His Bundle Electrograms in Patients with Short P-R Intervals, Narrow QRS Complexes, and Paroxysmal Tachycardias

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SUMMARY
His bundle electrograms were recorded in three patients with short P-R intervals, narrow QRS complexes, and a history of paroxysmal tachycardias. During sinus rhythm or atrial stimulation with long cycle lengths, the shortening of the P-R interval was due to a decrease in the low right atrium-His (LRA-H) interval (representing A-H conduction time). The latter was also short during retrograde (V-A) conduction. These findings support the existence of an A-V nodal bypass operation in both directions. In one patient, the LRA-H interval did not lengthen when the atrial rate was increased. Intermittent atrial pacing was performed in the two other patients. The LRA-H interval was short at long coupling intervals, but it started to increase (progressively) at a given Stimulus1-Stimulus2 interval. Apparently, the refractory period of the accessory bundle was encountered so that the impulse was propagated, with various degrees of delay, through the A-V node. A James bundle need not be present in all patients with similar electrocardiograms. Abnormalities of unknown origin could cause this phenomenon. Reciprocating tachycardias were induced by stimulation of the atria in one patient. The triggering beat consistently had a long A-V conduction time. Although in this case retrograde (V-A) propagation most probably occurred through the accessory communication, the possibility of a functional intranodal dissociation of a single anatomical pathway could not be excluded.

Additional Indexing Words:
A-V nodal bypass Reciprocating tachycardia

Intracardiac recordings and His bundle electrograms (HBE) have been most useful in the analysis of the various types of ventricular pre-excitation.1-8 For instance, the presence of two distinct types of accessory A-V communications (electrophysiologic Kent and Mahaim bundles) have been recently identified by these methods.2 Moreover, Durrer et al. have presented evidence supporting the existence of an A-V nodal bypass in a patient with a short P-R interval and normal QRS complexes.7, 8 In view of these findings it appeared of interest to present the information obtained from three patients with short P-R intervals and narrow QRS complexes who were studied with His bundle electrograms and electrical stimulation of the heart.

Methods
The technique of His bundle recordings used in our department has been described in previous communications.1, 2, 9 After we obtained consent, a tripolar catheter (Elecath Corporation, Rahway, N. J.) was introduced percutaneously through a femoral vein and positioned across the septal leaflet of the tricuspid valve. Two bipolar leads with interelectrode distances of 1 and 11 mm, respectively, were recorded (HBE1 and HBE11).9 In addition two bipolar catheters, with interelectrode distances of 1 mm (Elecath Corporation, Rahway, N. J.) were introduced percutaneously.

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through an antecubital vein and placed in the high right atrium (BAE lead) and in the coronary sinus ostia, respectively. Finally, a fourth bipolar catheter, introduced in the same fashion, was used for atrial or ventricular pacing as needed. The terminals from the electrodes were fed into a distribution switch box, the outputs of which were connected to the inputs of the recording instrument (Electronics for Medicine, White Plains, N. Y.). Leads I, II, III, and V₁ were obtained simultaneously with the filtered (400-2000 Hz) bipolar or unipolar intracardiac leads. The stimulator used for continuous, coupled or paired pacing delivered slightly underdamped pulses, 2.5 msec in duration and twice the diastolic threshold values. In one patient intermittent paired pacing was performed in the atria and ventricles. The interval between driving and testing stimuli was progressively shortened in order that the whole cycle might be scanned. The following intervals were measured during sinus rhythm and continuous atrial pacing:

(a) P-R interval (representing intra-atrial, A-V nodal, and His-ventricular conduction times) was measured as in conventional scalar electrocardiography.

(b) HRA-LRA interval (representing intra-atrial conduction time) was measured from the beginning of the P wave in the surface leads, usually coinciding with the onset of the local electrogram in the BAE lead, to the local atrial electrogram in the HBE leads.

