CASE REPORTS

Arrival of Excitation at the Right Ventricular Apical Endocardium in Wolff-Parkinson-White Syndrome Type B

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SUMMARY

His bundle and bipolar right ventricular apex (RVA) and right ventricular outflow tract (RVOT) catheter electrograms taken from sites 1 mm apart were recorded simultaneously with various surface leads in two patients with Wolff-Parkinson-White syndrome (WPW) type B. In “fusion” beats the presence of a normal H-RVA interval indicated that the apex of the right ventricle was activated by the impulse emerging from the right branch. On the other hand, a shorter-than-normal H-RVA interval implied that the RVA was depolarized by the activation front propagating from the pre-excited site. When this occurred, the V-RVA intervals gave a rough estimate of conduction time from pre-excited area to RVA. The values obtained (40 and 50 msec, respectively) were shorter than in two other patients with WPW type A.

The arrival of excitation patterns in the right ventricular endocardium were similar in WPW type B and in beats produced by RVA stimulation but differed markedly from that of left anterior hemiblock even when the surface electrocardiographic leads showed abnormal left axis deviation in all instances. This resemblance between ventricular complexes attributed to WPW type B and those resulting from stimulation of an inferior (apical) site suggests, but does not prove, that the impulses propagated from an equivalent region of the right ventricle. These simultaneously recorded His bundle and right ventricular endocardial electrograms during electrical stimulation of the heart have increased our knowledge of Wolff-Parkinson-White syndrome.

Additional Indexing Words:

His bundle electrogram
Right ventricular apical electrogram
Fusion beats
Left axis deviation

THE COMBINED USE of the newer catheter techniques for intracardiac recording and electrical stimulation of the heart has provided important information regarding the mechanisms of arrival of excitation at selected recording sites.1-3 Since earlier studies from our department dealt only with normal intraventricular conduction and some aspects of bundle branch block in man, it appeared of interest to extend these observations to patients with Wolff-Parkinson-White syndrome (WPW) type B, as was suggested by Latour and Peuch in 1957.4

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Materials and Methods

Our technique for obtaining His bundle and “local” ventricular electrocardiograms has been discussed elsewhere.1-3 After explaining the procedure and obtaining consent of patients, catheter electrodes were introduced to record from the His bundle area, high right atrium (HRA), right ventricular apex (RVA), and right ventricular outflow tract (RVOT). Electrodes 1 mm apart were used to record filtered (40-500 Hz) bipolar electrograms and were recorded simultaneously with four or five surface leads at paper speeds of 100 mm/sec. Electrical stimulation of atria or ventricles was performed through an additional catheter electrode. In beats both with and without pre-excitation, “V” represented the onset of ventricular depolarization in whichever lead (surface or intracardiac) it occurred first, regardless of the pathway through which the atrial impulse reached the ventricles.1-3 Data from two patients with WPW type B were compared. Actual values and the significance of the different intervals measured are given in the corresponding case descriptions.

![Diagram](image)

**Figure 1**

Surface and intracardiac recordings in patient in Case 1 during exclusive conduction through the normal pathway with a left anterior hemiblock (LAH) pattern. In this and all figures, paper speed was 100 mm/sec. Values are expressed in msec. Electrograms from: His bundle (H); right ventricular outflow tract (RVOT); right ventricular apex (RVA). V = onset of ventricular depolarization, whichever lead it occurs first; St = electrical stimuli delivered to the high right atrium; H = His bundle activation.

Case Reports

Case 1

Additional information regarding this 63-year-old male with intermittent WPW type B has been presented elsewhere.5 Several types of QRS complexes were observed in this patient (figs. 1-3). Beats without pre-excitation (fig. 1) showing exclusive A-V conduction through the normal pathway had a qR pattern in lead I with superior and leftward deviation of the electrical axis. The ventricular complexes, measuring 95 msec, suggest left anterior hemiblock (LAH). The duration and significance of intervals measured from figure 1 are as follows.

A-H interval: 180 msec (normal values: 50 to 120 msec1-3). This interval represents A-V nodal conduction time. The fact that this beat was recorded during atrial stimulation probably explains the above normal A-H interval.

H-V interval: 45 msec (normal values: 35 to 55 msec1-3). When normal A-V conduction is present, this interval gives a measure of conduction time from His bundle to whichever ventricular site is depolarized first.2

![Diagram](image)

**Figure 2**

Surface and intracardiac recordings in patient in Case 1 during predominant (left) and exclusive (right) A-V conduction through the accessory pathway with a WPW type B morphology. HRA = high right atrium.
CONDUCTION PATHWAYS IN WPW TYPE B

H-RVA interval: 65 msec (normal values: 55 to 75 msec). This interval gives an estimate of conduction time from His bundle to right ventricular apex through the right branch.1, 2

V-RVA interval: 20 msec (normal values: 15 to 30 msec).2 When normal intraventricular conduction is present, this interval represents simply the arrival of excitation at an undetermined ventricular site and at the RVA.

