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ORIGINAL ARTICLE

Upper turnaround point in a reentry circuit of the idiopathic left posterior fascicular ventricular tachycardia

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Abstract

Background: The anatomic extent of the reentry circuit in idiopathic left posterior fascicular ventricular tachycardia (LPF-VT) is yet to be fully elucidated. We hypothesized that entrainment mapping could be used to delineate the reentry circuit of an LPF-VT, especially including the upper turnaround point.

Methods: Twenty-three consecutive LPF-VT patients (mean age, 29 ± 9 years, 18 males) were included. We performed overdrive pacing with entrainment attempts at the left bundle branch (LBB) and the left His bundle (HB) region.

Results: Overdrive pacing from the LBB region showed concealed fusion in all 23 patients (post-pacing interval [PPI], 322.1 ± 64.3 ms; tachycardia cycle length [TCL], 319.0 ± 61.6 ms; PPI-TCL, 3.1 ± 4.6 ms) with a long stimulus-to-QRS interval (287.9 ± 58.0 ms, approximately 90% of the TCL). Pacing from the same LBB region at a slightly faster pacing rate showed manifest fusion with antidromic conduction to the LBB and minimal in-and-out time to the LBB potential (PPI-TCL, 21.3 ± 13.7 ms). Overdrive pacing from the left HB region showed manifest fusion with a long PPI-TCL (53.9 ± 22.5 ms).

Conclusions: Our pacing study results suggest that the upper turnaround point in a reentry circuit of the LPF-VT may extend to the proximal His-Purkinje conduction system near the LBB region but below the left HB region. The LPF may constitute the retrograde limb of the reentry circuit.

KEYWORDS
bundle of his, cardiac electrophysiologic study, idiopathic ventricular tachycardia, left bundle branch of his, posterior fascicle

Abbreviations: BBR-VT, bundle branch reentrant VT; ECF, entrainment with concealed fusion; ECG, electrocardiogram; EF, ejection fraction; EP, electrophysiology; HB, His-bundle; LAF, left anterior fascicle; LB, left bundle; LBB, left bundle branch; LPF-VT, left posterior fascicular ventricular tachycardia; LV, left ventricular; PCL, pacing cycle length; PPI, post-pacing interval; RB, right bundle; RBBB, right bundle branch block; RFCA, radiofrequency catheter ablation; RV, right ventricle; TCL, tachycardia cycle length.

1 | INTRODUCTION

Left posterior fascicular ventricular tachycardia (LPF-VT) is the most common type of idiopathic left ventricular (LV) tachycardia. Entrainment mapping study results have revealed that the tachycardia mechanism is reentry,[1,2] and now, hypothetical reentry circuit proposed by Nogami et al.[3] and Liu et al.[4] is accepted as the main reentry model of the LPF-VT. In this reentry model, the slow conduction zone of the reentry circuit is located between the ventricular myocardium and the proximal portion of the recorded diastolic Purkinje potential. In addition, with the two case reports on the negative participation of the LPF,[5,6] the proximal LPF was considered as a bystander of the reentry circuit.[7,8]

Meanwhile, participation of the LPF in the reentry circuit and a macro-reentry extending up to the basal septum of the LV have also been proposed in earlier studies.[9–11] In these studies, entrainment pacing results indicated that the septal Purkinje potential up to the mid-ventricle could be part of the circuit. In addition, the major conduction delay area is still unclear and needs to be elucidated. Hence, the exact reentry circuit, the area of critical conduction delay, and the role of the LPF are yet to be clarified. Therefore, in this research, we performed an overdrive pacing study with entrainment attempts at a proximal conduction system of the LV to investigate an anatomic reentry circuit of LPF-VT.

2 | MATERIAL AND METHODS

2.1 | Patient population

We included right bundle (RB) branch block (RBBB) morphology VT patients without structural heart disease who were referred to Asan Medical Center (Seoul, Korea) between January 2004 and December 2016 for electrophysiology (EP) study and radiofrequency catheter ablation (RFCA). An entrainment mapping study was attempted in 25 patients; of them, 23 patients in whom the study was possible were analyzed (Figure 1). The Institutional Review Board at Asan Medical Center approved this study (protocol code S2017-1294 and date of approval 2018.1.22), and written informed consent was obtained from all patients.

2.2 | Electrophysiological study

Procedures were carried out under a fasting state with light sedation after the withdrawal of antiarrhythmic drugs for at least five half-lives. Standard multi-electrode catheters were placed in the His-bundle (HB) region and the right ventricle (RV) apex. Bipolar electrograms were filtered at 30–500 Hz and recorded with a 12-lead surface electrocardiogram (ECG) using the Prucka Electrophysiology Lab system (Prucka, GE Medical Systems Information Technology, Milwaukee, WI, USA). Programmed ventricular stimulation for inducing VT was performed using a maximum of triple extrastimuli at two different drive cycle lengths (600 ms, 400 ms) from the RV apex and the RV outflow tract. Incremental burst ventricular pacing with constant cycle length was also performed. If sustained VT was not induced, the stimulation was repeated during intravenous isoproterenol infusion or after atropine injection.

2.3 | Entrainment and activation mapping

Mapping and ablation were performed with either a 7-Fr non-irrigated ablation catheter with a 4-mm tip or a 7.5-Fr irrigation catheter with a 3.5-mm tip. The catheter was introduced into the LV via the retrograde approach. Electrical potentials from the HB, left bundle (LB), left anterior fascicle (LAF) or LPF, and Purkinje fibers were attempted to trace and record during sinus rhythm and VT. In 13 patients, electro-anatomical mapping was performed using a three-dimensional mapping system (CARTO XP or CARTO 3, Biosense Webster, Diamond Bar, CA, USA; EnSite NavX or Velocity, Abbott/St. Jude Medical, St. Paul, MN, USA).

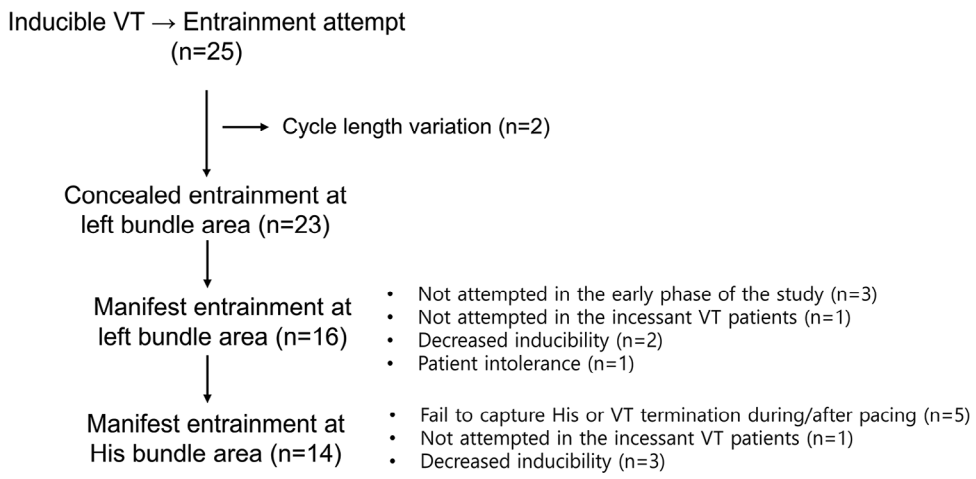

FIGURE 1 Patient enrollment flowchart and entrainment study results.

