Demonstration of Re-entry within the His-Purkinje System in Man

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SUMMARY

Re-entry within the His-Purkinje system (HPS) was consistently observed in 15/24 consecutive patients in whom retrograde refractory period studies were performed using His bundle electrograms and the ventricular extrastimulus method. Within a narrow range of ventricular coupling intervals (V1V2), V2 retrogradely conducted to the bundle of His (H2) with significant infra-His bundle conduction delay (V2H2 interval). At critical V2H2 delays another beat of ventricular origin (V3) followed V2 and was associated with H2V2 intervals greater than the H-V intervals of sinus beat. It is postulated that V2 retrogradely blocked within the right bundle branch and activated the bundle of His via the left bundle branch after which antegrade conduction occurred along the right bundle branch producing the V3 response. In support of re-entry within the HPS are the following: 1) V3 occurred in a narrow range of V1V2 intervals and critical V2H2 delays, 2) V3 did not occur when V2 retrogradely blocked below the bundle of His, 3) V3 was independent of retrograde A-V nodal delay, 4) V3 rarely occurred in patients with pre-existing complete right bundle branch block pattern. These results reasonably exclude local re-entry near the site of stimulation.

Additional Indexing Words:
Refractory periods Retrograde conduction

The suggested mechanisms of ventricular arrhythmias include ectopic impulse formation and re-entry within the ventricular specialized conduction system.1-15 In the animal heart, re-entry within the His-Purkinje system (HPS) has been frequently demonstrated in the setting of depressed conduction brought about by a variety of factors, most notably a high extracellular concentration of potassium10-12 and acute myocardial ischemia.5, 13, 15 This report deals with observations made in a group of 24 patients in whom unsustained re-entry within the HPS was observed during the performance of ventricular refractory period studies. In 15 patients re-entry was consistently observed within a relatively narrow range of ventricular coupling intervals and appeared to be entirely dependent upon retrograde conduction delay within the HPS and totally independent of retrograde A-V nodal conduction time. In this study, re-entry within the HPS and the A-V node could be easily distinguished even when they coexisted.

Methods

Right heart catheterization was performed in the postabsorptive, nonsedated state in a total of 24 patients. The experimental nature of the procedure was explained and signed consent was obtained. Using local anesthesia a quadripolar electrode catheter was percutaneously introduced into an antecubital vein and fluoroscopically positioned against the lateral wall of the right atrium near its junction with the superior vena cava. The proximal pair of electrodes was used to record a high right atrial electrogram and the distal pair to electrically stimulate the atrium. Similarly, a tripolar electrode catheter positioned in the region of the tricuspid valve was used to record bundle of His activity as previously described.16 Ventricular stimulation was performed with a bipolar electrode catheter fluoroscopically positioned at the apex or the outflow of the right ventricle.

Intracardiac electrograms (recorded at 40–500 Hz), standard ECG leads I, II, III and V1 (recorded at 0.1–200 Hz) and time lines generated at 10, 100, 1000 msec were displayed on a multichannel oscilloscope and recorded on magnetic tape. The records were subsequently replayed and recorded on photographic paper at a speed of 150 mm/sec. Using a programmed digital stimulator and the extrastimulus method, refractory periods were determined during both antegrade and retrograde conduction at the same basic cycle length (A1A1 or V1V1). Following every eighth beat of the basic drive, a premature beat (A2 or V2) was introduced at progressively decreasing coupling intervals (A1A2 or V1V2) up to the point of atrial or ventricular refractoriness.

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RE-ENTRY WITHIN THE HPS

During atrial and ventricular stimulation electrical impulses of 1.5 msec duration and minimum milliamperage permitting consistent atrial or ventricular capture were delivered through an isolation unit. All equipment was carefully grounded. The procedure was well tolerated by all patients and no untoward effects occurred. Sα, Vα, Hα, and Aα represent stimulus artifact, ventricular, His bundle, and atrial electrograms of the basic ventricular drive beats. Sβ, Vβ, Hβ, and Aβ represent stimulus artifact, ventricular, His bundle, and atrial electrograms of the premature ventricular beats.

