

AN INTRODUCTION TO THE GENERAL INTENSIVE CARE UNIT



WELCOME TO

ST GEORGE'S HOSPITAL

GENERAL INTENSIVE CARE UNIT

A Quick Who's Who

Consultants

Greg McNulty
Barbara Philips
Andy Rhodes
Jonathan Ball
Mark Hamilton
Maurizio Cecconi
Susannah Leaver
Vanessa Elliott
Nawaf al Subaie
Rafik Bedair

General Manager

Ruth Leighton

Management team

Nyrina Barwise x4164
Nana Frempomaa x4164

Senior Nursing Staff

Deborah Dawson
Lindsey Izard
Susan Reynolds

Step down & follow-up nurse lead

Paula O'Shea

Research nurses

Chris Ryan
Johannes Mellinghoff
Joao Mercedo
Helen Farrah
Vicky Barnes

Lead Physiotherapist

Lisa Ronson

Senior ICU pharmacist

Joanne Peh

Senior Technician

Sarah Piper

Administrative staff

Terri Walker
Edna Okyere
Jackie Williams

Audit team

Jag Singh
Claire Ostrowska

Where we are and how to make contact

General ICU

1st Floor

St James's Wing

St George's Hospital

Blackshaw Road

London SW17 0QT

Tel: 020 8725 1307

Fax: 020 8725 3296

Unit area extension nos.

ICU

HDU

Holdsworth IOR

The GICU SpR bleep is

Holdsworth OIR bleep is

Step down & follow up
nurse bleep is

1307 / 3294 / 3295

0883 / 0879

3717

7980

8278

7400

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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 any of the consultants 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. The consultant team 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.

Our environment is cramped and busy. It is everybody's responsibility to look after and maintain it. If you happen upon anything dirty, broken or "misplaced" please do something about yourself, then and there. It is not somebody else's responsibility.

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.

We hope you enjoy your time with us.

Jonathan Ball
On behalf of the GICU 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)

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 may 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 and the immediate availability of skilled medical staff.
ICU	Intensive Care Unit (ICU) – Is an area for the treatment of organ dysfunction / failure often including invasive ventilatory support. ICU's will usually have a nurse to patient ratio of 1:1 and the immediate availability of skilled medical staff.

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. Use <http://clincalc.com/icumortality/SOFA.aspx>
- It is our aspiration to have a daily SOFA score recorded between 8am and 10am based upon the worst value for each physiological variable within the past 24 hours.

THIS UNIT

- The General Adult Intensive Care Unit (GICU) at St George's currently has 21 beds.
- Of these:
 - 12 are located in the so-called ICU
 - 6 in the co-located, so-called HDU
 - 3 in the Overnight Intensive Recovery (OIR) on Holdsworth Ward (5th Floor, St James's Wing).
- At full capacity we can stretch to 18 level 3 patients. Ideally, we run with 11 level 3, 6 level 2 and one emergency level 3 bed.
- The 3 IOR beds are purely for the immediate post-operative care of high risk, elective, surgical patients.
- We have close working relationships with the Cardiothoracic and Neuro Intensive Care units. When necessary, patients can be transferred between units to facilitate acute admissions.

Nursing staff

- The nursing staff do 12.5-hour shifts (i.e. 2 per 24 hours).
- Their handover takes place at 7.30 and 19.30 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.

SHIFT PATTERNS

- Rota rules and day to day administration is co-ordinated by Nana Frempomaa.
- Dr Elliot is the consultant with lead responsibility for issues related working patterns.
- Full details are updated and available on the GICU website:
<http://www.gicu.sgul.ac.uk/i-am-about-to-start/doctors>

DAILY TIMETABLE

08:00-08:30	<p>HANDOVER end-of-the-bed ward round night shift to "Reg of the week" / long day shift. MUST FINISH PROMPTLY</p> <ul style="list-style-type: none"> • Final decisions regarding discharge from GICU and prioritising elective surgical admissions are crucial. • All other Drs should start clerking patients. • One doctor should check the Line Trolley. • Please book any procedures / scans as early as possible. <p>HANDOVER ward round on OIR second tier Dr to Dr</p> <ul style="list-style-type: none"> • Patients not fit for discharge must be transferred to GICU for continuing care
10:00	Multidisciplinary, "end of the bed", REHAB ward round
10:30	<p>Multidisciplinary, Consultant led, TEACHING ward round. (Coffee and biscuits).</p> <ul style="list-style-type: none"> • Sit down (seminar room) ward round followed by "tour of the unit." • Please ensure all plans are handed over to nurses at bedside. • PLEASE complete the "Ward Watcher" diagnostic codes during this WR.
15:30	Microbiology round (Mon-Fri)
18:00	EVENING end-of-the-bed ward round with on-call Consultants
20:00-20:45	<p>HANDOVER ward round Day shift to Night shift.</p> <ul style="list-style-type: none"> • Identify patients who are likely to be fit for discharge the following morning. Please start / complete Episode Summary. • Night Reg must review OIR patients with OIR SHO, preferably before 22:00.
~23:30	TELEPHONIC ward round with on call consultant

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.

DRESS CODE

- When on clinical duties, please wear GREEN Critical Care scrubs. They indicate to all staff on the unit and elsewhere that you are a qualified doctor working on 1 of 3 adult ICUs.
- These scrubs may be worn around the hospital without additional cover.
- They should be worn if accompanying a patient on an inter-hospital transfer.
- They must not be worn whilst travelling to and from St George's.
- Please remove all wrist watches and jewellery.
- If you have long hair, please ensure it is neatly tied back.
- If you are cold in scrubs, please wear a suitable base layer underneath the scrubs but maintain "naked below the elbow" for infection control purposes.

MINIMUM STAFFING LEVELS / LEAVING THE UNIT

- There should be at least one doctor on the unit at all times.
- Out of hours, the on call senior (1st) tier must inform the 2nd tier Drs and the nurse in charge if they are leaving the unit and ensure they can be contacted in an emergency.
- The on-call senior (1st) tier MUST carry bleep 7980.
- The second tier OIR Dr MUST carry bleep 8278. You may leave the OIR but should be available to return immediately if called.
- Detailed rota rules can be found at: <http://bit.ly/29C8czu>

NIGHT SHIFTS

- During the night, the junior doctor team on are welcome to use the consultants office opposite the coffee room.
- It is a shared resource for all the doctors on call.
- It MUST be left clean and tidy at the end of the shift.
- When unoccupied it must be locked - the Nurse in Charge of the shift holds the key.
- For patients identified as likely to be stepped down the following day, please start / complete the Episode Summary and prepare the patient as far as practical.

FOUNDATION YEAR 1 DOCTORS

- Your normal working hours are 08:00-18:00, 4 days out of 5, Monday to Friday, on a rolling rota, available at <http://bit.ly/29PjAxx>
- Regrettably, the Foundation School have removed the out of hours component of your posts, despite our protestations.
- During your first month, you'll be on GICU. In your successive 3 months, you be rostered to CTICU for 1 of the 3 months.
- When asked to perform the daily clerking of a patient, please ensure your findings and documentation are checked and countersigned in the notes by a more senior member of the team.
- Dr Bedair is the educational supervisor for Foundation doctors.

ANNUAL AND STUDY LEAVE

- Any leave taken outside of fixed days off, must be agreed with and covered by your colleagues.
- Difficulties with leave should be discussed in the first instance with Dr Elliot 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.
- For forms and obligatory return to work interviews, please contact Nyrina Barwise (x4164) or Nana Frempomaa (x4164).

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.
- On Wednesdays at 08:30, there is a Morbidity, Mortality, Re-admissions and Referrals meeting. These sessions are co-ordinated by Dr Bedair.
- On Thursdays at 08:30, there is a teaching presentation by one of the trainees. These sessions are co-ordinated and chaired by Dr Leaver.
- On Fridays at 08:30 there is a Journal club. These sessions are co-ordinated and chaired by Dr Elliot.
- There will be one whole afternoon, separate first tier and second tier, teaching sessions. These sessions are co-ordinated and chaired by Dr Leaver.
- There is a GICU website that contains many resources and useful links: www.gicu.sgul.ac.uk

MEDICAL STUDENTS AND WORK EXPERIENCE STUDENTS

- We frequently have local and visiting medical students and work experience students.
- Please make them feel welcome and task them with interesting things to do.
- They must be supervised at all times.
- Please ask them to wear GREY scrubs whilst on the unit. This indicates that they are not qualified doctors.
- They can only make entries into the medical notes if countersigned by a Dr, who must have personally ensured that the entry is accurate.
- Local students spend a week with us during a month long anaesthetic and critical care attachment. Toward the end of this week they should complete a case based discussion on a complex / long stay ICU patient and a CVP miniCEX.

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.**
- Once past the chart table in each bed space, all staff to be 'naked below the elbows', including watches and all rings.
- Gloves and either an apron or gown should be worn for all patient contact. Should they become soiled, they should be changed at the earliest opportunity.
- 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 MEDICAL NOTES IN GICU


- When admitting a patient to GICU please complete the iCLIP document "Z-JB ICU admission & daily". This is a pre-configured template you can locate by following the numbered highlighted areas below.

*Type: General Clinical Note **1**

*Date: 13/07/16 1830 BST

Title: ZJB ICU admission & daily

Document Template Existing **Pre-configured** Catalogue Recent Favourites

Search: **z** **2**  **3** Contains

Restricted display by:

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Title	Document template	Shared	Last Changed By	Perform/Service Dat
Hazelnut & almond -ve challenge	Dynamic Content Creation	Y	Wells , Karen Jane	10/06/15 11:29:45
Hazelnut +ve challenge	Dynamic Content Creation	Y	Bidgood , Sarah Maureen	20/11/15 12:53:32
Hazelnut +ve challenge	Dynamic Content Creation	Y	Bidgood , Sarah Maureen	20/11/15 12:53:51
Hazelnut -ve challenge	Dynamic Content Creation	Y	Bidgood , Sarah Maureen	06/11/15 13:18:34
Paediatric Seizure Chart	Dynamic Content Creation	Y	Barrett , Jodian Rossan	23/10/14 16:01:08
ZJB ICU admission & daily	Dynamic Content Creation	Y	Ball , Jonathan	11/07/16 19:15:41 4
ZJB ICU episode summary	Dynamic Content Creation	Y	Ball , Jonathan	11/07/16 18:26:13

SUGGESTIONS ON HOW TO COMPLETE NARRATIVE ELECTRONIC MEDICAL NOTES

- If you were reviewing this case in 6 months time AND had no prior knowledge of this patient, what would YOU want to know?
- ONLY include details that summarise events AND ARE NOT readily available elsewhere in the electronic medical record (EMR)
- Navigate using UP and DOWN arrow keys (or any pointing device) and AVOID using ENTER / RETURN which adds unnecessary blank lines.
- Please use COPY & PASTE FROM WITHIN CERNER intelligently BUT please REVIEW / MODIFY / CORRECT / UPDATE. If you copy and paste from any external source, the document often fails to modify and you LOOSE ALL YOUR WORK.

SUGGESTIONS ON HOW TO COMPLETE A DAILY REVIEW IN THE PAPER MEDICAL NOTES

- Patient's name, MRN, GICU consultant on call etc must be completed on every side of every history sheet in the patient's notes.
- Patient's age, date of admission to hospital and number of days on the unit
- Principal referring team and other teams involved, including contact details
- Brief history & update (comprehensive, chronological, concise)
 - Main diagnosis / diagnoses
 - Reason for ICU admission including details of injuries / surgical procedures*
 - **Current Problems and their Progress over the last 24 hours (including results of any imaging / investigations / procedures)**
 - *Relevant co-morbidities* (& source of history e.g. GP)*
 - *Pre-morbid level of function* (& source of history e.g. GP)*
- 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.

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.

<i>General</i>	End-of-the-bed-o-gram / "What would the cleaner notice?"
<i>Cardiovascular</i>	<p>Heart rate and rhythm</p> <p>Right atrial (RA) / central venous (CV) pressure</p> <p>Pulmonary artery pressures</p> <p>Systemic pressures, including mean arterial pressure (MAP)</p> <p>Cardiac output/index (CO/CI)</p> <p>Oxygen delivery (DO_2/DO_{2I})</p> <p>Mixed or central venous oxygen saturation ($SvO_2/ScvO_2$)</p> <p>Haemoglobin / lactate levels / central venous - arterial CO_2 gap</p> <p>Vasoactive drug infusion rates</p> <p>Positive examination findings</p> <p>Relevant 12 lead electrocardiograph (ECG), echocardiogram and angiogram findings</p> <p>Serum cardiac troponin levels</p>
<i>Respiratory</i>	<p>Nature and age of airway management</p> <p>Positive examination findings including site and state of any chest drains</p> <p>Mode of ventilation (if assisted) duration (days), settings, measurements and trends:</p> <ul style="list-style-type: none"> • FiO_2 • Tidal volumes (V_t) • Peak, plateau, and mean airway pressures • Positive end expiratory pressure (PEEP) • Breath timing [inspiratory:expiratory (I:E) ratio] <p>Recent arterial blood gas (ABG) result OR SpO_2 and $ETCO_2$</p> <p>Weaning regime (if prescribed)</p> <p>Nature and duration of all inhaled / nebulised therapy.</p> <p>Relevant findings from chest x-ray (CXR) series / CT scans.</p>

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.

<i>Renal</i>	<p>Last 6 hours urine output and today's urine dipstick analysis</p> <p>OR</p> <p>Renal replacement regime: mode, pre-dilution rate, post-replacement rate, fluid removal rate (set and achieved), anticoagulation prescription, time and date current circuit commenced / due to expire (max 80 hour lifespan)</p> <p>Today's, yesterday's and cumulative fluid balance.</p> <p>Trend of blood urea and creatinine.</p> <p>Abnormalities in blood sodium and any other relevant electrolyte results.</p>
<i>Gastro-intestinal</i>	<p>Abdominal examination, including site and output of any drains.</p> <p>Mode of feeding: naso-gastric (NG), naso-jejunal (NJ), gastrostomy and / or parenteral.</p> <p>Position of distal tip of NG tube and aspirate volumes.</p> <p>Nature and duration of prokinetics, if any.</p> <p>Nature and duration of stress ulcer prophylaxis.</p> <p>Bowel output.</p> <p>Nature and trend of any abnormal biochemistry.</p>
<i>Neurological</i>	<p>Nature and dose of analgesia and sedative medication.</p> <p>Glasgow coma score (GCS), sedation score and delirium assessment.</p> <p>Behaviour and orientation (if appropriate).</p> <p>Positive exam findings.</p>
<i>Infection control / microbiology</i>	<p>Skin / wounds - condition / abnormalities</p> <p>Age, location and appearance of exit site of ALL Lines / tubes / catheters / cannulas / drains etc</p> <p>Temperature.</p> <p>White blood cell count and C-reactive protein (CRP).</p> <p>Date, site and sensitivities of any positive cultures.</p> <p>Nature, indication and duration of any / all antimicrobials.</p>

MENTAL DAILY CLERKING CHECKLISTS

SIMPLE = F L A₃ T C H₂ U G

Feeding: Is the patient successfully being fed? Are they receiving prokinetics? Do they still need them (not if 4hrly gastric aspirates are <400mls for >24hours)?

Lines: For each line / 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)?

A₁nalgesia: Regular simple analgesia + / - opiates. What is the quality of pain control?

A₂ntibiotics: Indication and STOP date recorded in notes and on drug chart? De-escalate to narrower spectrum? IV to enteral switch? Levels? Toxicity - skin / renal / hepatic?

A₃ppariants: Are bowels working? Are senna 15mg NG od-bd and sodium docusate 200mg bd prescribed and being given?

Thromboembolic prophylaxis: dalteparin 5,000 units prescribed? TEDs? Calf compressors? TEG indicated / performed? Results seen and acted upon?

Communication: With the patient (if possible)? With the family / friends? With the parent team(s)? Documented in the notes?

H₁ead of bed elevated 30 – 45°: Can the patient be sat out of bed or even mobilised?

H₂ydration / fluid (sodium & chloride) balance: What was yesterday's fluid balance? What is the cumulative fluid balance? What is the fluid balance target for today? What is the sodium and chloride balance (how much NaCl is the patient receiving in their IV medications)?

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 ≤10.0 mmol/l over the last 24 hours?

ALTERNATIVE / MORE EXTENSIVE = D A S H E M F F U G B T T

Drug chart: legible and clear? Appropriate dose / frequency? Missed doses? Evidence of renal / hepatic toxicity? Restart chronic drugs? Stop anything? IV to enteral? Therapeutic monitoring required? Indications and stop / review dates for antimicrobials and any other short term drug courses?

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 gel + / - 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 <400mls for >24hours)?

Fluid balance: What was yesterday's fluid balance? What is the cumulative fluid balance? What is the fluid balance target for today? What is the sodium and chloride balance (how much NaCl is the patient receiving in their IV medications)?

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 ≤ 10.0 mmol/l over the last 24 hours?

Bowels: Are they working? Are senna 15mg NG od-bd and sodium docusate 200mg bd prescribed and being given?

Thromboembolic prophylaxis: dalteparin 5,000 units prescribed? TEDs? Calf compressors? TEG indicated / performed? Results seen and acted upon?

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)?

DAILY INVESTIGATIONS

- Most patients should have the following checked daily and / or on return from a major surgical procedure: full blood count (FBC), Clotting (including fibrinogen if coagulopathy suspected), urea & electrolytes (U&E's), Troponin T, Calcium, Phosphate, Albumin, Magnesium and Glucose. On iCLIP use the "ICU Admission" and "ICU Daily Bloods" bundles.
- Liver function tests (LFTs) and C-reactive protein should be checked daily in all acutely / severely unwell patients. The Biochemistry lab will not process repeated requests if samples received within 24 hours of each other.
- Patients receiving once daily aminoglycosides and/or continuous infusions of glycopeptides (vancomycin) require daily random levels. These should be performed simultaneously with the daily morning bloods..
- If a patient is likely to require blood products or is going to theatre, ensure that 2 samples are, or have been, sent to the blood bank. Blood bank will only cross match 2 units of packed red blood cells unless you discuss the case with them on x5471 &/or the Haematology SpR on bleep 6311.
- Screening swabs for colonising multi-resistant bacteria, in particular, methicillin resistant *Staphylococcus 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 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. 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).
- The Long Day Registrar is responsible for ensuring that each patient's diagnostic codes on Ward Watcher, are accurate and comprehensively completed.

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 evidence to support the use of sodium bicarbonate as the optimal crystalloid rather than sodium chloride [5]. Our policy, therefore, is to give 500mls of 1.4% NaHCO₃ or Hartmann's, a maximum of 30 minutes, prior to IV contrast, in patients with acute or chronic renal injury. The balance of evidence does not support the use of N-acetylcysteine (NAC) [6] or prophylactic renal replacement therapy to prevent contrast induced nephropathy [7].
- There is increasing evidence that the radiation dose patients receive from investigations results in CANCER in those patients. Always ask whether an investigation will change your management, if not, DON'T DO IT. As a guide, [these are the equivalent doses of common investigations using a routine CXR as the reference together with natural background radiation \(NBR\) in days](#) [8]:

EQUIVALENT DOSES OF COMMON RADIOLOGICAL INVESTIGATIONS

Investigation	Av. dose in mSv	Equiv. no. of CXRs	Equiv. no. days NBR
CXR	0.02	1	3 days
AXR	0.7	35	105 days
PXR	0.6	30	90 days
CT-brain	2	100	300 days
CT-neck	3	150	1.2 years
CT-chest	7	350	2.9 years
CTPA	15	750	6.2 years
CT-abdo pelvis	14	700	5.8 years
Trauma-CT	26	1300	10.7 years
CT-angio	15	750	6.2 years
coronary PCI	15	750	6.2 years

Key - NBR = normal background radiation

COMMUNICATION

- Whenever possible, spend time talking to our patients.
- Make every effort to communicate, at least daily, with the patient's relatives and all specialty teams involved in the case.
- 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.
- Numerous medical / surgical teams visit the ICU every day. To maintain effective communication all discussions must be documented. We operate as a closed unit, i.e. all prescribed therapies should only be instigated by the GICU team.

