Retrograde Activation of the His Bundle During Intermittent Paired Ventricular Stimulation in the Human Heart

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SUMMARY
Intermittent paired ventricular pacing was performed in five patients, one of them with Wolff-Parkinson-White (WPW) syndrome. Retrograde activation of the His bundle and of the atria was observed in all patients. Bipolar leads with interelectrode distances of 1 mm seemed to be slightly better than the conventional ones (10 mm apart) for the identification of His (H) deflections buried inside the QRS complexes. Diagnosis of retrograde atrial activation was enhanced by the use of a high bipolar right atrial lead. In cases 1 and 2, V2-A2 and V2-H2 intervals consistently lengthened as the V1-V2 interval was reduced. In two other patients, previously present retrograde P waves disappeared as the V1-V2 interval was reduced. Yet at even shorter intervals, retrograde conduction to the His bundle and to the atria was seen. Ventricular echoes occurred in two cases, and reciprocating tachycardia in one. In these instances the His bundle was engaged in both retrograde and forward conduction. Finally in the patient with WPW syndrome A2 preceded H2 at a given coupling interval, suggesting retrograde atrial pre-excitation through an extra-Hisian pathway.

Additional Indexing Words: Reciprocating tachycardias Ventricular echoes WPW syndrome Retrograde activation of atria

His bundle recordings have enhanced our knowledge of atrioventricular conduction in the human heart.1-5 The method introduced by Scherlag and associates has apparently been less useful in the analysis of ventriculo-atrial propagation. In fact, His bundle deflections buried inside the QRS complexes are difficult to identify with currently available technics. Therefore, the mechanisms of retrograde conduction occurring during spontaneous ventricular extrasystoles or continuous ventricular pacing have not been extensively studied. This is of electrophysiologic importance since in a recent article Mirowski and Tabatznik6 have re-emphasized the old theory which postulates that retrograde conduction of impulses through the A-V junction is usually not the cause of the inverted P waves following A-V junctional or ventricular ectopic beats.

On the other hand the work by Schuilenburg and Durrer7 on the human heart and Damato and associates on the dog's heart8 suggests that retrograde conduction through the A-V junction and His bundle is frequent during ventricular pacing. In addition, Schuilenburg and Durrer postulated that considerable infra-Hisian delay was present in some instances. If these assumptions are correct, it might be possible for a retrograde His bundle deflection to appear after the end of the ventricular complex, as shown by Moore9 in the rabbit's heart. Therefore, it seemed that a combination of the technic used by Schuilenburg and Durrer7 with an extension of the catheter technic of His bundle

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This work was supported in part by a grant from the Astra Pharmaceutical Company, Worcester, Massachusetts.
Received June 8, 1970; revision accepted for publication August 21, 1970.
recordings1-5 could give additional information regarding the mechanisms of retrograde conduction, ventricular echoes, and reciprocating tachycardias in man.

Methods

The technic of His bundle recordings used in our department is an extension of the one introduced by Scherlag and associates. It has been reported in previous communications.10, 11 After explaining the procedure and obtaining consent from the patients, a tripolar* or a bipolar catheter electrode (interelectrode distance: 5 or 10 mm) was positioned across the septal leaflet of the tricuspid valve. The tripolar catheter used in one patient (case 1) had the distal electrodes separated by a distance of 1 mm. The proximal electrode was 11 mm from the tip. Two additional bipolar catheter electrodes (interelectrode distance: 1 or 10 mm) were introduced percutaneously through an antecubital vein. One was placed high in the right atrium (BAE). The other was used for endocardial pacing from the right ventricular apex. Three filtered (40 to 500 Hz) His bundle electrograms, one BAE, and one unipolar ventricular electrogram (UVE) were recorded by attaching the catheter leads to an electrode distribution switch box which was then connected to a recorder.† Conventional leads I, II, III, and V1 were also recorded simultaneously with the former. To reduce the size of the figures and make the interpretation clearer, however, only the most pertinent leads will be presented in each case.

