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Recurrent ventricular tachycardia after cardiac sympathetic denervation: prolonged cycle length with improved hemodynamic tolerance and ablation outcomes

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Abstract

Introduction: Cardiac sympathetic denervation (CSD) is utilized for management of ventricular tachycardia (VT) in structural heart disease when refractory to radiofrequency ablation (RFA) or when patient/VT characteristics are not conducive to RFA

Methods: We studied consecutive patients who underwent CSD at our institution from 2009-2018 with VT requiring repeat RFA post-CSD. Patient demographics, VT/procedural characteristics and outcomes were assessed.

Results: Ninety-six patients had CSD, 16 patients underwent RFA for VT post-CSD. There were 15 male and 1 female patients with mean age 54.2±13.2 years. Fourteen patients had nonischemic cardiomyopathy. A mean of 2.0±0.8 RFAs for VT were unsuccessful prior to the patient undergoing CSD. The median time between CSD and RFA was 104 days (IQR=15-241). The clinical VT cycle length was significantly increased after CSD both spontaneously on ECG and/or ICD interrogation (355±73ms pre-CSD versus 422±94ms post-CSD, p=0.001) and intraprocedurally (406±86ms pre-CSD versus 457±88ms post-CSD, p=0.03). Two patients had polymorphic and fourteen had monomorphic VT (MMVT) pre-CSD, and all patients had MMVT post-CSD. The proportion of mappable, hemodynamically stable VTs increased from 35% during pre-CSD RFA to 58% during post-CSD RFA (p=0.038). At median follow-up of 413 days (IQR=43-1840) after RFA, eight patients had no further VT.

Conclusion: RFA for recurrent MMVT post-CSD is a reasonable treatment option with intermediate-term clinical success in 50% of patients. Clinical VT cycle length was significantly increased after CSD with associated improvement in mappable, hemodynamically tolerated VT during RFA.

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Conflict of interest: none

Introduction:

Radiofrequency ablation (RFA) provides significant clinical benefit for patients with structural heart disease and recurrent ventricular tachycardia (VT) when antiarrhythmic drugs (AAD) fail or are not tolerated.[1] However, success rates are modest, and many patients require multiple interventions to control their clinical arrhythmia and avoid recurrent implantable cardioverter-defibrillator (ICD) therapies. Increasing data support the use of cardiac sympathetic denervation (CSD) for the treatment of ventricular arrhythmias.[2, 3] In the published series on CSD, patients predominantly failed RFA or were found to have limited targets (lack of modifiable substrate/scar, pleomorphic/polymorphic VT as primary arrhythmia, etc.)

Nonetheless, recurrence of VT following CSD can occur in over 40% of patients based on the largest published datasets.[3] Clinical characteristics of patients with VT after CSD, and effectiveness of RFA for VT after CSD, have not been studied. We sought to assess whether autonomic modulation, even if not clinically successful in eliminating recurrent VT and ICD therapies, might lead to changes in VT characteristics, and potentially allow for subsequent successful VT RFA.

Methods:

We studied consecutive patients who underwent CSD at our institution from 2009-2018 who had recurrent VT and required initial or repeat RFA after CSD. In these patients, we analyzed demographics, procedural and VT characteristics, and outcomes. Data review was approved by the University of California Los Angeles Institutional Review Board.

Hemodynamic tolerance was defined as ability to perform activation mapping during the clinical VT without the use of mechanical circulatory support devices or additional sustained vasopressor agents during arrhythmia. All antiarrhythmic medications (IV and oral) were held at least 24-48 hours prior to RFA, if clinically appropriate. Clinical VT was determined by 1) assessment of spontaneous VT on 12-lead ECG or 2) ICD interrogation of VT events including cycle length and nearfield/farfield EGM morphologies. These data were then compared to VT (spontaneous or induced) at the time of RFA in order to determine the procedural clinical VT. In the event of multiple different clinical VTs, the average cycle length was obtained. Acute procedural success was defined as non-inducibility of any VT at the end of the procedure.

Partial success was defined as non-inducibility of the clinical VT, but with non-clinical VTs still inducible. A procedure was considered unsuccessful if the clinical VT could still be induced at the end of the procedure.

Cardiac sympathetic denervation

Bilateral or left-sided CSD was performed in all patients under general anesthesia via video-assisted thoracoscopic approach using previously reported methods.[3] Histopathologic analysis was performed to confirm sympathetic ganglia resection. Bilateral sympathectomy

was performed whenever possible, based on prior retrospective data suggesting greater efficacy of this strategy. (Table 1)

Statistical methods:

Continuous variables were expressed as mean ± standard deviation and compared using the student t test. Categorical variables compared with the Fisher exact test. Variables measured before and after CSD procedure were compared using the Wilcoxon signed rank test. Correlation between two continuous variables was determined with the Pearson correlation coefficient. Data were analyzed using SPSS version 25 (IBM, Armonk, NY, USA). A p-value of less than 0.05 was considered statistically significant.

