The Determinants of Atrioventricular Nodal Re-entrance with Premature Atrial Stimulation in Patients with Dual A-V Nodal Pathways

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SUMMARY In patients with dual atrioventricular (A-V) nodal pathways, atrial extrastimulus testing induces either no echoes, single atrial echoes (Ae), or repetitive re-entrance (repetitive atrial and ventricular beating). We examined the fast and slow pathways properties in 38 patients with dual pathways in order to delineate the determinants of re-entrance.

Seventeen patients had no Ae. Of these, six had no V-A conduction and 11, intact V-A conduction. The mean paced ventricular cycle length producing retrograde V-A block (VABCL) in this group (a measure of retrograde fast pathway refractoriness) was 552 ± 32 msec (mean ± SEM; 10 pts). In contrast, all 21 patients with Ae had intact V-A conduction with mean VABCL of 382 ± 21 msec (14 pts) (P < 0.05).

Repetitive re-entrance occurred only when Ae conducted to the ventricles. Seven patients had only single Ae. The mean paced atrial cycle length producing Wenckebach periodicity (CLAWP) in this group (a measure of antegrade slow pathway refractoriness) was 490 ± 31 msec (5 pts). Fourteen patients had repetitive re-entrance. The mean CLAWP in this group was 399 ± 18 msec (8 pts) (P < 0.05).

In conclusion, our results suggest that in patients with dual pathway, the occurrence of single or repetitive re-entry is dependent upon measurable slow and fast pathway properties.

DISCONTINUOUS ATRIOVENTRICULAR (A-V) NODAL CONDUCTION CURVES, suggestive of dual A-V nodal pathways, are found in most patients with A-V nodal re-entrant paroxysmal supraventricular tachycardia.1-4 Similar conduction curves are occasionally found in patients without paroxysmal tachycardia.4-6 The determinants of when A-V nodal re-entrance occurs in a patient with dual A-V nodal pathways have not been previously examined.

In this report we examine patients with dual A-V nodal pathways and compare patients with and without A-V nodal re-entrance. The fast and slow pathway properties allowing induction of single echoes, as well as repetitive A-V nodal re-entrance, are defined.

Methods

All patients undergoing electrophysiological studies in our laboratories between July 1971 and October 1976 were reviewed. Criteria for inclusion in the present study were: 1) assessment of antegrade conduction with incremental atrial pacing and atrial extrastimulus techniques; 2) assessment of retrograde conduction with incremental ventricular pacing; 3) presence of dual A-V nodal pathways, as diagnosed by the presence of discontinuous conduction curves;1-4 4) absence of either manifest or concealed pre-excitation.13-15

Thirty-eight patients fulfilled the above criteria: 27 males and 11 females with ages ranging from 18 to 82 years. The indications for electrophysiological studies in these 38 patients were as follows: 1) documented paroxysmal supraventricular tachycardia (16 pts); 2) a history of palpitations without documented supraventricular tachycardia (9 pts); 3) presence of intraventricular conduction defect (9 pts); 4) syncope or dizziness (4 pts).

Electrophysiological Studies

Electrophysiological studies were performed in the postabsorptive, nonsedated state. Informed consent was obtained prior to study. His bundle electrograms were obtained with a tripolar catheter placed across the tricuspid valve.19 A hexapolar catheter was also introduced via an antecubital vessel. The two distal electrodes of this latter catheter were placed at the right ventricular apex for ventricular pacing. The proximal four electrodes were used for high right atrial stimulation and recording. The hexapolar catheter was also used for recording of coronary sinus electrograms during induced supraventricular tachycardia. Multiple electrocardiographic leads and intracardiac electrograms were simultaneously recorded on a multichannel oscilloscopic photographic recorder (Electronics for Medicine DR-16, White Plains, New York) at paper speeds of 100 mm/sec. Stimuli were approximately twice diastolic threshold and provided by a programmable digital pulse generator (manufactured by M. Bloom, Philadelphia, Pa.).

The atria were paced at a rate slightly faster than spontaneous sinus rate and then at increasing rates in 10 beats/min increments until one or more of the following were demonstrated: 1) A-V nodal Wenckebach periodicity, 2) induction of echoes and/or paroxysmal supraventricular tachycardia, 3) achievement of a maximum paced rate of 200/min (cycle length of 300 msec). Antegrade refractory periods and echo zones were measured utilizing atrial extrastimulus technique during sinus rhythm at one or more atrial driven cycle lengths.1-6

The ventricles were paced at a rate slightly above sinus rate and then in 10 beats/min increments until one or more of the following were demonstrated: 1) ventriculo-atrial block; 2) achievement of a maximum rate of 140 to 200/min.
(cycle length of 429 to 300 msec). The maximum rate achieved was determined by the clinical status of the patient.

