Multiple exit sites identification by pace mapping with a grid catheter: Which bipolar pairs are in the critical ventricular tachycardia isthmus?



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Introduction

A recent study reported the effectiveness of scar-related ventricular tachycardia (VT) ablation guided by functional substrates via high-density mapping using multielectrode catheters.¹ In this study, VT induction was followed by strategic positioning of the catheter on the functional substrate with a pace map match for the target VT morphology or multiple exit sites (MES).¹ These responses to pace mapping are potential surrogates for VT isthmuses of VT reentry,^{2,3} so it can facilitate the rapid identification of the critical sites of VT circuits after VT induction. However, pace mapping cannot differentiate critical isthmuses from adjacent bystanders for VT reentry.^{2,3}

Case report

A 77-year-old man with ischemic cardiomyopathy underwent catheter ablation of VT. During atrial pacing, functional substrate mapping with a grid catheter (Advisor HD Grid mapping catheter; Abbott, St. Paul, MN) revealed a slow conduction region in the midposterolateral left ventricle (Figure 1A). The grid catheter positioned in this region had multiple late potentials (Figure 1B and 1C). Pace mapping from the proximal bipolar pair of spline C generated multiple QRS morphologies with different stimulus latencies (Figure 1D), and one of the pace maps matched the target VT morphology. Which bipolar pairs of the catheter are positioned in the critical isthmus of VT? Where is an additional pace map site before VT induction?

Discussion

A grid catheter allows simultaneous recording of multiple bipolar electrograms during each beat, even when overdrive pacing is delivered through a bipolar pair of the catheter.⁴

KEYWORDS Catheter ablation; Grid catheter; Isthmus; Pace mapping; Ventricular tachycardia

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KEY TEACHING POINTS

- A grid catheter allows simultaneous recording of multiple bipolar electrograms during each beat, even when pacing is delivered through a bipolar pair of the catheter.
- When attempting to identify the critical sites of reentrant ventricular tachycardia (VT) circuits, the use of a grid catheter can allow the visualization of local ventricular activation around a pacing site during pace mapping and pacing-induced termination of VT even with nonglobal ventricular capture.
- Analyzing the local activation sequences during pace mapping at a VT substrate with multiple exit sites can provide a complementary approach to explore the critical VT isthmuses.

Therefore, pace mapping with a grid catheter can provide information on activation sequences around the pacing site. In this case, pacing demonstrated a substrate with MES, and according to the pace maps, changes in activation sequences preceding the paced QRS complexes are observed. When an isthmus of VT reentry involves a substrate with MES (left panel in Figure 2A), pace mapping at an MES site attached to conduction pathways connecting each exit site can induce a shift in the direction of stimulated wavefront propagation through these pathways (Figure 2B). Therefore, the activation sequences should be different among the paced QRS morphologies. However, during the shift, the wavefront may propagate through those pathways to the other exit (and entrance) sites, and there can be no change in the activation sequences of the pathways preceding the paced QRS onset (Figure 2C). In fact, the same timing and morphology of local electrograms preceding the paced ORS onset were observed with the proximal bipolar pair of spline B (arrowheads in Figure 2G). On the other hand, local electrograms on all bipolar pairs of spline A preceded the

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Figure 1 A: Bipolar voltage and late activation maps of the left ventricle. **B**: Fluoroscopic images exhibiting the grid catheter positioned on the slow conduction region. **C**: Late potentials in this region. **D**: Pace maps obtained by pacing from the proximal bipolar pair of spline C at 10 mA/1 ms. The pace map with the shortest stimulus latency matched the target ventricular tachycardia. X1-X2, X2-X3, and X3-X4 represent the distal, middle, and proximal bipolar pairs of the relevant spline of the grid catheter, respectively. CS = coronary sinus; HB = His bundle; LAO = left anterior oblique; RAO = right anterior oblique; S = pacing stimulus.

paced QRS onset only when generating the pace map with the longest stimulus latency (Figure 2G). This finding indicates that these bipolar pairs can be in a distal region of the

pathway connecting the exit site of this pace map (areas with oblique lines in Figure 2C). Moreover, the large region covered by spline A suggests that the wavefront propagation



