

Age	Wt	Surface area	% of adult dose	Pulse 95% range	Mean BP 95% range
Term	3.5kg	0.23 m ²	12%	95-145	40-60
3 mo	6.0kg	0.31 m ²	15%	110-175	45-75
6 mo	7.5kg	0.38 m ²	20%	110-175	50-90
1 yr	10 kg	0.47 m ²	25%	105-170	50-100
3 yr	14 kg	0.61 m ²	33%	80-140	50-100
7 yr	22 kg	0.86 m ²	50%	70-120	60-90
10 yr	30 kg	1.10 m ²	60%	60-110	60-90
12 yr	38 kg	1.30 m ²	75%	60-100	65-95
14 yr	50 kg	1.50 m ²	80%	60-100	65-95
21 yr	60 kg	1.65 m ²	100%	65-115	65-105
21 yr	70 kg	1.80 m ²	100%	65-115	70-110

Age < 9 yr: wt (kg) approx = (2 x age) + 9

Age > 9 yr: wt (kg) approx = 3 x age

Surf area m² = sq root (Ht cm x Wt kg / 3600)

Age	Wght (kg)	ETT (Micro-cuff)	ETT (Mallinckrodt)		ETT length Microcuff ETT designed to have distal thick line at cords		Suction catheter FG
		Size	Diameter internal (mm)	Diameter external (mm)	At lip (cm)	At nose (cm)	
Newborn	<0.7		2.0	2.9	5.0	6	6
Newborn	<1		2.5+	3.6	5.5	7	6
Newborn	1.0		3.0+	4.3	6	7.5	7
Newborn	2.0		3.0	4.3	7	9	7
Newborn	3.0	3.0	3.0+	4.3	8.5	10.5	7
Newborn	3.5	3.0	3.5+	4.9	9	11	8
3 month	6.0	3.0	3.5	4.9	11	12	8
1 year	10	3.5	4.0	5.6	12	14	8
18 months	11	3.5	4.0	5.6	12	15	8
2 year	12	4.0	4.5	6.2	13	15	8
3 year	14	4.0	4.5	6.2	13	16	8
4 year	16	4.5	5.0	6.9	14	17	10
6 year	20	5.0	5.5	7.5	15	19	10
8 year	24	5.5	6.0	8.2	16	20	10
10 year	30	6.0	6.5	8.9	17	21	12
12 year	38	6.5	7.0	9.5	18	22	12
14 year	50	7.0	7.5	10.2	19	23	12
Adult	60		8.0	10.8	20	24	12
Adult	70		9.0	12.1	21	25	12

Intubated children should have a CXR in the neutral position, jaw midline and the tip of the ETT at vertebrae T2

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INTRODUCTION

This book contains the essential clinical information that registrars need to know to manage the spectrum of patients in a busy paediatric intensive care unit. It is not a text-book, and information on other clinical sciences: pathophysiology, anatomy, pharmacology, biochemistry, genetics, clinical and information technology are available from many other resources. There is often more than one clinical approach to the conditions included in this booklet; we describe a standardised way that is practiced at the Royal Children's Hospital in Melbourne. The diseases and conditions reflect the epidemiology of paediatric critical illness in Victoria, but with just a few exceptions this list is common to most middle and high income settings.

The rapid changes in medicine and health in this century are reflected in intensive care, so some of the information will be out of date in the next few years. However the basics of intensive care – the maintenance of airway, breathing, circulation, prevention of secondary brain injury, meticulous attention to clinical detail, prevention of hospital-acquired infections, minimizing antimicrobial resistance, quality improvement and good communication will always be key skills to learn and priorities on which to focus.

Since the first edition of this booklet in 2003 we now care for more children with chronic and complex underlying diseases; and for many children repeated ICU admissions or needing some ICU-technology at home are realities of life. It remains however that the minimal objective of treating children in ICU should be to restore them to the best possible level of health, which enables them to leave hospital and be at home with their families. Optimising function and quality of life are major outcomes in PICUs where mortality rates are now low. All who work in ICU need to focus on continuous quality improvement to avoid unnecessary complications, and to mitigate the effects of intensive care on development and psychological health.

We hope this book will be useful to doctors and nurses working in paediatric intensive care in many countries. Please note: these guidelines may not apply to an individual patient or every circumstance of illness. Doctors and nurses should not regard them as universally applicable.

Trevor Duke
Editor 4th Edition

THE GOLDEN RULES

1. The prime goal of intensive care is to preserve airway, breathing and circulation at all times
2. Identify high-risk children before they deteriorate; be proactive rather than reactive in treatment
3. Wash your hands - always observe the 5 moments of hand hygiene
4. Be polite and helpful to referring staff from both RCH and other hospitals
5. All visiting medical teams **must** speak with the PICU consultant or senior registrar when seeing a patient in the ICU
6. In a child with suspected severe sepsis, admit immediately to ICU if any *one or more of*:
 - Venous blood Lactate >3 mmol/L;
 - Neutropenia (neutrophil count <1000 / mm³), unexpected (i.e. not related to cancer chemotherapy)
 - Large pleural effusion (e.g. near white out of hemithorax)
 - Coagulopathy (INR>1.6, APTT>60, Fib <1)
 - Signs of shock persisting despite a total of 40ml/kg fluid
7. Admit to ICU: infants with bacterial meningitis; children with croup who needed more than 2 doses of adrenaline in the previous hour; children with acute leukaemia and a white cell count >300,000
8. If you order a pathology test you have to follow up. All pathology results must be reviewed, acted-upon or handed on to the next registrar to follow-up
9. Do not order diagnostic tests at regular intervals (such as every day), but rather in response to specific clinical questions
10. Do not transfuse red blood cells in hemodynamically stable, non-bleeding ICU patients with a haemoglobin concentration greater than 7 g/dL (Hb threshold in cardiac patients varies with lesion and age)
11. Do not deeply sedate mechanically ventilated patients without a specific indication and without daily attempts to lighten sedation
12. Do not continue life support for patients at high risk for death or severely impaired functional recovery without offering patients and their families the alternative of care focused entirely on comfort.
13. Do not continue antibiotics when clinical and laboratory signs of sepsis are absent or resolved and cultures are negative
14. Do not give oxygen for upper airway obstruction unless you have made the decision to intubate

15. Intubate for upper airway obstruction before cyanosis or exhaustion develops
16. Do not paralyse a child with upper airway obstruction when intubating
17. Take chest x-ray (and look at it) in all intubated admissions, and in all children intubated in the unit
18. Never paralyse a child without looking at the tidal volumes and adjusting the ventilator appropriately
19. Do not extubate after a severe hypoxic-ischaemic insult or suspected non-accidental brain injury without discussion with the ICU consultant
20. Give at least 2 ampoules of antivenom before giving FFP or cryoprecipitate for coagulopathy after snakebite

Cardiac

21. A child with a lactate >4 mmol at 4 hours after cardiac surgery has a 45% risk of a major complication: maintain blood volume, Hb 120-140, do not stop inotropes, optimise oxygen saturation, deep sedation & paralysis, correct arrhythmias, consider echo. Contact ICU consultant if the lactate is higher at 8 hours.
22. Single ventricle (eg Norwood, pulmonary atresia plus Blalock): balance pulmonary and systemic vascular resistance (saturation 70-85%). See protocol.
23. Oxygen delivery is usually best with dobutamine 5-10 mcg/kg/min plus a vasodilator; ensure adequate coronary perfusion pressure (diastolic BP - CVP).
24. If you think that a cardiac surgical patient needs an echo, call the ICU consultant.
25. After a shunt (central, Blalock, Norwood), start heparin 10u/kg/hr immediately on return to ICU. Do not wait for coagulation test results.
26. Internal jugular and subclavian central lines, and surgically-placed lines are not to be inserted without ICU consultant approval, and should use ultrasound guidance.
27. Call the ICU consultant if you take more than 30mins to insert a femoral line.
28. Unless there is a contraindication, give heparin 10u/kg/hr to a maximum of 250 U/hour to all children with a central line (neck or femoral).
29. Never place an ulnar or radial artery catheter without first ensuring the presence of dual blood supply to hand and fingers by other arteries (by ascertaining the presence of ulnar and radial pulses, and ideally by ultrasound).
30. If digits show signs of ischaemia associated with intra-arterial cannula, remove catheter immediately, call Plastic Surgeon and ICU consultant, and haematologist with view to

heparin therapy.

31. No child should go to theatre on parenteral nutrition – change to equivalent glucose infusion, mg/kg/min.

Administration

32. At least one registrar should stay in each Pod at all times, except in an emergency.
33. If you leave the unit, tell the nurse in charge, and hand over your patients to another registrar.
34. Inform the bedcard unit when a child is admitted to ICU, and on discharge.
35. For every patient, write a progress note at least twice a day, and when there is a change in status or an invasive procedure is performed. If you admit a patient, complete EMR admission before you leave the unit.
36. Discharges. Carefully complete all the tasks listed on the ICU Discharge Check List. Night registrars should write discharge summaries before the 8am handover.
37. If a child dies in ICU, the summary and medical certificate of death should be written by the ICU Registrar, and the ICU Consultant is responsible for a Coroner's submission. Complete the ICU Discharge Check List.
38. The AUM (nurse in charge of a shift) has the right to ask an ICU registrar to ring the ICU consultant about a patient (and the AUM may phone the consultant if the registrar refuses).

ADMIN - FELLOWS

The roles of the ICU Fellow include:

Provide continuity of clinical care

Provide supervision and teaching to ICU registrars

Assist and supervise ICU registrars in procedures

Facilitate communication between ICU registrars and the director of ICU

Supervise MET calls and review of PICU discharges.

The ICU Fellow should attempt to provide continuity of care, especially for long term or complicated patients.

The Fellow is sometimes not allocated to individual patients, so that he or she is free to provide overall supervision and teaching to the registrars.

Should ensure that each ICU registrar gets a lunch break, and covers the rooms if necessary.

Supervises all new admissions and discusses the management with the registrar – but lets the registrars do as much as possible themselves, and encourages them to formulate their own plan of management.

Can take PETS calls during the day

Ensure that the Boyle's machine has been checked daily.

The Fellow may respond to referrals from Emergency and Met calls during the day.

Inform the consultant about PETS, Emergency, and MET calls.

Teaching is an important part of the Fellow's job. The Fellow should do a teaching round with the registrars and medical students each day if there is time.

The Fellow should keep the ICU consultant informed of new admissions and important developments at all times.

ADMIN - CONTACTING THE PICU CONSULTANT

PURPOSE

To ensure ICU consultants are notified of key events and information.

The ICU consultant must be notified in the following circumstances:

Patient deterioration

Cardiovascular:

- Cardiac arrest or refractory significant arrhythmia
- Major bleeding requiring transfusion or activation of massive transfusion protocol
- VIS score doubled or adrenaline >0.1 or noradrenaline >0.15
- Lactate >4mmol/L or SvO₂<45mmHg or Delta SvO₂ >25 (Single ventricle)

Respiratory:

- Prior to endotracheal intubation or extubation, if unplanned
- Unanticipated commencement of mechanical ventilation or NIV
- Marked increase in ventilation or NIV requirements including increase of ≥ 30% in FIO₂ (e.g. increasing from a FIO₂ 30% to 60%)
- Upper airway obstruction persistent despite >2 doses of adrenaline, or hypoxic (SpO₂<90%)

Neurological:

- Refractory intracranial hypertension (notify Neurosurgeons as well)
- New focal neurological signs or marked deterioration in GCS
- Refractory seizures unresponsive to 2 doses of midazolam and loading with a long-active anticonvulsant agent

Renal / Metabolic

- Before commencing CRRT
- Urine output < 0.5 ml/kg/hour unresponsive to therapy for > 4 hours (unless on renal replacement therapy)
- Oliguria with hyperkalaemia or acidosis
- Hyperammonaemia: >150 micromol/L

Procedural

- Prior to any major invasive procedure planned in ICU
- Prior to any procedure in an ECMO patient
- If there is a complication of any procedure
- Prior to referral to an external unit e.g. Endocrinology, General Surgery, Neurosurgery

PETS / PIPER

- Referral of any patient who fulfils the "Go Now" criteria
- Where any PETS trip is being delayed for any reason: lack of staffing / transport difficulty / bed availability

- If on arrival the patient is sicker than was previously understood

ICU workflow

- Unexpected death in ICU or discharge from ICU
- Any referral to ICU (whether accepted or refused)
- Excessive workload for ICU junior medical staff resulting in unsafe conditions or inability to complete tasks
- Significant disagreement or conflict with ICU nursing staff, external medical staff, patient or patient's family
- Adverse clinical event leading to a VHIMS (Victorian Health Incident Management System) report

There should be a low threshold for notification. Even if you are able to manage the situation the consultant still needs to know that these significant events have occurred.

ANALGESIA AND SEDATION

Children tolerate short-lived discomfort remarkably well if they are treated sensitively – they fall over and run into things almost daily. Narcotic analgesics and heavy sedation inhibit movement (needed to get rid of excess body water) and coughing, and prolong intubation and mechanical ventilation with substantial additional risk of morbidity and mortality. On the other hand, inadequate analgesia for severe or prolonged pain is cruel and harmful.

Muscle relaxants do not prevent pain or awareness.

Although ideally a PICU patient should be alert, cooperative and free of pain and anxiety, in practice, painful procedures and alarming surroundings often make life unpleasant. In particular, being paralysed but aware and unable to move, communicate or avoid noxious stimuli may not be painful but is highly unpleasant and represents a failure of care.

Opiates, even in high dose, don't reliably prevent awareness or produce amnesia. An opioid plus a sedative relieves distress better than high opioid doses alone.

Treat every child as if he or she is aware: speak frequently to the child (not loudly enough to prevent sleep); warn the child of procedures; explain alarms and sudden loud noises; limit bedside conversations with parents and other staff to what you would want the child to hear.

In nonparalysed children use a scoring system (we use COMFORT B, see below) and document the required sedation score range. It will reduce the amount of drug use and minimize risks of withdrawal and polypharmacy. BIS monitors can be used over the age of 2 years and titrated to the level of sedation required. BIS > 65 is associated with awareness during anaesthesia but not distress unless in pain and paralysed.

Signs of pain if paralysed or apparently unaware

Autonomic: sweating, dilated pupils, tachycardia, hypertension, pulmonary hypertension. Exclude other causes of autonomic response (hypercarbia, hypovolaemia, hypoglycaemia).

Raised intracranial pressure.

Non-drug means of managing pain and distress

Remove obvious avoidable sources of pain, and minimise the distress caused by painful procedures:

Wrap and cuddle neonates if able. Give oral sucrose 12.5% before painful procedures in neonates. This may be more effective if the baby is wrapped. Topical amethocaine or EMLA creams, vibratory or cooling devices are all effective at reducing pain during cannulae insertion.

Electronic devices with games or movies can also be very effective as distraction.

Tell the child beforehand what is going to happen and why, even if the child is paralysed and appears too young or too unconscious to understand.

Don't say, "This won't hurt" if it will. Say, eg, "It will hurt a little, but only for a moment and then I'll get you a drink".

Use "rewards" after procedures: sip of fluid, dressings with a smiley face, TV, read the child a book.

Discuss painful procedures and the reasons for them with the parents (if present) beforehand. Parents should stay with the child if they are likely to remain calm.

Feed the child as soon as possible.

Promote sleep and keep the child warm and comfortable; if possible, schedule procedures to allow rest periods and preserve the child's day-night cycle.

Play the child's favourite music.

Speak gently to the child and keep the surroundings as non-threatening as possible. Consider contacting the Play Therapists or Comfort Kids for ideas

Drug management

Our standard regimen uses infusions of morphine and clonidine (<12 months), morphine and midazolam (children 12 months and older), often supplemented with paracetamol.

Dexmedetomidine is available for short term (<72 hour use)

Use the age appropriate Pain and Sedation algorithm (a specific algorithm available for ECLS patients usually requires larger initial dosing and reloading with circuit changes)

Paracetamol can greatly reduce the dose of narcotics needed, even with severe pain. Up to 90 mg/kg/day can be used for a maximum of 2-3 days.

Similarly tramadol 1-2 mg/kg 6 hrly and NSAID's such as ibuprofen 5-10 mg/kg 8 hrly (if feeding or on anti-ulcer prophylaxis, normal renal function, not bleeding) should be used. Parecoxib 1 mg/kg daily can also be used intravenously. When converting to oral opiates chart oxycodone rather than codeine.

Reduce morphine and midazolam well before you plan to wean the child from ventilation, especially if drug excretion is likely to be slow (eg newborns; poor liver or kidney function because of recent sepsis or low cardiac output; prolonged administration of the drug; large drug load because of ascites or oedema).

Midazolam has a short duration of action after a single dose, but a prolonged duration of action after several doses, so it should be stopped well before weaning from ventilation. It is associated with increased rates of delirium.

Consider using midazolam (0.1-0.2 mg/kg IV bolus) for procedures requiring brief sedation - beware myocardial and respiratory depression. If analgesia and sedation are required, consider ketamine.

It may be necessary to give multiple doses of morphine (100mcg/kg) and midazolam (100mcg/kg) before giving IV infusions, as maintenance infusions take 4-5 half-lives to reach plateau concentrations. IV boluses of morphine and midazolam cause hypotension and myocardial depression. Consider reducing the doses (to 50 mcg/kg) or giving them more slowly (over 10 mins) or if necessary, by means of a more rapid infusion over an hour.

In a critically unstable child, the best sedation is a compromise between ideal sedation and severe myocardial depression.

Morphine has lower rates of tachyphylaxis and withdrawal than fentanyl, so use morphine as first line opioid infusion. For proven morphine sensitivity in a ventilated child, use fentanyl 2 mcg/kg IV bolus, then infuse 2-4 mcg/kg/hr. Rotate with fentanyl or hydromorphone after 5-7 days.

Fentanyl is given as an IV bolus (1-4 mcg/kg) for rapid onset with minimal CVS effects. Cardiac output may be reduced, as fentanyl suppresses endogenous catecholamine output. It often causes apnoea in unventilated patients. Larger doses given rapidly cause apnoea with chest wall rigidity unless a muscle relaxant is given first. Give fentanyl slowly in an unstable patient.

Remifentanyl can be used for renal and hepatic failure or severe pulmonary hypertension. Infusions aim for 0.05 – 0.2 mcg/kg/min. Boluses of 0.1 – 0.5 mcg/kg/min can be given but there is a very high risk of bradycardia and hypotension. Tachyphylaxis occurs.

Hydromorphone (load 50mcg/kg slow IV over 1 hour 2mg max dose) Bolus <50kg: 5 – 10 mcg/kg
>50kg: 0.1-0.2mg, infusion 5-40 mcg/kg/hr

Dexmedetomidine is a newer (more specific) alpha₁ agonist with a shorter half life than clonidine. It is useful for short term sedation and also provides analgesia. If stable give 1mcg/kg over 1 hour then commence an infusion at 0.3-0.5 mcg/kg/hr (it can be increased upto 2mcg/kg/hr). Cease after 72 hours or transition to clonidine as associated with withdrawal if used longer than 96 hours.

Propofol is available for short term (<48 hours) use in those over 2 years of age. Small boluses of 0.5-1 mg/kg are effective for short term sedation and infusions should be limited to 4 mg/kg/hr. It should be avoided in mitochondrial diseases, malnourished and unexplained lactic acidosis.

TCl (target controlled infusions) can be used aiming for plasma (or effect site concentrations) of 1 - 4 mcg/ml titrated to level

of sedation required (or BIS if available)

Patient-controlled analgesia (PCA)

Used if the child can understand the concept of pressing a button to self-administer pain relief when required.

Consider its use in intelligent children > 6 years old, and in most children > 8 years.

Contact the Children's Pain Management Service (CPMS) for advice before use, document a referral and a PONV (post-operative nausea and vomiting protocol) *add MR number*.

Usual regimen: morphine 0.5 mg/kg (up to 50kg then use 50 mg) in 50 ml 0.9% saline; bolus dose 2 ml (= 20 mcg/kg morphine); lockout interval 5 minutes; background infusion (optional – rarely required and increase side effects) 0.5 ml/hour (= 5 mcg/kg/hr). Fentanyl or oxycodone PCA are alternatives refer to - CPMS.

Fill in the PCA prescribing sheet especially the naloxone orders; observe carefully for respiratory depression, excessive sedation, nausea or vomiting; monitor SpO₂; only the child may press the demand button. Adolescents may benefit from naloxone added to the PCA or prn if PONV, pruritis

Intubated, non-ventilated children (eg croup) or on CPAP

Usually need no sedation. If very agitated or distressed, try NG clonidine 2-5 mcg/kg 6 hourly, NG chloral hydrate 20- 30 mg/kg 6 hourly prn, NG chlorpromazine 0.25 mg/kg 6-8 hrly, or NG diazepam 0.1-0.15 mg/kg 6 hourly prn. Try feeding, comfort and entertainment. If a painful wound or injury (see below, trauma), use paracetamol, IV morphine infusion or PCA, or local anaesthesia.

Paralysed children

Morphine infusion: <3mo: 10-30 mcg/kg/hr; >3mo: 20-50 mcg/kg/hr.

Midazolam infusion usually 1-4 mcg/kg/min or dexmedetomidine 0.3-2 mcg/kg/hr. For long-term sedation, consider iv clonidine 0.5-2 mcg/kg/hr. NG diazepam 0.1 mg/kg 4-6 hourly.

Can use morphine 0.05 mg/kg boluses 10 mins (Fentanyl 1- 4 mcg/kg 5 mins) before ET suction.

Reassess analgesia and sedation needs frequently (hold relaxants if possible and perform COMFORT B).

Need increased doses of sedation if using IRV or PRV ventilation patterns.

For long-term paralysis, consider adding clonidine 0.5-2 mcg/kg/hr or chlorpromazine 0.25-0.5 mg/kg 6-8H slow IV, rather than increasing doses of morphine and midazolam.

Intubated and ventilated but non-paralysed children

Consider using PCA (qv) in children > 6 years of age, depending on cognitive development.

Morphine infusion: <3mo: 10-30 mcg/kg/hr; >3mo: 30-50 mcg/kg/hr.

Midazolam 1-4 mcg/kg/min, dexmedetomidine 0.3-2 mcg/kg/hr or clonidine 0.5-2 mcg/kg/hr.

Reassess sedation and analgesia needs frequently (COMFORT B)

If using pressure-support ventilation or ASB, use a backup ventilator rate if there is any possibility that the doses of sedative or opiate will suppress breathing.

Painful procedures

Don't forget to optimize all developmentally appropriate non-pharmacologic measures (eg music and play therapy, Comfort Kids, parents).

Wrap and cuddle neonates if able. Give oral sucrose 12.5% before painful procedures in neonates. This may be more effective if the baby is wrapped.

Topical amethocaine or EMLA creams, vibratory or cooling devices are all effective at reducing pain during insertion of peripheral IV or IA cannulae, even in a paralysed child.

Conventional infusion rates of morphine don't usually abolish the acute pain of IV cannulation.

Electronic devices with games or movies can also be very effective as distraction.

For intermittent painful procedures in a child who is comfortable the rest of the time, give IV boluses of fentanyl eg 1-2 mcg/kg 5 minutes before the procedure.

Ensure that local anaesthesia is given before isolated painful procedures (eg insertion of a central line or intercostal catheter, or bone marrow aspiration).

Surgery in ICU

For example, chest opening, cardiac catheter, balloon atrial septostomy, ECMO cannulation. Give full general anaesthesia sufficient to guarantee the absence of pain and awareness. A doctor must be continuously present with any anaesthetised patient. Use additional local anaesthesia with bupivacaine 0.5% ± adrenaline or ropivacaine 0.75% (max 0.5 ml/kg) if possible to prevent post-operative pain.

Cardiac surgery

In theatre, all children have received large bolus doses of opiate; the effect will continue for several hours in ICU.

Unless the child is to be weaned immediately from IPPV without any further doses of muscle relaxant, start morphine infusion (<3 mo 10-30 mcg/kg/hr; >3mo 20-40 mcg/kg/hr; >30kg 1-

5mg/hr). Add dexmedetomidine 0.3-2 mcg/kg/min (omit loading dose if unstable) or use clonidine 0.5-2 mcg/kg/min or midazolam 1-2 mcg/kg/min (>30kg 1-3m/hr) if paralysis is likely to last more than 4 hours.

If planning immediate weaning, infuse morphine 10-20 mcg/kg/hr (use nothing, low dose propofol or dexmedetomidine); increase if child is in pain.

Reduce or cease sedatives and reduce morphine 2-4 hours before starting to wean from ventilation.

Prolonged analgesia or sedation

Rather than increasing the dose of morphine, consider opiate rotation (fentanyl, hydromorphone, methadone) adding clonidine 0.5-2 mcg/kg/hr or chlorpromazine (beware hypotension with initial doses)

Ketamine 2-4(max) mcg/kg/min is useful for thoracotomy pain but tachyphylaxis develops quickly, use diazepam rather than midazolam. It will often be used in burns patients and doses will escalate during dressing changes – aim to de-escalate as soon as possible

Patients on opiate infusions for >5 days are at risk of opiate resistance and withdrawal (worse if also on midazolam). Use a WAT score (below) and document the weaning regimen with breakthrough prescriptions. Reduce the dose slowly and look for withdrawal symptoms (agitation, myoclonic jerks, sweating, fever, diarrhoea); increase the dose of morphine or add clonidine (or both).

Convert to oral morphine or methadone and diazepam. Add clonidine if not already charted.

Trauma and burns patients often have coexisting nerve injuries and neuropathic pain – amitriptyline (especially at night), gabapentin and or pregabalin may have a role.

During transport (PETS)

Do not sedate children unless they are ventilated, but ensure adequate analgesia for trauma victims (local blocks, paracetamol, and morphine 0.025 mg/kg boluses if no head injury).

COMFORT B SCORE

The Comfort B score indicates a child's level of pain and sedation.

Step 1: stimulate the patient: touch to the torso or score following general nursing

Step 2: watch for 5 minutes to assess how your patient responds to the stimulus and then assess them using the 6 categories. (If patient ventilated use the Respiratory Response category, if not ventilated use the Cry category)

Step 3: Sum numbers in each category to produce the overall Comfort B score

<p>Alertness</p> <ol style="list-style-type: none"> 1. Deeply asleep 2. Lightly asleep 3. Drowsy 4. Fully awake and alert 5. Hyper alert <p>Calmness/ Agitation</p> <ol style="list-style-type: none"> 1. Calm 2. Slightly anxious 3. Anxious 4. Very anxious 5. Panicky <p>Respiratory Response (Ventilated)</p> <ol style="list-style-type: none"> 1. No coughing/ no spontaneous ventilation 2. Minimal assistance from ventilator 3. Occasional cough, splinting or resistance to the ventilator 4. Actively breathes against the ventilator or coughs regularly 5. Fights ventilator, cough, gag or choking <p>Cry (Non-ventilated)</p> <ol style="list-style-type: none"> 1. Quiet breathing, no crying 2. Sobbing or gasping 3. Moaning 4. Crying 5. Screaming 	<p>Physical Movement</p> <ol style="list-style-type: none"> 1. No movement 2. Occasional, slight movement 3. Frequent, slight movements 4. Vigorous movement limited to extremities 5. Vigorous movement including torso and head <p>Muscle Tone</p> <ol style="list-style-type: none"> 1. Muscles totally relaxed, no muscle tone 2. Reduced muscle tone 3. Normal muscle tone 4. Increased muscle tone and flexion of fingers and toes 5. Extreme muscle rigidity and flexion of fingers and toes <p>Facial Tension</p> <ol style="list-style-type: none"> 1. Facial muscles totally relaxed 2. Facial muscle tone normal, no facial tension evident 3. Tension evident in some facial muscles 4. Tension evident throughout facial muscles 5. Facial muscles contorted and grimacing
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Score of 6-10: Deeply sedated patient

Consider weaning the sedation or analgesia by 10% if appropriate to the child's clinical state and natural history of illness

Score 11-22: Adequately sedated patient

Score 23-30: Inadequate sedation / analgesia

Bolus 0.1mg/kg of morphine and / or midazolam

Reassess 10 min after bolus

If still scoring high, repeat bolus

Reassess in a further 10 mins: if still scoring high, inform medical staff, increase background infusion, and consider another agent (clonidine, dexmetomidine, ketamine).

Look after the ETT and lines, so if the child is active have a syringe of propofol, midazolam, ketamine or fentanyl at the bedside.

WAT SCORE

Information from patient record in the previous 12 hours	
Any loose /watery stools	No = 0 Yes = 1
Any vomiting/wretching/gagging	No = 0 Yes = 1
Temperature > 37.8°C	No = 0 Yes = 1
2 minute pre-stimulus observation	
State	SBS1 ≤ 0 or asleep/awake/calm = 0 SBS1 ≥ +1 or awake/distressed = 1
Tremor	None/mild = 0 Moderate/severe = 1
Any sweating	No = 0 Yes = 1
Uncoordinated/repetitive movement	None/mild = 0 Moderate/severe = 1
Yawning or sneezing	None or 1 = 0 ≥2 = 1
1 minute stimulus observation	
Startle to touch	None/mild = 0 Moderate/severe = 1
Muscle tone	Normal = 0 Increased = 1
Time to gain calm state (SBS1 ≤ 0)	< 2min = 0 2 - 5min = 1 > 5 min = 2
Total Score (0-12)	

- Start **Withdrawal Assessment Test** (WAT-1) scoring from the first day of weaning in patients who have received opioids +/- benzodiazepines by infusion or regular dosing for prolonged periods (e.g., > 5 days). Continue twice daily scoring until 72 hours after the last dose.
- The WAT-1 should be completed along with the Comfort B at least once per 12 hour shift.
 - ✓ Loose/watery stools: Score 1 if any loose or watery stools were documented in the past 12 hours; score 0 if none were noted.

✓ Vomiting/wretching/gagging: Score 1 if any vomiting or spontaneous wretching or gagging were documented in the past 12 hours; score 0 if none were noted

✓ Temperature > 37.8°C: Score 1 if the modal (most frequently occurring) temperature documented was greater than 37.8 °C in the past 12 hours; score 0 if this was not the case.

2 minute pre-stimulus observation:

✓ State: Score 1 if awake and distressed observed during the 2 minutes prior to the stimulus; score 0 if asleep or awake and calm/cooperative (SBS1 ≤ 0).

✓ Tremor: Score 1 if moderate to severe tremor observed during the 2 minutes prior to the stimulus; score 0 if no tremor (or only minor, intermittent tremor).

✓ Sweating: Score 1 if any sweating during the 2 minutes prior to the stimulus; score 0 if no sweating noted.

✓ Uncoordinated/repetitive movements: Score 1 if moderate to severe uncoordinated or repetitive movements such as head turning, leg or arm flailing or torso arching observed during the 2 minutes prior to the stimulus; score 0 if no (or only mild) uncoordinated or repetitive movements.

✓ Yawning or sneezing > 1: Score 1 if more than 1 yawn or sneeze observed during the 2 minutes prior to the stimulus; score 0 if 0 to 1 yawn or sneeze.

1 minute stimulus observation:

✓ Startle to touch: Score 1 if moderate to severe startle occurs when touched during the stimulus; score 0 if none (or mild).

✓ Muscle tone: Score 1 if tone increased during the stimulus; score 0 if normal.

Post-stimulus recovery:

✓ Time to gain calm state: Score 2 if it takes greater than 5 minutes following stimulus; score 1 if achieved within 2 to 5 minutes; score 0 if achieved in less than 2 minutes.

Sum the 11 numbers in the column for the total WAT-1 score (0-12).

ANAPHYLAXIS

The clinical features are hypotension, bronchospasm, erythema, urticaria, angioedema (which may cause airway obstruction), pulmonary oedema, and GI symptoms (nausea, vomiting, abdominal pain and diarrhoea). Many children have the combination of food-related allergy or anaphylaxis and episodic or chronic asthma. The treatment is adrenaline.

1. Give oxygen by mask (or increase the FiO_2 on a ventilator).
2. Give 0.01 ml/kg of adrenaline 1:1000 (=10mcg/kg) by deep intramuscular injection. Repeat after 5 minutes if the child is not improving.
3. If angioneurotic oedema or laryngeal oedema do not respond to parenteral and inhaled adrenaline, early endotracheal intubation is indicated.
4. Give 10 ml/kg boluses of 0.9% saline for hypotension. Large volumes may be needed - in this situation, insert a central venous line and monitor central venous pressure.
5. If the child still has clinical signs of anaphylaxis after 2 IM doses of adrenaline commence an adrenaline infusion. In an emergency with no central line: put 1mg in 1000ml and run at 5ml/kg/hour (0.08 mcg/kg/min). Principle is to start with a large dose then reduce when symptoms relieved, rather than a small dose and escalate. Severe tachycardia or systolic hypertension should lead to dose reduction.
6. If hypotension persists despite adequate fluid therapy (as shown by CVP >10), obtain an echocardiogram and consider infusing noradrenaline as well as adrenaline.
7. If a child needs ongoing adrenaline infusion (sometimes needed for ingested allergens which continue to trigger mast cell activation for up to 24 hours), or if there is a need to reduce fluid volume infused because of risk of fluid overload, give adrenaline via central line (0.15mg/kg in 50ml 1ml/hour = 0.05 mcg/kg/min).
8. If there is severe bronchospasm, treat with nebulised adrenaline and ipratropium, IV adrenaline, IV aminophylline, and IV methylprednisolone.
9. Antihistamines and steroids are not given routinely. Antihistamines often reduce severe pruritis, but may cause hypotension.
10. If a child has to be given a drug when they are known to be allergic to it, give methylprednisolone 2 mg/kg IV and low-dose adrenaline infusion (as above) before administration of the drug.
11. **Emergency calls from theatre may be for refractory anaphylaxis** – don't forget to remove potential triggers eg latex, chlorhexidine.

12. Refractory anaphylaxis may require infusions of noradrenaline (0.1-2 mcg/kg/min), vasopressin (0.02-0.06 units/kg/hr), glucagon (40mcg/kg iv to max 1 mg); consider ECLS or cardiopulmonary bypass
13. ALWAYS take samples for tryptase (1-2 mls in a plain or serum tube) at 1, 4 and 24 hours to confirm the diagnosis of anaphylaxis

ANTIBIOTICS COMMUNITY-ACQUIRED INFECTIONS

CNS, EYE

Meningitis. Cefotaxime 50mg/kg/dose (max 2g) 6H IV (+ if <2mo, benzylpenicillin 50mg/kg/dose 4H + gentamicin IV).

HSV encephalitis. 12wk-12yr: aciclovir 500mg/m² 8H IV.

Periorbital cellulitis. See soft tissue cellulitis.

Orbital cellulitis (proptosis, chemosis, eye movement painful). Cefotaxime 50mg/kg/dose (max 2g) 6H IV + flucloxacillin 50mg/kg/dose (max 2g) 6H IV.

CVS

Prophylaxis. Before cardiac surgery: cefazolin 50mg/kg IV.

Dental and upper respiratory procedures: amoxicillin 50mg/kg (max 2g) oral 1hr before, or benzylpenicillin 50mg/kg (max 3g) IV at induction.

GI TRACT

Acute peritonitis or ascending cholangitis. Benzylpenicillin 50mg/kg/dose (max 3g) 4-6H IV + gentamicin daily IV + metronidazole 15mg/kg (max 1.6g) stat then 7.5mg/kg/dose (max 800mg) 8H IV.

Acute giardiasis. Tinidazole 50mg/kg (max 2g) stat oral; may repeat after 48 hr.

URINARY TRACT INFECTION

Sick, or <6mo, or acute pyelonephritis. Benzylpenicillin 50mg/kg/dose (max 3g) 6H IV + gentamicin daily IV.

Not sick and >6mo. Cotrimoxazole 4/20 mg/kg (max 160/800) 12H oral, or cephalexin 15mg/kg/dose (max 500 mg) 8H oral.

Prophylaxis. Cotrimoxazole 2/10 mg/kg (max 80/400) daily oral, or trimethoprim 2mg/kg (max 80mg) daily oral.

RESPIRATORY

Acute tonsillitis. Take throat swab for GAS. Penicillin V 30 mg/kg (max 1g) 12H oral for 10 days. Consider no antibiotic (especially <4yr).

Acute otitis media. None for 48hr if mild-mod and >12mo, or amoxicillin 15mg/kg/dose (max 500mg) 8H oral.

Pertussis prophylaxis. Clarithromycin 7.5mg/kg/dose (adult 500mg) 12H oral for 7 days.

Community acquired pneumonia

Mild: amoxicillin 15mg/kg/dose (max 500mg) 8H oral.*
Moderate: benzylpenicillin 50mg/kg/dose (max 3g) 6H IV.*
Severe pneumonia: benzylpenicillin 50mg/kg/dose (max 3g) 4H IV + gentamicin daily IV. If empyema see below).
Add roxithromycin if fever >=3 days in a child aged >=4 yr. Add Oseltamivir if known or suspected influenza.

Pneumonia with empyema (until pleural fluid cultures known). Flucloxacillin + ceftriaxone + clindamycin.

MRSA pneumonia. Linezolid, add rifampicin if necrotizing pneumonia, or extensive, or abscesses.

Pneumococcal necrotizing pneumonia / empyema. If pen sensitive, benzylpenicillin 50mg/kg/dose (max 3g) 4H IV. Consider azithromycin (anti-inflammatory effect).

SEPTICAEMIA, SKIN, SOFT TISSUE, BONE

Septicaemia, septic shock. Cefotaxime 50mg/kg/dose (max 2g) 6H IV + Flucloxacillin 50mg/kg/dose (vancomycin if central line) +/- clindamycin if toxin-mediated sepsis suspected +/- gentamicin daily IV.

Adenitis. Flucloxacillin 25mg/kg (max 1g) 6H IV.

Bites, animal or human. Procaine penicillin 50mg/kg (max 1.5g) IM stat, then amoxicillin/clavulanate 15mg/kg/dose (of amoxicillin) (500/125mg) 8H oral.

Cellulitis. Mild: cephalexin 25mg/kg/dose (max 500mg).

Mod/severe: flucloxacillin 50mg/kg/dose (2g) 6H IV.

Compound fracture. Fluclo 25mg/kg (max 1g) 6H IV.

Impetigo. Mupirocin 2% ointment 8H if localised, or cephalexin 25mg/kg/dose (max 500mg) 12H oral.

Lice, scabies. Permethrin 1% (head lice), 5% (scabies).

Osteomyelitis, septic arthritis. Flucloxacillin 50 mg/kg/dose (max 2g) 4-6H IV + (if <5yr and not fully Hib immunised) gentamicin daily IV.

CONTACT PROPYLAXIS

Group A strep infection – all household contacts receive cephalixin for 10 days.

N. meningitidis prophylaxis. Rifampicin 10mg/kg/dose (max 600mg) 12H for 2 days.

ANTIBIOTICS - HOSPITAL ACQUIRED SEPSIS

We have a problem with resistant Gram-negative bacteria in PICU, the extended-spectrum beta-lactamase producing *Klebsiella* and *Enterobacter* spp. These have emerged in the last few years, and lead to the increased tendency to use carbapenems and other high-grade antibiotics. We have also seen several deaths from carbapenem-resistant enteric Gram negatives. The unregulated use of vancomycin provides selection pressure for vancomycin-resistant enterococcus (VRE), which can be deadly in the PICU.

In deciding which antibiotics to give consider:

Probability of bacterial infection

Fever or temperature instability, increasing IT ratio or procalcitonin (see procalcitonin section)

Is infection likely to be community or hospital / PICU-acquired?

Are there any signs of severe sepsis?

Signs of severe sepsis: increased inotropic requirement with no other likely cause, worsening hypoxaemia associated with new CXR infiltrates.

Likely pathogens

Likely antimicrobial susceptibility

For hospital-acquired infections, see:

http://ww2.rch.org.au/emplibrary/picu_intranet/Antibiotics_in_the_PICU.pdf

First line treatment of PICU acquired sepsis should be flucloxacillin and gentamicin (or benzylpenicillin, gentamicin and metronidazole for GI tract sepsis)

Hospital-acquired MRSA is uncommon in RCH PICU, although increasing community-acquired MRSA is being seen, especially among high risk groups (indigenous, Pacific islanders, Indian sub-continent).

Although flucloxacillin won't cover the majority of coagulase-negative Staphs, many of these isolates are contaminants, or the infection indolent, so changing-up to vancomycin if an MRSE is isolated will not result in adverse clinical outcomes

Indications for vancomycin

Signs of serious sepsis PLUS

- Known methicillin-resistant *Staph aureus* colonization or infection

- High risk of methicillin resistant coagulase negative *Staph aureus* infection: such as a VP shunt or previous MRSE sepsis with CVC
- Risk factors for community acquired MRSA (indigenous, Pacific islander origin, Indian sub-continent, *plus* signs of Staph infection)

A short-term central line alone is NOT an absolute indication to use vancomycin rather than flucloxacillin as first-line treatment for PICU acquired sepsis

Indications for meropenem or amikacin

Signs of serious sepsis PLUS

- Known colonisation or infection with *Klebsiella* spp or *Enterobacter* spp
- Recent treatment with 3rd or higher gen cephalosporins, quinolones, pip/tazo, Timentin, carbapenems

Approval is required by the RCH Drug Usage Committee (DUC)

STOPPING ANTIBIOTICS or SCALING DOWN ANTIBIOTICS

Before antibiotics are started cultures (blood, urine +/- BAL) should be taken.

Antibiotics should be ceased after 48-72 hours if cultures are negative, and procalcitonin is low.

If it is not appropriate to cease antibiotics (because of a high probability of bacterial infection or signs of severe sepsis) antibiotic therapy can be scaled down when 48 hour cultures are negative. Appropriate antibiotics to scale down to depend on the clinical context, but include benzylpenicillin and gentamicin for culture-negative severe pneumonia or sepsis in the PICU. When scaling down antibiotics, the RCH Clinical Practice Guidelines can be followed for the treatment of focal infections.

Other considerations

Give a single dose of cephazolin 50 mg/kg just before any cardiac surgical procedure.

Do not give prophylactic antibiotics after the procedure. After 48-72hr of antibiotics, the patient has resistant flora; infection occurs at the same rate but with resistant bacteria.

Do not give antibiotics just because an organism has been isolated from an endotracheal or tracheostomy tube aspirate (colonisation is to be expected). Do a BAL.

Always give oral or NG nystatin when giving antibiotics.

All children who have chronic lymphopaenia, congenital or acquired T-cell immunodeficiency, or are on chronic immunosuppressive drugs, including corticosteroids and cancer

chemotherapy, should be on cotrimoxazole prophylaxis three days per week.

“The Sanford Guide to Antimicrobial Therapy” is a useful reference. To purchase the current edition, go to www.sanfordguide.com

ANTIBIOTICS – BY ORGANISM

These are suggestions for initial treatment until sensitivity-test results are available.

Acinetobacter. Ticarcillin or meropenem +/- tobramycin.
Actinomyces israelii (actinomycosis). Benzylpenicillin.
Aeromonas. Cotrimoxazole.
Bartonella kenselae (cat scratch). Ciprofloxacin or cotrimoxazole.
Bacillus anthracis (anthrax). Ciprofloxacin.
Bacteroides. Oral: benzylpenicillin. GI: metronidazole.
Bartonella. Azithromycin.
Bordetella pertussis. Clarithromycin or azithromycin.
Borrelia burgdorferi (Lyme disease). Doxycycline or amoxicillin.
Borellia recurrentis (relapsing fever). Tetracycline or benzylpenicillin.
Botulism: oral vancomycin.
Branhamella catarrhalis. See moraxella catarrhalis.
Brucella. Tetracycline + rifampicin, or cotrimoxazole.
Calymmatobacterium granulomatis (granuloma inguinale). Tetracycline.
Campylobacter fetus. Cefotaxime +/- gentamicin.
Campylobacter jejuni. Azithromycin or erythromycin.
Chlamydia trachomatis. Trachoma: azithromycin.
Lymphogranuloma venereum: tetracycline.
Chlamydia pneumoniae. Azithromycin.
Chlamydia psittaci. Tetracycline.
Citrobacter freundii. Meropenem.
Clostridia. Benzylpenicillin or metronidazole. C.difficile: oral metronidazole or oral vancomycin.
Corynebacteria. Erythromycin. JK group: vancomycin.
Ehrlichia. Doxycycline.
Eikenella corrodens. Amoxicillin +/- clavulanic acid.
Enterobacter. Cefotaxime or meropenem + gentamicin.
Enterococcus. Benzylpen + gentamicin or amikacin.
Uncomplicated urine tract infn: amoxicillin.
Escherichia coli. Cefotaxime +/- gentamicin.
Francisella tularensis. Ciproflox, or gent + tetracycline.
Fusobacterium. Benzylpenicillin or metronidazole.
Gardnerella (Haemophilus) vaginalis. Metronidazole.
Haemophilus ducreyi (chancroid). Erythromycin.
Haemophilus influenzae. Cotrimoxazole. Severe infn: cefotaxime or ceftriaxone.
Helicobacter pylori. Pantoprazole + clarithromycin + (amoxicillin or metronidazole).
Klebsiella pneumoniae. Cefotaxime +/- gentamicin.

Legionella. Azithromycin +/- rifampicin.
Leptospira. Benzylpenicillin.
Leptotrichia buccalis. Benzylpenicillin.
Listeria monocytogenes. Amoxicillin +/- gentamicin.
Moraxella catarrhalis. Cotrimoxazole or cefotaxime.
Morganella morganii. Cefotaxime +/- gentamicin.
Mycobacterium avium. Clarithromycin +/- rifampicin +/- ethambutol +/- clofazimine.
Mycobacterium fortuitum. Amikacin + doxycycline.
Mycobacterium kansasii. Isoniazid + rifampicin +/- either ethambutol or streptomycin.
Mycobacterium leprae (leprosy). Dapsone + rifampicin +/- clofazimine.
Mycobacterium marinum (balnei). Minocycline.
Mycobacterium tuberculosis. Isoniazid + rifampicin + pyrazinamide + ethambutol. Consider MDR-TB
Mycoplasma pneumoniae. Azithromycin or tetracycline.
Neisseria gonorrhoeae. Ceftriaxone.
Neisseria meningitidis. Benzylpenicillin.
Nocardia. Cotrimoxazole.
Pasteurella multocida. Benzylpenicillin.
Pneumocystis jiroveci. Cotrimoxazole.
All children who have chronic lymphopaenia, congenital or acquired T-cell immunodeficiency, or are on chronic immunosuppressive drugs, including corticosteroids or cancer chemotherapy, should be on cotrimoxazole prophylaxis three days per week.
Proteus. Cefotaxime +/- gentamicin. Indole neg: amoxy.
Providencia. Cefotaxime +/- gentamicin.
Pseudomonas aeruginosa. Ticarcillin + tobramycin. Urine: ciprofloxacin, or ticarcillin and/or tobramycin.
Pseudomonas cepacia. Cotrimoxazole.
Pseudomonas mallei (glanders). Streptomycin + either tetracycline or chloramphenicol.
Pseudomonas pseudomallei (melioidosis). Ceftazidime.
Rickettsia. Doxycycline.
Salmonella. Cefotaxime. S.typhi: ceftriaxone or ciproflox.
Serratia. Meropenem or cefotaxime +/- gentamicin.
Shigella. Ciprofloxacin or cotrimoxazole or amoxicillin or ceftriaxone.
Spirillum minus (rat bite fever). Benzylpenicillin.
Staphylococcus. Flucloxacillin +/- gentamicin. Resistant: vancomycin +/- gentamicin and/or rifampicin for bacteraemia / skin and soft tissue. Linezolid +/- rifampicin for severe MRSA pneumonia with empyema.
Stenotrophomonas maltophilia. Cotrimoxazole, or ticarcillin-calvulanic acid.
Streptobacillus moniliformis (rat bite fever). Benzylpenicillin.

