The role of Purkinje fibers in the emergence of an incessant form of polymorphic ventricular tachycardia or ventricular fibrillation associated with ischemic heart disease

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Background: The clinical and electrophysiological characteristic of ventricular premature contractions (VPCs) which trigger the incessant form of polymorphic ventricular tachycardia (VT), so-called “electrical storm” associated with ischemic heart disease, remains unclarified. The aim of this study was to evaluate those matters and the possible role of the Purkinje network in the emergence of an electrical storm.

Methods and results: We experienced 5 patients (68 ± 5 years, mean LVEF: 29%) with electrical storms which occurred during the acute phase of an infarction in 3 patients and the remote phase in 2. The triggering VPCs were multifocal in 3 patients and monofocal in the remaining 2. Radiofrequency (RF) catheter ablation was performed for a goal of eliminating the triggering VPCs. A total of 9 different kinds of VPCs differentiated by their morphology were successfully eliminated by the RF deliveries targeting the VPCs’ foci. At the successful ablation sites, Purkinje potentials preceded the QRS onset of the VPC by 67 ± 23 ms, suggesting the VPCs originated in the surviving Purkinje fibers. Moreover, the extensive RF deliveries applied at the surviving Purkinje network rendered the polymorphic VT unable to be induced by programmed stimulation which reproducibly induced it before the ablation in 2 patients.

Conclusion: A surviving Purkinje network might contribute not only to the initiation of the repetitive form of lethal ventricular arrhythmias, but also to the perpetuation of the arrhythmias in patients with ischemic heart disease.

(J Arrhythmia 2008; 24: 200–208)

Key words: Electrical storm, Polymorphic ventricular tachycardia, Ventricular fibrillation, Purkinje potential, Catheter ablation

Introduction
It has been recently shown that the ventricular premature contractions (VPCs) arising from the His-Purkinje system trigger polymorphic ventricular tachycardia (VT) and ventricular fibrillation.
(VF) in a variety of clinical situations with structural heart disease, such as ischemic heart disease and cardiomyopathy, or without structural heart disease, such as Brugada syndrome and Long QT syndrome. These VPCs are also shown to be eliminated by radiofrequency catheter ablation from the left or right ventricular endocardium. In patients with ischemic heart disease, such a mechanism of the tachyarrhythmias often forms clusters of arrhythmias, which are so-called “electrical storms” and have been shown to be associated with a poor prognosis.

We experienced 5 patients with an electrical storm triggered by VPCs originating from surviving Purkinje fibers located in the ischemic lesion. In all patients, catheter ablation targeting the VPCs was effective and lead to the successful suppression of the electrical storm. The aim of the present study was to elucidate the clinical and electrophysiological characteristics of the incessant form of polymorphic VT or VF and their triggering VPCs in patients with ischemic heart disease. The possible role of the Purkinje fibers in the development and perpetuation of this peculiar form of arrhythmia will be discussed.

### Methods

#### Study population

The population of this study was comprised of 5 retrospectively collected patients with ischemic heart disease who experienced electrical storms of polymorphic VT or VF and underwent successful suppression of the electrical storm by catheter ablation for the triggering VPCs. These arrhythmias were successfully cured by catheter ablation targeting the VPCs which triggered the electrical storms.

The demographic data of these 5 patients is demonstrated in Table 1. All patients were male, and the average age was 68 ± 5 years. In 3 out of 5 patients the electrical storm arose during the acute phase of a myocardial infarction (4 hours, 1 day and 4 days, respectively). The remaining 2 patients were diagnosed with ischemic cardiomyopathy since the cardiac enzymes were not elevated at the time of admission due to the incessant appearance of ventricular tachycardia. One patient (patient No. 4) experienced repeated ventricular arrhythmias after aggravation of congestive heart failure, and the remaining patient (pt No. 5) had them immediately after coronary artery bypass grafting (CABG) surgery. The mean left ventricular ejection fraction was 29 ± 5% (range 20–33%), suggesting that all the

