CHAPTER 23

PAEDIATRIC INTENSIVE CARE

Healthcare professionals engaged in intensive care must undergo appropriate training, with junior doctors working under appropriate supervision. Endotracheal intubation should usually be performed using the rapid sequence technique and must be undertaken by the most experienced clinician during emergencies.

For Neonatal Care see:
Chapter 19: Prematurity and Neonatal Conditions.

23.1 RAPID SEQUENCE INTUBATION (RSI)

DESCRIPTION

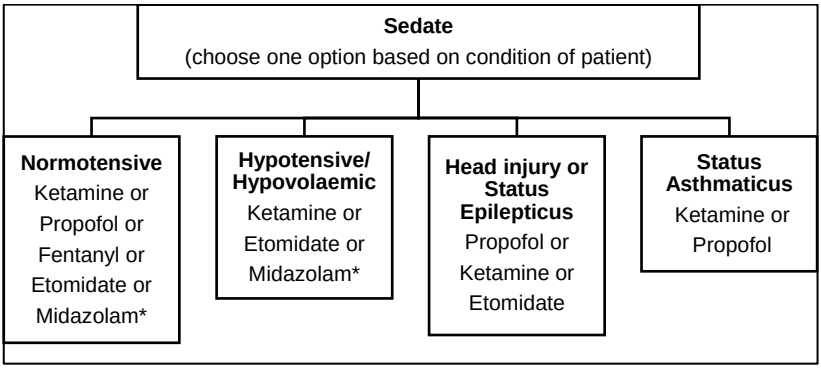
Rapid Sequence Intubation (RSI) is a coordinated, sequential approach to emergency endotracheal intubation designed to optimise success and safety. This technique is recommended for most scenarios necessitating endotracheal intubation. The sequential steps involved in RSI are (The 7 'P's):

1. **Preparation** (10 minutes pre-intubation) – best achieved with the use of a checklist. Focussed history and examination to assess for presence of a difficult airway and conditions which will influence choice of drugs for the procedure. All necessary equipment must be present and checked prior to the administration of any sedatives. A plan for what to do should intubation be unsuccessful is mandatory.
2. **Pre-oxygenation** (5 minutes pre-intubation) – 100% oxygen (or highest concentration available) should be started as soon as the decision to intubate is taken. A minimum of 3 minutes is needed to increase the time to desaturation during intubation.
3. **Pre-treatment** (3 minutes pre-intubation) – **optional**. Atropine may be beneficial to counteract bradycardia during intubation for the following: if suxamethonium is going to be used; for infants and in children with septic or late-stage hypovolaemic shock.
4. **Paralysis post sedation** (Induction) – Sedation must be administered prior to the paralytic agent. The choice of agents will be determined by the clinical characteristics of the patient.
5. **Protection and Positioning** (30 seconds post induction) – cricoid pressure is not recommended for airway protection. External laryngeal manipulation may be utilised to improve the view of the anatomy during intubation.

6. **Placement with confirmation** (45 seconds post induction) – end-tidal CO_2 detection is strongly recommended, in addition to auscultation and visualisation of symmetrical chest rise.
7. **Post intubation management** (60 seconds post induction) – secure the endotracheal tube appropriately and obtain a chest X-ray. Commence ongoing analgosedation.

NOTES:

» If intravenous (IV) access is not available, intraosseous (IO) is preferred to intramuscular (IM) administration.
» Pre-treatment (see table below) is no longer routinely recommended as there is limited evidence of routine benefit.
» Factors influencing choice of sedative:
  > Haemodynamically stable – propofol or ketamine or etomidate.
  > Haemodynamically unstable – ketamine or fentanyl or etomidate.
  > Status asthmaticus – ketamine or etomidate.
  > Status epilepticus – propofol or midazolam. If hypotensive – ketamine or etomidate.
» With respect to paralytic agents, suxamethonium (unless contraindicated) remains the agent of choice. Among the non-depolarizing agents, rocuronium is preferred to vecuronium.
» While etomidate does cause adrenal suppression, it remains an efficacious induction agent for most indications with the potential exception of sepsis.
» Fentanyl can be used in patients with haemodynamic instability, but at a lower starting dose and then titrated to effect (1 mcg/kg vs 3 mcg/kg in haemodynamically stable patients). It is a good option in patients with either suspected myocardial ischaemia or catecholamine depletion. Fentanyl must be administered slowly (30–60 seconds).
» **While midazolam remains a very popular induction agent, it is not recommended for routine use unless it is the only agent available**.



**Midazolam only to be used if no other agent available.*

Suxamethonium contraindications:

» Absolute:
  > Chronic myopathy or denervating neuromuscular disease.
  > 48 to 72 hours post burns, polytrauma, or an acute denervating event.
  > Extensive rhabdomyolysis.
  > Pre-existing hyperkalaemia.
  > Personal or family history of malignant hyperthermia.
» Relative:
  > Renal dysfunction with normal serum potassium.
  > Organophosphate poisoning or any other known pseudo-cholinesterase deficiency.

MEDICINE TREATMENT

Pre-treatment

Drug	IV Dose	Indication
Atropine or Glycopyrrolate	0.02 mg/kg Minimum: 0.1 mg Maximum: 0.5 mg (1 mg adolescents) 0.01–0.08 mg/kg	For patients at increased risk of bradycardia (vagally induced, reflex, succinylcholine). Prior to ketamine administration. As per atropine.
Lidocaine	1–2 mg/kg Maximum: 200 mg	Consider in patients with suspected raised ICP.
Fentanyl	2–3 mcg/kg	Consider in patients with suspected raised ICP.

Sedatives (Induction)

Drug	IV Dose	Time to Effect	Duration of Effect
Ketamine	1–2 mg/kg	60 seconds	5–10 minutes
Propofol	1–3.5 mg/kg	30 seconds	3–10 minutes
Fentanyl	1–5 mcg/kg	1–2 minutes	30–40 minutes
Etomidate	0.3 mg/kg Maximum: 20 mg	15–45 seconds	10–12 minutes
Midazolam	0.2–0.3 mg/kg Maximum: 10 mg	2–3 minutes	30–45 minutes

Paralytic Agents (Neuromuscular Blockers)

Drug	IV Dose	Time to Effect	Duration of Effect
Suxamethonium	1–2 mg/kg	30–60 seconds	5–10 minutes
Rocuronium	1 mg/kg	1–3 minutes	60–90 minutes
Vecuronium	0.1 mg/kg	2–3 minutes	30–40 minutes
Cisatracurium	0.1–0.15 mg/kg	3 minutes	30–45 minutes

Options if no IV or IO access available

Drug	IM Dose	Onset	Comments
Atropine	0.02–0.03 mg/kg	4–8 minutes	Not routinely recommended.
Ketamine	6–10 mg/kg	3–4 minutes	Peak effect after 10–30 minutes.
Midazolam	0.25–0.5 mg/kg Maximum: 10 mg	5– 10 minutes	Peak effect after 10–30 minutes.

Drug	IM Dose	Onset	Comments
Suxamethonium	4 mg/kg Maximum: 150 mg	3–6 minutes	Commonly results in significant local muscle pain.
Rocuronium	1–1.8 mg/kg	5–9 minutes	1 mg/kg for infants, 1.8 mg/kg for children > 1 year.

23.2 ANALGOSEDATION

DESCRIPTION

With respect to the comfort of critically ill children, the first priority is ensuring adequate analgesia, best assessed by validated pain assessment scales such as the revised FLACC (R-FLACC), Wong-Baker Faces, Numeric Pain Rating etc. However, sedation is routinely required to minimise anxiety and facilitate both the delivery of therapies, and performance of various invasive procedures associated with intensive care of children. To optimise the use of sedatives, it is essential to utilise age-appropriate sedation scales, e.g. COMFORT-B Scale or Richmond Agitation Sedation Scale, regularly (4–8 hourly). Please note that many sedatives may decrease blood pressure and prolonged usage is associated with delirium and other adverse outcomes.