STEP DOWN (DISCHARGE) FROM THE CRITICAL CARE ENVIRONMENT

- Please use “step down” and not “discharge” as the latter is confused (especially in iCLIP) with discharge from the hospital.
- Unless essential, do not step patients down between 22:00 and 08:00.
- Start planning for step down at the **earliest** opportunity. **Consider:** when / where to (including base hospital) / under whose care / are special arrangements required?
- Patients likely to be stepped down the following day should be identified as such, at the evening (18:00) ward round.
- Please start to complete the iCLIP document “Z-JB ICU episode summary” as soon as the patient has been identified as a potential transfer. This is a pre-existing template you can locate by following the numbered highlighted areas below. During the night shift, please complete this episode summary as fully as possible (time permitting).

*Type: General Clinical Note 1

*Date: 13/07/16 1830 BST

Title: ZJB ICU admission & daily

Document Template Existing **Pre-configured** Catalogue Recent Favourites

Search: z 2 3 Contains

Restricted display by:

☐ My notes only ☐ Note Type ☐ Associated Diagnosis

☐ Recent ☐ Favourites

Title	Document template	Shared	Last Changed By	Perform/Service Dat
Hazelnut & almond -ve challenge	Dynamic Content Creation	Y	Wells , Karen Jane	10/06/15 11:29:45
Hazelnut +ve challenge	Dynamic Content Creation	Y	Bidgood , Sarah Maureen	20/11/15 12:53:32
Hazelnut +ve challenge	Dynamic Content Creation	Y	Bidgood , Sarah Maureen	20/11/15 12:53:51
Hazelnut -ve challenge	Dynamic Content Creation	Y	Bidgood , Sarah Maureen	06/11/15 13:18:34
Paediatric Seizure Chart	Dynamic Content Creation	Y	Barrett , Jodian Rossan	23/10/14 16:01:08
ZJB ICU admission & daily	Dynamic Content Creation	Y	Ball , Jonathan	11/07/16 19:15:40
ZJB ICU episode summary	Dynamic Content Creation	Y	Ball , Jonathan	4 11/07/16 18:26:1

- On the 8am round clearly identify patients “fit for step down”, “possibly fit for step down” and “unfit for step down”. Please involve

the "Reg of the week" or one of the consultants, as soon as possible, for all "possibly fit for step down" patients to clarify / resolve any uncertainties.

- When performing daily reviews, prioritise patients fit for step down. Please check the step down board and / or liaise with the step down & follow up nurse regarding bed availability. As soon as a bed is identified, please contact the parent team to handover the patient.
- Please ask one of the registrars or consultants to review the episode summary before the patient leaves the unit.
- **Just prior** to the patient leaving, please review them to ensure that their condition hasn't changed. Please record this final check on the episode summary. Print off the summary, punch holes in it and FILE it in the contemporaneous paper medical notes. If, as is common, you cannot print the document due to IT / local printer issues, PLEASE write in the notes, "GICU Episode Summary has been completed in iCLIP."

CONTENT OF GICU EPISODE SUMMARIES AND HANDOVERS

- Ensure the summary is concise but comprehensive.
- Include a list of active problems and an explicit PLAN for each.
- Clearly document if the patient is considered to be unsuitable for re-admission to ICU, and if so, suggest that a DNA CPR form be completed by the receiving team as soon as practical. If this is unlikely / impractical, please ensure the form is completed and countersigned by the GICU consultant before the patient leaves. Please see the [unit policy](#) for a more detailed explanation.
- Record essential handover information on the Hospital at Night system on EPR.
- 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. This communication must be recorded on the episode summary.

DYING PATIENTS AND END OF LIFE CARE

Focus on the patient

- In the terminal phase of a patient's illness ensure optimal palliation of any / all sources of distress AND proactively institute any measures that might improve comfort / wellbeing.
- Stop everything that isn't contributing to a patient's comfort - this includes monitoring / physiological observations, drugs and all procedures.
- If already in place, consider continuation of enteral food and hydration, especially if death is not imminent. If not in place consider the need and options for hydration.
- Ask all those caring for the patient to record their timed observations / concerns on the ICU chart / "End of life & terminal decline" chart INCLUDING any interventions.
- Don't hesitate to seek senior / expert help if required. Only contact the Palliative Care Team if the patient is to be transferred off GICU.

Optimise the environment

- Turn all device alarms off. If unable to do so, set to maximum thresholds and minimum volume.
- If time permits, remove all unnecessary clutter, inform adjacent bed spaces of situation and ask them to minimise noise. If available, consider transferring the patient to a side room.
- If the dying process continues for >12-24 hours, consider transfer to a ward (preferably a side room.) Alternatively, we have, on occasion, transferred patients to Hospices or the patient's / their next of kin's homes.

Communication

- If a patient is terminally ill, ensure that key / close contacts (family & friends) are fully aware of the situation.
- Contact the admitting team to ensure they are also aware of the situation.

- If the patient could be a potential organ / tissue donor discuss referral with the ICU consultant. For additional information or to make a referral contact the Transplant Co-ordinator via 07659 100103.
- If appropriate, ask close contacts if they would like religious / spiritual support from the hospital chaplaincy team (covers all religions and denominations).

After death

- Use the "[Record of Death Form](#)" to record the clinical confirmation of death as per the Academy of Medical Royal Colleges' guideline [9]. Forms can either be downloaded, completed in MS Word, saved to your K drive, and attached to a blank General Clinical Note in the patient's iCLIP Documents **OR** blank forms should be found with the Death Certificate book, and can be completed by hand.
- Ensure a death certificate and cremation form 4 is completed as soon as practical and always before the end of that shift unless the patient is a mandatory coroner's inquest i.e. a death due to an "unnatural cause" such as an accident (trauma) or misadventure as opposed to a disease (non-industrial). Remember, if a death occurs out of hours, you may be the only person who can complete the forms. Copy this information onto the "Record of Death Form".
- If uncertain how to complete the death certificate, discuss this with a senior colleague and / or refer to [10].
- Always inform all relevant teams after a patient has died and whenever possible, inform the patient's general practitioner.
- Record all communications on the "Record of Death Form" so that everybody knows who has and who has not been contacted.
- Additional helpful resources can be found at: <http://bit.ly/29I7PaN>
- To refer a case to the coroner, complete this [FORM](#); ensure it is approved and countersigned by a consultant; and email it as described on the form. PLEASE note that the highlighted elements EITHER require modification e.g. correct ICU, location, direct phone no. etc OR are directions / explanations. PLEASE note the circumstances when you should or should not ALSO complete a death certificate and cremation forms. If you do complete a death certificate PLEASE remember to scan + email (or fax) it along with

the referral form. Once completed, PLEASE attach a copy to a blank document in the patient's iCLIP record.

- All deaths **MUST** have an episode summary completed either the standard iCLIP template OR the coronial referral form.

DO NOT ATTEMPT CARDIOPULMONARY RESUSCITATION ORDERS

The Trust's do not attempt cardiopulmonary resuscitation (DNA CPR) policy can be found [here](#):

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

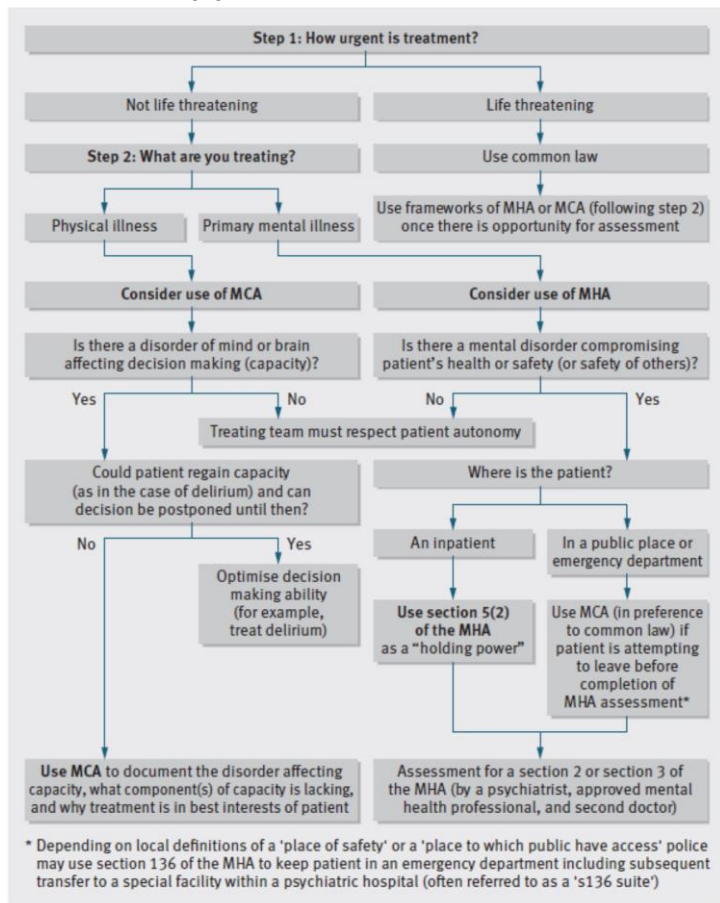
1. Ward referrals reviewed by GICU. If a patient is not considered appropriate for ICU care and is likely to deteriorate to the point of cardio-respiratory arrest, it is the responsibility of **the referring team** to complete the DNA CPR form. This must be communicated to that team at the time the patient is reviewed. If a patient is inappropriate for ICU care before they arrest then clearly, they should be made DNA CPR.
2. Patients on GICU. These patients **do not** require DNA CPR forms to be completed as patient specific plans are formulated and regularly reviewed that exceed the scope of the Trust DNA CPR 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 transferred off GICU to the ward. If it is the GICU team's opinion that a patient would not benefit from a return to ICU following transfer 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 DNA CPR form for the patient prior to their transfer. However, whenever possible, the DNA CPR 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 AND CAPACITY

- 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.
- A patient has capacity if you are satisfied that (CURB [11]):
 - They can **COMMUNICATE**
 - They can **UNDERSTAND**
 - They can **RETAIN** information
 - They can **BALANCE** or use information
- All capacity assessments **MUST** be clearly recorded in the medical notes.
- If capacity is impaired all decisions must be guided by the principle of best interests. In order to determine these, it is usually appropriate to consult (NOT gain consent from) the patient's nominated next of kin. You may need to balance the duty to consult with the duty to maintain patient confidentiality.
- If no such person exists and if time and circumstances permit, consider engaging the services of an independent, professionally trained, lay person (Independent Mental Capacity Advocate or IMCA). This **MUST** be a consultant decision. To request the services of an IMCA (office hours only) contact Voice Ability on 0845 175198.
- In a time limited situation, 2 senior members of the ICU team should discuss and agree upon what is in the patient's best interests, documenting this in the patient's notes.
- No-one except an appointed advocate can give consent on behalf of an incompetent (unconscious) adult.
- 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.
- For additional information regarding legal frameworks see [12] - full text available [HERE](#).

Flow chart from [12]



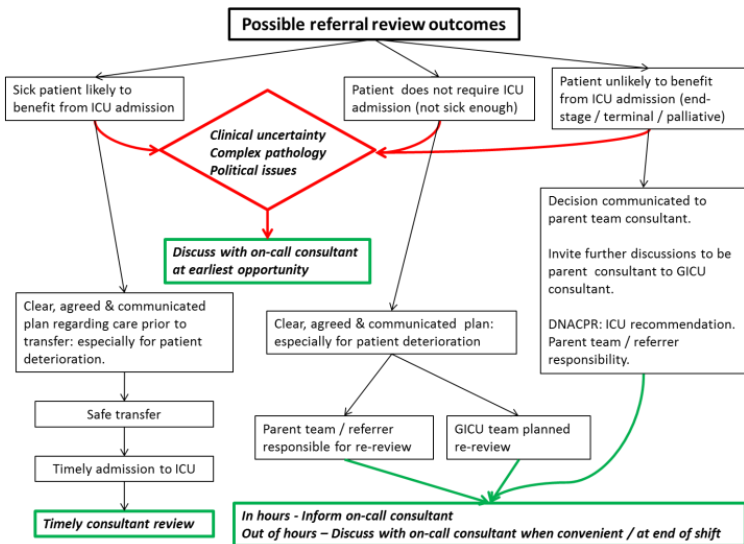
* Depending on local definitions of a 'place of safety' or a 'place to which public have access' police may use section 136 of the MHA to keep patient in an emergency department including subsequent transfer to a special facility within a psychiatric hospital (often referred to as a 's136 suite')

- In addition, following Montgomery v Lanarkshire Health Board (March 2015) <https://www.supremecourt.uk/cases/uksc-2013-0136.html> the following change in law should be observed:

“Any adult of sound mind is entitled to decide which, if any, of the available treatments to undergo, and consent must be obtained before treatment interfering with the bodily integrity is undertaken. The doctor is under a duty to take reasonable care to ensure that the patient is aware of any material risks involved in proposed treatment, and of reasonable alternatives. A risk is “material” if a reasonable person in the patient’s position would be likely to attach significance to it, or if the doctor is or should reasonably be aware that their patient would be likely to attach significance to it.”
- Many additional resources related to these complex and evolving issues are available here: <http://bit.ly/29Ey5zs>.

REFERRALS

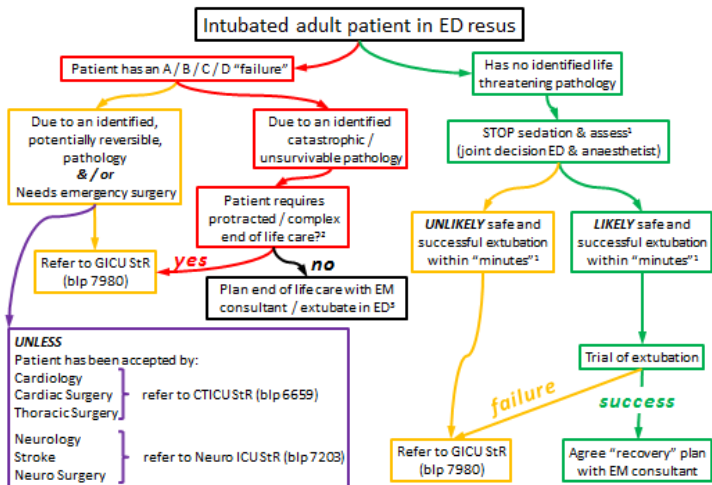
- As we don't have ICU outreach, are not part of the Cardiac Arrest or Trauma reception teams but receive many requests for advice / patient referrals from both within the hospital and beyond it is essential that we both log and audit this activity.
- All patients, with the exception of planned admissions of elective surgical patients, must have a referral audit form completed.
- Blank forms are kept in a box folder on the main desk OR can be printed off from: <http://bit.ly/29Fa7q8>
- Completed forms should be returned to the same box 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.
- **Our standards of care are:**
 - Between 8am-6pm Monday to Friday - 90% of referrals seen within 15 minutes of request.
 - Out of hours (all other times) - 90% of referrals seen within 30 minutes of request
 - No management advice should be given without review. This advice should be documented in the patient's notes.
 - Time from acceptance to admission:
 - Immediate – 90% within 30 minutes
 - Urgent – 90% within 60 minutes
- Though we do not mandate that every admission decision be agreed with the on call consultant, we encourage you to have a low threshold for discussing your opinion with us. This is particularly the case in frail patients, those with life limiting chronic disease and / or a poor prognosis (please see decision tree below).
- If you choose not to discuss a referral with a consultant at the time, PLEASE do discuss it before the end of your shift and record that conversation on the form.
- At the end of every shift, please send an email with the following information to stgh-tr.gicu@nhs.net: number of referrals received; any issues or concerns regarding these referrals.



REFERRALS REGARDING INTUBATED PATIENTS IN ED RESUS

Key personnel in the decision making process

- EM consultant in charge of Resus (bleep 8021) in collaboration with lead ED nurse for Resus
- Anaesthetic StR (bleeps 6111 or 7647) in collaboration with the Duty Floor Anaesthetist (bleep 8011) or the general anaesthetic consultant on call
- GICU StR (7980) in collaboration with the GICU consultant. [The GICU StR is usually best placed to act as the single point of contact for Adult Critical Care].



Admissions to CTICU and Neuro ICU from ED

- These are dependent upon the patient having been accepted by the relevant specialty team
- If the patient has not been accepted by one of these teams then the referral pathway is to GICU in the first instance. It is for the on call consultants for the 3 ICUs to determine the best ICU to admit the patient to and this is dependent upon both the patient's needs and the bed state of the Adult Critical Care service.

For patients who have no identified life threatening pathology: Sedation holds for assessment and planned extubation in ED within "minutes"

- As soon as safe and practical, a sedation hold should be performed to facilitate re-assessment. The decision to perform a sedation hold should be agreed by the ED and Anaesthetic key personnel.
- If it seems likely that the patient can be safely and successfully extubated within "60 minutes" then this should be undertaken in ED.

- The EM consultant in Resus, the senior nurse in Resus and the anaesthetist should agree a plan for the patient, including “stand down” criteria for the anaesthetist.

Patient requires protracted / complex end of life care?

- It may be appropriate to admit a patient to an ICU bed for end of life care.
- As each situation is unique, it is highly desirable if the EM consultant discusses the case with the GICU consultant.
- One reason might be active management whilst the potential for organ donation is considered.
- As a reminder, NHSBT’s absolute contra-indications to organ donation include:
 - Aged 86 and above (DBD and DCD)
 - Any cancer with evidence of spread outside affected organ (including lymph nodes) within 3 years of donation (however, localised prostate, thyroid, in situ cervical cancer and non-melanotic skin cancer are acceptable)
 - Melanoma (except completely excised Stage 1 cancers)
 - Choriocarcinoma
 - Active haematological malignancy (myeloma, lymphoma, leukaemia)
 - Definite, probable or possible case of human TSE, including CJD and vCJD, individuals whose blood relatives have had familial CJD, other neurodegenerative diseases associated with infectious agents
 - TB: active and untreated
 - HIV disease (but not HIV infection) HIV infection means people who have infection with HIV but none of the associated complications. Organs from such donors would be transplanted into individuals who are infected with HIV.
 - For more information see <http://bit.ly/29NMtrT>

Communication

- In the event of uncertainties or problems consultant to consultant discussions invariably result in the fastest possible resolution.

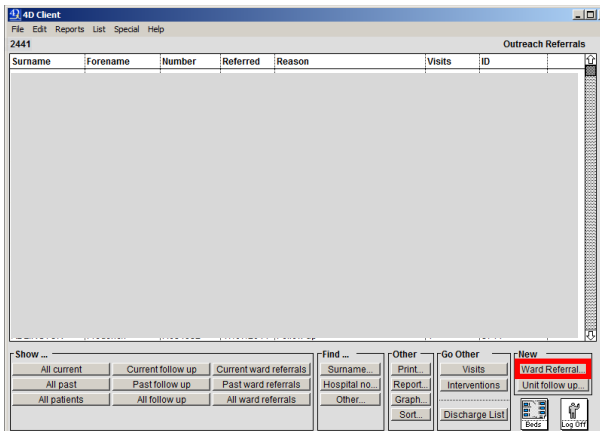
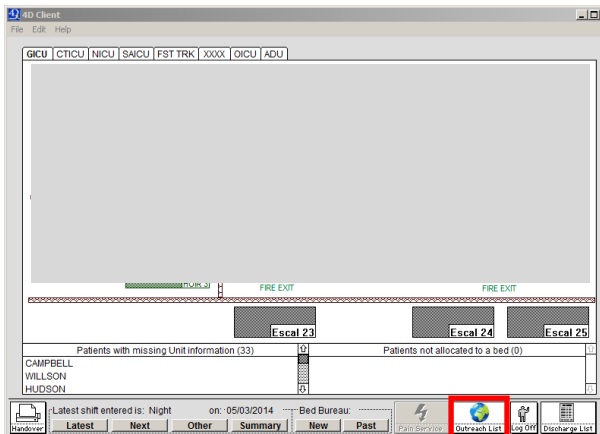
Considerations prior to ICU transfer

- Is there a clearly documented list of acute problems / diagnoses / injuries with a plan for each?
- Which specialty teams are actively involved in the patient's on-going care? Are their contact details clearly documented?
- Has a nasogastric (or orogastric) tube been inserted and the stomach effectively decompressed?
- Has a urinary catheter been inserted and urine drained?
- Has all necessary imaging been completed and reviewed (including repeat x-rays of lines / tubes etc)?
- If pertinent, has the spinal clearance form been completed and all unnecessary immobilisation removed?
- Have all wounds been cleaned, sutured and dressed, or is there a documented plan to go to theatre for this including the surgical team responsible?
- If there is going to be a delay in transfer whilst the ICU bed is cleared / cleaned / prepared, what could usefully be done for the patient during this time? [E.g. sedation hold; wean to spontaneous mode of ventilatory support; central line & / or vascath insertion; etc etc].

