Trust Guidelines for Acute Onset Supraventricular Tachycardia (SVT) in Children

Document Control:

For Use In:	Norfolk and Norwich University Hospitals (NNUH) Children's Assessment Unit (CAU), Children's Wards, Neonatal Intensive Care Unit (NICU), Accident & Emergency (A&E)		
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4	June 2023	Consultant Neonatologist & Paediatrician with Expertise in cardiology Consultant Paediatrician with expertise in cardiology (NNUH)	Author reviewed but no clinical changes at this time ; GOSH network guideline attached for reference (2023) Transferred to new Trust Docs template

Previous Titles for this Document:

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Joint Trust Guidelines for Acute Onset Supraventricular Tachycardia (SVT) in Children	Jun 2023

Note which Trust, where applicable.

Distribution Control

Printed copies of this document should be considered out of date. The most up to date version is available from the Trust Intranet.

Consultation

The following were consulted during the development of this document:
Dr Priya Muthukumar, Chief of service Paediatrics (Document authors' Line Manager)
Dr Graham Derrick and Dr Florian Moenkemeyer Consultant Paediatric Cardiologists at (NNUH) & Great Ormond Street Hospital for Sick Children
Consultants in Paediatric Medicine, Accident & Emergency

Monitoring and Review of Procedural Document

The document owner is responsible for monitoring and reviewing the effectiveness of this Procedural Document. This review is continuous however as a minimum will be achieved at the point this procedural document requires a review eg. changes in legislation, findings from incidents or document expiry.

Relationship of this document to other procedural documents

This document is a clinical guideline applicable to Norfolk and Norwich University Hospitals (NNUH); please refer to local Trust's procedural documents for further guidance.

Guidance Note

This guideline has been approved by the Trust's Clinical Guidelines Assessment Panel as an aid to the diagnosis and management of relevant patients and clinical circumstances. Not every patient or situation fits neatly into a standard guideline scenario and the guideline must be interpreted and applied in practice in the light of prevailing clinical circumstances, the diagnostic and treatment options available and the professional judgement, knowledge and expertise of relevant clinicians. It is advised that the rationale for any departure from relevant guidance should be documented in the patient's case notes.

The Trust's guidelines are made publicly available as part of the collective endeavour to continuously improve the quality of healthcare through sharing medical experience and knowledge. The Trust accepts no responsibility for any misunderstanding or misapplication of this document**.**

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Joint Trust Guidelines for
Acute Onset Supraventricular Tachycardia (SVT) in Children

Quick reference guideline for management of SVT in children

(Please refer to notes below the algorithm on adenosine doses as per cBNF)

Assess Airway, Breathing, Circulation
Continuous ECG monitoring/print out ECG traces

***<u>Neonate until 1 year</u>** – please use **Adenosine 150 micrograms/kg** with increments of 100 micrograms/kg if no response

1. Introduction

1.1. Rationale

Supraventricular tachycardia (SVT) is defined as an abnormally rapid heart rate and rhythm originating above the ventricles, often (but not always) with a narrow QRS complex. Most common forms of SVT in infants and children are

1. Atrioventricular re-entrant tachycardia (AVRT), including the Wolff-Parkinson-White (WPW) syndrome,
2. Atrioventricular nodal re-entrant tachycardia (AVNRT).
3. Atrial flutter – commonly seen in antenatal/neonatal period and rare in children
4. Ectopic atrial tachycardia and Junctional ectopic tachycardia are rare forms of SVT

SVT is the most common non arrest arrhythmia during childhood and is the most common arrhythmia that produces cardiovascular instability in infancy. Population-based study report a prevalence of supraventricular arrhythmia of 2.25/1000 persons with an annual incidence in children <19 years of age of 13/100 000.

Acute management of the child who presents in SVT can be a challenge because the exact mechanism of the tachycardia often is unknown. The treatment strategy depends upon the patient's presentation and clinical status (haemodynamic stability or instability). The approach consists of initiating therapy while continuing to assess the patient's condition. **ECG monitoring before, during and after treatment is crucial to ensure** correct rhythm identification and response to treatment.

1.2. Objectives

The objective of this clinical guideline is to

- Provide guidance on the prompt recognition and management of Supraventricular tachycardia (SVT) in children presenting acutely to A&E or the Jenny Lind Children's Department.

1.3. Scope

To be used in management of SVT in babies and children up to 16 years old in following areas NICU, Children's ED (CHED), Children's assessment unit and Buxton inpatient ward. Can be accessed by all staff groups who are treating the child.

