Electrophysiological and Electrocardiographic Characteristics of Focal Atrial Tachycardia Originating From the Pulmonary Veins

Acute and Long-Term Outcomes of Radiofrequency Ablation

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Background—The objective of this study was to describe the electrophysiological characteristics, anatomic distribution, and long-term outcome after focal ablation (RFA) of pulmonary vein (PV) atrial tachycardia (AT). Both atrial fibrillation (AF) and AT may be due to a rapidly firing focus in the PVs. Whether these represent two aspects of the same process is unknown.

Methods and Results—Twenty-seven patients with 28 PV(16%) ATs of a consecutive series of 172 undergoing RFA for focal AT are reported. The mean age was 39±16 years, with symptoms for 9±14 years resistant to 1.7±0.8 medications. AT occurred spontaneously or with isoproterenol in all patients and was not inducible with PES in any. The distribution of PV ATs was right superior PV, 11; left superior PV, 11; left inferior PV, 5; and right inferior PV, 1; 26 of 28 foci (93%) were ostial. RFA was successful in 28 of 28 PV ATs acutely. RFA was focal in 25 of 28, with PV isolation of a single target vein in 3. There were 4 recurrences at a mean of 3.3 months. Repeat RFA was performed in all 4 and successful in 3 of 4. All but one recurrence occurred from the same site. Long-term success was achieved in 26 of 27 (96%) patients at mean follow-up of 25±22 months. No patients have had subsequent development of AF or AT from a different site.

Conclusions—PV AT has a distribution similar to PV AF, with a propensity to upper veins. However, the majority of foci are ostial, and only a small percentage occur from deep in the PV. Focal RFA is associated with high long-term success, with freedom from both AT from other sites and from AF. PV AT is a localized process and therefore may be different from PV AF. (Circulation. 2003;108:1968-1975.)

Key Words: tachycardia ■ fibrillation ■ catheter ablation ■ fibrillation

The characteristic anatomic distribution for focal atrial tachycardia (AT) is well recognized. In the right atrium, these foci commonly originate from the crista terminalis, the tricuspid annulus, the os of the coronary sinus (CS), and the para-hisian region.1-3 In the left atrium, these foci particularly tend to cluster around the pulmonary veins (PVs)4 and more recently have been described at the mitral annulus.5 In recent years, the pioneering work of Haissaguerre and coworkers6 has focused our attention on the role of the PVs in the initiation of paroxysmal atrial fibrillation (AF). In patients with AF, we have learned that PV foci are usually multiple and usually involve multiple veins,7 that they are more common in upper than lower veins, and frequently originate from deep into the trunk of the vein,8 which, unless an approach is taken that isolates all 4 veins, the recurrence rate will be high.9 However, there is a paucity of similar such data describing the electrophysiological and electrocardiographic characteristics of PV tachycardia, the particular anatomic distribution of these foci, and the acute and long-term success rates of focal ablation. This study describes the electrophysiological and electrocardiographic characteristics, anatomic distribution, and long-term outcome after focal ablation (RFA) of PV AT in a consecutive series of patients undergoing RFA at a single center.

Methods

Study Population
The study population included 27 patients with 28 ATs of a consecutive series of 172 patients undergoing RFA for focal origin of 182 ATs. All patients had clinically documented paroxysmal or persistent AT for which they were having RFA. In these patients, detailed monitoring (at least two 24-hour monitors) demonstrated...
paroxysmal or persistent AT without any documentation of coexisting paroxysmal AF.

All patients underwent electrophysiological study after written informed consent was given. The study was approved by the Melbourne Health Research Ethics Committee. Patients were studied in the fasted awake state with minimal use of sedation. All antiarrhythmic drugs were ceased a minimum of 5 half-lives before the procedure. Three patients had been taking amiodarone.

Catheter positioning and the approach used in our laboratory for ablation of AT have been previously published.2 In brief, catheters were positioned in the following manner: (1) CS catheter (10-pole, 2 to 5 mm interelectrode spacing) positioned with the proximal bipole at the ostium of the CS by best septal left anterior oblique projection; (2) crista terminalis catheter (20-pole, 1 to 3 1-mm interelectrode spacing) positioned along the crista terminals; intracardiac echocardiography (9 MHz) was used to aid in positioning of the catheter when necessary; (3) His bundle electrogrom catheter; and (4) mapping and ablation catheter.

