Catheter Ablation of Paroxysmal Atrial Fibrillation Initiated by Non–Pulmonary Vein Ectopy

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**Background**—Most of the ectopic beats initiating paroxysmal atrial fibrillation (PAF) originate from the pulmonary vein (PV). However, only limited data are available on PAF originating from the non-PV areas.

**Methods and Results**—Two hundred forty patients with a total of 358 ectopic foci initiating PAF were included. Sixty-eight (28%) patients had AF initiated by ectopic beats (73 foci, 20%) from the non-PV areas, including the left atrial posterior free wall (28, 38.3%), superior vena cava (27, 37.0%), crista terminalis (10, 3.7%),ligament of Marshall (6, 8.2%), coronary sinus ostium (1, 1.4%), and interatrial septum (1, 1.4%). Catheter ablation eliminated AF with acute success rates of 63%, 96%, 100%, 50%, 100%, and 0% in left atrial posterior free wall, superior vena cava, crista terminalis, ligament of Marshall, coronary sinus ostium, and interatrial septum, respectively. During a follow-up period of 22±11 months, 43 patients (63.2%) were free of antiarrhythmic drugs without AF recurrence.

**Conclusions**—Ectopic beats initiating PAF can originate from the non-PV areas, and catheter ablation of the non-PV ectopy has a moderate efficacy in treatment of PAF. (Circulation. 2003;107:3176-3183.)

Key Words: atrial fibrillation • ablation • pulmonary vein

Recent studies have demonstrated that pulmonary vein (PV) is the major site of ectopic foci initiating paroxysmal atrial fibrillation (PAF), and isolation of PV from atrial tissue can cure 60% to 70% of patients with PAF.1–4 Several studies also addressed the importance of non-PV ectopic beats initiating PAF. The non-PV ectopic beats may arise from the superior vena cava (SVC), left atrial posterior free wall (LPFW), crista terminalis (CT), coronary sinus ostium (CSO), ligament of Marshall (LOM), and interatrial septum (IAS).5–9 However, the information about PAF originating from the non-PV area is limited, and the long-term follow-up results of catheter ablation in these patients are not available.

The present study describes the electrophysiological features and results of radiofrequency ablation in a large group of patients with AF initiated by ectopic beats originating from the non-PV areas.

**Methods**

**Study Patients**

This report included 68 patients (43 male and 25 female, age 61±13 years) with clinically documented episodes of PAF. AF was proven to be initiated by ectopic beats from the non-PV area. They were refractory to or intolerant of 2.8±1.2 antiarrhythmic drugs before the catheter ablation procedure.

**Electrophysiological Study**

Each patient underwent the electrophysiological study in the fasting, nonsedated state after written informed consent was obtained; all antiarrhythmic drugs except amiodarone were discontinued for at least 5 half-lives before the study.2,5,6,8 We tried to find ectopic beats initiating AF before or after infusion of isoproterenol or followed the previously designed algorithm used for facilitating initiation of AF.2 If a consistent ectopic focus and onset pattern of spontaneous AF was confirmed, the earliest ectopic site was considered to be the initiating focus of AF.2,6,8

**Mapping**

Mapping of the PVs was guided by selective PV angiography or the venous phase of selective pulmonary artery angiography, with the first pair of electrodes straddling the ostium; the catheters were first put into superior PVs and then the inferior PVs if the ectopic focus was suspected to be from the inferior PVs.2,5,6,8

If the initiating focus of AF was considered to be from the right atrium, we put 1 duodecapolar catheter (1-mm electrode length and 2-mm interelectrode spacing) along the crista terminalis to reach the atrio caval junction area or the superior vena cava (SVC) for simultaneous mapping of the PVs and SVC. The SVC mapping catheter was advanced to the site with the most distally recorded

