Demonstrating the deceleration zone associated with epicardial conduction using isochronal late activation mapping during right ventricular apex pacing

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Introduction

Substrate-based approaches for ventricular tachycardia (VT) ablation aim to ablate the channels that serve as the substrate for VT propagation without the requirement to map them during VT. Isochronal latest activation mapping (ILAM)[1,2] is a method by which the chamber of interest is mapped using a consistent wavefront and is useful for detecting the VT's critical zone. A previous study has shown that conduction-slowing areas such as deceleration zones (DZs) during sinus rhythm identified by ILAM are related to the exit site of the VT isthmus.[3] However, in several cases, DZs are not observed during sinus rhythm. In this case, the isochronal line and DZ that were not observed during sinus rhythm were unmasked during right ventricular apex (RVA) pacing and were useful for detecting the VT circuit. Moreover, recently, it has been reported in scar-related VTs that the VT circuit frequently exhibits a three-dimensional (3D) activation pattern. Here, we report a case in which the VT circuit was considered to have a 3D structure from the activation mapping performed on the endocardial surface using high-resolution mapping, called *omnipolar mapping* (Advisor HD Grid, Abbott, Green Oaks, IL).

Case report

A 75-year-old man with an implantable cardioverter defibrillator (ICD) for VT associated with prior myocardial infarction presented to the emergency department with shocks from his ICD. The patient was immediately hospitalized, and because of his resistance to drug treatment and frequent ICD shocks, we decided to perform catheter ablation. Informed consent was obtained from the patient.

KEY TEACHING POINTS

- Isochronal lines and deceleration zones, which were not visualized during sinus rhythm, were unmasked on isochronal latest activation mapping (ILAM) during right ventricular apex (RVA) pacing.
- The line of conduction block visualized on the ILAM during RVA pacing was colocalized with the lateral isthmus boundaries and was associated with the excitation propagation in the ventricular tachycardia (VT) isthmus.
- For the identification of reentry circuits with a three-dimensional structure, recording the activation gap during VT using omnipolar technology led to the elucidation of the mechanism of the VT origin.

A 12-lead electrocardiogram (ECG) revealed left bundle branch block during sinus rhythm. A transthoracic echocardiogram showed akinesis of the inferior-septal left ventricle (LV) wall. An ECG recorded by the ICD during a shock episode revealed a sustained monomorphic VT in which the tachycardia cycle length was 320–330 ms.

A voltage map of the LV during sinus rhythm was created with an HD Grid catheter (Figure 1A). When the low voltage area was set at $<$1.5 mV and scar at $<$0.3 mV, we observed a huge low voltage area from the inferior wall to the septal wall. On the ILAM during sinus rhythm, we observed a wavefront traveling from the basal region to the apex with a broad wavefront pattern (Figure 1B). In contrast, on the ILAM during RVA pacing, we found an isochronal line on the septal wall and several DZs that were not observed during sinus rhythm (Figure 1C). Furthermore, the intracardiac ECG in the DZs during RVA pacing exhibited local potentials that clearly differed from those during sinus rhythm. Fractionated