DIAGNOSTIC CRITERIA

COMFORT-B Scale Score

Alertness	**1** – Deeply asleep (eyes closed, no response to changes in environment). **2** – Lightly asleep (eyes mostly closed, occasional responses) **3** – Drowsy **4** – Awake & alert. **5** – Awake & hyper-alert.	How responsive is the patient to the ambient light, sound and activity around them? Monitors, phones, talking.
Calm/ Agitation	**1** – Calm **2** – Slightly anxious. **3** – Anxious **4** – Very anxious. **5** – Panicky	How would you rate the patient's level of anxiety?
Respiratory response *(intubated & ventilated)*	**1** – No spontaneous respiration, no cough. **2** – Spontaneous breathing no resistance to ventilator. **3** – Occasional cough or resistance to ventilator. **4** – Actively breathes against ventilator or coughs. **5** – Fights ventilator coughing or choking.	How comfortable and compliant is the patient with ventilation via ET tube?
Respiratory response *(crying & self-ventilated)*	**1** – Quiet breathing, no crying sound. **2** – Occasional sobbing or moaning. **3** – Whining or monotonous sound. **4** – Crying **5** – Screaming or shrieking.	How would you score the intensity of verbal response? (*Significance should be given to the characteristics of the cry, not to the presence of tears.)*

Physical Movement	**1** – No movement. **2** – Occasional (3 or fewer) slight movements. **3** – Frequent, (> 3) slight movements. **4** – Vigorous movements limited to extremities. **5** – Vigorous movements include torso & head.	What is the intensity & frequency of the patient's movements?
Muscle Tone	**1** – Muscles totally relaxed; no muscle tone. **2** – Reduced muscle tone; less than normal. **3** – Normal muscle tone. **4** – Increased muscle tone, increased flexion of fingers & toes. **5** – Extreme muscle rigidity & flexion of fingers & toes. *In cases of complex needs/CP/underlying neuromuscular condition, assess with a parent for the 1st assessment.*	How does the patient's muscle tone compare to a normal awake & alert child of the same age/stage of development? Flex/extend limb. (*Assess this section last.)*
Facial Muscles	**1** – Facial muscles totally relaxed. **2** – Normal facial tone. **3** –Tension evident in some muscles (not sustained). **4** – Tension evident throughout muscles (sustained). **5** – Facial muscles contorted and grimacing.	How does the patient's facial movement/tension compare to that of an awake & alert child of the same age/stage of development

Score uses 6 components; use only 1 respiratory score depending on whether the patient is intubated or not.

GENERAL AND SUPPORTIVE MEASURES

Pharmacotherapy is just part of the overall strategy to decrease the distress of critically ill children. It is crucial to also employ non-pharmacological strategies such as parental presence, calming music, distraction therapy, reduction of unnecessary alarms and environmental noise. Bundling of procedures also limits exposure to distressing situations.

MEDICINE TREATMENT

During continuous mechanical ventilation

	IV Bolus Dose	IV Infusion Dose	Comments
Analgesic			
Morphine	0.05–0.3 mg/kg	20–80 mcg/kg/hour	Significant vasodilator. Needs dose adjustment for renal failure.
Fentanyl	1–5 mcg/kg	1–4 mcg/kg/hour	Easier to titrate due to faster onset of action, but higher risk of tolerance/ tachyphylaxis.
Sedative			

	IV Bolus Dose	IV Infusion Dose	Comments
Midazolam	0.05–0.2 mg/kg	50– 200 mcg/kg/hour	Significant vasodilator. Needs dose adjustment for renal failure.
Lorazepam	0.05–0.1 mg/kg	0.05– 0.15 mg/kg/hour	Maximum daily dose 2 mg/kg or 100 mg. Useful for anticipated longer term sedation, t½ 10–20 hours.
Propofol	0.5–1 mg/kg	1.5–4 mg/kg/hour	For maximum of 48 hours continuously, significant vasodilator.
Ketamine	1–2 mg/kg	5–40 mcg/kg/min	Currently recommended as an adjunctive sedative.

Procedural Sedation

Take standard precautions for respiratory arrest.

Drug	Dose	Comments
Ketamine	0.25–1 mg/kg, IV 2–4 mg/kg, IM 6–10 mg/kg, oral	Both sedative and analgesic. Also results in a dissociative state. Oral dose must be administered 30 minutes before procedure.
Midazolam	0.025–0.1 mg/kg, IV 0.1–0.2 mg/kg, IM 0.25–0.5 mg/kg, oral 0.2–0.3 mg/kg, IN 0.3–0.5 mg/kg, PR	Purely sedative, therefore, will need to combine with an analgesic (opioid) for painful procedures. When combining with an opioid, reduce the dose by 30–50%. Time prior to procedure that dose should be administered: IM – 10 to 20 minutes, oral – 30 to 45 minutes, PR – 30 to 45 minutes.
Fentanyl	0.25 mcg/kg, IV 1–2 mcg/kg, IN	For painful procedures. Administer slowly to reduce risk of chest wall rigidity.

23.3 NUTRITIONAL CARE IN THE ICU

Malnutrition is common (25%) among critically ill children and is associated with increased morbidity, delayed recovery and increased mortality.

Nutritional goals in PICU include:
» Prevention and/or treatment of malnutrition – both macro and micronutrient deficiencies as well as overfeeding.
» Maintenance of gut integrity with enteral nutrition.
» Optimization of organ function.

23.3.1 PARENTERAL NUTRITION

» **Parenteral nutrition (PN) should be prescribed and administered under the supervision of a medical specialist and dietician.**
» PN should be used only when it is not possible to meet nutritional requirements enterally or when there is gastrointestinal dysfunction resulting in inability to tolerate enteral nutrition for a prolonged time: > 5 to 7 days.
» PN is the intravenous administration of amino acids (proteins), lipids, carbohydrates, electrolytes, minerals, vitamins, and trace elements necessary for metabolic requirements and growth.
» PN may be total parenteral nutrition (TPN) where all nutrients are administered, usually via a central venous line, until the child is able to tolerate enteral feeds.
» PN may also be partial parenteral nutrition (PPN) where it is used to supplement enteral feeds in children who cannot yet tolerate their full complement of enteral feeds.
» Whenever possible, the enteral route should still be used to deliver small volumes of feed, in order to protect the integrity of the gut mucosal barrier and preserve mechanical function of the gut. This is known as trophic feeding.
» Administer PN preferably via a central venous catheter (CVC), especially when it is expected that the child will require TPN for more than 7 days. A peripheral venous catheter may only be used for anticipated short term (< 7 days) partial parenteral nutrition. Only PN solutions with an osmolarity < 800 mOsm/L (non-lipid containing) and < 1000 mOsm/L (lipid containing) can be safely administered via a peripheral vein.
» Check peripheral vein infusion sites and patency of the catheter regularly for tissue infiltration.
» Transport and store PN solutions at 2–8 ºC. Start administration of the PN solutions within one hour after removal from the refrigerator.
» Do not make additions to a PN bag or decant contents as the stability and/or the sterility may be compromised.
» Do not use the PN catheter/lumen to collect blood samples.
» Administer PN through a dedicated catheter/lumen and do not administer medications, blood, etc. through the PN catheter/lumen.
» Use a 1.2 micron in-line filter for lipid containing PN solutions and a 0.2 micron filter for lipid-free PN solutions.
» Adhere to a strict aseptic technique when administering PN solutions. Check integrity of packaging before starting the infusion.
» PN bags must not be used beyond 24 hours after starting the infusion.

COMPLICATIONS OF PN

» Central or peripheral venous catheter complications, e.g., extravasation, blockage, infection and venous thrombosis.
» Metabolic complications, e.g., hyperglycaemia, hypoglycaemia, electrolyte and mineral disturbances, micronutrient deficiencies and hyperlipidaemia.

» Metabolic bone disease and growth impairment.
» Cholestatic hepatitis and liver failure.

Monitor:
» Vital signs and hydration.
» Blood glucose 12 hourly; maintain blood glucose at 4.0–10 mmol/L.
» Electrolytes, minerals, and acid-base on a daily basis or more regularly if necessary.
» Growth parameters and weight, twice weekly.
» Liver enzymes, bilirubin, lipids, urea and creatinine once weekly or more frequently, as indicated by the condition of the child.
» Regular screening for the presence of infection.

DOSE AND DURATION OF PN INFUSION
The maximum volume of TPN for a child depends on the age, weight and underlying disease and is based on the total daily fluid requirements.