(c) LRA-LA interval (representing conduction time from the low right atrium to the explored left atrial site) was measured from the onset of the atrial electrograms in the HBE lead to the beginning of the atrial electrogram in the coronary sinus lead.

(d) LRA-H interval (representing A-H conduction time) was measured from the onset of the local atrial electrogram in the HBE lead to the His (H) deflection.

(e) H-V interval (representing His-ventricular conduction time) was measured from the H deflection to the onset of ventricular depolarization, in whichever lead it occurred first.

(f) LRA-V interval (representing A-V conduction time from the local atrial electrogram in the HBE lead to the beginning of depolarization) was obtained by the addition of (d) and (e).

(g) St-LRA, St-H, and St-V intervals were measured from the moment in which the stimulus (St) artifact was delivered in the high right atrium to the corresponding recording sites.

During intermittent atrial or ventricular pacing, St₁ and St₂ were used in reference to driving and testing stimuli delivered to the atria or ventricles. The corresponding deflections were described as LRA₁ or LRA₂, H₁ or H₂, and V₁ or V₂. A similar nomenclature, but with the intervals in reversed

**Figure 1**

*Case 1. Surface electrocardiograms showing a short P-R interval with narrow QRS complexes.*

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![Sinus Rhythm Diagram](image)

QRS complexes of normal duration and width (80 msec). A slurring of the R wave was seen in II, III, and aVF. This was not a true delta wave since it was not observed in other leads. Left ventricular hypertrophy was also present. With the use of intracardiac recordings it was possible to subdivide the P-R interval into various components having the following durations (fig. 2): HRA-LRA, 50; LRA-H, 30; H-V, 35; and LRA-V, 65 msec. An LRA-H interval of 30 msec is definitely short (lower limit of normal in our department is 50 msec). In this case, "true" A-V conduction time (represented by the LRA-V interval) was shortened at the expense of the LRA-H interval, since the H-V interval was within normal limits (35-55 msec in our laboratory).1, 2, 9

Atrial pacing at all rates failed to increase the duration of the LRA-V interval. This is shown in figure 3, where the atria were stimulated at a rate of 230 beats/min. The first artificial stimulus (St), which was delivered to the high right atrium, fell on top of a sinus P wave; hence it was unable to stimulate the atria. However, the second, third, and fourth stimuli were able to produce propagated responses. The corresponding St-LRA and LRA-V intervals measured 75 and 65 msec, respectively. The latter value was similar to the one observed during sinus rhythm. The intra-atrial conduction time, which was longer than normal, (75 msec compared to 50 msec; see fig. 1) can be explained on the basis of slow propagation during the relative refractory period, a consequence of the fast pacing rate. Finally, the last stimulus was not followed by a P wave, because of either atrial refractoriness or exit block around the pacing catheter. For technical reasons, a His bundle deflection was not recorded during atrial pacing in this patient.

A His bundle deflection reappeared after atrial pacing when the corresponding catheter was repositioned. Hence, retrograde activation of the His bundle was detected during ventricular pacing in both driving and testing beats (fig. 4). Criteria for the diagnosis of

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**Case 1**

This 32-year-old male had atypical chest pain and a long history of palpitations. He was admitted for cardiac evaluation. The results of physical examination were negative. Hemodynamic studies gave results within normal limits. The electrocardiogram (fig. 1) showed sinus rhythm with slightly notched P waves, a short P-R interval (115 msec), and...
Case 1. Right atrial pacing at a rate of 230 beats/min, showing that the LRA-V interval did not increase during this procedure. This interval had the same duration as when sinus rhythm was present (see fig. 2). The vertical lines were artificially drawn to indicate the onset of ventricular depolarization.

Case 1. The duration of the H-LRA interval of both driving (H₁-LRA₁) and testing (H₂-LRA₂) stimuli was short: 40-45 msec. The values obtained during sinus rhythm are also shown for comparison (left panel).
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Case 2. Surface electrocardiogram in a patient with a short P-R interval, narrow QRS complexes, and a history of paroxysmal tachycardia.

