Site of Conduction Delay During Functional Block in the His-Purkinje System in Man

MASOOD AKHTAR, M.D., CAROL GILBERT, R.N., CCRN, MAMOUN AL-NOURI, M.D., AND STEPHEN DENKER, M.D.

SUMMARY Using recordings from the His bundle (H) and right bundle (RB), the site of functional conduction delay and/or block along the H-RB axis was analyzed in 14 patients with normal intraventricular conduction. During sinus rhythm, the HV and RB-V intervals had a mean value of 46.4 ± 3.6 msec (range 40–50 msec) and 22.8 ± 7.5 msec (range 5–30 msec), respectively, with an average H-RB interval of 23.6 ± 9.1 msec. In response to single atrial premature beats, all 14 patients developed right bundle branch block (RBBB), while nine of 14 also developed left bundle branch block (LBBB) as well as bilateral block within the His-Purkinje system (HPS). During RBBB, the RB potential disappeared in 13 of 14 patients with and without concomitant increase in the HV interval, suggesting an increase in the H-RB interval due to conduction delay distal to the H but proximal to the RB recording site. In two of these 13 patients and the remaining patient, RBBB also occurred without a corresponding increase in the H-RB interval, indicating conduction delay and/or block distal to the RB recording. An increase of 40–325 msec in the H-RB interval was noted during LBBB. During bilateral block within the HPS, a clearly recorded H potential was not followed by RB deflection. When either the RBBB (six patients) or LBBB (three patients) persisted for two or more successive premature beats, the H-RB interval lengthened preceding the first beat but normalized during subsequent beats. The data suggest that the site of functional conduction delay and/or block along the H-RB axis is generally proximal (i.e., distal H or proximal RB), but could vary in the same patient, and can quickly shift from a proximal to a distal location with successive short cycles.

FUNCTIONAL CONDUCTION DELAY or block within the His-Purkinje System (HPS) is frequently observed during antegrade propagation of premature impulses.1–6 In response to atrial premature beats, both right and left bundle branch block (RBBB and LBBB), as well as bilateral block within the HPS, have been noted in patients with normal intraventricular conduction.2, 4, 5 Using His bundle (H) electrograms alone it has not been possible to delineate the site of functional block within the HPS (i.e., within H, proximal bundle branches or more distally) in man. Using additional recordings from the RB or LBB, the location of functional block in the HPS has been studied in the intact canine heart.7–9 Although some information is available, few systematic studies have been carried out to determine the site of such a block in the intact human heart.10 The site of functional conduction delay and/or block along the His–right bundle (H-RB) axis was therefore analyzed in 14 patients with normal intraventricular conduction using simultaneous recordings from the H and RB. In this report we present findings in these cases and discuss the electrophysiologic implications of these observations.

Methods

Electrophysiologic studies were performed in unsedated, postabsorptive patients using previously described techniques.11 The procedure was explained to all patients and signed consents were obtained. Percutaneously introduced quadriolar electrode catheters were fluoroscopically guided to and positioned in the right atrium, the atrioventricular (AV) junction and the right ventricle. The simultaneous recordings from the H and RB were obtained either with a single quadriolar catheter (interelectrode distance 1 cm) or with two separate electrode catheters.12, 13 All intracardiac electrograms (filtered at 30–500 Hz), three surface electrocardiographic leads (I, II and V1) and time lines were simultaneously displayed on a multichannel oscilloscope and recorded on magnetic tape for later reproduction. Electrical stimulation was performed using a digital stimulator capable of delivering rectangular impulses of variable voltage and duration. All electrical equipment was carefully grounded and patient isolation existed during these studies; no problems were encountered. Aberrant conduction and block in the HPS in these patients was produced by using one or more of the following methods:

1) The conventional method of atrial premature stimulation in which a premature atrial beat (S2 or A2) was delivered after a number (usually eight) of spontaneous sinus or predetermined paced atrial cycle lengths (S1S1 or A1A1). To sustain aberrant conduction over several successive beats, additional premature beats (A2, A3, etc.) were used in some patients when feasible.

