Effects of Atropine on Induction and Maintenance of Atrioventricular Nodal Reentrant Tachycardia

DELon WU, M.D., PABLO DENES, M.D., ROBERT BAUERNFELD, M.D., RAMESH C. DHINGRA, M.D., CHRISTOPHER WYNDHAM, M.D., AND KENNETH M. ROSEN, M.D.

SUMMARY The electrophysiologic effects of atropine were studied in 14 patients with dual atrioventricular (AV) nodal pathways and recurrent paroxysmal supraventricular tachycardia (PSVT). During PSVT, all patients used a slow pathway (SP) for antegrade and fast pathway (FP) for retrograde conduction. Atropine enhanced both SP antegrade and FP retrograde conduction, shown by a decrease in paced cycle lengths (atrial and ventricular) producing AV and ventriculoatrial block.

Five patients had induction of sustained PSVT before and after atropine. Seven patients failed to induce or sustain PSVT before atropine, because of retrograde FP refractoriness. All seven had induction of sustained PSVT after atropine due to facilitation of FP retrograde conduction. Two patients had only single atrial echoes before atropine, reflecting SP antegrade refractoriness. After atropine, sustained PSVT was inducible in one, and nonsustained in the other. PSVT cycle length could be compared in seven patients before and after atropine and decreased from 383 ± 25 to 336 ± 17 (p < 0.05).

Thus, in patients with dual AV nodal pathways, atropine facilitated SP antegrade and FP retrograde conduction, shortened cycle length of PSVT and potentiated ability to sustain PSVT.

DUAL ATRIOVENTRICULAR (AV) nodal pathways can be demonstrated in most patients with AV nodal reentrant paroxysmal supraventricular tachycardia (PSVT).1-6 In these patients, the most common type of sustained AV nodal reentrance involves using a slow pathway for antegrade conduction and a fast pathway for retrograde conduction.1-4 Recent pharmacologic studies have demonstrated that digitalis, propranolol and verapamil may prevent induction of sustained AV nodal reentrant PSVT by increasing antegrade slow pathway refractoriness.5, 4, 7-9 Propranolol may prevent PSVT induction in patients with dual AV nodal pathways, by increasing retrograde fast pathway refractoriness.10

Limited data are available describing the effect of agents that facilitate conduction on AV nodal reentry. Akhtar and co-workers reported induction of AV nodal reentrant PSVT only after atropine in five patients with no previous history of PSVT, suggesting that vagolysis facilitated the development of AV nodal reentrant circus movements.11 In this study, we report the effects of atropine in patients with previously documented PSVT and electrophysiologically demonstrable dual AV nodal pathways. The effects of atropine on fast and slow pathways are quantitated, and the effects of this agent on PSVT induction noted.

Methods

Patient Selection

Criteria for inclusion in this study included: 1) a history of electrocardiographically documented recurrent PSVT; 2) absence of preexcitation on all available ECGs; 3) electrophysiologic demonstration of dual AV nodal pathways with demonstration of discontinuous A1-A2, H1-H2 curves during atrial extrastimulus testing (10 patients) or demonstration of two sets of A-H intervals at identical atrial paced cycle lengths (four patients).1-12-16 The latter demonstrations were during the same trial of incremental atrial pacing, the two sets of A-H reflecting fatigue (block) in the fast pathway with resultant slow pathway conduction, and 4) demonstration of AV nodal reentrant PSVT (see Results).1, 5, 17-19

Fourteen patients, nine females and five males, ages 24-81 years (mean ± sp 59 ± 16 years) were studied.

Electrophysiological Studies

Electrophysiological study was performed with the patient in the nonsedated supine state. Cardiac drugs were discontinued at least 72 hours before study. Informed written consent was obtained in each patient. A #7 quadripolar electrode catheter was placed across the tricuspid valve percutaneously via the right femoral vein. The proximal two electrodes were used for His bundle recording, while the distal two electrodes were used for ventricular pacing.20 A second #6 hexapolar electrode catheter was positioned in the distal coronary sinus via an antecubital vein. The distal two electrodes were used for recording of left atrial electrograms, the middle two electrodes for recording of high right atrial electrogram, and the proximal two electrodes for right high atrial pacing. Multiple surface and intracardiac electrograms were recorded simultaneously via a multichannel oscilloscopic recorder (Electronics for Medicine DR-16, White

From the Cardiology Section, Department of Medicine, Abraham Lincoln School of Medicine, University of Illinois College of Medicine, and West Side Veterans Administration Hospital, Chicago, Illinois.

