Mobitz Type II Block without Bundle-Branch Block

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SUMMARY
His bundle (H) electrograms were recorded in three patients with Mobitz type II block and narrow QRS. Block was secondary to digitalis intoxication in one patient. In the second patient, who had first-degree A-V block, type II block occurred with atrial pacing at a slightly increased heart rate. In the third patient, who had corrected transposition of the great vessels, type II block occurred spontaneously. In two additional patients, block simulating type II block was noted. In one, block of single P waves occurred with carotid massage. In the other, Wenckebach periods with small increments in P-R (and P-H) intervals resembled episodes of type II block. In all five patients, block was proximal to H, suggesting the A-V node as the site of block. The conduction defects in these patients were not progressive; none of the patients needed a pacemaker.

Although these mechanisms were identified in patients with narrow QRS complexes, they could occur with bundle-branch block suggesting an erroneous diagnosis of bilateral bundle-branch disease. It is concluded that His bundle recording is helpful in delineating these benign forms of block. The site of block may be a more important determinant of prognosis than the type of block.

Additional Indexing Words:
Wenckebach block Digitalis intoxication
His-Purkinje system Bilateral bundle-branch block

Mobitz type II, second-degree A-V block is characterized by the presence of fixed P-R intervals prior to the dropped beat.1, 2 This arrhythmia usually occurs in patients with established bundle-branch block and is felt to reflect sudden failure of conduction in the functioning bundle branch.3, 4 Type II block frequently is a precursor to more advanced conduction disturbance and has been related to irreversible destruction of the conduction system.1-4 Thus, type II block may be an indication for pacemaker insertion.5, 6

Experimental studies in isolated rabbit heart have suggested that the site of type II block is in or below the His bundle.7 Narula and Samet8 have recorded His bundle electrograms in 12 patients with type II block and have demonstrated block as occurring distal to the H recording site. Nine of their patients had bundle-branch block patterns in conducted beats, and they speculated that type II block represented bilateral bundle-branch
disease. In three of their patients, the QRS complex was narrow, and block was suggested to have occurred in the His bundle. We have recently reported a case of type II block and narrow QRS complex in which the block was proximal to the His bundle. Block in this patient reflected the occurrence of concealed premature His bundle depolarizations, which produced block of subsequent P waves by virtue of concealed retrograde penetration into the A-V junction (pseudo A-V block).

In the present report, we will describe three patients with type II block and narrow QRS complexes, and two additional patients with narrow QRS in whom type II block was simulated. In all the patients, block occurred proximal to the His bundle recording site. Conduction disease has not progressed in any of the patients, and this fact suggests that type II block with narrow QRS complexes can be a benign process. Similar forms of block could occur in patients with bundle-branch block leading to an erroneous diagnosis of serious bilateral bundle-branch disease.

Methods

The following electrocardiographic definitions were utilized: Second-degree A-V block was defined as incomplete A-V block with sinus rhythm and dropped ventricular beats. Second-degree block was classified into Mobitz types I and II. Type I block (Wenckebach) was characterized by progressive P-R prolongation preceding the dropped ventricular beat. Type II block was characterized by constant P-R intervals (which could be normal or prolonged) prior to the dropped beat. In the first one or two beats following the pause, the P-R interval could be variable. This variability could reflect either improvement in conduction following the dropped beat or the fortuitous occurrence of junctional escape beats a short time after the first P following the blocked P. In the latter instance, the P-R interval following the dropped beat could be extremely short, since it does not reflect A-V conduction time. It should be reemphasized that neither the QRS duration nor the site of block is a determinant of whether second-degree block is type I or type II. The differentiation of these types is only dependent upon the behavior of the P-R intervals prior to block.

Advanced A-V block was defined as incomplete A-V block with 2:1 or greater degree of block. QRS duration was considered narrow when the greatest QRS duration in the standard and augmented limb leads was 0.10 sec or less.

His bundle electrograms were recorded with a tripolar catheter passed percutaneously via a femoral vein and positioned at the tricuspid valve. Recordings were made at filter frequencies of 40 to 500 Hz on a multichannel oscilloscopic photographic recorder* at paper speeds of 100 and 200 mm/sec. Single or multiple simultaneous electrocardiographic leads were recorded.

P-H, representing the intraatrial and A-V-nodal conduction time, was measured from the onset of the P wave to the peak deflection of the His bundle electrogram (H) (normal 80 to 140 msec). If P waves were not well-visualized, the A-H conduction time was measured from the onset of the atrial electrogram. The H-Q interval, a measure of conduction time in the His-Purkinje system, was measured from the first high-frequency component of H to the onset of the QRS complex (normal, 35 to 55 msec). When block occurred, it occurred proximal or distal to the H recording site.

