Electrophysiological features and catheter ablation of symptomatic frequent premature atrial contractions

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Aims
Frequent premature atrial contractions (PACs) are associated with increased risk of atrial fibrillation (AF), stroke, and death. This study aimed to explore the electrophysiological features of PACs with and without inducing AF and to evaluate the effectiveness of catheter ablation for PACs.

Methods and results
Thirty-five consecutive patients with symptomatic, frequent, and drug-refractory PACs in the absence of AF (group A) and 35 patients with PACs-induced AF (group B) were enrolled. Coupling intervals (CoIs) of PACs were compared. Premature atrial contractions were mapped by the point-by-point and/or circular mapping technique. Focal ablation or pulmonary vein/superior vena cava isolation was applied as appropriate. A total of 35 ectopic foci were identified in group A. The majority of them were at pulmonary vein (PV) (n = 7), crista terminalis (n = 6), and para-Hisian area (n = 6). In group B, ectopic foci were in left-sided PVs in 21 patients, in right-sided PVs in 13 patients, and in SVC in 1 patient. There was significant difference in CoIs of PACs triggering AF and those from PVs and non-PV areas but without causing AF (362.8 ± 23.0 ms vs. 470.6 ± 60.1 ms vs. 515.6 ± 77.2 ms, P < 0.001). Premature atrial contractions were abolished in 32 of 35 patients from group A and in all patients from group B. At the end of follow-up, 29 patients in group A and 28 patients in group B were free of recurrence (off antiarrhythmic drugs) after the initial ablation (P = 0.97).

Conclusions
Frequent PACs in the absence of AF were characterized as having their predilection sites and longer CoIs than those inducing AF. Catheter ablation was effective to eliminate symptomatic, frequent, and drug-refractory PACs.

Keywords
Premature atrial contraction • Atrial fibrillation • Coupling interval • Pulmonary vein isolation • Focal ablation

Introduction
Frequent premature atrial contractions (PACs) are sometimes highly symptomatic and refractory to multiple antiarrhythmic drugs (AADs). Frequent PACs are possibly not ‘benign’ because they are associated with increased risk of atrial fibrillation (AF), stroke, and death. Moreover, excessive PACs in the absence of AF may be an underlying cause of stroke of undetermined etiology. It is well known that PACs arising from pulmonary veins (PVs) are the main triggers of AF and both PACs and AF can be abolished by PV isolation. However, it is unknown whether all PACs arising from PVs are AF related and those of a non-PV origin are not. Whereas it seemed applicable to ablate PACs guided by electroanatomic systems according to a few case reports, the effectiveness of catheter ablation for PACs in a patient cohort has not been established. Furthermore, it will be of value to know the electrophysiological characteristics of PACs with or without inducing AF.

This study aims to observe the electrophysiological features of frequent PACs with and without inducing AF and to evaluate the effectiveness of catheter ablation for the treatment of PACs.
What’s new?

- Frequent premature atrial contractions (PACs) in the absence of atrial fibrillation (AF) had their predilection sites. Pulmonary vein, crista terminalis, and para-Hisian area are the most common sites of origin.
- Premature atrial contractions from pulmonary vein (PV) or non-PV areas but without causing AF had significantly longer coupling intervals than those triggering AF.
- Catheter ablation was effective to eliminate symptomatic, frequent and drug-refractory PACs.

Methods

Patient characteristics

From June 2012 to January 2015 this study enrolled 35 consecutive patients with symptomatic PACs in the absence of AF (group A) and 35 patients with paroxysmal AF (group B). In group A, in each patient AF was carefully excluded by thorough review of the whole documentation of surface ECGs and 24-h Holter recordings. Premature atrial contractions burden was 25 567 ± 12 508 (range 8000–64 987) per day. Concomitant short atrial runs with an average paroxysms of 1197 ± 2466 (range 1–8914) per day were recorded in 22 of 35 patients from group A. Frequent PACs were refractory to an average of 2.5 ± 1.4 AADs prior to ablation.

