GUIDELINES FOR THE MANAGEMENT OF DIABETIC KETOACIDOSIS

We believe these guidelines to be as safe as possible in the light of current evidence.

These are general guidelines for management. Treatment may need modification to suit the individual patient and these guidelines do not remove the need for frequent detailed reassessments of the individual child’s requirements.

These guidelines are intended for the management of the children who have:

- hyperglycaemia (BG >11 mmol/l)
- pH < 7.3
- bicarbonate < 15 mmol/l

AND who are:

- more than 5% dehydrated
- and/or vomiting
- and/or drowsy
- and/or clinically acidotic

Children who are 5% dehydrated or less and not clinically unwell usually tolerate oral rehydration and subcutaneous insulin. Discuss this with the senior doctor on call.

A. GENERAL:
Always consult a more senior doctor on call as soon as you suspect DKA even if you feel confident of your management.

REMEMBER: Children can die from DKA
They can die from -

- **Cerebral oedema.**
  This is unpredictable, occurs more frequently in younger children and newly diagnosed diabetic patients and has a mortality of around 25%. The causes are not known, but this protocol aims to minimise the risk by producing a slow correction of the metabolic abnormalities.

- **Hypokalaemia.**
  This is preventable with careful monitoring and management

- **Aspiration pneumonia.**
  Use a naso-gastric tube in semi-conscious or unconscious children.

### B. EMERGENCY MANAGEMENT:

**B.1 General resuscitation: A, B, C**

**Airway**
- Ensure that the airway is patent and if the child is comatose, insert an airway.
- If comatose or has recurrent vomiting, insert N/G tube, aspirate and leave an open drainage.

**Breathing**
- Give 100% oxygen by face-mask.

**Circulation**
- Insert IV cannula and take blood samples (see below). Cardiac monitor for T waves (peaked in hyperkalaemia)
- If shocked (poor peripheral pulses, poor capillary filling with tachycardia, and/or hypotension) give 10 ml/kg 0.9% (normal) saline as a bolus, over 10-30 min and repeat until the peripheral circulation has improved to a maximum of 30 ml/kg.

**Extreme caution is required when exceeding 30ml/kg because of the fear of cerebral oedema**

*(There is no evidence to support the use of colloids or other volume expanders in preference to crystalloids)*
B.2 Confirm the diagnosis:

- **History**: polydipsia, polyuria
- **Clinical**: acidotic respiration, dehydration, drowsiness, abdominal pain/vomiting
- **Biochemical**: high blood glucose on finger-prick test, glucose and ketones in urine

B.3 Initial investigations:

<table>
<thead>
<tr>
<th>Degree of dehydration</th>
<th>Establishing cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (5%)</td>
<td>Dry mucous membranes, reduced skin turgor</td>
</tr>
<tr>
<td>Moderate (7.5%)</td>
<td>Above with sunken eyes, poor capillary return</td>
</tr>
<tr>
<td>Severe (10% - shock)</td>
<td>Severely ill with poor perfusion, thready rapid pulse, (reduced blood pressure is not likely and is a very late sign)</td>
</tr>
</tbody>
</table>

(DKA may rarely be precipitated by sepsis, and fever is not part of DKA.)

C. FULL CLINICAL ASSESSMENT AND OBSERVATIONS:

Assess and record in the notes, so that comparisons can be made by others later.

Careful and frequent monitoring to detect warning signs of complications is of paramount importance. Neurological observation, pulse and respiratory rate and blood pressure should be monitored at least hourly.

An accurate record of fluid input and output is essential. ECG monitoring is required.

C.1 Degree of dehydration -

<table>
<thead>
<tr>
<th>Assessment of severity</th>
<th>Establishing cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood glucose</td>
<td>Blood and urine cultures</td>
</tr>
<tr>
<td>Urea and electrolytes, creatinine</td>
<td>Chest x-ray</td>
</tr>
<tr>
<td>Capillary, venous or arterial blood gas</td>
<td></td>
</tr>
<tr>
<td>Haemoglobin and packed cell volume</td>
<td>Leukocytosis is a feature of DKA; it does not indicate infection</td>
</tr>
<tr>
<td>Urinary glucose and ketones</td>
<td></td>
</tr>
</tbody>
</table>
C.2 Conscious level
Institute hourly neurological observations whether or not drowsy on admission.

Assess conscious level according to AVPU (Alert, responds to Voice, responds to pain, Unresponsive) during initial assessment. A formal GCS should be done in cases of decreased conscious level or if there is any concern. Keep reassessing as treatment continues.

If in coma on admission, or there is any subsequent deterioration,
- record Glasgow Coma Score (see Appendix)
- transfer to PICU (x)
- consider instituting cerebral oedema management (see below) (x)

C.3 Full examination - looking particularly for evidence of–
- cerebral oedema headache, irritability, slowing pulse, rising blood pressure, reducing conscious level
  N.B. Examine fundi but papilloedema is a late sign.
- infection
- ileus

WEIGH THE CHILD
If this is not possible because of the clinical condition, use the most recent clinic weight as a guideline, or making use of formula (AGE in years + 4) x 2

C.4 All children with DKA should be nursed in HDU with adequate monitoring and nursing care.

Consider ITU for the following:
- Severe acidosis pH<7.1 with marked hyperventilation
- Severe dehydration with shock (see below)
- Depressed sensorium with risk of aspiration from vomiting
- Very young (under 2 years)
- Staffing levels on the wards are insufficient to allow adequate monitoring.
C.5 Observations to be carried out:
Careful and frequent monitoring to detect warning signs of complications is of paramount importance.

Ensure full instructions are given to the senior nursing staff emphasising the need for:

- An accurate record of fluid input and output
- Hourly BP, heart rate and respiratory rate
- Neurological observation should be done ½ hourly. Symptoms of headache or any change in either conscious level or behaviour should be taken seriously.
- ECG monitoring is required. Changes in the size of the T wave in lead II is a good indicator of hyperkalaemia.
- Measurement of volume of every urine sample, and testing for ketones.
- Capillary blood ketone levels may be available and may be a more sensitive measure of suppression of ketogenesis during treatment.
- Hourly capillary blood glucose measurements (these may be inaccurate with severe dehydration/acidosis but useful in documenting the trends. Do not rely on any sudden changes but check with a venous laboratory glucose measurement).
- Twice daily weight can be helpful in assessing fluid balance.
- Repeat blood gas 2-4 hourly until stable.
- Repeat urea and electrolytes at 2 hours and then every 4 hours until stable.
D. MANAGEMENT

D.1 Fluids

N.B. It is essential that all fluids given are documented carefully, particularly the fluid which is given in emergency department and on the way to the ward, as this is where most mistakes occur. (x)

a) Volume of fluid -
The total fluid requirement can be split into 3 categories:

1. Re-expansion of circulating volume if shocked (By this stage, the circulating volume should have been restored. If not, give a further 10 ml/kg 0.9% saline (to a maximum of 30 ml/kg) over 30 minutes. Discuss with a consultant if the child has already received 30 ml/kg. (x)
2. Deficit (ie fluid loss by dehydration)
3. Maintenance (standard weight-related formula)

Requirement = Maintenance + Deficit

Maintenance fluid

<table>
<thead>
<tr>
<th>Maintenance requirement for 24 hours</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Maintenance</td>
</tr>
<tr>
<td>0 - 2 yrs</td>
<td>80 ml/kg/24 hrs</td>
</tr>
<tr>
<td>3 – 5 yrs</td>
<td>70 ml/kg/24 hrs</td>
</tr>
<tr>
<td>6 – 9 yrs</td>
<td>60 ml/kg/24 hrs</td>
</tr>
<tr>
<td>10 – 14 yrs</td>
<td>50 ml/kg/24 hrs</td>
</tr>
<tr>
<td>adult (&gt;15 yrs)</td>
<td>30 ml/kg/24 hrs</td>
</tr>
</tbody>
</table>

Deficit
Deficit (ml) = % dehydration x body weight (kg) x 1000
Never use more than 10% dehydration in the calculations.
Fluid replacement over 48 hours on hourly basis

Add calculated maintenance for 48 hrs (24 hr maintenance x2) and estimated deficit, subtract the amount already given as resuscitation fluid, and give the total volume evenly over the next 48 hours.

| Hourly rate = | 48 hr maintenance + deficit - resuscitation fluid already given | 48 |

Example:
A 20 kg, 6 year old boy who is 10% dehydrated, and who has already had 20ml/kg saline, will require:

- **Deficit** - 10% x 20 kg = 2000 ml
- **Plus maintenance** each 24 hours - 60ml x 20kg = 1200 ml
- **Maintenance for 48 hr** - 1200ml x 2 = 2400 ml
- **Total** = 4400 ml
- **Minus** (fluid already given)20kg x 20ml - = -400 ml
  = 4000 ml over 48 hours
- **Hourly rate** (ml/hr)- = 83 ml/hour

DO NOT INCLUDE CONTINUING URINARY LOSSES IN THE CALCULATIONS (x)

b) Type of fluid -

- Initially use 0.9% saline. (x)
- Generally once the blood glucose has fallen to 12 mmol/l, replace fluid with 0.45% saline 5% dextrose. (x)
- If this occurs within the first 6 hours, the child may still be sodium depleted. Discuss this with consultant, who may wish to continue with normal saline and added dextrose.
- Check urea & serum electrolytes 2 hours after resuscitation is begun and then at least 4 hourly.
c) Oral fluids

- In severe dehydration, impaired consciousness & acidosis do not allow fluids by mouth. A NG tube may be necessary in the case of gastric paresis. (x)
- Oral fluids (e.g. fruit juice/oral rehydration solution) should only be offered after substantial clinical improvement and no vomiting.
- When good clinical improvement occurs before the 48hr rehydration calculations have been completed, oral intake may proceed and the need for IV infusions reduced to take account of the oral intake.

