Value of QRS alternation in determining the site of origin of narrow QRS supraventricular tachycardia

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ABSTRACT To determine the value of alternation of QRS morphology in determining the site of origin of sustained narrow QRS supraventricular tachycardia (SVT), we retrospectively studied 163 distinct tachycardias in 161 patients (ages 4 to 91 years) in whom the site of origin of SVT was proven by intracardiac electrophysiologic study. Sustained SVT was defined as lasting longer than 30 sec. Narrow QRS was defined as QRS width less than 0.12 sec. Atrial fibrillation and flutter were excluded. The presence or absence of QRS alternation was judged at least 10 sec after initiation of SVT. Circus movement tachycardia with anterograde AV node conduction and a retrograde accessory AV pathway was seen in 89 patients (58 with Wolff-Parkinson-White syndrome, 31 with concealed accessory pathway); intra-AV nodal reentrant tachycardia (AVNT) was present in 57 cases, and 17 tachycardias were atrial in origin. QRS alternation was present in 36 of 163 cases (22%). In only eight of these 36 did RR interval length alternation accompany alternation in QRS morphology. Thirty-three of 36 (92%) tachycardias with QRS alternation were circus movement tachycardias. Two were atrial in origin and one was AVNT. We conclude that the presence of QRS alternation during sustained narrow QRS SVT is highly indicative of a retrograde accessory AV pathway in the tachycardia circuit.

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ACCURACY in determining the site of origin of supraventricular tachycardia (SVT) with the 12-lead electrocardiogram (ECG) is important for correct treatment of the arrhythmia. Having seen electrical alternans of the QRS complex in patients with SVT, we wondered whether this finding could be of help in determining the site of origin of the tachycardia. Therefore we undertook a study to evaluate the diagnostic value of QRS alternation in patients with sustained SVT and a narrow QRS complex.

Patients and methods

All electrocardiographic tracings of all patients studied for SVT in Maastricht, The Netherlands, between February 1977 and December 1982 were retrospectively analyzed. In all patients the mechanism and site of origin of sustained narrow QRS tachycardia had been ascertained by an intracardiac electrophysiologic study, which included programmed electrical stimulation of the heart. Sustained tachycardia was defined as lasting longer than 30 sec, and narrow QRS was defined as a QRS width of less than 0.12 sec. Atrial fibrillation and atrial flutter were excluded. Our methods of stimulation, recording, and analysis of tracings have been previously described. The site of origin (intra-AV nodal reentrant tachycardia, atrial tachycardia, or orthodromic circus movement tachycardia with anterograde AV node conduction and a retrograde accessory atrioventricular pathway) was determined according to previously defined criteria.

A total of 161 patients met the entry criteria and were included in the study population. There were 71 women and 90 men (mean age 39 years). Two patients had more than one type of tachycardia and as a result there are 163 cases of tachycardia included in the study. One patient had two accessory AV pathways, and the tachycardia with the right-sided retrograde accessory pathway was considered separately from that with the retrograde left-sided pathway. One patient had a tachycardia with a concealed accessory pathway and an atrial tachycardia, and both had been documented clinically. Data on patients and their tachycardias are summarized in table 1.

There were 89 circus movement tachycardias, incorporating a retrograde accessory AV pathway and the AV node in the anterograde direction. Fifty-eight patients had Wolff-Parkinson-White syndrome and 31 had a concealed accessory pathway. Reentry within the AV node was present in 57 cases, and the remaining 17 tachycardias were atrial in origin.

All available leads in all recorded episodes of tachycardia were examined by the same observer for the presence or absence of QRS alternation. To avoid confusion by changes in QRS morphology seen at the initiation of tachycardia, QRS alternation was judged to be present only if it persisted for at least 10 sec and occurred at least 10 sec after the initiation of tachycardia.

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TABLE 1
Data on the patients and their tachycardias

<table>
<thead>
<tr>
<th></th>
<th>Age (yr)</th>
<th>Cycle length (msec)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Mean ± 1SD</td>
</tr>
<tr>
<td>CMT (n = 89)</td>
<td>4-70</td>
<td>34.2 ± 14.2</td>
</tr>
<tr>
<td>AVNT (n = 57)</td>
<td>23-91</td>
<td>48.2 ± 14.8</td>
</tr>
<tr>
<td>AT (n = 17)</td>
<td>5-73</td>
<td>36.5 ± 25.7</td>
</tr>
<tr>
<td>Total (n = 163)</td>
<td>4-91</td>
<td>38.9 ± 17.4</td>
</tr>
</tbody>
</table>

CMT = orthodromic circus movement tachycardia (retrograde accessory pathway); AVNT = intra-AV nodal reentrant tachycardia; AT = atrial tachycardia.

