Wolff-Parkinson-White Syndrome

Problems in Evaluation of Multiple Accessory Pathways and Surgical Therapy

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SUMMARY
Total atroioventricular bypass (Kent bundle) does not explain all the findings in some cases of Wolff-Parkinson-White (W-P-W) syndrome. Two cases are reported in which two accessory pathways, set in series or in parallel, could be demonstrated. In case 1, a short P-R interval, gap phenomenon, and presence of delta wave with either a short or long P-R interval suggested the hypothesis of an atrio-His accessory path (James fibers). The presence of a type-B delta wave during His stimulation demonstrated the takeoff of another bypass (Mahaim fibers) below or at the point of stimulation. The His-ventricle accessory path had a supernormal phase of conduction during either atrial or His stimulation. In case 2, the rapid spread of the impulse to the His bundle (P-H 65 msec) was responsible for a normal H-R interval (35 msec) during either reciprocating tachycardia or normal sinus rhythm with a nonwidened QRS and minimal W-P-W deformity (partial cancellation of the delta wave). During reciprocating tachycardia, alternatively short and long P'-H intervals with constant H-R (His bundle) and R-P' (Kent bundle) were due to alternate antegrade atrio-His conduction through the atrio-nodal bypass (James fibers) and the A-V node. Surgical findings confirmed the existence of a left bundle of Kent. The division of the His bundle resulted in a maximal W-P-W deformity (type A) and abolished the reciprocating tachycardia.

Additional Indexing Words:
Kent bundle James fibers Mahaim fibers Antegrade conduction
Retrograde conduction His bundle electrograms His bundle pacing
Reciprocating tachycardia Preexcitation of left ventricle Cancelled delta wave
His bundle division Supernormal conduction in accessory path

His bundle recording is a useful technic for measuring the timing of the impulse through the normal atrio-His axis in the Wolff-Parkinson-White (W-P-W) syndrome. On the other hand, inducing first-degree block into the normal atrioventricular (A-V) path by rapid or premature atrial stimulation is useful for comparing the respective influence of normal and accessory pathways on the shape of QRS complexes in various preexcitation syndromes. Finally, His bundle stimulation allows the investigator to check the hypotheses suggested by the two preceding technics.

Direct A-V bypass (bundle of Kent) is accepted as the underlying mechanism of the W-P-W syndrome. Furthermore the atrio-His bypass (James fibers) is thought to be responsible for the syndrome of short P-R interval with a normal QRS, and His-ventricle bypass (Mahaim fibers) seems to be responsible for the rare form of W-P-W syndrome with normal P-R interval.1 Conceivably, several accessory pathways may be associated, thus

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MULTIPLE ACCESSORY PATHS IN W-P-W

resulting in a W-P-W pattern. Two cases of such an association are described in the present report.

Methods

Two patients were studied, aged 40 (case 1) and 35 years (case 2), respectively, with a clinical history of paroxysmal tachycardia, but free from other cardiac anomalies.

The study was designed to provide recordings of His bundle potentials as described by Sherlag1 in control beats, premature atrial beats, and in runs of atrial tachycardia or reciprocating rhythm. His bundle stimulation was also performed in case 1. Recordings were displayed on a four-channel Elema Mingograph ink-jet writer at paper speeds of 25–100 mm/sec. Both the recording and stimulating catheters were bipolar (no. 6 French) with contacts 10 mm apart. The recording catheter was introduced percutaneously via the femoral vein, and the atrial-stimulating catheter introduced via an arm vein. Electrically induced premature atrial beats were introduced by means of an R-wave coupled-pulse generator (Cardioralentisseur Serdan no. 101). Stimuli were 2 ma in strength and 2 msec in duration.

In case 2, during the operation, bipolar potentials were recorded from the epicardial surfaces of both ventricles by a roving electrode. The interelectrode distance was 1 mm. The peak of monophasic potentials or the intrinsic deflection of biphasic tracings was identified as local activation time. The signals were recorded simultaneously with leads I and V1, and unipolar right atrial ECG.

