Reentry Within the His-Purkinje System

Elucidation of Reentrant Circuit Using Right Bundle Branch and His Bundle Recordings

MASOOD AKHTAR, M.D., CAROL GILBERT, R.N., C.C.R.N., FRANCIS G. WOLF, AND DONALD H. SCHMIDT, M.D.

SUMMARY Routes of impulse propagation during reentry in the His-Purkinje system (Re-HP) in 13 patients were studied using the right ventricular extra stimulus (V2) method and right bundle branch (RBB) recordings in addition to the conventional His (H1) bundle recordings. The H-V and RB-V intervals during sinus rhythm averaged 44.2 and 27.6 msec respectively, with the average antegrade H-RB interval 16.6 msec. All 13 patients demonstrated Re-HPs in the form of V3 showing a left bundle branch block (LBBB) pattern, and two of the 13 patients also manifested V3 with a right bundle branch block (RBBB) pattern. During Re-HPs showing a LBBB pattern, the retrograde activation of the His bundle (H2) in response to V2 occurred via the left bundle branch (LBB), as indicated by inscription of retrograde H2 prior to RB2 in nine of 13 cases (average H2-RB2 = 9.4 msec), and simultaneous inception of retrograde H2 and RB2 in the remaining four. When V3 showed a RBBB pattern the retrograde RB2 preceded H2, suggesting H2 activation via the RBB. These findings suggest that circuit of Re-HP incorporates both bundle branches and the bundle of His. The H-RB recordings were also useful in distinguishing sustained Re-HPs from atrioventricular nodal reentry when in both instances the QRS complex showed a LBBB pattern.

THE OCCURRENCE OF a reentrant beat of ventricular origin (V3) during right ventricular premature stimulation has been studied frequently in recent years. This type of reentry is relatively common, and is observed in most, though not all, patients with normal intraventricular conduction. Based on initial observations, it was proposed that V3 phenomenon represents a form of macro reentry wherein both bundle branches and the bundle of His are essential parts of the reentrant circuit. This form of reentry has been useful in studying the effect of cardioactive drugs upon the reentrant phenomenon in the His-Purkinje system. In previous studies, only His (HB) bundle electrograms were used, and it was felt that additional recordings from the His-right bundle axis would significantly improve the understanding of this phenomenon. This unique model of reentry was studied to document the routes of impulse propagation during this form of reentry more precisely by using additional recordings from the His-right bundle axis in 13 patients who manifested the V3 phenomenon.

This study was designed to demonstrate the activation sequence of HB and bundle branches during different types of V3 phenomena and to discuss some aspects of this form of reentry.

Materials and Methods

All patients were studied in a non-sedated, postabsorptive state. The nature of the procedure was explained, and signed consent was obtained. Electrode catheters were percutaneously introduced into peripheral veins and fluoroscopically positioned in the region of the high right atrium and right ventricle for local recordings and electrical stimulation. Simultaneous recordings from the His and right bundle branch (RBB) were obtained either with a single quadrilateral catheter with interelectrode distance of 1 cm or by using two separate catheters. The most proximal clearly identifiable recordings of the specialized conduction system from the atrioventricular (AV) junction in any given patient was accepted as representative of HB activity. For RBB recording, the catheters were advanced further into the right ventricle until the most peripheral possible recording could be obtained. When a single quadripolar electrode catheter was used, more importance was attached to the location of peripheral (RB) rather than the proximal recording. The proximal and distal temporal relationship between the HB and RB potentials during sinuses beats was considered more relevant to this study, rather than the precise location from where a given recording was obtained. Three surface electrocardiographic leads, 1, 2 and V1, as well as all intracardiac electrogram tracings and time lines, were simultaneously displayed on a multichannel oscilloscope and recorded on a magnetic tape. The records were subsequently reproduced on photographic paper at a speed of 100–250 mm/sec. Electrical stimulation was performed via a digital stimulator, which delivered a rectangular impulse of 1.5 msec and 10 V amplitude through an isolation unit. The sequence and timing of various stimulation techniques (see below) were modified, depending on the findings in any given case. In general, predetermined
basic ventricular cycle lengths (S1S1 or V1V1) were scanned with progressively decreasing ventricular coupling (S1S2 or V1V2) intervals until the effective refractory period (ERP) of the ventricular muscle (VM) was reached. In addition to this conventional method, retrograde refractory period studies were also performed with simultaneous atrial and ventricular pacing during the basic drive beats. This latter design was used exclusively in patients who displayed constant or intermittent AV dissociation during ventricular pacing. In patients with intact ventriculoatrial (VA) conduction, simultaneous atrial and ventricular stimulation during the basic drive beats allowed arrival of programmed atrial stimuli at the AV junction sooner than would be otherwise possible, mostly by altering the atrial activation sequence. Following V1 and V2, programmed atrial stimuli (AP) were also introduced to document the site of retrograde block of V2 or V3, as well as to clarify certain aspects of the V3 phenomenon. All electrical equipment was carefully isolated, and no complications were encountered while conducting these studies.

