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 intracardiac recordings made by Hecht (1), and demonstration of catheter-based cardiac stimulation by Furman and Robinson (2). Later, Wellens et al. (3) demonstrated that programmed electrical stimulation could reliably initiate and terminate ventricular tachycardia (VT) circuits in susceptible individuals. 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 et al. (4–6) demonstrated that intracardiac recordings from heterogeneous tissue identified by CMR. They have shown that sites that electrophysiology maneuvers from bystander areas adjacent to the VT circuit corresponded to a critical isthmus for VT. These results are remarkable. Perez-David et al. (25) have demonstrated the biologically plausible but up to now unproven connection regarding direct electrophysiology measures and maneuvers from heterogeneous sites identified by CMR (24).

In this issue of the Journal, Perez-David et al. (25) present the results of a study that compares LGE features using a 1.5-T magnetic resonance scanner, in 18 patients with sustained monomorphic VT, to 18 matched control subjects. The investigators chose to use Yan et al.’s (20) method for defining the infarct core and heterogeneous tissue as regions with signal intensity threshold >3 and between 2 and 3 standard deviations higher than remote normal myocardium, respectively. 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 1 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 investigators then created 3-dimensional color-coded shells displaying the subendocardial signal-intensity distribution. Continuous corridors of diseased but conducting tissue on endocardial voltage maps 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 et al. (25) 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 inducibility 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 marker of risk in a larger population may not offer adequate predictability 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 atrioventricular 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 is evident from the outstanding report by Perez-David et al. (25), solving the VT puzzle will still need an electrophysiologist who is adept at maneuvers to distinguish critical sites from bystanders.

REFERENCES


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