

Wolff-Parkinson-White syndrome type B with tachycardia-dependent (phase 3) block in the accessory pathway and in left bundle-branch coexisting with rate-unrelated right bundle-branch block

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SUMMARY A patient with Wolff-Parkinson-White syndrome type B developed 2:1 atrioventricular block resulting from the association of persistent right bundle-branch block with tachycardia-dependent (phase 3) left bundle-branch block. Electrophysiological studies disclosed the coexistence of a tachycardia-dependent (phase 3) block in the accessory pathway. This conduction disturbance was exposed, not by carotid sinus massage as in previous studies, but by pacing-induced prolongation of the interval between two consecutively conducted atrial impulses. Furthermore, the surface electrocardiogram showed, at different times, ventricular complexes resulting from: (1) exclusive atrioventricular conduction through the normal pathway without bundle-branch block; (2) predominant, or exclusive, atrioventricular conduction through a right-sided accessory pathway; (3) exclusive atrioventricular conduction through the normal pathway with right bundle-branch block; (4) exclusive conduction through the normal pathway, with left bundle-branch block; (5) fusion between (1) and (2); and finally, (6) fusion between (2) and (3).

However, QRS complexes resulting from simultaneously occurring Wolff-Parkinson-White syndrome type B and left bundle-branch block could not be identified. Future electrophysiological investigations should re-evaluate the criteria used to differentiate between true and false patterns of Wolff-Parkinson-White syndrome type B coexisting with left bundle-branch block.

The association of Wolff-Parkinson-White (WPW) syndrome, either type A or type B, with right bundle-branch block has been well recognised.¹⁻⁷ However, there are far fewer reports of the coexistence of left bundle-branch block with pre-excitation.^{1 2 8-13} Even more rare is the occurrence of both right and left bundle-branch block in a patient with WPW type B.

Case report

A 62-year-old woman with WPW syndrome type B and recurrent supraventricular tachycardias was referred for electrophysiological evaluation after the appearance of right bundle-branch block and 2:1 atrioventricular block. Electrocardiograms during previous admissions had also shown pure left

bundle-branch block, but exclusively when the sinus cycle lengths were shorter than 620 ms, that is when the rates were over 96/min (Fig. 1A). On the other hand, WPW type B was seen only with longer sinus cycle lengths. For example, Fig. 1B and C recorded while the rates ranged between 79 and 86/min show that the PR intervals were short (120 ms), the QRS complexes wide and of varying duration (100 to 130 ms), and the electrical axis deviated abnormally to the left (about -60°). Finally, Fig. 1D was obtained after pre-excitation had been abolished by the intravenous administration of 600 mg procainamide (given at a rate of 100 mg every five minutes). The sinus rates ranged between 79 and 83/min. The QRS complexes were narrow, there being no evidence whatsoever of left bundle-branch block.

In Fig. 1, physiological (rate-related) and pharmacological variations in the PR intervals prevented the comparison between the PJ (or PS) intervals occurring during left bundle-branch block, WPW type B, and normal conduction with narrow QRS complexes. Nevertheless, the effects of procainamide suggested that the pre-excitation complexes were fusion beats resulting from ventricular activation through both accessory and normal pathways (without left bundle-branch block in the latter). This assumption implies that the patient had a tachycardia-dependent (phase 3) left bundle-branch block.

Further evidence supporting the existence of a rate-related conduction disturbance in the left bundle-branch was obtained from intracardiac electrophysiological studies performed after development of right bundle-branch block. Thus, the first half of Fig. 2, recorded while the high right atrium was paced, shows 2:1 atrioventricular block. The first and third P waves, reaching the ventricles exclusively through the left bundle-branch, were followed 400 ms later by P waves blocked below the site from which the H deflection was recorded (presumably at both bundle-branches simultaneously).

In the beats conducted with a right bundle-branch block morphology the interval between the atrial (A) deflection of the His bundle electrocardiographic lead and the onset of ventricular (V) depolarisation (wherever it might have occurred) gave a measure of conduction time through the

normal (AV node—His bundle—left bundle-branch) pathway. The duration of the corresponding AH, HV, and H right ventricular apex (RVA) intervals was 110, 50 and 85 to 90 ms, respectively.

