Adenosine for wide-complex tachycardia: Efficacy and safety*

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Objectives: To determine whether adenosine is useful and safe as a diagnostic and therapeutic agent for patients with undifferentiated wide QRS complex tachycardia. The etiology of sustained monomorphic wide QRS complex tachycardia is often uncertain acutely.

Design: A retrospective observational study.

Setting: Treatment associated with emergency visits at nine urban hospitals.


Interventions: Treatment with adenosine infusion.

Measurements and Main Results: Measured outcomes included rhythm response to adenosine, if any, and all adverse effects. A positive response was defined as an observed change in rhythm including temporary atrioventricular conduction block or tachycardia termination. A primary adverse event was defined as emergent electrical or medical therapy instituted in response to an adverse adenosine effect. A rhythm diagnosis was made in each case. The characteristics of adenosine administration as a test for a supraventricular as opposed to ventricular tachycardia were determined, and the adverse event rates were calculated. A total of 197 patients were included: 104 (90%) of 116 (95% confidence interval, 93%–95%) and two (2%) of 81 (95% confidence interval, 0.3%–9%) supraventricular tachycardia and ventricular tachycardia patients demonstrated a response to adenosine, respectively. The odds of supraventricular tachycardia increased by a factor of 36 (95% confidence interval, 9–143) after a positive response to adenosine. The odds of ventricular tachycardia increased by a factor of 9 (95% confidence interval, 6–16) when there was no response to adenosine. The rate of primary adverse events for patients with supraventricular tachycardia and ventricular tachycardia was 0 (0%) of 116 (95% confidence interval, 0%–3%) and 0 (0%) of 81 (95% confidence interval, 0%–4%), respectively.

Conclusions: Adenosine is useful and safe as a diagnostic and therapeutic agent for patients with regular wide QRS complex tachycardia. (Crit Care Med 2009; 37:2512–2518)

KEY WORDS: tachycardia, ventricular; tachycardia, supraventricular; adenosine; anti-arrhythmia agents; diagnostic techniques; cardiovascular; adverse effects

Patients presenting with electrocardiographic (ECG) evidence of regular wide QRS complex tachycardia (WCT) pose both diagnostic and therapeutic challenges. Clinicians focus on distinguishing tachycardias with a supraventricular vs. a ventricular origin. A number of historical factors, such as a history of myocardial infarction (MI), physical examination findings, and ECG indicators like atrioventricular (AV) dissociation, can be helpful to distinguish the tachycardia origin, but none of these individual characteristics is both highly sensitive and specific, and combined algorithms have not proven reproducible (1–6). Medicines, such as procainamide and amiodarone, can be used to treat either type of underlying rhythm, but these agents may be poorly effective in some cases or have potential adverse effects, such as hypotension. Electrical cardioversion is both safe and effective but it can be painful without adequate sedation, does not prevent recurrence, and provides limited diagnostic information.

Adenosine, an endogenous nucleotide, causes rapid AV nodal blockade when administered intravenously with an ultrashort 9-sec half-life. Investigators have studied its utility to diagnose and treat regular WCT in small series of up to 30 patients primarily with tachycardias induced in the electrophysiology laboratory (7–10). It is expected that adenosine will 1) terminate the majority of supraventricular tachycardias (SVTs) that rely on the AV node to form a reentrant circuit; 2) cause transient AV block to permit the accurate diagnosis of atrial flutter; and 3) be unlikely to terminate most ventricular tachycardias (VTs). Nevertheless, use of adenosine has been tempered by a number of safety concerns including paradoxical enhancement of accessory tract conduction and acceleration of the ventricular response in the setting of atrial flutter or fibrillation with a bypass tract, persistent bradycardia or asystole, change in rhythm to atrial fibrillation, degeneration to ventricular fibrillation, myocardial ischemia due to coronary artery vasodilation and a secondary steal syndrome, or bronchospasm (11–16). It was hypothesized that the presence or absence of a change in heart rhythm in response to intravenous adenosine infusion would be a useful and safe way of distinguishing a supraventricular from ventricular origin in patients with stable, sustained regular WCT.
![Image](https://example.com/image.png)

