When Adenosine Does Not Work
Apparent and Real Adenosine-Resistant Tachycardia

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Abstract: Supraventricular tachycardia (SVT) is the most common arrhythmia in the pediatric population. Adenosine is widely accepted as the first-line pharmacological treatment for hemodynamically stable SVT, constituting a class I recommendation in the 2020 American Heart Association guidelines for pediatric life support (2020 American Heart Association Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care). As most pediatric SVTs are dependent on the atrioventricular node (AVN) for their propagation, and adenosine acts primarily on the AVN, adenosine will frequently terminate the arrhythmia. The term "adenosine failure" is often used to describe when its administration does not result in sustained termination of the tachycardia. Because of its very short half-life, there is confusion between improper delivery, failure to have any effect on the tachycardia, or transient termination. There are some pediatric SVTs, which are not AVN dependent, and which truly are refractory to adenosine. Simultaneous electrocardiogram recording during administration can provide important information to differentiate between adenosine resistance and transient adenosine effect, thus guiding further management.

Key Words: supraventricular tachycardia, adenosine, resistance

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TARGET AUDIENCE

This CME activity is intended for health care clinicians who manage the acute presentation of supraventricular tachycardia in pediatric patients. The target audience includes emergency medicine physicians, pediatricians, pediatric cardiology physicians, and nurse practitioners.

LEARNING OBJECTIVES

After completion of this article, the reader should be better able to:

1. Explain the mechanism of action for adenosine.
2. Describe the indications and contraindications for the use of adenosine in tachyarrhythmias.
3. Distinguish between apparent adenosine failure and adenosine resistant tachycardia.

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Supraventricular tachycardia (SVT) is typically a narrow complex tachycardia, and is the most common chronic arrhythmia seen in the pediatric population.[1,2] Supraventricular tachycardia accounts for 140:100,000 emergency department visits annually.[3] Prevalence is estimated at 1 in 250 children.[4] Supraventricular tachycardia most commonly presents in those with structurally normal hearts. The definition of a narrow QRS complex varies with age because of the progressive prolongation of the QRS duration. In neonates, the upper limit is 70 to 85 ms, increasing to 90 to 110 ms in adolescents.[5] Adult criteria consider a narrow complex tachycardia to be a QRS duration less than 120 ms. When there is a preexisting conduction abnormality, SVT can appear wide, and the morphology should be identical to the sinus QRS. In rate-related aberrant conduction, the QRS will also appear wide. Similarly, in preexcitation syndromes, such as Wolff-Parkinson-White syndrome, antidromic tachycardia or conduction of atrial fibrillation will have a wide QRS, which makes differentiation from ventricular tachycardia (VT) important. Wide complex tachycardia should be presumed to be VT until proven otherwise. Table 1 outlines features, which increase the likelihood of VT versus SVT.

Supraventricular tachycardias require tissue from the His bundle or above.[7] Atrioventricular reciprocating tachycardia (AVRT) and atrioventricular nodal reentrant tachycardia (AVNRT) are the most common forms of SVT in children, with AVNRT presenting more commonly in older children. Maturation of the AV node results in multiple pathways with different conduction properties and refractory periods, allowing reentrant circuits within the AV node. In many cases, P waves may not be visible on the electrocardiogram (ECG) because of their close proximity to the QRS complex, and may appear only as a terminal change in the QRS or an R' pattern.

Atrioventricular reciprocating tachycardia is the most common mechanism of arrhythmia presenting in neonates.[8] In its most common form, orthodromic SVT, the ventricles are activated through the normal His-Purkinje system, resulting in a normal complex tachycardia. The tachycardia circuit depends on activation of the atria via retrograde conduction through the accessory pathway (AP). The presence of an AP may be apparent if the pathway can also conduct antegradely, and there will be ventricular preexcitation noted on the baseline ECG in sinus rhythm.