Streptococcus Group A. Benzylpenicillin +/- clindamicin (if toxin-mediated sepsis). Cefalexin for household prophylaxis.
Streptococcus Group D (enterococcus) Vancomycin.
Strep.viridans: benzylpenicillin +/- gentamicin.
Treponema pallidum (syphilis). Benzylpenicillin.
Treponema pertenuae (yaws). Benzylpenicillin.
Ureaplasma urealyticum. Azithromycin.
Vibrio cholerae (cholera). Tetracycline or cotrimoxazole.
Vibrio vulnificus. Tetracycline or cefotaxime.
Yersinia enterocolitica. Cotrimoxazole.
Yersinia pestis (plague). Streptomycin +/- tetracycline.

ASTHMA

These guidelines are for severe asthma in ICU. Determine the need for ICU admission by the patient's clinical state and the response to treatment, not by the blood gases.

Consider diagnoses other than asthma, especially in infants with poorly responsive respiratory distress. High doses of salbutamol may be toxic if the child does not have small airways obstruction - consider this possibility if the child has minimal wheeze and develops marked tachycardia, tachypnoea, hypokalaemia and lactic acidosis.

Take a chest x-ray. Antibiotics are very rarely required. Inform the ICU consultant about any ventilated asthmatic, or any child with asthma who is likely to need ventilation soon.

Management

1. Oxygen. Removal of hypoxic drive is not a threat in children, and death is caused by hypoxia. Therefore give high concentrations of oxygen – if on a nebuliser, use 100% oxygen.
2. Nebulised salbutamol. Outside ICU, salbutamol is best given via a spacer device. In patients sick enough to need ICU, use 100% oxygen to drive a wet nebuliser (eg Hudson Micro Mist 1880). Use 0.5% undiluted respirator solution (5 mg/ml), not the 2.5 ml nebulisers (which are only 1 mg/ml or 2 mg/ml). Put 6 ml of 0.5% solution in the nebuliser (and refill when about 2 ml remain):
 - <2 years: oxygen at 10-12 L/min (tape connections)
 - >=2 years: oxygen at 8 L/minIf no salbutamol, use adrenaline 1/1000 (0.1%).
Note: The respirator solution can be added via a 3-way tap on the end of an NG tube passed into the nebuliser through a hole in the side of the face-mask.
3. Ipratropium (Atrovent) 250 mcg/ml. 1 ml added to salbutamol in nebuliser every 20 min for 3 doses, then every 4 hours.
4. Methylprednisolone 1 mg/kg/dose 6H IV for 24 hours, then 12H for 24 hours, then daily.
5. Aminophylline. If not on any theophylline product, give a loading dose of 10 mg/kg IV over 1 hour, and then a continuous infusion (see Drug Doses booklet). Use separate IV lines for aminophylline and salbutamol. Measure theophylline concentration after 12 hours and then daily (target 60-110 mmol/L).
6. Magnesium sulphate 50%. 0.1 ml/kg (50 mg/kg) IV over 20

min, then 0.06 ml/kg/hr (30 mg/kg/hr) to keep serum Mg 1.5-2.5 mmol/L.

7. **Severe asthma and anaphylaxis is increasingly common and causes deaths.** If the child with acute asthma has a history of food-related allergy or **anaphylaxis**, or has any signs of an allergic reaction (such as urticarial, hypotension, facial swelling) then adrenaline should be given. Give 0.01 ml/kg of adrenaline 1:1000 (=10mcg/kg) by deep intramuscular injection. Repeat after 5 minutes if inadequate response. If still symptoms after 2 doses of IM adrenaline commence adrenaline infusion (see anaphylaxis section for dilution and administration peripherally).
8. IV salbutamol. Give in addition to nebulised salbutamol. Give 5 mcg/kg/min for 1 hour, then 1 mcg/kg/min. Check for hypokalaemia and lactic acidosis at least 6 hourly.
9. Some children with mild asthma develop severe lactic acidosis (often with hyperglycaemia) when they are given large doses of sympathomimetics. The acidosis causes hyperventilation, which may be misinterpreted as asthma, so even more sympathomimetics are given. If a child has severe acidosis with minimal hyperinflation, reduce the dose of sympathomimetics.
10. Give 10 ml/kg boluses of 0.9% saline if required for hypotension or low CVP, with maintenance 4% dextrose in 0.9% saline + KCl 60 mmol/L restricted to 2 ml/kg/hr (1-9 yr), and 1.5 ml/kg/hr (10-16 yr). Monitor blood glucose 6 hourly.
11. Do not sedate an unintubated child with asthma.
12. Positive pressure can be helpful in asthma, but increases the risk of pneumothorax. High flow nasal prong oxygen therapy or CPAP may help some patients, but should not interrupt adequate therapy with beta-2 agonists (inhaled or IV) and other therapy. Indications for intubation are severe hypoxaemia and/or clinical signs of exhaustion from severe respiratory distress, despite 100% high flow oxygen by mask and adequate bronchodilator therapy, +/- after a trial of high flow / CPAP.
13. If intubation is required, induce with ketamine 2mg/kg, fentanyl 3mcg/kg and vecuronium IV 0.1mg/kg or rocuronium 1.5mg/kg, with cricoid pressure. Ventilate a child aged 1-9 yr at 16-20 breaths/min, IT 0.8 sec, PEEP 7, use pressure controlled ventilation – observe chest movement carefully, aim for a pCO₂ of 60-100 mmHg with pH 7.10-7.20. Sedate with ketamine 10-20 mcg/kg/min; try to stop vecuronium by 12 hours or as soon as possible because of the risk of myopathy. Cisatracurium may be associated with less myopathy.
14. Induction and intubation may be associated with significant

haemodynamic instability. Give 10ml/kg volume prior to induction and ensure that adrenaline is available. A common reason for decompensation after intubation is breath stacking with increased intrathoracic pressure and reduced venous return. Disconnection from ventilation to allow expiration may be helpful

15. Intrinsic PEEP and gas trapping can be estimated by stopping the ventilator at end expiration for 15 sec; intrinsic PEEP = pressure at end of a normal breath minus pressure at end of prolonged expiration, and trapped gas volume = tidal volume to end-expiration minus normal tidal volume. If you suspect gas trapping, (a) increase expiratory time, (b) pre-oxygenate then disconnect the ventilator tubing for 30-60 sec hourly – or more often if you suspect severe gas trapping (listen near the end of the ETT for exhaled gas flow).
16. Monitor CVP. If there is hypotension despite a normal or high CVP, consider obtaining a CXR (air leak) and an echo. Left atrial size and IVC distensibility with changes on passive leg raise may be used to assess volume responsiveness
17. Avoid rapid cessation of treatment just before discharge to the ward. Salbutamol and ipratropium inhalations can be continued in the ward, with daily prednisolone and aminophylline given IV over 1 hr via a burette every 6 hours.

BRONCHIOLITIS

The first episode of wheezing, apnoea or respiratory distress associated with an upper respiratory tract infection in a child less than 24 months of age.

Nurse in a separate area of ICU if possible.

1. Handle the child as little as possible.
2. Neutral thermal environment. Small infants should be fully clothed (with a hat) in a warm room.
3. Give oxygen if SpO₂ is <92%, give oxygen by nasal prongs. If respiratory distress is severe enough to warrant ICU admission, give Humidified High Flow Nasal Cannula Oxygen therapy
4. Feed with a NG tube unless severe respiratory distress or hypoxaemia despite oxygen or High Flow Nasal Cannula. IV fluids 1 ml/kg/hr for 24-48 hours, monitor blood glucose. An IA line is not required routinely unless the child is ventilated.
5. A chest x-ray should be taken on admission, but is not required every day unless there is deterioration.
6. If the child is becoming exhausted on HFNC oxygen therapy, give nasopharyngeal CPAP (NCPAP) of 6-12 cm H₂O.
7. If the child is becoming tired on NCPAP, consider endotracheal intubation and mechanical ventilation using pressure support. When a child is ventilated, a pCO₂ of 60-80 mmHg (pH 7.10-7.20) is often appropriate. See the advice about ventilation in the entry on Asthma.
8. Antibiotics should not be given routinely. If the neutrophil I/T ratio is high or PCT >2ng/ml, take cultures and give penicillin (or flucloxacillin) ± gentamicin. Check blood glucose in an infant who presents with apnoea. If there is apnoea, fever and PCT >2ng/ml, consider lumbar puncture when safe to do so.
9. A trial of nebulised adrenaline and dexamethasone may be useful for children with severe bronchiolitis. Give adrenaline 1% 0.05ml/kg diluted to 6ml plus dexamethasone 0.6mg/kg stat then prednisolone 1mg/kg 8 hourly. Adrenaline may worsen V:Q matching and causes tachycardia, so monitor closely and stop if worsens

BRONCHOALVEOLAR LAVAGE

Equipment

Combicath, simple dressing set, 10ml syringe, sterile saline, drawing-up needle, sterile scissors, mask, gloves, hat.

Measure the distance from the patient's xiphisternum to the tragus (ear), then to the outer end of the endotracheal tube. If the total distance is less than 35cm, use a Combicath 58223.19 (short), and if it is 35cm or more use a Combicath 58229.19 (long).

Procedure

1. Use a sterile technique (scrub, gloves, mask, hat).
2. Pre-oxygenate the patient.
3. Insert the Combicath through a valved swivel connector (*BodaTM*) and into the endotracheal tube until it is gently wedged.
4. While keeping the white outer protective sheath wedged, pull back the clear inner cannula 0.5 cm so you can remove the 5cm grooved tube that is between the end of the white protective sheath and the end of the clear inner cannula.
5. Push the inner cannula back in so it is gently wedged in the airway, and hold it wedged while pulling the white protective sheath out about 5cm (until it meets the Luer fitting on the end of the inner cannula).
6. While keeping the inner cannula gently wedged, inject 1ml/kg (maximum 10ml) of sterile saline over 5 seconds, and then aspirate gently for 10 seconds using the 10ml syringe.
7. Stop aspirating. To avoid contamination with tracheal secretions, **ONLY ASPIRATE WHILE THE INNER CATHETER IS WEDGED.**
8. After you have stopped aspirating, hold the outer protective sheath still and pull the inner cannula back about 5cm until you can see the end of the metal tube that is inside the inner cannula.
9. Pull out the whole Combicath (protective sheath and inner cannula).
10. Wipe the end of the protective sheath with a sterile swab, then cut off the distal 1cm of the protective sheath with sterile scissors.
11. Push the Luer fitting of the inner cannula back into the Luer fitting of the outer protective sheath so that the end of the inner cannula that was inside the patient is poking out of the protective sheath.
12. Inject 1ml of sterile saline through the inner cannula into a sterile sampling bottle.
13. Label the bottle and send it for a Gram stain, culture, viral PCR, and other tests as indicated (fungal wet-prep and culture,

Aspergillus antigen, pneumococcal PCR, 16SrRNA, CMV PCR,
PjP PCR if immune compromised, Z-N stain and GeneXpert
MTB/Rif if TB suspected).

BURNS

Many patients with severe burns are managed in the burns unit - the risk of infection is high in ICU.

Children with airway burns should be admitted to ICU (stridor, hoarseness, black sputum, facial swelling). Airway and oro-facial burns often need early intubation even if the airway is not yet compromised - always discuss this with the ICU consultant. Carefully document what is seen at laryngoscopy including presence of soot and colour changes. Get an ophthalmology consult early for facial burns.

Adequate analgesia is very important. Use morphine 0.2 mg/kg IM, or 0.1 mg/kg boluses IV titrated to effect - then an IV infusion.

Insert an IV (preferably through normal skin). Take blood for FBE, electrolytes, blood group and hold. After burns in an enclosed space, measure carboxyhaemoglobin and thiocyanate (1ml blood in gel tube).

If carbon monoxide >10% on admission, or if decreased level of consciousness, low blood pressure, or high blood lactate, treat as CO *and* cyanide (CN) poisoning (the CN result will come back too late to guide initial treatment). Discuss hyperbaric oxygen treatment (at the Alfred) with the ICU consultant – it may not be beneficial, and the risk of transporting the child is very great, especially <12 months of age.

If there are circumferential burns of the neck, trunk, limbs or digits, escharotomy should be discussed with the burns surgeon urgently. Escharotomy can be done in theatre or on the ICU if urgently required.

Blood transfusion is rarely required. Do not give prophylactic antibiotics or steroids.

Fluids

The goal is to minimize oedema, with albumin ≥ 30 g/l, Na > 140 mmol/l, and urine output >1 ml/kg/hour.

Initial resuscitation: treat shock with 10 ml/kg boluses of 0.9% saline. Give a total of 3 ml/kg per 1% area burnt for the first 24 hours from the time of the burn (half in the first 8hr, and the remainder over the next 16hr); use equal parts of 4% albumin and Hartmann's solution.

Maintenance: Plasma-lyte 148 and 5% dextrose.

After the first 24 hours, give about 1.5 ml/kg/day per 1% area burnt, plus usual maintenance fluid. Adjust fluid intake according to urine output, CVP, pulse and BP.

Do not use hypertonic saline for resuscitation.

Start enteral (NG or NJ) feeds early.

If there are signs of sepsis, antibiotic therapy should be based on cultures of skin biopsies, not surface swabs. Ask the surgeons to do biopsies when they are skin grafting or changing dressings.

CARDIAC DEFINITIONS

CARDIOPULMONARY BYPASS

Aortic cross-clamp time. Duration of clamping of the ascending aorta during CPB.

Cardioplegia. Cessation of myocardial activity on CPB using electrolyte solution delivered via coronaries.

Cardiopulmonary bypass (CPB). Systemic venous blood removed, pumped through oxygenator (gas exchange) and returned to aorta.

Circulatory arrest time. Period of no systemic blood flow (CPB pump stopped). Accompanied by protective hypothermia.

Open/closed case. Requires/does not require CPB.

Selective cerebral perfusion. CPB via innom artery only. Provides cerebral blood flow during arch repair.

SURGICAL PROCEDURES

Arterial switch (Jatene). (TGA) Divide and transpose Ao and PA, reimplant coronaries to Ao. PAs brought anterior to Ao before anastomosis ('Lecompte').

Bentall. (Ao root dilation and AR) Ao valve replacement, graft replacement of Ao root, coronary reimplantation.

Bidirectional cavo-pulmonary shunt (BCPS, bidirectional Glenn). (Single Ventricle) SVC to RPA (flow to both PAs). Bilateral BCPS: R & L SVCs to PAs.

Blalock-Taussig shunt, modified (mBTS). Subcl or innom art - PTFE tube - proximal branch PA.

Damus-Kaye-Stansel (DKS). Divide MPA. Anastomose proximal PA to asc Ao. (alternative supply to distal PAs).

David. (Ao root dilation/aneurysm, normal AV architecture) Graft replacement Ao root, reimplantation of native AoV.

Double Switch. (ccTGA) Atrial switch (usually Senning) and arterial switch operation.

Fontan. (Single ventricle) Systemic venous return to PA, bypassing ventricle. External conduit, usually fenestrated between conduit and atrium.

Hybrid Stage 1. (Single ventricle) Norwood alternative. Stent ductus; bilateral PA bands; atrial septectomy.

Kawashima. (Single Ventricle) BCPS with interrupted IVC (leaves only hepatic veins draining to RA).

Konno. (Ao stenosis) Enlarge Ao root and LVOTO, Ao valve prosthesis, homograft or Ross.

Maze procedure. Multiple atrial incisions to interrupt atrial reentrant pathways.

Nakaidoh (Bex). (TGA/DORV+PS). Translocate Ao to LV, close

VSD, anastomose PA to RV.
Norwood. (Single Ventricle). Reconstruct asc Ao with prox PA ± homograft; supply PAs via mBTS or RV-PA conduit (Sano); atrial septectomy.
Rastelli. (TGA+VSD+LVOTO). Divide PA; LV to Ao tunnel; RV to PA conduit.
REV (Reparation à l'étage ventriculaire). (TGA/DORV+PS). LV to Ao tunnel; anastomose PA to RV.
Ross. (Ao stenosis) Replace Ao valve with pul valve (autograft), homograft to pulmonary position.
Sano shunt. (Single Ventricle) RV to PA conduit.
Starnes. (Neonatal Ebstein's anomaly) Fenestrated patch closure of tricuspid valve, atrial septectomy, mBTS.
Warden. (PAPVD to SVC) Transect SVC. Baffle PAPVD to LA via ASD. Reconnect proximal SVC to RA.
Yasui. (IAA/LVOTO, 2 ventricles) Ao arch repair, DKS, baffle LV to pulm art via VSD, RV-PA conduit.

FORMULAE

Cardiac index (CI) = CO / BSA. Normal 3.5-5.5 l/min/m².
Ejection fraction (EF) = (EDV-ESV)/EDV. LV 55-75%; RV 50-60%.
Fractional shortening (FS) = (EDD-ESD)/EDD. LV: 28-45% + normal geometry.
Left ventricular stroke work index (LVSWI) = SI x (MAP-LAP) x 0.0136. Normal 50-62 g-m/m².
Modified Bernoulli. Echo estimate of pressure gradient (Δp) using doppler velocity measurement = $4 \times V_{max}^2$.
Oxygen consumption (VO₂) = CI x Hb (g/L) x 1.36 x ((SaO₂ - SmvO₂) / 100). Normal: infant 160-180, child 100-130, adult 120-150 ml/min/m².
Oxygen delivery (DO₂) = CI x Hb (g/L) x 1.36 x SaO₂. Normal 500 ml/min/m².
Oxygen extraction ratio = VO₂/DO₂
Pulmonary to systemic flow ratio (Qp/Qs) = (SaO₂ - SmvO₂) / (SpvO₂ - SpaO₂). Normal 1.0.
Pulmonary vascular resistance index (PVRI) = 79.9 x (MPAP - LAP) / CI (normal 80-240 dyn-sec/cm⁵/m²) = (MPAP - LAP) / (Qp/m²) (normal 1-3 Wood unit.m²).
RV systolic pressure (RVSP). From echo = 4 x TR Vmax² + RAP.
Stroke index (SI) = CI / HR. Normal 30-60 ml/m².
Stroke volume (SV) = CO / HR. Normal 50-80 ml.
Systemic vascular resistance index (SVRI) = 79.9 x (MAP - CVP) / CI (normal 800-1600 dyn-sec/cm⁵/m²) = (MAP - CVP) / CI (normal 15-30 Wood unit.m²).

ECG intervals (upper limits of normal) in milliseconds

	Newborn	12 mo	8 yrs	16 yrs
PR	100(120)	110(140)	130(160)	135(180)
QRS	65(85)	65(85)	80(100)	90(110)
QTc	420(450)	410(450)	410(445)	410(450)

QTc: Bazett's formula = $QT \sqrt{\text{heart rate}/60}$

CARDIAC ADMISSION: POST-OP

Preparation

Know what to expect. Before admission access details of diagnosis and conference discussions on Cardiobase. Imaging is available on Excelera.

On arrival from theatre

Receive handover from anaesthetist and surgeon. Anaesthetist remains in charge of patient until handover complete. Important points: details of heart lesion; preop functional state; previous problems; precise surgery performed; duration of CPB, cross-clamp and circulatory arrest (if any); problems during surgery or post-bypass; details of anaesthesia; coagulation and bleeding status; heart rate, rhythm, saturation and pressures before leaving theatre; *particular post-op problems anticipated in this child.*

After cardiac surgery, there is a high risk of arrest:

1. In infants <2yr with a high lactate (>5 mmol/l on arrival in ICU, or >4 mmol/l after 4 hr)
2. In children with a low central venous O₂ saturation (<60% in a fully corrected heart, or SaO₂-ScvO₂ gradient >35% in an uncorrected heart).

Treat for low cardiac output syndrome. Notify cardiac surgeon and ICU consultant if lactate still rising 4 hours post-op.

After handover examine the patient fully. Pay particular attention to site and security of all hardware, adequacy of ventilation, cardiac examination. Set ventilator and alarms. Check for bleeding from chest drains.

Take the first set of bloods on admission: ABG, ScvO₂, urea, creatinine, electrolytes, LFTs, FBC, coags.

Order and examine the initial CXR. Identify position of all hardware and look at lung fields and cardiac silhouette.

Perform a 12 lead ECG (and an atrial ECG if rhythm is unclear).

At the end of this process answer the following questions:

- *Is the child haemodynamically stable?*
- *Is the cardiac output adequate?*
- *Is the repair adequate?*
- *Are any complications present?*

GENERAL STRATEGIES IN EARLY POST-OP PERIOD

Analgesia/sedation

See full Analgesia and Sedation Guideline PICU Intranet

Targeted sedation – usually Comfort B 10-20.

Morphine 1mg/kg in 50 ml 5% dextrose (<10kg); 0.9% NaCl (>10kg). 0.5-3ml/hr = 10-60mcg/kg/hr. Additional sedation only if required (as per protocol): <12 months clonidine 0.5-2mcg/kg/hr; >12 months midazolam 1-2mcg/kg/min.

Antibiotics. Cephazolin 50mg/kg (max 2g) single dose prophylaxis given in theatre. Also for surgical procedures in ICU, including chest closure. No routine further doses.

Anticoagulation (see intranet protocol)

Heparin - Any patient with central line 10u/kg/hr, max 250u/hr.

BT shunt 10u/kg/hr, started immediately on admission.

Glenn/BCPC 10u/kg/hr. Fontan 15u/Kg/Hr. No APTT target, but ensure APTT<80s. Therapeutic anticoagulation for mechanical valve: APTT 60 – 80s.

Aspirin - Aortopulmonary shunt, RV-PA conduit, Glenn/BCPC, valve repair, PA patching, homograft valves/conduits 3-5mg/kg/dose daily when feeding. Stop heparin 6 hours later.

Clopidogrel – 3.0mm aortopulmonary shunt, in addition to aspirin.

Biochemistry

Glucose: May be low (newborns - limited reserve), or high (stress response). No evidence for tight glycaemic control.

Aim: 4-10mmol/L

Replacement: 2ml/kg 10% dextrose bolus and increase amount of glucose being given in fluids. If frequent administration required, start 50% dextrose 0.3ml/kg/hr and titrate to effect.

Dose being given (mg/kg/min) = (ml/hr x %dex)/(6xWt)

Hyperglycaemia: only treat if sustained (>6 hours in mid-teens) or if glucosuria and osmotic diuresis. Short-acting insulin 0.02-0.05u/kg/hr. Check glucose hourly and cease insulin when serum glucose<10mmol/L.

K⁺: **Aim:** 3.5-4.0mmol/L, 4.0-5.0 if arrhythmias.

Replacement: molar potassium 0.2-0.4mmol/kg/hr IV for up to 4 hours. Check K⁺ 1-2hourly.

Treatment of hyperK⁺ (>5.5 or rapidly rising): Remember acidosis produces hyperkalaemia. Check no K⁺ being given.

Give IV frusemide 1mg/kg, IV NaHCO₃ 8.4% 1 ml/kg IV over 30 minutes. Start PD. Other measures if still rising (rarely needed): resonium, dextrose/insulin, IV Ca gluc.

Ca²⁺: often low in first 24 hrs post CPB.

Aim: 1-1.2mmol/L, 1.2-1.3 if arrhythmias.

Replacement: 10% Ca gluconate 0.5ml/kg slow IV. If frequent replacement (?Di George), infuse at 0.1-0.4ml/kg/hr.

Mg²⁺: often low in first 24 hrs post CPB.

Aim >0.8mmol/L, 1-1.2 if arrhythmias.

Replacement: 50%MgSO₄ 0.2ml/kg slow IV. Beware hypotension.

Albumin: often low immediately post CPB, usually returns to normal after 12-24 hours. In infants, capillary leak and PD losses may prolong hypoalbuminaemia.

Chest drains

Underwater seal chest drains set at -10 to -20cmH₂O. Drains milked to encourage free drainage of intrathoracic fluid. Redivac drains have narrower, rigid tubing – difficult to milk. Watch for loss of vacuum.

Acceptable losses: up to 5ml/kg for first 2 hours, <1ml/kg thereafter.

Chest drain removal

Remove at surgeon's discretion – usually 4 days. Do not stop anticoagulation. Do not X-ray routinely post-removal.

Echo

Post-operative echo is not done routinely first night. Ask for the test if there is a specific question to answer (myocardial function, tamponade, residual lesion etc). Echo if escalating treatment for low cardiac output. All clinically indicated echos are done by on-call cardiology fellow.

Fluids

Total fluids:

Wt(kg)	3	5	7	10	15	20	25	30	40	50	60	70
ml/hr	5	7	10	14	17	21	25	28	32	40	45	50

Increase fluid as child recovers, up to full IV requirement when extubated (see Fluid and Electrolytes).

Maintenance: (<3mo) Plasma-Lyte 148, 10% dextrose; (>12mo) Plasma-Lyte 148, 5% dextrose.

Arterial line: (<2kg) heparin 1u/ml in 0.9%NaCl @ 1ml/hr; (2-20kg) heparin 5u/ml in 0.9%NaCl @ 1ml/hr; (>20kg) 2ml/hr.

Atrial, PA lines: heparin 1u/ml in 5%D @ 1ml/hr.

Fluid boluses

Give volume carefully; small aliquots (5ml/kg) with observation

and potential to repeat, rather than large boluses. Many patients will have significant increase in filling pressures with small volumes.

Fluid type: 0.9% NaCl if normal albumin; 4% albumin if low serum albumin; Plasma-Lyte 148 if hyperchloraemia.

Haematology

Hb and platelets: see Transfusion of blood products

WCC and ITR: both rise following surgery, peaking 24 hours post-op.

Coagulation: INR and APTT may be mildly deranged following CPB, normalizing with time. If bleeding aim for normal APTT and INR (see Post-op bleeding).

Inotropes/dilators/vasoconstrictors

- Dobutamine 2.5-7.5mcg/kg/min. First line to prevent/treat LCOS.
- Adrenaline 0.02–0.2mcg/kg/min. Additional inotrope if poor myocardial function
- Noradrenaline 0.02 – 0.2mcg/kg/min. Pressor if hypotensive and vasodilated
- Sodium nitroprusside 0.5-3mcg/kg/min. Vasodilator if poor myocardial function and preserved blood pressure.
- Milrinone 0.25-0.75mcg/kg/min Long acting vasodilator if poor myocardial function and preserved blood pressure; LCOS if unacceptable tachycardia with dobutamine.

Lines

- Arterial line: <2kg heparin 1u/ml @ 1ml/hr; 2-20kg 5u/ml @ 1ml/hr; >20kg 5u/ml@2ml/hr.
- Atrial and PA lines: heparin 1u/ml @ 1ml/hr.
- LA line enters heart through LA appendage or R upper pulmonary vein. Common atrial lines enter through R side of atrium. These lines have significant intrathoracic extracardiac course - risk of migration out of the heart with cardiac and lung movement. Do not access LA line for sampling.

Neuromuscular blockade

Only in severe pulmonary hypertension or haemodynamic instability. Intermittent vecuronium 0.1mg/kg. Always with deep sedation – Comfort B \leq 10.

Open sternum. Delayed sternal closure routine in some patients (post-Norwood) and common in other neonates following complex or problematic operations. Keep well sedated (Comfort-B 10-15) but not paralysed unless otherwise indicated. Do not give antibiotics routinely. Closure usually occurs in PICU post-op day 1-2.

Pacing wires

Temporary pacing wires are sutured onto the epicardium and exit skin through lower mediastinum. *Conventions:* blue=atrial, white=ventricular; if wires exit separately, R side=atrial, L side=ventricular. Do not touch uninsulated wires without gloves (microshock risk). Connect wires to pacemaker in all neonates and all patients with intraoperative arrhythmias or heart block.

Peritoneal dialysis (PD) catheter

PD catheter placed routinely in all neonates and most infants. Catheter usually sits in RUQ and may be palpable. Catheter not on free drainage when admitted. Allow drainage if significant 3rd spacing and ascites. For PD use, see oliguria and PD (qv). PD catheter is removed by cardiac surgical fellow when no longer required.

Removal of intracardiac lines and pacing wires

Remove pacing wires and intracardiac lines post-op day 4, regardless of presence or absence of chest drains. Cease heparin for 4 hours before removal, then check coags only if therapeutic anticoagulation. Check echo afterwards.

Routine blood tests. For initial tests, see above.

ABG 2-4 hrly, venous sat (best from: PA [SvO₂], SVC [ScvO₂] then RA, IVC [sats vary with position of line]) 4-6 hourly on the first night, more often if indicated. Albumin, magnesium, phosphate, creatinine and FBE daily for 2 days, then as indicated.

Clotting studies and liver function tests are checked on the first post-op morning, and thereafter only if indicated.

Transfusion of blood products

All cellular blood products to PICU are irradiated, except emergency O-ve blood and massive transfusion protocol.

Packed red cells. Post-op transfusion targets depend on age, circulation and severity of illness. Try to avoid transfusion in patients who have had a clear CPB prime (>8kg) and no exposure to donor blood. Transfusions in ICU are associated with worse outcomes; a lower Hb is increasingly tolerated in a stable child.

Suggested thresholds in stable child:

	2V
repair	UniV
repair	
Newborn	10
	12
Infant	8

Child 10
7
8-10

Transfusion may be useful to increase oxygen delivery in low cardiac output states, and to reduce left-to-right shunting in infants.

Pump blood. Heparinised blood collected from CPB circuit at end of bypass (Hb~100). Give within 6hr of return from theatre. Give 1mg of protamine for every 25 ml pump blood transfused (beware hypotension).

Platelets. Platelet function compromised by CPB so transfuse platelets (10ml/kg) if bleeding. Tolerate platelets >50,000 if no bleeding.

Ventilation

Most children admitted following cardiac surgery have normal lungs. Use sufficient PEEP to avoid atelectasis and minimise PVR and ventilate to normocarbia. Positive pressure ventilation improves cardiac output by reducing LV afterload. High pressure ventilation may increase RV afterload and may also reduce cardiac output by impairing venous return.

Suitable initial ventilator settings

<10kg pressure-limited; >10kg pressure or volume limited.

Rate 20-30 (<5yr), 15-20 (5-12yr), 12-15 (>12yr); PIP 15-25 cm H₂O; PEEP 5cm H₂O; tidal volume 6-8 ml/kg; trigger sensitivity <1L/minute; fresh gas flow 2-3 L/kg/min (newborn), 1-2 L/kg/min (child); FiO₂ 0.5.

Blood gas targets

In most cases, PaCO₂ 35-40mmHg and PaO₂ 95-100 mmHg is appropriate: aim for FiO₂ <0.5 and PIP <20.

In children with a known R-to-L shunt (eg BT shunt, Norwood), a PaO₂ of 95-100 usually indicates excessive pulmonary blood flow. In most of these patients aim for PaO₂ 35-40 mmHg and saturation 70-80%.

See notes on individual lesions for specific ventilation strategies (BTS, Norwood, Glenn, Fontan....)

Difficulty with achieving target gas exchange requires investigation. Start with examination and CXR: ensure airway patent, ETT in normal position, adequate ventilation, no collapse, consolidation, effusions, pneumothorax, and abdominal distension. Consider inadequate pulmonary blood flow as a problem (pulmonary hypertension, mechanical obstruction).

CARDIAC - POST-OPERATIVE PROBLEMS

CARDIOVASCULAR

Bleeding

More than anticipated chest drain (qv) losses. Drainage bloody, not haemoserous. Blood and clot may accumulate in pleura and mediastinum – tamponade (qv).

Causes: thrombocytopenia; poor platelet function; dilution or consumption of clotting factors; residual heparin (early post-op); hole in a blood vessel.

Investigation and treatment: Priorities are aggressive treatment of coagulation abnormalities and early communication with surgeon. Check coags, ACT, FBC. Consider tamponade/pleural collection – CXR, echo if signs. Give platelets 10ml/kg if platelets <150, protamine 0.5mg/kg if ACT>120s, FFP 10ml/kg. Cryoprecipitate if fibrinogen <1.5g/L. DDAVP if recently on aspirin. Recheck labs, aim to normalize. If ongoing bleeding and not returning to theatre consider tranexamic acid 100mg/kg over 1 hr, infusion at 10mg/kg/hr. Talk to surgeon again. Talk to haematologist about rFVII.

Bradycardia

Relative bradycardia common. Limits cardiac output.

Causes: Cold, AV block (qv), sinus bradycardia, drugs (β blockers, clonidine, antiarrhythmic drugs), acutely failing myocardium, hypothyroidism.

Investigation and treatment: check temperature, rhythm and conduction (ECG), drugs. Treat identified causes. If relative sinus bradycardia in early post-op period, pace to increase output (qv).

Coronary ischaemia

Any evidence of coronary ischaemia on monitoring must be investigated further. These problems are usually mechanical in nature.

Causes: manipulation of coronary arteries (ASO, Ross, Bentall), Ao V surgery, coronary compression (RV-PA conduit, PA band), coronary abnormalities.

Investigation and treatment: obtain ECG and Troponin I (uninterpretable immediately after surgery, but important baseline). Ensure adequate coronary perfusion pressure. Inform ICU consultant and cardiac surgeon. May need urgent intervention (chest opening/ECMO/theatre/cath).

High atrial pressures

Causes: awareness/pain, non-compliant atrium (LA in TAPVD, Shone's), myocardial dysfunction, AV stenosis or regurgitation, loss of AV synchrony (JET, CHB – qv), over-filling, tamponade (qv), tension pneumothorax (qv).

Investigation and treatment: Do not miss tamponade (qv) – high atrial pressures, tachycardia, hypotension with low pulse pressure. Ensure number is real (transducer), line in correct place (CXR, waveform), adequate sedation. Check waveform (canon waves in CHB, big a-wave in AV stenosis, big v-wave in AV regurg or tip at AV valve, damped if tip out of heart). Confirm rhythm (ECG). Echo if suspected tamponade, myocardial dysfunction or unexplained. Treat identified causes. Treat over-filling with diuretics, vasodilation or judicious removal of blood.

Hypertension

General causes: awareness, pain, agitation, seizures, vasoconstriction (drugs, cold)

Specific causes: after coarct repair (qv) or other LVOTO relief, BCPS (qv) or cardiac transplant (qv).

Investigation and treatment: ensure adequate analgesia and sedation, exclude seizures. If hypertension persists use short-acting vasodilator (SNP 0.5-3mcg/kg/min). Treat ongoing hypertension with short-acting beta blocker (esmolol 50-250mcg/kg/min) unless contraindication (bradycardia, myocardial dysfunction, asthma). Convert to bolus drugs when stable.

Hypotension

Causes: hypovolaemia; bleeding (qv); LCOS (qv); peripheral vasodilatation; drug delivery (inadequate inotrope/constrictor, bolus vasodilator); aortopulmonary runoff (through shunt, MAPCA, AV fistula); anaphylaxis.

Investigate and treat as per LCOS (qv). Use vasoconstrictor to treat excessive vasodilation (emergency - metaraminol 0.01mg/kg bolus, ongoing - noradrenaline 0.05–0.2mcg/kg/min infusion). Increase PVR if aortopulmonary runoff (FiO₂ 0.21, PEEP up to 10cmH₂O, PaCO₂ 45-55mmHg, Hb 140-160g/dl).

Low cardiac output syndrome (LCOS) (see intranet protocol)

Post-op oxygen delivery/consumption mismatch.

Clinical signs: tachycardia, hypotension, poor perfusion, oliguria.

Laboratory markers: high lactate (rising or >4mmol/L), low SvO₂ or ScvO₂ (<60%), increased SaO₂-S(c)vO₂ difference (>35%).

Causes: decreased myocardial function post surgery; fever; sepsis; arrhythmia; residual lesions; hypovolaemia; tamponade; pulmonary hypertension; drug delivery problem.

Investigations: clinical examination, review CXR, 12 lead (+/- atrial) ECG, echo (function/residual lesion/tamponade). Recheck

zeroing of transducers, delivery of drugs, patency of lines.

Treatment (see intranet protocol)

Optimise preload with adequate filling. Avoid hypovolaemia.

Reduce afterload if vasoconstricted. Add a vasodilator; use SNP (short half-life) initially.

Optimise contractility: AV synchrony, appropriate heart rate, treat tachyarrhythmias. Increase inotropy if poor function and no other cause.

Minimise O₂ consumption with deep sedation. Consider paralysis. Avoid fever and consider active cooling (min 34°C) to reduce oxygen consumption.

Notify ICU consultant early. Notify cardiac surgeons if persistent problem or rapid deterioration. Refractory cases may require chest opening in ICU or ECMO.

Pulmonary hypertension

Pre-op risk factors: newborn, high pulm blood flow (big L-R shunt), pulmonary venous hypertension (L heart obstruction).

CPB increases PVR & reactivity.

Episodic crises often with stimulation, ETT suction.

Signs: rising PAP, falling LAP & BP, tachycardia, hypoxia.

Treatment: Deep sedation, plus bolus sedation and paralysis for suction/cares. Consider regular neuromuscular blockade.

Provide inotropy for RV. Start iNO (20ppm) if episodes are haemodynamically significant. Ensure adequate ventilation, aim for PaCO₂ 30-40mmHg. Once stable, stop relaxants and bolus sedation. Then lighten sedation and wean ventilation. Reduce iNO over 12 hours to 2 ppm, then give sildenafil 0.4 mg/kg once and increase FIO₂ by 0.2 before ceasing iNO. Consider regular sildenafil in ongoing pulmonary hypertension.

Tachycardia

An important sign that something is wrong.

Causes: LCOS (qv), drugs (catecholamines), central (fever, pain, seizures), arrhythmia (qv), pulmonary hypertension (qv), tamponade (qv), anatomy (small LV), hypoxia, hypovolaemia, anaemia, hypoglycaemia.

Investigation and treatment: Check sedation, temp, haemodynamics, rhythm (ECG), ventilation (CXR, ETT suction, ABG), glucose, Hb. Echo if suspicion of pulmonary hypertension, LCOS or tamponade. Treat identified cause.

Tamponade

Accumulation of pericardial or mediastinal fluid compresses the heart, progressively limiting venous return and cardiac chamber size, reducing cardiac output.

Signs: tachycardia, high atrial pressures, hypotension, low pulse pressure, poor perfusion, acidosis, oliguria.

Causes: accumulation of blood in the post-op period. Beware drainage that suddenly stops – drain blocked.

Investigation and treatment: look for clinical signs. Milk chest drains, make sure they are on suction. Check ABG and coags. Echo and CXR if time allows. Tamponade is a clinical diagnosis and if rapidly progressive is a true emergency: call the ICU consultant and cardiac surgeon. Fluid boluses temporarily improve cardiac output.

Note that myocardial swelling may produce a tamponade-like picture, without significant collection of fluid or clot, particularly in neonates. Chest opening alone may be sufficient to improve cardiac output.

RESPIRATORY

Atelectasis

Causes: thick secretions; inadequate humidification, tracheal suction or PEEP; airway compression; tracheobronchomalacia (qv); phrenic nerve palsy (qv).

Investigation and treatment: check humidifier and tubing, hand-ventilate, suction with saline, Review PEEP, culture BAL, physiotherapy. Consider investigation for phrenic nerve palsy or tracheobronchomalacia (qv).

Chylothorax

Causes: damage to thoracic duct, high systemic venous pressure, venous thrombosis, pulm venous engorgement

Investigation and treatment: diagnosis - drain fluid triglyceride >1.1 mmol/L (if fed) & WBC >1000/ μ L, >80% lymphocytes. US neck veins for venous clot. Start enteral, low fat, MCT feed (Monogen). If high volume drain losses: change to TPN; Replace 50-75% of losses with 4% albumin; check albumin, protein, FBC, coags daily and immunoglobulins weekly. Give IVIG if low. Beware sepsis. Monitor for clot formation. Consider ligation of thoracic duct if persistent (>1 week) or pleurodesis.

Hypoventilation

Signs: rising PaCO₂, tachycardia, sweating, desaturation, rising PAP, BP may rise (hypercarbia) or fall (impaired myocardial performance).

Causes: drugs, brain injury, tracheal secretions, atelectasis, pneumothorax, pulmonary oedema, chest wall oedema, inadequate ventilation (leak in circuit, around ETT), abdominal distension.

Investigation and treatment: examine chest and abdo, ABG, hand-ventilate, suction ETT; CXR, check ETT and ventilator circuit for leaks, check ventilator settings, alter ventilation as necessary, aspirate NG tube, drain ascites.

Hypoxaemia

Respiratory causes: (pulmonary venous desaturation) lung disease, atelectasis, pneumonia, hypoventilation, pulmonary oedema, pneumothorax. *Cardiac causes:* (R – L shunt) – intracardiac (PFO, A/VSD), extracardiac (PDA, Ao-pulm shunt, SVC to IVC in BCPC), intrapulmonary (pulm AVM in BCPC). *Investigation and treatment:* Examine chest, ventilation, check sat probe. Verify with ABG. CXR. Ensure adequate ventilation and FiO₂. Treat identified causes. If cardiac cause suspected, check echo +/- bubble study, discuss cath or CT angio with cardiology.

Phrenic nerve palsy (paralysed hemidiaphragm)

Causes: surgical injury to phrenic nerve. Usually unilateral. *Investigation and treatment:* Suspect if high hemidiaphragm on CXR or no upper abdominal protrusion with spontaneous inspiration. Screen during spontaneous breathing with image intensifier (or ultrasound). Indications for plication: newborn with failed extubation or not extubatable and no other cause; older child if 2-3 failed extubation attempts.

Tachypnoea

Causes: pain, fever, sepsis, lung disease, pneumothorax/effusion, small lung volumes (CDH, Ebstein), pulmonary venous hypertension, cardiac failure, high pulm blood flow, metabolic acidosis, weakness, phrenic nerve palsy (qv). *Investigation and treatment:* examine (chest, abdomen, pupils, muscle tone and power, distress, ventilator synchrony); review HR, BP, toe temp, urine output; ABG; hand-ventilate & suction trachea; repeat CXR; consider trial bolus sedation; FBC & ITR, procalcitonin, culture blood, urine, BAL and drain fluid. Increase ventilation (IMV rate or support pressure) if muscle weakness present. Screen diaphragm if suspicious. Echo if indicated. Treat identified causes.

Tracheobronchomalacia

Suspect if likely lesion (APVS), prolonged expiration, hyperinflation, episodic desaturation on weaning. *Causes:* extrinsic compression (APVS); intrinsic malacia. *Investigation and treatment:* bronchogram (spontaneously breathing, ETT high in the trachea, varying CPAP). Treat with high enough CPAP to stent airway. Slow (weeks) wean with sedation. Consider tracheostomy and CPAP.

Ventilator dependence

Inability to wean from ventilation often multifactorial in chronic patients in cardiac ICU.

Causes: central – excess sedation, delayed excretion of drugs, encephalopathy, hypercarbia; *peripheral* – neuromuscular weakness (residual relaxant drugs, ICU myopathy, steroids, deconditioning and muscle loss); *airway* – vocal cord palsy (qv), post-extubation stridor (qv), tracheobronchomalacia (qv); *respiratory* – atelectasis, pneumonia, effusion, pneumothorax, pulm oedema, diaphragmatic palsy (qv); *cardiac* – residual lesion, cardiac failure.

Investigation and treatment: examine level of sedation, encephalopathy signs, respiratory drive, muscle strength. CT/MRI if abnormal neurology. Check leak around ETT, CXR, diaphragm movement (+/- screening), respiratory pattern. Nutritional assessment, appropriate feeding. Echo if evidence of residual lesions or myocardial dysfunction, may need further imaging and intervention. Treat identified causes. Young infants who have had a long complex stay may need a period of low level respiratory support while they grow and recover from surgery.

Vocal cord palsy

Causes: surgical damage to recurrent laryngeal nerve (usually L), 50-60% of Ao arch repairs.

Investigation and treatment: Suspect if arch surgery, hoarse voice, stridor, tachypnea, difficulty feeding. Diagnosis - flexible laryngoscopy (ENT). Tube feed, refer to speech pathology. May require CPAP/HFNP.

GI/GU

Abdominal distension

Causes: air in stomach or bowel; capillary leak, oedema and ascites; PD fluid; chyle; NEC (qv).

Investigation and treatment: examine chest and abdo; aspirate NGT; check PD drainage or release fluid; AXR & CXR, especially if tense/tender abdo. Leave PD on free drainage. Increase ventilation if necessary.

Oliguria and PD

Causes: ADH secretion following CPB, hypovolaemia, LCOS (qv), renal injury (cross-clamp, haemolysis).

Investigation and treatment: Oliguria is normal after surgery. Poor response to diuretics in first 12 hours. Do not use PD just to remove fluid in haemodynamically unstable infant early post-op. Start frusemide post-op day 1. PD used for control of hyperkalaemia, active cooling, fluid removal if required, short-term renal support. Start hourly cycles (40 minute dwell), 10ml/kg, 1.5% dialysate. More concentrated solutions will remove more fluid. Unstable neonates may have haemodynamic

changes with filling and emptying. Renal US for prolonged (>3 days) renal dysfunction. Post-op, post LCOS renal failure almost always recovers, but occasionally takes weeks.

Necrotising enterocolitis (NEC)

Risk factors: enteral feeding; newborn with single ventricle, left-sided obstruction or aortopulmonary shunt.

Investigation and treatment: AXR, refer general surgery. Stop feeds. Antibiotics (benzyl penicillin, gentamicin, metronidazole) 7 days. Daily AXR. Watch for perforation.

GENERAL

Fever

Causes: fever following cardiac surgery ("post-pump fever") now unusual. Think about other causes: SIRS, coincident infection (URTI), drug/transfusion reaction. Significant consequences in immediate post-op period: tachycardia, vasodilation, increased VO_2 . Fever several days after surgery is sepsis (qv) until proven otherwise.

Investigation and management: get pre-op history, examine chest and haemodynamics. CXR, ETT secretions. Blood cultures, BAL (qv), other cultures. FBC, ITR and procalcitonin. Both ITR and procalcitonin rise after cardiac surgery and peak on POD1. Treat excessive vasodilation with vasoconstrictors. Cool to normothermia in immediate post-op period. Treat sepsis (qv) if indicated.

