Linking Phenomenon in Dual Atrioventricular Nodal Pathways

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The linking phenomenon is an electrophysiological phenomenon of conduction between 2 different pathways, such as bundle branches, atrioventricular node (AVN) and accessory pathways. The present study used electrophysiological studies to investigate this phenomenon in 14 patients with dual AVN pathways. Decremental ramp pacing at intervals of 10 ms was carried out in high right atrium until the atrio-His (A–H) interval was abruptly prolonged (onset), then subsequent incremental ramp pacing at intervals of 10 ms proceeded until the A–H interval abruptly shortened (offset). The linking window (LW) was defined as the difference between the paced cycle lengths of the offset and the onset. The linking phenomenon occurred in 9 patients (64%). The onset depended on the functional refractory period of the fast pathway and once the linking was established in the dual pathways, the LW was related to the difference between the A–H interval immediately before and after the restoration of anterograde fast pathway conduction. These findings suggest that the linking phenomenon in dual AVN pathways occurs because of anterograde conduction block in the fast pathway and the subsequent concealed retrograde conduction of impulses propagated from the slow pathway.

Key Words: Dual atrioventricular nodal pathway; Linking phenomenon

Lehman et al first used the term ‘linking’ to describe an electrophysiological mechanism that perpetuates a functional anterograde block of a conduction pathway by repetitive retrograde concealed penetration of impulses propagating along the contralateral pathway. Recent studies of both the electrophysiological properties of the atrioventricular node (AVN) and radiofrequency catheter ablation treatment of AVN reentrant tachycardia (AVNRT) have indicated the presence of dual AVN pathways; namely, fast and slow pathways that are located at the anterior and posterior portions, respectively, of the compact node near the coronary sinus orifice.

The present study investigated the linking phenomenon in patients with dual AVN pathways and the electrophysiological mechanism is discussed.

Methods

Fourteen patients (7 men, 7 women) with a mean age of 54±19 years (mean±SD; range, 15–72 years) gave written informed consent to participate in the electrophysiological study. Ten patients had AVNRT, one had sick sinus syndrome, one had non-sustained ventricular tachycardia, one had Wenckebach type atrioventricular (AV) block, and one had complete AV block. Other underlying heart diseases included hypertension (2 cases) and angina pectoris (1 case).

The standard baseline electrophysiological study was carried out in all patients. All medications were stopped for a period equal to 5 or more half lives. Details of the technique are described elsewhere. Briefly, a quadripolar electrode catheter (EPT Inc, USA) was introduced percutaneously via the femoral vein and advanced under fluoroscopic guidance to the high right atrium (HRA), where one bipolar electrode catheter each was positioned in the His bundle region and right ventricle (RV) apex. Intracardiac electrograms were recorded using a Mingograph (Nihon-Koden, Inc, Japan) on paper at a rate of 100 mm/s. Programmed stimuli were elicited from the 2 sites using a cardiac stimulator (Nihon-Koden Inc, Japan). The effective refractory period (ERP) and functional refractory period (FRP) of the AVN and other electrophysiological parameters of AVN conduction were measured. The presence of dual AVN pathways was established by the jump up phenomenon; namely, a sudden prolongation of the atrio-His (A–H) interval or ventriculo-atrial (V–A) interval of at least 50 ms for a 10-ms decrement from the HRA or RV apex. We defined the FRP of the fast pathway as the minimum H–H interval before the jump up and the FRP of the slow pathway as the minimum H–H interval after the jump up obtained by extra stimuli from the HRA. To observe the linking phenomenon, decremental ramp pacing were established by slowly decreasing the paced cycle length (PCL) every 10 stimuli by 10-ms steps until the A–H interval was suddenly prolonged. We defined this point as the onset of linking. Incremental pacing was established by increasing the PCL every 10 stimuli by 10-ms steps until the A–H interval was suddenly shortened. We defined this point as the offset of linking (Fig 1). The linking window (LW) was defined as the difference in the PCL between offset and onset.

Results

The clinical and electrophysiological characteristics of
the patients are shown in Table 1. Ten patients had ortho-
dromic AVNRT; the remaining 4 did not, but were revealed
to have dual AVN pathways in the electrophysiological
study. Anterograde linking was evident in 9 of the 14
patients. Fig 1 shows an intracardiac electrogram from
patient 2 at the onset of the linking phenomenon. The A–H
interval was suddenly prolonged from 130 ms to 260 ms
after the paced cycle length shortened from 370 ms to 360 ms.
This is thought to be caused by anterograde conduction of the
fast pathway being interrupted and the ventricles becoming
excited by impulses through the slow pathway. The paced
cycle was gradually lengthened (incremental ramp pacing).
Fig 1B shows an intracardiac electrogram from the same
patient at the offset of the linking. The electrograms shown
in Fig 1 are continuous recordings. Immediately after the
PCL was extended from 460 ms to 470 ms, the A–H interval suddenly
shortened from 220 ms to 130 ms. The A–H interval decreased from 220 ms to 130 ms.

