MacroeXenterant Loop in Ventricular Tachycardia
From the Left Posterior Fascicle
New Implications for Mapping and Ablation

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Background—The underlying mechanisms of reentry during left posterior fascicular ventricular tachycardia (LPF-VT) remain unclear. The purpose of this study is to describe the components of LPF-VT reentry circuit and their electrophysiological properties.

Methods and Results—Fourteen consecutive patients with LPF-VT underwent electrophysiology study and radiofrequency ablation. Via a multipolar electrode catheter placed from a retrograde aortic approach, a sharp inflection, high-frequency potential (P1) was detected in 9 patients (64%). The ranges of length and velocity of recorded P1 were 9 to 30 mm and 0.5 to 1.2 mm/ms, respectively. MacroeXentry involving the ventricular myocardium was confirmed to be the mechanism in all patients by premature ventricular stimuli delivery or entrainment of LPF-VT with progressive fusion, or both. During LPF-VT, the earliest left posterior fascicle (LPF, P2) was considered to be the site of connection between P1 and P2, and the site of the earliest P2 along the left posterior ventricular septum correlated well with the His-ventricular interval during tachycardia. Radiofrequency ablation focused on the P1 potentials (9 patients with a recorded P1) or earliest P2 (5 patients without a recorded P1) was successful in all 14 patients. After 4.5±3.0 months of follow-up, no patients had recurrence of LPF-VT.

Conclusions—The LPF-VT macroreentrant loop involves the ventricular myocardium, a part of the LPF, a slow conduction zone, and in certain cases, a specially conducting P1 fiber. The His-ventricular interval during LPF-VT correlates with multiple electrophysiological measures and is a useful marker for identification of the optimal ablation site. (Circ Arrhythm Electrophysiol. 2016;9:e004272. DOI: 10.1161/CIRCEP.116.004272.)

Key Words: bundle of His • catheter ablation • electrophysiology • myocardium • tachycardia

See Editorial by Nogami

Nogami et al1 and Morishima et al2 have reported that the P1 potential is a critical component of reentry and could be detected in 75% patients with LPF-VT. They demonstrated that the mechanism of LPF-VT with recorded P1 potentials was a macroreentrant circuit involving abnormal Purkinje tissue with decremental properties (P1) acting as a slow conduction zone for the antegrade limb, left ventricular septal myocardium as the retrograde limb, and LPF (P2) as a bystander of the reentry circuit. However, based on these observations, we still cannot identify the reentrant mechanism of LPF-VT in the subgroup of patients that do not have a P1 potential recorded (estimated to be 25% of all such VTs).

The purpose of this study is to describe the components of LPF-VT reentry and their electrophysiological properties. Meanwhile, with a focus on the His-ventricular (HV) interval during LPF-VT, we describe the use of this parameter for mapping and ablation of LPF-VT.

Methods

Patient Population
Between April 2015 and February 2016, 14 consecutive patients with symptomatic LPF-VT and without structural heart disease underwent electrophysiology study and radiofrequency ablation in our center.
Macrocorticular Loop in Fascicular Tachycardia

WHAT IS KNOWN

• Left posterior fascicular ventricular tachycardia (LPF-VT) originates from the left ventricular conduction system and reentry is the mechanism.
• During LPF-VT, a diastolic potential (P1) can be recorded in some patients, and is involved in the reentry circuit in these patients.

WHAT THE STUDY ADDS

• The mechanism of LPF-VT is macro-reentry involving the ventricular myocardium, a part of the left posterior fascicle, a slow conduction zone, and a P1 fiber (in the cases with recorded P1).
• P1 potentials may not be recorded in some patients for several reasons.
• The HV interval during LPF-VT is a useful marker guide mapping and ablation of these arrhythmias.

Electrophysiology Study

Before electrophysiology study, all antiarrhythmic medications were discontinued ≥5 drug half-lives.

A 6F decapolar catheter (CSL; St. Jude Medical, Inc, St Paul, MN) was positioned in the coronary sinus via the left femoral vein. A 6F quadripolar catheter (CRD-2, St. Jude Medical, Inc) or a 6F octapolar catheter (Cordis Webster) was positioned at the His-bundle region via the left femoral vein. A multipolar electrode catheter (Duodecapolar, Livewire, St. Jude Medical, Inc; MEC) with 1-mm electrode length and 2-mm interelectrode spacing was positioned at the left posterior septum via a retrograde aortic approach.