Results:

Baseline characteristics and index RFA

Of 96 patients who had CSD at our institution for VT, 44 had recurrent VT. Of those with recurrent VT, seven received orthotopic heart transplant, ten were managed conservatively, and eleven were deceased. Sixteen patients underwent RFA for VT after CSD. There were 15 male and 1 female patients with mean age 54.2 ± 13.2 years. Average left ventricular ejection fraction was 39 ± 10%. There were 14 patients with non-ischemic and 2 with ischemic cardiomyopathy. Patients had previously undergone a mean of 2.0 ± 0.8 RFA procedures with 15/16 having undergone at least one prior EPS with or without RFA at our institution. (Table 1) On index RFA, only seven (44%) patients had acute success with non-inducibility of VT at procedure end. Three patients had no ablation performed due to PMVT or multiple VT morphologies. Six patients had incomplete ablation performed with VT inducible at procedure end.

CSD and VT recurrence

The median time between index RFA and CSD was 8 days (IQR 5-67). Ten patients underwent CSD within 10 days of RFA either due to unsuccessful procedural result or refractory VT. All patients underwent bilateral CSD except for one patient (patient 11) receiving unilateral (left) CSD due to intraoperative hypoxemia pre-empting bilateral denervation. In the patients who underwent CSD greater than ten days after index RFA, there was no significant difference in spontaneous VTCL comparing index RFA presentation and pre-CSD presentation (370 ± 64ms versus 362 ± 54ms, p = 0.66) (Supplemental table). Median time between index RFA and post-CSD recurrence was 148 days (IQR 32-390). Median time between CSD and post-CSD recurrence was 73 days (IQR 18-218). After CSD, eleven patients presented with VT storm, and five patients had recurrent ICD shocks prompting repeat RFA.

Post-CSD RFA

The median time between CSD and catheter ablation was 104 days (IQR 15-241). Ten patients had epicardial/endocardial RFA and six endocardial-only RFA performed. (Table 2). The intraprocedural clinical VT cycle length increased significantly after CSD compared to prior, excluding those with PMVT (406 ± 86ms pre-CSD versus 457 ± 88ms post-CSD, p = 0.03). The spontaneous VT cycle length by ECG or ICD was significantly increased post-

CSD compared to pre-CSD (355 ± 73ms pre-CSD versus 422 ± 94ms post-CSD, p = 0.001). (Figure 1A and B) There was no significant association between cycle length and time interval between CSD and RFA. Of the three patients who had no ablation performed at index ablation due to either PMVT or pleomorphic VT, all had monomorphic VT post-CSD and all had successful procedural outcome. Spontaneous VT CL in these three patients increased by a mean of 56 ± 20ms.

Regarding procedural VT CL, there were no significant changes in anesthetic plan or intraprocedural vasopressor use between pre-CSD and post-CSD RFA. Additionally, seven patients had no escalation in antiarrhythmic drug regimen (AAD), while five had decrease and four had increase in AAD in the immediate time period preceding RFA. (Table 3)

Two patients had polymorphic and fourteen had monomorphic VT prior to CSD, and all patients had monomorphic VT after CSD. The proportion of mappable, hemodynamically stable VTs increased from 35% during pre-CSD RFA to 58% during post-CSD RFA (p = 0.038). The clinical VT was more likely to be hemodynamically tolerated during RFA, though not statistically significant (50% pre-CSD versus 69% post-CSD, p = 0.28). Of the sixteen patients, seven had no mappable VTs during their pre-CSD RFA versus only four patients with no mappable VTs during post-CSD RFA. There was no significant difference in number of inducible VTs during pre-CSD RFA versus post-CSD RFA (3.5 ± 2.3 versus 3.3 ± 2.4, p = 0.28). Ablation time was less during pre-CSD RFA compared to post-CSD RFA (29 ± 24 minutes versus 49 ± 33 minutes), though this was not statistically significant (p = 0.08).

Outcomes

During post-CSD RFA, twelve out of sixteen (75%) patients had acute success during RFA with non-inducibility of any VT at procedure end. At median follow-up of 413 days (IQR=43-1840), eight (50%) patients were alive with no VT recurrence. There was no significant difference in median time to VT recurrence between patients who underwent endocardial ablation (658 days) versus combined endocardial/epicardial ablation (266 days) (p = 0.12). One patient succumbed to multiorgan failure within a week of RFA, two patients died of heart failure while awaiting transplant, four patients had recurrent VT which resolved with medical management, and one patient had recurrent VT that resolved with repeat RFA at an outside institution. There were no procedure related complications.

Electroanatomic mapping:

Electroanatomic mapping (EAM) data were reviewed when available before and after CSD. Of available cases for review, 22 were performed with NavX (Abbott Medical, Chicago, IL), and 9 were performed with Carto (Biosense Webster, Irvine, CA). Both pre- and post-CSD EAMs were available for 13 of 16 patients with mean area mapped 393.3 ± 241.1cm^2 pre-CSD and 283.7 ± 190.1cm^2 post-CSD (p = NS). Concordant mapping systems on the pre- and post-CSD procedure were used in all patients except for three in which this occurred for logistical reasons (i.e. - NavX on index and Carto on repeat procedure, or vice versa). The number of mapping points was greater on second procedure, though not statistically significant (682 ± 363 versus 1113 ± 1015 points, p = 0.15). Of these, 9 patients had at least

one surface that was mapped both pre- and post-CSD. In comparing same anatomic surfaces both pre- and post-CSD, there was increase in bipolar voltage percent scar (<0.5mV) from 21±25% to 35±21%, but this was not statistically significant (p=0.23). There was no significant difference in percent area border zone (0.5-1.5mV) or in percent area normal (>1.5mV) tissue. (Figure 2) There was no significant correlation between change in clinical VT cycle length and change in scar, border zone, or normal tissue area.

