“Supernormal Conduction” of a Premature Impulse Utilizing the Fast Pathway in a Patient with Dual Atrioventricular Nodal Pathways


SUMMARY
Electrophysiological studies with atrial extrastimulus technique suggested the presence of dual atrioventricular (A-V) nodal pathways in a patient with hypothyroidism, as evidenced by a sudden increase in H1-H2 intervals at critical A1-A2 coupling intervals. Following the atrial extrastimulus (A2), a third impulse (A3) occurred spontaneously. During slow pathway conduction of A3, an A3 appearing at a critically timed interval allowed fast pathway conduction, resulting in an earlier than expected QRS (a form of supernormal conduction). This demonstration of fast pathway conduction during slow pathway conduction adds strong evidence for the existence of dual A-V nodal pathways.

Additional Indexing Words:
His bundle electrogram Refractory periods Atrial extra-stimulus technique
Atrioventricular conduction Atrial pacing

The presence of dual atrioventricular (A-V) nodal pathways has been suggested in some patients, utilizing the atrial extrastimulus (A2) technique.1-3 In these patients, a sudden increase in H1-H2 intervals occurs at a critical range of A1-A2 coupling intervals, suggesting failure of a fast pathway and antegrade conduction via a slow pathway. Further substantiation of the existence of dual A-V nodal pathways in such patients would be provided if an additional extrastimulus (A3), delivered following an A2 conducted via the slow pathway, would result in production of an earlier than expected QRS (supernormal conduction). This would suggest that the fast pathway previously blocked was available for antegrade conduction following an appropriate recovery period after A2. The present report provides such evidence.

Report of Case
The patient was a 67-year-old male with hypertensive cardiovascular disease and documented hypothyroidism. There was no history of palpitation or paroxysmal supraventricular tachycardia. Electrocardiograms revealed first degree A-V block (P-R of 0.22 sec), complete right bundle branch block, and left axis deviation (−135°). Electrophysiological studies were performed because of intraventricular conduction defect.

Electrophysiologic Studies
Informed consent was obtained. A quadripolar catheter (1 cm interelectrode distance) was introduced through the right femoral vein and positioned in the high right atrium (HRA) for recording and atrial stimulation. A tripolar catheter was positioned across the tricuspid valve for recording of His bundle electrograms.4 Refractory periods were measured by the atrial extrastimulus technique.5 S1, HRA1, A1 and H1 denote the driving stimulus, and the corresponding high right atrial, low right atrial, and His bundle electrograms, respectively. HRA2, A2 and H2 denote the responses to an extrastimulus (S2) delivered at decreasing coupling intervals. S2 was an additional extrastimulus delivered after S1. HRA2 and A2 were used to denote responses to S2 or spontaneous atrial premature responses occurring after A2, respectively. HRA4 was used to denote another atrial impulse after A3.

The sinus rate was 73 beats/min, with the following conduction intervals: P-A = 61 msec; A-H = 110 msec; and H-V = 95 msec. Atrial pacing at rates from 80 to 120 beats/min increased the A-H interval to 175 msec. Atrioventricular nodal Wenckebach periods were noted at a paced heart rate of 130 beats/min. Extrastimuli were coupled to a driven rhythm with a cycle length of 667 msec.

The plot of A1-A2, H1-H2 and A1-A2, A2-H2 intervals is shown in Figure 1. As A1-A2 decreased from 670 to 480 msec, A2-H2 increased from 150 to 200 msec (figs. 1 (right) and 2A). At a coupling interval of 470 msec or less, a sudden increase in H1-H2 (and A2-H2) occurred (figs. 1 and 2B). Further decrease in A1-A2 resulted in further prolongation of A2-H2 until, at a critical A2-H2 interval, A-V nodal re-entry occurred (fig. 2C).

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The fast pathway curve is on the right, and the slow pathway curve on the left (fig. 1). Echo beats were elicited when the premature atrial beat was conducted via the slow pathway with a critically prolonged A3-H2. Thus, a clearly defined A-V nodal echo zone could be delineated coinciding with a portion of the slow pathway curve.

At close A1-A2 coupling intervals of 340 to 390 msec, early premature atrial responses (A3) were sometimes noted. These were characterized by short A2-A3 intervals (350 to 405 msec), and an abnormal sequence of atrial activation (low and high atrial electrograms recorded simultaneously). These were most likely due to atrial re-entrance, or sinus node re-entrance with an altered atrial activation sequence. The occurrence of spontaneous A3 might relate to the prolonged P-A interval.

