Conduction Disturbances Located within the His Bundle

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SUMMARY
Four patients are described with different degrees of conduction disturbance within the His bundle. In one patient with a Mobitz type II atrioventricular (A-V) block with normal QRS complexes the blocked atrial beats were followed by a His potential. Since the QRS complexes of the conducted beats were completely normal, the site of the block was thought to be in the distal part of the His bundle. In the other three patients with a 2:1 A-V block, a nearly complete A-V block, and a complete A-V block, two distinct His potentials could be discerned, one (H) following each atrial beat, the other (H') preceding each ventricular activation. In the patient with 2:1 A-V block a Wenckebach phenomenon within the His bundle could be produced at certain atrial driving rates. Impulse conduction through the A-V node was normal in all four cases, as could be concluded from the effect of increased atrial driving rate and of accurately timed atrial premature beats. The site of the block could not be predicted from the conventional electrocardiogram.

Additional Indexing Words:
A-V conduction Bundle-branch block A-V block Mobitz type II A-V block A-V Wenckebach phenomenon His bundle recordings Stimulation studies

SEVERAL recent studies1-5 have demonstrated that the recording of His bundle electrograms6 is a useful method for the localization of the conduction defect in patients with atrioventricular (A-V) conduction disturbances. It has been shown that most patients with complete heart block have in fact a bilateral bundle-branch block, a finding which is consonant with the outcome of histopathologic studies.7-10 In patients with a

The refractory periods of the A-V conducting partial (first-degree or second-degree) atrioventricular block the conduction defect may be localized between the atrium and the His potential recording site (that is, within the A-V node) or in the peripheral part of the conduction system, which means most frequently conduction impairment in both bundle branches. A few studies,4,11 however, mention the existence of a conduction disturbance within the His bundle as a third possibility.

This communication describes our findings in four patients with different degrees of atrioventricular block in whom the defect could be attributed to conduction impairment within the His bundle.

Methods
The stimulation procedure used was similar to that described in an earlier paper.9 Two bipolar catheters were introduced into the femoral veins using the Seldinger technique; one was positioned against the atrial septum, and the other in the apex of the right ventricle. These catheters were connected with the stimulator used in our catheterization laboratory for electrophysiologic studies.12

Another pair of electrode catheters were inserted for the recording of intracavitary potentials. From one catheter located high in the right atrium a unipolar atrial lead was obtained; the other (bipolar, with electrode distance 10 mm),

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positioned across the tricuspid valve ostium, was used for the registration of His bundle potentials.

In all cases, also leads I, II, III, V_{1}, and V_{6} were recorded on an eight-channel Elema direct-writing recorder and stored on tape on an Ampex FR-1300 tape recorder. Antegrade and retrograde A-V conduction were studied with stepwise increments of atrial or ventricular driving rate and with the application of atrial or ventricular premature stimuli during regular driving with gradual shortening of the premature beat interval. Time intervals were measured taking the stimulus artifact or the first visible deflection in any of the leads as a reference for ventricular and atrial activations, and the first visible deflection in the His bundle lead for the His bundle activation.

His bundle potentials were identified by their configuration, duration, and timing: by the influence of increasing atrial driving rate and of atrial premature beats on their timing; and by stimulation of the heart through the electrodes used for the derivation of these His potentials. For the registration of His bundle potentials a modified Elema EMT-12 amplifier was used. The response of the amplifier-recording system was 3 dB down at 30 Hz at the lower frequency range and at 725 Hz at the higher range.

Normal values for the A-H interval during sinus rhythm range in our laboratory between 85 and 150 msec; for the H-V interval they range between 35 and 55 msec. During atrial stimulation at rates just above the sinus rate the A-H interval was always shorter than during sinus rhythm, since the stimulation site (the midst of the atrial septum) was located nearer to the A-V junction than to the sinus node.

The refractory periods of the A-V conducting system were determined along the lines of earlier experimental work by the groups of Hoffman^{13} and Moe.^{14} The functional refractory period of the A-V node (FRP_{AV node}) for a specific basic driving rate was defined as the shortest interval between two His bundle activations (H_{1}-H_{2}) that could be produced by an induced atrial premature beat (A_{2}). The effective refractory period of the A-V node (ERP_{AV node}) was defined as the longest possible atrial premature beat interval (A_{1}-A_{2}) not giving rise to a His bundle activation (H_{2}). Both refractory periods are rate-dependent. The FRP_{AV node} tends to fall as rate increases; the ERP_{AV node} tends to rise.

