Electrophysiologic Effects of Adenosine in Patients With Supraventricular Tachycardia

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Background—We correlated the electrophysiologic (EP) effects of adenosine with tachycardia mechanisms in patients with supraventricular tachycardias (SVT).

Methods and Results—Adenosine was administered to 229 patients with SVTs during EP study: atrioventricular (AV) reentry (AVRT; n = 59), typical atrioventricular node reentry (AVNRT; n = 82), atypical AVNRT (n = 13), permanent junctional reciprocating tachycardia (PJRT; n = 12), atrial tachycardia (AT; n = 53), and inappropriate sinus tachycardia (IST; n = 10). There was no difference in incidence of tachycardia termination at the AV node in AVRT (85%) versus AVNRT (86%) after adenosine, but patients with AVRT showed increases in the ventriculoatrial (VA) intervals (13%) compared with typical AVNRT (0%), P < 0.005. Changes in atrial, AV, or VA intervals after adenosine did not predict the mode of termination of long R-P tachycardias. For patients with AT, there was no correlation with location of the atrial focus and adenosine response. AV block after adenosine was only observed in AT patients (27%) or IST (30%). Patients with IST showed atrial cycle length increases after adenosine (P < 0.05) with little change in activation sequence. The incidence of atrial fibrillation after adenosine was higher for those with AVRT (15%) compared with typical AVNRT (0%), P < 0.001, or atypical AVNRT (0%) but similar to those with AT (11%) and PJRT (17%).

Conclusions—The EP response to adenosine proved of limited value to identify the location of AT or SVT mechanisms. Features favoring AT were the presence of AV block or marked shortening of atrial cycle length before tachycardia suppression. Atrial fibrillation was more common after adenosine in patients with AVRT, PJRT, or AT. Patients with IST showed increases in cycle length with little change in atrial activation sequence after adenosine. (Circulation. 1999;99:1034-1040.)

Key Words: adenosine ■ tachycardia, supraventricular ■ tachycardia, inappropriate sinus

The electrophysiologic (EP) effects of adenosine include depression of sinus node automaticity, shortening of the atrial myocyte action potential duration and refractory period, slowing of atrioventricular (AV) nodal conduction, and suppression of catecholamine-induced triggered activity.1–4 Its negative dromotropic effects have made adenosine clinically useful for the termination of supraventricular tachycardias (SVT).5–9 There are relatively little data involving systematic examination of electrophysiologic characteristics or modes of tachycardia termination after adenosine.7–9 Also, few studies have been published that provide detailed electrophysiologic evaluation for the effects of adenosine in less common arrhythmias like atypical AV nodal reentrant tachycardia (atypical AVNRT)10,11 or permanent junctional reciprocating tachycardia (PJRT).12,13 Finally, to our knowledge, there are no published data on the response to adenosine in patients with inappropriate sinus tachycardia (IST).

Thus, the goal of this study was to determine if the electrophysiologic response to adenosine predicts the mechanism of the underlying tachycardia.

Methods

Patients

We retrospectively reviewed the records of all patients with SVT who underwent EP study at our institution between January 1993 and August 1997. Patients with atrial fibrillation, atrial flutter, or automatic junctional tachycardia were excluded from study. A total of 394 patients with SVT (excluding aforementioned arrhythmias) were studied during this period, of whom 229 (58%) received intravenous adenosine for treatment or diagnosis of the arrhythmia. Seventy patients (7%) had evidence of organic heart disease. Our study group included 82 patients with typical AVNRT, 59 with atrioventricular reentry (AVRT), 13 with atypical AVNRT, 12 with PJRT, 53 with atrial tachycardia (AT), and 10 with IST.
Electrophysiologic Studies

All patients were studied in the postabsorptive state after an overnight fast. Antiarrhythmic drugs were discontinued at least 5 half-lives before the study. The EP study protocol was described in detail previously.14 Most patients with a left free wall accessory pathway and all with left AT foci underwent transseptal catheterization for left atrial access. A 12-lead surface ECG and bipolar intracardiac electrograms were simultaneously recorded from the high right atrium, coronary sinus, His bundle region, right ventricular apex, and left atrium (where appropriate), and were stored on the optical disk of a Prucka computer (Prucka International, Inc). Programmed stimulation was performed by the use of both atrial and ventricular overdrive and programmed stimulation.15 All patients showed tachycardia cessation with adenosine except for some with AT or IST.