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Address for reprints: Kenneth M. Rosen, M.D., Cardiology Section, University of Illinois Hospital, P.O. Box 6998, Chicago, Illinois 60680.

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Plains, New York) at a paper speed of 100 and 200 mm/sec. Stimuli were provided via a programmable digital stimulator (manufactured by M. Bloom, Nar-beth, Pennsylvania) with a strength of approximately twice diastolic threshold and 2 msec long.

Antegrade and retrograde conduction properties, refractory periods and echo zones were studied with atrial and ventricular incremental pacing and extra-stimulus technique.1, 10, 14 Retrograde His bundle potentials could not be reliably recorded at all coupling intervals during ventricular extrastimulus testing in most of the patients. After control recording, studies were initiated 15 minutes after administration of 0.5–1 mg atropine intravenously.

Electrophysiological Definitions

HRA, A1, H1, and V1 were the high right atrial, low septal right atrial, His bundle, and ventricular responses to driven stimuli (S1), respectively. HRA1, A2, H2, and V2 were the high right atrial, low septal right atrial, His bundle and ventricular responses to test stimuli (S2), respectively. Ae, He and Ve were the atrial, His bundle and ventricular responses of AV nodal reentrant echo beats, respectively. Conduction intervals, refractory periods, echo zones, and critical A-H and V-A intervals (for induction of AV nodal reentrant echoes and PSVT) were measured and defined as previously described.1, 10, 14

Antegrade dual AV nodal pathways were diagnosed when discontinuous A-A2, H1-H2 curves were demonstrated, or when two sets of A-H intervals were demonstrated at identical atrial paced cycle lengths.1, 12-16 Both antegrade and retrograde effective refractory periods of the fast and slow pathway were defined as previously described.1, 10, 14

The diagnosis of AV nodal reentrant PSVT was made with combinations of the following diagnostic criteria: 1) induction of PSVT related to achievement of a critical A-H delay, with both incremental atrial pacing and with atrial extrastimulus testing;1, 2, 21-23 2) demonstration of discontinuous A1-A2, H1-H2 curves, suggesting dual AV nodal pathways, with induction of PSVT relating to antegrade block in the fast pathway;1, 2, 10-15 3) demonstration of atrial activation before or simultaneous with onset of ventricular activation during PSVT, suggesting that the ventricles were not part of an AV reentrant circuit movement;1, 2, 17 4) normal retrograde atrial activation sequence during PSVT, with low septal right atrium being activated earlier than all other atrial recording sites,24, 25 5) increase of ventriculo-atrial (VA) interval with incremental ventricular pacing with type I VA block at critical rate, suggesting retrograde AV nodal conduction;1, 10, 14 6) demonstration of His bundle activation (H1) preceding the atrial activation (A2) with ventricular extrastimulus testing during ventricular pacing and/or PSVT, suggesting retrograde AV nodal conduction;1, 17 7) absence of previously described criteria for diagnosis of concealed extranodal pathways, or sinoatrial reentry.1, 2, 26-33 All patients manifested criteria 4 and either 3 or 6.

Sustained PSVT was defined as induced PSVT that lasted longer than 2 minutes. Sustained PSVT was always terminated with single, double or multiple atrial extrastimuli. Nonsustained PSVT was defined as induced PSVT terminated spontaneously (within 2 minutes). In almost all instances, nonsustained PSVT terminated spontaneously within 10–20 seconds of PSVT induction. In patients with nonsustained PSVT, the site of block (weak-link) in the circuit was determined by noting whether PSVT was terminated with an atrial response (block in antegrade limb) or with a QRS complex (block in retrograde limb).10

The determinants of reentrance (antegrade slow pathway and retrograde fast pathway) were evaluated as previously described.3,4 The evaluation of antegrade slow pathway primarily depended on noting the paced atrial cycle length producing AV nodal block. Since in the patient with dual pathways, the antegrade fast pathway has a longer refractory period than the antegrade slow pathway, this paced cycle length reflects slow pathway refractoriness.

