Salutary effects of intravenous ajmaline in patients with paroxysmal supraventricular tachycardia mediated by dual atrioventricular nodal pathways: blockade of the retrograde fast pathway


ABSTRACT Electrophysiologic effects of 50 mg iv ajmaline were evaluated in 10 patients with atrioventricular nodal reentrant paroxysmal supraventricular tachycardia (PSVT) utilizing the slow pathway for antegrade and the fast pathway for retrograde conduction. Ajmaline terminated the PSVT in all 10 patients in 17 to 165 sec (mean 94 ± 49 sec): by ventriculoatrial block in eight, AH block in one, and intra-atrial reentry in one patient. The predrug mean PSVT cycle length of 289 ± 44 msec (range 240 to 350) increased significantly to 373.5 ± 60 msec (range 263 to 464; p < .01) before the tachycardia was terminated. The increase in cycle length was a function of both AH and HA prolongation. In all 10 patients ajmaline depressed conduction through the retrograde fast pathway, as evidenced by the increase in mean ventricular paced cycle length producing ventriculoatrial block from ≤280 ± 40 to 438 ± 93 msec (p < .001), and the increase in the effective refractory period of the ventriculoatrial conduction system from ≤241 ± 42 to ≤298 ± 62 msec (p < .05); the drug abolished ventriculoatrial conduction in four cases. The effective refractory period of the antegrade fast pathway was unchanged after ajmaline (≤281 ± 31 vs ≤275 ± 38 msec; p = NS), but conduction through the antegrade slow pathway was depressed (atrial paced cycle length producing AH block 269 ± 30 msec before and 312 ± 44 msec after drug; p < .05). PSVT could not be reinduced in eight subjects, predominantly because of inhibition of the retrograde pathway, although suppression of the antegrade slow pathway also contributed in one patient. In the remaining two subjects depression of retrograde conduction was insufficient to prevent sustenance of tachycardia. We conclude that ajmaline terminates atrioventricular nodal reentrant PSVT by blockade of the retrograde fast pathway, although effects on the antegrade slow pathway are also observed.


AJMALINE, an alkaloid derived from the Indian plant Rauwolfia serpentina, has been found useful in experimental and clinical atrial and ventricular arrhythmias.1-5 By virtue of its strong depressant action on accessory pathways, the drug has been extremely successful in management of arrhythmias associated with Wolff-Parkinson-White syndrome.4-5 No information is available concerning the efficacy of ajmaline in patients with paroxysmal supraventricular tachycardia (PSVT) that is mediated by dual atrioventricular nodal pathways. The present study was designed to evaluate the effects of ajmaline on termination and reinduction of PSVT resulting from reentry within the atrioventricular node.

Subjects and methods

Ten patients, one man and nine women from 18 to 60 years old (mean 45.4 ± 13), comprised the study group and were selected on the following criteria: (1) history of electrocardiographically documented recurrent PSVT, (2) absence of pre-excitation during sinus rhythm on all available electrocardiograms, and (3) electrophysiologic documentation of atrioventricular nodal reentry during PSVT. All patients were free of organic heart disease. Two patients (Nos. 2 and 10) had incomplete right bundle branch block; all others had normal 12-lead electrocardiograms.

Electrophysiologic studies were performed in patients in the postabsorptive nonsedated state. All cardiovascular drug therapy was stopped a week before the study. Informed written consent was obtained from all.

The method of study has been published elsewhere.6 Six No. 6F bipolar catheters were introduced transvenously and positioned at high, mid, and low right atrium, proximal coronary sinus, tricuspid valve (for His bundle electrogram), and right
ventricular apex of each subject. Multiple surface and intracardiac electrograms were recorded on a VR-12 photographic recorder (Electronics for Medicine) or on a Mingograf 8-channel ink-jet recorder (Siemens Elema) at paper speeds of 100 and 50 mm/sec. Stimuli 2 msec in duration and approximately twice diastolic threshold were delivered by a programmable stimulator (Digitimer arrhythmia investigating system, Neurolog 4279). Conduction intervals and refractory periods were measured as defined by Wu et al.7

The study protocol included: (1) incremental ventricular pacing to a paced cycle length producing ventriculoatrial block, (2) programmed ventricular extrastimulation at decreasing coupling intervals at a driven cycle length shorter than the sinus cycle length, (3) incremental atrial pacing to a paced cycle length producing AH block, (4) programmed atrial extrastimulus testing at decreasing coupling intervals during one or more atrial paced cycle lengths, (5) induction of PSVT by rapid atrial pacing, atrial extrastimulation, or rapid ventricular pacing, and delineation of the limits of the reentrant circuit by analysis of mode of initiation of PSVT, antegrade and retrograde conduction times, and sequence of retrograde atrial activation, and (6) ventricular programmed extrastimulation during PSVT. In one patient (No. 4) intravenous atropine was required to induce sustained PSVT.

