Barts Health NHS
NHS Trust

Acute Critical Care Unit
Royal London Hospital
Supplementary Induction Booklet

Reviewed 2022/2023

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Forward:

Starting a rotation on the Critical Care Unit, looking after the sickest patients in the hospital can be daunting, challenging and overwhelming.

We hope that this supplementary booklet written by trainees for trainees will provide an introduction to key critical care topics and be a reference guide. This supplementary booklet compliments the current ACCU trainees guide and focuses on areas of practice that are specific to Intensive Care Medicine at the Royal London Hospital: monitoring, organ support, troubleshooting and knowing when to call for help.

We hope that this makes your journey and training on the ACCU a little easier.

I'd like to thank each of my colleagues Dr Caroline Clapham, Dr Steve Cole, Dr Alex Fowler Dr Ryan Haines and Dr Sachin Mehta for helping me to put together this booklet.

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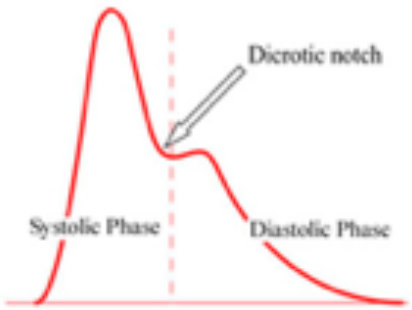
Section 1: Monitoring - Invasive blood pressure

Indications:

- Continuous BP monitoring (e.g. patient on vasoactive agent)
- Invasive cardiovascular monitoring (e.g. LidCo)
- Need for frequent blood sampling - ABGs/blood tests



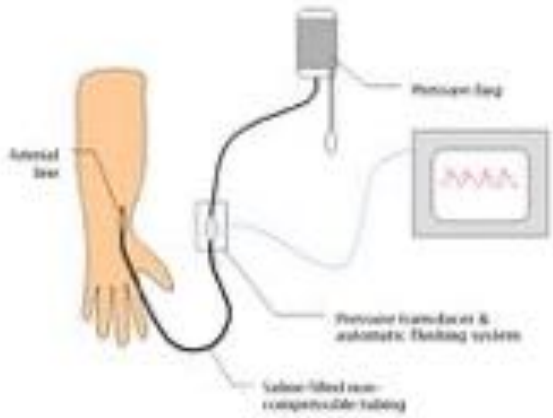
Normal arterial pulsation wave form.

Complications

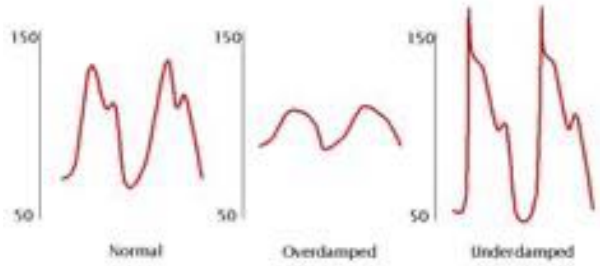
1. Bleeding – may have some leaking around the line site, especially in coagulopathic patients. Apply direct pressure with the line still in; can put some gauze under fresh dressing, ensure nursing staff monitor carefully.
2. Vasospasm - This can happen either during insertion, if blood is aspirated too quickly or sporadically, will lead to damped trace. **Important to monitor limb below (especially with brachial lines) and consider removal.**
3. Arterial occlusion
    a. If there is evidence of tissue ischemia below line (prolonged CRT, etc.), **this needs to be removed ASAP and discussed with the vascular team if it doesn't resolve rapidly** [worth comparing ischemic limb to the other as the patient may just be very shut down].
    b. Embolic disease also a risk; monitor for signs e.g. splinter haemorrhages in hands
4. 'Reading low'
    a. Check this isn't the actual blood pressure – Clinical assessment and do non-invasive BP
    b. As per 'no waveform' below
5. Pseudoaneurysm
    a. More likely to detect once out; needs vascular review.

Malfunctioning

1. No waveform – Check the set starting at the patient and ending at the monitor
    a. Clinical assessment & check line still in patient
    b. Check aspirating & give good flush
    c. Check transducer height
    d. Ensure pressure bag sufficiently inflated.
    e. Zero the line ('off to patient' → zero on the monitor)
    f. Check monitor cable attached (often worth changing monitor cable)



    g. May be asked to re-wire (passing a guide wire down the lumen of the line & passing new line; this rarely works)
2. Damped waveform
    a. Just describes how much the waveform has been damped; most important thing to know is that the MAP is most accurate to use if concerned line is over/under damped.
    b. Causes of overdamping: air bubbles, kinks, blood clots, vasospasm
    c. Causes of underdamping: Equipment artefact, dysrhythmia, hypothermia



3. Unable to aspirate
    a. If no need for regular ABGs & functioning for BP can just leave
    b. Check if able to flush with the built in flush system
    c. Use 20ml syringe to aspirate
    d. Un-suture and slowly withdraw while aspirating

Further reading

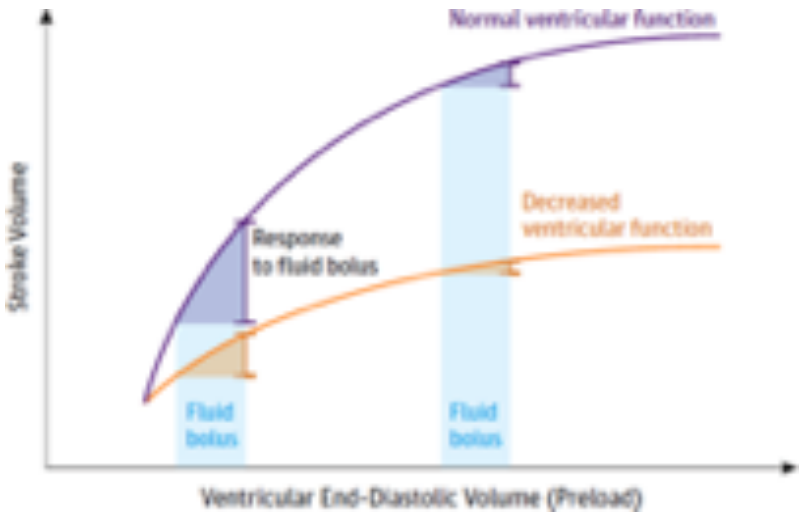
Matthew Ward, Jeremy A Langton; Blood pressure measurement, *Continuing Education in Anaesthesia Critical Care & Pain*, Volume 7, Issue 4, 1 August 2007, Pages 122–126.

Section 1: Monitoring – Central Venous monitoring

A central venous catheter (CVC) inserted via *Seldinger* technique into either the internal jugular, femoral or subclavian veins is used widely in ICU for:

- Invasive haemodynamic monitoring.
- Infusion of drugs that can cause damage to smaller veins such as noradrenaline.
- Emergency access when other veins cannot be used (NB rate of flow is inversely proportional to length of cannula; therefore resuscitation of blood products through a 20cm long CVC is *slower* than through an 18G 4cm peripheral cannula. Distal lumen (brown) on the CVC is the best for emergency fluid/blood administration.
- Access for specific treatments including; pacing wires, renal replacement therapy (a dedicated line), exchange transfusions, plasma exchange and total parenteral nutrition.

Central Venous Pressure (CVP): once used to guide fluid therapy in resuscitation, less importance is placed on CVP values now; however this form of invasive haemodynamic monitoring has some uses. It measures right atrial pressure which approximates to right ventricular end diastolic pressure which is related to ventricular end diastolic volume and therefore *ventricular preload.* Thus, during a fluid bolus, if CVP increases, this may result in increasing preload and a "fluid responsive" patient. On the other hand, if CVP remains the same then fluid may not help a "fluid unresponsive patient". High CVP may indicate fluid overload, which is increasingly associated with worse outcomes in ICU patients.



Central Venous Saturations (ScVO2)*:* If tissue blood flow is inadequate to meet oxygen demand more oxygen will be extracted from the blood by the tissues. This will lead to a fall in the oxygen saturation of blood returning to the heart. Sampling of blood from the right atrium (often where the tip of CVC lies) provides us with a central venous saturation. In patients with severe sepsis or septic shock early in resuscitation targeted to achieve a central venous saturation of greater than 70% may be associated with better outcomes.

Section 1: Monitoring – Intracranial Pressure

Overview

- Intracranial Pressure (ICP) monitoring is a core physiological monitor used in Traumatic Brain Injury (TBI)
- Cerebral Perfusion Pressure (CPP) is related to ICP (CPP = MAP - ICP)
- A core element of "Neuroprotective Care" is to maintain CPP >60 mmHg (and ICP <20 mmHg)
- The British Trauma Foundation (BTF) guidelines consider ICP monitoring mandatory for severe TBI with an abnormal CT (as intracranial hypertension develops in 60% of these cases)
- There is however currently no evidence of benefit from ICP monitoring in TBI
- The most common ICP monitors used are the "bolt" (transducer is placed in extradural/subdural space or in the parenchyma/CSF) and the External Ventricular Drain (see *"EVD Management and Troubleshooting"*)

Indications

- BTF Guidelines list the following indications:
    - Moderate to severe head injury who can't be serially neurologically assessed
    - Severe head injury (GCS < 8) + abnormal CT scan
    - Severe head injury (GCS < 8) + normal CT if 2 of the following are present:
        - *Age > 40 yrs, (2) SBP < 90mmHg, (3) Abnormal motor posturing*

Advantages

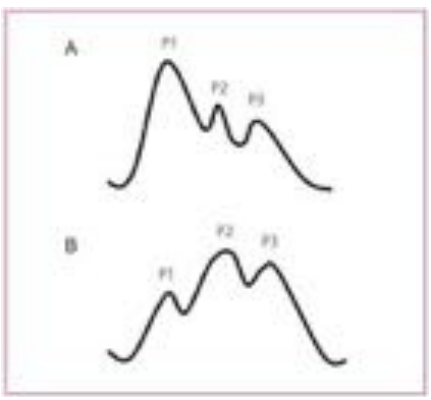
- Useful for CPP guided therapy
- Alerts clinicians to changes in the unconscious patient
- Easy to measure
- Number and waveform to evaluate
- Continuous measure

Disadvantages

- Invasive
- Intracranial bleeding (higher risk with EVD)
- Infections (higher risk with EVD)
- Malfunction/measurement error (may lead to over investigation)
- May be misleading (not a global measurement)
- No evidence of improvement in clinical outcomes
- Individual ICP monitors have specific limitations:
- Bolt: cannot calibrate, subject to drift, cannot drain CSF (for sampling/reducing ICP)
- EVD: requires expertise and resource availability for placement

ICP waveform

- An ICP >20 warrants urgent senior medical review (see *"Troubleshooting ICP"*)
- The ICP waveform can give additional information
- There are 3 distinct peaks:



    - P1: arterial pulse
    - P2: vaguely related to cerebral compliance
    - P3: aortic valve closure (dicrotic notch)
- Figure A demonstrates a normal ICP waveform
- Figure B demonstrates reduced cerebral compliance (P2>P1) and warrants senior medical review

Red flag (call 1113/45715): *An ICP > 20mmHg warrants an urgent senior medial review*

Remember that if the ICP bolt is working and there is an apparent spike in ICP, then do not ignore this, it is likely to be an actual real value. ICP takes a long time to fall spontaneously. You will notice that ICP will fall by increasing the angle of the head of the bed, ICP changes are consistent with CT findings and consistent with changes in motor scores.

Further reading

Kyle Pattinson, Guy Wynne-Jones, Christopher HE Imray; Monitoring intracranial pressure, perfusion and metabolism, *Continuing Education in Anaesthesia Critical Care & Pain*, Volume 5, Issue 4, 1 August 2005, Pages 130–133.