Standard electrophysiological criteria were used to diagnose AT.1 Attempts at AT induction were made including atrial programmed extrastimulation and burst atrial pacing. If this was unsuccessful or when AT was not occurring spontaneously, isoproterenol was infused (1 to 6 µg/min). Mapping of the earliest site of endocardial activity relative to surface P wave was performed with a 4-mm-tip mapping and ablation catheter. When a left-sided origin was suspected, a patent foramen ovale (PFO) was probed for using the ablation catheter, and in the absence of a P-Wave Morphology

Surface 12-lead ECG P-wave morphology was assessed as previously described.10 P waves were described on the basis of the deviation from baseline of the T-P interval as being (1) positive (+), if there was a positive deviation from the isoelectric baseline, (2) negative (−), if there was a negative deviation, (3) isoelectric, arbitrarily defined when there was no P-wave deviation from baseline of ±0.05 mV, (4) notched, when a double-positive deviation occurred, and (5) biphasic, if there were both positive and negative (+/− or −/+ ) deflections from baseline. P-wave amplitudes were measured from peak to nadir.

Right Atrial Endocardial Activation Sequence

The consistent deployment of a 20-pole catheter on the crista terminals, a decapolar catheter in the CS, and a catheter in the His position allowed characterization of the right atrial endocardial activation sequence maps. Activation timing was measured from the onset of the P wave in lead II of the surface ECG (arbitrarily assigned a time of 0 ms) to each of the intracardiac bipoles of these catheters. Activation times were measured in a standardized fashion to onset of the first rapid deflection from the baseline. Pacing from the putative vein of origin (through the mapping/ablation catheter) to confirm the activation sequence pattern was used when spontaneous tachycardia activity was infrequent.

Radiofrequency Ablation and Outcome

Radiofrequency ablation was performed with continuous temperature feedback control of power output to achieve a target temperature of 50° to 60°C, for a maximum power of 30 to 50 W, according to the anatomic location of the site of earliest endocardial activity. Acute procedural success was defined by the absence of tachycardia or ectopy 30 minutes after ablation despite infusion of isoproterenol (up to 6 µg/min) and burst atrial pacing. All variables are expressed as mean±SD. Statistical comparisons between groups were made by using the 2-tailed Student’s t test or the Mann-Whitney rank sum test. A probability value <0.05 was considered statistically significant. Sensitivity and specificity were calculated for P-wave morphology.

Results

Patient Characteristics

This study includes 27 patients with 28 ATs originating from a PV. These 27 patients represent 16% of a consecutive series of 172 patients undergoing RFA of a focal AT at a single center. Fourteen patients were women, and the mean age of the group was 39±16 (range, 9 to 78) years. Symptoms attributable to tachycardia had been present for 9±14 years, and patients had tried and failed a mean of 1.7±0.8 antiarrhythmic medications. Left atrial size was normal at 37±5 mm. Three patients were previously taking amiodarone, but this had been ceased in all 3, over 4 weeks before the procedure. Two patients had a mild dilated cardiomyopathy, one of which recovered on ablation of tachycardia.

Tachycardia Characteristics

In all patients, AT or ectopy from the tachycardia focus occurred spontaneously (23 patients) or during isoproterenol infusion (4 patients). Tachycardia was not inducible with programmed extrastimulation or burst atrial pacing in any patient. Interestingly, in one patient with a right superior PV (RSPV) focus, bursts of tachycardia were stimulated by coughing, and in another patient with a left inferior PV (LIPV), focus nonsustained AT was elicited by talking. Sustained tachycardia was not induced in 8 (28%), with mapping guided by frequent spontaneous unifocal ectopy. Mean tachycardia cycle length was 311±57 ms.

Anatomic Location

The RSPV was the site for AT in 11 patients (39%). Similarly, the left superior (LSPV) was the site of origin in 11 (39%) of PV tachycardias. Thus, a superior PV was the focal origin of 78% of all ATs in this series. The LIPV was the site...
of origin in 5 (18%), with the right inferior (RIPV) being represented in 1 (4%).

Within the PVs, the focus was ostial (Figure 1) according to the above definition in 26 (93%) patients. The remaining 2 cases occurred distally between 2 and 4 cm from the venoatrial junction, 1 in the LSPV (Figure 2) and 1 in the RSPV.

