THE EFFECT OF PACING SITE ON THE ECHO ZONE IN PATIENTS WITH CONCEALED WOLFF-PARKINSON-WHITE SYNDROME

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In 14 patients with the left-sided concealed Wolff-Parkinson-White syndrome, the effects of changing atrial pacing site on the echo zone were evaluated. In 12 patients, re-entrant tachycardia was induced by premature stimuli both in the coronary sinus (CS) and the high right atrium (HRA). In the remaining 2 patients, the tachycardia was induced by premature stimuli only in the CS. The lower limit of the echo zone was shifted to a longer coupling interval during CS pacing in 12 patients. The longer effective refractory period (ERP) of the CS was responsible for the shifting of the lower limit of the echo zone to a longer coupling interval. The upper limit of the echo zone was shifted to a longer coupling interval during CS pacing in 10 patients. The difference of atrial conduction times from the site of stimulation to the 2 conduction pathways (the normal conduction pathway and the accessory pathway) is thought to be responsible for the shifting of the upper limit of the echo zone.

It has been reported that the echo zone shifts with changes in pacing site in patients with Wolff-Parkinson-White syndrome. It is also relatively common experience during an electrophysiological study that re-entrant tachycardia occurs only with atrial stimulation near the site of atrial insertion of the accessory pathway, while pacing from a remote site fails to induce the tachycardia. However, there has been no systematic study as to the effects of changing pacing site on the echo zone in patients with concealed Wolff-Parkinson-White syndrome. Therefore, the purpose of this paper is to evaluate the effects of changing pacing site on the echo zone in patients with concealed Wolff-Parkinson-White syndrome and to clarify the mechanism responsible for the shift.

MATERIALS AND METHODS

Study Patients
Between July 1979 and January 1981, 35 patients without electrocardiographic evidence of ventricular pre-excitation were referred to our electrophysiological laboratory for an evaluation of recurrent episodes of tachycardia. At least one episode of tachycardia was documented electrocardiographically in all patients. Fourteen of these patients were determined to have a left-sided concealed Wolff-Parkinson-White syndrome by electrophysiological studies.

Electrophysiological Studies
A bipolar electrode catheter was passed percutaneously into a femoral vein and advanced across the tricuspid valve for His bundle
Fig. 1. Fourteen cases of the left-sided concealed Wolff-Parkinson-White syndrome. Black bars = echo zones during coronary sinus stimulation; white bars = echo zones during high right atrial stimulation. In Cases 5 and 11, an atrial echo was elicited only during coronary sinus stimulation. In all other cases except Cases 8 and 10, the echo zone was shifted by changing pacing site.

recordings? A quadrupolar electrode catheter was advanced from an antecubital vein into the coronary sinus. The distal 2 electrodes were used for left atrial pacing and the proximal 2 electrodes were used for recording left atrial electrogram. A second quadrupolar electrode catheter was introduced via a femoral vein for high right atrial pacing and recording. Electrocardiographic leads I, II, V1 and V5 as well as intracardiac electrograms were recorded simultaneously on a multichannel recorder (Siemens Mingograph) at a paper speed of 100 mm/sec. Atrial pacing was carried out at the high right atrium and at the site of atrial insertion of the accessory pathway (AP) which was predetermined by endocardial atrial mapping during the tachycardia. Pacing stimuli were provided by a programmable digital stimulator (San-ei Cardiac Stimulator, 3F51) with a strength of approximately twice the diastolic threshold and a duration of 2 msec.

**Definition of Terms**

S1, A1 and V1: The stimulus, atrial electrogram and ventricular electrogram of the basic drive beat.

S2, A2 and V2: The stimulus, atrial electrogram and ventricular electrogram of the induced extra-beat.

Effective refractory period of the A-V node: The longest A1−A2 interval not followed by an H2.

Effective refractory period of the atrium: The longest S1−S2 interval not eliciting an A2.

Upper limit of the echo zone: The longest coupling interval (A1−A2) with which A2 returns to the atrium via the accessory pathway, and initiates an atrial echo (Ae).