Section 1: Monitoring - Cardiac Output

LiDCO / LiDCO rapid

Cardiac output, unlike blood pressure, is more difficult to measure directly although we are able to use several devices to determine an estimate, including LiDCO and the Oesophageal Doppler probe. The goal of measurement and optimisation of cardiac output is ultimately to improve tissue oxygenation.

LiDCO (which stands for *Li*thium *D*ilution *C*ardiac *O*utput), uses an existing arterial line to analyse the pressure wave. An algorithm uses the 'pulse power' to compute a cardiac output and consequently changes in the shape and area under the pressure wave translate into changes in cardiac output. Accuracy is subject to the quality of the arterial line and the characteristics of the blood vessel in which it is inserted. To calibrate the measurements, Lithium Chloride is injected into an internal jugular CVC and this is in turn detected by a device connected to the arterial line, to determine concentration vs time. In LiDCO rapid, this step is omitted (so it can be used for patients without a central line) but as a consequence values are derived from estimates. Lithium calibration is not possible if there has been recent administration of muscle relaxant.

A typical approach might be to ask for LiDCO in a patient with hypotension of uncertain aetiology, or a patient in whom inotrope/vasopressor requirements are escalating. Several values will be given.

Firstly hypovolaemia should be corrected. The "stroke volume variation", if >10%, suggests that a fluid challenge may be helpful. If, after administration of e.g. 250 mL plasmalyte, there is an increase in stroke volume of >10%, then this is a positive result and a repeat fluid challenge should be considered.

Once fluid status is corrected, a low cardiac index (generally <2.2 L/min/m^2) suggests inotropy is required (eg dobutamine / adrenaline) whereas and adequate or high cardiac index coupled with a low systemic vascular resistance index (<1700 dyn.sec.cm^{-5}.m^2) suggests vasoconstriction is required (e.g. noradrenaline).

Always correlate the values with what you expect clinically. The values are only as good as the arterial line, and note that LiDCO is designed for intubated patients with mandatory ventilation at a tidal volume ~8 mL/kg (rarely the case in practice). Also, if clinically the patient is perfused with a good blood pressure, there is no need to fluid challenge simply because the device suggests to!

Further reading

Drummond K, Murphy E. Minimally invasive cardiac output monitors. Continuing Education in Anaesthesia Critical Care & Pain, vol 12, issue 1, 1 Feb 2012.

Section 1: Monitoring – Continuous EEG monitoring (Brainz)

Continuous EEG monitoring is an emerging technique that is not yet widely used. It is set up at the bed space by the critical care technologist, 3 electrodes are attached to the scalp (bi-parietal and one in the midline).

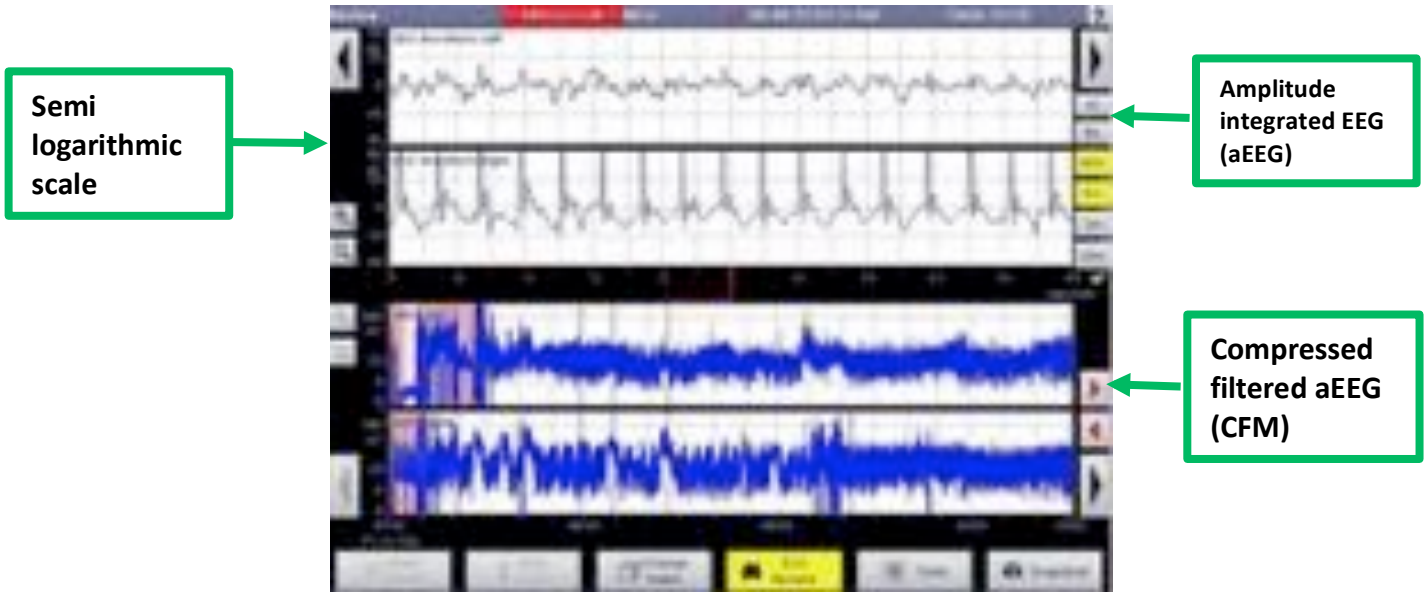
Indications:

- Suspected seizure activity: high risk of non-convulsive status epilepticus (NCSE)/monitoring/response to treatment
- Monitor sedation
- Patients with coma
- Post cardiac arrest
- Hypoxic ischaemic brain injury
- Metabolic encephalopathy
- To achieve burst suppression as part of a neuro-protective strategy in the management of high ICPs in TBI.

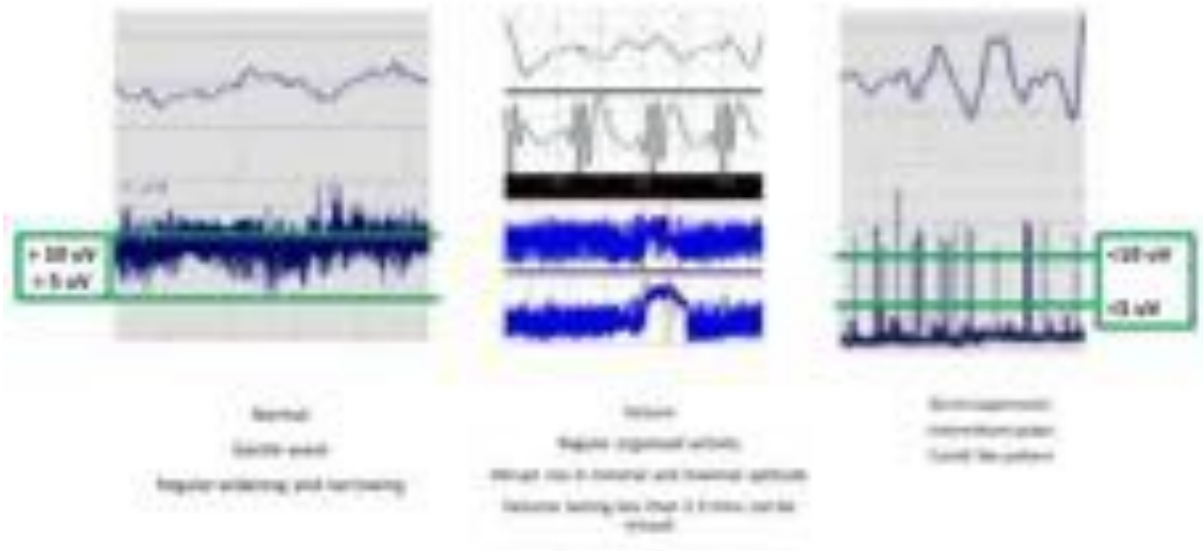
There are two features of the trace to look at:

1. Amplitude (deviation of the trace on the vertical axis)
2. Presence of seizure activity

The Brainz monitor displays two traces (one from each parietal lobe electrode) The record will be a dense trace and the width of the waveform will vary with normal brain activity:



Common continuous EEG monitoring patterns:



Burst suppression:

- Severely abnormal trace
- Characterised by general suppression of amplitude (narrow and low voltage)
- Intermittent bursts spikes of activity may seen
- Seen in encephalopathy and coma
- Coma can be induced with thiopentone (a barbiturate) as a neuro-protective strategy for high ICPs in TBI (see neuro-protection section)
- Often accompanied by seizure activity

Section 2: Organ support – Invasive Ventilation

Invasive ventilation could be described more thoroughly as invasive, positive-pressure, mechanical ventilation. It represents the process of gas being driven (positive-pressure) into the lungs (ventilation) via a definitive airway device (invasive) by a machine (mechanical). Invasive ventilation or some combination of the terms above are used interchangeably (confusingly!) to mean the same thing. Here, we will stick to using the term invasive ventilation. Understanding the underlying concepts is paramount at this stage. The various terminology will become clearer through your time in the ICU.

Aims of invasive ventilation

Patients are kept sedated and invasively ventilated for a number of reasons. In each case the objectives of invasive ventilation will always include the provision of adequate gas exchange (exchange of oxygen and carbon dioxide between inspired gas and blood in the alveoli).

Physiology

Gas exchange is governed by:

- **Minute Ventilation (MV):** the volume of gas expired from the lungs each minute. MV equals Tidal Volume (TV) multiplied by the Respiratory Rate (RR): *MV = TV x RR*. MV is proportional to CO_2 clearance and can therefore be manipulated by varying the depth and/or rate of breathing. (N.B. oxygenation is much less dependent on MV)
- **Alveolar Ventilation (AV):** The volume of gas reaching perfused alveoli each minute. It is even more closely related to CO_2 clearance as it describes the volume of gas actually taking part in gas exchange. Some inspired gas will not take part in gas exchange and is known as physiological deadspace (VD). Some gas remains in the airways (anatomical deadspace) and some will reach alveoli that are not perfused and therefore don't take part in gas exchange (alveolar dead space). *AV = (TV - VD) x RR.*
- **Ventilation/Perfusion (V:Q) matching:** The distribution of ventilation (V) and blood flow (Q) is closely matched (V:Q matching) throughout the lung, minimising physiological deadspace and maximising the efficiency of CO_2 clearance and oxygenation. Increased physiological deadspace is a type of V/Q mismatch. The other is known as "shunting": De-oxygenated, venous blood perfuses non-ventilated alveoli and passes back in to the systemic, arterial circulation still de-oxygenated. Increased shunting will proportionally decrease the oxygen content of arterial blood.
- Invasive ventilation should focus on maintaining 'normal lung physiology' by keeping alveoli open and ensuring adequate pulmonary blood flow. This is largely achieved by selecting the appropriate mode and settings of ventilation. Other important factors such as adequate position, sedation, etc. are covered in *'Ventilation Troubleshooting'*.

Modes of invasive ventilation

Terminology describing modes of ventilation is again confusing. To start with, focus on asking the following questions:

- How is the breath delivered: a preset pressure, or a preset volume?
- Are the breaths delivered at a set frequency (controlled mode), in response to patient's respiratory efforts (spontaneous mode) or a combination of both (assist-control or spontaneous-assisted)?

Volume Controlled (VC): set the ventilator to give a breath of a certain TV over a certain time and at a set frequency (RR). The *pressure* in the airways will be determined by how stretchy or "compliant" the lungs and chest wall are.

Pressure Controlled (PC): The ventilator is set to a certain pressure for a certain period of time and at a set frequency. The *volume* delivered will depend upon how compliant the lungs and chest wall are.

