SECTION 1

# MEDICAL EMERGENCIES

CHAPTER 1

# Recognising the Critically Ill Child

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## Introduction

Children are often unable or unwilling to verbalise complaints. In addition, symptoms and signs of sepsis or cardiopulmonary compromise are often vague and subtle in children.

The ability to assess and recognise an ill child early allows for timely interventions and therapy, such as respiratory support, fluid resuscitation or early antibiotics to reverse potentially life-threatening cardiopulmonary instability.

## Anatomic and Physiological Considerations

Respiratory arrest is the most common cause of cardiopulmonary collapse in children. This is largely because the paediatric respiratory system is ill-designed to cope with an increased work of breathing. The reasons are multi-factorial and include a relatively large tongue and floppy epiglottis, small airways with increased airway resistance, and increased chest wall compliance due to a cartilaginous chest wall. Respiratory failure is characterised by inadequate ventilation, insufficient oxygenation or both.

Cardiac output is a product of stroke volume and heart rate, and blood pressure is a function of cardiac output and systemic vascular resistance.

Circulatory shock is defined as the failure of the circulatory system to provide oxygen and nutrients to meet tissue metabolic demands. Shock can be classified into compensated, uncompensated or irreversible.

Compensatory mechanisms include tachycardia and increased systemic vascular resistance in an effort to maintain cardiac output and perfusion pressure (blood pressure), respectively. Decompensation occurs when these mechanisms fail and result in end-organ hypoperfusion and hypotension. Untreated, shock states can rapidly deteriorate into failure of multiple organ systems and lead to irreversible shock and death.

The pathophysiology of shock can be divided into distributive, hypovolaemic/haemorrhagic, cardiogenic or obstructive. It is not unusual for one patient to have a number of different pathophysiological patterns of shock, which can evolve depending on the time-course of their disease.

Recognition of pre-shock states is important so that early goal-directed therapy can be instituted. The regimen of resuscitation includes fluid boluses, airway intervention and inotropic support. The key is early shock recognition and prompt action.

## History

Functional status of the child is a simple but effective measure of how ill the child is. Questions to ask include:

- Level of activity/play
- Conscious level/irritability
- Feeding/fluid intake
- Urine output

Red flags in the history include:

- High-pitched cry/inconsolable crying
- Grunting
- Cyanosis
- Apnoeic episodes
- Pallor, cool and clammy peripheries
- Shortness of breath or dyspnoea
- Acute change in mentation
- Focal seizures
- Bloody stool in a neonate

There should be a high index of suspicion in the very young or if there is a significant medical history, such as:

- Maternal history of Group B Streptococcus (GBS) infection (for neonates)
- Congenital cardiac defects
- Primary immunodeficiency syndromes
- Chronic steroid usage
- Haematological-oncological disorders on active chemotherapy
- History of adreno-cortical deficiency, e.g. hypopituitarism, congenital adrenal hyperplasia, hypothalamic or pituitary lesions

## Vital Parameters

Hypotension is defined as systolic BP:

- <60 mmHg in a neonate
- <70 mmHg in infants (1–12 months)
- <70 mmHg + [2 x (Age in Years)] in children 1–10 years
- <90 mmHg in children older than 10 years

Mean arterial pressure can be calculated as {50 + [2 x (Age in Years)} mmHg.

There is significant inter-individual variability of pulse pressure (systolic minus diastolic BP), increasing with age and generally ranging between 20 and 40 mmHg. A widened pulse pressure is present in distributive shock. A narrow pulse pressure may suggest hypovolaemic or cardiogenic shock.

Unexplained tachycardia may be one of the first signs of compensated shock.

**Table 1.1 Table of Normal Heart Rate, Respiratory Rate and Systolic Blood Pressure (BP) by Age**

| Age | Heart Rate (beats/min) | Respiratory Rate (breaths/min) | Systolic Blood Pressure (mmHg) |
|---|---|---|---|
| Neonate | 120–180 | 40–60 | 60–80 |
| Infant (1 mth to 1 yr) | 110–160 | 30–40 | 70–90 |
| Toddler (1–2 yr) | 100–150 | 25–35 | 80–95 |
| Young child (2–7 yr) | 95–140 | 25–30 | 90–110 |
| Older child (7–12 yr) | 80–120 | 20–25 | 100–120 |

## Physical Examination

Clinical states which can rapidly progress and are life-threatening include:

- Severe respiratory distress
- Cardiovascular instability/cardiogenic shock
- Sepsis/septic shock
- Severe dehydration
- Seizures/altered mental state
- Trauma
- Any organ failure, such as renal/hepatic failure

Learn to integrate the signs of an unwell child as no single sign is definitive. If there are concerning features in the history and/or physical examination, consider the following:

- Admit for observation or transport patient to an appropriate care facility after initial stabilisation.
- Call for senior help if there is evidence of severe respiratory distress, poor perfusion and/or hypotension, obtundation/change in mentation, prolonged seizure or cardiac arrhythmias.
- Activate the paediatric Emergency Code Team if there is imminent cardiopulmonary arrest, severe respiratory compromise needing intubation, or if there is difficulty stabilising the patient, e.g. difficult venous access, no response to therapeutic interventions, potential difficult airway.

## Investigations

- Blood glucose: Exclude hypoglycaemia or diabetic ketoacidosis (DKA) as a cause for obtundation
- Blood gas analysis: Evaluate for metabolic or respiratory acidosis, sodium/potassium/calcium derangements
- Full blood count, renal panel and group and crossmatch
- Imaging: Chest X-ray (CXR) to exclude pulmonary pathology, cardiomegaly. Computed tomography (CT) of the head if there are concerns regarding intracranial pathology.

**Table 1.2 Care of the Critically Ill Child (adapted from RCH, 2021)**

| Assessment | Clinical Features | Management |
|---|---|---|
| **Airway** | | |
| Patency | If compromised:<br>• Abnormal breath sounds (stridor, stertor, snoring, gurgling, voice hoarseness)<br>• Drooling<br>• Tracheal tug<br>• Dyspnoea<br>• Respiratory distress<br>• Paradoxical breathing<br>• Inability to lie flat | Ensure airway patency<br>Avoid agitating child<br>Optimise head position:<br>• Continue child's adopted posture if possible<br>• Infant: neutral position<br>• Child: head tilt, chin lift<br>• Jaw thrust for suspected cervical injury<br><br>Consider:<br>• Suction<br>• Oropharyngeal or nasopharyngeal airway<br>• Oxygen (humidified if in respiratory distress)<br>• PEEP if upper airway obstruction<br>***(If severe, escalate to a senior/ anaesthetist for consideration for endotracheal intubation)*** |
| **Breathing** | | |
| • Adequacy of oxygenation and ventilation<br>• Respiratory muscle endurance | Vital signs<br>• Tachypnoea or bradypnoea<br>• Oxygen desaturation or cyanosis<br><br>Effort of breathing<br>• Limited ability to talk/fatigue<br>• Symmetry and quality of chest rise/fall<br>• Increased work of breathing (nasal flaring, grunting, head bobbing, accessory muscle use, retractions)<br>• Auscultation findings (reduced air entry, wheezing or crepitations)<br>• Abnormal breathing pattern (shallow breathing, Kussmaul breathing, Cheyne-Stokes)<br><br>Secondary signs of inadequate oxygenation or ventilation:<br>• Agitation<br>• Altered mental state<br>• Tachycardia/bradycardia<br>• Pallor<br>• Cool peripheries | Consider:<br>• Elevating head of bed<br>• Oxygen supplementation<br>• Institute specific therapy for the underlying cause of respiratory failure<br>• Medications, e.g. bronchodilators for asthma, diuretics in heart failure, antibiotics for pneumonia, etc.<br>• Arterial/Capillary blood gas<br>• Non-invasive ventilation, e.g. High flow nasal cannulae, CPAP, BiPAP<br>• Invasive mechanical ventilation<br><br>***If the child is not breathing, commence bag-mask ventilation and prepare for endotracheal intubation***<br><br>Indications for intubation:<br>• Reduced respiratory drive<br>• Loss of protective airway reflexes<br>• Intracranial hypertension<br>• Severe airway obstruction<br>• Respiratory failure/fatigue/apnoea<br>• Shock<br>• To reduce metabolic demand |

(*Continued*)