**References**


electrogram amplitude larger than 0.05 mV. The SVC orifice or SVC right atrium junction was determined fluoroscopically during SVC angiography. We determined the region of earliest activation in sinus rhythm, which was characterized by the initial negative rapid deflection or fusion of major atrial electrogram and the SVC muscular potential. If the atrial and SVC anatomic site were not clear, intracardiac ultrasound imaging (Boston Scientific Co) was used to delineate the atrio caval junction area in comparison with angiographic localization. In 7 patients with ectopic beats from SVC, a basket catheter with 64 electrodes (Boston Scientific Co) was put in the SVC for mapping and guidance of ablation. In patients with ectopic beats from the LOM, double potentials (DPs) are present at the orifice of or inside the left PVs, and distal CS pacing can help differentiate the LOM potential from the PV musculature potential. If the second deflection (D2) of DPs is attributable to activation of LOM, the CS ostium→D2 interval will be shorter during distal CS pacing compared with sinus rhythm. In contrast, if it is attributable to activation of the PV musculature, the CS ostium→D2 interval will be longer during distal pacing compared with sinus rhythm. In some patients with typical atrial flutter, a 7F, 20-pole, deflectable halo catheter with 10-mm paired spacing (Cordis-Webster Co) was positioned around the tricuspid annulus for mapping simultaneously.

**Catheter Ablation**

The presumed ablation site of non-PV ectopy from SVC, coronary sinus, atrial septum, and atrial free wall showed the earliest biphasic activity or a local unipolar QS pattern of the ectopic beats preceding AF recorded from the ectopic foci. For the ectopy from LOM, the earliest LOM activation potential preceding the onset of spontane-ous AF was targeted for ablation. For the ectopy from LPFW, the earliest LOM activation potential preceding the onset of spontane-ous AF was targeted for ablation. For the ectopy from LPFW, point ablation of the earliest activation site was tried first; if ectopy initiating AF still persisted, a box-shaped (1.5×1.5-cm square area) linear ablation was performed around the ectopy. For the 7 patients with the basket catheter facilitating mapping of SVC ectopy, segmental isolation was performed from the site proximal to SVC ectopy foci. The ablation catheter (4-mm tip electrode, Boston Scientific) was connected to an EPT-1000 generator (EP Technologies) delivering a 550-kHz sine wave output. Temperature-controlled (target temperature 50 to 55°C) radiofrequency energy was delivered for 20 to 40 seconds per pulse, but it was terminated immediately if the ablation catheter displaced or the patient complained of burning pain, coughed, or developed severe bradycardia. The ablation end point was total elimination or marked reduction (<50% of the initial amplitude) of ectopic focus electrogram amplitude. For the 7 patients with basket catheter mapping of SVC ectopy, the disappearance of ectopy and distal SVC potential was the end point. The protocols used to facilitate AF onset before ablation were repeated to assess the effects of radiofrequency ablation immediately after and 10 to 15 minutes after the last application of radiofrequency energy.

**Postablation Follow-Up**

Close clinical follow-up (2 weeks, 1 month, and then every 2 to 4 months) was arranged after ablation. If the patients experienced palpitation, 24-hour Holter monitoring or event recorder was performed to define the cause of tachycardia. Long-term follow-up information also was obtained from the operators, referring physicians, and through telephone interviews with the patients. If the patients agreed, the second electrophysiological study or catheter ablation was performed to identify the true cause of palpitation.

**Statistical Analysis**

Parametric data were presented as mean±1 standard deviation and were analyzed by t test or ANOVA, as appropriate. Nonparametric data were analyzed by the χ² test with Yates’ correction or Fisher’s exact test. P<0.05 was considered statistically significant.

**Results**

**Clinical Characteristics and Ablation Results**

Among the total 68 patients (28% of 240 patients) with 73 ectopic foci, 23 had structural heart disease, including 5 with coronary artery disease, 16 with hypertensive cardiovascular disease, 1 with hypertrophic obstructive cardiomyopathy, and 1 with previous repair of ventricular septal defect. The ectopic foci were in the LPFW (n=28, 38.3%), SVC (n=27, 37%), CT (n=10, 13.7%), LOM (n=6, 8.2%), CSO (n=1, 1.4%), and IAS (n=1, 1.4%) (Table 1). After follow-up of 22±11 months, 63.2% of patients remained in sinus rhythm without antiarrhythmic drugs.