After the verification of the participation of the dual atrioventricular nodal pathway in PSVT, 50 mg of ajmaline was injected intravenously over a period of 30 sec. Blood pressure was closely monitored by cuff. Intracardiac and surface electrograms were continuously recorded at a paper speed of 100 (eight patients) or 50 mm/sec (two patients) from the beginning of the injection to allow study of the mode of break of PSVT. The last 10 cycles preceding the break were analyzed in detail to identify electrophysiologic events leading to termination of the tachycardia.

After break of PSVT was achieved, atrial and ventricular pacing and extrastimulus studies were repeated in the same sequence described earlier and an attempt was made to reinduce PSVT. Driving cycle lengths during extrastimulus testing were kept identical before and after the drug. All postdrug studies were completed within 25 min after administration of ajmaline.

Diagnosis of atrioventricular nodal reentrant tachycardia was made as previously described.6, 8, 9 Since this is mainly a diagnosis made by exclusion of other causes, care was taken to exclude patients in whom there was participation of concealed accessory pathways during tachycardia.9 Specifically, in all of the 10 patients atrial activation occurred either simultaneously with or slightly before the onset of ventricular activation during the tachycardia, suggesting that the ventricle was not an essential component of the reentry circuit.10

Statistical analysis was done with Student's t test for paired data. All results are expressed as mean ± SD.

Results

No untoward effects were observed after administration of ajmaline.

Control sinus cycle length ranged from 490 to 930 msec (mean 636 ± 150), AH interval from 50 to 100 msec (mean 67 ± 14), HV interval from 30 to 50 msec (mean 40.5 ± 6), and QRS duration from 60 to 110 msec (mean 84 ± 19).

Induction of PSVT. At control sustained PSVT could be induced in all the 10 patients and cycle length of the tachycardia ranged from 240 to 350 msec (mean 289 ± 44). All patients had PSVT characterized by antegrade conduction through the slow pathway and retrograde conduction through the fast pathway. One patient (No. 1) developed left bundle branch block during the tachycardia.

Termination of PSVT. PSVT was terminated in all 10 patients within 17 to 165 sec (mean 94 ± 49) after administration of ajmaline (table 1). The break was caused by the lack of an atrial response after a QRS response (retrograde fast pathway block) in eight subjects (Nos. 2 to 9; figure 1), and by the lack of a His potential after an atrial response (AH block) in one (No. 1). In patient No. 10, before break of atrioventricular nodal reentry a second short run of atrial tachycardia was initiated by a spontaneous occurrence of a premature atrial impulse and thus resulted in an increase in atrial rate. As the atrial impulse conducted down the atrioventricular node to capture the ventricle, atrioventricular nodal reentry was terminated.

Cycle length responses before termination of PSVT. After ajmaline cycle length of the PSVT was significantly prolonged, from 289 ± 44 msec at control to 373.5 ± 60 msec (range 263 to 464; p < .01), as a result of AH and HA prolongation (table 1). The AH interval increased from 250.5 ± 44 to 323 ± 57 msec (p < .01) and the HA interval from 38.5 ± 5.8 to 50.5 ± 6.4 msec (p < .001) after ajmaline. Compared with control the mean increase in AH interval of 31 ± 27% was not significantly different from the mean increase in HA interval of 32.4 ± 15.8%. In individual patients, however, the relative increase in antegrade conduction time (range 3.8% to 89%) and retrograde conduction time (range 12.5% to 66.6%) was quite variable and did not correlate with the mode of termination of tachycardia, which was blockade of retrograde pathway in most cases. Alternation in cycle length was observed in two patients (Nos. 2 and 10). Patient 2 had both AH and HA alternans, and the AH time of the short cycles after the drug was similar to that observed at control.}

**FIGURE 1.** Patient 6. Termination of PSVT by ventriculoatrial (VA) block followed by sinus escape (A	extsubscript{s}). The last QRS complex of PSVT is not followed by an atrial response. 877

