

10

The child with an abnormal pulse rate or rhythm

LEARNING OBJECTIVES

In this chapter, you will learn:

- how to assess children with an abnormal pulse rate or rhythm
- how to resuscitate the child with life-threatening brady- or tachyarrhythmia

10.1 INTRODUCTION

In tachyarrhythmias in children, the rate is fast but the rhythm largely regular. Causes include:

- Re-entrant congenital conduction pathway abnormality (common)
- Poisoning
- Metabolic disturbance
- After cardiac surgery
- Cardiomyopathy
- Long QT syndrome

In bradyarrhythmias in children the rate is slow and the rhythm usually irregular. Causes include:

- Pre-terminal event in hypoxia or shock
- Raised intracranial pressure
- After conduction pathway damage during cardiac surgery
- Congenital heart block (rare)
- Long QT syndrome.

Presentations include:

- History of palpitations (verbal child)
- Poor feeding (preverbal child)
- Heart failure or shock

10.2 PRIMARY ASSESSMENT

This is dealt with in Chapter 7 “Structured Approach to the Seriously Ill Child”. Below is a summary.

Airway

Assess vocalisations – crying or talking indicate ventilation and some degree of airway patency.
Assess airway patency by

- *looking* for chest and/or abdominal movement, symmetry and recession,
- *listening* for breath sounds and stridor and
- *feeling* for expired air.

Reassess after any airway-opening manoeuvres. If there continues to be no evidence of air movement then airway patency can be assessed by performing an opening manoeuvre and giving rescue breaths (see “Basic Life Support”, Chapter 4).

Breathing

Effort of breathing

respiratory rate

recession

wheeze

accessory muscle use

gaspings

stridor

grunting

flaring of alae nasi

Exceptions

Increased effort of breathing DOES NOT occur in three circumstances:

- *exhaustion*
- *central respiratory depression*
- *neuromuscular disease*

Efficacy of breathing

chest expansion/abdominal excursion

breath sounds – reduced or absent, and symmetry on auscultation

SaO₂ in air

Effects of respiratory failure on other physiology

heart rate

skin colour

mental status

Circulation

Heart rate – This is the defining observation for this presentation. An abnormal pulse rate is defined as one falling outside the normal range given in Chapter 2.

In practice, most serious disease or injury states are associated with a sinus tachycardia. In

infants this may be as high as up to 220 beats per minute (bpm) and in children up to 180 bpm. Rates over these figures are highly likely to be tachyarrhythmias, but in any case of significant tachycardia, i.e. 200 in an infant and 150 in a child, an ECG rhythm strip should be examined and, if in doubt, a full 12-lead ECG performed. Very high rates may be impossible to count manually and the pulse oximeter is often unreliable in this regard. Again a rhythm strip is advised.

An abnormally slow pulse rate is defined as less than 60 bpm or a rapidly falling heart rate associated with poor systemic perfusion. This will almost always be in a child who requires major resuscitation.

Pulse volume
Capillary refill
Skin temperature

Disability

Mental status/conscious level
Posture
Pupils

Exposure – rash or fever

Features suggesting cardiac cause of respiratory inadequacy

- Cyanosis, not correcting with oxygen therapy
- Tachycardia out of proportion to respiratory difficulty
- Raised jugular venous pressure
- Gallop rhythm/murmur
- Enlarged liver
- Absent femoral pulses

10.3 RESUSCITATION

Airway

If the airway is not open, use:

- an airway-opening manoeuvre,
- an airway adjunct or
- urgent induction of anaesthesia followed by intubation to secure the airway.

Breathing

- Give high-flow oxygen through a face mask with a reservoir as soon as the airway has been shown to be adequate.
- If the child is hypoventilating or has bradycardia, respiration should be supported with oxygen via a bag–valve–mask device and consideration given to intubation and ventilation.

Circulation

If there is shock and

- the heart rate <60/min, start chest compressions.
- the ECG shows ventricular tachycardia, give up to one or two synchronous electrical shocks at 1 and 2 J/kg.
 - The child who is responsive to pain should be anaesthetised or sedated first
 - If the synchronous shocks for VT are ineffectual (because the defibrillator cannot recognise the abnormally shaped QRS complex), then the shocks may have to be given asynchronously recognising that this is a more risky procedure – because without conversion the rhythm may deteriorate to VF or asystole.

