Retrograde Conduction in the His-Purkinje System

Analysis of the Routes of Impulse Propagation Using His and Right Bundle Branch Recordings

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SUMMARY We analyzed routes of retrograde impulse propagation in the His-Purkinje system (HPS) in 24 patients with normal intraventricular conduction. Using intracardiac recordings from the His bundle and right bundle branch (RBB), predetermined paced ventricular cycle lengths were scanned with right ventricular extrastimuli ($S_2$). Electrophysiologic findings suggested that retrograde activation of the His bundle after premature stimuli ($H_1$) occurred via the left bundle branch (LBB) in 16 of 24 cases and via the RBB in three of 24 patients. The remaining patients demonstrated retrograde $H_2$ activation via both RBB and LBB at different times during the scanning. In general, a linear relationship existed between retrograde conduction delays in the HPS (i.e., $S_2H_1$ intervals) and ventricular coupling intervals ($S_2S_2$) when retrograde $H_2$ activation consistently occurred via the same bundle branch. However, we noted sudden and unexpected increases (30–90 msec) or decreases (10–55 msec) in $S_2H_1$ intervals in 11 of 24 patients; these changes occurred with and without concomitant changes in the retrograde His-right bundle (HRB) activation sequence. Retrograde $H_2$ activation during right ventricular premature stimulation occurred exclusively through the LBB in a majority (67%) and through the RBB in a minority (12%) of cases. In some patients (21%), however, the route of retrograde $H_2$ activation may vary and could occur via both the LBB and the RBB. Recordings from the HRB area should help to delineate the route of retrograde impulse propagation to the bundle of His.

ATRIAL EXTRASTIMULUS TECHNIQUE has been the standard laboratory method for assessment of drug effects upon the functional properties of the His-Purkinje system (HPS).\textsuperscript{1-10} Determination of the refractoriness of the HPS in the antegrade direction in man, however, is frequently precluded by longer refractoriness of the atrioventricular (AV) node, particularly after drugs like digitalis and propranolol.\textsuperscript{11-10} The ventricular extrastimulus method circumvents the above difficulties, provides a different yet practical approach to this problem, and allows determination of refractoriness of the HPS in almost all patients, albeit in the retrograde direction.\textsuperscript{11-16} While the ventricular extrastimulus method is simple, it has some inherent limitations, particularly the lack of sufficient electrophysiologic markers. The retrograde His bundle activation during basic drive beats ($H_1$) is generally not recognizable. The His bundle depolarization in response to closely coupled ventricular premature depolarization ($H_2$) is easily identified, but does not provide any insight into the route of retrograde impulse conduction through the HPS, i.e., via the right (RBB) or left bundle branch (LBB) systems. The manner of impulse propagation is fundamentally important for the interpretation of drug effects on the HPS when using the ventricular extrastimulus technique. The present study delineates the routes of impulse propagation to the bundle of His during right ventricular premature stimulation in 24 patients with normal intraventricular conduction.

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using recordings from the RBB and the His bundle recordings. In addition, the usefulness and limitations of the various electrophysiologic parameters used to assess the retrograde functional properties of the HPS are discussed.