Pressure Support (PS): The patient's spontaneous breath is assisted with a preset positive pressure from the ventilator. This is a spontaneous mode and the patient must have a normal respiratory drive as no breaths will be delivered if the patient stops breathing.

Mixed modes: These are controlled modes of ventilation with the ability to allow/assist patients' spontaneous breaths. They can be used when transitioning between controlled and assisted modes of ventilation and when patients fail to synchronise with either mode. They include Assist Control (AC) (pressure or volume), Synchronised Intermittent Mandatory Ventilation (SIMV) and BiLevel Ventilation.

Settings:

Ventilator settings are titrated to the patient's likely "baseline parameters", underlying pathology, vital signs, blood gases and more complex ventilator measurements (e.g. compliance curves, plateau pressures, auto-PEEP). A balance must also be struck between achieving certain parameters and minimising the damage/side effects of invasive ventilation.

Initial settings for an 'average adult' for mandatory ventilation include:

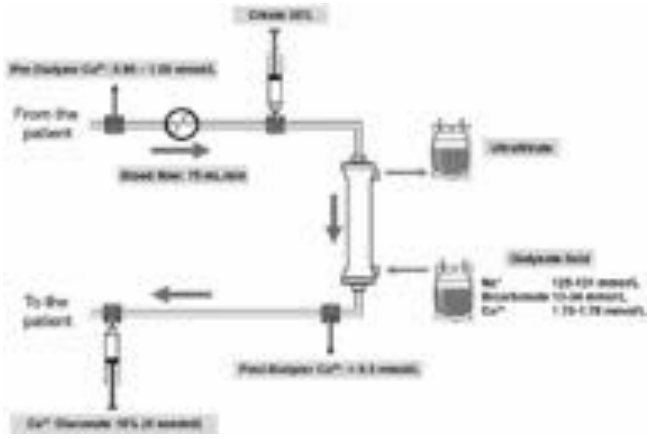
- Positive End Expiratory Pressure (PEEP): 5cmH_2O
- RR: 14-16
- TV: 6-8mL/kg (based on ideal bodyweight)
- Inspiratory to expiratory time ratio (I:E ratio): 1:2
- Fraction of inspired oxygen (FiO2): start high and reduce depending on monitored SaO_2

Further reading

www.ics.ac.uk/ICS/handbooks.aspx. – Ventilation handbook available as pdf file.

Section 2: Organ support – Renal Replacement Therapy

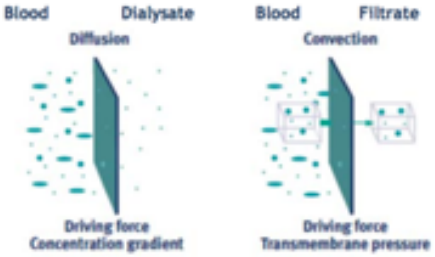
Treatment of established AKI is largely supportive. Severe AKI will require some replacement of kidney function. Renal replacement therapy (RRT) is not without complications and may have a substantial impact on drug pharmacology. RRT is the artificial replacement of some kidney functions: solute control (urea, creatinine, potassium, phosphate, acids etc.) and fluid balance. RRT is a supportive and is not a cure. RRT might also be used in some forms of poisoning, hence sometime referred to as "blood purification". All forms of RRT, apart from peritoneal dialysis, require an extracorporeal circuit. A typical RRT circuit is depicted below.



Patient's blood is drawn out through a double lumen catheter inserted in a central vein. This is made possible by the action of a roller pump ("blood pump"), which generates a negative pressure in the proximal part of the circuit. Blood is then circulated through a haemofilter where exchanges take place. Blood is finally returned to the patient through the return line.

Basic exchange principles:

Diffusion along a concentration gradient (Haemodialysis) vs Convection using solute drag (Haemofiltration)



When to start:

- Refractory hyperkalemia
- Fluid overload - the most common indication
- Several severe poisonings (e.g. Metformin, Lithium, Ethylene glycol)
- Metabolic acidosis with a pH < 7.15
- Complications of hyperuricaemia often when urea exceeds 40-50 mmol/L, rarely sole indication for RRT

When the decision to start CRRT is taken, the next step is to prescribe the machine settings which determine the safety and efficacy of RRT treatment.

The following parameters should be prescribed:

- The **dose** of exchange (sum of all effluent fluids) – usually 20-35 ml/kg/hr
- The ratio of pre- and post- filter **dilution** (standard 70:30)
- **Blood flow** rate (50-250ml/min)
- **Anticoagulation**:
    - 1^{st} choice citrate, 2^{nd} Choice heparin, 3^{rd} Choice none/Epoprostenol (flolan)
    - Blood circulation through non-biological materials, leads to an activation of platelets and coagulation pathways and formation of micro-thrombi that impair blood flow and result in circuit failure. To maximise circuit lifespan, effective anticoagulation is therefore required.
- **Fluid balance target and fluid removal rate** (up to 400ml/hr)

Further reading

Follow this link for more info and questions: bit.ly/2Jl2Yvc

Lisa Gemmell, Robert Docking, Euan Black; Renal replacement therapy in critical care, BJA Education, Volume 17, Issue 3, 1 March 2017, Pages 88–93.

Section 2: Organ support – Vasoactive drugs

Key principles

Organ support in critical care requires maintenance of adequate perfusion pressure and sufficient cardiac output. Cardiac output itself depends on preload, contractility and afterload. There are several therapies available in critical care to optimise these parameters, and is important to consider the cause of hypotension/shock to determine the best treatment. Hypotension can itself be broadly classified into four groups of causes:

- Hypovolaemic: restoration of circulating volume is required
- Poor contractility: optimisation of LV/RV function +- inotropy is required
- Obstructive (PE, tension PTX, tamponade): treatment of cause required
- Distributive: correction of reduced systemic vascular resistance required

Common pharmacological treatments

Noradrenaline (dose 0.01 – 1.00 microgram / kg /min via a CVC)
Predominantly alpha-adrenergic agonist (causing vasoconstriction); some beta-agonism
Used in conditions causing distributive hypotension after restoration of circulating volume (sepsis, 'sedation-related' hypotension)
Contra-indications / cautions: hypovolaemia, severe peripheral vascular disease

Vasopressin (dose 0.01 – 0.04 unit / min via CVC)
Potent vasoconstrictor, ADH-analogue
Used in conditions causing distributive hypotension after restoration of circulating volume e.g. sepsis. Often added in as second-line agent after noradrenaline
Contra-indications / cautions: chronic nephritis, severe peripheral vascular disease

Metaraminol (dose 0.5 – 5 mg / hour)
Predominantly alpha- agonist. Can be given peripherally, hence sometimes used perioperatively if a CVC is not present. However it does not have any beta agonistic effects therefore escalating doses will not demonstrate any benefit. Should be converted to noradrenaline via CVC if persistent requirement.

Adrenaline (dose 0.01 – 1.00 microgram / kg / min)
Mixed beta- (at lower doses) and alpha- (at higher doses) agonism.
Used in conditions of poor myocardial contractility / distributive hypotension (LVF, anaphylaxis). Often causes tachycardia and a lactic acidosis.
Contra-indications / cautions: arrhythmias

Dobutamine (dose 2.5 – 20 microgram / kg / min)
Predominant mechanism of action: beta-1 adrenergic agonism
Used to increase inotropy (eg in LVF) but vasodilation and tachycardia limit dose
Contra-indications / cautions: hypovolaemia, arrhythmias, HOCM

Milrinone *(dose 0.25 – 0.75 microgram / kg /min; loading dose usually omitted)*
Phosphodiesterase inhibitor causing increased inotropy
Also an inodilator causing vasodilatation
An alternative to dobutamine, possibly preferable in RV failure.
Contra-indications / cautions: hypovolaemia

Isoprenaline (dose 1-4 mcg/min infusion of 2mcg/ml (4mcg/ml via CVC)
Predominately a beta1 agonist
Contraindications/ cautions: tachyarrhythmias

Further reading

Bangash MN, Kong M-L, Pearse R. Use of inotropes and vasopressors in critically ill patients. Br J Pharmacol 2012 Apr; 165(7):2015-2033

Section 2: Organ support – Analgesia and Sedation

The general aim of sedation in ICU patients is to decrease anxiety and agitation with production of a calm but communicative state to minimise patient discomfort and facilitate treatment. We use the Richmond Agitation-Sedation Scale to measure the sedation level of our patients.

The following medications are commonly used for sedation and/or analgesia in the RLH ACCU:

Drug	Mechanism	Side Effects	Interactions	Hepatic/Renal	Doses	Other
Fentanyl	• Synthetic and highly lipid-soluble opioid • Potency ~ 80x morphine • Primarily μ-receptor agonist • Sedative and analgesic • Rapid onset	• Bradycardia • Respiratory depression • Apnoea • Pruritis/urticaria • Constipation • Rigidity (at high doses)	Additive or potentiating effect with other CNS depressants	Reduced clearance in renal impairment	As infusion to maintain sedation: 0.5-3mcg/kg/hr, typically 25-400mcg/hr	• Accumulates with prolonged infusion • Useful in patients with cardiovascular compromise
Propofol	• A unique sedative-hypnotic agent • Achieves this by positive modulation of the inhibitory function of the neurotransmitter gamma-aminobutyric acid (GABA) through GABA-A receptors • Not an analgesic • Rapid onset and offset	• Apnoea • Respiratory depression • Hypotension • Pain at injection site • Bradycardia • Discoloured urine	Sedation effects potentiated by other CNS depressants Caution in conjunction with other agents known to cause hypotension	Renal and hepatic impairment have minimal effect on pharmacokinetics	As infusion to maintain sedation: 1-4mg/kg/hr Highly variable doses often required (e.g. high doses in patients with history of alcohol excess, low doses in the elderly)	• Decreases ICP • Seizures listed as potential side-effect, however commonly used to suppress seizure activity • **Propofol infusion syndrome** (see below)
Midazolam	• Short-acting, water-soluble benzodiazepine • It is an anxiolytic, sedative, hypnotic, amnesic and anticonvulsant • Achieves this through potentiation of the neural inhibition mediated by GABA (different receptor site to Propofol) • Not an analgesic	• Respiratory depression • Confusion • Anterograde amnesia • Myoclonic jerks • Hypotension • Bradycardia	Midazolam is metabolised by CYP3A4. Inhibitors and inducers of CYP3A4 respectively increase and decrease the plasma concentrations (e.g. fluconazole, Ca channel blockers)	Accumulation especially with obesity or hepatic or renal impairment	Typical doses of 1-20mg/hr	• Useful in patients with cardiovascular compromise • Anticonvulsant • Accumulates with prolonged infusion • Tolerance and dependence can occur
Clonidine	• Centrally acting alpha2-agonist • Provides sedation with minimal respiratory depression and preserved arousability • Analgesic at higher doses	• Hypotension ++ • Bradycardia • AV block	Interacts with Methylphenidate Avoid in patients with Raynaud's or peripheral vascular disease	Accumulation occurs with renal impairment	Boluses of 25-100mcg (IV or oral/NG) TDS Infusion: 0.5-2mcg/kg/hr (up to 4mcg/kg/hr can be given)	• Sudden cessation may cause rebound hypertension • Tolerance • Tachyphylaxis

Propofol Infusion Syndrome (PRIS)

Common presenting features: unexplained new onset metabolic acidosis, cardiac dysfunction, rhabdomyolysis, renal failure, and hypertriglyceridaemia

Incidence: 1.1% of adult patients in mixed ICU. Occurred at a median of 3 days (range of 1–6 days) after the start of propofol

Risk factors: severe head injuries, severe burns, trauma, severe sepsis, pancreatitis, prolonged high dose propofol infusion, high exogenous or endogenous catecholamine and glucocorticoid levels, low carbohydrate to high lipid intake

Recommendations: Propofol infusions for sedation should not exceed 4 mg/kg/hr, routine monitoring of CK and triglycerides in the at-risk population

Treatment: nil specific treatment. Stop propofol and support organs as necessary

Analgesia:

Analgesia is typically provided to patients in ICU with the primary goal of optimising patient comfort, but also to attenuate the potentially deleterious physiological responses to pain.

