University Hospital of North Staffordshire NHS Trust Intensive Care Units

Clinical Guidelines version 7

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University Hospital of North Staffordshire

Contributors

Dr Bryan Carr
Dr Nick Coleman
Dr Stephan Krueper
Dr Paul Morrison
Dr Ross Kitson
Dr Sri Ngaiyan
Dr Peter Oakley
Dr Chris Thompson
Dr Ashley Miller
RN Ms Rebecca Amos

Neurocritical Care Guidelines consented with Mr. Simon Shaw (CD neurosurgery)

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Introduction

- The following guidelines are intended for use on the ITU and MIU at the University Hospital of North Staffordshire. They have been collated from a consensus opinion of the ICU consultants based on established local practice and current best evidence as well as various national protocols and standards.

- The remit is to provide a guide to the new trainee on the unit in managing the various problems particular to ICU. They are not a reference or full review of the literature and the depth in which they cover each topic will vary. Trainees are encouraged to read around the topics they encounter and to ask the ICM consultants about these topics.

Consultants

There are 8 Consultants providing cover to the Intensive Care and High Dependency unit (CGH) and the Multiple Injuries Unit (NSRI)

Dr B Carr	ICU / MIU Educational Lead
Dr N Coleman	ICU / MIU
Dr P Morrison	ICU / MIU
Dr S Krueper	ICU / MIU
Dr S Nagaiyan	ICU / MIU
Dr C Thompson	ICU / MIU / renal medicine
Dr P Oakley	MIU / Trauma / Neuroanaesthesia
Dr K Venkatesan	ICU/MIU

There is a duty consultant covering both the ICU/HDU and the MIU 08.00 – 18.00 Monday to Friday. On weekdays there is consultant cover for the HDU from 08.00 – 12.00, although this person will work flexibly across sites according to clinical need. Dr Oakley works a fixed day session on MIU only and does not cover ICU.

The duty consultant for each unit changes each week on a Monday morning. The duty consultant can be found on the anaesthetic rota. (via intranet- clinical section/divisional sites/anaesthesia/rotas.

Out of hours the consultant cover is provided by these consultants on a rota basis. The out of hours consultant can be found in the weekly emergency anaesthetic rota.

Nursing Staff

The team is led by Matron Karen Eptlett. She is responsible for all the nursing staff and practices within the units.

Patients are classed as level 3 (Intensive Care) and level 2 (High Dependency). During any one shift there will be one dedicated nurse per level 3 patient and one dedicated nurse per two level 2 patients. The provision of intensive care is totally dependant upon nursing staff and so the availability of beds reflects the availability of

nursing staff not physical bed space. Staff work flexibly between the three areas to accommodate the necessary patients.

Each shift is co-ordinated by a G grade nurse in charge who in liaison with the consultant manages the day to day running of the units.

Each shift also has a clinical support worker who whilst not directly responsible for any single patient will be involved in the care of all the patients.

Infection control
Infection control takes high priority. You will be expected to familiarize yourselves with the unit practices immediately which are:

- Safe working practice with regard to hand washing and use of alcohol gel
- Only wearing the designated coloured apron at the appropriate bed area and not on the nurses' station or elsewhere.
- Stethoscopes are provided at each bed area. You are requested not to use your own.
- Jewellery including wrist watches, bracelets and stoned rings are not to be worn in the clinical area.

Critical Care Outreach Team
In the Royal Infirmary there is an in hours service operating 0800-2000 hrs. Out of hours the nurse in charge on MIU carries the bleep in an advisory capacity. Patients at risk are identified to the team by ward staff using the PAR scoring tool. The team then advise and support the ward staff until the patient recovers or is moved to an appropriate area of care.

Follow up of all patients discharged from the MIU takes place for up to 48 hrs whereby the outreach team work with the ward staff to monitor the patients progress and help to settle them and their family into the ward environment.

Other staff
Technical support for the units is provided by Clinical Technology. Specific and urgent requirements are dealt with by Craig Shingler, Team Leader who is located on the ICU.

Physiotherapists visit each unit routinely and treat all patients where appropriate. This includes an evening service and weekends. Physiotherapy is available out of hours via switch board.

Dietetic advice can be sought if required on 2113 during the day. Other staff such as occupational or speech therapy are contacted when individual patients require it.

There is a regular weekly ward round with medical staff from the departments of Medical Microbiology and Infectious Diseases on a Wednesday on the ICU/HDU and Friday on the MIU

There is a dedicated pharmacist for both units who does daily rounds and is available for advice either in the Pharmacy or on bleep. For advice contact Lisa (bleep 470).

Section 1   Respiratory management

Section 1.1 Airway Management

- The majority of patients will come into the ICU already intubated with an oral endo-tracheal tube, or will require intubation shortly after admission.

- Intubation must only be carried out by personnel with appropriate training and experience. If you assess a patient and feel that they need respiratory support, call the consultant in charge for assistance.

- Tracheostomy is performed on patients who require ventilation for more than a few days, or in whom separation from the ventilator is thought unlikely without it.

- Tracheostomies are only performed by consultants or SPRs who have registered for ICU training.

- If either an endotracheal tube or a tracheostomy tube becomes dislodged, oxygenate the patient with a face mask and 100% oxygen and **call for help.**

- Do not try to reintubate unless you are experienced. Do not reinsert a tracheostomy tube as the danger of placing the tube outside the trachea is very high. Occlude the stoma with a dressing, **call for help** and oxygenate the patient with a face mask.

Section 1.2 Oxygen Therapy.

There are a number of devices available to deliver oxygen. (shown below).
Some provide a fixed inspired oxygen concentration, while in others the inspired
concentration will vary.

In general , use the lowest inspired oxygen concentration to achieve an adequate Pa02
(usually 8 KPA but depends on the patient)

Apparatus/Device	Oxygen flow (l/min)	Approximate F_IO_2 (%)
Nasal catheters	2 4 6	28 35 45
Semi - rigid masks (eg Hudson, CIG)	5 6 8 10 12	35 50 55 60 65
Venturi type mask (eg Ventimask, Accurox)	2 - 8	24 - 50 (per manufacturer)
Reservoir plastic masks (eg Non rebreathing bag)	6 - 15	FiO_2 = 21% + 4% per l/min
Anaesthetic circuits	Variable	21 - 100
Ventilators (include CPAP)	Variable	21 - 100
Oxylog	Variable	Airmix : 60 No airmix 100

Section 1.3   Mechanical Ventilation

- All new resident staff should attempt to understand the indications, complications, and practical aspects of mechanical ventilators and respiratory failure.

- They should familiarise themselves with the ventilators and understand the default settings and common modes of ventilation.

- However, incorrect ventilatory settings can cause serious morbidity or even mortality - if you have any questions about the ventilatory strategy of any patient, **ask the consultant in charge**.

- If you make any changes to the ventilators, notify the nurse at the bedside.

- The default FiO2 for any patient initially connected to a ventilator is 1.0 (100%). This is only changed after the first blood gas, which should be done as soon as possible.

1. 31   Indications for mechanical ventilation

The list of indications is long. Common ones include:

- Respiratory failure (failure of oxygenation, carbon dioxide clearance, or both)
- Control of intracranial pressure in head injury
- Airway protection following drug overdose
- Following cardiac arrest
- For recovery after prolonged major surgery or trauma

Criteria for starting mechanical ventilation are difficult to define and the decision is often a clinical one. Indicators include:

- Respiratory rate >35 or <5 breaths/ minute
- Exhaustion, with laboured pattern of breathing
- Hypoxia - central cyanosis, SaO_2 <90% on oxygen or PaO_2 < 8kPa
- Hypercarbia - $PaCO_2$ > 8kPa
- Decreasing conscious level
- Agitation in some patients.

Clinical assessment is the most important means of diagnosing respiratory failure. Do not delay institution of ventilation waiting for results of blood gases or mechanical measurements if the clinical condition warrants.

1. 32 Types of Ventilation

A detailed description of ventilators is beyond the scope of these guidelines. Fundamentally, there are two broad approaches to ventilation. Volume or pressure.

- Volume-controlled ventilation occurs when the ventilator delivers a preset tidal volume regardless of the pressure generated.
    - The 'constant' is volume, and the 'variable' is pressure.
    - The lung compliance (stiffness) of the lungs determines the airway pressure generated, so this pressure may be high if the lungs are stiff, with the resultant risk of barotrauma (rupture of the alveoli resulting in pneumothoraces and mediastinal emphysema).
    - SIMV is the volume mode on the ventilators in the ICU.

- Pressure-controlled ventilation is where the ventilator delivers a preset target pressure to the airway during inspiration.
    - The constant is pressure, and the variable is volume.
    - The resulting tidal volume delivered is therefore determined by the lung compliance and the airway resistance.
    - BILEVEL is the pressure mode on the ventilators in the ICU.

Unless indicated by the consultant in charge, SIMV is the first choice mode for any new admission. Initial settings should be as follows:

Initial ventilator settings:	
FiO$_2$	1.0 initially but then reduce
PEEP	5 cm
H$_2$O	Tidal volume 6-8 ml/kg IBW
Frequency	10 - 15 breaths per minute
Pressure support	20 cmH$_2$O (15cmH$_2$O above PEEP)
Inspiratory flow	60 litres per minute
Flow trigger	2.5 l/min

Alterations will need to be made as the patient's condition changes. Always consult with senior medical staff before making changes to a patient's ventilation.

Section 1.4   Specific Conditions.

1.41 Acute lung injury and Acute respiratory distress syndrome (ARDS)

Definitions

Acute lung injury is defined as a syndrome of acute lung inflammation with increased vascular permeability, characterized by the following:

- Bilateral diffuse pulmonary infiltrates on chest radiograph
- PaO_2 / FiO_2 < 40 kPa, irrespective of the level of PEEP
- No clinical evidence of elevated left atrial pressure or pulmonary capillary
- wedge pressure <18 mmHg

ARDS is defined as a syndrome of acute lung inflammation with increased vascular permeability, characterized by the following:

- Bilateral diffuse pulmonary infiltrate on chest radiograph
- PaO_2 / FiO_2 < 26 KPa, irrespective of the level of PEEP
- No clinical evidence of elevated left atrial pressure or pulmonary
- capillary wedge pressure <18 mmHg

ARDS and ALI represent the same disease spectrum but differ in severity

Causes of ARDS/ALI:

Direct Pulmonary injury

- Pneumonia
- Aspiration of gastric contents
- Pulmonary contusion
- Fat emboli
- Near-drowning
- Inhalation of toxic gases

Indirect Pulmonary injury

- Sepsis
- Severe trauma
- Massive transfusion of blood
- Cardiopulmonary bypass
- Drug overdose
- Acute pancreatitis

Management

- Patients with ARDS require a modified approach to ventilation that concentrates on limiting ventilator-induced lung injury by reducing tidal volume and plateau pressure. This must always be discussed with a consultant

- This can be achieved using either SIMV or BILEVEL modes

    The important goals are

    - Search for and treatment of disorders precipitating ARDS/ALI eg pneumonia
    - low tidal volume, appropriate PEEP and permissive hypercapnia
    - Low tidal volume to reduce "volutrauma", barotrauma
    - PEEP to reduce cyclic recruitment/derecruitment ("atelectrauma") of lung units and redistribute lung water

        - Levels of PEEP will be set by the consultant in charge. Opinion varies, but the following is a reasonable guide:

Fi02	PEEP (cm H20)
0.3	5
0.4	8
0.5	10
0.6	10
0.7	12
0.8	14
0.9	16
1.0	18-20

- Permissive hypercapnia – frequent consequence of ventilation with low tidal volumes. Do not try to reduce CO_2 back into the 'normal range'

- tidal volume 6ml/ Kg Ideal Body Weight
  Males = 0.91 x (height [cm] – 152.4) + 50
  Females = 0.91 x (height [cm] – 152.4) + 45.5

- Plateau pressure < 30 cm H20
- Deep sedation and occasionally paralysis is required to ensure adequate patient-ventilator synchrony.

On BILEVEL, the tidal volume will vary, so increases in tidal volume should prompt a reduction in 'High PEEP'
On SIMV, the volume is fixed.

1.42   Asthma

Patients with asthma have a low mortality on the ICU, but ventilation is the source of most morbidity and mortality. 'Normal' blood gases do not apply in these situations, and attempts to bring C02 into the 'normal' range can be disastrous.
A detailed description is beyond the scope of these guidelines

However the following is a useful starting point:

- **Initial settings**

  - SIMV
  - Tidal volume: 6 ml per kg body weight
  - SIMV rate 8-10 per min
  - I:E ratio: 1:4
  - Pause time = 0
  - PEEP=0

  - Increase Peak pressure alarm to appropriate level if the plateau pressure is not high e.g. 60 cm H2O *

Every 4 hours and after changes in ventilatory settings

- Measure   intrinsic PEEP (if patient is sedated and paralyzed) by pressing the end-expiratory breath hold button
- plateau pressure by pressing the end-inspiratory breath hold button
- Aim for intrinsic PEEP < 10 cm water and plateau pressure <25 cm water

Intrinsic PEEP may be decreased by:

- decreasing respiratory rate
- decreasing tidal volume
- increasing inspiratory flow   rate
- Bronchodilator therapy

Permissive hypercapnia is an important tool in the safe ventilation of asthmatics

All settings and adjustments must be approved by a consultant.

Section 1.5 Non-invasive Positive Pressure Ventilation (NIPPV)

Indications:

- Acute hypercapnic respiratory failure during acute exacerbations of COPD
- Acute respiratory failure due to cardiogenic pulmonary oedema
- Acute hypoxemic respiratory failure in immunocompromised patients
- Facilitation of weaning in patients with COPD

Contraindications to NIPPV

- Cardiac or respiratory arrest
- Non-respiratory organ failure e.g. encephalopathy with GCS < 10
- upper gastrointestinal bleeding and haemodynamic instability
- Facial trauma, injury and deformity
- Upper airway obstruction
- Uncooperative patient
- Unable to protect airway
- Unable to clear sputum
- High risk of aspiration

Protocol for NIPPV

- Sit patient up
- Explain to patient about NIPPV and what to expect
- Hold the mask over the patient's face gently
- Start with low inspiratory pressure (IPAP): 8 – 10 cm water and expiratory pressure (EPAP):5 cm water

- Gradual increase in IPAP as tolerated by patient up to 20 cm water
- Observe for change in respiratory rate, tidal volume, signs of respiratory distress

- Adjust FiO2 to maintain SpO2 > 90%
- Recheck arterial blood gases within 2 hours after application of NIPPV
- EPAP may be increased in cases of acute pulmonary oedema
- Apply strappings to the mask after the patient has get used to NIPPV
- Dressing eg Duoderm may be applied to nasal bridge or other pressure point to avoid the development of pressure sores

    For patients with cardiogenic pulmonary oedema without hypercapnia,

- CPAP 8 – 15 cm water via face mask can be tried.
- The FiO2 can be adjusted according to the arterial blood gases and SpO2

Contraindications to NIPPV might develop while patients on NIPPV e.g.
change in conscious state, vomiting. Conversion to invasive should be considered
after discussion with the consultant in charge.

Section 1.6   Weaning from Ventilation

Weaning is the process of gradual discontinuation of mechanical ventilation.

Patients with normal conscious state, respiratory mechanics and stable
haemodynamics do not need prolonged weaning and can be quickly extubated after a
brief trial of spontaneous ventilation. 30 mins is sufficient.

Patients who have had a longer ICU stay, chronic pulmonary disease, an underlying
neuromuscular problems or who have been severely ill may benefit from weaning

.

Is the patient ready for weaning?

- Precipitating factors for respiratory failure are under control
- sedation is not required
- Normal conscious state
- Adequate cough and gag reflexes
- Stable haemodynamics requiring minimal or no vasopressor or inotropes

If the patient is thought ready for weaning, the following algorithm is a useful
summary:
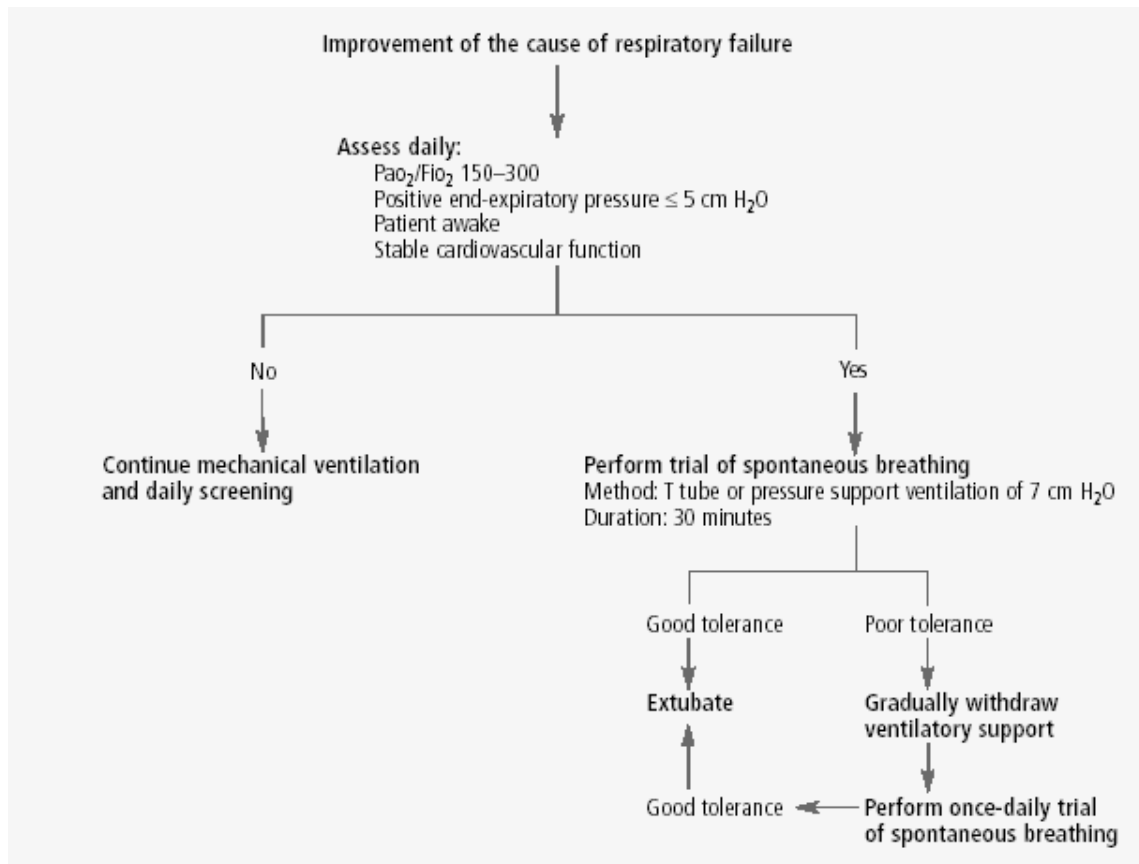
Failure of a spontaneous breathing trial can be identified by the following:

- Tachypnea (respiratory rate, > 35 for ≥ 5 min);
- Hypoxemia (oxygen saturation , $< 90\%$);
- Tachycardia (heart rate, > 140; or sustained rate increase $> 20\%$);
- Bradycardia
- Hypertension (systolic BP, > 180)
- Hypotension (systolic BP, < 90)
- Agitation, sweating etc

Rapid shallow breathing is associated with failure. The ratio of frequency in breaths per minute to tidal volume in litres (f/VT) is often used as a guide. A value of >105 is often cited as an indicator of failure.

1. 61 Pressure Support weaning

Patients who fail a spontaneous breathing trial require a more gradual withdrawal of ventilatory support.

- Patients should be established on Pressure Support Ventilation so that all ventilatory efforts are patient- initiated.
- Pressure support should be initially set at 20cm cm of water, and reduced by 2 cm of water at least twice a day.

- Pressure should be titrated to achieve a frequency of <25 breaths per minute.
- The pace of reduction can be increased if the patient has no signs of distress
- Patients on < 12 cm pressure support should have a spontaneous breathing trial

1.62   Factors contributing to difficulty in weaning

- Patient anxiety
- Over sedation or withdrawal
- Hypothyroidism
- critical illness polyneuropathy / Myopathy
- Electrolyte disturbances
- Malnutrition
- Unresolved sepsis
- Shock
- Bronchospasm
- Obesity
- Abdominal distension with diaphragmatic splinting
- Pleural effusion
- Pulmonary oedema
- Pneumonia
- Atelectasis

Section 1.7   Arterial blood gases

Normal values:

§   Arterial pO_2 with 21 % O_2: 9.3 kPa (elderly) to 13.3 kPa (young adults)
§   Arterial pCO_2 :   4.7 – 6.0 kPa

Oxygenation:

As inspiratory pO_2 is decreased by water vapour and CO_2 along the way down to the alveoles the alveolar pO_2 is roughly 10 kPa lower than the inspiratory pO_2.
In healthy persons the alveolar – arterial difference (AaDO2) is age - dependant in the range of 0.7 – 2,7 kPa (with 21% O_2) and 1.3 – 8.7 kPa (with 100% O_2).

As 1% normobaric oxygen   is approximately 1 kPa, it can , as a ruel of thumb, be said, that the arterial pO_2 (in kPa) in a person with normal lungs should be roughly :

Inspiratory O_2 concentration (%) - 10

(i.e with 60 % inspir. O_2 the arterial pO_2 should be around 50 kPa).

A method of describing the quality of oxygenation or quantifying the degree of an oxygenation failure, is the calculation of the pO2 / FiO2 ratio:

Normal value: 9.3 kPa / 0.21 = 44 (elderly) to
                     13.3 kPa / 0.21 = 63 (young adults)

A decreased arterial pO2 usually reflects an intrapulmonary (most common) or extrapulmonary shunt effect.
Intrapulmonary shunt is usually due to collapsed or fluid-filled alveoles and/or interstitiell infiltrates or edema and can often be improved by application of PEEP.
In the case of extrapulmonary shunt (e.g. septum defects with right-left shunt or PFO) PEEP will make the oxygenation worse as it increases the pulmonar - arterial- and right heart pressures and therefore the extrapulmonary shunt (unless there is an intrapulmonary shunt problem as well).

Ventilation

The qualitiy of ventilation (spontaneous or artificial) is reflected by the arterial pCO2.

Hypercapnia can be a symptom of very different clinical problems as:

Central: drug side effects (opioids)
                                      metabolic disorder with central hypoventilation
                                      brain injury or stroke
                                  spinal cord injury
                                  phrenic nerve paralysis
                                      oxygen triggered hypoventilation in COPD patients

Muscular :   Muscle fatigue in exacerbation of COPD
                                    Myasthenia gravis
                                    Residual effects of neuromuscular blocking agents
                                    Guillan Barre Syndrome
                                    Malignant hyperthermia (MH)
                                  Critical illness polyneuropathy

Perfusion problems: PE

| Lung conditions: | Bronchospasm |
| | Lung fibrosis |

Airway obstruction:	Foreign bodies, secretion, blood
	Swelling (edema, haematoma)
	Laryngospasm
	Recurrensparesis

The treatment obviously depends on the underlying problem.

CO2 narcosis:

pCO_2 levels above 8.0 kPa can cause neurological symptoms from drowsiness to coma.
Therefore assessments of patients consciousness level needs to take pCO_2 levels into account.

Section 1.8   Tracheostomy Care

1.81 General issues

- Tracheostomys are commonly used on ICU to
  - Facilitate weaning
  - Allow cessation of sedation whilst ventilation continues
  - Provide longer term airway access and protection in neurological impairment
  - Post operatively following major head and neck surgery
  - Rarely following emergency insertion

- Tracheostomy may be performed surgically (in theatre) or percutaneously (on the ICU)
  - Surgical tracheostomy is chosen when
    - § Difficult anatomy
    - § Previous neck surgery or tracheostomy
    - § Coagulopathy
    - § Concern regarding manipulation of the neck

- Percutaneous tracheostomy (PT) should be performed by a Consultant or Senior Intensive Care Trainee.

- A training package including a training video and manual is available and should be complemented by a study of the anatomy and observation of both surgical and percutaneous tracheostomy before performing the procedure under supervision.

- Currently two methods are employed

  - Griggs dilating forceps
  - Single dilator technique

- Consent cannot normally be sought from the patient. The patient's relatives should be informed of the procedure and it's risks (including bleeding and the need for surgical tracheostomy) and benefits.

- An Anaesthetist should provide anaesthesia for the procedure. Patients are usually sedated at time of tracheostomy and often increase in sedation with subsequent paralysis is all that is required.

- A carbon dioxide detector should be at the bedside. Bronchoscopy is utilised to visualise needle placement in the trachea and to assist in guiding the procedure.

- Tracheostomy involves risks of hypoxaemia and hypercarbia. As a result the patient must have reasonable ventilator settings prior to the procedure to minimise this risk and acceptable coagulation profile

  - $FiO2 < 0.4$
  - $PEEP < 8$ cmH2O
  - normal ICP (if monitored)
  - Platelets > 80
  - $INR < 1.5$
  - $APTTr < 1.5$

  There may be some flexibility with these parameters at the discretion of the consultant

1.82 Tracheostomy care

- Complications associated with tracheostomy include:

  Immediate
  Peri-operative e.g. haemorrhage, pneumothorax, surgical emphysema
  Misplacement of the tube in pre-tracheal tissues/right main bronchus
  Occlusion of tip of tube against carina/tracheal wall
  Accidental displacement of the tube

Recurrent laryngeal nerve injury

Delayed
Sputum plugging
Accidental displacement of tube
Bleeding
Infection
Tracheal distension
Mucosal ulceration
Tracheo-oesophageal or tracheo-vascular fistulas

Late
Granulation around tracheostomy tube
Tracheomalacia
Tracheal stenosis
Infection
Delayed healing of stoma
Revision of scar required

- Types of tracheostomy tube used on the unit include:

  - Portex
  - Flanged (adjustable)tracheostomy
  - Shiley (*fenestrated* with separate inner tubes)

- Typically Basic Portex tubes are inserted during PT and replaced after a week for Shiley tubes if the weaning is likely to be prolonged or if the tracheostomy is to be used on the ward.
- Flanged tubes may be inserted in theatre especially if the anatomy is difficult or the trachea deep. They are longer tubes with a variable fixation point to allow individualisation of the section of the tracheostomy tube that remains within the neck tissues. Care must be taken re their care and humidification.

- Fenestrated tubes allow airflow through the larynx and as such can facilitate weaning with cuff deflation. Patients at high risk of aspiration should not have a fenestrated tracheostomy. The changeable inner tube carries clear advantage in longer term weans by avoiding the necessity of repeated tracheostomy tube changes. However the use of an inner tube will reduce the diameter of the airway the patient ventilates through by 1-1.5 mm.

- Key aspects of tracheostomy care are:

  - Humidification
  - Suctioning practices
  - Change in dressings
  - Changes in inner cannula if present
  - Tube changes

1.83   Changes in tracheostomy tubes

- The literature describing the frequency of tracheostomy changes is sparse. It is our practice not to change tubes prior to the end of the first week. After this the need to change is individualised: it is typically performed prior to the patient going to the ward to allow use of a tube with a changeable inner, such as a Shyley.

- The first (and in a number of cases subsequent) tracheostomy tube change should be performed by an anaesthesist / intensivist.

- The following describes the method for changing the tube (extract from St George's hospital guidelines 2005):

Equipment
- Dressing pack
- Correct size tracheostomy tube and one size smaller within easy reach
- Tracheostomy tube holder
- 10ml syringe if tracheostomy tube is cuffed.
- Sterile water-soluble lubricant
- Sterile normal saline
- Pre-cut slim line key hole dressing
- Sterile or clean gloves, apron and protective eye wear
- Tracheal dilators and suction
- Functioning suction unit and appropriate sized suction catheters
- Stethoscope
- Resuscitation equipment

BOX 11.1 Changing a tracheostomy tube using an obturator

This is a two-person technique

Action	Rationale
Aspirate naso-gastric tube if present and keep patient nil by mouth for 3-4 hours prior to tube change	To reduce the risk of aspiration when the airway is unprotected
Two skilled practitioners should perform the procedure. Both must wear eye protection.	To ensure a swift and safe procedure
Explain the procedure and rationale to the patient. Inform the on-call anaesthetist of the time and place of the tube change	The patient should give their verbal consent to the procedure, unless unable or an emergency procedure. Ensuring the availability of an anaesthetist is safe practice should problems arise.
Wash hands and prepare dressing trolley	To reduce the risk of cross infection
Position patient in semi-recumbent position, extending the neck, removing any obstructive clothing.	Extending the neck will make the removal and insertion of the tube easier. To ensure adequate view of patients neck.
If the patient is dependent on oxygen, hyper-oxygenate the patient with 100% oxygen and monitor oxygen saturations. COPD patients should be assessed for necessity of hyper-oxygenation prior to delivery as they may only require an increase of their oxygen concentration of 20% (Day 2000)	During tube change the patient will not receive oxygen and will be at risk of hypoxia. Patients with COPD have an altered CO_2 response mechanism and should not routinely be given 100% O_2 flush (Day 2000).
If the new tracheostomy tube is cuffed, check the cuff by inflating it with air, and then fully deflate. Ensure a smaller tracheostomy tube is to hand	To check for air leaks and spontaneous deflation. In case the same size tube cannot be inserted easily.
Check obturator can be removed	To become familiar with removing obturator prior to insertion.
Lubricate the tube sparingly with a water soluble lubricant. Place on sterile surface.	To facilitate insertion, and maintain sterility
Remove old dressing and tapes, observe site and clean around stoma site	To clean skin of debris and potential skin contaminants. To enable removal of the tracheostomy tube
Suction patient if required. Suction oro-pharyngeal secretions. Using the synchronised cuff deflation and suction technique (see box 4.3) slowly deflate the cuff with a10ml syringe	Pooled secretions above the cuff may enter the lungs when the cuff is deflated. A fully deflated cuff reduces the risk of trauma on removal of the old tube.
Remove old tube in a firm upwards and downwards motion on expiration. Observe the stoma site.	To cause minimal trauma and reduce the risk of coughing. To identify signs of stoma infection and granulation tissue.
With clean gloves insert the new tracheostomy tube with the obturator in place as the patient exhales	The obturator guides the tracheostomy tube along the contour of the trachea. Relaxation of the neck muscles makes insertion easier.
Remove the obturator immediately	The patient is unable to breathe with the obturator blocking the lumen.
Inflate the cuff using the minimal occlusion technique (see box 4.1) and check with a hand pressure gauge (see box 4.2)	An inflated cuff reduces the risk of aspiration. Correct inflation reduces the risk of tracheal wall damage
Insert inner cannula if using a two piece system	To prevent secretions collecting on the inside of the outer cannula
Observe the patient for respiratory distress. Feel for respiration via the tube, where able, ask the patient to breath deeply and observe for chest movement	The flow of air is felt via the tracheostomy lumen if the tube is correctly positioned
Auscultate for equal air entry	To ensure bilateral inflation of the lungs
Clean stoma site if required, renew dressing and secure tracheostomy tube with a tube holder.	To reduce the risk of dislodgement, maintain patient comfort and reduce the risk of infection.

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If the patient is comfortable and there are no signs of respiratory distress, return oxygen to levels prior to procedure and observe respiratory rate and oxygen saturation	To ensure patient is breathing satisfactorily
Record tube change in medical notes, document time, date, size, type of tube and any complications	To ensure effective communication.
There may be a small amount of bleeding post tube change. If there is excessive bleeding, ensure cuff is inflated and call for medical assistance immediately.	Trauma can occur to stoma site during a tube change. An inflated cuff will protect the patients airway whilst tamponading the bleeding.

If tube insertion fails or patient becomes compromised and cyanosed

- Call the on-call anaesthetist immediately
- Use tracheal dilator and attempt to reinsert same tube
- If this fails, attempt to insert smaller size tube
- If this fails administer oxygen via the stoma or maintain patient's airway and administer oxygen via a facemask until the anaesthetist arrives

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- As detailed in the next section if a stoma is lost then the priority is maintenance of oxygenation and maintenance of a patent (usually upper) airway. Oral re-intubation may be necessary (if anatomically feasible).

1.84   Tracheostomy emergencies

- Tracheostomies can produce a number of problems that may seem more unnerving than other airway problems due to the unfamiliraty of most trainees with the tubes. These can include:
    - tube occlusion
    - tube displacement
    - suspected displacement
    - haemmorage

- the following algorithims are taken from the St Georges guidelines. By the time the trainee is called many steps may have been taken by the nursing staff. **Have a low threshold for seeking senior help**.

- **Maintenance of oxygenation and maintenance of patent airways are the priority.**

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                        ┌─────────────┐
                        │  Suspected  │
                        │    Tube     │
                        │  Occlusion  │
                        └──────┬──────┘
                               │
      ┌────────────────────────▼────────────────────────┐
      │  • Do not panic                                 │
      │  • Reassure patient                             │
      │  • Call for expert help                         │
      │  • Commence high flow oxygen                    │
      │  • Stop NG feed                                 │
      └────────────────────────┬────────────────────────┘
                               │
                        ┌──────▼──────┐
                        │   Assess    │
                        │   patient   │
                        └──────┬──────┘
                               │
      ┌────────────────────────▼────────────────────────┐
      │  • Ask patient to cough or suction the          │
      │    patient                                      │
      │  • Manipulate tube or patient's head to         │
      │    eliminate kinking or compression             │
      │  • Remove inner cannula if present              │
      │  • Occlusion still present?                      │
      └──────┬───────────────────────────────────┬──────┘
             │                                   │
       ┌─────▼─────┐                       ┌─────▼─────┐
       │    YES    │                       │    NO     │
       └─────┬─────┘                       └─────┬─────┘
             │                                   │
```

- **YES column:**
  - Assume the tube is occluded
  - Call for expert help
  - Delete the cuff to allow patient to breathe around tube
  - If this fails, remove occluded tube and either attempt to insert a new tube OR
  - Remove tracheostomy tube, occlude stoma and ensure patient can breathe through upper airway
  - Await medical assistance
  - Deliver high flow oxygen via tracheostomy or facemask whichever is patent until patient is recovered
  - Proceed to BLS if cessation of breathing occurs

  NB: If the patient has a disruption between the upper and lower airways ventilation must be through the tracheal stoma

- **NO column:**
  - Replace inner cannula
  - Check patient's respiratory status
  - Check if patient remains distressed, the whole tube may need replacing
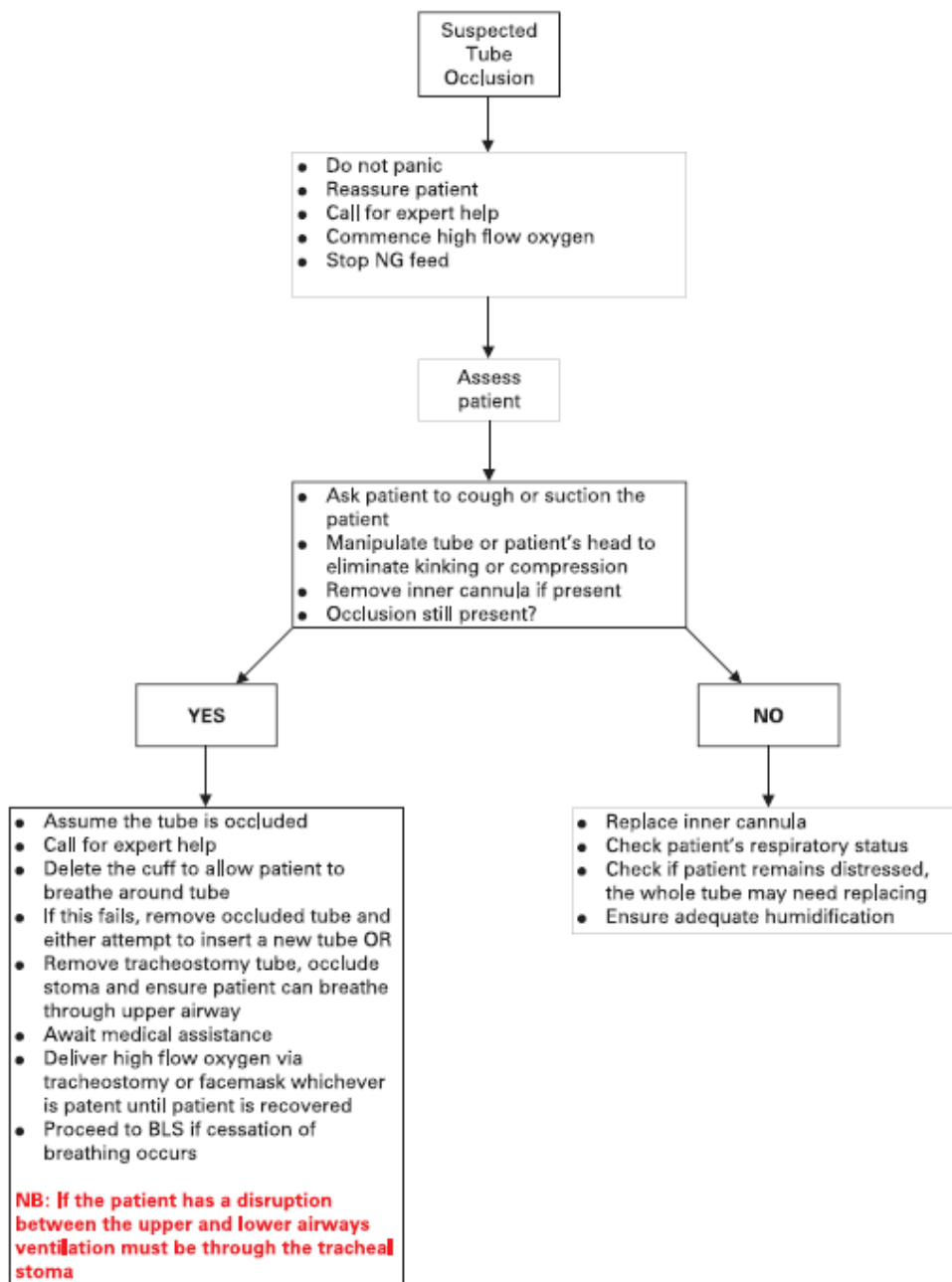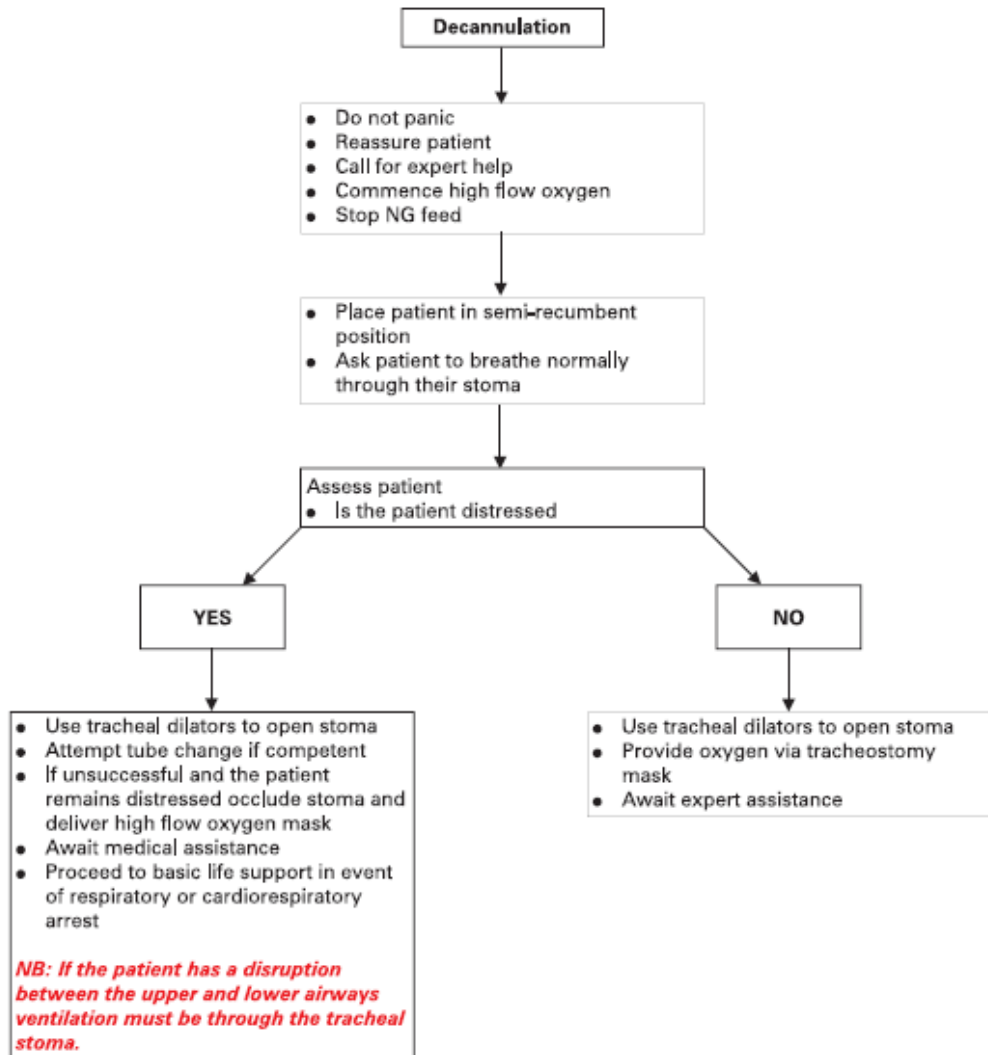 - Ensure adequate humidification

Figure 13.3 Tube Occlusion Emergency Algorithm

- **Process for suspected tube occlusion**. If in doubt do not persist with an occluded tube in a de-saturating patient, remove the tube and maintain airway and oxygenation via the upper airway.