2. Potassium

Potassium is mainly an intracellular ion, and there is always massive depletion of total body potassium although initial plasma levels may be low, normal or even high. Levels in the blood will fall once insulin is commenced.

Once the child has been resuscitated, potassium should be commenced immediately with rehydration fluid, provided anuria is not reported, the ECG does not show elevated T waves and serum potassium is not elevated(x).

(Therefore initially add 20 mmol KCl (10ml of KCl) to every 500 ml bag of fluid (40 mmol per litre).

Occasionally more potassium is required to maintain safe levels.

Check U & E’s 2 hours after resuscitation is begun and then at least 4 hourly, and alter potassium replacements accordingly. If serum potassium (unhaemolysed) exceeds 5.5mmol/l, potassium replacement should be withheld for 30min after which serum potassium should be repeated urgently.

3. Insulin

Once rehydration fluids and potassium are running, blood glucose will already be falling. However, insulin is essential to switch off ketogenesis and reverse the acidosis.

**Continuous low-dose intravenous infusion** is the preferred method. There is no need for an initial bolus. (x)
Make up a solution of 1 unit per ml. of human soluble insulin by adding 50 units (0.5 ml) insulin to 50 ml 0.9% saline in a syringe pump. Attach this using a Y-connector to the IV fluids already running. **Do not** add insulin directly to the fluid bags.

The solution should then run at **0.1 units/kg/hour** (0.1ml/kg/hour).

- If the rate of blood glucose fall exceeds 5 mmol/l per hour, or falls to around **12 mmol/l**, change the fluid to 0.45% saline with 5% dextrose (see “fluids” above). The insulin dose needs to be **reduced** to 0.05 U/kg/hour to switch off ketogenesis.
- **Do not** stop the insulin infusion and the dose is not reduced below 0.05 U/Kg/h while dextrose is being infused, as insulin is required to switch off ketone production. If the blood glucose is falling too rapidly or levels are less than 4mmol/l, a bolus of 2ml/kg of 10% dextrose should be given and increase the dextrose concentration of the infusion to allow insulin therapy to be continued safely. (x)
- If the blood glucose rises out of control, or the pH level is not improving after 4-6 hours, consult senior medical staff, re-evaluate (possible sepsis, insulin errors or other condition), and consider starting the whole protocol again.

4. **Bicarbonate**
This is not safe and rarely necessary in DKA. Continuing acidosis usually means insufficient resuscitation or insufficient insulin. Bicarbonate should only be considered in children who are profoundly acidic (pH< 6.9) and shocked with circulatory failure. Its only purpose is to improve cardiac contractility in severe shock.

Potential problems associated with bicarbonate therapy are paradoxical CNS acidosis, hypokalaemia, altered calcium ionisation and excessive osmolar load.

Before starting bicarbonate, discuss with senior staff. The quantity should be decided by the paediatric resuscitation team or consultant on-call.

5. **Antibiotics**
Broad spectrum antibiotics should be started pending culture isolates because indicators are often masked by DKA. (x)
E. CONTINUING MANAGEMENT

- Urinary catheterisation should be avoided but may be useful in the child with impaired consciousness.
- Documentation of fluid balance is of paramount importance. All urine samples need to be measured accurately and tested for ketones. All fluid input must be recorded (even oral fluids). (x)
- If a massive diuresis continues, fluid input may need to be increased. If large volumes of gastric aspirate continue, these will need to be replaced with 0.45% saline plus 10 mmol/l KCl.
- Check biochemistry, blood pH, and laboratory blood glucose 2 hours after the start of resuscitation, and then at least 4 hourly. Review the fluid composition and rate according to each set of electrolyte results. (x)
- If acidosis is not correcting, resuscitation may have been inadequate or sepsis or inadequate insulin activity. Check infusion lines, doses of insulin and consider giving more insulin, antibiotics and/or normal saline.

F. CEREBRAL OEDEMA

The signs and symptoms of cerebral oedema include:

- Headache
- Changes in neurological status (restlessness, irritability, increased drowsiness, incontinence)
- Specific neurological signs such as cranial nerve palsies, abnormal posture, rising BP, slowing of pulse are usually late signs.
- More dramatic changes such as convulsions, papilloedema, respiratory arrest are late signs associated with extremely poor prognosis
Management of cerebral oedema

If cerebral oedema is suspected **inform senior staff immediately**.

The following measures should be taken **immediately** while arranging transfer to PICU–

- Exclude hypoglycaemia as a possible cause of any behaviour change(x)
- Give mannitol 1 g/kg stat (= 5 ml/kg mannitol 20% over 20 minutes). This needs to be given as soon as warning signs occur. (x)
- Restrict IV fluids to 2/3 maintenance and replace deficit over 72 rather than 48 hours (x)
- Intubation and ventilation may be required whilst awaiting transfer to PICU.
- If assisted ventilation required maintain pCO₂ above 3.5kPa (26.25mmHg).
- Once the child is stable, exclude other diagnoses by CT scan - other intracerebral events (thrombosis, haemorrhage or infarction) may occur and present similarly.

- A repeated dose of mannitol should be given after 2 hours if no improvement.
- Document all events (with dates and times) very carefully in medical records.

**G OTHER COMPLICATIONS**

- **Hypoglycaemia and hypokalaemia** — Avoid by careful monitoring and adjustment of infusion rates.

- **Aspiration pneumonia** — Avoid by naso-gastric tube in vomiting child with impaired consciousness.

Other associations with DKA require specific management:

Continuing abdominal pain is common and may be due to liver swelling, gastritis, bladder retention, and ileus. However, beware of appendicitis
and ask for a surgical opinion once DKA is stable. A raised amylase is common in DKA.

Other problems are pneumothorax ± pneumo-mediastinum, interstitial pulmonary oedema, unusual infections (e.g. TB, fungal infections), hyperosmolar hyperglycaemic non–ketotic coma, and ketosis in type 2 diabetes. Discuss these with the consultant on-call.

Follow-up insulin therapy

Continue with IV fluids until the child is drinking well and able to tolerate food. Do not expect ketones to have disappeared completely before changing to subcutaneous insulin.

Discontinue the insulin infusion 60 minutes (if using soluble or long-acting insulin) or 10 minutes (if using bi-phasic insulin) after the first subcutaneous injection to avoid rebound hyperglycaemia. Subcutaneous insulin should be started according to local protocols for the child with newly diagnosed diabetes (1.0U/Kg/day), or the child should be started back onto their usual insulin regimen at an appropriate time (discuss with senior staff).

References


2. BSPED GUIDELINES 2004
APPENDIX 1

Glasgow Coma Scale

- **Best Motor Response**
  1 = none
  2 = extensor response to pain
  3 = abnormal flexion to pain
  4 = withdraws from pain
  5 = localises pain
  6 = responds to commands

- **Eye Opening**
  1 = none
  2 = to pain
  3 = to speech
  4 = spontaneous

- **Best Verbal Response**
  1 = none
  2 = incomprehensible sounds
  3 = inappropriate words
  4 = appropriate words but confused
  5 = fully orientated

Maximum score 15, minimum score 3

Modification of verbal response score for younger children:

<table>
<thead>
<tr>
<th>2 - 5 years</th>
<th>&lt;2 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = none</td>
<td>1 = none</td>
</tr>
<tr>
<td>2 = grunts</td>
<td>2 = grunts</td>
</tr>
<tr>
<td>3 = cries or screams</td>
<td>3 = inappropriate crying or unstimulated screaming</td>
</tr>
<tr>
<td>4 = monosyllables</td>
<td>4 = cries only</td>
</tr>
<tr>
<td>5 = words of any sort</td>
<td>5 = appropriate non-verbal responses (coos, smiles, cries)</td>
</tr>
</tbody>
</table>
Algorithm for the Management of Diabetic Ketoacidosis (DKA)

Clinical History
- polyuria
- polydipsia
- weight loss
- abdominal pain
- weakness
- vomiting
- confusion

Clinical Signs
-意识不清
- deep breathing (Kussmaul)
- smell of ketones
- lethargy, disorientation

Biochemical Signs
- ketones in urine or blood
- elevated blood glucose (>11mmol/L)
- acidosis (pH<7.3)
- take blood also for electrolytes, urinalysis
- perform other investigations if indicated

Resuscitation
- Airway + N/G tube
- Breathing (100% O2)
- Circulation (10ml/kg of 0.9% saline repeated until circulation restored, min 3 doses)

No improvement

Re-evaluate
- fluid balance + IV therapy if continued acidosis, may require further resuscitation fluid
- check insulin dose correct consider severe

Brainstem
- Shocks
- Reduced peripheral pulse volume
- Reduced conscious level
- Coma

Intravenous therapy
- calculate fluid requirements
- correct over 48 hours
- 0.9% saline
- add KCl 20 mmol every 500 ml
- insulin 0.1U/kg/hour by infusion

Observations
- hourly blood glucose
- neurological status at least hourly
- hourly fluid input: output
- electrolytes 2 hours after start of IV therapy, then 4-hourly

Blood glucose < 12 mmol/L

Management
- give mannitol 1.0 g/kg
- call senior staff
- source IV fluids by 2:3
- move to ITU
- CT scan when stabilized

Exclude hypoglycaemia
Is it cerebral oedema?

