ST. GEORGE'S HOSPITAL

AN INTRODUCTION TO THE

GENERAL INTENSIVE CARE UNIT

Version May 2010

WELCOME TO

ST GEORGE'S HOSPITAL

GENERAL INTENSIVE CARE UNIT

A Quick Who's Who

Consultants
Mike Grounds
Phil Newman
Greg McAnulty
Barbara Philips
Andy Rhodes
Tony Rahman
Jonathan Ball
Mark Hamilton
Maurizio Cecconi

Administrative Staff
Shanti Singh
Sue Brooks
Dorothy Thompson

Senior Nursing Staff
Deborah Dawson
Lindsey Izard
Susan Reynolds
Mark Russon

Lead Physiotherapists
Olive Wilkinson
George Momcilovic

Pharmacist
Joanne Peh

Senior Technician
Sarah Piper

Consultant Microbiologists
Aodhan Breathnach
Rick Holliman

Dietician
Catherine Collins

General Manager
Stephen Race

Where we are and how to make contact

General Intensive Care Unit
1st Floor, St James's Wing
St George's Hospital
Blackshaw Road
London SW17 0QT

Tel: 020 8725 1307
Fax: 020 8725 3296

For Hospital bleeps: 88 / blp no. / ext no.
For Aircalls (SG): dial #6321 or 07699 119700 or switchboard on 1000

The GICU SpR pager is 7980

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WELCOME TO THE UNIT

If this is your first exposure to intensive care you may find the environment a bit daunting. The clinical workload, vocabulary, technology and pace of working may all seem a bit alien. Please don't be put off and feel free to approach us at any time if you're having difficulties.

Your backgrounds will be different but we work as a multidisciplinary team. We also don't know what experience you have. You are likely to be asked to do things you either can't do or need help with. We all know and expect this so please don't be afraid to point it out to us. Most importantly, "If in doubt, ASK."

There is a very friendly and relaxed atmosphere within the Unit. There are no hierarchical or inter-professional boundaries. Decisions are generally made by consensus and everybody is entitled and encouraged to voice an opinion.

This book is our attempt to create a core of information necessary to help you through your first few days and weeks. It is not intended to be a text book of intensive care nor dogmatic. This guide is an evolutionary document and we welcome all feedback regarding its content.

As some of us are getting on a bit and all of the juniors wear scrubs, we ask you to present yourselves to Dr Ball's photo booth in Office 2, for your photo to be taken and then displayed in the seminar room. That way we have no excuse for forgetting who you are.

We hope you enjoy your time with us.

Tony Rahman & Jonathan Ball
On behalf of the ICU consultants

INTRODUCTION

Critically ill patients are those with acute, severe and potentially life threatening organ dysfunction and / or failure. Successful care of such patients requires a broad understanding of physiology, clinical medicine and surgery. Attention to detail and good communication skills are also essential. The critical care environment provides continuous monitoring, organ supportive therapies and a high staff to patient ratio. End of life care is often provided in this environment and requires complementary skills.

Good advice, "When initially faced with a critically ill patient, assume nothing, believe no one, give oxygen." (Origin unknown)

Table 1: LEVELS OF DEPENDENCY
A nomenclature describing the level of patient dependency is detailed below:

Level 0	Patients whose clinical needs are met by general ward care.
Level 1	Patients at risk of deterioration on a general ward who require critical care input.
Level 2	Patients requiring intensive observations and/or single organ support. (high dependency care)
Level 3	Patients requiring advanced respiratory support alone or ≥2 organ support (intensive care).
HDU	High Dependency Unit (HDU) – Is an area where patients requiring more intensive observation than can be provided on a normal ward are cared for. There may be some provision of organ support but generally not invasive ventilatory support. HDU's will usually have nurse to patient ratio of 1:2
ICU	Intensive Care Unit (ICU) – Is an area for treatment of organ dysfunction / failure often including invasive ventilatory support. ICU's will usually have a nurse to patient ratio of 1:1

SEVERITY OF ILLNESS SCORING SYSTEMS

A number of severity of illness scoring systems exist. They vary in terms of their complexity and applicability to particular patient groups. Most can be used to generate estimates of ICU mortality for a population of similar patients but cannot predict outcomes in individual cases. All are complex algorithms based on physiological patient data. They have been used to monitor and compare the performance of individual ICUs but their accuracy remains controversial. Below is a list of the more commonly encountered systems.

- Acute Physiology and Chronic Health Evaluation II (APACHE II) [1]. Originally published in 1985, this system rates the most deranged values in the first 24 hours of ICU admission, of 12 physiological variables. The total score is added to two further scores for age and chronic organ insufficiency. The original algorithm has been modified by a number of investigators, in particular, by the UK Intensive Care National Audit and Research Centre (ICNARC).
- APACHE III [2]. This is a proprietary system published in 1991 by the APACHE investigators. It added additional variables to the APACHE II dataset and their algorithm has continued to be developed by them. It is best validated in the United States.
- Simplified Acute Physiology Score II (SAPS II) [3]. This European and North American scoring system was published in 1993. It is significantly simpler than the APACHE III system but its applicability has been questioned.
- Sequential Organ Failure Score (SOFA) [4]. This system was devised in 1996 not as a mortality prediction score but a simple daily score that could accurately track severity of illness. Although the higher the SOFA score the worse the outcome, there is a much stronger association with poor outcome and a static or increasing score.

THIS UNIT

- The General Adult Intensive Care Unit at St George's has 17 beds.
- Of these, 11 are located in the so-called ICU and 6 in the HDU.
- At full capacity we can stretch to 17 level 3 patients. Ideally, we run with 10 level 3, 6 level 2 and one emergency level 3 bed.
- We have close working relationships with the Cardiothoracic and Neuro Intensive Care units.

NURSING STAFF

- The nursing staff do 12.5-hour shifts (i.e. 2 per 24 hours).
- Their handover takes place at 7.45 and 19.45 in the coffee room.
- Each shift has a designated leader, with whom you must liaise regarding any admissions, transfers, discharges etc.
- Any decisions regarding a patient's management MUST ALWAYS be passed onto the nurse looking after that patient.
- The nurses on the unit, like the doctors, range from the "new to ICU" to the very experienced. Please work WITH them. They are an excellent source of information and are very helpful.

Table 2: DAILY TIMETABLE

08:00	Business Ward Round with night shift SHO, on-coming on-call SHO and SpRs. Decisions regarding discharge from GICU and prioritising elective surgical admissions are crucial. The remaining SHOs should start clerking patients. Please book any procedures / scans as early as possible.
10:30	Multidisciplinary, Consultant led, Ward Round. (Coffee and biscuits). Sit down (Seminar room) ward round followed by "tour of the unit." Please ensure the blood results are documented for this WR. Please ensure all plans are handed over to nurses at bedside. Please complete the "Ward Watcher" diagnostic codes during this WR.
17:00	Evening Ward Round: handover for on-call Consultant / Registrar / SHO
20:00	Handover Ward Round for Night Shift SHO / Registrars
23:30	Telephonic ward round with on call consultant

Table 3: SHIFT PATTERNS

SHOs	*Registrars*
"Normal Day" 08:00 – 17:00	1 in 6 - "Reg of the week" 08:00-18:00 Monday-Friday
1 in 8 - "Long Day" 08:00 – 20:00 followed by 08:00 – 13-30 followed by day off	1 in 8 - "2 long days" (Monday - Thursday) 09:00-21:00 **followed** by 2 "Night shifts" 20:00-09:00
1 in 8 - "Week of nights" 20-00 – 08:00. Begins Friday. 2 weeks off at end, which encompasses annual leave entitlement	1 in 8 "Weekend days" (Friday - Sunday) 08:00-21:00 and "Weekend nights" (Friday - Sunday) 20:00-09:00. 18 in 8 week cycles - "Short day" (Monday-Friday) 08:00-18:00

MINIMUM STAFFING LEVELS / LEAVING THE UNIT

- Monday to Friday, office hours, there should be a minimum of 2 SpRs and 3 SHOs on duty.
- There should be at least one doctor on the unit at all times.
- Out of hours, the on call SpR must inform the SHO and the nurse in charge if they are leaving the unit and ensure they can be contacted in an emergency.
- The on-call SpR should carry bleep 7980.

ANNUAL AND STUDY LEAVE

- Any leave taken outside of fixed days off, must be agreed with and covered by your SHO / SpR colleagues.
- Difficulties with leave should be discussed in the first instance with Dr. Barbara Philips or the consultant in charge of the unit at the time when the problem occurs.

SICKNESS

- If you are sick and unable to come on duty, please contact the Intensive Care Unit at the earliest opportunity. On return to work a 'sick leave' form must be completed, which will then be forwarded to medical staffing.

TEACHING

- The daily multidisciplinary, consultant led ward round is a teaching ward round.
- If there are any procedures or pieces of equipment that you would like teaching on, please ask any senior member of staff.
- The hospital Grand Round is on Thursday lunchtime – 12-45hrs in the Monckton Lecture Theatre.
- There is an eLearning environment and eNotice-board for all trainees. This runs on the Medical School Moodle server. Please email Jonathan Ball (jball@sgul.ac.uk) with Moodle in the subject line to gain access and for further information.
- On Thursdays at 08:30, there is a teaching presentation by one of the trainees. These sessions are co-ordinated and chaired by Mike Grounds.
- Jonathan Ball is the clinical and educational supervisor for foundation year 1 & 2 doctors.
- Barbara Philips, Phil Newman and Mike Grounds are the local, regional and national advisors respectively, for the Intercollegiate Board for Training in Intensive Care Medicine.
- Barbara Philips is an examiner of the UK diploma in ICM.
- Mike Grounds, Barbara Philips, Andy Rhodes, Tony Rahman, Jonathan Ball and Maurizio Cecconi are examiners for the European diploma in ICM.
- Jonathan Ball is collaborating with the Cardiff University on their MSc in Critical Care.
- Please register and discuss any audit or research projects via the forums on Moodle.

GICU INFECTION CONTROL POLICY

Infection Control Guidance for preventing cross infection and for personal protection for the staff

Hand washing is the most important aspect of preventing cross infection between patients.

- Hands should be washed or gelled before and after leaving a patient bed space.
- **After any type of bowel care, hands must be washed not gelled, as gel is not effective against diarrhoea causing organisms.**
- Gloves and aprons are NOT to be worn all the time, but used appropriately.
- Once past the chart table in each bed space, all staff to be 'naked below the elbows', including watches and all rings. If staff are unable to comply they should completely cover all such areas with gloves +/- gowns.
- Gloves and aprons to be worn if coming into contact with any body fluid.
- Gowns to be worn if carer's upper body might come into contact with patient, if for example turning or washing.
- **Group regular individual patient care into clean or dirty, carrying out clean procedures first, reducing the times gloves need to be changed.**
- **Gloves, aprons or gowns to be changed between patients.**
- **Goggles to be worn at all times when dealing with bodily fluids.**
- Gloves, aprons or gowns to be removed and hands washed and gelled before leaving the bed space. Unless taking bedpans, catheter bags, drains etc to the sluice.

GUIDELINES FOR THE INITIAL* AND DAILY PATIENT ASSESSMENT RECORD

- Patient's name & age
- Number of days on the unit
- Principal referring team and other teams involved
- Brief history & update (comprehensive, chronological, concise)
    - Main diagnosis
    - Reason for ICU admission including details of injuries / surgical procedures
    - **Current Problems / Progress over last 24 hours (including results of any imaging / investigations / procedures)**
    - *Relevant co-morbidities**
    - *Pre-morbid level of function**
- Examination Record (A brief summary of positive findings, important negatives and physiological TRENDS. For guidance see following pages)
- Details of all current therapies including – organ support, medications, fluids and nutrition. Consideration should be given to stopping / restarting any of the patient's chronic medications.
- Details of all communications with next of kin and specialist teams.
- **The Plan.** This should include physiological / biochemical targets, investigation requests, and any other instructions. Relevant details must be communicated to the nurse caring for the patient and recorded on the paper observation chart at the bed space.

Current problems / Progress and **The Plan** are the most important elements. These should be revised during the 10:30 sit down ward round. The successful implementation of **The Plan** MUST be reviewed at regular intervals and amended as necessary. All amendments MUST be clearly documented including the rationale.

** Obtain this information and document it clearly at the earliest opportunity. Seek a co-lateral history from all available sources including the patient's GP.*

Table 4: The clinical parameters that should be considered as a part of the complete assessment of a critically ill patient. Part 1

Values are important. TRENDS are more informative.

General	End–of-the-bed-o-gram / "What would the cleaner notice?"
Cardiovascular	Heart rate and rhythm Right atrial (RA) / central venous (CV) pressure Pulmonary artery pressures Systemic pressures, including mean arterial pressure (MAP) Cardiac output/index (CO/CI) Oxygen delivery (DO_2/DO_2I) Mixed or central venous oxygen saturation (SvO_2/$ScvO_2$) Haemoglobin and lactate levels Vasoactive drug infusion rates Positive examination findings Relevant 12 lead electrocardiograph (ECG), echocardiogram and angiogram findings
Respiratory	Nature and age of airway management Positive examination findings including site and state of any chest drains Mode of ventilation (if assisted) duration (days), settings, measurements and trends: • FiO_2 • Tidal volumes (Vt) • Peak, plateau, and mean airway pressures • Positive end expiratory pressure (PEEP) • Breath timing [inspiratory:expiratory (I:E) ratio] Recent arterial blood gas (ABG) result *OR* SpO_2 and $ETCO_2$ Weaning regime (if prescribed) Nature and duration of all inhaled / nebulised therapy. Relevant findings from chest x-ray (CXR) series.

Table 5: The clinical parameters that should be considered as a part of the complete assessment of a critically ill patient. Part 2

	Values are important. TRENDS are more informative.
Renal	Maintenance intravenous fluid regime.
	Last 3 hours urine output or renal replacement regime [haemofiltration (CVVHF) or dialysis (iHD)], including anticoagulation prescription and relevant blood results.
	Previous day's fluid balance.
	Trend of blood urea and creatinine.
	Abnormalities in blood sodium and any other relevant electrolyte results.
Gastro-intestinal	Abdominal examination, including site and output of any drains.
	Mode of feeding: naso-gastric(NG), naso-jejunal (NJ), gastrostomy and / or parenteral.
	Position of distal tip of NG tube and aspirate volumes.
	Nature and duration of prokinetics, if any.
	Nature and duration of stress ulcer prophylaxis.
	Bowel output.
	Nature and trend of any abnormal biochemistry.
Neurological	Nature and dose of analgesia and sedative medication.
	Glasgow coma score (GCS), sedation score and delirium assessment.
	Behaviour and orientation (if appropriate).
	Positive exam findings.
Infection control / microbiology	Skin / wounds - condition / abnormalities
	Age, location and appearance of exit site of ALL Lines / tubes / catheters / cannulas / drains etc
	Temperature.
	White blood cell count and C-reactive protein (CRP).
	Date, site and sensitivities of any positive cultures.
	Nature and duration of any antimicrobials.

DAILY CLERKING CHECKLIST – D A S H E M F F U G B T T

Drug chart: legible and clear? Appropriate dose / frequency? Missed doses? Restart chronic drugs? Stop anything? IV to enteral? Therapeutic monitoring required?

Analgesia: Regular simple analgesia + / - opiates. What is the quality of pain control?

Sedation: Is there a need for sedation over and above analgesia? If so, when was it last stopped and what happened? What is the sedation plan for today? Does the patient have features of delirium e.g. inattention or disorganised thinking?

Head of bed up 30 – 45 degrees: Can the patient be sat out of bed or even mobilised?

Eye care: Simple eye ointment qds for all patients receiving mask or invasive ventilation (unless long term tracheostomy and awake).

Mouthcare: Chlorhexadine mouthwash + / - nystatin 4-6 hourly? Is there any peri-oral pressure injury from the endotracheal or other tubes?

Feeding: Is the patient successfully being fed? Are they receiving prokinetics? Do they still need them (not if 4hrly gastric aspirates are <200mls for >24hours)?

Fluid balance: What is the fluid balance target for today? Have any maintenance IV fluids been stopped? What is the sodium balance?

Ulcer prophylaxis: Patients who are intubated and/or coagulopathic should receive ranitidine prophylaxis 150mg NG bd (unless on PPI).

Glycaemic control: Is the blood sugar ≤8.0 mmol/l over the last 24 hours?

Bowels: Are they working? If not, senna 15mg NG od-bd and sodium docusate 200mg bd?

Thromboembolic prophylaxis: dalteparin 5,000 iu prescribed? TEDs? Calf pumps?

Tubes: for each tube (nasogastric, all vascular access, urinary catheter, drains etc) consider, is it still needed, is it working, is the exit site inflamed (look especially for pressure injury from NG tubes)?

