Pseudo A-V Block Secondary to Premature Nonpropagated His Bundle Depolarizations

Documentation by His Bundle Electrocardiography

By Kenneth M. Rosen, M.D., Shahbudin H. Rahimtoola, M.B., M.R.C.P.E., and Rolf M. Gunnar, M.S., M.D.

SUMMARY

His and right bundle electrograms were recorded in a patient with unexplained P-R prolongations and periods suggestive of both type I and II second degree A-V block. The mechanism of these conduction disturbances was shown to be due to multiple nonpropagated premature His bundle depolarizations (H') which produced their effects on the subsequent cycle by virtue of concealed conduction into the A-V junction. The effects on A-V conduction were related to the time of occurrence of H' in relation to the subsequent P wave: If H'-P was short, a more profound conduction defect occurred. H' was generally undetectable on the ECG because of both antegrade and retrograde block, but a few propagated H' produced typical junctional premature contractions. Propagation of H' depended on a short preceding cycle length and a late occurrence of H'. The site of origin of H' appeared to be in the bundle of His. The mechanism of production of H' did not appear to be either reentry or parasystole.

The term pseudo A-V block is used to describe the arrhythmia because there was no evidence of an intrinsic abnormality of A-V conduction. It is expected that other unusual electrocardiographic phenomena may be explainable with intracardiac recordings of specialized conduction tissue potentials.

Additional Indexing Words:

Cardiac conduction Junctional rhythms Wenckebach phenomenon
His-Purkinje system P-R interval

In 1947, Langendorf and Mehlman suggested that nonconducted junctional premature systoles could imitate first and second degree A-V block by virtue of concealed conduction to the A-V node. They reported a case of typical junctional premature systoles, blocked premature P waves of retrograde contour, a sudden prolongation of the P-R interval, and occasional block of

From the Department of Adult Cardiology of the Hektoen Institute for Medical Research and Cook County Hospital, the Division of Medicine of Cook County Hospital, and the Department of Medicine of the University of Illinois College of Medicine, Chicago, Illinois.

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normally conducted P waves. They postulated that the P-R prolongation and blocked P waves were due to the presence of junctional premature systoles which were concealed because of both antegrade and retrograde block. The existence of these was inferred by the presence of conducted junctional premature systoles and junctional premature systoles with retrograde block, and because of the unusual abnormalities of A-V conduction.

In 1958, Langendorf\(^2\) described a patient in whom alternation in P-R intervals was ascribed to concealed retrograde conduction of nonconducted interpolated A-V junctional premature systoles. Recently Langendorf and Pick\(^3\) also reported an instance of pseudo-Wenckebach periods believed to result from concealed A-V junctional premature contractions.

Until now the suggested mechanism of these arrhythmias has remained unproven. We report here on a patient who manifested sudden P-R prolongations and periods suggestive of both type I (Wenckebach) and type II (Mobitz II) second degree A-V block. With catheter recordings of His bundle potentials, the mechanism of this arrhythmia is shown to be due to the presence of nonconducted junctional premature systoles, manifesting their effects on normal sinus beats by mechanism of concealed conduction to the A-V junction. The effects of these premature were related to their timing in the cardiac cycle.

**Report of Case**

A 59-year-old woman was admitted to Cook County Hospital for evaluation and treatment of congestive heart failure. Blood pressure was 118/70. Neck vein distention at 45° was noted, and rales were heard at both lung bases. A grade II/VI ejection systolic murmur radiating to the carotids was presumed to be due to calcific aortic stenosis. Moderate anemia was present and examination of the bone marrow revealed tumor cells.

**Electrocardiograms**

Representative ECG strips are shown in figure 1. Normal sinus rhythm was present with an atrial rate of 80 to 90/min. QRS duration was 0.08 sec. P-R intervals varied from 0.10 to 0.40 sec. There were sudden unexpected P-R prolongations (fig.

![Figure 1](image)

Three representative electrocardiographic strips (lead II) showing variations in A-V conduction.

(A) There is unexplained sudden P-R prolongation on the fourth and seventh P waves. The second QRS arising right off the P wave is felt to represent a junctional premature contraction with aberrant conduction.

(B) Apparent type I block (Wenckebach). The third and sixth P waves appear to be blocked.

(C) Sudden P-R prolongation occurs with the second P wave. The seventh P wave is blocked without preceding P-R prolongation, suggesting type II A-V block (Mobitz). Time lines on this figure and all subsequent figures occur at 1 sec. Paper speed is 25 mm/sec.

1A and C) as well as periods of second degree A-V block mostly of type I (fig. 1B) but occasionally of type II (fig. 1C). There were occasional premature contractions of both normal and aberrant configurations without accompanying premature P waves (fig. 1A). In addition to the arrhythmia, there were nonspecific ST and T-wave changes.

**Electrophysiologic Studies**

Informed consent was obtained for right heart catheterization. Both His bundle and right bundle electrograms were recorded on a multi-channel oscilloscopic photographic recorder\(^*\) at paper speeds of 100 and 200 mm/sec, by previously described technic.\(^4\), \(^5\) Simultaneous lead II ECG was recorded.