Intravenous therapy
- change to 0.45% saline + dextrose 5%
- continue monitoring as above
- consider reducing insulin 0.05/kg/hour, but only when pH>7.3

Insulin start subcutaneous insulin then stop intravenous insulin 1 hour later

Observations
- hourly blood glucose
- neurological status at least hourly
- hourly fluid input: output
- electrolytes 2 hours after start of IV therapy, then 4-hourly

Management
- give mannitol 1.0 g/kg
- call senior staff
- source IV fluids by 2:3
- move to ITU
- CT scan when stabilized

Exclude hypoglycaemia
Is it cerebral oedema?
Diabetic ketoacidosis – Follow-up of children after discharge

The follow up of known diabetic children who present with diabetic ketoacidosis.

1. Home visits; explore reasons for admission

2. Re-educate
   - Self care
   - Sickness rules
   - Diet
   - 3 day rules

3. Arrange OPD appointment at least 1 month after discharge

4. Explore psychological reasons

**How should blood ketone levels be interpreted?**

Exact guidelines on the interpretation of and response to blood β–hydroxybutyrate levels have not yet been established. The current consensus of opinion, however, suggests that:

<table>
<thead>
<tr>
<th>Blood ketone level</th>
<th>Advice</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.6 mmol/l</td>
<td>No action required. Continue blood glucose testing schedule as suggested opposite.</td>
</tr>
<tr>
<td>0.6 – 1.5 mmol/l</td>
<td>Retest blood glucose and blood ketones in 2-4 hours</td>
</tr>
<tr>
<td>1.5 – 3 mmol/l</td>
<td>May be at risk of developing DKA. Call diabetes care team promptly for advice.</td>
</tr>
<tr>
<td>&gt;3 mmol/l</td>
<td>Requires immediate emergency treatment. <strong>Seek professional help.</strong> (If diabetes care team cannot be contacted, go to Casualty).</td>
</tr>
</tbody>
</table>

**Reference**

GUIDELINES FOR MANAGEMENT OF CARDIAC ARRHYTHMIAS

Introduction

Although primary cardiac events are uncommon in the paediatric age group, the ECGs of all critically ill or injured children should be continuously monitored. Most paediatric arrhythmias are the consequence of hypoxaemia, acidosis, and hypotension. Children with myocarditis or cardiomyopathy are at increased risk of primary arrhythmias, as are children after heart surgery. In addition, a number of drugs taken in therapeutic or toxic amounts may cause arrhythmias.

Among children with arrhythmias, most common dysrhythmias are sinus tachycardia (50%), supra ventricular tachycardia (13%), bradycardia (6%) and atrial fibrillation (5%).

The presentation of dysrhythmias can serve as a diagnostic challenge to most clinicians because most children present with vague and non specific symptoms such as “fussiness” or “difficulty in feeding” or even shock.

If unrecognized and untreated, dysrhythmias would lead to cardiopulmonary arrest.

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart rate (BPM)</th>
<th>PR interval (s)</th>
<th>QRS interval (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st wk</td>
<td>90-160</td>
<td>0.08-0.15</td>
<td>0.03-0.08</td>
</tr>
<tr>
<td>1-3 wk</td>
<td>100-180</td>
<td>0.08-0.15</td>
<td>0.03-0.08</td>
</tr>
<tr>
<td>1-2 mo</td>
<td>120-180</td>
<td>0.08-0.15</td>
<td>0.03-0.08</td>
</tr>
<tr>
<td>3-5 mo</td>
<td>105-185</td>
<td>0.08-0.15</td>
<td>0.03-0.08</td>
</tr>
<tr>
<td>6-11 mo</td>
<td>110-170</td>
<td>0.07-0.16</td>
<td>0.03-0.08</td>
</tr>
<tr>
<td>1-2 y</td>
<td>90-165</td>
<td>0.08-0.16</td>
<td>0.03-0.08</td>
</tr>
<tr>
<td>3-4 y</td>
<td>70-140</td>
<td>0.09-0.17</td>
<td>0.04-0.08</td>
</tr>
<tr>
<td>5-7 y</td>
<td>65-140</td>
<td>0.09-0.17</td>
<td>0.04-0.08</td>
</tr>
<tr>
<td>8-11 y</td>
<td>60-130</td>
<td>0.09-0.17</td>
<td>0.04-0.09</td>
</tr>
<tr>
<td>12-15 y</td>
<td>65-130</td>
<td>0.09-0.18</td>
<td>0.04-0.09</td>
</tr>
<tr>
<td>≥16 y</td>
<td>50-120</td>
<td>0.12-0.20</td>
<td>0.05-0.10</td>
</tr>
</tbody>
</table>
1. Bradyarrhythmias

Bradycardia is defined as a heart rate slower than the lower limit of normal for the patient’s age (Table 01)

Sinus bradycardia, sinus node arrest with a slow junctional or idioventricular rhythm, and atrioventricular (AV) block are the most common preterminal rhythms observed in infants and children.

All haemodynamically unstable slow rhythms require immediate treatment

Several forms of bradycardia due to prolonged atrioventricular conduction have been described as shown in the following ECGs. (Fig. 1 - 4)

Fig. 1  Electrocardiogram in first-degree heart block. Prolonged PR interval and one to one atrioventricular condition.

Fig. 2 Electrocardiogram in second-degree heart block, type I (Wenckebach). The PR interval lengthens each beat until conduction fails and a “beat is dropped.” The QRS is typically “regularly irregular.”
Fig. 3 Electrocardiogram in second-degree heart block, type II. The PR interval is normal and constant, until sudden failure of conduction from atria to ventricle occurs.

Fig. 4 Electrocardiogram in complete heart block. P waves and QRS complexes are occurring independently, and the ventricular rate is slow.

1.1 Causes of bradyarrhythmias
- Hypoxaemia – commonest cause
- Hypothermia
- Acidosis
- Hypotension
- Hypoglycemia

Other causes
- Excessive vagal stimulation eg, induced by suctioning or during endotracheal intubation
- Increased intracranial pressure or brain stem compression
- Post cardiac surgery - secondary to injury to the AV node or conduction system.
- Digoxin toxicity, yellow oleander poisoning and acute inflammatory injury from myocarditis.
1.2 Management of bradyarrhythmias

- Initial treatment should be directed to ensure Airway, Breathing and Circulation with high flow oxygen prior to pharmacological management of bradycardia.
- Reassess ABC
- Identification of the cause.
- Pharmacological management

If symptomatic bradycardia persists despite initial resuscitative measures, pharmacologic intervention is initiated.

**Epinephrine (Adrenaline)**

Is the most useful drug in the treatment of symptomatic bradycardia in an infant or child, except for bradycardia caused by heart block or increased vagal tone.

Bolus of epinephrine 0.01mg/kg (0.1ml/kg of 1 in 10,000 solution) is administered. If no response is seen epinephrine infusion is considered in a starting dose of 0.05 µg/kg/min. (x)

Drug is administered IV or intraosseous. If no circulatory access intra tracheal route can be used though it is less effective.

**Atropine sulphate**

For suspected vagally mediated bradycardia, atropine is the initial drug of choice. Although atropine may be used to treat bradycardia accompanied by poor perfusion or hypotension, epinephrine may be more effective in treating bradycardia accompanied by hypotension.

When indicated, give atropine to treat bradycardia only after ensuring adequate oxygenation and ventilation.

The recommended dose is **0.02 mg/kg**, with a minimum dose of 0.1 mg and a maximum single dose of 0.5 mg in a child and 1.0 mg in an adolescent. The dose may be repeated in 5 minutes, to a maximum total dose of 1.0 mg in a child and 2.0 mg in an adolescent. Larger intravascular doses may be required in special resuscitation circumstances such as organophosphate poisoning.

