Is the 12-lead electrocardiogram during antidromic circus movement tachycardia helpful in predicting the ablation site in atriofascicular pathways?

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Received 12 December 2013; accepted after revision 25 February 2014; online publish-ahead-of-print 28 March 2014

Aims
Unlike in the Wolff–Parkinson–White syndrome, there has been no systematic study on the role of the pre-excitation pattern in predicting the ablation site in patients with atriofascicular (AF) pathways. We assessed in a large cohort the value of the 12-lead electrocardiogram (ECG) during antidromic tachycardia (ADT) to predict the site of ablation.

Methods and results
Forty-five patients were studied, 23 males (51%), mean age of 27 ± 12 years with 46 AF pathways and 48 ADT using the AF pathway for A–V conduction. Inclusion required induction of a sustained ADT and successful ablation. Ablation site was assessed during LAO 45° projection and clockwise classified as hours in posteroseptal, posterolateral, lateral, anterolateral, and anteroseptal tricuspid annulus as follows: 05:00–07:00, >07:00–08:00, >08:00–09:00, >09:00–11:00, and >11:00–13:00 o’clock. The QRS axis was assessed during ADT and classified as normal (> +15°), horizontal (+15° to −30°), and superior (< −30°). During ADT axis was superior (−57° ± 10°) in 15 (31%), horizontal (−11° ± 14°) in 22 (46%), and normal (+45° ± 16°) in 11 (23%) patients. The correct ablation site did not differ between the different groups of QRS axis. QRS width during ADT was narrower in patients with a normal when compared with a horizontal and leftward axis (127 ± 14 vs. 145 ± 12 ms, P < 0.0001), and the V–H interval was shorter (4 ± 3 ms vs. 19 ± 22 ms, P = 0.03).

Conclusions
There was no correlation between the AF pathway ablation site and the QRS axis during ADT. The 12-lead ECG during maximal pre-excitation does not predict the proper site of tricuspid annulus ablation in patients with A–V conduction over an AF pathway.

Keywords
Atrofascicular pathway • Mahaim fiber • Catheter ablation • Radiofrequency • Mapping

Introduction
The role of the 12-lead electrocardiogram (ECG) in recognizing the presence of an atriofascicular (AF) pathway has been addressed in previous studies.1–3 In contrast to the widened left bundle branch block shaped QRS during a tachycardia with A–V conduction over the AF pathway, during sinus rhythm overt pre-excitation is usually absent. Only subtle ECG abnormalities suggesting the presence of pre-excitation of a small area of the anteroapical right ventricle (RV) (an rS or rsR pattern in Lead III, absence of septal q waves in Leads I and V6) were found in up to 72% of a cohort of 33 patients.5

The initial part of the AF pathway is usually ablated in the lateral aspect of the tricuspid annulus, guided by an accessory pathway potential, but it can also be located in other areas like the anterior, anterolateral, posterior, and posteroseptal regions of the annulus.4 During antidromic tachycardia (ADT), the frontal plane QRS axis

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What’s new?

- The present paper assesses in a large cohort of atriofascicular pathways whether the 12-lead electrocardiogram (ECG) during maximal pre-excitation (antidromic tachycardia, ADT) is helpful in predicting the site of ablation at the tricuspid annulus. There is a wide variability in the pre-excited QRS morphology due to a wide range of QRS axis during tachycardia (from +75° through −75°). The manuscript shows that despite this wide range of QRS frontal plane axis, the ablation site cannot be predicted by the 12-lead ECG.

- A normal QRS axis was present in ~25% of the cohort. Interestingly, such patients also show a narrower QRS complex and a shorter V–H interval during ADT. Atriofascicular pathways with a normal axis are not located in the anteroseptal aspect of the annulus in contrast to patients with Wolff–Parkinson–White. An alternate explanation for this finding is discussed in the paper.

shows wide variations among patients, from a superior to a horizontal and normal axis.24–6

Assessment of the delta wave in the 12-lead ECG in patients with the Wolff–Parkinson–White (WPW) syndrome can accurately predict the location of the rapidly conducting accessory A–V pathway, leading to location-specific algorithms.7,8 However, there has been no systematic study assessing the QRS configuration on the 12-lead ECG during maximal pre-excitation in relation to the proper site of ablation of AF pathways on the tricuspid ring. We therefore studied in a large cohort of patients with AF pathways, who underwent successful catheter ablation, if the 12-lead ECG during maximal pre-excitation in relation to the proper site of ablation of AF pathways on the tricuspid annulus.