**Table 1.2 (*Continued*)**

| Assessment | Clinical Features | Management |
|---|---|---|
| **Circulation** | | |
| Efficacy of circulation | Heart Rate/Rhythm:<br>• Tachycardia/bradycardia<br>• Arrhythmia<br><br>Blood Pressure:<br>• Narrow/wide pulse pressure<br>• Hypotension (late sign)<br><br>Assessment of Perfusion<br>• Pallor/cyanosis<br>• Weak/thready pulses<br>• Flash or delayed capillary refill time (>2 s)<br>• Cool/mottled peripheries<br><br>Secondary signs:<br>• Reduced conscious level<br>• Reduced urine output<br>• Tachypnoea or bradypnoea<br><br>Praecordium:<br>• New murmur<br>• Gallop rhythm<br>• Hyperdynamic praecordium | • Determine cardiac rhythm and treat accordingly<br>• Obtain large bore intravenous access x 2 (consider intraosseous access early)<br>• Arterial/capillary blood gas (including lactate if possible)<br>• Fluid resuscitation (10–20 ml/kg bolus of 0.9% sodium chloride for acute resuscitation or over 15–60 min if less acute (up to 40 ml/kg, with caution if suspicious of cardiogenic shock — consider early inotropic support)<br>• Consider blood products for haemorrhagic shock, severe anaemia or coagulopathic states<br>• Consider early antibiotics (within 1 h)<br>• Consider peripheral inotropes/ vasopressors<br>• Correct electrolyte imbalances<br><br>***Consider early endotracheal intubation and assisted ventilation in shock (reduces work of breathing and cardiac metabolic demands, especially in cardiogenic and septic shock)***<br><br>***If there are no signs of circulation (no pulse, bradycardia <60/min), commence cardiac compressions*** |
| **Disability** | | |
| Altered mental state | • Irritable<br>• Lethargic<br>• Asymmetrical pupillary reflexes<br>• Tense fontanelles<br>• Focal neurological deficits<br>• Rapidly decreasing conscious level<br>• Seizures | • Avoid hypoxia or hypercarbia<br>• Avoid hyperthermia<br>• Avoid hypo/hypertension<br>• Correct metabolic derangements (hypoglycaemia, symptomatic hypo/ hypernatraemia or hypo/hypercalcaemia)<br>• Manage seizures<br><br>Consider intracranial pathologies:<br>• Stroke/haemorrhage<br>• Meningitis/encephalitis/abscess<br>• Ventriculo-peritoneal shunt complications |
| **Exposure** | | |
| Assess child, equipment and environment systematically | • Hypo/hyperthermia<br>• Rash, petechiae, purpura<br>• Signs of injury (spinal injury, penetrating injury, flail chest, deformities, open wounds, burns, ongoing bleeding) | • Correct hypothermia<br>• Analgesia/sedation<br>• Secure tubes/lines<br>• Secure haemostasis<br>• Clean open wounds<br>• Stabilise injuries<br>• Consider cervical collar/spinal nursing |

- Septic screen including blood and urine cultures if sepsis is suspected. Consider cerebrospinal fluid cultures if an intracranial infection is suspected.
- Liver function test/coagulation profile if suspected liver dysfunction/bleeding diathesis.
- Serum lactate if available. This reflects tissue hypoperfusion and can be used as a marker of response to therapy.
- Metabolic screen if there is unexplained severe metabolic acidosis/hypoglycaemia.
- Drug toxicology screen if suspected.

## Monitoring

- Continuous pulse oximetry, heart rate and respiratory rate monitoring.
- Close BP monitoring: Consider invasive BP/central venous pressure (CVP) monitoring if there are concerns about haemodynamic status.
- Continuous cardiac monitoring if there are concerns about rhythm disturbances or myocardial dysfunction.
- Conscious level monitoring.
- Urine output as a marker of perfusion and end-organ function.

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CHAPTER 2

# Cardiopulmonary Resuscitation

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## Introduction

In children, cardiopulmonary arrests are usually a result of progressive respiratory failure or shock. While the survival of children with in-hospital cardiac arrests has improved over the years, with recent local data showing 45.6% survival-to-discharge, survival of children from out-of-hospital cardiac arrests remains poor (3.4%). Despite the improved outcome of in-hospital cardiopulmonary resuscitation (CPR), a substantial proportion of survivors have significant neurological deficits.

The provision of advanced paediatric life support usually takes place in a hospital environment where multiple responders can be rapidly mobilised and are capable of simultaneous actions. Important aspects of a successful resuscitation from cardiac arrest include early high-quality chest compressions, early assisted ventilation and early defibrillation (if indicated).

## Assessment

- Recognition of cardiac arrest should take no more than 10 seconds.
- The absence or presence of a pulse is not a reliable determinant of cardiac arrest; other determinants include unresponsiveness, gasping breaths and no spontaneous respirations.
- If cardiac arrest has occurred, activate the Emergency Code Team.

## Management (Infant <1 year, excluding newborns; child 1–12 years; adolescents >12 years)

### Airway/Breathing

- Open airway by appropriate positioning of head.
- Infant: Neutral head position
- Child: Head tilt-chin lift
- Jaw thrust for suspected cervical injury.
- Clear the airway by brief suctioning of mouth and pharynx, if secretions are present.
- Apply bag-mask ventilation with 100% oxygen (at least 15 L/min oxygen flow).

- Use an appropriately sized oropharyngeal airway only in the unconscious child. (Measure the oropharyngeal airway length from incisors to angle of mandible.)
- Ensure correct mask size, tight seal between mask and face, and assess for effectiveness of ventilation (saturations, chest rise, air entry) but avoid excessive ventilation.
- Deliver each breath with an inspiratory time of about 1 s.
- Ventilation should be synchronised with chest compressions at 30 compressions followed by 2 ventilations [i.e. 30:2 ratio] in un-intubated patients in cardiac arrest [if >12 years old and/or only 1 rescuer]; 15:2 ratio if ≤12 years old and at least two rescuers present. In intubated patients, ventilate asynchronously without interrupting chest compressions (30/min for infants, 20/min for children and 10–12/min for adolescents).
- In a patient with a perfusing rhythm but absent or inadequate respiratory effort, give rescue breathing (30/min for infants, 20/min for children and 12/min for adolescents).

## Circulation

- Place on cardiac monitor/defibrillator monitoring.
- Place the patient on a firm board and commence chest compressions if pulseless, or heart rate <60/min in child with evidence of shock (see algorithm on "pulseless arrest").
- High-quality CPR requires chest compressions to be of appropriate depth and rate, with complete chest recoil after each compression, minimal interruptions to the chest compressions and avoidance of excessive ventilation. (If manpower permits, it is a good practice to appoint a CPR coach to supervise and provide feedback for CPR.)
- Use "two fingers" (lone rescuer) or "thumb encircling" technique for an infant, placed 1 finger breadth below the intermammary line (lower half of sternum).
- Use the heel of 1–2 hands for an older child placed at the lower half of the sternum in the midline.
- Push Hard: Depress at least a third of the Anterior-Posterior (AP) diameter of the chest (target 3–4 cm for infants, 4–5 cm for children and 4–6 cm for adolescents).
- Push Fast: Chest compressions of at least 100–120/min.

## Endotracheal Intubation

- Endotracheal intubation should be performed by an experienced doctor. (See Appendix III "Useful Formulae" for endotracheal tube (ETT) size formula and lengths.)
- Use of Rapid Sequence Induction (RSI) drugs (See Appendix III "Useful Formulae") facilitates emergency intubation and reduces the risk of complications associated with intubation. RSI should be used only by trained, experienced providers. Patients in cardiopulmonary arrest and/or deep coma do not need RSI; otherwise, intubation should be preceded by oxygenation and RSI agents.
- RSI agents typically consist of an anaesthetic induction agent and a muscle relaxant. Maintenance of oxygenation and ventilation by bag-valve-mask

ventilation should be effective before paralysis is instituted. Use paralytic and sedative agents with caution in a patient with a difficult airway as sedating and paralysing such patients may render bag-mask ventilation more challenging. Plan ahead and consider early escalation to a more experienced provider.

- Both cuffed and uncuffed ETT may be used in infants and children. If cuffed tubes are used, cuff inflating pressures should be monitored and limited according to manufacturer's guidelines. Uncuffed ETTs are recommended for neonates.
- Indications for cuffed ETTs include large ETT leak, poor lung compliance and high airway resistance.
- Use both clinical assessment and confirmatory devices (where available) to verify correct ETT placement.

## Indications for Intubation

- Severe airway obstruction
- Airway protection in the presence of poor/absent airway reflexes, e.g. decreased conscious level, prolonged seizures, abnormal brainstem function, bulbar/pseudobulbar palsy
- Apnoea or ineffective respiratory drive
- Respiratory failure not responding to or not suitable for non-invasive ventilation
- To assist with airway toileting

## Vascular Access

- To obtain two large bore intravenous access for resuscitation. This attempt should take no longer than 60–90 sec.
- Intraosseous (IO) access into the anterior tibial bone marrow (about 1–3 cm below and medial to the tibial tuberosity) can be used if venous access is difficult. All intravenous medications/blood products can be given intraosseously.
- The endotracheal (ET) route can be used to give lipid-soluble emergency drugs (LEAN-lignocaine, epinephrine/adrenaline, atropine, naloxone) followed by a flush of 5 ml normal saline and five manual ventilations. Optimal ET drug doses are unknown, the general consensus is double or triple the dose of lignocaine, atropine or naloxone. For adrenaline, an ET dose ten times the intravenous dose (0.1 ml/kg of 1:1000 concentration) is recommended.
- Central venous cannulation or a venous cutdown may be performed if expertise is available.
- Drugs and fluids (See Appendix II — Drugs [Cardiovascular]). Use isotonic crystalloids as initial fluid for shock/fluid resuscitation. Give aliquots of 20 ml/kg boluses and assess for response.
- There is no added benefit to using colloids during early resuscitative efforts, and there is an associated risk of mortality with the use of colloids for resuscitation in children with trauma, burns or traumatic brain injury.
- Routine use of sodium bicarbonate is not recommended in cardiac arrest. It may be administered for treatment of tricyclic antidepressant overdose or hyperkalaemia.