INITIAL APPROACH TO THE PATIENT IN THE EMERGENCY DEPARTMENT

Presentation of SVT in the pediatric population varies according to age. Younger infants are likely to present with a tachycardia of over 220 bpm and may present after 24 to 48 hours with nonspecific signs, such as poor feeding or lethargy. School age children are likely to verbalize symptoms, such as palpitations, chest pain, or dizziness. Children who are nonverbal due to age or learning disabilities are more likely to have a delayed presentation and present with congestive cardiac failure.[4] In most pediatric patients, SVT is well tolerated, and it is essential to obtain a good quality 12 lead ECG to determine the mechanism of the arrhythmia. Children can present with hemodynamic instability, particularly with a

TABLE 1. ECG Criteria to Guide Differentiation of VT From SVT

AV dissociation	Dissociated P waves Fusion beats Capture beats
QRS duration	QRS duration >160 ms with a LBBB or >140 ms with a RBBB
QRS axis	Northwest axis. RBBB with LAD. LBBB with RAD. RBBB with a normal axis.
QRS precordial concordance	Entirely negative or positive QRS complexes throughout precordial leads, ie, no RS complexes
QRS morphology	Morphology in the precordial leads is not consistent for either RBBB or LBBB in V1-2 and V6.
Brugada Criteria	(a) If there is an RS pattern, an RS interval >100 ms in 1 precordial lead favors VT; (b) meets the morphology criteria for VT in both precordial leads V1, V2, and V6 c) AV dissociation d) Concordance present in precordial leads

Adapted from Vereckei et al.[6]

RBBB indicates right bundle branch block; LBBB, left bundle branch block; RAD, right axis deviation; LAD, left axis deviation.

delayed presentation. Signs of impending collapse are altered mental state and lethargy; blood pressure can be difficult to record at very rapid heart rates and should not be relied on as an indicator of stability. It is very easy to destabilize a compensated patient with medical maneuvers, including attempting intravenous access, administering medication, or giving sedation. All of these can precipitate significant decompensation. Adenosine can also cause sinus arrest and induce ventricular fibrillation so a defibrillator should be accessible when administered. Assessment of the patient in the emergency department must include an approach consistent with Advanced Pediatric Life Support.

In patients who present with hemodynamic instability, electrical cardioversion is the recommended route of therapy. In well-tolerated SVT, vagal maneuvers are the first recommended intervention for acute termination of the tachycardia. Adenosine is the recommended first-line pharmacological therapy for patients when vagal stimulation is unsuccessful or inappropriate[9] and constitutes a class I recommendation in the 2020 American Heart Association guidelines for pediatric life support.[10]

ADENOSINE PHARMACOLOGY AND MECHANISM FOR ACTION

Adenosine is a naturally occurring purine nucleoside, which produces a wide range of physiological actions. In cardiac tissue, adenosine binds to type 1 (A_1) receptors, which are coupled to Gi proteins. This opens potassium channels that hyperpolarize the cell. Activation of the Gi protein decreases cyclic AMP, inhibiting L-type calcium channels, thereby reducing the flow of calcium into the cell. This has a particular effect on cells in the atrioventricular node (AVN) as these L-type calcium channels are responsible for phase 2 of the action potential, and inhibition of these channels decreases conduction velocity.[11] Adenosine also acts to decrease conduction velocity in atrial cardiac myocytes within the sinoatrial node, by reducing intracellular cyclic AMP levels and therefore inhibiting the pacemaker current (I_f), which is responsible for phase 4 of the action potential in pacemaker cells and thereby decreases the rate of spontaneous depolarization.[12]

Adenosine has a very rapid onset of action and a short half-life of less than 10 seconds in human blood where it undergoes rapid intracellular metabolism.[11] This is key when using adenosine; it needs to be given with a rapid flush, preferably using a 3-way stopcock to allow more efficient administration. Adenosine should also preferentially be given in a large-bore cannula in large vein and as proximal to the central circulation as possible, eg, through a cannula in an upper limb.[13] Because of the rapid metabolism in blood, the cannula should be free of red blood cells. Although there are reports of using antiarrhythmic medications through an intraosseous needle, it is not particularly effective for adenosine. The side effects of adenosine are related to its vasodilatory action and include flushing and hypotension. Its short half-life makes it a safe drug to administer in hospital settings,[14,15] although it can cause provoke atrial fibrillation, which may require urgent cardioversion in the setting of preexcitation.