For patients who had tachycardia cessation after adenosine, preliminary observations showed that the tachycardia cycle length (CL) was relatively stable until the last 4 beats, with the greatest CL change occurring in the last beat before cessation. Using each patient as their own control, we measured atrial electrograms from the high right atrial catheter or from the proximal atrial electrogram in the coronary sinus. Ventricular electrograms were measured from the onset of the QRS complex from the surface lead ECG. All measurements were made with electronic calipers by the same observer. For this study, we measured changes in the last 4 beats preceding tachycardia cessation with adenosine including changes in the atrial CL (ΔAA), changes in the atrioventricular conduction time (ΔAV), or changes in the ventriculoatrial conduction time (ΔVA). In tachycardias that did not cease, we compared the ΔAA in 6 consecutive atrial CL 30 seconds before and within 30 seconds after adenosine during the period of maximal CL change, using each patient as their own control. For inclusion in the study, the patients remained in stable SVT for at least 3 minutes before adenosine.

Definitions

Short R-P tachycardias with R-P/R-R<50% were divided into typical (slow-fast) AVNRT and AV-reentrant tachycardias (AVRT) by use of standard criteria.13 Long R-P tachycardias R-P/R-R>50% were divided into ATs, atypical AVNRT, and PJRT by standard criteria.15-19 For patients with AT, transient change in atrial activation sequence consistent with sinus rhythm was termed tachycardia suppression.20 Tachycardia cessation by means of block in either the AV node or accessory pathway was termed tachycardia termination.

AT Mechanisms

1. Automatic AT could not be initiated or suppressed with programmed stimulation, generally exhibited the warm-up phenomenon, occurred spontaneously, and could be incessant.16,20

2. Reentrant or triggered AT were regularly initiated and suppressed with programmed stimulation.20,21 A subset of reentrant arrhythmias consisted of patients with surgical incisional intra-atrial reentrant tachycardias (SIART) that occurred after atrial surgery for correction or palliation of congenital cardiac lesions.

Adenosine Administration

During episodes of tachycardia, intravenous (IV) adenosine (Fujiwasa Pharmaceutical Company) was given as a rapid bolus injection into a peripheral vein, followed by a 10-mL bolus of normal saline. Patients weighing >50 kg were initially treated with 6 mg of intravenous adenosine and, if no observable response occurred, doses of 12 mg or (rarely) 18 mg were used. For pediatric patients <50 kg, 100 to 400 μg/kg IV adenosine were used. A total of 73 of 229 patients (32%) received adenosine more than once during the same EP study. Of these, after adenosine, 62 of 63 (98.4%) had reproducible termination of tachycardia with a nonectopic atrial or ventricular complex on >1 occasion, and 10 of 73 (14%) had tachycardia termination with either a nonectopic atrial or ventricular complex on 1 occasion and with ectopic premature complexes on other occasions. Nonspecific termination of SVT with premature complexes after adenosine has been previously reported.8,14