The onset of induced ventricular depolarization was taken from the corresponding stimulus artifact and therefore the S-H and S-A intervals were measured from the stimulus artifact to the peak or nadir of His bundle electrogram and to the beginning of low atrial electrogram, respectively. When Sβ resulted in more than one ventricular beat, the H-V interval of subsequent beats was measured from the peak or nadir of the preceding His bundle electrogram to the earliest detectable ventricular activity as noted on the ECG or the His bundle electrogram recording.

Results

Although both antegrade and retrograde period studies were performed in all patients except one (pt. 24), only the results obtained during retrograde refractory period studies will be presented. The essential clinical and electrophysiologic data for all patients are summarized in table 1. None of the patients had electrocardiographic evidence of spontaneous ventricular premature beats or Wolff-Parkinson-White syndrome.

Patients 1–19 had 1:1 retrograde conduction during the basic ventricular drive. Patients 20–23 consistently demonstrated A-V dissociation at all ventricular drive rates, and patient 24 had atrial fibrillation.

In all patients who demonstrated 1:1 retrograde conduction during the basic ventricular drive a progressive increase in ventriculo-atrial conduction time (Sβ Aβ interval) occurred as the premature ventricular beat (Vβ) was introduced at progressively shorter S1 S2 intervals. For relatively late ventricular premature beats (Vβ), the exact site of retrograde conduction delay (within the His-Purkinje system or the A-V node) could not be determined since the bundle of His electrogram was obscured within the ventricular electrogram for both the basic drive and premature beats. However, in five patients the retrograde His bundle electrogram was recognizable during both the basic ventricular drive and premature beats and it could be determined that, for relatively late premature ventricular beats, VA conduction delay was localized to the A-V node. At shorter coupling intervals, the bundle of His electrogram emerged from the ventricular electrogram of the premature beat in all patients. An increase in the S2 H2 interval documented conduction delay below the recording site of the bundle of His and also permitted retrograde A-V nodal delay (H2 A2 interval) to be more accurately assessed.

The exact site of infra-His bundle delay cannot be determined precisely in the intact human heart, and it is possible that the premature ventricular impulses may have encountered conduction delay within the ventricular myocardium, and/or muscle-Purkinje junction and/or Purkinje bundle branch-His system. When retrograde A-V nodal delay occurred it was dependent upon the relationship of the occurrence of S2 to previous A-V nodal depolarization, i.e., S1 H2 or H1 H2 interval. Within a certain range of S1 S2 intervals (350–210 msec), a critical degree of retrograde His-Purkinje conduction delay occurred (S2 H2 interval, range 120–325 msec) and resulted in spontaneous ventricular beats (V3) in 15/24 patients (table 1). The QRS morphology and axis orientation of V3 closely resembled V2 although its QRS duration was generally greater than V2. Once this critical degree of retrograde His-Purkinje conduction delay was achieved, V3 consistently occurred at shorter S1 S2 intervals up to the point of ventricular muscle refractoriness. If the S2 impulse was retrogradely blocked within the Purkinje bundle branch system, i.e., S2 was not followed by an H2 response, then the V3 response did not occur. However, at shorter S1 S2 intervals, resumption of conduction retrogradely to the His bundle (retrograde gap phenomenon),17 as well as resumption of the V3 phenomenon occurred. This was observed in patients 4, 12, 13, and 14.

Such zones of S1 S2 intervals, where S2 blocks retrogradely within the HPS, are commonly seen during retrograde refractory period studies. Figure 1 (taken from patient 4) is typical of these findings. The basic ventricular drive is constant at 700 msec in all panels, and retrograde conduction time (S1 A1) measures 150 msec. At a coupling interval of 330 msec (panel A), S1 is associated with a retrograde conduction time (S2 A2 interval) of 225 msec. Retrograde His-Purkinje (S2 H2) and A-V nodal conduction times (H2 A2) measure 175 and 50 msec, respectively. At a coupling interval of 320 msec (panel B), retrograde conduction time to the atrium (S2 A2) increases to 265 msec. The 40 msec increase in the S2 A2 interval in panel B compared to panel A results entirely from increase in infra-His bundle conduction time (S2 H2:215 msec) since A-V nodal conduction time remains constant at 50 msec. A spontaneous ventricular beat (V3) follows V2 and is preceded by an H-V interval of 85 msec (H2 V3 interval). Note the similarity of QRS morphology and axis orientation between V2 and V3, a fact which suggests that the spread of excitation during V3 occurs from the right ventricle also and has the same general direction as that of V2. When S1 S2 is further decreased to 300 msec (panel C), S2

