Patterns of Ventriculo-Atrial Conduction in the Wolff-Parkinson-White Syndrome

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SUMMARY
In 36 patients with the Wolff-Parkinson-White syndrome, ventriculo-atrial (VA) conduction was studied using the single test stimulus method. In 22 patients no significant change in VA conduction time occurred following test stimuli with increasing prematurity suggesting exclusive VA conduction by way of the accessory pathway, an accessory pathway with a shorter refractory period than the His-AV node pathway, or identical refractory periods in both pathways. In 14 patients showing this VA conduction pattern tachycardias could be initiated by a single early ventricular premature beat. This finding lends credence to the hypothesis that the accessory pathway is an essential link in the tachycardia circuit. In two of the five patients showing exclusive VA conduction over the His-AV node pathway single re-entrant beats with antegrade conduction over the accessory pathway could be elicited by appropriately timed ventricular premature beats. In one patient showing this pattern a sustained tachycardia with AV conduction over the accessory pathway could be initiated. A total of eight patients did not show VA conduction over the accessory pathway.

Additional Indexing Words:
Single test stimulus method Accessory pathway His-AV nodal pathway
Re-entry Tachycardia

In patients with the Wolff-Parkinson-White syndrome (WPW) epicardial excitation mapping,1–3 electrical stimulation of the heart,4 His bundle recordings5 and results of surgical interventions6,7 point to the presence of two conduction pathways between atrium and ventricle. Atrial pacing using both the single test stimulus method and driving of the atrium at increasing frequencies have demonstrated that 1) usually both pathways conduct in the atroventricular direction, 2) the electrophysiological properties of both pathways differ, and 3) because of these differences circus movement tachycardias using both pathways can frequently be initiated in the patients. Less is known about the ability of the two pathways to conduct an impulse from the ventricle to atrium.

Following an induced ventricular impulse one can theoretically expect four possible patterns of ventriculo-atrial (VA) conduction (fig. 1): 1) absence of VA conduction (both the accessory pathway [AP] and the His-AV nodal pathway [H-AV P] being blocked) (fig. 1A); 2) conduction via the AP only, (fig. 1B); 3) conduction both by the AP and the H-AV P (fig. 1C), with either the AP having the shortest refractory period (C1) or the H-AV P having the shortest refractory period (C2), or with identical refractory periods for both pathways. In the latter situation, because of the shorter time interval required for passage of the impulse through the AP, it will be impossible to distinguish this pattern from the one shown in C1; 4) conduction via the H-AV P only (fig. 1D).

In this article we would like to present results on VA conduction in patients with the WPW syndrome as studied by the single test stimulus method during right ventricular pacing.

Material and Methods
Thirty-six patients were studied. All fulfilled the classical criteria of the WPW syndrome: a delta wave, a P-delta interval of 0.12 sec or less, and a QRS width of 0.12 sec or more. According to Rosenbaum’s classification,8 26 patients showed a type A and 10 patients a type B WPW pattern. There were 17 males and 19 females ranging in age from 18 to 58 years. Twenty-four of them had had at least one electrocardiographically documented episode of tachycardia. In all patients both right atrial and right ventricular stimulation was performed using the single test stimulus method and

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VENTRICULO-ATRIAL CONDUCTION IN WPW

Figure 1

Possible patterns of VA conduction in the WPW syndrome. K = accessory pathway, H = His-AV nodal pathway.

regular pacing at increasing frequencies. For right ventricular stimulation the catheter was positioned in the apex. The stimulator, pattern of stimulation and characteristics of the stimulus given have been previously described. Simultaneous recordings were made of ECG leads I, II, III, V₁, and V₆, at least one uni- or bipolar right atrial lead, and a His bundle lead. For recording the electrical activity of the His bundle we used an Elema EMT 12. All data were stored on tape with help of an Ampex FR 1300. Only the data obtained by the single test stimulus method during right ventricular pacing will be presented. Using this method, pacing was continued until the effective refractory period of the right ventricle was reached (the shortest test stimulus interval resulting in ventricular activation). The shortest test stimulus interval followed by ventriculo-atrial conduction was considered the effective refractory period of the VA conduction system. Arguments as to the pathway used during VA conduction are discussed under results.