In group B, spontaneous PACs initiating AF were documented for at least three times during ablation procedures in 35 consecutive patients. In them, there were 3.2 ± 4.9 episodes of AF per month (refractory to 2.2 ± 0.9 AADs). Prior to ablation, the frequency of PACs was 5527 ± 4036 (range 31–19 002) per day by 24-h Holter recordings.

The number of PACs in each patient was counted in the daytime (7:00–19:00) and at night time (19:00–7:00) for both groups, and was designated as PAC_d and PAC_n, respectively. The proportion of PAC_d > PAC_n (PAC_d > PAC_n) was compared in two groups.

Warfarin anticoagulation was applied for 1 month in patients at high risk of thromboembolism (CHADS2 score ≥ 1) from group A and in all patients from group B with a target international normalized ratio of 2.0–3.0, and transesophageal echocardiography was applied to rule out left atrial thrombi in these patients. In both groups, all AADs except amiodarone were withdrawn for at least five half-lives. Amiodarone was paused for more than 1 month. All patients provided written informed consent.

Electrophysiological study and mapping

During Electrophysiological (EP) study, local anaesthesia with lidocaine was applied for vein catheterization. In group A, electrophysiological mapping was performed if PACs occurred spontaneously or was induced by isoproterenol infusion (2–20 μg/min). One octopolar mapping catheter was advanced in coronary sinus (CS) via subclavian vein access. If needed, one circular mapping catheter (Lasso, Biosense Webster, Diamond Bar, CA, USA) was positioned at PVs’ ostia to record PV potentials (PVP) via a L1-type Swartz sheath (St. Jude Medical, St. Paul, MN, USA) after a transseptal procedure. Heparin was titrated to maintain an activated clotting time (ACT) range of 300–350 s.

The ectopic P-wave pattern on surface electrograms (ECGs) and activation sequence of CS and PVPs were used to identify the site of origin. The ECG algorithm was primarily based on the morphology of ectopic P-wave on lead V1. Briefly, a negative P-wave indicated a right-side origin and a positive or negative one indicated a left-side origin. A positive/negative P-wave indicated PACs from crista terminalis (CT). An isoelectric P-wave indicated PACs from right septum or peri-nodal area. The proximal to distal CS activation suggested PACs of a right-side origin and distal to proximal indicated those of a left-side origin. If the PVP preceded A-wave and other atrial sites then this PV was considered to be the site of origin.

In group B, after two transseptal procedures, two L1-type Swartz sheaths were introduced into left atrium. Heparin anticoagulation was administered to maintain an ACT at 300–350 s. When spontaneous ectopic PV premature contractions initiated one episode of AF, intracardiac recordings as well as surface ECGs were recorded for offline analysis. If necessary, non-PV triggers were searched by mapping of superior vena cava (SVC) or other non-PV areas.

Electroanatomic mapping (CARTO 3 System, Biosense Webster) was performed by point-by-point technique for a non-PV focus. Activation sequence was displayed in a color-coded manner on the geometry of left or right atrium. Care was taken to exclude the PACs caused by mechanical bumping. The earliest activation site suggested a possible site of origin. When a para-Hisian focus was suspected, mapping was attempted at right atrial side of para-Hisian area and at non-coronary cusp (NCC) via retrograde aortic route. The left atrial geometry was reconstructed for mapping of PACs arising from the PV area.

The coupling intervals (Cols) of PACs were measured in both groups by Prucka 7000 Workstation (GE, USA) utilities during EP procedures. To exclude the impact of adrenergic nerve tone on CoI variation, Col was measured under baseline condition or after complete washout of isoproterenol. Coupling interval was defined as the time interval from the peak/nadir of sinus P-wave to that of ectopic P-wave on 1 of 12 leads of ECG on which the configuration of P-wave could be clearly seen (Figure 1A and B). Coupling interval measurement was repeated for three independent PACs.