Population and methods

Population: Tables 1 and 2 show the clinical and electrocardiographic characteristics of the 45 patients included in this study.

Data were collected retrospectively in 38 patients and prospectively in 7 patients. The study population had a mean age of 27 ± 12 years, with 23 males (Tables 1 and 2), and came from four Institutions (Biocor Instituto, Nova Lima, Brazil; Arrhythmia Associates, Mumbai, India; UN Mehta Institute of Cardiology and Research Centre, Ahmedabad, India; and University Hospital, Maastricht, The Netherlands).

Inclusion criteria: (i) presence of an AF pathway and inducibility of an ADT, with exclusive A–V conduction over the AF pathway and V–A conduction over the bundle branch-His-A–V node axis, (ii) a 12-lead ECG during ADT, (iii) successful radiofrequency catheter ablation targeting an accessory pathway potential at the tricuspid annulus, and (iv) a recorded fluoroscopy image of the ablation catheter at the site of successful ablation in left anterior oblique (40–50°) and right anterior oblique (30°) views (Figure 1).

Exclusion criteria: (i) Ebstein’s disease with major tricuspid valve displacement, (ii) absence of an accessory pathway potential at the annulus or sub-annular area.

Additional arrhythmia substrates: One patient (Case 10) had two AF pathways. Two patients (Cases 38 and 41) had two different ADTs (short and long ventriculo-His (V–H) tachycardia with different QRS axes). Five patients also had inducible atrioventricular nodal tachycardia (AVNRT), without having pre-excited bystander AVNRT. Four patients had an additional accessory A–V pathway. Two patients had Ebstein’s disease with mild displacement of the tricuspid valve.

Definitions

Antidromic decrementally conducting circus movement tachycardia (ADT): a tachycardia incorporating a slow and decrementally conducting accessory A–V pathway as antegrade limb of the tachycardia circuit.

Atriofascicular pathway: a decrementally conducting accessory A–V pathway connecting the right atrium with a part of the right bundle branch. Characterized by a QRS complex width during tachycardia ≤160 ms, and a short V–H interval (<40 ms), unless retrograde conduction occurs over the left bundle branch because of retrograde right bundle branch block (Cases 38 and 41, Table 2).

Categories of QRS axis: The QRS axis was categorized as normal (+15° or greater, positive in Lead II with an R wave in Lead II equal to or greater than R wave in Lead I), horizontal (+15° to −30°, positive in Lead II with R wave in Lead II less than R wave in Lead I), or superior (axis −30° or less). Two authors (E.B.S. and H.J.J.W.) classified the QRS axis and mismatches in the axis classification were discussed and a consensus agreement reached in all cases.

Classification of the site of ablation: The site of ablation at the tricuspid annulus was categorized into five regions based upon the left anterior oblique fluoroscopic view at 40–50°: posteroseptal and posterior aspect (>5 and ≤7 o’clock), posterolateral (>7 and ≤8

Table 1 Demographics and electrocardiographic parameters according to three pre-defined types of QRS frontal plane axis

<table>
<thead>
<tr>
<th>QRS frontal plane axis</th>
<th>N</th>
<th>%</th>
<th>Age</th>
<th>QRS axis</th>
<th>QRS width</th>
<th>V–H interval</th>
<th>Cycle length</th>
<th>Ablation site o’clock (LAO)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior</td>
<td>≤−30</td>
<td>15</td>
<td>31%</td>
<td>28 ± 14</td>
<td>−57 ± 10</td>
<td>153 ± 09</td>
<td>24 ± 26</td>
<td>322 ± 37</td>
</tr>
<tr>
<td>Horizontal (−30 to +15)</td>
<td>22</td>
<td>46%</td>
<td>28 ± 11</td>
<td>−11 ± 14</td>
<td>138 ± 11</td>
<td>16 ± 17</td>
<td>324 ± 39</td>
<td>8±24</td>
</tr>
<tr>
<td>Normal</td>
<td>&gt;+15</td>
<td>11</td>
<td>23%</td>
<td>28 ± 12</td>
<td>45 ± 16</td>
<td>127 ± 14</td>
<td>4 ± 3</td>
<td>298 ± 36</td>
</tr>
<tr>
<td>P value</td>
<td>ns</td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
<td>0.03</td>
<td>ns</td>
<td>ns</td>
<td></td>
</tr>
</tbody>
</table>
### Table 2 Clinical, electrocardiographic, and electrophysiological characteristics of the study subjects