Results

Short R-P Tachycardias: AVRT

Fifty-nine patients had an accessory AV pathway with atrioventricular reciprocating tachycardia (AVRT), of these, 22 of 59 (37%) had manifest conduction. Of these patients, 4 (7%) had decremental conducting pathways and 27 (46%) had concealed pathways. The location of the accessory pathways was left free wall in 43 (73%), right free wall in 7 (12%), and septal in 9 (15%). In twelve patients with AVRT, the tachycardia terminated with premature complexes (9 with atrial, 2 with ventricular, and 1 with junctional complexes) and were excluded from further analysis of cycle length change (Table 1). After adenosine, 40 patients with AVRT showed tachycardia termination in the AV node (40/47 or 85%) and 7 terminated retrogradely in the accessory pathway (7/47 or 15%), of which 2 of 7 (29%) were decrementally conducting (Table 2). Marked beat-to-beat oscillations or atrial cycle length alternans before tachycardia termination with adenosine were noted in 5 of 47 (11%). For the group, 6 of 47 (13%) had a significant change (>10 ms) in VA during the last 4 beats before tachycardia termination. The AV interval increased before termination at the AV node in 30 of 47 (64%) and in the retrograde pathway in 3 of 7 (43%) patients with AVRT. However, neither increases in AV nor in VA before termination predicted block in the AV node versus block in the accessory pathway (Table 2).

A total of 9 patients with AVRT (9/59, 15%) developed atrial fibrillation after adenosine (Figure 1). In 5 of the 9 patients, atrial fibrillation developed just after tachycardia termination, whereas in the remaining 4, orthodromic AVRT was interrupted by atrial fibrillation. Two of these patients had a prior history of spontaneous atrial fibrillation but none had a history of organic heart disease. Atrial fibrillation lasted an average 158 seconds (range, 6 to 681 seconds) and required direct-current (DC) cardioversion in 4 because of hemodynamic compromise. In the 5 patients with manifest preexcitation, the shortest preexcited R-R interval during

<table>
<thead>
<tr>
<th>TABLE 1. Tachycardia Mechanism</th>
<th>Patients, n*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short R-P tachycardias, n=118</td>
<td></td>
</tr>
<tr>
<td>AVRT</td>
<td>47</td>
</tr>
<tr>
<td>Typical AVNRT</td>
<td>71</td>
</tr>
<tr>
<td>Long R-P tachycardias, n=70</td>
<td></td>
</tr>
<tr>
<td>Atypical AVNRT</td>
<td>11</td>
</tr>
<tr>
<td>PJRT</td>
<td>11</td>
</tr>
<tr>
<td>AT-Automatic</td>
<td>24</td>
</tr>
<tr>
<td>AT-Triggered/reentrant</td>
<td>20</td>
</tr>
<tr>
<td>SIART</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate sinu tachycardia</td>
<td>10</td>
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</tbody>
</table>

*Excluded patients whose tachycardia was suppressed or terminated by premature complexes.
Atrial fibrillation was 225 ± 30 ms (mean ± SD) with an average preexcited R-R interval of 262 ± 40 ms.

**Typical AVNRT**

A total of 82 patients with typical AVNRT showed tachycardia termination after adenosine. Eleven terminated with premature complexes and were excluded from analysis (Table 1). A total of 61 of 71 (86%) (Figure 2A) terminated with antegrade slow pathway block whereas 10 of 71 (14%) terminated in the retrograde fast pathway (Figure 2B). Twenty-one patients (21/71 or 30%) displayed cycle length alternans before termination with adenosine (Figure 2A). The AV interval increased before termination in the retrograde pathway in 6 (6/10, 60%) patients with AVNRT. No patients with AVNRT developed atrial fibrillation after adenosine.

**Comparison Between Short R-P Tachycardias**

There was no statistically significant difference in the incidence of tachycardia termination in the AV node for patients with AVRT (40/47, 85%) and antegrade termination in the slow AV nodal pathway in patients with AVNRT (61/71, 86%); P > 0.05 (Table 2). There was a statistically significant higher risk for development of atrial fibrillation in patients with AVRT (9/59, 15%) compared with those with AVNRT (0/82, 0%); P < 0.001. Neither the mean dose of adenosine given (6.6 mg in AVRT versus 6.2 mg in AVNRT, P = 0.05) nor the incidence of premature complexes after adenosine differed between the 2 groups to explain this finding. The incidence of cycle length oscillations after adenosine was similar for both groups (AVRT, 11% versus AVNRT, 30%; P = 0.05). Finally, more patients with AVRT (13%) showed an increase in VA interval after adenosine than those with AVNRT (0%, P = 0.005).