The bipolar intracardiac ECGs were filtered at 30–500 Hz and displayed at a speed of 200 mm/s.

Radiofrequency ablation

The procedures were performed under conscious analgesia with continuous infusion of fentanyl. In group A, focal ablation was performed to abolish non-PV foci. Saline-irrigated radiofrequency (RF) energy was delivered at 30–35 W, 43 °C. If PACs disappeared within 20 s, then RF delivery would be prolonged to 90–120 s. Otherwise, ablation would be suspended and another attempt of mapping was applied. For para-Hisian foci, ablation was performed at right atrial side of para-Hisian area or at NCC if necessary. For PV foci, circumferential pulmonary vein isolation (CPVI) was performed to isolate the ‘culprit’ ipsilateral PVs. Pulmonary veins of the opposite side were not targeted for ablation. Prior to CPVI, an irrigated catheter (Navistar ThermoCool, Biosense Webster) was inserted into left atrium via another transseptal puncture.

The CPVI procedure was described in detail elsewhere. Briefly, the ostia of PVs were tagged on the left atrial geometry after selective PV angiography; saline-irrigated RF energy was delivered at 30 W for roof and posterior wall and at 30–35 W for anterior wall. Pulmonary vein isolation was confirmed by the disappearance of PVP or dissociation of PVP with atrial electrical activity. In group A, the endpoint of ablation was elimination of PACs and no recurrence with isoproterenol challenge during 30 min waiting time.

In group B, CPVI was the standard approach. If non-PV foci arising from SVC or other areas were detected, additional SVC isolation or focal ablation would be applied. After PV or SVC isolation, 30 min waiting time was needed to deal with acute PV/SVC re-connection.
Electrophysiological features and ablation of PACs

Post-ablation management and follow-up
Therapeutic warfarin anticoagulation was applied for 2–3 months in patients with left atrial ablation. Anticoagulation would be withdrawn for all patients in group A and for those without AF recurrence in group B. Aspirin 100 mg/day was lasted for 1 month for patients with only right atrial ablation in group A. All AADs were withdrawn if no PACs or AF were detected post-ablation, but could be continued otherwise. Surface ECG was recorded at 1, 3, 6, and 12 months. 24-h Holter monitoring was applied 3, 6, 12 and every 6–12 months post-ablation.

Blanking period was defined as 1 month post-ablation. After the blanking period, the clinical success was defined as freedom from recurrence of frequent PACs (typically < 100/24 h,² off AADs) in group A and from recurrence of atrial tachyarrhythmias in group B (off AADs) at the end of follow-up. A repeat procedure could be performed for recurrent PACs or atrial tachyarrhythmias after the blanking period.

Statistical analysis
Continuous variables were expressed as mean ± SD for normal distribution, or as median, first and third quartiles for non-normal distribution. Coupling interval difference between two groups was primarily compared by unpaired t-test for normal distribution or Wilcoxon–Mann–Whitney test otherwise. Patients from group A could be allocated to two sub-groups (PACs arising from PVs and from non-PV area). Coupling interval difference in two sub-groups of groups A and group B were further compared by analysis of variance for normal distribution or Kruskal–Wallis test otherwise. Discrete variables were expressed as counts or proportions (%), and were compared by the χ² analysis or Fisher exact test. Recurrence-free survival probability was estimated by Kaplan–Meier method and compared by log-rank test. A two-tailed P < 0.05 was considered statistically significant. Statistical analysis was performed with SPSS16.0 for Windows (SPSS Inc., Chicago, IL, USA).

