Unusual re-entry mechanisms in patients with Wolff-Parkinson-White syndrome

H. Neuss and M. Schlepper
From Kerckhoff-Klinik, Bad Nauheim, West Germany

Three patients with the Wolff-Parkinson-White (WPW) syndrome were studied using His bundle recordings and programmed atrial stimulation. In two of them conduction of premature atrial depolarizations via the accessory pathway was possible when the His-Purkinje system was still refractory. The consequent conduction delay in the His-Purkinje system resulted in re-entry phenomena at the ventricular level in one case, and possibly also in another. In the third patient short runs of supraventricular tachycardia could be elicited, which were due to a longitudinal dissociation within the atrioventricular node. During these paroxysms, ventricular activation occurred over both the anomalous and the normal paths. The resulting QRS complexes resembled fusion beats as indicated by their configuration and preceding H potentials.

Supraventricular tachycardias in patients with the WPW syndrome are usually thought to result from re-entry mechanisms involving both the normal and the accessory pathways of atrioventricular conduction. The increasing evidence favouring this mechanism has been largely obtained by studying these arrhythmias using intracardiac electrographic techniques (Castillo and Castellanos, 1970; Narula, 1973; Wellens, 1971).

Intracardiac electrograms combined with programmed atrial stimulation were used to elicit and analyse re-entry mechanisms in 3 patients with the WPW syndrome complicated by paroxysmal tachycardia. In none was the usual re-entry mechanism, involving accessory and normal pathways, responsible.

Methods
All three patients consented to the electrophysiological studies, were in a non-sedated state, and were not receiving antiarrhythmic medication at the time of the examination. A 5-polar catheter was passed via the right femoral vein and positioned to the right of the intraventricular septum adjacent to the bundle of His, according to the method of Scherlag et al. (1969). From this catheter filtered (40 to 200 Hz) low atrial (A) and His bundle depolarizations (H) were recorded. A second 4-polar catheter (4F) was positioned via the same femoral vein in the proximity of the sinus node. Two electrodes were used to record a high atrial electrogram; the two remaining electrodes served for stimulation. Simultaneously several surface electrocardiogram leads were registered. The atrial stimuli consisted of rectangular impulses of 2.0 msec duration and were twice diastolic threshold. They were applied either in the form of bigeminy or after each eighth beat during sinus rhythm or stimulated atrial rhythm. In the latter case the impulse following the premature stimulus was dropped to ensure a pause long enough for the study of re-entry mechanisms. Surface and intracardiac electrograms were recorded on magnetic tape and displayed at a paper speed of 100 mm/sec using an ink-writing system. The following intervals were measured.

a) PA interval: from the onset of the P wave in the electrocardiogram to the A potential in the His bundle electrogram — representing the intra-atrial part of atrioventricular conduction.

b) P delta interval: from the beginning of P in the electrocardiogram to the onset of the delta wave.

c) PJ interval: from the onset of the P wave to the junction of the QRS complex with the ST segment.

d) AH interval: represents the intranodal conduction time and is measured from the onset of the A potential in the His bundle electrogram to the beginning of the His spike.

e) HQ interval: from the beginning of the H potential to the earliest appearance of the ventricular deflection in the electrocardiogram. This interval represents the shortest conduction time in the His-Purkinje system.

Patients

Case I
A 21-year-old female patient exhibited the full picture of the WPW syndrome type B during sinus rhythm. P
Independent and retrograde potentials. Ventricular potentials, which were similar in morphological configuration to those observed in Wolff-Parkinson-White syndrome, were recorded at an AH interval of 185 msec. When the coupling interval was 290 msec or more, the His-Purkinje conduction time was prolonged, and the atrial and ventricular deflections were more apparent. When the coupling interval was 260 msec, only the atrial deflection could be recorded, in addition to the ventricular deflection, indicating that the refractory period of the accessory pathway was shorter than 260 msec.

In summary, the findings support the hypothesis that the Wolff-Parkinson-White syndrome is a manifestation of abnormal atrioventricular retrograde conduction. The results also indicate that the Wolff-Parkinson-White syndrome may be associated with an increased risk of sudden death, particularly in patients with a long QT interval. Further research is needed to clarify the mechanisms involved in these findings and their clinical implications.
repolarization (phase 2 to 3) in the His-Purkinje system (Hoffman and Cranefield, 1960; Trautwein and Dudel, 1954). It thus appears that premature atrial depolarizations may activate the ventricular myocardium via the accessory pathway when refractory dependent conduction delay is still present in the structures of the specialized conducting system. The importance of this observation for the initiation of re-entry mechanisms at the ventricular level is emphasized by the findings in the next case.