AVERAGE DAILY PARENTERAL REQUIREMENTS

	Birth–3 months	> 3 months –1 year	> 1–3 years	> 3–6 years	> 6–12 years
Fluid (mL/kg)	100–120	80–100	70–80	60–70	40–60
Energy (kcal/kg)	90–100	90–100	75–90	75–90	60–75
Protein (g/kg)	1.5–3	1.5–2.5	1.5–2.5	1.5	1.5
CHO (g/kg)	16-18	16–18	12–14	10–12	< 12
Lipid (g/kg)	3–4	3–4	2–3	2–3	2–3

The daily nutritional requirements are influenced by age, physical activity and underlying diseases/disorders, e.g., burns, liver failure, etc.

23.4 POST CARDIAC-ARREST SYNDROME

See Chapter 11: Emergencies and Trauma, section 1.1.5: Post resuscitation care.

23.5 FLUIDS IN ICU

- Fluids should be thought of as any other drug/medication in ICU; an under-resuscitated child as well as an overloaded child both have deleterious effects.
- Therefore, fluid therapy prescriptions must be tailored and appropriate for each child's clinical condition.
- Fluids need to be considered under:
  - Resuscitation – need for bolus therapy: 10–20 mL/kg, rapid administration, isotonic crystalloids.
  - Rehydration – slower administration with regular re-evaluation, decreasing infusion as the child improves.
  - Maintenance – restricted due to general lower requirements when in ICU.
  - Ongoing losses – the need to add additional specific fluids, e.g.: burns, DKA, cerebral salt wasting, dehydrating diarrhoeas, bowel fistulae etc.
- As soon as a child is able to feed enterally, a de-escalation approach to stopping all IV fluids as soon as possible and aiming for all fluids and feeds to be given enterally (orally or via gastric tubes).
- Critically ill children have important considerations specific to fluid prescriptions:
  - Sedated and ventilated.
  - Humidified ventilator circuits.
  - Increased ADH production (SIADH).
  - Overhead warmers for small infants.
  - Decreased energy expenditure.
  - Oliguria and anuria.
- These result in fewer insensible losses compared to normal children.
- In particular groups, i.e. head injuries, those with increased ADH (pneumonia, intracranial infection), and post-operative patients, this may be 60–80% of the maintenance volume outlined by the formulae below.
- Fluids should, therefore, be adjusted accordingly, on a patient-by-patient basis.
- **There must be a very good reason why you are giving crystalloid instead of nutritive enteral feeds to any paediatric ICU patient.**
- **Maintenance fluids must be isotonic crystalloids, (balanced solutions or 0.9% saline) with added dextrose.**
- **Note**: **The ideal maintenance fluid is enteral feed.**

Maintenance fluid requirements in ill children:

Age	Fluid requirements
1–3 months	100–120 mL/kg/day
3–12 months	80–100 mL/kg/day
1–5 years	70–80 mL/kg/day
5–12 years	40–70 mL/kg/day

Alternatively, for critically ill children: fluid dosage needs to be 80% of the below 4/2/1 rule.

1st 10 kg	100 mL/kg/24 hours	4 mL/kg/hour
2nd 10 kg	50 mL/kg/24 hours	2 mL/kg/hour
Each next 1 kg	20 mL/kg/24 hours	1 mL/kg/hour

Composition of commonly used crystalloid (mmol/L):

	Ringers Lactate	NaCl (0.9%)	RHS (0.45%NaCl/5% dextrose)	0.45% NaCl
Na	130	154	77	77
Cl	109	154	77	77
K	4	0	0	0
Mg	0	0	0	0
Bicarb.	0	0	0	0
Lactate	28	0	0	0
Glucose	0	0	5 g	0
Ca	0	0	0	0
Osmol.	272	308	406	154
Tonicity (approx. 275–295)	272 Isotonic	308 Isotonic	154 **Hypotonic**	154 **Hypotonic**

23.6 ELECTROLYTE ABNORMALITIES

23.6.1 DYSNATRAEMIAS IN ICU

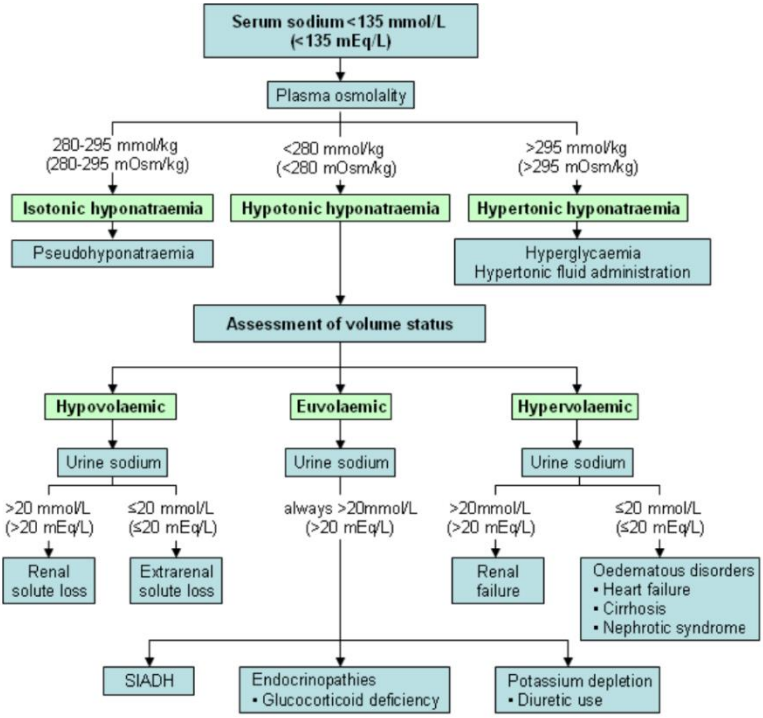
» Rapid changes in serum sodium are more likely to be symptomatic.
» Slow changes should be corrected slowly:
  > Hypernatremia – risk of cerebral oedema.
  > Hyponatremia – risk of central pontine myelinolysis.

Hyponatraemia
Defined as serum Na < 135 mmol/L

» Disorder of water balance, excess total body water relative to electrolytes.
» Caused by increased renal water reabsorption in response to ADH, along with water intake.
» Loss of sodium is minor compared to gain in water in most types of hyponatraemia.
» Hypotonic hyponatraemia is the commonest type.

Hyponatraemia may be classified according to the serum tonicity as being hypertonic, isotonic, or hypotonic:

» **Hypertonic hyponatraemia,** also known as redistributive hyponatraemia, occurs when the presence of excess levels of an osmolyte such as glucose or mannitol causes water to shift from the intracellular to the extracellular compartment, diluting extracellular sodium.
» **Isotonic hyponatraemia,** is an artefact caused by high lipid or protein levels and is usually called pseudohyponatraemia.
» **Hypotonic hyponatraemia,** encompasses all other causes of hyponatraemia and is classified as hypovolaemic, euvolaemic, or hypervolaemic.



** Aetiological diagnosis algorithm of hyponatraemia.*[1]

Hypovolaemic hypotonic hyponatraemia

» U_{Na} > 20 mmol/L: Renal salt losses occur due to diuretics (especially thiazides), salt wasting nephropathy, cerebral salt wasting syndrome and mineralocorticoid deficiency.
» U_{Na} < 20 mmol/L: Non-renal salt losses, usually GIT losses, e.g., vomiting and diarrhoea, burns, pancreatitis and severe hypoalbuminaemia resulting in third spacing of fluid.

Euvolaemic hypotonic hyponatraemia

» U_{Na} > 20 mmol/L: High fluid intake, primary polydipsia, intense exercise, iatrogenic.
» SIADH: Malignancy, CNS disorders and pulmonary disease.
» Urine osmolality > 300 mmol/kg.

Hypervolaemic hypotonic hyponatraemia

» Cardiac failure, cirrhotic liver, nephrotic syndrome.

Corrected sodium = measured sodium + (0.3 x (glucose - 5.6)) [all units in mmol/L]

Criteria for Diagnosing SIADH:

1. No drugs being administered which may mimic the condition.
2. Normal endocrine function (thyroid, adrenal).
3. No volume overload
4. Hyponatremia
5. Low serum osmolality (< 270 mosm/kg).
6. Inappropriately concentrated urine (> 150 mosm/kg).
7. Urine Na > 20 mmol/L.
8. Corrects with fluid restriction.