The stimulator used12 delivered slightly underdampered pulses, 2.5 msec in duration, and twice diastolic threshold values. Intermittent paired pacing was performed by means of a variable refractory delay which determined the intervals between test stimuli. The ventricles were driven at a rate high enough to permit 1:1 V-A conduction. After each eighth driving stimulus a test pulse with similar characteristics was delivered (in case 2 the intensity was five times threshold). The interval between the test stimulus (V2) and the previous driving stimulus (V1) was shortened by 5 to 20 msec, the amount depending on the findings. The post-extrasystolic stimulus was generally, but not invariably, delayed or postponed to facilitate the occurrence of echoes.

Criteria for Diagnosis of Retrograde Atrial and His Bundle Activation

With the technic used in our department, the retrograde His bundle and the retrograde atrial deflections do not appear as true mirror images of the ones recorded during sinus rhythm. Hence, specific criteria were developed for this purpose. During sinus rhythm the atrial electrogram was inscribed in the high right atrial lead (BAE) earlier than in the lead recording the activation of the low right atrium (His bundle electrogram [HBE]). This is shown in figures 1, 5, 10, and 12 to 15. During V-A conduction, on the contrary, the onset of the atrial electrogram in the HBE preceded that of the BAE. Retrograde activation of the His bundle was considered to be present when an H deflection appeared within, or after, a pacer-induced QRS complex provided that the

*Elecath Corporation. Rahway, New Jersey.

Figure 1

Case 1: Criteria for the diagnosis of retrograde activation of the His bundle and of the atria. St = stimulus artifact. BAE = bipolar atrial lead recording from high in the right atrium. H = His bundle deflection. HBE = His bundle electrogram obtained with a bipolar lead having an interelectrode distance of 1 mm. HBE11 = His bundle electrogram obtained with a bipolar lead having an interelectrode distance of 11 mm. During sinus rhythm the atrial activity started in the BAE earlier than in HBE11 (indicated by the oblique arrow pointing down). The P wave was hardly visible in HBE. The P-R, P-H, and H-R intervals measured 160, 120, and 40 msec, respectively. The first stimulus artifact (St) was ineffective, but the second one produced an ectopic ventricular beat with abnormal left axis deviation. An H deflection was clearly seen in the HBE. It was sharper than in HBE11. This H deflection must have been retrograde since the preceding (sinus) wave was too far ahead to suggest atrio-His propagation. In addition, it was followed by a retrograde P wave which was inscribed in the BAE later than in the HBE, as indicated by the oblique arrow pointing upward. Paper speed in this and all other figures was 100 mm/sec.
previous P wave was too far ahead to suggest atrio-His conduction (fig. 1). The existence of a retrograde P wave (see preceding paragraph) after the H deflection under consideration reinforced the diagnosis of retrograde His bundle excitation.

The following measurements were made (in msec) during ventricular pacing:

\[ V_1-V_1 = \text{interspike interval between two consecutive driving beats.} \]
\[ V_1-V_2 = \text{interspike interval between driving and testing beats.} \]
\[ V_1-A_1 = \text{R-P interval of driving beats, measured from the driving spike to the onset of atrial activity, in whichever lead it occurred first.} \]
\[ V_1-H_1 = \text{R-H interval of driving beats, measured from the driving spike to the onset of the retrograde His bundle deflection.} \]
\[ V_2-A_2 = \text{R-P interval of testing beats, measured from the driving spike to the onset of atrial activity.} \]
\[ V_2-H_2 = \text{R-H interval of testing beats, measured from the testing spike to the onset of the retrograde His bundle deflection.} \]
\[ A_2-R = \text{P-R interval of an echo beat.} \]

The stimulus-to-local ventricular electrogram was measured in the HBE lead with an interelectrode distance of 1 mm from the emission of the spike to the onset of the rapid ventricular deflection. Conventional measurements were performed when natural beats were present.\(^1\)\(^-\)\(^6\) Five patients were studied, one of them with WPW syndrome.