**Definition of Terms**

A complete list of definitions of terms that are used in this type of clinical investigation have been published previously.7 Only pertinent additional definitions are listed here.

**Antegrade Studies.** The H-V and RB-V intervals were measured from the onset of respective deflections to the earliest detectable ventricular activity measured on the surface electrocardiogram or local ventricular electrograms.

**Retrograde Studies.** The retrograde (Ret) HB and RB activation for the basic drive beats (H1 and RB1) were not identifiable, but at closer V1V2 intervals both H2 and RB2 potentials emerged from V2, and could be clearly recognized from their morphology and physiologic behavior. The S2H2 (V2H2) and S2RB2 (V2RB2) intervals were measured from the stimulus artifact (and local ventricular electrograms) to the onset of the respective deflections. When V3 followed V2, the H2V3 and RBV3 intervals were measured from the onset of H2 (and RB2) to the earliest recorded ventricular activity during V3.

**Retrograde Effective Refractory Period (ERP) of the His-Purkinje System (HPS).** The longest V1V2 interval where V2 retrogradely blocked below the His bundle.

**ERP of Ventricular Muscle.** The longest S1S2 interval where S2 did not produce a ventricular response.

**Zone of Reentry His-Purkinje System (Re-HPS).** The range of V1V2 intervals where V2 produced V3 phenomenon. The outer and inner limits of Re-HPS zone were the longest and the shortest V1V2 intervals which resulted in V3.

**Results**

Essential clinical and electrophysiologic data are outlined in tables 1 and 2. All patients were in sinus rhythm and had intact AV conduction at the time of study. Intraventricular conduction was normal in 11 patients, while two patients showed incomplete right bundle branch block (RBBB) pattern on resting surface ECG. Patients with acute myocardial infarction, electrolyte imbalance, frequent ventricular premature beats and pre-existing complete bundle branch block patterns were excluded from this study. In this report, data from only those consecutive patients in whom good proximal and distal recordings from the His-right bundle (H-RB) axis were obtained will be presented. H-RB interval of 15 msec or longer (table 1) during sinus rhythm was considered satisfactory, and this was the case in these 13 patients (mean H-RB 16.6 ± 2.4 msec, range 15–20 msec). Although complete antegrade and retrograde conduction and refractory period studies were performed in all patients, only results pertinent to this study are presented in this report.

