Non-Reentrant Fascicular Tachycardia
Clinical and Electrophysiological Characteristics of a Distinct Type of Idiopathic Ventricular Tachycardia

Ahmed Karim Talib, MD, PhD; Akihiko Nogami, MD, PhD; Itsuro Morishima, MD; Yasushi Oginosawa, MD, PhD; Kenji Kurosaki, MD; Shinya Kowase, MD; Yuki Komatsu, MD; Kenji Kuroki, MD, PhD; Miyako Igarashi, MD, PhD; Yukio Sekiguchi, MD; Kazutaka Aonuma, MD, PhD

Background—The most common form of idiopathic Purkinje-related ventricular tachycardia (VT) is the reentrant type. We describe the clinical and electrophysiological characteristics of focal non-reentrant fascicular tachycardia.

Methods and Results—Among 530 idiopathic VT patients who were referred for ablation, we identified 15 (2.8%) with non-reentrant fascicular tachycardia (11 men, 45±21 years). Sinus rhythm ECG showed normal conduction intervals with a His–ventricular interval of 41±4 ms. All patients had monomorphic VT (cycle length: 337±88 ms) with a relatively narrow QRS (123±12 ms), and they did not respond to verapamil during the initial presentation. VT exhibited right bundle-branch block/superior axis configuration in 11 patients (73%) and inferior axis in 3 (20%). In 1 patient (7%), VT exhibited left bundle-branch block/superior axis configuration. During ablation, spontaneous VT occurred in 3 patients (20%) and nonentraintable VT or identical premature ventricular complex was induced in 9 (60%). A high-frequency presystolic Purkinje potential was recorded during VT/premature ventricular complex, preceding the QRS by 25±16 ms. VT recurrence was observed in 4 patients (27%), and among them, 3 underwent pacemap-guided ablation during the first session. A second ablation with activation mapping guidance eliminated the VT during the 88±8-month follow-up.

Conclusions—Among idiopathic VT cases referred for ablation, 2.8% were focal non-reentrant fascicular tachycardia, which had distinct clinical characteristics and usually originated from the left posterior fascicle, and less commonly from the left anterior fascicle and right ventricular Purkinje network. Catheter ablation is effective, whereas pacemap-guided approach is less efficacious. (Circ Arrhythm Electrophysiol. 2016;9:e004177. DOI: 10.1161/CIRCEP.116.004177.)

Key Words: catheter ablation  ■  fascicular ventricular tachycardia  ■  Purkinje  ■  verapamil

Reentry is a common mechanism of ventricular tachycardia (VT) that originates from the Purkinje system, and verapamil-sensitive idiopathic left ventricular tachycardia (ILVT) is considered the most common form of Purkinje-related idiopathic VT. Although focal non-reentrant fascicular tachycardia (NRFT) was reported in patients with structural heart disease involving the Purkinje system, such as those associated with myocardial infarction, little is known about the prevalence, electrophysiological characteristics, or preferential sites of the tachycardia origin of NRFT in patients without structural heart diseases. The purpose of this study was to clarify the aforementioned points with the results of long-term follow-up after radiofrequency catheter ablation (RFCA).

Methods

Study Sample
Among 530 patients who were referred for RFCA of idiopathic VT, we identified 15 patients (2.8%) with distinct electrocardiographic and electrophysiological characteristics of focal NRFT. The study only included cases of VT, and those with isolated premature ventricular complex (PVC) were excluded. Of note, 17 patients had isolated PVCs of NRF type (14 left posterior fascicle [LPF] origin, 2 left anterior fascicle origin, and 1 right ventricle [RV]-Purkinje origin). For each patient, after a detailed medical history and examination were conducted, structural heart diseases were ruled out by using a standard investigation protocol, which included a 12-lead surface ECG, chest radiography, 2-dimensional (2D) echocardiography, 24-hour Holter recordings, cardiac computed tomography (when appropriate), coronary angiography, and right or left ventriculography. This study was approved by the local ethics committee and all patients provided written informed consent.

Electrophysiology Study and the Stimulation Protocol
The electrophysiology study was performed after obtaining written informed consent from patients, and antiarrhythmic drugs were withdrawn for ≥5 half-lives. Standard multielectrode catheters were placed in the high right atrium, His-bundle region, and RV apex. Programmed atrial and ventricular stimulation was performed...
WHAT IS KNOWN

• In normal hearts, reentrant fascicular tachycardia is the most common type of Purkinje-related arrhythmia.
• NRFT can be seen in structural heart diseases when the His-Purkinje system is diseased, but there is little data on this arrhythmia in the absence of heart disease.