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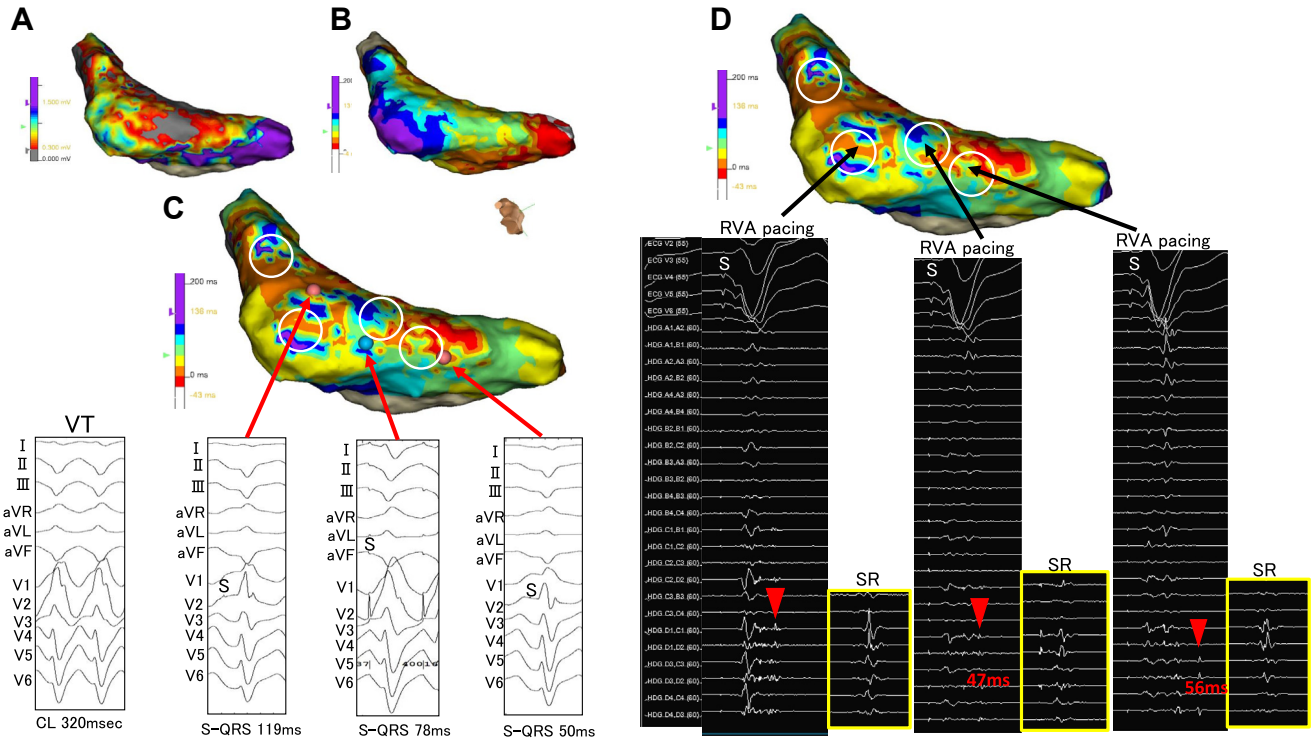

Figure 1 **A:** Voltage map of the left ventricle during sinus rhythm (right anterior oblique). A huge low voltage area from the inferior wall to the septal wall was observed. **B:** Isochronal latest activation mapping (ILAM) during sinus rhythm. The broad wavefront pattern from the basal region to the apex is shown, and there was no deceleration zone (DZ). **C:** ILAM during right ventricular apex (RVA) pacing. Several DZs became apparent in the area from the inferior wall to the septal wall during RVA pacing (white frame). The *pink and blue tags* indicate the good pace map sites. Moving from the basal region to the apex, the S-QRS becomes shorter. **D:** Intracardiac electrocardiogram in the DZ during right ventricular apex pacing and sinus rhythm. S-QRS = stimulus-QRS; SR = sinus rhythm; VT = ventricular tachycardia.

electrograms were markedly observed, and then several delayed potentials and split potentials[1] with an isoelectric segment more than 20 ms became apparent, suggesting the existence of a conduction block line (Figure 1D).

Programmed ventricular stimulation was performed at the RVA and right ventricle (RV) outflow tract with 1–3 extrastimuli until refractoriness and/or 200 ms after 8 basic stimuli with cycle lengths of 600 and 400 ms. Incremental pacing with a minimal cycle length of 240 ms was also performed as each ventricular site. These stimulation protocols were used before and after ablation in an attempt to induce VT. The monomorphic VT shown in Figure 1C, in which the QRS morphology exhibited right bundle branch block and a superior axis with a tachycardia cycle length of 320 ms, was induced reproducibly with double extrastimuli from the RVA. The VT was identified to be the clinical VT because of the cycle length. Since the VT terminated or changed into a polymorphic VT, it was difficult to perform entrainment pacing. The patient underwent pace mapping for a comparison with the 12-lead ECG of the clinical VT, resulting in satisfactorily matched pace-maps observed near the DZs during RVA pacing. The closer we moved from the basal region to the apex, the shorter the S-QRS became, and the former was 119 ms and the latter 50 ms (Figure 1C).