**PAF Initiated by LPFW Ectopy**

**Patient Population**

All of the 27 patients had multiple AF foci, and they were from left superior pulmonary vein (LSPV) (n=19, 48.7%), right superior pulmonary vein (RSPV) (n=15, 38.5%), right middle pulmonary vein (RMPV) (n=2, 5.1%), right inferior pulmonary vein (RIPV) (n=2, 5.1%), and IAS (n=2, 2.6%). In 5 patients, LPFW ectopy initiating AF appeared after ablation of PV ectopy.

**LPFW Activity During Sinus Rhythm and Ectopy**

During sinus rhythm, the LPFW atrial potentials with a rapid deflection were recognized and fused with the local PV potential at the PV ostium. A-PV potential could be found in the earliest ectopic beat and initiated atrial fibrillation. Alternating A-PV and PV-A potentials could also be found (Figure 1A). Two patients had spontaneous LPFW-AF, 14 patients needed isoproterenol infusion with pacing-triggered ectopic beat to induce LPFW-AF, and 17 patients had LPFW-AF after electrical cardioversion of AF. We divided the LPFW into 2 areas, near right side or left side PVs; 15 ectopic foci

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**TABLE 1. Clinical and Electrophysiological Characteristics and Ablation Results in Patients With PAF Initiated by Non-PV Ectopy**

<table>
<thead>
<tr>
<th>Group</th>
<th>Patients, n</th>
<th>Age, y</th>
<th>History, y</th>
<th>Other SHD, %</th>
<th>LA Size, mm</th>
<th>Multiple AF Foci, %</th>
<th>Late Recurrence, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>LPFW</td>
<td>27</td>
<td>63±14</td>
<td>5.2±4.0</td>
<td>50</td>
<td>39.5±5.9</td>
<td>100</td>
<td>56</td>
</tr>
<tr>
<td>SVC</td>
<td>27</td>
<td>57±12</td>
<td>4.7±4.8</td>
<td>22</td>
<td>36.8±5.1</td>
<td>44</td>
<td>26</td>
</tr>
<tr>
<td>CT</td>
<td>10</td>
<td>63±12</td>
<td>4.1±3.2</td>
<td>0</td>
<td>29.7±5.0</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>LOM</td>
<td>6</td>
<td>66±13</td>
<td>3.1±2.5</td>
<td>50</td>
<td>41.3±1.5</td>
<td>83</td>
<td>50</td>
</tr>
<tr>
<td>CSO</td>
<td>1</td>
<td>67</td>
<td>1</td>
<td>0</td>
<td>...</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>IAS</td>
<td>1</td>
<td>44</td>
<td>2</td>
<td>100</td>
<td>...</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

SHD indicates structural heart disease.
were near right side PV area, and 13 ectopic foci were near left side PV area.

Effect of Radiofrequency Ablation
Sixteen patients received catheter ablation of LPFW ectopy, and 10 (63%) ectopic foci were completely eliminated. One patient developed pericardial effusion during the ablation procedure, and it resolved after pericardiocentesis in the laboratory. After long-term follow up, only 12 (44%) patients were free of antiarrhythmic drugs without AF recurrence.

PAF Initiated by SVC Ectopy

Patient Population
Fifteen patients (55.6%) had only SVC foci (group A), and 12 patients (44.4%) had AF from SVC and extra-SVC foci (group B), including RSPV (n=8, 47%), LSPV (n=4, 23.5%), LIPV (n=2, 11.8%), CT (n=1, 5.9%), RMPV (n=1, 5.9%), and LOM (n=1, 5.9%). All of the SVC ectopy initiating AF were diagnosed before ablation of the associated AF foci.

