High Density Pace-Mapping for Scar-related Ventricular Tachycardia Ablation

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Conflicts of Interest :

Dr. Stevenson has received speaking Honoria from: Boston Scientific, Medtronic, Abbott, Johnson and Johnson, and Biotronik; he is co-holder of a patent for irrigated needle ablation that is consigned to Brigham and Women's Hospital.

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Despite advances in medical and interventional therapies, ventricular tachycardia (VT) due to reentrant activity within complex regions of myocardial scar remains a common late complication of myocardial infarction.[1] While implantable defibrillators (ICD) may prevent sudden death, ICD shocks are painful and impact quality of life[2]. Catheter ablation reduces the likelihood of ICD therapies and it's role early in the course of disease is expanding[3–5]. However, several factors limit the success and safety of catheter ablation procedures. Scar-related reentry circuits can be large with a critical isthmus shared by multiple loops. Ablation of the isthmus is associated with a low risk of recurrence of that VT[6,7]. The critical isthmus can be identified during VT by detailed activation mapping and entrainment. However, prolonged mapping during VT is often not feasible

or desired. Patients undergoing VT ablation often have severe systolic heart failure as well as other comorbid conditions. VT is often not hemodynamically tolerated and even when tolerated, prolonged time in VT may lead to decompensation. Strategies to limit initiation and mapping of VT may improve procedural safety[8]. Methods to guide ablation based on characterization of the sinus rhythm substrate alone have generally shown good results[9]. A number of approaches have been applied, including ablation over the entire low voltage area (scar homogenization)[10]. While this is often successful, areas of scar can be quite extensive, and undoubtedly this technique leads to ablation of more areas than absolutely necessary for success. This approach is also more effective if epicardial ablation is routinely included, which has the potential to increase procedural risk. A strategy to focus on the critical regions, particularly when a clinically relevant VT is known, remains a reasonable first step in the procedure. A variety of electrogram markers of critical regions have been described including late potentials, potentials that display variable coupling to surrounding tissue during programmed stimulation[11] , and areas of slow conduction identified by high density mapping [12,13]. While these are likely to increase the specificity of ablation targets compared to electrogram voltage alone, they are also seen at bystander areas[14].

Pace-mapping during sinus rhythm is useful to help identify the general location of focal arrhythmia sources,[15] and can also be used in scar related reentry.[16,17] At the reentry circuit exit region the paced QRS morphology often resembles the VT QRS, and this will also occur at sites proximal to the exit provided that the stimulated wavefront follows the reentry path to the exit. A stimulus – QRS $>$ 40 ms is also consistent with slow conduction away from the pacing site, that can be a marker for reentry substrate[17].

In this issue of the *Journal of Cardiovascular Electrophysiology,*Guenancia et al. review their technique of using high density pace mapping to guide VT ablation[18]. Their method takes advantage of software available in electroanatomic mapping systems that assigns a measure of correlation between two different QRS morphologies; in this case the VT and the paced QRS morphology.[19] A pacing correlation map is generated by pacing multiple sites within the ventricle and color coding the algorithmically derived score for display at each point on the anatomic map. Sites near the exit from the reentry circuit isthmus, typically along the border of a scar, will display good correlation with induced VT. As one moves along the isthmus deeper into the low voltage scar the S-QRS prolongs due to the conduction time between the pacing site and the exit region. If the isthmus is anatomically defined, such that it is present during VT and sinus rhythm, the QRS morphology remains similar to the VT as long as the paced wavefront follows the isthmus out to the exit. Moving to the entrance or adjacent sites outside the isthmus can produce an abrupt transition to a markedly different paced QRS because the wavefront can propagate away without following the path of the isthmus.[20] Thus, the pace-map correlation maps can outline the location of a reentry circuit isthmus during sinus rhythm, as they illustrate.

Their method can also help identify cases in which the critical isthmus is not located on the surface being mapped. When the VT circuit is epicardial or intramural, the earliest endocardial activation may appear focal. Similarly, the pace-map correlation maps may reveal a concentric or focal pattern of matching, potentially allowing recognition of this situation without the need for activation mapping during VT.

We agree with the fundamental principles described, and feel this technique can be a helpful substrate mapping approach. There are several caveats. Evaluation to clarify its specificity and sensitivity is limited. The authors report that in their unpublished experience an abrupt transition is seen in the majority of post-infarct cases, they have also published a series of 10 post-infarct patients undergoing VT ablation during which the pacing correlation maps visually matched VT activation maps.[21]

This technique is likely to be effective in cases where the VT isthmus is confined to the ventricular surface being mapped. Pacing can capture deep to the endocardium depending on current strength.[22] Whether this technique can detect intramural isthmuses and whether deep tissue that can be captured with pacing can also be ablated from the pacing site is not clear.

It is important to point out that very good correlations with VT can be observed pacing in an outer loop immediately adjacent to the exit where one would not anticipate RF ablation delivery would be effective.

If a focal pattern is seen on both the endocardial and epicardial surfaces very little can be inferred about the VT circuit; the site with better correlation would be expected to be closer to the exit. In this setting entrainment during a brief episode of induced VT with assessment of the post-pacing interval can potentially clarify the proximity to the reentry circuit.

During VT, areas of functional conduction block may be present that are absent during sinus rhythm. Functional block can also occur remote from the reentry isthmus and alter activation wavefronts during VT changing the QRS morphology. Theoretically it is then possible to have poor correlation between the VT and paced QRS at its exit. In animal models of post-infarction VT exit regions have been shown to harbor very slow areas of conduction which could be prone to altering total ventricular activation during VT.[23].

We would caution against generalizing these techniques to patients with dilated cardiomyopathies where confluent regions of low voltage scar are absent. Diffuse interstitial fibrosis may play a greater role in some of these VT circuit and anatomically fixed isthmus sites are less likely to be present.

Further study is needed before utilizing this technique when anatomical structures within the ventricle are involved in the VT circuit. Structures such as the moderator band may by definition have multiple exits and varied QRS morphologies[24], and papillary muscles may display large areas of similar paced morphology[25], potentially distorting pacing correlation maps.

This technique is unlikely to correctly characterize VT circuits that involve a portion of the cardiac conduction system as occurs in some scar-related VTs and in bundle-branch reentry.[26] These circuits may demonstrate a focal pattern at the left or right ventricular apical septum on pacing correlation maps due to the long, insulated nature of the reentrant circuit itself, and ablation at the exit site is very unlikely to be effective.

This strategy of high density pace mapping adds to the available substrate mapping methods for guiding VT ablation while limiting VT induction. This strategy does not rely on electrogram interpretation, making it of particular interest in regions of very low voltage. Indeed, when utilizing larger recording electrodes, such as an ablation catheter, pacing will often reveal the presence of excitable tissue where a local electrogram is not always apparent. In post-infarct ventricular tachycardia circuits with a well-defined scar and a short anatomically bounded isthmus, pacing correlation maps are likely to be revealing. More study is warranted to further assess this method in relation to other substrate mapping methods, in complex substrate with intramural components, and in other disease substrates. It is useful to have multiple tools in the tool box. More studies are needed to further define which tools work best for which substrate.

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