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Editorial commentary: Catheter ablation of ventricular arrhythmias: A changing landscape☆

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Clinical practice trends related to the management of idiopathic and malignant ventricular tachycardia (VT) have changed significantly in recent years. VT ablation is still more commonly performed at specialized quaternary care centers. However, over time, a large number of electrophysiologists have trained at experienced ablation centers and practice demands have encouraged a willingness to take on these complex cases. Therefore, a greater number of operators are presented with the procedural challenges of premature ventricular contraction (PVC) and VT mapping and ablation. Yet, the technique, technologic understanding and skill required to successfully and safely take on these cases continue to be a moving target. It is timely that Markman and Nazarian [1] provide a state-of-the-art review of PVC and ventricular VT management in this issue of *Trends in Cardiovascular Medicine*. The authors tackle complex and broad topics including PVCs and VT in structurally normal hearts, cardiomyopathy and inherited arrhythmia syndromes.

Important developments, some of which were noted in the review, include the following.

PVC management/ablation

1) Data on the risk of PVC-induced cardiomyopathy [2] and the predictors of ejection fraction improvement after successful ablation have increased the referral of patients for PVC ablation. Symptoms are no longer the only reason to consider ablation in idiopathic PVC cases.
2) The premise that the overwhelming majority of idiopathic PVCs originate from the right ventricular outflow tract (RVOT) is overstated and this presumption may effect procedural success as many operators are reluctant to perform more comprehensive mapping. Much of the early data suggesting RVOT origin did not include mapping of the left ventricular outflow tract, coronary cusps, coronary venous system and left ventricular (LV) summit. Successful ablation location does not necessarily imply that it is the exact site of origin or even the best location for ablation. Thin walled structures, intra-myocardial foci and preferential conduction may result in a PVC being labeled as 'RVOT' due to successful suppression with ablation there, without it being the true site of origin.
3) PVC functional characteristics such as coupling interval and circadian variability, and how those characteristics translate to PVC sites of origin, risk of PV-induced cardiomyopathy [4], and how PVCs will respond to intra-procedural drugs [3] at the time of ablation are becoming better understood. Previous literature has systematically characterized PVC morphology and relationship to sites of origin, but the understanding of PVC "behavior" remains limited.
4) Due to the increasing awareness of the anatomic complexity of the LV summit, a previously underappreciated site of PVC origin, expertise in new techniques such as the use of half normal saline, bipolar ablation, wire mapping (arterial and venous), alcohol ablation and coil embolization provide additional ablation options for PVCs that have a high failure rate with standard unipolar ablation only.

VT management/ablation

1) Active cardiac inflammation, often due to cardiac sarcoidosis, and in particular isolated cardiac sarcoidosis, may be more common than previously realized. Work in this area has led to more thorough assessments of patients with non-ischemic cardiomyopathy, particularly the use of PET-CT scans to assess for inflammation [5]. The best initial treatment for many of these patients may be immunosuppression with steroids, and not ablation.
2) For refractory VT, early intervention with ablation should be strongly considered. Procedural skills and experience have improved over recent years and the risks of VT ablation are relatively low when patients are hemodynamically compensated. However, when cardiomyopathy progresses, the risk of acute periprocedural decompensation increases. Retrospective data from Dinov and colleagues support the benefit of early intervention [6]. Further, the practice of waiting for multiple antiarrhythmics to fail prior to referral for VT ablation should be abandoned, as data show the addition of more than one antiarrhythmic has limited, if any, benefit [7].
3) Pre-operative imaging often changes the way we approach cases. For instance, epicardial mapping for many years was reserved for failed endocardial cases. However, MRI imaging often demonstrates transmural or mid-myocardial scars that are best approached with a combination of endocardial and epicardial mapping, regardless of whether it is the patient's first

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procedure. Alternatively, MRI may demonstrate only septal scar in which case the optimal approach may be better directed to bipolar septal ablation or other novel interventions that allow for deeper lesion formation. Cardiac MRI imaging in patients with non-conditional MRI devices has been shown to be safe and the use of wideband pulse sequences results in artifact free images in 90% of patients [8].

4) Bipolar ablation and the use of half-normal saline to form deeper lesion sets has greatly affected how we approach non-ischemic cardiomyopathy patients in particular. Given the propensity for mid-myocardial fibrosis and septal substrates especially in non-ischemic cardiomyopathy [9], these techniques must be available for targeting deep substrates, which unipolar ablation often cannot adequately reach.
5) The use of alternative or adjunctive therapies that involve modulation of the autonomic nervous system is becoming an essential component of a comprehensive VT management program [10]. Not all ventricular arrhythmias can be successfully ablated, and these alternative/adjunctive therapies play a critical role for challenging cases.

Future direction

We have made great strides in the management of PVCs and VT, but there remains much more to accomplish. Much of the research related to PVC-ablation involved ECG criteria for location of exit. While useful, we must now move toward a better understanding of PVC "behavior". What controls PVC coupling, preferential conduction, circadian oscillation and how can these characteristics be translated to better manage PVCs with both pharmacological and interventional strategies.

Structurally, VT involves three-dimensional circuits and a complex interplay of conduction block/slowing in the setting of structural (scar/fibrosis) and functional (autonomic) influences. We need mapping systems that can assess to some extent mid-myocardial conduction and the interplay of autonomic influences as well.

Conclusion

Scientifically we are making steady progress in understanding the structure-function interplay of PVCs and VT, which ultimately will lead to better ablation outcomes, while at the same time more clinicians are trained in complex VT ablation expanding the availability of this treatment option to patients.

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