**Report of Cases**

**Case 1**

This 53-year-old male had angina pectoris. The 12-lead electrocardiogram showed nonspecific ST-T changes and a questionable old inferior wall myocardial infarction. Coronary angiography revealed a 50% occlusion of the right coronary artery. During sinus rhythm, A-V conduction was within normal limits (fig. 1). The ventricles were driven at a rate of 100/min (cycle length of 600 msec). The corresponding \( V_1-A_1 \) and \( V_1-H_1 \) intervals measured 130 and 50 msec, respectively (figs. 2 and 3, table 1). These intervals did not change while the duration of \( V_1-V_2 \) was 400 msec or more (fig. 2, top panel). The stimulus-to-retrograde H interval was shorter than the stimulus-to-local ventricular electrogram interval (onset of ventricular complex in the 1-mm HBE lead). This indicated that the lead in the inflow tract of the right ventricle recorded the electrical activity of the His bundle before that of the ventricular muscle underneath the electrode. In other words, propagation from the stimulated site in the right ventricular apex to the inflow tract of the right ventricle was faster through the conducting system than through the ordinary ventricular muscle. When the \( V_1-V_2 \) interval was shortened to 390 msec (fig. 2, lower panel) \( V_2-A_2 \) increased to 180 msec. However, \( H_2 \) was not seen apparently because it was buried inside the ventricular electrogram.

The findings in the top panel of figure 3 tend to corroborate the latter assumption. The \( V_1-V_2 \) interval was 385 msec, that is, only 5 msec shorter than in figure 2 (lower panel). There was a lengthening of \( V_2-A_2 \) interval to 190 msec, but the H deflection now followed the local ventricular electrogram in the HBE lead. At this moment,
Figure 3

Case 1: Effects of intermittent ventricular pacing at $V_1-V_2$ intervals of 385 and 270 msec. In the top panel $H_2$ was seen in the HBE, but not in HBE11.

Conduction was slower through the Purkinje-bundle branch system than through the ordinary ventricular muscle. Finally, in the bottom panel of figure 3, the $V_1-V_2$ interval was shortened to 270 msec. There was a further increase in the $V_2-A_2$ and $V_2-H_2$ intervals to 270 and 200 msec, respectively. Conduction through the ordinary muscle was now more delayed than when the $V_1-V_2$ intervals were longer. This was manifested by a prolongation of the stimulus-to-local ventricular electrogram in the 1-mm HBE lead. The testing impulses failed to produce a propagated response at a $V_1-V_2$ interval shorter than 270 msec since they fell in the refractory period of the ventricles.

Table 1

<table>
<thead>
<tr>
<th>Intervals (msec)</th>
<th>$V_1-V_2$</th>
<th>$V_2-A_2$</th>
<th>$V_2-H_2$</th>
<th>Type of beat</th>
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<tr>
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<td>130</td>
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<td></td>
<td>Driving</td>
</tr>
<tr>
<td>400</td>
<td>130</td>
<td>50</td>
<td></td>
<td>Testing</td>
</tr>
<tr>
<td>360</td>
<td>130</td>
<td>50</td>
<td></td>
<td>Testing</td>
</tr>
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<td>Testing</td>
</tr>
<tr>
<td>270</td>
<td>270</td>
<td>200</td>
<td></td>
<td>Testing</td>
</tr>
<tr>
<td>Refractory period</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Case 2 (Figs. 4 to 6)

This 53-year-old patient had experienced several syncopal attacks in the month preceding his admission to the Miami Veterans Administration Hospital. Electrocardiograms during sinus rhythm showed complete right bundle-branch block, block in the anterosuperior division of the left branch and first degree A-V block. His bundle recordings were made when the patient was brought to the Cardiopulmonary Laboratory for the prophylactic insertion of a transvenous demand (QRS-inhibited) pacemaker. During sinus rhythm the P-R interval of 230 msec was associated with a prolonged H-R interval of 90 msec (fig. 5, last beat). (In our laboratory the upper limits of this interval is 55 msec.)

