Wenckebach and Mobitz Type II A-V Block Due to Block Within the His Bundle and Bundle Branches

By Onkar S. Narula, M.D., and Philip Samet, M.D.

SUMMARY

Fourteen patients with conduction defects were analyzed by using bundle of His (BH) recordings. The BH electrograms were validated by BH and right atrial pacing (AP). In 12 patients with Mobitz type II A-V block, failure in impulse transmission for the dropped beats was localized distal to the recording site of the BH. Three of these 12 patients showed normal QRS complexes. In two of these three, the QRS complexes remained unchanged during intermittent periods of complete heart block (CHB), and thus represent His bundle rhythm with subsidiary pacemaker arising above the bifurcation of the BH. The A-H time in this group of 12 patients ranged from 60 to 160 msec and the H-V time ranged from 40 to 90 msec. At any atrial rate (NSR or AP) conduction time through the A-V node (A-H), and His-Purkinje system (H-V) remained constant. With increasing atrial (A) rates during AP, the number of impulses blocked distal to the BH increased. At high AP rates Wenckebach phenomenon between A and BH occurred concomitantly with block distal to the BH.

Of the two additional patients studied, the one with Wenckebach type 2° A-V block during NSR and a narrow QRS complex had the delay localized between two recorded and validated BH potentials ("split" BH). The other patient, with 1:1 A-V conduction during NSR and left bundle-branch block, developed Wenckebach cycles during AP at 110 beats/min. The progressive delay in the P-R interval was localized in the His-Purkinje system (HPS) probably in the right bundle.

Our data support the clinical observations that Mobitz II A-V blocks are associated with bilateral bundle-branch block as well as with BH lesions. The ECG is of limited value in the localization of the delays occurring at two sites simultaneously: namely, in the A-V node and the HPS. Furthermore, demonstration of the Wenckebach cycles within the HPS (BH or either bundle branch), which cannot be determined from the surface ECG, has important clinical implications.

Additional Indexing Words:
Electrocardiogram His bundle recordings A-V nodal conduction time
His-Purkinje system conduction time Atrial electrograms Atrial pacing
Ventricular electrograms Bilateral bundle-branch block

Two types of second degree atrioventricular (A-V) block were originally described by Hay' and by Wenckebach.2,3 Type I block was characterized by the gradual prolongation of the a-c interval of the jugular pulse with eventual loss of the arterial pulse. In type II second degree heart block, ventricular beats were dropped without any preceding prolongation of the a-c interval. The introduction of the electrocardiogram enabled Mobitz to define the ECG criteria for

From the Division of Cardiology, Department of Medicine, Mount Sinai Hospital, Miami Beach, Florida.
Address for reprints: Onkar S. Narula, M.D., Division of Cardiology, Department of Internal Medicine, Mount Sinai Hospital of Greater Miami, 4300 Alton Road, Miami Beach, Florida.
Received December 2, 1969; revision accepted for publication February 10, 1970.
Table 1

**Electrocardiographic Findings**

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Degree of A-V block</th>
<th>P-P (msec)</th>
<th>P-R (msec)</th>
<th>Duration (msec)</th>
<th>Shape</th>
<th>Axis</th>
<th>Site of block*</th>
<th>A-H (msec)</th>
<th>H-V (msec)†</th>
<th>Medications</th>
<th>Symptoms</th>
<th>Follow-up (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>73</td>
<td>F</td>
<td>CHB, 2:1, 1:1</td>
<td>1020</td>
<td>200</td>
<td>80</td>
<td>Normal</td>
<td>N</td>
<td>Distal</td>
<td>90</td>
<td>55</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>75</td>
<td>M</td>
<td>CHB, 2:1, 1:1</td>
<td>730</td>
<td>150</td>
<td>120</td>
<td>RBBB</td>
<td>N</td>
<td>Distal</td>
<td>60</td>
<td>40</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>63</td>
<td>M</td>
<td>3:1, 2:1, 1:1</td>
<td>720</td>
<td>205</td>
<td>150</td>
<td>LBBB, RBBB</td>
<td>LAD</td>
<td>Distal</td>
<td>80</td>
<td>70</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>85</td>
<td>M</td>
<td>5:4, 4:3, 3:2, 2:1</td>
<td>910</td>
<td>260</td>
<td>150</td>
<td>RBBB</td>
<td>LAD</td>
<td>Distal</td>
<td>160</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>97</td>
<td>F</td>
<td>CHB, 3:1, 2:1</td>
<td>780</td>
<td>180</td>
<td>110</td>
<td>RBBB</td>
<td>RAD</td>
<td>Distal</td>
<td>60</td>
<td>90</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>73</td>
<td>M</td>
<td>1:1, 2:1, 5:4, 3:2</td>
<td>760</td>
<td>180</td>
<td>160</td>
<td>LBBB</td>
<td>LAD</td>
<td>Distal</td>
<td>65</td>
<td>70</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>76</td>
<td>M</td>
<td>1:1, 2:1, 1:1</td>
<td>600</td>
<td>270</td>
<td>160</td>
<td>RBBB</td>
<td>RAD</td>
<td>Distal</td>
<td>140</td>
<td>80</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>56</td>
<td>M</td>
<td>CHB, 1:1, 3:2, 4:2</td>
<td>670</td>
<td>200</td>
<td>120</td>
<td>LBBB</td>
<td>N</td>
<td>Distal</td>
<td>90</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>77</td>
<td>M</td>
<td>1:1, 2:1, 3:2</td>
<td>940</td>
<td>255</td>
<td>120</td>
<td>IVCD</td>
<td>LAD</td>
<td>Distal</td>
<td>160</td>
<td>55</td>
<td>Aldoril 25 mg 1 tab/day</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>73</td>
<td>F</td>
<td>CHB, 2:1, 4:1</td>
<td>800</td>
<td>200</td>
<td>70</td>
<td>Normal</td>
<td>N</td>
<td>Distal</td>
<td>80</td>
<td>H-V = 65</td>
<td>H-V = 35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>68</td>
<td>F</td>
<td>1:1, 2:1, 3:2</td>
<td>875</td>
<td>140</td>
<td>80</td>
<td>Normal</td>
<td>LAD</td>
<td>Distal</td>
<td>65</td>
<td>40</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>77</td>
<td>F</td>
<td>2:1, 1:1</td>
<td>930</td>
<td>190</td>
<td>120</td>
<td>RBBB</td>
<td>N</td>
<td>Distal</td>
<td>100</td>
<td>55</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>84</td>
<td>M</td>
<td>2:1, 3:2</td>
<td>950</td>
<td>170</td>
<td>80</td>
<td>Normal</td>
<td>N</td>
<td>Within BH</td>
<td>70</td>
<td>H-V = 70</td>
<td>H-V = 40</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Mobitz type II*

