A Moving Target for Catheter Ablation of Ventricular Tachycardia
Ablation of Scar or Arrhythmia?*

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Catheter ablation of ventricular tachycardia (VT) reduces recurrent VT across multiple clinical settings, from electrical storm to preemptive therapy after a first occurrence of VT (1). Approaches for ablation include targeting the arrhythmia using electrophysiological approaches (entrainment mapping, pacemapping, and electroanatomic mapping) and substrate modification to reduce the arrhythmogenicity of a scar. Superiority of a single approach has not been demonstrated consistently, although recent trends associate more extensive ablation with less VT recurrence (2–4). There is inconclusive evidence that mapping during VT improves long-term ablation success compared with a substrate-based approach in sinus rhythm (5,6).

“Substrate-based” approaches use electroanatomic mapping to carry out ablation predominantly during sinus rhythm and have variable, operator-dependent interpretations. Although many centers undertake a refined search for an induced VT morphology, others ablate the scar without any specific arrhythmia targeting. The rationale for the latter approach is that more extensive tissue destruction is necessary to emulate surgical resection. Furthermore, most patients have multiple VTs and the search for a single isthmus may be distracting and time-consuming. The counter argument is that not all low-voltage regions are critical components of re-entrant circuits, and methods that identify the most arrhythmogenic areas may allow focused radiofrequency delivery in higher-yield regions (7) and avoid unnecessary ablation at bystander sites.

The multicenter, prospective, randomized trial presented by Di Biase et al. (8), in this issue of the Journal, compares a “scar-based” with a “VT-based” strategy. In this study 118 patients with post-infarct cardiomyopathy and recurrent stable VT were randomized to ablation limited to “clinically stable” induced VTs versus an extensive homogenization strategy. In the limited strategy arm, only induced clinical and hemodynamically stable VT were randomized to ablation limited to “clinically stable” induced VTs versus an extensive homogenization strategy. In the limited strategy arm, only induced clinical and hemodynamically stable VTs were targeted using standard entrainment and pacemapping techniques, and ablation was confined to scar regions that were characterized as isthmuses. In the homogenization arm, extensive ablation was performed throughout scar, targeting all local potentials with fractionation and locally delayed activity and the low-voltage regions were remapped as an endpoint to demonstrate elimination of abnormal electrograms. Epicardial ablation was performed only in patients that remained inducible for clinical VT endocardial strategies.

Less recurrent VT occurred in the homogenization group compared with the limited approach for clinical VTs (84.5% vs. 51.7%; p < 0.001) at 1-year follow-up. No differences in mortality were observed, but a decrease in rehospitalization was observed in patients treated with a homogenization approach. No differences in post-ablation ejection fraction were observed between the 2 approaches, lessening concerns that deterioration in systolic function may result from extensive ablation.

Several aspects of the study warrant discussion. VT induction was not required in the scar

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homogenization group, yet 38% of patients underwent induction of VT at the operator’s discretion, suggesting that many patients had a hybrid approach including targeting of induced VTs. This may have enhanced the efficacy of the homogenization arm. The authors suggest that procedural time was shorter with a scar-based strategy compared with an arrhythmia-based approach once these cases were excluded from analysis. Additionally, 13% of patients assigned to ablation of clinically stable VTs had adjunctive hemodynamic support, illustrating that unstable VTs were likely targeted in such patients. Finally, an additional on-treatment variable of total radiofrequency application time (35 vs. 68 min) must be factored in to account for the difference in efficacy.

This study enrolled patients with drug-refractory, hemodynamically tolerated VT, and thus these data are not applicable to patients with only unstable VT, which comprise ~40% of patients referred for ablation (9). All patients had implantable cardioverter-defibrillators, and it is difficult to ascertain the hemodynamic stability of VT if therapies intervene rapidly. In contrast, the VTACH study enrolled patients with tolerated VT before implantable cardioverter-defibrillator implantation (10). Second, the determination of whether a clinical VT is “clinical” has significant limitations, because similarity in cycle length is not as predictive as 12-lead morphology. Sedation and anesthesia may influence the morphology and cycle length of induced VTs (11). The imperfect and relatively arbitrary identification of the clinical VT based solely on tachycardia rate is highlighted by a mean cycle length of 399 ms among the “clinical” VTs induced, compared with 376 ms among “nonclinical” VTs, with a wide standard deviation of 100 ms.

Although a scar-based strategy shifts the intraprocedural proportion of mapping to ablation time toward more therapeutic ablation time upstream, this approach may have limitations because it is estimated that up to 22% of patients with structural heart disease have focal mechanisms of arrhythmia that may not be addressed without identification of a target VT (12,13). Additionally, there may be intellectual resistance to treating VT with a purely anatomic approach. Focused approaches may obviate extensive ablation of nonarrhythmogenic regions of scar, which may be time-consuming and unnecessary. A more refined strategy targeting the entrances to downstream late activation within channels holds promise to achieve homogenization using fewer ablation lesions with 86% to 91% freedom from VT recurrence (14,15). Finally, there is no objective and reproducible measure to quantify and demonstrate elimination or modification of an abnormal electrogram within scar.

The current study convincingly demonstrates that more extensive ablation is more effective. Recurrence rates after ablation are as high as 50% to 70% in scar-related VT, which remains far inferior compared with early surgical success rates in the post-infarction setting (2,16-18). When ablating spatially complex scar with a small-tipped catheter, redundant, multiple, and expansive lesion sets are necessary to more closely mimic surgical resection. Refinements in current ablation technology and alternative energy sources allowing for greater destruction of arrhythmogenic tissue are needed.

The authors are to be congratulated on a well-designed, prospective, multicenter effort providing high-level evidence advocating extensive scar modification beyond targeted and induced VTs. As more examples of mortality reduction associated with successful VT ablation emerge (9,19), broader and earlier implementation is likely, which brings identification of the most optimal strategies to the forefront of clinical investigation. Randomized, multicenter studies in VT ablation are few to date and the present study sets the tone for the prospective collaborative work that lies ahead in this evolving field.

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