Seizures

Seizures often difficult to identify in early post-op period, with only hypertension, tachycardia and pupillary changes.

Causes: cerebral hemorrhage, hypoxia, ischaemia, embolism of thrombus or gas.

Investigation and treatment: examine tone, suppressibility of movements. Look for focal movements or deficits. Treat suspected seizures with IV midazolam bolus, recurrent seizures with levetiracetam or phenobarbitone. Check glucose and electrolytes. Perform CT brain urgently if decreased conscious state or abnormal neurological signs. Consider EEG if unsure of seizure diagnosis.

Sepsis

High-risk group with multiple barrier breaches, poor nutrition, implanted foreign material, immunosuppression (therapeutic, chylothorax, Di George).

Signs: Fever, vasodilation or poor perfusion, decreased cardiac output, hypotension, oliguria, lactic acidosis, increased PAP.

Rising procalcitonin, ITR, falling platelets.

Investigation and treatment. Examine chest, abdo, skin. Culture blood (CVC, art, percutaneous), urine, BAL, pleural fluid, peritoneal fluid. Swab red/oozing wounds, drain sites. Consider echo for endocarditis, LP, CT (mediastinal/abdo collection) Start antibiotics promptly (flucloxacillin and gentamicin initially for unknown organism), refine with further results. Stop after 48 hours if clinically resolved and cultures negative. Increase inotropic/pressor support as indicated. Review cultures, FBC, ITR daily. Consider deep-seated infection (mediastinitis, endocarditis, bone, sinus) and fungal infection in longer stay patients on antibiotics.

CARDIAC - TACHYARRHYTHMIAS

Treat unstable patient with wide QRS tachycardia as VT - synchronised DC shock.

Sinus tachycardia is more common than tachyarrhythmia.

Investigation

ECG: Do 12 lead ECG and compare to preop and immediately postop. Run limb lead rhythm strip with notation during diagnostic & therapeutic manoeuvres.

Atrial ECG: Connect one atrial pacing wire to the RUL ECG electrode and the other atrial wire to the LUL electrode, then run a rhythm strip on the ECG machine. Lead I is a bipolar atrial ECG, II & III are unipolar. This will help to identify the P wave during a tachycardia, and demonstrate the association between Ps and QRSs.

Answer the following questions:

Are the QRS complexes regular? Irregularity in 2nd degree heart block and atrial or ventricular ectopy.

Are the QRS complexes wide? Wide QRS suggests VT. QRS may be widened post-op (RBBB post TOF repair) and occasionally during supraventricular tachycardias (rate-related aberrant conduction).

Is there a visible P wave for every QRS complex? (sinus tachy, EAT, re-entry SVT, JET with retrograde conduction) *If not, are Ps faster than QRSs, or vice versa?* (P>QRS – atrial flutter with block; P<QRS – JET, VT).

Is the P wave morphology the same as previous ECGs? (abnormal in atrial flutter, EAT, retrograde conduction)

General measures in all tachycardias: normothermia; adequate sedation and analgesia; K⁺ 4-5mmol/L; iCa²⁺ 1.2-1.3mmol/L; Mg²⁺ 1-1.2mmol/L; reduce/cease contributing drugs (pancuronium, catecholamines).

Junctional ectopic tachycardia (JET) automatic tachycardia arising from AVN/His bundle. Narrow complex, AV dissociation (QRS>P) or retrograde conduction (P follows QRS). Self-limiting. General measures plus incremental cooling to min 34°C. Aim to slow JET, then pace above JET rate (AAI if AV conduction intact, DDD if not). Amiodarone if persistent and unstable – discuss with consultant. Back-up pacing (DDD, DVI, VVI) in case of heart block. ECMO if refractory LCOS.

Ectopic atrial tachycardia (EAT, AET) automatic tachycardia arising from atrium remote to SA node. Narrow complex, AV synchrony, abnormal P morphology.

Treatment: general measures – very sensitive to catecholamines. β -blocker or amiodarone may suppress automaticity – discuss with ICU and cardiology consultant.

Re-entrant SVT reentry circuit involving AV node and accessory pathway. Abrupt onset, narrow complex, AV synchrony, P follows QRS, abnormal P morphology, may have pre-excitation in sinus rhythm.

General measures. Try to interrupt re-entry circuit: vagal manoeuvres (ice to face, gag, ventilated Valsalva - hold inspiratory breath); rapid atrial pacing (qv); adenosine. If hypotensive and unstable synchronized DC shock 0.5J/kg.

Atrial flutter Re-entry circuit within atria, often 2:1 block at AV node. Narrow complex, P:QRS \geq 2:1. Adenosine diagnostic - increases block, unmasks flutter waves. Interrupt circuit by rapid atrial pacing (qv) faster than atrial rate (may be >300). If unsuccessful, DC shock. Amiodarone helpful if recurrent.

Ventricular tachycardia (VT)

Wide complex, QRS>P, may see capture beats, often unstable. Think about ischaemia, hypoMg²⁺, hyperK⁺, cardiomyopathy, myocarditis, channelopathies. Synchronised DC shock. Lignocaine 1mg/kg IV bolus, then 20-50mcg/kg/min. Consider amiodarone if resistant. Give Mg if torsades de pointes. Discuss with consultant.

Heart block

1st degree common, 2nd uncommon. Set backup ventricular pacing in case of progression to complete AV block (CHB). CHB: ensure secure pacing (DDD, DVI, VVI); treat expectantly for 1 week before permanent pacemaker; watch V wire thresholds. If no wires, use isoprenaline, transcutaneous pads, transvenous pacing wire.

TEMPORARY CARDIAC PACING

Definitions

Mode nomenclature. Chamber paced / chamber sensed / response to sensing

AV delay. Interval between sensed or paced atrial stimulus and delivered ventricular stimulus.

Maximum tracking rate. Maximum sensed atrial rate at which 1:1 ventricular pacing will occur (DDD).

Post ventricular atrial refractory period (PVARP). Period following paced/sensed ventricular event when atrial sensing will not occur (DDD).

Sensitivity. Level of electrical signal from atria/ventricles that the pacemaker will sense.

Threshold. Lowest output from the pacemaker that will consistently pace the atria/ventricles.

Common uses

Sinus bradycardia. AAI if intact AV conduction, DDD if AV block, DVI if poor atrial sensing, VVI if no atrial wires.

JET (once slow enough). AAI if intact conduction, DDD or DVI if AV block.

Complete heart block. DDD. VVI if poor atrial sensing.

Rapid atrial pacing (RAP). To terminate reentrant SVT or A Flutter/IART. Put pacemaker in RAP mode. Pacemaker continues to deliver previous settings until 'Start' is pushed. Set RAP rate 10-15% higher than tachycardia rate (atrial rate in flutter/IART). Press and hold 'Start' for 5-10s, then release. AAI delivered at RAP rate.

Pacemaker checking

Underlying rhythm. Turn rate down below native rate.

Sensitivity. Set rate below native rate. Start at lowest 'sense' setting and turn up until pacemaker fails to sense (paces instead). Set sensitivity at half this level. Do not check ventricular sensitivity in CHB.

Threshold. Set rate faster than native rate. Start at high output ('stim') setting, capturing appropriate chamber. Turn output down until loss of capture. Set at 2-3 times this number.

Pacing with a single ventricular wire. Use V wire in negative V terminal of pacemaker, insert skin wire or use atrial wire for positive terminal. VVI.

Troubleshooting

All sensing or pacing problems. Check electrical circuit: wires – connectors – leads – correct pacing terminals.

Failure to sense (usually atrial). Reduce sensitivity. Reverse lead polarity.

Failure to pace (no output). Hardware problem (pacemaker or circuit). Oversensing – increase sensitivity.

Failure to capture. Increase output, increase pulse duration, reverse lead polarity.

Pacing the diaphragm. Looks like hiccoughs at pacing rate. Often due to atrial output and position-dependant. Reduce output. Reverse polarity. Reposition patient.

CARDIAC - BY LESION / OPERATION

ALCAPA

ANOMOLOUS ORIGIN OF LEFT CORONARY ARTERY FROM PULMONARY ARTERY

LV dysfunction and ischaemia. MR almost always present.

Repair once diagnosis made. LCA direct transfer to aorta or via intrapulmonary aortocoronary tunnel (Takeuchi). ALCAPA much less common.

Important points

Don't miss this diagnosis in infant presenting with heart failure ?cardiomyopathy / myocarditis. Very sick pre-op. LV dysfunction and MR persist following repair. Usually return from theatre on ECMO/VAD.

Specific post-op problems

MR and LV dysfunction. Manage with inotropy and dilation.

Coronary ischaemia (qv).

Arrhythmias. LV may be a source of ectopy and VT (qv).

Usual course

ECLS 2-4 days. Inotrope and dilator to decannulate and continue through extubation (post-op day 4-6).

ATRIAL SEPTAL DEFECT

(ASD)

Extubated in theatre or soon after return to ICU (within 4 hours).

Inotropes not usually necessary. Complications (arrhythmia, bleeding, effusion) very uncommon. Discharge to ward same day/next morning.

AVSD

ATRIOVENTRICULAR SEPTAL DEFECT

Pre-op problems

Cardiac failure due to big L to R shunt, worse if severe AV valve regurgitation or L-sided obstruction. Pulmonary hypertension, worse if trisomy 21. Treat cardiac failure with diuretics, transfusion to Hb140-160, positive pressure ventilation.

Complete repair usually at 2-4 months.

Unblanced defect: common AV valve opens predominantly into one ventricle. May progress down single ventricle route, depending on degree of hypoplasia of non-dominant ventricle.

Specific post-op problems

Pulmonary hypertension (qv) (NB trisomy 21), JET (qv), CHB (qv), residual defects (shunt or AV valve regurgitation).

Usual Course

Sedation and observation for first 4-6 hours, then wean. Aim to extubate following day in uncomplicated repair. Modify approach if any of above complications present.

BIDIRECTIONAL CAVOPULMONARY SHUNT

(BCPS/bidirectional Glenn)

All SVC blood flows passively through pulmonary arteries, bypassing heart. Usually no other source of pulmonary blood flow. Removes volume load from ventricle. Usually done at 3 months as part of single ventricle pathway.

Important points

Intracardiac mixing– expect SpO₂ 70–85%. Positive pressure ventilation increases intrathoracic pressure and decreases pulmonary blood flow. Use low mean airway pressure. PEEP 5 to avoid atelectasis.

Specific post-op problems

Cyanosis. Causes: i) pulmonary; ii) increased pulmonary vascular resistance; iii) venovenous (SVC to IVC) collaterals; iv) obstruction to SVC-PA flow; v) low cardiac output (low SvO₂).

Look for high SVC pressure and clinical signs of SVC obstruction. Ensure no reason for high PVR (acidosis, hyperinflation, atelectasis, pneumothorax etc.). Echo to look at ventricular function, AV valve, anastomosis and PAs. Bubble study to identify SVC to atrial shunting, more formal imaging (catheter or CT angiogram) to clearly define VV collaterals or BCPS obstruction. Ensure adequate, low pressure ventilation and inotropy for impaired ventricular function. Allow PaCO₂ to rise to 50 (increases cerebral and SVC blood flow, improves oxygenation), but avoid acidosis. Trial of iNO if persistent signs of increased SVC pressure.

Systemic hypertension. Due to increased SVC pressure. Treat with vasodilator, many need ongoing ACE inhibition.

Headache. Patients are often sore and irritable, again probably because of high SVC pressure.

Usual course

Extubated first post-operative night, low inotrope requirement, remove IJ line post-op day 1. Start aspirin when feeding.

FONTAN

All systemic venous blood diverted to flow passively through lungs. Extracardiac conduit from IVC to underside of RPA. Final operation in single ventricle pathway, usually after a BCPC. Done at ~4years old.

Important points

Transpulmonary gradient = SVC-atrial pressure. Fenestration between conduit and atrium is only point of mixing. R-L fenestration flow ensures ventricular filling in the face of high PVR. SpO₂ usually 80s first night, rising to 90s over next few days. Positive pressure ventilation increases intrathoracic pressure and decreases pulmonary blood flow. Use low mean airway pressure. PEEP 5 to avoid atelectasis. Heparin 15u/kg/hr.

Specific post-op problems

Poor fontan flow. High venous pressures, big liver, increased chest drain losses, cyanosis. Causes: low cardiac output, increased pulmonary vascular resistance, conduit obstruction. Check ventilation, CXR. Bag & suction. Ensure no reason for high PVR (acidosis, hyperinflation, atelectasis, pneumothorax etc.). Echo to look at ventricular function, AV valve, Fontan conduit, fenestration and PAs. Treat respiratory causes and acidosis. Inotropes for ventricular dysfunction. iNO if persistent evidence of increased PVR.

Cyanosis. Causes: pulmonary, poor fontan flow (see above); pulmonary AV malformations (develop in some patients with BCPC). Investigate as above. Check preop cath for evidence of pulmonary AVMs.

Effusions. Ongoing high volume effusions may need partial replacement with intravenous 4% albumin. Chylothorax (qv) common.

Usual course

Most return to ICU on 0.1mcg/kg/min noradrenaline. Wean this off over first 12 hours, using filling to maintain BP in the face of peripheral vasodilation. Early extubation (within 4 hours). To ward post-op day 1. Transition to warfarin later.

HYPOPLASTIC LEFT HEART SYNDROME

(HLHS)

Generally delivered locally and transferred early in good condition - breathing spontaneously, alprostadil, SpO₂ 80s.

Pre-op problems

Balancing circulation. Systemic blood flow via PDA. Usually good systemic output at birth, but may change as PVR drops in first few days. If signs of excessive PBF and inadequate systemic perfusion (tachycardia, tachypnea, high SpO₂, hypotension, lactaemia) require intubation, control of ventilation. Intubate with dobutamine, volume and senior assistance. Manage as per systemic to pulmonary shunt post-op problems (qv).

Restrictive atrial septum. These babies are cyanotic, tachypnoeic and become rapidly unwell after birth. Require

intubation and ventilation, with discussion of urgent septectomy/Norwood or comfort care.

Important points

Surgery performed at 3-7 days. Routine is Norwood with mBTS. Sano in wt<3kg. All return to ICU with an open sternum. Fragile circulation, sensitive to small changes in initial post-op period. Aim for SpO₂ 70-85%. Manage as per mBTS (qv), with tendency for LCOS (qv).

Specific post-op problems

LCOS (qv). Common post-op problem. Likely to need increase in inotropy over first night. Echo if concerned (RV function, AV valve regurgitation).

Balancing circulation. Treat pulmonary overcirculation as per systemic to pulmonary shunt post-op problems (qv).

Desaturation. Causes: i) pulmonary, ii) inadequate shunt flow – as per systemic to pulmonary shunt post-op problems (qv), iii) restrictive shunt (3.0 or wrapped 3.5).

Ischaemia. Watch for any evidence of coronary ischaemia, especially if aortic atresia with tiny ascending aorta – ECG, troponin, echo and discussion of further imaging.

Usual course

Well sedated for first night. Chest closure post-op day 1 or 2, extubate 1-2 days later. Discharge to ward post-op day 5-7. Anticoagulation as per mBTS. Many patients have more complex and slower course through ICU.

INTERRUPTED AORTIC ARCH

(IAA)

Complete interruption of arch, always with VSD. Duct-dependant systemic perfusion. Neonatal repair. Strong association with 22q11 microdeletion.

Important points

Often shocked at presentation. Big, unrestrictive VSD – SpO₂ similar pre & post ductus.

Specific post-op problems

Pulmonary hypertension (qv). *Arrhythmia:* JET (qv), CHB (qv). *Vocal cord palsy (qv).*

Left main bronchus compression. Hyperinflation/collapse of L lung. Investigate with bronchogram and CT angiogram. May need prolonged respiratory wean or aortopexy.

Usual course

Targeted sedation and inotropy first night. Wean ventilation post-op day 1, extubate post-op day 2-3.

PULMONARY ARTERY BAND

Fixed restriction to pulmonary blood flow (PBF). Usually i) in neonates with unrepairable L-R shunts, ii) to protect pulmonary circulation during a period of growth before further surgery, or iii) to prepare LV for switch in late presentation TGA.

Important points

Difficult to get tightness of band right. Usually loose initially, with relative tightening with growth. Can be quite unstable post-op, with acute increase in afterload to banded ventricle.

Specific post-op problems

Tight band. Poor ventricular function, ventricular arrhythmias/bradycardia, +/- desaturation. Resuscitate, echo, call cardiac surgery.

Loose band. High saturations, with ongoing high PBF. Aim to increase PVR (FiO₂ 0.21, PEEP 10, PaCO₂ 50, Hb 140-160). Slow wean of respiratory support.

Pulmonary hypertension (qv). Often have had high PBF pre-op, susceptible to sudden increase in PVR postop.

Band placement. Injury to MPA or pulmonary valve. Ischaemia suggests compression of a coronary artery.

Usual course

Well sedated for first night with additional sedation for suction. Weaning first post-op day, extubation 24-48 hours later. Loose band with high PBF needs slow wean of respiratory support.

PULMONARY ATRESIA, IVS

(PA, IVS)

Very variable anatomy, with degrees of RV hypoplasia, tricuspid valve dysplasia and coronary abnormalities. Neonatal surgical priorities are to secure PBF, either by valvotomy, mBTS or both, and to maximise potential for growth of RV. Ultimate repair may be uni/biventricular or mixed, depending on RV & tricuspid anatomy.

Important points

Duct-dependant PBF. Thick, hypertensive, non-compliant RV. Fistulous connections develop between RV and coronary circulation, creating potential for coronary circulation that is dependant on high RV pressure.

Specific post-op problems

Depends on surgery

Cyanosis. Exclude other causes, but may be poor PBF. Minimize PVR - ventilation and iNO as per pulmonary hypertension (qv).

Avoid vasodilators. May need mBTS if not already done.

Coronary ischaemia, if RVOT opened with RV dependant coronary circulation. ECG, echo, troponin. Give volume, increase SVR (noradrenaline), call cardiac surgery.

Usual course

Again, very variable, depending on course taken. Some infants may spend considerable periods cyanotic in ICU, waiting for forward flow through RV to increase.

SYSTEMIC TO PULMONARY SHUNT

(mBTS, central shunt)

Means of securing pulmonary blood flow in neonatal period (R-sided obstruction or single ventricle). Flow dependant on i) fixed resistance through goretex shunt (proportional to length and inversely proportional to 4th power of diameter of shunt), ii) driving pressure (BP) and iii) downstream pressure (PAP).

Important points

Complete intracardiac mixing – expect SpO₂ 70-85%. Listen to the shunt murmur with the stethoscope bell at open end of ET tube or over ipsilateral 2nd intercostal space. Start heparin 10u/kg/hr immediately on admission. Ensure adequate sedation, including bolus for suction etc. Continue dobutamine. Wean alprostadil over first post-operative night.

Specific post-op problems

Desaturation. Can be pulmonary (examine, bag & suction, CXR) or due to poor shunt flow (blocked shunt or pulmonary hypertension), Abrupt desaturation may be shunt blockage. Give volume, increase systemic blood pressure with inotrope or vasoconstrictor. Examine for change in murmur, urgent echo. If profound or persistent call cardiac surgeons (cardiac callout) and give 50-100u/kg heparin. Consider a trial of iNO if unrestricted pre-op pulmonary blood flow.

Excess shunt flow. Tachycardia, high SpO₂, low diastolic BP, lactaemia, cardiac failure. Excessive pulmonary blood flow at the expense of systemic perfusion and increased myocardial work. Increase PEEP to 10, decrease FiO₂ to 0.21, transfuse to Hb 140-160. Consider allowing CO₂ to rise to 50 (may need to paralyse). Discuss adding N₂ to further reduce FiO₂. Surgeons may reduce shunt size by 'wrapping' – constriction along the length of a goretex sleeve around outside of shunt. These children may spend a long time on positive pressure 'growing into' their shunt.

Usual course

Keep well sedated for first 12 hours, then wean. No need to paralyse unless unstable. Aim for extubation post-op day 1-2. Start aspirin when feeding, add clopidogrel if 3.0mm shunt.

TAPVD

TOTAL ANOMOLOUS PULMONARY VENOUS DRAINAGE

All pulmonary venous blood drains to R atrium. Connection between pulm veins and systemic venous system supra-, intra-, or infracardiac. R-L via PFO maintains LV output. Usually isolated, also occurs as part of complex defects.

Important points

Severity of presentation in isolated TAPVD depends on degree of obstruction to PVs. Infracardiac almost always severely obstructed – cyanosis, hypotension, pulm hypertension - surgical emergency. Unobstructed – mild respiratory distress, SpO₂ 80s-90s.

LA & LV often relatively small and non-compliant.

Specific post-op problems

Pulmonary hypertension (qv). Very common after repair of obstructed veins. Echo to check for residual obstruction.

Non-compliant LA/LV. High LAP, very sensitive to volume administration. Small LV cavity – relative tachycardia.

Chylothorax (qv). Common after repair of obstructed veins.

Secondary pulmonary lymphangiectasis. Consequence of obstruction. Ongoing tachypnea, ventilator dependency and CXR changes. Rule out residual/re-obstruction. Very slow wean of respiratory support

Usual course

Depends on pre-op state/obstruction. Well, unobstructed – similar to repair of L-R shunt. Sick, obstructed – deep sedation, manage pulmonary hypertension (qv), slow wean.

TETRALOGY OF FALLOT

(TOF)

Physiology depends on degree of RVOTO: minimal obstruction - L-R shunt, acyanotic; obstruction – R-L shunt, neonatal cyanosis, spells.

Important points

Symptomatic neonates get mBTS. Aim for complete repair after 3 months. Transatrial repair with attempt to preserve pulmonary valve. Systolic ventricular function usually good.

Pre-op problems

Hypercyanotic spells. Escalating treatment: oxygen, posture (head down/knee-chest), sedation, volume bolus, vasoconstrictor (metaraminol 0.01mg/kg), β-blocker (esmolol 0.5mg/kg over 1 minute).

Specific post-op problems

Restrictive RV physiology. Diastolic RV dysfunction leads to continuous forward flow through RVOT in diastole. Confirm with

echo. Ensure RAP adequate (low-mid teens), AV synchrony, low ventilation pressures.

Arrhythmia/heart block. Prone to JET (qv), CHB (qv).

Usual course

Systemic to pulmonary shunt (qv).

Total repair. Targeted sedation. Low dose inotropes. Avoid vasodilation. Wean after 6 hours if complication-free. Extubate 12-24 hours.

TOF VARIANTS

PULMONARY ATRESIA, VSD, MAJOR AORTOPULMONARY COLLATERALS (MAPCAs)

Variant of Tetralogy with atretic pulmonary valve, small central PAs and variable, tortuous connections between systemic and pulmonary arterial circulations (MAPCAs).

Important points

RCH approach is early (few weeks) shunt from ascending aorta to central PA (to increase flow and promote growth of native PAs), often with further shunt procedures before complete repair (VSD closure, RV-PA conduit) at ~2 years. MAPCAs are not usually ligated or translocated onto PAs (unifocalisation).

Specific post-op problems

High PBF post central shunt. Unusual. L-R via MAPCAs and shunt. High SpO₂, low BP. Treat as systemic to pulm shunt, excess shunt flow (qv).

Segmental lung infarction post MAPCA ligation. Unusual. Bloody secretions, segmental CXR changes, potential infection.

Restrictive RV physiology (qv) post complete repair.

Usual course

As per systemic - pulmonary shunt (qv) or TOF repair (qv).

ABSENT PULMONARY VALVE SYNDROME

(TOF APVS)

TOF variant with pulmonary valve leaflet agenesis, small valve annulus and extensive dilatation of PAs.

Important points

Course dictated by degree of dilation of pulmonary arterial tree and consequent bronchomalacia. RV dilation due to PR.

Newborns requiring intubation and ventilation for widespread disease have poor prognosis. TOF repair often includes plication of PAs.

TRANSPOSITION OF THE GREAT ARTERIES

(TGA)

Usually isolated lesion. Coronary anatomy important, but no contraindication to surgery. TGA with IVS often deeply cyanosed at birth. Arterial switch operation (ASO) within first 10 days, longer (weeks) if VSD. Late presentation of TGA IVS needs PA band to prepare LV for ASO.

Important points

Oxygenation depends on L-R intracardiac shunting (VSD or PFO/ASD). PDA increases PBF, loads LA, increases L-R shunt.

Pre-op problems

Cyanosis Increase L-R shunt with alprostadil and balloon atrial septostomy (BAS) in PICU.

BAS. Intubate and sedate for procedure. Inflammatory response often after BAS with hypotension and tachycardia – give volume, low dose vasoconstrictor/inotrope. Wean ventilation and extubate when stable, try to cease alprostadil over next 1-2 days. If cyanosis persists despite successful BAS, may need ECMO or immediate ASO.

Specific post-op problems

Coronary ischaemia (qv) Urgent investigation and discussion with cardiac surgeons.

LCOS (qv); Arrhythmia. JET (qv), other SVTs (qv). CHB (qv) or ventricular arrhythmia (qv) - manage dysrhythmia and exclude coronary problem.

Usual course

Targeted sedation, inotropy first night. Start weaning at 12 hours if stable. Wean off alprostadil and GTN by post-op day 1. Extubate post-op day 1-2.

TGA variants

CONGENITALLY CORRECTED TGA

LA-RV-Aorta, RA-LV-PA. Usually associated with VSD, also subpulmonary stenosis, tricuspid valve abnormalities. Surgery to avoid progressive RV failure and TR. Senning (qv) + ASO (qv) = 'double switch'. Timing of surgery difficult and variable. Preparatory PA banding improves RV and tricuspid valve function and trains LV.

Important points

Associated defects dictate clinical course. CHB (qv) and reentrant SVT common (abnormal conduction pathway).

Specific post-op problems

Rhythm disturbance. CHB (qv), SVT(qv).

Systemic or pulmonary venous obstruction. Watch atrial pressures, echo if concerned.

LV dysfunction. Check post-op echo. Adjust inotropes and vasodilators accordingly.

Usual course

If stable rhythm & LV coping with systemic circulation, wean overnight and extubate post-op day 1.

TRUNCUS ARTERIOSUS

Single arterial trunk to systemic and pulmonary circulations (always with VSD). May have arch hypoplasia/interruption. 22q11 microdeletion common. Develop pulmonary overcirculation rapidly as PVR drops. Neonatal repair: PA removed from trunk, VSD closed, RV-PA conduit.

Important points

Big pre-op L-R shunt predisposes to pulmonary hypertension (qv) post-op. Coronary anomalies in ~50%. Truncal (neoaortic) valve may be abnormal and regurgitant. AV bundle position variable.

Specific post-op problems

LCOS (qv) common. *Pulmonary hypertension* (qv).

Coronary ischaemia (qv). *Arrhythmias*. JET (qv), CHB (qv).

Usual course

Targeted sedation first night (deeper if complications). Wean post-op day 1, extubate day 2-4.

VENTRICULAR SEPTAL DEFECT

(VSD)

Small (particularly muscular) VSDs likely to close. Large or multiple VSDs create L-R shunt, heart failure and pulmonary vascular disease. Patch closure at 2-6 months. PA band in multiple defects or premature infants.

Important points

Early (neonatal) presentation with heart failure – look for L sided obstruction. Transfusion to Hb 14-16 will reduce L-R shunt in unoperated infant with failure.

Specific post-op problems

Pulmonary hypertension (qv). Particularly in later repairs, large defects or Trisomy 21.

Arrhythmia. JET (qv), CHB (qv)

Respiratory problems. Some infants get clinical small airways disease/gas trapping after repair of big L-R shunt (poor compliance, prolonged expiration). Unresponsive to bronchodilators, settles with time and diuresis.

Usual course

Small VSD, older child. No inotropes, rapid wean and extubate.

Large VSD, infant heart failure. Targeted sedation first night, dobutamine. Wean first post-operative day, extubate post-op day 1-2.

CARDIOMYOPATHY

(Also see Myocarditis)

Investigation (see intranet protocol) FBE, ESR, glucose, ABG, pyruvate, lactate, liver and thyroid function, Ca, phos, Mg, CK, brain natriuretic peptide, Selenium, Ferritin, troponin; carnitine and acylcarnitine (2ml Li hep and Guthrie card). Urine for metabolic screen and GLC for organic acids. Urine / NPA / stool and 5ml clotted blood (serology) for viral PCR and culture; mention adeno, enterovirus, flu, parafu, Parvovirus B19 and CMV on form. Do ECG, echo (2D and M), 24hr Holter, chest X-ray. Consult Metabolic Unit

Management

CPAP, BIPAP or mechanical ventilation. These reduce left ventricular wall tension and work of breathing.

Cardiac echo and CVP should be used to ensure optimal intravascular volume. A high CVP may be needed in hypertrophic cardiomyopathy.

Treat arrhythmias (consult a cardiologist). Consider biventricular pacing if LVEF \leq 0.35, sinus rhythm, QRS \geq 120ms.

Rotation of dobutamine, milrinone and levosimendan may be helpful in dilated cardiomyopathy; watch for arrhythmias.

Inotropes usually worsen *hypertrophic* cardiomyopathy – rarely used.

In *dilated* cardiomyopathy, vasodilators (milrinone, levosimendan) are desirable, but introduce slowly (especially if there is hypovolaemia). When stable, introduce captopril slowly. Give spironolactone. Frusemide may also be needed (avoid hypovolaemia).

Beta-blockers (Carvedilol) may be beneficial if tolerated. IV esmolol should be considered in patients with hypertrophic cardiomyopathy. Beta-blockers are illogical if on catecholamines. Ensure normoglycaemia. Maximise nutrition; consider TPN, especially if signs of gut hypoperfusion.

In ICU, give low-dose heparin and an antiplatelet drug.

Long term anticoagulation is indicated for those with LVEF $<$ 25%.

Other treatments used occasionally include carnitine, coenzyme Q10, Selenium and L-arginine.

Durable VAD support is indicated only for potential transplant candidates. Consider centrifugal VAD or peripheral ECMO for evolving end organ dysfunction if primary durable VAD insertion not possible.

CATHETERS - INTRAVASCULAR

ARTERIAL LINES

Never place an ulnar artery catheter in small children, prefer a radial or femoral. If you are going to insert a radial or ulnar artery catheter first confirm the presence of dual blood supply to hand and fingers by other arteries (by ascertaining the presence of both ulnar and radial pulses, and ideally by ultrasound). Remove any arterial line if distal blanching or cyanosis occurs. Get urgent plastic surgery consult if distal ischaemia, and heparinise unless contraindicated.

In a small infant, rapid replacement of the fluid aspirated from the dead space of an arterial line can cause retrograde blood flow in the artery with a substantial risk of cerebral embolism. To sample from an arterial line:

Remove cap and keep clean. If debris present, clean tap with sterile swab stick and alcohol before starting. Aspirate dead space using a 2 ml syringe; at least 0.7 ml is necessary to guarantee an undiluted specimen. Inspect line and aspirate any thrombi. Keep dead space syringe sterile. Collect samples as required. Do not take more blood than is absolutely essential for the tests required – this is particularly important in small babies. Replace dead space *slowly* via arterial line unless thrombi or debris visible in aspirate. The rate of replacement should not be greater than 1 ml in 10 seconds. Resistance to injection may be due to thrombosis, spasm, or malposition or kinking of catheter; this should be investigated. Forced flushing is hazardous. Replacement of dead space results in heparinised solution throughout the extension and catheter. Further flushing is unnecessary. Clean tap with sterile swab stick and replace cap.

CENTRAL VENOUS LINES

Internal jugular and subclavian central lines must not be inserted without ICU Consultant approval.

The use of central lines means a significant risk of complications, such as pneumothorax, haemothorax, haemopericardium, perforation of the great veins or heart, SVC and IVC thrombosis, septicaemia and endocarditis, all of which have occurred in this Unit. Thrombosis and vessel perforation are more likely with double lumen than with single lumen catheters. Before inserting a central line, consider:

Does this child REALLY need a central line (would peripheral lines be safer and cheaper)?

If TPN is the reason for the central line, could the child be fed enterally? (it is safer and cheaper)

If the child really needs a central line, would a safer, cheaper single lumen catheter with or without peripheral lines be better than a double lumen catheter?

In an emergency, central venous access can usually be achieved rapidly via the umbilical vein in babies up to 5-7 days of age. While the anterior fontanelle is open, it can be used to gain venous access in an emergency.

Right internal jugular central line (inserted midway between mastoid and sternal notch): depth = (height in cm) / 10.

Central lines (neck or femoral) carry a very high risk of thrombosis in children <5 kg – either avoid using a central line, or give heparin 10 u/kg/hr (unless contraindicated).

Check the pressure trace on all central lines after insertion to check that they are venous rather than arterial.

Put heparin 1u/ml in all monitoring lines (IA, CVC, RA, LA, PA). Do not put heparin in infusions with betalactams, dobutamine, erythromycin, morphine or vancomycin, fluid boluses or high volume infusions (>100 ml/kg/day), or in children with life-threatening bleeding.

Put dobutamine in 0.9% saline.

Put adrenaline, dopamine, frusemide, glyceryl trinitrate, morphine, noradrenaline and nitroprusside in 5% dextrose for neonates and 0.9% saline for older children.

Infuse heparin 1u/ml even if on warfarin or aspirin.

Heparin reduces the risk of thrombosis and bacteraemia with central lines.

Do *not* change CVCs routinely to prevent infection

Do *not* remove a CVC on the basis of fever alone

UMBILICAL LINES

Catheter sizes:

Weight
size
size

UAC
UVC

<1.5kg	3.5Fr
, double lumen	3.5Fr
>1.5kg	5Fr
	5Fr

1. Supine position. Pay careful attention to thermoregulation. Sterile precautions.
2. Attach 3-way taps and flush each catheter with saline.
3. Grasp the cord clamp with a pair of artery forceps, hand to your assistant, prep and drape the umbilicus and surrounding skin while the umbilicus is held taut. Place and tie a sterile tie around the base of the cord.
4. Smoothly slice the cord (as you do this your assistant removes the clamp).
5. Identify vessels: the single UV sits at the 12 o'clock position, and has floppy walls. The two UAs have more muscular walls, and pout out of the cord's jelly.
6. Grasp the side of the cord with artery forceps to stabilise while you cannulate.
7. Dilate the umbilical artery with iris forceps: insert both tines of the forceps into the artery, separate slightly as you withdraw.
8. Insert the UAC. Distance from skin = (weight in kg x 3) + 9cm. Check that it aspirates, flush slowly.
9. Insert the UVC. Distance from skin = (weight in kg x 1.5) + 6cm. Check that each lumen aspirates and flushes.
10. Suture the UVC, then the UAC. Use 'goalposts' to anchor them to skin.
11. Xray: the UAC should sit between T6 and T9. The UVC should sit at T9-T10.

Tips:

- Insert the UVC second and suture it first. The UAC is unlikely to move once positioned, while the UVC moves easily.
- Tighten the umbilical tie if the stump is oozing; loosen if the catheters bounce at several centimetres. Remove it at the end of the procedure.
- A UVC which has fed to an intrahepatic position can be pulled back to a 'low' position, and used as short term (NOT CENTRAL) venous access.
- Patience is important: using force when attempting to insert umbilical lines can create a false passage.

Arterial. Tip above diaphragm, below the left subclavian artery; depth = 2cm more than distance from the tip of the shoulder to the umbilicus.

PERIPHERAL INTRAVENOUS LINES

No more than 2 peripheral veins should be used before escalating to a more experienced doctor.

Never run a calcium infusion through a peripheral IV line (even for a short time).

If TPN is run through a peripheral line, the risk of extravasation needs to be considered. NO child should ever go to theatre with TPN running through a peripheral line. If a child is on TPN prior to surgery, change to glucose infusion and ensure the equivalent mg/kg/min glucose infusion during surgery.

If extravasation occurs, tell a doctor immediately. Call plastic surgeons to review urgently, and follow the extravasation guideline:

http://www.rch.org.au/rchcpq/hospital_clinical_guideline_index/Extravasation_Injury_Management/

This includes hyaluronidase irrigation within 1 hour for extravasation of parenteral nutrition and calcium chloride. Hyaluronidase is NOT used for vasoconstrictor extravasation.

CHEST DRAIN

In children, chest drains should be inserted in the mid-axillary line (not the mid-clavicular line), in the 4th or 5th intercostal space.

To drain a pneumothorax in a supine infant, the tip of the drain should be positioned anteriorly near the xiphisternum.

Use a large drain for blood or pus (eg 16 gauge for a 5-10 kg child), and a smaller drain (preferably a 6F or 8F pigtail catheter) for pneumothorax or uncomplicated effusions (see Pneumonia – Empyema).

Lay the child supine with the arm above the head. Roll the child slightly away from you for a pneumothorax or slightly towards you for a collection of fluid.

Infiltrate the skin and intercostal space with 1% lignocaine.

For pigtail catheters, the technique is Seldinger: needle puncture through a small skin incision, then dilatation.

For non-pigtail drains: make a small incision (just bigger than the chest drain) in the skin in the 4th or 5th intercostal space in the mid-axillary line.

Use only blunt dissection of the intercostal space with a pair of artery forceps over the top of the rib (the neurovascular bundle lies just below each rib).

Hold the artery forceps about 2 cm from the tip (so that they cannot go in too far), angle them towards the xiphisternum, then push the forceps smartly through the pleura (you will feel a pop).

Remove the forceps, and use them to hold the chest drain and push it into the pleural space angled towards the xiphisternum.

Make sure that all the exit holes are inside the pleural space.

To keep the tip of the tube anterior, tape the outside part of the tube to the skin behind the shoulder (so the tube runs under the arm).

Seal the tube with a 3-0 silk purse-string stitch, and tie the silk firmly to the tube. Put an adhesive clear dressing over the entry site (with some gauze if there is ooze from the wound).

Take AP and lateral chest X-rays to check the position of the tube. Make sure that all the side holes are inside the pleural space.

Good analgesia can often be achieved after chest trauma or surgery by infusion of 0.5% bupivacaine into the pleural space via a chest drain: 0.5 ml/kg (max 20ml) 8-12H, or 0.5 ml/kg stat and then 0.1-0.25 ml/kg/hr (max 10 ml/hr).

COMA

Coma is a symptom, not a diagnosis. Consider trauma (which may be non-accidental), meningitis, encephalitis, cerebral tumor, vascular accident, poisoning, metabolic disease, DKA, severe sodium derangement, non-convulsive status epilepticus, tuberculoma and hypertensive encephalopathy. See specific sections on these conditions.

Maintain airway, breathing and circulation. Check blood pressure.

Intubate early, especially in young children, and in children who have signs of acute raised intracranial pressure.

Blood for glucose, culture, gas, electrolytes, creatinine, liver function tests, ammonia and cortisol. Urine for ward test (ketones), drug screen, and metabolic screen (and gas-liquid chromatography, if acidotic).

Do not do a lumbar puncture. Lumbar puncture can be done later when the child is conscious. Give cefotaxime and acyclovir.

Do a CT brain (to exclude haemorrhage or major stroke) or ideally MRI brain (if more likely encephalitis, ADEM, metabolic brain injury, early ischaemia, posterior reversible encephalopathy syndrome secondary to hypertension).

Consider a trial of naloxone 0.1 mg/kg (max 2 mg) IV. If responds, give 0.01 mg/kg/hr (0.2 mcg/kg/min) IV.

See sections on meningitis and encephalitis for further investigations.

Consider doing an EEG.

CONVULSIONS – STATUS EPILEPTICUS

Most convulsions stop spontaneously. ICU admission will usually be required for patients who have not responded to midazolam (0.2mg/kg x 2), or diazepam (0.2mg x 2) followed by a loading dose of a second-line drug: levetiracetam (50mg/kg), phenobarbitone (20-30 mg/kg IV over 30-60 min) or phenytoin (15-20 mg/kg IV over 60 min).

It is important to stop seizures quickly in patients with acute brain injury (eg trauma or meningitis or acutely raised ICP). Maintain airway, oxygenation and blood pressure at all times.

1. Give oxygen. Time seizures from its onset, monitor vital signs. Give benzodiazepine x 2 within first 5-30 minutes, then if still seizing give loading of second-line drug by 30-60 minutes.
2. Measure blood glucose. Consider blood gas, electrolytes, creatinine, liver function tests, full blood examination, drug screen, metabolic screen, ward test urine (ketones), blood culture, ammonia, CK. Do not do a lumbar puncture.
3. Respiratory depression caused by anticonvulsant therapy in an unintubated child is at least as dangerous as the convulsion. Children who have convulsions despite the above treatment usually need to be intubated and ventilated. Pre-oxygenate, cricoid pressure, thiopentone 4 mg/kg (as long as not hypotensive), vecuronium 0.1 mg/kg IV, wait 2 minutes then intubate quickly.
4. If no IV access, use midazolam 0.2 mg/kg IM or 0.5 mg/kg buccal, repeated as required.
5. Ensure adequate dose of one or 2 second-line drugs, then if still seizing give midazolam infusion 1 mcg/kg/min increasing if required (usually 2-3 mcg/kg/min, range 1-18 mcg/kg/min).
6. Give pyridoxine 100 mg IV to a child <18 mo with recurrent or refractory seizures. Pyridoxine-dependent convulsions should respond in 10-60 minutes.
7. Give cefotaxime and acyclovir (do not do a lumbar puncture), see encephalitis. CT or MRI brain when the child is stable.
8. Maintain core temperature 36-37°C.
9. Continuous EEG monitoring.
10. Thiopentone may be required if convulsions persist despite adequate loading of one or 2 second-line drugs (levetiracetam, phenobarb or phenytoin) plus high dose midazolam infusion. Give thiopentone 1-2 mg/kg slow boluses until control achieved, and then 1-5 mg/kg/hr. Monitor level of thiopentone needed with EEG (aim for burst

suppression - a level of anaesthesia at which all seizure activity is usually controlled) and monitor clinically.

11. Hypotension is a dangerous side effect of thio: maintain CVP 8-12 cm, and echo early to check myocardial contractility (dobutamine and noradrenaline may be required).
12. If status continues despite thiopentone infusion (or recurs on withdrawal of thio), consider magnesium sulphate infusion (safe), ketogenic diet, hypothermia, and immunotherapy (steroids, IViG, plasma exchange in specific cases likely to have immune-mediated refractory seizures: FIRES (febrile infection-related epilepsy syndrome)).

CROUP

High-pitched inspiratory stridor in a child with a hoarse voice and a harsh barking cough – usually with an upper respiratory tract infection. The differential diagnosis includes epiglottitis, foreign body, retropharyngeal or peritonsillar abscess, infantile larynx, haemangioma, subglottic stenosis.

In mild disease, the stridor is present only when the child is agitated. In more severe disease, there is stridor at rest – but the loudness of the stridor is a poor guide to severity. When airway obstruction is severe, there is marked inspiratory indrawing of suprasternal, intercostal and subcostal soft tissue. In very severe disease, there is cyanosis and reduced air entry, with stridor that may be expiratory as well as inspiratory, and may be soft despite severe obstruction. Cyanosis or apnoea require intubation.

1. Give dexamethasone 0.6 mg/kg IM or IV, then prednisolone 1 mg/kg 8H IV, oral or NG.
2. Nurse the child fully clothed in a warm room (to avoid cold stress), and disturb as little as possible.
3. Give 1% adrenaline (L isomer) 0.05 ml/kg/dose diluted to 4 ml by inhalation, or 1/1000 0.5 ml/kg/dose (max 6 ml) by inhalation as required. Outside ICU, a doctor must be in attendance whenever adrenaline is given (not because it is dangerous, but because the need for it implies severe airway obstruction).
4. Any child given adrenaline must be reviewed by ICU and considered for admission by ICU. If a child is considered to need another dose of adrenaline within 3 hours of a previous dose, the child should be reviewed by ICU before the dose is given, and, if the dose is required, the child must be admitted to ICU. If ICU cannot review the child immediately, the adrenaline should be given and the child should be admitted to ICU.
5. In ICU, adrenaline can be given repeatedly every hour or two if required to give the dexamethasone time to take effect.
6. Do not give oxygen unless a decision has been made to intubate (as oxygen may mask the signs of impending complete obstruction). Do not give steam or mist therapy.
7. The child should be intubated if he or she has severe obstruction, or has a poor response to inhaled adrenaline. Do not wait until the child is exhausted or very severely obstructed. It is often helpful to sit the child on the mother's lap, and get her to hold the face-mask near the child's face with 4 L/min of 100% oxygen. Gradually introduce

sevoflurane, increasing to 6% over 1-2 minutes. When the child goes to sleep, lie him or her down, place the mask firmly over the face (held by the middle, not the edge), pull the jaw forwards, and apply gentle positive pressure during inspiration if required (beware of inflating the stomach). It will take 5-10 minutes of 6% sevoflurane to achieve adequate anaesthesia in a child with severe croup. If possible, monitor end-tidal CO₂ and sevoflurane (aim for 4.5%). Apnoea and athetoid or epileptiform movements may occur. Vasodilation may cause hypotension if the child is even mildly dehydrated – be prepared to insert an IV and give 10-20 ml/kg of 0.9% saline quickly. If inhalational agents are not available, consider using ketamine 5-10 mg/kg and atropine 0.02 mg/kg IM.

8. Intubate the child with an oral endotracheal tube 0.5-1.0 mm smaller than the usual diameter: for example, use a 3.0 mm or 3.5 mm tube in a child aged 12-23 months. Most children will have copious yellow secretions when first intubated – this does not mean they have bacterial tracheitis. Instil 0.9% saline if necessary to enable all the secretions to be removed. Reduce the sevoflurane to 4.5%, and be careful to allow the child to breathe spontaneously (hand-ventilate briefly after suction).
9. When the child's trachea has been well sucked-out and there is adequate depth of anaesthesia, remove the oral tube and replace it with a nasal uncuffed endotracheal tube.
10. Insert an IV drip and a nasogastric tube. Firmly splint both arms with splints that reach from the axilla to the wrist – this must be done carefully. Allow the child to wake up, and do not give any sedation unless this is needed for transport.
11. Put a condenser-humidifier on the tube. Careful and frequent suction (often after putting 0.5-1 ml saline down the tube) is very important to prevent the copious thick secretions blocking the tube.
12. Children should be extubated when there is a leak around the tube, or at 24-36 hours (>2 yr) or 36-48 hours (<2 yr). Children with influenza or herpes simplex croup will need to be intubated for longer.
13. Do not give antibiotics unless there is bacterial tracheitis, which is very uncommon (many organisms of a single type seen on gram stain, with a heavy pure culture). Do not routinely culture tracheal aspirate for bacteria.
14. 95% of croup will resolve by 5 days. If a child has upper airway obstruction beyond 5 days, or if not typical croup (URTI symptoms), or cyanosis at any stage, do a chest xray and review for alternative diagnosis (subglottic stenosis or web, mediastinal mass, vascular sling).