Cases Studied

Case 1

Figure 1 A shows basic tracings during normal sinus rhythm. The rate is spontaneously variable. The W-P-W deformity (B type) is present in the second and fourth complexes. The others are normal in shape and duration, but the P-R interval is short. Measurements of the different intervals are

![Figure 1](http://circ.ahajournals.org/)

**Figure 1**

*Case 1: Basic tracings. (A) Intermittent W-P-W syndrome (B type) during normal sinus rhythm with varying cardiac cycle lengths. (B) His bundle electrograms (HBE) with narrow (first beat) and wide (second beat) QRS. Numbers indicate time intervals in 0.01 sec. See the text for explanation.*

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Figure 2

Case 1: Atrial and His bundle stimulation. (A) Atrial stimulation at increasing rate. (B) His bundle stimulation at constant rate. Abbreviations: Ps = stimulated P wave; Hs = stimulation of the bundle of His. See the text for explanation.

visualized in figure 1 B, where the first R wave is normal while the second is slurred and wide. In the first beat, the borderline normal P-R interval (120 msec) is in fact a borderline normal P-H interval (80 msec) while the H-R interval is quite normal (40 msec). In the second beat, the presence of a delta wave is responsible for a shorter P-R (90 msec), but the P-H remains constant.

In all spontaneous tracings recorded in this patient, the presence of the W-P-W deformity obviously depended on the cardiac rate. This fact is evident in figure 2 A where the atrial stimulation rate is progressively increased. As long as the cardiac cycle length is more than 560 msec the R waves remain narrow. The W-P-W syndrome appears (2:1) as the cardiac cycle is reduced. When it becomes less than 520 msec, wide R waves disappear again. It is likely that a supernormal period for the delta wave is present.

In figure 2 B, His stimulation is performed, the W-P-W deformity is present, and the supernormal period for delta wave is present, as in figure 2 A. Therefore, beats 2, 4, and 8 show that the W-P-W deformity as the cycle length is, respectively, 560, 540, and 520 msec. Beat 10 is normal because the preceding cycle is slightly shorter. Beats 5, 6, 11, and 12 are also narrow because the preceding cycle is clearly longer, as for beats 1, 3, 7, and 9 of sinus origin.

Figures 1 and 2 suggest the existence of two accessory pathways set serially. There is a bypass between the His bundle and the ventricle. Slurred and wide QRS during His stimulation can only be explained by the takeoff of the accessory path below the atrium, i.e. in the A-V node or the His bundle. Examination of the tracings permits exclusion of two differential diagnoses: (1) stimulation of both His bundle and atrium, since the interval between stimulation and wide QRSs is too short, and (2) stimulation of both His bundle and adjacent ventricular myocardium since the W-P-W deformity is exactly the same in the two panels of figure 2, and His
stimulation outside of the supernormal period never produces a wide QRS.

The presence of an isolated His-ventricular bypass should be responsible for the classical pattern of W-P-W syndrome with a normal P-R interval. On the contrary, in the present case, the P-R interval is 90 msec. This fact lends support to the hypothesis of another accessory path bypassing the A-V node itself. The values of 120 msec for P-R (without delta wave) and 80 msec for P-H may be either the lower values for normal A-V conduction or the higher values for conduction in the syndrome of short P-R interval with normal QRS.

Figures 3 and 4 support the preceding hypothesis. Figure 3 A shows basic complexes with or without a delta wave, to be compared with complexes recorded in strips B to J. Diagrams above tracings represent the A-V nodal axis and the two accessory pathways, either blocked or involved, as discussed below. Electrically induced atrial premature beats at progressively shorter coupling intervals produce a variety of results.

In figure 3B, the atrial stimulation is late and followed by a normal QRS. (It must be noted that the St-R interval [160 msec] is longer than the basic P-R interval [120 msec] by 40 msec, which represents a constant latency time between the stimulation and the resulting atrial premature beat. Thus, the “normalization” of the P-R interval is only apparent and must not be taken into account, but the comparison between the various tracings remains of value.)