Figure 2 Schematic of a ventricular tachycardia (VT) substrate with multiple exit sites (MES) showing the following: **A:** the wavefront propagation during VT; **B–F:** pace mapping at an MES site. **G:** the pace maps at the MES site. The black areas present scar. The double lines indicate functional conduction block. In panel A, showing VT circuits without (left panel) and with an adjacent bystander connecting an MES site (right panel), the wave lines indicate tachycardia wavefront propagation within the scar. In panels B–F, the solid and dashed wave lines indicate stimulated wavefront propagation before and after the paced QRS onset, respectively. In panels C and D, a shift in the exit site requires functional conduction block at least within the pathway connecting the exit site of the pace map with the shortest stimulus latency. In gray areas, the same activation sequences precede any paced QRS complex during the shift. In areas with oblique lines, the local electrograms precede or follow the paced QRS onset during the shift. In panel G, the arrowheads indicate the local electrograms with the same timing and morphology.



Figure 3 A: Pace map obtained by pacing from the proximal bipolar pair of spline B. B: Ventricular tachycardia termination with nonglobal ventricular capture induced by pacing from that of spline B. The arrowheads and asterisks indicate mid-diastolic potentials and local ventricular capture, respectively.

is likely to be limited to a proximal region of that pathway before generating the pace map with the shortest stimulus latency (Figure 2D). Therefore, the critical isthmus is likely to be in a substrate where the same activation sequences precede any paced QRS complex during MES site pacing (gray areas in Figure 2D). However, when an MES site connects an adjacent bystander (right panel in Figure 2A), the stimulated wavefront can propagate to a distal region of the bystander, whose activation sequences can be identical during the shift.

Pacing from the proximal bipolar pair of spline B resulted in a pace map match for the target VT morphology, and the stimulus latency is shorter than that during MES site pacing (Figure 3A). Therefore, the second pace map site is unlikely to be in an adjacent bystander, and a stimulus latency of over 40 ms suggests that this pacing site is likely to be in the critical isthmus.²

The target VT was easily induced with burst ventricular pacing from the MES site. During VT, the proximal bipolar pair of spline C and spline B had early and mid-diastolic potentials, respectively (Figure 3B). Overdrive pacing from that of spline B resulted in VT termination with nonglobal ventricular capture (Figure 3B), indicating that the critical isthmus involved the second pace map site.⁵ During this type of VT termination, evidence of local ventricular capture is rarely observed with an ablation catheter.⁵ However, the recording of activation sequences with a grid catheter showed local ventricular capture on the distal bipolar pair of spline B just after the first stimulus of overdrive pacing (Figure 3B). The target VT was successfully eliminated by radiofrequency ablation at the critical site. After additional ablation by targeting the late potentials around this site, any VT was rendered noninducible.

According to the pace maps at the MES site, small differences in the local electrograms were noted on spline A. In Figure 2G, these electrograms identical in the pace maps with the longest and longer stimulus latencies are slightly different from those following the onset of the paced QRS morphology with the shortest one. Therefore, MES site pacing may induce alternans in conduction properties of each pathway (Figure 2E), which causes the shift in the exit site. However, the critical site of the VT exhibited the same conduction property even during the shift (thick wave lines in Figure 2F). In the absence of functional conduction block within the pathways, the stimulated wavefront might propagate away from multiple exit sites during MES site pacing. However, the shift results in a distinct difference in the paced QRS morphology. Thus, in this setting, the wavefront propagating from one exit site may well depolarize most of the myocardium but that from the others might depolarize an insufficient mass of it to alter the QRS morphology in each pace map.

Conclusion

This case demonstrates that the use of a grid catheter allows the visualization of local ventricular activation around a pacing

site during pace mapping and pacing-induced termination of postinfarction VT. It also highlights that analysis of the activation sequences preceding paced QRS complexes during pace mapping at an MES site can provide a complementary approach to explore the critical VT isthmuses.

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