Definitions

Conduction intervals and refractory periods were defined as previously described.\textsuperscript{1-12} Dual A-V nodal pathway were diagnosed when discontinuous $A_1$-$A_2$, $H_1$-$H_2$ curves were demonstrated with atrial extrastimulus technique.\textsuperscript{1-12} Slow and fast pathway conduction times were defined as previously described.\textsuperscript{1-14} The longest slow pathway conduction time (max SPAH) was defined during atrial extrastimulus testing and was the longest $A_1$-$H_2$ obtained at all the tested $A_2$-$A_3$ coupling intervals.

Patients were subdivided into three groups, based upon response to atrial extrastimulus testing (figs. 1 and 2). Group A consisted of 17 patients with dual pathways, but without echoes, at all tested coupling intervals (figs. 1 and 2A). Group B consisted of seven patients with dual pathways and only single A-V nodal re-entrant atrial echoes ($A_{e}$), at one or more tested coupling intervals (figs. 1 and 2B). Group C consisted of 14 dual pathway patients with repetitive re-entrance, at one or more tested coupling intervals (figs. 1 and 2C). These patients all had an initial atrial echo with subsequent ventricular beat. In addition, all group C patients had induced paroxysmal supraventricular tachycardia (PSVT), as defined by the presence of three or more pairs of sequential A-V nodal re-entrant atrial and ventricular beats.

In all cases with echoes, electrophysiological mechanisms other than A-V nodal re-entry (i.e., retrogradely conducting extranodal pathways, sinus or atrial re-entry) were excluded, utilizing previously reported criteria.\textsuperscript{13-18}

Assumptions

The analysis presented in this study is based upon a number of assumptions, supported by previously reported work from our and other laboratories (fig. 3).\textsuperscript{1-12} Discontinuous A-V nodal conduction curves reflect the presence of dual A-V nodal pathways.\textsuperscript{1-12} This discontinuity reflects achievement of a fast pathway refractory period with antegrade conduc-

\begin{figure}[h]
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\caption{Schematic representation of patient grouping based upon responses to atrial extrastimulus testing. $A_1$ = atrium; $AVN$ = A-V node, $V$ = ventricle. $A_1$ is the basic atrial drive, $A_2$ is the atrial response to the extrastimulus. $A_3$ represents the atrial echo and $V_e$, the repetitive ventricular response.}
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\begin{figure}[h]
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\includegraphics[width=\textwidth]{figure2.png}
\caption{Electrophysiological demonstration of the three patient groups. Shown in each panel are electrocardiographic lead $V_1$, high right atrial electrogram (HRA), coronary sinus electrograms (CS) and His bundle electrograms (HBE). Paper speed is 100 mm per second and timelines are at one second on this and subsequent illustrations. In all panels, two sinus or driven impulses ($A_{e}$) are shown followed by an atrial extrastimulus and its response ($A_{e}$). Panel A, from patient 7, corresponds to figure 1 (top) and shows absence of echo response at close coupling intervals. Panel B, from patient 19, corresponds to figure 1 (middle) and shows a single atrial echo ($A_{e}$). Panel C, from patient 31, corresponds to figure 1 (bottom) and shows PSVT induction with sequential atrial and ventricular beats ($V_e$).}
\end{figure}
tion proceeding via a slow pathway. A-V nodal re-entry in patients with discontinuous conduction curves results from antegrade block in the fast pathway with antegrade conduction via the slow pathway. With sufficient antegrade slow pathway delay, the fast pathway recovers for retrograde conduction with resultant atrial echo. A-V nodal re-entrant PSVT usually results from a sustained circus movement involving fast and slow A-V nodal pathways. During PSVT antegrade conduction follows the slow pathway and retrograde conduction, a fast pathway (fig. 3A).