For evaluation of his heart condition. There was no history of hypertension, angina pectoris, or rheumatic fever. Physical examination revealed an S3 gallop and a type I crescendo-decrescendo systolic murmur in the apex transmitted to the fourth left intercostal space. Frontal and oblique X-rays showed moderate cardiomegaly, mainly at the expense of the left ventricle. Coronary angiograms were normal. Hemodynamic studies revealed a moderate elevation of the left ventricular end-diastolic and pulmonary artery pressures, as well as a slight reduction of the cardiac index. The electrocardiogram (fig. 5) showed sinus rhythm with a short P-R interval (120 msec) and normal QRS complexes. The durations of the various components of the P-R interval were as follows (fig. 6, left): HRA-LRA, 40; LRA-H, 35; and H-V, 45 msec. Hence, the LRA-H interval was shorter than normal. When the P waves were upright in the standard leads the sequence of atrial activation occurred in a superoinferior direction, from HRA to LRA to LA (fig. 6, left). However, mechanical stimulation of the coronary sinus (fig. 6, right) produced a short P-R interval with negative P waves in leads II, III, and aVF. Atrial depolarization was reversed: from LA to LRA to HRA. Therefore, in retrospect, the electrocardiogram shown in

retrograde His bundle activation have been presented elsewhere.9

The H1-LRA1 interval of 40 msec was definitely short (the lower limit of normal is 50 msec).9 Intermittent paired ventricular stimulation at an St1-St2 interval of 300 msec (fig. 4, right) showed that the St2-H2 interval had a duration 280 msec. The H2-LRA2 interval still measured 40 to 45 msec. This retrograde delay below the His bundle at short coupling intervals has also been noted in previous communications. It was ascribed to slow intraventricular propagation during the relative refractory period.9

In summary, the shortening of the P-R and LRA-V intervals observed during sinus rhythm was due exclusively to a reduction of the LRA-H interval. In addition, the LRA-V interval did not increase with atrial pacing with rates as high as 230 beats/min. Retrograde H-LRA conduction time was also short in both driving and testing beats. These findings are consistent with a rapidly conducting A-V nodal bypass operating in forward and retrograde directions.

Case 2

A 49-year-old alcoholic, who had sustained several documented attacks of paroxysmal atrial tachycardia and heart failure, was brought to the Cardiopulmonary Laboratory for evaluation of his heart condition. There was no history of hypertension, angina pectoris, or rheumatic fever. Physical examination revealed an S3 gallop and a type I crescendo-decrescendo systolic murmur in the apex transmitted to the fourth left intercostal space. Frontal and oblique X-rays showed moderate cardiomegaly, mainly at the expense of the left ventricle. Coronary angiograms were normal. Hemodynamic studies revealed a moderate elevation of the left ventricular end-diastolic and pulmonary artery pressures, as well as a slight reduction of the cardiac index. The electrocardiogram (fig. 5) showed sinus rhythm with a short P-R interval (120 msec) and normal QRS complexes. The durations of the various components of the P-R interval were as follows (fig. 6, left): HRA-LRA, 40; LRA-H, 35; and H-V, 45 msec. Hence, the LRA-H interval was shorter than normal. When the P waves were upright in the standard leads the sequence of atrial activation occurred in a superoinferior direction, from HRA to LRA to LA (fig. 6, left). However, mechanical stimulation of the coronary sinus (fig. 6, right) produced a short P-R interval with negative P waves in leads II, III, and aVF. Atrial depolarization was reversed: from LA to LRA to HRA. Therefore, in retrospect, the electrocardiogram shown in
Figure 6

Case 2. Shortening of the LRA-H interval during sinus rhythm (left) and mechanical coronary sinus stimulation (right). Sinus beats showed a normal sequence of activation (HRA to LRA to LA). Retrograde activation of the atria (LRA to HRA) occurred in coronary sinus beats. The coronary sinus bipolar electrogram (CSBE) recorded the activity of the left atrium (LA) from the coronary sinus os.