Intracardiac electrograms were recorded using a digital electrophysiological recording system (EP-Workmate Electrophysiology; St Jude Medical, Inc). All bipolar electrograms were filtered between a bandpass of 30 and 300 Hz. Pacing was performed at twice the diastolic threshold with a programmable stimulator using a pulse width duration of 2 ms.

Before the MEC was positioned, the baseline atrio-His and HV intervals were measured during sinus rhythm (SR) and programmed, or burst stimulation was performed from the coronary sinus catheter and RV catheter, or both. If sustained LPF-VT was noninducible from the coronary sinus catheter and RV catheter, or both, the stimulation procedure was repeated after isoproterenol infusion (1–3 μg/min). If sustained LPF-VT was induced, the HV interval, QRS width, and tachycardia cycle length (TCL) were measured. Then, the MEC was positioned gently along the left posterior ventricular septum with an attempt to record LPF potentials during SR and tachycardia.

Via the MEC, 2 distinct high-frequency potentials might be observed during LPF-VT and characterized by the following (Figure 1A):

P2 potentials: a high-frequency, presystolic potential that was observed during LPF-VT and sinus rhythm, which represents activation of the LPF.

P1 potentials: a sharp inflection, high-frequency potential activated from the proximal to distal MEC during LPF-VT preceding P2 potentials.

During electrophysiological study, the MEC was adjusted carefully along the left ventricular septum to record the actual P2 potential. For example, as in Figure 1, various fascicular potentials (FPs) were recorded at 2 different positions of the left ventricular septum via the MEC. Positioning the MEC at the left posterior ventricular septum (Figure 1A), a FP was recorded during SR, and a P1 was recorded with the FP acting as the real P2 preceding local ventricular potentials and the onset of QRS during LPF-VT. However, when the MEC was located at a slightly anterior portion of the ventricular septum (Figure 1B), a FP was observed during SR, while the FP was later than the local ventricular potentials and the onset of QRS during LPF-VT without a recorded P1, indicating that this was not an actual P2 recording.

If a P1 potential was recorded, the length of recorded P1, conduction time, and velocities were measured via the MEC based on the recording electrode distance. The conduction time of P2 was measured in all patients.

To categorize the location of potentials, the left ventricular septum was divided into 9 segments equally from the apex to the base near the His-bundle region (Figure 2).

Electrophysiological Stimulation Maneuvers During LPF-VT

1. Premature ventricular stimuli (PVCs) were introduced from the RV septum beginning at 10 ms shorter than the TCL, until loss of ventricular capture or termination of tachycardia.
2. Ventricular burst pacing was performed from the RV septum in an attempt at entrainment of LPF-VT with decreasing cycle length beginning at 5 ms shorter than the TCL.
3. Delivery of premature atrial stimuli and atrial burst pacing was performed from the proximal coronary sinus beginning at 10 ms shorter than the TCL, until loss of atrial capture or termination of tachycardia.

Mapping and Catheter Ablation

The left ventricular geometry was created using a 3-dimensional electroanatomic mapping system (Ensite-Velocity System, St Jude Medical Inc, St Paul, MN). The position of the MEC with recorded P1 and left posterior fascicle potentials and ablation points were marked on the 3D anatomic map.

Radiofrequency (RF) ablation was guided by 3-dimensional mapping and the MEC. A 4-mm saline-irrigated ablation catheter (Cool Flex; St Jude Medical, Inc) was introduced into the left ventricle via a large curve deflectable sheath (Agilis, St Jude Medical, Inc) with a trans-septal approach or via a retrograde aortic approach for ablation. In the cases with recorded P1 during LPF-VT, the ablation target was focused on the distal P1. Otherwise, in the cases without a recorded P1, ablation was targeted at the earliest P2 often with a fragmented pattern.

RF energy was delivered during either LPF-VT or sinus rhythm with a power of 30 to 35 W and flow rate of 13 mL/min. After every RF application, reinduction of LPF-VT was attempted using the same programmed stimulation procedure as before. The successful end point of RF ablation was noninducibility of LPF-VT.