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retrogradely blocks below the His bundle electrogram recording site, and V₃ does not occur. A sinus escape beat follows, the H-V interval of which measures 40 msec. In panel D, V-A conduction and the V₃ phenomenon resume at a closer coupling interval of 250 msec. Note the further delay in His-Purkinje conduction (S₂, H₂, 255 msec) whereas the A-V nodal conduction time is unchanged (H₂ A₂, 50 msec). When V₂ consistently continued to block retrogradely within the HPS up to the point of ventricular muscle refractoriness, V₂ was not followed by a V₃ response.

This occurred in patients 1, 4, 6-9, 12 and 17 and explains the clear range of V₁ V₂ intervals between the zone of re-entry and the ventricular effective refractory period in these patients as presented in table 1. Since the occurrence of V₃ was dependent upon retrograde infra-His bundle conduction delay and independent of A-V nodal conduction time, the V₂ phenomenon will henceforth be referred to as re-entry within the HPS.

Re-entry within the HPS was also observed in patients who demonstrated A-V dissociation during ventricular pacing, an example of which is shown in figure 2 (taken from patient 22). The re-entry occurred only at a longer cycle length of 850 msec (panel C), because sufficient His-Purkinje delay could not be achieved at a shorter ventricular cycle length of 700 msec (panel B). Both panels B and C show the longest S₂ H₂ intervals achieved during the respective basic ventricular drives. Note the QRS morphology and axis orientation of V₃ (LBBB pattern) compared to V₁ and V₂ when the site of stimulation was the right ventricular outflow tract as in this patient.

Figure 3 is an example of re-entry within the HPS in a patient with atrial fibrillation (patient 24).

In four patients in whom relatively late premature ventricular beats occurred, sufficient A-V nodal delay occurred to result in A-V nodal re-entry. At closer coupling intervals when S₂ blocked in the A-V node, A-V nodal re-entry was abolished. In other instances A-V nodal re-entry continued to occur while His-Purkinje conduction time progressively lengthened as the S₁ S₂ intervals were decreased. At a certain range of coupling intervals, both A-V nodal re-entry and His-Purkinje re-entry occurred from a single ventricular premature beat. The QRS morphology of the A-V nodal re-entrant beat was always aberrant when associated with a preceding V₃ response. Figure 4 taken from patient 5 is a representative example. The A-V nodal re-entrant beats in each panel are marked by an asterisk.

Re-entry within the HPS was generally manifest as a single re-entrant beat (V₃), and in only two patients (11 and 13) was re-entry sustained for up to three consecutive beats. These results are shown in fig. 5 (pt.11).
Discussion

Re-entry can occur in most cardiac tissues in which there exists slow conduction and unidirectional block.9-12, 18-20 There is overwhelming evidence that re-entry is the underlying mechanism responsible for some forms of cardiac arrhythmias, most notably A-V nodal re-entrant supraventricular tachycardia.21-26 Sufficient data is available to show that re-entry occurs in isolated canine Purkinje fiber in the background of depressed conduction. It has been suggested that re-entry within the ventricular specialized conduction system may occur over a small area (micro re-entry),30, 31 or over a larger circuit involving the bundle branches.32, 33 Although it is believed that some forms of ventricular arrhythmias in man may have a re-entrant basis, the role of re-entry in man is far from clear.