Lower limit of the echo zone: The shortest A1−A2 results in an atrial echo.

Echo zone: The difference between the upper limit of the echo zone and the lower limit of the echo zone.

**RESULTS**

**Effect of Changing Pacing Site on the Induction of Tachycardia**

In 12 patients, re-entrant tachycardia was induced by premature stimuli both in the coronary sinus (CS) and the high right atrium (HRA). In the remaining 2 patients (Cases 5 and 11), the re-entrant tachycardia was induced by premature stimuli in the CS, while premature stimuli in the HRA failed to induce the tachycardia (Fig. 1).

**Effect of Changing Pacing Site on the Lower Limit of the Echo Zone**

The lower limit of the echo zone was determined by atrial refractoriness in 11 patients. In these patients, the lower limit of the echo zone was shifted to a longer coupling interval during CS pacing, as the effective refractory period (ERP) of the HRA was shorter than the ERP of the CS (Table I). In the remaining 3 patients (Cases 8, 10 and 12), the ERP of the A-V node determined the lower limit of the echo zone. In these patients, the lower limit of the echo zone remained essentially unchanged regardless of the pacing site (Table I and Fig. 1).

**Effect of Changing Pacing Site on the Upper Limit of the Echo Zone**

In 10 patients (Cases 1–4, 6, 7, 9 and 12–14), the upper limit of the echo zone was shifted to a longer coupling interval with CS pacing (Table I.
Fig. 2. A-V conduction time of premature atrial beats as a function of premature coupling intervals (A1–A2) in Case 1. Circles indicate the A-V conduction times during HRA stimulation. Filled circles indicate atrial premature beats that resulted in atrial echo beats, and empty circles indicate atrial premature beats without echo beats. Triangle indicate A-V conduction times during CS stimulation. Filled triangles indicate atrial premature beats that resulted in atrial echo beats, and empty triangles indicate atrial premature beats without echo beats. Note that the lower limit of the echo zone is shorter during HRA stimulation, while the upper limit of the echo zone shifts to a longer coupling interval during CS stimulation. Line A indicates the minimal A-V conduction time required for the occurrence of atrial echoes during HRA stimulation, while line B indicates the minimal A-V conduction time required for the occurrence of atrial echoes during CS stimulation. Note that the line A determines the upper limit of the echo zone during HRA stimulation, and the line B determines the upper limit of the echo zone during CS stimulation.

CL = cycle length, HRA = high right atrium, CS = coronary sinus

As shown in Fig. 2, the minimal A-V conduction time required for the occurrence of an atrial echo (Ae) determines the upper limit of the echo zone. Therefore, the shifting of the upper limit of the echo zone to a longer coupling interval means that a shorter minimal A-V conduction time is required for the occurrence of Ae during CS pacing. A representative case is shown in Fig. 2, in which the filled circle corresponding to the shortest A-V conduction time represents the minimal A-V conduction time required for the occurrence of Ae during HRA pacing, while the filled triangle corresponding to the shortest A-V conduction time represents the minimal A-V conduction time required for the occurrence of Ae during CS pacing.

As shown in Fig. 3, the impulse arrived earlier at the A-V node than the AP during HRA pacing, while the impulse arrived later at the A-V node than the AP during CS pacing. The difference of the impulse arrival times at the A-V node and the AP during HRA pacing (a and b in Fig. 3) and the difference of the impulse arrival times at the A-V node and the AP during CS pacing (c in Fig. 3) were added in order to calculate the difference of impulse arrival time by changes of the pacing site (Table I). To test if the difference of impulse arrival time is responsible for the shift of the upper limit of the echo zone, it was compared with the difference of the minimal

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### TABLE I DETERMINANTS OF THE ECHO ZONE

