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INTRODUCTION

Welcome to the critical care department of Waikato Hospital.

The purpose of his handbook is to outline a standard approach to care as agreed by the senior medical staff. Intensive care is a twenty-four hour a day, seven days a week service. The majority of our work is unplanned acute work. It is essential that we provide a high standard of care, most often to time critical situations, at any time of the day or night. No individual member of staff can be available to care for any given patient for the entirety of their stay in our unit. Hence we must function as a team, providing consistent standardised care to the community we serve.

Our patient may originate from any specialty and now require intensive care support. Our expertise does not extend to the minutiae of knowledge in each of these specialty areas. We must work with the primary teams and other specialist teams involved in each patient’s care. We strive to remain on good terms with these teams and expect your behaviour to reflect this.

In parallel with our medical practice, every patient receives 24 hour care from our specialist nurses in intensive care. Good relations between our medical and nursing workforce is essential for the optimal function of our department. There are many tasks that are nursing led and there are many nursing guidelines and treatment protocols that also guide unit practice. Please, familiarise yourself with the content of these documents.

In reading and applying this book to your practice you will provide care consistent with the views and beliefs of the consultant staff of this department. Great care has been taken with this guide to accurately reflect our group practice. Of course the science and practice of intensive care medicine is ever changing and this book cannot be updated in real time. This book is a guide, and does not cover every eventuality in intensive care. We are not on site 24 hours a day, but we are always available for consultation and on-site patient review when needed. If you are using this book outside of Waikato intensive care we accept no liability for any factual errors that may be contained within this document.

We hope you enjoy your time in our intensive care unit, and become familiar with our particular approach to medicine. It is hoped that many of the skills and knowledge you learn in this department will be relevant for you in your chosen field of practice.

Jonathan Albrett. MBChB, FANZCA, FCICM.

Editor of the fifth edition.
WHO DO I REPORT TO?

Nurse Manager
Colleen Hartley

Secretary
Di T-D

CCD Charge Nurse Manager
Niki Houghton

Research Nurses
Mary La Pine
John Dunning

Nurse Educator Team
Mark Reynolds
Sue Jackson
John Bell

CCD Charge Technician
Paul Goble

CCD Associate Charge Nurse Managers
Matthew Hughes
Chris Craig
Sarah Walker
Simon Mehari
Christine Carter
Kate Smith
Deborah Trail
Toni-Marie Hancock

CCD Charge Nurse Technicians
Bradon Clark
Alan Hayman

Clinical Resource Nurses
Elaine Farr
Cindy Woodham
Tasha Palliser
Ginny Shelley
Elaine Fernandes
Keith O'Hara
David Aveyard

CCD Registered Nurses

CCD Health Care Assistants
Margaret Clarke
Lizzy Young
Leonie Swan
Andrea Skipper
Agnes Maletino
Luis Rhind
Leanne Ash-Tovatoa

CCD Attendant
Rae Whitehead

Consultants
Annette Forrest
Grant Howard
John Terrance
Nick Barnes
Tom O'Rourke
Robert Martynoga
Jonathon Albrett
Praneesh Jogia
(Supervisor of Training)

CCD Registrars

CCD SHO

Clinical Director
Dr Geoff McCracken

CCD Charge Technician
Paul Goble

CCD Technicians
Bradon Clark
Alan Hayman

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Jonathon Albrett
Praneesh Jogia
(Supervisor of Training)

CCD Registrars

CCD SHO

Clinical Director
Dr Geoff McCracken
ADMINISTRATION

STAFFING

DIRECTOR
Dr Geoff McCracken

CONSULTANT MEDICAL STAFF
Dr Jonathan Albrett
Dr Nicholas Barnes
Dr Annette Forrest
Dr Grant Howard
Dr Ryan Jang
Dr Pranesh Jogia
Dr Stephen Lo
Dr Robert Martynoga
Dr Tom O’Rourke
Dr John Torrance

NURSE MANAGER
Colleen Hartley

CHARGE NURSE
Niki Houghton
Associate Charge Nurse Manager
Christine Carter
Christine Craig
Mathew Hughes
Simon Mehari
Kate Smith
Sarah Walker

**SENIOR TYPIST / UNIT SECRETARY**
Dianne Takiari-Dawson
Reception: Jill Brough, Colette Sutherland, Rachel Rickard

**RESEARCH NURSE**
Jewel Barlow-Armstrong

**RESPIRATORY TECHNICIAN-CHARGE TECHNICAL ADVISOR**
Paul Goble

**NURSE EDUCATOR**
Mark Reynolds
John Bell

**ORGAN DONOR LINK NURSES**
Kate Smith
Sue MacAskill

**UNIT MANAGER – CRITICAL CARE DEPARTMENT**
Colleen Hartley

---

Senior Registrar
Up to 2

---

Advanced vocational trainees
Rostered according to seniority and experience.

---

Registrars
14

Vocational or Rotating registrars (76-9). Staff seconded from other disciplines to gain experience in Intensive Care Medicine. Portfolios and autonomy of practice will be determined by trainee experience and rostering requirements.
TRAINING POSITIONS

The College of Intensive Care Medicine has accredited The Waikato Hospital Intensive Care Unit for training towards the Fellowship in Intensive care. Trainees registered with the Faculty may have up to 24 months of service accredited towards their training.

NON-INTENSIVE CARE TRAINEES

Rotation through the intensive care is made by the following specialty based training programs;

- Physician trainee
- Emergency Medicine trainee
- Anaesthesia trainee
- Rural Hospital Physician trainee

ORIENTATION

At the start of the intensive care run, there is a 2 day formal orientation to the unit for all new registrars. Introductory sessions are conducted by the senior medical staff, the unit manager, senior registrar, head technician and senior nursing staff.

- Topics covered include:
  - General orientation to the unit
  - Data collection and computer programs
  - Transport
  - Invasive and non-invasive ventilation
  - Organ donation
  - Intubation including difficult and failed intubation
  - Cardiac Surgery
  - Neurological/neuro-surgical emergencies

DAILY PROGRAM OVERVIEW

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>08h00</td>
<td>Morning handover</td>
<td>(45 minutes)</td>
</tr>
<tr>
<td>09h00</td>
<td>Consultant led bedside ward round.</td>
<td></td>
</tr>
<tr>
<td>11h30</td>
<td>X-ray meeting (10h30 at the weekend)</td>
<td>30 minutes</td>
</tr>
<tr>
<td>15h45</td>
<td>Afternoon ward round and HDU review.</td>
<td>(30min-1hr)</td>
</tr>
<tr>
<td>20h00</td>
<td>Evening hand over and HDU review</td>
<td></td>
</tr>
</tbody>
</table>
In addition, attempts will be made to allow those registrars who feel that they require further experience with airway management and intubation to attend anaesthetic lists in theatre early in the run. On these days, the duty anaesthetist should be approached and asked if there are suitable lists available.

All time other than that allocated above should involve patient review, not only in response to request by nursing staff, but also in the interests of optimising patient care and progress.

**WEEKLY MEETINGS / EDUCATION**

---

**TRAUMA FORUM**

Day: Thursday  
Time: 0730 (before handover)  
Venue: Auditorium

---

**ICU MICROBIOLOGY MEETING**

Day: Thursday  
Time: 08h00 (before handover)  
Venue: ICU meeting room

Registrar presentation (10 min) and brief discussion of micro issues in the unit.

---

**AUDIT**

Registrars are expected to do an audit project in the six months they are in ICU. This may be presented at the Regional Meeting in the last week of the run.

---

**HOSPITAL GRAND ROUNDS (60MIN)**

Day: Thursday  
Time: 12h30  
Venue: Hospital auditorium

---

**TEACHING SESSIONS AND ICU MORTALITY MEETING / JOURNAL CLUB**

Day: Tuesday afternoons  
Time: 1300hrs  
Venue: ICU meeting room
ADDITIONAL TEACHING

Other teaching times may be arranged. In particular, every endeavour should be made to allow anaesthesia, medical and other trainees to attend their set sessions.

MAIL

You will have a named in-tray in the Workroom. An internal/external mailbox is outside reception. NZ Post box is available at the exit of B5 of the car park building.

MEAL VOUCHERS

You are entitled to x2 for a Long day, x1 for a Short day, and x1 for a Night shift.

NIGHT REGISTRAR SLEEP-ROOM

A sleep room is provided (in the HDU).

ROSTERS

At any one time there are 14 registrars on the ICU roster. Suggestions as to how the roster can be improved within the limits of the current Award/Employment Contract are always welcome. You will be paid fortnightly on a Thursday.

LEAVE

Annual / conference / course / exams

Please let us know of annual leave requests well in advance. These can usually be arranged at times requested, providing enough notice is given, e.g. several months.

These will normally be covered by the Reliever Registrar.

Sick leave

If you are sick for a rostered shift, you must ring:

Mon-Fri: 0700-1600 Operations Manager

After hours: Duty Intensivist

REGISTRAR ROSTER STRUCTURE

A handover is held at 0800 a.m. every day and is the time when the main transfer of information is made between registrars and seniors, and also when continuing policies are discussed.

It is necessary to be prompt at the 0800 a.m. handover and the registrar should be there before 0800 a.m. The main time is taken up in the 'paper handover' and the ward round moving between patients is then done
expeditiously. It is expected that the registrar will return to the patients and examine them in a full way later on.

**0800 Night Registrar**

Formal handover in the Workroom. X-rays / Lab results are put up on the screen by short day registrar

**LONG DAY REGISTRAR MAIN UNIT**

On duty from 0800 – 2000 hrs

Carries the Main Unit mobile phone

If the ward round is long, it is useful for the registrar to speak to the nurse in charge of each end once the formal round is finished (prior to morning tea and the hospital round) in case there are immediate issues of concern.

**REGISTRAR CARDIAC UNIT**

On duty from 0800 – 1700 and 1530 - 2300

Carries the Cardiac Unit mobile phone

Carries Cardiac Arrest 99-777 Pager

**SHORT DAY REGISTRAR**

On duty from 0800-1600 hrs

Carries Cardiac Arrest 99-777 Pager

Responsible for all referrals including ward assessments. Responsible for all inter-hospital transports 0800-1600hrs

**SHORT DAY REGISTRAR**

Available to review HDU patients and assist Long Day registrars as work load dictates. Available for insertion of line requests.

Responsible for ensuring all patients enrolled in research are consented.
CLINICAL DUTIES IN THE INTENSIVE CARE UNIT

INSTRUCTIONS TO REGISTRARS

Notify and discuss with the duty intensivist any concerns or change in a patient’s condition. Advice should be liberally sought.

It is expected that the registrars will function as a team and support each other and are expected to have a good overview of all patients in the Intensive Care Units.

The usual compliment of junior staff will include:

- Main Unit Registrar
- Cardiac Unit Registrar
- Referrals/Transport Registrar
- Advanced Trainee Registrar/Fellow 8am-4pm weekdays

Resident Staff will always shoulder a major part of the burden of continuity. Continuity is central to quality patient care and this expectation is not diminished with a decrease in working hours. The consultant staff also play a pivotal role in this process, but because of the prolonged periods of continuous call that they are expected to cover, they are not resident in the hospital and are therefore not continually in the unit. The responsibility for maintaining continuity and for effective communication both with other unit staff and with outside teams rests largely with the registrars. Effective communication is a basic medico-legal requirement.

DAILY MANAGEMENT ISSUES

The daily handover ward round at 08h00 is attended by the night Registrars, the incoming day staff, the Duty Intensivist and representatives of the nursing staff (Associate CNM and team leader).

The night Registrar will prepare a report and present in a concise and professional manner on the handover round.

Important decisions regarding patient discharge and specialist investigations are made at this meeting and it is important that junior staff have a good understanding of the patient status, including:

- Patient details and demographics
- Day of admission
- Diagnosis and major problems
- Relevant pre-morbid problems
- Progress and significant events
- Important results
Plan for the next 24 hours

Most of the above will appear on your daily “Problem List Report”.

This is followed by a consultant led bedside round.

**FAST HUG**

FAST HUG is a mnemonic used in the CCD as a checklist that highlights key aspects in the general care of the critically ill patient. It helps in preparation for patient rounds, helps to prevent and identify medication errors, and promote patient safety.

FAST HUG highlights the importance of the following clinical practices:

- Feeding
- Analgesia
- Sedation
- Thromboembolic prophylaxis
- Head of bed elevation
- Stress ulcer prophylaxis
- Glycaemic control

**FEEDING (NUTRITIONAL MANAGEMENT)**

Ensuring adequate nutritional intake in critically ill patients is essential for a number of reasons such as ensuring optimal wound healing and maintaining gastric motility. Two methods are available for ensuring adequate nutritional intake: these are enteral or parental nutrition. Enteral feeding options include naso/orogastric tubes (NG/OG) or Percutaneous endoscopic gastrostomy tubes (PEG) alternatively parental nutrition can be delivered via central venous access using Total Parental Nutrition solutions (TPN).

Nutritional assessment should be completed for all critically ill patients as soon as possible (preferably within the first 24-48 hours or once the patient is stabilised) and ongoing reassessment of nutritional demands should be made throughout the patient’s hospitalisation. The Adult Enteral Feeding Algorithm is a guide to commencing or restarting enteral feeding and can be found in the Enteral Feeding Procedure.

**PREVENTION OF ASPIRATION**

Preventing aspiration during enteral feeding is an important aspect of nurses the critically ill patient. A typical bundle of care would include:

- Maintaining head of bed (HOB) elevation at an angle of 30-45° (head injured patients should have HOB elevated to 30° as tolerated)
• Confirming gastric tube placement prior to starting or recommencing feeds by:
  • Injecting 10-20mL of air down the tube and auscultating the epigastric area
  • Using litmus paper
  • Radiography is the Gold Standard in checking gastric tube placement prior to commencing enteral feeding

ANALGESIA

No patient should be required to endure excessive pain.

If the patient is awake and alert consider in step-wise fashion the following:

• Regular paracetamol
• Codeine preparations (with or without paracetamol)
• Non-steroidal anti-inflammatory drugs unless contra-indicated. (ie bleeding diathesis, gastric ulcer / erosion, renal dysfunction)
• Patient controlled analgesia (PCA-see later section)

Where the patient is unable to co-ordinate the PCA mechanism, bolus analgesia should be administered by the nursing staff, titrated to the patients request for pain relief

In exceptional circumstances an infusion of narcotic may be appropriate.

SEDATION

Sedation in critically ill patients is principally used to control agitation, allow mechanical ventilation free of dysynchrony, and provide analgesia. These goals are quite distinct from the goals of anaesthesia, which are to provide short-term analgesia, sleep, and muscle relaxation to facilitate surgery. The point of distinction between intensive care sedation and anaesthesia is the primary goal of intensive care sedation is to prevent agitation; in anaesthesia it is to produce sedation. Specifically, different goals of treatment necessitate different methods of titration.

Patient sedation should be goal-directed. Generally sedation should fall into one of the categories listed below:

• Patient and nursing safety in the event of patient agitation: To enable effective care to be delivered and prevent occurrence of accidental extubation or removal of vascular access catheters
• Treat unacceptable treatment-related distress
• Where agitation or restlessness compromises patient haemodynamics
• To facilitate ventilation or minimise patient-ventilator dys-synchrony
- Control intra-cranial pressure
- Reduce metabolic rate (oxygen consumption) and sympathetic drive

In mild cases adequate sedation can be administered by regular and/or "prn" administration of one or more of:

- Haloperidol 2.5-20 mg 6 hrly (PO / NG / IVI) ?2.5-10 mg prn IVI
- Clonidine 50-200 µg 6-8hrly PO ? 15-30 µg prn IVI (max 600 µg / day)
- Diazepam 2.5-10 mg Brly (PO / NG / IVI) ? 5-10 mg prn IVI

Where an infusion is deemed necessary, this should be goal directed with a sedation end-point specified according to a designated sedation score. (See appendix – Sedation-Agitation score).

IN THE WAIKATO HOSPITAL ICU, PROPOFOL BOLUS. OPIOID OR INFUSION OF SHORT ACTING OPIOID, ARE THE FIRST LINE SEDATIVE AGENTS WHERE AN INFUSION IS REQUIRED.

THE MAXIMUM DOSE OF PROPOFOL IS 20MLS/HR IN WAIKATO ICU.

<table>
<thead>
<tr>
<th>COMMONLY ENCOUNTERED SEDATIVE AGENTS AND OPIOIDS</th>
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<tbody>
<tr>
<td><strong>Propofol</strong></td>
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<tr>
<td><strong>Remifentanil</strong></td>
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<td><strong>Midazolam</strong></td>
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<td><strong>Morphine</strong></td>
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<td><strong>Fentanyl</strong></td>
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<tr>
<td>Drug</td>
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</tr>
<tr>
<td>Diazepam</td>
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<tr>
<td>Dexmedetomidine</td>
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<tr>
<td>Haloperidol</td>
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</tbody>
</table>

Primarily sedation is given to reduce agitation, which most frequently results from a patient’s response to pain and other discomforts. This needs to be weighed against the adverse effects associated with over-sedating a patient:

- Respiratory depression
- Prolonged ventilation and associated risk of nosocomial infection
- Eventual emergence phenomena with sympathetic overdrive, delirium and withdrawal
- Hypotension
- Gastroparesis
- Prolonged stay with unnecessary use of resource, and increased risk of complications

There are many other reasons why a patient may become agitated which should be considered and are summarized below.
Thromboembolism prophylaxis is essential in critically ill patients due to the increased risk of venous thromboembolism (VTE) experienced in this patient group. This increased risk occurs for a number of reasons including the patients pre-existing medical conditions (e.g. obesity, family history of blood clots), their admitting diagnoses (especially sepsis and trauma), a history of recent surgery, interventions in the critical care environment (such as central venous catheterization, invasive tests and procedures) and finally drugs potentiating immobility (e.g. sedatives and paralytics).

**TREATMENT AND MANAGEMENT**

Due to the life-threatening nature of deep vein thrombosis (DVT) and pulmonary embolism (PE), thromboembolism prophylaxis should be implemented as soon as possible within the first 24 hours following admission. The treatment options currently used in the CCD include:

- Thigh length elasticised compression stockings (ECS)
- Sequential compression devices (SCD)
- Pharmacological agents such as:
  - Clexane
  - Fragmin
  - Low dose Heparin
- Placement of inferior vena cava filters (IVC filter)
- Mobilisation as able

**HEAD OF BED ELEVATION**

Maintaining head of bed (HOB) elevation at an angle of 30-45° (head injured patients should have HOB elevated to 30° as tolerated). Used to reduce the incidence of aspiration.

**ULCER PROPHYLAXIS**

**PROPHYLAXIS OF GASTRIC “STRESS ULCERATION”**

Cardiac Unit: All patients will receive post-operative omeprazole or equivalent agent

General Unit: Not indicated in enterally fed patients, even at low volumes, unless the patient is known to have pre-existing or subsequently (in-hospital) proven peptic ulceration.

Consider use of a prophylactic agent (omeprazole 40mg IV 12 hrly or ranitidine 50 mg IV 8 hrly) if patient is not enterally fed and:
- Pre-existing or intercurrent coagulopathy
- Mechanical ventilation > 48hrs

## USE OF GASTRO-INTESTINAL PRO-KINETIC AGENTS

Gastric stasis, colonic and small intestinal ileus are common management problems in the intensive care unit. It may be necessary to explore jejunal feeding tube placement and / or the use of prokinetic agents to facilitate enteral feeding (see algorithm on enteral feeding). Metoclopramide 10mg Q6H is used as first line followed by erythromycin 100mg Q6H.

### CONTRA-INDICATIONS

Erythromycin, sometimes used in this setting, interacts significantly with other drugs metabolised by the Cytochrome P450 enzyme system, with potentially lethal side effects (eg. arrhythmia).

Potential drug interactions must be reviewed prior to commencing erythromycin.

## ACTIVE GI BLEEDING

### DIAGNOSIS

- Revealed blood: Nasogastric blood, haematemesis, malaena
- A fall in systolic blood pressure > 20 mmHg
- Drop in Hb > 20 g / l in 24 hours, or requiring transfusion of blood

### MANAGEMENT

- ABC / resuscitate
- Correct coagulopathy / cease heparin
- Omeprazole 40-80 mg IVI 12-8 hrly, consider oral / nasogastric once stable. Gastroenterology may request an omeprazole infusion 80mg in 80ml at 8mg/hr post endoscopy
- Endoscopy and sclerotherapy / colonoscopy / angiography and attempted vessel embolism if clinically appropriate

## GLYCAEMIC CONTROL

Hyperglycaemia in critically ill patients is managed by following one of two insulin protocols depending on which specialist area the patient is admitted to.

## INTENSIVE ACTRAPID INSULIN THERAPY

The Intensive Actrapid Insulin Therapy protocol is based on an analysis of the results of both the Leuven study (Van den Berghe et al., 2001) and the NICE-SUGAR study (NICE-SUGAR Study Investigators, 2009) which showed that high and low levels of blood glucose are harmful in the critically ill population. Recently it has been modified to maintain less tight control. This is based on recent recommendations from the American...
College of Physicians Best Practice Advice. The protocol now aims to maintain a blood sugar level of between 8-11mmol/L.

The HDU follows the Waikato DHB wide ‘Intravenous Insulin Infusion Protocol for Adults’ in cases where the patient is a known or suspected diabetic. This protocol can be found on the Intranet (Clinical Guidelines → Diabetes → Intravenous Insulin Infusion - Adults). There is separate protocol recently developed for surgical patients called the perioperative insulin protocol (Clinical Guidelines → Diabetes → Perioperative management of diabetes mellitus)

REFERENCE:


MEDICATION TIME OUT

INTRODUCTION

Medication Time Out was a project undertaken by Annette Forrest (ICU Consultant) in 2012. The results of the study found four key areas that needed to be implemented in conjunction with the project to ensure its success:

1. An associated education package
2. Examples of medication incidents related to clinical practice
3. An associated evaluation package to check understanding of the process
4. Senior medical and nursing staff buy-in

It has therefore been decided that the Medication Time Out process will be reviewed and narrowed down to focus on eight key indicators:

1. Correct patient sticker or patients name and NHl if written
2. Allergy sheet has been filled in and perused
3. Prescription handwriting is legible
4. Medication is specified appropriately (no trade names)
5. Appropriate mass units are used (e.g. g, mg, units, micrograms)
6. Drug route of administration appropriate
7. Frequency correctly specified

8. Authorised abbreviations only – but particularly avoiding “high risk” abbreviations (see Medication and Allergies – Front Sheet)

While the Medication Time Out is not currently part of nursing practice the review and reimplementation of the process will be undertaken in 2014 when it will become ingrained into practice.

**REFERRALS**

The referrals registrar is expected to accept and review consults and attend trauma calls and cardiac arrest calls. They will also act as the flight retrieval registrar.

*All referrals should be discussed with the duty intensivist and documented in the patients clinical notes.*

Those patients not taken to the Intensive Care Unit should have their details recorded in the “Refusals Folder” in the main unit.

In general ICU staff should discourage other medical staff from letting them know about patients that are not being referred to the ICU. Where there is any doubt, the ICU registrar should treat the interaction as a consultation with all that entails.

When asked by a team to review a patient, registrars are required to obtain a full history from the patient and the patient notes, to perform a comprehensive examination of the patient and to formulate a differential diagnosis. They should then have an outline of a suggested investigation and treatment plan. The parent team should be consulted concerning their expectations for the patient, in particular what they are asking for from the ICU team. This information should be clearly documented in the patient record. This information is critical when presenting a patient at handover and to consultant staff as it is not possible to make quality decisions based on incomplete information.

It is important that there is a complete transfer of information at the handover between shifts. This will be facilitated by:

- Comprehensive admission note. Proforma sheets are available for cardiac and trauma admissions
- Completion of a standardised daily update note
- Daily review of all clinical laboratory tests, microbiology and radiological tests
- An update of the computerised problem list or daily progress notes by the night registrar. This will contain details of the presentation, the provisional diagnosis, investigations, consults and opinions and unresolved issues that require follow up
- Registrars should briefly familiarise themselves with the patients before the formal ward rounds
- When leaving the unit, registrars must inform their registrar colleague if applicable. The ICU floor must never be left unattended without proper reason, and the knowledge of the nursing team leader
PATIENT ADMISSION

PRIMARY PATIENT SURVEY

A: Ensure patient protecting airway / GCS / cognition
B: Breathing pattern acceptable, Pulse Oximetry acceptable
C: Patient cardiovascularly stable, venous access acceptable
D: Patient neurological condition

Obtain hand over information from the referring doctor

SECONDARY SURVEY

Examine patient thoroughly, then:

Notify Duty Intensivist if this has not already been done.