The results of this study provide evidence that re-entry within the HPS can be consistently demonstrated in most human hearts when a critical degree of conduction delay is present within the HPS. The origin of re-entry in this group of patients was suggested by the following:

1) Re-entry (V₃) occurred within a narrow range of S₁ S₂ intervals and was not observed at longer S₁ S₂ intervals or when the ventricular muscle was refractory.
2) The occurrence of V₃ appeared to be entirely dependent upon the attainment of a requisite retrograde conduction delay below the His bundle electrogram recording site.
3) Re-entry did not occur when the S₂ impulse was retrogradely blocked below the recording site of the His bundle electrogram.
4) The occurrence of V₃ bore no relationship to retrograde A-V nodal delay and in fact persisted even when the S₂ impulse retrogradely blocked in the A-V node (fig. 4).
5) Re-entry could also be demonstrated in patients in whom no V-A conduction occurred (fig. 2).

Although the exact circuit of re-entry in these cases is not known for certain, the available evidence indicates that the re-entrant pathway involves both bundle branches and the bundle of His. This is suggested by the fact that V₃ responses were almost always (except one patient, no. 7) preceded by H₂ V₃ intervals of greater than normal duration, i.e., longer than the H-V intervals of sinus beats (table 1). On the assumption that there is a close temporal relationship between the retrograde depolarization of the His bundle and the antegrade penetration of the right bundle by the S₂ impulse, the H₂ V₃ intervals fairly accurately reflect the antegrade conduction time of S₂ within the HPS.

Figure 6 diagramatically illustrates possible routes of re-entry involving both bundle branches. Panel A depicts the pathway of retrograde conduction for impulses of both the basic ventricular drive beats (S₁) and relatively late premature beat (S₂). Due to the lack of any significant retrograde delay within the HPS as well as the proximity of the stimulating site to the right bundle branch, both of these impulses preferentially depolarize the His bundle via the right bundle branch. The same impulse traversing the left bundle branch finds the bundle of His refractory. At closer coupling intervals (panel B), S₂ may retrogradely block within the right bundle branch or ventricular muscle-Purkinje fiber junction because it is still refractory from the preceding basic drive beat (S₁). Depolarization of the ventricular muscle proceeds and the arrival of the S₂ impulse at the left bundle branch permits retrograde conduction to the His bundle, after which the impulse is antegradeley conducted within the right bundle branch. If sufficient time has elapsed to permit recovery of both the right bundle branch and ventricular muscle, re-excitation will occur and the QRS morphology of the V₃ complex will be similar to that of V₁ and V₂ (figs. 1, 3, and 4). Incomplete recovery of the right bundle branch is indicated by the fact that V₃ complexes were almost always associated with H₂ V₃ intervals which were greater than those of normal sinus beats. Panel C depicts re-excitation of the ventricles due to longitudinal dissociation within the right bundle branch. It also shows A-V nodal re-entry as an additional mechanism of re-excitation of the ventricles following delayed retrograde A-V nodal conduction of V₃. The latter mechanism of re-excitation of the ventricles can be added to all panels. The events shown in panel D are theoretically possible if the refractoriness of the left bundle branch markedly exceeds that of the
Re-entry within the HPS in A-V dissociation. Panel A simply shows sinus rhythm and prolonged A-V nodal conduction time (A-H interval = 230 msec) of sinus beats (1st A-V nodal block). The H-V interval measures 45 msec. During right ventricular (outflow) pacing at a cycle length of 700 msec (panel B), the atria are dissociated, (high to low atrial activation sequence). At a coupling interval of 250 msec, V2 conducts retrogradely with an S2 H2 interval of 235 msec, which was the longest S2 H2 interval achieved at this cycle length. In panel C the basic ventricular cycle length is increased to 850 msec and the atria are still dissociated. V3 now conducts with longer His-Purkinje conduction time (S3 H2; 265 msec) and a spontaneous ventricular beat (V3) follows, which is preceded by an H-V interval (H2 V2) of 60 msec. The QRS morphology of V3 is compatible with ventricular activation occurring through right bundle branch system (left bundle branch block pattern). The beat following V3 (panel C) is a paced ventricular beat.
Re-entry within the HPS during atrial fibrillation. Panel A shows atrial fibrillation where the H-V interval of conducted beats measures 50 msec. During right ventricular pacing at a cycle length of 800 msec, the retrograde His deflection (H₂) is recognizable during basic drive, and S₁, H₁ interval is 55 msec. At an S₁, S₂ interval of 360 msec panel B, the His bundle deflection moves out of the ventricular electrogram and S₂ H₂ interval measures 175 msec. No re-entry occurred at this degree of His-Purkinje delay. At a closer coupling interval (panel C) the retrograde His-Purkinje conduction time measures 220 msec and V₂ is followed by a re-entrant beat (V₃). The H₂, V₃ interval measures 90 msec. Both HRA and HBE were recorded at the same filtering frequency settings, i.e., 40-500 Hz. The beat following V₃ (panel C) is a paced ventricular beat.