Ventricular driving was performed at a rate of 133/min (cycle length, 450 msec). The $V_1-H_1$ and $V_1-A_1$ intervals measured 130 and 330 msec.
Case 2: Ventricular echo, appearing at a $V_1-V_2$ interval of 250 msec, in a patient with right bundle-branch block. Pacing was stopped after the $V_2$ impulse to facilitate the occurrence of echoes. $\alpha$ and $\beta$ represent the corresponding intranodal pathways. Both sinus and echo beats had a right bundle-branch block pattern. In the diagram, A, H, and V indicate the onset of atrial, His bundle, and ventricular activation, respectively. A-H and H-V represent the atrio-His and His-ventricular regions. Oblique lines at the latter levels represent transmission through the respective regions. The deflection site of the retrograde impulse produced by $V_2$ could have occurred either in the atria or high in the A-H region.

Case 2: Appearance of a reciprocating tachycardia after cessation of ventricular pacing. In this diagram the final common pathway (FCP) was represented as being located at the lower levels of the A-H region, that is, between the $\alpha$ and $\beta$ pathways on one side and the His bundle, on the other. The deflection site of retrograde impulses could have occurred in the atria or high in the A-H region.
respectively (table 2). During retrograde conduction, atrial activity was first detected in the lower portions of the right atrium (HBE lead) indicating inferosuperior propagation throughout the atria. The V₂-A₂ intervals of testing stimuli were the same as those of driving stimuli as long as the V₁-V₂ interval ranged from 450 to 380 msec. Thereafter, the V₂-A₂ and V₂-H₂ intervals lengthened progressively as the coupling shortened (table 2, figs. 4 to 6). An interesting phenomenon occurred when the interval between driving and testing stimuli was reduced to 250 msec (fig. 5). At this moment, pacing was stopped after the testing beat to facilitate the appearance of ventricular echoes. It should be noted that the V₂-A₂ and V₂-H₂ intervals attained their maximal value: 470 and 210 msec, respectively (table 2). The retrograde impulse from V₂ activated the His bundle and the atria progressively. It also reversed its direction (either in the A-H region or in the atria) to reactivate the His bundle orthogradely, finally reaching the ventricles. The corresponding H-R interval and QRS morphology were similar to that of sinus beats. In the latter, the atrial electrogram was inscribed in the HBE lead earlier than in the high right atrium.

A reciprocating tachycardia had also been observed when the V₁-V₂ interval was in the range of 370 msec (fig. 6). Due to technical reasons pacing was stopped after the V₁ beat that followed the V₂ beat, instead of after the V₂ beat. The diagram shows that this driving beat (fourth QRS complex from the beginning of the strip) occurred later in the diastole than any of the preceding ventricular complexes. Its timing caused a recycling (shortening) of the retrograde intervals. In consequence, its R-P interval was considerably shorter than normal (300 msec vs. 330 msec). Since retrograde activation of the His bundle also occurred more prematurely, the H deflection must have fallen within the correspond-

Table 2

<table>
<thead>
<tr>
<th>Intervals (msec)</th>
<th>V₁-V₂</th>
<th>V₁-A₁</th>
<th>V₁-H₁</th>
<th>Type of beat</th>
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<td>450</td>
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<td>V₂-A₂</td>
<td>V₂-H₂</td>
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<td>Refractory period</td>
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Table 3

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<th>Intervals (msec)</th>
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<th>V₁-A₁</th>
<th>V₁-H₁</th>
<th>Type of beat</th>
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<td>600</td>
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<td>Driving</td>
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<tr>
<td>V₁-V₂</td>
<td>V₁-A₁</td>
<td>V₁-H₁</td>
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<td>410</td>
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<td></td>
<td>Testing</td>
</tr>
<tr>
<td>320</td>
<td>260</td>
<td>180</td>
<td></td>
<td>Testing</td>
</tr>
<tr>
<td>280*</td>
<td>300</td>
<td>230</td>
<td></td>
<td>Testing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Natural beat</td>
</tr>
</tbody>
</table>

*Repetitive firing occurred at this interval.
RETROGRADE ACTIVATION OF HIS BUNDLE 1085

Figure 7
Case 3: Intermittent ventricular pacing at V1-V2 intervals of 410, 370, and 360 msec. Testing stimuli (V2) did not show retrograde conduction when V1-V2 ranged between 370 and 360 msec.

msec V2-A2 increased to 170 msec (fig. 7, top panel). Retrograde activation of the His bundle and atria was not observed when V1-V2 was shortened to 370 and 360 msec (fig. 7, middle and lower panels).

