ISSN 0975-6299 Vol 3/Issue 2/April – June 2012

International Journal of Pharma and Bio Sciences

RESEARCH ARTICLE PHARMACOLOGY

# PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA IN NEONATES

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## ABSTRACT

Supraventricular tachycardia (SVT) is an uncommon event in the NICU. It may result from underlying cardiac malformation, Wolf-Parkinson-White syndrome, arrhythmogenic drugs or idiopathic in nature. They may or may not result in hemodynamic compromise, nevertheless, need to be treated appropriately. Recurrent neonatal SVT needs prophylactic therapy for about 9 – 12 months, as they tend to resolve spontaneously beyond 1 year.

## KEY WORDS

Supraventricular tachycardia, WPW syndrome, adenosine, propranolol.

## INTRODUCTION

Paroxysmal supraventricular tachycardia (PSVT) is one of the commonest arrhythmias in the neonatal age group. The clinical manifestations in this age group can be easily confused with sepsis, or respiratory distress resulting from a pulmonary lesion. A timely electrocardiograph (ECG) helps one to clinch the diagnosis and provide effective treatment. PSVT or more commonly simply referred to as supraventricular tachycardia (SVT) is the most common symptomatic arrhythmia in childhood that can be a recurrent and persistent condition. The quoted incidence of SVT in children of 1 in 25,000 is based on an estimate made in 1967,[1] but with a higher index of suspicion and better methods of detection, it now is estimated to be 1 in 100 for children of all ages and 1 in 250 for neonates.[2] SVT is a tachyarrhythmia originating proximal to the bundle of His. The typical infant who has SVT has a regular R-R interval, with rates often greater than 230 beats/ min and commonly 260 to 300 beats/ min.

### *Types of SVT*

The most common type of SVT in infancy and childhood, representing approximately 70% of SVT, results from a re-entry circuit between the atria and the ventricles and is called atrioventricular re-entry tachycardia (AVRT). In this circuit, the AV node generally forms the antegrade pathway, and an accessory connection between the ventricle and the atria serves as the retrograde pathway for conduction of impulses. The Wolff-Parkinson White (WPW) syndrome is SVT with evidence of pre-excitation. It is identified from a surface electrocardiogram (ECG) in sinus rhythm by a short P-R interval and a "delta wave." The delta wave is caused by fusion of ventricular complexes resulting from a portion of ventricular myocardium initially depolarized by antegrade conduction through the accessory pathway along with depolarization of the rest of the myocardium through the normal pathway. WPW syndrome comprises 12% to 56% of AVRTs.[2,3,4] WPW syndrome may be associated with Ebstein's anomaly or transposition of great vessels. The second type of SVT is AV nodal re-entry tachycardia (AVNRT) which represents about 13% of SVTs. The tachycardia circuit also involves dual pathways, but both are situated within or near the AV node. Typical AVNRT conducts in an antegrade direction slowly down to the ventricles through one pathway and has a rapid retrograde conduction via the other pathway. It is, therefore, called "slow-fast" AVNRT. On the ECG, this manifests as a QRS complex followed by a T wave, with the P wave often not visible (concealed in the QRS complex). Atypical AVNRT has a comparatively slower retrograde conduction, so the P wave is visible as an inverted complex after the QRS. The third most common type of SVT is atrial tachycardia (AT), comprising approximately 14% of SVTs which includes atrial flutter and atrial ectopic tachycardia (AET).