Document essential orders:

Ventilation
Sedation, analgesia, drugs and infusions
Fluid therapy
Discuss management with nursing staff and team: Everyone must be aware of the plan!
Basic monitoring and procedures:

ECG
Invasive / non-invasive monitoring
Urinary catheter / NG tube
Basic Investigations (usually a full blood count, coagulation profile, ICU specific electrolyte profile)
Advanced Investigations; CT, MRI, Angiogram as indicated
Case note documentation (see below)
Inform and counsel relatives in general terms

REGISTRAR DOCUMENTATION

Documentation is considered very important. Notes are a legal document that must be filled out daily.

As you are writing notes, please bear in mind that both the coroner and the patient can access these at a later date.
Registrars are responsible for documenting an admission note for all patients and a daily entry into the clinical notes as well as:

**ADMISSION NOTE**

Where a pro forma sheet exists this should be used (Cardiac and Trauma), otherwise include:

- Date / time
- Name of admitting officer
- Reason for admission
- Standard medical history including current medications
- Thorough examination findings
- Results of important investigations
- Assessment / severity / differential diagnosis
- Management plan
- Document notification of parent team and Duty Intensivist
- Discharge summary
- Death certificate
- Database form collection

Parent teams should be encouraged to write a short note at least.

If a patient deceases in ICU, transfers to another hospital or is discharged home, a discharge letter to the GP and referring hospital needs to be written as soon as possible. This can be done either from either the Clinical Results Viewer or dictated. The secretary will upload the dictated letter to the Clinical Results Viewer.

A copy of this letter is also used for our monthly ICU M&M meeting.

**DAILY ENTRY IN CLINICAL NOTES**

Ensure each page is dated and labelled with the patient’s name and hospital number. An ICU Template sheet is used

- Date / time / name of ICU Staff Specialist conducting the round
- A: Mental state, GCS, airway
- B: Ventilation, saturation (or PaO2), chest findings
- C: Pulse / BP / peripheral perfusion / Precordial exam
- Abdominal examination and description of feeding mode
- Peripheries
• Assessment or Impression
• Plan

Fill out the Ventilator orders sheet.

Immediately after completion, notes must be filed in the clinical record – This is the Registrars Responsibility.

**ADDITIONAL NOTATION MUST BE MADE IN THE NOTES WHEN**

• Invasive procedures are undertaken
• Important management decisions are made
• Significant interaction is made with the patients family

**ICU PROBLEM LIST FORMULATION**

We currently have a computerized handover sheet, updated daily to assist patient handovers. This must be updated and care must be taken not to leave them lying around the unit or hospital!

The Night Registrar is not on duty to simply fight fires until the next day dawns, but actually is the most important continuity pivot for the ICU. Night registrars must feel free to insist on an adequate handover from day staff including any tasks not yet accomplished.

**DATA COLLECTION FORMS**

**A) ICU DATA SHEETS**

ICU contributes to an ANZICS (Australian & NZ Intensive Care Society) database system. This enables us to benchmark ourselves against other Australasian units. Every patient admitted to ICU must have a datasheet completed. This is filled out at the time of discharge by the discharging registrar and handed to our receptionist.

The forms are checked by Dr Barnes and entered onto the ANZICS database by our receptionist.

**B) TRANSPORT OF THE CRITICALLY ILL**

These are completed on return from transports by the transport nurse, checked by Dr Torrance and entered onto our transport database by our receptionist.

Remember: Data is very important! What we put in is what we get out. Do the form filling conscientiously. Future staff will be writing papers on YOUR data and you will be writing papers on previous registrar data.
CLINICAL DUTIES OUTSIDE THE INTENSIVE CARE UNIT

CARDIAC ARREST CALLS

INDICATIONS

Cardiac arrest calls may be called for the following:

- In-hospital cardiac arrest or any severe clinical deterioration
- Collapse of unknown origin in the hospital environs
- Out of hospital arrest arriving in the emergency department

ARREST TEAM MEMBERS

- ICU Registrar
- Cardiology, Cardio-respiratory or Medical Registrar according to time of day
- Nurse practitioners: CCU nurse, Clinical resource nurses, ward staff

CPR (CARDIO PULMONARY RESUSCITATION)

The Waikato Hospital encourages the use of the International Consensus on Resuscitation guidelines for cardiopulmonary resuscitation. The ICU Registrar is responsible for securing the airway and establishing effective ventilation, whilst the Medical Registrar should concern themselves with cardiac and general aspects. It would be expected however that directing advanced life support be the responsibility of the more senior Registrar present.

Where CPR has been “successful” but further active treatment may not be in the interests of the patient, the admitting medical officer and ICU specialist must be consulted prior to withdrawing care.

All involvement in an arrest call must be documented in the patient case notes.

TRAUMA CALL

After receiving details from ambulance personnel patients will be designated according to ACEM triage category. The Emergency Department will initiate the call to the Hospital Operator. A 99-777 call is placed on the emergency pager.

The Intensive care department no longer response to all trauma calls. Therefore we will receive notice via the arrest pager but will only attend when asked to by the emergency department.
ROLE OF THE ICU REGISTRAR AT THE TRAUMA CALL:

The Emergency physicians are primarily responsible for the resuscitation and stabilisation of trauma patients including management of the airway.

However they will contact the ICU early if it is anticipated the patient will require ICU therapies.

The following applies to when we are asked to be involved and for multiple traumas:

- Primarily as a back-up for acute life threatening situations
- ICU staff manages the patients airway, providing they are adequately experienced to do so
- Secure the airway
- Establish ventilation
- Assist with vascular access

ICU Registrars usually escort patients to the ICU from the emergency department. They may also be required to transport the patient to radiology if the patient is destined for ICU thereafter.

Always document your involvement in the case notes.

Keep ICU senior medical and nursing staff up to date with patient progress if ICU admission is likely

REFERENCE:


TRAUMA CALL PROCEDURE

The trauma call usually precedes the patient arrival in ED. It is worthwhile to prepare everything that might be needed (i.e. calculate and draw up drugs, check intubation equipment and communicate with the rest of the team). It is particularly important to identify which nurse is helping with the airway.

On arrival in the E.D. the patient should be assessed according to ATLS Guidelines (ABCD...).

TRAUMA TEAM MEMBERS:

The team assembled will vary according to the number of cases expected, as below.

TRAUMA I (1 CASE)

- Emergency Department Senior (Trauma Leader)
- ICU Registrar
- Emergency Department Registrar (initiates resuscitation, assists with the management of the patient and makes sure all staff that have been called, attend)
- Surgical Registrar-(primary and secondary survey)

**TRAUMA II (2 CASES SIMULTANEOUSLY):**

- Emergency Department Senior (Trauma Leader)
- ICU Registrar (Team leader in absence of ED/ICU Senior)
- Emergency Department Registrar
- Surgical Registrar
- Anaesthetic Registrar

The ICU Senior is called in to lead the second trauma case if both cases ACEM Triage 1 or 2 or if ICU Registrar or Emergency Physician request.

**TRAUMA III (3 CASES):**

Call as for Trauma II with the addition of the Specialist Anaesthetist on call, Surgeon on call, and the orthopaedic registrar

**PRIMARY SURVEY**

- Airway: and total spine control. Do not forget to look in the mouth. Do not neglect the C-spine
- Breathing
- Circulation: and haemorrhage control. Resuscitation without controlling bleeding control is at best a temporary measure. Techniques such as FAST ((Focused Abdominal Sonography for Trauma) or DPL may be required before secondary survey
- Disability: brief neurological evaluation
- Exposure: completely undress the patient
- Adjuncts to primary survey include: Cervical Spine, Chest X-ray, and Pelvic X-ray.

**SECONDARY SURVEY**

Cover in the following order:

- Head and scalp/ maxillofacial
- Cervical Spine and Neck
- Chest
• Abdomen and Pelvis
• Back and Perineum
• Extremities
• Neurology

AIRWAY MANAGEMENT IN THE EMERGENCY DEPARTMENT

IF THERE IS AN ANTICIPATED OR ACTUAL DIFFICULT INTUBATION IN THE EMERGENCY DEPARTMENT THE ANAESTHETIC DEPARTMENT VIA THE DUTY ANAESTHETIST IS THE FIRST PORT OF CALL.

If the anaesthetic staff are all busy it is then reasonable for the ICU staff to help if able.

INTRA-HOSPITAL PATIENT TRANSPORT

No patient may be transported from the unit without the direction of the Duty Intensivist.

Medical escort is the rule. In a minority of circumstances a nurse escort may be sufficient, providing it is acceptable to the Duty Intensivist and the Nursing leader.

Registrars should ask the senior registrar to accompany them on their first in-hospital transport if they are available. It may not be appropriate for all registrars to undertake prolonged transport, or transport to unfamiliar areas (eg MRI). Always ask the Duty Intensivist if you are unsure.

Prior to embarking on an escort all equipment, oxygen supply and emergency drugs must be checked.

All problems encountered on the escort must be recorded in the notes, and an incident form completed if appropriate.

If a test is deemed urgent the medical escort should endeavour to get an informal report in the notes failing which they should request formal review and notification to the unit as soon as possible. For radiology, reports during the day should appear on the clinical results viewer within an hour.

There is a unit video outlining the process for intra-hospital transport of the ventilated patient.

REQUEST FOR INSERTION OF CENTRAL VENOUS ACCESS

Intensive Care Staff may be approached to facilitate central venous access in a patient not residing in intensive care. Most elective lines are now placed by the renal service but we may at times be asked to assist.

Request for CVC lines should come from Registrar level or above.

The person performing the line insertion is responsible for gaining informed consent.

CVC’s are generally elective procedures and do not take priority over ICU duties.
The indication for insertion must be reviewed, and alternatives discussed if appropriate.

The safety of the procedure must be reviewed, in particular the determinants of haemostasis (see relevant section on procedures).

The patient is currently brought to the ICU for insertion when possible.

Ultrasound is available within the unit for those familiar with its use.

**HIGH DEPENDENCY UNIT**

HDU patients are those who do not require services specific to an ICU. Specifically, the following modalities will not be available in HDU:

- endotracheal intubation
- intermittent positive pressure ventilation or CPAP by endotracheal or tracheostomy tube
- continuous renal replacement therapies (CRRT)
- pulmonary artery catheters
- inotropic support
- vasopressor (except for those patients with epidurals)
- BiPAP (except for those patients with longstanding COPD or CHF)

**HDU 10 COMMANDMENTS**

1. The HDU is an “open” unit. This means patients admitted to HDU are under the care of a named consultant who, along with his/her RMO team, coordinates the care of that patient, either alone or in consultation with other teams. Transfer to HDU is not a way of getting another team to take-over care

2. No patient in the HDU is being cared for by the ICU staff unless this is explicitly negotiated – this differs from many hospitals

3. Patients must be pre-booked whenever feasible using a written HDU Booking form. Unfortunately this still does not guarantee bed availability on the day

4. Management is meant to be performed predominantly through the leadership of the team registrar between consultant ward rounds

5. Hospital policy mandates notification of the responsible consultant (or the on-call consultant for that specialty if the former is unavailable). See protocol – Delegated responsibilities when to call the Consultant

6. With the exception of an anaesthetist booking a patient for postoperative care, a request for a bed in HDU should come from the registrar of the team intending to care for the patient in HDU, or as second best a House officer acting under explicit instruction of a more senior team member. Regardless, the registrar takes responsibility for the appropriateness of transfer
7. Anyone requesting an HDU bed should have a clear idea of the expected benefits of admission, have outlined (preferably in writing) a clear therapeutic plan, and be expecting to be called and respond in a timely fashion if these goals are not met.

8. Although teams are often requested to “clear” a patient for the ward, the reality is that the patient may need to be discharged regardless of a wish for that patient to remain in HDU, depending on available resource. This is the concept of triage and exists in every aspect of medical practice to maximise benefit in groups of patients.

9. Use of non-invasive ventilation and vasopressor/inotropes is tightly regulated. See High Dependency Admission and Discharge Policy.

10. Patients should be seen daily (prior to 10am is preferable) including the weekend, a weekend plan to be used when patients are expected to remain there over the weekend, and be handed over to on-call registrar staff. See High Dependency Admission and Discharge Policy. These factors are regularly audited.
PATIENT RETRIEVAL

INTRODUCTION

The Waikato ICU is frequently involved in inter-hospital patient transport within (and occasionally beyond) the North Island. Note that our formal involvement is limited to transport between public hospitals ONLY. The exception is attendance at unexpected emergencies while en route to another site. Retrievals may be undertaken from Private Hospitals in Hamilton.

The Waikato ICU Transport Service is a consultative and consultant lead service concerned with the safe transport of seriously and critically ill patients beyond the immediate newborn period, when we are able to and deem it appropriate. ICU Registrars with Transport Team Nurses perform the vast majority of these transports under the supervision of the appropriate Intensivist who is responsible for the Registrar’s performance. In every case, the appropriate Intensivist must be notified of the intention to perform a particular inter-hospital transport and authorise it.

It must also be stressed that in every case, another team must be expecting to assess and where necessary accept care of a patient transferred by our team, whether the patient is being admitted to our ICU or not. Exception: “single doctor on duty hospitals” within WDHB.

Effective and explicit communication is the principle that underlies inter-hospital patient transfer and every attempt must be made to foster this by team members.

SPECIFIC RETRIEVAL SITUATIONS

BALLOON PUMP

Balloon pump - under emergency situations, new guideline 2016.

CHILDREN

See table below and transport compendium. Below 5kg, a Hamilton T1 is the most suitable ventilator. Beyond 5kg, the Hamilton T1 is suitable. Above 10kg, the Parapac or Hamilton T1 is suitable. Transport of critically ill children by our team is a rare event indeed, and yet is required from time to time. Whenever possible and appropriate, children leaving our ICU bound for an Auckland hospital should be transported by Starship Hospital Retrieval Services if not destined for PICU at Starship. Urgency for definitive care may preclude this however.

DYSBARIC ILLNESS

If mild, transport should happen by standard escort and road ambulance. If severe, critical care transport as close to sea level as possible.

DIVERSION TO ROADSIDE

Perform in usual doctor “Good Samaritan” role.
ED DESTINATION

Make sure ED physician on duty is aware the patient is coming to ED.

HDU DESTINATION

Check bed availability with HDU Co-ordinator. If no bed available, inform accepting team.

OBSTETRIC PATIENTS

Strict control of transport of these patients is required. Patients notified to us from the Obstetric Unit should already have an appropriate decision made by duty Obstetrician as to what type of obstetric escort is necessary. If not, this should be encouraged as a matter of urgency. If a referring clinician rings our ICU initially, we will take details and call Consultant Obstetrician on duty who will be asked to specify the obstetric escort. Callers notifying of patients who are not in a public hospital should be advised to dial 111 and ask for an ambulance.

RAPID RESPONSE TURNOU T

Used whenever extreme emergency- notify all concerned to facilitate rapid transport.

RELATIVELY WELL PATIENTS

After verification that the patient can be transported without a critical care transport team (achieved by assessing patient and liaising with duty Intensivist), ward staff are advised to explore other options with Duty Manager.

Trauma within 48 hours injury- should be taken through the ED. For ICU patients beyond this period, transport direct to ICU is preferable.

WEIGHT OVER 150KG

Fixed wing or road ambulance necessary for at least return trip.

When suitable transport or team unavailable: responsibility lies with medical staff attending patient to continue exploring other ways to transport patient. This may involve liaising with transport services in other cities.

DOCUMENTATION

A transport summary form is to be completed for every patient. There is space for clinical details, charting drugs and fluids, regular recordings and to list any problems encountered.

On delivery of the patient the record is copied and the copy left in the patient’s clinical record. The original is returned to the coordinator’s office – information is entered on the database, the job reviewed and then the summary filed.
## SUMMARY OF TRANSPORTING SERVICES

<table>
<thead>
<tr>
<th>Referral Source</th>
<th>Trauma a In ED</th>
<th>All admitted and non-trauma Patients</th>
<th>All Interhospital Transfers</th>
<th>Flight Time (mins)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taupo</td>
<td>ED A Zero</td>
<td>ICU Transport Team</td>
<td></td>
<td>35</td>
</tr>
<tr>
<td>Tokoroa</td>
<td></td>
<td></td>
<td></td>
<td>30</td>
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<tr>
<td>Taumarunui</td>
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<td>35</td>
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<tr>
<td>Thames</td>
<td></td>
<td></td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>Te Kuiti</td>
<td></td>
<td>ED A Zero</td>
<td></td>
<td>30</td>
</tr>
<tr>
<td>All other Midland Hospitals</td>
<td></td>
<td>ICU Transport Team</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tauranga</td>
<td></td>
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<td></td>
<td>40</td>
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<tr>
<td>Rotorua</td>
<td></td>
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<td>35</td>
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<tr>
<td>Gisborne</td>
<td></td>
<td></td>
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<td>45</td>
</tr>
<tr>
<td>New Plymouth</td>
<td></td>
<td></td>
<td></td>
<td>50</td>
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<tr>
<td>Neonates and children &lt;5kg</td>
<td></td>
<td>Neonatal Transport Team</td>
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<td></td>
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<tr>
<td>Obstetric Referrals from hospital</td>
<td></td>
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<tr>
<td>Interhospital transfers but not to Waikato</td>
<td></td>
<td>ICU Transport Team</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Roadside Retrievals</td>
<td></td>
<td>ED A Zero</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
SUMMARY OF THE TRANSPORT PROCESS

Initial call to ICU by referring hospital

Clinical details obtained by Registrar/Specialist

Acceptance of transport by ICU Specialist

Pilot contacted by ICU Helicopter 06 100 663
Fixed wing 026 107 380
Ambulance control 8055

ICU transport doctor notified

ICU transport nurse notified

Duty Manager notified

ADDITIONAL MATERIAL:

The transport compendium, held in a red folder in the bay of ICU2 is the most comprehensive reference on our retrieval services.

MRI – PRACTICAL ASPECTS OF MRI TRANSPORT

INTRODUCTION

Referral and transport of a patient to the MRI scanner should be initiated by the Duty Intensivist, who will also take responsibility for the transport unless able to delegate to a suitably qualified registrar. A number of factors make the MRI a technically demanding area to work in:

- The MRI scanner is remote from the ICU
- Indications for MRI scanning usually involve pathology of the spinal cord, or areas of the brain not well visualised on CT scan
- There are specific restrictions on practice by virtue of the MRI technology

SAFETY

There are three main mechanisms, in addition to the standard risks of patient transport, by which patient and staff may be injured:

- The effects of magnetism: The magnetic strength of an MRI is measured in Tesla, with an average machine developing 1.5 -2.0 T. This is similar that required in a scrap yard to lift a car of the ground
The more ferrous an object, the more likely it is to be affected by the magnetic coil. All metallic objects should be removed if possible. Those that cannot be must be discussed with the MRI staff. Of note, metallic heart valves are non-ferrous, and are said to experience less shear stress than that exerted by heart contractility

- Magnetic flux: A changing magnetic field strength induces electrical current in conductors with consequences such as heating in a prosthesis. Rapidly changing fields may even cause nerve depolarisation
- Radio-frequency: Generally in the region of 15-20 kW. This may cause heating or electrical conduction where a coil exists. For this reason cables etc should not be coiled or kinked if excessively long

### RELEVANCE FOR ICU

MRI transport is a team event, requiring a competent nurse, an ICU technician, and an experienced medical escort.

Basic ICU equipment must be removed from the room, with consequences for ventilation (dead space in tubing), infusions (long lag time for any change to take effect), and monitoring (damping of trace).

Some ICU indwelling equipment such as Codman ICP monitors and PiCCO catheters are considered unsafe in the MRI and may have to be replaced or removed.

### PROCEDURE

Registrar to complete patient consent paperwork / MRI risk screening document.

### IN ICU

Liaise with nursing and technical staff so that adequate preparation time is available and an appropriate ventilator is available in MRI. Careful planning is mandatory to ensure adequate sedation, vasopressor or other substance is available, as these may not be able to be sourced in the MRI scanner rooms. In addition to a standard transport, note the following:

- Non-vital infusions are disconnected and capped
- Vital infusions (vasopressors and sedation) will require the addition of extension sets, which are usually pre-coiled with a total length of around 5m (may require preparation of new syringes and pumps)
- Add 3 extra rigid lines to the arterial line. The resulting trace will be damped, but with a reasonably accurate mean pressure
- Take two further 3 way taps for attachment to the infusion lines outside of the MRI room (for boluses) and 4 extra luer plugs for keeping the extension lines sterile during their passage through the scanner wall

### IN THE MRI ANTE-ROOM

- Transfer onto the MRI gurney
- Remove and exchange ICU non-invasive BP cuff for MRI equivalent
- Remove ECG dots

**IN THE MRI**

Attach the MRI oximeter, NIBP and capnograph.

In an aseptic manner, disconnect arterial line extensions using luer plugs, prior to patient movement into scanner gantry. Where vasopressors are in use, the pump should be left infusing (i.e not turned off), the line disconnected using a luer plug and passed through the wall for immediate re-attachment. Use a paper tape measure to establish height of phlebostatic axis prior to movement of patient into scanner gantry.

Move patient into scanner gantry, check there is no fouling of lines or equipment through the range of movement required. Pass through any further lines required for use outside the scanning room itself. Pass through the arterial line extension set for re-attachment to transducer and monitor outside the room. Use height measurement as above to “level” transducer.

When departing from the scanner, perform above in approximately reverse order.
CONSENT IN THE INTENSIVE CARE SETTING

A competent patient may give or withhold consent for any medical treatment (Code of Health and Disability Consumer’s Rights). This includes life-saving treatment. Lack of capacity to consent should not be assumed-talk to the patient if at all possible.

Unfortunately, patients in ICU often cannot have their competency established with certainty. This is because the elements of establishing capacity to consent are altered by illness or medication—e.g. ability to understand, process and retain information on treatment.

When a patient cannot give consent in an emergency, in the absence of convincing evidence to the contrary (e.g. presence of a person with enduring power of attorney for personal care and welfare (EPOA) who can categorically state that the person does not wish to receive the treatment in question, or applicable advance directive) consent to treatment is implied.

PROCEDURES THAT REQUIRE CONSENT

This is a difficult area. Expectation of patients who are competent is that they give verbal consent for virtually every procedure. Keeping the competent patient well informed, allowing questions when possible, usually suffices for all procedures. When patients cannot consent, relatives often expect to receive prior information when the procedure is not routine or when it entails risk. Both of these are a matter of judgement.

A written record of informed consent is unnecessary for the vast majority of bedside procedures in ICU—in practice it is only universally obtained for elective percutaneous tracheostomy.

CONSENT BY RELATIVES

Relatives or friends cannot give or withhold consent for the performance of a medical treatment (unless they hold a relevant EPOA). An EPOA may only represent what the patient would have wanted.

New Zealand statute does however mandate that the treating doctor takes other views into account in deciding whether to perform a particular treatment when the patient cannot consent. In this setting, “those with an interest in the welfare of the patient” must be consulted to ascertain the patient’s likely views. Note that this deliberately provides an informal definition of the concept of family. No family hierarchy in the process is specified. These people can only be consulted if they are reasonably available.

CONSENT AT THE WAIKATO HOSPITAL

A “Right 7(4) Consent Form” is available to try to take the frequent inability of others to give consent for an incompetent adult into account. Completion of the appropriate form is necessary to comply with hospital policy in certain procedures (see Waikato Hospital Policy for Informed Consent).

A written record of informed consent is unnecessary for the vast majority of bedside procedures in ICU—in practice it is only universally obtained for elective percutaneous tracheostomy.

When it is necessary to obtain consent for a particular procedure to be performed on an ICU patient, it is appropriate for ICU medical staff to play a role in this process. This may mean ensuring that the staff, intending
to perform a procedure, make the requisite information available to the ICU registrar to enable them to get consent, or in most cases obtain consent themselves.

MISCELLANEOUS

Consent to participate in research is a specialised area which this document cannot cover.

Issues of a child’s competence to give or withhold consent are likewise too complex to cover in full here. In difficult situations where a child (<16 years) has their safety risked by withholding of parental consent for treatment that a clinician believes is necessary, involvement of the patient’s paediatrician (if applicable), other colleagues, Chief Medical Advisor and in-house Legal Advisor may be necessary (accessed via telephone operator out-of-hours).

Consent is additionally required for a patient to participate in teaching.

A medical practitioner working in a hospital does not require a patient’s consent to take a blood alcohol level if requested by an enforcement officer, providing taking the sample is not prejudicial to the patient’s care (Land Transport Act, 1998).

REFERENCES:

“Health Care Law in New Zealand” Johnson, S (held in ICU and main library).


RESEARCH IN THE INTENSIVE CARE UNIT

Research in The Waikato Hospital ICU is strongly encouraged.

PERSONNEL

Coordinator of Research: Dr Annette Forrest
Nursing Research: Jewel Barlow
Research Committee: ICU Director, ICU Manager, Research Coordinator, ICU Nurse Educator,
Annette Forrest, Tom O’Rourke, Robert Martynoga, Pranesh Jogia

The research committee meets once a month to discuss ongoing issues regarding research, assess validity of proposed projects and managing funding of studies appropriately.