Follow-Up

All patients were scheduled for visits to the arrhythmia clinic at 2 weeks and 12 weeks postprocedure. During each visit, 12-lead ECG was performed for each patient. After the 12-week visit, follow-up information was obtained through telephone interviews with patients.

Results

Patient Characteristics

The patients’ baseline characteristics are shown in Table. Previous ablation had been performed in 3 of the 14 patients (Patient 1, 9, and 11). During electrophysiology study,
Figure 1. Example of mapping of the left ventricular (LV) septum with the multipolar electrode catheter (MEC). Various potentials were recorded via the MEC at 2 different positions along the LV septum during sinus rhythm (SR) and left posterior fascicular ventricular tachycardia (LPF-VT). A, The MEC was located at the left posterior ventricular septum. During SR (left), a fascicular potential was recorded via the MEC. During LPF-VT (middle), P1 was recorded with the fascicular potential preceding local ventricular potentials and the earliest portion of P2 preceding the onset of QRS. Right anterior oblique fluoroscopic images and the diagram of the MEC position are shown in right. B, The MEC was located at more anterior portion of the LV septum. During SR (left), a fascicular potential was recorded via the MEC. During LPF-VT (middle), there was no P1 recorded, and the fascicular potential was buried in the local ventricular potentials. Right anterior oblique fluoroscopic images and the diagram of the MEC position are shown in the right. CS indicates coronary sinus electrogram; HIS, His-bundle electrogram; electrogram; RAO, right anterior oblique; and RV, right ventricular electrogram.
LPF-VT was induced in all patients. LPF-VT could be induced by programmed or burst ventricular stimulation in all but one patient (patient 14), whereas by programmed atrial stimulation in 4 patients (patient 5, 12, 13, and 14). In 3 of 14 patients (21%), LPF-VT induction also required isoproterenol infusion. In all patients, the range of HV interval during LPF-VT was −38 to 0 ms.

Conduction Properties of P1 and P2 During LPF-VT

In 9 of all patients (64%), P1 potentials were recorded with a conduction sequence from proximal to distal MEC during LPF-VT at the left posterior ventricular septum. The range of length and velocity of recorded P1 were 9 to 30 mm and 0.5 to 1.2 mm/ms, respectively. Additionally, the velocity of P2 via the MEC was also measured in all 14 patients with the range between 1.4 and 3.0 mm/ms. In all patients during LPF-VT, the locations of earliest P2 were located at the left posterior ventricular septum at segments between 3 and 6 (Table and Figure 2). A correlation was observed between the HV interval and location of earliest P2 during LPF-VT, the more negative HV interval during LPF-VT correlated with a more distal location of earliest P2. The range of interval between the earliest P2 and QRS was between 17 and

Table. Patient Characteristics and Electrophysiological Study Data

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Patients are listed according to HV interval during LPF-VT. AH indicates atrio-His; AHSR, AH interval during sinus rhythm; HV, His-ventricle; HVSR, HV interval during sinus rhythm; HVVT, HV interval during LPF-VT; P2-QRS, the interval between the earliest P2 and onset of QRS during LPF-VT; P2V1, earliest P2 potential during LPF-VT; QRSVT, QRS duration during LPF-VT; RBBB, right bundle branch block; TCL, cycle length of tachycardia; and VSR, ventricular potential during sinus rhythm.

*The left ventricular septum was divided into 9 segments equally from the apex to the base near the His-bundle region (Figure 2).
†Patients with previous ablation.
Figure 3. In patient 11 with a slightly negative His-ventricular (HV) interval of −13 ms during left posterior fascicular ventricular tachycardia (LPF-VT) a relatively short P1 was recorded via the multipolar electrode catheter (MEC). A, During sinus rhythm (SR), the LPF was recorded via the MEC along the left posterior ventricular septum. B, During LPF-VT, P1 was recorded with a length of 9 mm along 3 pairs of electrodes at the distal MEC, relatively shorter than most of the patients with a recorded P1 and a more negative HV interval. Some low amplitude, far-field potentials (arrow) with an antegrade conduction sequence were also noted at the mid and proximal MEC region. In this case, the proximal portion of the P1 fiber may be nonparallel and slightly distant from the LPF and MEC. C, In the same patient with slightly proximal movement of the MEC toward the His bundle, a premature ventricular stimulus introduced from the midright ventricular septum during tachycardia advanced the immediate septal ventricle (V*) via the right bundle branch retrogradely, without alteration of the immediate P1 or P2. The subsequent P1 was advanced by 15 ms, and the following P2 and QRS were advanced sequentially with rest of the tachycardia. CS indicates coronary sinus electrogram; HIS, His-bundle electrogram; LV, left ventricular electrogram; and RV, right ventricular electrogram.
During LPF-VT (Table). During SR, the earliest ventricular potentials after the LPF recorded via the MEC were located between segments 3 and 5.