Useful numbers

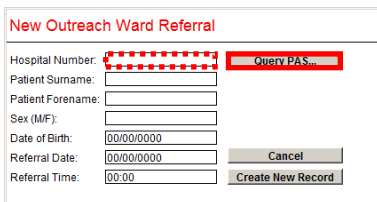
- General Intensive Care Unit (GICU), 1st Floor St James's Wing, Ext 1307 / 3294 / 3295
- Neuro Intensive Care Unit (NICU), 2nd Floor Atkinson Morley Wing, Ext 4195 / 4196
- Cardiothoracic Intensive Care Unit (CTICU), 1st Floor Atkinson Morley Wing, Ext 1495 / 1504 / 1507

HAVING COMPLETED A REFERRAL EPISODE, PLEASE RECORD THE DETAILS ON **WARDWATCHER**. TO ACHIEVE THIS:



THEN a pop up box appears.

Enter Hospital number and press the **Query PAS...** button.



New Outreach Ward Referral

Hospital Number: **Query PAS...**

Patient Surname:

Patient Forename:

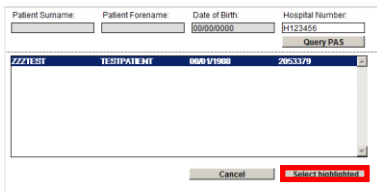
Sex (M/F):

Date of Birth:

Referral Date: **Cancel**

Referral Time: **Create New Record**

Select highlighted patient

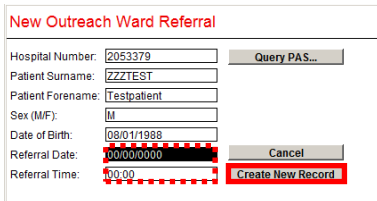


Patient Surname: Patient Forename: Date of Birth: Hospital Number: **Query PAS**

Patient Surname	Patient Forename	Date of Birth	Hospital Number
ZZZTEST	TESTPATIENT	08/01/1988	2053379

Cancel **Select highlighted**

THEN complete referral date and time boxes and press **Create New Record** button



New Outreach Ward Referral

Hospital Number: **Query PAS...**

Patient Surname:

Patient Forename:

Sex (M/F):

Date of Birth:

Referral Date: **Cancel**

Referral Time: **Create New Record**

Please enter essential details / a summary of the case in the **Referral Notes** box. If you have time and the enthusiasm, please complete as many of the other boxes as you can.

4D Client File Edit Help

Outreach Referral ID: 11797

Patient identity and referral details

Surname: **ZZZTEST** Date referred: **25/11/2015** Time referred: **18:15**
 Forename: **Testpatient** Referred by:
 Hospital no.: **2053379** Reason referred:
 DOB: **08/01/1988** Age: **27** Sex (M/F): **M** Details taken by:
 NHS number known (Y/N): **Y** NHS no.: EWS on referral: **0** Hospital admit date: **00/00/00**
 Hospital: Diagnosis:
 Referral location (type): Outreach category:
 Referral location (name): Initial management:
 Consultant: Previously seen by Outreach (Y/N):
 Specialty: Excluded from Outreach performance analysis (Y/N): **N**
 Surgery: Reason excluded:
 Date referral triggered: **00/00/00** Time trigger: **00:00**
 EWS on trigger: Gap (hrs) trigger to referral: **0**

Referral notes

Current user: balljo00 0

Referral Details Patient Visits Custom Questions Discharge Details Previous Patient Next Patient Referral List Bed Plan Log Off

A summary of each week's referrals must be presented at the Wednesday morning M, M, R & R meeting. This should include the total number of referrals received and any issues or concerns related to individual cases and / or systems.

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 [13].

RESEARCH

- GICU is an active research unit.
 - We have a team of 5 research nurses delivering a portfolio of NIHR and Commercial studies both observational and interventional.
 - Details of the studies we are currently recruiting to are posted on the Research notice board outside the staff room.
- Patients that are enrolled into interventional studies may, in addition to our usual standards of care, be treated according to a strict research protocol.
 - We ask that you stringently adhere to these protocols. If you need advice regarding a protocol, need to break a protocol or unblind a patient, please do not hesitate to contact the Research Team.
 - In daylight hours, one of the team is available on the unit. A rota with individual's contact details is posted on the Research notice board.
 - Out of hours, try the person rostered in the first instance, followed by the principal investigator.
 - Please always inform and involve the on-call consultant if there are any problems related to patients enrolled in interventional research studies.
- Enrolled patients often require additional clinical and laboratory assessments. The Research team may request your assistance in ensuring these are performed / ordered and documented. Your cooperation is greatly appreciated.
- Essential information related to all actively recruiting interventional trials should be available on the website in the [Research folder](#).

GENERAL CONSIDERATIONS FOR PATIENT CARE IN THE ICU

Many of the items detailed below are being brought together in so-called care bundles [14]. 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, SEDATION, SLEEP AND DELIRIUM [15]

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 [16].

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 some suppression of airway reflexes.
- Most ICUs employ continuous infusions of [analgesics](#) and [sedatives](#). The choice of agents depends upon a number of factors including drug pharmacokinetics, cost and personal preference. 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.
- [Neuromuscular blockade](#) should only be considered in patients in whom analgesia / sedation does not achieve the defined goals, most commonly, failure to achieve adequate ventilation. Intermittent bolus dosing is usually preferable to IV infusions. 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 deprivation is probably one of the principle causes of ICU delirium [17].
- 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, risperidone and quetiapine have also been used ([see bolus sedation table](#)) [18].

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.

Assessing the quality of analgesia, depth of sedation and presence of delirium

- These are essential skills in the assessment of all ICU patients.
 - The effectiveness of the analgesia regime should ideally be judged using a [numerical rating scale](#) (NRS). If the patient is not able to use such scale, please assess pain related behaviour using the [Critical Care Pain Observation Tool](#) (CPOT). If the NRS is ≥ 4 / 10 or the CPOT ≥ 2 / 8, then bolus &/or increase analgesic medication and reassess in ~30 minutes.
 - Next, the depth of sedation should be assessed using the [Richmond Agitation-Sedation Scale](#) to communicate and set goals. Our standard goal, even in intubated and ventilated patients is 0 to -1.
- Procedure
1. Observe the patient. Is the patient alert and calm (score 0)?
 - If the patient exhibits restless or agitated behaviour then score +1 to +4 using the these criteria.
 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. If there is no response to shaking shoulder then gently rub sternum.
 - Patient has any movement to physical stimulation (score -4).
 - Patient has no response to voice or physical stimulation (score -5).
- If RASS ≥ -3 proceed to [delirium assessment](#)
 - At present, there is no useful tool to evaluate the quantity or quality of sleep. Please ask the patient and / or the bedside nurse for their subjective impression.

Richmond Agitation–Sedation Scale

Score	Term	Description
+4	Combative	Overtly combative / violent &/or immediate danger to self &/or staff
+3	Very agitation	Pulls on / removes tubes / catheters &/or aggressive behaviour
+2	Agitated	Frequent non-purposeful movement &/or fights ventilator.
+1	Restless	Anxious or apprehensive but movements not aggressive or vigorous
0	Alert and calm	
-1	Drowsy	Not fully alert, but has sustained (>10sec) awakening, with eye contact, to voice
-2	Light sedation	Briefly (<10sec) 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 movement (eye opening) to physical stimulation
-5	Unroutable	No response to voice or physical stimulation

Sleep

- Physiological sleep is an essential requirement for physical and mental health. A basic review of the neurobiology of sleep can be found here [19] and a more detailed review here [20].
- Studying sleep quality and quantity requires continuous multimodal monitoring including EEG. Understandably therefore, research in the critically ill has been impeded by the logistics of employing such techniques and the interpretation of the accumulated data [21]. However, all of the studies published to date, agree that critical illness, sedation, mechanical ventilation and the ICU environment all contribute to dramatic reductions / obliteration, disruption / fragmentation and altered architecture of sleep [22, 23].
- Exactly how important this is remains unknown. How ICU interventions / environment can be altered to minimise this likely iatrogenic injury is also unknown but several common sense strategies are widely recommended, if perhaps, all too rarely employed. These include:
 - Minimising sedation including an early morning cessation of all sedative drugs.

- Adopting a daytime regime of stimulation / physical work (e.g. reduced ventilatory support) alternating with planned quiet periods and minimal interventions with optimal support during the night.
- Sensory minimisation using foam ear plugs and eye shades during quiet periods and / or to simulate a day night cycle.
- The only pharmacological intervention to show promise is melatonin [21] although much work remains to be done before this becomes established as a useful therapy.

Delirium

- Delirium [24] is an acute, (at least partially) reversible and fluctuating disturbance of consciousness, cognition and behaviour and by inference, maybe multifactorial. Delirium may be a manifestation of encephalopathy. A diagnosis of delirium during any acute illness is associated with a greater morbidity, length of ICU and hospital stay, prevalence of chronic neuropsychiatric sequelae and mortality.
- Delirium has been classified into 3 subtypes based on psychomotor activity: hyperactive, hypoactive and mixed or fluctuating. A number of bedside examination tools have been developed to diagnose and characterise delirium, including several, designed for use in ICU patients, including those intubated and ventilated. These tools assess conscious level, concentration / inattention and some aspects of cognition, often termed, organised thinking.
- Prevalence studies utilising these methods have reported incidence rates of 20-80% in ICU patient populations. Despite this wide range, it is generally acknowledged that delirium has historically been under diagnosed and that in part, this is due to the hypoactive subtype being the most prevalent and the purely hyperactive active subtype being comparatively rare.
- Delirium is a common manifestation in all acute illnesses. The best established risk factors are increasing age and any form of chronic cognitive decline, perhaps best considered a reduction in functional cognitive reserve. In the ICU population, severity of illness, direct brain injury and chronic hypertension have been identified as patient risk factors. There is also some evidence linking chronic alcohol misuse and acute nicotine withdrawal with the risk of developing delirium. In addition, a number of ICU interventions, in particular, the

cumulative dose of benzodiazepines has been shown to significantly increase the risk of developing delirium. Unsurprisingly, sleep deprivation has also been linked to the risk of delirium [25], however, the pathophysiology of sleep disturbance and delirium have a great deal in common and may be two manifestations of the same process rather than cause and effect.

- The pathophysiology of delirium is not well characterised [26] but inflammation, impaired oxidative metabolism, and alterations in the balance of amino acids and other neurotransmitters, in particular dopamine and acetylcholine, all appear to have a role. Studies into the long term neuro-cognitive outcomes of patients following critical illness have been limited but suggest a high incidence of chronic dysfunction associated with a diminished quality of life [27, 28]. The incidence, severity and duration of delirium are all associated with an increased risk of long term neuro-cognitive dysfunction. Some have hypothesised that critical illness accelerates age related brain atrophy and thereby unmasks otherwise occult, functionally compensated, structural abnormalities [29].
- Management recommendations include:
 - Daily screening for signs using the [Confusion Assessment Method for the ICU](#) (CAM-ICU).
 - Minimising all sedative medication [30] and avoidance of benzodiazepines altogether
 - Identical measures to those described above to optimise the quality and quantity of physiological sleep
 - Rescue therapies for hyperactive or mixed subtypes, where agitation impedes care or places the patient at risk of self harm include both traditional and atypical antipsychotics. To date, no single drug or combination of drugs has been demonstrated as superior. However there is some evidence to support the newer, atypical agents due to a reduction in side effects [18, 31, 32].

PATIENT POSITIONING [33, 34]

- 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.
- We have stopped ROUTINE use of chlorhexadine mouth gel +/- oral nystatin (topical antifungal). However please prescribe for patients with poor oral hygiene &/or acute pathology.

NUTRITIONAL SUPPORT [35]:

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 > 400mls after 4 hours) commence prokinetic therapy.
- First line therapy is metaclopramide 10 mg IV 8hrly.
- Second line therapy is erythromycin 250 mg IV on each subsequent occasion that the gastric aspirate is >400mls/4hours with a maximum dose frequency of 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 >400ml every 4 hours, for > 48h despite regular prokinetic administration.

Parenteral nutrition (PN)

- To be of benefit, PN has to be 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 PN should be undertaken [36].
- A dedicated central line or lumen should be set aside for PN. Close monitoring of electrolytes, liver function tests (for cholestatic jaundice) and line related sepsis should be undertaken.

Considerations that may warrant initiation of PN

These presume that enteral tube feeding has failed or is not possible / appropriate and cannot predictably be initiated in the next 7 days.

- Severe chronic mal / under nutrition. For example:
 - low body mass index (≤ 18)
 - significant recent weight loss
 - chronic inadequate intake
- Acute high nutritional needs / acute severe hypercatabolic state. For example:
 - severe / extensive polytrauma
 - extensive surgical “trauma”
- Small bowel failure. For example:
 - failure of upper GIT integrity - proximal perforation(s) / high output fistulae
 - significantly reduced small bowel length ($<1\text{m}$)
 - mesenteric ischaemia
 - acute severe pancreatitis
- The decision to commence PN should be reached by consensus between the “parent team”, the ICU team and the ICU dietician / the nutritional support team (NST). If consensus is not immediately forthcoming, the on call ICU consultant’s decision holds whilst consensus is sort.
- PN should be commenced within 24 hours of the decision to start.
- To request PN or a review by the NST, contact the NST dietician on bleep 6171.
- To obtain PN that day (Mon-Fri), this contact needs to be made as early as possible and has to be before 11:30 to allow sufficient time for pharmacy manufacturing to create a bespoke bag.
- If after 11:30, pharmacy will normally supply a bespoke bag the following afternoon.

If the ICU consultant considers that urgent (out of 'business' hours*) PN is warranted:

- Contact the on call pharmacist on bleep 6267
- Within 8 hours they will supply:
 - A basic bag (e.g. Kabiven 5gN 100kcal in 1440mL)
 - Parenteral micronutrients: Solivito N® and Additrac® in 250mL 5% Glucose
- Pabrinex I + II and parenteral micronutrients should be given BEFORE starting the PN.
- The PN should be commenced through a clean (unused), dedicated, central venous lumen at:
 - Patients <50Kg: infuse at 30mL/hour
 - Patients >50kg: infuse at 60mL/hour
- A close watch should be kept for electrolyte disturbances, in particular K⁺ and PO₄³⁻ together with other signs of re-feeding syndrome [37].
- The on call pharmacist will hand the patient over to the NST pharmacist on the next working day.

*out of hours = 17:00 to 09:00 weekdays and anytime at weekends/bank holidays

UPPER GASTROINTESTINAL TRACT

- All intubated patients, those receiving mask / helmet positive pressure ventilatory assistance and any other patient not able to eat (or eat enough) should have either an oro or a nasogastric tube. The preferential site and bore of the tube need to be considered carefully.
- Whenever possible, the position of an enteral access tube should be confirmed using bedside examination, which must include a reasonable volume aspirate with a pH<6.
- If you cannot confirm the position then request senior help. **Consider** whether an x-ray would be helpful, taking into account any structural abnormality the patient is known or is suspected to have e.g. hiatus hernia with intra-thoracic stomach.

- The following groups of patients should receive stress ulcer prophylaxis [34, 38]:
 - those not receiving nasogastric feeding
 - those shocked / on vasopressors
 - those with a coagulopathy (including uraemia) / anticoagulated
 - those receiving gastric mucosal irritants e.g. enteral steroids or NSAIDS
 - burns / polytrauma
 - 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 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 1mg every 4 to 6 hours until bleeding is controlled, for up to 5 days.

GLYCAEMIC CONTROL [39]

- There has been extensive investigation of tight glycaemic control in critically ill patients. The optimal target range remains controversial but we aim for 4.5-10.0mmol/l. Have a low threshold for commencing IV insulin infusion if blood glucose is persistently >10.0mmol/l.
- Never stop insulin infusions, instead reduce rate and start dextrose infusion to avoid hypoglycaemia. **DO NOT give boluses** of 20% or 50% dextrose to treat hypoglycaemia as these cause injurious (if transient) hyperglycaemia.
- 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 carefully. 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, mild, hypoglycaemia [40]. If the patient is cognitively normal, treat enterally. If not, start a dextrose infusion (100mls of 10% peripherally or 50mls of 20% centrally). The blood glucose must be rechecked within 15 minutes and the infusion rate titrated accordingly.

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. If, despite these measures, patients remain "BNO" for >1-2 days consider: PR examination / glycerine suppositories / phosphate enemas / laxido (NG).
- Any patient who has liquid stool diarrhoea must have a documented clinical assessment of the most likely cause. The principal classification is between diarrhoea due to an infection (norovirus, C. diff., others) and functional diarrhoea i.e. due to laxatives, NG feeding, pancreatic dysfunction, bowel inflammation, critical illness, constipation with overflow etc etc). If classified as likely to be infectious you must implement optimal infection control precautions to minimise cross contamination AND send stool sample for norovirus, C. diff., M,C & S. If classified as likely to be functional adopt sensible / proportionate hygiene measures but DO NOT send stool specimens for micro testing.
- Persistent / infected diarrhoea may be optimally managed by insertion of a bowel management system. This is often initiated and inserted by nursing staff. Severe coagulopathy or local pathology are relative contra-indications. Do not prescribe loperamide / codeine (except for high output stomas). Consider change of / increasing [Na⁺] in, NG feed.

MAINTENANCE [FLUIDS](#) – INTRAVENOUS AND ENTERAL

- Inputs: Patients with normal losses 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 may 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 [41]:

- 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 renin-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](#). 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 the [comparison Table](#) for more detailed information).
- Daily electrolyte requirements for a normal adult are:
 - Sodium 1-2 mmol/kg/day
 - Potassium 1 mmol/kg/day

Adverse effects of tissue oedema

<i>Tissue</i>	<i>Effects of oedema</i>
Brain	Confusion, delirium, agitation, coma and death
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 hypertension
Skin and soft tissues	Pressure area injury, impaired wound healing

Comparison of plasma to intravenous fluid therapies

[Electrolyte] in mmol/l	<i>Plasma</i>	<i>0.9%NaCl</i>	<i>5% Dextrose</i>	<i>4% Dextrose 0.18% NaCl</i>	<i>Hartmann's</i>	<i>Ringer's</i>
Na ⁺	140	154	0	30	131	130
K ⁺	4.0	0	0	0	5.0	4.0
Mg ²⁺	0.9	0	0	0	0	0
Ca ²⁺	2.5	0	0	0	2.0	2.5
Cl ⁻	98	154	0	30	111	109
PO ₄ ³⁻	1.2	0	0	0	0	0
Lactate	1.0	0	0	0	29	28
Osmolality mOsm/l	275-295	308	252	262	275	273
pH @ 37°C	7.40	5.0	4.0	4.0	6.5	6.5
Calories kcal/l			170	136		

PACKED RED CELL TRANSFUSION [42, 43]

- 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 [44].

- 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 70-100g/l, 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 <70 g/l [45]. Although this is an oxygen carrying capacity trigger it should also be considered a viscosity trigger [46]. 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 [45]. Be aware that most blood gas analysers are significantly less accurate at [Hb] measurement than formal haematology laboratories (errors as much as $\pm 2\text{g/dl}$).
- Try to minimise iatrogenic losses, in particular, blood sampling and during line insertion. Be conscious of the effects of iatrogenic haemodilution.
- Transfusion is not a benign intervention [47, 48]. 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 lose 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 [49] and trauma [50], the early use of PRBCs may 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) [50] although this is not without controversies [51-53].
- The place of intravenous iron and recombinant erythropoietin in the management of anaemia remains undefined in the critically ill population [54].

THROMBO-PROPHYLAXIS

- Every patient should receive an appropriate prophylactic dose of low molecular weight heparin (LMWH) unless they have a hypocoagulable TEG or they are receiving therapeutic anticoagulation or heparin (or argatroban) as anticoagulation for renal replacement therapy.
- Routine practice for patients >50kg is dalteparin 5,000units od.
 - For patients <50kg, consider a dose reduction to 2,500units od.
 - For patients >100kg increase the dose to 5,000units bd.
 - For patients >150kg increase the dose to 7,500units bd.
- For patients with an eGFR<30ml/min, use unfractionated heparin and titrate dose against daily aPTTr (target 1.3 - 1.6). Suggested starting doses:
 - <100kg 5,000units bd
 - >100kg 7,500units bd
 - >150kg 10,000units bd
- 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 unless they have peripheral vascular disease, chronic skin / soft tissue inflammation or acute injury / pathology.
- Compression boots may be useful alternative, especially in high risk patients.