Since the pre-excitation had become concealed, attempts had to be made to expose, or unmask, atrioventricular conduction through the accessory pathway (Fig. 2 and 3). This was accomplished by increasing the intervals between two consecutively conducted atrial impulses by means of a single premature atrial stimulus delivered after every eighth blocked P wave. When these intervals were prolonged beyond 1000 ms, they were terminated by QRS complexes (last in Fig. 2 and in Fig. 3, left panel) which were different from those with a right bundle-branch block morphology in that: (a) lead I changed to a predominantly positive deflection (not followed by a wide S wave) with a slurring in its upstroke (delta wave); (b) the electrical axis was deviated abnormally to the left; and (c) lead V₁ changed to an Rsr' pattern (Fig. 2) or to an Rs morphology (Fig. 3, left panel). In these complexes AH remained at the control value (110 ms) but the H deflection was inscribed as the ventricles started to be depolarised by the impulse emerging from the accessory pathway. In consequence, the AV interval of 110 ms was used as a rough estimate of conduction time through the accessory pathway.^{14 15}

These findings suggested that the last QRS complexes in Fig. 2 and 3, left panel, reflect fusion resulting from ventricular activation through both

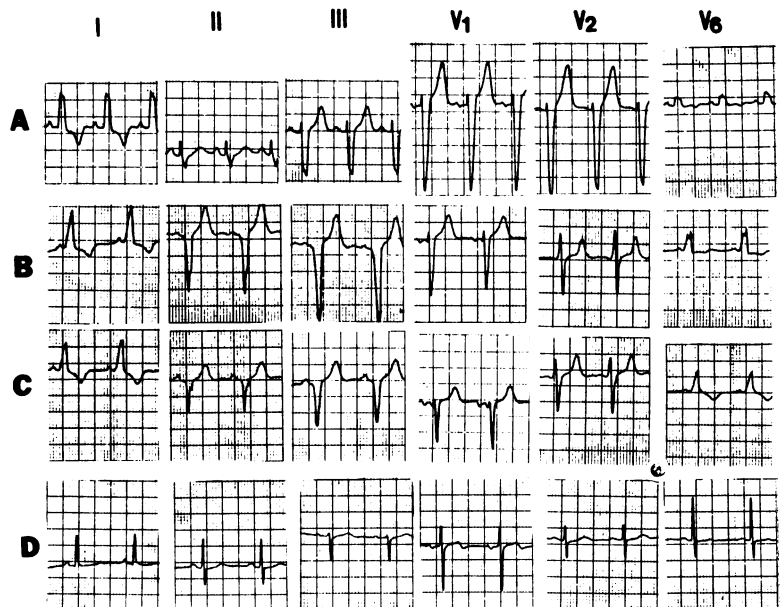


Fig. 1 Tachycardia-dependent block in the left bundle-branch and (possibly) in the accessory pathway (A); different degrees of pre-excitation with Wolff-Parkinson-White type B morphologies (B, C) and narrow QRS complexes after procainamide (D).

the accessory and normal pathways (with right bundle-branch block in the latter). On the other hand, the last QRS complex in Fig. 3, right panel, resulted from exclusive, or almost exclusive, conduction through the accessory pathway. It thus had a morphology similar to that in Fig. 1B.