### Table 1 Clinical Demographic Data of the Patients

<table>
<thead>
<tr>
<th></th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
<th>Patient 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>67</td>
<td>71</td>
<td>74</td>
<td>60</td>
<td>67</td>
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<tr>
<td>Gender</td>
<td>Male</td>
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<tr>
<td>Diagnosis</td>
<td>AMI</td>
<td>AMI</td>
<td>AMI</td>
<td>ICM</td>
<td>ICM</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>32</td>
<td>20</td>
<td>29</td>
<td>32</td>
<td>33</td>
</tr>
<tr>
<td>Killip Class</td>
<td>III</td>
<td>III</td>
<td>I</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>NYHA Class</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>III</td>
<td>II</td>
</tr>
<tr>
<td>Infarction Site</td>
<td>anterior</td>
<td>inferior</td>
<td>anterior</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Coronary Artery Status</td>
<td>LAD occlusion</td>
<td>RCA occlusion</td>
<td>LAD occlusion</td>
<td>LCX occlusion</td>
<td>LAD occlusion</td>
</tr>
<tr>
<td>Intervention</td>
<td>PCI</td>
<td>CABG</td>
<td>PCI</td>
<td>PCI</td>
<td>CAGB</td>
</tr>
<tr>
<td>Interval between MI and VT</td>
<td>4 hours</td>
<td>1 day</td>
<td>4 days</td>
<td>—</td>
<td>—</td>
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<tr>
<td>VT or VF</td>
<td>PVT &amp; VF</td>
<td>PVT &amp; VF</td>
<td>PVT &amp; VF</td>
<td>PVT &amp; MVT</td>
<td>PVT &amp; MVT</td>
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<tr>
<td>Number of PVT/VF episodes</td>
<td>45</td>
<td>8</td>
<td>200</td>
<td>10</td>
<td>6</td>
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<tr>
<td>Number of DC deliveries</td>
<td>48</td>
<td>10</td>
<td>185</td>
<td>5</td>
<td>4</td>
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<tr>
<td>Anti-arrhythmic drugs (pre)</td>
<td>Mex, Lido</td>
<td>Nife, Lido</td>
<td>Nife, Lido, Proc</td>
<td>Nife, Mex, Lido</td>
<td>Nife, Lido</td>
</tr>
<tr>
<td>Anti-arrhythmic drugs (post)</td>
<td>—</td>
<td>AMD, Carv</td>
<td>AMD, Mex</td>
<td>AMD, Mex</td>
<td>AMD, Carv</td>
</tr>
<tr>
<td>ICD implantation</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Prognosis (F/U period, cause)</td>
<td>D (1M, sepsis)</td>
<td>D (1Y, stomach ca)</td>
<td>S (2Y)</td>
<td>S (2Y)</td>
<td>D (5M pneumonia)</td>
</tr>
</tbody>
</table>

patients had a severely reduced systolic function at the emergence of the electrical storm. The observed arrhythmias were polymorphic VT and VF in 3 patients (pts Nos. 1–3) and polymorphic VT which was spontaneously organized to monomorphic VT in 2 patients (pts Nos. 4 and 5). The average number of episodes of ventricular tachyarrhythmias was 54 ± 83 (range: 6–200) and that of direct current deliveries for life-saving cardioversion was 50 ± 77 (range: 4–185), respectively. In 2 out of 5 patients, the electrical storm occurred after a coronary intervention (1 percutaneous catheter intervention [PCI], and 1 CABG), and in the remaining patients PCI was indicated for the cure of the arrhythmias and did not suppress them.

