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BILATERAL CARDIAC SYMPATHETIC DENERVATION FOR THE MANAGEMENT OF ELECTRICAL STORM

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To the Editor: The sympathetic nervous system plays an important role in ventricular arrhythmogenesis. Left cardiac sympathetic denervation (LCSD) decreases the incidence of ventricular arrhythmias (VAs), and sudden cardiac death in patients with severe VAs [1,2] However, when LCSD is ineffective in suppressing VAs, adjunctive right cardiac sympathetic denervation (RCSD) may be an option. In humans, the safety and feasibility of BCSD in the management of VAs remains unclear. The present study was undertaken to assess the benefit of BCSD for the acute management of persistent VAs.

We reviewed the records of patients who underwent BCSD (or RCSD after prior LCSD failed to control arrhythmias). Review of patient data was in accordance with the guidelines of the institutional review board. These patients presented with electrical storm characterized by incessant ventricular tachycardia (VT), or repeated episodes of ventricular fibrillation (VF).

Five males and 1 female were included in the study. Mean age 60.1 years (47 to 75 years), mean LV EF 25.8% (15–40%) (Table). Five patients presented with monomorphic VT (MMVT), and 1 patient had polymorphic VT (PMVT). Of patients with MMVT, four had undergone previous endocardial VT ablation and one had undergone an epicardial VT ablation. The arrhythmia burden and number of therapies (automated or external defibrillator shocks, and anti-tachycardia pacing episodes) suffered by each patient is shown in the table.

After presentation, VAs persisted despite intensive investigation and correction of all reversible causes. All patients received maximal tolerated β-blockade (metoprolol 50%, carvedilol 50%) and amiodarone. Lidocaine and/or mexiletine was utilized in 50% of the patients. Other antiarrhythmics were contraindicated or had failed previously. Of the five patients with MMVT, we performed catheter ablation in three, including one combined endocardial and epicardial approach. One patient underwent three endocardial ablations. In summary, all patients with MMVT underwent catheter ablation (mean 2.2±0.5 ablations/

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patient) either prior to or during the hospitalization where BCSD was performed. TEA was utilized in two patients, with little to no response noted, despite repositioning of the epidural catheter. The patients were all deemed poor transplant candidates. Only after these measures failed were patients considered for BCSD (or RCSD as an adjunct to prior LCSD).

After BCSD, a complete response was observed in 66.7% of patients (4/6), while a partial response was seen in 16.7% of patients (1/6) and no response in 16.7% (1/6). ICD shocks and ATPs decreased to 0 shocks or ATPs in 3 patients, and decreased by greater than 50% in one patient (Figure, Table). External shocks decreased from 11 to 0 in another patient. Frequency of therapies before and after BCSD are shown in the Figure. Only one patient showed no response to BCSD. All five patients who showed a reduction in VAs to BCSD survived to discharge (Table), while the only non-responder expired after withdrawal of care, at the family's request. After discharge, two deaths were noted (Table), neither of which were related to arrhythmias. Patient 1 continued to have heart failure exacerbations and elected to go on hospice care. Patient 6 expired at home for unknown reasons. Interrogation of his ICD showed no atrial or ventricular arrhythmias before or at the time of death.

No significant electrocardiographic changes or events consistent with adrenergic insufficiency were documented in patients subsequent to bilateral denervation. Operative complications occurred in two patients (post-operative heart failure and poor tolerance of single lung ventilation during VATS).

To our knowledge, this is the largest cohort of BCSD reported to date. Limitations to this study include its small size, which precludes broad conclusions regarding the applicability of these results. Further, due to the lack of randomization and retrospective approach, biases which may have been involved in the decision making process cannot be excluded.

Mechanisms underlying the benefit of BCSD may include the interruption of adverse stellate ganglion remodeling, or mitigation of pro-arrhythmic neural signaling within the myocardium or stellate ganglia. Multiple lines of evidence suggest a potent anti-arrhythmic effect of BCSD on ventricular myocardium. In canine studies comparing left, right, or bilateral sympathectomy, the most profound anti-arrhythmic effects were seen with bilateral sympathectomy.[3,4] Studies on spinal cord stimulation or thoracic epidural anesthesia[2,5,6] all of which decrease global cardiac sympathetic activity have shown a profound protective effect. Compared to asystole and pulseless electrical activity, VT and VF are less frequent modes of SCD in patients after cardiac transplantation[7], as these hearts are completely denervated,

Our study suggests that patients with incessant ventricular arrhythmias for whom no other therapeutic options exist, bilateral cardiac sympathetic denervation may be beneficial. This procedure does not appear to result in adverse outcomes. Further studies examining the role of BCSD in suppressing human arrhythmias are warranted.

