Double Accessory Pathways in Wolff-Parkinson-White Syndrome

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SUMMARY

Intracardiac electrophysiological studies were performed in two patients with Wolff-Parkinson-White (WPW) syndrome. Atrial pacing at increasing rates or shorter coupling intervals produced inscription of the forward His bundle deflection at progressively longer intervals after the onset of ventricular depolarization. There was an associated increase in QRS duration without any change in the P-R (or St-V) interval. This response was consistent with a Kent bundle. Case 1 also had a short A-H interval which did not show the expected prolongation with stimulation at progressively faster rates. This suggested the presence of a James bundle in addition to the Kent bundle. In Case 2 beats conducted exclusively through the atrioventricular (A-V) node had a short H-V interval but a delta wave was not inscribed. Absence of an initial slurring was attributed to the existence of an infra-His bundle bypass of the Mahaim type causing only slight pre-excitation, which was not of sufficient magnitude to be recorded by body surface leads. However, the existence of a congenitally short anterosuperior division of the left bundle could not be excluded.

Additional Indexing Words:
Kent bundle
Reciprocating tachycardias
James bundle
Short H-V interval
Mahaim bundle

Several authors have described the coexistence of two accessory pathways in some patients with Wolff-Parkinson-White (WPW) syndrome. This combination can occur “in parallel” (Kent and James bundle or Kent and Mahaim bundles) as well as “in series” (James and Mahaim bundles). We have studied two cases in which the former associations seemed to have been present. The possible electrophysiological implications of this infrequently found association will be discussed.

Material and Methods

The techniques for obtaining His bundle and other intracardiac electrograms used in our department were discussed previously. The studies were performed after explaining the procedure to the patients and their relatives and obtaining consent. Information from two symptomatic cases of WPW syndrome will be presented.

Case 1

An otherwise normal 64-year-old male had a long history of repetitive tachyarrhythmias. The typical features of WPW type B were noted during sinus rhythm (fig. 1). The P-R interval and QRS complexes measured 90 msec and 140 msec, respectively. Inscription of the forward H deflection almost coincided with the beginning of ventricular depolarization. This finding favored the presence of an electrophysiologically active extra-A-V and extra-His-bundle communication (Kent bundle). In addition, the short A-H interval (45 msec; our lower limits of normal are 50 msec) suggested the coexistence of an electrophysiologically active atrioventricular (A-V) node bypass (James bundle).

When the right atrial lead was stimulated at a rate of 67 beats/min, the forward H deflection appeared 50 msec after the beginning of QRS (fig. 2). The St-V interval was similar to the P-R interval of sinus beats (fig. 1). The shorter (185 msec) and more distorted ventricular complexes suggested that greater parts of the ventricles were activated by the impulse traversing the Kent bundle.

The A-H interval was still short (45 msec). At this moment the low right atrial electrogram recorded by the HBE lead was inscribed simultaneously with the onset of ventricular depolarization. This means that pacing was performed close enough to the atrial end of the Kent bundle to allow the wavefront traversing the accessory pathway to reach the ventricles at more or less the same time as the wavefront reaching the right low atrium (in the vicinity of A-V node) through the atria.

Although there was no further change in the St-V interval as the atrial rate was increased to 135 beats/min, the QRS complexes became slightly wider and more distorted (fig. 3). Apparently the ventricles were now activated almost exclusively through the Kent bundle. Therefore the QRS complexes shown in figures 1 and 2 were “fusion” beats in which ventricular activation occurred through both the Kent and His bundles.

The A-V interval remained at more or less the same value (45 to 50 msec) as the atrial rate was increased (fig. 4). But at a pacing rate of 125 beats/min the H spike was lost within the ventricular deflections of the HBE lead. This need not

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Case 1. Sinus rhythm (rate 55 beats/min) showing WPW type B. Both P-R and A-H intervals were short. In this and all figures paper speed was 100 mm/sec. HRA = Bipolar electrogram from high right atrium; HBE = His bundle electrogram. Values, expressed in msec, are P-A = 40; A-H = 45; H-V = 5; P-R = 90; QRS = 140.

Figure 1

have been due to significant A-H prolongation. For instance, at a rate of 67 beats/min, the H electrogram was already so close to the ventricular deflections that only a minor increase in the A-H intervals would result in its merging with these deflections. Hence the disappearance of the His bundle electrogram at a relatively slow rate does not necessarily rule out a James bundle. However, the increase in A-H interval which did occur might have been due to the fact that the James bundle did not completely bypass the A-V node. Moreover, the possibility of block in the James bundle with conduction through the normal A-V nodal pathway cannot be excluded.