The P-H interval was measured from the onset of the P wave to the peak deflection of the His bundle electrogram (H), H-Q interval from the peak deflection of H to the onset of the ECG QRS complex, and RB-Q, from the peak of the right bundle potential (RB) to the onset of Q wave. Basic cycle length was measured from H to H

\(^*\)Electronics for Medicine (model DR8), White Plains, New York.
(H-H). All intervals were measured in milliseconds.

Several hundred cardiac cycles were analyzed. The basic rhythmic mechanism was normal sinus with an atrial rate of 80 to 90/min. P-H intervals varied from 110 to 140 msec in the normally conducted beats. H-Q and RB-Q intervals were 42 msec and 15 msec.

H' occurred between the atrial and the ventricular electrograms of conducted beats. In addition another electrical depolarization, similar in contour to H, occurred at varied times in the cardiac cycle but always between the ventricular electrogram of one beat and the atrial electrogram of the next beat (figs. 2 and 3). This depolarization will be referred to as H'.

H' was recorded generally between every second to every fourth cycle (occasionally H' occurred in two consecutive cycles). There was varying coupling between H' and the preceding beat, H' occurring from 320 to 640 msec from the preceding H. The occurrence of H' was not related to the preceding P-H or H-H intervals, and a parasystolic relationship with H' spikes was not demonstrable.

No atrial electrogram or P wave could be attributed to H' and most H' depolarizations were not related to ventricular activation. (See further discussion below.) However, there was a striking relationship between H' and the cardiac cycle following it. P-H prolongation or complete block of the P wave occurred in the cycle following the

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**Figure 2**

Three representative strips of simultaneous lead II ECG and His bundle electrogram (HBE) showing the effects of premature His bundle depolarization (H') on the subsequent cardiac cycle, and also demonstrating the propagation of an H' to the ventricles. Paper speed is 100 mm/sec. P-H intervals are listed at the top of each strip over the cycle in which it was measured. H'-P intervals are noted just under HBE. H-H' intervals are listed on the bottom of each strip. See text for definition and discussion of these intervals.

(A) H'-P is 220 msec and the subsequent P wave is blocked. There are both retrograde and antegrade blocks of the H' potentials with no related atrial or ventricular activity. The ECG resembles type II A-V block (Mobitz).

(B) The first H'-P interval is 290 msec, and the subsequent P-H is prolonged to 160 msec. The second H'-P is 160 with block of the P wave. The ECG resembles a Wenckebach period, with P-R and P-H prolongations before the blocked P.

(C) The first H'-P of 245 msec produced P-H prolongation to 200 msec. The second H' propagates to the ventricle because of a long H-H' interval (460). In tracings A and B, H' does not propagate because of shorter H-H' intervals. The P wave following the propagated H' is blocked because of the short H'-P interval.
H’. The degree of P-H prolongation or the occurrence of block was related to the H’-P interval (from H’ to the following P wave). This relationship is shown graphically in figure 4. The longer H’-P intervals (240 to 290) tended to be associated with lesser degrees of P-H prolongation, in the range of 140 to 200 msec (fig. 2B and C). When H’-P was 210 to 240, P-H was often markedly prolonged (> 300 msec), or the P wave was nonconducted (figs. 2A and 3). When H’-P was 210 msec or less, the subsequent P wave was always blocked (fig. 2B and C), and all of the nonconducted P waves were blocked above H.

All P-R prolongations as well as blocked P waves could be accounted for by the presence of H’ in the preceding cycles. The electrocardiographic appearance depended on where and when H’ occurred. Type I block was simulated when H’ occurred in two succeeding cycles, with the second H’-P being short enough to produce block (fig. 2B). When H’ occurred in an isolated cycle, the H’-P determined whether unexpected P-R prolongation occurred (fig. 2C and 3) or type II block was simulated (fig. 2A).

Eighteen per cent of H’ potentials appeared to be conducted to the ventricles, with either normal or aberrant conduction. H’-Q intervals of the propagated H’ were from 55 to 75 msec. Propagation of H’ to the ventricles was likely when preceding cycle length (H-H) was short and when H’ occurred relatively late (long H-H’). This relationship is shown graphically in figure 5. Examples of nonpropagation with short H-H’ are shown in figure 2A and B, and propagation with longer H-H’ is shown in figure 2C.

The polarity of the recorded H’ varied from being similar to H (fig. 2A, B, and C) to being the opposite from that of H (fig. 3). Small changes in catheter position would change the polarity of H’ while not affecting the polarity of H.

Intracardiac recordings of right bundle-branch potentials revealed no evidence of propagation of H’ (fig. 6) except when H’ was propagated to the ventricles.

**Discussion**

Premature His bundle depolarizations (H’) resembled typical diphasic or triphasic His...
Minimal changes in catheter position could cause the polarity of H' to vary from that of antegrade contour (similar to the H of normally conducted beats) to complete reversal of polarity. The changes in polarity of H' occurred without concomitant change in polarity of the normally conducted H. This phenomenon is explained by postulating a site of origin of H' in the bundle of His. Small changes in catheter position could place the poles slightly above or below the locus of origin, changing the polarity of the recorded H', but not affecting the polarity of H.