Normal values for the FRP_{AV node} at a driving rate just above the spontaneous sinus rate in our laboratory range between 330 and 500 msec (65% between 330 and 425 msec; 27% between 425 and 475 msec). At a driving rate just above the sinus rate, A-V conduction of atrial premature beats remained present until the refractory period of the atrium was reached in 24% of normal subjects.

The ERP_{AV node} in the remaining 76% ranged between 230 and 390 msec (82% between 230 and 325 msec).

The effective refractory period of the distal part of the His bundle (ERP_{His}) was defined as being equal to the longest possible interval between two His bundle activations (H_{1}-H_{2}) in which the second (H_{2}) is not conducted to the ventricles. Normally the ERP_{His} cannot be measured by atrial premature stimulation since it is shorter than the functional refractory period of the A-V node.

Results

Case 1

This 71-year-old woman had a history of syncopal attacks. The electrocardiogram showed a sinus rhythm with a P-R interval of 0.19 second and QRS complex of normal duration and configuration (fig. 1, left side). At one examination a Mobitz type II A-V block was found without alteration of QRS configuration. Intracavitary electrograms showed that during sinus rhythm the A-H and H-V intervals were normal (resp. 140 and 50 msec, fig. 1).

During atrial stimulation, stepwise increments of the driving rate resulted in prolongation of the A-H interval from 100 msec at rate 110 to 165 at rate 190, the highest rate studied (fig. 1). At rate 120 a Mobitz type II A-V block was seen several times, as is shown in the bottom strip of figure 1. In this strip the fifth His bundle potential is not followed by a ventricular activation. There is no alteration of the preceding A-H and H-V intervals. It is interesting that there was 1:1 H-V conduction at higher rates up to 180 per minute, the H-V interval remaining constant (50 msec). At rate 190, 1:1 A-H conduction was present, but a 2:1 H-V block existed (fig. 1). The refractory period of the conduction tissue below the level of registration of the His potential could be estimated by premature atrial stimulation (A_{2}) after each eighth beat (A_{1}) of a regularly driven atrial rhythm. The A_{1}-A_{2} interval was gradually shortened until the refractory period of the atrium was reached.

The first strip of figure 2 shows that at basic cycle length of 500 msec a premature beat (A_{2}) with an A_{1}-A_{2} interval of 300 msec was conducted through the A-V node with some retardation (H_{1}-H_{2}, 325 msec), but arrived at the ventricle at normal speed (V_{1}-V_{2} is equal to H_{1}-H_{2}). When the A_{1}-A_{2} interval was shortened by 10 msec (290 msec, fig. 2, second strip), the H_{1}-H_{2} interval was also 10 msec shorter (315 msec), but activation of H_{2} was not followed by a ventricular activation.

At A_{1}-A_{2} 270 msec, H_{1}-H_{2} was 310 msec. This was, in fact, the shortest H_{1}-H_{2} interval that

Atrial pacing: On increasing the driving rate a prolongation of the A-H interval (AH) is observed from 100 msec at rate 110 to 135 msec at rate 170, the H-V interval remaining constant: 50 msec. No changes in duration or configuration of the ventricular complex. At rate 190 a 2:1 H-V block occurs. The H-V interval of the propagated impulses is 50 msec. The lowest strip shows a Mobitz type II block at atrial driving rate 120. The fifth atrial impulse is blocked below the level of recording of the His potential (H), without preceding prolongation of the A-H or H-V interval. RA = right atrium; I, II, III, V₁, V₆ = electrocardiogram leads.

Figure 1
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could be produced by atrial premature stimulation at this particular basic rate and therefore, by definition, the functional refractory period of the A-V node. Again the premature impulse was blocked below the level of registration of the His potential. So at this rate the effective refractory period of the conduction tissue below the level of registration of the His potential (ERP_{His}) was estimated to be 315 msec, and the functional refractory period of the A-V node (FRP_{AV node}) was 310 msec.

As can be concluded from table I, both values decreased when the basic driving rate increased, the ERP_{His} more than the FRP_{AV node}. At rate 160, H_2 potentials with the shortest possible H_1-H_2 interval (290 msec) were still conducted to the ventricles; at this rate, therefore, the ERP_{His} was shorter than the FRP_{AV node}.