2) Premature atrial stimulation coupled to a spontaneous sinus beat, producing a rhythm in which every other beat was premature.2 This technique of stimulation appreciably increases the frequency of aberrant conduction and/or bilateral block in the HPS by producing a long HH cycle preceding an atrial premature beat (unpublished observations).
3) Sudden initiation of rapid atrial pacing after a regular sinus cycle or a sinus escape cycle. A similar sequence of events was also initiated during constant cycle length atrial pacing with either deliberate omission of atrial stimulation for one or two cycles or spontaneous occurrence of missed atrial captures.

Definitions of Terms

The HV and RB-V intervals were measured from the onset of respective deflections to the earliest detectable ventricular activity, whether recorded on surface ECG or one of the local electrograms.

Patient Population

The patients were 20–68 years old (eight males and six females) and were studied for various reasons, predominantly for palpitations or syncope. The underlying cardiac disease was arteriosclerotic in six patients and mitral valve prolapse in two, while the remaining patients had no clinically evident heart disease. No patient in this series was taking cardioactive medication at the time of or within 72 hours before the study. All patients had normal intraventricular conduction. Patients with preexisting bundle branch block (BBB) or catheter-induced RBBB pattern were excluded.*

Results

Although stable recordings from both the H and RB were obtained in several patients, inclusion in this study required 1) HV ≥ 40 msec, 2) RB-V ≤ 30 msec, 3) H-RB ≥ 15 msec and 4) the occurrence of functional aberrancy and/or block in the HPS. Both antegrade and retrograde refractory period data were available for all patients. However, only the results dealing with antegrade conduction in the HPS will be presented here.

HPS Conduction Times During Sinus Rhythm

The HV and RB-V intervals ranged from 40–50 msec (mean 46.4 ± 3.6 msec) and 5–30 msec (mean 22.8 ± 7.5 msec), respectively. (table 1). The mean H-RB interval in these patients was 23.6 ± 9.1 msec (range 15–40 msec).

RBBB Pattern After Single Atrial Premature Beats

All patients in this series developed a RBBB pattern with one or more of the atrial stimulation techniques listed above. In eight of 14 patients, various degrees of RBBB, ranging from minimal terminal delay to a complete RBBB pattern, were observed during atrial premature stimulation. When the QRS complex showed an incomplete RBBB pattern, a concomitant increase in the H-RB interval was noted without simultaneous change in the HV intervals, suggesting conduction delay proximal to the RB recording site (figs. 1B and C). The RB-V interval therefore showed a corresponding decrease in values compared with normally conducted beats (fig. 1). When the QRS complex showed a more complete RBBB pattern (QRS duration ≥ 120 msec), the RB potential disappeared (figs. 1 and 2) in 13 of 14 patients (patients 1–13, table 1), indicating conduction delay and/or block proximal to or within the area of RB recording. The disappearance of RB deflection occurred with or without corresponding prolongation in the HV intervals. In patient 14, the H-RB interval did not change during RBBB, suggesting conduction delay or block distal to the location of RB recording (fig.

<table>
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<th>Pt</th>
<th>HV (msec)</th>
<th>RB-V (msec)</th>
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<th>Site of block after A by</th>
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Abbreviations: HPS = His-Purkinje system; RBBB = right bundle branch block; LBBB = left bundle branch block; + or = presence or absence of a given finding proximal (P) or distal (D) to right bundle recording.


In general, the site of conduction delay and/or block after A2 remained the same in a given patient over a wide range of cycle lengths (i.e., both H1H1 and H1H2). However, patients 12 and 13, who predominantly had conduction delay between the H-RB recordings during RBBB, on occasion also had a RBBB pattern associated with conduction delay distal to the RB potential. Although no consistent pattern of site of conduction delay could be identified in these two patients, the conduction delay was more frequently proximal to the RB with longer preceding (H1H1) cycles and distal to the RB with shorter preceding cycle lengths.

LBBB Pattern After Single Atrial Premature Beats

Nine of the 14 patients (patients 2, 6–13, table 1) also had a LBBB pattern. Frequently both RBBB and LBBB patterns were observed during scanning of sinus cycle length or after A2 coupled to the sinus escape beats (fig. 4). Unlike findings during RBBB, the RB potential during LBBB was always clearly identifiable, providing an opportunity to measure the magnitude of H-RB delays directly during functional BBB. The H-RB intervals preceding the beat showing a LBBB pattern were always longer than the H-RB intervals of the sinus beats. The increase in the H-RB intervals (compared with sinus beats) preceding the QRS complexes and displaying a more complete LBBB pattern (QRS duration ≥ 120 msec) was 40–325 msec (figs. 4 and 5). During incomplete LBBB pattern (seen rarely), however, lesser degree of H-RB interval prolongation (relative to the sinus beats) occurred. While the HV intervals preceding LBBB increased in all patients, the RB-V intervals remained the same in all patients during LBBB after single atrial premature beats.
Bilateral Block in the HPS
After Single Atrial Premature Beats