For more information see later section, **GENERAL CONSIDERATIONS FOR PATIENT CARE IN THE ICU**

DAILY INVESTIGATIONS

- Most patients should have the following checked daily and on return from a major surgical procedure: full blood count (FBC), Clotting (including fibrinogen if coagulopathy suspected), urea & electrolytes (U&E's), liver function tests (LFTs), Troponin I, Calcium, Phosphate, Albumin, Magnesium and Glucose. [For biochemistry request, "ITU profile"]
- Patients receiving once daily aminoglycosides and/or continuous infusions of glycopeptides (vancomycin) require daily random levels.
- Patients receiving digoxin, aminophylline or phenytoin may require frequent blood level monitoring. Caution is required in interpreting drug levels as most assays measure total drug not free drug (i.e. non-protein bound). As many critically ill patients have markedly reduced serum protein levels, especially albumin, total drug levels which would normally be considered subtherapeutic or therapeutic, may actually be toxic.
- If a patient is likely to require blood products or is going to theatre, ensure that a serum sample is, or has been, sent to the blood bank.
- Screening swabs for colonising multi-resistant bacteria, in particular, methicillin resistant Staphloccus aureus (MRSA) are usually sent on admission and on a fixed day of the week thereafter.
- The results of all laboratory investigations, including microbiology, should be checked frequently, flow-charted and abnormalities addressed.
- Patients receiving mechanical ventilation may require a daily CXR (preferably erect) during the acute phase of their illness. Try to plan for these to occur after any relevant procedures, such as central venous line insertion or percutaneous tracheostomy, have been performed. In the more sub acute phase, always articulate what question(s) you want the CXR to address.
- Any patient with known or suspected cardiac problems should have a daily 12 lead (ECG).

RADIOLOGICAL INVESTIGATIONS

- Make requests as early in the day as possible.
- Discuss complex patients with a radiologist and clarify that the optimal investigation has been requested to address the question raised.
- Establish and inform all concerned of the agreed time and date for the investigation.
- Some radiological investigations require intravenous (IV) and / or oral contrast. Most IV contrast agents are nephrotoxic. The mainstay of prevention is good hydration. Consider giving an additional bolus of an appropriate crystalloid prior to giving the agent. There is some evidence to support the use of N-acetylcysteine (NAC) as a prophylactic agent against contrast induced nephropathy. If given by the enteral route, it must be commenced at least 48 hours prior to the procedure. As many critically ill patients do not enjoy the luxury of this time interval, NAC must be given intravenously to be effective. There is also some evidence to support the use of sodium bicarbonate and prophylactic renal replacement therapy in patients with severely diminished renal function. The most recent trial recommends 500mls of 1.4% $NaHCO_3$ with 2.4g of NAC [5].

COMMUNICATION

- Make every effort to communicate, at least daily, with the patient's relatives and the admitting team.
- Numerous medical / surgical teams visit the ICU every day. To maintain effective communication all discussions must be documented.
- All discussions with relatives must be documented. Whenever possible the nurse looking after the patient should also be present. If this is not possible, always inform the nurse concerned regarding the content and outcome of the discussion.

DISCHARGE FROM THE CRITICAL CARE ENVIRONMENT

- Ensure a concise but comprehensive discharge summary is completed prior to the patient's discharge. Include an explicit PLAN. Clearly document if the patient is considered to be unsuitable for re-admission to ICU, and if so, suggest that a DNAR form be completed by the receiving team as soon as practical.
- Speak to a senior member of the team who will be taking over the care of the patient, emphasising any continuing problems and the management PLAN.
- Unless essential, do not discharge patients between 22:00 and 08:00.

***Suggested outline of discharge summary**:*

- Date and duration of ICU admission
- Reason for ICU admission
- List diagnoses / active problems, including a brief chronological history (include important results / diagnostic procedures / interventions) of each and the plans for future management
- Brief description of clinical state
- Age and plans for any lines and drains
- List of current medications (including dose, frequency, route of administration, need for therapeutic monitoring, intended duration of treatment or plans to increase / decease dose) and any chronic drugs that may need to be reinstated
- Plans for nutritional and fluid management, as appropriate
- Summary of patient awareness of condition and communication with relatives and other clinical teams
- Predictable risks post discharge and re-escalation plan

DYING PATIENTS AND END OF LIFE CARE

- In the terminal phase of a patient's illness ensure optimal palliation of any / all sources of distress. Seek expert help if required. Only contact the Palliative Care Team if the patient is to be discharged from GICU.
- After due consideration and discussion, withhold / withdraw any unnecessary interventions, including continuous monitoring.
- If a patient is terminally ill, ensure that the family are fully aware of the situation.
- Contact the admitting team to ensure they are also aware of the situation.
- If appropriate, always consider organ / tissue donation and adhere strictly to protocols. Contact the Transplant Co-ordinator via main switchboard to discuss or refer the patient.
- Always inform all relevant teams after a patient has died and whenever possible, inform the patient's general practitioner.
- If uncertain how to complete the death certificate, discuss this with a senior colleague. Many patients will require discussion with the coroner prior to completion of the death certificate.
- Ensure death certificates and cremation forms are completed promptly. Always ensure that all forms are completed for all patients. Remember, if a death occurs out of hours, you may be the only person who can complete the forms.
- Record all communications in the notes so that ever body knows who has and who has not been contacted. Record the cause of death clearly in the notes.

DO NOT ATTEMPT RESUSCITATION ORDERS

As of 26 March 2007 there is a new Trust do not attempt resuscitation (DNAR) policy (*see* http://stginet/Policies/Clin_1-Ethics/Clin_1_1.pdf).

This policy affects three groups of patients that the GICU medical team will be dealing with.

1. Ward referrals reviewed by GICU. If a patient is not considered appropriate for ICU care and is likely to deteriorate to the point of cardio-respiratory arrest, it is the responsibility of the referring team to complete the DNAR form. This must be communicated to that team at the time the patient is reviewed. If a patient is inappropriate for ICU care before they arrest then clearly they should be made DNAR.
2. Patients on GICU. These patients do not require DNAR forms to be completed as patient specific plans are formulated and regularly reviewed that exceed the scope of the Trust DNAR form. Immediate clarification can always be sort by calling the on-call SpR, who can, if necessary, contact the on-call consultant.
3. Patients being discharged from GICU to the ward. If it is the GICU team's opinion that a patient would not benefit from a return to ICU following discharge and / or their discharge is for end of life care on a ward and this has been discussed with the team assuming on-going care of that patient, the GICU team can complete a Trust DNAR form for the patient prior to their discharge. However, whenever possible, the DNAR form should be completed by the receiving team prior to the patient leaving GICU.

If there are any difficulties with this policy they should be reported to the on-call GICU consultant.

CONSENT

- In the UK, consent is now governed by the Mental Capacity act of 2005 (*see* http://www.opsi.gov.uk/acts/acts2005/20050009.htm)
- A patient can only give consent if they have capacity. However, the Act makes the assumption that capacity exists, hence, your reason for considering there to be a lack of capacity must be documented.
- No-one except an appointed advocate can give consent on behalf of an incompetent (unconscious) adult.
- It is a good practice to obtain 'informed assent' from the next of kin, but this has questionable legal validity in the UK, unless this person has been appointed by the patient as their advocate.
- In a situation where a patient can give informed consent, it must be taken by someone capable of performing the procedure for which consent is being sort.
- In the ICU consent issues must be taken seriously. If conflicts or misunderstandings arise seek senior help early.

CALLING THE CONSULTANT AT NIGHT

- Please do not hesitate to contact the on call consultant to discuss any issues.
- Please take a moment to collect your thoughts and all the available information before picking up the phone.
- If you cannot contact the consultant-on-call, ring one of the other consultants.
- We don't expect you to know everything.

REFERRALS

- All patients, with the exception of planned admissions of elective surgical patients, must have a referral audit form completed.
- Blank forms are kept in the box folder on the main desk.
- Completed forms should be returned to the same folder.
- Please fill in as much detail as possible. The outcome at 24 hours data is completed by one of the audit team.
- Use the back of the form to record any other useful information and when taking down details.

TRANSPORTATION OF THE CRITICALLY ILL PATIENT

- Transfer of any critical ill patient out of the critical care environment requires careful planning and specialist skills. Even short journeys may provoke significant deterioration. Many areas present a hostile environment to the critically ill patient and the ICU team caring for them.
- The team escorting the patient must ensure that all necessary equipment is taken on the transfer including sufficient supplies to deal with all predictable adverse events. In particular, the quantity of oxygen and battery power for ventilators, monitors and infusion pumps must be carefully considered and a plan in place to deal with supply and / or device failure.
- Patients who are intubated should always be escorted by a doctor with established airway skills. An experienced nurse should also always accompany the patient.
- Try to minimise the amount of equipment taken with the patient. For example disconnect from feeding pumps and non-essential infusions.
- Position monitors, ventilators, pumps etc where they can be easily seen and accessed. Whenever possible, position these devices such that the patient can be transferred off their beds with minimal repositioning of equipment.
- Prior to leaving perform a N E W S (necessary, enough, working, secure) checklist [6].

GUIDELINES FOR THE INSERTION AND CARE OF LINES

- Prepare all materials required prior to insertion to avoid interruption and contamination
- Position patient optimally, don't accept inadequate arrangement
- Choice of site is not vital, however, consider the accessibility and ease of exit site contamination. Always balance the risks and benefits. If unfamiliar with subclavian line insertion please seek help / supervision.
- Presence of a nurse or other doctor to assist is essential.
- No shaving of area; hair cutting is acceptable
- Prior to insertion wash hands thoroughly with chlorhexadine based surgical scrub (hibiscrub).
- Clean skin with 2% Chlorhexidine in 70% alcohol; NOT alcoholic iodine
- Allow 2 minutes drying time
- Use full sterile technique including gown, gloves, large drapes (plus goggles/mask with visor for high risk patients)
- If the line insertion is difficult don't use up every site – leave at least one for a fresh pair of hands! We have an U/S machine, which should be used to assess central veins and guide line insertion if necessary. For training in this see / find Dr Ball.
- All central lines must be sutured in place
- Lines should only be changed as clinically indicated, not routinely.
- All blood should be removed from lines, skin and internal lumens as this acts as a nidus for infection.
- Cover the line exit site with an occlusive dressing.
- Swab all access ports prior to each use using chlorhexadine soaked gauze. Ask any senior nurse to demonstrate the correct technique.
- Please document in notes when any line is inserted.
- If replacing a potentially infected / colonised line, always take a set of blood cultures from the new line at the time of insertion and send the old line tip for culture.

SPECIFIC LINE RELATED ISSUES

Arterial Lines

- The radial artery is the preferred site. Femoral and dorsalis pedis are the usual alternatives. Axillary, brachial and ulnar are all possible but generally avoided as arterial injury at these sites can result in limb threatening distal ischaemia.
- There are 3 recognised methods of insertion: transfixion, standard cannulation and the Seldinger technique.

Central Lines

- The internal jugular and subclavian sites are preferred but femoral veins can be useful in a crisis. If using the femoral approach always avoid the skin crease and effectively tunnel the line a few centimetres.
- Quadruple lumens preferred for access.
- Do not insert wires too far as you risk vascular perforation and arrhythmias.
- Please attach clean caps to all lumens.
- Please ensure all lumens are flushed prior to insertion.
- Please ensure that lines are well secured.
- A chest X-ray must be performed and reviewed for pneumothorax and line malposition after any SVC central line is inserted.

Double Lumen Dialysis Catheters (VasCaths)

- For haemofiltration or dialysis. Flush both lumens prior to insertion, and cautiously "lock" each lumen with the appropriate volume of 5,000iu/ml heparin. Leave a label on the line to confirm hep-lock including time and date. Anyone considered for haemofiltration is a potential dialysis patient and must have their hepatitis B+C status checked. Please note that there are 2 sizes available, a 15cm for IJ/subclavian insertion and a 20cm for femoral use. You MUST ensure the correct size is used at the correct site.

GENERAL CONSIDERATIONS FOR PATIENT CARE IN THE ICU

Many of the items detailed below are being brought together in so-called care bundles [7]. This approach has been shown to increase compliance.

THINK THEN ACT

- Take a holistic approach.
- Always introduce yourself to patients and their visitors and explain what your about to do.
- Patient comfort and dignity should be everybody's priority.
- Be conscious of the loss of the patients' privacy and autonomy.
- A significant proportion of critical care is attention to detail.
- Review the effectiveness of management plans at frequent intervals. If you initiate or change something decide when you'll need to review the patient's response.
- Record everything you do in the notes and / or on the charts including a rationale and the response. Start every entry with the date, time and PRINT your name.

PRESCRIBING

- Write legibly.
- Document any premorbid medications clearly and make a plan to continue, stop or re-introduce each drug.
- Clearly document all suspected adverse drug reactions.
- Stop all unnecessary medications. Be conscious and cautious of the hazards of polypharmacy.
- Multiple drug charts increase the risk of drug errors occurring therefore prescription charts should be rewritten rather than changed and multiple charts should ideally be combined to have as few charts as possible.
- For antimicrobial prescriptions, state the indication / culture details and the intended duration of therapy.

ANALGESIA AND SEDATION [8]

Analgesia, with or without sedation, is an essential component of the holistic care of critically patients. With the exception of immediate life saving interventions, patient comfort should be the first priority.

Aims of analgesia-sedation regimes:

- Patients should be comfortable and pain free.
- Anxiety should be minimised.
- Patients must be able to tolerate appropriate organ system support / nursing care.
- Patients should not be paralysed and aware.
- Ideally, patients should be calm, co-operative, able to communicate and to sleep when undisturbed. From a clinical perspective, the ideal state is to be able to complete a neurological assessment. Coughing and moving are not in themselves reasons to sedate a patient, unless such activity places the patient at risk.

Guidelines:

- The commonest indication for the initiation of analgesia-sedation is endotracheal intubation and ventilation. Some patients may tolerate this without any drugs but most will require analgesia and suppression of airway reflexes.
- Most ICUs employ continuous infusions of opiates and sedatives. The choice of agents depends upon a number of factors including drug pharmacokinetics, cost and personal preference (see Tables 7 and 8). Some advocate analgesia only sedation with the addition of non-analgesic sedatives only as necessary.
- Start with a small bolus dose prior to commencing an infusion. If this is insufficient to achieve the desired level of analgesia / sedation, repeat the small bolus prior to each increase in infusion rate. This is to allow steady state drugs levels to be achieved more quickly and reduces total cumulative dosage.
- All drugs accumulate to some degree, if given to critically ill patients for prolonged periods. It is standard practice to perform a daily cessation of the drug regime, which should only be re-started as clinically indicated.

- Regular simple analgesia should always be considered in critically ill patients regardless of pathology as immobility and critical care interventions are uncomfortable and can be distressing (see Table 9).
- Neuromuscular blockade should only be considered in patients in whom sedation / analgesia does not achieve the defined goals, most commonly, failure to achieve adequate ventilation or as part of a cooling protocol. Intermittent bolus dosing is usually preferable to IV infusions (see page 91). If given by infusion, daily cessation is mandatory. Prolonged use of neuromuscular blocking agents is associated with a higher incidence of critical illness neuromyopathy.
- Be aware that sedative drugs do not achieve physiological sleep (as assessed by EEG) and that sleep depravation is probably one of the principle causes of ICU delirium [9].
- Prolonged use of sedation / analgesia drugs is associated with tachyphylaxis and some degree of neurochemical dependence, and therefore withdrawal syndromes. Weaning from prolonged use may require staged reduction over a period of days and may be enhanced by the use of alternative drugs, in particular, methadone (prolongs QTc), a benzodiazepine and clonidine. Haloperidol, chlorpromazine, olanzapine and risperidone have also been used (see Table 10).

Before increasing sedation and / or adding neuromuscular blockade:

- Exclude any avoidable source of physical discomfort.
- Review the need for all uncomfortable or disturbing interventions.
- Consider whether the increase in sedation is an index of clinical deterioration.
- Consider non drug measures e.g. patient positioning.
- Consider analgesia.
- Consider a bolus dose rather than an increase in infusion rate, especially if prior to an unpleasant intervention.
- Over sedation is associated with a higher incidence of ventilator associated pneumonia, prolonged weaning from mechanical ventilation, colonisation with multiply resistant organisms, an increased requirement for neurological investigations, prolonged ICU stay and death.

Assessment of the depth of sedation

- This is an essential skill in the assessment and review of all ICU patients receiving any form of sedative.
- We use the Richmond agitation-sedation scale to communicate and set goals.

Proceedure

1. Observe patient. Is patient alert and calm (score 0)?
   If the patient exhibits restless or agitated behaviour then score +1 to +4 using the criteria listed below, under description.
2. If patient is not alert, in a loud speaking voice state patient's name and ask them to open their eyes and look at you. Repeat once if necessary.
   Patient has eye opening and eye contact, which is sustained for more than 10 seconds (score -1).
   Patient has eye opening and eye contact, but this is not sustained for 10 seconds (score -2).
   Patient has any movement in response to voice, excluding eye contact (score -3).
3. If patient does not respond to voice, physically stimulate patient by shaking shoulder and then rubbing sternum if there is no response to shaking shoulder.
   Patient has any movement to physical stimulation (score -4).
   Patient has no response to voice or physical stimulation (score -5).

