Ablation for Atrial Fibrillation

Characterization of non-pulmonary vein foci with an EnSite array in patients with paroxysmal atrial fibrillation

Takanori Yamaguchi¹, Takeshi Tsuchiya¹*, Koji Miyamoto¹, Yasutsugu Nagamoto¹, and Naohiko Takahashi²

¹EP Expert Doctors-Team Tsuchiya, Koto 3-14-28, Kumamoto 862-0909, Japan; and ²Department of Internal Medicine, 1 Faculty of Medical Oita University, Oita, Japan

Received 11 May 2010; accepted after revision 9 August 2010

Aims
Non-pulmonary vein (PV) foci are sometimes difficult to identify and eliminate. The EnSite array (EA) reveals the detailed beat-to-beat virtual activation. This study aimed to characterize non-PV foci using the EA.

Methods and results
Sixty-five patients with paroxysmal atrial fibrillation (AF) were included. All had ectopy initiating AF and/or focal atrial tachycardia analysed using the EA. All patients underwent PV isolation (PVI) and additional ablation of non-PV foci if present. The EA revealed 59 PV foci in 48 patients (Group P) and 19 non-PV foci in 17 patients (Group N). In Group N, 12 patients (71%) also had 17 PV foci. The non-PV foci were frequently distributed in the left atrial (LA) roof (n = 5) and superior vena cava (n = 5). Pulmonary vein isolation during on-going AF terminated AF in 34 of 37 in Group P (92%) and 4 of 14 in Group N (29%) patients (P < 0.0001). All non-PV foci were eliminated by an EA-guided ablation. During a 23 ± 10 month follow-up, 11 patients (17%) had AF recurrences, mainly due to LA–PV reconnection.

Conclusion
Non-PV foci are prevalent in the LA roof and SVC sites, but can originate from other sites as well. When non-PV foci are observed, PVI may be insufficient and should be supplemented with non-PV foci ablation.

Keywords
Atrial fibrillation • Catheter ablation • EnSite array • Non-PV foci • PV isolation

Introduction
Although the pulmonary veins (PVs) are recognized as a crucial source of atrial fibrillation (AF) in most cases, non-PV foci play an important role in initiating and maintaining AF in ~20% of patients.¹⁻⁵ Non-PV foci are located at sites including the superior vena cava (SVC), left atrial (LA) posterior wall, crista terminalis (CT), coronary sinus (CS), ligament of Marshall (LOM), and interatrial septum.¹⁻³ The presence of non-PV foci closely relates to the recurrence of paroxysmal AF (PAF) after PV isolation (PVI).⁶⁻⁷ Episodes from non-PV foci are frequently unpredictable and transient and can easily change into AF. Also, since non-PV foci are located in diverse sites, it is possible that conventional techniques using point-to-point recording under fluoroscopic guidance underestimate the true features of non-PV foci. In contrast, the EnSite array (EA) visualizes the virtual activation of any tachyarrhythmia on a beat-to-beat basis in the cardiac chamber of interest.⁸⁻¹⁴ Therefore, the EA is expected to provide a rapid and precise identification of the AF initiation from PVs and non-PV foci irrespective of the sustainability and multiplicity. In this study, we aimed to characterize the non-PV foci with an EA.

Methods
Patients
This study included 65 patients with PAF (55 males and 10 females; age, 58 ± 10 years; range, 31–76 years) referred for radiofrequency AF ablation. All had clinically documented symptomatic PAF refractory to 2 ± 1 antiarrhythmic drugs. None had been treated with amiodarone or had undergone any prior AF ablation. All had ectopic beats initiating AF and/or focal atrial tachycardia (AT). The mean echocardiographical dimension of the left atrium (LA) was 38 ± 5 mm (range 31–46 mm)

* Corresponding author. Tel: +81 96 368 0403; fax: +81 96 368 0414, Email: tsuchiya@s1.kcn-tv.ne.jp

Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2010. For permissions please email: journals.permissions@oxfordjournals.org.
and mean left ventricular fractional shortening was 37 ± 6% (range 23–53%). Eleven patients had underlying heart disease, including a decreased left ventricular systolic function in three, sick sinus syndrome in three, prior myocardial infarction in two, idiopathic hypertrophic cardiomyopathy in two, and previous repair of an atrial septal defect in one. Patients in whom no ectopic beats were observed or induced, or who had a gigantic LA were excluded from this study.