Members of the medical and nursing staff are encouraged to become involved in research during their stay in the unit. Registrars are expected to be aware of active studies in the unit, obtaining consent for which is seen as part of their responsibility within the unit. Information on active studies can be obtained from the Director of Research, or the research coordinator. Research in the unit falls into 3 categories:

- “In House”: Research that originates, and is conducted by staff within the unit
- Contract research: Research conducted and funded by drug companies
- ANZICS Clinical Trials Group (CTG) trials—these should be of particular interest to trainees

PRESENTATIONS

Medical progress relies on dissemination of information to colleagues, and many training programs indeed require presentation of a completed project to a suitable audience as part of training. For this reason completed projects should be presented at a scientific meeting and, if appropriate, publication sought. Some funding will be available for medical / nursing staff who present at any meeting.

SUITABLE FORUMS FOR PRESENTATION

- Annual Scientific Meeting on Intensive Care (Held in October each year)
- Abstracts to be submitted by preceding July
- Prizes are awarded for
- Best free paper (med / nurse)
- Best review (med / nurse)
- Best Poster
- Best paper by a trainee
- Thoracic Society of Australia and New Zealand (March)
INTERNATIONAL MEETINGS

- Society of Critical Care Medicine, North America (Feb)
- International Symposium on Intensive Care and Emergency Medicine, Brussels (March)
- European Society of Intensive Care Medicine, various (Sept)

ETHICS SUBMISSIONS

Worldwide, the trend towards gaining ethical approval prior to commencing a study is becoming increasingly important. Many journals will not publish research done without prior ethics committee approval.

With the exception of some audits, most projects must obtain approval from the Regional Ethics Committee prior to commencement. The clinical trials coordinator should be viewed both as a resource person and an advisor in negotiating ethics committee approval.

Research is a slow process, therefore registrars that wish to do their college project whilst in ICU should obtain ethics approval, and in some cases funding, before the start of the ICU rotation.
OTHER IMPORTANT INFORMATION

INFORMATION TECHNOLOGY

There are numerous terminals in the intensive care unit that offer access to the hospital network from which patient results can be obtained.

There is a hospital intranet which allows access to the library which has an excellent selection of online journals available. All critical care drug protocols are currently available on the intranet.

You will be given a login by the IT department. This allows you access to the laboratory data and to your own account. You will have an e-mail address with access to the GroupWise mail program and its address book which has a list of most company employees and their contact details.

The local area network provides access to the “World Wide Web”. This is controlled and closely monitored by the IT department. Numerous sites of medical interest are readily accessible. Other sites are however blocked by “border manager”. If you feel that a particular site should be available, the URL should be submitted to the system controller who will look at the site and unblock access to it if appropriate.

MEDICO-LEGAL ASSISTANCE

In those situations where you may require medico-legal advice, a legal advisor may be contacted through the switchboard. From time to time you may be asked to prepare reports for an internal investigation, the Coronial Service, the Health and Disability Commissioner or some other agency.

There are very few situations where you should submit a report without the oversight and involvement of a Senior Medical Officer involved in the care of that patient, or the scenario, whatever that may be.

If you are unsure of who you should approach, or for whatever reason do not want to approach the Senior Medical Officer involved, then we suggest you seek advice from the Director of the ICU, the Deputy Director or Supervisor of Training, whichever is the most appropriate person in your estimation.
ENDOTRACHEAL INTUBATION

INTRODUCTION

Endotracheal intubation in ICU patients is a high risk but vital emergency procedure in patients who often have limited reserve, are difficult to position and may have a difficult airway.

All staff should familiarise themselves with the intubation trolley and equipment.

Whenever possible make sure that you have capable and trained staff to assist you. If you are inexperienced (e.g. fewer than 20 intubations), always call for assistance. If the Duty Intensivist cannot be reached for some reason, or is detained, then assistance should be sought from an anaesthetic colleague.

Rapid sequence induction is the rule in ICU patients.

INDICATIONS

- Institution of mechanical ventilation
- To maintain an airway
- Upper airway obstruction or threat
- Control of arterial carbon dioxide content (e.g. in the setting of traumatic brain injury)
- Patient transportation
- To protect an airway
- Patients at risk of aspiration
- Altered conscious state
- Tracheal toilet

TECHNIQUES

Oro-tracheal intubation is the rule.

Awake Fibreoptic intubation, may be indicated in selected patients with cervical spine injury, limited mouth opening or oro-facial surgery / trauma. This technique should only be undertaken by staff with current experience of these techniques, and only after discussion with the Duty Intensivist.

STANDARD ENDOTRACHEAL TUBE CHOICE

All patients in the Waikato Hospital Intensive Care Unit should be intubated with a low pressure high volume PVC tube (e.g. Portex blue line oral nasal tube). This may be changing to supraglottic suction tubes shortly.
NON-STANDARD TUBES

Patients returning from theatre may have a different ET tube (eg. armoured ETT) in situ. Where there is no good reason for this to remain, it should be changed to the standard ETT if it is anticipated that the patient will require intubation > 48 hours, and would not be exposed to significant risk during the ETT change. The armoured tube currently in use on theatre has an appropriate cuff for longer term use.

INTUBATION GUIDELINE

PERSONNEL

Skilled assistance is mandatory; where possible a team of 4 is required

- “Intubator” who controls and co-ordinates the procedure
- “Drug administration”
- A person to apply in-line immobilisation where the stability of the cervical spine is unclear
- Cricoid pressure (CP) is recommended in all emergency situations and should be applied at the commencement of induction. CP may distort the larynx requiring it’s removal. CP is controversial, and its relative risks and benefits should always be considered

PREPARATION

Check all equipment prior to intubation:

- Secure adequate IVI access
- Adequate lighting
- Selection of oropharyngeal airways
- Working suction with Yankauer attachment
- AMBU bag assembly and appropriate mask
- 100% oxygen with flow capability > 15 l/min
- 2 working laryngoscopes with appropriate choice of blade
- Magill forceps
- Malleable introducer and gum-elastic bougie
- 2 x ETT: estimated patient size and one smaller size. (Female = 7-8 mm, Male = 8-9 mm)
- A selection of laryngeal masks + oropharyngeal airways
- Emergency cricothyroidotomy kit: (#15 scalpel and 6.0mm cuffed ETT)
- Ensure adequate monitoring
• Pulse oximetry
• Reliable blood pressure monitoring (eg. invasive if necessary)
• ECG telemetry
• Capnography must always be Immediately Available

**DIFFICULT INTUBATION KIT**

A kit can be found in a yellow bag on the side of the Unit 2 intubation trolley containing:

- An intubating LMA
- McCoy laryngoscope
- Light wands
- Emergency cricothyrotomy kit

Jet ventilation system – NOT recommended for adult cricothyroidotomy, only paediatric.

*Our difficult intubation kit is currently undergoing review.*

A video laryngoscope is available within the unit. It is certainly a useful adjunct for patients with a poor Cormack-Lehane view on direct laryngoscopy. It must be cautioned that a good view with a video laryngoscope does not guarantee the ETT will pass easily through the cords. A stylet can be useful in this regard. Indirect video techniques count towards the no more than three attempts rule. A cricothyroidotomy in a hypoxic patient should never be delayed, especially after three supraglottic attempts, for any perceived benefit of videolaryngoscopy.

**DRUGS FOR INTUBATION**

**INDUCTION AGENT**

eg. Etomidate (0.2mg/kg), Thiopentone, Fentanyl, Ketamine, Midazolam.

**MUSCLE RELAXANT**

Suxamethonium 1-2 mg/kg

Consider rocuronium 1-2 mg/kg if suxamethonium contra-indicated i.e:

- Burns patients > 48 hrs post injury
- Spinal cord injury patients >72h injury or where spasticity is present
- Some acute neuromuscular disease (e.g. GBS)
- Hyperkalaemic states
- Malignant hypothermia
- Prolonged immobilisation

**MISCELLANEOUS**

- Atropine 0.6-1.2 mg
- Adrenaline 10 ml of 1:10 000 solution
- Phenylephrine 10mg in 100mls then drawn up into 10mls. 1-2mls PRN

**PROCEDURE- RAPID SEQUENCE INDUCTION AND OROTRACHEAL INTUBATION**

Pre-oxygenate for 3-4 minutes with 100% oxygen. Patients receiving non-invasive ventilation should continue on this form of ventilation until the point of induction, and a PEEP valve applied to the AMBU-bag mask assembly.

- Administer induction agent and suxamethonium
- Apply cricoid pressure
- Intubation under direct visualisation
- Inflate ETT cuff until there is no air leak during ventilation
- Confirm ETT placement with capnograph and chest auscultation with manual ventilation
- Release cricoid pressure
- Secure ETT at correct length (Female = 19-21cm at incisors, Males = 21-23 cm at incisors)
- Do not cut ETT
- Connect patient to ventilator
- Ensure adequate sedation and analgesia to cover period of muscle relaxant and continue as indicated by clinical scenario
- Insert naso-/oro-gastric tube if not already present

**INTUBATION CHECK LIST**

- Personnel (4 people)
  1. Intubator
  2. Cricoid pressure person
  3. Drug/Equipment person
  4. Spare person
• Ideally 2 x MO
• ICU Consultant

Drugs

• Large IV access (x2 people if possible)
• 1000ml N/Saline running
• Induction agent
• Neuromuscular blocker
• Maintenance sedation (MnM/Propofol etc)
• Long acting NMB
• (if not given in “2”)
• Vasoactive agents (adrenaline/ephefrine etc)

INTUBATION CHECK LIST

• Equipment
  • Bag valve mask
  • O2 attached and on at 15 L/min
  • Working Suction
  • Bougie / stylet
  • Guedel airway
  • Working Laryngoscope x2 (Mac 3+4 blades)
  • Checked ETT plus lubricant (plus smaller size ETT)
• 10 ml syringe
• Tube tie
• NG tube
• End-Tidal CO2
• Monitor Saturation

FINAL PREPARATION

• Airway assessed, position optimised and well pre-oxygenated
• Plan B discussed with personnel and equipment available
• Communication: All personnel know roles and responsibilities

**DOCUMENTATION**

• Time and date
• Drugs
• Intubation
• ETT position on CXR
• ETT cuff pressure (20-30cm H2O)
• NIBP 3-5min cycle

*A follow-up CXR should be performed as soon as convenient post intubation.*

**FAILED INTUBATION AND DIFFICULT AIRWAY ALGORITHM**

Assess the likelihood and clinical impact of basic management problems

• Difficult ventilation
• Difficult intubation
• Difficulty with patient co-operation or consent
• Difficult tracheostomy

If intubation is likely to be difficult and time allows seek expert anaesthetic assistance which is often best performed in theatre.

Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management, for example nasal prongs with high flow oxygen after pre-oxygenation once facemask removed.

Consider the relative merits and feasibility of basic management choices:

• Awake intubation vs. Intubation after induction of general anaesthesia
• Non-invasive techniques as an initial approach vs. Invasive technique as initial approach
• Preservation of spontaneous ventilation Vs ablation of spontaneous ventilation
• Develop primary and alternative strategies

The Canadian airway focus group (2013) divides patients into two groups: the unconscious patient Vs the anticipated difficult tracheal intubation.

Many of our patients cannot be woken up so need a secured airway. There should be no more than three attempts at intubation. Further attempts risk bleeding and worsening of the airway. It may be appropriate to establish bag mask ventilation or pass a supra-glottic device for oxygenation. If the patient cannot be oxygenated or intubated then a cricothyroidotomy is indicated.
AWAKE INTUBATION

An awake intubation in the ICU should be directed by the Duty Intensivist, or directly delegated by the same. Intubation with the patient awake may be achieved by a variety of methods. Establishment of a surgical airway is an alternative.

FAILED INTUBATION ALGORITHM
Anticipated difficult tracheal intubation

Airway exam or history predicts difficult tracheal intubation

If general anesthesia is induced...
1. Is tracheal intubation predicted to succeed in no more than 3 attempts?
2. If tracheal intubation fails, will face mask or SGD ventilation succeed?

...and are other patient and contextual issues favorable?
1. Rapid oxygen desaturation unlikely with onset of apnea?
2. Little risk of aspiration once unconscious?
3. No obstructing airway pathology?
4. Additional skilled help available?
5. Clinician skilled in planned technique(s) and equipment available?

Low risk of failed oxygenation if induced
Consider intubation after induction of general anesthesia
- e.g., IV induction (e.g., RSI)
- e.g., inhalational induction

Significant risk of failed oxygenation if induced
Is awake intubation feasible?
- Patient can cooperate
- Situation acuity permits

Consider awake intubation/technique
- e.g., awake oral/nasal
- e.g., awake tracheotomy

Other options
- e.g., induction with ‘double set-up’ preparation for immediate cricothyrotomy

Is local or regional anesthesia feasible for surgical case?

Figure Flow diagram: anticipated difficult tracheal intubation. SGD = supraglottic device; IV = intravenous; RSI = rapid sequence induction/ intubation
Fig. 1 Flow diagram: difficult tracheal intubation encountered in the unconscious patient.
SGD = supraglottic device
REFERENCES


MALLAMPATI CLASSIFICATION

For grading airways from the least difficult airway (I) to the most difficult airway (IV).

- Class I = visualization of the soft palate, fauces, uvula, & anterior and posterior pillars
- Class II = visualization of the soft palate, fauces, & uvula
- Class III = visualization of the soft palate and the base of the uvula
- Class IV = soft palate is not visible at all

There are many ways to estimate difficult intubation. None are perfect. However an assessment of likelihood of difficult ventilation and/or intubation should always occur.

MAINTENANCE OF ENDOTRACHEAL TUBES

TAPES

- ETT are generally secured with white tape
- Tapes are changed daily or PRN by nursing staff
- In certain circumstances personalised ETT security may be required

CUFF INTEGRITY

Sufficient air should be placed into the cuff to prevent an air leak, as assessed by auscultating over the trachea. A technique which prevents cuff over-inflation should be used.
PERSISTENT CUFF LEAKAGE

Any ETT that constantly requires additional air instilled into the cuff should be reviewed for:

- Herniation above the cords
- Cuff damage (rare)
- Malfunctioning pilot tube valve (which can be excluded by placing distal pilot tube into container of water and observing for bubbling)

AIRWAY SUCTIONING

Airway suction is performed prn.

Routine suctioning should be avoided especially where:

- it requires disconnection of PEEP (open suction system)
- May exacerbate the patient’s condition (asthma, reactive Intra-cranial pressure, florid pulmonary oedema)

ENDOTRACHEAL TUBE CHANGE

EQUIPMENT AND ASSISTANCE

- The procedure / setup is the same as for intubation de novo
- Ensure patient is adequately oxygenated
- Ensure adequate anaesthesia and muscle relaxation

PROCEDURE

Perform direct laryngoscopy. If a good view of the larynx and vocal cords is obtained then proceed to manual exchange of ETT with application of cricoid pressure, or proceed as below using gum-elastic bougie.

If direct laryngoscopy reveals abnormal or swollen anatomy, or only partial view of anatomy, then proceed as follows:

- Place gum elastic or ventilating bougie through the ETT and insert to a length corresponding to a few cm distal to the end of the ETT
- With an assistant stabilising the bougie, and applying cricoid pressure, remove faulty ETT under direct laryngoscopy, while maintaining bougie in the same position
- Confirm the bougie is still in place through cords once ETT removed, and then replace new ETT over the top of the bougie apparatus
- If the ETT does not progress smoothly through the cords, rotate 90 degrees anti-clockwise and attempt again (i.e. Realign bevelled edge of ETT along upper border of bougie)
• Check position of ETT and secure as for de novo intubation procedure

**APPROACH TO MUSCLE RELAXANTS IN THE INTENSIVE CARE UNIT**

*Muscle relaxant use in the intensive care setting is to be discouraged unless specifically indicated.*

There is no circumstance where a patient should receive muscle relaxant without covering sedation where the possibility of consciousness exists.

Where doubt exists with regard lingering effects of muscle relaxant, this should be confirmed with a nerve stimulator, prior to stopping sedation. The indications for using muscle relaxants in ICU are limited to:

- Endotracheal intubation and acute control of ventilation post intubation
- Patient transport
- Selected patients with complicated ventilatory parameters including early ARDS
- Facilitate acute procedures: tracheostomy, bronchoscopy
- In selected patients with severe head injury and uncontrolled intra-cranial pressure

**REFERENCE:**

Neuromuscular blockers in early ARDS. NEJM 2010;363:1107-16.

**COMMONLY USED MUSCLE RELAXANTS**

<table>
<thead>
<tr>
<th>Relaxant</th>
<th>Dose</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suxamethonium</td>
<td>100-200 mg IVI or 1-2 mg/kg IVI</td>
<td>Consider pre-treatment with atropine (0.6-1.2 mg) if potential bradycardia and always with second dose; Contraindicated in burns (&gt; 3 days post burn), chronic spinal and neuromuscular syndromes, hyperkalaemic states (K &gt; 5.5 mmol/L); Prolonged immobilisation; Malignant hyperpyrexia.</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>2-8 mg IVI</td>
<td>First line non-depolarising agent; Repeated dosing leads to accumulation, and possibly increased risk of myopathy/neuropathy.</td>
</tr>
<tr>
<td>Atracurium</td>
<td>0.5 mg/kg IVI</td>
<td>Clearance independent of renal or hepatic metabolism.</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>0.6 mg/kg IVI</td>
<td>Rapid onset (60 seconds) non depolariser; Duration of action: 30-40 minutes</td>
</tr>
</tbody>
</table>
**ROCURONIUM AS AN ALTERNATIVE TO SUXAMETHONIUM FOR RAPID SEQUENCE INTUBATION.**

- Used when suxamethonium contra-indicated
- May also consider where emergence is not a viable outcome, ie comatose state. Sugammadex can be used for immediate post-rocuronium reversal (dose 16mg/kg)
- Contraindicated in renal failure (Creatinine clearance <30 mL/minute)
- Lower doses required for residual paralysis
- Sugammadex is available to Anaesthetists, in theatre

**EXTUBATION GUIDELINE**

Ensure adequate assistance, monitoring and equipment as for intubation.

Extubation should generally not be performed overnight if the responsibility to re-intubate might fall on a less experienced staff member. Patients may be extubated if this action is part of an established care plan or algorithm (eg. cardiothoracic), or at the direction of the Duty Intensivist.

No patient should be extubated without medical staff being aware and available to assist.

**PATIENT SELECTION**

For a more extensive description see section on mechanical ventilation.

- The patient must be awake enough to maintain their own airway.
- Any threat to airway patency as a result of surgery or injury may require consultation with the co-managing team (ENT or Plastic surgery) prior to extubation.
- Patient should demonstrate adequate pulmonary reserve. There are a number of ways of assessing pulmonary reserve although none is perfect:
  - Resp rate < 30
  - FVC > 15 ml/kg
  - PaO2 / FiO2 ratio > 200
  - Resp rate / tidal volume 1 min after disconnection from ventilator (use T-piece)

The last method has the best predictive value.

**REFERENCE:**

FIBREOPTIC BRONCHOSCOPY

POLICY

Only to be performed by adequately trained staff, after authorisation by the Duty Intensivist.

INDICATIONS

• Persistent lobar collapse that is refractory to normal bronchial toilet

• Foreign body in airway

• Diagnostic broncho-alveolar lavage (BAL)

• Fibre-optic intubation

CRICOTHYROIDOTOMY

POLICY

This is the recommended procedure for urgent surgical airway access (not percutaneous tracheostomy).

When urgent surgical airway is required, call for help then proceed without delay.

As cricothyroidotomy is a rarely used skill for most practitioners regular practice/simulation is recommended.

INDICATIONS

• Failed intubation drill

• Inability to maintain an airway despite basic manoeuvres

EQUIPMENT

Purpose made kits exist in the unit using direct access and/or a Seldinger technique (Melker kits). In the event of these not being available, the simplest technique is described below:

• # 15 scalpel and handle

• Size 6.0 cuffed ETT loaded on to a gum elastic bougie

• Oxygen delivery circuit and ventilation device (eg. Laerdal bag)

PROCEDURE

• Palpate cricothyroid membrane

• Perform 2cm vertical incision through skin in the midline and then a horizontal puncture through the cricothyroid membrane
• Using the blunt side of scalpel blade, apply traction and create a small window to enter the trachea with a bougie

• Insert bougie then ETT into trachea. All of the ETT cuff should be in trachea but only just

• Connect oxygen circuit

• Confirm correct placement with end-tidal CO2, auscultation, and if possible CXR

• Perform tracheal toilet as soon as adequate oxygenation achieved

Arrange definitive surgical airway as soon as possible. A cricothyroidotomy can be used for up to 72 hours. Other therapies may be of higher priority.

TRACHEOSTOMY - PERCUTANEOUS

POLICY

Percutaneous tracheostomy is the preferred method for elective tracheostomy in suitable critically ill patients.

The decision to perform percutaneous tracheostomy rests with the Duty Intensivist.

Where appropriate the co-managing team should be consulted prior to performing the procedure.

Consent should be obtained as outlined in the unit guidelines.

Percutaneous tracheostomies may only be performed by experienced specialist staff or ICU vocational trainees under supervision.

INDICATIONS

• As for surgical tracheostomy

• Airway maintenance:
  
  o Prolonged intubation (> 10 days) or anticipation thereof
  
  o Prolonged upper airway obstruction
  
  o Laryngeal pathology
  
  o Subglottic stenosis
  
  o Airway protection
  
  o Delayed return of glottic reflexes
  
  o Tracheal toilet / ineffective cough mechanism

RELATIVE CONTRAINDICATIONS

• Elevated or unstable measured intra-cranial pressure
• Coagulopathic state:
  o Platelets < 100 000 (or abnormal function eg. following aspirin)
  o APTT > 40 sec
  o INR > 2.0
• Renal failure with uncorrected uraemic state
• Previous neck surgery
• Unstable cervical spine injury
• Unsuitable anatomy

**PROCEDURE**

Percutaneous tracheostomy is commonly performed using two experienced operators:

- Anaesthetist / endoscopist: Responsible for administering a suitable anaesthetic and managing the airway
- Surgeon-operator

**EQUIPMENT:**

- Monitoring and drugs are as for standard endotracheal intubation, with the recommended addition of the fibreoptic bronchoscope
- Adequate lighting essential
- Patient ventilated on 100% oxygen and a pressure controlled ventilation mode
- A Cook kit using a “blue rhino” dilatational technique is standard. The guide-wire forceps technique using the Griggs forceps should only be used by operators trained in this technique
- Tracheostomy tubes: The “Portex” tracheostomy tube is the standard tube used in this unit

*No patient should leave the ICU without the inner cannula being inserted prior to discharge in an effort to confirm patency.*

Inner tube does not need to stay in place- it is just used to confirm patency.

**EDUCATION AND TRAINING**

Senior Registrars and selected advanced trainees will be invited to learn how to perform percutaneous tracheostomies. This will involve hands-on training with a skilled operator scrubbing alongside the trainee.

**AIRWAY MANAGEMENT**
Endoscopic confirmation of surgical technique is not practiced universally, but it is a useful adjunct to correct placement.

**METHOD 1**

- Place the fibreoptic bronchoscope in the trachea beyond the distal tip of the ETT
- Under direct laryngoscopy retract the ETT (with deflated cuff) so that the cuff is above the vocal cords and inflate the cuff with 10-15 ml of air
- Use an assistant to secure tube in place and apply slight downward force on the ETT to maintain a seal to ventilate the patient
- Retract bronchoscope to a point proximal to planned tracheal puncture

**METHOD 2**

- Place the fibreoptic bronchoscope in the trachea beyond the distal tip of the ETT
- Withdraw the ETT 2-3 cm with the cuff deflated, then reinflate cuff
- Request the surgeon-operator apply digital pressure over intended tracheal puncture site, and confirm this is distal to ETT tip and bronchoscope
- Beware ETT puncture or bronchoscope damage
- Observe correct placement of needle-guidewire by Seldinger technique, and sequential dilatation
- Once tracheal tube in situ, connect to ventilator and insert bronchoscope into tracheostomy
- Confirm tip of tracheostomy clear of carina, and absence of ongoing haemorrhage

**TRACHEOSTOMY INSERTION TECHNIQUE**

- Position patient: 30 degrees head up, with neck in extension but supported
- Adopt strict aseptic technique
- Infiltrate with 10 ml of 1% lignocaine / 1:100 000 adrenaline over the pre-tracheal rings
- Check tracheostomy tube cuff, lubricate and insert dilator into tracheostomy tube making sure there is a good fit
- Perform a 1-2cm incision over the 2nd tracheal ring
- Dissect bluntly to fascia
- Insert sheathed needle catheter in to trachea at midline. Confirm placement by aspirating air and confirming with endoscopist
- Remove needle, and feed guidewire through sheath
- Remove sheath and dilate with mini-dilator
• Place white dilator-guide over sheath
• Proceed to dilatation with “rhino” (to appropriate size according to desired size tracheostomy)
• Remove dilator and use guidewire to insert dilator and tracheostomy into the trachea
• Remove dilator and wire, inflate cuff and confirm placement with bronchoscope
• Secure with tapes
• Perform a control CXR if the procedure has been difficult or accomplished without bronchoscopy

**COMPLICATIONS**

- Haemorrhage (may be delayed as lignocaine / adrenaline wears off)
- False passage, posterior wall tear
- Loss of airway
- Pneumothorax
- Cricoid fracture (often tracheal ring fracture occurs as “normal part of procedure”)
- Laryngeal dysfunction
- Tracheal stenosis
- Infection
- Cuff leak (see cuff leak policy under intubation)
- Dilatation of Murphy’s eye
- Innominate artery fistula
RESPIRATORY THERAPY

Traditionally the major reason for referral to intensive care, respiratory failure and our understanding of how best to manage it is constantly evolving.