P-Wave Morphology
The P-wave morphology for each of the PV sites is presented in the Table, with typical examples of each site in Figure 3. The typical P-wave features of the PV tachycardias included the following P wave characteristics:

1. Positive across precordial leads from V1 to V6 in all patients. The precordial leads became consistently less positive from V1 (mean amplitude, 0.17 ± 0.06 mV) to V6 (mean amplitude, 0.10 ± 0.05 mV; P < 0.001) in PV tachycardias regardless of their vein of origin.

2. Negative or isoelectric in aVL in 24 of 28 (86%). The exceptions were 3 RSPV and 1 RIPV tachycardia.

3. Negative in aVR in 27 of 28 (96%). The exception was a LIPV tachycardia.

In addition, the following P-wave characteristics assisted localization of the tachycardia focus to a particular PV:

1. Notching was characteristically seen in the left-sided PVs, particularly in the inferior leads, but also in the precordial leads (Figure 3). This occurred in both LSPV and LIPV. This morphology was seen in 16 of 16 left-sided PV tachycardias but in only one right-sided PV tachycardia. A bifid P wave in at least 2 leads thus had a specificity of 93% and sensitivity of 100% for tachycardias arising from the left-sided PVs.

2. The width of V1 was significantly greater in left-sided veins (0.12 ± 0.02 cm) than in right-sided veins (0.07 ± 0.02 cm; P < 0.001).

3. A positive P wave in lead I was highly suggestive of a focus in a right-sided PV. This was present in 11 of 12 (92%) with a right-sided origin and in 2 of 16 (12%) with a left-sided origin (sensitivity = 92%; specificity = 87%). Both left-sided PV tachycardias (LSPV) with a positive P wave in lead I had a P-wave amplitude in that lead of 0.05 mV.

4. The inferior leads were markedly positive in all superior PVs, with a mean lead II amplitude of 0.18 ± 0.07 mV compared with a flatter but still positive appearance in the inferior veins of 0.10 ± 0.04 (P = 0.04). A lead II amplitude of ≥0.10 mV had a sensitivity of 95% and specificity of 66% for detection of a superior vein.

5. The lead III/II ratio was >0.8 in 10 of 11 LSPV but in only 2 of 11 RSPV (sensitivity = 90%; specificity = 82%).

We applied the algorithm developed by Yamane et al., using pace mapping from the PVs to the spontaneous PV P-wave morphology seen in patients in this study. The use of this algorithm correctly identified the native PV of origin in 22 of 28 (78%) tachycardias. The 6 exceptions were (1) 2 patients with an LSPV AT who had a positive P wave in lead I (P-wave amplitude 0.05 mV) but could have been correctly identified by noting in lead II; (2) 2 patients correctly identified as left-sided but with lead II amplitudes <0.10 mV allocating them inferiorly rather than superiorly; (3) 1 patient with LIPV AT who had a markedly bifid P wave in the precordial leads but not in lead II; and (4) an RSPV AT with...
a P-wave amplitude in lead I of 0.03 and a lead III/II ratio of 1 and was thus incorrectly allocated as a left-sided PV.

In the present series, if notching in 2 or more leads had been used in the Yamane et al algorithm at the first stage, all left-sided ATs would have been correctly identified.

**Atrial Endocardial Activation Sequence Mapping**

All catheters in the activation maps were located distant from the PV sites of tachycardia origin and therefore absolute activation times from these catheters were relatively late compared with the activation time at the ablation site. However, 3 of the PVs (LUPV, LLPV, and RUPV) showed a characteristic pattern of endocardial activation. Typical examples of the intracardiac activation patterns are represented in Figure 4.

Some simple observations aided in giving a general indication of the likely PV of tachycardia origin.

**Right Versus Left PVS**

For right PV tachycardias, activation at CS9,10 preceded activation at CS1,2. (31.6 ± 16.1 ms versus 56.0 ± 19.7 ms, P = 0.006). Not only was the activation time significantly earlier, but CS9,10 activation occurred before CS1,2 for all RPV tachycardias. Thus, this observation was highly predictive of a right-sided PV.