RVA-RVOT interval: 10 msec (normal values: 10 to 20 msec).2 This interval represents differences in arrival of excitation at two right ventricular sites without necessarily implying linear conduction from one to the other.2

QRS complex: 90 msec.

The first P-R interval and QRS complex (with WPW type B morphology) in fig. 2 measured 120 and 130 msec, respectively. Inception of the forward H deflection at the onset of ventricular depolarization indicated that pre-excitation of the ventricles (through an accessory pathway) was almost simultaneous with activation of the His bundle (by an impulse traversing the A-V node). The H-RVA interval (30 msec) was shorter than normal (65 msec), a fact suggesting that the RVA was activated (from the pre-excited site) ahead of the impulse traversing the right branch. Nevertheless this beat was most probably a "combination" or "fusion" beat in which the ventricles were activated through both normal and accessory pathways as previously described6 since the second QRS complex (although similar) had a longer duration. The V-RVA interval was 40 msec. If the RVA was activated from the pre-excited site (as stated above), the V-RVA interval could be used as a rough measurement of conduction time from the pre-excited area to the RVA. The RVA-RVOT interval was 50 msec, a longer-than-normal interval indicating that differences in arrival of excitation at the recording electrodes were greater in beats without pre-excitation (10 msec, in fig. 1).

Surface and intracardiac recordings in the patient in Case 1 during right ventricular apical stimulation. The pacemaker stimulus artifact (St) produced considerable distortion of the RVA electrogram which, therefore, was not adequate for measurement. However, the St-RVOT interval gave a rough estimate of conduction time from the paced site (at the RVA) to the RVOT.

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Surface recordings in the patient in Case 2 during atrial fibrillation. The first and last QRS complexes resulted from exclusive or predominant accessory pathway conduction. The second and third beats (showing some degree of right bundle branch block in lead V1) resulted from exclusive or predominant normal pathway conduction. Left anterior hemiblock might also have been present.
In the second QRS complex, the His deflection was not seen. It was probably buried in the ventricular electrogram in which case it follows that this beat resulted from a greater degree of ventricular excitation by way of the wave front originating through the accessory pathway.\(^7\) Whereas the electrical axis was deviated to the left and superiorly, lead \(V_2\) showed a predominantly negative deflection. The H-RVA interval was not measured since His deflection was not seen. Other pertinent intervals in this beat were P-R (120 msec), V-RVA (40 msec), and RVA-RVOT (50 msec).

Figure 3 was obtained during right ventricular apical pacing. The wide (170 msec) ventricular complex with superior and leftward deviation of the electrical axis and a negative deflection in \(V_1\) resembled the one attributed to pure WPW type B conduction (fig. 2). The duration of the St (RVA)-RVOT interval (60 msec) was similar to that of the RVA-RVOT interval (50 msec) measured in the beat with WPW type B (fig. 2).

**Case 2**

The tracings in figures 4 and 5 were obtained from a 53-year-old male with WPW type B in which conduction through the normal pathway was recorded only during runs of atrial fibrillation (fig. 4). The first beat in fig. 5 was of sinus origin. The short (110 msec) P-R interval, wide (155 msec) QRS complex (with predominantly negative deflection in \(V_1\)), and the appearance of the forward H deflection 45 msec after the onset of ventricular depolarization supported the diagnosis of WPW type B.

The second beat had a wider QRS complex followed by an electrically-induced premature atrial impulse. The P-R interval had the same (short) value (110 msec) as in the first beat. Therefore, this second beat probably resulted from exclusive, or predominant accessory pathway conduction.\(^7\) Because of the A-H interval prolongation (100 msec as compared to 100 msec) related to normal A-V nodal refractoriness, the H deflection moved further into the QRS complex, and now appeared 65 msec (as compared with 45 msec for the sinus beat) after the onset of ventricular depolarization. The H-RVA interval was 5 msec and H deflection occurred after RVA during the premature beat. This interval was too short to imply linear conduction from H to RVA. Hence the RVA must have been activated from the pre-excited site. In both beats the V-RVA interval was 50 msec and gave a rough measurement of conduction time from pre-excited area (wherever it might have been) to the recording electrodes at the RVA.