Figure 7

Case 2. Intermittent paired atrial stimulation. Cycle length of driving (St₁) beats was 750 msec. Testing stimuli (St₂) were delivered at St₁-St₂ intervals of 330 and 480 msec. The St₂-R intervals were longer than the St₁-R intervals, the increase being proportional to the reduction of cycle length. The left panel shows aberration with the morphology of right bundle-branch block with left anterior hemiblock. A more detailed description of the results of this procedure is presented in figure 8.

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Figure 5 probably represented sinus rhythm with a short P-R interval rather than an ectopic atrial rhythm.

Atrial pacing at rates over 160 beats/min produced the typical iatrogenic A-V nodal Wenckebach in which the prolongation of the St-V interval was due exclusively to a lengthening of the LRA-H interval. Additional information regarding A-V transmission is presented in figures 7 and 8. The atria were driven at a rate of 80 beats/min (cycle length of 750 msec). Testing stimuli, delivered at St₁-St₂ intervals between 750 and 540 msec, showed St-LRA₂, LRA₂-H₂, and H₂-V₂ intervals with the same duration as that of driving beats (35, 35, and 45 msec, respectively). At St₁-St₂ intervals below 520 msec, the LRA₂-H₂ and H₂-V₂ intervals became progressively longer (figs. 7 and 8). Finally, at a delay of 300 msec the atrial impulse failed to activate the His bundle; the absolute refractory period of the A-V node was reached. It should be noted that the St-LRA₂ interval started to increase at a delay of 360 msec. The latter, therefore, indicated the beginning of the relative refractory period of the atria.

Right ventricular apical pacing was performed at a rate of 80 beats/min (figs. 9 and

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Case 2. Duration of the St1-LRA2, LRA2-H2, and H2-V2 intervals (ordinate) as a function of the St1-St2 intervals during intermittent atrial paired pacing (abscissa). Values are expressed in msec. The duration of the corresponding intervals at any delay can be determined as follows: St2-LRA2, from the bottom line to the solid circles; LRA2-H2, from the solid circles to the open circles; and H2-V2, from the open circles to the triangles. Note that the LRA2-H2 interval was short (35 msec) when the cycle length was long (between 750 and 540 msec). At St1-St2 intervals of 520 msec or less, the LRA2-H2 and H2-V2 intervals showed a gradual increase. Aberrant ventricular conduction, with the morphology of right bundle-branch block (RBBB) and left anterior hemiblock (LAH), appeared when the St1-St2 intervals were shorter than 470 msec. Finally, at a delay of 310 msec the atrial impulse failed to activate the His bundle.

Case 2. Intermittent paired ventricular stimulation at various St1-St2 intervals. At a delay of 400 msec, St1 either activated (retrogradely) the His bundle (left panel) or failed to do so (middle panel). In the former instance H2 was followed by LRA2; in the middle panel retrograde activation of the atria did not occur.
Figure 10

*Case 2. Duration of the St₂-H₂ and St₂-LRA₂ intervals (ordinate) as a function of the St₁-St₂ intervals (abscissa) during intermittent paired ventricular stimulation. The H₂-LRA₂ interval had the same short duration (25 msec) at all St₁-St₂ intervals. Note that as the latter decreased, the St₁-H₂ interval increased. At delays of 390 and 400 msec some of the testing (St₅) stimuli did not propagate (retrogradely) to the His bundle. In these instances, retrograde activation of the atria also failed to occur.*

10). Retrograde activation of the His bundle was not observed within driving beats, due to the distortion of the HBE leads produced by the movements of the tricuspid leaflets.

