Pre-excitation with Normal PR Intervals
A Case Secondary to Slow Kent Bundle Conduction*

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His bundle electrograms were recorded in a patient with pre-excitation and normal PR intervals to determine whether conduction was via anomalous A-V connection (Kent bundle) or infranodal bypass (Mahaim tract). During sinus rhythm (62/min) both P-delta and P-H measured 155 msec. With atrial pacing at 70 and 75/min, P-H prolonged while P-delta remained constant. At 80/min, the anomalous pathway failed and the QRS normalized. The effective refractory period (ERP) of the anomalous pathway was 700 msec. In summary, the normal PR interval represented slow Kent bundle conduction. Additional findings of depressed Kent bundle conduction included a prolonged ERP and block at a slow paced rate, implying that tachycardia would be unlikely. In WPW, the functional properties of both normal and anomalous pathways can be determined by His bundle recording and atrial pacing.

The classic electrocardiogram in Wolff-Parkinson-White syndrome (WPW) is characterized by a PR interval of 0.12 sec or less, a QRS duration of 0.12 sec or greater, and initial slowing of the QRS (delta wave). Current evidence strongly suggests that the usual case of WPW relates to the presence of an anomalous A-V connection (Kent bundle) bypassing the A-V node and inserting directly into one or the other ventricles. Additional accessory pathways, other than the classic Kent bundle have recently been implicated. Short PR intervals with normal QRS may be explained by “James tracts” bypassing the A-V node but re-entering the His bundle. The presence of delta waves with normal PR intervals may be explained by Mahaim tracts, exiting from the His bundle and inserting into the ventricular septum.

In the present communication, we describe a patient with a normal PR interval and pre-excitation. Electrophysiologic studies suggested that slow Kent bundle conduction and not Mahaim conduction was responsible for the patient’s unusual electrocardiogram. The clinical and physiologic implications of these findings are discussed.

CASE REPORT
A 25-year-old man was admitted to the Veterans Administration West Side Hospital for evaluation of recurrent episodes of dizziness and headaches. Physical examination and routine laboratory evaluation were within normal limits. Neurologic investigation revealed no evidence of central nervous system disease.
Atrial excitation. The P-delta interval, from the onset of the P wave supplied by a battery powered coupled pulse generator (Medtronics 5837) in both the demand and coupled modes. Stimuli were supplied by a battery powered coupled pulse generator (Medtronics 5837) in both the demand and coupled modes. All intervals were measured in msec.

Electrocardiograms (Fig 1)

The heart was in sinus rhythm with a PR interval of 0.16 sec. The QRS was 0.12 sec in duration with initial slowing typical of pre-excitation. Delta waves were positive in I, II aVL, and all the precordial leads, suggesting a diagnosis of type A pre-excitation using the classification of Rosenbaum et al.14

Although most P waves were conducted to the ventricles with pre-excitation, occasional P waves were conducted normally (Fig 1). This finding is consistent with intermittent block of an anomalous pathway.

No tachyarrhythmias were demonstrated during the patient's hospitalization.

Electrophysiologic Studies

His bundle (H) electrograms were recorded on a multichannel oscilloscopic photographic recorder (Electronics for Medicine, model DR8) using previously described catheter techniques, at paper speeds of 100 and 200 mm/sec.15 Atrial pacing was performed with a bipolar pacing catheter placed against the lateral wall of the right atrium. Stimuli were supplied by a battery powered coupled pulse generator (Medronics 5837) in both the demand and coupled modes. All intervals were measured in msec.

The presence of delta waves were used as a marker for pre-excitation. The P-delta interval, from the onset of the P wave to the onset of the delta wave, was used as a measure of conduction time in the anomalous pathway. The P-H interval, from the onset of the P wave to the His bundle electrogram, was used as a measure of A-V nodal conduction time (normal 80-140 msec). During atrial pacing, both P-delta and P-H were measured from the atrial pacing spike.

H-V interval, from the H potential to the onset of the QRS, was also measured. During pre-excitation, H often followed the onset of the QRS and H-V was recorded as a negative value. When conduction normalized, H preceded the QRS and H-V was recorded as positive, and used as a measurement of conduction time from His bundle depolarization to the onset of ventricular activation (normal 35-55 msec).