The groups were compared for continuous and discrete variables by Student’s t tests for unpaired data and chi-square tests, respectively.

Results

QRS alternation was found in 36 of 163 tachycardias (22%). Thirty-three of these 36 (92%) were circus movement tachycardias with anterograde AV node conduction and a retrograde accessory AV pathway (figures 1 to 3, table 2).

As shown in table 3, the mean tachycardia cycle length and mean age were significantly lower (p < .001) in the group with QRS alternation. Table 4 shows the incidence of QRS alternation in relation to the tachycardia rate.

Alternation in the length of the RR interval during tachycardia, varying from 10 to 30 msec, was present in only eight of the 36 tachycardias with QRS alternation and in four of the 127 without QRS alternation.

The specificity of QRS alternation for predicting the use of an accessory AV pathway in the circuit was 96% (true negatives × 100/true negatives + false positives). The presence of QRS alternation had a predictive accuracy of 92% for the use of an accessory AV pathway (true positives × 100/true positives + false positives). The sensitivity of QRS alternation for detecting the use of an accessory AV pathway was 37% (true positives × 100/true positives + false negatives).

Because 12-lead ECGs during tachycardia were not available in all patients with QRS alternation, no information could be obtained as to the incidence and degree of QRS alternation in the different extremity and precordial leads.

Discussion

At present, several electrocardiographic criteria have been used for determination of mechanism and

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**FIGURE 1.** Sinus rhythm (A) and tachycardia (B) in a patient with QRS alternation during orthodromic circus movement tachycardia in a concealed, retrograde left-sided accessory AV pathway. The tachycardia cycle length was 300 msec. Note the marked alternation in QRS morphology, especially in lead V5. Surface ECG leads I, II, III, V1, V5, and V6 were recorded simultaneously.
FIGURE 2. QRS alternation during orthodromic circus movement tachycardia in a patient with a concealed left-sided accessory AV pathway. Note the cycle length (CL) alternation from 280 to 300 msec. Surface ECG leads I, II, III, V₁, V₅, and V₆ were recorded simultaneously.

site of origin of SVT. These include analysis of the morphology and position of the P wave in relation to the QRS complex, the AV ratio, and the effect of bundle branch block on intervals during tachycardia. The results of the present study suggest that QRS alternation during tachycardia should also be considered as a useful electrocardiographic criterion and should be sought during narrow QRS tachycardia.

Our findings indicate that the presence of QRS alternation during narrow QRS tachycardia has a specificity of 96% and a predictive accuracy of 92% for the incorporation of an accessory AV pathway in the tachycardia circuit. On the other hand, the absence of QRS alternation was of no value in distinguishing between atrial, AV nodal, and circus movement tachycardias.

The explanation for the observed differences in the presence of QRS alternation in orthodromic circus movement tachycardia, intra-AV nodal reentrant tachycardia, and atrial tachycardia (37%, 2%, and 12%, respectively) is not obvious. The pathways of anterograde ventricular activation are the same in all the narrow QRS tachycardias with anterograde conduction over the AV node, His bundle, and bundle branches. In the absence of differences in input to the His-Purkinje system, the observed differences must be explained by other mechanisms.

One possible explanation is the generally faster tachycardia rates seen in patients with QRS alternation. This could be expected to produce a higher incidence of functional delay somewhere within the conduction system. Although there were significant differences in cycle lengths of tachycardias between the groups with and without QRS alternation during

<table>
<thead>
<tr>
<th>Type of Tachycardia</th>
<th>QRS ALT+</th>
<th>QRS ALT-</th>
</tr>
</thead>
<tbody>
<tr>
<td>CMT (n = 89)</td>
<td>33</td>
<td>56</td>
</tr>
<tr>
<td>AVNT (n = 57)</td>
<td>1</td>
<td>56</td>
</tr>
<tr>
<td>AT (n = 17)</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>Total (n = 163)</td>
<td>36</td>
<td>127</td>
</tr>
</tbody>
</table>

CMT = orthodromic circus movement tachycardia (retrograde accessory pathway); AVNT = intra-AV nodal tachycardia; AT = atrial tachycardia; QRS ALT = QRS alternation.

