Preexcitation and Tachycardias in Wolff-Parkinson-White Syndrome, Type B

A Case Report

By John W. Lister, M.D., Francis X. Worthington, Jr., M.D.,
Thomas O. Gentsch, M.D., John A. Swenson, M.D.,
David A. Nathan, M.D., and Arthur J. Gosselin, M.D.

SUMMARY
Electrophysiologic events in a 52-year-old man with Wolff-Parkinson-White (W-P-W) syndrome, type B, recurrent supraventricular tachycardias, and coronary artery disease were studied during cardiac catheterization and at open-heart surgery.

During cardiac catheterization reciprocal tachycardias were repeatedly initiated by premature atrial beats and terminated by rapid right atrial pacing. Our results confirm that the tachycardia usually seen in the W-P-W syndrome is reciprocal tachycardias with orthograde conduction to the ventricles through the normal atrioventricular conduction system and retrograde conduction to the atria via the Kent bundle.

The epicardial surface of the ventricles was “mapped” at surgery. The earliest site of excitation was the posterior base of the left ventricle near the crux of the heart. Kent bundle conduction was temporarily ablated with lidocaine (Xylocaine) hydrochloride.

This is the first case of W-P-W syndrome, type B, in which the anomalous A-V bundle entered the left ventricle. Our results indicate that the analysis of the electrocardiogram in localizing an abnormal A-V connection cannot be relied upon completely.

Additional Indexing Words:
Kent bundle  Atrial stimulation  Epicardial “mapping”
Ventricular activation sequence  Reciprocating tachycardias

The pathophysiology of the Wolff-Parkinson-White (W-P-W) syndrome and associated tachycardias is currently under intensive investigation. Application of new technics of His bundle recordings, “mapping” of ventricular activation times, and surgical attempts at interruption of the Kent bundle have contributed much toward clarifying the sequence of electrophysiologic events in the preexcitation syndromes.

Presented below are electrophysiologic data obtained from a patient with the W-P-W type-B syndrome. This information will help in the further elucidation of the mechanisms of recurrent supraventricular tachycardia and the variability of ventricular preexcitation.

Case Report

A 50-year-old man with known W-P-W syndrome was admitted to the hospital for evaluation of anginal pain. He had sustained a myocardial infarction 4½ years previously. After recovery, he was symptom-free for 3 years when he experienced his first anginal attack. This progressed until pain occurred at rest, during sleep, and with minimal exertion or anxiety.

For a prolonged period prior to his infarction, the patient had had frequent bouts of supraventricular tachycardia lasting from 5 min to 24 hours. During the year preceding his admission...
for evaluation, this tachycardia had been associated with severe angina and necessitated cardioversion on two occasions. Pharmacologic attempts at its control were unsuccessful.

Examination of the precordium revealed the apical impulse in the fifth intercostal space at the midclavicular line. The first heart sound was normal. The aortic and pulmonic component of the second sound were also normal and split physiologically. An S₄ was present at the apex. The remainder of the physical examination, including the chest X-ray, was unremarkable.

The ECG findings fulfilled all the criteria for the W-P-W type-B syndrome. The P-R interval was less than 0.12 sec, there was a prominent delta wave, and the QRS interval was greater than 0.12 sec. As interpreted from the ECG, the delta-wave vector was oriented to the left, superiorly and anteriorly (fig. 1).

Cardiac catheterization revealed hypokinesis of the anterior wall of the left ventricle and a left ventricular end-diastolic pressure of 18 mm Hg. There was a 90% occlusion of the left anterior descending coronary artery and an 80% occlusion of the obtuse marginal branch of the circumflex coronary artery.

Saphenous vein aortocoronary artery bypass surgery was performed with separate vein bypass graft to the distal circumflex and left anterior descending arteries. Since surgery the patient has had no further anginal episodes.

**Investigative Methods**

*His Bundle Recordings and Arrhythmia Studies at Cardiac Catheterization.* During cardiac catheterization, shortly after coronary angiography, three bipolar electrode catheters with electrodes 1 cm apart were positioned in the right heart: (1) to record the low right atria (LRA) and His bundle (BH) bipolar electrograms; (2) to record the high right atrial (HRA) bipolar electrogram; and (3) to stimulate the atria electrically. The catheter through which the LRA and BH