Management of hyponatremia:

Also see Chapter 2: Alimentary Tract, section 2.2.4: Diarrhoea, acute: metabolic disturbances.

1. Depends on the cause (see algorithm below).
2. Pseudohyponatraemia does not need treatment.
3. SIADH should be fluid restricted.
4. Sodium deficit requires replacement (e.g. renal/cerebral salt wasting, vomiting/diarrhoea).

Calculating sodium deficit:

(Na desired - Na actual) x weight x 0.6 = mmol Na required.

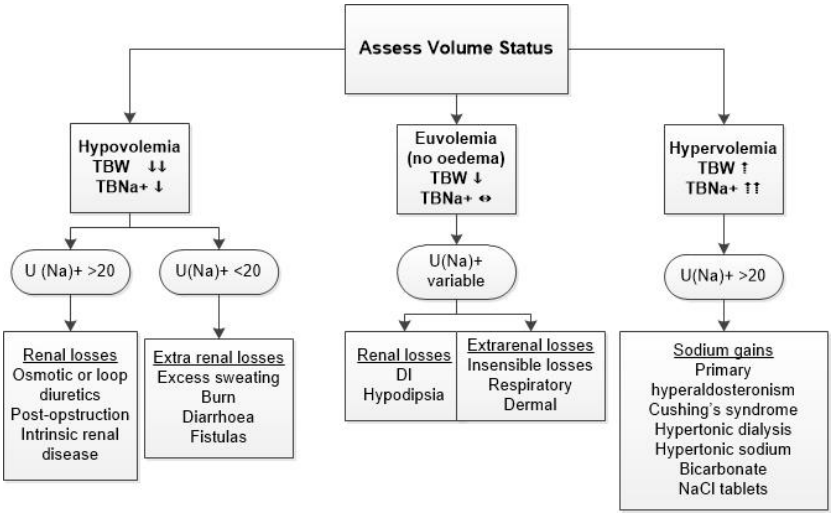
Serum sodium should not increase by greater than 10–12 mmol/L in a 24 hour period.

8.5% $NaHCO_3$: 1 mmol Na/mL
5% NaCl: 0.85 mmol Na/mL
3% NaCl: 0.5 mmol Na/mL
0.9% NaCl: 0.15 mmol Na/mL

Hypernatraemia

Defined as serum Na > 145 mmol/L.

See Chapter 2: Alimentary Tract, section 2.2.4: Diarrhoea, acute: metabolic disturbances.



Hypervolaemic hypernatraemia

» Administration of bicarbonate, hypertonic saline and other fluids containing excess solute relative to free-water.

Hypovolaemic hypernatraemia

» Conditions that cause excessive amounts of free-water losses via the kidneys include central and nephrogenic diabetes insipidus (chronic kidney disease and nephrogenic diabetes insipidus (NDI), respectively).
» Acquired causes of NDI include amphotericin, lithium, hypokalaemia and hypercalcaemia. Mannitol and hyperglycaemia – osmotic diuresis.
» Non-renal free-water losses – from the GIT via suctioning, diarrhoea, vomiting; skin (drains, burns, hyperthermia), and from the respiratory tract in intubated patients.

Management of hypernatraemia:
In children, the two most common aetiologies are diabetes insipidus and hypernatraemic gastroenteritis.

1. Diabetes insipidus requires extra fluid and desmopressin (if central). See Chapter 7: Endocrine System, section 7.4: Diabetes insipidus.
2. Hypernatraemic dehydration treatment:
   a. Calculate free-water deficit and replace, aiming to decrease sodium slowly (maximum 10–15 mmol/L/24 hours). May, therefore, need to administer free-water deficit over 24 to 72 hours (depending on severity).
   b. Oral 'tap water' preferable, otherwise D5W, IV, (taking note of increased glucose delivery).
3. Stop offending drugs.
4. Replace excessive volume losses with fluid containing equivalent electrolyte concentrations (drains, fistulae, renal).

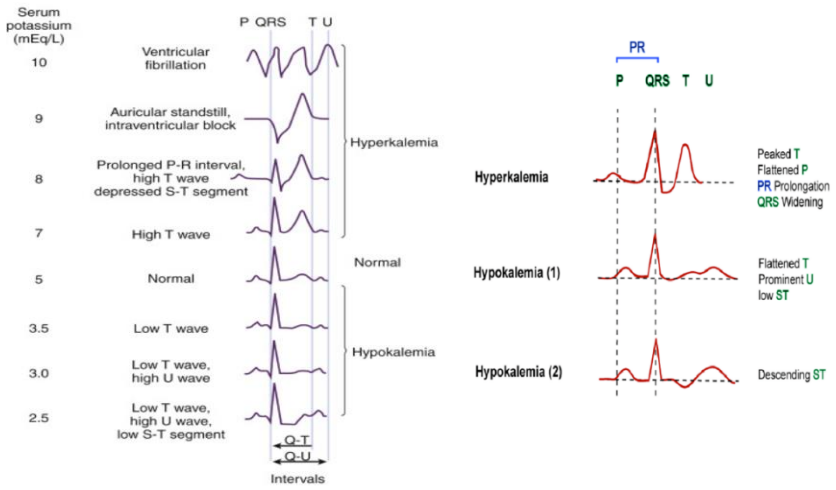
Water deficit calculation:

Water deficit = (current Na/140 -1) x 0.6 x weight (kg) = total litres of water required to normalize sodium.

Amount to be administered per 24 hours is total water deficit divided by planned duration of correction (in days).

23.6.2 POTASSIUM ABNORMALITIES IN ICU

Rapid changes in serum potassium result in the critical clinical features, mainly cardiac and therefore, absolute values may not reflect commonly described clinical changes associated with absolute levels, e.g.: children with chronic renal failure may tolerate very high levels of potassium with little clinical effect.



Hypokalaemia:
See Chapter 2: Alimentary Tract, section 2.2.4: Diarrhoea, acute: metabolic disturbances.

Normal potassium = 3.5–4.5 mmol/L.
Replacement of potassium in ICU:
If potassium 2.5–3.4 mmol/L – replace orally – 1 mmol/kg per dose.

- Potassium chloride tablets (600 mg) = 8 mmol.
- Mist. potassium citrate 30% suspension = 2.8 mmol/mL.

If potassium **< 2.5 mmol/L**, will need intravenous replacement.

»	IV potassium only to be used where appropriate monitoring is available which must include continuous ECG and bedside serum potassium/blood gas analysis.
»	Ensure slow administration, over 4 hours.

Always discuss with a specialist first before commencing any IV potassium.

- Replacement dose = 1–2 mmol/kg (given slowly over 4 hours).
- Maximum rate of replacement = 0.5 mmol/kg/hour.
- **Recommended dose = 1.2 mmol/kg = 0.3 mmol/kg/hour for 4 hours.**
- 15% KPO_4 and 15% KCL both contain 2 mmol/mL of potassium.
- ECG monitoring is strongly recommended during IV potassium replacement.

Example of IV replacement in a 10 kg child:
Dose = 1.2 mmol x 10 kg = 12 mmol of potassium.
= 6 mL of 15% potassium solution.
Recommended: Add the 6 mL of potassium (either KCl or KPO_4 - depending on the patient's clinical characteristics) to 14 mL of 0.9% saline, to create a 20 mL solution. Then administer the solution at 5 mL/hour over 4 hours.

Please note that this only holds true for a child who is not receiving any additional potassium intravenously.

Note: In stable patients with severe hypokalaemia, slow correction with oral potassium supplementation can be considered in non-ICU environments.

Acute hyperkalaemia in ICU:
Hyperkalaemia: K > 5.5 mmol/L.
Moderate hyperkalaemia: K > 6 mmol/L.
Severe hyperkalaemia: K > 7 mmol/L.

Management of hyperkalaemia:
» Stop any potassium containing fluids/supplemental potassium.
» Treat the cause.
» Treatment of hyperkalaemia:
- Calcium gluconate 0.6 mL/kg diluted 1:4, IV, slowly.