Commonly prescribed agents include: paracetamol, morphine, fentanyl, oxycodone, gabapentin, clonidine and occasionally ketamine

Regional anaesthesia in the form of epidurals or paravertebral infusions is also frequently used in our post-operative and trauma patients.

Opioid analgesia is frequently administered via a PCA (Patient Controlled Analgesia) pump.

Further reading:

1. Local RLH ACCU prescribing guidelines
2. Ne-Hooi Will Loh, Priya Nair, Propofol infusion syndrome. Continuing Education in Anaesthesia Critical Care & Pain, Volume 13, Issue 6, 1 December 2013, Pages 200–202, can be found at: https://academic.oup.com/bjaed/article/13/6/200/246704
3. Intensive Care Society Review of Best Practice for Analgesia and Sedation in the Critical Care, 2014. Can be found at: https://www.ics.ac.uk/ICS/guidelines-and-standards.aspx

Section 3: Post – Operative management: Extra-ventricular drain (EVD)

An EVD is the gold-standard device for Intracranial Pressure (ICP) monitoring and allows CSF drainage. It requires surgical placement in a lateral ventricle via a burr hole thus passes through brain tissue (risk of damage and haemorrhage) The transducer is usually remote and the device is "zeroed" at the external auditory meatus (EAM). It is a common piece of equipment in neurosurgical ICU patients.

Indications

- Measurement and treatment of raised ICP
- Hydrocephalus or at risk of hydrocephalus following Traumatic Brain Injury (TBI)

Contraindications

- Coagulopathy or infection at insertion site

Design

Management

Check the indication and date of insertion of any EVD along the with the patient's current neurological status. Ensure CSF is draining by inspecting the drip chamber and whether the EVD column is oscillating. Ensure the drainage manometer is set at the correct height and hourly drainage instructions are clear (check the Neurosurgical operative note/documentation). Example settings would be 10-20 cmH_2O at level of EAM and drainage of up to 10ml/hr of CSF. Check ICP measurement and recent output and follow steps below if abnormal. CSF should be intermittently sampled to detect infection (not uncommon, referred to as "ventriculitis").

Section 3: Post-operative management – Epidural

"Epidurals" (epidural catheters), effective means of analgesia, are placed under sterile conditions into the epidural space and must be connected to an anti-bacterial filter. Epidural pumps are programmed to provide a continuous infusion of local anaesthetic +/- opioid and in theory this diffuses in the epidural space to bathe nerve roots as they exit.

Standard prescription is with **0.1% levobupivicaine and 4 microgram / mL fentanyl at a rate of 4-15 mL/hour.** Pre-printed stickers should be used to avoid potential for drug errors. Although often highly effective there are a number of potential hazards

- Inadvertent intrathecal/subdural placement of the catheter. This should have been identified prior to coming to ACCU, but suspicion should be aroused if the sensory/motor block is much higher than expected particularly after a bolus.

> ***Red flag (call 1113/45715):*** *Local anaesthetic can rapidly spread upwards to cause profound hypotension, bradycardia and loss of consciousness so* ***call for senior anaesthetic help ASAP.***

- Intravenous placement of catheter / inadvertent connection of the epidural infusion to an intravenous cannula. The latter is a never-event and both are potentially fatal. The epidural infusion should only be connected by two trained staff members.
- High "block". Sensory level and presence of motor blockade should be regularly checked and documented. A sensory block higher than T4 or a motor block should prompt temporary cessation/reduction of infusion rate – see epidural recording charts for management.
- Epidural haematoma. Rare, but serious complication. A motor block which does not improve soon after stopping infusion should prompt an urgent senior review. Motor block is monitored by the Brommage score: 1 (no motor block), 2 (just able to flex knees), 3 (unable to flex knees) 4 (unable to move feet either).
- Ineffective block – patient in pain. Consider anaesthetic colleague review (see the troubleshooting section).
- Hypotension. Correct fluid status and consider vasopressors if analgesia provided by the epidural is effective. A slight reduction in rate of epidural infusion (2mL/hr) may be required.

Epidurals should generally be removed by day 5, or sooner if the patient is septic or has signs of infection at the insertion site. It is imperative that removal is in the presence of normal clotting / platelet counts, and >12 hours after last dose of prophylactic LMWH or >24 hours after treatment dose LMWH; the next dose should also be deferred for >=4 hours after removal.

Section 3: Post-operative management – Lumbar spinal drain

Indications for Lumbar Drain (LD) insertion

- Management of CSF leak
- Following SAH haemorrhage
- During/ following aortic aneurysm surgery - For spinal cord protection, preventing spinal cord ischaemia (SCI). Reduction of spinal blood flow due to oedema and placement of endografts/stents over thoracic aorta can lead to cord ischaemia. CSF drainage reduces risk of SCI.
- Following spinal surgery
- Post-operatively (e.g. after dural repair or removal of tumour from sinuses, etc)

Management of LD	
• Unless otherwise specified the drain should be left open with continuous drainage of CSF (+/- intermittent pressure monitoring) • The chamber should be setup at the prescribed height • The drain should be kept in the open position • Adjust height as required to avoid excessive drainage (>15-20 ml/hour) or as directed by the neurosurgeons	

Red flag (call 1113/45715): *The following signs should prompt discussion with a consultant or 1113 SpR or neurosurgeon:*

- *Reduced GCS*
- *Frank blood in CSF drainage*
- *Altered sensation/loss of power to lower limbs*
- *Leakage around the site*
- *Loss of patency*
- *Over-drainage (>15-20 ml/h)*

Other tips:

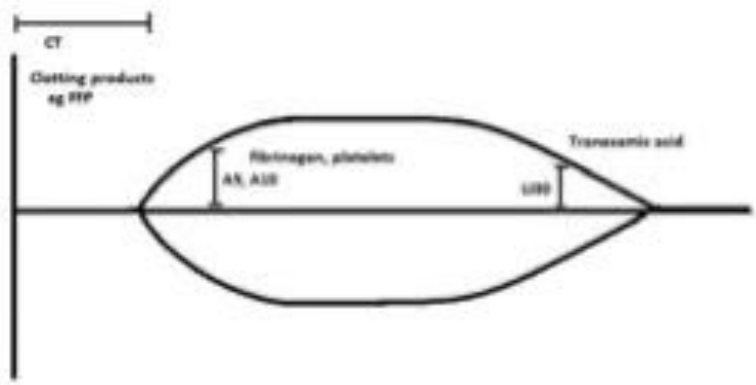
- If a change of system is indicated, this needs to be carried out under strict aseptic technique
- CSF sampling is only to be carried out by neurosurgeons under strict aseptic technique
- Remove lumbar drain at least 12 hours after the last dose of LMWH
- Do not give LMWH until 6 hours after removing the LD catheter

Section 3: Post-operative management: Point of care coagulation testing (ROTEM)

Traditional measurement of clotting parameters involves measurement of platelet count by FBC and clotting status by coagulation screen and fibrinogen. Although often appropriate for elective surgery or procedures and for monitoring on ACCU, in situations of massive and ongoing haemorrhage these tools are limited by speed of turn-around in the lab. Additionally, the platelet count tells us nothing about platelet function, which is relevant for example in those taking antiplatelet medications.

*Ro*tational *t*hrombo*e*lasto*m*etry is often useful in such situations. A sample sent to the machine in theatres can deliver a result within 10-20 minutes. It gives several parameters that quantify clot formation, strength and fibrinolysis and consequently suggest whether platelets, clotting products or tranexamic acid are appropriate therapies. A repeat ROTEM can be sent after 15 minutes of blood product administration.

A small pin is rotated within the sample of blood and over time, a clot is formed. As a gross simplification, the y-axis below represents the size of the clot and the x-axis is time. The initial time to formation of clot (clotting time) is dependent predominantly on clotting factors; the strength/amplitude and speed of formation of clot on fibrinogen and platelets; and any subsequent deterioration in clot size is dependent on thrombolytic activity – hence the suggested therapies in the diagram.



The algorithm for "correction" can be hard to commit to memory but is displayed in the 4E office and is on the Box account.

In situations of massive haemorrhage, amongst other derangements in physiology, remember also to anticipate and correct hypocalcaemia, hyperkalemia and hypothermia; as well as addressing any surgical cause to bleeding.

Further reading

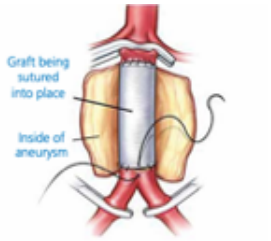
Srivastava A, Kelleher A. Point-of-care coagulation testing. Continuing Education in Anaesthesia Critical Care & Pain.

Section 3: Post- operative management: Post Abdominal Aortic Aneurysm (AAA) repair

Open AAA repair: Major abdominal surgery during which the aorta is clamped; denying blood flow to a number of organs and sections of the spinal cord. Peri-operative mortality varies widely and complication rates are high (15-30%).

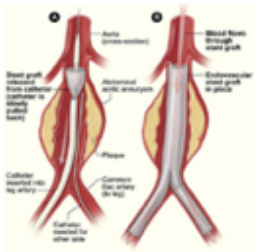
Common complications:

- Abdominal compartment syndrome (post repair of ruptured AAA)
- Ischaemic colitis
- Acute kidney injury
- Acute limb thrombosis
- Spinal cord ischaemia
- Infection



EVAR (Endovascular Aortic Repair): Radiologically guided placement of an expandable stent graft within the aorta. Associated with a three-fold reduction in perioperative mortality (however increased rates of long-term complications and no over-all long-term mortality benefit).

Common complications:



- Endoleak (30-40%)
- Continued enlargement of the aneurysm
- Delayed aneurysm rupture
- Stent migration
- Access site complications
  (e.g. bleeding, haematoma, false aneurysm, infection)
- Acute limb thrombosis and ischaemic colitis are less common
- Infection

Note: Patients whom develop AAA are typically elderly, have associated co-morbidities such as coronary artery disease, heart failure, COPD, chronic renal failure. Thus are at high risk of post-operative complications such as AMI, stroke, pneumonia and acute kidney injury.

Post-operative monitoring and management in ICU:

- Continuous ECG monitoring
- Invasive BP monitoring (arterial-line)
- Pulse oximetry
- Urinary catheter and hourly documentation of urinary output
- Regular arterial blood gases (particularly noting lactate and Hb)
- Regular neurovascular observations of lower limbs (often require doppler USS for pulses)
- Adequate analgesia (often with an epidural)
- Targeted blood pressure management (as guided by surgeon specified targets)
- Wide-bore nasogastric tube in situ

Additional:

- Monitoring of intra-abdominal pressures
- Low threshold to start renal replacement therapy
- May require cardiac output monitoring to guide fluid administration
- Some patients may have a lumbar drain in situ

When to call the surgeons:

- Rising lactate (despite adequate fluid resuscitation)
- Sudden drop in haemoglobin
- Raised intra-abdominal pressures
- Absent pedal pulse(s) or other clinical features of lower limb ischaemia

Further reading

1. Choudhury M. Postoperative Management of Vascular Surgery Patients: A Brief Review. Clin Surg. 2017; 2: 1584 available at: http://www.clinicsinsurgery.com/pdfs_folder/cis-v2-id1584.pdf
2. https://radiopaedia.org/articles/endovascular-aneurysm-repair?lang=gb

Section 4: Troubleshooting – Renal Replacement Therapy

RRT is set-up by experienced nursing staff, some of which have attended the Royal London training course specific to our equipment and are called **Link Nurses**. Filter problems should therefore be directed to them. Below are frequent filter issues that having an awareness of is important for all involved (*red flags in red - when consultant discussion is essential*)

Should we use a higher dose mode for this patient?