Whenever pre-excitation occurred, the HV intervals, as well as the His-right ventricular apex (H-RVA) intervals, were shorter than in control complexes, indicating that the apex of the right ventricle was not activated by the impulse traversing the normal pathway, but by that emerging from the accessory pathway.^{14 15} Therefore, the time elapsing between the onset of the delta wave and arrival of excitation at the RVA (35 ms) was used to estimate conduction time from pre-excited site to the apex of the right ventricle.^{14 15} Moreover, if the AV interval (of 105 ms) preceding the ventricular complexes with a delta wave indeed reflected the conduction time through the accessory pathway, then the fusion complexes in Fig. 2 and 3 can be explained by assuming that the corresponding AH interval of 110 ms still allowed certain portions of the left ventricle to be activated by the impulse descending through the left bundle-branch.^{14 15}

However, the increase of the AH interval to 140 ms which occurred in the last complex of

the right panel of Fig. 3 (in turn resulting from the concealed penetration, into the atrioventricular node, of the preceding, premature, atrial beats) produced sufficient delay in the conduction of the impulse traversing the normal pathway so as to allow the impulse traversing the accessory pathway to activate all or most of both ventricles.^{14 15}

Discussion

DIFFICULTIES IN DETERMINING LOCATION OF ACCESSORY PATHWAY

The major problem in diagnosing the location of the accessory pathway in this case was related to the occurrence of pre-excitation only at very long cycle lengths (Fig. 2 and 3). Nevertheless, analysis of the surface electrocardiographic and intracardiac electrophysiological events suggested that the accessory pathway was right sided. Vectorial analysis of standard and chest leads showed that the abnormal ventricular depolarisation occurred in a superior, leftward, and posterior direction (Fig. 1 to 3). Moreover, during maximal pre-excitation the initial slurring was located partly in the left anterior quadrant and partly in the left posterior quadrant. Even in the absence of spatial vectorcardiograms these findings can be construed to indicate that the

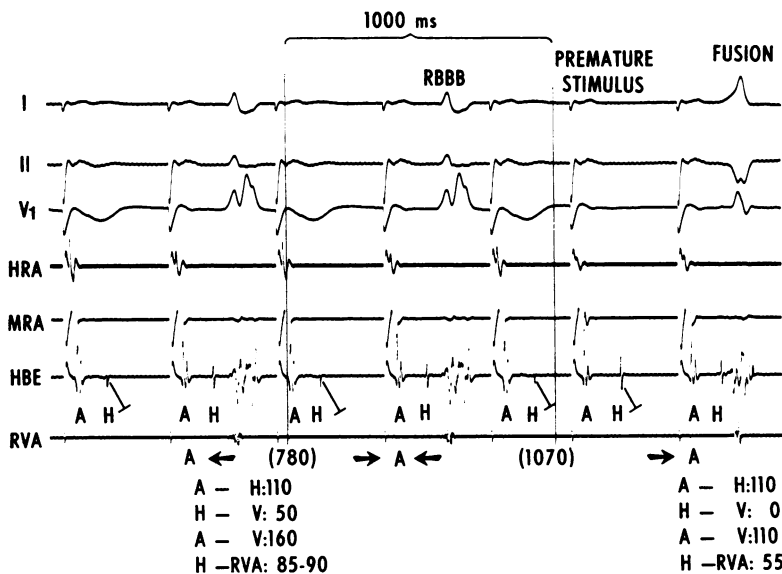


Fig. 2 Intracardiac recordings (obtained during atrial pacing) showing: (a) pure right bundle-branch block (RBBB) in conducted impulses (first two QRS complexes); (b) 2:1 (infra-Hisian) AV block resulting from the association of persistent RBBB with tachycardia-dependent (phase 3) left bundle-branch block (LBBB); (c) tachycardia-dependent (phase 3) block in the accessory pathway; and (d) fusion resulting from the coexistence of right bundle-branch block with WPW type B (last QRS complex). Numbers between arrows refer to the interval between two consecutively conducted atrial (A) impulses. All values are expressed in ms. HRA: high right atrium; MRA: mid-right atrium; RVA: right ventricular apex; A: atrial electrogram recorded in the His bundle electrogram (HBE) lead; V: onset of depolarisation, regardless of the pathway responsible for initial ventricular activation.

accessory pathway ended in a right-inferior, probably mid-septal, site.¹⁶

On the other hand, the intracardiac studies showed that the estimated conduction time between the pre-excited site and the right ventricular apex in beats with delta waves (35 ms) was similar to that previously reported in patients with WPW type B (40 to 50 ms).¹⁵ These values were significantly shorter than those reported in patients with WPW type A (120 to 160 ms).¹⁴

