Mechanisms of Posterior Fascicular Tachycardia
The Relationship Between High Frequency Potentials and the Ventricular Myocardium

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Case Description

Case 1
A 17-year-old man presented with a history of palpitations and surface ECG demonstrating wide QRS tachycardia (QRS duration=140 ms) with a right bundle branch block pattern and left-axis deviation (Figure 1A). The 12-lead ECG was normal during sinus rhythm.

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During electrophysiology study, mapping was performed using a 20 pole multielectrode catheter (MEC) via a retrograde aortic approach (Figure 1C). The baseline A–H and H–V intervals were 57 and 55 ms, respectively (Figure 1B, left). A wide QRS tachycardia identical to the clinical tachycardia was observed with cycle length of 320 ms, H–V interval of −20 ms and 2:1 retrograde conduction (Figure 1B, right). This was consistent with a diagnosis of fascicular ventricular tachycardia (FVT) with origin from the left posterior fascicle. Three distinct potentials could be recorded from the MEC during FVT: A) sharp inflection, high frequency potentials (P1) activated from midproximal to distal MEC; B) presystolic LPF potentials (P2) activated from distal to proximal MEC; and C) left-septal ventricular (V) potentials (Figure 1B, right).

Right ventricular programmed stimulation (S1S2, 400/300 ms) was used to induce FVT (Figure 2A). During S1 stimulation, P1 was recorded with a stable S1–P1 interval (S1–P1=255 ms) and the same conduction sequence as during tachycardia. With delivery of an extrastimulus, the S2–P1 interval increased to 320 ms, followed by P2 and V potentials, and induction of sustained FVT.

Additional pacing maneuvers were performed during tachycardia.

Delivery of Premature Ventricular Contraction
Premature ventricular contractions (PVCs) were introduced from the right midventricular septum beginning at 10 ms shorter than the tachycardia cycle length, until loss of ventricular capture or termination of tachycardia. A PVC advanced the immediate septal ventricle and His bundle via the right bundle branch retrogradely, without advancing the immediate P1 and P2, however, advancing the subsequent P1 by 10 ms. The following P2 and QRS were also advanced sequentially. The P1 and P2 intervals during tachycardia were 315 ms before the PVC. However, the P1 and P2 intervals both shortened to 305 ms after the PVC (Figure 2B).

Atrial Overdrive Pacing
Atrial overdrive pacing was delivered from the proximal coronary sinus at 300 ms (Figure 2C and 2D). The tachycardia converted to a narrow QRS morphology with the His bundle conducting antegradely (noted by a longer A–H interval and antegrade P2 conduction). At the onset of atrial pacing, the proximal portion of P2 was activated antegradely, whereas the distal P2 was still activated retrogradely. The septal ventricular potentials (V*, beat 4) at the mid MEC were advanced by antegrade P2 conduction without a change in cycle length at the distal MEC, and the subsequent P1 (beat 5) was also advanced (Figure 2C). Timing of P1 was simultaneous with the antegrade His bundle activation after the last atrial stimulus (beat 7) without termination of tachycardia (Figure 2D).

Radiofrequency ablation targeted at the distal P1 potentials rendered tachycardia noninducible.

Case 2
A 19-year-old man presented with a history of palpitations and surface ECG demonstrating wide QRS tachycardia (QRS duration=135 ms) with a right bundle branch block pattern and left-axis deviation. During sinus rhythm, the 12-lead ECG was normal.