TABLE 6: Richmond agitation–sedation scale

Score	*Term*	*Description*
+4	Combative	Overtly combative or violent; immediate danger to staff
+3	Very agitation	Pulls on or removes tube(s) or catheter(s) or has aggressive behaviour toward staff
+2	Agitated	Frequent non-purposeful movement or patient–ventilator dys-synchrony
+1	Restless	Anxious or apprehensive but movements not aggressive or vigorous
0	Alert and calm	
-1	Drowsy	Not fully alert, but has sustained (more than 10 seconds) awakening, with eye contact, to voice
-2	Light sedation	Briefly (less than 10 seconds) awakens with eye contact to voice
-3	Moderate sedation	Any movement (but no eye contact) to voice
-4	Deep sedation	No response to voice, but any movement to physical stimulation
-5	Unarousable	No response to voice or physical stimulation

Table 7: Continuous infusion sedative analgesic regimes

Drug	*Regime*	*Notes*
Alfentanil **First choice**	Loading 15 - 50 mcg/kg Maintenance 30 - 85 mcg/kg/hr (1 - 6 mg/hr)	Rapid onset. Relatively short acting. Accumulates in hepatic failure.
Morphine **Second choice**	Loading 5 - 15 mg Maintenance 1 - 12 mg/hr	Slow onset. Long acting. Active metabolites. Consider bolus dosing / PCA in place of infusion. Accumulates in renal and hepatic impairment.
Fentanyl	Loading 25 - 100 mcg Maintenance 25 - 250 mcg/hr	Rapid onset. Modest duration of action. No active metabolites. Renally excreted. Patches available for longer term use.
Remifentanil	Dose 0.4 – 45 mcg/kg/hr	Rapid onset and offset of action with minimal if any accumulation of the weakly active metabolite. Significant incidence of problematic Bradycardia with bolus dosing.
Clonidine	Dose 1-10 mcg/kg/hr	An α_2 agonist. Has sedative and analgesic effects. Infusion doses up to 25 mcg/kg/hr AND slow bolus doses of 10-20 mcg/kg have been described as being safe with a surprisingly low incidence of hypotension and bradycardia [10].
Ketamine [11, 12]	Analgesia 0.2 mg/kg/hr Induction 0.5 - 2.0 mg/kg Maintenance 1 - 2 mg/kg/hr	Atypical analgesic with hypnotic effects at higher doses. Sympathomimetic; associated with emergence phenomena when given at hypnotic doses when usually co-administered with a benzodiazepine. Potentially useful adjunct to opiates (opiate sparing) as part of a mixed regime

Table 8: Continuous infusion sedative regimes

Drug	*Regime*	*Notes*
Propofol 1% **First Choice**	Loading 1.5 - 2.5 mg/kg Maintenance 0.5 - 4 mg/kg/hr (0 - 200 mg/hr)	Intravenous anaesthetic agent. Causes vasodilatation and hence hypotension. Extra hepatic metabolism, thus does not accumulate in hepatic failure. Has no analgesic properties. Made in Intralipid hence maximum long term (hours) infusion rate should be ≤200 mg/hr. Propofol infusion syndrome is a serious complication of prolonged and high dose administration with a significant fatality rate [13].
Midazolam	Loading dose 30 - 300 mcg/kg Maintenance 30 - 200 mcg/kg/hr (0 - 14 mg/hr)	Shortest acting benzodiazepine. Active metabolites accumulate in all patients especially in renal failure. Consider intermittent bolus dosing rather than an infusion.

Table 9: Regular / bolus dose analgesia

Drug	*Regime*	*Notes*
Paracetamol	1 g NG / PO 6 hourly or 1 g IV 6 hourly	Starting regime for simple analgesia Only use IV if enteral route unavailable / unreliable OR as part of an opiate sparing regime. Note 1g IV paracetamol ≡ 2.5 – 5mg IV morphine
Diclofenac	50 mg NG / PO 8 hourly or 75 mg IV 12 hourly	As part of an opiate sparing regime BUT only in well hydrated patients with normal renal function. Usually requires PPI cover. NSAIDs may have a role in reducing hypertropic acetabular ossification post acetabular fracture repair.
Codeine, Dihydrocodeine Oramorph	Starting regime: Oramorph 2.5 – 10mg PRN Max. 60mg / 24 hrs	Essentially the same drug (codeine is metabolised to morphine BUT only by 70% of the population). Use regularly in post-op patients to wean from PCA infusions. Avoid in renal failure. Patient must receive aperients. Oramorph is the preferred agent.
Oxycodone	5 – 30 mg NG / PO 4 -12 hourly	Safer in renal failure as extensive hepatic metabolism to less active drug.
Methadone	Start 15-30 mg NG / PO daily	Useful daily opiate. Can prolong QT interval.
Tramadol	50 – 100 mg 6 hourly	**PLEASE AVOID**. Mixed weak opiate and noradrenaline re-uptake inhibitor. Highly emetogenic, causes delirium, especially in elderly, and SIADH. Multiple drug interactions therefore contra indicated in patients on any antihypertensives, SSRIs, tricylics and warfarin.

Table 10: Regular / bolus dose sedation

Drug	*Regime*	*Notes*
Haloperidol	2.5 – 5 mg NG / PO / IV Max daily dose 2 0mg	Often delayed onset of action in patients with agitated ICU delirium.
Chlorpropazime	10 – 250 mg NG / PO / IM	Alternative to haloperidol.
Olanzapine	5 – 15 mg NG / PO daily	Alternative to haloperidol.
Risperidone	1 – 4 mg NG / PO / s/l	Alternative to haloperidol.
Lorazepam	0.5 – 4 mg s/l / NG / PO / IV PRN	Tablets work well s/l. IV preparation is in ethylene glycol. Give 8 – 12 hourly. Fewer active metabolites / more predictable half life in multiple organ failure (~14 hours) compared to diazepam.

PATIENT POSITIONING [14, 15]

- To prevent passive aspiration and enhance respiratory mechanics all sedated patients should be positioned at least 30° head up. As soon as practical, sit patients out of bed for a portion of every day.

EYE CARE

- Prescribe simple eye ointment 6 hourly for all unconscious, sedated and / or mask / helmet ventilated patients.
- Check the eyes daily for injury / inflammation in sedated / unconscious patients.

MOUTH AND NOSE CARE

- Always examine the mouth and nose of patients with endotracheal and NG / NJ tubes. Look for signs of pressure necrosis, oral colonisation and sinusitis. Start chlorhexadine mouthwash +/- oral nystatin (topical antifungal) and discuss changing the offending tube. (Topical antiseptics are increasingly being adopted as primary prophylaxis and added to existing care bundles.)

NUTRTITIONAL SUPPORT [16]:

Early enteral feeding

- Early initiation (within 12 hours) is the key nutritional factor influencing clinical outcome.
- 'Late' feeding (i.e. feeding commenced within 36h of admission) is associated with increased gut permeability and an increase in late multi-organ failure.
- Timing, rather than amount / volume of feed administered is the key factor.

Prokinetic therapy / enteral feeding failure

- If delayed gastric emptying is evident (gastric aspirates > 200mls after 4 hours) commence prokinetic therapy.
- First line therapy is metaclopramide 10 mg IV 8hrly.
- Second line therapy is erythromycin 250 mg IV 8 hourly.
- Prokinetics should be reviewed daily and crossed off once patients have been absorbing for 24 hours.
- If enteral feeding cannot be established within 12 hours of admission consider a hypertonic dextrose infusion either 25 mls/hr of 20% or 10 mls/hr of 50%.
- Consider post pyloric feeding if gastric aspirates remain >200ml every 4 hours, for > 48h with regular prokinetic administration

Total parenteral nutrition (TPN)

- To be of benefit, TPN has to given for a minimum of 10-14 days. Unless there is good reason to suspect that enteral feeding cannot be successfully established in this time frame, then serious consideration of the risk benefit ratio of commencing TPN should be undertaken.
- A dedicated central line or lumen should be set aside for TPN. Close monitoring of electrolytes, liver function tests (for cholestatic jaundice) and line related sepsis should be undertaken.

UPPER GASTROINTESTINAL TRACT

- All intubated patients, those receiving mask / helmet positive pressure ventilatory assistance and any other patient not able to eat should have either

an oro or a nasogastric tube. The preferential site and bore of the tube need to be considered carefully.

- The following groups of patients should receive stress ulcer prophylaxis [15, 17]:
    - those not receiving nasogastric feeding
    - those shocked / on vasopressors
    - those with a coagulopathy (including uraemia) / anticoagulated
    - those receiving gastric mucosal irritants e.g. steroids, NSAIDS
    - burns / polytrauma
    - prolonged intubated and sedated
- Recommended starting regime: enteral ranitidine 150 mg 12 hourly. Consider IV ranitidine 50mg 8 hourly (reduce to 12 hourly in renal failure) **ONLY** if the enteral route is unavailable.
- For those deemed to be at very high risk or with proven peptic ulcer disease consider lansoprazole fasttabs 30 mg daily.
- Intravenous PPIs are reserved for patients with **suspected or proven upper GI bleeding**. Give omeprazole 80mg IV over 1 hour then 8mg/hr for 71 hours. Then switch to enteral PPI. Always consider H. pylori eradication therapy.
- In suspected or proven **variceal haemorrhage**, in addition to IV omeprazole, give terlipressin 2mg IV followed by 1 or 2mg every 4 to 6 hours until bleeding is controlled, for up to 72 hours.

GLYCAEMIC CONTROL [18]

- There is significant evidence to support tight glycaemic control (blood glucose levels between 6.0 and 8.0 mmol/l) in all critically ill patients, although the exact definition of "tight" remains controversial. Have a low threshold for commencing IV insulin infusion. Rarely, if ever, stop infusions, instead reduce rate and give dextrose as required.
- Keep a close watch on serum potassium concentration and supplement intake to maintain levels >4.0 mmol/l. Sando K via NGT &/or 40mmol/l in IV

maintenance fluid are the preferred methods. **AVOID** infusions of concentrated KCl whenever possible

- Avoid hypoglycaemia and if present, treat aggressively. Continuous feeding regimes, or dextrose infusions, should be used to reduce / avoid this problem. However, recent studies in tightly controlled diabetics have failed to demonstrate any long term neuro-cognitive decline in patients with recurrent hypoglycaemia [19].

BOWEL CARE

- Constipation and diarrhoea are common complications of critical illness.
- Most patients benefit from stool softeners e.g. sodium docusate 200mg twice daily and mild aperients e.g. sennakot 5-15mls twice daily. Osmotic laxatives, e.g. lactulose, are avoided (except in decompensated liver patients) as they can significantly contribute to bowel gas formation.

MAINTENANCE FLUIDS – INTRAVENOUS AND ENTERAL

- Inputs: Patients with normal loses will require 2000 to 3000 ml of water per day. Whenever possible this should be administered enterally. Additional losses are usually replaced intravenously.
- Losses - normal routes: (quantities many be low, normal or high): renal, gastrointestinal, respiratory and intact skin (sweat).
- Losses - additional considerations in critical illness: bleeding, wounds / burns and 3rd space accumulation due to leaky microvasculature and reduced oncotic pressure.
- The aim of fluid therapy is to maintain adequate intravascular volume, patient hydration, and normal electrolyte concentrations.

Assessing the adequacy of fluid replacement [20]:

- For intravascular volume see later section on cardiovascular support.
- Plasma / serum biochemistry (measured at least daily)
- Urine output > 0.5 ml/kg/hour, except in the immediate stress response (due to ADH and rennin-angiotensin-aldosterone) and oliguric or anuric renal failure.

- In critically ill patients, fluid replacement often results in tissue oedema. This has a number of detrimental effects (see Table 11). This is often unavoidable and can result in total body excess but effective intravascular hypovolaemia.
- Accurate measurement of fluid input and output is fundamental.
- A fluid balance target (including enteral intake) should be set daily. Any additional boluses of IV fluids should be taken into account. Such boluses should be minimised and given against strict physiological criteria see later section on dynamic fluid challenges.
- To achieve the target balance may require regular low doses or continuous infusion of loop diuretics or renal replacement therapy.
- Close attention should be paid to serum concentrations of sodium and chloride. Both tend to accumulate as a result of IV fluids and drugs. Iatrogenic hyperchloraemic acidosis is a common problem (see Table 12).
- Daily electrolyte requirements for a normal adult are:
    - Sodium 1-2 mmol/kg/day
    - Potassium 1 mmol/kg/day

Table 11: The clinical systems that may function inefficiently in the presence of oedema.

Tissue	*Effects of oedema*
Lungs and pleural cavities	Deterioration in gas exchange, predisposition to pneumonia
Bowel and peritoneal cavity	Malabsorption, ileus, ischaemia, anastomotic breakdown, stress ulceration, bacterial translocation, intra abdominal hypertentension
Skin and soft tissues	Pressure area injury, impaired wound healing

Table 12: Comparison of plasma to intravenous fluid therapies

	Plasma	*Intravenous fluids*			
		0.9% NaCl	*5% Dex*	*Hartmann's*	*Volpex*
[Electrolyte] All in mmol/l	Na^+ 140 K^+ 4.0 Mg^{2+} 0.9 Ca^{2+} 2.5 Cl^- 98 PO_4^{3-} 1.2 Lactate 1.0	Na^+ 154 Cl^- 154	Na^+ 0 K^+ 0.0 Mg^{2+} 0.0 Ca^{2+} 0.0 Cl^- 0	Na^+ 131 K^+ 5.0 Mg^{2+} 0 Ca^{2+} 2.0 Cl^- 111 PO_4^{3-} 0.0 Lactate 29	Na^+ 154 K^+ 0.0 Mg^{2+} 0.0 Ca^{2+} 0.0 Cl^- 125 PO_4^{3-} 0.0 Lactate 0
Osmolality mOsm/l	290	308	278	279	284
pH @ 37ºC	7.40	5.0	4.0	6.5	7.4
Notes			200 kcalories per litre		

See also http://www.iv-partner.com/index.cfm?887EDD57B0E745469F7063BB9503A30A

PACKED RED CELL TRANSFUSION [21, 22]

- Erythrocytes are highly deformable, biconcave discs. They have 2 principal roles. Firstly, the carriage of haemoglobin, which is essential for the efficient transport of oxygen. Secondly, they are the major determinant of blood viscosity, maintenance of which is essential for effective microcirculatory function [23].
- Blood loss results in a reduction in oxygen carrying capacity and blood viscosity. This can be compensated for by increases in flow (cardiac output and microcirculatory autoregulation) but may be further compromised by resuscitation with crystalloids and / or colloids.
- The optimal haemoglobin (Hb) concentration / haematocrit is a balance of rheology and oxygen carrying capacity. A target value is usually set at 8-10g/dl, even in patients with critical myocardial or other organ ischaemia.
- Current evidence suggests that a transfusion trigger for PRBCs should be a haemoglobin concentration of <7.0 g/dl [24]. Although this is an oxygen carrying capacity trigger it should also be considered a viscosity trigger [25]. In the setting of critical illness, no benefit of a higher transfusion threshold has

been demonstrated, even in patients with critical coronary (or other organ) arterial disease [24]. Be aware that most blood gas analysers are significantly less accurate at [Hb] measurement than formal haematology laboratories (errors as much as ±2g/dl).

- Try to minimise iatrogenic loses, in particular, blood sampling and during line insertion. Be conscious of the effects of iatrogenic haemodilution.
- Transfusion is not a benign intervention [26]. Stored red blood cells become 2,3-DPG depleted resulting in higher O_2 avidity, which causes a leftward shift in the oxygen dissociation curve i.e. a reduction in oxygen release to the tissues. In addition, they loose their highly deformable biconcave morphology and come to resemble spiky balls, which fail to enter capillaries and can become impacted, obstructing the micro-circulation. There is also the potential risk of disease transmission and allergic reactions.
- In resuscitation however, in the setting of both sepsis [27] and trauma [28], the early use of PRBCs appears to be of benefit as part of the package of care. It should be stressed that in resuscitating haemorrhagic shock, the recommended ratio of PRBCs to fresh frozen plasma (to platelets) is now 1:1(:1) [28] although this is not without controversies [29-31].
- The place of intravenous iron and recombinant erythropoietin in the management of anaemia remains undefined in the critically ill population [32].

THROMBO-PROPHYLAXIS

- Every patient should receive an appropriate prophylactic dose of low molecular weight heparin (LMWH) unless they have a coagulopathy / thrombocytopaenia or they are receiving therapeutic anticoagulation or heparin / epoprostenol (Flolan) as anticoagulation for renal replacement therapy.
- Routine practice for patients >50kg is dalteparin 5.000iu od. For patients <50kg, consider a dose reduction to 2,500iu od.
- Once daily LMWH should be prescribed at 18:00 so that any necessary surgical procedures (e.g. removal of epidural catheters, tracheostomies, line insertions / removals) are not delayed the following day.
- Appropriately sized TED stockings should be fitted to all patients where possible. Compression boots may also be useful.

INITIAL ASSESSMENT: ABC

In any critically ill patient the hierarchy of assessment and resuscitation for life threatening pathology starts with the airway followed by breathing and circulation, the so-called ABC approach. Over time, the alphabet of resuscitation has been extended to D for disability (rapid neurological assessment including measurement of blood glucose) and E for exposure and examination.

Initial assessment

- First assess the patency of the upper airway:
    - LOOK – is there any visible obstruction and / or seesaw breathing?
    - LISTEN – is there stridor or silence?
    - FEEL – is there any gas flow?
- If the airway is partially or completely obstructed, institute a jaw thrust and / or chin lift manoeuvre.
- Consider whether the patient is at risk of cervical spine injury and, if so, institute immobilisation precautions.
- If airway patency remains suboptimal, carefully insert an oral or nasopharyngeal airway adjunct paying attention to size and effectiveness.
- Administer oxygen at the highest available concentration.
- Assess breathing:
    - LOOK – is the chest rising and falling, is it symmetrical?
    - LISTEN – are there breath sounds, are they symmetrical?
    - FEEL – is there any chest movement, is it symmetrical?
- Simultaneously feel for a carotid pulse, if present obtain a blood pressure measurement as soon as practical.
- If there is no pulse, call for assistance, initiate chest compressions and follow basic and advanced life support algorithms.
- If there is a pulse, but no respiratory effort, call for assistance and supply rescue ventilation, ideally with a bag mask valve system connected to high flow oxygen. Again, follow life support algorithms.

- If there is some respiratory effort, determine the adequacy by clinical examination, attaching a pulse oximeter and if practical, obtain an arterial blood gas specimen. Be aware of the limitations of pulse oximetry especially in hypotensive patients. Arterial blood needs to be sampled into an anti-coagulated syringe and any air in the sample should be expelled before the sample is safely capped. The sample should be analysed immediately or transported in ice to minimise cellular metabolism in the sample from consuming oxygen and producing carbon dioxide.
- Immediate management of cardiovascular and neurological abnormalities are discussed in later sections.

