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Magnetic Resonance Identification of the Ventricular Tachycardia Critical Isthmus: Finding the Needle in the Haystack

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Significant strides have been made in development of catheter based therapeutics for cure of malignant ventricular arrhythmias. Notable initial milestones included the first intra-cardiac recordings made by Hecht (1), and demonstration of catheter based cardiac stimulation by Furman and Robinson (2). Later, Wellens demonstrated that programmed electrical stimulation could reliably initiate and terminate ventricular tachycardia (VT) circuits in susceptible individuals (3). These tools allowed electrophysiologists to grow in their understanding of VT mechanisms, critical pathway and sites of origin, and potential new therapeutics. In 1978, Josephson demonstrated that intra-operative mapping of VT circuits can lead to identification and curative resection of the critical isthmus for VT maintenance (4–6). Once excisional cure was demonstrated, techniques were developed for non-surgical destruction of targeted tissue by high-energy shocks (7–9), and later by radiofrequency energy (10–13). Since then, numerous important contributions have been made to optimize techniques for VT ablation. Of utmost importance, is the development of electrophysiology criteria for distinguishing the critical isthmus from bystander areas adjacent to the VT circuit (14). The critical isthmus for VT is often in diseased tissue that exhibits slow conduction properties. However, identification of a slow conduction zone does not necessarily imply vulnerability of the patient to, or participation of that slow zone in a VT circuit. Electrophysiology maneuvers from the slow zone in question are an integral part of the mechanistic study of VT and the formulation of an approach to eliminate it. Due to the complex nature of the information derived from such studies, three-dimensional mapping systems have been developed to summarize the electrical and anatomic information gathered during the study (15,16). Such systems simplify the localization of diseased and slow conduction zones via voltage and activation mapping, but the solution to the VT puzzle is

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only attainable through the appropriate use and understanding of electrophysiology maneuvers.

Cardiac magnetic resonance (CMR) is capable of providing high resolution images of intra-myocardial scar through the late gadolinium enhancement (LGE) technique (17–19). Heterogeneous tissues surrounding areas with dense LGE are hypothesized to be the imaging equivalent of slow conduction zones in patients with ischemic cardiomyopathy (20,21). However, while the relationship between induciblity of VT and extent of LGE in ischemic cardiomyopathy has previously been reported (22,23), little data exists regarding direct electrophysiology measures and maneuvers from heterogeneous sites identified by CMR (24).

In this issue of the *Journal*, Perez-David et al present the results of a study that compares LGE features using a 1.5 Tesla magnetic resonance scanner, in 18 patients with sustained monomorphic VT, to 18 matched controls. The authors chose to use Yan et al's method for defining the infarct core and heterogeneous tissue as regions with signal intensity threshold greater than 3 and between 2–3 standard deviations above remote normal myocardium, respectively (20). Continuous heterogeneous corridors were then defined as the presence of heterogeneous tissue in consecutive planes surrounded by scar and connected to normal myocardium by at least one side. The underlying hypothesis that slow conduction channels during electrophysiology study would correspond to continuous heterogeneous corridors defined by CMR was then tested. The VT and control groups were similar in scar, heterogeneous tissue, and overall myocardial mass. However, patients in the VT group were more likely to exhibit continuous corridors of heterogeneous tissue. To make the images most suitable for comparison to endocardial voltage maps, the authors then created 3-dimensional color coded shells displaying the subendocardial signal intensity distribution. Continuous corridors of diseased but conducting tissue on endocardial voltage mapping corresponded to continuous corridors of heterogeneous tissue identified by LGE. Of 26 total corridors in 17 of 18 patients with VT, 15 corresponded to a critical isthmus for VT. These results are remarkable. Perez-David and colleagues have demonstrated the biologically plausible but up to now unproven connection between heterogeneous tissue on CMR and slow conduction zones identified by endocardial mapping. Additionally, they have shown that sites that electrophysiology maneuvers identify as critical VT isthmus sites often reside in heterogeneous tissue identified by CMR.

These findings have important differences with previous studies. In contrast to findings of previous publications (22,23), the VT and control groups of the current study were not different in scar and heterogeneous tissue mass. This inconsistency may be attributable to differences in patient populations and definition of infarct core and heterogeneous tissue on LGE, or the use of induciblity at electrophysiology study as a surrogate of spontaneous VT. Alternatively, the inconsistency may reflect the fact that a marker of risk in a larger population may not offer adequate resolution in an enriched sample of at risk individuals. If validated in future studies, the association of continuous corridors of tissue with spontaneous VT may improve the arrhythmic risk stratification of patients with ischemic cardiomyopathy. Another inconsistency with previous studies resides in the location of critical VT zones in the current study versus that performed by Desjardens et al (24). Desjardens et al found that the majority of critical VT isthmus sites were within the infarct core as defined by LGE, whereas Perez-David et al found all critical isthmus sites within heterogeneous tissue. This difference may reflect current CMR shortcomings in scar resolution and volume averaging in the infarct core and border zones. Until higher resolution images capable of resolving the intricate architecture of fibrotic tissue and surrounding healthy myocytes are clinically feasible (25), electrophysiologic characterization of CMR surrogates will be limited by the reality that the currently visualized "infarct core" and

"heterogeneous tissue" is a mixture of true infarct core, true heterogeneous tissue, and artifacts, particularly those produced by volume averaging of infarct core with healthy tissue.

The authors have presented data to support further research in visualization of the substrate for VT. Identification of the infract core and its associated heterogeneous tissue prior to the electrophysiology study and integration of such information into mapping systems will likely decrease procedure and fluoroscopy duration by focusing our attention on areas with diseased tissue. However, the electrophysiologist must remember that LGE image signal intensity is very sensitive to the assigned inversion time, poor ECG gating in the setting of arrhythmia, artifacts from fat in the atrio-ventricular groove or epicardium, and artifacts from respiratory motion. Additionally, volume averaging may represent a perfectly sharp but slanted scar border as "heterogeneous tissue" on LGE. Once such CMR limitations are addressed, we will know where on the LGE image to focus our attention. However, as evident from the outstanding report by Perez-David and colleagues, solving the VT puzzle will still need an electrophysiolgist who is adept at maneuvers to distinguish critical sites from bystanders.

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