Table 1 Electrophysiological Parameters

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Diagnosis</th>
<th>FERP (ms)</th>
<th>FFRP (ms)</th>
<th>SERP (ms)</th>
<th>SFRP (ms)</th>
<th>PSVT (A–H/H–A)</th>
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<tbody>
<tr>
<td>Linking (+)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>M</td>
<td>71</td>
<td>AVNRT</td>
<td>340</td>
<td>405</td>
<td>&lt;270</td>
<td>440</td>
<td>340 (300/40)</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>32</td>
<td>AVNRT</td>
<td>320</td>
<td>370</td>
<td>&lt;160</td>
<td>440</td>
<td>340 (300/40)</td>
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<tr>
<td>3</td>
<td>F</td>
<td>66</td>
<td>AVNRT</td>
<td>330</td>
<td>370</td>
<td>&lt;210</td>
<td>420</td>
<td>320 (300/20)</td>
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<tr>
<td>4</td>
<td>F</td>
<td>72</td>
<td>SSS</td>
<td>340</td>
<td>380</td>
<td>290</td>
<td>440</td>
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</tr>
<tr>
<td>5</td>
<td>F</td>
<td>52</td>
<td>NSVT</td>
<td>340</td>
<td>415</td>
<td>240</td>
<td>440</td>
<td>ND</td>
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<tr>
<td>6</td>
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<td>300</td>
<td>290</td>
<td>220</td>
<td>370</td>
<td>320 (250/70)</td>
</tr>
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<td>AVNRT</td>
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<td>200</td>
<td>430</td>
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<td>460</td>
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<td>9</td>
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<td>AVNRT</td>
<td>460</td>
<td>525</td>
<td>&lt;250</td>
<td>510</td>
<td>400 (320/80)</td>
</tr>
<tr>
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<td>&lt;240</td>
<td>510</td>
<td>380 (305/75)</td>
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<tr>
<td>12</td>
<td>F</td>
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<td>AVNRT</td>
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<td>330 (260/70)</td>
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<tr>
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<td>15</td>
<td>2AVB</td>
<td>400</td>
<td>480</td>
<td>350</td>
<td>620</td>
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</table>

ERP, effective refractory period; FRP, functional refractory period; FERP, ERP of fast pathway; FFRP, FRP of fast pathway; SERP, ERP of slow pathway; SFRP, FRP of slow pathway; PSVT, paroxysmal supraventricular tachycardia; AVNRT, atrioventricular nodal reentrant tachycardia; SSS, sick sinus syndrome; NSVT, nonsustained ventricular tachycardia; CAVB, complete AV block.
shortened in steps, and at a specific cycle length the A–H interval suddenly prolonged without continuity. Thereafter, as the PCL gradually prolonged, the A–H interval gradually decreased until it suddenly shortened discontinuously at a specific cycle length. The duration of the LW in each patient was 30–110 ms (median, 40 ms).

The PCL at the onset appeared to be similar to the FRP of the fast pathway (Tables 1, 2).

Discussion

The major finding of this study was the observation of the linking phenomenon in 9 patients (64%) with dual AVN pathways. Several investigators have found the linking phenomenon in accessory pathways and macroreentrant circuits.1–3,9 When 2 discrete pathways of conduction exist, impulses in one can influence the conduction properties of the other.

The onset of this phenomenon seemed to be related to the FRP of the fast pathway. Nodal refractoriness can be related to the refractory properties of cells in different regions of the node, and so the FRP of the node may reflect the refractoriness of cells located in the distal portion of the node,10 which is consistent with our results.

Lehman et al described the following mechanisms of linking: (1) persistent retrograde functional conduction delays in the His–Purkinje system during right ventricular pacing, (2) anterograde Kent bundles at rapid rates dependent on a prior block in the normal pathway, (3) persistent anterograde functional infra-His block of atrial impulses during rapid ventricular pacing in the presence of a retrograde conducting accessory pathway, and (4) transient advancement of His activation with ventricular fusion complexes during overdrive ventricular pacing of bundle branch reentrant tachycardia. The linking phenomenon has many variations because of the many types of macroreentrant tachycardia found in humans. We certainly observed this phenomenon in dual AVN pathways.

However, the linking phenomenon was not evident in 5 patients (36%) with the dual AVN pathways for the following possible reasons: the conduction time of the fast and slow pathways did not differ (patients 10 and 11); the conduction time of the slow pathway was so long that reentry initiated immediately after the fast pathway was blocked (patients 12 and 13); or the fast pathway had no retrograde conduction (patient 14). A Wenckebach type AV block occurred immediately after conduction of the fast pathway was blocked because the FRP values of the slow and fast pathways were almost equal (patient 14). Several conduction properties of the fast and slow pathway are required in order to observe the linking phenomenon in dual pathways.

Whether or not the reentry circuit of AVNRT includes atrial tissue has been a matter of conjecture and the present study could not deny the presence of an upper common pathway.

Study Limitations

The number of patients in this study was relatively small. Because changes in autonomic tone may influence AVN conduction during differently paced cycle lengths, an autonomic blockade should be applied to all patients. The linking phenomenon was observed in one patient under autonomic blockade; anterograde conduction was blocked in the fast pathway exactly as in the absence of an autonomic blockade.

Clinical Implications

Diagnosis of AVNRT is usually made when the jump up phenomenon observed with extra stimuli from the right atrium. But detection of anterograde dual AVN pathways is difficult in some patients with typical AVNRT because of the absence of conduction curves with a discontinuity (ie, jump). In the present study, we observed the linking phenomenon in 2 patients with obvious AVNRT whose dual AVN pathways did not show the jump up phenomenon clearly. Therefore, we consider that this phenomenon in the dual AVN pathways might be useful for diagnosing AVNRT in patients with obscure dual pathways.

Conclusions

We observed the linking phenomenon in 9 of 14 patients (64%) with dual AVN pathways. We propose that the anterograde conduction block in the fast pathway, as well as subsequent concealed retrograde conduction of impulses from the slow pathway, causes the linking phenomenon in the dual pathways.

References

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