Catheter Ablation of Fascicular Ventricular Tachycardia: Long Term Clinical Outcomes and Mechanisms of Recurrence

Running title: Liu et al.; Fascicular ventricular tachycardia ablation

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Abstract:

Background - Fascicular ventricular tachycardia (FVT) is a common form of sustained idiopathic left ventricular tachycardia with an Asian preponderance. This study aimed to prospectively investigate long term clinical outcomes of patients undergoing ablation of FVT and identify predictors of arrhythmia recurrence.

Methods and Results - Consecutive patients undergoing FVT ablation at a single tertiary center were enrolled. Activation mapping was performed to identify the earliest pre-systolic Purkinje potential (PP) during FVT which was targeted by radiofrequency ablation. Follow-up with clinic visits, ECG and Holter monitoring was performed at least 6 monthly. 120 consecutive patients (mean age 29.3 ± 12.7 years old, 82% male, all patients with normal ejection fraction) were enrolled. FVT involved left posterior fascicle (LPFVT) and left anterior fascicle (LAFVT) in 118 and 2 subjects respectively. VT was non-inducible in 3 patients and ablation was acutely successful in 117 patients. With a median follow up of 55.7 months, VT of a similar ECG morphology recurred in 17 patients and repeat procedure confirmed FVT recurrence involving the same fascicle. Shorter VT cycle length was the only significant predictor of FVT recurrence (P = 0.03). Six other patients developed new-onset upper septal FVT which was successfully ablated.

Conclusions - Ablation of FVT guided by activation mapping is associated with a single procedural success rate without the use of anti-arrhythmic drugs of 80.3%. Arrhythmia recurrences after an initially successful ablation were caused by recurrent FVT involving the same fascicle in two-thirds of patients or new onset of upper septal FVT in the remainder.

Key words: ventricular tachycardia, radiofrequency, idiopathic left ventricular tachycardia, fascicular ventricular tachycardia, outcomes, radiofrequency ablation
Since the first description of fascicular ventricular tachycardia (FVT) involving the left posterior fascicle (LPF) in 3 patients by Zipes et al in 1979, our understanding of this common form of idiopathic left ventricular tachycardia has expanded particularly over the last 2 decades.

Through detailed electrophysiological studies, FVT has been determined to be re-entrant in mechanism, can be responsive to verapamil, involves various branches of Purkinje network emanating from left fascicles with differential conduction properties participating in the tachycardia and can be successfully treated by localized ablation in the left ventricular septum. Based on reports of small single-center cohorts undergoing ablation, acute success rates are generally high although limited data exist about electrophysiological findings and clinical outcomes in the few patients that do develop arrhythmia recurrence. More recently, case reports have described FVT patients re-presenting years after ablation with different variants of FVT.

This study aimed to prospectively investigate long term clinical outcomes of a large cohort of patients undergoing catheter ablation of FVT in a regional referral center in China, analyze mechanisms of arrhythmia recurrence and identify electrophysiological or clinical predictors for FVT recurrence.

**Methods**

**Population**

The study was approved by an institutional ethics committee and that all subjects gave informed consent. From January 2008 to December 2012, consecutive patients undergoing clinically indicated electrophysiological studies (EPS) and ablation for FVT at the Affiliated Hospital of...
Nanjing Medical University, China were enrolled into a prospective clinical database. All patients fulfilled all of the following criteria:

a. Symptomatic sustained ventricular tachycardia

b. Documented clinical VT with right bundle branch block-like morphology

c. Absence of ischemic or structural heart disease based on transthoracic echocardiography and clinical evaluation

**Electrophysiological Study**

Antiarrhythmic medications were withdrawn for at least a period of 5 half-lives. After written informed consent was obtained, the electrophysiological study was performed in the fasting state under conscious sedation. Six French quadripolar catheters were positioned at the bundle of His and right ventricular (RV) apex. AH, HV intervals and ECG frontal axis during sinus rhythm were recorded at baseline and at the end of the procedure. Particular attention was paid to the development of fascicular block after ablation. Specifically, LPF block was defined as frontal axis between 90° to 180°, rS pattern in leads I and aVL, qR pattern in leads III and aVF and QRS duration of less than 120ms as per the most recent international guidelines.17