Whenever A3 occurred, H1-H2 was consistently shorter when compared to similar H1-H2 without intervening A3. An example is shown in figure 3. At an A1-A2 of 385 msec, A2-H2 was 660 msec (slow pathway conduction time), and H1-H2 was 890 msec (fig. 3A). When A1-A3 was decreased by 10 msec, an A3 occurred, and H1-H2 (denoted as H1-H2A) interval shortened (fig. 3B). The only reasonable explanation for this shortening of H1-H2A depends upon the existence of dual pathways. If this patient had only a single A-V nodal pathway, then the long A3-H2 would have to reflect relative refractoriness of this single pathway. Unexpected shortening of conduction time A3-H2 after A3 would have to reflect interaction of the impulses of A2 and A3, resulting in faster than expected A-V nodal conduction. Summation of impulses A2 and A3 in a single pathway is not likely to occur, since A3 would probably be blocked because of decremental conduction in the A-V node, rather than catch up to the previous impulse (A2). Therefore, the shortening of H1-H2 (and of necessity, A3-H2) probably reflects conduction of A3 via a previously blocked fast pathway. Thus, the H1-H2 in figure 3B is really H1-H2. The resultant A3-H2A of 140 msec is consistent with fast pathway conduction (fig. 1, right).

Since the impulse propagated from A3 arrives at the His bundle before A2 would have arrived, the final common pathway (which probably includes a portion of the A-V node and His bundle) is already engaged and is refractory to the delayed slow pathway impulse. However, summation of A2 and A3 could possibly occur with dual A-V nodal pathways, allowing A2 to "catch up" to A3 and shorten A-V nodal conduction.

Further support for the presence of dual pathways was provided by using a third extrastimulus (S3). S1-S3 coupling interval was constant at 390 msec in the three panels of figure 4. In figure 4A, A2 was conducted through the slow pathway with an A3-H2 interval of 640 msec, and H1-H2 of 890 msec. S3 was introduced 670 msec after S2 and was conducted to the ventricles via the slow pathway with a conduction time of 580 msec. Although the S2-S3 interval of 670 msec allowed more than enough time for recovery of the antegrade fast pathway, this same time interval allowed retrograde penetration of the fast pathway (following antegrade slow pathway conduction). With the fast pathway thus refractory, S3 (and HRA3) were therefore conducted via the slow pathway.

In panel 4B, a spontaneous A3 occurred 350 msec following A2 and S3 and failed to capture the atria because of atrial refractoriness. With the occurrence of A3, H1-H2A shortened to 740 msec, suggesting that A3 was conducted via the fast pathway. The resultant A3-H2A interval of 170 msec preempted slow pathway conduction and the conducted A3 controlled the ventricles. In figure 4C, a spontaneous A3 similarly conducted via the fast pathway. S3, however, resulted in an atrial capture (HRA3) with HRA2-HRA3 intervals of 690 and A2-HRA3 intervals of 315 msec. HRA3 was conducted to the ventricles via the slow pathway with a slow pathway conduction time of 585 msec (A-H interval). This conduction time was similar to that of the HRA3 in figure 4A, indicating that in both panels (HRA3 in figure 4A, and HRA3 in figure 4C), the impulse was conducted via the slow pathway. In panel C, the slow pathway was utilized because of antegrade refractoriness of the fast pathway, the A2-HRA of 315 msec being less than the fast pathway effective refractory period. It should also be noted that previous fast pathway conduction did not affect subsequent slow pathway conduction.

An alternate explanation to these findings can be postulated by assuming that the long H1-H2 intervals represent junctional escape beats following a blocked A2, rather than slow pathway conduction times. If A2 was blocked in the A-V node, A3 would have a short A-V nodal conduction time, due to recovery of A-V nodal conduction. The postulated A-V nodal re-entrant echoes (A3) could then reflect sinus return beats following interpolation of A3. However, these assumptions seem to be a less likely explanation for the findings in this case, since 1) Conducted H-H intervals of up to 1120 msec were observed without intervening junctional escape beats. Examples of H-H intervals of 1000 and 1045 msec in duration are shown in figures 2C and 3B. These H-H intervals are well in excess of the postulated junctional escape times of 860 to 930 (fig. 1A). 2) Atrial responses to S3 (HRA3 in figure 4A, and HRA3 in figure 4C) were conducted with long A-H intervals, indicating that the A-V node (slow pathway) was capable of delayed conduction. 3) Mean sinus node recovery time at the driven cycle length of 667 msec was 1050 with a range of 990 to 1120 msec. Atrioventricular re-entrant atrial echoes (A3) occurred at A1-A3 intervals of 1150 to 1190 msec, which is longer than sinus return cycles without A3, suggesting that A3 was not interpolated.

It could also be argued that shortening of A-V conduction following A3 depended upon the site of origin of A3, which
in turn could affect A-V nodal conduction. However, such pronounced shortening of A-V nodal conduction would be unlikely to occur from an impulse arising above the A-V node, in the absence of a fast pathway. Furthermore, the A-V nodal conduction times of A₃ were compatible with fast pathway conduction time, as seen in figure 1 (right panel).