Mapping procedure

Each patient after giving informed consent underwent an electro-physiological study in a fasting and conscious-sedated state. All anti-arrhythmic drugs were discontinued for at least five half-lives prior to the study.

A duodecapolar catheter was inserted through the right jugular vein. The proximal portion was positioned along the SVC and CT and the distal portion was placed in the CS for pacing and recording. Using the standard Brockenbrough technique, an atrial transeptal puncture was performed under fluoroscopic guidance and a multielectrode array (MEA; St. Jude Medical, Minnetonka, MN, USA) was transeptally introduced into the LA through a 10-Fr long transeptal sheath (Multilum, Medtronic, Minneapolis, MN, USA). The tip of the MEA catheter was placed in the LA appendage to ensure complete coverage of the entire LA by the MEA. In addition, two transeptal long sheaths were introduced through another septal opening made by another transeptal puncture. Two catheters including a 20-pole circular mapping catheter (Optima; St. Jude Medical) and deflectable 7-Fr quadripolar, non-irrigated 8-mm tip electrode ablation catheter (Fantasista; Japan Lifeline Co. Ltd., Tokyo, Japan) were introduced into the LA through the sheaths. The former catheter was placed in the ostium of either the right or left superior PV and was used to record PV potentials. The latter was introduced into the LA and was used for mapping, pacing, and radiofrequency ablation (RFA).

The boundaries of the PVs were set at the site where an apparent change was observed in the morphology from the PV antrum to the LA body. When such a change was not observed it was determined as a site ~1 cm away from the ostium of the PV tubular portion.

Intravenous heparin was administered immediately after the first atrial transeptal puncture to maintain an activated clotting time (ACT) ≥ 300 s. The ACT levels were monitored every 30 min, and if they were <300 s a suitable amount of heparin was injected.

After the MEA was placed in the LA, three-dimensional geometries of the LA and PVs were separately depicted and combined by the EA (version 3.0 in the first 17 patients and version 6.0J in the remaining 54). The tip of the MEA catheter was placed 1 cm away from the PV ostium to 30 W and 20 s at the LA posterior wall near the oesophagus to confirm the site of origin. The virtual activation map of the EA as a focal discharge followed by centrifugal activation of the surrounding tissue. Any solitary ectopic beat that did not initiate AF or AT was excluded from the analysis.

Ablation

Radiofrequency ablation was performed during induced AF (n = 51) or sinus rhythm (n = 14) after attempting to induce ectopic beats. In the first 11 patients, PV ostial segmental ablation was performed. In the remaining 54, PV antrum isolation was performed ~1 cm away from the ipsilateral superior and inferior PV ostia under navigation using an EA. The endpoint of the PVI was electrical isolation of the PVs in which the disappearance of the PV potentials and/or dissociation of the PV potentials from the LA were confirmed.

When a non-PV focus was identified, focal ablation was performed at the focus except for in the SVC where segmental isolation of the SVC was performed. When AF persisted after these procedures, a roof line ablation was performed in the LA until complete conduction block was achieved. If AF was not terminated by this procedure, additional ablation line(s) were created, including a bottom line connecting both inferior PVs and/or a mitral isthmus line. The endpoint for those linear lesions was complete conduction block and it was confirmed by pacing from appropriate sites and virtual activation mapping with the EnSite. Linear ablation at the cavitricuspid isthmus was performed in patients with documented and/or inducible cavitricuspid isthmus-dependent atrial flutter.

Radiofrequency energy applications were limited to 50°C and 40 W for 30 s at each site. However, the power and duration were reduced to 30 W and 20 s at the LA posterior wall near the oesophagus to reduce the risk of oesophageal thermal injury. The entire course of the oesophagus was depicted by a nasally introduced standard mapping catheter in the last 48 patients using an EA version 6.0J. Finally, an ISP challenge of ectopic beats with and without rapid atrial pacing was performed in patients with non-PV foci.

Follow-up

Follow-up was at 2 weeks, 1 month, and every 1–3 months thereafter using 24-h Holter monitoring and telephone interviews with all patients. Any symptomatic or asymptomatic atrial tachyarrhythmias were considered a recurrence. When a recurrence occurred and the patients agreed, a second catheter ablation was performed.

Statistical analysis

The continuous variables are expressed as the mean ± SD or number and percentage, as appropriate. Data were analysed with unpaired t-tests. Categorical variables were compared with χ² tests or a Fisher’s exact test as appropriate. Results with a P-value of <0.05 were considered statistically significant.