Methods

Patient Population

All studies were performed with the patient in a nonsedated, postabsorptive state. The procedure was explained to all patients and signed consents were obtained. Electrode catheters were percutaneously introduced into peripheral veins and fluoroscopically guided to positions in the region of the high right atrium, AV junction and right ventricle for local bipolar recordings and electrical stimulation. Recordings from the His bundle and the RBB were obtained either from two separate catheters or from the proximal and distal pairs of the same quadripolar catheter (interelectrode distance 1 cm). We considered the most proximal, clearly identifiable recording of the specialized conduction system from the AV junction to be representative of His bundle activity. For RBB recording, the catheter was advanced farther into the ventricle until the most peripheral recording possible was obtained. When using a single quadripolar catheter, we attached more importance to the location of the distal (right bundle) recording than to the proximal recording as long as the interval between proximal and distal recording was \( \geq 15 \) msec.\(^{17}\) We considered the proximal and distal temporal relationship of the recordings during sinus rhythm to be more important than the precise location of a given proximal recording (i.e., from the proximal, middle or distal portion of the His bundle). Complete electrophysiologic studies in these patients required two or three catheters and, therefore, no additional catheters were needed to complete the present protocol. In addition to the intracardiac electrograms (filtered at 30-500), three surface electrocardiographic leads (I, II and VI), and time lines were simultaneously displayed on a multichannel oscilloscope (Electronics for Medicine VR-12) and recorded on a magnetic tape (Honeywell Model 96) for later retrieval. The records were subsequently reproduced on photographic paper at 100-250 mm/sec. Electrical stimulation was performed using a digital stimulator (DTU, Bloom Associates, Philadelphia, Pennsylvania) capable of delivering rectangular impulses of variable voltage and duration. An isolation unit was also used for all electrical stimulation. To determine the retrograde refractory periods, we scanned the basic ventricular cycle lengths (S, S\(_r\), or V\(_r\),V\(_r\)) with progressively shorter S\(_r\)S\(_r\) (V\(_r\),V\(_r\)) intervals until no ventricular response could be elicited. In addition, we introduced premature ventricular stimuli (S\(_r\) or V\(_r\)) while atria and ventricles were paced simultaneously during the basic drive beats. Only the latter method was used to determine the retrograde refractory period studies in patients who had AV dissociation during ventricular pacing, thereby avoiding interruption of the basic drive due to intermittent ventricular captures from sinus beats. Programmed atrial extrastimuli (Sp or Ap) were also introduced after V\(_r\), when the latter demonstrated a retrograde block, in order to document the site of block, i.e., the AV node or the HPS. All electrical equipment was grounded and isolated and there were no untoward effects during these studies.

Twenty-four patients (14 males and 10 females), average age 57.7 years (range 19-81 years) were studied. Thirteen patients had had episodes of dizziness and lightheadedness, and syncope was documented in six of 13. The remaining patients had symptoms of paroxysmal rapid heart action. The underlying heart disease was arteriosclerotic in nine, primary myocardial in two, and mitral valve prolapse in two; the remaining 11 patients had no clinically detectable heart disease.

Definitions

A complete list of definitions of terms for both antegrade and retrograde conduction and refractory period studies has been published.\(^1\)\(^{11}\) Only the intervals pertinent to this report are defined below.

**Antegrade HPS Conduction.** The HV and RBV intervals were measured from the onset of the respective deflections to the earliest detectable ventricular activity on the surface ECG or on local electrogram recordings.

**Retrograde HPS Conduction.** The retrograde His bundle and RBB potentials for the basic drive beats (H\(_r\) and RB\(_r\)) could not be identified. When the two deflections emerged from local V\(_r\) electrogram, the S\(_r\)H\(_r\) (V\(_r\)H\(_r\)) and S\(_r\)RB\(_r\) (V\(_r\)RB\(_r\)) intervals were measured from the stimulus artifact (and local ventricular electrogram) to the onset of respective potentials.

In the absence of identifiable retrograde H\(_r\) potential, S\(_r\)H\(_r\) intervals were measured in lieu of retrograde H\(_r\)H\(_r\) intervals to determine the duration and magnitude of retrograde delays in the HPS at any given ventricular coupling interval. If the S\(_r\)H\(_r\) has a constant and fixed value, the S\(_r\)H\(_r\) intervals should parallel H\(_r\)H\(_r\) intervals, the former exceeding the latter by a constant amount in any given patient.

**Bilateral Retrograde Block HPS.** The longest V\(_r\),V\(_r\) interval where V\(_r\) retrogradely blocked below the His and RBB recording sites.

**Effective Refractory Period (ERP) of the Ventricular Muscle.** The longest S\(_r\),S\(_r\) interval when S\(_r\) did not produce a ventricular response.