- The dose is standardised on the protocol. There is an option to use a higher dose of RRT (60ml/hg/hr) which needs to be confirmed with the consultant.

This patient's sodium is 165 and they need urgent RRT for renal failure with severe fluid overload, how should we commence RRT?

- Extremes of sodium can be too quickly corrected on RRT with sometimes-fatal consequences so these cases need to be discussed with the consultant before starting RRT.

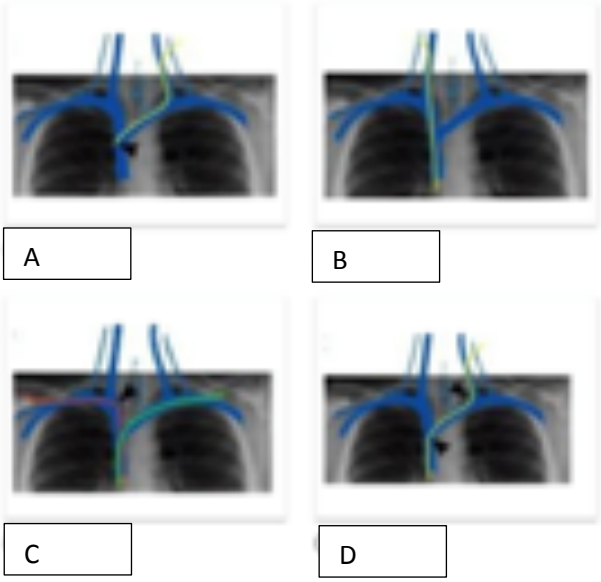
What anticoagulation should we use?

- The protocol states the first line as regional **citrate** anticoagulation. The nursing team will often assess the contraindications to this mode of anticoagulation and if in doubt will ask the clinical team. This needs to be confirmed with the consultant or senior ICM trainee.

Difficult to treat hypocalcaemia OR persistent alkalosis are signs of citrate toxicity and need to be flagged to the consultant.

The line is not working, what should we do?

- Access alarms are very common and a major cause of reduced treatment for patients on RRT. The line tip should be within or close to the right atrium.



A: this vascath is not within the right atrium and likely to cause alarms
B: shows the ideal line placement - Right Internal jugular (this is the first option)
C: subclavian line is the fourth option (1st right, 2nd left internal jugular, 3rd femoral)
D: demonstrates why left is second due to multiple turns in the line to reach the right atrium limiting flow of blood

Section 4: Troubleshooting - High ICPs

Raised ICPs should be reviewed and treated urgently as they are associated with increased mortality: Look for a cause, low threshold for repeat CT scan and urgent neurosurgical input is essential.

1st line therapy: if ICP >20mmHg for >15mins/regular spiking

- *Inform Neurosurgeons/ ACCU consultant/ 1113 registrar*
- Sedate; to enable ventilation and reduce metabolic demands of brain tissue
  - [Propofol 4mg/kg/hr + Fentanyl 4mcg/kg/hr +/- midazolam 0.1mg/kg/hr]
  - Be wary of propofol infusion syndrome – consider checking lipids (See the analgesia and sedation section)
  - Fluids (sparingly) and vasopressors (noradrenaline) to achieve a MAP keeping CPP 60-70mmHg
  - Cardiac output monitor if >0.2micrograms/kg/min, send ACTH (review scan, ? pituitary involvement)

2nd line therapy:

- Bolus sedation [e.g. 50mg propofol/100micrograms fentanyl] – assess response
- Neuromuscular blockade – increases venous return, support ventilation, stop coughing
  - Atracurium 50mg or Rocuronium 50mg +/- infusion if helpful
- Consider introducing anticonvulsants (e.g. Benzodiazepines, Leviteracetam 500mg loading + 500mg BD)
  - Brainz monitor may be useful ? seizing, request in hours EEG
- Prevent hyperthermia – aim normothermia which may requiring active cooling
- Consider therapeutic trial of hyperventilation (if on ITU >24hrs, can aim PCO2 4.5-5kPa)

3rd line therapy: A scan should have happened by this time and a decision whether for theatre or not

- Burst suppression:
  - Thiopentone challenge of 3mg/kg bolus aiming to burst suppress on Brainz (see section on continuous EEG monitoring)
- Hypertonic osmotherapy - bridging therapy
  - Hypertonic saline (2ml/kg 5% NaCl)
    - May be repeated every 4-6 hours
    - Provided Na^+ <150mmol/L
  - Mannitol (2ml/kg 20%)
    - Can be repeated if needed if serum osmolality <320 mOsm/kg & CV stable

Neurosurgeons may consider urgent decompressive craniectomy for intractable intracranial hypertension

Section 4: Troubleshooting - Invasive ventilation

Introduction

Ventilators can seem daunting and ventilation problems may arise anywhere along the chain from the patient to the ventilator to the power/gas supplies. However, as with any emergency in ICU, a structured approach will make things a lot more manageable.

Whilst troubleshooting increase the oxygen to 100%: assess patient first, then look at the ETT, then the tubing and finally the ventilator.

Assessment

The acronym 'DOPES' may help you when there is a failure in ventilation.

Displacement	- Inadvertent extubation - Endobronchial migration
Obstruction	- Endotracheal tube (ETT) kinked - ETT gripped by teeth - Secretions or blood within lumen of tube - Mucus plug in airways
Pulmonary	- Atelectasis & alveolar de-recruitment - Pneumothorax - Bronchospasm - Pulmonary oedema - Pulmonary embolus - Worsening of underlying pulmonary pathology
Equipment	- Disconnection - Leaks - Ventilator failure - Oxygen failure
Sedation	- Inadequate sedation may cause agitation, coughing and ventilator asynchrony - Over-sedation may cause hypoventilation in a spontaneously breathing patient

Management

If the patient is compromised, e.g, hypoxic/tachycardic, **start 100% oxygen and call for help immediately** (the quickest way may be to shout or pull the emergency buzzer at the bedspace). As you can see from the acronym, assessing the Airway patency and then Breathing are paramount. If you identify a problem at any stage, e.g. inadvertent extubation, address the situation as best you can before moving on. You are **not** expected to be intubating patients. This is rarely a life-saving procedure. However, performing effective airway manoeuvers, inserting an airway adjunct and supplying bag-mask-valve ventilation with 100% oxygen will regain control in the majority of situations. This will then allow time for the drugs, equipment and personnel to arrive and allow re-intubation to occur.

Getting a brief history of the patient and examining the chest will reveal most pulmonary problems. The management of pulmonary problems varies widely therefore your assessment is crucial to get the correct diagnosis. As well as the history and examination urgent bedside investigations (CXR, ultrasound) and/or a trial of treatment (e.g. bronchodilator, chest physiotherapy, diuretic, recruitment manoeuver) may be warranted. Pay attention to the alarms on the ventilator too as they may offer clues to other pathology, e.g. high airway pressures (? obstruction), apnoea (? oversedation). Other ventilator alarms are more obvious e.g. disconnection, leak, oxygen failure and low battery (portable ventilators). These issues will require urgent assessment from the ICU Registrar or Consultant. The Senior Nursing staff are sometimes the most familiar with the ventilator equipment (as they set them up) and you should always ask for their help if you are unsure of anything.

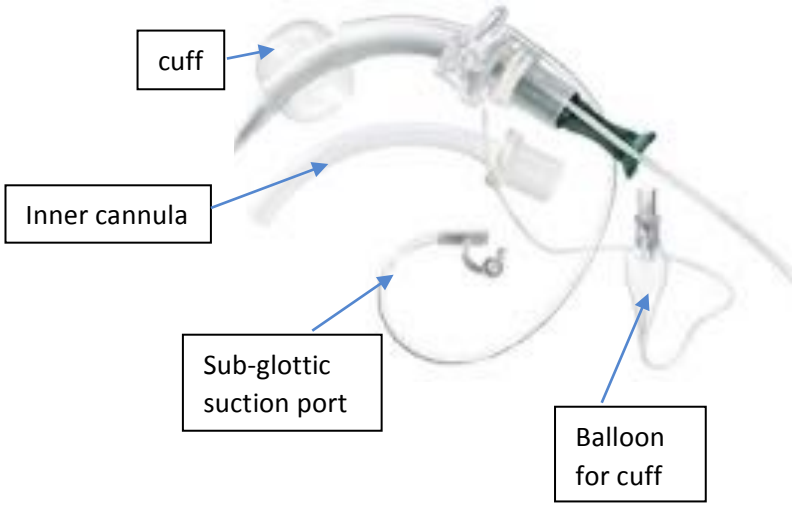
Finally, assess the patient's sedation level and check that the IV sedation is running and connected to the patient through a patent venous cannula. Inadequate sedation may require a sedative bolus and should only be done by those familiar with the drug. Over-sedation will require a reduction in sedative infusion(s) and a mandatory/mixed mode of ventilation (see *'Invasive Ventilation'* section) until they start breathing adequately.

<u>An approach to de-saturation:</u>

Section 4: Troubleshooting - Tracheostomy

Three common types in ACCU:

- TRACOE *twist* plus size 7-9mm (cuffed, non-fenestrated, sub-glottic suction port)
- Adjustable flange
- Laryngectomy stoma/tube (rare)

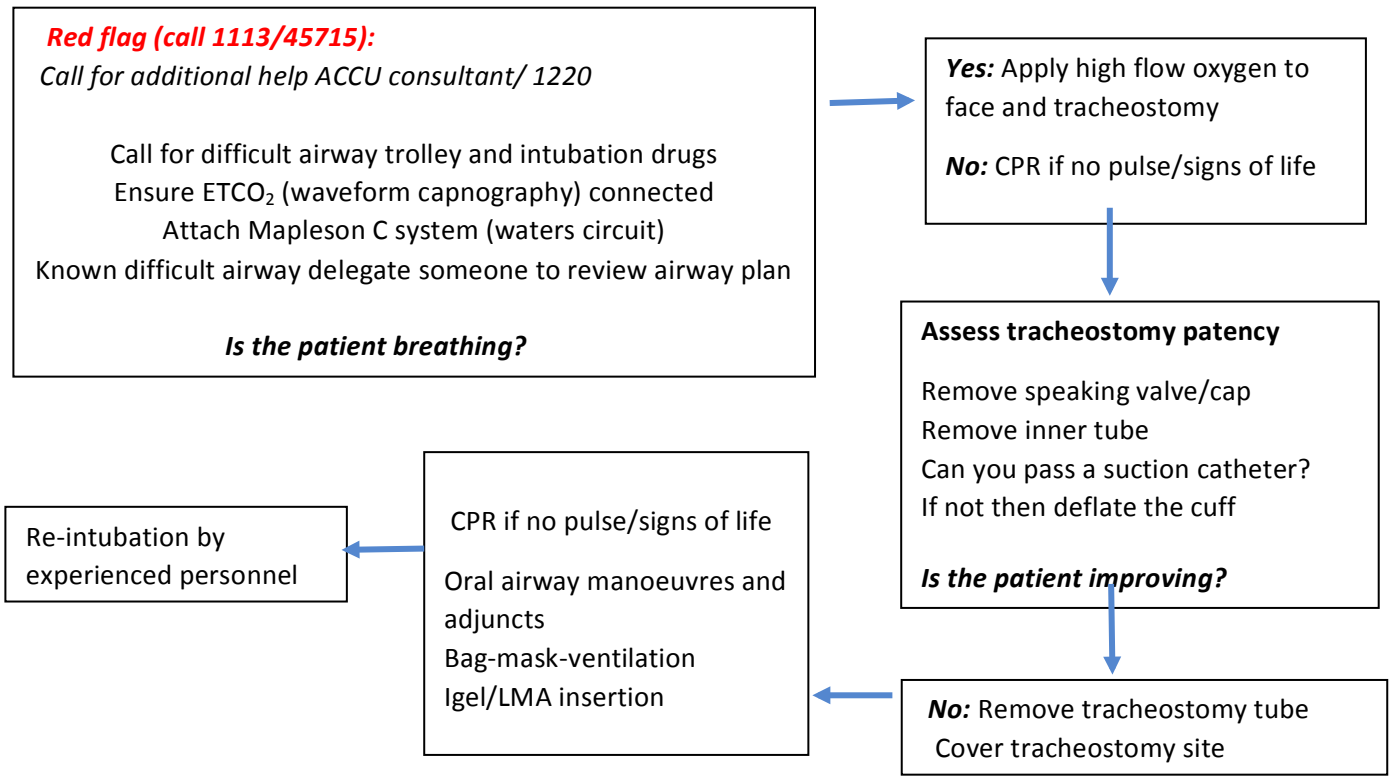


Complications:

Immediate	Delayed	Late
Hypoxia	Infection	Bleeding
Tube dysfunction	Tube displacement/migration	Tracheal injury/dysfunction
Malposition	Tube obstruction	Speech problems
Damage to local structures	Erosion/ulceration	
Air-related complications		

Signs of a tracheostomy problem: Change in $ETCO_2$ trace, hypoxia +/- cardiovascular instability or cuff leak.

Emergency tracheostomy management (known patent upper airway):



If successful reintubation doesn't allow easy ventilation consider:

- Tracheal obstruction – secretions, blood, foreign body, bronchoscopy +/- lavage
- Tracheal false passage – Maxillo-facial surgeons +/- reintubation with manual guidance
- Pneumothorax – bilateral thoracostomies if in doubt

Further reading

1. Hunt K and McGowan S. Tracheostomy management, pages 149-153. BJA Education Volume 15, No 3, 2015.
2. ICS TICS. Standards for the care of adult patients with a temporary tracheostomy Standards and Guidelines 2014.
3. Cook TM, Woodall N, Harper J, Benger J Major complications of airway management in the UK: results of the 4th National Audit Project of the Royal College of Anaesthetists and the Difficult Airway Society. Part 2 Intensive Care and Emergency Departments. Br J Anaesth 2011;106:623-42.
4. http://www.tracheostomy.org.uk (for difficult airway algorithms)

Section 4: Troubleshooting – Epidural

Epidurals when working provide excellent analgesia, however there are many reasons why an epidural fails and this requires careful consideration and troubleshooting to maximise the chance of it working before considering taking it out.

A thorough assessment should be carried out prior to any intervention that includes:

- History from the patient – pain score, type of pain and location of pain
- Examination – check the position of catheter (compared to what was documented) check sensory/motor level
- Optimise patient position

Serious complications, which should be identified promptly:

- **High block** – see epidural chart for management, but generally involves temporarily stopping infusion / reducing infusion rate.
- **Epidural haematoma** – non-resolving motor block. MRI is the investigation of choice. Seek senior input. Urgent decompression may be required.
- **Hypotension** – correct fluid status, consider reducing infusion rate (if high block) or adding noradrenaline to support BP

The reasons for epidural failure in ICU is ineffective block and the patient is in pain:

- **No clinical discernible sensory block**
  - Check epidural hasn't fallen out or leaking!
  - Consider a bolus +/- increase in infusion rate by airway-trained colleague
- **Unilateral block**
  - Consider a 5ml bolus on "bad side down" if block is one-sided
  - If ineffective consider epidural catheter migration and an anaesthetic colleague may consider withdrawing the catheter 1 cm (timed around LMWH) and repeating
- **Low sensory level block**
  - Consider a bolus 5-8 mL (must be given by an anaesthetic-trained colleague) and then increasing infusion rate. Ensure patient has cardiovascular monitoring
- **If still ineffective**, an IV PCA may be started; either alone, or with a change of the epidural infusion prescription to (just) plain levobupivacaine.

Section 4: Troubleshooting – Extra-ventricular drain

EVDs allow interpretation of ICP waveforms, *see 'ICP Monitoring'.* A flat ICP trace suggests compression or kinking of the transducer.

- Check the length of the tubing from insertion point (use aseptic technique) to transducer and relieve any kinks/pressure.
- If CSF drainage is absent check the manometer height and that the 3-way stopcocks are open and tubing is free of obstruction.
- If no issues are found then Neurosurgical opinion should be sought urgently along with alerting the ACCU Senior Registrar/Consultant.
- Likewise, a high CSF output warrants the same escalation and review.
- A rounded appearance of the waveform suggests a raised ICP, check the height and connections.
- If the trace remains rounded or the ICP measurement is high (>20mmHg) in a properly set up system escalate and seek urgent Neurosurgical review (and refer to the guidance in the *'Neuroprotection'* section).

Things to watch out for:

- **Drop in GCS in a patient with an EVD**
  - Inform neurosurgeons/ACCU consultant/1113 registrar
  - Check that EVD is draining
  - If it is not draining after discussion with a senior member of the team consider dropping the height of the manometer and observe to see if CSF drains
  - Check the pupils of the patient (high risk of hydrocephalus)
  - Arrange an urgent CT head
- **Clots/tissue fragments in the EVD catheter**
  - Inform neurosurgeons/ACCU consultant/1113 registrar
  - Check that the EVD is draining
  - Check that there is pulsation of the CSF meniscus in the drain tubing
  - Damping of ICP waveform, reduction/absence of CSF drainage and lack of pulsation all insinuate catheter obstruction
  - Neurosurgical team will need to attempt: flushing the catheter in sterile conditions or change of EVD.
- **Cloudy CSF fluid**
  - EVD associated meningitis or ventriculitis can occur
  - Risk factors for EVD infections include: systemic infection, depressed skull fractures, catheter leak/migration, frequent CSF sampling and duration of EVD placement.
  - Neurosurgical team may sample the CSF and consider withdrawal of EVD.

Section 5: Other key topics – Neuroprotection

Neuroprotective management strategy is key in the management of Traumatic Brain Injury. There are a number of mechanisms by which the injured brain may suffer secondary insult: reduced cerebral oxygen delivery, increased cerebral oxygen requirement, exocitoxity, inflammation, hyperglycaemia and free radical damage.

Physiological targets: **ICP <20mmHg & CPP 60-70mmHg are key goals**

ICP	<20mmHg	$P0_2$	>10kPa
CPP	60-70mmHg	Glucose	5-8mmol/L
MAP	To achieve CPP (>90mmHg if no ICP bolt in situ)	Temp	Normothermic
PCO_2	4.5-5kPa	Na^+	135-145 (up to 155 often acceptable if consultant agrees)

CPP = Cerebral perfusion pressure; = MAP – ICP

Standard care:

- Head in neutral position, 30 degrees elevation of head, no tight neck constraints
- Arterial transducer at level of mastoid for CPP evaluation
- NGT & early feed, ensure laxatives start early
- DVT, GI prophylaxis – LMWH typically held for 48-72hrs depending on injury
- Note many patients will potentially develop Diabetes Insipidus; monitor urine output and paired serum and urine osmolarities if needed. Note that hypertonic solutions often cause profound diuresis.

Before each step:

- Check ICP transducer height; ensure not coughing/dysynchronous with ventilator and there is no shivering
- Think: **IS A SCAN NEEDED? SHOULD I BE DISCUSSING THIS WITH THE NEUROSURGICAL TEAM, WHAT ARE THE PUPILS DOING?**
- If pyrexial, cool to normothermia
- Potential neurosurgical interventions are: EVD placement, bone flap removal, haematoma evacuation, decompressive contusionectomy/craniectomy.

Management of high ICP – see troubleshooting high ICPs.

Further reading

Brain Trauma Foundation. 2016. Guidelines for the management of Severe TBI. 4th ed.

Section 5: Other key topics – Neuro-prognostication

We have many patients who suffer traumatic brain injuries and complex cerebrovascular disease. Many patients die before leaving the intensive care unit, and those that survive are often left very disabled. Below is a quick guide to the steps typically taken in neuro-prognostication, and what you may be asked to do for this. Please bare in mind that neuro-prognostication is very difficult and requires a multi-disciplinary approach.

Clinically:
GCS at baseline (prior to intubation) is important and it's worth finding this information out. Is the patient coughing on suction when off sedation? Do they have corneal reflexes? What is the GCS (table below) and are the pupils reactive?

EEG: Electroencephalogram
Requested via CRS and normally happens the same day (won't need chasing). ?Seizures, ?cortical activity, ?cerebral dysfunction

SSEPs: Somatosensory evoked potentials
Tests pathways of cutaneous nerves and subsequent intracranial activity, typically used in people who are breathing, but have likely devastating intracranial insult.

MRI head:
Typically performed a few days after insult, this needs to be requested on CRS, then protocolled with the radiologist and a slot arranged with MRI (typically one slot a day for ventilated patients). This should be done first thing in the morning.

Refer to Dr Liu
Dr Liu is a neurology consultant with a special interest in neurorehabilitation and neuroprognostication. Once the above tests are performed, he can review the patient and provide some idea of prognostication and suitability for neurorehabilitation. He typically reviews patients on Thursday afternoons, and a referral is made via. email (Clarence.liu@bartshealth.nhs.uk).

Make sure that you know how to assess a GCS quickly:

Score	Eye opening	Verbal response	Motor response
1	None	None	No movements
2	Open to painful stimulation	Incomprehensible sounds	Extends to pain
3	Open to voice	Inappropriate words	Abnormal flexion to pain
4	Open spontaneously	Confused, disoriented	Withdraws to pain
5	-	Oriented, converses	Localises to painful stimulus
6	-	-	Obeys commands

Legend: The GCS is obtained by adding the value for each category: minimal = 3, maximum = 15.

Further reading
BMJ Best Practice: Coma https://bestpractice.bmj.com/topics/en-gb/417

Section 5: Other key topics – Spinal precautions

Many patients suffer from poly-trauma, with multiple organ systems affected. Often patients are intubated and therefore formal neurological assessment isn't possible. As such, patients are often left on spinal precautions until woken, or until spine radiologically cleared.

Neurogenic shock – Bradycardia, hypotension, respiratory failure, spinal shock = loss of reflexes below level of injury.

- Intubated and ventilated for respiratory failure
- Noradrenaline for cardiovascular instability; Atropine 500microgram/ Glycopyrronium 200-400microgram boluses if bradycardic.

Spinal Clearance:

- If GCS 15/15, no spinal pain, no distracting injury or ETOH → Clear clinically via. NEXUS guidelines (https://radiopaedia.org/articles/nexus-criteria)
- If GCS 15/15, with focal tenderness, neurology or high-risk mechanism → any appropriately experienced clinician can review imaging to clear.
- If above not met:
    - Radiological clearance: On CT spine; must be reported by ST4+ Radiologist (must hold FRCR) and **typically discussed with neurosurgical team before stopping precautions.** If CT scan abnormal or indeterminate, neurological deficit or high-risk mechanism → MRI.

Precautions depend on the level/type of injury and typically include:

- Log roll with head hold
- Hard collar, nursed in supine position
- Held hold on transfer to/from bed
- These may differ for patients with thoraco-lumbar injuries and may include sitting restricted to certain angles, orthotic braces or similar.