Figure 1. A, Initiation of AF from PV ostium and left atrial posterior free wall. The first beat is sinus beat, the second beat is premature beat originating from LSPV-2 with conduction to the atrial tissue, the third beat is sinus beat, and the fourth beat is atrial premature beat originating from the atrial tissue (A) with conduction to the PV. Alternating activation from PV ostium and atrial wall was noted too. Ectopy beats from other PVs were excluded, because we have put the mapping catheters in the other 3 PVs for comparison with atrial ectopy. B, Ectopic beat originating from the LOM. The LOM potential can be found in the ablation catheter, and the ablation catheter is outside the ostium of the LSPV (LSPV-O). The typical triple potentials (LOM-A-PV) were noted. C, Diminished LOM potential after RFA. LSPV-1, -2, and -3 indicate the first, second, and third pairs of electrodes in the LSPV; ABL, ablation catheter; and CSD, distal coronary sinus.

Figure 2. Initiation of AF from superior vena cava (SVC). A, Bigeminal ectopic beats (arrow) followed by initiation of tachycardia in the surface ECG. B, Ectopic foci initiating tachycardia from SVC-D (distal) and conducting to SVC-P (proximal). C, Ectopic beats initiating AF from SVC after isoproterenol infusion (basket catheter recording inside SVC, with the earliest ectopic beat from E3 and F3).
**SVC Activity During Sinus Rhythm and Ectopy**

During sinus rhythm, the SVC potentials with a rapid deflection (duration \(<50 \text{ ms}, \text{amplitude } >0.05 \text{ mV}\) were recognized along the SVC in a proximal-to-distal activation sequence above the junction of the SVC and right atrium. The SVC potential was fused with the local atrial electrogram at the ostium (Figure 2). Two patients had spontaneous SVC-AF, 11 patients needed isoproterenol infusion with pacing-triggered ectopic beat to induce SVC-AF, and 15 patients had SVC-AF after electrical cardioversion of AF. In comparison of the 2 groups, there were no differences in LA size (33.3±4.4 versus 39.2±4.4 mm), SVC-O diameter.

**TABLE 2. Clinical and Electrophysiological Characteristics and Ablation Results in Patients With SVC-AF**

<table>
<thead>
<tr>
<th>Ectopy</th>
<th>Patients, n</th>
<th>AGE, y</th>
<th>SHD, %</th>
<th>LA Size, mm</th>
<th>SVC-O Diameter, mm</th>
<th>SVC Myocardial Ectopy Above M/S Late Ectopy</th>
<th>Ectopy Above SVC-O, mm</th>
<th>M/S Foci</th>
<th>Late Recurrence, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVC</td>
<td>15</td>
<td>55±12</td>
<td>6.7</td>
<td>33.3±4.4</td>
<td>27.5±4.3</td>
<td>31.6±6.9</td>
<td>25.3±9.7</td>
<td>2/13</td>
<td>13.3</td>
</tr>
<tr>
<td>SVC-others</td>
<td>12</td>
<td>61±12</td>
<td>50*</td>
<td>39.2±4.4</td>
<td>25.5±5.7</td>
<td>36.2±17.8</td>
<td>29±19.9</td>
<td>2/10</td>
<td>33.3</td>
</tr>
</tbody>
</table>

SHD indicates structural heart disease; PA, posterior-anterior fluoroscopic view; LA, lateral fluoroscopic view; M/S foci, multiple/single foci; and SVC-O diameter, SVC orifice diameter in the junction with right atrium.

\*P<0.05.