Discussion:

The key findings of this study are the following:

1. Patients with prior CSD who are referred for repeat attempt at VT RFA have success rates similar to patients initially referred for VT ablation
2. Data suggests that post CSD patients have a prolongation of the cycle length of monomorphic VT.
3. There were more hemodynamically tolerated, mappable VTs during RFA after CSD.
4. Changes in VT characteristics were independent of electroanatomic voltage changes.

Treatment options for refractory VT and VT storm are limited. The primary treatment for AAD-refractory VT is RFA. Success rates for VT RFA range from 50-70%[1, 4] but can vary depending on etiology with non-ischemic cardiomyopathy (NICM) patients having a lower overall success rate. CSD, a well described therapy for inherited channelopathies, has been further utilized for patients with cardiomyopathy and recurrent VT when patients are refractory to RFA or are not good candidates for RFA. VT-free survival at 1-year was 58% in a large series, and patients had an overall decrease in VT events by 88%.[3] Given that almost half of patients still have VT episodes after CSD, it is important to understand whether the autonomic modulation, even when unsuccessful in completely controlling VT events, alters the VT substrate/characteristics in such a way as to make them more amenable to future RFA. This is the first experience assessing VT RFA after previous CSD and demonstrates an increase in VT CL and an improved hemodynamic tolerance. Importantly, clinical success was achieved in 50% of cases. This is comparable to overall reported outcomes for catheter ablation;[5] however, the patients in this series had failed multiple prior RFA procedures and CSD, indicative of a more intractable disease process.

Prolongation of VT cycle length can make clinical VT more hemodynamically tolerated and the associated patient stability may increase the success rate of subsequent VT RFA. The rate of VT induced in patients undergoing programmed electrical stimulation has been shown to be associated with patients' ability to remain conscious.[6] However, in another observational study by Landolina and colleagues, baroreflex sensitivity was a more important factor in predicting hemodynamic tolerance of ventricular arrhythmias.[7] This potentially implicates the cardiac neuraxis as an important determinant of whether or not a patient will deteriorate during sustained VT. The tolerability of VT and the presence of a clear ablation target significantly impacts the outcome of RFA.[8] In our series, nine patients

were deemed to be poor candidates for VT RFA and either had no RFA (3 patients) performed or very limited RFA (6 patients) prior to referral for CSD. Of these patients 44% had successful RFA after CSD.

Progression of cardiac disease can lead to increased cardiac scar, which can affect VT cycle length.[9, 10] Ejection fraction and left ventricular size did not differ pre- and post-CSD. Electroanatomic mapping data did not demonstrate significant increase in low voltage regions (Figure 2). While there was numerically increased percent and mapped scar area encountered at post-CSD compared to pre-CSD, this was not statistically significant. It is possible that progression of substrate may account for a portion of the changes seen in our patient series; however, this likely does not account for the entirety of these findings (Figure 3). Additionally, the effect of the initial RFA may partially explain some of the change in cycle length. Importantly, as previously stated, a majority of patients had no or limited ablation performed prior to CSD (due to PMVT, multiple VT morphologies, hemodynamically unstable, etc.). Of the patients who had no ablation performed, all still had increases in the VT CL. Furthermore, no significant change was seen in patients comparing the VTCL pre-CSD RFA and the clinical VT CL prior to CSD, which argues against a significant role of RFA in the change in VTCL seen in these patients. Ablation times were also greater on repeat RFA versus the pre-CSD RFA. This may have been due to a number of factors including greater hemodynamic stability of VTs, more complex substrate, and greater operator impetus given the multiple prior unsuccessful therapies.

Procedural anesthesia level (general vs. moderate sedation and type of anesthetic) and use of vasoactive medications can affect intraprocedural hemodynamic stability, but we also did not see any significant change in anesthesia strategy or vasopressor use. Additionally, anti arrhythmic drugs or changes in regimen could prolong cycle length, and indeed, the majority of these patients had their medication regimens modified or even escalated (Table 3). In order to isolate this effect on VTCL from the effect of the CSD, we analyzed both VTCL during RFA procedure as well as spontaneous VTCL based on ECG or device interrogation. Three of the four patients that had an escalation in AAD had an increase in VT CL during RFA; however, spontaneous VTCL based on 12-lead ECG and ICD intracardiac tracings also increased. These were obtained prior to initiation of any IV antiarrhythmic medications or escalation in drug regimen, as well as prior to anesthetic or intraprocedural influences, suggesting a potential role of CSD in the prolongation of VTCL.

