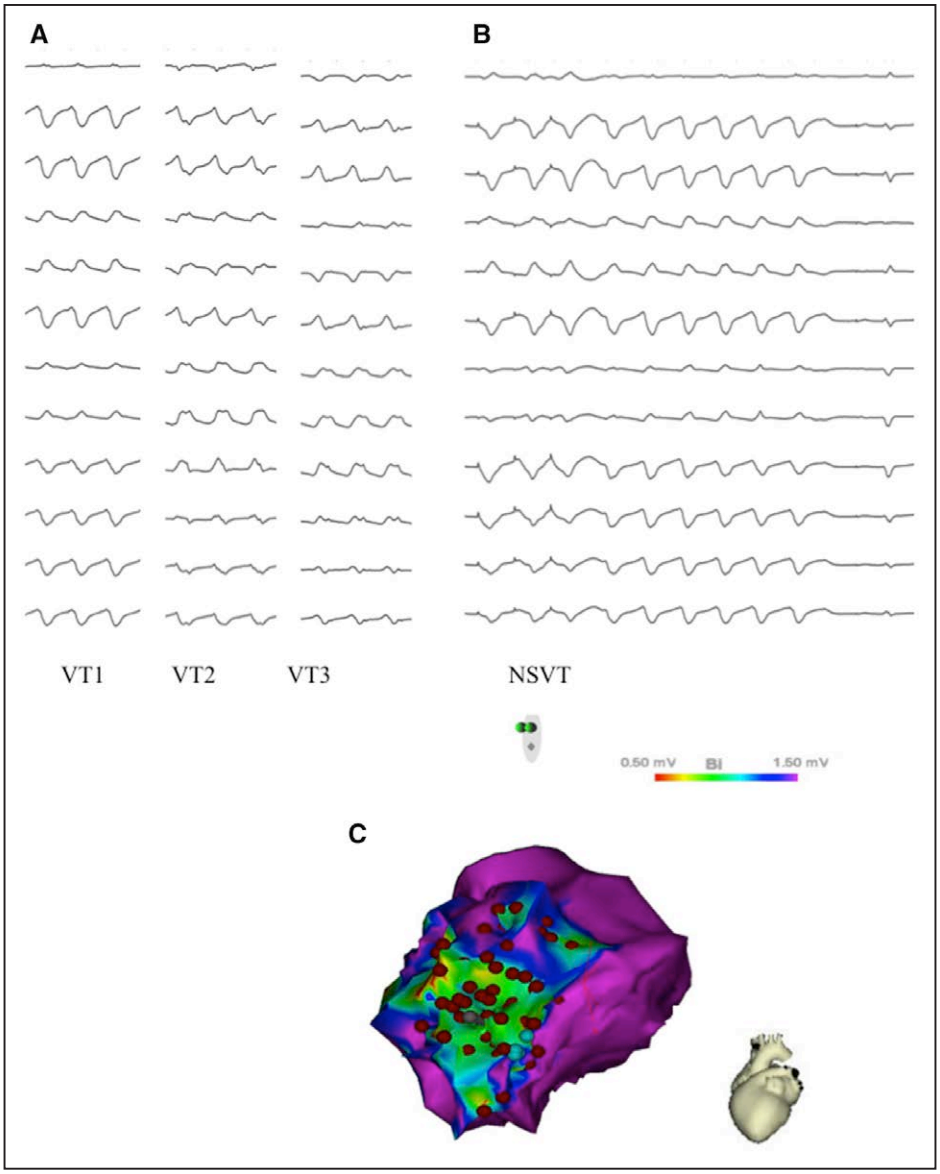

Figure 4. Tracings and mapping data from 1 patient who had monomorphic nonsustained ventricular tachycardia (NSVT) induced at the end of the ablation procedure and subsequently had a VT recurrence.
A, Twelve-lead ECG morphologies of 3 different sustained VTs induced during the procedure and (**B**) NSVT induced at the end of procedure. In the first beat and monomorphic beats of inducible NSVT, both morphologies resemble that of VT1. **C**, Bipolar voltage map of the left ventricle (LV). The purple area indicates voltage area of >1.5 mV and the area of <0.5 mV is colored red. Unexcitable scar are indicated by grey tags. Ablation sites are indicated by dark red tags.

also indicate residual arrhythmogenic substrate in some patients undergoing VT ablation. This consideration is also supported by our observation of recurrences in the small number of patients with only polymorphic VT/VF induced, as stated above. A previous study noted that polymorphic VTs induced in patients without a history of VT were faster and had a longer coupling interval between the last stimulus and first VT beat compared with polymorphic VT induced by programmed stimulation in patients with no structural heart disease or history of VT.[10] These features did not seem to be useful for distinguishing patients who would have recurrences in our study.