Results

Stable recordings from the His bundle and RBB were obtained in 30 cases. Inclusion in this study required an RBV interval \( \leq 30 \) msec and a His-right bundle (HRB) interval \( \geq 15 \) msec. The 24 patients who met the above criteria are the subject of this report (table 1). The HV and HRB intervals in these 24 patients are within the range previously described by other investigators.\(^1\)\(^{19}\)\(^{20}\) We did not include the re-
remaining six patients because their HRB intervals measured < 15 msec. At the time of study, all patients were in sinus rhythm and had intact AV conduction except one patient, who had third-degree AV nodal block. Intraventricular conduction was normal in 22 of 24 patients and the remaining two patients (3 and 22) had an incomplete RBB block pattern on the resting surface ECG. Cardioactive medications were withheld for 48–72 hours before the study in all but two patients (4 and 11), who continued taking maintenance doses of digoxin. Patients with preexisting complete right or LBB block pattern, ventricular preexcitation, electrolyte imbalance and acute myocardial ischemia or infarction were excluded from this study.

Complete anterograde and retrograde conduction and refractory period data are available; however, only data pertinent to the main theme of this paper are presented.

Anterograde HPS Conduction Times (table 1)

The HV and RBV intervals were 40–50 (mean 45.0 ± 4.3) and 25–30 (mean 27.8 ± 2.5) msec, respectively, and the mean HRB interval was 17.2 msec (range 15–25 msec). In three patients (7, 13 and 14), an additional recording from the peripheral RBB was obtained which preceded the ventricular activation by 5–10 msec. However, these values were not included in table 1 because peripheral recordings were not available for most patients.

Retrograde HPS Conduction

Retrograde HPS conduction times could not be measured until the H2 and RB2 potentials emerged from the local V2 electrograms. The magnitude of retrograde HPS conduction delays (and consequent emergence of H2 or RB2) for any given S2S2 interval (at a given basic cycle length) was not predictable. The initial emergence of H2 or RB2 deflections from local V2 was gradual in 17 of 24 patients (fig. 1), but in seven of 24 patients (3, 5, 6, 10, 11, 14 and 15) both deflections simultaneously and abruptly cleared the end of local V2 by more than 50 msec. In patients who had gradual emergence of H2 or RB2, the clearance of the two deflections from the respective electrograms did not always occur at the same S2S2 intervals. The RB2

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**Table 1. Electrophysiologic Data**

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All values are in milliseconds.

Abbreviations: BCL = basic cycle length; ERP = effective refractory period; LBB = left bundle branch; RBB = right bundle branch; ret = retrograde; HPS = His-Purkinje system; VM = ventricular muscles.
potential was identifiable in six of 17 cases at S1S2 intervals that had not produced a recognizable emergence of H2 from local V2 (fig. 1). Conversely, in two of 17 patients the retrograde H2 potential could be identified before clearance of RB2 deflection. In the remaining nine cases, both H2 and RB2 emerged from local V2 at the same S1S2 interval. Table 1 lists the shortest S1H2 and S1RB2 delays when both deflections could be simultaneously delineated along with the ventricular coupling intervals that produced this emergence. At the earliest simultaneous emergence of retrograde H2 and RB2, the S1H2 interval measured less than S1RB2 in nine patients (1–8 and 19) with a mean H2RB2 value of 10.0 ± 3.5 msec and a range of 5–15 msec (table 1). The antegrade HRB intervals in the same nine patients ranged from 15–20 msec (mean 16.7 ± 2.5 msec). In another nine patients (9–16 and 20), when the two deflections were first recognized, the retrograde S2H2 and S2RB2 intervals were equal, such that the H2RB2 measured zero (figs. 1C and 1D), while an HRB interval ≥ 15 msec was recorded during sinus rhythm. The sequence of retrograde H2RB2 activation in the above 18 patients suggested retrograde depolarization of the His bundle and RB2 via the LBB. The reasons for such a conclusion along with validation of the HRB recordings have been published. In seven patients (17–19* and 21–24), when both H2 and RB2 were initially identified, the S2RB2 intervals were less than S2H2 by a mean of 15.0 ± 0.0 msec, indicating retrograde activation of the His bundle via the RB2. The antegrade HRB interval in these seven patients averaged 15.7 ± 1.9 msec (range 15–20 msec).