Sympathetic input interacts with tissue substrate causing alterations in activation wavefronts and functional block which can perpetuate VT.[11–14] The effect of global sympathetic modulation on local activation recovery intervals varies depending on scar-border zone characteristics. In general, sympathetic stimulation exerts greater enhancing effect on activation recovery in normal tissue and lesser effect on border zone tissue, which promotes the heterogeneous electrophysiological properties that can sustain arrhythmias.[11] Bilateral cardiac sympathetic denervation has become an established therapy for patients with refractory ventricular arrhythmias; however, many patients still suffer recurrence.[3] In recent data by Yamaguchi and colleagues, a swine model showed increased VT induction threshold after CSD, but with added autonomic modulation by vagus nerve stimulation, the VT induction threshold increased even further, rendering most animals non-inducible.[15]

Therefore, CSD may decrease but not completely eliminate potential VT circuits possibly explaining the change in cycle length seen in our data. The sympathetic nervous system modulates dromotropic properties of normal and infarcted myocardium in complex ways.[14] Changes secondary to sympathectomy (reduced norepinephrine levels in the heart) could play a role in cycle length changes. Whether further sympathetic modulation can provide benefit in these patients is not known. Renal denervation is another promising therapy for autonomic modulation in treatment of ventricular arrhythmias, with a recent case series demonstrating benefit.[16] However, this requires further study before broader application.

Limitations:

This is a single center series given the limited number of centers with such data available. While the increase in VT cycle length and improvement in hemodynamic stability may have been due to autonomic modulation from CSD, alternative causes such as improved response to antiarrhythmic medications, effect of prior RFA, progression of VT undetectable substrate/scar changes (microscopic) cannot be excluded and likely did contribute to some extent. The determination that a patient was not a good candidate for RFA and/or required CSD was at the discretion of the treating physician/team. It cannot be excluded that a patient deemed not to have RFA targets pre-ablation could have been felt to have appropriate targets by another physician. However, at our institution complex VT cases are discussed as a group by several high volume experienced operators to devise a treatment plan. Detailed characteristics of prior VT mapping procedures are always included in the assessment of suitability for catheter ablation.

Conclusion:

Catheter ablation for recurrent monomorphic VT after CSD is a reasonable treatment option with clinical success in 50% of patients at median 14-month follow-up. Clinical VT cycle length was significantly increased at post-CSD RFA with associated improvement in hemodynamically tolerated VT during RFA. These findings are hypothesis generating and potential benefit of CSD allowing previously unmappable VTs to be mapped and successfully ablated should be further investigated.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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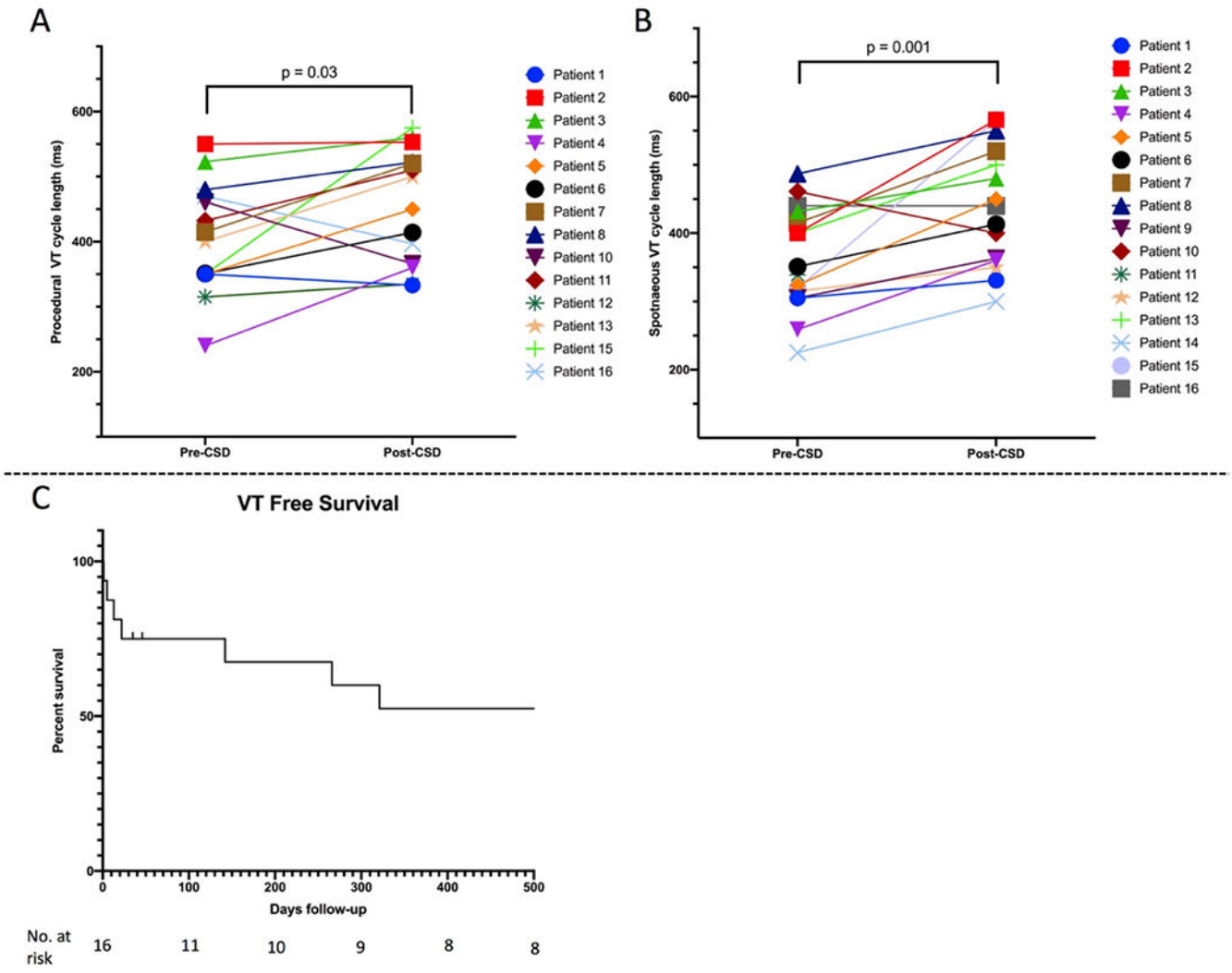

