Observations on Atrioventricular Conduction in Patients with Bilateral Bundle-Branch Block

By R. M. Schulenburg, M.D., and Dirk Durrer, M.D.

SUMMARY

Atrioventricular conduction was studied in three patients, each representing a different grade of bilateral bundle-branch block. His bundle activity was recorded by a catheter technic. Two of the three patients had experienced Adams-Stokes attacks.

The first patient had right bundle-branch block with left axis deviation at the time of catheterization. Increases in the atrial driving rate resulted in progressive lengthening of the A-H interval, the H-V interval remaining constant and within normal limits. Second degree A-V block occurred at atrial rates higher than 140/min. The site of this block was located above the His bundle. On no occasion was the impulse blocked distally to the His bundle.

The second patient had a Mobitz type II A-V block with complete left bundle-branch block and a prolonged P-R interval in the conducted beats. The block of the nonconducted beats could be located distal to the His bundle. H-V block occurred more frequently when the atrial rate was increased. Second degree A-H block occurred at rates above 140/min. At these rates interesting patterns were seen due to conduction impairment at two levels in the A-V conduction system.

The third patient presented with complete A-V block. The site of this block could be located at a level lower than the His bundle. A-H conduction studied with increases of atrial rate and with atrial premature beats seemed to be normal. There was evidence for the existence of retrograde V-A conduction in this patient.

Additional Indexing Words:

- Complete A-V block
- Mobitz type II A-V block
- Wenckebach phenomenon
- Right bundle-branch block with left axis deviation
- V-A conduction in A-V block
- His bundle recordings
- Atrial pacing
- Atrial premature beats
- A-V conduction
- Heart rate

In recent years it has been appreciated that some of the more advanced types of A-V conduction disturbance may be caused by functional abnormalities of both bundle branches rather than of the A-V node.1-8 In its most extreme form bilateral bundle-branch block presents itself as a complete A-V block. The electrocardiographic features of the potential forms of bilateral bundle-branch block have been defined,1,2 and the potential progression into the complete form has been stressed.1,2,4,6-8 Direct catheter recording of His bundle potentials in the human heart, as described by Giraud and associates,9 Watson and co-workers,10 and later by Scherlag’s group,11 who developed a simple recording technic, opened a way to the study of impulse propagation in the specific conduction tissue in patients suspected of having bilateral bundle-branch block. Two cases of bilateral bundle-branch block studied with this method were mentioned by Damato and associates.12 The physiologic characteristics of A-V conduction in the intact human heart can be analyzed by investigating the influence of changes in...
driving rate of the heart and of accurately timed premature beats elicited during regular driving, upon the time relations of atrial, His bundle, and ventricular action potentials.

In three patients with bilateral bundle-branch block, we studied A-V conduction in this way in order to analyze the changes in conduction in the specific conduction system yielding this condition.

Methods

The heart was stimulated with a bipolar electrode catheter, positioned in the right atrium with its tip against the atrial septum. The method described by Scherlag and associates11 was used for recording His bundle activity. A tripolar electrode catheter introduced into the right femoral vein was positioned across the tricuspid valve orifice. In this catheter one electrode was located at the tip. The two other electrodes were a distance of 1.2 and 2.4 cm respectively from the tip electrode. A bipolar electrogram was derived from two adjacent electrodes. This electrogram and leads I, II, III, V1 and V6 and an intra-atrial lead were recorded on an Elema direct-writing recorder and stored on tape with an Ampex FR 1300 tape recorder. The intervals between atrial activation and the His electrogram (A-H interval), between the His electrogram and the ventricular activation (H-V interval), and between atrial and ventricular activation (A-V interval) were measured; for this the first visible deflection of each activation in any of the used leads was employed. The stimulator described earlier13 was used. The atrial septum was subsequently stimulated at stepwise increasing rates. In some experiments extra stimuli were applied after each eighth stimulus of the basic rhythm during regular driving of the heart.