Placing the site of origin in the His bundle is consistent with current concepts regarding automaticity in the specialized conduction tissue. Action potential studies suggest that A-V node cells are not automatic, and that automaticity in the A-V junction resides in His-Purkinje cells. Damato and Lau utilizing catheter recordings of A-V node and His bundle potentials in patients with junctional rhythms, have also suggested origin of these rhythms from the His bundle.

The mechanism of production of H' could not be determined. The premature His bundle depolarizations did not appear to represent reentrant activation of the His bundle for several reasons. Fixed coupling, a typical finding of reentry, was not present. H' occurred relatively late after ventricular depolarization of normally conducted beats. There was no relationship between the occurrence of H' and the preceding cycle length, or the preceding P-H and H-Q cycles.

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**Figure 5**
The effect of timing of H' on propagation to the ventricles. Abscissa: H-H' intervals; ordinate: preceding H-H intervals. Note that both short preceding cycle length and late occurrence of H' favor the propagation of H' to the ventricles. See text for further discussion.

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**Figure 6**
Simultaneous ECG and right bundle electrogram (RBE) showing lack of penetration of H' to the RB. Note that there is no evidence of H' before the blocked P wave suggesting that the zone of concealment of the forwardly conducted H' is above the site of the recording of RBE. Paper speed is 200 mm/sec. Time calibration is shown in milliseconds.
intervals. Parasytrole also did not appear to explain the random occurrence of H.

Both antegrade and retrograde block of H were present. No P wave or atrial electrogram was associated with premature His bundle activity, and so complete retrograde block of H was present. The occurrence of retrograde block with junctional rhythm is quite common, with the zone of concealment probably residing in the A-V node region.11

Antegrade block of H was incomplete with conduction of 18% of the recorded H to the ventricles. Refractory periods of both the His bundle and bundle branches are a function of basic cycle length.12 Circumstances were ideal for propagation to the ventricles when a short cycle length (H-H) was followed by a late occurring H. The relationship of these variables to the conduction of H can be noted on the graph (fig. 5). QRS contour of the conducted H varied from normal to aberrant.

As mentioned above, there was antegrade block of most H potentials. Since no evidence of the blocked H was noted on the right bundle electrogram, the site of concealment is presumed to be low in the His bundle or in both bundle branches (high in the right bundle branch and either proximal or distal in the left bundle-branch system).

The effect of H on the subsequent cardiac cycle is most interesting. Unexplained P-R prolongations and blocked P waves were noted on the electrocardiogram, suggesting an unusual type of A-V block. All of these apparent abnormalities of the A-V conduction were explainable by the premature His bundle depolarizations. On the His bundle recording it was noted that in the cycle following an H, the P wave was conducted with a prolonged P-H or blocked above the bundle of His. The occurrence of prolongation or block was related to the timing of H, the greater conduction disturbance occurring after the shorter H-P intervals. This was due to concealed retrograde conduction of the premature His bundle depolarization into the A-V junction. H, although blocked in the A-V junction, modified conduction of the subsequent impulse. The closer to the subsequent P wave that H occurred, the more refractory the A-V junction became until complete decrement of the conducted atrial impulse occurred. The phenomenon resembled that of coupled concealed pacing described by Sheiner and Stock,13 who with coupled ventricular pacing were able to slow the ventricular response in a case of atrial fibrillation. Their coupling interval was short enough so that the coupled stimulus produced no propagated electrical response but by concealed conduction into the A-V junction slowed A-V conduction, thus reducing ventricular rate. In our case, instead of a pacer impulse, the H produced concealed retrograde conduction.

No intrinsic disturbance of A-V conduction was present. P-H and P-R intervals of beats that were not interfered with were normal, and neither first nor second degree block would have been simulated if premature His bundle depolarizations had not occurred. The term pseudo A-V block as suggested by Langendorf and Pick3 seems appropriate for this arrhythmia.

Several unusual electrocardiographic findings that suggest the diagnosis of pseudo A-V block secondary to nonpropagated His bundle premature contractions are: (1) the sudden appearance of unexplained P-R prolongations1; (2) the appearance of both type I and type II block in the same patient; and (3) the occurrence of type II block in a patient with normal duration QRS complexes.14 The presence of junctional premature contractions is a clue to the nature of the arrhythmia.1-3 But proof depends upon recording of premature His bundle potentials.

It is intriguing to speculate that other unusual electrocardiographic phenomena may be explainable with intracardiac recordings of specialized conduction tissue potentials. There is evidence of automaticity in varying atrial sites and throughout the His-Purkinje system.8 It seems likely that premature activation can occur throughout the conduction system, which because of local variations in refractory periods may be concealed, yet may manifest effects on the conduction or automaticity of other cardiac conduction tissue.
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References

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KENNETH M. ROSEN, SHAHBUDIN H. RAHIMTOOLA and ROLF M. GÜNNAR

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