The eight patients who had both RBBB and LBBB patterns and patient 14 showed a bilateral block in the HPS in response to $A_2$ (table 1). In most instances the block along the H-RB axis was between the H and RB recording sites: the $H_2$ was recorded but the $RB_2$ deflection could not be identified (figs. 4 and 5A). However, patient 14 had block distal to the RB recording site (fig. 3B).

In six of nine patients a bilateral block in the HPS was also induced during rapid atrial pacing. In none of the six cases did an identifiable RB potential follow the H potential during the first blocked atrial impulse. When the 2:1 block was sustained over several cycles, however, the RB deflection gradually reappeared (fig. 6), which suggests a shift in the site of block from a proximal to a distal location (see also below) despite continuation of a 2:1 block.

Sustained Bundle Branch Block Pattern

In patients 1, 5, 8, 9, 12 and 13 (table 1), RBBB could be sustained for two beats or more, either by rapid atrial pacing or by introducing successive atrial premature beats ($S_2S_3$, etc.). The first conducted beat with the RBBB pattern was not preceded by an identifiable RB potential regardless of whether the accompanying HV interval was prolonged (figs. 6B and 7). In all instances, however, the RB potential reappeared during the subsequent beats with RBBB pattern. The HV, RB-V and H-RB intervals preceding the second, third and subsequent beats with RBBB pattern had the same value as the sinus beats (fig. 7). Patients 8, 9 and 13 also had a sustained LBBB pattern over several
FIGURE 3. The basic atrial cycle length is 700 msec in both panels, and the $H, V_1$ and $R_B, V_1$ measure 45 and 25 msec, respectively. The $A_2$ in panel $A$ conducts with a right bundle branch block (RBBB) pattern, while in panel $B$, $A_2$ shows a block in the His-Purkinje system (HPS). In both panels the $H-R_B$ interval is the same as sinus beats, and conduction delay and block are distal to the $R_B$ recording site. $HRA = \text{high right atrial electrogram}; \ HB = \text{His bundle electrogram}; \ RB = \text{right bundle electrogram}$. All measurements are in milliseconds.

FIGURE 4. The basic atrial cycle length in panels $A$ and $B$ measures 700 msec. At an $A_1A_2$ of 370 msec (panel $A$), $A_2$ conducts with a left bundle branch block (LBBB) pattern. The $H_2V_2$ intervals measure 80 msec more than $H_1V_1$, but the $R_BV_2$ remains the same (25 msec) as $R_BV_1$. With a 10-msec decrease in $A_1A_2$ (panel $B$), the $A_2$ fails to produce a ventricular response. In panel $B$, $H_2$ is clearly identifiable but no $R_B$ is recorded. Panel $C$ shows induction of both LBBB and right bundle branch block (RBBB) patterns with atrial premature beats coupled to sinus escape beats. All atrial coupling intervals measure 480 msec and only slight variation is seen during the cycle length preceding the premature beats. The right bundle ($RB$) potential is recognizable preceding the LBBB and normal conduction but not preceding the RBBB. The $H-R_B$ increases by 55 msec (not labeled) preceding the beat showing the LBBB pattern. $HRA = \text{high right atrial electrogram}; \ HB = \text{His bundle electrogram}; \ RB = \text{right bundle electrogram}$. 
successive cycles. The HV and H-RB intervals preceding the first aberrantly conducted beat were prolonged, and both intervals normalized during subsequent beats despite continued LBBB pattern (fig. 6B).

