Non-contact mapping-guided ablation of ventricular arrhythmias originating from the pulmonary artery

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Aims
To identify unique electrophysiological characteristics of pulmonary artery (PA) ventricular arrhythmias (VA) and determine long-term clinical outcomes following non-contact mapping (NCM)-guided ablation.

Methods and results
The NCM array was deployed in consecutive patients undergoing clinically indicated ablation of outflow tract (OT) VA with left bundle branch block morphology, inferior axis and the precordial lead transition zone ≥V3. Activation, pace and NCM mapping parameters, and electrocardiogram analysis of PA VA patients were compared with 50 patients with right ventricular OT (RVOT) or aortic coronary cusps (ACC) foci. Of 170 consecutive patients, 20 (12%) patients (8 male, 39.7 ± 12.8 years old) with PA VA were identified. Electrocardiogram morphologies of PA ventricular tachycardia (VT) (located 10.8 ± 15.1 mm above the PV) were indistinguishable from RVOT VT, particularly those arising from the septal RVOT. Pulmonary artery VT can be mapped and ablated by targeting the site of earliest activation on NCM maps, with success rates of 90% after a single procedure, without anti-arrhythmics and mean follow-up of 5 years.

Pace-mapping in the PA is complicated by frequent inability to capture (P < 0.01). Small far-field atrial potentials and smaller ventricular electrograms were more frequently recorded at successful sites of ablation in the PA (P < 0.05).

Conclusion
Non-contact mapping is a useful technique to map PA VT and ablation at sites of earliest activation above the pulmonary valve is associated with excellent long-term clinical success.

Keywords
Non-contact mapping ● Pulmonary artery ● Premature ventricular complexes ● Right ventricular outflow tract ● Ventricular tachycardia

Introduction
Anatomically, the pulmonary artery (PA) and pulmonary valve (PV) form a continuum with the right ventricular outflow tract (OT). Radiofrequency catheter ablation is an effective therapy for drug-refractory ventricular tachycardia (VT) or symptomatic PVCs originating from the OTs of structurally normal hearts. Most of these arrhythmias originated from the RVOT but VT/PVC originating from the PA has been very seldom reported.¹–⁶

In our institution, the non-contact mapping (NCM) array (Ensite, St Jude Medical, MN, USA) is used routinely as part of our institutional clinical protocol to map right-sided ventricular arrhythmias (VA).¹–⁹ The array affords unique capability of globally mapping electrical activation in an entire chamber within a single beat. The aims of this study are two-fold: firstly to identify unique electrophysiological characteristics of VA arising the PA to distinguish them from those arising from the RVOT or aortic coronary cusps (ACC) and secondly to determine long-term clinical outcomes following NCM-guided ablation of PA VA.

Methods
Study population
Patients included in this study underwent ablation between May 2006 and December 2011. The inclusion criteria included patients with symptomatic idiopathic VT or PVC with left bundle branch block morphology, inferior axis, and the precordial lead transition zone ≥V3. For patients with frequent PVC, PVC burden was ≥20% on 24-h Holter monitoring.¹⁰ All patients who failed or are intolerant of β-blockers, class IC, or III
anti-arrhythmic agents. Patients with structural heart disease and polymorphic VA were excluded from this study.

Consecutive patients with VA electrocardiogram (ECG) morphology fulfilling the inclusion criteria and undergoing clinically indicated ablation were recruited. Age- and gender-matched patients in whom VA originated from the RVOT septum (RVOT-SP) and RVOT free wall (RVOT-FW) as well as patients with ACC foci were used as controls. The study protocol conforms to the ethical guidelines of the 1975 declaration of Helsinki as reflected in a priori approval by our institution’s human research committee.

