The use of ventricular tachycardia (VT) ablation in patients with structural heart disease remains reserved primarily for those who experience repeated implantable cardioverter defibrillator shocks despite pharmacological and attempted pacing therapy. This fact is confirmed by the comprehensive 8-year report of the experience with VT ablation by the group from the Brigham and Women’s Hospital. Even at this very experienced center, most patients attempted pacing therapy. This fact is confirmed by the cardioverter defibrillator shocks despite pharmacological and primarily for those who experience repeated implantable ejection fraction.

Follow-up of 29% and 39%, respectively, would seem high low, and a recurrence rate after a median of 1 month of ischemic heart disease and NICM, respectively, would seem for eliminating all VT of 65% and 51% for patients with cardiomyopathy group and 59% in the nonischemic cardiomyopathy (NICM) group—were treated with amiodarone. This tendency to “hold back” on catheter ablation therapy for VT seems to be even more dramatic as a current practice standard than previously noted. In our own institutional experience, when comparing the clinical characteristics of consecutive patients referred for VT ablation with coronary disease over a comparable time period from the mid-1990s with the current decade (Table), we observed a significant increase in the use of implantable cardioverter defibrillator therapy and amiodarone before ablation. Patients also appear to have poorer left ventricular function as indexed by a decline in left ventricular ejection fraction.

It is important to determine the basis for this tendency to withhold or at least delay ablative therapy and determine whether it is justified. On a cursory review of the report by Sacher et al, published in this issue of Circulation: Arrhythmia and Electrophysiology, the success rate of VT ablation for eliminating all VT of 65% and 51% for patients with ischemic heart disease and NICM, respectively, would seem low, and a recurrence rate after a median of 1 month of follow-up of 29% and 39%, respectively, would seem high and perhaps justify a conservative approach. Importantly, although the ability to eliminate all inducible VT appeared to be a challenge, the ability to eliminate the targeted clinical VT was reported to be 80% for patients with NICM and 90% for patients with ischemic heart disease. Thus, the true clinical impact of ablative therapy, even in patients with structural heart disease, should be viewed with a greater degree of optimism. Furthermore, the reported elimination of amiodarone therapy in nearly two thirds of patients who were previously treated with the drug is reason enough to consider early ablative therapy in an effort to avoid the high risk of end-organ toxicity from long-term use of the drug. Finally, nearly half of the patients undergoing VT ablation in the report by Sacher et al had one or more unsuccessful ablation attempts before referral. The referral for ablation frequently represented a “last ditch” effort in a uniquely difficult group. The higher-than-anticipated mortality during long-term follow-up is consistent with the fact that many of the patients currently referred to tertiary care institutions for VT ablation will represent a unique subset of particularly ill patients. The fact that most of these patients still had VT control with ablation emphasizes its potential clinical utility.

Obviously, electrophysiologists who focus on efforts to advance VT ablation techniques and improve efficacy must also share some responsibility for the reluctance of the general medical community to embrace enthusiasm for VT ablation. Outcome data in large cohorts of patients has not been previously described. Additional reports that corroborate or dispute the efficacy and risk data noted need to be generated. Also of importance is the recognition that the widespread use of VT ablation techniques will only come with additional training and experience in the general electrophysiology community. Efforts to make this training a more routine part of the electrophysiology fellowship experience should be encouraged. The evolution of effective ablation techniques is still a work in progress and these efforts need to be hastened. It has only been within the last decade that a substrate-based ablation strategy was initially described. Sacher et al document that unstable VT is present in more than two thirds of patients with structural heart disease who are undergoing ablative therapy. These data, coupled with the acknowledged frequent use of amiodarone before ablation, which can suppress some VT during the procedure that will become manifest late after drug withdrawal, suggest that an aggressive substrate-based ablation strategy must be the rule rather than the exception. This substrate-based approach appears to be warranted, regardless of inducibility or hemodynamic tolerance of the VT. Only recently has a detailed delineation of the endocardial substrate for VT in a variety of subgroups of patients with NICM

Editorial

Ventricular Tachycardia Ablation
Moving Beyond Treatment of Last Resort

Francis E. Marchlinski, MD

It is important to determine the basis for this tendency to withhold or at least delay ablative therapy and determine whether it is justified. On a cursory review of the report by Sacher et al, published in this issue of Circulation: Arrhythmia and Electrophysiology, the success rate of VT ablation for eliminating all VT of 65% and 51% for patients with ischemic heart disease and NICM, respectively, would seem low, and a recurrence rate after a median of 1 month of follow-up of 29% and 39%, respectively, would seem high and perhaps justify a conservative approach. Importantly, although the ability to eliminate all inducible VT appeared to be a challenge, the ability to eliminate the targeted clinical VT was reported to be 80% for patients with NICM and 90% for patients with ischemic heart disease. Thus, the true clinical impact of ablative therapy, even in patients with structural heart disease, should be viewed with a greater degree of optimism. Furthermore, the reported elimination of amiodarone therapy in nearly two thirds of patients who were previously treated with the drug is reason enough to consider early ablative therapy in an effort to avoid the high risk of end-organ toxicity from long-term use of the drug. Finally, nearly half of the patients undergoing VT ablation in the report by Sacher et al had one or more unsuccessful ablation attempts before referral. The referral for ablation frequently represented a “last ditch” effort in a uniquely difficult group. The higher-than-anticipated mortality during long-term follow-up is consistent with the fact that many of the patients currently referred to tertiary care institutions for VT ablation will represent a unique subset of particularly ill patients. The fact that most of these patients still had VT control with ablation emphasizes its potential clinical utility.

