Demonstration of Dual Atrioventricular Nodal Pathways Utilizing a Ventricular Extrastimulus in Patients with Atrioventricular Nodal Re-entrant Paroxysmal Supraventricular Tachycardia

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SUMMARY

In patients with atrioventricular (A-V) nodal re-entrant paroxysmal supraventricular tachycardia (PSVT), atrial extrastimulus technique frequently reveals discontinuous A₁-A₂, H₁-H₂ curves suggestive of dual A-V nodal pathways. To further test the hypothesis that these curves in fact reflect dual A-V nodal pathways, a ventricular extrastimulus (Vₜ) was coupled either to A₁ at a fixed A₁-A₂ interval which reliably produced an A-V nodal re-entrant atrial echo (E) with a constant A₂-E interval in two patients, or to QRS complex (V) during sustained PSVT with a constant E-E interval in one patient. Three response zones were defined: at longer A₂-Vₜ or V-Vₜ coupling intervals, Vₜ manifested no effect on the timing of E (Zone 1). At closer A₂-Vₜ or V-Vₜ coupling interval, Vₜ conducted to the atrium, shortening the apparent A₂-E or E-E interval (Zone 2).

The ability of Vₜ to preempt control of the atria (Zone 2 response) strongly suggests the presence of dual A-V nodal pathways in these PSVT patients. If only a single pathway were present, Vₜ would of necessity collide with the antegrade impulse and could not reach the atria. The Zone 3 response occurs because of retrograde refractoriness of the fast pathway. Failure of the echo during Zone 3 probably reflects concealed conduction to the fast pathway, or possibly interference in the slow pathway.

Our Laboratory has recently demonstrated discontinuous atrioventricular (A-V) nodal curves (A₁-A₂, H₁-H₂) in a number of patients with A-V nodal re-entrant paroxysmal supraventricular tachycardia (PSVT). We suggested that these curves reflected the occurrence of longitudinal dissociation of the A-V node into fast and slow pathways. We further postulated that block in the fast pathway accompanied by antegrade slow pathway conduction allowed the fast pathway to recover and conduct retrogradely. It was suggested that A-V nodal reentrant PSVT reflected sustained intranodal re-entrance with antegrade slow pathway and retrograde fast pathway conduction.

In this study, we have attempted to support further the hypothesis that discontinuous conduction curves reflect the presence of dual A-V nodal pathways (as opposed to relative refractoriness within a single pathway) utilizing critically timed ventricular extrastimuli. Ventricular extrastimuli were either coupled to atrial extrastimuli, which induced A-V nodal re-entrant atrial echoes, or coupled to QRS complex during sustained PSVT. If only a single pathway were present, ventricular extrastimuli would collide with the antegrade impulse and could not reach the atria. If dual A-V nodal pathways were present, ventricular extrastimuli could be expected to preempt the atria, resetting the expected atrial echo response.

Methods

Three patients with documented recurrent PSVT and discontinuous A-V nodal conduction curves are reported. All three patients met the following electrophysiological criteria for A-V nodal re-entrance: 1) induction of echoes with or without PSVT at a critical coupling interval; 2) induction of echoes with or without PSVT during pacing-induced A-V nodal Wenckebach periods; 3) induction of echoes with or without PSVT related to a critical A-H interval achieved by coupled atrial stimulation or rapid atrial pacing; 4) absence of the echo phenomenon when atrial extrastimuli were blocked proximal to the His bundle; 5) absence of criteria suggesting sinus node re-

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Electrophysiological Studies