WHAT THIS STUDY ADDS

• In a large series, NRFT represents ≈3% of all idiopathic ventricular tachycardia cases referred for catheter ablation.
• Electrocardiographically, NRFT is similar to reentrant FT but is verapamil resistant. Difficulty in inducing NRFT and the potential for multiple QRS morphologies are important challenges for ablation.

using a maximum of triple extrastimuli at 2 different driven cycle lengths from the right atrium and the RV apex, respectively. In addition, incremental atrial and ventricular stimulation with constant cycle length was performed. The stimulation protocol was terminated when sustained VT was induced. If sustained VT was not induced, stimulation was repeated after a bolus intravenous injection of 0.5 mg of atropine. If VT or PVC of the same morphology was still not induced, stimulation was repeated during intravenous isoproterenol administration (0.5–2 mg/min). During the episodes of clinical arrhythmias, activation mapping was performed using the CARTO system (Biosense-Webster, Diamond Bar, CA) or the NavX Ensite Velocity system (St. Jude Medical Inc, Milwaukee, WI). In patients with sustained monomorphic VT, overdrive pacing was performed locally from the ablation catheter at the area of interest and remotely from the right atrium and RV. Right atrial pacing was attempted in the presence of consistent (1:1) atrioventricular nodal conduction (ie, tachycardia cycle length is shorter than the atrioventricular nodal Wenckebach cycle length). To achieve this, intravenous atropine was injected. Although atropine has no effect on the VT circuit itself, it enhances the atrioventricular nodal conduction. Therefore, atropine can increase the maximum stimulation rate to the Purkinje network during atrial stimulation. We think that atrial pacing is also useful for non-reentrant VT because burst pacing can induce triggered activity–related arrhythmias while it suppresses arrhythmias caused by automaticity.\(^1\) Practically, atropine has another advantage in the stability of the mapping/ablation catheter because, unlike isoproterenol, atropine does not cause hyperkinetic contraction of the left ventricle (LV). Intracardiac echocardiogram with 3D reconstruction (SoundStar, Biosense Webster, Inc) was used in 6 patients, 8 to 12 and 15, to confirm the ablation catheter position and stability and exclude the possibility of contribution of intracavitary structures, such as the papillary muscles or false tendons to the VT origin.

Mapping and Ablation

In patients with LV VT, a transaortic approach was used to access the LV. Mapping was performed with a 7-French nonirrigated ablation catheter with an 8-mm tip (NaviStar, Biosense Webster) or a 7.5-French irrigation catheter with a 3.5-mm tip (NaviStar ThermoCool, Biosense Webster). Ablation was performed with a maximum power of ≤50 W and a target temperature of 58°C for the nonirrigated catheter. When an acceleration or reduction in the incidence of VT or PVCs was observed during the first 10 s of the application, the radiofrequency delivery was continued for 60 to 180 s while carefully monitoring the surface ECG and conduction intervals. Otherwise, radiofrequency delivery was terminated, and the catheter was repositioned. If VT or isolated PVCs with identical morphology to the clinical VT were induced, activation mapping was performed. The presence of Purkinje potentials was assessed at the site of earliest activation during ventricular arrhythmias (ie, VT/PVCs). Purkinje potential was also assessed during sinus rhythm. Pacemapping around the presumably successful site was performed. A successful ablation site was defined as the site of complete suppression of the clinical arrhythmia. Several additional radiofrequency energy applications were delivered around the successful site to consolidate the lesion. A good pacemap was confirmed before applying these additional radiofrequency applications. Successful ablation was defined as the disappearance of spontaneous VT/PVCs and the noninducibility of the VT or PVCs using the same protocol as that before ablation.

Follow-Up

Patients were monitored for 1 to 3 days in the hospital after ablation. Then, they were followed for an 8±8-month period (range: 6–132 months). A 24-hour Holter recording was obtained at approximately yearly intervals.

Statistical Analysis

Values are given as mean±SD.

Results

Among 530 patients who were referred for RFCA of idiopathic VT, 15 (2.8%) had idiopathic NRFT. Eleven patients (73%) were men and 4 patients were women between the age of 18 and 70 years (mean age: 45±21 years). Figure 1 outlines the ablation flowchart for the study cohort. Fourteen patients presented with palpitation and had normal systolic function (LV ejection fraction: 64±5%), whereas 1 patient (patient no. 15) presented with shortness of breath associated with heart failure due to tachycardia-induced cardiomyopathy (LV ejection fraction: 45%). LV false tendon was observed in 5 patients (patients 1–5). Representative cases with LV false tendon detected by transthoracic echocardiography and cardiac computed tomography are shown in Figure 2.

Electrocardiographic Characteristics

Baseline ECG showed sinus rhythm with normal conduction intervals in all patients. Eleven patients initially presented with symptomatic sustained monomorphic VT, whereas 4 had salvos of nonsustained VT (cycle length: 337±88 ms), with a QRS duration of 123±12 ms (Table 1). Verapamil was administered in 10 patients when they initially presented with VT, but it was ineffective for VT termination in all of them. The VT exhibited 3 distinct QRS morphologies: (1) pattern 1 (Figure 3A), right bundle-branch block (RBBB) configuration and superior axis in 11 patients (73%); (2) pattern 2 (Figure 3B), RBBB configuration and inferior axis in 3 patients (20%); and (3) pattern 3 (Figure 3C), left bundle-branch block (LBBB) configuration and superior axis in 1 patient (7%). In lead V1, among the 14 LV-origin NRFT cases, an rR pattern was observed in 9 cases (64%), Rr′ in 2 cases, monophasic R in 2 cases, and qR pattern in 1 case (Table 1).