DEFINITIVE AIRWAY MANAGEMENT: ENDOTRACHEAL INTUBATION

- Indications for endotracheal intubation include:
  - Protection of the airway
  - Preventing or relieving airway obstruction
  - Respiratory failure requiring mechanical ventilation
- Unless experienced in advanced airway skills, call for assistance whilst maintaining airway patency, delivering high flow oxygen at maximal concentration and providing rescue ventilation as described above.
- Out of hours immediate help is available via **BLEEP 6111**. This is the on-call anaesthetist for St James's Wing. Intubations on GICU are emergencies and should be treated as such.
- Ensure all necessary equipment is available and functioning. Brief any assistants. Have a well formulated failed intubation plan and be able to execute it.
- Although there are many similarities between the elective intubation of a patient for surgery and the intubation of a critically patient with one of the above indications, it is vital to consider the specific circumstances and modify the technique accordingly.
- Sedation and suppression of the airway reflexes: The choice of drug/s with which to sedate the patient for intubation must be tailored to the individual patient's needs. Many critically ill patients require minimal, and sometimes no, sedation. Whatever drug/s are used, be very familiar with their pharmacodynamics, pharmacokinetics and side effects. Be prepared to deal with immediate haemodynamic instability, in particular hypotension, by having fluid resuscitation and vasopressor therapy immediately available. Drugs to consider include, propofol, thiopentone, ketamine, fentanyl and alfentanil. Etomidate is generally considered a suboptimal choice due to its association with adrenal suppression [33].
- Muscle relaxants: As with sedation, the choice and need for a muscle relaxant must be considered carefully. Rapid onset, duration of action and side effects must all be considered carefully (see Table 13).
- Technique: Pre-oxygenate the patient with 100% oxygen and a tight fitting face mask. This should take place for at least 3 minutes, and is intended to achieve

complete nitrogen wash out. Ensure the patient is optimally positioned, and if practical, is placed in the 'sniffing the morning air position'.

- Consider asking an assistant to apply cricoid pressure to reduce the risk of aspiration. Be prepared to deal with secretions, passive regurgitation and vomiting.
- To visualise the larynx, insert an appropriately sized laryngoscope into the right side of the mouth and sweep the tongue to the left. While advancing the scope blade, be careful not to damage the teeth or lips. By advancing the scope blade along the tongue, the epiglottis should come into view. Gently apply pressure along the axis of the handle until the optimal view of the vocal cords is achieved. Insert the cuffed endotracheal tube through the cords noting the distance the cuff is below them. Inflate the cuff and manually ventilate to check endotracheal location and optimal position above the main carina (bilateral chest movement, equal air entry, absence of gastric ventilation and a classic capnograph trace).
- A bougie or airway exchange catheter can be used if the view of the vocal cords is limited. If successfully placed then a lubricated endotracheal tube maybe placed over the bougie.
- The cuff balloon inflation pressure should be checked. High inflation pressures may lead to tracheal injury.
- A nasogastric tube should be passed and a chest radiograph ordered to check endotracheal and nasogastric tube position.

Table 13: Neuromuscular blocking drugs

Drug & bolus dosing	*Bolus dose*	*Onset & Duration*	*Side effects*
Suxamethonium	1 - 2 mg / kg Ampoule 100 mg	30 s 5 mins	Depolarisation. Histamine release. Elevation of plasma K^+ by ~1 mmol / l hence contraindicated in hyperkalaemia.
Atracurium	0.3 - 0.6 mg / kg Ampoule 50 mg	90 - 120 s 60 mins	Racemic mixture. Broken down by serum esterases hence predictable pharmacokintics in renal and hepatic failure. Causes histamine release hence contra-indicated in acute severe asthma. Inactive metabolite, laudanosine, lowers seizure threashold
Vecuronium	0.08 - 0.1 mg / kg Ampoule 10 mg	60 - 120 s 20 - 60 mins	Lipid soluble *hence* accumulates.
Rocuronium	0.6 mg / kg Ampoule 50 mg	< 60 s 30 - 60 mins	Most rapid onset of non-depolarising blockers. Low incidence of histamine release. Low, but significant incidence of anaphylaxis.

For more detailed information on neuromuscular blocking drugs see [34-36]

RESPIRATORY FAILURE AND SUPPORT

- There are 2 principle functions of the respiratory system, uptake of oxygen and elimination of carbon dioxide.
- Although these functions are closely linked, respiratory failure can result in hypoxaemia, hypercapnia or both.
- Arterial O_2 tension is principally determined by the fraction of inspired oxygen (FiO_2), ventilation / perfusion matching and the blood's oxygen carrying capacity (essentially haemoglobin concentration).
- Assuming a fixed rate of CO_2 production, arterial CO_2 tension is primarily determined by alveolar minute ventilation i.e. (tidal volume – anatomical & physiological dead space) x the respiratory rate.

RESPIRATORY SUPPORT

A number of techniques exist to support the failing respiratory system. There follows a description and concise guide to each technique.

Mask / helmet continuous positive airway pressure (CPAP)

- This is a high flow circuit providing a variable FiO_2 delivered at a set pressure.
- A wide variety of specific patient interface devices exist including helmets, nasal and full face masks. The patient interface device needs to be considered carefully as this is the primary determinant of patient tolerability.
- The pressure remains constant during the patient's respiratory cycle, providing positive end expiratory pressure (PEEP). The advantage of this system is that it recruits and retains the patency of smaller airways and alveoli by splinting them open. This process principally improves oxygenation.
- It should be considered an intermittent therapy.
- Failure to achieve a sustained clinical improvement within one hour should prompt the clinician to change therapy, usually by considering intubation and mechanical ventilation.
- CPAP has been demonstrated as efficacious in the management of atelectasis and pulmonary oedema.

- Therapy is usually commenced at +5 cm H_2O and escalated to a maximum of +15 cm H_2O.
- Have a low threshold for inserting an NG tube to mitigate against aerophagia and gastric distension, which can result in nausea, vomiting, aspiration and ventilatory failure secondary to diaphragmatic splinting.
- Other detrimental effects include: impediment to patient communication, ocular injury, nasal injury, facial injury (from straps) and secretion drying and retention. The latter may result in proximal airway obstruction.
- Contraindications include: a GCS < 14, an uncooperative patient, recent upper airway or upper GI surgery, a severe respiratory acidosis (commonly pH < 7.2).

Non-invasive ventilation

- This technique adds some form of ventilatory assistance to CPAP. Most commonly, this takes the form of patient triggered inspiratory pressure support (PS). Alternatively, a time cycled bilevel of CPAP is delivered.
- All of the same precautions and contraindications apply.
- There are a wide range of delivery devices available starting with small, domiciliary units that merely provide a set expiratory and inspiratory pressure (EPAP and IPAP respectively) to full ICU ventilators, which have the ability to deliver a set FiO_2, higher levels of IPAP, continuous monitoring and alarms. At the simplest end of this range, supplemental oxygen can be provided by mask entrainment.
- Non-invasive ventilation is considered the standard of care in patients with acute exacerbations of chronic obstructive airways disease. It has been successfully employed in many other conditions including as a weaning strategy from invasive ventilation. The broad indications are mild to moderate hypercapnia with or with or without hypoxaemia.
- Therapy is usually commenced with an IPAP of 8-10 cm H_20) and an EPAP of 4-5 cm H_20.. These settings are then incremented based upon efficacy and tolerability.

Remember, these techniques are supportive care, you must determine the cause of the respiratory failure and initiate definitive therapy as soon as possible.

Mechanical ventilation

- The mainstay of respiratory support is intermittent positive pressure ventilation (IPPV).
- Modern ICU ventilators are complex devices, which provide continuous monitoring and alarms as well as support.
- The terminology surrounding IPPV has become unnecessarily complex, especially ventilatory modes and their acronyms.
- The value of regular patient review, including respiratory examination, cannot be overstated.
- There has been a huge philosophical shift in the last few years away from trying to normalise gas exchange and towards minimising ventilator induced lung injury (VILI), which should perhaps more appropriately be termed physician induced lung injury.
- Regional lung ventilation and perfusion are not homogenous and this heterogeneity increases in disease. Positive pressure ventilation can cause lung injury via overdistension (volutrauma), excessive pressure (barotrauma) and cyclical recruitment and derecruitment (alectratrauma or biotrauma).
- Be aware of heart lung interactions and the effects on these of IPPV. In particular, be aware of the detrimental effects of sustained high airway pressures on right heart function and the risks of inducing right heart failure.

Nomenclature

- In simple terms IPPV can be set up as either volume controlled (pressure monitored) or pressure controlled (volume monitored). This somewhat arbitrary distinction has become blurred with the advent of complex software in modern ventilators and the development of such concepts as volume targeted pressure control, pressure limited volume control, volume support, proportional assist and assisted spontaneous ventilation. In essence it doesn't matter which mode of ventilation you select as long as you understand what you need to set and what you need to monitor (see Table).

GOALS OF SUPPORTIVE CARE

- PaO_2 > 7 kPa and pH > 7.20 whilst minimising VILI and cardiovascular compromise.

To treat hypoxaemia

- Consider performing a recruitment manoeuvre [37]
- Increase the CPAP / PEEP.
- Consider increasing the I:E ratio.
- Consider changing the patient's position
- Increase the FiO_2

To fix hypercapnia

- DON'T if the pH is normal or within acceptable limits (permissive hypercapnia)
- Increase respiratory rate and / or tidal volume – watch the effect on peak pressures, inspiratory and expiratory flows. Remember that the volume of dead space ventilation is fixed hence increasing the rate at the expense of tidal volume will result in a reduction in alveolar minute ventilation and a rise in $PaCO_2$.

Table 14: Mechanical ventilation guidance. Part 1

Things you need to set	*Guidance notes*
FiO_2	• Start high and reduce gradually. • Minimise to achieve PaO_2 8-10 kPa / SpO_2 88-92%.
Modes *(what effort is the patient making?)*	• Mandatory ventilation (MV) sometimes referred to as assist / control. The ventilator delivers a set number of breaths per minute. The inspiratory flow rate and pattern, the length of inspiration the presence and duration of any inspiratory pause and the length of expiration are all set. • Synchronised Intermittent Mandatory Ventilation (SIMV) delivers a set number of breaths per minute, which the ventilator will try to synchronise with the patient's inspiratory efforts, if any. In addition, if the patient initiates a breath in between the SIMV breaths they can be given a set level of inspiratory pressure support. In SIMV, the minimum rate is set. Use this mode if patients are making some but not enough respiratory effort. • "Pressure support + CPAP" / "Spont" / "Assisted spontaneous breathing" are all the same mode. Inspiratory pressure support is set for each patient initiated breath. No rate is set.
Cycling *(decide what to set & what to measure)* Tidal volume target	• Pressure cycled or pressure control: peak pressure is set, volume achieved is measured. Aim to keep peak / plateau / end inspiratory pressures ≤ 30 cmsH2O. • Volume cycled or volume control: tidal volume is set, peak / plateau pressure is measured / limited. Inspiratory flow pattern can be set to continuous, decelerating (miniking pressure control) or sinusoidal. In continuous flow, a variable length end inspiratory pause must be set. • 6-8 ml/kg ideal body weight in injured lungs • otherwise ≤ 10 ml/kg ideal body weight
Set rate	• Set rate to achieve target minute volume and $PaCO_2$ (4.5-6.5 kPa or higher as long as pH > 7.2)
I:E ratio OR Inspired time "Tinsp"	• 1:2 normally • consider 1:1-2:1 to recruit / optimise oxygenation (termed inverse ratio ventilation) • consider 1:3-1:4 in the presence of expiratory airflow imitation / high intrinsic PEEP / air trapping

Table 15: Mechanical ventilation guidance. Part 2

Things you need to set	*Guidance notes*
Pressure support	• Initially set pressure support to achieve the same as peak inspiratory pressure and titrate to desired tidal volume
PEEP	• Externally applied PEEP is used to recruit and retain alveolar units. Thus PEEP is used to improve oxygenation. • See table below for rough guide but remember high PEEP can compromise cardiovascular function [38]. • How to best determine the optimal level of PEEP in a patient at a particular time point remains controversial. In patients with potentially recruitable lung units consider performing a recruitment manoeuvre, usually a single or series of sustained inspiratory pauses e.g. 35cmsH_2O for 30-60s followed by a decremental PEEP trial starting at either 10 or 12 cmsH_2O. The optimal PEEP is set at the level above which, derecruitment is judged to have occurred, often judged by a deterioration in dynamic compliance and / or peripheral oxygen saturations [39-41]. • Remember that total PEEP delivered is the sum of any intrinsic PEEP and any externally applied PEEP. Patients with airflow limitation due either to disease e.g. asthma, COPD or ventilator settings e.g. too short an expired time to reach zero flow will develop intrinsic PEEP. The optimal setting of extrinsic PEEP in the presence of significant intrinsic PEEP is often a matter of trial and error see [42]

FiO_2	0.21-0.4	0.4-0.5	0.5-0.7	0.7-1.0
PEEP	5	8	10	12-15

Alarms	• Always check to see that all alarm settings are appropriate, especially the high pressure alarm.
Patient Position	• Sit up as far as possible to maximise functional residual capacity • Turn the bad side down to ventilate it or the good side down to maximise oxygenation in severe hypoxaemia • In resistant hypoxaemia, consider proning the patient [43].

UNCONVENTIONAL VENTILATION

Airway pressure release ventilation (APRV) [44]

- Airway Pressure Release Ventilation is a time cycled mode of ventilation conceived to minimise ventilator induced lung injury (perhaps better called physician induced lung injury!).
- It is perhaps best described as "CPAP plus." Like CPAP, a continuous distending pressure is delivered (P_{high}) with the addition of regular releases to washout dead space gas.
- The number or releases per minute (similar too but distinctly different from respiratory rate) are determined by setting a T_{high} (the time between airway releases).
- The duration of each release is determined by setting T_{low}. The duration of the release (T_{low}) should be set to achieve a maximum expired volume of 6ml/kg and minimum of 150ml (estimated anatomical dead space in an adult).
- Ideally, T_{low} should permit <50% of passive expiration, best determined by watching the expiratory flow curve, the idea being that <50% of the area under the curve elapses before the return to P_{high}. Note, the area under the curve is not the same as the duration, remember that the curve is exponential.
- P_{low} should always be set at zero.
- APRV is most efficacious when the patient makes some spontaneous breathing effort. Even if this effort generates very small tidal volumes, their efforts create valuable intrapulmonary gas mixing and dependent area recruitment and retention.
- Although no inspiratory pressure support is set, the ventilator will provide up to maximal inspiratory flow to maintain the set pressure.
- The airway releases washout dead space, thereby reducing the work of breathing.
- APRV can be used as a mandatory mode in patients making no spontaneous effort. It can also be used to provide a safe recruitment manoeuvre. It is very well tolerated by patients, and therefore, unlike alternative modes, doesn't in itself require analgesia / sedation.

Table 16: APRV TYPICAL PARAMETER SETTINGS

P_{high} 10-30 cmH_2O

Start at 25-30 cmH_2O / recruit then perform a decremental trial (*see* **weaning** section below). ***CAUTION*** sustained high airway pressures may compromise pulmonary perfusion, and right ventricular function with consequent reduction in left sided filling and systemic hypotension. If this occurs, consider a fluid bolus and / or alternative modes of ventilation.

P_{low} ALWAYS ZERO

T_{high} 4 - 6 seconds ($60 \div T_{high}$ = no. of releases per minute)

T_{low} 0.2 - 0.8 seconds

Start at too low a setting and increase gradually, up to a maximum of 50% of the area under the expiratory flow curve

To improve oxygenation

Recruitment manoeuvre: set **P_{high}** to 30 cmH_2O & **T_{high}** to 30s for 2-5mins ***see CAUTION above***. Then perform decremental **P_{high}** trial to a level above previous settings.

Increase **P_{high}** & / or Increase **T_{high}**. ***REMEMBER*** to reduce **T_{low}** so as not
OR to exceed 6ml/kg during an airway release.

(Increase FiO_2 as needed)

To improve CO_2 clearance

Decrease **T_{high}** i.e. increase the number of releases per minute

Reduce analgesia / sedation to increase spontaneous breathing efforts

Add "automatic tube compensation" (ATC on Draeger) *OR* inspiratory pressure support, in order to increase the tidal volume of spontaneous breaths

as a last resort

Increase **T_{low}** to **maximum** of 75% of expiratory flow (area under the curve)

Table 17: WEANING USING APRV

As oxygenation improves: reduce $\mathbf{P_{high}}$ and FiO_2. **REMEMBER** to reassess and reduce T_{low} every time you reduce $\mathbf{P_{high}}$. Employ a decremental trial by reducing the level of $\mathbf{P_{high}}$ in a stepwise fashion (e.g. 2 cmH_2O every 15mins) until SpO_2 is seen to fall, then go back 1 - 2 steps (with or without a recruitment manoeuvre).