For the retrograde fast pathway, this evaluation primarily depended on noting the paced ventricular cycle length producing VA block. This procedure is based on the assumption that the shortest VA retrograde refractory period in these patients is that of the retrograde fast pathway, rather than the retrograde slow pathway. This assumption is based on the following observations: 1) Retrograde conduction curves (H1-H2, A1-A2) are smooth and suggest one retrograde pathway; 2) VA conduction times are short (there are not two populations of retrograde conduction times) and are consistent with observed antegrade fast pathway conduction times; 3) retrograde conduction is via AV node (normal retrograde activation sequence, and H2 and A2 are both driven out of the QRS with closely coupled ventricular extrastimuli). Retrograde fast pathway effective refractory period cannot be directly measured in most patients with dual AV nodal pathways because of either limiting ventricular refractoriness or limiting refractoriness between the ventricle and His bundle.

Results

PSVT Induction (Table 1)

In seven of the 14 patients, sustained AV nodal reentrant PSVT could not be induced before atropine administration, despite achievement of sole conduc-
tion in the antegrade slow pathway (antegrade failure of the fast pathway) with atrial extrastimulus testing and/or rapid incremental atrial pacing (cases 1–7). In three of these patients (cases 1–3), nonsustained AV nodal reentry was induced before atropine, with spontaneous termination of PSVT due to block in the retrograde fast pathway (inadequate retrograde fast pathway conduction) (fig. 1A). In four of the patients (cases 4–7), no AV nodal reentrant atrial echoes were noted because of inadequate retrograde fast pathway conduction (fig. 2B). After atropine, all seven patients had induction of sustained AV-nodal PSVT due to enhanced retrograde fast pathway conduction (figs. 1B, 2C).
Table 1. Effects of Atropine on Induction and Maintenance of Atrioventricular Nodal Reentrant Tachycardia

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Abbreviations: PSVT = paroxysmal supraventricular tachycardia; CL = cycle length; FP-ERP = effective refractory period of the fast pathway; SP-ERP = effective refractory period of the slow pathway; CL-AVB = longest atrial paced cycle length producing atrioventricular block; CL-VAB = longest ventricular paced cycle length producing ventriculoatrial (VA) block; C = control; A = atropine; Ant = antegrade; Ret = retrograde.

1C and 2C). PSVT was induced with either rapid atrial pacing (all seven patients) or with double atrial extrastimuli (cases 1–3, 6, 7). In only two patients was PSVT inducible with single atrial extrastimulus testing after atropine (cases 2 and 5).

In two of the 14 patients, sustained PSVT was not induced before atropine due to inadequate antegrade slow pathway conduction (cases 8 and 9). In both patients only single AV nodal reentrant atrial echoes were induced after achievement of sole antegrade slow pathway conduction (fig. 3A). After atropine, one of these patients had induction of sustained PSVT due to enhanced antegrade slow pathway conduction (case 8) (fig. 3B), while the other continued to have induction of only single AV nodal reentrant atrial echoes (case 9).

Five of the 14 patients had induction of sustained PSVT before atropine (cases 10–14) (fig. 4A). In all five patients, PSVT was inducible with rapid atrial pacing and in four with single atrial extrastimulus testing (cases 10–12 and 14). After atropine administration, all five patients had induction of sustained PSVT with either rapid atrial pacing (at faster paced rates than those necessary for induction prior to atropine) or with double atrial extrastimuli (A1, A2, A3, testing) (figs. 4B and 4C). In only one patient was PSVT inducible with single atrial extrastimulus testing after atropine (case 14).

Conduction Curves and Echo Zones (table 1)

Discontinuous A1-A2, H1-H2 curves suggesting dual AV nodal pathways were demonstrated in 10 of the 14 patients before atropine (cases 1–3, 7–12 and 14) (figs. 2A, 2B and 5). In eight of these 10 patients, an echo zone could be defined which coincided with either the whole slow pathway curve or the leftward portion of the slow pathway curve. In two patients, an echo zone was not defined, despite demonstration of the discontinuous curve (cases 1 and 7) (fig. 5, left panel). After atropine, discontinuous A1-A2, H1-H2 curves were demonstrated in four of these 10 patients (cases 1–3 and 14). Three of these four had echo zones before atropine, and two of four had echo zones (and PSVT...
induction) after atropine (cases 2 and 14). In six patients (cases 7-12), discontinuous A₁-A₂, H₁-H₂ curves became continuous after atropine due to facilitation of fast pathway conduction (see below) and echo zones were not defined (fig. 5, middle panel). Antegrade block in the fast pathway with antegrade slow pathway conduction (with echoes and PSVT) in these six patients was achieved with rapid atrial pacing and/or double atrial extrastimuli after atropine (figs. 2C, 3B, 4B, 4C and 5, right panel).