It is assumed that antegrade slow pathway properties can be assessed by noting the response to incremental atrial pacing at fast rates (fig. 3B). In patients with discontinuous conduction curves (dual pathways), there is a sudden large increment in A-V nodal conduction time (A-H interval) at a critical paced atrial cycle length, suggesting failure of a fast A-V nodal pathway and antegrade conduction via a slow pathway. Further shortening of atrial paced cycle length produces A-V nodal Wenckebach periodicity. This presumably reflects failure of the slow pathway and is thus related to slow pathway refractoriness (fatigue of the slow pathway due to repetitive impulse propagation).

It is assumed that retrograde fast pathway conduction can be assessed by noting the response to rapid ventricular pacing (fig. 3C). In most dual pathway patients, the fast pathway has the shortest retrograde refractory period (Wu et al., unpublished data). Thus the ventricular paced cycle length producing failure of ventriculo-atrial conduction is a measure of retrograde fast pathway refractoriness.

It should be noted that the above assumptions, as well as the data reported in this study, are based upon observations of His bundle electrograms and responses to electrical stimulation of atria and ventricles. More precise proof of the determinants of conduction in various portions of the A-V transmission system will have to await further experimental work.

### Results

#### Determinants of the Atrial Echo

We hypothesized that retrograde fast pathway properties would determine whether or not patients could have atrial echoes. Specifically, the occurrence of ventriculo-atrial conduction at short cycle lengths should allow the echo phenomenon. To test this hypothesis, retrograde conduction was examined in patients without echoes (group A), and those patients with echoes (groups B and C) (table 1).

There were 17 patients without A-V nodal re-entrant echoes during atrial extrasystolic testing (group A) (figs. 1 and 2A). Of these 17, six (35%) had no V-A conduction at any tested ventricular paced cycle length (fig. 4A). The remaining 11 patients (65%) had ability for retrograde conduction. Of the latter 11 patients, ten manifested retrograde ventriculo-atrial block at a paced ventricular cycle length of 375 to 667 (mean ± SEM, 552 ± 32 msec) (fig. 4B). One patient maintained intact V-A conduction up to a paced cycle length of 430 msec and was not tested at shorter cycle lengths.

#### Table 1. Electrophysiologic Data in Groups A, B and C

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Abbreviations: max SPAH = maximal slow pathway A-H; VABCL = cycle length of pacing-induced ventriculo-atrial block; AWPCCL = cycle length of antegrade Wenckebach periodicity; PSVT = paroxysmal supraventricular tachycardia; (R) = interruption of tachycardia in the antegrade limb of the re-entrant circuit; (A) = interruption of tachycardia in the retrograde limb of the re-entrant circuit; = no; + = yes.
Maximum slow pathway A-H was also evaluated in the 17 patients without echoes, since this might be a variable in determining whether or not enough antegrade slow pathway delay existed for the fast pathway to recover for retrograde conduction. Maximum slow pathway A-H ranged from 280 to 620 (388 ± 27 msec) in this group.

Twenty-one patients had one or more echoes (groups B and C) (figs. 1, 2B and C). All 21 (100%) had ability for ventricular-atrial conduction (fig. 4C). In seven of the 21 patients (33%), ventriculo-atrial conduction was present at the shortest tested ventricular paced cycle lengths of 300 to 400 msec (342 ± 13 msec) (fig. 4C). Shorter cycle lengths were not tested in this group. The cycle length inducing retrograde block in the remaining 14 patients ranged from 300 to 600 msec (382 ± 21 msec). Maximum slow pathway A-H in the 21 (groups B and C) patients ranged from 260 to 670 msec (399 ± 22 msec).

The comparison of patients without echoes (group A) and those with echoes (groups B and C) is presented in table 2. Group B and C patients had a significantly higher incidence of ability for retrograde conduction. In addition, the ventricular paced cycle length producing retrograde block was significantly shorter in groups B and C. There was no significant difference in maximum slow pathway A-H, when comparing patients with and without echoes.

Determinants of Repetitive Re-entrance

An early ventricular beat only occurred in those patients having an initial atrial echo with atrial extrastimulus testing (table 1). We hypothesized that antegrade slow pathway properties should determine whether or not the ventricular beat would occur. Specifically, the ability for A-V conduction at short cycle lengths should allow occurrence of ventricular beating. This hypothesis was tested by comparing the atrial paced cycle length producing Wenckebach periodicity in patients with single atrial echoes (group B) (figs. 1 and 2B) and patients with repetitive re-entrance (group C) (figs. 1 and 2C).