In figure 3C, the atrial stimulation is slightly earlier: the P-R interval is short, and the W-P-W deformity is present. In D, there is a short P-R interval followed by a narrow QRS, slightly different in shape (but not in

**Figure 3**

Case 1: Effects of progressively earlier premature atrial stimulation. (A) Basic tracing. (B–J)
See the text and figure 4 for explanation. Diagrams above tracings represent the A-V nodal-His axis and the two accessory pathways. Abbreviation: St = stimulation.
duration) from basic complexes (left-axis shift). In E, the P-R interval is short, and the QRS is slightly prolonged (100 msec) with an incomplete right bundle-branch block (RBBB) pattern (rSr' in V1). In F, the P-R is short, and the QRS is clearly prolonged (120 msec) with a complete RBBB pattern. In G, there is a “gap phenomenon”: with this precise timing the stimulated P wave is blocked, while it is conducted when it is either earlier or later. In H, the A-V conduction is resumed with a prolonged P-R interval (280 msec), and again an incomplete RBBB is present. In I, the P-R interval is long, with a complete RBBB pattern. In J, after a very premature P wave, the P-R interval is very long (320 msec) and the QRS later than in I; the W-P-W deformity reappears, as in C.

Figure 4 shows the duration of the different intervals and R waves recorded in figure 3. The P-R interval depends on the timing of the stimulated P waves, and the shape and duration of R waves depend on the timing of R. These timings are measured with respect to the sinus P waves of the preceding beat (zero time in abscissa). The lack of His bundle recordings for technical reasons at this time of study does not disturb the interpretation of the tracings.

The shape of R waves depends on both intraventricular conduction and ventricular preexcitation. Intraventricular conduction is normal in B and borderline normal in C. An incomplete RBBB is present when the timing of R is slightly above 600 msec (610 in E and 620 in H), with either a short or long P-R interval. A complete RBBB is present when the timing of R is slightly under 600 msec (580 in F and 560 in I), with either a short or long P-R interval. Strips C and J strongly support the hypothesis of a His-ventricle accessory path, since the W-P-W deformity is observed with either a short or long P-R interval. The fact that in both strips the timing of QRS is exactly the same (600 msec) stresses the existence of a supernormal state for conduction through this bypass, and the timing of this supernormal phase in figure 3 fits with that observed in the two panels of figure 2. Finally, there is a possibility of a “canceled” RBBB in strips C and J, since the timing of the QRS is intermediate between E and F for C, and between H and I for J. However, the B-type W-P-W syndrome, with
right ventricular preexcitation, may mask the RBBB. The existence of a dual atrio-His conduction system may explain the A-V conduction pattern, if the effective refractory period in the A-V node is assumed to be shorter than in the accessory path. In strips B through F, it can be assumed that the atrial impulse involves the atrio-His bypass. The duration of the P-R interval is borderline normal and short, but the most important fact is that it is constant, a characteristic feature of the accessory paths. In strips H through J, the impulse is assumed to involve the A-V node itself. The P-R interval is long and increases as the P wave appears earlier.

Then, the underlying mechanism of the gap phenomenon as seen in strip G of figure 3 might be the nodal retrograde concealed conduction of the impulse having reached the His bundle via the still responsive accessory path. It has been shown that in such cases the P-H interval remains short, and the impulse is blocked distal to the His bundle. A functional left BBB then probably occurs, in addition to the right BBB, leading to a bilateral BBB, as shown on the diagram.

In this patient, reciprocating tachycardia could be initiated and terminated by electrically induced atrial premature beats. The narrowness of the QRS complexes during the tachycardia was not surprising, since the cardiac rate was too rapid and explained by itself the failure of W-P-W deformity. A circus movement probably involved the A-V node antegrade and the atrio-His bypass retrograde (P'-R interval longer than R-P') as suggested in Durrer's study. A circus movement between the bundle of His and the His-ventricle bypass could be ruled out because of the insufficient responsiveness of the latter.