**Long R-P Tachycardias: Atypical AVNRT**

Thirteen patients had atypical AVNRT and received adenosine, 2 of whom terminated with PVCs and were excluded from further analysis (Table 1). Two (18%) terminated.

### Table 2. Adenosine Response by Tachycardia Mechanism

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Suppressed/Terminated After Adenosine</th>
<th>Block with A by Atrial Deflection</th>
<th>ΔAA*</th>
<th>ΔAV*</th>
<th>ΔVA*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short R-P tachycardia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Typical AVNRT, n = 71</td>
<td>71/71 (100%)</td>
<td>61/71 (86%)</td>
<td>19±4</td>
<td>17±4</td>
<td>2±4</td>
</tr>
<tr>
<td>AVRT, n = 47</td>
<td>47/47 (100%)</td>
<td>40/47 (85%)</td>
<td>16±5</td>
<td>21±5</td>
<td>1±17</td>
</tr>
<tr>
<td>Long R-P tachycardia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atypical AVNRT, n = 11</td>
<td>11/11 (100%)</td>
<td>2/11 (18%)</td>
<td>18±5</td>
<td>20±6</td>
<td>14±52</td>
</tr>
<tr>
<td>PJRT, n = 11</td>
<td>11/11 (100%)</td>
<td>3/11 (27%)</td>
<td>46±31</td>
<td>32±43</td>
<td>25±20</td>
</tr>
<tr>
<td>AT-Automatic, n = 24</td>
<td>14/24 (58%)</td>
<td>5/24 (21%)</td>
<td>28±45</td>
<td>39±50</td>
<td>-11±53</td>
</tr>
<tr>
<td>AT-Triggered/reentrant, n = 20</td>
<td>6/20 (30%)</td>
<td>4/20 (20%)</td>
<td>14±49</td>
<td>18±36</td>
<td>-6±47</td>
</tr>
<tr>
<td>SIART, n = 4</td>
<td>1/4 (25%)</td>
<td>0/4 (0%)</td>
<td>10±20</td>
<td>23±49</td>
<td>0.5±24</td>
</tr>
</tbody>
</table>

*Mean ± SD, ms. ΔAA indicates mean change in atrial cycle length in ms comparing the fourth to the last beat prior to tachycardia suppression; ΔAV and ΔVA, mean change in AV and VA conduction in ms, as above. There is no statistically significant difference among the groups for each interval.

![Figure 1](image1.png)

**Figure 1.** Initiation of atrial fibrillation in a patient with orthodromic AVRT supported by a left free wall accessory pathway with adenosine. Simultaneous surface electrograms in lead V6 and intracardiac electrograms from the high right atrium (HRA), His bundle region (HBE), and coronary sinus (CS). AV intervals lengthen and VA intervals remain constant before termination in retrograde pathway. Sinus or ectopic atrial beat followed by atrial premature complex (APC) initiates atrial fibrillation with high grade block in AV node.

![Figure 2](image2.png)

**Figure 2.** Termination of typical (slow-fast) AVNRT with adenosine in 2 different patients. Surface electrogram lead I and intracardiac electrograms from high right atrium (HRA), His bundle region (HBE), and coronary sinus (CS) are displayed. Panel A demonstrates termination in the slow (antegrade) pathway. Marked oscillations in the AV intervals are observed before block. Note the reciprocal relationship between AV and VA before termination. The same activation sequence in atrial electrograms makes atrial premature complexes unlikely. Panel B shows that the greatest slowing of conduction occurs in the antegrade slow pathway conduction (increasing AV intervals) but tachycardia termination occurs in the retrograde fast pathway.
antegradely in the AV node and 9 (82%) in the retrograde direction (Figure 3). All demonstrated a progressive lengthening of atrial CL in the 4 beats before tachycardia termination (18 ± 65 ms, mean ± SD) (Table 2). Of those with retrograde termination, 7 showed progressive increases in VA and 2 showed variations in VA interval (Figure 3) during the 4 beats before termination with adenosine, whereas 2 also showed increases in the AV interval. No patients with atypical AVNRT developed atrial cycle length alternans, acceleration of atrial rate, or atrial fibrillation with adenosine.

