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EDITORIAL COMMENT

Open Chest Epicardial VT Ablation After LVAD

Ounce of Prevention or Pound of Cure?*

Joshua D. Moss, MD, Ayhan Yoruk, MD

Drug-refractory ventricular arrhythmias (VAs) are common in patients with a continuous-flow left ventricular assist device (LVAD). The acute and subacute hemodynamic effects of VA after LVAD implantation can be highly variable, introducing additional management uncertainties and challenges. The safety and efficacy of endocardial catheter ablation have been demonstrated in patients with a variety of continuous-flow LVAD models, although none previously with the Jarvik 2000 device (Jarvik Heart, Inc., New York, New York) (1).

In this issue of *JACC: Case Reports*, Corona et al. (2) describe successful epicardial ablation through a left anterior thoracotomy for refractory VA in a patient who had undergone LVAD implantation as destination therapy 3 years earlier. After lysis of adhesions and exposure of the left ventricular (LV) surface, substrate and activation mapping was achieved with a decapolar catheter and the CARTO 3 mapping system (Biosense Webster, Irvine, California). One episode of ventricular tachycardia (VT) that was induced by right ventricular (RV) stimulation terminated during ablation, and another was targeted during sinus rhythm by scar homogenization. Radiofrequency lesions were delivered at 30 W and using a 3.5-mm-tip irrigated ablation catheter. The surgical procedure was well tolerated, with no recurrent VT through 9 months of follow-up while on beta-blocker monotherapy. Corona et al. (2) should be commended for coordinating a technically challenging hybrid procedure with an excellent outcome. The patient's presentation and the ablation strategy chosen highlight a variety of factors to consider in the management of VA in this patient population: the clinical significance of such arrhythmias, potential obstacles to interventional therapy, and preventative techniques that can be used before or during LVAD implantation.

Some patients with an LVAD have no immediate symptoms or hemodynamic instability, even with rapid and sustained VAs. Retrospective analyses have failed to demonstrate the mortality benefit of an implantable cardioverter-defibrillator (ICD) in these patients, thus raising questions about the true impact of sustained VAs (3,4). The ASSIST-ICD (Determination of Risk Factors of Ventricular Arrhythmias After Implantation of Continuous Flow Left Ventricular Assist Device) investigators showed no difference in transplant-free survival between patients with and without VAs occurring more than 30 days after LVAD implantation (5). However, "survival to transplantation" is a dramatically different outcome from "death while supported by LVAD"; in that study, the endpoint included a higher rate of transplantation in patients without late VAs versus those with late VAs and a higher rate of death in patients with late VAs versus those without late VAs. In patients with late VAs post-LVAD who died, cardiovascular death was implicated in 61.7%, mostly as a result of RV failure, electrical storm, and LVAD thrombosis. In patients without late VAs post-LVAD who died, non-cardiovascular causes of death were more common. In other observational studies, post-implantation VAs were associated with increased hospitalizations and

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From the Section of Cardiac Electrophysiology, Division of Cardiology, University of California-San Francisco, San Francisco, California, USA.

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morbidity related to ICD shocks and RV failure (6,7), as well as increased mortality (8,9). Indeed, the patient highlighted in the case reported by Corona et al. (2) had reduced cardiac output, manifest in part as ischemic colitis, resulting from recurrent VT at a relatively slow cycle length of 400 ms. There is clearly a subset of patients with an LVAD who require definitive treatment of VAs, particularly if heart transplantation is not an option.

Once the decision is made that nonpharmacological intervention is necessary, endocardial catheter ablation with an LVAD in place can have multiple challenges. Bundle branch re-entry VT, which may have a higher incidence after LVAD, can be effectively diagnosed and treated with RV access alone (10). LV ablation, however, can be limited by occasional obstacles to retrograde aortic access, difficult catheter manipulation within a decompressed LV cavity, and electromagnetic interference with surface electrocardiography, intracardiac electrogram recordings, and 3-dimensional (3D) mapping systems. Nevertheless, LV endocardial catheter ablation can be safe and effective. Experienced operators can avoid catheter entrapment in the inflow cannula without difficulty, although previously published case series have used devices with pump mechanisms remaining outside the chamber (1,10). The Jarvik 2000, a nonpulsatile axial-flow LVAD, has a miniaturized intraventricular blood pump, further reducing space in the chamber for catheter manipulation and theoretically increasing the risk for catheter entrapment. Epicardial access, now routinely achieved in the electrophysiology laboratory with percutaneous subxiphoid pericardiotomy in many patients, is more limited after LVAD implantation because of pericardial adhesions and intervening hardware. Surgical approaches using subxiphoid access or anterior thoracotomy have been previously described in LVAD recipients without the Jarvik 2000 device, although such hybrid procedures are not readily available in all centers, and short-term procedural complications were not uncommon (11,12). These factors and the case reported by Corona et al. (2) highlight the need for close interdisciplinary collaboration among the mechanical circulatory support team, cardiac surgeons, and electrophysiologists. Success is contingent on careful procedural planning, including preoperative imaging and arrhythmia analysis, acceptable LVAD pump speed parameters while trying to minimize electromagnetic interference, optimal surgical exposure, 3D mapping set-up, and goals for induction and ablation.

Some of these challenges in epicardial access after LVAD implantation may be mitigated by addressing the arrhythmogenic epicardial substrate before the LVAD is in place. We previously demonstrated that intraoperative high-density epicardial mapping during LVAD implantation is safe and efficient, and that an increased burden of epicardial scar may be associated with a higher incidence of post-implantation VA (13). The technique did not require a specialized hybrid operating room, only a stand-alone 3D mapping system, and the mapping process added a median of only 11.8 min to the LVAD implantation procedure. Empirical epicardial ablation, without arrhythmia induction, was performed in several patients, and a surgical cryoablation wand was used to treat relatively large areas over a short period of time. For nonurgent LVAD implantation procedures, standard percutaneous endocardial and epicardial mapping could also be considered pre-operatively. Particularly in patients who will not be candidates for heart transplantation and who have a known history of VAs (the most consistent predictor of post-LVAD arrhythmias), such an approach could yield long-term dividends in reduced morbidity and mortality.

In this case, the achievement of Corona et al. (2) represents a well-planned and expertly executed ounce of prevention for their patient—hopefully avoiding years of hospitalizations, ICD therapies, and morbidity. However, it also could be considered the pound of cure—a complex and risky procedure that is readily available in only a minority of centers and that perhaps could have been avoided with epicardial substrate homogenization at the time of LVAD implantation years earlier. What we need are more prospective, randomized studies on interventions to treat and prevent VAs in such patients, to determine whether there is a positive impact on morbidity and/or mortality. Patients who undergo pre-operative or intraoperative arrhythmia ablation at the time of LVAD implantation are excellent candidates for larger investigations.

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ADDRESS FOR CORRESPONDENCE: Dr. Joshua D. Moss, Department of Cardiac Electrophysiology, University of California-San Francisco, 500 Parnassus Avenue, MUE 4th Floor, San Francisco, California 94143, USA. E-mail: joshua.moss@ucsf.edu.

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