Supraventricular Tachycardia in Infancy and Childhood

Andrew D. Spearman, MD; and Paula Williams, MS, MD

Abstract

Supraventricular tachycardia (SVT) is the most common arrhythmia in the pediatric population. Despite its commonality, presentation of SVT can be nonspecific and varies based upon age with infants demonstrating fussiness or irritability and older children reporting vague perceptions of tachycardia or palpitations. Furthermore, SVT may manifest as self-limited paroxysms or with prolonged runs of SVT with subsequent development of cardiac dysfunction, heart failure, and multiorgan shock. Clinicians must maintain high levels of suspicion for SVT given the potentially dire consequences of untreated SVT. When diagnosed, there are effective acute and chronic treatments for SVT, with potential for spontaneous resolution in many infants. [Pediatr Ann. 2014;43(11):456-460.]

You are admitting a patient to the pediatric intensive care unit (PICU) from the emergency room with tachycardia and possible sepsis. He is a 28-day-old, full-term infant with no previous medical problems. His mother reports that he has had increasingly poor feeding and fussiness over the past 2 days, as well as decreased activity and alertness, decreased wet diapers, and increased difficulty breathing. His mother reports that he has had increasingly poor feeding and fussiness over the past 2 days, as well as decreased activity and alertness, decreased wet diapers, and increased difficulty breathing. His mother also reports visiting another hospital emergency department 2 days ago at the onset of symptoms with a reported normal examination and work-up; the child was discharged home with supportive care. She denies any reports of elevated heart rate during that evaluation.

INITIAL HOSPITAL COURSE

Initial point-of-care venous blood gas on 100% oxygen is significant for pH of 6.78, pCO₂ of 38.6 mm Hg, pO₂ of 20 mm Hg, bicarbonate of 6.4 mmol/L, and lactate of 16.32 mmol/L. He is emergently given two 20 mL/kg of intravenous (IV) normal saline boluses for hypotension, as well as 2 mL/kg of 10% IV dextrose bolus for a bedside glucose of 51 mg/dL with no significant change in heart rate or blood pressure. Telemetry demonstrates narrow complex tachycardia of approximately 52/35 mm Hg. Cardiac monitoring demonstrates narrow complex tachycardia persistently ranging from 227 to 233 bpm. His examination is notable for a listless infant with a weak cry, nasal cannula in place with blood coming from his mouth, regular tachycardia, no murmurs, tachypnea with severe suprasternal and subcostal retractions, abdominal distention with coffee-ground nasogastric output, hepatomegaly approximately 4 cm below the right costal margin, and palpable femoral and brachial pulses with capillary refill of 3 to 5 seconds.

PHYSICAL EXAMINATION

Upon arrival to the PICU, the child is listless and minimally responsive to stimulation. His vital signs are significant for rectal temperature of 33.5°C, heart rate of 229 beats per minute (bpm), respiratory rate of 37 breaths per minute, saturation of 100% on 100% oxygen via high-flow nasal cannula, and manual cuff blood pressure of 52/35 mm Hg. Cardiac monitoring demonstrates narrow complex tachycardia persistently ranging from 227 to 233 bpm. His examination is notable for a listless infant with a weak cry, nasal cannula in place with blood coming from his mouth, regular tachycardia, no murmurs, tachypnea with severe suprasternal and subcostal retractions, abdominal distention with coffee-ground nasogastric output, hepatomegaly approximately 4 cm below the right costal margin, and palpable femoral and brachial pulses with capillary refill of 3 to 5 seconds.

Figure 1. Electrocardiogram demonstrating narrow complex tachycardia in precordial lead V1, with a rate of 231 beats per minute and QRS interval of 68 milliseconds.
220 bpm (Figure 1), consistent with sinus tachycardia versus supraventricular tachycardia (SVT). While preparing for central line placement, the patient is given a diagnostic dose of adenosine (0.2 mg/kg) via rapid push through peripheral IV, followed by a flush of normal saline without heart rate response. After intubation, a femoral line is placed and used to give a rapid IV push of adenosine (0.3 mg/kg) followed by normal saline flush. Following the rapid, central administration of adenosine, the patient demonstrates a 2- to 3-second atrioventricular (AV) node block then resumes normal sinus rhythm of approximately 120 bpm. He is then started on dopamine and amiodarone drips for hypotension and sustained supraventricular tachycardia, respectively.