In patients without spontaneous VT, programmed stimulation was performed from the RV apex, RV outflow tract and right atrium at drive trains of two different cycle lengths (500 or 400 ms, and 330 ms), with up to three extra stimuli, with and without the use of isoproterenol (1-3ug/min). If necessary, incremental burst pacing up to cycle lengths of 280 ms and 200ms in the RV and atrium respectively was performed for VT induction. The filter setting for the bipolar intracardiac electrograms was 30 to 500 Hz. Differential diagnoses such as a typical atrioventricular re-entrant tachycardia and bundle branch re-entry were excluded by established deductive criteria during EPS in combination with diagnostic maneuvers, activation mapping and
entrainment.

**Activation Mapping**

A 7-French deflectable, non-irrigated, quadripolar catheter with a 4-mm distal electrode, an embedded thermistor and 2-5-2 mm inter-electrode spacing (Cordis Webster Inc., Diamond Bar, CA, USA or EP Technologies Inc., San Jose, CA, USA) was introduced retrogradely for mapping and ablation. If a three-dimensional mapping system (Ensite NavX System, St Jude Medical Inc., St Paul, MN, USA) was used, endocardial LV geometry was first created. Activation mapping during VT was performed and specifically, sites with His or Purkinje potentials associated with local ventricular electrograms, extending from left basal septal sites with bundle of His recordings to most apical sites with pre-systolic Purkinje potentials (apical PP), were marked on the LV geometry (Figure 1). During cases where only conventional mapping was performed, sites with the earliest pre-systolic Purkinje potential during VT was identified (Figure 2). Whenever possible, entrainment was performed to confirm participation of the targeted site in VT. If VT was not inducible, catheter ablation was not performed.

**Radiofrequency Ablation**

Temperature-controlled radiofrequency energy was delivered at the site with the earliest pre-systolic Purkinje potential during VT. The power output was titrated to as high as 35W to achieve a target temperature of 40 to 60°C for up to 120 seconds. The ablation procedure was considered successful if FVT terminated during ablation and/or any VT was not inducible 30 minutes after ablation despite repeat programmed stimulation and isoproterenol infusion.

**Follow-Up**

After the procedure, continuous ECG monitoring was performed for 24 hours. All anti-arrhythmic agents were not restarted in the absence of post-operative arrhythmia recurrence.
All patients were reviewed in clinic monthly after the procedure for the first three months, and then followed up every 6 months. A 12-lead ECG and 24-hour Holter monitoring were performed at every clinic visit. Arrhythmia recurrence was defined as symptomatic recurrence with ECG documentation of recurrent VT on either the 12-lead ECG and/or Holter monitoring.

**Statistical Analysis**

Continuous variables and categorical variables were described by means ± standard deviation and percentages, respectively. An independent samples t-test and paired samples t test were performed for comparisons of the corresponding parameters and paired data. Chi-square test was used for comparison of categorical data. To determine the association between procedural parameters and VT recurrence, univariable and multivariable Cox proportional regression analyses were performed and results were expressed as hazard ratios (HRs) with 95% confidence intervals (CIs). P-value of less than 0.05 was considered as statistically significant.

**Results**

**Patient Characteristics**

From January 2008 to December 2012, 120 consecutive patients underwent EPS and ablation for FVT, refractory to a mean of 1.3 ± 0.9 anti-arrhythmic medications (Figure 3). 56 patients received intravenous verapamil during episodes of VT, prior to EPS which terminated VT in all patients. Two patients had previously undergone unsuccessful ablation attempts at their referring hospitals. The mean age was 29.3 ± 12.7 (range from 7 to 69) years old and they were predominantly male (81.7%). The median duration of symptoms prior to EPS was 14 (25th to 75th percentile - 3 to 63 months) months. All but 3 patients reported palpitations whilst the remaining 3 experienced presyncope or syncope. Echocardiography confirmed structurally normal hearts in all cases with a mean left ventricular ejection fraction of 65.3 ± 4.5 %.
Electrophysiological Studies