Continuous A₁-A₂, H₁-H₂ curves were demonstrated in four of the 14 patients before atropine (cases 4-6 and 12). In three of these four patients, atrial functional refractory period was longer than the effective refractory period of the fast pathway (cases 4, 6 and 13), and in the other (case 5) only the slow pathway was used for antegrade conduction at the tested driven cycle length. Echo zones were not demonstrated in these four patients, although in one patient (case 13) sustained PSVT was inducible with rapid atrial pacing. After atropine, continuous curves without definition of an echo zone were demonstrated in three of the four patients (cases 4, 6 and 13). In all three patients, sustained PSVT could be induced after atropine with rapid atrial pacing and/or double atrial extrastimuli. In the remaining patient (case 5, the patient with only slow pathway conduction with extrastimulus testing), A₁-A₂, H₁-H₂ curve became discontinuous after atropine due to shortening of antegrade fast pathway refractory period. An echo zone was...
defined, and coincided with the whole slow pathway curve.

Antegrade Fast Pathway Properties (table 1)

Antegrade fast pathway effective refractory periods were measured in 11 patients before and after atropine (cases 1–3, 5, 7–12 and 14) (fig. 5, left and right panels). Antegrade fast-pathway effective refractory period was ≤340 ± 21 msec (mean ± SEM, range 270 to <500) before atropine, and decreased to ≤276 ± 9 msec (range <200 to 300) after atropine (p < 0.01).

Antegrade Slow Pathway Properties (table 1)

Refractoriness of the antegrade slow pathway could be evaluated in 12 patients before and after atropine by noting the longest atrial paced cycle length producing AV nodal block (cases 1, 3–12 and 14). The atrial paced cycle length producing AV block was 373 ± 18 msec (range 300–500 msec) before atropine, and decreased to ≤301 ± 11 msec (range <273–375) after atropine (p < 0.01).

Antegrade effective refractory period of the slow pathway could be compared with extrastimulus tech-
nique before and after atropine in only two patients, and was decreased in both patients after atropine (cases 1 and 5).

Retrograde Fast Pathway Properties (table 1)

Retrograde fast pathway conduction could be evaluated in nine patients before and after atropine by noting the longest ventricular paced cycle length producing VA block (cases 1–9) (figs. 1B and 1D). VA conduction was via fast pathway at short paced cycle lengths in most patients before atropine, and in all patients after atropine (see below). The ventricular paced cycle length producing VA block was $429 \pm 36$ msec (range $375-667$ msec) before atropine, and decreased to $\leq 313 \pm 7$ msec (range $284-333$) after atropine ($p < 0.01$).

The retrograde effective refractory period of the fast pathway could be compared before and after atropine in seven patients (cases 1, 2, 4–7 and 13) and decreased from $390 \pm 51$ msec (range $250-650$ msec) to $< 271 \pm 14$ msec (range $230-325$ msec) after atropine ($p < 0.05$). The retrograde effective refractory period decreased in six of these seven patients. In seven patients (3, 8–12 and 14), retrograde fast pathway effective refractory periods could not be measured because of limiting ventricular refractoriness.

Retrograde Slow Pathway Properties (table 1)

Although retrograde effective refractory period of the slow pathway was achieved in four patients before atropine (1, 4, 6 and 7), it was not achieved in any of the 14 patients after atropine because retrograde effective refractory period of the fast pathway was either shorter than that of the slow pathway or shorter than the ventricular functional refractory period (continuous $V_1-V_2$, $A_1-A_2$ curves).