**Table 1. Enrollment information**

<table>
<thead>
<tr>
<th>Hospital</th>
<th>Location</th>
<th>Beginning Search Date</th>
<th>Ending Search Date</th>
<th>Search Criteria</th>
<th>SVT Patients Enrolled</th>
<th>VT Patients Enrolled</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of New Mexico Hospital</td>
<td>Albuquerque, NM</td>
<td>Jan-91</td>
<td>Feb-95</td>
<td>A</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Veterans Affairs Medical Center</td>
<td>Albuquerque, NM</td>
<td>Jul-93</td>
<td>Nov-95</td>
<td>All ECGs with WCT stored in the hospital ECG database, and patient diagnosed with VT</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>R.E. Thomason Hospital</td>
<td>El Paso, TX</td>
<td>Jul-90</td>
<td>Jan-96</td>
<td>A</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Columbia West Medical Center</td>
<td>El Paso, TX</td>
<td>Jan-93</td>
<td>Mar-96</td>
<td>A</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>New York University Hospital</td>
<td>New York, NY</td>
<td>Dec-97</td>
<td>Jan-03</td>
<td>All ED patients with a pharmacy charge for intravenous adenosine</td>
<td>11</td>
<td>4</td>
</tr>
<tr>
<td>SUNY Downstate Medical Center</td>
<td>New York, NY</td>
<td>Jul-99</td>
<td>Feb-05</td>
<td>A</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Massachusetts General Hospital</td>
<td>Boston, MA</td>
<td>Oct-93</td>
<td>Jan-08</td>
<td>A and B</td>
<td>59</td>
<td>27</td>
</tr>
<tr>
<td>Brigham and Women's Hospital</td>
<td>Boston, MA</td>
<td>Jan-94</td>
<td>Oct-06</td>
<td>A and B</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>Mount Auburn Hospital</td>
<td>Cambridge, MA</td>
<td>Sep-96</td>
<td>Dec-06</td>
<td>Digital text search of all ED physician charts for the consecutive letters “VT,” “vent tach,” “aden,” “amio,” or “procain”</td>
<td>29</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>Total</strong></td>
<td>116</td>
<td>81</td>
</tr>
</tbody>
</table>

SVT, supraventricular tachycardia; VT, ventricular tachycardia; ECGs, electrocardiograms; WCT, wide QRS complex tachycardia; A, primary discharge diagnosis “paroxysmal ventricular tachycardia”; ED, emergency department; ICD-9 427.1; B, all ED patients with a pharmacy charge for intravenous adenosine, amiodarone, or procainamide.

**MATERIALS AND METHODS**

**Study Design**

This report describes a retrospective observational study of consecutive patients with sustained WCT treated with intravenous adenosine (adenocard, Astellas Pharma US, Deerfield, IL).

**Setting and Selection of Participants**

This was a multicenter study performed at nine hospitals in five cities (Table 1). Patients were primarily identified at their emergency department (ED) presentation, but the first administration of adenosine (ranging from treatment by emergency medical services [EMS] to administration in another hospital unit) was analyzed. A variety of search criteria were used to identify cases depending primarily on the medical record documentation, storage, and search capabilities available at each facility (Table 1).

This study was performed concurrently with a retrospective comparison of amiodarone and procainamide for the treatment of VT. It is conceivable that a patient could have been treated with one of these agents in the ED, and could have received adenosine before ED arrival by EMS providers. Thus, these patients were also included in the enrollment criteria. Patients included in New Mexico and Texas were previously described (17). Institutional Review Board approval with waiver of informed consent was obtained from all participating institutions.

All patients >16 yrs old who presented with sustained regular WCT and were treated with intravenous adenosine were eligible for inclusion. A sustained rhythm was defined as continuous for at least 2 mins to minimize the risk of mislabeling spontaneous termination of a transient WCT as a response to adenosine. A regular rhythm was defined as having ≤5% beat-to-beat variability in R-to-R interval for contiguous beats. Wide QRS tachycardia was defined as a QRS width ≥120 msecs and a heart rate (HR) of at least 120 beats/min. All patients who received a 12-mg bolus of adenosine were eligible for inclusion. Patients who exhibited any response to a 6-mg bolus and exhibited no response were included, as it is unknown if they would have responded to a full therapeutic dose of 12 mg. If a patient received multiple boluses with different doses for a single WCT episode, then the response to the highest dose was included. If a patient received multiple boluses of the same dose of adenosine, then the response to the first bolus was included in the primary study analysis. If a patient was treated on multiple occasions, only the first event for which there was complete data was included.