### *Clinical features of SVT in infants*

Presenting features of SVT in neonates can include fetal SVT, unexplained nonimmune hydrops in the newborn, sudden cardiovascular collapse, symptomatic heart failure developing over a few hours to days or incidental detection during cardio respiratory monitoring. A cardiac cause is found in about 9% of nonimmune hydrops, 60% of which is due to fetal SVT.[5] Congenital heart disease is reported in about 6.5% to 37% of neonates who have SVT.[2,3,6] SVT has been reported as the initial presenting feature of cardiac involvement in patients who

have tuberous sclerosis with cardiac rhabdomyomas.[7,8] Natural history studies in patients who have SVT have demonstrated that approximately 70% of infants lose SVT inducibility by 1 year of age, and clinical recurrences are uncommon.[9]

### *Treatment options for SVT in infants*

Acute termination of SVT is critical in patients who develop signs and symptoms of hemodynamic instability, including lethargy, pallor, poor perfusion, hypotension, acidosis, and signs of cardiac failure. Immediate restoration of sinus rhythm is achieved best by cardioversion. An initial synchronized shock of 0.5 J/kg is recommended. In subsequent attempts, the energy is doubled. If conversion to sinus rhythm is not achieved after two attempts, drug therapy should be initiated before attempting cardioversion again. Although cardioversion can restore sinus rhythm, it cannot maintain sinus rhythm. Therefore, it is always prudent to have a pharmacologic agent available to maintain sinus rhythm after conversion. Adenosine can be administered if a neonate is hemodynamically stable and can be provided to the unstable neonate in whom intravenous (IV) access has been established while preparing for cardioversion. Adenosine is administered as a rapid IV or even intraosseous (I/O) bolus followed by a saline flush with continuous ECG monitoring and availability of cardiopulmonary resuscitation equipment. Adenosine is successful in terminating SVT in about 42% to 86% of cases.[4, 10] The advantages of adenosine are its very short half-life (10 to 15 sec) and the rapid onset of action (within 10 to 20 sec). Administration can be repeated immediately if required, and the drug has no prolonged negative inotropic effect. The disadvantage is immediate recurrence of tachycardia in approximately 30% of cases.

For patients who are hemodynamically stable, a more graded approach is appropriate. Initially, vagal maneuvers, which are simple, quick, and safe to perform, can be attempted. Placement of an ice pack on the face and nose for 5 to 10 seconds at a time is used commonly. Success rates vary from 63% to 96%.[2, 11] Esmolol, a short acting beta blocker (T ½ = 3 minutes) is a very useful pharmacological agent in patients who experience recurrent or sustained SVT. (Table 1) Digoxin has been the drug of choice for neonatal SVT in the past. This agent is avoided in patients who have WPW syndrome because of their predisposition to develop atrial fibrillation, which may lead to ventricular arrhythmias due to enhanced conduction through the accessory pathway. If beta blockade is ineffective in controlling the rhythm, a class I antiarrhythmic agent may be appropriate. Procainamide (IA), flecainide and propafenone (IB) are alternatives for patients not responding to beta blockade (Table 1). For patients who are unresponsive to the beta blockade or class I agents, a class III drug such as amiodarone may be successful in terminating SVT. For cases of SVT that do not respond to single pharmacologic agents, a combination of different medications can be tried. Commonly used combinations include digoxin with propranolol and digoxin with amiodarone. The reported success rate for the combination of amiodarone and propranolol is 80% and flecainide with sotalol is 100% in controlling single or multidrug refractory SVT.[12, 13]