After initial emergence of the H2 and RB2, retrograde H2 activation continued to occur via the same route in patients 1–18; however, we noted a change in patients 20–24 as further scanning of the ventricular cycle length was carried out (fig. 2). The remaining patient (19) demonstrated retrograde H2 activation via the LBB during right ventricular apical pacing.

*Patient 19 had retrograde H2 activation via the LBB during right ventricular apical pacing and via the RBB during inflow pacing.
pacing, while retrograde H₂ activation occurred via the RBB during right ventricular inflow pacing through almost the full range of S₁S₂ intervals (fig. 3). The longest S₁H₂ intervals achieved via the RBB and LBB are shown in table 1. When retrograde H₂ activation occurred exclusively via the LBB (patients 1-16, table 1), the mean longest S₁H₂ value was 231.9 ± 43.9 msec (range 170-310 msec) for a basic cycle length range of 500-1000 msec (mean 712.5 ± 108.8 msec). The mean value for the longest S₁H₂ delays in patients 17-19, who demonstrated exclusive retrograde H₂ activation via the RBB, was 245.0 ± 8.7 msec (range 240-255 msec) at basic cycle length ranges of 700-900 msec (mean 766.7 ± 115.5 msec). Six patients (including patient 19) had retrograde activation of H₂ via both the RBB and LBB (at different times) at shorter S₁S₂ intervals (figs. 2 and 3; table 1). The shift in retrograde H₂ activation (i.e., from RBB to LBB or vice versa) had no particular pattern and occurred unpredictably (fig. 2). Table 1 gives the longest available S₁H₂ values when retrograde H₂ activation occurred via the RBB or LBB, but does not
indicate that such delays via the two bundle branch occurred at the same S₁S₂ intervals. Only patient 1 showed retrograde His bundle activation via both LBB (apical pacing) or RBB (inflow pacing) through almost the full range of S₁S₂ intervals and provided unique opportunity to compare the magnitude of S₂ delays via the two bundle branches at the same cycle length and ventricular coupling intervals (fig. 3).

Once the H₂ and RB₂ potentials emerged from the respective ventricular electrograms, further decrease in S₂S₂ generally produced almost linear increases in S₁H₂ and S₁RB₂ intervals, i.e., a 10–20-msec decrease in S₂S₂ resulted in a 10–20-msec increase in S₁H₂ and S₁RB₂ intervals in most cases (fig. 4A). However, in seven patients (7, 11, 16, 17, 19, 20, and 23), we noted sudden unexpected increase ≥ 30 msec (range 30–90 msec) in the S₁H₂ interval, with a small (10 msec decrease in S₁S₂ intervals (fig. 4B; table 2). Unexpected increases in S₁H₂ delays occurred with and without concomitant changes in the retrograde H₂ and RB₂ activation sequence (figs. 4B and 5; table 2). More surprising were the decreases in S₁H₂ delays of 10–55 msec noted in 10 patients at shorter ventricular coupling intervals (table 2). Such decreases in S₁H₂ intervals also occurred with and without accompanying changes in the retrograde H₂ and RB₂ activation sequence. The magnitude of S₁H₂ interval shortening was generally greater when a change in the retrograde HRB activation sequence accompanied S₁H₂ decrease (fig. 2) or when retrograde H₂ activation consistently occurred via the RBB (fig. 6 and table 2). When we noted shortening of S₁H₂ during the activation of H₂ via the LBB, the magnitude of S₁H₂ decrease was usually small, i.e., ≤ 20 msec (fig. 4B and table 2). The abrupt and unexpected increases or decreases in S₁H₂ delays noted in these patients were usually not main-
tained as the ventricular coupling intervals were further decreased. In most instances the S₂H₂ intervals returned to values which would be expected from the reciprocal and linear relationship between S₁S₂ and S₂H₂ intervals in most of the patients (figs. 4A and 4B). No patient in this series had a retrograde conduction delay or block between the His and right bundle recording sites during ventricular premature stimulation.