During electrophysiology study, a 20 pole MEC was used via a retrograde aortic approach. The baseline A–H and H–V intervals were 58 ms and 60 ms, respectively (Figure 3A, left). A wide QRS tachycardia identical to the clinical tachycardia was observed with cycle length of 310 ms, H–V interval of −20 ms and 2:1 retrograde conduction (Figure 3A, right). Three distinct potentials P1, P2, and...
**Figure 1.** A, Surface ECG of tachycardia showed wide QRS complex (QRS duration=140 ms) with a right bundle branch block pattern and left-axis deviation. B, During sinus rhythm (left), A–H and H–V intervals were 57 and 55 ms, respectively. P2 was recorded with an antegrade conduction sequence from proximal to distal multielectrode catheter (MEC). The ventricular potentials at the mid MEC were earlier than proximal and distal ventricular potentials. During tachycardia (right), the H–V interval was −30 ms. P1 and P2 were recorded with opposite conduction sequences with P1 activated antegradely, and P2 activated retrogradely. P1 transition to P2 activation occurred toward the distal MEC. C, Right and left anterior oblique fluoroscopic images of catheter positions. The 20 pole MEC was positioned at the left septal ventricle via a retrograde aortic approach. CS indicates coronary sinus electrogram; HIS, His bundle electrogram; His, His bundle; LAF, left anterior fascicle; LAO, left anterior oblique; LPF, left posterior fascicle; LV, left ventricular electrogram; RAO, right anterior oblique; RB, right bundle; and RV, right ventricular electrogram.
Figure 2. A, Ventricular programmed stimulation from the septal right ventricle (S1S2, 400/300 ms) to induce FVT. During S1 stimulation, P1 was recorded with an antegrade conduction sequence and a S1–P1 interval of 255 ms. After the S2 stimulus delivery, the S2–P1 interval increased to 320 ms with induction of FVT. A schematic diagram shows the sequence of FVT induction. The site of possible slow conduction (wavy line) may be located at the proximal P1 region. P1 is the antegrade limb of the reentry circuit and activation of proximal P1 can occur either from retrograde conduction of P2 via the ventricular myocardium (solid arrow) or directly from ventricular myocardium at the distal MEC (open arrow). B, Premature ventricular contractions (PVC) introduced from the midright ventricular septum during tachycardia. A PVC advanced the immediate septal ventricle (V*) and His bundle via the right bundle branch retrogradely, without alteration of the immediate P1 or P2. The subsequent P1 was advanced by 10 ms and the following P2 and QRS were advanced sequentially. (Continued)
Figure 2 Continued. C and D, Atrial overdrive pacing from proximal CS catheter at a cycle length of 300 ms during FVT. C, Progressive antegrade capture of P2 and the Purkinje system was observed with narrowing of the surface QRS morphology. At beat 4, the septal ventricle (V*) at the mid-MEC was advanced by antegrade conduction of P2 without advancement of the ventricular potential at the distal MEC, and the subsequent P1 in beat 5 was advanced. D, The earliest ventricular activation at beat 7 activated by the antegrade conduction of P2 was observed at the mid-MEC. The subsequent P1 (beat 8) initiated FVT with retrograde P2 conduction. Schematic diagrams depict conduction patterns during atrial overdrive pacing. C shows that the LPB is gradually activated antegrady by atrial pacing and that each P1 activation follows previous ventricular activation. D depicts the initiation of tachycardia with the last paced beat 7 activating P1 antegradely (beat 8) and re-entry begins either with retrograde conduction of P2 via the ventricular myocardium (solid arrow) or directly from ventricular myocardium at the distal MEC (open arrow). AVN indicates atrioventricular node; CS, coronary sinus electrogram; FVT, fascicular ventricular tachycardia; HIS, His bundle electrogram; His, His bundle; LV, left ventricular electrogram; LAF, left anterior fascicle; LPF, left posterior fascicle; MEC, multielectrode catheter; RB, right bundle; and RV, right ventricular electrogram.
Figure 3. A. During sinus rhythm (left), A–H and H–V intervals were 58 and 60 ms, respectively. P2 was recorded with an antegrade conduction sequence from proximal to distal MEC. The earliest ventricular potential was located at the distal MEC. During tachycardia (right), the H–V interval was −20 ms with 2:1 retrograde conduction. P1 and P2 were recorded with opposite conduction (Continued)
Figure 3 Continued. Sequences with P1 activated antegradely and P2 activated retrogradely. P1 transition to P2 activation occurred toward the mid-distal MEC, simultaneous with the earliest recorded ventricular potential. B, During tachycardia, radiofrequency ablation was delivered at the location where the distal P1 fused with P2 and ventricular myocardium. After radiofrequency delivery, tachycardia was terminated with disconnection of the distal P1 potentials. Schematic diagrams show the potential reentrant circuit (left) and the termination of the tachycardia with antegrade block of distal P1 after ablation (right). C, After cessation of ablation, a delayed antegrade conduction of P1 was observed during either programmed ventricular pacing or sinus rhythm. Schematic diagrams show the P1 was antegrade activated during RVA pacing (left) and sinus rhythm (right). D, During ablation at the level of the proximal P1 potentials, automaticity arising from the P2 or local ventricle was noted with subsequent elimination of the P1 potentials. Schematic diagram shows the antegrade and retrograde block of the P1 fiber. ABL indicates ablation catheter; AVN, atrioventricular node; CS, coronary sinus electrogram; HIS, His bundle electrogram; His, His bundle; LAF, left anterior fascicle; LPF, left posterior fascicle; LV, left ventricular electrogram; MEC, multielectrode catheter; RB, right bundle; and RV, right ventricular electrogram.
V potentials of the left septum were recorded at the MEC (Figure 3A, right). During tachycardia, PVCs were delivered from the right ventricular apex. A PVC advanced the immediate septal ventricle without advancing the immediate P1 and P2, however, advancing the subsequent P1, and the following P2 and QRS were also advanced sequentially (not shown).