Report of Cases

Case 1

The patient, a 63-year-old male with aortic insufficiency, was admitted to the hospital because of second-degree A-V block. He had been receiving 0.25 mg of digoxin per day prior to admission. There was no history of chest pain or electrocardiographic evidence of myocardial infarction.

Admission electrocardiograms revealed type I block with occasional 2:1 block (fig. 1A). Digoxin was discontinued and A-V block diminished in severity over the next several days. On the fifth hospital day, the patient was in sinus rhythm with a P-R interval of 0.38 sec. At this time, episodes of type II block were noted. In figure 1B, the last 10 beats of a sequence of over 60 beats are shown. During the total sequence of beats, the P-R interval was fixed at 0.38 sec. The eighth P of the figure is unexpectedly blocked, and is followed by a junctional escape (eighth QRS) 1.44 sec after the previous QRS. The next QRS (ninth QRS) may also be a junctional escape, occurring at the same escape interval. The last QRS (tenth) occurs earlier and is conducted with a P-R interval of 0.38 sec. The ninth P and possibly the tenth P waves are not conducted, the former P occurring right after the QRS and the latter 0.28 sec in front of the ninth QRS. The possibility that the tenth P conducts the ninth QRS cannot be excluded. Several

*DR16—Electronics for Medicine, White Plains, New York.
CASE 1. Electrocardiographic rhythm strips: (A) On admission showing 4:3 and 3:2 Wenckebach periods and 2:1 block. (B) On fifth hospital day demonstrating type II block. P-R intervals are constant at 0.38 sec. The eighth P wave is suddenly blocked and is followed by a junctional escape (eighth QRS). There is an interfered (blocked) P in the S-T segment of the escape beat. The ninth QRS may also be a junctional escape. See the text for further discussion. (C) On the seventh hospital day showing sinus rhythm with first-degree A-V block (P-R of 0.34 sec).

Electrophysiologic Studies

The patient was studied on the sixth hospital day. The heart rate was 70/min without variation in cycle length. A-H intervals ranged from 290 to 320 msec except in the first one or two beats following blocked P waves, when they were shorter. Several examples of type II block proximal to H were recorded. An example is shown in figure 2A. In this illustration, A-H varied from 290 to 320 msec. The A-H intervals remain relatively constant prior to sudden block of the fifth P wave. This blocked P was followed by a junctional escape 1.5 sec after the last conducted QRS, and 140 msec after the next P. Escape beats in this patient were always preceded by H potentials which differed from conducted H potentials in that they were wider and of less amplitude. This is shown in both figure 2A and 2B; the latter, which was recorded at a faster paper speed, shows a blocked P, a junctional escape following the next P (with an A-H interval of 70 msec), and a conducted beat with an A-H of 250 msec. The A-H intervals of 140 msec (fig. 2A) and of 70 msec (fig. 2B) do not reflect A-V conduction times but represent the fortuitous occurrence of junctional escape beats shortly after P waves. The duration of the A-H of the conducted beat in figure 2B is slightly shortened to 250 msec, and this slight degree of shortening is a reflection of improvement in A-V conduction following the two nonconducted P waves.

Comment

This patient had episodes of type II block recorded during routine electrocardiographic monitoring as well as at the time of electrophysiologic study. The diagnosis was based on the occurrence of second-degree A-V block without progressive prolongation of either P-R or A-H prior to the dropped beats. The occurrence of Wenckebach periods in this patient does not negate the diagnosis of type II block, which is a descriptive categorization of those episodes of second-degree block in which the P-R and A-H intervals remained fixed prior to block.

The site of block was proximal to the H recording site, probably in the A-V node. Second-degree A-V block was secondary to digitalis intoxication, which was superimposed on chronic first-degree A-V block. To our knowledge, this is the first reported case of type II block secondary to this drug.
Case 1. Simultaneous His bundle electrograms (HBE) and electrocardiographic lead V1 demonstrating type II block proximal to H. Time lines on this and subsequent illustrations are at intervals of 1 sec.

(A) P wave and atrial electrograms are labeled P, His bundle electrogram labeled H, and QRS labeled R. A-H intervals are listed on the recording. Note slight variation in A-H with constant A-H in the two beats prior to block. The fifth P is blocked proximal to the His bundle and is followed by a junctional escape with somewhat aberrant-appearing H spike. The P-H of 140 msec in this latter beat is spurious and does not represent A-V conduction time. Paper speed is 100 mm/sec.

(B) Recording at 200 mm/sec paper speed showing blocked P wave followed by a junctional escape with aberrant-appearing H spike. The A-H of 70 msec is spurious. The third P is conducted with an A-H of 250 msec.