SUSPECTED or PROVEN DVT / PE

- Commence full anticoagulation as soon as practical with once daily dalteparin (see weight based dosing in BNF).
- If at high risk of bleeding consider continuous unfractionated heparin infusion & / or an IVC filter.
- If haemodynamically unstable, consider thrombolysis. Give alteplase as a 10mg IV injection over 1-2 minutes, followed by infusion of 90mg over 2 hours; maximum dose 1.5mg/kg in patients weighing less than 65kg.

PRACTICAL/ INVASIVE PROCEEDURES

- The following procedures require the completion of an ["ICU Safety Checklist"](#) analogous to the "WHO Safety Checklist":
 - Percutaneous tracheostomy
 - [Bronchoscopy](#) - via any route
 - Insertion of pleural, peritoneal or other indwelling drain
- Pre-printed stickers of the Checklist should be available in the stationery draws in front of the main desk on the unit

GUIDELINES FOR THE INSERTION AND CARE OF LINES

- Prepare all materials required prior to insertion to avoid interruption and contamination. There is a dedicated line trolley - IT IS YOUR RESPONSIBILITY TO CLEAN AND RE-STOCK IT AFTER EACH USE.
- 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).
- A checklist is performed to ensure compliance with best practice.
- 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 using the audit sticker.
- If replacing a potentially infected / colonised line, always take a blood culture 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 try to 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 transduce the pressure waveform before stitching the line in to ensure venous placement as opposed to right ventricular or arterial placement.
- Please attach clean needleless ports to all lumens, bar the distal, monitoring lumen.

- 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 5,000units/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 3 sizes available, a 15cm for **RIGHT** IJ/subclavian, a 20cm for **LEFT** IJ/subclavian and a 24cm for **femoral** use. You **MUST** ensure the correct size is used at the correct site.
- Dialysis lines with a **THIRD**, small, central lumen (for drug administration in long term patients) are also available.

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 & 7647**. These are the on-call anaesthetic SpRs. Intubations on GICU are emergencies and should be treated as such.
- In order of escalating emergency, intubating equipment is available in the store room, the difficult airway trolley and the crash trollies. If you deplete one or more of the trollies, PLEASE ensure either you or another member of the team immediately restocks.
- 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 [55].
- 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 [NMB Table](#)).

- 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 may be 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.
- Any patient with a known or suspected difficult airway should have a difficult airway passport http://bit.ly/diff_arwy_psp completed. Having done so, please ensure you inform the bedside nurse and write boldly on the ICU chart "DIFFICULT AIRWAY PASSPORT".

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 O₂ tension is principally determined by the fraction of inspired oxygen (FiO₂), ventilation / perfusion matching and the blood's oxygen carrying capacity (essentially haemoglobin concentration).
- Assuming a fixed rate of CO₂ production, arterial CO₂ 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.

High flow, heated humidified oxygen - "Optiflow"

- Up to 80L/min flow with FiO₂ 0.21-1.0. Delivers the FiO₂ set and CPAP of 2.5-5.0cmH₂O via specialised nasal cannulae (2 sizes available) or tracheostomy.
- Very comfortable and well tolerated. Unlike mask CPAP / NIV, patients can eat, drink and talk during therapy. ENHANCES expectoration.
- Should be considered first line therapy when "wall" O₂ (max 15L/min) fails to achieve target SpO₂ / PaO₂.

Mask / helmet continuous positive airway pressure (CPAP)

- This is a high flow circuit providing a variable FiO₂ 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 H₂O and escalated to a maximum of +15 cm H₂O.
- 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, time cycled, 2 level 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 FiO₂, 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 without hypoxaemia.
- Therapy is usually commenced with an IPAP of 8-10 cmH_2O and an EPAP of 4-5 cmH_2O . 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 (atelectrauma 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 8).

GOALS OF SUPPORTIVE CARE

- $\text{PaO}_2 > 7 \text{ kPa}$ and $\text{pH} > 7.20$ whilst minimising VILI and cardiovascular compromise.

To treat hypoxaemia

- Consider performing a recruitment manoeuvre [56]
- 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 .

Mechanical ventilation guidance - Part 1 of 2

Things you need to set	Guidance notes
FiO ₂	<ul style="list-style-type: none"> Start high and reduce gradually. Minimise to achieve PaO₂ 8-10 kPa / SpO₂ 88-92%.
Modes (what effort is the patient making?)	<ul style="list-style-type: none"> 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)	<ul style="list-style-type: none"> Pressure cycled or pressure control: peak pressure is set, volume achieved is measured. Aim to keep peak / plateau / end inspiratory pressures ≤ 30 cmH₂O. Time cycled: A variant of pressure control in which 2 levels of pressure are set with durations $>2-3\times$ the spontaneous respiratory rate. Volume cycled or volume control: tidal volume is set, peak / plateau pressure is measured / limited. Inspiratory flow pattern can be set to continuous, decelerating (mimicking pressure control) or sinusoidal. In continuous flow, a variable length end inspiratory pause must be set.
Tidal volume target	<ul style="list-style-type: none"> 6-8 ml/kg ideal body weight in injured lungs otherwise ≤ 10 ml/kg ideal body weight

Mechanical ventilation guidance - Part 2 of 2

Things you need to set	Guidance notes			
Set rate	<ul style="list-style-type: none"> Set rate to achieve target minute volume and PaCO_2 (4.5-6.5 kPa or higher as long as $\text{pH} > 7.2$) 			
I:E ratio OR Inspired time " T_{insp} "	<ul style="list-style-type: none"> normally 1:2 consider 1:1-2:1 to recruit / optimise oxygenation (termed inverse ratio ventilation) consider 1:3-1:4 in the presence of expiratory airflow limitation / high intrinsic PEEP / air trapping 			
Pressure support	<ul style="list-style-type: none"> Initially set pressure support to achieve the same as peak inspiratory pressure and titrate to desired tidal volume 			
PEEP	<ul style="list-style-type: none"> 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 [57]. 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. 35cmH₂O for 30-60s followed by a decremental PEEP trial starting at either 10 or 12 cmH₂O. 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 [58-60]. 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 [61] 			
FiO ₂	0.21-0.4	0.4-0.5	0.5-0.7	0.7-1.0
PEEP	5	8	10	12-15

Additional considerations

- 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 [62].

UNCONVENTIONAL VENTILATION (APRV & HFOV)

Airway pressure release ventilation (APRV) [63, 64]

- 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 of 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.

APRV typical parameter settings

P_{high} 10-30 cmH₂O

Start at 25-30 cmH₂O / 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 ($60 \div T_{\text{high}} = \text{no. of releases per minute}$)

The effectiveness of **P_{high}** is often enhanced by longer values of **T_{high}**, such that to gain the same benefit with shorter values may require higher pressures. However, the optimal target is that **P_{high}** should be maintained for 90% of the time i.e. $T_{\text{high}} / (T_{\text{high}} + T_{\text{low}}) \times 100 = 90$. Hence the same effect can be achieved for a given **P_{high}** and a shorter **T_{high}** by shortening **T_{low}**.

T_{low} 0.2 - 0.8 seconds (THIS IS THE MOST CRITICAL SETTING)

Start at 0.2 seconds and titrate up / down until the termination of the release coincides with **75% of the peak expiratory flow rate** (PEFR). To determine this value, record / freeze the ventilator screen after a series of releases and use the cursor function on the expiratory flow-against-time graph to determine the value of the PEFR. Multiply this value by 0.75 and set **T_{low}** such that the ventilator cycles back to **P_{high}** at (or before) the PEFR has decayed to this value.

To improve oxygenation

Recruitment manoeuvre: set **P_{high}** to 30 cmH₂O & **T_{high}** to 30s for 2-5mins **see CAUTION above**. Then perform decremental **P_{high}** trial to a level above previous settings.

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

(Increase FiO₂ as needed)

To improve CO₂ 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 50% of expiratory flow (area under the curve)

Weaning using APRV

As oxygenation improves: reduce P_{high} and FiO_2 . **REMEMBER** to reassess and reduce T_{low} every time you reduce P_{high} . Employ a decremental trial by reducing the level of P_{high} in a stepwise fashion (e.g. 2 cmH₂O every 15mins) until SpO₂ is seen to fall, then go back 1 - 2 steps (with or without a recruitment manoeuvre).

As spontaneous tidal volume / PaCO₂ improves: increase T_{high} to reduce the number of airway releases, ultimately aiming for CPAP

Parameters to record

Settings FiO_2 P_{high} T_{high} T_{low}

Measurements Minute volume

Spontaneous respiratory rate which = total resp. rate -
($60 \div T_{high}$)

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 H₂O, 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 dyssynchrony 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 minimum 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.
- We routinely use conventional Portex Blue Line tubes. For large / complex patients we use Portex Uniperc tubes (flexible, armoured, adjustable flange tubes WITH inner cannula).
- For safe discharge to the ward, any patient with an ongoing need for their tracheotomy must either have an inner cannula in-situ OR have an uncuffed tube.
- 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) [65]

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 [66].

Pre 2011 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 (e.g. PAWP \leq 18mmHg)
- Pulmonary hypertension (common)
- ALI - $\text{PaO}_2 / \text{FiO}_2 < 40\text{kPa}$ ARDS - $\text{PaO}_2 / \text{FiO}_2 < 26\text{kPa}$

Berlin definition 2011 [67]

Timing - Within 1 week of a known clinical insult or new/worsening respiratory symptoms

Chest imaging - Bilateral opacities - not fully explained by effusions, lobar/lung collapse, or nodules

Origin of Oedema - Respiratory failure not fully explained by cardiac failure or fluid overload; Need objective assessment (e.g., echocardiography) to exclude hydrostatic oedema if no risk factor present

Oxygenation ($\text{PaO}_2 / \text{FiO}_2$) with PEEP / CPAP $\geq 5\text{cmH}_2\text{O}$

- Mild 26.6-40kPa (200-300mmHg)
- Moderate 13.3-26.6kPa (100-200mmHg)
- Severe $<13.3\text{kPa}$ (100mmHg)

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 [68]
 - 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
 - “Optimal” PEEP – recruit then decremental trial (see earlier section)
 - Minimise peak / plateau pressures ($\leq 30\text{cmH}_2\text{O}$)
 - Minimise FiO_2
 - Maintain spontaneous breathing
- Controversial ventilatory rescue therapies
 - Prone positioning
 - Unconventional ventilation (APVR)
 - Extracorporeal lung assist
- Adjunctive therapies with evidence of no benefit:
 - HFOV
 - Inhaled nitric oxide [69]
 - Nebulised prostacyclin
 - Almitrine
 - Recombinant surfactant
 - Salbutamol
- Adjunctive therapies under active investigation:
 - Sildenafil
- Controversies:
 - Open lung biopsy [70]
 - The role of high dose corticosteroids [71, 72]

FLEXIBLE BRONCHOSCOPY

Flexible bronchoscopy is an essential diagnostic and therapeutic procedure in the ICU. The majority of procedures are performed in intubated patients receiving mechanical ventilation. For generic guidance on awake fiberoptic bronchoscopy see [73, 74]. For training please approach the consultant of the week or Dr Ball.

ICU indications:

- Direct visualisation of the upper airway in difficult endotracheal intubation.
- Direct endotracheal visualisation and guidance during percutaneous tracheostomy.
- Inspection of the distal portion of an endotracheal or tracheostomy tube to assess patency and position.
- Inspection of the distal trachea and proximal bronchial tree for mucosal pathology / extrinsic compression.
- Removal of material obstructing one or more major bronchi.
- Performing sampling of distal airways for microbiological and / or cytological specimens.
- Guiding the placement of an endobronchial blocking / isolation catheter.

Equipment preparation and aftercare

- Prior to use, a bronchoscope must be thoroughly cleaned and disinfected. Cross infection between patients is a serious hazard [75]. Our clean scopes are stored, on trays, in a special clean cabinet. By manual inspection of the barcoded stickers in the central, flip top, opening section of each tray, take the scope that has been in the cabinet for the longest time i.e. has the oldest "washed on" time / date.
- The scope should be handled aseptically and transported in a clean grey container, lined with a new, single use liner and covered with a GREEN (CLEAN) plastic cover.
- Each scope should be carefully disconnected from the cabinet tubing, which attaches at 2 points, the suction valve opening and the suction tubing connection. A clean suction valve, made up of a 2 piece valve

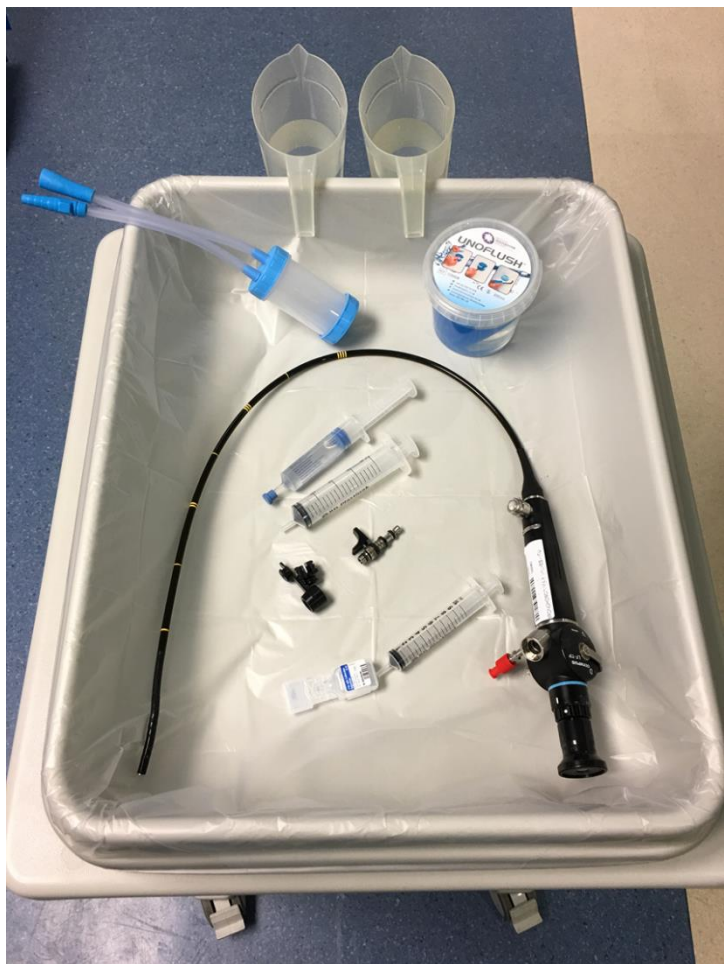
and a single patient use syringe cap should be retrieved from the basket in the centre of the tray, along with a dual barcoded sticker.

- Every scope removed from the cabinet must be tracked for infection control purposes using the barcoded stickers. Use the paper forms located on the cabinet door. One copy of the sticker goes in the patient's notes, the other goes on the form together with the patient's details back to decontamination with the scope. The template for the forms can be found at http://bit.ly/bscope_track
- The scope should be checked for clear vision and a functioning suction system immediately prior to use. The outside of the scope should be wiped with saline soaked gauze. Avoid water based gel lubricants as these can dry out and become sticky rather than lubricate. In situations where there is problematic sticking of the scope to the inside of an endotracheal or tracheostomy tube use sterile liquid paraffin, a supply of which should always be available in the lotions and potions section of the drug cupboard on the main unit.
- The bronchoscopist and any assistants should wear full protective clothing including gowns, gloves and face / eye cover. Appropriate and sensible precautions should be made regarding the potential aerosolisation of infected material.

Essential equipment (this should be clean or sterile single use):

- Light source
- Camera / videoscope stack (optional)
- A packet of sterile gauze
- A sterile 1 litre jug.
- A 500ml bag of 0.9% sodium chloride
- A 20ml syringe
- Sputum traps
- Dedicated suction
- A bronchoscopy catheter mount

see picture on next page



- At the end of the procedure, gently clean the outside of the scope using the special sponges provided. Then, suction up the entire contents of the specialist cleaning solution. In hours, it is the responsibility of the bronchoscopist, to return the used scope to the decontamination area of the endoscopy unit. If out of hours, please ensure the scope is returned for decontamination at the earliest opportunity. The scope should be left in a prominent position with the RED (DIRTY) plastic cover over the top of the grey container. If used, the stack must be cleaned and stored appropriately.

Patient preparation and aftercare

- Whenever practical, inform the patient regarding the proposed procedure and gain their consent.
- Depending upon the indication, likely duration and clinical condition of the patient, an appropriate plan regarding topical anaesthesia, sedation and neuromuscular blockade should be made.
- At a minimum, continuous ECG and SpO₂ monitoring together with intermittent, automated non-invasive blood pressure should be used.
- The FiO₂ should be increased to 60-100%. If ventilated, a pressure control mode is preferable although a strictly pressure limited volume control mode can be used. Hypoventilation is an inevitable occurrence during the procedure. In patients in whom even transient hypercapnia needs to be avoided, a series of timed, short bronchoscopies can usually be safely performed. In such circumstances, continuous end tidal capnography is essential. Alternatively, consider using high frequency ventilation.
- At the end of the procedure it is the responsibility of the bronchoscopist to ensure that there is a record of the procedure (Indication / Findings / Procedures / Specimens sent) together with a clear sedation and ventilatory management plan recorded in the patient's notes and communicated. They must also ensure that all specimens are appropriately labelled, request forms completed and specimens sent urgently, preferably via the tube system, to the labs. Please phone the labs to ensure the specimens have arrived safely.

Procedure

- Before starting, ensure all necessary equipment is available and working, the patient is comfortable and the bronchoscopist is ergonomically positioned. Consider slowly injecting 3-5ml of sterile saline into the endotracheal / tracheostomy tube to lubricate the passage of the scope. At least one assistant must be present to watch the patient, the ventilator and the monitoring, in addition to being able to assist the bronchoscopist.
- First navigate the endotracheal / tracheostomy tube and ascertain whether the tube is encrusted with secretions. If so, this may inhibit both the procedure and present the patient with unwanted additional resistance. It may be possible to effectively clean the tube using the scope, however, electively changing the tube is sometimes preferable.
- Next, consider whether the tube opens centrally within the trachea and is a sufficient distance from the main carina. If this is not so, reposition the tube under bronchoscopic guidance and make note of the optimal position using the visible reference markers on the tube. Always consider whether moving the patient will adversely affect the position of the distal end of the tube and leave a detailed description in the patient's notes.
- Then go on to examine the distal trachea, in particular, looking for mucosal trauma caused by the distal tip of the tube and blind suction catheter insertion. Continue by inspecting the remainder of the accessible bronchial tree in a logical order. It should be possible to visualise the first 2-5 divisions of each lobar bronchus. Always examine the "normal" before progressing to the "abnormal".
- For visible secretions, try to sample / remove without causing trauma to the mucosa and WITHOUT using saline lavage. If required, collect a specimen for microbiological examination. Difficult to clear secretions can often be removed piecemeal. Large pieces can often be held against the tip of the scope by application of continuous suction. The scope, together with the offending mass, can then be removed en mass, by slowly withdrawing the scope whilst maintaining suction. If this fails, a trial of a SLOW injection of a few mls of 0.9% NaCl can be tried to dislodge / break up masses BUT DO NOT inject in such a manner as to push such masses distally and cause them to impact / occlude bronchi. The use of biopsy forceps

and cytology brushes may be useful but require skill and patience. Should flexible bronchoscopy fail, consider using a rigid scope. Obstructing aggregations of inspissated secretions, blood clots or mucosal sloughing may require prolonged or multiple procedures.