Results

Case 1

This 75-year-old man was admitted to our clinic 2 hours after he experienced a typical Adams-Stokes attack. The day before admission he had taken some digoxin tablets on his own initiative because he observed an irregularity in his radial pulse. On admission the ECG showed sinus bradycardia (rate, 60/min) with a PR interval of 0.18 sec. The QRS complex showed a complete right bundle-branch block (QRS duration, 0.12 sec) and left axis deviation (−80°). There were no signs of previous myocardial infarction. Four days after admission (in the meantime no digitalis was given), heart catheterization for the study of A-V conduction was performed. The right atrium was stimulated at the middle of the atrial septum. The influence of increases in rate upon A-V conduction was studied.

Table 1 shows some of the results in this patient. At sinus rhythm (rate, 61/min) the A-H interval was 120 msec and the H-V interval was 55 msec (fig. 1A). During driving of the atrium at the rate of 78/min the A-H interval was somewhat shorter, 100 msec. This is due to the fact that the atrium was stimulated in the middle of the atrial septum. The H-V interval was unchanged. Increasing the rate resulted in a gradual lengthening of the A-H interval, the H-V interval remaining constant (55 msec, fig. 1B and C). At the rate of 143/min (cycle length, 420) a Wenckebach phenomenon was observed (fig. 2). This record clearly shows that the Wenckebach phenomenon occurred at a level upstream from the His bundle. The first five atrial beats in this record are conducted to the ventricle with a progressive delay in the region between atrium and His bundle, while the H-V interval is the same for each beat. The sixth atrial beat is not conducted to the His bundle. The seventh atrial beat is again conducted to the ventricles with the same time characteristics as the first atrial beat. The small variation in the configuration of the His complex in this figure was due to discrete variations in location of

Table 1

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<th>Rate</th>
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<th>H-V interval (msec)</th>
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<tr>
<td>AP</td>
<td>174</td>
<td>345 2:1 A-H block</td>
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Abbreviations: SR = sinus rhythm; AP = atrial pacing.

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Figure 1

Case 1. Complete right bundle-branch block and left axis deviation. (A) Sinus rhythm rate, 61/min; A-H interval, 120 msec; H-V interval, 55 msec. (B) Right atrial driving, rate, 91/min; A-H, 115 msec; H-V, 55 msec. (C) Right atrial driving, rate, 123/min; A-H, 145 msec; H-V, 55 msec. (D) Right atrial driving, rate, 174/min; 2:1 A-H block. The conducted beats have an A-H interval of 125 msec and an H-V interval of 55 msec. Because the A-H interval of the conducted beats was longer than that found at rate 90/min, concealed conduction in the A-V node of the blocked atrial beats has to be assumed.

the electrodes which can be deduced from the accompanying variations in the configuration of the ventricular complex in the same lead. At rate 174/min (cycle length, 345 msec) a 2:1 A-H block occurred (fig. 1D). The conducted atrial beats had an A-H interval of 125 msec and an H-V interval of 55 msec.
Case 2

This 72-year-old man had experienced several Adams-Stokes attacks, but on admission the ECG showed sinus rhythm (rate, 75/min) with a Mobitz type II A-V block. The P-Q interval of the conducted beats was 0.24 sec. The ventricular complexes had a complete left bundle-branch block configuration with a QRS width of 0.14 sec. Figure 3 shows the Mobitz II type of A-V block occurring during regular driving of the right atrial septum at a rate of 80/min. The fifth atrial beat is followed by a His potential with the same A-H interval as the preceding beats, but there is no subsequent ventricular activation. The A-H and H-V intervals in the preceding beats are constant. The H-V interval was 100 msec.

When the driving rate was increased (stepwise increments of 10/min), a gradual prolongation of the A-H interval was found, varying between 110 at rate 80 and 180 at rate 130/min. Blocking of conduction at a level below the His bundle occurred more frequently at the higher driving rates; sometimes a 3:2 H-V block was seen over longer periods of time. The H-V interval of the conducted beats showed a variation between 90 and 110 msec.