TABLE 1 Baseline clinical and electrophysiological characteristics.

	Total ($n = 23$)
Age, y	29.1 ± 8.7
Male sex	18 (78%)
LV EF (%)	59.0 ± 10.3
SR cycle length (ms)	786.4 ± 168.6
QRS duration during SR (ms)	92.3 ± 10.9
QTc interval during SR	403.0 ± 50.5
AH during SR (ms)	92.3 ± 17.1
HV during SR (ms)	48.9 ± 5.0
VT cycle length (ms)	353.7 ± 71.0
QRS duration in VT (ms)	143.0 ± 14.0
HV during VT (ms)	−21.5 ± 13.0
Follow-up, month (median, IQR)	15, 6–47
Recurrences	4 (17.4%)

Abbreviations: AH, Atrio-Hisian interval; HV, His-ventricular interval; IQR, interquartile range; LV EF, left ventricular ejection fraction; SR, sinus rhythm; VT, ventricular tachycardia.

Entrainment mapping was performed during sustained VT to investigate tachycardia's mechanism and elucidate the reentry circuit's extent. The pacing was performed with a pacing cycle length (PCL) 10–20 ms shorter than the tachycardia cycle length (TCL) and a pacing threshold of 8–15 mA. Then the difference between the post-pacing interval (PPI) and the TCL and between the stimulus-to-QRS interval and the electrogram-to-QRS interval were measured.

2.4 | Radiofrequency catheter ablation

Ablation targeted the abnormal diastolic Purkinje potential or the earliest presystolic Purkinje potential in the mid to apical septum during VT. If catheter contact was unstable or VT termination was not achieved, ablation was attempted in sinus rhythm. When VT became non-inducible during the EP study and mapping procedure, an empirical linear ablation strategy was performed.[12] The ablation was considered successful if VT was terminated during ablation and if any VT or ventricular echo beat was not inducible 30 min post-ablation during repeat programmed stimulation with or without isoproterenol infusion.

3 | RESULTS

3.1 | Patient characteristics

Baseline clinical and electrophysiological characteristics are shown in Table 1. The mean age was 29.1 ± 8.7 years, and 18 (78%) patients were male. Two patients presented with incessant VT refractory to antiarrhythmic drugs and showed decreased LV ejection fraction (EF), which was normalized after catheter ablation. Other patients had structurally normal hearts, with a mean LV ejection fraction of 61.7 ± 4.1%.

3.2 | Baseline electrophysiological study

The patients' AH (92.3 ± 17.1 ms) and HV (48.9 ± 5.0 ms) intervals were within normal ranges. The VTs were induced in the baseline condition in 10 patients or during isoproterenol infusion (1–10 μg/min) in 8 patients. Atropine was used in one patient. In two patients, VT was induced by spontaneous premature ventricular complexes in the baseline condition, and another two patients presented with incessant VT, as described above. The mean VT cycle length and QRS duration were 319.0 ± 61.6 ms and 143.0 ± 14.0 ms, respectively, and the mean HV interval during VT was −21.5 ± 13.0 ms.

3.3 | Entrainment study

To identify the mechanism of tachycardia and determine the proximal extent of the reentry circuit, we attempted entrainment mapping from the proximal His-Purkinje system, especially from the left HB and the left bundle branch (LBB) region, which encompasses distal LB to very proximal LPF.

3.3.1 | Entrainment mapping from the LBB region

The ablation catheter was retrogradely advanced via the aorta and was located at the left HB region, where the HB potential was visible along with atrial electrograms. Then, the ablation catheter was advanced slightly downward (0.5–1.0 cm) until the atrial electrogram was almost invisible while a clear LB potential was recorded. This area was considered as the LBB region (Figure 2A,B; Figure 3A–C). The LB-to-QRS interval at this LBB region was 29.2 ± 4.2 ms.

- Pacing at the LBB region with a PCL 10–20 ms shorter (304.3 ± 61.6 ms) than the TCL showed concealed fusion. Paced beats with the QRS complex identical to that of the tachycardia were accelerated to the pacing CL, and the PPI (322.1 ± 64.3 ms) measured at the "estimated" capture point matched the TCL (319.0 ± 61.6 ms). The capture point in the pacing catheter electrogram was inversely estimated by measuring the electrogram-to-QRS interval during the tachycardia that matched the stimulus-to-QRS interval during the entrainment pacing. As the pacing stimuli were recorded after the inscription of the QRS complex and the morphology of the QRS complex did not change during pacing, this pacing from the tip of the ablation catheter was presumed to have captured only the conduction system while the refractory ventricular myocardium was left unaffected. In addition, presystolic LBB potential was observed in the pacing catheter electrogram during the entrainment pacing. The stimulus-to-QRS interval (287.9 ± 58.0 ms) was 90.2 ± 4.8% of the TCL, suggesting that the pacing site is

