Sinus Rhythm Targeting of Channels for Ablation of Postinfarction Ventricular Tachycardia

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Scar-related reentry is the most common cause of sustained monomorphic ventricular tachycardia (VT) in patients with structural heart disease. These scars are comprised not only of dense fibrosis but also contain surviving myocyte bundles that support reentry. The separation of myocytes creates a circuitous path (zig-zag conduction) that slows conduction despite the presence of relatively normal action potential upstrokes.\textsuperscript{1,2} Gap junction remodeling and ion channel alteration may also contribute to slow conduction.\textsuperscript{3} After myocardial infarction, the scar evolves as part of the ventricular remodeling process, and acute reperfusion favorably influences this process.

Of 689 patients with acute ST elevation myocardial infarction who received acute percutaneous intervention for reperfusion reported by Zaman et al,\textsuperscript{4} only 17% had a left ventricular ejection fraction <40%, and of these, only 32 (=5% of the total group) had inducible VT when studied early after the infarction. By 1 year, only 19% of the patients with inducible VT had spontaneous VT. Interestingly, the VTs that occurred spontaneously (a mean of 11 months after the infarct) were substantially slower as compared with those that were induced early after infarction. Thus, it seems likely that the ongoing remodeling efforts result in conduction slowing in or around the channels of surviving myocytes in the infarct that foster reentrant VT. The risk of VT may increase over years. The time between infarction and referral for VT ablation was an average of 16 years for patients who receive acute infarct reperfusion in one study.\textsuperscript{5}

Once VT is clinically manifest, the reentrant substrate is relatively stable, such that the risk of recurrence exceeds 40% by 2 years. An implantable defibrillator is protection against sudden death, but recurrent VT is associated with worse outcomes.\textsuperscript{6} Catheter ablation has an important role in reducing the frequency of recurrent VT and has also been shown to reduce recurrences after the initial presentation of VT, although whether this would translate to a survival benefit is not known.\textsuperscript{7-9} The inability to reach and permanently transect intramural portions of reentry circuits, failure to detect all relevant reentry circuits, and further remodeling of the infarct with the emergence of new circuits may all contribute to recurrences.

The optimal strategy for catheter ablation is not clear. When a channel for a reentrant VT can be identified and confirmed by entrainment, ablation of that channel generally abolishes that VT. However, these maneuvers must be performed during VT, which is often limited by hemodynamic intolerance of VT. In addition, most patients have multiple VTs inducible, indicative of multiple potential reentry circuits and possibly multiple channels. These considerations have fostered substrate mapping approaches in which ablation targets are selected during stable sinus or paced rhythm, seeking to ablade all potential channels that could support a VT reentry circuit.\textsuperscript{10} The infarct region is identifiable as an area of relatively low voltage (<1.5 mV bipolar electrograms), and markers of potential channels in and adjacent to the low-voltage region include electrograms inscribed after the end of the QRS (late potentials), multicomponent electrograms that split out during pacing, consistent with asynchronous activation of myocyte bundles and slow conduction, and long stimulus to QRS (S-QRS) intervals during pace mapping.\textsuperscript{11-13}

These sinus rhythm mapping, substrate-guided ablation approaches maintain hemodynamic stability during the procedure, but there are limitations. Substrate mapping does not distinguish abnormal bystander areas that are not involved in a VT circuit from clinically relevant channels.\textsuperscript{14,15} Although a bystander for one VT may be involved in another VT circuit, it seems likely that bystanders lead to unnecessary ablation, prolonging procedures with the potential to increase risks. Channels defined by functional conduction block may escape detection with substrate mapping. Furthermore, a typical 3.5-mm electrode used for ablation may not be able to detect all low-amplitude electrograms from potential channels. At some sites where no potentials are measurable by the 3.5-mm electrode, pacing captures with a long S-QRS, indicating the presence of viable myocytes, and these areas can be part of a reentry circuit. Even with extensive substrate ablation approaches, a VT recurs in 20% to 53% of patients and often seems to be due to the inability to achieve durable ablation of portions of VT circuits.\textsuperscript{16,17}

In the current issue, Nayyar et al\textsuperscript{18} attempt to relate potential channels in infarct scars to channels supporting inducible VT. High-density mapping was achieved with a multipolar, 5-splined catheter. This catheter has 1-mm electrodes facilitating the detection of low-amplitude potentials that may not be appreciable with ablation catheters using larger electrodes. The authors attempted to reconstruct the course of channels within the low-voltage infarct region by plotting the location of adjacent sites where pacing produced the same QRS morphology, requiring that ≥1 site has a prolonged S-QRS >40 ms.
consistent with slow conduction away from the pacing site, and emerging from the infarct to the surrounding myocardium at an exit region in the infarct border. Patients had multiple channels identified by these criteria, but only 24% of channels (a median of 2 per patient) had QRS morphologies during pace mapping that matched an induced VT (which they refer to as VT channels), suggesting that the majority were bystanders. Although one would assume that the channel exit should connect to the border of the infarct, only 48% of channels had a segment identified in the border zone (defined by an electrogram voltage of 0.5–1.5 mV). The reasons for this finding are not clear. Perhaps, these exits were epicardial to the portion of the channel identified or require greater density mapping along the border zone for detection. Compared with non-VT channels, VT channels were longer and associated with slower conduction as indicated by longer S-QRS intervals and longer differences in S-QRS over the length of the channel. Although the authors attempted to calculate conduction velocity for the channels, it should be recognized that, with this resolution of mapping, any calculation is only a crude reflection of conduction velocities.