In all patients, pre-procedural ECGs demonstrated sinus rhythm with normal axes and QRS durations. AH (80.4 ± 15.6 ms) and HV (44.4 ± 8.2 ms) intervals were within normal limits before ablation. VT was not inducible in 3 patients whom therefore did not proceed to catheter ablation. In the remaining 117 patients, monomorphic VT involved the left posterior fascicle (LPFVT) (RBBB morphology with superior axis; mean VT cycle length 362.1 ± 79.4 ms; mean QRS duration 123.9 ± 14.0 ms and HV intervals during VT -7.5 ± 2.1 ms) and the left anterior fascicle (LAFVT) (RBBB morphology with right inferior axis; mean VT cycle length 364.0 ± 26.9 ms; mean QRS duration 124.6 ± 12.0 ms and HV intervals during VT -11.9 ± 13.0 ms) in 115 and 2 patients respectively. In all patients, bundle of His was activated retrogradely during VT (often with VA dissociation), ruling out alternative diagnosis such as bundle branch re-entry and supraventricular tachycardia with aberrancy.

Mapping and Ablation

The mean earliest pre-systolic Purkinje potential preceded the onset of QRS during VT by 30.1 ± 9.3 ms (Figure 2). A 3D mapping system was utilized in 71.6% of cases. In LPFVT subjects, the earliest pre-systolic Purkinje potential was located 61.3 ± 23.7 mm (range 40 to 116 mm) from the most basal septal left ventricular site where a His potential could be measured or 69.0 ± 10.8 % of the course which extended along the septum from the bundle of His to the end of the LPF (His-RF/His-apical PP) (Figure 1). Similarly for patients with LAFVT, the earliest pre-systolic Purkinje potential was located 61.0 ± 8.5 mm (55 to 67 mm) from the most basal LV site where a His potential could be measured and 68.5 ± 5.4 % of the course that extended along the septum from the bundle of His to the end of the left anterior fascicle. Entrainment was performed successfully in 20% of cases, demonstrating concealed fusion and participation of the
ablation site in FVT.

Ablation was acutely successful by targeting the earliest pre-systolic Purkinje potential in all 117 patients with inducible FVT, with non-inducibility of any VT despite programmed stimulation and isoproterenol infusion achieved as the electrophysiological endpoint. At the end of the procedure, HV intervals during sinus rhythm remained unchanged in all patients.

ECG changes during sinus rhythm were seen in the majority of patients who underwent ablation of LPFVT. 23 (20%) subjects developed new onset LPF block whilst 67 (58.3%) patients exhibited rightward shift in their frontal axis compared to baseline (frontal axis 31° to 84°). The axis remained unchanged in the remaining 25 (21.7%) patients. Development of LPF block was associated with more proximal ablations (His-RF / His-apical PP ratio of 0.57 ± 0.13 versus 0.77 ± 0.10 in patients whose frontal axis remained unchanged, $P = 0.03$) (Table 1). ECG remained unchanged in the 2 LAFVT patients after ablation. No complications occurred during the index procedure.

**Arrhythmia Recurrences During Follow up and Repeat Ablation**

With a median follow up of 55.7 months (25th to 75th percentile: 38.9 to 71.7 months), VT recurred in 23 LPFVT patients. There was no recurrence of LAFVT. The median time to recurrence was 2.5 months (25th to 75th percentile: 1.5 to 17 months) after the index ablation. In 17 patients, the recurrent VT had ECG morphology similar to the clinical LPFVT targeted at the index ablation, although with longer tachycardia cycle lengths and shorter VH intervals. (Table 2) All consented to repeat ablation. The VT induced during the repeat procedure had similar electrophysiological parameters including cycle length, HV intervals and prematurity of the earliest PP in relation to QRS onset. The earliest PP during FVT was again mapped to the vicinity of the LFP, to a location in close proximity to the ablation site of the index procedure.
(Figures 4A and 4B). Ablation was acutely successful again in all 17 patients and FVT has not recurred during a median follow-up of 40.3 months. (25\textsuperscript{th} to 75\textsuperscript{th} percentile - 25.9 to 55.4 months). The development of LPF block did not confer any protection from recurrent FVT (Table 1 and 3).