If intravenous access is not readily available, atropine (0.02 mg/kg) may be administered tracheally, although absorption into the circulation may be unreliable.
Bradycardia algorithm

**BRADYCARDIA**

With a Pulse
Causing cardiopulmonary compromise

- Support ABCs as needed
- Give oxygen
- Attach monitor/defibrillator

**Bradycardia still causing cardiopulmonary compromise?**

- Perform CPR if despite oxygenation and ventilation
  HR < 60/min with poor perfusion

- Support ABCs; give oxygen if needed
- Observe
- Consider expert consultation

**Persistent symptomatic bradycardia?**

- Give epinephrine
  - IV/IO: 0.01 mg/kg
  - Endotracheal tube: 0.1 mg/kg
  - (1:1000: 0.1 mL/kg)
  - Repeat every 3 to 5 minutes

- If increased vagal tone or primary AV block:
  - Give atropine, first dose: 0.02 mg/kg, may repeat.
  - Minimum dose: 0.1 mg; maximum total dose for child: 1 mg.

- Consider cardiac pacing

If pulseless arrest develops, go to Pulsatile Arrest Algorithm

**Reminders**

- During CPR, push hard and fast (at least
  100/min)
- Ensure full chest recoil
- Minimize interruptions in chest compressions
- Support ABCs
- Secure airway if needed, confirm placement

- Look for and treat possible contributing factors:
  - Hypovolemia
  - Hypothermia
  - Hypoxia or ventilation problems
  - Hypoglycemia (in pediatrics)
  - Hypocalcemia
  - Hypomagnesemia
  - Hypokalemia
  - Trauma
  - Torsade de pointes
  - Torsades de pointes (cardiac arrest)
2. Tachydysrhythmias

Tachycardia is defined as a heart rate beyond the upper limit of normal for the patient’s age. (Table 1)

2.1 Initial evaluation, when tachycardia is recognised

- Evaluate the ECG tracing with a view to identify the following
  (a) Regular or irregular?
  (b) QRS complex narrow or wide?
  (c) Is every P wave followed by a QRS complex?

- Check whether the patient has pulse
- If pulse is palpable look for haemodynamical stability

2.2 Classification of Tachyarrhythmias

The tachycardias can be classified on the appearance of the QRS complex.

- Narrow–QRS-complex tachycardias (QRS \(< /= 0.08\) second)
  - Supraventricular tachycardia
  - Sinus tachycardia
  - Atrial fibrillation
  - Atrial flutter
  - Junctional tachycardia

- Wide–QRS-complex tachycardias (QRS > 0.08 second)
  - Ventricular tachycardia (VT)
  - SVT with aberrancy
2.3

Table 2

Diagnosis of tachyarrhythmias

<table>
<thead>
<tr>
<th>Electrocardiographic findings</th>
<th>Heart rate (beats/min)</th>
<th>P wave</th>
<th>QRS duration</th>
<th>Regularity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus tachycardia</td>
<td>Infants &lt;220 Children &lt;180</td>
<td>Always present</td>
<td>Normal</td>
<td>Rate varies with respiration</td>
</tr>
<tr>
<td>Supraventricular tachycardia</td>
<td>Infants ≥220 Children ≥180</td>
<td>Present in 50%</td>
<td>Normal or prolonged (RBBB pattern)</td>
<td>Regular</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>120-180</td>
<td>Fibrillatory waves</td>
<td>Normal or prolonged (RBBB pattern)</td>
<td>Irregularly irregular</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Atrial: 250-400 Ventricular response variable: 100-320</td>
<td>Saw-toothed flutter waves</td>
<td>Normal or prolonged (RBBB pattern)</td>
<td>Regular ventricular response</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>120-240</td>
<td>Absent or atrioventricular dissociation</td>
<td>Usually prolonged</td>
<td>Slightly irregular</td>
</tr>
</tbody>
</table>

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 sometimes as high as 300 bpm. Lower heart rates may be observed in children during SVT. The QRS complex is narrow (i.e., <= 0.08 seconds) in >90% of involved children, making differentiation between marked sinus tachycardia (ST) due to shock and SVT somewhat difficult, particularly because either rhythm may be associated with poor systemic perfusion.

Table 3

<table>
<thead>
<tr>
<th>History</th>
<th>Sinus tachycardia (Fig.5)</th>
<th>Supraventricular tachycardia (Fig. 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Consistent with known cause e.g. hemorrhage; dehydration</td>
<td>Often vague and non specific</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Infants &lt;220 Children &lt;180</td>
<td>Infants ≥220 Children ≥180</td>
</tr>
<tr>
<td>P waves (difficult to identify in ST and SVT when rate &gt;200)</td>
<td>Always present Usually upright in LI and aVF</td>
<td>Present in 50%. Negative in II, III, and aVF</td>
</tr>
</tbody>
</table>

![Fig. 5 Electrocardiogram of sinus arrhythmia. Each QRS complex is preceded by a P wave, but the interval between each P wave is variable, usually changing with the respiratory cycle.](image-url)
Cardiopulmonary stability during episodes of SVT depends on the child’s age, duration of SVT, prior ventricular function, and ventricular rate.

Older children will typically complain of light headedness, dizziness, or chest discomfort, or simply note the fast heart rate. Infants, present with non-specific complaints “fussiness”, lethargy, poor feeding, pallor, sweating with feeds or simply “not being well”.

Very rapid rates may be undetected for long periods until low cardiac output and shock develop.

**SVT can produce signs of shock in a relatively short time.**

**Wide-QRS SVT**

Wide-QRS SVT (ie, SVT with aberrant conduction) is uncommon in infants and children. Correct diagnosis and differentiation from VT depends on careful analysis of at least a 12-lead ECG that may be supplemented by information from an oesophageal lead. In most circumstances, wide-complex tachycardias should be treated as if they are VT.
Management of SVT

- Ensure ABC
- Administer high flow oxygen
- Obtain 12-lead ECG with a rhythm strip
- Differentiate whether the patient is **stable** or **unstable** and manage accordingly
- Long term management

(A) Treatment of stable SVT

Those who are asymptomatic with good perfusion and also those who are in mild heart failure are considered to be stable.

1. Vagal manoeuvres may be tried

Obtain a 12-lead ECG before and after the vagal manoeuvre and monitor the ECG continuously.

- Ice water applied to the face is most effective in infants and young children. One method uses crushed ice mixed with water in a plastic bag or glove. Use care to apply the ice water mixture to the infant’s face without obstructing ventilation.
- One sided carotid body massage
- Valsalva manoeuvre is effective and appear to be safe. In children one technique for performing a Valsalva manoeuvre is to have the child blow through a straw.

*Note that application of external ocular pressure may be dangerous and should not be used to induce a vagal response.*

2. Medications are indicated if these manoeuvres are unsuccessful.

**Adenosine**

- Adenosine is the drug of choice for SVT in children. (x)
- To enhance delivery of the drug to its site of action in the heart, the injection site should be as close to the heart as possible.
- Because of its extremely short half life, adenosine must be pushed and flushed quickly.
- A 2-syringe technique is recommended, 1 syringe containing the drug and 1 containing a saline flush of at least 5 mL.
- With continuous ECG monitoring, administer 50 µg/kg (0.05mg/kg) as a rapid intravenous bolus preferably into a large peripheral vein.
If there is no effect, increase the dose to 100 µg/kg (0.1mg/kg) after 2 minutes.

- The next dose should be 250 µg/kg (0.25mg/kg).
- The maximum recommended is 500 µg/kg (0.50mg/kg) (300µg/kg - 0.30mg/kg, under 01 month).
- A single dose of adenosine should not exceed 12 mg.
- Adenosine may also be given by the intraosseous route.

3. If no response to adenosine, consult a cardiologist and consider alternative drugs or cardioversion

(a) Alternative drugs
- **Verapamil** could be used in a dose of 100-300µg/kg to a maximum of 05mg. Contraindicated under 01 year of age as associated with irreversible hypotension and asystole. Do not use if the patient has already received β blockers, flecainide or amiodarone.
- **Flecainide** in a dose of 2mg/kg over 20 min. This drug is particularly useful in refractory WPW type tachycardia.
- **Amiodarone** can be used in refractory SVT in a dose of 5mg/kg over 30 min diluted in 4ml/kg of 5% dextrose.
- **Propranolol** in a dose of 50 µg/kg slow IV. Only if pacing is available as asystole may occur.
- **Digoxin** could be used. Refer BNF for children for dosage

(b) Cardioversion
- Refer unstable SVT for details.

(B) Treatment of unstable SVT

Those who are in severe heart failure with or without poor perfusion are considered to be unstable

1. **Cardioversion**
   In a child presenting with unstable SVT with severe heart failure and poor perfusion synchronized cardioversion is initiated.

   Synchronized electrical cardioversion is recommended at a starting dose of 0.5 J/kg to a maximum dose of 2J/kg
2. Adenosine

If vascular access is already available, adenosine may be administered before electrical cardioversion.

Cardioversion should not be delayed for attempts at IV access or sedation.

(C) Long-term management

Once stabilised, the majority of patients who present with SVT will need to be admitted to be hospital, to investigate the underline cause of SVT and the need for long-term medical management or radiofrequency ablation.

