Adenosine and Pediatric Supraventricular Tachycardia in the Emergency Department: Multicenter Study and Review

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Study objective: To determine the frequency of successful cardioversion and the adverse effects of adenosine treatment in pediatric emergency department patients with supraventricular tachycardia (SVT).

Methods: This was a multicenter descriptive study with both prospective (convenience sample) and retrospective (chart review) patient entry. The setting was 7 urban pediatric EDs with a yearly census range of 22,000 to 70,000 visits. Pediatric patients 18 years of age and younger who received intravenous adenosine for presumed SVT were eligible.

Results: Six investigators from 7 pediatric EDs entered 82 patients with 98 presumed SVT episodes (52 prospective and 46 retrospective) into the study. Twenty-five episodes occurred in children younger than 1 year of age. Eight patients had congenital heart disease, 59 had a history of SVT, 43 were taking cardiac medications (digoxin in 27), 13 had a history of asthma, and 25 presented in compensated cardiogenic shock. A total of 193 intravenous doses of adenosine were administered; doses were classified as low (<.1 mg/kg [n=18]), medium (.1 to <.2 mg/kg [n=116]), or high (≥.2 mg/kg [n=59]). The dose range was .03 to .5 mg/kg, and only 2 doses were higher than .3 mg/kg. A total of 95 patient-events were determined to be SVT, all but 5 of which were atrioventricular (AV) node–dependent; 3 events were ventricular tachycardia. The overall cardioversion success rate of adenosine was 72% (71/98), and that for AV node–dependent SVT was 79% (71/90). Cardioversion was successful for 4 patient-events at a low dose, 44 at a medium dose, and 23 at a high dose of adenosine. Adverse effects occurred in 22 patients, and no patient had bronchospasm or hemodynamically significant arrhythmia.

Conclusion: Intravenous administration of adenosine led to successful cardioversion in 72% of pediatric ED patient-events that were presumed to be SVT. A dose range of .1 to .3 mg/kg was found to be most effective. Adenosine was not associated with significant adverse effects.

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INTRODUCTION

Supraventricular tachycardia (SVT) occurs in 1 of 250 to 1 of 1,000 children, making it the most common arrhythmia in the pediatric population. Adenosine is an endogenous nucleoside that transiently blocks atrioventricular (AV) conduction in the heart.[1-5] Almost 90% of SVT in children is based on a re-entrant mechanism.[6] For these reasons, adenosine should be effective for the termination of most pediatric SVT episodes. Reports of the use of adenosine in children are limited to hospitalized patients, many of whom received adenosine during electrophysiologic testing.[7-14] No studies have focused on the use of adenosine in the pediatric emergency department, where many episodes of SVT are treated. Despite the lack of studies, adenosine has become first-line therapy for SVT in the pediatric patient who presents to the ED.[15]

The purpose of this study was to determine the frequency of successful cardioversion and the adverse effects of adenosine treatment in pediatric ED patients with SVT. The associations of several clinical variables with successful cardioversion and with adverse effects were evaluated.

MATERIALS AND METHODS

Through the Collaborative Research Committee, Emergency Medicine Section, American Academy of Pediatrics, 6 of 23 pediatric emergency medicine physician committee members offered to participate in this study. The study protocol was approved by the review board of each center at which patients were entered prospectively. Consent beyond the standard consent for treatment in the ED was waived because adenosine is the drug of choice for SVT.

Children from birth to 18 years of age who received intravenous adenosine for treatment of presumed SVT in a participating pediatric ED were eligible. Patients were enrolled prospectively by convenience sample and retrospectively by chart review based on the presence of Clinical Modification Code 427 (cardiac dysrhythmias) of the *International Classification of Diseases,* 9th revision. ED census logs were not reviewed. Patients were excluded from the study if one or more of the following conditions were present: congestive heart failure with uncompensated shock (hypotension for age), atrial fibrillation, atrial flutter or sick sinus syndrome, use of carbamazepine and dipyridamole (both are adenosine uptake inhibitors and prolong the effects of adenosine[2]), or use of methylxanthines (which decrease the effects of adenosine by blocking adenosine receptors[2]). SVT was defined as a narrow QRS complex tachycardia with a fixed RR interval, absence of normal P waves, and a rate exceeding the upper limit of sinus tachycardia for age (< 1 year, 180; 1 to 2 years, 150; 3 to 12 years, 140; 13 to 18 years, 120).