DEATH OF A CHILD

From within RCH, detailed information and all the necessary forms can be found under "Death of a Child" in the Clinical Practice Guidelines on the intranet at:

www.rch.org.au/clinicalguide/cpg.cfm?doc_id=5305

All cases

Notify ICU Consultant, Bedcard Unit (who should arrange followup), referring doctor, GP. Complete ICU discharge procedures. If organ donation does not occur, record in the notes (1) why organ donation was not requested, or (2) that it was requested but that permission was refused. Ask the family if they will consent to a post-mortem.

No report to Coroner

Complete a Medical Certificate of Cause of Death, RCH autopsy form and consent form. Do not call the Medical Certificate of Cause of Death a "death certificate" – the death certificate is a document issued by the Registrar of Births, Deaths and Marriages.

Report to Coroner

Report the death to the Coroner if it is unexpected, unnatural, violent or an accident; occurred during anaesthetic (even from natural causes); as a result of an anaesthetic or operative procedure; a person held in care (eg. care order with DHS); a person of unknown identity, or the second child death in a family (reviewable death). It is not always necessary to report a death to the Coroner just because it occurred within 24 hours of an anaesthetic (unless it occurred during or as a result of the anaesthetic or procedure).

<http://www.coronerscourt.vic.gov.au/resources/1e321087-60c8-4c56-beb3-63d29d263c81/flowchart+to+determine+whether+a+death+is+reportable+or+reviewable.pdf>

If in doubt about whether to report a death, ask the ICU consultant (who may discuss the case with the Coroner, or discuss with a senior colleague).

Ring 96844444 to report the death.

Record the Coroner's case number in the UR.

Fill out a Coroner's deposition (summary of the case), with a copy for the Coroner, a copy for the notes, and a copy for you to keep.

Ask family to complete a Statement of Body Identification.

Lodge a request for no autopsy if family wants this.

A Medical Certificate of Cause of Death is not required.

Inhibition of lactation

If the mother requires inhibition of lactation, prescribe cabergoline (Dostinex) 1mg oral once.

DEATH - ORGAN DONATION

Organ donation can occur after brain death (DBD), or following circulatory death (DCD) and should be considered in all children having withdrawal of intensive care.

For the complete guideline for Donation after Circulatory Death visit:

http://www.rch.org.au/picu/Donation_after_cardiac_death_protocol/

1. Diagnosis of brain death. The diagnosis of brain death requires unresponsive coma, absent brainstem reflexes and absent respiratory function. (See ANZICS Statement on Death and Organ Donation Edition 3.2) The necessary preconditions for clinical suspicion of brain death must be present:

- The child has a condition known to cause brain death
- Normal blood pressure (use age appropriate SBP)
- Body temperature >35°C and normoglycaemia
- No significant electrolyte or endocrine abnormalities
- Neuromuscular transmission is normal by train-of-four stimulus testing
- Significant drug intoxication (including morphine, benzodiazepines and barbiturates) has been excluded. (flumazenil and naloxone can be used to reverse benzodiazepine and opiate activity)

The following are clinical tests of brainstem function (In Victoria - must be performed by 2 doctors of 5 years experience):

- Test both eyes for corneal reflex + pupil response to light
- Vestibulo-ocular (caloric) test on at least one side (observe for at least 60 seconds)
- Do not perform an oculo-cephalic (doll's eye) test
- Gag and cough reflexes
- Test response to painful stimuli to limbs and supraorbital region (V cranial nerve distribution)
- Note: **we no longer test for respiratory movement** in response to hypercarbia and acidosis as this may lead to increased intracranial pressure. **Instead a test of cerebral blood flow, usually a radionuclide scan using Ceretec is done.**

Radionuclide scanning or 4-vessel angiogram is used to demonstrate absence of blood flow above the carotid siphon.

The time of death is recorded as the time when the second medical officer has confirmed the absence of cerebral blood flow

(ie reviewed the reported complete Ceretec scan)

For children over 30 days of age: determination of brain death is the same as adults

For neonates ≥ 36 weeks-29 days: determination of brain death is to be performed after 48 hours from birth. Second clinical exam should occur 24 hours after the first.

All aspects of DBD can be performed by the treating intensivist.

Management of haemodynamic instability secondary to brain death

Brain death can lead to a neuro-humoral response leading to profound circulatory instability which can last hours. Diabetes Insipidus, loss of sympathetic tone, hypothermia and lost pituitary function contribute to this.

Diabetes Insipidus – Vasopressin 2-5 units in 1litre of fluid: replace urine output + 10% per hour OR 1 unit/kg in 50ml at 1-3ml/hr (0.02-0.06 units/kg/hr)

Circulatory dysfunction – inotrope and vasopressor (minimum necessary) as required. Methylprednisolone 15mg/kg.

Triiodothyronine (T3) for myocardial dysfunction: based on echocardiographic findings and in consultation with the cardiac intensivist or cardiologist.

Maintain normoglycaemia with infusion of 50% dextrose, or an infusion of insulin (0.05-0.1 unit/kg/hr) as needed.

Treat coagulopathy with FFP.

Give platelets if bleeding due to platelet count $< 50 \times 10^9$

Stable gas exchange (PaCO₂ + PaO₂ as close to normal as possible); prevent atelectasis (PEEP 5-10 cm + ETT suction); use low tidal volumes to avoid volutrauma.

2. Donation following Circulatory Death (DCD)

Main considerations

DCD is to be considered for all patients where withdrawal of life sustaining therapies is going to occur.

The intensivist, the treating unit consultant and the family must agree on withdrawal of life sustaining therapies first before consideration of organ donation. All units involved must document this in the medical record.

Designated intensivist – a second intensivist provides the donation counselling and seeks consent for DCD.

Designated officer (representing the hospital) – a number of senior medical staff are registered as designated officers and their consent on behalf of the hospital is required to permit DCD to occur. Usually a member of the RCH executive.

Medical suitability – is determined by transplantation teams so referral to the organ procurement agency (Donatelife) is indicated even to discuss potential suitability. DCD is not possible if the child is likely to breathe for 90 minutes following withdrawal of life sustaining therapy.

See PICU intranet for complete protocol.

3. Coroner. In a Coroner's case, the ICU consultant should discuss organ donation with the duty coroner via the on-call pathologist at the Coroner's office. ICU medical staff should complete a Coroner's Deposition (found at <https://secure.vifm.org/meddep/>) and a Statement of Body Identification. Police will attend as agents of the Coroner, and they may or may not wish to speak to parents in the ICU. Warn parents of this possibility, and that this is routine practice.

4. Serology and other tests. Most ante-mortem tests are conducted with the instruction of the donor co-ordinator from Donatelife if the parents have agreed to organ donation: 10 ml clotted blood (gel tube) should be sent via RCH Core Laboratory for urgent hepatitis B, C & D; HIV 1 & 2 and CMV serology; LFTs, lipase, urea, creatinine and electrolytes (unless recent results available); CXR; ECG; cardiac echo for contractility, valve function, vegetation and anatomical abnormality. Bronchoscopy may be indicated.

5. Donor coordinator (Donatelife) notification. After brain death has been certified or DCD has been consented for, RCH staff (usually the ICU consultant) will conduct the initial discussion with the parents about the meaning and significance of brain death and about organ donation. If parental consent is obtained, the donor coordinator will be contacted by the ICU consultant and will meet the parents. The **24 hour contact phone number** for the duty Donatelife donor coordinator is **9347 0408**.

The donor coordinator needs the child's age, weight, diagnosis and previous medical history, blood group, all current medications, LFTs, urea and creatinine, hepatitis B, C and D, HIV 1 and 2, and CMV status.

The donor coordinator will: meet the parents and discuss the process of organ donation; notify the recipient coordinators; coordinate the activity of the organ removal teams; book theatres for organ removal; notify the anaesthetist for the donor; notify ICU of the likely theatre time.

Provide the Donateliflife donor co-ordinator a space with computer access, supervised access to the child's EMR and introduce them to the nurse in charge. They may not be familiar with the PICU or staff.

5. Requirements of recipient transplant teams. These should be discussed by the donor coordinator with the individual recipient teams and then with the RCH ICU consultant. Important considerations (apart from ABO group, serology and exclusion criteria such as disseminated malignancy, active auto-immune disease and presence of some inborn metabolic errors such as mucopolysaccharidoses or primary lactic acidoses) include:

Kidney: age, hypotension, high-dose inotropes, serum urea, creatinine, electrolytes.

Liver: age, weight, hypotension, inotrope dose, use of vasoconstrictors including vasopressin, electrolytes, LFTs, clotting profile, glucose.

Pancreas: age, Low BP, high dose inotropes, blood glucose and serum amylase, abdominal trauma.

Heart: age, weight, hypotension, use of high dose inotropes, cardiac echo examination including assessment of contractility, ECG, CXR.

Lungs: age, size, weight, hypotension, high dose inotropes, cardiac echo, bronchoscopy, ECG, CXR, and blood gases after 15 minutes on 100% O₂ and 5 cm PEEP.

Corneas: age, eye injury or ulceration.

Age should not be considered a contraindication to donation. Case by case consideration will be applied to potential infant and neonatal donors.

6. Booking of theatre time. The donor coordinator will contact theatre nursing staff and the anaesthetist in charge, in order to book the case. In-hours, this can be done by ringing the duty anaesthetist (ext 52000). The organ procurement procedure needs to be booked on the electronic medical record.

7. Planning for transfer of the child from intensive care unit to theatre. For donor patients who are brain dead, intensive care should be notified of the anticipated theatre time by the donor coordinator as soon as possible. For DCD donors a pre-withdrawal planning meeting will occur with all relevant operating theatre staff, anaesthetists, surgeons, and the designated intensivist.

8. Parents and visitors. Well in advance the ICU consultant will discuss with parents and visitors the procedure for taking the child to theatre. Parents and visitors will say their farewells in the patient's ICU room and will then leave the ICU. They should not accompany the child to the theatre.

9. Intra-operative management of brain dead donors. In

general the medical management commenced in ICU should be continued. Muscle relaxants should be given to prevent spinal reflex movements. The order of organ procurement will be decided by surgeons. Drugs that are usually required intraoperatively include heparin, methylprednisolone and antibiotics. The exact dosage and timing of dosage should be discussed with the surgeons involved and/or the donor coordinator. Femoral lines will not be useful if and when the IVC is cross-clamped, so intravenous access in the upper body must be available.

10. Postoperative management. The duty social worker will arrange for the parents and friends to view the child's body after the procedure if they wish to. At the end of the procedure, the donor is taken to the mortuary to prepare for transfer to the funeral director or Coroner's office. For Coroner's cases the office should be informed when the patient arrives at the mortuary.

11. Pastoral care – participation in organ donation is stressful and can be distressing. PICU can facilitate debriefs soon after the event. Staff are encouraged to seek help, internally or externally, for persistent or unresolved distress.

12. Hospital Donation Specialists – Any concerns regarding organ donation at RCH can be relayed to the PICU medical donation specialist or nursing donation specialist

DEATH – TISSUE DONATION

Tissue donation can occur within 24 hours of death. This is feasible for patients who die in PICU or other locations in the hospital. Enquiries in relation to tissue donation can be made at the Donor Tissue Bank of Victoria (DTBV; Victorian Institute of Forensic Medicine) ph 9684 4444 in hours or 0407326705 after hours.

Standard tissue donation and age criteria

Cardiovascular tissue –

Standard donation: aortic valve, pulmonary valve and pericardium

Age: 3 months (no weight criteria as long as babies were born at full term with no growth restrictions in utero).

Skin

Standard donation: superficial layer of skin from upper back, flanks, posterior upper/lower legs and anterior upper legs

Age: no specific age limit but depends on size. Very small body surface areas and bony prominences may be excluded.

Bone

Standard donation: femur, tibia, iliac crest and humerus

Age: From 18 years

Tendon

Standard donation: Achilles, tibialis (anterior) and patella

Age: From 18 years

Eyes:

Corneas- will consider 2-6 years. Confirm with Lions Eye Donation Service (LEDS)

Sclera and eye research- May accept any age. Confirm with LEDS

Procedures to be followed

1. When heart valve donation is proposed, the transplant coordinator should be notified as soon as possible. Note that for tissue donation *an autopsy will be required*, and the DTBV Medical and Social History Questionnaire (F000) must be completed.
2. Potential donors should meet all the criteria listed in Part B, Medical Acceptance for Heart Valve Donors. The suitability of the donor will be discussed with the treating doctor by the donor coordinator at the time of referral.

3. When the suitability of the donor has been established, the transplant coordinator will coordinate with the cardiac surgeon on-call and arrange transport details with ward and anatomical pathology.
4. The transplant coordinator will be responsible for discussing the option of donation with the family and completing the necessary forms for consent or denial.
5. A copy of the consent will be provided for inclusion in the donor medical history.
6. When the donor is a Coroner's case, the transplant coordinator will be responsible for dealing with the family. However, in such cases, the autopsy if ordered by the coroner will be performed at the Victorian Institute of Forensic Medicine.
7. Blood for serology will be collected at autopsy and forwarded to DTBV for testing by a TGA licensed lab.
8. The parents should be informed that the child will be tested for HIV, and that a brain biopsy will be needed for CJD (although this may change).

PART B. MEDICAL ACCEPTANCE FOR HEART VALVE DONATION

The following criteria must be met:

Death certified and recorded in the patient's history.

The patient does not have active systemic infection.

There is no evidence of active infection involving the tissues to be retrieved.

There is no evidence of present or past slow virus disease.

There is no clinical history of active tuberculosis.

There is no clinical history of syphilis.

There is no evidence of malignancy.

There is no history of active hepatitis or unexplained jaundice.

There is no diffuse connective tissue disorder, metabolic bone disease, or other serious disorder or disease of unknown aetiology, including rheumatoid arthritis.

The cause of death is known.

There is no history of non-medical parenteral drug use in the patient, or in the parents if the patient is being breast-fed.

There is no history of irradiation to the mediastinum.

There is no history of ingestion or administration of toxic substances that might be transferred in heart valves or arteries.

There is no history of dementia or neurologic degenerative disease.

There is no history of living in the UK for a cumulative period of more than 6 months between 1980 and 1996.

There is no clinical evidence of HIV infection.

The patient has not received pituitary growth hormone

The patient has not received human derived clotting factor concentrates for treatment of haemophilia.

The patient has not been identified as being at high risk for HIV infection.

If the patient is being breast fed, the parents have not been identified as being at high risk for HIV infection.

DIABETIC KETOACIDOSIS

Children with diabetic ketoacidosis should be admitted to ICU if they are less than 2yr old, or if there are signs of cerebral oedema. The major risks are cerebral oedema, rapid changes in serum osmolality (usually caused by overzealous rehydration and/or excessive insulin), hypoglycaemia, worsening acidosis (due to hypovolaemia or lack of insulin), and severe hyperkalaemia or hypokalaemia.

Insulin 2.5u/kg in 50ml 4% albumin. Start at 1ml/hr (0.05 u/kg/hr), increase to 0.1u/kg/hour if blood sugar not falling.

When the blood glucose is less than 12 mmol/L:

- give more intravenous dextrose
- do NOT stop insulin or reduce <0.05 u/kg/hr.

While on an insulin infusion:

- do glucose hrly, gas + elec 2-4H, blood or urinary ketones 6H
- do hourly neuro obs (bradycardia a major sign)
- calculate effective plasma osmolality 2-4 hrly = $2[\text{Na}^+] + [\text{glucose}]$.

Do not give insulin routinely for hyperosmolar nonketotic diabetes (ketonuria no more than one plus).

1. Assess clinical condition. If only very mildly dehydrated and not vomiting or acidotic, proceed to protocol for stabilisation rather than emergency treatment.
2. Give nil by mouth (except ice to suck). Insert a nasogastric tube if the patient is comatose or has repeated vomiting, and leave on free drainage.
3. Take blood for glucose, electrolytes, plasma osmolality, acid-base studies. In newly diagnosed patients, do islet cell antibodies, insulin antibodies, GAD antibodies, total IgA, anti-endomysial IgA antibody, and thyroid function.
4. Check urine for ketones and presence of infection.
5. Commence resuscitation without waiting for results.
6. Nurse head-up 15° (in coma position if unconscious).
7. Check for precipitating cause, eg infections (check injection sites, urine, consider blood culture and CXR).
8. Use diabetic ketoacidosis chart to improve monitoring.
9. Blood transfusion is almost never necessary.

Resuscitation and rehydration

1. Children with DKA who are hypotensive should be given 10 ml/kg boluses 0.9% saline until blood pressure is normal. A guide to minimum acceptable systolic BP is:

Age	6m	1y	2y	4y	6y	8y	10y	12y	14y	16y	18y
SBP	65	70	75	75	75	80	80	85	90	95	95

2. Because of the risk of DVT, do not put in a central line unless it is absolutely necessary. If a CVC is used, give heparin 10 u/kg/hr through the line.
3. After restoration of blood pressure, all children with DKA and unequivocal signs of dehydration should be given the following amount of IV fluid (based on maintenance requirements of 80% of normal, and 3-5% dehydration corrected over 48 hours) irrespective of their apparent degree of dehydration:

Wt (kg)	5	6	7	8	9	10	12	14	16	18	20	25	30	35	40	45
ml/hr	24	28	32	36	40	45	50	55	65	70	75	80	90	95	105	110

This fluid rate (ml/hr) includes deficit AND maintenance fluid needs. The degree of dehydration is often overestimated in children. Fluid therapy should be reviewed if oliguria develops: tubular necrosis caused by severe hypotension before resuscitation may require fluid restriction, but persistent hypovolaemia may require extra fluid.

4. Type of fluid

- a. 0.9% NaCl is to be used for the first 24 hours, then Plasma-lyte 148 in 5% dextrose. At the time of starting the insulin infusion, add KCl to the saline at 40 mmol/L if body weight <30kg, and at 60 mmol/L if >30kg.
- b. When the blood glucose falls below 12 mmol/L, either add dextrose to the maintenance fluid (i.e. make it 5% or change 0.9% NaCl to Plasma-lyte 148 with 5% glucose), or (only if there is a central line) give 50% dextrose IV at 0.5 ml/kg/hr (maximum 10 ml/hr). Adjust the dose of dextrose to keep the blood glucose at 8-12 mmol/L. If the blood glucose falls below 8 mmol/L, increase the dextrose infusion rate - DO NOT STOP THE INSULIN INFUSION.

Insulin Infusion

- a. Start the insulin infusion together with the KCl replacement after the initial resuscitation has been completed and the patient is in ICU.
- b. Add 2.5 unit/kg of clear or rapid-acting insulin (Actrapid HM or Humulin R) to 50 ml 0.9% NaCl, and run at 1 ml/hr (0.05 u/kg/hr of insulin). This dose provides near maximal glucose clearance; lower doses may be needed to prevent a rapid fall in blood glucose. The insulin infusion may be run as a sideline with the rehydrating fluid via a three-way tap providing a

volumetric or syringe pump is used. Ensure that the insulin infusion is clearly labelled. Insulin is infused in 4% albumin to reduce adsorption to the tubing.

- c. Adjust the dextrose infusion rate to keep the glucose level at 8-12 mmol/L. Remember that insulin is needed to clear the acidosis (ketonaemia) and adequate insulin must be continued IV or SC until the ketones have cleared. The insulin infusion rate can be increased if the acidosis does not improve by at least 0.03 pH unit/hour, increase the % dextrose infused if necessary.
- d. The best time to change to SC insulin is just before a meal, when the child is alert and metabolically stable (glucose <8-12 mmol/L, pH >7.30 and HCO₃ >15). The insulin infusion should only be stopped 30 minutes after the first SC injection of insulin.

Potassium Replacement

Potassium replacement should begin at the same time as the insulin infusion is commenced. Start KCl at a concentration of 40-60 mmol/L (40 mmol/L if body wt <30 kg, and 60 mmol/L if 30 kg or above). If K levels are tending low, check with venous or arterial K (rather than capillary). Extreme care should be taken if the initial serum K is >5.5 mmol/L or if the patient is anuric.

Bicarbonate

Do not give bicarbonate. Administration of bicarbonate may increase the risk of cerebral oedema. A low pH is not in itself harmful if the child has a good cardiac output and good peripheral perfusion. Worsening acidosis usually means hypovolaemia, insufficient insulin, hyperchloraemia from 0.9% NaCl, or tissue necrosis from ischaemic injury before adequate resuscitation.

CLOSE CLINICAL AND BIOCHEMICAL MONITORING

Clinical

- a. Document fluid balance carefully. All urine should be measured, and tested for ketones at least 6 hourly. All fluid input should be recorded (including oral).
- b. Record observations hourly for 24 hr: pulse, blood pressure, respiratory rate, level of consciousness and pupils. See cerebral oedema, below.
- c. Record temperature 4 hourly.

Biochemical

These tests may have to be performed more frequently. Use the DKA flow chart. While on an insulin infusion do:

- a. Hourly blood glucose levels, and 6 hourly urine ketones

- b. 2-4 hrly blood gases, sodium, potassium, chloride and urea. Calculate the effective plasma osmolality 2-4 hrly = $2[\text{Na}^+] + [\text{glucose}]$. If capillary bloods are done, beware of falsely high K^+ levels. Aim to prevent the effective plasma osmolality falling faster than 0.5 mmol/L per hour; the plasma Na^+ will have to rise to compensate for the fall in glucose (and K^+). It may be necessary to give 3% saline (4 ml/kg over 6 hr raises sodium by 3 mmol/L). Do not use the "corrected sodium" to monitor therapy.

HAZARDS OF THERAPY

1. HYPOGLYCAEMIA. Treat hypoglycaemia with 50% dextrose 1 ml/kg infused over 3 minutes, and by increasing the amount of dextrose infused. Do not discontinue the insulin infusion or reduce it to less than 0.05 u/kg/hour. If venous access cannot be obtained, give oral or NG 50% dextrose 1 ml/kg, glucagon 1 mg IM and notify the ICU consultant. Prevention includes adding dextrose to the IV fluids when the blood glucose has fallen to 12-15 mmol/L

2. CEREBRAL OEDEMA. Subclinical brain swelling is present during most episodes of diabetic ketoacidosis. Clinical signs of cerebral oedema occur suddenly, usually 6-12 hr after starting therapy (range 2-24hr). Mortality or severe morbidity is very likely without early treatment.

Risk factors: first presentation, long history of poor control, age <5 yr; failure of sodium to rise as glucose levels decline, development of hyponatraemia, initial low osmolality; HEAD-ACHE, behavioural changes such as irritability, lethargy and depressed level of consciousness. BRADYCARDIA, INCREASED BP, AND RESPIRATORY IMPAIRMENT are late signs of raised intracranial pressure.

Prevention: The most important factor is SLOW correction of the fluid and biochemical abnormalities – osmolality ($2\text{Na} + \text{glucose}$) should not fall faster than 0.5 mmol/L per hour. Nurse 15° head up.

Treatment:

- a. Mannitol 20% should always be readily available; the dose is 2.5 ml/kg (0.5g/kg) IV stat. This can be repeated every 15-20 minutes. It should be given as soon as the clinical diagnosis is made - it should NOT be delayed for confirmatory brain scans.
- b. Fluid input should be severely reduced and changed to 0.9%

- NaCl with added dextrose.
c. Transfer immediately to ICU.

HYPEROSMOLAR NONKETOTIC DIABETES

Some children have very high blood glucose levels without marked ketosis (ketonuria no more than one plus); the pH is usually > 7.10; they are often obese, and may have axillary freckling. There may be severe dehydration. These children are at very high risk of cerebral oedema. Start with boluses of 0.9% saline 10ml/kg IV to correct hypovolaemia, and then slower as for ketoacidosis (see Resuscitation and Rehydration, above). The effective osmolality (2Na + glucose) must not fall faster than 0.5 mmol/L per hour. Insulin is NOT needed initially, and is dangerous if it causes an abrupt fall in effective osmolality. Rhabdomyolysis, renal failure and gut ischaemia may occur. If insulin is required for severe hyperkalaemia (consider haemofiltration rather than insulin) or for moderate ketosis that does not resolve with rehydration (mixed DKA and hyperosmolar syndrome), start with only 0.01 u/kg/hr (0.5 u/kg in 50ml 0.9% NaCl at 1ml/hr).

DISASTER - CODE BROWN

See the Emergency Displan Folder in each room in ICU.

The ED Consultant is likely to be the first person aware of the Emergency. They communicate to the Director of Operations, or Nursing Hospital Manager, who in-turn communicates with the Hospital Commander who determines if a Code Brown, or Code Brown Standby is announced. The ED Consultant will activate a trauma page as required, and will liaise directly with the PICU General consultant regarding the event ASAP.

PICU Notification

PICU Consultant will notify:

- AUM
- Clinical Technologist
- Other Consultant medical staff as indicated below and ICU registrars

AUM will notify:

- NUM
- Nursing staff on duty
- Ward Clerk
- PSAs

PICU areas of responsibility

PICU general unit / flexipod

PICU cardiac unit

PICU will set up and staff an annex in operating theatre recovery area to decant less unwell patients. Some children can be moved to the wards, or to this PICU annex staffed by PICU nurses and an ICU registrar. This will free up PICU beds for Code Brown admissions. The PICU General consultant will oversee the transfer of children to this area, and their subsequent care.

If needed PICU will also staff 2 beds in the Medical Imaging Recovery bay, requiring 3 nurses and a senior registrar. The intention is to hold no more than 2 patients in this area while waiting for CT, but with full ventilation, inotrope, resuscitation facilities as needed. If a child is likely to need to wait more than 2 hours before CT or OT, they will be transferred to PICU and returned when ready.

If necessary (eg at the request of State Displan Coordinator) PICU will provide a team to go to the disaster site (ICU registrar + a nurse from ICU or Emergency Department or recovery) for triage and site resuscitation. Will use PETS equipment, plus site team equipment as needed.

ROLE DESCRIPTIONS FOR KEY ICU PERSONNEL

ICU Associate Unit Manager (AUM). Allocate tasks to nurses in PICU; give out job instruction cards from Displan Folder; call in extra nurses in consultation with Nurse Coordinator of Disaster; supervise transfer of moveable current patients to NNU or wards; move whiteboard to central area; collect tabbards and magnets; fill-in details of current ICU population and nursing staff on white board; supervise documentation of new arrivals; contact Services to get extra beds, cots and equipment; direct PSAs to clean and assemble used or old ventilator circuits and other equipment; with ICU technologist and delegated nurses monitor adequacy of supplies – sterile packs, dressings, fluids, disposables, drugs, linen, hardware (eg beds, monitors, pumps, ventilators). Obtain extra supplies if needed; arrange stand-downs and organise extra staff for next 2 shifts. Liaise with ICU registrar and consultants regarding transfers to wards, NICU, theatre recovery annex, home; decide which ICU bed spaces to use for resuscitation and which staff will man them; identify 3 nurses to manage 2 Code Brown patients in Medical imaging area with PICU registrar / Fellow; and allocate appropriate nursing staff to manage lower-acuity patients in operating theatre recovery annex if needed; nominate helipad triage nurse.

The ICU general consultant on duty. Call in ICU medical staff for roles listed below; liaise with ICU cardiac consultant; consider transfer of patients to neonatal unit, cardiology or other wards; liaise with the Emergency Department and triage point; arrange teams of ICU registrar / fellow and a nurse from ICU to assist in ED. Supervise work of the ICU registrars in ICU: resuscitation and management of critically ill patients; full assessment of patients; and arrange investigations and consultations as per EMST-APLS guidelines. Decide with ICU cardiac consultant whether additional ICU consultant staff needed to manage existing ICU patients in cardiac and general units.

PICU consultant / fellow stationed in the ED

Work with consultant anaesthetist, surgeon and ED consultant in the ED. The role of PICU consultant / fellow stationed in ED is to provide clinical support, assist with triage, and supervise procedures (intubation, line insertion) and transfer of patients to Medical Imaging, OT and PICU. Triage in this setting includes prioritisation of patients for distinct segments of care including theatre and CT. Supervise the management of intubated or critically injured patients in ICU areas outside PICU, including the Medical imaging holding bay. Relay status of the Code

Brown and number and severity of expected patients to staff in ICU. Liaise with PICU consultant executive for Code Brown, the cardiac ICU consultant, AUM.

ICU consultant executive for Code Brown. Main role is organization and liaison. Attend control centre meetings to give details of PICU response/needs and to find out more about the emergency. Liaise with the triage team (ED team and helipad); liaise with control centre and ED about expected numbers of patients, and estimate the number needing PICU; assess the need for a site team and allocate tasks to the ICU registrar and the ICU-emergency nurse who would form the site team; liaise with RCH Communication / Public Relations officer; liaise with surgeons and anaesthetists as needed; liaise with PIPER. Call in additional ICU consultants or registrars as needed to supervise patient transfers to CT, assist in ED, or manage intubated / critically injured patients in ICU areas, including Medical imaging recovery bay, and Theatre recovery ICU annex. Maintain close contact with other PICU Consultants, share information.

Team Support Nurse (TSN)

Before children arrive make sure all empty rooms are set up with basic equipment including pens, torches, documentation. Collect used equipment and ask PSA's to clean it and return it to shelves. Prepare IV fluids, arterial and central lines. Delegate tasks: e.g. nurses with less sick children to make up fluids. Record ages and weights of patients as they are available. Set up ventilators; arrange cots and beds; obtain extra Code Brown supplies from Material Resources, Pharmacy, Clinical Technologists and linen room. Assist with transfers and discharges as required. Be aware that PICU may need (i) to open an annex in the Theatre recovery area for less sick children, and (ii) PICU may staff 2 beds in Medical imaging holding area with 3 nurses and a senior PICU registrar if needed. These patients may be very unwell, requiring ventilation, resuscitation; TSN should provide support there as well.

ICU ward clerk, or person designated as clerk. Get staff telephone numbers and discuss with AUM what the text message should say. Keep a list of staff contacted and responses. Call in extra nursing staff as directed; answer ICU phones; arrange transport of supplies and pathology specimens; assist AUM with communication with other areas of the hospital; assist AUM with documentation of all new arrivals; with rostering of next 2 nursing shifts and with arranging extra nurses as needed; liaise with public relations about families and visitors; arrange accommodation for relatives if possible.

ICU technologist. Locate and assemble equipment required (in ICU, from other wards, or from outside RCH); perform special tasks as needed (eg. set up haemofiltration); ensure adequate gas supplies and electrical connections for patients managed in remote locations (eg. NNU and DSU); assist in transfer of patients.

See the RCH Intranet home page: Emergency Procedures – Code Brown

DIURETIC THERAPY

Intravenous frusemide should be given slowly, at a *maximum* rate of 0.05 mg/kg/min (eg 1 mg/kg over at least 20 min).

Frusemide has a half-life of only 30-120 minutes so, in comparison to 3-6 hourly intermittent administration, a continuous infusion gives less fluctuation in urine output and CVP, lower dose requirements, and less urine sodium and chloride loss. However, frusemide is incompatible with dobutamine, esmolol, fentanyl, gentamicin, midazolam, noradrenaline, phentolamine, phenytoin and vancomycin – so another IV line is often needed for a continuous infusion. If mixed at a Y connection just before the patient, frusemide is compatible with adrenaline, alprostadil, aminophylline, GTN, insulin in albumin, isoprenaline, levosemindan, lignocaine, magnesium sulphate (in dextrose), naloxone, nitroprusside, vasopressin, KCl and lipid.

To prevent hypokalaemia and improve the diuresis, spironolactone can be given with frusemide (unless low doses are being used for just a few days).

If a strong diuretic effect is required, give hydrochlorothiazide (a tubular diuretic) as well as frusemide (a loop diuretic) and spironolactone. The risk of severe hypokalaemia with this combination can be reduced by adding acetazolamide.

Frusemide + spironolactone + hydrochlorothiazide + acetazolamide is a very potent combination that may cause hyponatraemia and hypovolaemia. It should be used for no more than a few days, with close monitoring of electrolytes, acid-base and intravascular volume.

ECLS - ECMO

Indications: Progressive respiratory (VV-ECMO) and/or cardiac (VA-ECMO) failure not responsive to maximal medical management. cVAD for single ventricular failure (ALCAPA, TGA) or bridge to long-term VAD.

Contraindications: Irreversibility of underlying condition, GA<34 weeks, intracerebral bleed, post-BMT. Every ECMO referral should be discussed with the ECLS consultant on call, especially where there are relative contraindications or uncertainty

DP3 circuit: <10 Kg ¼" Hilite 2400LT oxygenator; >10 kg 3/8" Hilite 7000LT oxygenator. Blood prime if <10kg.

Flows: VA:150ml/kg/min (<10kg) or 2.4L/m²/min (≥ 10kg). VV: about 70% of VA flows. Higher flows needed in sepsis and CHD with systemic to pulmonary shunts. Monitor blood flow & SvO₂ with Spectrum monitor. Target inlet pressure > - 20mmHg, plasma Hb <0.1g/L.

Gas Exchange: Initial sweep gas to blood flow ratio 0.7:1 if <10kg & 1:1 if >10kg. FiO₂ 0.5. Resting lung ventilation: 10 x 20/10 with FiO₂ 0.3-0.5. Target PaO₂ 80-100mmHg and PaCO₂ 40-45mmHg.

Anticoagulation with Heparin bolus 50-100 U/Kg at cannulation followed by 10-40u/kg/hr infusion, adjusted to maintain ACTs 150-170sec (<15 kg) or aPTT 70-90sec (≥15kg).

Epoprostenol 5ng/kg/min & **NO** 20 ppm. Maintain Plt>80-100x10⁹/L, Hb 80-100g/L, Fibr>1.5g/L.

If **bleeding** (>4ml/kg/h for 4 hours), reduce heparin to 10 u/kg/h, stop epoprostenol, maintain Plt> 100-120x10⁹/L, Hb>120g/L, Fibr>2g/L, surgical exploration.

Haemofiltration in line with ECMO circuit (drainage post-oxygenator, return pre-oxygenator), no heparin prime and heparin infusion 2U/Kg/h pre-filter.

ECMO management

Order 1 unit of RBC if cross match available or 1 unit of O-Neg irradiated blood if cross match not available. A clear prime circuit can be used during ECPR and if ≥10kg.

Adminster 50mg/kg of cephazolin before cannulation; no antibiotic prophylaxis is needed during the ECMO run.

Ensure good central venous access with extension so taps accessible when patient draped, volume attached. Ensure good arterial access & obtain ABG soon post cannulation.

Cannulation site based on type of support, patient age and underlying condition (see Intranet guideline).

Cannulae position and adequacy of support to be reviewed by team members through CXR & echo within 1 hour from cannulation (Huddle).

Invasive procedures on ECMO are discouraged and need to be discussed with ECLS consultant.
For sedation on ECMO & weaning off support see Intranet guideline.

ECLS - DURABLE (LONG-TERM) VAD

Eligibility criteria for durable VAD insertion:

1. Transplant candidacy confirmed by formal assessment
2. Cardiac or end organ recovery likely within 4 weeks of adequate circulatory support

Patient selection and timing:

Timing and indication for device support should be defined, and destination

Risk factors include weight <5kg, end organ dysfunction, need for biventricular support, or single ventricle physiology

Most children receive ECMO or centrifugal VAD prior to conversion to durable VAD

Primary durable VAD insertion should be considered for those without end organ dysfunction

Centrifugal VAD (< 14 days) may be used to allow for end organ recovery

Chronic inotrope or ventilator dependence can be indications for durable support

Device selection is determined by patient and ventricular size

Long term venous access inserted at time of durable VAD cannulation

VAD Configuration (In order of increasing risk)

Left ventricular (LV apex to ascending aorta)

Right ventricular (Right atrium to pulmonary artery)

Biventricular (RA:PA and LV:Aorta)

Single ventricular (Single ventricle apex to ascending aorta)

Cavopulmonary (SVC-IVC conduit to pulmonary arteries)

Surgical considerations:

Optimal inflow cannula placement must be confirmed by VAD operation and TOE

Closure of residual intracardiac shunts

Aortic incompetence is poorly tolerated, consider valve repair or replacement (not closure)

Tricuspid valve repair may reduce the risk of right heart failure

VAD devices used at RCH

Berlin Heart (EXCOR)

Paracorporeal pulsatile device

Patient size < 20kg / LVEDD < 40mm
Pneumatic driving system (IKUS)
Pump size 10 / 15 / 25 / 30 / 50 ml
Initial anticoagulation with Heparin, aspirin and clopidogrel start
6-24 hours post op, then convert to long term Warfarin or
Clexane for infants

Usual operating parameters (discuss with VAD team)
VAD rate 60 - 120 bpm

- systolic pressure +50 mmHg above patient systolic
- diastolic pressure -20 to -50 mmHg
- systolic time 30 - 40%
- it is not appropriate to titrate VAD settings to MAP

Berlin Heart clinical practice tips

1. Poor chamber filling;
 - Assess preload / rhythm / RV function / PVR / pleural effusion or tamponade / cannula position
 - High mean airway pressure will affect venous return
 - Consider volume / ECHO / decrease VAD rate or % systole / diastolic pressure more negative
2. Poor chamber emptying;
 - Assess afterload / outlet cannula position / chamber for thrombus
 - Consider dilator / increase systolic pressure or % systole / decrease VAD rate
3. Fibrin / clot in chamber;
 - Review anticoagulation / notify VAD team
 - Consider CT brain even for subtle neurological changes
4. Pneumatic or driver malfunction;
 - Hand pump (60 - 90 bpm) / cardiac call-out / change IKUS unit
 - Oxygenate, ventilate and support the RV

Heartware HVAD

- Intracorporeal centrifugal continuous flow pump (160g)
- Patient size > 20kg / LVEDD > 45mm
- Driveline connects to controller and batteries
- Anticoagulate with warfarin and aspirin

Usual operating parameters (discuss with VAD team)
- Speed 2200 - 3200 rpm (device range 1800 - 4000 rpm)
- Estimated flow determined by speed, power (variable) and blood viscosity (HCT)
- Continuous flow pumps are preload dependent and afterload sensitive

- MAP should be maintained < 80mmHg (< 70 mmHg for younger child)
- It is not appropriate to titrate VAD settings to MAP

Heartware clinical practice tips

1. Baseline flow and amplitude;
 - Maintain baseline >2L/min, increase speed
 - Maintain amplitude > 2 L/min, amplitude will decrease with increasing speed
 - Baseline flow should never fall below 0 (regurgitant region)
2. Decreased estimated flow;
 - Low amplitude waveform = RV failure or tamponade

consider volume / iNO / inotropes / ECHO / CT mediastinum

- High amplitude waveform = systemic hypertension
- Anaemia presents as decreased estimated flow over days (check VAD HCT)

3. Increased estimated flow;
 - Low amplitude waveform = vasodilatation or aortic incompetence
 - High amplitude waveform = hypervolaemia
 - Pump thrombosis presents as increased estimated flow over days
4. Aortic valve function;
 - Intermittent aortic valve opening may be beneficial
 - Aortic incompetence is poorly tolerated, reduce after load and device speed
 - Increasing device speed with significant aortic incompetence will lead to low cardiac output
5. Patient may not have a palpable pulse
6. Oscillometric BP not reliable, use Doppler method
7. No external 'hand pump' and CPR is not recommended
8. Usual Mx for defibrillation and APLS - oxygenate, ventilate and support the RV

ENDOTRACHEAL TUBES

<10yr: use a nasal endotracheal tube (but not if there is a possible fractured base of skull, or coagulopathy).

>10yr: use an oral endotracheal tube (unless there is a particular reason to use a nasal tube).

We now use Microcuff ETTs for all children, except those with croup.

- On insertion of an ETT, listen for an air leak around the tube. If no leak is heard, insert a smaller tube
- Once a leak is heard, inflate the cuff with air via the one way valve until the leak just disappears. Record the amount of air in the cuff on the vital signs chart
- With the introduction of Microcuff ETTs, which have a low pressure cuff, it is no longer necessary to deflate the cuff for short periods frequently.
- Remember that air can be absorbed into the cuff, even when it is not intentionally inflated.

LARYNGEAL MASK AIRWAY SIZES

Size 1	<5kg	Size 3	30-50kg
Size 1.5	5-10kg	Size 4	50-70kg
Size 2	10-20kg	Size 5	70-100kg
Size 2.5	20-30kg	Size 6	>100kg

CHEST PHYSIOTHERAPY

Definite benefit. In chronic diseases with large amounts of sputum: cystic fibrosis, bronchiectasis, chronic bronchitis.

Probable benefit. For lobar atelectasis in ventilated patients; benefit may be from bagging-suction rather than vibration-percussion

Possible benefit. Deep breathing exercises for atelectasis after cardiac surgery. Vibration-percussion probably harmful.

No benefit, potential harm. Pneumonia, bronchiolitis, acute asthma

EPIGLOTTITIS

There is usually a low-pitched expiratory stridor in an unwell, febrile child who is sitting up with an open mouth and drooling saliva. If there is a cough, it is usually just to expel saliva from the mouth, and it does not have the harsh barking quality found in children with croup.

Epiglottitis is now very rare. This increases the risk that, when epiglottitis does occur, the diagnosis will be missed or management will be inappropriate. Almost all cases were caused by *Haemophilus influenzae* type b, but now occasional cases are due to other organisms (e.g. Staph or group A strep) or other cause of epiglottic injury / inflammation.

Call the ICU consultant immediately if there is any suspicion of epiglottitis.

1. Do not lay the child down.
2. Do not examine the child's throat.
3. Give two doses of ceftriaxone: 100 mg/kg IM or IV stat, and 50 mg/kg after 24 hours, plus flucloxacillin if Staph suspected.
4. Steroids and adrenaline are not helpful.
5. Most children with epiglottitis will need to be intubated, but the procedure can be difficult. Obtain expert help before attempting intubation. It is performed as for croup. Achieve deep anaesthesia, do not put a pad under the shoulders, apply cricoid pressure, be prepared to aspirate copious saliva, and, if the tube sticks just below the cords, flex the neck and twist the tube on its long axis while gently pushing it down the trachea. **Do not intubate without informing the ICU consultant.**
6. Do not give paracetamol. Extubate when the child is afebrile, usually at 12-24 hours.

FEVER – TREATMENT IN ICU

	Unparalysed*	Paralysed
Brain injury (head injury) meningitis, encephalitis, febrile fit), cardiac failure.	Paracetamol if >38.5°C, aim for 36-37.5°C. Paracetamol often ineffective; consider low dose chlorpromazine or paralysis and cooling.	Paracetamol (± cooling blanket), aim for 36-37°
Respiratory failure (croup, pneumonia, bronchiolitis).	Paracetamol if >38.5°C, aim for 37-38°C.	Paracetamol (± cooling blanket), aim for 37-38°
After cardiac surgery	Paracetamol to 38°	Paracetamol achieve 37-38° (± cold PD) to achieve 37-38°C. May need hypothermia for some tachyarrhythmias.
Epi-glottitis	Paracetamol only if > 40°C.	

***Do NOT nurse naked or use topical sponging, fans or cooling blankets in an unparalysed child – even if cooling to normothermia.**

Paracetamol may be needed to treat pain or discomfort (regardless of the temperature).

- Fever does not cause pain (although many infections cause significant discomfort as well as fever). Do not give paracetamol routinely to treat fever (unless it is contributing to respiratory or cardiac failure, is causing an arrhythmia, or there is traumatic or hypoxic brain injury, or pain). Antipyretics increase viral shedding and impair the antibody response; they prolong the illness in influenza, chickenpox, malaria, and perhaps measles; and paracetamol does not prevent febrile convulsions.

FILTRATION

Haemofiltration (veno-venous)

Indications: Renal failure with fluid overload, hyperkalemia, acidemia or severe uremia and removal of toxins (inborn errors of metabolism and drug-toxicity non plasma-bound).

Cannula: 6.5 Fr double lumen in newborn/infants (blood flow 8-10 ml/Kg/min), 8 Fr in child (blood flow 5-8 ml/Kg/min), 11.5 Fr in teenager (blood flow 4-6 ml/Kg/min), 13.5 Fr in adolescent or adult (blood flow 2-4 ml/Kg/min). Max blood flow 200-250 ml/min.

Blood prime if weight < 10 Kg, Hb < 100 g/L or priming volume > 15% of circulating volume.

Filter: Removes particles of MW up to approx 30,000 da.

Filtrate: 20-35 ml/kg/hr (monitor serum creatinine), maintain ratio of blood/filtrate flow of 5 or more.

In sepsis keep filtrate \geq 50 ml/kg/hr.

In Hyperkalemia \geq 500 μ mol/L, rhabdomyolysis with CK >5,000 U/L and associated renal dysfunction (see Rhabdomyolysis) or fulminant/hyperacute liver failure use **high-flow filtration with UFR>200 ml/Kg/h**. To achieve such high filtration rate, accept a blood/filtrate ratio of 3:1 and/or increase the filter surface area by using a bigger filter or 2 filters in series. For hyperammonaemia also lactulose 1ml/kg of 3.3g/5ml every hour until loose stools are produced, then 6-8 hourly, and ammonia scavengers (see METABOLIC – HYPERAMMONAEMIA).

Heparin: start infusion at 10 U/Kg/h pre-filter and titrate to keep ACT 1.5 x normal (Actalyke 140-160 seconds); post-filter Heparin is set at 10% of pre-filter Heparin.

Replacement fluid: < 2 years: RCH standard solution (Dextrose 0.18%, Na 140 mmol/L, K 3 mmol/L, Ca 2 mmol/L, Mg 1 mmol/L, Bic 25 mmol/L, Phos 1 mmol/L, Cl 110 mmol/L, Acetate 23 mmol/L); **\geq 2 years** Haemosol B0 (Na 140 mmol/L, Ca 1.75 mmol/L, Mg 0.5 mmol/L, Bic 32 mmol/L, Cl 109.5 mmol/L, Lactate 3 mmol/L).

Complications: Hypotension, Fluid imbalance, Electrolyte abnormalities, Metabolic Acidosis or Alkalosis, unwanted clearance (water-soluble vitamins and amino acids), Hypothermia, Thrombocytopenia.

Regional Anticoagulation

Indicated in patients with high risk of bleeding. 2 methods:

1-Acid citrate dextrose solution, formula A (ACDA): ACD pre-filter at 1.8 ml/hr per 1 ml/min blood flow.

Maintain pre-filter Ca^{++} 0.25-0.3 mmol/L (measure hourly).

Replacement fluid: < 2 years RCH standard solution, ≥ 2 years

PrismOcal.

Calcium Chloride 10% infusion post-filter at approx 3-5% of ACD flow rate. In children < 2 years receiving RCH solution start Calcium Chloride 10% infusion at 1-2 ml/h.

Maintain patient arterial ionized Ca 1.0-1.25 mmol/L.

Monitor Magnesium and Bicarbonate levels.

Contraindications: Severe hepatic dysfunction, metabolic condition and patient with "Citrate lock".

2-Heparin-Protamine: start Heparin infusion at 10 U/Kg/h pre-filter and titrate to keep the circuit ACT 1.5 x normal (Actalyke 140-160 seconds); post-filter Heparin is set at 10% of pre-filter Heparin.