**AF: Multiple Migrating Foci with Conduction Block within SVC**

**Figure 3.** Basket catheter recording inside SVC during AF. A, The first beat is sinus beat, and the second beat is ectopic beat from SVC-B1 and SVC-C2, followed by SVC-A2 (arrowheads). B, After termination of SVC-AF, reinitiation of ectopic beats from SVC-B1, SVC-C2, and SVC-A1 (arrowheads). The coupling intervals of SVC depolarizations in SVC-B1 and SVC-C2 were significantly shorter than the proximal part (SVC-B4 and SVC-C4); this means conduction block between distal and proximal SVC. Furthermore, the distal bipole of D spine (D1, D2, and D3) in the posterolateral wall do not show SVC potentials. C, Another case with burst activities from SVC. Arrowheads show the earliest ectopic beats changed from A2, A1, and A2 to B2. D and E, AF termination during segmental isolation of the proximal SVC-right atrium area, but concealed discharge from SVC is still present (black spot). In the inset drawing in the upper right corner, A indicates anterior wall of SVC; D, posterolateral wall of SVC. 1, 2, 3, and 4 are the bipolar recordings from distal to proximal SVC.
(27.5±4.3 versus 25.5±5.7 mm), SVC myocardial sleeve length (31.6±6.9 versus 36.2±17.8 mm), or number of SVC foci (multiple versus single was 2 versus 13 in group A and 2 versus 10 in group B, respectively). However, the patients with SVC and extra-SVC ectopic foci had a higher incidence of structural heart disease (6.7% versus 50%, \(P<0.05\)) (Table 2). In the 7 patients with a basket catheter mapping of the SVC, a mean of 2±1 splines in the posterior wall did not show SVC potential or only showed low voltage of SVC potential (<0.05 mv). SVC conduction block and concealed discharge were also noted in the 7 patients (Figure 3).

**PAF Initiated by CT Ectopy**

**Patient Population**

Initiation of PAF by ectopic beats after isoproterenol infusion originating from the CT was demonstrated. Six patients had only CT ectopy, and another 4 patients (40%) had multiple AF ectopic foci, including 2 patients associated with SVC ectopy, 1 patient associated with RSPV ectopy, and 1 patient associated with both RSPV and LSPV ectopy. The locations of 10 ectopy were in the high (n=5), middle (n=3), and low (n=2) CT.

**CT Activity During Sinus Rhythm and Ectopy**

During sinus rhythm, the double potentials with a rapid deflection were recognized along the CT in a high-to-low activation sequence. Reversal of the double potentials was noted during the atrial premature beat (Figure 4).

**Effect of Radiofrequency Ablation**

AF from the CT ectopic foci were completely eliminated. No complication was noted during the ablation procedure. After long-term follow-up, 1 of the 6 patients with only CT ectopy and 1 of the 4 patients with other AF ectopy had AF recurrence; thus, 8 patients (80%) were free of AF without any antiarrhythmic drugs.

**PAF Initiated by LOM Ectopy**

**Patient Population**

Five patients (83%) had multiple ectopic foci, including 3 patients with LSPV ectopy, 1 patient with LIPV ectopy, and 1 patient with both LSPV and LIPV ectopy.

**LOM Activity During Sinus Rhythm and Ectopy**

Initiation of PAF by ectopic beats (after isoproterenol infusion) originating from the LOM was demonstrated. During sinus rhythm, the LOM potentials could be found near the LSPV ostium (n=5) or inside the LSPV (n=1), and reversal of triple potentials sequence was demonstrated (Figures 1B, 1C, and 5). Continuous bursts of atrial premature beats with fibrillatory conduction in the atria, or degenerating to sustained AF, was observed.

**Effect of Radiofrequency Ablation**

Three LOM ectopic foci were completely eliminated. The other 3 LOM ectopic foci were only partially eliminated and still showed burst depolarizations. No complication was noted during the ablation procedure. After long-term follow-up, 3 patients (50%) were free of antiarrhythmic drugs without AF recurrence.

**PAF Initiated by CSO and IAS Ectopy**

Only 1 patient (man, refractory to 2 antiarrhythmic drugs) had PAF by ectopic beats (after isoproterenol infusion plus pacing-triggered ectopy) originating from CSO. The CSO ectopy was eliminated completely. No complication or recurrence was noted. Only 1 patient (woman, refractory to 2 antiarrhythmic drugs) had initiation of PAF by ectopic beats (after isoproterenol infusion plus pacing-triggered ectopy).
originating from the IAS. She also had other ectopic foci from RSPV and LPFW. The IAS potential could not be eliminated completely. No complication was noted during the procedures.