At a rate of 140/min (fig. 4) a Wenckebach phenomenon, occurring at a higher level than the His bundle, was found. There was an increase in A-H interval in three successive atrial beats. The fourth beat was not conducted to the His bundle. The third atrial beat was blocked each time at a lower level than the His bundle.

Since correct positioning of the electrode catheter in order to obtain good His bundle recordings was very difficult in this patient, the variations in configuration of the His electrograms are presumably caused by slight variations in the position of the recording electrodes, due to the abnormal mechanical interaction of atrial and ventricular contractions. This was also seen in figure 5.

When the atrium was driven at 170/min, three patterns could be discerned successively.
(fig. 5). In the first pattern (fig. 5A) two of each three atrial beats were conducted to the His bundle, the first with an A-H interval of 140 msec, the second with an A-H interval of 205 msec. Only this second His activation was followed by a ventricular complex (H-V interval, 110 msec).

The second pattern showed a 2:1 A-H block and a 2:1 H-V block (fig. 5B), resulting in a 1:4 ventricular response. The A-H interval of the conducted beats was 170 msec, and the H-V interval was 95 msec. Transition to a pattern with a 2:1 A-H block and a 1:1 H-V response (fig. 5C) was found. The A-H interval was at that time 165 msec; the H-V, 110 msec.

Case 3

This 81-year-old man was admitted to our department because of complete atrioventricular block accompanied by fatigue and dyspnea on exertion. There was no history of Adams-Stokes attacks. Eight months before admission the ECG showed a sinus rhythm (rate, 56/min) with 1:1 atrioventricular conduction (P-R interval, 0.22 sec), and complete left bundle-branch block. On admission he had complete atrioventricular block with a ventricular rate of 34/min. In this case, during driving of the atrium each atrial complex was followed by a His electrogram (fig. 6). The propagation of the atrial impulse obviously was blocked at a lower level than the His bundle. When the atrial rate was increased, the A-H interval showed gradual prolongation from 130 msec at an atrial rate of 90/min to 215 msec at a rate of 140/min. At a rate of 145/min (fig. 7) and at higher rates (fig. 6) an A-H Wenckebach phenomenon was seen. A 2:1 A-H block occurred at a rate of 200/min. The A-H interval of the conducted atrial beats was 180 msec. This interval is 40 msec longer
than when the atrium was driven at a rate of 100/min. This finding suggests concealed conduction within the A-V node of the atrial beat not followed by a His activation.

The A-H conduction was further studied in this case by the application of premature stimuli to the right atrium. The atrium was driven regularly with a cycle length of 590 msec. After each eighth stimulus an extra stimulus was applied. The time interval of this extra stimulus $A_2$ to the foregoing stimulus of the basic rhythm $A_1$ was decreased gradually. Figure 8 shows that shortening of the $A_1$-$A_2$ interval resulted in progressive lengthening of the $A_2$-$H_2$ interval. The A-H interval of the atrial beats of the basic rhythm was 130 msec. At an $A_1$-$A_2$ interval of 300 msec, the $A_2$-$H_2$ interval was 290 msec. The atrium was found to be refractory at an $A_1$-$A_2$ interval of 275 msec. $A_2$ complexes with an $A_1$-$A_2$ interval only 5 msec longer (280 msec) were still conducted to the His bundle. The marked broadening of the $A_2$ complex at this $A_1$-$A_2$ interval, however, suggests that the impulse propagation in the atrium, which presumably is still partly refractory as a result of activation $A_1$, is considerably retarded. It may be postulated, therefore, that the arrival of the impulse at the atrionodal junction is delayed, and an estimation of the functional refractory period of the upper part of the A-V conduction system, therefore, is not possible.