Results

Baseline characteristics
The baseline characteristics in two groups were shown in Table 1. In group A, the proportion of female patients was higher, PACₙᵢ > PACᵢᵦ% was lower, and the left atrium size was smaller than in group B. There were more average PACs in group A than in group B. The mean age, history, left ventricular ejection fraction (LVEF), AADs use and the prevalence of comorbidities were comparable between two groups. In group A, LVEF was within normal range in all but one patient despite high PACs burden. Left ventricular ejection fraction was 45% in this patient, and was attributed to remote anterior myocardial infarction.

Procedural data
The procedures were accomplished in 70 patients. The total procedural and fluoroscopic time was significantly shorter in group A than in group B (106.5 ± 8.3 min vs. 145.6 ± 9.2 min and 10.7 ± 8.3 min vs. 18.4 ± 5.9 min, P < 0.05 for both). The time for ectopic foci mapping was 20.4 ± 10.3 min in group A and 10.1 ± 3.5 min in group B, P < 0.05.

Results of premature atrial contractions mapping in two groups
In group A, PACs occurred spontaneously in 32 patients and were induced by isoproterenol infusion in 3 patients. A total of 35 atrial ectopic foci were identified. No patients had >1 ectopic focus in the cohort of this study. Concomitant premature ventricular contractions (PVCs) were mapped to be of para-Hisian origin in one male patient with PACs of high CT origin. There were 18 (51.4%) right atrial ectopic foci and 17 (48.6%) left atrial ones. The anatomic
The distribution of atrial ectopic foci was shown in Figure 2. The most common sites of origin were PVs (n = 7, 20.0%), CT (n = 6, 17.1%), and para-Hisian area (n = 6, 17.1%), followed by mitral annulus (n = 4, 11.4%) and left atrial posterior wall (n = 3, 8.6%). This was followed by cavo-tricuspid annulus (n = 2, 5.7%) and ostium of left atrial appendage (n = 2, 5.7%). The rare sites of origin were right atrial postero-inferior wall (n = 1, 2.9%), high right atrial septum (n = 1, 2.9%), left atrial roof (n = 1, 2.9%), Koch’s Triangle area (n = 1, 2.9%), and CS ostium (n = 1, 2.9%).

In group B, circular mapping showed ectopic foci were located in left-sided PVs in 21 patients, in right-sided PVs in 13 patients, and in SVC in 1 patient. The patient with an SVC trigger also had a trigger at lateral free wall of left atrium, which initiated AF episodes following frequent PACs of this origin.

**Table 1**: Baseline characteristics in two groups

<table>
<thead>
<tr>
<th></th>
<th>Group A (n = 35)</th>
<th>Group B (n = 35)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>61.0 (46.0, 66.0)</td>
<td>64.0 (56.0, 70.0)</td>
<td>0.10</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>10 (28.6)</td>
<td>24 (68.6)</td>
<td>0.001*</td>
</tr>
<tr>
<td>LAD (mm)</td>
<td>37.1 ± 5.0</td>
<td>41.8 ± 5.0</td>
<td>0.001*</td>
</tr>
<tr>
<td>History (m)</td>
<td>6.0 (3.0, 72.0)</td>
<td>24.0 (4.0, 36.0)</td>
<td>0.41</td>
</tr>
<tr>
<td>Class I/III AADs prior to ablation</td>
<td>2.5 ± 1.4</td>
<td>2.2 ± 0.9</td>
<td>0.21</td>
</tr>
<tr>
<td>Average PACs burden/day</td>
<td>25567 ± 12 508</td>
<td>5527 ± 4036</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>PAC_{n}\text{, PACs }n\text{, PACs number at night time (19:00–7:00)}</td>
<td>16 (45.7)</td>
<td>26 (74.3)</td>
<td>0.03*</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>63.1 ± 7.1</td>
<td>65.9 ± 6.1</td>
<td>0.08</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>11 (31.4)</td>
<td>9 (25.7)</td>
<td>0.60</td>
</tr>
<tr>
<td>CAD, n (%)</td>
<td>1 (2.9)</td>
<td>1 (2.9)</td>
<td>1.00</td>
</tr>
<tr>
<td>HCM, n (%)</td>
<td>1 (2.9)</td>
<td>1 (2.9)</td>
<td>1.00</td>
</tr>
<tr>
<td>Prior history of stroke, n (%)</td>
<td>1 (3.0)</td>
<td>0 (0)</td>
<td>0.31</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>2 (5.7)</td>
<td>4 (11.4)</td>
<td>0.39</td>
</tr>
</tbody>
</table>