Figure 1:

Change in VT cycle length and VT Free Survival (A) Comparison of clinical VT cycle length during pre-CSD RFA versus during RFA procedure post-CSD. (B) Comparison of spontaneous VT cycle length from 12-lead ECG or ICD interrogation comparing pre-CSD presentation to post-CSD recurrence. (C) Kaplan Meier survival curve for VT free survival in patients undergoing RFA after CSD.

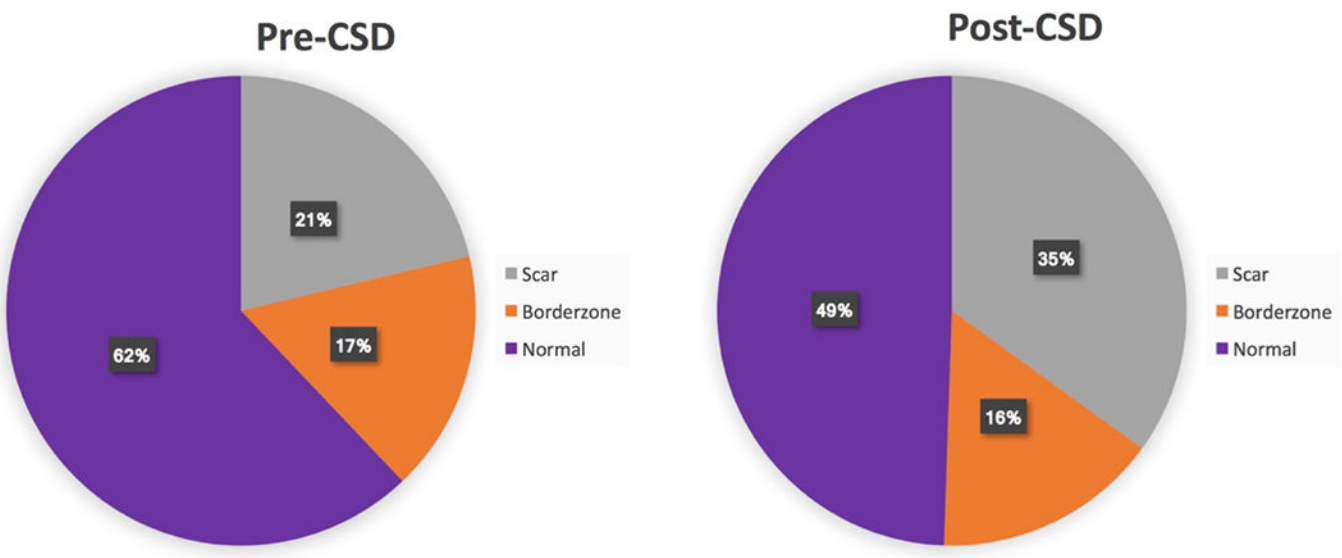

Figure 2:

Pie chart comparison of electroanatomic mapping data for patients who underwent mapping of same surface pre-CSD and post-CSD. Percent scar increased from 21% to 35%, but this was not statistically significant. There was no significant change in percent border zone or normal tissue pre- versus post-CSD. Standard bipolar voltage settings were used for analysis (<0.5mV for scar, 0.5-1.5mV for border zone, and >1.5mV for normal tissue).