Ablation of the P1 Potentials
A large curve deflectable sheath and a 4-mm saline-irrigated ablation catheter (SJM) were used to ablate via a trans-septal approach. Radiofrequency ablation was delivered at the location where the distal P1 fused with P2 and ventricular myocardium. After radiofrequency delivery, tachycardia was terminated with disconnection of the distal P1 potentials (Figure 3B). After cessation of ablation, P1 potentials were recorded during either sinus rhythm or ventricular pacing, and delayed conduction to P1 was observed (Figure 3C). Further attempts were then made to ablate the proximal P1 during sinus rhythm. During ablation, automaticity arising from the P2 or local ventricle was noted with elimination of the P1 potentials (Figure 3D). Tachycardia could not be induced after ablation.

Case 3
A 33-year-old man presented with a history of palpitations and surface ECG demonstrating wide QRS tachycardia (QRS duration=125 ms) with a right bundle branch block pattern and left-axis deviation (Figure 4A). During sinus rhythm, the 12-lead ECG was normal.

A 20 pole MEC was used via a retrograde aortic approach during electrophysiology study.

The baseline A–H and H–V intervals were 75 and 60 ms, respectively (Figure 4B, left). A wide QRS tachycardia identical to the clinical tachycardia was observed with cycle length of 330 ms, H–V interval of ~5 ms and 1:1 retrograde conduction (Figure 4B, right). No manifest P1 potential could be recorded even with adjusting the MEC position along the septum. P2 potentials were observed along the MEC with earliest potentials seen at the midseptal left ventricle fluoroscopically.

Delivery of PVC
During tachycardia, PVCs were delivered from the midright ventricular septum. A PVC advanced the immediate proximal septal ventricle without advancing the immediate P2 and the septal ventricle from mid to distal; however, there was advancement of the subsequent P2 and QRS by 15 ms (Figure 4D).

Ventricular Overdrive Pacing
Ventricular overdrive pacing was delivered from the mid-right ventricular septum at different cycle lengths (Figure 4E). Entrainment of tachycardia and progressive fusion of QRS morphology were observed during decreasing pacing cycle length.

Atrial overdrive pacing failed to conduct through the His bundle antegradely. Radiofrequency ablation targeted at the earliest portion of P2 rendered tachycardia noninducible.

Discussion
Left posterior FVT is the most common type of fascicular tachycardia involving the left ventricular conduction system and may have multiple underlying mechanisms. Nogami et al and Morishima et al suggested that P2 may be a bystander of the FVT, and the left ventricular myocardium and P1 may comprise the reentry circuit.

In the first 2 cases, 2 distinct sharp potentials (P1 and P2) were recorded during tachycardia with a negative H–V interval (H–V= ~30 and ~20 ms, respectively). Tachycardia demonstrated a negative H–V interval indicating activation of the P2 at the mid to low LPF where P1 inserted antegradely (Figure 1B, right; Figure 3A, right). In the third case, the H–V interval during tachycardia was less negative (~5 ms) suggestive of a more proximal initial activation of the LPF (Figure 4B, right).