```
                        ┌─────────────────────┐
                        │    Decannulation    │
                        └─────────────────────┘
                                   │
                                   ▼
        ┌──────────────────────────────────────────────┐
        │  • Do not panic                              │
        │  • Reassure patient                          │
        │  • Call for expert help                      │
        │  • Commence high flow oxygen                 │
        │  • Stop NG feed                              │
        └──────────────────────────────────────────────┘
                                   │
                                   ▼
        ┌──────────────────────────────────────────────┐
        │  • Place patient in semi-recumbent           │
        │    position                                  │
        │  • Ask patient to breathe normally           │
        │    through their stoma                       │
        └──────────────────────────────────────────────┘
                                   │
                                   ▼
        ┌──────────────────────────────────────────────┐
        │  Assess patient                              │
        │   • Is the patient distressed                │
        └──────────────────────────────────────────────┘
              │                                  │
              ▼                                  ▼
        ┌──────────┐                        ┌──────────┐
        │   YES    │                        │    NO    │
        └──────────┘                        └──────────┘
              │                                  │
              ▼                                  ▼
```

YES
- Use tracheal dilators to open stoma
- Attempt tube change if competent
- If unsuccessful and the patient remains distressed occlude stoma and deliver high flow oxygen mask
- Await medical assistance
- Proceed to basic life support in event of respiratory or cardiorespiratory arrest

NB: If the patient has a disruption between the upper and lower airways ventilation must be through the tracheal stoma.

NO
- Use tracheal dilators to open stoma
- Provide oxygen via tracheostomy mask
- Await expert assistance

- In the critical care setting decannulation may occur accidentally via the staff or via the patient. If the patient is on IPPV then maintain the airway and oxygenation via a bag and mask and consider
  - Whether to attempt re-insertion of trachy (only if > 7 days old and you are skilled)
  - Whether to occlude the stoma and re-intubate the patient
  - Whether to manage with the patient breathing spontaneously

```
                    ┌──────────────────┐
                    │    Suspected     │
                    │   Displacement   │
                    └──────────────────┘
                             │
                             ▼
```

- Do not panic
- Reassure patient
- Call for expert help
- Commence high flow oxygen
- Stop NG feed