Electrophysiologic study and catheter ablation procedures

Written informed consent for electrophysiologic (EP) study and catheter ablation was obtained from all the patients through legal representatives. The EP study was performed in the fasting state and all patients were sedated by an intravenous administration of midazolam. In all patients, these procedures were applied urgently to suppress the incessant appearance of the life-threatening ventricular tachyarrhythmias which 2–3 kinds of antiarrhythmic drugs failed to suppress (Table 1). Therefore, it was considered ethical to indicate such invasive diagnostic and therapeutic tools in the critically ill patients. Three out of the 5 patients were mechanically ventilated, and 2 were supported by intra-aortic balloon pumping at the beginning of the catheter ablation session. Multi-electrode catheters were inserted from the femoral veins and positioned at the right high atrium, His bundle region and right ventricular apex. Surface and intracardiac electrograms were continuously monitored and recorded using an EP-WorkMate (EP MedSystems, Inc, Mt. Arlington, NJ, USA) recording system at a filter setting of 30 to 500 Hz. Left ventricular mapping and catheter ablation were performed using a Navi-Star catheter with a 4-mm-tip electrode (Johnson & Johnson) in 2 patients (pts Nos. 2 and 3) and in the remaining 3 patients, a standard 4-mm-tip electrode catheter (7 Fr; RF Marinr, Medtronic Inc., Minneapolis, MN, USA) was used. In the former 2 patients (pts Nos. 2 and 3), the electrogram mapping and substrate mapping were constructed using a 3-D electro-anatomical mapping system (CARTO, Johnson & Johnson). Pace mapping was used to identify the origin of the VPCs in the LV in 4 patients, and activation mapping was also attempted in all patients. In this case, the earliest activation (earliest Purkinje potentials observed) was searched. The catheter ablation was performed with a radiofrequency (RF) generator (Atakr RF Power generator, Medtronic). The ablation target sites for the triggering VPCs were determined mainly by referring to the activation map, namely, how the Purkinje potential preceded the QRS complex during the spontaneous VPCs, with supplementary information from the pace-mapping. For the pace-mapping, the QRS configuration of each of the 12-lead ECG was compared with that of the VPCs, and scored with the following criteria (Pace-map score): Score 1, a QRS configuration showing complete matching; score 0.5, the polarity of the QRS complex was same, but minor deflections were not similar; score 0, the polarity of the QRS complex was different. Then, the scores of each lead were summed. Radio-frequency (RF) current was delivered in the temperature control mode (60°C) with a maximum power setting of 50 W. If the VPCs which triggered the polymorphic VT or VF were suppressed, the RF current was delivered for a maximum of 120 seconds. The goal of the catheter ablation was set at the elimination of the triggering VPCs in all patients. In 2 patients (pts Nos. 2 and 3), multiple RF current deliveries were repeatedly applied on the sites where survived Purkinje potential was detected in the infarction region. Consequently, the polymorphic VT became non-inducible by programmed ventricular pacing in these 2 patients.

Statistical analysis

The data are expressed as the mean ± standard deviation for continuous variables and as the frequency (%) for categorized variables.

Results

1) ECG morphology of the VPCs triggering polymorphic VT (Electrical storm)

In 3 of the 5 patients, 2 or more morphologies of the VPCs were documented to have induced polymorphic VT (2, 3 and 2 for pts Nos. 1, 2 and 3, respectively). The remaining 2 patients (pts Nos. 4 and 5) had only a single morphology of the VPCs triggering the VT (Table 2). A total of 9 morphologies of the VPCs were recorded to have induced ventricular tachyarrhythmias while monitoring the ECGs in the 5 patients. All of the VPCs exhibited a right bundle branch block (RBBB) morphology suggesting a left ventricular origin, and either a superior axis (6 VPCs) or inferior axis (3 VPCs). The mean QRS duration of the triggering VPCs was 133 ± 22 ms (range: 100–170 ms), suggesting
Figure 1
Representative records of the spontaneous occurrence of the polymorphic VT triggered by a VPC (indicated by arrow) with relatively narrow QRS complex of right bundle branch block morphology and left axis deviation in patient 4 in the left panel (A) and in patient 5 in the right panel (B), respectively.

Table 2  ECG and Electrophysiological Data of the Patients

<table>
<thead>
<tr>
<th>VPCs triggering PVT/VF</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
<th>Patient 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of morphology</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>QRS Morphology (VPCs)*</td>
<td>RBBB, Sup, 100</td>
<td>RBBB, Sup, 140</td>
<td>RBBB, Sup, 120</td>
<td>RBBB, Sup, 130</td>
<td>RBBB, Sup, 170</td>
</tr>
<tr>
<td>VPC 1</td>
<td>RBBB, Sup, 115</td>
<td>RBBB, Sup, 140</td>
<td>RBBB, Sup, 120</td>
<td>RBBB, Sup, 130</td>
<td>RBBB, Sup, 170</td>
</tr>
<tr>
<td>VPC 3</td>
<td>—</td>
<td>RBBB, Inf, 130</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Origins of VPCs</td>
<td>VPC1:MS</td>
<td>VPC1:MS, VPC2:PS</td>
<td>VPC1:PS</td>
<td>VPC1:PS</td>
<td>VPC1:MS</td>
</tr>
<tr>
<td>Pacemap score</td>
<td>VPC1:12</td>
<td>VPC1:12, VPC2:11.5</td>
<td>VPC1:11</td>
<td>VPC1:12</td>
<td>VPC2:11</td>
</tr>
<tr>
<td>No. of RF energy deliveries</td>
<td>10</td>
<td>24</td>
<td>61</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Achieved ablation results</td>
<td>VPC elimination</td>
<td>Non-inducibility</td>
<td>Non-inducibility</td>
<td>VPC elimination</td>
<td>VPC elimination</td>
</tr>
<tr>
<td>Purkinje potential (Pp)** during VPCs (ms)</td>
<td>VPC1: 55, VPC2: 45</td>
<td>VPC1:40, VPC2:60</td>
<td>VPC1: 62, VPC2:60</td>
<td>VPC1: 58</td>
<td>VPC1: 100</td>
</tr>
</tbody>
</table>