**PJRT**

Twelve patients with PJRT received adenosine during tachycardia, 1 of whom terminated with an APC and was excluded from analysis (Table 1). Three (3/11, 27%) showed tachycardia termination in the AV node and 8 (73%) in the retrograde pathway. All demonstrated progressive increases in atrial CL before termination (46 ± 31 ms, mean ± SD) (Table 2). Seven patients (7/8, 88%) with termination in the retrograde direction showed progressive increases in the VA interval. Two patients with AV-node termination showed increases in both AV and VA intervals before tachycardia termination. No patient displayed cycle length alternans or shortening of the atrial CL during termination. Two patients with PJRT (2/12, 17%) developed atrial fibrillation after adenosine. Neither patient had a history of atrial fibrillation or organic heart disease and both required DC cardioversion to terminate the atrial fibrillation.

**AT: General Features**

Fifty-three patients had AT, 5 of whom terminated with premature complexes and were excluded from CL analysis, whereas 27 of 48 (56%) terminated or suppressed with adenosine. The mechanism of AT was automatic in 24 of 48 (50%), triggered or reentrant in 42%, and SIART in 8% (Table 1). In 9 of 27 patients (33%), the tachycardia suppressed with an atrial complex and with a ventricular complex in the rest. There was an increase in tachycardia cycle length in the fourth beat compared with the last beat before AT suppression (Table 2). Four (4/27, 15%) showed no change. The mode of AT suppression failed to correlate with tachycardia location. In patients who had tachycardia suppression with an atrial complex, 1 of 9 (11%) had a septal AT, 6 of 9 (67%) had a right-sided AT, and 2 of 9 (22%) had a left-sided focus, \( P > 0.05 \) between groups.

**AT: Suppression and Mechanism**

Of those patients with tachycardia suppression after adenosine, 14 of 24 (58%) had an automatic mechanism, 6 of 20 (30%) were triggered/reentrant, and 1 of 4 (25%) was SIART (Table 2). The mode of tachycardia suppression failed to correlate with tachycardia mechanism. In patients with suppression by an atrial complex, 5 of 24 (21%) had an automatic mechanism, 4 of 20 (20%) were triggered/reentrant, and 0 of 4 (0%) had SIART (Table 2).

**Comparison Between Long R-P Tachycardias**

Among the long R-P subtypes, there was no correlation between termination or suppression with an atrial complex after adenosine and tachycardia mechanism (AT 19%, PJRT 27%, and atypical AVNRT 18%; \( P > 0.05 \)). There was no significant difference among the groups with respect to changes in AA, AV, or VA after adenosine (Table 2). Patients with AT who showed tachycardia suppression were not more likely to exhibit a reciprocal relationship between changes in AV and VA after adenosine (12/48, 25%) versus those with tachycardia termination and atypical AVNRT (2/11, 18%) or PJRT (1/11, 9%); \( P > 0.05 \). Additional features that distinguished AT from both atypical AVNRT and PJRT were that only patients with AT showed either AV block (27%, \( P < 0.007 \)) (Figure 4), atrial CL alternans (23%, \( P < 0.02 \)), or marked decreases in the atrial CL after adenosine (2%, \( P > 0.05 \)).
Short R-P Tachycardias