*Circulation, Volume XLII, June 1970*

**Wenckebach type of A-V block during NSR**

<table>
<thead>
<tr>
<th>Follow-up (months)</th>
</tr>
</thead>
</table>
type I and type II blocks.4 Today type I block is commonly known as Wenckebach or Mobitz I, and type II block as Mobitz II block. Several studies 5–11 have been reported relative to second degree heart block. Attempts to correlate experimental findings with individual clinical varieties of second degree A-V block have been limited. Intracardiac recordings from the specialized conducting system12–15 during periods of second degree A-V block (types I and II) offer opportunity to determine the site of block(s). In addition to the analysis of Mobitz type II blocks, we are presenting two cases of typical Wenckebach A-V block with dropped ventricular beats occurring in the His-Purkinje system (HPS). The Wenckebach type of A-V block localized in the main bundle of His or bundle branches has not been previously described in man. This evidence also demonstrates some of the pitfalls of the electrocardiographic (ECG) criteria for the localization of A-V block.

Methods

Fourteen patients with A-V conduction defects were studied during right heart catheterization. The ages of these patients varied from 56 to 97 years. The clinical ECGs in 12 patients (cases 1 to 12, table 1) exhibited Mobitz type II block. One patient (case 13, table 1) with a normal QRS complex demonstrated Wenckebach type of second degree (3:2 and 2:1) A-V block. The other patient (case 14, table 1) showed left bundle-branch block (LBBB) during normal sinus rhythm (NSR) with 1:1 A-V conduction and a normal P-R interval of 0.18 sec. Of the 12 patients with type II block, three exhibited QRS complexes of normal duration (≤0.10 sec).

All 14 patients were studied in the postabsorptive state and were premedicated with 100 mg of pentobarbital (Nembutal) administered intramuscularly 1 hour before catheterization. His bundle (BH) electrograms were recorded as described previously.13,14 The BH recordings were validated by demonstrating the progressive lengthening of the A-H time as the P-P interval was shortened by right atrial pacing at progressively increasing rates (up to 180/min or until Wenckebach cycles developed) or by placement of premature atrial beats. In addition to atrial pacing, in selected cases, bundle of His pacing was utilized to validate the BH deflection and to differentiate it from the right bundle electrogram.15
Case 3. Recording of the His bundle (BH) electrogram in a patient with Mobitz type II block. The block was localized distal to the BH. The rhythm strips I, III, and V1 (leads, L-I, L-III and V1) show period of 3:1 A-V conduction with left bundle-branch block (LBBB) and 2:1 A-V conduction with right bundle-branch block (RBBB) pattern. Also of interest are the 12 ECG leads exhibiting RBBB and a normal axis in presence of partial bilateral bundle-branch block (BBBB), alternating RBBB and LBBB.

(A) Simultaneous recordings of bipolar (BE) electrograms from the area of the A-V junction and standard leads aVR, aVF, and I (aVR, aVF, and L-1) ECG. The first ventricular (V) activity is at the end of the ventricular pacing impulse (VPI) followed by normal sinus beats with 2:1 conduction. The first A (atrial electrogram) wave is followed by a BH deflection with an A-H interval of 140 msec as compared with the following three A-H intervals of 80 msec, suggesting retrograde concealed conduction after the VPI. Each A wave is blocked distal to the BH deflection. The BH to V activation (H-V) time of 70 msec for the two conducted beats is abnormal. The P-R interval of 205 msec and the interval (720 msec) between two consecutive P or A waves (P-P) remain constant.