**Data Collection**

Five unblinded investigators collected data, using standardized abstraction forms following a prospectively written protocol. The principal investigator trained the other abstractors and reviewed all of the forms in periodic meetings. For variables noted multiple times on the chart, the first documented value was recorded. Unavailable information was documented as missing and the decreased total number was noted in the appropriate category in the results in Table 2. Study data were collected from each record by a single abstractor, and there was no measure of intrarater reliability.

**Methods of Measurement**

Patients' demographics, including age and gender, the date of the WCT presentation, the hospital, and hospital unit were recorded. History of cardiac disease including dysrhythmias, implantable cardioverter defibrillator (ICD) implantation, congestive heart failure, and measured left ventricular ejection fraction, MI, or coronary artery bypass grafting, was obtained from the medical record. Medications at presentation including antidysrhythmics and medications known to alter adenosine effect, such as aminophylline, theophylline, and dipyridamole, were recorded.

The duration of the WCT before treatment, if known, the initial HR and blood pressure, and any medications administered before adenosine were recorded. Symptoms of chest pain, shortness of breath, or lightheadedness, and physical signs of pulmonary rales or lower extremity edema before treatment were noted.

The HR, QRS duration, frontal QRS axis, and
Table 2. Patient characteristics

<table>
<thead>
<tr>
<th>Rhythm Adenosine Response</th>
<th>SVT</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, years</td>
<td>66.7 (12)</td>
<td>70.5 (16)</td>
</tr>
<tr>
<td>Gender, male</td>
<td>75% (9)</td>
<td>55% (57)</td>
</tr>
<tr>
<td><strong>Past medical history</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of MI</td>
<td>33% (4)</td>
<td>23% (24)</td>
</tr>
<tr>
<td>History of CABG</td>
<td>8% (1)</td>
<td>17% (18)</td>
</tr>
<tr>
<td>EP ≤ 35%</td>
<td>46% (5/11)</td>
<td>24% (22/93)</td>
</tr>
<tr>
<td><strong>Dysrhythmia history</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supraventricular</td>
<td>33% (4)</td>
<td>44% (46)</td>
</tr>
<tr>
<td>Spontaneous sustained VT</td>
<td>8% (1)</td>
<td>1% (1)</td>
</tr>
<tr>
<td>Other ventricular</td>
<td>17% (2)</td>
<td>8% (8)</td>
</tr>
<tr>
<td>Uncertain</td>
<td>0% (0)</td>
<td>6% (6)</td>
</tr>
<tr>
<td>None</td>
<td>58% (7)</td>
<td>44% (46)</td>
</tr>
<tr>
<td>Theophylline or aminophylline therapy</td>
<td>0% (0)</td>
<td>1% (1)</td>
</tr>
<tr>
<td><strong>Physical examination</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic pressure (mm Hg)</td>
<td>144 (23)</td>
<td>151 (21)</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>135 (36) (n = 11)</td>
<td>124 (26) (n = 102)</td>
</tr>
<tr>
<td>Patient weight, kg</td>
<td>81.4 (22)</td>
<td>77.7 (20) (n = 95)</td>
</tr>
<tr>
<td>Shortness of breath</td>
<td>50% (6)</td>
<td>37% (37/99)</td>
</tr>
<tr>
<td><strong>Automated ECG characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>QT interval (msec)</td>
<td>140 (21)</td>
<td>136 (16) (n = 103)</td>
</tr>
<tr>
<td>R/S axis between 180° and 270°</td>
<td>0% (0)</td>
<td>9% (9/100)</td>
</tr>
<tr>
<td>Corrected QT interval (msec)</td>
<td>502 (59) (n = 12)</td>
<td>489 (43) (n = 97)</td>
</tr>
<tr>
<td><strong>Serum electrolytes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potassium, mmol/L</td>
<td>4.3 (.5)</td>
<td>4.2 (.5) (n = 103)</td>
</tr>
<tr>
<td>Magnesium, mEq/L</td>
<td>1.7 (.1) (n = 7)</td>
<td>1.8 (.3) (n = 94)</td>
</tr>
<tr>
<td>Calcium, mg/dL</td>
<td>9.4 (.7) (n = 7)</td>
<td>9.1 (.6) (n = 58)</td>
</tr>
</tbody>
</table>

SVT, supraventricular tachycardia; VT, ventricular tachycardia; MI, myocardial infarction; CABG, coronary artery bypass graft; EF, ejection fraction; ICD, implantable cardioverter defibrillator; ECG, electrocardiogram.