- Synchronisation relies on the ability of the defibrillator to recognise the QRST complex, and is designed to avoid shock delivery at a point in the cardiac cycle likely to precipitate ventricular fibrillation.
- Gain intravenous or intraosseous access.
- If the tachyarrhythmia is SVT then give intravenous/intraosseous adenosine (see formulary for more on adenosine) 100 micrograms/kg to a maximum single dose of 500mcg/kg (300mcg/kg under one month) if this can be administered more quickly than a synchronous electrical shock.
- Take blood for FBC, renal function, glucose stick test and glucose laboratory test.
- Give a bolus of 20 ml/kg IV of crystalloid to a patient with bradycardia who is in shock.

While the primary assessment and resuscitation are being carried out, a focused history of the child's health and activity over the previous 24 hours should be gained. Certain key features which will be identified clinically in the primary assessment, from the focused history, from the initial blood tests and from the rhythm strip and 12-lead ECG can point the clinician to the likeliest working diagnosis for emergency treatment.

From the ECG the arrhythmia can be categorised by the following simple questions:

1. Is the *rate*
 - too fast?
 - too slow?
2. Is the *rhythm*
 - regular?
 - irregular?
3. Are the QRS *complexes*
 - narrow?
 - broad?

○ *Bradycardia*

- is usually a pre-terminal rhythm. It is seen as the final response to profound *hypoxia* and *ischaemia* and its presence is ominous.
- is precipitated by *vagal stimulation* as occurs in tracheal intubation and suctioning and may be found in post-operative cardiac patients. The rhythm is usually irregular.
- may be seen in patients with *raised intracranial pressure*. These patients will have presented with coma and their management can be found in Chapters 11 and 16.
- can be a side effect of *poisoning* with *digoxin* or *beta-blockers* and the management can be found in Appendix H.

○ *Tachyarrhythmia*

- with a narrow QRS complex on the ECG is *supra-ventricular tachycardia*. The rhythm is usually regular.
- with a wide QRS complex on the ECG is *ventricular tachycardia*; this can be provoked by
 - *hyperkalaemia*.
 - poisoning with *tricyclic antidepressants*, or a combination of *cisapride* and *macrolide antibiotics*. Additional details on the management of the poisoned child with ventricular tachycardia can be found in Appendix H.

10.4 APPROACH TO THE CHILD WITH BRADYCARDIA

In paediatric practice bradycardia is almost always a pre-terminal finding in patients with respiratory or circulatory insufficiency. Airway, breathing and circulation should always be assessed and treated if needed before pharmacological management of bradycardia.

Emergency Treatment of Bradycardia

Reassess ABC

- If there is hypoxia and shock, treat with
 - high concentration of oxygen, bag–mask ventilation, intubation and IPPV;
 - volume expansion (20 ml/kg of 0.9% saline repeated as recommended in the treatment of shock);
 - if the above is ineffective give a bolus of adrenaline (epinephrine) 10 micrograms/kg IV and
 - if the above is ineffective try an infusion of adrenaline (epinephrine) 0.05 to 2 micrograms/kg/min IV.
- If there has been vagal stimulation, treat with
 - adequate ventilation;
 - atropine 20 micrograms/kg IV/IO (minimum dose 100 micrograms; maximum dose 600 μ g)
 - the dose may be repeated in 5 minutes (maximum total dose of 1.0 mg in a child and 2.0 mg in an adolescent) and
 - if IV/IO access is unavailable, atropine (0.04 mg/kg) may be administered tracheally, although absorption into the circulation may be unreliable.
- If there has been poisoning, seek expert toxicology help.

10.5 APPROACH TO THE CHILD WITH SUPRAVENTRICULAR TACHYCARDIA

Supraventricular tachycardia (SVT) is the most common non-arrest arrhythmia during childhood and is the most common arrhythmia that produces cardiovascular instability during infancy. SVT in infants generally produces a heart rate >220 bpm, and often 250–300 bpm. Lower heart rates occur in children during SVT. The QRS complex is narrow, making differentiation between marked sinus tachycardia due to shock and SVT difficult, particularly because SVT may also be associated with poor systemic perfusion.

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 bpm in infants and children whereas infants with SVT typically have a heart rate greater than 220 bpm.
2. P-waves may be difficult to identify in both sinus tachycardia and SVT once the ventricular rate exceeds 200 bpm. 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.
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.
5. A history consistent with shock (e.g. gastroenteritis or septicaemia) is usually present with sinus tachycardia.