Case 2
A 24-year-old woman with a WPW syndrome, type B, and a history of tachycardia had a P delta interval of 130 and a PJ interval of 250 msec during sinus rhythm. PJ was only slightly prolonged to 270 msec during atrial stimulation up to 180 a minute while the P delta interval remained unchanged. When atrial bigeminy was induced during sinus rhythm, the effective refractory periods of both the accessory and the normal pathways were reached at a PS interval of 290 msec (Fig. 2a). At a PS interval of 300 msec the premature atrial depolarization traversed the anomalous pathway. Following the ventricular potentials a predominant negative deflection was registered (H') at an interval of 320 msec after the preceding stimulus. The next premature atrial depolarization, with an identical PS interval, elicited a similar deflection after V; the SH interval was reduced to 300 msec, the preceding RR interval being 710 msec.
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FIG. 2 (Case 2) a) Bigeminal premature atrial depolarizations (PAD) with a PS interval of 290 are ineffective. On increasing the PS interval to 300 msec, PADs are exclusively propagated via the accessory pathway. These V potentials were followed by retrograde His depolarizations (H'). b) and c) At a paced rate of 150/min (S1S1 = 400 msec) the PADs with an S1S2 interval of 290 and 270 msec, respectively, were now propagated over the accessory pathway. The V potentials were followed by retrograde H spikes.

FIG. 3 (Case 2) Paced heart rate 120/min (S1S1 = 500 msec). a) and b) Premature atrial depolarizations (PADs) with an S1S2 interval of 330 and 290 msec, respectively, were propagated via the accessory path; the S2H' interval showed a reversed relation to the S1S2 interval. c) Shortening of the S1S2 interval to 280 msec resulted in a prolongation of S2H' to 340 msec. Under this condition the H' potential was followed by a QRS of left bundle-branch block morphology and an atrial echo. The ladder diagram shows the analysis of the conduction sequences.
This H' potential became visible only at PS intervals of 300 to 350 msec during induced atrial bigeminy. At a paced basic heart rate of 150 a minute (S1S1: 400 msec) the effective refractory period of the accessory pathway was reduced to 220 msec, therefore, premature atrial depolarizations with an S1S1 interval of 290 msec (Fig. 2b) and 270 msec (Fig. 2c), respectively, were now conducted via the accessory pathway.

H' potentials were only recorded at S1S2 intervals between 290 and 260 msec. At S1S3 of 290 msec the resulting S2H' interval was 250 msec and became 260 msec when S1S3 was reduced to 270 msec.

At a basic heart rate of 120 a minute (S1S1: 500 msec) H' deflections were registered at S1S2 intervals of 330 to 270 msec. Again the longest S2H' interval was observed after the shortest S1S2 interval (Fig. 3). After an S2H' interval of 340 msec an early ventricular depolarization occurred, 50 msec after the H' deflection, and showed left bundle-branch block without evidence of pre-excitation; 160 msec after H' an atrial echo appeared (Fig. 3c).

Intermittent block of the accessory pathway appeared when the driving frequency was increased to 200 a minute. Atrial stimuli were sometimes exclusively propagated via the normal pathway. Preceding the QRS complexes (no. 1 and 8, Fig. 4) were H potentials which showed a predominantly positive deflection, as opposed to the H' potentials seen in Fig. 2 and 3. If the RR distance was about 600 msec (QRS 4 to 5) the two following excitations were propagated via the accessory pathway and succeeded regularly by a QRS complex of left bundle-branch block configuration without pre-excitation (no. 7). This triplet of QRS complexes was identical to that in Fig. 3c. Hence, similar mechanisms of excitation can be assumed.

The following analysis appears to explain these findings. H' potentials are caused by retrograde activation of the bundle of His. This assumption is favoured by 3 observations:

1) The different polarity of H and H' potentials;
2) An increase in basic heart rate shortens the S2H' interval apparently because refractory-dependent conduction delay in the His-Purkinje system diminishes with increasing heart rate (Schuilenburg and Durrer, 1973; Moe and Mendez, 1971; Moe, Mendez, and Han, 1965). In the case of anterograde His activation the opposite would be expected, because of atrioventricular nodal conduction delay.
3) At higher basic heart rates H' potentials occurred at shorter S1S2 intervals. This is in accordance with the well-known frequency-dependent shortening of refractory periods in the His-Purkinje system (Hoffman and Cranefield, 1960; Trautwein and Dudel, 1954). VH conduction delay, which results in the occurrence of retrograde H' potentials should become evident at longer coupling intervals when basic heart rate is slow and at shorter S1S2 intervals when basic heart rate is faster.