At this point, blood work results are available. They demonstrate evidence of multi-organ failure with acute kidney injury (AKI; blood urea nitrogen of 29 mmol/L and creatinine of 0.9 mg/dL), coagulopathy (International Normalized Ratio of 4.7), heart failure (pro-B-type natriuretic peptide of 73,029 pg/mL), and uncompensated metabolic acidosis (serum lactate of 18.4 mEq/L, bicarbonate of 8 mEq/L), as well as a normal white blood cell count (12.3 K/mcL). Chest radiograph is notable for mild cardiomegaly without focal opacity, and bedside transthoracic echocardiography demonstrates severely depressed biventricular wall motion of a structurally normal heart.

**DIAGNOSIS**

Our patient’s history was notable for many nonspecific findings, including increased fussiness, decreased alertness and activity, decreased intake by mouth, and decreased urine output. Notably, 2 days prior, he presented for evaluation due to the same parental concern, with a thorough and negative work-up, including complete blood count, basic metabolic profile, urinalysis, and chest roentgenogram. His symptoms may have been caused by intermittent tachycardia that was not captured during his period of observation.

On presentation to our hospital, he was clearly in shock with altered mental status, respiratory distress, marked hypothermia, and refractory hypotension. In neonates, septic shock must always be considered, but cardiogenic shock was suggested by narrow complex tachycardia without variation in rate, and without leukocytosis or leukopenia, and no obvious site of infection. SVT was confirmed by a 12-lead electrocardiogram (ECG), as well as the diagnostic and therapeutic dose of adenosine.

He was weaned off pressor support and transitioned to milrinone for depressed cardiac function. He was transitioned from amiodarone continuous infusion to oral propranolol for maintenance suppression of SVT. He had several episodes of breakthrough SVT that resolved with IV adenosine and were treated with upward titrations of propranolol. His lactate acidosis, AKI, coagulopathy, and heart failure normalized with return of normal sinus rhythm and the above supportive measures. He was discharged home on hospital day 7.

In summary, the patient is a 1-month-old male who presented in cardiogenic shock with multorgan failure due to supraventricular tachycardia that resolved after restoration of sinus rhythm with intravenous adenosine and supportive care.

**SUPRAVENTRICULAR TACHYCARDIA**

SVT is the most frequent dysrhythmia in the pediatric population, with an estimated prevalence of 1 in every 250 to 1,000 pediatric patients. SVT is a heterogeneous collection of dysrhythmias and is traditionally characterized as a narrow-complex tachycardia. As described by O’Connor and Dick, SVT is a generic term for an accelerated, non-sinus rhythm originating above the level of the AV junction with a rate of 200 to 300 bpm. This heterogeneous group all categorized as SVT can be artificially organized by electrophysiology or anatomic origin (Table 1). Classification tends to be based on electrophysiology, rather than structure, because mechanism directs medical therapy.

Under normal physiologic conditions, the ventricles are protected from a rapid atrial rate because of the innate delay in the AV node. This delay is bypassed by abnormal reentrant circuits in the myocardium, resulting in SVT. Reentrant rhythms create a rapid ventricular rate by either allowing a reentrant circuit to directly connect the atria and ventricles (ie, atrioventricular reentrant tachycardia [AVRT]) or activating the ventricles via the normal His-Purkinje system but rapidly with a reentrant circuit within the AV node itself (ie, atrioventricular node reentrant tachycardia [AVNRT]). Of note, a small subset of SVT is caused by an abnormal focus of atrial automaticity, which will not be discussed here. Traditionally, AVRT tends to predominate in infants, with AVNRT more common in older children and adolescents. Specifically, Ko et al. found SVT due to AVNRT in approximately 5% of infants, compared with nearly 80% due to AVRT. One recent study, however, found that 30% of 2-year-old children with SVT had AVNRT.

Wolfe-Parkinson White (WPW) syndrome is a type of AVRT. WPW is diagnosed in patients with a history of SVT and a baseline ECG that demonstrates sinus rhythm, shortened PR interval, and widened QRS complex with a characteristic delta wave. These ECG findings in WPW are due to fusion of the QRS of normal sinus conduction as well as slight antegrade conduction via the accessory pathway (AP). SVT is initiated if a sinus impulse conducts normally down the AV node and retrograde through the AP at just the right interval, termed orthodromic reciprocating tachycardia (ORT). ORT
is by far the most common manifestation of AVRT (95%). A less common cause of SVT is when the sinus impulse is conducted antegrade down the AP and then backward through the His-Purkinje system, which is termed antidromic reciprocating tachycardia (ART). ART is worth understanding, because it can cause wide complex tachycardia. It is important to note that ART is exceedingly rare, and wide complex tachycardia should be treated as ventricular tachycardia (VT) until proven otherwise.

