Successful catheter ablation for verapamil-sensitive idiopathic left ventricular tachycardia guided by dual post–QRS wave P1 potentials after catheter-induced mechanical block

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Introduction

Verapamil-sensitive left posterior fascicular ventricular tachycardia (LPF-VT) was first described in 1979.[1] This tachycardia is characterized by a right bundle branch block morphology with left and superior axis deviation and originates in the region of the left posterior fascicle of the left bundle. Previous studies have also shown that the tachycardia is often sensitive to verapamil.[2,3] To determine the optimal ablation site for LPF-VT during ventricular tachycardia (VT), it is important to locate the diastolic potential (P1). If visible, the optimal ablation site is where both a P1 and a presystolic potential (P2) can be recorded simultaneously, and the interval between them is shortest. If P1 cannot be visualized, the optimal ablation site is where the earliest P2 during VT can be recorded.[4]

Catheter ablation of LPF-VT is highly successful, but there is a major limitation to the use of this strategy. The VT must be inducible to allow for mapping and searching for the optimal ablation site. If catheter-induced mechanical block, known as the "bump" phenomenon, occurs, it may be impossible to induce the tachycardia and difficult to determine the ablation site.

To the best of our knowledge, the case presented here is the first to demonstrate a novel ablation strategy for verapamil-sensitive LPF-VT in which P1 potentials appearing after the QRS wave are used for guidance after the bump phenomenon has occurred.

KEY TEACHING POINTS

- Appearance of a late P1 potential during sinus rhythm may indicate occurrence of catheter-induced mechanical block during catheter ablation for idiopathic left ventricular tachycardia.
- If catheter-induced mechanical block is suspected, the intracardiac electrogram and 3-dimensional mapping system used during the procedure should be retrospectively reviewed to analyze the electrical potentials and positional information at the moment of the bump phenomenon.
- Appearance of 2 types of post–QRS wave P1 potentials during sinus rhythm might provide additional guidance for identifying the optimal ablation site after the bump phenomenon occurs.

Case report

A 19-year-old woman visited our hospital complaining of frequent palpitations. An electrocardiogram revealed wide QRS tachycardia (cycle length, 430 ms), the morphology of which showed a right bundle branch block configuration and a superior axis (Figure 1). Intravenous administration of verapamil 2.5 mg terminated the tachycardia with deceleration of heart rate. She had no history of syncope or family history of sudden cardiac death, and the echocardiogram indicated a structurally normal heart without valvular disease. The tachycardia was thought to be LPF-VT. After informed written consent was obtained, we performed catheter ablation for LPF-VT. For the electrophysiologic study, we placed a 5F quadripolar electrode catheter at the high right atrium (HRA) and a 14-polar electrode catheter from the right

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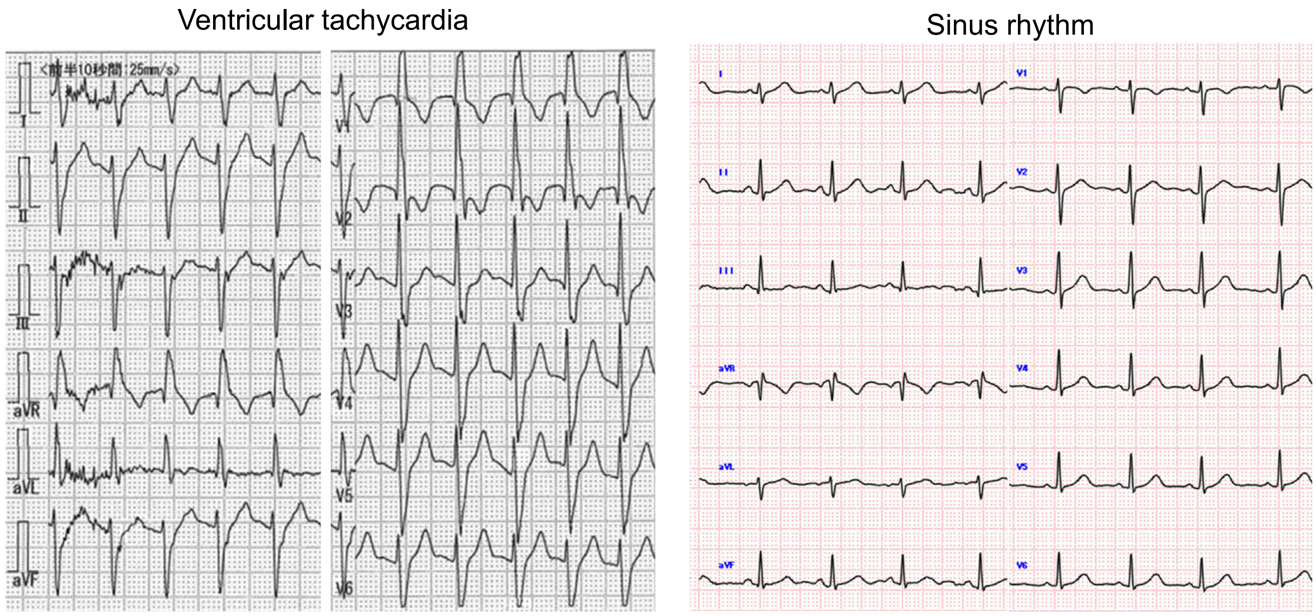