In Fig. 3c the premature atrial depolarization leads to an early excitation of the right ventricle due to WPW syndrome type B. Propagated via the myocardium the excitation wave reaches the specialized conduction system of the left ventricle at a time when the His-Purkinje system is out of its effective refractory period. After the delayed retrograde conduction the bundle of His is depolarized and the right bundle-branch is anterogradely penetrated causing a ventricular echo of left bundle-branch block configuration. The succeeding retrograde atrial activation most probably occurs via the accessory pathway. The similarity of the configuration

![Image](http://heart.bmj.com/)
of QRS complex 7 in Fig. 4 as well as the time relation suggest that the excitation mechanisms are identical. The common features of Cases 1 and 2 are that induced premature atrial depolarizations lead to pre-excitation of the ventricular myocardium at a time when conductivity of His-Purkinje system has not been fully restored. In Case 1 this is indicated by the appearance of anterograde H potentials following the V potentials and the occurrence of physiological interference at the level of atrioventricular node/bundle of His. In the second case the impairment of conductivity was indicated by the pronounced delay of retrograde VH conduction, especially at lower basic heart rates. This functional impairment of conductivity is the most important precondition for the development of a re-entry mechanism in the His-Purkinje system such as could be demonstrated in Case 2.

Case 3
Another re-entry mechanism was observed in a 63-year-old male patient with WPW syndrome type A. He had experienced brief paroxysms of tachycardia for at least 10 years. Using atrial stimulation there was a stable conduction via the accessory pathway up to a rate of 200 a minute. At all frequency ranges the effective refractory period of the accessory pathway was found to be shorter than that of the nodal conduction path. At sinus rhythm with RR intervals between 800 and 1050 msec H potentials were noted at the beginning of the delta wave in the surface electrocardiogram, AH measuring from 100 to 130 msec. Premature atrial depolarizations with a PS interval of 420 to 360 msec were always able to elicit short runs of tachycardia.

In Fig. 5a a premature atrial depolarization with a PS interval of 420 msec produces maximal ventricular pre-excitation, which after a delta-A' interval of 440 msec is followed by a retrograde atrial depolarization. QRS complex 4 shows less pre-excitation as shown by the QRS configuration and an H spike simultaneous with the delta wave (A'H: 170 msec). 440 msec after this ventricular echo there is another atrial echo which in

| FIG. 5 (Case 3) a) A premature atrial depolarization (PAD) with a PS interval of 420 msec produces a QRS with maximal pre-excitation, which is followed by an atrial echo. The succeeding QRS complexes no. 4 and 5 are fusion beats resulting from excitation via the normal and anomalous pathway. This is indicated by the preceding H spikes. b) A PAD with a coupling interval of 350 msec initiated a supraventricular tachycardia. c) A PAD with a coupling interval of 350 msec secondly causes one atrial echo without initiating a tachycardia. d) At a further decreased coupling interval (340 msec) anterograde nodal conduction was delayed so that the bundle of His could be depolarized retrogradely. |
turn leads to another ventricular activation. This QRS (no. 5) also showed evidence of propagation via both the normal and anomalous pathway. QRS 5 and 4 are therefore fusion beats as are QRS 1 and 2. The more pronounced degree of pre-excitation is caused by the rate-dependent prolongation of AH interval from 120 to 170 msec. There is no retrograde atrial activation following QRS 5, so the re-entry mechanism is terminated. The next stimulated excitation is not relevant to the explanation of the underlying mechanism. A premature atrial depolarization with a PS interval of 350 msec (Fig. 5b) was followed by a fully pre-excited QRS (no. 3), which was in turn succeeded by retrograde activation of the atria. This in consequence produces a fusion beat with an AH interval of 150 msec initiating a spell of tachycardia. Because of the shorter RR intervals (490 to 450 msec) and the rate-dependent prolongation of AH, the next QRS complexes show maximal pre-excitation, but are always preceded by retrograde atrial activation. Apparently at this PS interval borderline conditions are reached. In Fig. 5c the same PS interval only leads to an atrial echo but does not initiate a tachycardia. Contrary to Fig. 5b the anterograde H spike (AH: 280 msec) succeeds the fully pre-excited stimulated QRS. The fact that the observed atrial echo was followed by a ventricular depolarization with pronounced pre-excitation (QRS complex no. 4), while no His potential was discernible, favours the interpretation that anterograde block supra His has occurred and prevented repetitive reciprocation. By reducing PS to 340 msec (Fig. 5d), the stimulated QRS complex is succeeded by a predominant positive deflection (AH': 280 msec) which probably presents a retrograde His spike (due to block or greater delay in anterograde atrioventricular nodal conduction). An atrial echo could not be elicited.