At St₁-St₂ intervals of 470 msec, the St₂-H₂ and St₂-A₂ intervals measured 70 and 95 msec. Thus the retrograde H₂-LRA₂ interval was abnormally short (25 msec). Figure 10 shows that the St₁-H₂ and St₂-LRA₂ intervals became progressively longer as the St₁-St₂ intervals shortened; yet the H₂-LRA₂ interval remained unchanged (25 msec).

It is interesting to note that at coupling intervals of 390 and 400 msec, St₂ sometimes failed to reach the His bundle (fig. 9 left and middle panels, fig. 10). Whenever H₂ was absent, retrograde activation of the atria did not occur. This indicated that the impulse reached the atria through a communication located above the His bundle. Moreover, at coupling intervals of 310 msec, the St₂-H₂ interval was slightly shorter (210 msec compared to 230 msec) than when the coupling interval was 320 msec (fig. 10). However, the corresponding H₂-LRA₂ intervals maintained their constant value of 25 msec.

In this case, the shortening of the P-R and LRA-V intervals during sinus rhythm and paired atrial pacing (with long St₁-St₂ inter-

vals) was due to a decrease in the LRA-H intervals (35 msec). However, this patient showed the "normal" response to atrial pacing as well as to paired pacing when the cycle lengths were short. The retrograde H-LRA intervals had consistently short (and fixed) values at all St₁-St₂ intervals. These findings are consistent with an A-V nodal bypass operating in both forward and backward directions.

**Case 3**

This 41-year-old female had suffered frequent episodes of paroxysmal tachycardia during the last 15 years. There was no history of rheumatic fever, hypertension, diabetes, or coronary artery disease. The results of physical examination were negative. The patient was referred for His bundle studies. The electrocardiogram (fig. 11) showed a short P-R interval (115 msec), normal QRS complexes, and nonspecific ST changes. During sinus rhythm the durations of the various

Figure 11

*Case 3. Intracardiac recordings showing that the shortening of the P-R interval was due to a reduction of the LRA-H interval.*

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components of the P-R interval were as follows: HRA-LRA, 40; LRA-H, 40; and H-V, 35 msec (fig. 11). Therefore, the shortening of the P-R interval was due to a reduction in LRA-H interval. This shortening persisted until the cycle length was reduced below 550 msec. Thereafter, the LRA-H became progressively longer, as in case 2. However, in contrast to the first two patients, several episodes of reciprocating tachycardia were induced by atrial pacing in this patient. The arrhythmia invariably occurred after a long St-R interval.

The durations of the LRA-H and H-V intervals of the first paired atrial beat in figure 12 were similar to that of sinus beats. LRA-H intervals became progressively longer after the second and third stimuli due to the reduction of the cycle lengths. In fact, the LRA-H interval produced by the third stimulus measured 260 msec. The corresponding His bundle deflection was followed by retrograde activation of the low right atrium. The corresponding H-LRA interval measured 45 msec. This phenomenon occurred because the fourth stimulus artifact was unable to produce a P wave, for, otherwise, retrograde activation of the atria would have been impossible. As shown in figure 12, two possible mechanisms can explain this premature, retrograde, atrial deflection. The impulse could have returned through a bypass extending from the upper portion of the His bundle to the atria (represented by the oblique broken lines extending from H to A). However, the possibility of a functional longitudinal intranodal dissociation cannot definitely be excluded. In the diagram this second mechanism is indicated by the oblique solid line emerging (in a superior direction) from the downwards oriented (oblique) line, representing transmission through the A-H region.

This beat, irrespective of its mechanism, initiated a run of reciprocating tachycardia during which forward conduction occurred via the His bundle. The slight deformity of the second and fourth QRS complexes was ascribed to a minor degree of aberrant
ventricular conduction. Spontaneous cessation of the arrhythmia occurred when an impulse was blocked within the A-V node in its forward journey toward the His bundle.

The findings in this patient are compatible with an A-V nodal bypass operating in both directions. Although the reciprocating tachycardias probably involved two separate anatomical communications, the presence of a single, functionally dissociated anatomical (A-V nodal) pathway cannot be excluded.