OR if life threatening hyperkalaemia, consider:
- Calcium chloride (10%) 0.2 mL/kg (maximum 10 mL) diluted 1:4 with sterile water, IV, slowly over 10 minutes **(must be administered via a CVC line)**.
- Salbutamol nebulisation or salbutamol 4 mcg/kg, IV, over 20 minutes. (Note – ineffective in severe metabolic acidosis.)
- Sodium bicarbonate 8.5% 1 mL/kg diluted 1:1 with sterile water, IV. (Note – more effective with severe acidosis of pH < 7.15.)
- Dextrose/insulin: 1 mL/kg of 50% dextrose + 1 mL/kg of sterile water + 0.1 units/kg rapid acting insulin. (this concentration is only suitable for administration via a central line, use a 10% solution via peripheral lines)

23.6.3 MAGNESIUM ABNORMALITIES IN ICU

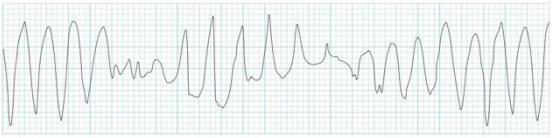
40–60% of ICU patients have hypomagnesaemia.

Hypomagnesaemia:
Clinical effects of hypomagnesaemia:
» Electrolyte abnormalities:
  > Hypokalaemia
  > Hypocalcaemia

- Hypophosphataemia

» Arrhythmias:
- Prolonged QT interval.
- Polymorphic VT – Torsade de pointes.

» Neurologic:
- Altered mentation, generalized seizures, tremors, hyper-reflexia, weakness.



Torsade de pointes

Magnesium supplementation:
IV: Ampoules of 2 g in 1 mL (50% magnesium sulphate solution).

Replacement dose:
- Magnesium sulphate, 25–50 mg/kg, IV.
  - Maximum rate of IV administration: 1 g/7 minutes.

Life threatening hypomagnesaemia – Torsade de pointes:
1. Infuse 50 mg/kg $MgSO_4$, IV, over 2 to 5 minutes.
2. Follow with 25–50 mg/kg $MgSO_4$ in 250–500 mL N/S over the next 6 hours (maximum 5 g).
3. Continue with 25–50 mg/kg $MgSO_4$ every 12 hourly for 5 days (Note – to replenish total body stores).

Hypermagnesaemia:
» Serum magnesium > 2 mEq/L.
» Usually in patients with renal insufficiency and haemolysis, closely associated with increased potassium and low calcium.

Clinical effects of hypermagnesaemia:
Serious effects of hypomagnesaemia are due to calcium antagonism in the cardiovascular system:
» Weakness/hyporeflexia/paralysis > 4 mEq/L.
» 1st degree AV block > 5 mEq/L.
» Complete heart block > 10 mEq/L.
» Cardiac arrest > 13 mEq/L.

Treatment of hypermagnesaemia:
» Stop extra oral/IV intake.
» In patients with normal renal function, will correct once supplemental intake is stopped.

- IV calcium gluconate (0.6 mL/kg over 2–3 minutes) can be used for hypotension and respiratory depression (only transient effect).

» Haemodialysis for severe cases with significant cardiac signs.

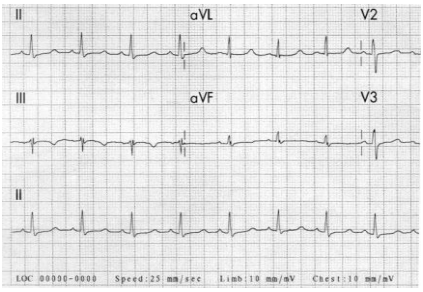
23.6.4 CALCIUM ABNORMALITIES IN ICU

Effects of albumin and pH
Alkalosis results in increased bound calcium – and decreased ionized calcium.
In hypoalbuminaemia, total serum calcium measurement may be falsely low.

Corrected calcium = measured calcium + (40 - albumin) x 0.025.

Hypocalcaemia:
See Chapter 7: Endocrine System, section 7.8: Hypocalcaemia in children.

In ICU, the focus is on low ionized calcium concentrations (< 1.0 mmol/L).
Clinical effects of hypocalcaemia:
» Tetany
» Seizures, confusion.
» Laryngospasm
» Cardiac arrhythmias (heart block and ventricular tachycardia).
» Prolonged QTc interval.



Management of hypocalcaemia in ICU:
1. Check magnesium, replace if low.
2. Replace calcium, IV:
   - 10% calcium gluconate 0.5 mL/kg/dose.
   - 10% calcium chloride 0.2 mL/kg/dose (must use a central line).
3. IV replacement targeted to the ionized calcium value on blood gas – > 1.2 mmol/L.

Intravenous calcium replacement in ICU:
» Replacement dose = 0.1 mmol/kg/dose (for seizures/arrhythmias – infusion 0.1 mmol/kg/hour after initial IV slow bolus).

- Calcium gluconate 10%:
  - 1 ampoule (10 mL) = 1 g = 2.2 mmol = 0.22 mmol/mL.
  - 0.6 mL/kg/dose (maximum 10 mL).
  - Mix 1:4 with sterile water and inject IV, slowly over 10 minutes.

OR if severe or refractory:
- Calcium chloride10%:
  - 1 ampoule (10 mL) = 1 g = 7 mmol = 0.7 mmol/mL.
  - 0.2 mL/kg/dose (maximum 10 mL).
  - Mix 1:4 with sterile water and inject IV, slowly over 10 minutes.
  - **Must be administered via a CVC**

Note:
Monitor IV sites for extravasation, never mix with fluids containing bicarbonate or phosphate, never give IM or SC.

Hypercalcaemia:
» Key principle: Effective management of hypercalcaemia begins with adequate rehydration and treating the underlying cause.
» Refer to a specialist for consideration of furosemide and/or haemodialysis in the setting of severe renal failure.

23.6.5 PHOSPHATE ABNORMALITIES IN ICU

Hypophosphataemia:
» Incidence is 60–80% of septic ICU patients.
» Mortality increases 30% in patients with severe hypophosphataemia (< 0.32 mmol/L).

Clinical presentation of hypophosphataemia can include:
» Confusion, coma, seizure.
» CCF
» Respiratory failure.
» Shift of Hb-dissociation curve left (hypoxia).
» Haemolysis, rhabdomyolysis.

Treatment of hypophosphataemia:
Intravenous replacement is recommended for severe hypophosphataemia.

- Potassium phosphate: 1 mmol/mL phosphate and 2 mmol/mL potassium.
  - 0.15–0.6 mmol/kg/day, IV, over 6 hours.
  - Dilute to ratio 1:4 (potassium phosphate: 0.9% sodium chloride).
  - Repeat at 6 hourly intervals until phosphate > 0.6 mmol/L.

Note:
Rate limiting fact will usually be potassium concentration/rate.

  - Example: 20 kg child:

0.15 x 20 kg = 3 mmol/day = 3 mL $KP0_4$.
i.e. add 3 mL $KP0_4$ to 12 mL normal saline over 6 hours (3 mL/hour).
= 0.05 mmol/kg/hour potassium.
= 0.025 mmol/kg/hour phosphate.

Hyperphosphataemia:

Clinical presentation of hyperphosphataemia:
» Decreased mental status.
» Seizures (secondary to hypocalcaemia).
» Weakness, tetany (secondary to hypocalcaemia).
» ECG changes: prolonged QT, polymorphic VT, Torsade de pointes (secondary to hypocalcaemia).
» Anorexia, nausea, vomiting.
» Soft tissue deposition of calcium-phosphate product – renal dysfunction.
» Metabolic acidosis, hypomagnesaemia.

Management of hyperphosphataemia:
1. Treat the underlying cause.
2. Fluid administration (rhabdomyolysis/tumor lysis).
3. Reduce phosphate intake.
4. Phosphate binders (calcium carbonate).
5. Refer and consult with specialist for consideration for haemodialysis.

23.6.6 HYPERGLYCAEMIA

R73.9

DESCRIPTION

Critical illness is a state of metabolic stress associated with elevated serum catecholamines and is often associated with the use of continuous feeding. Hence the target blood glucose is 4–10 mmol/L. Serum glucose > 10 mmol/L is common, occurring in up to 20% of critically ill children.

Clinical significance

» Increased morbidity and mortality.
» Osmotic diuresis – fluid and electrolyte abnormalities.
» Increased infections and sepsis.
» Poorer neurological outcomes.

