An Unusual Variety of Atrioventricular Nodal Re-entry due to Retrograde Dual Atrioventricular Nodal Pathways

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SUMMARY Three patients with paroxysmal supraventricular tachycardia (PSVT) had discontinuous ventriculo-atrial conduction curves (V1-V2, A2-A3), suggesting dual A-V nodal pathways. Ventricular echoes occurred simultaneously with sudden increase of V-A interval. These echoes were characterized by retrograde P waves occurring in front of QRS, suggesting utilization of a slow pathway for retrograde conduction and a fast pathway for antegrade conduction. In case one, atropine improved retrograde slow pathway and antegrade fast pathway conduction and made A-V nodal re-entry sustained, resulting in PSVT (with retrograde P in front of the QRS). In cases 2 and 3, atropine markedly shortened retrograde fast pathway refractory period and slightly improved antegrade slow pathway conduction. The discontinuous V1-V2, A2-A3 curves and echoes were no longer demonstrable. However, with improvement of retrograde fast pathway and antegrade slow pathway conduction, A-V nodal re-entrant echoes and PSVT were observed, utilizing the slow pathway for antegrade conduction and the fast pathway for retrograde conduction (P simultaneous with QRS).

In this study of three patients with PSVT, we demonstrate an unusual form of A-V nodal re-entry utilizing an A-V nodal fast pathway for antegrade conduction and an A-V nodal slow pathway for retrograde conduction. All three patients demonstrated retrograde discontinuous conduction curves (V1-V2, A2-A3). The electrophysiological mechanisms of this unusual variety of A-V nodal re-entry are discussed.

Methods

Electrophysiological Studies

Three patients with documented recurrent PSVT were studied. Electrophysiological studies were performed in the nonsedated, supine state. Cardiac medications were discontinued at least 72 hours prior to the study. Informed written consent was obtained. A percutaneously passed tripolar electrode catheter was placed at the tricuspid valve for His bundle recording. A hexapolar electrode catheter was placed at the right ventricular apex via an antecubital vein. The distal two electrodes were utilized for ventricular
stimulation (tip), the middle two electrodes for atrial pacing (10 cm from tip), and the proximal two electrodes for recording of high right atrial electrograms (13 cm from tip). Multiple electrocardiographic leads and intracardiac electrograms were recorded on a multichannel oscilloscopic recorder (Electronics for Medicine DR-16) at paper speeds of 100 and 200 mm/sec. Electrical stimuli were provided by a programmable digital stimulator (manufactured by M. Bloom, Philadelphia, Pa.) and were approximately twice diastolic threshold and 2 msec in duration.

Antegrade and retrograde conduction were evaluated with atrial and ventricular incremental pacing and extrastimulus techniques. In all patients, 0.5 to 1.0 mg atropine was administered intravenously after control study, and measurements were repeated.

Electrophysiological Definitions

HRA₁, A₁, H₁, and V₁ were high right atrial, low septal right atrial, His bundle and ventricular responses, respectively, to the driven stimuli (S₁); HRA₂, A₂, H₂ and V₂ were the high right atrial, low septal right atrial, His bundle, and ventricular responses to the extrastimulus (S₂). HRA₃, A₃, H₃, and V₃ were high right atrial, low septal right atrial, His bundle and ventricular electrogram of single echo beats and also repetitive beats during PSVT.

Conduction intervals, refractory periods, echo zones, and critical A-H interval (for echo and PSVT induction) were measured and defined as previously described. Retrograde echo zones were defined as the zone of V₁-V₂ coupling intervals at which V₂ induced an A-V nodal re-entrant ventricular echo (narrow QRS) with or without sustained PSVT. Critical V-A interval was defined as the shortest V-A interval at which echoes (narrow QRS) were observed during incremental ventricular pacing or coupled ventricular stimulation.

Antegrade dual A-V nodal pathways were diagnosed when discontinuous A₁-A₃, H₁-H₂ curves were demonstrated. The effective refractory periods of the fast and slow pathways were measured and defined as previously described. Retrograde dual pathways were diagnosed when discontinuous V₁-V₂, A₁-A₃ curves were demonstrated (see Results). The curve to the right of the discontinuity reflected a fast pathway, and to the left, a slow pathway. The effective refractory period of the fast pathway was defined as the longest V₁-V₂ at which V₂ was blocked in the fast pathway. The effective refractory period of the slow pathway was defined as the longest V₁-V₂ at which V₂ was blocked in the slow pathway.