Results

Differences in the patients with pulmonary vein and non-pulmonary vein foci

The EA revealed 76 PV foci and 19 non-PV foci in 65 patients. Forty-eight patients had 59 PV foci but no non-PV foci (Group
P), whereas 17 patients had 19 non-PV foci (Group N). In Group N, 12 patients (71%) had 17 PV foci in addition to non-PV foci and 7 patients (29%) had non-PV foci alone. The mean age was significantly higher in Group N than in Group P (62 ± 8 vs. 56 ± 10 years old, \( P \approx 0.0131 \)). Additionally, Group N also had a greater incidence of underlying heart disease (35% vs. 10%, \( P \approx 0.0284 \)).

There was no significant difference between the groups in terms of other patient characteristics (Table 1). The coupling intervals of the PV and non-PV foci were similar (313 ± 41 ms and 289 ± 63 ms, \( P \approx 0.8661 \)).

### Occurrence of atrial fibrillation or repetitive activity from the pulmonary veins or non-pulmonary vein foci

The form of the ectopic beats was classified as ectopy triggering AF and focal AT. The timing was classified as before the ablation, after ISP, and after AF termination by DC shock or ablation. In total, 72 PV foci were identified as origins of ectopic beats triggering AF. The ectopy was spontaneous at 26 foci (34%) before the ablation, induced after ISP at 12 foci (16%), and spontaneous after AF termination at 34 foci (45%). The remaining 4 PV foci (5%) were identified as origins of focal AT occurring during the PVI.

For non-PV foci, 12 (63%) were identified as origins of ectopic beats triggering AF and the other 7 (37%) were identified as origins of focal AT. Ectopies triggered AF spontaneously at 3 foci (16%) before ablation, were induced after ISP at 7 foci (37%), and after AF termination by DC shocks at 2 foci (11%). No focal AT occurred spontaneously before ablation. However, focal ATs were induced by ISP and rapid atrial pacing at 2 foci (both from the CS ostium) and spontaneously transformed from AF during ablation at 5 foci. These focal ATs (except for 2 from the CS ostium) had a mean cycle length of 229 ± 31 ms. The cycle lengths of the two ATs from the CS ostium were 361 and 338 ms, respectively, and both patients also had PV foci. All ectopies followed by sustained AT originated from the same site as the AT focus in all patients.

When the timing and form of ectopic beats were compared between the PV and non-PV foci groups, the timing was found to be similar but non-PV foci were more frequently identified as origins of focal AT than PV foci (37% vs. 5%, \( P \approx 0.0009 \)). Ectopic beats from PV foci tended to occur spontaneously more frequently than those from non-PV foci (34% vs. 16%) and were less likely to be induced by ISP alone (16 vs. 37%). However, these differences were not statistically significant.

### The distribution of non-PV foci

In Group P, 22 episodes occurred from the right superior PV, 22 from the left superior PV, 9 from the right inferior PV, and 6 from the left inferior PV. In Group N, six episodes occurred from the right superior PV, eight from the left superior PV, and three from the left inferior PV.

Ten non-PV foci (53%) were observed in the LA and 9 (47%) outside the LA (Table 2). The sites of non-PV foci were the LA roof \( n = 5 \), LA posterior wall \( n = 1 \), LA anterior wall \( n = 1 \), interatrial septum \( n = 1 \), and left atrial appendage (LAA; \( n = 1 \)). Non-PV foci outside the LA were located in the SVC \( n = 5 \), CT \( n = 1 \), and LOM \( n = 1 \). Figures 1–3 show examples of ectopic beats from the LA posterior wall, LA anterior wall, and interatrial septum, respectively. All ectopic beats from non-PV foci in the LA were clearly elucidated by the EA even though a single ectopic beat triggered AF (Figures 1–3). Figure 4 shows an example of a focal AT that

---

**Table 1** Patient characteristics \((n = 65)\)

<table>
<thead>
<tr>
<th></th>
<th>Group P</th>
<th>Group N</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males (%)</td>
<td>55 (85)</td>
<td>42 (88)</td>
<td>0.4336</td>
</tr>
<tr>
<td>Age, years (range)</td>
<td>58 ± 10</td>
<td>56 ± 10</td>
<td>0.0131</td>
</tr>
<tr>
<td>Left atrial dimension, mm (range)</td>
<td>38 ± 5 mm (31–46)</td>
<td>38 ± 5 mm (31–46)</td>
<td>0.6577</td>
</tr>
<tr>
<td>LVFS, % (range)</td>
<td>37 ± 5</td>
<td>37 ± 5</td>
<td>0.8160</td>
</tr>
<tr>
<td>Underlying heart disease, n (%)</td>
<td>11 (17%)</td>
<td>5 (10)</td>
<td>0.0284</td>
</tr>
</tbody>
</table>

LVFS, left ventricular fraction shortening.

