Problems in the Recognition of Conduction Disturbances in the His Bundle

By Reinier M. Schuilenburg, M.D., and Dirk Durrer, M.D.

SUMMARY

Two cases with conduction disturbances in the His bundle are described. In each it was not possible to obtain both components, H and H', of the "split" His bundle potential at one time from a single bipolar electrode catheter with an interelectrode distance of 10 mm. Initial failure to record the proximal His component (H) led to the incorrect diagnosis of block located within the atrioventricular (A-V) node, of third degree, in patient A, and of first to second degree in patient B. However, careful withdrawal of the catheter resulted in the appearance of proximal components (H), accompanied by disappearance of the distal components (H') of the His bundle potential. Apparently the lesion in the His bundle and the recording electrodes were spatially related in such a way that the bipolar electrode could not override the lesion. In view of these findings it is advocated that in patients with A-V conduction disturbances the His bundle catheter should be withdrawn carefully, if the initial recording reveals His bundle potentials bearing a time relation to the ventricular complexes, or advanced carefully, if these His potentials are related to the atrial activations, in order to demonstrate the (co)existence of a lesion in the His bundle. Search for a proximal His potential by withdrawal of the catheter should be done, if possible, while the atrium is paced at a rate in excess of the spontaneous sinus rate since the H potential may be obscured by the atrial complex if the A-V nodal transmission time is short at the spontaneous rate, as was the case with patient A.

In patient B the not yet described phenomenon of bradycardia-dependent block within the His bundle was observed.

Additional Indexing Words:

Atrioventricular block
Bradycardia-dependent block
Carotid sinus massage
Atrioventricular conduction
His bundle rhythm
Atrial and ventricular stimulation
Atropine
His bundle recordings

CATHETER RECORDINGS of His bundle activity have demonstrated that the His bundle itself may be subject to conduction impairment. A finding of so-called "split" His bundle potentials is diagnostic of such disorders. The recording of this type of His bundle potentials requires careful manipulation of the electrode catheter.

In this paper two patients with conduction disturbances in the His bundle are described, in whom the lesion-electrode relations were such that it was impossible to obtain both components of the split His potential at one time from one pair of electrodes. This condition might lead to an erroneous localization of the block.

Material

Patient A

This 65-year-old man had a history of repeated Adams-Stokes attacks. His ECG showed complete atrioventricular (A-V) block with narrow QRS complexes of normal configuration (QRS width 0.09 sec, mean electrical axis in the frontal plane 20°). The ventricular rate was low, 35 to 40 beats per minute.

A diagnostic electrophysiological study was performed after informed consent had been obtained. During catheterization, using a bipolar electrode catheter with an interelectrode distance of 10 mm, His bundle activations preceding each ventricular complex at 35 msec were recorded. No other His bundle potentials were detected during spontaneous rhythm. From this finding it was concluded that the complete A-V block was located above the His bundle, i.e., in the A-V node.

This conclusion proved to be incorrect, however. During right atrial stimulation at a rate of 120 beats per minute a fortuitous change in the position of the leads in the catheter resulted in the appearance of His bundle potentials following each atrial activation at a constant interval of 125 msec. These His potentials shifted into the atrial complex (recorded in the His lead) on termination of atrial pacing, due to a rate dependent shortening of the A-V nodal transmission time.

The fact that the His potentials became obscured by the atrial complexes at lower atrial rates explained why initially (during sinus rhythm) this type of His potentials was not recognized, although the electrode catheter was manipulated carefully. The catheter position at which (during atrial pacing) His bundle potentials bearing a constant time relation to the atrial activity (H) could be found did not

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demonstrate His potentials related to the ventricular activations (H'). The introduction of a second bipolar catheter (with an interelectrode distance of 10 mm) into the His bundle area made it possible to obtain a simultaneous recording of both types of His potentials H and H' (fig. 1).

A frontal and lateral X-ray was taken of the position of the two His lead catheters. From these photographs the spatial distance between the electrodes of those two pairs which were the closest to each other was calculated, using the measured catheter diameter for the estimation of the magnification factor. This calculated distance was 13 mm.

Validation that the H deflections were indeed His bundle potentials could be made in three ways. First it was shown that the A-H interval could be prolonged by carotid sinus massage (fig. 2). Increasing the atrial driving rate resulted in prolongation of the A-H interval, with occurrence of an A-H Wenckebach phenomenon at a critical driving rate (fig. 3). Finally, atropine shortened the A-H interval for each atrial rate studied and increased the rate at which an A-H Wenckebach phenomenon occurred (fig. 3).