At all basic rates A-H conduction remained present until the refractory period of the atrium (RP_{Atrium}) was reached. The third, fourth, and fifth strips of figure 2 demonstrate that the H_2 impulse resulting from A_2, with A_1-A_2 270 msec is blocked in the tissue below the level of registration but still penetrates into this part of the conduction pathway, giving rise to the phenomenon of concealed conduction. A second atrial premature beat (A_3), elicited 545 msec after A_1, is blocked below the recording site of the His potentials when H_3 is present (fourth strip), but is conducted to the ventricles when H_2 is absent by the omission of A_2 (fifth strip).

Comment

In this patient a Mobitz type II A-V block and 2:1 A-V block could be produced by increasing the atrial driving rate. The block was located below the level of registration of the His potentials. The QRS complex of the conducted beats, however, was completely normal. It is therefore likely that in this patient the distal part of the His bundle was the site of the A-V conduction disturbance.

It could be established that in this patient rate increase had a greater influence on the duration of the effective refractory period of the His bundle than on that of the functional refractory period of the A-V node. Both values decreased with rising driving rate, the ERP_{His} more than the FRP_{AV node}. These findings are in accord with the results of Moe and co-workers, who showed in the dog heart that the inverse relation between rate and refractory period was more pronounced in the right bundle branch and the His bundle than in the A-V node. Studies by the groups of Moore and Myerburg suggest that a similar mechanism exists at the more distal level of the Purkinje network and the ventricular muscle.

Concealed conduction in the His bundle or in lower parts of the A-V conduction system could be demonstrated in the dog heart by Hoffman. Our patient gives a rare clinical example of this phenomenon.

Case 2

This 27-year-old man had a relaxation of the left side of the diaphragm of unknown origin, and a dextroposition and dextrorotation of the heart. The ECG showed a 2:1 A-V block with prolonged P-R interval of the conducted beats (0.22 second), which showed a complete right bundle-branch-block configuration with QRS duration 130 msec (fig. 3). Hemodynamic studies of the right and left side of the heart revealed no abnormalities aside from the positional anomaly. There were no signs suggestive of a corrected transposition of the great vessels.

Recording of His bundle electrograms during sinus rhythm with rate 75 revealed that every atrial beat was followed at 100 msec by a His potential (H) and that every ventricular beat was preceded at 30 msec by a second, smaller deflection (H') (fig. 3). The H-H' interval was at this rate 90 msec. The H-V interval of the conducted beats was 120 msec.

When the right atrium was driven with rate 100 per minute a 2:1 H-H' block was seen (fig. 4). The H-H' interval of the conducted beats was 135 msec. At rate 110 an interesting phenomenon was observed. In the strip shown (fig. 4) the first, third, and fifth H complexes are followed by an H' deflection and a ventricular activation, however with a gradually increasing H-H' interval (resp. 100, 140, and 170 msec). The seventh H deflection is not followed by a H' potential, but the eighth again shows conduction to the ventricles, and the sequence described repeats itself.

This rare phenomenon can be explained as an H-H' Wenckebach period superimposed upon a 2:1 H-H' block. This explanation is based on the supposition that the zone of tissue with conduction impairment has a very long refractory period, which could be validated by the application of atrial test stimuli, as will be shown below. A 3:1 H-H' block was found at rates 120 to 140 and a 4:1 H-H' block at rate 160. The H'-V interval remained 30 msec at all rates. The A-H interval showed a gradual increase from 90 msec at rate 100 to 200 msec at rate 160. At rate 170 an A-H Wenckebach phenomenon was found (not shown.
Case 1. Atrial premature beats (A₂) induced after each eighth beat (A₁) of a regular driven atrial rhythm with basic cycle length (BCL) 500 msec. An atrial premature beat (A₂) with A₁-A₂ interval 300 msec is conducted with an A-H prolongation of 25 msec (H₁-H₂ 325 msec) through the A-V node. There is no additional delay below the level of recording of the His
Table 1

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<th>Rate</th>
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<th>FRPAV node (msec)</th>
<th>ERPHis (msec)</th>
<th>RPAtrium (msec)</th>
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Abbreviations: FRPAV node = functional refractory period of the A-V node; ERPHis = effective refractory period of conduction tissue below the level of registration of the His potential; RPAtrium = refractory period of the atrium.