Limitations

Although the VT ablation data are collected prospectively, this study was retrospective. Although stimulation protocols were the same within an individual patient, the precise sites of stimulation varied between RV apex, outflow tract, and septum, and also LV. The association of NSVT with recurrences raises the question of whether patients with inducible NSVT might benefit from further ablation targeting any the residual substrate.[11–13] Whether abolition of inducible NSVT, beyond abolishing inducible sustained VT, is achievable and would improve ablation outcomes

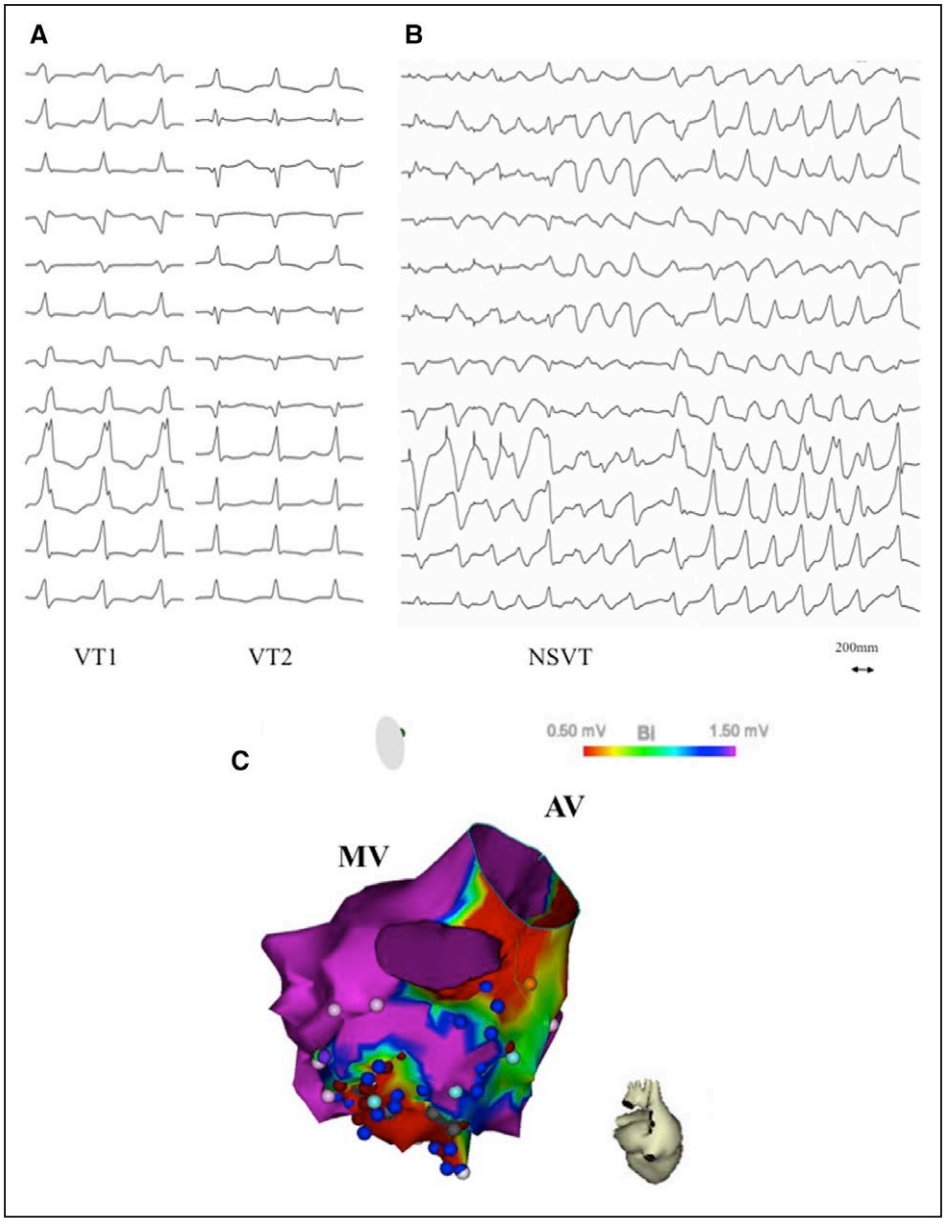

Figure 5. Tracings and mapping data from a patient who had polymorphic nonsustained ventricular tachycardia (NSVT) induced at the end of ablation procedure and subsequently had a VT recurrence after procedure.
A, Twelve-lead ECG morphologies of 2 different sustained VTs induced during the procedure and (**B**) polymorphic NSVT induced at the end of procedure. The morphology of the first beat of inducible NSVT resembles that of VT2. **C**, Bipolar voltage map of the left ventricle. The purple area indicates voltage area of >1.5 mV and the area of <0.5 mV is colored red. Blue tags indicate late potentials and pink tags indicate fragmented potentials. Ablation sites are indicated by dark red tags. Fractionated potentials are indicated by blue tags. AV indicates aortic valve; and MV, mitral valve.

warrants prospective study. The crude origin of NSVT was estimated from the QRS morphology, which is a limited indicator of reentrant VT exit sites. Whether the morphology of the induced NSVT would provide guidance as to the location of residual substrate was not established from our study. The median follow-up was short, as follow-up ended with the first recurrence of VT. The number of patients in subgroups is relatively small and there are some differences among groups, with the inducible sustained VT patients (group C) tending to have worse ventricular function and more inducible VTs.

Conclusions