Interestingly, in the remaining 6 patients, recurrent VT exhibited a different ECG morphology, with narrower QRS and variable inferior axis (Figure 5, Supplemental Figure 2) (Table 2). Five out of these 6 patients had developed new onset LPF block after the first ablation. Only 3 patients agreed to undergo a repeat EPS. During the second ablation procedure, the LPF was activated in an anterograde direction with a resultant positive HV interval that was shorter during VT compared to sinus rhythm. Electrophysiological studies and activation mapping confirmed the diagnosis of upper septal VT. Concealed entrainment was only successful in 1 patient with a PPI (320 ms) minus tachycardia cycle length (315ms) of 5 ms (Supplementary Figure 3). The lower turnaround of the upper septal VT circuit, as marked by the site with the most premature diastolic potential was targeted. The ablation site was significantly more basal compared to the index ablation (Table 2, Figures 4C, 4D and 6). Radiofrequency ablation was initiated cautiously at 10 watts, gradually increased to a maximum of 20 watts and was acutely successful in all 3 patients without any AV nodal injury or ECG changes. All 3 patients remain free from recurrent arrhythmias after a median follow-up of 46.1 months.

Three patients in whom VT was not inducible at the first procedure did not return for repeat EPS. Therefore the single procedural success rate of FVT ablation without the need for anti-arrhythmic agents in an unselected cohort of patients is 80.3% (94/117). Allowing for repeat procedures, 97.4% (114/117) patients were free from recurrent VT during a median follow-up of 55.7 months.
Predictors of ILVT Recurrence

Multivariable analysis was performed to identify risk factors for LPFVT recurrence after an acutely successful ablation procedure (Table 3). Only faster VT cycle lengths predicted LPFVT recurrence. (VT cycle length in patients with and without LPFVT recurrence, 307.7 ± 49.6 ms vs. 361.7 ± 62.7, \( P = 0.03 \)). Development of LPF block did not protect against LPFVT recurrence.

Discussion

Major Findings

Our strategy of targeting the most premature pre-systolic Purkinje potential with limited ablation during ongoing FVT is associated with high acute procedural success (100%) although approximately 20% of patients subsequently developed arrhythmia recurrence. Two-thirds of FVT recurrence was caused by the re-appearance of same FVT that was treated during the index ablation whereas the rest was caused by new-onset upper septal VT. Shorter tachycardia cycle length was the sole predictor of LPFVT recurrence. The development of new LPF block was associated with more basal ablations and did not protect against LPFVT recurrence.

Identifying Targets for FVT Ablation

The re-entrant pathway of LPFVT is understood to consist of a decrementally conducting, abnormal Purkinje fiber located in the vicinity of the distal third of the left posterior fascicle-Purkinje network that gives rise to a diastolic potential (often termed P1 potential) serving as the anterograde limb and a faster conducting, “normal” fiber which accounts for the pre-systolic Purkinje potential (also known as P2) serving as the retrograde limb. It is highly probable that ventricular myocardium serves as the bridge between the anterograde and retrograde limbs.\(^3\),\(^10\) When activation mapping can be performed during ongoing VT, LPFVT can be successfully ablated by targeting either diastolic (P1) or the earliest PP (P2 potentials), the
latter representing the lower turnaround of the LPFVT circuit.\textsuperscript{8, 10} The advantages of targeting the earliest PP include their more apical locations, reducing the risk of atrioventricular nodal or bundle branch injury and need for fewer radiofrequency energy applications.\textsuperscript{18} Furthermore, diastolic (P1) potentials may not be recorded in all patients.\textsuperscript{8} Although different smaller series have selected varying ablation targets namely P1, earliest PP or even the exit site (site with earliest ventricular activation), acute success rates are consistently high, suggesting that the reentrant FVT can be interrupted at multiple points along the re-entrant circuit.\textsuperscript{8, 9, 11, 18} Consistent with these prior reports, our pre-defined strategy of targeting the earliest PP during LAFVT or LPFVT was acutely successful in all patients.