During sinus rhythm in case 1, P2 conducted with an antegrade activation sequence was recorded preceding the onset of local V potential without recording of P1 (Figure 1B, left). Earliest local ventricular activation appeared to be at the middle portion of the MEC suggesting a connection of LPF to ventricular myocardium at the midseptal location. Right ventricular programmed stimulation was performed, and P1 was noted following the local V potential with antegrade conduction and a stable S1–P1 interval (S1–P1 = 255 ms). We also noted that P1 was not conducted antegradely to the distal MEC during ventricular stimulation suggestive of conduction block at the distal P1 or concealed retrograde conduction. With extrastimulus delivery, the S2–P1 interval increased by 65 ms (S2–P1=320 ms), and tachycardia was induced with retrograde P2 conduction from distal to proximal MEC after P1 conduction from proximal to distal MEC (Figure 2A).

During tachycardia, PVCs were introduced from the right midseptal ventricle. The local V potentials were advanced without advancement of the immediate P1 or P2, and the subsequent P1 and P2 were advanced by 10 ms (Figure 2B). These findings confirm the involvement of ventricular myocardium as part of the reentry circuit.

With atrial overdrive pacing from the coronary sinus during tachycardia, gradual antegrade activation of the His-Purkinje system was noted after the second paced beat (Figure 2C and 2D). The fourth beat demonstrates LPF (P2) activation from proximal to mid poles and slightly advanced local ventricular activation at the mid MEC poles. The distal retrograde activation of P2 and timing of ventricular activation at the distal MEC remained unaffected; however, the subsequent P1 (beat 5) was advanced. The proximal P1 must be activated by the mid portion of the LPF (P2) via ventricular myocardium and a slow conduction zone. After the last atrial stimulus, the timing of antegrade P1 (beat 7) was simultaneous with the His bundle potential which indicated that P1 was activated from the previous ventricular complex (beat 6). The antegrade activation of P2 (beat 7) from the last atrial stimulus conducts through ventricular myocardium that was connected to the subsequent P1 (beat 8) fiber with continuation of tachycardia. This suggests the involvement of 2 distinct reentry circuits in FVT. Both involve a slow conduction zone linked to the proximal P1 region. However, one circuit may include only the ventricular myocardium and the proximal P1 fiber with retrograde P2 conduction acting as a passive bystander. An alternative circuit utilizing
Figure 4. A, Surface ECG of tachycardia showed wide QRS complex (QRS duration=125 ms) with a right bundle branch block pattern and left-axis deviation. B, During sinus rhythm (left), A–H and H–V intervals were 75 and 60 ms, respectively. P2 was recorded with an antegrade conduction sequence from proximal to distal MEC. The earliest ventricular potential was located at the mid-distal MEC. During tachycardia (right), the H–V interval was −5 ms with 1:1 retrograde conduction. P2 potentials were recorded with a retrograde conduction sequence from mid-distal to proximal MEC. C, Right and left anterior oblique fluoroscopic images of catheter positions. D, Premature ventricular contractions (PVC) introduced from the midright ventricular septum during tachycardia. A PVC (Induced by the second stimulus) advanced the immediate high septal ventricle (V*) without alteration of the immediate P2. The subsequent P2 is advanced by 15 ms, and the following QRS was advanced sequentially. Schematic diagram in the right depicts the potential reentrant circuit of the tachycardia. E, Overdrive pacing from the right midseptal ventricle during tachycardia at cycle lengths of 305, 300, 290, and 270 ms. Surface ECG demonstrates entrainment of tachycardia with progressive fusion of QRS morphology during decreasing pacing cycle length. F, Theoretically, ablation targeting the distal or proximal LPF away from the optimal target may result in different changes in QRS morphology during tachycardia. Schematic diagram shows the potential mechanisms. Left, the reentrant circuit may involve both the right bundle branch and the left anterior fascicle after ablating the distal part of the LPF away from the target, a narrow QRS morphology may be observed (Continued)
Figure 4 Continued. with positive H–V interval; Right, a wider QRS morphology with left-axis deviation and more negative H–V interval may emerge after ablating the proximal part of the LPF away from the target because of loss of antegrade conduction from the right bundle branch and left anterior fascicle. AVN indicates atrioventricular node; CS, coronary sinus electrogram; HIS, His bundle electrogram; His, His bundle; LAF, left anterior fascicle; LAO, left anterior oblique; LPF, left posterior fascicle; LV, left ventricular electrogram; MEC, multielectrode catheter; RAO, right anterior oblique; RB, right bundle; RV, right ventricular electrogram; and SS, overdrive pacing cycle length.
Cardiac, the successful ablation target should be the earliest H–V interval and no manifest recorded P1 during tachycardia, the successful ablation target should be the P1 potentials. However, the ideal level of P1 for ablation may be at a more distal site of P1 to avoid possible delayed conduction at the more proximal LPF.