LAD, left atrial diameter; AAD, anti-arrhythmic drug; LVEF, left ventricular ejection fraction; CAD, coronary artery disease; HCM, hypertrophic cardiomyopathy; PAC_{n}, PACs number in the daytime (7:00–19:00).

*Statistically significant.

**Analysis of coupling interval in two groups**

The mean CoI was significantly longer in group A than in group B (506.6 ± 76.0 ms vs. 362.8 ± 23.0 ms, P < 0.001). Premature atrial contractions arising from within PVs had markedly shorter CoI than those from non-PV areas (470.6 ± 60.1 ms vs. 515.6 ± 77.2 ms, P = 0.02) in group A. However, PACs of PVs origin in group A had significantly longer CoI than those in group B (470.6 ± 60.1 ms vs. 362.8 ± 23.0 ms, P < 0.001).

**Results of ablation**

In group A, focal ablation was performed for 28 non-PV foci and "culprit" ipsilateral CPVI was applied for 7 PV foci. RF ablation successfully abolished PACs in 32 patients, but failed in the remaining 3 patients. RF ablation was aborted due to transient AV conduction block in 1 patient with PACs at mid-Koch’s triangle, which recovered immediately after ablation cessation. Ablation failed in 1 patient with PACs at postero-inferior RA due to inability to find an ideal target point, and failed in 1 patient with PACs at lateral mitral annulus due to no response to endocardial and epicardial ablation within CS. At the end of the procedure, no acute recurrence of PACs was detected for 32 patients.

In group B, CPVI in 34 patients and SVC isolation/focal ablation at left atrial lateral free wall in 1 patient successfully abolished PACs and subsequent episodes of AF. At the end of 30 min’ waiting time, PV/SVC re-connection in >1 PVs was observed in 18(51.4%)
patients, and was successfully eliminated after additional ablation of 1.5 ± 0.7 gaps in circumferential lesion lines.

Clinical effectiveness during follow-up
Two patients with PACs of PVs origin and one patient with para-Hisian PACs experienced symptom relapse after 7, 25, and 35 days post-ablation in group A. After the blanking period, 24-h Holter monitoring showed no improvement of PACs in all of them. Re-ablation was successful after closing an anterior conduction gap of left PVs in one patient. The other two patients refused to accept re-ablation. Taking 3 patients with acute ablation failure into account, 29 (82.9%) patients were free of recurrence of PACs/short atrial runs (off AADs) after a single ablation procedure at the end of 459.3 ± 307.7 days of follow-up. Zero to <100 PACs were recorded by 24-h Holter in all 29 patients. However, 1 of 29 patients with para-Hisian PACs experienced palpitation 2 days after ablation, and 24-h Holter monitoring recorded frequent PVCs (1200/day pre-ablation vs. 5749/day post-ablation).

In group B, 28 (80.0%) patients were free of recurrent atrial tachyarrhythmias after initial procedure during 565.7 ± 243.5 days of follow-up. Five of seven patients with recurrence underwent repeated ablation. During re-ablation, PV re-connection in >1 PVs was identified in all of them. After closing 1.9 ± 0.6 conduction gaps, all PVs were re-isolated. Further recurrence was detected in 2 of 5 patients during subsequent follow-up. The recurrence-free survival curve in two groups after the first ablation was shown in Figure 3.