The study provides further interesting data on the relation of channels identified by pace mapping to electrogram markers of the VT substrate. Only about a quarter of the sites with abnormal fractionated or late electrograms were in a VT channel. Abnormal electrograms had poor specificity but high sensitivity for locating VT channels. However, VT channels were noted to have longer fractionated potentials and more poorly coupled late potentials. Additionally, it was not possible to identify channels visually in the electroanatomic mapping system by manipulating voltage thresholds, a technique occasionally attempted clinically. Thus, it seems that electrogram markers and pace mapping markers of channels provide complementary information. The limited relation of late potentials to VT channels is consistent with a previous study. The use of rapid pacing or extrastimuli to induce delay or conduction block between myocyte bundles causing split potentials, as referred to by Jaïs et al in their definition of local abnormal ventricular activities, likely improves the detection of channels by electrogram criteria.

There are several limitations and caveats to this interesting study, as acknowledged by the authors. It is important to recognize that electrogram characteristics are importantly influenced by the recording techniques. The small electrodes with relatively narrow spacing used have a smaller field of view compared with larger electrodes on ablation catheters, which might thereby record fractionated electrograms over larger areas. Applying the same voltage parameters for border zone and dense scar may not identify precisely the same region as when mapping with a larger electrode. The morphology of VTs for comparison with pace mapping was obtained by inducing VT with programmed stimulation. It is likely that not all potential VTs could be induced. As the authors recognize, pace mapping near a reentry circuit exit produces a QRS similar to VT, but pacing at more proximal channel sites may allow the stimulated wavefront to take a different exit from the scar, producing a different QRS morphology. If the same circuit gives rise to 2 different VTs, a single channel may be double-counted. If one of the VTs was not induced, one part of the channel would have been considered a non-VT channel. Their analysis of shared isthmuses based on pacing is important in this regard, but may nonetheless fail to detect some interactions between channels. The pacing rate can influence conduction and development of conduction block that may define reentry circuits, and all pacing in this study was relatively slow, as is common to avoid undesired hemodynamic consequences and initiation of VT during mapping. As for all catheter mapping studies in this population, monomorphic VTs are often relatively slow. Although these reentry circuits often seem to have anatomically determined channels, it is likely that functional conduction block contributes to other reentry circuits, the substrate for which may not be identified by these methods. Determining the QRS morphology, which is needed to apply this pace-mapping method, is not always possible for fast VTs.

It would seem that these methods may allow a more parsimonious approach to selecting ablation targets, but this study did not specifically assess ablation of channels identified by these criteria for abolishing VT. The authors targeted sites with abnormal scar-related electrograms and sites with long S-QRS intervals for ablation. All inducible VTs were abolished in 64% of patients, and another 32% had inducible VTs modified. During follow-up, one quarter of patients had recurrence of VT, which is fairly consistent with other studies. Interestingly, at repeat ablation in 4 patients, the majority of inducible VTs were new morphologies, 2 matched a previous VT and 1 matched a previous shared non-VT channel pacing morphology, suggesting that some of the potential channels that do not correspond to induced VT morphologies should be targeted for ablation. A systematic comparison of approaches to the selection of ablation sites is needed.

Postinfarction VT has a complex substrate that slowly evolves over years after infarction. The methods to identify potential arrhythmogenic substrate during stable sinus or paced rhythm have enabled ablation of multiple and poorly tolerated VTs in many patients with recurrent VT. Ablation has also been shown to reduce recurrences of VT after an initial episode. However, the procedure is still often perceived as a complex undertaking in a sick patient population, and optimal methods are not yet completely defined. Thus, we and others continue to encounter patients who are not considered for VT ablation until they have had frequent episodes or VT storm with adverse consequences, including posttraumatic stress disorder and toxicities of antiarrhythmic drugs. Studies such as this one, which help better understand the VT substrate, will hopefully lead to improved, defined ablation approaches with better outcomes.

Disclosures
Dr Stevenson is coholder of a patent for needle ablation that has been assigned to Brigham and Women’s Hospital. Dr Tedrow has received consulting fee from St Jude Medical and Boston Scientific, as well as nonsalary research grant from Biosense Webster and St Jude Medical.

References


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