Control heart rate averaged 62/min. Pre-excitation was present with a QRS duration of 120 msec and prominent delta waves (Fig 2). P-delta interval measured 155 msec and P-H varied from 150-160 msec (average 155). Thus, the H potential occurred within 5 msec of the onset of the QRS (H-V of -5 to +5 msec), suggesting that initial ventricular activation and His bundle activation occurred simultaneously.

With atrial pacing, P-delta and P-H intervals dissociated (Table). With increase in rate, P-delta remained fixed at 170-180 msec in all beats with delta waves. The increase in P-delta of 15-25 msec from control value represented the additional conduction time from the pacing spike to the onset of the P wave. P-H intervals increased with increasing heart rates, a normal response. At a paced rate of 70/min, P-delta was 180 msec while P-H increased to 190 msec (Fig 3-top). The slight increase in P-H relative to P-delta was accompanied by an increase in QRS duration to 135 msec. At 75/min P-H further prolonged so that the H potential was masked in the ventricular electrogram.

At a paced rate of 80/min, total block occurred in the anomalous pathway, with normalization of the QRS. A complete sequence of beats at this rate is shown in Figure 4. Following the onset of pacing, P-H increased from 165-210 msec before stabilizing at the latter value. P-delta remained fixed at 170 msec for six paced beats, until sudden failure of conduction in the anomalous pathway occurred on the seventh beat. The gradual increase in P-H relative to P-delta was accompanied by left axis deviation and increasing QRS duration. The first two beats are fusion beats, with the delta wave reflecting ventricular pre-excitation and the rest of the QRS reflecting ventricular depolarization over the normal pathway. With slowing in the normal pathway, more of the ventricles were depolarized via the anomalous pathway. The third and fourth beats also appear to be fusion beats, but predominantly anomalous in origin. The fifth and sixth beats were dissociated with a normal P-H interval.

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Figure 1. Electrocardiogram demonstrating normal PR interval (0.16 sec) with ventricular pre-excitation. Delta waves are seen with a QRS duration of 0.12 sec. The rhythm strip (below) shows sinus rhythm with sudden normalization of conduction on the third and seventh beats.

Figure 2. Control recordings during sinus rhythm. Shown are lead II, lead I and simultaneous His bundle electrogram (HBE). P waves are labeled P, delta wave labeled D, QRS labeled R, atrial electrogram labeled A, and His bundle electrogram labeled H. Both the P-delta (PD) and P-H (PH) intervals are 155 msec. His bundle depolarization is coincident with the onset of ventricular activation. PR prolongation represents prolongation of P-delta interval. Time lines on this and all subsequent illustrations are at 100 msec. Paper speed is 200 mm/sec.
Table 1—Effect of Atrial Pacing on Conduction Intervals

<table>
<thead>
<tr>
<th>Paced Atrial Rate (beats/min)</th>
<th>P-Delta (msec)</th>
<th>P-H (msec)</th>
<th>H-V (msec)</th>
<th>QRS Duration (msec)</th>
<th>QRS Morphology</th>
</tr>
</thead>
<tbody>
<tr>
<td>70</td>
<td>180</td>
<td>190</td>
<td>-10</td>
<td>135</td>
<td>Anomalous</td>
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<tr>
<td>75</td>
<td>170</td>
<td>-</td>
<td>50**</td>
<td>100**</td>
<td>Normal</td>
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<tr>
<td>145</td>
<td>-</td>
<td>***</td>
<td>50</td>
<td>100</td>
<td>Normal</td>
</tr>
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</table>

*Prior to failure of conduction in the anomalous pathway; **after conduction normalized; ***2:1 + 3:2 block proximal to H.

Failure of conduction in the anomalous pathway persisted at all rates above 80/min. P-H continued to increase with increasing heart rates (Fig 3-bottom). At a paced rate of 145/min, second degree block developed in the normal pathway, with development of 3:2 and 2:1 block proximal to the His bundle (Fig 5). The anomalous pathway was totally blocked and did not protect against second degree A-V block.