Count data are provided as a percentage along with the absolute count. If the history of present illness, physical examination, ECG, or ECG-QRS axis was not specifically stated as positive or negative, or was unavailable, it was treated as unknown. In this case, the number of patients with the characteristic divided by the total number known is provided. Interval data are provided as an average along with the standard deviation in parentheses. If the data were unavailable for some patients, then the number of patients, n, upon which the data are based is provided.

QT interval corrected for HR were recorded from automated measurements from the WCT ECG, with manual correction for obvious errors. Measured patient weight was noted as well as the serum potassium and magnesium. Serum calcium was added to the data collection form for the Massachusetts hospitals.

The number and dose of adenosine infusions, whether a post bolus flush was specified, and the response to each infusion were recorded. Adenosine response was classified as: none, tachycardia termination with return to native rhythm, transient change in AV nodal conduction, new atrial or ventricular dysrhythmia, or other. Any documented adverse effects of adenosine infusion and the subsequent and ultimate tachydysrhythmia treatments were recorded.

Acute MI was diagnosed in association with the WCT event if the patient demonstrated characteristic elevation of cardiac markers and the diagnosis was confirmed by the cardiologic team caring for the patient. Any patient death and the reason for death during the hospitalization were recorded.

The underlying mechanism of the WCT rhythm was determined by the investigators using the following methods and criteria in descending order, as available: ECG evidence of AV dissociation demonstrating VT, review of the tachydysrhythmia from ICD recordings, reproduction of the dysrhythmia during an electrophysiologic study, diagnostic response to carotid massage or adenosine, such as transient AV block revealing atrial flutter waves, ECG analysis by the treating cardiology team with clear evidence of the diagnosed rhythm confirmed by the investigators, or if necessary, ECG analysis using standard criteria by an electrophysiologist investigator blinded to all clinical data.

**Outcome Measures**

The primary outcome was the response to adenosine as observed by the medical care team based on the following predetermined criteria.
tachycardia; RV, right ventricular; RVOT, right ventricular outflow tract.

### Table 3. Adenosine dose and response

<table>
<thead>
<tr>
<th>Adenosine Dose and Response</th>
<th>SVT</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 mg, no response</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>6 mg, positive response</td>
<td>58</td>
<td>1</td>
</tr>
<tr>
<td>Up to 12 mg, no response</td>
<td>11</td>
<td>73</td>
</tr>
<tr>
<td>Second 12 mg, no response</td>
<td>3</td>
<td>21</td>
</tr>
<tr>
<td>Second 12 mg, positive response</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Up to 12 mg, positive response</td>
<td>46</td>
<td>1</td>
</tr>
<tr>
<td>Up to 18 mg, no response</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Up to 18 mg, positive response</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

### Table 4. How was the diagnosis proven?

<table>
<thead>
<tr>
<th></th>
<th>SVT</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG with AV dissociation</td>
<td>0% (0)</td>
<td>15% (12)</td>
</tr>
<tr>
<td>ECG with VT with variable retrograde VA conduction</td>
<td>0% (0)</td>
<td>4% (3)</td>
</tr>
<tr>
<td>ICD interrogation</td>
<td>1% (1)</td>
<td>5% (4)</td>
</tr>
<tr>
<td>EP study with tachydysrhythmia reproduced</td>
<td>15% (17)</td>
<td>54% (44)</td>
</tr>
<tr>
<td>EP study with tachydysrhythmia reproduced and AV dissociation</td>
<td>0% (0)</td>
<td>6% (5)</td>
</tr>
<tr>
<td>Carotid massage response diagnostic</td>
<td>1% (1)</td>
<td>0% (0)</td>
</tr>
<tr>
<td>Adenosine response diagnostic</td>
<td>48% (56)</td>
<td>0% (0)</td>
</tr>
<tr>
<td>ECG criteria per cardiologist team</td>
<td>20% (23)</td>
<td>0% (0)</td>
</tr>
<tr>
<td>ECG criteria per electrophysiologist</td>
<td>16% (18)</td>
<td>16% (13)</td>
</tr>
</tbody>
</table>