Results

No VA Conduction

Three patients, each of whom showed AV conduction during atrial pacing both by the AP and the AV-HP, did not have VA conduction during ventricular pacing. None of these patients (whose ages were 18, 22, and 42 years) had a history of tachycardia and no tachycardia could be elicited during atrial or ventricular pacing.

VA Conduction Via the AP Only

In two patients we could be certain that the AP was the only pathway because the AV-HP had been surgically dissected for treatment of tachycardias that could not be controlled by drug therapy. Both patients suffered from circus movement tachycardias in the presence of WPW type A and will be described elsewhere.

During ventricular pacing no increase in VA conduction time was seen following premature beats up to very short premature beat intervals. Very early premature beats elicited in a 30 msec interval range immediately following the effective refractory period of the ventricle showed a gradual increase in VA conduction time, which could be
attributed to: 1) an increase in latency time (the interval between the pacing stimulus and the beginning of subsequent ventricular activation); 2) widening of the QRS complex due to slowing of interventricular conduction at very short premature beat intervals; 3) slowing of conduction at the bypass connection between ventricle and atrium; 4) slowing of intraatrial conduction.

Both patients showed the same pattern of VA conduction during the study performed prior to dissection of the His bundle, suggesting that VA conduction at that time occurred by the accessory pathway only, or that this pathway had a shorter refractory period than the His-AV nodal pathway (see below).

Conduction Via the AP Only or Conduction by Both the AP and the H-AV P With the AP Having the Shortest Refractory Period or Both Refractory Periods Being Identical

Twenty patients showed no increase in VA conduction times even when premature beats were elicited at very short coupling intervals, when factors discussed above could have caused an increase in VA conduction time. In these patients

Figure 2

Initiation of tachycardia by a ventricular premature beat (elicited after 400 msec) during driving of the ventricle at a basic cycle length of 600 msec. Simultaneous recording of ECG leads I, III, V₁, and V₆, and right atrial (RA) and His bundle (His) electrograms in the top panel. Ladder diagrams are depicted in the bottom panel. Note that the interval between atrial activation following the last stimulus of the basic ventricular rhythm and atrial activation following the premature stimulus is actually 15 msec shorter than the premature beat interval. This could be explained by assuming that the testing stimulus, following changes in cardiac dimensions after the last beat of the basic rhythm, was not given at the same stimulus site as during basic driving. This is favored by the observation that the QRS complex following the test stimulus is not completely identical to the QRS complex during basic rhythm. During tachycardia the Q-P interval is almost the same as the P-H interval suggesting that the atrial end of the accessory pathway was located close to the AV node.
we do not know whether VA conduction also occurred, up to a certain premature beat interval, along the H-AV P, with the His bundle complex being buried within the QRS complex. Depending upon the distance between the site of ventricular stimulation and the location of the H-AV P and AP and the conduction properties of the two pathways, one might theoretically expect that at certain premature beat intervals atrial activation starts both at the atrial end of the AP and at the atrial end of the AV node.

On increasing the prematurity of the test pulse, increase in VA conduction time over the H-AV P, with constant VA conduction time over the AP, will lead to less and eventually to no contribution to atrial excitation via the H-AV P.

In 14 of the 20 patients who did not show an increase in VA time during ventricular pacing, tachycardias could be initiated by a single ventricular premature beat. An example is given in figure 2. This pattern of initiation of tachycardia suggests that at a critical premature beat interval VA conduction occurred only via the AP. Following atrial activation, AV conduction by way of the AV-H P completes the first reentry cycle of the ensuing tachycardia. The absence of any increase in VA conduction time makes reentry in the AV node very unlikely.

![Figure 3](https://example.com/figure3.png)

**Figure 3**

VA conduction patterns at three different premature beat intervals during regular pacing of the right ventricle at a basic cycle length of 600 msec. At a premature beat interval of 275 msec (Panel A), VA conduction time following the premature beat is the same as during basic driving. At a premature beat interval of 265 msec (Panel B) sudden prolongation of the VA conduction time occurs. Further prolongation of VA conduction time could be observed on shortening the premature beat interval (Panel C) until the ventricle became refractory at a premature beat interval of less than 215 msec.
We do not know the exact site of the VA block in the H-AV P at the time of initiation of tachycardia. His bundle activation might have been hidden in the QRS complex. It is therefore not possible to localize the block at the muscle-Purkinje junction, the bundle branches, the His bundle or the AV node.