Inducible NSVT after catheter ablation for SMVT in patients with prior myocardial infarction is associated with an increased risk of sustained VT recurrence. Further study is warranted to assess the underlying reasons for these findings and consider whether additional

ablation in patients with inducible NSVT might improve outcomes of ablation.

AFFILIATIONS

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DISCLOSURES

Dr Stevenson is coholder of a patent for needle ablation that is consigned to Brigham and Women's Hospital and receives consulting fees/honoria from Boston Scientific and Abbott Medical. Dr Tedrow receives consulting fees/honoraria from Boston Scientific Corp and St. Jude Medical and research funding from Biosense Webster, Inc and St. Jude Medical. Dr John receives consulting fees/honoraria from St. Jude Medical. Dr Michaud receives consulting fees/honoraria from Boston Scientific Corp, Medtronic, Inc, and St. Jude Medical and research funding from Boston Scientific Corp and Biosense Webster, Inc.

FOOTNOTES

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Significance of Inducible Nonsustained Ventricular Tachycardias After Catheter Ablation for Ventricular Tachycardia in Ischemic Cardiomyopathy

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SUPPLEMENTAL MATERIAL

Supplemental Table 1. Characteristics of patients excluded

	Patients excluded from this study (n = 44)
Age, yrs	69 ±10
Male	40 (91)
Body mass index, kg/m²	27.3 ± 3.6
NYHA ≥ III	9 (20)
Time since MI, months	144 (90-231)
No. of vessels	2.1± 0.9
PCI	22 (50)
CABG	21 (48)
Prior ablation	17 (39)
VT storm	17 (39)
ICD	43 (98)
CRT	18 (41)
LVEF, %	29 ± 11
No. of induced VT	2 (1-4)
Epicardial ablation	3 (7)
Radiofrequency time, min	41 ± 20
Fluoroscopy time, min	28 ± 15
Procedure time, min	225 ± 73

Values are the mean ± SD, median (25th, 75th interquartile range) or n (%).
NYHA = New York Heart Association; MI=myocardial infarction; PCI= percutaneous coronary intervention; CABG = coronary artery bypass graft; VT = ventricular tachycardia; ICD=implantable cardiac defibrillator; CRT = cardiac resynchronization therapy; LVEF= left ventricular ejection fraction.