**Discussion**

The availability of simultaneous recordings from the H and RBB during this study made it possible to analyze the site of conduction delay during functional block in the HPS. Although the precise location of the H and RB recordings in a given case is uncertain with the technique used, the HV and RB-V intervals in these patients were within the ranges expected for patients with normal intraventricular conduction.10 12 15

We examined whether the disappearance of RB deflection during the present study represented unstable local recording or a true electrophysiologic phenomenon. Conceivably, sufficient displacement in catheter position can take place with atrial contraction before or coincident with the timing of H or RB activation, rendering the local depolarizations unrecordable by the catheter. Such catheter displacement could vary at different coupling intervals and preceding cycle lengths, as well as with variation in respiratory cycles. The following reasons, however, suggest that the changes in H-RB intervals and disappearance of RB deflections during this study were true electrophysiologic events:

1) Disappearance of RB potential occurred only during RBBB and bilateral block HPS and not during LBBB or normal conduction. The coupling intervals and preceding cycle lengths during RBBB and bilateral block HPS were frequently closely comparable to coupling intervals and preceding cycle lengths during LBBB or normal conduction (figs. 1–4). These observations suggest that disappearance of RB deflection cannot be solely ascribed to changes in
coupling intervals and preceding cycle lengths. The reappearance of RB potential during sustained 2:1 AV block in the HPS without any changes in the cycle lengths also supports the above reasoning.

2) Reappearance of RB deflection during sustained RBBB suggests that the RB potential was recordable if local conduction delay did not exist.

3) Recordability of the RB potential or lack thereof appeared to correlate with the type of conduction delay or block (RBBB vs LBBB, etc.) rather than with respiratory cycles; therefore, the role of the respiratory cycle, if any, was inconsequential in determining the disappearance of RB potential. Even though not presented here, both the H and RB recordings in all of these cases remained stable during retrograde refractory period studies as well, further attesting to the stability of both recordings over a wide range of basic cycle lengths, coupling intervals and methods of stimulation.\textsuperscript{12, 13}

Consequently, the disappearance of RB potential during RBBB represented 1) conduction delay proximal to the RB, with effective propagation to the RB recording site producing an increase in the H-RB interval (the RB deflection being obscured within the succeeding ventricular electrogram); 2) block proximal to the RB recording site; and 3) markedly slow conduction or block in the region of RB recording rendering the RB potential unrecordable.

\textbf{FIGURE 6.} \textit{A} A period of stable functional 2:1 atrioventricular block in the His-Purkinje system (HPS) before resumption of 1:1 conduction. The second, fourth and sixth atrial impulses are followed by His (H) but no right bundle (RB) potentials. The eighth atrial impulse, however, is followed by both H and RB potentials and the HRB interval measures the same as sinus beats. The tenth atrial impulse conducts with a left bundle branch block (LBBB) pattern and is preceded by prolonged HV and RBV intervals and a normal HRB interval. The association of delay distal to RB recording in association with LBBB at this moment is because the site of delay had already shifted from a proximal to a distal location (beyond the RB potential) before resumption of 1:1 conduction. Perpendiculars are drawn in appropriate places to show the timing of the HRB activation. \textit{B} Atrial pacing at a constant cycle length of 380 msec and two missed atrial captures (seventh and eleventh stimuli). The second atrial impulse is followed by H but no RB deflection, while the third atrial impulse conducts normally. The next two beats conduct with a LBBB pattern; the HV and H-RB intervals preceding the first aberrant beat are prolonged but return to normal with the second aberrant beat. The RB-V interval preceding both beats with LBBB pattern measure the same as sinus beats (not labeled). During the first beat with a RBBB pattern (seventh QRS complex), the HV interval is prolonged and no RB potential is recorded; however, the HV and RB-V intervals preceding the second beat with RBBB measure the same as normal beats and are shown by perpendiculars. HRA = high right atrial electrogram.
The existence of conduction delay proximal to the RB recording resulting in H-RB prolongation was directly documented during LBBB and may also have existed during RBBB, although the RB potential could not be identified within the corresponding local ventricular electrogram. Whether the complete disappearance of RB potential shown during bilateral block HPS (or RBBB) represented failure of impulse propagation to the RB recording site or markedly slow conduction at the recording site is uncertain because the two may be indistinguishable with the methods used. It is reasonable to state, however, that disappearance of RB deflection indicated conduction delay or block proximal to or at the RB recording site.

Data from isolated canine preparations consisting of RBB and Purkinje system muscle indicate that the action potential duration is longer in more peripheral Purkinje network compared with the RBB. Because the local refractory periods generally parallel the changes in action potential durations, the refractoriness of the peripheral Purkinje network (i.e., the area of gate) exceeds the more proximal RBB. However, in isolated preparations containing both the H and its bifurcation in addition to the more distal bundle branches and Purkinje network, the limiting area for antegrade conduction is located higher at the level of bundle branches. Similar observations have been made in the intact canine heart as well.