Electrophysiological Characteristics

The electrophysiological characteristics of VT and the successful ablation site are summarized in Table 2. All patients had normal His–ventricular interval during sinus rhythm. During the electrophysiological study, clinical VT spontaneously occurred in 3 patients (20%), and it was induced in
5 (33%) using isoproterenol infusion (atropine in 1 patient) or burst pacing.

In 4 patients (27%), isolated PVCs, with an identical morphology to clinical VT, were induced. In the remaining 3 patients (20%), all the aforementioned protocols failed to induce VT/PVC, thus pacemap-guided ablation was performed (Table 2).

In patients with sustained monomorphic VT, overdrive pacing was performed from the right atrium, RV, and locally from the ablation catheter at the area of interest; however, VT could not be entrained by overdrive pacing. Figure 3D shows the absence of constant fusion during RV apex overdrive pacing (patient no. 13). This finding proves that NRFT is a non-reentrant tachycardia.

During clinical arrhythmias, His potential was recorded during VT/PVC in 10 patients. It was recorded after the onset of QRS complex with an His–ventricular interval of −20±12 ms.

Figure 1. Flowchart of the study cohort. Among the 530 patients referred for radiofrequency catheter ablation (RFCA) of idiopathic ventricular tachycardia (VT), 15 (2.8%) had non-reentrant fascicular tachycardia. VT exhibited 3 distinct QRS morphologies: (1) pattern 1: a right bundle-branch block (RBBB) configuration and superior axis in 11 patients (73%); (2) pattern 2: an RBBB configuration and inferior axis in 3 patients (29%); and (3) pattern 3: a left bundle-branch block (LBBB) configuration and superior axis in 1 patient (7%). Patients were treated by (1) or (2) ablation procedures. IA indicates inferior axis; ILVT, idiopathic left ventricular tachycardia; NRFT, non-reentrant fascicular tachycardia; PVC, premature ventricular contraction; and SA, superior axis.

Figure 2. Representative cases showing left ventricular false tendons. False tendons (arrows) were detected on trans-thoracic echocardiogram in patient no. 1 (left) and on cardiac computer tomography in patient no. 5 (right).
Mapping and Ablation
A nonirrigated tip catheter was used in 8 patients (patients 1–7 and 14), and an irrigated tip catheter was used in 7. Among the 7 patients, the irrigated tip catheter failed to eliminate VT/PVC in 2 patients; however, a nonirrigated tip catheter was successfully used instead (patients 8 and 10). Table 3 shows the results of RFCA. At the successful ablation site, the presystolic Purkinje potential preceded the QRS onset of the VT/PVCs by 25±16 ms. In 3 patients with noninducible VT/PVC, pacemap-guided RFCA was performed.

For all VTs with an RBBB configuration and superior axis, the successful ablation site was at the LPF vicinity, namely the mid- to apical-inferior septum of the LV. Figure 4A and 4B shows the successful ablation site in patient no. 7. By positioning the ablation catheter against the LV septum at the midthird of the LV septum, a presystolic Purkinje potential was recorded during sinus rhythm. During PVC mapping, the same potential was recorded in the reverse direction, during which the Purkinje potential preceded the QRS by 12 ms, whereas the His-bundle was recorded after both the Purkinje potential and the QRS onset. Figure 4C and 4D. For VT with LBBB configuration and superior axis (patient no. 11), the successful ablation site was at the RV midanteroseptum, where the Purkinje potential preceded the QRS onset of the PVC by 30 ms (Figures 4E and 4F). During sinus rhythm, Purkinje-like potential was also recorded; however, it was buried within the ventricular potential (Figure 4E). Although intracardiac echocardiogram did not reveal any endocavitary structure at the site of origin, the recorded site might be close to the septal papillary muscle of the RV.3

Pacemapping was also performed. Because there was no mappable VT/PVC (spontaneous or induced) during the RFCA session, pacemap-guided ablation was performed in 3 patients (patients 2, 4, and 6). Notably, excellent pacemap was obtained in only 1 patient (no. 6). The best pacemap site did not always represent the successful ablation site. Figure 5 shows that despite obtaining a perfect pacemap match at the third of the LV septum (Figure 5A), VT could not be terminated. However, radiofrequency application at a more proximal part (ie, the middle third of the LV septum) successfully terminated VT within 4 s after radiofrequency energy application delivered though pacemapping did not match clinical VT (Figure 5B).

Ablation End Point
The ablation end point was to eliminate clinical arrhythmia and the newly induced ventricular arrhythmias. After repeating the same induction protocol, 3 patients developed a new VT of a different morphology than the original one, and the new VT was successfully ablated in all 3 patients (Table 3; Figure 1). At the end of the session, all inducible VTs in 12 patients became noninducible, and among them, only isolated PVCs were observed in 2 patients. In 3 patients, clinical VT could not be induced at baseline; therefore, the ablation end point was unclear. After ablation, the local Purkinje potential was delayed in 9 patients (Figure 6), and it was not changed in 6 without any change in the His–ventricular interval (Table 3).