The brief paroxysms are most probably initiated and maintained by a re-entry mechanism sparing the accessory pathway. Premature atrial depolarizations with a PS interval of 420 to 350 msec (echo range) meet the atrioventricular node in the state of longitudinal dissociation. The excitation reaches the bundle of His via fibres with a short refractory period (alpha-path) while anterograde conduction was blocked in other fibres with a longer refractory period (beta-path). If the distal atrioventricular node was reached with a critical delay, reciprocation via the now recovered beta-path is possible and leads to an atrial echo (Mendez and Moe, 1966; Denes et al., 1973; Schuilenburg and Durrer, 1968; Goldreyer and Damato, 1971). This is again propagated over the alpha-pathway and the accessory pathway (bundle of Kent), eliciting ventricular fusion beats.

Discussion

A predisposing mechanism for the initiation of reciprocating tachycardias is the presence of two parallel conducting atrioventricular pathways with different functional properties (Schamroth and Yoshonis, 1969). In patients with WPW syndrome reciprocating tachycardia are thought to involve two separate anatomical communications (Durrer et al., 1967), one of them the accessory path and the other the normal atrioventricular pathway. Premature atrial depolarizations with a critical coupling interval may find one path refractory (path A) while atrioventricular conduction via the other path (path B) is possible (Durrer et al., 1967; Castil and Castellanos, 1970; Narula, 1973). On the ventricular level the impulse may enter path A and return to the atria. If the initial path is responsive once again circus movement may continue. If the accessory pathway is traversed retrogradely and the normal path is used for anterograde conduction pre-excitation will disappear. If, however, the anomalous path is used for atrioventricular conduction QRS complexes will show maximal pre-excitation during supraventricular tachycardia.

Generally re-entry tachycardias involving parallel conducting structures may occur in any part of the conducting system (Mendez and Moe, 1966; Wellens, Schuilenburg, and Durrer, 1972). Their analysis is impossible to achieve by use of the surface electrocardiogram. Special techniques with intracardiac registration of electrograms and programmed atrial and ventricular stimulation are necessary to differentiate between intranodal re-entry, re-entry in the His-Purkinje system, and re-entry involving the atrioventricular node/His system and an accessory atrioventricular connexion.

According to our analysis, Case 2 demonstrates a re-entry mechanism at the ventricular level which is thought to be the underlying mechanism in certain forms of ventricular tachycardia. Circus movement in these tachycardias, which can include the bundle-branches as well as the bundle of His, have been studied with programmed stimulation (Wellens et al., 1972). However, documentation of the conduction sequence within His-Purkinje system is still lacking. In animal experiments this mechanism was well established by the studies of Moe et al. (1965).

Although in the first patient no re-entry phenomenon could be provoked, the findings support the conclusions drawn in Case 2. It could be demonstrated that in both patients the functional properties of the accessory pathway were quite different from those of the His-Purkinje system. This permits the propagation of premature atrial depolarizations to the ventricle at a time when a repolarization of the His-Purkinje system had not been fully completed. Phase 3 dependent conductivity impairment is the premise for re-entry mechanisms in the His-Purkinje system. It seems possible that atrial premature beats may not only cause single echo phenomena as in the second patient but may initiate self-maintaining ventricular tachycardia in the WPW syndrome.
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The existence of longitudinal dissociation in the normal atrioventricular conduction pathway and the presence of an accessory pathway of the Kent bundle type is the explanation for the re-entry mechanism observed in the third patient. A similar assumption has been made by Wellens (1971), to interpret the initiation of a supraventricular tachycardia by double ventricular stimuli in a patient with WPW syndrome. However, the explanation is limited to the initiation of the tachycardia. Findings suggesting a coexistence of accessory atrioventricular pathways, and dual atrioventricular nodal pathways are described by Spurrell, Krikler, and Sowton (1973) and by Friedberg and Schamroth (1973). In our patients the findings clearly indicate that the accessory pathway takes part neither in the initiation nor in the maintenance of the tachycardia and that conduction over the accessory pathway was incidental.

References


Requests for reprints to Dr. M. Schlepper, Kerkhoff-Klinik, 635 Bad Nauheim, Benekestr. 6–8, West Germany.
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H Neuss and M Schlepper

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