Obviously, electrophysiologists who focus on efforts to advance VT ablation techniques and improve efficacy must also share some responsibility for the reluctance of the general medical community to embrace enthusiasm for VT ablation. Outcome data in large cohorts of patients has not been previously described. Additional reports that corroborate or dispute the efficacy and risk data noted need to be generated. Also of importance is the recognition that the widespread use of VT ablation techniques will only come with additional training and experience in the general electrophysiology community. Efforts to make this training a more routine part of the electrophysiology fellowship experience should be encouraged. The evolution of effective ablation techniques is still a work in progress and these efforts need to be hastened. It has only been within the last decade that a substrate-based ablation strategy was initially described. Sacher et al document that unstable VT is present in more than two thirds of patients with structural heart disease who are undergoing ablative therapy. These data, coupled with the acknowledged frequent use of amiodarone before ablation, which can suppress some VT during the procedure that will become manifest late after drug withdrawal, suggest that an aggressive substrate-based ablation strategy must be the rule rather than the exception. This substrate-based approach appears to be warranted, regardless of inducibility or hemodynamic tolerance of the VT. Only recently has a detailed delineation of the endocardial substrate for VT in a variety of subgroups of patients with NICM
been reported.6–8 A common link in the pathogenesis related to the perivascular anatomic distribution of scars has been noted, and unique characteristics associated with the different arrhythmia syndromes has been described.5,6,9,10 Importantly, substrate-based ablation techniques continue to be refined and the “best” strategy needs to be defined through carefully designed prospective, comparative studies rather than opinion.5,11–13 Also of note, an almost equally short period of time has elapsed since a percutaneous epicardial mapping and ablation technique was originally described by Sosa et al.14 This technique is now beginning to be more widely deployed, even at centers with significant experience in VT ablation. The importance of the epicardial substrate for VT in a variety of subgroups of patients with NICM has been reported.6,8,15,16 The increased success in VT control using epicardial ablation strategies, not only for VT in Chagas disease but also in right ventricular cardiomyopathy/dysplasia and hypertrophic cardiomyopathy, are indeed encouraging.17–19 Based on the distribution of the anatomic substrate for VT, it is anticipated that at least 50% of patients with NICM will benefit from epicardial mapping and ablation.15 This degree of use is nearly double that deployed by Sacher et al1 during the span of the current report. Finally, in addition to advances in technology that might allow us to better image the ventricular substrate, track online lesion formation, and ablate more deeply, there needs to be a further elucidation of the relationship between the VT substrate and VT arrhythmogenesis to better identify the best ablation targets in sinus rhythm and more uniformly guarantee acute success and long term efficacy.

The report by Sacher et al1 should be considered an important reference point on which we can build. It would have been helpful in that report to exclude or analyze separately young patients with right ventricular cardiomyopathy and congenital heart disease in the mortality analysis. If this were done and transplants were identified as a mortality equivalent, it would be possible to anticipate that mortality outcome profiles after VT ablation for patients with nonischemic and ischemic structural heart disease would have been more closely matched. The impact of recurrent VT on clinical outcome might also have been important to identify. If it can be demonstrated that mortality was indeed reduced in those patients who achieved VT control after controlling for ejection factor, then the argument for pushing forward with treatment that is not only palliative but also “lifesaving” would be even more pressing. The improved mortality outcome after VT ablation in patients with incessant VT has been recently demonstrated by Carbucicchio et al.20 Regardless of any minor shortcomings, the current report is a landmark article that gives us insight into the characteristics and outcome of patients undergoing VT ablation during the last decade and supports the consideration of VT ablation therapy earlier in the course of arrhythmia events in patients with structural heart disease. It also sets the bar high and identifies the final hurdles that we must overcome to provide uniformly safe and effective VT ablation therapy.

### Table. Comparison of Patient Characteristics Undergoing VT Ablation

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<tr>
<td>Male, n (%)</td>
<td>59 (91)</td>
<td>58 (89)</td>
<td>NS</td>
</tr>
<tr>
<td>Age (mean±SD), y</td>
<td>67±9.2</td>
<td>67±8.9</td>
<td>NS</td>
</tr>
<tr>
<td>LVEF (mean±SD), %</td>
<td>27±7</td>
<td>23±9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Prior ICD, n (%)</td>
<td>35 (54)</td>
<td>61 (94)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Prior antiarrhythmic therapy, n (%)</td>
<td>49 (75)</td>
<td>62 (96)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Prior amiodarone therapy, n (%)</td>
<td>32 (49)</td>
<td>51 (79)</td>
<td>&lt;0.01</td>
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</tbody>
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HbD indicates ischemic heart disease; NS, not significant; LVEF, left ventricular ejection fraction; ICD, implantable cardioverter defibrillator.

### References


**Keywords:** ablation, arrhythmia, tachycardia, ventricles
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