**Table 2** The distribution of non-PV foci

<table>
<thead>
<tr>
<th>Location of non-PV foci</th>
<th>Episodes from non-PV foci</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AF trigger</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
</tr>
<tr>
<td>LA</td>
<td>10 (53)</td>
</tr>
<tr>
<td>LA roof</td>
<td>5 (26)</td>
</tr>
<tr>
<td>LA posterior wall</td>
<td>1 (5)</td>
</tr>
<tr>
<td>LA anterior wall</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Interatrial septum</td>
<td>2 (11)</td>
</tr>
<tr>
<td>LA appendage</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Outside LA</td>
<td>9 (47)</td>
</tr>
<tr>
<td>SVC</td>
<td>5 (26)</td>
</tr>
<tr>
<td>CS ostium</td>
<td>2 (11)</td>
</tr>
<tr>
<td>CT</td>
<td>1 (5)</td>
</tr>
<tr>
<td>LOM</td>
<td>1 (5)</td>
</tr>
</tbody>
</table>

Values are represented by \( n \) (%).
Ablation of non-pulmonary vein foci

Radiofrequency ablation strategy
Electrical PVI was achieved in all PVs under navigation using an EA. In Group P (n = 48), an LA roof line was created after the PVI in 9 patients (19%) and additional LA line(s) consisting of an LA bottom line and/or a mitral isthmus line were created in five patients. In Group N, all 17 patients underwent RFA at the non-PV foci in addition to the PVI, including focal ablation of non-PV foci in 13 and segmental SVC isolation in 5. One patient underwent both an SVC isolation and focal ablation of a non-PV focus. An LA roof line was created in seven patients (41%) and additional LA line(s) were created in four patients. In terms of the non-PV foci in the LA (n = 10), 6 ± 2 RF pulses eliminated the non-PV foci and no ectopic beats occurred thereafter either spontaneously or following an ISP challenge with or without rapid atrial pacing. In two patients who had AF originating from the interatrial septum, frequent RF applications, in total 8 and 10 deliveries, from both sides of the interatrial septum, respectively were needed to eliminate the AF (Figure 5). In terms of the non-PV foci from outside the LA (n = 9), an SVC isolation was achieved by 9 ± 5 RF pulses in five patients, and in the remaining four, the non-PV foci were eliminated by 2 ± 2 RF pulses. After these procedures, no ectopic beats occurred either spontaneously or after an ISP challenge with or without rapid atrial pacing. There were no procedural complications.

Acute effect of the radiofrequency ablation during on-going atrial fibrillation
The acute RFA results were analysed in 51 patients (78%) undergoing AF ablation during on-going AF [37 in Group P (77%) and 14 in Group N (82%)]. Pulmonary vein isolation resulted in acute AF termination in 34 of 37 patients in Group P (92%) and in 4 of 14 patients in Group N (29%, P < 0.0001). In the latter group, additional focal RFA of non-PV foci (n = 2) or a segmental SVC isolation (n = 4) resulted in acute AF termination in six patients (43%). In total, AF termination was eventually achieved in 10 patients (71%) in Group N. Figure 5 shows an example of AF termination by focal RF pulses applied to the interatrial septum.

After the PVI in Group P and PVI plus RFA of non-PV foci in Group N, an LA roof line was created in seven patients with...
persistent AF after the PVI (three Group P and four Group N patients). This resulted in direct AF termination in one Group P patient and transformation to focal AT originating from the LA roof in one Group N patient. The latter was terminated by a subsequent focal RFA to the focus. Additional ablation applications to the LA were performed in the remaining five patients (two Group P and three Group N patients). AF termination was not achieved in Group P, whereas in one Group N patient the AF converted into a focal AT originating from the LA roof; the arrhythmia was terminated by a subsequent focal RFA to the focus. In the other four patients, internal DC shocks were performed to terminate the AF persisting despite the stepwise procedures. In summary, acute AF termination was achieved in 35 of 37 patients (95%) in Group P and 12 of 14 patients (86%) in Group N (P = NS).