```
                    ┌──────────────────┐
                    │     Assess       │
                    │     patient      │
                    └──────────────────┘
```

- Is the patient distressed?
- Can the patient speak and the tube is not capped?
- Are you <u>unable</u> to pass a suction catheter down the tube?

YES

- Assume the tube is displaced
- Call for expert help
- Deflate the cuff and attempt to reposition the tube if competent
- If this fails either attempt to insert a new tube OR
- Remove tracheostomy tube, occlude stoma and ensure patient can breathe through upper airway
- Await medical assistance
- Proceed to BLS if cessation of breathing occurs

NB: If the patient has a disruption between the upper and lower airways ventilation must be through the tracheal stoma.

NO

- Assume tube is not displaced
- Call for medical review
- Ensure the patient is able to breathe
- A check chest x-ray may be performed to confirm correct position of tube
- Attempt to establish alternative cause of respiratory compromise

- this algorithm is similar to previous. If you suspect displacement the options to check patency are attempting to pass suction catheter or placing on a bag-valve circuit and observing for bag movement. If available fibre-optic bronchoscopy may be used to assess patency.

Section 2   Cardiovascular Management

Section 2.1 Shock – basic principles

2.11 Recognition of shock

Shock can be defined as a state of inadequate organ perfusion. It ma y occur due to a variety of aetiologies which may co-exist:

- Cardiogenic shock   e.g. post MI, post cardiac surgery
  - o Obstructive shock   e.g. cardiac tamponade, massive PE
- Hypovolaemiac shock   e.g. massive fluid loss
  - o Haemmoragic shock
- Anaphylactic shock
- Septic shock

It may be recognised by a constellation of features the occurrence of which may be influenced by co-morbidities, patient physiology (e.g. the young compensate very well until volume loss is pronounced and then decompensate rapidly) and medication (e.g. beta blockade).

Typical signs include: Cold, clammy, sweaty, drowsy, confused, tachycardia, tachypnoea, hypotensive, acidotic, oliguric.

2.12 Management of Shock

2.121   Immediate Treatment:

- *Airway, Breathing, Circulation,*

- Airway: Ensure adequate airway especially in the unconscious patient.

- Breathing: Ensure adequate oxygenation and gas exchange.
  100% oxygenation until full assessment can be made
  Assist ventilation if unconscious and rate < 10 or > 30

- Circulation: Establish large bore IV access and institute fluid therapy based on likely aetiology.
  The majority of patients will require fluid resuscitation unless the diagnosis is clearly cardiogenic shock and/or LVF. Even then cautious fluid replacement may be required.

- Exclude or consider the following (ALS guidelines) before pursuing more obscure diagnosis:

Hypoxia	Tension Pneumothorax
Hypovolaemia	Tamponade
Hyper/hypokaleamia, metabolic disorders	Toxic/therapeutic disturbances
	Thromboembolic/mechanical

2.122 Subsequent management

- Proceed with cardiovascular resuscitation as follows

  1. ensure a filled intravascular compartment with optimal haemoglobin
  2. Ensure optimal cardiac output
  3. Ensure optimal perfusion pressure

  o *Ensure a filled intravascular compartment with optimal haemoglobin*: This can be achieved by assessing the patient's response to a fluid challenge either clinically or with invasive monitoring.

  o *Ensure optimal cardiac output*: Either inferred from clinical examination or if high levels of inotropic support are required confirmed by a direct measure of cardiac output using a PICCO monitor or the oesophageal doppler (**Discuss with ICU Consultant at this stage**). In addition central $ScvO_2$ has been used as a surrogate for determining need for increased cardiac output in sepsis.

    The most useful information is often found from observing the response to treatment in particular fluid resuscitation.

    In the emergency situation when the underlying picture is unclear use adrenaline 4 mg in 50 mls 5% dextrose or 0.9% saline titrated to effect until therapy can be guided by haemodynamic monitoring.

    This should be changed to noradrenaline, dobutamine or both as soon as possible guided by clinical assessment or monitoring.

    In a low cardiac output state use dobutamine 250 mg in 50mls 5% dextrose at 0-20 mls/hr, if high cardiac output use noradrenaline 8 mg in 100 mls 5% dextrose and titrate to effect.

- o *Ensure optimal perfusion pressure*. If perfusion pressure remains low in the face of the above start noradrenaline 8 mg in 100 mls 5% dextrose or normal saline and titrate to effect.

- Specific treatments may be aimed at specific types of shock i.e. steroids and activated protein C for septic shock , revascularisation and intra-aortic balloon pump in the face of cardiogenic shock after an acute myocardial infarction and surgery and fluids and blood products for haemorrhagic shock.

2.123 Optimal values for resuscitation

- There is a large amount of literature on goal directed therapy aiming for different endpoints, using different monitoring and with differing outcomes. In summary it would appear that patients are more likely to benefit from being treated and resuscitated *early* in the course of their illness than being left moribund for many hours and then receiving resuscitation. Once in established multi organ failure then goal directed therapy has not been shown to reduce mortality.

- On a more philosophical basis we all must have goals when treating our patients whether it be that we are aiming for a patient with warm peripheries, well oxygenated, well perfused, good urine output and normal lactate or the use of goals from small randomised trials such as CVP > 8 -12 mmHg, Central $ScvO_2 > 70\%$ (mixed venous saturation $SvO_2 > 65\%$) and mean arterial blood pressure > 65 mmHg urine output > 0.5mls/kg/hr used by Rivers in a trial on acute sepsis.

- This highlights the need during resuscitation which may go on for hours to frequently review the following parameters: Peripheral perfusion, urine output and lactic acidosis.

- Peripheral perfusion can readily be assessed by checking the lower limb temperature and noting at what level there is a warm/cold demarcation. The core peripheral temperature will normally decrease over an 8 hour period on re-warming following surgery. The rate of re-warming can be increased with warm fluids and forced air heating.

- Urine output should be at least 0.5 mls/kg and is a useful sign that the renal bed is being perfused as long as diuretics (including dopamine) are not administered.

- In the poorly perfused patient lactate levels will rise and their fall to normal is a good prognostic sign. It should be noted that lactate metabolism is complex and production may be increased by adrenaline, stress or severe liver dysfunction.

- The values for blood volume, haemoglobin, cardiac output and perfusion pressure will depend on the underlying disease process and the patients pre-existing state. These are discussed below:

2.124 Determination of Optimal Blood Volume

- **See appendices for details of CO monitors**

- To determine if the intravascular compartment is full or not the simplest assessment is clinical. This is often followed by use of a CVP. Placing a CVP in a patient who is clinically hypovolaemic apart from enabling access is of no benefit and should be preceded by fluid resuscitation.

- A single CVP measurement is not felt to be a good measure of volume status but initially a CVP of greater than 8 in the non ventilated patient and 10-12 in the ventilated patient should be aimed for. Following this optimal volume expansion is generally assumed when there is a sustained rise in CVP of 3 mmHg or more after a fluid challenge. Despite this both CVP and PAOP are poor at predicting fluid responsiveness.

- The most accurate measure of volume responsiveness is the pulse pressure variation. (The ΔPP is calculated as the difference between the maximal and the minimal value of pulse pressure over a single respiratory cycle, divided by the mean of the two values, and expressed as a percentage. %PPV = (PPmax - PPmin)/[(PPmax + PPmin)/2] *100%. This only holds true if the patient is fully ventilated with no arrhythmias.

- Pulse pressure variation is calculated by the PICCO. A figure of over 11% - 13% suggests there is room for further fluid resuscitation.

- The oesophageal doppler can be used to assess fluid status using the corrected flow time (FTc) and estimated stroke volume. An FTc of 340-360 reflects adequate blood volume however this can be further assessed by seeing if the stroke volume increases by more than 10% in response to a fluid challenge. (*See Oesophageal Doppler in Appendices*)

- The PICCO will also give an indication of blood volume and extra vascular lung water which are also used to determine further fluid requirements as outlined by their algorithm. (*see PICCO in appendices*)

- Again other parameters should be taken into account such as the state of the peripheral circulation, urine output and change in lactic acidosis although these will not show an immediate change with adequate fluid volume. Having obtained an optimal filling pressure the fluid status should continue to be assessed particularly when the patient requires inotropes.

- **Optimal Haemoglobin Concentration.**

  A study has not shown any major benefit between aiming for a haemoglobin of 7-9 or 10-12 in the critically ill. There are a number of papers suggesting that patients with ischaemic heart disease may benefit from a higher haemoglobin concentration. We would therefore aim for a haemoglobin greater than 7 unless evidence of heart disease (see section 2.4).

2.125   Increasing Cardiac Output:

- First establish and treat the cause:

  - Hypovolaemia.
  - Poor contractility – ischaemic heart disease, cardiomyopathy, myocardial depression secondary to sepsis.
  - Tamponade
  - Pulmonary embolus
  - Arrythmias

- If when using inotropes to increase cardiac output there is excessive tachycardia, tachyarrythmias or ST depression then the patient may have myocardial ischaemia and the choice of inotrope and its use must be reconsidered.

- What is the optimal Cardiac Output?

  - The optimal cardiac output will vary depending on the individual patients pre-existing cardiovascular status and the underlying pathology.

  - A cardiac index that is within normal limits 2.5 - 3.5 and is associated with a normal blood pressure and perfusion for the patient is acceptable.

  - Using inotropes to increase it further in the face of an acute myocardial infarction is likely to increase myocardial workload and ischaemia and in this situation if the patient is under-perfused a balloon pump should be considered (**Discuss with consultant and with Cardiologist**)

  - Surgical and Trauma patients are known to have a catabolic response to their insult and some studies have suggested that a cardiac index greater than 4.5 is associated with better survival.

  - In sepsis, trials have not been able to demonstrate any improvement in outcome compared to aiming for a cardiac index of 3.5 as compared to 4.5. The cardiac output should only be increased if low to reverse clinical signs of hypoperfusion and not to chase specific targets.

2.126   Optimal perfusion pressure.

  - The optimal perfusion pressure is generally considered that at which the patient normally functions at. To achieve this the above steps should first be followed prior to raising the pressure with noradrenaline. When using noradrenaline its effect on cardiac output and perfusion must be monitored. If the patient is refractory to noradrenaline then Vasopressin should be introduced at a dose of 0.01 -0.04 units per minute (0.6 to 2.4units/hr). Note vasopressin may cause a decrease in cardiac output and its use must be

carefully monitored.

o In sepsis it is often associated with slowing of the heart rate and improvement of perfusion. However if used in patients with poor cardiac contractility it can dramatically reduce cardiac output and in sepsis significantly worsen perfusion if the patient is underfilled.

o In cardiogenic shock lower perfusion pressures than normal may be accepted (see cardiogenic shock) whereas higher perfusion pressures may be required in patients with head injury to maintain cerebral perfusion pressure or when there is concern regarding cerebral vasospasm (see section 6 : Neurosurgical guidelines).

Section 2.13 Cardiogenic shock

- The major cause is inadequate myocardial function related to ischemic heart disease. In this situation optimising fluids can still improve function particularly with right heart failure and also following institution of ventilation. In addition improvements can be made by treatment of arrhythmias. Always consider specific cardiac anatomical abnormalities which may be exacerbating or causing poor heart function. If this is a concern early consultation with the cardiology team and echocardiography is important in establishing the diagnoses.

- Inotropes must be used carefully in order to avoid further myocardial ischaemia. Dobutamine has been suggested as a first line drug as it does not have the same vasoconstrictor properties as adrenaline. Other drugs which have been used are enoximone and amrinone although both these can cause major falls in SVR.

- Failing response to this, the use of a balloon pump may help when there is evidence of reversibility – specifically angioplasty should be discussed in the face of acute myocardial infarction.

- Normally in cardiogenic shock there will be a high diastolic pressure maintaining mean arterial pressure however in prolonged shock a vasodilatory state may ensue necessitating support with vasoconstrictors.

2.131 Inotrope use in cardiogenic shock :

- For patients with a high SVR use dobutamine.
- If no response to dobutamine, adrenaline is more efficacious.
- If on using adrenaline SVR increases glyceryl trinitrate can be used.
- An alternative is enoximone - if used venous system may need support with fluid and the arterial system with noradrenaline.
- Balloon pump may need to be considered in certain cases. (Discuss with consultant)

For information re management of Myocardial Infarction, Acute Coroanry Syndrome, Cardiac Tamponade, Arrythmias etc see UHNS medical guidelines.

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Section 2.14 Guidelines for Permissive Hypotension in Trauma

- In trauma patients who are suspected of having uncontrolled haemorrhage, permissive hypotension is the appropriate temporary management strategy. This involves accepting a low blood pressure and restricting fluids until haemostasis can be achieved. The benefits of this approach are: less bleeding than would result from a higher blood pressure; less dilution of clotting factors; less immuno-suppression from transfused blood. However, this deliberate under-resuscitation will tend to lead to poor tissue perfusion and acidosis. It is essential to act quickly to identify and treat (or exclude) the causes of uncontrolled haemorrhage and to restore adequate organ perfusion as soon as any sources of bleeding have been controlled. This is an emergency time-limited situation and a complacent acceptance of low blood pressure must be avoided.

- If there is an associated serious head injury, achieving an adequate cerebral perfusion pressure is of paramount importance and it may be acceptable to allow a higher blood pressure in the face of uncontrolled bleeding. This is still a controversial area and a careful judgement by senior personnel is required in order to manage these two conflicting demands.

- In suspected uncontrolled bleeding following blunt trauma, a systolic blood pressure of 80-90 mm Hg is accepted temporarily and fluids are withheld if this level is achieved. In the pre-hospital phase, fluids may be withheld if the radial pulse is palpable. In penetrating trunk trauma, a systolic blood pressure of 70-80 mm Hg is accepted temporarily and fluids are withheld if this level is achieved. In the pre-hospital phase, fluids may be withheld if the carotid or femoral pulse is palpable.

- If the blood pressure is lower than the temporarily acceptable levels, small aliquots (e.g. 250 mL) of crystalloid, colloid or blood should be administered and the situation reviewed continuously, repeating the aliquots if not achieving the acceptable level. Such fluids must be warmed, preferably through a counter-current warmer or equivalent, so that they are administered at (or very close to) normal body temperature. There is some experimental evidence that fluids can be safely given at higher temperatures and this may be appropriate in the future if the benefits can be demonstrated in clinical trials. Fluids from a blood warming cabinet are warmer than those at room temperature but cool down within a few minutes. Cold blood should not be administered to the patient. There is some experimental evidence (in animals) that profound cooling to 10º in the face of haemorrhagic shock may produce a state of suspended animation that allows surgical repair of damaged structures with preservation of neurological function. This may be appropriate in the future if technological advances make it feasible and if the benefits can be demonstrated in clinical trials. In the meanwhile, hypothermia is best avoided as there is extensive evidence of harm from its associated coagulopathy and risk of infection (especially pneumonia) in clinical practice. Clotting products are less effective at low temperatures.

- It is important to monitor the patient closely during the implementation of a permissive hypotension policy. This requires the use of ECG monitoring, pulse oximetry (though the probe may not pick up an adequate signal in some

compromised patients) and invasive arterial pressure monitoring. Even in shocked patients, arterial line insertion can usually be achieved, but in cases of difficulty unnecessary delay must be avoided if a life-saving intervention is required. Non-invasive blood pressure monitoring is useful for the initial observations and as a backup if arterial cannulation cannot be achieved in a timely fashion.

- Some sources of bleeding can be controlled using simple, readily-available methods and such interventions must not be overlooked. These include direct pressure on external wounds, the application of a pelvic sling to an unstable pelvic ring fracture, reduction of open long bone fractures, temporary suture of bleeding scalp lacerations and nasal packing.

- Early diagnosis (or exclusion) of uncontrolled bleeding can usually be achieved rapidly using clinical examination, chest and pelvic plain x-rays, and ultrasound examination of the trunk. These must be immediately available in the Emergency Department.

- If they are not part of the Trauma Team itself, early referral to appropriate Surgical Specialists is essential in this emergency situation. This should be pre-emptive: the referral should be made as soon as a source of bleeding is suspected in that surgeon's area of expertise – before definitive diagnosis is achieved. Early referral to the Anaesthetic/Intensive Care Service is equally important, though they should always be represented on the Trauma Team. Interventional Radiologists should be involved as soon as a source of bleeding that is amenable to embolisation or other radiological intervention is identified.

- Early provision of cross-matched blood (or Group-O Rhesus-negative blood if needed immediately) and clotting products is essential, although the initial plan will be to administer these after control of the sources of bleeding (or exclusion of uncontrolled bleeding).

- A CT scan may be desirable to clarify or confirm the diagnosis. Taking a shocked patient to the CT Scanner has high risks and the benefits of the investigation must outweigh the risks. A senior clinician acting as the Trauma Team Leader should make this decision, in liaison with senior specialist team members when appropriate. A full team should accompany the closely-monitored patient to the scanner. The organisation of transfer must be slick to avoid unnecessary delays. Full resuscitation equipment must accompany the transfer and a backup plan must be pre-agreed e.g. to divert immediately to a waiting Operating Theatre for immediate surgical intervention if the patient should deteriorate.

- Interventional radiology may be required to stop uncontrolled bleeding, especially from the pelvis or retroperitoneum. As for the CT Scanner, taking a shocked patient to the Angiography Suite has high risks and the benefits of the investigation must outweigh the risks. A senior clinician acting as the Trauma Team Leader should make this decision, in liaison with senior specialist team members. Similar plans for monitoring and resuscitation must be in place – the Angiography Suite should serve as an intensive care environment. Even though angiographic embolisation is an intervention rather than just an investigation, there must also be a pre-agreed backup plan e.g. to divert immediately to a waiting

Operating Theatre for immediate surgical intervention if the patient should deteriorate.

- As soon as it is clear that surgical intervention is required, the patient should be transferred rapidly to the Operating Theatre to control the bleeding. A system should be in place to achieve this within 30 minutes of admission to the Emergency Department in obvious cases, assessable by clinical examination, plain chest and pelvic x-rays and trunk ultrasound. Agreed communication plans to involve all the relevant specialists, to procure blood and blood products (to be sent directly to the Operating Theatre) and to alert and equip the Theatre are of central importance.

- Prolonged hypotension is harmful. As soon as uncontrolled bleeding is excluded or as soon as it is controlled surgically or by radiological intervention, vigorous administration of warmed fluid, blood or blood products will be an urgent priority to restore tissue perfusion and limit the damage from the deliberate under-resuscitation. Most cases of suspected uncontrolled bleeding will not reveal an uncontrolled source – it is important to achieve this diagnosis of exclusion quickly to minimise harm. On the other hand, recognising the occasional case of true uncontrolled bleeding and managing it in a careful but urgent manner, using permissive hypotension coupled with prompt surgical and/or radiological intervention, will save lives.

Section 2.2 Cardiovascular monitoring

- Cardiovascular monitoring is one of the aspects of care unfamiliar to many new trainees. Invasive monitoring of the circulation is required on ICU for several key reasons:
    - Use of inotropes and vasopressors
    - Shock states
    - Repeated sampling of blood gases
    - Difficulty estimating volume status in critical illness

- The available monitors on ICU at UHNS are

    - Arterial pressure monitoring (arterial lines)
    - Central venous lines (CVC)
    - Oesophageal Doppler monitors (ODM)
    - PICCO
    - Pulmonary artery catheters (PAC; Swan-Ganz catheters)

- The methods of inserting these are dealt with in the **appendices**.

2,21 Arterial lines

- Arterial lines are indicated on ICU for the following reasons:

    - Use of vasopressors and inotropes
    - Shock
    - Need for repeated blood gas sampling
    - As aspect of PICCO monitoring system

- In practice this means that virtually all ICU patients will have an arterial line for the majority of their stay although the need for the line must be reviewed regularly

- Relative contraindications may include
    - Severe coagulopathy
    - Local site infection
    - Severe peripheral vascular disease

- The following sites are available for use:
    - Radial artery
    - Brachial artery (also used for PICCO)
    - Femoral artery (also used for PICCO)
    - Dorsalis pedis artery

- There are three types of arterial line available: Flowswitch (used routinely), Vygon leadercaths and PICCO lines. The latter two are suitable for brachial and femoral. Flowswitch can be used for all except femoral.

- Potential complications are rarely observed but include

    - Distal ischemia
    - Soft tissue infection
    - False aneurysm formation
    - Inadvertent drug administration

2.22 Central venous catheters

- Similarly to Arterial lines, CVCs are commonly used and inserted on ICU.
  The indications may include:
    - Delivery of vasoactive drugs
    - Delivery of concentrated or caustic drugs
    - IV access for multiple infusions
    - Need for TPN
    - Determination of CVP
    - Sampling of ScvO2
    - As part of PICCO system

- The insertion and care of CVC is dealt with in the appendices

- Contra-indications are relative but may include:

    - Severe coagulopathy
    - Local site infection
    - Anatomical problems

- Complications are myriad but include

    - Related to insertion
        - Pneumothorax
        - Haemothorax
        - Cyclothorax
        - Arrhythmias
        - Arterial puncture
        - Haematoma
        - Nerve injury (femoral, vagal, phrenic)
        - Air embolism

    - Related to maintenance
        - Infection (catheter related blood stream infection)
        - Thrombo-embolism

- The risks are minimised by training and experience, use of ultrasonography, strict asepsis, good line care post-insertion and daily review of the necessity for CVC presence and usage.

- Potential routes for insertion are

- Internal jugular vein
- Subclavian vein
- Femoral vein

- Central venous pressure is of some use in determination of volume status and may be of some use as a resuscitation goal (see section 2.124). Its absolute value may be of use in extremes (e.g. if extremely high in the setting of suspected tamponade) but typically more can be gained from evaluating its response to repeated therapeutic challenges, such as fluid boluses.

2.23 Oesophageal Doppler monitors

- The Oesophageal Doppler Monitor (EDM) is a non-invasive cardiac function and volume status monitor that utilises descending aortic flow to provide a real-time indication of cardiovascular status.

- The EDM consists of two major components: a single-patient oesophageal probe and a colour waveform monitor that provides an on-screen display of blood flow velocity profiles. The single-patient, 6mm probe contains two, continuous-wave Doppler transducers. Following the entering of patient height, weight and age it is inserted into the oesophagus and focused on the patient's descending aorta and the signal optimised.

- The colour waveform spectrum and the green line that conforms to the contour of the EDM's waveform ensure an immediate, continuous indication of signal quality.

- The monitor displays a continuous display of cardiac output and stroke volume --as well as a visual and quantitative representation of aortic blood flow, preload, contractility and afterload-- allowing early diagnosis of changes in patients' cardiac condition and providing immediate feedback on the effects of therapeutic interventions.

2.231 Interpreting EDM Waveforms

- See schematic diagram

- The base of the EDM waveform is used as an index to preload and is displayed as FTc (systolic Flow Time corrected for heart rate).
- The waveform height is an index to contractility and appears quantitatively as PV (Peak Velocity).
- Changes in afterload are indicated by shifts in both FTc and PV values.

Normal values

- FTc (systolic Flow Time corrected for heart rate). Normal 330-360 msec

- Peak velocity   Age 20:   90-120 cm/sec
          Age 50:   70-100 cm/sec
          Age 70:   50-80 cm/sec

Clinical Interpretation

- Hypovolemia is indicated visually by a narrow waveform base and quantitatively by a decrease in corrected time flow (fig. 1a). Restoration of normovolemia results in a widening of the waveform base and a lengthening of the flow time (fig. 1b).

- A poorly contractile left ventricle displays reduced waveform height with abnormally low peak velocity (fig. 2a). Effective inotropic therapy increases waveform height and restores peak velocity (fig. 2b).

- Excessive afterload is indicated by both a reduced waveform height and narrowed waveform base (fig. 3a). Appropriate vasodilation is evidenced by increases in both the peak velocity and flow time (fig. 3b).

FTc, systolic flow time corrected for heart rate; SV, stroke volume.
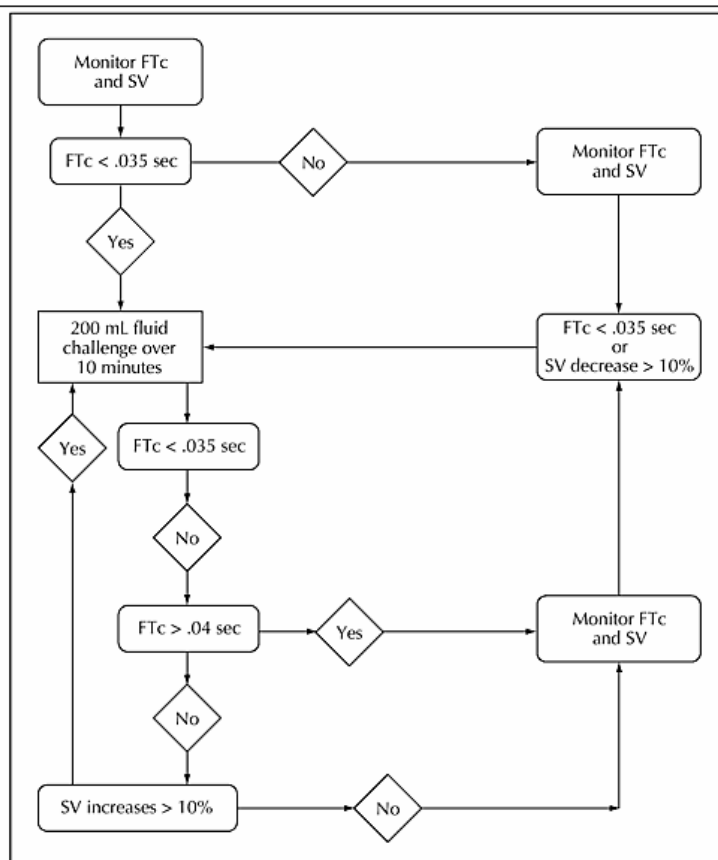
<u>Tips For More Effective EDM Use</u>

1. When possible, insert the EDM probe prior to positioning other oesophageal tubes/probes to reduce the possibility of EDM signal disruption.
2. Insert lubricated probe with the bevelled edge facing up toward the back of patient's throat.
3. Include EDM's audible signal when focusing the probe.
4. If waveform intensity varies with respiration, probe is probably placed too low. Adjust for the most consistent signal.
5. The default FILTER setting of 300 Hz is appropriate for most patients. At times it may be necessary to select the 900 Hz filter to eliminate low frequency interference. Engage the 900 Hz filter when: "below the line" flow appears during systole; high intensity diastolic flow is present, indicated by excessive white in waveform; diastolic flow is greater than .2 m/sec.
6. If a shorter time period is desired for calculations, reduce the NUMBER OF CYCLES FOR CALCULATIONS parameter on the Screen set up display three cycles per calculation is recommended during excessive body activity.
7. The VELOCITY RANGE default setting of 1.0 m/sec is appropriate for most patients. The 2.0 m/sec setting should be used to obtain calculations for patients with very high peak velocities. If the waveform appears larger than the screen will allow, alter the range setting to display the entire waveform. The 0.5 m/sec range may allow for easier detection of change in patients with very low peak velocities.

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When using the Doppler please record the following on the ICU chart after use:

Peak Velocity
Corrected flow time
Stroke Volume
Cardiac Index

Diagram 1 : interpretation of the waveform (styilised)

Oesophageal doppler wave form interpretations

Oesophageal doppler wave form interpretations

Fig 1A

Hypovolaemia

Fig 1B
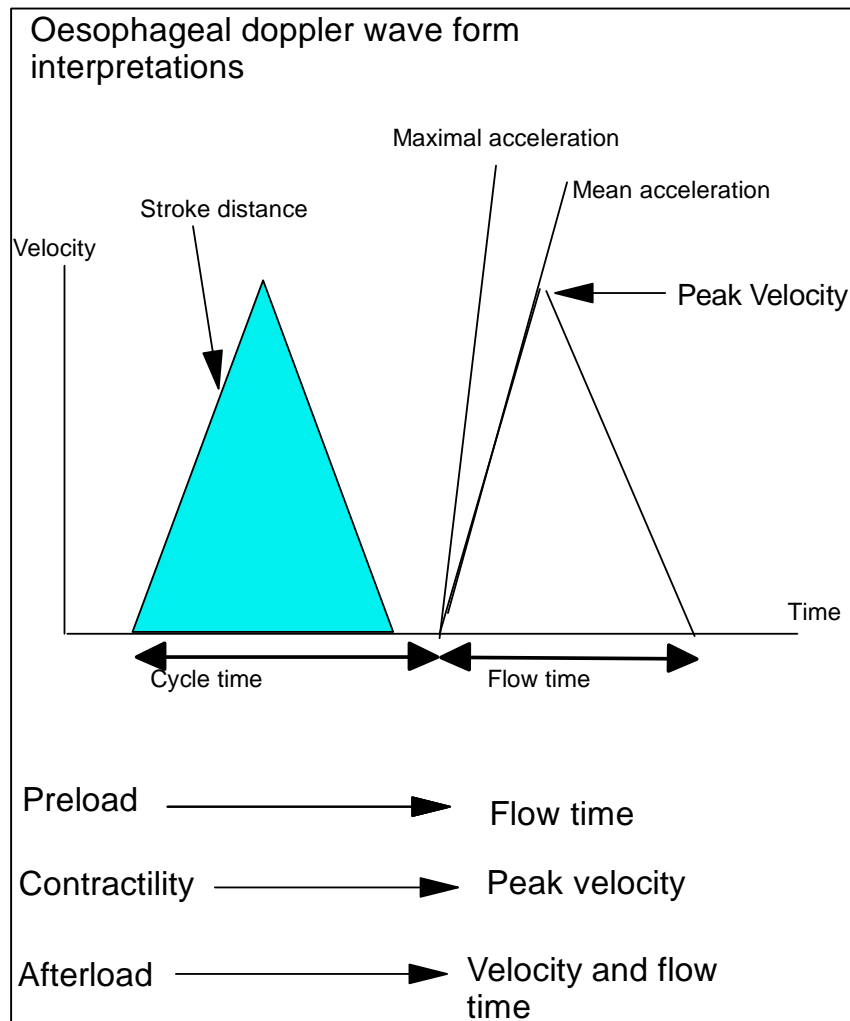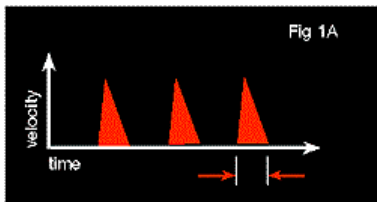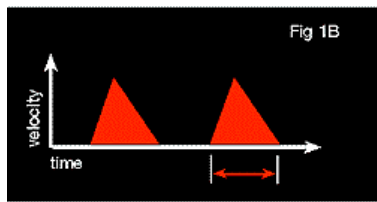
Fluid repletion

Oesophageal doppler wave form interpretations

Fig 3A
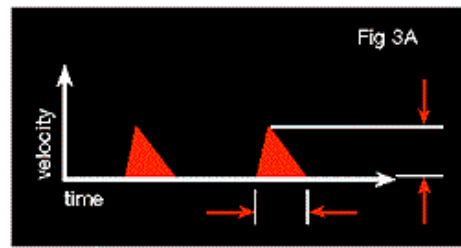
High systemic vascular resistance

Fig 3B
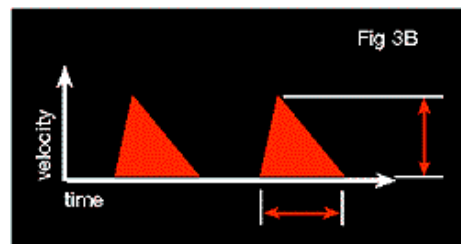
Afterload reduction

Oesophageal doppler wave form interpretations


Fig 2A

Left ventricular failure

Fig 2B

After inotrope

2.24 PICCO

- PiCCO stands for Pulse Contour Cardiac Output and is a device useful for monitoring cardiac output continuously as well as providing other useful information on volume status, likely fluid responsiveness and distribution of fluid within the body.

- PICCO system comprise of
    - Arterial cannula   inserted femorally or brachially
    - injectate temp sensor   placed on the CVC flush line
    - PICCO plus monitor   a portable unit which can integrate with bedside monitors
- The PICCO system analyses the arterial waveform to provide an estimate of cariac output. Although initially providing information based on a population algorithim it requires individual calibration in each patient. Usefully this calibration process (injection of cold saline which is sensed by the arterial thermistor : a form of thermodilution) also provides data on lung water and " pre-load."

- Indications for insertion are:
    - All kinds of shock or imminent shock

    - Acute Respiratory Distress Syndrome (ARDS)

    - Acute severe cardiac insufficiency

    - Major Surgery

    - Acute multi-trauma/severe burns

    - Transplantations

- In practice we tend to use PICCO in preference to EDM when information about lung water may be useful, such as in ARDS/ALI or severe sepsis where lung injury is at risk of developing.
- Contra-indications and cautions include:
    - Patients in whom there are arterial access restrictions, for example due to femoral artery grafting or severe burns in areas where the arterial catheter would normally have been placed.
    - The PiCCO-Technology may give incorrect thermodilution measurements in patients with
        - intracardiac shunts,
        - aortic aneurysm,
        - aortic stenosis, mitral or tricuspid insufficiency,
        - pneumonectomy,
        - macro lung embolism
        - extracorporeal circulation (if blood is either extracted from or infused back into the cardiopulmonary circulation).
        - On intra-aortic balloon pumps
        - Rapidly changing body temperature
        - Marked arrhythmias

## 2.41 Insertion of PICCO and calibration

- The arterial cannula is sited aseptically using the Seldinger technique via the brachial (16cm) or femoral (20cm) route.
- A PICCO flush system is connected to the arterial cannula
- from the monitor there are three cables:
    - arterial thermistor   connects to the red plastic connection on the arterial line
    - pressure transducer   the white connector on the flush line connects to this cable
    - injectate thermistor   this cable runs to the special thermistor supplied with the flush sets, which is connected to the flush line of the CVC
- Once connected patient data is entered on the input screen (catheter length, height and weight, CVP, volume of injectate and temperature).
- The arterial line is zeroed at the transducer to air (screen AP zeroing)
- The screen is then displayed with an arterial waveform: in some cases a cardiac output/index will be displayed (*pulse contour display page*)
- The PiCCO is calibrated with thermodilution (*thermodilution display page*)
    - 15ml of cold saline (from fridge) connect to injectate port
    - select thermodilution display page
    - press *start*
    - wait until the screen says *stable*
    - inject steadily and swiftly (in under 7 seconds)
    - screen registers the injection and a curve will be displayed
    - machine then calculates the indices and calibrates the cardiac index
    - repeat 3 times for initial calibration
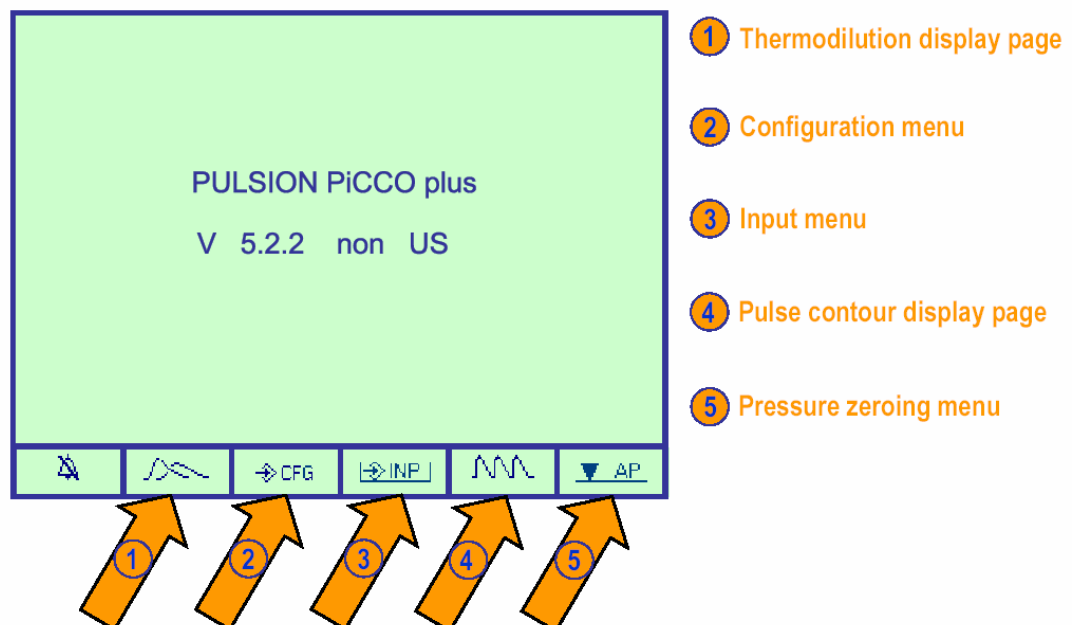
fig 1 main PiCCO screen
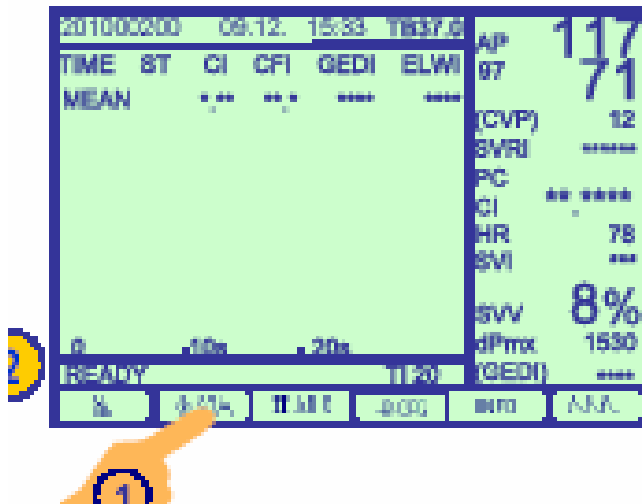
Fig 2 : thermodilution screen
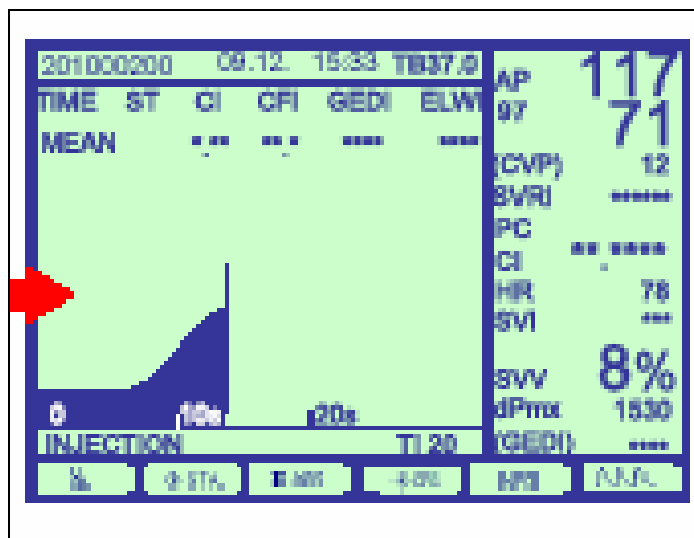
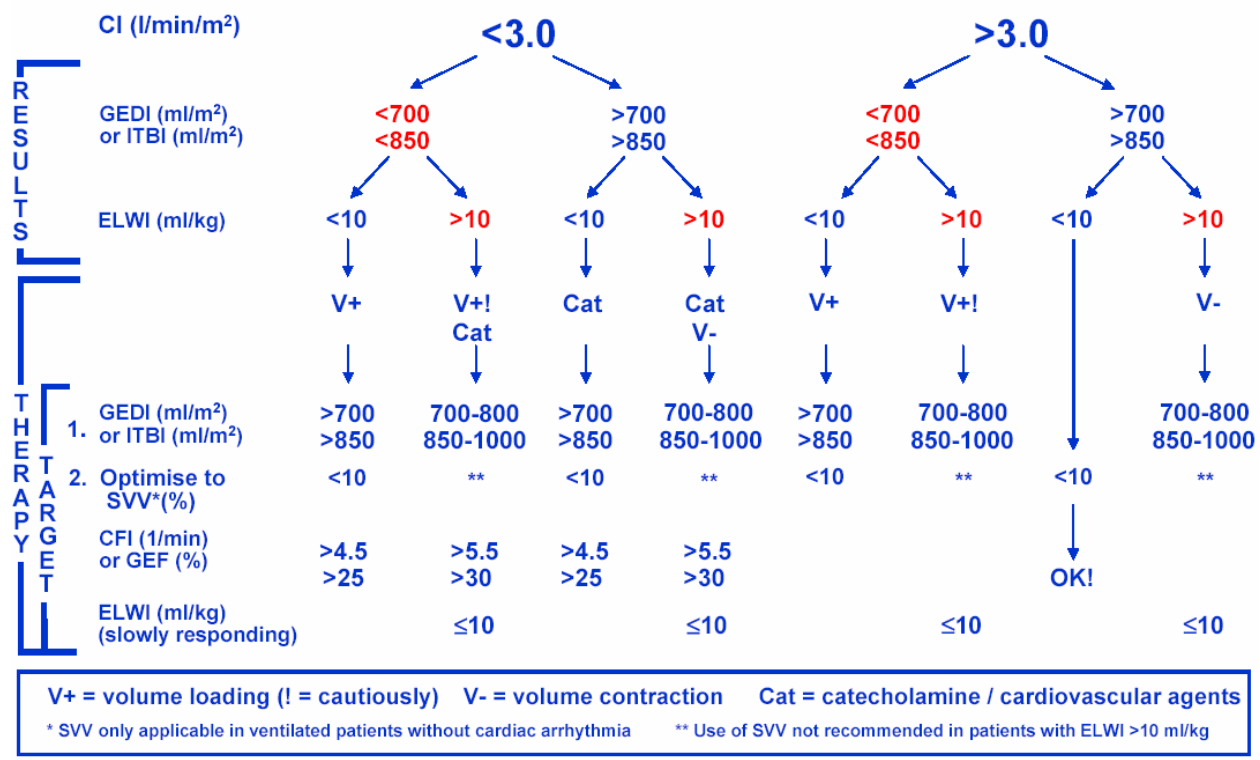
Fig 3 : injection of cold saline
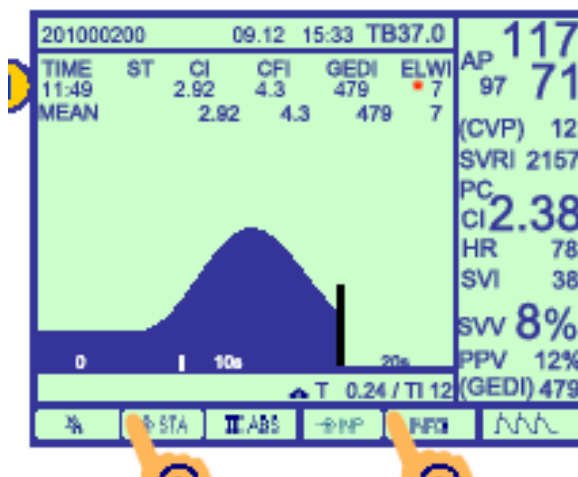
Figure 4: thermodilution indices

2.42   Measured values on the PiCCO

- the following data is obtained by the PiCCO

    - **cardiac index**   *this is obtained from TD measurement and continuously*
    - **stroke volume index**
    - **intra-thoracic blood volume index**   *indicator of pre-load*
    - **stroke volume variation**   *an indicator of potential volume responsiveness (only in ventilated pts with no arrhythmias)*
    - **dPMx**   *an indicator of contractility*
    - **cardiac function index**   *an indicator of contractility*
    - **extravascular lung water index**   *an indicator of the excess water present in the lungs (not including effusions, just parenchymal)*
    - **SVRI**   *the systemic vascular resistance index, a derivative of mean arterial pressure and cardiac index.*

- Normal values for the indices are:

    - **CI 3.0 – 5.0 l/min/**
    - **SVI 40 – 60 ml/m2**
    - **GEDI 680 – 800 ml/m2**
    - **ITBI 850 – 1000 ml/m2**
    - **ELWI 3.0 – 7.0 ml/kg**
    - **PVPI 1.0 – 3.0 -**
    - **SVV ¡Ü 10 %**
    - **PPV ¡Ü10 %**
    - **GEF 25 – 35 %**
    - **CFI 4.5 – 6.5 1/min**
    - **MAP 70 – 90 mmHg**
    - **SVRI 1700 – 2400 dyn*s*cm-5*m2**

- The exact response to the values must be considered along with other patient observations (e.g. appearance, perfusion, dug usage, urine output, lactate etc). Pulsion have produced a decision tree based on CI, ITBVI, EVLWI and CFI/SVV. This is available on the unit and may provide some guide. Generally correct low ITBVI first with fluid and this may improve CI if low. Once optimised ITBVI assess contractility and cardiac index to judge if inotropes are indicated. If MAP is below target and has not been improved by volume resuscitation then consider pressors if index normal or pressors and inotropes concurrently if both low. **Until you are experienced it is better to seek senior help.**

- For reference the decision tree is reproduced below:

The PiCCO decision tree:

RESULTS

CI (l/min/m²): **<3.0** | **>3.0**

GEDI (ml/m²) or ITBI (ml/m²):
- **<700 <850** | **>700 >850** (under <3.0)
- **<700 <850** | **>700 >850** (under >3.0)

ELWI (ml/kg): **<10** | **>10** | **<10** | **>10** | **<10** | **>10** | **<10** | **>10**

Actions:
- V+ | V+! Cat | Cat | Cat V- | V+ | V+! | (→) | V-

THERAPY / TARGET

	<10 (CI<3.0, GEDI<700/<850)	>10	<10	>10	<10 (CI>3.0, GEDI<700/<850)	>10	<10	>10
1. GEDI (ml/m²) or ITBI (ml/m²)	>700 >850	700-800 850-1000	>700 >850	700-800 850-1000	>700 >850	700-800 850-1000		700-800 850-1000
2. Optimise to SVV*(%)	<10	**	<10	**	<10	**	<10	**
CFI (1/min) or GEF (%)	>4.5 >25	>5.5 >30	>4.5 >25	>5.5 >30			OK!	
ELWI (ml/kg) (slowly responding)		≤10		≤10		≤10		≤10

V+ = volume loading (! = cautiously)   V- = volume contraction   Cat = catecholamine / cardiovascular agents
* SVV only applicable in ventilated patients without cardiac arrhythmia   ** Use of SVV not recommended in patients with ELWI >10 ml/kg

- Further information on PiCCO is available on the unit and also on the website:
  http://www.pulsion.com/index.php?id=46

- **2.25 Pulmonary artery catheters**

  - Although formerly a popular form of monitoring and cardiac output determination on critical care in the UK the use of PAC has declined markedly over the past decade. This is due to both emergence of alternative forms of monitoring and also a series of trials evaluating the usefulness of PAC as a monitor and as a guide to therapeutics in critical care. The lack of evidence that they influence outcome on ICU coupled with the definite morbidity associated with their use has largely lead to their abandonment in ICU at UHNS.

  - There are several instances where they are still used:
    - Presence of Intra-aortic balloon pump
    - Cardio-thoracic critical care
    - Contra-indication to PICCO or ODM
    - Therapeutic interest in pulmonary artery pressure

  - Insertion is detailed in the appendices

  - The usual data obtained is PAOP (pulmonary artery occlusion pressure or wedge pressure), stroke volume, CO / CI and derived values such as oxygen delivery indices, left ventricle stroke work and systemic vascular resistance. SvO2 may also be sampled.

  - PAOP may provide some limited information on volume status in a manner akin to CVP and its response to therapy may be of use. It may have some usage in fluid management in cardiogenic shock.

  - CO/CI determination is via thermodilution techniques and again with stroke volume may provide some guide to therapeutics (e.g. titration of IABP, titration of inotropes).

  - SvO2 is used in some protocols as an indication of the balance between global O2 delivery and O2 demand. It is now more common to use central venous O2 saturations for this purpose in the belief the two are related.

  - The complications of PACs are as for CVCs but also additional risk of

    - Pulmonary artery rupture
    - Endocarditis
    - Ventricular arrythmias
    - PE

  - For those interested in learning about PACs a useful resource is
    - www.pacep.org

2.26 Vigileo Monitor and Flotrac sensor

This system provides a minimally invasive way of assessing cardiac output and volume status.
The Edwards FloTrac system algorithm is based on the
principle that aortic pulse pressure is proportional to stroke
volume (SV) and inversely related to aortic compliance.

Using of complex mathematical formulae based on many observations a stroke volume is determined. For full details see
http://www.edwards.com/eu/products/mininvasive/vigileo.htm and study the quick guide to cardiopulmonary care which is full of interesting detail on cardiopulmonary monitoring as well as the underlying principals of the flotrac sensor.

Once stroke volume is obtained then cardiac output and cardiac index can be derived. The vigileo also will give information on stroke volume variation which can be used to assess volume status.

Guidance on fluid management can be gained in two ways.
1) By using fluid boluses, filling pressures and cardiac output to plot a sterling curve until no further benefit is gained.
2) By using the stroke volume variation reading from the flotrac. An im of < 13% is used. This does not apply if the patient has arrhythmias or is spontaneously breathing.

Section 2.3 Inotropes / vasopressors

2.31 General points about vasoactive drugs

- Vasocative drugs are not without their risks, hence the severity of the patients condition must justify their usage and appropriate levels of monitoring are required. Risks include:

    o Damage to tissues
    o Very potent (depending on concentration)
    o Need for invasive monitoring
    o Potential for harm unless volume resuscitated
    o Arrhythmias
    o Increased myocardial oxygen demand

- These agents must be given via a CVC unless in an emergency situation. Invasive arterial monitoring is indicated and CO monitoring may be required if there are multiple inotropes.

2.32 Adrenaline

- Acts on $\alpha 1, \beta 1, \beta 2$ receptors. At very low doses the $\beta 2$ effect predominates (dilator action on sphlancic circulation, liver bed, bronchial muscle) although this range is rarely used on ICU

- At more usual doses the $\beta 1$ then $\alpha 1$ effects begin producing inotropism, chronotropism and then vasoconstriction

- PROs
    o Good all rounder and initial choice until nature of shock becomes apparent
    o Familiarity and availability (e.g. on general ward)

- CONs
    o Mixed receptor agonism
    o Hyperglycaemia
    o Lactic acidosis (due to both aerobic and anaerobic glycolysis)

- Main indications and uses

    o resuscitation drug

Cardiac arrest	1mg (1ml of 1:1000 or 10ml of 1:10 000)
Anaphylaxis	0.5mg IM or 50-100 mg IV

- o Initial ino-pressor
  5-25 ml/h of 4mg in 50ml or 8 mg in 100 ml (0.01-0.47 mg/kg/min)

- o Second line agent in severe shock states
  0.5-25 ml/h of 4mg in 50ml or 8 mg in 100 ml

2.32 Noradrenaline

- Endogenous precursor to adrenaline. Physiologically it is found as a neurotransmitter at synapses, rather than as a circulating hormone

- Acts at α1 predominantly with some β1 action. Hence acts as potent vasoconstrictor with some lesser inotropic action.

- PROs   predictable vasoconstrictor effect

- CONs   occ reflex bradycardia
              decreased sphlancic perfusion

  - Main indications are

    - o Vasodilatory shock   e.g. sepsis, liver failure,
                                  SIRS (e.g. pancreatitis, burns)

    - o Need for high perfusion pressures
                                  e.g. targeting cerebral perfusion

  - Usual starting dose is

    8 mg in 100ml @ 0.5-25 ml/h (0.01-0.47 mcg/kg/min in 70kg adult) "single strength." Increased as required to target MAP.

2.33 Dobutamine

- Synthetic catecholamine acting on β1and β2 receptors
- Action is thus inotrope with potential for some vasodilation (ino-dilator)
- Effect on BP will depend on filling status and current cardiac output. In some cases the increase in CO will offset the vasodilation and BP will hold or improve, in other cases there may be a fall in BP.

- PROS   good inotrope

- CONs   tachycardia, inc myocardial O2 demand, occ hypotension

- Indications
  - o Inotropism   septic shock (cold), cardiogenic shock

- o Inc DO2　　　　pre-optimisation strategies
- Doses　　250mg in 50ml @ 1-20 ml/h

2.34 Vasopressin

- Vasopressin (ADH) is utilised as a potent second line vasoconstrictor in refractory vasodilatory shock
- Acts via V_1 receptors (which are PLC linked similar to $\alpha 1$ receptors)
- Degree of vasoconstriction varies according to vascular bed. Skin & gut are particularly affected.
- Some evidence that sepsis is a vasopressin deficient state and that small doses of vasopressin act as HRT

- Main concerns are skin complications, sphlancic ischaemia, coronary vasoconstriction. **Discuss the use of vasopressin with a senior prior to commencement.**

- Initial doses
  - o 20 units/40ml i.e. 0.5 U/ml
  - o Commence at low dose of 1.2 ml/hr and increase as required up to 4.8 ml/h.

2.35 Enoximone

- Phosphodiesterase (PDE-III) inhibitor which act to increase myocardial cAMP and hence act as inotropes
- Also act to increase cAMP in vascular smooth muscle which acts to vasodilate (peripheral and pulmonary)
- Act as ino-dilators

- PRO　　　-do not depend on β receptors
　　　　　-assist coronary blood flow
　　　　　-aid diastolic relaxation (lusitropy)
　　　　　-additive effect with β agonists

- CON　　　- dilator action may produce hypotension
　　　　　- arrythmogenic

- Dose　　　90 mcg/kg/min over 10 – 30 min followed by 5 – 20 mcg/kg/min
　　　　　(not more than 24 mg/ kg per day)

2.36 Which inotrope to choose ?

- Septic shock / SIRS   NORAD +/- DOBUT
    Second line   vasopressin

- Cardiogenic shock   DOBUT +/- NOR/ADR
    Second line   milrinone/enoximone

- Anaphylactic shock   ADRENALINE

- Vasodilation (e.g. sedation, liver failure)
    NORADRENALINE

Section 3   Sepsis and Host defence issues

Section 3.1   Severe Sepsis and Septic Shock

- Severe sepsis is sepsis associated with organ dysfunction, hypoperfusion or hypotension. There may be one or more of:

arterial hypoxaemia	oliguria
increased serum creatinine	coagulation abnormalities
thrombocytopenia	hyperbilirubinaemia
hyperlactataemia	metabolic acidosis
arterial hypotension	

- Septic shock is severe sepsis with hypotension refractory to fluid loading. The usual haemodynamic picture in septic shock is that of an increased cardiac output (after correction of hypovolaemia) and vasodilatation resulting in hypotension. Mixed venous oxygen saturations will usually be high.

- The septic patient will be hypovolaemic both from vasodilatation (both arteriolar and venous capacitance vessels) and increase in capillary leak.

3.11 Sepsis (initial) resuscitation at a glance

- Initial resuscitation and treatment (first 6 hours) is as follows:

Secure the airway and administer 02 to ensure adequate oxygenation, maintain SaO2 > 90%.

Correct hypovolaemia. Fluid resuscitate with isotonic crystalloid. Insert a central venous catheter and fluid load. Aim for haemodynamic goals of:

  CVP ≥ 8 mmHg spontaneously breathing, ≥12 mmHg ventilated

  MAP > 65 mmHg

  Urine output > 0.5 ml/kg

  Central venous saturation > 70%

If central venous saturation is < 70% and haematocrit < 30% transfuse packed red cells.

If central venous saturation is < 70% and haematocrit > 30% commence dobutamine infusion at 5 μg/kg/min up to maximum of 20 μg/kg/min.

Life threatening hypotension requires treatment with a vasopressor to allow further fluid resuscitation and implementation of more advanced haemodynamic monitoring.

Obtain relevant samples for microbiology including 2 sets of blood cultures.

Measure serum lactate (if elevated identifies patient at increased risk of mortality)

Administer appropriate antibiotics within 1 hour of presumptive diagnosis

Source control – remove infected lines, drain collections (may involve interventional radiology or surgery)

Further sepsis management at a glance

- Subsequent resuscitation and treatment (first 24 hours) is as follows:

    o Continue fluid resuscitation with isotonic crystalloid.

    o Transfuse packed cells to maintain Hb > 7 g/dl (unless acute myocardial infarction, acute coronary syndrome, or acute stroke – aim for > 8 g/dl)

    o Maintain perfusion pressure with norepinephrine to maintain appropriate MAP. Previously fit young patients may require MAP 60-65 mmHg, more elderly patients MAP 70 mmHg, hypertensive patients 80mmHg or higher. The optimum MAP is the pressure that ensures adequate end organ perfusion (see shock general principles).

    o Patients with a low cardiac output (< 2.5 l/min/m2) should receive dobutamine as previously described.

    o Insulin should be infused as per protocol to maintain blood glucose $\leq$ 10 mmol/l

    o Ventilated patients should not receive plateau airway pressures > 30 cm H2O.

    o Drotrecogin alpha (activated protein C) administration should be considered according to guideline. **Prescription should be by a consultant**. Drotrecogin alpha is contraindicated if the patient is systemically anticoagulated with heparin. On commencing drotrecogin alpha prophylactic heparin should be discontinued. If the patient requires renal replacement therapy no additional anticoagulant is required. Heparin should be avoided for the duration of the infusion.

    o In patients who appear refractory to norepinephrine consider arginine vasopressin 20 units to 40 ml normal saline at 1.2-4.8 ml/hour (0.01-0.04 units per minute) and steroids given as Hydrocortisone 50 mg qds. **Discuss with a consultant prior to use.**

    o Haemodynamic resuscitation and manipulation may be guided by use of cardiac output measurements. In patients without significant pulmonary pathology oesophageal Doppler monitoring or Vigileo should be `first line`. For patients with acute lung injury or acute respiratory distress syndrome PiCCO monitoring should be `first line`.

    o Do not use FFP to correct laboratory clotting abnormalities unless there is bleeding or a planned surgical procedure

    o Treat thrombocytopenia with platelet transfusion, with goal of count > 50 if post surgery or requiring invasive procedure, otherwise goal > 20.

Sepsis treatment: The detailed guidance

Surviving Sepsis Campaign : International guidelines for management of severe sepsis and septic shock: 2008

(Crit Care Med 2008 Vol. 36, No.1)

Initial resuscitation and infection issues

Table 3. Initial resuscitation and infection issues

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in parentheses after each guideline
- ● Indicates a strong recommendation, or "we recommend"
- ○ Indicates a weak recommendation, or "we suggest"

Initial resuscitation (first 6 hrs)
- ● Begin resuscitation immediately in patients with hypotension or elevated serum lactate >4 mmol/L; do not delay pending ICU admission (1C)
- ● Resuscitation goals (1C)
  CVP 8–12 mm Hg[a]
  Mean arterial pressure ≥ 65 mm Hg
  Urine output ≥0.5 mL·kg^{-1}·hr^{-1}
  Central venous (superior vena cava) oxygen saturation ≥70% or mixed venous ≥65%
- ○ If venous oxygen saturation target is not achieved (2C)
  Consider further fluid
  Transfuse packed red blood cells if required to hematocrit of ≥30% and/or
  Start dobutamine infusion, maximum 20 μg·kg^{-1}·min^{-1}

Diagnosis
- ● Obtain appropriate cultures before starting antibiotics provided this does not significantly delay antimicrobial administration (1C)
  Obtain two or more BCs
  One or more BCs should be percutaneous
  One BC from each vascular access device in place >48 hrs
  Culture other sites as clinically indicated
- ● Perform imaging studies promptly to confirm and sample any source of infection, if safe to do so (1C)

Antibiotic therapy
- ● Begin intravenous antibiotics as early as possible and always within the first hour of recognizing severe sepsis (1D) and septic shock (1B)
- ● Broad-spectrum: one or more agents active against likely bacterial/fungal pathogens and with good penetration into presumed source (1B)
- ● Reassess antimicrobial regimen daily to optimize efficacy, prevent resistance, avoid toxicity, and minimize costs (1C)
- ○ Consider combination therapy in *Pseudomonas* infections (2D)
- ○ Consider combination empiric therapy in neutropenic patients (2D)
- ○ Combination therapy ≤3–5 days and de-escalation following susceptibilities (2D)
- ● Duration of therapy typically limited to 7–10 days; longer if response is slow or there are undrainable foci of infection or immunologic deficiencies (1D)
- ● Stop antimicrobial therapy if cause is found to be noninfectious (1D)

Source identification and control
- ● A specific anatomic site of infection should be established as rapidly as possible (1C) and within first 6 hrs of presentation (1D)
- ● Formally evaluate patient for a focus of infection amenable to source control measures (e.g. abscess drainage, tissue debridement) (1C)
- ● Implement source control measures as soon as possible following successful initial resuscitation (1C) (exception: infected pancreatic necrosis, where surgical intervention is best delayed) (2B)
- ● Choose source control measure with maximum efficacy and minimal physiologic upset (1D)
- ● Remove intravascular access devices if potentially infected (1C)

GRADE, Grades of Recommendation, Assessment, Development and Evaluation; ICU, intensive care unit; CVP, central venous pressure; BC, blood culture.

[a]A higher target CVP of 12–15 mm Hg is recommended in the presence of mechanical ventilation or preexisting decreased ventricular compliance.

Hemodynamic support and adjunctive therapy

Table 4. Hemodynamic support and adjunctive therapy

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in parentheses after each guideline.
- ● Indicates a strong recommendation, or "we recommend"
- ○ Indicates a weak recommendation, or "we suggest"

Fluid therapy
- ● Fluid-resuscitate using crystalloids or colloids (1B)
- ● Target a CVP of ≥8 mm Hg (≥12 mm Hg if mechanically ventilated) (1C)
- ● Use a fluid challenge technique while associated with a hemodynamic improvement (1D)
- ● Give fluid challenges of 1000 mL of crystalloids or 300–500 mL of colloids over 30 mins. More rapid and larger volumes may be required in sepsis-induced tissue hypoperfusion (1D)
- ● Rate of fluid administration should be reduced if cardiac filling pressures increase without concurrent hemodynamic improvement (1D)

Vasopressors
- ● Maintain MAP ≥65 mm Hg (1C)
- ● Norepinephrine and dopamine centrally administered are the initial vasopressors of choice (1C)
- ○ Epinephrine, phenylephrine, or vasopressin should not be administered as the initial vasopressor in septic shock (2C). Vasopressin 0.03 units/min may be subsequently added to norepinephrine with anticipation of an effect equivalent to norepinephrine alone
- ○ Use epinephrine as the first alternative agent in septic shock when blood pressure is poorly responsive to norepinephrine or dopamine (2B).
- ● Do not use low-dose dopamine for renal protection (1A)
- ● In patients requiring vasopressors, insert an arterial catheter as soon as practical (1D)

Inotropic therapy
- ● Use dobutamine in patients with myocardial dysfunction as supported by elevated cardiac filling pressures and low cardiac output (1C)
- ● Do not increase cardiac index to predetermined supranormal levels (1B)

Steroids
- ○ Consider intravenous hydrocortisone for adult septic shock when hypotension responds poorly to adequate fluid resuscitation and vasopressors (2C)
- ○ ACTH stimulation test is not recommended to identify the subset of adults with septic shock who should receive hydrocortisone (2B)
- ○ Hydrocortisone is preferred to dexamethasone (2B)
- ○ Fludrocortisone (50 μg orally once a day) may be included if an alternative to hydrocortisone is being used that lacks significant mineralocorticoid activity. Fludrocortisone if optional if hydrocortisone is used (2C)
- ○ Steroid therapy may be weaned once vasopressors are no longer required (2D)
- ● Hydrocortisone dose should be ≤300 mg/day (1A)
- ● Do not use corticosteroids to treat sepsis in the absence of shock unless the patient's endocrine or corticosteroid history warrants it (1D)

Recombinant human activated protein C
- ○ Consider rhAPC in adult patients with sepsis-induced organ dysfunction with clinical assessment of high risk of death (typically APACHE II ≥25 or multiple organ failure) if there are no contraindications (2B, 2C for postoperative patients).
- ● Adult patients with severe sepsis and low risk of death (typically, APACHE II <20 or one organ failure) should not receive rhAPC (1A)

GRADE, Grades of Recommendation, Assessment, Development and Evaluation; CVP, central venous pressure; MAP, mean arterial pressure; ACTH, adrenocorticotropic hormone; rhAPC, recombinant human activated protein C; APACHE, Acute Physiology and Chronic Health Evaluation.

Other supportive therapy of severe sepsis

Table 5. Other supportive therapy of severe sepsis

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in parentheses after each guideline
- ● Indicates a strong recommendation, or "we recommend"
- ○ Indicates a weak recommendation, or "we suggest"

Blood product administration
- ● Give red blood cells when hemoglobin decreases to <7.0 g/dL (<70 g/L) to target a hemoglobin of 7.0–9.0 g/dL in adults (1B). A higher hemoglobin level may be required in special circumstances (e.g., myocardial ischaemia, severe hypoxemia, acute hemorrhage, cyanotic heart disease, or lactic acidosis)
- ○ Do not use erythropoietin to treat sepsis-related anemia. Erythropoietin may be used for other accepted reasons (1B)
- ○ Do not use fresh frozen plasma to correct laboratory clotting abnormalities unless there is bleeding or planned invasive procedures (2D)
- ● Do not use antithrombin therapy (1B)
- ○ Administer platelets when (2D)
  Counts are <5000/mm^3 (5 × 10^9/L) regardless of bleeding
  Counts are 5000–30,000/mm^3 (5–30 × 10^9/L) and there is significant bleeding risk
  Higher platelet counts (≥50,000/mm^3 [50 × 10^9/L]) are required for surgery or invasive procedures

Mechanical ventilation of sepsis-induced ALI/ARDS
- ● Target a tidal volume of 6 mL/kg (predicted) body weight in patients with ALI/ARDS (1B)
- ● Target an initial upper limit plateau pressure ≤30 cm H$_2$O. Consider chest wall compliance when assessing plateau pressure (1C)
- ● Allow Paco$_2$ to increase above normal, if needed, to minimize plateau pressures and tidal volumes (1C)
- ● Set PEEP to avoid extensive lung collapse at end-expiration (1C)
- ○ Consider using the prone position for ARDS patients requiring potentially injurious levels of Fio$_2$ or plateau pressure, provided they are not put at risk from positional changes (2C)
- ● Maintain mechanically ventilated patients in a semirecumbent position (head of the bed raised to 45°) unless contraindicated (1B), between 30° and 45° (2C)
- ○ Noninvasive ventilation may be considered in the minority of ALI/ARDS patients with mild to moderate hypoxemic respiratory failure. The patients need to be hemodynamically stable, comfortable, easily arousable, able to protect/clear their airway, and expected to recover rapidly (2B)
- ● Use a weaning protocol and an SBT regularly to evaluate the potential for discontinuing mechanical ventilation (1A)
  - ● SBT options include a low level of pressure support with continuous positive airway pressure 5 cm H$_2$O or a T piece
  - ● Before the SBT, patients should
    be arousable
    be hemodynamically stable without vasopressors
    have no new potentially serious conditions
    have low ventilatory and end-expiratory pressure requirement
    require Fio$_2$ levels that can be safely delivered with a face mask or nasal cannula
- ● Do not use a pulmonary artery catheter for the routine monitoring of patients with ALI/ARDS (1A)
- ● Use a conservative fluid strategy for patients with established ALI who do not have evidence of tissue hypoperfusion (1C)

Sedation, analgesia, and neuromuscular blockade in sepsis
- ● Use sedation protocols with a sedation goal for critically ill mechanically ventilated patients (1B)
- ● Use either intermittent bolus sedation or continuous infusion sedation to predetermined end points (sedation scales), with daily interruption/lightening to produce awakening. Re-titrate if necessary (1B)
- ● Avoid neuromuscular blockers where possible. Monitor depth of block with train-of-four when using continuous infusions (1B)

Glucose control
- ● Use intravenous insulin to control hyperglycemia in patients with severe sepsis following stabilization in the ICU (1B)
- ● Aim to keep blood glucose <150 mg/dL (8.3 mmol/L) using a validated protocol for insulin dose adjustment (2C)
- ● Provide a glucose calorie source and monitor blood glucose values every 1–2 hrs (4 hrs when stable) in patients receiving intravenous insulin (1C)
- ● Interpret with caution low glucose levels obtained with point of care testing, as these techniques may overestimate arterial blood or plasma glucose values (1B)

Renal replacement
- ○ Intermittent hemodialysis and CVVH are considered equivalent (2B)
- ○ CVVH offers easier management in hemodynamically unstable patients (2D)

Bicarbonate therapy
- ● Do not use bicarbonate therapy for the purpose of improving hemodynamics or reducing vasopressor requirements when treating hypoperfusion-induced lactic acidemia with pH ≥7.15 (1B)

Deep vein thrombosis prophylaxis
- ● Use either low-dose UFH or LMWH, unless contraindicated (1A)
- ● Use a mechanical prophylactic device, such as compression stockings or an intermittent compression device, when heparin is contraindicated (1A)
- ○ Use a combination of pharmacologic and mechanical therapy for patients who are at very high risk for deep vein thrombosis (2C)
- ○ In patients at very high risk, LMWH should be used rather than UFH (2C)

Stress ulcer prophylaxis
- ● Provide stress ulcer prophylaxis using H2 blocker (1A) or proton pump inhibitor (1B). Benefits of prevention of upper gastrointestinal bleed must be weighed against the potential for development of ventilator-acquired pneumonia

Consideration for limitation of support
- ● Discuss advance care planning with patients and families. Describe likely outcomes and set realistic expectations (1D)

GRADE, Grades of Recommendation, Assessment, Development and Evaluation; ALI, acute lung injury; ARDS, acute respiratory distress syndrome; PEEP, positive end-expiratory pressure; SBT, spontaneous breathing trial; ICU, intensive care unit; CVVH, continuous veno-venous hemofiltration; UFH, unfractionated heparin; LMWH, low-molecular weight heparin.

Resources

1. Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008 Dellinger RP, Mitchel M. Levy, Carlet JM et al. Critical Care Medicine 2008 36 No.1, 296-327

2. Septic shock. Annane D, Bellisant E et al. Lancet 2005 365 63-78

3. Sepsis care bundles via the institute for health improvement website as www.ihi.org/IHI/Topics/CriticalCare/

4. Bernard GR, Vincent JL, Laterre PF, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. New England Journal of Medicine. 2001;344:699–709.

5. Fourrier F. Recombinant human activated protein C in the treatment of severe sepsis: An evidence-based review. Critical Care Medicine. 2004;32 (Suppl.):S534–S541.

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Section 3.2 Infection control on ICU

Section 3.21   General issues

- One of the salient concerns in modern healthcare is the emergence of pathogens resistant to commonly used antimicrobials and the involvement of these pathogens in infections which are acquired in the healthcare setting.

- Intensive care units, by virtue of the critically ill nature of the patients and the severity of their pathology have been one of the areas of greatest concern in the evolution of this phenomenon. The frequent use of potent antibiotics has created a selection pressure for resistant organisms in the ICU. The smaller contained nature of critical care units does, however, provide the intensivist an opportunity to become heavily involved in all aspects of infection control in the unit and an understanding of the aetiology of the infections will allow the intensivist to impact upon this.

- A significant proportion of critical care admissions relate to infection. The recent European SOAP study detailed a two week period on 198 European ICUs and found 24.7% of ICU admissions had sepsis on admission, 37% has sepsis during their stay and 64% received antibiotics whilst on ICU. Previously the EPIC study, a one day point prevalence study of 1417 ICUs in 17 European countries in 1992 had detailed a 44.8% incidence of infection on ICU with 20.6% having an ICU acquired infection. A similar proportion were on antibiotics at any given time (62.3%).

- Full details on the UHNS infection control policy can be found on the Intra-Net website under *clinical section/ infection control*. Within this site are trust guidelines on care of CVCs, catheters, handwashing practice and guidelines for management of an outbreak. A key document is the Trust's Infection control strategy which summarises recent policies on the topic of hospital acquired infections.

o All infection control policies can be found on the intra-net at http://uhns/policies and select infection control from the drop down menu.

Section 3.211 Infection control team

o The infection control team provide a supportive service for the critical care areas. The contacts are

- o Consultant microbiologist Dr George Orendi (bleep via switch or ring 4654)
- o Infection control nurses extension 4282 bleep 934
- o ICU infection control nurse lead/link nurse Sr Alex Worsey
- o two ward rounds are held per week
- o ICU   Wednesday 12pm   attended by ICU team, IC team and infectious disease consultant