Complications
One patient in group A developed left hemothorax 24 h post ablation, which was treated by thoracentesis and drainage without any sequelae. Transient AV conduction block occurred during ablation at mid-Koch’s triangle in one patient in group A and recovered when ablation suspended. There were no other complications in two groups.

Discussion
Main findings of this study
To the best of our knowledge, this was the first study focusing on PACs ablation in a consecutive cohort of patients. The main findings of this study were as follows: first, frequent PACs/short atrial runs were characterized as having their predilection sites. Anatomically, PVs, CT, and para-Hisian area were the most common sites of origin. Second, there was significant difference in Col between PACs inducing AF and those without. Premature atrial contractions triggering AF (mostly of PV origin) had the shortest Col than those from PVs and non-PV areas but without causing AF. Third, the effectiveness of catheter ablation for PACs was similar to that of AF ablation.

Rationale of catheter ablation for premature atrial contractions
Unlike PVCs ablation, PACs ablation was not commonly performed, probably because they were less symptomatic or caused less severe consequence. However, frequent PACs were possibly not ‘benign’ because they were associated with increased risk of AF and stroke.2-6 Rarely, frequent PACs could even induce cardiomyopathy and heart failure.15 Furthermore, PAC ablation might be more challenging than PVC ablation due to the difficulty in identifying the P wave morphology and ablating results during the procedure. However, it was already proved applicable to ablate PACs by three-dimensional mapping technique in previous case reports.19,10 Hence, we conducted a pilot study to evaluate the safety and effectiveness of PAC ablation in a cohort of 35 patients.

Comparison of coupling interval of premature atrial contractions arising from pulmonary veins with and without inducing atrial fibrillation
It was not surprising to see that 7 of 35 PACs in group A were from PVs due to the arrhythmogenesis of myocardial sleeves of PVs.7,8 However, it was interesting to find that there existed significant differences between PACs arising from PVs-induced AF episodes. It was unknown why PACs arising from PVs-induced AF in group B but not in group A. The higher proportion of female patients, lower PACn, > PAC90%, and smaller LA dimension in group A seemed unable to explain this phenomenon, whereas Col difference might be a reasonable explanation. As was reported in a previous study,16 PACs arising from PVs and inducing AF had the shortest Col in comparison with those from PVs and non-PV areas but without inducing AF. However, the underlying mechanisms of the short Col of PACs arising from PVs needed to be further explored.
Techniques for premature atrial contractions mapping and ablation

In this cohort of 35 patients, catheter ablation was highly effective for PACs elimination guided by three-dimensional mapping system. The morphology of P′ wave on ECGs as well as activation sequence of CS provided enough information for a prior judgment for PACs’ site of origin. When performing point-by-point mapping, PACs caused by mechanical bumping should be excluded after careful examination of P′ wave configuration and intracardiac activation sequence, in order to acquire a precise activation map.

Ablation near the conduction systems was challenging. In our series, para-Hisian PACs recurred in one of six patients, and RF ablation at mid-Koch’s Triangle in one patient failed to avoid AV conduction impairment. It was advisable to map para-Hisian PACs in NCC via retrograde aortic approach, but unfortunately they could not be eliminated in NCC in any of the patients in group A. Cryomapping and ablation for para-Hisian PACs might be advantageous over radiofrequency energy for safety consideration. Of note, the risk-benefit of para-Hisian PACs ablation should be considered. The prevalence of AV block was not neglectable if repeated RF ablation at this area was performed.

In our study ‘culprit’ ipsilateral rather than bilateral CPVI or SVCI was applied for ablating PACs arising from PVs or SVCs, in order to avoid unnecessary tissue injury and potential complications. The high long-term success rate of PACs ablation supported the application of this conservative CPVI procedure for treating PACs arising from PVs.