- For visible mucosal bleeding, haemostasis will usually occur spontaneously. Haemostasis can be augmented by topical vasoconstriction using adrenaline (epinephrine). Gently instil / irrigate with a 1 in 10,000 (0.1mg ml⁻¹) solution. Topical antifibrinolytics, such as neat tranexamic acid (100mg ml⁻¹) can also be useful.
- If the bleeding is distal to the main carina and either unstoppable and / or distal to the limit of visualisation, temporary isolation and tamponade can be achieved by wedging the tip of the bronchoscope into the origin of the identified bronchus. The efficacy of instilling vasoconstrictors and / or antifibrinolytics is uncertain. The value of prolonged continuous suction is also debatable. If this fails to achieve haemostasis, then a balloon tipped bronchial isolation catheter can be inserted parallel to the bronchoscope, which can then be used to guide catheter placement. This can be a difficult procedure due to the aerosolisation of blood within the airway masking any vision. Be careful not to dislodge the blocking catheter when withdrawing the scope. Consider paralysing the patient and applying a high level of PEEP. If practical, turn the patient bleeding side down. As a further adjunct, connect and instil oxygen through the bronchoscope suction channel at a high flow rate. Definitive treatment for persistent haemorrhage is either selective bronchial angiography and embolisation or surgery.

Specimens

- Whenever possible, send undiluted secretions for micro-biological investigation. To obtain a specimen from a region of interest beyond visualisation, first locate the nearest lobar, segmental or subsegmental division, then gently wedge the tip of the bronchoscope into it. Before any sampling, ensure that the suction channel is clear of any proximal secretions, which might contaminate the specimen. This may require the scope to be fully with-drawn, the channel flushed and rinsed with clean saline and the scope reinserted and positioned. Next, apply the specimen trap as close to the bronchoscope as possible. Slowly instil 20-60mls of 0.9% sterile

saline, wait for a few seconds, then apply continuous low level, suction. If the airway completely collapses, ask an assistant to gently turn the strength of the suction down until some fluid is drawn into the specimen trap. When no further fluid flows, slowly withdraw the scope whilst maintaining continuous suction. Make a note of the volume instilled and the volume of the specimen. Examine the specimen for adequacy, looking for the presence of mucoid or infected airway secretions. It is worth discussing the optimal handling of specimens with the labs receiving them, in particular, if qualitative or semi-quantitative microscopy are required or specific pathogens are suspected. If a good quality, large volume specimen is obtained, this can often be divided in the laboratory for both microbiological and cytological examination if required. This prevents repeated saline lavage and scope trauma both of which are injurious.

- Blind brush specimens may be useful in both cytological and microbiological testing. Brush and biopsy specimens of visible lesions can also be taken. In patients receiving positive pressure ventilation, transbronchial biopsy and transbronchial needle aspiration specimens carry a small but significant risk of pneumothorax and pneumomediastinum and are best avoided.
- Protected lavage and brush catheters are available but are of questionable value. There is conflicting evidence regarding the value of bronchoscopic specimens over and above those obtained by blind endotracheal suctioning, most especially in the diagnosis of ventilator associated pneumonia.

Complications

The following complications can occur and should be prepared for:

- Displacement of the endotracheal / tracheostomy tube out of the trachea.
- Obstruction of the endotracheal / tracheostomy tube.
- Obstruction of the trachea or major bronchus
- Hypoxia / derecruitment / increasing ventilatory requirements post procedure
- Hypercapnia / underventilation
- Coughing
- Bronchospasm

- Haemorrhage
- Pneumothorax
- Pneumomediastinum
- Sepsis secondary to translocation (bacteraemia) [76]
- Hypo or hypertension / cardiac arrhythmias

CARDIOVASCULAR FAILURE AND SUPPORT

- The principle role of the cardiovascular system is to deliver adequate amounts of 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 MAY include:
 - Low volume peripheral pulses
 - Cold peripheries, especially with proximal extension / peripheral capillary refill >2s (except in distributive shock states where peripheries may 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)
 - A lactic acidosis / a reduced central or mixed venous oxygen saturation
- 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. 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. Rate of change from abnormal to normal are useful indicator of response to therapy and are prognostic [77-79]
 - Central venous-to-arterial carbon dioxide difference (Pcv-aCO_2) $\leq 0.8\text{kPa}$ [80]
 - See also [81]

For detailed information on the following topics please follow the links:

- Atrial fibrillation [82]
<http://onlinelibrary.wiley.com/doi/10.1111/anae.12050/full>
- Acute myocardial infarction [82]
<http://onlinelibrary.wiley.com/doi/10.1111/anae.12050/full>
- Pulmonary embolus [83]
<http://onlinelibrary.wiley.com/doi/10.1111/anae.12051/full>

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
	Tachy-arrhythmia	Anti-arrhythmic drugs / cardioversion	optimal mechanical efficiency is achieved at a heart rate of ~90/min in sinus rhythm, <90 in any other rhythm
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.
	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)	- ≥3cmH ₂ O 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 & / or Hct 0.3-0.4
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
	Distributive shock (septic, spinal, anaphylactic)	Vasopressor drugs	These are arbitrary targets that require individualisation to the patient and the pathology

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 (DO₂) is the amount of oxygen delivered to the tissues per unit time.
- DO₂ = CO x arterial oxygen content (CaO₂)
- DO₂ = (HR x SV) x (([Hb]/100 x 1.34 x SaO₂/100) + (PaO₂ 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 O₂ each gram of Hb can carry if 100% saturated

SaO₂ = arterial oxygen saturations divided by 100 to convert from %

PaO₂ = the partial pressure of oxygen in arterial blood in kPa

0.000225 is ml of O₂ dissolved per ml of blood per kPa of O₂

- DO₂ = 950-1150ml/min
- It is worth noting that the major determinants of DO₂ 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/m² CI = 2.2-3.5 l/min/m² DO₂I = 500-600 ml/min/m²

- Oxygen Consumption (VO₂) is the amount of oxygen consumed by the tissues per minute. VO₂ = Cardiac Output x (arterial oxygen content) – (mixed venous oxygen content-SvO₂)
- VO₂ = 180-320ml/min
- Central venous saturation (ScvO₂) is a guide to the difference between oxygen being delivered to the tissues and its extraction or use. The normal value is 70-75%. This measurement has been successfully used to guide resuscitation resulting in improved morbidity and mortality [49].
- A useful additional marker is Pcv-aCO₂, normal value ≤0.8kPa. For more information regarding this and pitfalls of ScvO₂ see [80, 81, 84].

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 subclavian. 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 semirecumbent 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.
- Patients with dysrhythmias should have the following electrolyte levels targeted: serum Mg^{2+} >0.9 mmol/l & K^+ 4.5-5.5mmol/l.
- The commonest arrhythmia in critically ill patients is atrial fibrillation (AF). Iatrogenic precipitants should be considered and acted upon (e.g. furosemide, insulin, salbutamol, inotropes etc).
- 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: metoprolol, esmolol, sotalol, digoxin (may control rate) or amiodarone. Verapamil and flecainide are usually avoided as they are both profoundly negatively inotropic.
- For more detailed information see [82]
<http://onlinelibrary.wiley.com/doi/10.1111/anae.12050/full>

Intravenous fluid resuscitation, the dynamic fluid challenge or preload optimisation*

- 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 4-8cmH₂O (caution in interpretation is required in patients receiving positive pressure ventilation).
 1. No or minimal response \Rightarrow relative hypovolaemia.
 2. Gradual and sustained rise (≥ 2 cmH₂O) 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 999-1,500ml/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 [85].
- 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 [49]. 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 vasoconstrictors, 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 [86] and methylene blue [87]. 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 dependent 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 vasopressor resistant / refractory vasoplegic 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 to gluco / mineralo corticoids is unclear. Use of “physiological replacement” dose hydrocortisone (60-240mg/24hrs) remains controversial [88].
- 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 $\geq 15\%$ 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/m²). 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 ($\geq 20\%$ 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 $\geq 20\%$ 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 vasopressor support

CARDIOVASCULAR EFFECTS OF VASOACTIVE DRUGS AND DEVICES

Drug	Receptor avidity				Physiological effects				
	DA	α	B ₁	B ₂	HR	CI	MAP	SVR	MO ₂
Dopamine									
<5 g/kg/min	3+	+	+		=/+	=/+	=/+	=	+
5-10 g/kg/min	3+	2+	2+	+	+	+	+	+	+
>10 g/kg/min	3+	3+	2+	+	2+	+	2+	2+	2+
Dobutamine	+/-	+	3+	2+	+	2+	=/-	=/-	3+
Dopexamine	2+		+	3+	2+	+	=/-	=/-	2+
Adrenaline									
<0.2 g/kg/min		+	+	+	+	+	+	+	2+
>0.2 g/kg/min		3+	2+	+	2+	2+	2+	2+	3+
Noradrenaline									
<0.2 g/kg/min		2+	+	+/-	=/+	=/+	2+	2+	2+
>0.2 g/kg/min		3+	+	+/-	=/+	=/+	3+	3+	2+
Metaraminol		2+	+		+	+/-	2+	3+	2+
Ephedrine		+	2+		2+	2+	+	+/-	2+
Phenylephrine		2+			=/+	-	3+	3+	2+
Milrinone					2+	2+	2-	2-	2+
Levosimendan					+	2+	2-	2-	?=
Vasopressin / analogues					=	-	3+	3+	2+
Methyl blue					=	-	2+	2+	2+
IABP (device)						2+	+	-	2-
Metoprolol			++	+	2-	-	-	-	-
Labetalol		++	++	+	-	-	--	--	-
Amiodarone									
Enteral					-	- / +	=	=	-
Parenteral					-	2-	-	-	-
Digoxin					-/=	=/+	=	=	=/+
GTN					=/+	-/+	-	-	-
Hydralazine					-/+	+	--	--	-
Sodium nitroprusside					=/+	-/+	-	-	-
Nimodipine					=/+	-/+	-	-	-

[Key: DA dopamine receptors; α B₁ B₂ adrenergic receptor types; MO₂ myocardial oxygen demand; + increased, = unchanged, - decreased; IABP intra-aortic balloon pump]

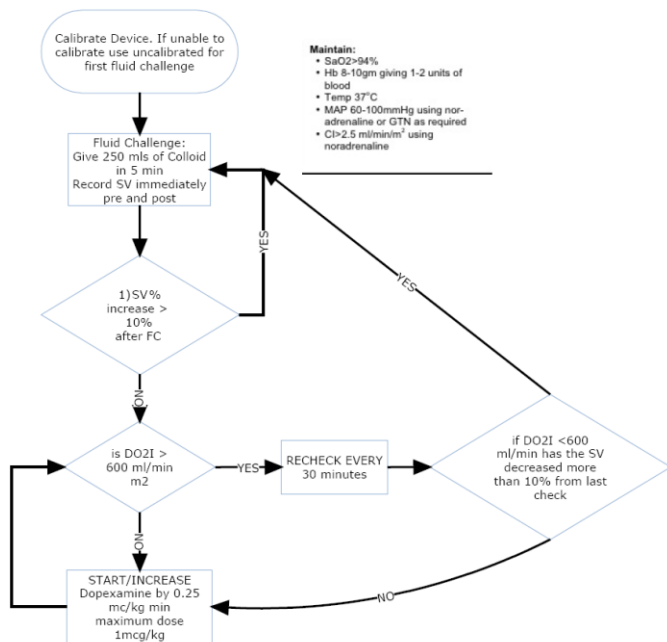
DOSAGE REGIMENS OF COMMONLY USED VASOACTIVE DRUGS

<i>Drug</i>	<i>Dosage range</i>	<i>Notes</i>
Dopamine DON'T EVER USE THIS	1-20 mcg/kg/min	Dominant effect dependent upon dose. Some evidence of worse outcome as compared to other drugs. 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-inflammatory?
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.
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	1unit bolus (repeated if necessary) then 0.5-4units/hr	Vasoconstrictor. 2nd line vasopressor in norepinephrine resistant shock [89] (see also Functional hypoadrenalism)
Terlipressin (vasopressin analogue)	0.25-2 mg bolus OR 1.3 mcg/kg loading then 1.3-5.2mcg/kg/hr	Vasoconstrictor. Duration of action 4-6 hours. Alternative to vasopressin (>V1a & <V2 affinity). infusion maybe superior to bolus dosing [86, 89, 90].
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 [87] (see also Functional hypoadrenalism)
GTN	0.5-30 mg/hr [91]	Rapid tachyphylaxis over 12-24hrs
Hydralazine	Bolus 1-10 mg slowly Infusion 200-300 µg/min reduce to 50-150 µg/min	Arterial vasodilator
Labetalol	15-160 mg/hr	Alpha and beta blockade
SNP	0.5-8 mcg/kg/min	Arterial and venodilator
Metaraminol	0.5-2 mg bolus	Emergency "noradrenaline"
Phenylephrine	100-500 mcg bolus	"Pure" vasoconstrictor = more pressure but less flow

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/m² respectively [92].
- 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/m² has been demonstrated to be a successful strategy in reducing morbidity and mortality [93].
- $\text{DO}_2\text{I} = \text{Cardiac Index (CI)} \times \text{Arterial oxygen content}$. 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-existing limiting co-morbidities especially ischaemic heart disease.

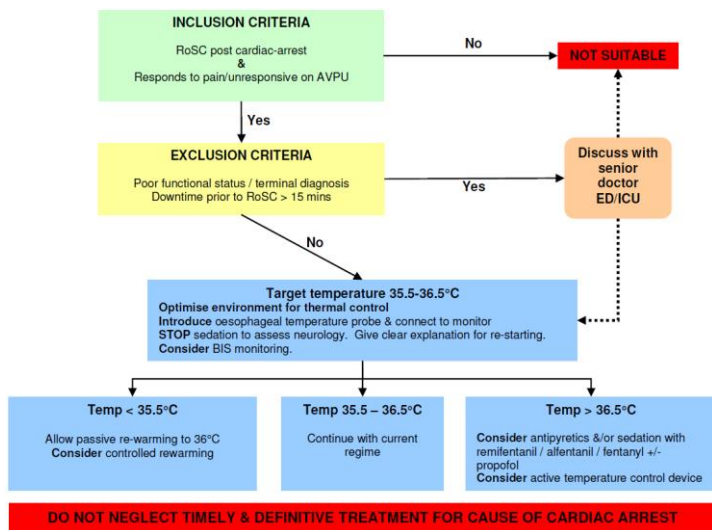
POST OPERATIVE HAEMODYNAMIC GOAL DIRECTED THERAPY FLOW CHART



POST CARDIAC ARREST - TARGETTED TEMPERATURE MANAGEMENT (TTM)

- We target **36.0°C** rather than 32-34°C.
- Spontaneous / unintentional hypothermia is common and should be managed with passive rewarming. However, please be aware that rewarming at $>0.5^{\circ}\text{C}$ per hour probably causes a secondary brain injury following a global hypoxic-ischaemic insult.

BASIC TTM ALGORITHM



ADDITIONAL NOTES & PHYSIOLOGICAL TARGETS

System	Target	Notes
Airway	Intubate	Don't forget nasogastric tube
Breathing <i>ABG temp corrected</i>	Mechanical ventilation PaO ₂ 8.0-13.0kPa (SpO ₂ 92-97%) PaCO ₂ 4.5-5.5kPa	Maintain spontaneous breathing efforts if possible Avoid both hypoxia & hyperoxia (\downarrowtemp$\Rightarrow\downarrow$VO₂) Avoid both hypo and hypercapnia (\downarrowtemp$\Rightarrow\downarrow$PaCO₂)
Circulation	Heart rate 60-90bpm (sinus rhythm) MAP 60-80mmHg ScvO ₂ \geq 75% or SvO ₂ \geq 70%	Optimal treatment for cause of cardiac arrest Use flow monitoring to assess fluid responsiveness, adequacy of cardiac output & oxygen delivery by trending indices of global oxygen balance (i.e. central or mixed venous oxygen saturations).
Disability (<i>& dextrose</i>)	Minimal / no sedation. Maintain ability to assess GCS / neurology / seizures / myoclonus. If possible, use BIS monitoring.	Use remifentanyl / alfentanil / fentanyl +/- propofol. Treat shivering with opiates first, then atracurium 50mg boluses. There is no effective treatment for myoclonic status, if present the prognosis is very poor. See seizure management below.
	Maintain blood sugar 6.0-8.0mmol/l	Commence and maintain standard NG feeding.
Electrolytes [Measure 6hrly]	Na ⁺ 135-145mmol/l ----- K ⁺ 4.0-5.0mmol/l ----- Mg ²⁺ >0.8mmol/l ----- Ca ²⁺ 1.0-1.3mmol/l ----- PO ₄ ³⁻ >0.8mmol/l -----	Avoid hypo- and hyper-natraemia <i>Ionised</i> from gas machine NOT <i>total</i> from lab
Seizures / myoclonus	No seizures or Intermittent, self terminating seizures lasting <5mins with recovery to baseline Differentiating between seizures and myoclonus can be challenging. Seek senior advice, use BIS monitoring and request a formal EEG as soon as practical.	For generalised tonic clonic seizures: Termination of seizures lasting >5mins use either propofol infusion (+/- boluses) OR midazolam boluses 2-10mg. For recurrent / refractory seizures use sequentially: 1. Load with IV sodium valproate 30mg/kg then commence 15mg/kg NG (IV) within 12 hours. 2. Load with IV levetiracetam 40mg/kg then commence 20mg/kg NG (IV) within 12 hours. 3. Clonazepam 2mg qds with potential escalation to 5mg qds.

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

PREVIOUS DEFINITIONS (2003) [94]

- 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 from 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 2 or more of the following clinical findings:
 - Body temperature $>38^{\circ}\text{C}$ or $<36^{\circ}\text{C}$
 - Heart rate $>90/\text{min}$
 - Hyperventilation evidenced by respiratory rate higher than 20/min or $\text{PaCO}_2 <4.2\text{kPa}$
 - White blood cell count $>12,000\text{cells}/\mu\text{l}$ or $<4,000/\mu\text{l}$
- 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 2015

www.survivingsepsis.org &

http://www.survivingsepsis.org/SiteCollectionDocuments/SSC_Bundle.pdf

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, revised in 2008, 2012 and 2015. It represents a set of evidence based recommendations, detailed below:

To be completed within 3 hours (of triage in the emergency department or, if presenting from another care venue, from the earliest chart annotation consistent with all elements of severe sepsis or septic shock):

- Measure serum lactate
- Obtain blood cultures prior to antibiotic administration
- Administer broad-spectrum antibiotic(s)
- Administer 30 ml/kg crystalloid for hypotension or lactate ≥ 4 mmol/L

To be completed within 6 hours:

- Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) ≥ 65 mmHg
- In the event of persistent hypotension after initial fluid administration (MAP < 65 mm Hg) or if initial lactate was ≥ 4 mmol/L, re-assess volume status and tissue perfusion by performing and documenting:
 - A repeat physical examinationAnd / or 2 of the following:
 - CVP / ScvO₂ / Bedside cardiovascular ultrasound / Dynamic assessment of fluid responsiveness with passive leg raise or fluid challenge
- Re-measure lactate if initial lactate elevated.

As a unit we broadly support these goals, as they are “common sense”. However, these recommendations represent the bare minimum rather than the best care possible for an individual.

NEW SEPSIS DEFINITIONS 2016 [95]

In February 2016, the 3rd international consensus conference published its conclusions. The pragmatic ramifications of these are beginning to emerge.

BRAIN INJURY AND SUPPORT[96]

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, normocarbida, normotension, normothermia, normoglycaemia and normonatraemia. In particular, hypercarbia, hyperthermia, hyponatraemia, and both hypo and hyperglycaemia are associated with secondary brain injury. Hypocarbida causes cerebral ischaemia and should only be considered ONLY as a short term temporising measure to permit safe transfer to CT or theatre.
- Position the patient 30° 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 PaCO₂ and core temperature.
 - Patient positioning (see above)
 - High dose propofol or thiopentone infusion (barbiturate coma) to reduce cerebral metabolic demand.
 - Aggressive management of seizures (valproate / keppra NOT phenytoin).
 - 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 [9]

- This term has different definitions in different countries.
- It is an emotive area that may be difficult to communicate to next of kin. 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.'
- The process in diagnosing brain death is divided into three parts:

Preconditions

- The patient must be in an apnoeic coma and on a ventilator making no 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

- Hypotension - MAP <60mmHg
- Hypothermia - core temperature <34°C
- Dysnatraemia - serum Na⁺ <115mmol/l OR >160mmol/l
- Hypokalaemia - serum K⁺ <2.0mmol/l
- Dysmagnesaemia / dysphosphataemia - serum levels <0.5 OR >3.0mmol/l **AND** no muscle response to a peripheral nerve stimulator
- Dysglycaemia - serum glucose <3.0 OR >20.0mmol/l
- Poisoning, use of sedative and neuromuscular blocking drugs must be excluded or accounted for.
- No clinical suspicion of severe hypo or hyper thyroidism OR severe adrenal insufficiency

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 this area. The time of death is time when the first set of tests confirms the presence of brain stem death.