Results

Case One

This was a 31-year-old female with documented recurrent PSVT. Spontaneous episodes of PSVT were characterized by retrograde P waves (biphasic in II and negative in III and aVF) occurring in front of the QRS complex during PSVT with a P-R interval of 0.12 sec. Cardiac examination, chest X-ray and resting electrocardiograms were normal. His bundle recordings during sinus rhythm revealed an A-H of 65 msec and an H-V of 38 msec.

Incremental right atrial pacing revealed intact A-V conduction up to a paced rate of 105 beats/min. A-H interval

![Figure 1](http://circ.ahajournals.org/)  
**Figure 1.** Recordings from case 1, showing responses to rapid atrial pacing before and after atropine. Shown are electrocardiographic lead II, right high atrial and His bundle electrograms (HRA and HBE). S, A, and H are stimulus artifacts, atrial and His bundle responses. A₁, H₁, and V₁ are atrial, His bundle and ventricular responses of the echo beat. Time lines are at one second and paper speed at 100 mm/sec in this and subsequent illustrations. A-H intervals are listed. Panel A) Before atropine at an atrial paced heart rate (HR) of 110/min, 2° A-V nodal Wenckebach block was noted. A-H interval increased minimally from 105 to 130 msec prior to block. Panel B) After atropine at an atrial paced rate of 220 beats/min, sustained PSVT was induced upon achieving a critical A-H of 125 msec. The cycle length of PSVT was 400 msec. The PSVT was characterized by an atrial activation (A₁) occurring before His bundle (H₁) and ventricular activations (V₁). During PSVT, A₁-H₁/V₁-A₁ ratio was 0.21 (70/330) and retrograde P waves preceded QRS complexes.
Figure 2. $A_1-A_2, H_1-H_2$ and $A_2-H_2$ curves before and after atropine in case 1. Left) Before atropine, the curves were continuous at a driven cycle length (CL) of 600 msec. $A_2-H_2$ increased from 100 to 130 msec as $A_1-A_2$ was shortened from 590 to 405 msec. $A-V$ nodal effective refractory period was 400 msec. Right) After atropine, the curves were still continuous at a driven cycle length of 500 msec. $A-V$ nodal effective refractory period shortened to 250 msec.

Increased from 80 to 110 msec as the paced rate was increased from 80 to 105 beats/min. Type I second degree A-V nodal block was noted at a paced rate of 110 beats/min.

The maximal differences in A-H intervals during Wenkebach periods was only 25 msec (fig. 1A). Atrial extrastimulus studies at a driven cycle length of 600 msec revealed a continuous $A_1-A_2, H_1-H_2$ curve with an $A-V$ nodal effective refractory period of 400 msec (fig. 2). The maximal $A_2-H_2$ achieved was 130 msec as compared to the basic driven $A_1-H_1$ of 100 msec. Atrioventricular nodal re-entrant echoes were not induced with either atrial incremental pacing or extrastimulus testing.

Incremental ventricular pacing revealed 1:1 V-A conduction up to a paced rate of 110 beats/min. V-A interval increased from 120 to 150 msec as the paced rate was increased from 100 to 110 beats/min. Retrograde Wenkebach periodicity with unexpected sudden increase of V-A interval (from 135 to 350 msec) was noted at a paced rate of 120 beats/min. A-V nodal re-entrant ventricular echoes occurred simultaneously with the sudden increase of V-A interval. Ventricular extrastimulus studies at a driven cycle length of 600 msec revealed a discontinuous $V_1-V_2, A_1-A_2$ curve due to a sudden increase of $V_2-A_2$ interval at $V_1-V_2$ of 480 msec, suggesting retrograde dual A-V nodal pathways (figs. 3A, 3B and 4). The fast and slow pathway retrograde effective refractory periods were respectively 480 and 230 msec. A-V nodal re-entrant ventricular echoes occurred when $V_2$ was conducted via the slow pathway (fig. 3B). The echo zone coincided with the entire slow pathway.