Electrophysiological studies were performed with the patients in the postabsorptive, nonsedated state with cardiac drugs discontinued 72 hours prior to study. Informed consent was obtained. A tripolar electrode catheter was placed across the tricuspid valve via a femoral vein for His bundle recording. A hexapolar electrode catheter was introduced via the antecubital vein and advanced into the right ventricle. The distal two electrodes were utilized for right ventricular pacing. The middle two and the proximal two electrodes were positioned against the lateral wall of the high right atrium (close to the sinus node). The middle two electrodes were utilized for pacing and the proximal two electrodes were utilized for recording of high atrial electrograms. A third bipolar electrode catheter with an interelectrode distance of 1 cm was also introduced into the antecubital vein, advanced into the right atrium, and positioned in the coronary sinus for recording of posterior left atrial electrograms. Multiple electrocardiographic leads, high right atrial, coronary sinus, and His bundle electrograms were simultaneously recorded on a multichannel oscilloscopic recorder (Electronics for Medicine DR-16, White Plains, New York) at paper speeds of 100 and 200 mm/sec. Recordings were also stored on an 8 channel tape system to facilitate subsequent analysis. Extrastimuli were delivered via a programmable digital stimulator (manufactured by M. Bloom, Philadelphia, Pa.).

The atria were paced at rates slightly faster than sinus rhythm. The paced rate was then increased at 10 beats/min steps until type I A-V block was noted. The pacemaker was then turned on and off repeatedly and randomly at this rate and at rates slightly above and below in order to delineate the presence or absence of concealed re-entry. A similar protocol was utilized for right ventricular pacing when retrograde properties were studied.

Both antegrade and retrograde refractory periods and echo zones were measured utilizing extrastimulus technique with a test stimulus introduced after every tenth sinus or driven beat. The coupling interval was decreased in 5-20 msec increments. In order to insure the reproducibility of observed phenomena, extrastimuli were repeated several times at critical coupling intervals.

After these measurements, the following special studies were performed: In two patients with induced echoes, but without sustained PSVT, a ventricular extrastimulus (V2) was coupled to the atrial extrastimulus (A2) at a fixed A1-A2 coupling interval, in which A2 always induced an echo (E) with a constant A2-E interval (fig. 1A). In one patient with induced echoes and sustained PSVT, the ventricular extrastimulus was coupled to the QRS complex (V) during PSVT (fig. 1B). In both, the coupling intervals (A2-V or V-V,) were shortened in 5 msec decrements until V, failed to capture the ventricle.

Electrophysiological Definitions

HRAa, CSa, Aa, Hs, and Va were the high right atrial, coronary sinus, low right atrial, His bundle, and ventricular electrograms, respectively, of the sinus or driven beat (Sa). HRA2, CSa, Aa, Hs, and V, were respectively the high right atrial, coronary sinus, low right atrial, His bundle, and ventricular responses to the extrastimulus (S2). V, Hs, Aa, CSa, and HRAa were the ventricular, His bundle, low right atrial, coronary sinus, and high right atrial responses to the ventricular extrastimulus, respectively.

Conduction intervals, refractory periods, antegrade echo zones, and critical A-H intervals were measured and defined as previously described. Retrograde echo zones were defined as a zone of V1-V, intervals in which V, induced a ventricular echo (narrow QRS) with or without PSVT. Critical V-A (or H-A when retrograde H was visible) interval was defined as the shortest V-A (or H-A) interval inducing ventricular echoes with or without PSVT either during ventricular pacing or during coupled ventricular stimulation.

Dual pathway cases were suspected when discontinuous A1-A2, H1-H2, curves were demonstrated by curve-fitting analysis with definition of fast and slow pathways. Functional and effective refractory periods of the fast and slow pathways were defined as previously described.

Results

Case One

The patient was a 63-year-old male with recurrent PSVT. Electrophysiological studies during sinus rhythm revealed an A-H of 109 msec and H-V of 35 msec.

Atrial extrastimuli were coupled at a driving cycle length of 600 msec (fig. 2A). As A1-A2 coupling intervals were decreased from 590 to 390 msec, H1-H2 intervals decreased from 590 to 485 msec. A sudden jump of H1-H2 interval, reflecting a sudden increase of A1-A2 interval, occurred at an A1-A2 interval of 385