The S₁H₂ Interval

The S₁H₂ interval, representing a sum of ventricular coupling intervals and retrograde delays in the HPS, is obviously dependent upon S₁S₂ and S₂H₂ values. The measurements of S₂H₂ during full scanning of a basic cycle length provides a quantitative estimate of the degree of S₂H₂ prolongation for a given change in prematurity of the ventricular extrastimulus. The relationship between S₁S₂ and consequent S₂H₂ delays can be analyzed anywhere within the range of ventricular coupling intervals. For this study, however, only the shortest S₁H₂ delays achieved via the LBB and RBB are given (table 1). In patients 1–16, who showed retrograde H₂ activation via the LBB, the shortest S₁H₂ intervals had mean value of 481.6 ± 50.3 msec (range 385–590 msec) for the basic cycle length range of 500–1000 msec. The same parameter in patients 17–19, who demonstrated retrograde H₂ activation exclusively via the RBB, had a mean value of 490.0 ± 13.2 msec (range 480–505 msec) for the basic cycle length range of 700–900 msec. Of six patients (19–24), in whom the retrograde H₂ activation occurred via both the LBB and RBB, the minimum S₁H₂ values were obtained from the LBB in four and the RBB in two. In all of these patients, conduction via the contralateral bundle occurred only intermittently and the shortest achievable S₁H₂ intervals via the two bundle branches could not be compared except in patient 19 (fig. 3 and table 1).

Bilateral Retrograde Block HPS

At certain S₁S₂ intervals in 12 of 24 patients, V₂ retrogradely blocked bilaterally within the HPS, i.e.,
no H2 or RB2 was recorded while the two deflections were present at longer coupling intervals. During the present study, the site of retrograde block (the AV node or the HPS), was also confirmed with programmed atrial stimulation (Sp or Ap) after the blocked V2. This method permitted determination of retrograde block in the HPS even before the emergence of H2 and RB2 from the local V2 electrogram (fig. 7). The values presented in table 1 represent ventricular coupling intervals where V2 initially blocked in the HPS and do not imply that V2 continued to block at shorter S1S2 intervals (see below).

**Retrograde Gaps HPS (table 2)**

The retrograde block of V2 in the HPS was not maintained in any of the 12 patients as further scanning of the basic cycle length was carried out with shorter S1S2 intervals. Thus, in all 12 patients, conduction to the His bundle resumed after a zone of initial block (gap zone). Ten patients (1-4, 16, 17, 19, 20, 23 and 24) had retrograde activation of the H2 via the same bundle branch before and after the zone of block (figs. 3, 4, and 7; table 2). In three patients (21, 23 and 24*), retrograde activation of the His bundle occurred via the RBB before the block and via the LBB during resumption of conduction (fig. 2).

Table 2 lists the maximum change in S2H2 produced by a given change in S1S2, with or without a shift of conduction to the contralateral bundle branch and in both the presence and absence of the gap phenomenon. The increases and decreases in S2H2 delays before and after the zone of block are comparable whether or not a change in HRB activation accompanied resumption of retrograde conduction to H2. In five of these 12 patients, V2 again showed a retrograde block in the HPS at shorter coupling intervals and continued to block up to the point of the ERP of the ventricle in four of five (figs. 3 and 4).

**Reproducibility of Retrograde Electrophysiologic Parameters**

In all patients, the ventricular premature depolarizations with the same S1S2 interval were introduced two or three times in quick succession, and the magnitude of delays was quite reproducible in most patients. Some patients, however, had unexpected changes in S2H2 delays as outlined above, and in these cases the degree of such delays at the same S1S2 was not reproducible. The analysis of S2H2 intervals over the full range of S1S2 intervals showed that the shortest S2H2 delays in any given patient were the most consistent and reproducible parameter of HPS. The zones of retrograde block in the HPS showed variations of 10-20 msec on repeat scanning; in some patients, however, the values were quite reproducible.