(B) During periods of 2:1 and 1:1 conduction with a fixed P-P interval (720 msec), the P-R (205 msec), A-H (80 msec), and H-V (70 msec) intervals remain constant. The second A wave is blocked distal to the BH at the same A-H time (80 msec) as the conducted A waves. On this and subsequent figures the paper speed = 100 mm/sec. Interval between time lines = 1 sec.

**Definition of Terms**

The A and the V complexes are evident, respectively, in the atrial and ventricular electrograms recorded from the area of the A-V junction. The BH bipolar electrogram is recorded from the bundle of His. The A-H time is the interval from the first rapid deflection of the A wave to the first rapid deflection of the BH bipolar electrogram. Since the A wave is recorded from the area adjacent to the A-V node, the A-H time represents the conduction time through the A-V node. The His bundle to ventricular activation.
(H-V) time was measured from the first rapid BH deflection to the earliest ventricular depolarization recorded on any of the intracardiac electrograms or standard leads. The site of the block in these studies has been characterized as occurring distal to the site of the recorded BH deflection. For the sake of brevity, it is described as block distal to the BH. We recognize that blocks distal to the site of the recorded BH deflection may still be localized in the distal portion of the BH. On the basis of previous studies in patients with normal A-V conduction, the range for normal A-H time is 50 to 120 msec and for normal H-V time 35 to 45 msec.

Results

Mobitz Type II Block in the His-Purkinje System

In these 12 patients the conduction defect was distal to the recording site of the BH electrogram. Each A wave was conducted through the A-V node and was followed by a BH deflection with a constant A-H time (fig. 1). The A and BH deflections of the blocked beats were not followed by ventricular (V) activity. The P-R interval as well as the A-H time was constant for the conducted QRS complexes preceding and following the blocked P waves. The conduction time through the A-V node (A-H time) as well as the P-R interval for the conducted beats was within normal range in all but three patients (cases 4, 7 and 9). Mobitz II A-V block in a patient with prolonged P-R interval and an abnormal A-H time is shown in figures 2 and 3. The conduction time from the BH to the ventricular activation (H-V) for the conducted beats was abnormal in all except two

![Figure 1](H-V) time was measured from the first rapid BH deflection to the earliest ventricular depolarization recorded on any of the intracardiac electrograms or standard leads. The site of the block in these studies has been characterized as occurring distal to the site of the recorded BH deflection. For the sake of brevity, it is described as block distal to the BH. We recognize that blocks distal to the site of the recorded BH deflection may still be localized in the distal portion of the BH. On the basis of previous studies in patients with normal A-V conduction, the range for normal A-H time is 50 to 120 msec and for normal H-V time 35 to 45 msec.

Results

Mobitz Type II Block in the His-Purkinje System

In these 12 patients the conduction defect was distal to the recording site of the BH electrogram. Each A wave was conducted through the A-V node and was followed by a BH deflection with a constant A-H time (fig. 1). The A and BH deflections of the blocked beats were not followed by ventricular (V) activity. The P-R interval as well as the A-H time was constant for the conducted QRS complexes preceding and following the blocked P waves. The conduction time through the A-V node (A-H time) as well as the P-R interval for the conducted beats was within normal range in all but three patients (cases 4, 7 and 9). Mobitz II A-V block in a patient with prolonged P-R interval and an abnormal A-H time is shown in figures 2 and 3. The conduction time from the BH to the ventricular activation (H-V) for the conducted beats was abnormal in all except two
patients (case 11, fig. 4, and case 2). In one patient (case 10, fig. 5) two BH deflections (BH and BH′) were recorded from sites proximal and distal to the focal lesion in the BH, respectively.

During atrial pacing at progressively higher rates, there was a progressive increase in the degree of block beyond the BH deflection; thus, 2:1 block became 3:1 and 4:1 (figs. 6 and 7) or 3:2 block progressed to 4:2, 5:2, and 5:1 (figs. 8 and 9). In addition, at high atrial pacing rates Wenckebach phenomena in the A-V nodal region (between A and BH) were also seen (fig. 10). The conduction time through the His-Purkinje system (HPS), in other words, H-V time, remained constant for all conducted beats at all paced rates. A unique pattern during NSR and atrial pacing was seen in case 8 (figs. 8 and 9) which showed periods of 3:2, 4:2, and 5:2 conduction. After the dropped P wave, the following P waves were always conducted in pairs. Only when the atrial pacing rate was increased from 140 to 150/min did the conduction change from 5:2 to 5:1 block. Only three (cases 1, 10, and 11) of the 12 patients with Mobitz type II block showed normal QRS complexes (≤0.10 sec; fig. 4, case 11). In two of these three patients (case 10, fig. 5, and case 1) with normal QRS complexes during Mobitz II A-V block and intermittent periods of complete heart block (CHB) distal to the BH, the “idioventricular” QRS complexes were also normal and remained unchanged in comparison to the conducted beats. The remaining nine patients with abnormal QRS complexes (≥0.12 sec) showed LBBB, right bundle-branch block (RBBB), or intraventricular conduction defect (IVCD).