Figure 1

Diagrammatic representation of the experimental protocol. At, AV, and V respectively represent the atrium, A-V node, and ventricle. A1 and A2 are the atrial responses to driven and test stimuli. E represents an atrial echo. Aa is the atrial response to the ventricular extrastimulus (V2). Solid lines reflect fast pathway conduction and interrupted lines reflect slow pathway conduction. Zone 1, Zone 2, and Zone 3 are respectively zones of non-reset, reset, and termination. In panel A, V1 is coupled to Aa. In panel B, V2 is coupled to V during an induced episode of paroxysmal supraventricular tachycardia (PSVT).
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Conduction curves in case 1 suggesting dual A-V nodal pathways. In panel A, H1-H2 responses were plotted against A1-A2 coupling intervals. The driving cycle length (CL) was 600 msec. The fast and slow pathway effective refractory periods were respectively 385 and 300 msec. Echoes occurred at A1-A2 between 340 and 310 msec (open circles). In panel B, A2-E (or A2-A8) responses were plotted against A2-V8 coupling intervals, at an A1-A2 of 325 msec, with an atrial driving cycle length (A1-A2) of 600 msec. Zone 1 responses occurred at A2-V8 between 460 and 370 msec. Zone 2 responses occurred at A1-V8 between 365 and 255 msec. Zone 3 responses occurred at A2-V8 between 250 and 150 msec.

msec. At A1-A2 intervals between 385 to 310 msec, H1-H2 intervals increased from 535 to 640 msec. Echoes occurred at A1-A2 intervals of between 340 and 310 msec with achievement of a critical A-H interval of 385 msec or greater (figs. 2A and 3A). Examination of the A1-A8, H1-H2 curve suggested dual A-V nodal pathways with fast and slow pathway effective refractory periods of 385 and 300 msec, respectively. The atrial functional refractory period was 285 msec.

Ventricular extrastimuli were coupled to a ventricular driving cycle length of 600 msec. At V1-V2 coupling intervals between 590 to 220 msec, A1-A2 intervals were between 600 and 425 msec. The ventricular functional refractory period of 220 msec limited V-A conduction. Analysis of V1-V2, A1-A2 curves revealed a smooth continuous curve. Echoes with or without PSVT were not induced.

The nature of the A-V nodal echo phenomenon was studied with a ventricular extrastimulus (V8) following A2 (figs. 2B and 3B-D). Ventricular extrastimuli were delivered at an A1-A2 interval of 325 msec and an atrial driving cycle length (A1-A2) of 600 msec. At this A1-A2 interval, A-V nodal re-entrant atrial echoes always occurred with a mean A2-H2 interval of 400 msec (range 390–410 msec) and a mean A2-E interval (measured from A2 to the onset of atrial echo observed from low right atrial electrogram) of 605 msec (range 585–615 msec); (atrial extrastimuli were delivered 16 times at this coupling interval) (fig. 3A). At A2-V8 intervals between 460 to 370 msec, A2-E (A2-A8) remained unchanged and were between 585 to 615 msec (Zone 1 response) (figs. 2B and 3B). At A2-V8 intervals between 365 to 255 msec, A2-E (A2-A8) intervals progressively shortened from 580 to 520 msec (Zone 2 response) (figs. 2B and 3C). At A2-V8 intervals between 250 to 150 msec, E (or A8) disappeared (Zone 3 response) (figs. 2B and 3D). When V8 was delivered without the presence of A2, V8-A8 interval was always slightly shorter than the corresponding V8-A8 interval when A2 was present.

Comment

In this patient, discontinuous A1-A2, H1-H2 curves suggested that longitudinal dissociation of the A-V node into dual pathways accounted for the occurrence of the echo phenomenon. Delivery of ventricular extrastimuli coupled to A2 disclosed three types of responses (fig. 1A): 1) a zone of nonreset (or Zone 1), in which V8 did not interfere with A-V nodal re-entrant atrial echoes and A2-E interval remained unchanged; 2) a zone of reset (or Zone 2), in which atrial responses (A8) to V8 occurred earlier than the expected echo (shortening of A2-E or A2-A8 intervals); 3) a zone of termination (or Zone 3) in which V8 resulted in no atrial response and A-V nodal re-entrant atrial echoes were eliminated. The presence of Zone 2 response with shortening of A2-E intervals suggests the presence of dual A-V nodal pathways with retrograde conduction along the fast pathway being preempted by V8. If there were only a single pathway, V8 should either not interfere with the re-entrant impulse (Zone 1 response) or collide with the antegrade impulse before the site of reflection (Zone 3 response). V8 should never result in earlier atrial activation (Zone 2 response). Lengthening of V8-A8 intervals with the presence of A2 compared to the corresponding V8-A8 intervals without the presence of A2 can be explained by antegrade concealment to the fast pathway by A2 blocked in this pathway. Inability to demonstrate discontinuous V1-V2, A1-A2 curves and failure to induce echo phenomenon during ventricular extrastimulus technique can be explained by a long retrograde effective refractory period and/or a unidirectional block of the slow pathway.6,8