**Table 1**
***Pharmacotherapy for SVT in neonates.***

| SNo | Drug | Mechanism of action | Dosage | Side effects |
|---|---|---|---|---|
| 1 | Adenosine | It produces AV block (may appear as a brief episode of asystole), | 0.05 to 0.2 mg/kg rapid IV bolus followed by a saline flush | Flushing, bronchospasm, transient premature ventricular contractions, premature atrial contractions, sinus bradycardia, tachycardia, as well as varying degrees of AV nodal block. |
| 2 | Digoxin | Digoxin acts by decreasing conduction through the AV node | Oral total digitalizing dose (TDD) is 20 to 30 mcg/kg in preterm neonates and 25 to 35 mcg/kg in term neonates.* | Nausea, vomiting, heart block, and rhythm disturbances. |
| 3 | Esmolol | Increase in refractory period of the AV node, thus interrupting the re-entry circuit. | 100 to 500 mcg/kg loading dose over 1 minute followed by a continuous infusion of 50 to 100 mcg/kg per minute.† | Bradycardia and bronchospasm |
| 4 | Propranolol | - do - | 0.25 mg/kg every 6 to 8 hours and can be increased slowly to a maximum of 4 mg/kg per dose | Bradycardia, hypotension, impaired myocardial contractility and bronchospasm |
| 5 | Procainamide | decreases myocardial excitability and conduction velocity and depresses myocardial contractility | 3 to 6 mg/kg IV in a loading dose of over 5 minutes, repeated every 5 to 10 minutes to a maximum of 15 mg/kg, and followed by a continuous infusion of 20 to 80 mcg/kg per minute. | ventricular tachycardia, lupuslike syndrome, nausea, vomiting, diarrhea, and liver dysfunction. |
| 6 | Flecainide | effects on the accessory connection and the AV node | 2 mg/kg IV over 10 minutes during the acute phase followed by a dose of 3 to 6 mg/kg per day orally in two divided doses. | negative inotropic effect and can be proarrhythmic |
| 7 | Propafenone | - do - | IV dose of 1 to 2 mg/kg can be used for acute control of SVT. The oral dose of propafenone is 8 to 10 mg/kg per day divided in three to four equal doses | negative inotropic effects, proarrhythmic effects, sinus node dysfunction, blood dyscrasias, and neurologic effects. |
| 8 | Amiodarone | Inhibits adrenergic stimulation, prolongs the action potential and refractory period in myocardial tissue, and decreases AV conduction and sinus node function. | IV loading dose of 5 mg/kg over 1 hour, followed by a continuous infusion of 5 mcg/kg per minute, with dose increases until either conversion of SVT or attainment of a maximum of 15 mcg/kg per minute | arrhythmias, hypothyroidism, liver dysfunction, pulmonary fibrosis, and corneal opacities |

*

***The parenteral TDD is 75% of the oral TDD. The dose is reduced by 50% in patients receiving concomitant amiodarone or patients who have end-stage renal disease.***

***† Esmolol can be titrated upward by 50-mcg/kg per minute increments every 5 to 10 minutes to a maximum dose of 1,000 mcg/kg per minute until either there is control of the ventricular rate or hypotension develops.***

Chronic medical treatment is appropriate for neonates who have hemodynamically significant SVT, frequent SVT requiring medical management, pre-excitation on ECG, or congenital cardiac defect. For neonates who have had self-terminating asymptomatic SVT with no evidence of pre-excitation on ECG and with normal cardiac anatomy, chronic medical

treatment may be deferred. A beta blocker such as propranolol is most appropriate for patients responding to esmolol or who have evidence of pre-excitation on ECG. Patients who achieved control of SVT with class I or class III agents are treated with oral preparations of the same medications. Because 70% of neonates who have SVT lose inducibility by 1 year of age, most cardiologists treat for about 6 to 9 months, adjusting dosage for size, and subsequently allow the patient to outgrow the drug dose by about 1 year of age. Rarely, radiofrequency catheter ablation is required for SVT recalcitrant to medical therapy.

## CONCLUSION

SVT is an uncommon, yet life threatening event in the NICU, which necessitates a standard protocol for its management. SVT, which can be recurrent and persistent, is the most common symptomatic arrhythmia. The most common type of SVT in infancy results from a re-entry circuit between the atria and the ventricles. SVT inducibility is lost in 70% of infants by 1 year of age. Twelve-lead ECG during and after SVT along with the response to adenosine can facilitate accurate diagnosis. SVT can have varying clinical presentations, ranging from incidental detection to hemodynamic collapse. Hemodynamic instability warrants immediate restoration to sinus rhythm, best achieved by synchronized electrical cardioversion. Medical management of SVT consists of a trial of vagal maneuvers, adenosine, and medications to maintain sinus rhythm such as beta blockers and class I or class III antiarrhythmic medications. For neonates who have hemodynamically significant SVT, frequent SVT requiring medical management, pre-excitation on ECG, or congenital cardiac defect, chronic medical treatment is appropriate

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