Wenckebach Type of Block in the His-Purkinje System

Two patients (cases 13 and 14, table 1) showed this phenomenon. Case 13 (figs. 11 and 12) with normal QRS complexes showed periods of 3:2 (fig. 11) and 2:1 (fig. 12) Wenckebach type of block during normal sinus rhythm (sinus rate, 63 beats/min). The BH recordings revealed two BH deflections (BH and BH′). Each A wave was conducted to the BH through the A-V node with a normal A-H time of 70 msec. The first conducted beat had an abnormal H-V time of 70 msec with a normal P-R interval of 170 msec. The BH and BH′ electrograms were recorded from sites proximal and distal,
WENCKEBACH AND MOBITZ TYPE II A-V BLOCK

Figure 4

Case 11. His bundle recordings in a patient with a narrow QRS complex (0.08 sec) and Mobitz type II A-V block. The 12-lead standard ECG with a rhythm strip (L-2) shows dropped beats resulting in 2:1 A-V conduction with a constant P-P interval and a fixed P-R interval in the conducted beats.

(A) Simultaneous recordings of BE and L-1 exhibiting the block distal to the BH in the dropped beats. The A-H (65 msec) and H-V (40 msec) times were normal. As the A-A interval was shortened to 375 msec by an induced (PI) premature atrial beat, the A-H time lengthened to 125 msec.

(B) Right bundle-branch (RB) electrogram with an RB to V (RB-V) interval of 20 msec is recorded only in the conducted beats. Note the absence of A and BH deflections because of the catheter placement adjacent to the RB.

respectively, to the focal lesion in the bundle of His. The BH' to ventricular activation (H'-V) time of 40 msec was normal. In figure 11 the time interval between the BH and BH' deflections for the first conducted beat was 30 msec. The following beat was conducted with a prolonged P-R interval (345 msec) and lengthening of the interval between BH and BH' deflections to 205 msec. The A-H and H'-V intervals remained constant at 70 and 40 msec, respectively. The next A wave was blocked distal to the BH deflection and was not followed by BH' and ventricular deflections. This blocked impulse was followed by similar Wenckebach cycles with 3:2 and 2:1 conduction (fig. 12). The atrial rate (P-P interval, 920 to 960 msec) during this phenomenon was essentially unchanged. The shape and normal duration of the QRS complexes remained unchanged despite the lengthening of the BH and BH' interval. The BH and BH' deflections were validated by atrial and BH pacing, respectively.

Case 14 (figs. 13 and 14) with normal sinus rhythm (NSR) and 1:1 A-V conduction showed an LBBB pattern with a normal P-R interval (180 msec). His bundle recordings during NSR showed a normal A-H time (85 msec), but the H-V time (65 msec) was abnormal. During atrial pacing at 110 (fig,
13) and 120 (fig. 14) beats/min Wenckebach phenomena occurred distal to the recording site of the BH. There was progressive prolongation of the H-V time, till finally the impulse was blocked distal to the BH deflection. In the blocked beat the A wave was followed by the BH deflection but not by ventricular activity. Wenckebach cycles with atrial pacing at 110/min showed 4:3 conduction. When the atrial pacing rate was increased to 120/min Wenckebach cycles of 3:2 and 2:1 conduction distal to the BH were observed. Despite production of Wenckebach cycles, the conduction time through the A-V node (A-H) as well as the shape of the QRS complexes remained constant during NSR and

Figure 5

Case 10. Mobitz type II A-V block due to focal lesion in the bundle of His, and with His bundle escape beats. The 12 standard ECG leads on top show a narrow QRS complex.

(A) Simultaneous recordings of BE and L-1, L-2, and L-3. The first atrial (A) impulse is conducted with a normal A-H (80 msec) and an abnormal H-V (65 msec) time. Note the two BH and BH' deflections recorded proximal and distal to focal lesion in the BH, respectively. The H'-V time is 35 msec. The second A wave is blocked distal to the BH deflection and is not followed by BH' and V deflections. The next beat, a BH escape beat, is preceded by BH' deflection at an interval of 35 msec and the sinus A wave is buried in the V of the escape beat. A conducted beat (third QRS complex) follows with the same A-H and H-V conduction times as the first conducted beat. P-R = 200 msec.

(B) Recordings during intermittent periods of complete heart block with an R-R interval = 1510 msec. Each ventricular (V) beat is preceded by a BH' deflection by 35 msec. The atrial impulses blocked distal to the BH are barely visible between the spontaneous ventricular beats (BH' and V) on the BE, but P waves are obvious in the ECG leads L-1, L-2, and L-3. This is due to the slight movement of the catheter into the right ventricular cavity. During His bundle rhythm the H'-V time of 35 msec is similar to H'-V time during the conducted beats.
at each atrial pacing level. In addition the A-H time remained normal despite the Wenckebach cycles.