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### TABLE I

Effect of ajmaline on termination of PSVT

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Control Before termination&lt;sup&gt;a&lt;/sup&gt; (range/mean)</th>
<th>Before termination</th>
<th>Increase in AH (%)</th>
<th>Control Before termination</th>
<th>Increase in HA (%)</th>
<th>Mode of termination</th>
<th>Time to termination (sec)</th>
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<tr>
<td>1</td>
<td>60</td>
<td>F</td>
<td>250 260–280/263</td>
<td>210 218</td>
<td>3.8</td>
<td>40 45</td>
<td>12.5</td>
<td>AH BI</td>
<td>17</td>
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<td>2</td>
<td>55</td>
<td>F</td>
<td>350 410–520/460&lt;sup&gt;b&lt;/sup&gt;</td>
<td>310 405</td>
<td>30.6</td>
<td>40 55</td>
<td>37.5</td>
<td>VA BI</td>
<td>165</td>
</tr>
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<td>39.1</td>
<td>40 50</td>
<td>25</td>
<td>VA BI</td>
<td>105</td>
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<tr>
<td>4</td>
<td>28</td>
<td>M</td>
<td>340 390–400/395</td>
<td>290 335</td>
<td>15.5</td>
<td>50 60</td>
<td>20</td>
<td>VA BI</td>
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<td>5</td>
<td>52</td>
<td>F</td>
<td>350 370–390/377</td>
<td>315 332</td>
<td>5.4</td>
<td>35 45</td>
<td>28.6</td>
<td>VA BI</td>
<td>55</td>
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<tr>
<td>6</td>
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<td>F</td>
<td>270 310–320/314</td>
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<td>18</td>
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<td>240 360–380/368&lt;sup&gt;c&lt;/sup&gt;</td>
<td>200 318</td>
<td>59</td>
<td>40 50</td>
<td>25</td>
<td>Intra-atrial reentry</td>
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</table>

Mean 289 373.5 250.5 323 31 38.5 50.5 32.4 94

±SD ±44 ±60 ±44 ±57 ±27 ±5.8 ±6.4 ±15.8 ±49

CL = cycle length.
<sup>a</sup>10 cycles.
<sup>b</sup>Both AH and HA alternans.
<sup>c</sup>AH alternans.

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**FIGURE 2.** For legend see opposite page.
dia occurred in the retrograde pathway after a short antegrade cycle (figure 2). Patient 10 showed alternation in the antegrade limb only.

**Escape rhythm and conduction intervals after termination of PSVT.** Conversion of the tachycardia was succeeded by sinus rhythm in eight patients (Nos. 2, 3, and 5 to 10). One patient (No. 1) showed a single junctional escape beat followed by sinus rhythm, and another (No. 4) developed sinus arrest with junctional rhythm (cycle length 800 msec) that persisted for 20 min after break of PSVT. Mean cycle length during sinus rhythm immediately after termination of PSVT was 583 ± 91 msec (range 500 to 765), not significantly different from control sinus cycle length of 636 ± 150 msec (p = NS). The AH interval (measured in nine) increased from a mean of 67.7 ± 15 to 88.8 ± 17 msec (range 70 to 110; p < .02) and the HV interval (measured in 10 subjects) also increased, from 40.5 ± 6 to 52 ± 9.5 msec (range 40 to 70; p < .01), after the drug. The QRS complexes widened significantly from 84 ± 19 to 114 ± 19.5 msec (range 90 to 140; p < .001) after ajmaline.

**Antegrade conduction properties (table 2).** Atrial paced cycle length producing AH Wenckebach block increased from 269 ± 30 msec (range 240 to 310) during control to 312 ± 44 msec (range 240 to 360) (p < .05) after ajmaline. The effective refractory period of the antegrade fast pathway ranged from ±240 to 320 msec (mean ±281 ± 31) before, and ±210 to 330 msec (mean ±275 ± 38; p = NS) after the drug. The effective refractory period of the antegrade slow pathway could be compared in only one patient (No. 10) and increased from 200 to 220 msec.

**Retrograde conduction properties.** The longest ventri-

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### TABLE 2

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Time of measurement</th>
<th>Antegrade (msec)</th>
<th>Retrograde (msec)</th>
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<td>Driving CL</td>
<td>FP-ERP</td>
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<tr>
<td></td>
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<td>230</td>
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</table>

C = control; A = after ajmaline; CL = cycle length; CL-AVB = longest atrial paced cycle length producing atrioventricular block; CL-VAB = longest ventricular paced cycle length producing ventriculoatrial block; ERP-VACS = effective refractory period of the ventriculoatrial conduction system.

*Effective refractory period could not be determined because ventriculoatrial conduction was abolished.