On the propagation map during VT, it was observed that the wavefront which broke out from the left ventricular apical inferior wall area with a conduction delay during RVA pacing, circled around the inferior wall with an activation gap at the distal isthmus, and it was also found that the wavefront passed through part of the line of block (LOB) formed by multiple split potentials (>20 ms), which made up the isthmus boundaries (Figure 2A). When we observed the local potentials at the activation gap site using omnipolar mapping, we found that along the same spline, split potentials on the proximal side and long-lasting fractionated presystolic potentials on the distal side were simultaneously recorded across the gap site (Figure 2B). The latter potentials were estimated to be far-field potentials, and a continuous propagation map that filled the tachycardia cycle length was demonstrated when the potentials were annotated manually (Supplemental Video). We ablated the endocardial site using an anatomic approach. An irrigated-tip catheter (Tacti Cath; Abbott) was inserted into the left ventricle using a transaortic approach. Next, we applied 35–45 W of radiofrequency energy until LSI 5.0 to the regions exhibiting the earliest potentials and the gap site, and VTs were no longer inducible. This patient has been free of VT episodes during a follow-up period of 2 years.

Discussion

In this case, the isochronal line and DZ that were not observed during sinus rhythm were unmasked during RVA

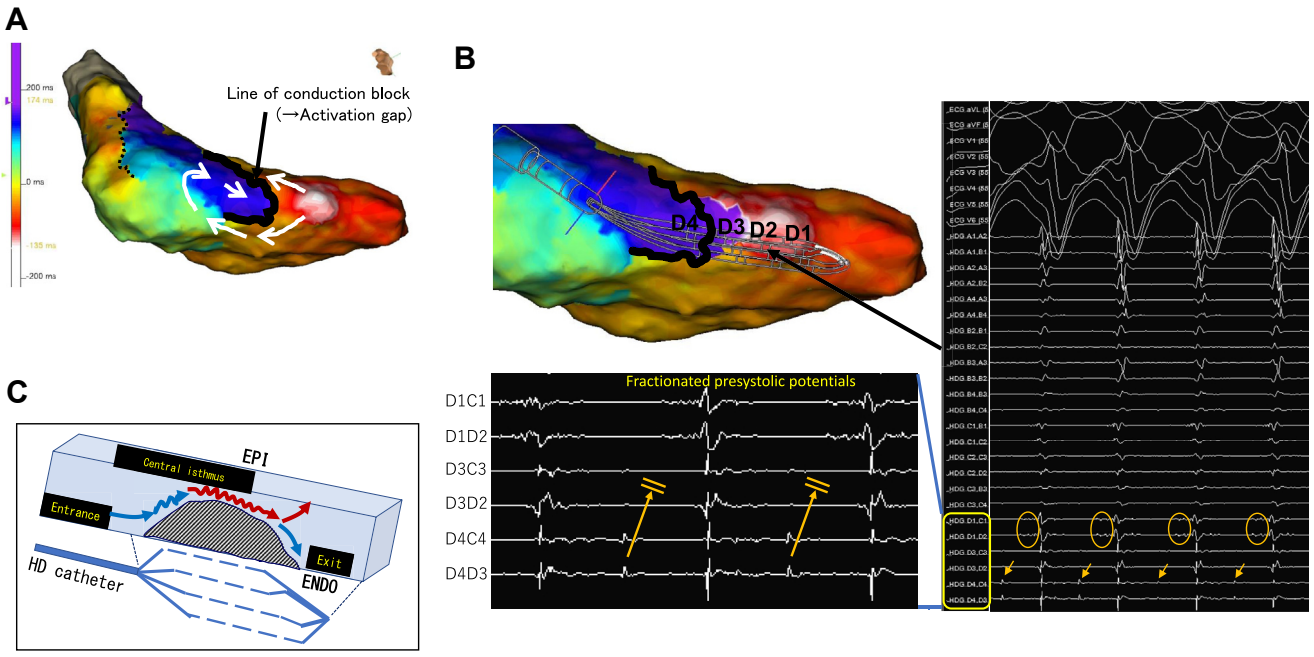

Figure 2 **A:** Propagation map during ventricular tachycardia (VT). A wavefront breaking out from the left ventricular apical inferior wall area with a conduction delay during right ventricular apex (RVA) pacing circled around the inferior wall with an activation gap in the distal isthmus. The *black line* indicates conduction block; the *white arrow* indicates propagating excitation. **B:** Omnipolar mapping at the activation gap site during VT. On the same D spline, a split potential on the proximal side and long-lasting fractionated presystolic potentials on the distal side were simultaneously recorded across the gap site. **C:** Isthmus activation during 3-dimensional VT reentry. The mechanism by which the gap occurred on the endocardial side was presumed to be that part of the slow conduction area (central isthmus) that mediated the epicardial conduction. The *arrows* indicate propagating excitation.

pacing at a site near the exit site of the VT reentry circuit. Moreover, the VT circuit was considered to have a 3D structure because the presence of an activation gap recorded by the coaxial spline electrodes was observed on the VT activation map recorded from the endocardial surface using omnipolar mapping.