<table>
<thead>
<tr>
<th>Pt</th>
<th>CMT</th>
<th>Age</th>
<th>Gender</th>
<th>Other Circuit</th>
<th>Ebstein</th>
<th>Site</th>
<th>AP Pot</th>
<th>LAO 45</th>
<th>ADT CL</th>
<th>VH</th>
<th>QRS width</th>
<th>QRS axis</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>AVF</th>
<th>V1</th>
<th>R/S &gt;1</th>
</tr>
</thead>
</table>
| Superior axis | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | ...
Predicting ablation site in Mahaim fiber

<table>
<thead>
<tr>
<th>Patient</th>
<th>Gender</th>
<th>Age</th>
<th>Tachycardia Cycle Length</th>
<th>Ablation Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>28</td>
<td>300 ms</td>
<td>8 o'clock</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>30</td>
<td>320 ms</td>
<td>9 o'clock</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>32</td>
<td>340 ms</td>
<td>10 o'clock</td>
</tr>
</tbody>
</table>

Statistical analysis

Values are given as mean ± standard deviation. The significance of differences (P < 0.05) between the groups of clinical and electrophysiological parameters was assessed by Fisher’s exact test for categorical variables and by t-test for continuous variables, using the STATA statistical software package (STATA 11 Software).

Results

Forty-five patients, mean age 27 ± 12 years, 23 males, with 46 AF pathways and 48 ADTs were included in the study. During tachycardia all 45 patients showed V–A conduction over the right bundle branch–His bundle axis as documented by the short (V–H bundle activation interval of ≤ 40 ms. Patient 10 had two AF pathways. Patients 38 and 41 had in addition an ADT with a longer V–H interval because of retrograde block in the right bundle branch. The mean V–H interval in all 48 ADT was 15 ± 20 ms. All patients showed a predominant R (or r) wave in Lead I during tachycardia.

Catheter ablation at the tricuspid annulus: the site of ablation of the 46 AF pathways is shown in Figure 2. No AF pathway was ablated in anteroseptal or midseptal regions.

A tachycardia with a normal QRS axis: ranging from +25° to +75° (45° ± 16°) was present in 11 patients. The mean tachycardia cycle length was 298 ± 36 ms, the mean V–H interval during ADT was 4 ± 3 ms, and the mean QRS width was 127 ± 14 ms (Table 1). The site of ablation distribution of each case is shown in Figure 2. The average site of ablation in patients having an ADT with a normal axis was at 07:57 o’clock, as assessed in LAO 45°.

Tachycardia with a horizontal QRS axis: 21 patients (mean age of 28 ± 11 years, 38% males) with 22 tachycardias (46% of the 48 ADT) fell in this subgroup with a QRS axis ranging from +10° to −30° (−11° ± 14°). The mean tachycardia cycle length was 298 ± 36 ms, the mean V–H interval during ADT was 16 ± 17 ms, and the mean QRS width was 138 ± 11 ms. The average ablation site was at 08:24 o’clock.

Tachycardia with a superior QRS axis: 15 patients (mean age of 28 ± 14 years, 73% males) with 15 tachycardias (31% of the 48 ADT) were classified in this subgroup with the axis ranging from −30° to −75° (−57° ± 10°). The mean tachycardia cycle length was 332 ± 37 ms, the mean V–H interval during ADT was 24 ± 26 ms, and the mean QRS width was 153 ± 9 ms. The average ablation site was at 08:06 o’clock.

