Idiopathic Premature Ventricular Contraction Ablation
Prime Time or Second Line?*

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Repetitive premature ventricular contractions (PVCs) cause significant morbidity in a minority of affected individuals. Although most patients with symptomatic ectopy endorse non-disabling palpitations, more severe manifestations are relatively common. A reduction in cardiac output during ectopic beats can produce concealed mechanical bradycardia with a symptom complex similar to severe sinus node dysfunction (1). Frequent ventricular ectopy also can negatively and reversibly affect objective quality-of-life assessments (2,3).

Given the gravity inherent in the diagnosis of left ventricular (LV) cardiomyopathy, transformative therapies are of great interest to practitioners and patients alike. Before recognition of the link between repetitive PVCs and cardiomyopathy, many patients with potentially reversible systolic dysfunction were committed to escalating medical and device therapies while the causative arrhythmias were largely ignored. Several important studies have demonstrated marked improvement in LV dimension and function after PVC elimination (4–6). Catheter ablation provides an attractive alternative to medical therapy in these patients; however, previous reports often have presented single-center experiences with homogenous arrhythmia phenotypes.

The recent report by Latchamsetty et al. (7), in this issue of JACC: Clinical Electrophysiology, provides important insights into the efficacy and safety of catheter ablation in patients with idiopathic PVCs. They describe the procedural characteristics, complications, and outcome in a cohort of 1,185 patients who underwent PVC ablation at 8 international centers over a 10-year period. Approximately 90% of patients had symptomatic arrhythmias, and 21% of patients had a clinical suspicion for a PVC-related cardiomyopathy (defined as an ejection fraction [EF] <50%). Acute procedural success was defined as a >80% reduction in preoperative PVC burden. Although this cutoff may seem arbitrary in patients with symptomatic ectopy, it effectively limits the cumulative daily PVC burden in a patient with ventricular bigeminy to 10%. This metric is important, because separate series have found a 10% to 13% daily PVC threshold to provide 100% sensitivity in discriminating patients with PVC-related cardiomyopathy (6,8).

The site of PVC elimination was relatively diverse and included the right ventricular outflow tract (RVOT), aortic root, papillary muscles, and epicardium. Acute procedural success was achieved in 84% of patients, although significant heterogeneity in outcome was observed on the basis of PVC location. Procedural complications were relatively infrequent (5.2%), and most were due to vascular injury. Pericardial effusions occurred in 1% of cases, and a single patient had permanent atrioventricular block. Of note, no procedure-related embolic events or deaths were reported.

In the patients undergoing post-operative Holter monitoring (41%), persistent suppression was achieved without antiarrhythmic drugs in 71% at a mean follow-up of 20 months. Unfortunately, repeat ablation (mean: 1.3; range: 1 to 6) was required in 22% of patients, most commonly for PVCs originating from the papillary muscle (36%) or epicardium (38%). An RVOT PVC origin was associated with chronic PVCs.
elimination in multivariate analysis. In patients with presumed PVC-related cardiomyopathy, the mean daily PVC burden decreased from 27% to 5% post-ablation with a concomitant increase in LV EF from 38% to 50%. The EF increased by more than 10% in 67% of these patients.

This study (7) provides several important observations. First, it represents the largest experience to date of catheter ablation for idiopathic PVCs. The multicenter design and diversity of PVC sites of origin included enhanced the generalizability of the outcomes reported. The profile of procedural complications was favorable, with only one single patient experiencing permanent harm. These data are particularly powerful when one considers that the study procedures were performed over a decade, encompassing vastly different catheter and imaging technologies. One might anticipate more contemporary outcomes to be superior.

Other interesting findings related to the ablation outcomes bear further discussion. First, procedural outcome was strongly influenced by PVC location. The inherent complexity of ablating non-RVOT (e.g., epicardial or intracavitary) structures is familiar to operators, and these sites were more likely to be acutely unsuccessful and to require multiple procedures. Chronic recurrence rates were highest (~20%) for aortic root and papillary muscle PVCs. The use of real-time imaging (e.g., intracardiac echocardiography) may improve the procedural success in such cases by enhancing catheter contact with intracavitary structures (9). Despite multiple procedures, 19% of the cohort required antiarrhythmic agents to achieve PVC suppression; the specific agents used are not enumerated in the manuscript. A previous report found that class I and III antiarrhythmic agents reduced PVC burden by 82%; the response was fairly consistent for different sites of PVC origin (10). Thus, it is important to consider the anticipated PVC site of origin based on 12-lead electrocardiography when counseling patients about procedural success, and trialing an antiarrhythmic agent is a reasonable initial strategy in more complex cases. The relative efficacy of antiarrhythmic drugs and ablation cannot be determined with the design of the current study.

The duration of RF required to eliminate the clinical PVC (mean of 12 min) was surprisingly long; papillary muscle PVCs required an average of 26 min of ablation. The long-term implications of (potentially extensive) collateral damage to adjacent tissue are poorly understood and typically not considered when estimating the risks and benefits of ablation. Although the majority of patients with presumed PVC-related cardiomyopathy had significant EF improvement after ablation, 15% demonstrated further decline. Whether this was related to ineffective elimination of PVCs with ablation or a non-PVC-related cardiomyopathy (i.e., misclassification) cannot be determined from the data presented.

The current report (7) has several important limitations that should be highlighted. The determination of the primary efficacy outcome is confounded by a lack of Holter data pre-ablation in 27% of patients and post-ablation in 41% of patients; it is unclear to what extent these 2 populations overlap. The presence of PVC-related symptoms was the predominant indication for ablation; however, no follow-up data regarding subjective or objective reduction in symptom burden are presented. Thus, it cannot be determined whether achieving the primary ablation outcome of PVC burden reduction effectively mitigated arrhythmia symptoms. Approximately one-quarter of targeted sites were classified as “other,” with no specific information on the PVC location or ablation outcome. Primary echocardiographic data from the study centers were not available for independent review, which may limit the accuracy of the comparisons made (11).

Future studies are needed to better elucidate the temporal relationship of PVC onset, burden, and phenotype with the development of cardiomyopathy. In patients with pre-existing cardiomyopathy, relatively low burdens of PVCs could adversely affect cardiac function and may represent a novel therapeutic target. The mechanism of late recurrence of PVCs after ablation also requires further clarification, particularly because it confers a risk of recurrent LV dysfunction in affected patients (12).

In summary, Latchamsetty et al. (7) have provided a benchmark for the safety and efficacy of catheter ablation of idiopathic PVCs. There remains substantial room for improvement in single procedure efficacy; thus, it seems most prudent to individualize the treatment strategy, particularly in patients with more challenging sites of arrhythmia origin. The current report empowers providers to engage in these discussions with greater specificity.

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REFERENCES


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