As spontaneous tidal volume / $PaCO_2$ improves: increase $\mathbf{T_{high}}$ to reduce the number of airway releases, ultimately aiming for CPAP

PARAMETERS TO RECORD

Settings	$\mathbf{FiO_2}$ $\mathbf{P_{high}}$ $\mathbf{T_{high}}$ $\mathbf{T_{low}}$
Measurements	Minute volume
	Spontaneous respiratory rate which = total resp. rate - (60 ÷ T_{high})

High frequency oscillatory ventilation (HFOV)

- This method of ventilation requires a specialist ventilator. CPAP is provided with the addition of a piston driven diaphragm in the circuit, which oscillates at frequencies of 3-6 Hz. This results in the generation of multiple forms of gas diffusion and effective ventilation.
- It is currently used as a second line therapy in severe lung injury although its optimal use remains controversial.
- Safely establishing a patient on this mode of ventilation requires specialist training / experience. Dr Ball and Dr McAnulty have considerable expertise in its use.
- Initial settings should be conventional mean Paw +2 cm H_2O, frequency 5Hz and maximum power.
- Spontaneous breathing is poorly tolerated due to a limited bias flow through the circuit.

- If you think this mode of ventilation is indicated ASK for help.

Cuirass ventilation

- An alternative to mask / helmet CPAP is continuous negative pressure ventilation, which can be delivered via a thoraco-abdominal shell or cuirass.
- This device can also deliver expiratory support, either mandatory, or patient synchronised.
- In addition, a high frequency mode can be employed (5-20Hz).
- A secretion clearance mode of alternating high frequency and assisted coughs may also be valuable.
- As with other modes of non-invasive ventilation, this is an intermittent supportive therapy.
- Currently, it is considered a second line therapy in patients requiring NIV.

Time cycled, bilevel, pressure ventilation

- This mode of ventilation provides 2 levels of CPAP sequentially.
- It can be useful in patients who exhibit ventilator dysynchrony or as a weaning mode.

Helium oxygen gas mixtures

- Cylinders of 21% helium : 79% oxygen are available on the unit for use as rescue therapy in upper airway compromise, acute severe asthma and acute COPD.
- There are guidelines for the use of helium oxygen mixtures in the resources section of Moodle.

WEANING FROM IPPV

- The weaning process starts the moment IPPV is initiated.
- Allow the patient to make spontaneous breathing efforts (albeit assisted) at the earliest possible stage of respiratory support. This is the best way to recruit and retain basal and dorsal areas and thereby improves gas exchange permitting reductions in FiO_2 and peak pressures.
- As oxygenation improves, wean FiO_2 and PEEP sequentially.
- As hypercapnia improves, wean from MV / SIMV to PS. Then gradually reduce PS.
- Once established on PS, consider a daily trial of support withdrawal. This needs to be co-ordinated with daily sedation holds. Three methods are widely used, CPAP +5 cm H_2O, PS +5-7 cm H_2O with 0cms H_2O PEEP and just a T-piece. The usual duration is a maximum of 15mins, which if successful should prompt extubation.
- In patients who require prolonged support (> 5-7 days), tracheostomy is often performed. This can invariably be performed utilising a percutaneous technique on the ICU. The optimal timing and individual patient's risks and benefits need to be carefully considered.
- In tracheostomised patients, "training periods" of reduced or absent support can be employed as part of a daily weaning routine. The routine should be reviewed at least daily and titrated according to progress. Complete rest overnight and establishing a day night cycle, are essential.

ACUTE LUNG INJURY (ALI) AND ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS) [45]

ALI and ARDS are syndromes and represent a spectrum of disease. The syndrome is defined as: A specific form of lung injury with diverse causes, characterised pathologically by diffuse alveolar damage and pathophysiologically by a breakdown in both the barrier and gas exchange functions of the lung, resulting in proteinaceous alveolar oedema and hypoxaemia [46].

Diagnostic criteria:

- Identifiable cause or associated condition
- Hypoxaemia (usually refractory to supplemental oxygen)
- Bilateral, diffuse, interstitial and alveolar infiltrates on CXR (+/or CT)
- Reduced respiratory compliance (optional)
- No evidence for cardiac factors as the principle cause of the pulmonary oedema (PAWP ≤18mHg)
- Pulmonary hypertension (common)
- ALI - $PaO_2/FiO_2 < 40kPa$ ARDS - $PaO_2/FiO_2 < 26kPa$

Management

- Do the basics well, including:
  - Early and aggressive treatment of underlying condition
  - Optimal cardiovascular support with particular attention to fluid balance and use of a restrictive strategy [47]
  - Early enteral nutritional support
  - Minimise sedation and neuromuscular blockade
  - Adherence to ventilator care bundle
- Ventilatory strategy: “open the lung and keep it open” and minimise VILI
  - Low tidal volumes 6-8mls/kg ideal body weight
  - “Adequate” (high) PEEP (see earlier section)
  - Minimise peak / plateau pressures (≤30cmH2O)
  - Minimise FiO2
  - Maintain spontaneous breathing
- Unproven ventilatory rescue therapies
  - Prone positioning
  - Unconventional ventilation, in particular HFOV
  - Extracorporeal lung assist
- Adjunctive therapies with no evidence of benefit:
  - Inhaled nitric oxide [48]
  - Nebulised prostacyclin
  - Almitrine
  - Recombinant surfactant
- Adjunctive therapies under active investigation:
  - Sildenafil
  - Salbutamol [49]
- Controversies:
  - Open lung biopsy [50]
  - The role of high dose corticosteroids [51, 52]

CARDIOVASCULAR FAILURE AND SUPPORT

- The principle role of the cardiovascular system is to deliver oxygen and glucose to the tissues and remove carbon dioxide and other waste products. Failure of this principle role is termed shock.
- Clinical manifestations of cardiovascular insufficiency / shock include:
    - Low volume peripheral pulses
    - Cold peripheries, especially with proximal extension / peripheral capillary refill >2s (except in distributive shock states where peripheries way be warm with capillary refill time <2s)
    - Tachycardia and hypotension (not invariable and the least reliable signs)
    - Altered mentation / confusion / diminished level of consciousness (cerebral hypoperfusion)
    - Urine output <0.5 ml/kg/hr (renal hypoperfusion)
- When faced with a patient who has haemodynamic instability and / or shock, prompt clinical assessment is required and urgent efforts made to initiate cardiovascular support.
- This can be achieved by assessing and optimising four key components of the cardiovascular system in strict order (see Table 18). More than one component failure may exist. For example, a patient with septic shock may be in fast atrial fibrillation, be hypovolaemic, have myocardial depression and abnormally low arteriolar vascular tone, hence not merely distributive shock.
- Inotropic and / or vasopressor support should only be instituted after volume resuscitation. Exceptions to this suggestion include severe or drug induced hypotension.
- Remember, the heart is 2 pumps in series. Of the 2 pumps, the right heart usually has less physiological reserve than the left and is more vulnerable to acute decompensation.
- At an organ level shock may result in microcirculatory failure. This complex phenomenon arises from, among other processes, endothelial cell damage, formation of capillary occlusive microthrombi and neutrophil margination. This results in arterio-venous shunting and tissue ischaemia. This phenomenon is hard to monitor in almost all organs and no specific therapy exists to treat it.

- At a cellular level, shock may induce a variety of effects including, necrosis, apoptosis and hibernation. The later may manifest as reversible mitochondrial failure. This is currently an area of active research but again, monitoring and therapy remain distant goals.
- Measures of global oxygen delivery (DO_2) and consumption (VO_2) are useful in assessing the adequacy of resuscitation and include (see later for definitions):
    - direct measurements of DO_2 and VO_2
    - Central or mixed venous oxygen saturations ($ScvO_2$ and SvO_2 respectively)
    - Blood pH, lactate levels and calculated base excess.

Table 18: The four key assessable and treatable components of shock

Assessable and treatable components of shock	*Aetiology of shock*	*Therapy*	*Physiological targets*
Heart rate and rhythm	Brady-arrhythmia	Positive chronotropic drugs / pacing	HR >30 is rarely the cause of shock optimal mechanical efficiency is achieved at a heart rate of ~90/min in sinus rhythm, <<90 in any other rhythm
	Tachy-arrhythmia	Anti-arrhythmic drugs / cardioversion	
Preload (intravascular volume status)	Hypovolaemic shock	**RAPID** 3-5ml/kg fluid bolus (balanced crystalloid or colloid), repeated depending on physiological response)	- ≥15% increase in stroke volume / cardiac output implies fluid responsiveness. REPEAT until <15% increase. - ≥3cmsH2O increase in CVP implies fluid responsiveness. Cautiously repeat whilst also monitoring HR, BP, capillary refill etc. STOP if no improvement in global assessment of perfusion adequacy - [Hb] 8-10 g/dl & Hct >0.3
	Haemorrhagic shock	- **Hypotensive resuscitation until bleeding has been controlled.** **-** replacement of RBCs, clotting factors and platelets (1:1 +/- 1 ratio) - RAPID 3-5ml/kg fluid bolus (balanced crystalloid or colloid)	
Cardiac contractility	Cardiogenic shock SIRS / sepsis related myocardial depression	- Inotropic drugs - Intra-aortic balloon pump - Re-vascularisation - Structural repair	cardiac index (output per m^2 body surface area) ≥2.0 l/min/m^2. "Survivor physiology" 3.0-5.0 l/min/m_2
Afterload / vascular resistance (both pulmonary and systemic)	Obstructive shock (PE, tamponade)	Relieve obstruction	-..systolic blood pressure ≥90mmHg -..mean arterial pressure ≥60mmHg These are arbitrary targets that require individualisation to the patient and the pathology
	Distributive shock (septic, spinal, anaphylactic)	Vasopressor drugs	

TERMINOLOGY AND NORMAL VALUES

Note – normal value ranges are for healthy human adults at rest. There are no normal ranges for shocked patients. Therapeutic targets are best considered by assessing the dynamic response to an intervention and using all available measures of both organ specific and global hypoperfusion.

- Cardiac Output (CO) = SV x HR (normal range at rest 4-6l/min)
- Oxygen delivery (DO_2) is the amount of oxygen delivered to the tissues per unit time.
- DO_2 = CO x arterial oxygen content (CaO_2)
- DO_2 = (HR x SV) x (([Hb]/100 x 1.34 x SaO_2/100) + (PaO_2 x 0.000225))

where HR = heart rate in beats per minute, SV = stroke volume in ml
[Hb] = haemoglobin concentration in g/dl divided by 100 to convert to g/ml
1.34 is the maximum mls of O_2 each gram of Hb can carry if 100% saturated
SaO_2 = arterial oxygen saturations divided by 100 to convert from %
PaO_2 = the partial pressure of oxygen in arterial blood in kPa
0.000225 is ml of O_2 dissolved per ml of blood per kPa of O_2

- DO_2 = 950-1150ml/min
- It is worth noting that the major determinants of DO_2 are CO and [Hb].
- For standardisation between individuals all of these variables are commonly indexed to (divided by) body surface area, hence normal values of:
  SVI = 33-47 ml/m^2 CI = 2.2-3.5 l/min/m^2 DO_2I = 500-600 ml/min/m^2
- Oxygen Consumption (VO_2) is the amount of oxygen consumed by the tissues per minute. VO_2 = Cardiac Output x (arterial oxygen content) – (mixed venous oxygen content-SvO_2)
- VO_2 =180-320ml/min
- Mixed venous saturation (SvO_2) is a guide to the difference between oxygen being delivered to the tissues and its extraction or use. This is measured by taking a sample of blood from the distal port of a pulmonary artery catheter (PAC) and measuring the saturations using a blood gas analyser. If a PAC is not available, samples from a central venous catheter in the superior vena cava will provide an approximate guide. This variable is called central venous oxygen saturation ($ScvO_2$). The normal value is 70-75%. This measurement has been successfully used to guide resuscitation resulting in improved morbidity and mortality [27].

METHODS OF CONTINUOUS HAEMODYNAMIC MONITORING

An important principle with all types of monitoring is that care should be taken in the interpretation of individual data points. The dynamic response of measured variables to a therapeutic action is far more informative.

The insertions of peripheral arterial and central venous lines are common first steps in the monitoring of critically ill patients.

Invasive arterial and venous pressure monitoring

- Arterial cannulation allows real time blood pressure measurement, beat-to-beat display of the arterial waveform and facilitates regular arterial blood sampling.
- A modified Seldinger technique or direct cannulation can be used.
- Favoured sites are the radial and femoral arteries.
- The central venous line serves two important functions. Primarily, it provides information on right heart filling pressures and also allows estimation of mixed venous saturations. The other practical function is to act as vascular access. National Institute of Clinical Excellence (NICE) guidelines now recommend that insertion of CVP lines should be guided by ultrasound. Central venous pressures should correlate well with left ventricular filling pressures, however, this is not always so in the critically ill. Ischaemic cardiomyopathy, valvular pathology, pulmonary hypertension and pulmonary embolism are potential causes of disparity.
- A Seldinger technique is routine for multi-lumen catheters. Strict aseptic technique should be employed. Skin preparation with 2% chlorhexadine in 70% alcohol is recommended. Exit sites should be dressed so as to prevent contamination. Prior to any use, any access port should be cleaned with chlorhexadine. Lines should be removed at the earliest opportunity.
- Favoured sites are the internal jugular and subcalvian. The latter has a lower risk of line related bacteraemia but is associated with a high risk of complications at insertion and chronic stenosis and thrombosis. The femoral site should be avoided in the first instance whenever possible.

Oesophageal Doppler

- This technique relies on the phenomenon that sound undergoes a frequency shift with respect to a fixed receiver when the source is moving at a different

speed to the receiver. The degree of frequency shift is directly proportional to the speed of the sound source (The Doppler effect).

- A probe inserted 30-40cm down the oesophagus and an ultrasound beam is focussed on the blood flow in the descending aorta. In this area aorta lies parallel to the oesophagus and has predictable cross-sectional area. The frequency of reflected sound waves undergoes Doppler shift dependent on the speed of blood flow. By quantifying this shift and incorporating the estimated cross-sectional area and ejection time (stroke distance), the stroke volume can be calculated. The aortic stroke volume is a percentage of the actual cardiac output, which can be derived from patient normograms based on height and weight.
- The advantages of this system include relative ease of use, minimal invasiveness and the fact that this is the only method of haemodynamic monitoring to provide real time data based on the actual (and not derived) cardiac output.
- Conditions prohibiting use include severe aortic pathology, an intra-aortic balloon pump (IABP)and oesophageal disease.

Pulse contour analysis

- The pressure wave displayed by an invasive arterial line is amenable to pulse contour analysis. The pressure waveform is converted to a volume-time waveform. The area under the curve gives a derived estimate of beat-to-beat SV. Combination with the HR is used to calculate CO. For accuracy, several of the systems calibrate them estimates of CO using transpulmonary dilution methods.

CARDIOVASCULAR SUPPORTIVE THERAPIES

Rate and rhythm control

- Optimal cardiac efficiency in a semirecumbant adult with no active peripheral venous muscle pump and positive pressure ventilation is an HR of ~90/min in sinus rhythm. Rates of <70 or >110 are inefficient and all reasonable attempts should be made to reach the target rate of 90/min.
- If the patient is haemodynamically unstable treat as per the Advanced Life Support algorithm. DC cardioversion is relatively straightforward in intubated patients and can be considered early.
- If haemodynamically stable, correct electrolytes and perform a dynamic fluid challenge prior to drug treatment and / or DC cardioversion.
- The commonest arrhythmia in critically ill patients is atrial fibrillation (AF). In the majority of cases there is an identifiable iatrogenic precipitant (frusemide, insulin, salbutamol, inotropes etc). Consequently, aim to keep serum Mg^{2+} >0.9 mmol/l, & K^{+} > 4.4mmol/l
- If the onset of AF is known to have been within 12-24hrs consider DC cardioversion. If the AF has been paroxysmal, has been present for >24 hours or fails DC cardioversion consider the following drugs: digoxin (may control rate), esmolol, metoprolol, sotolol or amiodarone. Verapamil and flecanide are usually avoided as they are both profoundly negatively inotropic.

Intravenous fluid resuscitation, the dynamic fluid challenge or preload optimisation [20, 53].* ***MUST READ*** http://www.ics.ac.uk/downloads/2008112340_GIFTASUP%20FINAL_31-10-08.pdf

- These are interchangeable terms that should be used to describe the assessment of fluid responsiveness rather than as assessment of the adequacy of intravascular volume. It is performed by administering a rapid 100-500 ml bolus of IV fluid and measuring the instantaneous response in HR and stroke volume (and hence cardiac output), CVP and MAP. Of all of these variables, stroke volume is most useful measure.
- There is no proven benefit in using any particular crystalloid or colloid.
- With regard to CVP, 3 patterns of response are looked for, in patients with a starting CVP in the normal range of 4-8cmsH2O (caution in interpretation is required in patients receiving positive pressure ventilation).
    1. No or minimal response $\Rightarrow$ relative hypovolaemia.
    2. Gradual and sustained rise ($\geq$2cmsH_2O) followed, over a period of minutes, by a return towards, or even reaching, baseline $\Rightarrow$ relative euvolaemia.
    3. Rapid and sustained rise with little or no fall $\Rightarrow$ relative hypervolaemia.

The following further points are noteworthy.

- Ideally, a rapid fluid bolus should not be given via an automated volumetric pump. Such pumps usually have a maximal delivery rate of 999ml/hr, which is too slow to properly assess volume responsiveness.
- A volume challenge can be simulated without actually giving a fluid bolus by the method of passive leg raising [54].
- In patients receiving positive pressure ventilation, who demonstrate no inspiratory effort and are in sinus rhythm, a maximum variation in systolic or pulse pressure of greater than 15% over each respiratory cycle is a validated method of predicting volume responsiveness, as defined by an increase in stroke volume in response to a subsequent fluid bolus. Extrapolating this finding to patients, who fail to meet these strict criteria, is inappropriate and inaccurate. Many of the newer cardiac output monitoring devices (see below) measure maximal variation in either arterial pressures or stroke volume over a fixed time period with no reference to respiratory cycle monitoring or the presence, or absence, of the above criteria. Such measurements do not accurately predict volume responsiveness.