**Table 1. Clinical Data**

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<th>No.</th>
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<td>M</td>
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Abbreviations: SB = sinus bradycardia; PSVT = paroxysmal supraventricular tachycardia; IHBD = ischemic heart disease; MVP = mitral valve prolapse; PAF = paroxysmal atrial fibrillation; SSS = sick sinus syndrome; IRBBB = incomplete right bundle branch block.
Patterns of Emergence of Ret H2 and RB2 Potentials

The Ret H2 and RB2 deflections did not emerge from respective ventricular electrograms at the same V1V2 intervals in all cases. This was in part related to different durations of corresponding local ventricular electrograms. The longest V1V2 intervals at which both Ret H2 and RB2 could be clearly identified ranged from 230–500 msec (mean 344.6 ± 61.7) at basic ventricular cycle lengths of 500–1000 msec (mean 726.9 ± 142.3). The shortest Ret H2 and RB2 delays when both deflections were recognized are shown in table 2. At the earliest simultaneous recognition of the two potentials, the Ret H2 preceded RB2 in six cases (patients 1–6) (table 2), and the H2-RB2 interval averaged 10.8 ± 3.7 msec (range 5–15 msec). Antegrade H-RB intervals in the same six patients on the average measured 16.7 ± 2.6 msec (range 15–20 msec), and in only one (patient 1) did the Ret H2-RB2 intervals equal antegrade H-RB intervals (tables 1 and 2). In three patients (nos. 7–9), Ret RB2 preceded H2 by 15.0 msec in each case when both deflections cleared the local V2 electrograms. Antegrade H-RB intervals in all three patients also measured 15.0 msec. The remaining four patients (nos. 10–13) displayed simultaneous activation of Ret H2 and RB2, i.e., no detectable difference existed in V2H2 and V2 RB2 intervals, although a clear difference in H-RB interval was registered during sinus rhythm (mean H-RB 17.5 ± 2.9 msec, range 15–20 msec).

Retrograde H-RB Activation Sequence, V2H2 and V2RB2 Delays at Closer Ventricular Coupling Intervals

As further scanning of the basic ventricular cycle lengths were carried out with progressively shorter ventricular coupling intervals, the sequence of Ret H-RB activation reversed in the three patients (nos. 7–9) in whom initially Ret RB2 preceded H2 (table 2). In other cases the Ret H-RB activation sequence generally stayed unchanged at closer ventricular coupling intervals. However, three of these patients (nos. 1, 10 and 12) demonstrated changes in H-RB activation sequence in an unpredictable manner during the course of ventricular premature stimulation (not presented in the table). As a rule, linear increases in V2H2 and V2RB2 delays of 5–20 msec were noted as the V1V2 intervals were shortened by 10–20 msec intervals. However, at times sudden unexpected increases and decreases in V2H2 intervals of 40 msec or more were seen in three patients (nos. 1, 5 and 10). Such marked and unpredictable changes in V2H2 intervals occurred with and without changes in Ret H-RB activation sequences (fig. 1, panels A and B). The longest V2H2 and V2RB2 intervals (which did not necessarily occur at shortest V1V2) in individual patients are outlined in table 2. In all patients, and at all times, V2H2 and V2RB2 delays occurred in a parallel fashion, and no conduction delay or block were noted between H2 and RB2 deflections. Similarly, no split potentials from H or RB recordings
were noted in this series of patients, either during antegrade or Ret conduction.

Reentry His-Purkinje System

A reentrant beat of ventricular origin (V3) occurred after the induced V2 in all 13 patients (table 2). Certain features of V3 phenomenon (Re-HPS) have been previously described, and are not presented here.1,2 The ranges of V1V2 intervals and V2H2 (also V2RB2) delays which accompanied V3 are summarized in table 2. The occurrence of V3 in all cases was sudden, and could not be anticipated from the degree of V2H2 delays in any patient for any given cycle length. Generally, the V2H2 and V2RB2 intervals showed a gradual increase before and with the appearance of V3. However, sudden increases in V2H2 delays were noted in three patients, and in two of the three, such increases in V2H2 intervals accompanied V3. A Ret block of V2 below the HB recording site, i.e., no Ret H2, was encountered in eight of the 13 patients, and V3 first appeared after resumption of conduction to the HB at shorter V1V2 intervals in five of eight patients (fig. 1, panels C and D), whereas in the remaining three V3 was noted before Ret block of V2 in the HPS. As previously reported, no instance in this study was V3 noted when V2 did not depolarize the bundle of His (no H2).1,2