The reverse was true for left-sided PVs. For left PVs, activation at CS1,2 always preceded activation at CS 9,10. The mean activation time at CS1,2 was significantly earlier than at CS9,10 (0.7 ± 32.0 ms versus 24.0 ± 13.1 ms, P = 0.05). Thus, this observation was highly predictive of a left-sided PV tachycardia.

**RSPV Versus LSPV**

For RSPV tachycardia, the first crista terminalis activation occurred significantly earlier than earliest CS activation (6.9 ± 11.9 ms versus 23.3 ± 13.9 ms, P = 0.003) (Figure 5). For LSPV tachycardia, earliest crista terminalis activation and earliest CS activation occurred relatively simultaneously, and these activation times were not significantly different. Thus the dispersion of activation between the crista terminalis and the CS (earliest crista terminalis activation on any bipolar minus earliest CS activation on any bipolar) was significantly greater for RSPV than for LSPV tachycardias. (31.2 ± 13.6 versus 12.2 ± 7.8 ms, P = 0.01).

Because of small patient numbers, similar measurements were not made for the inferior veins.

By using these observations and the PV pace-mapping sequences previously described by Deen et al for the same catheter set, of the 22 patients in whom catheter positioning was verified, 20 of 22 veins were successfully identified by a blinded observer.

**Use of Paced Endocardial Activation Sequence Mapping**

In 10 patients with very infrequent activity, the paced endocardial activation sequence map could be compared with the spontaneous map to assist in localizing the PV of origin. In these 10 patients, the paced endocardial activation map correctly identified the vein of focal origin in 8 of 10. In the remaining 2 patients, catheter movement and inability to capture prevented comparison.

**Radiofrequency Ablation**

Radiofrequency ablation was initially successful in 24 of 28 patients. In 4 patients, the initial procedure was unsuccessful because of infrequent ectopy or tachycardia.

After a second procedure in these 4 patients, successful ablation was achieved in all.

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**P-Wave Morphology**

<table>
<thead>
<tr>
<th>ECG Lead</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>aVL</th>
<th>aVR</th>
<th>V1</th>
<th>V3</th>
<th>V6</th>
</tr>
</thead>
<tbody>
<tr>
<td>RSPV</td>
<td>+(10), -(1)</td>
<td>+(11)</td>
<td>+(11)</td>
<td>-(8), +(1), biphasic(2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LSPV</td>
<td>iso(8), +(2), -(1)</td>
<td>+(11)</td>
<td>+(11)</td>
<td>-(11)</td>
<td>-(11)</td>
<td>+(11)</td>
<td>+(11)</td>
<td>+(10), iso(1)</td>
</tr>
<tr>
<td>RIPV</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>LIPV</td>
<td>iso(3), -(1), +(1), +(4), -(1), +(4), -(1)</td>
<td>-(3), iso(2)</td>
<td>-(4), +(1)</td>
<td>+(5)</td>
<td>+(5)</td>
<td>+(3), iso(2)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Figure 3. Tachycardia P-wave morphology at each PV site. Left-sided compared with right-sided PVs demonstrate positive notching in $\simeq$2 surface leads, an isoelectric or negative P wave in lead 1, and a lead III/II ratio >0.8, and a broad P wave in lead V1. Inferior veins were flatter or negative in leads II/III compared with superior veins.
Ablation was focal, with no attempt to isolate the PV in 25 of 28 (89%) tachycardias. The mean activation time at the successful ablation site measured to onset of the P wave in lead II was $-35 \pm 19$ ms (Figure 6). In the presence of a distal focus (1 patient) or when ectopy or tachycardia had become quiescent despite isoproterenol (2 patients), PV isolation was performed after deployment of a lasso catheter. In one patient with a distal focus in the RSPV, successful ablation was carried out by using a focal approach at this distal site without complication.

Mean RF duration for the initial procedure was $215 \pm 176$ seconds, with a fluoroscopy time of $29 \pm 8$ minutes.

There were no complications from the procedure, and in particular, no PV stenoses were detected by postablation contrast injection.

Follow-Up

Of the 28 tachycardias successfully ablated, 4 patients had a recurrence. In all 4 patients, recurrent symptoms were noted within 1 month of the initial procedure. The recurrences occurred at the RSPV in 2 patients, LSPV in 1 patient, and LIPV in 1 patient.