Cycle Lengths of PSVT (table 1)

Cycle lengths of PSVT could be compared in seven patients before and after atropine (cases 2, 3, 10–14) (figs. 1A and C and 4A and C). Cycle lengths of PSVT ranged from 305–480 msec (mean $\pm$ SEM $383 \pm 25$ msec) before atropine, and ranged from 300–400 msec (mean $\pm$ SEM $334 \pm 17$ msec) after atropine. The cycle lengths of PSVT shortened significantly after atropine ($p < 0.05$). This shortening reflected decrease in both antegrade slow pathway conduction time (mean $\pm$ SEM). The A-H interval during PSVT decreased from $294 \pm 18$ to $226 \pm 12$ msec after atropine ($p < 0.05$), and retrograde fast pathway conduction time H-A during PSVT decreased from $89 \pm 16$ to $71 \pm 12$ msec after atropine ($p < 0.05$). A-H and H-A are only approximate measures of antegrade slow and retrograde fast pathway conduction time (H is distal to the final common pathway).

Discussion

AV Nodal Reentrant PSVT

Dual AV nodal pathways are demonstrable in most patients with AV nodal reentrant PSVT. In these patients, critically timed atrial premature stimuli block in a fast AV nodal pathway resulting in sudden increase in A-H interval (sole antegrade slow pathway conduction). If the blocked fast pathway is available for retrograde conduction, PSVT induction may result. The usual circus movement in these patients
FIGURE 4. Recordings from case 11 demonstrating induction of sustained paroxysmal supraventricular tachycardia (PSVT) before and after atropine. RA = right atrial electrogram; CSA = coronary sinus electrogram; HBE = His bundle electrogram. Panel A demonstrates induction of sustained PSVT before atropine after cessation of rapid atrial pacing at a atrial paced cycle length (CL) of 333 msec, which achieved a slow-pathway A-H of 450 msec. CL of PSVT was 355 msec. Panel B demonstrates no induction of PSVT after atropine at a shorter atrial paced CL of 273 msec with an A-H (fast-pathway conduction) of 130 msec. Panel C demonstrates induction of sustained PSVT after atropine with atrial extrastimulus testing. The basic driven CL was 400 msec. A1-A2 was 220 msec, and A3 was still conducted via the fast-pathway. PSVT was induced by a premature atrial beat (PAB) (A3) after A2. The A3 achieved slow-pathway conduction with an A2-H3 of 340 msec. CL of PSVT was 300 msec.

In patients with depressed slow pathway conduction, only single AV nodal reentrant atrial echoes or nonsustained PSVT can be induced with atrial stimulation. Termination of PSVT in these patients occurs when atrial responses are not followed by His bundle or ventricular responses, suggesting block in the antegrade limb of the circuit. In patients with depressed retrograde fast pathway conduction, induction of AV nodal reentrant atrial echoes may not be possible, or only nonsustained PSVT may be induced with atrial stimulation. Termination of PSVT in these patients occurs when QRS complexes are not followed by atrial responses, suggesting block in the retrograde limb of the circuit.
Antiarrhythmic Agent and AV Nodal Reentrance

Previous electrophysiological studies have demonstrated that antegrade and retrograde properties of both fast and slow pathway can be modified by antiarrhythmic agents, and the ability to induce or sustain PSVT may be suppressed or enhanced after drug administration. Propranolol, digitalis and verapamil increase antegrade slow pathway refractoriness. There is little information regarding the effects of propranolol and verapamil on retrograde fast pathway conduction. Digitalis appears to have little effect on retrograde fast pathway refractoriness. All of these drugs inhibit the ability to sustain PSVT in some patients with AV nodal reentrance, by increasing refractoriness in the antegrade limb of the circus movement. Procainamide depresses retrograde fast pathway conduction and inhibits the ability to induce or sustain PSVT in the majority of the patients with AV nodal reentrant PSVT. However, procainamide may enhance antegrade slow pathway conduction due to vagolytic effect and potentiates the ability to sustain PSVT in a minority of patients with AV nodal reentrance.

Effects of Atropine

In this study, we demonstrated that atropine enhanced retrograde fast pathway and antegrade slow pathway conduction, and thus potentiated the ability to induce sustained PSVT. In seven patients with either no induction of AV nodal reentrant atrial echoes or induction of nonsustained PSVT due to depressed retrograde fast pathway conduction, improvement of retrograde fast pathway conduction was sufficient to allow induction of sustained PSVT after atropine. In one of the two patients with induction of only single AV nodal reentrant atrial echoes due to depressed antegrade slow pathway conduction, the improvement of antegrade slow pathway conduction after atropine was sufficient to allow induction of sustained PSVT.