- o MIU   Friday 12pm   attended by ICU team and IC team.

o These rounds provide excellemt opportunity to review microbiology results, ask advice on antibiotic presecribing and duration and feed back to the IC team about clinical progress. At other times in the week the IC team are very approachable and available for advice.

- A brief summary of general aspects of infection control are given below. The ones highlighted are elaborated.

Section 3.22   Prevention of antimicrobial resistance and nosocomial infection

Preventative measures may be divided into three broad categories:

- o general (environmental) preventative measures
    - § handwashing
    - § gowning / barrier methods
    - § cleaning environment
    - § architecture of unit/ unit lay-out
    - § isolation / cohorting
    - § workload on ICU

- o reduction in selection of MDR pathogens
    - § antibiotic policy
    - § antibiotic rotation
    - § infection control consultants
    - § source control
    - § eradication therapy

- o specific (patient related) preventative measures
    - § aseptic techniques
    - § use of prophylactic antibiotics
    - § reduction in ventilator associated pneumonia
    - § reduction in catheter related blood stream infection
    - § use of care bundles
    - § selective decontamination of the digestive tract (SDD)

3.221 Handwashing   see IC policy 03

The importance of handwashing in infection control has been recognised since the 19th century. It is still considered a key aspect in the prevention of horizontal transmission of pathogens between patients. Recommendations have developed over the past four decades on the importance of strict hand asepsis and the evolution of agents and methods to achieve this have similarly evolved.

A number of investigations have demonstrated a benefit to handwashing in decreasing the spread of nosocomial infections. Despite the widespread enforcement of such policies the impact on nosocomial infection rates has been disappointing. This may be due to a number of factors:

- o **Poor compliance with guidance**. This may be due to lack of education or awareness of the importance of handwashing or the large number of individuals involved with each given patient on ICU. It is everyones responsibility to enforce compliance.

- o **Workload.** There is evidence to suggest that increased staff workload or decreased staffing levels may alter compliance with handwashing guidance. Alternative methods of handwashing may offer less time consuming options, such as the use of alcohol gel on the hands rather than traditional sink and soap.

- o **Inadequate technique**. The efficiency of potential pathogen removal from the hands during handwashing varies widely by technique, time taken and agent used. Even with a thorough and prolonged technique clearance is rarely complete. There may be reluctance to repeatedly wash the hands due to irritation of the skin or dryness.

- o **The contribution of horizontal transmission to nosocomial infection on ICU is overestimated.** Studies using this criteria estimate that only 40% of nosocomial infections on ICU are due to the later pathogens and are amenable to prevention by good handwashing practice and the other 60% being established prior to arrival on ICU are not.
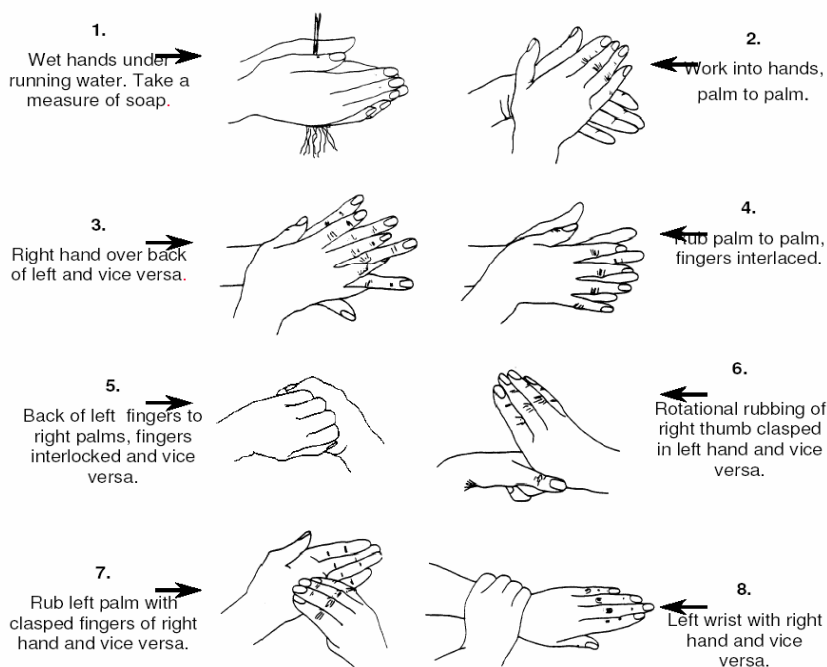
Current guidelines recommend

§ use of soap and water when hands are visibly dirty or contaminated and for any patient with Diarrhoea.   § use of alcohol gel for routine decontamination.   § It is recommended prior to each patient contact, prior to invasive procedures, after removing gloves and after hand contamination.   § Alcohol gels are found on ICU at every bed space and are mounted on the walls at entry-exit points
§ Removal of wrist watches and stoned rings is also important prior to patient contact   § Don't lift bin lids with hands!   § Full details on technique of handwashing are available in IC policy 03. The technique is shown below.

Handwashing Technique

Wash hands using the following 8 steps.
Each step consists of five strokes rubbing backwards and forwards.

1.
Wet hands under running water. Take a measure of soap.

2.
Work into hands, palm to palm.

3.
Right hand over back of left and vice versa.

4.
Rub palm to palm, fingers interlaced.

5.
Back of left fingers to right palms, fingers interlocked and vice versa.

6.
Rotational rubbing of right thumb clasped in left hand and vice versa.

7.
Rub left palm with clasped fingers of right hand and vice versa.

8.
Left wrist with right hand and vice versa.

Rinse hands under running water and dry thoroughly

3.222 Barrier methods   see IC policy 04

- In a similar vein to hand-washing it is recognized that staff transfer potential pathogens from patient to patient via items of clothing and medical instruments

- The use of gowns and gloves as a barrier method reduces the colonisation of the health care worker and the transmission of pathogens already colonising the staff member. Up to 65% of healthcare workers will contaminate their clothes when routinely caring for patients with MDR pathogens such as MRSA. Studies have evaluated the addition of gowns (disposable aprons) to gloves alone as a method and the majority show a benefit.

- It is therefore policy on UHNS critical care units to

  > - Don a disposable apron at each bed space whilst engaging in patient contact
  > - Don disposable gloves at each separate bed space
  > - Consider eye or face protection
  > - Remove outdoor coats prior to entering the unit

- Utilize stethoscopes individual to each bed space for examination. If a stethoscope is taken from bed space to bed space it should be cleaned with alcohol
- Ensure visitors to the unit and medical staff form outside the unit adhere to the above policy: nursing staff are encouraged to regard intentional breaches of the policy as an adverse incident.

3.223 Isolation and cohorting   **see IC policy 06**

- There are two types of isolation employed in critical care units:

  o Protective isolation   the isolation of immunocompromised or neutropenic patients to reduce the potential for opportunistic infections.

  o Source isolation   the isolation of colonised or infected patients to minimise potential transmission to other patients or to staff on an ICU

- The gold standard for isolation is the geographical isolation of the infected/colonised patient in a side room on the ICU. Limited availability of rooms and other organisational issues have lead to the isolation within an area on the open ward and grouping of such infected patients together with dedicated staff, a practice referred to as cohorting.

o Each intensive care unit has isolation facilities: MIU (ward 11) has a side room and ICU (ward 115) has two side rooms.

o **Isolation** of patients either colonised or infected with multi-drug resistant pathogens is a common and historical practice employed in healthcare. The clear rationale is that by physically restricting access to the patient the spread of the MDR pathogen via the carriage route (such as on healthcare staff) is reduced, that the contamination of the environment adjacent to the patient is restricted and the visual and psychological reinforcement to practice good basic infection control measures, such as hand-washing and donning gowns is maximised.

o Isolation within separate rooms on ICU is not without problems. The separation from the remaining patients will increase nursing workloads and alter allocations and dependence. There is some evidence that isolated patients suffer more preventable adverse outcomes (pressure sores, falls, fluid or electrolyte disorders), are more dissatisfied with their care and have less documentation in their notes.

o There is a lack of consensus in the literature as to whether isolation is effective in controlling spread of nosocomial infections and its exact effect is likely to depend on the pathogen, the population and the compliance with other infection control measures.

o **Cohorting** of patients is the practice of grouping patients in area of the unit or ward in whom similar MDR pathogens have been isolated, with nursing by dedicated staff. It has the advantage of not requiring separate rooms and has less of an impact on nursing dependencies than isolation but would require more vigorous enforcement of standard infection control measures than isolation.

o Practical aspects of isolation include

> o Hands washed prior to and exiting room
> o Aprons donned on entrance to room
> o Appropriate notices
> o Room specific equipment
> o Separation of waste bags and sharps containers

o The indications for isolation are

> o On advice of Infection control team
> > e.g. certain cases of MRSA where potential for spread is high
> > Other communicable diseases
> o Isolation of MDR pathogen in sputum e.g. ESBL gram negative, acinetobacter, VRE etc.
> o Clostridium difficile

3.224 Eradication therapy

o See section 3.36 for eradication regimes for MRSA

o Currently patients are screened on admission to ICU or MIU via swabs of nose, mouth, perineum and axilla and thereafter weekly. Tracheostomy swabs are indicated if the patient has a long term tracheostomy.

3.225 Aseptic technique

o This is detailed in the section on invasive procedures (section 11).

3.226 Specific infections and their prevention

o The following infections are considered in section 3.4 :

- o Ventilator associated pneumonia
- o Catheter related blood stream infections
- o Surgical site infections

o Section 3.4 also considers the methods which are implemented to attempt to reduce these infections.

Section 3.3 Antimicrobials and microbiology on ICU

- The frequency of antibiotic resistant health care associated infections has increased over the past two decades and nowhere is this more apparent than in ICU and in the acutely ill patient.

- Most studies show that the proportion of gram positive infections as a primary cause of the nosocomial infection has been increasing and the proportion of gram negative decreasing. The nature of the gram negative pathogens is, however, concerning as there has been a growth in the proportion of resistant bacteria in that proportion. Also of note is the increasing rate of fungal infections on ICU.

- In the recent SOAP study of European ICUs the incidence of infecting organisms for patients with sepsis are summarised below. This includes patients admitted with sepsis and those developing sepsis on ICU.

3.31 Risk factors for nosocomial infection

- Independent risk factors have been determined in a number of studies of nosocomial infections on ICU. Most of these are deductive and relate to factors which depress host immunity or breach natural defence mechanisms.

- These may be divided as follows

Patient factors

- o Severity of illness

- o Shock on admission
- o Age > 60 yrs
- o Neurological failure at day 3

Therapeutic factors

- o Parenteral nutrition
- o Antimicrobial therapy
- o Central venous access
- o Days with arterial line
- o Mechanical ventilation
- o Tracheostomy
- o ICP monitoring

Organisational factors

- o Prolonged length of ICU stay
- o Size of ICU (> 10 beds)
- o Understaffing of unit

- The impact of these general factors is modified by more specific risk factors for certain infections. The impact of organisational aspects must not be underestimated. Several studies have shown that overcrowding on ICU or understaffing of ICU correlates with increased nosocomial infections. This may be due to pressures on individuals workload resulting in sub-optimal infection control procedures, such as hand washing, donning of aprons and care of CVC lines. This encourages cross transmission of pathogens between patients via staff members.

- More severely ill patients are susceptible to nosocomial infections due to a relative immunodeficiency (such as the immunoparesis seen in sepsis). The breaching of natural body defences with invasive devices (such as endotracheal tubes, CVC, urinary catheters, ICP bolts) promotes this. Scoring systems such as APACHE II/III or SAPS II / 3 allow some quantification of illness severity but it must be bore in mind that the systems were designed for mortality prediction in populations and perhaps sequential organ failure scores such as SOFA may be more appropriately used.

- Specific nosocomial infections also carry specific risk factors. For example risk factors for ventilator associated pneumonia (VAP) includes supine positioning, enteral feeding, use of stress ulcer prophylaxis, re-intubation and prolonged ventilation.

3.32 Organisms producing nosocomial infections on ICU

- The most common organisms involved in nosocomial infection on ICU are

    - Gram positive bacteria
        - Methicillin-sensitive *staph aureus*
        - Methicillin-resistant *staph aureus*
        - Coagulase negative staphylococcus
        - *Enterococcus* (a proportion of which are vancomycin resistant)
    - Gram negative bacteria
        - *Pseudomonas aeruginosa*
        - *Eschiceria coli* (some of which are ESBL producing)
        - *Klebsiella* (some of which are ESBL producing)
        - *Stenotrophomonas maltophillia*
        - *Enterobacter*
        - *Acinetobacter*
    - Anaerobes
    - Fungi
        - *Candida albicans*
        - *Candida* (non-albicans)

- Of particular concern is the increasing incidence of resistant pathogens producing infection in the acutely unwell patient. These include: MRSA, Coag negative staphylococcus, vancomycin resistant *enterococci* (VRE), ESBL producing *Klebsiella*, *enterobacter*, *Ps aeruginosa*, *Acinetobacter* and *Stenotrophomonas maltophillia*.

3.33 Colonization versus infection

- Always consider in patients when results are returned from microbiology whether the result represents infection or colonization. The definitions of colonisation versus infection are important in discussing the management of the ICU or critically ill patient.

    Contamination is the presence of bacteria at a site (e.g. a surgical wound) prior to multiplication taking place.

    Colonisation is the presence of multiplying pathogens with no overt host response or clinical symptoms. At critical colonisation the host defences are unable to maintain the balance of organisms at colonisation.

    Infection occurs when the multiplying pathogen overwhelms the host defence. The definition of infection in the 1992 ACCP/ SCCM guidelines is "microbial phenomena characterised by inflammatory response to the presence of micro-organisms or to the invasion of normally sterile host tissue by those organisms".

- Colonisation occurs readily in hospital environments, particularly in areas such as critical care where the colonisation pressure is high. Critically ill patients are susceptible to colonisation with MDR pathogens for several reasons:

    i. Prolonged hospital stay
    ii. Use of broad spectrum antibiotics
    iii. Close proximity to other colonised patients
    iv. Immunosupression (absolute and relative)
    v. Co-morbidity and chronic disease
    vi. Use of CVCs, use of endotracheal tubes

- One of the key difficulties in critical care is determining whether isolation of a potential pathogen from a sample represents colonisation or infection. A second areas of interest is whether colonisation requires treatment, namely does colonisation lead to infection.

So is it infection?

- Infection is a both a microbiological and clinical diagnosis. Infection may be suspected in the absence of microbiological evidence in patients whom have typical features of an infective response. Unfortunately in the critically ill many of these are non-specific.

- Signs of infection may include:

    - General signs of an inflammatory response
        o Pyrexia or hypothermia
        o Leucocytosis or leucopenia
        o Tachycardia, tachypnoea
        o Raised inflammatory markers (CRP, PCT)
    - Specific signs relevant to infective site
        o Sputum production
        o Localised erythema, pain, induration
        o Presence of pus

Isolation of a potential pathogen in absence of the above is likely to represent colonisation not infection. Isolation with the above may also represent colonisation with some other process driving an inflammatory response. In these situations clinical judgment must be used to decide whether to treat the pathogen. This will be guided by knowledge of usual infection patterns, other symptoms and signs, prior antimicrobial usage and magnitude of the host response (e.g. organ dysfunction).

Section 3.34 Antibiotic regimes

- The UHNS antimicrobial guidelines can be found on the intranet in clinical section / guidelines / antimicrobial guidelines / adults treatment and antimicrobial drugs. This should be used when staring any antibiotics and provides the most up to date guidance. If in doubt discuss with ICU or Microbiology Consultant.

- For signs and severity of infection see section 3.1

- In the vast majority of cases antimicrobials should be given initially IV on intensive care. Absorption and hence bio-availability can be poor in unwell critically ill patients. Conversion to oral antibiotics may be considered at later stages of treatment.

- Antibiotic prescriptions should be reviewed regularly. A typical course of antibiotics on ICU will be 5-7 days. The need should be reviewed prior to this based on clinical picture and microbiology. In some cases the course will be longer than 7 days: for example CNS infections or C.difficile. Ideally this should be on microbiology advice.

- A microbiology round takes place once a week on each unit: Wednesday 12pm at the CGH ICU and Friday 12pm at the MIU. This provides an opportunity to discuss new results, antibiotic regimes and other aspects of care.

Section 3.4 Specific infections on ICU

o Although virtually all infective processes can be encountered in critical care there are a few nosocomial infections that predominate in the ITU. These generally relate to the key interventions on critical care, that of ventilation and cardiovascular monitoring.

o The commonest nosocomial infections on ICU are thus:

   o Ventilator associated pneumonia (VAP)
   o Catheter related bloodstream infection (CRBSI) – ' line sepsis'
   o Surgical site infections (SSI)

o Each of the subsections below briefly details the aetiology, diagnosis of, prevention of and treatment of the specific infections.

Section 3.41   Ventilator associated pneumonia

o VAP is a subtype of hospital acquired / healthcare associated pneumonia particular to patients receiving invasive ventilation.

o The etiology of VAP has been extensively investigated and appears to be due to introduction of potentially pathogenic micro-organisms (PPM) into the pulmonary tree and subsequently the lung parenchyma. This introduction may occur via:
   o Pooling of contaminated secretions above the ETT cuff and micro-aspiration
   o Introduction of contaminated secretions via suctioning
   o Haematogenous spread (rarely)

   o In turn the PPM are colonizing pathogens that grow in the upper GI tract, oral flora, nasal flora or skin. The growth is predisposed by various factors found in ICU patients, such as altered gastric pH, enteral feeds, impaired host defences and use of antibiotics.

o Prevention of VAP focuses on these factors. However not all are remediable to correction. These are detailed below and then the ventilator care bundle is detailed.

   1. **Avoiding intubation.** As intubation and ventilation are significant risk factors strategies employing alternatives such as *non-invasive ventilation* may reduce VAP rates. Failed trials of extubation and re-intubation greatly increase the risk of VAP and should be avoided if possible and the use of *tracheostomy* may allow this. The benefit of early versus late tracheostomy appears to favour early intervention but work is still ongoing into this aspect of care.

      Duration of ventilation correlates with risk of developing VAP, with the initial period carrying the greatest increase in risk. Thus the use of *weaning protocols* could be considered a preventative measure as should the use of *sedation holds.*

   2. **Aspiration of subglottic secretions**. Pooling of secretions around the cuff leads to micro-aspiration and subglottic suctioning may reduce VAP rates at the expense of potential mucosal trauma.

   3. **Body positioning**. Early work has suggested that semi-recumbent position reduces the rate of VAP and mortality when compared with supine position. However very few patients remain supine for prolonged periods on ICU and maintaining semi-recumbent posture throughout the day on ICU can be difficult and more recent work indicates the value of this intervention may have been over-estimated.

   4. **Enteral feeding**. By neutralising the pH of the gastric contents and promoting bacterial growth as well as increasing the aspirates enteral feeding is a risk for VAP. Alternatives such as parenteral nutrition

unfortunately increase the risk of other infective complications and as such this is not an easily modifiable risk factor. There is some evidence that *small bowel* versus gastric feeding may reduce aspiration rates and VAP.

5. **Selective decontamination of the digestive tract**. This is not practiced in our unit. We use oral decontamination with Chlorhexidine mouth Gel.

6. **Stress ulcer prophylaxis**. Both H2 antagonists and antacids have been identified as independent risk factors for VAP. The results of the numerous trials comparing the risks of stress ulceration and haemorrhage versus the benefits of reduced VAP rate have been conflicting and many ICU will have individual policies depending on their interpretation of the evidence and risk of their population.

7. **Transfusion practice**. The use of a *conservative transfusion strategy* may reduce the incidence of VAP in the ICU population. This may be related to the nature of the blood transfusions utilised in the study and the effects of a conservative strategy using leuco-depleted blood is not as clear.

o These measures are incorporated into a package of simple evidence based interventions known as a care bundle (see www.ihi.org/IHI/Topics/CriticalCare for more information on the approach). These "bundle" concepts have been further developed by the UK modernization agency via clinical networks and via the "Saving lives" campaign as high impact changes.

o Further details of care of the ventilated patient can be found in section 1

o The ventilator care bundle is

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1. **Elevation of the head of the bed to 30-45 degrees**
2. **Daily sedation holding**
3. **DVT prophylaxis** *ventilation is a risk factor for ulceration on ICU and hence benefits outweigh the risk of increased VAP*
4. **Gastric ulcer prophylaxis**
5. **Appropriate humidification of inspired gases**
6. **Clean suctioning of respiratory secretions** *wear examination gloves and decontaminate hands before and after the procedure*
7. **Tubing management** *only replace when soiled or damaged*

---

o Points 1,2,5,6 and 7 relate to prevention of VAP.
o Diagnosis of VAP can be difficult in an ICU patient on the ventilator. The diagnosis can be made on clinical suspicion +/- microbiological evidence.

o Diagnosis of VAP

1. radiological evidence of new or progressive infiltrates on CXR plus two out of

2. abnormal white cell count (WCC < 4 or WCC > 11)
3. abnormal temperature (temp < 36 or > 38)
4. purulent secretions

o this is only a guide and ultimately the decision is based on clinical suspicion. Other considerations may be:
      -microbiological evidence (e.g. significant growth of pathogens)
      -deteriorating gas exchange

o the treatment of VAP depends upon the time frame of onset and prior history (see section 3.33 and 3.35)

o if VAP at < 4 days then use antibiotics for community acquired pathogens
o if VAP at 4 days or more or if prior antibiotic usage / recent hospitalization/ care in long term facility (see 3.33) then use antibiotics to cover hospital acquired pneumonia

Section 3.42   Catheter related blood stream infections

o Catheter related blood stream infections (CRBSI) are nosocomial infections related to central venous lines (including vas-caths and pulmonary artery catheters). They are often referred to as "line sepsis".

o Full guidance on hospital protocols for CVC and CRBSI can be found on the intranet at clinical section/infection control/general infection control in patients/guidelines/central venous catheterisation. These guidelines are heavily based on the CDC guidelines for prevention of CRBSI [see references].

o CRBSI are the second commonest nosocomial infection on the ICU not least because of the ubiquitous nature of the CVC in ICU care. This section considers the aetiology of CRBSI, the prevention of CRBSI (again via the care bundle approach), the diagnosis of CRBSI and the treatment.

o CRBSI develop due to colonisation of the CVC and spread via the CVC into the patients blood stream. There are two ports of entry: the insertion site of the CVC where it breaches the skin (extra-luminal) and the lumen(s) of the CVC where colonisation may spread from the catheter hub (intra-luminal). CVCs may also become colonized as a secondary phenomenon to haematogenous spread from distant sites, although this is unusual.

o Prevention of CRBSI focuses on cleanliness at time of insertion and continuing cleanliness of the line after insertion.

Site of insertion. Current evidence appears to indicate the incidence of CRBSI is lower with subclavian CVCs versus femoral CVCs. Comparison with IJV catheters is limited in the literature. Logically SCV catheters should be the least prone to site infection as IJV catheters may well be contaminated with oral spillage from intubated patients and may be subject to greater movement at insertion sites fixation points. Femoral lines in addition to being located in an area prone to colonisation have a significantly higher incidence of thrombosis formation which may promote colonisation of the line. However the relative risk of SCV insertion compared with the other two routes in critically ill ventilated patients must be borne in mind.

Catheter features. The use of anti-microbial agent impregnation of the CVCs has increased over recent years. Catheters may be impregnated with minocycline-rifampicin or silver sulphadizine-chlorhexidine. The use of these catheters decreases colonisation and may decrease CRBSI. They are recommended in high risk patients and in ICU where CRBSIs persist despite good adherence to other preventative measures. There is concern that they may increase colonisation with non-bacterial pathogens such as *Candida*.

Aseptic technique. Attention to aseptic technique is paramount for preventing line sepsis. Migration from the skin insertion site into the cutaneous tract is the most common route of infection in CRBSI followed by contamination and colonisation of the catheter hub via exogenous sources. Good hand hygiene and maximal barrier precautions (gloves, hats, mask, gown, large drapes) improve the incidence of CRBSI when compared with more basic precautions (e.g. just gloves and a small drape). The use of skin preparation is mandatory and both povidine-iodine and chlorhexidine may be used. Some evidence indicates chlorhexidine results in less colonisation than iodine but the CRBSI rates did not differ.

Use of ultrasound. The use of ultrasound to assist in the siting of internal jugular CVCs has been shown to reduce the number of attempts required, the complication rate and time taken to insert the CVC. The reduction in mechanical complications may be accompanied by a reduction in haematomas and localised tissue trauma which may lead to fewer CRBSI.

Post-insertion care. Excessive manipulations of CVCs post-insertion increase the risk of CRBSI. Inappropriate post-insertion care similarly increases the risk. Evidence indicates that continuing quality improvement programmes reduce the incidence of CRBSI, primarily by re-iterating good practice in the care and handling of the CVC. Potential benefit exists in the implementation of specialised CVC nursing teams whom take responsibility for all CVC care within an institution and education regarding infection control issues. On ICU some evidence exists to demonstrate the relationship between nursing staff levels and the risk of CRSI.

Removal of CVCs. Although most guidelines do not recommend routine replacement/ exchange of CVCs in critical care or guide wire exchanges for suspected CRBSI, the probability of colonisation and infection in CVCs does

increase with time. Therefore it is prudential to remove CVCs as early as possible. The risk increases mainly after 5 to 7 days post-insertion.

Parenteral feeding. The risk of CRBSI increases when a CVC lumen is utilised for parenteral nutrition. Meticulous infection control procedures must be undertaken when handling lines for TPN and ideally the lumen utilised must be dedicated for TPN. Some ICUs insist on a new CVC if parenteral nutrition is to be commenced to minimise the risk of CRBSI.

o The care of these lines on ICU is protocolised in High Impact intervention number 2 (a care bundle) with 9 points

1. catheter type   single lumen unless indicated otherwise (not usually possible on ICU)
2. insertion site – subclavian or IJV
3. alcoholic chlorhexidine gluconate solution skin prep
4. prevent microbial contamination
        hand hygiene
        personal protective equipment (gloves and apron)
        safe disposal of sharps
        aseptic technique
5. regular (daily or more) observation of the insertion site
6. catheter site care   intact clean dressing
7. catheter access   aseptic technique when accessing cathere ports
8. fluid administration   fluid bags for no longer than 24 hours
9. no routine catheter replacement

10. consider need for CVC daily and remove line as soon as possible

11. if TPN needed then dedicated lumen required .If one is not reserved at time of insertion for this purpose then a fresh CVC must be inserted

o CRBSI can be a difficult diagnosis to make. The problem comes with colonisation versus infection. A liberal approach to line replacement is not without risk however and thought should be given before deciding to remove a CVC because of suspected infection. Some indicators may be

1. line in- situ > 5 days
2. line placed in sub-optimal conditions (e.g. ward setting, A&E)
3. patient on TPN via CVC
4. femoral line

5. Evidence of infection at insertion site (erythema, induration, pus)
6. positive blood cultures from separate site
7. positive cultures from CVC, especially if matching blood cultures from other site
8. evidence of sepsis
    a. raised or low WCC
    b. pyrexia or low temperature

   c. hypotension, increasing vasopressor requirements, tachycardia
   d. raised lactate
   e. other organ dysfunction

- o if in doubt the line should be removed and replaced if necessary. It is advisable to place a fresh CVC at a new site if a line is removed for suspected infection.

- o Treatment of CRBSI involves

1. remove suspect CVC; send tip for MC & S
2. re-site CVC in fresh location
3. draw two sets of blood cultures from fresh line
4. commence antimicrobials
  a. vancomycin (as per protocol see section 3.36)
  b. tazocin or meropenem
  c. consider fluconazole (if previous cultures showed candida colonisation, if severe sepsis, if neutropenic, surgical patients)
5. review microbiology results at day 2-3 and narrow spectrum as appropriate
  a. usual pathogens are coag negative staphylococci, staph aureus, gram negative bacilli and candida
6. ensue documentation of line change in notes and in record at back of notes

Section 3.43 Surgical site infections see surgical guidelines 2006 p172

- o Surgical site infections (SSI) are an infrequent infection on ICU occurring in post-operative patients and in all ICU patients from old cannula and CVC /ART sites.

- o Suspicion of SSI will come from repeat examination of the surgical wound and may include

1. superficial erythema around wound edges
2. swelling of wound with discharge
3. fluctuation to the wound
4. increasing wound tenderness
5. spreading erythema
  o this indicates a cellulitic process
6. systemic symptoms of sepsis (see section 3.1)

o Typically wound infections present 3-4 days post-operatively

o Management would include

> o Swab wound and send for MC&S
> o Discuss with surgical team regarding draining, packing or removal of sutures
> o Consider antibiotics if
> > § Spreading erythema (suggesting cellulitus)
> > § Possible deep infection
> > § Systemic symptoms
> o If antibiotics indicated give
> > § Flucloxacillin 1 gram IV 6hrly
> > § Benzylpenicillin 1200 mg IV 6hrly
> > § If previous MRSA colonisation give vancomycin
> > § If penicillin allergic consider clindamicin

o A severe form of SSI / soft tissue infection is that of necrotizing fasciitis. **Seek senior advice on managing this condition**. There are two types
   o Type 1 involves gram negative bacilli and anaerobes
   o Type 2 involves β haemolytic streptococcus

§ Classically type 2 involves a toxic shock like syndrome. In practice type 1 patients are usually severely septic and it can be difficult to distinguish the two. Urgent gram staining can allow rationale prescribing in some cases. Antibiotic treatment according to hospital guidelines / Microbio advise.

Section 3.5 Immunosupressed patients on critical care

- The immunocompromised patient provides a number of challenges to the intensivist, not least because of the poor general prognosis of this group of patients.

- Many patients on ICU have some degree of immunocompromise. This may be overt such as admissions with haematological malignancy or immunosupression with drugs or more covert such as the relative immunosupression occurring with recovering / late phase sepsis, metabolic processes or use of sedatives and inotropes.

3.51 Causes of immunosupression

- One way of classifying immune suppression on ICU is presented. There are two broad categories

    1. Treatment induced immune dysfunction
        § Physical therapies (plasmapheresis, whole body irradiation)
        § Immunosuppressant drugs e.g. post transplant
        § Chemotherapeutic agents
        § Immune modifying drugs (e.g. steroids, immunoglobulins)
        § Subtle immune modifying drugs (e.g. sedatives, inotropes)

    2. organic immune impairment
        § inherited defects in immune system
            1. innate system abnormality
            2. antibody deficiency *e.g. common varied immunodeficency*
            3. cellular immune deficiency *e.g. thymic aplasia, SCID*
            4. complement deficiency
            5. phagocyte defects
        § isolated neutropenias (*e.g. in sepsis, burns*)
        § haematological malignancies *e.g. leukaemia, lymphoma, myeloma.*
        § T cell dysfunction *e.g. HIV/ AIDS, CMV, measles*
        § B cell dysfunction
        § Other (non haematological) malignancies
        § Chronic diseases
        § Chronic infection
        § Chronic alcohol addiction
        § Critical illness immune dysregulation (seen in severe sepsis)

- In general patients will either be referred to ICU because of complications of their immunosuppressive process (e.g. opportunistic infection) or be patients who have another critical illness process and the immunosuppression as a separate process. **All admissions of these patients must be discussed with senior ICU clinician.**

3.52 Management of the neutropenic patient

- The neutropenic patient and their management provides a paradigm for the management of immunosupressed individuals.
- Neutropenia is defined as a neutrophil count below $1 \times 10^6/ L$ and risk of opportunistic infection increases as the count falls, being particularly high below 0.1.

- Typically neutropenia occurs 7-14 days post chemotherapy. It may also occur in the context of haemotological malignancies and due to overwhelming sepsis.

- The management of the neutropenic patient is detailed in the UHNS Medical Guidelines 2006 page 48. In brief:

  o In first instance resuscitate as per guidelines in section 3.1 Sepsis

    § Fluid resuscitation
  o Consider inotropes and vasopressors
  o Take cultures as appropriate
    § Sputum
    § Urine
    § Blood cultures (x2)
    § Line cultures +/- line removal and tips for MC&S
    § Consider CSF cultures via LP

  o Also consider need for anti-fungal agents (e.g. liposomal amphotericin, voriconazole, caspofungin) and the risk of MRSA sepsis (wher vancomycin would remain the first choice). Advice from microbiology should be sought.

- Neutropenic patients are typically referred to ICU for one of two reasons:

Sepsis

- Severe sepsis (see section 3.1) is a common presenting syndrome for the neutropenic patient and unfortunately the source is not always apparent. As a result a broad approach as detailed above is recommended and an aggressive search for the source advocated if the patient is appropriate for critical care.
- The appropriateness of the patient will depend on a number of factors including age, previous admissions, duration of admission prior to referral, cause of the Neutropenia and prognosis and reversibility of the underlying disease process. This is valuable information to glean prior to discussion with the consultant.

- Treatment of the sepsis follows the guidelines in section 3.1. Xigris (rhAPC) would probably not be appropriate for many of these patients on the basis of co-existing coagulation abnormalities, survival to 28 days being unlikely and the exclusion from the PROWESS study of HIV patients.

Respiratory failure

- Respiratory failure is the commonest reason neutropenic and immunosupressed patients are referred to ICU. A detailed approach to managing the problem can be found in Azoulay 2006 (see references).

- Its incidence is surprisingly high in this patient group: up to 50% of hematological malignancy patients, especially those following bone marrow transplant and 30% of neutropenic patients.

- Its cause can be

    o Infective (bacterial, viral, fungal, mycobacterial)
    o Non-infective
        § Alveolar hemorrhage
        § Cardiogenic pulmonary oedema
        § ALI / ARDS
        § Lung infiltration
        § Drug toxicity (e.g. bleomycin)
        § Bronchiolitis
        § Cryptogenic organizing pneumonia (COP)
        § Radiation injury
        § Secondaries/ metastases

- The prognosis of this patient group is amongst the worst of all ITU patients. This poor prognosis is greatly worsened if the patient is invasively ventilated and may approach 90-100%. As a result management is based primarily around use of non-invasive strategies e.g. CPAP, BiPAP.

- The precise management strategy depends on the likely cause and reversibility of that condition.

3.53 The HIV + patient on ICU

- Admission to ICU with the complications of HIV or AIDS is unusual in the UHNS critical care unit. HIV may be detected incidentally in the investigation of sepsis / infections in critically ill patients and should always be suspected if infections appear atypical. Finally HIV positive patients may be admitted for other reasons and have HIV as an incidental chronic disease state.

- Generally the care of these patients involves close links with the Infectious Diseases consultants. For an in depth review see Rosen 2006 [ref 12] and Huang 2006 [ref 13].

- With the advent of effective HAART the incidence of HIV associated infections as a reason for ICU admission is declining and non-HIV associated infections are increasingly the reason for admission. The admission CD4 count provides some indicator as to the potential causes of infection or decline in the HIV patient and treatment pathway:

    - CD4 200-250   TB, Bacterial pneumonia, oral candiadisis
    - CD4 125-200   Kaposi's sarcoma (from HHV8)
    - CD4 75-125   PCP, cerebral toxoplasmosis, cryptococcal meningitis
    - CD4 < 50   CMV retinitis

- End stage HIV infection /AIDS is clearly not an indication for ICU admission.

- It can be seen that with CD4 counts < 200 that the incidence of AIDS defining infections increases. In these patients there may well be benefit in instituting HAART even if the patient has not been on the treatment prior to admission. The ID team will advise on this.

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Section 4.1 Guidelines for Management of ARF in ITU

4.11 Recognition and Assessment

- Acute renal failure (ARF) is a rapid decline in renal excretory function, acid-base balance and removal of solutes and water
- Acute kidney injury is defined as an abrupt (within 48 hr) reduction in kidney function determined by an absolute increase in serum creatinine of either 26.4 µmol/L or a percentage increase > 50% (1.5 x baseline), or a reduction in urine output documented as oliguria <0.5 mL/kg for 6 hr

Recognition

1	Increase in creatinine >26.4µmol/L or 1.5-2 fold increase from baseline	<0.5 mL/kg for 6 hr
2	Increase in creatinine >2-3 fold from baseline	<0.5 mL/kg for >12 hr
3	Increase in creatinine >3 fold or serum creatinine >350 µmol/L or acute rise of >44 µmol/L	<0.3 mL/kg for 24 hr or anuria for 12 hr

Groups at higher risk
- Pre-existing chronic kidney disease
- Age >60 yr
- Sepsis
- Cardiac failure
- Diabetes
- Cirrhosis
- Myeloma

4.12 Prevention

- Be alert to recognize patients at risk (e.g. hypovolaemic states and presentation with sepsis)
- Once identified as at risk or has developed AKI start MEWS scoring if patient seen outside ICU to detect further deterioration at early point. Write appropriate monitoring plan in notes and inform nursing staff.
- Do not overlook simple interventions (e.g. adequate fluid replacement and discontinuing nephrotoxic drugs in at-risk individuals)
- Minimize risk of acute kidney injury associated with radiographic contrast media – see **Prevention of contrast induced nephrotoxicity** guideline on intranet.
- Discontinue NSAIDs, diuretics, ACE inhibitors and angiotensin-receptor blockers (ARBs), in at-risk groups
- ensure adequate hydration with IV fluids.
- consider alternative imaging modality, ultrasound or MR

4.13 Causes

The development of ARF increases morbidity and mortality. In the intensive care setting it is most often associated with multiple organ failure and the presence of sepsis. If the need for Renal Replacement therapy occurs in ITU then mortality is in the order of 40-60%.

The causes of ARF are almost always multifactorial but usefully broken up into:

Ø Pre-Renal   Decreased effective arterial blood volume
                              Poor Cardiac function
                              Renal artery or vein occlusion

Ø Renal   Intrarenal Vascular
                              Glomerulonephritis
                              Ischaemic ATN
                              Toxic ATN
                              Tubulointerstitial Nephritis
                              Intrarenal obstruction

Ø Post-Renal   Urinary tract obstruction

> *Hospital-acquired renal failure is often multifactorial, with contributions from hypotension, sepsis and drugs*
> *Risk of ARF resulting from obstruction or renovascular disease is greater in patient with single kidney*

4.14 Initial Management

Most cases in ITU will be as a result of Pre-Renal ARF. Therefore the aim should be a restoration of adequate renal perfusion in the first instance.

- Correction of Hypoxia

- Correction of Hypovolaemia

- Optimise Cardiac Output

- Aim Mean Arterial Blood Pressure >65mmHg

- Urinary Catheter to monitor Urine output and response to above interventions

- Check catheter is not blocked with lavage

Then

- Urine Dipstix Testing - Presence of haematuria/proteinuria may indicate acute glomerulonephritis/vasculitis

- Blood and other body fluid cultures as sepsis common precipitant

- Drug chart – Nephrotoxins stopped and dose reductions as needed

- Consider Vascular disease and Parenchymal Renal disease i.e. think about underlying diagnosis.
  -
- Consider a) Plasma protein and urine electrophoresis and b) Immunology screen (ANA/ANCA/C3/C4/Anti-GBM)

- Urgent Renal Ultrasound (daytime)

- Gastric protection with Ranitidine dose adjusted.

The use of Furosemide to convert from oliguric to non-oliguric renal failure maybe considered if haemodynamics have been optimised or there is evidence of Fluid overload.

Institute an appropriate fluid regime after resuscitation to manage patient e.g. INPUT = URINE OUTPUT + 30ml/hr

Adjust for fluid deficit or higher insensible losses.

4.2 Renal Support

The use of the Renal Drug Handbook is important in dose adjustment.
4.21 Indications for Renal Replacement Therapy

- Uraemia – Urea >25mmol/l in ITU patients
- Oliguria with fluid overload
- Hyperkalaemia – K >6.5mmol/l
- Severe Acidosis
- Extracorporeal Drug Removal

4.22 Continuous Renal Replacement Therapy

All patients on CRRT should have

- Gastric protection with Ranitidine 25mg tds
- 4 hourly Arterial or Venous Blood Gas to check potassium level
- Daily monitoring of Magnesium and Phosphate levels
- Commence replacement of water soluble vitamins with Pabrinex preparation 2 vials daily or started on day 1 with full replacement if thought to be nutritionally at risk.

Access

The most important part of the Renal Replacement Therapy circuit is the vascular access, because if it is poor the result will be inefficient therapy and multiple circuit losses. **If multiple line circuits are lost this should prompt consideration of line replacement.**

The current line used by the unit is Medcomp Silicone 13.5 french gauge line and is available in 15, 20 and 24cm lengths. The following choice of line should be applied as per access site:

RIJ 15 cm length
LIJ 15cm length if patient height < 165cm or 20cm length if >165cm
Femoral always 24cm line

Please ensure that the dialysis line is locked with Heparin 1000u/ml as per the line volume given on each limb of the line after insertion.

Modality

The 3 types of Renal replacement therapy currently used at UHNS are:

a) CVVHD

CVVHD uses diffusive solute clearance against a counter current flow of dialysis fluid. This is current practice on MIU/ICU with plans to move to CVVHDF in July 2010.

The standard prescription of CVVHD uses a 2000ml dialysis fluid cycle per hour which gives a default clearance of 33ml/min.

If the patient is less than 50kg actual body weight then 1500ml/hr dialysis fluid flow rates maybe sufficient to maintain biochemical control. Patients greater than 80kg will often need higher dialysis doses and therefore rates of 2500 ml/hr should be used.

With CVVHD a fixed Blood flow rate should be targeted to 150ml/min.

b) CVVH

CVVH uses convective solute clearance. This is current practice on Cardiothoracic ICU with plans for change to CVVHDF in July 2010. Current practice is aiming for Haemofiltration rates of 35ml/kg/hr with titrated blood flow rates according to fig 4.22.1

c) **CVVHDF (this will become default treatment from July 2010)**

CVVHDF uses both diffusive clearance of solute and convective clearance of solute with bulk solvent movement. Prescription using CVVHDF should be dosed according to actual body weight and prescribed to deliver 30 ml/kg/hr total dose. This total dose should be split 50:50 to give to the nearest 100ml equal amounts of Haemofiltration and Haemodialysis. For example a 70 kg man would require 2100ml total dose / hour and receive 1100ml dialysis and 1100ml Haemofiltration. This strategy effectively ensures a minimal dose of 25ml/kg/hr for 24 hours allowing for >80% efficiency with down time off CRRT.

Blood flow rates with CVVHDF will need to be adjusted according to Haemofiltration dose to avoid a filtration fraction greater than 0.30, but with a minimum blood flow rate of 150ml/min. See figure 4.22.1

Fig 4.22.1 Filtration Fraction

Exchange Rate (l/h)

Blood Pump Speed (ml/min)	0.5	1	1.25	1.5	2	2.5	3	3.5	4	4.5	5	6
50	17%	33%	42%	50%	67%	83%	100%	100%	100%	100%	100%	100%
60	14%	28%	35%	42%	56%	69%	83%	97%	100%	100%	100%	100%
75	11%	22%	28%	33%	44%	56%	67%	78%	89%	100%	111%	100%
100	8%	17%	21%	25%	33%	42%	50%	58%	67%	75%	83%	100%
125	7%	13%	17%	20%	27%	33%	40%	47%	53%	60%	67%	80%
150	6%	11%	14%	17%	22%	28%	33%	39%	44%	50%	56%	67%
175	5%	10%	12%	14%	19%	24%	29%	33%	38%	43%	48%	57%
200	4%	8%	10%	13%	17%	21%	25%	29%	33%	38%	42%	50%
225	4%	7%	9%	11%	15%	19%	22%	26%	30%	33%	37%	44%
30%	3%	7%	8%	10%	13%	17%	20%	23%	27%	30%	33%	40%
275	3%	6%	8%	9%	12%	15%	18%	21%	24%	27%	30%	36%
300	3%	6%	7%	8%	11%	14%	17%	19%	22%	25%	28%	33%
325	3%	5%	6%	8%	10%	13%	15%	18%	21%	23%	26%	31%
350	2%	5%	6%	7%	10%	12%	14%	17%	19%	21%	24%	29%

Fluids

Choice of Dialysate/replacement fluid should be a Bicarbonate buffered solution as default. Were patients develop metabolic alkalosis the renal replacement dose can be reduced.

Fluid balance is controlled by Transmembrane pressures in both modalities and a desired fluid balance is set e.g. neutral or a negative rate in ml/hr to achieve a daily fluid balance required. Net Fluid balance should be set for every patient and calculated on an hourly basis.

It is often the case that patients are fluid overloaded when they start CRRT. The evidence certainly suggested that Renal outcome is not adversely affected by negative fluid balance to attain normovolaemia. In ARDSnet trial for ARDS and fluid balance a negative balance did not significantly increase the need for CRRT.

Potassium replacement will be needed and this can be injected into the 5litre dialysate fluid bag as a sliding scale prescription and adjusted per patient.

KCl 20mmol *(4mmol/l)*   if   Serum K 4.5-5.0mmol/l
KCl 40mmol *(8mmol/l)*   if   Serum K 4.0-4.5mmol/l

Phosphate replacement should be provided as per the unit's Protocol see Section .

4.23 Anticoagulation for CRRT

Anti-coagulation is often necessary to prevent clotting of the extra-corporeal circuit. Recurrent circuit loss however is almost always secondary to vascular access problems and should be addressed by performing a change of vascular access. The main risk with anti-coagulation is bleeding. Anticoagulation however is not always needed particularly if there is any coagulopathy.

Anti-coagulation is used as standard with a sliding scale Heparin regime as below unless Platelets <50 or INR >1.5 or APTT >1.5 in which case no anticoagulation is used.

Anti-coagulant: Heparin 10000 units in 0.9% Saline in a volume of 20ml see prescription chart.

APTT ratio	Change in infusion rate
> 5.0	Stop infusion for 1hour, then reduce by 1 ml/hr or if infusion < 1.0ml/hr decrease rate by 1/3
4.1 – 5.0	Reduce by 0.6 ml/hr
3.6 – 4.0	Reduce by 0.2 ml/hr
3.1 – 3.5	Reduce by 0.1 ml/hr
2.0 – 3.0	No Change
1.5 – 1.9	Increase by 0.2 ml/hr
1.2 – 1.4	Increase by 0.4 ml/hr
< 1.20	Increase by 0.8 ml/hr

4.24 Heparin induced Thrombocytopaenia

Clinical aspects

Heparin-induced thrombocytopenia (HIT) is the most frequent drug-induced thrombocytopenia. Heparin can form a neo-antigen complex with platelet factor 4 (PF4). HIT is an immune-mediated disease in which antibodies are formed against the neo-antigen complex heparin/PF4 (HEP/PF4). These antibodies bind to the platelet membrane resulting in platelet aggregation. The incidence in non-ICU patients is about 3% with use of Unfractionated Heparin. In critically ill patients with MODS, the incidence increases to 4.7% or even higher. The antibodies can remain detectable up to 85 days.

HIT can occur with both unfractionated Heparin and Low Molecular weight Heparins (LMWH). The typical clinical features consist of a 50% or more fall in platelet count 4-14 days after starting heparin and/or evidence of new or progressive thrombosis. The thrombocytopenia can be asymptomatic, but should be regarded as a pro-coagulant state. HIT can be complicated by acute life threatening arterial and venous thrombo-embolic complications – referred to as heparin-induced thrombocytopenia and thrombosis (HITT). In CRRT, early haemofilter clotting and catheter clotting are prominent features. Mortality, in part attributable to HIT(T), is 30% in non-ICU patients and can be as high as 70% in MODS patients.

When the antibodies have disappeared after an episode of activity of HIT(T), mostly after 3 months or more, rechallenge with heparin does not necessarily result in a reappearance of antibodies and clinical manifestations of HIT(T). The exact risk of relapse is unknown. It is advocated to test the presence of antibodies again, to use the safest anticoagulation dependent on the clinical situation, and to perform daily platelet counts.

Risk of HIT should be estimated clinically (see scoring system below). If the clinical risk is intermediate/high consider stopping UFH/LMWH and start alternative anticoagulant in therapeutic doses while the HIT antibody result is awaited. Therapy with Danaparoid /Lepirudin should be started.

Table I. Estimating the pretest probability of HIT: the 'four Ts'.

| | Points (0, 1 or 2 for each of four categories: maximum possible score = 8) | | |
	2	1	0
Thrombocytopenia	> 50% fall or platelet nadir 20–100 × 10⁹ per l	30–50% fall or platelet nadir 10–19 × 10⁹ per l	fall <30% or platelet nadir <10 × 10⁹ per l
Timing* of platelet count fall or other sequelae	Clear onset between days 5 and 10; or less than 1 d (if heparin exposure within past 100 d)	Consistent with immunisation but not clear (e.g. missing platelet counts) or onset of thrombocytopenia after day 10	Platelet count fall too early (without recent heparin exposure)
Thrombosis or other sequelae (e.g. skin lesions)	New thrombosis; skin necrosis; post heparin bolus acute systemic reaction	Progressive or recurrent thrombosis; erythematous skin lesions; suspected thrombosis not yet proven	None
Other causes for thrombocytopenia not evident	No other cause for platelet count fall is evident	Possible other cause is evident	Definite other cause is present

Pretest probability score: 6–8 = high; 4–5 = intermediate; 0–3 = low. Reprinted from Warkentin and Heddle (2003) Laboratory diagnosis of immune heparin-induced thrombocytopenia. Current Hematology Reports, 2, 148–157. Copyright Current Medicine, used by permission.
*First day of immunising heparin exposure considered day 0; the day the platelet count begins to fall is considered the day of onset of thrombocytopenia (it generally takes 1–3 d more until an arbitrary threshold that defines thrombocytopenia is passed.

4.25 HIT and CRRT

Lepirudin is a direct irreversible Thrombin inhibitor binding both free and clot-bound Thrombin.
The experience of use of Lepirudin in patients with the combination of HIT(T), renal failure and haemodynamic instability therefore requiring CRRT is limited to a few case series and there is no consensus on how it should be administered. There are two alternatives bolus dose and continuous intravenous infusion. The risk of bleeding with CRRT and anticoagulation in the face of MODS is relatively high. Lepirudin is excreted via the kidneys, and therefore can accumulate in renal failure with a half life increasing from 1.5 hours in normal function to as much as 50hours. Clearance does occur via CVVH but efficiency of clearance varies according to membrane type Polysulphone membranes are more efficient than AN69 materials.

The monitoring of Lepirudin can be done effectively with APTT ratio.

Dialysis Line Locks in HIT

All line locks in patients with HIT should be made with 0.9% saline only.

Dosing Regime for Lepirudin in CRRT

Bolus dose at start of therapy of 0.1mg/kg then check APTT at 6 hours aiming APTT 2.0 - 3.0 and then 12hourly APTT with repeat dose of 0.1mg/kg when APTT <1.5.

Dose adjustment should be aimed at hitting the target APTT range of 2.0-3.0 post dose and any dose adjustment should be followed by a check APTT 6 hours later. Dose adjustment of 20% incrementally is suggested.

Preparation of lepirudin for use as anticoagulant in CRRT

- Using a 3ml luer lock syringe and blue needle draw up 1ml of sodium chloride 0.9% and add to the 50mg vial of lepirudin. Shake the vial gently
- This solution contains 50mg/ml of lepirudin
- **Further dilute to 10ml with sodium chloride 0.9% to make a final concentration of 50mg in 10ml (5mg per ml)**
- Calculate the volume of solution needed for the prescribed dose and withdraw the required volume from the vial
- Dilute to 2mls with sodium chloride 0.9% (if necessary) and administer as a bolus via the dialysis machine at the start of the session.

References:

Acute renal failure in Critically ill patients: A multinational multicentre study. Unchino,S et al. JAMA 2005; 294: 813-818

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Continuous Renal Replacement Therapy. NVIC Committee Nephrology and
Intensive Care.
JPJ Wester. Department of Intensive Care Medicine, Onze Lieve Vrouwe
Gasthuis, Amsterdam, The Netherlands

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S50

SECTION 5 SPECIAL REGIME (METABOLIC)

SECTION 5.1 Nutritional Support

Home

Malnutrition is common in ICU patients and is associated with increased morbidity and mortality. Consequences of malnutrition include impaired immune function, decreased ventilatory drive, respiratory muscle weakness, prolonged ventilator dependence, and increased infectious morbidity and mortality. Screening patients for nutritional status is recommended. All ventilated critically patients are at high risk and should be considered for nutrition support.

Benefits of nutritional support include improved wound healing, decreased catabolic response to injury, and reduction in complication rate and length of stay. Nutritional support is not without risk. Enteral nutrition may be associated with increased gastric residual volumes, bacterial colonisation of the stomach, and increased risk of pneumonia. Parenteral nutrition may be associated with gut mucosal atrophy, overfeeding, hyperglycaemia, and increased risk of infectious complications.

On admission ensure patients actual weight and height are measured, and ideal body weight and body mass index recorded. All patients not expected to be on full oral diet within 3 days should receive nutritional support. Enteral feeding should be initiated within the first 48 hours of ICU stay. Prior to initiation it should be established that the patient is haemodynamically stable, not requiring high volume fluid or blood product resuscitation. Haemodynamically stable ventilated patients with a functioning gastrointestinal tract should have gastric enteral feed prescribed and initiated within 24 hours of admission.

There should be a clearly identified goal for caloric requirement, and the aim should be to provide > 50% of requirement within the first week.

If it is anticipated that EN can be initiated within the first 7 days of hospital stay PN should not be used. In the decision not to use PN, this presumes a patient whom is normally nourished, and has had no `run in` time without nutrition prior to their admission. If the patient is malnourished, and use of EN is not feasible PN should be initiated as soon as the patient is adequately resuscitated. If it is anticipated that the duration of PN will be less than 5 days its prescription should be avoided.

If the patient is not established on 100% of requirement by day 7-10 use of supplemental PN should be considered to achieve goals.

All ventilated patients should receive the components of the ventilator care bundle to minimise the risks of aspiration. EN by the gastric route should not be withheld for gastric residual volumes less than 500 ml in the absence of other signs of feed intolerance (patient complains of pain or distension,

abdomen distended). On initiation of enteral feed, prescribe prokinetics if gastric residual volumes are greater than 200 ml 6 hourly. Erythromycin 200 mg 12 hourly intravenously is first choice. If there is a contraindication to its administration (e.g. administration of amiodarone, prolonged QT interval) prescribe metoclopramide 10 mg 8 hourly. If there is no response to prokinetics, or the patient is judged at high risk of aspiration a small bowel feeding tube should be placed.

In the majority of cases this can be accomplished simply at the bedside. If there is failure to site a jejunal tube by this method, endoscopic placement is required (refer to Lynne Timmis, clinical nurse specialist, gastroenterology page number 1948 or extension 3406).

All small bowel feeding tubes, irrespective of their route and method of placement require 30 ml water flush 8 hourly to prevent blockage.

Amount of Enteral Feed

The amount of feed should be titrated to patient weight. Care should be taken to avoid hyperalimentation particularly in the acute phase. Patients who are nutritionally replete at the onset of their critical illness should receive 20-25 kcal/kg/day (0.83 – 1.04 ml/kg/hour Osmolite), increasing to 25-30 kcal/kg/day (1.04 - 1.25 kcal/kg/hour Osmolite) during the recovery phase. The aim should be to provide 1.2 – 2 g protein/kg/day. Catabolic patients may require greater provision of protein.

Malnourished patients nutritional `target` should be 25 kcal/kg/day during the acute stage of their illness, increasing to 30 kcal/kg/day during the recovery phase. The weight taken to calculate `target` should be actual, not ideal body weight.

Obese patients (those with a BMI > 25) should receive the same nutritional target as those who are normally nourished but targets should be based on ideal, not actual, body weight. For obese patients those with a BMI 30 – 40 should receive 2g protein per kg IBW per day. For those with a BMI greater than 40 2.5 g protein per kg IBW should be provided.

Osmolite contains 1 kcal/ml, and 4 g/100 ml protein. Fibre supplementation (Jevity feed – identical caloric and protein content to Osmolite) may be of benefit if there is constipation. For patients requiring fluid restriction the options are Osmolite with 1.5 kcal/ml and 6.27 g/100ml protein, or Nepro with 2 kcal/ml.

Patients not established on nutrition support should receive dextrose infusion 100 g/day (e.g. 20 ml/hour 20% dextrose) until enteral feed tolerance of at least 30 ml/hour is confirmed or parenteral nutrition is commenced. Patients without central access will require an increased volume of 10% dextrose. In this circumstance, care should be taken to avoid hyponatraeamia.

Nutrition in ARDS/ALI:

Patients with ARDS and severe acute lung injury (ALI) should be placed on an enteral formulation characterized by an anti-inflammatory lipid profile (ie omega 3 fish oils, borage oil) and antioxidants.

Diarrhoea

An attempt should be made to establish a diagnosis and exclude infective causes. Prodiarrhoeal drugs should be discontinued. Having excluded infective cuases, treat with Loperamide (4 to 16 mg / day) unless contraindicated (e.g. obstruction). Intractable diarrhoea may require cessation of EN.

Monitoring
Glucose hourly initially reducing frequency if glycaemic control stable (see Section 5.2 Glycaemic control). Electrolytes daily with particular care over potassium, magnesium, and phosphate in those at risk of refeeding syndrome (see Section 5.3). Liver function tests initially daily.

ENTERAL NUTRITION ESSENTIALS:

<u>Admission</u>
Screen nutritional state
Record ideal body weight, actual body weight, and BMI
Assess risk of refeeding syndrome

<u>Within 48 hours</u>
Commence enteral nutrition within 24-48 hours unless contraindicated
Prescribe prokinetics if gastric residuals > 200 mls

<u>Within 1 week</u>
Aim to provide 100% of estimated requirements/day UNLESS HIGH RISK REFEEDING SYNDROME
Failure to achieve target enterally consider parenteral feed

Parenteral nutrition

The indication for and decision to prescribe TPN should be discussed with a consultant. Administration should be via a dedicated lumen of a central venous catheter. If prolonged administration is anticipated a PICC line or a tunnelled central venous catheter may be inserted.

Calculation of caloric requirements should be identical to that used for prescribing enteral nutrition. Great care should be taken to avoid overfeeding.

Protein intake should aim to provide 1.5 g/kg/day (1g N is equal to 6.25g protein).

Lipid can be given up to 1.5 g/kg/day and glucose up to 4 g/kg/day.

For electrolytes aim to provide 1-2 mmol/kg/day sodium. 1-2 mmol/kg/day potassium, 0.1 mmol/kg/day calcium, 0.1 mmol/kg/day magnesium and 0.5 mmol/kg/day phosphate.

When parenteral nutrition is prescribed it should be supplemented with glutamine 0.5 g/kg/day. Glutamine, whilst functionally non-essential in health, becomes essential in times of metabolic stress. Low plasma glutamine levels may be associated with immune dysfunction, increased risk of infection, and significantly increased hospital mortality. Benefits of glutamine administration to supplement TPN include decreased mortality, decreased infection rates, and improved glycaemic control.

Requests for parenteral nutrition should be made to pharmacy manufacturing before mid-day (extension 2289). Patients who are at risk of refeeding syndrome (see Section 5.3) should have a `phased` escalation of caloric intake, commencing at 50% requirements for the first 48 hours, 75% requirements for the 3rd day increasing to estimated requirement after this time.