Activation and entrainment mapping of VT is the gold standard for identifying critical sites for ablation of VT[4]; however, those strategies are not useful for unmappable VTs, such as hemodynamically unstable VTs and nonsustain VTs. In contrast, ILAM is a method by which the chamber of interest is mapped using a consistent wavefront. LAT maps are then divided into isochrones with the same unit of time, with areas of isochronal crowding representing slow conduction. In a report by Aziz et al.,[1] 8 isochrones were used with 3 isochrones within a 1-cm area used to define a DZ. Using this method to identify and ablate critical zones within the substrate, 70% of patients had no VT recurrences at 12 ± 10 months of follow-up. Conduction slowing areas, such as DZs during sinus rhythm identified with ILAMs, are related to the exit portion of VT isthmuses[3]; however, in several cases, DZs are not able to be observed during sinus rhythm. In this case, the isochronal lines and DZs that were not observed during sinus rhythm were visualized during RVA pacing. Diseased myocardial tissue may exhibit nonuniform anisotropy, with slow conduction only when the excitation propagates from a specific wavefront. Furthermore, it has also been reported that since the direction of the wavefront in the left ventricular myocardium affects the site and degree of the conduction delay, mapping during RV and LV pacing in addition to sinus rhythm visualizes the conduction delay sites and critical isthmus sites, increasing the accuracy of the substrate mapping.[5] Animal research has shown that the mechanism of anisotropy in pathologic myocardial tissue is in the infarcted region, gap junctions tend to degenerate, and the conduction difference between the long and short axes of myocardial fibers increases, which is likely to cause anisotropy.[6] Furthermore, it has been reported that an impedance mismatch that occurs because of thinning of the remaining myocardium in the infarct border region might cause unidirectional conduction block and reentry.[6]

Figure 3 shows the propagation excitation during sinus rhythm and RVA pacing in this case. During RVA pacing, the wavefront traveling toward the isthmus during the VT traveled in a different direction than that during sinus rhythm (Figure 3B); therefore, its involvement increased, leading to a decrease in the conduction velocity and prolongation of excitation time. It was estimated that this resulted in the formation of a LOB by the lateral isthmus boundary along with the DZ and functional block (Figure 3A and 3C). In other words, DZs and LOB that were not visualized during sinus rhythm were unmasked during pacing at a site near the exit of the VT reentry circuit. Furthermore, stimulation from that site caused conduction block within the central isthmus, forming a reentrant circuit and inducing VT (Figure 3D). Nishimura et al.[7] reported that such an existence explains the relationship between the LOB and VT lateral isthmus boundaries in a recent report with multiple cases. In short, it has been shown that a concealed LOB can be visualized with a high probability not during sinus rhythm, but during pacing