Theoretically, the distance between the ventricular end of the accessory pathway and the recording electrodes can be determined if the conduction time, conduction velocity, and location of conduction pathways are known. Unfortunately, these indices have not been adequately defined for the human ventricles: the reported conduction times have ranged widely between 440 mm/s (in revived hearts) to 1200 mm/s (in patients with implanted left ventricular pacemakers).^{17, 18} In canine hearts Lewis found that the conduction velocity through the ordinary muscle of the free right ventricular wall was approximately 400 mm/s.¹⁹

Calculations based on these values suggest that the distance between the site of emergence from the accessory pathway and the right ventricular apex in our two cases could have ranged between 15.4 and 42 mm.

TACHYCARDIA-DEPENDENT (PHASE 3) BLOCK IN ACCESSORY PATHWAY

The absence of pre-excitation (before the development of 2:1 AV block) in all tracings showing sinus tachycardia (Fig. 1A) could have been the result of tachycardia-dependent (phase 3) block in the accessory pathway.²⁰ However, this assumption was

not proved as electrophysiological studies were not performed.

In our opinion the diagnosis of the conduction disturbance under consideration cannot always be made exclusively from the surface electrocardiogram since multiple factors can produce false patterns of tachycardia-dependent (phase 3) block in the accessory pathway.²¹ Foremost among these are the differences in conduction time through normal pathway and anomalous pathway.

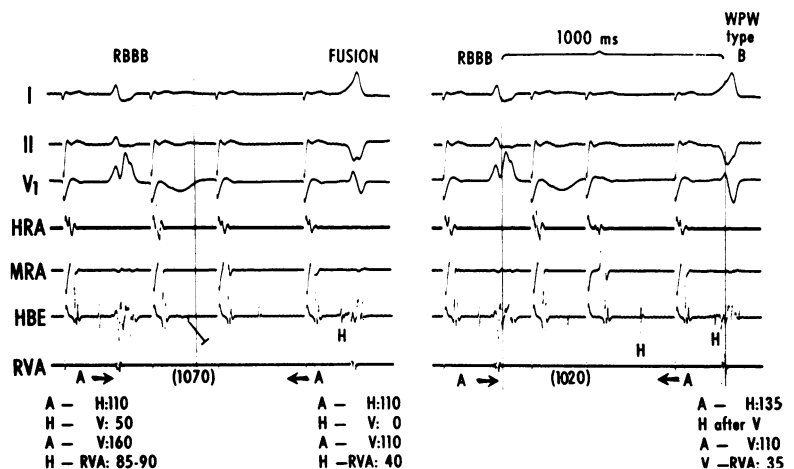
Disappearance of pre-excitation at relatively high rates may simply be an expression of a sympathetic enhancement of AV nodal conduction without any change in the physiological properties of the accessory pathway. In addition, this phenomenon has also been ascribed to a shift in the site of impulse initiation, to differential intra-atrial delays, and to changes in the site and mode of entry into the AV node and accessory pathways.^{21, 22} Similarly, the exposure of pre-excitation during sinuatrial slowing produced by carotid sinus massage²⁰ may be explained entirely by vagal-induced AV nodal block of high degree.

The method used to unmask the pre-excitation in Fig. 2 and 3 was different from that used by Przybylski *et al.*²⁰ Fig. 2 and 3 also show that the occurrence of pre-excitation at long cycle lengths was related to: (a) the duration of the phase 3 block in the accessory pathway; and (b) differences in conduction time through the latter and through the normal pathway.

RIGHT BUNDLE-BRANCH BLOCK IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME

The association of right bundle-branch block with WPW type A, or type B, has been well recognised and thoroughly discussed.¹⁻⁷

Fig. 3 Atrial pacing with: (a) pure right bundle-branch block in conducted impulses (first QRS complex in the left, and in the right panel); (b) 2:1 AV block resulting from the association of persistent right bundle-branch block and tachycardia-dependent (phase 3) block in the accessory pathway; (d) fusion resulting from the coexistence of right bundle-branch block with WPW type B (second QRS complex in the left panel); and (e) ventricular complex (last in the right panel) with a pure, or almost pure, WPW type B morphology.