Discussion

Mobitz Type II A-V Block

Hay and Wenckebach ascribed type I and type II blocks to impaired atrioventricular conduction and depressed ventricular excitability, respectively. Earlier studies failed to offer a precise explanation for this phenomenon, although the causative role of bilateral bundle-branch block was suspected in cases with type II. Katz and Pick attributed type I to a moderate prolongation of both the absolute and relative refractory periods of the A-V junctional tissues while type II was identified with an increase in the absolute refractory phase. Recently Langendorf and Pick have subscribed to the idea that an infra-A-V nodal lesion is the cause of type II A-V block. It had also been suggested that type II may be the result of concealed nodal extrasystoles. Whether similar concealed reexcitation mechanisms within the lower regions of the A-V conduction system (distal HPS) can produce this type of block is unknown. The present study, by demonstrating the increase in block distal to the BH deflection during atrial pacing, suggests that the latter possibility is unlikely since the
Case 6. Simultaneous recordings of BE and lead L-1 during atrial pacing (PI) at progressively increasing rates in the same patient as in figure 6.

(A) Tracings 3:1 A-V conduction with an A-A (P-P) interval of 475 msec are shown. All three atrial (A) impulses were conducted through the A-V node, but two were blocked distal to the BH. The interval between two consecutive QRS complexes (R-R) was 1,425 msec.

(B and C) Periods of 3:1 A-V block (panel B) alternating with 4:1 A-V block (panel C) appeared with R-R of 1,155 and 1,540 msec, respectively, as the A-A interval was shortened to 385 msec. The block remained distal to the BH deflection. The conduction time through the HPS (H-V) remained constant (70 msec) throughout.

Concealed A-V junctional extrasystoles now must be associated with the atrial rate.

Our data also explain the original observations of a constant P-R interval despite dropped beats during type II second degree heart block. All 12 patients in this series with the electrocardiographic diagnosis of type II A-V block demonstrated that each P wave was conducted through the A-V node to the bundle of His at a constant A-H time. The failure in A-V transmission for the dropped P waves (during normal sinus rhythm), irrespective of their number, was localized distal to the recording site of the BH deflection. The
conducted beats had a constant conduction time through the HPS (H-V time). It has been previously\(^1\),\(^2\),\(^28\)-\(^34\) documented that as the P-P interval is shortened, the P-R interval prolongs due to the increased refractoriness of the A-V node, whereas the conduction time through the HPS usually remains constant at all heart rates. The fewer the impulses transmitted through the A-V node, the shorter the P-R interval. However, in type II block the number of impulses transmitted through the A-V node, with or without dropped beats, remains unchanged. Thus, there is a constant A-H time as well as a stable P-R interval for the conducted beats. In contrast, previous studies\(^1\),\(^4\),\(^15\),\(^30\),\(^34\),\(^35\) have shown that type I block occurs proximal to the BH deflection (either in AN, N, or NH regions of the A-V node) and the number of impulses completely transmitted through the A-V node is variable, hence changing the A-H and the P-R intervals.

Our findings support the experimental work\(^35\) and the clinical observations\(^36\) that Mobitz type II blocks are due to lesions in the HPS either in the BH or the bundle branches,
Case 8. Simultaneous recordings of BE and lead L-3 during atrial pacing at progressively increasing rates in the same patient as in figure 8.

(A) Atrial pacing (AP) at 110/min produced periods of 3:2 conduction distal to the BH without any change in the shape of the QRS complexes.

(B and C) With AP at 130 and 140/min the block distal to the BH increased to 4:2 and 5:2 respectively. The relationship of two paired consecutively conducted beats persisted despite the increased block. Note that the second of the paired conducted beats showed slight aberration in the shape of the QRS complex.

(D) With further increase in AP to 150/min the block distal to the BH increased to 5:1 without paired conducted beats. H-V time remained constant throughout.

by demonstrating block distal to the BH deflection. In the majority of cases the abnormal H-V time during the conducted beats (table 1) further confirms disease of the HPS. The patients with narrow QRS complex and type II block localized distal to the BH deflection most probably have His bundle lesions. In case 11 (fig. 4) the recording of a proximal right bundle (RB) deflection, only during the conducted beats as opposed to the beats blocked distal to the BH deflection, further indicates that the block is situated in the distal BH but before its bifurcation into bundle branches. In another patient (case 10, fig. 5, table 1) the recording of “split” BH (BH and BH’ deflections) is a further evidence of this site of lesion. This type of block is not as rare as considered previously.10

In the patients with an abnormal QRS complex and block distal to the BH, the lesion could be either situated in the distal BH or in
WENCKEBACH AND MOBITZ TYPE II A-V BLOCK

Figure 10

Case 12. Simultaneous recordings of BE and lead L-1 in a patient with Mobitz II A-V block localized distal to the BH with two sites of block occurring concomitantly during AP.

(A) Normal sinus rhythm with sinus arrhythmia and a normal P-R interval. The third A wave is suddenly dropped and is blocked distal to the BH with an A-A interval of 940 msec in comparison to the conducted beats (first and last) at shorter A-A intervals of 860 and 910 msec, respectively. The A-H time (100 msec) and the H-V time (55 msec) were normal and abnormal, respectively.

(B) Mobitz I between the A and BH complexes is shown together with Mobitz II between the BH and V complexes. During AP at 135/min, the A-V block increased to 3:1, as an example of Mobitz II block between the BH and V complexes. The majority of the atrial (A) impulses are conducted beyond the A-V node, and are blocked distal to the BH. A 6:5 Wenkebach type of block is also shown, as exhibited by progressive prolongation of the A-H interval from 160 to 360 msec until finally the A is blocked proximal to the BH, illustrating Mobitz I block. Note the variable P-R intervals (260 and 415 msec) for the conducted beats due to the mixed sites of block.