Key points

Management of SVT

- Adenosine is the drug of choice in stable SVT
- Cardioversion is the treatment of choice in unstable SVT; however adenosine can be tried if IV access is already available.
- Cardioversion should not be delayed for attempts at IV access or sedation.
SVT Algorithm

SVT
- Assess and support ABCs as needed
- Give oxygen
- Attach monitor/defibrillator
- Check stable/unstable

Stable
- Vagal manoeuvres
- Adenosine 0.05 mg/kg rapid IV
  - No response
  - Adenosine 0.1 mg/kg rapid IV
    - No response
    - Adenosine 0.25 mg/kg rapid
      - No response
      - CONSIDER Synchronous DC shock
        - Verapamil
        - Digoxin
        - Amiodarone
        - Flecanide
        - Propranolol

Unstable
- IV access available
- Synchronous DC shock 0.5J/kg
  - No response
  - Synchronous DC shock 1J/kg
    - No response
    - Synchronous DC shock 2J/kg
      - No response
      - Consult cardiologist & consider anti-arrhythmics

- IV access unavailable
Ventricular tachycardia and ventricular fibrillation

Fig. 7 Electrocardiogram of ventricular tachycardia. Wide QRS complexes occurring at regular intervals without evidence of atrial activity.

A - Monomorphic ventricular tachycardia, the most commonly seen.
B - Torsades de pointes (literally, “twisting of the points”).

Fig. 8 Electrocardiogram of ventricular fibrillation. Wide, bizarre, irregularly occurring wave forms of various amplitudes

VT and VF are uncommon in children. When seen, consider congenital heart disease, cardiomyopathies, or acute inflammatory injury to the heart (eg, myocarditis). Other causes are drug toxicity (eg, recreational drugs, tricyclic antidepressants, digoxin overdose, or toxicity, cisapride with macrolide antibiotics combination), metabolic causes (eg, hyperkalemia, hypermagnesemia, hypocalcemia, or hypoglycemia), or hypothermia.
Management of VT and VF

- Assess ABC and administer high flow oxygen (x)
- Attach 12 lead ECG monitor/defibrillator (x)
- Determine the origin of the tachycardia based on analysis of the 12-lead ECG and on a carefully obtained history, including family history for ventricular arrhythmias or sudden deaths (x)
- Identify whether the child has pulse and in shock or not in shock and manage accordingly (x)

The treatment goal is to keep the heart rate at < 150 BPM in infants and less than 130 BPM in older children

(A) Treatment of child with stable VT/VF
Those who have VT/VF with palpable distal pulses are considered to have stable VT/VF

1. Pharmacological agents are the first line of treatment

- Careful evaluation and early consultation with a cardiologist before any therapy is given.
- Amiodarone (5 mg/kg over 60 minutes) should be administered. (x)
- Procainamide (15 mg/kg over 30 to 60 minutes) or lidocaine (1 mg/kg over approximately 2 to 4 minutes) may be considered as alternative agents.
- A cautious approach is appropriate as amiodarone and procainamide can cause hypotension, and procainamide is a potent negative inotrope.
- Close haemodynamic and ECG monitoring are required during and after the infusion of either agent. (x)
- Amiodarone and procainamide generally should not be administered together because both prolong the QT interval.
(B) Treatment of child with unstable VT/VF with pulses
Those who have VT/VF with palpable distal pulses in shock are considered in this group.

1. Immediate synchronized cardioversion is the treatment of choice
   - Pretreat conscious patients with light sedation eg. midazolam 0.1mg/kg before cardioversion. (x)
   - If the child is appropriately responsive and not in distress, there is often time to have IV access and opinion from a cardiologist.
   - It is important to consider drug or metabolic causes of the VT, especially in a child without a known predisposing cause for the arrhythmia.
   - The rhythm should be examined for a torsades de pointes appearance. If torsades de pointes is suspected, administer 25-50mg/kg up to 2g of magnesium sulphate by a rapid intravenous bolus over 10 to 20 minutes. (y)

2. Pharmacologic interventions include
   - Amiodarone 5mg/kg/IV over 30 min, procainamide 15mg/IV over 30-60 min or lidocaine 1mg/kg IV bolus which can be repeated every 5-10 min.
   - After cardioversion as return to normal rhythm is transient continue the medication used to achieve sinus rhythm as an infusion. Lidocaine (20-50µg/kg/min), amiodarone (7-15mg/kg/day) or procainamide (20-80µg/kg/min)

(C) Treatment of a child with pulseless VT/VF

1. Immediate defibrillation is the definitive therapy for pulseless VT and VF
   - Ventilation, oxygenation, and chest compressions should be delivered and vascular access may be attempted until the defibrillation is commenced, but these interventions should not delay shocks. (x)
• If the patient fails to defibrillate after 3 shocks, administer intravenous epinephrine in a dose of 0.01 mg/kg (or 0.1 mg/kg for the tracheal route) and attempt defibrillation again within 30 to 60 seconds. (x)

• If VF or pulseless VT continues after this epinephrine dose plus shock(s) or if pulseless VF/VT recurs, amiodarone (5 mg/kg by rapid intravenous bolus) may be used followed by another defibrillation attempt within 30 to 60 seconds after closed-chest compression to deliver the drug to its site of action.

• It is recommended that not more than 30 to 60 seconds of chest compressions should be given before the next shock.

Note that the pattern of treatment after the initial 3 shocks is “CPR-drug-shock, CPR-drug-shock”.


Amiodarone.
Loading infusion of 5 mg/kg is recommended over several minutes to 1 hour, depending on the need to achieve a rapid drug effect. Repeated doses of 5 mg/kg up to a maximum of 15 mg/kg per day may be used as needed. The main acute side effect from intravenous administration is hypotension. (x)

Lidocaine.
Lidocaine may be considered in children with shock-resistant VF or pulseless VT.

The recommended dose is 1 mg/kg by rapid intravenous injection followed by an infusion; Infusions are given at a rate of 20 to 50 µg/kg/minute. If there is more than a 15-minute delay between the bolus dose and start of an infusion, a second bolus dose of 0.5 to 1 mg/kg lidocaine may be given to rapidly restore therapeutic concentrations. Therapeutic concentrations raise the VF threshold and therefore may protect against re-fibrillation after successful defibrillation. If reduced lidocaine clearance is expected or suspected, the infusion rate generally should not exceed 20 µg/kg/minute.
**Procainamide**

Procainamide is effective in the treatment of atrial fibrillation, flutter, and SVT and it may be useful in the treatment of postoperative junctional ectopic tachycardia. It also has been used to treat or suppress VT.

Procainamide must be given by a slow infusion to avoid toxicity from heart block, myocardial depression, and prolongation of the QT interval (which predisposes to torsades de pointes tachycardia),

Procainamide is not indicated in the treatment of VF or pulseless VT.

In children with VT with pulse, procainamide may be considered. Infuse the loading dose of 15 mg/kg over 30 to 60 minutes with continuous monitoring of the ECG and frequent blood pressure monitoring. If the QRS widens to >50% of baseline or hypotension occurs, stop the infusion.

Since procainamide increases the likelihood of polymorphous VT developing, it generally should not be used in combination with another agent that prolongs the QT interval, such as amiodarone.

**Epinephrine and vasopressin.**

A vasoconstrictor regimen may be considered in shock-resistant VT/VF, since if systemic vasoconstriction is inadequate with routine therapy, coronary perfusion is limited and the myocardium is unlikely to respond to shocks. For these reasons high-dose epinephrine (0.1 to 0.2 mg/kg) may be considered in shock-resistant VF/pulseless VT.

**Key Points**

**Management of Ventricular Tachycardia**

- Pharmacological agents are the first line of treatment in a stable child with palpable pulse without shock
- Immediate synchronized cardioversion is the treatment of choice in an unstable child with palpable pulses with shock
- Immediate defibrillation is the definitive therapy for pulseless VT and VF.
Atrial flutter

Fig. 9  Electrocardiogram of atrial flutter. Regular P waves with less than 1-to-1 atrioventricular conduction.

Is an uncommon rhythm seen in paediatric population. Atrial rates may present in the range of 240-450 BPM with the ventricular response depending on the AV nodal conduction. Mostly seen in children with structural heart disease and notably seen as a complication of cardiac surgery.

Management of atrial flutter

- Identify whether the child is haemodynamically stable
- If unstable, electrical cardioversion is indicated and consider adding heparin to prevent embolization.

If patient is already on digoxin avoid cardioversion unless the condition is life threatening as this combination is associated with malignant ventricular arrhythmias. Alternatives for patients who are receiving digoxin are rapid atrial pacing.

- If haemodynamically stable, digoxin is administered. Oral propranolol 1-4mg/kg/day in 3–4 times a day in divided doses may also be added.
- Recurrences are prevented by administering quinidine.
Atrial fibrillation

Fig. 10 Electrocardiogram of atrial fibrillation. Wave isoelectric line reflects the irregular and rapid atrial activity. Typically, the ventricular rate is “irregularly irregular”.