---

**Figure 1**

A 12-lead ECG typical of the W-P-W syndrome, type B. The P-R interval is less than 0.12 sec; the QRS duration is greater than 0.12 sec; the mean electrical axis of the delta wave is depicted to the left, superiorly and anteriorly; while the mean QRS vector is directed to the left superiorly and posteriorly. Paper speed 50 mm/sec. Time lines 1 sec. Abbreviations for all figures: HRA = right atrial electrogam; LRA = low right atrial electrogram; BH = bundle of His electrogam; A-A = intraatrial conduction time; A-H = conduction time from LRA to BH (approximate A-V nodal conduction time); H-V = interval from BH activation to onset of ventricular activation; and V = ventricles.
electrograms were obtained was positioned adjacent to the septal leaflet of the tricuspid valve. The HRA electrogram was obtained through a catheter placed in the right atrium near the entry of the superior vena cava. The catheter used for atrial stimulation was also situated high in the right atrium. Recording catheters were connected to a distribution box which was in turn attached to a multichannel oscillographic photographic recorder. Tracings were obtained at a paper speed of 50 or 100 mm/sec. The atrial-stimulation catheter was connected to a paired pulse generator (Medtronic model 5837). For the placing of premature beats, a specially designed instrument was employed, designed and built by the Biomedical Engineering Department of the Miami Heart Institute. The methods used for recording the BH electrograms have been described elsewhere.  

Epicardial "Mapping" at Surgery. Prior to coronary artery bypass surgery, fixed close bipolar plaque electrodes with five silver contacts 2–5 mm apart were sutured to the epicardial surface of the sinus node and on the high lateral posterior epicardial surface of the right ventricle. An L-shaped bipolar roving electrode was used to record electrograms from 10 predetermined points on the ventricular epicardium. One cc of 1% lidocaine (Xylocaine) hydrochloride (10 mg) was injected into the ventricular myocardium at

![Image of a figure showing electrocardiogram tracings]

**Figure 2**

Premature A-V junctional beats. Simultaneous recordings of bipolar high right atrial (HRA), low right atrial (LRA), and bundle of His (BH) electrograms and lead I ECG. The first, second, fifth, sixth, and seventh beats are typical W-P-W beats (fusion beats) and exhibit delta waves. The third and fourth beats are premature A-V junctional beats which arise above the BH recording site and are solely conducted to the ventricles through the normal A-V conduction system. In the beats which exhibit ventricular preexcitation the BH is activated simultaneously with the delta wave, and the A-H time is normal. In the premature A-V junctional beats, the H-V time is normal, and the interval between the preceding sinus beat and BH deflection is too short to permit normal A-V conduction. Therefore, these are dissociated beats. The sinus rhythm is undisturbed throughout the record. Paper speed 50 mm/sec. Time lines 1 sec.

*Circulation, Volume XLV, May 1972*
Figure 3

(A) Initiation of a reciprocal tachycardia by a paced premature atrial beat. The first and second beats are typical W-P-W beats. They exhibit a short P-R interval, and the His bundle is simul-
the point of earliest ventricular activation. The sinus node electrogram became the reference point in determining the ventricular activation times. The leads from the electrodes were connected to a distribution box which was, as before, attached to a multichannel oscillographic photographic recorder. Electrograms used to plot epicardial activation times were obtained at a paper speed of 200 mm/sec while rhythm strips were recorded at 50 mm/sec. The detailed steps for recording epicardial electrograms have been previously described.16, 17

Results

Arrhythmia Studies at Cardiac Catheterization. During normal sinus rhythm, preexcitation of the ventricles was constantly present. The intracardiac electrograms and ECGs during sinus rhythm were typical of the W-P-W syndrome (figs. 1-3; see also fig. 5). The sequence of atrial activation and the A-H time were normal. The His bundle deflection occurred simultaneously with the delta wave on the ECG, and the temporal relationship between the BH and ventricular deflections on the LRA and BH electrogram were shorter than normal (35-55 msec)8 (figs. 2, 3). Occasionally, A-V junctional beats originating proximal to the BH recording site occurred spontaneously (fig. 2). These beats invariably exhibited a narrowed QRS complex (normal ventricular activation) and normal H-V times. The typical reciprocal supraventricular tachycardia (RSVT) was repeatedly initiated by a properly timed premature atrial beat (PAB) and terminated by atrial pacing (× 8) (fig. 3A). The PABs which initiated the RSVT were placed 285-305 msec after the previous sinus beat and exhibited a markedly prolonged A-H time. They were blocked at the anomalous atrioventricular pathway and entered the ventricles via the normal atrioventricular conduction system (AVCS). In every instance ventricular activation during the RSVT occurred via the normal AVCS, and there was 1:1 ventriculoatrial conduction (retrograde atrial activation) which was transmitted through the anomalous atrioventricular connection. The RSVT was terminated by atrial pacing at the slightly greater rate than the intrinsic rate of the RSVT. In each instance it required the apparent complete pacemaker capture of two atrial beats to terminate the RSVT. An example of termination of the tachycardia is depicted in fig. 3B.