Coupled atrial pacing was utilized for measurement of effective refractory periods. At a coupling interval of 720 msec (measured from the spontaneous P wave to the coupled paced P wave) conduction was maintained in the anomalous pathway (Fig 6A). Shortening the coupling interval to 700 msec produced block in the anomalous pathway, with normalization of the coupled QRS (Fig 6B). The effective refractory period of the anomalous pathway was thus 700 msec. This anomalous pathway refractory period seems long when compared to the normal A-V nodal refractory period, (250-350 msec), and when compared to other patients with pre-excitation. However, further work is necessary before the value of 700 msec can definitely be considered prolonged. As coupling intervals were further shortened, increasing P-H prolongation was noted in the coupled beat (Fig 6C). At a coupling interval of 290 msec, failure of atrial capture occurred. The effective refractory period of the A-V node was less than 290 msec, but could not be precisely measured because of conduction failure at the atrial level. No echoes or tachyarrhythmias were elicited by coupling pacing.

In summary, conduction in the anomalous pathway was depressed as evidenced by: 1) prolonged conduction time of 155 msec (first degree block in the anomalous pathway); 2) complete block at a relatively slow paced rate; 3) a relatively prolonged effective refractory period. A-V nodal conduction was also slightly depressed as shown by slight prolongation of P-H interval.

The normal PR interval accompanying pre-excitation reflected the long P-delta interval. If the patient did not have pre-excitation, he would have had first degree A-V block with a PR interval of 0.20 sec (P-H of 155 and an H-V of 50).

DISCUSSION

The classic electrocardiogram of Wolff-Parkinson-White syndrome is characterized by a PR interval of 0.12 sec or less, a delta wave, and a QRS duration of 0.12 sec or greater. Most workers accept that this electrocardiographic pattern reflects...
pre-excitation of one of the ventricles because of an anomalous A-V connection (Kent bundle) bypassing the A-V node.5-12

Several electrocardiographic variants of pre-excitation have been described including the occurrence of short PR intervals with normal QRS, and the occurrence of pre-excitation (delta wave and wide QRS) with normal PR intervals.2-4 The present report is concerned with this latter variant.

Wood and Wolfeth17 suggested that changes in conductivity of the anomalous pathway could account for variations in the PR interval and QRS duration. Ohnell10 postulated that pre-excitation occurring with a normal PR interval could reflect a delay in "starting time" of the anomalous pathway. Ferrer11 suggested that such cases were due to functioning Mahaim tracts. These tracts, which arise from the His bundle and insert into the ventricular septum can theoretically produce ventricular pre-excitation. Since they are infranodal in location, the normal A-V nodal delay is preserved and PR intervals are normal.

The recording of His bundle electrograms and simultaneous electrocardiograms allow the definition of whether pre-excitation is occurring via pathways bypassing the A-V node or by pathways which are infranodal in location.9,10 The presence of delta waves are used as a marker for the occurrence of ventricular pre-excitation, and the P-delta interval is used as a measure of conduction time in the anomalous pathway. The His bundle electrogram is used as a marker for the normal conduction pathway, and the P-H interval is used as a measure of conduction time in the normal A-V pathway.

In most cases of Wolff-Parkinson-White syndrome, electrophysiologic studies using His bundle technique suggest pre-excitation via pathways by-
PRE-EXCITATION WITH NORMAL PR INTERVALS

A. 720

P

P1

B. 700

P

P1

C. 360

P

P1

Figure 6. Determination of refractory periods with coupled atrial pacing. Pacing speed 100 mm/sec. Shown are leads II, I and HBE. A) Coupling interval (from P to P1) of 720 msec. Pre-excitation is present in the coupled beat. P-H is 180 msec. B) Coupling interval of 700 msec. The refractory of the anomalous pathway is reached, conduction normalizing on the coupled beat. C) Coupling interval of 360 msec. The anomalous pathway remains blocked while P-H is markedly prolonged in the coupled beat. The coupled beat now shows aberrant conduction (functional right bundle branch block) as demonstrated by the development of an S wave in I.

passing the A-V node, and inserting into the ventricles. Usually the P-delta interval is shorter than the P-H interval, the H potential being recorded after the onset of the delta wave. With increase in heart rate with atrial pacing, P-delta interval remains constant while P-H interval increases. Frequently, the QRS becomes more anomalous in appearance as the discrepancy in P-delta and P-H becomes greater, suggesting that more of the ventricles are depolarized via the anomalous pathway.