Recent advances in ventilatory strategy, and their impact on not only lung injury but also on other organ dysfunction, necessitates that all staff within the ICU acquire some understanding of the pathophysiology involved.

While Registrars and residents are encouraged to understand the principles of ventilation, and indeed participate in the management of ventilated patients; decisions regarding ventilation, weaning, extubation and other extra-ordinary actions (such as patient proning) remain the domain of the Duty Intensivist.

RESPIRATORY FAILURE

Definition: Failure of efficient gas exchange. Either failure to oxygenate adequately, or failure to ventilate.

FAILURE TO OXYGENATE ADEQUATELY – TYPE I FAILURE

\( P_{aO_2} < 60 \text{ mmHg} \) under the following conditions:

- \( F_{O_2} 21\% \) (i.e. room air)
- Barometric Pressure 760 mmHg (sea level)
- No intracardiac shunt

NB: This does not mean taking a patient off oxygen to perform an arterial blood gas, but rather inferring the need for “assistance” as stated below. Examples include pulmonary oedema, pneumonia and pulmonary haemorrhage.

FAILURE TO VENTILATE ADEQUATELY – TYPE II FAILURE

\( P_{aCO_2} > 50 \text{ mmHg} \), unless in the presence of a primary metabolic alkalosis (pH normal or elevated).

AETIOLOGY

- Lung insult
  - Pulmonary oedema (hydrostatic-cardiogenic, or leaky capillary-ARDS)
  - Pneumonia
  - Contusion
  - Haemorrhage
  - Airway pathology
  - Proximal
  - Distal: COAD, asthma, bronchiectasis, sputum retention
• Neuromuscular
• Depressant drugs
• Intra-cranial pathology
• Guillan Barré
• Myasthenia Gravis
• Skeletal
• Loss of chest wall integrity: flail chest
• Loss of chest wall elasticity: severe kyphosis or scoliosis
• Intra-thoracic space occupying lesion: Pneumo-/ Haemothorax, pleural effusions

WHEN SHOULD I CONSIDER VENTILATING (± INTUBATING) PATIENTS?

INDICATORS

Clinical assessment outweighs any “result” such as an ABG / CXR or other objective measurement (see below).

Consider institution of ventilation in the presence of:

• Threatened airway
• Fatigue or imminent exhaustion
• Inability to effectively cough or clear secretions
• Respiratory failure (as above)

OBJECTIVE MEASUREMENTS

In the appropriate clinical setting, and where time allows a combination of the following may assist your decision:

• Resp rate > 35 breaths per minute
• Tidal volume < 5 ml/kg
• Vital capacity < 15 ml/kg
• Abnormal oxygenation as indicated by:
  • $P_aO_2 < 75\text{ mmHg on an } F_iO_2 > 0.4 (40\% O_2)$
  • $P_aO_2$ to $F_iO_2$ ratio < 150
• Abnormal ventilation as indicated by:
  • $P_aCO_2 > 60\text{ mmHg}$
## OXYGEN DELIVERY SYSTEMS

<table>
<thead>
<tr>
<th>Class</th>
<th>Device</th>
<th>Oxygen Flow (l/min)</th>
<th>Approx F\textsubscript{O2} as percent</th>
<th>Comment / Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Variable delivery devices</strong></td>
<td>Nasal Catheter</td>
<td>2</td>
<td>28%</td>
<td>Not suitable for acutely ill patients.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td>35%</td>
<td>Indicated to provide supplementary oxygen only in stable patients</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6</td>
<td>45%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Semi-rigid masks</td>
<td>5</td>
<td>35%</td>
<td>Inspired fraction of oxygen variable with minute volume</td>
</tr>
<tr>
<td>(eg Hudson)</td>
<td></td>
<td>6</td>
<td>50%</td>
<td>Popular because of low cost, not accurate oxygen delivery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8</td>
<td>55%</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10</td>
<td>60%</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>12</td>
<td>65%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Reservoir plastic masks</td>
<td>6-15</td>
<td>F\textsubscript{O2} = 21% + 4% per l/min</td>
<td>Fixed delivery device at all but extreme of respiratory effort.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>More expensive than Hudson type mask, but preferable in unstable patients</td>
</tr>
<tr>
<td><strong>Fixed Delivery Devices</strong></td>
<td>Venturi type masks</td>
<td>2-8</td>
<td>24-50% according to product specs</td>
<td></td>
</tr>
<tr>
<td></td>
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<tr>
<td></td>
<td>CPAP devices</td>
<td></td>
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<td>Most of these devices make use of turbine devices or high wall flow to provide oxygen / gas mixtures at flow rates in excess of that achieved at peak respiratory effort. They usually all allow reasonably accurate oxygen / gas blending and therefore control of patient F\textsubscript{O2}</td>
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<td>BiPAP system</td>
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<td>Ventilator</td>
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**HUMIDIFICATION**

**GENERAL**

Poor conditioning of the temperature and humidity of inspired gases leads to airway damage, sputum plugging and may even increase morbidity and mortality of during an ICU stay. Any patient ventilated in the ICU requires adequate humidification. Deleterious sequelae can develop with in hours of exposure to dry gas during mechanical ventilation.

All patients that are intubated / tracheostomised must have adequate humidification of inspired gases using one of two mechanisms.

**HEAT AND MOISTURE EXCHANGERS (HME’S)**

Effective first line humidifier: Conserves patient’s exhaled water vapour and temperature (gas re-inspired at about 20 deg C). Still requires patient to be able to warm and humidify inspired gas to some degree.

- Not effective at minute volume in excess of 10 l/min
- Must be changed daily
- Cannot be used with an in-line nebuliser
- Incorporates a bacterial filter

**HEATED WATER HUMIDIFIERS (FISHER AND PAYKEL EVAPORATIVE HUMIDIFIER)**

Where any doubt exists about adequate humidification, a heated water humidifier should be the default humidifier, particularly those patients in whom there is bronchorrhoea, sputum inspissation or haemoptysis.

Generally these devices supply gas to the upper proximal airways at 29-32 °C and 95-100% relative humidity, requiring minimal modification within the lungs.

**MECHANICAL VENTILATION**

Mechanical ventilation is one of the mainstays of Intensive Care Medicine and you should attempt during your stay to develop an understanding of the basic principles and practice of ventilation.

Registrars are not expected to manage patient ventilation alone. While most patients can be ventilated using a “default” setting (see below), ventilation of complex patients remains the domain of the Duty Intensivist.

Senior Critical Care Nursing Staff may be useful resource people to aid in troubleshooting, and assisting with instituting ventilation using a default setting.

*No change may be made to a ventilator without clear written order on the appropriate chart and communication with bedside staff.*

*Certain patients may be pre-designated for nurse initiated ventilation changes-see nursing guidelines.*
INDICATIONS FOR MECHANICAL VENTILATION

- Respiratory failure
- Maintenance of cardiopulmonary homeostasis in an unstable or high risk environment
- Following cardiac arrest
- Post-operative support in high risk surgical patients
- Control of intracranial pressure
- Patient Transport / assessment
- Muscle relaxant anaesthesia

OBJECTIVES OF MECHANICAL VENTILATION

- To improve alveolar ventilation and reduce $P_aCO_2$
- To improve oxygenation and ventilation perfusion mismatch
- To increase end expiratory lung volume to prevent or treat lobar or pulmonary collapse and atelectasis
- To increase functional residual capacity through the use of PEEP, which may help improve oxygenation or reduce lung injury through adequate recruitment with the prevention of repeated opening and closing of alveoli
- To unload the respiratory muscles when there is an unbalance between load and the ability to cope. This results in respiratory muscle insufficiency or ventilatory failure
- To allow adequate sedation and paralysis of the patient to aid control to enable the underlying disease state to be adequately treated

In some conditions such as trauma where there is loss of chest wall integrity such as in a flail chest, ventilation may be needed to stabilise the chest wall and to initiate other treatment such as analgesia with safety

COMPLICATIONS OF MECHANICAL VENTILATION:

HAEMODYNAMIC

- Increased intrathoracic pressure-unmasking of hypovolaemia (although there is significant benefit to LV performance with application of PEEP)

RESPIRATOR

- Nosocomial pneumonia
- Volutrauma
- Barotrauma
VENTILATOR SETTINGS

Mechanical ventilation serves two basic functions: ventilatory support and oxygenation support. Ventilatory support is designed to provide either total or partial gas transport between the environment and the alveoli. Usually this is done by providing positive airway pressure in a manner that mimics the normal tidal volume and breathing frequency pattern.

In contrast oxygenation support is designed to supplement the FIO₂ and to optimise ventilation perfusion matching to effect alveolar gas transport. The most common technique to accomplish this is the application of positive end expiratory pressure (or PEEP), but manipulations of the ventilatory pattern and other strategies can also be used.

CLASSIFICATION OF VENTILATORS

Mechanical ventilators have been classified according to the characteristics of the inspiratory phase:

- If they provide a constant inspiratory pressure they are known as pressure generators
- If they provide a constant inspiratory flow they are known as flow generators

FLOW GENERATORS

These usually deliver a preset volume of gas to the lung independent of the change in pulmonary or chest wall compliance or airway resistance. The pulmonary and chest wall compliance and airway resistance determine the proximal airway pressure produced by these machines.

PRESSURE GENERATORS

These deliver gas at a pre-set pressure. They are often simple, small, robust and cheap. The volume of gas that they deliver can be altered by a change in the patient’s lung or chest wall compliance or airway resistance.

Modern ventilators encompass both types of generator. They can ventilate the patient either by pre-set volume, independent of compliance, or pre-set pressure which is interactive with pulmonary compliance and resistance, thus altering tidal volume.

It is important to become familiar with the mechanics of both modes.
VENTILATORY STRATEGIES TO PROVIDE TOTAL VENTILATORY SUPPORT

Current approaches to total ventilatory support generally attempt to duplicate the normal bulk flow ventilatory pattern and use tidal volumes (VT) of 5-10 ml/kg.

- Machine breath rates of 10-30 breaths per minute and inspiratory to expiratory ratios (I:E) of 1:4 to 1:2
- These positive pressure breaths are generally delivered as either flow limited volume cycle breaths or pressure limited time cycle breaths
- Positive pressure ventilatory support is usually used in conjunction with elevations in baseline (end expiratory) pressure (PEEP) and supplementary oxygen

These settings generally provide safe and effective total ventilatory support in most patients in respiratory failure.

In more complex patients, conventional approaches do not provide ideal blood gas values, or airway pressures may be excessively high. Under these circumstances other strategies may be considered.

CONTROLLED MECHANICAL VENTILATION

This is the most basic form of mechanical ventilation supplying all ventilation in the apnoeic patient. Spontaneous breaths are not available.

During pressure control ventilation (PCV) each breath is delivered as time pressure controlled breaths and tidal volume varies, dependent on the resistance of the airway, elastance and the total PEEP.

ASSIST CONTROL VENTILATION (ACV)

In addition to a pre-set background rate of CMV breaths the patient’s inspiratory effort initiates a standard CMV breath.

The ability to control respiratory rate means that less sedation is required, however the respiratory muscles continue to contract during these assisted breaths with only a small reduction in work compared to unassisted spontaneous breaths.

INTERMITTENT MANDATORY VENTILATION (IMV)

This was introduced to allow unimpaired spontaneous breaths while still ventilated with intermittent CMV breaths to minimise sedative use and to reduce respiratory muscle disco-ordination, allowing more rapid weaning.

SYNCHRONISED INTERMITTENT MANDATORY VENTILATION (SIMV)

SIMV is designed in an attempt to avoid “breath-stacking.”. This mode of ventilation controls the inspired flow rate, pattern, time and volume. Providing the inspired pressure limits are not exceeded (30cm H2O) it will deliver a pre-set tidal volume. Patients are unable to breath against the ventilator during the inspired phase. This may result in patient-ventilator dysynchrony. If the respiratory demand exceeds the delivered inspiratory flow rate, this causes flow starvation, and a negative pressure will develop in the circuit. This is distressing for the patient; increases risks of micro aspiration and may result in haemodynamic disturbances.
SIMV is delivered with a refractory period where the next mandatory breath can not be triggered. Most commonly pressure support is employed during this period (SIMV + PSV). If this is not sufficient the patient should be switched to another mode to allow breathing during all phases of ventilation. Typically spontaneous modes; (assisted spontaneous breathing), or Bi-Level (controlled pressure) are used. During Bi-level the pressure support can be manipulated during time low and is fixed at a very low level of extra support during time high. Alternatively the patient may be paralysed if they continue to have significant respiratory distress. This is rarely necessary.

**PRESSURE SUPPORT VENTILATION (PSV)**

During this type of ventilation the patient breaths are supported to a pre-set pressure using additional gas flow.

- Inspiration is usually terminated when the inspiratory gas flow falls to about 25% of the initial flow rate.
- The ramping of acceleration of initial inspiratory support, flow or pressure trigger for inspiratory support, and termination of inspiratory support can all be manipulated to potentially improve patient dysynchrony.

The main disadvantage of pressure support ventilation is that the tidal volume may alter so that minute volume will alter depending on respiratory drive, pressure support level and respiratory system compliance.

Excessively large tidal volumes resulting in overstretch of the lung may occur, possibly contributing towards ventilator associated lung damage.

The trans-pulmonary pressure gradient is not measured by the ventilator. The measurement does not account for the intrapleural pressure being generated by the patient. If the patient is generating -15 cm H2O of intrapleural pressure and the inspiratory pressure is +15 cmH2O the trans-pulmonary pressure is 30 cm H2O. One should be mindful of this when switching a patient from mandatory to support modes.

On some ventilators there is a similar type of ventilation called volume support (VS) which is a mode of adaptive pressure support ventilation where breath to breath logic is used to assure pre-set tidal volume.

There are many other forms of ventilation which at the moment are still being investigated. These include airway pressure release ventilation, bi-level ventilation and proportional assist ventilation.

**BILEVEL PRESSURE CONTROL**

This mode provides set inspiratory and expiratory pressures, and allows the patient to breathe at any time during the respiratory cycle. Generally speaking patients find this more comfortable as flow starvation is rare and they can determine their own respiratory patterns. However in all pressure control modes, the tidal volume is then dependent on the inspiratory resistance, and compliance of the lung, chest wall and abdomen. It is therefore important the tidal volumes of patients on pressure control ventilation are carefully monitored and the inspiratory pressures adjusted as necessary. I.e. \( \text{Paw} = \text{E.Vt + Q.Resinsp} \).

- Where \( \text{Paw} \) = Peak airway pressure, \( \text{E} \) = Lung and chest wall Elastance, \( \text{Q} \) = inspiratory flow rate, \( \text{Resinsp} \) = inspiratory resistance.

**PSV and VSV are spontaneous modes. They cannot be used in paralysed patients.**
OTHER VENTILATORY STRATEGIES

Rarely when conventional ventilation strategies described above have failed alternative ventilation strategies may be employed. These should only be initiated by the Duty Intensivist with clearly documented instructions given.

REVERSE I:E RATIO

The conventional inspiratory to expiratory (I:E) ratio is generally 1:2 to 1:4. This range of I:E ratio tends to synchronise with the patient’s spontaneous ventilatory drive and permits adequate expiratory time for the lung to return to functional residual capacity (FRC) using the recoil pressure of the lung. Lengthening the inspiratory time to I:E ratios approaching 1:1 or even exceeding it (inverse ratio ventilation) can be accomplished in either volume or pressure cycled modes.

Prolonging inspiration has several physiological effects. The alveolus is held at its inspiratory volume for a longer period. This should allow more mixing time between the alveolus and the conducting airway and more exposure of the capillary blood to fresh gas. Some studies have shown an improvement in ventilation perfusion (V/Q) mismatching with this technique and increases in the PaO2.

Incomplete lung emptying: Under these conditions the lung cannot return to its normal FRC and intrinsic PEEP or auto-PEEP develops.

Many of the studies on long inspiratory time and inverse ratio ventilation showing an improvement in gas exchange have probably had this occur as a consequence of auto or intrinsic PEEP. Long inspiratory times with air trapping may also improve V/Q mismatching because it functions like applied PEEP, however there is often a trade-off to allow permissive hypercapnoea. This is largely a consequence of lower set respiratory rates to allow adequate expiration per breath.

Baseline alveolar pressure rises and thereby this raises maximum alveolar pressure for a constant tidal volume. The main role of inverse ratio ventilation is in alveolar recruitment in acute lung injury and ARDS. In these conditions it is used as a ventilatory strategy in an attempt to improve oxygenation.

It is important to realise that once the I:E ratio has been inverted the need for increased sedation and neuromuscular paralysis starts to increase. The mode is inherently uncomfortable and is poorly tolerated in lightly sedated patients.

There may be negative effects on cardiac output (increased intrathoracic pressure impeding venous return). Highly elastant lung parenchyma is much less likely to impact high airway pressures onto the cardiovascular system.
VENTILATION IN THE PRONE POSITION

Until recently ventilating a patient in the prone position has not been shown to improve mortality, however in up to 60% of selected patients there is a significant improvement in oxygenation, often persisting beyond the period spent prone. In 2013 the PROSEVA study published a scarcely believable reduction in 28 and 90 day unadjusted mortality in patients with severe ARDS. Unadjusted mortality at 90 days was 23.8% in the prone group and 40% in the supine group. The proning protocol mandated patients were prone for at least 16 hours per day in the intervention group.

It is unclear how long a patient should be ventilated in the prone position. The majority of patients that do respond do so quickly, however up to 30% may exhibit delayed improvement. Available evidence supports >12hrs. 

The decision to prone a patient should not be made lightly, and is the domain of the ICU specialist

Once the decision has been made to prone a patient, this should be done following ICU nursing guidelines, under the direction of an experienced nursing team

RATIONALE FOR PRONE VENTILATION:

- Increased uniformity of regional pleural pressure gradient
- Improvement in dorsal ventilation with a reduction in shunt fraction
- Improved ventilation-perfusion heterogeneity
- Uniform distribution of lung water and exudate
- Improvement in FRC with further alveolar recruitment
- Reduction in diaphragmatic splinting and improved movement of the posterior diaphragm
- Non-restriction of abdominal contents

INDICATIONS:

- Severe ARDS as given by: \( P_{a}O_2: F_{O_2} \) ratio < 100
- Non response to standard supportive / ventilatory care
- Local or anatomical factors (eg. posterior burns)

RELATIVE CONTRAINDICATIONS:

- Inadequate staff to perform procedure safely
- Anterior intercostal catheter
- Continuous renal replacement therapy
- Intra-aortic balloon counter-pulsation
- Morbid obesity

**HAZARDS:**

- Difficult airway management and access (including ETT kinking and dislodgement)
- Accidental removal of invasive catheters (and possible occult haemorrhage)
- Obstruction or disconnection of abdominal / thoracic drains
- Pressure necrosis, pressure neuropraxia and blindness
- Labour intensive procedure-distraction from other patients

**REFERENCE:**


**HYPERBARIC OXYGEN THERAPY (HBO) IN CRITICALLY ILL PATIENTS**

Hyperbaric oxygen treatment is indicated in significant air embolism and severe decompression illness. It may be indicated in carbon monoxide poisoning and clostridial soft tissue infections. Hyperbaric oxygen therapy referral is accessed via 0800 4337 111, 021 915462, or the operator at Waitemata District Health Board.

Even in patients where HBO may be indicated, the isolated nature of the facility and the unfamiliarity of our staff with the hyperbaric environment make this mode of therapy less feasible.

Unintubated, non-ICU patients can be referred by their managing services to the Hyperbaric Unit as above and to North Shore Hospital or Auckland Hospital for subsequent management.

**REFERENCE:**

Weaver LK et al. NEJM 2002; 347:1057.
VENTILATION MECHANICS

TIDAL VOLUMES

- Traditionally an average tidal volume of approximately 10-15 ml/kg was used. The ARDS-net trial suggested 6 mLs/kg ideal body weight was the preferred strategy in those patients with acute lung injury or ARDS. Limited evidence supports this target in other patient groups.

RESPIRATORY FREQUENCY

The respiratory frequency required in an adult varies between 8 and 25 inflations per minute and depends on the patient’s expiratory time and lung compliance.

PEAK INSPIRATORY AIRWAY PRESSURE

Generally this should be set on the ventilator at or below 35 - 40 cmH2O to reduce side-effects of barotrauma. Plateau pressure is probably a more reliable guide to risk of barotrauma than peak pressure and should not be more than 32 in the short term or preferably not more than 28 for more than a few hours.

POSITIVE END EXPIRATORY PRESSURE

PEEP is defined as the pressure above atmospheric maintained at the airway at the end of expiration. It is a supportive technique used to increase arterial oxygen content without increasing the FIO2 and maintain alveolar / small airways recruitment.

USING PEEP

- PEEP may be indicated in patients with pulmonary oedema of cardiogenic or non cardiogenic origin
- The usual range of applied PEEP varies anywhere from 5 to 15 cmH2O and rarely up to 20

PROBLEMS WITH USING PEEP

PEEP is generally contraindicated in patients with a bronchopleural fistula, or severe barotrauma as it may further predispose to barotrauma including mediastinal air leak and pneumothorax.

PEEP may also:

- Increase physiological dead-space
- Reduce the capacity to excrete carbon dioxide
- Reduce cardiac output: Due in part to a decrease in venous return and an increase in alveolar and therefore pulmonary blood pressure, ie an increase in Right Ventricular afterload, and alteration of left ventricular geometry (intraventricular septum shifted towards the left)
- PEEP can be of benefit in the setting of LV failure
- Other complications may include a decrease in renal blood flow and possibly a reduction in portal blood flow
DEFAULT VENTILATOR SETTINGS, AND PRINCIPLES IN OPTIMIZING VENTILATION IN ICU PATIENTS

Where there is no reason to expect mechanical ventilation will be complex the following settings should be chosen:

**MODE**

SIMV, volume control.

**OXYGEN**

Titrate delivered oxygen to provide an oxygen saturation > 90% (an equivalent arterial PO2 of > 60 mmHg), unless otherwise specified by the Duty Intensivist.

**TIDAL VOLUME**

The ARDS-net trial suggested 6-8 ml per kilogram ideal body weight per breath (450-550 ml per delivered breath in a 60 kg woman, 500-650 ml in a 70 kg male). There is no ‘safe’ airway pressure with IPPV, therefore aim for the lowest possible pressure, but certainly under 35cmH2O.

**REFERENCE:**


**RESPIRATORY RATE**

10 to 25 breaths per minute adjusted to an arterial blood gas P_{a}CO_{2} in the normal range or approximating pre-morbid level.

Rapid ventilatory rates are not appropriate in patients with prolonged expiratory phase (ie Acute status asthmaticus: 5-8 breaths per minute adequate and perhaps desirable, allowing for prolonged expiratory times).

In some patients attempting to normalize arterial carbon dioxide content may expose the patient to the risk of barotrauma and / or volu-trauma. It is important that the Duty Intensivist is notified if this is suspected.
PEEP

As a guideline, PEEP may be applied using the normogram below, titrated to arterial oxygen content:

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<thead>
<tr>
<th>( F_1O_2 )</th>
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<tr>
<td>PEEP</td>
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<td>12</td>
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SPONTANEOUS MODE OF VENTILATION

MODE

“Pressure support”

SUPPORT LEVEL

Titrate level of pressure support 5-15 cmH\(_2\)O to achieve acceptable tidal volume (see above) and respiratory rate below 30 breaths per minute (preferably < 25 / min).

PEEP

5-12 cmH\(_2\)O according to \( F_1O_2 \), see chart above.

POSITIVE PRESSURE VENTILATION AND HYPOTENSION

Positive pressure ventilation may exacerbate or induce hypotension by increasing relative intrathoracic pressure and therefore decreasing venous return to the heart. ie:

- Mild – moderate: Loss of negative phase of inspiration and initiation of PEEP
- Extreme: Excessive increase in intrathoracic pressure (auto-PEEP or tension pneumothorax)

WEANING FROM MECHANICAL VENTILATION

INTRODUCTION

In many patients, especially those requiring short-term support, mechanical ventilation can be removed quickly and easily. In more complex cases however considerable difficulty may be encountered.