**ECG analysis**

All ECGs recording were analysed at a paper speed of 50 mm/s. Analysis of the VA ECG focused on the following parameters: coupling interval or tachycardia cycle length of VA, frontal axis, QRS duration, R wave amplitude in lead aVF, transitional zone (defined as the precordial lead position in which the amplitudes of the R and S waves were equal), R wave duration index (defined as a percentage by dividing the R-wave duration by QRS complex duration in lead V1), R/S amplitude ratio (defined as the amplitude of the R wave expressed as a ratio to the S wave amplitude ratio in lead V1), intrinsicoid deflection time (defined as interval from earliest ventricular activation to the peak of R wave in V2), and RS complex duration (defined as interval from earliest ventricular activation to nadir of the first S wave in any of precordial lead). The TP segment was considered the isoelectric baseline for measurement of R and S wave amplitude. The QRS duration was measured from the site of earliest initial deflection from the isoelectric line in any lead to the time of latest activation in any lead. For VT cases, QRS measurements were performed on the first beat of VT or isolated PVC representative of the clinical VT before the induction of sustained VT.7,11–17

**Non-contact mapping**

The NCM system was deployed with the multi-electrode array (MEA) placed at the optimal mapping position such that one-third of the MEA was above PV. The three-dimensional geometry of RVOT and PA was constructed by navigating the ablation catheter within the RVOT and PA around the MEA. The PV was defined by a combination of voltage mapping, assessment of local potentials (typically small atrial potentials visible), and contrast pulmonary angiography.7,8 The EA site was marked on the geometry and defined by the earliest unipolar deflection from baseline forming a single spot on the isopotential map and characterized by a QS pattern of non-contact unipolar electrogram.7,8 The breakout (BO) site was marked as the site along the depolarization pathway identified by the colour-coded activation map where rapid centrifugal electrical propagation originated from and local unipolar electrograms exhibited the maximum negative dV/dt. For identification of the EA and BO sites, a broad colour band setting was used with colour high (defined as unipolar electrogram baseline) at $-0.1$ mV and colour low at $-2$ mV. The virtual unipolar high-pass filters set at 4 Hz.7,8 The following parameters were assessed: distances between EA, BO sites, and PV distances. Local activation at EA and BO sites based on contact bipolar electrograms from the mapping catheter was recorded. Bipolar pace-mapping, at twice diastolic threshold and 0.5 ms pulse width, to determine the degree of matching with the clinical VA was performed. In the ACC group, the Ensite Array was not re-deployed within the aorta. Instead contact mapping was performed using Ensite NavX system (St Jude Medical, St. Paul, MN, USA).

**Ablation**

As part of a previously reported study protocol, patients were randomized to have the first ablation performed at either the EA or BO sites.7 If this was acutely unsuccessful, the ablation was performed at the alternative site. Radiofrequency energy was applied in temperature-control mode with the power output titrated up to 35 W to achieve a target temperature of $50–60$ °C for 60 s using standard 4 mm-tipped non-irrigated catheters (Biosense Webster Inc., Diamond Bar, CA, USA or EP Technologies Inc., San Jose, CA, USA). Ablation was considered to be acutely successful if the VT or PVC were eliminated during the ablation and/or became non-inducible with programmed electrical stimulation and isoproterenol infusion.

**Follow-Up**

After the procedure, continuous ECG monitoring was performed for 24 h. Anti-arrhythmic agents were not restarted in the absence of arrhythmia recurrence. All patients were reviewed in an outpatient clinic monthly after the procedure for the first 3 months, and then followed up every 6 months. A 12-lead ECG and 24-h Holter monitoring were performed at every clinic visit. Arrhythmia recurrence was defined as symptomatic recurrence with ECG documentation of VA on either the 12-lead ECG and/or Holter monitoring, without the use of anti-arrhythmic agents.

**Statistical analysis**

Continuous variables and categorical variables were described by means ± SD and percentages, respectively. An independent samples t-test was performed for comparisons of two groups. Chi-square test was used for non-parametric analysis. One-way ANOVA was used for comparisons among three or more groups. A P-value of $<0.05$ was considered to be statistically significant.