### Table 5. Rhythm and response to adenosine

#### Tachycardia Termination

<table>
<thead>
<tr>
<th>Rhythm</th>
<th>Supraventricular</th>
<th>Atrial flutter</th>
<th>Atrial tachycardia</th>
<th>Atrioventricular tachycardia</th>
<th>AVNRT</th>
<th>Node-fascicular tachycardia</th>
<th>VT, unspecified</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post Adenosine</td>
<td>0</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>17</td>
</tr>
</tbody>
</table>

#### Any Response to Adenosine

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supraventricular</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>54</td>
<td>4</td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Atrioventricular tachycardia</td>
<td>11</td>
<td>0</td>
</tr>
<tr>
<td>AVNRT</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Nodal-fascicular tachycardia</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>VT, unspecified</td>
<td>17</td>
<td>4</td>
</tr>
</tbody>
</table>

### Table 6. Characteristics of Study Subjects

- A total of 197 patients who presented during the 16-yr span from January 1991 to December 2006 were included (Table 1). Twenty-one patients who demonstrated no response to the administration of 6 mg of adenosine and received no further adenosine were excluded (Table 3). There was no evidence of a serious complication from adenosine in any of these excluded patients. The distribution of tachydysrhythmic treatment locations included: 13 (7%) in the field treated by EMS, 155 (79%) in the ED, seven (4%) in clinic, one (1%) in the treadmill laboratory, 16 (8%) on an in-patient unit, and five (3%) in the intensive care unit. Techniques used to determine the tachydysrhythmia mechanisms are listed in Table 4. Patient characteristics are listed in Table 2. Exact duration of the tachycardia and infusion of a flush post adenosine bolus were rarely explicitly documented and thus are not listed.

### Main Results

Response to adenosine infusion by rhythm is listed in Table 5. One patient each with SVT and VT had a history of Wolff Parkinson White Syndrome. The primary endpoint, any response to adenosine administration including termination or transient alteration, is listed along

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with termination alone. When all variants were combined, 104 (90%) of 116 (95% CI = 83%–95%) and two (2%) of 81 (95% CI = 0.3%–9%) SVT and VT patients demonstrated a response to adenosine, respectively. In terms of likelihood ratios, a positive response to adenosine increased the odds of SVT by a factor of 36.3 (95% CI = 9.2–142.9) whereas a negative response to adenosine increased the odds of VT by a factor of 9.4 (95% CI = 5.5–16.1). Response to adenosine infusion as a function of dose is listed in Table 3.

Details of the two patients with VT, who responded to adenosine, are as follows: the first patient was a 39-yr-old man with a history of Wolff Parkinson White Syndrome and an idiopathic dilated cardiomyopathy. His HR decreased from 216 to 90 beats/min after 6 mg of adenosine infusion. Electrophysiologic study revealed inducible sustained VT identical to the patient’s clinical WCT and a posteroseptal accessory pathway with a long effective refractory period that did not participate in the tachydysrhythmia. The second patient was a 48-yr-old man with a history of an inferior MI, who was noted by EMS to have a transient decrease in HR from 160 to 120 beats/min after 12 mg of adenosine in the field. No rhythm strips of the event were available. The patient arrived at the ED with an HR of 167 beats/min and was treated successfully with intravenous amiodarone. His WCT ECG demonstrated AV dissociation. Because there was no definitive documentation by EMS of this reported transient response of VT to adenosine, a sensitivity analysis was performed of the primary outcome after removal of this case. One (1%) of 80 (95% CI = 0.03%–7%) VT patients demonstrated a response to adenosine. A positive response to adenosine increased the odds of SVT by a factor of 71.7 (95% CI = 10.2–503.3) whereas a negative response to adenosine increased the odds of VT by a factor of 9.5 (95% CI = 5.6–16.3).

All documented adverse effects are listed in Table 6. No patient suffered a sustained ventricular tachycardia or bradycardia, or required an emergent intervention, such as cardioversion, defibrillation, or pressor medication infusion in response to adenosine administration. The rate of the primary adverse events for SVT and VT patients was 0 (0%) of 116 (95% CI = 0%–3%) and 0 (0%) of 81 (95% CI = 0%–4%), respectively.