The present results suggest that in patients with normal intraventricular conduction, the refractoriness of the distal H and/or proximal RBB generally equals or exceeds that in the more distal area of the RBB. Conduction delay proximal to the RB recording during functional RBBB has also been noted in man. Findings of conduction delay and block distal to the RB recording site in some cases, however, indicate that the refractoriness of tissues beyond the RB recording may occasionally exceed that of the proximal HPS. It is also possible that cycle-length–induced changes in the refractoriness may be quantitatively different in the proximal H-RB vs distal RB-Purkinje muscle system in some patients. The site of conduction delay and/or block after a single atrial premature beat may therefore vary in the same subject with changes in the basic cycle length. The technique used here only permitted analysis of conduction delay or block along the H-RB axis. Conduction delay or block along the H-LB axis could also have similar distribution, although we cannot confirm this directly.

To what extent the delay between the H and RB contributed to the RBBB pattern in a given patient at a given moment cannot be determined from the data available. Nor are we certain whether conduction delays within the distal H rather than the proximal RB induced by atrial premature beats contributed to RBBB pattern in some cases (longitudinal dissociation within the distal H). Spontaneous occurrence of a BBB pattern due to longitudinal dissociation in the H has been shown in the human heart. Although a direct correlation of the magnitude of delay along the
H-RB axis with QRS morphology cannot be made, it is clear that marked degrees of conduction delay can exist within a relatively small area of the HPS (fig. 5), with a normal activation time as short as 15-20 msec. The magnitude of such delays is certainly more than adequate to allow total ventricular activation via the contralateral bundle branch.

Whereas conduction delays both proximal and distal to the RB recording were noted after single atrial premature beats in some patients, the site of delay or block was uniformly distal to the RB recording during successive aberrant beats (figs. 6 and 7). The exact mechanism for the above findings is not clear; however, some explanations can be offered as follows:

1) If the first premature impulse activates the RB (regardless of succeeding QRS morphology), it will shorten the refractoriness of the RB, and subsequent impulses will conduct more rapidly due to the newly abbreviated refractory period of the RB, producing a return of recordable refractory period of the RB. Very slow conduction at the RB recording site during RBBB pattern could render the local potential unrecordable by the catheter even if the actual block occurred more distally.

2) When the first premature atrial impulse blocks at the site of RB recording, the refractoriness of the H and proximal RB activated by the impulse may still shorten, permitting conduction of subsequent impulses past the RB recording site.

3) If the first atrial impulse blocks proximal to the area of RB recording, retrograde activation of the RB during ventricular activation via the LB will still occur prematurely and could shorten the refractory period of the RB.

The antegrade shift in the site of conduction delay or block with successive cycles should eventually abolish the BBB. A BBB pattern, however, could persist over prolonged periods due to retrograde penetration distal to the area of antegrade block.

The more gradual transfer in the site of block from a proximal to a distal location during 2:1 block in the HPS (fig. 6) can best be explained by a gradual shift in the antegrade penetration level of the consecutive non-conducted atrial impulses. During 2:1 AV block in the HPS, the RBB Purkinje system distal to the site of block registers a cycle length that is equal to the conducted beats, i.e., the RR intervals. Shortening of the refractoriness in a given area along the H-RB axis (with consequent conduction to or beyond it) is therefore primarily dependent upon the antegrade penetration level of the prior blocked atrial impulse, because retrograde penetration of RB is not expected to occur. The antegrade shift in the level of penetration with consecutive blocked beats along the two bundle branches appears asynchronous, as suggested by aberrant conduction of the first beat during resumption of 1:1 AV conduction (figs. 6A and B). The site of block during the initial aberrant beat may be proximal to the RB recording site (fig. 6B, third QRS) or distal to it (fig. 6A, sixth QRS), depending upon the level of impulse penetration before resumption of 1:1 AV response.

Acknowledgments

The authors thank Kathryn Corriere and Robert Walters for their assistance and special thanks to Dr. Andrew L. Wit for his critical review of the manuscript.

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Circulation. 1980;61:1239-1248
doi: 10.1161/01.CIR.61.6.1239

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

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