**Results**

**Patients’ characteristics**

One hundred and seventy consecutive patients with VA ECG morphology fulfilling the inclusion criteria and undergoing clinically indicated ablation were recruited. Of them, VA originated from the RVOT (below the PV) in 136, ACC in 10, LV summit in 4 subjects and the PA in 20 subjects. Twenty age- and gender-matched patients in whom VA originated from RVOT-SP and RVOT-FW together with all 10 patients with ACC foci (five from the left coronary cusp, three from the right coronary cusp, and two from the anterior wall between the left and right coronary cusps) were used as controls. The demographics of these four groups are listed in Table 1. The ACC group had a significantly higher PVC burden.
In the PA group, consecutive 20 patients (40% male, 39.7 ± 12.8 years old) underwent mapping and catheter ablation for symptomatic VA. Fourteen (85%) patients had non-sustained VT and PVC, and six (15%) had only frequent PVC. Five patients have had undergone unsuccessful ablation procedure previously, whilst the remaining 15 patients were undergoing their index ablation. VA originated from the PA free wall in 8 (40%) patients and the septal PA in the remaining 12 (60%) (Figures 1 and 2).

**ECG morphology**

The chosen ECG parameters were unable to distinguish between VA arising from the RVOT-SP and PA (Table 2). As expected, VA arising from the RVOT-FW had more leftward axis due to lower R wave amplitude in the inferior leads. Ventricular arrhythmias arising from the RVOT-FW, PA free wall, and ACC-exhibited broader QRS complexes. Aortic coronary cusp VA transited earlier in the precordial plane and had a more prolonged interval to the first deflection as measured by either the time to first intrinsicoid deflection or RS plane and had a more prolonged interval to the first deflection as compared with lead II (II/III ratio of 1.4 ± 0.5). In contrast, PVC that originated from the septal aspect of the PA had an rSr’, qRs, rS, or Qr morphology in lead I and taller/equivalent R waves in lead III compared with lead II (II/III ratio of 1.0 ± 0.2, P < 0.01).

**Mapping and ablation**

In the PA group, VA arising from the right free wall of the PA exhibited an R/R’ morphology in lead I and taller R waves in lead II compared with lead III (II/III ratio of 1.4 ± 0.5). In contrast, PVC that originated from the septal aspect of the PA had an rS’ morphology following a steam-pop during ablation (temperature-control mode 30 W, 43 °C, non-irrigated ablation). The resultant pericardial effusion was initially drained by pericardiocentesis but the perforation eventually necessitated surgical repair.

**Follow-up**

All patients completed electrocardiographic follow-up. Two patients declined to attend the clinic physically but Holter recordings were performed at their local hospitals and sent to our institution for analysis.
analysis. The remaining patients completed follow-up for at least 37 months. With a mean follow-up of 69.8 ± 17.7 months, PVC re-occurred in two subjects: one patient in PA group (14,243 PVC in 24 h after the index ablation) and one patient in RVOT-FW group (5,228 PVC in 24 h after the index ablation). Both patients underwent successful repeat ablation. In the remaining 68 patients, a significant reduction in PVC burden (732.9 ± 3295.2 PVC in 24 h before ablation compared with 28,608 ± 18,229.3 PVC in 24 h before ablation; \( P < 0.01 \)) was seen in all groups (Tables 1 and 3).

**Discussion**

**Major findings**

Ventricular arrhythmias arising from the PA exhibits ECG morphologies that are indistinguishable from the RVOT. These uncommon foci are located at a mean of 10.8 mm above the PV, propagate at the same speed as sub-valvular foci before breaking out to activate the ventricular myocardium at a mean of 15.9 mm before the PV. These PA foci can be mapped and ablated using the NCM array with excellent long-term success rates of 90% with a single procedure, without the concomitant use of anti-arrhythmic medications. Pace-mapping in the PA is complicated by frequent inability to capture. Small far-field atrial potentials and smaller ventricular electrograms were often recorded at the successful sites of ablation.