Figure 5

Case 2: Basic tracings. Top tracings during sinus rhythm. The third beat in each lead is an electrically induced atrial premature beat. Bottom tracings show supraventricular tachycardia.
In short, the existence of an His-ventricular accessory pathway is accurately supported by the presence of the delta wave with either a short or long P-R interval and the results of the His bundle stimulation, with a supernormal period of conduction. On the other hand, the existence of an atrio-His accessory path is possible, but the disposition "in series" of two accessory paths is not absolutely proved.

Case 2
Basic tracings are presented in figure 5. In the top tracings, during sinus rhythm, the P-R interval is short. The QRS complexes are narrow, and normal in appearance, except for the tall R wave in V1. In each lead the third beat is an electrically induced atrial premature beat. The stimulated P wave is followed by a short P-R interval, and the QRS is slurred and wide, with a maximal W-P-W deformity (A type). Thus, the tall R wave in V1 during sinus rhythm reflects, in fact, a small delta wave. In addition, bottom tracings show a supraventricular tachycardia observed 10–40 times a day in this patient. The QRS complexes differ from sinus beats by left-axis shift in standard leads, and normal pattern of R in V1. Thus, one of the three usual criteria for W-P-W syndrome (i.e. wide QRS) is not present in this case.

The different conduction times can be studied more precisely in figures 6 and 7, in leads I, III, V1, and His bundle electrograms. Besides the usual landmarks (P, H, R, J), the S wave in V1 has been taken into account because it allows for the relative variations between initial and terminal parts of the ventricular depolarization.

Figure 6 shows the effect of a premature atrial stimulation on the P-H and P-R intervals and on shape and duration of the QRS. The duration of the different intervals is depicted

Figure 6
Case 2: Effects of progressively earlier premature atrial stimulations on H-R interval and delta wave. See the text and figure 7 for explanation.
on figure 7. Electrically induced atrial premature beats (labeled Ps) at varying coupling intervals (strips A through H) produce a variety of results.

The P-R interval remains constant. In fact, it is a Ps-delta interval, and the latency time between the stimulation and the resulting atrial beat explains its apparent prolongation to 160 msec instead of 100 msec for basic sinus beats. So, the comparison between the different strips remains of value, and the constancy of the P-R interval supports the concept that there is a direct A-V bypass responsible for the delta wave.

The P-H interval is progressively prolonged, so that His deflections move further into the QRS. The P-H interval is short during sinus rhythm (65 msec), which suggests the probability of a bypass of the A-V node, but its progressive lengthening strongly suggests that the accessory pathway is atrio-nodal rather than atrio-His. Therefore, a first-degree block in the lower part of the A-V node is easily conceived.

S wave and J point are progressively delayed in the same proportion as H wave so that, as the onset of R (delta) does not move, the QRS duration increases progressively from 75 to 155 msec. The lengthening of the QRS concerns its initial part (R-S) and not the terminal one (S-J).

As the exaggerated W-P-W deformity is associated with the prolongation of the P-H interval, it seems obvious that the QRSs are fusion complexes resulting from activation in parallel of the ventricle via the two pathways: direct A-V accessory path and atrio-nodal accessory path. The latter corrects more or less the two effects of the former on the QRS, delta deformity and widening.

The initiation of paroxysmal supraventricular tachycardia could easily be obtained in this patient by either atrial or ventricular stimulation. When two atrial beats are induced in quick succession (see fig. 9 A), the first one is followed after a short P-R interval by a QRS with a maximal W-P-W deformity. The second one is followed after a long P-R interval by a normalized QRS indicating the block of the direct A-V accessory bundle. Therefore, the latter is now able to conduct retrogradely the impulse with a short R-P' interval: reciprocal beating of the atrium which initiates the reciprocating tachycardia. In the same way, a single spontaneous ventricular premature beat was able in this patient to initiate the tachycardia, explaining the frequent recurrence of the episodes.