1.4. Glossary

The following terms and abbreviations have been used within this document:

Term	Definition
NNUH	Norfolk and Norwich University Hospitals
CAU	Children's Assessment Unit
NICU	Neonatal Intensive Care Unit
A&E	Accident and Emergency
SVT	Supraventricular tachycardia
WPW	Wolff-Parkinson-White
AVRT	Atrioventricular re-entrant tachycardia
AVNRT	Atrioventricular nodal re-entrant tachycardia

CHF	Congestive Heart Failure
VT	Ventricular tachycardia
ECG	Electrocardiogram
CRT	Capillary refill time
BP	Blood pressure
GOSH	Great Ormond Street Hospital
AV	Arteriovenous
TDS	Three times a day
LFT	Liver function test
TFT	Thyroid function test
BD	Twice daily
PMHx	Past medical history
PECs	Paediatrician with Expertise in cardiology
EIA	Equality Impact Assessment

2. Responsibilities

Document authors:
Dr Rahul Roy, Consultant Neonatologist & Paediatrician with Expertise in cardiology
Dr Aravind Shastri, Consultant Paediatrician with expertise in cardiology

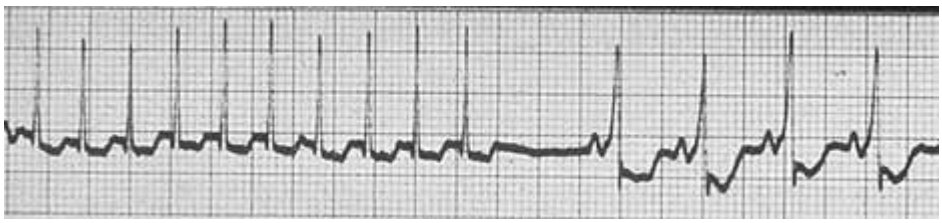
3. Processes to be followed

3.1. Presentation

Older children usually complain of light-headedness, dizziness, chest discomfort, or note the fast heart rate (palpitations), but very rapid rates may be undetected for long periods in young infants until they develop a low cardiac output state, shock or cardiac failure. Presentation in infants may include poor feeding, irritability, tachypnoea and mottled skin. In utero SVT is a known cause of hydrops foetalis.

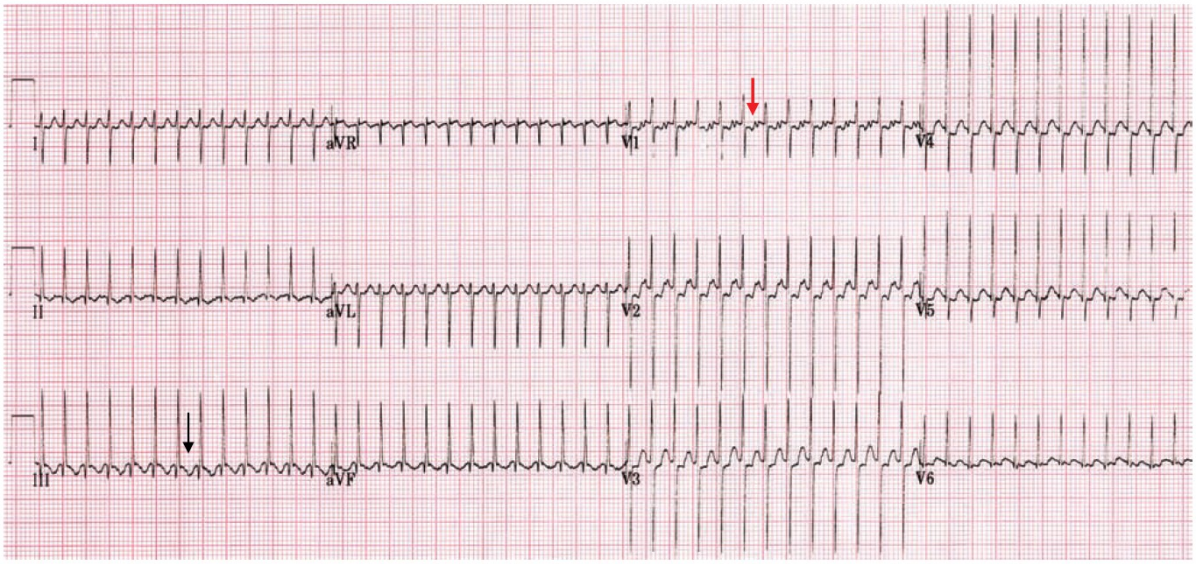
Below are example of SVT rhythm on ECG recording:

1) **AV re-entrant tachycardia**



AV re-entrant tachycardia breaking to sinus rhythm with pre-excitation (Wolff-Parkinson-White syndrome).