Case 2

The patient, a 22-year-old asymptomatic female, was referred to the cardiology service for electrophysiologic evaluation because of conduction disease. Electrocardiograms revealed varying first-degree A-V block with narrow QRS. The etiology of the conduction disturbance was not apparent.

Electrophysiologic Studies

During control recordings, the heart rate was between 52 and 68 beats/min. The A-H interval was 190 to 200 msec and the H-V was 38 msec. One-to-one A-V conduction was present. Atrial pacing at 80/min produced episodes of both type I and type II block proximal to the His bundle. An episode of the latter is shown in figure 3. The illustration shows a full sequence of beats following a blocked P. The first P-H (measured from the atrial pacing spike) is 210 msec. Subsequent P-H's are 250, 270, 270, 250, and 250; then block suddenly occurs proximal to H. The first P-H following the block beat is slightly shortened at 190 msec.

Comment

This patient with idiopathic first-degree A-V block had episodes of type II block with atrial pacing at relatively low heart rates. The diagnosis was based upon the occurrence of second-degree A-V block without progressive increases of either P-R or P-H interval prior to block. The slight decrease in P-H following the cycles with block is acceptable in type II block as defined by Katz and Pick.10

Case 3

The patient, a 26-year-old male with corrected transposition of the great vessels and ventricular inversion, was admitted for cardiac catheterization. Electrocardiograms revealed first-degree A-V block with a P-R interval of 0.30 sec and absence of normal septal forces.

Electrophysiologic Studies

Studies were performed during diagnostic cardiac catheterization. His bundle electrograms
were recorded with a tripolar catheter positioned across the right A-V valve (bicuspid). The rhythmic mechanism was sinus with a prolonged P-H of 220 msec and a normal H-Q of 52 msec. Unexpected blocked P waves were noted without progressive prolongation of conduction intervals prior to block (fig. 4). Block was always proximal to H.

Comment
This is the first report of intracardiac recordings of H potentials in a patient with corrected transposition. First-degree A-V block was demonstrated to represent prolongation of the P-H interval. During cardiac catheterization, type II second-degree A-V block was also noted with block proximal to the H recording site. In corrected transposition, the positions of both the A-V node and His bundle are inverted. The site of block may have been the A-V node, N-H region, or proximal His bundle.

Case 4
The patient, a 48-year-old male with chronic obstructive lung disease, was admitted because of leg swelling. Admission electrocardiograms revealed sinus tachycardia (120/min) with P-R interval of 0.18 sec. Carotid massage, attempted in order to rule out a paroxysmal tachycardia, produced slight sinus slowing and sudden A-V dissociation which lasted several beats until 1:1 A-V conduction returned (fig. 5 top). P-R intervals prior to and following A-V block were fixed.

Electrophysiologic Studies
His bundle electrograms were recorded after diagnostic right heart catheterization. Heart rate
was 110/min, A-H was 130 msec, and H-Q was 48 msec. With gentle carotid massage, single blocked beats were noted with block occurring proximal to H. In figure 5A, the A-H interval increased 10 msec prior to the dropped beat. With moderate carotid massage, both A-V block proximal to H and simultaneous sinus slowing were noted (fig. 5B). The A-H interval was fixed both prior to and following the A-V block.

Comment

A-V block in this patient was mediated via increased vagal tone produced with carotid massage. The site of block was presumably at the A-V node. A-V conduction appeared to be all or none in response to this vagal stimulus, with either 1:1 conduction being preserved or complete A-V block. Conduction intervals did not prolong prior to the occurrence of block. When only one blocked beat occurred (fig. 5A), the tracing simulated type II block. In this patient, A-V block did not present a clinical problem since it was obviously iatrogenic. Sudden increases in vagal tone with production of A-V block have occurred, however, in other patients in association with such maneuvers as hiccuping and swallowing, particularly in situations where conduction may already have been depressed. Thus, it is expected that type II block secondary to increase in vagal tone could exist as a clinical entity.

Case 5

The patient, a 52-year-old male with previous mitral valve replacement, was admitted because of second-degree A-V block. He had been taking 0.25 mg of digoxin per day prior to admission. Admission electrocardiograms revealed typical Wenckebach periods (fig. 6A). Digitalis was withheld, and the conduction disturbance slowly subsided. On the fourth hospital day, the patient was in sinus rhythm with a P-R interval of 0.28 sec, with occasional dropped beats. At first glance, the electrocardiographic strips appeared to show type II block (fig. 6B). However, careful measurement of P-R intervals revealed an almost imperceptible increase in P-R intervals over six or seven beats prior to the dropped beat. In fig. 6B, the initial P-R interval was 260 msec with gradual increase to 280 msec prior to block. Thus, a 20-msec increment in P-R interval was divided over six cardiac cycles. On the sixth hospital day, stable sinus rhythm prevailed with a P-R interval of 240 msec.