```
PARENTERAL NUTRITION ESSENTIALS:

To reduce risk of infection:
        • Use of single central venous catheter or one lumen exclusively for
        purpose of TPN
        • Replace tubing at end of bag infusion or within 24 hours (whichever is
        shortest).
        • There should be no 3-way tap or ports to enable injections in line, i.e.
        the giving set should connect directly to the designated lumen.

To promote safe administration:
        • TPN must be administered via a volumetric pump
        • Bag should be light protected
        • The fluid should only be administered by personnel assessed able to
        give i.v. drug therapy.

Blood sugar monitoring:
        • Patients requiring continuous infusion of sliding scale insulin therapy
        should have their blood sugars recorded as per unit protocol
        • Critical care patients receiving TPN but not insulin therapy should have
        their blood sugars recorded 4 hourly.
```

Re-establishing oral intake

Patients with a tracheostomy and intact protective upper airway reflexes are able to eat and drink. After confirmation that these are present oral intake should be encouraged. Where there is concern that gag, swallow or cough are inadequate the patient should remain nil by mouth and consideration given to Speech and Language Therapy referral.

In patients able to manage some oral intake, enteral feed should be continued at target rate until the patient is able to take > 50% of target requirements.
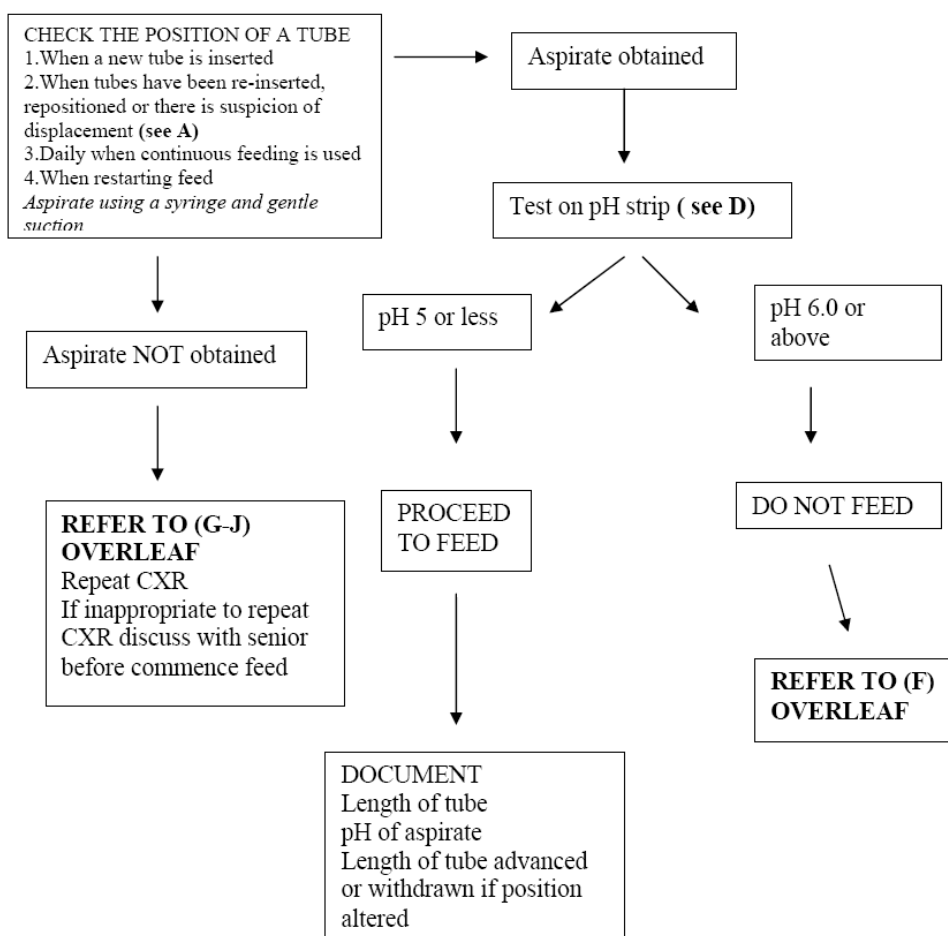
Dietetic referral

All complex patients should be referred for dietetic review and advice.

Nutritional content of commonly used enteral feeds/1000ml:

Feed	Energy (kcal/ml)	Protein g	Carb g	Fat g	Na+ mmol	K+ mmol	Ca^{2+} mmol	PO$_4$$^{3-}$ mmol	Mg^{2+} mmol
Osmolite	1010	40	136	34	38	36	17	22	8
Jevity	1050	40	148	35	40	40	23	23	9
Ensure Plus	1510	63	204	50	61	41	25	32	13
Nepro	2000	70	206	96	36	27	35	23	10

Confirming the correct position of NG feeding tubes in critically ill adults

CHECK THE POSITION OF A TUBE
1.When a new tube is inserted
2.When tubes have been re-inserted, repositioned or there is suspicion of displacement (see A)
3.Daily when continuous feeding is used
4.When restarting feed
Aspirate using a syringe and gentle suction

Aspirate obtained

Test on pH strip (see D)

Aspirate NOT obtained

pH 5 or less

pH 6.0 or above

REFER TO (G-J) OVERLEAF
Repeat CXR
If inappropriate to repeat CXR discuss with senior before commence feed

PROCEED TO FEED

DO NOT FEED

REFER TO (F) OVERLEAF

DOCUMENT
Length of tube
pH of aspirate
Length of tube advanced or withdrawn if position altered

The recommended procedure for checking the position of nasogastric feeding tubes in critically ill adults

Action	Rationale
A) Check for signs of tube displacement	Documenting the external length of the tube initially and checking external markings prior to feeding will help to determine if the tube has moved. The tube should be marked initially with permanent marker pen. A change in the marked tube position suggests that the tube may have migrated. Also check that mouth is free of coiled tube.
B) Obtain radiographic confirmation of initial tube placement	Mark and document the tubes length from the nose or mouth immediately after radiographic confirmation. Confirmation of the tube should be done by appropriately trained clinician and documented in the notes prior to commencement of feed.
C) Aspirate	Injecting air down the tube prior to aspiration should clear the tube of debris and or feed. Aspirates from small bowel feeding tubes are usually less than 10ml, an increase up to 50 ml or higher suggest the tube is in the stomach. CAUTION there have been reports of large volumes of aspirate from tubes that have been located in the lungs (Kauffman et al 2001).
D) Record pH levels	Apply aspirate to an area on the single, double or triple reagent panels of pH testing strips/paper. Allow ten seconds for any colour change to occur. Ensure you use colour chart on the box supplied. Record pH level.
E) Aspirate is pH 5 or below	Commence feed
F) Aspirate pH 6 or above	• Check the tube is the same length as documented on initial check x-ray • Medication may increase the pH level of gastric contents e.g. antacids, H2 antagonists or proton pump inhibitors eg pantoprazole. If the patient has only a one-off reading of a pH of 6 or is giving consistently high pH readings then consider why. • Dilution of the gastric acid by the enteral feed may cause pH to be 6 or above. Stopping the feed for up to 1 hour may allow time for the gastric pH to fall. This procedure should only be carried out with extreme caution in patients on insulin infusions for tight glycaemic control and only if the all of above methods have failed. CAUTION: Ensure the insulin infusion is turned off while the feed is stopped. If inconclusive consider options: consider replacement of tube or x-ray confirmation of placement. Do not feed before thorough risk assessment and team discussion, document decision and rationale.
G) Problems obtaining aspirate?	Use the largest size tubes (e.g. size 10 or 12) Have the tubes got multiple ports? If the tube only has one port then the side of the tube with the exit port can get lodged on the side of the gastric mucosa. Using a tube with multiple ports will limit this problem.
H) Turn adult onto their side	This will allow the tip of the nasogastric tube to enter the gastric fluid pool.
I) Inject 10-20ml of air using a syringe. This is NOT a testing procedure: DO NOT carry out auscultation of air('whoosh' test) to test tube position	Injecting air through the tube may dislodge the exit-port of the nasogastric feeding tube from the gastric mucosa. It is safe practice to use nasogastric tubes and enteral syringes that have non luer connectors (Building a Safer NHS for Patients: Improving Medication Safety published22/01/2004 available at www.dh.gov.uk)
J) Advancing the tube	Is the tube length long enough to reach the gastric body? A naso gastric tube will usually be around 60cm length. If the tube is in the oesophagus, advancing it 5 to 10cm may allow it to pass into the stomach. If the tube has been inserted too far, it may be in the duodenum (pH 7-8).. Document the length of tube if moved.

NUTRITION SUPPORT GUIDELINE:

```
┌─────────────────────────────┐      ┌──────┐      ┌──────────────────────────────────────┐
│ At ICU admission:           │ ──── │  NO  │ ───► │ Acceptable conditions:               │
│ Should this patient be fed? │      └──────┘      │   • Tolerating adequate oral diet    │
└─────────────────────────────┘                    │   • Less than 72 hours to full oral  │
              │                                     │     intake                           │
          ┌───────┐                                 │   • Palliative care                  │
          │  YES  │                                 └──────────────────────────────────────┘
          └───────┘
              │
┌─────────────────────────────┐      ┌──────┐      ┌──────────────────────────────────────┐
│ Can enteral nutrition be    │ ──── │  NO  │ ───► │ Acceptable conditions:               │
│ started within 24 hours?    │      └──────┘      │   • Enteric anastomosis (may still   │
└─────────────────────────────┘                    │     opt for enteral feeding)         │
              │                                     │   • Short bowel syndrome             │
          ┌───────┐                                 │   • High output small bowel fistula  │
          │  YES  │                                 │   • Imminent GI surgery              │
          └───────┘                                 │   • Imminent endoscopy               │
              │                                     │   • Bowel obstruction                │
┌─────────────────────────────┐                    │   • High output GI losses            │
│ Use full strength feed      │                    │   • Severe exacerbation              │
│ Consider Metoclopramide     │                    │     inflammatory bowel disease       │
│ 10 mg 8 hourly              │                    └──────────────────────────────────────┘
│ Goal: at least 80% of       │                                        │
│ requirements at 3 days.     │                                        │
│ Assess 6 hourly             │                                        │
└─────────────────────────────┘                                        │
              │                                                         │
        ┌────────────┐                                                  │
        │ At 24 hours│                                                  │
        └────────────┘                                                  │
              │                                                         │
┌─────────────────────────────┐                                        │
│ Aspirates > 200 ml 6        │                                        │
│ hourly?                     │                                        │
│ Add erythromycin 200 mg     │                                        │
│ 12 hourly                   │                                        │
└─────────────────────────────┘                                        │
              │                                                         │
        ┌────────────┐                                                  │
        │ At 48 hours│                                                  │
        └────────────┘                                                  │
              │                                                         │
┌─────────────────────────────┐                                        │
│ Aspirates > 200 ml 6        │                                        │
│ hourly?                     │                                        │
│ Insert nasojejunal tube and │                                        │
│ commence small bowel feed   │                                        │
└─────────────────────────────┘                                        ▼
              │                              ┌──────────────────────────────────────┐
        ┌────────────┐                       │ Begin TPN                            │
        │ At 72 hours│                       │ Reassess 12 hourly for EN eligibility│
        └────────────┘                       │                                      │
              │                              │                                      │
┌─────────────────────────────┐             │                                      │
│ Intolerant of small bowel   │ ──────────► │                                      │
│ feed?                       │             │                                      │
│                             │             │                                      │
│ Unable to meet 80% of       │             │                                      │
│ requirements by EN?         │             └──────────────────────────────────────┘
└─────────────────────────────┘
```

PHOSPHATE replacement in critical care

INDICATIONS
à Hypophosphatemia
• Reference range for inorganic phosphate is 0.7 – 1.34 mmol/L

MILD HYPOPHOSPHATEMIA (INORGANIC PHOSPHATE 0.6 – 0.7 mmol/L)

Oral / enteral administration
à Use Phosphate-sandoz® tablets. Each tablet contains:-
• Phosphate 16.1 mmol
• Sodium 20.4 mmol
• Potassium 3.1 mmol
à Prescribe 2 tablets orally / via nasogastric tube 8 hrly
à Tablets are effervescent and must be dissolved in water prior to administration

Parenteral administration

Only use if unable to take oral / enteral phosphate

à Use Addiphos® injection. Each 20 ml vial contains:-
• Phosphate 40 mmol
• Sodium 30 mmol
• Potassium 30 mmol
à **Peripheral line (use a dedicated line)**
• Prescribe Addiphos® 20 mmol (10 ml) in 500 ml sodium chloride 0.9% or glucose 5% by IV infusion at 62.5 ml/hr. Administer using an infusion pump
à **Central line (use a dedicated lumen)**
• Prescribe Addiphos® 20 mmol (10 ml) in 100 ml sodium chloride 0.9% or glucose 5% by IV infusion at 12.5 ml/hr **via central line.** Administer using an infusion pump

MODERATE OR SEVERE MILD HYPOPHOSPHATEMIA (INORGANIC PHOSPHATE < 0.6 mmol/L)

Parenteral administration
à Use Addiphos® injection. Each 20 ml vial contains:-
• Phosphate 40 mmol
• Sodium 30 mmol
• Potassium 30 mmol
à **Peripheral line (use a dedicated line)**
• Prescribe Addiphos® 40 mmol (20 ml) in 500 ml sodium chloride 0.9% or glucose 5% by IV infusion at 62.5 ml/hr. Administer using an infusion pump
à **Central line (use a dedicated lumen)**
• Prescribe Addiphos® 40 mmol (20 ml) in 100 ml sodium chloride 0.9% or glucose 5% by IV infusion at 12.5 ml/hr **via central line.** Administer using an infusion pump

MONITORING
à Check phosphate
• daily if taking oral phosphate
• after completion of infusion if parenteral phosphate has been given
à Check calcium and potassium daily

Key References:

Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Adult Critically Ill Patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.)
JPEN 2009 33 277-316

ESPEN Guidelines on Enteral Nutrition: Intensive Care
Kreymann KG, Berger MM et al
Clinical Nutrition 2006 25 210-223

Practical Management of Parenteral Nutrition in Critically Ill Patients
Standards and Guidelines
Intensive Care Society

Evidence-based guidelines for nutrition support of the critically ill: results of a bi-national guideline development conference.
Carlton (Australia): Australian and New Zealand Intensive Care Society 2005
Doig GS

www.guideline.gov
Nutrition Support for Adults
Oral Nutrition Support, Enteral Tube Feeding and Parenteral Nutrition
National Institute for Clinical Excellence 2006

Evaluation of a technique for blind placement of post-pyloric feeding tubes in intensive care:
application in patients with gastric ileus
Lee AJ, Eve R, Bennet MJ
Intensive Care Medicine 2006 32 4 553-556

SECTION 5.2 Glycaemic Control and Insulin Therapy

As well as its `classical` actions promoting anabolism and glucose utilisation it has been demonstrated that insulin has other less known effects which may be significant in critical illness including:

- Immunomodulation – e.g. decreasing production and reducing the effects of tumour necrosis factor
- Decreasing platelet aggregation
- Promoting vasodilatation
- Promoting preservation of mitochondrial ultrastructure and reducing malfunction of the electron transport chain

In addition hyperglycaemia is pro-inflammatory, impairs neutrophil function and decreases release of endothelial nitric oxide.

When to prescribe insulin:

Only prescribe insulin if:
• blood sugar is raised to greater than 10 mmol/l over 4 hours or
• patient is insulin dependant diabetic or none insulin dependant diabetic on oral hypoglycaemics

Aim to maintain blood sugar between 4.0 and 10 mmol/l. Use 50 units of insulin (Human Actrapid) to 50 ml with 0.9% sodium chloride. Suggested regime is:

Blood sugar (mmol/l)	Insulin infusion rate (units/hour)
0 – 6.0	Omit
6.1 – 8.0	1
8.1 – 10.0	2
> 10.0	4
> 10.0 for 2 hours	6

Blood sugar should be checked hourly for the first 4 hours, decreasing to 2 hourly for 8 hours, then 4 hourly if the blood sugar is between 4 and 10 mmol/l and there has been no change to the insulin regime or nutritional input. If there is a change to the rate of administration of insulin or feed blood sugar should be measured hourly until stable.

Hypokalaemia must be corrected prior to commencing insulin therapy. Insulin therapy may precipitate a refeeding syndrome. Monitor and correct hypokalaemia, hypomagnesaemia, and hypophosphataemia.

For patients who are receiving nutritional support (either enteral or parenteral) there is a risk of hypoglycaemia if the feed is interrupted. The insulin prescription should include the requirement to **STOP** the insulin infusion if there is interruption, and monitor blood sugar hourly until feed is recommenced. Insulin should only be administered if the blood sugar is greater than 10, at ½ the previous rate.

Insulin requirements vary between patients depending upon presence or absence of diabetes mellitus, body mass index, admission blood sugar, admission diagnosis, and caloric intake. Requirements also vary in a given patient over the course of their illness.

The insulin regime should be regularly reviewed and modified to ensure that the target is met. 10% dextrose 50 ml intravenous bolus `stat` if blood sugar less than 4 mmol/l should be prescribed with repeat blood glucose measured 20 mins later

Patients should not be discharged to a ward area with the preceding regime. Insulin should be discontinued prior to discharge where the patients' condition allows.

Key references:

Van den Berghe G et al NEJM 2001 345 1359-67

Van den Berghe G et al NEJM 2001 354 449-61

Nice-sugar investigators NEJM 2009 360 1283-97

Preiser JC et al ICM 2009 351 738-48

SECTION 5.3 Re-feeding Problems

Re-feeding problems encompass life-threatening acute micronutrient deficiencies, fluid and electrolyte imbalance and disturbances of organ function and metabolic regulation resulting from over-rapid or unbalanced nutrition support. The 2 most widely recognised complications of re-feeding are the re-feeding syndrome and Wernicke-Korsakoff Syndrome.

Re-feeding syndrome:

Up to 1/3 of ICU patients fasted for greater than 48 hours may develop the re-feeding syndrome. A range of life-threatening clinical and biochemical problems may occur:

Cardiac failure, pulmonary oedema, dysrhythmias
Acute fluid overload or depletion
Hypokalaemia
Hypophosphataemia
Hypomagnesaemia
Hypocalcaemia (uncommon)
Hyperglycaemia

Patients at risk:

> Little food intake over 5 days

Patients at high risk:

Any one of the following;
> BMI < 16
> Unintentional weight loss >15% over < 6/12
> Little or no nutrient intake for > 10 days
> Hypokalaemia, hypophosphataemia, or hypomagnesaemia prior to feeding

Or any 2 of the following:
> BMI < 18.5
> Unintentional weight loss > 10% over < 6/12
> Little or no nutrient intake for > 5 days
> Alcohol abuse, chemotherapy, insulin, diuretics

For patients at high risk:
Aim should be to provide 50% of requirements for first 2 days, 75% requirements for 3rd day, and full requirement from the 4th day. Ensure appropriate biochemical monitoring. Prescribe oral thiamine 300 mg once daily, vitamin B either intravenously (Pabrinex iv high potency) or enteral Vit. B co strong 2 tablets three times daily, and a balanced multivitamin/trace element supplement for first 10 days.

Extreme cases e.g. BMI < 14 should have dietetic review.

Wernicke-Korsakoff syndrome:

Caused by acute thiamine deficiency upon re-feeding due to increased metabolic demands.
The syndrome comprises one or more of the following:
Apathy and disorientation
Nystagmus, opthalmoplegia, other eye movement disorders
Ataxia
Severe impairment of short term memory often with confabulation

For patients at high risk (alcoholics or chronic vomiting) prescribe Pabrinex IV high potency 1 and 2, 2 pairs three times daily for 9 doses then enteral Vitamin B co strong as above.

Key References:

Kraft MD Nutr Clin Pract 2005 20 6 625-33
Marik PE Arch Surg 1996 131 10 1043-7
Nutrition Support for Adults
Oral Nutrition Support, Enteral Tube Feeding and Parenteral Nutrition
National Institute for Clinical Excellence 2006 80-

Section 5.4 Acid – Base Analysis

Introduction

pH is vitally important as most metabolic processes are directly or indirectly affected by changes in pH.
Severe disorders of acid-base balance lead to organ dysfunction, especially when these derangements develop quickly.
Neurological - Cerebral oedema, seizures and coma.
Cardiovascular - Variable - altered sympathetic tone with arrhythmias, increases myocardial oxygen demand, reduces myocardial contractility, pulmonary vasoconstriction and systemic vasodilatation or constriction.
Respiratory - increases RR , reduces minute ventilation , respiratory muscle fatigue , respiratory failure or diversion of blood flow from vital organs to the respiratory muscles.
Immune system - Emerging evidence suggests that changes in acid-base variables influence immune effector cell function.

It is vitally important to understand that it is the underlying disorder rather than just the value of the pH that is important. For example, a patient with DKA with a pH of 6.8 will get better with fluid resuscitation and insulin, whereas someone with septic shock, and the same pH, will probably die.
It may also be beneficial to treat the acidosis directly but this should be of secondary importance.

Knowing how to interpret an ABG means being able to know the magnitude, the cause and what to do about an acid-base disturbance.

Magnitude

pH

The $\log_{10}$ of the reciprocal of the hydrogen ion activity' .
Thus it is simply a measure of the hydrogen ion concentration.

pH is logarithmic so it is therefore difficult to appreciate the magnitude of the change in H^+. A fall in pH from 7.4 to 7.1 represents a doubling of H^+ from 40 to 80 nmol/l, whereas a rise in pH from 7.4 to 7.7 represents a fall in H^+ by only 20 nmol/l, from 40 nmol/l to 20nmol/l.

The normal pH of plasma is 7.35-7.45
<7.35 = acidaemia
>7.45 = alkalaemia

The terms acidosis and alkalosis describe the process leading to acidaemia or alkalaemia. Thus they can be present with a pH that is in the normal range due to respiratory or metabolic compensation.

It is interesting to note that the pH of pure water (which is of course acid-base neutral) at 37 degrees is 6.8 which is close to the pH of intracellular fluid.

Actual and standard bicarbonate

These can be ignored as Base Excess is of more use.

Base Excess

It is independent of CO2 and therefore allows us to quantify the magnitude of the metabolic component.
It is defined as the amount of acid or base that must be added to a sample of whole blood in vitro to restore the pH of the sample to 7.40 while the PCO2 is held at 5.33 kPa at a given Hb concentration.
The BE of oxygenated blood with a Hb of 15 at a pH of 7.4 and PCO2 of 5.33 is zero.
BE is influenced by Hb concentration so accuracy is increased by using a Hb value of 5 which estimates the average content of Hb across the entire extracellular fluid space. This is called the standard BE (SBE) or BE(ecf).
BE does not tell us about the mechanisms of a metabolic acid–base disturbance.

Cause

Disturbances in acid-base balance are caused by 3 independent variables – CO2, Strong Ion Difference (SID) and the total concentration of weak acid (Atot).

CO2

CO2 is an independent determinant of plasma pH. Its level is normally maintained at 5.3kPa by a balance of cellular metabolic production and alveolar ventilation.
An increase in CO2 leads to an increase in the concentration of hydrogen ions (pH falls) while a decrease will reduce the hydrogen ion concentration (pH rises)

$$CO_2 + H_2O \longrightarrow H_2CO_3 \longrightarrow H + HCO_3$$

Intracellular, as well as extracellular, acidosis will result as CO2 freely diffuses through cell membranes.
Thus a raised CO2 causes a respiratory acidosis while a low CO2 causes a respiratory alkalosis. These may or may not result in acidaemia or alkalaemia depending on metabolic compensation.

SID (Strong Ion Difference)

The human body is 60% water which provides an inexhaustible source of hydrogen ions.
Strong ions are ions that completely dissociate in water.
In plasma strong cations outnumber strong anions – the difference between them is termed SID

$$SID = (Na + K + Ca + Mg) - (Cl + lactate)$$

It is mathematically provable that the SID causes water to dissociate or associate, thus determining the hydrogen ion concentration.
A reduced SID causes an acidosis; an increased SID causes an alkalosis.
The normal SID in plasma is 42mEq/L.
Because the concentrations of the ions other than Na and Cl is so small and varies little, the SID is basically the difference between Na and Cl.

$$SID = Na - Cl$$

The normal difference between these 2 ions is 38mEq/L
As these ions and the BE are measured in mEq/L it makes it easy to calculate the effect on the BE of any change in SID.

Increasing SID by 1 will increase the BE by 1.
Decreasing the SID by 1 will decrease the BE by 1.
It can therefore be seen that an acidosis will be caused by a reduced gap between Na and Cl concentration.
This can be caused by:
- Water overload (eg TURP syndrome). Diluting plasma will reduce SID.
- Failure of chloride excretion – renal failure.
- Administration of chloride in excess of sodium – giving 0.9% saline which has 154mmol/l of Na and Cl will proportionally increase Cl more than Na.
- Diarrhoea and pancreatic drainage (large bowel and pancreatic fluid has a high SID so more cations than anions will be lost).

Alkalosis will be caused by an increased Na-Cl gap.
- Vomiting with loss of chloride.
- Administration of Na without Cl eg sodium bicarbonate.

It is of note that the kidney and liver will compensate for changes in CO2 and Atot to keep pH in the normal range by altering SID (primarily through chloride balance). This will of course be impaired in renal and hepatic failure.

Atotal

It can again be proved mathematically that the total concentration of weak acids (**A**total) determines pH.
In plasma **A**total is predominantly albumin.
Hypoalbuminaemia has an alkalinizing effect while hyperalbuminaemia would cause an acidosis.
As critically ill patients all have an albumin of half normal (20) or less, this will disguise a significant presence of strong anions (ie they would be more acidotic than they actually are if they had a normal albumin).
It is doubtful that the body compensates for a metabolic acidosis by albumin loss. Hypoalbuminaemia in the critically ill is the result of the switch of hepatic albumin production to the production of acute phase proteins, poor nutrition and the loss of albumin through leaky endothelium. It is therefore most likely just a lucky coincidence that hypoalbuminaemia is protective against an acidosis.
You will see that hypoalbuminaemic patients on ICU with a BE in the normal range have compensated by altering their chloride levels (and thus SID).

Bedside calculations

The cause of an acidosis can quickly be determined by the bedside.

The value of the BE can be accounted for by 3 simple calculations which take into account the above independent variables:

Sodium - chloride effect on BE = Na - Cl - 38 (normal Na-Cl gap is 38)
Albumin effect on BE = 0.25 x (42 - albumin)
Unmeasured ion effect on BE = BE - (Na - Cl effect) - (albumin effect)

(Remember to use SBE (BEecf) if alternatives are displayed on your ABG)

So you now know what effect the Na-Cl difference is having on the BE, what effect albumin is causing (albumin in ICU patients is usually around 20 which equates to a +ve BE of around 6) and what effect other ions are causing.
Possible unmeasured strong anions will be:

Ketones (hydroxybutyrate and acetate)
Lactate
Renal failure and hepatic failure anions (sulphate, phosphate, urate etc)
Methanol (formate)
Ethylene glycol (oxalate)
Aspirin (salicylate)

Lactate is of course measured on many ABG machines so can be factored into your calculation.

You are now a long way towards knowing the true cause. Methanol, ethylene glycol and aspirin poisoning are rare and will be suggested by the history. Ketones and lactate can be easily measured which leaves renal failure as the alternative cause (which you will know from the creatinine, urine output etc)
Eg
pH 7.2
BE -12

Na – Cl (135 - 105 – 38) = -8
0.25 x (42 - 20) = 5.5
–12 - (-8) – 5.5 = -9.5

Here you can see that the Na-Cl effect on the BE is almost as great as the unmeasured ion effect (which in this example could come from acute renal failure). You can also see this patient would be more acidotic if not for the low albumin.

It can be seen from the above information that if each time you look at an ABG you look at the pH, BE, Na, Cl, albumin and lactate and do the above calculation you will know the cause and magnitude of any acid base disturbance.

What to do about it

Treat the underlying cause first.

Only use 0.9% saline if there is a hypochloraemic alkalosis. 0.9% saline has a SID of zero and will increase Cl proportionally more than Na causing an acidosis.

Use Hartmanns as your fluid of choice. This has an SID of 29 (assuming metabolism of the lactate which occurs very quickly). Although this SID is lower than normal for plasma it will cause haemodilution and a fall in albumin concentration thus having a neutral effect on acid-base balance.
Remember that Hartmanns will *decrease* plasma potassium if the plasma value is greater than 5 and that only a negligible amount of the lactate will be metabolised to glucose. This means it is safe (indeed desirable) to use it in renal failure and DKA.
If there is a high lactate it depends on the cause whether Hartmanns should be used. If the high lactate is caused by hepatic failure then it should probably be avoided (although only 1mmol of its 29mmol would need to be metabolised for it to have less of an acidifying effect than 0.9% saline). If it is due to hypoperfusion then Hartmanns will treat the cause and the lactate will fall.

Sodium Bicarbonate will reverse an acidosis by increasing the sodium concentration (the bicarb is irrelevant)
This will permanently treat an acidosis cased by a narrowed Na-Cl gap (the kidneys will do this themselves if working normally)

In an acidosis caused by other strong anions than chloride it should be seen as a temporising measure only to allow institution of renal replacement therapy.
The hypothesis that sodium bicarbonate worsens intracellular acidosis by generating extra CO_2 is overstated. Its administration over 30-60mins will easily permit the removal of any additional CO_2 load.
Eg
A BE of -10 can be corrected by increasing the SID of the ECF by 10 (accomplished by increasing the ECF Na by 10)
BE -10
ECF volume 15L
10mmol Na required for each litre of ECF
So 150mmol $NaHCO_3$ will bring the BE to zero. This is theoretical and in practice it often requires more.
Give half the calculated dose of 8.4% sodium bicarb via a central line over 30-60mins and then reassess the patient.

Anion gap

The other less accurate, but traditional, way of working out the cause of an acid-base disturbance is to calculate the anion gap.
There is in reality no 'gap' - as electrical neutrality has to exist in any solution:

measured anions + unmeasured anions = measured cations + unmeasured cations

Routinely measured anions are Cl^- and HCO_3^-, and routinely measured cations are Na^+ and K^+.

$$AG = ([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-])$$

More recently, the reference range for AG has shifted downwards to *3–11* mmol litre^{-1} due to the improved accuracy of chloride measurement.

The unmeasured anions in a normal sample are mostly accounted for by albumin so the 'gap' is grossly underestimated in hypoalbuminaemia.
It is therefore essential to correct for albumin.
Assuming a normal albumin concentration of 40 g litre^{-1}:

$$AG \text{ (albumin corrected)} = AG + (0.25 \times [40 - albumin])$$

You can work out that for every 10g/L of albumin rise or drop, you can correspondingly adjust the AG by about 3 mmol/l.

Changes in the concentrations of measured ions will leave the AG unaffected as the effect of changes in Na and Cl will be countered by changes in HCO_3. Therefore a metabolic acidosis with a normal anion gap will be caused by a narrowed Na-Cl gap.
Only unmeasured ions will alter it. The unmeasured anions that will cause an increased anion gap are those strong anions listed above.

Section 6   Neurocritical Care

Section 6.1 Management of traumatic brain injury

6.11 Initial management and resuscitation

Epidemiology:

About 120.000 hospital admissions / year with head injuries in England + Wales:

- 70 % male patients

- in up to 65 % of adult head injuries alcohol involved

- about 10 % of brain injury patients have an additional spine trauma (mainly cervical spine)

Basics:

Initial GCS (before sedation) is important for prognosis and therapeutical decisions (e.g. ICP monitoring)

Trauma classification

Initial GCS	Classification
3 - 8	severe brain injury
9 - 12	moderate head injury
13 - 15	mild head injury

GCS should always be documented as sum of its three components: E, V, M

Main therapeutical target is the strict avoidance of secondary brain damage (= cerebral tissue hypoxia) by avoiding [1,7]

1. Hypotension (systolic BP < 90 mmHg)
2. Hypoxemia (arterial pO2 < 8,0 kPa)

In patients with severe brain injury (GCS 3 – 8): [2]
- prolonged periods of hypotension increase mortality from 27% to 60 %
- prolonged periods of hypoxemia increase mortality from 27% to 33 %
- the combination of both increases mortality from 27% to 75 %

To avoid these incidents:

- a sufficient fluid resuscitation (e.g. CVP and/or central venous saturation guided)
- use of inotropic/ vasopressor support (mainly noradrenaline)
- early intubation (rapid sequence induction) under in-line immobilisation and
- controlled ventilation

are important factors of success.

Arterial line and CVC should be inserted early for close blood pressure monitoring and, if required, application of vasopressors , mannitol and CVP measuring.

The decision to insert an intracranial pressure probe is made by the neurosurgical team. According to the Brain Trauma Foundation the usual indications are:

1. GCS $\leq$ 8 and pathological CT head or
2. normal CT and two or more of the following features:
   - patients age > 40 years
   - Systolic blood pressure < 90 mmHg
   - Uni- or bilateral motor posturing

Targets of therapy:

For the acute phase of brain trauma management aim for [1,2,3,4,7] :

- mean arterial pressure > **80 mmHg** (measured on ear level)
- arterial sO2 $\geq$ **95 %** or arterial pO2 $\geq$ **13 kPa** (= 100mmHg)
- ventilation adjusted to arterial pCO2 of **4.5 kPa**
- intracranial pressure $\leq$ **20 – 25 mmHg**
- cerebral perfusion pressure (CPP= MAP – ICP) **60 mmHg**. No artificial attempts to achieve CPP > 70 mmHg because of increased risk of ARDS
- strict avoidance of hyperthermia (> 36,5°C) à cooling and paracetamol possible benefits and harm of hypothermia are currently still under investigation
- normoglycaemia: **6.0 – 10.0** mmol/l

6.12   Management of raised ICP

ICP treatment to be initiated if ICP > 25 mmHg for > 5 min

C

second tier options (d/w neurosurgical team)
• Thiopentone coma:
   bolus : 1,5 – 3 mg/kg, start infusion only if ICP responds
   infusion: 0.5 – 4 mg/kg/h
• decompressive craniectomy
• EVD or lumbar drain insertion
• shortterm hyperventilation to pCO_2 < 4.0 kPa
• paralysis (infusion only if ICP responds to bolus dose)

yes

ICP > 25 mmHg ?

no → go back to A

B

• if EVD in situ: drain a few ml of CSF to control ICP
• otherwise initiate shortterm moderate hyperventilation: target pCO_2 4.0 kPa
• if serum osmolarity < 320 mosm/l : Mannitol 20% 3 ml / kg over 15 min i.v

yes

ICP > 25 mmHg ?

no

A

continously maintain / restore:
• MAP, CPP, pO_2 , pCO_2 in target range
• appropriate sedation
• normothermia (cooling, if required)
• head 30° up and in in line position

Section 6.13   Seizures treatment and Seizure prophylaxis

- Phenytoin and Carbamazepine can be used for **prophylaxis** of early posttraumatic seizures within the first 7 days post trauma. After 7 days a prophylactic treatment is not recommended.

- This does not affect the **therapeutic use** of these drugs according to anti-epileptic treatment guidelines if seizures are evident.

- If patients show prolonged wake up times nonconvulsive seizures must be excluded by EEG.

Section 6.14   Diabetes insipidus

- Cranial DI is caused by post-traumatic failure of ADH secretion.

- Symptoms:

  Polyuria (> 3000 ml / 24h) **plus**
  Hypernatraemia
  Urine osmolarity : < 300 mOsmol/l,   rises > 50 % after Desmopressin (DDAVP)
  Serum osmolarity: > 300 mOsmol/l

- Management:

  If diuresis < 4000 ml /24 h : only fluid replacement

  If diuresis > 4000 ml /24 h : fluid replacement + DDAVP 1 mcg i.v.

  Correct hypovolaemia with Normal Saline (0,9%), then use Glucose 5 % for correction of hypernatraemia.

  Plasma sodium lowering not faster than 0.7 mmol/L/h (10 mmol/L /24hrs).

Diabetes insipidus needs to be managed with caution to avoid overcorrection of sodium and / or problems with the patients fluid balance. As Diabetes insipidus in the context of brain injury usually reflects a critical situation discuss situation with Consultant.

Section 6.15   Hyponatraemic syndromes after head injuries

Section 6.151   Syndrome of Inappropriate ADH Secretion (SIADH)

- Symptoms:

    - hyponatraemia
    - persistent increased renal sodium excretion: Urine sodium level > 25mmol/l
    - serum osmolality < 280 mOsmol/l
    - urine osmolality > serum osmolality
    - **normo- or hypervolaemia** without peripheral oedemas

- Therapy:

    - Fluid restriction (1000 ml isotonic fluid/24 h)

    - if fluid restriction alone fails or hyponatraemia < 115 mmol/l:
        - à add infusion of **NaCl 3 % (1.2 ml/kg/h)** + frusemide
           aim for **slow serum sodium correction (1 mmol/h), max. 12 mmol/l within first 24 h** to avoid central pontine myelinolysis.

Section 6. 152   Cerebral Salt Wasting Syndrome (CSW)

- Symptoms:

    - hyponatraemia
    - **hypovolaemia**
    - excessive natriuresis with urine sodium levels > 50 mmol/l

- Therapy:

    - Fluid replacement with normal saline 0,9 % (CVP or CV sO2 guided)
    - if necessary infusion of NaCl 3 % for slow serum sodium correction (see SIADH)

Section 6.16 Further issues of neurotrauma care:

1. **CNS**:
   - § parameters to maintain see above, if ICP is normal for > 24 h without interventions stop sedation in consens with neurosurgical team (unless non – neurological reasons for sedation exist).

   - § For endotracheal suctioning, difficult ventilator adjustment or acute intervention for raised ICP **single boluses of relaxants** might become necessary. **Relaxant infusions should be avoided** because they have shown to induce outcome relevant complications (higher pneumonia rates, longer duration of ventilator support, longer ICU stay, higher rates of critical illness polyneuropathy) and make it difficult to assess the quality of sedation.

2. **CVS**:
   - § parameters to maintain see above,

3. **Respiratory system**:
   - § parameters to maintain see above, PEEP levels up to 10 cm H_2O do usually not affect the ICP and can therefore be used even in brain injured patients [6].

4. **GIT / Metabolism**:
   - § Gastroparesis and ileus are common problems after severe head injuries. Therefore prokinetics should be started early. If no contraindications from the GIT are present enteral feeding (+ prokinetics) should be started within 24 h, beginning with small volumes (30 ml/h).

   - § Strict avoidance of hyperglycaemia by i.v. insulin infusion (see nutrition guidelines).

   - § Stress ulcer prophylaxis should be given at least until full enteral nutrition is established.

5. **Coagulation**
   - § If clotting and platelets are in normal range and no evidence of ongoing intra- or extracranial haemorrhage exists, prophylactic heparinisation should be considered 24 h post trauma unless otherwise directed by neurosurgical team.

6. **Nursing**:

§ Patients to be nursed in 30° head up position. Prone position can be used under ICP monitoring.

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[1] Management and Prognosis of Severe Traumatic Brain Injury. The Brain Trauma Foundation (2000) (www.braintauma.org)

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[3] EBIC- guidelines for management of severe head injuries in adults. European Brain Injury Consortium. Acta Neurochir (Wien) 1997; 139(4):286-94

[4] Update Notice Management of Severe Traumatic Brain Injury: Cerebral Perfusion Pressure, The Brain Trauma Foundation, Approved by the AANS on March 14 , 2003

[5] Alderson P, Gadkary C, Signorini DF : Therapeutic hypothermia for head injury. The Cochrane Database of systematic reviews 2006 Issue 4

[6] Mc Gurie G, Crossley D, Richards J et al.: Effects of varying levels of positive end expiratory pressure on intracranial pressure and cerebral perfusion pressure. Crit Care Med 25 (1997) 1059 – 1062

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Section 6.2   Subarachnoid haemorrhage

Epidemiology:

- incidence around 6 / 100.000 per year

- causes: 85% aneurysm rupture, 10% non-aneurysmal perimesencephalic bleed (good prognosis), 5% other rare conditions (Angioma, AVM etc)

- most patients < 60 years, women > men

- prognosis: case fatality rate about 50 %, severe handicapped survivors about 15 %, survivors who remain independent 35 %

Assessment of severity : The World Federation of Neurosurgery (WFNS) Scale:

WFNS grade	GCS	motor deficit
I	15	-
II	14 - 13	-
III	14 - 13	+
IV	12 - 7	+ / -
V	6 - 3	+ / -

Basics of diagnosisc:

- CCT   will detect SAH with 98 % sensitivity. This is often followed by CT angiography or DSA to identify the vascular lesion.

- if clinical suspect of SAH exists but CCT is negative (~ 2%) LP should be performed 12 h after onset of symptoms (not earlier to allow red cell lysis to take place and increase of bilirubin- and oxyhaemoglobin concentration in CSF). Otherwise traumatic spinal tap and SAH can not be discriminated.

General measures in SAH patients

CNS: - consider intubation / ventilation in WFNS grade II - IV patients, grade V
      will always require intubation

    - if patient is not intubated provide sufficient analgesia (paracetamol 1g/6hr +/-
     opioids)

    - ICP monitoring, if patient is on sedation/ventilation (CPP 80 – 90 mmHg).

    - continuous observation (GCS, temperature, ECG, pupils, focal deficits) to detect
    complications as early as possible

CVS: - do not treat hypertension unless MAP $\geq$ 130 mmHg or systolic. BP > 180
    mmHg
    (whatever occurs first) or if clinical or laboratory evidence of progressive end
    organ failure (e.g. left ventricular failure)

    - regular monitoring of arterial blood pressure, CVP, urine output

    - high endogenous stress level after SAH affects cardiac function: ischaemic
    ECG

    - changes are found in 50 – 75 % , arrhythmias in up to 80% and left
    ventricular wall dysfunction in 27 – 33%

    - maintain normovolaemia or mild hypervolaemia

Respirat.:   - maintain: sO2 $\geq$ 95 % or arterial pO2 $\geq$ 13 kPa (= 100mmHg)
    - ventilation adjusted to arterial pCO2 of 4.5 kPa

GIT: - oral feeding, if cough and swallowing reflexes are working properly,
    otherwise nasogastric tube, start NG feeding within 24 hrs, beginning with
    low volumes (20 ml/hr) + prokinetics

    - maintain normoglycaemia (6.0 – 10.0 mmol/l)

    - keep stool soft (adequate fluid intake, laxatives)

Temperature:- maintain normothermia, if necessary with paracetamol and cooling,
    no hypothermia

The SAH patient is mainly threatened by three possible events:

- rebleed

- hydrocephalus

- delayed cerebral ischemia / vasospasm

6.21 Rebleed

- without intervention rebleed will occur in:

15 % within the first few hours (sudden deterioration of consciousness, apnoea)
40% within the first 4 weeks

à poor prognosis: 80 % of patients die or remain disabled

- main strategy for reducing the risk of rebleed is early intervention (coiling or clipping) within the first 3 days.

6.22 Hydrocephalus

- caused either by occlusion (blood in ventricular system) or decreased resorption of CSF secondary to blood in subarachnoid space in ~ 70 % of all SAH patients

- typical presentation: initially alert patient who drops GCS over a few hours or (if patient is unconscious the whole time or on analgosedation) downward deviation of eyes, small unreactive pupils (dilatation of aqueduct).

- Diagnosis: repeat CT scan

- Treatment: external ventricular drainage or lumbar drain as directed by neurosurgeon

- management of CSF diversion see 6.23

6.23 Care and management of the patient with an External Ventricular Drain (EVD) (for nursing staff and doctors)

(written by SN Becky Amos)

This guidance has been produced in order to assist the bedside nurse with observations, to identify potential complications and courses of action when caring for a patient with an EVD. Should more information be required about anatomy & physiology, please ask.

An EVD is a neurosurgical specific piece of equipment that is used for the temporary drainage of cerebrospinal fluid (CSF) from the lateral ventricles situated deep within the brain (Littlejohns & Trimble 2005). This piece of equipment should be thought of as **important** as a ventilator or haemofiltration.

ONLY A NEUROSURGEON SHOULD TAKE SAMPLES OF CSF, ADMINISTER INTRATHECAL ANTIBIOTICS AND REMOVE AN EVD.

Nursing staff are to assist with the collection of equipment to enable procedures to be carried out and to ensure infection control principles are adhered to throughout.

Please ensure that the following things are completed during your bed space check when caring for a patient with an EVD. If unsure, please speak to the nurse in charge of the shift:

1. **Check the height/level at which the EVD has been set and prescribed within the medical notes. Please record on the MIU chart, daily and record any changes to that level on the chart as they occur.**

Rationale: the level at which the drain is set will determine CSF drainage; the EVD will drain to maintain the level set which corresponds to the patient intracranial pressure (ICP). Its is also to ensure shift to shift consistency and to identify any changes that may have not been communicated by the neurosurgical team to the nurse caring for the patient, if not present at the bedside when changes are made.

2. **Check that the EVD is easily visible and is attached to a separate IV pole on the bed itself, away from any other infusions.**

Rationale: to ensure the EVD is easily identifiable, drainage can be observed regularly and is not confused with infusion devices.

3. **Check the level at which the EVD has been placed on the IV pole. This 'zero' point determines the amount of CSF drained and should be in line with mid-brain; the external auditory meatus is the anatomical reference point with foramen of Monro. If unsure, please ask the nurse in charge. This level <u>MUST</u> be checked using a spirit level (located on the MIU) or using a laser which may be attached to one of the Codman EVD sets, against the 3-way tap towards the bottom of the drip chamber. <u>DO NOT RELY ON YOUR ARM.</u>**

(CODMAN)
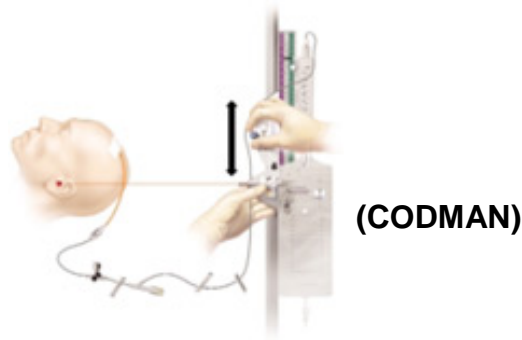
Rationale: if the EVD is too high above the 'zero' point (foramen of Monro), drainage of CSF will reduce, potentially causing complications associated with a rise in intracranial pressure (ICP) due to enlargement of the ventricles (Woodward, 2002). If the EVD is too far below the 'zero' point, a siphoning effect may be evident. This may result in over-drainage of CSF which may lead to collapse of the ventricles and tearing of small blood vessels leading to re-bleeding (Oman & Levine, 2009). Excessive over or under-drainage of CSF may both lead to **herniation** of brain tissue. This level must also be checked following each patient turn.

Lumbar drains use the same system CSF drainage system as normal EVD's, however, the 'zero' point in this case may need to be discussed with the neurosurgeon. According to the rep for the new EVD systems however, the level must be set at the site in which the catheter is inserted in the case of lumbar drains.

4. **Check that CSF if oscillating/pulsating within the EVD tubing. This indicates the patency of the drain itself.**

Rationale: if oscillation cannot be seen, check the clamps on the EVD itself to make sure the ones that should be open, are. Check to see if the tubing is kinked or contains debris and check if the drainage bag is full.

The EVD can be removed from the IV pole and temporarily lowered to see if CSF can still drain. Following this, the EVD must immediately be replaced into the IV pole and level checked against the patient and results of this procedure reported to medical staff.

Observe the insertion site and check that the drain has not come out; if the drain is insitu, medical staff must be informed as the EVD may be blocked; this is a neurosurgical emergency. This scenario must be taken into account alongside the patients Glasgow Coma Score (GCS) and pupil reactions. If the drain has come out, place a sterile dressing over the insertion site and contact the neurosurgical team immediately. If the drain has become disconnected, clamp the catheter using non-serrated clamps and place sterile gauze over the end of the catheter tubing. Lay the patient on their back, in the supine position, and contact the neurosurgical team immediately.

On an hourly basis, please ensure that the following things are carried out:

1. **Observe the amount of CSF drainage on an hourly basis. Ensure this is drained hourly from the chamber and documented on the fluid balance section of the MIU chart.**

Rationale: If the drip chamber is allowed to fill, CSF drainage may be inhibited due to potential blockage of the air filter at the top. Notify medical staff if drainage is >25 - 30mls/hr or if you have no output. CSF drainage of >50mls/hr is seen as excessive but may be seen as acceptable in cases of severe hydrocephalus. Ensure that the drain is checked for the oscillation of CSF following the emptying of CSF from the drip chamber.

2. **Observe the colour and consistency of CSF within the drip chamber of the EVD. Please ensure this is documented and recorded hourly on the fluid balance section of the MIU chart.**

Rationale: Normal CSF should appear translucent/clear and straw coloured. In patients with an EVD, CSF may appear different:

- o Cloudy (turbid) CSF may indicate infection such as meningitis
- o Faint yellow, orange or pink (xanthochromia) CSF indicates red blood cell lysis (breakdown)
- o Brownish CSF may indicate the presence of old blood or hyperbilirubinemia
- o Blood stained (Rose) is usually as a result of surgery or recent haemorrhage
- o If sudden blood is seen this may indicate a further bleed and a neurosurgeon should be contacted immediately
- o Normal CSF will have the consistency of water. CSF that is more viscous may indicate meningitis or some forms of cancer

3. **Check that CSF if oscillating/pulsating within the EVD tubing. This indicates the patency of the drain itself.**

Rationale: please see above rationale within the bed space check.

4. **Check the entry site of the EVD.**

Rationale: this is to check the patency of the drain alongside checking the oscillation of CSF within the EVD tubing. If there has been no drainage of CSF into the burette, you will be able to see if the drain has been dislodged, if the site is leaking CSF or if there are any signs of infection at the entry point. If there are any signs of infection (redness, swelling, visible pus) or CSF leakage, please alert the medical staff and neurosurgical team.

5. **Please ensure that you record your patient's GCS and pupil reaction on an hourly basis.**

Rationale: complications of EVD usage can be identified early, such as, blockage of the drain, over/under-drainage of CSF. By doing this, patient

condition is monitored closely and continuously. Alert the medical team of any alterations to your patients' level of consciousness.

EVD safety

1. EVD's should not be lay flat unless the drip chamber has been emptied and the drain clamped. This is to prevent the filters within the EVD becoming blocked, affecting the efficiency of drainage and creating the risk of introducing infection when it means that the EVD drainage system needs to be changed.

2. It has been recommended that non-serrated clamps, gauze and a chlorhexidine steret (2% according to infection control guidance) be kept at close hand, in case of accidental disconnection of the drain from the catheter (Great Ormond St, 2009). If this happens, clamp the catheter immediately and nurse the patient in a supine position. Steret the end of the catheter and wrap in sterile gauze until the patient can be seen by the neurosurgeons (Leeds RI, 2001) (this may be a neurosurgical emergency, therefore notify the nurse in charge immediately, the doctor caring for the patient and the neurosurgical team).

TO CLAMP OR NOT TO CLAMP

3. Clamping the EVD may result in inadequate drainage of CSF. EVD's should not be clamped for more than 30 minutes at a time, due to the risk of a rise in ICP and deterioration in the patients neurological condition, caused by the build up of CSF within the ventricles. Your

patients clinical condition should be considered when deciding whether to clamp the EVD. EVD's should only be clamped following discussion with medics:

- When ICP measurements are required
- When CSF samples are being collected
- During the administration of intrathecal antibiotics (clamped for 60 minutes following administration)
- During turning – clamps must immediately be released and level checked once repositioning has finished
- During transfer e.g. from one bed to another, in CT/MRI scan (ensure the drain is transferred 'ON' if possible, in an upright position and attached to an IV pole. The drain must be observed throughout the transfer to prevent accidental disconnection.
- During extensive, vigorous episodes of chest physiotherapy - coughing/suctioning may cause over drainage of CSF and therefore ventricular collapse.

The drain must be unclamped and re-zeroed after each episode of clamping.

Please ensure that you adhere to all infection control guidelines when caring for a patient with an EVD by ensuring that a closed system is maintained at all times, that Personal Protective Equipment (PPE) and alcohol hand gel is used at all times, that dressings are observed regularly and changed aseptically only when soiled or loose, that the drainage bag is changed aseptically when no more than ¾ full and that the neurosurgical team is contacted if there is any sign of infection, CSF leakage from the insertion site or deterioration in your patients' GCS.

The above points are to be used as a tool to guide and support your care of a patient with an External Ventricular Drain. This will support continuous and thorough assessment of your patient, ensuring that any deterioration in your

patient's condition is determined and acted upon early. If unsure about any of the points raised above, please speak with the nurse in charge of the shift, your PDN team or Senior Staff Nurse Becky Amos. Further education will be provided on this topic; keep your eye on the education programme.

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6.24 Vasospasm

- Vasospasm is one important reason for onset of delayed cerebral ischemia and neurological deficits.
  - 40 % - 60 % of SAH patients develop vasospasms, mainly those with an initial loss of consciousness and higher amounts of subarachnoid blood.

  - 1/3 of vasospasms remain asymptomatic

  - 2/3 of vasospasms go along with a Delayed Ischemic Neurological Deficit (DIND)

  - Onset of vasospasm after 48 – 72 h, peak frequency from 5 to 14 days after SAH, but vasospasms can continue up to 20 days after SAH.

Therapeutic strategy:

- **Nimodipine 60 mg orally/NGT every 4 h for 3 weeks**, if this is not possible (high aspirates, ileus) consider i.v. application via central venous line:

  1 mg/hr for 2 hours, if blood pressure remains stable increase to 2 mg/hr, change to oral medication as soon as possible.

- keep magnesium levels in normal range

- post intervention : **triple H therapy**: (hypertension, hypervolaemia, haemodilution)

  1. **h**ypertension: systolic arterial blood pressure 160 – 180 mmHg, MAD 110 mmHg, CPP 80 – 90 mmHg (noradrenalin + fluid)
  2. **h**ypervolaemia: CVP 12 cm H2O   (> 3 L/day normal saline)
  3. **h**aemodilution: (Hct 30 %) (see hypervolaemia)

- There is no clear evidence for better neurological results / survival due to triple H therapy up to now but it is still an accepted strategy. Therefore this concept should be handled very cautiously to avoid negative side effects (e.g. left ventricular failure due to hypervolaemia in patient with chronic cardiac failure).

6.24   Hyponatraemia after SAH

see section 6.15

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3. Rinkel GJE, Feigin VL, Algra A, van Gijn J. Circulatory volume expansion therapy for
 aneurismal subarachnoid haemorrhage.Cochrane Database of Systematic Reviews 2007 Issue

4. Bederson JB et al. Guidelines for the Management of Aneurysmal Subarachnoid Haemorrhage.
    http://stroke.ahajournals.org, Jan 22, 2009

Section 6.3 Care of the post cardiac arrest patient

Post-resuscitation syndrome

Post-resuscitation syndrome following return of spontaneous circulation after cardiac arrest is a common reason for patients to be admitted to the intensive care unit. The pathophysiological changes occurring as part of the syndrome are complex and involve multiple body systems.

Syndrome	Pathophysiology	Clinical Manifestation
Post–cardiac arrest brain injury	• Impaired cerebrovascular autoregulation • Cerebral edema (limited) • Postischemic neurodegeneration	• Coma • Seizures • Myoclonus • Cognitive dysfunction • Persistent vegetative state • Secondary Parkinsonism • Cortical stroke • Spinal stroke • Brain death
Post–cardiac arrest myocardial dysfunction	• Global hypokinesis (myocardial stunning) • ACS	• Reduced cardiac output • Hypotension • Dysrhythmias • Cardiovascular collapse
Systemic ischemia/reperfusion response	• Systemic inflammatory response syndrome • Impaired vasoregulation • Increased coagulation • Adrenal suppression • Impaired tissue oxygen delivery and utilization • Impaired resistance to infection	• Ongoing tissue hypoxia/ischemia • Hypotension • Cardiovascular collapse • Pyrexia (fever) • Hyperglycemia • Multiorgan failure • Infection
Persistent precipitating pathology	• Cardiovascular disease (AMI/ACS, cardiomyopathy) • Pulmonary disease (COPD, asthma) • CNS disease (CVA) • Thromboembolic disease (PE) • Toxicological (overdose, poisoning) • Infection (sepsis, pneumonia) • Hypovolemia (hemorrhage, dehydration)	• Specific to cause but complicated by concomitant PCAS

Patients will only be admitted if deemed appropriate by a consultant intensivist. The decision for supportive care will have been made at this time and if deemed appropriate hypothermia should be induced as soon following admission as is practicable, if the target core temperature is exceeded.

Haemodynamic management

Patients with ST elevation myocardial infarction require angiography with a view to percutaneous coronary intervention +/- IABP insertion for myocardial revascularisation. Other patients with ECG evidence of ischaemia after VF arrest should be discussed with cardiology to ascertain possible need for PCI.

There is often conflict between the requirements to maintain an adequate mean arterial pressure for cerebral perfusion with the need to minimise exposure of the ischaemic myocardium to catecholamines. A MAP of 65 mmHg is adequate. A lower mean pressure may be tolerated if cardiac function is poor and vital organ perfusion is maintained. Adequacy of cardiac output should be assessed clinically, guided by urine output and serial lactate measurements. Central venous haemoglobin saturation measurement may be useful. Fluid resuscitation is often required to correct hypovolaemia. Isotonic crystalloid should be used. Advanced haemodynamic monitoring may be appropriate in complex cases – discuss with consultant.

Management of dysrythmias is by correction of electrolyte abnormalities, and pharmacological or electrical treatment as appropriate.

Oxygenation

Hyperoxia, particularly in the early post resuscitation phase, should be avoided. Aim to maintain SpO2 between 93 and 96% (PaO2 8-10 KPa).

Ventilation

Hypocarbia should be avoided. Maintain PaCO2 5.0 - 5.5 KPa.

Hypothermia

Induced hypothermia should be avoided in patients with cardiogenic shock (hypotension with poor peripheral perfusion despite the IABP +/- catecholamine infusion). If in doubt discuss with consultant.

Cooling is achieved through the `Arctic Sun` device. Aim for core temperature of 33 degrees C (range 32-34) as soon as is practicable. There may still be benefit to cooling induced up to 24 hours after ROSC. Hypothermia may be induced by administration of ice cool isotonic crystalloid 2 litres over 1 hour. Consider administration of magnesium sulphate 5g over 5 hours as prophylaxis for shivering and assisting induction of hypothermia through vasodilatation.

Hypothermia should be maintained for 24 hours. Hypothermia induces a diuresis. Hypovolaemia should be avoided. Monitor for and correct hypokalaemia, hypomagnesaemia, hypocalcaemia, and hypophosphataemia. Treat hyperglycaemia as per unit guidelines. Monitor carefully for and treat shivering. Deep sedation is appropriate. Administration of neuromuscular blocking agents may be required.

Great care must be taken during rewarming to avoid too rapid warming. A rate of rise of temperature of 0.25 degrees C per hour is the target. Care should be taken on rewarming to avoid `overshoot` and resultant pyrexia.

Seizures and myoclonus

Seizures should be treated conventionally. Myoclonus should be treated with propofol boluses, phenytoin 15 mg/kg then further boluses of 5 mg/kg up to a maximum of 30 mg/kg. Failure to respond to phenytoin is common. Sodium valproate is 2nd line. Clonazepam may be required if there is no response to valproate.

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Madl C, Holzer M. Brain function after resuscitation from cardiac arrest. Curr opinion critical care 2004; 10: 213- 217.

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Bernard SA, Gray TW, Buist MD et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. NEJM 2002; 346: 557-63.

Section 7   Trauma

(See Section 2.14 for Guidelines for Permissive Hypotension in Trauma)

Trauma Lecture Notes
Final FRCOA Course, Royal College of Anaesthetists

General principles

Characteristics of trauma
Anatomical disruption with physiological consequences
Complexity: concurrent, consecutive, concealed injuries
Frequency: blunt *versus* penetrating, serious injuries *versus* severe trauma; age, sex, evening/weekend
Level of intent: rarely completely accidental
Outcome: co-morbidity; morbidity; mortality (immediate, early or late, not tri-modal)

Trends in trauma care
Operative/non-operative control of bleeding: Pringle 1908
Permissive hypotension: Cannon 1918
If the pressure is raised before the surgeon is ready to check any bleeding that may take place, blood that is sorely needed may be lost. Cannon WB, *et al*. Preventative treatment of wound shock. *JAMA* 1918;**70**:618.
Advanced Trauma Life Support: Steiner 1978
Early complete definitive care
Damage control: Rotondo/Schwab 1993
Rotondo MF, Schwab CW, *et al*. Damage control: an approach for improved survival in exsanguination penetrating abdominal injury. *Journal of Trauma* 1993;**35**:375-383
Maintaining blood pressure: *sine qua non* of head injury management: Chesnut 1997
Chesnut RM. Avoidance of hypotension: conditio sine qua non of successful severe head-injury management. *Journal of Trauma* 1997;**42**:Suppl.4-9
Diagnostic and interventional radiology

Generic model of trauma care
Control of anatomical structures

- Decompress, drain, decontaminate, debride, dress, damage control
- Reduce, restrain, repair, remove, replace, rehabilitate

And control of physiological systems

- Respiration, circulation, nervous system, metabolism, host defence

Along a care pathway
Locations with attendants in multi-disciplinary teams that assess and intervene

- Scene → Primary transfer → Primary emergency department → Imaging suite → Operating theatre → Critical care unit → Acute ward → Rehabilitation unit
- If secondary transfer is indicated, the pathway includes Secondary transfer → Secondary emergency department, etc.

Involving three early in-hospital phases
Assessment (including adjuncts) and intervention

- Primary assessment (or survey) and intervention – identify and treat immediately life-threatening conditions – resuscitate (ABCDE)

- Secondary assessment (or survey) and intervention – identify and treat potentially life-threatening, potentially incapacitating, and limb-threatening conditions – start structural control (D's and R's)
- Tertiary assessment and intervention – missed injuries and physiological system failure – continue system control (ABCDEFGHI[6])

Primary Phase

Primary survey (look, feel, listen, percuss and elicit verbal/pain response), adjuncts to the primary survey (monitoring, imaging and blood samples) and resuscitation

- Airway with cervical spine control
- Breathing with ventilatory support
- Circulation with haemorrhage control (and position control in late pregnancy)