The last QRS complex in figure 3, although similar to the first three ventricular complexes, did not occur in response to the atrial impulse elicited by the fourth stimulus artifact because the latter appeared 30 msec after the onset of ventricular depolarization. Note that the fourth St-LRA and St-HRA intervals had the same duration as the first three. It is possible that the last QRS complex resulted from exclusive, mechanical, stimulation of the Kent bundle.

In summary, the findings in this patient are consistent with the presence of two distinct electrophysiologically active accessory A-V pathways located "in parallel" (Kent and James bundles).

Case 2

A 52-year-old male without other evidence of heart disease had WPW type A and a 26-year history of repetitive supraventricular arrhythmias. Both sinus and driven beats showed P-R and A-H intervals measuring 120 and 70 msec respectively (first beat in fig. 5). A deflection, presumably originating in the His bundle, was inscribed at the onset of ventricular depolarization.

Figure 2

Figure 3

Case 1. Atrial pacing at a rate of 67 beats/min showing an increase in QRS duration and distortion without concomitant prolongation of the St-V (equivalent to the P-R) interval. This response is compatible with the finding of a Kent bundle. In addition, the short A-H interval (of similar duration to that measured during sinus beats, fig. 1) suggests the coexistence of a James bundle, i.e., double accessory pathways located "in parallel." Interval between time markers was 1 sec. St = pacemaker stimulus artifact. St-A = 90; A-H = 45; H-V = H after V; St-V = 90; QRS = 165.

Case 1. Atrial pacing at a rate of 135 beats/min showing what seemed to have been solely Kent bundle conduction. The last QRS complex was not induced by the atrial impulse triggered by the last stimulus artifact. It could have resulted from exclusive mechanical stimulation of the Kent bundle.
Atrial pacing with the extrastimulus method showed that progressively shorter coupling intervals resulted in a gradual increase in QRS duration and distortion (second beat in fig. 5 and second beat in left-sided panel of fig. 6). The inscriptions of the H deflection came progressively later than the beginning of ventricular depolarization, a response in keeping with an electrophysiologically active Kent bundle.

The WPW morphology was not present at a coupling interval of 330 msec (last beat in fig. 6) when the Kent bundle became effectively refractory. In consequence, A-V conduction through the A-V node was considerably delayed. The St-V interval measured 210 msec. The corresponding QRS complex showed the triphasic morphology in lead V1, the pattern characteristic of right bundle branch block. Note that the H-V interval was definitely short (25 msec; lower limits of normal in our department: 35 msec).

A reciprocating tachycardia was induced by the premature stimulus at a coupling interval of 310 msec. In figure 7, the first QRS complex (produced by the driven impulse) showed a WPW type A morphology. The premature impulse, blocked at the Kent bundle, reached the ventricles through the A-V node with a prolonged A-V conduction time and re-entered the Kent bundle in a retrograde direction. It thereafter re-activated the atria to initiate a run of reciprocating tachycardia. During this arrhythmia the H-V interval and QRS duration measured 25 msec and 70 msec respectively. The ventricular complexes showed an electrical axis of close to +90° with a qR pattern in leads II and III. In absence of chronic or acute lung disease, thoracic chest wall deformities, or an extremely vertical anatomical heart position, this morphology resembles the left posterior hemiblock pattern.

The identification of the His bundle origin of this deflection was supported by its relatively large size, occurring in association with a low right atrial electrogram of normal voltage in two HBE leads. Its appearance at the same interval (25 msec) in front of beats with exclusive A-V node conduction irrespective as to whether functional right bundle branch block was or was not present tends to exclude a right bundle branch deflection. The latter would have been inscribed closer to QRS in beats with right bundle branch block. Besides, pacing through the catheter located over the His bundle region resulted in St-V intervals and QRS morphologies similar to the H-V intervals and ventricular complexes of beats conducted exclusively through the A-V node without functional right bundle branch block (fig. 8).

The short H-V intervals seen in beats with exclusive His bundle conduction (regardless of whether right bundle branch block was present or not) can be explained by assuming the existence of a congenitally short anterosuperior division (or fascicle) of the left bundle branch.18 However, theoretically, this morphology can also indicate a Mahaim bundle extending from the beginning of the left bundle to the anterosuperior regions of the left septal or paraseptal surface18 (see Discussion).