Cardiopulmonary stability during episodes of SVT is affected by the child's age, duration of SVT and prior ventricular function and ventricular rate. Older children usually complain of lightheadedness, dizziness or chest discomfort or they note the fast heart rate, but very rapid rates may be undetected for long periods in young infants until they develop a low cardiac output state and shock. This deterioration in cardiac function occurs because of increased myocardial oxygen demand and limitation in myocardial oxygen delivery during the short diastolic phase associated with very rapid heart rates. If baseline myocardial function is impaired (e.g. in a child with a cardiomyopathy), SVT can produce signs of shock in a relatively short time.

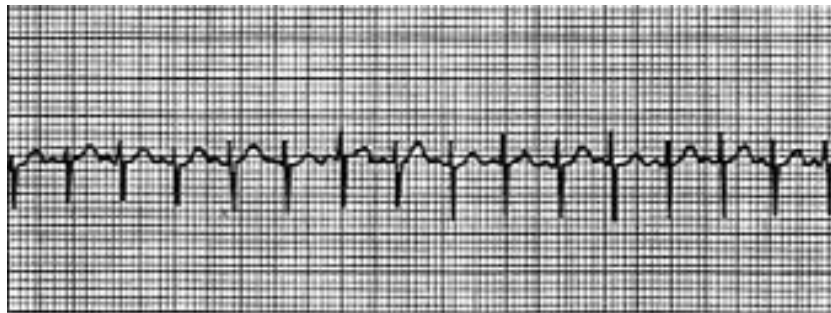


Figure 10.1. Sinus tachycardia

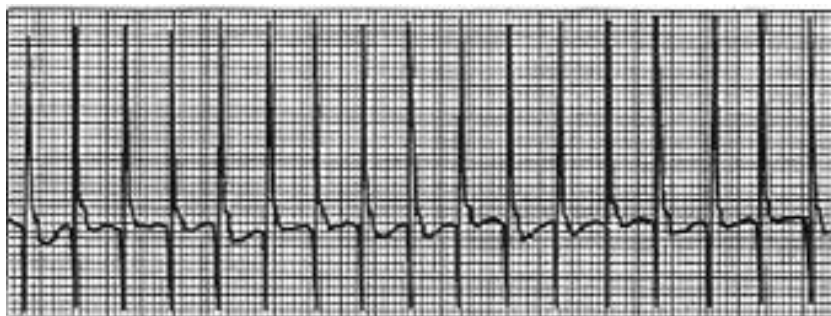


Figure 10.2. Supraventricular tachycardia

Emergency Treatment of Supraventricular Tachycardia

Reassess ABC

- Try vagal stimulation while continuing ECG monitoring. The following techniques can be used:
 - Elicit the “diving reflex”, which produces an increase in vagal tone, slows atrioventricular conduction and interrupts the tachycardia. This can be done by the application of a rubber glove filled with iced water over the face, or if this is ineffectual, wrapping the infant in a towel, and immersing the face in iced water for 5 seconds.
 - One-sided carotid sinus massage.
 - Older children can try a Valsalva manoeuvre. Some children know that a certain position or action will usually effect a return to sinus rhythm. Blowing hard through a straw may be effective for some children.
- Do not use ocular pressure in an infant or child, because ocular damage may result.
- If these manoeuvres are unsuccessful, give:

Intravenous adenosine: Start with a bolus dose of 100 micrograms/kg intravenously and increase the dose to 200 micrograms/kg after 2 minutes if success is not achieved. The next dose should be 300 micrograms/kg. The maximum single dose that should be given is 500 micrograms/kg (300 micrograms/kg under 1 month) up to a maximum of 12 mg. Adenosine is a very rapidly acting drug with a half-life of less than 10 seconds. This means that side effects (flushing, nausea, dyspnoea, chest tightness) are short-lived. It also means, however, that the effect may be short-lasting and the supraventricular tachycardia may recur.

Owing to the same reason, if the drug is given through a small peripheral vein, an insufficiently high concentration may reach the heart and therefore a larger dose may need to be given. Preferably, the drug should be injected into a large peripheral vein and rapidly followed by a saline flush. Adenosine is the drug of choice for supraventricular tachycardia because of its efficacy and safety record.

If the stable supraventricular tachycardia of a child 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. The use of one of the following may be suggested. Only use one drug. If this is unsuccessful seek further cardiological advice. Many of these drugs are unsafe to use in the same patient. The patient can be treated by cardioversion with a general anaesthetic.