**PRESENTATION AND DIAGNOSIS**

Signs and symptoms of SVT presentation tend to vary greatly with age and ability to perceive and communicate symptoms. Infants, for example, tend to present with sustained runs of SVT, and they demonstrate more nonspecific signs, as did our patient in this case. SVT sustained over hours to days causes symptoms of congestive heart failure (CHF); thus, infants present with fussiness, irritability, poor feeding, tachypnea, diaphoresis with feeds, and hepatic congestion. Older children and adolescents, on the other hand, may present with vague descriptions of their perceptions of palpitations, including chest pain, racing heart, “buzzing,” nausea, dizziness, and light-headedness. Additionally, they may have complaints of decreased exercise tolerance, fatigue, and lack of energy.

The timing of presentation is likewise affected by the rate of SVT and perceptions of symptoms. Infants are unable to communicate their symptoms, which are often mistaken for colic or other illness. Additionally, the rate of SVT in neonates tends to range from 220-300 bpm. As a result, infants with SVT tend to go undiagnosed and may have sustained tachycardia at high rates, which leads to CHF. Conversely, older children and adolescents have heart rates of 180-220 bpm during episodes of SVT. The lower heart rate and further developed myocardial muscle helps delay onset of tachycardia-induced cardiomyopathy. Older children often report their symptoms and present for evaluation.

Diagnosis can be based on symptoms alone, but most cardiologists require documented SVT prior to initiating treatment. Intermittent tachycardia can be captured with 30-day event monitoring. If the tachycardia is sustained, patients are encouraged to present for evaluation and documentation of SVT with 12-lead ECG. The only acceptable scenario to defer ECG documentation is when you must perform synchronized cardioversion of an unstable patient with SVT.

**TREATMENT**

When considering treatment for acute SVT, the most important diagnostic decision is hemodynamic stability. Stable SVT has many noninvasive treatment modalities that should be employed first, whereas unstable SVT, by definition, is a medical emergency warranting immediate action due to hemodynamic compromise.

Stable SVT is characterized by SVT with preserved hemodynamics (i.e., normal end-organ perfusion with normal blood pressure, mental status, and brisk capillary refill). The hallmark of noninvasive treatment for stable SVT is increasing vagal tone, which can be done with a number of vagal maneuvers.

The Valsalva maneuver, named after physician Anton Maria Valsalva (who described it at the turn of the 18th century), is achieved by forced expiration against a closed glottis. Specifically, it has four phases that help explain how it works. First, there is the onset of strain, followed by maintenance of strain, then release of strain, and finally relaxation. During relaxation, arterial pressure increases and overcompenses (i.e., the vasovagal response causing vasovagal syncope), resulting in vagal stimulation and decremented conduction through the AV node. A second vagal maneuver, and one of the most common vagal maneuvers used in young children with SVT, utilizes the diving reflex. The diving reflex is an aquatic mammalian and bird response to cold or cold water in order to decrease oxygen consumption. This trait is also preserved in many nonaquatic mammals. We can achieve this response by placing a slurry of bagged ice over the nasal bridge and forehead for up to 30 seconds at a time. The cold-water stimulation of afferent branches of the trigeminal nerve causes efferent stimulation of the vagal nerve, again slowing conduction through the AV node. Additional vagal maneuvers include knee-to-chest positioning and assistance in a headstand. Other maneuvers that are no longer recommended include initiating a gag reflex (via nasogastric or orogastric tube insertion), applying ocular pressure (which carries risk of retinal detachment), and carotid massage (which has been shown to have limited effectiveness in children). Vagal maneuvers are effective early but not when initiated late. Prolonged SVT results in ever-increas-

**TABLE 1.**

**Supraventricular Tachycardia Organized by Anatomic Origin**

<table>
<thead>
<tr>
<th>Sinoatrial nodal origin</th>
<th>Atrial origin</th>
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<tbody>
<tr>
<td>Sinoatrial nodal reentry tachycardia</td>
<td>Automatic atrial tachycardia</td>
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<tr>
<td>Atrioventricular nodal origin (junctional tachycardia)</td>
<td>Ectopic (unifocal) atrial tachycardia</td>
</tr>
<tr>
<td>Atrioventricular nodal reentry tachycardia</td>
<td>Multifocal atrial tachycardia</td>
</tr>
<tr>
<td>Junctional ectopic tachycardia</td>
<td>Atrial flutter with rapid ventricular response</td>
</tr>
<tr>
<td>Persistent junctional reciprocating tachycardia</td>
<td>Atrial fibrillation with rapid ventricular response</td>
</tr>
</tbody>
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**FEATURE**
ing sympathetic tone, thus rendering increased vagal tone null.

