Demonstration of Dual A-V Nodal Pathways in Patients with Paroxysmal Supraventricular Tachycardia

By Pablo Denes, M.D., Delon Wu, M.D., Ramesh C. Dhingra, M.D., Ruben Chuquimia, M.D., and Kenneth M. Rosen, M.D.

SUMMARY
Electrophysiological evidence suggestive of dual atrioventricular (A-V) nodal pathways is presented in two patients with normal P-R interval and reentrant paroxysmal supraventricular tachycardia (PSVT). His bundle recordings and atrial stimulation were used to obtain this electrophysiological evidence.

Refractory periods were measured with the atrial extra-stimulus technique. Plotting of H1-H2 responses against A1-A2 coupling intervals revealed that as A1-A2 decreased, H1-H2 decreased appropriately. At a critical A1-A2, a sudden marked increase in H1-H2 occurred, suggesting failure of fast pathway, (defining the fast pathway effective refractory period ERP). Further shortening of A1-A2 defined a second H1-H2 curve. The longest A1-A2 with no H2 response was defined as the slow pathway ERP. Echo zones coincided with A1-A2 intervals equal to or less than the fast pathway ERP.

These results provide the first electrophysiological demonstration of dual A-V nodal pathways in patients with normal P-R interval and PSVT, as manifest by dual A-V nodal conduction times and refractory periods. Antegrade failure of the fast pathway with subsequent availability for retrograde conduction could allow A-V nodal reentry. These findings provide a basis for reentry in some patients with reentrant PSVT.

Additional Indexing Words:
Paroxysmal atrial tachycardia
Atrial echoes
His bundle electrogram
A-V nodal bypass
A-V nodal reentry

The demonstration of dual atrioventricular (A-V) pathways is most easily accomplished in patients with Wolff-Parkinson-White syndrome. Both electrophysiological and pathological studies suggest the presence of two A-V connections, these being: 1) an anomalous pathway in the A-V ring, and 2) the structures of the normal A-V junction. In patients with preexcitation, the two pathways have different conduction times and, frequently, different refractory periods. Patients with short P-R intervals, narrow QRS complexes, and paroxysmal tachycardia form another group in which dual A-V pathways have been suggested. Some of these patients may have accessory connections between the atria and the lower A-V node.

Animal studies by Rosenblueth and Moe, Preston and Burlington, suggest that the A-V node itself may undergo longitudinal dissociation into two pathways demonstrable under appropriate conditions. Rosen et al. recently reported a patient with two P-R intervals, and two sets of A-V nodal conduction times and refractory periods, suggesting dual A-V nodal pathways.

Recently reported studies in man suggest that A-V nodal reentrance is a common mechanism of paroxysmal supraventricular tachycardia (PSVT). A-V nodal reentrance implies either the presence of dual A-V nodal pathways or reentry by "reflection" in an area of depressed conduction.

The purpose of this report is to describe two patients with history of PSVT and normal P-R
interval studied with His bundle recording and atrial stimulation. These patients had evidence suggestive of both a fast and a slow A-V nodal pathway, each with its own effective and functional refractory period. Failure of the fast pathway was often accompanied by occurrence of A-V nodal echoes, implying that this pathway was utilized for retrograde propagation during episodes of PSVT.

Materials and Methods

Patient Selection

We have studied eight patients with PSVT and normal P-R interval in the past year. In two of these patients, electrophysiological studies suggested the presence of dual A-V nodal conduction pathways. These two are the subjects of the present report. Both patients were male, ages 54 and 50, and each had a history of recurrent episodes of documented PSVT. The resting electrocardiograms showed normal P-R intervals and narrow QRS complexes. The physical examination, radiologic and routine laboratory studies were within normal limits.

Recording Technique

Patients were studied in supine, postabsorptive, and nonsedated state. Under local anesthesia, a tripolar catheter was introduced percutaneously through the right femoral vein and positioned across the tricuspid valve. A second quadripolar catheter was introduced percutaneously through the left femoral vein and positioned in the right atrium. Electrodes 1, 2, and 3 of the tripolar catheter, and 3 and 4 of the quadripolar catheter were connected to a distribution switchbox. The terminals were connected to the A-C inputs of the ECG amplifiers of an Electronics for Medicine Recorder. Frequencies below 40 and above 500 Hz were filtered. ECG leads V1 and II and the atrial (A) and His bundle (H) electrograms were displayed on an oscilloscope and recorded at 100 and 200 mm/sec paper speeds.