Amiodarone: This drug can be used in refractory atrial tachycardia. The dose is 5mg/kg over 30 min diluted in approximately 4ml/kg of 5% dextrose.

Procainamide: This is a calcium channel blocking antiarrhythmic agent that prolongs the refractory period in both atria and ventricles. The loading dose is 15mg/kg given slowly over 30-60 minutes with ECG and BP monitoring. Stop the infusion if the QRS widens or hypotension occurs.

Flecainide (2 mg/kg over 20 minutes): It is a membrane stabiliser but can be proarrhythmic and has a negative inotropic effect.

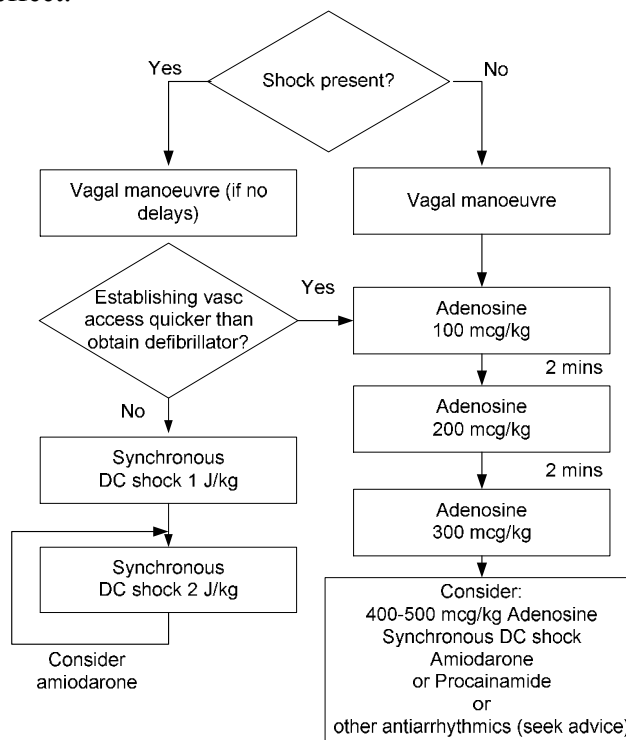


Figure 10.3. Algorithm for the management of supraventricular tachycardia

Digoxin: Dosage schedules vary with age and underlying condition. Seek advice.

Verapamil: This drug has been associated with irreversible hypotension and asystole when given to infants. It therefore should *not be used in children under 1 year of age*. The dose is 100–300 µg/kg, maximum 5 mg. The drug should be terminated when sinus rhythm is seen, even if the calculated dose has not been given. *Do not use if a patient has received β-blockers, flecainide or amiodarone*.

Propranolol (50 micrograms/kg slowly intravenously): Only if pacing is available, because asystole may occur. *Do not give propranolol if the patient has been given verapamil*.

It is unsafe to give verapamil and propranolol to the same patient because they both have negative inotropic actions. It is, however, safe to give propranolol and digoxin.

10.6 APPROACH TO THE CHILD WITH VENTRICULAR TACHYCARDIA

In the haemodynamically stable child with ventricular tachycardia a history should be carefully obtained to identify an underlying cause for the tachycardia because this will often determine ancillary therapy.

Consider underlying causes:

- Congenital heart disease and surgery
- Poisoning with tricyclic antidepressants, procainamide, quinidine, cisapride and macrolide antibiotics, or terfenadine with grapefruit juice
- Renal disease or other cause of hyperkalaemia
- Long QT syndrome

Look for:

- Characteristics of the ECG indicative of torsade de pointes: polymorphic VT with QRS complexes which change in amplitude and polarity so that they appear to rotate around an isoelectric line. Seen in conditions characterised by a long QT interval quinine, quinidine, disopyramide, amiodarone, tricyclic antidepressant and digoxin poisoning and cisapride with erythromycin.

Check serum K, Mg and Ca levels.