Electrophysiological Maneuvers During LPF-VT
In all patients, attempts were made at delivery of PVCs and premature atrial stimuli during LPF-VT. Detailed information of the electrophysiological maneuvers had been presented in our previous article. In 12 patients with a stable TCL, PVCs from the RV septum reset tachycardia without affecting the immediate P1 and P2 (or P2 alone in patients without a recorded P1; Figure 3C). Entrainment of LPF-VT with progressive fusion during RV pacing with decreasing cycle length was performed and was observed in 7 patients. An example with schematic diagram

Figure 4. Example of overdrive pacing from the right midseptal ventricle with decreasing pacing cycle length during left posterior fascicular ventricular tachycardia (LPF-VT) in a patient with a tachycardia cycle length (TCL) of 315 ms and without a recorded P1 (patient 13). A. Surface ECG demonstrates pacing during tachycardia with progressive fusion of QRS morphology at pacing cycle lengths of 305, 300, 290, and 270 ms, respectively. B–D, Intracardiac electrograms during entrainment of LPF-VT at different pacing cycle lengths. The surface ECG and intracardiac electrogram were accelerated to the pacing rate, and the conduction sequence of recorded P2 via the multipolar electrode catheter (MEC) remained unchanged. After cessation of overdrive pacing, the first postpacing interval of P2 (Continued)
Macr oreentrant Loop in Fascicular Tachycardia has been shown in Figure 4. In the remaining 7 patients, attempts at burst RV pacing resulted in tachycardia termination. In 2 of 7 with successful entrainment and continuous resetting, the maneuver of PVC delivery could not be evaluated because of unstable TCL. Macroreentry involving ventricular myocardium was confirmed to be the mechanism in all patients by PVCs delivery or entrainment of LPF-VT with progressive fusion, or both.

Premature atrial stimuli or burst pacing delivered from the coronary sinus that captured the LPF antegradely without termination of tachycardia was observed in 4 patients. In other 10 patients, LPF could not be captured antegradely by atrial stimulation during LPF-VT.

Ablation
RF ablation was performed successfully in all patients.

In 9 patients with a recorded P1, RF energy was delivered at the distal P1 adjacent to the connection of P2. In 8 of the 9 patients, a trans-septal approach was adopted with recording by MEC during ablation, and a delayed P1 after ventricular potentials was observed during SR after successful RF ablation (Figure 5). In 1 of the 9 patients (patient 1), a
retrograde aortic approach was adopted without MEC recording during ablation because of difficulty with ablation via the trans-septal approach. In patient 8 (Figure 6), prolongation of the conduction time between the LPF and ventricle during SR and between P1 and P2 during LPF-VT were observed after initial ablation at a more distal portion of P1. A delayed P1 potential after ventricular potentials during SR was observed with noninducibility of LPF-VT after further ablation at a slightly more proximal site to the previous ablation. In this case, delayed P1 potentials could be eliminated by further ablation at the proximal portion of P1 during SR as was previously published.3

In 5 patients without a recorded P1, RF energy was delivered at the location of the earliest P2 during LPF-VT. A trans-septal approach was adopted in 4 patients, and a retrograde aortic approach was adopted in 1 patient (patient 10).

After successful ablation, in 9 of all patients, a QRS axis shift (5 rightward and 4 leftward) was observed. In 13 of all patients, there was the development of small Q-waves in the inferior leads noted after successful ablation. In 1 patient (patient 10)
with right bundle branch block before the study, the QRS axis and QRS morphology in the inferior leads remained unchanged. No significant prolongation of the QRS duration during SR was observed in any patients after successful ablation (Table).