Supplemental Table 2. Characteristics of VT Recurrences

	Group A: No VT (12)	**Group B: NSVT (15)**	**Group C: Sustained VT (18)**
VT storm	1/12 (8)	5/15 (33)	6/18 (33)
TCL, ms	375±81	380±104	492±111
Monomorphic VT	5/12(42)	9/15 (60)	12/15 (80)
Polymorphic VT/VF	3/12 (25)	1/15 (7)	0/15 (0)

Values are the mean ± SD, median (25th, 75th interquartile range) or n (%).
VT = ventricular tachycardia; VF = ventricular fibrillation; TCL=tachycardia cycle length.

Supplemental Table 3. Characteristic between those with and without recurrence.

	Recurrence (n=15)	No recurrence (n=19)	P value
Age, yrs	65±12	70±10	0.22
Male	15/15	18/19	0.28
Body mass index, kg/m²	27±4	27±3	0.95
NYHA ≥ III	6	4	0.32
Time since MI, months	138 (33-219)	210 (132-282)	0.16
No. of vessels	1.9±0.9	2.3±0.8	0.23
PCI	7	10	0.73
CABG	6	9	0.36
LVEF, %	32±3	37±3	0.14
No. of inducible VT	2 (2-3)	2 (1-4)	0.90
Epicardial ablation	0	2	0.12
RF ablation time, min	42±20	30±16	0.09
Procedure time, min	220±46	198±71	0.35
Induced Nonsustained Monomorphic VT	11 (53)	10 (47)	
No. of beats	7 (6, 11)	7.5 (5.8, 10)	1.00
No. of monomorphic beats	5 (4, 6)	4 (3, 7.5)	0.61
The monomorphic beats			
Similar to pacing QRS morphology	3 (27)	1 (10)	0.30
Similar to area to scar	10 (91)	5 (50)	0.03
Similar to VT induced during procedure	6 (55)	3 (30)	0.25
CI to the last stimulus (ms)	405±71	388±123	0.71
Induced Polymorphic NSVT	4 (31)	9 (69)	
No. of beats	6 (5, 12)	5(5, 7)	0.67
CI to the last stimulus (ms)	366±54	383±77	0.71

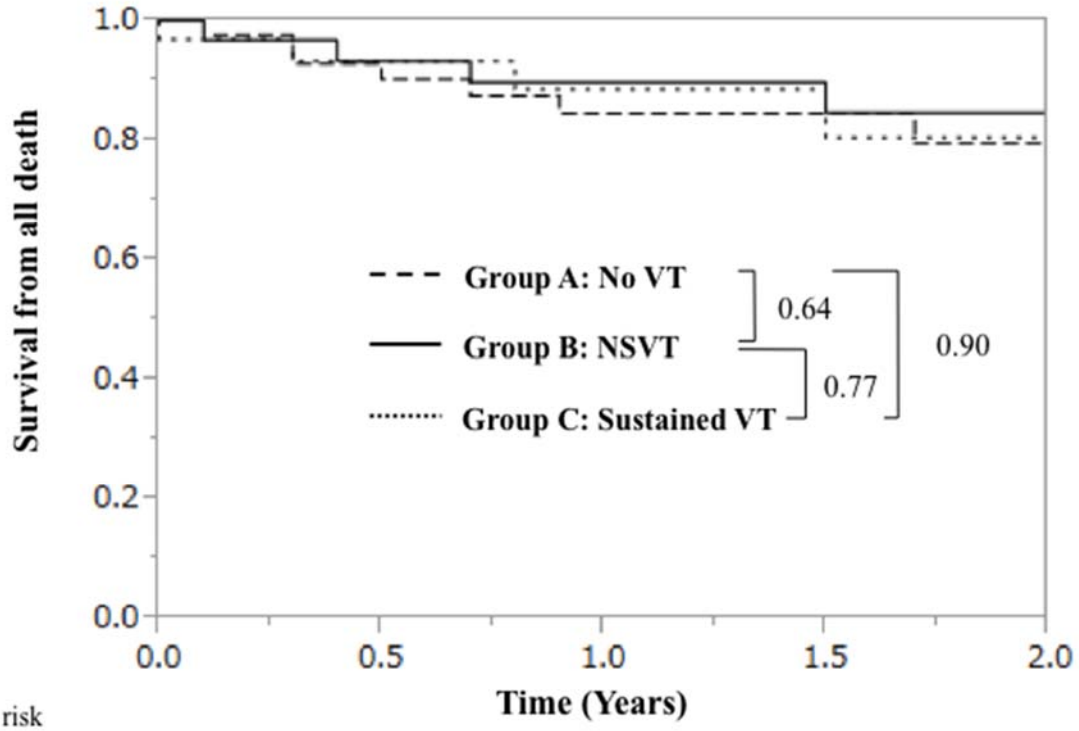
Values are the mean±SD, median (25th, 75th interquartile range) or n (%). RF, radiofrequency; VT, ventricular tachycardia; NSVT, nonsustained VT; CI, coupling interval.