No patients suffered an ST elevation MI in association with their tachycardic event. One patient with SVT and one with VT died in association with their tachycardic event.

DISCUSSION

The optimal treatment and prognosis for patients with SVT and VT differ considerably. Thus, determining the tachydysrhythmia mechanism is useful to optimize emergent care. A number of historical factors may be predictive. Sustained VT occurs most commonly in the setting of a myocardial scar with localized altered conduction properties that develop over time after an MI. In this sample, male gender, history of MI, low ejection fraction, and ICD implantation were more commonly present in patients with VT; however, as demonstrated in Table 2, individually, these factors would have poor test characteristics in distinguishing VT from SVT. Not surprisingly, a history of prior sustained VT, which was present in 26% of patients with VT and 2% with SVT, was most strongly associated with current VT.

Patients with VT tended to have a faster HR and lower blood pressure. Exceptions to this include patients with VT who have an ICD or are taking an anti-dysrhythmic in whom the mean HRs were 147 beats/min and 158 beats/min, respectively. In our facilities, electrophysiologists most commonly program ICDs empirically to pace terminate or cardiovert faster VT only.

This study was not designed to assess the utility of ECG findings to distinguish SVT from VT; however, automated ECG values were recorded. Although VT tended to have a wider QRS interval as expected, an extreme right axis deviation between 180° and 270° was a less specific finding for VT than previously reported (19).

A response to a single bolus of 12 mg of adenosine, including transient AV nodal block or tachycardia termination, was highly associated with SVT as opposed to VT. Of 116 patients, 104 (90%) patients with SVT exhibited a response to adenosine, and eight (89%) of nine patients with VT proven to involve the AV node experienced termination. This is comparable to the rate of adenosine termination of narrow complex VT involving the AV node (8, 20). Similar to prior studies, 98% (79 of 81) of patients with VT demonstrated no observable response to adenosine (7–9).

In contrast to all of the historical factors, physical examination, and ECG findings, nonresponse to adenosine is the only finding that is both highly sensitive and specific for VT. This is because adenosine acts rapidly and briefly to slow conduction in the AV node, and VT generally does not involve or require AV nodal conduction. In contrast, VT most commonly either involves the AV node in a reentrant circuit or requires AV nodal conduction to excite the ventricles.

A notable exception to the lack of response of VT to adenosine is the relatively rare right ventricular outflow tract type (21, 22). Right ventricular outflow tract VT was diagnosed in 4% (three of 81) of patients in this study and was, surprisingly, unresponsive to adenosine in all cases. Adenosine may also cause transient retrograde ventricular-atrial con-

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**Table 6. Adverse effects associated with adenosine administration**

<table>
<thead>
<tr>
<th>Rhythm</th>
<th>VT</th>
<th>SVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertigo and diaphoresis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Nausea and dry mouth</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Flushed sensation</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Flushed sensation with headache and nausea</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Transient increased chest pain</td>
<td>1</td>
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<tr>
<td>Transient increased chest pain with 6 mg adenosine and transient PVCs with 12 mg</td>
<td>1</td>
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<tr>
<td>Asymptomatic PVCs</td>
<td>1</td>
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<tr>
<td>Asymptomatic NSVT</td>
<td>1</td>
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<tr>
<td>Decreased blood pressure and shortness of breath associated with simultaneous nitropaste administration</td>
<td>1</td>
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<tr>
<td>Decreased blood pressure not requiring therapy</td>
<td>1</td>
<td>111</td>
</tr>
<tr>
<td>None</td>
<td>76</td>
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</tbody>
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SVT, supraventricular tachycardia; VT, ventricular tachycardia; PVCs, premature ventricular contractions; NSVT, nonsustained ventricular tachycardia.
duction block in the setting of VT, but this was not observed in our patient sample (8, 9, 13).