**Electrophysiological Endpoints of FVT Ablation**

Locating the optimal site for ablation becomes more difficult when FVT is difficult to induce or is non-sustained. Numerous ablation strategies performed during sinus rhythm to overcome this hurdle have been reported. Purkinje potentials visible after the QRS complex, which may be characteristic of delayed retrograde activation of abnormal Purkinje fibers that act as the anterograde limb of LPFVT, have been targeted during sinus rhythm. Whilst these potentials seem unique to FVT patients compared to a small group of control patients without FVT, such electrograms are recordable over a sizeable area of the septum and can be detected remotely in the left anterior fascicle Purkinje network in patients presenting with LPFVT.\textsuperscript{4, 10} Thus, undertaking such a strategy would result in a substantial septal area being ablated with the risk of injury to the conducting system. A more anatomically guided approach of placing 7 to 15 ablation lesions in a linear pattern, perpendicular to the long axis of the ventricle approximately midway from the base to the apex in the region of the mid to mid-inferior septum, guided by the presence of Purkinje potentials and pace mapping was reported to be effective in 6 patients with
non-sustained LPFVT.\textsuperscript{6} Consistent with our results that the successful ablation site is located on average 69% of the septal course extending from the bundle of His to the end of the left fascicle, it is highly plausible that such lesion sets could transect the lower turnaround of the LPFVT circuit, preventing arrhythmia recurrence. Finally, a few groups have suggested creation of partial or complete LPF block as a reproducible, easily demonstrable electrophysiological endpoint for LPFVT ablation.\textsuperscript{7, 14, 19} In our study, the development of complete LPF block was associated with more basally located ablations. Furthermore, the formation of LPF block did not reduce the risk of LPFVT recurrence. These findings, in conjunction with other observations that selective capture of the LPF during LPFVT did not affect tachycardia cycle length; LPFVT can still develop in patients with pre-existing LPF block and the vast majority of patients remained arrhythmia-free after LPFVT ablation without developing new onset LPF block; would suggest that the LPF does not participate in LPFVT circuit.\textsuperscript{5, 13, 20} Hence rightward shift in the ECG axis is most likely due to inadvertent collateral damage to the LPF, particularly when ablation is delivered more basally. Therefore, our results would imply that creation of LPF block is unlikely to be an effective electrophysiological endpoint for FVT ablation, although this hypothesis was not tested directly in the experimental design. It is also noteworthy that 5 out of 6 patients who developed arrhythmia recurrence with upper septal VT developed LPF block after their first index. It is speculative but plausible that the inadvertent creation of sites of slow conduction within the Purkinje network could be pro-arrhythmic, promoting re-entry along different septal circuits.

**Predictors of FVT Recurrence**

In an earlier study of 79 patients whom underwent ablation of idiopathic VT which included 40 FVT patients, 9 patients (5 FVT subjects) developed VT recurrence.\textsuperscript{12} The only significant
predictor of arrhythmia recurrence was the endocardial activation time at the successful ablation. In our study with a larger sample size, only faster tachycardia cycle length but not prematurity of the local PP potential, predicted FVT recurrence. More rapid ventricular contractions may lead to reduced catheter stability and tissue contact, impairing ablation lesion size and quality.

Subsequent recovery of conduction between the anterograde and retrograde limbs of the LPFVT circuit allowed for VT recurrence. Hence during the repeat procedure, the successful ablation site was once again mapped to a similar location and additional ablation rendered once again FVT non-inducible.

**Potential for Multi-fascicular VT**

Upper septal VT is the most uncommon variant of fascicular VT, characterized by a tachycardia circuit that utilizes parts of the LPF as the anterograde limb (with simultaneous bystander activation of the right bundle and left anterior fascicle to produce a narrow QRS) and septal Purkinje fiber or auxiliary fascicle as the retrograde limb.\(^{15,16}\) Such VT has also been described as fast-slow fascicular VT whereby the slower conducting septal fascicle serves as the retrograde limb as opposed to the fast-slow fascicular VT seen in LAFVT and LPFVT whereby anterograde limb is the slower conducting abnormal Purkinje fibre.\(^{21}\) It has been reported by Nishiuchi *et al.* recently that upper septal VT was responsible for arrhythmia recurrence in a patient who had previously undergone successful ablation of LPFVT, similar to 6 patients in our cohort.\(^{15}\)

However, upper septal VT can also present as the *de novo* arrhythmia.\(^ {16}\) Unlike LPFVT, it is important to target the earliest diastolic potential originating from the septal fascicle which marks the lower turnaround of the upper septal VT circuit. Ablating the earliest pre-systolic Purkinje potentials around the anterograde limb, where concealed entrainment can be achieved, could result in fascicular or bundle branch block. Moreover VT can remain inducible with the
tachycardia switching to revolve around the left anterior fascicle as upper septal VT with a
different ECG morphology or around the right bundle branch to form bundle-branch reentrant
VT.  