**Discussion**

Dual A-V nodal pathways can be defined utilizing the atrial extrastimulus technique, with construction of A₁-A₂, H₁H₂ curves. Dual A-V nodal pathways are characterized by a sudden jump in H₁-H₂ at a critical range of A₁-A₂ coupling intervals. The portion of the curve to the right of the jump represents the fast pathway curve, and to the left, the slow pathway curve. Electrocardiographic manifestations of dual A-V nodal pathways include: 1) two P-R intervals on the surface electrocardiogram; 2) occurrence of A-V nodal re-entrant echo beats with or without paroxysmal A-V nodal re-entrant tachycardia; 3) atypical Wenckebach periods.

Mendez and coworkers studied the effect of premature atrial stimulation upon the ventricular echo phenomenon in the canine heart. Ventricular echo responses were obtained, with premature stimulation of the His bundle at critical H₁-H₂ intervals. If the atrium was prematurely stimulated prior to the arrival of retrograde A₂, the premature atrial extrastimulus was conducted to the ventricle with a relatively short A-H interval, arriving at the ventricles before the occurrence of the expected ventricular echo, indicating that the premature atrial stimulus was conducted to the ventricles through a pathway.

**Figure 2**
The effect of shortening of the A₁-A₂ coupling intervals on the A-V nodal conduction of premature beats (A₁-H₂) and the H₁-H₂ responses. Electrocardiographic leads I, II, III, V₁, high right atrial electrogram (HRA). His bundle electrogram (HBE) are shown in each panel. CL represents the basic driving cycle length. S₁, A₁, H₁, and S₂, A₂, H₂ represent the stimulus, atrial and His bundle electrograms of the basic drive and premature atrial stimuli, respectively. A₃ (panel C) represents an A-V nodal re-entrant atrial echo. To the right of each recording, a ladder diagram representing atrial, A-V node, and ventricles is shown. Shortening of A₁-A₂ by 10 msec results in a sudden increase of A₁-H₂ interval, representing slow pathway conduction time (panels A and B). At a critical A₁-H₂ interval, A-V nodal echo beats occur (panel C). Time lines are at one second, and paper speed is 100 mm/sec on this and subsequent illustrations.

**Figure 3**
Simultaneous slow and fast pathway antegrade conduction. HRA₁, HRA₂ represent high right atrial electrogram of basic driven and premature beats. A₃ represents a spontaneous atrial premature impulse. H₂₃ represents His bundle electrogram in response to A₃. Other symbols and abbreviations are similar to figure 2. Note that the appearance of A₃ results in shortening of H₁+H₂₃. This suggests that A₃ conducts through the fast pathway.
which was not engaged during retrograde conduction from His bundle to atrium.

In a subsequent study in the opened-heart dog, Moe et al. demonstrated that conduction time of a premature atrial stimulus (A2V2) was shortened by the occurrence of a third atrial response (A3). They suggested that A3 traversed a pathway that had not been entered by A2, since it was hardly likely that A3 produced a supernormal pathway for the preceding response. Thus, Moe et al. postulated that longitudinal dissociation of the A-V node could produce apparent supernormal conduction.

In the present report, dual A-V nodal pathways were suggested from experiments made with the atrial extra-stimulus technique. At a critical A1-A2 (fast pathway effective refractory period), the A2-H2 suddenly lengthened (slow pathway conduction time). At a critical A2-H2, A-V nodal re-entry became apparent. A2 was blocked in the fast pathway and conducted antegradely through the slow pathway. The slow pathway conduction time (critical A2-H2) allowed for recovery of the fast pathway for retrograde conduction. At an A1-A2 interval less than the fast pathway effective refractory period, A2 conducted via the slow pathway. Occurrence of a spontaneous atrial premature beat (A3) demonstrated that during slow pathway conduction, the atrial impulse could simultaneously conduct through the fast pathway, pre-empting control of the ventricle. This demonstration of dual pathways resembled somewhat the previous demonstration of Mendez et al. and was identical to the previous demonstration of Moe et al.

This demonstration of unexpected shortening of A-V nodal conduction time with A2 can be considered a form of "supernormal conduction." The mechanism appears to reflect longitudinal dissociation of the A-V node, i.e., the existence of dual pathways.

References


Figure 4

The S-S-S stimulation pattern in the demonstration of dual A-V nodal pathways. An atrial premature stimulus A2 is introduced in all three panels at an S-S coupling interval of 390 msec. Panel A) An S2 is introduced at an S2-S3 coupling interval of 670 msec. Both S2 and S3 capture the atrium and are conducted through the slow pathway. Panel B) An atrial echo (A3) occurs 350 msec following HRA2; the H-H2-3 interval shortens by 140 msec (in spite of constant S2-S3 coupling interval), indicating that H2-3 results from fast pathway conduction of the A3. S3 failed to capture the atrium. Panel C) Similar to panel B, except that S2 is effective in capturing the atrium, resulting in a fourth atrial depolarization (HRA4) which is conducted to the ventricles through slow pathway with a conduction time similar to the one obtained in the absence of the atrial echo (A3), suggesting that fast pathway conduction did not interfere with subsequent slow pathway conduction.
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