Further manual handling guides:

https://www.mascip.co.uk/wp-content/uploads/2015/02/MASCIP-SIA-Guidelines-for-MH-Trainers.pdf

Nursing supine with a hard collar makes management of TBI/chest injuries very challenging, so it is **important to clear spines as quickly as possible, or get a timely management plan if requires fixation/conservative care.**

Types of injuries typically requiring spinal precautions:

- Any fracture of C1/C2 (minimal ligament support) / Atlanto-Occipital subluxation
- Fractures involving >2 columns
- Bilateral facet joint dislocations
- Flexion teardrop
- Subluxation or fracture dislocation of vertebral bodies
- Burst fractures with associated instability
- 'Seatbelt' fractures

Types of injuries typically not requiring spinal precautions:

- Fractures involving single columns
- Isolated wedge fractures
- Transverse process fractures
- Spinal process fractures
- Isolated compression type burst fractures

Spinal Cord Injury care:

- There is a dedicated pathway for patients with spinal injuries (guidance on BOX), including bowel management, respiratory weaning, physiotherapy and onward care.

Further reading

Ackland HM. The Alfred Spinal Clearance Management Protocol. The Alfred, Melbourne June 2006 – Updated November 2009.

Section 5: Other key topics – Setting up for an Emergency Rapid Sequence Intubation

Emergency rapid sequence intubation (RSI) on ACCU is a common procedure, either semi-electively for deteriorating respiratory status, or as a true emergency (eg seizures). All ICU patients are considered "non-fasting" although a true RSI (that is, completely avoiding bag-mask ventilation) is rarely appropriate as ACCU patients are prone to hypoxia.

A number of simultaneous tasks need to be executed to perform rapid yet safe intubation. There is an intubation checklist poster at each bedside to act as an aide-memoire and this should be read aloud to ensure all team members are prepared for what can be both an anatomically and physiologically challenging procedure. In essence, the following need to be acquired:

Drugs

There are three large trays of drugs kept in the pharmacy fridge ready for use in intubations. Most commonly ketamine, midazolam, thiopentone or propofol are used to induce loss of consciousness – but ketamine will need to be acquired from the controlled-drugs cupboard, and extreme caution with thiopentone and propofol will need to be exercised for a majority of ACCU patients as these medications can profoundly lower BP. Fentanyl is also often given peri-induction of anaesthesia. Typically rocuronium is given as a muscle relaxant. Vasopressors will be required in most patients.

Airway equipment

There are both airway and difficult-airway trolleys on 4E and 4F. It is good practice to have both available at the bedside. An appropriately sized endotracheal tube will need to be prepared and the cuff checked along with any aids to intubation such as a Bougie. A 'Waters-circuit" with facemask and end-tidal CO_2 monitoring should be available at the patient's bed as should suction via a Yanker.

Every patient with a difficult airway should have a difficult airway plan at the bedside.

Sedation and a ventilator

To maintain comfort and ventilation following intubation.

A number of staff members will be required (sometimes also a staff member to maintain immobilisation of the neck in C-spine trauma patients). As a general principle there needs to be maintenance of oxygenation (with either the Waters-circuit applied tightly, NIV, or high-flow nasal oxygen), followed by induction of anaesthesia and then intubation.

Further reading

Sinclair R, Luxton M. Rapid Sequence Induction. Continuing Education in Anaesthesia Critical Care & Pain, vol 5, issue 2, 1 April 2005 - for 'classical RSI'

Section 5: Other key topics – Delirium

THINK Delirium: Delirium is very common affecting 70-80% patients in critical care. Use the 4AT/CAM-ICU/ICDSC screening tool.

Risk factors in critical care:
Modifiable: Blood transfusions, benzodiazepine use, and mechanical ventilation
Non-modifiable: Greater age, dementia, prior coma, emergency surgery, trauma, increasing APACHE.

Non- pharmacological multicomponent management:

A	Assessment, prevention and management of pain
B	Both spontaneous awaking trials and spontaneous breathing trials
C	Choice of sedation and analgesia
D	Delirium assessment, prevention and management
E	Early mobility and exercise
F	Family engagement and empowerment

Red flag (call 1113/45715):

1. Benzodiazepines have a limited role in the treatment of delirium. May be indicated when anti-psychotics contraindicated, if there is benzodiazepine withdrawal or alcohol withdrawal.

2. Unmanageable agitation requiring physical restraint despite the non-pharmacological and pharmacological measures above. May require further chemical restraint such as propofol, ketamine or general anaesthetic.

Pharmacological treatment:

Can be considered to treat any psychotic component/agitation only if non-pharmacological measures have not worked or the patient is at risk to themselves and/or others. No strong evidence for use of pharmacological agents in preventing delirium or in hypoactive delirium.

Drug		Dexmedetomidine	Olanzepine	Haloperidol	Quetiapine
Dose	Oral/IM	N/A	2.5mg -5mg PO/IM 2 hourly 24 hrs max 20mg (10mg if age > 75 yrs)	0.5mg PO/IM 2 hourly 24 hrs max 20mg (10mg if age > 75 yrs)	25mg BD PO Double dose every 24 hrs. 24hrs max 750mg
	Intravenous bolus	N/A	N/A	1 - 5mg IV Up to max 10mg per sedation event (5mg if age > 75 yrs)	N/A
	Infusion	Concentration: 4mcg/ml (dilute with 0.9% saline or 5% Dex). Loading dose: 1mcg/kg 10 mins Maintenance: Start 0.6mcg/kg/hr titrate to effect (0.2 – 1mcg/kg/hr)	N/A	N/A	N/A
Mechanism of action		Hight selective α_2-agonist. Minimal respiratory depression.	D_1, D_2, D_4, 5-HT_2, histamine -1 and muscarinic -receptor antagonist	D_2, 5-HT_2, , α_1 adrenoreceptor antagonist	D_1, D_2, 5-HT_2 and histamine 1 receptor antagonist
Side effects		Hypotension Bradycardia Tachyphylaxis Tolerance (> 24hrs)	Increased risk of stroke Prolonged QTc Hypotension	Extrapyramidal effects, prolonged QTc	Prolonged QTc
Interactions/CI		Potentiate other sedatives/hypnotics/opioids	Caution in cardiovascular disease and dysrhythmia	Parkinsons and lewy body dementia Caution in cardiovascular disease and dysrhythmia	Caution in cardiovascular disease and dysrhythmia

Further reading

1. Clinical Practice Guidelines for the Prevention and Management of Pain, Agitation/sedation, delirium, immobility and Sleep Disruption in Adult Patients in the ICU. Devlin et al. Critical Care Medicine. September 2018. Volume 46, Issue 9 p e825-e873.
2. Intensive Care Guidelines on the Detection, Prevention and Treatment of Delirium. https://www.ics.ac.uk/ICS/guidelines-and-standards.aspx

Section 5: Other key topics – Violent and aggressive patient

Maintaining safety of staff members and patients on the ACCU is paramount. Often it is difficult to rule out a reversible cause of violence and aggression without physical and/or pharmacological restraint.

Identify at risk patients: Mental health illness, previous violent/aggressive tendencies, extensive forensic history, admitted under Mental Health Act or substance/alcohol abuse.

Steps to undertake prior to de-sedation/extubation of high-risk patient:

- Ensure appropriate pre-extubation risk assessment is conducted
- Psychiatrist/RAID have been informed and have reviewed the patient
- Appropriate sedation/anti-psychotics have been prescribed/given
- Ensure post-extubation plan is made and documented
- Ensure additional health care assistant/ registered mental health nurse is available
- Highlight the at risk patients at morning handover daily

Red flag (call 1113/45715): *For any patient who is acutely violent, aggressive or extremely agitated requiring physical restraint.*

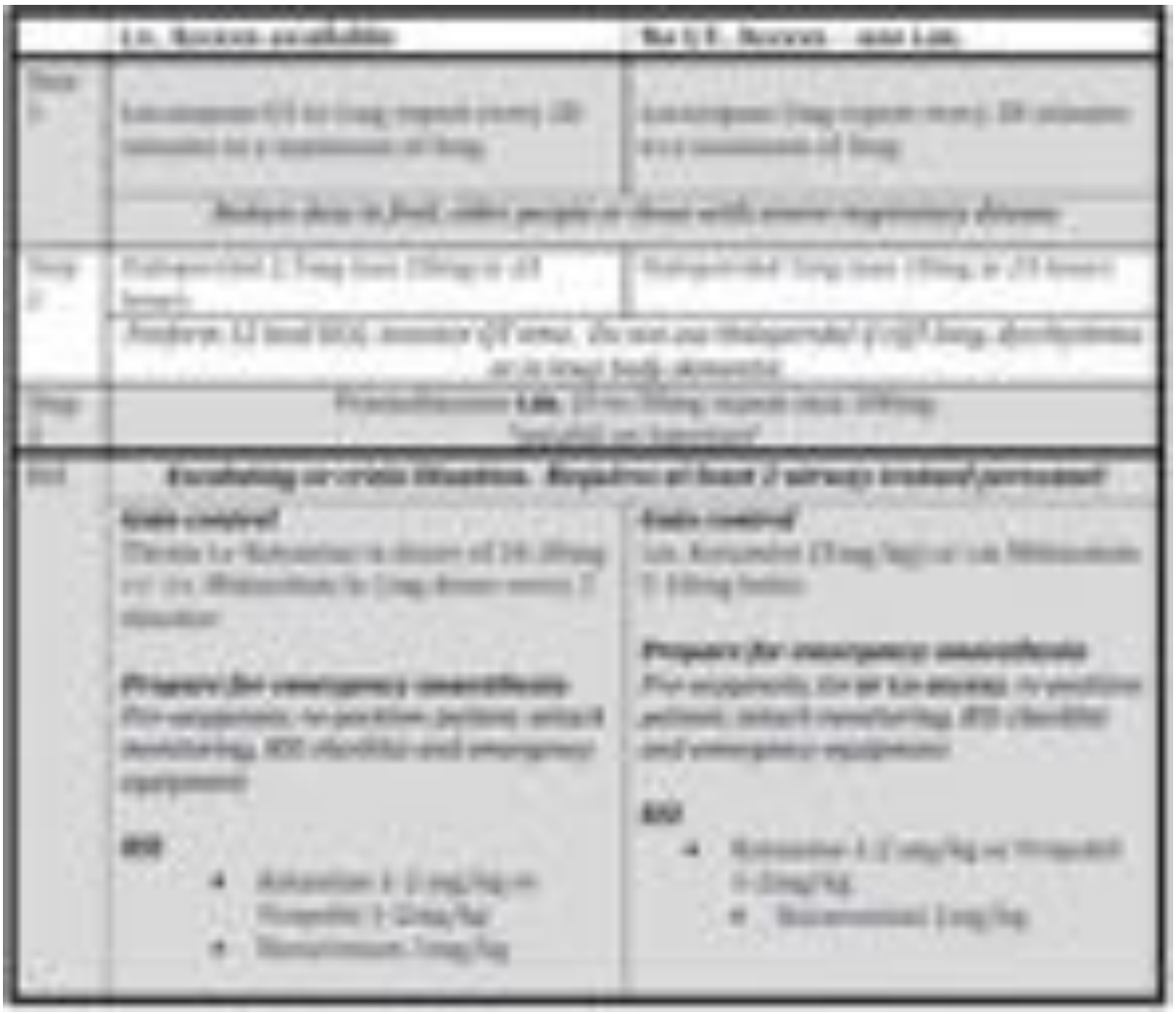
Action points on management:

- Physical restraint (1 person for each limb, 1 person for airway and 1 person to lead/manage)
- Call security
- Deploy staff to ensure: arrest/airway trolleys nearby and to obtain Rapid Tranquilisation protocol drugs and Rapid Sequence Intubation drugs.
- Ensure senior airway doctor present
- Inform psychiatry and ACCU consultant

Drugs used for emergency management of violence and aggression:

Drug	Lorazepam	Haloperidol	Promethazine	Ketamine
Mechanism of action	$GABA_A$ agonist	D_2, 5-HT_2, , α_1 adrenoreceptor antagonist	Sedating anti-histamine Inhibit peripheral and central H1 receptors	NMDA receptor antagonist
Side effects	Apnoea Withdrawal effects	Extrapyramidal effects, prolonged QTc	Lower seizure threshold Dysrhythmias Hypotension	Emergence delirium – hallucinations Increased ICPs (new data questions this) Liver injury
Interactions/CI	Marked respiratory weakness/depression CNS depression	Parkinsons and lewy body dementia Caution in CVS disease/dysrhythmia	Epilepsy	Pre-eclampsia Intra cranial mass

Local ACCU Rapid Tranquilisation Protocol:



Further reading

ACCU local Rapid Tranquilisation protocol on intranet and Box. Archary C, Wood A, Barrass L, Pennington J, Healey M. ACCU Guidelines 2018.