Follow-up
During a 23 ± 10 month follow-up, 11 patients (17%) had AF recurrences, including 7 (15%) in Group P and 4 (24%) in Group N. No AT recurrence was observed. The recurrence rate was similar between the 2 groups. Six of the 11 patients with AF recurrence underwent a second session including four in Group P and two in Group N. No non-PV foci were observed in these sessions but all patients had LA–PV reconnection and in four patients ectopic beats from PV foci triggered AF. No recurrence was observed after the second session.

Discussion
Main findings
The EA virtual activation map quickly identified the location of the non-PV foci in the LA and clearly elucidated any subsequent activation of ectopic beats initiating AF and focal AT. Non-PV foci were prevalent at the LA roof as well as in the SVC, but they were also observed in the LA, RA, SVC, possibly the LOM, and CS ostium. PVI performed during on-going AF terminated the AF in 92% of the Group P and 29% of the Group N patients. Additional RFA of non-PV foci and/or LA linear ablation increased the acute AF termination rate to 86% in Group N. Recurrence after ablation was due to AF, which resulted mainly from LA–PV reconnection in the patients with PV foci and also in patients with non-PV foci during the index ablation procedure. The recurrence rate during the long-term follow-up of the patients with non-PV foci was 24%, which was similar to that of the patients who only had PV foci.

Figure 2 Atrial fibrillation initiation from the anterior LA. The left upper panel shows serial virtual activation maps of an ectopic beat originating from the anterior LA viewed from the superior direction. The time period of the upper left map is that of the yellow bar indicated by an asterisk. The tracings in the lower panel indicate the electrocardiographic leads I and V1, bipolar electrograms recorded from the mapping catheter (MAP 1–2 and 3–4), and virtual unipolar electrograms recorded at sites 6 to 10 (corresponding site number is in the upper left panel). The tracking virtual and the virtual unipolar electrograms at site 6 show a QS pattern suggesting that site 6 is probably the origin of the ectopic beat. The upper middle panel shows the subsequent activation of the LA. The upper right panel shows the fluoroscopic image of the ablation catheter during the radiofrequency delivery. CS, coronary sinus; HRA, high right atrium; ABL, ablation catheter; MEA, multielectrode array; MV, mitral valve; LAO, left anterior oblique. The other abbreviations are as in Figure 1.
Superiority of the EnSite array over the conventional mapping methods

Initiation of ectopic beats from non-PV foci is usually unpredictable in terms of the timing. The episodes were sometimes transient or easily changed into AF. Further, non-PV foci were located at diverse sites. Therefore, it is possible that the conventional technique under fluoroscopic guidance underestimates the true features of the non-PV foci because it requires a time consuming and deliberate process of accumulating the bipolar electrograms using the point-to-point recording method. This is also true for electroanatomical mapping because this system requires the accumulation of point-to-point sampling points.

In contrast, the EA visualizes the detailed activation of >3000 VUEs of any tachyarrhythmia on a beat-to-beat basis in the cardiac chamber of interest. We previously reported that the EA virtual activation map was useful for identifying sustained and non-sustained AT irrespective of whether or not this was associated with organic heart disease. In this study, the EA provided us with information on the rapid activation of the initiation of all atrial tachyarrhythmias from both the PVs and non-PV foci even though they were transient in nature or from multiple origins. Further, EA-guided RFA eliminated all non-PV foci.

The distribution of the non-pulmonary vein foci

In the present study, 26% of the patients had non-PV foci. Some researchers have reported that the prevalent sites of non-PV foci were the SVC, posterior LA, CT, LOM, and CS ostium, which is also consistent with our data. In this study, the LA roof was one of the most prevalent sites of non-PV foci in five patients (26%) who had focal discharges including ectopic beats initiating AF (n = 3) and focal ATs (n = 2). However, the roof location could have been described as the anterior or posterior left atrium in the previous studies.