Statistical analysis: There was no correlation between the axis during ADT as classified into three groups (superior, horizontal, and normal) and the ablation site as categorized into the regions posteroseptal, posterolateral, lateral, anterolateral, and anteroseptal (Fisher’s exact test yielded P = 0.24). Figures 3 and 4 show examples of patients with ADT and different QRS axis ablated at the same sector at the tricuspid ring. There was no statistical difference between age and tachycardia cycle length between the groups of
ADT as classified by axis. However, we found a significant difference between the QRS complex width in ADT with a normal axis when compared with the other two axis categories \((P < 0.0001)\) (Table 1). We also found that the V–H interval during ADT and normal QRS axis was significantly shorter than V–H during ADT with both horizontal and superior axis \((P = 0.03)\); however, there was no difference between the V–H interval during ADT with horizontal or superior axis \((P = 0.2)\) (Figure 5).

**Discussion**

Our major findings are (i) no correlation between the frontal QRS axis (superior, horizontal, and intermediate) during ADT and the location of the AF pathway at the tricuspid annulus; (ii) tachycardias showing an intermediate axis were associated with an AF pathway located at a lateral or posterolateral site on the tricuspid annulus and not on the anterosetal site as is the case in a rapidly anterograde conducting accessory A–V pathway.
Why does the location of the atriofascicular pathway at the annulus not correlate with a specific frontal plane axis as it does in patients with Wolff–Parkinson–White?

In the WPW patient, the short anterogradely, rapidly conducting A–V pathway is a muscular bridge inserting into the ventricle just beneath the A–V annulus, therefore the atrial and ventricular ends are close together. This results during A–V conduction over the accessory pathway in a frontal QRS axis related to the accessory pathway location at the annulus. This also holds for a decrementally conducting short AV fibre which inserts close to the AV annulus. Such a situation is not the case in the AF pathway, where there is a considerable distance between the atrial part and the distal end of the AF pathway inserting into the RV. As recently published by Gandhavadi et al. all induced ADT’s in their AF cohort had their earliest ventricular activation at the RV free wall, near the insertion of the moderator band into the anterior papillary muscle. He and others suggested that nearly all AF pathways insert into the RBB, and variations in the frontal plane axis could be explained by a variable degree of fusion of ventricular activation between anterograde conduction over the AF pathway and following retrograde invasion into the RBB partial left ventricular activation over the left sided conduction system, especially the anterior fascicle. The proximal end of the AF pathway gives rise to an accessory pathway potential (the so-called proximal Mahaim potential, which is targeted for ablation but plays no role in the QRS configuration during tachycardia). In other words, no matter where in the tricuspid annulus the proximal end is located, the frontal plane QRS axis during maximal pre-excitation will not be affected by its annular location.

Figure 3 Twelve-lead ECG of three different patients with ADT showing normal, horizontal, and superior frontal plane QRS axis ablated from the same sector of the tricuspid annulus (site of ablation between 07:00 and 07:30 o’clock).
How to explain the differences in the frontal plane QRS axis during antidromic tachycardia in patients with atriofascicular pathways?

We did not have a single AF pathway in our cohort ablated in an anteroseptal location, in spite of the fact that 11 patients showed a normal frontal plane QRS axis during ADT. There was no difference in location at the tricuspid ring in ADT displaying a normal, horizontal, or leftward axis. The most likely explanation for the differences in QRS axis during ADT is, as already indicated, that in the short V–H ADT following retrograde invasion in the right bundle branch may be followed by activation of the left bundle branch, especially the anterior fascicle, with anterograde conduction over the fascicle. The contribution to ventricular activation over the anterior fascicle results in a fused QRS. The amount of ventricular activation over the anterior fascicle will determine the width of the QRS and the frontal QRS axis. Of interest is the finding of, on average, a shorter V–H interval in the patients with a normal QRS axis, suggesting earlier arrival at the take-off site of the anterior fascicle. Sternick et al. reported QRS configuration changes in patients with ADT over an AF pathway upon the occurrence of retrograde right bundle branch block. Not only slowing in tachycardia rate occurred, because of a larger tachycardia circuit, but there was also a change in QRS axis, which became more leftward, because of lack of fusion from anterograde conduction over the left bundle branch system. This also happened in two patients of our cohort, who had both a short and a long V–H tachycardia during the same electrophysiological study. In both there was a significant change in the frontal plane axis from $-15^\circ$ to $-60^\circ$ (Case 38) and from $15^\circ$ to $-15^\circ$.