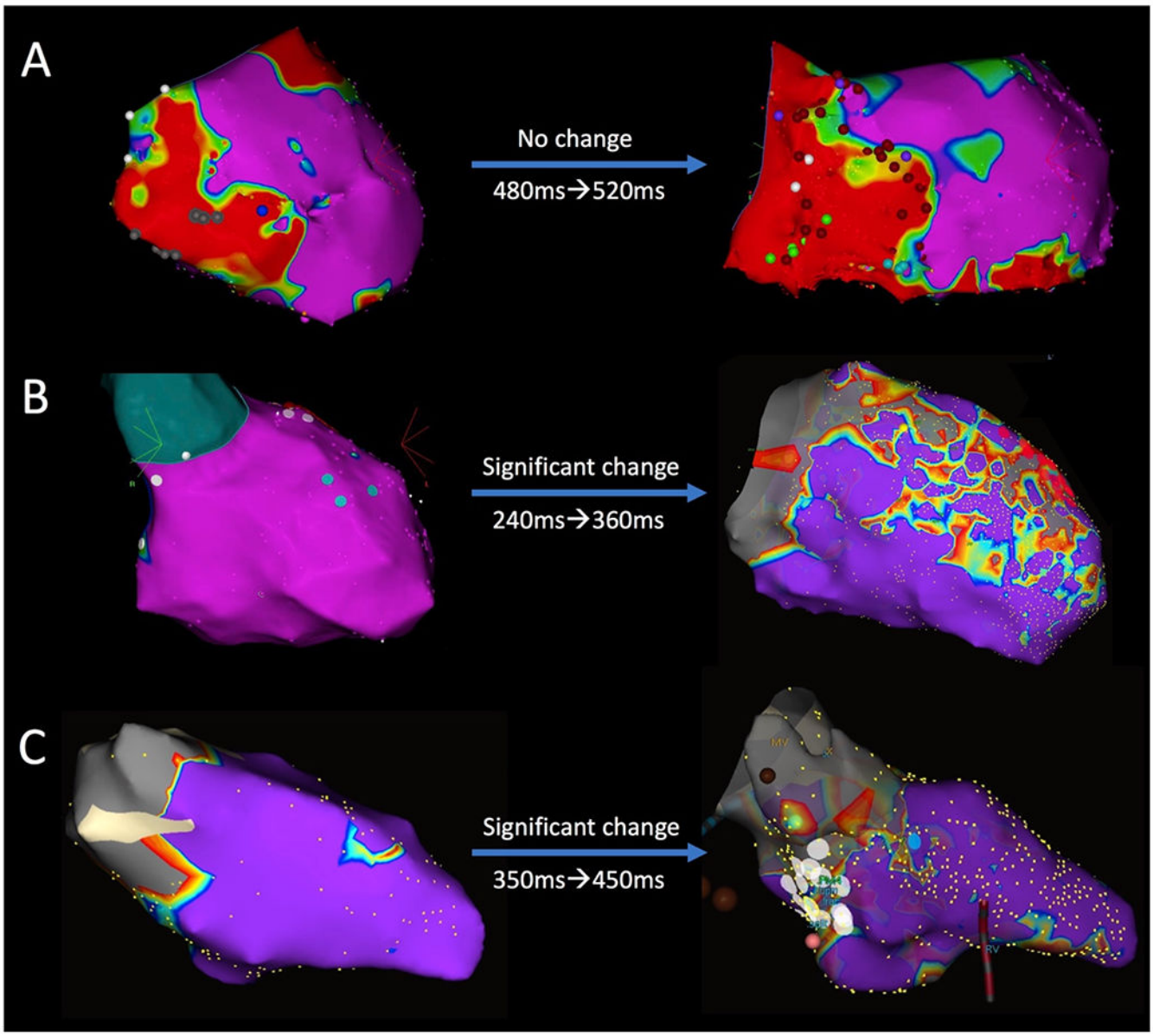

Figure 3: Electroanatomic map and VT cycle length correlation.

Voltage maps shown at standard scar settings at 0.5-1.5mV. (A) A patient with non-ischemic cardiomyopathy in whom VT CL increased but there was no change in EAM scar findings. Pre-CSD and post-CSD maps both made with Carto mapping system (Biosense Webster, Diamond Bar, CA) (B) A patient with non-ischemic cardiomyopathy in whom VT CL significantly increased and there was corresponding change in voltage mapping data with significantly more scar encountered during post-CSD mapping. Pre-CSD map made with Carto mapping system (Biosense Webster, Diamond Bar, CA). Post-CSD map made with NavX (Abbott Medical, Chicago, IL). (C) A patient with non-ischemic cardiomyopathy in whom VT CL significantly increased but there was no significant change in voltage mapping

data, with similar area of scar and border zone tissue. Pre- and post-CSD maps both made with NavX (Abbott Medical, Chicago, IL).