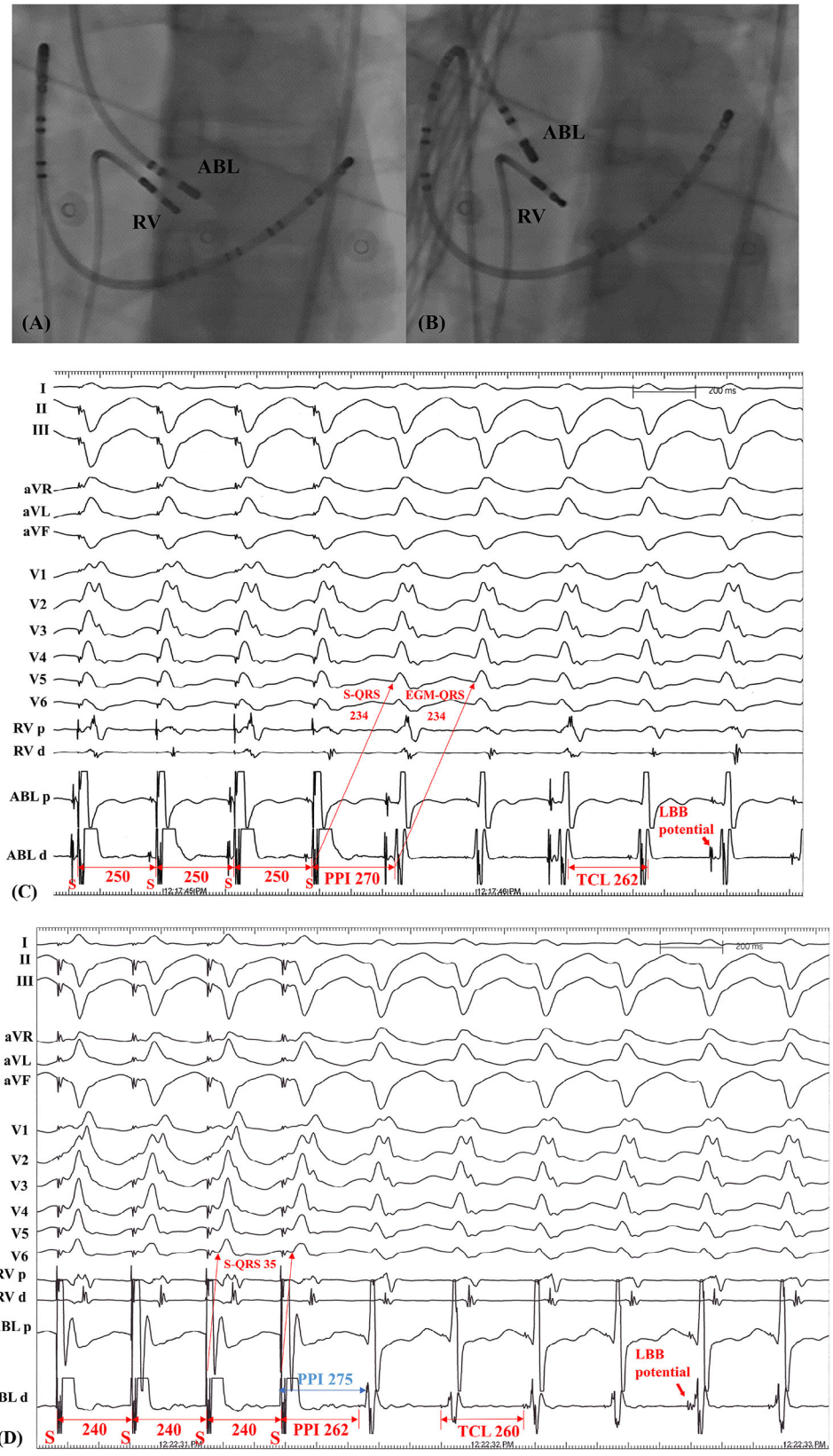

FIGURE 2 An example of entrainment mapping at the left bundle (LB) to LB branch (LBB) region which encompasses distal LB to very proximal left posterior fascicle (LPF). Figures (A) and (B) show left anterior oblique fluoroscopy images of the catheter position. The distal tip of the ablation catheter (ABL) was located at the LBB (A) and the His bundle (HB) region (B). A quadripolar catheter was located at the right ventricular (RV) septum. Figure (C) shows the surface ECG and intracardiac electrogram tracing during entrainment pacing from the LBB region. Surface ECG leads I, II, III, aVR, aVL, aVF, and V1–V6 are shown with intracardiac electrograms from the RV septum (RVp, RVd) and ablation catheter (ABL). The distal tip of the ablation catheter (ABL d) was located at the LBB region, and the presystolic LBB potential was recorded. The pacing was performed from

proximal to the slow conduction zone. This entrainment with concealed fusion (ECF) at the LBB region was observed in all 23 patients (Figures 2C and 3D), and these results (electrogram fusion and resetting of the tachycardia) illustrate the reentry mechanism of the tachycardia. Therefore, the VT was considered LPF-VT, and further entrainment pacing attempts were made.

- When the PCL was slightly shortened (279.1 ± 36.3 ms compared to the VT TCL 302.3 ± 35.5 ms, 20–30 ms shorter than the TCL), overdrive pacing from the LBB region yielded manifest fusion. The morphology of the entrained QRS complex was slightly different from that of the tachycardia, resulting in a minor reduction of the QRS complex duration and exaggeration of the left anterior fascicular block pattern (accentuation of the terminal R' in V1). Unlike in the ECF, the presystolic LBB potential was not observed at the pacing catheter during pacing, and the QRS complex appeared 30.7 ± 10.3 ms after the pacing stimuli with a clear isoelectric line between the pacing stimulus and the QRS complex, suggesting that the LBB potential (but not the surrounding ventricular myocardium) was activated antidromically by the pacing stimuli. Then, we could assume that this antidromic activation collided with the incoming wavefront of the previous beat, resulting in the fusion of the QRS complex, that is, manifest fusion. The PPI measured to the "estimated" capture point (323.8 ± 29.5 ms) matched the TCL (PPI-TCL = 21.3 ± 13.7 ms) ("PPI" presented with a blue arrow in Figures 2D and 3E). The PPI measured to the LBB potential ("PPI" presented with a red arrow in Figures 2D and 3E, 311.7 ± 37.8 ms) was not prolonged (PPI-TCL = 9.4 ± 10.2 ms). PPI would have been much more prolonged if the reentry circuit were located well below the LBB, as in Liu et al.'s model. This pacing with the manifest fusion was observed in 16 out of the 23 patients. In seven patients, this was not performed. The reasons were as follows: three patients in the early phase of our study when the concept of this research was not yet fully developed, one patient with incessant VT and decreased LV EF, two patients who showed markedly decreased inducibility of VT after previous entrainment attempts, and one patient who did not tolerate the further pacing attempts. Therefore, all 16 patients who attempted the pacing with the shorter PCL at the LBB region showed manifest fusion with PPI matching TCL.

3.3.2 | Entrainment pacing from the HB region

After the entrainment study at the LBB region, the ablation catheter was withdrawn slightly upward to the left HB region, where a clear HB potential and distinct atrial electrograms were recorded (Figure 3A–C). Pacing from the left HB area at a PCL 10–20 ms shorter (318.9 ± 62.7 ms) than the TCL (336.9 ± 67.1 ms) revealed manifest fusion with further increment in the stimulus-to-QRS interval (44.9 ± 6.0 ms), suggesting that the HB was selectively captured without direct ventricular myocardial stimulation. The QRS morphology was identical to that of the sinus rhythm, and PPI (391.5 ± 58.8 ms) and PPI-TCL (53.9 ± 22.5 ms) were much prolonged, suggesting that the left HB is off from the reentry circuit (Figure 3F). This manifest fusion at the left HB region was observed in 14 out of the 23 patients and was not attempted in 6 patients. The reasons were as follows: one patient was in the incessant VT state and decreased LV EF, the entrainment pacing attempt failed to capture the HB or VT was terminated during/after overdrive pacing in five patients, the VT became non-inducible during this entrainment pacing attempt in three patients. The results of the entrainment pacing study are summarized in Table 2.

3.4 | Catheter ablation

For ablation, we explored the sharp, discrete Purkinje potentials in the mid-to-apical septum. The area recording the diastolic potential (P1) 30–40 ms before the QRS onset and earliest presystolic Purkinje potential (P2) was aimed first, but the earliest P2 was targeted when the diastolic potential (P1) was not seen. Due to the risk of heart block, we did not attempt ablation at the earliest diastolic potential where the entrainment pacing attempt was performed. In 12 patients, the diastolic potential preceding the earliest Purkinje potential was found, and ablation was successfully done in this area; the mean diastolic potential-to-QRS interval was 48.6 ± 17.4 ms. Diastolic P1 potential was not recorded in six patients, and the earliest P2 was targeted. The mean Purkinje potential-to-QRS interval at the ablation site was 18.6 ± 9.3 ms. Ablation was performed during tachycardia in 12 patients and sinus rhythm in six patients. In five patients, VT was