Of the 4 recurrences, all elected to undergo repeat ablation, which was performed at a mean follow-up of 3.3 months. Repeat ablation was successful in 3 of 4 without further recurrence. Thus, at a mean follow-up of $25 \pm 22$ months, the long-term success was 96% (26 of 27 patients).

Of the 4 recurrences that underwent repeat ablation, 3 were from the original tachycardia focus. The 4th patient recurred with 2 distinct PV foci; the original focus in the LSPV and a second focus in the RSPV. RFA in this patient was unsuccessful. No patients have had development of AF after successful ablation of PV tachycardia at a mean follow-up of $25 \pm 22$ months.

Discussion

It has been extensively documented that focal AT tends to have a characteristic anatomic distribution, and in the left atrium, a distribution to the PVs is well recognized. However, most published series have included relatively small numbers of patients with PV tachycardia, and the electrocardiographic and electrophysiological characteristics are not well described. In addition, there is a paucity of data describing the long-term success of ablation for PV tachycardia, of particular relevance in the era of ablation of PV AF.

The current study describes the characteristic P-wave morphology and electrophysiological features of PV tachycardia in a consecutive series of patients. It documents a high long-term success rate for ablation through the use of a focal approach with a low incidence of recurrent AT and with no patients with development of late AF. These PV tachycardias represented 16% of all focal ATs and 78% of all left ATs from a consecutive series at a single center.

Comparison With Prior Studies

To date, published studies of AT originating from the PVs have included small numbers of patients only, and, as such, the electrocardiographic and electrophysiological features and long-term response to ablation have not been well characterized. Tang et al described 11 PV tachycardias (10 from upper PVs) of 14 LA tachycardias. This study did not describe the precise location of these foci nor the long-term outcome of ablation. Anguera et al included 6 PV tachycardias of 14 left ATs, with successful ablation only achieved in 3. Other series have included fewer than 5 PV tachycardias, and no definite conclusions could be drawn.

P-Wave Morphology

P-wave morphology provided a good general guide to the left atrial location of the tachycardia and more specifically to the likely PV of origin. In the current series, an entirely positive P wave was found in lead V1 in 100%, with lead aVL isoelectric or negative in 86%, consistent with the findings of Tang et al. Lead aVL may be biphasic or positive in right-sided PV tachycardias. Lead aVR was negative in
the present series, there was considerable overlap of patients in the lead II voltage range of 0.10 and 0.15 mV (9 superior and 2 inferior veins). Thus, if the cutoff for a superior vein was increased to a lead II amplitude of ≥0.15 mV, specificity would be increased but sensitivity significantly reduced. Therefore, P-wave morphology was of greater accuracy in distinguishing right-sided from left-sided veins, in contrast to superior from inferior. This is in keeping with the known limitations of spatial resolution in P-wave morphology as in many instances, the upper and lower veins are separated only by a fold of atrial myocardium.

Electrophysiological Characteristics
In this series, all PV tachycardias demonstrated onset that was either spontaneous or occurred in response to isoproterenol. In several patients, onset also occurred with unusual maneuvers such as coughing or talking. Tachycardia could not be induced by programmed extrastimulation or burst atrial pacing in any patient. Although a definitive determination of AT mechanism is generally beyond the scope of clinical electrophysiology, the mode of onset of AT in this study would nevertheless be most suggestive of abnormal automaticity. Although triggered automaticity or microreentry cannot be completely excluded, the absence of initiation in any patient in response to either programmed stimulation or burst atrial pacing would make these mechanisms less likely. Ongoing debate exists as to whether PV foci initiating AF are due to reentry or triggered automaticity, but this mechanism is not necessarily the same as that in isolated focal PV tachycardia.

Activation Sequence Mapping
Prior studies have used endocardial activation sequence mapping to localize right ATs and more recently for...
determining the PV origin of focal AF. Deen et al demonstrated that an evaluation of the endocardial activation pattern could identify the PV of origin in 94% to 97% of cases when patterns were retrospectively analyzed. Similarly, in the present study, the PV of origin could be distinguished on the basis of a characteristic activation sequence pattern and some stereotypic observations. CS activation occurred from proximal to distal for right PVs and distal to proximal for left PVs. Variations in activation timing between the CS and crista terminalis catheters and on the latter catheter itself could be used to further differentiate left from right and upper from lower PVs. The endocardial sequence can also be used with paced activation sequence mapping to confirm the likely vein of origin. Pappone et al found a concordant pace map sequence to be highly sensitive (92.8%) but relatively non-specific (47.8%) for determining the site of successful RF ablation for focal AT originating from right and left atria. Thus, although these patterns do provide a guide to tachycardia origin, they do not obviate the need for careful mapping. In particular, although PV tachycardia accounts for the majority of left ATs (78% in this series), it should be remembered that non-PV sites with differing and nonstereotypic activation sequences may be present.