Table 1. Electrocardiographic Characteristics of Clinical VT

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Clinical VT</th>
<th>VT Morphology</th>
<th>V1 QRS Morphology</th>
<th>VT-CL, ms</th>
<th>VT-QRSd, ms</th>
<th>Verapamil Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>SMVT</td>
<td>RBBB-SA</td>
<td>rR'</td>
<td>250</td>
<td>119</td>
<td>No effect</td>
</tr>
<tr>
<td>2</td>
<td>NSMVT</td>
<td>RBBB-SA</td>
<td>rR'</td>
<td>283</td>
<td>145</td>
<td>No effect</td>
</tr>
<tr>
<td>3</td>
<td>SMVT</td>
<td>RBBB-SA</td>
<td>qR</td>
<td>250</td>
<td>125</td>
<td>No effect</td>
</tr>
<tr>
<td>4</td>
<td>SMVT</td>
<td>RBBB-SA</td>
<td>rR'</td>
<td>267</td>
<td>140</td>
<td>No effect</td>
</tr>
<tr>
<td>5</td>
<td>NSMVT</td>
<td>RBBB-SA</td>
<td>monophasic R</td>
<td>480</td>
<td>132</td>
<td>No effect</td>
</tr>
<tr>
<td>6</td>
<td>SMVT</td>
<td>RBBB-SA</td>
<td>Rr</td>
<td>210</td>
<td>129</td>
<td>Not used</td>
</tr>
<tr>
<td>7</td>
<td>Incessant MVT</td>
<td>RBBB-SA</td>
<td>rR'</td>
<td>400</td>
<td>110</td>
<td>Not used</td>
</tr>
<tr>
<td>8</td>
<td>SMVT</td>
<td>RBBB-SA</td>
<td>Rr</td>
<td>500</td>
<td>129</td>
<td>Not used</td>
</tr>
<tr>
<td>9</td>
<td>NSMVT</td>
<td>RBBB+IA</td>
<td>monophasic R</td>
<td>400</td>
<td>98</td>
<td>No effect</td>
</tr>
<tr>
<td>10</td>
<td>SMVT</td>
<td>RBBB+IA</td>
<td>rR'</td>
<td>330</td>
<td>116</td>
<td>No effect</td>
</tr>
<tr>
<td>11</td>
<td>NSMVT</td>
<td>LBBB+SA</td>
<td>QS</td>
<td>315</td>
<td>127</td>
<td>Not used</td>
</tr>
<tr>
<td>12</td>
<td>SMVT</td>
<td>RBBB-SA</td>
<td>rR'</td>
<td>430</td>
<td>125</td>
<td>Not used</td>
</tr>
<tr>
<td>13</td>
<td>SMVT</td>
<td>RBBB-SA</td>
<td>Rr</td>
<td>360</td>
<td>120</td>
<td>No effect</td>
</tr>
<tr>
<td>14</td>
<td>SMVT</td>
<td>RBBB-SA</td>
<td>rR'</td>
<td>275</td>
<td>115</td>
<td>No effect</td>
</tr>
<tr>
<td>15</td>
<td>Incessant MVT</td>
<td>RBBB+IA</td>
<td>rR'</td>
<td>300</td>
<td>120</td>
<td>No effect</td>
</tr>
</tbody>
</table>

Mean±SD 337±88 123±12

IA indicates inferior axis; LBBB, left bundle-branch block; MVT, monomorphic ventricular tachycardia; NSMVT, nonsustained monomorphic ventricular tachycardia; QRSd, QRS duration; RBBB, right bundle-branch block; SA, superior axis; SMVT, sustained monomorphic ventricular tachycardia; VT, ventricular tachycardia; and VT-CL, ventricular tachycardia cycle length.
Figure 3. Representative surface ECG showing 3 different patterns of idiopathic non-reentrant fascicular tachycardia. **A**, Pattern 1: right bundle-branch block (RBBB) configuration and superior axis (patient no. 14). **B**, Pattern 2: RBBB configuration and inferior axis (patient no. 10). **C**, Pattern 3: left bundle-branch block configuration and superior axis (patient no. 11). In this patient, nonsustained (Continued)
Regarding complications, right-axis deviation was observed in 1 patient (no. 8), and mild left-axis deviation was observed in another patient (no. 15); however, neither atrioventricular block nor LBBB was found during or after RFCA.

Follow-Up
During the 4±3-month follow-up after the first RFCA session, VT recurrence was seen in 4 patients (27%; nos. 2, 3, 4, and 6) for whom a second RFCA session resulted in VT elimination during the 88±8-month follow-up (Figure 1). Notably, all 3 patients who underwent pacemap-guided ablation during their first procedure developed VT recurrence. Specifically, 2 patients developed the same VT morphology and 1 patient developed VT of a different morphology. However, 2 patients who had their local Purkinje potentials significantly delayed during the first procedure developed VT of a different morphology (Table 3).

In 1 patient who presented with tachycardia-induced cardiomyopathy (no. 15), a dramatic improvement in the patient’s symptoms and LV function was observed (LV ejection fraction was 54% 6 months after ablation).