Figure 9 demonstrates the remarkable finding that ventriculo-atrial conduction was
possible in this patient with complete antegrade bilateral bundle-branch block. The right ventricle was driven with a cycle length of 665 msec (rate, 90/min). Over longer periods of time the ventricular complexes were followed at 160 msec by atrial complexes. During driving at higher rates ventriculo-atrial conduction still occurred; however, it was not in a 1:1 fashion, and interference with sinoatrial activity complicated the picture. The occurrence of ventriculo-atrial conduction in patients with a high degree of atrioventricular block is not an isolated phenomenon and has been observed several times in our laboratory.14, 15

Discussion
The combination of complete right bundle-branch block and left axis deviation, as seen in case 1, is considered to be one of the possible manifestations of bilateral bundle-branch block.1, 6, 7 Anatomic studies by Lenègre4 and Unger and associates8 on hearts of patients with this electrocardiographic pattern showed widespread pathologic changes in both bundle branches with minimal or no involvement of the A-V node and common bundle. The left axis deviation is thought to be caused by a conduction disturbance in the anterior division of the left bundle-branch block. This view is supported by the studies of Grant16 and Pryor and Blount17 on left axis deviation. Moreover, the electrocardiographic pattern of complete right bundle-branch block and left axis deviation has been produced experimentally in dog and primate hearts by Watt's group18 by division of the right bundle branch and the anterior fascicle of the left bundle branch.

Several authors1, 4, 7, 8 described the possible progression of this form of partial bilateral bundle-branch block into complete bilateral bundle-branch block eventually accompanied by Adams-Stokes seizures.

Our patient 1 experienced an Adams-Stokes attack shortly before admission. Possibly the digoxin ingested by the patient on his own initiative was responsible for a depression of the conduction in the posterior fascicle of the left bundle branch, resulting in temporary complete atrioventricular block. At the time of catheterization 4 days after admission no complete atrioventricular block could be elicited by carotid sinus massage or by increasing the atrial rate. The gradual prolongation of the A-V conduction time following increases in driving rate of the atrium was caused by changes in the conduction time from atrium to His bundle (A-H interval). The conduction times from His bundle to ventricle (H-V interval) during sinus rhythm with a rate of about 60/min and during driving of the atrium with rates up to 140/min were constant and compared to the findings of Damato and co-workers19 and those of our laboratory,20 were within normal limits. At rates between 140 and 170/min an A-H Wenckebach phenomenon occurred. This does not necessarily indicate a disturbance of conduction of the A-V nodal level. In our experience a Wenckebach phenomenon may be observed in apparently normal hearts during driving of the atrium with rates as low as 130/min. At a rate of 174/min a 2:1 A-H block was seen. At all times a His activation was followed by a ventricular activation. Thus, in this patient with partial bilateral bundle-branch block, evident by complete right bundle-branch block and left axis deviation, it was not possible to produce complete interruption of atrioventricular conduction by increasing the atrial rate.

In case 2 a combination of prolonged P-R interval and complete left bundle-branch block suggested (according to Lepeschkin1 the existence of bilateral bundle-branch block. Moreover, this patient had a Mobitz type II A-V block. The frequent occurrence of this type of second degree A-V block in patients with right bundle-branch block and left axis deviation has led to the view that the site of the block might be located in the bundle branches.21 Experiments of Watanabe and Dreifus22 on rabbit heart preparations showing a Mobitz type II A-V block supported this view. In our case of Mobitz type II block, His bundle recordings showed that those atrial beats which were not conducted to the ventricle still were followed by a His complex with the same A-H interval as the preceding
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Case 3. Complete A-V block. Right atrial driving; lead V1 and "His lead." Each atrial complex is followed by His activation; thus, the site of the A-V block is located at a level lower than the His bundle. Increases in driving rate are accompanied by increases in A-H interval. At rates higher than 140/min an A-H Wenckebach phenomenon occurs. A 2:1 A-H block is seen at the rate of 200/min.

beats (fig. 3). The impulse, therefore, was blocked at a level lower than the His bundle, presumably in the right bundle branch. The A-H and H-V intervals of the preceding beats were constant. The H-V interval (100 msec) was prolonged, also suggesting that the