the distal ablation catheter at a pacing cycle length (PCL) of 250 ms. The Tachycardia cycle length (TCL) was 262 ms. Surface ECG and intracardiac electrograms were accelerated to the pacing rate, and the tachycardia resumed after cessation of overdrive pacing. The LBB potential was not captured during the pacing. Paced QRS morphology was identical to that of the tachycardia. The postpacing interval (PPI) matched the TCL when measured to the point (estimated capture point) at which the pacing stimulus to QRS interval (S-QRS) was equal to the interval from the captured electrogram to QRS interval (EGM-QRS). This pacing result is consistent with concealed fusion and matching PPI (270 ms). Therefore, we can conclude that the tachycardia had a reentry mechanism and the pacing site is on the reentry circuit. In (D), the ablation catheter was still located at the same LBB region, and the pacing was performed with a PCL of 240 ms while the TCL was 260 ms. Surface ECG and intracardiac electrograms were accelerated to the pacing rate, and the tachycardia resumed after cessation of overdrive pacing. The QRS morphology during pacing was slightly different from that of the tachycardia, and the LBB potential was not observed during pacing. There was an isoelectric interval from the stimulus artifact to the QRS complex (35 ms). PPI measured to the estimated capture point ("PPI" presented with the blue arrow, the estimated capture point is the EGM of distal ablation catheter which has the 35 ms after the QRS onset) matched the TCL (PPI 275 ms—TCL 260 ms = 15 ms), implying that the estimated capture point is on the reentry circuit. Therefore, the impulse from the estimated capture point seems to have been conducted antidromically to the LBB. The PPI measured to the LBB ("PPI" presented with red arrow) was not prolonged (PPI 262 ms—TCL 260 ms = 2 ms), and this minimal (2 ms) in-and-out time from the capture point to the LBB suggests that the LBB is not far from (and most likely within) the reentry circuit. ABL p = electrogram at the proximal ablation catheter; ABL d = electrogram at the distal ablation catheter; RV p = electrogram at the proximal RV catheter; RV d = electrogram at the distal RV catheter. [Color figure can be viewed at wileyonlinelibrary.com]

not inducible after entrainment pacing attempt or activation mapping; therefore, a linear ablation strategy during sinus rhythm was applied in these patients. After ablation, all patients achieved non-inducibility of any VT during programmed stimulation with or without isoproterenol infusion.

3.5 | Follow-up

The median follow-up duration was 15 months (interquartile range, 6–47). Four patients had recurrence: two patients had an early recurrence of VT at 9 and 10 days after the procedure, and the other two had a late recurrence of VT at 2.1 and 4.6 years after the procedure. The patients with recurrent VT underwent successful redo catheter ablation and did not experience recurrence of VT after the procedure. Two patients with incessant VT initially showed heart failures that were resolved after successful ablation. No procedural complications were observed during the follow-up period.

4 | DISCUSSION

4.1 | Main findings

The main findings of our study are as follows: (1) Pacing attempts at the LBB region with a PCL 10–20 ms shorter than the TCL selectively captured the proximal conduction system and showed concealed fusion with a long stimulus-to-QRS interval (approximately 90% of the TCL). (2) Pacing from the same LBB region with a slightly faster PCL showed manifest fusion, and the electrogram/ECG analysis suggested antidromic activation of the presystolic LBB. In addition, the PPI measured to the LBB was not prolonged, suggesting that the proximal LBB is not far from (and likely a part of) the reentry circuit. (3) Pacing from the left HB region showed manifest fusion with a long (> 30 ms) PPI-TCL.

These results can be translated into the following electrophysiological implications: (1) The proximal conduction system near the LBB region is a part of the reentry circuit and constitutes the entrance of the slow conduction zone. (2) Considering the no or minimal "in-and-out" conduction time to the adjacent LBB during manifest fusion, the retrograde limb of the tachycardia likely extends upward to the LBB region, and the LPF seems to constitute the retrograde limb of the reentry circuit. (3) The HB is not a part of the reentry circuit, and the upper turnaround point of the LPF-VT may extend to the proximal His-Purkinje conduction system near the LBB but below the left HB.

4.2 | Proposal of new hypothetical reentry circuit of LPF-VT

Because the entrainment mapping results were reproducible in most of the studied LPF-VT patients, we propose a new model of the reentry circuit of LPF-VT to explain our study results. Figure 4 shows the schematic diagram of our hypothetical reentry circuit in relation to the entrainment pacing. In our model, the upper turnaround point is located in the LBB region, and the retrograde limb of the tachycardia circuit is presumed to be the LPF. The slow conduction zone of the reentry circuit arises from anisotropic lateral conduction in the LBB and the Purkinje fibers (most probably LV mid-septal fascicles) that constitute the anterograde limb.

4.3 | Concealed fusion at the LBB

As described above, attempts to entrainment pacing at the LBB region during LPF-VT resulted in concealed fusion with a long stimulus-to-QRS interval (Figures 2C and 3D). As the pacing stimuli were recorded after the inscription of the QRS complex and the morphology of the QRS complex did not change during pacing, the pacing stimuli might selectively capture the conduction system. According to the overdrive pacing model of Barbhaiya et al.,[13] our overdrive pacing at the LBB region advanced (or reset) the tachycardia by entering the slow conduction zone just downstream to the LBB activation. The matching PPI and long stimulus-to-QRS interval imply that the pacing electrode was located in the reentry circuit just downstream of the upstream LBB (Figure 4B).

4.4 | Manifest fusion at the LBB

Pacing with a slightly faster rate demonstrated an isoelectric line between the stimuli and the QRS complex, which indicates that pacing stimuli did not directly capture the ventricular myocardium (Figures 2D and 3E). Therefore, this faster pacing might have activated the conduction system antidromically (LBB and likely LPF, Figures 4C and 4D). Meanwhile, the PPI measured to the LBB potential matched the TCL. If the upper turnaround of the LPF-VT were located well below the LBB region (as in Liu et al.'s model), this matching PPI could not be possible considering the "in-and-out" conduction time through the LPF. Based on our model, this antidromically activated LBB potential should have yielded a PPI shorter than the TCL by the time the pacing stimulus was conducted antidromically from the capture point to the LBB. PPI measured to the "estimated" capture point was slightly (21.3 ± 13.7 ms) longer than the TCL, and the rate-dependent conduction delay in the reentry circuit might explain these differences.

4.5 | Participation of LPF as a reentry circuit of LPF-VT

Our entrainment pacing attempts were reproducible, especially with findings suggestive of selective conduction system capture. These results are explained only when a well-established track of the reentry circuit is present. The most plausible (anatomically reasonable) candidate is a circuit incorporating the LPF as the retrograde limb and the LV mid-septal fascicle as the anterograde limb.