If pre-excitation were occurring via Mahaim tracts, conduction should be characterized as follows: 1) P-H interval should be within the normal range while H-V should be short; 2) PR interval (P-delta) should be normal since P-H interval is normal; 3) increase in rate with atrial pacing should be accompanied by increase in P-H and PR (P-delta) while H-V remains unchanged and the degree of pre-excitation remains constant; 4) P-H and P-delta should not dissociate as in the usual case of WPW.

Castillo and Castellanos reported one patient with pre-excitation who partially fulfilled the criteria specified above. Their patient had two types of pre-excitation complexes, one typical of Kent bundle conduction, with short P-delta and normal P-H intervals, and the other suggestive of Mahaim tract conduction with normal P-H and prolonged P-delta with a short H-V. Atrial pacing in their patient produced prolongation of P-H and P-delta while the H-V remained unchanged, further supporting the occurrence of Mahaim tract conduction. Rosen and co-workers reported a child with congenital heart block and a QRS suggestive of type B pre-excitation. His bundle electrograms revealed total block proximal to the His bundle and a short H-V interval. These findings were interpreted as supporting the occurrence of infranodal pre-excitation via Mahaim tracts. Atrial pacing could not be performed because of complete A-V block.

The present case was characterized by pre-excitation with normal PR intervals. Studies suggested that pre-excitation occurred via pathways bypassing the A-V node. With increase in heart rate, P-H prolonged (moving into the QRS), while P-delta remained constant. As P-H prolonged, the QRS widened, and the axis shifted leftwards, suggesting that ventricular activation was occurring through an anomalous pathway (Kent bundle).

The normal PR interval in this patient appeared to represent slow conduction in the anomalous pathway, with a P-delta (PR) of 155 msec during control recordings. One could describe this finding as first degree block in the anomalous pathway. Other findings consistent with conduction in the anomalous pathway were also noted, including: 1) spontaneous second degree block in the anomalous pathway noted electrocardiographically; 2) a prolonged effective refractory period (700 msec) of the anomalous pathway and 3) total block of the anomalous pathway at paced atrial rates of 80/min or faster. The normal A-V pathway in this patient was also slightly depressed as manifest by slight P-H prolongation. The cause of slow Kent bundle conduction may have been either congenital or acquired.

Clinical Implications

The findings in this patient underscore the importance of evaluation of conduction properties of both normal and anomalous pathways in patients with WPW. The PR interval and degree of pre-excitation reflect the relationship of conduction times in both pathways. The PR interval will reflect the fastest pathway to the ventricles, whether it be anomalous or normal. The QRS morphology will reflect the two pathways, with fusion complexes when the conduc-
tion times are relatively close, and either predominantly normal or anomalous conduction if one pathway is considerably faster than the other.

The finding of depressed anomalous pathway conduction in a patient implies that re-entrant arrhythmias utilizing the pathway are unlikely. A pathway that cannot support conduction at a paced rate of 80/min or faster, as in the present case, should protect against paroxysmal tachycardia. On the other hand, a depressed anomalous pathway might not protect against development of acquired A-V block later in life. In the present case, it is even possible that the slight A-V nodal depression and marked anomalous pathway depression, are the earliest manifestation of degenerative disease involving the specialized conduction system. Further observations of this patient for many years will be necessary to determine if the present findings are the forerunner of clinically significant conduction disease.

It is pertinent to inject a note of caution regarding the application of findings in this patient to other patients with normal PR intervals and pre-excitation. Similar electrophysiologic evaluation is necessary to determine whether Mahaim tract conduction or slow Kent bundle conduction is present. We expect that cases secondary to Mahaim tract conduction will also be discovered.

References
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