Discussion

Electrophysiological Characteristics of AF from the Non-PV Areas

Previous embryological studies have demonstrated that the sinoatrial node is derived from the sinus venosus, and the other remnants of the embryonic sinus venosus are present in several areas of the mammalian heart, including the musculature of the SVC, CS, venous valve, and an area embedded in the proximal sulcus terminalis.10

Superior Vena Cava

The proximal SVC contains cardiac muscles connected to the right atrium, and atrial excitation or sinus node impulse can propagate into the SVC.11–13 SVC cardiomyocytes were found to have pacemaker activity, and the enhanced automaticity and afterdepolarization play a role in the arrhythmogenic activity of SVC.14 This clinical study showed that all the 7 patients who received 3D mapping of SVC had less myocardial tissue in the SVC posterior wall. This finding can be supported by our basic study, which showed a thinner layer of myocardial tissue with more fibrotic and fatty tissues in the dorsal surface of dog SVC.15

Ligament of Marshall

In 1850, Marshall16 first described the ligamentous fold, a vestigial tissue encompassing portions of the embryonic sinus venosus and left cardinal vein, running between the superior and inferior left PVs. In 1972, Scherlag et al17 demonstrated the electrical activity within the LOM in dogs, and the 2 terminal ends of this atrial tract may have insertions into the left atrial musculature and coronary sinus. Recently, Doshi et al18 found that the LOM has focal automatic activity induced by isoproterenol, and it may contribute to the development of AF. We also found that all of the LOM-AF needed isoproterenol infusion to provoke ectopy or bursts of AF, and this finding was similar to the finding by Hwang et al.7

Coronary Sinus

In 1907, Erlanger and Blackman19 described a “high degree of rhythmicity” in the area near the rabbit CS orifice. CS fibers have automatic activity, and it can be triggered into sustained, rapid rhythmic activity in the presence of norepinephrine.20 Because the patient number with CS-AF is very small, it is difficult to prove the electrophysiological characteristics.

Crista Terminalis

Hogan et al21 found a type of specialized fiber in the canine right atrium. This so-called atrial plateau fiber is found consistently along the border of the CT, with inherent slow diastolic depolarization during phase 4; the other rhythm is triggered by delayed afterdepolarizations and occurs either spontaneously or after epinephrine superfusion.23 The myocardial cells of diseased atria are significantly hypopolarized compared with those of normal atria.24,25 Thus, it is possible that LPFW could be the site of spontaneous ectopy.

Left Atrial Free Wall

Previous studies described 2 types of sustained rhythmic activity in human atrial fibers from normal and diseased hearts.23–25 One rhythm is automatic and depends on slow diastolic depolarization that can be enhanced by catecholamines to the point of spontaneous discharge, and is responsible for development of atrial ectopic foci.22 This study showed the 10 CT ectopy were catecholamine sensitive and that the ectopy accelerates to a very high depolarization rate and induces fibrillatory conduction in the atrium.

Mapping and Ablation of PAF Originating From Non-PV Areas

Several reports have demonstrated that most of the PAF initiated by ectopic beats originates from the PV.1–4 This laboratory first proposed the important concept of non-PV ectopy from different areas initiating PAF.5,6,8 Thereafter,
several investigators demonstrated non-PV ectopy-initiating AF, with the incidence varying from 3.2% to 47%.\textsuperscript{3,4,26–28} The present study showed that 68 patients (28%) had PAF initiated by non-PV ectopies. However, only 23 (33.8%) of 68 patients had a single focus from the non-PV areas. In an earlier study, Haissaguerre et al\textsuperscript{1} reported 8.9% of patients had AF from non-PV areas, including 6.7% from RA and 2.2% from LPFW. However, the same group reported a surprisingly high (47%) incidence of non-PV foci after PV disconnection in 100 patients with PAF, and these non-PV areas included the adjacent posterior wall around PV ostium in 25 cases, atrial tissue in 23 cases (posterior LA, 13; other parts of the LA, 6; RA or septum, 4), from the coronary sinus in 1, from the left SVC in 1, and 9 foci that could not be localized.\textsuperscript{27} Natale et al\textsuperscript{26} also reported 18 (37.5%) of 48 chronic AF patients had right-sided foci, and most of the right-sided foci were located in the proximity of the sinus node region along the superior and mid-portions of the crista terminalis. Recently, Schmitt et al\textsuperscript{28} used biatrial mapping technique to localize AF initiators and found 47% of the ectopies were in the non-PV areas. Therefore, based on these reports and the present study, non-PV ectopy is important in AF initiation.