Supplemental Table 4.

Case	Available ECG/EGM	1st beat of NSVT	Findings
1	ECG	different	similar to inducible VT
2	EGM		different TCL of inducible VT
3	NA		
4	EGM		similar to TCL of inducible VT
5	NA		
6	EGM		similar to TCL of inducible VT
7	NA		
8	ECG	different	similar to inducible VT
9	NA		
10	ECG	similar	
11	EGM		similar to TCL of inducible VT
12	NA		
13	EGM		similar to TCL of inducible VT
14	EGM		similar to TCL of inducible VT
15	ECG	different	similar to inducible VT

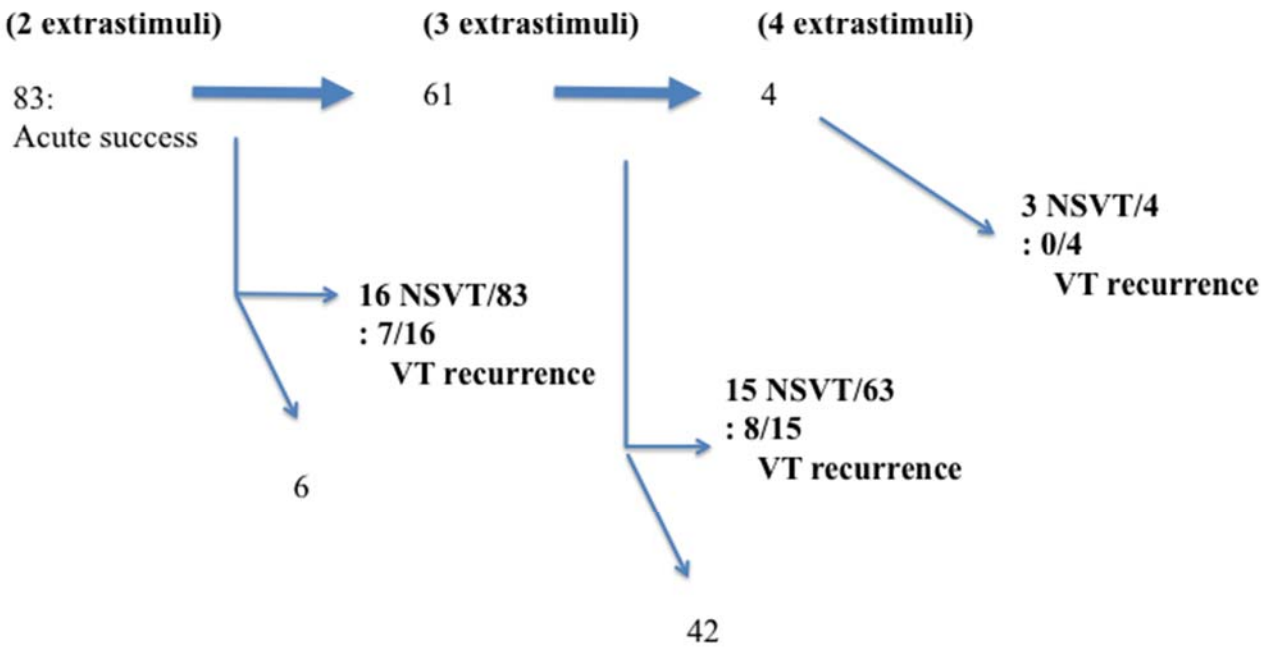
ECG = electrocardiogram; EGM = electrogram of device; NA=not available for review; NSVT = non-sustained ventricular tachycardia; VT = ventricular tachycardia; TCL=tachycardia cycle length.