- Routine use of calcium is not recommended for paediatric cardiopulmonary arrest in the absence of documented hypocalcaemia, calcium channel blocker overdose, hypermagnesaemia or hyperkalaemia.

### Gastric Insufflation

- Insert a nasogastric tube if abdominal distension is marked and/or oxygenation and ventilation are compromised.
- Pass the tube after intubation as it may interfere with gastroesophageal sphincter function, increasing risk of aspiration during intubation

### Termination of Resuscitative Efforts

- Details of events and treatment must be recorded. Where there is no response after 20 minutes of adequate resuscitation, the senior doctor should decide how much longer resuscitative efforts should continue.

## Special Resuscitative Conditions

### Ventilation with a Tracheostomy

- Use the tracheostomy tube for assisted ventilation and watch for adequate chest rise.
- If the tracheostomy tube does not allow effective ventilation even after suctioning, change it.
- If still unable to maintain effective ventilation, attempt alternative ventilation methods such as bag-mask ventilation through the nose and mouth while occluding the tracheal stoma.

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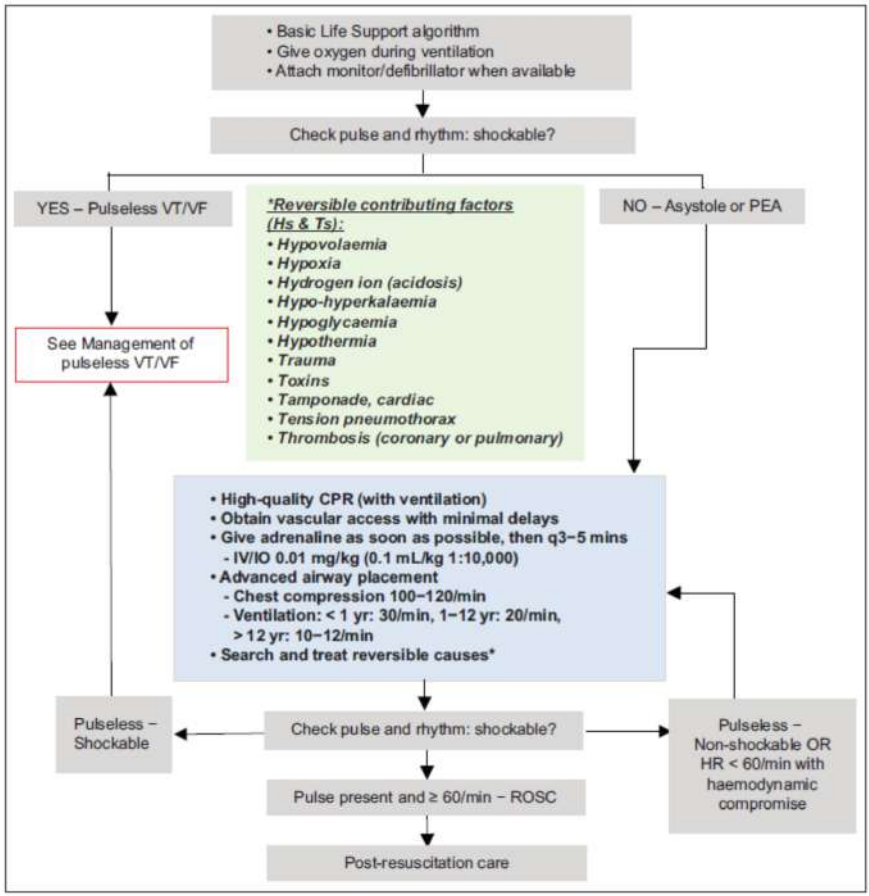
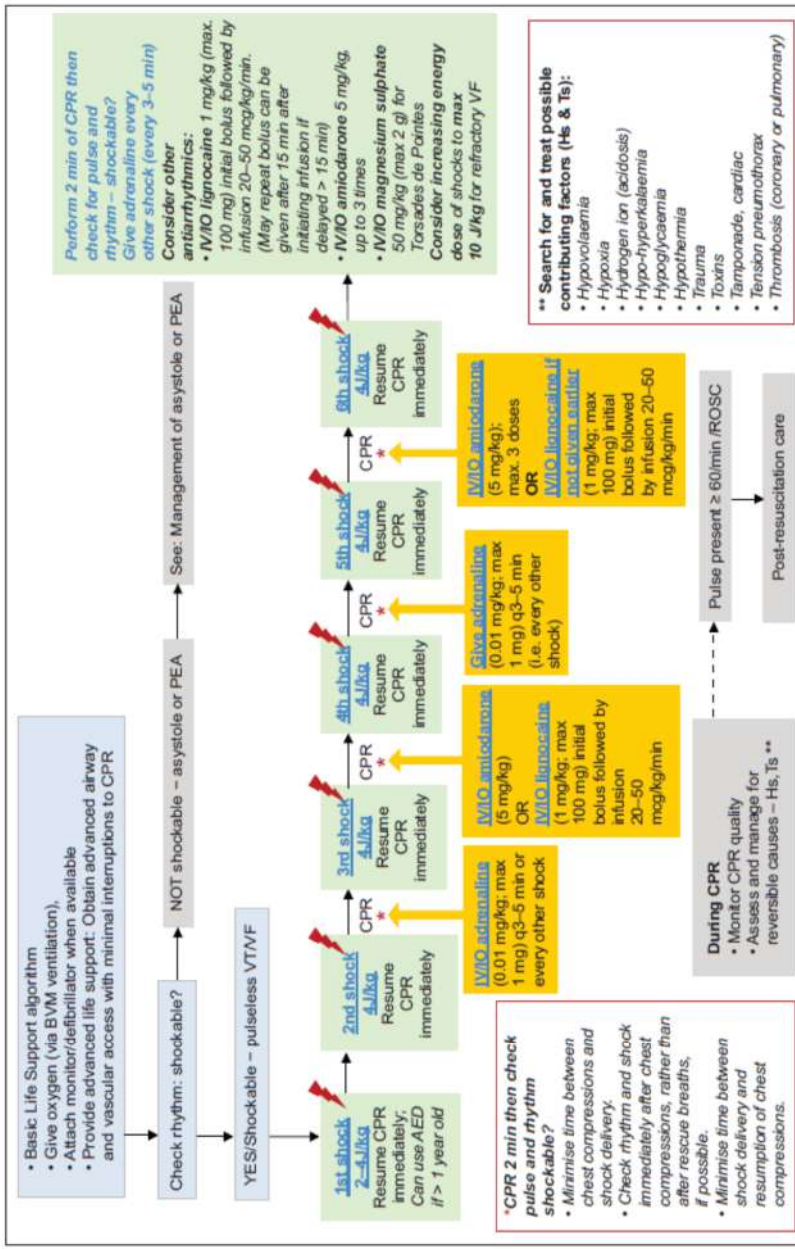
**RESUSCITATION ALGORITHMS**