Complication and Follow-Up
No study-related complications occurred in any patients. After 4.5±3.0 months of follow-up, no patients had recurrence of LPF-VT. Values are expressed as mean±SD.

Figure 6. Patient 8. A, Before ablation, the LPF (P2) was recorded preceding local ventricular potentials via the multipolar electrode catheter (MEC) during sinus rhythm (SR; left). During left posterior fascicular ventricular tachycardia (LPF-VT; right), P1 and P2 were observed with a more distal connection between P1 and P2 at the MEC. B, After initial ablation at the site of the connection, prolongation of the conduction time between the LPF and ventricle during SR (left) and between P1 and P2 during LPF-VT (right) were observed. C, Schematic diagram shows the possible network at the connection between P1 and P2. Before ablation, P2 was activated via the proximal channel of the connection during LPF-VT (left). After initial ablation (right), the proximal channel of the connection (open arrow), and the proximal exit of LPF to ventricular myocardium (solid arrow) were eliminated. The reentry circuit continued using the distal channel of connection between P1 and P2. AVN indicates atrioventricular node; CS, coronary sinus electrogram; HIS, His-bundle electrogram; LAF, left anterior fascicle; LV, left ventricular electrogram; RB, right bundle; and RV, right ventricular electrogram.

Discussion
The mechanism of LPF-VT is macroreentry involving the ventricular myocardium, a part of the LPF, a slow conduction zone, and in some cases, a P1 fiber with a slow conduction zone located between the proximal portion of recorded P1 and ventricular myocardium. The HV interval during LPF-VT is well correlated with the connection of P1 and the LPF or the level of the earliest LPF potential when P1 was not recorded.

Figure 7. Schematic diagram of the left posterior fascicular ventricular tachycardia (LPF-VT) reentry circuit. The reentry circuit of LPF-VT includes ventricular myocardium, a part of the LPF, a P1 fiber, and a slow conduction zone connecting the ventricular myocardium and proximal P1. A, In the cases with a recorded P1 and a more negative His-ventricular (HV) interval during LPF-VT, the P1 fiber is parallel and adjacent to the LPF, and the connection between P1 and the LPF (P2) is located at a more distal portion of the LPF. B, In the cases with a recorded P1 and a slightly negative HV interval during LPF-VT, the P1 fiber is parallel and adjacent to the LPF, and the connection between P1 and the LPF (P2) is located at the middle or proximal portion of the LPF. C, In the cases without a recorded P1, and HV interval that is slightly negative, the P1 fiber may be short in length or nonparallel in orientation to the LPF, or both. AVN indicates atrioventricular node; HIS, His-bundle electrogram; LAF, left anterior fascicle; and RB, right bundle.
Components of the Reentrant Circuit

In our study, P1 potentials were recorded in 9 of 14 patients (64%). To demonstrate the reentrant mechanism, 2 stimulation maneuvers from the RV septum were adopted: PVCs and burst pacing in an attempt at entrainment of LPF-VT at multiple pacing lengths. One or both of the maneuvers were performed successfully in all 14 patients with the following effect: (1) after advancement of the local ventricular potential by a PVC, without affecting the immediate P1 and P2 (or P2 alone in patients without a recorded P1), tachycardia was reset; and (2) entrainment of LPF-VT with progressive fusion was observed during RV pacing with decreasing cycle length. On the basis of the 2 pacing maneuvers, we confirmed that the mechanism of LPF-VT was macroreentry involving ventricular myocardium as we have previously demonstrated.3

Similar results after these maneuvers were observed in all 14 cases with or without a recorded P1. We speculate a reentry circuit composed of ventricular myocardium, a P1 fiber, a slow conduction zone connecting the ventricular myocardium and proximal P1, and a part of LPF as a bridge connecting the distal portion of P1 and the ventricular myocardium (Figure 7).

During LPF-VT, the site of earliest P2 indicates the site of connection between P1 and P2. The LPF (P2) was conducted bidirectionally from the connection site, distally to the ventricle and proximally to the His bundle, respectively (Figure 7). On the basis of this conduction sequence, the HV interval during LPF-VT reflects the site of connection between P1 and P2 along the LPF. In our study, the left ventricular septum was divided into 9 segments equally from the apex to the base near
the HV-bundle region. A correlation was observed between the HV interval and location of earliest P2 during LPF-VT, with a more negative HV interval during LPF-VT correlating with a more distal location of earliest P2 (Table and Figure 2).

