Intrascar ventricular tachycardia: New concept of scar-reentrant ventricular tachycardia

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Introduction
The circuit of scar-reentrant ventricular tachycardia (VT) in the context of structural heart disease usually consists of 2 components, an isthmus within a low-voltage scar and an outer loop of healthy myocardium, that are connected by an entrance and exit in the scar border zone.1 Because slow conduction in the critical isthmus plays a crucial role in maintaining the reentry, VT ablation usually targets the area from the critical isthmus to the exit identified by voltage and entrainment mapping.2 We, however, encountered a VT case in which the entire reentrant circuit was confined to the scar area remote from the exit.

Case report
A 75-year-old woman with ischemic cardiomyopathy managed by a cardiac resynchronization therapy defibrillator was referred to our institution for catheter ablation of a recurrent VT storm. She had previously received endocardial ablation for a VT originating from an extensive left ventricular (LV) apical scar. The QRS complex morphology of this hemodynamically tolerant VT exhibited a left bundle branch block configuration with a superior axis with a tachycardia cycle length (TCL) of 430 ms. The right ventricular (RV) and LV activation maps were created with a multipole, multispline mapping catheter (2-6-2 mm interelectrode spacing; PentaRay NAV; Biosense Webster, Diamond Bar, CA) and guided by the CARTO3 mapping system (Biosense Webster). The LV activation map revealed a relatively small reentrant circuit accounting for 97% of the TCL at the anterior aspect of the LV apex (Figure 1). The activation at the RV apex was also early and preceded the QRS complex by 15 ms (Figure 1). Entrainment pacing from the RV apex revealed the earliest activation exhibiting manifest but nearly concealed entrainment with a short stimulus-QRS interval (≤30% of tachycardia cycle length [TCL]) and a considerably long postpacing interval (PPI); and (2) all sites with a PPI identical to the TCL exhibit a long stimulus-QRS interval (>70% of TCL).

When we encounter the intrascar VT, we need to change the ablation target from the exit site to the critical isthmus exhibiting a PPI identical to the TCL despite a long stimulus-QRS interval.

KEYWORDS
Activation mapping; Catheter ablation; Entrainment mapping; Ischemic cardiomyopathy; Ventricular tachycardia

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LV with the exit site at the apical septum remote from the VT circuit (Figure 2A). A radiofrequency energy application to the LV entrainment pacing site immediately terminated the VT (Figure 2F). The pace maps after the VT terminated, utilizing an output of 10 mA within the scar area, revealed that the paced-QRS morphologies relatively matched the VT morphology at the successful ablation site to the RV earliest site accompanied by a shortening of the stimulus-QRS interval (Figure 3). After extensive substrate modification guided by the pace map, further VT was rendered noninducible.

Discussion
The VT circuit is generally considered to consist of an isthmus within the scar and a healthy outer loop. However, an entire VT reentrant circuit confined to the scar area (defined as “intrascar VT”) can be possible. In that situation, a careful interpretation of the electrograms and observation during entrainment pacing would be required. In our case, the findings supporting the diagnosis of the intrascar VT were as follows: (1) the activation map revealed the VT circuit was confined to the LV apical scar, which was confirmed by an estimated PPI identical to the TCL and VT termination by a radiofrequency application; (2) the RV pacing site conventionally appeared to be close to the VT exit on the basis of nearly obtaining concealed entrainment and a short stimulus-QRS interval, but the PPI was considerably longer than the TCL; and (3) a morphology-matched pace map and stimulus-QRS interval suggested that the critical isthmus extended from the VT termination site in the LV to the exit site on the apical septum. Given these findings, the estimated VT circuit was confined to the LV scar area, but the exit site to healthy myocardium was located on the apical septum remote from the circuit through the scar area. Although the

![Figure 1](image-url) The right ventricle (RV) and left ventricle (LV) activation maps of the ventricular tachycardia. The circuit was located in the anterior aspect of the LV apex, where it was confined to an extensive scar area. The fractionated potentials on the circuit account for 97% of the tachycardia cycle length. The slowest conduction zone is found within the slow or nonconducting zones, colored blue to red. The activation of the RV apex is early and precedes the QRS complex by 15 ms. EGM = electrogram.
VT was terminated by a radiofrequency energy application at the LV pacing site, that site left open the possibility of being a part of the inner loop because the local electrogram was not visible, which hindered the PPI measurement. The VT termination might have been due to conductive heating of the critical isthmus adjacent to the inner loop where the ablation was performed. Nonetheless, the VT circuit seemed to be confined to the scar.

VT ablation generally targets the area from the critical isthmus to the exit, which is determined by (1) concealed entrainment with (2) a short stimulus-QRS and (3) PPI identical to the TCL. Nonetheless, in our case, mapping failed to locate the exit site, because no sites fulfilled all 3 of those entrainment pacing–related criteria. In this situation, we needed to target the critical isthmus site mimicking the entrance, which was not initially targeted. The long stimulus-QRS interval did not necessarily mean it was the entrance site of the VT. High-density 3D activation mapping would help to identify the VT circuit.

Based on the discussion, we proposed 2 criteria for the diagnosis of intrascar VT: (1) the presumed exit site, where concealed entrainment and a short stimulus-QRS interval (<30% of TCL) are obtained, exhibits a considerably long PPI; and (2) all sites with a PPI identical to the TCL exhibit a long stimulus-QRS interval (>70% of TCL). When we encounter a case fulfilling both of those criteria, we need to change the ablation target from the exit site to the critical isthmus exhibiting a PPI identical to the TCL despite a long stimulus-QRS interval because the exit site is remote from the reentry circuit.

Conclusions
The VT case, in which the entire reentrant circuit was confined to the scar area and the exit was located at the remote site from the circuit, would add new insight into VT mapping and ablation.
Figure 3  The bipolar voltage maps during sinus rhythm and QRS morphologies observed in the pace map. The purple area indicates a high bipolar voltage area $\geq 1.5$ mV. The scar area, colored red, shows a bipolar voltage of $<0.5$ mV, and the scar border zone areas, colored orange, yellow, green, and blue, have a voltage of 0.5–1.5 mV. The pace maps utilizing an output of 10 mA within the scar area revealed that the paced-QRS morphologies relatively matched the ventricular tachycardia (VT) morphology at the successful ablation site to the right ventricle earliest site accompanied by a shortening of the stimulus-QRS interval.

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