**VA Conduction over Both Pathways with the H-AV P Having the Shortest Refractory Period**

In five patients no change in the VA interval was observed until at a critical premature beat interval sudden marked prolongation in VA conduction time occurred. The premature beat intervals at that time varied between 230 and 300 msec (an example is given in fig. 3). In four patients the basic cycle length during ventricular pacing measured 600 msec, in one 650 msec. In the latter patient tachycardias could be initiated. As shown in figure 4, a premature beat elicited after an interval of 290 msec was followed by atrial activation, 360 msec later than atrial activation following the last beat of the basic ventricular rhythm. This ventricular premature beat is followed after 280 msec by a ventricular complex which is now preceded by a His bundle deflection. In the His bundle lead the time between atrial activation following the ventricular premature beat and the His bundle complex is only 40 msec, a finding which excludes the possibility that this ventricular complex is the result of VA conduction over the anomalous pathway, atrial activation and AV conduction over the AV-HP. Most probably reentry in the AV junction is responsible

**Figure 4**

Initiation of a tachycardia by a ventricular premature beat (given after 290 msec) during driving of the right ventricle at a basic CL of 650 msec. The VA conduction time following the premature beat is longer than the VA conduction time during basic rhythm. As shown in the His bundle lead following the premature beat there is a very short interval of time between atrial activation and His bundle activation, excluding the atrium as an essential link during the initiation of the tachycardia. The exact site of re-entry during the initiation of the tachycardia is not known. We also do not know whether, after the tachycardia has been initiated, the accessory pathway is incorporated in the tachycardia pathway during the subsequent beats of the tachycardia.
for the initiation of the tachycardia. Left aberrant conduction could explain the configuration of the first beat of the tachycardia.

Gradual Prolongation of VA Interval, Suggesting VA Conduction by Way of the H-AV P Only

Five patients showed a gradual increase of the VA interval on shortening the premature beat interval. In two patients this pattern of VA conduction was followed at a certain range of premature beat intervals by a ventricular complex showing pre-excitation. As shown in figure 5 this sequence seems to be the result of VA conduction by way of the H-AV P only, followed by a reciprocal beat with AV conduction over the accessory pathway. From the same figure it becomes apparent that a delay in transmission in the area between the His bundle and atrium was essential for the occurrence of the reciprocal beat. As shown, at a premature beat

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

**Figure 5**

VA conduction at four different premature beat intervals during pacing of the ventricle at a basic CL of 600 msec. A right atrial lead (RA), a His bundle lead (His), and lead V₅ are shown. As demonstrated in panel A at a V₃-V₈ interval of 500 msec the corresponding A₁-A₂ interval measures 660 msec, indicating VA conduction via the His-AV nodal pathway. At a V₃-V₈ interval of 420 msec (panel B) the atrial complex (A₂) is followed after a very short interval by a ventricular complex, where configuration is identical to the pre-excitation complex brought out during atrial pacing and shown on the left side of the figure. This ventricular complex is considered the result of re-entry over the accessory pathway. In panel C (premature beat interval of 390 msec) the His bundle complex can be seen following the premature ventricular complex. Re-entry over the accessory pathway was present up to a premature beat interval of 320 msec. As shown in panel D at this premature beat interval retardation of impulse conduction in the area between site of stimulation and His bundle results in later arrival at the AV node, faster transmission through this area and prevention of re-entry over the accessory pathway. The ventricle became refractory at a premature beat interval of 260 msec.
interval of 320 msec when the zone of delay in VA conduction is situated lower in the specific conduction system (in the area between site of stimulation and His bundle) the impulse arrives later at the AV node and is able to pass faster through this area. The subsequent atrial activation finds the anomalous pathway refractory and no reciprocal beat occurs.

In one patient tachycardias demonstrating AV conduction over the accessory pathway could be initiated by a single ventricular premature beat (fig. 6). As shown in the diagram we assume that at a critical premature beat interval exclusive VA conduction over the His-AV pathway is followed by AV conduction over the accessory pathway. Perpetuation of this circus movement resulted in tachycardia which could be terminated by a single ventricular premature beat.