The correlation between the HV interval during LPF-VT and a recorded P1 was also observed in our series. Patients with a clear P1 recording demonstrated a more negative HV interval during LPF-VT (Table). In 7 of 8 patients with a HV interval during tachycardia of \(-<20\) ms, there was a recorded P1 of \(>15\) mm length via the MEC. In one of these 8 patients (patient 2), there was no P1 recorded, and there seemed to be a relatively horizontal orientation of the heart (Figure 8). In 2 of 3 patients with a HV interval between \(-10\) and \(-20\) ms, P1 was recorded with a length of 9 mm via the MEC and the remaining 1 patient had no P1 recorded. In 3 patients with a HV interval \(>-10\) ms, there was no P1 recorded.

Anatomically, in most of the patients with a recorded P1, the P1 fiber was adjacent and parallel to the LPF. Some of the reasons that a P1 may not be recorded include that it is present with poor contact because of variation in P1 length, orientation, or septal anatomy.

One possibility is that the P1 fiber may be short in length or nonparallel in orientation with the LPF, or both. A nonparallel P1 fiber orientation may also result in a short recorded P1 length. In patient 11 (Figure 3), P1 potentials were recorded in 3 pairs of electrodes at the distal MEC, with a P1 length of 9 mm, shorter than most of the patients with a recorded P1. Some low amplitude, far-field potentials were observed at the mid-MEC region. In this case, the proximal portion of the P1 fiber may be nonparallel and slightly distant from the LPF (P2) and MEC.

A second possible reason may be poor contact between the MEC and ventricular septum, especially near the proximal region. At the proximal septum, this may be because of the aortic valve or chordae tendineae. In the patients with a slightly negative HV interval, the P1 fiber and the connection site of P1 and P2 may be located at a more proximal region of the left posterior ventricular septum. In these cases, because of the tilting effect of the aortic valve, P1 may be more difficult to record via the MEC from a retrograde aortic approach.

One other remaining reason may be anatomic variations in cardiac axis. A horizontal orientation of the heart may result in difficulty recording P1 as shown in patient 2 (Figure 8).

**Property of P1 and Slow Conduction Zone**

Thus far, the histopathologic properties of the P1 fiber and the slow conduction zone involved in the reentry circuit remain unknown. Previous studies have reported the conduction velocity of normal ventricular myocardium and bundle branches to be 0.3 to 1.0 and 2 to 4 mm/ms, respectively.\(^4\) In our 9 patients with a recorded P1, the velocity of recorded P1 conduction was measured, and it was slower than that of the LPF but similar to the conduction velocity of normal ventricular myocardium (Table). Suwa et al\(^5\) reported one LPF-VT case who was cured by surgical resection of the false tendon and left ventricular myocardium where the false tendon was attached. In this case, histopathologic examination revealed that the resected tendon contained numerous Purkinje fibers and a few monocytes possibly related to some degeneration, and Purkinje fibers ran longitudinally within the tendon and connected to the endocardial surface. We speculate that the P1 fiber is an insulated tissue containing ventricular myocardium and Purkinje fibers which connect with the LPF at its distal portion and the ventricular myocardium with some slow conduction zone at its proximal region.

Nogami et al\(^6\) considered that the slow conduction zone was the P1 fiber. In our study, the velocity of recorded P1 was invariable during either TCL alteration or ventricular pacing in most of patients. In our previously published report (patient 3),\(^3\) LPF-VT was induced by programmed stimulation introduced from the midnight ventricular septum. After S2 stimulation delivery, the S2–P1 interval increased from 255 to 320 ms with induction of LPF-VT, and the velocity of recorded P1 was consistent during the stimulation. We hypothesize a unique slow conduction zone exists between the proximal recorded P1 and ventricular myocardium. The essence of the slow conduction zone is unknown but may be some ventricular myocardium or abnormal Purkinje fiber with decremental conduction properties. Herkommer et al\(^7\) reported local areas of fibrosis or scar corresponded to the successful ablation sites of LPF-VT in 3 of 9 patients seen via magnetic resonance imaging. In certain LPF-VT cases, myocardial scar may represent an alternative reason for the presence of a slow conduction zone.