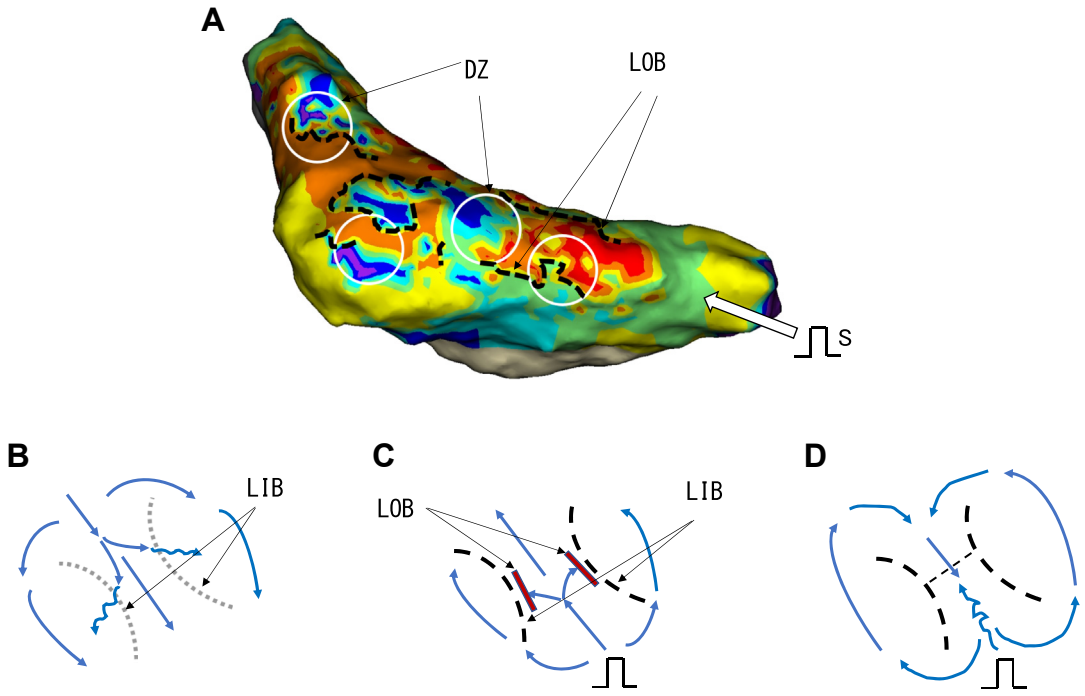

Figure 3 **A:** Isochronal latest activation mapping (ILAM) during right ventricular apex pacing (RAO). Several deceleration zones (DZs) and lines of conduction block (LOBs) were observed. The *white frame* indicates the DZ; the *black dotted line* indicates the LOB. **B:** Propagation excitation during sinus rhythm. The main wavefront conducted longitudinally along the lateral isthmus boundaries (LIBs), and some wavefronts propagated across the LIB with a conduction delay. The *blue arrow* indicates propagating excitation. **C:** Propagation excitation during right ventricle apex pacing. It was estimated that an LOB accompanied by a DZ and functional block was formed because the wavefront moving toward the isthmus during the ventricular tachycardia (VT) traveled in a different direction than that during SR. *Black dotted lines* indicate LIBs; *red lines* indicate LOBs. **D:** VT induction and excitation propagation during right ventricular apex (RVA) stimulation. Stimulation from the RVA caused conduction block within the central isthmus, leading to the induction of a reentrant VT.

from other sites, and that colocalizes with the lateral isthmus boundaries in two-dimensional activation patterns or with the depth boundary during a 3D VT.

Recent studies have reported that the majority of VTs exhibits a 3D reentry circuit. Tung et al.[8] reported that when the VT circuit has a reentrant isthmus confined to below the surface of the epicardium, incomplete reentry is seen on the endocardium with a breakout and curved propagation along the outer loops with an activation gap at the distal isthmus. In this case, as previously reported by Tung et al.,[8] in the recording from the endocardial side, an activation gap with a conduction block was observed, and at the exit site, centrifugal activation was observed on the endocardial side (Figure 2B). The fractionated presystolic potentials between the conduction block site and the earliest excitation site during VT were suggested to be far-field potentials from the epicardial or mid-myocardium conduction. Since no ventricular tachycardias were any longer inducible after ablation on the endocardial side using an anatomic approach, the mechanism by which the gap occurred was presumed to be that a part of the slow conduction area (central isthmus) mediated the epicardial conduction, and the VT was estimated to be a reentrant VT that activated a site that was the isthmus (Figure 2C).