Section 5: Other key topics – Disclosure to the police

If Police have a suspect in custody for an offence leading to a patient's admission, there is a 24-hour timeframe to charge the suspect. Evidence taken from the patient may be what is required to meet the criteria for the charge. This is often why information is required quickly.

Injuries can be defined to the police in two ways:

Life threatening: An injury which has been the subject of a full and formal assessment by a Doctor and as the result of that assessment has been deemed to be, on the balance of probability, a critical and high risk to life.

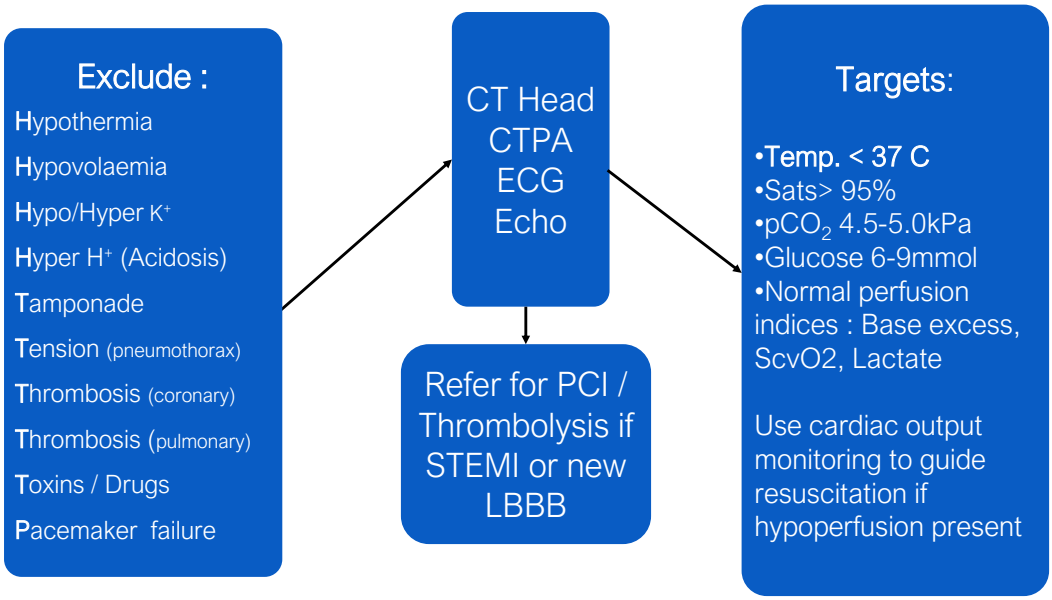
Life changing: Expected to result in the loss of a limb or is likely to result in a permanent significant physical impairment or disability which will prevent that person from being independent.

When talking to the police remember:

- Check the ID of the officer you are talking to.
- Abide by the confidentiality guidance toolkit from the GMC Guidelines on information disclosure.
- We have a duty not to impede the process of law.
- It is acceptable to provide a very broad and brief factual verbal report of the state of a patient. The officers can choose to write down your verbal report.
- Contact the Legal Department if you are uncertain what to do. An out of hours a legal service is available – contact the site manager.
- Any more detailed information should be sought in writing through the appropriate channels via the legal department.

Section 5: Other key topics – Post-cardiac arrest management

The complex pathophysiological processes that occur following whole body ischaemia during cardiac arrest and the subsequent reperfusion response have been termed the **post-cardiac arrest syndrome**. The treatment patients receive during the post-resuscitation period influences the overall outcome, particularly the quality of **neurological recovery.**



3 Elements to Post Cardiac Arrest Syndrome :

1) Myocardial Dysfunction – Early echo, inotropes / IABP support, BBs / ACEI once haemodynamically stable
2) Ischaemia - Reperfusion injury – SIRS response ➔ ↑CRP, risk of GIT translocation, MOF, may require vasopressor support
3) Neurological Prognostication – Poor prognostic features :
Clinical – absent pupil / corneal reflexes, generalised (face and limbs together) myoclonic jerks, GCS Motor score < 4 on days 1-3
EEG – unresponsive to external stimuli, generalised seizure activity, burst suppression or isoelectric trace after day 1
SSEP – bilateral absent N20 with normal N9 & N13 (confirming normal spinal cord / peripheral nerve function) after day 2
CT – loss of grey:white differentiation day 1
MRI – hyperintensity on DWI / FLAIR indicative of cytotoxic oedema partic. affecting cortex, thalami, basal ganglia, hippocampus, cerebellum on day 2-5

Further reading

1. Trust guidance can be found on box or ACCU protocol on shared drive
2. Resuscitation Council (UK) https://www.resus.org.uk/resuscitation-guidelines/post-resuscitation-care/

Section 5: Other key topics – Brain stem testing

Brain stem testing (diagnosis of death by neurological criteria)

Definition of death: the irreversible loss of the capacity for consciousness, combined with irreversible loss of the capacity to breathe

Although there are no universal criteria for the **determination of brain death**, codes of practice around the world are very similar and require (at least) these three elements:

1. The presence of a aetiology known to produce severe and irreversible brain damage
2. Exclusion of potentially reversible contributions to a state of apnoeic coma
3. Determination by clinical examinations, that there is profound unresponsive coma, apnoea, and the absence of brainstem reflex activity. (Clinical interrogation of the brainstem serves to demonstrate the absence or otherwise of brainstem functions)

In the UK death by neurological criteria is performed in keeping with recommendations made by the Academy of Medical Royal Colleges, *A Code of Practice for the Diagnosis and Confirmation of Death.* There is a **form for the Diagnosis of Death using Neurological Criteria {abbreviated guidance version}** that specifically details the process of brainstem death testing and should be used for all cases at the RLH.

Two doctors should carry out the tests; one of these doctors should be a consultant and both should have been fully registered with the GMC for at least 5 years. The tests must be undertaken by the two doctors together and completed successfully on two occasions.

Equipment that may be required

- Thermometer (to ensure patient normothermic)
- Pen torch (to assess pupillary response)
- Otoscope (to visualise the ear drum prior to performing the caloric testing)
- 50ml syringe of ice cold water (to perform caloric testing to assess vestibulo-occular reflex)
- Sterile gauze to assess corneal reflexes
- Tongue depressor
- Waters-circuit to do the apnoea test

Ancillary tests

The UK Code recognizes that there may be circumstances where clinicians feel unable to confirm brain death based on clinical assessment alone. Ancillary tests can be used - these tests are generally aimed at confirming the presence or absence of cerebral blood flow or cerebral function and commonly include EEG, cerebral angiography, CT angiography.

Section 5: Other key topics – Organ donation after brainstem death

Organ donation is possible in most situations where brain death has been confirmed (provided consent is given)

Contraindications:

- Primary intra-cerebral lymphoma
- Active malignancy within 3 years of donation
- Melanoma
- Active haematological malignancy
- Definite, probable or possible CJD
- TB
- West Nile Virus
- HIV disease (not HIV infection)

How to organise

The Specialist Nurse in Organ Donation (SNOD) is responsible for co-ordinating the organ donation process from consent to last offices of the deceased in the operating theatre.

You should refer any patient who is a potential organ donor early in their hospital admission. Referral to the SNOD is through the extension 40336 or 07659100103 or via switchboard (they are available 24hours a day).

Donor Optimisation

Brain death is often associated with marked physiological instability, which, if not managed, can lead to deterioration in organ function before retrieval. Moderation of these pathophysiological changes by active management can maintain organ function, thereby increasing the number and functional quality of organs available for transplantation.

Priorities to address include:

1. Assess fluid status and correct hypovolaemia
2. Introduce vasopressin infusion and where required introduce cardiac output/flow monitor
3. Respiratory optimisation
4. Identify, arrest and reverse effects of diabetes insipidus
5. Administer methylprednisolone (all donors)

Typical target parameters:

- MAP 60-80mmHg
- PaO_2 > 10 kPa, pH > 7.25
- Temperature 36 - 37.5
- TV 4-8ml/kg
- Urine output 0.5-2ml/kg/hr
- Blood sugar 4-10mmmol

Further reading

1. Trust policies can be found on the ACCU shared drive or in 'box'
2. Academy of Medical Royal Colleges, *A Code of Practice for the Diagnosis and Confirmation of Death* http://www.aomrc.org.uk/wp-content/uploads/2016/04/Code_Practice_Confirmation_Diagnosis_Death_1008-4.pdf
3. John Oram, Paul Murphy; Diagnosis of death, *Continuing Education in Anaesthesia Critical Care & Pain*, Volume 11, Issue 3, 1 June 2011, Pages 77–81, https://academic.oup.com/bjaed/article/11/3/77/257231
4. D. W. McKeown, R. S. Bonser, J. A. Kellum; Management of the heartbeating brain-dead organ donor, *BJA: British Journal of Anaesthesia*, Volume 108, Issue suppl_1, 1 January 2012, Pages i96- i107 https://academic.oup.com/bja/article/108/suppl_1/i96/237125
5. Donor optimisation guideline for management of the brain-stem dead donor, NHSBT, 2012 http://odt.nhs.uk/pdf/donor_optimisation_guideline.pdf

Section 5: Other key topics – Organ Donation after cardiac death

The Specialist Nurse in Organ Donation (SNOD) is responsible for co-ordinating the organ donation process from consent to last offices of the deceased in the operating theatre.

You should refer any patient who is a potential organ donor early in their hospital admission. This includes patients with whom there is an intention to withdraw life-sustaining treatment, which will, or is, expected to, result in circulatory death.

Referral to the SNOD is through the extension 40336 or 07659100103 or contact via switchboard (available 24hours a day).

Specific guidance on Donation after Circulatory Death (DCD):

- SNOD will liaise with operating theatres to book theatre and to arrange the relevant staff.
- SNOD will liaise directly with the National Organ Retrieval Service (NORS) team members to co-ordinate their arrival at the RLH.
- When the NORS team are present and an operating theatre is available the patient should be transferred from the ACCU to the Anaesthetic Room with respiratory and cardiovascular support and accompanied by medical, nursing and SNOD staff (family may also be present).
- Palliative analgesia and sedation must be continued in accordance with the existing withdrawal of treatment plan. The dignity and comfort of the patient are of paramount importance.
- Once the NORS team are ready, treatment should be withdrawn by a medical member of ACCU who should remain with the patient until asystole.
- Death should be certified 5 minutes after circulatory death by ACCU medical staff.

Further reading

1. Trust policies can be found on the ACCU shared drive or in ‘box’