- Disability with prevention of secondary insult
- Exposure with temperature control

Conditions not to be missed in the Primary Assessment:

- Airway obstruction
- Tension pneumothorax
- Open pneumothorax
- Decompensating flail chest
- Massive haemothorax
- Exsanguinating haemorrhage
- Cardiac tamponade
- Neurogenic shock
- Decompensating head injury

Secondary Phase

Secondary survey (head to toe, front and back; surface, orifice, cavity, extremity), adjuncts to the secondary survey (imaging, signal recording, diagnostic sampling of blood/body fluids, and specialist referral), and definitive care (anatomical structure control)

- Head & neck (head, face, eyes, neck soft tissues)
- Trunk (chest, abdomen, pelvic contents, retro-peritoneum)
- Spine (cervical, thoracic, lumbar)
- Limbs (upper extremity, shoulder girdle, lower extremity, pelvic girdle)

Conditions not to be missed in the secondary assessment (e.g. chest):

- Disruption: tracheo-bronchial, diaphragmatic, aortic, oesophageal
- Contusion: pulmonary, myocardial

Tertiary Phase

Tertiary survey and continuing care

- Clinical re-examination
- Imaging review
- Critical care

Conditions not to be missed in the Tertiary Assessment:

- Missed injuries (concealed, delayed, simply missed through inexperience or distraction by other priorities)
- Secondary organ damage

From the Scene to the Operating Theatre

Scene

Flying squad – control of inaccessible airway, analgesia/anaesthesia for extrication, giving permission to fire service to extricate rapidly in time-critical situations, operative interventions extremely rare
Scene behaviour – appropriate clothing, personal safety, liaison with Emergency Services, cautious intervention (the scene is not a hospital!)
Problems – limited access, environmental hazards, limited resources, limited time (swoop and scoop *versus* stay and play *versus* swoop and scoop and play on the way)
Extrication – immobilise vehicle; immobilise spine, evaluate clinically and judge urgency; oxygenate, cannulate, relieve pain, reassure, give judicious fluids; dismantle vehicle at appropriate speed; coordinate rapid side extrication by spinal board, package for transfer

Emergency Room

Preparation
Ambulance alert. Team call-out→Team assembly→Role assignment→Briefing
Equipment check and drug preparation
Emergency Room Drugs
Fentanyl (25-50 mcg increments) or morphine (much slower onset)
Induce with ketamine (±fentanyl), thiopentone (±fentanyl) or propofol (+fentanyl). Etomidate now less favoured, but still used by many.

Annane D. ICU physicians should abandon the use of etomidate! *Intensive Care Medicine* 2005;**31**:325-326

Morris C, McAllister C. Etomidate for emergency anaesthesia; mad, bad and dangerous to know? *Anaesthesia* 2005;**60**:737

Suxamethonium (or rocuronium in major crush injury, etc.)
Propofol infusion, midazolam increments/infusion, atracurium (or cis-atracurium) afterwards
Epinephrine (or metaraminol) to counteract sedation-related hypotension in severe head injury
Immediate Care
Oxygen with reservoir mask and bag-valve-mask to hand
Handover and history from pre-hospital personnel
Continue or instigate spinal precautions if indicated or if history uncertain
Verbal contact → airway manoeuvre if unresponsive
Continue primary assessment and intervention, including (further) vascular access and drawing blood
Order head CT, chest and pelvic radiographs now (if indicated) to avoid delay later
Start (anterior) secondary assessment with early analgesia when primary assessment complete and resuscitation underway
Log-roll after head and neck assessment and complete (posterior) secondary assessment
Arterial line when convenient after immediate resuscitation
Urinary catheter (after rectal/perineal examination) and gastric tube (after considering basal skull fracture)

Spinal imaging before or as part of CT scan

Spinal Precautions

Manual in-line immobilisation (at outset or when performing airway manoeuvre)

Hard collar, firm surface, head blocks and straps

Firm surface with temporary use of spine board

Log-rolling to examine or re-position

Return to manual in-line immobilisation for any airway intervention

The Thrasher

Hard collar, if feasible, but avoid restraining the head (neck acts as fulcrum)

Assume/exclude hypoxia/hypotension

If failing, rapid sequence induction, then wake (if appropriate) after secondary assessment and investigations complete

What If the Patient Vomits?

Tip trolley head-down and use suction

Don't turn on spinal board unless fully strapped in!

Don't suddenly log-roll on side unless at least 5 people already in place!

Rapid Sequence Induction

Rapid primary survey with adjuncts

Manual in-line immobilisation (from above)

Collar removed

Selected induction agents in judged dose (reduce if altered consciousness or shock)

Relaxant

Cricoid pressure

Oral intubation with bougie/stylette loaded or available

Secure tube and re-package C-spine

Immediate vascular access

- Peripheral (2 cannulae, 1 fluid infusion unless in the face of extreme hypovolaemia or in the midst of operative, resuscitative haemorrhage control)
- Central – rarely needed immediately (femoral if no major abdominal, pelvic or ipsilateral femoral injury; internal jugular if no neck injury; subclavian if no coagulopathy and no ipsilateral upper rib or clavicular fractures). For subclavian access, choose the side of lower rib fractures with or without pre-existing chest drain to spare the normal side; choose the other side from high rib or clavicular fractures to avoid distorted vessels and the fracture haematoma.
- Cut-down (long saphenous at ankle or groin)
- Intra-osseous (tibia or sternum)

External compression of bleeding sites
Immediate management of major blood loss

This is perhaps the biggest change in the management of the major trauma patient in the last decade. Vigorous fluid therapy championed by Advanced Trauma Life Support has given way to a more cautious approach. NICE recommends that no fluid is administered pre-hospital if there is a palpable radial pulse (or central pulse in penetrating trunk trauma). **Permissive hypotension**, better referred to as **deliberate temporary under-resuscitation**, is appropriate in the face of uncontrolled bleeding, though definitive evidence is still elusive. The likely sites of bleeding are easily defined and immediate surgery or, increasingly, angiographic embolisation may be needed to stem the bleeding. The latter option requires a critical care environment to be established in the relative isolation of the angiography suite and a surgical backup plan. In open book pelvic fractures, temporary pelvic splinting combined with internal

rotation of the femurs assists may help to limit the bleeding, depending on the mechanism of injury and fracture pattern.

Dutton RP. Fluid management for trauma; where are we now? *Continuing Education in Anaesthesia, Critical Care & Pain* 2006;**6**:144-147

Emergency Imaging
Adjuncts to the Primary Survey
- Chest and pelvic x-rays
- Ultrasound of the trunk

Adjuncts to the Secondary Survey
- Other x-rays
- CT of the head and cervical spine
- Other CT scans
- Other imaging (angiography, other contrast studies, echocardiography, MRI)

What to look for on Chest X-ray
Adequacy (extent, rotation – medial ends of clavicles in relation to spinous processes, penetration – vertebrae seen through heart)
Airway (including tubes)
Breathing
- Lungs (including pleural space) – haemo-pneumothorax, contusion, aspiration
- Ribs
- Diaphragm
Circulation (mediastinum and vascular lines)
Surrounding structures (interstitial emphysema, clavicles, scapulae, spine, stomach)

What to look for on head CT
Air in skull
Blood: haematoma (extradural, subdural, intra-cerebral), intra-ventricular or subarachnoid blood
Contusion including petechiae
Displacement: midline shift, ventricular compression, basal cistern patency
Edema: loss of grey/white differentiation, hypo-density
Fracture (on bone windows) including fluid in sinuses

Plain views of the C-spine – adjunct to secondary assessment: cross-table lateral with gentle arm traction, antero-posterior, trans-oral peg, obliques or swimmer's

What to look for on C-spine X-ray
Adequacy (extent, rotation, penetration)
Alignment (anterior/posterior longitudinal ligaments, spino-laminar line)
Bones
Connective tissue spaces: pre-vertebral (vertebral body depth below larynx), pre-dental (3 mm in adults, 5 mm in children), disk spaces, inter-spinous)

CT scan of C-spine
Although it provides imperfect visualisation of injury to ligaments and disks, CT scanning has an increasing role in spinal clearance. Indiscriminate use involves unnecessary thyroid irradiation. Patients with multiple injuries and cardio-respiratory instability should undergo CT scanning of the cervical spine and of the entire trunk,

with sagittal and coronal reconstructions of the cervical, thoracic and lumbar spine. Patients with isolated severe head injury should have the cervical spine imaged with sagittal and coronal reconstructions. Patients with a less severe head injury should have the upper cervical spine scanned (from the occiput to C3) at the same time as the head CT scan, sparing the thyroid if possible.

Clearing the spine in the obtunded trauma patient

Clinical history and examination are not reliable. Standard imaging (plain views with targeted CT) can miss isolated ligamentous injury. Alternative imaging (e.g. flexion-extension on image intensifier) is controversial and unproven. Prolonged spinal immobilisation has complications and limitations. The cervical spine of a patient who continues to be obtunded or sedated after obtaining standard imaging can be 'fully' cleared by one of four methods: **clinical assessment when the patient is eventually awake** (with modified spinal precautions in the interim); **performing an MRI scan**; **performing flexion-extension views** under image intensifier control; accepting a normal **CT of the cervical spine coupled with a Trust risk assessment declaration**. Each strategy has advantages and risks, though the last option is currently gaining favour. **Cervical spinal clearance algorithm** (British Trauma Society) – see appendix.

Clearing the abdomen

Clinical assessment is unreliable if the conscious level is altered or if there are painful distracting or adjacent injuries. Diagnostic peritoneal lavage is less sensitive and specific than ultrasound and is no longer favoured. Ultrasound is more appropriate than CT for immediate use in unstable patients, but remains operator dependent. CT is more sensitive and specific, but has a radiation risk and takes place in a more difficult environment.

Getting mobile

Propofol ± alfentanil infusion
Fentanyl (or morphine), atracurium or cis-atracurium, ± midazolam increments
Portable monitor with invasive BP/NIBP, capnography and charger
Portable ventilator with PEEP, alarms and extra length of hose to reach wall outlet in CT
Repeated blood gas analysis

Operating Theatre

In transition – brief theatre staff, maintain impetus, plan operative sequence. In serious situations, consider simultaneous procedures, blood salvage or damage control.
During the operation: In **extreme urgency**, stay 'mobile' until under control. In **isolated head injury**, use neuroanaesthetic technique. Note that propofol or servoflurane requirements may be much lower if patient was obtunded pre-sedation and this may change after decompression. Do not over-anaesthetise and cause unnecessary and harmful hypotension. Avoid switching from fixed rate propofol infusion in the ER to TCI in theatre. In **combined head and other injuries**, use core neuroanaesthetic technique (e.g. air, sevoflurane, opioid. If post-operative ventilation is planned or high analgesic requirements are anticipated (for other injuries), longer-acting opioids may be used instead of remifentanil. Consider ICP measurement during extra-cranial surgery. Keep looking at the pupils if feasible. Don't trust end-tidal CO_2

as an estimate of arterial pCO_2. In **major abdominal, chest or head injuries**, avoid nitrous oxide. Anticipate lower anaesthetic requirements in accordance with shock or altered consciousness. In **chest injuries** – think pneumothorax. Consider alternative theatre ventilators and PEEP in severe pulmonary contusion.

In **unstable patients**, check blood gases (with lactate, haemoglobin, potassium, calcium) at least hourly and clotting at least every few hours. The gravity of the situation should be matched by the training and experience of the staff and by the complexity of surgery undertaken. Keep the patient warm and consider damage control if losing physiological system control.

Damage Control
Original definition: *Capacity of a ship to absorb damage and maintain mission integrity*
Sum total of the manoeuvres necessary to ensure patient survival above all else. Rarely needed in British trauma practice but understanding essential when indicated. Mainly refers to general surgical problems in the abdomen but increasingly recognised as a technique for other specialties in severe cases. Damage control surgery is **deliberately abbreviated emergency surgery** (usually to the abdomen) to achieve haemostasis and prevent uncontrolled spillage of intestinal contents or urine.

Rotondo MF, Schwab CW, *et al*. Damage control: an approach for improved survival in exsanguination penetrating abdominal injury. *Journal of Trauma* 1993;**35**:375-383

Shapiro MB, et al. Damage control: collective review. *Journal of Trauma* 2000;**49**:969-978

Packs may be left in situ and the wound may be left open. Physiological control in the Intensive Care Unit then assumes priority over achieving definitive closure and anatomical repair, which can be achieved at re-operation, typically after 48 hours. Similar concepts have been developed in orthopaedic surgery, though their implementation is still limited in the UK.

Damage Control – Phase I
Abbreviated resuscitative surgery
Control of haemorrhage (manual compression, resuscitative packing, clamping, etc)
Exploration
Control of contamination
Definitive intra-abdominal packing if necessary
Rapid (temporary) abdominal closure if necessary

Resuscitative packing: initial short-term measure (minutes) to control or minimise blood loss while dealing with higher priority injuries or catching up
Therapeutic packing: provides tamponade if coagulopathy present or if injury surgically unmanageable; allows longer resuscitation time to correct the metabolic derangements and access other means of definitive vascular control e.g. angiography

Damage Control – Phase II (ICU)
On-going re-warming
Correction of coagulopathy
Fluid resuscitation
Optimisation of haemodynamic status
Re-examination/further investigation to diagnose all injuries

Damage Control – Phase III

Re-exploration

Definitive management of injuries

Abdominal closure if previously left open

Re-operation

Typically at 48 hours, but earlier if evidence of on-going haemorrhage, un-correctable acidosis, or evolving abdominal compartment syndrome

Damage Control Orthopaedics

Not yet widely practised in UK, except for immediate external fixation of open-book pelvis.

Temporary external fixation of femur with intra-medullary nailing later when patient stable.

Avoid major definitive long bone procedures and other challenging interventions between days 2 and 4 (interleukin-6 and the second hit).

Scalea TM, et al. J Trauma 2000;48:613-21

Pape HC, et al. J Trauma 2002;53:452-462

Harwood PJ, *et al*. Alterations in the systemic inflammatory response after early total care and damage control procedures for femoral shaft fracture in severely injured patients. *Journal of Trauma* 2005;**58**:446-454

Hildebrand F, et al. Damage control: extremities. *Injury* 2004;**35**:678-689

Specific Conditions

Primary Survey

Airway Compromise in trauma

Obstruction, contamination, disruption, distortion

Airway obstruction

Intra-luminal (tongue, foreign body, teeth, blood, vomit), intra-mural (larynx), extramural (sublingual, C-spine, vascular)

Treat with suction, jaw manoeuvres, foreign body removal, pharyngeal/tracheal tubes, cricothyroidotomy (needle *versus* surgical) as back-up

Alternative Intubation Methods

Blind nasal (no longer favoured), flexible fibre-optic, rigid fibre-optic (Bullard, Wu), tracheostomy under local anaesthetic, cricothyroidotomy (needle, surgical)

Manual Jet Ventilation Via Needle Cricothyroidotomy

Transcricoid cannula (13G adults, 18G neonates), oxygen supply 4-10 bar, typical pressure regulator (3 bar adults, 0.5 bar neonates)

Tension pneumothorax

Relieve immediately, without x-ray confirmation:

Needle thoracocentesis (2nd intercostal space in the mid-clavicular line), followed by chest drain, or

Scalpel/clip thoracostomy (5th intercostal space, just anterior to mid-axillary line, using rapid access technique) with/without immediate chest drain

Open pneumothorax

Cover temporarily with:

Asherman chest seal, or

Occlusive dressing, taped on three sides, or

Occlusive dressing with separate chest drain

Call thoracic surgeon and prepare for thoracotomy

Flail Chest

2 or more ribs in 2 or more places. May be concealed by pain (splinting) or positive pressure ventilation and may not be evident on chest x-ray

Provide analgesia, according to the site, extent and severity of the pain: Titrated intravenous opioids, initially by incremental boluses, and then by infusion/PCA Inter-costal, para-vertebral or intra-pleural blocks Epidural analgesia, provided that spinal clearance has been achieved and hypovolaemia and coagulopathy can be excluded Non-steroidal anti-inflammatory drugs, provided that hypovoalemia and coagulopathy can be excluded
Search for associated pulmonary contusion, pneumothorax or haemothorax
Treat with added oxygen and if necessary by ventilatory support according to gas exchange and respiratory rate/tidal volume: Face mask CPAP or BIPAP mask ventilation Mechanical ventilation via an endotracheal tube In severe cases, ventilatory support may need to be continued for more than 2 weeks
Insert a chest drain if there is any evidence of a pneumothorax or of a significant haemothorax, or as prophylaxis if the patient is ventilated and the chest is inaccessible for a significant period (e.g. helicopter retrieval or prolonged surgery)
Provide intensive physiotherapy
When a subclavian or internal jugular central venous line is required in patient with a unilateral chest injury, use the ipsilateral side for lower rib fractures and the contralateral side for high rib fractures or clavicular fracture (to avoid fracture haematomas and distorted vessels)

Massive haemothorax

Treat by simultaneous chest drain insertion and blood volume replacement, without waiting for a chest x-ray. Judge volume replacement in context of uncontrolled bleeding.
In an adult, insert a large bore chest drain using blunt dissection. Place the drain in the fifth intercostal space, just anterior to the mid-axillary line, and direct it superiorly and posteriorly towards the apex of the lung.
In general, if 1500 mL is obtained immediately or more than 200 mL per hour for 2-4 hours, involve a cardiothoracic surgeon immediately and consider emergency exploratory thoracotomy
Consider auto-transfusion if available

Cardiac tamponade

Suspect cardiac tamponade and decompress the pericardium in pulseless electrical activity, unless responding immediately to volume replacement or thoracocentesis. Consider this diagnosis in poorly responding shock.
Decompress the pericardium immediately in the acutely compromised patient: Subxiphoid needle pericardiocentesis (catheter-over-needle or Seldinger technique), using ECG monitoring to detect injury pattern changes on penetrating the myocardium (but recognise limitations of this technique: "clot at both ends"), Surgical pericardial window, using the sub-xiphoid or inter-costal technique, or Immediate thoracotomy and pericardiotomy
Summon a cardiothoracic surgeon immediately, or if one is not available locally, a general surgeon capable of performing an emergency thoracotomy
Prepare for immediate thoracotomy in the Emergency Room or emergency thoracotomy/median sternotomy in the Operating Theatre, depending on the patient's condition

Traumatic cardiac arrest

Blunt *versus* penetrating – blunt has bleak outlook, penetrating better if arrest witnessed.

Immediate thoracotomy: incision (5th space ± trans-sternal extension) à interventions (pericardial decompression, haemorrhage control, open cardiac massage, aortic compression). Patient may wake up and need immediate anaesthesia if manoeuvres successful!

Shock

Fluid-restricted resuscitation/delayed fluids/permissive hypotension *versus* classic ATLS fluid resuscitation (20 mL.kg^{-1} boluses, class I-IV haemorrhage and responder status) in haemorrhagic shock. Problems of vigorous fluid infusion/blood transfusion – clot dislodgement, further bleeding; dilutional coagulopathy; hypothermia; immune reaction or suppression

Animal models of hypotensive resuscitation

Improves short-term survival at expense of tissue perfusion

Moderate hypotension better than severe

Moderate hypotension better than normotension

Mortality, haemodynamics and haemorrhage same for similar sodium loads of normal and hypertonic saline

Stern SA. Low-volume fluid resuscitation for presumed hemorrhagic shock: helpful or harmful? Current *Opinion in Critical Care* 2001;**7**:422-30

Stern S, *et al*. Under-resuscitation of near-lethal uncontrolled hemorrhage: effects on mortality and end-organ function at 72 hours. *Shock* 2001;**15**:16-23

Stern S, *et al*. *Shock* 2001;**15**:16-23. *J Neurosurgery* 2000;**93**:305-14

Stern S, *et al*. Effect of initially limited resuscitation in a combined model of fluid-percussion brain injury and severe uncontrolled hemorrhagic shock. *Journal of Neurosurgery* 2000;**93**:305-14

Human studies on hypotensive resuscitation

Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries

Bickell WH, *et al*. *New England Journal of Medicine* 1994;**331**:1105-1109

Hypotensive Resuscitation: Prospective, randomised study of 110 patients in haemorrhagic shock – Target SBP 100 *versus* 70 mmHg; measured 114 *versus* 100 mmHg; only 4 deaths in each group, so inconclusive!

Dutton RP, Mackenzie CF, Scalea TM. *J Trauma* 2002;**52**:1141-1146

Delayed fluid resuscitation and permissive hypotension are well-accepted in penetrating trunk trauma. Many regard it to be appropriate in blunt cases too, though this is more controversial and further evidence is awaited. This treatment strategy demands prompt surgical haemostasis.

Alternative causes of shock

Blood on the outside
- Obvious and visible?
- At the scene?
- In clothes?
- Underneath the patient, on the trolley or bed, or in drains?

Blood in a cavity
- In the chest? Examine the chest and get a chest x-ray
- In the abdomen? Examine the abdomen (unreliable) and get a portable ultrasound or CT (if the patient is fit for transfer to the Imaging Suite)

- In the pelvis or retroperitoneum? Reconsider external fixation, angiography and embolisation for pelvic injuries. Think bladder and kidney (especially if haematuria present but even if none).

Blood in both thighs (bilateral femoral fractures)

Not hypovolaemic or not bleeding at all
- Pericardial tamponade
- Tension pneumothorax
- Spinal cord injury
- Brain stem compromise
- Septic shock (not likely unless delayed presentation)
- Over-sedation
- Allergy to administered drugs or fluids

Transfusion and blood products

Pre-emptive administration of clotting factors and platelets are appropriate in massive haemorrhage. Much interest has been shown in the use of **activated factor VII** to control non-surgical bleeding, though the evidence has not yet been convincing enough to justify a product license in trauma. It acts at the site of tissue damage and requires the presence of other clotting factors and platelets. It is much less effective in a cold, acidotic patient. **Prothrombin complex concentrate** is an overlooked product that is much more effective than vitamin K or fresh frozen plasma in reversing warfarin immediately. It has a definite place in a life-threatening haemorrhage such as a subdural haematoma in a warfarinised patient, allowing prompt neurosurgical intervention. Unlike factor VIIa, it acts throughout the circulation, though the short term risk in patients with artificial heart valves is often overestimated. **Tranexamic acid** is currently undergoing evaluation as an adjunct to haemorrhage control following trauma in a multi-centre controlled trial (CRASH II trial).

Boffard KD, Riou B, Warren B, *et al*. Recombinant factor VIIa as adjunctive therapy for bleeding control in severely injured trauma patients: two parallel randomized, placebo-controlled, double-blind clinical trials. *Journal of Trauma* 2005;**59**:8-15

Ketchum L, *et al*. Indications for early fresh frozen plasma, cryoprecipitate and platelet transfusion in trauma. *Journal of Trauma* 2006;**60**:S51-58

Restrictive transfusion to a haemoglobin of $7g.dL^{-1}$ is appropriate in the post-resuscitation phase, but with caution in the elderly and in those with ischaemic heart disease.

Decompensating head injury

Maintain perfusion pressure
Avoid secondary insult
Decompress (temporary – mannitol and respiratory control; definitive – surgery if appropriate: burr hole, craniotomy, craniectomy)

Restoring blood volume and increasing osmolality are important therapeutic manoeuvres in the immediate care of decompensating head injuries. Hypertonic saline is a convenient, highly portable alternative to isotonic saline, but by increasing blood pressure, it may exacerbate uncontrolled bleeding in a similar way to larger volumes of isotonic saline solutions. Mannitol remains the most widely used agent for reducing intracranial pressure prior to emergency surgical decompression.

There may be a conflict of priorities if there is a severe head injury and uncontrolled bleeding elsewhere. This is thankfully rare! Golden rule – if in doubt, keep to ABCD order.

Secondary Survey

Head (dealt with separately in head injury section)
European Brain Injury Consortium (EBIC) guidelines (now old) and Brain Trauma Foundation and Association of Anaesthetists guidelines (updated):

- Avoid hypoxia (EBIC/AAGBI: $p_aO_2 > 13$ kPa)
- Avoid hypercapnia and severe hypocapnia (AAGBI: p_aCO_2 4.5 – 5.0 kPa, but reduce to 4.0 kPa if clinical or radiological evidence of raised intracranial pressure)
- Maintain blood pressure (AAGBI: MAP > 80 before ICP available)
- Keep CPP > 60 mm Hg (BTF: aggressive attempts to keep CPP > 70 mmHg with fluids and pressors was associated with increased risk of ARDS).

Intracranial haemorrhage (extradural, subdural, intra-cerebral, subarachnoid, intra-ventricular), diffuse axonal injury (not always evident on CT), contusion, oedema, fracture, hypoxic insult (from airway obstruction, respiratory depression or shock)

Face
Airway bleeding, sublingual haematoma, trismus
Nasal pack/balloon (beware basal skull fracture); manual reduction of maxilla (beware bleeding may $\uparrow$ or $\downarrow$)

Neck
Vascular tiger country; concealed by collar; not to be explored under local anaesthetic

Chest
Pneumothorax (see above for tension/open)

Drain every traumatic pneumothorax unless miniscule and perform a check chest x-ray afterwards
Do not perform positive pressure ventilation or take the patient in an air ambulance without draining a pneumothorax
In persistent pneumothorax, check position of drain (e.g. side hole at skin), consider applying suction (2-5 kPa), inserting further drain and investigating for tracheo-bronchial or oesophageal injury

Tracheo-bronchial rupture

Anticipate difficult airway management. The trachea may be compressed, partially severed and contain blood and debris. Fibre-optic intubation may be required but difficult: summon an expert to navigate across a proximal lesion or enter a distorted, blood-stained contra-lateral main-stem bronchus to achieve adequate oxygenation
Involve a thoracic surgeon as soon as the condition is suspected and liaise closely
Diagnose bronchoscopically by an expert diagnostic bronchoscopist if the patient's condition allows
Thoracotomy and surgical repair should be performed as soon as possible. Double lumen tube positioning may be difficult because of the anatomical disruption and distortion. Liaise with the surgeon to assess risk-benefit. In some cases, the surgeon may be able to help guide tube placement

per-operatively from within the chest.

Pulmonary contusion

Maintain a high level of suspicion: contusion evolves over several hours. The initial chest x-ray may underestimate the severity of the lesion, though the rapidity of onset of radiological signs correlates with severity. Try to distinguish from aspiration, haemothorax, and chest wall soft tissue swelling
Do not use CT scanning to diagnose pulmonary contusion, but use it to confirm the diagnosis and its extent, when a CT is indicated for other suspected thoracic injuries
Invasively monitor the blood pressure with frequent arterial blood gas estimations
Treat with added oxygen and if necessary by ventilatory support according to gas exchange, respiratory rate, tidal volume and effort: Face mask CPAP or BIPAP mask ventilation Mechanical ventilation via an endotracheal tube In unilateral cases with severe shunting, consider independent lung ventilation via a double-lumen tube Uncomplicated pulmonary contusion improves within 72 hours
Restrict post-resuscitation intravenous fluids
Provide intensive physiotherapy to reduce the risk of sputum retention and pneumonia. Prophylactic antibiotics are not needed.

Diaphragmatic rupture

Maintain a high index of suspicion in multiple trauma, including: Careful examination of the chest x-ray, noting an apparently raised hemi-diaphragm, basal atelectasis, a solid or gas-filled opacity above the apparent diaphragm and the course of the nasogastric tube Thorough examination of the diaphragm at laparotomy Awareness of low diagnostic sensitivity of ultrasound and may be missed on CT
Insert a naso- or oro-gastric tube
Involve thoracic or general surgeons as soon as the diagnosis is suspected
If uncertain on CT, confirm the diagnosis by upper GI contrast study or thoracoscopy
Repair the defect surgically, usually by laparotomy but may require thoracotomy or combined approach. Laparoscopic or thoracoscopic repair may be possible in small penetrating injuries

Causes of a wide mediastinum

Artefact from supine antero-posterior projection Aortic rupture Bleeding from other mediastinal vessels Sternal fracture Spinal fracture Pre-existing heart disease Thymus (in small children)

Aortic rupture – evidence on x-ray

Wide mediastinum Obliteration of the aortic knob

Obliteration of the space between the pulmonary artery and the aorta (the AP window)
Depression of the left main-stem bronchus
Deviation of the oesophagus
Presence of a pleural cap
Left haemothorax

Aortic rupture

In cases of uncertainty, to help *exclude* the diagnosis, consider: A repeat chest x-ray, performed erect after clearing the spine   CT angiogram   Trans-oesophageal echocardiography is regarded as a sensitive investigation, comparable with angiography in proximal aortic rupture
For definitive diagnosis, with sufficient anatomical detail for planning surgical procedures, angiography still considered the gold standard investigation
Prior to repair, including during transfer to a cardiothoracic centre, restrict the systolic blood pressure to 100-120 mmHg using agents such as nitroprusside, labetalol, propranolol and esmolol. This will necessitate invasive monitoring.
Refer promptly to a cardiothoracic surgeon for surgical management (primary repair or resection and grafting), using simple clamp repair, heparin-bonded bypass shunts during the repair, or cardiopulmonary or femoro-femoral bypass and cooling
In clamp techniques, consider optimal site for arterial line (e.g. right radial for proximal pressure measurement in tears at the aortic isthmus)
In cardiopulmonary or femoro-femoral bypass techniques, exclude other sources of internal haemorrhage (especially intra-abdominal) prior to full heparinisation

Myocardial contusion

Administer oxygen and analgesia as in medical cases of myocardial ischaemia
Monitor carefully for at least 24 hours, even in mild cases
Investigate using: Serial 12-lead ECG   Cardiac enzymes (e.g. troponin T), though the sensitivity and specificity have been questioned in major trauma   Echocardiography   Combining all three serially improves the sensitivity and specificity.
Consider carefully the medical history, the mechanism of injury and the physical pattern of injury to help distinguish myocardial contusion from (non-traumatic) myocardial infarction, which may indeed have caused the accident.
Treat symptoms and complications as in myocardial infarction (except of course, thrombolysis is unnecessary and contraindicated). Avoid hypotension and hypertension.

Oesophageal rupture

Involve a thoracic or gastrointestinal surgeon as soon as the diagnosis is suspected, as the time to definitive repair influences the prognosis
May be suspected on CT, but confirm by gastrografin contrast imaging or oesophagoscopy
Prophylactic antibiotics to provide Gram negative and anaerobic cover
Prompt direct repair at thoracotomy with wide drainage of pleural space and mediastinum

Mediastinal traversing wounds

Immediately involve a cardiothoracic surgeon or, if not available, a general surgeon capable of performing exploratory thoracotomy.
Treat tension pneumothorax, massive haemothorax and cardiac tamponade
Insert bilateral chest drains
Perform immediate or emergency thoracotomy in decompensating patients. Prepare for bilateral thoracotomy, generally exploring the side with the most evident blood loss first.
Investigate mediastinal structures, even in haemodynamically normal patients with no specific clinical or radiological signs: CT scan, aortic arch angiography, water-soluble contrast oesophagography, oesophagoscopy, bronchoscopy, and echocardiography. Consider neurological injury if close to the spinal cord or involves the aorta

Abdomen
Abdominal compartment syndrome

This is a previously under-recognised (and still controversial) condition, causing gut, renal and hepatic failure, impairing venous return and compromising ventilation.
Consider monitoring bladder pressure to recognise early:

- < 20 mm Hg: decompression not indicated
- 20-30 mm Hg: be concerned and consider decompression especially if high airway pressure, low urine output or poor cardiac output
- > 30 mm Hg: decompression indicated

Expedient decompression when abdominal compartment syndrome recognised. If three or more severe abdominal injuries, plan to leave abdomen open as prophylactic decision. If abdomen tight, avoid deep tension sutures and leave open or use mesh closure instead.

Intra-peritoneal bleeding

Liver

Treat as a source of major haemorrhage and consider hypotensive resuscitation especially in penetrating trauma. Cross-match for at least 8 units with promptly available type-specific blood.
Immediately involve an experienced general or hepatic surgeon
When emergency laparotomy is required as a resuscitative procedure, identify the injuries directly and consider packing in preference to exploration, according to surgical judgement.
Otherwise, identify intra-peritoneal bleeding using ultrasound or CT. If urgency permits, CT provides the best evaluation of liver parenchyma on which to base a judgement of conservative or surgical management.
Liaise with a specialist Liver Unit in cases of uncertainty and consider transfer
Manage the patient in an intensive care or high dependency unit, especially during conservative management of injuries capable of severe haemorrhage
Monitor coagulation and liver function carefully in cases involving major haemorrhage, hypotension, or when there is a risk of extensive hepatic ischaemia or infarction on CT. Treat derangements in coagulation with fresh frozen plasma, cryoprecipitate and platelets.
Monitor haemodynamic status and perform serial ultrasound and/or CT scans during conservative management, ensuring rapid access to surgical intervention if indicated.

Spleen

Involve surgical team as soon as potential injury identified, if not already involved in trauma team response
Proceed to laparotomy in a deteriorating patient without delaying to perform CT. A rapid ultrasound may be performed if immediately available.
In more stable patients, assess on ultrasound and proceed to CT scan. If conservative management feasible on scan evidence, observe in high dependency environment with rapid access to appropriate surgical intervention. Repeat ultrasound/CT scan at intervals.
If feasible and safe, preserve the spleen at laparotomy or, if impossible, consider omental splenic implantation
After splenectomy, maintain treatment with penicillin or an equivalent antibiotic and immunise against infection with *Streptococcus pneumoniae*, *Neisseria meningitidis* and *Haemophilus influenzae* at an appropriate stage

Perforated hollow viscus

Stomach, bowel

Maintain a high index of suspicion: Ultrasound will not show free gas but may reveal some intra-peritoneal fluid Consider free gas on the CT scan or sub-phrenic gas on an erect chest x-ray as indicative, although the gas may have come from an external wound Carefully examine the full extent of the gastrointestinal tract at laparotomy performed for other indication
Proceed to laparotomy: Primary closure, with or without resection, is possible in most injuries Exteriorisation in destructive colonic wounds, massive contamination and poor risk patients
Provide antibiotic prophylaxis with Gram negative and anaerobic cover

Bladder

Suspect the diagnosis in association with pelvic fracture or a high seat belt mark and search for adjacent injuries, especially to the rectum
If there is blood at the meatus, perineal bruising, a high riding prostate, or failure of gentle catheterisation, perform water-soluble contrast urethrography then cystography
Confirm the diagnosis with cystography, using 150-300 mL of water-soluble contrast (antero-posterior and oblique views). Combine with cystoscopy in cases of uncertainty.
Management varies with the site and extent of the injury: Serious contusions and small extra-peritoneal ruptures may be managed conservatively, with an indwelling urethral catheter, provided that careful monitoring for sepsis is maintained Larger extra-peritoneal ruptures supra-pubic catheterisation for up to two weeks, peri-vesical drainage and antibiotics Intra-peritoneal rupture requires surgical repair and usually supra-pubic catheterisation

Retroperitoneal injury

Pancreas

Consider the diagnosis in all cases of blunt abdominal trauma, and inspect ultrasound and CT scans with this in mind, recognising their lack of sensitivity.
Measure serum amylase but recognise its insensitivity.
Proceed to laparotomy acutely according to general criteria of peritonism or intra-peritoneal haemorrhage, or if there is CT evidence of severe disruption/ impaired perfusion, according to

surgical judgement.
In equivocal cases, consider emergency ERCP.
Conservative management is similar to that of acute (non-traumatic) pancreatitis, with particular care in maintaining fluid balance and urine output, and careful monitoring for the development of a pseudocyst or abscess with repeated ultrasound and/or CT.

Kidney

Always inspect the flank during the log roll and check for macroscopic haematuria as part of the secondary survey (recognising that its absence does not exclude renal injury). Consider other urinary tract injuries in the differential diagnosis of haematuria
Proceed to laparotomy in cases associated with haemorrhagic shock and consider an on-the-table IVU if necessary
Use ultrasound in the Emergency Department but, in the non-shocked patient, rely more on CT with contrast urography, supplemented by formal IVU or renal angiography according to surgical judgement. Be cautious with contrast administration in shocked patients. A contrast CT or IVU should be performed if there is macroscopic haematuria.
Manage conservatively or operatively according to the evidence of injury: Manage renal contusions and small cortical lacerations conservatively Shattered kidneys and renal pedicle injuries may require immediate surgery In deep parenchymal and collecting system lacerations, proceed to surgical exploration or plan conservative management with careful monitoring on an intensive care or high dependency unit, according to surgical judgement. In renal vascular injury with on-going haemorrhage, consider arterial embolisation

Spine
Fractures and subluxations in the cervical spine
Jefferson blow-out (C1) – axial compression
Odontoid (C2) – type I = tip (minor, stable); type II = waist (unstable, cortical bone
à poor healing); type III = body (unstable, cancellous bone à good healing)
Hangman (C2) – posterior elements
Uni-/bi-facet subluxation (less than/more than 50% displacement)
Pseudo-subluxation (children)
Other fractures (e.g. inferior tear drop – looks like minor anterior fracture, but may be
unstable with posterior element ligamentous disruption)

Useful descriptive terms – angulation, loss of lordosis, displacement
anteriorly/posteriorly on underlying vertebra, loss of anterior height, retro-pulsion of
bone fragments into spinal canal, pre-vertebral soft tissue swelling

Treatment of cord injury – Bracken: methyl prednisolone 30mg/kg then
5.4mg/kg/hr was previously standard in North America (and failure to administer it a
suable offence). The evidence for its use has been questioned [15] and many centres
have now abandoned it.

Bracken MB. Steroids for acute spinal cord injury. *Cochrane Database of Systematic Reviews*. 2002;(3):CD001046

Short DJ, El Masry WS, Jones PW. High dose methylprednisolone in the management of acute spinal cord injury - a systematic review from a clinical perspective. *Spinal Cord* 2000;**38**:273-86

Maintenance of cord perfusion pressure. Role of MRI. Consider decompressive surgery if cord injury incomplete.

The limbs

Mechanism of unstable pelvic fracture

Antero-posterior compression, causing diastasis of the pubic symphysis, sacro-iliac fracture/dislocations and sacral fractures. These are associated with haemorrhage from the posterior pelvic venous complex and more rarely from branches of the internal iliac artery. The pelvic volume is increased in the 'open book' injury and the urethra is vulnerable.

Lateral compression, leading to internal rotation of the hemi-pelvis and predisposition to bladder and urethral injury, but less risk of major haemorrhage.

Vertical shear, disrupting the sacro-tuberous and sacro-spinous ligaments, leading to major instability and significant risk of haemorrhage.

Unstable pelvic fracture

Assess pelvic tenderness/stability at an early stage. Avoid repeated examination so as not to risk displacing clots and worsening haemorrhage
Perform abdominal ultrasound and pelvic x-ray in the Resuscitation Room. Look for evidence of two breaks in the pelvic ring, especially at the sacro-iliac joints and the arcuate lines of the sacral foramina.
In haemodynamically unstable patients: Secure large-bore venous access   Cross-match at least 8 units and arrange for type specific blood to be rapidly available   Summon an experienced orthopaedic surgeon   Consider immediate pelvic binder or splint (custom or makeshift sling or pneumatic anti-shock garment) depending on fracture configuration
Consider rapid external fixation, angiographic embolisation or pelvic packing in haemodynamically unstable patients. Consider other causes of bleeding too. CT scan is valuable but hazardous in a shocked patient.
In less urgent cases, arrange CT scan and plan definitive surgical repair if indicated (typically on days 4-7, though some injury patterns are amenable to emergency surgical fixation). Associated rupture of pelvic viscera demands prompt surgical intervention, debridement, repair and antibiotic cover. Open pelvic fractures have a high mortality.

Van Vugt AB, *et al*. An unstable pelvic ring – the killing fracture. *Journal of Bone and Joint Surgery Br*. 2006; **88-B**: 427-433

The Angiography Suite should be prepared as a mini-ICU with close monitoring, on-going resuscitation and re-assessment of other bleeding sources by dedicated clinicians

Velmahos GC, *et al*. A prospective study on the safety and efficacy of angiographic embolization for pelvic and visceral injuries. *Journal of Trauma* 2002;**53**:303-308

Long bone fracture fixation

Early intra-medullary fixation of femoral shaft fractures remains the treatment of choice in most patients. Although the evidence is scanty, it may be wise (but still controversial) to avoid immediate fixation in severe chest injuries and in severe head injuries with intracranial hypertension. In unstable, critically injured patients, orthopaedic damage control of long bone fractures should be considered. Temporary internal fixation is associated with less systemic inflammatory response than early definitive plating or nailing. Conversion to intra-medullary fixation should be considered (if appropriate) when the inflammatory response has subsided.

While **complete definitive fracture fixation** (early total care) is often appropriate, orthopaedic damage control must be considered in unstable patients, especially in the face of hypothermia, acidosis and an evolving coagulopathy. With new evidence from inflammatory markers (e.g. interleukin 6) and the concept of the 'second hit', major interventions should be avoided if possible between days 2 and 4 following trauma, because of an increased susceptibility to organ failure.

<div align="right">Hildebrand F, et al. Damage control: extremities. Injury 2004;35:678-689</div>

Vascular impairment: consider need for emergency fracture/dislocation reduction if tissues at risk, or angiography to define vascular injury. Remember compartment syndrome and avoid prolonged local anaesthesia if limb is at risk (e.g. closed tibial fracture).

Fat embolism (Georgopoulos D, *et al*. Fat embolism syndrome: clinical examination is still the preferable diagnostic method. *Chest* 2003;**123**:982-983)

<u>**External**</u> (burns dealt with in separate lecture)

Airway burn – inhalational injury

Body surface burn – area, depth; fluid estimate 4 mL/kg/%/day

Complicating factors – myoglobinuria (electrical injury), physical injury (escaping/explosion)

Appendix

A. Audit

Trauma Audit & Research Network (TARN)

NCEPOD study in progress (3 month national survey)

ICNARC for intensive care

Physiological Severity Scoring

Revised Trauma Score (respiratory rate, systolic blood pressure, Glasgow Coma Score)

Probability of survival $Ps = 1/ (1+e^{-a})$ where $a = a0 + a1.RR + a2. SBP + a3. GCS$ (logistic regression)

Anatomical Severity Scoring

Abbreviated Injury Scale

9 body regions: head, face, neck, chest, abdomen, spine, upper limbs, lower limbs, external

Score for each injury (1-6): minor, moderate, serious, severe, critical, currently untreatable

Injury Severity Score (6 regions, condensed from AIS)

Head, neck (including C-spine), face, chest (including T-spine), abdomen (including L-spine), limbs, external

ISS = sum of squares of AIS scores in the three most severely injured ISS regions

Combined Anatomical and Physiological Severity Scoring

TRISS (Revised Trauma Score, Injury Severity Score, Age – using logistic regression)

Probability of survival $Ps = 1/ (1+e^{-a})$ where $a = a0 + a1.RTS + a2.ISS + a3.age$

Severity-outcome Scoring

Ps not applicable to individual patients. **W statistic** = excess survivors /100 patients, used to stratify performance of different centres. **Ws** is a weighted adjustment used by TARN. TARN's new logistic model uses ISS, age and GCS (as the only physiological parameter), overcoming the problem of omitted respiratory rate values.

B. Other References

Advanced Trauma Life Support

- 7th Edition, American College of Surgeons Committee on Trauma 2004

Royal College of Surgeons of England
- Better Care for the Severely Injured 2000 (downloadable from website)
- Management of Patients with Head Injuries 1999 (downloadable from website)

Royal College of Anaesthetists
- Guidelines for the Provision of Anaesthetic Services 1999 – Trauma Anaesthesia

Association of Anaesthetists of Great Britain and Ireland
- Recommendations for the safe transfer of patients with brain injury 2006 (http://www.aagbi.org/publications/guidelines.htm)

Intensive Care Society
- Transport of the Critically Ill Adult 2002

Brain Trauma Foundation – braintrauma.org
- The Management and Prognosis of Severe Traumatic Brain Injury 1995, 2000, 2003
- Surgical Management of Traumatic Brain Injury 2006

Eastern Association for the Surgery of Trauma (EAST) – *east.org*
- Various guidelines e.g. c-spine, pelvis, abdominal blunt trauma

NICE Guidelines – *nice.org.uk*
- Head Injury 2003, Pre-hospital Intravenous Fluid Therapy in Trauma 2004

Hypertonic saline
- Stern S, *et al*. Comparison of the effects of bolus vs. slow infusion of 7.5% NaCl/6% dextran-70 in a model of near-lethal uncontrolled hemorrhage. *Shock* 2000;**14**:616-22
- Cooper DJ, *et al*. Prehospital hypertonic saline resuscitation of patients with hypotension and severe traumatic brain injury. *JAMA* 2004;**291**:1350-1357
- White H, *et al*. The use of hypertonic saline for treating intracranial hypertension after traumatic brain injury. *Anesthesia & Analgesia* 2006;**102**:1836-1846.
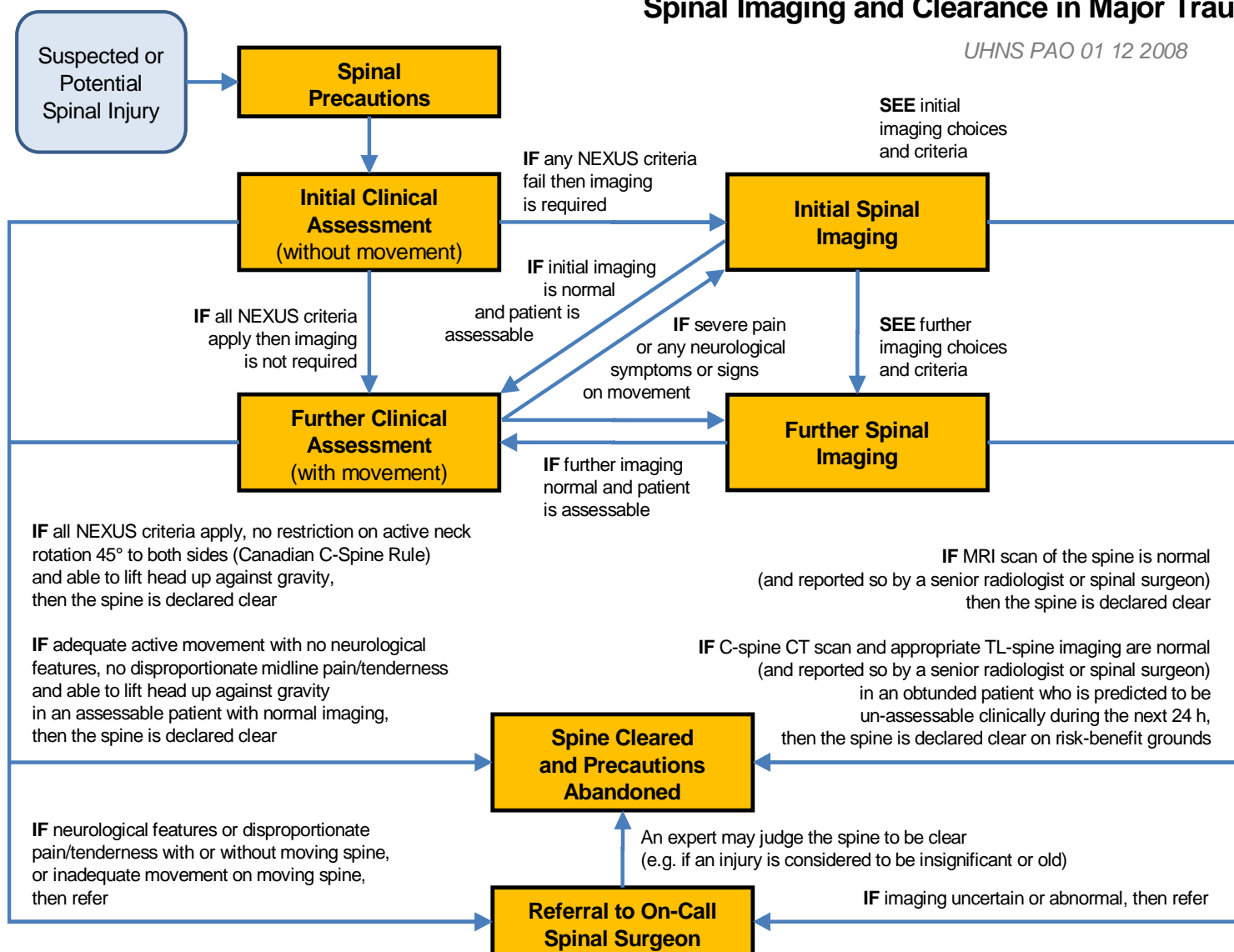
Fluid resuscitation in pre-hospital trauma care
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- Greaves I, Porter K, Revell M. *JR Coll Surg Edinb* 2002;**47**:451-7

- JRCALC Pre-hospital Guidelines (2000, updated 2002 version 2.2)

Spinal clearance
- British Trauma *Society*. Guidelines for the initial management and assessment of spinal injury. *Injury* 2003;**34**:405-425
- C. G. Morris and B. Mullan. Clearing the cervical spine after polytrauma: implementing unified management for unconscious victims in the intensive care unit. *Anaesthesia* 2004;**59**:755-761
- C. G. T. Morris and E. McCoy. Cervical immobilisation collars in ICU: friend or foe. *Anaesthesia* 2003;**58**:1051

Suspected or Potential Spinal Injury

Spinal Precautions

Initial Clinical Assessment (without movement)

IF any NEXUS criteria fail then imaging is required

SEE initial imaging choices and criteria

Initial Spinal Imaging

IF all NEXUS criteria apply then imaging is not required

IF initial imaging is normal and patient is assessable

IF severe pain or any neurological symptoms or signs on movement

SEE further imaging choices and criteria

Further Clinical Assessment (with movement)

IF further imaging normal and patient is assessable

Further Spinal Imaging

IF all NEXUS criteria apply, no restriction on active neck rotation 45° to both sides (Canadian C-Spine Rule) and able to lift head up against gravity, then the spine is declared clear

IF adequate active movement with no neurological features, no disproportionate midline pain/tenderness and able to lift head up against gravity in an assessable patient with normal imaging, then the spine is declared clear

IF MRI scan of the spine is normal (and reported so by a senior radiologist or spinal surgeon) then the spine is declared clear

IF C-spine CT scan and appropriate TL-spine imaging are normal (and reported so by a senior radiologist or spinal surgeon) in an obtunded patient who is predicted to be un-assessable clinically during the next 24 h, then the spine is declared clear on risk-benefit grounds

Spine Cleared and Precautions Abandoned

IF neurological features or disproportionate pain/tenderness with or without moving spine, or inadequate movement on moving spine, then refer

An expert may judge the spine to be clear (e.g. if an injury is considered to be insignificant or old)

Referral to On-Call Spinal Surgeon

IF imaging uncertain or abnormal, then refer

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SPINAL PRECAUTIONS

Hard collar, head blocks and firm surface
Log-rolling to change position
Manual in-line immobilisation of head and neck when collar or blocks removed
(e.g. for airway procedures)

Spinal Imaging and Clearance in Major Trauma

UHNS PAO 01 12 2008

ASSESSABILITY WITHOUT IMAGING – *NEXUS CRITERIA

No posterior midline spinal tenderness
No focal neurological deficit
No evidence of intoxication (including presence of significant alcohol or sedative drugs)
No painful distracting injury
Normal mental status

* modified to apply to thoraco-lumbar, as well as cervical spine

ASSESSABILITY AFTER NORMAL IMAGING

Careful judgement by senior clinician on case-by-case basis if any of the NEXUS criteria still do not apply.
The patient must be at least:

Alert when roused (E ≥ 3)
Appropriate (V ≥ 4)
Able to obey commands (M = 6)

IMAGING MODALITIES

CT scan: C-spine 2-3 mm slices and chest/abdomen/pelvis 5 mm slices, all reformatted to 1 mm for sagittal and coronal spine reconstructions
Plain views: C-spine antero-posterior (AP), lateral and open-mouth odontoid peg ± 45° obliques of cervico-thoracic junction; TL-spine AP and lateral
MRI: Sagittal STIR to screen for soft tissue and bony injury; high resolution (sagittal/axial T1/T2) to assess areas of concern on CT or STIR

INITIAL IMAGING CHOICES AND CRITERIA

IF high risk mechanism with suspected serious trunk injury or any cardio-respiratory instability, then **CT from occiput to pelvis**, irrespective of conscious level

IF cord or nerve root symptoms or signs, or evidence of posterior circulation syndrome, then **MRI entire spine** (± CT of appropriate areas for bone detail)

IF GCS < 13, then **CT entire C-spine** from occiput (C0) and extend down to T4

IF GCS 13-14, then **AP and lateral plain C-spine views** (omitting peg and oblique views) and **CT upper C-spine** from C0 to C3 (sparing thyroid from radiation). If age ≥ 65 or clinical evidence of cord/nerve root injury/posterior circulation syndrome, or if plain views (done first) are inadequate/abnormal/uncertain, then **CT entire C-spine instead**

IF GCS = 15 and no cord, nerve root or vascular features, then **plain C-spine films**

IF mechanism puts TL-spine at risk, then **plain TL-spine films**, but if CT of chest or abdomen is indicated separately, then replace corresponding plain views with **CT scan**

IF pregnant, consider **MRI** TL-spine (± C-spine) **instead of plain views or CT scan**. Note that plain views or C-spine CT (extended down to T4) are not contraindicated. MRI of the entire spine may cause significant heating that may affect the fetus. Senior judgement is required to tailor the imaging protocol.

FURTHER IMAGING CHOICES AND CRITERIA

IF C-spine plain films normal, but severe localised posterior midline tenderness or disproportionate neck pain with or without movement, then consider **CT scan ± MRI scan**

IF plain films show fracture, mal-alignment or soft tissue swelling or findings uncertain, then **CT scan**

IF CT shows:

Fracture involving more than 1 column, or
Significant soft tissue abnormality, or
High risk factors for disco-ligamentous/vascular injury:

– Subluxation or dislocation
– Fracture through foramen transversarium

then **MRI scan**

IF neurological features on moving neck on further clinical assessment, then **MRI scan**

IF MRI scan shows unsuspected bony injury, consider **CT scan** of this area to reveal bone detail (if not already done)

Section 8   Sedation

§   Sedation is a primary consideration in the care of critically ill patients. Adequate sedation facilitates effective ventilation, tolerance of invasive procedures and is a vital component of treating some critical illnesses, such as raised intracranial pressures. Conversely excessive sedation extends stay on ICU with the concomitant risks of hospital acquired infections and carries increase risks of drug related effects, such as hypotension. Withdrawing sedatives can be difficult in the critically ill, due to altered metabolism ,altered distributions and altered elimination. These issues and the specific agents are dealt with below.

8.1 Principles of sedation on ICU

Sedative and analgesic agents are utilised on virtually every ICU patent at some point in their care and represent the key pharmacological expenditure on most units. An understanding of the requirement for good sedation practice is vital.

Indications for sedation & analgesia

- tolerance of endotracheal tube and IPPV
- tolerance of invasive monitoring (insertion)
- tolerance of interventions e.g. suctioning, prone positioning
- relief of psychological distress
- analgesia post-operatively or post-trauma
- relief of agitation
- reduction in raised intracranial pressure/ prevention of surges in ICP
- reduction in oxygen demand and oxygen consumption
- simulation of sleep wake cycles

Effects of inadequate sedation/ analgesia

- agitation and confusion
- inadvertent removal of invasive monitoring
- inadvertent removal of endotracheal tube
- ventilator dysynchrony
- rises in ICP
- CVS instability
- Increased O2 demand
- Post traumatic stress disorder
- Sympathetic activation (tachycardias, hypertension, tachypnoea)
- Poor pain control
  - Adverse physiological responses (hypercoagulability, immunosupression)

Effects of excessive sedation/ analgesia

- Prolonged weaning from ventilator and increased LOS on ICU
- Inadequate cough / clearance of secretions
- Increased exposure to side effects (e.g. hypotension, bradycardia, increased lipid loads)

- Difficulty assessing neurological function or changes in status
- Risk factor for ventilator associated pneumonia
- Post ICU psychological stress (related to prolonged amnesia)

§ Sedation and analgesia can be delivered via a number of routes in the critically ill patient and these include intravenously (IV), enterally (PO/ NG), rectal (PR), epidurally (ED), inhaled and intramuscularly (IM).

§ The choice of route will depend on the clinical state of the patient. Traditionally IV has been the usual route due to the variable absorption of the critically ill patient, the better bio-availability of the IV route, the need for rapid titration in critical illness and the nature of the preferred agents (i.e. only IV formulation). Oral and other routes tend to be used during the weaning phase of the sedation and analgesia administration.