Limitations

Only patients with inducible FVT underwent ablation and therefore our findings during
activation mapping would not be applicable for patients with non-inducible VT. Intravenous
verapamil was not administered during EPS to avoid rendering FVT non-inducible. Therefore the
possibility of focal Purkinje VT being the diagnosis in a few cases cannot be excluded.

Conclusions

Ablation of FVT guided by activation mapping is associated with a single procedural success
rate without the use of anti-arrhythmic drugs of 80.3% which increases to 97.4% with repeated
ablations. Only faster tachycardia cycle length but not development of new LPF block predicted
LFPVT recurrence. Arrhythmia recurrences after an initially successful ablation were caused by
recurrent FVT involving the same fascicle in two-thirds of patients and new onset of upper septal
VT in the remainder.

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Conflict of Interest Disclosures: None.

References:


Table 1: Procedural Details according to Changes in ECG Frontal Axis after Successful Left Posterior Fascicular Ventricular Tachycardia Ablation

<table>
<thead>
<tr>
<th></th>
<th>New onset LPFB (n = 23)</th>
<th>Rightward shift in frontal axis (n = 67)</th>
<th>Axis unchanged (n = 25)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedural time (min)</td>
<td>138.4 ± 54.8</td>
<td>138.4 ± 54.8</td>
<td>122.4 ± 57.3</td>
<td>0.66</td>
</tr>
<tr>
<td>Fluoroscopy time (min)</td>
<td>12.9 ± 5.5</td>
<td>15.9 ± 8.3</td>
<td>11.0 ± 4.5</td>
<td>0.83</td>
</tr>
<tr>
<td>Fluoroscopy dose (cym²)</td>
<td>63.5 ± 62.9</td>
<td>90.0 ± 24.5</td>
<td>53.4 ± 21.8</td>
<td>0.37</td>
</tr>
<tr>
<td>Axis before ablation (°)</td>
<td>59.4 ± 21.8</td>
<td>53.8 ± 20.7</td>
<td>47.3 ± 24.5</td>
<td>0.33</td>
</tr>
<tr>
<td>Axis after ablation (°)</td>
<td>101.8 ± 13.5</td>
<td>64.1 ± 16.1</td>
<td>44.9 ± 26.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>AH during SR (ms)</td>
<td>75.0 ± 32.5</td>
<td>77.0 ± 15.0</td>
<td>83.6 ± 12.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>HV during SR (ms)</td>
<td>46.0 ± 14.1</td>
<td>44.8 ± 7.9</td>
<td>45 ± 9.8</td>
<td>0.98</td>
</tr>
<tr>
<td>VT cycle length (ms)</td>
<td>382.0 ± 70.7</td>
<td>347.3 ± 84.3</td>
<td>379.2 ± 77.1</td>
<td>0.72</td>
</tr>
<tr>
<td>VH interval during VT (ms)</td>
<td>-10.7 ± 9.3</td>
<td>-13.5 ± 9.4</td>
<td>-14.6 ± 3.9</td>
<td>0.31</td>
</tr>
<tr>
<td>His-RF site/His-apical PP(%)</td>
<td>0.57 ± 0.13</td>
<td>0.61 ± 0.16</td>
<td>0.77 ± 0.10</td>
<td>0.03</td>
</tr>
<tr>
<td>Earliest PP-QRS interval during VT (ms)</td>
<td>31.4 ± 5.4</td>
<td>30.5 ± 8.3</td>
<td>30.7 ± 9.2</td>
<td>0.87</td>
</tr>
<tr>
<td>RF energy (W)</td>
<td>30.0 ± 4.4</td>
<td>31.1 ± 5.3</td>
<td>29.7 ± 6.0</td>
<td>0.65</td>
</tr>
<tr>
<td>Temperature achieved (°C)</td>
<td>52.2 ± 3.5</td>
<td>51.5 ± 6.1</td>
<td>49.0 ± 4.5</td>
<td>0.26</td>
</tr>
<tr>
<td>VT recurrence during follow-up (%)</td>
<td>9 (39.1)</td>
<td>10 (14.9)</td>
<td>4 (16.0)</td>
<td>0.04</td>
</tr>
<tr>
<td>VT recurrence during follow-up (per 100 person-years)</td>
<td>0.62</td>
<td>0.06</td>
<td>0.19</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