We observed that ≈15% of patients with either AVNRT or AVRT will have tachycardia termination with a ventricular complex after adenosine and this alone cannot be used to determine the tachycardia mechanism. However, changes in VA interval after adenosine were seen only in those with AVRT (13%) and not with AVNRT (0%), which provides a useful clinical tool to differentiate between the short R-P tachycardias. Retrograde block in the accessory pathway occurred in both those with decrementally conducting pathways (2 patients) and in those without decremental conduction (5 patients). Previous reports have mainly emphasized tachycardia termination in the antegrade direction but clear-cut examples of retrograde block have also been described. We found marked beat-to-beat oscillations in CL before termination in patients with AVRT (11%) and in those with AVNRT (30%) (P > 0.05). Cycle length alternans was not seen in those with long R-P tachycardia due to atypical AVNRT or PJRT. Marked oscillation of tachycardia CL suggests a lengthening of the refractory period which then impinges on the tachycardia cycle length. The absence of marked oscillations in most patients with SVT supports the notion that adenosine may in large measure be effective by abrupt induction of conduction block rather than by lengthening the refractory period. The rapid onset and termination of adenosine effects preclude steady state measurements of conduction or refractoriness.

Adenosine-Induced Atrial Fibrillation

Previous studies have described induction of atrial fibrillation as an infrequent phenomenon for patients with SVT treated with adenosine. However, a recent study of 200 consecutive patients with SVT who were given adenosine during electrophysiologic evaluation reported atrial fibrillation in 11% of patients with typical, 10% of atypical AVNRT, and 13% with AVRT. We did not observe initiation of atrial fibrillation in patients with AVNRT but did find an incidence of atrial fibrillation in patients with accessory pathways (15% AVRT) similar to this study. This finding was true whether the AVRT pathways were manifest (38%) or concealed (62%) or associated with PJRT (17%). The differences in results may be partly due to the fact that all of their patients received 12 mg of adenosine centrally via a femoral sheath and >50% required isoproterenol to initiate tachycardia, whereas our patients received lower doses of adenosine (mean, 6.6 ± 3.4 mg), only 14% required 12 mg, and 36% required isoproterenol to initiate tachycardia. Additionally, there was no difference in the mean doses of adenosine used to terminate typical AVNRT versus AVRT nor in the incidence of premature complexes after adenosine. We speculate that the higher incidence of atrial fibrillation in patients with accessory pathways compared with patients with AVNRT observed in our study may be caused by both the decrease in atrial refractory period due to adenosine plus the anatomic substrate in AVRT provided by the arborization of the atrial accessory pathway insertion sites.

Long R-P Tachycardias

PJRT and Atypical AVNRT

The most important findings in our study relate to our observations in patients with long R-P tachycardias who had tachycardia termination after adenosine. We found that the mode of termination of adenosine in the long R-P subtypes did not predict the tachycardia mechanism. There was no significant difference between groups in the incidence of termination with an atrial complex. The magnitude of change in either AV or VA intervals after adenosine failed to predict the site of block.

Most prior reports on the use of adenosine in patients with long R-P tachycardias consist of relatively small numbers and have emphasized that use of the drug results in block either in the retrograde slow AV-nodal pathway or in the accessory pathway. The largest comparative trial reported for patients with long R-P tachycardias caused by PJRT or atypical AVNRT who were given adenosine consisted of 5 patients with atypical AVNRT and 5 with PJRT. In contrast, in our series we found that ≈20% of patients with atypical...
AVNRT and 30% of those with PJRT had tachycardia termination in the AV node. Our results support the preliminary observations of Hill et al., who studied patients with adenosine-sensitive AT and atypical AVNRT. They found that adenosine induced block in the antegrade (fast) pathway in 2 of 7 (29%) patients with atypical AVNRT. Our data support these observations and have extended these findings to patients with PJRT.

**AT**
A total of 21 patients with AT did not show AT cessation with adenosine, although 62% had associated AV-node block. Changes in atrial cycle length or cessation of tachycardia with an atrial complex after adenosine did not predict the anatomic location of the AT in our study. We hypothesized that these results might predict location because of the different embryologic origins for right versus left AT. Our observations highlight the difficulty in the use of adenosine to differentiate among the mechanisms of long R-P tachycardia. We demonstrate that in patients with atypical AVNRT or PJRT, either the antegrade nodal pathway or the retrograde pathway may prove to be the weak link of the tachycardia circuit. Similarly, we found that 33% of those with AT who had cessation after adenosine did so with an atrial complex. The most likely explanation is that adenosine simultaneously suppressed the AT focus while coincidentally inducing block at the AV node.