§ The option between bolus sedation versus continuous sedation depends upon the patient. Bolus will result in less sedation given but has the disadvantage of peaks and troughs that may result in agitation and other adverse effects. In UHNS we tend to give sedatives via infusion with boluses for interventions or for when rapid deepening is needed.

§ Critically ill patients have a variety of problems with handling all pharmacological agents, not just sedatives. However the nature of most sedative and analgesic agents means they are prone to accumulation, especially form continuous administration and this is extenuated in critical illness due to:

- Altered absorption (as noted above) : due to decreased sphlancic perfusion and altered bowel micro-environment
- Altered protein binding : decreased albumin, decreased carrier proteins,
- Altered volume of distribution : capillary leak, intravascular depletion, tissue oedema, third space loss (e.g. ascites)
- Altered receptor populations : down regulation of receptor channels
- Altered drug metabolism : enzyme dysfunction, altered pH, hepatic dysfunction
- Altered elimination : hepatic and renal dysfunction

Section 8:2   Drugs used for sedation

A large variety of drugs can be employed for sedation and analgesia on ITU. In practice we utilise only a few, with the majority been used occasionally for specific indications or for difficult weaning sedation. The drug strategy utilised for general usage ans specific strategies is dealt with in section 7.3

8.21 Propofol

- Propofol is a phenolic compound prepared as a lipid emulsion and given intravenously. Its popularity as an anaesthetic induction and maintenance agent has carried over into critical care where it is the most commonly used sedative.
- It is given as a 1% solution (10mg/ml) IVI typically as 50ml syringes or as 100ml bottles
- Typical infusion rates are 1- 20 ml/h but higher rates may be needed in some patients
- Its advantages are titratibilty and rapid onset of effect (q.v. other sedatives on ICU) and acceptable levels of accumulation. It is very effective at obtunding the

pharyngeal and laryngeal responses allowing tolerance of ETT and can produce apnoea or ventilatory depression in some patients. It produces some amnesia and anxiolysis. It is also an anticonvulsant.

- These properties make it ideal for sedation that needs to be rapidly titrated, such as in neurocritical care.
- Its main disadvantage is that of hypotension. This occurs due to vasodilatation, increased venous capacitance, mild myocardial depression and reduction in sympathetic tone.
- Other problems are the high lipid content may lead to hyperlipidaemia in some patients and the occurrence of a rare "propofol infusion syndrome" in children. Historically there has been concern regarding possible immunosuppressive effects.
- Propofol is metabolised mainly in the liver but also has significant extra-hepatic metabolism and elimination
- **Propofol infusion <u>syndrome (PRIS)</u>** is a rare and often fatal syndrome described in critically ill children and increasingly in adults, undergoing long-term propofol infusion at high doses. The main features of the syndrome consist of cardiac failure, rhabdomyolysis, severe metabolic acidosis and renal failure. Adult case reports usually involve patients with acute neurological illnesses or acute inflammatory diseases complicated by severe infections or even sepsis, and receiving catecholamines and/or steroids in addition to propofol sedation at doses higher than 5 mg/kg per hour, for more than 48 hour. In these cases, alternative sedative agents should be considered.

8.22 Midazolam

- Midazolam is a short acting benzodiazepine prepared as a water-soluble preparation but becoming lipid soluble in-vivo.
- It acts rapidly after bolus injection and the infusion on ITU is typically given as 1-10 ml/h of a 0.1% solution (1 mg/ml)
- Midazolam rarely produces the CVS instability of propofol but has a tendency to accumulate after prolonged infusions, especially in patients with renal or hepatic failure (it is metabolised in the liver then excreted by the kidneys)
- Midazolam is also a potent anticonvulsant and has strong amnesic properties

8.23 Alfentanil

- Alfentanil is a potent and short acting opioid commonly used in infusion on ICU. Its shorter duration of action and relatively rapid clearance from the plasma mean that it is often used in combination with 1% propofol in patients where duration of sedation is likely to be short or where rapid emergence and re-sedation is required (such as in assessing neurosurgical patients)
- Alfentanil is prepared as a 500 mcg/ml solution (0.05%) and infused at rate of 1-5 ml/h. In some patients tolerance can be displayed and higher rates may be required
- Alfentanil can cause hypotension in some patients. It has the advantage of diminishing airway reflexes and facilitating ventilator / ETT tolerance

8.24   Morphine

- Morphine is given on ICU typically by infusion for it's sedative and analgesic properties.
- Typically it is prepared as a 0.1 % solution (1mg/ml) and infused at 1-10 ml/h
- Morphine readily accumulates in critically ill patients and the time taken for the drug to clear from the system progressively increases with the duration of infusion, moreso than alternative opioids such as alfentanil and remifentanil. This becomes more apparent in patients with renal and hepatic failure.
- Morphine is an effective analgesic, as are some of its metabolites. It is also a good sedative and its well known respiratory depressant effects are of use in most critically ill patients.
- Its adverse effects are few in ICU patients but can include hypotension and bradycardia. In higher infusion rates there is evidence it can act as an immunosupressant.

8.25   Clonidine

- Clonidine is an α2 agonist with analgesic and sedative properties. It can be given as an IV infusion or as oral bolus doses.
- It is typically used on UHNS ICU as a secondary sedative agent either in patients with high sedative needs or in patients weaning form sedation. The variable response in patients can be marked. Its main side effect is that of hypotension although this is not seen as often as one would expect. In the long term depression is a side effect although rarely a problem on ICU.
- IV it is given as a infusion of 600 mcg in 48ml 0.9% saline at 2- 5 ml/h. Most patients will get adequate effects from the starting dose of 2 ml/h.
- Orally it is given as 600 mcg/day in divided doses, typically as 200 mcg tds. This is then reduced as required.

8.26   Ketamine

- Ketamine is a phencyclidine derivative producing potent analgesia and dissociative anaesthesia
- It acts on NMDA receptors in the thalamus, cortex and reticular system. It also acts as a symapthomometic agent having mild vasopressor and bronchodilator properties.
- Its metabolism is primarily hepatic
- It is infused at a rate of 1 – 2 mg/kg/h. Hence prepare 2000mg in 40 ml to create a 50 mg/ml solution and run at appropriate rate (e.g. 1.4 to 2.8 ml/h for a 70 kg patient).
- Its main indications on ICU at UHNS are
    - Status asthmaticus
    - Profound CVS instability
    - Major trauma (by some consultants)
- Its main side effects relate to emergence phenomenon (visual hallucinations), which can be reduced by concomitant benzodiazepine infusions.

UHNS Guideline for remifentanil analgosedation in ICU

Is the patient suitable for remifentanil? CONSULTANT DECISION
REMIFENTANIL SHOULD NOT BE USED FOR LONGER THAN 72HRS

Indications for use
- Head injuries/patients with low GCS needing neuro assessment (needing to be regularly assessed)
- Raised ICP (>20mmHg) – resistant to medical management
- Severe Hepatic impairment
- Assessment of neurological function in a ventilated patient (or to make an assessment of optimal cerebral perfusion pressure)

Drug Preparation

5mg in 50mls Sodium Chloride 0.9%
= 100micrograms per ml

Via burette on a dedicated line

Drug dose range between 0.1 and 0.25micrograms/kg/min

Calculate patient's IDEAL body weight (kg) and determine remifentanil dose limits
UPPER RATE LIMIT 0.25micrograms/kg/min
Commence infusion at 0.1µg/kg/min (see examples)

ASSESS patient using sedation and pain score ← REASSESS HOURLY

Increase or decrease remifentanil infusion rate by 1 ml/hr every 5 min. until desired sedation score achieved

Remifentanil 0.2micrograms/kg/min

Is the patient adequately sedated? → YES

NO

Pain or tube/ventilator intolerance?

Anxious or agitated?

Increase remifentanil rate 1ml/hr every 5 minutes until adequate **pain relief** or **max dose** of 0.25micrograms/kg/min has been reached

Start propofol 1% at 6ml/hr (0.5mg/kg/hour)

Increase infusion rate by 1 ml/hr until desired **sedation score** achieved

If max propofol infusion rate reached (20ml/Hour) give Midazolam 0.5mg increments until patient settled

Is the patient adequately sedated? → YES

NO

Example rate ranges:
(from product data sheet)

3 – 8 ml/hr for a 50kg patient

4 – 10 ml/hr for a 70kg patient

6 - 15 ml/hr for a 100kg patient

START AT LOWEST RATE

Authors: Dr P. Glover, P. Caddell, N. Walker, Royal Victoria Hospital Regional ICU, Belfast

Modified by Imran Hanif, Pharmacist Team Leader for Surgery, February 2009

CAUTIONS

- Remifentanil is a potent drug TREAT LIKE AN INOTROPE
- NO BOLUSES - For special procedures increase infusion rate by 25%
- Watch for **bradycardia** and **hypotension** in over administration. Should resolve rapidly
- Use a dedicated iv line
- IV sites should be aspirated (CVP) or flushed (peripheral) after discontinuation
- Consider IV morphine 10-30mg 30 mins prior to discontinuing Remifentanil infusion

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8.27 Thiopentone

- Thiopentone is a barbiturate anaesthetic agent primarily used in anaesthetic practice for induction of anaesthesia in emergency settings or where rapid securing of the airway is required as part of a rapid sequence induction.
- Its use in critical care is reserved for
  - Intractable status epilepticus
  - Refractory raised ICP (generally prior to surgical intervention)
- In these circumstances it is given to achieve an iso-electric EEG. However in most ICUs (ours included) continual EEG monitoring is not used and hence dosing is arbitrary.
- The dose involves initial bolus dose of 4- 6 mg/kg and then infusion commenced at 0.5 – 4 mg/kg/h.
- The main disadvantages to thiopentone infusions are hypotension, which may be marked and accumulation, which may mean residual sedation for a prolonged period after its cessation.

8.28 Diazepam

- Diazepam is a well known benxodiazepine sedative utilised in critical care in the oral and IV formulations. Its main utility is in supplemental sedation for patients weaning from sedatives.
- Dosages are titrated to the individual but may range from 2-5 mg tds – qds. This dose is then tapered as the patient weans.

8.29 Haloperidol

- Haloperididol is a butyrophenone neuroleptic agent popular in psychiatric practice and as a first line sedative for confused patients in ward practice. Its use on ICU is typically for similar problems during the sedative weaning process or where delirium is a key feature of the process.
- It is prepared either orally or parenterally, where it can be given IM or IV.
- Dosages vary but typically 2-5 mg is given up to 3-4 hrly with caution exercised if daily doses more than 20 mg are needed.
- Side effects may include dysphoria, hypotension, anticholinergic side effects (dry mouth, tachycardias, retention), rigidity or dyskinesias, although these latter effects are rare on ICU.

8.210 Carbamazepine

- Carbamazepine is a relatively new usage of an established anticonvulsant. The sedative properties of carbamazepine can be used during the weaning process from sedation.
- The drug is typically given in its oral/NG formulation at doses of 200 mg tds and then is gradually weaned.

Section 8.3   Strategies for sedation

As noted above, most patients have very simple sedation regimes titrated to sedation scores.

8.31   Common strategy

- Combination of propofol 1% @ 1-20 ml/h and alfentanil (0.5 mg/ml) @ 1-5 ml/h
- If inadequate sedation on this regime is the problem:
    - Dysynchrony with the ventilator?   Increase alfentanil range to 1-10ml/h
    - Too light for targeted sedation score?   *E.g. neuro patient, young patient, alcohol/drug abuser*
- Increase the propofol range to 1-30 ml/h
- If now maximal on propofol / alfentanil ?   Consider adding midazolam **(seek senior advice)**

- Patient likely to be prolonged stay/ slow wean (e.g. > 5 days)
    - Combination of midazolam @ 1-10 ml/h and morphine @ 1-5 ml/h

8.32   Neuocritical care

- In most cases the plan is to provide a period of deep sedation to allow opportunity for CNS recovery and control of ICP then a period of lightening to assess neurological functioning
- The combination of propofol / alfentanil is well suited for this

- In cases where large amounts of sedation are not adequate sedating the patent and ICP control is critical (see section on neurocritical care) the options are
    - Addition of midazolam
    - Paralysis
    - Addition of Carbamazepine
    - Thiopentone coma

    In each of these cases you should **seek senior advice**

8.32   Asthma

- Most asthmatics are sedated adequately with either propofol / alfentanil or midazolam / morphine
- In some cases due to refractory bronchospasm and dynamic hyperinflation further options are considered and may include
    - Ketamine infusion
    - Inhaled isoflurane

    In each of these cases you should **seek senior advice**

8.34   Difficult to sedate patents

- Rarely despite good doses of sedatives patients are not adequately sedated for their needs

- Before resorting to additional agents a thorough review of potential causes should be sought
  - Problem with delivery e.g. cannula tissued, CVC blocked or misplaced, GI tract non-functional
  - Tolerance to sedatives e.g. prolonged stay, IVDU, alcohol abuse, chronic sedative usage
  - Increasing pain e.g. peritonitis, wound infections, CVC infections, pressure areas, joint stiffness due to immobility
  - Increased discomfort with the ventilator e.g. ETT moved distally, wrong ventilatory mode
  - Drug interactions e.g. enzyme inducers
  - Improving clinical state e.g. resolving raised ICP, resolving organ failure improving drug tolerance

- Next consider what the exact problem is. Is it problems with the ventilator synchronising? Is it tube irritation? Is it pain? Is the patient too light and in discomfort or disorientated? This will allow rationale addition of further analgesics / respiratory depressants / sedatives.
- Additional agents/ alternative agents can include
  - Clonidine
  - Haloperidol
  - Diazepam
  - Clonazepam
  - Carbamazepine
  - Ketamine
  - Fentanyl

8.4 sedation scores

8.41 sedation scores

- Due to the concerns regarding over and under sedation it is important we titrate sedation to the optimum level for the patient. There is some evidence that this is facilitated by the use of sedation scores and they provide an objective means of assessment for medical and nursing staff alike.

- There are many sedation scores in use internationally. We use the RASS Score (Richmond Agitation and Sedation Scale) see below

Richmond Agitation and Sedation Scale (RASS)

+4	Combative	violent, immediate danger to staff
+3	Very Agitated	Pulls or removes tube(s) or catheter(s); aggressive
+2	Agitated	Frequent non-purposeful movement, fights ventilator
+1	Restless	Anxious, apprehensive but movements not aggressive or vigorous
0	Alert & calm	
-1	Drowsy	Not fully alert, but has sustained awakening to *voice* (eye opening & contact ≥ 10 sec)
-2	Light sedation	Briefly awakens to *voice* (eye opening & contact < 10 sec)
-3	Moderate sedation	Movement or eye-opening to *voice* (but no eye contact)
-4	Deep sedation	No response to voice, but movement or eye opening to *physical* stimulation
-5	Unarousable	No response to *voice or physical* stimulation

Source: Pain Manag Nurs © 2009 W.B. Saunders

- In addition patients may be paralysed. **Patients who are paralysed must have sedation depth assessed prior to paralysis to minimise the risk of awareness and should be at the deeper levels of sedation**. **Seek senior advice if you are unsure about this.**

- The ideal RASS score depends on the patient and their pathology. Generally 0 or -1 is the goal for patients weaning.
  A score of – 4 or - 5 should be reserved for patients with

    - Raised intracranial pressure
    - Status epilepticus
    - High ventilator pressures and FiO2

- The required score should be prescribed daily on the ICU chart

8.42 Sedation holds

- Based on the evidence that over sedation is detrimental for patients and results in prolonged stays there should be daily review on the need for sedation, sedation score required and a sedation hold should be considered.

- Sedation holds may be of two forms. The first is to assess underlying neurological status and is dealt with in section 6. The second is to permit a period of lightening from sedation and an opportunity for the patient to metabolise and eliminate the drugs. There is good evidence that this shortens the ICU stay and duration of ventilation and may impact on ventilator-associated pneumonia. It is a recommendation in the surviving sepsis guidelines and in some care bundles.

- Consider it in

  - Patients on minimal to moderate respiratory support (PEEP < 10cmH2O, FiO2 < 0.5)
  - Patients on minimal CVS support (NORAD < 10 ml/h, DOBUT < 5 ml/h)
  - No concern regarding raised ICP or cerebral oedema

- Sedation should be stopped until the patient is awake and cooperative (sedation score 1). Re-sedation should occur if the patient becomes agitated or distressed. Concerns that patients on sedation holds have higher rates of CVC or ETT displacement are unfounded in studies.

8.5 Weaning sedation

- The process of weaning sedation patients incorporates a spectrum of ease from the short stay post surgical patient in whom sudden cessation is accompanied by smooth emergence to the slow titration of oral sedatives in the long stay ICU patient complicated by agitation and confusion

- What should be the criteria for initiating sedative withdrawal? Clearly there should have been some improvement in the presenting pathology and sedative weaning should be an aspect of the weaning of other organ support such as ventilatory weaning and CVS support weaning. The co-existence of a tracheostomy may facilitate the sedative wean as the requirement for sedation is much less compared with an ETT.

- In the majority of patients sedatives may be gradually reduced until stopped and the patient will clear the sedatives from their system and begin to lighten.

- In some patients however reduction in the sedatives provokes agitation, dysphoria, delirium, CVS responses, ventilator dysynchrony and other such problems. Even after short periods on sedatives some individuals may develop a relative dependence on the drugs and withdrawal may have to far more gradual.

- Suggested supplemental sedative regimes for sedative withdrawal can include:

  - Regular PO/NG diazepam; gradually titrated. Alternative benzodiazepines include clonazepam.
  - Clonidine infusion or oral/NG clonidine
  - Haloperidol prn
  - Regular oral morphine in de-escalating doses
  - Regular carbamazepine on de-escalating doses

- If agitation and confusion are the key problems always consider potential other causes (see 8.8 ICU delirium).

8.6 Neuromuscular blockade

- The use of neuromuscular blockade in ITU patients has steadily decreased over the past decades with an awareness of the short and long term consequences of its use. This has been facilitated in part by improvements in ventilator technology allowing better patient ventilator harmony.

- There remains a number of indications for neuromuscular blockade in critically ill patients on ICU

  - Short term boluses to facilitate transport of patients or to facilitate interventions
  - Severe respiratory dysfunction with difficulty settling patient on ventilatory support
  - Raised ICP refractory to adequate sedation, consider infusion only if ICP reduction occurs with bolus dose.
  - To counteract shivering during therapeutic hypothermia

- It is vital the patient is adequately sedated prior to paralysis so as to minimise the risk of awareness. **If in doubt about this or the indications seek senior help.**

- For short term (bolus) paralysis the following are suitable

  - Atracurium   0.5 – 1 mg/kg
  - Vecuronium   0.1 mg/kg
  - Rocuronium   0.6 mg/kg
  - Cisatracurium   0.15 mg/kg

- If short term usage is planned and lightening of sedation is also planned ensure the patient has had adequate time for the drug to be eliminated. The profiles for atracurium and cisatracurium are more favourable in this area. If in doubt either maintain sedation or give reversal (neostigmine 0.05 mg/kg (max 5mg) + glycopyrolate 200mcg per 1mg of neostigmine). The disadvantage of reversal is usually tachyarrythmias.

- For longer term paralysis infusions are more appropriate. Any of the above agents can be used to paralyse the patient. Our default drug is cisatracurium due to its favourable elimination profile and low risk of histamine release. Regimes include:

  - Cisatracurium (5mg/ml) at 1-5 ml/h
  - Rocuronium (10 mg/ml) at 300- 600 μg/kg/h (aprox 2- 4 ml/h in 70 kg pt)
  - Atraucurium (10 mg/ml) at 4.5 to 29.5 μg/kg/min (1.8 – 12.6 ml/h in 70 kg pt)
  - Vecuronium (2 mg/ml) at 0.8 to 1.5 μg/kg/min (1.6 ml – 3.1 ml/h in 70 kg pt)

- Regular reviews of the need for neuromuscular blocking agents should be undertaken and every opportunity to reduce or remove them taken

- The long term consequences of NMB agents are
  - Increased rate of VAP
  - Prolonged periods of ventilation
  - Increased incidence of critical illness polyneuropathy (especially with concomitant use of steroids and occurrence of sepsis)

8.7 Epidurals and analgesic regimes on ICU

- The majority of patients are analgesed on ITU with IV infusions of opioids, typically alfentanil or morphine. Patients who have returned from theatre for post-op care (see section 10) may have other methods of analgesia in place such as PCA or epidurals.

8.7.1 <u>Epidural analgesia</u>

- Epidurals in ICU patients are usually thoracic epidurals inserted per-op for post op analgesia. Lumbar epidurals are less often seen but may be in-situ following surgery for trauma or elective procedures for lower limb joint replacements. Full information can be obtained from the UHNS surgical guidelines (p 119-124).

- The advantages of epidural analgesia are good pain relief in the majority of cases, facilitation of physiotherapy and expectoration in abdominal surgery, reduced incidence of DVT and avoidance of systemic opioids.

- The current vogue for epidural analgesia is administration of low dose epidural infusions utilising a mixture of opioids and local anaesthetic. The benefit is of optimal analgesia with minimal motor blockade and minimal sympathetic blockade. This reduces the incidence of hypotension. From a safety perspective the use of lower concentrations of local anaesthesia also minimised the effects of inadvertent overdoses with these agents.

- The standard concentration utilised at UHNS is

  - 0.125 % levobupivicane + 100 µg/ml diamorphine
  - this can be run at 3- 8 ml/h

- in some elderly patients it is appropriate to reduce or omit the diamorphine dosages as respiratory depression can occur.

- If analgesia is inadequate with an epidural **seek anaesthetic or senior assistance if you are not familiar with their usage**. Potential problems may include:

  - Inadequate block height e.g. top of incision not covered. For abdominal surgery a block height of T5 is generally desirable, T2 if a thoracotomy is involved. Require bolus and increased rate e.g. bolus 5 – 10 ml and increase by 2-3 ml/h
  - Inadequate analgesia in area covered by epidural. Consider bolus of more concentrated solution and/or opioid e.g. 10 ml 0.25% levobupivicaine and/or 500 µg of diamorphine.
  - Excessive sedation or respiratory depression. This is most likely due to sensitivity to the opioids. Initially stop infusion, consider naloxone 50 µg increments and reduce the dose of diamorphine in the infusion.
  - Bradycardia is typically due to excessive sympathetic blockade (levels above and including T4). Assess CVS stability. If compromised then stop infusion and give atropine 500 µg titrated to response =/- ephedrine 3-6 mg IV. If not compromised reduce infusion rate and review.
  - Hypotension may be due to the usual causes of fluid depletion, bleeding, excessive sedation or sepsis (see section 2) and/or excess sympathetic blockade. Sympathetic blockade will be generally unresponsive to fluid

boluses which should be the initial strategy. It will require some form of vasopressor, the choice of which will depend largely on the patient and their general condition but may include:

- Ephedrine 3-6 mg IV boluses
- Metaraminol 0.5 – 1 mg IV boluses (note patients may be surprisingly sensitive to this)
- Phenylephrine
- Noradrenaline (as infusion of 4mg in 50 ml (80 μg/ml)) rarely more than a few ml/h unless other conditions involved (e.g. sepsis).

8.7.2   Opioid PCA

- See surgical guidelines p116-117.

- In a proportion of post surgical patients epidurals may be contra-indicated (coagulopathy, severe sepsis, local infections, refusal, anatomical considerations) and an alternative is patient controlled analgesia.
- In addition PCAs may be utilised when weaning post surgical patients from morphine infusions when their cognitive states permit.

- The standard settings on the PCA are
  - 1 mg boluses
  - 5 min lock outs
  - 40 mg 4 hourly maximum
  - via a Graseby 3300 pump
- it is important given the kinetics of morphine that a loading dose is provided: 5-15 mg IV is a typical range.

- Anticipate side effects such as N & V, drowsiness/sedation, respiratory depression and potential retention of urine (although it is unusual for ICU patients not to be catheterised) and prescribe appropriate prn medication (i.e. anti-emetics, naloxone).

8.7.3   Supplemental Analgesia

- In a similar fashion to other post-operative patients ICU patients may require supplemental analgesia to the epidural / PCA methods. Also when weaning from more potent analgesics oral analgesia may be utilised to facilitate withdrawal.
- In general terms the use of the WHO analgesic ladder is acceptable, with the caveat that a proportion of patients have some contra-indications to NSAIDs on ICU. For more detail see surgical guidelines p 105 – 108.

- A typical escalation may involve

  - Paracetamol 1 gram QDS (can be given PO/PR/NG and in selected cases IV)
  - Ibuprofen 400mg TDS
  - Co-codamol (8/500) TDS
  - Co- codamol (30/500) TDS
  - Dihydrocodeine 30-60 mg TDS or codeine phosphate 30-60 mg TDS.

8.8. ICU delirium

8.8.1 Delirium on ICU

- Delirium in ICU patients is (not surprisingly) common, particularly during the phase where sedative regimes are weaned. Its exact incidence is difficult to evaluate as the diagnostic criteria for delirium have varied between studies and the range depends heavily on the patient population studied. Estimates range from 19 to 80 % of patients experiencing delirium at some point in their care.

- Delirium is characterised by acutely changing and fluctuating mental status, inattention, disorganised thinking and altered consciousness, which may or may not be accompanied by agitation. Hallucinations may also occur. Passivity may be more difficult to notice than agitation.

- A validated method for assessing confusion is the CAM-ICU method (Ely 2001). It is detailed for the interest of the reader and is not a tool used on UHNS ICU at present.

    o Delirium is diagnosed when features 1 and 2 are present plus either feature 3 or 4.

    o Feature 1: Acute onset of mental state changes or fluctuating course
        § Assessed by sedation scores, GCS and family / staff observation

    o Feature 2:: inattention
        § Observation or formal assessments

    o Feature 3 : disorganised thinking
        § Observation or formal testing

    o Feature 4: altered level of consciousness
        § Any level of consciousness other than alert would include vigilant, lethargic, stupor, comatose.

- Delirium on ICU has a myriad number of causes:

    o Metabolic disturbances
        o Hepatic failure
        o Renal failure
        o Corticosteroids

    o Physiological abnormalities
        o Hypotension / decreased cerebral perfusion
        o Hypoxaemia
        o Hypercarbia
        o Anaemia
        o
    o Electrolyte imbalances
        o Sodium abnormalities
        o Calcium abnormalities

    o Withdrawal syndromes

- o Sedative withdrawal
- o Alcohol withdrawal

- o Acute infections
  - o CNS infections
  - o Systemic sepsis
  - o Pyrexia

- o CNS disorders
  - o Vascular disorders
  - o Seizures /post-ictal
  - o Encephalopathy (notably septic encephalopathy)

- o Pre-existing problems
  - o Chronic cognitive impairment
  - o Cerebrovacsular diseases
  - o Chronic substance dependence
  - o Nicotine withdrawal

- o Sleep disorders

8.8.2 Management of delirium

- Management of delirium can be difficult in ICU patients due to the multiple causes and variable presentations.

- If any reversible or remediable causes are present address those

- If despite correcting the above the delirium continues consider

  - o Haloperidol 2.5 to 5 mg IV
  - o Midazolam 1-2 mg IV
  - o Morphine 1-5 mg IV (if pain a consideration or respiratory distress)

- In some patients it may be appropriate to use physical restraints e.g. "boxing gloves." Discuss this with senior nursing staff and SpR/ Consultant prior to instituting.

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4. Kress JP, Pohlman AS, O'Connor MF, Hall J. Daily interruption of sedative infusions in critically ill patients undergoing mechanical ventilation. NEJM 2000; 342: 1471-7.
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6. Griffiths R, Jones C. Delirium and confusion: more than ICU syndrome. Critical care focus 2005; 12 : 28-38.
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Section 9   Ethical issues on ICU

9.1 Overview of ethics on ICU

- Ethics on the intensive care unit and the application of ethical principles differs in several ways from that on the general ward. A significant number of our patients are sedated and therefore lack mental capacity and are unable to enter discussions with us about treatment pathways. These patients should be treated on the basis of what would be in their best interests after discussions with friends and families to ascertain patients prior expressed wishes, their beliefs and values and advance decisions if any. If the patient lacks family or friends then the involvement of an Independent mental capacity advocate may be necessary.

- This section aims to provide an overview of the principles in these areas. It is a difficult area and the trainee should seek senior advice at most times.

- Modern medical ethics at institutional level is primarily deontological, following the ethics of a set of principles. The priority of each principle in practice depends on the situation and the clinical setting. There are four key principles / duties:

    - Autonomy
    - Beneficence
    - Non- maleficence
    - Distributive justice

- Autonomy is felt by many to be the over-riding principle in medical ethics. However on ICU patients inevitably have diminished autonomy and as a result the other three principles come to the fore. The duty of beneficence (acting for the good of the patient) becomes one of the key duties and this is referred to as (soft) paternalism. The duty of non-maleficence, that of doing no harm, tends to come into discussion when considering the benefits versus harm of ongoing treatments and in consideration for withdrawal decisions.

9.2 Withdrawal of treatment   see also Trust Policy C13

- Decisions to withdraw treatment are made at senior levels but the junior trainee will benefit from knowledge of the ethical and legal considerations and knowledge of the process of withdrawing treatment.

- The decision to withdraw is a clinical one but is made in consultation with families. This is not just a point of common courtesy. Legally if a family object to withdrawal of treatment the issue can escalate to the courts which
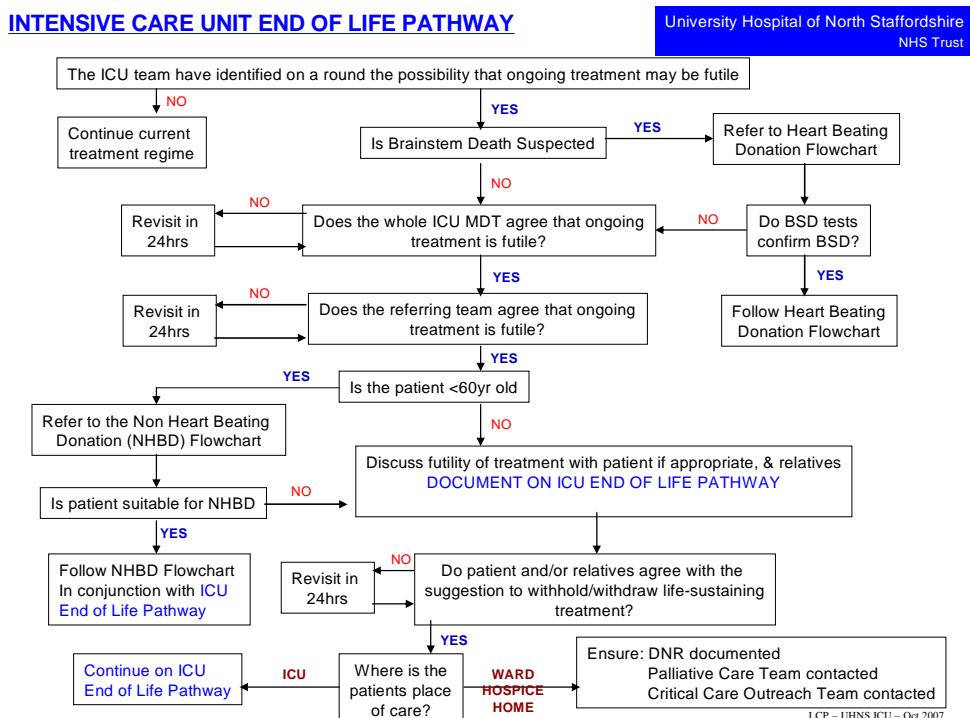
may prolong the process markedly. It is important however to engage the family in the process but to make it clear the decision is a clinical one so as to avoid them feeling responsible for the death of their relative.

- Ethically the justification for withdrawing life sustaining treatment revolves once more in the four duties noted above. If a continuing treatment offers no benefit to the patient, namely it is *futile* then in all likelihood it constitutes a *harm*. This can be rationalised with ventilation: IPPV may involve infections, discomfort, repeated suctioning etc. On the principle of "do no harm" this can be seen to be untenable. Hence death can be considered preferable to suffering without prospect of improvement.

- Thus it is when the patients condition renders ongoing treatment futile and burdensome that withdrawal is considered. Inevitably this is based on both opinion and evidence and is a complex decision.

- Legally the position remains essentially unchanged sine the case of Tony Bland. It has been reiterated in case law and in a recent challenge to the GMC guidance by Lesley Burke. Physicians are not obliged to provide treatment that they feel is inappropriate. Stopping life sustaining treatment does not constitute manslaughter because it is regarded in law as an omission to continue treatment rather than an act to stop it.

- The recent case of Ann David (ongoing with the GMC) highlights the difficulties that can arise when withdrawal is allegedly performed badly. To this end it is worth understanding the process of withdrawing treatment, although this will vary between consultants.

- One method of treatment withdrawal is:

- Most cases will be sedated when the decision is made. If not one should consider the need for sedation/ analgesia for patient comfort and commence this prior to altering other agents (such as inotropes)

  o The choice of sedative varies: all are acceptable.
  o If de novo many consultants will start diamorphine (25 mg in 50 ml @ 1-10 ml/h) or morphine. This is because the respiratory suppressant nature of the agents will ensure comfort when hypoxia and hypercarbia ensue.

- Once comfort is ensured the family are typically invited into the area with curtains drawn. The monitor alarms should be silenced and the monitor ideally easily accessible to the ITU staff to ensure minimal disturbance.

- Families should be aware that the process may range from very rapid to a number of hours.

- Ventilatory support is discontinued in a variety of ways. The choice depends on quality of spontaneous ventilation.
  - ETT remains, patient continues to be ventilated, placed on FiO2 of 0.21 and rate turned down and alarms re-set. Disadvantage is that the ventilator will continue after death and will need to be switched off.
  - ETT remains, patient disconnected from ventilator and placed on Swedish nose at ETT with no oxygen flow. Alternatives such as CPAP circuits or anaesthetic bags can be used but involve some extra fresh gas flow and thus more than "room air"
  - ETT removed. It is advisable to inform family that following extubation upper airway sounds similar to snoring can occur. This can be distressing to relatives but unlikely to cause the patient discomfort. Every effort to alleviate pain, discomfort, nausea and restlessness should be made to maintain patient comfort and dignity.T

- Inotropic/ vasopressor support is discontinued. Stop all medication other than those necessary for comfort.

- Opinion on whether nutritional support or fluids should continue varies but is unlikely to be a key issue in ICU withdrawals. If in doubt discuss with consultant.

- A good resource is the document produced by the Intensive care society [ref 1].

If the decision for palliative care / withdrawal is made and death is not imminent we are following the algorithm of the "Liverpool Care Pathway". From that moment on the LCP form replaces all other medical ICU documents for the further course. The LCP form contains treatment algorithms for pain control, nausea, dyspnoea and restlessness as well.

INTENSIVE CARE UNIT END OF LIFE PATHWAY

University Hospital of North Staffordshire
NHS Trust

The ICU team have identified on a round the possibility that ongoing treatment may be futile

NO → Continue current treatment regime

YES → Is Brainstem Death Suspected

YES → Refer to Heart Beating Donation Flowchart → Do BSD tests confirm BSD?

NO → Does the whole ICU MDT agree that ongoing treatment is futile? — NO → Revisit in 24hrs

NO → Does BSD tests confirm BSD? — NO → Does the whole ICU MDT agree that ongoing treatment is futile?

YES → Follow Heart Beating Donation Flowchart

Does the referring team agree that ongoing treatment is futile? — NO → Revisit in 24hrs

YES → Is the patient <60yr old

YES → Refer to the Non Heart Beating Donation (NHBD) Flowchart

NO → Discuss futility of treatment with patient if appropriate, & relatives DOCUMENT ON ICU END OF LIFE PATHWAY

Is patient suitable for NHBD — NO →

YES → Follow NHBD Flowchart In conjunction with ICU End of Life Pathway

Do patient and/or relatives agree with the suggestion to withhold/withdraw life-sustaining treatment?

NO → Revisit in 24hrs

YES → Ensure: DNR documented Palliative Care Team contacted Critical Care Outreach Team contacted

Where is the patients place of care?

ICU → Continue on ICU End of Life Pathway

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9.3 Organ donation on ICU

- Full guidance on the trust policy on organ donation can be found on the Trust intra-net via http://uhns/clinical section/ organ and tissue donation. The key policies are C55 and C13. The site contains extensive literature on all aspects on tissue and organ donation.

- The ICU has extensive involvement with the Transplant coordinators and they are very approachable and available for advice. With the advent of non-heart beating donation in the trust it is likely they will become even more involved in the unit.

- Contact details are

    - On call donor transplant co-ordinator 07659 137 821 (a 24 hour pager)
    - Regional donor transplant coordinator 0121 462 1311

- At present donation is discussed with families at time of Brain Stem Death Testing, either prior to first set or between sets. Changes in legislation give families less scope to object if the potential donor had previously expressed desire to donate (evidenced by donor card or placement on a national register).

- A good resource is the UK transplant website www.uktransplant.org.uk

- For discussion on confirmation of brain stem death see section 6.

- If a decision is made the patient is for organ donation then a process commences of arranging recipients, coordinating teams, arranging theatres and anaesthetists and switching the focus of care of the patient (donor) to that of organ preservation. Advice will be provided by the coordinator but is detailed below:

9.4 Brain stem death testing

- Although trainees will not be expected to perform brain stem tests it is useful to know the current practice. On our unit the usual practice is to have an intensive care consultant and a member of the neurosurgical team to perform the tests. In the unusual scenario that the patient is not a neurosurgical patient a senior SpR form the intensive care team is appropriate.

- Personnel

    - Two testers (may test separately or simultaneously). One must be a consultant and the other a doctor of at least 5 years post-registration standing.

- - - Tests are performed twice (to remove the risk of observer error). The gap between the tests is discretionary.
    - Death is pronounced at the completion of the second set. *Legal time of death is the time of the first set.*

- Criteria

  - - Irremediable brain damage of known aetiology
    - Patient is deeply unconscious
      - § Confounding causes must be excluded (see exclusions)
    - Patient is ventilated

- physiological preconditions and drug levels for valid brain stem tests as per "Code of Practice for Diagnosis and Confirmation of Death" (Med. Royal Colleges 2008)

  - § Temp. > 34.0 °C
  - § MAP > 60 mmHg
  - § pO2 $\geq$ 10 kPa
  - § pCO2 $\leq$ 6 kPa
  - § pH 7.35 – 7.45
  - § Na 115 – 160 mmol/L
  - § K > 2.0 mmol/L
  - § Mg and Phosph. 0.5 – 3.0 mmol/L
  - § Glucose 3.0 - 20.0 mmol/L
  - § Drug levels:
      Thiopentone: < 5 mg/L
      Midazolam : < 10 mg/L
  use Flumazenil and Naloxone, if in doubt about opioid and benzodiazepine effects

- Components of test

  - - Cranial nerves
      - § Pupils fixed
      - § No corneal reflex
      - § Absent vestibulo-ocular reflex (50ml ice cold water over one minute into EAM with head flexed to 30 degrees. Ensure clear EAM)
      - § No motor response to pain in any area within cranial nerve distribution. No limb response to supra-orbital pressure.
      - § No gag reflex or response to tracheal suction

§ The Apnoea Test:

1. Hypoventilate with 100 % O2 until pCO2 $\geq$ 6.0 kPa and pH < 7.40

2. Take patient off ventilator with 5L/min O2 flow via tube (or CPAP device if necessary) and watch 5 min for respiratory efforts

9.5 Care of the potential organ donor

- Once brain stem death has occurred a process of gradual organ system shutdown begins. This process varies in its time frame and is unpredictable. As a result if the patient is to donate their organs it is a priority to (a) support and sustain the organs and (b) be aware of the potentially narrow time frame for organ viability

- Various guidelines exist which detail the care of the potential donor and these are referenced below [4-5].

- Typical features seen in patients post-brain stem death are [5]

  - Hypotension (81%)
  - Diabetes insipidus (65%)
  - DIC (28%)
  - Cardiac arrythmias (27%)
  - Pulmonary oedema (18%)
  - Metabolic acidosis (11%)

- General good supportive care is the backbone of supporting the donor organ systems. However there are specific issues related to potential donors that if managed aggressively will improve organ viability (by up to 60% in some studies).

- Particular care is

  - Ventilation   lung protective strategy (Tv 6-8 ml/kg, adequate PEEP) Aim for PaO2 > 10 kPa. Minimise FiO2 if possible.

  - Circulation   prone to pulmonary oedema; keep CVP ~ 6 mmHg if lungs being considered and CVS state permits. CVP 10-12 mmHg if not. CI > 2.2-2.5 if measured. MAP > 70 mmHg.
  Wean NORAD if present and start vasopressin infusion as pressor (range 0.5 to 2.5 U/h)
  Routine management of arrhythmias

  - Metabolic   consider intermittent DDAVP if diabetes insipidus Evident (high Na and polyuria). Infusions of 5% dextrose or NG water may be required in some cases.

  - Haematological
  Normal transfusion practices apply. Correct coagulopathy.

Resources and references

1. http://www.ics.ac.uk/icmprof/downloads/LimitTreatGuidelines2003.pdf
2. www.uktransplant.org.uk
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4. http://www.uktransplant.org.uk/ukt/about_transplants/donor_care/policy_documents/ICS_guidelines_for_adult_organ_and_tissue_donation_chapter_5(nov2004).pdf
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6. A Code of Practice for Diagnosis and Confirmation of Death. Medical Royal Colleges, Oct. 2008

Section 10 Transfer of critically ill patients

Guidelines for the Transfer of Adult Patients with Serious Head Injury and Other Emergency Neurosurgical Conditions
The following provisional guidelines have been developed by the Neurosciences Unit at the University Hospital of North Staffordshire (UHNS) in conjunction with colleagues from the Acute Hospitals in Shropshire and South Staffordshire. They have been accepted by the Staffordshire and Shropshire Critical Care Network.

National Guidelines
The following national guidelines should be followed in the local Neurosurgical System:

- Recommendations for the Safe Transfer of Patients with Brain Injury, Association of Anaesthetists of Great Britain and Ireland (updated 2006)
- Transfer Guidelines, Intensive Care Society (updated 2002)
- Head Injury Guidelines, National Institute for Clinical Excellence (2003)
- Report of the Working Party on the Management of Patients with Head Injuries, Royal College of Surgeons of England (1999)

Existing Local Guidelines for the Transfer of Patients with Acute Life-threatening Intracranial Space-occupying Lesions
This was established on 12/01/2001 and was updated on 18/08/2006 (see Appendix 1).

Overview of Time Frame
The following time frame relates to head injuries, but similar considerations apply to other neurosurgical emergencies.

- Arrival in primary hospital from scene (usually within about ¾ hour)
- Head CT scan within 1 hour of arrival (or within 1 hour of deterioration if not initially compromised) – see *indications for emergency CT scan*, *spinal imaging guidelines*
- Referral within ¼ hour of CT scan – see *indications for emergency neurosurgical consultation/referral*
- Reply back from neurosurgical team within ½ hour
- Preparing for transfer (generally already started as transfer will generally have been anticipated and simply transferring to the scanner requires similar preparation) – further ½ hour
- Transfer to Neurosciences Unit – about 1 hour

With this time frame, the total anticipated time from the pre-hospital emergency call to arrival in the Neurosurgical Unit will be less than 4 hours. While this is generally acceptable, it may not be so in some emergency situations. Every attempt should be made to reduce the time delay in life-threatening situations. It may be appropriate to dispatch some head-injured patients directly to the Neurosciences Unit, bypassing the Primary Hospital, on the basis of the clinical findings at the scene.

Detailed Arrangements

1. A Head CT scan should be obtained within an hour of arrival for serious head injuries with an altered conscious level or within an hour of deterioration if not initially obtunded. Consultation with/referral to the Neurosciences Unit should take place within 15 minutes of the CT scan being performed. The CT images should be transmitted as soon as possible to the Neurosciences Unit, before consultation/referral takes place.

2. A minimum of a Specialist Registrar/ Staff Grade (or Consultant) in Emergency Medicine, Surgery, Orthopaedics, Anaesthetics, Intensive Care (depending on local arrangements), who is caring for the patient at the time, should consult with/refer to the on-call Specialist Registrar/Staff Grade in Neurosurgery.

3. If the on-call Specialist Registrar/Staff Grade in Neurosurgery is unable to give a prompt opinion (e.g. is scrubbed in the Operating Theatre), the on-call Consultant Neurosurgeon should be contacted. The Consultant Neurosurgeon should then decide whether the opinion can wait until the on-call Specialist Registrar/Staff Grade in Neurosurgery is available to view the CT scan, whether the patient should be transferred anyway (without waiting for the scan to be seen), or whether the Consultant Neurosurgeon should come in to view the scan.

4. The reply back from the on-call Specialist Registrar/Staff Grade in Neurosurgery after viewing the transmitted images and discussing the case with the on-call Consultant Neurosurgeon should be within 30 minutes. In cases that are likely to require transfer to the Neurosurgical Unit, the availability of a suitable bed should be checked within this time. During the reply back, the on-call Neurosurgeon should agree an initial management plan with the referring clinician.

5. If transfer is agreed to be appropriate and a suitable bed is currently available in the Neurosciences Unit, transfer should be arranged. In general, trauma cases will be transferred to the Emergency Department at the University Hospital of North Staffordshire. Other emergency neurosurgical cases may be transferred to the Emergency Department, the Multiple Injuries Unit (a Level-3 facility that includes Neuro-Critical Care beds), the Neurosurgical Special Care Unit (a Level-2 Neurosurgical High Dependency Unit), the Neurosurgical Ward, the Paediatric Intensive Care Unit or a Paediatric Ward, depending on the patient's needs. Occasionally the transfer may be directly to the Operating Theatre. The Neurosurgical Specialist Registrar/Staff Grade should inform the receiving area of the expected case and the transferring team should provide the receiving area with an expected time of arrival when leaving the Primary Hospital.

6. If transfer is agreed to be appropriate, but no suitable bed is available, transfer may still take place providing that the case meets the criteria in the *Guidelines for the Transfer of Patients with Acute Life-threatening Intracranial Space Occupying Lesions*. These guidelines should then be followed.

7. If transfer is agreed to be appropriate for transfer, but no suitable bed is available in the Neurosciences Unit and the case does not meet the criteria in the *Guidelines for the Transfer of Patients with Acute Life-threatening Intracranial Space Occupying Lesions*, the referring team in the Primary Hospital should identify an available bed in the nearest alternative Neurosciences Unit by contacting the Emergency Bed Service and then contacting a unit that has declared free beds.

[*Proposed Alternative*: If Transfer … *Space Occupying Lesions*, the neurosurgical team should identify an available bed in the nearest alternative Neurosciences Unit by contacting the Emergency Bed Service and then contacting a unit that has declared free beds. The Neurosurgical Specialist Registrar/Staff Grade (or Consultant) should refer the patient to the alternative centre.] … *continue as alternative below*.

8. The referring Specialist Registrar/Staff Grade in the Primary Hospital should then refer the patient to the alternative unit, explaining that the University Hospital of North Staffordshire considered transfer to be appropriate. The name of a Neurosurgical Specialist Registrar/Staff Grade (or Consultant) at the University Hospital of North Staffordshire (who has seen transmitted films) should be given to the alternative unit to allow the CT findings to be discussed neurosurgeon-to-neurosurgeon, if the alternative unit cannot receive transmitted images from the Primary Hospital. It should be noted that the neurosurgical management policies may vary from unit to unit: the alternative Neurosurgical Unit may advise a different management plan from that agreed with the Neurosurgical team at the University Hospital of North Staffordshire.

[*Continuation of alternative above* … When a neurosurgeon at an alternative centre has provisionally accepted the patient, he or she contacts the original referring clinician at the Primary Hospital for an update with any further clinical details and, if agreed, to accept the patient for transfer.]

9. The Neurosurgical Specialist Registrar/Staff Grade (or Consultant) at the University Hospital of North Staffordshire should remain available for ongoing advice in any changing situation before the referral has been accepted by alternative unit.

10. The on-call Consultant Anaesthetist in the Primary Hospital should be informed of any planned emergency transfer to the Neurosciences Unit that requires an Anaesthetic escort, before transfer takes place. The duty Consultant responsible for head injuries in the Primary Hospital (or the Consultant responsible for the patient's overall care) should be informed of any emergency transfer to the Neurosciences Unit.

11. A transfer form and audit form should be completed for all transfers. Copies of both should be sent to the Critical Care Network Coordinator for collation.

12. The on-call Consultant Neurosurgeon should be informed of any planned emergency transfer to the Neurosciences Unit before transfer takes place. The Consultant Intensivist in the Neurosciences Unit should be informed of any planned emergency transfer to the Neurosciences Unit before transfer takes place, if the patient is currently receiving or will require intensive care. The Third On-call Anaesthetist in the Neurosciences Unit should be informed of any planned emergency transfer to the Neurosciences Unit before transfer takes place, if the patient will need emergency surgery on or soon after arrival.

13. If a patient with a serious head injury to be transferred to the Neurosciences Unit has other serious injuries, the appropriate specialists in the Primary Hospital should contact their counterparts in the University Hospital of North Staffordshire to inform them of these injuries and to plan future management.

Supporting Arrangements
Coordinated CT scanner links between the hospitals
An effective CT scanner link between the Neurosciences Unit and the surrounding hospitals must be maintained at all times.
Standard transfer records
A standard referral form, ideally electronic, should be used to document timings of the communication between the Primary Hospital and the Neurosciences Unit, the advice given and the agreed management plan. The standard Critical Care Network transfer record should be used during the transfer itself.
Transfer training programme
Personnel undertaking critical care escort duties should have received specific training in inter-hospital transfer, either on a recognised national course (e.g. Safe Transfer and Retrieval – STaR) or on a customised local hospital or network course.
Defined consultant responsibility
There should be a nominated Consultant responsible for transfers in each Primary Hospital and in the Neurosciences Unit, together with the provision of resources to allow the responsibilities to be fulfilled.
System meetings
A programme of system meetings should be scheduled to allow constructive feedback of consultations made, transfers undertaken, transfers refused and any adverse incidents relating to transfers, together with the opportunity to revise the guidelines accordingly.

Appendix 1: Guidelines for the Transfer of Patients with Acute Life-threatening Intracranial Space-occupying Lesions

In the following circumstances, the Consultant Neurosurgeons are empowered to accept an emergency transfer from those hospitals in the Transfer Group for which the Neurosurgical Service is specifically responsible (i.e. Stafford, Telford, Shrewsbury, Oswestry, Leighton), **even if there is no available intensive care bed** in the University Hospital of North Staffordshire, provided that an appropriately staffed and equipped operating theatre can be made available for emergency neurosurgical intervention:

Acute life-threatening intracranial space-occupying lesions:

- Which are predicted to deteriorate rapidly with a high risk of death or disability unless treated surgically without delay
- Which are judged to be sufficiently urgent to transfer straight to the operating theatre for life-saving surgery before admission to a ward
- In which a favourable outcome is potentially achievable with prompt surgical treatment.

In all other circumstances, the availability of an intensive care bed must be established before accepting a transfer from another hospital with a neurosurgical emergency which is likely to need intensive care.

If the outcome is considered to be so poor that transfer for acute neurosurgical intervention is futile, the Consultant Neurosurgeon may refuse the patient, irrespective of bed availability. Advice on continuing care or withdrawal of care should still be provided to the referring clinician.

Whenever a transfer is agreed for emergency neurosurgical intervention without a guaranteed intensive care bed, the on-call manager should be informed immediately and work with the Consultant Neurosurgeon and Consultant Intensivist to facilitate arrangements for providing a suitable bed post-operatively and to contribute to the audit of the individual case.

If a patient is transferred for emergency neurosurgical intervention without a guaranteed intensive care bed and still requires an intensive care bed post-operatively, the following options should be considered:

- Admission into an intensive care bed in the University Hospital of North Staffordshire, if one has by then become available
- Transferring the patient out into the nearest available neurosurgical intensive care bed
- Transferring out another, more stable intensive care patient into the nearest appropriate intensive care bed, if the patient is too unstable for transfer.

Appendix 1 guidelines updated 18/08/2006 (Jag Singh, Peter Oakley, Simon Ellis)

Appendix 2: Referral and Transfer Checklists
Indications for emergency Head CT scan
Refer to:

- Head Injury Guidelines, National Institute for Clinical Excellence (2003)
- Report of the Working Party on the Management of Patients with Head Injuries, Royal College of Surgeons of England (1999)]

Indications for emergency neurosurgical consultation/referral
Refer to:

- Head Injury Guidelines, National Institute for Clinical Excellence (2003)
- Report of the Working Party on the Management of Patients with Head Injuries, Royal College of Surgeons of England (1999)]

Guidelines for cardio-respiratory support during head injury transfer
Refer to:

- Recommendations for the Safe Transfer of Patients with Brain Injury, Association of Anaesthetists of Great Britain and Ireland (updated 2006)
- Transfer Guidelines, Intensive Care Society (updated 2002)

Local variation (Neurosciences Unit, University Hospital of North Staffordshire):

- Keep $p_aO_2 > 13$ kPa (relaxation to 10 kPa under consideration)
- Keep mean arterial pressure > 80 mm Hg when raised intracranial pressure (ICP) suspected and not (yet) measured
- Keep cerebral perfusion pressure > 60 mm Hg (when ICP available)

Other recommendations for system support during transfer

Refer to:

- Recommendations for the Safe Transfer of Patients with Brain Injury, Association of Anaesthetists of Great Britain and Ireland (updated 2006)
- Transfer Guidelines, Intensive Care Society (updated 2002)
- Head Injury Guidelines, National Institute for Clinical Excellence (2003)
- Report of the Working Party on the Management of Patients with Head Injuries, Royal College of Surgeons of England (1999)