Ablation Technique
Patients in this study underwent focal ablation of AT with a high acute success rate and with a long-term success achieved in 93% at a follow-up of more than 2 years. These outcomes are similar to those of focal ablation of atrial tachycardia originating from other sites. Recent advances in the ablation of AF originating from PVs have included the description of PV isolation. Although this has represented a quantum advance in AF ablation, the current study suggests that its routine application to focal PV tachycardia is unnecessary. In the present study, we performed PV isolation only when the focus was located distally or when activity became quiescent after a clear determination of the vein of origin.

Indeed, there are a number of possible advantages in using a focal approach in these circumstances.

(1) The risk of PV stenosis has been clearly correlated with the extent of PV ablation. Thus, a focal approach would be expected to carry a much lower stenosis risk.

(2) A focal approach obviates the need for double transeptal puncture in the majority of cases.

(3) The current study highlights the very proximal location of the majority of these foci. If empiric PV isolation were performed even just inside the PV mouth, there would be a significant chance of missing the proximal focus.

PV Tachycardia Compared With PV Fibrillation
In patients with paroxysmal AF originating from the PVs, extensive monitoring will frequently document coexisting paroxysms of both AT and also atrial flutter. Similarly, at electrophysiological evaluation, many patients with paroxysmal AF (PAF) will have nonsustained paroxysmal tachycardia or spontaneous or inducible atrial flutter. Thus, in the PAF patient population, a wide spectrum of atrial arrhythmias may coexist. Furthermore, patients with PAF usually have multiple PV foci in multiple veins, and many of these foci originate distally in those veins. In the current series, we studied a different patient population including only those patients whose clinical arrhythmia was AT. In contrast to patients with PAF, these patients demonstrated a largely focal process, without evidence of a more progressive and diffuse process as observed in the PAF population and without a tendency to development of further atrial arrhythmias during long-term follow-up. Notably, when patients with PV AT presented with recurrence, in almost all instances this was from the original focus. In contrast, patients with PAF have recurrences from foci in other PVs and from within the body of the left atrium. In addition, in the vast majority of patients, the focus originated from the ostium of the vein (or within 1 cm of the designated ostium) rather than from further distally (2 to 4 cm), as described in the landmark study of PV AF by Haissaguerre et al. These observations suggest that patients with focal AT may represent a population different from patients with PV AF and that they have a discrete and focally curable process. However, there were some similarities to patients with PV AF and in particular a similar preference for upper versus lower PVs and a greater frequency in the left inferior than the right inferior PV.

It is interesting to speculate as to why patients with focal atrial tachycardia behave differently from those with AF.

(1) The cycle length of PV tachycardia in patients in this study (311±57ms) was longer than that reported for PV tachycardia in patients with AF (130±30 ms).

(2) It is probable that patients with PAF have a more generalized process affecting the muscular sleeves in all 4 PVs in a diffuse manner compared with the focal nature of the process in patients with isolated PV tachycardia.

(3) Patients with PAF also may have atrial substrate, which maintains AF initiated by paroxysms of rapid PV tachycardia.

On the basis of follow-up data presented in this study, we suggest that the difference is not simply rate-related but suggests a highly localized abnormality in patients with PV tachycardia compared with the more diffuse process affecting multiple PVs of PV AF.

Limitations
The relatively small number of patients with tachycardia originating from inferior PVs limited the power of observations pertaining to P-wave morphology and activation patterns for these sites.

Conclusions
PV AT has a distribution similar to PV AF, with a propensity to upper veins. However, the majority of foci are ostial, and only a small percentage occur from deep in the PV. Focal RFA is associated with a high acute and long-term success rate, with freedom from both AT from other sites and from AF. PV AT is a localized process and therefore may represent a process different from PV AF.

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