Figure 1 Twelve-lead electrocardiogram (left, ventricular tachycardia; right, sinus rhythm). HIS = bundle of His; HRA = high right atrium; LAO, left anterior oblique; RV = right ventricle.

ventricular apex (RVA) to the His bundle area via the right femoral vein. The CARTO3 electroanatomic mapping system (Biosense Webster, Diamond Bar, CA) was used during the procedure. The atrio-Hisian and His-ventricular intervals were 84 ms and 36 ms, respectively. The tachycardia occurred spontaneously and incessantly, and we administered 20 mg of adenosine intravenously to distinguish between VT and supraventricular tachycardia. After the infusion, atrioventricular dissociation occurred without termination of the tachycardia. During the VT, entrainment pacing was performed from the RVA; however, the VT terminated and could not be evaluated. Considering that this patient had no structural heart disease and her tachycardia showed characteristics of verapamil sensitivity and right bundle branch block with left axis deviation, we diagnosed verapamil-sensitive LPF-VT.

Therefore, we attempted to map the posterior septal wall of the left ventricle (LV), manipulating the steerable decapolar catheter with 2-8-2-mm interelectrode spacing (DECANAV; Biosense Webster) via a retrograde aortic approach.

Immediately after the DECANAV catheter was placed in the LV longitudinally, LPF-VT was no longer inducible, even in response to an isoproterenol infusion and a programmed stimulus from both the HRA and RVA. Specifically, our VT stimulation protocol involved continuous stimulation (in both HRA and RVA, ranging from 600 ms to 240 ms), single premature extrastimulation (in both HRA and RVA, S1 at 600 ms or 450 ms, S2 from 400 ms to the effective refractory period [ERP]), and double premature extrastimulations (in both HRA and RVA, S1 at 600 ms or 450 ms, S2 from 300 ms to ERP, S3 from 300 ms to ERP). Under the administration of isoproterenol, the same stimulation protocol was used in an attempt to induce VT. Based on these observations, we suspected catheter-induced mechanical block (ie, the bump phenomenon).

We looked retrospectively at the position of the DECANAV catheter when the bump phenomenon occurred. On the intracardiac electrogram, the P1 potential was observed before the P2 potential during a premature ventricular contraction (PVC), which might have been the initiation of the LPF-VT (shown in the first beat on Figure 2A). The next beat was a catheter-induced PVC (shown in the second beat of Figure 2A), and the subsequent PVC had the same morphology as the LPF-VT (shown in the third beat of Figure 2A).

Immediately after the bump phenomenon, a late-appearing P1 potential, which is one of the indicators of successful catheter ablation for verapamil-sensitive LPF-VT, was recorded during sinus rhythm by the DECANAV catheter and was reproducible (Figure 2A). We removed the DECANAV catheter from the LV for 30 minutes. The bump phenomenon still occurred after 30 minutes of follow-up; the late-appearing P1 potential was still observed and we could not induce LPF-VT even by an isoproterenol infusion and a programmed stimulus from both the HRA and RVA. Atrial programmed pacing demonstrated decremental conduction in the late-appearing P1 potential (Figure 2B).

