

## EDITORIAL COMMENT

# Ablation for Ventricular Tachycardia

## Is More Always Better? How Much More Is Too Much?\*

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Ventricular tachycardia (VT) remains one of the major causes of morbidity and mortality after myocardial infarction (MI) (1,2). Surviving myofibers within scar tissue have been shown to represent the arrhythmogenic substrate of post-infarction VT (3). Identification of these myocytes and myofiber bundles is possible by recording electrograms from the scarred endocardium with a mapping catheter (4). This can be best achieved while the patient is in VT. Unfortunately, most VTs are not hemodynamically tolerated, and therefore mapping during VT is not possible (5). Identification of specific electrograms, mainly isolated potentials during sinus rhythm, has been linked to critical sites of VT,

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thereby avoiding the search for critical areas during nontolerated VT (6). Although the majority of critical areas of post-infarction VT are located in the endocardium, not all components critical to a VT circuit can be reached from the endocardium, and characterization of the entire circuit is often not possible. It may be best to imagine the VT circuit as a 3-dimensional structure that may have components in the endocardium (most often the exit area), the mid-myocardium, and the epicardium. How much of the circuit is accessible endocardially is not known. Epicardial and intramural re-entry circuit locations are well recognized in the surgical literature and are an important cause of failure of endocardial ablation (7,8).

Another problem of the mapping/ablation procedure for VT is the difficulty and often unreliability of VT inducibility. Procedural endpoints may therefore be unreliable as well. Noninducibility is the preferred outcome of an abla-

tion procedure, especially because there are reports that link this endpoint with improved long-term outcome (9). Lack of inducibility of VT after ablation, however, does not unequivocally predict a better outcome after ablation (10). Therefore, other endpoints and ablation techniques must be sought. Scar homogenization might be a such a technique that addresses the entire scar and may improve long-term outcome, provided that patients are noninducible after the ablation procedure. This innovative ablation approach for patients presenting with electrical storm (ES) is presented by Di Biase et al. (11) in this issue of the *Journal*. Ninety-two consecutive patients with ischemic cardiomyopathy and ES were enrolled at 5 centers. Forty-nine patients underwent conventional endocardial mapping and ablation of select areas based on activation, entrainment, substrate mapping, and pace mapping (Group 1). The subsequent 43 patients (Group 2) underwent both percutaneous endocardial and epicardial mapping and ablation of all abnormal potentials within and around the scar (homogenization procedure). Four patients in Group 1 also underwent epicardial mapping and ablation, as VT was still inducible after endocardial ablation. In both groups the electrophysiological endpoint was VT noninducibility. Although in Group 2 all patients underwent epicardial mapping, only 14 (33%) had ablation performed in the pericardial space due to the presence of delayed, fragmented, or low voltage potentials. The authors report a 100% VT noninducibility rate with 3 extrastimuli and isoproterenol (up to 5  $\mu\text{g}/\text{min}$ ) at the end of the ablation in both groups. Ablation with endo- and epicardial homogenization was associated with dramatically lower VT recurrence rate (hazard ratio: 0.38,  $p = 0.019$ ). However, before adopting this technique, several issues need to be addressed:

- This is not a randomized study. The first group was more of a historical comparison, with a longer follow-up than the second group, although the analysis performed with a shorter follow-up time tried to adjust for this fact. However, more long-term follow-up is necessary in order to fully assess the value of different ablation techniques.
- The endpoints of the scar homogenization technique are not fully described, and it is not clear how much ablation is required at each site where radiofrequency energy is delivered. If the endpoint was to render the entire scar nonexcitable, then this would have been a clear endpoint; however, this is difficult and potentially impossible to achieve with current ablation catheters. Several areas probably would need more ablation than other areas to render a particular VT noninducible, but no data are provided regarding this issue. For example, if VTs remain inducible in the “homogenization group” after the entire scar is covered with ablation lesions, identifying the areas that require more ablation requires a more localizing approach, such as pace mapping. This argues for a procedure that would take both electrogram characteristics and localizing information of VTs into consideration.

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However, the homogenization technique has particular advantages if the clinical VTs are not inducible.

- A large part of the population currently undergoing VT ablation in the United States has had prior coronary artery bypass grafting and therefore would not be eligible for a percutaneous epicardial ablation procedure (12). The true value of the epicardial approach is not clear from the study design, although it appears that patients who underwent an epicardial approach did not have a better outcome compared with patients who underwent an endocardial approach only.
- The investigators ought to be congratulated in achieving 100% noninducibility in all patients with both approaches. The participating centers are very experienced in complex ablation procedures, and one needs to be careful in extrapolating the results to other centers. Although the technique of ablating the entire scar may seem appealing, it remains to be seen whether the excellent results of midterm follow-up can be replicated with essentially no periprocedural complications despite an epicardial approach in half of the patients. This is surprising in view of the complications described even in experienced centers with an epicardial approach (13,14). Furthermore, no additional anticoagulation was used after ablation despite a more encompassing ablation approach. Current expert opinions do call for at least temporary anticoagulation if more extensive ablation is performed (15).

In summary, the study by Di Biase et al. (11) presents a novel approach to ablation of ES after myocardial infarction. This approach provides some evidence in favor of tissue homogenization as an ablation strategy. Further studies are needed, however, to determine the incremental benefit—if any—of the epicardial ablation portion used in this method, in light of the potential complications that such an approach carries.

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