At the longest V1V2 intervals which produced Re-HPS, the QRS morphology and axis orientation of V3
FIGURE 2. The overall format is the same as figure 1. With simultaneous atrial (open arrow) and ventricular (closed arrow) pacing during the basic cycle length (BCL), premature ventricular stimuli are introduced (panels A–D). Site of right ventricular stimulation is apical in panels A and B and inflow in panels C and D. Reference sinus beats are displayed in panels A and C, and the H-RB interval measures 15 msec. The V2 retrogradely blocks in the HPS (panels A and C, i.e., no Ret H2 or A2), whereas at closer coupling intervals V2 conducts to the His bundle (panel B and D) and is followed by V3 (Re-HPS). The QRS morphology of V3, i.e., left bundle branch block pattern and right bundle branch block pattern in panels B and D, is quite obvious. Note that the Ret H2 precedes RB2 in panel B, while Ret RB2 precedes H2 in panel D. Pertinent intervals are labeled. CS = coronary sinus electrogram.

resembled V2, i.e., a pattern of left bundle branch block (LBBB) and left axis deviation (LAD) when right ventricular apex was the site of stimulation. With right ventricular outflow pacing (two patients), V3 showed a LBBB and LAD in one and LBBB and right axis deviation (RAD) in the other. At shorter V1V2 intervals, changes in QRS complexes of both V2 and V3 were sometimes noted. The most apparent change in V2 was an increase in the QRS duration compared with V1. The more obvious change in V3 was a decrease in QRS duration and a change in its axis toward normal as the inner limits of the Re-HPS zone were approached. The changes in V3 could be clearly appreciated in only those cases where the zone of Re-HPS was relatively long (40 msec or more) (patients 5, 8, 9 and 11).

When Re-HPS showed a LBBB pattern regardless of the axis orientation the Ret H2 preceded RB2 in nine of the 13 (fig. 2, panels A and B) by an average of 9.4 msec (range 5–15 msec), while the two potentials occurred simultaneously (no detectable H2-RB2 interval) in four of 13 patients in association with V3 phenomenon (fig. 1, panel D). Two patients in this series (nos. 1 and 8) also displayed V3 showing a RBBB pattern, and in one of these (no. 8), V3 showing both left and RBBB pattern were noted during pacing from the same site. In the other patient (no. 1), V3 with a LBBB pattern occurred during right ventricular apical pacing (fig. 2, panels A and B) while V3 showing a RBBB pattern was noted during inflow pacing (fig. 2, panels C and D). The sequence of H-RB activation preceding V3 displaying a RBBB pattern in both cases was the reverse (i.e., RB2 preceded H2) of what was seen when V3 had the more commonly observed LBBB pattern (compare panels B and D, fig. 2). At no time did Ret RB2 precede H2 in association with V3 showing a LBBB pattern or Ret H2 precede RB2 with V3 displaying a RBBB pattern in this series of patients.

A reciprocal relationship existed between V2-H2
shortening in H2V3 intervals, such that V2V3 intervals increased as the V1V2 intervals were decreased (fig. 3). In eight of 13 patients, therefore, at the outer limits of Re-HPS zone the V1V2 exceeded the V2V3 intervals, whereas at the inner limits the V1V2 intervals frequently measured less than V2V3 intervals. In four of the patients, the Re-HPS zone was too narrow to draw such conclusions. Only one patient (no. 2) demonstrated V2V3 < V1V2 through the full zone of Re-HPS.

While no conduction delay was demonstrable between the stimulus and local ventricular response at S1S2 intervals when V3 first appeared, some degree of local latency (5–15 msec) was common at the inner limits of Re-HPS zone which did not appear to have any influence upon the occurrence of V3 phenomenon. In two patients, however, the V3 disappeared at such close coupling intervals despite longer V2H2 delays, although the phenomenon was clearly demonstrable at longer S1S2 intervals.