Table 1:

Baseline Patient Characteristics

Patient	Age	Gender	Cardiomyopathy etiology	Ejection fraction	NYHA Class Pre-CSD	Number of prior ablations	Access at prior ablation(s)	Prior RFA challenges	CSD
1	54	M	Ischemic	30-35%	3	1	Endo	Outside ablation	Bilateral
2	36	M	Non-ischemic	40-45%	2	2	Endo Endo	N/A	Bilateral
3	52	M	Non-ischemic	35-40%	2	4	Endo Endo Endo Endo/epi	Multiple VT morphologies	Bilateral
4	26	M	Non-ischemic	25-30%	2	2	Endo Endo	N/A	Bilateral
5	65	M	Non-ischemic	40-45%	2	1	Endo	Intraseptal substrate	Bilateral
6	45	M	Non-ischemic	40-45%	2	2	Endo Endo/epi	Intramyocardial substrate; phrenic nerve proximity	Bilateral
7	66	M	Non-ischemic	<20%	3	2	Endo/epi	Multiple VT morphologies	Bilateral
8	37	M	Non-ischemic	25-30%	3	2	Endo/epi Endo/epi	Multiple VT morphologies	Bilateral
9	66	M	Non-ischemic	30%	2	2	Endo Endo	Minimal scar	Bilateral
10	65	M	Non-ischemic	20-25%	2	3	Endo Endo/epi Endo	N/A	Bilateral
11	50	M	Ischemic	35%	2	2	Endo Endo/epi	N/A	Left
12	64	M	Non-ischemic	45-50%	1	1	Endo	Multiple VT morphologies	Bilateral
13	50	F	Non-ischemic	45%	2	2	Endo Endo/epi	N/A	Bilateral
14	60	M	Non-ischemic	55%	2	2	Endo Endo	Multiple VT morphologies	Bilateral
15	56	M	Non-ischemic	45-50%	2	1	Endo	Intraseptal substrate	Bilateral
16	75	M	Non-ischemic	25-30%	2	3	Endo Endo Endo	Intraseptal substrate	Bilateral

Abbreviations: CSD = cardiac sympathetic denervation, RFA = radiofrequency ablation

Table 2:

Pre- and Post-CSD VT Procedural Characteristics

			Pre-CSD RFA				Post-CSD RFA				
Patient	**Recurrence presentation**	**RFA Access**	**Morphology**	**HDS VT/ Tota VTs induced**	**Ablation strategy**	**Acute ablation outcome**	**Morphology**	**HDS VT/ Total VTs induced**	**Ablation strategy**	**Acute ablation outcome**	**Clinical Outcome**
1	VT storm	Endo	MMVT	0/1	No ablation	Unsuccessful	MMVT	0/2	Substrate	Success	No recurrence
2	VT storm	Endo	MMVT	1/2	Substrate + VT mapping	Success	MMVT	3/4	Substrate + VT mapping	Success	No recurrence
3	Recurrent VT	Endo/ epi	MMVT	0/3	Substrate	Success	MMVT	3/4	Substrate + VT mapping	Success	No recurrence
4	Recurrent VT	Endo/ epi	MMVT	0/1	Substrate	Partial	MMVT	0/1	Substrate + VT mapping	Success	No recurrence
5	Recurrent VT	Endo/ epi	MMVT	0/3	Substrate	Partial	MMVT	0/4	Substrate	Success	VT recurrence repeat RFA
6	VT storm	Endo/ epi	MMVT	1/4	Substrate + VT mapping	Success	MMVT	2/5	Substrate + VT mapping	Partial	No recurrence
7	VT storm	Endo/ epi	MMVT	3/9	Substrate + VT mapping	Unsuccessful	MMVT	4/6	Substrate + VT mapping	Success	Died of pump failure
8	VT storm	Endo/ epi	MMVT	5/5	Substrate + VT mapping	Success	MMVT	5/5	Substrate + VT mapping	Success	VT recurrence medically managed
9	VT storm	Endo/ epi	PMVT	1/5	Substrate + VT mapping	Unsuccessful	MMVT	1/2	Substrate	Success	VT recurrence medically managed
10	VT storm	Endo	MMVT	1/3	Substrate + VT mapping	Success	MMVT	2/2	Substrate + VT mapping	Success	No recurrence
11	VT storm	Endo/ epi	MMVT	2/2	Substrate + VT mapping	Success	MMVT	3/3	Substrate + VT mapping	Unsuccessful	Died of multiorgan failure
12	Recurrent VT	Endo	MMVT	0/7	No ablation	Unsuccessful	MMVT	1/1	Substrate + VT mapping	Success	No recurrence
13	VT storm	Endo	MMVT	1/1	Substrate + VT mapping	Success	MMVT	1/1	Substrate + VT mapping	Success	No recurrence
14	Recurrent VT	Endo/ epi	PMVT	0/1	No ablation	Unsuccessful	MMVT	2/3	Substrate + VT mapping	Success	VT recurrence medically managed
15	VT storm	Endo/ epi	MMVT	0/3	Substrate	Unsuccessful	MMVT	0/10	Substrate + VT mapping	Unsuccessful	Died of pump failure

			Pre-CSD RFA				Post-CSD RFA				
Patient	**Recurrence presentation**	**RFA Access**	**Morphology**	**HDS VT/ Tota VTs induced**	**Ablation strategy**	**Acute ablation outcome**	**Morphology**	**HDS VT/ Total VTs induced**	**Ablation strategy**	**Acute ablation outcome**	**Clinical Outcome**
16	VT storm	Endo	MMVT	1/1	Substrate + VT mapping	Unsuccessful	MMVT	1/2	Substrate + VT mapping	Unsuccessful	VT recurrence medically managed

Abbreviations: CSD = cardiac sympathetic denervation, HDS = hemodynamically stable, MMVT = monomorphic ventricular tachycardia, PMVT = polymorphic ventricular tachycardia, RFA = radiofrequency ablation, VT = ventricular tachycardia

Table 3.