Figure 3. Recordings from case 1, showing induction of $A-V$ nodal re-entrant ventricular echo with ventricular extrastimulus before (panels A and B) and after (panels C and D) atropine. $S_1, A_1,$ and $V_1$ are stimulus artifact, atrial and ventricular responses to the basic driven stimuli. $S_2, A_2,$ and $V_2$ are stimulus artifact, atrial, and ventricular responses to the test stimulus. $V_1-V_2, A_1-A_2$ intervals are listed on top and $V_2-A_2$ at the bottom of each panel. The driven cycle length (CL) was 600 msec before atropine and 500 msec after atropine. Panel A) At $V_1-V_2$ coupling interval of $485$ msec, $V_2-A_2$ was 180 msec and $A_1-A_2, 530$ msec. An echo did not occur. Panel B) At $V_1-V_2$ of $480$ msec, $V_2-A_2$ suddenly increased to $370$ msec and $A_1-A_2, 715$ msec. $A-V$ nodal re-entrant ventricular echo occurred. This echo was characterized by a short $A_2-H_2$ interval of 85 msec. Panel C) At $V_1-V_2$ of $470$ msec, $V_2-A_2$ was 165 msec and $A_1-A_2, 475$ msec. An echo did not occur. Panel D) At $V_1-V_2$ of $460$ msec, $V_2-A_2$ suddenly increased to 370 msec and $A_1-A_2$ to 670 msec. $A-V$ nodal re-entrant PSVT was induced.
curves (zone of $V_1-V_2$ intervals with long $V_2-A_2$) with a critical V-A interval of 360 msec (fig. 4).

Atropine 0.5 mg was administered intravenously and studies were repeated. After atropine, incremental atrial pacing revealed 1:1 A-V conduction up to a paced rate of 210 beats/min. The A-H interval increased from 90 to 120 msec as the paced rate was increased from 120 to 210 beats/min. PSVT was repetitively induced with sudden cessation of atrial pacing at a rate of 220 beats/min. The A-H interval associated with PSVT induction was 125 msec (fig. 1B). Atrial extrastimulus studies at a driven cycle length of 500 msec revealed a continuous $A_1-A_2$, $H_1-H_2$ curve with an A-V nodal effective refractory period of 250 msec (fig. 2) without definition of an echo zone.

Retrograde studies with incremental ventricular pacing revealed 1:1 V-A conduction up to a paced rate of 130 beats/min (increase in V-A interval from 155 to 160 msec). Wenckebach periodicity with unexpected sudden increase of V-A interval occurred at a paced rate of 140 beats/min. Paroxysmal supraventricular tachycardia induction coincided with the sudden increase of V-A interval. Ventricular extrastimulus studies at a driven cycle length of 500 msec revealed a discontinuous $V_1-V_2$, $A_1-A_2$ curve (figs. 3C, 3D and 4). The effective refractory period of the fast pathway was 440 msec and the slow pathway less than 220 msec. Paroxysmal supraventricular tachycardia induction occurred throughout the slow pathway curve (figs. 3D and 4). The critical V-A interval was 350 msec.

Induced episodes of PSVT (both antegradely and retrogradely induced) had a cycle length of 400 msec and were characterized by a retrograde P wave occurring before the QRS complex with an $A_2-H_2$ of 70 msec and $H_2-A_1$ of 330 msec (a ratio of 0.21) (fig. 1B). This PSVT was identical to spontaneously observed episodes of PSVT.

Comment

The following interpretation of electrophysiological results in this patient seems reasonable. The patient had dual A-V nodal pathways with a major discordance of antegrade and retrograde properties. In the antegrade direction, the A-V nodal fast pathway had a shorter refractory period than the A-V nodal slow pathway, resulting in a continuous (fast pathway) conduction curve. In the retrograde direction, the fast pathway refractory period was longer than that of the slow pathway, resulting in the discontinuous retrograde curve. The discontinuity reflected retrograde failure of the fast pathway with resultant slow pathway conduction. The echo phenomenon occurred with block of the fast pathway. This pathway then became available for antegrade conduction. The inability to induce sustained PSVT prior to atropine reflected the inability of both A-V nodal pathways to conduct sequential impulses.

Atropine produced the following demonstrable electrophysiological changes: 1) improvement in antegrade A-V nodal fast pathway conduction (decrease in refractory period with maintenance of intact A-V conduction up to a paced rate of 220 beats/min), 2) slight improvement in retrograde slow pathway conduction (decrease in refractory period and increase in the paced rate allowing intact V-A conduction). Paroxysmal supraventricular tachycardia could be sustained after atropine because of improved antegrade fast pathway conduction and improvement of retrograde slow pathway conduction. Induction of PSVT after atropine could be demonstrated from the ventricle, with coupled impulses that blocked in the fast pathway (see above) and also from the atrium with rapid stimulation. The latter induction appeared to reflect antegrade block of the impulse in the slow pathway (a concealed event) at a critical rate. This resulted in the slow pathway being available for retrograde conduction upon sudden cessation of pacing (concealed re-entry).