Figure 4  Twelve-lead ECG of three different patients with ADT showing normal, horizontal, and superior frontal plane QRS axis ablated from the same sector of the tricuspid annulus (site of ablation between 08:30 and 09:00 o’clock).
Interestingly, Patient 10 had two different AF pathways, one ablated at 08:30 o’clock showing a normal axis (60°), and another ablated close to 07:30 o’clock with 250° QRS axis (Table 2).

The explanation of differences in QRS axis, width, and ventriculo-His interval without a change in tachycardia rate

The differences in QRS axis and width during ADT were the result of the presence and amount of ventricular activation over the left-sided conduction system. A normal QRS axis indicates that an appreciable amount of left ventricular activation occurs by way of anterograde conduction over the anterior fascicle of the left bundle branch, leading to less widening of the QRS complex. As already explained, a short V–H interval will allow that mechanism. Indeed, in our 11 patients with ADT showing a normal QRS axis, the QRS width (127 ± 14 ms), because of a greater amount of ventricular fusion, was significantly narrower than the QRS width of the 15 patients with a superior axis (153 ± 9 ms) as well as the 22 ones with a horizontal axis (138 ± 11 ms) (P < 0.0001). As shown in Table 1, there were no significant differences in tachycardia rate in spite of a shorter V–H interval in patients with ADT and normal axis (4 ± 3 ms) when compared with the longer V–H during AT with a horizontal (16 ± 17 ms) or a superior axis (24 ± 26 ms) (Figure 5). This suggests that in the different components of the tachycardia circuit consisting of atrium-AF fiber-right bundle branch-His-A–V node-atrium, size and conduction velocity compensated for the shorter V–H interval.

Clinical implications

Despite wide axis variability, AF pathways distal end cluster at the lateral and posterolateral aspect of the tricuspid annulus and axis patterns are not helpful in guiding the electrophysiologist. All patients in this large cohort were successfully ablated by mapping the accessory pathway potential at the annulus or sub-annular region. Three-dimensional mapping systems were not required.

Conclusions

There is no correlation between the site of tricuspid annulus location of the AF pathway and the frontal plane QRS axis during ADT. A normal QRS axis during maximal pre-excitation is not related to an anteroseptal tricuspid annulus location. The 12-lead ECG during maximal pre-excitation does not predict the correct site of tricuspid annulus ablation in patients with an AF pathway.

Conflict of interest: none declared.

References


Figure 5 Examples showing the relation between the frontal QRS axis and the V–H interval. A long V–H interval (32 ms) in an ADT with leftward axis (A), and a shorter (4 ms) V–H interval and QRS width in an ADT with a normal axis (B).
Osteomyelitis of the clavicle following to a pacemaker implantation

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Two weeks after dual-chamber pacemaker implantation, a 51-year-old man presented typical signs of pacemaker pocket infection and sharp pain in the left shoulder with functional impotence of the limb. Device and leads were explanted. Pseudomonas aeruginosa was isolated in culture samples obtained from the pocket tissue and both lead tips; blood cultures remained sterile. Specific antibiotic therapy with intravenous ciprofloxacin and cefepime was started.

Transoesophageal echocardiography ruled out intracardiac vegetations. Magnetic resonance imaging (MRI) showed acute osteomyelitis of the left clavicle (Panel A). After discharge, a further MRI revealed a mild reduction of the infected focus; antibiotic therapy was discontinued. Nine months later, a nearly complete resolution of the osteomyelitic focus was observed and the patient regained a full functionality (Panel B). The quick beginning of specific antibiotic therapy, the good susceptibility profile of the microorganism and the absence of comorbid conditions probably contributed to the good prognosis.

The full-length version of this report can be viewed at: http://www.escardio.org/communities/EHRA/publications/ep-case-reports/Documents/Osteomyelitis-of-the-clavicle.pdf.

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