Of the seven patients with only atrial echoes, five patients manifested antegrade Wenckebach periodicity at a paced atrial cycle length of 400 to 545 msec (490 ± 31 msec) (fig. 5). In the remaining two, Wenckebach periodicity was not achieved because of the occurrence of pacing-induced atrial echoes.

In eight of the 14 patients with repetitive re-entrance, antegrade Wenckebach periodicity occurred at paced cycle lengths of 316 to 462 msec (399 ± 18 msec) (fig. 6). In the remaining six patients, Wenckebach periodicity was not achieved because of atrial pacing-induced PSVT.

When groups B and C were compared, the cycle length producing antegrade Wenckebach periodicity was significantly shorter (P < 0.05) in group C.

Determinants of Sustained PSVT

All 14 patients in group C had induced PSVT during atrial extrastimulus testing (figs. 1 and 2C; table 1). In eight of the patients, induced PSVT was self sustaining, necessitating conversion with single or double atrial or ventricular extrastimuli. In six group C patients, induced PSVT was self terminating (within 30 seconds of induction). In two of the patients with self terminating PSVT, termination always occurred following the QRS (retrograde block in the fast pathway). In four of the patients with self terminating PSVT, termination always ended with the atrial echo (antegrade

![Table 2. Fast and Slow Pathway Properties](data:image/png;base64,iVBORw0KGgoAAAANSUhEUgAAAIQAAADhCAYAAAD97j9KAAAgAElEQVR42u1QZT2Rg4gAAAAASUVORK5CYII=)

See table 1 for abbreviations.
block within the slow pathway). In neither of the two patients with self termination in the fast pathway (retrograde limb of the circus), was ventriculo-atrial conduction observed at paced ventricular cycle lengths of less than 400 msec. In contrast, intact ventriculo-atrial conduction was observed at paced ventricular cycle lengths of less than 400 msec in seven of the eight patients (88%) with sustained PSVT.

In none of the four patients with self termination in the slow pathway (antegrade limb of the circus) was intact A-V conduction noted at paced atrial cycle lengths of 350 msec or less. In contrast, intact A-V conduction was observed at paced atrial cycle lengths of less than 350 msec in six of eight patients (75%) with sustained PSVT.

Although the above observations have been made in small numbers of patients (precluding meaningful statistical analysis), it appears that ability for sequential antegrade slow pathway and retrograde fast pathway conduction at short cycle lengths are electrophysiological properties pre-disposing to sustained PSVT in patients with dual A-V nodal pathways.

Discussion

Most patients with A-V nodal re-entrant PSVT have discontinuous conduction curves suggestive of dual A-V nodal pathways. With atrial extrastimulus testing at close coupling intervals, patients with dual A-V nodal pathways may demonstrate a number of responses, these being induction of no echoes, induction of single atrial echoes, induction of nonsustained PSVT (nonsustained repetitive re-entrance), and induction of sustained PSVT (figs. 1 and 2).

The following hypothesis may explain the presence of discontinuous A-V nodal conduction curves and the different types of responses to atrial extrastimulus testing. In patients with dual A-V nodal pathways, one can consider the A-V node to be dissociated (functionally or anatomically) into fast and slow pathways. The former pathway has a
longer refractory period than the latter, so that at close coupling intervals, antegrade conduction occurs via the slow pathway (fig. 1, top panel). With enough antegrade slow pathway delay, the fast pathway recovers for retrograde conduction with resultant atrial echo (fig. 1, middle panel). For this atrial echo to occur, there must be a distal final common pathway by which the antegrade impulse in the slow pathway can be transmitted to the fast pathway for retrograde conduction. Once retrograde fast pathway conduction occurs (with resultant atrial echo), a ventricular response may occur if the retrograde impulse in the fast pathway can re-enter the slow pathway for antegrade conduction. The slow pathway must be able to conduct a second sequential impulse (the first impulse being that in response to the extrastimulus) for the ventricular response to occur (fig. 1, lower panel). Sustained PSVT results from a circus movement involving sequential retrograde fast pathway, and antegrade slow pathway conduction (utilizing proximal and distal final common pathways).

In the present study, retrograde fast pathway properties were evaluated to determine whether these related to the occurrence or lack of occurrence of atrial echoes with atrial extrastimulus testing. Specifically, the ventricular paced cycle length producing ventriculo-atrial block was examined. Measurement of fast pathway retrograde refractoriness, as measured with ventricular extrastimulus technique, was not utilized for several reasons, these being: 1) the difficulty in reliably recording retrograde His bundle potentials during ventricular stimulation, a necessity for accurate measurement of fast pathway refractoriness; 2) the probability of limitation of measurement of retrograde conduction by ventricular limited refractoriness, or retrograde His-Purkinje refractoriness.