Although atropine facilitated the induction of sustained AV nodal reentrant PSVT in patients with dual pathways with either inadequate antegrade slow pathway, or inadequate retrograde fast pathway conduction, PSVT induction in the laboratory was frequently more difficult after atropine administration. This reflected the effects of atropine on fast and slow pathways. Facilitation of fast pathway conduction frequently changed discontinuous to continuous conduction curves (decrease of fast pathway effective refractory period so that it was less than atrial functional refractory period). This prevented achievement of antegrade slow pathway conduction with the atrial extrastimulus technique. Despite the continuous conduction curves in these patients, failure of the fast pathway (with resultant antegrade slow pathway conduction and PSVT induction) could be achieved after atropine using either rapid incremental atrial pacing (repetitive rapid conduction may produce block in the fast pathway) or SSsSs stimulation. Even when discontinuous conduction curves were present after atropine administration, echo zones might not be delineated because of facilitation of antegrade slow pathway conduction, making achievement of the slow critical pathway A-H necessary for reentry difficult. Critical slow-pathway A-H could be achieved after atropine using other forms of atrial stimulation (rapid incremental atrial pacing and/or SSsSs technique).

In patients who had PSVT before and after

FIGURE 5. Atrioventricular (AV) conduction curves before and after atropine in case 7. The basic driven cycle length (CL) was 400 msec. Solid circles represent responses without echoes, while open circles represent responses with echoes. Left panel demonstrates discontinuous A1-A2, A2-H2 and A1-A2, H1-H2 curves, suggesting dual AV nodal pathways before atropine. The effective refractory periods of the fast and slow pathway were 420 msec and < 290 msec, respectively. An echo zone was not defined. Middle panel demonstrates continuous A1-A2, A1-H1 and A1-A2, H1-H2 curves after atropine. The curves became continuous because of shortening in antegrade fast-pathway effective refractory period (< 260 msec). Right panel demonstrates discontinuous A1-A2, A2-H2 and A1-A2, H1-H2 curves after atropine, suggesting dual AV nodal pathways. The driven CL (A1-A1) was 400 msec, and A1-A2 was 270 msec. The effective refractory periods of the fast and slow-pathway were 290 msec and < 255 msec. The echo zone coincided with the whole slow-pathway curve.
atropine, atropine shortened the cycle length of PSVT, primarily due to shortening antegrade slow pathway conduction time (A-H). Although retrograde fast pathway conduction time (H-A) was also shortened after atropine, the amount of shortening was relatively small compared with shortening of antegrade slow pathway conduction time. Since induction of sustained PSVT requires the cycle length of PSVT to be longer than the effective refractory periods of any component of the reentrant circuit, it is theoretically possible (although not noted in our study) that atropine could also abolish the ability to sustain PSVT if shortening of PSVT cycle length exceeded shortening of refractory period of a portion of the reentrant circuit.

**Electrophysiological Applications**

This study has increased our knowledge of the nature of the fast AV nodal pathway. In a recent study, we demonstrated that procainamide selectively depressed retrograde and non antegrade fast pathway conduction, an unexpected response. We could not distinguish whether the retrograde fast pathway was an intranodal, atrial nodal or extranodal structure. We also did not know whether the retrograde fast pathway was anatomically identical to the antegrade fast pathway. The present demonstration of marked facilitation of both antegrade and retrograde fast pathway conduction with atropine strongly suggests that the fast pathway is partially or totally within the AV node.

In conclusion, atropine enhances antegrade slow pathway conduction, antegrade and retrograde fast pathway conduction, and allows induction of sustained PSVT. Facilitation of both antegrade and retrograde fast pathway conduction by atropine is consistent with the hypothesis that the fast pathway is partially or totally within the AV node. Induction of PSVT after atropine usually requires faster atrial paced rates (than before atropine) and/or double atrial extrastimuli. Inability to induce sustained PSVT in the catheterization laboratory in some patients with known PSVT due to AV nodal reentry probably reflects inadequate antegrade slow pathway or retrograde fast pathway conduction on the day of study (autonomically mediated). Administration of atropine in these patients may be useful in potentiating PSVT induction, thus helping to delineate the mechanism of tachycardia.

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Effects of atropine on induction and maintenance of atrioventricular nodal reentrant tachycardia.

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