Retrograde P waves reappeared at intervals between 350 and 320 msec (fig. 8, two top panels). In addition, they were now preceded by H deflections—the corresponding V2-H2 intervals measuring 140 and 180 msec. When the testing interval was shortened to 280 msec (fig. 8, lower panel and table 3), repetitive firing occurred (V2 was followed by spontaneous V3), and the study was discontinued. The corresponding V2-A2 and V2-H2 intervals had lengthened to 300 and 230 msec, respectively. Retrograde conduction of V2 failed below the His bundle.

Case 4 (Figs. 9 and 10)
This patient was a 45-year-old male with a history of chronic alcoholism who was admitted because of heart failure. Tendency to low voltage, nonspecific ST-T wave changes, and a P-R interval of 200 msec were the most conspicuous electrocardiographic findings. Hemodynamic studies performed after apparent clinical compensation showed a low cardiac index, an elevated left ventricular end-diastolic pressure, and moderate pulmonary hypertension. Coronary angiograms were normal. The diagnosis of primary (alcoholic?) cardiomyopathy was made.

Figure 8
Case 3: Intermittent ventricular pacing at V1-V2 intervals of 350, 320, and 280 msec, respectively. A retrograde His bundle deflection (H2) appeared "sandwiched" between the V2 and A2 impulses.

During ventricular pacing at a rate of 100/min (cycle length of 600 msec) the V1-A1 intervals measured 250 msec (table 4). Retrograde His bundle deflections were not observed within these beats. R-P intervals of testing and driving beats were similar until the V1-V2 interval was shortened to less than 480 msec. However, when it was reduced to 470 msec, the V2-A2 interval lengthened to 300 msec (fig. 9, top panel). At ranges between 400 and 280 msec retrograde conduction to the atria did not occur (fig. 9, middle panel). However, His bundle deflections (H2) appeared progressively after the end of the QRS as the V1-V2 interval shortened from 350 to 280 msec (fig. 9, bottom panel).

Further reduction of the V1-V2 interval to 280 msec (fig. 10) resulted in repetitive firing since V2 was followed by two extra beats (V3 and V4). The impulse from V2 was conducted retrogradely to the His bundle (V2-H2 interval of 170 msec) but was blocked in the A-V node. Retrograde conduction time from V3 to the His bundle was even longer (270 msec). Hence, in these two beats there was a retrograde infra-Hisian Wenckebach phenomenon. V4 was blocked below the His bundle. However, the retrograde stimulus produced by V3 propagated through the A-H
region very slowly (presumably through the α pathway) finally reaching the atria with an R-P interval of 520 msec. At some point in the A-H region it was also able to change its direction, reactivate the His bundle orthogrady, finally reaching the ventricles producing a QRS complex of normal contour. The latter was an echo beat.

No further studies were performed on this patient after the appearance of repetitive firing. The P wave preceding the echo beat was inscribed in the HBE earlier than in the BAE indicating retrograde atrial activation. The reverse occurred in the last (sinus) beat. The corresponding QRS complex was similar to that of the echo.

Case 5

A 25-year-old medical student had chest pain on exertion and a 2-year history of palpitations. Electrocardiograms showed WPW type A syndrome (fig. 11). None of the tracings, including continuous monitoring during ambulation, however, had revealed evidence of ectopic tachycardias. Right and left heart catheterization and coronary angiograms were within normal limits.

This subject showed the usual response to atrial stimulation observed in our department in patients with WPW syndrome: The P-H interval and the degree of pre-excitation increased proportionally to the atrial rate and the prematurity of the stimuli.10, 11 The forward H deflection followed the onset of the delta wave in lead II (fig. 12) indicating ventricular pre-excitation through a bypass of the main His bundle.