To summarize, this patient either had two independent accessory pathways located "in parallel" or a Kent bundle associated with an abnormally short (probably congenital) anterosuperior division (or fascicle) of the left bundle branch.

Discussion

The diagnosis of simultaneous coexisting Kent and James bundles can be made in a patient with WPW syndrome, when, as in case 1, atrial stimulation and His bundle studies are consistent with the presence of an extra-A-V node, extra-His bundle A-V communication (Kent bundle) if, in addition, the A-H interval is short.3,5 The latter is considered the hallmark of an A-V nodal bypass (James bundle).14,15

In patients who have short P-R and A-H intervals, atrial pacing at progressively higher rates does not produce the expected (qualitative or quantitative) increase of the A-H interval.14,17 The duration of the A-H interval may in rare instances remain more or less the same until the effective refractory period of the accessory pathway is encountered.15 In other cases in-
increasing atrial stimulation produces a gradual prolongation of the A-H interval, but while the direction of the change is similar to normal findings, the degree of change is not as great as that found in patients without accessory pathways.\textsuperscript{19} When, as in case 1, the St-V interval stays the same (because of the presence of a Kent bundle) as the cycle length is reduced, it does not take a significant increase in rate for the H deflection to become lost within the ventricular electrogram of the HBE lead (fig. 4).

Case 2 had an electrophysiologically active Kent bundle. In addition, the H-V interval was abnormally short during beats conducted exclusively through the A-V node. Although the His bundle origin of the corresponding deflection could be reasonably proved, the presence of the short H-V interval was more difficult to explain except by the presence of a second accessory pathway. However, the existence of a congenitally short anterosuperior division or fascicle of the left bundle branch could conceivably cause a short H-V. Excitation at the endocardial surface of the anterosuperior left ventricular regions arriving earlier than usual could have produced the left posterior hemiblock pattern seen in figure 7 (vertical, close to

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure6.png}
\caption{Case 2. The second QRS complex in the far left panel shows the maximal degree of QRS duration and distortion observed in this patient (at an St1-St2 interval of 360 msec). Although the St-V interval did not increase, the H deflection was inscribed significantly after the beginning of ventricular depolarization. When the St1-St2 interval was reduced to 330 msec (right), St2 reached the ventricles (after being considerably delayed at the A-V node) with a (functional) right bundle branch block morphology. The corresponding H-V interval measured 25 msec.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure7.png}
\caption{Case 2. Reciprocating tachycardia elicited by a testing stimulus (St2) conducted with a longer St-V interval. During the arrhythmia the narrow ventricular complexes showed a vertical axis with a qR pattern in leads II and III (left posterior hemiblock pattern). The duration of the H-V interval was the same (25 msec) as when right bundle branch block was present (last QRS complex in fig. 5).}
\end{figure}

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+90°, electrical axis and the qR morphology in leads II and III). This phenomenon can be considered the counterpart of the short H-V interval and left anterior hemiblock pattern reported in some patients with incomplete endocardial conduction defects. These findings have been attributed to a congenitally short posteroinferior division (or fascicle) of the left bundle branch.18

According to conventional electrocardiographic theory, postulation of the existence of a Mahaim bundle cannot be used to explain the short H-V because a delta wave was not present. Classically, Mahaim bundle conduction is characterized by the association of a normal or prolonged P-R interval with a delta wave during sinus rhythm or to “WPW deformity when QRS is nodal in origin.”20 Atrial stimulation at increasing rates has been said to increase the A-V conduction time (due to a corresponding lengthening of the A-H interval) without changing either the H-V interval or QRS contour.7

Yet, if a Mahaim bundle extending from the beginning of the left bundle branch to the ventricular septum19 had caused a pre-excitation of only 10 or 15 msec, the latter might not have been of great enough magnitude to produce a definite delta wave in the surface leads, especially if the bypass ended close to the Purkinje network into which the anterosuperior fascicle subdivided.

In fact, Moore has observed that in canine hearts artificially-induced pre-excitation of certain right septal areas can be electrically silent (not recorded) on the body surface leads.31 Although a delta wave was not seen in Moore’s studies, a slight deformity of the narrow QRS complexes occurred when the right septal surface was pre-excited at the time that the supraventricular impulse reached the His bundle.21

These hypotheses need further corroboration. In fact, the exact mechanism of the short H-V in case 2 remains conjectural.

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Figure 8

Case 2. His bundle stimulation showing that the St-V interval and QRS contour were similar to the H-V interval and QRS morphology of beats conducted exclusively through the A-V node (fig. 7).
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