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 may be an option. In humans, the safety and feasibility of bilateral cardiac sympathetic denervation (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 right cardiac sympathetic denervation 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.

Five men and 1 woman were included in the study. The mean age was 60.1 years (range: 47 to 75 years), and the mean left ventricular ejection fraction was 25.8% (range: 15% to 40%) (Table 1). Five patients presented with monomorphic VT (MMVT), and 1 patient had polymorphic VT. Of patients with MMVT, 4 had undergone previous endocardial VT ablation, and 2 had undergone epicardial VT ablation. The arrhythmia burden and number of therapies (automated or external defibrillator shocks and antitachycardia pacing episodes) experienced by each patient are shown in Table 1.

After presentation, VAs persisted despite intensive investigation and correction of all reversible causes. All patients received maximal tolerated beta-blockade (metoprolol 50%, carvedilol 50%) and amiodarone. Lidocaine and/or mexiletine was used in 50% of patients. Other antiarrhythmic agents were contraindicated or had failed previously. Among the 5 patients with MMVT, we performed catheter ablation in 3, including 1 combined endocardial and epicardial approach. One patient underwent 3 endocardial ablations. In summary, all patients with MMVT underwent catheter ablation (mean 2.2 ± 0.5 ablations/patient) either before or during the hospitalization that included BCSD. Thoracic epidural anesthesia was used in 2 patients, with partial to no response noted, despite repositioning of the epidural catheter. The patients were all deemed poor transplantation candidates. Only after these measures failed were patients considered for BCSD (or right cardiac sympathetic denervation as an adjunct to prior LCSD).

After BCSD, complete response was observed in 66.7% of patients (4 of 6), while partial response was seen in 16.7% of patients (1 of 6) and no response in 16.7% (1 of 6). Implantable cardioverter-defibrillator shocks and antitachycardia pacing decreased to no shocks or episodes in 3 patients and decreased by >50% in 1 patient (Fig. 1, Table 1). External shocks decreased from 11 to none in another patient. Frequencies of therapies before and after BCSD are shown in Figure 1. Only 1 patient showed no response to BCSD. All 5 patients who showed reductions in VAs with BCSD survived to discharge (Table 1), while the only nonresponder expired after withdrawal of care, at the family’s request. After discharge, 2 deaths were noted (Table 1), neither of which was related to arrhythmias. Patient 1 continued to have heart failure exacerbations and elected to receive hospice care. Patient #6 expired at home for unknown reasons. Interrogation of his implantable cardioverter-defibrillator showed no atrial or VAs 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 2 patients (post-operative heart failure and poor tolerance of single-lung ventilation during video-assisted thoracoscopic surgery).

To our knowledge, this is the largest cohort of patients undergoing BCSD reported to date. Limitations of this study include its small size, which precludes broad conclusions regarding the applicability of these results. Furthermore, because of the lack of randomization and the retrospective approach, biases that 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 the mitigation of proarrhythmic neural signaling within the myocardium or stellate ganglia. Multiple lines of evidence suggest a potent antiarrhythmic effect of BCSD on ventricular myocardium. In canine studies comparing left, right, and bilateral sympathectomy, the most profound antiarrhythmic effects were seen with bilateral sympathectomy (3,4). Studies on spinal cord stimulation and thoracic epidural anesthesia (2,5,6), both of which decrease global cardiac sympathetic activity, have shown a profound protective effect. Compared with asystole and pulseless electrical activity, VT and ventricular fibrillation are less frequent modes of sudden cardiac death in patients after cardiac transplantation (7), because these hearts are completely denervated. Our study suggests that for patients with incessant VAs for whom no other therapeutic options exist, BCSD 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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