The possibility that the discontinuity in retrograde conduction curves reflected achievement of a retrograde refractory period in the His-Purkinje system seems unlikely for the following reasons: 1) the increment in V-A interval with a small change in coupling interval was over 150 msec, a relatively large increment if one were to postulate sudden block in a segment of the His-Purkinje system. 2) The demonstration of identical increments in V-A interval during ventricular pacing at critical heart rates. This demonstration is identical to the demonstration of sudden increments in A-H interval during atrial pacing in patients with dual antegrade A-V nodal pathways. 3) the decrease in retrograde slow pathway refractory periods with atropine, suggesting that dual A-V nodal pathways were infranodal, and 4) The simultaneous occurrence of marked increase in V-A with induction of echoes (control) and PSVT (after atropine). The normal H-V and narrow QRS of echo beats is inconsistent with re-entry occurring in the His-Purkinje system.

This case also does not fulfill criteria for other varieties of PSVT, e.g., low atrial re-entrance, re-entrance utilizing a
concealed Kent bundle, His bundle re-entrance, and low atrial ectopic firing.18

Case Two

This was a 60-year-old female with recurrent PSVT. During PSVT, P waves were not seen; they probably occurred simultaneously with QRS complexes (see below). Cardiac examination, chest X-ray and resting electrocardiogram were normal. Electrophysiological studies during sinus rhythm revealed an A-H of 112 msec and an H-V of 45 msec.

Antegrade studies with incremental atrial pacing revealed intact A-V conduction up to a paced rate of 170 beats/min. At paced rates of 180 and above, Wenckebach periodicity with unexpected sudden A-H increments in the midst of paced Wenckebach periods were noted without echoes (fig. 5A). Atrial extrastimulus testing at a cycle length of 740 msec revealed a continuous A1-A2, H1-H2 conduction curve with atrial-limited A-V conduction (fig. 6), without echoes.

Retrograde studies with incremental ventricular pacing revealed intact V-A conduction up to a paced rate of 90 beats/min. Retrograde Wenckebach periodicity with sudden unexpected increase in V-A interval (from 290 to 460 msec) was noted at a ventricular paced rate of 100 beats/min (fig. 5B), with simultaneous occurrence of ventricular echoes (fig. 5B).

Ventricular extrastimulus testing (driven cycle length of 740 msec) revealed a discontinuous V1-V2 curve due to sudden increase in V1-A2 at critical V1-V2 coupling intervals of 650 msec, suggesting dual retrograde A-V nodal pathways (figs. 7A, 7B and 8). The effective refractory periods of the fast and slow pathways were 620 and 560 msec, respectively. Single A-V nodal re-entrant ventricular echoes occurred when V2 was conducted via the slow.

Figure 5. Recordings from case 2, showing responses to rapid atrial and ventricular pacing before and after atropine. Panel A) Before atropine, at an atrial paced rate of 190 beats/min, second degree A-V nodal Wenckebach block occurred, but echoes were not induced, despite achieving an A-H of 420 msec. Panel B) Before atropine at a ventricular paced rate of 100 beats/min, retrograde atypical Wenckebach periodicity with a sudden increase of V-A interval from 290 to 460 msec occurred. An A-V re-entrant ventricular echo occurred with this sudden increase of V-A interval. This echo was characterized by a short A-H, interval of 140 msec. Panel C) After atropine at an atrial paced rate of 170 beats/min, sustained PSVT was induced upon achieving a critical A-H interval of 260 msec. This PSVT was characterized by an A2-H2/H2-A1 ratio of 4.67 (280/60) and the retrograde P waves occurred simultaneously with the QRS complexes. Panel D) After atropine at a ventricular paced rate of 170 beats/min, second degree retrograde block occurred but atypical Wenckebach periodicity and echoes were no longer observed.
pathway with a critical V-A of 490 msec (figs. 5B and 7B).

