Atrioventricular and Ventriculoatrial Excitation in Wolff-Parkinson-White Syndrome (Type B)

Temporary Ablation at Surgery

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SUMMARY

A patient with an atrial septal defect, paroxysmal tachycardia, and the Wolff-Parkinson-White syndrome (type B) had epicardial exploration to determine the nature of the excitation anomaly. Right bundle-branch block in association with the WPW syndrome (type B) was evidenced by the late activation (0.12 sec) of the epicardium over the outflow tract of the right ventricle. Early activation of the base of the right ventricle (near the atrioventricular groove at the right border of the heart) was interpreted as indicating an actively conducting atrioventricular muscle bridge (bundle of Kent) in this region. During paroxysms of tachycardia, the ventricular area excited much later than when sinus mechanism was present, and the adjacent right atrium was excited in sequence. This sequence supported the concept of a circus movement, that is, movement from atrium to ventricle via atrioventricular bundle (His) and ventricle to atrium via a muscle bridge (Kent). Injection of procaine into the base of the right ventricle abolished the pre-excitation of the ventricle.

Additional Indexing Words:
Paroxysmal tachycardia
Bundle of Kent
Congenital heart disease
Atrial septal defect

Durrer AND Roos1 have reported the pattern of epicardial excitation of the ventricles in a patient with Wolff-Parkinson-White syndrome (type B). The dominant QRS vectors in this variety of WPW, directed principally to the left and posteriorly, would fit neatly into the concept that an atrioventricular muscle bridge (bundle of Kent) at the right border of the heart was the anatomic base for the electrocardiographic abnormality. The case reported by Wood and associates,2 in which a bundle of Kent was demonstrated and believed to be responsible for the electrocardiographic display in the WPW syndrome, was similar to that reported by Durrer and Roos.1 The muscle bridge in the case of Wood and associates was in the proper location to produce the aberration in the electrocardiogram, and a similarly located atrioventricular bridge could cause the right ventricular excitation in the case reported by Durrer and Roos. Such a muscular bridge (bundle of Kent) cannot be held responsible for all cases of WPW syndrome, as evidenced by a case carefully studied by Lev and associates3 in which no atrioventricular bridges were found. They explained the unusual sequence of electrocardiograms by the finding of fibers of atrial septal muscle bypassing the A-V node, and additionally, unusually copious Mahaim fibers from the A-V bundle to the posterior portion of the muscular ven-

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Figure 1

(A) Routine electrocardiogram. Recording speed 50 mm/sec. (B) X, Y, and Z leads. (C, D, and E) Vectorcardiogram as computer plotted from X, Y, and Z modified Frank system (Mayo).

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tricular septum. In addition, the occurrence of WPW syndrome on catheterization of the right ventricle, as, for example, reported by Kossmann and associates, suggests that other mechanisms may be responsible for pre-excitation, specifically a possible nascent ventricular pacemaker from the catheter moved by atrial contraction. In the isolated dog heart, pre-excitation was observed by Burchell, and he believed the data pointed to the A-V bundle (His) area as the locus of the problem in this instance. The report of Durrer and Roos gave further support to the viewpoint that the WPW syndrome might be surgically ablated, if an aberrant A-V pathway could be identified by electrophysiological means.

When surgery was recommended for a patient with an atrial septal defect, who had in addition a WPW aberration similar to that of the patient investigated by Durrer and Roos, a concomitant exploration of epicardial excitation was also planned with the possibility of surgically modifying the WPW syndrome.

Preparing the protocol, we categorized the approach as a feasibility study and believed that a simple multichannel type of electrocardiographic recorder and unipolar exploring electrodes would be adequate for our purposes. Some hazard related to direct exploration of the heart from induced alternating current was acknowledged but, with awareness of the hazard, it was believed that any complication could be readily managed. A second hazard contemplated was the possibility of complete loss of A-V conduction, owing to a block in the normal A-V conduction pathways (His bundle) which would be unmasked by surgical interference of the pre-excitation pathway (Kent bundle). The patient's disability from the tachycardia was not such that the production of complete heart block and implantation of an electronic pacemaker would have been justified.