Induced premature beats (V3) at a driving rate of 100/min produced interesting effects. Driving QRS complexes consistently showed retrograde conduction with a V1-A1 interval of 100 msec (fig. 13). Therefore, onset of atrial activity in the low right atrium preceded that of the BAE. Retrograde activation of the His bundle could not be detected in these beats. At a V1-V2 interval of 350 msec (fig. 13, bottom panel), V-A conduction time measured 190 msec, and the QRS complexes became wider and more distorted. This additional retrograde delay of 90 msec does not exclude conduction through the Kent bundle since it could have been due to slow conduction through the nonspecific ventricular myocardium (intraventricular retardation below the Kent bundle itself), a consequence of propagation during the relative refractory period.18, 19 This type of intramyocardial delay was shown in figure 3 (lower panel). An H deflection was not seen at this interval.

Table 4

<table>
<thead>
<tr>
<th>Intervals (msec)</th>
<th>Type of beat</th>
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<td>V3-V2</td>
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<td>600</td>
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<td>250</td>
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<td>330</td>
<td>—</td>
</tr>
<tr>
<td>300</td>
<td>—</td>
</tr>
<tr>
<td>280 (V2)*</td>
<td>—</td>
</tr>
<tr>
<td>— (V2)</td>
<td>520</td>
</tr>
<tr>
<td>— (V4)</td>
<td>—</td>
</tr>
</tbody>
</table>

*V2 produced two extra beats, V3 and V4 (repetitive firing).
Case 4: Intermittent paired ventricular pacing at a V_1-V_2 interval of 280 msec. The V_2 impulse fell in the vulnerable period of the previous beat and produced repetitive firing (natural beats V_3 and V_4). A ventricular echo was induced by V_2. Note that both sinus and echo beats showed a similar (normal) QRS complex. Arrows between HBE and BAE leads indicate the onset of atrial activity.

The V_2-A_2 was still the same when V_1-V_2 was shortened to 340 msec (fig. 14, top panel). Yet, a retrograde (H_2) deflection now appeared “sandwiched” between the V_2 and A_2. The V_2-H_2 interval measured 160 msec. The long interval (530 msec) between H_2 and the previous retrograde P wave (A_1) suggested that they were unrelated.

Unexpected events occurred when carotid sinus pressure was exerted at the same V_1-V_2 interval (fig. 14, lower panel). First, the stimulus artifact produced repetitive firing. Second, the V_2-H_2 interval lengthened to 230 msec. Third, the H_2 deflection appeared later than A_2. These findings suggested that further intraventricular retardation was responsible for both repetitive firing and for the infra-Hisian delay, as in figure 10. Moreover, the atria were activated before the His bundle (retrograde atrial pre-excitation?), since A_2 appeared before H_2. The short H_2-A_2 interval (30 msec) in the top panel probably indicated that these deflections were not related, since retrograde nodal conduction should last more than 30 msec. Similarly, the short P-H interval (35 msec) in the lower panel tends to exclude forward nodal conduction.

In figure 15 (top panel) the V_1-V_2 interval was shortened to 330 msec. The V_2-H_2 interval measured 160 msec. H_2 was followed, after a delay of 350 msec, by a negative P wave (A_2). The long V_2-A_2 interval might have been a result of delayed retrograde nodal conduction in the presence of complete retrograde block in the Kent bundle. Therefore, the atria were activated after the His bundle. Finally, at a V_1-V_2 interval of 320 msec retrograde conduction must have failed through both pathways since V_2 was not followed by a negative P wave.

Discussion

His bundle recordings have provided important information regarding the mechanisms of atrioventricular propagation in man. However, this technic has apparently been less useful in the study of ventriculo-atrial conduction. This limitation is technical, since
an H deflection buried inside a QRS complex is not always identified when a bipolar lead with an interelectrode distance of 10 mm is used. Figures 1 to 3 (case 1) show that the close bipolar (1 mm) leads are superior in this respect. This finding should be stressed because it validates the assumptions made in cases 2 to 5 regarding the His deflections buried inside the ventricular electrograms. In other words, case 1 offers enough information to suggest that, as the V1-V2 interval shortened, the V2-H2 lengthened. In consequence, H2 which was initially inscribed within ventricular deflections, progressively emerged after the QRS complexes. Such an assumption can be proved by comparing table 1 with tables 2, 3, and 4. Applying the criteria established in the proper section, retrograde activation of the His bundle was proved in one moment or another in all cases.