One mg of atropine was administered intravenously. Atrial incremental pacing revealed 1:1 A-V conduction up to a paced rate of 170 beats/min. A-H intervals ranged from 75 to 110 msec at paced rates of 80 to 150 beats/min, and suddenly increased to 250 msec at a paced rate of 150 beats/min, suggesting failure of an antegrade fast A-V nodal pathway, with conduction via an antegrade slow A-V nodal pathway. Sustained PSVT with simultaneous P and QRS was induced with sudden cessation of atrial pacing at a rate of 170 beats/min with a slow pathway A-H of 260 msec (fig. 5C). This appeared to reflect antegrade slow pathway and retrograde fast pathway conduction (the common type of A-V nodal re-entrant PSVT). Atrial extrastimulus testing at a cycle length of 740 msec revealed a continuous A-V nodal conduction curve which was limited to atrial conduction. Only fast pathway A-H intervals were obtained (fig. 6).

Retrograde studies with incremental ventricular pacing revealed 1:1 V-A conduction up to a paced rate of 160 beats/min; V-A interval increased from 140 to 150 msec as the paced rate increased from 110 to 160 beats/min. Second degree V-A block occurred at a paced rate of 170 beats/min (fig. 5D), without sudden increase of V-A interval. Ventricular extrastimulus studies at a cycle length of 600 msec revealed a continuous V1-V2, A1-A2 curve with a ventricular limited V-A conduction (fig. 8). Atrioventricular re-entrant ventricular echoes were not induced.

Comment

This patient had dual A-V nodal pathways with a major discordance of antegrade and retrograde properties. In the antegrade direction (at the tested driven cycle length), the fast pathway refractory period was shorter than the slow pathway refractory period, producing the continuous antegrade conduction curve. Block in the antegrade fast pathway could be achieved with rapid atrial pacing, presumably reflecting fast pathway refractoriness from multiple impulse propagation. The lack of A-V nodal re-entrant atrial echoes during cessation of pacing during antegrade slow pathway conduction reflected relatively long retrograde fast pathway refractoriness.

Retrogradely, the fast pathway refractory period was longer than that of the slow pathway, producing the discontinuous conduction curve. The echo phenomenon reflected retrograde block in the fast pathway with retrograde slow pathway conduction, and return to the ventricles via the failed fast pathway. The inability to induce PSVT upon retrograde failure of the fast pathway reflected slow pathway refractoriness (the inability of the slow pathway to conduct two sequential impulses).

Atropine produced the following demonstrable electrophysiological effects: 1) improvement of antegrade fast pathway conduction with marked decrease in fast pathway A-H at equivalent coupling intervals; 2) improved antegrade slow pathway conduction, with maintenance of intact slow pathway conduction up to a paced rate of 170 beats/min and shortening of retrograde fast pathway refractory period so that the discontinuous retrograde conduction curve was changed to continuous (fast pathway retrograde refractory period shorter than slow pathway retrograde refractory period).

Paroxysmal supraventricular tachycardia induction after
lowing QRS complexes. Cardiac examination, chest X-ray, and resting electrocardiograms were normal. His bundle recordings during sinus rhythm revealed an A-H of 105 msec and an H-V of 45 msec.

Antegrade studies with incremental atrial pacing revealed intact A-V conduction up to a paced rate of 190 beats/min. A-H interval increased from 100 to 160 msec as the paced rate was increased from 105 to 150 beats/min and suddenly increased to 260 msec at a paced rate of 160 beats/min, suggesting failure of an antegrade fast A-V nodal pathway with conduction via an antegrade slow A-V nodal pathway. Second degree A-V nodal Wenckebach block occurred at a paced rate of 200 beats/min. Atrioventricular nodal re-entrant echoes occurred upon achieving a critical A-H interval of 325 msec (fig. 9A). The echo was characterized by a long A-H of 210 msec and a short H-A of 145 msec and the P wave occurred slightly after the QRS complex. Atrial extrastimulus studies at a driven cycle length of 460 msec revealed a discontinuous A-A, H-H curve, suggesting dual A-V nodal pathways (fig. 10). The effective refractory periods of fast and slow pathway were 320 and 280 msec, respectively. The maximal A-H interval achieved with atrial extrastimulus was 275 msec and echoes did not occur.

