Ventriculoatrial Shortening Achieved in Wolff-Parkinson-White Syndrome by Programmed Right Ventricular Pacing

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A 47-year-old woman with a left-sided Kent bundle showed marked reduction of the ventriculoatrial conduction time during extrastimuli at the right ventricular apex and outflow tract. The degree of reduction was greater than 6 years ago. A 'supernormal conduction' in the ventricle and the Kent bundle could have been responsible for this phenomenon. (Jpn Circ J 1999; 63: 404 – 406)

Key Words: Supernormal conduction; Ventriculoatrial conduction; Wolff-Parkinson-White syndrome

In programmed ventricular stimulation, the ventriculoatrial (VA) conduction time that passes the Kent bundle usually remains constant! This in turn is used to signify the presence of the Kent bundle based upon electrophysiologic (EP) studies. Multiple accessory pathways should be considered when interpreting cases where the presence of the Kent bundle is known and the VA conduc-
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Case Report

A 47-year-old woman with palpitation as the chief complaint had had an EP study at age 41 and had been diagnosed as a concealed WPW with a left-sided Kent bundle. Because intravenous verapamil was effective in controlling her attacks, 120mg/day orally dose or as a potion was prescribed. In the course of the oral drug therapy, attacks started to occur twice a month in July 1996 and she was admitted to hospital in October 1996 for radiofrequency catheter ablation (RF-CA) therapy. No abnormality was indicated on the general check-up or in the biochemical blood study performed on admission. Informed consent was obtained from the patient and her family for a diagnostic intracardiac EP test and RF-CA.

Fig 1 shows the 12-lead ECG at rest, and during the supraventricular paroxysmal tachycardia induced for the EP study. The heart rate was 68 beats/min at rest, exhibiting no notable abnormality. The heart rate during the tachycardia was 167 beats/min with a narrow QRS complex and the p wave was negative in I and aV1, and positive in V1. The EP test was performed and the left atrium was mapped by a catheter placed through the foramen ovale. An accessory pathway in the left lateral free wall was suggested because the earliest atrial excitation was recorded from the left atrial mapping catheter by ventricular stimulation (Fig 2). As indicated in Fig 2, extrastimulus (S2) at the right apical ventricle exhibited a shorter conduction time from a pacing spike to an atrium potential (SA time) than that in 8 basic cycle beats (S1). Concurrently, the interval between ventricular potential and atrial potential became shorter, suggesting a conduction time reduction in the accessory pathway. Fig 3 shows the relationship of the coupling interval and SA time during right apical ventricular extrastimuli. As the coupling interval shortened, SA time gradually and continuously shortened without drastic changes up to 40ms. However, SA time prolongation was recorded once the coupling intervals reached less than 380 ms. We then tried extrastimuli from the right ventricular outflow, and observed that the SA time shortened compared with the basic cycle beat, just as in apical stimulation. Concurrently, the interval between the ventricular potential and atrial potential shortened, and the retrograde atrial activation pattern was unchanged (Fig 4). The relationship between coupling intervals and SA time in right ventricular outflow stimulation is shown in Fig 5. The SA time shortening pattern was almost same as that in apical stimulation. After changing the basic cycle length (667 to 545ms), the same SA shortening pattern was revealed by programmed right ventricular extrastimuli. But we could not find the SA shortening pattern in the constant ventricular pacing because ventricular atrial conduction block occurred with a pacing rate of 140beats/min.

Because the present patient had had an EP test 6 years ago, 5 ventricular programmed pacings, including her previous test, were compared (Table 1). The SA time at the basic cycle beat in 1996 was slightly shorter during the right ventricular outflow stimulation, but during the apical stimulus the SA time was almost the same as the one recorded in 1990. The effective refractory period (ERP) of the right ventricle remained almost the same, but the ERP...
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of the retrograde Kent bundle was prolonged in 1996. Similarly, maximal SA time shortening was slightly longer in 1996. The coupling interval of extrastimuli that exhibited a 10-ms or longer reduction of SA time moved to the late phase from 1990 to 1996, and right ventricular outflow stimulation exhibited intermediate values.