This laboratory has demonstrated that use of endocardial atrial activation sequences from the high right atrium, His bundle, and coronary sinus (CS) catheters can predict the location of AF initiation foci. With the difference in the time interval between high right atrium and His bundle activation obtained during sinus beats and atrial premature beats <0 ms, the accuracy for discriminating the SVC and CT from PV ectopy is 100%.\textsuperscript{29} Furthermore, true activation potentials and the far-field potentials can appear in the same multipolar catheter, and the true origin of ectopy can be differentiated clearly when 2 multipolar catheters are simultaneously put in the SVC and RSPV.\textsuperscript{6} To localize the accurate site of ectopy and the mechanism of SVC-AF, 3D mapping using Carto system or basket catheter would be useful.\textsuperscript{30,31} However, isolation of SVC ectopy from atria may be better than focal suppression of ectopy inside SVC.

Because LOM may have multiple insertion sites in the LPFW or near the PV ostium, it is difficult to differentiate LOM ectopy from PV or LPFW ectopy. This laboratory has demonstrated that distal CS pacing can help differentiate LOM potential from PV potential, and the possibility of LOM ectopy should be considered when the so-called triple potentials are recorded around PV ostium.\textsuperscript{8} However, the best method for differentiating LOM ectopies from other ectopies would be recording the continuous activations of LOM from the multipolar microcatheter in the Marshall vein.\textsuperscript{7} We found a low success rate of curing LOM ectopy initiating AF in the long-term follow-up, because we did not routinely cannulate the Marshall vein and the ablation sites were only limited to the endocardial area. Hwang et al\textsuperscript{7} used the direct recording of LOM potentials from the Marshall vein to guide ablation sites, and Katritsis et al\textsuperscript{9} used combined endocardial and epicardial approach to ablate LOM ectopy initiating AF; the results showed that approximately 60% to 70% of patients were free of AF.

Although the present study demonstrated that application of radiofrequency energy in the non-PV areas was feasible, the success rate varied in these 6 groups. The higher success rate was in the right atrium, including SVC and CT, but the LPFW group had a higher recurrence rate because of anatomic limitation and multiple ectopic foci. There are 3 possible reasons for this. First, electrophysiologists are more sophisticated in manipulating electrode catheter in the right compared with left atrium. Second, right atrial structure was easier to be identified accurately compared with left atrium. Third, right side non-PV ectopy has a higher incidence of true focal AF (the ectopy is the driver of AF) and lower incidence of substrate problem; thus, elimination of the ectopy is more effective in curing AF. However, the true incidence of AF recurrence is uncertain because the occurrence of AF was paroxysmal, and sometimes it is difficult to detect asymptomatic AF. Furthermore, we did not routinely perform electrophysiological study in all of the patients with recurrent AF, thus the recurrent AF could not be demonstrated to originate from non-PV or PV foci.

Conclusions

At the present time, most PV ablation procedures are performed anatomically by isolating all PV ostia. Thus, recurrent AF should be considered from non-PV foci if 4 PVs were successfully isolated. This study demonstrated that PAF can be initiated by ectopic beats originating from the non-PV areas, and the application of radiofrequency energy in the non-PV areas is effective and safe to treat PAF.

Acknowledgments

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