Discussion

The term "short P-R interval" used in this communication implied a total A-V conduction time (from the beginning of the P wave to the onset of ventricular depolarization) of 120 msec or less. However, it has to be stressed that P-R intervals of 120, 110, and 100 msec have been reported in 0.4 to 2% of presumably normal adult individuals. Hence, it is possible that electrocardiograms similar to the ones shown in figures 1, 5, and 11 (without the history of paroxysmal tachycardia) could have represented a normal variant. However, for practical purposes a division between normal and abnormal was necessary.

According to Scherf and Cohen, the electrocardiographic syndrome of short P-R intervals and normal QRS complexes has been described in many conditions, namely: coronary nodal rhythm, A-V nodal rhythm with positive P waves, beri-beri heart disease, hyperthyroidism, hypertension, "active" rheumatic fever, myocardial infarction, "hyperexcitability and erythema" of the heart, and delayed activation of the atria (presumably due to what is now known as sinoventricular conduction).

Several mechanisms have been implied in the genesis of this entity. Lown and coworkers, who pointed out that some of these patients were prone to develop paroxysmal tachycardias, suggested that the electrocardiographic changes were due to an endocrine and autonomic imbalance. Yet, Scherf and Cohen stated that "most authors considered such electrocardiograms as a variant, atypical, or partial form of pre-excitation syndrome." Burch and Kimball postulated the existence of an anomalous pathway connecting the atra with the ventricular septum. Ferrer and Durrer believed that the short P-R interval could be due to the presence of an A-V nodal bypass, perhaps one of the groups of fibers described by James (hereby designated as James bundle). Durrer et al. estimated that the degree of P-R shortening depended on the conduction velocity through the James bundle, the activation time of its atrial end, and the site of insertion.

In the patient studied by Durrer et al. the impulse propagated normally within the atria, while the P waves were positive in the standard leads. This finding suggested that the rhythm was sinoatrial in origin, thereby ruling out an ectopic mechanism. A similar activation sequence was also noted in one of our patients in whom coronary sinus stimulation produced a short P-R interval with negative P waves in leads II and III (fig. 6). In addition, atrial stimulation in Durrer's case did not produce the expected P-R prolongation. Retrograde (V-A) conduction time was also extremely short.

In the three patients reported in this communication His bundle recordings showed that, during sinus rhythm, the shortening of the P-R interval was due exclusively to a shortening of the LRA-H interval. The latter has been used as a rough estimate of A-V nodal conduction time in cases (without preexcitation) in which A-V nodal electrograms were not recorded. Bagdonas et al. stated that if no slowing of conduction in the A-V node was present, activity in the low right atrium and His bundle would occur at more or less the same time. According to these authors, the fact that His bundle depolarization occurred at a given delay (35 to 60 msec in the dog, 50 to 120 msec in man) after local activity in the low right atrium suggested that the LRA-H interval represented the magnitude of the A-V nodal delay. It should be emphasized that when the A-V node is bypassed, the LRA-H interval does not measure A-V conduction time. In these cases it
only represents the time elapsed between activation of the low right atrium and His bundle. The shortening of the forward and retrograde LRA-H intervals observed in the patients studied is a valid proof that the usual A-V junctional delay did not occur and that the forward or retrograde impulses propagated towards the His bundle or atria at a speed faster than normal. These findings can be explained by the presence of a communication between the atria to the His bundle.

Although His bundle deflections were not recorded during rapid atrial pacing in case 1, the fact that the LRA-V interval remained unchanged indicated that A-V nodal transmission time had not increased. Hence, the area of normal A-V nodal delay was bypassed. The slight prolongation of the St-V interval noted at high rates was due to the increase in intra-atrial conduction, which was in turn a consequence of slow propagation during the relative refractory period of the atria.