After the diagnostic EP study, RF-CA was performed by the transatrial septum approach. The Kent bundle was successfully ablated at only 1 site, and atrioventricular conduction was not detected after-wards. Thus it was unlikely that retrograde AV nodal conduction was involved in the SA time shortening during ventricular extrastimuli. The existence of multiple accessory pathways was also unlikely. The Kent bundle in the present patient might have had several components with different conduction times, but in spite of the different stimulation sites (ie, different conduction time or direction) at the right ventricular apical or outflow, SA (also VA and SV) conduction time reduction patterns remained almost the same, and retrograde atrial sequences were also the same. Therefore, it is likely that this phenomenon was caused by slanting, multicomponents of the Kent bundle or shortening of the distance by contraction. It is more likely that these phenomena were attributable to a ‘supernormal excitability-derived supernormal conduction’ mechanism.

Discussion

Induced VA conduction time in tachycardia was constant in the present patient. The atrial excitation sequence during tachycardia was the same as that during ventricular pacing and this indicated that the tachycardia was an atrioventricular reciprocating tachycardia via a left-sided accessory pathway. Using the transatrial septum approach, RF-CA successfully ablated the Kent bundle at only 1 site, and atrioventricular conduction was not detected afterwards. Thus it was unlikely that retrograde AV nodal conduction was involved in the SA time shortening during ventricular extrastimuli. The existence of multiple accessory pathways was also unlikely. The Kent bundle in the present patient might have had several components with different conduction times, but in spite of the different stimulation sites (ie, different conduction time or direction) at the right ventricular apical or outflow, SA (also VA and SV) conduction time reduction patterns remained almost the same, and retrograde atrial sequences were also the same. Therefore, it is likely that this phenomenon was caused by slanting, multicomponents of the Kent bundle or shortening of the distance by contraction. It is more likely that these phenomena were attributable to a ‘supernormal excitability-derived supernormal conduction’ mechanism.
Cinca et al reported 6 cases of left-sided Kent bundle with the SA conduction time shortening phenomenon and these phenomena were eliminated by antiarrhythmic medications. We did not use any medications for catheter ablation, so the effect of drugs on the SA conduction time shortening was unknown in the present patient. They further suggested that the pattern of the reduction in conduction time exhibited a similar pattern to past experimental data in which Spear and Moore showed 'supernormal excitability-derived supernormal conduction' in the canine heart. Spear and Moore also revealed that premature beats evoked during the time when the proximal cell was supernormally excitable were conducted to the distal cell with a shorter conduction time than those beats evoked during diastole. In a clinical setting, supernormal conduction is usually manifested by conduction that is better than expected, but not more rapid than normal. Many studies have shown this type of supernormality, but in some studies, the conduction time reduction, as in the present case, was reported in the human atria and ventricle. Therefore, we considered that in the present case conduction shortening might also occur in the ventricle and Kent bundle. The phenomenon was becoming more prominent with age, suggesting its dependency on some degenerative changes in the muscle fibers.

Although SA time shortening has been reported in previous studies, reduction patterns were not analyzed individually. It is possible that the reduction mechanism varied between cases. To clarify the underlying mechanisms in SA or VA time shortening, further studies are needed.

References


Table 1 Results of Five Series Programmed Right Ventricular Pacings

<table>
<thead>
<tr>
<th>Year</th>
<th>BCL</th>
<th>Pacing site</th>
<th>SA time (ms)</th>
<th>ERP (VA/V) (ms)</th>
<th>Maximal shortening (ms)</th>
<th>Coupling interval (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1990</td>
<td>667</td>
<td>RV apex</td>
<td>200</td>
<td>320/240</td>
<td>40</td>
<td>380–480</td>
</tr>
<tr>
<td></td>
<td>545</td>
<td>RV apex</td>
<td>200</td>
<td>310/220</td>
<td>30</td>
<td>350–440</td>
</tr>
<tr>
<td></td>
<td>545</td>
<td>RV outflow</td>
<td>170</td>
<td>280/NA</td>
<td>20</td>
<td>320–370</td>
</tr>
<tr>
<td></td>
<td>545</td>
<td>RV apex</td>
<td>200</td>
<td>270/220</td>
<td>25</td>
<td>290–360</td>
</tr>
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BCL, basic cycle length; SA, conduction time from pacing spike to local atrial potential; ERP, effective refractory period; NA, not available.