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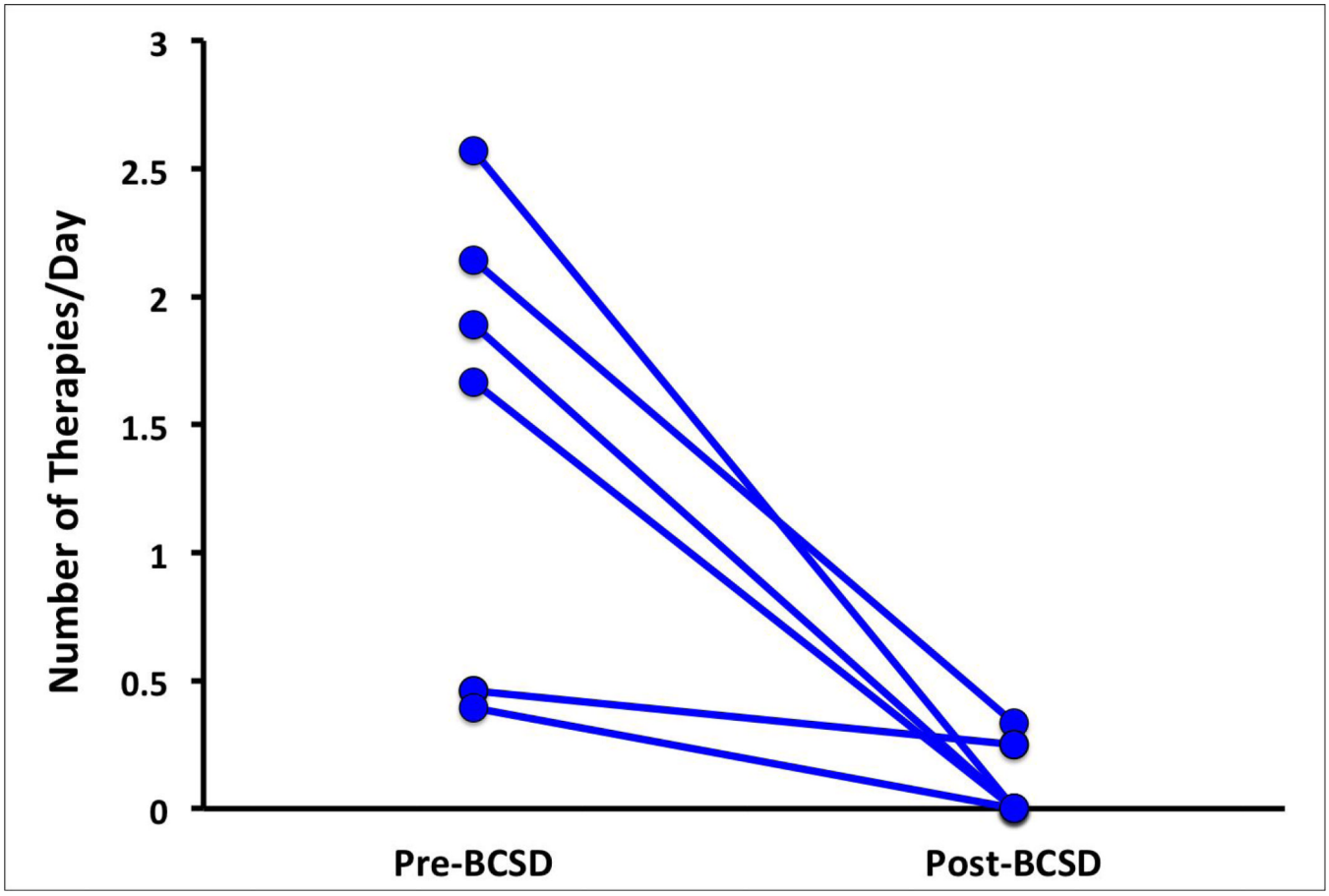

Figure 1.
The acute improvement in therapies per day (number of therapies/number of days) before and after bilateral cardiac sympathetic denervation are shown. Therapies included antitachycardia pacing episodes and automated or external defibrillation.

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Table

Baseline characteristics, arrhythmia burden, clinical management, acute and intermediate outcomes are shown for patients in the study.

Patient	Age/Gender	Substrate/ LV EF	VT Type	Anti-arrhythmics	GA /TEA	VT RFA	Arrhythmia Episodes / Arrhythmia Therapies* Pre-BCSD	Arrhythmia Episodes / Arrhythmia Therapies* Post BCSD	Response to BCSD	Survival to Discharge	intermediate Follow up (Days post-Discharge)
1	69 / M	Sarcoid / 20%	MMVT	Carvedilol Amiodarone	No / No	2 Endo	45 / 30 (14 Days)	3 / 2 (6 Days)	Partial	Yes	Death CHF (89 Days)
2	66 / F	NICM / 20%	PMVT	Amiodarone Lidocaine Metoprolol	Yes / Yes	n/a	51 / 6 (13 Days)	27 / 2 (8 Days)	Poor	No	n/a
3	55 / M	NICM / 20%	MMVT	Amiodarone Carvedillol Lidocaine, Mexiletine	No / No	2 Endo 1 Epi	39 / 17 (9 Days)	0 / 0 (3 Days)	Complete	Yes	Alive (207 Days)
4	47 / M	NICM / 15%	MMVT	Amiodarone Esmolol Diltiazem Carvedilol Verapamil Lidocaine Procainamide	Yes / Yes	3 Endo	87 / 11 (28 Days)	0 / 0 (7 Days)	Complete	Yes	Alive (153 Days)
5	49 / M	NICM / 40%	MMVT	Amiodarone metoprolol	No / No	2 Endo	43 / 25 (15 Days)	0 / 0 (14 Days)	Complete	Yes	Alive (129 Days)
6	75 / M	ARVC / 40%	MMVT	Amiodarone Metoprolol	No / No	2 Endo 1 Epi	36 / 36 (14 Days)	0 / 0 (14 Days)	Complete	Yes	Death Unknown Cause (21 Days)

*Therapies: Automated or Manual Defibrillation, and Anti-Tachycardia pacing.

ARVC – Arrhythmogenic Right Ventricular Cardiomyopathy, BCSD – Bilateral Cardiac Sympathetic Denervation, CHF – Congestive Heart Failure, Endo – Endocardial, Epi –Epicardial, GA – General Anesthesia, Ventricular Ejection Fraction, VT – Ventricular Tachycardia, NICM – Non-Ischemic Cardiomyopathy, TEA – Thoracic Epidural Anesthesia, MMVT – Monomorphic Ventricular tachycardia, PMVT –Polymorphic Therapies: Automated or Manual Defibrillation, and Anti-Tachycardia pacing. Anesthesia, M – Male, F – Female, LV EF – Left tachycardia, PMVT – Polymorphic Ventricular Tachycardia