Discussion

Main Findings
This is the first study to demonstrate that 2.8% of idiopathic VT cases referred for ablation are of non-reentrant Purkinje origin, with men being more commonly affected. Idiopathic NRFT is characterized by the following: (1) normal electrocardiographic and intracardiac conduction intervals; (2) right and less commonly, left-branch block morphology, depending on the site of origin with a relatively narrow QRS complex; (3) inducibility of VT or PVCs with intravenous isoproterenol infusion and burst ventricular stimulation; (4) the absence of criteria of reentrant ILVT, such as transient entrainment and verapamil sensitivity; and (5) the presence of high-frequency presystolic Purkinje potentials at the site of origin.

Table 2. Electrophysiological Characteristics of Idiopathic NRFT and the Successful Ablation Site

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Induction Mode</th>
<th>Mappable Arrhythmia</th>
<th>PP-QRS, ms</th>
<th>12-Lead Pacemap</th>
<th>Sinus HV, ms</th>
<th>VT-HV, ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ISP+burst VP</td>
<td>PVC</td>
<td>20</td>
<td>9/12</td>
<td>38</td>
<td>−15</td>
</tr>
<tr>
<td>2</td>
<td>Noninducible</td>
<td>None</td>
<td></td>
<td>Pacemap-guide</td>
<td>9/12</td>
<td>42</td>
</tr>
<tr>
<td>3</td>
<td>Burst VP</td>
<td>PVC</td>
<td>22</td>
<td>10/12</td>
<td>50</td>
<td>−13</td>
</tr>
<tr>
<td>4</td>
<td>Noninducible</td>
<td>None</td>
<td></td>
<td>Pacemap-guide</td>
<td>11/12</td>
<td>38</td>
</tr>
<tr>
<td>5</td>
<td>Spontaneous</td>
<td>VT</td>
<td>16</td>
<td>11/12</td>
<td>38</td>
<td>−12</td>
</tr>
<tr>
<td>6</td>
<td>Noninducible</td>
<td>None</td>
<td></td>
<td>Pacemap-guide</td>
<td>12/12</td>
<td>42</td>
</tr>
<tr>
<td>7</td>
<td>Burst VP</td>
<td>PVC</td>
<td>12</td>
<td>n/a</td>
<td>42</td>
<td>−32</td>
</tr>
<tr>
<td>8</td>
<td>Burst AP</td>
<td>VT</td>
<td>32</td>
<td>9/12</td>
<td>36</td>
<td>−10</td>
</tr>
<tr>
<td>9</td>
<td>Spontaneous</td>
<td>VT</td>
<td>68</td>
<td>9/12</td>
<td>42</td>
<td>Immeasurable</td>
</tr>
<tr>
<td>10</td>
<td>Atropine+burst VP</td>
<td>VT</td>
<td>17</td>
<td>9/12</td>
<td>42</td>
<td>−30</td>
</tr>
<tr>
<td>11</td>
<td>ISP</td>
<td>PVC</td>
<td>30</td>
<td>10/12</td>
<td>42</td>
<td>−35</td>
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<tr>
<td>12</td>
<td>Spontaneous</td>
<td>VT</td>
<td>30</td>
<td>10/12</td>
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<td>−9</td>
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<tr>
<td>13</td>
<td>ISP</td>
<td>VT</td>
<td>15</td>
<td>8/12</td>
<td>44</td>
<td>−5</td>
</tr>
<tr>
<td>14</td>
<td>ISP</td>
<td>VT</td>
<td>n/a*</td>
<td>11/12</td>
<td>40</td>
<td>−38</td>
</tr>
<tr>
<td>15</td>
<td>ISP+burst VP</td>
<td>VT</td>
<td>12</td>
<td>11/12</td>
<td>42</td>
<td>Immeasurable</td>
</tr>
<tr>
<td>Mean±SD</td>
<td></td>
<td></td>
<td>25±16</td>
<td>41±4</td>
<td>−20±12</td>
<td></td>
</tr>
</tbody>
</table>

AP indicates atrial pacing; HV, His-ventricle; ISP, isoproterenol; NRFT, non-reentrant fascicular tachycardia; PP, presystolic Purkinje potential; PVC, premature ventricular complex; VP, ventricular pacing; and VT, ventricular tachycardia.

*n/a=not available (PP–QRS interval at the successful site could not be recorded because VT was terminated by the mechanical bump).
where RFCA at the earliest Purkinje potential can eliminate the clinical arrhythmias. NRFT has been reported in patients with structural heart diseases, such as ischemic cardiomyopathy and fulminant myocarditis; however, the prevalence and electrophysiological characteristics of idiopathic NRFT have not been fully clarified. Although sporadic cases of idiopathic NRFT have been reported, to the best of our knowledge, this is the first study to demonstrate the prevalence, clinical and electrophysiological characteristics of this type of VT in patients with structurally normal hearts.

ECG Characteristics of NRFT
Baseline sinus ECG showed normal conduction intervals and relatively narrow QRS duration during VT (123±12 ms). Clinical VT exhibited RBBB or LBBB configuration, depending on the affected Purkinje fibers. In the majority of the patients, the QRS morphology during VT exhibited an RBBB configuration and superior axis, consistent with its origin from the LPF where VT was successfully eliminated. In 2 patients, VT had RBBB configuration and inferior axis, indicating its origin from the left anterior fascicle. In 1 patient, VT exhibited LBBB and superior axis configuration, indicating RV origin, and PVC was successfully ablated at the RV midseptum.