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Figure 3

Recordings from case I suggesting dual A-V nodal pathways as the mechanism of A-V nodal re-entrance with ventricular extrastimulus coupled to A2. Shown are electrocardiographic lead II, high right atrial electrogram (HRA), and His bundle electrogram (HBE). A1 and H1 are the low right atrial and the His bundle electrograms of the driven beats (S1). A2 and H2 are the low right atrial and His bundle responses to the atrial extrastimulus (S2). E represents an A-V nodal re-entrant atrial echo. V1 and A2 are the ventricular and low right atrial responses to the ventricular extrastimulus (S). Time lines are at one second in this and all subsequent illustrations. Paper speed is 100 mm/sec. Conduction intervals were listed on the top of each panel. The basic A1-A1 driving cycle length was 600 msec. A1-A2 interval was 325 msec. In panel A, V1 was not delivered. A2 induced an A-V nodal re-entrant atrial echo with an A2-E of 605 msec. In panel B, A2-V1 was 400 msec; A2-E (or A2-A2) remained unchanged. In panel C, A2-V1 was 270 msec; A2-A2 was shortened to 520 msec. In panel D, A2-V1 was 250 msec; A2 was not followed by an A3.

Case Two

The patient was a 67-year-old male with recurrent PSVT. Electrophysiological studies during sinus rhythm revealed an A-H of 155 msec and an H-V of 48 msec.

Atrial extrastimuli were coupled to sinus rhythm at a cycle length of 835 msec (fig. 4A). At A1-A2 coupling intervals between 715 to 420 msec, H1-H2 intervals were between 720 and 535 msec. At A1-A2 coupling intervals between 410 and 400 msec, H1-H2 intervals were either between 880 and 840 msec, or between 545 and 520 msec. At A1-A2 coupling intervals between 390 and 365 msec, H1-H2 intervals were between 810 to 935 msec. Examination of the A1-A2, H1-H2 curve suggested dual A-V nodal pathways with the effective refractory periods of fast and slow pathway being 395 and 360 msec, respectively. The echo zone coincided with the total slow pathway curve with a critical A-H interval of 580 msec. Overlapping of the fast and slow pathway curve occurred at A1-A2 intervals between 400 and 410 msec. The atrial functional refractory period was 340 msec.

Ventricular extrastimuli were coupled to a ventricular driving cycle length of 680 msec. As V1-V2 coupling intervals were shortened from 650 to 240 msec, H1-H2 intervals shortened from 650 to 340 msec and A1-A2 shortened from 650 to 440 msec (fig. 5A). Ventricular echoes occurred at V1-V2 coupling intervals less than 370 msec which achieved a critical H2-A2 interval of 225 msec or greater (fig. 5B). The ventricular functional refractory period of 240 msec limited V-A conduction. The V1-V2, A1-A2 curve was continuous and smooth.