Proper candidates for premature atrial contractions ablation

So far there was no guideline recommendations or expert consensus with respect to PACs ablation. Generally, PACs frequency, symptoms, response to AADs as well as concomitant diseases should be taken into account when considering the indication for ablation. In our study, PACs were highly frequent, symptomatic, and refractory to multiple AADs. Similar to PVCs ablation, the high frequency of PACs served as one of the prerequisites for a successful ablation procedure. Furthermore, PACs were mono-focal in all patients from group A. This might because the patients enrolled in group A had fewer concomitant diseases. Hypertension, as the most common comorbidity, might play a less important role in the genesis of pleomorphic or multifocal PACs. However, the probability of pleomorphic or multifocal PACs did exist in some sub-categories of patients, especially in those with chronic obstructive pulmonary disease. These patients might not be the proper candidates for PACs ablation.

Study limitations

There were several limitations in this study. First, the sample size was not big enough to describe the overview of spatial distribution of PACs’ sites of origin and the effectiveness of catheter ablation. However, the major findings of this pilot study provided sufficient information that frequent PACs had their specific sites of predilection and could be ablated effectively by the available navigation systems. Second, patients in group A were collected by analysing surface ECG and 24-h Holter monitoring. This might miss asymptomatic AF episodes and lead to patients misclassification. Even so, the main results of this study might not have changed, since Cols in group A were always measured in ‘isolated’ PACs and those in group B measured in PACs triggering AF episodes. Third, the point-by-point mapping technique might be time-consuming and non-contact mapping might be a better choice for a rapid mapping process. As we have mentioned, the morphology of P′ wave and CS conduction sequence provided enough information for a correct prior localization of PACs’ site of origin, and it took only ≈20 min to acquire an entire three-dimensional activation map. Therefore, we thought that the point-by-point technique was applicable in routine ablation practice. Third, the effectiveness of catheter ablation for AF might be over-estimated, because trans-telephone ECGs or implanted loop recorders were not applied to rule out asymptomatic or short episodes of AF in this study. However, regular ECGs and 24-h Holter recordings were sufficient to detect the frequency of PACs/short atrial runs (similar to the follow-up protocols for PVCs ablation), and the ablation success rate for PACs would not be over-estimated.

Conclusions

In conclusion, PACs in the absence of AF had their predilection sites. PV, crista terminalis, and para-Hisian area are the most common sites of origin. Premature atrial contractions from PV or non-PV areas but without causing AF had significantly longer Cols than those triggering AF. Catheter ablation was effective to eliminate symptomatic, frequent, and drug-refractory PACs.

Conflict of interest: none declared.

References

Successful catheter ablation of ventricular ectopy in a young patient with implanted Melody valve

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A 17-year-old male after transcatheter implantation of Melody pulmonary valve (TPV) (Medtronic) was admitted for catheter ablation (CA) of frequent and highly symptomatic premature ventricular complexes (PVCs—Figure) refractory to treatment with bisoprolol. The patient had a history of surgical correction of truncus arteriosus. Due to moderate stenosis and severe regurgitation of the valved conduit, a TPV Melody was implanted and was functioning properly at the time of the procedure. Morphology of PVCs implied origin from the right ventricular outflow tract.

The catheter (CoolFlex, SJM) manipulation was guided by intracardiac echocardiography (Siemens Medical Solutions) and NAVX (SJM) electroanatomical mapping system (EAM). Ablation at the best pace-map and the earliest intracardiac bipolar electrogram at the proximal end of the TPV stent (Figure, site 1) was attempted, but was not successful due to frequent impedance changes (Figure lower panel). Ablation at a more proximal location (Figure, site 2) underneath the stent, where the tip of the ablation catheter was not touching the stent, resulted in stable impedance drop and successfully abolished PVCs. There were no procedure-related complications. The patient was free of palpitations at the 3-month follow-up, and 24-h ECG monitoring revealed very sporadic PVCs.

The full-length version of this report can be viewed at: http://www.escardio.org/Guidelines-&-Education/E-learning/Clinical-cases/Electrophysiology/EP-Case-Reports.

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