- The reason why, fluid responsiveness rather than volume status, has emerged as a central idea and target for cardiovascular monitoring and therapy is a combination of pragmatism and a belief in the virtue of preload optimisation. There is evidence to support the idea that early and aggressive fluid resuscitation is beneficial [27]. In the short term, fluid administration is simple, quick and often effective and iatrogenic fluid overload is rare. In responsive patients, fluid therapy increases global oxygen delivery for the smallest increase in myocardial work.

Inotropes, vasopressors and other vasoactive drugs

- If rate and rhythm are acceptable and / or resistant to fluid and electrolyte resuscitation, estimation and continuous monitoring of cardiac output are highly desirable to guide pharmacological therapy.
- If there is evidence to suggest a low or inadequate CO and / or perfusion pressure (MAP), then vasoactive drugs should be commenced. A simple guide to available agents, their pharmacodynamics and standard dosage regimens are detailed in the following Tables.
- Dopexamine or Dobutamine are the inotropes of first choice. They are both strongly positive chronotropes and arterial vasodilators. Alternatives include the inodilators milrinone and levosimendin. Due to the vasodilation these drugs cause, combination with a vasopressor is often required. All are pro-arrhythmogenic.
- Dopamine and epinephrine, both inoconstrictors, have lost favour over recent times for a complex series of reasons.
- In ischaemic cardiogenic shock, inotropic drugs should be considered as providing a temporal bridge to definitive revascularisation and / or mechanical support, most commonly an IABP.
- Norepinephrine is the first choice vasoconstrictor with weakly positive chronotropic and inotropic activity. Alternatives, other than dopamine and epinephrine include, vasopressin analogues [55] and methylene blue [56]. Excessive doses of vasopressors can lead to over centralisation of the circulation, left ventricular failure, splanchnic and distal extremity ischaemia / infarction.
- In immediate resuscitation, ephedrine, phenylephrine, metaraminol and 1:10,000 - 1:100,000 adrenaline (epinephrine) can be given in small bolus doses to maintain blood pressure.

- The management of hypertensive cardiovascular compromise is dependant upon the underlying cause. In the immediate management of decompensated left ventricular failure, intravenous nitrates are a useful first line agent in off loading the left side of the heart. This is predominantly via venodilation. Second line agents in hypertensive cardiovascular crises include hydralazine and labetalol.

"Low dose" corticosteroids and functional hypoadrenalism in shock

- There is evidence to suggest that a proportion of patients with distributive shock have functional hypoadrenalism. This is manifest as vasopressor resistance with patients requiring rapidly escalating / high dose infusions. Whether such patients have functional hypoadrenalism and / or peripheral resistance is unclear. Use of "physiological replacement" dose hydrocortisone (200-240mg/24hrs) remains controversial.
- Random cortisol and short ACTH stimulation tests are no longer performed as they do not predict responsiveness to replacement therapy.
- Pragmatic definitions of functional hypoadrenalism:
    - Patients with septic shock requiring high dose vasopressors. Defined as: ≥0.2 mcg/kg/min norepinephrine, who are not volume responsive (defined as a ≥10% increase in stroke volume following a 3 ml/kg fluid bolus administered in ≤ 5 min) and hyperdynamic (defined as a cardiac index ≥2.8 l/min/m^2). Patients with evidence of acute myocardial depression or chronic insufficiency should be considered separately.
    - Patients, who, having been stable for ≥2 hours on a dose of vasopressor, but develop increasing dose requirements (≥20% increase), unresponsive to a volume bolus (as above) and are hyperdynamic (as above).
    - Patients whose dose of vasopressor cannot be weaned ≥24 hours following initiation of appropriate broad spectrum antimicrobial therapy and / or effective source control.
- Pragmatic definition of "steroid responder": A patient who demonstrates a ≥20% decrease in vasopressor requirement to maintain the same mean arterial pressure, 4 hours after a 100mg bolus dose of hydrocortisone. Responders should be commenced on hydrocortisone infusions at 10mg/hr. This should be weaned 6 hours after successful withdrawal of vassopressor support

Table 19: Cardiovascular effects of vasoactive drugs and devices

	Receptor avidity				*Physiological effects*				
Drug	DA	α	B_1	B_2	HR	CI	MAP	SVR	MO_2
Dopamine									
<5μg/kg/min	+++	+	+		=/+	=/+	=/+	=	+
5-10μg/kg/min	+++	++	++	+	+	+	+	+	+
>10μg/kg/min	+++	+++	++	+	++	+	++	++	++
Dobutamine	+/-	+	+++	++	+	++	=/-	=/-	+++
Dopexamine	++		+	+++	++	+	=/-	=/-	++
Adrenaline									
<0.2μg/kg/min		+	+	+	+	+	+	+	++
>0.2μg/kg/min		+++	++	+	++	++	++	++	+++
Noradrenaline									
<0.2μg/kg/min		++	+	+/-	=/+	=/+	++	++	++
>0.2μg/kg/min		+++	+	+/-	=/+	=/+	+++	+++	++
Milrinone					++	++	- -	- -	++
Levosimendan					+/++	++	- -	- -	?=
Vasopressin / analogues					=	- / - -	+++	+++	++
Methyl blue					=	- / - -	++	++	++
IABP (device)						++	+	-	- -
Metoprolol			++	+	- / - -	- / - -	- / - -	- / - -	- / - -
Labetalol		++	++	+	- / - -	- / - -	- -	- -	- / - -
Amiodarone I									
Enteral					-	- / +	=	=	-
Parenteral					-	- -	-	-	-
Digoxin					-/=	=/+	=	=	=/+
GTN					=/+	-/+	-	-	-
Hydralazine					-/+	+	- -	- -	-
Sodium nitroprusside					=/+	-/+	-	-	-
Nimodipine					=/+	-/+	-	-	-

[Key: DA dopamine receptors; α B_1 B_2 adrenergic receptor types; MO_2 myocardial oxygen demand; + increased, = unchanged, – decreased; IABP intra-aortic balloon pump]

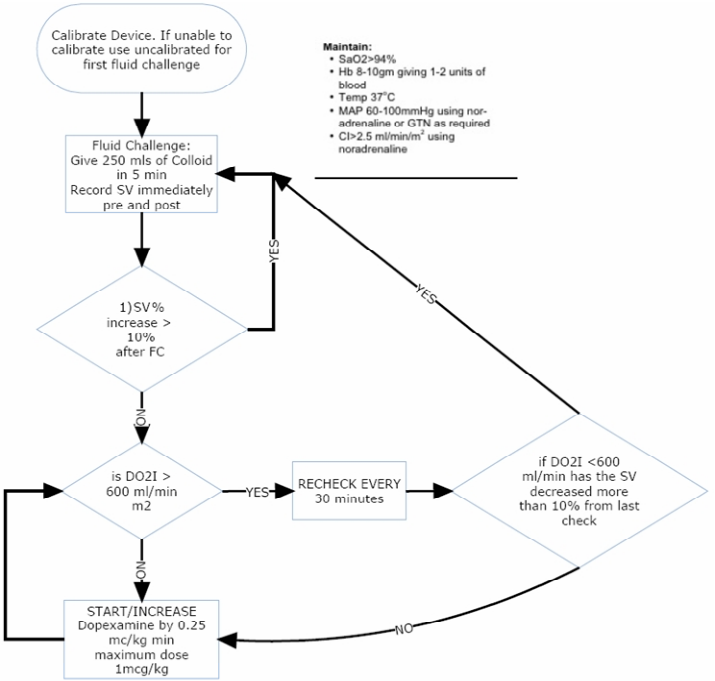
Table 20: Dosage regimens of commonly used vasoactive drugs

Drug	*Dosage range*	*Notes*
Dopamine **DON'T EVER USE THIS**	1-20 mcg/kg/min	Dominant effect dependant upon dose. Some evidence of worse outcome as compared to other drugs, therefore out of favour. Possible association with ICU delirium.
Dobutamine	5–20 mcg/kg/min	Inodilator
Dopexamine **1st line on GICU**	0.25-2.0 mcg/kg/min	Positive chronotrope / Inodilator / Anti-inflamatory?
Adrenaline (Epinephrine)	0.01–1 mcg/kg/min	Inoconstrictor. In resuscitation, use 1:100,000 peripherally (add 1mg to 100mls bag via volumetric pump or 0.5mg to 50ml syringe via driver)
Noradrenaline (Norepinephrine)	0.01–1 mcg/kg/min	Vasoconstrictor (some inotropic activity) 1st line vasopressor.
Milrinone (phosphodiesterase inhibitor)	150 - 750 ng/kg/min	Inodilator. Significantly longer onset and elimination half-life than dobutamine. Accumulates in renal failure. Do not give loading dose.
Levosimendan (Ca^{2+} sensitizer and K channel opener)	0.05–0.2 mcg/kg/min	Inodilator. Active metabolite with long elimination half life, hence 24hr infusion will have measurable effects for up to 7 days. Do not give loading dose.
Vasopressin	0.01-0.05 units/min	Vasoconstrictor. 2nd line vasopressor in norepinephrine resistant shock (see also Functional hypoadrenalism)
Terlipressin (vasopressin analogue)	0.25-2 mg bolus	Vasoconstrictor. Duration of action 4-6 hours. Alternative to vasopressin.
Methylene blue (nitric oxide antagonist)	loading 2 mg/kg maintenance 0.25-2 mg/kg/hr	Vasoconstrictor. 2nd/3rd line vasopressor in norepinephrine resistant shock (see also Functional hypoadrenalism)
GTN	0.5-30 mg/hr	Rapid tachyphylaxis over 12-24hrs
Hydralazine	Bolus 1-10 mg slowly Infusion 200-300 mcg/min reducing to 50-150 mcg/min	Arterial vasodilator
Labetalol	0.25-2 mg/min	Alpha and beta blockade
SNP	0.1-10 mcg/kg/min	Arterial and venodilator

Post-operative care of patients with a high risk of morbidity and mortality

- On the GICU this concept is often referred to as "optimisation" or "Goal directed therapy".
- It is easy to identify a group of patients with a high risk of post-operative morbidity and mortality. The more extensive the surgery, the more physiologically deranged the patient, be it acute, acute on chronic or merely chronic, the higher the risk. Many such patients are often referred to critical care environments for their immediate post-operative care.
- There is a large body of evidence to support the provision of cardiovascular optimisation of this group of patients.
- The concept arose from the observation that survivors had a different physiological profile to non-survivors. The median values of DO_2I and VO_2I in survivors was 600 and 170 ml/min/m^2 respectively [57].
- Maximising a high risk patient's DO_2I in the immediate post-operative period (for 8hours) using the therapies described above, in particular intravascular fluid responsiveness, with the addition of low dose inodilators where necessary, in an attempt to reach 600ml/min/m^2 has been demonstrated to be a successful strategy in reducing morbidity and mortality [58].
- DO_2I = Cardiac Index (CI) x Arterial oxygen content. Thus to increase DO_2I the important variables are stroke volume (SV), heart rate (HR) and haemoglobin concentration (Hb). Stroke volume is determined by end diastolic volume, heart rate, cardiac contractility and afterload. End diastolic volume is influenced by intravascular volume status, heart rate and rhythm.
- To increase SV optimise HR (~90bpm) and rhythm (sinus) and test for volume responsiveness with a rapid bolus of colloid using a direct measure of SV and surrogate markers (HR, BP, CVP). If not volume responsive consider manipulation of HR, cardiac contractility and afterload with appropriate pharmacological and non-pharmacological therapies. Always pay attention to analgesia, core body temperature and any pre-exiting limiting co-morbidities especially ischaemic heart disease.

Post operative haemodynamic goal directed therapy flow chart



THE SYSTEMIC INFLAMMATORY RESPONSE SYNDROME, SEPSIS, SEVERE SEPSIS AND SEPTIC SHOCK

CURRENT DEFINITIONS (2003) [59]

- The systemic inflammatory response syndrome (SIRS) became a defined clinical entity after the consensus conference of 1992. The term provided a reference for the complex findings that result from a systemic activation of the innate immune response, regardless of cause. The statement form that conference hypothesized that SIRS is triggered by localized or generalized infection, trauma, thermal injury, or sterile inflammatory processes, e.g. acute pancreatitis. SIRS is considered to be present when patients have more than one of the following clinical findings:
    - Body temperature > 38°C or < 36°C
    - Heart rate > 90/min
    - Hyperventilation evidenced by respiratory rate higher than 20/min or $PaCO_2$ < 4.2kPa
    - White blood cell count > 12,000 cells/ µl or < 4,000/ µl
- Sepsis is defined as SIRS with a documented or suspected infection. Severe sepsis is sepsis with evidence of organ dysfunction (as defined by SOFA or equivalent). Septic shock is defined as severe sepsis with hypotension despite adequate fluid resuscitation (see reference for definition of hypotension).
- Severe sepsis and septic shock are the commonest conditions requiring critical care. Their incidence is high and increasing. They are associated with a very high morbidity and an overall mortality of 30-60%. Despite the heterogeneity of precipitating events, these syndromes encompass a burden of disease equivalent to ischaemic heart disease and cancer. Accordingly, there have been major efforts over recent years to raise both awareness of these conditions and promote optimal and timely therapy. An obvious parallel can be drawn between this campaign and that created to promote revascularisation (thrombolysis and / or primary angioplasty) in acute ST elevation myocardial infarction.

THE SURVIVING SEPSIS CAMPAIGN'S RECOMMENDATIONS 2004 [60] (CURRENTLY UNDER REVIEW) www.survivingsepsis.org.
This international campaign is aimed at improving the care and hence outcome of all patients with severe sepsis / septic shock. It was launched in 2004 and represents a set of evidence based recommendations, detailed below:

Initial resuscitation - Goals during first 6 hours:

- Measure serum lactate
- Obtain blood cultures prior to antibiotic administration
- Administer broad-spectrum antibiotic(s), within 1 hour of admission
- In the event of hypotension and/or a serum lactate > 4 mmol/L
    - Deliver an initial minimum of 20 ml/kg of crystalloid or an equivalent
    - Commence vasopressors for hypotension not responding to initial fluid resuscitation to maintain MAP > 65 mm Hg
- In the event of persistent hypotension despite fluid resuscitation (septic shock) and/or lactate > 4 mmol/L
    - Continue fluid resuscitation until CVP $\geq$ 8mmHg
    - Continue resuscitation until $ScvO_2 \geq 70$ % $SvO_2 \geq 65$ %

Management goals within the first 24 hours

- Administer low-dose steroids for septic shock in accordance with a standardised ICU policy. If not administered, document why the patient did not qualify for low-dose steroids based upon the standardized protocol. Please note that this recommendation is currently under review in light of more recent evidence (see earlier section).
- Maintain blood glucose at $\geq$ 6mmol/l but $\leq$ 8mmol/l
- In mechanically ventilated patients, maintain the inspiratory plateau pressure at < 30 $cmsH_2O$.

Activated Protein C or drotrecogin alfa [61]

- In severe sepsis / septic shock there is activation of the coagulation cascade including the fibrinolytic pathway.
- It has been observed that there is a depletion of activated protein C and that lower levels are associated with a worse outcome.
- There is also evidence that protein C plays an important role in modulating the inflammatory process in addition to its role in the clotting system.
- This led to the development of recombinant human activated protein C (rhAPC) and a series of trials in patients with severe sepsis and septic shock. The first of these trials [62] remains the subject of much controversy but continues to be used as the basis for administering this drug. Subsequent trials and observational studies have raised doubts regarding both the safety and efficacy of rhAPC.
- Current indications for its use are patients at high risk of death (APACHE II $\geq$ 25) from sepsis-induced multiple organ failure (2 or more organs). It must be administered within 24 hours of the onset of organ dysfunction.
- It is contraindicated in patients with active internal bleeding, intracranial pathology, concurrent heparin use or significant thrombocytopenia (platelet count $\leq$ 30x109/l).
- Dosage - 24mcg/kg/hr (based on actual body weight) for 96 hours

BRAIN INJURY AND SUPPORT

Regardless of the nature of brain injury certain universal principles of care apply. In essence, minimise secondary brain injury and optimise any penumbral chance of recovery.

- First and foremost adopt an ABC approach to resuscitation.
- Aim for normoxia, normocarbia, normotension, normothermia, normoglycaemia and normonatraemia. In particular, hypercarbia, hyperthermia, hyponatraemia, and both hypo and hyperglycaemia are associated with secondary brain injury. There is no therapeutic benefit in hypocarbia.
- Position the patient 30º head up. This is considered the best compromise between the increased gravitational gradient placed on the arterial pressure and enhanced gravitational gradient for venous drainage. Try to avoid any intervention that may reduce or obstruct cerebral venous return e.g. internal jugular lines, high intrathoracic pressures (high mean airway pressures) created by mechanical ventilation
- With regard to blood pressure, the concept of cerebral perfusion pressure (mean systemic pressure – intracranial pressure (ICP)) is helpful. A minimum target CPP of 60mmHg is considered optimal. A lower CPP is associated with a higher incidence and extent of secondary brain injury. If possible, measure and continuously monitor ICP.
- Medical management of raised ICP includes:
    - Aggressive normalisation of $PaCO_2$ and core temperature.
    - Patient positioning (see above)
    - High dose propofol or thiopentone infusion (barbiturate coma) to reduce cerebral metabolic demand.
    - Aggressive management of seizures.
    - Hypertonic saline and / or mannitol.
    - Therapeutic hypothermia to 32-34°C
- Surgical management of raised ICP includes:
    - CSF drainage (placement of an external ventricular drain)
    - Decompressive craniectomy

BRAIN STEM DEATH

- This term may have different definitions in different countries.
- It is an emotive area that is often difficult to communicate and understand for family and staff at the bedside. In the United Kingdom this term refers to 'the irreversible loss of the capacity for consciousness combined with the irreversible loss of the capacity to breath.' This may be anatomically defined as brain stem death. In different states in the United States brain death also requires cardiopulmonary death as part of the definition.
- The process in diagnosing brain death is divided into three parts:

Preconditions

- The patient must be in an apnoeic coma and on a ventilator unable to make spontaneous breathing effort.
- The presence of irremediable structural brain damage contributing to brain stem death should be present, for example traumatic head injuries or haemorrhage.