Therefore, we decided to perform catheter ablation using an anatomical approach and guided by the late-appearing P1 potential during sinus rhythm. Interestingly, 2 types of isolated P1 potentials were observed after the His potential, appearing at 110–120 ms and 340–350 ms, respectively (Figure 2C); these P1 potentials were respectively labeled the “early-to-mid-appearing P1 potential” and the “late-appearing P1 potential.” We also retrospectively reviewed the

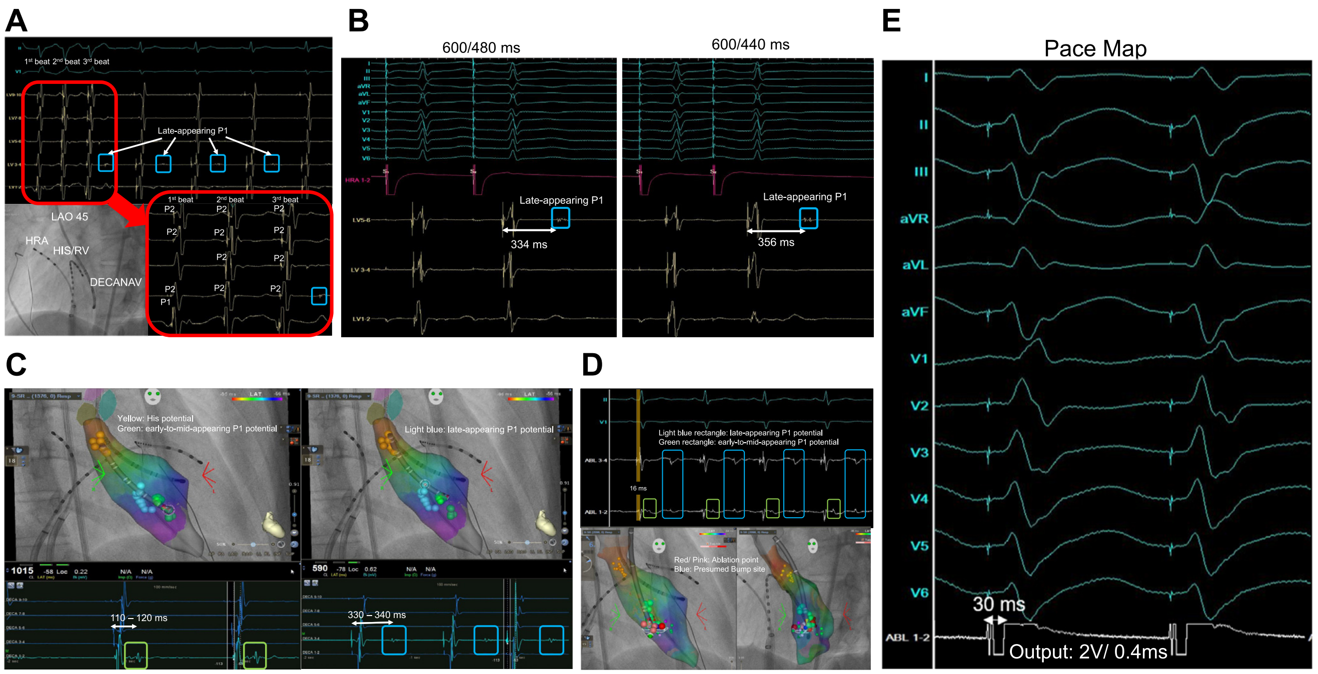

Figure 2 **A:** Intracardiac electrogram and fluoroscopic image at the moment of the "bump" phenomenon. **B:** Atrial programmed pacing that demonstrates decremental conduction in the late-appearing P1 potential. **C:** Two types of delayed P1 potentials, recorded by the DECANAV (DECA) catheter in the CARTO3 system (Biosense Webster, Diamond Bar, CA). The left panel displays the site recorded for the early-to-mid-appearing P1 potential, marked with a light blue tag, while the right panel displays the site recorded for the late-appearing P1 potential, marked with a green tag. **D:** The upper panel displays the local potentials of the ablation catheter at the beginning of radiofrequency ablation. The presumed bump site is identified by the blue tag and the ablation sites are identified by red or pink tags in the lower panel. **E:** Best pace map at the ablation site.

position of the DECANAV catheter in the CARTO3 system at the time when the bump phenomenon occurred and marked it with a blue tag (Figure 2D).

An irrigated contact force ablation catheter (ThermoCool SmartTouch SF; Biosense Webster) was inserted into the LV transaortically, and radiofrequency energy was applied at the site where both the early-to-mid-appearing and the late-appearing P1 potentials were simultaneously recorded. In view of the high score of 95 points (Figure 2E) obtained at this location using an automatic pace-mapping algorithm (PASO™; CARTO; Biosense Webster), we started radiofrequency ablation at this site. Using the conventional anatomical approach, we performed additional ablation targeting the mid-to-inferior septum in a linear fashion using radiofrequency energy positioned approximately two-thirds toward the apex and perpendicular to the septal plane (Figure 2D). Radiofrequency energy of 30–40 W was applied for 60 seconds for each to obtain an impedance drop of 10 Ω. Neither left bundle branch block nor a prolonged PQ interval was observed following the procedure.