The nature of the antegrade echo phenomenon was studied with a ventricular extrastimulus (V3). V4 was delivered at a fixed A1-A3 interval of 380 msec during sinus rhythm (figs. 4B and 6). At this A1-A3 interval, A3 reliably induced A-V nodal re-entrant atrial echoes with a mean A-R-H2 interval of 628 msec (range 580-645 msec), and a mean A2-E interval of 771 msec (range 735-800 msec); tests were repeated six times (fig. 6 A). At A2-V8 coupling intervals greater than 560 msec, A2-E (or A2-A3) intervals remained unchanged and were between 790 and 805 msec (Zone 1 response)
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A-V node was the mechanism of re-entry. Furthermore, the phenomenon observed in figure 6C suggests that under proper conditions, $V_a$ could preempt the fast pathway to depolarize the atria without interfering with the re-entrant impulse. The ladder diagrams in figure 7 demonstrate the postulated mechanism. In panel A, $A_2$ encountered the effective refractory period of the fast pathway and conducted via the slow pathway with occurrence of an atrial echo. In panel B, $V_a$ was delivered, preempting the fast pathway, resulting in an early atrial response ($A_3$). Meanwhile, $V_a$ encountered retrograde block in the distal slow pathway. Despite preempted utilization of the fast pathway and retrograde concealment of the distal slow pathway by $V_a$, antegrade slow pathway delay was sufficient to allow recovery of both the slow and the fast pathway for reactivation. However, $A_2-H_2$ and $A_2-E$ were lengthened slightly, probably due to the concealment of $V_a$. Evidence of retrograde block in the slow pathway was suggested during ventricular extrastimulus studies with demonstration of a continuous $V_1-V_2$ $A_1-A_2$ (or $H_1-H_3$, $A_1-A_2$) curve and occurrence of echo phenomenon, which were always characterized by a relatively short $H_1-H_2$ and a long $A_2-H$ (fig. 5B). We postulate that at a critical $V_1-V_2$ coupling interval, $V_2$ was blocked in the distal slow pathway, while retrograde conduction continued to occur via the fast pathway. Conduction delay in both the fast (retrograde) and slow (antegrade) pathways was sufficient to allow recovery of the previously blocked distal slow pathway for reactivation.

**Case Three**

This was a 24-year-old female with recurrent PSVT. Electrophysiological studies during sinus rhythm revealed an A-H of 60 msec and H-V of 35 msec.

Atrial extra stimuli were coupled to sinus rhythm at a cycle length of 540 msec (fig. 8A). At $A_1-A_2$ coupling intervals between 530 to 300 msec, $H_1-H_2$ intervals were between 560 and 360 msec. At $A_1-A_2$ coupling intervals between 290 and 260 msec, $H_1-H_2$ intervals were either between 375 and 350 msec or between 575 and 550 msec. Echoes and PSVT occurred at $A_1-A_2$ coupling intervals between 290 and 280 msec with a critical A-H interval of 350 msec. The inner limit of the echo zone was limited by early atrial responses provoked by $A_2$ during atrial vulnerability. The A-V nodal effective refractory period was 265 msec and atrial functional refractory period was 250 msec. The discontinuous $A_1-A_2$, $H_1-H_2$ curves suggested dual A-V nodal pathways with fast and slow pathway effective refractory periods of 260 and 265 msec, respectively (fig. 8A). Overlapping of the two pathways was noted.

Ventricular extrastimuli were coupled to a ventricular driving cycle length of 470 msec. As $V_1-V_2$ was

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**Figure 4**

Conduction curves in case 2 suggesting dual A-V nodal pathways. In panel A, $H_1-H_2$ responses were plotted against $A_1-A_2$ coupling intervals at a sinus cycle length of 835 msec. The fast and slow pathway effective refractory periods were respectively 380 and 380 msec. Overlap of fast and slow pathway curve occurred at $A_1-A_2$ between 410 and 400 msec. The echo zone (unfilled circles) coincided with the total slow pathway curve. In panel B, $A_2-E$ (or $A_2-A_3$) responses were plotted against $A_2-V_a$ coupling interval at an $A_1-A_2$ of 380 msec during sinus rhythm at a cycle length of 835 msec. Zone 1 responses occurred at $A_2-V_a$ between 625 and 580 msec. Zone 2 responses occurred at $A_2-V_a$ between 550 and 140 msec. Zone 3 responses occurred at $A_2-V_a$ between 135 and 120 msec.