Substrate-based VT ablation now includes modalities such as voltage-based scar determination, identification of functional substrates via areas of fractionation, deceleration zones, and decrement-evoked potentials, which correlate highly with critical isthmus sites for VTs.[9] For the evaluation of a substrate-based mechanism, the relative utility of various substrate mapping techniques for the identification of the critical sites for VT ablation using the omnipolar technology, has shown that together with other electroanatomic modalities including ILAM, fractionation maps provide a useful adjunct to substrate mapping.[10] A recent study has shown that a high-density, whole-chamber double-extrastimuli protocol enhance the identification of VT isthmus on the basis of DZ and late potential assessment and enables improved identification of ablation targets.[11] In this case, for the identification of a reentry circuit with a 3D structure, it was useful to reveal the DZ by ILAM using omnipolar mapping during continuous RVA pacing without the application of extrastimuli. Furthermore, recording the activation gap during the VT led to the elucidation of the mechanism of the VT origin.

Conclusion

Isochronal lines and DZs visualized during pacing at a site near the exit site of the VT reentrant circuit were useful for detecting the VT circuit. Moreover, the VT circuit was estimated to have a 3D structure because an activation gap recorded by the coaxial spline electrodes was identified on the VT activation map recorded from the endocardial surface using omnipolar mapping.

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References

1. Aziz Z, Shatz D, Raiman M, et al. Targeted ablation of ventricular tachycardia guided by wavefront discontinuities during sinus rhythm: a new functional substrate mapping strategy. Circulation 2019;140:1383–1397.
2. Irie T, Yu R, Bradfield JS, et al. Relationship between sinus rhythm late activation zone and critical sites for scar-related ventricular tachycardia: systematic analysis of isochronal late activation mapping. Circ Arrhythm Electrophysiol 2015; 8:390–399.
3. Ueda A, Soejima K, Fukuda R, et al. Conduction slowing area during sinus rhythm harbors ventricular tachycardia isthmus. J Cardiovasc Electrophysiol 2020;31:440–449.
4. Stevenson WG, Friedman PL, Sager PT, et al. Exploring postinfarction reentrant ventricular tachycardia with entrainment mapping. J Am Coll Cardiol 1997; 29:1180–1189.
5. Anter E, Neuzil P, Reddy VY, et al. Ablation of reentry—vulnerable zones determined by left ventricular activation from multiple directions: a novel approach for ventricular tachycardia ablation: a multicenter study (PHYSIO-VT). Circ Arrhythm Electrophysiol 2020;13:e008625.
6. Ciaccio EJ, Anter E, Coromilas J, et al. Structure and function of the ventricular tachycardia isthmus. Heart Rhythm 2022;19:137–153.
7. Nishimura T, Shatz N, Weiss JP, et al. Identification of human ventricular tachycardia demarcated by fixed lines of conduction block in a 3-dimensional hyperboloid circuit. Circulation 2023;148:1354–1367.
8. Tung R, Raiman M, Liao H, et al. Simultaneous endocardial and epicardial delineation of 3D reentrant ventricular tachycardia. J Am Coll Cardiol 2020; 75:884–897.
9. Aziz Z, Tung R. Novel mapping strategies for ventricular tachycardia ablation. Curr Treat Options Cardiovasc Med 2018;20:34.
10. Khan H, Bonvissuto MR, Rosinski E, et al. Comparison of combined substrate-based mapping techniques to identify critical sites for ventricular tachycardia ablation. Heart Rhythm 2023;20:808–814.
11. Guichard JB, Regany-Closa M, Vazquez-Calvo S, et al. Substrate mapping for ventricular tachycardia ablation through high-density whole-chamber double extra stimuli: the S3 protocol. JACC Clin Electrophysiol 2024;10:1534–1547.