Demographic information included age, sex, race, name of treating hospital, date of treatment, presence of structural heart disease (except patent ductus arteriosus or spontaneously closed ventricular septal defect), previous cardiac surgery (except closure of patent ductus arteriosus in a premature infant), previous ECG-documented SVT, medications taken within the past 24 hours, and history of wheezing requiring bronchodilators within the past year. Clinical interventions recorded before treatment included vital signs, symptomatic complaints, and physical examination findings.

Compensated cardiogenic shock was defined as normal systolic blood pressure for age (< 2 years, 70; 2 to 12 years, 80; 13 to 18 years, 90 mm Hg) or higher, associated with 1 or more of the following: respiratory distress, altered level of consciousness, anxiety, irritability, diaphoresis, pallor, or hepatomegaly. Respiratory distress was defined as the presence of 1 or more of the following: retractions, rales, or tachypnea (breaths per minute) for age (<1 year, >60; 1 to 2 years, >50; 3 to 12 years, >40; 13 to 18 years, >30).

Documentation of interventions included a 12-lead ECG before and after conversion to sinus rhythm for patients with unknown conduction mechanism of SVT, a 12-lead ECG or lead II rhythm strip during conversion (reading of ECGs was not blinded), number of adenosine doses, adenosine dosage, and other modes of treatment (drugs versus synchronized electrical cardioversion).

Doses of adenosine were administered by rapid intravenous bolus followed by 5 to 10 mL of flushing with normal saline solution. The recommended first dose was .1 mg/kg, with a maximum dose of 6 mg. Subsequent doses were .2 mg/kg, with a maximum single dose of 12 mg. Adenosine treatment success was defined as SVT converted to sinus rhythm for a minimum of 5 minutes.

The following were considered systemic adverse effects of adenosine if they occurred within 5 minutes of administration: bronchospasm, dyspnea, cough, chest pain, flushing, headache, nausea, or vomiting. Cardiac adverse effects included the following if they were present for longer than 10 seconds after adenosine administration: asystole,

bradycardia for age (<2 years, <100; 2 to 12 years, <70; 13 to 18 years, <60), AV block, atrial fibrillation, atrial flutter, ventricular tachycardia, or ventricular fibrillation.

The medical records were reviewed to ascertain type and mechanism of tachycardia, and therefore the exclusions for arrhythmias were determined retrospectively. Tachycardias were classified as ventricular or supraventricular. Supraventricular tachycardia was further divided into AV node–dependent and non–AV node-dependent types, the latter including junctional tachycardia, ectopic atrial tachycardia, atrial flutter, atrial fibrillation, sinus tachycardia, and sinus node reentry.

Summary statistics included absolute numbers and percentages for successful conversion and adverse effects. Univariate analyses (χ^2 or Fisher's exact test, 2-tailed), with success as an outcome, were used to identify the individual variables with the greatest association. A probability value lower than .05 was considered to be indicative of a significant effect.

RESULTS

Six pediatric emergency medicine physicians from 7 urban pediatric EDs (annual census, 22,000 to 70,000 visits) participated. There were 82 patients and a total of 98 presumed SVT events. One patient had 5 events, 3 patients had 3 events each, and 6 patients had 2 events each. There were 52 prospective and 46 retrospective patient-events. The range of patient entry dates for each investigator is presented in Table 1. Prospective and retrospective cases were combined, and total patient-events were used for data analysis. No patient was excluded because of concurrent use of dipyridamole, carbamazepine, or methylxanthines.