Statistical comparisons (vs CMT): *p < .01; **p < .05 vs CMT.
tachycardia, there was no significant difference in tachycardia cycle lengths between the AV nodal reentry group and the accessory pathway group when these were analyzed by type of tachycardia (tables 1 and 3). In addition, there was a marked overlap of the ranges of tachycardia rates (tables 1 and 4). Therefore the differences in the presence of QRS alternation cannot be explained by differences in cycle length alone.

Theoretically, alteration in HH intervals might cause functional conduction delay in the bundle branch system during alternate beats, thus causing alternation in QRS morphology. However, alteration in HH interval was observed in only eight of the 36 tachycardias with QRS alternation (figure 2). The majority of our patients with QRS alternation (28/36) showed no such cycle length alternation in either the HH or RR interval (figures 1, 3, and 4).

The relative infrequency of cycle length alternation in our group suggests that alternating differences in timing of input into the conduction system is an unusual cause of QRS alternation. It also suggests that the site of alteration in ventricular activation is located more distally, such as in the bundle branches, the Purkinje system, or even within the ventricular muscle.

**TABLE 3**

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Cycle length (msec)</th>
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<tbody>
<tr>
<td>QRS ALT+ (n = 36)</td>
<td>26.7 ± 13.1</td>
</tr>
<tr>
<td>QRS ALT- (n = 127)</td>
<td>42.4 ± 17.1</td>
</tr>
<tr>
<td>Total (n = 163)</td>
<td>38.9 ± 17.4</td>
</tr>
</tbody>
</table>

QRS ALT = QRS alternation.

**TABLE 4**

<table>
<thead>
<tr>
<th>Tachycardia rate (bpm)</th>
<th>110–150</th>
<th>&gt;150–180</th>
<th>&gt;180–210</th>
<th>&gt;210</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVNT (n = 57)</td>
<td>0/16(^a)</td>
<td>0/22</td>
<td>0/10</td>
<td>1/9</td>
</tr>
<tr>
<td>CMT (n = 89)</td>
<td>2/21</td>
<td>8/27</td>
<td>11/21</td>
<td>12/20</td>
</tr>
<tr>
<td>AT (n = 17)</td>
<td>0/5</td>
<td>1/10</td>
<td>1/2</td>
<td>—</td>
</tr>
<tr>
<td>Total (n = 163)</td>
<td>2/42</td>
<td>9/59</td>
<td>12/33</td>
<td>13/29</td>
</tr>
</tbody>
</table>

AVNT = intra-AV nodal tachycardia; CMT = orthodromic circus movement tachycardia (retrograde accessory AV pathway); AT = atrial tachycardia.

\(^a\)Number of patients with QRS alternation during tachycardia over the total number of patients in each group.
Some of our observations during tachycardia may be pertinent to this issue.

For example, figure 3 shows disappearance of QRS alternation after the development of right bundle branch block and reappearance of alternation after disappearance of right bundle branch block. This suggests that the site of QRS alternation in this case was within the right bundle branch.

Our observations suggest that patients with tachycardias in accessory AV pathways may have anatomically or functionally different conduction systems than those of other patients with SVT and that these differences might cause changes in conduction at faster heart rates. Indeed a tendency toward functional bundle branch block during tachycardia has been previously observed and this may be playing a role in producing QRS alternation in some of our group.2, 9, 10

Unfortunately, the role of changes in heart rate and the effects of drugs on QRS alternation could not be systematically studied in our group. However, we did observe that several patients showed persistence of alternation in QRS morphology when they were re-studied on drugs that slowed the tachycardia rate.

QRS alternation was observed in only one patient with intra-AV nodal tachycardia (figure 4). In this patient the mechanism of QRS alternation may have been caused by changes in superimposition of the P wave and not by change in QRS morphology itself.

In summary, our findings show that the presence of QRS alternation during sustained narrow QRS tachycardia is indicative of an accessory AV pathway in the tachycardia circuit. This might be a helpful clue in determining the site of origin of the tachycardia, even when detected in a single lead such as on a Holter recorder or rhythm strip.

References

![Figure 4](http://circ.ahajournals.org/)

**FIGURE 4.** ECG recordings from the only patient with intra-AV nodal reentrant tachycardia with QS alternation. Atrial and ventricular activation are simultaneous. The HH and HV intervals are constant, although there is 10 msec alternation in AA intervals. There is also alternation in the atrial electrograms (A). In this case, QRS alternation may actually be caused by changes in atrial activation with alternating changes in superimposition of the P wave on the QRS complex. Surface ECG leads I, II, III, V₁, and V₆ were recorded simultaneously with bipolar intracavitary electrograms from the high right atrium (HRA), coronary sinus (CS), and the tricuspid valve in the region of the His bundle (HIS).


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