Figure 3

Case 2. Spontaneous rhythm. P-P interval 750 to 850 msec; 2:1 A-V block. Each atrial beat is followed after 100 msec by a His potential (H). This potential H is followed in a 1:2 fashion at 90 msec by a second potential (H'), which precedes a ventricular complex with a constant interval of 30 msec. The GRS complex shows a complete right bundle-branch block (width 140 msec) with a mean electrical axis of about -130°. The A-V interval of the propagated beats is 220 msec. RA = right atrium; I, II, III = electrocardiogram leads.

potential. When A2 is induced 10 msec earlier (A1-A2 290 msec), the retardation in the A-V node is again 25 msec (H1-H2 315 msec), but the impulse is blocked below the recording site of the H potential. The same phenomenon is seen at A1-A2 interval 270 (the H1-H2 interval is now 310 msec).

A second atrial test stimulus (A2) induced 545 msec after A1 is also blocked below the His recording electrodes. That this is due to concealed conduction of H1 in the His-Purkinje system becomes clear when A2 is omitted (last strip). A2 is now transmitted to the vehicles. The V1-V4 interval is 10 msec shorter than the A1-A2 interval due to faster impulse propagation through the A-V node, caused by the fact that the A1-A2 interval is longer than the basic cycle length. RA = right atrium; III = lead III of the electrocardiogram.

Influence of Atrial Driving Rate on Refractory Periods of the Atrioventricular Junction (Case 1)
Figure 4

Case 2. Influence of increasing atrial driving rate upon A-V conduction. At rate 100 a 2:1 block in the His bundle is seen (H-H' interval [HH'] of propagated beats 135 msec). At rate 110 an H-H' Wenckebach phenomenon is superimposed upon a 2:1 H-H' block. Note gradual prolongation of H-H' interval until the seventh atrial beat is not propagated to the ventricles. The eighth atrial beat is conducted again and restarts the Wenckebach cycle. At rates 120 and 140 a 3:1 H-H' block is found; at rate 160 a 4:1 H-H' block. There are increases in A-H (AH) and H-H' intervals accompanying the increases in driving rate; the H'-V interval, however, remains constant (30 msec). At rate 180 a 2:1 A-H block and 2:1 H-H' block are observed. RA = right atrium; I = lead I of the electrocardiogram.
ventricular activation ($V_2$); the $H_r-H'_r$ interval, however, was longer: 125 msec. When $A_2$ was advanced only 5 msec ($A_1-A_2$ 900 msec), the impulse was blocked at a site lower than that giving rise to the $H_2$ deflection (fig. 5, second and third strips).

The $H_1-H_2$ interval was 895 msec at $A_1-A_2$ 905 msec, and 890 msec at $A_1-A_2$ 900 msec. The effective refractory period of the tissue below the site of inscription of the H potential ($ERP_H$) therefore was 890 msec at this rate, which is considerably longer than the H-H interval of the basic rhythm (665 msec). This explains the existence of a 2:1 A-V block at this rate and the fact that a Wenckebach phenomenon could be superimposed upon a 2:1 A-V block at rate 110 (fig. 4).

Further shortening of $A_1-A_2$ never resulted in $H_1-H'_1$ conduction. At $A_1-A_2$ 305, the shortest possible $H_1-H_2$ interval was found: 410 msec, the functional refractory period of the A-V node (fig. 5, fourth strip). At $A_1-A_2$ 295, the $A_2$ impulse was blocked within the A-V node (effective refractory period of the A-V node; fig. 5, fifth strip). The refractory period of the atrium was reached at $A_1-A_2$ 250 msec.

Comment
In this patient with a presumably congenital A-V conduction anomaly, the spontaneous rhythm showed a 2:1 A-V block with complete right bundle-branch block. In the His bundle lead two distinct potentials (H and H’) could be discerned, the H deflection following each atrial beat at a normal A-H interval, the H’ deflection preceding each ventricular beat at a rather short interval (30 msec). This registration of two distinct deflections could be explained by assuming that the bipolar electrode had a location overriding a zone of conduction impairment within the His bundle.

The possibility that the H’ deflection was not a potential produced by the distal part of the His bundle, but a right bundle-branch potential, cannot be excluded completely. The H’-V interval of 30 msec was in fact slightly shorter than the lower limit of 35 msec for the H-V conduction time accepted to be normal in our laboratory and by other workers,2,3 but much longer than the right bundle-branch–ventricle (RB-V) interval that would be expected in the presence of a complete right bundle-branch block.4,5 We did not succeed in localizing the site of origin of the H’ deflection by stimulation of the heart by way of the His bundle recording electrodes, because on stimulation the ventricles were always activated immediately. Of course, one has to consider the possibility that in a right bundle-branch conduction disturbance the RB-V interval might be prolonged by coexisting disease of the left bundle branch. In order to explain the long H-RB (H-H’) interval, an additional delay has to be postulated in the proximal part of the right bundle branch (proximal to the alleged site of the recording electrode), in the distal part of the His bundle, or in both.