Antiarrhythmic and anesthesia characteristics

	Pre-CSD RFA			Post-CSD RFA				
Patient	**Antiarrhythmic regimen at at pre-CSD RFA**	**Anesthesia**	**Vasopressors**	**Antiarrhythmics at post-CSD RFA**	**Anesthesia**	**Vasopressors**	**Antiarrhythmic Pre- v. Post-CSD comparison**	**Discharge med regimen**
1	Lidocaine IV Amiodarone 200 bid Mexiletene 400 tid	General	Not available	Amiodarone 200 daily Mexiletine 200 bid Esmolol IV	General	Phenylephrine	No increase	Amiodarone 200 daily Metoprolol 25 daily
2	Amiodarone 200 bid Mexiletine 150 tid Metoprolol 50 bid	General	Phenylephrine	Amiodarone IV	General	None	No increase	Amiodarone 200 daily Metoprolol 25 bid
3	Sotalol 160 bid Fleciainide 100 bid Carvedilol 12.5 bid	MAC	Dopamine Epinephrine	Amiodarone 200 bid Ranolazine 500 bid	General	Dopamine	No increase	Metoprolol 25 bid Ranolazine 500 bid
4	Amiodarone 400 daily Carvedilol 6.25 bid	MAC	None	Carvedilol 25 bid	General	None	Decrease	Carvedilol 25 bid
5	Mexiletine 200 tid Carvedilol 25 bid	General	None	Lidocaine IV Amiodarone 200 daily Mexiletine 200 tid Carvedilol 12.5 bid	General	None	Increase	Amiodarone 400 bid Mexiletine 400 tid Metoprolol 25 bid
6	Mexiletine 150 tid Metoprolol 50 bid	General	Dopamine	Mexiletine 150 tid Metoprolol 50 bid	MAC	Dopamine	No increase	Metoprolol 50 bid
7	Amiodarone IV Lidocaine IV Mexiletine 200 tid Metoprolol 25 bid	General	Epinephrine	Amiodarone IV Lidocaine IV Metoprolol 50 bid	General	Vasopressin	Decrease	Amiodarone 400 bid Mexiletine 150 tid Metoprolol 25 bid
8	Lidocaine IV Carvedilol 50 bid	MAC	Dopamine	Carvedilol 25 bid	MAC	Phenylephrine	Decrease	Carvedilol 25 bid
9	Amiodarone 200 daily Mexiletine 150 tid Carvedilol 3.125 bid	General	Phenylephrine	Amiodarone 200 daily Mexiletine 200 bid Carvedilol 3.125 bid	General	Phenylephrine	No increase	Amiodarone 200 daily Mexiletine 200 bid Carvedilol 3.125 bid
10	Sotalol 80 bid Carvedilol 50 bid	MAC	Phenylephrine Dopamine	Lidocaine IV Sotalol 160 bid Carvedilol 50 bid	MAC	Epinephrine	Increase	Mexiletine 150 tid Carvedilol 50 bid
11	Procainamide IV Metoprolol 25 bid	General	Phenylephrine	Procainamide IV Metoprolol 25 bid	General	Phenylephrine	No increase	N/A

	Pre-CSD RFA			Post-CSD RFA				
Patient	**Antiarrhythmic regimen at at pre-CSD RFA**	**Anesthesia**	**Vasopressors**	**Antiarrhythmics at post-CSD RFA**	**Anesthesia**	**Vasopressors**	**Antiarrhythmic Pre- v. Post-CSD comparison**	**Discharge med regimen**
12	Amiodarone 400 daily Mexiletine 150 bid Metoprolol 150 bid	MAC	Phenylephrine	Verapamil 100 daily Metoprolol 50 bid	MAC	None	Decrease	Verapamil 100 daily Metoprolol 50 bid
13	Procainamide IV Lidocaine IV Esmolol IV Mexiletine 150 tid	General	Phenylephrine	Amiodarone IV Esmolol IV Mexiletine 150 tid	MAC	None	No increase	Amiodarone 200 bid Metoprolol 200 bid
14	Dofetilide 500 bid Metoprolol 50 bid	General	None	Dofetilide 500mcg bid Mexiletine 150 bid Metoprolol 50 daily	General	Phenylephrine	Increase	Dofetilide 500mcg bid Mexiletine 150 bid Metoprolol 50 daily
15	Amiodarone 200 daily Metoprolol 25 bid	MAC	Epinephrine	Amiodarone IV Procainamide IV Esmolol IV	General	Vasopressin	Increase	N/A
16	Amiodarone IV Sotalol 80 bid Mexiletine 200 bid	MAC	Epinephrine	Sotalol 80 tid Mexiletine 300 tid	MAC	Phenylephrine	Decrease	Amiodarone 200 tid Mexlietine 200 tid Ranolazine 500 bid Metoprolol 50 bid

Abbreviations: CSD = cardiac sympathetic denervation, MAC = monitored anesthesia care, RFA = radiofrequency ablation