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**Comment**

Similar to Case One, the presence of Zone 2 responses in this patient with discontinuous $A_1-A_2$, $H_1-H_2$ curve suggested that longitudinal dissociation of the...
Ventricular extrastimuli (V₂) were coupled to QRS complexes during induced episodes of sustained PSVT (induced with atrial extrastimuli) (fig. 8B and 9). During these episodes of PSVT, cycle length was constant at 355 msec and atrial activation occurred simultaneously with ventricular activation. Since low septal atrial electrograms were masked by ventricular electrograms, timing of coronary sinus electrograms (CS) was noted to determine the effects of ventricular extrastimuli. At V-V₂ coupling intervals equal to or greater than 270 msec, CS-CS₂ interval remained unchanged (Zone 1 response) (figs. 8B and 9A). At V-V₂ coupling intervals between 260 and 165 msec, CS-CS₂ intervals were shortened from 350 to 295 msec (Zone 2 response) (figs. 9B and 9C). Zone 3 response could not be defined because the ventricular functional refractory period of 165 msec limited retrograde conduction.

Comment
In this patient with discontinuous A₁-A₂, H₁-H₂ curves, the presence of Zone 2 responses with ventricular extrastimuli coupled during PSVT suggested dual A-V nodal pathways as the mechanism of sustained A-V nodal re-entry. However, the possibility of a retrograde concealed anomalous pathway (Kent bundle) bypassing the normal pathway must be considered. The following evidence mitigates against this possibility: 1) an appropriate increase in V₂-A₂ as V₁-V₂ coupling intervals were decreased, suggesting retrograde conduction via the A-V node; 2) a normal retrograde atrial activation sequence (low septal right atrium, coronary sinus,
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Figure 6

Recordings from case 1 suggesting dual A-V nodal pathways as the mechanism of A-V nodal re-entrance, with ventricular extraventricleus coupled to A2. H2 is the His bundle response to Vn. A1-A2 was 835 msec (sinus rhythm) and A1-A2 was 380 msec. In panel A, Vn was not delivered; A2 was followed by an A-V nodal re-entrant atrial echo with an A2-E of 795 msec. In panel B, A2-V2 was 360 msec; V2-A2 was shortened to 605 msec. In panel C, A2-V2 was 135 msec; V2 was followed by an A2 with an A2-A2 of 490 msec. However, the A2 was followed by a His bundle potential (labeled as H2), QRS complex and atrial echo (E) with an A2-H2 interval of 655 msec and A2-E of 905 msec. In panel D, A2-V2 was 130 msec; V2 was not followed by an A2.

and high right atrium) (fig. 9C); 3) the occurrence of low atrial and coronary sinus activation almost simultaneously with the onset of ventricular activation during PSVT, suggesting that the ventricles could not be necessary links for re-entry.  The inability to demonstrate discontinuous V1-V2, A1-A2 curves and failure to induce echoes and PSVT with ventricular extrastimuli suggested a long retrograde effective re-

Figure 7

Ladder diagrams presenting the postulated mechanisms for figure 6C.

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fractory period and/or unidirectional block of the slow pathway. 6-8

Discussion

The concept of longitudinal dissociation of the A-V node into two pathways with different functional properties has been used to explain a number of electrophysiological phenomena, including discontinuous atrioventricular conduction curves (A1-A2, H1-H2, or A1-A2, V1-V2 curves) demonstrated with atrial extra-stimulus technique, 1-4, 10 and echo phenomenon. 1-12 However, controversies remain. The above mentioned electrophysiological phenomena can be explained with alternative mechanisms. Firstly, inhomogeneity of the A-V node with different degrees of conduction delay and recovery in the proximal and distal A-V node could occur. 29-31 Atrial beats occurring within a critical range of A1-A2 coupling intervals could arrive at the A-V node during its relative refractory period, resulting in sudden prolongation of H1-H2 intervals producing discontinuous conduction curves. Secondly, when an impulse enters an area of depressed conduction, after a considerable delay, the impulse could reflect back to re-excite the proximal conducting tissue. 62 Possibly, reflection could occur in a single pathway without implying longitudinal dissociation.