In cases 2 and 3, the LRA-H interval was short during sinus rhythm and atrial stimulation at long cycle lengths. However, it increased when the cycle length was reduced. These findings suggested that the "normal" A-V nodal delay was not encountered until the cycles became sufficiently short, that is, when the absolute refractory period of the James bundle was reached. This phenomenon can occur only if the refractoriness of the bypass is greater than that of the A-V node. In fact, a similar response to atrial pacing was observed in patients with ventricular preexcitation presumably due to a Kent bundle (hereby defined as a total bypass of the normal A-V conducting system irrespective of its anatomical location).\textsuperscript{1, 2, 5-8, 19} In some of these patients, A-V conduction occurred through both Kent and His bundles when the cycle lengths were long. Shortening of the cycles produced increasing degrees of preexcitation due to the smaller contribution of the stimulation propagating via the His bundle.\textsuperscript{19} The latter was a consequence of the progressive prolongation of A-V nodal conduction time. At even shorter cycles, propagation through the Kent bundle failed. Hence, A-V conduction occurred exclusively through the normal pathways, although with a long P-R interval due to the normal A-V nodal refractoriness.

In cases 1 and 2, retrograde conduction time was shorter than normal, indicating that the ventricular impulse also used the nodal bypass in its propagation toward the atria. A detailed analysis of retrograde conduction in case 2 showed that the St\textsubscript{2}-H\textsubscript{2} intervals increased as the St\textsubscript{1}-St\textsubscript{2} intervals decreased. This response was noted in a previous communication from our department in the absence of preexcitation.\textsuperscript{9} It was ascribed to slow propagation through the ventricles and/or bundle branch-Purkinje system during the relative refractory period. Figure 10 showed that the H\textsubscript{2}-LRA\textsubscript{2} intervals were the same (short) at all St\textsubscript{2}-H\textsubscript{2} intervals, indicating that once the impulse reached the His bundle, it propagated back to the atria at a rapid speed. On the other hand, retrograde activation of the atria did not occur if St\textsubscript{2} failed below H\textsubscript{2} (fig. 9). This behavior gives further support to the assumption that the bypass extended from the atria to the upper His bundle. In contrast, the V-A conduction pattern in a patient with preexcitation due to a Kent bundle was such that retrograde activation of the atria could occur even before that of the His bundle, suggesting retrograde atrial preexcitation through an extra-nodal, extra-Hisian pathway.\textsuperscript{9}

Atrial stimulation induced several episodes of paroxysmal tachycardia in the third patient. The arrhythmias invariably appeared when the atrial cycle was short and the LRA-V interval prolonged. Apparently the triggering (atrial) impulse occurred at a moment in which the bypass was refractory and hence, unable to be activated in a forward direction (fig. 12). Therefore, the impulse was conducted through the A-V node, traversed the His bundle, and finally reached the ventricles. In addition, it also penetrated the lower end of the James bundle and propagated (in an inferosuperior direction through this structure) in its journey towards the atria. Atrial activation was possible because the fourth (and last) artificial stimulus was ineffective. A
reciprocating tachycardia was thus initiated. Although we favor this mechanism, a functional longitudinal (intranodal) dissociation cannot be excluded. Both possibilities are represented in figure 12.

It has to be stressed that an A-V nodal bypass need not be the only explanation for the electrocardiographic syndrome of short P-R interval, narrow QRS complexes, and paroxysmal tachycardias. As mentioned by Durrer et al., "A-V nodal abnormalities of unknown origin may cause this entity. The possibility that a group of nodal fibers could (because of congenital abnormalities or acquired disease) conduct faster than those constituting the so-called α and β groups has to be considered.

The analysis of the various patterns of forward and retrograde conduction presented in figures 1 to 12 could not have been made from the surface electrocardiogram alone. This confirms the value of intra-atrial and His bundle recordings in localizing the site(s) in which an impulse can be delayed or accelerated in its propagation from atria to ventricles or vice versa.

References


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