So if H’ is in fact a right bundle-branch potential, delay of conduction must exist at three levels: the distal part of the His bundle or the proximal part of the right bundle branch; the distal part of the right bundle branch; and the left bundle branch. As is clear from figures 4 and 5, increases in atrial driving rate and atrial premature beats then modify the impulse transmission in only one of these three sites of impaired conduction, the distal part of the His bundle or the proximal part of the right bundle branch, while they have no influence on the distal part of the right bundle branch and the left bundle branch, since the H’-V interval remains constant and the QRS configuration of $V_2$ is unchanged. Although it cannot be denied that such a rather complicated mechanism might be present, the assumption that H’ is indeed a His bundle potential and that there is a zone of impaired conduction within the His bundle coexistent with a complete right bundle-branch block seems to be more attractive by its greater simplicity.

Another possibility to be considered is that the H’ potential originates from delayed activation of a portion of the His bundle, with (due to longitudinal dissociation in this structure) normal conduction through the rest of it and with delay or block of conduction in the bundle branches. With this model, however, it is difficult to explain why the impulse propagation to the compromised part of the His bundle and that within the bundle branches are blocked at exactly the same time when atrial driving rate is increased or when
Figure 5

Case 2. Estimation of refractory periods of A-V node and His bundle. Regular driving right atrium (RA) with basic cycle length (BCL) 665 msec. A test pulse (A₂) is applied to the atrium after each 16th beat (A₁) of the basic rhythm. A₂ is followed by an H, an H', and a V complex at A₁-A₂ intervals greater than 900 msec. At A₁-A₂ 900 msec, A₂ is followed only by an H complex. The H₁-H₂ interval is then 890 msec; this is the effective refractory period of the distal part of the His bundle (ERP₃). At all A₁-A₂ intervals between 900 and 295 msec, A₂ gives rise to an H complex, but never to an H' potential. At A₁-A₂ 295 msec,
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appropriately timed atrial premature beats are elicited, unless one assumes that the block is located at a higher level in the His bundle.

The particularly interesting finding in this case was that a Wenckebach phenomenon could be produced within the His bundle.

Case 3

This was a 78-year-old man with a history of Adams-Stokes attacks. His ECG showed a complete A-V block with QRS complexes with normal configuration and a duration of 0.09 second.

The ventricular rate was about 36 per minute. His bundle recordings revealed that there were two H deflections, one (H) following each atrial activation at 125 msec, the other (H') preceding each ventricular complex at 45 msec. Figure 6 (upper strip) shows this phenomenon at atrial driving with rate 100 per minute. The A-H interval at this atrial rate is 135 msec. The H'-V interval is 45 msec. When the atrial driving rate was increased stepwise from 70 to 170, a gradual prolongation of the A-H interval from 110 msec at rate 70 to 175 msec at rate 130 did occur. An A-H Wenckebach periodicity was observed at rates 140 to 160, and a 2:1 A-H block at rate 170. The findings at rates 100, 120, 150, and 170 are shown in the upper four strips of figure 6. At all rates the H'-V interval was 45 msec. The V-V interval was 1,650 to 1,655 msec at all atrial rates. Sometimes, however, ventricular beats with a shorter interval to the foregoing ventricular activation were seen (second ventricular beat in the second strip of figure 6, and third and fifth beats in the fifth strip of figure 6). These beats, having a QRS configuration identical to that of the foregoing ventricular beats, were always preceded by an H and H' deflection with a constant time relation (H-H' 30 msec).

His bundle pacing resulted in 1:1 conduction to the ventricles at rates up to 100 per minute, with a conduction time of 35 msec (fig. 7). The QRS complexes had the same configuration as those of the basic rhythm. Atrial muscle adjacent to the His bundle electrodes was also stimulated with the stimulus strength needed to activate the distal part of the His bundle (10 ma). The fact that the artifact-V interval was 10 msec shorter than the H'-V interval may indicate that the pacemaker in the distal part of the His bundle was located at a somewhat higher level than the distal electrode of the His bundle catheter.