Exclusions

- Hypothermia - core temperature greater than 35ºC
- Metabolic or endocrine derangement must be excluded. (e.g. hypoglycaemia)
- Poisoning, use of neuromuscular drugs, and sedatives must be excluded and accounted for.

BRAIN STEM DEATH TESTS

These should be performed and repeated by two separate senior doctors (ideally one should be a consultant or equivalent). This may be done on separate occasions or together. The two doctors should not be part of a transplantation team and should be competent in the area. The time of death is time when the first set of tests confirms the presence of brain stem death.

- Oculocephalic Reflex: This should be absent. This is tested by rolling the head from side to side. In a functioning brain stem the eyes will move relative to the orbit (Dolls eyes present). In brain stem death the eyes move with the head in the direction of travel of the movement (Dolls eyes absent).
- Pupillary Reflex: Pupils are fixed and unresponsive to light stimulus. Direct and consensual light reflexes are absent. Pupils may or may not be dilated.
- Corneal Reflexes: Cotton wool may be used to illicit a response to corneal stimulation by light touch. This response will be absent
- Vestibulo-ocular Reflexes: Caloric testing is used to assess function of the labyrinth, it is essential that the tympanic membrane is visualised prior to the test. Fifty mls of ice cold water is injected into the left ear over one minute. Intact brain stem response will elicit nystagmus with fast movements to the right (away from the injected ear). The opposite response will take place in the intact brain stem of an individual if water is injected into the right ear. In patients with brain stem death no response to ice cold water injection is seen.
- Gag and Cough Reflex: Pharyngeal, laryngeal (throat spatula) and tracheal (suction catheter down the endotracheal tube to carina) stimulation would normally illicit a cough / gag response. This is absent in brain stem death.
- Motor Reflex: Centrally or peripherally applied painful stimulus would normally illicit a motor response in the cranial nerve territory. In brain stem death this is absent.
- Apnoea testing: Intact brain stems will initiate spontaneous respiratory effort in the presence of hypercarbia. The patient is placed on 100% oxygen on the ventilator and pre-oxygentaed for 10 minutes. The minute volume should be reduced to achieve a $PaCO_2$ > 5kPa before the test. The patient is then disconnected from the ventilator and oxygen is attached to the endotracheal tube at 6l/min (to maintain oxygenation in the presence of hypercarbia). The $PaCO_2$ must rise to greater than 6.65kPa. The chest is visualised and inspected for movements. In brain stem death no movements are present and the patient is then re-connected to the ventilator.

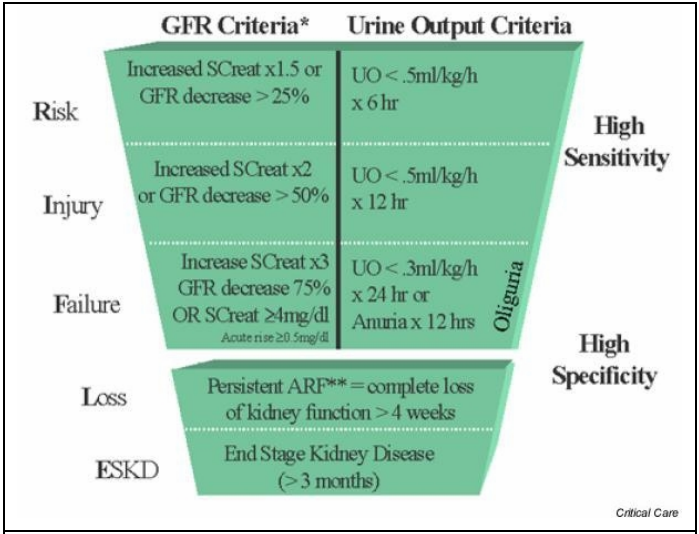
Organ and tissue donation:
In the UK, organ and tissue donation are governed by the Human Tissue Act 2004 (see http://body.orpheusweb.co.uk/HTA2004/20040030.htm). Always consider donation in dying patients, especially in those with brain stem death and, in particular, in the brain injury is the sole organ failure. In the UK, to discuss any aspect of donation, contact your local transplant co-ordinator through your hospital switchboard.

RENAL FAILURE AND SUPPORT

- Acute renal injury is a common complication of critical illness.
- In 2004, an expert panel proposed the so-called RIFLE severity of injury criteria of at Risk, with Injury, with Failure, with sustained Loss and with End-stage status [63] see Figure.
- Renal injury is associated with increased morbidity and mortality regardless of the primary pathology, with a direct correlation between the severity of renal injury and poor outcome.
- Clinically, oliguria is the first presenting sign. This may lead promptly to anuric renal failure.
- Prevention, by maintaining intravascular volume and adequate renal perfusion pressure together with the avoidance of nephrotoxic drugs e.g. NSAIDs, in the at risk population is well established.
- X-ray intravascular contrast is another potent precipitant of acute renal injury. Prevention with intravenous prehydration is well established. The role of N-acetyl cysteine and its optimal dosing regime is emerging as is the role of sodium bicarbonate [5].

MEDICAL MANAGEMENT OF ACUTE OLIGO/ANURIC RENAL FAILURE

- "Low dose" dopamine has no place in the prevention or treatment of acute renal failure [64].
- Frusemide may aide in the management of fluid overload in non-oliguric patients but has no effect on disease progression or outcome of acute oligo/anuric renal failure [65].
- Optimise renal perfusion (intravascular volume, cardiac output, perfusion pressure)
- Initiate renal replacement therapy early, specifically to manage acidosis (pH < 7.2, in particular, acidosis associated with cardiovascular compromise), hyperkalaemia (K^+ > 6.5), uraemia (urea > 35mmol/l) or fluid overload. Other indications include encephalopathy, hyperpyrexia, and possibly in vasopressor resistant septic shock.



Proposed classification scheme for acute renal failure (ARF). The classification system includes separate criteria for creatinine and urine output (UO). A patient can fulfill the criteria through changes in serum creatinine (SCreat) or changes in UO, or both. The criteria that lead to the worst possible classification should be used. Note that the F component of RIFLE (Risk of renal dysfunction, Injury to the kidney, Failure of kidney function, Loss of kidney function and End-stage kidney disease) is present even if the increase in SCreat is under threefold as long as the new SCreat is greater than 4.0 mg/dl (350 µmol/l) in the setting of an acute increase of at least 0.5 mg/dl (44 µmol/l). The designation RIFLE-FC should be used in this case to denote 'acute-on-chronic' disease. Similarly, when theRIFLE-F classification is achieved by UO criteria, a designation of RIFLE-FO should be used to denote oliguria. The shape of the figure denotes the fact that more patients (high sensitivity) will be included in the mild category, including some without actually having renal failure (less specificity). In contrast, at the bottom of the figure the criteria are strict and therefore specific, but some patients will be missed. *GFR = Glomerular Filtration Rate; ARF Acute Renal Failure. Copied from [63]

RENAL REPLACEMENT THERAPY

- Continuous renal replacement therapy (CRRT) is performed either as haemofiltration (convection - CVVHF), or as haemodiafiltration (convection and diffusion - CVVHDF) i.e. haemofiltration with a counter current for solutes to diffuse down.
- Dose: there is some evidence that higher rates of filtration produce better outcomes in patients with severe sepsis. Aim for 35 ml/kg/hr minimum. More if the patient will tolerate it. A good starting point is 2,000- 2,500 ml/hour of CVVHF.
- Replacement fluids are usually bicarbonate based buffer solutions with a fixed concentration of potassium. Alternatives include acetate buffered solutions. If using high volume replacement be aware of increasing the ventilatory demands on the patient, hypomagnesaemia and hypophosphataemia.
- Make a fluid balance plan indicating how much, if any, fluid to take off.
- Consider intermittent haemodialysis (iHD) in haemodynamically stable patients. In the acute phase of critical illness, daily iHD has been shown to be of greater benefit than alternate day treatment. Patients must have their hepatitis B and C status established prior to commencing therapy as there is a risk of cross infection.
- Approxiamtely 80-90% of patients with acute renal failure who require renal support will recover some if not all renal function at three months

CIRCUIT ANTICOAGULATION [66]

- Extra-corporeal circuits require anticoagulation.
- Unless the patient is known or suspected to have a hypersensitivity to unfractionated heparin, circuits should be primed with a dilute heparin solution, which avidly binds to the circuit tubing.
- Once connected, first line therapy is a continuous infusion of unfractionated heparin into the proximal end of the circuit. The usual dose is 500-1,000 units per hour. Contraindications include thrombocytopaenia (Plts < 50) of any cause.
- Second line therapies include continuous infusions of epoprostenol, low molecular weight heparin, citrate and lepirudin.

- In problematic circuits, minimising the procoagulant stimulus by maximising the blood pump speed and prediluting the patient's blood with the replacement fluid should also be considered.

TRAUMA

- St George's is the major (level 1) trauma centre for South West London and North Surrey. At present, St George's receives 2-4 multiply injured patients per week. This is likely to increase to 1 per day.
- More than any other group of patients, those with polytrauma require "time critical" care.
- The GICU SpR is responsible for arranging the timely admission of these patients to the most appropriate ICU. The guideline (overleaf) should assist you. If in doubt, contact the GICU consultant.
- You are responsible for co-ordinating the care of these patients with all of the other teams involved. There are always logistical difficulties so be proactive.
- For practical assistance liaise with the trauma nurse co-ordinator on bleep 8091.

QUICK ICU TRAUMA PATIENT CHECKLIST
(*see* http://emedicine.medscape.com/article/434445-overview *for more detail*)

A. Airway secured. COETT (if present) long enough to safely allow for increased facial swelling). C-spine control – *see* spinal clearance checklist
B. Assisted ventilation as for lung injury (Vt 6ml/kg IBW etc)
C. Haemodynamically stable – all sources of haemorrhage identified and controlled (surgery + / or interventional radiology). Coagulopathy actively managed – 1:1 RBC to FFP transfusion + / - platelets. Fibrinogen >2.0 g/dl, ionised Ca^{++}>1.0 mmol/l (on blood gas). Temp >36.5.
D. If GCS≤13 treat as diffuse head injury (see earlier section). Complete the spinal clearance checklist.
E. Ensure secondary and tertiary surveys completed at earliest possible opportunity and documented in trauma booklet. For each injury there must be a management plan.

ST GEORGE'S HOSPITAL CRITICAL CARE REFERRAL GUIDELINES FOR TRAUMA PATIENTS

Patients with ANY of the following criteria should be discussed with the GICU SpR via pager 7980 or extension 1307.

Patient criteria

- >65 years of age with 1 or more major injuries (NOT including # neck of femur)
- Any limiting / severe co-morbidities

ABCD (physiological) criteria

A. Injury to, or that might compromise, the airway
B. Hypoxaemia and / or hypercapnia post resuscitation
C. Haemodynamically unstable (persistent tachycardia and / or hypotension post resuscitation and surgical control of bleeding)
D. GCS ≤13 (any cause) post resuscitation

Injury criteria

- 2 or more major injuries
- Suspected or proven, unstable, spinal injuries

Treatment criteria

- >4 units of packed RBC transfusion during resuscitation
- >2 hours in theatre

Post treatment criteria

- Any significant acute organ or metabolic dysfunction post resuscitation
- High risk of deterioration or complications

The GICU SpR will decide (in consultation with the GICU consultant, if necessary) which of the 3 adult ICUs the patient should be admitted to. As a general guide:

- Isolated head or spinal injuries should go to Neuro ICU (whether they need neurosurgery or not).
- Isolated chest injuries should go to CTICU.
- Polytrauma patients should go to GICU.
- Polytrauma patients requiring neurosurgical intervention (craniotomy) and / or intra-cranial pressure monitoring and management, can be managed on either Neuro ICU or GICU and each case should be judged on it own merits.

In order to take an acute admission onto any of the 3 units, a stable patient can be transferred to one of the other units to create a bed for the acute admission.

Immediate secondary transfers (from base hospital A&E) should be delivered to St George's A&E and the patient treated in identical manner to primary reception:

- Please inform GICU SpR at the earliest opportunity.
- Immediate lack of an ICU bed should not delay transfer to St George's (Vascular surgery model).

Delayed secondary transfers requiring 1 or more surgical specialties and ICU / HDU care:

- Whoever takes referral must get a comprehensive list of injuries, co-morbidities and current clinical state (GICU & Pelvic surgery template).
- Team accepting patient must liaise with trauma team and other specialist teams at the earliest opportunity.
- Patients should arrive with radiological spinal clearance / diagnosis and appropriate immobilisation. (ICS guidelines)
- Inform the relevant ICU of the patient and the acuity of the need to transfer.
- Ensure all radiology travels with patient.
- Ensure tertiary survey completed within 24 hours of patient arrival.

Spinal Clearance Checklist

Patient's Name	
Date of birth	
St G. Hosp. No.	

To be fully completed at admission clerking and amended over time.

1. Given the mechanism of injury is there a risk of spinal injury? If uncertain, then the answer is YES. Are symptoms or signs of spinal injury reported or evident (from history, medical notes, secondary or tertiary survey)?

	Risk		Symptoms & / or signs of injury (bony & / or neurological)		Date	By whom (PRINT)
	No	*Yes*	*No*	*Yes. Symptoms / signs were …*		
C-spine						
T & L spine						

2. Plain x-rays (or CT scanograms). Have they been performed? Are they adequate? Have they been reported by a radiologist OR consultant? Is the spine radiologically cleared or are there injuries noted?

	Performed		Adequate		RADIOLOGICAL CLEARANCE		Date	By whom (PRINT)
	No	*Yes*	*No*	*Yes*	*Yes*	*No. Injuries are … Stable / unstable*		
C-spine								
T & L spine								

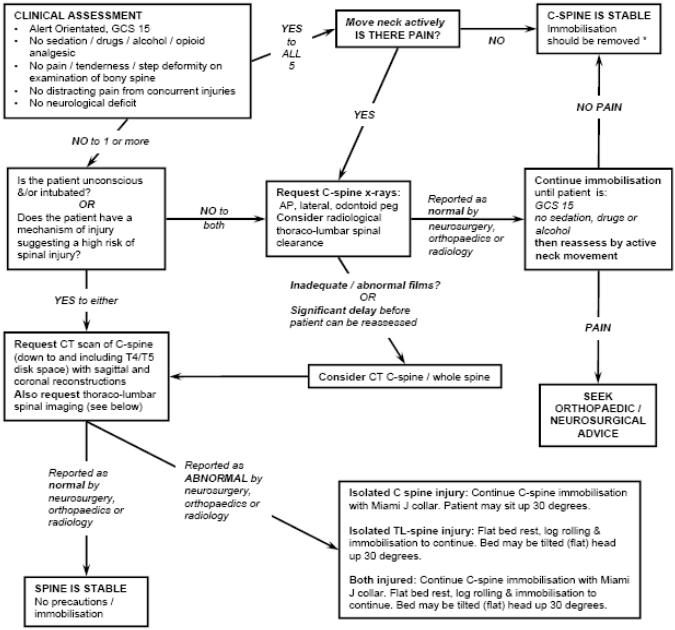
3. CT scans with planar reconstruction. Are these necessary? Have they been performed? Have they been reported by a radiologist OR consultant? Is the spine radiologically cleared or are there injuries noted?

	Necessary		Performed		RADIOLOGICAL CLEARANCE		Date	By whom (PRINT)
	No	*Yes*	*No*	*Yes*	*Yes*	*No. Injuries are … Stable / unstable*		
C-spine								
T & L spine								

4. **Management plan.** It is intended that the plan will progress to no precautions over time.

Precautions (circle)	Details	Time & Date	Name (PRINT)	Sign
Full	Miami J collar / spinal mattress / log roll / scoop stretcher / supine			
Limited / special instructions				
None				
Updates / changes				

SPINAL CLEARANCE FLOW DIAGRAM



Thoracolumbar spinal assessment
Image the TL-spine if **ANY** of the following apply:

- Given the mechanism of injury, is there a risk of thoracic and / or lumbar spine injury?
- Is there pain, bruising, swelling, deformity or abnormal neurology attributable to the thoracic or lumbar spinal regions?
- Is there a fracture anywhere else in the spine?
- Is the patient unconscious?

AP and lateral films *OR* CT scanograms (preferably at time of CT C-spine) may be adequate. If not, request CT whole spine.

****Close observation is required during mobilisation (removal of immobilisation). Development of weakness, paraesthesia or pain may indicate a missed injury***

Neurological deficit referable to spine injury requires CONSIDERATION of urgent MRI

GICU FORMULARY (see also specific sections)

The GICU IV drug guide is available on Moodle, in the *Resources* section under the *Pharmacology and poisons* heading.

The following brief guide is laid out in sections in the order used by the BNF.