Administer 1 mg/h of Protamine for every 100 units/h of total Heparin into the returning arm (venous side) of the VasCath to gain normal ACT in patient (90-120 seconds).

In regional Anticoagulation, if red and blue lumen of the VasCath are swapped, the risk of recirculation is high and Calcium or Protamine must be administered directly to the patient via CVL.

Plasmafiltration (veno-venous)

Indication: Removal of large molecular weight substances from blood (autoantibodies, immunocomplexes,) and intoxications with drugs that are highly protein bound (beta-Blockers, Calcium-channel blockers, phenytoin, mushroom).

Cannula: 6.5 Fr double lumen in newborn/infants (blood flow 8-10 ml/Kg/min), 8 Fr in child (blood flow 5-8 ml/Kg/min), 11.5 Fr in teenager (blood flow 4-6 ml/Kg/min), 13.5 Fr in adolescent or adult (blood flow 2-4 ml/Kg/min).

Filter: Removes particles of MW up to 2,000,000 da.

Filtrate: 1.5-2 plasma volume exchange (max 3-3.5 L) over 4-6 hours. Maintain ratio of blood/filtrate flow of 5 or more.

Heparin: start infusion at 10 U/Kg/h pre-filter and titrate to keep the circuit ACT 1.5 x normal (Actalyke 140-160 seconds); post-filter Heparin is set at 10% of pre-filter Heparin.

Acid citrate dextrose solution, formula A (ACDA): 1 ml/hr pre-filter for every 1 ml/min of blood flow.

Replacement fluid: Albumin 30 g/L, Dextrose 0.3%, Na 135 mmol/L, K 3.5 mmol/L, Ca 2 mmol/L, Mg 0.7 mmol/L, Bic 25 mmol/L, Phos 1.5 mmol/L, Cl 100 mmol/L, Acetate 9.2 mmol/L.

Give 1 bag FFP (about 230ml) per 800ml replacement. Use cryoprecipitate 5ml/kg if fibrinogen <2g/L and in TTP.

Complications: Hypotension, Electrolyte abnormalities, Metabolic Acidosis or Alkalosis, unwanted clearance (water-soluble vitamins, amino acids and protein-bound drugs), Hypothermia.

FLUID AND ELECTROLYTES

Anion gap = Na – (bicarb + Cl). Normal <12.

Anuria (noncatabolic): urea rises 3-5 mmol/L/day, creatinine rises 0.05-0.1 mmol/L/day.

Blood volume = 85 ml/kg in neonate, 70 ml/kg in adult.

Chloride deficit: ml 20% NaCl = Wt x 0.2 x (104 – [Cl]).

Extracellular fluid: birth 400 ml/kg, >1 yr 250 ml/kg.

No. mmoles = mEq/valence = mass (mg) / mol. Wt.

Osmolality serum = 2Na + 2K + gluc + urea (mmol/l).

Plasma volume = 45ml/kg in neonate, 35 ml/kg in adult.

Na deficit: ml saline = Wt x 4 x (140 – [Na]) / (% saline).

Sodium in hyperglycaemia = Na + 0.3 (glucose – 5.5)

Urine: minimum acceptable is 0.5-1.0 ml/kg/hr.

Water deficit ml = 600 x Wt(kg) x (1 – 140/sodium).

Intravenous fluid requirements (ml/hr)

Wt(kg)	3	5	7	10	15	20	25	30	40	50	60	70
Active	12	20	30	40	50	60	65	70	80	90	95	100
Sick	6	10	14	20	25	30	35	40	45	55	60	70
Intubated	5	7	10	14	17	21	25	28	32	40	45	50

Neonates: 2ml/kg/hr (day 1 of life), 3ml/kg/hr (day 2 of life),

4ml/kg/hr (day 3 to 12mo).

Post-cardiac surgery: see Cardiac Admission – Post-Op.

Modification of the IV fluid volume given to an active child:

Renal failure x0.3 + urine output; basal x0.7; high ADH (IPPV, brain injury) x0.7; high room humidity x0.7; humidified gases x0.75; hypothermia -12% per °C; burns +4% per 1% burnt 1st day, then +2% per 1% burnt; hyperventilation x1.2; neonates preterm x1.2, radiant heater x1.5, phototherapy x1.5; normal activity (oral intake) x1.5; fever +12% per °C; room temperature over 31°C +30% per °C.

Usual fluid: Plasma-lyte 148 in 10% dextrose (<12mo), Plasma-lyte 148 in 5% dextrose (>12mo).

Brain injury (trauma, meningitis, encephalopathy): 0.9% saline in 10% D (<12mo), 0.9% saline in 5% D (>12mo).

Know the total fluid intake (TFI) for every child every day

Hypoglycaemia

Each day calculate and document the amount of glucose every child on IV fluids is receiving in mg/kg/min.

Glucose mg/kg/min = mls/hr of infusions x % glucose infusion) / (6 x body weight in kg)

eg 12 mls/hr of 10% glucose + 0.45% NaCl in a 4 kg infant = $(12 \times 10)/(6 \times 4) = 120/24 = 5 \text{ mg/kg/min}$. Aim for 4-6mg/kg/min
Give enteral nutrition wherever possible

If needed in infants on restricted fluids, infusions of 50% glucose at low rates can be used to maintain normoglycaemia. 50% dextrose must be given by a central line. It should not be given as a bolus.

For the treatment of hypoglycaemia, use 2ml/kg of 10% dextrose, then increase glucose intake (start or increase enteral feeds, increase glucose concentration in maintenance fluid, add low rate infusion of 50% dextrose if CVC).

Never give insulin without a source of intravenous glucose in a non-diabetic patient

Peritoneal dialysis

Cycles. Ventilated: 10-20 ml/kg/cycle (in/dwell 20 min, out 10min). Normal: 10ml/kg/cycle (in 40min, out 20min).
1.5% isotonic, 4.25% hypertonic. Potassium 0-4 mmol/L.

GENETIC TESTING COMPLEX CONDITIONS

Recently whole exome sequencing (WES) has become available and evaluated in children in ICU, showing a high yield of recognisable single gene defects for children with undiagnosed syndromes, and that the identification of a genetic diagnosis often changes management.

Early referral to Genetics for consideration of WES should occur for children with complex undiagnosed conditions who are seriously ill. These conditions include:

- Neuromuscular diseases
- Syndromic cardiovascular malformations
- Hypertrophic cardiomyopathy
- Skeletal malformations and/or dysplasia
- Neonatal cholestasis and liver failure
- Cystic renal disease
- Metabolic disorders with lactic acidosis
- Immunodeficiency or bone marrow failure

Many of these disorders can be diagnosed with simpler, more targeted tests, and often Genetics and other departments will already be involved, so check what diagnostic tests have already been done. If management may change on the basis of a specific genetic diagnosis, discuss with the bed-card unit and refer to Genetics for WES.

GUILLAIN-BARRE SYNDROME

Progressive weakness with reduced reflexes, often starting in legs and arms about 10 days after an infection of respiratory tract (eg. mycoplasma) or bowel (eg. *C jejuni*). Usually symmetrical, often muscle pain and parasthesiae, cranial nerves may be involved, usually afebrile, may be severe autonomic dysfunction. CSF protein >0.9 g/l, cells <10 per mm³. CK normal or moderately raised.

Exclude cord compression, transverse myelitis, tick paralysis, botulism, myasthenia gravis, polio, lead poisoning, porphyria.

Serology for CMV, EBV, *C jejuni* and *M pneumoniae*; faeces PCR for enterovirus, culture for *C jejuni*; NPA for *M pneumoniae* PCR if respiratory illness. Consider tests for ANF, HIV, Zika virus in certain circumstances.

Immunoglobulin 1g/kg IV over 8 hr daily for 2 doses. If no response after 3-5 days, consider plasmapheresis 50ml/kg exchange alternate days to total 250ml/kg, then give (or repeat) immunoglobulin.

1. Steroids are probably not effective.
2. Measure vital capacity using spirometer if possible. Ventilate if vital capacity below 20-25 ml/kg, or if drooling, no cough or poor bulbar function. Low pO₂ and high CO₂ are late signs; ventilate before they occur. Weak children may not appear distressed despite maximal respiratory effort.
3. Autonomic dysfunction may cause serious hypertension, hypotension, tachycardia or bradycardia; sudden death may occur. Monitor ECG and blood pressure closely. Avoid hypovolaemia. Pacing is seldom needed for bradycardia in children, sometimes aminophylline useful for bradycardia.
4. Catheterisation may be needed for urinary retention. Constipation may be severe; erythromycin or neostigmine may help.
5. Pain may be severe. Try paracetamol, and gabapentin 15mg/kg daily NG. If this is inadequate, add tramadol 2-8 mcg/kg/min. Morphine may be required, but worsens constipation. Carbamazepine is less effective than gabapentin; amitriptyline is probably more effective than gabapentin, but may exacerbate autonomic instability. Steroids probably do not reduce pain.
6. Enoxaparin and stockings should be used in adults, but not in children.
7. Children with GBS will almost all recover. Delayed recovery or poor outcome more likely if (a) a ventilated patient has not started to improve within 18-21 days of reaching peak deficit; (b) if ventilation is needed early; (c) EMG shows absent motor

responses or axonal involvement; or (d) there is *C jejuni* or CMV infection.

HAEMATOLOGY

Compatible blood groups

PATIENT	WHOLE PACKED			CRYO- PRECIP
	BLOOD	RED CELLS	FFP	
O	O	O	Any	O
A	A or O*	A or O	A or AB	A
B	B or O*	B or O	B or AB	A
AB	Any*	Any	AB	A

*Low titre gp O blood; do not use whole blood.

Platelets: donor preferably has same ABO and Rh groups as recipient, otherwise usually as for FFP.

Avoid giving FFP or platelets rapidly for hypovolaemia: they may cause vasodilation and severe hypotension.

Except in an emergency, all blood given to ICU patients should be irradiated just before administration.

In ICU, unless the patient is known to be CMV +ve, give CMV – ve blood or use an Imugard III-RC filter.

Platelets will usually have been leucocyte filtered by central blood bank. If giving unfiltered platelets (from interstate), use an Imugard III-PL filter.

Contact Blood Bank and on-call haematologist and activate massive transfusion protocol if transfusion requirement >40ml/kg of PRBC.

https://www.transfusion.com.au/disease_therapeutics/transfusion

IMMATURE / TOTAL NEUTROPHIL RATIO (I/T RATIO)

The immature to total neutrophil ratio is a useful guide to the presence of sepsis in ICU patients – it is much more accurate than the total neutrophil count. The I/T ratio is calculated from the number of immature neutrophils (blasts and myelocytes + bands) divided by the total number of neutrophils (immature + mature) in the peripheral blood.

The I/T ratio is usually <0.2 in young infants, and <0.15 over about 3 months of age. After cardiopulmonary bypass, the I/T ratio is usually <0.5 (day 1), <0.35 (day 2), <0.3 (day 3), <0.25 (days 4-5), and <0.2 thereafter. The change from day to day is more informative than a single absolute value – eg. a ratio of 0.25 on day 5 would be reassuring if it had been 0.40 the day before, but suggestive of sepsis if it had been only 0.10 the day before. In addition, the acute onset of neutropenia strongly suggests sepsis (even if the I/T ratio is normal). Integrate the results of the I/T ratio with serum procalcitonin in deciding to scale down or cease antibiotics if cultures negative at 48 hours.

HEAD INJURY

Summary: severe traumatic brain injury: if flexor (decorticate), extensor or flaccid: intubate and ventilate (CO₂ 35-40, O₂ sat >90%), sedate (morphine, midazolam), paralyse, cool to normothermia and until ICP <20mmHg, ICP catheter (if ICP >20: vent CSF, cool, 3% saline/mannitol 12.5% 2ml/kg, thiopentone), Na 140-150 (3ml/kg 3% saline over 1hr prn, can give over 5 min if ICP spike), normovolaemia (CVP 5-10), maintain CPP (neonate >30 mmHg, 1-6mo >35, 6-11mo >40, 1-4yr >45, 5-9yr >50, 10-15yr >55, >15yr >60), Noradrenaline for hypotension/ CPP, levetiracetam, glucose 4-10 mmol/L (dextrose and/or insulin), consider SSEP day 2, MRI.

A. INTUBATION AND VENTILATION

For all patients who present with:

- a. flaccid or extensor (decerebrate) or flexor (decorticate)
- b. or deteriorating conscious state
- c. or respiratory failure.

Maintain a PaCO₂ of 35-40 mmHg, oxygen saturation >90%, and provide PEEP of 5 cm H₂O.

B. PARALYSIS, SEDATION, ANALGESIA

Following initial neurological assessment:

1. Analgesia

- a. morphine by intravenous infusion at 40-80 mcg/kg/hr
- b. morphine bolus 50mcg/kg (2.5ml/kg of 1mg/kg in 50ml) IV before painful procedures e.g. turning in a child with fractures, leave adequate time to take effect (5-10 minutes).

2. Sedation

- a. midazolam 1-4mcg/kg/min
- b. give extra boluses of midazolam 0.2 mg/kg if response to stimuli (eg. tachycardia, increased blood pressure or lacrimation on passive movement of limbs) or high ICP, and consider increasing infusion (up to 18mcg/kg/min used rarely) or adding diazepam 0.1 mg/kg 4H slow IV.

3. When thiopentone is used all other sedative drugs should be ceased. (Morphine/fentanyl can be left on a small dose if significant pain likely). If thiopentone used, ideally do continuous EEG monitoring.

3. Paralysis

- a. all patients until verification of head injury status is obtained through CT scan
- b. all patients when it is decided to insert an ICP catheter
- c. all patients with sustained intracranial hypertension (paralyse)

for at least 72 hours from the time of injury)

- d. can use pancuronium: 0.1-0.15 mg/kg prn or vecuronium 0.1mg/kg.

Do not continue to paralyse the patient if a decision has been made not to insert an ICP catheter. Avoid vecuronium infusions, as likely to lead to prolonged neuromuscular paralysis.

C. TEMPERATURE CONTROL

1. All patients should have active interventions to preserve normothermia (36-37°C), including a cooling blanket if required. While hypothermia (<35°C) reliably reduces ICP, it has unproven effects on mortality. Therefore outside of a research context, therapeutic hypothermia should be reserved for elevations of ICP refractory to other treatments.
2. For active cooling to therapeutic hypothermia, patients are ventilated, paralysed and cooled using a cooling blanket to first 35°C, then 32-33°C if ICPs remain uncontrolled.
3. Hypothermia is maintained until the ICP is stable and less than 20mmHg.
4. Rewarm gradually via a servo-controlled warming-cooling blanket. The patient's core temperature should be allowed to rise 0.5° every 3hr up to 36-37°C (not over 37°); recool if the ICP rises sharply and the CPP falls.
5. Vasodilation is common during rewarming, and intravascular filling ± noradrenaline is often needed.
6. Do not wean sedatives until ICP stable for 12 hr with cooling blanket turned off.
7. Nutritional requirements are significantly reduced while temperature <36°C
8. With hypothermia infection risk is increased (and clinical signs are masked – i.e. no fever), cease hypothermia if signs of VAP, neutropenia, increased procalcitonin or rising IT ratio.

D. MAINTAIN CARDIAC OUTPUT

1. Correct hypovolaemia: IV 0.9% saline, 5-10 ml/kg bolus prn. Do not use 4% albumin for resuscitation
2. Provide maintenance hydration:
 - a. Total intravenous fluid volume (0.9% saline with KCl 20 mmol/l or Plasmalyte + morphine + IA + inotrope + 50% dextrose):
Wt(kg) 3 5 7 15 20 25 30 40 50 60 70
ml/hr 5 7 10 17 21 25 28 32 40 45 50
- b. Maintain urine output \geq 1.0 ml/kg/hr
3. Biochemistry
 - a. Serum Na 140-150 mmol/l. If <140 mmol/l give 3 ml/kg of 3% saline IV over 1hr; consider reduced water intake.
- b. Maintain blood glucose at 4-10 mmol/L; use 50% dextrose 0.5

ml/kg/hr adjusted as required, or insulin 0.05 u/kg/hr adjusted as required. If BSL consistently >12, reduce IV sources of glucose and commence insulin.

4. Mean arterial pressure:
 - a. Normovolaemia (CVP 5-10, Na 140-150) using 0.9% saline (not albumin).
 - b. Meticulously avoid hypotension and maintain CPP (see age specific targets below), particularly for the first 5 days. After this period a specific CPP aim is controversial. Use Noradrenaline 0.05-0.5mcg/kg/min IV if required.
 - c. Consider addition of adrenaline 0.05-0.1 mcg/kg/min IV (especially if giving thiopentone IV or if cardiac echo demonstrates decreased ventricular function)

E. ICP MONITORING (VIA INTRA-VENTRICULAR CATHETER or CODMAN CATHETER)

1. Instituted for all patients who have:
 - a. extensor or flexor posturing or flaccidity
 - b. or a swollen brain at craniotomy
 - c. or non-purposeful movements, posturing or flaccidity and require a prolonged surgical procedure e.g. laparotomy or orthopaedic procedure.

If ICP is elevated, a catheter placed in the lateral ventricle is *much* preferred to a Codman monitor (which does not allow for removal of CSF). Decompressive craniectomy with duraplasty is associated with lower ICP but more severe disability in survivors. Its utility is restricted to evacuation of haematoma where bone cannot be returned.

2. General principles of management:
 - a. The arterial pressure transducer and ICP transducer are zeroed at the level of the external acoustic meatus to allow ICP and CPP (cerebral perfusion pressure) to be measured from the same baseline.
 - b. Venting of cerebrospinal fluid (CSF) is regulated by the height of attachment of the drainage burette (at the level of the drip point) above the level of the external acoustic meatus. For example, to lower the ICP to 15mmHg pressure whenever the ICP is over 20mmHg, the height of the burette above the external acoustic meatus = $15\text{mmHg} \times 1.36 = 20.4\text{cm}$, so the top of the burette (at the level of the drip point) is secured 20cm above the external acoustic meatus to drain at 15mmHg.
 - c. When continuously venting, the ICP should only be measured after the venting has been turned off at the distal 3-way tap for at least 30 seconds.
 - d. Daily culture of cerebrospinal fluid (CSF): see Procedure Manual - Intracranial Pressure Monitoring.

3. Treatment of raised intracranial pressure.

The primary objective is maintenance of an adequate cerebral perfusion pressure (CPP) = mean arterial pressure - mean intracranial pressure. Adequate CPP is:

- Neonate: >30 mmHg
- 1 month – 6 mo: >35mmHg
- 6 month – 11 mo: >40 mmHg
- 1 year - 4 years: >45 mmHg
- 5 years – 9 years: >50 mmHg
- 10 years - 15 years: >55 mmHg
- >15 years: >60 mmHg.

If there is sustained intracranial hypertension (ICP 20-24 mmHg for 30min, or ICP 25-29mmHg for 10min, or ICP 30mmHg or more for 1min) without hypertension:

- treat immediately with: sedation bolus; bagging; mannitol or hypertonic saline; and venting
- consider urgent CTB to rule out evolving haematoma/hydrocephalus which may be amenable to neurosurgical intervention.
- after surgery, give full medical management (see below).

If there is high ICP and a high CPP, there must be high blood pressure. Look for the cause of the high BP (eg pain, seizures) and treat it; do NOT give antihypertensives. Avoid wide swings in blood pressure or CPP (low CPP is bad, but very high CPP can have adverse effects also).

Aim for ICP 20 mmHg or less

Medical management of elevations:

- i. Ensure adequate sedation and analgesia, paralysed, CO₂ 35-40, serum Na 140-150, temperature control.
- ii. Moderate hyperventilation via hand bagging only if there is cerebral herniation or an ICP >30 mmHg. Use either mannitol OR hypertonic saline:
Mannitol 0.25-0.5g/kg/dose IV (2-4ml/kg of 12.5%, 1.25-2.5ml/kg of 20%).
Precautions: ensure serum osmolality is no greater than 320 mOsm to prevent risk of renal failure, and do not give more than 3 doses of mannitol per 24 hours due to the risk of cerebral accumulation of mannitol and potentiation of cerebral oedema; OR:
Hypertonic Saline 3%, given in boluses of 2-4mL/kg over 5 minutes. Repeat to maximum 2-4mL/kg 6 hourly PRN. Hold 3% saline if severe hyperosmolar state develops (ie Na >150). Do not give boluses of hypotonic solutions to lower sodium.
- iii. Intermittently vent CSF for five minutes.

- iv. Continuously vent CSF (inform neurosurgical team).
- v. Thiopentone. Avoid hypotension. Slow bolus dose of 1mg/kg IV, repeated not more than 4 times if needed to control seizures or ICP, then 1-5mg/kg/hr IV via central venous catheter. Total cumulative dose to achieve burst suppression may be up to 20-40mg/kg. Level: 150-200 ummol/L (x 0.24 = mcg/ml); Biochemistry Department to be notified of request for thiopentone levels before blood sampling.

Based on the lack of evidence for ICP-directed management for more than the early part of ICU admission, there should be consideration for ceasing all ICP-directed management after 7-10 days (including ICP monitoring). Ceasing ICP-directed management should be agreed by both ICU and neurosurgery.

F. CERVICAL COLLAR AND USE OF SAND BAGS

1. Use sand bags beside the head and a cervical collar on all patients; remove them only on the written instruction of the consultant neurosurgeon or consultant orthopaedic surgeon. Immobilise with tape if transporting a paralysed child. Avoid compression of soft tissues and venous obstruction.
2. X-ray cervical spine: AP, lateral, peg (and right and left 30° oblique if unconscious or uncooperative).
If X-rays normal in a child old enough to clinically assess:
 Aspen collar until conscious and cooperative, then remove collar and assess for pain, tenderness, muscle spasm, or reduced movement:
 - if assessment normal (fully conscious, no neck pain, swelling, tenderness or muscle spasm, full range of movement) no further action
 - if assessment abnormal or the child is unable to be assessed because of young age, lack of cooperation or encephalopathy: replace Aspen or Philadelphia collar, do an MRI to look at soft tissues and cord.*If X-rays abnormal or will be unable to assess clinically:*
 Philadelphia or Aspen collar, MRI (+/- CT). Avoid compression of soft tissues and venous obstruction.

G. ANTIBIOTICS

1. Prophylactic cefazolin only for external compound skull fracture or if surgeons request prophylaxis for EVD.
2. Antibiotics are NOT routinely required for:
 - a. children with a fractured base of skull opening into the nose, middle ear or paranasal sinus. Use high dose penicillin if concerns.
 - b. children with a Codman pressure monitor.
3. Use high dose vancomycin (target level 20-25) and

ceftazidime for suspected or confirmed ventriculitis (>1 WCC to every 500 RBC from EVD CSF).

H. ANTICONVULSANTS

1. Evidence that prophylaxis reduces seizures. Prophylactic anticonvulsants should be ceased after 7 days unless there has been evidence of seizures. Levetiracetam provides equivalent seizure prophylaxis to phenytoin, with the added advantage of: reliable dosing without a requirement for levels; equivalent bioavailability given enterally with no requirement to stop feeds before or after administration; fewer drug interactions; no derangement to LFTs.
 - a. prophylaxis dose levetiracetam 10mg/kg (max 500mg) 12 hourly IV/oral.
2. Monitor for and document all suspected seizure activity.

I. GI/NUTRITION

1. Early enteral feeds if possible, although may not be well tolerated if temp maintained <36°C. If NG feeds poorly tolerated: reduce narcotics, and give IV erythromycin 3mg/kg 8H.
2. Recommended preparations for NG feeding (nil evidence of cows milk protein allergy): ≤1yr maintenance infant formula or EBM, 1-6 years (8-20kg) Nutrini or 7+ years (>20kg) use Nutrison. Use Nutrison Energy (hyperosmolar) if high calorie intake required.
3. Naso-jejunal (NJ) feeding may succeed if NG feeding fails. Recommended preparations for NJ feeds are described in Nutrition.
4. If diarrhoea occurs, investigate cause before stopping feeds.
5. Patients deeply sedated for ICP management should be started on early aperients (ie BD movicol, coloxyl and senna)
6. Stress ulcer prophylaxis (pantoprazole 1mg/kg: max 40mg, IV/oral daily)

J. POSITIONING

All patients should be:

1. Nursed head elevated 30°
2. Maintained with their head in a neutral position with all flexion, extension, lateral flexion, axial traction and rotation avoided during turning.
3. Log rolled from side to side every 2-4 hours and maintained at a 45° angle. The patient's head and shoulders should be supported throughout the turn.
4. Cooling blankets under the trunk only, so that pressure relief is available to the head at all times.
5. Bed can be broken once thoracolumbar spine and pelvis cleared.

K. SPECIFIC DIAGNOSTIC MEASURES

1. CT or MRI of brain (+/- spine) at the time of admission, upon subsequent deterioration in neurological condition, sustained elevations of ICP and at the discretion of the intensivist or neurosurgeon. If doing an MRI brain, consider whether it will be possible to clear the cervical spine clinically – if not do MRI cervical spine also.
2. SSEP (somatosensory evoked potentials) 24-48 hr post injury if not brain dead but poor outcome seems likely. For accurate SSEP ensure: no focal lesion preventing the stimulus reaching the cortex, no subdural or extradural haemorrhage preventing the cortical response being recorded, and no craniectomy in the previous 24hr. Absent SSEPs should be repeated after 24hr. Bilaterally absent SSEPs in setting of severe global injury on CT or MRI, history of severe injury (low motor score at scene), and sustained high ICP indicates very poor outcome.
3. Monitoring of (1) jugular venous oxygen saturation or (2) brain oxygen partial pressure (using a Licox monitor) may improve the management of patients with traumatic brain injury. However, their role is uncertain and they are not used routinely at RCH.

HYPOXIC INJURY

From drowning, cardiac arrest or SIDS, for example. Admit to ICU if there is impaired conscious state or lung disease.

Do a chest X-ray, gas, electrolytes, creatinine, lipase, liver function tests, FBE, coagulation, group and hold. Consider ECG (check for prolonged QT syndrome) and EEG.

Prognosis is determined by multi-modal evaluation – history of event (including rhythm, first response CPR, number of doses of adrenaline needed to achieve return of spontaneous perfusing rhythm), objective clinical neurological signs (in absence of sedative or paralyzing drugs), imaging (MRI) and electrical information (EEG, SSEPs).

1. Maintain airway, breathing and circulation at all times.
2. Pass a large NG tube and remove as much stomach contents as possible.
3. Intubate and ventilate if there is impaired conscious state or evidence of significant lung disease. Pre-oxygenate, use cricoid pressure, give thiopentone 1-2 mg/kg (if circulation stable), vecuronium 0.1 mg/kg IV, wait 2 minutes then intubate quickly.
4. Insert a femoral venous line, and use 0.9% saline to maintain a CVP of 7-10 cm. Correct hypovolaemia if it occurs. If there is hypotension, get a cardiac echo to guide fluid and inotrope therapy – is the left atrium small (give more fluid) or large (restrict fluid); is left ventricular contractility poor (increase inotropes) or too good (reduce inotropes). Correct hyponatraemia by maximum 0.5 mmol/L/hr with 3% saline (4 ml/kg raises serum Na by 3 mmol/L). Give dobutamine (and noradrenaline if required). If there is persistent hypotension, give hydrocortisone 1 mg/kg/dose 6H IV.
5. Maintain serum glucose 3.5-5.5 mmol/L (65-100 mg/100 ml). If glucose is low, infuse 50% dextrose at 0.5 ml/kg/hr, adjusted as required. Hypoglycaemia is more dangerous in children than in adults, and hyperglycaemia is less dangerous: if glucose is >10 mmol/L for over 12hr, consider giving insulin 0.025 u/kg/hr (1.25 u/kg in 50 ml at 1 ml/hr) and monitor glucose hourly.
6. There is limited evidence that hypothermia improves the outcome in children after in-hospital cardiac arrest, but some evidence of improved outcome for adults after out-of-hospital VF and in asphyxiated neonates. If it is used, hypothermia should be induced as soon as possible after the insult, preferably within 6 hr.

7. After near-drowning, give penicillin 50 mg/kg/dose 6 hourly IV for 5 days. If there is severe lung disease, consider urgent bronchoscopy.
8. Do not routinely give steroids or mannitol.
9. Do not insert an ICP catheter (unless traumatic brain injury is present as well). If the ICP is very high, the outlook is very poor, and there is no evidence that treatment of the ICP improves outcome.
10. Check carefully for seizure activity. At the very least, allow relaxants to wear off every 12-24 hours to check for abnormal movements. However, non-convulsive seizures are common and dangerous, and EEG monitoring is required to detect them. Treat seizures with levetiracetam (50mg/kg then 10mg/kg 8 hrly) or phenobarbitone (20 mg/kg IV over 30 min stat, then 10 mg/kg over 30 min as required) or phenytoin (15 mg/kg IV over 1 hr); if required, add midazolam 1-4 mcg/kg/hr.
11. Use PD or haemofiltration early if there is oliguria for more than 24-48 hours, or hyperkalaemia.
12. Be careful not to wean too quickly or too soon. A child who has had a severe hypoxic insult should be ventilated for at least 72 hr, and much longer periods may be needed.
13. After asystolic arrest caused by hypoxia, the prognosis is poor (but not necessarily hopeless) if the pupils remain fixed and dilated for more than 6 hours. A daily full neurological examination should be done, including the tests of brain-stem function (apart from the apnoeic oxygenation test). Lack of motor response to pain, or extensor response, bilateral lack of pupillary reflexes or lack of corneal reflexes at 72 hours, in the absence of sedation are indicators of poor prognosis. However a multi-modal approach to prognosis should be taken (history, clinical signs, MRI +/- electrical)
14. Bilaterally absent somatosensory evoked potentials 24 hours or more after the event suggest a poor prognosis, repeat in further 24 hours if absent. EEG changes indicating a poor prognosis include status epilepticus (despite treatment), burst suppression, alpha coma, or an EEG that is non-reactive to patient stimulation in the absence of sedation.
15. An MRI scan done at 3-5 days will identify the severity and extent of restricted diffusion, which indicates dysfunctional cell membranes seen in ischaemic neurones.

IMMUNE SUPPRESSIVE MONOCLONAL AND BIOLOGICAL AGENTS

Immune suppressive monoclonal antibodies and other biologic immunomodulatory agents are increasingly used in complex patients in the ICU. They are used by many departments: Oncology, Rheumatology, Immunology, Nephrology, Gastroenterology, transplant services and Dermatology. These drugs have complex effects on the immune system, and many potential interactions and adverse effects in critically ill patients. Before commencement of a biologic immunomodulatory agent have a case conference involving the bed-card unit, the unit wishing to prescribe the immune suppressive therapy, the ICU consultant and PICU pharmacist.

Answers to the following questions will be clearly defined.

<p>DRUG PROPOSED TO BE USED Dose and duration</p>
<p>DIAGNOSIS What is the diagnosis or condition being treated? What are the objective diagnostic criteria? What international criteria are being used to confirm diagnosis? What further investigations are needed to be as sure as we can be about the diagnosis?</p>
<p>INDICATION FOR DRUG What is the indication for a biologic immunomodulatory agent in <i>this</i> diagnosis? What is the evidence What other therapies (including less immune-suppressive) should be considered?</p>
<p>OBJECTIVE THERAPEUTIC GOALS What are the objective therapeutic goals? These should be clearly stated, such as reduction in A-a gradient, improvement in renal or liver function, increase in platelet count, and resolution of haemolysis on blood film. There will usually be several objective metrics to assess the effect of any immune therapy, which when triangulated enable a valid assessment of whether the overall therapeutic goal is being achieved.</p>
<p>LABORATORY MONITORING What laboratory monitoring is needed to assess the effects of the drug on the immune system? Specify the exact tests, and frequency</p>
<p>TIME FRAME What is the time-frame for the expected therapeutic goals to be reached?</p>
<p>SIDE EFFECTS AND DRUG INTERACTIONS</p>

<p>What are the common side effects of these drugs? How can these be mitigated? What are the possible interactions of these drugs in this child:</p> <ul style="list-style-type: none"> • interactions with other immune dysfunction the child may have • interactions with other drugs • exacerbation of existing organ injury • potential for systemic effects (neuro-myopathy, SIRS, nutrition)
<p>INFECTION PROPHYLAXIS What prophylaxis of infection is needed?</p>
<p>OPT OUT CRITERIA What criteria will result in cessation of the immune modulation therapy?</p>

Immune suppressive monoclonal antibody and other biologics require the approval of the Drug Usage Committee. A form outlining the answers to the above questions should be submitted to DUC as evidence of a case conference.

Drug	Mechanism of action	Conditions approved for use / experimental uses	Effects	Adverse effects
Adalimumab	Anti-TNF	JIA, IBD		infection, malignancy, arrhythmia, heart failure, HT, headache, rash, vomit, neutropenia, hepatic necrosis, allergic reaction
Alemtuzumab (Campath)	Anti-CD52, expressed on lymphocytes, monocytes, macrophages & NK cells	multiple sclerosis, acute GVHD	Extensive lympholysis by apoptosis of targeted cells	pancytopenia, infusion reaction, fever, infection, malignancy, autoimmune disease, headache, paraesthesia, vomit, rash, thyroid disease, limb pain
Anakinra	IL-1 inhibitor	JIA, KD, FMF, neonatal-onset multisystem inflammatory disease		headache, rash, vomit, thrombocytopenia, leukopenia, infection, allergic reaction

Basiliximab	Anti-IL-2 receptor, anti-CD25	Solid organ transplant	Prevent cell-mediated allograft rejection (IL-2 receptor is critical for cell-mediated rejection)	infection, arrhythmia, heart failure, chest pain, hypertension, hypotension, agitation, headache, cytopenia, CRS, allergic reaction
Blinatumomab	Anti-CD3/CD19	(relapsed/refractory) ALL, B cell lymphoma	Engages T-cell activity by linking CD3+ (T-effector) cells with CD19+ cells. Resultant cytotoxic T cell engagement leads to target cell lysis	Cytokine release syndrome, neurotoxicity (seizures, tremor, encephalopathy), liver toxicity
Chimeric antigen receptor T cells (CART)	anti-CD19 (can be specified)	(relapsed/refractory) ALL, CLL, B cell lymphoma	Engineered T-lymphocytes link to CD19, resultant cytotoxic T-cell engagement leads to target cell lysis	Cytokine release syndrome, neurotoxicity (seizure, encephalopathy, ischemic stroke)
Cyclosporine	Calcineurin inhibitor	Solid organ transplant, GVHD, KD, lupus nephritis, nephrotic syndrome	Inhibit T cell activation	Infection, malignancy, HT, nephrotic, liver toxic, seizure, encephalopathy, BM suppression, TMA, gum hyperplasia, hypertrichosis, GI upset
Daclizumab	Anti-IL-2 receptor (anti-CD25) antibody	Multiple sclerosis		Liver toxic, autoimmune hepatitis, immune-mediated disorder (skin, LN, colitis), seizure, anaemia, lymphopenia, allergic reaction, infection, fever
Eculizumab	anti-C5 monoclonal antibody	PNH, atypical HUS, thrombotic microangiopathy post-BMT	Binds to C5 preventing haemolysis and coagulation	Major: headache, thrombocytopenia, leukopenia, haemolysis, GE complaints, infections, fever & chills, asthenia, oedema, oral herpes, URT infections, anaphylaxis, skin reactions, myalgia

Etanercept	Anti-TNF	JIA, idiopathic pneumonia syndrome, KD, FMF		Infection, malignancy, rash, allergic reaction, injection site reaction, heart failure, CNS demyelination
Infliximab	Anti-TNF	JIA, KD, IBD		Infection, malignancy, cytopenia, rash, heart failure, hepatitis, CNS demyelination, infusion reaction
Mycophenolate mofetil	Mycophenolate mofetil is an ester prodrug that is hydrolyzed to the active immune suppressor mycophenolic acid (MPA). MPA inhibits the activity of inosine monophosphate dehydrogenase, a key enzyme in the de novo pathway of guanosine nucleotide synthesis in B and T lymphocytes that slows their proliferative response	Solid organ transplant, lupus nephritis		hyperlipidaemia, cytopenia, GI upset, infection, malignancy
Polyclonal antithymocyte globulin (ATG)	Cytotoxic antibodies that bind to CD2, CD3, CD4, CD8, CD11a, CD15, CD25, CD44, CD45 and HLA class I and II molecules on the surface of human T lymphocytes	Bone marrow transplant, rejection of kidney transplants	T-cell lympholysis	Major: fevers, dyspnoea, stomatitis, sepsis, CMV infections, UTI, neutropenia, headaches, tremor, GE complaints, malignancy, pancytopenia, anaphylaxis, hyperlipidaemia, hyperbilirubinemia, paraesthesia, veno-occlusive disease, bleeding disorders, myalgia, angina, asthenia, hyperthermia, RTN, haematuria, oedema

Rituximab	Anti-CD20 antibodies	NHL, B cell lymphoma/leukaemia, rheumatoid arthritis, nephrotic syndrome	B-cell lympholysis	Major: infections, CRS, infusion reactions, lymphopenia. Sometimes pancytopenia, opportunistic infections, angioedema, nausea, skin reactions and itch, alopecia, fever, asthenia, headache, low IgG, hyperglycaemia, increase LDH, hypocalcaemia, paraesthesia, pneumonitis (granulomatous or desquamative interstitial)
Sirolimus	Engage FKBP12 (FK binding protein) and modulate activity of mammalian target of rapamycin (mTOR). mTOR inhibits IL-2 mediated signal transduction, resulting in cell cycle arrest.			Cytopenia, hyperlipidaemia, diabetogenic, interstitial pneumonitis, nephrotoxic, proteinuria, FSGS, hyperkalaemia, neurotoxic, hypertension, TMA, skin eruption
Tacrolimus	Binds to FK506-binding protein 12 (FKBP12) to create a complex that inhibits calcineurin, ultimately reduces T cell proliferation	Solid organ transplant		Nephrotoxic, hypertension, seizure, encephalopathy, hyperglycaemia, hyperlipidaemia, hyperkalaemia, liver toxic, hypomagnesaemia, infection, malignancy
Tocilizumab	IL-6 inhibitor	JIA		infection, macrophage activation syndrome, leukopenia, thrombocytopenia, elevated LFT, hyperlipidaemia, injection site reaction, infusion reaction

IMMUNIZATION (VICTORIA)

Birth. Hepatitis B (HB Vax II Paediatric) 0.5 ml IM.

2 months. Diphtheria + tetanus + acellular pertussis + polio (DTPaP) (Infanrix IPV) 0.5 ml IM; Hib (PRP-OMP) + hepatitis B (Comvax) 0.5 ml IM; pneumococcus (Prevenar) 0.5 ml IM; rotavirus (RotaTeq) 2 ml oral.

4 months. DTPaP (Infanrix IPV) 0.5 ml IM; Hib + hep B (Comvax) 0.5 ml IM; pneumococcus (Prevenar) 0.5 ml IM; rotavirus (RotaTeq) 2 ml oral.

6 months. DTPaP (Infanrix IPV) 0.5 ml IM; Hib + hep B (Comvax) 0.5 ml IM; pneumococcus (Prevenar) 0.5 ml IM; rotavirus (RotaTeq) 2 ml oral.

12 months. Measles + mumps + rubella (Priorix) 0.5 ml SC; Hib + hepB (Comvax) 0.5 ml IM; meningococcus gpC (NeisVacC) 0.5 ml IM.

18 months. Chickenpox (Varilrix) 0.5 ml SC.

4 years. DTPaP (Infanrix IPV) 0.5 ml IM; MMR (Priorix) 0.5 ml SC.

School Year 7 (age 12yr). Hep B (HB VaxII Adult) 1 ml IM; chickenpox (Varilrix) 0.5 ml SC; human papillomavirus (Gardasil) 0.5 ml IM at 0, 2 and 6 mo (3 doses).

School Year 10 (age 15yr). Diphtheria + tetanus + pertussis (Boostrix) 0.5 ml IM.

50 years. Diphtheria + tetanus (ADT) 0.5 ml IM.

Over 65 years. Pneumococcus (Pneumovax 23) 0.5 ml IM every 5 yr; Influenza 0.5 ml SC every year.

Aboriginal and Torres Straight Islanders. Additional vaccines: hepatitis A at 12-18mo, and 18-24mo; pneumococcus (Pneumovax 23) 0.5 ml IM at 18mo and 50yr. Aboriginal

Chronic cardiac or pulmonary disease. Additional vaccines: pneumococcus (Prevenar) 0.5 ml IM at 12mo (fourth dose); pneumococcus (Pneumovax 23) 0.5 ml IM at 4yr; influenza vaccine 0.125 ml (3mo-2yr), 0.25ml (2-6yr); 0.5ml (>6yr) SC with a second dose after 4wk the first year, then annual.

Severe immune deficiency. Give non-live vaccines (HepB, DTPaP, Hib, HepB, pneumococcus) on schedule; extra doses may be needed. Avoid live vaccines (varicella, measles, mumps, rubella, BCG, oral polio); give before immunosuppression, if possible. Can give live vaccines if on prednisolone 2 mg/kg/day for <7 days (or lower doses for up to 4wk).

INOTROPES AND VASOACTIVE AGENTS

Adrenaline: (0.01-0.1mcg/kg/min)

Powerful inotrope, vasodilator at low dose. Increases DO₂.

Dobutamine: (2.5-10mcg/kg/min)

First-line inotrope. Lusitropic effects and some vasodilation at low dose. Chronotropic effects can be limiting.

Dopamine: (5-10mcg/kg/min)

Very rarely used inotrope and chronotrope. Worsens VO₂/DO₂ ratio. Does not protect from renal failure.

Glyceryl trinitrate: (0.5-5mcg/kg/min)

Weak vasodilator of mostly venous capacitance vessels. Still used in potential coronary ischaemia or as NO donor.

Isoprenaline: (0.05-1mcg/kg/min)

Chronotrope (and vasodilator) used occasionally for severe bradycardia. Normal myocardium can be very sensitive.

Levosimendan: (12.5mcg/kg over 10 mins, then 0.2mcg/kg/min for 24 hrs)

Ca sensitizer and PDEIII inhibitor with very long half-life. Beware hypotension and arrhythmias.

Metaraminol: (0.01mg/kg bolus)

Short-acting α agonist used to acutely increase BP.

Milrinone: (0.25-0.75mcg/kg/min)

PDEIII inhibitor with long half-life. Powerful vasodilator, modest inotrope and lusitrope. Minimal chronotropy.

Noradrenaline: (0.01-0.1mcg/kg/min)

First-line vasoconstrictor with minimal inotropic effect.

Sodium nitroprusside: (0.05-3mcg/kg/min)

Short-acting powerful arteriolar vasodilator. Rapid tachyphylaxis and risk of toxicity.

Vasopressin: (0.02-0.06u/kg/hr)

Second-line vasoconstrictor.

SUGGESTED APPROACHES

Cardiogenic shock: use adrenaline

Heart failure – see CARDIOMYOPATHY.

Rotating inotrope schedule may be useful: milrinone day 1-7,

levosimendan day 8, dobutamine day 9-14, repeat.

Post-op cardiac – see CARDIAC – general strategies in post-op period.

Brain death – see DEATH – ORGAN DONATION –

Management of haemodynamic instability.

Sepsis – see SEPSIS – SEVERE.

LIVER FAILURE – ACUTE FULMINANT

Multi-system illness with **severe derangement of liver function**

Coagulopathy (INR >2) OR

Coagulopathy (INR >1.5) AND Encephalopathy

Within 8 weeks of liver insult

Causes: Infective, metabolic, drugs, auto-immune, vascular, ischaemic, infiltrative, unknown (almost 50%)

Neonatal: Haemochromatosis (>33%), viral (HSV), haematological malignancies (HLH), liver-based metabolic defects

Indicators of higher risk:

- INR >4 despite 1 dose of vitamin K
- Any coagulopathy in neonates
- Under 5 yrs old
- Bilirubin >235 µmol/L
- Grade III/IV coma in paracetamol overdose
- WBC >9 x 10⁹/L
- Elevated lactate

N/B Fixed dilated pupils may be reversible

Grade of ENCEPHALOPATHY

0. No detectable change in conscious state, behaviour or neurological findings

I. CONFUSED: lethargic; irritable; sleep reversal

II. DROWSY: odd behaviour; mood swings; photophobia, asterixis

III. STUPOROSE: somnolent but rousable; may be aggressive; hyperreflexic

IVa. COMATOSE: rousable to pain; areflexic

IVb. DEEP COMA: unrousable; decerebrate or decorticate

ENCEPHALOPATHY: if encephalopathy ≥Grade 2 or GCS ≤8:

Intubate and insert CVC and vascath for CVVH

Hourly arterial blood gases including Na, K, glucose, lactate

Ventilate to normocapnia: PaCO₂ 35-40 mmHg or to the pre-intubation PaCO₂ if <35

Maintain Cerebral Perfusion: Target mean arterial pressures >70mmHg if over 4yrs, >60mmHg if <4yrs, >50mmHg if <1yr; consider vasopressors

Osmotherapy: aim for Na 148-152mmol/L; infuse 3% NaCl 1 ml/kg over 1 hour then 0.1-1.0 ml/kg/hour via CVC

Assume Raised ICP if: hypertensive, bradycardic or dilated pupil(s)

Aggressively manage signs of raised ICP: bolus 3 ml/kg 3% NaCl. Hyperventilation if ICP crisis
Seizure control: load with IV phenytoin 20mg/kg; and consider phenobarbitone 5mg/kg or thiopentone 3mg/kg
Temperature control: Aim for 35°C: Cooling blanket
Consider CVVH if worsening hyperammonaemia >100µmol/L or grade 2 encephalopathy. Aim for NH₃ ≤60
Routine measures for raised ICP: head-up 30°; head in mid-line

COAGULOPATHY:

Check coag screen + FBE 4 hourly

Aim for: Fibrinogen 1.0 g/L; INR < 5; platelets > 20

Vitamin K 1mg/kg (maximum 10 mg) daily

FFP 10 ml/kg. Platelets 15 ml/kg. Cryoprecipitate 5ml/kg

Use ultrasound for line insertion

BLEEDING DIATHESIS: Invasive procedures; spontaneous intracranial (rare: ~1%); upper GI; menstrual

Prevention: IV pantoprazole.

Check βHCG and give norethisterone 5 mg tds enterally to all girls >11 y.o.

If persisting significant bleeding: provided Fibrinogen >1g/L, consider rFVIIa (Nova-7) 80 ug/kg or prothrombinex 20 IU/kg

Consider endoscopy for large or ongoing upper GI bleed

CARDIOVASCULAR:

Vasodilatory shock ± SIRS occurs commonly

Obtain early **cardiac echo** to assess ventricular filling and systolic performance

Consider infusion of **noradrenaline** to maintain mean BP >70 (if > 4 y.o.); >60 (if 1-4 y.o.); >50 (if < 1 y.o)

Consider infusion of **adrenaline** if ventricular systolic performance is reduced

Consider **hydrocortisone** 1 mg/kg IV 6 hourly if needing high dose inotrope/vasopressors

Avoid vasopressin and terlipressin which increase cerebral blood volume and raise ICP

Maintain CVP 5-8 to maintain adequate cardiac output but avoid hypervolaemia

HYPOGLYCAEMIA: Aim for blood Glucose to 4-7 mmol/l.