The lower strip of figure 7 demonstrates that right ventricular pacing had no influence on the A-H conduction time of the independently beating atrium.

Comment

In this patient, showing at first sight a complete A-V block with normal QRS complexes, the conduction defect could be localized within the His bundle. The H'-V interval of 45 msec and the fact that His bundle pacing resulted in 1:1 conduction with unaltered normal QRS configuration prove that the H' deflection originated from the distal part of the His bundle, which area acted as the pacemaker site for the ventricular rhythm. Although the ventricular rhythm was basically regular, QRS complexes were sometimes seen with a shorter preceding V-V interval. Since the QRS configuration of this beat was identical to that of the basic ventricular rhythm, the occurrence of these beats might be explained as sporadic successful conduction through the diseased part of the His bundle, or as premature impulse formation within the distal part of the His bundle. The H' deflections of these beats had the same H'-V interval as the beats of the basic rhythm, but they were always preceded at 30 msec by an H deflection. The first possibility, sporadic conduction, therefore seems to be the more probable.

The fact that the minimal H'-H' interval found in all tracings was 1,575 msec suggests that the refractory period of the compromised part of the His bundle was extremely long. Because of interference of sinus node activity the effective refractory period could not be estimated exactly, as was done in case 2. Since concealed conduction of H activations within the zone of block could therefore not be excluded, the refractory period of this zone might be much shorter than the above-mentioned value of 1,575 msec.

A, is no longer followed by an H complex; this is the effective refractory period of the A-V node (ERP, A node), or of the proximal part of the His bundle. FRP, A node = functional refractory period of the A-N node; RP, atrium = refractory period of the atrium; I = lead I of the electrocardiogram.

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Figure 6

Case 3. The upper strip shows the complete atrioventricular dissociation (also existing in the spontaneous sinus rhythm) at atrial pacing with rate 100 per minute. Note normal configuration and width of QRS complexes. In the His lead every atrial complex is seen to be followed at 135 msec by a His activation (H), and every ventricular complex to be preceded at 45 msec by a His activation (H').

The next four strips show the influence of increasing atrial driving rate. The A-H interval (AH) increases from 135 msec at rate 100 to 175 msec at rate 130. At rate 150 an A-H Wenckebach phenomenon is seen; at rate 170 a 2:1 A-H block. The ventricular activations are always at 45 msec, preceded by an H' deflection. At rate 120 ventricular beats were sometimes seen with a shorter interval to the foregoing ventricular beat than the interval of the basic ventricular rhythm. These beats were always preceded by an H and H' deflection with constant time relation (H-H' 30 msec). This is shown in the strip of rate 120 by the second ventricular beat, which has an interval to the foregoing ventricular beat of 1,575 msec and to the next of 1,655 msec. This phenomenon was more pronounced at rate 130 (lowest strip). At this rate one in every two ventricular beats showed a constant time relation of the H and H' deflections (in strip 130, arrows at the first, third, fifth ventricular beats).
Case 3. His bundle pacing with rate 100 per minute, basic cycle length (BCL) 600 msec. Stimulus artifacts (art.) are at 35 msec, followed by ventricular complexes (V), identical to those of the spontaneous rhythm (fig. 6, note different paper speed). There is a 1:1 ventricular response, in contrast to the high degree of atrioventricular conduction disturbance during atrial pacing with the same rate. The adjacent part of the atrium was also stimulated.

Right ventricular (RV) pacing with the same rate gives no retrograde conduction to the atria. That there is not even concealed retrograde conduction to the upper part of the A-V junction is shown by the fact that the first atrial beat, occurring 55 msec after a ventricular activation, has the same A-H interval (AH) as the other atrial beats. RA = right atrium; I, II, III, V₁, V₆ = electrocardiogram leads.

Case 4
This was an 82-year-old man with a complex A-V block and a history of repeated Adams-Stokes attacks. The ventricular activations, having a rate of 46 per minute, showed a complete right bundle-branch-block configuration with a duration of 0.12 second (fig. 8).

In the His bundle lead, again two types of potentials (H and H') could be discerned. An H deflection followed every atrial beat at 95 msec; an H' potential preceded every ventricular activation at 45 msec. An increase in the transmission time through the A-V node (A-H interval) was found, resulting from fastening the atrial rate. The A-H interval rose from 90 msec at rate 100 to a maximum value of 145 msec at rate 170.