**Termination of Re-HPS**

In 11 of the 13 patients the Re-HPS was limited to only V3, which retrogradely blocked in the HPS, i.e., there was no Ret H3 or RB3 recorded. In two patients (nos. 5 and 7), however, the V3 did conduct to the bundle of His (H3) and was followed by another beat (V4) showing the same general morphologic features (fig. 4, panel A). The V4 in turn blocked retrogradely below the His bundle (H4), terminating the reentrant process. Only at one occasion, in patient 7, was V4 followed by H4 and V5 was noted at that moment. The sequence of Ret H and RB activation during V4 and V5 in these patients remained the same as with V3. In another patient (no. 2, fig. 4, panel B), V4 showing a LBBB pattern was seen due to AV nodal reentry. The latter origin was suggested by a change in temporal relationship of H-RB activation preceding V4 compared to V3.

**Other Types of Ventricular Responses**

Two patients demonstrated the occurrence of ventricular response showing RBBB pattern that were not preceded by H2 or RB2 activation. None of the patients in this series demonstrated V3, V4 or V5 showing a LBBB pattern that was not preceded by Ret H deflections. Catheter-induced ventricular extra beats with a LBBB pattern were observed on occasion, were randomly distributed and were easy to identify. The term Re-HPS used in this text excludes types of ventricular responses just described.

**Discussion**

These data strongly suggest that Re-HPS does indeed have a macro reentrant basis wherein both bundle branches and the bundle of His are essential links in the reentry circuit. It is important to carefully examine the possible location of the recordings obtained from the H-RB axis in this series of cases. Considering that the intervals between the proximal and distal recordings along the H-RB axis ranged between 15–20
The sequence of activation of H-RB in the remaining cases (proximal recordings preceded distal recordings during both antegrade and Ret conduction) is compatible with locations of the two recordings shown in panel C and panel D. However, since the conduction time from the proximal deflection to the earliest ventricular activation ranged from 40–50 msec, it is unlikely that the proximal recordings originated from RBB (panel D), which is also suggested by the observation that Ret H2-RB2 measured less than antegrade H-RB in most of these patients. If the two recordings were from proximal and distal RBB, then the H2-RB2 and H-RB should measure the same, regardless of whether the impulse approached the proximal recording from LBB or HB. In either case the two deflections will be antegrade in the LBB.

msec, the four main possible locations are schematically depicted in figure 5, panels A-D.

The first possibility (panel A) that the H-RB recordings were in fact proximal and distal His bundle recordings can be summarily dismissed, since the distal His potential should precede the proximal His potential at all times during Ret conduction regardless of whether Ret HB activation occurred via the left or right bundle branch, a finding not noted in any of the 13 patients. The results obtained in four patients (nos. 10–13), simultaneous activation of Ret H2 and RB2, can best be explained by locations of proximal and distal recordings shown in panel B. In two of these patients (nos. 10 and 12) a change in Ret activation sequence (RB2 preceding H2) on occasion further attests to the fact that at other times, Ret H2 activation occurred via the LBB.

**Figure 4.** These tracings were obtained from two patients. Panel A, taken from patient 7, shows induction of sustained Re-HPS (V3 and V4) at a basic cycle length (BCL) of 1000 msec and V1V2 interval of 320 msec. Note that the H2 and H3 precede RB2 and RB3 deflections respectively by 5 msec, which is significantly shorter than H-RB of 15 msec during antegrade conduction (not labeled but marked by vertical line). Panel B (patient 11) shows induced V3, which is preceded by simultaneous activation of retrograde H2 and RB2, a sequence quite different than the one noted during antegrade (ant) conduction (ant H-RB = 20 msec, not labeled, marked by perpendicular in next to last beat). The V3 (Re-HPS) is followed by another beat showing a left bundle branch block pattern, and is not considered to have the same origin as V3. The H-RB preceding this beat equals the sinus beats (20 msec), which suggests its origin to be above the HB recording site. Since the H is preceded by retrograde atrial activation sequence, AV nodal reentry is the most likely mechanism although intraatrial reentry cannot be totally excluded. Sp and/or Ap = programmed atrial extra stimulus.