It is rare in children. Defined as disorganized rapid atrial activity with rates ranging from 350-600 bpm. Children at an increasing risk of developing atrial fibrillation include those with structural heart disease and those who have undergone intra-atrial surgery.

Management of atrial fibrillation

- If haemodynamically unstable consider cardioversion immediately.
- If haemodynamically stable administer digoxin. If no response is seen in 24 hours a second medication such as Propranolol is added.
Tachycardia Algorithm

No pulse

Vaso/IV/Trach

No pulse

Defibrillation
28kg ⇒ 2-4J/kg ⇒ 48kg

Epinephrine
0.01IU/kg/1000

STABLE
Adenosine IV/IO 0.1 mg/kg (max 6mg)
Consider Alternative Medication
Amiodarone 5mg/kg IV over 20-60min

UNSTABLE
Cardioversion
0.3-1J/kg

UNSTABLE
Cardioversion
0.3-1J/kg

Pulse

SVT

Stable Tachycardia

VT/VTx

Defibrillation
48kg

Epinephrine
1/1000 ⇒ 5/1000 ⇒ 25/1000

Stable
Adenosine IV/IO 0.2 mg/kg (max 12mg)
Defibrillation is the untimed (asynchronous) depolarization of the myocardium that successfully terminates VF or pulseless VT.

Cardioversion is the timed (synchronous) depolarization of myocardial cells that successfully restores a stable rhythm. It is used to treat the symptomatic patient with SVT or VT (with pulses) accompanied by poor perfusion, hypotension, or heart failure. It also may be used electively in children with stable VT or SVT at the direction of an appropriate cardiology specialist.

In order to achieve the optimum outcome, defibrillation must be performed quickly and efficiently. This requires the following:

- Correct paddle position
- Correct paddle placement
- Good paddle contact
- Correct energy selection

**Correct paddle selection**

Most defibrillators are supplied with adult paddles attached (13-cm diameter, or equivalent area). Paddles of 4.5-cm diameter are suitable for use in infants, and ones of 8-cm diameter should be used for small children.

**Correct paddle placement (Fig. 11 & 12)**

The usual placement is antero-lateral. One paddle is put over the apex in the mid axillary line and the other is placed just to the right of the sternum, immediately below the clavicle.
If the antero-posterior placement is used, one paddle is placed just to the left side of the lower part of the sternum and the other just below the tip of the left scapula.

Fig. 12  Antero-posterior paddle placement

- **Good paddle contact**
  Gel pads or electrode gel should always be used (if the latter, care should be taken not to join the two areas of application). Firm pressure should be applied to the paddles.

- **Correct energy selection**
  Refer text for the recommended levels

- **Safety**
  A defibrillator delivers enough current to cause cardiac arrest. The user must ensure that other rescuers are not in physical contact with the patient (or the trolley) at the moment the shock is delivered. The defibrillator should only be charged when the paddles are either in contact with the child or properly in their storage positions.

  Disconnect the oxygen supply to the patient.
### Procedure

**Basic life support should be interrupted for the shortest possible time (5-9 below)**

1. Apply gel pads or electrode gel
2. Select the correct paddle
3. Select the energy required
4. Place the paddles on the gel pads, and apply firm pressure
5. Press the charge button
6. Wait until the defibrillator is charged
7. Shout “Stand back!”
8. Check that all other rescuers are clear
9. Deliver the shock
GUIDELINES FOR OPTIMAL MANAGEMENT OF SHOCK IN CHILDREN

Shock can be defined as “acute circulatory failure with inadequate or inappropriately distributed tissue perfusion resulting in generalized cellular hypoxia.” It is a life threatening medical emergency.

Key Point
Failure of successful therapy is very often as a result of too little being done, too late by a too junior officer.

1. Causes of shock

- Cardiogenic: “pump failure” (acute myocardial infarction).
- Obstructive: mechanical impediment to forward flow (pulmonary embolus and cardiac tamponade).
- Hypovolaemic: loss of circulating volume (gastroenteritis, haemorrhage, burns and intestinal obstruction).
- Distributive: abnormalities of the peripheral circulation (sepsis and anaphylaxis).
- Dissociative: inadequate oxygen releasing capacity (profound anaemia, methaemoglobinemia and carbon monoxide poisoning).

These factors are often combined. For example, in sepsis and anaphylaxis, vascular dilatation and sequestration in venous capacitance vessels lead to relative hypovolaemia, which is compounded by true hypovolaemia due to fluid losses through increased microvascular permeability.

In all forms of shock there may be activation of the coagulation pathway, with the development of disseminated intravascular coagulation (DIC). The disseminated inflammatory response and microcirculatory changes may lead to progressive organ failure (multiple organ dysfunction syndrome MODS), also known as multiple organ failure (MOF)); the lungs are usually affected first, with the development of the acute respiratory distress syndrome (ARDS). The mortality in MODS is high and treatment is supportive.
The history will often indicate the cause of shock.

- A history of vomiting and/or diarrhoea points to fluid loss.
- The presence of fever and/or rash points to septicaemia or dengue shock.
- The presence of urticaria and angio-neurotic oedema and a history of allergen exposure points to anaphylaxis.
- A history of major trauma points to blood loss, cardiac tamponade or tension pneumothorax.
- A history of polyuria and a very high blood sugar points to diabetes.
- The presence of tachycardia and an abnormal rhythm points to an arrhythmia or a cardiogenic shock.

1.1 Septic shock

- Patients in septic shock are likely to be febrile.
- May have a focus of infection.
- May have relative intravascular volume depletion despite a lack of history of inadequate intake or volume loss.
- Cardiac function may be depressed, worsening potential cardiovascular compensation.
- There may be a mal-distribution of blood flow, such that normal autoregulatory mechanisms to preserve perfusion to vital organ vascular beds are ineffective.
- In the early phases of sepsis, the skin may appear very briskly perfused with warm extremities, bounding pulses, and brisk capillary refill (warm shock).
- As shock progresses and perfusion worsens, distal pulses are diminished, the skin appears cool, and capillary refill is prolonged (cold shock).
- Systemic inflammatory response syndrome (SIRS) is often associated with sepsis.
1.2 Cardiogenic shock
Cardiogenic shock is relatively rare in the general pediatric population.

Findings in cardiogenic shock:
- Tachycardia
- Hepatomegaly
- Cardiac gallop
- Cardiac murmurs
- Precordial heave
- Cardiomegaly on chest radiography
- Cardiac hypertrophy on echocardiography
- Jugular venous distention
- ECG abnormalities

1.3 Anaphylactic shock
- Profound vasodilatation leads to warm peripheries and low blood pressure.
- Erythema, Urticaria, angio-oedema, bronchospasm, and oedema of the face and larynx may be present.

2. Compensated, decompensated and Irreversible shock
- Compensated shock - perfusion is poor while the systolic blood pressure remains normal. Will progress to decompensated shock, if not treated.
- Decompensated shock - When perfusion is poor and the blood pressure below normal.
- Irreversible shock - When there is additional organ damage in a child who has not been aggressively resuscitated. May progress to and death.
3. Assessment of a child in shock

<table>
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<th>Primary assessment</th>
<th>Assessment of tissue perfusion</th>
<th>Effects of circulatory inadequacy on other organs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assess the adequacy of</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Airway</td>
<td>Tachycardic initially</td>
<td>Reduced urine output &lt; 1ml/kg/hr in children</td>
</tr>
<tr>
<td>Breathing</td>
<td>Bradycardic terminally</td>
<td>&lt; 2ml/kg/hr in infants</td>
</tr>
<tr>
<td>Circulation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Poor pulse volume:</td>
<td>Acidotic sighing breathing</td>
</tr>
<tr>
<td></td>
<td>Centrally and peripherally</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Capillary refill &gt;2 sec when blanching pressure is applied for 5 sec on sternum/ digit</td>
<td>Pale, cyanosed or cold skin</td>
</tr>
<tr>
<td></td>
<td>Normal blood pressure initially</td>
<td>Agitated and confused due to brain hypoperfusion</td>
</tr>
<tr>
<td></td>
<td>Hypotension terminally</td>
<td></td>
</tr>
</tbody>
</table>

Symptoms and Signs of Shock

- Cold mottled skin
- Pale or cyanosed
- Tachypnoea with acidotic sighing breathing
- Tachycardia with poor pulse volume
- Agitated, confused and decreasing conscious state
- Capillary refill over 2 seconds
- Peripheral to core temperature gradient >2°C
- Poor urine output <0.5ml/kg/hr
- Hypotension is a late manifestation

4. Management of Shock

Regardless of the cause, the ABC must be stabilized immediately.

Do not delay this initial stabilization for further workup and imaging studies.
4.1 Airway

The patient’s airway must be patent.

4.2 Breathing

- Oxygenate adequately with high flow oxygen
- If in respiratory distress, consider intubation and ventilation.
- Place the patient on a pulse oximeter and cardio-respiratory monitor.