Of the 98 patient-events, 54 (55%) occurred in boys. Sixty-six occurred in white patients, 22 in black patients, 6 in Hispanics, and 4 in patients of other ethnic backgrounds. Twenty-five events (26%) occurred in children younger than 1 year of age. In 8 cases (8%), congenital heart disease was present; 7 patients (7%) had undergone cardiac surgery; and 43 (44%) were taking cardiac medications, including 27 who were taking digoxin. In 59 cases (60%), there was a history of SVT; in 13 (13%), there was a history of asthma. In 25 events (26%), the patient presented in compensated cardiogenic shock. Vagal maneuvers were performed in 45 cases; in 6 of these, the rhythm temporarily converted to sinus rhythm but reverted to SVT.

Ninety-five events were SVT, and 3 were ventricular tachycardia. Of the SVT events, 90 were AV node-dependent and 5 were non–AV node-dependent. The latter group included 2 cases of atrial tachycardia, 1 automatic atrial tachycardia, 1 ectopic atrial tachycardia, and 1 junctional tachycardia. Twenty-two (24%) of the AV node-dependent SVT events were caused by Wolff-Parkinson-White (WPW) syndrome.

One to 4 doses of adenosine were administered per episode. Of the 193 doses of adenosine administered, 18 were classified as low dose (<.1 mg/kg), 116 as medium dose (.1 to < than .2 mg/kg), and 59 as high dose (≥.2 mg/kg). The range was .03 to .5 mg/kg, or .23 to 12 mg. Only 2 doses were greater than .3 mg/kg. The patient was initially treated with a low dose in 16 episodes, with a medium dose in 78 episodes, and with a high dose in 4 episodes. Additional doses were equal to or greater than preceding doses for patients receiving more than 1 dose. The number of doses administered per patient event averaged 2.44, 1.90, and 1.5 for patients who initially received low, medium, and high doses, respectively.

The overall success rate of adenosine per patient-event in presumed SVT was 71 (72%) of 98. Of the 90 AV node-dependent SVT events, 71 (79%) were treated successfully. In patients with non–AV node-dependent SVT or ventricular tachycardia, cardioversion with adenosine was not successful. Demographic, clinical, and therapeutic factors in relation to success of treatment are summarized in Table 2. There were no significant associations between any of these factors and treatment success. The medium- and high-dose ranges of adenosine resulted in greater success rates than did the low-dose treatment. Among the 10 patients with multiple events, cardioversion with adenosine treatment was successful in 23 events (88%).

Adverse effects occurred in 22 of the 98 cases; no patient with more than 1 event had recurrent adverse effects. The most common noncardiac adverse effect (8 patients) was

Table 1.
Method of patient entry and dates.

Investigator	Patients Entered (Prospective/Retrospective)	Date Range
1	27 (9/18)	09/03/92–04/28/95
2	26 (16/10)	09/10/91–09/15/96
3	9 (9/0)	02/26/92–03/16/94
4	12 (12/0)	08/19/93–04/12/95
5*	12 (6/6)	10/18/91–09/08/95
6	12 (0/12)	10/17/91–10/02/95
Total	98 (52/46)	

*Two different EDs.

vomiting (Table 3). Dyspnea occurred in 2 patients, neither of whom had a history of asthma. Both patients with arrhythmia had self-limited bradycardia (duration, 5 and 15 seconds) before successful conversion to sinus rhythm. The adverse effect probability values were not determined due to the low prevalence of adverse effects. Trends included a greater rate of adverse effects in children 1 year of age or older, in those who had undergone cardiac surgery, and those who were not in cardiogenic shock (Table 2). The number of adverse events was similar in the medium- and high-dose groups and did not depend on the number of doses received.