(C) As the AP was further increased to 155/min, the A-V block increased to 4:1 with the majority of the atrial impulses still blocked distal to the BH. Wenkebach type of block through the A-V node also increased to 4:3 (6:5 in panel B). The P-R intervals in the conducted beats were again variable. The conduction time through the His-Purkinje system (H-V = 55 msec) remained constant throughout.

the bundle branches. For practical purposes these cases, especially the cases in which the H-V time and the QRS complex are abnormal during the conducted beats, most likely represent bilateral bundle-branch block. The present series indicates that all types of abnormal QRS complexes (RBBB, LBBB, and IVCD) were associated with this type of block. Of interest is case 2 which showed a pattern of RBBB with a normal axis in the conducted beats and a normal H-V time. The site of block in case 2 is probably in the distal portion of the His bundle since an abnormal H-V time would have been anticipated in the conducted beats if the left bundle branch were also diseased.

The cases of type II block distal to the BH and a constant H-V time, regardless of the
Case 13. His bundle recordings in a patient with a narrow QRS complex during 3:2 Wenkebach type of block within the BH.

The 12 standard ECG leads are shown on top. Simultaneous recordings of BE with ECG leads L-1, L-2, and L-3 show 3:2 Wenkebach type of block with prolongation of the P-R interval from 170 to 345 msec before the dropped beat and followed by similar cycles. Two BH deflections (BH and BH') were recorded, one proximal and one distal to the focal lesion in the BH. The prolongation of the P-R interval was contributed by the delay between the BH and BH' deflections which increased from 30 to 205 msec. The A-H time (70 msec) and H'-V time (40 msec) remained constant throughout. During periods of 3:2 Wenkebach type of block the atrial rate was slightly slower (A-A = 940 to 960 msec) in comparison to periods of 2:1 block (A-A = 920 to 930). (See fig. 12.)

In the majority of the type II cases the P-R interval was normal. In these cases the A-H time was within the normal range, indicating normal conduction through the A-V node and lack of involvement of this region by the disease process. Similar observations were made in a recent histopathologic study of patients with CHB due to diseased HPS, in whom the A-V node was shown to be normal.

When the block occurs at two different sites simultaneously, that is in the A-V node and the HPS, any combination of electrocardiographic patterns can be seen. The diagnosis of mixed lesions such as Wenkebach type of block in the A-V node and type II block in the HPS is quite difficult on the basis of a surface electrocardiogram (fig. 10). Although the given example exhibited mixed lesions during atrial pacing only, the possibility of such a phenomenon occurring spontaneously does exist and may mimic CHB.

A single case purported to be Mobitz II in a patient with LBBB and 2:1 A-V block has recently been reported. On the basis of localization of block distal to the BH, the case does represent type II block. But in the reported case as well as another study, the electrocardiographic criteria for type II block were inadequate because of the presence of a fixed 2:1 A-V block. It should be stressed that 2:1 A-V block per se does not satisfy the electrocardiographic criteria of type II block (constant P-R with or without blocked impulses) unless periods of changing conduction (3:1, 1:1, or 3:2) with the same P-R interval (constant P-P interval) are demonstrated. During BH recordings in 350 cases, it has been observed that fixed 2:1, 3:1 A-V block
may be localized either proximal or distal to the BH, irrespective of the shape and duration of the QRS complexes. In cases in which only one bundle branch such as LBBB is involved, lesions can subsequently develop in the A-V node (rather than in the opposite bundle) giving rise to 2:1 or 3:1 A-V block or even CHB proximal to the BH. Reports of studies have emphasized that "bilateral bundle-branch block should always be suspected whenever second degree A-V block is associated with either a right or left bundle-branch block pattern." As noted above, the P-R interval merely reflects the sum of the conduction disturbances which occur during impulse transmission through the atria, A-V node, and HPS. The present study shows the limitations of these concepts by demonstrating in addition that even in type I block (see below) the site of the lesion cannot always be accurately localized from the electrocardiogram.

Case 8 (figs. 8 and 9) with conduction ratios of 3:2, 4:2, and 5:2 may be an example of Wedensky phenomenon. In this case the second of the paired conducted beats may be dependent in some way on the conduction of the preceding beat. With atrial pacing up

Figure 12

Case 13. His bundle recordings in the same patient as in figure 11 during 2:1 Wenckebach type of block within the BH. The 12 standard ECG leads on top show sinus rhythm and narrow QRS complex.

(A) Simultaneous recordings of BE with ECG leads L-1, L-2, and L-3, showing 2:1 A-V conduction. The blocked atrial (A) impulse was localized distal to the BH with a normal conduction through the A-V node (A-H = 70 msec). Distal to the blocked BH, note a slow deflection with a lower amplitude probably indicating decremental conduction through the diseased HPS (see fig. 11). The conducted beats had an abnormal H-V time (70 msec) with an intra-BH (BH-BH') delay of 30 msec with a H-V' time of 40 msec. The P-R interval for the conducted beats was normal (170 msec).