- 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 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.
 - Prior to the test increase the PEEP to 10cmH₂O and commence capnography monitoring. Perform an ABG to establish the ETCO₂-PaCO₂ gap (usually 0.5-1.0kPa).
 - Reduce the Vmin to achieve a steady state ETCO₂ corresponding to a PaCO₂ 6-7kPa and a pH of 7.30-7.40 then check an ABG to confirm.
 - Increase the FiO₂ on the ventilator to 70-100%, for at least 3 minutes prior to the test, aiming for an SpO₂ of 100%.
 - Using the NIV / Mask mode of ventilation, set the ventilator to deliver CPAP at 10cmH₂O, disable all of the alarms, in particular the apnoea backup. Observe the patient for any respiratory effort for 5 minutes before repeating the ABG. The PaCO₂ must have increased by >0.5kPa with no detectable respiratory effort for the test to be positive.

- This test must be conducted twice, so restart IPPV, aiming for a steady state ETCO_2 corresponding to a PaCO_2 6-7kPa then continue as above.
- NOTE: the time of death is the time the first apnoea test is positive.

A form to record brainstem death testing can be found at:

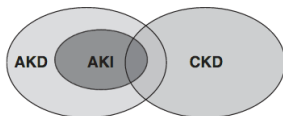
<http://www.gicu.sgul.ac.uk/resources-for-current-staff>

ORGAN AND TISSUE DONATION

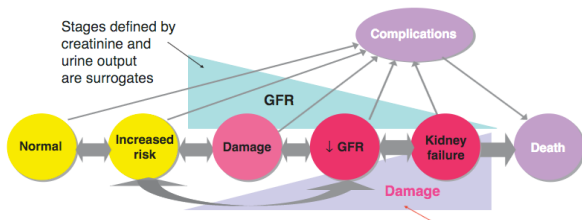
- In the UK, organ and tissue donation are governed by the Human Tissue Act 2004, see: <http://body.orpheusweb.co.uk/HTA2004/20040030.htm>
- Always consider donation in dying patients, especially in those with brain stem death and, in particular, if brain injury is the sole organ failure.
- The decision to refer a patient to the Transplant co-ordination service MUST be discussed with the on-call consultant before contact is made.
- To discuss any aspect of donation, contact the local transplant co-ordinator through the hospital switchboard.
- For specific guidance on the management of patients prior to and following brain stem death testing, who are potential organ donors please see: <http://www.gicu.sgul.ac.uk/resources-for-current-staff>

RENAL FAILURE AND SUPPORT

- Acute kidney injury (AKI) is a common complication of critical illness. It may, or may not be associated with acute kidney disease (AKD).



Overview of AKI, CKD, and AKD. AKI is a subset of AKD. Both AKI and AKD without AKI can be superimposed upon CKD. Individuals without AKI, AKD, or CKD have no known kidney disease



Conceptual model for AKI. Red circles represent stages of AKI. Yellow circles represent potential antecedents of AKI, and the pink circle represents an intermediate stage (not yet defined). Thick arrows between circles represent risk factors associated with the initiation and progression of disease that can be affected or detected by interventions. Purple circles represent outcomes of AKI. “Complications” refers to all complications of AKI, including efforts at prevention and treatment, and complications in other organ systems.

- 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 [97] see Figure. Very minor modifications to these criteria were made in 2007 by the AKI network and are termed the AKIN criteria [98]. These have now been superseded by the KDIGO criteria see Table below.

- AKI is associated with increased morbidity and mortality regardless of the primary pathology, with a direct correlation between the severity and outcome.
- Oliguria is defined as an hourly urine output $<0.5\text{ml/kg}$ ideal body weight, for >6 hours.
- Clinically, oliguria is the first presenting sign but occurs late in the natural history of AKI. But note, oliguria may also be a sign of “renal success” in response to hypovolaemia / hypotension.
- Prevention, by maintaining adequate intravascular volume and 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 potential precipitant of acute renal injury. Prevention with intravenous prehydration is well established. Whenever safe and feasible, give 500ml 1.4% NaHCO_3 immediately (<30 mins) prior to contrast injection in all patients with acute or chronic renal injury.
- The extent of renal damage can be inferred from the combination of the extent of function loss (estimated GFR) AND the degree of proteinuria, which at the lower end of the damage spectrum is best assessed by urinary albumin loss.

Urine albumin to creatinine ratio (ACR)

- Adult reference range: $<3.0\text{ mg/mmol}$
- $3.0 - 30.0\text{ mg/mmol}$: Moderately increased
- $>30.0\text{ mg/mmol}$: Severely increased

Urine protein creatinine ratio

- Adult reference range: $<15\text{mg/mmol}$
- In pregnancy: $<30\text{mg/mmol}$ indicates significant proteinuria

Problems associated with sampling:

- Results can be affected by physiological factors such as erect posture, exercise or acute diuresis
- Urine samples should not be collected after undue exertion or acute fluid loads.
- Urine ACR is specific for albumin; it is however possible to lose significant amounts of other proteins of lower molecular size (e.g. in renal tubular disease or in light chain disease) without necessarily seeing an increase in albumin loss.
- Some drugs may cause an analytical interference in assessing protein levels.

AKI-KDIGO and CKD staging criteria

Acute kidney injury - see [99] & review articles [100, 101]		
Stage	Serum creatinine	Urine output
1	a 1.5-1.9x increase from baseline over <7 days OR ≥26.5μmol/l increase from baseline over <48 hours	<0.5ml/kg/hr for 6-12 hrs
2	a 2.0-2.9x increase from baseline over <7 days	<0.5ml/kg/hr for 12-24 hrs
3	a ≥3.0x increase from baseline OR Value ≥ 353.6μmol/l over <7 days	<0.3ml/kg/hr for ≥24hrs OR Anuria for ≥12hrs

Chronic kidney disease		
Functional Stage	Definition	GFR (ml/min/1.73m ²)
1	Kidney damage with normal GFR	≥90
2	Kidney damage with mild decrease in GFR	60-89
3A	Mild-to-moderate decrease in GFR	45-59
3B	Moderate-to-severe decrease in GFR	30-44
4	Severe decrease in GFR	15-29
5	End-stage renal disease	<15

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/minol	30-300 mg/g 3-30 mg/minol	>300 mg/g >30 mg/minol
GFR categories (ml/min/ 1.73 m ²) Description and range	G1	Normal or high	≥90			
	G2	Mildly decreased	60-89			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

MEDICAL MANAGEMENT OF ACUTE OLIGO / ANURIC RENAL FAILURE

- Optimise renal perfusion (intravascular volume, cardiac output, renal perfusion pressure).
- Actively manage fluids, electrolytes and drugs to avoid iatrogenic / preventable injury.
 - Avoid indiscriminate / untargeted fluid boluses .
 - Avoid maintenance fluids in excess of needs / losses.
 - Avoid excessive loading of Na, Cl, K and PO₄
 - Consider the effects of altered drug pharmacokinetics
- Medical management of hyperkalaemia
 - defined as: K⁺ >6.5mmol/l OR K⁺ >5.5mmol/l and rapidly rising at >0.25mmol/hr for 2 or more hours below
 - **GIVE - CONTINUOUS** insulin (actrapid) infusion starting at 2units/hr together with a **CONTINUOUS** infusion of dextrose (20-50mls of 10% peripherally OR 10-30ml of 20% via a central venous line).
 - **DO NOT GIVE** a "one off" infusion of 10-15units of insulin in 50ml of 50% dextrose as this results in **REBOUND HYPERKALAEMIA** within 30-60minutes and frequently causes problematic dysglycaemia.
 - In the event of ECG changes, give 20mls of 7.35% (10mmol) of CaCl₂, preferably over 10 minutes via a central venous line.
 - CONSIDER the use of adjunctive medical therapies such as IV sodium bicarbonate 1.4% (peripherally) or 8.4% (centrally)
- To manage worsening renal acidosis (bicarbonate loss) give IV sodium bicarbonate 1.4% (peripherally) or 8.4% (centrally)
- Furosemide may aide in the management of fluid overload in non-oliguric patients BUT has no beneficial effect on disease progression or outcome of acute oligo/anuric renal failure [102]. If used indiscriminately, it can worsen outcome by reducing renal perfusion.
- "Low dose" dopamine has no place in the prevention or treatment of acute renal failure [103].
- For guidance on heptorenal syndrome see [104] - full text available at: <http://www.gicu.sgul.ac.uk/resources-for-current-staff/renal-replacement-therapy/>

CONTINUOUS RENAL REPLACEMENT THERAPY (cRRT)

Common indications for initiating / continuing cRRT

- Hyperkalaemia (K^+ $>6.5\text{mmol/l}$ OR K^+ $>5.5\text{mmol/l}$ and rapidly rising at $>0.25\text{mmol/hr}$ for 2 or more hours). **This is time critical.** USE potassium free replacement fluid as the first bag.
- Correction of severe / unresolving acidosis ($\text{pH} <7.1$) in particular, acidosis associated with cardiovascular compromise (shock i.e. end organ hypoperfusion) / high vasoactive drug requirements (noradrenaline $>0.5\text{mcg/kg/min}$ / dobutamine $>10\text{mcg/kg/min}$).
- Uraemia (urea $>40\text{mmol/l}$ or rising by $>12\text{mmol/24hrs}$)
- Fluid overload causing severe hypertension and / or problematic oedema (e.g. abdominal compartment syndrome) and / or contributing to hypoxaemia / poor lung compliance.
- Other indications include encephalopathy, hyperpyrexia (note even with maximal circuit warming, patients usually lose a minimum of 1°C core body temperature during cRRT)

Size of kidney and target blood pump speed

- There are 2 sizes of kidney available HF 12 (1.2m^2) and HF19 (1.9m^2). The default option should be HF12 with a target blood pump speed of $\geq 250\text{ml/min}$.
- If clearance targets are not achieved with an HF12 and / or the patient is very tall / muscular / catabolic then use an HF19 BUT the target blood pump speed should be $\geq 300\text{ml/min}$.
- Failure to achieve the target blood pump speed results in blood stasis + haemoconcentration within the kidney and both treatment failure and circuit loss due to clot obstruction within the kidney.

Modes of cRRT

- Haemofiltration (convection only - CVVH) - usual mode - BECAUSE, permits predilution hence longer circuit life AND convection has greater efficiency than diffusion
- Haemodiafiltration (convection and diffusion - CVVHDF) - when enhanced SMALL solute clearance is needed e.g. when CVVH fails

to achieve target goals in 6-24 hours or some drug overdoses (e.g. salicylate) -

- Slow continuous ultra-filtration (SCUF) - if fluid removal is all that is required - USE CVVH, 10ml/kg/hr, split 90% predilution + 10% post replacement, NOT "SCUF" setting on machine in order to preserve circuit life.

NOTE - our current machines can switch mode of cRRT at any time.

Prescribing cRRT

PLEASE USE THE SPECIAL CHARTS. These can be found at::

<http://www.gicu.squl.ac.uk/resources-for-current-staff/renal-replacement-therapy/>

- For patients, in whom their metabolic derangement is felt to be contributing to their acute condition / instability, START at a DOSE of 20ml/kg/hour of "replacement fluid". This fluid principally contains sodium bicarbonate - Na 140mmol/l HENCE be very cautious if the patient's Na is <130 or >150mmol/l.
- Actively titrate dose AND / OR mode to achieve predefined goals of therapy. Suggested goals:
 - K^+ <6.0mmol/l within 2 hours (using potassium free replacement fluid)
 - pH rising by 0.5 within 6 hours
 - MINIMUM SOLUTE CLEARANCE should be 12mmol/l of urea every 24 hours
 - Fluid balance goals will depend upon the patient's ability to tolerate removal
- For all other scenarios start at 15ml/kg/hour of "replacement fluid".
- Our standard starting practice for CVVH is to apportion 1/3 of "replacement fluid" as "pre-dilution" and 2/3 as "post replacement".
- Our standard practice for CVVHDF is to apportion half of the "replacement fluid" as the counter current and half as "post replacement".
- Our standard practice is to set fluid removal at a MINIMUM of 50mls/hr. The rate of fluid removal can be increased up to a maximum of 2000ml/hr
- Circuit anticoagulation:

- Unless the patient is known or suspected to have a hypersensitivity to unfractionated heparin (including heparin induced thrombocytopenia), circuits should be primed with a dilute heparin solution (10,000 units in 1000ml of 0.9% NaCl).
- PLEASE ensure that there is NO AIR in the bubble trap as any air-blood interface is highly thrombogenic
- The circuit should then be placed in "RECIRCULATION" mode for a minimum of 20 minutes before connection to a patient (UNLESS treatment is time critical).
- Once connected, first line therapy is a continuous infusion of unfractionated heparin into the proximal end of the circuit. The usual dose is 400-1,000 units per hour. The target aPTT is 1.5-2.0.
- HOWEVER, if the patient requires THERAPEUTIC anticoagulation - ensure the target is set and achieved.
- In problematic circuits, minimising the procoagulant stimulus by maximising the blood pump speed and increasing the proportion (up to 90%) of replacement fluid that predilutes the patient's blood should also be considered.
- cRRT circuits have a maximum 80 hour lifespan. Unplanned, premature circuit loss, usually through problematic vascular access, is both very expensive and commonly results in 10-20g/l loss in the patient's [Hb], necessitating pRBC transfusion. Do everything possible to avoid this and seek specialist help early.
- Whenever a circuit dose fail and / or reaches the end of its life, always consider a period OFF cRRT to assess the patient's renal recovery.

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.
- Polytrauma patients, especially those with brain injury and / or haemorrhagic shock 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 - ICU ADMISSION AND DAILY THEREAFTER

(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 (Peak airway pressure <30cmH₂O and mandatory breath Vt 6-8ml/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 Ca²⁺>1.0 mmol/l (on blood gas). Temp >36.5. Tranexamic acid given [105]. TEG performed and acted on (see later section).
- 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.

POLICY FOR THE MANAGEMENT OF INTUBATED VENTILATED PATIENTS IN ED **WITH NO** SIGNIFICANT LIFE THREATENING INJURY / PATHOLOGY

Background

With the advent of the helipad, it is estimated that 20-30% of patients delivered by helicopter have only minor injuries. However, for safety during transfer, they are often intubated and ventilated at scene. It is anticipated that unless haemodynamically unstable, these patients will go direct to CT in ED and from there to resus. In the event that no significant injuries are detected and there is no clear indication for ongoing sedation / airway protection / ventilatory support, then the patient should be woken so that they can be assessed with a view to extubation, if appropriate. This policy aims to outline the pathway for such patients.

Referrals

As soon as the patient is stable and in resus, the trauma team leader should make contact with the GICU SpR on bleep 7980 or via x1307 to inform them of the details of the case. Unless the patient has an obvious single system, non-orthopaedic injury, they should be admitted under the Trauma team

Plan A

The patient is transferred to GICU to be woken up and assessed.

Before transfer, it is expected that the following will have been completed and documented in the patient's Trauma booklet. The original booklet should stay with the patient and the photocopy retained in ED.

- A CT "primary assessment", including radiological spinal clearance
- A repeat primary survey (compared to the on scene HEMS primary survey)
- As comprehensive a secondary survey as possible, including a log roll
- A list of injuries and plan for each
- A completed spinal clearance form and removal of all unnecessary immobilisation
- All wounds cleaned, sutured and dressed, or a documented plan to go to theatre for this including the surgical team responsible

- A nasogastric (or orogastric) tube inserted and the stomach effectively decompressed
- A urinary catheter inserted and urine drained (if there is a distended bladder on CT)
- A clear management plan regarding Tetanus prophylaxis made and acted upon
- Blood sent for FBC, clotting profile (including fibrinogen), U&E, LFTs, Ca, PO₄, Mg, Troponin, alcohol, group and save
- Urine sent to biochemistry for a drugs of abuse screen

It is suggested that this list be used as a checklist.

The reasons for these expectations are as follows:

- Regrettably, there is often an unavoidable delay of 30-60 minutes whilst an ICU bed is prepared.
- Most patients who are intubated at scene have an altered level of consciousness / agitation and the assumption should be that they HAVE a diffuse brain injury.
- Spinal immobilisation is a significant cause of preventable pressure area injury and should be removed at the earliest opportunity.
- If a peripheral injury requires an x-ray this can be done more easily in ED
- The GICU team are not well equipped nor necessarily skilled, at wound closure
- Many trauma patients arrive in ICU without gastric decompression, which compromises their ventilation, places them at risk of aspiration and is essential if the patient is to be woken up. Similarly, the urinary catheter.
- Tetanus vaccine and human anti-tetanus immunoglobulin are kept in ED
- The blood alcohol and urinary drugs of abuse screen may inform the differential diagnosis of the on scene, or, on waking, agitation.

Plan B

GICU does not have a bed but CTICU and / or Neuro ICU have a bed

The GICU SpR will liaise with the CTICU / Neuro ICU consultant to arrange that the patient be admitted to the unit with the least bed pressures. This is preferable to transferring a GICU patient to one of the other units to facilitate the admission of the ED trauma patient to GICU. The reason this is preferable is that the latter scenario involves moving 2 patients and involves a non-clinical transfer for the ICU patient, which cannot be in their best interests and for which we are penalised by commissioners.

Plan C

There is no bed that can be made available on any of the 3 units within 60 minutes

If the available ED and Anaesthetic personnel believe that there is a high clinical probability that the patient should be a straight forward to wake, wean and extubated then they should proceed with this, in resus. If the Anaesthetist feels that transferring the patient to a recovery area is more practical / desirable, then they should liaise with senior members of the team in that area and execute their plan. If neither of these options are practical then the patient should be kept on minimal sedation in ED whilst the ICU consultants and senior nurse (SG591) create a bed solution.

GUIDELINES FOR REFERRAL OF TRAUMA PATIENTS WITH INJURIES

The Trauma team leader should refer any patient with one or more of the following criteria:

Patient criteria

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

ABCD (physiological) criteria - post resuscitation

- A. Injury to, or that might compromise, the airway
- B. Hypoxaemia ($\text{PaO}_2/\text{FiO}_2 \leq 40 \text{ kPa}$) **and / or** hypercapnia ($\text{PaCO}_2 \geq 6.5 \text{ kPa}$ despite a minute volume $\geq 100 \text{ ml}$ per kg ideal body weight)
- C. Haemodynamically unstable (persistent tachycardia and / or hypotension post resuscitation and surgical control of bleeding)
- D. GCS ≤ 13 (any cause)

Injury criteria

- 2 or more major injuries
- Suspected or proven, unstable, spinal injuries **with or without** spinal cord injury

Treatment criteria

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

Post resuscitation & intervention 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 its 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's EDs) should be delivered to St George's ED 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, preferably pre-transfer, via the Image Exchange Portal (IEP).
- Ensure tertiary survey completed within 24 hours of patient arrival.

Copy of the Trauma Booklet spinal clearance form

The purpose of this form is to clearly document the current status of the patient to ALL members of the multidisciplinary team. It should be completed by the Trauma Team Leader before the patient leaves the ED and amended as soon as further information becomes available.

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. 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? If there are injuries are they stable or unstable?

	Necessary		Performed		RADIOLOGICAL CLEARANCE		Date	By whom (PRINT)
	No	Yes	No	Yes	Yes	No. The injuries are The spine is stable / unstable		
C-spine								
T & L spine								

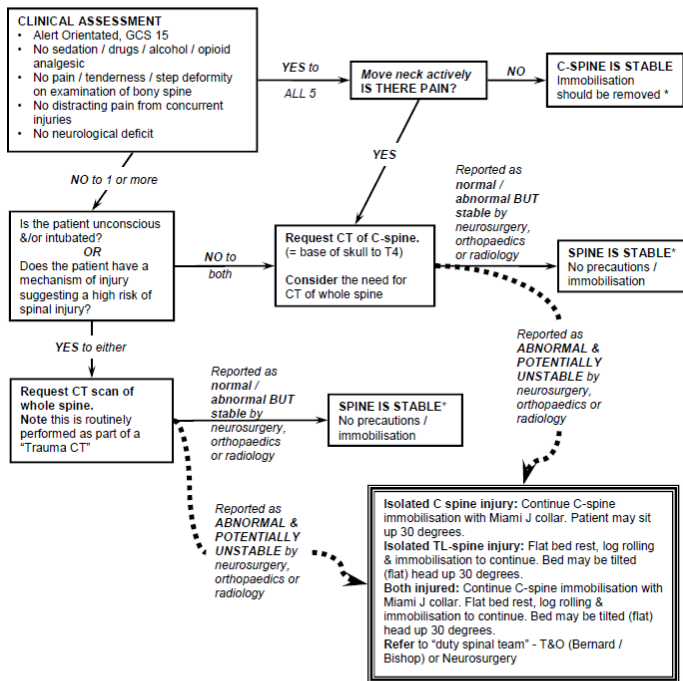
3. Management plan. It is intended that the plan will progress to no precautions over time.