**Report of a Case**

A 43-year-old man was seen at the Mayo Clinic in August 1966 with the main complaint of episodes of rapid heart action. The first paroxysm had occurred 20 years previously, but the episodes had occurred several times a month for about 4 years. He had been admitted twice to a hospital because of the attacks, but the overall disability was slight, and he was able to fulfill his duties as a mail carrier, walking 6 to 8 miles daily. The most successful therapeutic procedure for an attack, in his opinion, was to take a barbiturate sedative, and when he awoke from the induced sleep, the heart rate would be normal. He believed that digitalis given prophylactically during a year had decreased the number of attacks, whereas quinidine taken for 3 weeks had seemed to increase the frequency.

On examination, the precordium was moderately overactive, owing presumably to an accentuated right ventricular pulsation. There was an ejection murmur of grade II and moderate duration to the left of the upper sternum. The second sounds were of normal intensity with very narrow splitting.

The electrocardiogram (fig. 1) showed a classic pattern of the WPW syndrome of type B. Analysis of the vectorcardiogram showed the initial (delta) vector to be oriented to the left, superiorly, and anteriorly. This suggested early excitation of the right ventricle proceeding from the atrium to the ventricle at the right lateral border of the heart.

<table>
<thead>
<tr>
<th>Cardiac Catheterization</th>
<th>Superior vena cava</th>
<th>Inferior vena cava</th>
<th>Right atrium</th>
<th>Pulmonary artery</th>
<th>Femoral artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂ saturation, %</td>
<td>86</td>
<td>89</td>
<td>92</td>
<td>94</td>
<td>96</td>
</tr>
<tr>
<td>Pressures, mm Hg</td>
<td>—</td>
<td>—</td>
<td>10/2</td>
<td>30/8</td>
<td>130/70</td>
</tr>
</tbody>
</table>

The points on the vector loops are at 10-msec intervals, the delta wave being mainly inscribed between 0 and point 9. (Courtesy of Dr. Ralph E. Smith.) It will be recognized that the sagittal component, recorded as the scalar record Z, has inverted polarity to the recommendations of the AHA Committee on Electrocardiography.6

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The primary electrophysiological objective at thoracotomy was exploration of the right ventricle below the A-V groove to determine the time of activation (depolarization). Unipolar records were obtained from epicardial points according to a prepared chart (fig. 2). The results (figs. 3 and 4) revealed that, at point 5, at the extreme right border of the right ventricle, there was premature excitation of the right ventricle. During the time when the heart was exposed by thoracotomy, frequent short paroxysms of tachycardia at a rate of approximately 150/min occurred, during which there was reversal of the excitation direction, the right atrial excitation apparently following the excitation of the adjoining right ventricle. On several occasions, pressure on the right ventricle next to the A-V groove at point 5 terminated the tachycardia. This result was not obtained when the pressure was exerted anterior to point 5. During the tachycardia, the standard leads and chest leads of the electrocardiogram changed their form to that of a right bundle-branch block which fitted the finding of a delayed excitation of the base of the right ventricle (fig. 5). The onset of tachycardia was heralded by a premature atrial contraction with a P wave that was negative in leads II and III, the peak of which was 0.42 sec after the peak of the preceding sinus P wave. The inverted P wave was followed by a ventricular complex after a P-R of 0.22 sec, and the tachycardia ensued. Cessation of the tachycardia was recorded once and was associated with P wave upright in leads II and III, with its peak 0.13 sec after the peak of the preceding R wave, which was followed by an aberrant QRS after a short P-R (0.09 sec).