VPC: Ventricular premature contraction; PVT: polymorphic ventricular tachycardia; VF: ventricular fibrillation; RBBB: right bundle branch block; MS: midseptal; PS: posteroseptal; AS: anteroseptal; NA: not applied

*ECG morphology of triggering VPCs include QRS morphology (LBBB or RBBB pattern), QRS axis (superior or inferior axis) and QRS duration (ms).

The time phase of Purkinje potential was assessed at the successful ablation sites for VPCs.

In both patients, the triggering VPCs were successfully eliminated by a small number of RF deliveries applied at the posteroseptal region where the Purkinje potential preceded the QRS complex by 58 ms in patient 4, and midseptal region where the Purkinje potential preceded the QRS complex by 100 ms in patient 5.

In patient 1, a severe electrical storm which required a total of 48 counter shocks led us to perform a rescue catheter ablation. In that session, the origin of the triggering VPCs was searched along the left posteroseptal region, using the electrogram mapping and pace mapping methods. As shown in Figure 2, the pace mapping revealed an almost complete match of the QRS morphology between the electrogram during pacing and that of VPC #1 in the standard 12 lead ECG (2B and 2C). It was noted that there was significant latency (approximately 50 ms) between the pacing spike and QRS complex (2C), which corresponded well to the Purkinje potential-QRS onset interval at that site (P-QRS = 55 ms;2D). An RF current delivery applied at that point eliminated VPC #1. Because the pace mapping was not effective in finding the origin of VPC #2 (the best pace map was 10.5 points), a total of 10 RF current deliveries were applied along the mid-apical
region of the left posterior septum under electrogram guidance. An RF delivery applied at the site where the Purkinje potential-QRS onset of VPC #2 was 45 ms resulted in the disappearance of both VPCs.

In a patient with 3 different QRS morphologies of the triggering VPCs (pt No. 2), an EP study and catheter ablation were performed using a CARTO mapping system. Figure 3 shows the actual surface and intracardiac recordings during a spontaneous emergence of ventricular tachycardia triggered by the 3 kinds of VPCs with different morphologies. Polymorphic VT could be easily induced by programmed stimulation from the left ventricular septum. Figure 4 shows such a recording during the provocation by double extrastimuli. It was noted that the time phase and morphology of the Purkinje potential of the initial several beats exhibited beat-to-beat changes. The catheter ablation initially targeted the origin of the 3 VPCs indicated in Figure 5 by the arrows, and it successfully decreased the frequency of the VPCs. Then, the RF current

Figure 2

(A) A record of monitoring ECG during the emergence of polymorphic VT which is induced by a VPC (indicated by arrow) with relatively narrow QRS complex in the patient 1.
(B) A standard 12-lead ECG record of the VPC which repeatedly triggered the polymorphic VT.
(C) ECG record during the pacing from a successful ablation site for the VPC, showing almost complete match of QRS morphology with that of the VPC (panel B). There is significant latency after the pacing spike to the QRS onset, that is approximately 50 ms.
(D) An intracardiac record during the spontaneous occurrence of the VPC from the successful ablation site. From top, leads I, aVF and V1 and intracardiac records from the distal pair of mapping catheter and multiple electrodes’ pair of decapolar catheter which was positioned along the anatomical running of the right sided specialized tissue (His bundle-right bundle branch) are shown. It is noted that a Purkinje potential preceded the QRS onset by 55 msec. which corresponds well to the latency during the pacing from this site suggesting that the pacing was supposed to result in a selective capture by the Purkinje fiber. It is also recognized that Purkinje potential preceded both His bundle and right bundle branch activations.

applications were repeatedly delivered to the points where a Purkinje potential was detectable in the infarct zone (dark-brown dots in Figure 5). A total of 24 RF deliveries rendered the polymorphic VT unable to be induced. After the catheter ablation, a non-sustained ventricular tachycardia which was not observed clinically was induced only once by a triple extrastimuli applied from the interventricular septum (Figure 4B). Similarly, repeated RF currents deliveries applied extensively to the surviving Purkinje fibers under the guidance of CARTO system made the tachycardia non-inducible in patient 3, in which the programmed pacing was applied from the left ventricular septum.