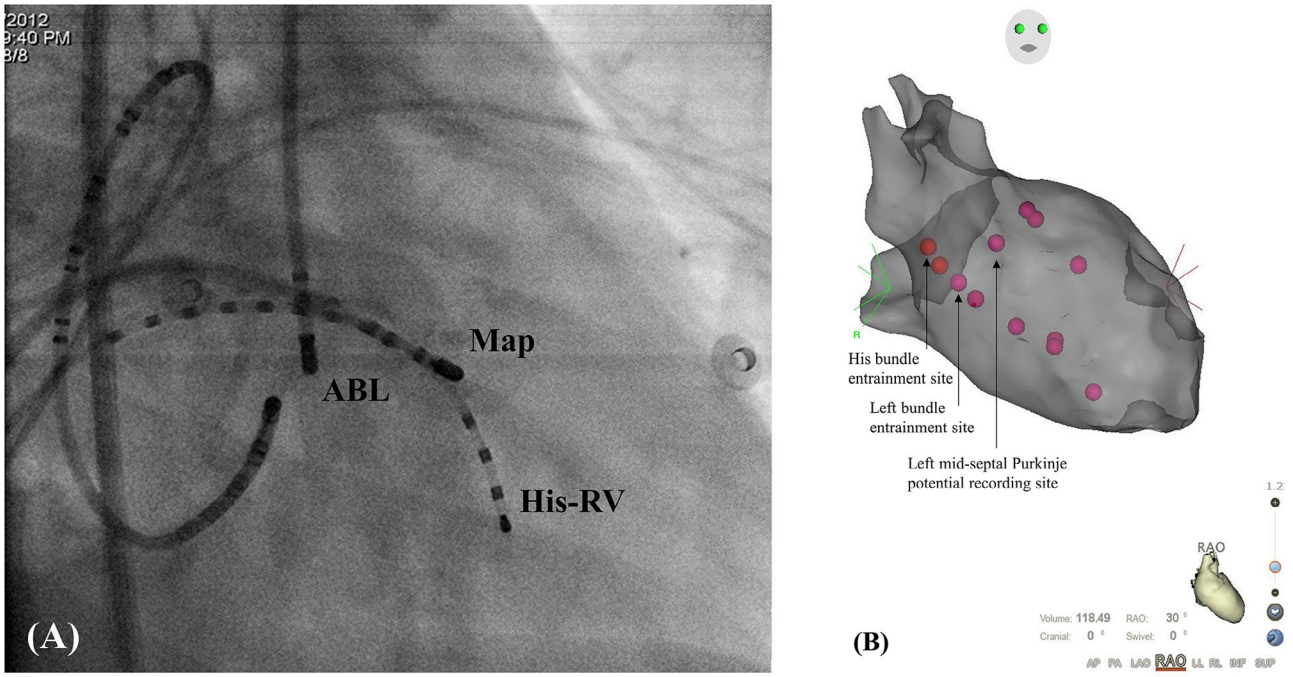

FIGURE 3 An additional example of entrainment pacing attempt from the left bundle branch (LBB) region and His bundle (HB) region during left posterior fascicular (LPF) ventricular tachycardia. A fluoroscopic image of the catheter position (right anterior oblique view) is shown in (A). One ablation catheter (ABL) was introduced into the left ventricle (LV) via retrograde aortic approach and located at the left side HB. Another ablation catheter (Map) was introduced into the LV via trans-septal puncture and located at the LV mid-septum region, which recorded LV mid-septal Purkinje potential. A His-RV catheter was located and recorded the right HB electrogram together with the RV apex electrogram. In (B), the three-dimensional reconstruction of LV geometry is displayed using the Carto 3 system (Biosense Webster). The red dots indicate the locations where the HB potential was observed, and the pink dots indicate the locations where the LB/LV mid-septal Purkinje potentials were observed during sinus rhythm. Recorded electrograms from the catheters before pacing are shown in (C). The left HB potential was seen at the distal ablation catheter (ABL d). The LV mid-septal Purkinje potential was seen at the distal electrode of another ablation catheter (Map, presumed location of this catheter is marked as a blue asterisk in Figure (4). Figures (D)–(F) show surface ECG and intracardiac electrogram tracing during entrainment pacing. Surface ECG leads I, II, aVF, V1, and V6 are shown with intracardiac electrograms from the high right atrium (HRA), right atrial septum (His-RV 7—8), right His bundle (His-RV 5—6), right ventricle proximal septum (His-RV 3—4), LV mid-septum (Map), and the LB region (ABL p, d). In (D) and (E), the ablation catheter was advanced just below the aortic cusp and located at the LBB region. In (D), the pacing was performed from the distal ablation catheter at a PCL of 300 ms. Tachycardia cycle length (TCL) was 316 ms. Surface ECG and intracardiac electrograms were accelerated to the pacing rate, and the tachycardia was resumed after cessation of overdrive pacing. Paced QRS morphology was identical to that of tachycardia, therefore, entrainment with concealed fusion was suggested. The pacing stimulus to the onset of the last entrained QRS complex interval was 277 ms (S-QRS). Then, it is possible that the distal ablation catheter EGM located 277 ms backward from the onset of the first QRS complex of the resumed tachycardia represents the estimated capture point. The PPI (320 ms) measured to the estimated capture point closely matched TCL. Entrainment pacing attempt from the same region with a slightly faster pacing rate (PCL 280 ms) was done and the result was shown in (E). Tachycardia with a cycle length of 315 ms was accelerated to the pacing rate and resumed after cessation of overdrive pacing. Paced QRS morphology was slightly different from the tachycardia, and there was a 34 ms interval between the stimulus and the QRS complex. We assumed that this phenomenon suggests that the LBB-LPF was antidromically activated and this antidromic wavefront (Nth beat) collided with the incoming orthodromic waves of the previous (N-1)th beat, resulting in fusion of the QRS complex. In the LV mid-septal region (Map proximal and distal), the Purkinje potentials at the proximal and distal Map catheters were orthodromically activated by the pacing stimuli (compare Purkinje activity of the last entrained QRS complex and the first QRS complex of resumed tachycardia), but the ventricular activation was advanced and reversed during pacing (distal to proximal during pacing while proximal to distal during tachycardia). This "fusion and reset" phenomenon also suggests a reentrant mechanism of fascicular VT and the presence of a slow conduction zone between the ablation catheter and the Map catheter. PPI measured to the estimated capture point (Abl d, "PPI" presented with blue arrow) closely matched the TCL (PPI 343 ms—TCL 315 ms = 28 ms), indicating that the pacing site (near the LBB) was on the reentry circuit. PPI measured to the LBB potential ("PPI" presented with red arrow) was not prolonged (PPI 330 ms—TCL 315 ms = 15 ms) and was slightly shortened due to the conduction time from the LBB to the estimated capture point. These findings (minimal in-and-out conduction time to the tachycardia circuit) imply that the LBB potential is close to the reentry circuit, possibly being located just upstream to the pacing site. In (F), the pacing was performed from the distal ablation catheter (slightly withdrawn compared to the pacing site of (D) and (E) at a PCL of 290 ms while the TCL was 315 ms. Surface ECG and intracardiac electrograms were accelerated to the pacing rate, and the tachycardia resumed after cessation of overdrive pacing. The QRS complex during pacing was narrow, and there was a clear isoelectric line between the pacing stimuli and the QRS complex (stim to QRS, 54 ms). One-to-one retrograde VA conduction was also present as the atrial electrograms at the ABL p and HRA electrodes. Considering the narrow QRS complex, we assumed that the pacing electrode captured the left side of His bundle. The PPI measured to the left HB was prolonged (386 ms) by 71 ms compared with the TCL (315 ms). This manifest fusion with long PPI indicates that the pacing site (left His bundle region) was distant from the reentry circuit. LBB, left bundle branch; LPF, left posterior fascicle; PCL, pacing cycle length; PPI, postpacing interval; TCL, tachycardia cycle length. [Color figure can be viewed at wileyonlinelibrary.com]