The actuarial risk of nosocomial pneumonia increases by about 1\(^{st}\) per each day of MV, being 6.5\(^{th}\) at 10 days and 19\(^{th}\) at 20 days. It is crucial to discontinue ventilatory support and extubate at the earliest time that a patient can sustain spontaneous ventilation safely. Planning for weaning should start as soon as the patient is intubated, using the following parameters:

- How long can we expect this patient to require mechanical ventilation (MV)? Is a tracheostomy likely to be needed?
• What is the underlying disease process and how may this impact on weaning?
• Premature attempts at weaning can result in respiratory muscle fatigue and atelectasis
• Premature extubation with resultant reintubation carries an appreciable risk to the patient

In general ventilatory patterns should not be reduced overnight unless there is an agreed plan to do so. All changes made to ventilator settings must be documented and clearly communicated to the bedside staff.

**PREDICTING SUCCESSFUL WEANING FROM MECHANICAL VENTILATION**

In a small percentage of patients, there may be some doubt as to whether the patient will cope with removal of respiratory support despite meeting the above criteria. A number of parameters have been studied, however at present a “spontaneous breathing trial is considered the most useful, with a positive predictive value of about 80%.

**REDUCTION OF VENTILATORY RATE**

Consideration should be given to reducing the controlled rate to the lowest that can be safely tolerated from as early in the course of ventilatory management as is possible. Spontaneous breaths are supported by pressure support at the lowest level consistent with adequate tidal volume and observed work of breathing, usually as evidenced by the spontaneous respiratory rate.

**ASSISTED SPONTANEOUS BREATHING (ASB)**

Many patients will be able to have their controlled rate reduced to zero before they are otherwise able to be extubated. This is desirable and in general should be considered if the following parameters are achieved:

• \( F_{O_2} \leq 0.5 \) and \( PEEP \leq 12cm \text{ H}_2\text{O} \) and \( PEEP \) and \( F_{O_2} \) values are otherwise trending downwards
• Spontaneous breathing effort is present
• Haemodynamic stability exists
• All spontaneous breaths should be assisted by at least 5cm H\(_2\)O pressure support

**REDUCTION OF INSPIRED OXYGEN LEVEL AND PEEP**

The inspired oxygen level should be progressively titrated to maintain saturations in the range of 90 to 95%.

In general the established level of PEEP is not reduced until such time as the inspired oxygen has been reduced to 50%. The level of PEEP is then reduced in aliquots of 2.5 to 5cm H\(_2\)O in conjunction with the inspired oxygen level providing that the saturations remain in the range of 90 to 95%. The above table provides a general guide to levels of PEEP associated with varying inspired oxygen concentrations.

**SPONTANEOUS BREATHING TRIAL**

The patient should receive no more than a PEEP of 5 cmH\(_2\)O through a T-piece system. Generally if an SBT is conducted while on the ventilator, no more pressure support than is sufficient to overcome “system” resistance to flow should be allowed (see ETT compensation mode on newer generation Puritan-Bennett)
MARKERS OF SUCCESSFUL SPONTANEOUS BREATHING TRIAL

Objective

- Gas exchange acceptable (Oxygen sat > 90%, \( P_{O_2} > 50 – 60 \text{ mmHg} \), Increase in \( P_{\text{CO}_2} < 10 \text{ mmHg} \))
- Stable ventilatory pattern (RR < 30 – 35 / min, RR not changed > 50%)
- Haemodynamically stable

Subjective

- No onset or worsening of discomfort
- Diaphoresis
- Clinical evidence increased work of breathing

REFERENCE:


CHOOSEN MODE OF WEANING TO EXTUBATE

There is no evidence that a trial of unsupported breathing using a T-piece apparatus is any better or worse than decremental levels of pressure support ventilation. Both may be used in The Waikato Hospital ICU prior to planned extubation.

The duration of the trials is not defined but those that fail usually do so early on. Probably 30 minutes to two hours is all that is needed.

CLINICAL SIGNS OF FAILURE INCLUDE

- Tachypnoea
- Tachycardia
- Hypertension
- Obtundation
- Desaturation

As breathing through an artificial airway increases the work of breathing a successful trial of spontaneous breathing should be terminated at 30 or 120 minutes. This may reduce the chance of ongoing fatigue contributing to a “failed extubation” later in the day.

FACTORS THAT INFLUENCE SUCCESS OF WEANING (WEANING FAILURE)

- Weakness
- Critical illness weakness (polyneuropathy, myopathy)
- Cord injury
- Drug induced weakness from steroids, statins, aminoglycosides
- Electrolyte abnormalities (Hypokalaemia, hypomagnesaemia, hypophosphataemia, metabolic alkalosis)
- Fever, sepsis
- Low Pulmonary or Chest Wall Compliance
- Stiff lungs from pulmonary oedema, inflammation, interstitial lung disease
- Pleural effusions, pneumothorax
- Mechanical disruption of ribs / diaphragm
- Intra-abdominal hypertension - Obesity, abdominal distension
- Airflow obstruction
- Mucus plugging
- Acute bronchospasm
- Anaphylaxis
- Blocked ETT or tracheostomy tube
- Central Nervous System Conditions
- Acute Delerium (eg. drug induced, encephalopathy, encephalitis)
- Psychiatric
- Pain
- Brain injury
- Excess respiratory depressant drugs
- Environmental

**EXTUBATION**

Requirements for extubation:

- Improving clinical condition.
- Patient stable on FIO₂ < 0.4 with a P₉O₂ > 60 mmHg and/or sats >90%
- Assisted spontaneous breathing with PEEP < 5-8 cmH₂O
- The spontaneous respiratory rate is less than 30/min
- Acceptable neurological state: ie the expectation exists that the patient will be awake enough to protect their airway, and have the local ability (intact cranial nerve function or tracheostomy) to do so
- Haemodynamically stable
- No prospect of major intervention planned in the ensuing 24 hours
NON-INVASIVE VENTILATION (NIV)

Strict control of application of NIV exists for HDU Patients- see relevant procedure on Intranet for current version.

Mechanical ventilation not requiring endotracheal intubation may avoid many of the complications of invasive ventilation (ie. generally associated with less ICU acquired infection, results in shorter ICU and hospital length of stay, and may be more acceptable to patients. Appropriate patient selection is important (restrict to indications below), as is appropriate monitoring and a controlled environment with the capacity to initiate invasive ventilation without delay where necessary.

MODES

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)
- Single continuous positive airway pressure
- No augmentation of tidal volume
- Generally seen to be useful in hypoxic states

BIPHASIC POSITIVE AIRWAY PRESSURE (BIPAP)
Usually PEEP plus augmentation of tidal volume. Useful in the treatment of acute hypercarbic states.

This section does not attempt to address the role of NIV outside of acute conditions. The groups outlined below are those that have been studied, often in a limited way.

As the technology improves and the area is studied more extensively, previous absolute contra-indications (Hypoxic respiratory failure, acute severe asthma, respiratory distress post-extubation) have become relative. The same pitfalls exist for these conditions and clinical experience helps inform individual therapy. NIV should still be consultant led therapy.

ACCEPTED INDICATIONS
- Acute exacerbations COAD: Good evidence that NIV useful in acutely hypercapnoeic patients (ie pH <7.3)
- Pulmonary oedema
- Patients with underlying neuromuscular, parenchymal or restrictive lung disease: NIPPV useful only if decompensation is a result of reversible infection and not disease progression
- Immunocompromised patients

LESS ACCEPTED INDICATIONS
- NIV should only be applied in the following situations on a trial basis, with well defined end points, and in a well controlled environment (ICU generally). The duty Intensivist should be made aware of these decisions in real time
- Weaning or early discontinuation of invasive ventilation
- Stable airway obstruction (eg: post operative patient with obstructive sleep apnoea)
- Pneumonia or ARDS
• Asthma

**CONTRA-INDICATIONS TO NIV**

- The patient with impaired level of consciousness (including those that are in-extremis)
  - In setting of CO2 narcosis with COPD a short trial of NIV with a clear rapid decrease in arterial pCO₂ may be reasonable, but this should be a consultant led decision, with clear documentation of what constitutes success and failure of this therapy
- Haemodynamic instability
- Bowel obstruction or upper GI haemorrhage (increased risk of aspiration)
- Agitation such that mask not tolerated well
- Impaired cough, including low GCS and bulbar dysfunction
- Non-reversible disease process
- Untreated pneumothorax

**PRE-REQUISITES**

- The patient must be able to protect their own airway sufficiently
- The patient must be accepting of the face mask
- There must be a reversible problem requiring “bridging” respiratory support
- There must be adequate monitoring
- Continuous pulse oximetry, telemetry and at least intermittent blood pressure and ABG recording
- Nursing ratio no worse than 1:2

**COMPLICATIONS**

- Aerophagia or gastric distension - Aspiration lung injury
- Mask intolerance and heightened anxiety
- Pressure necrosis of the face

**REFERENCE:**


**PLEURAL PROCEDURES**

**INDICATIONS FOR ACCESSING PLEURAL SPACE**

- Pneumothorax (? temporising procedure if under tension)
- Haemothorax
- Symptomatic or infected pleural effusion

**NEEDLE THORACOSTOMY FOR TENSION PNEUMOTHORAX**

- 16G cannula placed in mid clavicular line, 2nd intercostal space
• Tension is a clinical diagnosis. Do not delay to obtain chest X-ray
• Proceed to formal intercostal drain insertion

PLEUROCENTESIS

INDICATIONS
Diagnostic procedure.

Therapeutic procedure: Drainage of an infected collection requires an underwater seal drain. It may not be appropriate to perform “once-off” drainage. The practice of draining non-infected pleural collections by pleurocentesis is controversial and should not be performed without direction by the Duty Intensivist.

TECHNIQUE
Local anaesthesia and sterile technique.

Unless the fluid collection is grossly detectable on clinical examination and on plain radiology, pleurocentesis should be ultrasound directed.

INVESTIGATION OF PLEUROCENTESIS FLUID
Aspirated fluid should, at the very least, be submitted for:

• pH: analysed in Laboratory (NOT ICU) blood gas analyser (pH < 7.20 = empyema, 7.20-7.25 = equivocal). Send sample on ice to laboratory
• Protein (<25g/L transudate, >35g/L exudate. Ratio of pleural fluid protein to serum protein greater than 0.5 = exudate
• LDH (Pleural fluid LDH is greater than 0.6 or ? times the normal upper limit for serum
• Cell counts and MC&S

INTERCOSTAL CATHETER / UNDERWATER SEALED DRAIN

INSERTION
• Local Anaesthesia is mandatory in awake patients, and should be used in sedated patients
• Strict aseptic technique
• 28F catheter inserted into 3-4th intercostal space, mid-axillary line (within “triangle of safety”), using blunt dissection as described and recommended in the ATLS guidelines
• The catheter must be guided through the ribs without use of sharp instruments (preferably finger). Trochar aided insertion techniques are not acceptable
• Chest drains must be secured with both sutures and tape
MAINTENANCE

- Drains placed in un-sterile environs should be removed as soon as possible
- Drains should remain in-situ until radiological resolution has occurred and there is no further bubbling or drainage of significance (< 150 ml / 24hrs)
- Drains placed electively in theatre are the responsibility of the surgeon

COMPLICATIONS

- Incorrect placement
- Pulmonary laceration
- Pneumothorax
- Bleeding as a result traumatic drain insertion (intercostal or, lateral thoracic artery, lung etc)
- Empyema

NASOJEJUNAL TUBE INSERTION

INDICATIONS

- Instillation of feed into the jejunum is an effective way of feeding patients with:
  - Prolonged gastric stasis ( > 3 days)
  - Gastric stasis resistant to treatment with pro-kinetic agents (erythromycin, metoclopramide)
  - Pancreatitis or other scenario’s where feeding distal to the duodenum is desired

PROCEDURE

This is most frequently performed endoscopically by a gastroenterologist

COMPLICATIONS

- Endobronchial placement
- Other ectopic placement
- Migration, kinking or knotting
INTRA-ABDOMINAL PRESSURE MANOMETRY

POLICY

Renal perfusion pressure may be compromised by raised intra-abdominal pressure following:

- Surgery
- Trauma
- Intra-abdominal pathology (e.g., pancreatitis)

The occurrence of acute renal failure in an intensive care patient significantly increases the risk of adverse outcome. The measurement of intra-abdominal pressures in patients that are at risk of developing abdominal compartment syndrome may allow renal salvage in patients where there is a remedial cause. A measured pressure of > 20 mmHg (referenced to the symphysis pubis) may precipitate acute abdominal compartment syndrome and renal failure.

PROCEDURE

- Connect a 100ml bag of saline to a “metriset” which is then connected to a manometer. A 20G needle is then attached to the manometer tubing.
- Place patient supine.
- Empty bladder.
- Clamp indwelling catheter distal to the culture aspiration point. Clean aspiration point with an alcohol swab and insert 20G needle (prepared as above).
- Inject 100 ml warmed sterile saline into patient’s bladder.
- Open stopcock to transducer and allow 30 seconds to equilibrate.
- Once pressure measurement completed, remove 20G needle from aspiration point, unclamp urinary catheter and allow free drainage of the bladder.

COMPLICATIONS

- Instillation of bacteria into the bladder.
- Triggering autonomic dysfunction (NB vagal) on injecting into the bladder, particularly if the bladder is incompletely drained.
- Patient discomfort (if awake).
- Artificially elevated readings due to bladder spasm or local pelvic haemorrhage may precipitate interventions that are associated with significant morbidity.
VASCULAR ACCESS

ARTERIAL CANNULAE

INDICATIONS

• Invasive measurement of systemic blood pressure

• Multiple blood gas sampling and laboratory analysis

SITE AND CATHETER CHOICE

• 1st choice: Radial

• 2nd choice: Femoral - Doralis pedis may also be considered

• Site of choice for PiCCO catheter monitoring (Pulsiocath 5F 16 cm catheter) is generally the femoral artery

The axillary artery may be considered (usually 4F catheter for PICCO monitoring, 3F for blood pressure monitoring). The introduction of air into the circulation, especially from the axillary artery risks a cerebral vascular event.

The Brachial artery is an end-artery, and catheterisation has been considered a risk for distal arterial complication (although this has also been disputed). It may be used if there are no alternatives.

TECHNIQUE

All catheters should be inserted with full sterile technique (gown, sterile gloves, topical antiseptic). The arterial line must be firmly anchored. The insertion site and all connectors must be visible through the applied dressing.

COMPLICATIONS

• Infection

• Thrombosis

• Digital Ischaemia

• Vessel trauma and fistula formation.

PERIPHERAL IV CATHETERS

INDICATIONS

• Initial IV access for resuscitation
• Stable or convalescent patients, where more invasive access is not warranted

**MANAGEMENT**

All lines placed in situations where aseptic technique was not followed must be removed (eg. Placement by emergency staff at the roadside). Acceptable aseptic technique must be followed including:

- Thorough hand-washing
- Skin preparation with alcohol swab
- Occlusive but transparent dressing

All lines should be removed if not being actively used, or if > 2 days old. An exception may be made where venous access is challenging (eg. paediatric patients).

**COMPLICATIONS**

- Infection
- Thrombosis
- Extravasation

**CENTRAL VENOUS CANNULAE**

**TRAINING**

Central venous catheterisation may not be attempted by any member of staff without adequate training or supervision. The unit only stocks antibiotic impregnated catheters, both 3 and 5 lumen.

**INDICATIONS**

- Reliable IV access in ICU patients
- Fluid administration
- TPN, hypertonic solutions (amiodarone, nimodipine)
- Infusions of inotropes or other vasoactive substances
- Monitoring of right heart pressures (CVP, Pulmonary Artery Catheter)
- Access for renal replacement therapy
- Large bore resuscitation catheter: PA sheath or dialysis catheter
**TECHNIQUE**

All staff are expected to view and familiarize themselves with insertion techniques as described in standard texts. All procedures must be performed under STRICT sterile conditions – thorough hand washing, gloves, gown, hat, mask, whole body drape, USS probe cover and chlorhexidine with 70% alcohol skin prep. Where a junior member of staff is familiar with a certain technique, they should continue to use that technique.

If you suspect that you have mistakenly cannulated an artery rather than a vein, seek assistance from the senior Registrar or Duty Intensivist prior to removing the offending line.

**CHOICE OF ROUTE**

The internal jugular route represents less risk than subclavian in un-practised hands. Subclavian catheterisation may be the route of choice from an infective risk perspective, followed by internal jugular and then femoral. Each site has characteristics that make it preferable under certain circumstances and where the operator is in any doubt this should be discussed with senior staff members.

**ULTRASOUND**

Ultrasound is now used for all central lines. It can also be useful for arterial cannulation and peripheral vein cannulation. It is important visualize the tip of the needle, hopefully seeing it tent the vessel as the vein is punctured. As the ultrasound is a two dimensional image it is possible for the tip of the needle to be well outside the 2-D plane causing damage to other structures.

**SUBCLAVIAN**

Avoid in situations where pneumothorax would be fatal. (eg. severe respiratory failure, lung hyperinflation). Avoid in patients therapeutically anticoagulated or coagulopathic, Platelets <50, INR >2, APTT >50. It may be appropriate to attempt to reverse abnormal clotting prior to insertion of a CV catheter; however this should be discussed with the Duty Intensivist.

**Always choose side of chest that is least effective for ventilation, or in which there is already an intercostal catheter**

Ultrasound imaging of the subclavian vein is difficult. One approach is to cannulate the proximal section of the axillary vein as it joins the subclavian. Another approach is to position the ultrasound probe above the clavicle aiming caudad to see the needle approach from under the clavicle.

**INTERNAL JUGULAR**

This route is associated with a higher risk of infection than subclavian access. However there is a lower risk of pleural puncture (for high jugular approach) and better control of haemorrhage than the subclavian approach. Internal Jugular approach is the route of choice for dialysis catheter insertion, although femoral access is also acceptable. The safety of jugular puncture is improved with ultra-sound guidance.

**EXTERNAL JUGULAR**

In certain circumstances it may be appropriate to attempt cannulation of the external jugular vein in order to achieve central venous access. This route may be advantageous when the patient is coagulopathic, or in
certain emergency situations where other access may be difficult. This route has the lowest rate of complications, but is associated with a 20% failure rate due to inability to cannulate vein or malposition.

EJ access is less suitable for PA Catheter or Dialysis catheter insertion.

FEMORAL

Femoral catheterisation has traditionally been thought to confer a high risk of infection relative to subclavian access. This has not been proven, although in certain patients (eg: the obese, or those with infected/open abdominal wounds) this may still hold true. The incidence of thrombosis is probably similar to other sites. Good flow characteristics for dialysis catheters, using a 20 cm or longer catheter.

Relatively low risk route for inexperienced operators in high risk patients (i.e. uncorrected coagulopathy, severe respiratory failure). This vein is a safer choice if you are asked to place a CVL urgently where ultrasound is not readily available, patient retrieval for example. If the back of the vein is punctured during insertion it is preferable that this is distal to the inguinal ligament or a retroperitoneal haematoma may develop.

REFERENCE:


LINE MANAGEMENT

Routine line replacement is not required.

Non-antibiotic coated lines and central lines inserted in other hospitals should be changed within 24 hours of admission to our ICU.

Lines should be removed as soon as:

- They are not required any longer
- The patient has evidence of unexplained systemic infection (pyrexia, ?WCC). Take a set of blood cultures from the line and a peripheral site at the same time
- Insertion site infection or positive blood culture with likely organism (Staph epidermidis)
- Guide-wire exchanges should not be performed unless discussed with the Duty Intensivist

COMPLICATIONS

- AT INSERTION
  - Arterial puncture
  - Pneumothorax
  - Neural injury (phrenic nerve, brachial plexus, femoral nerve, cervical plexus)
  - Guidewire induced atrial ectopy, arrhythmia
• **DURING CATHETER PRESENCE:**

  • Infection: Infection risk increased with increased catheter size, choice of site (femoral > jugular > subclavian) and use of TPN or dextrose containing fluids
  
  • Thrombosis
  
  • Embolism
  
  • Pulmonary infarct or pulmonary arterial rupture (PA Catheter)

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**THE PULMONARY ARTERY CATHETER**

The PAC is used to measure hemodynamic variables (flow and pressure) within the right heart and pulmonary vasculature.

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**MEASURED VARIABLES**

- Cardiac Output (using the average of three thermodilution measurements, discarding measurements with >10% variance)
- Central Venous Pressure (CVP)
- Pulmonary Artery Pressure (PASP, PADP, PAM)
- Pulmonary Artery Wedge Pressure (PAWP, a surrogate for left ventricular preload under specific conditions)
- Core temperature
- Mixed venous oxygen saturation (SvO2, not routinely used in our ICU)

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**CALCULATED VARIABLES**

Calculated variables use values gained from direct measurement and are therefore susceptible to measurement errors (see Useful Equations Index).

- Cardiac Index (CI)
- Stroke Volume/Stroke Volume Index (SV, SVI)
- Systemic Vascular Resistance/Systemic Vascular Resistance Index (SVR, SVRI)
- Pulmonary Vascular Resistance/Pulmonary Vascular Resistance Index (PVR, PVRI)
CLINICAL USES

Therapeutic use of the PAC is controversial and widespread use has dwindled in the absence of clear evidence to support the use of the PAC to predict fluid responsiveness and guide the use of vasoactive medications in critically ill patients with hemodynamic instability.

Understanding of the catheter’s limitations and usefulness varies widely among doctors and nursing staff and requires ongoing education to reduce morbidity associated with its use, and correct interpretation of the data it provides.

In our ICU the PAC is most commonly used in post-cardiac surgical patients (indications include right ventricular dysfunction, pulmonary hypertension, IABP or LVAD, severe left ventricular dysfunction with EF<30%, acute VSD).

The PAC can aid diagnosis:

- Differentiating the origin of shock
- Differentiating the origin of pulmonary oedema (cardiogenic, non-cardiogenic)
- Differentiating the origin of pulmonary hypertension (pre-capillary, post-capillary)
- Diagnosing cardiac tamponade
- Diagnosing left to right shunt

INSERTION

Under sterile conditions the PAC is inserted via a wide bore introducer sited in a large central vein (RIJ provides the most direct route). With the distal port transduced and the balloon inflated, the catheter tip is floated through the right atrium and right ventricle into the pulmonary trunk. Floating the PAC requires understanding of the characteristic waveforms encountered as the catheter tip floats through the right heart into the pulmonary artery. Once in position the catheter tip can be intermittently wedged within one of the pulmonary arteries by temporary balloon inflation to create a continuous column of blood between the pulmonary artery and left atrium.

SWAN-GANZ-HAEMODYNAMIC WAVEFORMS
MONITORING THE PAC TRACE

The PAC trace should be visible on the monitor at all times.

Waveform features can be used to differentiate between a right ventricular (RV) and pulmonary artery (PA) trace:

- RV waveform has a symmetrical steep upstroke and steep downstroke. A small rise in pressure (caused by ejection of blood into the RV during atrial contraction) precedes the steep systolic upstroke.
- PA waveform is similar to the arterial pressure waveform with a rapid systolic upstroke and a more gradual downstroke. The downstroke has a dicrotic notch which indicates closure of the pulmonary valve.

A damped trace may represent:

- Wedged catheter (ensure balloon is deflated and withdraw catheter a couple of centimetres until PA trace is visible)
- Clot at the catheter tip (flush distal port generously)
- Kinking of the PAC as it exits the introducer (withdraw introducer slightly)
- Equipment error (check transducer and monitor)

COMPLICATIONS OF PAC

Complications may occur at the time of insertion or while the PAC remains insitu.

- Time spent inserting the catheter may distract from resuscitating the patient
- Complications associated with standard CVL insertion (bleeding, arterial cannulation, pneumothorax, lost wires, infection)
- Arrhythmia (risk of inducing RBB in 5% of patients during flotation resulting in CHB if pre-existing LBBB)
- Catheter knotting
- Perforation/Pulmonary Artery rupture
- Pulmonary/Tricuspid Valve damage
- Air embolism
- Thrombus
- Endocarditis
- Pulmonary infarction
- Misinterpretation or misuse of data
**SPECIAL POINTS**

The PAC should only be inserted by medical staff trained in the use of the PAC, after discussion with the Duty Intensivist.

The PAC should only be manipulated or the balloon inflated by medical and nursing staff who are trained and familiar with its use (infrequent use has resulted in reduced familiarity).

The PAC should NEVER be withdrawn with the balloon inflated.

Wedge pressures should only be performed at the request of the Duty Intensivist (Pulmonary Artery Diastolic Pressure can be used as a surrogate for PAWP in patients without pulmonary hypertension).

Pressures should be referenced to the mid-axillary line.

**REFERENCES**


**TRANSPULMONARY THERMODILUTION (PICCO, VOLUME VIEW)**

Both the PiCCO (Pulse Contour Cardiac Output) and Edwards Volume View (EV1000) combine continuous pulse contour analysis with intermittent transpulmonary thermodilution, as less invasive alternatives to PAC for hemodynamic monitoring.

**INDICATIONS**

Transpulmonary thermodilution is the first choice for cardiac output monitoring of hemodynamically unstable critically ill patients in our ICU.