Electrophysiological Characteristics of Idiopathic NRFT
This type of VT is usually induced by exercise, emotional stress, and fever, and it is classified as propranolol-sensitive VT. It is thought to be because of abnormal automaticity in most cases. In our cases, the majority of PVCs/VT could be induced by intravenous isoproterenol or less commonly, atropine infusion or by burst atrial/ventricular pacing, and the induced VT could not be entrained. Although tachycardia induction by burst atrial/ventricular pacing or by using Isoproterenol infusion may indicate triggered activity as an underlying mechanism of NRFT, abnormal automaticity cannot be excluded because NRFT was nonsustained in the majority of the cases in which many maneuvers such as overdrive pacing could not be performed to rule out the presence of abnormal automaticity. Hence, it remains difficult to differentiate between these 2 mechanisms in our cohort.

Rodriguez et al reported a case of a focal VT originating from the left anterior fascicle in a patient without structural heart disease. β-blockers slowed the VT, adenosine, and verapamil had no effect, and lidocaine and procainamide terminated the VT. In our cohort, verapamil was used in 10 patients, and it had no effect on VT. LV false tendon was observed in 5 patients on echocardiography, enhanced computed tomography, or intracardiac echocardiogram. In all these cases, the false tendon was located around the area where the ablation was performed. However, it was unclear whether the arrhythmogenic focus of the Purkinje network existed in the false tendons.

Differential Diagnosis of NRFT
Electrocardiographically, idiopathic NRFT should be differentiated from reentrant fascicular VT. There are many
similarities between NRFT and reentrant ILVT, including male predominance, a relatively narrow QRS tachycardia with an RBBB and left axis QRS morphology in the majority of cases. Hence, it is difficult to differentiate between the 2 arrhythmias based on surface ECG.

Although reentrant fascicular VT is verapamil sensitive, it can be reproducibly initiated and terminated by programmed stimulation and classical criteria of entrainment, and both presystolic and diastolic potentials can be demonstrated. VT in our patients was verapamil-resistant and

Figure 4. Successful ablation sites of the 3 subtypes of idiopathic non-reentrant fascicular tachycardia (NRFT). A, Pattern 1 (patient no. 7): during sinus rhythm, the ablation catheter at the midapical left posterior fascicle demonstrated a presystolic Purkinje potential, which was recorded after His potential and followed by ventricular activation. During premature ventricular contraction (PVC) of the same morphology of clinically documented VT (right bundle-branch block [RBBB] configuration and superior axis), the Purkinje potential preceded the QRS onset of the PVC by 12 ms. The PVC/VT was eliminated by radiofrequency catheter ablation (RFCA) at the same site. Note that the Purkinje potential was activated antegradely (proximal to distal) during sinus rhythm, while during PVC, the Purkinje potentials were activated retrogradely (arrows). B, Right anterior oblique (RAO, 35°) and left anterior oblique (LAO, 45°) views of the successful ablation site. (Continued)
inducible by isoproterenol infusion or burst pacing, and it could not be entrained. Moreover, detailed VT mapping did not show any diastolic potential that suggests a reentrant circuit. Because fascicular reentrant circuits are notoriously bumpable during mapping, the VT stimulation protocol was performed before introducing the mapping/ablation catheter into the ventricle, rendering noninducibility of NRFT unlikely to be bump related. Yet, lack of inducibility cannot definitely determine VT mechanism. Other clues of a non-reentrant mechanism are the pattern of NRFT initiation and variations in its cycle length (Figure 3C). Another important differential diagnosis is papillary muscle VT, which has a wider QRS complex than NRFT. A wide QRS probably represents a site of origin at the Purkinje–myocardial interface, where conduction is slower than in the Purkinje system. Moreover, the presence of a V1 r<R′ pattern in the majority of our NRFT cases may support the diagnosis of fascicular arrhythmia as opposed to papillary muscle VT, as suggested by others. In the electrophysiology laboratory, high-frequency Purkinje potentials are usually absent at the site of origin of papillary muscle VT. However, this is not the case when Purkinje fibers are distributed at the surface of papillary muscles. Furthermore, unlike NRFT, papillary muscle VT usually originates somewhere deep relative to the