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**FIGURE 2.** Patient 2. Cycle length (CL) alternation preceding break of PSVT by ventriculoatrial (VA) block. Upper panel, induction of PSVT at A1A2 of 290 msec. PSVT is regular at a cycle length of 350 msec, AH of 310 msec, and HV of 40 msec. Lower panel, after ajmaline. The R-R intervals alternate between 410 and 520 msec. HV has increased to 50 msec. Both AH and HA alternans are present. The AH interval (350 msec) in short cycles is followed by the longer HA interval (60 msec). Long AH intervals (460 msec) are followed by shorter HA intervals (50 msec). Termination of PSVT occurs by VA block after a short AH cycle.
tricular paced cycle length that produced ventriculoatrial block increased from \( \leq 280 \pm 40 \) msec (range \( \leq 240 \) to 350) during control to \( 439 \pm 93 \) msec (range 270 to 590) after ajmaline (\( p < .001 \)). In four patients (Nos. 2, 3, 6, and 9) ventricular pacing at a cycle length just shorter than the sinus cycle length revealed complete abolition of ventriculoatrial conduction. In all four retrograde concealed conduction into the atrioventricular node was seen, suggesting that the atrioventricular node was the site of block (figure 3). The effective refractory period of the ventriculoatrial conduction system during control was \( \leq 241 \pm 42 \) msec (range \( \leq 210 \) to 330). The effective refractory period of the ventriculoatrial conduction system could be compared in only six patients (Nos. 1, 4, 5, 7, 8, and 10) and increased from \( \leq 230 \pm 35 \) msec (range \( \leq 210 \) to 300) to \( \leq 298 \pm 62 \) msec (range \( \leq 230 \) to 370) after ajmaline (\( p < .05 \)).

**Reinduction of PSVT.** In eight patients tachycardia could not be induced after ajmaline. This effect was directly related to depression of the retrograde fast pathway, as shown by the marked increase in ventricular paced cycle length producing ventriculoatrial block in all the eight and total abolition of ventriculoatrial conduction in four. In five of these patients no atrial echoes were noted despite the fact that AH intervals longer than the critical AH interval observed during control study were attained with either incremental atrial pacing or atrial extrastimulus testing. This suggests a retrograde weak link (figure 4). In two patients single atrioventricular nodal reentrant echo beats conducted to the ventricle but were not followed by a second atrial echo. In one patient (No. 8) additional effects of ajmaline on antegrade slow-pathway refractoriness also contributed to failure of induction of tachycardia (table 2).

During the control study in all the patients the cycle length of PSVT was longer than the atrial paced cycle length that produced AH block. Each of five of the eight patients in whom PSVT could not be reinduced after ajmaline had a cycle length of PSVT (before drug) similar to or longer than the atrial paced cycle length producing AH block after the drug. These findings are consistent with the observation that after ajma-

![FIGURE 3](http://circ.ahajournals.org/)

**FIGURE 3.** Effect of ajmaline on retrograde conduction in patient 6. **Upper panel,** Before ajmaline. One-to-one VA conduction at ventricular paced cycle length (CL) of 280 msec, and induction of PSVT on cessation of ventricular pacing. **Lower panel,** After ajmaline. VA conduction is abolished. Ventricular paced cycle length is 470 msec and sinus cycle length varies from 550 to 740 msec (normal sequence of atrial activation), showing VA dissociation. In addition, retrograde concealed conduction into atioventricular node (first sinus beat not followed by H, AH interval of second sinus beat of 150 msec, and third sinus beat of 110 msec) shows atioventricular node to be the site of block.
line the antegrade limb is not the limiting factor in the induction or sustenance of PSVT.

In two patients (Nos. 7 and 10) PSVT could be reinitiated after ajmaline. The cycle length of the tachycardia increased (from 260 to 310 and 240 to 290 msec, respectively) as a result of slowing of conduction in both limbs of the circuit. In both cases the AH interval increased by 40 msec and HA interval increased by 10 msec. In patient 7 the atrial paced cycle length producing AH block remained unaffected by the drug, while retrograde conduction was not sufficiently inhibited to prevent reinduction of PSVT. Although blood levels of ajmaline were not measured in our study, it is probable that in patient 10 the initiation of PSVT was related to the short duration of action of drug. Immediately after termination of arrhythmia by ajmaline, the ventricular paced cycle length producing ventriculoatrial block increased from 240 (control) to 370 msec. PSVT was, however, initiated during atrial extrastimulus testing 15 min after administration of ajmaline. On repeat incremental ventricular pacing 1:1 retrograde conduction could be achieved up to paced cycle length of 250 msec, suggesting that the effect of drug on retrograde conduction had significantly decreased.