**ECG morphologies of ventricular arrhythmias arising from the pulmonary artery**

Ouyang and co-workers demonstrated that V1 R wave duration and amplitude index can be used to differentiate between VA originating from the pulmonary artery and RVOT. These patterns are characterized by a prominent R wave in V1 with a duration greater than 70 ms and an amplitude greater than 0.05 mV. The presence of these markers can help in identifying the origin of ventricular arrhythmias in the PA.
Table 2 ECG parameters

<table>
<thead>
<tr>
<th>Variables</th>
<th>PA group (n = 20)</th>
<th>Septal PA group (n = 12)</th>
<th>PA free wall group (n = 8)</th>
<th>RVOT-SP group (n = 20)</th>
<th>RVOT-FW group (n = 20)</th>
<th>ACC group (n = 10)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT/PVC QRS axis (°)</td>
<td>90.9 ± 7.3</td>
<td>92.0 ± 5.6</td>
<td>88.7 ± 9.1</td>
<td>87.9 ± 9.4</td>
<td>76.2 ± 10.2</td>
<td>88.9 ± 15.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>aVF R wave amplitude (mv)</td>
<td>1.5 ± 0.4</td>
<td>1.5 ± 0.3</td>
<td>1.8 ± 0.6</td>
<td>1.5 ± 0.4</td>
<td>1.1 ± 0.4</td>
<td>1.7 ± 0.7</td>
<td>0.02</td>
</tr>
<tr>
<td>Coupling interval (ms)</td>
<td>446.9 ± 69.6</td>
<td>432.6 ± 72.7</td>
<td>481.6 ± 70.8</td>
<td>401.4 ± 69.1</td>
<td>447.9 ± 63.9</td>
<td>477.2 ± 92.6</td>
<td>0.04</td>
</tr>
<tr>
<td>II QRS duration (ms)</td>
<td>145.1 ± 12.9</td>
<td>142.2 ± 12.6</td>
<td>158.7 ± 6.1</td>
<td>140.6 ± 13.7</td>
<td>161.1 ± 17.4</td>
<td>161.8 ± 10.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Transitional zone</td>
<td>3.6 ± 0.7</td>
<td>3.5 ± 0.7</td>
<td>4.0 ± 0.8</td>
<td>4.0 ± 0.5</td>
<td>4.0 ± 0.75</td>
<td>2.7 ± 1.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>V1 R-wave duration index (%)</td>
<td>26.3 ± 13.9</td>
<td>25.6 ± 14.4</td>
<td>33.5 ± 12.1</td>
<td>20.8 ± 15.8</td>
<td>27.1 ± 8.6</td>
<td>43.2 ± 11.1</td>
<td>0.001</td>
</tr>
<tr>
<td>V1 R/S amplitude ratio (%)</td>
<td>0.2 ± 0.1</td>
<td>0.2 ± 0.1</td>
<td>0.2 ± 0.1</td>
<td>0.1 ± 0.1</td>
<td>0.1 ± 0.1</td>
<td>0.5 ± 0.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Intrinsicoid deflection time V2</td>
<td>49.2 ± 13.1</td>
<td>51.0 ± 13.9</td>
<td>45.0 ± 11.5</td>
<td>52.2 ± 7.2</td>
<td>57.8 ± 11.5</td>
<td>73.1 ± 11.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RS complex duration</td>
<td>71.9 ± 14.8</td>
<td>73.6 ± 14.9</td>
<td>66.8 ± 16.3</td>
<td>74.0 ± 8.3</td>
<td>79.9 ± 12.5</td>
<td>100.5 ± 15.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are shown as mean ± SD.
ACC, aortic coronary cusps; PA, pulmonary artery; RVOT-FW, right ventricular outflow tract free wall; RVOT-SP, right ventricular outflow tract septum; PVC, premature ventricular contractions; TZ, transition zone; VT, ventricular tachycardia.

Figure 3 ECG morphology, bipolar, unipolar electrograms and prematurity of local potential at EA site of RVOT-SP, RVOT-FW, septal PA, and PA free wall PVC. Far-field atrial potentials (arrow indicates) are visible at the origin of the septal PA and free wall PA PVC (C, D). FW, free wall; PA, pulmonary artery; PV, pulmonary valve; PVC, premature ventricular complexes; RVOT, right ventricular outflow tract; SP, septum.