After ablation, we performed postablation mapping using the DECANAV catheter and confirmed that the early-to-mid-appearing P1 potential could no longer be observed and that only the late-appearing P1 potential remained. Left posterior fascicular block was not observed. LPF-VT could not be provoked even by isoproterenol infusion and a programmed stimulus from both the HRA and RVA. The ablation session was ended without any complications. There has been no recurrence of LPF-VT during a year of follow-up.

Discussion

Although the P1 potential is the usual target for catheter ablation of LPF-VT, it cannot be recorded in up to one-third of cases, even when using a linear catheter.[5] In patients without a recorded P1 potential, radiofrequency energy should be delivered at the location of the earliest P2 potential during LPF-VT.[5] However, both of these indicators require induction of VT during the procedure, which is a limitation. When VT cannot be induced or sustained, an anatomical approach to ablation has been reported to be safe, though development of left posterior fascicular block was observed in 2 out of 6 cases.[6] The ablation site in our case was indeed similar to the one suggested by an anatomical approach, and the appearance of 2 types of post-QRS wave P1 potentials provided further guidance for our ablation.

It is not unusual for idiopathic left VT to become nonsustained or even noninducible as a result of catheter-induced mechanical block occurring during the mapping process. In our case, immediately after insertion of the mapping catheter into the LV, the VT could no longer be induced because of the bump phenomenon. However, the moment when the bump phenomenon occurred could be identified during the procedure by reviewing the intracardiac electrogram and

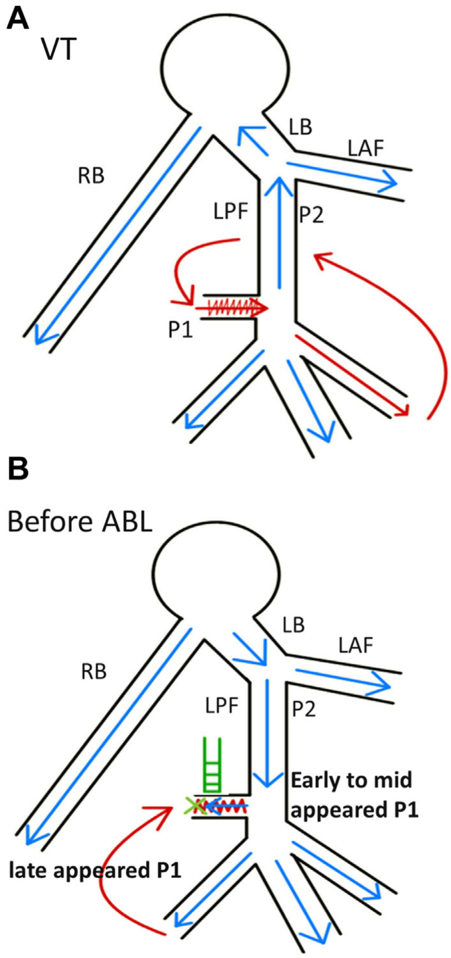

Figure 3 **A:** Illustration showing the appearance of the P1 potential in typical idiopathic left ventricular tachycardia (left, ventricular tachycardia; right, sinus rhythm). **B:** Illustration showing the mechanisms that cause the 2 types of delayed P1 potentials (left, before ablation; right, after ablation). ABL = ablation; LAF = left anterior fascicle; LB = left bundle; LPF = left posterior fascicle; RB = right bundle; SR = sinus rhythm; VT = ventricular tachycardia.

identifying the post–QRS wave P1 potentials. After the bump was recognized, the DECANAV catheter was immediately removed from the LV. When the DECANAV catheter was reinserted and placed in the green tag and light blue tag areas (Figure 2C), 2 different reproducible types of post–QRS wave P1 potentials were recorded consistently during sinus rhythm. Given that these observations were made before radiofrequency ablation, it is reasonable to speculate that the delay in the P1 potential was caused by catheter-induced mechanical block. By careful mapping of the area of interest using the DECANAV catheter, 2 different types of reproducible post–QRS wave P1 potentials at different phases were recorded. Figure 3A and 3B illustrates the mechanism by which these different potentials could be recorded. The late-appearing P1 potential in this case corresponds to a His potential–to–P1 interval of 340–350 ms, which is consistent with the definition of the late-appearing P1 potential in a previous study[4] and considered one of the indicators of successful ablation.