Figure 3
ECG and intracardiac records (Map) from the left anterior interventricular septum during the spontaneous emergence of the clinical VTs in the patient 2 induced by three types of VPCs with different QRS morphologies (VPC #1, #2, #3). The Purkinje potentials precede the QRS onset of VPC #1, #2 and #3 by 10 ms, 5 ms and 50 ms, respectively. Pp: Purkinje potential.

Figure 4
(A) Body-surface ECG and intracardiac records during the provocation of clinical VT by double extrastimuli (S1–S2 interval: 340 ms, S2–S3 interval: 270 ms). Note that the timing and morphology of the Purkinje potential (Pp: indicated by arrows) changed in a beat-to-beat fashion during the initial consecutive beats of induced tachycardia.
(B) After the extensive catheter ablation targeting the surviving Purkinje fibers, the clinical polymorphic VT was no longer inducible, instead a monomorphic ventricular repetitive activity was induced only once by an aggressive triple extrastimuli applied at same site (S1–S1 interval: 400 ms, S1–S2: 240 ms, S2–S3: 240 ms, S3–S4: 240 ms).
3) Acute consequences of catheter ablation, and follow-up data (Table 2)

In all 5 patients, the catheter ablation procedures successfully suppressed the electrical storms with a mean of 21 ± 24 applications of RF energy. The sites where the triggering VPCs were eliminated by the RF deliveries were located in the posterior-septum for 5 VPCs, mid-septum for 3 VPCs and anterior-septum for the remaining 1 VPC (Table 2). The pace-mapping score for the VPCs ranged from 10.5 to 12 points (average: 11.5 points). The Purkinje potential-QRS interval at the successful ablation sites for the VPCs ranged from 40 to 100 ms (average: 67 ± 23 ms). The final achieved results of the ablation procedures were the elimination of the VPCs in 3 patients and non-inducibility of the sustained ventricular tachyarrhythmias in the remaining 2 patients.

All patients were discharged from the Coronary Care Unit, however one patient (pt No. 1) died of sepsis on the general ward (Table 1). The remaining 4 patients were discharged from the hospital under the administration of antiarrhythmic drugs with (3 pts) or without (1 pt) an implantable cardioverter defibrillator (ICD). Out of those, one patient died of stomach cancer 1 year after being discharged and another died of pneumonia 5 months after discharge. There were no appropriate shocks delivered in the patients with an ICD, nor any documentation of sustained ventricular tachyarrhythmias during the follow-up period.

Discussion

The main findings in this study are as follows. The electrical storms occurring in the patients with ischemic heart disease could be successfully suppressed by catheter ablation targeting the origins of the VPCs which triggered the sustained VT/VF. Additional RF deliveries applied at the surviving Purkinje fibers located in the extensive infarction zone may have achieved another endpoint, i.e., the non-inducibility of the sustained VT or VF.

The Purkinje fibers have been shown to survive in the event of a coronary occlusion in both an experimental animal model11,12) and after a myocardial infarction in humans. 13) Multiple mechanisms of such a greater tolerance of Purkinje fibers to the ischemia as compared with regular muscle cells have been proposed. First, Purkinje tissue is commonly located at the surface of the endocardium, and thus the necessary oxygen and nutrition are supplied via diffusion or collaterals from the left ventricular cavity.14,15) Second, Purkinje cells contain more nutrition stores (glycogen) than the regular muscle cells. Finally, the Purkinje cells do not contain less myofibrils, and therefore their oxygen demand is smaller than that of the regular muscle cells.16) Although Purkinje cells are likely to survive over severe coronary events, they undergo an unignorable histological change caused by the ischemia, leading to the formation of an arrhythmogenic substrate as a consequence of significant electrophysiological
modifications, such as a decrease in the resting membrane potential and $|\dot{V}_{\text{max}}|$. In our patients with myocardial infarctions, distinct spiky potentials were detectable from a wide range of sites on the left ventricular septum (Figure 5A) where the ventricular potentials exhibited a decreased amplitude and fragmented activity, suggesting the location was inside the infarction zone. Those potentials are most likely to have reflected the electrical activation of surviving Purkinje fibers.