Electrophysiologic Studies

The patient was studied on the fourth hospital day. Atrial rate was 90/min. His bundle
MOBITZ TYPE II BLOCK

Case 4. Recordings showing block induced by carotid sinus massage. (Top) Rhythm strip during carotid massage. The eighth P wave is blocked without preceding P-R prolongation. The eighth and ninth QRS complexes could be junctional escapes with an escape interval of 1.32 sec. The P-R interval of the conducted beats is 0.18 sec.

(A) Gentle carotid massage producing sudden block of the fifth P wave, proximal to H. There is slight increase in P-H prior to the block. The QRS following the blocked P may be a junctional escape or may be conducted with a P-H of 110 msec. Paper speed 100 mm/sec.

(B) Moderate carotid massage producing sudden block of three consecutive P waves (the third, fourth, and fifth P's) proximal to the His bundle. The QRS following the long pause is probably a junctional escape. Note the absence of P-H prolongation either prior to or following the three blocked P waves.

Discussion

In the present study, we have reported the occurrence of type II block proximal to the His bundle in three patients with narrow QRS. In the first case, block occurred secondary to digitalis intoxication. In the second case, a case of first-degree A-V block, type II block occurred upon atrial pacing at increased heart rate. Type II block occurred spontaneously in the third case, that of a patient with first-degree A-V block and corrected transposition of the great vessels. In addition, we have described two additional cases in which block proximal to the His bundle closely simulated type II block. In one, sudden block of single P waves was produced by sudden transient

Comment

In this case, an arrhythmia-simulating type II block on electrocardiogram was demonstrated to be type I block. The cause of confusion was the relatively small increments in A-V conduction time which electrocardiographically could be measured only with some difficulty. This is another case of digitalis intoxication with block proximal to the His bundle recording site, probably in the A-V node.

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increase in vagal tone with carotid massage. In the other, type II block was simulated by type I block with very small increments in conduction times. This latter case was secondary to digitalis intoxication.

Previous workers have suggested that the site of type II block is in the His-Purkinje system. In this study, block occurred proximal to the His bundle recording site in all of the patients, and this suggests block either in the A-V node or proximal His bundle. The association of block with digitalis intoxication in two of the patients, the relationship to carotid massage in one of the patients, and the occurrence of type II block with chronic first-degree block with prolonged A-H in the other patients, all suggested the A-V node as the site of the block. In the patient with corrected transposition, block may have occurred at the A-V node, in the N-H region, or in the proximal His bundle.

The clinical course of these patients is of interest. In all of the patients, the episodes of type II block were short-lived. In the patients with digitalis intoxication, they occurred just prior to recovery from a more advanced conduction disturbance. None of the patients has developed more advanced conduction disturbances or needed pacemaker therapy. The benign course in these patients is in marked contrast to that of previously described patients with type II block, in whom advanced and progressive conduction disease was noted.

It is important to recognize these benign forms of type II block so that unnecessary pacemaker implantation can be avoided. In the present series of cases, the A-V-nodal site of block could be suspected because of the narrow QRS complexes and the occurrence of Wenckebach periods in these patients. However, similar forms of type II block could occur in association with bundle-branch block, leading to an erroneous diagnosis of bilateral bundle-branch disease.

These cases demonstrate the potential fallibility of the electrocardiographic diagnosis of site of block. Type I block, generally suggestive of A-V-nodal disease, has recently been demonstrated to occur also in the His-Purkinje system.

Figure 6

Case 5. Recordings demonstrating type I block simulating type II block. (A) Admission electrocardiographic strip showing 6:5 Wenckebach period. (B) Rhythm strip taken on fourth hospital day showing simulated type II block. The initial P-R interval is 260 msec and the P-R interval prior to block is 280 msec. (Bottom) Electrocardiogram and His bundle electrogram showing type I block with P-H increments of 10 to 15 msec prior to block of the fourth P proximal to H. Paper speed is 200 mm/sec.
MOBITZ TYPE II BLOCK

Type II block, which had previously been demonstrated to occur only in the His-Purkinje system, is now demonstrated to occur also proximal to the His bundle. Although the electrocardiographer will generally be correct in predicting the site of block, the recording of His bundle potentials obviously allows a greater degree of accuracy in the localization of conduction disturbance. The site of block is probably a more important determinant of prognosis than the type of block. Furthermore, work is necessary in delineating the relationship of clinical course to both the site and type of second-degree A-V block.

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References

4. DONOSO E, ADLER LN, FRIEDBERG CK: Unusual forms of second-degree atrioventricular block, including Mobitz type II block, associated with the Morgani-Adams-Stokes syndrome. Amer Heart J 67: 150, 1964
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