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

SPINAL CLEARANCE FLOW DIAGRAM

Use this flow chart IF:

- from the mechanism of injury there is a risk of spinal injury AND / OR
- there are symptoms or signs of bony spinal / spinal cord injury



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

Neurological deficit referable to spinal injury requires CONSIDERATION of urgent MRI and ICU review regarding risk and management of ascending injury and spinal perfusion pressure / spinal shock.

For printable version goto: http://bit.ly/spine_clear

THROMBO-ELASTOGRAPHY (TEG)

TEG is a near patient, assessment of whole blood, coagulation and fibrinolysis [106]. Fresh blood is withdrawn into a PLAIN syringe, 1ml added to a kaolin reagent bottle, the sample gently mixed and 0.36ml pipetted into a prepared cup. The cup oscillates through a fixed small angle every 10 seconds. An initially stationary pin connected to a torsion wire is immersed in the clotting blood. As the clot progressively strengthens it binds the pin to the cup resulting in increasing torque on the pin, which is transduced by the wire. Points plotted at the extremes of these excursions describe a bifurcating curve whose components yield a variety of information. The distance (in millimetres) between the arms of a TEG curve varies with the strength of the clot, reaching a maximum, and then reconverging as the clot undergoes fibrinolysis.

TEG analysis measures various components of clotting time and strength. This analysis is much more representative of clotting *in vivo* than conventional PT / aPTT tests. However, TEG “assumes” a core temperature 37°C, pH of 7.35-7.45, haematocrit 0.3-0.5 and an ionised Ca^{2+} 1.0-1.3mmol/l (blood gas). Furthermore, TEG cannot detect the *in vivo* contribution of endothelial cells or shear forces of blood flow on local clot formation and fibrinolysis.

The precise role of TEG in managing the bleeding patient is evolving. It should be considered complimentary to conventional coagulation tests, of which, platelet count and fibrinogen level are the 2 most important in ongoing haemorrhage. Both GICU and CTICU have TEG machines. In order for an effective clot to form *in vivo*, 3 stages of coagulation occur, initiation, amplification and propagation - for animation see:

<http://www.hemophilia.org/> hover over, “Researchers and Healthcare Providers” tab then select “Cell based Coagulation Animation” from the menu, then follow the on-screen instructions.

In short, initiation requires both platelet function (minor) and sufficient clotting factors (major), in particular factors VII and X. In the amplification phase, platelet activation occurs, forming the platform for the activation of numerous factors. During propagation, the factor cascade rapidly generates large quantities of first thrombin then fibrin, which polymerises. The stability of the forming clot is dependent upon both platelet function and haematocrit.

A TEG should be performed on every patient with any of the following conditions, in whom **treatment** of the apparent problem is being contemplated **and** to measure the response to therapy:

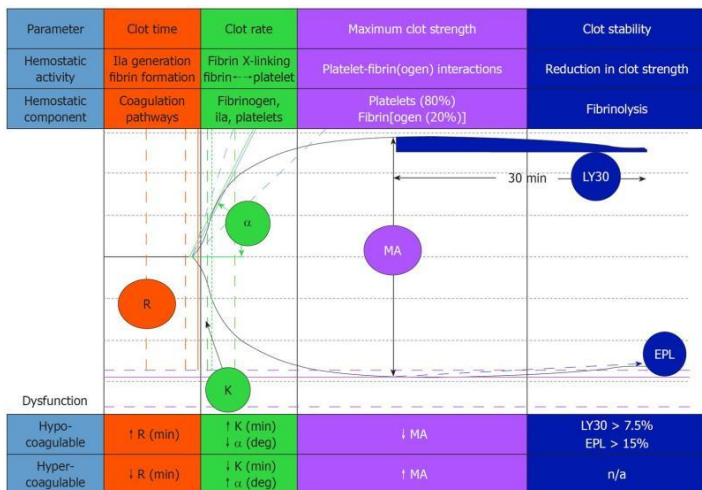
- Haemorrhagic shock
- Post massive transfusion
- A bleeding patient with a clinical diagnosis of apparent coagulopathy (any cause, including iatrogenic e.g. heparin, anti-platelet therapy etc)
- Suspected or proven Disseminated Intravascular Coagulation (DIC)
- Suspected or proven hypercoagulable state

Unless familiar with our machine, you will need training to be able to:

- Perform the test
- Interpret the test

Dr Cecconi and Dr Ball are the expert users.

A QUICK GUIDE TO INTERPRETATION:



“R time” is the time taken for a sufficient clot to form in the cup, so that the cup’s rotation starts to turn the pin. It is principally a measure of the enzymatic clotting cascade (aPTT + PT). A prolonged “R-time” is most commonly due to inadequate quantities of clotting factors OR the presence of anticoagulants (heparin, warfarin etc). If the former, then treat with fresh frozen plasma (15ml/kg), for the later, reverse the anticoagulation (protamine, beriplex +/- vitamin K, etc). In complex cases, you can establish whether heparin is the cause by running a second sample in a “heparinase” cup.

“K time” is the time taken for a clot to reach an amplitude of 20mm. The **“ α angle”** is calculated as the angle from the horizontal between “R time” and “K time”. Both are principally measures of fibrin polymerisation but are secondarily dependent upon the speed and extent of the thrombin burst, which in turn is partially dependent upon platelet number and function. A prolonged “K time” / “ α angle” suggests hypofibrinogenaemia, the treatment for which is cryoprecipitate. However, if there is also a prolonged “R-time” fresh frozen plasma may be more appropriate.

Maximum amplitude (**MA**) is the maximum diameter of the clot formed. It is ~80% dependent upon platelet number / function and 20% on fibrin polymerisation. **“G”** is calculated from MA and represents the shear elastic modulus strength and is deemed to be a more sensitive measure of platelet number and function. These parameters are measures of clot strength. A low value implies either thrombocytopenia or the presence of platelet blocking drugs. A high value, especially in the context of a short “R time” and / or “ α angle” suggests a hypercoagulable state that might warrant therapeutic anticoagulation +/- anti-platelet therapy. Note that haematocrit (Hct) also influences MA. As fibrin polymerises it traps RBCs, which disrupt clot growth. Thus as Hct falls below 0.2, the MA will start to increase. Thus interpretation of the test must include consideration of this variable

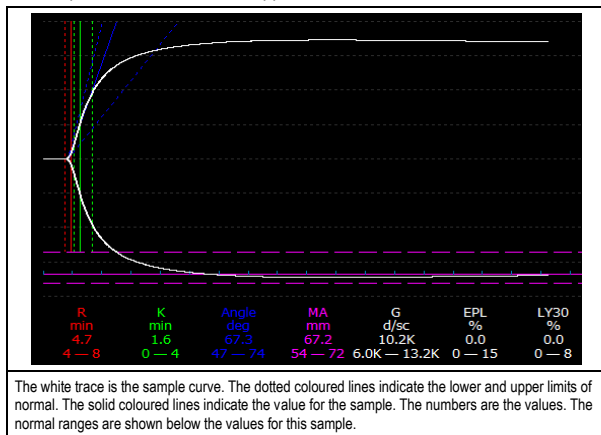
“LY30” is the percentage of MA that lyses in 30 minutes. The estimated percent lysis (**EPL**) is the estimated rate of change in amplitude after MA is reached. In the coagulopathy of trauma / haemorrhagic DIC, there is often hyperfibrinolysis. If detected, then treat with tranexamic acid.

If in any doubt, please seek expert guidance including a discussion with the Haematology SpR on call.

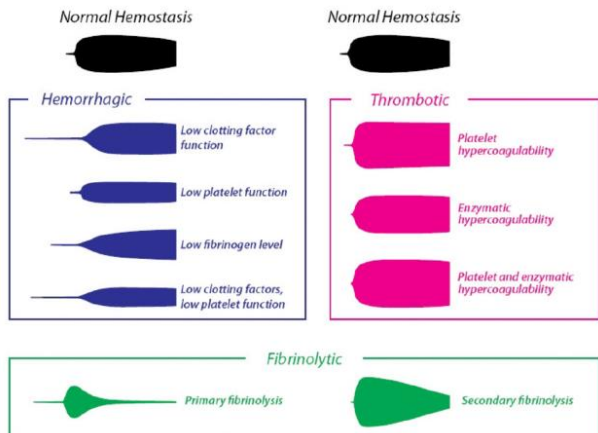
For more information goto:

<http://www.gicu.sgul.ac.uk/teaching/resources/haematology>

An example of a TEG trace as it appears on the machine



Examples of commonly encountered curve shapes and their interpretation



SUGGESTED MANAGEMENT ALGORITHM BASED UPON TEG ABNORMALITIES

In a patient who appears to be, or is at risk of, bleeding (e.g. about to undergo a surgical procedure).

TEG values	Probable cause BUT consider "assumptions"	Suggested treatment
R time > 8mins	Inadequate clotting factors	FFP 12-15ml/kg
$\alpha^\circ < 47^\circ$ (K time > 4mins)	Hypofibrinogenaemia	Cryoprecipitate x2
MA < 54mm	Inadequate platelet function (&/or numbers) ~80% + / - Hypofibrinogenaemia ~20%	Platelets x1 AND CONSIDER Cryoprecipitate x2
LY30 $\geq 8\%$	Primary hyperfibrinolysis	Tranexamic acid 1g IV bolus then 1g IV infusion over 8 hours

Notes

- If R time 4-8mins **AND** α° 47-74° **AND** MA 54-72mm and the patient **IS** bleeding, the cause is **NOT** a lack of clotting factors / platelets. Consider, core temperature, pH and ionised Ca²⁺ level (blood gas), NOT FORGETTING occult vascular / organ injury.
- If R time > 8mins **AND** $\alpha^\circ < 47^\circ$ **AND** MA < 54mm **GIVE** FFP 12-15ml/kg **AND** Platelets x1.
EXPLANATION - FFP contains some fibrinogen hence cryoprecipitate **MAY NOT** be necessary. However, in massive transfusion, recent data **SUGGESTS** an advantage to the early use of cryoprecipitate.
- In primary fibrinolysis **EXPECT** R time > 8mins **AND** $\alpha^\circ < 47^\circ$ **AND** MA < 54mm. If R time, α° and MA are normal OR (more commonly) R time < 4mins **AND** $\alpha^\circ > 74^\circ$ **AND** MA > 72mm, then a state of secondary hyperfibrinolysis exists and tranexamic acid is contra-indicated.
- If R time < 4mins **AND** / **OR** $\alpha^\circ > 74^\circ$ **AND** / **OR** MA > 72mm the patient is hypercoagulable and maximal mechanical + / - chemical thromboembolic prophylaxis should be a priority.

GICU FORMULARY

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

<i>Indication</i>	<i>Rx</i>	<i>Notes</i>
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	Metaclopramide 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 [107] OR Neostigmine 2mg IV bolus over 3-5mins [108]	5mg in 50mls NS @ 4-8ml/hr
Acute liver failure* [Encephalopathy, INR>3.5, lactate>3.5mmol/l & glucose <4.0mmol/l]	N-acetylcysteine 150 mg/kg/h over 1 hour, followed by 12.5 mg/kg/h for 4 hours, then continuous infusions of 6.25 mg/kg 67 hours	NOT for paracetamol poisoning, ischaemic liver failure or pregnancy associate liver failure [109]

CARDIOVASCULAR SYSTEM

Dosage regimens of commonly used vasoactive drugs - "uppers"

Drug	Dosage range	Notes
Dobutamine	5–20 mcg/kg/min	Inodilator
Dopexamine 1st line in GICU	0.25–2.0 mcg/kg/min	Positive chronotrope / Inodilator / Anti-inflammatory?
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	1unit bolus (repeated if necessary) then 0.5–4units/hr	Vasoconstrictor. 2nd line vasopressor in norepinephrine resistant shock [89] (see also Functional hypoadrenalism)
Terlipressin (vasopressin analogue)	0.25–2 mg bolus OR 1.3 mcg/kg loading then 1.3–5.2mcg/kg/hr	Vasoconstrictor. Duration of action 4–6 hours. Alternative to vasopressin (>V1a & <V2 affinity). Some evidence to suggest infusion superior to bolus dosing [86, 89, 90].
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 [87] (see also Functional hypoadrenalism)
metaraminol	0.5–2 mg bolus 5–20 mcg/kg/min infusion	Emergency "noradrenaline" = more pressure but less flow 10mg in 20mls (low pH = veno-irritant)
phenylephrine	100–500 mcg bolus 40–200 mcg/min infusion	"Pure" vasoconstrictor = more pressure but less flow 10mg in 500ml = 20mcg/ml = 120–600ml/hr
ephedrine	5–25 mg every 5–10 min	Max 150 mg per 24 hours

Dosage regimens of commonly used vasoactive drugs - “downers”

GTN	0.5-30 mg/hr [91]	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	15-160 mg/hr	Alpha and beta blockade
Esmolol	load with 500 mcg/kg in 60s then 50-250 mcg/kg/min	Beta blocker broken down by serum esterases hence very short duration of action
Metoprolol	1-15mg bolus qds infusion 1-3mg/hr	1 st line IV beta blocker. Safe in renal failure. Use maximum tolerated dose
SNP	0.5-8 mcg/kg/min	Arterial and venodilator
Amiodarone	<p>Loading IV</p> <p>Max rate 150 mg in 15 mins</p> <p>Usual 300 mg in 60 mins then 900 mg over 23 hours</p> <p>then 600-1600 mg daily</p> <p>Min cumulative loading dose 10g</p>	<p>IV preparation made in solvent mixture of polysorbate 80 and benzyl alcohol, which are both potent negative inotropes. Rapid IV loading may result in hypotension due to this -ve inotropy. Treat by slowing the rate of infusion or stopping.</p> <p>Due to unpredictable and very variable pharmacokinetics total loading doses in excess of 10g over 1-3 weeks are required. Enteral bioavailability is 35-65% of IV</p> <p>The IV preparation can cause an acute hepatitis. Chronic therapy may cause pulmonary fibrosis, cirrhosis, thyroid dysfunction and skin photosensitivity.</p> <p>As monotherapy, only cardioverts ~50% of AF to SR. Median time to cardioversion AF to SR 7 hours.</p> <p>In short, second line therapy, load aggressively, enterally, if possible, consider co-therapy (beta blockers), use for shortest possible time as end organ toxicity related to total cumulative dose.</p>

RESPIRATORY

<i>Drug</i>	<i>Dosage range</i>	<i>Notes</i>
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 elimination 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)
Ipratropium bromide	250-500mcg nebulised 4-6 hourly	Bronchodilator
Budesonide	1mg nebulised bd	Inhaled steroid for intubated / ventilated patients.
Carbocysteine	1.5-2.5g/day NG in divided doses	Mucolytic. Probably of no benefit in ventilated patients.
Hypertonic saline NOT 0.9% "normal" saline	5% NaCl 500ml polyfusor OR Add 10 x 10mls of 30% NaCl to 400ml of 0.9% NaCl (remove 100mls from a 500ml bag first) 10ml nebulised 4-6 hourly	<i>Probably</i> the best therapy for thick secretions [110, 111]. DO NOT USE nebulised N-acetylcysteine [110, 112]
Sildenafil	25-50mg tds NG	POTENTIAL rescue therapy in acute or acute on chronic pulmonary hypertension.
Epoprostenol (flolan)	50ng/kg/min inhaled	POTENTIAL rescue therapy in acute or acute on chronic pulmonary hypertension. Administer as a continuous infusion into the ventilator circuit immediately distal to the HMEF [113].

see also second table overleaf

<i>Drug</i>	<i>Dosage range</i>	<i>Notes</i>
Lidocaine (lignocaine)	10ml 1% solution NEBULISED up to 2 hourly	POTENTIAL rescue therapy in acute severe asthma / acute decompensated COPD (with bronchospastic features) [114]
Lidocaine (lignocaine)	Load with 2mg/kg IV over 5 min, followed by 3mg/kg/hour for 10 min see also CNS dosing	POTENTIAL rescue therapy in acute severe asthma / acute decompensated COPD (with bronchospastic features) [115]
Methylpredisolone	Loading 1 mg/kg Maintenance 1 mg/kg/day for 14 days	In refractory ARDS patients before day 14, preferably before day 7 [72]. De-escalate by gradual reduction over 14 days. For full protocol see [71]
Heparin	10,000-25,000units nebulised 4-6 hourly	Consider in ALI / ARDS secondary to inhalational injury. DOES have variable systemic absorption so APTT must be monitored closely [116].
Sodium bicarbonate 4.2% (50% dilution of 8.4% with sterile water)	5-10ml nebulised 4-6 hourly	Consider as an alternative / adjunctive mucolytic. May also have a role in toxic inhalation / inhalational burns [117].

Continuous infusion sedative analgesic regimes (continues overleaf)

<i>Drug</i>	<i>Regime</i>	<i>Notes</i>
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.
Remifentanyl	Dose 0.025 – 2.0 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 [118-120].
Ketamine [121, 122]	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. Consider using as a second line anticonvulsant anaesthetic agent [123].
Dex- medetomidine	0.2-1.4 mcg/kg/hr	Similar to clonidine but has a much greater affinity for α_2 -receptors over α_1 -receptors (1,620:1 compared to 200:1 for clonidine). Hence higher incidence of bradycardia especially with loading doses / high dose infusions. Consultant approval required for use.

<i>Drug</i>	<i>Regime</i>	<i>Notes</i>
Lidocaine	load 1.5mg/kg over 10mins then 1.5mg/kg/hr [124, 125] dose range 0.25- 1.5mg/kg/hr	Potential sedative adjunct / opiate sparing agent in complex patients. Be aware of accumulation of active (hepatic) metabolites, effects of protein binding, possible negative inotropic (and chronotropic) effects, induction of seizures and potentiation of NMBs (and weak NMB effect) hence risk of CINM? MUST have daily cessation to avoid accumulation. May reduce rate of cerebral oedema formation [126]
Thiopental sodium	Load with 0.5-5.0mg/kg over 10-15s Then infusion at 0.5-5.0mg/kg/hr	Rapid onset general anaesthetic. Can cause severe cardiovascular depression. Very lipophilic so cumulative dose sequestered in tissues resulting in prolonged recovery. Considered as a last resort in intracranial hypertension and status epilepticus. Infusion should always be titrated to continuous EEG variables.

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 [127]
Midazolam	Loading dose 30 - 300 mcg/kg Maintenance 30 - 200 mcg/kg/hr (0 - 14 mg/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 \equiv 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 hypertrophic 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, epileptogenic and causes SIADH. Multiple drug interactions therefore contra indicated in patients on any antihypertensives, SSRIs, tricyclics and warfarin.

Regular / bolus dose sedation

<i>Drug</i>	<i>Regime</i>	<i>Notes</i>
Haloperidol	2.5 – 5 mg NG / PO / IV Max daily dose 20mg	Often delayed onset of action in patients with agitated ICU delirium. Can prolong QT interval.
Chlorpropazime	10 – 250 mg NG / PO / IM	Alternative to haloperidol.
Olanzapine	5 – 15 mg NG / PO daily	Alternative to haloperidol. Max 30mg/24hr
Risperidone	1 – 4 mg NG / PO / s/l	Alternative to haloperidol. Max 16mg/24hr. Use half the dose in renal failure.
Quetiapine	50 – 200mg NG / PO	Alternative to haloperidol [32]. Max 800mg/24hrs
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.