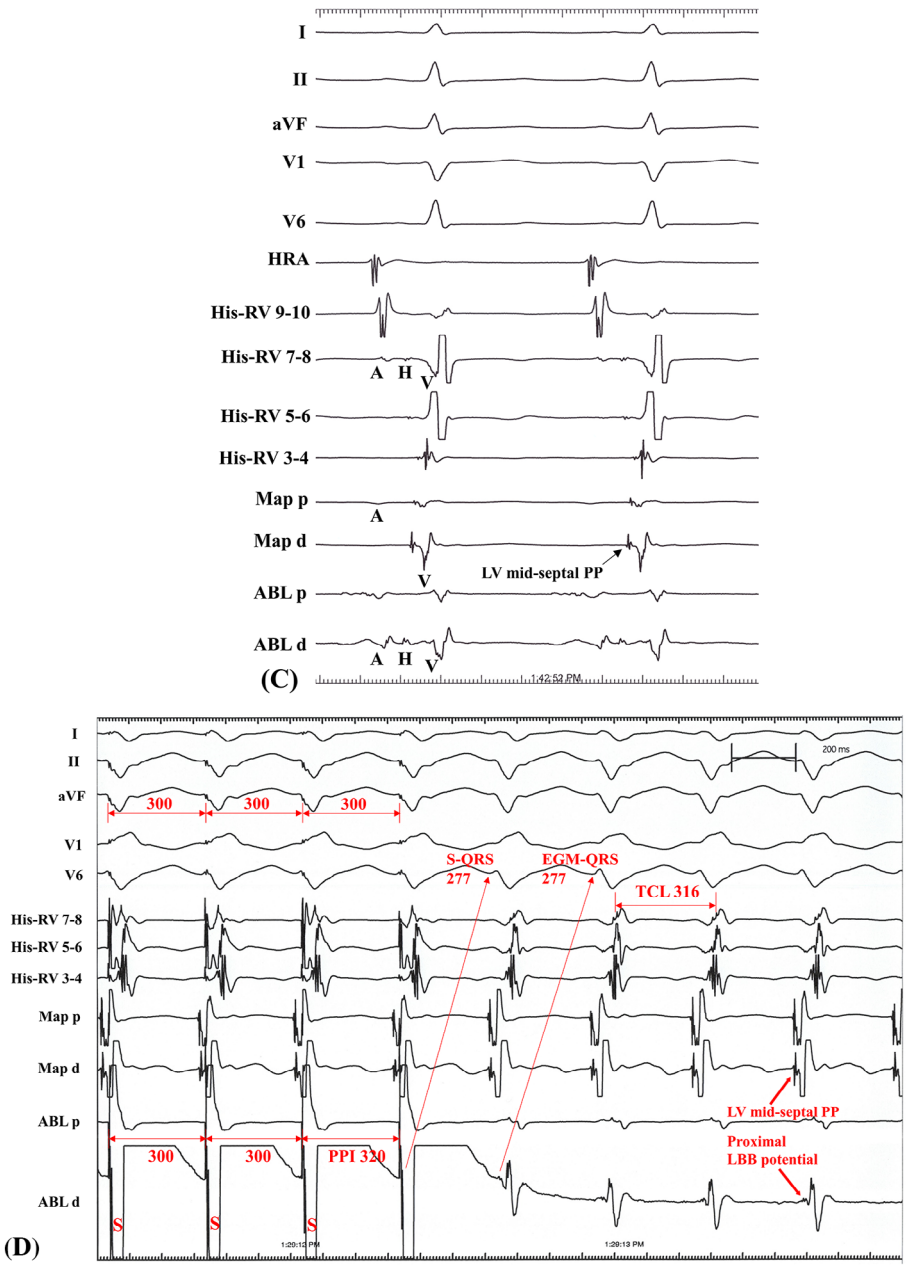

FIGURE 3 Continued

Participation of the LPF in the tachycardia circuit has been controversial. However, early studies and case reports suggested that the LPF and conduction system are part of the reentry circuit. In 1998, Lai et al.[9] used entrainment mapping to demonstrate that the entrance of the slow conduction zone of the LPF-VT was located away from the tachycardia exit site in one patient. In this patient, the entrance was LV mid-septal region, and the activation of presystolic Purkinje potential was later at the entrance site than the exit site. Therefore, the authors concluded that the electric activity was conducted from the exit site to the entrance site through the Purkinje or fascicular conduction system. Aiba et al.[10] recorded Purkinje and pre-Purkinje potentials during LPF-VT and showed that the Purkinje potential was activated bidirectionally toward the proximal (basal) and distal (apical) sites along the LPF. In addition, the authors performed the entrainment mapping at the proximal LV conduction system as in our study. It showed concealed fusion, and they were the first group to suggest that the reentry circuit is extended to a more proximal site along the LPF. However, their pacing site is considered to be located at the proximal-to-mid LPF, which is lower than the pacing site in our study.

4.6 | Slow conduction zone of the reentry circuit in an LPF-VT

The location of the slow conduction zone in an LPF-VT has yet to be identified. If the LBB is the entrance of the slow conduction zone