It is useful when PAC is contraindicated and/or monitoring of pulmonary artery pressures is not required.

**COMPONENTS**

- A standard CVL (specialized PiCCO or Volume View injectate temperature sensor attaches to distal lumen) inserted under sterile conditions into a large central vein

- Specialized PiCCO or Volume View thermistor-tipped arterial line inserted under sterile conditions into a large proximal artery (usually femoral but axillary and brachial arteries can be used)

- Specialized arterial transducer (PiCCO or Volume View)

- Specialized central processor/electronic display (PiCCO monitor or EV1000)
MEASURED VARIABLES

- Cardiac Output (CO, measured using intermittent transpulmonary thermodilution where a bolus of thermal indicator is injected into the distal lumen of the CVL travels through the lungs to the thermistor located on the tip of the specialized arterial line. The modified Stewart Hamilton equation is used to calculate cardiac output using the thermodilution curve)
- Arterial Blood Pressure
- Core Temperature

CALCULATED VARIABLES

Volumetric parameters are calculated by advanced analysis (extrapolation and log transformation) of the slope and duration of the thermodilution curve.

This assumes a model of circulation in which the thermal indicator solution undergoes mixing in a series of chambers (intra-thoracic thermal volume or ITTV).

- The mean transit time (MTT) of the indicator is the time taken for half the thermal indicator to pass the thermistor and is proportional to the total intra-thoracic thermal volume
The exponential decay time (EDT) is proportional to the volume of distribution of the largest chamber (pulmonary thermal volume or PTV)

- \( \text{PTV} = \text{EDT} \times \text{CO} \)

Global End Diastolic Volume (GEDV) is the volume of blood in all 4 chambers of the heart at end-diastole

Intra-thoracic Blood Volume (ITBV) is the volume of blood in all 4 chambers of the heart at end-diastole (GEDV) plus the volume of blood in the pulmonary circulation

Extravascular Lung Water (EVLW) is the water content outside the pulmonary vasculature (pulmonary interstitium and alveolar fluid) and can be used to quantify pulmonary oedema although the clinical benefit of this is unproven

Pulmonary Vascular Permeability Index (PVPI) is used to help differentiate between causes of pulmonary oedema. PVPI is increased with increased pulmonary permeability due to lung injury and normal in patients with cardiogenic pulmonary oedema or increased hydrostatic pressure

Cardiac Function Index (CFI) and Global Ejection Fraction (GEF) are derived values that have some correlation with cardiac function

Together GEDV and/or ITBV are used to predict preload and volume responsiveness and guide fluid administration and use of vasoactive medications in hemodynamically unstable patients (see Decision Model).

**PULSE CONTOUR ANALYSIS**

Pulse Contour Analysis requires calibration by thermodilution (this determines a calibration factor for continuous cardiac output monitoring). The following variables can then be calculated:

- Cardiac Output/Cardiac Index (CO/CI) is measured using characteristics of the arterial pressure waveform. Cardiac output is dependent on stroke volume, aortic impedance and a calibration factor (aortic impedance and calibration factor are measured during transpulmonary thermodilution)

- Stroke Volume (SV)

- Systemic Vascular Resistance (SVR, uses cardiac output measured by transpulmonary thermodilution)

- Pulse Pressure Variation (PPV) is cyclical change in pulse pressure over the respiratory cycle

- Stroke Volume Variation (SVV) is the cyclical change in stroke volume with respiration

- Left Ventricular Contractile Index (LVCI) is derived from the maximal upslope of the arterial pressure time curve in systole (role not established)
USEFUL FORMULAS AND NORMAL VALUES

<table>
<thead>
<tr>
<th>Variable</th>
<th>Formula</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global End Diastolic Volume</td>
<td>GEDV = ITTV - PTV</td>
<td>600-800 ml/m²</td>
</tr>
<tr>
<td>Intra-thoracic Blood Volume</td>
<td>ITBV = GEDV x 1.25</td>
<td>850-1000 ml/m²</td>
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<tr>
<td>Extravascular Lung Water</td>
<td>EVLW = ITTV - ITBV</td>
<td>3-7 ml/kg</td>
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<tr>
<td>Pulmonary Vascular Permeability Index</td>
<td>PVPI = EVLW/GEDV x0.25</td>
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<tr>
<td>Systemic Vascular Resistance Index</td>
<td>SVRI = MAP x CO/BSA</td>
<td>1700-2400 dyn/s/cm²/m²</td>
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<tr>
<td>Cardiac Index</td>
<td>Pulse Contour Analysis</td>
<td>3-5 l/min/m²</td>
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<tr>
<td>Cardiac Function Index</td>
<td>CFI = CO/GEDV</td>
<td>4.5-6.5 1/min</td>
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<tr>
<td>Global Ejection Fraction</td>
<td>GEF = (SV/GEDV) x4</td>
<td>25-35%</td>
</tr>
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</table>

ACCURACY OF TRANSPULMONARY THERMODILUTION

Transpulmonary thermodilution correlates well with cardiac output measurements from the PAC. The presence of a large aortic aneurysm results in over estimation of ITBV and GEDV. Intra-cardiac shunts, rapid changes in body temperature and aortic regurgitation will affect the accuracy. Injectate administered through a CVL located in the femoral vein adjacent to a femoral PICCO will affect the accuracy of transpulmonary thermodilution (proximity to thermistor). EVLW is under-estimated in obese patients and post-pneumonectomy.

Transpulmonary thermodilution remains useful in the presence of an intra-aortic balloon pump (IABP). Use of a CVL located in the SVC is preferable (the accuracy of transpulmonary thermodilution and calculated volumetric variables is affected by use of a femoral CVL for injectate).

ACCURACY OF PULSE CONTOUR ANALYSIS

Accurate Pulse Contour Analysis relies on an optimal arterial waveform therefore is affected by over-damping, under-damping and arterial line site (ideally large proximal artery). Arrhythmia, aortic regurgitation and intra-aortic balloon pumps (IABP) will affect accuracy.

Recalibration is required every 8 hours and following sudden changes in aortic compliance (resuscitation of the hemodynamically unstable patient).

ADVANTAGES OF TRANSPULMONARY THERMODILUTION VERSUS PAC

Comparable accuracy of cardiac output determination using the PAC and transpulmonary thermodilution.

Volumetric measures of preload (ITBV and GEDV) are better at predicting fluid responsiveness and less affected by ventilation than CVP and PAWP.

Transpulmonary thermodilution is less invasive than PAC.

Transpulmonary thermodilution can remain in situ for up to 10 days (PAC use is usually limited to 3 days) although the transducer and injectate sensor need to be changed every 3-5 days.
This decision model is not obligatory. It cannot replace the individual therapeutic decisions of the treating physician.

<table>
<thead>
<tr>
<th>Measured Values</th>
<th>CL (l/min/m²)</th>
<th>&lt; 3.0</th>
<th>&gt; 3.0</th>
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<tbody>
<tr>
<td>GEDI (ml/m²) or ITBI (ml/m²)</td>
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<td>&gt; 700</td>
<td>&lt; 700</td>
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<tr>
<td>ELWI (ml/kg)</td>
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<td>&gt; 850</td>
<td>&lt; 850</td>
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<td>Therapy Options</td>
<td></td>
<td>V+?</td>
<td>Cat?</td>
</tr>
<tr>
<td>Targeted Values</td>
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<td>700-800</td>
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<td>(slow response)</td>
<td>≤ 10</td>
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V+ = volume loading  V = volume reduction  Cat = catecholamines / cardiovascular agents

*SVV is only applicable in fully ventilated patients without cardiac arrhythmia

This decision model is not obligatory. It cannot replace the individual therapeutic decisions of the treating physician.

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## PERICARDIOCENTESIS

### INDICATIONS

- Haemodynamically significant pericardial effusion
- Traumatic pericardial tamponade

### TECHNIQUE

Pericardial access and drainage may not be performed in ICU except under the most dire circumstances. Echocardiographic guidance by staff experienced is the preferred method.

Suspected cardiac tamponade in a patient who has undergone cardiac surgery is an indication for chest re-opening and not needle aspiration.
CARDIO-PULMONARY RESUSCITATION

Whilst we have little control at present over community cardiac arrests, and to a lesser extent hospital cardiac arrest, it must be stressed that vigilance and pro-active management of critically ill patients may abort a process precipitating a cardiac arrest within the ICU.

**KEY POINTS IN THE MANAGEMENT PLAN FOR AN ADULT COLLAPSE**

In adult cardiac arrest, VF / VT is the most likely rhythm, and a defibrillator the only effective treatment.

Start effective CPR as soon after the circulatory arrest as possible. Effective artificial circulation requires controlled, uninterrupted chest compression. The ratio of compressions to ventilation is 30:2 in all instances except when an ETT is in place, when it is 15:1 with no compression pause. The rate of compression is 100 / minute.

As soon as possible (especially in unmonitored patient) switch the defibrillator on and check or confirm the rhythm via the paddles.

Defibrillate as soon as possible.

**Defibrillation should take precedence over all other interventions**

Assess for, and shock, VF / pulseless VT, (200J monophasic) if necessary.

Endotracheal intubation, IV insertion and ECG electrode placement / replacement should occur between defibrillation attempts. The order of priority for these adjuncts is:

1. Secure airway (plus waveform capnography). Endotracheal intubation is the gold standard for a secure airway. Compressions should not be ceased for more than 20 seconds. An ETT increases the continuity of chest compressions after placement. Oesophageal intubation may occur up to 12% of the time. The balance of these considerations should be tailored to the patient

   **If you are not well practised in intubation early placement of an Laryngeal mask airway is advisable, especially if an intubation attempt is likely to pause chest compressions for more than twenty seconds**

2. Ventilate with 100% oxygen

3. IV / Intraosseous access and administration. Consider IO if two failed attempts at IV placement

4. Augmentation of aortic diastolic pressure should be an adjunctive goal of therapy since coronary perfusion is low during conventional CPR. Adrenaline and other alpha agonists will significantly increase aortic diastolic pressure. Administer adrenaline to maintain coronary blood flow if the second defibrillation fails. 1 mg of adrenaline every 2nd shock is an acceptable minimum. Adrenaline is given after the FIRST CYCLE in PEA arrest. Adrenaline is given after the SECOND CYCLE in pulseless VT/VF

5. If VT / VF persists after 3 shocks, give Amiodarone 300 mg IV

Consider and correct if possible any reversible causes of circulatory arrest. (see 5H’s and 5T’s on algorithm below)
During CPR, adequate ventilation is the mainstay of therapy for acid-base abnormalities. The indications for Sodium Bicarbonate are:

- Hyperkalaemia
- Tricyclic antidepressant overdose where metabolic acidosis existed prior to arrest
- Late in cardiac arrest situation (at least > 10 minutes) in intubated hyperventilated patients

Reference:
TARGETED TEMPERATURE THERAPY POST-CARDIAC ARREST

Patients subjected to therapeutic hypothermia as soon as possible following resuscitation from cardiac arrest may have a better outcome (15-25% absolute survival advantage). Following consultation with the Duty Intensivist, short term hypothermia should be induced as below. More recent studies suggest that avoidance of fever is crucial, as opposed to a therapeutic benefit of cooling to 33 degrees centigrade.

PATIENT SELECTION

Apply to patients with:

- Suspected hypoxic-ischaemic encephalopathy
- Motor score of GCS 4 or less (ie flexion to pain or worse)

COOLING GUIDELINE

- Cool as soon as possible following return of circulation
- Use up to 40ml/kg of fridge cold isotonic crystalloid if necessary
- Sedate with propofol, with intermittent muscle relaxant to ablate shivering if prominent
- Actively cool, using water cooled blanket to core temperature target of less than 36 degrees centigrade for at least 24 hours, as measured with rectal temp probe
- After at least 24 hours re-warm to 36.5 degrees centigrade. Passively is ideal and certainly no faster than 0.5 degree per hour
- Avoidance of fever (temperature > 37.5 degrees) should continue for up to 72 hours post-arrest. (Antipyretics, cooling fans etc)
- Conduct sedative free neurological assessment
- Progress to somato-sensory evoked potentials if awakening slow or absent

RAPID COOLING

Rapid onset cooling can be achieved by administering up to 40ml / kg of Ringers lactate or other isotonic crystalloid, cooled to 4 degree centigrade, over a 30 minute period. Rapid cooling has not been shown to beneficial in the most recent trials.

REFERENCE:

WITHDRAWAL OF TREATMENT IN THE INTENSIVE CARE

Withdrawal of treatment, or the decision not to initiate treatment, is a consultant responsibility.

Resident staff are not expected, nor encouraged, to begin an end-of-life discussion with a patient or their family unless on the instruction of the Duty Intensivist.

Patient and family may initiate such a discussion which may involve RMOs after hours. If it is necessary to address this after hours the Consultant should be contacted.

PRINCIPLES

Patients have a right to receive quality end of life care including appropriate palliative care and help making decisions regarding life-sustaining treatment.

Health providers are not however obliged to provide treatments that would be perceived to be futile, or otherwise not in the best interests of a given patient.

DECIDING NOT TO TREAT (OR TREAT ANY FURTHER)

The goal of intensive care is to prevent unnecessary suffering and premature death by treating reversible illnesses for an appropriate period of time.

Patients in whom treatment is to be withdrawn or not initiated generally fall into one of the following categories:

- Imminent death: A patient with an acute illness whose reversal or cure would be extremely unlikely
- Lethal condition: Progressive, unrelenting terminal disease incompatible with survival longer than 3-6 months. Life sustaining treatment should not be provided for the underlying disease. Where treatment is provided for superimposed, reversible illness, this should have clear goals and limitations
- Lack or loss of consent for intensive care treatments: patient with-holds or withdraws consent or it can be established through other means, e.g. advanced directive, reliable family member that consent for such treatment would have been with-held by the patient had they been well enough to do so

THE DECISION MAKING PROCESS

- Generally, there should be inter-professional team consensus to withdraw therapy
- The Duty Intensivist or primary specialist should
- As early as possible discuss with patients while capable, their prognosis and wishes for treatment
- Explore why the patient or substitute decision maker wishes treatment to be continued
Discuss with the patient or decision maker the rationale for withholding or withdrawing of life support systems

Describe palliative measures and emphasize patient comfort and dignity

Offer hospital resources such as social work, chaplaincy or bioethics to assist the patient / family with their psychosocial, cultural, spiritual and informational needs

Document pertinent details of this communication in the patient notes

Where there is no consensus between the patient / family and staff, then:
  - Negotiate a plan of care acceptable to all parties
  - Obtain a second opinion should this be appropriate
  - Initiate a clearly defined trial of therapy

If none of these are successful then external mediation may become necessary although this would be extremely rare

REFERENCES:

ANZICS Statement on Care and Decision-Making at the End of Life for the Critically Ill.pdf

BRAIN DEATH AND ORGAN DONATION

The activities around declaration of brain death and organ donation in general are in part codified and in part guided by the activities of Organ Donation New Zealand (ODNZ). A smartphone app available at http://moacreative.com/odnz contains most of the relevant information.

DECLARATION OF BRAIN DEATH

This procedure is an absolute requirement prior to organ donation in a beating heart donor.

Where clinical examination is to be used alone, this must be performed by 2 doctors of the status prescribed by local jurisdiction. In Waikato ICU, we take this to be the Duty Intensivist plus at the minimum, an experienced registrar (Senior Registrar and/or experienced ICU trainee).

The two doctors may choose to be present at each examination; however, each must perform ALL of the brain death studies independently, and be responsible for one of the examinations.

In some circumstances, a clinical examination may be replaced by investigations as given below.

CLINICAL CERTIFICATION OF BRAIN DEATH

as per ANZICS document available at:


PRE-CONDITIONS

- A cause of coma that is consistent with brain death must be identified and documented
- Reversible causes of coma must be excluded:
  - Coma caused/contributed to by drugs / poisons-Morphine, Midazolam, barbiturates etc.
  - Unresponsive state caused by neuromuscular blocking agents - vecuronium, pancuronium etc.
  - Coma caused by/contributed to hypothermia - core temperature must be ? 35o C
  - Coma caused by/contributed to metabolic or endocrine disturbance

A minimum of four hours observation (24 hours for hypoxic-ischaemic injury) with pre-conditions met and, during which the patient has been comatose (Glasgow Coma Score 3), had non-reactive pupils, absent cough and gag reflexes, and no spontaneous breathing efforts
CLINICAL ASSESSMENT OF BRAIN FUNCTION

It is recommended that this procedure is performed separately by 2 doctors in separate examinations. Thus brain death is not confirmed until a minimum of 4 (but practically speaking usually 5-6 ) hours by the time 2 tests have been performed) after onset of coma (or 24 hours if due to hypoxic - ischaemic brain insult).

TESTING BRAIN FUNCTION

A response due to cranial nerve stimulation at any stage deems the patient is not brain dead and further testing does not proceed. Access to at least one eye and one ear able to be meaningfully tested is required for the clinical diagnosis of brain death.

1. Absent pupillary responses to light (direct and consensual)
   tests cranial nerves II, III

2. Absent corneal reflexes
   tests cranial nerves V, VII

3. Absent vestibulo-ocular reflex: (the tympanic membrane must be inspected and noted to be intact before proceeding).- no nystagmus (no eye deviation to the stimulated side) on the injection of 50 ml of iced water into the ear
   tests cranial nerves VII, VIII

4. Absent gag reflex
   tests cranial nerves IX, X

5. Absent cough reflex
   tests cranial nerves IX, X

6. Absent response to painful stimuli within the cranial nerve distribution

7. Absent respiratory function: should always be done last, and the following must be adhered to following the disconnection of the ventilator:
   - pre-oxygenate the patient and rely on bulk transport of oxygen to maintain oxygenation during the test
   - look for apnoea clinically
   - sample ABG 10-15 minutes following disconnection from the ventilator.
   - the PaCO2 should be > 8 kPa with a pH < 7.30 (these thresholds may need altering with chronic lung disease-see ANZICS document)

TIME OF DEATH

The legal time of death is at the time of the completion of the second test of brain death studies.
Objective demonstration of the absence of cerebral blood flow is required if brain death is suspected and the preconditions or requirements for clinical certification cannot be met. For example:

- Facial trauma or obstruction of the external auditory canals may not allow assessment of all the brain stem reflexes
- A high cervical injury may not allow assessment of the respiratory centre
- Where the effects of sedation agents cannot be excluded

A 4 hour period of observation of absent brain function is necessary as in clinical confirmation of brain death. Haemodynamic stability (systolic pressure >90 is necessary during the intracranial blood flow study). Clinical examination of all available cranial nerve reflexes should be performed by two medical practitioners as above prior to radiological examination when the absence of cerebral blood flow may be established by either:

- Radionuclide cerebral perfusion scan
- 4 vessel catheter angiography

**CT angiography is not sufficient and can be misleading**

Certification of brain death is then undertaken after the scan has been verified by a practitioner certified to do so. The radiologist cannot constitute one of the doctors involved in brain death certification. These doctors must be suitably qualified practitioners as above.

The legal time of death is at the time of certification by the second practitioner.

**FREQUENTLY ASKED QUESTIONS: EXCLUSIONS TO THE DIAGNOSIS OF BRAIN DEATH**

The following observations do not exclude a diagnosis of brain death:

- Spontaneous “spinal” movements of the limbs
- Respiratory - like movements (shoulder elevation and adduction, back arching or intercostal expansion without significant tidal volume)
- Sweating
- Blushing
- Tachycardia
- Absence of diabetes insipidus (normal osmolar control mechanism)
- Deep tendon reflexes
- Up-going plantar reflex

**ORGAN SUPPORT IN DONORS**

See ODNZ guidelines.
ADMISSION TO ICU OF POTENTIAL DONORS

Patients may be referred for admission where brain death seems very likely and the only potential gain for the patients’ family and society is organ donation. In this section we are not talking about patients where the prognosis is unclear, but rather patients who currently are refused admission to ICU and extubated in the ED / Ward instead.

In these cases, the registrar should make a complete assessment of all the relevant medical / radiological details as for any other patient prior to discussion with the Intensivist on duty. In addition though, it would be reasonable to check the donor status on the drivers licence (obtained through the phone number in the Organ Donation Folder in ICU - NOT by asking relatives).

Potential medical exclusions to donation should be sought discreetly including advanced age (e.g. over 75 years), some malignancies with potential for distant spread, HIV, active viral hepatitis etc. (see Organ Donation Folder).

It is not an expectation of registrars that they initiate a formal examination to establish brain death or initiate a request for organ donation or discussion about organ donation or brain death in the Emergency Department (or indeed ICU), unless directed to by the Duty Intensivist and when willing and sufficiently experienced to do so. In nearly every instance, this discussion would be conducted directly by the specialist.

The focus must remain on the medical needs of the patient and the psychological needs of family at all times.

An indication that survival seems highly doubtful at this stage (once confirmed by the Intensivist in person or by phone) should suffice at this stage when it is felt inappropriate to initiate more direct discussion in the ED. It is currently not unusual to admit patients in this circumstance irrespective of their potential donor status.

The Intensivist may decide not to admit the patient if it is suspected that the patient may not progress to brain death, or in relation to any medical or social factors that would make donation unlikely, or if there is an immediate prospect that another patient’s life would be jeopardised by admitting this patient. The Link Nurse should be involved by Nursing or other staff to examine staffing possibilities if insufficient nurses are available and / or to provide assistance in any discussion subsequent to the Intensivists assessment / family discussion.

Patients should not be intubated to allow for the possibility of donation.

Sound clinical decision making must continue to apply.

DONATION AFTER CARDIAC DEATH

Waikato Hospital is accredited as a centre to undertake donation after cardiac death-i.e. non-beating heart donation. This only occurs rarely and occurs under the direction of a Consultant Intensivist. At present in New Zealand, it only happens in the setting of severe brain damage where withdrawal of treatment has already been accepted by those involved.

The relevant guideline should be consulted for detail (via ODNZ app or ICU guideline). The support and agreement of ODNZ is necessary for this process.
FLUIDS AND ELECTROLYTES

PRINCIPLES OF FLUID MANAGEMENT IN ICU

Intravenous fluid prescription is a core management tool in the critically ill. Poor fluid management can result in significant morbidity, such as fluid overload, pulmonary oedema, hyponatraemia and acute kidney injury. Positive fluid balance is associated with prolonged ICU length of stay and persistently elevated organ failure scores. There is a lack of high-quality evidence to guide routine IV fluid management, although some evidence regarding resuscitation now exists.

FLUID CHARTING

- Fluids are drugs and must be treated as such
- All fluid prescriptions must be reviewed daily.
- Fluid orders must be charted individually

FLUID ORDERS SHOULD BE CONSIDERED IN TWO COMPONENTS:

1) MAINTENANCE OR REPLACEMENT FLUIDS

Daily total fluid administration including enteral feeding = 25-30 mL/kg / day or 60-90 mL/hr, selected according to patient serum Sodium and total fluid balance + additional fluid tailored to excessive losses as appropriate. Remember that normal requirements are 25-30mL/kg/day H2O; Na, K and Cl: up to 1mmol/kg/day and glucose 50-100g/day. Options include:

- 4% dextrose and one fifth (0.18%)
- 0.45% saline
- 0.9% saline
- 5% dextrose
- Hartmann’s solution

Patients who are anuric or fluid overloaded should not necessarily receive “maintenance” fluids

2) RESUSCITATION FLUIDS

The intensive care community is divided on the relative suitability of various fluids in the resuscitation of a critically ill patient. In general if crystalloid (Hartmann’s solution or 0.9%NaCl) is chosen in the first instance, no more than 2000 ml should be administered, followed by blood products and/or vasopressor infusion according to the clinical situation. Balanced crystalloids are generally favoured in this unit (the chloride excess delivered by 0.9%NaCl is well-described elsewhere), with some exceptions (eg brain injury).

Synthetic colloids (eg HES, Voluven) have no place in the management of the critically ill patient. This is substantiated by the CHEST and 6S trials and by subsequent meta-analyses and now by international guidelines/licensing authorities.
Fluid boluses should optimally be titrated against a measurable end-point, although most in current use are at best imperfect.