Monitoring

» Routine blood glucose monitoring: 4 to 6 hourly.
» Urine dipstix to check for glycosuria.
» If the patient is requiring insulin then hourly monitoring until blood glucose < 10 mmol/L.

Management

» Tight glycaemic control (maintenance of blood glucose between 4.4–6.1 mmol/L) is NOT recommended.
» Look for and treat any potential underlying cause, e.g. sepsis, pain, steroid treatment etc.
» If HGT is > 10 mmol/L for 2 consecutive readings (60 minutes apart) or for 1 reading in the presence of glycosuria, then give a stat dose of IV short acting insulin 0.05 units/kg.
» Calculate glucose delivery. If glucose delivery > age-appropriate range, reduce glucose delivery to within normal range and monitor HGTs.
» If a second dose of IV insulin is required within a 4-hour period, then seek advice from a paediatric endocrinologist.
» Insulin infusions are not routinely recommended.

23.6.7 HYPOGLYCAEMIA

The target blood glucose of critically ill children is 4–10 mmol/L. It is essential to check the blood glucose of all critically ill children as hypoglycaemia (< 3.5 mmol/L) is an emergency. Immediate treatment is with an IV glucose bolus and depends on the nature of the available IV access.

- Peripheral line: 5 mL/kg of a 10% dextrose solution.
- Central line: 2 mL/kg of a 25% dextrose solution.

The bolus will need to be repeated until the blood glucose ≥ 4.0 mmol/L.

For additional information see Chapter 7: Endocrine System, section 7.6: Hypoglycaemia in children.

23.6.8 DIABETIC KETOACIDOSIS

See Chapter 7: Endocrine System, section 7.5.2.2: Diabetic ketoacidosis

23.7 TRAUMATIC BRAIN INJURY (TBI) AND NEURO-PROTECTION IN THE ICU

S06.2

DEFINITION

Traumatic brain injury (TBI) is a form of Acquired Brain Injury. It is a non-degenerative, non-congenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions, with an associated diminished or altered state of consciousness. Severity of the initial insult is based clinically on the Glascow Coma Scale (GCS): Severe = GCS < 9.

Patients should be referred and managed in a specialised centre.

Summarized therapeutic goals – strict avoidance of the 5 'H's.

» Hypoxaemia
» Hypotension
» Hyperthermia
» Hypoglycaemia
» Hypercarbia

MEDICINE TREATMENT

Principles for neuroprotection in 1st 24–48 hours.

1. Airway & Ventilation

1. Secure the airway.
2. Optimise oxygenation – Sats > 94% but < 100%.
3. Use PEEP cautiously – keep PEEP < 10 cmH_2O to prevent inhibited venous return – individualise according to patient response. NB: Hypoxia kills neurons.
4. P_aCO_2 manipulation
   » Target P_aCO_2 35–40 mmHg (4.7-6 kPa).
   » Avoid prophylactic hyperventilation P_aCO_2 < 35 mmHg.

2. Haemodynamics

Prevent hypotension

1. Ensure adequate Cerebral Perfusion Pressure (CPP).
2. CPP = MAP - ICP
   a. Therefore, placing an ICP monitoring catheter is mandatory, if available.
   b. ICP directed therapy – target MAP to a CPP of 40–50 mmHg.
3. If no ICP catheter – target MAP = 65 + 1.5 x age (years).
4. The use of inotropes and vasopressors should be considered early if needed to achieve the target CPP level.

Fluids

1. Optimal fluid resuscitation to ensure normal circulating blood volume.
2. Use isotonic fluids in patients with raised ICP for fluid resuscitation, e.g. Ringers lactate, normal saline.
3. Avoid hypo-osmolar and dextrose containing IV fluids, e.g. 5% dextrose water, ½ normal saline, in the resuscitation phase of care (see fluid contents table).

Corticosteroids

- Available evidence does not support the use of corticosteroid.

3. Ongoing management

Analgosedation

Goal is to avoid ICP spikes from restlessness and irritability, especially if ventilated; avoid ventilator/patient dyssynchrony.

Objective measures for analgesia and sedation should be used, e.g.: RASS and Comfort score targets.

» Provide adequate sedation and optimal pain relief to avoid anxiety and pain – helps decrease ICP. See Chapter 20: Pain Control.
» Morphine/fentanyl and midazolam are the mainstay in the first 24 hours.
» Titrations or infusions are acceptable.
» Paracetamol decreases the need for opioids by 30% and is recommended as initial therapy.
» Avoid NSAID in the 1st 24–48 hours – impaired platelet function may aggravate intracranial bleeding.
» If above steps are inadequate, consider ketamine and propofol (note – haemodynamic instability may occur and require vasopressor support).
» Neuromuscular blocking agents are not indicated as routine therapy.

Seizure Prophylaxis

Phenytoin can be considered in patients with severe TBI to reduce the incidence of early onset (< 7 days) post traumatic seizures.

- Phenytoin, IV, 20 mg/kg over 20 minutes (loading dose) followed by 5–10 mg/kg/day in 3 divided doses (maintenance).

Positioning and care

1. Optimise venous drainage from the brain:
   » Keep head in neutral position with neck in mid-line.
   » Elevated head of bed – 30 degrees.
   » Remove cervical collars as soon as possible, current prehospital guidelines – hard cervical collar usage is contraindicated.
2. Limit handling when possible.
3. Tracheal suctioning:

- » Protection of the brain takes precedence over protection of the lungs.
- » Avoid routine suctioning – a potent elevator of ICP.
- » Suction only if secretions are visible and causing ventilation problems.
- » Do not insert the catheter beyond the end of the ETT or tracheostomy tube.
- » Provide analgosedation and consider neuromuscular blockade with a non-depolarising agent (cisatracurium, vecuronium or rocuronium) when suctioning patients with increased ICP.

4. Relieve Abdominal Compartment Syndrome (ACS) if present, as abdominal hypertension increases ICP.

Temperature

- » Maintain normothermia – 36.0–37.5 °C.
- » Hyperthermia > 38 °C must be avoided.
- » Hypothermia – cooling reduces the cerebral metabolic rate of oxygen consumption but published data are controversial.
  Targeted temperature management is recommended after prolonged cardiac arrest followed by return of spontaneous circulation.

Feeds

- » Patients with TBI are frequently hyper-metabolic and hyper-catabolic.
- » Initiation of early enteral nutrition within 72 hours from time of injury is indicated. Build feeds slowly as tolerated to achieve full enteral nutrition. Avoid nasogastric tubes if a base of skull fracture is suspected.

Stress-ulcer prophylaxis

- » Patients with raised ICP are at high risk for stress ulceration.
- » Consider prophylaxis with a proton pump inhibitor.
- » Stop prophylaxis once feeds have been established.

Glucose

- » Aim for glucose levels 4–10 mmol/L.

Family Information

- » Allow family contact as this helps calm and reassure the patient.
- » Prepare family with advice on infection control and ICU practices.
- » Commence counseling early in the patient's stay – regarding high risk of short-term adverse outcomes in patients with TBI and raised ICP requiring ICU, and the likelihood of protracted recovery and permanent disability even in survivors of initial ICU stay.

4. Acute management of raised ICP

» Controlled hyperventilation ($PaCO_2$ no less than 30 mmHg – risk cerebral vasoconstriction and induced ischaemia) only transiently for management of very acute and serious elevation of intracranial pressure.
» Osmotic therapy for children with suspected raised ICP (avoid use if sodium > 160 mmol/L).

Hypertonic saline should be considered for acute treatment of TBI with raised ICP (> 20 mmHg).

- Sodium chloride 5%, IV, 3 mL/kg as a stat dose over 20 minutes.
- Continuous infusion of sodium chloride 3%, IV, 0.1–1 mL/kg/hour, titrated to maintain ICP < 20 mmHg).