If <4 mmol/l: bolus 5ml/kg 10% dextrose IV but avoid repeated boluses which cause rebound hypoglycaemia

Increase dextrose concentration in maintenance fluid (give via CVC if > 10% dextrose)

Consider infusion of 20% dextrose via CVC and calculate glucose intake in mg/kg/min.

RENAL FAILURE (due to SIRS or to toxin such as paracetamol):

Fluid bolus 10 ml/kg + frusemide 1mg /kg IV and assess response

Aim for urine output 1 ml/kg/hour

Consider haemofiltration (q.v.) if no response to frusemide

FLUID AND ELECTROLYTES

Check electrolytes including Na, K, Mg, PO₄ every 4 hours and correct abnormalities

Fluid overload occurs commonly in acute liver failure. Monitor fluid balance and CVP (aim for 5-8) and intervene early (frusemide ± haemofiltration)

CONTINUOUS VENO-VENOUS HAEMOFILTRATION (CVVH):

See RCH PICU RRT guideline

Start CVVH early (grade 2 encephalopathy or NH₃>100).

Use as large a catheter as possible and a large filter.

For NH₃ >150, use as high a blood flow rate and filtrate flow rate as possible

No heparin in the prime

Lactate-free replacement solution; regional anticoagulation (heparin/protamine); avoid ACD as a failing liver will not handle a citrate load.

INFECTION: mortality risk and a risk factor for cerebral oedema

Culture blood, urine, tracheal aspirate daily; WBC count, I/T ratio and procalcitonin

Empirical broad-spectrum antibiotics for patients from time of admission with encephalopathy grade ≥3, SIRS or renal failure
Consider antifungal therapy in patients with new signs of sepsis after 5 days in PICU.

SEDATION: use the minimum amount needed to suppress coughing and movement

Consider early use of muscle relaxant: use atracurium rather than vecuronium/pancuronium

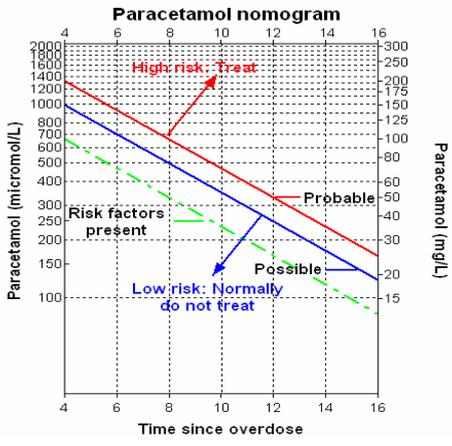
GIT:

Pantoprazole 1mg/kg 12 hourly IV

Consider **low protein feed** + pro-kinetic + neomycin + lactulose

PARACETAMOL overdose: A Lactate >3.5mmol/L at presentation or >3mmol/L after 12hrs of fluid resuscitation is associated with high mortality.

Give N-Acetylcysteine 150 mg/kg over 1st 15 min, then 50 mg/kg over next 4 hrs, then 100 mg/kg over next 16 hrs. Less likely to be effective if >10 hours post overdose.



LONG-STAY PICU PATIENTS

Chronically ill children now make up a high proportion of PICU bed days. Many long-stay patients have chronic neurological problems or chronic respiratory or cardiac needs. These include children with cerebral palsy, post op spinal surgery, neuromuscular disease, or other brain injury.

Children who stay in PICU longer than 28 days make up about 1% of all ICU patients, but nearly 20% of bed days. Just over a quarter of long-stay patients (LOS >28 days) have a favourable outcome (normal, functionally normal or mild disability), while nearly 50% die.

Patients with PICU LOS >7 days and with complex needs are supported by the ICU care manager to ensure consistency of communication and care. Each week there is a long-stay patient meeting.

Some children have a long-term ICU consultant, whose role is to maintain consistency of management, be the person who generally has discussions with the family, sub-specialty units and the duty ICU consultant about overall management, but not in day-to-day medical management.

Transition from PICU to ward

Chronic respiratory support

Children requiring long-term invasive ventilation may be appropriate for transition to the ward. The Sugar-glider (General Medical) and other wards will provide nocturnal BiPAP, +/- BiPAP with physiotherapy, or BiPAP when children are asleep during the day (e.g. some children with neuromuscular disease). However the child must first be established on BiPAP in ICU and stable. CPAP or BiPAP can be initiated on the ward in a child who has an acute deterioration, but the child will then be managed in the PICU until stable. BiPAP or CPAP can also be given in the context of palliative care on the wards. Currently Sugarglider is the only ward outside PICU where children requiring invasive ventilation or CPAP via tracheostomy are accepted.

All patients requiring invasive or non-invasive ventilation must be referred to the Respiratory Unit, who will then manage ventilation and weaning outside PICU.

There is a weekly ventilation meeting on Wednesday morning organized by the Respiratory Unit at which ward patients or those soon to be discharged from PICU requiring ventilation are discussed.

Children who are on home oxygen or non-invasive respiratory support who are having surgical procedures should generally have a bed booked in ICU post-op, as they may have worse respiratory function after surgery. For some procedures where post-op respiratory function is uncertain (such as spinal surgery, or after fundoplication where CPAP/ BiPAP may carry a risk) children with chronic respiratory requirements should return to ICU intubated, and weaning and extubation can be planned and occur after a proper assessment of analgesia and ventilation requirements.

BiPAP / VPAP

Children in PICU may be on BiPAP Vision (Philips), or their home BiPAP machine. Patients needing transition to the ward are put on the VPAP (ResMed).

Modes of operating

CPAP: only for children who do not require tidal volume augmentation and do not retain CO₂, e.g. obstructive sleep apnoea

Spontaneous mode: a child who needs augmentation of tidal volume, but who is able to trigger the device. The patient breathing in and out triggers transition between IPAP and EPAP

Spontaneous / Timed mode: a child who sometimes is unable to trigger the device, and is needing to rest the respiratory muscles

Timed: a child who is unable to trigger the device, the breath rate, IPAP Min and IPAP Max are set

IPAP Max time: the time set as the maximum time the flow generator will remain in IPAP before switching to EPAP. This should be no greater than 50% of the cycle length. IPAP Min

time: the minimum time the flow generator will remain in IPAP before switching to EPAP (if the patient's inspiration is too short, the IPAP will remain for the IPAP minimum time, before changing to EPAP. This should be no less than 0.3 seconds.

Ventilator parameters to be set:

EPAP pressure

IPAP pressure

Respiratory rate (only in timed or spontaneous/timed modes): usually set 4-6 breaths below the patient's relaxed rate.

Count the patient's respirations for 60 seconds with the child breathing quietly, calculate the respiratory cycle length (60 / RR) seconds

Setting inspiratory times:

Maximum IPAP time (IPAP Max) rise time = respiratory cycle length / 2

Minimum IPAP time (IPAP Min)

Weaning from BiPAP: generally allow children to have several short periods (30-60min) off BiPAP, then progressively lengthen time off (1-2 hours x 3 per day, 2-3 hours x 3 per day). Continue night-time BiPAP until off for the whole day when awake.

Consider tds BiPAP with physio during the day if the patient requires night-time BiPAP

Before discharge ensure patients have VPAP orders prescribed, Respiratory Unit and physiotherapy referrals have been made and the ICU technologists are aware of impending transfer

Other things to consider for long-term or chronic PICU patients:

- Weaning plan for sedation and analgesia
- All drugs orally where possible
- Activity, physiotherapy, wheelchair, communication tools, play
- Nutrition: regular weighing, head circumference, length, plot growth
- Providing optimal calories, see Nutrition section
- Consider risk of aspiration – feed upright
- Feed intolerance: if large gastric residual volumes, consider metaclopramide or erythromycin
- Constipation: consider lactulose, movicol
- Routine sleep times, consider melatonin or night time sedation
- Osteopenia: risk factors prematurity, corticosteroids, disuse, diuretics, chronic lung disease, TPN, organ transplantation. Measure alkaline phosphatase (>800 IU seen in osteopenia), and phosphorous (<1.8 mmol/L), Vitamin D levels (25-hydroxy vit D decreased; 1-25 hydroxy vitamin D may be increased), Prevention: adequate amounts and ratio (1.3-1.7:1) of Ca and P intake with an adequate caloric (> 80 Kcal/kg/d) and nutritional (2.5-3 g/kg/d amino acid and 400 IU/d vitamin D); consider ceasing frusemide or changing to anti-calciuric diuretic, such as chlorthiazide; limit use of corticosteroids; encourage physiotherapy and activity, consider bisphosphonates
- Rationalise tests: reduce frequency of x-rays, blood tests, have a weekly timetable
- Prevent anaemia: essential blood tests only, haematinics (iron and folic acid, darbepoietin if renal impairment)

- Immunizations, newborn screening

PALLIATIVE CARE

Palliative care is an important part of ICU care. This requires different things for different children and families, but the following are important:

Effective communication, children with chronic illness generally have many sub-specialty units caring for them. ICU Care Manager can coordinate meetings. Some of these children will have a long-term ICU consultant who can provide consistency in discussions with families. **A documented treatment plan** outlines the care that will be provided if there is deterioration.

RCH has a Palliative Care service.

Do not repeatedly ask families if their child is “for full resuscitation”, and do not tell families that living is “not in the child’s best interests”. This can be wearing and distressing for families. Discuss with the ICU and bed-card Consultant. The person who is best to talk to the family is usually someone senior who knows the child and the family well, whom they trust.

For more information about Palliative Care, see

http://www.rch.org.au/clinicalguide/guideline_index/Withholding_or_Withdrawal_of_LifeSustaining_Treatment/

MENINGITIS AND ENCEPHALITIS

A child <1yr old with meningitis or encephalitis should usually be admitted to ICU because of the potential for deterioration or acute complications; contact the ICU consultant if you think a child <1 yr does not need admission to ICU. Admit any child with coma, hypotension, sodium <130 mmol/L, multiple convulsions or a convulsion lasting longer than 15 minutes. See also Meningococcal Sepsis, and Sepsis - Severe.

Lumbar puncture is contraindicated if there is absent or non-purposeful response to squeezing the ear lobe hard (the child should push you away or seek a parent), or if the child has focal neurological signs. Do blood culture, FBE and I/T ratio, gas, U&E, glucose.

1. Antibiotics. Give cefotaxime 50 mg/kg/dose (max 3g) 6H IV. If <2 mo, also give penicillin and gentamicin.
2. Fluids. Avoid both hypovolaemia (and hypotension) and water overload (cerebral oedema). Correct hypotension with 10 ml/kg boluses of 0.9% saline, and consider inserting a femoral venous cannula to monitor CVP. Give maintenance fluid of Plasma-lyte 148 and 5% dextrose at 1 ml/kg/hr (less if there is hyponatraemia).
3. Monitor electrolytes and glucose 4 hourly for at least the first 24 hours. Hyponatraemia suggests water overload.
4. Usually give dexamethasone 0.15 mg/kg 6H IV, but give hydrocortisone 1 mg/kg 6H IV instead if there is septic shock. In addition, give a single dose of methylprednisolone 10 mg/kg or dexamethasone 2mg/kg if this can be given before the first dose of antibiotics.
5. Intubation and ventilation. Do this early for even mild impairment of consciousness, convulsions, focal neurological signs, acidosis or hypotension. Pre-oxygenate; use cricoid pressure; give ketamine 2 mg/kg, fentanyl 5 mcg/kg and vecuronium IV; wait 2 mins then intubate quickly.
6. Do a CT or MRI of the brain in any child who is intubated. If there is any clinical or radiological evidence of raised intracranial pressure, the ICU consultant may consult neurosurgery about an intraventricular catheter (and early cerebral decompression if the pressure is high).
7. Treat repeated or prolonged convulsions with intubation, and a long-acting anticonvulsant - levetiracetam or phenobarbitone or phenytoin (see Convulsions). If convulsions persist despite a long-acting anticonvulsant, give midazolam infusion increased until controlled (range 1 up to 10 mcg/kg/min in refractory cases) – see CONVULSIONS - STATUS.
8. The bedcard unit should report any notifiable disease.

ENCEPHALITIS

Causes: HSV, VZ (cerebellitis), EBV, mycoplasma, influenza (myositis, esp with influenza B measure CK), enterovirus (look for papular lesions on hands and feet, neurogenic pulmonary oedema), rotavirus, HHV6, parechovirus (neonates). Consider parainfectious immune mediated, autoimmune, metabolic, vascular, paraneoplastic, toxic (alcohol, paracetamol, salicylate, tricyclic, and heavy metals), and septic encephalopathy.

Decision to do LP is based on clinical features not only on a CT. If clinical contraindications to an LP (above) are present, an urgent CT or MRI should be done to identify brain oedema, tight basal cisterns, mass lesions, abscess, empyema. A normal CT does not exclude intracranial hypertension.

Investigations

Should be selective based on the clinical presentation, and staged according to treatment response and initial tests.

- CSF microscopy, Gram stain, bacterial culture, cell count and type, protein, glucose
- CSF PCR for HSV (1&2), enterovirus, parechovirus, mycoplasma, varicella-zoster virus
- CSF lactate (mitochondrial disorder, meningitis)
- CSF oligoclonal bands (non-specific indication of CSF inflammation)
- CSF Anti-NMDA receptor antibodies
- NPA for respiratory viruses, enterovirus PCR
- Throat and rectal swabs for enterovirus
- Faeces: PCR or antigen testing for enterovirus, adenovirus, rotavirus; enterovirus culture/typing
- Acute and convalescent serum if mycoplasma, EBV, flavivirus, lyme disease, cat scratch (Bartonella), rickettsiosis or erlichiosis suspected
- If immunocompromised, also consider HHV-6, JC, CMV, cryptococcal
- Vesicle fluid PCR and culture (HSV, VZV, enterovirus)
- Neonates: also HSV-2, parechovirus, CMV, toxoplasmosis, *T. pallidum* (syphilis), *L. monocytogenes*, HHV-6.
- MRI more sensitive than CT at detecting early cerebral changes of viral encephalitis

Treatment

Supportive, antibiotics until bacterial meningitis excluded
Acyclovir: 3 months – 12 years: 500mg/m² 8 hourly; >12 years 10mg/kg 8 hourly

Consider: erythromycin if mycoplasma suspected;
Immunotherapy (steroids, IVIg, plasma exchange for immune-mediated encephalopathy)

MENINGOCOCCAL SEPSIS

See also Sepsis, and Meningitis. Do not do lumbar puncture if there is a strong suspicion of meningococcal disease (typical rash in an ill child). Do a blood culture, blood smear on slide for gram stain, throat swab (in transport medium), 2ml blood in an EDTA tube (for meningococcal PCR), FBE with diff and I/T ratio, coags, blood group, gas, Na, K, Cl, Ca, phosphate, creatinine, glucose and chest X-ray.

1. Children with meningococcal disease often need very large amounts of fluid initially (and antibiotics may make the hypovolaemia worse). Give 10-20 ml/kg boluses of saline; 60-100 ml/kg is often needed. For septic shock, see Sepsis - Severe.
2. If it can be given before the first dose of antibiotic, give methylprednisolone 10mg/kg.
3. Give hydrocortisone 1 mg/kg 8H IV.
4. Intubate early (eg more than 40 ml/kg fluid, BE <-10, progressive skin lesions); do not wait until very ill. Pre-oxygenate, use cricoid pressure, give ketamine 1-2mg/kg and fentanyl 2-3mcg/kg slowly IV if conscious and circulation stable, vecuronium 0.1mg/kg, wait 2 minutes, and intubate quickly.
5. Give cefotaxime 50 mg/kg/dose (max 3g) 6H IV.
6. Give immunoglobulin 0.5 g/kg IV over 2 hours.
7. If oliguria >12-24 hours, consider haemofiltration.
8. If there is purpura fulminans, give FFP 1 bag per 15 kg (about 15 ml/kg) every 8 hours, and heparin 20 u/kg stat then 10 u/kg/hr (unless the child is heparinised for filtration)
9. Because large volumes of FFP are often needed, give 50% dextrose infusion if required to maintain blood glucose at 4-6 mmol/L. If glucose >10 mmol/L, add insulin 0.05 u/kg/hr.
10. Do urgent fasciotomy if compartment pressure >40 mmHg (or > DBP - 30 mmHg) in adult-sized.
11. Isolate the child for 12hr after starting antibiotics. The risk to staff is very low after just a few hours of antibiotic treatment.
12. ICU staff rarely need to take prophylactic antibiotics. They are only needed if staff have had unprotected exposure to respiratory droplets (generated from coughing or during airway management) within 24hr (and perhaps less) of the patient starting antibiotic treatment.
13. The bedcard unit should report to the Health Department, and arrange prophylaxis for contacts.

METABOLIC – HYPERAMMONAEMIA

Contact the metabolic consultant on call to discuss the management of these patients. Metabolic patients have individualised management plans and different thresholds for various treatment modalities.

Below is a general guideline for treatment options at different ammonia thresholds.

Enteral feeds should not be interrupted for even a short time without providing adequate intravenous energy

Ammonia \leq 250 $\mu\text{mol/l}$

Inform intensive care and metabolic consultants

Enteral feeds (energivit 20%) preferred

If enteral feeds are not tolerated or even temporarily interrupted give iv glucose (10%, 12%, 15%) and intralipids (20%) to prevent catabolism.

Continue medications (intravenous or enteral) after discussion with metabolic team. These may include: sodium benzoate, arginine, biotin, carnitine, hydroxycobalamin.

Monitor for changes in conscious level; consider ICP monitoring.

Monitor ammonia level hourly until downward trend is observed.

Start haemofiltration if ammonia level is rising at a rate of $>50 \mu\text{mol/l}$ per hour or fails to fall / stabilize within 3 hours

Note that in states other than inborn errors of metabolism, such as liver failure, a serum ammonia >100 is neurotoxic and an indication for haemofiltration.

Ammonia 250 – 400 $\mu\text{mol/l}$:

Inform intensive care and metabolic consultants

Enteral feeds (energivit 20%) preferred

If enteral feeds are not tolerated or even temporarily interrupted give iv glucose (10%, 12%, 15%) and intralipids (20%) to prevent catabolism.

Specific metabolic medications (such as listed above) to be given intravenously

Prepare haemofiltration but do not start immediately

Monitor for changes in conscious level; consider ICP monitoring.

Monitor ammonia level hourly.

Start haemofiltration if ammonia level is rising at a rate of $>50 \mu\text{mol/l}$ per hour or fails to fall / stabilize within 3 hours

Ammonia \geq 400 $\mu\text{mol/l}$:

Inform intensive care and metabolic consultants

Enteral feeds (energivit 20%) preferred

If enteral feeds are not tolerated or even temporarily interrupted give IV glucose (10%, 12%, 15%) and intralipids (20%) to prevent catabolism.

Start haemofiltration ASAP

Specific metabolic medications (such as listed above) to be given intravenously

Ammonia $\geq 1000 \mu\text{mol/l}$

Inform intensive care and metabolic consultant

Evaluate whether to start curative treatment or palliative care

Consider the following and discuss with Metabolic Team

1. ICP monitoring

2. Lower threshold for haemofiltration in patients with **organic acidaemia** or with hyperammonaemia of unknown cause

Reference: Haberle et al. Suggested guidelines for the diagnosis and management of urea cycle disorders. Orphanet Journal of Rare Diseases 2012; 7:32 <http://www.ojrd.com/content/7/1/32>

MET: MEDICAL EMERGENCY TEAM

The aims of the MET service are to prevent unexpected cardio-respiratory arrest and to reduce unplanned admissions to ICU. The MET is composed of an ICU doctor, ICU nurse, The ICU doctor is the team leader.

Responsibilities of MET members.

Attend immediately (as for cardio-respiratory arrest). ICU nurse will take MET Trolley. Equipment is on wards.

Most calls are not arrests. Introduce yourself as the ICU doctor, find out who the other people are in the room, listen carefully to what ward staff say about the patient and the reasons for the call.

Politely ask people who are not assisting to leave.

Ensure the parents are informed about what is done.

Liaise with ICU ANUM if patient requires ICU admission.

Call the ICU consultant if assistance with disposition is required or extra medical personnel are required.

For patients remaining on ward – ensure ward nurse and unit doctor have an agreed follow up plan.

Assign MET modifications as required.

Complete the MET navigator in the Electronic Medical Record.

Please do not criticise or embarrass the staff who called MET – be supportive and take the opportunity to educate. Enquire why the MET was called for the purpose of resolving the issue. Encourage and commend staff for calling a MET

Multiple METs can occur concurrently. Call the PICU fellow or consultant to arrange attendance to all METs

See **Escalation of Care** procedure on the RCH intranet

PICU MET will attend all calls to:

North Building - Level 1 and above, including Kelpie and Banksia (level 1), Sugar Glider and Kookaburra (level 2), Rosella and Koala (level 3), Platypus and Cockatoo (level 4), Butterfly (level 5), the clinical & service (silver) lifts and the helipad.

East Building - Level 3 and above, including the Operating Theatres, Recovery Rooms, MRI Suite, Possum (level 3) and Laboratory Services (fourth floor).

Standard MET call criteria

VICTOR chart – vital signs in the purple zone

OR

Any one or more of:

1. Parent, nurse or doctor worried about clinical state.
2. Airway threat.
3. Hypoxaemia: SpO₂ <90% in any FiO₂, or cyanotic heart disease and SpO₂ <60% in any FiO₂.
4. Severe respiratory distress, apnoea or cyanosis.
5. Tachypnoea. Respiratory rate >60 (<4mo), >50 (4-12mo), >40 (1-4yr), >30 (5yr or more).
6. Tachycardia or bradycardia. Pulse outside range 100-180 (<12mo), 90-160 (1-4yr), 80-140 (5-12yr), 60-130 (>12yr).
7. Hypotension. Systolic BP <50 (<4mo), <60 (4-12mo), <70 (1-4yr), <80 (5-12yr), <90 (>12yr).
8. Acute change in neurological status or convulsion.
9. Cardiac or respiratory arrest.

Some of the values for respiratory rate, heart rate and blood pressure are outside the normal ranges for age: they represent values that may indicate serious illness, and that require expert review.

It is also important to look for worsening trends in vital signs and report these.

If a child fulfils any of these criteria, the treating medical team and the MET service are notified (via switch on 777).

MET modifications

MET criteria can only be modified under the following circumstances:

1. In a stable patient, where there is a clear underlying clinical reason for one variable existing outside the normal range.
2. Any criteria can be modified by no more than 20%.
3. Do not modify more than one criteria (this effectively puts a child outside the protection afforded by the MET service).
4. By a doctor of registrar level or above.
5. For a defined period of time (not more than 24 hours).
6. Inform the bedcard consultant of any modification
7. Inform the PICU outreach registrar

MYOCARDITIS

1. Initial investigations and treatment as for cardiomyopathy (qv).
2. Consider early administration of immunoglobulin 2g/kg (35 ml/kg of 16% solution) IV over 16 hours.
2. The use of immunosuppressive drugs is controversial. Therapies include prednisolone, prednisolone plus azathioprine, or cyclosporin.

If the patient dies of suspected myocarditis or cardiomyopathy

A full PM is desirable, but it can be limited to biopsies. Tissue should be collected rapidly. Inform pathology. The autopsy (tissue) kit is in the core lab. Dry ice is on the 10th floor (call Security if out-of-hours).

Cardiac + skeletal muscle: 1 piece of each in glutaraldehyde for EM (store at 4°C); 1 piece of each in formalin or paraffin for histochemistry + microscopy; 2 x 0.5cm cubes of each (wrap in foil, place in screw cap tube, cover in dry ice, store at -70°C).

Liver: 1 piece in glutaraldehyde for EM (store in 4°C fridge); 2 cores or 2 cubes (wrap in foil, place in screw cap tube, cover in dry ice, store at -70°C).

Skin. 1 piece full-thickness (2-3mm surface diameter) in tissue culture or viral medium. Store at 4°C.

Blood. 10ml in heparin tube for DNA tests. Store at room temperature if it will be received <24hr, otherwise freeze.

NEEDLE AND CATHETER SIZES

Needle and catheter sizes and gauges

Ext dia mm	Needles SWG	Catheters FG,FR,CH	Ext dia mm	Needles SWG	Catheters FG,FR,CH
0.32	30	1	3.0	-	9
0.43	27	-	3.3	-	10
0.51	25	-	4.0	8	12
0.61	23	-	4.7	6	14
0.67	-	2	5.3	-	16
0.81	21	-	6.0	4	18
0.91	20	-	6.7	-	20
1.0	-	3	7.0	2	-
1.3	18	4	7.3	-	22
1.6	16	5	8.0	1/0	24
2.0	14	6	8.7	2/0	26
2.3	-	7	9.3	3/0	28
2.7	12	8	10.0	-	30

SWG = Standard Wire Gauge = British Imperial Gauge
 = 20 - 20 (log of external diameter) approx
 FG = FR = French = Charriere (CH) = 3 x ext dia mm

NON-ACCIDENTAL INJURY

Clinical findings that suggest non-accidental injury are:

- injuries that do not fit with the history
- bruising or injuries at unusual sites
- multiple fractures
- unexplained oral bleeding
- burns or scalds (especially small burns at unusual sites)
- injuries in a child with failure to thrive
- subdural haematomas, especially with retinal haemorrhages

1. Discuss any suspicion of non-accidental injury with the ICU consultant. The ICU and bedcard consultants will decide whether to refer the child to the on-call social worker and the Victorian Forensic Paediatric Medical Service (VFPMS). **In Australia reporting of suspected child abuse is mandated by law.**
2. The bedcard unit registrar is responsible for formally admitting the child, and a VFPMS paediatric consultant should see the child early and document injuries. However, ICU staff should carefully record any statements made to them by the family about the cause of the injuries.
3. Carefully examine the child all over, and record your findings in the notes in words and diagrams.
4. Investigations will usually include clotting studies (PT, PTT, fibrinogen, platelets), a skeletal survey (and often a bone scan when the child is well enough), ophthalmology (to look for fundal haemorrhages – dilate the pupils with short-acting **tropicamide**, and **not** with long-acting cyclopentolate). Contact VFPMS and the RCH photographer for clinical photographs of any skin lesions or external physical injuries.
5. Remember that unlike accidental trauma, with a non-accidental injury you can never be sure of when it occurred, or the force that was applied, or whether there were multiple injuries over time, or whether the child suffered secondary injury (hypoxic, hypotensive) following the abuse. Therefore, the natural history is much less certain than with injuries where these things are known, and children with NAI may deteriorate unexpectedly.

NORMAL VALUES

Blood and sweat

Acid phosphatase (prostatic): 0-0.8 IU/l
ACTH (0800hr): <20 pmol/l
Activated partial thromboplastin time (APTT): 22-39 sec
Alanine aminotransferase (ALT, SGPT): 0-35 IU/l.
Albumin (>1y): 33-47 g/l (x0.1 = g/100ml)
Alkaline phos (0-2y): 100-350IU (x0.041=KA/100ml)
Ammonia: <50 umol/l (x1.703 = ug/100ml)
Amylase: 8-85 IU (x0.546 = SU/100ml)
Antithrombin III: 0.8-1.2 U/ml
Aspartate transaminase (AST) (1-3y): 15-60 IU
Base excess: -4 to +3 mmol/l (x1 = mEq/l)
Bicarbonate: 18-25 mmol/l (x1 = mEq/l)
Bilirubin (>1m): <10 umol/l (x0.0585 = mg/100ml)
Caeruloplasmin: 200-430 mg/l (x0.001143 = ODU)
Calcium ionized: 1.2-1.3 mmol/l (x4.008 = mg/100ml)
Calcium total: 2.0-2.7 mmol/l (x4.008 = mg/100ml)
Carboxyhaemoglobin: <5% total
Chloride: 98-110 mmol/l (x1 = mEq/l)
Chloride, sweat (<12y): <50 mmol/l (x1 = mEq/l)
Cholesterol (>1y): 3.1-5.4 mmol/l (x38.66 = mg/100ml)
Cholinesterase: 600-1500 IU
Copper (1-9y): 14-28 umol/l (x6.354 = ug/100ml)
Coprotophyrin: <0.3 umol/l (x654 = ug/l)
Cortisol: 150-600 nmol/l
Creatine kinase (CK, CPK): 40-240 IU
Creatinine (1-9y): 0.01-0.06 mmol/l (x11.3 = mg/100ml)
Cross-linked degradation products: <0.25 mg/l
Cyanide: <8umol/ml, >40 fatal (x0.026 = mcg/ml)
Ferritin: 18-300 mcg/l (x1 = ng/ml)
Fibrinogen: 1.9-5.0 g/l (split products <10 mg/l)
Free fatty acids: 0.1-0.6 mmol/l
Free thyroxine index (>4m): 60-155%
Globulins (>3y): 17-38 g/l (x0.1 = g/100ml)
Glucose (>1m): 3.6-5.4 mmol/l (x18.02 = mg/100ml)
Gamma-glutamyltransferase (GGT) (>3m): <40 IU
Immature/total neutrophil ratio: <0.15 (neonate <0.2)
Iron: 9-27 umol/l (x5.585 = ug/100ml)
Iron binding capacity: 45-72 umol/l (x5.585 = ug/100ml)
Lactate (venous): 1.0-1.8 mmol/l (x8.904 = mg/100ml)
Lactate dehydrogenase (LD): 210-420 IU
Lead: 0.2-1.2 umol/l (x20.72 = ug/100ml)
Lipase: 0-2.66 ukat/l (x60 = IU/l)
Magnesium: 0.7-1.0 mmol/l (x2.432 = mg/100ml)
Methaemoglobin: <2% total

Osmolality: 270-295 mmol/kg (x1 = mOsm/kg H₂O)
pCO₂: 32-45 (x0.1317 = kPa)
pH (>1m): 7.34-7.43
Phosphate (>2y): 1.1-1.8 mmol/l (x3.098 = mg/100ml)
pO₂ (>2w): 80-100 mmHg (x0.1317 = kPa)
Potassium (>1y): 3.5-5.0 mmol/l (x1 = mEq/l)
Protein (>3y): 57-80 g/l (x0.1 = g/100ml)
Protein C: 0.7-1.4 U/ml.
Protein S (free): 0.55-1.40 U/ml
Prothrombin time (PT): 9-14 sec, INR 0.8-1.3
Protoporphyrin: 0.3-1.0 umol/l (x56.2 = ug/100ml)
Pyruvate: <0.1 mmol/l (x8.702 = mg/100ml)
Renin activity: 1-4 ng/ml/h
Sodium: 135-145 mmol/l (x1 = mEq/l)
Thyroid bind glob (adult): 12-28mg/l (x0.078=ugT4/100ml)
Thyroid stimulating hormone (TSH) (>14d): <5 nmol/l
Thyroxine free (adult): 9-26 pmol/l
Thyroxine total (>1y): 70-155 nmol/l (x0.078 = ug/100ml)
Triglycerides: 0.9-2.0 mmol/l (x88 = mg/100ml)
Triiodothyronine (T3) (adult): 1.0-2.7 nmol/l
Triiodothyronine (T3) uptake (0-adult): 70-115%
Troponin I (cardiac troponin): 0-0.29 mcg/l
Urea (>4y): 2.1-6.5 mmol/l (x6.006 = mg/100ml)
Uric acid (<12y): 0.13-0.4 mmol/l (x16.81 = mg/100ml)
Zinc: 11-22 umol/l (x6.538 = ug/100ml)

Urine

Adrenaline (1-6y): <0.05 umol/d
Delta ALA: <40 umol/l (x0.13 = mg/l)
Amylase: 50-500 IU (x0.546 = SU/100ml)
Calcium: <0.12 mmol/kg/d (x40.08 = mg/kg/d)
Calcium/creatinine ratio: <0.7 mmol/mmol
Copper: <0.3 umol/d (x63.5 = ug/d)
Coprotoporphyrin: <0.3 umol/l (x654=ug/l)
Creatinine clearance: 1.4-2.4 ml/s/1.73m²
Dopamine (1-6y): 0.11-1.16 umol/d
Dopamine (1-6y): <1.1 umol/mmol creat
5HIAA: <0.05 mmol/d (x0.19=mg/d)
Homovanillic acid (HVA) (1-6y): 3-16 umol/mmol creat
MHMA, VMA or HMMA (1-6y): <12 umol/d
Oxalate: <0.6 mmol/d (x90 = mg/d)
Phosphate index (<12y): -0.2 to +0.04
Porphobilinogen: <9 umol/l (x0.23 = mg/l)
Protein: <4 mg/h/m²
Uroporphyrin: <0.05 umol/l (x830 = ug/l)

Cerebrospinal fluid

Glucose: >2.5 mmol/L (>=60% blood level)

Protein: 0.5-4 (prem), 0.4-1.7 (<1wk), 0.05-0.4 g/L (>2mo)
Lymphocytes ($\times 10^6$): <20 (<1wk), <6 (>1wk)
Neutrophils ($\times 10^6$) <10 (<1wk), 0 (>1wk)

NUTRITION

Enteral Nutrition

Feed children in ICU enterally as soon as possible. Feed using 3 hourly boluses initially. Start with half the calculated feed volume and increase over the next 6 hours.

Approximate daily energy requirements in PICU:

Age	KJ/Kg/Day
1-2 mth	353 – 441
3 mths	320 – 400
4-6 mths	281 – 348
7-9 mths	266 – 332
10-12 mths	271 – 338
13-24 mths	275 – 345
2 yrs	278 – 346
3-6 yrs	193 – 234
7-10 yrs	152 – 181
11-14 yrs	122 – 141
15-18 yrs	109 – 118

Consider increasing energy provided in non-ventilated, burns, complex cardiac patient, chronic sepsis, prolonged ICU stay.

Suggested rates for grading up continuous feeds

Age	Initiation	Advance	Tolerance
Prem	1-2ml/kg/hr	10-20ml/kg/d	120-200ml/kg/d
<1 yr	5-10ml/hr	5ml 4hrly	100-150ml/kg/d
1-6 yr	10-20ml/hr	10ml 4hrly	75-100ml/kg/d
>7 yr	20-25ml/hr	10-20ml 2-8hrly	35-75ml/kg/d

Standard feed choices

Weight	Standard energy concentration	Increased energy concentration
<8kg	Breast milk (280kJ/100ml) Standard formula (280kJ/100ml)	Arrange with dietician
8-20kg	Nutrini (420kJ/100ml)	Nutrini Energy (630kJ/100ml)
>20kg	Nutrison Standard (420kJ/100ml)	Nutrison Energy (630kJ/100ml)

If NG bolus feeds poorly tolerated: reduce narcotics, give erythromycin 3mg/kg 8H. Or insert a nasojejunal tube for continuous post-pyloric feeding.

Do not give ranitidine routinely to ventilated patients. If enteral feeds cannot be given, give ranitidine to high risk patients (burns, trauma, shock, sepsis, renal failure, spinal cord lesion,

corticosteroids, coagulopathy). Give omeprazole if GI hge occurs.

Parenteral Nutrition

In a well nourished child give parenteral nutrition if enteral nutrition is not possible for:

- > 2 days in a neonate
- > 3 days in infants
- > 5 days in an older child

Earlier PN carries higher risk of infection.

Consider PN earlier in malnutrition or weight loss >10% and it is not possible to feed enterally.

Lipid solutions:

ClinOleic if anticipated use <2 weeks

SMOF if long term use anticipated

Start lipid at 0.5-1g/kg/day (older children may need 1-2g/kg/day)

Contraindicated: fungal infection, thrombocytopenia, hypertriglyceridemia

Monitoring

Baseline: U&E, Ca, Mg, PO₄, BSL, TG, VBG, LFT, FBC

Daily while grading up: U&E, Ca, Mg, PO₄, BSL, TG

Weekly: U&E, Ca, Mg, PO₄, BSL, TG, VBG, LFT, urinary Na, K and CL

Risk of refeeding syndrome: 6hrly bloods

Interruptions to PN

If parenteral nutrition has to be suddenly discontinued, infuse at least half the amount of glucose (in mg/kg/min) to prevent hypoglycaemia (especially in infants).

NO child should ever go to theatre on peripheral PN. The risk of unrecognized extravasation is very high. Change to equivalent dextrose-electrolyte solution (mg/kg/min glucose) and notify anaesthetist. No child should go to theatre on central PN, except for short procedures and only if the anaesthetist is aware.

Refeeding Syndrome

At risk after >7 days fasting, malnourished, recent weight loss (>10% body weight), baseline low K, PO₄, Mg

Manifests as low PO₄, K, Mg and thiamine deficiency.

Observe for arrhythmias, weakness, sepsis.

Monitor electrolytes 6hrly initially and replace, give thiamine, reduce energy intake if PO₄ <0.6mmol/L. Slowly grade up over 4 days.

Nutritional content of PN formulas

Infants <5kg (Primene amino acid solution)

Each litre provides	N1: 25/100	N2: 30/125	N3: 50/200
Amino acids (g/L)	25	30	50
Glucose (g/L)	100	125	200
Sodium (mmol/L)	0 or 20	30	Nil
Potassium (g/L)	0 or 20	30	Nil
Calcium (mmol/L)	8	8	8
Phosphate (mmol/L)	10	10	10
Magnesium (mmol/L)	2	2	2

Also multivitamins and trace elements.

Children >5kg (Synthamin amino acid solution)

Each litre provides	P1: 20/100	P2: 25/150	P3: 30/200
Amino acids (g/L)	20	25	30
Glucose (g/L)	100	150	200
Sodium (mmol/L)	30	30	30
Potassium (g/L)	30	30	30
Calcium (mmol/L)	6	6	6
Phosphate (mmol/L)	6	6	6
Magnesium (mmol/L)	4	4	4

Also multivitamins and trace elements.

Nutrient and lipid flow rates

Nutrient ml/hr for 3 of 4 hours = ml/kg/day x Wt / 18

Nutrient ml/hr constantly = ml/kg/day x Wt / 24

Lipid 20% ml/hr of 1 of 4 hours = g/kg/day x Wt x 0.84

Lipid 20% ml/hr constantly = g/kg/day x Wt x 0.21

ONCOLOGY EMERGENCIES

Acute leukaemia and hyperleucocytosis

Very high white cell count (AML more than 100,000 and ALL more than 300,000). Leucostasis is more common in AML at lower white cell counts as blast are larger and coagulopathy more severe.

All treatment decisions in consultation with oncologist.

Admit directly to ICU, such children should not be managed on the ward until they deteriorate, it is common that they will.

Baseline CT head scan on admission if any neurological signs.

Intubate early if the child has headache, is irritable, has oedema or bleeding on CT scan, don't wait for neurological deterioration.

Meticulous attention to correcting coagulation and thrombocytopenia. For all leukaemia with high white cell counts maintain platelets counts over 50,000. If any bleeding on CT scan correct coagulopathy and give Nova 7, keep platelets above 100,000.

Give rasburicase early (0.2mg/kg DAILY for 5 days). Do not prescribe it as a PRN order, as second and subsequent doses may not be given (duration can always be reviewed).

Use mannitol with pre-chemotherapy hyper-hydration. 500 ml Plasma-lyte 148 and mannitol 20g (100 ml of 20%). Run at 1.5 L per m² per 24 hours.

If the child is referred from a regional centre, transfer as soon as possible. If the chemotherapy is going to be delayed by transfer, consider Hydroxyurea for AML or corticosteroids (methylprednisolone) for ALL until transfer and initiation of chemotherapy.

Induction chemotherapy should be as intense as possible to reduce the WCC as rapidly as possible (Oncology will consider this on an individual basis)

Monitor for tumour lysis syndrome (see below).

For large mass disease lymphoma or high risk of tumour lysis syndrome

Give rasburicase early (0.2mg/kg DAILY for 5 days). Do not prescribe it as a PRN order, as second and subsequent doses may not be given (duration can always be reviewed)

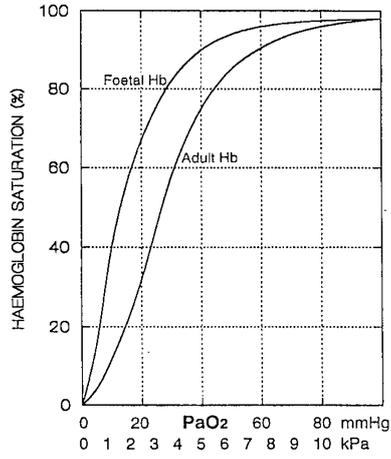
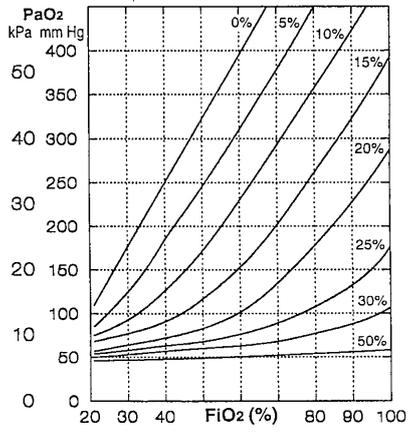
Use mannitol with pre-chemotherapy hyper-hydration (as above). Alkalinisation should be avoided when phosphate is elevated, as this can cause calcium phosphate to precipitate. With rasburicase the problem of hyperuricemia is rare.

Monitor potassium, uric acid, calcium, phosphate, creatinine, and urea. Do not give calcium unless symptomatic (risk of calcium phosphate deposition).

Treat hyperkalaemia according to guideline (see Cardiac – post-op and hyperkalaemia)

Consider haemofiltration if electrolyte abnormalities, progressive fluid overload or oliguria refractory to treatment.

OXYGEN GRAPHS



OXYGEN THERAPY

- In bronchiolitis administer oxygen when SpO₂ <90%.
- For critically ill children with multiple organ dysfunction or potential deficits in oxygen delivery, administer oxygen to maintain SpO₂ >94%.
- Wean oxygen therapy if SpO₂ >97%.
- Humidify oxygen if flow rates >2 litres/min in infant, or >4 litres/min in an older child.
- Always humidify oxygen therapy given through a tracheostomy, nasopharyngeal airway or endotracheal tube.
- If giving oxygen via face mask have flow rates >5 L/min to avoid re-breathing CO₂.
- Avoid hyperoxaemia, which can exacerbate ARDS – aim for FiO₂<0.6 in an intubated patient (use PEEP and other strategies to improve SpO₂ in order to reduce FiO₂<0.6).
- Before giving oxygen to children with cyanotic congenital heart disease, find out what their usual SpO₂ is when they are well. Beware of giving oxygen to infants with balanced circulations (univentricular hearts) or large VSDs (which can increase shunt and worsen pulmonary congestion. Aim for SpO₂ within their usual range (in balanced circulation usually 75-85%); for VSD with lung disease (such as bronchiolitis) SpO₂ 88-92% is adequate.
- In very pre-term infants (<32 weeks GA) maintain SpO₂>88% but not greater than 95%.
- Use the Respiratory Distress Score (below) to monitor patients on oxygen therapy: If RDS>8 consider the need for higher level of support (e.g. High flow or CPAP), refer to ICU registrar, fellow or consultant.

OXYGEN – HIGH FLOW NASAL CANNULA

Humidified high flow nasal cannula oxygen therapy (HHFNC) used for the same indications as the traditional method of CPAP using a nasopharyngeal tube:

- Severe respiratory distress from bronchiolitis, pneumonia, asthma, congestive heart failure
- hypoxaemia (SpO₂ <90%) despite standard oxygen nasal cannula therapy (2 L/min <1 yr; 4 L/min older children)
- respiratory support post extubation and mechanical ventilation
- weaning therapy from mask CPAP or BIPAP
- respiratory support to children with neuromuscular disease
- apnoea of prematurity

Equipment

- Oxygen and air source, blender
- Flow meter
 - <10Kg use standard 0-15L/min flow meter
 - >10Kg use flow meter which delivers up to 100L/min flow (available from clinical technologists)
- Humidifier (Fisher and Paykel® MR850)
- Circuit tubing to attach to humidifier
 - Children <10kg: small volume circuit tubing (RT 329)
 - Children >10kg: adult oxygen therapy circuit tubing (RT203)
- Nasal cannula (prongs) to attach to humidifier circuit tubing
 - Newborn, full term: Infant cannula BC2745 or BC 2755 (size to fit nares comfortably)
 - Infants and children up to 10kg: Paediatric cannula BC3780
 - Children >10kg: Adult cannula OPT542 (available from clinical technologists)

Secure nasal cannula on patient using Comfeel™ and tape, ensuring the prongs sit well into the nares.

Start the high flow nasal cannula system at the following settings:

Flow rates:

2 L/kg/min for the first 10kg: i.e. 6 kg child = 12 L/min;
8kg=16L/min; 10kg child = 20 L/min

0.5 L/kg/min for each kg above that (max 50 L/min): 16kg child will receive 20+3 = 23 L/min; 30kg = 20+10 = 30 L/min; 40kg = 20+15=35 L/min; 60kg = 20+25=45 L/min

Flow 2 L/kg/min in infant gives 4-8 cmH₂O PEEP.

For <10kg start at target flow, for >10kg start at 6L/min and increase up to target flow rate over a few minutes to allow patient to adjust to high flow

FiO₂: **Always use a blender**, never use flow meter off wall delivering FiO₂ 100%. Start at 50-60% for bronchiolitis and respiratory distress

Lower FiO₂ (e.g. 21% – 25%) may be needed for cyanotic congenital heart disease with balanced circulation

Target range for SpO₂ of 93%-98%, use lowest FiO₂ to achieve this

75-85% in cyanotic congenital heart disease with balanced circulation

Humidification: Because flows used are high, heated water humidification is needed. Set humidifier on 37° C invasive setting
Most children have an NG tube

Monitor HR, RR, SpO₂, work of breathing (use **Respiratory Distress Score**, below). If within 2 hours SpO₂>90%, HR and RR↓ by 20% or both to normal range for age escalation of respiratory support – e.g. to CPAP or intubation is unlikely to be needed.

If RDS>8 consider the need for higher level of support, refer to ICU fellow or consultant

Use the respiratory distress score for monitoring patients on HFNC and Oxygen therapy

RESPIRATORY DISTRESS SCORE						
	Hypoxaemia	Chest wall retraction	Respiratory sounds (Audible)	Respiratory sounds (Auscultated)	Respiratory rate / min **	Heart rate / min **
Mild =1	Mild hypoxaemia SpO ₂ 90–93%	None or minimal	None or minimal external sounds	Good air entry on auscultation, some wheeze	<40	<140
Moderate =2	Moderate hypoxaemia SpO ₂ 85–90%	Moderate chest wall retraction	Intermittent grunting and/or nasal flaring	Moderately reduced air entry. Wheeze and some crepitation on auscultation	40-60	140-170
Severe =3	Severe hypoxaemia SpO ₂ <85%	Marked chest wall retractions, tracheal tug	Grunting with every breath, or audible wheeze, nasal flaring	Widespread crepitations. Poor air entry on auscultation	>60	>170
Score / 18						

Note that on high flow if high FiO₂ is used, oxygen saturation may be maintained in an infant despite the development of hypercarbic respiratory failure.