The 19 AV node–dependent SVT events unsuccessfully treated with adenosine were converted to sinus rhythm with the following treatments: procainimide (3), verapamil (3), β-blocker (2), digoxin (2), flecainide (2), further doses of adenosine during hospitalization (1), electrical synchronized cardioversion (1), esophageal overdrive pacing (1), spontaneous cardioversion (1), and unknown (3). There were no deaths in this study population.

DISCUSSION

The overall success rate for conversion of 98 presumed SVT events in 82 pediatric patients treated in the ED was 72%. This compares with a rate of 77% (90/117) reported by Till et al[10] in 50 hospitalized pediatric patients (1 to 17

Table 2.
Patient characteristics and results of treatment with adenosine.

Category	Factor	No. of Patient-Events	Successful Cardioversion No. (%)	*P*	Adverse Effects No. (%)
Demographic					
Sex	Male	54	40 (74)	.690	11 (20)
	Female	44	31 (70)		11 (25)
Race	White	66	46 (70)	.235	13 (20)
	Black	22	15 (68)		9 (41)
	Hispanic	6	6 (100)		0
	Other	4	4 (100)		0
Age (y)	<1	25	17 (68)	.564	1 (4)
	≥1	73	54 (74)		21 (29)
Clinical					
Congenital heart disease	Yes	8	6 (75)	.866	4 (50)
	No	90	65 (72)		18 (20)
Cardiac surgery	Yes	7	5 (71)	1*	4 (57)
	No	91	66 (73)		18 (20)
Cardiac medications	Yes	43	33 (77)	.400	10 (23)
	No	55	38 (69)		12 (22)
Digoxin	Yes	27	22 (81)	.217	5 (19)
	No	71	49 (69)		17 (24)
Prior SVT	Yes	59	46 (78)	.133	13 (22)
	No	39	25 (64)		9 (23)
WPW syndrome	Yes	22	18 (82)	.264	3 (14)
	No	76	53 (70)		19 (25)
Asthma	Yes	13	12 (92)	.085	1 (8)
	No	85	59 (69)		21 (25)
Cardiogenic shock	Yes	25	20 (80)	.328	2 (8)
	No	73	51 (70)		20 (27)
Therapeutic					
No. of doses	1	98	34 (35)	.919	7 (7)
	2	60	24 (40)		8 (13)
	3	29	11 (38)		6 (21)
	4	6	2 (33)		1 (17)
Dosage	Low	18†	4 (22)	.401	0
	Medium	116†	44 (38)		10 (9)
	High	59†	23 (39)		12 (20)

*Fisher's exact test, 2-tailed.
†Total number of doses given.

years of age). Till et al reported an 86% (88/102) success rate for AV node–dependent SVT, compared with 79% in our study. Two ED studies of adult patients showed overall success rates of 54% and 85% and AV node–dependent SVT success rates of 85% and 96%.[16,17] Five prehospital care studies of adult patients reported adenosine success rates ranging from 45% to 68%, with AV node–dependent SVT success rates of 73% to 90%.[18-22]

Our study, like others, showed no decrease in adenosine effectiveness in the presence of WPW, congenital heart disease, or concurrent digoxin use.[8,10] The 68% success rate for children younger than 1 year of age was lower than the rate of 74% for older children, but the difference was not significant. This is consistent with a report by Dorostkar et al,[14] who indicated a 60% success rate for infants. Possible explanations for this lower success rate are that smaller-gauge intravenous catheters used in infants may not permit rapid delivery of adenosine and that the AV nodes of infants may be relatively more resistant to adenosine.