As per American Heart Association guidelines, dosage of adenosine is 100 μg/kg with an increased dose administered when ineffective. However, in our clinical experience, a higher dose is frequently needed, if only to assure the prescribers that insufficient dosing was not the reason for apparent noneffect. In a study by Lewis et al,[2] although the overall conversion to sinus rhythm rate with adenosine was 79%, the response to the first dose in infants younger than 12 months was much lower with only 1 of 17 infants responding to the initial dose. It was, therefore, recommended that an initial dose of 200 μg/kg should be given to infants younger than 12 months. Similar results were reported in a study by Dixon et al[16] who found that the median effective dose was 150 μg/kg. Adenosine is increased in increments of 100 μg/kg until a response of AV block is seen on the electrocardiogram. The maximum dose is 500 μg/kg for a child and 300 μg/kg for a neonate; however, it is important to note that this refers to the maximum singly administered dose and should not be mistaken for a cumulative total administration.[17]

WHEN IS ADENOSINE LIKELY TO BE EFFECTIVE?

The use of adenosine in the acute management of stable narrow complex SVT is well illustrated in pediatric life support guidelines as the first-line pharmacological treatment if vagal maneuvers fail to terminate the tachycardia. Adenosine acts on the AVN and, therefore, is indicated in tachycardias that are AVN-dependent, which include AVRT and AVNRT (Table 2). As these tachycardias are typically quite stable in their rate, a regular narrow complex tachycardia is the most likely to respond to adenosine.

Important information can be gained by how the tachycardia terminates after adenosine. Adenosine blocks antegrade conduction in the antegrade slow pathway in AVNRT and in the AVN in AVRT, therefore, if the tachycardia terminates with a P wave then the mechanism is likely AVRT/AVNRT. Repeated termination with a P wave also rules out atrial tachycardia as this usually terminates with a QRS before AV block due to the antiadrenergic effects of adenosine. Atypical AVNRT can block in the slow pathway retrogradely, terminating with a QRS. Orthodromic AVRT can also terminate with either a P wave (block is in the AVN) or with a QRS (block occurs in the AP)[19] (see Fig. 1).

If the ventricular rate during the arrhythmia is quite irregular, it is likely atrial in origin and less likely to stop with adenosine. Adenosine will, therefore, not usually be effective on arrhythmias such as atrial fibrillation, atrial flutter, focal atrial tachycardia, or, in the case of repaired congenital heart disease, intra-atrial reentrant tachycardia. Some atrial tachycardias are adenosine sensitive because their site of origin may resemble AV nodal tissue electrophysiologically if the site is perinodal or periannular.[19] Although adenosine is also not usually effective for macro reentrant tachycardias, such as atrial flutter, because they are not AVN dependent, the transient AVN blockade can unmask the flutter waves.

TABLE 2. Classification of Regular, Narrow Complex SVT and Their Response to Adenosine