Experimental procedures were similar to those described by Wit et al. Basic drive (S1) and test stimuli (S2) were delivered to the right atrium through electrodes 1 and 2 of the quadripolar catheter and supplied by a programmable stimulator. The test stimulus was introduced after every 8th driven or spontaneous beat and the S1-S2 interval was decreased in 5-20 msec steps. The stimulus pulse was rectangular, 2 msec in duration, and approximately twice diastolic threshold intensity.

Definitions

A1, H1, and V1 were the atrial, His bundle and ventricular electrograms of the basic drive impulse. A2, H2, V2 were the atrial, His bundle and ventricular electrograms of the test impulse. A-H, H-V, H2-H2, and V1-V2 intervals were defined and measured as described previously by Wit. The atrial effective refractory period (ERP) was the longest S1-S2 interval, not capturing the atrium. The A-V nodal ERP was the longest A1-A2 not propagated to the His bundle. The A-V nodal functional refractory period (FRP) was the minimum interval between two successive His bundle responses (H2-H2), both propagated from the atrium.

Results

Case 1

Electrophysiological Studies: The resting sinus rate was 65 beats/min and conduction intervals were as follows: P-A 30 msec, A-H 70 msec, and H-V 45 msec. Atrial test responses (A2) were elicited by coupling stimuli to the patient's spontaneous sinus rhythm (fig. 1). H1-H2 responses were plotted against the A1-A2 coupling intervals (fig. 2). As A1-A2 was progressively shortened from 770 msec to 470 msec, H1-H2 decreased from 820 to 540 msec (fig. 2, left) and then increased slightly, closely resembling the type 1 curve previously described by Wit et al. At an A1-A2 interval of 460 msec, one of two H1-H2 responses was noted, either fast (820 msec) (fig. 1B) or slow (880 msec) (fig. 1C). At coupling intervals of 450 msec or less, only slow responses were defined (fig. 1D and E, and fig. 2). The limiting factor for A-V conduction was the A-V nodal ERP (fig. 1F).

Examination of the graph (fig. 2) suggests two curves, each representing an A-V nodal pathway, with its own ERP and FRP. Curve fitting analysis supported this hypothesis. Analysis of the points coinciding with the curve representing the fast pathway (A1-A2 between 770 and 460 msec) fit a second degree polynomial. The second set of points (A1-A2 of 460-340 msec) were all above the 95% confidence limits of predicted values for the "fast" pathway curve.

The ERP of the fast pathway may be defined as 450 msec, since coupling intervals of this magnitude and less do not conduct via this pathway. The FRP of this pathway, defined as the shortest H1-H2 on this curve, is 540 msec. Similar determinations for the slow pathway reveal an ERP of 340 msec and an FRP of 820 msec.

Conduction velocities of the two A-V nodal pathways can also be examined by plotting A2-H2 intervals against A1-A2 coupling intervals (fig. 2, right). Two groupings of A2-H2 intervals are discernible with increasing prematurity of A2.

Echo beats were elicited with every atrial test stimulus conducted via the slow pathway (fig. 1C-E, fig. 2). Thus, a clearly defined echo zone could be delineated coinciding with the slow pathway curve. When antegrade block occurred in the fast
pathway, the impulse propagated through the slow pathway and returned to the atrium via the fast pathway. Two episodes of PSVT were induced during premature atrial stimulation (fig. 1E). These were reentrant since the preceding A-H interval was prolonged, the premature beat was located in the echo zone, and timed single atrial premature impulses could terminate the tachycardia.5-8

Wenckebach periods proximal to the His bundle recording site were noted at a paced rate of 108 beats/min. These were both 3:2 and 4:3 (fig. 3). The latter did not demonstrate typical Wenckebach periodicity in that the largest increment occurred in the last conducted beat of the group. The A-H intervals in a 4:3 sequence were as follows: 80 msec, 150 msec, 510 msec and the fourth atrial stimulus was blocked proximal to the His bundle recording site (fig. 3). The sudden increase in A-H interval is consistent with dual A-V pathways. We would postulate that during the first beats of the Wenckebach period, both pathways are antegrade invaded by the atrial impulse. The fast pathway will determine the A-H interval. The slow pathway will only be apparent if the fast pathway fails. The expected atrial echo is concealed because of the next paced atrial beat.