Neuromuscular blocking drugs

<i>Drug</i>	<i>Bolus dose</i>	<i>Onset & Duration</i>	<i>Side effects</i>
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 pharmacokinetics in renal and hepatic failure. Causes histamine release hence contraindicated in acute severe asthma. Inactive metabolite, laudanosine, lowers seizure threshold
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 [128-130]

Anticonvulsants

Drug	Regime	Notes
Valproate FIRST LINE	Loading 30 mg / kg IV @ 10 mg / kg / min Maintenance 15 mg / kg / hr, 12 hrly	Maintenance doses should be NG unless gut dysfunction suspected. Do not use if the patient is receiving carbapenems as they induce valproate metabolism.
Levetiracetam SECOND LINE	Loading dose 40 mg/kg IV @ 6 mg / kg / min Maintenance 20 mg / kg / hr, 12 hrly	Maintenance doses should be NG unless gut dysfunction suspected.
Clonazepam	1-5mg 6 hrly	Only enteral formulations available
Lacosamide	Loading dose 200mg IV over 15-60 mins Maintenance 100mg IV 12 hrly	Only after discussion with a senior clinician. Gradual increases in maintenance therapy up to 200mg BD can be considered.

DO NOT ROUTINELY USE phenytoin. Only use phenytoin IF the patient has been established on phenytoin for a prolonged period (months - years)

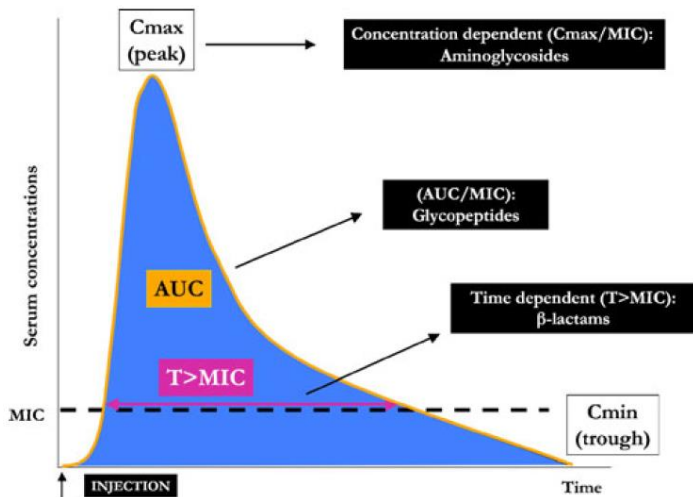
For refractory convulsant status epilepticus - seek Neurology advice

ANTIMICROBIALS

Adult Critical Care Empirical Antimicrobial Therapy Policy - July 2016

Also available via Microguide [online](#) and as an App

- All antimicrobial prescriptions must include the indication and stop / review date in both the medical notes AND on the drug chart OR in Cerner Millennium.
- Empirical antibiotics must be stopped after 48hrs **UNLESS** there is clear clinical **AND** / **OR** microbiological evidence to continue treatment.
- There have been increasingly frequent isolates of carbapenem resistant G-ve organisms in long stay ICU patients at St George's (all 3 units). PLEASE limit the use of carbapenems whenever possible.
- 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 48 hours
 - Enteral 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 ICU on antibiotics should have the intended duration of treatment written in the notes / ICU discharge summary and on the drug chart.
- Optimal dosing of antibiotics in critically ill patients remains an area of active research, with under dosing and drug toxicity are real concerns. For a review of this topic see [131]. As a starting point see diagram below. In order to optimise beta-lactam antibiotic therapy we give a rapid loading dose followed by extended infusions. Please refer to the drug specific notes below.



- **High dose dexamethasone in suspected bacterial meningitis** is controversial. To be effective it **HAS TO BE** given **BEFORE** (or with) the first dose of antibiotics. After this time point, the negative effects outweigh any benefit. Retrospective analysis of the limited data on which this recommendation is based suggests that the patients that probably benefit are those with a high CSF bacterial load, with *Strep. pneumoniae* and *H. influenzae* BUT NOT *N. meningitidis*. **Contra-indications include:** pre-treatment with parenteral antibiotics; recent brain injury (including neurosurgery +/- CSF shunt); AND septic shock. The recommended dose is 8mg, 6 hourly for 4 days. Please discuss the use of dexamethasone with a consultant **BEFORE** giving [132].
- **Corticosteroids in severe pneumonia** remains controversial. The best evidence suggests such therapy is safe but of **NO BENEFIT** [133].

Adult Critical Care Empirical Antimicrobial Therapy Policy - Part 1 of 2

Indication	1st line	If reliable Hx of TRUE penicillin allergy (see notes)	For
Community acquired pneumonia	Co-amoxiclav 1.2g 8hrly + Clarithromycin 500mg 12hrly	Levofloxacin 500mg daily	5-10 days
Hospital acquired pneumonia	Co-amoxiclav 1.2g 8hrly + Amikacin 15mg/kg daily (if severe / shocked)	Levofloxacin 500mg daily + Amikacin 15mg/kg daily (if shocked)	5-7 days
Ventilator associated pneumonia (VAP)	Tazocin 4.5g 8hrly + Amikacin 15mg/kg daily (if shocked)	Meropenem 1g 8hrly + Amikacin 15mg/kg daily (if shocked)	5-7 days
VAP if MRSA colonised	Tazocin 4.5g 8hrly + Vancomycin 500mg-3g/24hr + Rifampicin 600mg 12hrly	Meropenem 1g 8hrly + Vancomycin 500mg-3g/24hr + Rifampicin 600mg 12hrly	5-7 days
Aspiration pneumonitis CONSIDER NO antibiotics	Co-amoxiclav 1.2g 8hrly	Meropenem 1g 8hrly	5 days
Intra-abdominal sepsis	Amoxicillin 1g 8hrly + Amikacin 15mg/kg daily +	Meropenem 1g 8hrly + Amikacin 15mg/kg daily +	5-10 days
Necrotising pancreatitis	Meropenem 1g 8hrly		5-10 days
Polytrauma ONLY open / contaminated wounds	Co-amoxiclav 1.2g 8hrly + Amikacin 15mg/kg daily (if shocked) + <i>consider</i> Tetanus prophylaxis	Cefuroxime 1.5g 8hrly + Amikacin 15mg/kg/daily (if shocked) + <i>consider</i> Tetanus prophylaxis	3 days
Polytrauma Including abdominal trauma	Co-amoxiclav 1.2g 8hrly + Amikacin 15mg/kg daily + <i>consider</i> Tetanus prophylaxis	Meropenem 1g 8hrly <i>consider</i> Tetanus prophylaxis	5-10 days
Isolated penetrating cranio-cerebral injury	Cefuroxime 1.5g then 750mg 8hrly + Metronidazole 500mg 8hrly		3-5 days
Skin /Soft tissue Infections	Flucloxacillin 2g 6hrly	Clindamycin 600mg-1.2g 6hrly	5-10 days
Necrotising soft tissue infection <i>See IVlg section below</i>	Meropenem 1-2g 8hrly + Clindamycin 600mg-1.2g 6hrly + Amikacin 15mg/kg daily + Metronidazole 500mg 8hrly		10-14 days

Adult Critical Care Empirical Antimicrobial Therapy Policy - Part 2 of 2

Indication	1st line	If reliable Hx of severe penicillin allergy *	For
Neutropenic infections	Tazocin 4.5g 8hrly + Amikacin 15mg/kg daily	Meropenem 1g 8hrly + Amikacin 15mg/kg daily	Ask
Community acquired meningitis / meningo-encephalitis	Ceftriaxone 4g daily + Aciclovir 10mg/kg 8hrly (if viral encephalitis suspected) + Amoxicillin 2g 4hrly (if immunosuppressed OR >55 years old, to cover Listeria)	Meropenem 2g 8hrly + Aciclovir 10mg/kg 8hrly (if viral encephalitis suspected)	5-7 days
Infections post neurosurgery +/- Intrathecal Device	Intrathecal Vancomycin 10mg od (if no EVD 500mg-3g/24hr IV) + Meropenem 2g 8hrly		Ask
Vascular line associated infection Remove / change lines	ONLY if severely unwell Vancomycin 500mg-3g/24hr + Amikacin 15mg/kg od		5-14 days
Urinary tract sepsis	Co-amoxiclav 1.2g 8hrly + Amikacin 15mg/kg x1	Meropenem 1g 8hrly + Amikacin 15mg/kg x1	5-7 days
Suspected Clostridium difficile associated diarrhoea	PO/NG Vancomycin 125mg 6hrly. If patient has ileus +/- large gastric aspirates (>400mls 4hrly) then GIVE IV metronidazole 500mg 8hrly		10-14 days
Proven Clostridium difficile associated diarrhoea	Proof = "toxin positive" PO/NG fidaxomicin 200mg 12hrly		10 days

Drug specific notes

Beta-lactam antibiotics	<p>Beta-lactam antibiotics exhibit time dependent bacterial killing. Maintaining free beta-lactam levels above the minimum inhibitory concentration (MIC) for a percentage of the dosing interval (40% for carbapenems, 50% for penicillins), will ensure a near maximal bactericidal effect. There is increasing evidence to suggest that administering beta-lactams as a prolonged or continuous infusion produces a drug concentration in excess of the MIC for a longer period than standard intermittent dosing which may achieve improved outcomes in critically ill patients. For more information see [134, 135].</p>
Principals of prescribing	<ul style="list-style-type: none"> • Always prescribe the first dose of antibiotics as a "LOADING DOSE" which will appear on the "Once Only" section of the medication administration record (Paper chart or iCLIP), to reduce any potential delay in achieving therapeutic concentrations. • Administer the first antibiotic dose as a 30min infusion. • Subsequent doses should be prescribed as regular scheduled medication as per dosing protocols. For tds drugs this should be 06:00, 14:00 and 22:00. • In patients with severely reduced renal clearance (eGFR <20ml/min/1.73m²) CONSIDER increasing the dose interval to 12 hourly (10:00 and 22:00) BUT ONLY AFTER 24-36 hours, BUT NOT, if the patient is receiving renal replacement therapy. • DO NOT hold / omit the first continuous infusion dose on the regular scheduled medication chart, following the "LOADING" dose as delaying the first infusion dose can result in sub optimal treatment in the first 24 hours. <p>For the time being we are only applying these principals to Tazocin and Meropenem, NOT Co-amoxiclav, Flucloxacillin, Amoxicillin, Cephalosporins or Ertapenem.</p>
Tazocin	<ul style="list-style-type: none"> • LOADING DOSE 4.5g over 30 mins followed immediately by 4.5g over 8 hours continuously.
Meropenem	<ul style="list-style-type: none"> • LOADING DOSE 1g over 30 mins followed by 1g over 8 hours continuously. <p>In ventriculitis +/- EVD / post neurosurgery</p> <ul style="list-style-type: none"> • LOADING DOSE 2g over 30 mins followed by 1g over 4 hours continuously

Drug specific notes	
Aminoglycosides	<p>Aminoglycoside dosing should be based on body weight (BW). For safety and simplicity USE ideal BW.</p> <ul style="list-style-type: none"> • However, in very underweight patients CONSIDER dose reduction to actual BW • In very overweight patients CONSIDER increasing the dose to adjusted BW, where: $\text{adjusted BW} = \text{ideal BW} + (0.4 \times (\text{actual BW} - \text{ideal BW}))$ • For more information goto http://clincalc.com/aminoglycoside/ • Note - PLEASE use the Urban-Craig formulas for extended dosing - SELECT this in the advanced settings
Amikacin	<ul style="list-style-type: none"> • 1st line, empirical aminoglycoside • 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. • For empirical cover when changing a colonised urinary catheter, GIVE 250mg x1
Gentamicin	<ul style="list-style-type: none"> • Give first dose STAT, 5mg/kg whatever the renal function. (Maximum dose 400mg) • Thereafter, chart for 12 noon administration when blood levels taken @~6am are <1mg/ml.
Vancomycin	<ul style="list-style-type: none"> • Load with 1g ($\leq 65\text{kg}$) or 1.5g ($> 65\text{kg}$) over 2 hours then start continuous infusion at 500mg-3g/24hours depending upon renal function. • Measure blood levels daily and titrate dose to achieve 20-25mg/ml • For full protocol see
Colistin	<ul style="list-style-type: none"> • Load with 9 million units then 3 million units 8 hourly IV • 2 million units nebulised for colonised patients. Use nebulised & systemic therapy for severe pneumonia

Fungal infections – suspected or proven	
Candidiasis	<p><i>FIRST LINE</i></p> <ul style="list-style-type: none"> Fluconazole 800mg day 1 & 2 400mg daily thereafter for 7-10 Load IV. Convert to enteral ASAP. If >130kg increase dose to 1.2g and 600mg respectively <p><i>SECOND LINE</i></p> <ul style="list-style-type: none"> Anidulafungin 200mg day 1 then 100mg daily IV
For all other proven or suspected fungal infections refer to Trust antifungal policy via the intranet & /or seek micro advice:	

Penicillin “allergy”

- Many claims of penicillin “allergy” turn out to be spurious. Approximately 10% of patients report a history of penicillin allergy. However, up to 90% of these individuals are able to tolerate penicillin and are designated as having “penicillin allergy” unnecessarily [136, 137].
- Penicillins are potent bacteriocidal antibiotics and are often the best therapy, hence should be used whenever possible. Use of broad-spectrum antibiotics in patients designated as being “penicillin allergic” is associated with higher costs, increased antibiotic resistance, and may compromise optimal medical care [136, 137].
- “TRUE” penicillin allergies may be clinically sub divided into:
 1. Anaphylaxis: <1 hour following exposure, severe erythema, urticaria or angioedema & / OR hypotension & / OR bronchospasm
 2. Three syndromes of non-immediate reactions with systemic involvement:
 - Drug reaction with eosinophilia and systemic symptoms (DRESS). Onset usually 2–6 weeks after first drug exposure or within 3 days of second exposure.
 - Toxic epidermal necrolysis or Stevens–Johnson syndrome. Onset usually 7–14 days after first drug exposure or within 3 days of second.
 - Acute generalised exanthematous pustulosis (AGEP). Onset usually 3–5 days after first drug exposure.
 3. Non-immediate reactions without systemic involvement: Onset usually 6–10 days after first drug exposure or within 3 days of

second exposure. 2 patterns, widespread red macules or papules (exanthem-like) **AND** Fixed drug eruption (localised inflamed skin). Should be confirmed by skin biopsy.

- If there is a reliable history of anaphylaxis **DO NOT GIVE ANY** beta lactam antibiotic and discuss empirical therapy with the on call microbiologist.
- If there is a reliable history of a serious non-immediate reaction (2. or 3.) to a specific antibiotic then **DO NOT GIVE IT** and discuss empirical therapy with the on call microbiologist.
- If there is an unreliable history suggestive anaphylaxis, perform intradermal testing before administering the first dose of 1st line therapy as follows:

Intradermal test for anaphylactic reactions to penicillins

Inject 0.02mL intradermally of 0.9%NaCl and standard antibiotic solution into the volar surface of the forearm using an orange needle. The margins of the wheals induced by the injections should be marked with a ball point pen. An intradermal test is positive if the average wheal diameter 15 minutes after injection is >2 mm larger than the initial wheal size and also is >2 mm larger than the negative controls. Otherwise, the tests are negative

- [For more information](#) or see [138]
- If the skin test is positive, use the TRUE penicillin allergy column advice.
- If the skin test is negative OR if the history is of ANY OTHER reaction, GIVE first line therapy

IVIg

Is indicated in SOME cases of the following rare conditions.

- Severe invasive group A streptococcal disease (necrotising fasciitis)
- Staphylococcal toxic shock syndrome
- Necrotising (PVL-associated) staphylococcal sepsis / pneumonia / soft-tissue infection

This is a time critical intervention. To be of benefit it should be commenced within 6 hours of disease onset.

- Give 1g/kg daily for 48 hours..

- IT MUST NOT BE USED without ICU AND micro consultant approval.
Contact the on-call pharmacist to obtain supplies.

Tetanus prevention

For full guidance see:

Tetanus-prone wounds include:

- visibly contaminated wounds or burns that require surgical intervention that is delayed for more than six hours
- wounds or burns that show a significant degree of devitalised tissue or a puncture-type injury, particularly where there has been contact with soil or manure (faeces)
- wounds containing foreign bodies
- compound fractures
- wounds or burns in patients who have systemic sepsis.

Immunisation status	Clean wound		Tetanus-prone wound	
	Vaccine	HaTI	Vaccine	HaTI
Fully immunised, i.e. has received a total of five doses of vaccine at appropriate intervals	None required	Only if high risk	None required	Only if high risk
Primary immunisation complete, boosters incomplete but up to date	None required	Only if high risk	None required	Only if high risk
Primary immunisation incomplete or boosters not up to date	Give	One dose	Give	One dose
Not immunised or immunisation status not known or uncertain	Give	One dose	Give	One dose

HaTI = human anti-tetanus immunoglobulin

One dose = 250units IM or 500units IM if >24hrs have elapsed since injury / heavy contamination / contaminated burns

Trust guidelines regarding treatment and prophylaxis

All guidelines can be found on the Trust Intranet - Homepage > Bottom of Central Column > Quicklinks > [Antibiotic prescribing](#)

ODDS & SODS

Indication	Drug and dosing	Notes
Antipyretic (esp. following acute brain injury)	Diclofenac[139]	Load with 0.2mg/kg diluted in 100ml 0.9%NaCl over 30min Continuous infusion (75mg in 50ml 0.9% NaCl) Commence @ 0.04mg/kg/hr & titrate 12 hourly aiming for minimum effective dose. Dose range 0.004-0.08 mg/kg/hr
Prevention of x-ray contrast induced nephrotoxicity	500ml of 1.4% NaHCO ₃	[5]
Eclampsia	Magnesium sulphate	Load 16mmol (4g) over 10-15mins then 4mmol/hr (1g) [140]
Acute pulmonary hypertension	Sildenafil 25mg NG 8 hourly	Increasing dose to 50mg / 100mg is of no proven additional efficacy
Beta blocker and / or calcium channel blocker overdose	Insulin (short acting) Loading 1unit/kg IV Maintenance 1-10units/kg/hr	Give 20ml 20% dextrose then 20-50ml of 20% dextrose per hour. Maintain K ⁺ 4.5-5mmol/l and Mg ²⁺ >1.0mmol/l Boluses up to 10units/kg and infusions up to 22units/kg/hr have been used with good outcomes. Reviewed [141].
Lipophilic drug toxicity	20% Intralipid (available St James' Theatres) 1.5 ml/kg as an initial bolus, followed by 0.25 ml/kg/min for 30-60 minutes Bolus could be repeated 1-2 times for persistent asystole Infusion rate could be increased if the BP declines.	Based ONLY on animal studies and human case reports. Safety does not appear to be an issue BUT efficacy has NOT been clearly established. Hence should be considered as a rescue therapy only, NOT a standard antidote, possibly with the exception of bupivacaine toxicity. Other drugs included verapamil, chlorpromazine, some tricyclic antidepressants and propranolol [142]. http://www.lipidrescue.org/

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IMPORTANT & USEFUL CONTACT NUMBERS

For Hospital bleeps: dial 88 / blp no. / ext no.

For Aircalls (SG): dial #6321 or 07699 119700 or switchboard on 1000

Airway / anaesthetic emergency

bleep 6111 / bleep 7647

Anaesthetic co-ordinator (DFA) bleep 8011

A&E consultant bleep 8021

Blood bank* dial 5471

Cardiology SpR bleep 6002

Cardiothoracic ICU dial 1495 / 1504 / 1507

Cardiothoracic surgical SpR bleep 7370

Code RED

(massive haemorrhage)

dial 6789

ECMO referral (ICU2, St Thomas's) 020 7188 2511

General surgical SpR bleep 7655

Haematology SpR bleep 6068

King's Liver ICU 020 3299 3406

National Burn Bed Bureau (NBBB) 01384 215666

Neuro ICU dial 4195 / 4196

Neurosurgical SpR bleep 7242

Orthopaedic SpR bleep 7439

Radiographer for portables bleep 6284 / 6345 / 6955

Security emergencies

dial 3333

Switchboard dial 1000

Trauma co-ordinator bleep 8091

Transplant co-ordinator pager via 07659 590529
expect call back within 15 mins

Websites

GICU <http://www.gicu.sgul.ac.uk>

username password

Yammer <https://www.yammer.com/stgeorges.nhs.uk> none

Medical devices <http://med> Usual StGH

Burns <http://www.lsebn.nhs.uk/>

Toxbase <http://www.toxbase.org> H1432 KR66C2