Classification	Pathophysiology	ECG Signs	Effect of Adenosine	Tachycardia Termination With Adenosine
Atrial tachycardias				
Sinus tachycardia	Physiologically driven due to sympathetic tone	Normal P wave axis and a physiological rate for age	Transient AV block with nonconducted P wave seen at a rate of 220 bpm or less or no effect	No
Focal AT	Automatic focus within the atrium	Narrow complex regular tachycardia and may have a variable conduction to the ventricles. P wave morphology will be uniform. P waves may be hidden in the QRS or Twave	Transient AV block revealing P waves at a rapid rate. P waves are separated by an isoelectric line	May terminate if focus is perinodal
Multifocal AT	Automatic focus from multiple atrial foci	Narrow complex irregular tachycardia. P wave morphology is variable with a variable P-P intervals	Transient AV block revealing P waves of multiple morphologies separated by an isoelectric line	No
Atrial flutter	Macro reentry circuit within the right atrium	Narrow complex regular tachycardia. The atrial rate is usually over 250 bpm with variable block to the ventricles, ie, 1:2 or 1:3. There is no isoelectric line	Transient AV block revealing flutter waves with no isoelectric line	No
Atrial fibrillation	High frequency excitation of the left atrium	Narrow complex irregularly irregular ventricular rhythm with no distinguishable P waves. Fibrillary waves may be seen	Transient AV block revealing fibrillation waves with no isoelectric line	No
AV nodal tachycardia				
AVNRT	Reentry circuit within the AVN due to 2 pathways with different conduction speeds and refractory periods	Narrow complex regular tachycardia with abrupt onset and offset. No obvious P waves seen	Termination of tachycardia with a retrogradely conducted P wave followed by a short period of AV block after sinus beat	Yes
JET	Automatic focus from within the AVN	Narrow complex regular tachycardia. P waves may be conducted retrogradely after the QRS or there may be ventriculoatrial dissociation	There is temporary block of the retrograde conduction to the atria but no slowing of the ventricular rate	No
Atrioventricular reentrant tachycardia				
Orthodromic	Reentry mechanism with impulses conducted antegradely down the AVN and retrogradely back up the AP	Narrow complex regular tachycardia. Retrogradely conducted P waves may be seen in 1:1 relationship with the QRS complexes or may be buried.	Termination of tachycardia with a short period of AV block followed by a sinus beat or an escape beat	Yes
Antidromic	Reentry mechanism with impulses conducted antegradely down the AP and retrogradely up the AVN	Regular broad complex QRS (>120 ms) tachycardia due to delta waves. May be difficult to ascertain from VT. Preexcitation will be seen on sinus ECG	May terminate if tachycardia is using the AVN as retrograde limb	Yes

Adapted from Brugada et al.[18]

AT indicates atrial tachycardia; AF, atrial flutter; AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reentrant tachycardia; JET, junctional ectopic tachycardia.

WHEN SHOULD ADENOSINE NOT BE USED?

Adenosine should not be administered to patients who are hemodynamically unstable during the arrhythmia; this means a reduced level of consciousness. In pediatric patients presenting with an irregular broad complex tachycardia, this is usually preexcited atrial fibrillation (see Fig. 3B). This should be treated with IV procainamide. It is reasonable to administer adenosine to hemodynamically stable pediatric patients presenting with a regular wide complex tachycardia. This is because the majority of such cases in pediatrics will be due to SVT with aberrancy[20] (Fig. 2B). If the patient is stable and they are known to have antidromic AVRT,

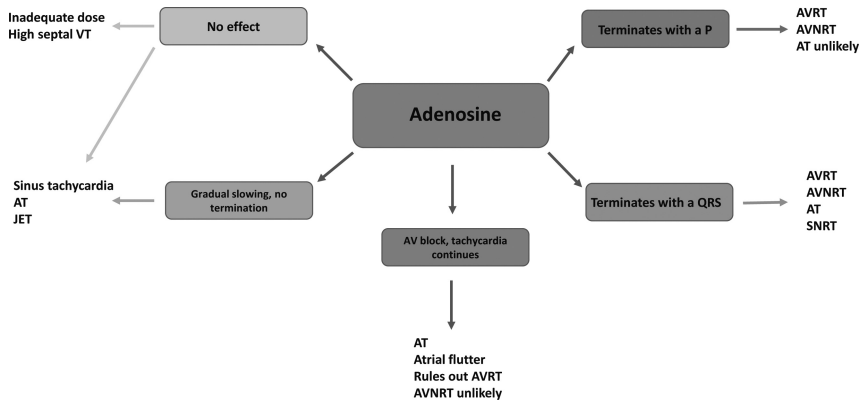

FIGURE 1. Differential diagnosis of narrow QRS tachycardia based on the response to adenosine. Modified from Brugada et al.[18] AT indicates atrial tachycardia; JET, junctional ectopic tachycardia; SNRT, sinus node reentrant tachycardia.

then it is reasonable to try adenosine, with the ability to cardiovert, if the tachycardia does not respond or accelerates. An alternative approach would be to use drugs acting mainly on the AP, such as procainamide or flecainide, as first-line pharmacological therapy.[7] The contraindications to adenosine are adult focused and include second or third-degree AV block without the presence of a pacemaker, sick sinus disease or symptomatic bradycardia, severe hypotension, known hypersensitivity to adenosine previously. A relative contraindication is a history of bronchoconstriction, such as asthma[21]; large multicenter reviews of pediatric emergency departments have reported no episodes of bronchospasm with adenosine.[22]