2) **Narrow complex Tachycardia in a neonate**



3.2. Diagnosing SVT in infants and children

SVT in infants generally produces a heart rate >220 bpm, and sometimes as high as 300 bpm. Lower heart rates like 180 bpm or above may occur in children during an attack of SVT. The QRS complex is narrow, making differentiation between marked sinus tachycardia due to shock and SVT difficult, particularly because SVT may be associated with poor systemic perfusion.

SVT, seen in the first year of life i.e. in infancy is more likely to have accessory AVRT, and an adolescent who has first SVT is more likely to have nodal AVNRT. AVNRT is more influenced by increased sympathetic tone than AVRT. AVNRT is more likely triggered by physical activity, emotional stress, and abrupt changes in body position. AVNRT are less likely to be incessant and therefore rarely causes a tachycardia induced cardiomyopathy.

The following characteristics ***may*** help to distinguish between sinus tachycardia and SVT

1. Sinus tachycardia is typically characterised by a heart rate less than 200 per minute in infants and children, whereas infants with SVT typically have a heart rate greater than 220 beats per minute. P waves are usually present.
2. P-waves may be difficult to identify in both sinus tachycardia and SVT, once the ventricular rate exceeds 200 beats per minute. If P-waves are identifiable, they are

usually upright in leads I and AVF in sinus tachycardia while they are negative in leads II, III and AVF in SVT, although this is not always reliable. P waves are usually invisible in SVT.

3. In sinus tachycardia, the heart rate varies from beat to beat and is often responsive to stimulation, but there is no beat-to-beat variability in SVT.
4. Termination of SVT is abrupt whereas the heart rate slows gradually in sinus tachycardia in response to treatment such as fluid resuscitation in shock scenarios.
5. A history consistent with shock (e.g. gastroenteritis or septicaemia) is usually present with sinus tachycardia.

Many infants tolerate SVT well. If the tachycardia is sustained for 6 to 12 hours, signs of cardiac heart failure (CHF) usually develop in infants. When CHF develops, the infant's condition can deteriorate rapidly. Older children may complain of chest pain, palpitation, shortness of breath, light headedness, and fatigue

Note: A wide QRS complex tachycardia should always be managed as VT (ventricular tachycardia) until proven otherwise.

3.3. Assessment

3.3.1. History

- Onset
- Associated pain, dyspnoea, syncope or dizziness
- Infants – poor feeding, pallor, tachypnoea, irritability
- Older children – palpitations, chest discomfort
- Medication
- PMHx – Congenital cardiac problems/surgery
- Sometimes diagnosed antenatally (atrial flutter)

3.3.2. Clinical Assessment

- **A**irway and **B**reathing
- **C**irculation
    - ECG strip and 12 lead ECG
    - Assess for signs of cardiogenic shock
        - Prolonged CRT
        - Low BP
        - Acidotic Blood Gas
        - Gallop rhythm
        - Enlarged liver
    - Discuss with cardiology team early
- **D**isability

Agitation, confusion

- **E**xposure

  Rule out other causes of presentation (as above)

- **E**lectrolytes

  Check electrolytes (including Mg, PO_4, Ca, K)

- Check drug levels (if on theophylline or digoxin)
- Infection

  May be a presenting feature of myocarditis

Notes to guide the use of algorithm above

- **Assess Airway, Breathing and Circulation. Inform Duty Paediatric Consultant**
- **Continuous ECG monitoring which allows printouts of traces.**

Try **vagal stimulation** while continuing ECG monitoring. The following techniques can be used.

1. Elicit the "diving reflex" in an infant with a narrow complex tachycardia who is not haemodynamically compromised ; diving reflex produces an increase in vagal tone, slows atrioventricular conduction and interrupts the tachycardia. In the case of a baby, the infant should be wrapped in a towel and his/her whole face immersed into a bowl of cold water for about five seconds. There is no need to obstruct the mouth or nostrils as the baby will be temporarily apnoeic. For an older child an ice-water soaked cloth is placed on the nose and mouth.

2. Older children can try a Valsalva manoeuvre. Some children know that a certain position or action will usually effect a return to sinus rhythm like a headstand. Blowing hard through a straw may be effective for some children. *Ocular pressure or carotid body massage* should not be attempted in infants or children. Carotid massage rarely works and is not advisable (in infants could cause airway obstruction).