<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>CL (ms)</th>
<th>Lower HRA</th>
<th>Limit CS</th>
<th>Determinants lower limits</th>
<th>Upper HRA</th>
<th>Limit CS</th>
<th>Difference minimal AV for Ae</th>
<th>Difference total arrival time</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Y.Y.</td>
<td>39</td>
<td>F</td>
<td>750</td>
<td>190</td>
<td>230</td>
<td>Atrium</td>
<td>300</td>
<td>370</td>
<td>70</td>
<td>70</td>
</tr>
<tr>
<td>2</td>
<td>U.G.</td>
<td>47</td>
<td>M</td>
<td>750</td>
<td>270</td>
<td>280</td>
<td>Atrium</td>
<td>340</td>
<td>400</td>
<td>65</td>
<td>70</td>
</tr>
<tr>
<td>3</td>
<td>H.H.</td>
<td>44</td>
<td>M</td>
<td>500</td>
<td>240</td>
<td>270</td>
<td>Atrium</td>
<td>300</td>
<td>420</td>
<td>80</td>
<td>80</td>
</tr>
<tr>
<td>4</td>
<td>J.K.</td>
<td>46</td>
<td>M</td>
<td>950</td>
<td>180</td>
<td>210</td>
<td>Atrium</td>
<td>300</td>
<td>340</td>
<td>115</td>
<td>130</td>
</tr>
<tr>
<td>5</td>
<td>S.Y.</td>
<td>62</td>
<td>M</td>
<td>700</td>
<td>—</td>
<td>290</td>
<td>Atrium</td>
<td>—</td>
<td>310</td>
<td>—</td>
<td>60</td>
</tr>
<tr>
<td>6</td>
<td>O.K.</td>
<td>54</td>
<td>F</td>
<td>600</td>
<td>180</td>
<td>240</td>
<td>Atrium</td>
<td>330</td>
<td>390</td>
<td>100</td>
<td>110</td>
</tr>
<tr>
<td>7</td>
<td>E.M.</td>
<td>31</td>
<td>M</td>
<td>500</td>
<td>180</td>
<td>220</td>
<td>Atrium</td>
<td>330</td>
<td>370</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>8</td>
<td>Y.E.</td>
<td>58</td>
<td>M</td>
<td>400</td>
<td>250</td>
<td>250</td>
<td>A-V node</td>
<td>290</td>
<td>290</td>
<td>0</td>
<td>70</td>
</tr>
<tr>
<td>9</td>
<td>K.M.</td>
<td>61</td>
<td>F</td>
<td>750</td>
<td>280</td>
<td>290</td>
<td>Atrium</td>
<td>310</td>
<td>340</td>
<td>50</td>
<td>60</td>
</tr>
<tr>
<td>10</td>
<td>T.I.</td>
<td>65</td>
<td>M</td>
<td>750</td>
<td>300</td>
<td>300</td>
<td>A-V node</td>
<td>440</td>
<td>440</td>
<td>0</td>
<td>80</td>
</tr>
<tr>
<td>11</td>
<td>M.T.</td>
<td>24</td>
<td>M</td>
<td>500</td>
<td>—</td>
<td>220</td>
<td>Atrium</td>
<td>—</td>
<td>310</td>
<td>—</td>
<td>80</td>
</tr>
<tr>
<td>12</td>
<td>N.T.</td>
<td>23</td>
<td>M</td>
<td>750</td>
<td>250</td>
<td>250</td>
<td>A-V node</td>
<td>360</td>
<td>380</td>
<td>50</td>
<td>60</td>
</tr>
<tr>
<td>13</td>
<td>E.N.</td>
<td>47</td>
<td>F</td>
<td>500</td>
<td>180</td>
<td>210</td>
<td>Atrium</td>
<td>300</td>
<td>340</td>
<td>65</td>
<td>70</td>
</tr>
<tr>
<td>14</td>
<td>O.M.</td>
<td>32</td>
<td>M</td>
<td>600</td>
<td>190</td>
<td>260</td>
<td>Atrium</td>
<td>270</td>
<td>460</td>
<td>75</td>
<td>70</td>
</tr>
</tbody>
</table>

All measurements are in msec.