LPFVT: left posterior fascicular VT; LPFB: left posterior fascicular block; SR: sinus rhythm; VT: ventricular tachycardia; His-RF/His-apical PP - linear distance between the most basal left-sided His potential to the site of successful radiofrequency ablation expressed as a percentage of the distance between the most basal left-sided His potential and the most apically recorded pre-systolic Purkinje potentials during LPFVT; PP: pre-systolic Purkinje potential.
**Table 2: Characteristics of Recurrent Ventricular Tachycardia**

<table>
<thead>
<tr>
<th></th>
<th>Recurrence with LPFVT (n=17)</th>
<th>Recurrence with upper septal VT (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Index ablation</td>
<td>Repeat ablation</td>
</tr>
<tr>
<td>Left posterior fascicular block during sinus rhythm pre-ablation</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>VT cycle length (ms)</td>
<td>337.7 ± 98.9</td>
<td>369.8 ± 104.4</td>
</tr>
<tr>
<td>VT Frontal axis (º)</td>
<td>-98.3 ± 17.2</td>
<td>-76.2 ± 73.3</td>
</tr>
<tr>
<td>VT QRS duration (ms)</td>
<td>121.8 ± 7.6</td>
<td>121.8 ± 13.2</td>
</tr>
<tr>
<td>HV interval (ms)</td>
<td>-10.5 ± 10.7</td>
<td>-6.2 ± 10.2</td>
</tr>
<tr>
<td>Earliest PP-QRS / DP-QRS* interval (ms)</td>
<td>34.0 ± 8.4</td>
<td>34.7 ± 10.2</td>
</tr>
<tr>
<td>His-RF site / His-apical PP (%)</td>
<td>62.3 ± 6.4</td>
<td>61.1 ± 1.7</td>
</tr>
</tbody>
</table>

DP: diastolic potential; LPFVT: left posterior fascicular VT; PP: pre-systolic Purkinje potential; RF: radiofrequency; VT: ventricular tachycardia; His-RF/His-apical PP - linear distance between the most basal left-sided His potential to the site of successful radiofrequency ablation expressed as a percentage of the distance between the most basal left-sided His potential and the most apically recorded pre-systolic Purkinje potentials during LPFVT.
Table 3: Predictors for Successful Left Posterior Fascicular Ventricular Tachycardia Recurrence

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Univariable HR (95%CI)</th>
<th>P value</th>
<th>Multivariable HR (95%CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.99(0.92-1.01)</td>
<td>0.72</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Sex</td>
<td>0.99(0.99-1.01)</td>
<td>0.99</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Earliest PP-QRS interval</td>
<td>0.99 (0.94-1.05)</td>
<td>0.82</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>New onset LPFB after first ablation</td>
<td>2.57 (0.58-11.38)</td>
<td>0.21</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>His-RF site / His-apical PP</td>
<td>0.10 (0.01-17.98)</td>
<td>0.39</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>VH interval during VT</td>
<td>1.04 (0.98-1.12)</td>
<td>0.21</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>VT cycle length</td>
<td>0.98 (0.97-0.99)</td>
<td>0.02</td>
<td>0.98 (0.97-0.99)</td>
<td>0.03</td>
</tr>
<tr>
<td>VT QRS duration</td>
<td>0.91 (0.84-0.99)</td>
<td>0.04</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

HR: hazard ratio; LPFVT: left posterior fascicular VT; PP: pre-systolic Purkinje potential; LPFB: left posterior fascicular block; SR: sinus rhythm; VT: ventricular tachycardia; His-RF/His-apical PP - linear distance between the most basal left-sided His potential to the site of successful radiofrequency ablation expressed as a percentage of the distance between the most basal left-sided His potential and the most apically recorded pre-systolic Purkinje potentials during LPFVT.
**Figure Legends:**

**Figure 1:** Activation map using the NavX system was created during fascicular ventricular tachycardia (FVT). Septal sites with His or Purkinje potentials, extending from most basal septal sites with bundle of His recordings to most apical sites with pre-systolic Purkinje potentials (apical PP), were marked on the left ventricular geometry. The site with the earliest pre-systolic Purkinje potential (earliest PP) during FVT was targeted by catheter ablation. The distance between the most basal His recording to the earliest PP was expressed as a ratio of the distance between the His bundle and apical PP, as an objective measure to indicate the position of ablation sites along the course of the left fascicles.