Figure 8 shows an unusual phenomenon during tachycardia. Beats 1 to 6 are regular (cycle length, 310 msec), then short pauses occur before beats 7, 9, and 11 (400 msec)
Figure 8

Case 2: Short and long cardiac cycles during tachycardia. R-P' and H-R intervals remain constant. P-H interval is alternatively short (James fibers) and long (A-V node) from beats 6 to 11. Abbreviation: P' = retrograde P wave. See the text for explanation.

while the R-R interval is unchanged for beats 8 and 10. During these alternatively short and long cardiac cycles the retrograde conduction times (R-P') remain unchanged (120 msec), and the His-ventricle conduction time (H-R) is constant (35 msec). P'-H is the only changing interval (155 and 245 msec alternatively). This finding strongly suggests that the spread of the impulse from the atrium to the His bundle involves either the A-V node (long P'-H) or the atrio-nodal accessory path (short P'-H), while the retrograde impulse involves the direct A-V accessory path.

The termination of tachycardia could be obtained by either atrial or ventricular stimulation, as commonly observed in reciprocating rhythms. It should be noted that either atrial or ventricular captures are easily induced, and this fact favors a circus movement involving atrial and ventricular myocardium themselves,7 without initial and final common pathways.8

Because of the ineffectiveness of drug treatment, the surgical therapy of tachycardia was decided. During the operation, bipolar potentials were recorded from the epicardial surfaces of the right and left ventricles. In this study, particular attention was focused on the posterior wall of the left ventricle, along the atrio-ventricular margin, between the interventricular septum and the left border of the heart. The epicardial surface of the left ventricle adjacent to the interventricular septum was activated 200 msec after the onset of P. The timing of the intrinsic deflection decreased progressively when the electrode was shifted to the left. The earliest region of ventricular activity was found to be located 2 cm from the interventricular septum, 5 mm below the posterior atrioventricular margin. The activation spread radially from this focal point.

In figure 9 A the intrinsic deflection in this area occurs 100 msec after the P wave, before the onset of the QRS in lead I and V1, and does not move when a premature atrial stimulation (beat 3) exaggerates the W-P-W deformity. On the other hand, after a second stimulated P wave (beat 4), the W-P-W syndrome disappears, and the reciprocating tachycardia is induced after a long P-R interval. When the ventricles are activated via
the His bundle alone, the intrinsic deflection in the same area is late (beats 4, 5, and 6).

Figure 9 B demonstrates the transition to the normal excitation which occurred when digital pressure was applied to the site of earliest ventricular activity. This procedure was repeated with similar results several times. Pressure applied to the adjacent regions along the posterior atroventricular margin failed to abolish the preexcitation. This observation confirms the focal location of the preexciting pathway determined electrophysiologically. However, no evidence was found of “preventricular spike” traducing the electrical activity of the bypass tract itself as in the case of Boineau and Moore.9

In addition, when the ventricular activation is “normalized” by digital pressure (fig. 9 B), the intrinsic deflection is not only late in the previous preexcitation area, as it is during tachycardia, but also the initiation of tachycardia by two premature atrial stimulations (beats 3 and 4) is no longer possible.

In spite of these facts strongly suggesting the presence of a direct A-V accessory path, its surgical division was not attempted. The risk of venous or arterial coronary or left ventricular or even mitral valve injury was too high.

Thus, the ligature of the His bundle was performed after right heart bypass. The resulting ECG (fig. 10 A) is a maximal W-P-W deformity (A type) during sinus rhythm. The exaggeration of the delta wave and the
initiation of tachycardia by premature atrial stimulation are observed no more. In figure 10 B after infusion of ajmalin (50 mg), which is known to interrupt the conduction within the preexcitation path, the fourth P wave is blocked while the fifth is normally conducted (Mobitz type II second-degree A-V block), after which there is a complete A-V block. Then the escape beats are narrow.