Supplemental Figure 1: Kaplan-Meier curves showing all cause mortality among the 3 groups by the response to programmed stimulation after ablation.
There was no significant difference among all groups (Group A vs B, P=0.64), (Group A vs C, P=0.90) and (Group B vs C, P=0.77).

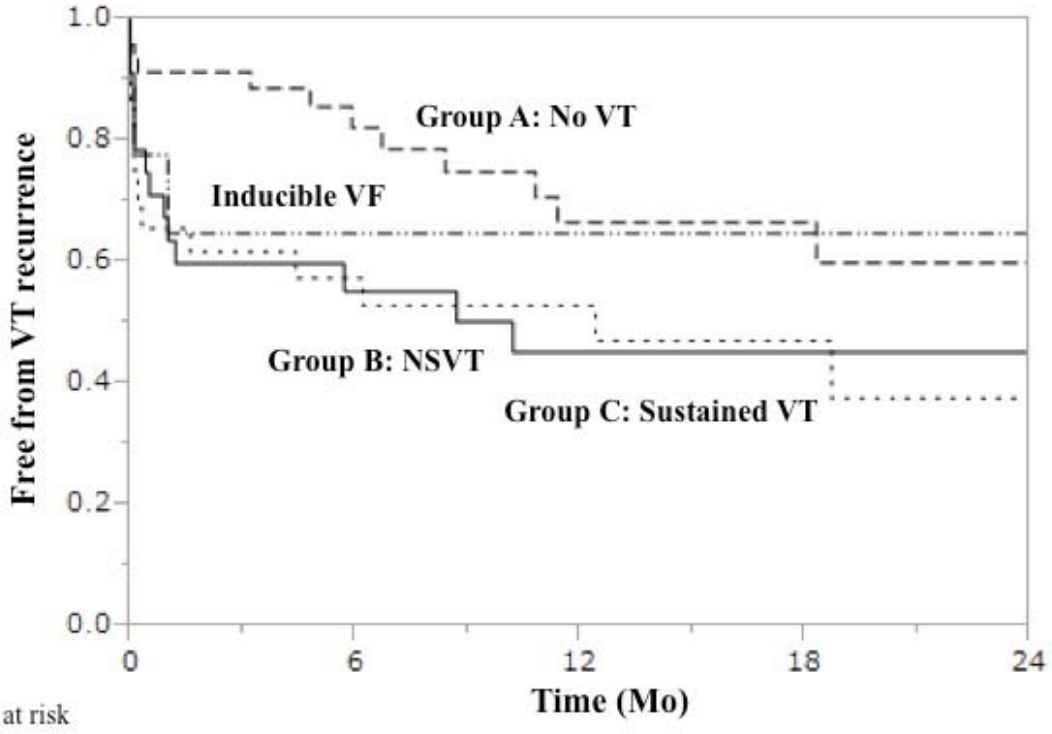


No. at risk					
No VT	49	38	28	20	14
NSVT	34	28	23	18	10
Sustained VT	38	24	17	11	9

Supplemental Figure 2: Flow chart showing the number of ventricular extrastimuli required to initiate the 1st NSVT in patients without inducible SMVT after ablation (acute success) (group A and B). VT, ventricular tachycardia; NSVT, non-sustained VT. In 83 patients with 2 extrastimuli: 16 patients had inducible NSVT, 7 of whom had a recurrence of VT; 6 patients without inducible NSVT did not receive more than 2 extrastimuli. In 61 patients with 3 extrastimuli: 15 had inducible NSVT, 8 of whom had a recurrence of VT. Four extrastimuli were used in only 4 patients, and induced NSVT in 3, none of whom had a recurrence of VT



Supplemental Figure 3: Kaplan-Meier curves showing the recurrence of ventricular tachycardia among the 4 groups including inducible VF by the response to programmed stimulation after ablation.
In Group inducible VF, 44% had VT recurrence, while there was no difference compared to Group A (P=0.08), Groups B (P=0.91) and C (P=0.78).



No. at risk

No VT	49	24	16	10	7
NSVT	34	12	8	7	5
Sustained VT	38	13	9	5	3
VF	9	2	1	1	1