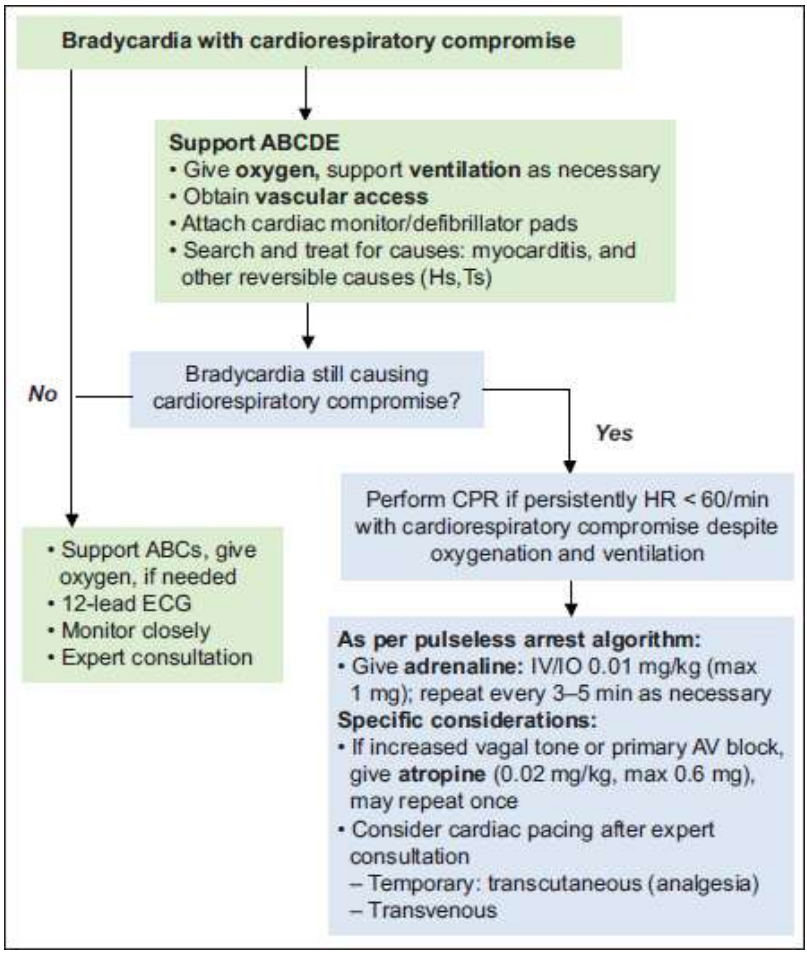
**ALGORITHM FOR PULSELESS ARREST**



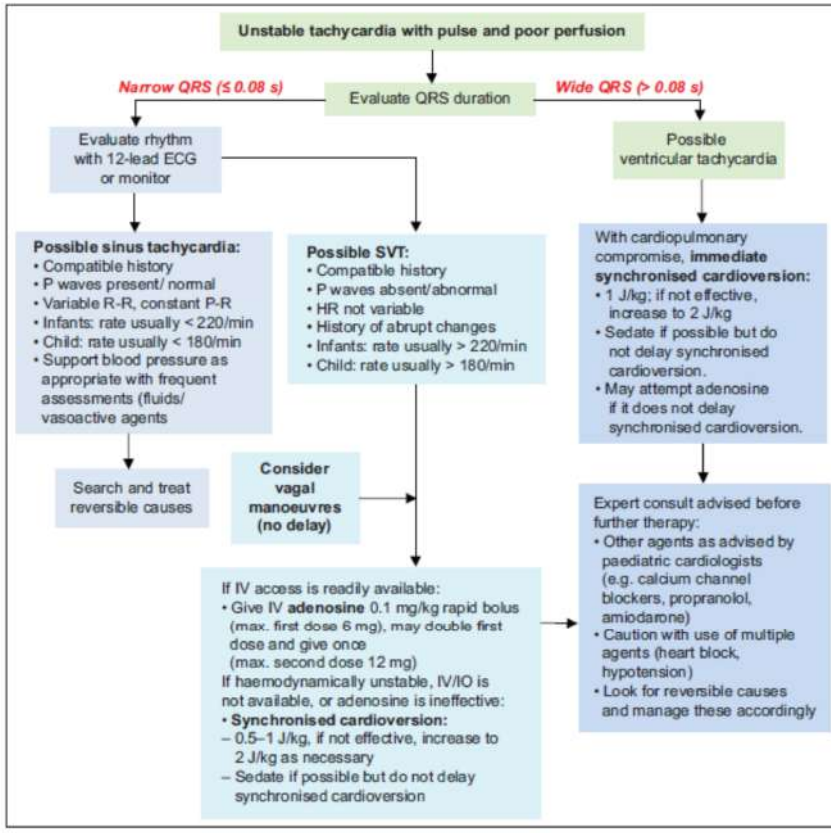



**ALGORITHM FOR PULSELESS ARREST (SHOCKABLE)**

**ALGORITHM FOR BRADYCARDIA**



**ALGORITHM FOR UNSTABLE TACHYCARDIA WITH POOR PERFUSION**



CHAPTER 3

# Drug Overdose and Poisoning

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## Epidemiology

There is usually a bimodal age peak distribution for paediatric poisoning. The first peak includes younger children (<6 years old) and the second peak includes the adolescents (>12 years old).

Toddler toxicology is usually unintentional ingestion of pharmacological and non-pharmacological products and mostly benign due to the small quantities involved.

## General Approach

The general approach to the poisoned patient can be summarised as stabilisation, history, examination, diagnosis, decontamination and enhanced elimination, poison-specific treatment and disposition (Figure 1.1).

### Stabilisation

Stabilisation is priority in managing toxic ingestion. The patient should be rapidly assessed to determine patency of airway and adequacy of ventilation, mental status and cardiovascular stability. Correction of any associated hypoxia and hypoglycaemia is crucial. Initial management priorities are maintenance and protection of the airway, supporting ventilation and circulation. Unstable patients should be placed on a cardiac monitor with measurement of vital signs every 5 to 15 min until the patient is stabilised. The potential for rapid changes in the patient's condition should be considered in making decisions about airway and ventilatory support.

With regard to toxicology, prolonged resuscitation should be attempted in drug-induced cardiac arrest. Extracorporeal membrane oxygenation (ECMO) should be considered if resources and expertise allow.

## History

- History and physical examination are performed concurrently with stabilisation.
- History should include
  - Who: Patient characteristics, age, weight, relationship to others present and gender
  - What: Name and dosage of medication(s) or substance(s) of abuse, co-ingestants and amount ingested
  - When: Timing and date of ingestion

Where: Route which toxins was taken
Why: Whether intentional or accidental and associated details

- For paediatric ingestions, thorough history from caregivers is crucial.
- If the toxic ingestion is unwitnessed (time, dose), always manage as for worst-case scenario even if the patient is asymptomatic, especially if the alleged drug is highly toxic.

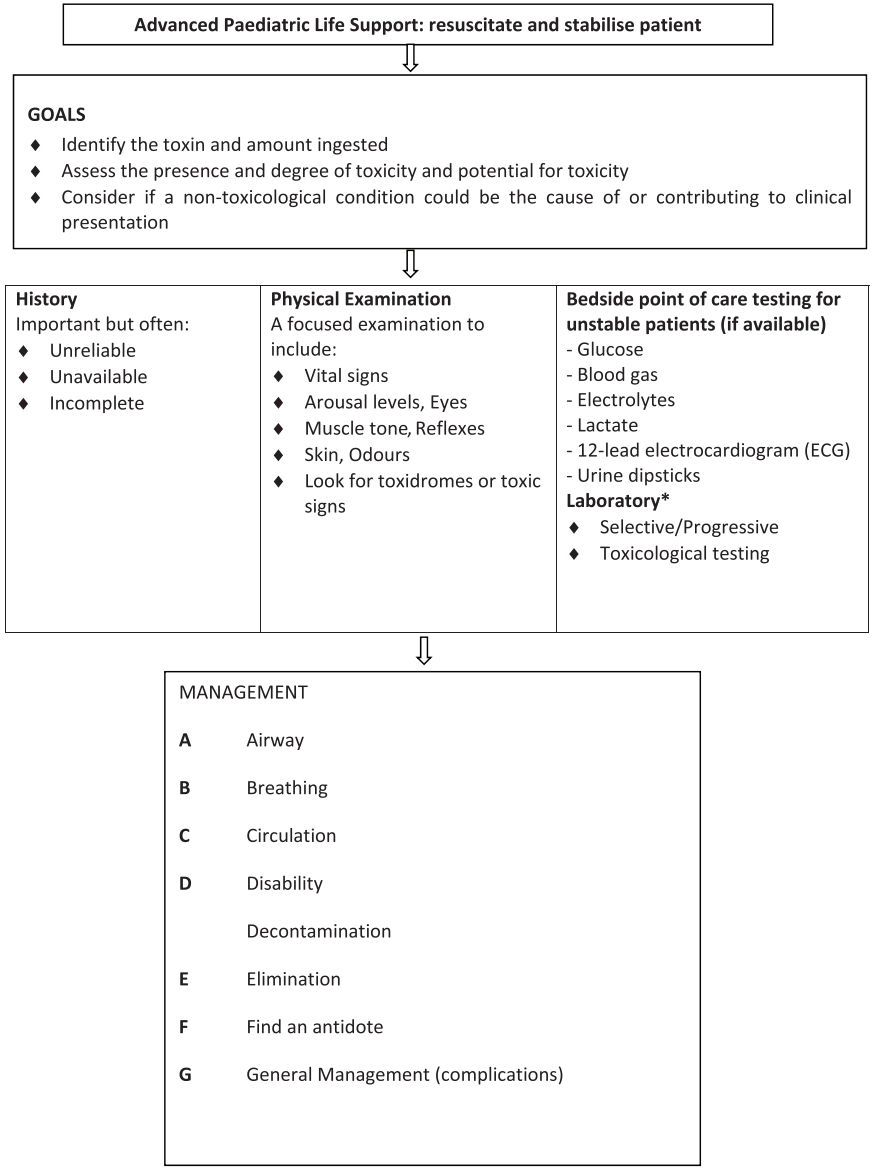


**Figure 1.1** Assessment and management of a poisoned child.

- Other important historical points from the caregivers include a systemic review, specifically the presence of seizures, agitation, coma, vomiting, headache and shortness of breath.
- Clinicians should be aware that in some cases, the history may be unreliable for teenagers, especially if the toxic ingestion was intentional or if there was ongoing inappropriate substance use. Patients may not know what they ingested, the patient's altered mental status may impede communication, or the patient may intentionally mislead the clinician.

## Clinical Examination

- A thorough physical examination should be performed to help with the diagnosis and identify complications.
- Specific attention to vital signs, mental status (depressed or agitated), respiration (depressed, evidence of pulmonary oedema or aspiration), pupils (size, reactivity, presence of nystagmus), skin (diaphoresis or abnormally dry, blisters), bowel sounds (increased or decreased).
- Based on findings from physical examination, the clinician should specifically consider the presence of a toxidrome.
- Note that for young paediatric patients, the classical findings may be different from adolescents and adults. For example, for organophosphate poisoning in young children, nicotinic symptoms may predominate over muscarinic symptoms, and they may present predominantly with muscle weakness with respiratory insufficiency, altered mental status and seizures. The pupils of the organophosphate poisoned young child may not exhibit the classical miosis.