Retrograde studies with incremental ventricular pacing revealed 1:1 V-A conduction up to 140 beats/min; V-A interval increased from 140 to 160 msec as the paced rate was increased from 100 to 140 beats/min. Second degree Wenckebach periodicity with a sudden unexpected increase of V-A interval (from 165 to 360 msec) simultaneous with the occurrence of A-V nodal re-entrant ventricular echoes occurred at a ventricular paced rate of 150 beats/min (fig. 9B). Ventricular extrastimulus studies at a driven cycle length of 460 msec revealed a discontinuous V1-V2, A2-A2 curve, suggesting dual A-V nodal pathways (figs. 11 and 12). The effective refractory periods of the fast and slow pathways were 340 and 330 msec, respectively. A-V nodal re-entrant ventricular echoes occurred when V2 was conducted

**Figure 8.** $V_1-V_2, A_1-A_2$ and $V_1-V_2, V_2-A_2$ curves before and after atropine in case 2. Left) Before atropine, the curves were discontinuous at a driven cycle length of 740 msec. The effective refractory period of the fast pathway was 620 msec and the slow pathway 560 msec. Overlap of the fast and slow pathway occurred at $V_1-V_2$ between 650 and 630 msec. Echo zone coincided with the whole slow pathway curve. Critical $V-A$ interval was 490 msec. Right) After atropine, the curves were continuous at a driven cycle length of 600 msec. Ventricular functional refractory period of 250 msec limited $V-A$ conduction. Echo did not occur.

**Case Three**

This was a 61-year-old female with recurrent PSVT. During PSVT, retrograde P waves were noted immediately following QRS complexes. Cardiac examination, chest X-ray, and resting electrocardiograms were normal. His bundle recordings during sinus rhythm revealed an A-H of 105 msec and an H-V of 45 msec.

Antegrade studies with incremental atrial pacing revealed intact A-V conduction up to a paced rate of 190 beats/min. A-H interval increased from 100 to 160 msec as the paced rate was increased from 105 to 150 beats/min and suddenly increased to 260 msec at a paced rate of 160 beats/min, suggesting failure of an antegrade fast A-V nodal pathway with conduction via an antegrade slow A-V nodal pathway. Second degree A-V nodal Wenckebach block occurred at a paced rate of 200 beats/min. Atrioventricular nodal re-entrant echoes occurred upon achieving a critical A-H interval of 325 msec (fig. 9A). The echo was characterized by a long A-H of 210 msec and a short H-A of 145 msec and the P wave occurred slightly after the QRS complex. Atrial extrastimulus studies at a driven cycle length of 460 msec revealed a discontinuous A-A, H-H curve, suggesting dual A-V nodal pathways (fig. 10). The effective refractory periods of fast and slow pathway were 320 and 280 msec, respectively. The maximal A-H interval achieved with atrial extrastimulus was 275 msec and echoes did not occur.

Retrograde studies with incremental ventricular pacing revealed 1:1 V-A conduction up to 140 beats/min; V-A interval increased from 140 to 160 msec as the paced rate was increased from 100 to 140 beats/min. Second degree Wenckebach periodicity with a sudden unexpected increase of V-A interval (from 165 to 360 msec) simultaneous with the occurrence of A-V nodal re-entrant ventricular echoes occurred at a ventricular paced rate of 150 beats/min (fig. 9B). Ventricular extrastimulus studies at a driven cycle length of 460 msec revealed a discontinuous V1-V2, A2-A2 curve, suggesting dual A-V nodal pathways (figs. 11 and 12). The effective refractory periods of the fast and slow pathways were 340 and 330 msec, respectively. A-V nodal re-entrant ventricular echoes occurred when V2 was conducted

**Figure 9.** Recordings from case 3, showing responses to rapid atrial and ventricular pacing before and after atropine. Panel A) Before atropine at an atrial paced rate of 200 beats/min. A-V nodal re-entrant echoes occurred upon achieving a critical A-H interval of 325 msec. This echo was characterized by a long A-H of 210 msec and a short H-A of 145 msec. The retrograde P wave (A2) occurred at the end of the QRS complex. Panel B) Before atropine at a ventricular paced rate of 150 beats/min. Atypical retrograde Wenckebach periodicity with a sudden increase of V-A interval from 165 to 360 msec occurred. Atrioventricular nodal re-entrant echo was induced. This echo had a short A-H of 150 msec and was different from the antegrade induced echo seen in panel A. Panel C) Atropine at an atrial paced rate of 200 beats/min, sustained PSVT was induced upon achieving a critical A-H of 250 msec. The PSVT had a cycle length of 300 msec, and was characterized by a long A-H of 190 msec and a short H-A of 110 msec (A-H/A-A ratio of 1.72) with the retrograde P waves occurring at the end of the QRS complexes identical to the echo seen in panel A.
via the slow pathway with a critical V-A interval of 330 msec. The echoes induced either with rapid ventricular pacing or with ventricular extrastimulus (figs. 9B and 11B) were different from the antegrade induced echoes (fig. 9A) in that the retrograde P wave occurred in front of the QRS complex with a short A2-H2 of 145 msec.