If the vagal manoeuvre is ineffective, give:

3.4. Intravenous Adenosine cBNF dosing schedule

3.4.1. Neonates:

150 micrograms/kg; increase dose every 2 minutes by 50-100micrograms/kg until tachycardia terminated or until maximum single dose of 300 micrograms/kg given.

3.4.2. Child 1 month-1 year:

150micrograms/kg; increase dose every 2 minutes by 50-100micrograms/kg until tachycardia is terminated or maximum single dose of 500micrograms/kg given.

3.4.3. Child 1-12 years:

100micrograms/kg; increase dose every 2 minutes by 50-100 micrograms/kg until tachycardia terminated or maximum single dose 500micrograms/kg (max. 12 milligrams) given.

3.4.4. Child 12-18 years:

Initially 3 milligrams; if necessary increase dose to 6 milligrams after 2 minutes, then to 12 milligrams after further 2 minutes.

Intravenous adenosine (to be given rapidly into large peripheral or central vein and followed promptly by 0.9% sodium chloride flush). Refer to dose schedule above, as per cBNF.

Starting dose of 100 micrograms/kg rapid (<2 seconds) rapid IV bolus

Wait 2 mins → 200 micrograms/kg rapid IV bolus

Wait 2 mins → 300 micrograms/kg rapid IV bolus

Maximum single dose is 500 micrograms/kg IV (300 micrograms/kg under one month) up to a maximum of 12mg.

Stop at anytime as soon as the child reverts to sinus rhythm.

Adenosine should be given as a rapid (<2 seconds) bolus into a large peripheral vein and rapidly followed by a 5mL 0.9% sodium chloride flush(so that it reached the heart quickly as adenosine is metabolised very quickly).

Obtain a continuous printed ECG recording during adenosine administration and wait for 20-30 secs post administration before discontinuing recording.

Discuss with Paediatric Consultant/Paediatric Cardiologist regarding consideration of an alternative antiarrhythmic agent if SVT recurs despite higher adenosine doses (400-500 micrograms/kg). Please involve GOSH cardiology team in all resistant SVT cases and seek their advice.

Consider conference call between Cardiology/Transport teams in sick children.

Side effects are short-lived but include flushing, nausea, dyspnoea and chest tightness. There will be AV block and ventricular asystole (the drug works by blocking the AV node and interrupting a reciprocating tachycardia) if the drug is given correctly, this is the intention and is short lived. It is worth warning parents and staff that this will happen.

If a child with stable supraventricular tachycardia has not been converted to a normal rhythm with intravenous adenosine it is essential to seek the advice of a paediatric cardiologist before further treatment. After discussion with duty Paediatric Consultant, contact the on call cardiology registrar at Great Ormond Street Hospital and fax copy of ECG recording for further advice on management.

The use of one of the following may be suggested, only to be given with intensive monitoring, regular 12 lead ECGs recording and with the knowledge and approval or recommendation of a paediatric cardiologist in a tertiary cardiology centre.

1. Amiodarone

This drug can be used in refractory atrial tachycardia. The dose is 5mg/kg given intravenously over 20-30 minutes diluted in approximately 4mLs/kg of 5% dextrose.

Can use a longer safer loading dose of 25 micrograms/kg/min for 4 hours (make infusion so that 1mL/hr =10micrograms/kg/min i.e. run this at 2.5mL/hour for 4 hours) and then a maintenance of 5-15 micrograms/kg/min i.e. 0.5-1.5ml/hour (maximum 1.2 grams/24 hours). It is thrombophlebotic and therefore best given through a central line. Least negatively inotropic.

Maintenance infusion continues for at least 24 hours and overlaps with oral amiodarone for 24 hours, once tolerating feeds.

Dose for oral amiodarone as recommended by Great Ormond Street Hospital: Amiodarone 5 milligrams/kg TDS for 1 week, 5 milligrams/kg BD for further 1 week, then 5 milligrams/kg OD to continue (please discuss length of treatment with Dr Derrick's team at Great Ormond Street Hospital

Before starting Amiodarone, it is important to counsel the family about blood tests to include LFT's and TFT's as part of the monitoring. Also, education about increased susceptibility to sunburn and therefore the importance of using sunblock protection and shade is recommended.

3. **Magnesium sulphate**

Magnesium Sulphate is used as an adjunct at dosages of 25 -50 mg/kg(maximum of 2 g) in resistant atrial tachycardia as infusion over 15 minutes.