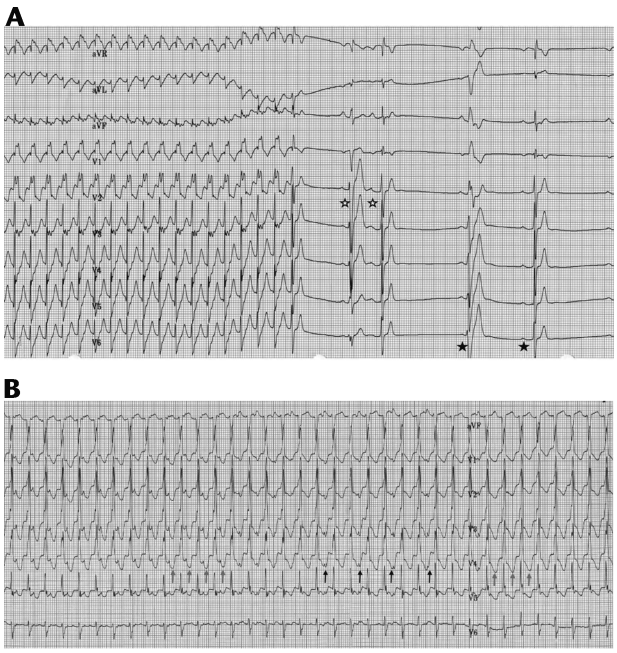

FIGURE 2. A, Adenosine effect in regular narrow complex tachycardia demonstrating abrupt termination. Initial sinus beats conduct with progressive PR prolongation (white stars) indicating AVN block. Black stars, sinus P waves. B, Adenosine effect in a wide complex regular tachycardia with 1:1 VA association. The administration of adenosine results in 2:1 VA block with persistent tachycardia, indicating the atrium does not need to participate in the tachycardia. In this case, this is VT. Note that the QRS in an infant can look relatively narrow. Light grey arrows, P waves with 1:1 VA association. Black arrows, P waves with 2:1 VA block.

COMMON REASONS FOR APPARENT ADENOSINE FAILURE/RESISTANCE

Adenosine failure is often labeled as occurring when its administration does not result in definitive or sustained termination of the tachycardia. There are three scenarios that cause this:

1. Failure of administration

In this scenario, adenosine is given, but there is no evidence of effect. There is no change in VA relationship, no transient slowing of the atrial rate, no PR interval change, and no increase in heart rate after the medication. There are no patient reported symptoms, such as headache, nausea, flushing, or "feeling of impending doom." This means that there is no clear evidence of any Adenosine action. This can be due to either

a. insufficient dose of adenosine;
b. incorrect administration, eg, IV push is too slow or the presence of red blood cells in the line;
c. access is too distant from central circulation.

2. Failure of the arrhythmia to terminate: adenosine resistance tachycardia

In this case, the arrhythmia does not respond to adenosine. This occurs in automatic tachycardias, such as sinus, atrial, or junctional, as they are not dependent on reentry using the AVN. There may be a transient response, such as temporary suppression of AVN conduction, with continuation of the atrial or ventricular arrhythmia.

The difference between an insufficient dose versus a refractory arrhythmia can be difficult to determine, and recording a rhythm strip while administering adenosine is essential. In the case of an atrial tachycardia, AVN block will lead to observation of P waves continuing on at the tachycardia rate before the conduction through the AVN resumes (Fig. 3A). The exception to this are some focal reentrant perinodal atrial tachycardias, these usually terminate before the effect on the AV node; therefore, the tachycardia terminates after a QRS complex. A transient slowing of the ventricular rate sometimes leads to the erroneous conclusion that the tachycardia has stopped. In junctional tachycardia and VT, there may be a change of the VA relationship, including VA block. This can be quite subtle. Most VTs fall into this category.