The possibility that the deflection attributed to reversed activation of the His bundle may represent a retrograde right bundle potential cannot be completely excluded, even though this is improbable because of similarity to the forward H deflection. This possibility, however, does not invalidate the assumptions made in the present communication.

With the technic employed in our department, \(^{10, 11}\) retrograde activation of the atria did not appear as the mirror image of the patterns observed during normal sinoventricular conduction. (figs. 1, 5, 10, 12 to 15) For this reason, high BAE and low bipolar atrial (HBE) leads were recorded to differentiate between superoinferior and inferosuperior propagation throughout the atria.\(^{10, 11}\) The morphology of the P wave in the standard leads was not helpful when the latter fell before the end of the T wave.

In the first two patients there was progressive prolongation of the V2-A2 and V2-H2 interval.

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**Figure 11**

*Case 5: Sinus rhythm and WPW type A.*
intervals as the coupling interval between driving and testing beats shortened (tables 1 and 2). Conduction was delayed both below, and above, the His bundle. Infra-Hisian delay can occur in the myocardium itself as well as in the Purkinje-bundle branch (specialized intraventricular conduction) system. An impulse originating during the relative refractory period\textsuperscript{18,19} could (because of true latency or inhomogeneous intraventricular conduction) reach the Purkinje system with more delay than a beat originating later in diastole (fig. 3, bottom panel). Moreover, Moore observed that the specialized intraventricular conduction system was an important area for retrograde conduction delays and concealed conduction\textsuperscript{9} in the rabbit's heart.

The second patient had significantly longer \(V_1-H_1\) and \(V_1-A_1\) intervals than case 1. This difference might be related to the fact that patient 2 had extensive conducting system disease (right bundle-branch block, block in the anterosuperior division of the left branch, and prolonged P-R and H-R intervals).

The pattern of retrograde conduction in cases 1 and 2 resembled that in case B reported by Schuilenburg and Durrer.\textsuperscript{7} These authors suggested that progressively longer \(V_2-A_2\) intervals indicated that retrograde (supra-Hisian) conduction took place through what Moe and associates\textsuperscript{14-17} have called the \(\alpha\) intranodal pathway, the faster \(\beta\) pathway being unable to respond at these moments. Thereafter, the corresponding impulses could engage the \(\beta\) pathway in a forward direction. An echo beat appeared (as in fig. 5) when this impulse reached the final common intranodal pathway after the end of its refractory period, provided, of course, that another ventricular (driving) beat had not been delivered. The intracavitary recordings presented in this communication did not permit an exact determination of the site (above the His bundle) where the retrograde impulse changed its course to proceed, again, in a forward direction toward the ventricles. According to Moe and associates\textsuperscript{14-17} the atria are essential for the production of echoes. But Mignone and Wallace\textsuperscript{20} and Coumel and associates\textsuperscript{21} considered that the atria are not a

\[\text{Figure 12}\]

\begin{center}
\includegraphics[width=\textwidth]{image12}
\end{center}

\textit{Case 5: His bundle electrograms during sinus rhythm (left) and premature atrial stimulation (right). The forward H deflection coincided with the onset of the delta wave in lead II (first beat) but followed the latter in the second beat.}

\[\text{Figure 13}\]

\begin{center}
\includegraphics[width=\textwidth]{image13}
\end{center}

\textit{Case 5: Intermittent paired ventricular pacing at varying (500 to 350 msec) \(V_1-V_2\) intervals. Both driving \(V_1\) and testing \(V_2\) beats showed retrograde conduction to the atria. Ventricular echoes did not occur.}

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necessary link for the production of ventricular echoes.

In case 2 (fig. 6) a reciprocating tachycardia was initiated when the returning impulse traversing the \( \beta \) pathway reached the deflection point and found that (1) the final common (intranodal) pathway (FCP) was nonrefractory, (2) the \( \alpha \) pathway was responsive toward the atria (fig. 6), and (3) an effective artificial ventricular stimulus had not been delivered.