Atropine 0.5 mg was administered intravenously and the study was repeated immediately. After atropine, incremental atrial pacing revealed 1:1 A-V conduction up to a paced rate of 200 beats/min. The A-H interval increased from 100 to 120 msec as the paced rate increased from 100 to 170 beats/min and suddenly increased to 260 msec at a paced rate of 180 beats/min, suggesting failure of an antegrade fast A-V nodal pathway with conduction via an antegrade slow A-V nodal pathway. Atioventricular nodal re-entrant echoes and PSVT were induced at a paced rate of 200 beats/min which achieved a critical A-H interval of 250 msec (fig. 9C). The PSVT had a cycle length of 300 msec with an A2-H2 of 190 msec and an H2-A2 of 110 msec (a ratio of 1.72); a retrograde P wave occurred slightly after the QRS complex. This PSVT was identical to spontaneous PSVT. Atrial extrastimulus studies revealed a discontinuous A1-A2, H1-H2 curve with effective refractory periods of fast and slow pathway, 300 and 250 msec respectively (fig. 10). The maximal A-H interval achieved with atrial extrastimulus was 245 msec and echoes or PSVT did not occur.

Incremental ventricular pacing revealed 1:1 V-A conduction up to a paced rate of 180 msec; V-A interval increased from 120 to 130 msec as the paced rate increased from 120 to 180 beats/min. 3:2 V-A block occurred at a ventricular paced rate of 190 beats/min without a sudden increase of V-A interval (V-A increased from 120 to 140 msec) or induction of echoes or PSVT. Ventricular extrastimulus testing at a driven cycle length of 460 msec revealed a continuous V1-V2, A2-A3 curve with a retrograde A-V nodal effective refractory period of 260 msec (fig. 12). A-V nodal re-entrant echoes were not induced.

Comment

This patient differed from patients 1 and 2 in that both antegrade and retrograde A-V nodal pathways were demonstrable with extrastimulus testing. This reflected the fact that fast pathway refractory periods (antegrade and retrograde) were longer than slow pathway refractory periods, allowing critically timed extrastimuli (atrial or ventricular) to block in the fast pathways, traversing the slow A-V nodal pathway with resulting sudden increase in A-H or V-A. Atrioventricular nodal re-entrant atrial echoes were not seen during antegrade extrastimulus testing because slow pathway conduction times were not long enough for recovery of the fast pathway for retrograde fast pathway conduction. Longer antegrade slow pathway conduction times were achieved with rapid atrial pacing (slow pathway fatigue), allowing the demonstration of A-V nodal re-entrant atrial echoes (antegrade slow pathway and retrograde fast pathway). Atrioventricular nodal re-entrant ventricular echoes during ventricular extrastimulus testing were similar to those demonstrated in case 2 (retrograde slow pathway and antegrade fast pathway conduction). Sus-

Figure 10. A1-A2, H1-H2 and A2-H2 curves before and after atropine in case 3. Left) Before atropine, the curves were discontinuous at a driven cycle length of 460 msec. The effective refractory period of the fast pathway was 320 msec and the slow pathway 280 msec. An echo zone was not defined. Right) After atropine, the curves were still discontinuous at a similar driven cycle length of 460 msec. The effective refractory period of the fast pathway shortened to 300 msec and the slow pathway to 250 msec. An echo zone was not defined.

Figure 11. Recordings from case 3, showing induction of A-V nodal re-entrant echo with ventricular extrastimulus before atropine. The driven cycle length was 460 msec. Panel A) At V1-V2, coupling intervals of 375 msec, V1-A2 was 160 msec and A1-A2 380 msec. An echo did not occur. Panel B) At V1-V2 coupling interval of 370 msec, V1-A2 suddenly increased to 355 msec and A1-A2 to 550 msec. An A-V nodal re-entrant echo was induced. Note that this echo had a short A2-H2 interval of 145 msec.
tained PSVT was not induced because of the inability of both pathways to sustain sequential impulse propagation.

Atropine produced the following demonstrable electrophysiological effects: 1) facilitation of antegrade fast pathway conduction, with shorter A-H intervals at equivalent coupling intervals; 2) facilitation of antegrade slow pathway conduction with shorter A-H intervals at equivalent coupling intervals and decrease of antegrade slow pathway refractory period; 3) marked decrease of retrograde fast pathway refractoriness so that discontinuous retrograde conduction curves could no longer be demonstrated.