Validation of the H' deflections by stimulation through the electrodes from which these deflections were obtained failed because ventricular muscle was stimulated simultaneously. Although the duration of the H'V interval (35 msec) — in the absence of signs indicating conduction delay in the bundle branches — and the configuration of the H' potential suggests that these H' deflections were derived from the His bundle, the possibility that they may represent proximal right bundle branch activity cannot be completely excluded. However, even if this were the case, the block must be located within the His bundle because of the normal QRS configuration.

Thus, in this patient the correct diagnosis of block in the His bundle was difficult to arrive at for two reasons: the failure to record splitting of the His bundle potential when only a single pair of electrodes was used and the inability to record the proximal His potential during spontaneous rhythm because it was obscured by atrial activity.

![Figure 1](image1)

**Figure 1**

**Patient A showing complete A-V block.** His leads 1 and 2 are obtained from two bipolar catheters advanced into the His bundle area. The catheter giving the His 2 lead had a position more proximal to the A-V ring than the other catheter. During spontaneous rhythm His potentials (H') preceding each ventricular complex are only observed in the distal His lead 1. During atrial pacing at a rate of 120 beats per minute His bundle potentials (H) following each atrial complex become visible in the proximal His lead 2. This establishes the diagnosis complete block within the His bundle. The atrial complexes obscure the H potentials during the slower spontaneous sinus rhythm due to a short A-V conduction time.

![Figure 2](image2)

**Figure 2**

**Patient A.** Carotid sinus massage (CSM) during atrial pacing at a rate of 130 beats per minute. During CSM (lower panel) there is an increase of the A-H interval and some slowing of the pacemaker focus in the distal part of the His bundle. The H'V interval remains constant.

The focus in the His bundle activating the ventricles below the zone of block was to some degree susceptible to parasympathetic influences as the slowing effect of carotid sinus massage (fig. 2) and the accelerating effect of atropine (fig. 3) indicate. The transient change in cycle length during carotid sinus massage was small but reproducible and greater than the short-term spontaneous variations of cycle length (which were 10 msec or less). In the course of the catheterization there was a long-term gradual slowing of the His bundle focus, so that at the time of the administration of atropine the ventricular cycle length was longer (fig. 3, upper panel) than at the start of the study (fig. 2, upper panel). Two minutes after the injection of 1 mg of atropine sulfate, the cycle length shortened by 230 msec, an effect which persisted throughout the remaining part of the procedure. We therefore feel that these changes in rate, although small, are significant.

The somewhat unusual finding that the H potentials were obscured by atrial activity could have been caused by a long duration of the atrial complex in the His lead recording, a

![Figure 3](image3)

**Figure 3**

**Patient A.** (Upper panel) An A-H Wenckebach phenomenon occurs at a driving rate of 145 beats per minute. Lower panel) After administration of atropine the rate at which an A-H Wenckebach periodicity occurs is higher — 176 beats per minute — and the H'H' interval decreases.

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short A-V nodal transmission time, or both. The duration of atrial activity measured from its earliest detectable activity in any of the recorded leads to the end of the atrial complex in the His lead was 125 msec during the spontaneous rhythm and 100 msec during atrial pacing. At the lowest atrial driving rate (100 beats/min) at which H potentials could be discerned in the terminal part of the atrial complex, the A-H (artifact-His) interval was 90 msec. When the driving rate was decreased, the H potential moved into the atrial complex. Thus, the P-H interval during spontaneous rhythm was shorter than 125 msec and the A-H interval during atrial pacing at rates between 70 and 100 beats/min was shorter than 90 msec.

In view of these figures A-V nodal transmission time was presumably short and the possibility of an accessory A-V nodal bypass tract, connecting atrium and proximal part of the His bundle, could be postulated. However, no other direct or indirect evidence corroborating this supposition was found during atrial pacing and the single test stimulus study. Apart from the presumably short A-H interval at low atrial rates, conduction through the A-V node was normal, as shown by the normal value obtained for the rate at which an H-Wenckebach phenomenon occurred and from the normal values for the functional and effective refractory periods of the A-V node (FRPAVN and ERPAVN, see table 1).