Comment: In this case, dual A-V nodal pathways were suggested by demonstration of two A-V nodal refractory periods and conduction velocities. One pathway had faster conduction and a longer ERP, and the other had slow conduction and a shorter ERP. Echo beats were seen when the fast pathway failed. Another manifestation of dual pathways appeared to be the atypical Wenckebach periods.
Case 1: Dual A-V nodal refractory periods and conduction times. Premature atrial coupling to the patient's sinus rate. $A_1-A_2$ in the abscissa is plotted as a function of the $H_1-H_2$ responses in the ordinate (left panel). Numerical values on the top indicate the effective and functional refractory periods (ERP and FRP) of the fast and slow pathways expressed in msec. CL indicates basic drive cycle length. Filled circles (●) indicate responses to atrial premature beats introduced at varying prematurity during the basic cycle length, empty circles (○) indicate atrial premature beats that resulted in echo beats. At a coupling interval of 460 msec two $H_1-H_2$ responses (left panel) and A-V nodal conduction times (right panel) can be observed. Further decrease in prematurity results in a second curve.

Figure 3

Case 1: Atypical Wenckebach period during atrial pacing. Atrial pacing at a rate of 108 beats/min (CL of 555 msec). (Top) Electrocardiographic lead V$_1$ (ECG), His bundle electrograms (HBE), high atrial electrogram (HAE). Atrial stimuli are indicated by arrow. His bundle depolarizations are labeled H. A-H intervals are expressed in msec. (Bottom) Atrial (A), A-V nodal (A-V) and ventricular (V) conduction. Solid lines reflect fast pathway and dotted line slow pathway. R-R intervals are expressed in msec. 3:2 and 4:3 Wenckebach cycles are displayed. The first two beats engage both the fast and slow pathways and arrive at the His bundle via fast pathway. The third beat of the 3:2 sequence is blocked in both pathways. The third beat of the 4:3 sequence is blocked in the fast pathway and conducted antegradely through the slow pathway. Retrograde conduction through the fast pathway is concealed by the subsequent atrial stimulus. The fourth beat is blocked in both pathways.
An alternative explanation for atypical Wenckebach phenomena would be concealed reentry of the beat preceding the sudden change in A-H interval. PSVT was reentrant, utilizing the slow pathway as antegrade, and the fast pathway as the retrograde route.

Case 2

Electrophysiological Studies: Intervals were as follows during sinus rhythm: P-A 20 msec, A-H 80 msec, H-V 40 msec. With atrial pacing at a cycle length of 667 msec, progressive shortening of the H1-H2 intervals was noted as A1-A2 coupling intervals were shortened, until an A1-A2 interval of 315 msec was reached. At this point, H1-H2 was 425 msec. At an A1-A2 interval of 305 msec, sudden prolongation of H1-H2 to 515 msec was noted. At all A1-A2 intervals less than 305 msec, only long H1-H2 responses were noted (fig. 4, left). Atrial echoes were noted at A1-A2 intervals of 275, 270, and 265 msec.

Examination of the graph (fig. 4, left) suggests two pathways. Curve fitting analysis, identical to that described in the first case, supported this premise. The fast pathway has an ERP of 305 msec and a FRP of 415 msec; the slow pathway has an ERP of less than 265 msec (the limiting factor for conduction was the atrial ERP) and an FRP of 495 msec.

A2-H2 increased in the expected manner as A1-A2 intervals were shortened (fig. 4, right). At an A1-A2 of 315 msec, the A2-H2 interval was 185 msec. At an A1-A2 of 305 msec, A2-H2 interval suddenly increased to 295 msec. After this, A2-H2 intervals continued to increase as A1-A2 was further shortened.

When the premature atrial beats were elicited by coupling stimuli to the patient sinus rhythm (750 msec cycle length) no break in the H1-H2 or A2-H2 curves could be demonstrated and no echo beats were seen.

Comment: In this patient, dual A-V nodal pathways were suggested by demonstration of two conduction times and refractory periods. These findings were demonstrated at a cycle length of 667 msec. There was no evidence of dual pathways during sinus rhythm (cycle length of 750 msec). The ability to demonstrate dual pathways in this patient at shorter cycle length could reflect a differential response of slow and fast pathways to change in rate. One could postulate that at longer cycle lengths, slow pathway ERP was shorter than fast pathway ERP, thus preventing demonstration of dual pathways with the extra-stimulus technique. With increase in rate, lengthening of the fast pathway ERP relative to the slow pathway ERP (increase of fast pathway ERP and/or decrease of slow pathway ERP) could allow demonstration of
dual pathways. It is possible that the spontaneous episodes of PSVT in this patient occur with sinus rates of 90 or greater (cycle length of 667 or less).