LEFT BUNDLE-BRANCH BLOCK IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME TYPE A

In contrast, as recently emphasised by Krikler *et al.*¹³ fewer reports have dealt with left bundle-branch block coexisting with WPW type A.¹⁰⁻¹² Moreover, Krikler *et al.*¹³ also reviewed the published reports and presented information from two personal cases in which electrophysiological studies were performed.

RATE-UNRELATED LEFT BUNDLE-BRANCH BLOCK IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME TYPE B

In the very few reports dealing with this combination, the diagnosis was made by electrocardiographic and (or) vectorcardiographic analysis. However, as stated by Krikler *et al.*¹³ 'final proof requires electrophysiological techniques'. Therefore, it is possible to question some tracings (showing short PR intervals and very wide QRS complexes) which were attributed to WPW type B coexisting with left bundle-branch block either because 'after the endpoint of pre-excitation there was further prolongation of the QRS',²³ or because 'horizontal QRS loops with leftward and posteriorly oriented delta and maximal vectors were followed by additional mid-delays and slurrings'.²

In the light of recently acquired information they can be explained by assuming exclusive AV conduction through a right-sided accessory pathway, without necessarily postulating the concomitant existence of left bundle-branch block.¹⁶ This accessory pathway could be atrioventricular ('Kent') as well as fasciculoventricular ('Mahaim'). Electrophysiological verification is essential, because similar patterns can occur when the ventricles are activated almost simultaneously by impulses emerging from a right anterior (parietal or septal) accessory pathway and from the right bundle-branch (in cases where the left bundle-branch is completely blocked). This was shown in the studies of Latour and Puech²⁴ and Mendoza *et al.*²⁵ where simulated (catheter-induced) right septal pre-excitation was produced in patients with left bundle-branch block. The ventricular complexes resulting from iatrogenic right ventricular pre-excitation were as wide as (but of different morphology from) those occurring when sinus rhythm (with left-bundle-branch block) was present.

TACHYCARDIA-DEPENDENT (PHASE 3) LEFT BUNDLE-BRANCH BLOCK IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME

As far as we know there is only one reported case

where the authors postulated that this conduction disturbance might have been present in a patient with WPW syndrome. According to Krikler *et al.*¹³ the left bundle-branch block occurring in one of the cases (with WPW type A) reported by Pick and Fisch¹ was probably tachycardia-dependent. In our patient this diagnosis was possible because, regardless of the underlying electrophysiological mechanism, pre-excitation did not occur during sinus tachycardia (Fig. 1A). Furthermore, left bundle-branch block was not seen when pre-excitation was abolished with intravenous procainamide while the rate was slower (Fig. 1D). After development of persistent right bundle-branch block, the tachycardia-dependent (phase 3) left bundle-branch block naturally coexisted with the former, thus being manifested as infra-Hisian (bilateral bundle-branch) block in alternate beats (Fig. 2 and 3).

VARYING QRS MORPHOLOGIES

The patient discussed in this communication is unique, in that she had, at one time or another, ventricular complexes representing: (a) exclusive normal (AV node-His Purkinje) pathway conduction without bundle-branch block (Fig. 1D); (b) predominant or exclusive AV conduction through a right-sided accessory pathway (Fig. 3, right panel); (c) pure right bundle-branch block (Fig. 2 and 3); (d) pure left bundle-branch block (Fig. 1A); (e) fusion complexes resulting from the coexistence of (a) and (b) (Fig. 1B and C); and (f) fusion complexes resulting from the coexistence of (b) and (c) (Fig. 2 and 3). However, fusion caused by simultaneously occurring WPW type B and left bundle-branch block could not be identified, and this association still requires further electrophysiological evaluation.

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