In the second case, a similar conduction sequence of P1 and P2 was observed during tachycardia (Figure 3A, right). However, we observed that there was a more distal connection between the LPF (P2) and ventricular myocardium during sinus rhythm (Figure 3A, left). Before ablation, P1 was retrogradely activated via the distal LPF, and P1 potentials were fused with V potentials at the MEC during sinus rhythm. After ablation at the site where distal P1 connected with P2 and ventricular myocardium, P1 was recorded during either sinus rhythm or right ventricular apex pacing, with significant delayed antegrade conduction following ventricular potentials (Figure 3C). After ablation of the proximal P1 region, total elimination of the proximal P1 activation from ventricular myocardium was observed (Figure 3D). This is evidence that P1 is critical to the reentry circuit and that ablation is feasible by either targeting the proximal or distal P1 region.

In contrast to the above 2 cases, during tachycardia, the third case had a slightly negative H–V interval (~5 ms), a relatively narrow QRS duration, and no manifest P1 could be recorded. The response to PVC delivery and the progressive fusion of QRS morphology noted during entrainment with decreasing pacing cycle length suggests a re-entrant mechanism of tachycardia with ventricular myocardium as part of the reentrant circuit, similar to those cases with a manifest recorded P1 and more negative H–V interval during tachycardia. The earliest LPF potentials were observed at the location of the midseptal left ventricle, and successful ablation was targeted at this area. We hypothesize a P1 fiber with a short length anatomically or nonparallel in orientation to the LPF and inserts into the LPF at the mid septum, with a slow conduction zone linked to the proximal P1 region. The potential reentrant circuit is shown as a schematic diagram (Figure 4D, right).

Catheter ablation guided by the relationship of P1 and P2 potentials recorded by the MEC may be useful for evaluation of the underlying substrate of FVT. For the type of FVT with a more negative H–V interval and manifest recorded P1 during tachycardia, the successful ablation target should be the P1 potentials. However, the ideal level of P1 for ablation may be at a more distal site of P1 to avoid possible delayed conduction at the more proximal LPF.

For the other type of FVT with a slightly negative H–V interval and no manifest recorded P1 during tachycardia, the successful ablation target should be the earliest P2 potential. Theoretically, ablation targeting the distal or proximal LPF away from the optimal target may result in different changes in QRS morphology during tachycardia. The schematic diagram (Figure 4F) shows the potential mechanisms of these alterations. A narrow QRS morphology with positive H–V interval may be observed after ablating the distal part of the LPF away from the target, because the reentrant circuit may involve both the right bundle branch and the left anterior bundle branch after the ablation (Figure 4F, left). Contrarily, a wider QRS morphology with left-axis deviation and more negative H–V interval may emerge after ablating the proximal part of the LPF away from the target (Figure 4F, right).

Conclusions

It is possible that left posterior FVT may involve more than one form of reentrant circuit. We describe 2 types of FVT characterized by H–V interval. The first type has a more negative H–V interval with manifest recorded P1. The second type has a slightly negative H–V interval without recorded P1. Re-entry was the common mechanism of the 2 types of FVT. The reentrant circuit consists of ventricular myocardium, a Purkinje fiber (P1), a slow conduction zone exists at the proximal P1 region, and a part of LPF (P2) that connects distal P1 and ventricular myocardium. This unique slow conduction region may be part of ventricular myocardium, proximal P1, or other Purkinje fibers, which demonstrate delayed conduction properties and may represent a substrate for this arrhythmia. The successful ablation target should be the P1 potentials for the type of FVT with recorded P1, whereas the earliest P2 potential should be the successful target for the type of FVT without manifest recorded P1. Ablation at the different positions of P2 away from optimal target may change the reentrant circuit and the QRS morphology during tachycardia.

Disclosures

None.

References


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