FiO₂ adjustment and weaning

Wean FiO₂ but do not wean flow rate until need for PEEP support resolved

For infants <10Kg

- First step is to wean the FiO₂ to <40% (usually within the first 1-2 hours)
- Once respiratory distress improved, change to **standard low flow 100% oxygen** (1 to 2 L/min) or cease oxygen therapy

For children >10Kg

- Wean FiO₂ to 40% (usually within the first 1-2 hours)

- Once the indication for using high flow has resolved, and the patient is stable in 40% oxygen, change to standard flow 1-2 L/min (up to 4L/min for older children) with FIO₂ of 100% via standard nasal prong therapy, or mask oxygen if preferred.
- There is no need for a prolonged weaning process, either be on humidified high flow, or standard low flow.

PERTUSSIS

Pertussis (whooping cough) usually presents as episodes of severe, prolonged coughing, but may present as apnoea in young infants. A whoop (loud stridor on inspiration after a paroxysm) is rare in children <6 mo old. Patients often appear well between paroxysms; persistent respiratory distress suggests that there is pneumonia. Mortality is highest in very young infants, and with a white cell count >100,000 (especially neutrophils >50,000). Diagnosis is by immunofluorescence or PCR of nasopharyngeal aspirate.

Severe illness may be associated with circulatory failure (from pulmonary hypertension), respiratory failure (often pneumonia), and encephalopathy with coma or convulsions (probably from hypoxic-ischaemic injury).

1. Give clarithromycin 7.5 mg/kg/dose 12H for 7 days oral or NG. If parenteral treatment is required, give azithromycin 15 mg/kg (max 500 mg) IV day 1, then 5 mg/kg (max 200 mg) daily for 7 days. Treat contacts if they are not fully immunised and either <12 mo old, or chronic cardio-respiratory illness, or in last month of pregnancy.
2. Oxygen therapy is often indicated even though the child is well oxygenated between paroxysms; pre-oxygenation reduces cyanosis during paroxysms of coughing.
3. There are copious thick secretions, and effective suctioning is very important (particularly after intubation).
4. In severe disease, consider giving methylprednisolone.
5. Humidified high flow or Nasal CPAP may be helpful for apnoea (but may stimulate coughing paroxysms).
6. Pulmonary hypertension may cause severe circulatory or respiratory failure; it may be caused by leukocyte aggregates in pulmonary vessels. If there is leukocytosis (especially neutrophilia) with pulmonary hypertension, consider urgent exchange transfusion or apheresis to remove white cells. For pulmonary hypertension, consider nitric oxide and sildenafil.
7. In very severe disease, ECMO should be considered providing there is not severe hypoxic-ischaemic injury. However, mortality rates are high, especially in infants <6 weeks old.
8. Hypotension may respond to vasopressin 1 u/kg in 50ml given at 0.5-2 ml/hour.

PETS

The Victorian Paediatric Emergency Transport Service (PETS) offers retrieval of critically ill children in Victoria, southern NSW and Tasmania, and advice about their management. The service is amalgamated with PIPER, to be the Paediatric Infant Perinatal Emergency Retrieval service.

Managing PIPER Referral Calls

PIPER calls should be taken by the PETS consultant when available, or the most senior registrar or fellow when not available. All PIPER calls are answered and scribed by a Clinical Coordinator in PIPER. You will receive a page directing you to call into a conference room. If you take the PIPER call, you should state your name and role (eg ICU registrar). Use the 'PETS Activity Activity Notepad' as a guide. Obtain essential details (name, age, hospital, referring doctor's name and contact telephone number), provisional diagnosis, current condition, treatment and response to treatment... At the finish of the phone conversation, ask the referring doctor to speak directly with the receiving bedcard registrar (eg general paed, or general surgery) if they have not already done so.

If the PETS consultant is not immediately available: listen to the referring doctor's concerns; offer advice on management; advise on preparations for transfer (see below); arrange to transfer the child if required; ring back with your ETA at the referring hospital (for very unstable children, ring back yourself in 15-30 minutes, or get another experienced ICU doctor to do so). Always be polite, diplomatic and never condescending. Double-check the name of the town and the name of the hospital.

The most appropriate doctor should perform retrievals. For example, a child with a difficult airway should always be retrieved by a senior ICU registrar or a consultant. Only the PETS consultant may decide that we are not able to perform a requested transport. The consultant should discuss the best method of transferring the child with the referring doctor.

Prioritisation. There are certain conditions for which the PETS team should be mobilized immediately, these are referred to as the Go Now criteria. The team deployed may need to be a second team if the PETS team are already out. The criteria are:

PETS GO NOW CRITERIA

- Lactate >6 mmol/L or pH <7.0
- Upper airway obstruction persistent despite >2 doses of adrenaline, or hypoxic (SpO₂<90%)
- Pneumonia with hypoxaemia (SpO₂<90%) despite locally

- available non-invasive respiratory support
- Sepsis or shock requiring intubation
- Sepsis or shock despite >40ml/kg fluid
- Large pleural effusion (e.g. near white-out of hemi-thorax)
- Ongoing seizures despite 2 doses of midazolam and loading with a long acting agent (phenytoin, levetiracetam, phenobarbitone)
- Signs of raised intracranial pressure
- Unconsciousness with worse than flexion motor response
- Any arrhythmia with haemodynamic compromise (shock, hypotension, signs of heart failure)
- Any child with suspected systemic to pulmonary shunt who is about to be intubated or needing inotropes
- Any child with suspected cardiomyopathy / myocarditis who is about to be intubated or needing inotropes
- Cardiac or respiratory arrest
- Serum ammonia >200 mcg/dL

ECLS retrievals must always be co-ordinated by the PETS and ECLS consultant. They require an ECLS PIPER nurse, senior ICU doctor, cardiac surgeon and perfusionist. They take up to 16hr, so are a significant resource consideration. All interstate ECLS retrievals must first be ratified by the exec on-call for RCH.

Advising referring hospital staff

Secure the Airway, Breathing and Circulation. Intubate if necessary (nasal tube if appropriate); check ETT position on chest x-ray (tip at medial end of clavicles); strap ETT securely; humidification and frequent ETT suction; NG tube; adequate plasma expansion; guidelines on when to start inotrope infusion; check blood glucose and give dextrose if necessary; stop fits; continue ventilating any child after cardiac arrest even if they appear to be breathing adequately; maintain body temperature; urine catheter if necessary; check electrolytes if appropriate; splint arms. Doctor's letter to RCH; copy of observation chart and drug chart if possible; copy of x-rays if possible; ring RCH ICU if there is a problem before the PETS team arrives.

Mode of transport

All transport is organised through PIPER:
 1300 659 803 (Non Emergency calls), 1300 137 650
 (Emergency calls)
 Metro- Non-urgent: PIPER vehicle or taxi
 Urgent: Ambulance
 City Fringe & rural <150km- Non-urgent: PIPER vehicle or taxi
 Urgent: Helicopter

Rural >150km: Fixed Wing

Equipment to take

Complete a checklist (found in the PIPER Equipment room) for every retrieval. Standard equipment to take on all trips:

Sealed PIPER bag

Zoll Monitor

ECG leads, BP cuffs & cable, saturation probe and cable,

Invasive cables, ETCO₂, defib pads

3 Braun syringe pumps: 50ml syringesx3, long viggos (extension tubing) x3

Laerdal bag and mask

LTV/Hamilton ventilator and circuit - check oxygen inlet hose is attached

Spare LTV battery for interstate retrievals

ISTAT blood gas analyzer and cartridges

Plastic container for spare syringes, needles, and medications

Retrieval drugs from locked safe

Telephone / i-pad

PIPER folder, survey forms

Pedimate / Stryker Harness / Vacmat

Stretcher Bridge

Resus Chart

Map

Additional equipment or drugs to be taken for specific conditions

Asthma

Salbutamol-intravenous and nebulized

Atrovent

Aminophylline

Ketamine

Magnesium sulphate

Salbutamol metered dose inhaler and chamber

Nebuliser

Istat cartridge no: CG 4+

EzPap & Mask

HFNP

Bronchiolitis

EzPap

Suction catheters

Wrap around arm splints

Chloral Hydrate

High flow nasal prongs

Croup / epiglottitis / difficult airway

Extra long ETTs (croup tubes) if available
Suction catheters
Wrap-around arm splints
Glidescope
Bougies

Status epilepticus

Phenytoin
Diazepam
Phenobarbitone
Levetiracetam
Midazolam (enough for infusion)

Severe sepsis

0.9% saline
Dobutamine, adrenaline, noradrenaline
Central line: 3-lumen
Extra pressure transducer and cable
Extra 50 ml syringes x3
Extra long IV extension tubing x3
Extra Braun infusion pump
Urine catheter & bag
Istat cartridge No: CG4+

Head injury / multi trauma

Mannitol
0.9% saline, 3% saline
Central line: 3 lumens
Extra pressure transducer and cable
Extra 50 ml syringes x3
Extra long IV extension tubing x3
Extra Braun infusion pump
Chest drain catheters
Heimlich valve/pneumostat
Istat cartridge No: EG 8+
Vac Mat
Aspen collar of appropriate size

Picking up the patient at the referring hospital

The PIPER team should introduce themselves to anyone they speak to; listen to the history; be polite, interested and diplomatic. Unless inappropriate to do so (resuscitation), perform a 'huddle' between PIPER team and local team to discuss the expected plan and who will do what in case of expected or unexpected deterioration. Do not initiate discussion about things you consider to be mistakes in management (on return to RCH,

discuss these with the ICU consultant, who will arrange feedback later). If you are asked to comment, be diplomatic and consider the stress under which referring staff have been working. Use local expertise if appropriate. For example, a local anaesthetist may be more experienced at intubation than you.

Explain any changes in treatment you feel are needed before departure (eg changing oral to nasal tube). Ask the referring hospital staff to help in the preparations for departure. Use referring hospital consumables when appropriate. Go carefully through the pre-departure checklist on the retrieval form. Make sure the patient and equipment are stabilised before departure. Contact the ICU Consultant and receiving Consultant before departure. Check: water in ET tube cuff; avoid air splints; pneumothorax drained; adequate supplies of drugs, IV fluids, oxygen and batteries. Thank the referring staff, and ring them on arrival. Ensure the patient, all lines, equipment, bags and staff are secured in the vehicle.

At least one parent should be carried in the transport vehicle whenever possible- ask driver/pilot. Briefly discuss the child's illness and the retrieval and management plan. Mention any problems anticipated during retrieval. Tell the parents how to find RCH/MonashGive them the receiving hospital information handouts.

Analgesia and sedation during transport

Upper respiratory tract obstruction. Splint the arms securely; secure the ETT to with adequate strapping; ensure adequate ETT humidification and suction; NG chloral or IV midazolam if all other measures fail to reduce the risk of self-extubation.

Asthma, bronchiolitis, pneumonia, ARDS. If not ventilated, do not sedate during transfer: it suppresses respiratory drive and precipitates respiratory failure. If ventilated (not just CPAP or NCPAP), adequate analgesia and sedation (with NG chloral or IV midazolam) should be given. Muscle relaxant drugs are usually used during transport in these circumstances.

Conditions with raised intracranial pressure (head injury, meningitis, blocked VP shunt, tumour, intracranial haemorrhage). Unless they are ventilated, these children should not be sedated - sedation depresses consciousness, obscuring the main sign of progression of the intracranial hypertension, and suppresses the respiratory drive (raising PaCO₂ and ICP). If they are paralysed and ventilated, use morphine infusion or boluses of morphine (0.01-0.05 mg/kg/hr) to prevent increases in ICP caused by pain or distress. Midazolam 1-2 mcg/kg/min may be given IV if necessary unless neurological assessment

will be needed in the next 2-4 hours. If the PIPER team believes that a patient with raised ICP should be sedated before or during transport, this should be discussed with the ICU consultant on call.

Trauma. Children without head injury may be given a morphine infusion or small IV boluses of morphine (0.025 mg/kg) repeated as necessary up to 0.15 mg/kg per 4 hours. Children with a head injury and painful fractures or other painful injuries may require doses of morphine large enough to suppress spontaneous ventilation, and may require IPPV to allow adequate analgesia without secondary brain injury. Beware using intercostal nerve blocks before travel in aircraft (risk of pneumothorax).

Miscellaneous disorders (eg septic shock). Unless the child is clearly in pain or agitated, sedative or analgesic drugs should not be given as they may cause hypotension or impaired cardiac output during transport when observation and monitoring are difficult. If analgesia is required, consider giving ketamine by infusion (1 mg/kg/hr) or repeated IV boluses (0.5-1 mg/kg every 30 minutes). Otherwise, give small IV boluses of fentanyl (eg 0.2 mcg/kg) or morphine (0.025 mg/kg) repeated as necessary, allowing at least 2 minutes between doses to assess the effect of the drug on blood pressure and circulation. Repeated small doses of midazolam (0.05 mg/kg) to a maximum of 0.2 mg/kg may be given, especially if ketamine is used (beware reduction of blood pressure and cardiac output).

Retrieval Paperwork

Complete and thorough documentation must be completed for every retrieval. 15 min observations; note all drugs and fluids given, any changes in the child's condition, any problems occurring at the referring hospital and during transfer, times of arrival and departure. Whenever possible, leave a photocopy of the PIPER notes (up to departure) you have made with the referring hospital just prior to departure.

On return to RCH

Notify the receiving medical or surgical unit immediately. Discuss the child with the ICU consultant. Contact the referring hospital staff and let them know the child's condition on arrival and the management plan. Replace any items used from the PIPER bags. Clean the equipment and charge the monitor, ventilator, and syringe pumps.

PIM SCORE

The paediatric index of mortality (PIM2) is a statistical model that predicts the number of children who die after admission to intensive care. PIM can be used to assess quality of care in individual PICUs, and compare different ways of organising intensive care. PIM can also be used to assess risk-of-mortality in groups of children with a specific disease, but is not ideal for this purpose.

To assess the quality of care in an ICU, we use the standardized mortality rate (SMR). The SMR is obtained by dividing the number of patients who actually died by the number of deaths predicted by PIM. An SMR <1.0 suggests good quality care, and an SMR >1.0 suggests poor quality care (but the variation from 1.0 may well be due to chance if the 95% CI of the SMR includes 1.0). The SMR compares the actual number of deaths in the ICU with an estimate of the number of deaths that would have occurred if the same patients had been looked after in the units that derived the score (at the time the score was derived).

Before using the SMR results, check the performance of PIM in the ICU being assessed by using the area under the ROC plot – an area over 0.7 suggests that PIM is probably an appropriate model.

PIM (and PRISM) are designed to be used to describe groups of patients, *not* individual patients. They should never be used to decide that an individual patient is too sick to treat, or not sick enough to need intensive care.

It is important that the information used to calculate PIM is accurate – so it has to be collected very carefully. Data from a random sample of 20-50 patients should be collected in duplicate annually. At least 50 deaths are needed to obtain a reliable estimate of the SMR.

To allow for valid comparison, report results for a standard age range (such as 1 mo to 15 yr inclusive), and for ventilated and unventilated children. It is also important that paediatric intensivists obtain population-based information about children from defined regions who receive intensive care, and those who die without receiving intensive care.

PNEUMONIA

Indications for ICU admission:

- hypoxaemia ($SpO_2 < 90\%$) or severe respiratory distress despite oxygen therapy
- sepsis with hypotension or need for 40ml/kg bolus of fluid
- large effusion (e.g. near white out of a hemi-thorax or midline shift)
- Lactate $> 3\text{mmol/L}$

Do an NPA, blood culture, procalcitonin, FBE. Distinguish pneumonia (lung consolidation, small lung volumes) from bronchiolitis (gas trapping, prolonged expiratory phase, large lung volumes). If clinical / radiographic picture of bronchiolitis: antibiotics only if toxic, very unwell, Pct > 2 , IT ratio > 0.2 or high fever ($> 39\text{ C}$).

Antibiotics for pneumonia: benzylpenicillin and gentamicin are first-line therapy, except if you suspect Staph infection (flucloxacillin) or atypical (azithromycin), or hospital-acquired (see Sepsis – Hospital Acquired).

If empyema, give ceftriaxone, flucloxacillin and clindamycin (see below).

Give oxygen by mask or nasal prongs

If persistent respiratory distress give humidified high flow nasal cannula oxygen therapy or nasal CPAP.

Restrict fluids. If needing IV fluids give no more than 50% of normal maintenance requirements in the first 24 hours IV, unless dehydrated or hypovolaemic. Enteral feeding if possible.

Intubate if severe respiratory distress or persisting hypoxaemia despite HFNC or non-invasive respiratory support (CPAP / BiPAP), or severe sepsis *and* hypoxaemia. **Notify ICU**

consultant if need to intubate any child with pneumonia

- Assess cardiac function
- Be prepared for deterioration in oxygenation (de-recruitment with induction)
- Beware of over-bagging
- Pre-oxygenate, PEEP
- Fentanyl, vecuronium
- Stabilise with oral intubation first, re-recruit, suction, then change to nasal, insert NGT
- Beware ETT obstruction with secretions
- Chest x-ray to check ETT & NG position, no PTX

- Sedation with morphine / midazolam, as per sedation guidelines

Often after intubation of a child with pneumonia there is respiratory or cardiovascular instability. This can be mitigated by maintaining lung inflation with during induction, avoiding derecruitment, suctioning ETT early and gentle recruitment with long inflations, avoiding overinflation which will reduce venous return, pre-induction low dose vasopressor or inotrope if hypotension or poor perfusion. Decide if there is a component of airflow obstruction (such as in viral pneumonia) that may need a different ventilation strategy.

Ventilation

- PIP < 30
- PEEP 8 - 10
- FiO₂ < 0.6
- I-time 1 sec
- Ensure adequate expiration (no gas trapping)

Paralysis or HFOV if these parameters cannot be achieved, with PaO₂ > 60, pCO₂ 50-70 and pH > 7.2

Do echo if signs of heart failure (tachycardia, cardiomegaly, hepatomegaly) or murmur, measure lactate and SvO₂

Do a BAL in any child intubated with pneumonia.

Drainage of effusion / empyema (see below)

Consider V-V ECMO for refractory hypoxaemia despite HFOV

PNEUMONIA – EMPYEMA

Do not delay draining a large effusion in a child who has significant cardiac or respiratory compromise.

Do an ultrasound scan to confirm presence of effusion, look for septation, fibrin strands that suggest complex empyema, underlying lung for evidence of haemorrhagic change, abscess or necrotizing pneumonia.

Intubation of a child with large empyema often leads to severe deterioration. If evidence of cardiorespiratory compromise, drain the effusion early. Many children stabilise after primary drainage and avoid ventilation. On PETS retrieval, drain effusion in any unstable child with severe pneumonia prior to transport.

Chest drain: pigtail catheter if simple effusion, or if complex effusion: chest drain using blunt dissection. Send pleural fluid for Gram stain, culture, pneumococcal PCR, 16sRNA; antibiotics (flucloxacillin + cefotaxime + clindamycin)

+/- VATS in theatre either primarily or if no improvement after 48 hours of effective antibiotics.

Consider Urokinase if no improvement, but only do if no coagulopathy, and no evidence of haemorrhagic lung underlying empyema (do a CT if needed to identify this).

Urokinase - 40,000 units in 40 mL 0.9 percent saline for children one year and older, and 10,000 units in 10 mL 0.9 percent saline for children younger than one year. This dose should be administered twice daily (with a four-hour dwell time) for three days only.

Additional doses may be considered if the response is incomplete after six doses of Urokinase, but only if no bleeding complications or coagulopathy.

Intrapleural bupivacaine (0.5 to 1.0 mL/kg of a 0.25 percent solution) can be administered with urokinase if the child finds it uncomfortable.

If no improvement, do a CT, consider resistant organisms, TB, discuss with surgeons regarding thoracotomy.

PNEUMONIA / ARDS IMMUNOCOMPROMISED HOST

Diagnostic possibilities include bacterial (primary or VAP), viral, fungal, oedema (fluid overload or cardiogenic), immune, transfusion or chemotherapy related, baro-trauma, thrombo-embolic.

Many cases of ARDS in immunocompromised children are multifactorial. Investigate early, to avoid polypharmacy. Fluid overload and cardiac causes (cardiomyopathy, tamponade) may mimic ARDS

Risk factors for fungal and PjP include prolonged corticosteroid therapy, hematopoietic stem cell transplantation, prolonged neutropenia, and lymphopenia.

Do BAL, CT and consider lung biopsy early.

Investigations:

BAL bacterial Gram stain and culture, fungal wet prep and culture. Viral PCR: influenza, parainfluenza, RSV, adenovirus, human metapneumovirus, plus

PCR for CMV, pneumococcal 16s ribosomal RNA, Pneumocystis jiroveki PCR, consider HHV6, EBV, toxoplasmosis.

If vesicles: PCR for HSV, VZV, bacterial culture on vesicle fluid

If suspect fungal infection: as above, plus fundoscopy, urine microscopy and fungal culture, renal ultrasound, serum galactomannan (aspergillus), CT of sinuses

Atypical (chlamydia, mycoplasma, legionella)

If suspect Mycobacteria: GeneXpert MTB, Zeil Neelsen stain for AFB and culture

Consider *non-infective* causes:

Fluid overload

Transfusion related lung injury

Tumour infiltrate (cytology on BAL)

Pulmonary haemorrhage (haemosiderin-laden macrophages)

Engraftment syndrome: bone marrow recovery, GCSF related neutrophil infiltrate

GVHD (wide spectrum, to severe bronchiolitis obliterans in chronic GVHD)

Chemotherapy induced lung injury (alkylating agents, antimetabolites, bleomycin, others). Can cause various lung pathologies: diffuse alveolar damage, bronchiolitis obliterans (BO) or BO organizing pneumonia (BOOP), hypersensitivity pneumonitis, pulmonary oedema or haemorrhage, veno-occlusive disease, eosinophilic pneumonitis.

Chest CT with contrast early: fungal on CT (pulmonary nodules with ground glass halo); PjP: ground glass perihilar opacities. All non-specific.

Other investigations:

Procalcitonin

Serum ferritin, LDH, triglycerides, ALT, fibrinogen for HLH

Echocardiograph

Lung biopsy

POISONING

The severity of poisoning depends on the toxicity of the substance, its formulation, the quantity, and the time since exposure. These notes refer only to potentially serious cases.

Information about poisoning can be obtained from the ICU consultant, the Poisons Information Centre (phone 131126) and from Poisindex (www.clinicians.vic.gov.au/electlib.htm) and click on Micromedex then Toxicology).

1. Ipecac use is controversial, even if it is given within 1 hour of ingestion – so it is very rarely indicated in ICU.
2. Gastric lavage removes less than half of most substances even if performed promptly. Lavage should be discussed with the ICU consultant for potentially serious cases within 2 hours of ingestion (or longer if the drug impairs gastric emptying); it is contraindicated for corrosives, hydrocarbons and petrochemicals. Children who are not fully alert should be intubated before lavage (but note that intubation does not guarantee that aspiration will not occur). Lay the child on the side, head down. Use a single-lumen 24-32 French orogastric tube, and check position carefully (return of gastric contents and auscultate air injected into stomach). Instill 10 ml/kg aliquots of saline and then aspirate; repeat until the aspirate is clear (several litres may be required).
3. Several specific adsorbants are used: Fuller's earth (for paraquat), potassium ferrocyanate (for thallium), milk (for fluoride), sodium polystyrene sulfonate (for lithium), and cholestyramine (for lindane).
4. Activated charcoal adsorbs many poisons, but not common electrolytes, iron, mineral acids and bases, alcohols, cyanide, most solvents, hydrocarbons, pesticides, or lithium. It is most effective in the first hour after ingestion, with little benefit from a single dose of charcoal after 2-4 hours. Charcoal causes severe lung damage if aspirated. Children who are not fully alert should be intubated (but this does not guarantee protection against aspiration). Check that bowel sounds are present, pass an NG tube (minimum size 10 FG), check the position carefully (preferably by X-ray), empty the stomach as much as possible. Lie the child on the side, give charcoal 1 g/kg via NG tube, then (if slow-release preparation ingested) give 0.25 g/kg hourly. If multiple doses of charcoal are given, also give sorbitol 1 g/kg (1.4 ml/kg of 70%) NG repeated after 4hr.
5. Whole-bowel irrigation (with polyethylene glycol and electrolytes) is reserved for use in (a) delayed presentation after ingestion of slow-release preparations, or (b) substances

not adsorbed by charcoal (eg iron).

Eucalyptus and essential oils

Cause coughing, vomiting and aspiration pneumonia. Coma usually occurs within 30-60 min, but may be delayed. Do not use lavage, charcoal or bowel irrigation.

Iron

Take chest and abdominal X-rays (may see tablets). Charcoal is useless. Gastric lavage <2 hr. Whole bowel irrigation if no ileus, obstruction or erosion. Give desferrioxamine 10 mg/kg/hr for 12-24 hr (max 6g/24 hr) if serum iron >60-90 umol/L at 4 hr or 8 hr (x 5.59 = ug/dl).

Paracetamol

Discuss gastric lavage with the ICU consultant if it can be performed within 2 hours of ingestion. Give activated charcoal. Give acetylcysteine if plasma paracetamol >1000 umol/L at 4 hr, 500 at 8 hr, 200 at 12 hr, 80 at 16 hr or 40 at 20 hr (x 0.15 = ug/ml); give even if presentation is delayed: 150 mg/kg over 1 hour, then 10 mg/kg/hr IV. Monitor liver function tests and potassium.

Paraquat

>30 mg/kg severe toxicity, >50 mg/kg usually fatal: O₂ free radicals cause multi-organ failure over several days. Immediate charcoal 1g/kg NG, haemofiltration or charcoal haemoperfusion (within 2hr if possible), add N₂ so sats 70-80% (or V-V ECMO with FiO₂ <<0.10), desferrioxamine 3mg/kg IV over 24hr, acetylcysteine 10mg/kg/hr IV, dexamethasone 0.2 mg/kg 8H IV, vitamin E 300mg 12H NG.

Salicylates

Correct dehydration, acidosis and hypokalaemia. Bicarbonate to keep urine pH >7.5. Haemofiltration if serum level >25 mmol/L (x 13.8 = ug/ml). Monitor glucose, K, pH.

Theophylline

Consider gastric lavage (<2 hr), activated charcoal, or whole-bowel irrigation (delayed presentation of slow-release form). Consider plasmapheresis with levels over 500 umol/L. Metoclopramide for severe vomiting. Monitor ECG and potassium (low early, high later).

Tricyclic antidepressants

Monitor ECG (rate, QRS, QT). Adjust pH to 7.45-7.50 with bicarbonate or hyperventilation or both. Midazolam for con-

vulsions (or phenytoin if arrhythmia). Noradrenaline for hypotension (avoid beta agonists). DC shock then amiodarone up to 5 mg/kg or beta blocker for VT, VF. For torsade, give magnesium 0.1-0.2 mmol/kg (0.05-0.1 ml/kg of 50% MgSO₄). Consider trying 3 ml/kg of 3% saline slowly IV for severe arrhythmias or CNS toxicity. Monitor ECG in ICU until 12-lead ECG shows normal duration of QRS at normal pH (7.35-7.45).

PROCALCITONIN

Measure PCT in patients where temperature or IT ratio are unreliable, or high risk of sepsis:

- ECMO
- Haemofiltration
- Burns
- Traumatic brain injury
- Induced hypothermia
- Post-cardiopulmonary bypass
- Immunocompromised

Measure PCT if you are prepared to change management based on the result, i.e. if you will withhold, cease or scale back antibiotics if the result is <2ng/ml.

If PCT >2 ng/ml consider antibiotics according to PICU Antibiotic Protocol.

If the PCT is substantially rising (such as from 0.5 to 1.8 ng/ml over 24 hours) and there are other signs of sepsis, also consider antibiotics according to PICU Antibiotic Protocol.

The higher the PCT gets, the more likely bacterial sepsis, and the more strongly antibiotics are recommended. For example a PCT of >5 ng/ml mandates antibiotics.

Measure PCT in a child with fever T>38 C, if there are no absolute indications for antibiotics:

If <2 ng/ml consider to withhold, cease or scale back antibiotics.

If >2 ng/ml consider antibiotics according to PICU Antibiotic Protocol.

PCT should be measured no more frequently than 24 hours.

Key facts

No marker of infection can rule in or rule out sepsis in all children
Procalcitonin (PCT) is an early marker of sepsis; it is more sensitive and specific than C-reactive protein (CRP)

In normal healthy children the reference range for PCT is <0.05 ng/ml (or microgram per L) but this reference range is not appropriate for PICU

In local infection the PCT is typically >2 ng/ml, but may be in the range 0.5-2 ng/ml

In systemic inflammatory response without bacteraemia PCT is typically 0.5-2 ng/ml

In bacteraemic sepsis PCT is almost always >2 ng/ml, usually >10 ng/ml, and often in the 100s

In pneumonia or bronchiolitis a PCT is not very helpful in distinguishing between bacterial and viral aetiology, i.e. a child can have RSV bronchiolitis and a PCT of 2 or more; it is only if very high that this is suggestive of bacterial pneumonia.

Post cardiopulmonary bypass the PCT increases, peaking on day 2. In the absence of infection the PCT level post-CPB is typically <2.2 ng/ml

In neonates in the first 48 hours of life PCT levels are higher than the normal range in older children. A PCT of <10ng/ml can be seen in normal healthy newborns at 24-48 hours, but in a sick neonate a PCT >10 ng/ml strongly suggests sepsis. After 3 days of age threshold of >2 ng/ml should be used.

PCT decreases after infection is treated adequately

As it can take 2 hours to get a PCT result, a decision to start antibiotics in a potentially septic child should not be delayed.

Local infection

PCT is *not* needed to diagnose local infections. In many local infections the PCT range overlaps with the range of PCT in uninfected children in intensive care, so it will not be a strong discriminator of local bacterial infection in this population. Also, there are more specific clinical or laboratory signs referable to the site of the local infection (thrombophlebitis, sternal wound inflammation, cellulitis, white cells in urine, new opacity on chest x-ray). If these signs are present local infection is likely. Blood and other cultures should be taken, and the local infection should be treated appropriately (remove the IV drip or catheter, remove the infected sutures, treat the UTI, etc), without the need for using less-specific biomarkers.

Do not order PCT unless (a) there are clinical signs suggestive of infection and (b) you are prepared to withhold, cease, scale back or not escalate antibiotics if the PCT is <2ng/ml. PCT is *not* a "routine daily screening test" for sepsis.

Do not order CRP

Always take blood cultures before starting antibiotics

RHABDOMYOLYSIS AND MYOGLOBINURIA

Causes: prolonged seizure, heat stroke, extreme exercise, sepsis (influenza, group A strep, Staph aureus), crush injury, pressure effects of prolonged surgery, hypokalaemia, snake, spider or scorpion bite, neuroleptic malignant syndrome / malignant hyperthermia, metabolic abnormalities: LIPIN1 mutation (rare), mitochondrial disorders (long-chain fatty acid oxidation defects, phosphorylase kinase deficiency, MELAS), drugs (propofol, valproate, clarithromycin, cholesterol-lowering statins), DKA with severe hypophosphataemia.

Investigations: CK, acid-base, lactate, electrolytes, urine myoglobin, blood culture, viral PCR, serum ammonia, urinary organic and amino acids, other metabolic tests as directed by metabolic unit.

Early aggressive resuscitation with 0.9% saline is crucial. Maintain normovolaemia: CVP 5-8 cmH₂O and BP.

1. Forced alkaline diuresis: 500 ml N/2 (0.45% NaCl in 2.5% glucose; add sodium bicarbonate 25 mmol, 50% glucose 30 ml, and mannitol 20g (100 ml of 20%). Give 10 ml/kg/hr IV until there is no myoglobinuria (usually by the third day). Aim for urine output >2ml/kg/hour.
2. Keep urinary pH >6.5. Give acetazolamide if the blood pH >7.45.
3. Do urgent fasciotomy if compartment pressure >40 mmHg (or > diastolic BP – 30 mmHg) in adult.
4. High-flow CVVHF (see Filtration): if CK>5000 U/L & oligoanuria (UO≤ 0.5 ml/Kg/h for >6 hours, creatinine >2x baseline or rapidly rising) or fluid overload or metabolic derangement (acidosis and/or K≥6.5 or rapidly rising) or poor response to medical therapy.
5. Consider Rasburicase if uric acid level high
6. Metabolic consultation if cause uncertain. Consider desferrioxamine.
7. Calcium and loop diuretics (eg frusemide) may be harmful.

QUALITY

Quality in intensive care requires

- care delivered to accepted standards
- care based on evidence
- meticulous attention to detail
- adequate staff numbers and skills
- credentialing
- a continuous quality assurance program including a system of audit, including reporting and investigation of adverse events
- continuing education
- research
- a multidisciplinary approach and an open culture where people's views are respected
- highly effective communication
- family-centred care and care planning
- an ethical approach, including a focus on relief of suffering when cure is not possible
- a safe environment

Reporting of adverse events

All adverse events should be reported through the Victorian Health Incident Management System (Riskman Q) or through the ICU Quality, Data and Research team

The PICU has several **internal quality processes**:

The ICU Quality, Data and Research team collect data regarding admissions, adverse events and complications of disease, and discharges daily

Monthly morbidity meeting

Monthly quality meeting, which reports quality indicators, including:

- standardized mortality rates
- cardiac arrest data
- ECMO cannulation in PICU
- accidental extubations
- hand hygiene compliance
- central line infections
- ventilator associated pneumonia
- nosocomial bacteraemia
- wound infections
- accidental device removal
- pressure areas
- patient identification compliance
- sentinel events that occur around MET calls
- readmission or MET <24 hours post PICU discharge

SEPSIS - SEVERE

In sepsis-induced hypoperfusion, at least 30 mL/kg of IV crystalloid fluid be given within the first 3 h. Continue fluids resuscitation as long as there is positive effect. If more than 40ml/kg fluid needed, there is an urgent need to differentiate aetiology, address source control, and provide therapy based on the pathophysiology (i.e. vasoplegia, myocardial failure, warm or cold shock, hypovolaemia). Additional fluids guided by frequent reassessment of hemodynamic status: BP, HR, neurologic status, SvO₂, lactate, CVP trend. Echocardiography enables a more detailed assessment of the causes of the hemodynamic issues

Target mean arterial pressure is age based: MAP >40 (1-4 mo), >45 (5mo-5yr), >50 (6-7yr), >55 (>7yr).

Use noradrenaline 0.01-0.5 mcg/kg/min as the first-choice vasopressor. Use either vasopressin 0.02-0.06 units/kg/hr (especially if contractility acceptable) and adrenaline 0.01-0.5 mcg/kg/min (especially if contractility poor) or dobutamine (up to 15 mcg/kg/min) as next agents. Consider albumin 4% if > 40ml/kg required. If persistently poor contractility, rising lactate (not due to adrenaline), persistent hypotension diastolic BP <25 (<12mo) or <30 (<=8 yr) or <40 (>=9 yr): consider ECMO (VA, central).

Don't use low-dose dopamine for renal protection.

All patients requiring vasopressors need an arterial catheter placed as soon as practical.

Consider steroids (hydrocortisone, 1 mg/kg 6-8 Hr) if adequate fluid resuscitation and vasopressor therapy are unable to restore hemodynamic stability.

Respiratory: give high flow mask oxygen, or high flow nasal cannula oxygen or CPAP. Intubation. Indicated if there is severe respiratory distress or hypoxaemia despite oxygen, hypoventilation, impaired mental status or the presence of a severe shock state despite 60ml/kg and inotropes. Intubation can be hazardous, with many children deteriorating significantly on induction. Use ketamine, vecuronium or small doses of fentanyl. Have inotrope running, metaraminol and adrenaline ready (see also section on intubation of the child with pneumonia). Do not use thiopentone or propofol. **Ventilation.** Use minimum FiO₂ to maintain SpO₂ >90% (PEEP as needed). Ventilate to keep pH >7.25 (but minimum pCO₂ 35 mmHg, and max PIP 30 if <12 mo, or 35 if >12 mo). If pH <7.25 and base deficit >10 mmol/L, consider bicarbonate (IV over 1 hour).

Diagnosis and source identification

Send at least two sets of blood cultures (aerobic and anaerobic) without delaying antibiotic treatment. Send urine culture. No LP in a shocked patient. Consider other cultures as appropriate

(BAL). If a long term line is in-situ, send culture from lines + peripheral culture.

Blood cultures volume: collect 0.25 ml/kg (minimum 1ml, maximum 20ml) per sample. Higher volumes and multiple sampling times both increase the diagnosis of bacteraemia. There is a higher yield if 2 or 3 (but not more) percutaneous cultures can be performed at least 1hr apart. If only one culture is taken and coagulase negative *S. epidermidis* is isolated, contamination cannot be excluded. If subacute bacterial endocarditis is suspected, take 3-5 cultures over 48 hours.

Percutaneous blood sampling. Wash and glove, keep the skin moist with alcoholic chlorhexidene for at least 2 minutes, allow skin to dry, then use a no-touch technique.

Line culture. Wash and glove, use a swab-stick to carefully remove all visible debris from the 3-way tap, fill the luer fitting with 70% alcohol for 2 minutes, empty out the alcohol and allow to dry, remove dead space, aspirate the sample, replace the dead space, clean blood from tap.

Antimicrobial therapy

Antibiotic therapy should be initiated within one hour: specific antibiotics if organism known; otherwise give flucloxacillin (or vancomycin if hospital-acquired), cefotaxime and gentamicin. If a gut source is suspected, give penicillin and gentamicin and metronidazole. Give clindamycin and immunoglobulin if signs of toxin-mediated disease (toxic shock syndrome). If relevant, see on-line Febrile Neutropenia protocol. If relevant see Infections – Hospital acquired for indications for carbapenems.

Source control

Specific anatomic diagnosis of infection requiring emergent source control should be identified or excluded as rapidly as possible in patients with sepsis or septic shock, and any required source control interventions should be done as a matter of urgency.

Immunoglobulin. Give immunoglobulin if evidence of toxin-mediated disease (Group A strep or Staph): IVIg 0.5 g/kg IV over 2 hours stat, and after 36 hours.

Adjuvant therapies. Consider IV thiamine (if high lactate, malnutrition, vasodilated sepsis); steroids (as above); give G-CSF if neutropenia

Renal replacement. Consider venovenous haemofiltration if oliguria for >24 hours, or creatinine >0.4 mmol/L or increasing >0.1 mmol/L/day. Consider plasma filtration in severe sepsis (see below).

Nutrition. Use enteral feeding early, when organ perfusion restored consider early trophic feeds. Use RCH PICU feeding protocol in case of feed intolerance. Avoid early TPN. Insulin 0.05 u/kg/hr if reqd to keep glucose 4-12 mmol/L.

Heparin. Consider giving heparin 10u/kg/hr.

Blood products. RBC transfusion occur only when hemoglobin concentration decreases to <7.0 g/dL, in the absence of extenuating circumstances, such as myocardial ischemia, severe hypoxemia, single ventricle physiology, prematurity or acute haemorrhage.

Consider platelet transfusion when counts are < 10,000/mm³ in the absence of apparent bleeding and when counts are < 20,000/mm³ if the patient has a significant risk of bleeding.

Higher platelet counts (≥ 50,000/mm³) are advised for active bleeding, surgery, or invasive procedures.

Plasma filtration

Consider in severe sepsis.

- cannula: 8 Fr in child (blood flow 30-60 ml/min), 11.5 Fr in a teenager (blood flow 50-100 ml/min)
- filter: Gambro PF 1000 or PF 2000 (or equivalent)
- filtrate: 20 ml/kg/hr for 6 hr, then 10 ml/kg/hr for 30 hr
- replace any filter that clots before 24 hours
- Heparin: 1000u in 50ml saline at 1-10ml/hr prefilter, 1000 u in 50 ml saline at 1-10 ml/hr postfilter. Keep ACT x 1.5 normal (Hemochron 160-180)
- acid citrate dextrose, formula A (ACDA): 1 ml/hr prefilter for every 1 ml/min of blood flow
- replacement fluid: dextrose 0.3%, Na 135 mmol/L, K 3.5, Ca 2.0, Mg 0.7, Bic 25, Phos 1.5, Cl 100, acetate 9.2, albumin 30g/L; give 1 bag of FFP (about 230ml) after every 800 ml of replacement, and cryoprecipitate 5 ml/kg (1 bag/4kg) if fibrinogen <2.0 g/l.

SCOLIOSIS REPAIR POST-OP

Some children after scoliosis repair will have prolonged weaning from mechanical ventilation. Check for lung collapse, pneumonia, chest flail (if more than one rib removed), diaphragm paralysis (especially if anterior approach).

Positioning. Keep the back straight at all times. Beds should generally not be broken, due to the risk of patients sliding down and putting pressure on the instrumentation, which is often in soft bone. If you must break the bed i.e. post extubation then ensure that the flexion is only at the hips and not in the lower back. Never leave a patient in such a position that they can slide down the bed. **A much safer position is with bed tilted** rather than broken in the middle.

Considering the importance of physiotherapy after extubation, and to increase the chances of successful extubation, aim to extubate these patients early in the day. This will often be day 1 post-op.

Children on pre-existing respiratory support (e.g. nocturnal CPAP or BiPAP) or those with other risk factors for respiratory failure, should be extubated to NIV.

Once extubated, all children can be hoisted into a sitting position in a chair (with the help of a physiotherapist).

SNAKEBITE

Fang marks may not be visible, bite site may not be painful. There may be headache, nausea, vomiting, abdominal pain, tender local lymphadenopathy, weakness (ptosis, blurred vision, bulbar palsy, general or respiratory weakness), coagulopathy (bleeding, or just abnormal tests), hypotension, rhabdomyolysis and renal failure.

Always inform the ICU consultant about any patient with potential or confirmed envenomation.

Examine the patient carefully; insert an IV; take blood for FBE, PT, PTT, fibrinogen, FDPs, CK, creatinine.

When adequate amounts of antivenom are available:

- **possible bite** (asymptomatic, normal coag tests): remove pressure bandage and observe for 4 hours if well
- **definite bite** (asymptomatic, normal coag tests): remove pressure bandage and observe for 12 hours if well
- **definite bite** (mild symptoms, or abnormal coagulation): admit to ICU, test for venom on swab of site (and in urine if swab negative), discuss with ICU consultant
- **very ill** (hypotension, bleeding, paralysis): full resuscitation, admit to ICU, leave bandage on (or apply), give 2 amp of tiger and brown snake antivenom (total 4 amp), test for venom on swab of site (and in urine if swab negative). Any further antivenom should be for that snake (see below). Obtain more supplies of antivenom (RCH holds only 4 amp tiger and 4 amp brown). Only remove bandage when stable (may then need more antivenom).

Venom detection kit. Used to determine whether envenomation has occurred (but *not* to decide whether to give antivenom), and which antivenom to use. Do not rely on an observer to identify the snake, use the kit.

Antivenom. In Victoria, all snakes are covered by giving antivenoms against tiger snake (covers red-bellied black, copperhead and tiger snakes) plus brown snake. Give only for significant symptoms or coagulopathy (not minor symptoms or minor coagulopathy). The dose of antivenom depends on the amount of venom injected, NOT the size of the patient. Repeated doses may be needed if symptoms or coagulopathy persist (after 30min) or recur.

Adrenaline premedication. Give 0.01 ml/kg of 1/1000 SC *before* the first dose of antivenom.

FFP and platelets. Do NOT give these until adequate antivenom has been given.

Tetanus. Give prophylaxis if indicated.

SPINAL INJURY

Assume any child with significant trauma has a spinal injury

Injuries commonly occur at several vertebral levels.

< 8 y.o.: mostly C1-2; >8 y.o.: C6-T1 or low thoracic.

Lap belt: consider Chance fracture: flexion; crush T12-L2 body; duodenum; pancreas

Assess and maintain ABCD: prevent hypoxia and hypotension

Intubate if necessary: manual inline immobilisation without distraction; RSI + pre-oxygenate

Assess for bradycardia and hypotension; pain/tenderness of spine; weakness; numbness; paraesthesiae; lax anal tone; priapism; distended bladder.

Hypotension + tachycardia: consider blood loss: give 10 ml/kg boluses

Hypotension + bradycardia; check for blood loss; give one 10 ml/kg bolus then noradrenaline infusion + atropine or benzotropine or benzhexol boluses; insert CVC and measure CVP

Beware ongoing paroxysmal bradycardia; esp. with ET suction; consider regular benzhexol or aminophylline.

Avoid hard collar < 2 y.o.

Maintain spine in alignment without distraction

Spine board in children <8 y.o. should have 2-4 cm deep pad extending from knees to shoulders

Remove collar; manual immobilisation and log roll to examine spine.

Examine for other injuries: head; neck; trunk; pelvis; limbs

Plain X-rays: A-P; Lateral; Odontoid (if >5 and cooperative); CT sensitive if suspect C0-C1#

MRI if suspect cord injury, including SCIWORA

Nil by mouth until bowel sounds appear; give PPI for 4 weeks

Maintain body temp $\geq 36^{\circ}\text{C}$

Urine catheter: indwelling for 3-4 days, then intermittent

Pressure area care: log roll 2 hourly

Pain control: opioids; consider gabapentin

DVT prophylaxis in adolescents, especially girls

LMW heparin unless surgery is likely; calf compressors

Don't give steroids

SPLENECTOMY OR ASPLENIA

1. Patients should be informed about the risk of infection, and advised to wear a form of medical alert (eg bracelet). Patients should be advised to see their doctor when unwell, when planning to travel to a malaria-endemic area or if bitten by any animals.
2. The patient's medical records should be prominently labelled with the information that he or she has had a splenectomy, or is asplenic.
3. Conjugate vaccines against pneumococcus, meningococcus type c and *Haemophilus influenzae* type b should be given at least 14 days before splenectomy or as soon as possible after the operation. Booster injections every five years should be considered. Measurement of antibody levels as a guide to the need for revaccination has been suggested, but interpretation is often difficult.
4. Oral phenoxymethylpenicillin (adults: 250 mg or 500 mg twice daily), or, for patients allergic to penicillin, erythromycin (adults: 250 mg to 500 mg twice daily) should be given for at least one to two years after splenectomy. Immunocompromised patients should be advised to take lifelong prophylaxis.
5. Upon cessation of penicillin prophylaxis, patients should have an amoxicillin 3 g tablet (for adult) available to take if a fever develops, especially if immediate medical attention is not at hand.