The effectiveness of adenosine in the treatment of pediatric patients with compensated cardiogenic shock (normal systolic blood pressure for age) secondary to SVT has not been reported previously. We found the success rate of adenosine treatment for 25 patients in compensated cardiogenic shock to be similar to the rate for patients without cardiogenic shock (80% versus 70%, respectively). No significant adverse effects occurred in patients with compensated cardiogenic shock. Studies of adults with SVT and associated hypotension (systolic pressure <90 mm Hg) reveal that the presence of hypotension does not increase the incidence of adverse effects or alter the effectiveness of adenosine in achieving cardioversion and normal blood pressure.[16,18,22,23]

In our study, the rate of success for medium- or high-dose adenosine (≥.1 mg/kg) was 38%, compared with 22% for low-dose adenosine (<.1 mg/kg). Also, initial low-dose treatment resulted in a greater number of doses being given than when the starting dose was .1 mg/kg or higher. Of the 8 doses of .3 mg/kg or more (administered to 8 different patients), only 1 (at .3 mg/kg) was successful. Five of these 8 patients had AV node–dependent SVT, and 3 had VT. The Emergency Cardiac Care Committees and the American Heart Association recommend a dose of .1 to .2 mg/kg.[24] Our study supports a dose range of .1 to .3 mg/kg (maximum, 12 mg) as most effective for treatment of SVT. If higher doses are unsuccessful, a non–AV node–dependent arrhythmia should be considered. Higher doses may be necessary for patients receiving theophylline or aminophylline. Berul[25] reported that a 16-day-old female infant with WPW who was receiving aminophylline and caffeine required .4 to .8 mg/kg per dose of adenosine for cardioversion of SVT after multiple doses less than .4 mg/kg were unsuccessful.

In our study, 32 adverse effects occurred in 22 (22%) of 98 patient-events. Eight patients had more than 1 adverse effect. Vomiting, the most common adverse effect, was reported in 8 patients. No sustained arrhythmia or bronchospasm was observed in our study. Overholt et al[11] reported adverse effects in 6 pediatric patients (25%), including 5 with dyspnea, flushing, or irritability and 1 with bradycardia. No other pediatric study has delineated nonserious adverse effects. Studies of adult patients that listed nonserious adverse effects and limited the maximum adenosine dose to 12 mg have reported adverse effects rates of 9.3% to 26.6%. In these studies, the most common adverse effect was chest pain or discomfort; this complaint accounted for 36% of the nonserious adverse effects. These effects were transient (<1 min) and were associated with successful cardioversion or AV block.[17-19,22]

Factors in our study that had a greater rate of adverse effects of adenosine treatment were age 1 year or older, absence of cardiogenic shock, and cardiac surgery. The greater rate of adverse effects in older children can probably be attributed to the subjective nature of adenosine's adverse effects (nausea, light-headedness, dizziness, impression of impending doom, weakness, headache, and chest pain or discomfort). Younger children would be less likely to report these adverse effects. The greater rate of adverse effects in patients with noncardiogenic shock is possibly related to the fact that the signs of cardiogenic shock are similar to those of adverse effects, so that adverse

Table 3.
Type and frequency of adverse effects.

Effect	No. of Patients
Nausea/vomiting	4
Vomiting	3
Chest pain	3
Nausea	2
Headache	2
Flushing	2
Chest pain/flushing	1
Chest pain/flushing/headache	1
Dyspnea	1
Dyspnea/chest pain	1
Arrhythmia*	1
Arrhythmia/nausea/vomiting†	1

*Bradycardia, 15 s.
†Bradycardia, 5 s.

effects are more apparent in the patients without noncardiogenic shock.

The greater rate of adverse effects among patients with previous cardiac surgery is consistent with reports showing that cardiac transplantation patients are hypersensitive to adenosine. Ellenbogen et al[26] studied the effects of adenosine in 28 orthotopic cardiac transplantation patients and found that the denervated transplanted donor sinus and AV nodes demonstrated an increased duration of electrophysiologic effect. In a case series by Crosson et al[27] of 38 children with 50 episodes of SVT undergoing electrophysiologic testing, 1 cardiac transplantation patient became asystolic with adenosine and required 1 minute of resuscitation. Because of these findings, the recommended adenosine dose given to transplantation patients to treat SVT is approximately 67% to 80% of the generally recommended dose.[26,27] Our findings indicate that this reduced dose recommendation may also be applicable to children who have previously undergone cardiac surgery.