In the strip of rate 170 it can be seen that an H' activation did not inhibit the occurrence of an H activation (second ventricular beat), so there was no retrograde concealed conduction to the proximal part of the His bundle. I, III, V₁, V₆ = electrocardiogram leads.
150. At rates 160 and 170 an A-H Wenkebach phenomenon was seen. The V-V interval remained constant at all atrial driving rates.

The functional refractory period of the junctional tissue between atrium and His bundle recording site (FRP_{AV} node) was estimated at an atrial rate of 100 per minute by the application of atrial test stimuli (A_2) with gradually shortening interval to the foregoing atrial beat (A_1). Shortening of the A_1-A_2 interval resulted in an increase of the A_2-H_2 interval. A minimum value of 385 msec for the H_1-H_2 interval (the FRP_{AV} node) was found. A_2-H_2 conduction remained intact until the refractory period of the atrium was reached at A_1-A_2 200 msec, so the effective refractory period of the A-V node was less than 200 msec.

The fact that an H' potential sometimes preceded an H potential without disturbing the A-H time relation (as shown in the upper right inset in fig. 8) proved that there was no retrograde conduction from the distal part of the His bundle to the proximal part. This can also be concluded from figure 9, showing that the A-H conduction time of an atrial test stimulus (A_1) given during a basic rhythm in which right atrium and ventricle were stimulated synchronously was independent from the presence of an earlier given ventricular test stimulus (V_1). When V_1 was moved forward (gradual shortening of V-V_1), a His deflection H_1' emerged from the ventricular complex (V_1) at V-V_1 intervals shorter than 390 msec, indicating that there was retrograde conduction to the distal part of the His bundle (fig. 9, lower strip).

Comment

In this patient with complete A-V block, His bundle recordings showed that the conduction defect was located within the His bundle. As in case 3, the pacemaker center for the ventricular rhythm presumably was located in the His bundle downstream of the diseased area. The QRS configuration of the resulting ventricular beats indicated an additional impairment of impulse conduction to or in the right bundle branch. The complete independence of H and H' potentials proved that in the retrograde direction there was also a high degree of block within the His bundle. The presence of retrograde conduction from ventricle to distal part of the His bundle could be established.

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Figure 9

Case 4. The two upper strips show that there is no retrograde concealed conduction from the distal to the proximal part of the His bundle. Right atrium (RA) and ventricle (V) are driven synchronously with a cycle length of 600 msec. A test stimulus (V₁) is applied at an interval of 600 msec only to the right ventricle. A second test stimulus (A₁) is then applied 150 msec after V₁ to the right atrium. The A₁-H₁ interval is 90 msec in the presence of V₁ (first strip) and in its absence (second strip), which makes it unlikely that the ventricular impulse V₁ is propagated into the proximal part of the His bundle. That there is, however, retrograde conduction into the distal part of the His bundle is shown in the third strip, where the test stimulus V₁ is moved forward (V-V₁, 360 msec); it is followed by an H′ deflection at 165 msec.
Discussion

Each of the four patients described had a different degree of A-V conduction disturbance. From the conventional ECG it could not be predicted that this conduction defect was localized within the His bundle, as was revealed by the recording of His bundle electrograms. Only in case 1, who had normal QRS complexes in a Mobitz type II A-V block, could localization of the block within the His bundle or its bifurcation be deduced on indirect grounds from the standard ECG since (1) the Mobitz type II block is a conduction disturbance typically occurring below the level of the A-V node;\textsuperscript{5, 11, 20, 21} and (2) the normal QRS configuration of the conducted beats suggests that conduction was impaired above the level of both bundle branches, a sudden simultaneous block in all three fascicles being improbable. His bundle recordings showed indeed that the area of the block had to be sought lower than the His bundle recording site, that is, in the distal part of the His bundle. This case was therefore identical to the cases described by Narula and Samet\textsuperscript{11} and showed again that a Mobitz type II A-V block may occur without signs of bilateral bundle-branch block in the ventricular complex.

In case 2 the 2:1 A-V block of the spontaneous rhythm could be attributed to a very long refractory period of a zone of conduction impairment within the His bundle. At an atrial driving rate of 110 per minute impulse conduction through this zone showed Wenckebach periods superimposed on 2:1 conduction. This case and a case described by Narula and Samet\textsuperscript{11} demonstrate that the Wenckebach phenomenon is not restricted to the A-V node, as was suggested by earlier studies,\textsuperscript{1, 2, 20, 22, 23} but may also occur in the His bundle. Even at a lower level, in the bundle branches, a Wenckebach type of conduction may be found,\textsuperscript{4, 11} which phenomenon was encountered several times in our laboratory. In our experience the Wenckebach phenomenon occurring at a lower level than the A-V node is an expression of a severe functional and presumably also organic disturbance of the A-V junction, contrary to the Wenckebach phenomenon within the A-V node, which can be produced easily in completely normal hearts by increasing the atrial driving rate.