Regarding the early-to-mid-appearing P1 potential, it is possible that the 30-minute interval before reinserting the mapping catheter allowed for recovery of the catheter-induced mechanical block, resulting in formation of a slow conduction zone and appearance of the early-to-mid P1 potential (Figure 3B). Moreover, by performing radiofrequency ablation, the slow conduction zone was modified to a complete unidirectional block, which resulted in disappearance of the early-to-mid-appearing P1 potential and left only the late-appearing P1 potential (Figure 3B).

Although there are no reports on the recurrence rate in patients who have experienced the bump phenomenon during catheter ablation for idiopathic left VT, a previous study found that catheter-induced mechanical block in an accessory pathway was associated with a less favorable late outcome and that the recurrence rate was significantly higher than in patients without this type of block.[7]

A previous report described a method called “playback” ablation, in which the positional information of catheters in a 3-dimensional mapping system and the local potential of catheters are recorded continuously.[8] This method allows the physician to locate the bump site using a 3-dimensional mapping system and perform ablation, even if the catheter moves away from the bump site. In our case, the location where both the early-to-mid-appearing and late-appearing

P1 potentials were recorded closely matched the bump site identified by retrospective review and was the site targeted for ablation. The occurrence of the bump phenomenon during the procedure resulted in loss of inducibility of VT, which is the most reliable indicator of success. In addition to the traditional anatomical approach and retrospective identification of the bump site using the 3-dimensional mapping system, analysis of Purkinje potentials after catheter-induced mechanical block on the intracardiac electrogram may further improve the success rate of ablation of verapamil-sensitive idiopathic left VT.

Lastly, previous reports suggest that the HV interval during VT can be used to find the ablation site.[9,10] In our case, the retrograde His wave during VT could not be recorded. Nonetheless, based on the average HV interval during VT or PVCs from a previous report (-4.5 ms),[9] the value of the earliest retrograde presystolic potential can be predicted as 15.75 ms. This value is indeed consistent with 16 ms, as recorded using the ablation catheter (Figure 2D). Thus, our ablation site can be further backed up from this perspective.

Conclusion

When an irreversible bump phenomenon occurs during LPF-VT ablation procedure, it is important to carefully observe the Purkinje potentials and to recognize the moment of the bump phenomenon if possible. Dual post–QRS wave P1 potentials may provide additional guidance for identifying the optimal ablation site.

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Consent

The authors confirm that written consent for submission has been obtained from the patient in line with COPE guidance.

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References

1. Zipes DP, Foster PR, Troup PJ, Pedersen DH. Atrial induction of ventricular tachycardia: reentry versus triggered automaticity. Am J Cardiol 1979;44:1–8.
2. Klein GJ, Millman PJ, Yee R. Recurrent ventricular tachycardia responsive to verapamil. Pacing Clin Electrophysiol 1984;7:938–948.
3. Okumura K, Matsuyama K, Miyagi H, Tsuchiya T, Yasue H. Entrainment of idiopathic ventricular tachycardia of left ventricular origin with evidence for reentry with an area of slow conduction and effect of verapamil. Am J Cardiol 1988; 62:727–732.
4. Nogami A, Naito S, Tada H, et al. Demonstration of diastolic and presystolic Purkinje potentials as critical potentials in a macroreentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. J Am Coll Cardiol 2000; 36:811–823.
5. Liu Q, Shehata M, Jiang R, et al. Macroreentrant loop in ventricular tachycardia from the left posterior fascicle: new implications for mapping and ablation. Circ Arrhythm Electrophysiol 2016;9:e004272.
6. Lin D, Hsia HH, Gerstenfeld EP, et al. Idiopathic fascicular left ventricular tachycardia: linear ablation lesion strategy for noninducible or nonsustained tachycardia. Heart Rhythm 2005;2:934–939.
7. Bastani H, Insulander P, Schwieler J, et al. Cryoablation of superoparaseptal and septal accessory pathways: a single centre experience. Europace 2010; 12:972–977.
8. Ishidoya Y, Yotsukura A, Sano F, et al. Novel use of a 3-dimensional mapping system in cryoablation of right-sided and septal accessory pathways: playback ablation. J Interv Card Electrophysiol 2018;53:91–101.
9. Chen H, Zhang F, Yang B, et al. A novel method to identify the origin of ventricular tachycardia from the left fascicular system. Heart Rhythm 2016;13:686–694.
10. Ma W, Wang X, Cingolani E, et al. Mapping and ablation of ventricular tachycardia from the left upper fascicle: how to make the most of the fascicular potential. Circ Arrhythm Electrophysiol 2013;6:e47–e51.