Clinically significant cardiac adverse effects associated with adenosine are extremely uncommon in children but have been reported. ED personnel must be prepared to treat life-threatening arrhythmias when administering adenosine. In our report, 2 patients had episodes of bradycardia lasting less than 1 minute. Till et al[10] reported that 1 patient experienced a 40-second bradycardic adverse effect. In a case series of 25 pediatric patients (age range, 6 hours to 17 years), a 10-year-old child with Down's syndrome, AV canal, and pulmonary hypertension had sinus bradycardia for 2 to 3 minutes and required temporary pacing.[11] Ventricular fibrillation is another, very uncommon adverse reaction to adenosine. Mulla and Karpawich[28] reported that an 8-day-old child with WPW treated with digoxin received .2 mg/kg of adenosine for an SVT episode after no response was obtained with smaller doses. The patient had asystole for 1 second, followed by ventricular fibrillation and hypotension that was converted to sinus rhythm by electrical defibrillation.

The most serious noncardiac adverse effect of adenosine is bronchospasm. Adenosine causes bronchospasm by enhancing the release of preformed mediators from mast cells via the adenylate cyclase receptor.[29] Mast-cell stimulation can cause immediate or delayed bronchospasm. Adenosine has not been associated with bronchospasm in patients who do not have asthma. DeGroff and Silka[30] described a 13-year-old child with asthma who had had no asthma episodes in 3 years but experienced severe bronchospasm 90 seconds after a 12-mg dose of intravenous adenosine during electrophysiologic testing. The bronchospasm was reversed with subcutaneous epinephrine and inhaled albuterol. In contrast to adenosine-related bronchospasm, Cook et al[31] reported conversion to sinus tachycardia with administration of .1 mg/kg adenosine in a 4-year-old with albuterol-induced SVT. None of the patients in our study had bronchospasm, and this group included 13 asthmatic patients. From our experience, asthma is not a contraindication to adenosine, although emergency physicians should be prepared to treat immediate bronchospasm and to consider delayed bronchospasm when managing asthmatic patients who receive adenosine treatment.

In our study, 3 patients (3%) had ventricular tachycardia misinterpreted as SVT. Two patients received doses of .1, .2, and .3 mg/kg, and 1 patient received doses of .2 and .3 mg/kg. One patient complained of chest pain, and none of these patients had successful cardioversion with adenosine. Crosson et al[27] described 2 children (5%) with ventricular tachycardia misinterpreted as SVT. One had successful cardioversion with adenosine, and no adverse effects occurred. In prehospital and ED studies of adult patients, 10 (1.4%) of 691 patients had ventricular tachycardia misinterpreted as SVT and were treated with adenosine. Conversion was successful in 1 patient, and no adverse effects were reported.[16-22] Therefore adenosine treatment of ventricular tachycardia misinterpreted as SVT does not appear to be associated with serious adverse effects.[32]

Our study has 2 limitations. First, the original design was prospective, but retrospective patients were added to increase the study population and to include patients not entered prospectively. The 2 centers that entered patients only prospectively (21 patient-events) may have missed eligible patients. Therefore the results of this study might not be generalizable to the target population (pediatric patients with presumed SVT presenting to the ED). Second, clinical factors not recorded in the medical records of patients entered into the study retrospectively were considered to be negative. For this reason, the true number of nonserious adverse effects among such patients may have been greater than reported, although the rate of adverse effects was similar for patients entered retrospectively (20%) and those entered prospectively (25%).

In summary, adenosine is effective treatment for children who present to an ED with presumed SVT. Adenosine doses of .1 to .3 mg/kg (maximum dose, 12 mg) were more successful than doses of less than .1 mg/kg. Life-threatening adverse effects of adenosine treatment were not found in this study. However, emergency physicians must be prepared to treat hemodynamically compromising arrhythmias for all patients and immediate or delayed bron-

chospasm for asthma patients when administering adenosine.

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