Patient B

A 56-year-old man who had experienced three Adams-Stokes attacks prior to admission was studied. Alternating 2:1 A-V block and 1:1 A-V conduction were seen on his ECG, dependent on the heart rate. Although signs suggestive of some conduction delay in the right bundle branch were present, the QRS width was normal (0.10 sec). The mean electrical axis in the frontal plane was 40°. A His bundle recording made during 2:1 A-V conduction initially showed His bundle deflections (H') preceding each ventricular complex at 35 msec (fig. 4, strip 1). This finding suggested that the block was located within the A-V node. However, when the catheter was withdrawn 1½ cm, His bundle deflections, following each atrial activation (H) became visible, while the H' deflections preceding the ventricular complex disappeared (fig. 4, strip 2). Since the spontaneous atrial rate varied considerably, the atrium was paced at a rate comparable to the sinus rate in the top panel.

It was not possible to record both His deflections simultaneously from a single His bundle lead. Therefore a second electrode catheter was introduced and positioned at the site where the H' deflections could be obtained. When the atrial driving rate was progressively increased, a gradual prolongation of the A-H interval was observed (from 95 msec at rate 50 beats/min to 175 msec at rate 120 beats/min), until at a rate of 130 beats/min an H-Wenckebach phenomenon occurred. This demonstrated that the H deflection was indeed a His bundle complex, and that the 2:1 A-V block found earlier was located within the His bundle.

At low driving rates 1:1 conduction through the His bundle occurred, with an H-H' interval of 25 msec. Figure 5 shows that there was a narrow range of heart rates between 1:1 and 2:1 conduction through the His bundle. No block was found at rates between 60 and 65 beats per minute. At a rate of 65 beats per minute a Mobitz II type of block within the His bundle occurred. A small increase in driving rate (to 66 beats/min) resulted in transition into a 2:1 His bundle block.

These results, which were reproducible, are summarized in table 2. With the single test stimulus method applied to the atrium, the refractory periods of the A-V node (FRPAVN and ERPAVN) and the effective refractory period of the His bundle (ERPHis) could be measured (tables 1, 5, and fig. 6). The normal values for the FRPAVN and ERPAVN indicate that conduction through the A-V node was normal, although the atrial driving rate at which an A-H Wenckebach phenomenon occurred was slightly below the normal lower

| Table 1 |

**Characteristics of Antegrade Conduction in Patients A and B**

<table>
<thead>
<tr>
<th>Patient</th>
<th>A-H Wenckebach rate (b/min)</th>
<th>FRPAVN (msec)</th>
<th>ERPAVN (msec)</th>
<th>His bundle</th>
<th>ERPHis (msec)</th>
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<tbody>
<tr>
<td>A</td>
<td>145</td>
<td>465</td>
<td>320</td>
<td>120</td>
<td>2:1 His block (V-V 1640)</td>
</tr>
<tr>
<td>Atropine</td>
<td>195</td>
<td>335</td>
<td>210</td>
<td>120</td>
<td>compl. His block (V-V 1610)</td>
</tr>
<tr>
<td>B</td>
<td>130</td>
<td>440</td>
<td>320</td>
<td>66</td>
<td>935 (90)</td>
</tr>
<tr>
<td></td>
<td>140</td>
<td>440</td>
<td>330</td>
<td>915</td>
<td>80</td>
</tr>
</tbody>
</table>

Abbreviations: A-H Wenckebach rate = heart rate at which Wenckebach pattern occurs; FRP = functional refractory period; ERP = effective refractory period; AVN = atrioventricular node.
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Figure 5

Patient B. Influence of atrial driving rate on A-V conduction. Recordings from two bipolar His leads are obtained from two different catheters in the His bundle area. His lead 2 is from a position more proximal to the A-V ring than His lead 1. At a rate of 63 beats per minute there is 1:1 A-V conduction with an H-H' interval of 25 msec. A Mobitz type II block in the His bundle occurs at rate of 65 beats/min, a 2:1 block at 66 beats/min. BCL = basic cycle length.

Table 2

The Effect of Atrial Driving Rate on A-V Conduction in Case B

<table>
<thead>
<tr>
<th>Rate</th>
<th>A-A</th>
<th>A-H</th>
<th>H-H'</th>
<th>H'-V</th>
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<td>465</td>
<td>A-H Wenckebach</td>
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</table>

All values are in msec.

limit of 140 beats per minute. The ERP\textsubscript{His} was very long at both rates studied (935 msec at rate 60, and 915 at rate 80).

Figure 6 shows that very late atrial test depolarizations were also blocked in the His bundle. At a driving rate of 60 beats/min, the critical H\textsubscript{1}–H\textsubscript{2} interval at which the H\textsubscript{2} activation was blocked was 1425 msec. Presumably this type of block is due to phase 4 depolarization in the compromised part of the His bundle.