GIT

Indication	*Rx*	*Notes*
Routine aperients	Sodium docusate 200mg 12hrly NG + Sennakot 15mls 12 hrly NG	
Aperient in liver failure	Lactulose 20-30mls 8-12hrly NG	
Prokinetics STOP once enteral feed established for >24hrs	Metaclopromide 10mg 8hrly IV & / OR Erythromycin 250mg 8hrly IV	 Only prokinetic at low dose
Suspected or confirmed upper GI bleed	Omeprazole 80mg IV over 1hr then 8mg/hr for 71 hrs	
Suspected or confirmed variceal haemorrhage	Terlipressin 2mg IV followed by 1 or 2mg every 4 to 6 hours until bleeding is controlled, for up to 72 hours.	
Constipation / pseudo-obstruction / ileus	Neostigmine 0.4-0.8mg/hr for 24 hours [67] OR Neostigmine 2mg IV bolus over 3-5mins [68]	 5mg in 50mls NS @ 4-8ml/hr

Dosage regimens of commonly used vasoactive drugs

Drug	*Dosage range*	*Notes*
Dopamine	1-20 mcg/kg/min	Dominant effect dependant upon dose. Some evidence of worse outcome as compared to other drugs, therefore out of favour. Possible association with ICU delirium.
Dobutamine	5–20 mcg/kg/min	Inodilator
Dopexamine	0.25-2.0 mcg/kg/min	Positive chronotrope / Inodilator / Anti-inflamatory?
Adrenaline (Epinephrine)	0.01–1 mcg/kg/min	Inoconstrictor. In resuscitation, use 1:100,000 peripherally (add 1mg to 100mls bag via volumetric pump or 0.5mg to 50ml syringe via driver)
Noradrenaline (Norepinephrine)	0.01–1 mcg/kg/min	Vasoconstrictor (some inotropic activity) 1st line vasopressor.
Milrinone (phosphodiesterase inhibitor)	150 - 750 ng/kg/min	Inodilator. Significantly longer onset and elimination half-life than dobutamine. Accumulates in renal failure. Do not give loading dose.
Levosimendan (Ca^{2+} sensitizer and K channel opener)	0.05–0.2 mcg/kg/min	Inodilator. Active metabolite with long elimination half life, hence 24hr infusion will have measurable effects for up to 7 days. Do not give loading dose.
Vasopressin	0.01-0.05 units/min	Vasoconstrictor. 2nd line vasopressor in norepinephrine resistant shock (see also Functional hypoadrenalism)
Terlipressin (vasopressin analogue)	0.25-2 mg bolus	Vasoconstrictor. Duration of action 4-6 hours. Alternative to vasopressin.
Methylene blue (nitric oxide antagonist)	loading 2 mg/kg maintenance 0.25-2 mg/kg/hr	Vasoconstrictor. 2nd/3rd line vasopressor in norepinephrine resistant shock (see also Functional hypoadrenalism)
GTN	0.5-30 mg/hr	Rapid tachyphylaxis over 12-24hrs
Hydralazine	Bolus 1-10 mg slowly Infusion 200-300 mcg/min reducing to 50-150 mcg/min	Arterial vasodilator
Labetalol	0.25-2 mg/min	Alpha and beta blockade
SNP	0.1-10 mcg/kg/min	Arterial and venodilator

Respiratory

Drug	*Dosage range*	*Notes*
Salbutamol	2.5mg nebulised **maximum** 2 hourly after loading with a **maximum** of 10mg in the first hour.	A 50:50 racemic mixture of R and S isomers. R-salbutamol is a bronchodilator. S-salbutamol is a bronchoconstrictor. The S isomer has a significantly slower elliminination from the lungs than the R isomer. HENCE, salbutamol is TOXIC to asthmatics in overdose. IV salbutamol has no proven efficacy, especially NOT as a rescue therapy in acute severe asthma. It also causes lactic acidosis and hypokalaemia (an excellent therapy in acute renal failure)
Ipratopium	250-500mcg nebulised 4-6 hourly	Bronchodilator
Budesonide	1mg nebulised bd	Inhaled steroid for intubated / ventilated patients.
Carbocysteine	1.5-2.5g/day in divided doses	Mucolytic, preferred to nebulised n-acetylcysteine
Hypertonic saline	5% NaCl 500ml polyfusor (from Neuro ICU) *OR* Add 10 x 10mls of 30% NaCl to 400ml of 0.9% NaCl (remove 100mls from a 500ml bag first) 5ml nebulised 4-6 hourly	*Probably* the best therapy for thick secretions

CNS

Continuous infusion sedative analgesic regimes

Drug	*Regime*	*Notes*
Morphine	Loading 5 - 15 mg Maintenance 1 - 12 mg/hr	Slow onset. Long acting. Active metabolites. Accumulates in renal and hepatic impairment.
Fentanyl	Loading 25 - 100 mcg Maintenance 25 - 250 mcg/hr	Rapid onset. Modest duration of action. No active metabolites. Renally excreted. Patches available for longer term use.
Alfentanil	Loading 15 - 50 mcg/kg Maintenance 30 - 85 mcg/kg/hr (1 - 6 mg/hr)	Rapid onset. Relatively short acting. Accumulates in hepatic failure.
Remifentanil	Dose 0.4 – 45 mcg/kg/hr	Rapid onset and offset of action with minimal if any accumulation of the weakly active metabolite. Significant incidence of problematic bradycardia.
Clonidine	Dose 1-10 mcg/kg/hr	An α_2 agonist. Has sedative and analgesic effects. Infusion doses up to 25 mcg/kg/hr AND slow bolus doses of 10-20 mcg/kg have been described as being safe with a surprisingly low incidence of hypotension and bradycardia [10].
Ketamine [11, 12]	Analgesia 0.2 mg/kg/hr Induction 0.5 - 2.0 mg/kg Maintenance 1 - 2 mg/kg/hr	Atypical analgesic with hypnotic effects at higher doses. Sympathomimetic; associated with emergence phenomena when given at hypnotic doses when usually co-administered with a benzodiazepine. Potentially useful adjunct to opiates (opiate sparing) as part of a mixed regime

Continuous infusion sedative regimes

Drug	*Regime*	*Notes*
Propofol 1%	Loading 1.5 - 2.5 mg/kg Maintenance 0.5 - 4 mg/kg/hr (0 - 200 mg/hr)	Intravenous anaesthetic agent. Causes vasodilatation and hence hypotension. Extra hepatic metabolism, thus does not accumulate in hepatic failure. Has no analgesic properties. Propofol infusion syndrome is a serious complication of prolonged and high dose administration with a significant fatality rate [13].
Midazolam	Loading dose 30 - 300 mcg/kg Maintenance 30 - 200 mcg/kg/hr (0 - 14 mgs/hr)	Short acting benzodiazepine. Used with morphine. Active metabolites accumulate in all patients especially in renal failure.

Regular / bolus dose analgesia

Drug	*Regime*	*Notes*
Paracetamol	1 g NG / PO 6 hourly or 1 g IV 6 hourly	Starting regime for simple analgesia Only use IV if enteral route unavailable / unreliable OR as part of an opiate sparing regime. Note 1g IV paracetamol ≡ 2.5 – 5mg IV morphine
Diclofenac	50 mg NG / PO 8 hourly or 75 mg IV 12 hourly	As part of an opiate sparing regime BUT only in well hydrated patients with normal renal function. Usually requires PPI cover. NSAIDs may have a role in reducing hypertropic acetabular ossification post acetabular fracture repair.
Codeine, Dihydrocodeine Oramorph	Starting regime: Oramorph 2.5 – 10mg PRN Max. 60mg / 24 hrs	Essentially the same drug (codeine is metabolised to morphine BUT only by 70% of the population). Use regularly in post-op patients to wean from PCA infusions. Avoid in renal failure. Patient must receive aperients. Oramorph is the preferred agent.
Oxycodone	5 – 30 mg NG / PO 4 -12 hourly	Safer in renal failure as extensive hepatic metabolism to less active drug.
Methadone	Start 15-30 mg NG / PO daily	Useful daily opiate. Can prolong QT interval.
Tramadol	50 – 100 mg 6 hourly	**PLEASE AVOID**. Mixed weak opiate and noradrenaline re-uptake inhibitor. Highly emetogenic, causes delirium, especially in elderly, and SIADH. Multiple drug interactions therefore contra indicated in patients on any antihypertensives, SSRIs, tricylics and warfarin.

Regular / bolus dose sedation

Drug	*Regime*	*Notes*
Diazepam	2 – 10mg NG / PO / IV PRN	Lipid emulsion. If giving regularly interval 6 12 hourly. Active metabolites accumulates quickly.
Lorazepam	0.5 – 4mg s/l / NG / PO / IV PRN	Tablets work well s/l. IV preparation is in ethylene glycol. Give 8 – 12 hourly. Fewer active metabolites / more predictable half life in multiple organ failure (~14 hours) compared to diazepam.
Haloperidol	2.5 – 5mg NG / PO / IV	Often delayed onset of action in patients with agitated ICU delirium. Can prolong QT interval.
Chlorpromazine	10 – 250mg NG / PO / IM	Alternative to haloperidol.
Olanzapine	5 – 15 mg NG / PO daily	Alternative to haloperidol.
Risperidone	1 – 4mg NG / PO / s/l	Alternative to haloperidol.

Neuromuscular blocking drugs

Drug	*Bolus dose*	*Onset & Duration*	*Side effects*
Suxamethonium	1 - 2 mg / kg Ampoule 100 mg	30 s 5 mins	Depolarisation. Histamine release. Elevation of plasma K^+ by ~1 mmol / l hence contraindicated in hyperkalaemia.
Atracurium	0.3 - 0.6 mg / kg Ampoule 50 mg	90 - 120 s 60 mins	Racemic mixture. Broken down by serum esterases hence predictable pharmacokintics in renal and hepatic failure. Causes histamine release hence contra-indicated in acute severe asthma. Inactive metabolite, laudanosine, lowers seizure threashold
Vecuronium	0.08 - 0.1 mg / kg Ampoule 10 mg	60 - 120 s 20 - 60 mins	Lipid soluble *hence* accumulates.
Rocuronium	0.6 mg / kg Ampoule 50 mg	< 60 s 30 - 60 mins	Most rapid onset of non-depolarising blockers. Low incidence of histamine release. Low, but significant incidence of anaphylaxis.

For more detailed information on neuromuscular blocking drugs see [34-36]

ANTIMICROBIALS

Empirical therapy policy April 2010

Notes

- Empirical antibiotics must be stopped after 72hrs unless there is clear clinical and microbiological evidence to continue treatment.
- All doses are IV unless otherwise stated. Switch to enteral route at earliest opportunity. Usual criteria for switch are:
    - Temperature less than 38°C for 48hours
    - Oral food/fluids tolerated, with no evidence of impaired absorption
    - Patient is clinically stable with improving clinical parameters such as WBC count & CRP
    - Not treating infections that require high antibiotic tissue concentration such as endocarditis, meningitis, necrotising fascitis, mediastinitis, brain abscess etc.
- Patients discharged from ITU on antibiotics should have the intended duration of treatment written in the notes and drug chart.
- Clindamycin, cefotaxime, ceftazidime and ciprofloxacin- switch to appropriate oral agents that are less likely to cause C.difficile diarrhoea prior to discharge Treatment should be discussed with microbiology on a daily basis.

Indication	*1st line*	*If penicillin allergic*	*Duration*
Community acquired pneumonia	Benzyl penicillin 1.2g 4hrly + Clarithromycin 500mg 12hrly	Ertapenem 1g daily + Clarithromycin 500mg 12hrly	5-10 days
Hospital acquired pneumonia	Co-amoxiclav 1.2g 8hrly + Gentamicin 5mg/kg daily	Meropenem 1g 8hrly + Gentamicin 5mg/kg daily (if shocked)	7-10 days
Ventilator associated pneumonia	Tazocin 4.5g 8hrly + Gentamicin 5mg/kg daily (if shocked)	Meropenem 1g 8hrly + Gentamicin 5mg/kg daily (if shocked)	5- 10 days
If MRSA colonised	Tazocin 4.5g 8hrly + Vancomycin 500mg-2g/24hr + Rifampicin **PO** 600mg 12hrly	Meropenem 1g 8hrly 1g 8hrly + Vancomycin 500mg-2g/24hr + Rifampicin **PO** 600mg 12hrly	5-10 days
Aspiration pneumonitis	Co-amoxiclav 1.2g 8hrly	Ertapenem 1g daily	5-10 days
Intra-abdominal sepsis	Amoxicillin 1g 8hrly + Gentamicin 5mg/kg daily + Metronidazole 500mg 8hrly	Ertapenem 1g daily + Gentamicin 5mg/kg daily + Metronidazole 500mg 8hrly	5-10 days
Necrotising pancreatitis	Meropenem 1g 8hrly	Ciprofloxacin 400mg 12hrly + Metronidazole 500mg 8hrly	5-10 days

Indication	*1st line*	*If penicillin allergic*	*Duration*
Polytrauma Orthopaedic only	Benzyl penicillin 1.2g 6hrly + Flucloxacillin 1g 6hrly + Gentamicin 5mg/kg daily (if shocked) + Tetanus prophylaxis	Clindamycin 600mg 6hrly + Gentamicin 5mg/kg/daily (if shocked) + Tetanus prophylaxis	3-5 days
Polytrauma Including abdominal trauma	Co-amoxiclav 1.2g 8hrly + Gentamicin 5mg/kg daily + Tetanus prophylaxis	Meropenem 1g 8hrly Tetanus prophylaxis	5-10 days
Skin /Soft tissue Infections	Benzylpenicillin 1.2g 4hrly + Flucloxacillin 1-2g 6hrly	Clindamycin 600mg-1.2g 6hrly	5-10 days
Necrotising soft tissue infection	Benzylpenicillin 1.2g 4hrly + Clindamycin 600mg 6hrly + Gentamicin 5mg/kg daily Metronidazole 500mg 8hrly	Call Micro	10-14 days
Neutropenic infections	Tazocin 4.5g 8hrly + Amikacin 15mg/kg daily	Meropenem 1g 8hrly + Vancomycin 500mg-2g/24hr	Discuss
Meningitis	Cefotaxime 2g 6hrly + Aciclovir 10mg/kg 8hrly if viral encephalitis suspected + Amoxicillin 2g 4hrly (if >55 years old to cover Listeria)	Meropenem 2g 8hrly + Acyclovir 10mg/kg 8hrly if viral encephalitis suspected	5-7 days
Vascular line associated infection	**Remove / change lines** **If severely unwell** Co-amoxiclav 1.2g 8hrly + Gentamicin 5mg/kg od (+ Vancomycin 500mg-g/24hr **if MRSA colonised**)	Vancomycin 500mg-2g/24hr + Gentamicin 5mg/kg od	5-14 days
Clostridium difficile associated diarrhoea	**PO/NG** Metronidazole 400mg 8hrly	**PO/NG** Vancomycin 125-250mg 6hrly	10-14 days
Urinary tract infections	Co-amoxiclav 1.2g 8hrly + Gentamicin 5mg/kg x1	Ertapenem 1g daily + Gentamicin 5mg/kg x1	5-7 days
Sternal wound infections	Vancomycin 500mg-2g/24hr + Meropenem 1g 8hrly	Call Mirco	Discuss
Infections post neurosurgery +/- Intrathecal Device	Intrathecal Vancomycin 10mg od + Meropenem 2g 8hrly	Call Mirco	Discuss

Gentamicin

- Give first dose STAT, 5mg/kg whatever the renal function. (Maximum dose 450mg)
- Thereafter, chart for 12 noon administration when blood levels taken @~6am are <1mg/ml.

Amikacin

- Give first dose STAT, 15mg/kg whatever the renal function. (Maximum dose 1500mg)
- Thereafter, chart for 12 noon administration when blood levels taken @~6am are <5mg/ml.

Vancomycin

Prophylaxis (endovascular stents etc combined with 3 x co-amoxiclav 1.2g 8hrly)

- 1g repeated once after 12 hours (normal renal function)
- 1g only if significant renal impairment

Treatment

- Load with 1g over 2 hours then start continuous infusion at 500mg-2g/24hours depending upon renal function.
- Measure blood levels daily and titrate dose to achieve 20mg/ml

Fungal infections – suspected or proven

Infection	*Regime*	*Notes*
Candidiasis	*FIRST LINE* Fluconazole 800mg day 1 & 2 400mg daily thereafter for 7-10 days *SECOND LINE* Caspofungin 70mg daily	Load IV. Convert to enteral ASAP. If >130kg increase dose to 1.2g and 600mg respectively
Cryptococcus / Aspergillus	Liposomal amphotericin 3mg/kg daily	Watch for anaphylaxis

IVIg

In some cases of the following rare conditions, IVIg at 1g/kg daily for 48 hours may be indicated. A small emergency supply is kept in the GICU drug fridge. IT MUST NOT BE USED without consultant approval. The conditions are: Severe invasive group A streptococcal disease; Staphylococcal toxic shock syndrome; Necrotising (PVL-associated) staphylococcal sepsis; Severe or recurrent C diff colitis; CMV-induced pneumonitis following transplantation.

ODDS & SODS

Indication	*Drug*	*Notes*
Prevention of x-ray contrast induced nephrotoxicity	500ml of 1.4% $NaHCO_3$ with 2.4g of N-acetylcysteine	[5]
Eclampsia	Magnesium sulphate	Load 16mmol (4g) over 10-15mins then 4mmol/hr (1g) [69]
Acute pulmonary hypertension	Sildenafil 25mg NG 8 hourly	Increasing dose to 50mg / 100mg is of no proven additional efficacy

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