Epicardial "Mapping" at Surgery. This patient's 12-lead ECG is typical of the W-P-W type-B syndrome (fig. 1). The early sequence of ventricular activation during normal sinus rhythm and ventricular preexcitation is shown in figure 4. The duration of the delta wave on the ECG is 70 msec. The first point on the
Epicardial “map” of activation obtained at cardiac surgery. The anomalous A-V bundle enters the left ventricle approximately 1 cm to the left of the posterior descending coronary artery. The area encompassed by the broken line approximates the area of the ventricular preexcitation. The first point activated on the ventricular epicardial surface was excited 113 msec after the sinoatrial node and was simultaneous with the onset of the delta wave on the ECG. From this point it appears that activation spread slowly across the posterior base of both ventricles. The slow spread of activation during the delta wave of the ECG suggests muscle conduction. One cc of 1% lidocaine (Xylocaine) hydrochloride was injected into the myocardium and almost immediately abolished ventricular preexcitation (see fig. 5). SAN = sinoatrial node; FE = fixed.

Circulation, Volume XLV, May 1972
epicardial surface of the ventricles to be activated was situated at the posterior base of the left ventricle, approximately 1 cm to the left of the posterior descending coronary artery and just below the A-V groove. This area was activated 113 msec after depolarization of the sinoatrial node and was simultaneous with the onset of the delta wave on the ECG. Although the impulse entered the left ventricle through the anomalous A-V pathway, the spread of activation was such that the posterior basal portions of both ventricles showed early activation. In figure 4, the epicardial area activated during the delta wave of the ECG was assumed to be the area of ventricular preexcitation. To further document the site of entry of the anomalous A-V pathway into the left ventricle, 1 cc of 1% Xylocaine hydrochloride (10 mg) was injected into the left ventricular myocardium at the point of earliest activation (fig. 4). This procedure almost immediately eliminated ventricular preexcitation, and inverted the rhythm. Three minutes after lidocaine administration, there was an A-V junctional rhythm with an apparently normal QRS and 1:1 retrograde atrial activation (fig 5A and B). Five minutes later, normal sinus rhythm with exclusive conduction through the normal AVCS ensued (fig. 5C). Approximately 2½ hours later normal sinus rhythm with ventricular preexcitation recurred. No attempt was made to transect the anomalous A-V conduction pathway because of its precarious anatomic location in the immediate vicinity of the crux of the heart and the normal AVCS.

Discussion

As early as 1926, de Boer18 proposed that tachycardias in patients with anomalous A-V connections may be caused by a circus movement. If antegrade conduction is only possible in the normal AVCS because of temporary refractoriness of the Kent bundle, normal excitation of the ventricles will occur with activation of their basal part at a time when the Kent bundle is no longer refractory. This will be followed by retrograde activation of the atria via the Kent bundle.

Clinical evidence in favor of retrograde conduction through an accessory pathway during RSVT was first presented by Wolf19 and Harnischfeger.20 The first experimental data to support the above hypothesis derived from the work of Butterworth and Poindexter.21 These investigators produced tachycardias in the feline heart by retrograde atrial stimulation with an amplified QRS signal. In recent years electrical stimulation of the heart,2, 19 His bundle recordings,3, 4 and the “mapping” of the sequence of epicardial activation,4, 6, 14 have been used to study in detail the preexcitation syndrome. All the observations made with these newer techniques, as well as our findings, support the earlier concepts that in the W-P-W syndrome the tachycardias are reciprocal supraventricular rhythms, usually with orthograde ventricular activation through the normal AVCS and with retrograde conduction to the atria via the accessory pathway.

Massumi10 reported a case in which simultaneous block occurred in the normal AVCS (bilateral bundle-branch block) and in the Kent bundle. He proposed that in his patient the AVCS and the Kent bundle were in close proximity to each other. In our patient, the close anatomic location of the normal AVCS and the anomalous A-V connection have been documented by direct observations (epicardial “mapping” and blocking of the Kent bundle with lidocaine hydrochloride). In our case, in the absence of tachycardia, there appeared to be a complete functional separation of the Kent bundle and normal AVCS. During preexcitation, fusion beats with normal conduction across the A-V node were present.