**Ablation Strategy**

Various ablation procedures with successful outcomes have been previously described including targeting the earliest Purkinje potential or diastolic potential during LPF-VT,\(^8\)\(^–\)\(^10\) linear ablation at the midleft ventricular septum during SR,\(^11\) or ablation at the middle segment of the LPF.\(^12\) However, none of these studies have clearly defined the relationship between the ablation target and the mechanism of LPF-VT. In fact, based on our model of the LPF-VT circuit, the earliest Purkinje potential targeted by previous studies was the earliest P2 (the connection of P1 and P2). In most patients with a more negative HV interval during LPF-VT, a P1 fiber may be adjacent and parallel to the LPF along the left posterior ventricular septum (Figure 7A), and a linear ablation at the midleft ventricular septum or ablation at the middle segment around the LPF will eliminate conduction through the P1 fiber. However, in patients without a recorded P1, usually with a slightly negative HV interval during LPF-VT, the P1 fiber may be short or nonparallel to the LPF, or there is no P1 involved in the circuit. In these patients, the successful ablation target should be the earliest P2 during LPF-VT, and the critical position should be confirmed carefully by adjusting the MEC along left posterior ventricular septum. Ablation targeted at different portions of the LPF away from the connection between P1 and P2 may be unsuccessful and result in changes in QRS morphology during tachycardia, as we detailed in our previous publication.\(^3\)

Recently, Zhan et al reported fragmented Purkinje potentials that could be recorded at the left ventricular septum during SR in LPF-VT patients and could be used for guiding successful ablation. We postulate that retrograde conduction through the P1 fiber during SR acts as the fragmented Purkinje potential after antegrade conduction of the LPF. In
our patients with a recorded P1 during LPF-VT, the P1 potential could not be recorded during SR via the MEC before ablation. A reasonable explanation was that the P1 fiber conducts retrogradely via the LPF, and the P1 potential is obscured in the local ventricular potentials.

In theory, as an antegrade limb and critical component in the LPF-VT circuit, ablation targeted at any portion of P1 should be successful and result in the elimination of the connection between P1 and P2. However, we suggest the optimal ablation target should be slightly proximal from the connection between P1 and P2. Anatomically, a complex network exists at the distal Purkinje system. In patients with a recorded P1 and a more negative HV interval during LPF-VT, the connection between P1 and P2 could also be relatively complex. Ablation targeted at a more distal portion of the P1 fiber may be difficult. As in patient 8 (Figure 6), after initial ablation at the distal portion of P1, the LPF-VT was inducible with a prolongation between P1 and P2 during LPF-VT and a slight change in QRS morphology. We hypothesize that a proximal connection between P1 and P2 was eliminated by the initial ablation, and there still remained a distal connection which allowed reentry to propagate. In this type of patient, the optimal ablation target should be slightly proximal to the P1–P2 connection site, where there usually is a clear separation between P1 and P2 potentials (Figure 5). After successful ablation, a delayed P1 after ventricular potentials should be observed because of the retrograde conduction block of the distal P1 with only antegrade conduction observed. Even though ablation targeted at any portion of P1 may be successful, a more proximal ablation of the P1 fiber should be avoided because of the risk of His-bundle injury or proximal LPF block.

Limitations

There are several limitations in the present study. (1) The conduction time and velocities of P1 and P2 were measured via the MEC based on the recording electrode distance. This measurement may not be accurate in some cases as the MEC may not be in good contact or not in parallel with the fascicle and/or P1 fiber; (2) a limitation of using the MEC should be noted, as the contact or the position of the catheter may not be in good contact or not in parallel with the fascicle and/or P1 fiber; (3) the follow-up period was relatively short (4.5±3.0 months), and a longer follow-up is needed.

Conclusion

The LPF-VT macroreentrant loop involves the ventricular myocardium, a part of the LPF, a slow conduction zone, and in certain cases, a specially conducting P1 fiber. The HV interval during LPF-VT correlates with multiple electrophysiological measures and is a useful marker for identification of the optimal ablation site.

Disclosures

None.

References

Macroreentrant Loop in Ventricular Tachycardia From the Left Posterior Fascicle: New Implications for Mapping and Ablation
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