Induction of ectopic beats

The mechanism of ectopy originating from the PVs has been explained by late phase 3 early afterdepolarization-induced triggered activity, although other cellular mechanisms including abnormal automaticity and delayed afterdepolarizations have also been
The former requires a shortened action potential duration, which is usually obtained by promoting sympathetic and/or parasympathetic nerve activity. On the other hand, it appears that it is better to discuss the mechanism of ectopic beats originating from non-PV foci apart from that for the thoracic veins and working atrial muscle. In this study, non-PV foci were distributed not only in the thoracic veins, including the SVC, CS, and LOM, but also in the working atrial myocardium including the LA roof, posterior wall, septal wall, base of the LA appendage, and CT. The ectopic beats originating from these thoracic veins were reported to result from triggered activity due to delayed afterdepolarizations. Ectopic beats originating from diseased atrial myocardium associated with underlying heart disease have been reported to result from delayed afterdepolarizations and abnormal automaticity. Both delayed afterdepolarizations and abnormal automaticity are facilitated by ISP through an increased I_{ca-L} current, and thus, an ISP injection was anticipated to facilitate induction of spontaneous ectopic beats from these thoracic veins and also from damaged working atrial muscle.

Therefore, ISP is anticipated to facilitate the induction of atrial tachyarrhythmias both from the PVs and non-PV foci. This occurred in the current study and the induction was similar for PV foci and non-PV foci (16 vs. 37%).

The role of non-pulmonary vein foci
Non-PV foci have been considered to act as initiators of AF, focal drivers of AF, and origins of concomitant arrhythmias. In this study, 12 non-PV foci (63%) acted as AF initiators. It was noted that four non-PV foci in the SVC (n = 3) and interatrial septum (n = 1) appeared to function not only as initiators but also as focal drivers because RFA applied directly to those foci terminated the AF. Five of seven focal ATs transformed from AF during the ablation of AF, which originated from the LA roof (n = 2), interatrial septum (n = 1), LA appendage (n = 1), and SVC (n = 1) and RFA to those sites terminated the ATs. Those ATs may be related to the driver mechanisms of AF, although the possibility of a simple concomitant tachycardia cannot be ruled out. The remaining two ATs originated from the CS ostium and were directly induced by an ISP injection or by rapid atrial pacing.
before the PVI and did not trigger AF. Therefore, these ATs appear to be simply concomitant arrhythmias.

Radiofrequency ablation results

Pulmonary vein isolation terminated the AF in 92% of the patients with PV foci but in only 29% of those patients with non-PV foci. Therefore, PVI seems insufficient when non-PV foci are observed or induced by an ISP injection. Interestingly, 71% of the Group N patients had non-PV foci in addition to PV foci but the percentage of AF termination achieved by PVI alone was 29%, suggesting that non-PV foci play an important role in maintaining AF as well as triggering AF.

There was no significant difference in the recurrence rate of AF between Groups P and N during a 23 ± 10 month follow-up. It was noted that no non-PV foci were observed or induced by ISP after a second session. All the patients involved had LA–PV reconnection possibly related to AF recurrence. No recurrence was observed after the second session. Thus, EA-guided ablation of non-PV foci with PVI seems to be an effective strategy for PAF.

Limitations

Patients with gigantic LAs were excluded from the study because areas where the distance from the centre of the MEA is >4.0 cm cannot be analysed by the EA. Therefore, our study may have missed a large group of patients with dilated LAs and more extensive damage of the atrial myocardium. Mapping in the RA was performed using the conventional mapping method and point-to-point recording under fluoroscopic guidance (except for in SVC), and therefore, underestimation of non-PV foci is possible in terms of the ectopy originating from the RA. Further, it seems difficult to clarify where the site of origin is within the PVs with the EA, but it can reveal the exit from the PVs.

Most of the non-PV foci were artificially induced and may not be clinically relevant, because only 3 foci (6%) were spontaneously triggering AF before the ablation, and PVI only can be associated with a success rate as high as 90% (including redo procedures for re-isolation) in PAF. Further, the rate of patients with non-PV foci was 26%, so only a small number of patients had non-PV foci associated with PAF.

The relative inaccuracy of the EnSite mapping should be emphasized, given the mean number of RF deliveries required to eliminate these foci.

Finally, to confirm the efficacy of the EA for PAF ablation, a randomized study comparing the EA-guided ablation and conventional PVI will be needed. However, the former approach will require an additional cost.

Conclusion

Non-PV foci frequently coexist with PV foci. The prevalent sites of non-PV foci are the LA roof and SVC. When only PV foci are observed, PVI alone is sufficient, whereas when non-PV foci are
observed. PVI seems insufficient and additional non-PV foci ablation would be required, even in patients with PAF.

**Conflict of interest:** T.T. has served as a speaker and consultant for Nihon Kohden and St. Jude Medical.

**References**