Case 3 is of interest because it shows that a practically complete A-V block with normally configurated QRS complexes may be caused by a conduction disturbance within the His bundle. Localization of the block within the A-V node would have been anticipated had only the standard ECG been available.

In the patient described by Narula and Samet (their case 10)\textsuperscript{11} with intermittent complete A-V block with normal QRS width, H' potentials were visible, preceding the ventricular complexes both during the periods of complete A-V block and during the periods in which a Mobitz type II block existed. H potentials following each atrial beat were seen only during the Mobitz II periods and not when a complete A-V block was present. This was possibly due to catheter movement.

In another study Narula and co-workers\textsuperscript{24} found among a group of 18 patients with complete H-V block only one patient with narrow QRS complexes. Not in this patient, however, nor in a similar patient described by the group of Puech (fig. 9 in their article),\textsuperscript{4} was “splitting” of the His bundle electrogram, as was present in our case 3, observed. In a second patient mentioned by Puech and collaborators (their fig. 6b)\textsuperscript{4} with a complete H-V block, the QRS complexes were broad (0.13 second) or narrow (0.09 second). The latter type of complex was always preceded by a second kind of His deflection, but the first type never. It was thought that the block was located within the His bundle and that the ventricles were activated from a focus in the distal part of the His bundle, resulting in a narrow QRS complex, or from an intraventricular focus, causing a broad complex.

“Splitting” of the His electrogram was also recorded in our case 4. In this patient there was also a complete right bundle-branch block. By looking at the standard ECG one might have anticipated the existence of a complete bilateral bundle-branch block. The
His bundle recording, however, revealed that there was in fact a combination of a complete block within the His bundle and a complete right bundle-branch block.

In two of our four patients with conduction disturbances within the His bundle, the ventricular activation had a complete right bundle-branch-block pattern. One might argue whether there was indeed a coexisting bundle-branch anomaly or that the lesion in the His bundle also comprised fibers already differentiated to feed the right bundle branch, a possibility favored by experimental findings of Sciacca and Sangiorgi.25 In both patients (cases 2 and 4) the possibility that the recorded H' potential was in fact a right bundle-branch potential could not be excluded completely. If this were true, one still has to assume, as outlined in the comment on case 2, that there was conduction impairment at a higher level than the recording site of the H' potential—that is, in the very proximal part of the right bundle branch or in the distal part of the His bundle. In order to explain the right bundle-branch-block configuration and the prolonged RB-V interval, one should then also postulate an additional conduction disturbance in the distal part of the right bundle branch and in the left bundle branch, a construction which is more complicated and therefore less attractive.

It is worthwhile to consider that the recording of two different His bundle potentials in patients with a lesion within the His bundle is in fact the result of a coincidental situation in which such factors as location and extension of the lesion, site of the focus activating the ventricles, electrode distance, and angle between catheter and His bundle play a role. This explains that in some patients with complete H-V block and narrow QRS complexes no H' potentials are recorded. On the other hand, the question may arise whether in some cases with complete H-V block and broad QRS complexes considered to be caused by a complete bilateral bundle-branch block, the lesion is in fact located at a higher level within the His bundle. The focus activating the ventricles might then be situated in the Purkinje system (the second patient of Puech's group mentioned above4 is an example) or in the distal part of the His bundle with a coexisting bundle-branch block. H' deflections might be “missed” in the last possibility due to the extreme delicacy of the recording process.

Probably conduction impairment within the His bundle is not a rare condition. We found four cases among a total of 55 electrophysiologic studies of patients with different grades of A-V conduction disturbance. It is important to recognize that the impulse propagation through the A-V node was normal in all four patients, as was reflected by the normal response of A-H conduction to increases in atrial driving rate and to atrial premature excitation.

Addendum

Since this paper was submitted another patient with complete A-V block, who also showed “splitting” of the His bundle potentials, has been studied. As in the four cases described in this paper, conduction through the A-V node was normal.

References

Conduction Disturbances Located within the His Bundle
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