Figure 3

The premature ventricular impulse (S₂) may therefore block in the left bundle, propagate along the right bundle and re-excite the left bundle branch system. The mechanism depicted in panels D provides an explanation for the infrequent finding of V₃ responses showing a RBBB pattern. Likewise, panel C provides an explanation for the infrequent occurrence of V₃ responses showing a LBBB pattern and short H₂ V₃ intervals.

One cannot entirely exclude the possibility that re-excitation of the ventricles resulted from re-entry occurring in local circuits established at the site of premature ventricular stimulation and that the retrograde His deflection (H₂) bears a merely fortuitous relationship to V₃. However, the consistency with which retrograde His deflection preceded single or multiple re-entrant QRS complexes makes local re-entry a less likely possibility to explain our results. In addition, as shown in figure 1, re-excitation of the ventricles disappeared at short S₁ S₂ intervals when the S₂ impulses retrogradely blocked in the Purkinje bundle branch system and reappeared at shorter S₁ S₂ intervals when retrograde conduction to the bundle of His resumed. Furthermore, the similarity of the QRS complexes for V₃ responses in all patients in whom V₃ showed LBBB pattern, whether ventricular stimula-
Sustained re-entry within the HPS. Panel A shows a reference sinus beat. In panel B a premature ventricular beat is coupled to the basic ventricular drive beat at an S1, S2 interval of 220 msec. S2 conducts retrogradely with a S1, H2 interval of 260 msec and a sustained re-entry is initiated. The H-V intervals of the three subsequent beats (i.e., V3, V4, V5) measure 60, 70, and 50 msec respectively. A2 on the HBE coincides with the onset of H2 and it is very unlikely that V4 is an A-V nodal re-entrant beat. The cycle of sustained re-entry within the HPS is followed by an A-V nodal re-entrant beat (asterisk). The fact that the first beat in panel B does not retrogradely capture the atria should have no influence on the subsequent events since it is unlikely that the sinus beat could have antegrade depolarized the HPS. Similarity of V3 to V4 suggests their similar origin; however A-V nodal re-entry and aberrant conduction during V4 cannot be entirely excluded.

In the fifth patient with complete RBBB pattern (last beat in panels B and C of fig. 7), re-entry occurred on only two occasions and is shown in figure 7. The QRS morphology (i.e., RBBB pattern) is compatible with the mechanism shown in panel D of figure 6 or reciprocation within the left bundle branch. If the most likely mechanism for this form of re-entry is that which is presented in panel B of figure 6, then it is tempting to postulate that most patients with a complete RBBB pattern will not demonstrate the phenomenon following premature stimulation of the right ventricle since the right bundle branch would be refractory to antegrade conduction of the re-entrant impulse.

Re-entry as demonstrated in this study was a self-limiting phenomenon. Most often only a single re-entrant ventricular beat occurred but on occasion

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Re-entry within the HPS and the A-V node. The basic ventricular cycle length is 700 msec, panel A to D. In panel A, S1 conducts retrogradely with an S1, A2 interval of 270 msec and is followed by a ventricular beat with a normal QRS complex. The ventricular activation is preceded by a normal H-V interval of 40 msec and a low to high sequence of atrial activation which suggest A-V nodal re-entry as the underlying mechanism responsible for the origin of this beat. At a closer coupling interval (panel B), S2 blocks in the A-V node (the retrograde His bundle potential emerges from the ventricular electrogram and is not followed by an atrial electrogram) and A-V node re-entry is abolished. At still closer coupling interval of 270 msec (panel C), S2 continues to block in the A-V node and is followed by another beat of ventricular origin (V3).