Cases 3 and 4 had some of the features observed in case C reported by Shuilenburg and Durrer.\(^7\) There was a progressive lengthening of the \( V_2-A_2 \) intervals at the longer \( V_1-V_2 \) ranges. At shorter intervals (370 and 360 msec) retrograde conduction to the atria first failed completely (figs. 7 and 9, middle and lower panels), but later reappeared at even shorter intervals (fig. 8). In addition, there was a gradual increase in the \( V_2-H_2 \) intervals.

It is possible that at the longer coupling intervals retrograde conduction was occurring through only one of the intranodal pathways (possibly the faster \( \beta \) pathway). At a shorter interval, both routes failed completely. When the coupling was further reduced, a significant delay in activation of the His bundle determined that the oncoming ventricular impulse could reach the FCP after the end of its refractory period hence propagated retrogradely to the atria through the \( \alpha \) or \( \beta \) routes. This phenomenon in a general sense represents "supernormal" retrograde conduction to the atria since an impulse originating earlier in diastole (where conduction is supposed to be more impaired) could be propagated successfully, whereas, other beats produced later in diastole failed to do so.

This possibility was also mentioned by Moore\(^8\) who emphasized that if conduction velocity was diminished enough the impulse could reach the His bundle after the latter had recovered its excitability.

*Figure 15*

Case 5: In the upper strip complete retrograde block occurred in the accessory communication. Delayed V-A propagation occurred through the normal A-V pathway. The lower strip shows complete retrograde block in both normal and accessory communications.
In case 4, V₂ induced repetitive firing (V₃ and V₄) when the V₁-V₂ interval was reduced to 280 msec (fig. 10). This created a model similar to Moore's, who systematically used an artificial V₃ impulse.⁹ The spontaneous V₃ produced an abrupt shortening of the R-R cycle. This shortening in turn led to an impairment of conduction between ventricles and His bundle (within the myocardium itself, in the Purkinje bundle-branch system, or in both), therefore permitting a longer than usual V₃-H₃ interval. Hence, the impulse from H₃ reached the FCP after the end of its refractory period, thereafter propagating to the atria and also reactivating the His bundle to produce an echo beat.

Patient 5 had WPW syndrome. During atrial pacing (forward conduction) the ventricles were pre-excited ahead of the His bundle, suggesting an extra-Hisian communication.ⁱ⁰,¹¹ (fig. 12). The pattern of retrograde His bundle activation occurring in this patient followed that of cases 1 and 4 in that H₂ appeared after V₂ only when the V₁-V₂ interval had been sufficiently shortened (fig. 14, top panel). Yet, unexpectedly, at a given moment (fig. 14, lower panel) A₂ preceded H₂; that is, the atria were depolarized (retrogradely) earlier than the His bundle. This phenomenon suggests retrograde atrial pre-excitation through an extra-Hisian communication.

Although the electrical activity of the accessory pathway was not recorded directly, deductive reasoning supports retrograde Kent bundle conduction, as had been postulated from the analysis of clinical electrocardiograms for many years.

Our findings are in disagreement with those of Mirowski and Tabatznik.⁶ These authors recently revived older theories which postulated that “retrograde” P waves were due to stimuli originating in various (usually left atrial) centers, induced in some way by the preceding ventricular contractions. They stated that retrograde conduction through the normal A-V junction was the exception rather than the rule. Our figures 1 to 15, on the contrary, convincingly show that retrograde transmission through the A-V conducting tissues was the rule instead of the exception, even in the presence of trifascicular block. However, more work is still necessary to ascertain whether this mechanism also applies to V-A conduction of spontaneous ventricular extrasystoles or idioventricular complexes in established complete A-V block and to locate the site of deflection of the retrograde impulse.

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Circulation, Volume XLII, December 1970
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Circulation. 1970;42:1079-1092
doi: 10.1161/01.CIR.42.6.1079

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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/42/6/1079

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