This mechanism could explain the peculiar phenomenon of complete A-V dissociation after cessation of atrial pacing at a rate at which 1:1 conduction was previously present (fig. 7). The ventricles were activated by an escape focus apparently located in the distal part of the His bundle. The cycle length of this His bundle rhythm was 1730 to 1740 msec. It seems likely that this complete A-V dissociation in which phase 4 and phase 3 block both played a role is similar to the one proposed by Rosenbaum and coworkers\textsuperscript{7} in their explanation of paroxysmal atrioventricular block located at the level of the bundle branches. Following their argument one might speculate that the escape activation in figure 7 (third ventricular complex) invaded retrogradely and activated part of the area of depressed conduction at a time when antegrade conduction was blocked by phase 4 depolarization. The next atrial (and corresponding proximal His bundle) activation may then be blocked because it arrives at the zone of depressed conduction before the refractoriness caused by the retrograde activation of the escape beat had dissipated (phase 3 block). If an atrial activation arrives at the injured area after the end of the period of phase 3 block and before the start of the phase 4 block induced by the escape beat, that is, in the "normal conduction range," it may be conducted and terminate the "paroxysm" of A-V block. The way in which the reproducible paroxysms of intra-His block ended in this patient strongly suggests the existence of such a mechanism.

The escape beat therefore plays an important role by its resetting the area of depressed conduction, an activation

Table 3

A-V Conduction of Atrial Test Stimuli (A\textsubscript{2}) in case B (basic cycle length 1000 msec)

<table>
<thead>
<tr>
<th>A\textsubscript{2}-A\textsubscript{1}</th>
<th>H\textsubscript{1}-H\textsubscript{2}</th>
<th>H\textsubscript{1}'-H\textsubscript{2}'</th>
<th>V\textsubscript{1}-V\textsubscript{2}</th>
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All values are in msec.

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which creates a time span conducive to successful conduction. If the time relations between escape beat and following atrial activation are favorable, conduction may take place and the period of A-V block is extended. If these time relations are unfavorable (the atrial beat comes too early or too late), antegrade conduction is blocked and the period of A-V block is extended.

Complete block within the His bundle could also be produced by massage of the left carotid sinus during atrial driving with 1:1 A-V response (fig. 8, rate 55 beats/min). In this figure the second atrial activation after carotid sinus massage is begun is blocked in the His bundle. A ventricular activation follows after the next atrial beat with a prolonged H-H' interval (75 msec). The next five atrial beats are blocked until, 3180 msec after the last H' activation, a His bundle escape beat occurs. It is clear that the first cycle after beginning of carotid sinus massage has a longer H-H' interval due to conduction delay within the A-V node. Therefore, the possibility that the block in the His bundle occurring in this first cycle during CSM was caused not by a direct nervous effect on the His bundle but by the slowing of the rate of the proximal part of the His bundle (phase 4 block) had to be considered. However, an H activation elicited without carotid sinus massage by a late atrial test stimulus (fig. 9, panel B arrow), with an H-H' interval identical to the first H-H interval during CSM (fig. 9, panel A), was conducted through the His bundle. At this basic driving cycle length (1100 msec) the H-H interval at which conduction was blocked by phase 4 block was 1430 msec. From figure 9A it is clear that, during CSM, block had occurred at an H-H' interval shorter by 230 msec. This finding indicates that at least in the first cycle CSM influenced His bundle conduction directly. For the block of the next six atrial beats in figure 8 (if one considers the second ventricular activation during CSM as an escape beat from the lower part of the His bundle and not a conducted beat), the mechanism of phase 3 and phase 4 block might have played an important role. The long interval between the second and third ventricular activations during CSM suggests that CSM also influenced impulse formation in the lower part of the His bundle. The effect of CSM on conduction through the His bundle was reproducible. As in case A it was impossible to stimulate the distal part of the His bundle selectively.

Discussion

Splitting of the His potential into two components, one bearing a time relation to the atrial activation, the other to the ventricular activation, has been
Patient B. In order to analyze the mechanism of the His bundle block occurring during carotid sinus massage (CSM, figure 8 and this figure panel A), a test impulse to the atrium was given without CSM (panel B, arrow). This impulse gave rise to a His activation (H) with the same H-H interval of 1200 msec as the first H-H interval after initiation of CSM (panel A). Since this H activation is successfully conducted through the His bundle, the block during CSM must be attributed to a direct nervous influence upon the His bundle and not to the existence of a bradycardia-dependent block.

recognized as a diagnostic sign of conduction impairment within the His bundle. It is thought that the recording of the two components of the His potential is brought about when the catheter is in the critical position in which the bipolar electrodes override the lesion in the His bundle.