**Figure 2:** Intracardiac electrograms during electrophysiological studies of left posterior fascicular ventricular tachycardia (LPFVT). Note the more rounded diastolic potential (DP) of the anterograde limb and sharper pre-systolic Purkinje potential (PP) of the retrograde limb of LPFVT recorded on the ablation catheter (ABL). Radiofrequency ablation delivered at this site with the earliest PP terminated LPFVT. CS, coronary sinus; His, His bundle; ABL, ablation catheter.

**Figure 3:** Flow chart of consecutive patients with fascicular ventricular tachycardia enrolled in this study.

**Figure 4:** Panel A – Activation map in patient 1 recorded during the first ablation procedure for left posterior fascicular ventricular tachycardia (LPFVT). Panel B – Activation map in patient 1
recorded during the second ablation procedure after presenting with recurrent LPFVT. Panel C – Activation map in patient 2 recorded during the first ablation procedure with LPFVT. Panel D – Activation map in patient 2 recorded during the second ablation procedure after presenting with upper septal VT. Note in patient 1 that the earliest pre-systolic Purkinje potential during the first and second procedure were similar in location, strongly suggestive that the index LPFVT had recurred. In comparison, the site of successful ablation during upper septal VT in patient 2 is much more basal compared to the original site of ablation during LPFVT. In panels A to C, the ablation catheter was recording the site with the earliest PP (white arrow) whereas during mapping of upper septal VT (Panel D), the earliest DP is targeted (white arrow). The local activation at each site was measured by annotating the local His / Purkinje potential, rather than the ventricular electrogram.

**Figure 5:** 12 lead ECG of index and recurrent fascicular ventricular tachycardias (FVT). Both patients labelled A and B, presented with LPFVT (left panels) which was successfully ablated during their index ablation. ECG axis was unchanged after the index ablation in Patient A whereas there was onset of left posterior fascicular block after the first ablation in Patient B. LPFVT of near-identical morphology recurred in patient A and was successfully re-ablated. In patient B, VT recurrence was due to upper septal VT that was ablated during the second procedure.
Figure 6: Right anterior oblique (left panel) and left anterior oblique (right panel) of the ablation catheter (ABL) position during upper septal VT ablation. Note the proximity to the right sided His catheter. RAA, right atrial appendage; RVA, right ventricular apex.
Catheter Ablation of Fascicular Ventricular Tachycardia: Long Term Clinical Outcomes and Mechanisms of Recurrence
Yaowu Liu, Zhen Fang, Bing Yang, Pipin Kojodjojo, Hongwu Chen, Weizhu Ju, Kejiang Cao, Minglong Chen and Fengxiang Zhang

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Supplemental Figure Legends

**Supplemental Figure 1.** The pre-systolic potential (PP) occurs typically pre-QRS and the diastolic potential (DP) were recorded by twenty-polar catheter extends from left basal septal sites to most apical sites during fascicular ventricular tachycardia.

**Supplemental Figure 2.** The ECGs of upper septal VT were printed on paper from 3 patients. The ECGs from the remaining 3 patients were too faint to be seen clearly. Left panel shows 12 lead ECG during sinus rhythm after the first radiofrequency ablation and right panel shows upper septal VT which was inducible during the second ablation procedure.

**Supplemental Figure 3.** Entrainment mapping was performed in a patient with upper septal VT with a tachycardia cycle length of 315 ms. Concealed entrainment was achieved at the left basal septal site (A) with a post-pacing interval (PPI) of 320 ms. In comparison, manifest fusion was achieved at the middle anterior septum (B) and the LV apex (C) with PPIs of 378 ms and 392 ms, respectively.
Supplemental Figure 1
Supplemental Figure 2

Case 1

Case 2

Case 3

I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
SR
VT
SR
VT
SR
VT