**Effect of Simultaneous Atrial and Ventricular Pacing on Retrograde HPS Conduction**

In addition to the conventional method (see Methods), retrograde refractory period studies were also done using simultaneous atrial and ventricular...
Table 2. Maximum Changes in $S_2H_2$ in Response to Changes in $S_1S_2$

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All values are in milliseconds.
The changes are the maximum for the degree of $S_1S_2$ shortening available (10-20 msec).
LB-LB and RB-RB indicate no change in retrograde His-right bundle activation; RB-LB and LB-RB indicate a change in retrograde His-right bundle activation.

*Route of $H_2$ activation before retrograde block.
†Route of $H_2$ activation after resumption of $S_2H_2$ conduction.

Abbreviations: LB = left bundle; RB = right bundle; $\Delta$ = change; HPS = His-Purkinje system; gap zone = $S_1S_2$ ($V_1V_1$) intervals where $V_1$ retrogradely blocks bilaterally in the His-Purkinje system.

Discussion

In patients with normal antegrade intraventricular conduction, premature ventricular impulses can depolarize the His bundle by either the RBB or the LBB. Knowing the route of retrograde impulse conduction both during the basic drive beats and the
FIGURE 6. Patient 17. At a basic ventricular cycle length (VCL) of 900 msec and $S_1S_2$ of 340 msec, $H_2$ activation occurs via the right bundle branch (RBB). Panel B shows a 55-msec decrease in the $S_2H_2$ interval at a shorter $S_1S_2$ of 310 msec without a change in the sequence of retrograde His-right bundle (HRB) activation. Sequence of antegrade and retrograde HRB activation is shown with perpendiculars.

premature beats, therefore, is important in order to draw any meaningful conclusions concerning the effect of drugs upon retrograde HPS conduction and refractoriness.

Retrograde Activation of His Bundle
During Constant Cycle Length Pacing

During right ventricular basic drive beats, retrograde activation of the His bundle ($H_1$) is expected to occur via the RBB and it has been demonstrated in the intact canine heart.\(^1\) The same impulse also conducts transseptally to engage the LBB with delayed arrival at the His bundle such that the two wave fronts probably collide somewhere along the LBB-Purkinje-muscle system; the exact location is variable.

Retrograde Activation of the His Bundle
During Right Ventricular Premature Stimulation

Although retrograde $H_1$ activation is expected to occur consistently via the same route, retrograde $H_2$
Figure 7. Patient 1. The ventricular cycle length (V1, V2) is 700 msec in panels A-F. The basic drive consists of ventricular pacing alone (panels A-C) and simultaneous atrial and ventricular pacing (panels D-F). After the last beat of the basic drive, a programmed atrial extra stimulus (Sp or Ap) is introduced at an S,Sp (or V1,Sp) interval of 800 msec in all panels. The Sp conducts to the His bundle (HB) with SpH interval of 130 msec in panel A and 120 msec in panel D when V2 is omitted after the last V1. When premature ventricular depolarizations are introduced at S1,Sp intervals of 400 msec (panels B and E), V2 is not followed by A2 (ventriculoatrial block). The site of retrograde block (i.e., the atrioventricular (AV) node or the His-Purkinje system (HPS) cannot be directly determined in panels B and E because retrograde H2 has not yet emerged from the V2 electrogram. However, the SpH intervals in panels B and E is prolonged (compared with panels A and D, respectively), which suggests that V2 retrogradely reached and blocked in the AV node (retrograde concealed conduction AV node). The V2 continues to block retrogradely at shorter S1,S2 intervals of 380 msec (panels C and F). However, a return of SpH values in panels C and F to those seen in panels A and D, respectively, suggest that V2 blocked distal to the AV node (i.e., HPS). The figure shows 1) indirect determination of the site of retrograde block after V2 with the use of programmed atrial extra stimuli, and 2) that the onset of retrograde block in the HPS is identical to the two methods of stimulation shown. BCL = basic cycle length; HRA = high right atrial.