In animal studies using a myocardial infarction model, the ventricular arrhythmias have been shown to originate from the subendocardial Purkinje fibers in both the phase I and Phase II post-coronary occlusion periods.\(^{17-19}\) Arner et al. previously reported the results of their 3-dimensional evaluation of the origin of ventricular tachycardia by extended transmural mapping utilizing multiple plunge electrode catheters over the infarction and border zone in the canine coronary occlusion model. They clearly demonstrated\(^{19}\) that most (80%) ventricular tachycardias occurring during the acute phase of an infarction (10–30 minutes after the coronary occlusion) exhibited a focal activation pattern and originated in the Purkinje tissue. The same group also demonstrated\(^{20}\) using a similar experimental model that in more than half of the dogs (58%) with documented reperfusion arrhythmias, those arrhythmias originated in the Purkinje tissue. Since the VPCs triggering an electrical storm also exhibited focal activation patterns from the Purkinje fibers in our patients, and since the electrical storm emerged in the acute phase of the infarction or after coronary artery intervention or CABG surgery, the mechanism of the VPCs might be analogous to that experimental model in which the authors speculated that the mechanism may be triggered activity.

Up to the present, there have been several reports on the successful suppression of electrical storms by catheter ablation procedures targeting the VPCs triggering either VT or VF.\(^{1-5}\) Bansch et al.\(^{2}\) reported 4 cases of electrical storms that were cured by catheter ablation targeting the triggering VPCs with a relatively narrow QRS complex (120–160 ms), which corresponds to our data (Table 2). All of the patients experienced the electrical storms in the acute phase of a myocardial infarction. Since the authors confirmed the suppression of the VPCs either by overdrive pacing at a rate of $<120\text{ bpm}$ or by an intravenous administration of adenosine, it was speculated that triggered activity was the underlying mechanism of the VPCs. It was also shown that the VPCs were not suppressed by the intravenous administration of class I antiarrhythmic agents, which is similar to our observation. The triggering VPCs were successfully eliminated by ablation targeting the anteromedial region in 2 patients and inferomedial region in the remaining 2 patients, where Purkinje potential preceded the QRS complex by a maximum of 120–160 ms during VPCs. Szumowski et al.\(^{3}\) demonstrated 5 cases of electrical storms which occurred during the acute phase of a myocardial infarction in 3 and remote phase in the remaining 2 patients. The authors also confirmed that either VT or VF was triggered by the VPCs originating in the Purkinje tissue of the myocardial infarction region or the border zone, since the Purkinje potential preceded the QRS complex by 20 to 160 ms during VPCs, that is consistent with our data (Table 2). It is noteworthy that the repetitive Purkinje activity was observed to precede each QRS complex during the initiation of the VT, and the morphology and polarity of the Purkinje potentials changed in a beat-to-beat manner. Thus they speculated a possible role of the Purkinje network in both the initiation and maintenance of the polymorphic VTs. We also confirmed similar findings during the initiation of the polymorphic VTs. Moreover, it was clearly shown in our study that successive RF deliveries applied to the surviving Purkinje fibers over the extensive infarction scar could render the polymorphic VT and VF unable to be induced by programmed stimulation in 2 patients. This might be further evidence of the contribution of the Purkinje network not only in the initiation, but also the perpetuation of the ventricular arrhythmias. Thus, the maintenance of polymorphic VT might, at least in the initial part, be due to a reentry mechanism over the Purkinje network with multiple Purkinje-muscle connections, expressing the polymorphism of the QRS complex.

**Study limitation**

Because the number of patients was small in this study, it might an overestimation to draw a final conclusion that the extensive ablation of the surviving Purkinje network may have modified the arrhythmia substrate in addition to eliminating the trigger. However, we could demonstrate that the polymorphic VT that was reproducibly inducible before the ablation became non-inducible after the extensive ablation targeting the surviving Purkinje fibers, suggesting that the arrhythmia substrate was modified.

The region of the ablation target was almost always located inside the infarction zone, therefore it was unlikely that the repeated RF deliveries influenced the cardiac function itself.
Acknowledgment

The authors express sincere gratitude to Murata H MD, Yamamoto T MD, Okazaki R MD, Tsutomu Horie MD, Junko Abe MD, Yasuhiro Hirasawa MD, Mitsunori Maruyama MD and Toshihiko Ohara MD, for their technical assistance, and Mr. John Martin for his linguistic assistance.

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