Stabilizing the airway and providing mechanical ventilation may facilitate elimination of carbon dioxide, helping to compensate the coexistent metabolic acidosis.

4.3 Circulation

- Immediately improve circulation for systemic oxygen delivery, once the airway and ventilation are stabilized.
- Circulatory improvement is achieved via volume expansion and, if necessary, with Pharmacologic therapy.

4.3.1 Volume expansion

The major physiologic abnormality in most forms of pediatric shock is either an absolute or a relative intravascular hypovolemia. Dehydration, hemorrhage, sepsis, and other distributive etiologies all cause intravascular hypovolemia with a reduction in cardiac ventricular filling volume (preload).

Children with hypovolemic shock who receive appropriate aggressive fluid replacement within the first hour of resuscitation have the most optimal chance of survival and recovery. Unlike adults, children do not have an apparent increase in fluid-related complications such as pulmonary oedema. Therefore, the therapy of choice is rapid and aggressive fluid replacement. Children, who receive appropriate yet aggressive fluid replacement early, have the best chance of surviving severe septic or hypovolaemic shock.

- Place 2 large-bore free-flowing IV/ intraosseous needle.
- Administer 20 mL/kg of an isotonic crystalloid infusion (0.9% isotonic sodium chloride or Hartmanen solution) over 5 minutes or less.
- Re-evaluate and administer additional 20 mL/kg infusions of isotonic crystalloid or colloid if signs of poor perfusion exist.
- Severe hypovolemia or sepsis may require more than 60 mL/kg of volume in the first hour of resuscitation, often within the first 15 minutes.
Cardiogenic shock - As myocardial failure is the root cause of poor cardiac output cardiotropic medications are indicated after an initial 20 mL/kg of crystalloid solution.

Anaphylaxis - IM adrenaline should be administered after initial 20ml/kg of colloids.

Tachyarrythmia - Up to 03 synchronous electrical shocks at 0.5, 0.5 and 1 Joule should be given. If IV access is already available for SVT, IV/intraosseous adenosine is preferred to synchronous electrical shock.

4.3.2 Pharmacologic Therapy for maintenance of circulation

- Myocardial contractility is impaired in cardiogenic shock and at a later stage in other forms of shock as a result of hypoxaemia, acidosis and the release of mediators.
- Drugs that impair cardiac performance, e.g. _-blockers, should be stopped.
- When a patient remains hypotensive despite adequate volume replacement inotropic agents are administered.
- This must be via a large central vein and the effects carefully monitored.
- Many consider dopamine to be the inotrope of choice in critically ill patients, but dobutamine is a better choice when vasoconstriction caused by dopamine could be dangerous.
- Norepinephrine (noradrenaline) in combination with dobutamine (depending on the cardiac output) is used for shocked patients with a low peripheral resistance, e.g. septic patients.
Most infusions may be calculated on the basis of the “Rule of 6” as illustrated in the table. Alternatively, a standard concentration may be used to provide more dilute or more concentrated drug solution, but then an individual dose must be calculated for each patient and each infusion rate as follows: Infusion rate (mL/h) = [weight (kg) x dose (µg/kg per minute) x 60 min/h]/concentration (µg/mL). Diluent may be 5% dextrose in water, 5% dextrose in half-normal saline, normal saline, or Ringer lactate unless noted otherwise.

**Dopamine**

- **Initial dose** - 2 to 5 µg/kg/minute (infused through a secure intravenous line). Increased to 10 to 20 µg/kg/min in an effort to improve blood pressure, perfusion, and urine output.
- **Infusion rates exceeding 20 µg/kg/ min** may result in excessive vasoconstriction and a loss of renal vasodilating effects.
- **Hence, if further inotropic support is needed**, either epinephrine or dobutamine may be added
- **Dopamine infusions may produce tachycardia, vasoconstriction, and ventricular ectopics.**
- **Infiltration of dopamine into tissues can produce local tissue necrosis.**
- **Dopamine and other catecholamines are partially inactivated in alkaline solutions and therefore should not be mixed with sodium bicarbonate.**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Comments</th>
<th>Dose range</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>Low dose (1-3) Moderate (3-10) High dose (&gt;10)</td>
<td>At low dose general vasoconstriction which may increase urine output and preserve function of vital organs. Increases cardiac output at all doses, but at high doses this beneficial effect may be offset by vasoconstriction, thus increasing afterload and ventricular filling pressure.</td>
<td>IV/IO infusion: 2-20 µg/kg per minute; 6x body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 1 µg/kg per minute</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Dopamine analogue. Most useful in patients with a low cardiac output and peripheral vasoconstriction. E.g. Cardiogenic shock</td>
<td>IV/IO infusion: 2-20 µg/kg per minute; 6x body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 1 µg/kg per minute</td>
<td></td>
</tr>
<tr>
<td>Epinephrine (Adrenaline)</td>
<td>Low dose (0.06-0.1) Moderate dose (0.1-0.18) High dose (&gt;0.18)</td>
<td>Not responding to dobutamine or dopamine. At high doses Vasodilation of coronary arteries leads to decreased cardiac output, oliguria and peripheral gangrene. Agent of choice in septic shock when hemodynamic monitoring not available.</td>
<td>IV/IO infusion: 0.1-1.0 µg/kg per minute; 0.6x body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 0.1 µg/kg per minute</td>
</tr>
<tr>
<td>Norepinephrine (noradrenaline)</td>
<td>Particularly useful in septic shock as administration leads to increased inotropy and an increase in peripheral vascular resistance. Requires full hemodynamic monitoring</td>
<td>IV/IO infusion: 0.1-2.0 µg/kg per minute; 0.6x body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 0.1 µg/kg per minute</td>
<td></td>
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</table>
**Dobutamine**

- An inotrope with primarily beta_1_-agonist effects, that increases cardiac contractility. Provides relatively weak beta_2_-mediated peripheral vasodilation that might reduce systemic vascular resistance and afterload and improve tissue perfusion.
- Dobutamine is most appropriate in cardiogenic shock
- For fluid refractory shock, dobutamine is given along with dopamine.
- Dobutamine is less likely to precipitate ventricular dysrhythmias than epinephrine.
- Initial dose is 5 mcg/kg/min IV and gradually increased to 20 mcg/kg/min IV.

**Epinephrine (Adrenalin)**

- Recommended for fluid refractory dopamine resistant shock. (septic/anaphylactic)
- Stimulates both alpha- and beta-receptors improving myocardial contractility and peripheral circulation.
- May be preferable to dopamine in patients with marked circulatory instability, particularly in infants.
- Initial dose is 0.1 to 0.3 µg/kg/min (infuse into a secure intravenous line) and titrated up to 1 µg/kg/min based on the observed hemodynamic effects.
- Severe cases, the dose may be 2-3 mcg/kg/min IV or even higher.
- Known complications are atrial/ventricular tachyarrhythmias, severe hypertension, hyperglycaemia, metabolic acidosis and hypokalaemia.

**Norepinephrine**

- Also recommended for fluid refractory dopamine resistant shock. (septic/anaphylactic)
- Norepinephrine is predominantly an alpha-agonist that results in increased peripheral vasoconstriction and thus increased peripheral vascular resistance.
- Initial dose is 0.1mcg/kg/min IV (secure vascular line, preferably central) and increased according to effect and adverse effects.
- Toxic effects are hypertension, organ ischemia (including distal extremity vascular beds), and arrhythmias.
4.4 Antibiotics
- Unless an alternative diagnosis is very clear, an antibiotic is given immediately after blood culture is taken as sepsis is one of the commonest causes of shock in paediatric practice.
- Empiric coverage vary depending on the age of the patient and previous antibiotic exposure.
- Neonates are often begun on a combination of ampicillin and gentamicin.
- Older infants and children are covered with a third-generation cephalosporin, possibly along with expanded Gram-positive organism coverage with vancomycin.
- Contact microbiologist for opinion.

4.5 Dextrose
- Perform a glucose test on all patients in shock.
- Neonates and infants are at a risk of hypoglycaemia during shock as glycogen stores are limited.
- Alternatively, high levels of catecholamines may result in hyperglycaemia.
- If the glucose level is low, 5-10ml/kg of 10% Dextrose infusion is given.

4.6 Sodium bicarbonate
- Use of sodium bicarbonate is controversial.
- Acidosis impairs myocardial contractility and catecholamines function.
- Treatment with bicarbonate may worsen intracellular acidosis while it corrects serum acidosis.
- Bicarbonate administration may result in hypernatraemia and hyperosmolality.
- For persistent shock or ongoing loss, careful replacement of bicarbonate may be indicated. (eg.severe diarrhea)
- Dose of bicarbonate $\text{HCO}_3^-$ (mEq) = Base deficit X patient’s weight (in kg) X 0.30.
- Half the bicarbonate deficit may be administered initially and repeat acid-base status determined.
- Alternatively, 0.5-1 mEq/kg/dose IV infused over 1-2 minutes.
Acidosis should ideally be corrected with increased perfusion from volume supplementation and judicious use of cardiotropic medications together with optimal ventilation.