Figure 4 Continued. C, Pattern 2 (patient no. 10): during sinus rhythm, a presystolic Purkinje potential was recorded at the midleft anterior fascicle (LAF). During VT (RBBB configuration and inferior axis), the Purkinje potential preceded the QRS of the VT by 17 ms. RFCA at the same site successfully suppressed the VT. D, RAO and LAO views of 3-dimensional electroanatomical mapping of pattern 2 idiopathic NRFT. After mapping, the His (green tags) and Purkinje potentials (yellow tags) sites during sinus rhythm, VT activation mapping showed that the early activation site was within the LAF vicinity, which was separated from the anterior papillary muscle (APM) as is clearly shown in the LAO view. RFCA at the earliest Purkinje potential sites (red tags) completely suppressed VT. E, Pattern 3 (patient no. 11): the ablation catheter was (Continued)
placed at the right ventricular midseptum. During sinus rhythm, Purkinje-like potential was buried within the ventricular potential. During PVC with the same morphology of clinical VT, the Purkinje potential preceded surface QRS by 30 ms, and a good pacemap was obtained at the same site. RFCA at the same site suppressed VT successfully. F, Fluoroscopic images (top) and 3-dimensional electro-anatomical mapping (bottom) of the successful ablation site in the RAO (right) and LAO (left) views. The blue tags represent the sites of good pacemap. Intracardiac echocardiography (ICE) was used to exclude the presence of endocavitary structures that may contribute to the VT origin. 3–4 indicates proximal bipole; 1–2, distal bipole; A, atrium; ABL, ablation catheter; H, His potential; HBE, His-bundle electrogram; HRA, high right atrium; P, Purkinje potential; PPM, posterior papillary muscle; and RV, right ventricle.
endocardial surface of the papillary muscle, as evidenced by the requirement for cooled ablation to achieve long-term success. Conversely, focal NRFT can be ablated using non-irrigation catheters, indicating that NRFT origin is subendocardial and can be ablated without creating deep lesions. Although cooled tip catheters produce a larger lesion size with sufficient depth when compared with that produced by nonirrigated catheters, the irrigation system reduces the ablation electrode temperature and the temperature at the tissue interface, thus relatively sparing the endocardium. Although this advantage reduces the risk of clot formation and charring, a reduced endocardial temperature may prevent sufficient energy delivery to the Purkinje/fascicular origin because Purkinje tissue is a subendocardial structure. Such a problem can be overcome by using nonirrigated catheters.

**Challenges in Idiopathic NRFT Ablation**

Only sporadic cases of successful idiopathic NRFT ablation have been reported to date. Ablation may be hampered by a relatively high recurrence rate compared with other forms of idiopathic VTs, such as outflow tract VT. Our data suggest that the high recurrence rate is mainly because of 3 factors: the nature of VT, mapping limitations, and potential risk of complications. Owing to the lack of reproducible VT induction, noninducibility has to be considered rather a weak end
Figure 6. Changes in the presystolic Purkinje potentials (PPs) after successful radiofrequency catheter ablation. A, PPs were recorded both during sinus rhythm and premature ventricular contraction of a similar morphology to that of clinically documented ventricular tachycardia (patient no. 1). After successful ablation, the PP appeared after the diminished myocardial potential without changes in the surface QRS morphology or His–ventricular interval. B, PPs were recorded during sinus rhythm (patient no. 4). After the successful third radiofrequency (RF) application (RF no. 3), splitting of the PP was observed without changes in the surface QRS morphology or His–ventricular interval. After additional RF applications at the adjacent sites (RF no. 4 and RF no. 5), the PPs were significantly delayed during sinus rhythm. Because non-reentrant fascicular tachycardia (NRFT) originates from a focus within a mesh-like subendocardial Purkinje network, ablation of the earliest Purkinje focus, which was presumably the origin of NRFT, caused fragmentation and delay of that Purkinje focus (P′) from the local ventricular myocardium. This indeed resulted in successful NRFT elimination. This indicates a local Purkinje–Purkinje (intra-Purkinje) block and Purkinje–myocardial exit block. After ablation, that site was activated by inputs from a different Purkinje arborization.
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Point in NRFT ablation. If VT is noninducible, pacemapping would be a reasonable alternative option. A pacemap-guided approach is generally used for other idiopathic focal VTs, such as outflow VT and atrioventricular annular VT. However, in NRFT, we have found that pacemapping at the successful ablation site cannot produce an excellent QRS match in the vast majority (93%) of cases, because selective pacing of the Purkinje potential of interest is difficult, and local myocardial capture may result in different QRS morphologies. In addition, good pacemapping does not always indicate the site of origin. Pacing at the exit site from the Purkinje network to the ventricular myocardium may reproduce an identical QRS configuration. Indeed, VT recurrence was observed in 4 patients, 3 of whom had noninducible VT, and pacemap-guided ablation was performed during the first ablation session. Gonzalez et al reported a low success rate of catheter ablation at the exit site (2 successful cases out of 5). In our cohort, 2 cases developed a new VT of a different morphology, suggesting that ablating the exit site, as determined by pacemapping, did not necessarily eliminate the VT origin; instead, it may have only changed the VT exit to an adjacent Purkinje arborization or to the myocardium. This notion is depicted in Figure 5, which shows that pacemapping at the apical third of the LV inferior septum reproduced an excellent QRS match, but the ablation was ineffective, whereas rapid and effective VT termination could be performed at the mid part of the LV septum, although pacemapping resulted in a different QRS match at the same site.

Although noninducible reentrant fascicular tachycardia can be ablated anatomically by transecting the reentrant circuit of left posterior or anterior fascicle, empirical ablation is not effective for focal NRFT. Although pacemap-guided ablation has limitations, it is still reasonable to ablate Purkinje focus that reproduces the best pacemap-match, especially if ablation reproduces repetitive ventricular responses of similar morphology to that of clinical arrhythmia. Of note, various pacing inputs should be attempted to selectively capture the focal Purkinje focus.