The His bundle electrograms are recorded during either sinus rhythm or A-V block. During sinus rhythm (fig. 10 C) a diphasic (+ -) retrograde H’ wave occurs just after the end of QRS, while during A-V block (fig. 10 D) a diphasic (− +) antegrade H wave occurs 40 msec before narrow escape beats.

The follow-up of the patient is now 6 months, and no recurrent tachycardia has ever been observed.

In short, numerous facts support the hypothesis that three pathways of conduction do exist in this patient: A-V nodal-His axis, atrio-nodal accessory path, and direct A-V bypass. During sinus rhythm the rapid spread of the impulse through both direct A-V bypass and atrio-nodal bypass, and His bundle and its branches, results in a partly canceled ventricular preexcitation, so that the delta wave is small, and the QRS complex is narrow. During reciprocating rhythm, circus movement involves: either A-V node or atrio-nodal accessory path to His bundle and its branches to ventricular myocardium to direct A-V accessory path retrogradely and finally to atrial myocardium. After division of the bundle of
His, the W-P-W syndrome becomes obvious, and the epicardial mapping is in favor of the existence of a bundle of Kent between the left atrium and the left ventricle.

Discussion

The purpose of the present study is a functional rather than anatomic evaluation of multiple accessory pathways bypassing partly or entirely the normal A-V nodal-His axis. Since anatomic\textsuperscript{11} and electrophysiologic\textsuperscript{12, 13} data are not easily compared, the semantic question remains whether or not the terms "bundle of Kent," "James fibers," and "Mahaim fibers" are to be used to indicate preexciting pathways. On clinical grounds, the evaluation of the functional setting of the different accessory pathways requires a careful study of the various intervals and their changes during atrial stimulation and reciprocating rhythm.

Direct A-V Bypass

For the existence of a bundle of Kent, the following criteria are needed\textsuperscript{14–16} (table 1): (1) His potentials usually following the onset of QRS, within delta wave, because of the normal value of the P-H interval (more than 80 msec); (2) slurred and wide QRS, with exaggeration of the W-P-W deformity when a first-degree block in the normal A-V nodal-His axis is produced by rapid or premature atrial stimulation with lengthening of the P-H interval, so that QRS appears to be a fusion complex; and (3) particular features of reciprocating rhythm when present, i.e. constant coincidence of normalized QRS with initiation of the tachycardia, long anterograde conduction time contrasting with short retrograde conduction time and the possibility of eliciting atrial or ventricular captures by a single ventricular or atrial stimulation (different from intranodal reciprocating tachycardia where initial and final common pathway "filter" the impulses).\textsuperscript{8, 17} In exceptional cases\textsuperscript{7, 18} where antegrade conduction involves the bypass, a maximal W-P-W deformity is present, and R-P' interval is longer than P'-R.

The lack of these three criteria is complete in case 1, so that the hypothesis of a direct A-V bypass can be accurately ruled out. In case 2, criteria 2 and 3 are present; the lack of criterion 1 does not eliminate the existence of a bundle of Kent, and suggests that two accessory pathways are present instead of one.

In case 2, the sequence of epicardial activation, with a left ventricular preexcitation, differs from the findings of Boineau and Moore\textsuperscript{9} in a dog with A-type W-P-W syndrome. This location makes difficult and dangerous the surgical division of left bundle of Kent, not reported at the present time. The division of the His bundle is a more accurate technic.\textsuperscript{19}

### Table 1

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Abbreviations: NEB = nodal escape beats; HS = His stimulation; PAS = premature atrial stimulation; L-G-L syndrome = Lown-Ganong-Levine syndrome.