## Decontamination

After the patient is stabilised, consideration should be given to removing the toxin. The choice of decontamination should be made based on the clinical status and the suspected toxin. Patients with an inhalation exposure should be removed from the source, with care taken to avoid exposure of the rescuers. When toxic compounds have been splashed into the eyes or onto the skin, copious irrigation should be performed.

### Paediatric Gastrointestinal Decontamination

1. Single-dose activated charcoal

The preferred method of GI decontamination. It is indicated as a gastric decontaminant agent if a patient has ingested a potentially toxic amount of a poison up to 1 hour following ingestion.

However, it can be considered for paracetamol toxic ingestion up to 2 hours. Other toxins for which delayed administration can be given include aspirin, digoxin, phenobarbitone, anticholinergics, tricyclics and sustained release tablets.

Dose: 1 g/kg (maximum of 50 g if less than 12 years old).

Drugs which are not absorbable by charcoal — **PHAILS**: Pesticides, Hydrocarbons, Acids/Alkalis, Iron, Lithium and Solvents.

Activated charcoal is contraindicated in patients who are unable to protect their airway. The use of orogastric and nasogastric tubes for administration of activated charcoal

in a drowsy or vomiting patient has been associated with pulmonary complications, unless the airway has been secured.

2. Whole bowel irrigation

Whole bowel irrigation (WBI) is not routinely performed in the management of the poisoned patient. WBI is best used in patients who are not vomiting and have normal intestinal function. They are useful in patients with ingestions of highly toxic substances that are poorly bound to activated charcoal, such as iron, lithium or lead, as well as for sustained-release medications (e.g. certain formulations of lithium, theophylline or calcium channel blockers).

WBI is contraindicated in patients with bowel obstruction, gastric haemorrhage, perforation and ileus. It is also contraindicated in unstable patients or inpatients with compromised unprotected airways, haemodynamic instability and intractable vomiting.

The procedure is administered by giving polyethylene glycol bowel preparation at a rate of 1 to 2 L/h in adolescents and 500 ml/h in children through a nasogastric tube, to induce osmotic diarrhoea till rectal effluent is clear (usually about 6 hours). The electrolytes of small children must be closely monitored. For radio-opaque compounds (**CHIPES**: chloral hydrate, heavy metals, iron, phenothiazines, enteric-coated tablets, salicylates/substances in containers, e.g. body packers), continue until repeat radiographs are clear.

3. Gastric lavage

Gastric lavage is not routinely performed in the management of poisoned patients. In certain cases where the procedure is of attractive theoretical benefit (e.g. recent ingestion of a very toxic substance within 1 hour), the substantial risks should be weighed carefully against the sparse evidence that the procedure is of any benefit.

## Enhanced Elimination

Enhanced elimination techniques aim to increase the rate of removal of an agent from the body with the aim of reducing the severity and duration of clinical intoxication.

Enhanced elimination techniques are only indicated for poisonings when the following requirements are met: severe toxicity, poor outcome despite good supportive care/antidote administration, slow endogenous rates of elimination, suitable pharmacokinetic properties, benefit exceeds risks, and will not distract from other priorities (e.g. resuscitation, supportive care, decontamination and antidote therapy).

Techniques for enhanced elimination include:

1. Multiple-dose activated charcoal (MDAC)
    - This causes interruption of entero-hepatic circulation and gastrointestinal dialysis for small, lipid soluble molecule drugs with a small volume of distribution and low protein binding.
    - Drugs (ABCD): Antimalarials (quinine), Aminophylline (theophylline), Barbiturates, Carbamazepine, Dapsone.
    - Contraindications: decreased consciousness/anticipated decreased level of consciousness without prior airway protection, bowel obstruction.
    - Give an initial dose of activated charcoal 1 g/kg (children) or 50 g (adolescents) and repeat doses of 0.25 g/kg/h (paediatrics) or 12.5 g/h in divided doses over 2–4 hours.

- Check bowel sounds prior to administration of each dose and reconsider the indications and clinical endpoints for therapy every 6 hours. Multiple-dose activated charcoal should rarely be required beyond 6 hours.
- Complications include emesis (30%), charcoal aspiration, constipation, charcoal bezoar formation, bowel obstruction, bowel perforation (rare) and corneal abrasions. MDAC also risks distracting manpower from resuscitation and supportive care priorities.

2. Urinary alkalinisation
   - Urinary alkalinisation enhances renal elimination of weak acids such as salicylates, phenobarbital and chlorpropamide.
   - Loading dose of IV sodium bicarbonate 1–2 mmol/kg followed by infusing bicarbonate-containing fluids: 3 ampules (150 mmol) of sodium bicarbonate to 1 L of dextrose 5% and run at 250 ml/h (run the IV at 1.5 times maintenance in children).
   - Achieving urinary alkalinisation is impossible if there is total body potassium depletion, even in the presence of normal potassium levels on the renal panel. Potassium supplementation is required.
   - Follow urinary pH hourly. Blood pH, salicylate levels and electrolytes should be monitored regularly. The frequency of monitoring is dependent upon the severity of the clinical situation
   - The goal is to achieve urine pH of 7.5–8.5.

3. Extracorporeal elimination (e.g. haemodialysis, haemofiltration, haemoperfusion, plasmapheresis and exchange transfusion)
   - Effectively enhances elimination of any drug that is a small molecule, has a small volume of distribution, rapid redistribution from tissues and plasma and slow endogenous elimination. Continuous renal replacement therapy (CRRT) has lower drug clearance per unit of time but slow continuous removal may be preferred when the patient is haemodynamically unstable (unable to tolerate rapid solute and fluid loss), the drugs are highly protein/tissue bound toxicant with large volume of distribution with very long plasma half-lives and exhibit 'rebound' phenomenon (e.g. lithium)
   - Indications for haemodialysis/haemofiltration are: carbamazepine (if MDAC is inadequate or infeasible), valproate, lithium, concomitant severe hyperkalaemia, metformin lactic acidosis, methotrexate (if in acute renal failure or leucovorin is not available), paraquat (within 2 hours of ingestion), salicylates, toxic alcohols and theophylline.
   - Exchange transfusion can be considered for severe drug-induced methaemoglobinaemia, or if refractory to IV methylene blue and IV ascorbic acid, if antidote is contraindicated (e.g. methylene blue in G6PD deficiency) or antidotes are unavailable.

## Antidotes

- Antidotes are typically given after stabilisation and when the diagnosis was made.

- In certain cases, prompt administration of antidote is imperative. E.g. prompt administration of naloxone may avoid the need for endotracheal intubation in opiate overdose.



## Investigations

In general, toxicological testing can serve a number of purposes including:

- Confirming a toxic aetiology and avoiding the need for further diagnostic studies
- Identifying a specific agent
- Assessing the severity of the intoxication
- Assessing efficacy of treatment
- Guiding management decisions
- Prognostication
- Guiding transfer decisions

## Disposition

Consider transfer to higher centre in the following circumstances:

- Inability to stabilise patient or deteriorating patient
- Inability to provide patient with ongoing maintenance treatment
- Lack of availability of toxin-specific therapy
- Management and disposition of patients following decontamination is toxin and patient-specific, e.g. interventions such as dialysis, haemodialysis and haemoperfusion.
- All patients who have taken a suicidal ingestion require assessment of suicidal risk prior to discharge, possibly transferring to Institute of Mental Health if still actively suicidal.

**Table 1.3 List of Antidotes for Initial Management of Common and Dangerous Poisons (Referral to clinical toxicologists for more detailed management is advised)**

| Toxin | Antidote/Paediatric dose |
|---|---|
| **Analgesia Poisoning** | |
| Paracetamol | Paediatric dosing for IV N-acetylcysteine<br>Most effective within 8 hr but has significant effects post 8 hr for documented toxicity<br>See section on paracetamol. |
| Salicylates | IV Sodium bicarbonate1–2 mmol/kg bolus<br>Followed by infusion: 150 mmol Sodium bicarbonate + 40 mmol KCl in total volume of 1 L of dextrose 5% infused to maintain urine output at 1.5–2 ml/kg/hr and urine pH at 7.5–8.5<br>Repeat IV sodium bicarbonate bolus as necessary, keep serum potassium normal. |
| Opioid | Correct hypoxia and assist ventilate as main priority<br>IV Naloxone hydrochloride 0.1 mg/kg/dose (max 2 mg/dose). Can repeat up to 0.5 mg/kg or max 10 mg total. Consider infusion in view of short half-life. |

(*Continued*)