Two patients showed a VA conduction pattern which could not be classified in the categories given above. Both showed a small but gradual increase in VA conduction time up to a certain premature beat interval when sudden prolongation of the VA conduction time occurred. A possible explanation for these findings could be that the initial gradual increase in VA conduction time was either the result of intraventricular delay or delay in the AP, the sudden further prolongation in VA conduction time being the result of block in the AP with exclusive VA conduction via the H-AV P.

**Discussion**

In approximately 90 percent of patients with normal AV conduction and the AV nodal-His pathway as their only connection between atrium and ventricle, ventriculo-atrial conduction can be demonstrated by ventricular pacing at rates slightly in excess of the sinus rate. Studies on AV nodal conduction using the single test stimulus method during atrial pacing have revealed three different conduction patterns. The single test stimulus method has also been used during ventricular pacing, although the published results are mainly concerned with the initiation of single or sustained AV nodal re-entry at certain premature beat intervals. Apart from the

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**Figure 6**

Initiation of a tachycardia showing AV conduction over the accessory pathway. As shown in the diagram we assume that at a critical premature beat interval (250 msec) ventricular activation is followed by conduction to the atrium via the His-AV nodal pathway only. After activation of the atrium AV conduction over the accessory pathway results in a ventricular complex showing a typical pattern of pre-excitation (see the QRS configuration during sinus rhythm on the left side of the figure). Continuation of this sequence of activation results in a tachycardia with QRS complexes having the characteristic configuration of pre-excitation.
patients reported by Goldreyer and Bigger, we do not have a systematic study on ventriculo-atrial conduction using the single test stimulus method in patients with normal AV conduction, without bundle branch block or tachycardias. In ventriculo-atrial conduction several structures are involved: in transmitting the impulse from ventricle to atrium, they use ventricular muscle, Purkinje tissue, bundle branches, His bundle, AV node proper and atrium. The functional characteristics of each of these structures will play a role in the VA conduction patterns and will obviously complicate such a study in the intact human heart. With His bundle recordings during ventricular pacing (if the His bundle electrogram is not hidden in the ventricular complex) one can at present study conduction patterns only in the area between the site of stimulation and the His bundle recording site and in the AV node proper. Using these techniques, conduction delay following ventricular premature beats has been demonstrated in both areas.

In the Wolff-Parkinson-White syndrome two pathways are present between atrium and ventricle, obviously increasing the possible patterns of VA conduction in these patients. In the absence of data on VA conduction patterns in the normal intact human heart, we have considered the absence of a change in VA conduction time following induced ventricular beats with increasing prematurity as typical for conduction over the accessory pathway, similar to the constant atrium-delta interval following premature test stimuli during atrial pacing. Twenty-two of our patients showed this pattern. In two of them the AV nodal-His pathway had been surgically dissected. From one of the latter patients graph A (fig. 7A) was constructed. In this graph the relation between the ventricular premature beat interval (V1-V2) and the distance between atrial activation following the last beat of the regular driven ventricular rhythm and atrial activation following the ventricular premature beat (A1-A2) is shown. Only at very short premature beat intervals (in the range 270 to 240 msec) does the curve deviate from the line of identity. As described earlier, increase in latency time and increase in ventricular, bypass, or atrial conduction times are probably responsible for this finding.

In 14 patients showing this pattern, tachycardias could be initiated by a single ventricular premature beat. Because of our inability to register a His bundle electrogram following the ventricular premature beat (which might have been hidden in the ventricular complex) we do not know whether at this coupling interval, block in the His-AV nodal pathway occurred in the area below the His bundle or in the AV node proper. We feel that the absence of increase in VA conduction time on initiating the tachycardia is a very strong argument for involvement of the accessory pathway in the tachycardia circuit. Figure 7B shows the relation between V1-V2 intervals and corresponding atrial activations (A1-A2) from the patient shown in figure 3. In view of the events shown in the His bundle electrogram the sudden deviation from the line of identity at a V1-V2 interval of 265 msec was interpreted as indicating block in the accessory pathway followed by VA conduction over the His-AV node pathway. Conduction over the latter pathway was possible to a V1-V2 interval of 215 msec. From figure 3 a marked increase in latency time and changes in the QRS complex following an early premature stimulus become apparent. The lead from the right atrium was not very helpful in this patient in demonstrating different atrial activation patterns when VA conduction occurred via the accessory pathway as compared to conduction over the His-AV node pathway. As described by Grolleau et al. two intraatrial leads are frequently necessary to show these changes.