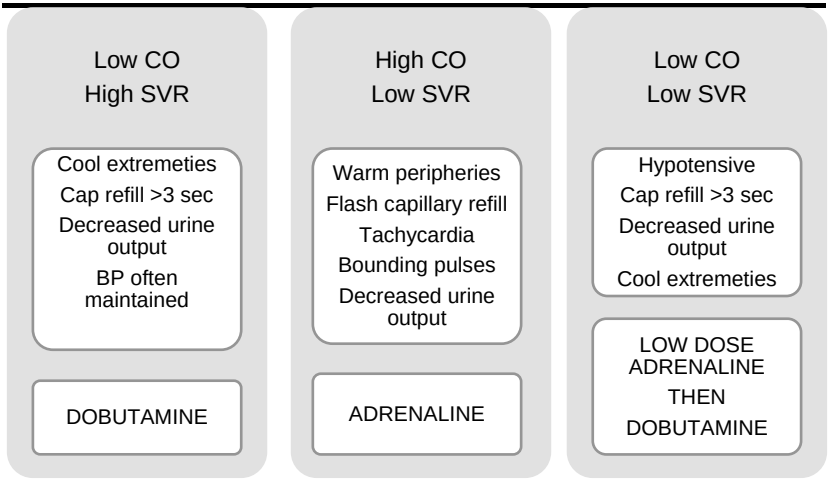
Mannitol can also be used for the treatment of raised ICP, although hypertonic saline has a better efficacy and side effect profile.

- Mannitol, IV, 0.5–1 g/kg over 10 minutes, may be repeated 6 hourly.

» Urgent neurosurgical consultation if raised ICP is refractory to medical management.
» High dose barbiturate therapy in refractory intracranial hypertension.

23.8 INOTROPES AND VASOPRESSORS

» Choice depends on desired effect.
» There is no 'one size fits all' strategy for inotrope/vasopressor choice.
» Each agent has dose-dependent effects on inotrope (improved contractility) or vasopressor effect (increasing the SVR).
» Important components of the cardiovascular system:
  > Preload – is the child pre-load replete (not overfilled and not under-filled) – fluid therapy.
  > Contractility – does the heart need support to pump effectively – inotropic support.
  > Afterload – how vasoconstricted or vasodilated is the child – need for vasopressor or vasodilator support.
» Septic shock patients may present in any of the following ways, and may change clinical appearance from hour to hour:



Paediatric cardiovascular considerations:

» Reduced myocardial mass means that the myocardium is limited in its ability to increase size and contractility, therefore, the main way that children are able to increase CO is by increasing HR, not SV.
» Children start at higher baseline heart rates, therefore, they are limited by how much they are able to increase HR and, therefore, CO.
» Children maintain blood pressure by increasing SVR (systemic vascular resistance). A drop in blood pressure is a late sign in shocked children.
» Vagal parasympathetic tone is most prominent, therefore, they are more prone to bradycardia.

Drug	Dosage	A1	B1	B2	DOPA	Side effects	Indications
Adrenaline	0.01–1 mcg/kg/min	++ ++ +	+++ +	+++	N/A	Ventricular arrhythmias -Cardiac ischaemia -Hyper-tension -Sudden cardiac death -Lactic acidosis -Tissue ischaemia	-Shock (vasodilatory, cardiogenic) Note: Broncho-spasm – SC adrenaline Anaphylaxis – IM adrenaline initially: (0.15 mg < 6 years, 0.3 mg > 6 years)
Dopamine	2–20 mcg/kg/min	++ +	+++ +	++	+++ ++	-Hyper-tension -Ventricular arrhythmia	-Cardiogenic shock -Heart failure

						-Cardiac ischaemia -Tissue ischaemia/ gangrene	-Symptomatic bradycardia unresponsive to atropine
Dobutamine	2–20 mcg/kg/min	+	+++ ++	+++	N/A	-Tachy-cardia -Ventricular arrhythmia -Cardiac ischaemia -Hyper-tension -Hypo-tension	-Low CO, shock -Symptomatic bradycardia unresponsive to atropine
Phenylephrine	0.4–9 mcg/kg/min	++ ++ +	0	0	N/A	-Reflex bradycardia -Hyper-tension -Peripheral vaso-constriction -Tissue necrosis	-Hypotension (vagally mediated) -Increase afterload in Tetralogy of Fallot -Decrease LVOT gradient in HCM -Increase MAP in AS with hypo-tension - Hypotension in spinal shock (**Not for use in septic shock)**

Concentrations and formula

Vasopressors and Inotropes	Recommended concentration	Formula to calculate rate (mL/hour)
Adrenaline	100 mcg/mL (5 mg in 50 mL normal saline)	$\frac{\text{mcg/kg/min x weight x 60}}{100}$
Dobutamine	2500 mcg/mL (125 mg in 50 mL normal saline)	$\frac{\text{mcg/kg/min x weight x 60}}{2500}$
Dopamine	1000 mcg/mL (50 mg in 500 mL normal saline)	$\frac{\text{mcg/kg/min x weight x 60}}{1000}$
Phenylephrine	100 mcg/mL (5 mg in 50 mL normal saline)	$\frac{\text{mcg/kg/min x weight x 60}}{100}$

5% Dextrose water to be considered for use in patients where sodium needs to be avoided (e.g. nephrotic syndrome).

Additional medicine therapies:

- Corticosteroids:
  - Limit adrenoceptor down-regulation.
  - The role of adjunctive hydrocortisone in catecholamine refractory septic shock is unclear. Use is currently not supported by high-level evidence.
- Calcium:
  - Use in the setting of hypocalcaemia and hypotension.
  - Side effects: cardiac myocyte apoptosis, over constriction, arrhythmias.
    - Infusion dose: 0.5–2.0 mmol/kg/day = 0.02–0.08 mmol/kg/hour. Mix 2 mmol/kg of 10% calcium chloride to make a total volume of 100 mL to run at 1–4 mL/hour (0.02–0.08 mmol/kg/hour).

    Example: 8 kg child: 2 mmol/kg = 2 mmol x 8 = 16 mmol.
    10% calcium chloride is 7 mmol/10 mL.
    16 mmol, therefore = 22.8 mL of 10% calcium chloride.
    Mix this with 77 mL of normal saline to make a total volume of 100 mL.
    Run at 1–4 mL/hour (0.02–0.08 mmol/kg/hour).

Pearls:

» There is more to fixing shock than fixing the blood pressure.
» All catecholamines alter immune response.
» All catecholamines have short half-lives, therefore, achieve your goal quickly at the bedside and titrate to targeted goals.
» Steady state is achieved after 5–10 minutes.
» **Start** administering inotrope/vasopressor through a peripheral line but **switching to a central venous line** is paramount as soon as possible.
» Cardiac output monitoring is the most accurate way to assess the cardiovascular system. Consider in the patient with refractory shock – therefore, the need for possible referral to a tertiary ICU.
» Make sure your patient is adequately 'filled' before starting a vasopressor.
» Do not leave the bedside until the patient has improved perfusion markers – improved LOC, adequate urine output, improved skin perfusion, HR normalizing and appropriate BP.

23.9 VENOUS THROMBO-EMBOLISM (VTE)

23.9.1 THROMBOPROPHYLAXIS IN ICU

The incidence of venous thrombosis is likely underappreciated in critically ill children. All children with at least one organ failure and central venous access likely require pharmacological prophylaxis against venous thrombo-embolism.

Drug	Dose	Comments
Low molecular weight heparin (LMWH), e.g. enoxaparin.	< 2 months of age: 0.75 mg/kg/dose, SC, 12 hourly. > 2 months of age: 0.5 mg/kg/dose, SC, 12 hourly.	Avoid with renal insufficiency. Monitoring: 0.2–0.4 anti-Xa U/mL (sample must be drawn in a non-heparinised syringe, 3–4 hours post dose).
Unfractionated heparin (UFH).	10 units/kg/hour, IV, as a continuous infusion.	Not for routine use. Can be used in children with renal insufficiency, those requiring surgery or if they have a high risk of bleeding.

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23.9.2 TREATMENT OF VTE

The duration of treatment (anticoagulation) depends on the cause of VTE: 3 months for CVC-related or secondary VTE and 6 months for idiopathic or recurrent VTE. Initial anticoagulation with LMWH or UFH for 5–10 days followed by maintenance warfarin or LMWH. Routine use of thrombolytic therapy is not recommended unless there is a major vessel occlusion. Protamine sulphate can be used to reverse the effects of both LMWH and UFH.