**Table 1.3 (*Continued*)**

| Toxin | Antidote/Paediatric dose |
|---|---|
| **Cardiovascular drug toxicity** | |
| Calcium channel blocker<br>Beta-blockers | • IV Calcium<br>- IV 10% Calcium gluconate 0.5 ml/kg (max 20 ml/dose)<br>- IV 10% Calcium chloride 0.2 ml/kg (max 10 ml/dose)<br>• IV Glucagon (more effective for beta-blockers than calcium channel blockers)<br>- Paediatric: Initial bolus of 150 mcg/kg and repeat as necessary (max 2 mg/dose up to a max of 10 mg). If effective, may be followed by an infusion of 50–100 mcg/kg/hr (max 5 mg/hr)<br>• High-dose Insulin Euglycaemic Therapy (HIET)–IV Insulin 1 unit/kg with 0.5 g/kg dextrose bolus<br>- Followed by insulin infusion 0.5–2 unit/kg/hr together with a dextrose 10%-saline infusion<br>• 20% MCT emulsion: Intralipid/lipofundin<br>- Bolus: 1.5 ml/kg and may repeat as needed.<br>- Followed by infusion: 0.25 ml/kg/min over 30 to 60 min<br>- Max total dose: 12 ml/kg |
| Digoxin | Digoxin immune antibody fragment: Empiric dosing: 10–20 vials IV bolus for life-threatening toxicity (See package insert for other dosing regimens) |
| Tricyclic antidepressants and other sodium channel blockers | IV sodium bicarbonate: 1–2 mmol/kg<br>Repeat boluses titrate to QRS duration (do not exceed arterial pH 7.55) |
| **Cholinergic Poisoning** | |
| Carbamates | IV Atropine 0.05–0.1 mg/kg |
| Organophosphate | IV Atropine 0.05–0.1 mg/kg<br>Consider IV Pralidoxime 20–50 mg/kg (max 2 g/dose) for Diethyl organophosphate poisoning and specific nerve agent poisoning |
| **Heavy Metals** | |
| Iron | IV Deferoxamine 5–15 mg/kg/hr infusion |
| Arsenic | PO Succimer (DMSA) 10 mg/kg/dose TDS<br>IM British Anti-Lewisite (BAL) (dimercaprol) 3–5 mg/kg/dose Q4-6H (only if unable to tolerate succimer). BAL is contraindicated in peanut allergic patients. |
| Lead | Lead encephalopathy:<br>IM BAL (dimercaprol) 4 mg/kg or 50–75 mg/m$^2$ Q4H;<br>(BAL is contraindicated in peanut allergic patients.)<br>Followed by<br>IV Calcium disodium EDTA 50–75 mg/kg, to be administered 4 hr after BAL (1 g of Calcium disodium EDTA can be diluted with 250 ml dextrose 5% or normal saline. Administer over 8–12 hr or continuously over 24 hr).<br>Succimer (DMSA) (if patient is able to tolerate oral medication) 10 mg/kg/dose TDS, repeat doses are often needed<br><br>Further management needed for chelation and referral is advised. |

**Table 1.3 (*Continued*)**

| Toxin | Antidote/Paediatric dose |
|---|---|
| **Toxic Alcohols** | |
| Methanol<br>Ethylene Glycol | Ethanol (10%): Loading dose 10 ml/kg IV or orally followed by maintenance dose 1–2 ml/kg/hr orally<br>OR IV Fomepizole: 15 mg/kg over 30 min, followed by maintenance dose. |
| **Others:** | |
| Bupivacaine (L.A. toxicity) | 20% MCT emulsion: Lipofundin/Intralipid<br>- Bolus: 1.5 ml/kg and may repeat as needed.<br>- Followed by infusion: 0.25 ml/kg/min over 30 to 60 min<br>- Max total dose: 12 ml/kg |
| Cyanide | - Hydroxocobalamin: IV 70 mg/kg (max 5 g/dose) over 15–30 min. Repeat once 70 mg/kg as needed depending on response (total dose 140 mg/kg or max 10 g).<br>- Sodium thiosulphate: Used in conjunction with hydroxocobalamin or sodium nitrite (synergistic). IV of 25% sodium thiosulphate 1–2 ml/kg; max 50 ml/dose.<br>- Sodium nitrite (not for cyanide toxicity in smoke inhalation): IV 3% sodium nitrite 0.2 ml/kg; max 10 ml. Repeat 2 hr later or as necessary, 0.1 ml/kg; max 5 ml. Watch for methaemoglobinaemia. |
| Isoniazid | Pyridoxine: IV 70 mg/kg (max 5 g/dose) over 5 min |
| Methemoglobinemia | Methylene blue: IV 1–2 mg/kg slow infusion, repeat as needed every 30 to 60 min |
| Sulphonylureas | Octreotide: IV 1–1.5 mcg/kg bolus (max 150 mcg/dose) Q6H or continuous infusion |
| **Drug toxicities with antidotes to be used with caution** | |
| Anticholinergics | Physostigmine salicylate 0.02 mg/kg (max 0.5 mg/dose) slowly over 5 min (may cause bradycardia, respiratory distress and seizures).<br>Its use as an antidote for anti-cholinergic toxicity is controversial due to reports of associated seizures and life-threatening arrhythmias. |
| Benzodiazepines | Flumazenil IV 0.01 mg/kg (max 0.2 mg). It should not be routinely administered in the patient presenting with coma or altered mental status.<br>Reversal of benzodiazepine effect in mixed drug ingestion involving cyclic antidepressants and chloral hydrate may result in seizures or arrhythmias with fatal outcomes. |



## Pre-Hospital Paediatric Toxicology — Advice to Caregivers

### Primary Prevention

Most poisonings occur when parents or caregivers are home but children may not be supervised.

Advise caregivers to store medicine, cleaning and laundry products, paints/varnishes and pesticides in their original packaging in locked cabinets or containers, out of sight and reach of children.

Caregivers should never refer to medicine as "candy" or other appealing names.One should be reminded not to give or take medication in the dark. They should also check the label each time a child is given medicine to ensure proper dosage as the same drug may come in different concentrations.

### Home Management if Toxic Exposure has Occurred

If a child is unconscious, not breathing, or having convulsions or seizures due to poison contact or ingestion, they should be advised to call for emergency services immediately. Caregivers or any one nearby should initiate basic life support if they are able to.

### Immediate Management for Home Poisonings

- Toxic ingestion — Advise the caregivers to immediately remove the item away from the child and have the child spit out any remaining substance. Do not induce vomiting or use syrup of ipecac.
- Skin exposures — Remove the child's clothes and rinse the skin with lukewarm water for at least 15 minutes.
- Ocular exposures — Flush the child's eye by holding the eyelid open and pouring a steady stream of room temperature water into the inner corner for 15 minutes.
- Inhalation exposures — The child should be evacuated outside or into fresh air immediately. If respiratory effort is compromised, start cardiopulmonary resuscitation (CPR) and do not stop until the child breathes on his or her own or until someone takes over.

## Clinical Presentations

### Drug-Induced Bradycardia and Hypotension

- Paediatric advanced life support
  - » Perform CPR if despite adequate oxygenation and ventilation heart rate <60/min with poor perfusion
- Treat reversible causes like hypoxia, electrolyte disturbances
- IV fluids
- Inotropic support
- Atropine
- External or internal cardiac pacing
- Resuscitative Extra-Corporeal Life Support (ECMO)

### Drug-Induced Seizures

- The priority still is the management of reversible causes of seizures like hypoxia, hypoglycaemia, electrolyte disturbances and if the history and physical findings are not consistent with toxic seizures, intracranial infections, bleeds, space occupying lesions should be appropriately excluded.

**Table 1.4 Causes of Drug-Induced Bradycardia and Hypotension**

| |
|---|
| Calcium channel blockers |
| Beta-blockers |
| Digoxin |
| Opiates |
| Clonidine |
| Severe tricyclic antidepressant poisoning |
| Severe organophosphate poisoning |

**Table 1.5 Causes of Toxic Seizures**

| |
|---|
| Isoniazid (INH) |
| Gyromitra mushroom or false morel |
| Hydrazine |
| Sulphonylureas — refractory hypoglycaemic seizures |
| Salicylates |
| Tricyclic Antidepressants |

- Toxic seizures are often transient and no specific treatment is required. However close monitoring may be required depending on the half-life of the ingested toxin and other potential life-threatening conditions, e.g. arrhythmias.
- Benzodiazepines (e.g. diazepam or lorazepam) are usually effective for toxic seizures.
- If benzodiazepines are ineffective, barbiturates such as phenobarbital usually will suffice.
- Phenytoin has no role in toxin-induced seizures and is theoretically harmful in poisoning with substances with sodium channel blockade such as tricyclics. It is also contraindicated in theophylline-induced seizures as it worsens toxicity.
- Pyridoxine may be required only in unusual poisonings such as isoniazid toxicity.
- It is essential that the patients who have or had toxic seizures to be on cardiac monitoring and have a 12-lead ECG performed.

## Drug-Induced Altered Mental Status

- Treatment of the most common causes of altered mental status that can be recognised and treated immediately.
- Exclude reversible causes like hypoxia, hypoglycaemia, electrolyte disturbances, sepsis, intracranial pathologies.