Activation could occur via either bundle branch. The route of retrograde impulse propagation to the His bundle during ventricular premature stimulation cannot be delineated with certainty using His bundle recordings alone. Even using both His and RBB recordings, we could not detect the routes of impulse propagation to the His bundle until both retrograde H2 and RB2 could be clearly defined. The manner of H2 emergence from V2 (gradual or abrupt) also did not distinguish whether H2 activation was via the RBB or
the LBB. Although the linear relationship between S1S2 and S1H2 intervals suggested H2 activation via the same bundle branch, abrupt increases or decreases in the S1H2 delays did not necessarily indicate a shift in H2 activation via the contralateral bundle branch. The linear relationship between S1S2 and S1H2 intervals suggested consistent conduction via the same bundle branch, but the findings were not particularly helpful in determining whether H2 activation occurred via the RBB or LBB. From the His bundle recordings alone, the route of impulse propagation (RBB or LBB) could only be delineated when macroreentry in the HPS (V2 phenomenon) accompanied retrograde H2 activation.11, 22

At the initial emergence of the H2 and RB2 deflections and subsequently at shorter coupling intervals, retrograde H2 activation occurred more frequently via the LBB (table 1). We do not know the reason for this phenomenon, but there are several possible explanations.

1) For the same S1S2 interval, proximity of the pacing site may allow delivery of closer consecutive ventricular impulses (V1V2) to the RBB (and hence within its ERP) than to the LBB. While this mechanism might operate at very short S1S2 intervals where a stimulus to ventricular response latency may exist, it appears an unlikely explanation at longer coupling intervals where no intramural conduction delay can be shown.

2) If the two consecutive impulses (S1S2) reach both the RBB and LBB at the same coupling interval (V1V2) (although in a different temporal sequence), we postulate that the inherent retrograde refractoriness of the LBB is generally shorter than that of the RBB. The term refractoriness as used here implies that the RBB either had a complete retrograde block or sufficiently slow conduction such that S2 impulse reached the His bundle sooner via the LBB.

3) The partial antegrade penetration of the LBB during the basic drive beats could alter the retrograde conduction along the LBB. Dual excitation of the LBB from the two wave fronts may facilitate conduction of V2 along the LBB. Whether these or other mechanisms are responsible in a given circumstance are not clear from the data available.

In one-third of the cases, the H2 activation occurred via the RBB either exclusively (patients 17-19) or intermittently (patients 20-24). Exclusive activation of the H2 via the RBB in patient 19 appeared to be related to the site of stimulation, whereas in the remaining two cases (patients 17 and 18), exclusive activation of H2 via the RBB might have the same basis, or retrograde refractoriness of the LBB was equal to or exceeded that of the RBB. Intermittent activation

Figure 8. Patient 9. The tracings show scanning of the same cycle length at the same S1S2 intervals as in figure 1, except that the atria are also being paced during the basic drive. The pattern of emergence of retrograde H2 and RB2, the sequence of His-right bundle (HRB) activation and degree of S1H2 and S1RB2 delays are identical to those in figure 1. BCL = basic cycle length; HRA = high right atrial; HB = His bundle.
of H_2 via the RBB in patients 20–24 probably resulted from: 1) excessive retrograde conduction delay, or block in the RBB, i.e., when H_2 activation via the RBB occurred in association with S_2H_2 prolongation; or 2) sudden facilitation of retrograde conduction along the RBB, i.e., when H_2 activation was accompanied by shortening of S_2H_2 intervals. A complete retrograde block in the RBB or LBB is not an unexpected finding, because at least 50% of the patients in this series had complete bilateral retrograde block in the HPS (i.e., both RBB and LBB systems).

**Retrograde Gap Phenomenon HPS**

The retrograde gap phenomenon in the HPS has been described; here it is analyzed in greater detail. We saw that retrograde gaps frequently occur within the same bundle branch system, i.e., the V_2H_2 conduction occurred via the same bundle branch before and after the zone of block (figs. 3 and 4). Sometimes, however, H_2 activation occurred via the RBB before and via the LBB after the zone of bilateral block in the HPS (fig. 2).