(B) During AP = 120/min, the block distal to the BH increased to 3:1 and the A-H interval lengthened to 115 msec (70 msec in panel A). The H-V time (70 msec) in the conducted beats remained constant.
Case 14. His bundle recordings in a patient with normal sinus rhythm (NSR) with 1:1 conduction and LBBB who developed Wenckebach type of A-V block distal to the BH during AP. LBBB is evident in the 12 standard ECG leads on top.

(A) Simultaneous recordings of BE and L-1, L-2, and L-3 during NSR at a rate of 92/min with 1:1 A-V conduction. Each A wave is conducted with a normal A-H (85 msec) but an abnormal H-V (65 msec) time. P-R = 180 msec.

(B) During AP = 110/min Wenckebach type of A-V block (4:3) developed distal to the BH. There was progressive prolongation of the P-R interval (185 to 210 msec) before the dropped beat occurred. This prolongation of the P-R interval was contributed by the progressive prolongation of the H-V time (65 to 90 msec) with eventual block distal to the BH. Each atrial (A) impulse was conducted through the A-V node to the BH with a constant A-H interval of 90 msec.

to 140/min the number of blocked impulses preceding the first conducted beat increased and the pairs of conducted beats persisted. However, at an atrial pacing rate of 150/min, the second beat of the pair was no longer conducted.

Previous reports10, 11 have indicated a high incidence of Stokes-Adams attacks with type II blocks. The present series supports this observation. These patients are likely to develop chronic complete heart block due to the pathologic involvement either of the BH or of both bundle branches. Patients 6 to 8 and 10, when studied 6 mo later, showed complete heart block distal to the BH.

Our data indicate the existence of His
bundle lesions as a cause of Mobitz type II A-V blocks with a subsidiary pacemaker (His bundle rhythm) arising above the bifurcation of the bundle of His. In addition, our findings confirm the clinical observations that Mobitz type II blocks are usually associated with bilateral bundle-branch block.

**Wenckebach Type of Block**

Any site in the specialized conduction system where a difference in the functional refractory period occurs can be expected to produce block if an impulse reaches a region which has not repolarized sufficiently. Usually the functional refractory period of the A-V node (proximal A-V junction) is longer than that of the BH and bundle branches. Atrio-ventricular block, especially of the Wenckebach type, is generally considered to occur in the A-V node. Experimental and clinical work has indicated that HPS is also an important site of block. A recent editorial suggested that type I A-V block is a characteristic of the disease in the A-V nodal region. However, recent case reports have described “Wenckebach cycles” in the bundle branches as indicated by progressive prolongation of the QRS duration at a constant atrial rate. These cases did not exhibit progressive prolongation of the P-R interval but did show such prolongation of the P-S interval (P to S wave of the QRS complex). In contrast cases 13 and 14 in the present study demonstrated the classic prolongation of the P-R interval as well as the dropped beats characteristic of the Wenckebach phenomenon. Furthermore, the site of this delay and block in conduction was in the BH (case 13, figs. 11 and 12) or main right bundle branch or both (case 14, figs. 13 and 14) and not in the A-V node. The latter case showed LBBB during NSR. With atrial pacing at 110/min, the Wenckebach type of A-V block developed with progressive prolongation in the H-V time (HPS) with subsequent dropped beats exhibiting block distal to the BH deflection. The shape and duration of the QRS complexes remained unchanged from the control LBBB pattern. This Wenckebach type of A-V block was either occurring in the distal BH or the main right bundle branch. Since no “split” BH was recorded, this case
may be an example of type I A-V block occurring in the right bundle.

These findings demonstrate that in some cases localization of block on the basis of the clinical electrocardiogram is not possible. The clinical significance of these findings should be considered in the light of recent suggestions that patients with type I block during acute myocardial infarction need not be treated with pacemakers unless the ventricular rate is below 50/min. This statement was made on the assumption that type I A-V block always occurs in the A-V nodal region. However, Wenckebach type of A-V block in the HPS is probably not rare. The Wenckebach type of second degree A-V block in the HPS has not previously been described because of the unavailability of BH recordings. It must be emphasized that this phenomenon probably occurs only during disease states of the HPS. The prognosis for the two patients in our series with type I A-V block should not be different from that for patients with type II A-V block because they both have infra-A-V nodal lesions within the HPS. Actually, CHB developed in case 14 within 6 mo of the initial study. Therefore, CHB may follow both Mobitz I and Mobitz II in the HPS.

The type I A-V block is simply an electrocardiographic sign which can be manifested by lesions involving different regions of the A-V conduction system, in other words, A-V node and HPS. Therefore, the distinction in second degree A-V blocks (type I and type II) perhaps should be made on the basis of the site of block such as nodal (A-H block) or HPS (H-V block) rather than Mobitz type I or type II. This differentiation is of value from a prognostic point of view. The findings of CHB distal to BH in four of 12 patients with Mobitz type II block when restudied after a 6-mo interval are of therapeutic significance.