A solution of 1% procaine injected into the right ventricular muscle at area 5, presumably the site of a Kent bundle, was followed by disappearance of the pre-excitation of the right ventricle. With the disappearance of the WPW phenomenon, there was initially an atrioventricular dissociation, with ventricular rate (94/min) more rapid than the atrial rate (78/min). After a few minutes of observation, a drop of epinephrine (1/1,000) was placed on the right atrium at the base of the superior vena cava, the objective being to increase the rate (and establish the dominance) of the sinus node pacemaker. Sinus tachycardia (142/min) transiently ensued with the capture of the ventricle, the P-R interval being 0.26 sec. In the hope of permanently interrupting the Kent bundle, a transverse cut, 1 cm in length, was made on the inside of the right atrium, close to and parallel to the atrioventricular ring. This was accomplished while the atrioventricular conduction was still normal after the procaine injection. At the end of the intracardiac repair, the cardiac mechanism was by an atrial pacemaker with a

**Figure 2**

Diagram, as utilized in operating theater, for identification of points of exploration by unipolar electrode. Values for excitation are given in reference to earliest intrinsic deflection (marked .000) on right border of right ventricle near groove. Measurements are between sites on base-line intercept by sharp intrinsic deflection. The rectangle enclosing CO refers to excitation deflection at zero time within the right ventricular cavity.

A roentgenogram of the chest showed moderate cardiac enlargement and moderate enlargement of the pulmonary vascular shadows.

The absence of prominent and fixed splitting of the second sound (confirmed by phonocardiography), which is unusual in the patient with atrial septal defect with a large shunt, was considered possibly related to the accelerated excitation and contraction of the right ventricle.

The data on oxygen saturation and pressures can be seen in table 1. The systemic blood flow was 10.9 L/min (systemic flow index of 5.6), and the pulmonary blood flow was large (left-to-right shunt estimate of 60%). The onset of P wave to the foot of the right ventricular pressure rise was 0.20 sec; from the delta wave onset, 0.13 sec. Electrocardiograms, taken after mild exercise and with carotid sinus pressure, did not vary in any detail from the original one.
normal P-R interval, without evidence of ventricular pre-excitation (fig. 6). However, just after the final closing of the chest, the electrocardiogram showed a return of the WPW complexes. The postoperative period was without complications. The day after dismissal from hospital the patient reported that he had had a short episode of tachycardia.

**Discussion**

Our study indicates the feasibility of a surgical approach to the treatment of some varieties of the pre-excitation (WPW) syndrome. The occurrence of A-V dissociation (as dissociation when the nodal rate is higher than the sinus rate) with disappearance of ventricular pre-excitation in this patient after pharmacological block is not considered an integral part of the response to interruption of Kent conduction and is a common phenomenon. With the recording equipment utilized, we have reported “time-of-excitation”
The paroxysms of tachycardia during the operation had an equivalent rate to those that had occurred prior to operation—approximately 150 beats per minute, or a cycle length of approximately 400 msec (0.4 sec). The presence of a circus movement would fit the observations, and some of the components of the time period could be supplied; namely, 50 msec from ventricle to atrium (Kent pathway), 200 or more msec from atrium to ventricle (P-R interval, His bundle), and 70 msec or more for right ventricular excitation (onset of R wave to base of right ventricle), and a remainder of 80 msec for conduction along an intra-atrial pathway.

The observation of the delayed activation of the epicardium overlying the right ventricular outflow tract despite the early excitation of the base of this ventricle is of particular pertinence to the questions as to whether WPW of type B can coexist in the right bundle-branch block. Despite the late activation of the epicardial area over the outflow portion of the right ventricle, the right ventricular cavity potential was without a prominent initial R wave; this indicates that the pre-excitation had gained access to the right bundle ramifications but that propagation was blocked to the outflow portion of the ventricle. This patient thus appeared to have delayed excitation in the right bundle ramifications whether the WPW mechanism was present or absent.

Extensive discussions have occurred pertaining to the possibilities of coexistent right bundle-branch block and WPW of type B. Our studies indicate that the association may be present and not be manifest in the surface potentials of the body. Such a view does not necessarily conflict with the observation of Gamboa and associates, who found that electrically induced right bundle-branch block in man with the WPW syndrome did not modify the end of ventricular activation.