Figure 6

Case 2. Right atrial driving, rate, 170/min. (A) A-H Wenckebach and 2:1 H-V block; H-V interval of the conducted beats, 110 msec. (B) 2:1 A-H block and 2:1 H-V block; H-V interval of the conducted beats, 95 msec. (C) 2:1 A-H block and 1:1 H-V conduction with H-V interval of 110 msec.
impulse conduction in the right bundle branch was compromised. At higher atrial rates blocking of A-V conduction distally to the His bundle occurred more frequently. Impulse conduction at a higher level (in the A-V node, in the His bundle, or in both structures) also became impaired at atrial rates higher than 140/min. At rates 140 to 170 an A-H Wenckebach phenomenon was found (fig. 4); at rate 170 a 2:1 A-H block was also seen (fig. 5B and C). This is in accordance with the microelectrode studies of Watanabe and Dreifus indicating that in the Wenckebach type of A-V conduction disturbance the delay occurs within the A-V node. Conduction disturbances at two levels at rate 170/min in our case gave rise to the interesting combinations of 3:2 A-H Wenckebach with 2:1 H-V block, 2:1 A-H block with 2:1 H-V block, and 2:1 A-H block with 1:1 H-V conduction.

As mentioned before, the occurrence of A-H conduction disturbances at an atrial rate of 140 and higher does not necessarily implicate a functional abnormality of the A-V node. Although in this patient involvement of the A-V node to some degree cannot be fully excluded, it is obvious that the main site of the pathologic process has to be sought in both bundle branches, leading to complete block in the left bundle branch and conduction impairment in the right bundle branch. At the time of preparation of this paper we found no published cases of Mobitz II A-V block studied with His bundle recordings in the literature. The case described by Damato and associates, actually showing a 2:1 A-V block (fig. 9 in their paper) did not exhibit the pattern generally considered to be typical for Mobitz type II A-V block, in which one or more ventricular beats are dropped occasionally following cycles with fixed P-R intervals.

In case 3 the recording of a His complex after each atrial complex made it clear that the existing complete A-V block was in fact a complete bilateral bundle-branch block. An
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Figure 8
Case 3. Right atrial driving with cycle length 590 msec. After each eighth beat (A1) of this rhythm an extra stimulus (A2) was applied to the right atrium by way of the same electrodes. The A1A2 interval was gradually shortened. Only A1 and A2 are shown. His lead. The A-H interval of the beats of the basic rhythm (A1H1) was 130 msec. Shortening of A1A2 is accompanied by a progressive lengthening of A2H2. See text.

analogous case was described by Damato's group in 1969 (fig. 10 of their article).12 In our case we examined the effect of stepwise increases in atrial rate and of atrial premature beats on A-H conduction. The A-H interval was normal at an atrial rate of 90/min. The gradual prolongation of the A-H interval accompanying stepwise increments of rate did not qualitatively or quantitatively differ from findings in patients with normal atrioventricular conduction.

Atrial premature beats elicited during regular driving of the atrium with a cycle length of 590 msec were conducted to the His bundle even while the interval to the preceding atrial beat was as short as 300 msec (fig. 8). This finding makes an important participation of the A-V node in the pathologic process improbable. It is, therefore, very likely that in this patient also the conduction disturbance was confined to the bundle branches. The complete form of bilateral bundle-branch block in this patient was preceded by left bundle-branch block.

Each of the three patients described showed a different grade of bilateral bundle-branch block. The conduction through the A-V node, as studied by changing the driving rate and by the application of atrial premature beats during regular driving rate and by the application of atrial premature beats during regular driving of the atrium (case 3), was thought to be within normal limits in all three cases. Possibly they also represent different stages in the development of complete atrioventricular block. The recording of His bundle electrograms was of great value for the understanding of atrioventricular conduction disturbances in these three patients.
Figure 9

Case 3. Retrograde (V-A) conduction. Right ventricular driving, rate, 90/min. As the right atrial and "His leads" show, each ventricular complex is followed at 160 msec by an atrial complex.

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