Sudden prolongation and shortening in S_2H_2 delays occurred with and without concomitant gap phenomena. Although an increase in S_2H_2 interval may be expected with a switch of H_2 activation from the RBB to LBB (due to additional transseptal conduction time), this finding was not common. In fact, we could identify no uniform pattern of retrograde conduction in the HPS, as we noted both decreases and increases with and without change in HRB activation sequence and with and without associated gap phenomenon.

Sudden shortening in S_2H_2 at closer S_2S_2 intervals with or without changes in HRB activation sequence is even more difficult to explain. To provide a unified concept for this observation, the explanation of proximal conduction delay and distal recovery of excitability used to explain the gap phenomenon does not appear to be adequate. During antegrade gap where the initial site of block and area of proximal delay are both in the HPS, the H_2V_2 intervals during resumption of AV conduction are generally longer than they are before the block (personal observation). Only narrow zones of antegrade H_1H_1 intervals are generally available and it is not known if shortening in H_2V_2 will also occur during antegrade gaps if closer coupling intervals could be achieved.

The concept of summation and inhibition introduced by Cranefield and Hoffman may provide an alternate explanation. The HPS, consisting of extensive number of fibers both in the form of Purkinje network and more compact strands and bundles, may not function as a single unit. Different fibers could potentially show different conduction and refractory period properties depending upon the time, direction and magnitude of the arriving impulse. Progressive and linear increase in S_2H_2 intervals may represent periods of more uniform propagation of the impulse along different wave fronts. A retrograde block in one or both bundle branch systems could occur with the inhibition of a potentially propagable impulse due to depolarization of tissue ahead by a decremental impulse (which fails to propagate). Summation of the two or more wave fronts could theoretically produce effective propagation (and gap phenomenon), and even shortening of S_2H_2 delays if summation took place in relatively faster conducting fibers. The precise location of retrograde conduction delay and block is not known, but does not appear to be within the proximal portion of the bundle branches as suggested by the present results. Another explanation for abrupt shortening of S_2H_2 intervals is the existence of true supernormal conduction, a possibility that we cannot exclude, based on the data available.

Although there is no clear explanation for the sudden increases and decreases in retrograde conduction delays, they are probably true electrophysiologic phenomena. A variable level of antegrade penetration of the LBB during right ventricular basic drive could artificially influence the S_2H_2 conduction via the LBB and account for such results. However, the occurrence of such findings during retrograde H_2 activation via the RBB and the shift of conduction via the contralateral bundle suggest their existence as true electrophysiologic events. Similarly, it is possible that simultaneous atrial and ventricular pacing during the basic drive beats in some way might have influenced the retrograde HPS conduction. In these patients, H_2 activation occurred in the retrograde direction (i.e., from ventricular impulse), even with simultaneous atrial pacing. This observation is supported by the fact that the parameters of retrograde HPS conduction and refractoriness were the same (whenever comparison was possible) regardless of the method of study.

**Role of Ventricular Extrastimulus Technique in Assessing Drug Effects on Retrograde HPS Conduction**

The consistent or intermittent activation of H_2 via the RBB at close coupling intervals in a significant number of cases makes the route of impulse propagation in the HPS without using RBB recordings uncertain unless macroreentry HPS co-exists. Since H_2 activation via the LBB incorporates transseptal conduction time, a basic physiologic difference exists between S_2H_2 intervals obtained via the RBB and those achieved via the LBB. The parameters of retrograde HPS conduction, (S_2H_2 and S_2H_2 intervals) could be significantly influenced by shifts in routes of impulse propagation to the bundle of His, and analysis of these parameters can be further complicated by a therapeutic agent that can produce a shift of conduction to the contralateral bundle branch. Therefore, when using the V_2 method to assess drug effects on the HPS, we recommend comparing control data and drug effects within the zone of macroreentry HPS or using additional (RBB) recordings from the AV junction.

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