References
1. Hay J: Bradycardia and cardiac arrhythmia produced by depression of certain of the functions of the heart. Lancet I: 139, 1906
10. Donoso E, Adler LN, Friedberg CK: Unusual forms of second degree atrioventricular block, including Mobitz type II block, associated with the Morgagni-Adams-Stokes syndrome. Amer Heart J 67: 150, 1964
Faisseau de His-Tawara. Paris, Masson et Cie, 1931

20. YATER YM, CORNELL VH, CLAYTON T: Auriculo-
ventricular heart block due to bilateral bundle-
branch lesions: Review of the literature and
report of three cases with detailed histopatho-
logical studies. Arch Intern Med 57: 132, 1936

21. LEV M, UNGER PN: Pathology of the conduction
system in acquired heart disease: I. Severe
atrioventricular block. Arch Path (Chicago)
60: 502, 1955

22. LENÈGRE J: Contribution à l’étude des blocs de
branche comportant notamment les confronta-
tions électriques et histologiques. Paris, J. B.
Baillière et Fils, 1958

23. LENÈGRE J, MOREAU PH: Le bloc auriculo-
ventriculaire chronique: Étude anatomique,
clinique et histologique. Arch Mal Cœur 56:
867, 1963

24. MCNALLY ME, BENCHIMOL A: Medical and
physiological considerations in the use of
artificial cardiac pacing: Part I. Amer Heart J
75: 380, 1968

25. KATZ LN, PICK A: Clinical electrocardiography:
Part I. The arrhythmias. Philadelphia, Lee and
Fibiger, 1956

26. LANGENDORF R, PICK A: Atrioventricular block,
type II (Mobitz): Its nature and clinical

27. LANGENDORF R, MEHLMAN JS: Blocked (non-
conducted) A-V nodal premature systoles
imitating first and second degree A-V block.
Amer Heart J 34: 500, 1947

28. LISTER JW, STEIN E, KOSOWSKY BD, ET AL:
Atrioventricular conduction in man: Effect of
rate, exercise, isoproterenol, atropine on P-R
interval. Amer J Cardiol 16: 516, 1965

29. LINHART JW, BRAUNWALD E, ROSS J JR:
Determinants of the duration of the refractory
period of the atrioventricular nodal system in

30. HOFFMAN BF, CRANEFIELD PF: Electrophysiol-
ogy of the Heart. New York, McGraw-Hill
Book Co., 1960, p 132

31. ROSENBLUTH A: Mechanism of the Wenckebach-
Luciani cycles. Amer J Physiol 196: 491,
1958

32. HOFFMAN BF, PAES DE CARVALHO A, DE MELLO
WC, ET AL: Electrical activity of single fibers
of the atrioventricular node. Circulation Re-
search 7: 11, 1959

33. CRANEFIELD PF, HOFFMAN BF, PAES DE
CARVALHO JF: Effect of acetylcholine on single
fibers of the atrioventricular node. Circulation
Research 7: 19, 1959

34. ALANIS J, LOPEZ E, MANDOKI J, ET AL:
Propagation of impulses through the atrioven-
tricular node. Amer J Physiol 197: 1171, 1959

35. WATANABE Y, DREIFUS LS: Second degree A-V

36. DAVIES M, HARRIS A: Pathological basis of
primary heart block. Brit Heart J 31: 219,
1969

37. STOCK RJ, MACKEN DL: Observations on heart
block during continuous electrocardiographic
monitoring in myocardial infarction. Circula-
tion 38: 987, 1968

38. NARULA OS, SCHRLAG BJ, JAVIER RP, ET AL:
Analysis of the A-V conduction defect in
complete heart block utilizing His bundle
electrograms. Circulation 41: 437, 1970

39. LEPESSCHIN E: Electrocardiographic diagnosis
of bilateral bundle branch block in relation to
heart block. Progr Cardiovasc Dis 6: 445,
1964

40. LOPEZ JF: Electrocardiographic findings in
patients with complete heart block. Brit Heart J
30: 20, 1968

41. MULLER OF: Electrocardiographic interpretation
of A-V block. In: Mechanisms and Therapy of
Cardiac Arrhythmias, edited by LS Dreifus, W
Likoff, and JH Moyer. New York, Grune &
Stratton, Inc., 1966, p 461

42. CASTELLANOS A JR, LEMBERG L, JOHNSON D, ET
AL: Wedensky effect in the human heart. Brit
Heart J 28: 276, 1966

43. SCHAMROTH L, FRIEDBERG HD: Wedensky
facilitation and the Wedensky effect during
high grade A-V block in the human heart.
Amer J Cardiol 23: 893, 1969

44. MOE GK, MENDEZ C, HAN J: Aberrant A-V
impulse propagation in the dog heart: A study of
functional bundle branch block. Circulation
Research 16: 261, 1965

45. FRIEDBERG HD, SCHAMROTH L: The Wencke-
bach phenomenon in left bundle branch block.
Amer J Cardiol 24: 591, 1969

46. ROSENBAUM MB, GERARDRO NJ, LEVI RJ, ET AL:
Wenckebach periods in the bundle branches.
Circulation 40: 79, 1969
Wenckebach and Mobitz Type II A-V Block Due to Block Within the His Bundle and Bundle Branches

ONKAR S. NARULA and PHILIP SAMET

Circulation. 1970;41:947-965
doi: 10.1161/01.CIR.41.6.947

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1970 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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/41/6/947

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/