For a brief period in the electrocardiographic records of our patient in the early operative period, there was a sequence of nodal rhythm, with a R-P period (Fig 7) and absence of any pre-excitation. The latter effect
EXCITATION IN WPW SYNDROME

Figure 5

Representative records during episode of tachycardia; standard leads in panel on left. Unipolar electrograms from right atrium and from site 5 on ventricle in panels in middle and on right, respectively. Vertical line has been drawn on middle and right panels to facilitate demonstration of relative timing of the excitation of adjacent atrial and ventricular sites. It is believed that excitation spread retrograde into atrium for reasons discussed in text and this was not a nodal tachycardia.

Figure 6

Electrocardiogram taken after repair of heart and resumption of its normal function, illustrating absence of pre-excitation. P vectors indicate an anomalous site of the intrinsic atrial pacemaker.

of “normalization” would be an obvious result of nodal rhythm, as pointed out years ago by Öhnell and by Rosenbaum and associates, if pre-excitation were related to an atrioventricular (Kent) pathway. Our observations give further support to the generalizations of

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Pick and Fisch, who pointed out how pre-excitation may obscure the electrocardiographic pattern of bundle-branch block if the chamber with the interrupted bundle is activated prematurely. However as Robertson and associates have demonstrated, if the right bundle-branch block results in very late activation of part of the right ventricle, the late vectors still may indicate right bundle-branch block despite pre-excitation of type B.

The protocol included plans for recording potentials from within the left ventricle, but the electrode was too stiff to be guided into it. The lack of such records prevents a conclusion concerning the mechanism of excitation of the whole heart, but we suspect that excitation was taking place entirely through the anomalous pathway. Further evidence for this supposition was afforded by the configuration of the QRS complexes (V-7) of premature ventricular beats produced occasionally by pressure of the electrode on area 5, the configuration being virtually identical to the QRS of the WPW complexes. This mechanism was suggested to be occurring by Kossmann and Goldberg in their case from measurements of the total P-QRS interval. Their case, also of WPW syndrome of type B, demonstrated disappearance of splitting of the second sounds when WPW complexes occurred.

March and associates were hesitant to make any generalization concerning the mechanical consequences of anomalous excitation in the two cases they clearly illustrated, but in one (type B) pulmonary closure preceded the aortic closure, and in the other (type A) the reverse was true. It should be noted that in our case there was no demonstrable right ventricular pressure rise associated with the pre-excitation potentials. Ferrer and associates studied two cases of the WPW syndrome in which the anomalous conduction was present and, when tachycardia was present, found no evidence of ventricular asynchronous-
ity. The tachycardic mechanism was believed to be nodal, with P waves demonstrated by an intra-atrial electrode, but inspection of their records would lead us to believe that the mechanism could have been a circus one, with a cycle of approximately 0.3 sec.

In retrospect, with the recurrence of WPW after closure of the chest, the incision designed to transect the bundle of Kent seems unduly timid, but may be defended because the patient was not disabled by his episodes of tachycardia and one could neither jeopardize the results of the operation recommended for the cure of the atrial septal defect nor risk the remote chances of complete heart block. The intra-atrial surgical incision was believed at the time to be safer than a ventricular epicardial incision, the former being further away from the right coronary artery, which was difficult to visualize in the A-V groove. It will be our intention in any future similar operation to localize the anomalous bundle by the epicardial electrocardiographic tracings, avoid pharmacological block, and attempt ablation of the anomalous pathway of conduction by completely separating the atrial wall from the ventricle by an incision near the tricuspid ring in the appropriate area, which may be extended about 2 cm on either side of the suspected anomalous bundle, and which would then be repaired by suturing.

In essence, our investigations support the mechanisms of pre-excitation and of tachycardia in some patients with the WPW syndrome as early conjectured by Holzmann and Scherf,15 illustrated by Wolferth and Wood,16 and supported by the observations of Hamischfeger.17

References


Stability of Bundle-Branch Block (31 years)
Simultaneous ECG Leads (1925)

Record of an electrocardiogram by means of a capillary electrometer taken on June 15, 1894, i.e. before the construction of the string galvanometer. Lead I.

The same patient but investigated 31 years later with three leads simultaneously. The disease has remained unchanged. One notes the similarity of the curves taken with the capillary electrometer and the galvanometer with Lead I.

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