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from the ACC and RVOT, which is consistent with our findings. However, ECG algorithms are less accurate when sub-localizing within the RVOT. In this study, no significant distinguishing electrocardiographic feature were detected between VA arising from the PA and the RVOT, particular the septal RVOT. Only when the PA foci were further sub-divided into those arising from the right free wall of the PA and leftward septal wall of the PA, those arising from the latter demonstrated a broader QRS complex. These findings are in agreement with previously published but smaller case series. To the contrary, Sekiguchi and colleagues measured significantly larger
Table 3  Procedural and NCM details

<table>
<thead>
<tr>
<th>Variables</th>
<th>PA group (n = 20)</th>
<th>RVOT-SP group (n = 20)</th>
<th>RVOT-FW group (n = 20)</th>
<th>ACC group (n = 10)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute success (n)</td>
<td>19</td>
<td>20</td>
<td>20</td>
<td>10</td>
<td>0.47</td>
</tr>
<tr>
<td>Procedure time (min)</td>
<td>184.2 ± 64.9</td>
<td>171.4 ± 60.8</td>
<td>160.5 ± 58.5</td>
<td>183.1 ± 60.5</td>
<td>0.66</td>
</tr>
<tr>
<td>Fluoroscopy time (min)</td>
<td>11.0 ± 8.7</td>
<td>13.2 ± 13.4</td>
<td>21.5 ± 32.5</td>
<td>14.8 ± 7.3</td>
<td>0.64</td>
</tr>
<tr>
<td>EA BO distance (mm)</td>
<td>16.7 ± 9.7</td>
<td>7.5 ± 5.5</td>
<td>10.0 ± 5.4</td>
<td>–</td>
<td>0.03</td>
</tr>
<tr>
<td>EA PV distance (mm)*</td>
<td>–10.8 ± 15.1</td>
<td>17.2 ± 10.3</td>
<td>21.1 ± 10.0</td>
<td>–</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BO PV distance (mm)</td>
<td>15.9 ± 9.2</td>
<td>23.5 ± 10.3</td>
<td>22.9 ± 8.2</td>
<td>–</td>
<td>0.04</td>
</tr>
<tr>
<td>EA BO time (ms)</td>
<td>11.1 ± 6.6</td>
<td>7.3 ± 4.0</td>
<td>7.8 ± 1.7</td>
<td>–</td>
<td>0.20</td>
</tr>
<tr>
<td>Conduction velocity between EA and BO sites (ms)</td>
<td>1.6 ± 1.1</td>
<td>1.2 ± 1.2</td>
<td>1.3 ± 0.7</td>
<td>–</td>
<td>0.53</td>
</tr>
<tr>
<td>Prematurity of local electrogram at EA site (ms)</td>
<td>24.6 ± 12.2</td>
<td>22.3 ± 8.5</td>
<td>23.0 ± 9.3</td>
<td>37.1 ± 28.0</td>
<td>0.08</td>
</tr>
<tr>
<td>Prematurity of local electrogram at BO site (ms)</td>
<td>19.9 ± 5.5</td>
<td>18.8 ± 7.4</td>
<td>20.4 ± 8.3</td>
<td>–</td>
<td>0.81</td>
</tr>
<tr>
<td>Ablation at EA site only</td>
<td>10/20</td>
<td>9/20</td>
<td>8/20</td>
<td>10/10</td>
<td>0.01</td>
</tr>
<tr>
<td>Ablation at BO site only</td>
<td>0/20</td>
<td>4/20</td>
<td>6/20</td>
<td>–</td>
<td>0.03</td>
</tr>
<tr>
<td>Ablation at EA and BO sites</td>
<td>10/20</td>
<td>7/20</td>
<td>6/20</td>
<td>–</td>
<td>0.40</td>
</tr>
<tr>
<td>RF ablation deliveries (n)</td>
<td>4.3 ± 3.0</td>
<td>3.7 ± 2.1</td>
<td>5.6 ± 7.2</td>
<td>2.6 ± 0.9</td>
<td>0.37</td>
</tr>
<tr>
<td>RF ablation power (W)</td>
<td>33.5 ± 5.6</td>
<td>36.2 ± 6.5</td>
<td>38.1 ± 6.9</td>
<td>20.1 ± 7.0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>RF ablation temperature (°C)</td>
<td>46.9 ± 6.2</td>
<td>47.6 ± 5.6</td>
<td>48.6 ± 3.0</td>
<td>44.8 ± 7.2</td>
<td>0.41</td>
</tr>
<tr>
<td>PVC post-ablation (n)</td>
<td>1709 ± 3755</td>
<td>9 ± 24</td>
<td>288 ± 1164</td>
<td>6 ± 18</td>
<td>0.06</td>
</tr>
</tbody>
</table>

The italic values indicate that number of this NCM parameter in the ACC group.

