EDITORIAL COMMENTARY

Managing ventricular arrhythmias after failed catheter ablation: Interrupting the reentrant loop of repeat ablation

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The autonomic nervous system exerts profound control on cardiac function. Perturbations in cardiac function are sensed and relayed to neural control centers via afferent nerves, while efferent nerves relay reflex output to the heart. Alterations in autonomic nervous system function, as seen in disease states such as myocardial infarction and heart failure, result in dysfunctional neural processing, inducing excessive sympathetic tone and withdrawal of parasympathetic drive. These consequences have been directly related to pump dysfunction and arrhythmogenesis and form the basis for the use of β-adrenergic receptor blockers.

Cardiac sympathetic denervation (CSD), traditionally used in the treatment of ventricular arrhythmias (VAs) in long QT syndrome, is increasingly used in managing catecholaminergic polymorphic ventricular tachycardia (VT) and VAs related to structural heart disease. CSD reduces repolarization heterogeneity and shortens the QT interval in patients with long QT syndrome type 1. In patients with catecholaminergic polymorphic VT, CSD mitigates the adrenergically driven initiation and maintenance of recurrent arrhythmias that characterize this condition, especially during implantable cardioverter-defibrillator shocks. In structural heart disease, CSD imparts several benefits, including interrupting a significant source of catecholamines exposed to myocytes, eliminating adverse neuronal remodeling within stellate ganglia, and interrupting cardiac afferent neurotransmission, a major driver of cardiac adrenergic neuroremodeling and adverse cardiac remodeling.

In this issue of HeartRhythm, Richardson et al expand the potential uses of CSD for VAs not previously reported. They presented a series including 7 patients with a variety of VAs in structurally normal or abnormal hearts for which catheter ablation failed or was limited by anatomical factors. In these patients arrhythmia control was achieved with CSD, although 1 patient underwent orthotopic heart transplantation shortly after CSD (the patient was listed for orthotopic heart transplantation before CSD). In the study, no complications occurred, and in the follow-up period, changes in the sweating pattern were reported as a side effect in 1 patient.

This small series raises a few important points for consideration regarding CSD for VAs. It highlights some of the limitations of catheter ablation. Although catheter ablation has profoundly affected the management of VAs positively, there are clinical scenarios where ablation fails. These may be due to anatomical factors that limit access, catheter stability, or energy delivery to the arrhythmia focus or a critical isthmus. Strategies such as alcohol injection or coil embolism of arterial branches supplying the critical site for the VT may be limited by inadequate identification of such a branch or by collateral damage to uninvolved myocardium. In these cases, CSD offers an alternative therapeutic approach.

Another notable point is the variety of VAs for which CSD had a beneficial effect. This included not only monomorphic and polymorphic VTs in structural heart diseases such as arrhythmogenic right ventricular cardiomyopathy and ischemic cardiomyopathy but also fascicular premature ventricular contraction-triggered ventricular fibrillation in hypertrophic cardiomyopathy and exertion-mediated VT in the structurally normal heart. These VAs spanned automaticity, or energy delivery to the arrhythmia focus or a critical isthmus. Strategies such as alcohol injection or coil embolism of arterial branches supplying the critical site for the VT may be limited by inadequate identification of such a branch or by collateral damage to uninvolved myocardium. In these cases, CSD offers an alternative therapeutic approach.

Perhaps the most consequential lesson from the present study is the need to consider other options after an adequate and thorough attempt at catheter ablation fails. Certainly, repeat ablation may be the answer if, for example, only endocardial mapping and ablation was performed initially. Epicardial mapping and ablation may yield additional target sites for ablation. As illustrated by the authors, there are cases where repeat catheter ablation is unlikely to yield significantly different results, and many
of these cases can be identified a priori. CSD offers an important therapeutic alternative in such cases.

A notable consideration to make regarding the study is the somewhat low burden of VAs in the population, with a median of 1 VA episode per month in the population. Other CSD reports that predominantly focused on patients with electrical storm or end-stage VA included patients with higher VA burden. Although this is a small study, it suggests that CSD may not need to be reserved for electrical storm or after extensive attempts at arrhythmia control. While little can be drawn from the small sample size, the low recurrence rates of sustained arrhythmias or implantable cardioverter-defibrillator therapies compared to studies of CSD for electrical storm with recurrence rates of up to 30% raises the possibility that “early” utilization of CSD may be more beneficial.

In summary, this data set adds to a growing body of literature on the use of CSD in managing VAs when catheter ablation fails or is not achievable and is unique in the breadth of arrhythmias and arrhythmic mechanisms for which CSD is applied. Neuromodulation, whether achievable by CSD or other means, holds great promise for managing arrhythmias. Large randomized trials are needed to assess these strategies for arrhythmia control compared to current best practices.

References