---

table: reference electrode; RE = roving electrode; AUE = atrial unipolar electrogram recorded from SAN electrode; ECG = lead I ECG; and A through J = electrograms recorded from corresponding points shown on map. The SAN ECG was used for the reference point (0 point) in determining the activation time of the ventricular epicardium. Records were obtained at 200 mm/sec.

Circulation, Volume XLIV, May 1972
Figure 5

(A) Termination of ventricular preexcitation by administration of Xylocaine hydrochloride. After the second beat, 1 cc of 1% Xylocaine hydrochloride was injected into the myocardium at the point of earliest ventricular activation (see fig. 4). The subsequent four beats show a progressive diminution in the magnitude of the delta wave of the QRS. From the fifth beat on, there is an A-V junctional rhythm during which all the beats exhibit A-V dissociation except the last, which shows retrograde atrial activation. In each beat, except the last, the SAN is activated prior to the LA. In the last beat, the SAN and LA are activated simultaneously documenting that there has been a sudden change in atrial activation (retrograde atrial excitation). Paper speed 50 mm/sec. Time lines 1 sec. (B) At 3 min after Xylocaine hydrochloride administration. There is an A-V junctional rhythm with 1:1 retrograde atrial activation. The retrograde atrial excitation is documented by the simultaneous activation of the LA and
Occasionally, when the QRS normalized, the beats arose in the A-V junction above the His bundle recording site and were conducted to the ventricles solely through the normal AVCS (fig. 2). However, our observations indicate that during RSVT the reentry pathway was short. In figure 3B, the first beat which appeared to capture the entire atrium “peeled” back the refractory period so that the next beat could penetrate to a deeper level and interrupt the reentry pathway of the tachycardia. This suggests that either the atrium is excluded from the reentrant pathway or only a small portion of the atrium is involved.

In previous reports of the W-P-W type-B syndrome,4,14 studied at open-heart surgery, the anomalous A-V conduction pathway entered the lateral margin of the right ventricle. Although the delta wave in our subject had the same electrocardiographic spatial orientation as the case studied by Burchell,4 the entry of the anomalous A-V connection into the ventricles was not at the lateral margin of the right ventricle but at the basal posterior medial area of the left ventricle (fig. 4). Lev and his associates1 carefully studied a case of W-P-W type-B syndrome at postmortem in which no atrioventricular bridges were found. They explained their patient’s ECG by the finding of atrial septal muscle fibers bypassing the A-V node and the existence of numerous Maheim fibers extending between the A-V node and the posterior portion of the ventricular septum. In our case, the incongruity of the ECG findings of W-P-W type-B syndrome and the anomalous A-V connection entering the left ventricle near the crux of the heart may best be explained by the vector resulting from the slow spread of depolarization (muscle conduction) during the period of the delta wave and/or Maheim fibers entering the right side of the ventricular septum or right ventricular endocardium. These Maheim fibers if present, could not be detected by our method of ventricular “mapping” because we only explored the epicardial surface of the heart. Since publication of the successful treatment of RSVT in patients with preexcitation syndrome by transection of anomalous A-V bundles14 or of the normal AVCS,8 the determination of the exact location of the anomalous A-V connecting pathway has assumed clinical importance. The analysis of the ECG in localizing the abnormal A-V connection cannot be relied upon completely. This is the first reported case of the W-P-W type-B syndrome in which the deviant A-V connection did not enter the right ventricle.

Acknowledgments

We wish to acknowledge Dr. Minor Duggan for his editorial assistance in the preparation of the manuscript and Mrs. Klara Soos for her skilled typing help.

References


SAN. (C) At 8 min after Xylocaine hydrochloride administration. There is normal sinus rhythm with a normal P-R interval and normal ventricular excitation. These tracings demonstrate that Xylocaine only blocked the anomalous A-V conduction pathway, and at the point of entry into the ventricle the normal A-V conduction system and anomalous A-V connection are anatomically separate.


Preexcitation and Tachycardias in Wolff-Parkinson-White Syndrome, Type B: A Case Report
JOHN W. LISTER, FRANCIS X. WORTHINGTON, JR., THOMAS O. GENTSCH, JOHN A. SWENSON, DAVID A. NATHAN and ARTHUR J. GOSSSELIN

Circulation. 1972;45:1081-1090
doi: 10.1161/01.CIR.45.5.1081

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/45/5/1081

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/