In one patient (fig. 4) a tachycardia could be initiated by a single ventricular premature beat given in an interval range of 30 msec, starting at a V1-V2 interval 10 msec shorter than the V1-V2 interval showing sudden deviation from the line of identity. As described under results we think that in the initiation of this tachycardia neither the accessory pathway nor the atrium was involved in the re-entry circuit. We do not know whether the actual site of re-entry was the bundle branches, the His bundle or the AV node. We are also not informed about the pathway of the impulse during the tachycardia. Following initiation of the tachycardia, was the AV node pathway or the accessory pathway involved in the tachycardia circuit?

Five patients showed a gradual increase in A1-A2 interval following shortening of the V1-V2 interval. We have regarded this as indicative of VA conduction over the His-AV node pathway only. Two patients showed re-entry over the accessory pathway within a certain V1-V2 interval range. This phenomenon has also been described by Castellanos, Castillo and Agha. A graph relating V1-V2 and A1-A2 intervals from one of these patients is given on figure 7C. As shown in figure 5 (which is from the same patient as fig. 7C), a critical delay in AV nodal transmission was essential for the
occurrence of these phenomena. When shortening the $V_1-V_2$ interval led to an increase in $V_2-H_2$ interval, with later arrival of the impulse at the AV node and faster transmission through this structure, re-entry disappeared. In one patient showing this pattern of VA conduction a tachycardia could be initiated with AV conduction over the accessory pathway (fig. 6). To our knowledge this is the first example of a circus movement tachycardia with antegrade conduction over the accessory pathway following a critically timed ventricular premature beat.

A very interesting finding in this study was that in three patients where atrial pacing revealed AV conduction over both pathways, no VA conduction could be demonstrated at all. Added to the five

Figure 7
Relation between $V_1-V_2$ and $A_1-A_2$ intervals in four patients showing different VA conduction patterns. Graphs B and C correspond to patients shown in figures 4 and 5. The cross hatched area in graph C indicates the zone of $V_1-V_2$ intervals during which $A_2$ was followed by re-entry over the accessory pathway.
patients who showed only VA conduction via the His-AV nodal pathway, this means that eight of the 36 patients studied did not show VA conduction by way of the accessory pathway during ventricular pacing. In two patients another VA conduction pattern was found. As shown in fig. 7D, taken from one of them, on shortening the premature beat interval a small but gradual increase in VA conduction time occurred. At a certain premature beat interval VA conduction time suddenly became markedly prolonged. As postulated earlier this sudden prolongation of VA conduction time at a critical premature beat interval might indicate delay in the ventricle or the accessory pathway with block in the latter followed by exclusive VA conduction over the His-AV nodal pathway.

In patients in whom the accessory pathway had the shortest refractory period of the two pathways, we compared these data with the refractory periods of the accessory pathway obtained during atrial pacing using the single test stimulus method. In order to make this comparison we required: a) that the basic pacing frequency during ventricular and atrial pacing during which the test stimuli were applied were the same; and b) that the refractory period of the right atrium and the right ventricle was shorter than that of the accessory pathway. Only 11 patients fulfilled these criteria. Ten of them showed that the \( V_1-V_2 \) interval at which the accessory pathway became blocked was shorter than the \( A_1-A_2 \) interval at which this occurred during atrial pacing. This is in contrast to the findings of Goldreyer and Bigger\(^ {10} \) and Damato, Lau and Bobb\(^ {10} \) that the refractory period of the normal AV junction during ventricular pacing is longer than during atrial pacing. Possibly this phenomenon is explained by a more marked increase in latency time and widening of the ventricular complex following early ventricular premature beats, with subsequent later arrival at the ventricular end of the accessory pathway.

References

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