Using likelihood ratios, the odds of SVT increase by a factor of 36 (95% CI = 9 – 143) after a positive response to adenosine. In many cases, transient AV block is diagnostic of underlying atrial flutter or tachycardia. The odds of VT increase by a factor of 9 (95% CI = 6 – 16) when there is no response to adenosine infusion. The additional information gained by a nonresponse to adenosine may not be fully appreciated by clinicians. For example, based on the likelihood ratio of 9, if the pretest probability of VT is 50% with 1:1 odds, then the odds of VT would be approximately 9:1 with a 90% probability after a nonresponse to 12 mg of adenosine infusion. This information could be used to guide further therapy primarily aimed at the treatment of VT as opposed to SVT, thus avoiding the poor therapeutic response and adverse effects that may occur due to misguided treatment.

Twenty-seven patients who did not respond to a single 12-mg bolus of adenosine received a second 12-mg bolus (Table 3). Three of six patients with SVT and none of 21 patients with VT responded to the second bolus. In this sample, the sensitivity for SVT would be increased by a second bolus with no cost in specificity. There may be some benefit to this common procedure. There was no apparent benefit from a higher 18-mg adenosine dose.

Cases of destabilization due to hypotension or an adverse change in rhythm have been reported after the administration of essentially every agent used to treat VT (17, 23–25). Remarkably, there is only a single case report of destabilization of VT to ventricular fibrillation after treatment with adenosine (26). The absence of destabilization of VT in our study sample is consistent with the observations in prior series (7–9, 21, 22). The greatest concern in the treatment of patients with regular wide complex SVT with adenosine is accelerated conduction over an accessory pathway with possible deceleration to ventricular fibrillation (11–14). Neither this nor other reported dysrhythmic complications of adenosine, such as torsades de pointes or sustained bradyarrhythmia, were observed in this patient sample.

Dipyridamole competitively inhibits adenosine metabolism, and the risk of adverse adenosine effect may be increased in the presence of dipyridamole (27). Three patients were taking dipyridamole in this study and they received adenosine without event. Other potential risks of adenosine include cardiac ischemia induced by coronary vasodilation and steal syndrome, and dyspnea possibly due to bronchospasm (15, 16, 28). These adverse effects were not observed. Flushing and chest discomfort were occasionally described in this sample. These side effects likely occurred commonly but were not documented because they are well known, expected, and transient without lasting repercussions. Nevertheless, it is likely that clinically important adverse effects would be documented and treated. The low rate and narrow confidence interval of such adverse effects in this sample is reassuring and should encourage adenosine’s appropriate use.

Sustained monomorphic VT is most commonly due to scar from a prior MI, but as observed in this sample, it is rarely associated with an acute STEMI. As has been observed in other series of spontaneous wide complex and ventricular tachycardia, mortality associated with this condition was low, but not zero (17).

Because of the retrospective nature of this study, there were multiple potential limitations, including the risk of bias in enrollment and assessment of adenosine response by the primary physicians and the investigators, incomplete or inaccurate data collection, and nonstandardized treatment regimens. The utility and safety of adenosine administration could differ when applied to a different sample of SVT and VT patients with WCT.

Patients who only received 6 mg adenosine and had no response were excluded. This was decided before initiation of the study because the primary interest was in the response to 12 mg of adenosine, and this outcome would remain unknown in this subgroup. It was felt likely that patients who responded to 6 mg of adenosine would also have responded to 12 mg. Nevertheless, if the patients with no response to 6 mg of adenosine were included, then the sensitivity for VT would have increased from 79 of 81 to 89 of 91; however, the sensitivity for SVT would have decreased from 104 of 116 to 104 of 127.

Normal saline flush post adenosine bolus was rarely explicitly documented in the chart. However, it is the experience of the investigators that nurses and doctors in all of the facilities were well trained regarding the importance of this, and that it was routinely administered.

CONCLUSIONS
The primary evaluation of patients with regular WCT should be based on an educated history, physical examination, and ECG analysis. If, based on this analysis, the mechanism is likely to be VT, then adenosine is unlikely to be helpful. If there is a history of Wolff Parkinson White Syndrome or the WCT is actually irregularly irregular and thus consistent with atrial fibrillation, then adenosine should not be used. If the mechanism is likely SVT or uncertain, then adenosine is safe and may be useful to terminate or to diagnose the rhythm. As for all antidysrhythmics administered to patients with WCT, before adenosine administration, defibrillation pads should be attached to the patient and the defibrillator should be prepared for immediate use in the event of cardiovascular decompensation. Response or nonresponse to 12 mg of adenosine infusion significantly alters the likelihood of SVT vs. VT.

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REFERENCES