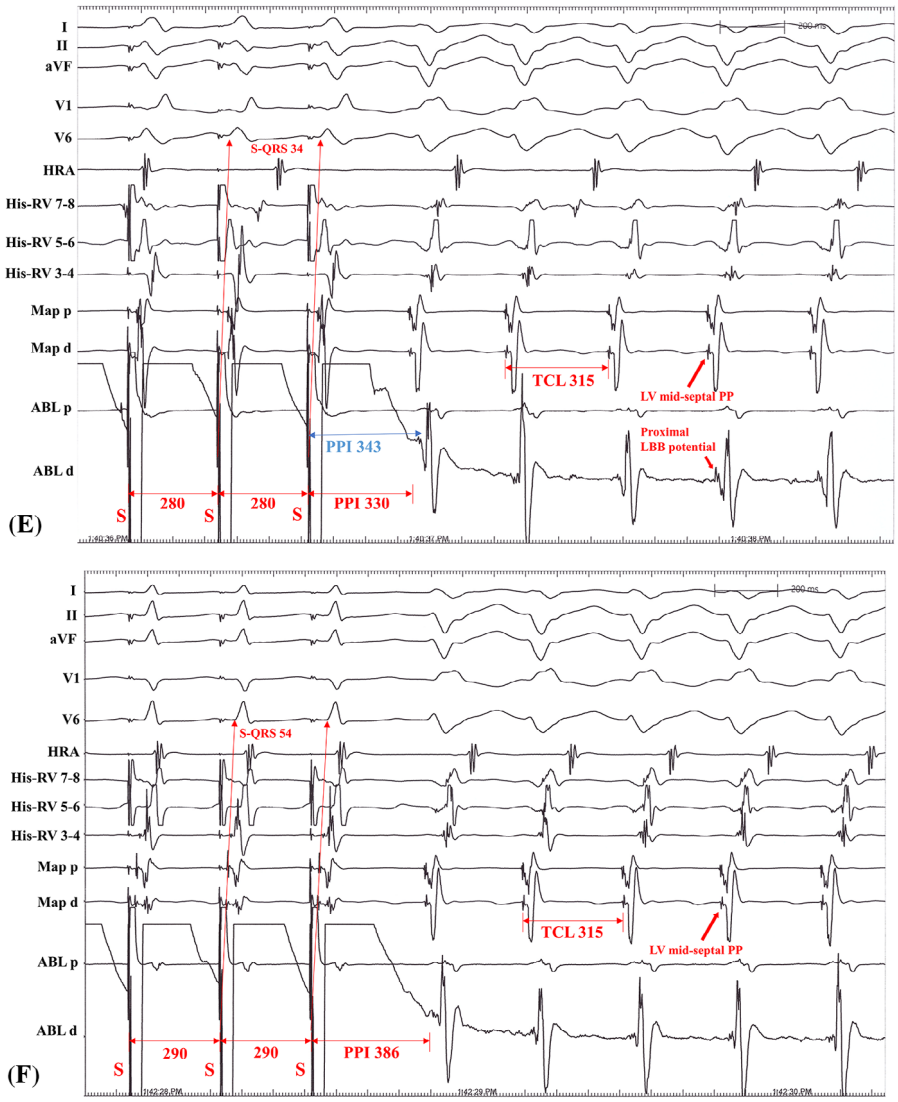

FIGURE 3 Continued

of the reentry circuit, the slow conduction zone would be located between the LBB and the tachycardia exit. Although diastolic potentials are often observed, the interval between the earliest diastolic potential and the latest Purkinje potential covers only a small proportion of the TCL even when recorded in the proximal LPF or LBB regions where concealed fusions were observed (average 16.2 ± 7.8% [(earliest diastolic potential to latest Purkinje potential)/TCL], Figures 2C and 3D). Therefore, the location of the significant conduction delay of LPF-VT seems to reside in a more proximal region of the conduction system. Considering that the interval from the diastolic potential to Purkinje potential (P1 to P2) is shorter than the long Purkinje potential-to-diastolic potential interval (P2 to P1) recorded near the proximal conduction system (Nogami et al.[3]), the major conduction delay seems to reside between the retrogradely conducted LBB potential (P2) and the P1 diastolic potential. In the bundle branch reentrant VT (BBR-VT), the HV interval during tachycardia is usually equal to or longer than the HV interval during sinus rhythm, and some patients with BBR-VT show apparently normal His-Purkinje conduction time during sinus rhythm. Anisotropic conduction in the upper turnaround point of the tachycardia (conduction from the proximal LB to the proximal RB) or functional conduction abnormalities within the LBB (presence of a split His or left bundle potential) is presumed to underlie this electrophysiologic observation of the BBR-VT.[14,15] We assume that these unique conduction properties of the proximal His-Purkinje system could also apply to patients with LPF-VT. During entrainment mapping, split or fragmented HB potential was observed in some of our patients (during sinus rhythm in four patients [Figure S1A] and during LPF-VT in five patients [Figure S1B]).

4.7 | The hypothesized antegrade limb of a reentry circuit in the LPF-VT

The left His-Purkinje system is a fan-like structure covering the endocardial surface of the basal septal LV with three major divisions (LAF, LPF, and middle or septal fascicle) or a bifascicular structure and

TABLE 2 Results of the entrainment pacing study.

	Mean ± SD (ms)
Concealed entrainment at LB ($n = 23$)	
Tachycardia cycle length	319.0 ± 61.6
Pacing cycle length	304.3 ± 61.1
TCL-PCL	14.7 ± 5.5
Postpacing interval to ECP	322.1 ± 64.3
doi:PPI-TCL	3.1 ± 4.6
S-QRS/TCL	90.2 ± 4.8 (%)
Manifest entrainment at LB ($n = 16$)	
Tachycardia cycle length	302.3 ± 35.5
Pacing cycle length	279.1 ± 36.3
TCL-PCL	23.2 ± 11.4
Postpacing interval to ECP	323.8 ± 29.5
doi:PPI-TCL	21.3 ± 13.7
Postpacing interval to LBB	311.7 ± 37.8
doi:PPI-TCL	9.4 ± 10.2
S-QRS	30.7 ± 10.3
Manifest entrainment at His bundle ($n = 14$)	
Tachycardia cycle length	337.7 ± 64.8
Pacing cycle length	318.7 ± 60.5
TCL-PCL	19.0 ± 9.3
Postpacing interval	391.5 ± 58.8
PPI-TCL	53.9 ± 22.5
S-QRS	44.9 ± 6.0

Abbreviations: ECP, estimated capture point; LB, left bundle; PPI, postpacing interval; S-QRS, interval from pacing stimulus to next QRS onset; TCL, tachycardia cycle length.

functional interconnection between the fascicles.[16–18] We assume that the LV mid-septal fascicle could act as an antegrade limb in our model (Figure 4), but any smaller fascicle or Purkinje fiber connected with the proximal LB may behave as the antegrade limb of the reentry circuit. During entrainment mapping in the LBB region, mechanical contact of the catheter frequently induced the "bump" phenomenon.[19] Because of this bump, we could not perform the entire entrainment study in some of our patients. Also, in 6 out of the 23 patients, we had to deliver empirical linear ablation at the LV posteroinferior septum due to the non-inducibility of tachycardia after the entrainment study at the proximal conduction system. This "bump" phenomenon may be evidence that the reentry circuit is composed of a conduction system in the high septal region located at the superficial endocardium, which is vulnerable to mechanical trauma.

4.8 | Comparison with the proposed reentry circuit by Liu and colleagues and others

Recently, Liu et al. published an article describing the hypothetical reentry model involving the ventricular myocardium based on the response to premature ventricular stimuli or entrainment pacing from the RV septum.[4] Specifically, the authors proposed the presence of a slow conduction zone which is presumed to be in the proximal portion of the diastolic potential. Entrainment of tachycardia and orthodromic activation of P2 with a long conduction time (PPI of P2 was equal to the PCL) was demonstrated during overdrive pacing at the RV mid-septum. The authors concluded that the ventricular septum is involved in the reentry circuit of LPF-VT. However, their pacing results do not necessarily indicate that the RV mid-septum is within the circuit. Their pacing results may be explained as follows in our point of view: the pacing at the RV septum retrograde conducted through the RB and penetration of the reentry circuit in the proximal conduction system (from the RB to the right side His, then to the left side HB and the LBB). This concept is supported by the advanced timing of the right HB, LBB, and LPF potentials after the delivery of premature ventricular stimulation or entrainment pacing in their tracings (Figures 3C, 4C, and 4D in Liu et al.[8]).

More recently, the same authors demonstrated that false tendons (FTs) provided an electrophysiologic substrate for LPF-VT using intracardiac echocardiography imaging.[20] They observed a 100% prevalence of FTs at the posterior septal region in all patients and recorded P1 potentials from FTs via a multielectrode catheter in 22/28 of the study patients. This may strongly indicate that the FTs are the electroanatomic basis for the anterograde arm of LPF-VT. However, FTs may not be a specific structure for LPF-VT,[21] and, as the authors mentioned, distinguishing the FTs from the endocardium of the posterior septum is practically difficult.[20] Participation of the left septal fascicle has also been proposed.[22] The complex septal fascicular network with its multiple anastomoses and substantially slower conduction velocity caused by load mismatch might facilitate reentry involving the mid-septal fascicles in patients with FVT. If all the reported electrophysiologic finding be considered, the true reentry circuit of LPF-VT might by much more complex than those proposed hitherto, and a "figure-of-8" like model may be necessary to integrate all these contradictory findings.