SURGICAL PROCEDURES IN THE GENERAL ICU

The following surgical or interventional procedures may be done under certain circumstances in the ICU

- Percutaneous tracheostomy
- Chest drain insertion
- Bronchoscopy - flexible
- Emergency escharotomy for burns
- Wound suturing
- Cut-down vascular access
- Bone marrow aspirate

Other routine medical procedures done by ICU staff

- Lumbar puncture
- Broncho-alveolar lavage
- Bronchoscopy
- Vas-cath insertion

Not every time or in all circumstances will it be appropriate for these procedures to be done in PICU.

If there are multiple procedures to be done on the one patient it is often better to do these in OT. If OT time is available without delay which would compromise the safety of the patient, procedures such as burns escharotomy are better not done in ICU.

The same considerations apply as in cardiac procedures: procedures can only be performed in ICU if there are adequate staff and facilities to do so safely, and if they will not cause excessive disruption to the running of the ICU and the care of other patients. The anticipated duration and complexity of procedures must be taken into account on any given day.

The procedure should be planned by the surgeon and ICU consultant together and communicated by the ICU consultant with nursing and other staff. There must be an assessment of feasibility, availability of staff and timing, before finalising arrangements.

If the procedure requires a surgeon (General, Plastic, Neuro, etc) the procedure should be booked as routine through the OT.

TRACHEOSTOMY CHANGE

Equipment needed before attempting the procedure: oro-pharyngeal airway, suction, replacement tracheostomy tube and smaller reserve tracheostomy tube, lubricant, stomal dilators, flexible tubing which can be used as a tracheal guide (eg a nasogastric tube or tracheal suction catheter with the proximal end cut off).

Procedure to be followed:

1. Place a rolled towel or pillow beneath the shoulders of the patient so that the neck is in a position of extension. Do not place the towel or pillow beneath the head of the patient. Position the head, neck and body in-line supine. Lubricate the new tracheostomy tube.
2. Suction the trachea before attempting tube change.
3. Undo the tracheostomy tapes.
4. Ventilate with 100% oxygen for at least 5 breaths so that the lungs are filled with oxygen.
5. Pass the flexible catheter being used as a guide down through the old tracheostomy tube to the carina.
6. Remove the old tracheostomy tube, making sure that the tracheal guide remains in the trachea.
7. Pass the new tracheostomy tube over the guide into the trachea, and remove the tracheal guide.
8. Bag with 100% oxygen.
9. Re-tie the tracheostomy tapes.

Course of action if difficulty is experienced in replacing the tracheostomy tube:

1. If the stoma is constricted, the stomal dilator may be used to aid insertion of the new tube. Alternatively, graduated dilators may be used (for example, a series of smaller to larger size endotracheal tubes).
2. Avoid hypoxaemia at all costs. Remember that unless there is obstruction above the tracheostomy site, the patient can always be ventilated by bag and mask (with the tracheostomy stoma occluded by a second person using their fingers). Do not persist in your attempts to insert the new tube if the patient is cyanotic; bag and mask until the patient is pink, and then try again with a new or smaller tube. As a last resort, if the new tracheostomy tube cannot be inserted, oral endotracheal intubation may be necessary.

The key to an anxiety-free tracheostomy change is to be prepared. If difficulty is experienced, ventilate the patient by bag and mask to avoid hypoxaemia between successive attempts.

TRACHEOSTOMY SIZES

Tracheostomy uncuffed tube sizes

	Portex			Shiley			Bivona		
	ID	OD	Lth ^a	ID	OD	Lth ^a	ID	OD	Lth ^a
Prem	-	-	-	-	-	-	2.5	4.0	30/38
Term	3.0	4.2	35	3.0	4.5	30/39	3.0	4.7	32/39
<6mo	3.5	4.9	39	3.5	5.2	32/40	3.5	5.3	34/40
1yr	4.0	5.5	43	4.0	5.9	34/41	4.0	6.0	36/41
2-3yr	4.5	6.2	46	4.5	6.5	36/42	4.0	6.0	36/41
4-5yr	5.0	6.9	50	5.0	7.1	44/50	4.5	6.7	42
6yr	5.0	6.9	50	5.5	7.7	46/52	5.0	7.3	44/60
8yr	6.0	8.2/9.2	55/65	6.0	8.3	54	5.5	8.0	46
10yr	6.0	8.2/9.2	55/65	6.5	9.0	56	6.0	8.7	70
12yr	7.0	9.6/10.5	65/70	6.5	9.0	56	6.5	9.4	70
14yr	7.0	9.6/10.5	65/70	5.0	9.4	65	7.0	10.0	80
16yr	7.5	10.3/11.3	71/73	6.4	10.8	76	7.5	10.4	80
Adult	8.0	11.0/11.9	76/76	6.4	10.8	76	8.0	11.0	88
Adult	8.0	11.0/11.9	76/76	7.6	12.2	81	8.5	11.8	88
Adult	9.0	12.3/12.3	87/81	7.6	12.2	81	9.0	12.3	98
Adult	10.0	13.7/14.0	98/88	8.9	13.8	81	9.5	13.3	98

a. Length from mid-stoma to tip of tube

TRANSPLANT – HEART

Postop immunosuppressive regime (basic)

Cyclosporin 0.1-0.2 mg/kg/hr by continuous IV infusion for first 24-48 hours, then 5-7 mg/kg/12hr orally. Lower doses will be required in renal failure. Adjust dose to obtain trough levels of 250-400 nanogram/ml, depending on urine output.

Azathioprine 1-2 mg/kg IV or oral daily. Withhold if WBC drops below 4000.

Methylprednisolone 10 mg/kg/dose IV 12 hrly for 4 doses.

Aspirin 75-150 mg daily, start day 2.

Acyclovir IV: 5 mg/kg/dose over 1 hour 8 hourly. NG or oral: <2 yr 100 mg qds, >2 yr 200 mg qds.

Cotrimoxazole 2.5 mg/kg/dose (trimethoprim) 12hrly IV over 1hr or oral for 2-4 wk, then 3 times a week for 3mo.

Nystatin 500,000u NG 6 hourly.

Cyclosporin levels

1 ml in heparinised tube to Biochemistry. Send on return from theatre and then at least every day until level stable. Cyclosporin dose determined by cardiologist or cardiac surgeons.

BLOOD PRODUCTS AND CMV

All blood products must be irradiated.

Recipient CMV +ve

No filtration and no CMV immunoglobulin.

Recipient CMV -ve, organ donor CMV +ve

- give CMV immunoglobulin (from City Blood Bank or CSL) 100 mg/kg over 2 hours, 50 mg/kg over 1 hour day 2, then 50 mg/kg over 1 hour each week for 4 weeks
- filter red cells (Pall leucocyte filter)
- filter platelets (Pall platelet filter)
- FFP, SPPS, and albumin do not need to be filtered
- Ganciclovir: 10 mg/kg daily for 14 days, then 5 mg/kg daily.

Recipient CMV -ve, organ donor CMV -ve

- no CMV immunoglobulin
- filter red cells (Pall leucocyte filter)
- filter platelets (Pall platelet filter).

Procedures

When taking blood samples, eg for a blood gas, full sterile procedure to be followed.

TRANSPLANT - LIVER

Pre-transplant workup will have been completed before transplant for all patients except fulminant hepatic failure - check with the liver transplant coordinator or hepatologist on call that the work-up is complete.

Post-op management: day of transplant

The child will return from theatre with an ETT, NG tube, urinary catheter, triple lumen CVC, arterial line, 2 peripheral IV lines, 1-2 abdominal drains on closed suction, bile duct T-tube on closed drainage, and a Swann Ganz catheter in larger children.

Routine care. Nil orally until surgeon says to feed; routine oral hygiene; NG tube on free drainage with hourly aspiration; strict fluid balance, daily weigh after transfer to ward.

Fluid balance. Postoperative fluid requirements are variable. Vasodilatation from rewarming, intra-abdominal bleeding from extensive raw surfaces, and the reaccumulation of ascites cause extravascular losses that must be corrected. Give sufficient crystalloid to replace urine, insensible and third space losses. The haematocrit should be maintained at around 30% (Hb 90-100) to optimise perfusion and minimise the risk of hepatic artery thrombosis. Serum albumin should be kept at 28-30 g/L and right atrial pressure at 9-10mm Hg

Renal function. Often impaired after liver transplantation. Patients may arrive with grossly positive fluid balance yet low filling pressures because of intra-operative blood loss, IVC clamping, veno-venous bypass, and nephrotoxic drugs (cyclosporin, tacrolimus, acyclovir, amphotericin, cotrimoxazole). Give mannitol intraoperatively, and low dose noradrenaline to defend MAP for 24hr post-op. Closely monitor fluid balance and volume status, urine output. If the urine output drops, give fluid challenge: 4% albumin 10 ml/kg (or 20% albumin 5 ml/kg if serum albumin < 30 g/l). If urine osmolality < plasma, give frusemide. If no response, give mannitol 0.25 gm/kg, then a large dose of frusemide + hydrochlorothiazide. If oliguria persists, use haemofiltration early. Cardiac output may be 2-3 times normal with the new liver. Maintain CVP approximately 9-10 mmHg. Check Na⁺, K⁺, urea and creatinine 4-6 hourly. In established renal failure, replace urine output, insensible losses, NG aspirate, drainage and bile losses and review all drugs.

Abdominal drains. The Jackson Pratt drains are connected to Minivacs. There may be substantial loss, with ascites, chylous

peritoneal fistula due to coeliac axis dissection, leakage from raw surface if cut down liver. Bleeding or bile leakage may require reoperation (ascitic fluid can be easily mistaken for bile. The JP drains should be milked hourly (wet with Hexol or Alcowipe) and a sample of drain fluid taken at least daily; the drain is disconnected, emptied, wiped with Betadine and reconnected using a no-touch technique. The surgeons should be notified if the haemoglobin concentration of drain fluid >3gm/100 ml. The drain is removed on day 2-10. Drain losses should be replaced ml for ml with 4% albumin. Check electrolyte and protein content daily to guide replacement. T-tube. In patients who have had duct-to-duct reconstruction, a T-tube is left in place and drained into a closed system. Because of delayed healing due to protein malnutrition and high dose steroids, the T-tube is left in place for at least 3 months. The bag is only changed as necessary. Early withdrawal is associated with a high risk of biliary leak. In patients in whom the recipient common bile duct cannot be used for reconstruction, a choledocho-jejunostomy is constructed using a Roux loop. This will have external drainage but radiological definition will be suboptimal as the tube has end and side holes. The tube is clamped around day 10 to 12. Clamping the T-Tube and cholangiography will usually result in a jump in hepatic enzymes. Clamping the T-Tube will increase absorption of cyclosporin (but not tacrolimus). All invasive radiology of the bile tract must be covered with prophylactic antibiotics to reduce the risk of septicaemia.

CVS monitoring. Cardiac monitoring includes ECG, BP, toe temp, urine output and plasma lactate. Rarely, cardiac output is measured by thermodilution via a Swann Ganz catheter. Arterial lines and 4F central lines are flushed with hep saline 1u/ml at 1 ml/hr. No heparin is used in any other line, including a haemofilter (but see Haemofilter protocol below). Hypertension is always present post-transplant and usually lasts some weeks. The BP is very labile and is affected by volume loading, cyclosporin, tacrolimus and steroids. Exclude correctable causes (hypoventilation, hypoglycaemia, fits, awareness, pain). Hypertension is controlled (due to the risk of bleeding) using SNP (measure plasma cyanide) and esmolol. Later, control BP with captopril, nifedipine and atenolol orally.

Central nervous system. Full neuro obs chart is important post-op, especially when coma was present before operation. Development of encephalopathy may mean a non-functioning hepatic graft, but is difficult to recognise in a paralysed and sedated patient. Dilated or sluggishly reactive pupils plus irritability may be the only signs. Papilloedema is uncommon. Circulatory instability may indicate coning and may require

urgent measures to control ICP: consider craniectomy and hypothermia; elevate head of bed 20°; ventilate to PaCO₂ 35 - 40 mmHg; mannitol 0.25 - 0.5 g/kg; aim for a serum Na 145-150 mmol/l and plasma osmolality 300-310 mOsm/kg.

Respiratory. Take chest x-ray on return to ICU, then daily as required. PEEP should be kept low, as high PEEP levels reduce hepatic blood flow. 2 hrly suctioning of ETT while intubated. Ventilation is usually needed only for 24-48 hrs postop. Spontaneous ventilation will be allowed as soon as conscious state and gas exchange allow, with early extubation. The patient should remain in ICU following extubation and be reintubated promptly if respiratory function is inadequate. Pulmonary infections are a major problem because of decreased vital capacity, the subcostal incision, pleural effusions and respiratory muscle weakness due to preoperative malnutrition. Atelectasis is treated with aggressive physiotherapy. Chest physiotherapy with face mask CPAP and deep breathing and coughing exercises should start immediately after extubation. Pleural effusions, particularly on the right side are common after liver transplantation, but usually resolve spontaneously; occasionally thoracentesis is required if ventilation is impaired. Ascites can rapidly traverse the diaphragm and cause tension hydrothorax. Pulmonary oedema often occurs postoperatively and may be difficult to diagnose, as it may occur with low filling pressures and with a normal or high urine output: a dose of furosemide, and repeat examination and chest x-ray may clarify the diagnosis. Pulmonary capillary injury similar to ARDS may develop with fulminant hepatic rejection, hepatic infarction or sepsis. High cyclosporin concentrations administered into a central vein may precipitate wheezing, so cyclosporin must be given by infusion into a peripheral vein. Older children with long-standing cirrhosis may have substantial pre-transplant intrapulmonary shunts (hepatopulmonary syndrome) or rarely pulmonary hypertension (porto-pulmonary hypertension).

Temperature. Hypothermia is common on arrival in ICU because of long operation time, large volume replacement, veno-venous bypass, and rewarming of a cold liver graft. Nurse babies <7 kg on a radiant heater. Rewarming may require a heating blanket, heated humidification, and warmed fluid infusions. Any fever is abnormal and should be investigated.

Infection control. More than 50% of patients develop bacterial, fungal or viral infections after liver transplantation: most pathogens originate from endogenous flora. The risk is greatest in the first 6 wks. Reverse barrier nurse in a single room for the first 48 hours postoperatively. Use gowns while the child is

ventilated and invasively monitored, but hats and masks are not required. All IV, IA and CVC lines, drain tubes, tracheal aspirate and urine are cultured daily. Tips should be cultured on removal of lines. All procedures will be carried out by using RCH PICU protocols, adhering to them very strictly (3rd daily CVC and peripheral line changes with scrubbing to change them, scrubbing to break into a central line, clean no-touch method to give IV drugs via a drug bung.) After extubation and removal of invasive monitoring lines, reverse barrier nursing is replaced by routine precautions. This means: a single room; very strict hand washing protocols; gowns to protect clothing; restrict visitors to two per child (none with infections).

Patient movement and data. Most patients stay in ICU for two or more days before transfer to the ward. A flow chart of pathology results and clinical data will be maintained in the patient's room. Once mobile, the patient may go outside the ward with mask on for limited periods in non-crowded areas (eg Avoid Starlight room, McDonald's etc).

Electrolytes and glucose. Patients with end stage liver disease often have low plasma Na, K, Ca, Mg and PO₄ because of prolonged diuretic therapy. This will be complicated by intraoperative changes in serum K and ionised Ca.

Potassium. Reperfusion of the new liver is associated with severe but transient hyperkalaemia. Serum K will fall as the hepatocytes take up K, and molar KCl infusion may be required. Persistent hyperkalaemia often means poor liver function. Graft necrosis can result in sudden release of large amounts of K and consequent hyperkalaemia.

Sodium. An accurate assessment of total Na loss can be made from the volume and measured Na content of drain fluid, bile and urine output. The Na content of infusions and drugs should be noted. Hypernatraemia and hyponatraemia should be corrected no faster than 0.5 mmol/L/hr.

Calcium, phosphate, magnesium. Low Ca, Mg and PO₄ after operation may require correction. PO₄ falls as liver function recovers post-transplant as a general re-feeding phenomenon. Both cyclosporin and tacrolimus cause high urinary loss of Mg. Check Na, K, Mg, PO₄ 4-6 hourly. Check liver enzymes, proteins, bilirubin and NH₄ on arrival from theatre and 12 hourly thereafter (6 hourly if there is a problem). Citrate intoxication and acidosis is common following massive transfusion and is an early sign of poor liver function. The resulting acidosis is corrected with bicarbonate and hypocalcaemia with Ca. A

normally functioning graft metabolises citrate and converts a mild to moderate metabolic acidosis to a mild alkalosis within a few hours. Rewarming and restoration of peripheral perfusion further reduces the acidosis.

Glucose. Revascularisation of the grafted liver is associated with increased glucose uptake. Hyperglycaemia immediately postop is due to the glucose content of infused blood, and decreased utilisation due to hypothermia and high dose steroids: insulin infusion may be needed.

Nutrition. Most children requiring liver transplant have pre-existing protein-calorie malnutrition. Post-op energy requirements are high. Enteral feeding will commence as soon as normal gut activity returns, 24-72 hours post-op. TPN may be required 36-72 hours post-op, if there is no gut activity. Consult the hepatologist before using Intralipid. Insulin may be needed for hyperglycaemia.

Coagulopathy. Children with liver failure have elevated PT and APTT and decreased factors (II, VII, IX, X and XII), low platelet count, and elevated FDP with fibrinolysis. Fibrinogen and factor VIII are usually normal. These abnormalities are only corrected pre-op if the patient is bleeding. During transplantation, administration of FFP, platelets, blood, and cryoprecipitate is titrated against bleeding and test abnormalities, especially thromboelastography. Low antithrombin III, protein C, protein S levels can lead to thrombosis of hepatic artery, hepatic and portal veins. Routine Management: check Hb, WCC, platelets, PT, APTT and fibrinogen 4-6 hrly; maintain Hb at 90-100 g/L; give platelets only after discussion with the transplant unit; FFP is given for coagulopathy only if INR >3 and after discussion with the transplant unit; heparin is given SC or by IV infusion post-op when the PT and APTT fall below 1.5 times normal (maintain anti-Xa levels of 0.1 -0.3 IU/ml, mild anticoagulation). Heparin is replaced by aspirin when oral intake is established and no short term need for liver biopsy. All patients are given dextran 40 for 5 days postop. Measure antithrombin III (AT III), protein C and protein S levels daily. Maintain AT III level at >80% control - use 1000u daily. Maintain protein C and S at >60% activity using FFP 15ml/kg. There should be 2 units of cross-matched blood available at all times. Severe coagulopathy: bleeding from all puncture sites often indicates deteriorating liver function. Intra-abdominal bleeding (increased abdominal girth and falling haematocrit): obtain urgent ultrasound; correct abnormalities of coagulation and platelet count; notify surgeons (usually needs laparotomy).

Haemofiltration. If the liver is still functioning and haemofiltration is required, use ACD solution instead of heparin in the circuit. If the liver is not functioning, only albumin or saline solution should be used. ACT should still be checked hourly. Watch the circuit closely for the formation of clots. (1) If clotting normal and platelets normal: give replacement fluid pre-filter, prime with total 10u/kg heparin, infuse 10 units/kg/hour heparin pre-filter. (2) If coagulopathy or platelets <60,000 but no clinical bleeding: no heparin in prime, infuse replacement fluid pre-filter, infuse prostacyclin 5 ng/kg/min pre-filter. (3) If clinical bleeding and coagulopathy: no heparin in prime, infuse replacement fluid pre-filter, no heparin or prostacyclin. (4) If fulminant hepatic failure (no anti-thrombin III production): regional heparinisation with heparin 10 u/kg/hr prefilter, protamine 0.1 mg/kg/hr postfilter, cryoprecipitate to replace AT3.

Immunosuppression

Methyl prednisolone. 2.5mg/kg/dose (max 25 mg) 6hrly x 4; 2.0mg/kg/dose (max 20mg) 6hrly x 4; 1.5mg/kg/dose (max 15mg) 6hly x 4; 1.0mg/kg/dose (max 10mg) 6hrly x 4; 1.0mg/kg/dose (max 10mg) 12hrly x 2; 1.0mg/kg/dose (max 10mg) daily oral.
Tacrolimus. 0.05mg/kg/dose enteral 12hrly, starting within 24hr of transplant OR cyclosporin 5mg/kg 12hrly via NG tube. IV cyclosporin is only used in exceptional circumstances. Take blood for level just before 9am dose (do not wait for result); Gastroenterology will obtain the result and adjust the 9pm dose to achieve tacrolimus level 10-12mg/L or cyclosporin 300 ng/ml.
Azathioprine. 1.5 mg/kg nocte.
Rejection. Manage in consultation with hepatologist on call. Ensure the cyclosporin/ tacrolimus level is adequate. Pulse methylprednisolone, if fails OKT3.

Antibiotics

Preoperative prophylaxis. To be given in theatre and repeated at hepatectomy as necessary, then for 48 hours post-op.
Cefotaxime 25mg/kg 8hrly x 6 doses; amoxycillin 25 mg/kg 6hrly x 8 doses; vancomycin if MRSA positive.
Biliary prophylaxis (before T-tube cholangiogram, PTC, and T-tube removal). Cefotaxime 25 mg/kg before procedure; amoxycillin 25 mg/kg before procedure. Antibiotics changed according to the sensitivities of any organisms recently grown in the bile.
Antifungal prophylaxis (for 1 month post-op). Nystatin 1ml oral 6hrly plus 2ml via NG tube 8hrly. Nystatin cream to skin creases, groin, axilla and nose 8hrly.
Antiviral. Acyclovir 500 mg/m² 6hrly oral for 3 months. CMV seronegative recipients of CMV positive donors will receive

gancyclovir (instead of acyclovir) for 3 months, then acyclovir: 5mg/kg/dose bd IV, or 20-40mg/kg/dose tds oral. Monitor CMV antigenemia (Buffy coat) weekly.

Toxoplasma prophylaxis. Give pyrimethamine to toxoplasma seronegative recipient of seropositive donor.

Pneumocystis prophylaxis (should commence within one week of transplant and continue for 6 months). Cotrimoxazole (40/200) <2yr 2.5ml daily oral, >2yr 5ml daily oral. If allergic to cotrimoxazole, give pentamidine 3mg/kg monthly by inhalation for 6 months.

Other drugs

Analgesia. Morphine infusion starting at 20 mcg/kg/hr and increased cautiously if necessary, titrated against pain, wakefulness and breathing, reduce if poor liver or kidney function. Avoid barbiturates in encephalopathy.

Prostaglandin E1. Used as immunosuppressant. Causes splanchnic vasodilatation, decreases acid secretion in the gut, protects hepatocytes and platelets. Dose 10ng/kg/min.

Gastric cytoprotection. Sucralfate: 0-2yr 250mg 6hrly; 3-12yr 500mg 6hrly; >12yr 1g 6hrly oral. Omeprazole if GIT bleeding: 0.4-0.8mg/kg/dose 12-24 hrly IV or oral.

Anticoagulation. Heparin 10 u/kg/hr IV infusion when PR and PTTK <1.5 times normal. Maintain anti-Xa levels of 0.1-0.3 IU/ml (mild anticoagulation).

Antithrombin III. Once daily if the antithrombin III level <80%. Dose 1000 International units (ie 1 vial).

Dextran 40 in N/saline or 5% dextrose <10kg 2.5ml/hr for 5 days. 10kg 5ml/hr.

Aspirin. Commenced when heparin ceases. 8-15kg 50mg daily orally; >15kg 100mg daily oral.

Vitamins A, E, and K. Vitamin K on return from theatre and daily until established oral intake. Micelle A & E 1 ml daily by mouth. Folate 2.5 mg/5ml/day oral for 2 wk.

Investigations

Radiology. CXR on admission to ICU and daily. Ultrasound or Doppler of hepatic artery, hepatic veins and portal vein daily in ICU.

Haematology. FBE, differential and coags on admission, then 6 hourly in ICU (x1-2/day in ward); antithrombin III, protein C&S daily (at 9 am, 2 citrate tubes, sent to Alfred lab); weekly serum folate and B12.

Biochemistry. On admission to ICU: U+E, glucose, lactate, blood gas, LFTs, Ca, PO4, Mg, NH3; 4-6 hrly in ICU: U+E, glucose, lactate, blood gas; daily in ICU: LFT, Ca, PO4, Mg, amylase,

NH3. Once or twice daily in ward: U+E, LFTs, Ca, PO4, Mg.
Tacrolimus (1ml EDTA blood) or cyclosporin level daily
predose; aim for tacrolimus level 10-15mg/L.

Microbiology. Daily in ICU: bile, drain fluid, sputum for M+C and
fungi, urine for M+C, blood culture.

CMV. Saliva swab in viral transport medium, urine and blood:
weekly for 6 wk, then 2wkly for 3 months. CMV antigenemia
and PCR weekly.

Liver Biopsy. Performed only when indicated.

TRANSPLANT – IMMUNOSUPPRESSIVE DRUGS

The following table outlines the commonly used immunosuppressants likely to be encountered in the ICU during the acute post transplantation phase of management of solid organ transplants, particularly liver and heart transplants.

Confirm all immunosuppressant drug doses from the official intranet protocols

[Liver Transplant Post-op Management](#)

[Cardiac Transplant](#)

Drug	Sample Dosing	Target levels	Toxicities
Calcineurin inhibitors: <i>Reduces effector (killer) T-cells. Leaves sufficient immune activity to combat infection</i>			
Cyclosporin	Heart: IV:1 mg/kg BD then Oral 2.5mg/kg BD titrated to keep target 12-hr trough levels. In PICU: Daily mane level (and give)	Heart: Day 1: 100 ng/mL (lower if AKI) end week 1-3: 300-400 ng/mL 3-12 months: 200-300 ng/mL >12 months: 100-150 ng/mL Diltiazem used to improve pharmacokinetics	Nephrotoxicity Hypertension Dyslipidaemia Low K & Mg Hyperuricemia Neurotoxicity
Tacrolimus	0.1 mg/kg/dose 12 hourly oral/NG. titrated to keep target 12-hr trough	Liver: <3m post 10-15g/L 3-12m post 8-12g/L >12m post 5-10g/L	Nephrotoxicity Hypertension Hyperglycaemia Dyslipidaemia Hyperkalaemia

	levels. In PICU: Daily mane level (and give)	Heart: <6m post: 9-13ng/ml >6m post: 7-9ng/ml	Low Mg Neurotoxicity
Cell cycle agents (Purine/Pyrimidine synthesis) <i>Reduced B and T lymphocytes as well as monocytes in the blood</i>			
Azathioprine	1.5 mg/kg/dose nocte oral/NG	Dose alteration/ withheld if cytopaenia or pancreatitis	Cytopaenias Hepatitis (rare) Pancreatitis Malignancy
Mycophenolate mofetil	IV or oral 600mg/m ² /d ose BD	Dose alteration/ withheld if WCC<4 x10 ⁹ /L OR Neut <1x10 ⁹ /L IV and oral bioequivalence	GI Leukopenia
Proliferation signal inhibitors (MTOR inhibitors) <i>Inhibit T cell activation via a different pathway to Calcineurin inhibitors Not commonly used immediately post transplant due to poor wound healing</i>			
Sirolimus	3 mg/m ² stat then 1mg/m ² daily oral,		Poor wound healing Oral ulcerations Dyslipidaemia Oedema Lung toxicity Pleural effusions Cytopaenias Nephrotoxicity
Everolimus	Dependent	Heart:	Poor wound

	on trough Blood levels Usual Adult dose 1.5mg-3mg BD	No cyclosporin No Tacro: 8- 12ug/l With cyclosporin With Tacro: 3- 8ug/l Levels after 4 days of dose Δ	healing Acne Mouth ulcers Pleural effusions
Corticosteroids			
Methylpredni solone	Heart: starting 10mg/kg/dose BD Liver:: starting 2.5mg/kg/dose (max 25mg) IV 6 hourly tapering to 1mg/kg/dose (max 10mg) IV 12 hourly. Then:	None	Weight gain Hypertension Hyperlipidaemia Osteopenia Hyperglycaemia Poor healing Salt/water retention Prox. myopathy Cataracts Peptic ulcers Poor growth
Prednisolone	Start prednisolone 1mg/kg/dose (max 10mg) daily oral/NG, weaning from week 4.		
Monoclonal Antibody			
Anti- thymocyte globulin Rabbit.	Initial Dose 1- 1.5mg/kg/day	For severe rejection or highly sensitised patients	Fever Rigors Allergic reactions

(ATG Thymoglobulin)	Treatment of Rejection Up to 2.5mg/kg/day Up to 14 days	Discontinue or modify dose if platelet count is less than 50x10 ⁹ /L	Low platelets
Anti-thymocyte globulin Rabbit (ATG Fresenius)	3-5mg/kg/day IV Up to 14 days		
Rituximab humoral rejection.	375mg/m ² Given weekly for 4 weeks		Rash Fever Chills Headache Chest Pain Dyspnoea Hypertension/ Hypotension

TRAUMA

The doctor who receives the first call from the Ambulance Service about a major trauma should obtain the following details: age, what happened, obvious injuries, BP, conscious state, best movement, pupils, saturation or central colour, intubated, ventilation, IV fluids, and estimated time of arrival (ETA).

For all cases of major trauma, the trauma team should be activated, which will notify the ICU AUM, ICU consultant, Emergency Dept registrar, General Surgery registrar (or consultant), radiographer, blood bank, and Neurosurgery registrar (if appropriate). The trauma team is activated from the emergency department by the ED AUM or ED resuscitation consultant. Notification should not be too early (eg don't tell the neurosurgeon at 3am about a patient arriving at 6am).

A senior PICU doctor (fellow or senior registrar) should attend all ED trauma calls, to assist with airway management and provide specialized guidance on overall management.

Role Allocation:

Team leader (TL): Usually ED resus consultant. Allocate roles and responsibilities according to skill/experience; perform trauma team activation; Ensure PPE including lead worn by all team members; pre-arrange radiographer, order xrays and discuss CT; contact blood bank for blood in motion/MTP; obtain pre-hospital handover and involve PETS consultant if transport not being undertaken by MICA; allocate family support; contact other units (eg Neurosurgery, orthopaedics) as needed; formally handover to appropriate medical team member at the appropriate time and place.

***Team leader support:** ICU registrar role if anaesthetics managing airway: Discuss management and offer advice to TL; Liaise with specialty units; Assist with appropriate investigation ordering and review results; Liaise with family to gain further info; ensure blood bank aware; consider longer term requirements of care.

***Airway doctor:** ICU registrar or anaesthetic registrar. Introduce self. Assess and manage airway and breathing. Pre-arrival: PPE (glove, gown, lead, goggles), airway equipment and RSI drugs with nurse/TL, airway plan A, B, C, D discussed with nurse/TL ; Post arrival: control log roll, patslide and transfer; apply oxygen, airway adjuncts and assess/perform intubation if required, in discussion with TL; cervical immobilization all times. Monitor CNS and update TL of any changes; post intubation: orogastric, ETT secure. Adjust ventilator to target normoxia and

normocapnoea with safe ventilation parameters; ensure appropriate sedation; accompany intubated patient to destination (theatre/PICU via CT).

Assessment doctor: Pre-arrival: PPE; ultrasound; discuss anticipated injuries with TL ; On arrival: assists transfer; A,B – vitals, detect thoracic threats; C – vitals, assess for massive haemorrhage, abdo/pelvis (Binder with Procedure Dr), limbs, extended FAST (if appropriate); D– assess neurology, check temp and keep warm, BSL. Post arrival: secondary survey. +/- procedures. Request x-rays and follow up.

Procedure doctor: . Pre-arrival: PPE; Prepare 2 large bore IV, rapid infuser, IO +/-pelvic binder +/- chest drain; On arrival: assists transfer; IV access x2, order and take blood for path; chest decompression. Post arrival: Aid airway Dr if required; ICC; pelvic binder (with assessment Dr); call Blood Bank for blood.

Nurse team leader: Pre-arrival: PPE; trauma callout; nursing role allocation. On arrival: Logistics; Organise additional staff/ PSA/ Blood bank/ Equipment/ SW; Bed management – OT/CT/PICU; family coordination.

Scribe: Pre-arrival: PPE; Identify to role; Documentation in room; Document team members and allocated roles. On arrival: Document: Time, MIST handover from ambulance, staff present and roles, 1⁰ and 2⁰ survey, physical findings, fluids/drugs/blood administration, injuries, interventions and procedures; Monitor response to therapies; Activate MTP on instruction; record regular vitals, GCS/AVPU; document transfer time and location.

Airway Nurse: Senior nurse, PICU/ED. Pre-arrival: PPE; clarify airway plan with airway Dr and TL; pre-intubation checklist; set-up for intubation; glidescope in room; clarify intubation drugs with Airway Dr and TL; set-up ventilator with settings per airway doctor and TL; ensure thoracic elevation in place (children <8) On arrival: assist with log roll transfer; cervical spine immobilization with appropriate collar; assist intubation and airway Mx; insert orogastric, confirm gastric aspirate; accompany in transport.

Circulation nurse(s) (roles allocated depending on need)

Procedure: Pre-arrival: PPE; special procedure trolley; prepare IV fluid/access/IO On arrival: assist log roll; assist pelvic binder, chest drain, IDC, splinting; apply pressure pads if exsanguinating; sets up art line/CVC. **Infusion:** Pre-arrival: PPE prime level 1 rapid infuser; call blood bank On arrival: assist log roll; manage rapid infuser, blood administration including checks. Liaise with blood bank, TL and scribe. **Infusion** Pre-arrival: PPE; prepare requested RSI drugs & analgesia; prepare infusion and bolus sedation; trauma shears, patient property, warm blanket and Bear hugger. Ensure transport ready (transport bag, drug

box, monitor). **On arrival:** start clock, assist log roll transfer; remove clothing, apply warm blankets; attach monitors and obtain 1st vitals; assist IV access collecting and labelling bloods; confirm and prepare correct medication doses; chest compressions in arrest

Radiographer: Preload plates for imaging; trauma series (CXR, lateral C-spine, +/- pelvis); additional imaging after secondary survey; remain with trauma team until no longer required. Liaise with CT radiographer.

Social work: greet family and escort to designated area for ongoing support; psychosocial history; provide information package; Follow up next day. If patient dies, or is DOA, SW present during family meeting, assist with contacting family, friends, clergy; support funeral arrangements, autopsy, organ donation etc; support family during notification to coroner, identifying body to police etc.

*Likely ICU doctor roles.

Helicopter reception. In most cases the flight paramedics will transfer the patient from the helipad to ED assisted by RCH PSAs. In the unlikely event that the child is too unstable, a MET call will be called to the helipad reception area: Both Emergency Department team (nurse and doctor) and ICU team (nurse and doctor) should attend. ED team brings an oxygen cylinder with Twin-O-Vac and tubing for oxygen and suction. RCH reception teams wait behind the sliding door at the helipad until beckoned.

Principles of primary reception of an injured child:

Airway, Breathing, Circulation, Cervical spine, Disability (GCS), and Exposure (completely undress patient but keep warm).

2 large bore cannulae in upper limb veins.

Insert intra-osseous needle if IV takes more than 90 sec.

Immobilise cervical spine: Aspen + sandbags (see Spinal cord injury)

Take blood for glucose and X-match and any other necessary tests as soon as the IV cannula is inserted.

Cross-match amount depends on body size and amount of bleeding: usually at least 4 units for a school-age child.

Persistent tachycardia may represent inadequate resuscitation, occult bleeding, intrathoracic injuries (pneumothorax/pericardial tamponade) or inadequate analgesia/sedation.

Arrange FFP and platelets if anticipate loss > 40 ml/kg (activate massive transfusion protocol).

Ensure stable airway, breathing and circulation before transfer to CT, but otherwise ensure no other delays to CT scan (eg insertion of arterial and central lines must not delay CT scan

unless essential for stability).

If head injury suspected or confirmed, defend blood pressure and target normoxia and normocapnoea (CO₂ 35-40, SpO₂ 95-99%) with safe ventilation.

Chest X-ray immediately on arrival (get out of the radiographer's way). X-rays of pelvis, cervical spine, thoracolumbar spine and extremities could wait until CT done if this will delay transfer.

Spine boards are radiolucent, take X-rays through them. Log-roll every 30 min while on spine board.

Patient transfers between trolley, bed and CT scanner require log roll and strict in line spine immobilization, with the airway doctor in control at the child's head.

Log-roll to examine the spine before transferring to CT scan: use correct log-roll technique: Airway doctor looks after head; at least 2 other people (3 others for an older child); one turns the shoulders, one the pelvis, one the legs (overlapping arms, the person minding the head calls the time of turning).

Turn all trauma patients hourly unless a consultant provides written orders to the contrary.

Transfer to CT scan. Portable monitor. Hand-ventilate, or portable ventilator if lungs are abnormal. At minimum, one doctor, one nurse + emergency dept orderly accompany the child to CT. Log roll and inline stabilization for transfers. Don't attempt to place the child's head in the head-rest on the CT scanner (risk of neck movement). Don't use either the ventilator or the anaesthetic machine that is kept in CT (use the portable ventilator, or hand-ventilate).

Radiology. Notify a radiographer within 15min prior to the child's arrival, so that he or she is waiting in ED when child arrives. If child has a head injury, mention to the radiographer that the child will need an urgent head CT scan. This can be done without a consultant radiologist present. Obtain a chest X-ray immediately on arrival (get out of the radiographer's way). X-ray pelvis, cervical/thoracolumbar spine, and extremities shouldn't delay transfer. If the surgical consultant or registrar wants a CT scan of the abdomen, they should notify the consultant radiologist well before the child is transferred to Radiology for head CT. If first chest X-ray shows wide mediastinum, and question of thoracic aortic tear arises, obtain a second chest X-ray in full inspiration and 15° head-up (tilt the whole bed, not just the head end and watch the blood pressure).

Identification. Each child (unless known to be an old RCH patient) is given an Unknown Male or Unknown Female identifier including wrist bands in ED

Notification and consultation. The team leader (or support) notifies the specialty surgical units (eg neurosurgery, orthopaedics, plastics, ophthalmology) of the child's arrival, and notifies theatre and duty anaesthetist as necessary.

Disposal. An ICU patient should return from the CT scanner to ICU (or occasionally, directly to Theatre). Some children with an intracranial haematoma can be extubated in the operating theatre after craniotomy or burr holes, and go directly to the ward from there.

Longer term management. At 0700 every day there will be a trauma huddle to assist with coordination of the early surgical management of any patients admitted within the prior 24 hours. A representative from ICU should attend for any patients admitted to ICU.
Consider MRI to investigate C-spine if unable to clear clinically because of young age, agitated state, sedation, unable to cooperate;
Spinal clearance according to hospital protocol (Orthopaedics unless Neurosurgery managing TBI);
Consider DVT prophylaxis in post-pubertal children;
Tertiary survey to be completed by the Trauma team unless delegated to PICU.

VENTILATION

Start with a tidal volume or PIP that produces visible chest movement, then measure blood gases after 15 minutes and adjust the ventilator according to PaCO₂ and PaO₂.

Pressure-limited ventilation should be used if child <15 kg.

If a child has lung disease, use controlled hypoventilation (unless there is brain injury) and try to keep the FiO₂ <0.5 to avoid iatrogenic lung damage.

Gas exchange is usually better when the good lung is uppermost in infants and children <5 years, and when it is dependent in adults.

Triggered ventilation is more comfortable than un-triggered, and uses lower tidal volumes and airway pressures.

Ensure that the apparatus dead space is minimal.

Use lightweight tubing in small children to avoid accidental extubation. Do not use narrow tubing in patients < 20 kg.

When using volume-cycled ventilation, record the peak pressure.

With pressure-limited ventilation, record tidal volume. Volume-cycled pressure-limited ventilation yields little information about lung compliance.

Respiratory rate per min: neonate 30-60, 6mo 25-30, 1-5yr 20-25, 5-12yr 15-20, >12yr 12-15.

$A-aDO_2 = PAO_2 - PaO_2 = ((P_{bar} - P_{water}) \times FiO_2) - (PaCO_2 / 0.8) - PaO_2$. Usually = $(716 \times FiO_2) - (PaCO_2 / 0.8) - PaO_2$. Child <10 mmHg, adult >15 mmHg, elderly >40 mmHg.

$MAP = ((PIP \times IT) + (PEEP \times ((60/RR) - IT))) / (60/RR)$.

Oxygenation index (OI) = $MAP \times FiO_2 / PaO_2$.

Ventilation index (VI) = $pCO_2 \times RR \times PIP / 1000$.

WEANING

Infants should usually be extubated from a rate of 5, and not have a period of endotracheal CPAP before extubation.

Infants: usually wean by rapid reduction in IMV rate to 5/min (followed by extubation if indicated), rather than a very gradual reduction in assist pressures or IMV rate. **Older children:** a trial of several hours CPAP + PS allows assessment of adequacy of gas exchange pre-extubation.

Children who have been intubated for (say) >3 days, are often extubated to nasal CPAP or High Flow rather than nasal prongs. This is particularly the case for neonates post-cardiac surgery.

AVEA VENTILATOR

Modes:

Control. Ventilator delivers set pressure or tidal volume at a set inspiratory flow and rate. If assist sensitivity set to off, patient

prohibited from triggering breaths between the machine breaths. Suitable only for paralysed patients.

Assist-control. A machine breath of pre-set pressure or tidal volume given if patient does not trigger a breath within a time determined by the set rate. If patient effort is sufficient to trigger during expiration, after expiration has finished an assist breath given. Breath period is reset when the patient trigger is detected.

SIMV. Machine-generated breath only delivered if there is no patient inspiratory effort at or above the preset sensitivity level (trigger) in the previous breath period. The first patient trigger during a breath period results in an assist breath (pre-set volume or pressure). If there are additional patient triggers in the breath period, pressure support is given (or no assistance if PS is off). Breath period is not reset when the patient initiates a breath.

CPAP. Patient initiates all breaths. If PS activated, triggers assist breaths. Only for spontaneously breathing patients.

APRV-Biphasic. Airway pressure release ventilation. Similar to BiPAP. Patient can breathe spontaneously at two pressure levels. Adjust time high and low, pressure high and low, and pressure support.

Five breath types

TCPL (time cycled, pressure limited). Ventilator delivers set inspiratory flow to reach set inspiratory pressure for set time (i-time). No volume guarantee; tidal volume varies with changes in compliance and resistance in patient's airway.

Pressure control. Ventilator regulates inspir flow to maintain set pressure for set period (difference to TCPL is that rate is set, rather than the i-time). No volume guarantee.

Volume control. Set tidal volume, breath rate and flow rate. Pressures vary with lung compliance and resistance.

Volume assured pressure support. Combines pressure support with tidal volume guarantee. Set mode (AC or SIMV), set breath rate, set sensitivity, set insp pressure above PEEP. Turn dial to VAPS, adjust tidal volume. Set high peak insp press alarm 5-8 cmH₂O above insp press. Breath will terminate when maximal tidal volume delivered.

Pressure support. Breaths always initiated by the patient. Set assist sensitivity. Set PS level (above PEEP).

Triggering

Pressure triggering. 1-20cmH₂O.

Flow triggering. Infant flow sensor detects 0.2-3.0 L/min; placed between patient Y and ETT. Paediatric flow sensor detects 1-5 L/min; placed between exhalation valve and patient breathing circuit (all modes except TCPL).

Termination sensitivity

Active only for TCPL breaths. With termination sensitivity on, inspiratn terminated at 5-25% of peak flow, or time cycled

(breath rate based on set I time), whichever first.

Cycling: the variable that terminates inspiration (pressure, volume, flow or time cycling)

Flow cycle. (In Advanced Settings) Terminates TCPL or pressure support breath when flow falls to set % of initial flow. Start with 5%

Volume limit. Terminates a pressure-limited breath when tidal volume reaches a set value.

Inspiratory rise. Determines the slope of pressure rise during a mandatory breath (not active in TCPL).

Bias flow. Sets the gas flow in the tubing between breaths.

Pressure trigger. Pressure below PEEP which triggers a mandatory or pressure-supported breath. Instead of flow trigger.

HIGH FREQUENCY OSCILLATORY VENTILATION

Infant: MAP 18-25, 8-12 Hz, delta-P 30-40 cmH₂O, IT 33%. Aim for wiggle down to upper thigh.

Child: MAP 20-30, 6-8 Hz, delta-P 40-60, IT 33%

Adolescent-adult: MAP 25-40, 4-6 Hz, delta-P 50-90, IT 33%

SpO₂ 80-85% usually adequate. To improve oxygenation, increase MAP or FiO₂. Aim for FiO₂ <0.5. Use chest X-ray to adjust MAP (avoid under or over inflation).

To improve ventilation (lower pCO₂), reduce frequency (Hz) or increase amplitude (delta-P). Frequency <5Hz unusual.

ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)

Sedate with morphine, and midazolam or diazepam. Try patient-triggered ventilation first, paralyse if gases poor. If gases still poor, try HFOV. Do BAL, start antibiotics. Insert central and arterial lines. Echo to assess cardiac function and pulmonary hypertension.

Start with low tidal volumes of 6-8ml/kg, peak pressure <30cm, PEEP 8-10cm, FiO₂ <0.5.

If no brain injury, accept pCO₂ 60-80mmHg and pH > 7.2 unless this causes cardiac failure from pulmonary hypertension.

SpO₂ 80-85% will be adequate for most patients.

Recruit collapsed alveoli using 15-30sec inflation 5-10cm above PEEP; clamp ETT when reconnecting to ventilator.

Consider prone positioning for 4 hours at a time to recruit posterior lung segments.

Consider adrenaline infusion 0.03-0.1 mcg/kg/min to improve pulmonary blood flow.

Consider trial of NO (stop after 30min if no measured response).

Give enteric feeds (jejunal tube often needed) or TPN.

Haemofilter if oliguria >24hr, high K, or creatinine >0.4.

WARD ROUND CHECKLIST

The following items should be discussed for every patient as part of the ward round to ensure quality care and communication with team members:

Airway

- ETT – Can it be secured better, position on x-ray?

Breathing

- Review ventilator settings, alarms
- CXR: ETT, lines, & drains positioned correctly?
- Ventilation: ready for weaning or extubation?
- Head of bed elevated

Circulation

- Review inotropes
- Lactate and SvO₂ reviewed

Pain and sedation

- Goal for level of analgesia & sedation
- Comfort B Score
- Appropriate prescriptions, prn drugs
- Documented weaning plan

Fluids and nutrition

- Calculate TFI
- Calculate glucose intake (mg/kg/min)
- Plan for nutrition, calories
- Bowels working: lactulose, movicol

Infection

- Infection markers (IT ratio, PCT) and culture results reviewed
 - Antibiotics: appropriate to likely infection, review or cease date documented, can the antibiotics be ceased or scaled down?
- CVAD, IDC or surgical drains still required?
If not, remove

Document targets for:

- Vital signs
- Blood gases, electrolytes & glucose

Pressure areas, IV sites

Stress ulcer prophylaxis

DVT prophylaxis

Discharge preparation

Communication / referrals (pain service, allied health, medical and surgical teams)

Eligible for research project enrolment

Address any nursing concerns

Update parents