Drug	Dose	Comments
Low Molecular Weight Heparin (LMWH), e.g. enoxaparin.	< 2 months of age: 1.5 mg/kg/dose, SC, 12 hourly. > 2 months of age: 1 mg/kg/dose, SC, 12 hourly.	Avoid with renal insufficiency. Monitoring: 0.5–1.0 anti-Xa U/mL (sample must be drawn in a non-heparinised syringe, 3–4 hours post dose).
Unfractionated Heparin (UFH).	Bolus: 75–100 units/kg, IV, followed by 20 units/kg/hour, IV, as a continuous infusion.	Monitoring: PTT 2–3 times baseline value (60–85 seconds). Check platelet count (heparin induced thrombocytopenia is an infrequent complication).
Warfarin	0.1 mg/kg (maximum 10 mg on day 1).	Must follow treatment with a heparin. Dose can be increased by 0.5 mg per dose, in response to INR (target 2.0–3.0). Heparin is discontinued when warfarin produces target INR for 2

		consecutive days. Monitoring: INR at least 2–4 weekly.

23.10 ICU MEDICATIONS

Drugs given in ICU are often diluted and given as an infusion. It is essential that one considers intravenous fluids to be drugs and as such need to consider compatibility with other medications, role of ambient light, temperature and stability of drugs once diluted, characteristics of diluents and the physical characteristics of the infusion set.

Agent	Compos-sition	Pharmaco-logical action/ Indications	Route of admin.	Dose	Compatible fluids	Incompatible fluids/drugs
Adrenaline (epinephrine)	1 mg/mL ampule, clear fluid.	Vasopressor, inotrope, shock, anaphylaxis, cardiac arrest, upper airway obstruction.	Continuous IV infusion via a central line; IM, for ana-phylaxis; nebulized for upper airway obstruction.	0.05–1 mcg/kg/min, IV, (titrated to effect). 0.01 mg/kg, IV, for cardiac arrest. 0.01 mg/kg (maximum 0.5 mg), IM, for anaphylaxis. 2–5 mL (1 mg/mL) for nebulization.	Normal saline 0.9%, 5% dextrose saline, 5% dextrose water.	
Amiodarone	150 mg/ 3 mL (50 mg/mL), clear pale yellow fluid.	Specific tachy-dysrhythmias (refractory SVTs and VTs).	IV infusion, preferably via a central line	Emergency (VT/VF): 5 mg/kg, IV, bolus over 3 minutes (maximum 300 mg bolus dose). Other arrhythmias: Initial dose: 5 mg/kg (150 mg + 7 mL of 5% dextrose water = 10 mL, so 15 mg/mL) over 20–60 minutes; maximum 300 mg. Infusion: 10 mg/kg over 24 hours with maximum daily dose of 15 mg/kg over 24 hours.	5% dextrose water only.	Normal saline, sodium bicarbonate.

Agent	Compos-sition	Pharmaco-logical action/ Indications	Route of admin.	Dose	Compatible fluids	Incompatible fluids/drugs
Atropine	0.5 mg/mL; 1 mg/mL.	Cholinergic causes of bradycardias, organo-phosphate/ carbonate poisoning.	IV infusion, IV bolus.	Bolus: 0.02 mg/kg Infusions: 1 mg into 9 mL of normal saline giving a concentration of 0.1 mg/mL and the rediluted 1 mL of this solution into 9 mL normal saline giving a concentration of 0.02 mg/mL and give at a rate of 0.012–0.1 mg/kg/hour.		
Dexametha-sone	4 mg/1 mL vial	Allergic and anti-inflammatory conditions. Croup/ Airway oedema. Note: Long-acting steroid (half-life up to 54 hours), 30 x more potent than hydrocortisone.	IV bolus.	0.6 mg/kg, stat. May be followed by 0.15 mg/kg 6 hourly in airway oedema.		
Dobutamine	250 mg/20 m L ampoule, clear fluid.	Inotrope vasodilator (< 7.5 mcg/kg/min) Vasopressor (> 10 mcg/kg/min)	Continuous IV infusion via a central line.	5–20 mcg/kg/min titrated to effect.	Normal saline 0.9%, 5% dextrose water, 5% dextrose saline.	Sodium bicarb, furosemide, heparin, hydrocortisone, penicillin, cefazolin.

Agent	Compos-sition	Pharmaco-logical action/ Indications	Route of admin.	Dose	Compatible fluids	Incompatible fluids/drugs
Furosemide	10 mg/mL in 2 mL/20 mg ampoules or 5 mL/50 mg ampules.	Fluid overload.	IV infusion, IV bolus (Note: IV boluses are associated with hearing loss).	Bolus 0.5–1.5 mg/kg slowly Infusions: 0.05–0.2 mg/kg/hour (higher doses to be used only in consultation with specialists).	Normal saline 0.9%, 5% dextrose saline 5% dextrose water, Ringers lactate.	
Glycopyrrolate	0.2 mg/mL in a 1 mL or 2 mL vial.	Organo-phosphate poisoning in the absence of neurological involvement due to the organo-phosphate (off-label).	IV infusion, IV bolus.	0.01 mg/kg2 mg into 9 mL normal saline resulting in a concentration of 0.02 mg/mL.		
Hydrocortisone	100 mg/ 2 mL (powder). Add 2 mL WFI or normal saline.	Adrenal corticosteroid, Anti-inflammatory agent (glucocorticoid). Refractory septic shock. Adrenal insufficiency. Status asthmaticus. anaphylaxis	IV bolus.	5 mg/kg in acute asthma and anaphylaxis. 50 mg/m^2/day in divided doses for catecholamine refractory septic shock.		

Agent	Compos-sition	Pharmaco-logical action/ Indications	Route of admin.	Dose	Compatible fluids	Incompatible fluids/drugs
IVIG Intravenous polyvalent human immune-globulin.	3 g powder in 100 mL normal saline, 6 g powder in 200 mL normal saline and 12 g powder in 400 mL normal saline (packaged with diluent).	Autoimmune and inflammatory disorders (GBS, Kawasaki, MIS-C, TTP).	IV infusion.	2 g/kg (in total) given over 2–5 days. Depending on indication, seek specialised advice in toxic shock.		
Labetalol	5 mg/mL in 20 mL ampules, clear liquid.	Hypertensive emergency.	IV infusion.	200 mg (2 amps) in 200 mL normal saline = 1 mg/mL.	Normal saline 0.9%, 5% dextrose saline.	Bolus: 0.2–1 mg/kg, IV, maximum 40 mg Infusion: 0.25–3 mg/kg/hour.
Lidocaine (Lignocaine)	10 mg/mL (1%), 20 mg/mL (2%) clear colourless fluid.	Specific ventricular dysrhythmias. Alternative to amiodarone in VF/VT refractory cardiac arrest.	IV bolus or IV infusion.	Bolus:1 mg/kg, with a maximum of 100 mg/dose. Dilute to 1 mg/mL solution and inject at a rate of 25–50 mg/minute. Can be repeated at 5 to 10 minute intervals with a maximum of 3 mg/kg or 300 mg. Infusions: 20–50 mcg/kg/min. If have liver dysfunction, renal dysfunction or persistent poor	Normal saline 0.9%, 5% dextrose. water, 5% dextrose saline.	Normal saline, sodium bicarbonate.

Agent	Compos-sition	Pharmaco-logical action/ Indications	Route of admin.	Dose	Compatible fluids	Incompatible fluids/drugs
				cardiac output, do not exceed 20 mcg/kg/minute.		
Magnesium sulphate	1 g in 2 mL plastic ampoules (2mmol/ml = 4mmol per 2 ml ampoule)	Bronchodilation Hypo-magnesaemia, cardiac arrest from Torsades de pointes, digoxin toxicity. Pre-eclampsia, eclampsia.	Intravenous infusion, intravenous bolus.	Bolus: 20–40 mg/kg, over 20 minutes. Infusions: 10–50 mg/kg/hour. Asthma/Digoxin tachycardia/Pulmonary HT – 50 mg/kg, over 20 min. Polymorphic VT 25–50 mg/kg, over 3–5 min. Magnesium deficiency 100 mg/kg, IV, over 4–6 hours 12 hourly.		
Salbutamol	0.5 mg/mL (500 mcg/ mL)	Hyperkalaemia Bronchodilator	IV bolus, IV infusion,	Bolus: 2–4 mcg/kg over 20 min. Infusion: Dilute 500 mcg into 49 mL of normal saline giving a concentration of 10 mcg/mL. 1–2 mcg/kg/min. Higher doses only in consultation with a specialist.		

References

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