Ventriculo-atrial conduction had to be present for the echo phenomenon to occur. No patient who had absent V-A conduction (at all tested cycle lengths) had atrial echoes. This lack of ability for retrograde conduction is unexplained at present. In addition, the quality of V-A conduction also determined whether or not echoes could occur. Even if patients had intact V-A conduction, the development of V-A block at relatively long ventricular paced cycle lengths mitigated against the occurrence of atrial echoes with atrial extrastimulus testing. In contrast, most patients with echoes had intact V-A conduction at relatively short cycle lengths. These observations suggested that the presence and quality of V-A conduction over the fast pathway determined whether or not atrial echoes could occur. Good or excellent V-A retrograde fast pathway conduction allowed the occurrence of atrial echoes.

One other possible determinant of whether or not atrial echoes might occur was also examined, this being slow pathway conduction time. One could hypothesize that the ability for very long slow pathway conduction might predispose to the echo phenomenon, since this would enable the fast pathway to have time to recover for retrograde conduction. This did not appear to be a major variable when comparing patients with and without atrial echoes, since maximum slow pathway A-H was not significantly different between the two groups.

The determinants of repetitive re-entrance were also examined. It was first noted that repetitive re-entrance (atrial and ventricular responses) occurred with atrial extrastimulus testing only in those patients who had atrial echoes. Thus, we compared patients with only atrial echoes to those with repetitive re-entrance. The atrial paced cycle length producing antegrade Wenckebach periodicity was used as a measure of antegrade slow pathway refractoriness. As hypothesized, the cycle length producing antegrade Wenckebach periodicity was less in those patients who had repetitive re-entrance than in those with just atrial echoes. It was noted that there was overlap between groups, so that knowing the atrial paced cycle length producing Wenckebach periodicity could not precisely predict which patients would or would not have ventricular echoes.

Induced PSVT, as defined by the presence of three pairs of sequential A-V nodal re-entrant atrial and ventricular beats, occurred in all patients with one demonstrated pair of A-V nodal re-entrant atrial and ventricular beats. Thus, fast and slow pathway properties determining whether or not PSVT would occur were identical to those predicting whether or not repetitive re-entrance would occur. However, the ability to induce sustained (non self-terminating) PSVT did appear to relate to measurable fast and slow pathway properties. Induction of sustained PSVT was usually associated with both ability for retrograde conduction at paced ventricular cycle lengths of less than 400 msec, and ability for intact antegrade conduction at atrial paced cycle lengths of less than 350 msec.

In regard to the question of whether final common pathway properties were a determinant of the echo phenomena, we can make only limited observations. Since all patients with ability for retrograde fast pathway conduction at cycle lengths of 400 msec or less had atrial echoes, this group appears to have a distal common pathway. The present study does not allow us to comment on the location of the distal common pathway.

The proximal common pathway is less susceptible to scrutiny. Because of the overlap of patients in regard to slow pathway properties (comparing those with only atrial echoes and those with both atrial and ventricular echoes) the possibility exists that this overlap reflects unmeasured properties of a proximal final common pathway. This study sheds no light on whether the atrium is part of the proximal common pathway.

The present study is limited to the determinants of A-V nodal re-entrance with atrial extrastimulus technique. Our data in the presently reported group of patients was not adequate to evaluate the occurrence and determinants of A-V nodal re-entry with premature ventricular stimulation. There does appear to be another type of A-V nodal re-entry elicited by ventricular premature stimulation, in which retrograde conduction is via the slow A-V nodal pathway, and antegrade conduction via the fast pathway. This suggests the possibility of two types of A-V nodal reciprocation: one type induced from the atria, and the other induced from the ventricles.

Clinical Implications

In patients with dual A-V nodal pathways, the occurrence of single re-entry, repetitive re-entry or PSVT appears to be not a random event, but dependent upon measurable slow and fast pathway properties. The predictability of
responses allows a more rational understanding of why some dual pathway patients do or do not have clinically significant PSVT.

References

The determinants of atrioventricular nodal re-entrance with premature atrial stimulation in patients with dual A-V nodal pathways.

P Denes, D Wu, F Amat-y-Leon, R Dhingra, C R Wyndham and K M Rosen

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