In an earlier communication we expressed our view that the actual incidence of conduction disturbances in the His bundle might be higher than indicated by the number of successful recordings of split His potentials, since the recording of the two components of the His bundle activation depends on a delicate interplay of several factors, including location and size of the lesion, interelectrode distance, spatial angle between catheter and His bundle, and distance between the electrodes and the “proximal” and “distal” normally conducting parts of the His bundle, respectively.

It is not improbable that in a substantial number of blocks in the His bundle the diagnosis is missed, due to a less than optimal relationship between electrodes and lesion. Problems of this kind in recording of split His bundle electrogram were encountered frequently by Scherlag and coworkers in their experiments on ischemia-induced His bundle block in the dog heart. Furthermore, in third degree block in the His bundle the diagnosis can be made only if the focus activating the ventricles is located within the distal part of the His bundle. For these reasons the successful recording of splitting of the His potential may consume an appreciable amount of time. Careful positioning of the catheter and sometimes exchange of the catheter for one with a different bend of the tip are required.

In this paper, two patients with a conduction disturbance in the His bundle in whom it was not possible to obtain both components of the His potential at one time from a single bipolar electrode lead with an electrode distance of 10 mm are described. In both cases the finding of His potentials preceding the ventricular complexes led to the initial, wrong impression that the block was located within the A-V node. When the catheter was withdrawn slowly, these potentials (H') disappeared and potentials (H) bearing time relationships to the atrial depolarizations became visible. This finding suggests that in these two patients the electrode-lesion relationships were such that the bipolar electrode could not override the lesion.

In patient A the H potentials were only visible if the atrium was driven at a rate higher than the sinus rate, since the A-H interval during sinus rhythm was so short that the H potential was hidden in the terminal part of the atrial complex. The H and H’ potentials could be recorded simultaneously by the positioning of a second bipolar electrode catheter in the His bundle area. The calculated smallest electrode distance of the two catheters in patient A of 13 mm suggests that the lesion in the His bundle was too long to permit “overriding” by the two electrodes of a single bipolar catheter with an electrode distance of 10 mm. Although it was not possible to stimulate the His bundle by way of the “distal” electrodes, the H’ potentials observed in both patients were acceptable as true His bundle potentials by their configuration and timing.

In view of these findings in these two patients, we advocate that in every patient in whom initial His bundle recordings suggest a conduction disturbance located in the A-V node the catheter should be withdrawn carefully in order to exclude the possibility of a His bundle block. Preferably this should be done while the atrium is being driven at a rate well over that the sinus rate because the A-H interval during sinus rhythm might be short enough to obscure the H potential (patient A). Extrapolating from our findings, we also propose that in every patient in whom the initial His bundle recordings suggest a conduction disturbance at the level of the bundle branches (H-V block), the catheter should be advanced carefully. The finding of H’ potentials would then lead to the correct diagnosis. This procedure may prove to be valuable not only in cases with a narrow QRS complex but also when the QRS complex is broad since the bundle block may coexist with bundle branch block.

Atropine had an accelerating effect upon the focus in the distal part of the His bundle in patient A (fig. 3).
Although the rate increase was small (from 32 to 37 beats per minute) as compared to the changes in rate found in cases with a complete block in the A-V node, this effect of atropine indicates that the distal part of the His bundle is susceptible to parasympathetic influences. Moreover, carotid sinus massage resulted in a discrete slowing of the His bundle focus in patient A and in the initiation of block in the His bundle in patient B.

In patient B block in the His bundle was produced not only by increasing the atrial rate but also by diminishing it (figs. 6 and 7). To our knowledge this is the first documented case of bradycardia-dependent block within the His bundle. Bradycardia-dependent block has been described in the bundle branch system. It is thought that this type of conduction impairment is caused by a reduction of the diastolic membrane action potential and to slow (phase 4) depolarization. Possibly the same mechanism is operative in the His bundle of patient B. It is interesting that as soon as block in the His bundle occurs, an escape focus in the distal part of the His bundle becomes active (fig. 7). It seems attractive to attribute both the block and the escape mechanism to phase 4 depolarization at one level or at two slightly different levels in the His bundle. Presumably also phase 3 block plays a role in maintaining the dissociation between atrial and ventricular activity by inhibiting conduction of atrial activations occurring early after His bundle escape beats.

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