The key diagnostic feature that differentiated patients with AT from the other long R-P subtypes was the finding of adenosine induced AV block without suppression of the atrial focus (Figure 4). The presence of AV block during tachycardia excluded the diagnosis of PJRT and was never observed in those with atypical AVNRT. Additionally, patients with PJRT and atypical AVNRT did not display marked atrial cycle length alternans observed in 23% of patients with AT after adenosine. The latter feature may also serve to further differentiate AT from the other long R-P subtypes. Presumably, the baseline conduction and refractory properties intrinsic to the long R-P tachycardias helped determine the response to adenosine.

Our results are in accord with previous studies that showed tachycardia suppression after adenosine for patients with automatic ATs. Theoretically, the EP effects of adenosine should act to maintain reentrant circuits by shortening the atrial refractory period. One patient with SIART (clearly a reentrant mechanism) had tachycardia suppression after the drug. We speculate that the tachycardia circuit in this patient may have contained slow response or depolarized atrial tissue that was adenosine-sensitive.

**Inappropriate Sinus Tachycardia (IST)**
To our knowledge, our study is the first to report effects of adenosine in patients with IST. The response of IST to adenosine is consistent with what is known about the cellular actions of adenosine on automatic AT. Transient suppression followed by resumption of the automatic AT is expected because of hyperpolarization of the automatic focus, whereas a slowing of the IST cycle length is observed as predicted since the atrial cells are presumably not depolarized. Of interest was the observation that in spite of marked slowing of atrial cycle length, there was no (or only slight) shift in the IST focus after adenosine was given in the 6 patients for whom crista catheter recordings were available. Isoproterenol has been shown to shift the pacemaker focus superiorly along the crista terminalis, whereas enhanced vagal tone shifts the focus inferiorly. One possible explanation is that adenosine may depress automaticity throughout the pacemaker region relatively equally without a strong preferential depression of the dominant pacemaker, although differential sensitivity to adenosine in rabbit pacemaker cells was reported. Additionally, it is possible that higher doses of adenosine would indeed further suppress and shift the dominant pacemaker focus. Finally, we cannot exclude the possibility that the crista catheter might have shifted slightly during adenosine administration.

**Study Limitations**
This study represents a retrospective analysis of all patients with SVT referred to our center for EP study and ablation between January 1993 and August 1997 who received adenosine during tachycardia and therefore may represent a somewhat biased population not generalizable to all patients with SVT. However, for patients with short R-P tachycardias, this possibility seems unlikely given that the incidence of both pathway location and SVT mechanism described in our study compares favorably to those in the published literature. Our series would appear to be biased somewhat by inclusion of larger numbers of patients with less common tachycardias (such as IST, PJRT, and AT), but this is actually reflective of our particular referral pattern. Also, there were insufficient data at times to regularly distinguish between triggered versus reentrant ATs. Regardless of these considerations, we were able to confirm the atrial origin of the tachycardia and to describe the effect adenosine has on the tachycardia. This limitation does not extend to the examination of the effects adenosine has on the short R-P, other long R-P, or inappropriate sinus tachycardias.

**Clinical Implications**
We found that the response to adenosine for tachycardia termination proved to be of limited value in defining either the mechanism of the SVT or the anatomic location of the AT. For patients with short R-P tachycardias, the presence of changes in VA intervals before termination after adenosine predicted AVRT as the tachycardia mechanism but occurred in only 13% of patients. We reported a high incidence of atrial fibrillation in patients with AVRT compared with typical AVNRT who were given adenosine, which emphasize the desirability of an available defibrillator when such patients are given adenosine. The mode of termination for the long R-P tachycardias was similar among the subtypes. However, features favoring AT include the presence of AV block (27%) and atrial CL oscillations (23%) before suppression with adenosine. Finally, we describe the first report, to our knowledge, in the use of adenosine for patients with IST. In these patients, we found slowing of the tachycardia cycle length with only a slight shift in the atrial focus.
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References


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