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Atrio-Nodal or Atrio-His Bypass

The syndrome of a short P-R interval with a normal QRS is due to the bypass of the normal conducting system up to the His bundle. No agreement has been reached as to whether the accessory path is intranodal or extranodal (posterior internodal pathway of James).11 His bundle recordings21, 22 show that the H-R interval always remains normal, and that the shortening of P-R is in fact the shortening of P-H. If the maximal value of P-R in this syndrome is admitted to be 120 msec,23 the maximal value of P-H would be about 80 msec and should be considered as "short." Furthermore, this characteristic is not the only one observed in this syndrome:21 rapid or premature atrial stimulation results in either progressive (atrio-nodal bypass) or sudden (atrio-His bypass) lengthening of the P-H interval. The gap phenomenon is often observed, more frequently than in normal patients,6 thus suggesting a dual atrio-His conduction system, even though such explanation is not exclusive.

Thus, James fibers seem to be present in both cases. In case 1, it may explain His potentials preceding the delta wave, the shortness of P-H with either normal or slurred and wide QRS during sinus rhythm, and the sudden prolongation of the P-R interval with a gap phenomenon when atrial premature stimulation is earlier.

In case 2, the existence of James fibers is suggested by the shortness of the P-H, and explains the partial cancellation of the delta wave and the narrowness of the QRS during sinus rhythm. When premature atrial stimulation progressively lengthens and "normalizes" the P-H interval, all criteria for the presence of a bundle of Kent are fulfilled. The maintained A-V conduction after division of the bundle of His confirms the existence of a direct A-V accessory path. Three similar cases have been reported previously,24 and the hypothesis of two accessory pathways "in parallel" is also admitted by Massumi16 for two of his cases. In fact, the simultaneous mapping of both left atrium and ventricle would be the only way to support accurately the existence of a left bundle of Kent; unfortunately, this technic cannot be used in the human heart in situ. Thus, another hypothesis cannot be accurately ruled out: an extension of the James fibers terminating in the basal posterior segment of the left ventricle, so realizing a functional direct A-V accessory path, but not an anatomic one, clearly separated from the normal nodal-His axis.

His-Ventricle Bypass

Whether or not the P-R interval is long, the only criteria needed by Pick and Katz25 for the presence of Mahaim fibers is the existence of W-P-W deformity when QRS is nodal in origin. When A-V nodal escape beats are not present, the stimulation of the His bundle is a useful technic to demonstrate this form of conduction, as in case 1. In addition, the presence of a delta wave with either a short or long P-R interval fits in with the hypothesis of the takeoff of Mahaim fibers in the A-V node or the bundle of His. Finally, the lack of exaggerated W-P-W deformity by atrial stimulation is easily explained, since the all-or-none phenomenon is commonly observed in both the His bundle and the anomalous pathways. This fact does not mean that QRS is not a fusion complex in this case. On the contrary, the fusion of impulses can explain the cancellation of the RBBB in figure 3 J.5, 4

In our case, a delta wave follows the H wave during sinus rhythm, but disappears during supraventricular tachycardia, in contrast to the case reported by Castillo and Castellanos.26 Lev et al.20 also reported the association "in series" of two accessory pathways, with anatomic confirmation. Furthermore, in our case, the 2:1 conduction in Mahaim fibers might be explained by a very long refractory period in the accessory path, followed by a supernormal state of conduction.27

Our goal is to emphasize the importance of a precise evaluation of the functional disposition of accessory pathways when the surgical treatment of paroxysmal supraventricular
tachycardia is considered.\textsuperscript{19, 24, 28, 29} The co-existence of W-P-W syndrome with paroxysmal tachycardia is not sufficient to conclude that a Kent bundle exists. The type of W-P-W syndrome is not helpful since we have encountered, among 21 cases studied,\textsuperscript{14} "Kent bundle" alone, or "Kent bundle plus James fibers," or "James fibers plus Mahaim fibers" disposition (table 1) in any type of W-P-W syndrome. In the reverse, the evaluation of the "functional" disposition of accessory pathways does not allow precise anatomic conclusions, except for cases in which epicardial potentials or even the accessory path itself\textsuperscript{9, 16} have been recorded directly.

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