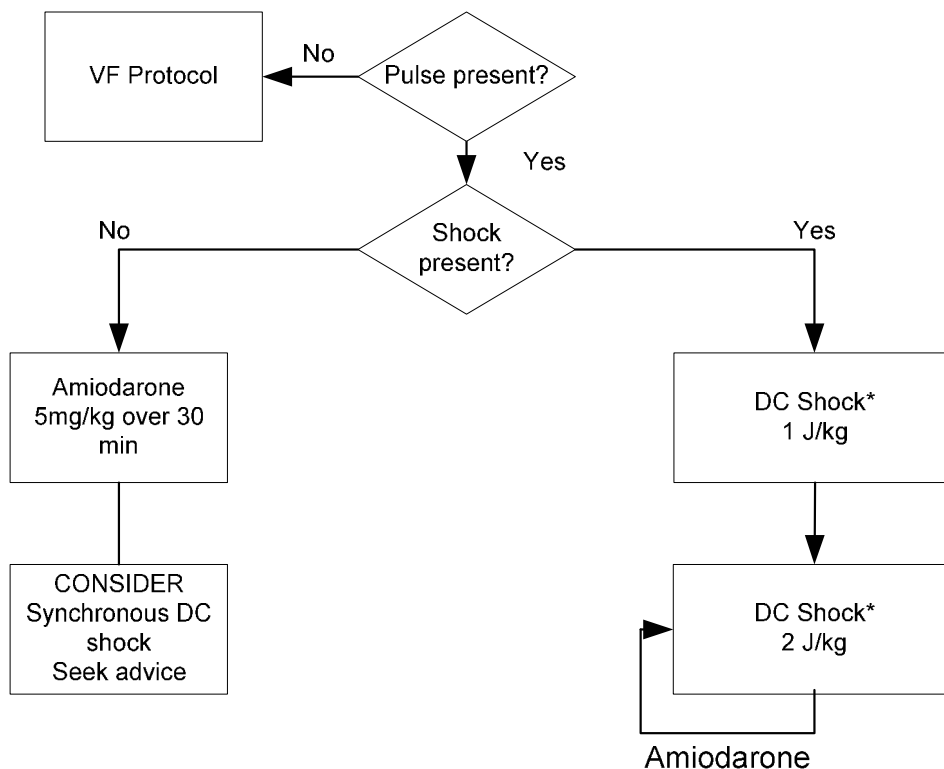
Analysis of the ECG should be done in consultation with a paediatric cardiologist, who should be sent a copy urgently.

Emergency Treatment of Ventricular Tachycardia

Reassess ABC

- The treatment of the haemodynamically stable child with ventricular tachycardia should always include early consultation with a paediatric cardiologist. They may suggest
 - amiodarone (5 mg/kg over 60 minutes) or
 - intravenous procainamide (15 mg/kg over 30–60 minutes, monitor ECG and BP).Both can cause hypotension, which should be treated with volume expansion.

- In cases where the ventricular arrhythmia has been caused by drug toxicity, sedation/anaesthesia and DC shock may be the safest approach. Use synchronous shocks initially, as these are less likely to produce ventricular fibrillation than an asynchronous shock. If synchronous shocks are ineffectual, subsequent attempts will have to be asynchronous if the child is in shock.



* See text for further clarification

Figure 10.4. Algorithm for the management of ventricular tachycardia

- The treatment of torsade de pointes ventricular tachycardia is magnesium sulphate in a rapid IV infusion (several minutes) of 25–50 mg/kg (up to 2 g).
- Amiodarone 5mg/kg may be given over a few minutes in ventricular tachycardia if the child is in severe shock.

It is important not to delay a safe therapeutic intervention for longer than necessary in VT as the rhythm often deteriorates quite quickly into pulseless VT or VF.

Wide-QRS SVT (i.e. SVT with aberrant conduction) is uncommon in infants and children. Correct diagnosis and differentiation from ventricular tachycardia depends on careful analysis of at least a 12-lead ECG that may be supplemented by information from an oesophageal lead. The patient and family history should be evaluated to help identify the presence of an underlying condition predisposing to stable ventricular tachycardia. Because either SVT or VT can cause haemodynamic instability, assumptions about the mechanism (i.e. ventricular versus supraventricular) should not be based solely on the haemodynamic status of the patient.

A dose of adenosine may help identify the underlying aetiology of the arrhythmia, but should be used with extreme caution in haemodynamically stable children with wide-complex tachycardia because acceleration of the tachycardia and significant hypotension are known risks and should not delay definitive treatment in children with shock. Seek advice.

10.7 SUMMARY

You should use the structured approach in the assessment and management of the child with an abnormal pulse rate or rhythm:

- Primary assessment
- Resuscitation
- Secondary assessment and looking for key features
- Emergency treatment
- Stabilisation and transfer to definitive care