Induction of PSVT after atropine was similar to case 2 and was dependent upon cessation of atrial pacing while conduction was taking place via the slow pathway. As in case 2, the ability to induce and maintain PSVT reflected facilitation of antegrade slow pathway and retrograde fast pathway conduction.

Discussion

In the usual case of A-V nodal re-entrant paroxysmal tachycardia, antegrade conduction curves with atrial extrasystolic techniques are discontinuous, suggesting dual A-V nodal pathway.24 In these cases, the discontinuity in conduction curves reflects antegrade failure of a fast A-V nodal pathway with resultant antegrade slow pathway conduction. The failed fast pathway is used for retrograde conduction. The resultant PSVT is thus characterized by antegrade slow pathway and retrograde fast pathway conduction. Typically, in this type of PSVT, the P wave appears simultaneous with or slightly following the QRS, with an A-H to H-A ratio of greater than one.58 Retrograde conduction curves in the usual case of A-V nodal re-entrant PSVT are usually continuous, reflecting the fact that retrograde fast pathway refractory periods are shorter than those of retrograde slow pathways.9, 15, 21, 27 Induction of PSVT in such cases can sometimes be achieved with ventricular extrastimuli which conduct in the fast pathway (a concealed event, since it is not demonstrable by a sudden increase in V-A) and return to the ventricles via the blocked slow pathway. The resultant PSVT is still characterized by antegrade slow pathway conduction and retrograde fast pathway conduction.15, 28

The present study extends observations concerning A-V nodal re-entry. All three of the present cases had retrogradely discontinuous conduction curves. These reflected the presence of a retrograde fast pathway refractory period which was longer than that of the retrograde slow pathway.21, 23 Thus, critically-timed ventricular extrastimuli were blocked in the fast pathway and conducted via the slow pathway. The ventricular echo resulted from utilization of the blocked fast pathway for antegrade conduction. Induction of PSVT was achieved in case 1 after treatment with atropine, which facilitated antegrade and retrograde conduction in both pathways.27 The induced PSVT was of an uncommon type, and characterized by antegrade fast pathway and retrograde slow pathway conduction. This PSVT was therefore characterized by an unusual P-to-QRS relationship, with retrograde P wave in front of the QRS, with an A-H to H-A ratio of much less than one.59 This type of A-V nodal re-entrant PSVT could not be induced in cases 2 and 3. However, in the latter cases, injection of atropine allowed induction of the common type of A-V nodal re-entrant PSVT (antegrade slow pathway and retrograde fast pathway conduction). In the latter cases the drug facilitated retrograde fast pathway and antegrade slow pathway conduction so that the more common A-V nodal re-entrant PSVT could be induced.

The present cases also extend our knowledge regarding the type of PSVT which may be associated with discontinuous antegrade conduction curves. Previously, continuous curves were associated with induction of PSVT utilizing concealed retrogradely conducting Kent bundles, and with induction of sinus node or atrial re-entrant paroxysmal tachycardia.10-13, 18 Cases 1 and 2 of the present study revealed A-V nodal re-entrant PSVT, despite smooth conduction curves. Both cases had discontinuous retrograde conduction curves and retrograde Wenckebach periodicity with sudden unexpected increases in V-A interval, a diagnostic clue to the presence of A-V nodal re-entry.1, 9

In the present cases, the relationship of P to QRS during PSVT can also be examined. The unusual variety of PSVT characterized by retrograde P in front of the QRS was seen in case 1. This type of PSVT is thus consistent with A-V nodal re-entry (antegrade fast and retrograde slow pathway conduction). Electrocardiographically identical PSVT has been noted in some cases of low atrial re-entrance and in cases with apparent low atrial ectopic firing.18 Another observation of interest in the present study was the demonstration of a retrograde P in case 1, with antegrade P wave morphology (- +). Similar retrograde P waves have been described previously.16, 29

In summary, the present three cases provide additional insight into A-V nodal re-entry in man. Discontinuous conduction curves, although most commonly seen with
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antegrade stimulation, may sometimes be demonstrated with retrograde stimulation. In rare cases, both antegrade and retrograde conduction curves may be discontinuous. The type of PSVT observed in a patient with A-V nodal re-entrance will depend upon antegrade and retrograde properties of A-V nodal fast and slow pathways, as well as the site of origin of the initiating beat (atrial or ventricular).

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An unusual variety of atrioventricular nodal re-entry due to retrograde dual atrioventricular nodal pathways.

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