A useful means of supporting the presence of dual pathways involves utilization of additional extrastimuli. Moe et al., in the canine heart, and Denes et al., in a patient, demonstrated that when a premature atrial beat (Aa) was conducted to the ventricles (or His bundle) via a slow A-V nodal pathway, A2-V2 (or A2-H2) could be shortened by an immediately succeeding additional atrial premature beat (As). 33-36 They suggested that As was conducted via a fast pathway which was still refractory at the time Aa was evoked, and that faster conduction of As preempted the final common pathway and resulted in an earlier activation of the His bundle and ventricles.

The present study provides additional evidence supporting dual A-V nodal pathways as the mechanism of discontinuous A1-A2, H1-H2 curves and echo phenomenon. Demonstration of Zone 2 responses with shortening of A2-E or E-E interval is difficult to explain utilizing a single pathway, since retrograde and antegrade impulses should collide and Vb should
not conduct to the atria. Case 2 further suggests that $V_s$ could activate the atria without abolishing the echo phenomenon. If the antegrade delay in the slow pathway was sufficient, the part of the re-entrant circuit depolarized by $V_s$ could recover for reactivation and the subsequent echo could still occur. Failure to demonstrate discontinuous $V_s$-$V_a$, $A_1$-$A_2$, and $H_1$-$H_2$-$A_1$-$A_2$ curve in this study can be explained by postulating long retrograde effective refractory period and/or unidirectional block of the slow pathway. Evidence supporting this hypothesis was seen in Case 2. During re-entrance in this patient, the slow pathway was always used for antegrade conduction and the fast pathway for retrograde conduction, regardless of whether echoes were induced from atria or ventricle.

Recently, the presence of concealed retrograde bypass tracts has been demonstrated in a number of documented paroxysmal supraventricular tachycardia. Caution should be used in explaining the data obtained from $V_s$ study. Zone 2 responses could occur if a concealed retrograde bypass tract was present. This possibility was excluded in the present study by the fact that all the three patients showed appropriate lengthening of V-A intervals and development of retrograde Wenckebach periodicity during incremental ventricular pacing and appropriate increase in $V_s$-$A_2$ with ventricular extrastimulus testing. In Case 3, we also demonstrated that a normal retrograde atrial activation sequence was present and that the ventricles were not a necessary link for re-entry.

The present study sheds little light on the exact nature of longitudinal dissociation. Zipes suggested that the effect of summation may play a significant role in the demonstration of discontinuous $A_1$-$A_2$, $H_1$-$H_2$ curves. He suggested that at longer $A_1$-$A_2$ coupling intervals, the wavefront of $A_2$ could engage the entire A-V node and result in short $H_1$-$H_2$ intervals (fast pathway). At shorter $A_1$-$A_2$ coupling intervals, block in part of the A-V node could eliminate the effect of summation and result in sudden prolongation of $H_1$-$H_2$ interval (slow pathway). Our $V_s$ study appears to refute this hypothesis, since $V_s$-$E$ or $V_s$-$A_2$ intervals were always consistent with fast pathway conduction, at a time when a part of the A-V node (slow pathway) was engaged in antegrade conduction.

In conclusion, we have presented additional evidence suggesting that longitudinal dissociation of the A-V node into two pathways is the mechanism of discontinuous $A_1$-$A_2$, $H_1$-$H_2$ curves and A-V nodal echo phenomenon (with or without paroxysmal supraventricular tachycardia). The ventricular extrastimulus technique utilized in this study provides a means for the diagnosis of dual A-V nodal pathway reentrant paroxysmal tachycardia. The technique should also differentiate the existence of a concealed retrograde pathway bypassing the A-V node, in which the ventricular extrastimulus would activate the atria without depolarization of the His bundle during an episode of induced echoes with or without paroxysmal supraventricular tachycardia.

References

17. Wit AL, Cranefield PF: Effect of Verapamil on the sinoatrial and atrioventricular nodes of the rabbit and the mechanism by which it arrests reentrant atrioventricular nodal tachycardia. Circ Res 35: 413, 1974


32. CRANEFIELD PF, WIT AL, HOFFMAN BF: Genesis of cardiac arrhythmias. Circulation 47: 190, 1973


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