Discussion

In patients with reentrant PSVT drugs that increase refractoriness of one or more components of the circuit can terminate or prevent induction of tachycardia. Ajmaline terminated PSVT in all of the 10 patients in our study. Prolongation of cycle length preceded termination of PSVT in all subjects. Although conduction was depressed in both the antegrade and retrograde limbs, the magnitude of depression in the two directions was quite variable in individual patients and did not correlate with the mode of termination, which in most cases was achieved by block in retrograde pathway. This suggests that, compared with the antegrade slow pathway, the retrograde fast pathway is more vulnerable to blockade by ajmaline. This effect of ajmaline resembles those of procainamide,11 quinidine,12, 13 and disopyramide,6, 14 all of which specifically depress the retrograde fast atrioventricular nodal pathway. Neither procainamide nor disopyramide, however, depress conduction through the antegrade slow pathway after

![Figure 4](image-url)

**FIGURE 4.** Patient 6. Upper panel, Induction of sustained PSVT by atrial extrastimulus (A1A2 = 230 msec) at control. Cycle length of PSVT is 270 msec. Lower panel, Inability to induce PSVT after ajmaline. At A1A2 of 300 msec the A2H2 (350 msec) is longer than the critical A2H2 (210 msec) that precipitated PSVT during control. No atrial echo is observed.
intravenous administration. Cycle length alternation before termination was observed in two patients. Possible explanations for alternating cycle lengths could be (1) the unmasking of a third intranodal pathway conducting the longer cycles, and (2) the occurrence of a form of resonance related to the effects of changes in cycle length on atrioventricular nodal conduction time. Recently, Ross et al. have demonstrated in a computer model that the cycle length alternation in a circus-motion tachycardia can be explained by the characteristics of a single curve of atrioventricular nodal function without postulating the presence of an additional antegrade accessory pathway.

Antegrade atrioventricular nodal conduction has been reported to be variably affected by ajmaline, while infra-Hisian conduction is depressed. The effective refractory period of the antegrade fast pathway was not significantly affected in the present study. Conduction through antegrade slow pathway, however, was depressed. The predominant effect of ajmaline was depression of retrograde conduction (reflecting conduction through the fast pathway), as demonstrated by the lowering of the paced ventricular rate necessary to produce ventriculoatrial block. The demonstration of retrograde concealed conduction in the atrioventricular node in four patients in whom no ventriculoatrial conduction was noted after ajmaline suggests that the atrioventricular node was the site of block. The effective refractory period of the ventriculoatrial conduction system was also increased by ajmaline.

In eight patients ajmaline inhibited induction of PSVT. Although the drug had demonstrable effects on both the antegrade and retrograde limbs of the reentrant circuit, prevention of PSVT was usually the result of an inability of the retrograde limb to support a circus movement, as evidenced by failure to induce atrioventricular nodal reentrant tachycardia or sustained PSVT despite achievement of an AH interval equivalent to or longer than the one that precipitated PSVT at control. In two patients reinduction of the PSVT could not be prevented because of less pronounced inhibition of the retrograde pathway. In both cases ajmaline failed to significantly lower the retrograde block rate. The half-life of intravenous ajmaline ranges from 3 to 8 min and its effect on cardiac tissue lasts for from 15 to 45 min.

The efficacy of other drugs in preventing induction of atrioventricular nodal reentrant PSVT has been reported to be as follows: intravenous propranolol 41% to 44%, intravenous ouabain 44% to 54%, intravenous ouabain plus propranolol 58%, intravenous procainamide 65%, oral quinidine 78%, oral disopyramide 67%, and intravenous disopyramide 60%. Intravenous verapamil converts 80% to 100% of episodes of PSVT into sinus rhythm, but is less effective in preventing induction of PSVT after oral administration. One peculiar feature observed in dual atrioventricular nodal pathways has been that drugs like procainamide, quinidine, and disopyramide, which inhibit the accessory pathway in Wolff-Parkinson-White syndrome, prevent atrioventricular nodal reentry by selective inhibition of the retrograde fast pathway. Ajmaline is also a strong suppressant of conduction through extranodal bypass tracts. The present study demonstrates its unidirectional depressant effects on the fast pathway in atrioventricular nodal reentrant PSVT.

Reports on toxicity of ajmaline at therapeutic doses refer mostly to its intravenous administration. Mild side effects include blinding of eyes, flushing, and nausea. Severe toxicity may lead to worsening of existing congestive heart failure, shock, and sinoatrial block and occasionally ventricular tachycardia, ventricular fibrillation, and asystole have been noted. The drug should be used with great caution in patients with conduction disturbances of the atrioventricular node and His Purkinje system.

In conclusion the results of our study show that intravenous ajmaline is highly effective in terminating acute episodes of PSVT mediated by dual atrioventricular nodal pathways. Further studies of the effect of oral ajmaline on induction of tachycardia are needed to evaluate its usefulness in preventing recurrence of PSVT in patients on long-term oral therapy.

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