Discussion

Dual Pathways: Moe, Preston and Burlington,4 studying the effect of premature atrial stimulation on A-V transmission in dogs, plotted atrial impulses (A1-A2) against ventricular responses (V1-V2). They described three types of curves, all of which showed a linear decrease of V1-V2 as A1-A2 was shortened until reaching a minimal V1-V2, where further decrease in the prematurity of the atrial beat resulted in one of the following responses: a) no change in V1-V2 interval up to the earliest propagated atrial beat (type A curve); b) an abrupt increase in V1-V2 to a new level which remained constant despite decreasing A1-A2 intervals (type B curve); c) gradual increase in V1-V2 after reaching the minimal V1-V2 interval (type C curve). Type B and C curves were felt to reflect two different A-V nodal pathways with different refractoriness and conduction velocities.

Hoffman et al.,13 using the extra-stimulus method in dogs, recorded impulse propagation at several sites in the conduction system and offered the following interpretation of Moe’s curves. Type A curves could be seen in those experiments where the A-V node had the longest functional refractory period of the A-V conduction system. Type B curves reflected delays of impulse propagation in the peripheral Purkinje fibers. Type C curves reflected delays of impulse propagation in the A-V node. Wit et al.,11 studying the propagation of premature atrial responses in humans found similar curves. The type B curve was characterized by a sudden increase in ventricular response (V1-V2 interval) while H1-H2 remained smooth, reflecting a delay in conduction localized to the His-Purkinje system (H-V interval). A break in the H1-H2 curve has only been described in one patient, a child with two ranges of P-R intervals.5 The electrophysiologic findings in the latter case supported the existence of dual A-V nodal pathways, with two A-V nodal conduction times and refractory periods.

The curves described in this report also show sudden increase in H1-H2 interval with small decreases in the prematurity of the atrial impulse. This occurred without change in intra-atrial conduction of H-V interval, suggesting a site of delay in the A-V node.

The dual pathways demonstrated in this study could reflect two anatomically definable pathways within or around the A-V node or could be secondary to functional longitudinal dissociation.

Echo Phenomena: White,14 Barker, Wilson and Johnston,15 Scherf,16 Katz and Pick,17 and Kistin18 have postulated that longitudinal dissociation of the A-V node explains the occurrence of atrial echoes, ventricular echoes, and reentrant tachycardia. Mendez et al. provided experimental evidence for this.19 A premature impulse blocked in a fast pathway could conduct via a slow pathway and return to the chamber of origin via the originally failed fast pathway.

Schulenburg and Durrer,20 utilizing premature ventricular stimulation in two patients, demonstrated sudden increases in retrograde conduction times at critical coupling intervals. This sudden increase was accompanied by ventricular echo beats. The genesis of the ventricular echoes was attributed to dual conduction pathways. However, without His bundle electrograms, it was not possible to localize the site of sudden increase. In our cases, A-V nodal echoes occurred only with beats conducted antegradely through the slow pathway. This suggested that failure of the fast pathway was necessary for the echo phenomenon, allowing it to be available for retrograde propagation back to the atria.

Paroxysmal Supraventricular Tachycardia: Recent studies6-8 have shown that PSVT can be induced and terminated by single atrial premature beats failing within a specific portion of the A-V nodal relative refractory period. Goldreyer and Damato6 pointed out that it is the critical A-V nodal delay (A-H) of the premature beat, rather than its coupling interval, that determines whether echo beats will result. This suggested that PSVT in man is frequently caused by A-V nodal reentry.

Reentry can imply “reflection” of the impulse through a single pathway, if the impulse is delayed enough at one point so that it can reenter the original pathway.10 Previously published A-V refractory period curves in patients with reentrant PSVT show smooth curves without sudden increases of A-V nodal conduction time simultaneous with the occurrence of the echo phenomenon.8 These earlier cases are consistent with the “reflection” mechanism. The critical A-H interval necessary for induction of reentry in these cases, would be equal to the amount of A-V nodal delay necessary to allow “reflection.”

Reentry could also imply either anatomic or functional dissociation into dual pathways, with
unidirectional block in one pathway, which then becomes available for retrograde propagation. The two cases in this report are consistent with this mechanism for A-V nodal reentrance. In our patients, the initiation of PSVT appeared to be dependent upon the premature impulse occurring at or less than the fast pathway ERP. The critical A-H for induction of reentry in these cases would reflect slow pathway conduction time.

References
10. CRANEFIELD PF, WIT AL, HOFFMAN BF: Genesis of cardiac arrhythmias. Circulation 47: 190, 1973
Demonstration of Dual A-V Nodal Pathways in Patients with Paroxysmal Supraventricular Tachycardia

PABLO DENES, DELON WU, RAMESH C. DHINGRA, RUBEN CHUQUIMIA and KENNETH M. ROSEN

_Circulation_. 1973;48:549-555
doi: 10.1161/01.CIR.48.3.549

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/48/3/549

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/