4.9 | Clinical implication

We found that most of the LPF-VTs are entrained from the proximal conduction system; these results may be used for differential diagnosis of the RBBB morphology-wide QRS complex tachycardias. Concealed fusion at the LBB region can be a good indicator for identifying the LPF-VT with a reentry mechanism. If the PPI is long, the possibility of other mechanisms, such as papillary muscle VT or focal mechanism, should be considered. In addition, the possible involvement of the LPF in the reentry circuit may provide a rationale for the proposed empirical ablation transecting the LPF in patients with non-inducible tachycardias.

4.10 | Limitation

First, our data is from a single-center study with a relatively small number of patients. Second, we did not use multipolar electrode catheters used in previous studies and did not obtain a mapping of the

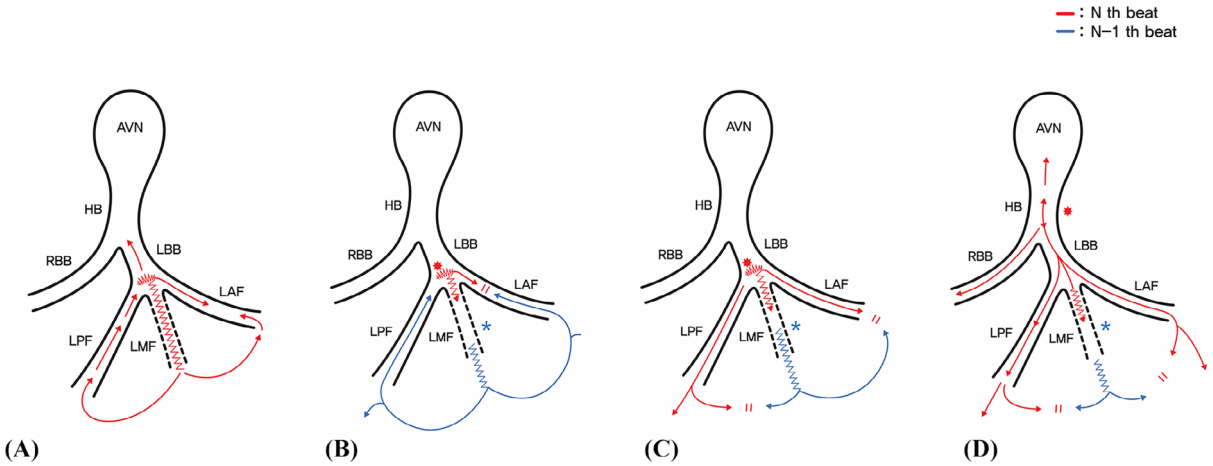

FIGURE 4 Proposed reentry circuit of left posterior fascicular ventricular tachycardia (LPF-VT) and diagrammatic presentation of our pacing results according to the newly proposed reentry circuit model during VT. Arrows indicate the direction of activation, serpentine lines indicate slow conductions, blue lines indicate the orthodromic wavefronts of the previous (N-1)th beat, and red lines indicate the wavefronts from the pacing impulse entering the VT circuit (Nth beat). (A) In our hypothesis, the retrograde limb of the tachycardia circuit is presumed to be the LPF, and the upper turnaround point is located around the left bundle branch (LBB) region. The slow conduction zone of the reentry circuit seems to arise from anisotropic lateral conduction in the LBB and/or the Purkinje fiber that constitutes the anterograde limb (e.g., left middle or septal fascicle) of the circuit or intervening ventricular muscle between fascicles. (B) Schematic diagram of the concealed fusion of VT during pacing at the LBB region. The pacing stimulus was delivered at a late diastolic phase just after the inscription of the LBB electrogram, which was activated by the previous (N-1)th beat and collided with it between the LBB and the capture point. The Nth pacing stimulus captures the region slightly downstream to the LBB and advances the tachycardia. This enters the slow conduction zones (likely created by the anisotropic lateral conduction in the LBB) and travels along the antegrade limb of the circuit (left ventricular (LV) mid-septal fascicle) and the left anterior fascicle (LAF). This antegrade wavefront of the Nth beat collides with the retrograde wavefront of the (N-1)th beat in the LAF as well. (C) Schematic diagram of the manifest fusion with a slightly faster pacing cycle length (PCL). As the PCL is shortened, a larger proportion of the reentry circuit is occupied by the Nth beat. The LBB and the LPF are antidromically activated and the excitable gap of VT in the antegrade limb (LV mid-septal fascicle) is reduced. The paced wavefront conducting to the LAF collides with the orthodromic wavefront of the (N-1)th beat in a more distal part of the LAF but is still concealed in the fascicle. This results in altered QRS morphology during entrainment pacing (LAF block pattern) and some isoelectric interval (30.7 ± 10.3 ms) from the stimulus-to-QRS complex. (D) Schematic diagram of the manifest fusion during pacing from the left His bundle region. The pacing impulse simultaneously captures LAF and LPF, which leads to normal conduction as in sinus rhythm. The pacing stimulus also enters the slow conduction zone of the circuit (septal or middle fascicle) and resets or maintains the tachycardia (Nth beat). Impulse wavefront from the previous (N-1)th beat collides with the antidromic waves of the Nth beat conducting through the LPF and LAF. As the pacing site is remote from the turnaround point, pacing in this left His bundle region shows manifest fusion with long post-pacing interval. AVN, atrioventricular node; HB, His bundle; LAF, left anterior fascicle; LBB, left bundle branch; LMF, left mid-septal fascicle; LPF, left posterior fascicle; RBB, right bundle branch. [Color figure can be viewed at wileyonlinelibrary.com]

sequential Purkinje potential activation. Third, our entrainment pacing was not performed in other (more distal) parts of the Purkinje system and thus could not directly demonstrate the participation of the LPF in the reentry circuit. The possibility of different paths running parallel to the LPF and joining the LPF near the LBB region cannot be excluded. Fourth, our results may not be applicable to all patients with LPF-VT, and the possibility of bystander LPF should be considered in a subset of LPF-VT patients.[5,6] Fifth, our pacing protocol has not been performed in other forms of idiopathic LV tachycardias, such as LAF tachycardia and upper septal fascicular tachycardia. Understanding the anatomic extent of the reentry circuit and the location of the upper turnaround point in these variants of fascicular tachycardias awaits further study.

5 | CONCLUSION

In patients with LPF-VT, entrainment mapping at the LBB region showed concealed fusion with matching PPI, whereas pacing from the HB region resulted in manifest fusion with long PPI. In addition, entrainment pacing from the LBB at a faster pacing rate resulted in manifest fusion with an antidromic activation of the presystolic LBB and minimal in-and-out time to the LBB. These results suggest that the upper turnaround point of the LPF-VT may extend to the proximal His-Purkinje conduction system near the LBB, and the LPF may constitute the retrograde limb of the reentry circuit.

AUTHOR CONTRIBUTION

Conceptualization: Gi-Byoung Nam. *Formal analysis*: Jongmin Hwang. *Investigation*: June Hong Kim, Jun Kim, Kee-Joon Choi, You-Ho Kim. *Writing–original draft*: Jongmin Hwang. *Writing–review and editing*: Gi-Byoung Nam.

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The authors have nothing to report.

CONFLICT OF INTEREST STATEMENT

Authors have no conflict of interest.

DATA AVAILABILITY STATEMENT

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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