4.7 Calcium

- Serum ionized calcium could be low despite normal total calcium.
- Blood products which contain citrate may decrease ionized calcium.
- Calcium therapy may be useful in a patient with hypocalcemia.
- Also indicated for shock caused by arrhythmias secondary to hyperkalemia, hypermagnesemia, or calcium channel blocker toxicity.
- Calcium chloride produces higher and more consistent levels and, is recommended for acute resuscitation.
- Dose is 10-20 mg/kg (0.1-0.2 mL/kg of calcium chloride 10%) IV infusion at a rate not exceeding 100 mg/min.
- May be repeated guided by ionized calcium.

4.8 Corticosteroids

- Place of corticosteroids is controversial.
- Cortisol level may be done prior to first dose of corticosteroids; replacement is beneficial if the random level is low.
- Initiation of stress-dose hydrocortisone, in the range of 1-2mg/kg/IV every 6 hours may be beneficial and lifesaving.

4.9 Other therapies

- Underlying etiology should be identified and treated.
- If the cause is sepsis, isolate and treat the infectious organism with appropriate antibiotics.
- If the cause is trauma, ongoing bleeding needs surgical opinion.
- Convert malignant arrhythmias to normal sinus rhythm as soon as possible.
- Optimize nutritional support.
- Organ support may be required, including mechanical ventilation, renal dialysis, or even extracorporeal circulatory support.
5 Additional workup

- While stabilization of the airway and breathing and an aggressive expansion of circulation takes precedence over any other workup. Additional investigations may ultimately help identify an etiology and guide ultimate therapy.

5.1 Full blood cell count

- Haemoglobin determines the amount of oxygen carrying capacity.
- Transfuse an anemic patient as soon as possible.
- An elevated or depressed white cell count, along with a white cell differential, supports the diagnosis of septic shock.
- Thrombocytopenia supports a bleeding disorder that could result in internal hemorrhage or diffuse intravascular coagulation

5.2 Biochemical evaluation

- Hypernatraemia suggests intravascular volume contraction seen in hypovolaemia.
- Hypovolaemia may result in an elevated blood urea nitrogen and creatine.
- Hypoxic/ischaemic damage to other organs such as the liver, results in elevated liver enzymes.

5.3 Chest radiography

- Evaluation of the cardiac silhouette may help delineate cardiogenic shock from hypovolaemic shock in which the heart size appears small.
- Look for changes of ARDS and focus of sepsis.

5.4 Blood gas

- Determine the PaO₂, to assist in titration of supplemental oxygen and acid-base status to monitor the degree of shock and the response to therapy.
- A decreased serum carbon dioxide suggests a metabolic acidosis.
6 Monitoring in ICU

While taking into account the overall clinical appearance, acid-base status, arterial oxygen tension and venous oxygen saturation, central venous pressure (CVP) and/or pulmonary capillary wedge pressure, and CO or cardiac index should be monitored.

6.1 Acid-base status

- Metabolic acidosis is either due to lactic acid production (shock) or direct bicarbonate loss (diarrhoea).
- Serum lactate level to identify the etiology.

6.2 Central venous pressure and pulmonary capillary wedge pressure

- Low central venous pressure (CVP) or pulmonary capillary wedge pressure (PCWP) reflects inadequate intravascular volume.
- A normal CVP in a normal compliant heart is 1-3 cm H$_2$O.
- Pressures higher than 10 cm H$_2$O reflects volume overload.
- Volume administration is generally thought to be maximal at PCWP measurements of 12-18 cm H$_2$O in patients with adequate left-sided heart function.

6.3 Mixed venous oxygen saturation

- A blood sample from the right atrium through a central venous catheter or blood from a pulmonary oxygen catheter (Swan-Ganz catheter) sampled from the port placed in the right atrium is mixed venous blood returning to the heart.
- By comparing the mixed venous oxygen saturation (SvO$_2$) with the SaO$_2$, a determination of the arteriovenous oxygen saturation difference can be noted.
- In a patient with a relatively normal SaO$_2$ (90-100%), the normal SvO$_2$ is 70-80%. The tissues typically extract 28-33% of oxygen delivered to them.
- If the oxygen extraction difference is greater than 33%, perfusion to the tissue capillary beds may be inadequate, reflecting a state of shock.
- Alternatively, if the oxygen extraction difference is less than 25%, oxygenated blood may be shunting past tissue capillary beds as a result of inappropriate distribution of blood flow (ie, distributive shock with arterio-venous shunts resulting from vasodilation).
6.4 Cardiac index

- A pulmonary artery catheter may be useful in determining a measurement of cardiac output.
- The CO divided by body surface area yields the CI.
- Normal CI is 3.5-5.5 L/min/m².
- Monitoring changes in CI together with changes in intravascular volume administration or cardiotropic infusions may help guide and optimize administration of these therapies.

Key points

- Early and rapid recognition is crucial
- Treatment must be instituted early, before patients have developed irreversible peripheral vascular failure.
- Aggressive and adequate volume replacement is essential in all cases.
- Mean arterial pressure should be maintained at adequate levels
- Circulatory support should aim to achieve normal haemodynamics and restore tissue perfusions.
- For patients with continued impaired tissue oxygenation, inotropes and vasoconstrictors are used to further increase tissue perfusion.

Management of anaphylactic shock

- Anaphylaxis is a potentially life-threatening condition, which may progress to shock, but in most cases rash is the only symptom.

- The most common causes are drug and food allergy.

- Prodomal symptoms of flushing, itching, facial swelling, urticaria, abdominal pain, diarrhea, wheeze and stridor may precede shock or may be the only manifestation of anaphylaxis.

- Anaphylaxis can be life-threatening because of the rapid onset of airway compromise due to laryngeal oedema, breathing difficulty due to severe broncho-constriction and / or development of shock.
Management

- Manage ABC.
- If airway obstruction with stridor - call for ENT and Anaesthetic help.
- Remove the precipitating cause.
- High concentration inhaled oxygen.
- Colloid, e.g. Starch solution/ Haemaccel or crystalloid eg. N. saline (0.9%)/ Hartmann solution 20ml/kg IV/IO as a rapid infusion and continue depending on the response.
- Administer 10micrograms/kg adrenaline IM =0.1mL/kg of a 1 in 10,000 solution.
- Administer further doses of IM epinephrine every 05 mints until improvement occurs.
- If no response, administer intravenous adrenaline 10 micrograms/kg over several minutes with full ECG monitoring till response is seen.
- Chlorpheniramine IV over 1–2 min (Table 2 for dosage) and Hydrocortisone 04 mg/kg IV are given though there is no proven benefit.
- Admit patient to hospital for 24 hours for monitoring and treatment even after full recovery as there is a risk of relapse.
- Patients who have had an attack of anaphylaxis and who are at risk should carry a preloaded syringe of epinephrine for subcutaneous self-administration.
- Should wear an appropriate information bracelet.

Table 2

<table>
<thead>
<tr>
<th>Drug Dosage in Anaphylaxis</th>
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<tbody>
<tr>
<td><strong>Adrenaline</strong></td>
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<td><strong>Chlorpheniramine</strong></td>
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<td><strong>Hydrocortisone</strong></td>
</tr>
</tbody>
</table>
Anaphylactic Shock

- Assess ABC
- High flow O₂
- Remove precipitating cause

Shock

No stridor

- Colloid / Crystalloid 20ml/kg IV/IO

- Adrenaline IM 0.1 ml/Kg 1:10,000

- Reassess ABC

If no improvement

- Repeat Adrenaline IM 0.1ml/Kg 1:10,000
- Adrenaline every 5 min.

- Chlorpheniramine IV
- Hydrocortisone - 4mg/kg IV

- Admit & observe for 24 hrs

- Parent education
- Information leaflet /bracelet

Stridor

- Call ENT/ Anaesthetist

No stridor

- Call ENT/ Anaesthetist

Shock

Not in shock

No stridor

- Colloid / Crystalloid 20ml/kg IV/IO

- Adrenaline IM 0.1 ml/Kg 1:10,000

- Reassess ABC

If no response with hypotension & severe dyspnoea

- IV adrenaline 1:10,000 10mcg/kg over several minutes till response is seen.

>12yrs 10-20mg
6-12yrs 5-10mg
1-5yrs 2.5-5mg
1mo-1yr 0.25mg/kg
Neonates do not use

Stridor

- Call ENT/ Anaesthetist

No stridor

- Call ENT/ Anaesthetist

Shock

Not in shock

No stridor

- Colloid / Crystalloid 20ml/kg IV/IO

- Adrenaline IM 0.1 ml/Kg 1:10,000

- Reassess ABC

If no improvement

- Repeat Adrenaline IM 0.1ml/Kg 1:10,000
- Adrenaline every 5 min.

- Chlorpheniramine IV
- Hydrocortisone - 4mg/kg IV

- Admit & observe for 24 hrs

- Parent education
- Information leaflet /bracelet
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