4. **Flecainide**

2mg/kg intravenously over 30 minutes with continuous monitoring of ECG looking particularly at QRS duration.

This drug is particularly useful in refractory Wolff-Parkinson-White type tachycardia (resistant re-entry supraventricular tachycardia). It is a membrane stabiliser but can be pro-arrythmic and has a negative inotropic effect, therefore do not give if myocardial function is impaired. Levels need monitoring if continued for more than 24 hours.

5. **Propranolol and Verapamil (rarely used)**

IV propranolol may be used to treat SVT in the presence of WPW syndrome.

IV verapamil (may be indicated for fascicular ventricular tachycardia) should be avoided in infants younger than 12 months of age because it may produce extreme bradycardia and hypotension in infants.

6. **Patient information leaflet ;** please print the info leaflet from GOSH website

(link as below)
https://www.gosh.nhs.uk/conditions-and-treatments/conditions-we-treat/supraventricular-tachycardia

7. **GOSH network guidance**

GOSH team have produced a more detailed network guidance for use by PECs or other paediatricians for a detailed reference ; see PDF below

4. Training & Competencies

SVT is an important condition and is covered under APLS, EPLS life support courses and is also regularly taught in medical/postgraduate programmes in paediatrics.

5. References

1. Pharmacological and non-pharmacological therapy for arrhythmias in the paediatric population. EHRA and AEPC-Arrhythmia Working Group joint consensus statement. EP-Europace. 2013; Europace (2013)
2. Clinical guideline – SVT in children from Children Acute Transport Service
3. Update January 2011 to Advanced Paediatric Life Support: The Practical Approach 5th Edition
4. Cochrane Review on Supraventricular Tachycardia
5. Sherwood MC, Lau KC, Sholler GF. Adenosine in the management of supraventricular tachycardia in children. *Journal of Paediatrics and Child Health* 1998 Feb;34(1):53-56
6. Dixon J, Foster K, Wyllie J, Wren C. Guidelines and adenosine dosing in supraventricular tachycardia. *Archives of Disease in Childhood.* 2005;90:1190-1191
7. Josephson ME, Wellens HJ. Differential diagnosis of supraventricular tachycardia. Cardiol Clin 1990; 8:411
8. Salerno J, Seslar P. Supraventricular Tachycardia (review article). Arch Pediatr Adolesc Med/Vol 163(No 3), March 2009
9. cBNF 2018 edition
10. Drug doses, Intensive Care Unit, Royal Children's Hospital, Melbourne

6. Audit of the service

Regular audits are necessary; if there is any cases that have deviated from standard guideline, we will come to know about them via Datix system and will be discussed in morbidity/mortality meetings

Compliance with the process will be monitored through the following:

Key elements	Process for Monitoring	By Whom (Individual / group /committee)	Responsible Governance Committee /dept	Frequency of monitoring
Case discussions in morbidity meetings if the management has deviated significantly from above guidelines	Morbidity meeting Departmental educational meeting	Dr Roy an Dr Shastri	PECs at NNUH	3 years

7. Equality Impact Assessment (EIA)

Type of function or policy	Existing

Division	Paediatrics	Department	Womens and Childrens

Name of person completing form	Aravind Shastri	**Date**	11th August 2023

Equality Area	**Potential** **Negative Impact**	**Impact** **Positive Impact**	**Which groups are affected**	**Full Impact Assessment Required YES/NO**
Race	N/A	N/A	N/A	N/A
Pregnancy & Maternity	N/A	N/A	N/A	N/A
Disability	N/A	N/A	N/A	N/A
Religion and beliefs	N/A	N/A	N/A	N/A
Sex	N/A	N/A	N/A	N/A
Gender reassignment	N/A	N/A	N/A	N/A
Sexual Orientation	N/A	N/A	N/A	N/A
Age	N/A	N/A	N/A	N/A
Marriage & Civil Partnership	N/A	N/A	N/A	N/A
EDS2 – How does this change impact the Equality and Diversity Strategic plan (contact HR or see EDS2 plan)?		N/A		

- **A full assessment will only be required if: The impact is potentially discriminatory under the general equality duty**
- **Any groups of patients/staff/visitors or communities could be potentially disadvantaged by the policy or function/service**
- **The policy or function/service is assessed to be of high significance**

IF IN DOUBT A FULL IMPACT ASSESSMENT FORM IS REQUIRED

The review of the existing policy re-affirms the rights of all groups and clarifies the individual, managerial and organisational responsibilities in line with statutory and best practice guidance.