Abbreviations: CL = cycle length; HRA = high right atrium pacing; CS = coronary sinus pacing; difference minimal AV for Ae = difference of the minimal A-V conduction time required for the occurrence of an atrial echo by change of pacing site; difference total arrival time = the sum of the arrival time difference at the A-V node and at the accessory pathway during HRA and CS pacing.

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**Stimulation at HRA**

![Stimulation at HRA](image1)

**Stimulation at CS**

![Stimulation at CS](image2)

Fig. 3. During HRA stimulation, the impulse arrives at the A-V node earlier than the AP, while the impulse arrives at the A-V node later than the AP during CS stimulation.

a: Conduction time from the stimulus to the AP during HRA pacing.
b: Conduction time from the stimulus to the A-V node during HRA pacing.
c: Conduction time from the stimulus to the A-V node during CS pacing.

The difference of impulse arrival time at the A-V node and CS by changes of the pacing site can be calculated as \( a - b + c \).

NP = normal pathway, AP = accessory pathway

A-V conduction times required for the occurrence of Ae by changes of the pacing site in each patient, as the minimal A-V conduction time required for the occurrence of Ae determined the upper limit of the echo zone. The difference of impulse arrival time was equal (0 ± 15 msec) to the difference of the minimal A-V conduction times required for the occurrence of Ae by changes of the pacing site. This suggests that the difference of impulse arrival time to the A-V node and the AP by changes of the pacing site was responsible for the shifting of the upper limit of the echo zone in these patients.

In Cases 8 and 10, the shift of the upper limit of the echo zone was not recognized by changes of the pacing site. In these patients, dual A-V nodal pathways were demonstrated and their echo zones were confined to the interval during which A-V nodal conduction occurred through the slow pathway with marked A-V conduction delay.

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**DISCUSSION**

It is well known that re-entrant tachycardia is most easily produced by stimulation near the site of the AP, and not infrequently stimulation near

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the site of the AP initiates re-entrant tachycardia while stimulation at a distant site fails to do so. In the present study, in 2 of the 14 patients with the left-sided concealed WPW syndrome, re-entrant tachycardia was initiated by premature stimuli in the CS while premature stimuli in the HRA failed to initiate the tachycardia.

It has been reported that the range of the echo zone is different according to the pacing site in patients with WPW syndrome. In the present study, the lower limit of the echo zone was shorter during HRA pacing than during CS pacing in 12 patients (Table 1). In these patients, the lower limit of the echo zone was determined by atrial refractoriness and the ERP of the HRA was shorter than the ERP of the CS. In the remaining 3 patients, the lower limit of the echo zone remained unchanged regardless of the pacing site. In these patients, block in the A-V node was responsible for determining the lower limit of echo zone.

As to the shift of the upper limit of the echo zone, Morris et al. have reported that the range of the echo zone in the patient with the concealed left-sided WPW syndrome was wider during stimulation in the CS than during stimulation in the HRA. Likewise, Prichett et al. have attributed the shift of the echo zone in patients with manifest WPW syndrome to the trans-atrial conduction time difference (the difference of the impulse arrival times at 2 pathways by changes of the pacing site). They have noted that the shift of the upper limit of the echo zone by changes of the pacing site is due to the difference of impulse arrival time at 2 pathways by changes of the pacing site. Our present study showed that the mechanism noted by Prichett et al. was also operative in patients with concealed WPW syndrome. As shown in Table 1, the difference of the minimal A-V conduction times required for the occurrence of A-V by changes of the pacing site was equal (0 ± 15 msec) to the difference of the impulse arrival times at 2 pathways. In Cases 8 and 10, the upper limit of the echo zone was equal regardless of the pacing site. This was explained by the fact that both of them had dual A-V nodal pathways and their echo zone were confined to the interval during which A-V nodal conduction occurred through slow pathway with marked A-V conduction delay. Thus, in these patients, the difference of the impulse arrival times was masked by sudden great prolongation of the A-V conduction time.

Our results suggest that stimuli should be given near the site of the atrial insertion of the AP in order to determine proper drugs which narrow or eliminate the echo zone in patients with concealed WPW syndrome.

REFERENCES