3. The duration of adenosine effect is insufficient

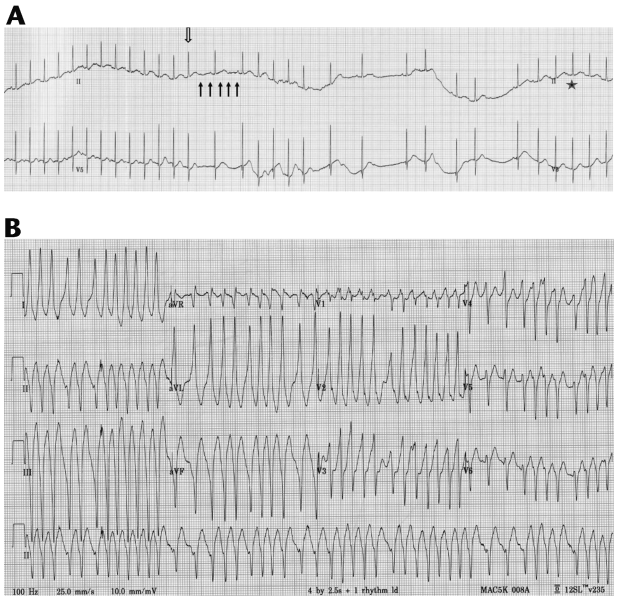

FIGURE 3. Adenosine resistant tachycardias. (A) 12-lead electrocardiogram demonstrating the effect of adenosine on atrial tachycardia. White arrow, adenosine effect on the AV node seen with slowing of AVN conduction. Black arrows, nonconducted P waves unmasked by adenosine. Black star, tachycardia resumes. (B) Preexcited atrial fibrillation. This presents as an irregular wide QRS tachycardia. Adenosine is contraindicated.

In some patients with AVRT and AVNRT, the tachycardia may stop with adenosine, but then resume. This is common in patients who have been in the arrhythmia for a while, in whom endogenous catecholamines may promote continuation of the tachycardia. Similarly, long RP tachycardias can be incessant. Because of its short half-life, adenosine may result in a short period of AV block before the tachycardia quickly resumes. This reinitiation of tachycardia can be seen in up to 28% of pediatric patients after effective adenosine administration.[5] In such cases, a higher dose of adenosine may be effective; however, it is more likely that a drug with a longer acting effect is needed. It is often possible to use an oral medication, such as flecainide or propafenone, in a patient tolerating the arrhythmia well. Ascertaining whether administration of adenosine failed or whether the tachycardia was resistant is a key distinction, so it is crucial that a 12-lead electrocardiogram is taken before and during the administration of adenosine. If there is no evidence of AVN effect from adenosine then the next management steps should be focused toward optimizing delivery. If there is AV block but only short-lived conversion to sinus rhythm (refractory SVT) despite optimization of the delivery method and the maximum dose of adenosine, then a cardiology opinion should be sought to discuss other pharmacological treatment.[17]

In some tachycardias, there will truly be no observable response, even with proper administration; sinus tachycardia is such a rhythm.

SUMMARY

Adenosine produces a wide range of electrophysiological effects in the myocardium and has been used for decades by cardiologists across the globe to treat and diagnose narrow complex tachycardias in the acute setting and also as part of electrophysiology studies. The term adenosine failure is often used to describe when adenosine does not terminate a tachyarrhythmia, which infers that this is because of the arrhythmia mechanism being focal or triggered. However, we recommend that this term should only be used when there is no effect on the AV node seen on the ECG, such as AV conduction prolongation or block. This is due to inadequate administration of adenosine through an intraosseous line or a small peripherally placed IV line, the adenosine bolus being given too slowly or the presence of red blood cells in the line causing rapid metabolism before the drug reaches the central circulation.

When adenosine is administered effectively, there are several potential outcomes. If there is successful adenosine therapy then AV block is seen preceding SVT termination. This indicates that the SVT is due to an AV node-dependent reentry tachycardia, such as AVRT, AVNRT, or a perinodal atrial tachycardia.

The other 2 outcomes are either (1) gradual slowing but no termination of the tachycardia, which can be seen in sinus tachycardia and automatic atrial or junctional tachycardias or (2) AV block will be seen with continuation of the tachycardia but a slower ventricular rate, which is seen in atrial flutter and atrial tachycardia.[19]

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