*Despite relatively large atrial deflection on the distal recording, a clear proximal and distal temporal relationship between the so labeled H and RB potentials is obvious and the RB-V interval measures 25 msec.*
recordings obtained in this series of patients do, in fact, represent activation of the HB and RBB, respectively. Figure 5 is only a schematic representation and does not necessarily indicate the precise location of the recordings in any given patient. Although the H-V and RB-V intervals in the present series are comparable to previously published data, it is the activation sequence of the bundle branches and the HB during antegrade and Ret conduction that is pertinent here, rather than the exact H-V or RB-V intervals in any given patient. 10, 11

Macro Re-HPS vs Micro or Local Reentry

The results show that the activation of Ret H2 occurred via the LBB whenever a V3, V4 or V5 showing a LBBB pattern were inscribed, whereas Ret H2 activation occurred via the RBB in association with V3 displaying a RBBB pattern. The breakthrough of ventricular activation wave front from right and left ventricles, along with appropriate activation sequence of both bundle branches and the bundle of His, cannot be considered a fortuitous combination. Such a coincidence is particularly unlikely if one considers the fact that Ret RB2 activation never preceded H2 with inscription of V3 showing a LBBB pattern and Ret H2 inscription did not occur before RB2 in conjunction with V3 displaying the RBBB pattern. It was further observed that when similar V2H2 delays were available for comparison, V3 showing a LBBB pattern occurred only when H2 activation occurred via LBB (compare panels A and D, fig. 1).

Reasons other than the ones listed above which also support the idea that Re-HPS incorporates both the RBB and LBB and the bundle of His, and is not a local phenomenon (micro circuits near the site of stimulation), are as follows:

1) An inverse relationship existed between V2H2 and V2RB2) and H2V3 (and RB2-V3) intervals as well as V1V2 and V2V3 intervals when the zone of Re-HPS was sufficiently wide (fig. 3).

2) No evidence of local conduction delay was noted.

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**Figure 5.** The enclosed clear and shaded areas are representative of the overall range of proximal and distal recordings. The four main possible combinations of proximal and distal recordings obtained from the H-RB axis are depicted. Panel A shows that the two potentials could represent electrical activity from the proximal and distal His bundle. Recordings from middle portion of the His bundle and proximal right bundle branch (RBB) are shown in panel B, whereas panel C displays that the two recordings might reflect distal His and more peripheral RBB activity. Panel D shows the last possibility, that the two recordings obtained were from proximal and distal RBB. The figure is a schematic representation and is not reflective of precise anatomic location of recordings in any given case. AVN = atrioventricular node; HB = His bundle; LB = left bundle branch; RB = right bundle branch.
at the outer limits of Re-HPS zone and at closer S1S2 intervals V3 disappeared in two patients, despite presence of local conduction delay.

3) Neither type of Re-HPS occurred whenever V2 retrogradely blocked below the bundle of His. Other ventricular responses noted which were not preceded by an H deflection are compatible with a micro reentrant process.

It may still be argued that close similarity of QRS complex morphology and axis between the paced V2 and V3 phenomenon might be indicative of a local (micro reentrant) origin of V3.14 For the reasons listed below the axis orientation of V3 does not appear to be of great value in determining the reentrant circuit.

1) Incomplete recovery of excitability of the RBB-Purkinje-muscle system existed at the outer limits of Re-HPS and in some way contributed to overall axis of V3 since changes in QRS axis of V3 were frequently noted in association with shortening of RB2-V3 intervals at the inner limits of Re-HPS. Some element of intramuscle conduction delay during V3 at the longer V1V2 intervals is also indicated by the QRS duration of V3, which exceeded V2 at the beginning and showed a decrease at the inner limits of Re zone; QRS duration of V3 decreased when V2V3 intervals exceeded V1V2 interval.