*Surface and intracardiac recordings in the patient in Case 2 in beats with predominant, or accessory pathway conduction with a WPW type B morphology. The second (premature) beat was produced by electrical stimulation of the right atrium. In this patient a more proximal and a more distal His bundle electrogram was recorded.*
Discussion
Significance of the H-RVA and V-RVA Intervals in Beats with WPW Type B

Previous studies from our department have indicated that when A-V conduction occurred through the normal pathway, the H-RVA interval gave a rough estimate of conduction time from His bundle to RVA through the right branch (fig. 1).\(^1\)\(^2\) On the other hand, inscription of the H deflection after the onset of ventricular depolarization indicated that the ventricles were pre-excited through an extra-A-V nodal, extra-His bundle pathway (figs. 2 and 5).\(^6\) Whenever the latter finding was associated with a shorter than normal H-RVA interval (figs. 2 and 5) it was assumed that the RVA was not activated by the impulse emerging from the right branch (RB). In these beats the H-RVA “interval” was not a true interval representing conduction time from one site to another, rather it reflected the time elapsing between activation of His bundle (from A-V node) and of RVA (from pre-excited area). This occurred, at given moments of the cycle, when conduction through the A-V node was sufficiently delayed to allow the impulse propagating from the pre-excited area to reach the RVA ahead of the impulse traversing the RB. Consequently, the corresponding V-RVA interval could be used as an estimate of conduction time from the pre-excited area to RVA.

The V-RVA values observed in the two patients with WPW type B studied—40 msec (case 1) and 50 msec (case 2)—were shorter than those recorded from patients with WPW type A (120 to 160 msec)\(^8\). However, these findings do not preclude obtaining different values for other persons with WPW syndrome.

In theory, the distance between ventricular end of the bypass pathway and recording electrodes can be determined if the conduction time, conduction velocity, and intraventricular conduction pathways are known. Unfortunately, conduction velocity of ectopic right ventricular impulses has not been measured in the intact human heart. Values for the intraventricular septum and left ventricle have ranged between 440 mm/sec (in revived hearts)\(^9\) and 1200 mm/sec (in patients with two implanted ventricular pacemakers)\(^9\). In canine hearts Lewis found that conduction velocity through the ordinary muscle of the free right ventricular wall was approximately 400 mm/sec\(^11\).

Calculations made on the basis of the above values indicate that the distance between the site of emergence from the accessory pathway and the RVA could be as short as 18 mm or as long as 60 mm. The former value (18 mm) is appealing since conduction velocity along the right branch is at least three times the calculated speed within the ordinary muscle. Thus for RVA activation without the right branch, the length of the bundle would have to be greater than 60 mm.

Surface Electrocardiographic Morphology and Right Ventricular Endocardial Arrival of Excitation Pattern in WPW Type B

The surface electrocardiogram from one of the patients with WPW type B (fig. 2) resembled the one produced by RVA pacing (fig. 3). Both showed wide QRS complexes with superior and leftward orientation of the electrical axis and a deep S wave in lead V\(_1\).

Considering that the “electrical axis” (in the absence of right ventricular hypertrophy and extensive lateral wall myocardial infarction) predominantly reflects left ventricular potentials, the similar duration of electrical axis and RVA-RVOT and St(RVA)-RVOT intervals in figures 2 and 3 suggest that the sequence of activation was more or less the same through the left ventricular myocardium as contrasted with the sequence of right ventricular endocardial activation. On the other hand, the QRS complex showing LAH (fig. 1) had a similar (left and superior) axis deviation with a different (normal) sequence of right ventricular endocardial activation. These findings corroborate conventional electrocardiographic theory since changing but electrically silent right ventricular events (detected only by the catheter electrodes) are overpowered, at body surface, by the predominant left ventricular forces.

The resemblance between the QRS complexes attributed to WPW type B and those resulting from stimulation of an inferoapical right ventricular endocardial site suggests, but does not prove that the corresponding impulses propagated from the inferior regions of the right ventricle. The term “inferior” is used here as suggested by Barker, McLeod, and Alexander,\(^12\) “in reference to the cephalic and caudal aspects of the heart as it lies on the body rather than to apex and base.” These authors showed that a superior and leftward electrical axis can be elicited from the right ventricle from a rather large “inferior” area, which can be almost as large as the maximal value mentioned in reference to the distance between ventricular end of bypass and recording electrodes (60 mm). This statement can be corroborated by a

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careful study of the ectopic beats induced by the relatively widely spread “inferior” points 4, 5, 6, and 8 in figures 7a, b of the article by Barker et al.12

**Importance of Catheter Recordings**

Recent studies using catheter electrodes have dealt mainly with the electrical activity of the specialized conducting system. Although previous investigators had been concerned with the arrival of excitation at several ventricular sites, their technique stressed the value of intrinsicoid deflection as recorded by nonfiltered unipolar leads,4 which generally are considered to be less accurate than filtered bipolar leads with relatively short interelectrode distances. Nevertheless, the information obtained with the use of the latter (as presented in this paper) still requires confirmation by direct epicardial or endocardial bipolar recordings to determine its value.

**References**

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