**REMEMBER THE “5RS” OF FLUID PRESCRIBING:**

FROM BMJ 2014;350:g7620

**BASIC RULES:**

- Consider whether the patient needs IV fluids AT ALL
- Can their fluid (and calorific) requirements be met by the enteral route? If so – use this route
- Fluid prescriptions should not exceed 1mL/kg/hr IDEAL BODY WEIGHT, unless there are clear indications to exceed this
- Remember to add K+ when required
- Don’t target a CVP number
- Don’t target a urine output “goal”
- Consider lactate as a marker of perfusion
- Utilise a balanced crystalloid (Hartmann’s) in the first instance, unless there is a clear indication not to
- Regularly consider whether it is appropriate to de-escalate fluid administration or initiate fluid removal (actively diurese/RRT)
<table>
<thead>
<tr>
<th>Solution</th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
<th>Ca</th>
<th>Lac</th>
<th>Glu (g)</th>
<th>Osm</th>
<th>Prot (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.9% Saline</td>
<td>154</td>
<td>154</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>308</td>
<td></td>
</tr>
<tr>
<td>0.45% Saline</td>
<td>75</td>
<td>75</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>150</td>
<td></td>
</tr>
<tr>
<td>0.18% Saline/4% Dextrose</td>
<td>30</td>
<td>30</td>
<td></td>
<td></td>
<td></td>
<td>40</td>
<td>282</td>
<td></td>
</tr>
<tr>
<td>5% Dextrose</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>50</td>
<td>278</td>
<td></td>
</tr>
<tr>
<td>Hartmann’s (Lactated Ringer’s/Compound sodium lactate) solution</td>
<td>129</td>
<td>5.0</td>
<td>109</td>
<td>2.0</td>
<td>29</td>
<td>274</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gelofusine (500 ml) (NOT RECOMMENDED)</td>
<td>144</td>
<td>120</td>
<td></td>
<td></td>
<td></td>
<td>283</td>
<td></td>
<td>Gelatin 20</td>
</tr>
<tr>
<td>Albumin 4%</td>
<td>70</td>
<td></td>
<td>62.5</td>
<td></td>
<td></td>
<td></td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Voluven 6% (500ml)“HES”</td>
<td>154</td>
<td>154</td>
<td></td>
<td></td>
<td></td>
<td>304</td>
<td></td>
<td>Starch 30</td>
</tr>
</tbody>
</table>
ASSESSMENT OF FLUID BALANCE AND HYDRATION

CLINICAL MARKERS

- Skin turgor, mucous membrane hydration (poor indicator)
- Heart rate and blood pressure
- Peripheral perfusion, capillary refill

BIOCHEMICAL MARKERS:

- Serum Na+, Cl-, osmolality and trends
- Urea / creatinine and trends
- Bicarbonate and trend
- Lactate and trend
- Haematocrit and trend
- Charted fluid balance - at best a rough guide
- Charted intake minus (charted losses of all types + estimated insensible losses)

PREDICTORS OF INCREASED CARDIAC OUTPUT IN RESPONSE TO ADMINISTRATION OF FLUID

STATIC MEASURES (UNRELIABLE)

- JVP / CVP: very poor indicator of fluid-responsiveness. Very low CVP may indicate hypovolaemia. A trend in pressures may be useful
- Pulmonary artery pressures (particularly diastolic) & pulmonary capillary wedge pressure. At best these are poorly related to a response to further fluid (pre-load), but are still widely employed
- PiCCO parameters: derived estimates of intra-thoracic blood volume and extra vascular lung water

DYNAMIC MEASURES (MORE RELIABLE IF PRECONDITIONS MEET)

- Pulse pressure variation/stroke volume variation: Variation in arterial waveform with positive pressure ventilatory cycle, or SV by Vigileo or PiCCO. MUST be in sinus rhythm and FULLY controlled IPPV, without significant right heart failure
- Passive leg raising (PLR): An increase in Cardiac output (Vigileo or PiCCO) with PLR indicates volume responsiveness. However our current beds do not allow the correct technique. PLR may be more useful with most patients spont breathing and arrhythmia common (AF)
Volume status and fluid responsiveness are notoriously difficult aspect of critical care practice. If you are in any doubt consult the Duty Intensivist, but do not delay the administration of titrated fluids in the acute resuscitation phase.

**BODY FLUID AND ELECTROLYTE PHYSIOLOGY**

A working knowledge of the distribution of fluid and electrolytes throughout the body is required before any rational prescribing process can begin. It is beyond the scope of this handbook to describe in detail the physiology involved. What follows are salient notes on fluid and electrolyte distribution, and some of the more common disorders encountered in the intensive care setting.

**FLUID DISTRIBUTION**

**FLUID DISTRIBUTION IN THE NORMAL PERSON**

Fluid Distribution in the Normal Person

<table>
<thead>
<tr>
<th>Fluid Compartment</th>
<th>Volume (ml/kg)</th>
<th>Volume (l / 70 kg)</th>
<th>% total body weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma</td>
<td>45</td>
<td>3.15</td>
<td>4.5</td>
</tr>
<tr>
<td>Blood</td>
<td>75</td>
<td>5.25</td>
<td>7.5</td>
</tr>
<tr>
<td>Interstitial</td>
<td>200</td>
<td>14.00</td>
<td>20</td>
</tr>
<tr>
<td>Extracellular</td>
<td>250</td>
<td>17.50</td>
<td>25</td>
</tr>
<tr>
<td>Intracellular</td>
<td>350</td>
<td>24.50</td>
<td>35</td>
</tr>
<tr>
<td>Total body fluid</td>
<td>600</td>
<td>42.00</td>
<td>60</td>
</tr>
</tbody>
</table>

NB: these figures may be significantly altered in critical illness
<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Extracellular Fluid Compartments</th>
<th>Intracellular Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Plasma</td>
<td>Interstitium</td>
</tr>
<tr>
<td></td>
<td>Whole plasma</td>
<td>Plasma “water”</td>
</tr>
<tr>
<td>Sodium</td>
<td>140</td>
<td>150</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.7</td>
<td>4</td>
</tr>
<tr>
<td>Chloride</td>
<td>101</td>
<td>109</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>27</td>
<td>29</td>
</tr>
<tr>
<td>Calcium</td>
<td>1.1</td>
<td>1.2</td>
</tr>
<tr>
<td>Magnesium</td>
<td>0.7</td>
<td>0.8</td>
</tr>
<tr>
<td>Phosphate</td>
<td>1.1</td>
<td>1.2</td>
</tr>
<tr>
<td>Osmolality</td>
<td>291</td>
<td>291</td>
</tr>
</tbody>
</table>
Electrolyte derangement should be considered as resulting from one of the following:

- Erroneous results / Lab error (uncommon)
- Haemolysed specimen
- Factitious results: eg hyperglycaemia and hyponatraemia; lipaemic serum.
- Blood taken in proximity to an intravenous infusion
- Decreased or increased intake
- Decreased or increased loss (renal versus extra-renal)
- Shifts between compartments: eg potassium driven intra-cellularly by insulin/salbutamol

Treatment of electrolyte disturbance should be aimed at not only the apparent problem but also the underlying cause. Consideration should be given to the consequences of rapid correction of measured plasma...
electrolyte imbalances. Particularly those which have developed over a longer period of time, for which there may have been some intracellular accommodation.

**HYPONATRAEMIA: NA+ < 130 MMOL/L**

### AETIOLOGY

*Measured plasma osmolarity > 290 mmol/L:*

- Hyponatraemia in hyperglycaemia: For every 10 mmol/L increase in glucose, serum Sodium falls 3 mmol/L. It is in a sense a real hyponatraemia, however treatment aimed at correcting the blood glucose will resolve the hyponatraemia

- Mannitol: not usually a clinical issue, however later diuresis and hypernatraemia may be seen

- Alcohol (including methanol)

- Measured plasma osmolarity 270-290 mmol/L

- Hyperlipidaemia and hyperproteinaemia. (Neither should be a problem with current ion-specific electrodes for measurement of Na)

*Measured plasma osmolarity < 270 mmol/L:*

- Hypovolaemia with Sodium depletion

- Renal

- Diuretics

- Addisons

- Polyuric renal failure or diuretic recovery phase of renal dysfunction

- Extra-renal

- GIT loss (vomiting, diarrhoea, stoma output)

- Burns

- Hyperpyrexia & sweating

- Hypervolaemia (water excess)

- Renal failure: acute or chronic

- Extra-renal

- Excessive intake (IVI 5% dextrose for example). This is the commonest in-hospital cause of hyponatraemia

- Oedematous states: CCF, cirrhosis, nephrotic syndrome, hypoalbuminaemia
• Normovolaemia
• Psychogenic polydipsia
• SIADH
• Hypothyroidism
• Acute adrenal insufficiency

It is also important to measure urine osmolality. Urine osmolality is <100 mOsm/kg H2O in cases of excessive water intake, but >100 mOsm/kg H2O in all other causes. Differences in urine osmolality between SIADH and cerebral salt-wasting syndrome may be helpful in distinguishing these conditions. However, in general, urine osmolality is measured primarily to assess disease severity and is not useful for elucidating the underlying cause. It should be noted that causes such as endocrinopathies (glucocorticoid deficiency), potassium depletion, and diuretic use may present with either a euvolemic or hypovolemic state depending on the severity of the disease.

**EVALUATION ALGORITHM FOR HYponatraemia**
MANAGEMENT OF SEVERE HYponatraemia with fitting or decreased LOC

Resuscitative measures and ABC principles should not be delayed.

Hypertonic saline (23.4% (10ml amp=40mmol)) may be indicated but should not be used without prior discussion with the ICU Duty Intensivist, unless the patient is actively fitting. A typical dose would be 20mls (80mmol) over 10-15mins. Hypertonic saline is very irritant and is best administered via central venous access where time allows.

An infusion of 50-70 mmol / hr of Sodium should increase the serum Sodium by approximately 2 mmol/L / per hour.

The serum Sodium should not be allowed to increase more than 8-12 mmol/L in the first 24 hours, and certainly should not be overcorrected (max serum Sodium <= 130 mmol/L). (Risk of pontine myelolysis)

In very rare circumstances where fitting or encephalopathy are life threatening, 500 ml of 20% mannitol has been used. Bolus dosing of hypertonic saline may be just as effective. Presumably this is due to treatment of cerebral oedema.

REF:

Worthley LG. Hyponatraemia and speed of correction: why is there a dilemma? Crit Care & Resus 2000;2:173-180

HYPOVOLAEMIC STATES

Restore volume with normal saline or albumin according to clinical estimate (fluid balance, weight, JVP, CVP, underlying cause). Urine Sodium may be misleading in the context of diuretic administration or use of catecholamines.

HYPERVOLAEMIC STATES

Most common scenario clinically.

Fluid restriction if safe to do so (< 15 ml/kg/day).

Excess should correct as ADH levels re-set.

Address underlying cause (cardiac failure, etc).

SIADH

Often misdiagnosed

Diagnosis:

- Low serum osmolarity
- Urine osmolarity > plasma osmolarity
• Urine Sodium >20-40 mmol/L with normal renal, hepatic and cardiac function, and no diuretic use

Management:

• Fluid restriction (< 1000 ml / day)
• ADH receptor antagonists may have a role in the future (-vaptan drugs)

HYPERNATRAEMIA: NA+ > 145 MMOL/L

AETIOLOGY

Water depletion:

Virtually all body fluids have a Sodium concentration less than that of plasma

Renal loss:

• Diuretics or osmotic diuresis
• ARF / CRF
• Diabetes insipidus:
  • Neurogenic (including Guillain-Barre)
  • Nephrogenic: Hypercalcaemia, hypokalaemia, drug related (lithium), congenital

GIT losses:

• Diarrhoea
• Vomiting
• Fistulae
• Small bowel obstruction

Skin losses:

• Fever
• Vasodilated states
• Burns
• Thyrotoxicosis

Inappropriate fluid restriction or under administration in the elderly,( ie post-operative nil by mouth).

MANAGEMENT IN WATER DEPLETION STATES

Resuscitate if necessary.
“Restore” volume over 24-28 hrs using a relatively hyponatraemic fluid (half normal saline or 5% dextrose), if necessary a rough estimate of fluid deficit can be calculated:

\[
\text{Water deficit} = \frac{(\text{measured serum Na}^+ - 140)}{140} \times (\text{Body Weight} \times 0.6)
\]

For example, a 70kg male with a serum Sodium of 160 mmol/L might be expected to have a fluid deficit of 6 litres.

**Do not correct Sodium by more than 2 mmol / hour**

Aim for 0.5mmol/L/hr. Consider DDAVP if central diabetes insipidus has been confirmed.

---

**SALT GAIN**

Iatrogenic administration of Sodium containing feed or IV fluids.

Mineralocorticoid excess.

---

**HYPOKALAEMIA: K+ < 3.5 MMOL/L**

**AETIOLOGY / CLASSIFICATION**

Increased loss:

- Renal
- Diuretics
- Decreased serum Magnesium
- Decreased serum Calcium
- Steroids and mineralocorticoid excess
- Renal tubular acidoses
- GIT: diarrhoea, hypersecretory states (villous adenoma, small bowel fistulae)
- Inadequate dietary intake or daily administration

Transcellular shifts:

- Beta-andrenergic stimulants (catecholamines, salbutamol)
- Insulin (endogenous or exogenous)
- Familial periodic paralysis and related syndromes (consider thyrotoxic states)
- Decreased pH, hypomagnesaemia
Intravenous or oral potassium replacement. Intravenous replacement should not exceed 40 mmol/hr, concentrated solutions should be administered centrally and the patient carefully monitored. Concentrated solutions should not be administered peripherally.

Address cause of K+ loss.

**A low threshold should be adopted for co-administration of Magnesium as an essential co-factor in Na+-K+ pumps. Patients that are Magnesium deficient will remain hypokalaemic despite generous administration of potassium**

### HYPERKALAEMIA: K+ > 5.5 MMOL/L

Aetiology.

Changes in blood composition:

- Sampling in proximity to venous infusion
- Haemolysis
- Extremes of thrombocytosis and leukocytosis.

Release from intra-cellular compartments:

- Acidosis: decreased pH by 0.1 = serum potassium increased by 0.5 mmol/L
- Tissue disruption: tumour lysis syndromes, rhabdomyolysis, intravascular haemolysis, burns
- Suxamethonium
- “Insulin deficiency”: the hyperkalaemia associated with diabetic ketoacidotic states is related to lack of insulin and a change in serum pH but is usually associated with a total body potassium deficit

Increased intake: Not usually a problem unless patient has impaired renal function.

Reduced potassium clearance:

- Acute renal failure
- Renal tubular acidosis: type 4
- Potassium sparing diuretics: (spironolactone, amiloride)

**Patients with a slow rise in serum potassium usually tolerate elevated levels better than following an acute rise.**
Where elevated serum potassium (generally > 6.0 mmol/L) is associated with acute ECG changes or haemodynamic compromise this should be considered a medical emergency and treated as follows:

- Calcium gluconate 10% 10mL IV over 5 minutes
- Insulin/Dextrose infusion (eg 10U actrapid insulin in 50mL 50% Dextrose IV over 20-30 minutes)
- Consider salbutamol nebuliser (avoid if already tachycardic)
- Renal replacement therapy may be urgently required

Calcium resonium as a chelating agent can be considered, either NG or rectally.

**HYPOPHOSPHATEMIA**

Relatively common in the critically ill: associated with reduced intake (eg TPN), re-feeding syndrome, acute respiratory alkalosis, insulin infusion (eg DKA), glucagon and catecholamine administration and renal replacement therapy. Primary causes are very rare in ICU patients. Low phosphate will contribute to muscle weakness and potentially slow ventilator weaning.

**PHOSPHATE REPLACEMENT IN THE ICU**

<table>
<thead>
<tr>
<th>Serum phosphate:</th>
<th>Dose (IVI):</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 0.7 mmol/L</td>
<td>0.16 mmol/kg over 4-6 hrs</td>
</tr>
<tr>
<td>0.5-0.7 mmol/L</td>
<td>0.32 mmol/kg over 4-6 hrs</td>
</tr>
<tr>
<td>&lt; 0.5 mmol/L</td>
<td>0.64 mmol/kg over 8-12hrs</td>
</tr>
<tr>
<td></td>
<td><strong>Given as Potassium Di-Hydrogen Phosphate</strong> (10mmol in 10mls) Sodium dihydrogen phosphate is also available</td>
</tr>
</tbody>
</table>

**HYPOMAGNESEMA**

Magnesium is a major intracellular cation involved in a wide variety of cellular functions being an important cofactor for a wide range of enzymes, transporters and nucleic acids required for normal cellular function, replication and energy metabolism.

**AETIOLOGY**

There are many potential causes of low magnesium. More common causes in the ICU include:

- Intestinal malabsorption
- Protracted vomiting
• Diarrhoea
• Drugs (gentamicin)
• Phosphate depletion
• Metabolic acidosis
• Pancreatitis
• Burns
• Defective renal tubular reabsorption / acute renal injury

**CLINICAL EFFECTS**

Usually asymptomatic at levels >0.5mmol/L. Cardiac arrhythmias may occur at lower levels including sinus tachycardia, SVT and VT. Prolonged PR or QT intervals may also be seen with T wave flattening or inversion.

**Other electrolyte abnormalities including hypocalcaemia and hypokalaemia may not be easily corrected unless magnesium is also administered**

**MAGNESIUM REPLACEMENT**

1 ampoule 49.3% = 5mls = 10mmol = 2.47G.

Symptomatic treatment: give 10mmol over 5 to 60 minutes depending on urgency. Rapid administration may lead to flushing and hypotension. Maintenance infusion may be required to correct deficiency using 20mmol over 3 hours.

**MAGNESIUM IS TREATMENT OF CHOICE IN PRE-ECLAMPSIA/ECLAMPSIA**

• Loading dose of 4 g should be given intravenously over 20 minutes
• Followed by an infusion of 1 g/hour maintained for 24 hours
• Recurrent seizures should be treated with a further dose of 2g given over 5-20 minutes

**HYPOCALCAEMIA**

Mild degrees of hypocalcaemia are usually asymptomatic. Levels of ionised Ca++ <0.8mmol/L may cause neuromuscular irritability and result in clinical symptoms. Relatively less hypocalcaemia, (ionised Ca++ <0.9mmol/L) may reduce the efficacy of inotropes. Treatment should be considered in view of the clinical situation.

**AETIOLOGY**

There are many potential causes of low serum calcium. Calcium chelation and hypoparathyroidism constitute the common mechanisms in the ICU setting. This is frequently accompanied by other biochemical abnormalities therefore pattern recognition will often point to the aetiology.
### Aetiology vs. Biochemical Pattern

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Biochemical Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Serum Albumin</td>
<td>Reduced total Ca, normal ionised Ca</td>
</tr>
<tr>
<td>Alkalosis</td>
<td>Normal total Ca, reduced ionised Ca</td>
</tr>
<tr>
<td>Hypomagnesaemia</td>
<td>Reduced ionised Ca &amp; Hypokalaemia</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Hypocalcaemia, raised Lipase &amp; Glucose</td>
</tr>
<tr>
<td>Renal Failure</td>
<td>Raised Urea, elevated Phosphate</td>
</tr>
<tr>
<td>Rhabdomyolysis</td>
<td>Hypocalcaemia, elevated Phosphate, CK urinary Myoglobin</td>
</tr>
<tr>
<td>Tumour Lysis Syndrome</td>
<td>Hypocalcaemia, elevated Phosphate, Potassium &amp; Urate</td>
</tr>
<tr>
<td>Calcium chelation eg</td>
<td>Reduced total Ca, reduced ionised Ca</td>
</tr>
<tr>
<td>blood products transfusion</td>
<td></td>
</tr>
</tbody>
</table>

### CLINICAL EFFECTS

Usually asymptomatic at levels >0.8mmol/L

- CNS effects: paraesthesia, muscle cramps, tetany, proximal myopathy
- Cardiovascular effects: arrhythmias, hypotension, inotrope unresponsiveness, prolonged QT, T wave inversion
- Respiratory effects: Laryngospasm, bronchospasm, apnoea

### INDICATIONS

- Symptomatic hypocalcaemia (cardiac depression, vasodilatation, muscle weakness, tetany)
- Ionised calcium <0.8mmol/L
- Hyperkalaemia
• Calcium channel antagonist or beta block overdose
• Hypocalcaemia in setting of high inotrope requirement
• Massive blood/products transfusion

**CALCIUM REPLACEMENT**

Individualise according to serum level, indication and response. It is available as calcium gluconate 10%, or calcium chloride 10%. The main difference is the amount of elemental calcium in each preparation (Gluconate 2.3mmol, Chloride 6.8mmol). Do not mix with other drugs.

- Calcium Gluconate 10ml ampoule 10% solution at 2mls/minute
- Calcium Chloride 10ml ampoule 10% solution 1ml/minute
- As an infusion 10mls of 10% solution in 50mls D5W given over 10 – 20 minutes

May cause hot flushes, vasodilatation, hypotension, bradycardia and syncope.

**HYPERCALCAEMIA**

Less commonly seen in the critically ill: generally due to an underlying disease process. Symptoms include GI upset, confusion and polyuria.

**AETIOLOGY**

- Malignancy
- Iatrogenic
- Hyperparathyroidism
- Sarcoidosis

**TREATMENT**

Rehydration is the initial treatment. Consider forced diuresis to aid excretion, calcitonin, bisphosphonates (work over days). Corticosteroids may have a role in malignancy or sarcoid-induced hypercalcaemia.

**ACID-BASE DISTURBANCES IN THE ICU**

Critically ill patients commonly have a deranged acid base status. Despite this, explanations of the physiology behind the process are not universally accepted. It is necessary to have an approach to the clinical importance of each of the common major abnormalities, even given the complex and often mixed scenario's you might encounter. You are encouraged to read widely on the subject of acid-base disorders and the opposing ideologies put forward to explain them.
Correction of acid-base disturbance should be aimed at the underlying cause, and not at correction of the superficial abnormality.

**GENERAL PRINCIPLES - REGULATION OF PH**

The concept of pH: pH = negative log of the hydrogen ion concentration. Normal range = 7.35 - 7.45

Without regulation of acid-base, the daily production of non-volatile H+ in a normal person (about 70 mmol) would reduce the pH in a volume of water similar to that of a 70kg man (42 l) from 7.4 to a pH of 2.78.

\[
[H^+] = \frac{K \times CO_2}{[HCO_3^-]}
\]

\[
\text{Henderson / Hasselbach Equation} \quad \text{pH} = \frac{6.1 + \log[HCO_3^-]}{P_{CO_2} \times 0.03}
\]

The human body is an “open” system in which other organ systems and tissues contribute to the maintenance of the free [H+] within a narrow, biologically tolerable range.

From both the above it is clear that any mechanism responsible for regulating or affecting pH does so by changing the relative concentrations of HCO3-, PaCO2 or H+ directly.

The response of the body to an enforced change in one of these parameters takes place in three broad groups:

1. Adjusting minute ventilation (increasing respiratory rate or tidal volume) to manipulate PaCO2
2. Buffering systems:
   - Bicarbonate ion
   - Haemoglobin
   - Protein substrates
   - Phosphate
3. Renal compensation: delayed > 6-12hrs

**PRIMARY AND SECONDARY ACID-BASE DERANGEMENTS**

End point: “constant” PCO2 : HCO3- ratio.
CHARACTERISING ACID - BASE DISTURBANCES

<table>
<thead>
<tr>
<th>Acid-Disorder</th>
<th>Primary Change</th>
<th>Compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory acidosis</td>
<td>↑PCO₂</td>
<td>↑HCO₃⁻</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>↓PCO₂</td>
<td>↓HCO₃⁻</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>↓HCO₃⁻</td>
<td>↓PCO₂</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑HCO₃⁻</td>
<td>↑PCO₂</td>
</tr>
</tbody>
</table>

Compensatory changes are “never” complete, and certainly “overcompensation” does not occur.

ADEQUACY OF COMPENSATION

Expected magnitude of compensation for a primary abnormality is given below. In critically ill or ventilated patients compensation may not be possible, presenting as a mixed or complex problem.

Expected Compensation Following Acid-Base Disturbance

<table>
<thead>
<tr>
<th>Primary disorder</th>
<th>Expected Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>PCO₂ in mmHg = (1.5 × HCO₃⁻ in mmol/L) + 8</td>
</tr>
<tr>
<td></td>
<td>PCO₂ in kPa = HCO₃⁻/5 + 1</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>PCO₂ = (0.7 × HCO₃⁻) + 21</td>
</tr>
<tr>
<td></td>
<td>PCO₂ in kPa = HCO₃⁻/10 + 2.6</td>
</tr>
<tr>
<td>Acute respiratory acidosis</td>
<td>ΔpH = 0.008 × (PCO₂-40) or approx 1 mmol/L ↑ in HCO₃⁻ per 10 mmHg increase in P₂CO₂</td>
</tr>
<tr>
<td></td>
<td>In kPa: HCO₃⁻ (mmol/L) = (PCO₂-5.3)*3/4 +24 or, “up three quarters per kPa”</td>
</tr>
<tr>
<td>Chronic respiratory acidosis</td>
<td>ΔpH = 0.003 × (PCO₂-40) or approx 4 mmol/L ↑ in HCO₃⁻ per 10 mmHg increase in P₂CO₂</td>
</tr>
<tr>
<td></td>
<td>In kPa: HCO₃⁻ (mmol/L) = (PCO₂ – 5.3)*3 + 24 or, “up three per kPa”</td>
</tr>
<tr>
<td>Acute respiratory alkalosis</td>
<td>ΔpH = 0.008 × (40-PCO₂)</td>
</tr>
</tbody>
</table>
|                          | In kPa: HCO₃⁻ (mmol/L) = 24 – 1.5*(5.3-PCO₂) or, “down one and a half
Chronic respiratory alkalosis

\[ \Delta pH = 0.017 \times (40 - PCO_2) \]

In kPa: \( HCO_3^- \ (\text{mmol/L}) = 24 - 4 \times (5.3 - PCO_2) \) or, “down four per kPa”

**METABOLIC ACIDOSIS**

**THE ANION GAP**

Classically metabolic acidoses are classified according to the concept of anion gap. Whilst the body must maintain overall electrical neutrality there are a number of unmeasured ions which result in a difference when the major cations are compared to the major anions.

ie. Anion Gap = \( [\text{Na}^+ + \text{K}^+] - [\text{Cl}^- + \text{HCO}_3^-] \) = 12-17 mmol/L = unmeasured anions

**Determinants of the Anion Gap**

<table>
<thead>
<tr>
<th>Unmeasured anions</th>
<th>Unmeasured cations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proteins (albumin) 15 mmol/L</td>
<td>Calcium 2.5 mmol/L</td>
</tr>
<tr>
<td>Organic acids (lactate, ketones) 5 mmol/L</td>
<td>Magnesium 1.2 mmol/L</td>
</tr>
<tr>
<td>Phosphates 2 mmol/L</td>
<td>IgG</td>
</tr>
<tr>
<td>Sulphates 1 mmol/L</td>
<td>Other</td>
</tr>
</tbody>
</table>

An increase in anion gap usually means an increase in an organic acid. In some patients with low serum albumin this may be masked unless you adjust accordingly.