2) More importantly, the areas of initial excitation during V2 might also be the areas of earlier recovery; the spread of activation front during V3 could be quite similar to V2.

Re-HPS vs Reentry within the Bundle of His

Intra-His reentry, which cannot be entirely excluded in any given case at a given moment, is an unlikely mechanism of Re-HPS for the following reasons:

1) No evidence of intra-His conduction delay in the form of wide or split His potentials8 or conduction delay between the H-RB deflections was observed during antegrade or retrograde refractory period studies.

2) The HB, which is quite remote from stimulation site, will be an unlikely area of conduction delay unless local conduction abnormalities preexist.

3) The route of Ret activation of the HB (via RBB or LBB) would not be expected to influence intra-His bundle reentrant process. However, in this study, a consistent association existed between a certain type of V3 and a certain sequence of bundle branch activation.

Re-HPS vs AV Nodal Reentry

When Re-HPS occurs as a single beat (V3), its origin is easily distinguished from AV nodal reentry, since the HB activation must occur twice before registration of AV nodal reentrant ventricular echo. However, when Re-HPS appears to be sustained beyond V3, to V4, V5, etc., it is possible that the latter ventricular responses could originate from reentry within the AV node.2 Although generally ventricular echo beats due to AV nodal reentry are preceded by prolonged AV nodal conduction, such beats can occur without demonstrable Ret AV nodal conduction delays.2, 7, 13-16 The true origin of V4 and subsequent ventricular responses may be difficult or impossible to delineate unless recordings from both the HB and RBB are obtained, as exemplified by findings in patient 11 (fig. 4, panel B).

Spontaneous Termination

The Re-HPS whether limited to one beat (V3) or more (V4, V5) spontaneously terminated with a Ret block below the HB, an expected finding if the suggested mechanism is true. Although not seen here, spontaneous termination of Re-HPS might sometimes occur in the antegrade limb of the circuit (personal observation) — H3 but no V4. In none of these patients did Re-HPS terminate due to conduction delay between the H-RB recording sites.

Clinical Implications

In the overwhelming majority of cases with chronic recurrent ventricular tachycardia studied by HB electrogram recordings, Re-HPS does not appear to be the mechanism for arrhythmia.17-20 However, if Re-HPS is sustained, the pattern will closely simulate the so-called supraventricular tachycardia with aberrant conduction, since in both instances a HB deflection will precede the QRS complex by H-V and/or RB-V intervals ≥ sinus beats. During such a tachycardia, if the atrial and HB activation are dissociated, then Re-HPS is possible. On the other hand, a 1:1 VA (or AV) relationship during such an arrhythmia may not necessarily mean the arrhythmia is supraventricular in origin at any given moment. The distinction between Re-HPS and supraventricular tachycardia with aberrant conduction may be difficult at times, particularly in patients with Wolff-Parkinson-White syndrome (where ventricular muscle also is an essential part of the Re circuit as during Re-HPS). If a question arises regarding the circuit of reentry, recordings from both the HB and RBB may be helpful in distinguishing sustained Re-HPS and supraventricular tachycardia with aberrant conduction. In this regard, more proximal recordings from the H-RB axis (panel B, fig. 5) will permit a clearer distinction between the two impulses approaching the HB from different directions, i.e., one via the LBB vs the other via the AV node (panels B, figs. 4 and 5). If the recordings were obtained from more distal locations in the HB (panel C, fig. 5), the Ret H2-RB2 intervals may equal antegrade H-RB (fig. 2), and such a distinction will be more difficult.

This study provides strong support to the previous hypothesis of the nature and Re circuit of V3 phenomenon, which lends itself to easy analysis for evaluation of drug effect on reentrant phenomenon in the HPS.1 Confirmation of the precise routes of Re-HPS is important, since it represents the only model of its type in the intact human heart.

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