**Table 1.6 Drugs Causing Altered Mental Status**

| Clinical Situation/Symptoms |
|---|
| **Depressed sensorium with small pupils and hypotension** |
| Opioids<br>Clonidine<br>Phenothiazines<br>Benzodiazepines<br>Barbiturates<br>Nerve agents<br>Organophosphates<br>Carbamates<br><br>*Classically small pupils are expected in cholinergic poisoning in adults but literature suggests this is variable, with miosis and mydriasis both reported in paediatrics* |
| **Causes of toxidromes with hyperthermia and altered mental status** |
| Sympathomimetic syndrome<br>*(This is usually differentiated from the anticholinergic toxidrome by the presence of marked diaphoresis)*<br>Amphetamines<br>Salbutamol<br>Cocaine<br><br>Anticholinergic toxidrome<br>Antihistamines<br>Anticholinergic plants (e.g. Dhatura)<br>Antiparkinsonian agents<br>Antiemetics<br>Antispasmodics<br>Antipruritic<br>Cyclic antidepressants<br><br>Serotonin syndrome<br>Fluoxetine<br>Citalopram<br>Sertraline<br>Bupropion<br><br>Malignant neuroleptic syndrome<br>*Every class of neuroleptic drug has been implicated, including the low potency (e.g. chlorpromazine) and the newer "atypical" antipsychotic drugs (e.g. clozapine, risperidone, olanzapine)*<br><br>Antipsychotics, e.g. haloperidol, fluphenazine.<br>Metoclopromide<br><br>Malignant hyperthermia<br>Succinylcholine<br>General anaesthesia |

**Table 1.7 Other Drug-Induced Clinical Presentations**

| **Agitated delirium** |
|---|
| Anticholinergics<br>Phencyclidine<br>Sympathomimetics (e.g. cocaine)<br>Hallucinogens<br>Alcohol toxicity |
| **Drug-induced acute movement disorders** |
| Dyskinesia<br>Amphetamines<br>Anticholinergics<br>Antihistamines<br>Cocaine<br>Gamma-hydroxybutyrate<br><br>Dystonia<br>Metoclopramide |



## Paracetamol Poisoning

I) Toxic ingestion if
   a) Single, acute ingestion of ≥200 mg/kg or 10 g (whichever is lesser)
   b) Repeated Supratherapeutic Ingestion (RSTI): defined as more than 1 dose of paracetamol ingested in the last >24–72H. (if repeated dosing ≤24 hours, to follow Acute Ingestion pathway)

      In symptomatic patients — irrespective of dose, if ≥daily therapeutic dose per day for more than 48 hours.

      In asymptomatic patients — cumulative dose of ≥10 g or ≥200 mg/kg (whichever is lesser) within a 24 hour period; or ingestion of ≥4 g/24 hours or ≥100 mg/kg/24 hours beyond 24 hours.

II) Presumed toxic ingestion in
   c) Symptomatic patients (even with known time of ingestion)

## Clinical Presentation

| | |
|---|---|
| Early: | Non-specific with nausea and vomiting |
| 24–48 hours: | Tender hepatomegaly with jaundice |
| Day 4–5: | Acute liver and renal failure |
| Other features: | Erythema, urticaria, haemolytic anaemia, pancreatitis, haemorrhage (prolonged prothrombin time) |

## Investigations

Serum paracetamol level: taken at least 4 hours post-ingestion

For acute ingestions, refer to the Rumack–Matthew nomogram

Liver function test (LFT):

- Transaminases (ALT, AST) begin to rise by 12 hours and peak at 72 to 96 hours
- Serum bilirubin (SB) rises more slowly
- Prothrombin time (PT)/International Normalised Ratio (INR) abnormal by 24 to 36 hours

Other investigations: Renal panel/electrolytes, blood gas

## Treatment

Activated charcoal 1 g/kg (most useful within 2 hours of ingestion).

Specific antidote: IV N-acetyl-cysteine (NAC)

NAC is indicated if:

- Serum paracetamol level lies above the Treatment Nomogram Line on the Rumack–Matthew nomogram ("Probable Toxicity") for acute ingestion
- Asymptomatic with delayed presentation: ≥200 mg/kg or 10 g of paracetamol has been ingested and serum paracetamol level is not likely available within the 8-hour window. Effectiveness depends on initiation of NAC with maximal benefit within 8–10 hours of acute ingestion, diminishing value after 12–16 hours. If paracetamol levels can be made known within 8 hours, need not start till levels are available.
- Symptomatic — unusual for symptoms to occur if ingestion is less than 8 hours (inaccurate timing or dose reporting).
- Sustained-release (SR) preparation or co-ingestion of agents which affect gastric motility. In such cases, start IV NAC and follow RSTI workflow

### Dosage of N-Acetyl-Cysteine (NAC)

For paracetamol poisoning, a total dose of NAC 300 mg/kg body weight was administered over 3 phases traditionally. The current recommended regimen is a 2-bag IV NAC over 20 hours. Reported benefits of this 2-bag regime include significantly reduced non-immune mediated allergic reactions, and it has been shown to be as effective as the 3-bag regimen.

This involves combining the first 2 bags of the traditional 3-course NAC infusion into the loading dose. i.e.

- 1st bag is 200 mg/kg in TOTAL 500 ml (maximum concentration of 22 g over 4 hours, then followed by
- 2nd bag 100 mg/kg over 16 hours.

Please refer to your institution's guidelines for IV NAC infusion and dilution charts for details.

Similar to the 3-bag regimen, the last dose may be repeated if there is ongoing hepatotoxicity. If the initial paracetamol concentration is more than double the nomogram line following acute ingestion, increase NAC dose to 200 mg/kg over 16 hours.

The administration of N-acetyl-cysteine will be dependent on the child's weight for dosing and dilution.

- Phase 1: 200 mg/kg over ≈4 hours
  - » ≤25 kg: Add IV NAC 200 mg/kg in total volume of 100 ml
  - » >25–80 kg: Add IV NAC 200 mg/kg to 500 ml

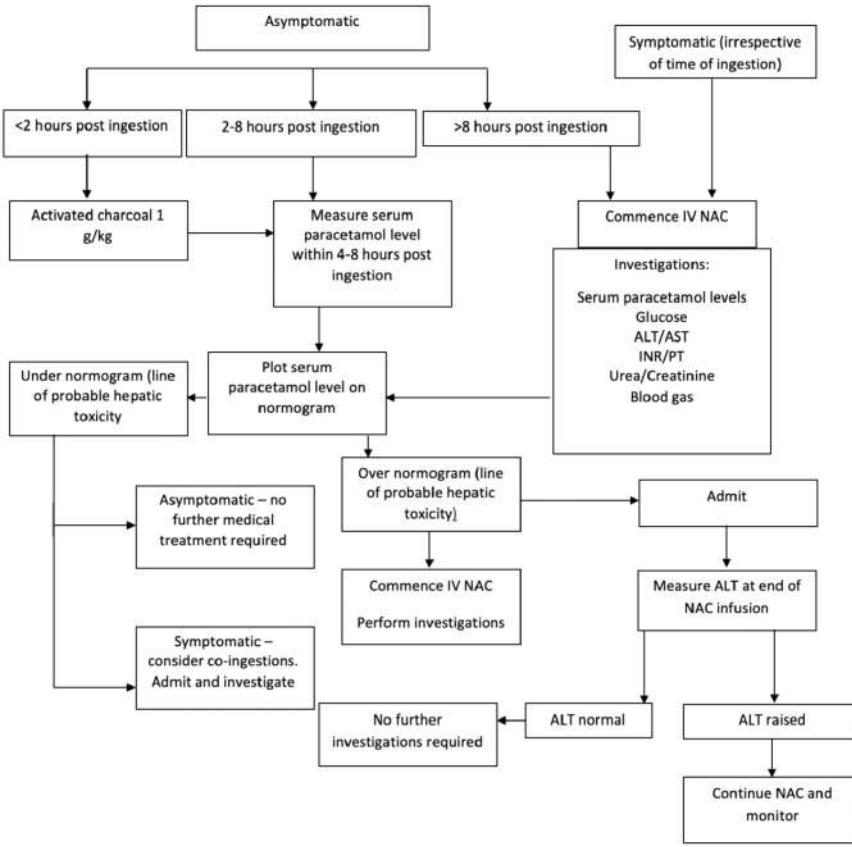


**Figure 1.2** Single, acute toxic ingestion of paracetamol.

  - » >80 kg: Add IV NAC 200 mg/kg to 1000 ml (in 2 bottles × 500 ml)
- Phase 2: 100 mg/kg over ≈16 hours
  - » ≤ 10 kg: Add IV NAC 100 mg/kg in total volume of 100 ml
  - » >10–50 kg: Add IV NAC 100 mg/kg to 500 ml
  - » >50 kg: Add IV NAC 100 mg/kg to 1000 ml (in 2 bottles × 500 ml)

Total dose: 300 mg/kg
Total duration: ≈20 hours

In all cases, additional maintenance fluids can be given if required, or NAC may be administered in larger volume bags if more convenient. At 18 hours into the NAC infusion (2 hours before completion), send bloods for:

- Paracetamol level
- ALT
- Urea, electrolytes, creatinine (5% of patients with paracetamol toxicity will develop acute renal injury)

The NAC infusion should be discontinued only once the:

- ALT < 50U/L, reached their peak levels and are declining

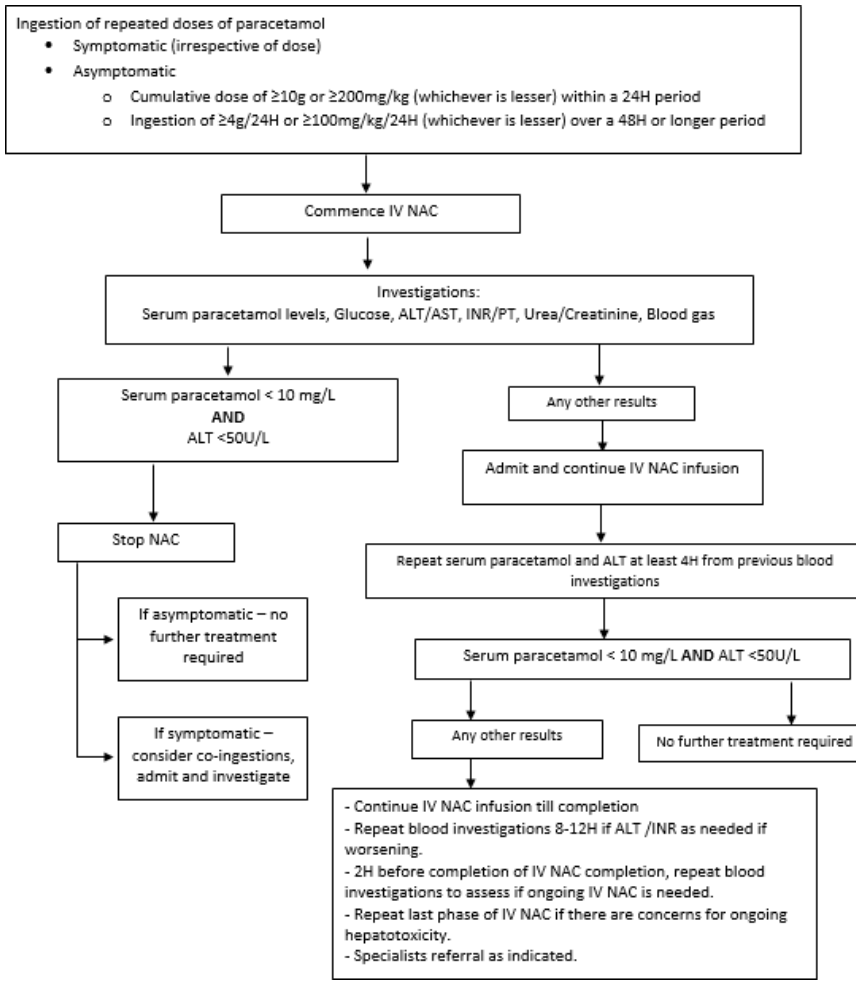


**Figure 1.3** Repeated Supratherapeutic Ingestion of paracetamol.



- Urea, electrolytes and creatinine are normal or have normalised (if previously abnormal)
- Paracetamol level has returned to normal (i.e. below <10 mg/dL)

## Other Considerations

### 1) Massive Paracetamol ingestion and its management

Massive paracetamol ingestion is defined by ingestion of ≥30 g or ≥500 mg/kg (whichever is less). Patients should receive IV NAC till completion and increased doses may be needed. Consult clinical toxicologists and intensive care specialists early for further management. Patients may be symptomatic early and present with severe lactic acidosis with or without liver injury (direct effects on mitochondria).

In addition to the standard paracetamol management, if the patient is haemodynamically unstable and with concerns of end-organ dysfunction or refractory

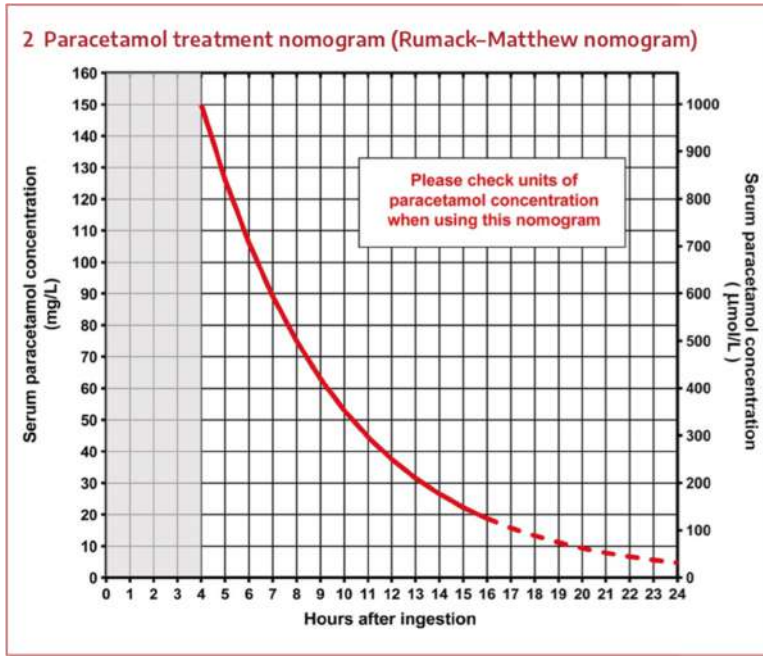


**Figure 1.4** Nomogram-relating plasma or serum paracetamol concentration and probability of hepatotoxicity at varying intervals following ingestion of a single toxic dose of paracetamol.

acidosis not responsive to standard resuscitation, consider haemodialysis early. If haemodialysis is needed, IV NAC dosing may need to be adjusted (consult pharmacists/toxicologists).

Complete IV NAC 2-bag regimen but increased doses needed for the 2nd bag.

If serum paracetamol at 4-hour post ingestion is:

- 2X probable toxicity, i.e. levels are ~300 mg/dL at 4H post ingestion, double the 2nd bag to 200 mg/kg over 16 hours.
- 3X probable toxicity, i.e. levels are ~450 mg/dL at 4H post ingestion, triple the 2nd bag to 300 mg/kg over 16 hours (consult pharmacists/toxicologists)

  Repeat paracetamol levels 4H apart to guide need for further decontamination (stat dose of activated charcoal) and IV NAC dose.

At 18 hours into the NAC infusion (2 hours before completion), send bloods for: Paracetamol level, ALT, and Urea, electrolytes, creatinine.

The NAC infusion should be discontinued only when the:

- ALT < 50U/L, reached their peak levels and are declining
- Urea, electrolytes and creatinine are normal or have normalised (if previously abnormal)
- Paracetamol level has returned to normal (i.e. below <10 mg/dL)

The 2nd bag over 16H may be repeated if there is ongoing hepatotoxicity at completion of IV NAC at 20 hours and dosing adjusted to serum paracetamol (should be <10 mg/dL at 20H).

Refer Gastroenterologists for review and possible need for liver transplant if liver injury is refractory to management.

**2) Acute ingestion of Modified-Release paracetamol or co-ingestion of drugs that significant slow GI transit.**

Dose of modified release preparation ingested

i) If ingestion is <10 g or <200 mg/kg (whichever is less), i.e. non-toxic dose.
- If ≤4 hours post ingestion, administer activated charcoal, obtain bloods at 4 hours post ingestion.
- If presentation is >4 hours post ingestion, activated charcoal is not required. Obtain bloods for ALT, renal panel and paracetamol levels.
- If ingestion occurred close to 8 hours post ingestion with concerns of toxic ingestion to start IV NAC (2-bag standard dosing).

ii) If ingestion is ≥10 g or ≥200 mg/kg (whichever is less)
- If ≤4 hours post ingestion, administer activated charcoal if no contradictions (e.g. ileus) and start IV NAC. Obtain bloods at 4-hour post ingestion.
- If presentation is >4 hours post ingestion, obtain bloods for ALT, renal panel and paracetamol levels and commence IV NAC (2-bag standard dosing). Measure 2 paracetamol concentrations: (i) at least 4 hours post-ingestion and (ii) 4 hours apart.
  - » If either serum paracetamol levels are ≥150 mg/dL at 4H line (possible hepatotoxicity), to complete IV NAC (2-bag standard dosing). If serum paracetamol levels are increasing, consider stat dose activated charcoal and serum levels and ALT re-assessed as needed.
  - » If either serum paracetamol levels are ≥2X probable toxicity, i.e. levels are 300 mg/dL at 4H post-ingestion, double the 2nd bag to 200 mg/kg over 16 hours.

Stopping IV NAC

At 18 hours into the NAC infusion (2 hours before completion), send bloods for: Paracetamol level, ALT, Urea, electrolytes, creatinine.

The NAC infusion should be discontinued only when the:
- ALT < 50U/L, reached their peak levels and are declining
- Urea, electrolytes and creatinine are normal or have normalised (if previously abnormal)
- Paracetamol level has returned to normal (i.e. below <10 mg/dL)

The 2nd bag over 16H may be repeated if there is ongoing hepatotoxicity at completion of IV NAC at 20 hours and dosing adjusted to serum paracetamol (should be <10 mg/dL at 20H).

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