If vagal maneuvers are unsuccessful in effectively terminating SVT, medical therapy is recommended. There are several pharmacologic antiarrhythmic options, but the mainstay of medical therapy of acute SVT is IV adenosine. Adenosine, when administered in the correct dose with the correct means, is very effective, with success rates of 85% to 100%. There are several common pitfalls when administering adenosine, as seen in the initial treatment efforts in our patient. American Heart Association and Pediatric Advance Life Support guidelines recommend 0.1 mg/kg, followed by 0.2 mg/kg (to a maximum of 12 mg). New evidence suggests that 0.2 mg/kg might be the optimal starting dose, with 0.3 mg/kg as an appropriate second dose. The short half-life of adenosine (<2 seconds) makes the means of administration critical. Adenosine must be given in the most central location possible via a rapid IV push with immediate saline flush using a stopcock. For example, this patient received three appropriately dosed rounds of adenosine without evidence of AV node block because they were given via peripheral IV. Once given through a centrally placed femoral line with appropriate flush, his SVT converted to normal sinus rhythm.

In addition to IV adenosine, other medical options include amiodarone, verapamil, digoxin, propranolol, procainamide, and flecainide. These other medications, however, carry significantly increased risks of side effects and should be undertaken only after consultation with a pediatric cardiologist and/or intensivist. Notably, verapamil should not be used in infants, as it has been associated with irreversible electromechanical dissociation.

If medical therapy with adenosine is unsuccessful, or stable SVT deteriorates to unstable SVT, synchronized cardioversion is warranted. Unstable SVT is defined by acutely impaired hemodynamics, with signs including hypotension, impaired mental status, and delayed capillary refill. Synchronized cardioversion applies a direct current of electricity to the heart, precisely timed to the R wave on ECG, which reduces the risk of inducing ventricular fibrillation. Cardioversion should be applied at 0.5 to 1 J/kg on the first attempt, and doubled until effective cardioversion (up to 2 J/kg).

Diagnosis and termination can also be achieved with transesophageal electrophysiology (TEP) testing. TEP involves transnasal placement of a small pacing catheter in the esophagus posterior to the heart. Basic pacing maneuvers can be performed allowing evaluation of SVT, overdrive pacing to terminate SVT, and evaluation of the need for medication or effectiveness of suppressive medical therapy.

**PROGNOSIS**

Despite the myriad of presentations, prognosis for SVT in the pediatric population is excellent for younger and older patients alike, although for different reasons. Infants with SVT, like our patient, can sometimes present in cardiogenic shock. Once the initial presentation is stabilized and resolved, most infants are controlled with oral medications and many actually have spontaneous resolution. Up to two-thirds of patients with AVRT SVT presenting in infancy may fully resolve by age 1 year. Most infants gradually outgrow their oral medical therapy around age 1 year and can be monitored via cardiac event monitors for further arrhythmias.

Newer data indicate that duration of therapy to age 1 year may not be necessary. A recent randomized, blinded clinical trial comparing digoxin versus propranolol demonstrated 6-month recurrence-free status for 79% of patients on digoxin and 67% of patients on propranolol (P = .34). More impressively, no first recurrences of SVT occurred after 110 days of treatment.

Although there are not consistent patterns *a priori* to suggest which patients will have resolution of SVT and which will not, TEP can be a helpful prognostication tool. Rhodes et al. previously demonstrated a 96% negative-predictive value, with a lack of inducibility during TEP as a marker for patients unlikely to require medical therapy after a patient’s first birthday. Furthermore, a recent study confirmed these findings when 15 of 42 patients who were not inducible remained free of SVT recurrence for an average of 2 years without further medical treatment after negative TEP (median follow-up of 25 months, range of 3 to 97 months). Unfortunately, the positive-predictive value of TEP in multiple studies has not been determined because patients with inducibility are continued on medical treatment. Children or adolescents who are inducible may undergo electrophysiological study to identify and map the errant pathway causing the arrhythmia. Immediately after mapping, the accessory pathway is eradicated with radiofrequency ablation. Pharmacologic therapy is rarely required after ablation.

**CONCLUSION**

The patient has been followed as an outpatient with continued propranolol therapy, with dose titrations for somatic growth and no evidence of breakthrough tachycardia. We plan to wean his medication at age 12 months.

In conclusion, supraventricular tachycardia is relatively common and presents with occasional palpitations or in fulminant cardiogenic shock. Diagnosis, acute treatment, supportive care, and maintenance of sinus rhythm are the mainstays of treatment for SVT cases.

**REFERENCES**


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