Panels B and C also show reference sinus escape beats with a high to low atrial activation sequence. In the bottom panel (D) S3 impulse resumes conduction to the atria and encounters both His-Purkinje and A-V nodal delay and results in both His-Purkinje and A-V nodal re-entry. Note the similarity of V3 in panel C and D; and in both panels V3 is preceded by H3. V3 interval of 55 msec. A-V nodal re-entrant beats are indicated by asterisks both in panel A and D. In panel D the QRS complex shows a left bundle branch block pattern and is preceded by an H-V interval of 55. This type of aberrancy can be explained if one assumes that V3 either penetrated the left bundle branch system retrogradely and blocked in the left bundle branch system which was found refractory by the A-V nodal re-entrant impulse, or simply that the left bundle branch system had a longer recovery time.

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RE-ENTRY WITHIN THE HPS

Observed in clinical cases of spontaneous ventricular extrasystoles or tachycardia. In these situations, the bundle of His is also retrogradely activated but its inscription immediately precedes or occurs some time after the onset of ventricular activation so that the H-V interval is either nonexistent or significantly less than in normal sinus rhythm. These differences suggest that the mechanism of re-entry observed in this study is not commonly operative in most clinical cases of ventricular tachycardia, which may have as their basis ectopic impulse formation or micro-re-entry. It is, however, conceivable that if re-entry utilizing the bundle branches is sustained, the His bundle electrogram may precede the following ventricular activation by a normal or more than normal H-V interval. Theoretically it is also possible that H-V intervals may be normal or longer during ectopic ventricular tachycardia when there is associated antegrade conduction delay or block from the point of impulse discharge. This is more likely to occur when the ectopic focus is located in the bundle branches.

Results obtained in this study also suggest that multiple successive ventricular beats seen on standard ECG with wide bizarre QRS complexes may not necessarily originate from the ventricles but may represent A-V nodal re-entry or dissociated sinus captures (fig. 4).

Figure 6

Diagrammatic representation of possible events which follow right ventricular stimulation. The possible sites of block and the circuits of re-entry are depicted. See discussion for details. Abbreviations: A = atrium; AVN = A-V node; H = bundle of His; RB = right bundle branch system; LB = left bundle branch system; S1 = basic ventricular drive stimulus; S2 = premature ventricular stimulus.

Re-entry was observed for three consecutive beats. Spontaneous termination of the re-entrant phenomenon could result because successive ventricular depolarizations (V2 V3) produce a decrease in refractoriness of the HPS which in turn alters the critical balance between refactoriness and delayed conduction which is necessary for sustaining a re-entrant phenomenon. One may therefore postulate that a sustained re-entry involving a long re-entrant pathway, i.e., both bundle branch systems, may be more difficult to maintain.

The results of this study differ from what is usually

References
2. Hoffman BF: The genesis of cardiac arrhythmias. Prog Cardiovase Dis 8: 319, 1966

Figure 7

Re-entry within the HPS in a patient with antegrade right bundle branch block. At a constant ventricular cycle length of 700 msec, and an S1, S2 interval of 360 msec, S2 initially conducts retrogradely with an S1, H2 interval of 235 msec (panel A). Note the relatively short time of 35 msec interval in the H1, A2 interval. S2 blocks retrogradely below the bundle of His between S1, S2 interval of 350 to 330 (panels B and C). These last beats in both panels B and C are sinus escape beats and show complete right bundle branch block pattern. At closer coupling of 320 msec (panel D), S2 encounters marked His-Purkinje delay which results in re-entry within this system, and the H2, V3 interval measures 45 msec. The relatively long S1, H2 interval of 300 msec and the QRS morphology of V1 (right bundle branch block pattern) suggests reciprocation either in the left bundle branch system or due to unidirectional block in the right bundle with V1 conducted along the right bundle and re-entered through the left bundle.

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15. Durrer D, Van Dam RTH, Freud GE, Janse MJ: Reentry and ventricular arrhythmias in local ischemia and infarction of the intact dog heart. Proc Kon Nederl Akad Van Wetensch Amsterdam 74: 4, 1971
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