Data are shown as mean ± SD.

ACC, aortic coronary cusp; EA, earliest activation; BO, breakout; PA, pulmonary artery; RVOT-FW, right ventricular outflow tract free wall; RVOT-SP, right ventricular outflow tract septum; PVC, premature ventricular contractions; RF, radiofrequency; VT, ventricular tachycardia.

*Negative values denote distance superior to the PV.

inferior lead R wave amplitudes, aVL/aVR Q-wave amplitude, and R/S ratio on lead V2 in a cohort of 24 patients with PAVA when compared with 48 RVOT VA patients.1 However, these authors also acknowledge whilst significant differences could be seen in ECG characteristics between the PA and RVOT groups, there was marked overlap of characteristics between these groups, rendering it difficult to identify a cut-off value in any of these ECG parameters to allow for sufficient predictive value. It is also interesting to note in the study by Sekiguchi et al., only 2 of 24 PA foci were located in the right free wall of the PA, compared with 40% in our cohort. Nonetheless, even when our PA group was sub-divided into those with septal or right free wall foci, our conclusions remained unchanged.

Non-contact mapping and ablation for pulmonary artery VT/PVC

We have previously demonstrated the effectiveness of the NCM system in mapping and targeting VA arising from the RVOT with long-term (mean of 3 years follow-up) success rates in excess of 86% after a single procedure without the use of anti-arrhythmic agents.7 Non-contact mapping-guided mapping and ablation of PA VA is associated with similar effectiveness. The unique ability of the NCM system to record global electrical activation within a single chamber also affords additional insights into these uncommon arrhythmic foci. The site of earliest activation (EA site) during PA VT recorded by the NCM array is located at a mean of 10.8 mm above the PV. We have shown in a previously randomized study that ablation of RVOT foci is acutely more successful if the first lesions were delivered at the EA (88%) rather than the BO (66%) sites.7 In comparison, initial ablation performed at BO sites in 10 randomly assigned PA VT patients were all acutely unsuccessful. Procedure success was only achieved by ablation at the EA sites. This greater disparity could be due to the larger EA-BO site distances and thus minimal impact on the focus by such distant ablation. In a post-mortem study of 95 PA PVC from subjects without cardiac disease, ventricular myocardial extensions beyond the PV annuli were found in 17% of subjects, twice more frequent than extensions beyond the aortic valve annuli.25 These PA myocardial extensions were usually found in the adventitial layer, continuous with underlying ventricular myocardium, and most commonly located in anterior pulmonary sinus (50% vs. 39% in the left pulmonary sinus and 11% in the right pulmonary sinus). Given such contiguous muscle fibre arrangements, the PA should be practically considered as an electrical continuum of the RVOT. It is thus not surprising to record conduction velocities in the PA which are similar to the RVOT.

Study limitations

Intra-cardiac echocardiography was not utilized in these cases which would have provided a more accurate localization of the PV and confirmation that the sites of ablation were supra-valvular. Instead, voltage mapping, qualitative assessment of local potentials, and contrast pulmonary arteriography were performed in all patients to document the exact ablation site in relation to the PV. Twenty-five per cent of patients with PA VA had unsuccessful ablation attempts prior to referral to our centre. It is possible that prior ablation may
have affected their ECG characteristics but excluding these five patients from the analysis did not alter the findings.

Conclusion

Non-contact mapping is a useful technique to map PAVT and ablation at sites of earliest activation above the PV is associated with long-term success of 90%. Ventricular tachycardia arising from the PA cannot be distinguished electrophysiologically from those originating from the RVOT.

Conflict of interest: none declared.

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