It is also possible that the Purkinje focus has several exit sites that produce different morphologies. Of note, after

Figure 7. Ventricular tachycardia (VT) recurrence with a different QRS morphology (patient no. 4). A, ECG comparison between the original non-reentrant fascicular tachycardia (VT1) and another VT that recurred 2 mo after the first session (VT2). The VT cycle length and morphology in limb leads were almost identical, whereas the precordial recording was different. VT2 exhibited an atypical right bundle-branch block (RBBB) configuration. B, CARTO mapping of the first and second sessions. The red tags indicate the ablation sites, and the green tags represent presystolic Purkinje potential sites. During the first session, the ablation site (blue arrow) was determined by pacemapping because VT/premature ventricular contraction (PVC) could not be induced. During the second session, PVC with the same morphology of VT2 could be mapped, and the earliest presystolic Purkinje potential was observed slightly proximal to the site of the midapical left posterior fascicle (LPF) lesion. PVC was eliminated by radiofrequency delivery to this lesion. The yellow tags indicate the sites of presystolic Purkinje potential during sinus rhythm. These findings suggest that both VTs had the same origin but ablation of the exit site, as determined by pace mapping, does not necessarily eliminate the origin of non-reentrant fascicular tachycardia (NRFT), but may change its exit to an adjacent Purkinje arborization or to the myocardium. C, Schematic presentations of VT1 and VT2. The focal origin of VT1 and VT2 was the same and located at the distal site of Purkinje arborization near the LPF. Radiofrequency catheter ablation (RFCA) at the first session created a conduction block in the Purkinje network and conduction from the origin to another Purkinje arborization via the septal muscle occurred. This explains why VT2 had a similar cycle length, slightly less superior axis, and less RBBB configuration. RFCA was successful at the proximal site of the Purkinje potential during PVC.
suppressing the original VT, a new VT of a different morphology was observed during the procedures in 3 patients. In 2 of them, the new VT had a different axis (Table 3). This indicates that the Purkinje focus may have preferential conduction through the Purkinje system, and it may change its exit in an antidromic direction when its orthodromic exit is blocked and vice versa. This is supported by the finding that 1 patient with superior axis VT developed inferior axis VT, which was induced after blocking the distal Purkinje conduction, as indicated by the blocked local Purkinje potential after ablation (Figure 6). Because NRFT originates from a focus within a mesh-like subendocardial Purkinje network, ablation of the earliest Purkinje focus, which was presumably the origin of NRFT, caused fragmentation and delay of that Purkinje focus (P′) from the local ventricular myocardium. This indeed resulted in successful NRFT elimination. Figure 6 provides the evidence of a local Purkinje–Purkinje (intra-Purkinje) block and Purkinje–myocardial exit block. After ablation, that site was activated by inputs from a different Purkinje arborization.

Another explanation for the high recurrence rate is that the recurrent VT may have originated from a residual Purkinje network proximal to the ablation site. Fascicular block has been reported in the majority of successfully ablated cases.3,5 In contrast, our strategy was to start ablation as distal as possible to the Purkinje arborization not only to avoid injuring the proximal fascicles but also to avoid creating iatrogenic VT in the proximal vicinity. Indeed, we have recently reported that 50% of upper septal ILVTs are because of ablation-related conduction disturbances that create a substantial substrate for reentrant VT.14 The optimal ablation site in NRFT is the site with the longest local fascicular–ventricular interval. As we had tried to ablate as distal as possible to avoid injuring the proximal fascicles, cases with recurrent VT might have had the longest local fascicular–ventricular interval more proximally.

Limitations
Some study limitations should be addressed. The most significant one is the sample size. However, this is the first study to demonstrate the prevalence of idiopathic NRFT; hence, our sample size reflects the rarity of the disease. Moreover, understanding the mechanism and electrophysiologic characteristics of NRFT may warrant the identification of unrecognized cases, especially those who may have been misdiagnosed as reentrant ILVT or papillary muscle VT. Therefore, the actual incidence of idiopathic NRFT may have been underestimated. Intracardiac echocardiography was used in only 6 cases; hence, contribution of endocardial structures to the tachycardia mechanism cannot be completely ruled out. Importantly, papillary muscle VT was certainly pointed out in all the spectrum of NRFT origin (intracardiac echocardiogram was used in 2 LAF origin, 3 LPF origin, and 1 RV-Purkinje origin NRFT cases). Finally, difficulty in inducing this type of VT is an important limitation that led to a relatively high recurrence rate after the first ablation session.

Conclusions
Approximately 3% of cases of idiopathic VT referred for catheter ablation were because of focal NRFT, which had distinct clinical characteristics and usually originated focally from the LPF, and less commonly from the left anterior fascicle and RV Purkinje arborization. This type of VT is verapamil resistant and can be induced by catecholamine infusion or burst ventricular pacing; however, it cannot be entrained. Obscurity of the ablation end point and the presence of multifocal VT represent important challenges in ablating this type of VT.

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Ahmed Karim Talib, Akihiko Nogami, Itsuro Morishima, Yasushi Oginosawa, Kenji Kurosaki, Shinya Kowase, Yuki Komatsu, Kenji Kuroki, Miyako Igarashi, Yukio Sekiguchi and Kazutaka Aonuma

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