**AETIOLOGY**

Raised anion gap metabolic acidosis:

- Lactic acidosis
- Ketoacidosis
- Rhabdomyolysis
- Drugs or toxins:
  - Aspirin  (may result in elevated salicylate, lactate, ketones)
- Ethanol
- Methanol
- Ethylene glycol
- Paraldehyde
- Renal failure: usually only mildly elevated anion gap ( < 23)

**Low or normal anion gap acidosis:**

- Hyperchloraemic metabolic acidosis
- Infusion IVI of NaCl
- Resolving renal failure
- Renal tubular acidosis / carbonic anhydrase inhibitors
- GIT losses including fistulae
- Hypoalbuminaemia
- Myeloma

**MANAGEMENT**

**High anion gap**

Address cause. **Bicarbonate administration is not indicated**

**Normal anion gap**

- Address underlying cause
- In some situations (eg. renal tubular acidosis) it may be appropriate to replace / administer bicarbonate directly

**Approx deficit = (24-[HCO3-]) x (body weight x 0.6) in mmol**

- Generally one third to one half of the estimated deficit should be replaced and then acid-base status reviewed

**METABOLIC ALKALOSIS**

**AETIOLOGY**

Common causes

- Diuretics
- Vomiting
• Post hypercapnoea > 48 hrs
• Any fluid loss replaced with insufficient Na+, associated with H+ loss (contraction alkalosis).
• Association with hypovolaemia and / or hypokalaemia
• H+ / PROTON LOSS
• Renal
• ? Na+ reabsorption
• Cushings syndrome including exogenous steroid administration
• Proximal tubulopathies: Bartter’s syndrome, Liddle’s syndrome
• Hypercalcaemia / hypomagnesaemia associated with diabetes insipidus
• Diuretics
• GIT
• NG suctioning or protracted vomiting
• Diarrhoea (acidosis more likely)
• INCREASED ADMINISTRATION OF BASES
• CVVHDF-lactate buffered solution

**MANAGEMENT**

Correct hypovolaemia and electrolyte abnormalities. Review drugs, and administration of exogenous bases (lactate buffered dialysate, citrate). Acetazolamide has been used to increase renal losses of bicarbonate, however this should not be considered routine practice.

**RESPIRATORY ACIDOSIS**

**AETIOLOGY**

Any cause of hypoventilation, whether respiratory failure or planned (permissive hypercapnoea ventilation)

**TREATMENT**

Address underlying respiratory pathology

**RESPIRATORY ALKALOSIS**

**AETIOLOGY**

• Any cause of hyperventilation in ICU eg. early sepsis
- Early hypoxic situations
- Anxiety
- Hysteria (NB this is a diagnosis by exclusion, and presumes normal oxygenation)
- Neurogenic hyperventilation: usually a marker of severity of head injury.

**TREATMENT**

Treat underlying problem
GENERAL PRINCIPLES

In general the following rules should be followed where possible before instituting inotropic support.

ESTABLISH THE PRESENCE OF HYPOTENSION

- **Absolute**: Systolic Blood Pressure < 90 mmHg
- **Relative**: Systolic Blood Pressure decrement > 30 mmHg below normal for that patient

Despite these definitions, we often quote mean arterial blood pressure (MAP) as being more relevant to organ perfusion. A MAP of > 60-70 mmHg would be considered adequate in most instances.

ORGAN PERFUSION

When hypotension is deemed to exist, assess organ perfusion:

- Renal: urine output (minimum 0.5 ml/kg/min)
- Cerebral: cognitive state
- Peripheries (unreliable in septic patients)
- Surrogate markers: eg. Metabolic acidosis on a blood gas, measured lactate or venous oxygen saturation

ASSESS AND CORRECT HYPOVOLAEMIA

The only reason to challenge a patient with fluid is to increase a patient’s stroke volume (SV). If there is no increase in SV in response to fluid, it serves no purpose and may well be harmful. In practice it is difficult to predict which patients are likely to be volume responders (ie. increase SV by 10–15%) and both hypovolaemia and hypervolaemia increase mortality and morbidity. Volume responsiveness does not equate to hypovolaemia and fluid should be limited despite ongoing fluid responsiveness if the patient is assessed as no longer being hypovolaemic. In the ICU there are a number of ways to assess intravascular volume status have been used, however these have their shortcomings.

- Clinical assessment of fluid status including JVP
- Right heart catheterisation with pulmonary artery occlusion pressure
- Measurement of dynamic and static measurements CVP
- Echocardiographic techniques (IVC collapse)

Have all been shown to poor predictors of volume responsiveness. Variation of arterial waveform characteristics with mechanical ventilation (pulse pressure variation and stroke volume variation) have been
shown to be useful predictors of volume responsiveness under controlled conditions but recent studies have shown that in average ICU patient they are poor predictors of volume responsiveness.

The best predictors of volume responsiveness in ICU are:

- Measured changes in SV in response to a passive leg raising manoeuvre and
- Measured changes in SV in response to a fluid challenge

Both of these techniques however have a requirement for measuring the stoke volume.

**INSTITUTING INOTROPIC THERAPY**

Only once the above steps have been considered should inotrope therapy be considered.

No single inotrope (or mixture of inotropes) has been shown to be superior to another.

**CATECHOLAMINES**

There is marked inter-individual variation in response to a chosen inotrope, due to:

- Qualitative and quantitative changes in adrenergic receptor kinetics in both acute illness (sepsis) and chronic conditions (heart failure)
- Underlying variability in disease state (ie cardiogenic shock, sepsis, hypovolaemia)

**SHORT NOTES ON USING COMMON AGENTS**

Dopamine / adrenaline / noradrenaline: For ease of application many claim these three agents have a beta-adrenergic action in low dose and a progressive alpha-effect in increasing doses. Each however has a characteristic feature worth noting.

Dopamine in low doses (2.5 microg / kg / min) has a direct diuretic effect which may result in increased urine volume; there is no evidence of a reno-protective effect.

Adrenaline is a useful alpha / beta-agonist, however it does have significant beta2-effect which may result in unwanted metabolic effects (hyperglycaemia, lactic acidosis).

Noradrenaline is generally held to have a predominant alpha-effect and is therefore useful as an inotrope-vasopressor, particularly in septic shock but does have significant beta-agonist activity.

Dobutamine, a synthetic inotrope, does not have significant alpha-effects (may have some myocardial beta1-effect) and is therefore useful in increasing heart rate and stroke volume, but may cause a paradoxical fall in blood pressure due to peripheral beta2-adrenergic activity.

Adrenaline and noradrenaline infusions should be started at 3-5 microg / min and titrated to response. Infusions of these agents require 3-5 minutes to achieve steady state. Changes in rate more frequently than every 3-5 minutes (unless in an emergency) should be discouraged as it may lead to a “roller-coaster” effect.
PHOSPHODIESTERASE INHIBITORS

Phosphodiesterase inhibitors increase cAMP by non-adrenergic mechanisms. They are not therefore affected by down-regulation of adrenoreceptors as occurs in sepsis or heart failure. For this reason milrinone is useful for refractory (ie following adequate volume resuscitation) low cardiac output states.

They result in:

- Increased myocardial contractility
- Systemic and pulmonary vasodilatation (often requires co-administration of a vasopressor / noradrenaline)
- Improved diastolic relaxation (useful in patients with diastolic heart failure)

NOTES ON PHARMACOLOGY OF MILRINONE

These drugs usually require a loading dose on commencement which may predispose to additional hypotension by virtue of vasodilatation. A smaller loading dose may be used at times.

The relatively long half-life of these agents requires forethought before administration, as their action is not easily reversed, and titration of infusions to effect cannot be effected rapidly.

LEVOSIMENDAN

Calcium sensitising inotrope with prolonged duration of action. Possibly less increase in myocardial oxygen consumption. As with PDEi, levosimendan is a potent pulmonary vasodilator and leusitropic agent. Studies however have not shown any benefit over milrinone.

INOTROPIC AGENTS COMMONLY USED IN ICU

<table>
<thead>
<tr>
<th>Agent</th>
<th>Standard Infusion</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>1 mg / 50 ml D5W ratio.</td>
<td>CPR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe sepsis syndrome / shock</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiogenic shock</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute severe asthma (adjunct)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anaphylaxis (correct hypovolaemia !!)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Medical Pacing (1st line drug)</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>8 mg / 50 ml D5W ratio. 0-30mls/hr (ml/hr = μg / min)</td>
<td>Conditions where mixed α- / β-effect is required with a predominant α-effect</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ie. septic shock</td>
</tr>
<tr>
<td>Drug</td>
<td>Dose &amp; Type</td>
<td>Notes and Indications</td>
</tr>
<tr>
<td>-----------------</td>
<td>---------------------------------</td>
<td>--------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Dopamine</td>
<td>200 mg / 50 ml D5W</td>
<td>No advantage over adrenaline or noradrenaline&lt;br&gt;May induce more tachycardia than adrenaline (through stimulation of D-receptors)&lt;br&gt;Not reno-protective, but may have direct diuretic effect</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>250 mg / 50 ml D5W</td>
<td>Pure β adrenergic agent used in low cardiac output / high vascular resistance states&lt;br&gt;Effect may be diminished in sepsis and chronic heart failure due to down regulation of receptors</td>
</tr>
<tr>
<td>Levosimendan</td>
<td>12.5 mg in 500ml 5% dextrose.</td>
<td>Inotrope, predominantly by Ca²⁺ sensitisation of myocardium&lt;br&gt;Recommended loading dose (up to 24 µg/kg in 10 min) often omitted or given more slowly</td>
</tr>
<tr>
<td>Milrinone</td>
<td>Loading dose:&lt;br&gt;12.5 - 50µg / kg over 20 min&lt;br&gt;Infusion:&lt;br&gt;10 mg / 50 ml D5W (200µg / ml)&lt;br&gt;infuse at 0.375 – 0.75 µg / kg / min&lt;br&gt;(8-15 ml / hr in 70 kg pt)</td>
<td>Cardiogenic shock due to diastolic failure&lt;br&gt;Pulmonary hypertension following cardiac valve replacement&lt;br&gt;Rescue following catecholamine induced down regulation of receptors in patients requiring ongoing chrono-inotropy&lt;br&gt;These agents may accumulate in renal failure</td>
</tr>
<tr>
<td>Isoprenaline</td>
<td>2mg (10mls) in 500mls 5% dextrose&lt;br&gt;infuse at 1.25ml/min adjust to effect. (Up to 7.5ml/min in advanced shock)</td>
<td>B-Adrenoreceptor agonist used in Heart Block, Stokes Adams Attacks, cardiac arrest</td>
</tr>
</tbody>
</table>

If more concentrated solution needed:<br>3mg in 50mls D5W 1-25mls/hr
VASOPRESSOR AGENTS

INTRODUCTION
Noradrenaline is the vasopressor of choice in the ICU. Indirect acting agents such as metaraminol and phenylephrine should generally be restricted to peri-operative practice where temporary vasodilatation results from specific intervention (spinal, epidural, local block) and as a peripherally administered vasopressor until central access has been achieved for noradrenaline administration.

GENERAL PRINCIPLES
- These agents are used primarily to induce vasoconstriction and thus elevate blood pressure
- They may increase cardiac afterload and thus cardiac wall stress
- These agents should not be used to treat hypotension due to hypovolaemia

INDICATIONS
Hypotension following sympathetic block (epidural anaesthesia), where vasodilatory effect is likely to be temporary, and excessive fluid administration is not recommended.

Hypotension resistant to catecholamine use (ie. vasopressin).

COMPLICATIONS
- Rebound hypotension
- Vagal response (with possible bradycardia)
Elevated blood pressure should be viewed in the context of each patient, and should include an appraisal of pre-morbid blood pressure.

Hypertension in the intensive care should not elicit direct treatment, but rather a review of the cause of blood pressure elevation.

Elevated blood pressure is commonly seen in patients who are agitated, delirious, or who have some other cause for overt sympathetic drive. This should be addressed with analgesia and sedation where appropriate. A dual purpose drug such as an alpha2-agonist (clonidine or dexmedetomidine) may be useful.

Hypertension in the setting of intracranial pathology may be self driven. No attempt should be made to actively lower elevated blood pressure with anti-hypertensive agents unless cerebral perfusion pressure is not threatened. Vasodilators may increase intracranial pressures further while dropping cerebral perfusion pressure to dangerous levels.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Standard Infusion</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenylephrine</td>
<td>10 mg made up to 50 ml 5% dextrose. Start infusion at 5 ml/hr. Titrate to BP at 1 – 10 ml/hr</td>
<td>Predominantly used in anaesthetics for short periods of predictable hypotension associated with epidural or spinal anaesthesia. Can be administered peripherally</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>20 units / 40 ml D5W at 2.0 ml/hr (approx =0.04 units / min)</td>
<td>Catecholamine resistant hypotension - limited availability. Efficacy appears to be above a given threshold and not linear – “do not titrate” to effect</td>
</tr>
<tr>
<td>Metaraminol</td>
<td>0.01mg/kg stat Dilute 10mg to 10mls. 1ml boluses. IVI: 10mg / 50ml D5W (200mcg/ml) 0-30mls/hr</td>
<td>Advantages of bolus dose - often 1mg increments PRN May be given as infusion to maintain desired BP 0-30mls/hr Can be given peripherally</td>
</tr>
</tbody>
</table>
INDICATIONS

ACUTE

Peri-operative control of hypertension post-cardiac, carotid or other vascular surgery, or for patients with critical myocardial ischaemia. In this instance target blood pressures should be discussed with the surgeon involved, and confirmed with the Duty Intensivist.

- Accelerated hypertension: “Malignant Hypertension”
- Hypertensive Proteinuric Pregnancy states (Eclampsia)
- Active Phaeochromocytoma (NB: always precede β-blockade with alpha blocker)
- Non-hypertensive indications
- Reduction of afterload in cardiac ischaemia and failure
- Decrease dP / dt in patients with aortic dissection using β-blockers

COMPLICATIONS

- Hypotension
- Tachyphylaxis
- Cyanide Toxicity - Sodium Nitroprusside
- Pulmonary vasodilatation causing increased pulmonary shunting and hypoxia

VASODILATORS AND OTHER ANTI-HYPERTENSIVE AGENTS

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Uses</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GTN (glyceryl tri-nitrate)</strong></td>
<td>50 mg in 50 ml. Use non-PVC giving set.</td>
<td>Venodilation &gt; arterial. Unpredictable hypotensive effects</td>
</tr>
<tr>
<td></td>
<td>Range 1-20 ml / hr (0-20 mg / hr)</td>
<td>Useful in cardiac ischaemia (no proven effect on outcome)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tachyphylaxis when used &gt; 24hrs</td>
</tr>
<tr>
<td><strong>Sodium Nitroprusside</strong></td>
<td>50 mg / 50 ml D5W.</td>
<td>Rapid control hypertension by direct arteriolar action</td>
</tr>
<tr>
<td></td>
<td>Range 0-20 ml / hr IVI</td>
<td>Cyanide toxicity may be seen at total dose &gt; 1.5</td>
</tr>
<tr>
<td>Drug</td>
<td>Dose/Route</td>
<td>Action/Adverse Effects</td>
</tr>
<tr>
<td>------------</td>
<td>----------------------------</td>
<td>---------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Captopril</td>
<td>Dose 6.25-50 mg up to 6 hrly po; Syrup 5 mg/ml also available for smaller dose adjustment</td>
<td>After load reduction / anti-hypertensive; Dose adjustment required in renal failure; May aggravate renovascular insufficiency; Beware first dose effect (↓ BP &gt; expected)</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5-40 mg daily po</td>
<td>Longer acting than captopril, only use once patient stable, or captopril therapy established</td>
</tr>
<tr>
<td>Amlodipine</td>
<td>5-10 mg daily po</td>
<td>Long acting dihydropyridine Ca channel blocker; Use as anti-hypertensive in stable patients (not anti-arrhythmic)</td>
</tr>
<tr>
<td>Labetalol</td>
<td>Bolus: 5 mg bolus. No faster than 20-50 mg over 1-5 min. (&lt; 300 mg/d); Infusion: 200 mg in 40 ml DSW, infuse at 5-30 ml/hr (25-150 mg/hr)</td>
<td>Non-selective β-blocker, some α-blocker activity; Useful in bolus temporising treatment of acutely elevated blood pressure; Not shown to increase ICP in head injured patients (unlike GTN and Nitroprusside)</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>Oral: 47.5 mg – 190 mg per day in 1–2 doses (CR formula); NG: 25 – 200 mg / day in divided doses; IVI: 1-5 mg slowly up to 15 mg</td>
<td>Commonly used β-blocking agent; Metabolised by the liver, useful in conditions where renal function uncertain or deteriorating</td>
</tr>
<tr>
<td>Esmolol</td>
<td>Bolus: 0.25 – 0.5 mg / kg; IVI commonly in 10mg increments; Infusion: 50 – 200 ug / kg / min.</td>
<td>Ultra short acting β-blocker used in cardiac surgery, and where a short trial of the patients ability to tolerate a negative inotrope is useful</td>
</tr>
<tr>
<td>Drug</td>
<td>Bolus/Infusion Details</td>
<td>Action/Notes</td>
</tr>
<tr>
<td>---------------</td>
<td>----------------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| Clonidine     | Bolus: 150µg diluted in 10 ml NS, administer 1-5 ml IVI prn                             | Centrally acting α2-agonist  
Useful in peri-operative centrally mediated hypertension, with anti-agitation benefits |
| Hydralazine   | 10-20 mg as a bolus IVI titrated in 1-2mg increments                                     | Direct acting arterial vasodilator  
Mild positive inotrope but often associated with a reflex tachycardia  
Half-life 4-6 hrs - Metabolic slow acetylators at risk to develop Lupus Syndrome with prolonged use, or dose > 200 mg / day |
| Methyldopa    | 250 mg - 2 g / day bd po                                                              | Traditional agent still useful in gestational proteinuric hypertension       |
| MgSO₄         | Bolus: 10-20 mmol IVI slowly (may cause sensation of flushing, and hypotension)  
Infusion: 40 mmol in 500 ml of D5W 6 hrly IVI (1 ampoule = 10 mmol = 2.5 g)  
Preeclampsia/eclampsia 4g bolus over 5-10 min followed by infusion of 1g per hour | Gestational proteinuric hypertensive states ("eclampsia")  
Torsades  
Phaeochromocytoma  
? Status Asthmaticus, seizures  
Beware of hot flush on administering IVI bolus in awake patients  
Toxicity: monitor for “weakness” in non-ventilated patients: ie falling respiratory rate and loss of deep tendon reflexes |

**ANTIARRHYTHMIC DRUGS IN CRITICAL CARE**

**GENERAL PRINCIPLES OF TREATMENT**

In therapy of arrhythmias, prior consideration should be given to causal or aggravating factors:

- Hypoxæmia
- High, occasionally low, pH
- Hypokalæmia, hypomagnesæmia
- Pre-existing drug effects or toxicity (including bronchodilators and inotropes)
- Presence of right heart lines (including pacemakers)
- Myocardial or coronary compromise, especially pulmonary œdema

The need for therapy should be carefully evaluated. For instance atrial tachyarrhythmias with a ventricular response similar to that of the preceding sinus rhythm need not be slowed down or abolished at the cost of additional hypotension. Reperfusion VT associated with myocardial infarction (fascicular or otherwise) or non-paroxysmal junctional tachycardia (NPJT) at 130 / min may be as acceptable as sinus or junctional bradycardia at 40 / min and equally self-limiting.

Non-pharmacological therapy should also be always considered -if only to be dismissed, as in the case of precordial thump for VT (repeated thumps for pacing asystole may be OK). In patients with permanent pacemakers, a magnet may abolish both VT or SVT by fixed overdrive pacing; post-CABG patients with temporary wires may be more reliably overdriven. DC cardioversion is always available.

The value of precise diagnosis is increasingly deconstructed by the likes of sotalol or amiodarone. Still, as a general rule, if antiarrhythmics are to be used, a 12-lead ECG should be obtained.

Arrhythmias can only be defined electrocardiographically. On the other hand, a full ECG obtained on a pulseless patient is medico-legally indefensible.

In many ICU patients the need for continued therapy ceases as they improve and the drug (often the ubiquitous amiodarone) can be stopped.

REFERENCE:

DRUG THERAPY OF BRADYARRHYTHMIAS

Pacing is preferred, but there are clinical situations when pacing is either impracticable or fails. A number of drugs have some utility in the setting of bradycardia – bradyarrhythmia.

ADRENALINE

A mixed alpha- / beta- agonist, adrenaline has declined in popularity mainly due to its metabolic side effects mediated by beta2-receptor stimulus (increased lactic acid production, hyperglycaemia). In emergency situations where bradycardia is associated with hypotension and patient compromise adrenaline remains the first line agent, at least in the short term.

ATROPINE

Alkaloid from Atropa belladonna, competitive acetylcholine antagonist at post-ganglionic parasympathetic endings. It comes in 0.6 mg ampoules. Doses smaller than 0.6 mg in an adult may paradoxically cause bradycardia.
Its vagolytic action is useful in the very early stages of (usually inferior) myocardial infarction complicated by significant bradycardia or block. Intraventricular, Möbitz II block is made worse. Escape-capture bigeminy may be replaced by slower 2:1 block.

It may also be used in reflex bradycardia associated with upper airway manoeuvres, such as suctioning. In brady-asystolic cardiac arrest, it is next to useless.

ISOPRENALINE

Isoproterenol is a “pure” β agonist producing marked vasodilatation and cardiac stimulation; these actions have long ago necessitated its replacement by selective β-2 bronchodilators in asthma. It is sometimes used for:

- 3rd degree or advanced 2nd degree AV block as a bridge to pacing
- to promote tachycardia and shorten the QT interval, against potential or manifest torsade de pointes. Here, too, pacing offers greater flexibility and stability

SUPRAVENTRICULAR ARRHYTHMIAS

Some agents control the ventricular response through AV blocking action, some interrupt the re-entry circuit and abolish the paroxysm; many do both.

ADENOSINE

Endogenous adenosine production is enhanced by ischæmia and it may well be the mediator of sustained AV block following inferior infarction. Its half-life is only 0.6-1.5 sec, requiring larger dose with peripheral access, e.g. 6 mg where 3 mg given centrally would do.

In SVT, both AV nodal and non-nodal re-entrant tachycardias (AVNRT and AVRT), the slow pathway is blocked and cycle length alternans may occur. With incremental doses, over 90% effectiveness is seen. The response can also be used to differentiate broad complex tachycardia due to aberrancy from its ventricular look-alike, even though adenosine-sensitive VT needs consideration.

Chest pain induced by adenosine, like that of dipyridamole, can be severe; other side effects include flushing, headache, dyspnœa and cough. Sinus bradycardia or arrest and ventricular arrhythmias are frequent, but almost never require action. AF or flutter may follow cardioversion of SVT; they are less durable than with verapamil. SVT recurs early in 10-30% of cases.

INDICATIONS FOR ADENOSINE

- Narrow complex tachycardia
- Adenosine may be the drug of choice in investigating and or treating such arrhythmia. It may terminate AV-nodal and AV re-entry tachycardia, or reveal underlying atrial flutter or fibrillation
- Broad complex tachycardia

Adenosine may terminate SVT with intra-ventricular conduction block. It will not cardiovert true VT. It may be useful therefore in treatment of regular broad complex tachycardia not thought to be of ventricular origin

DOSE:
• Adults: Incremental 3 mg, 6 mg, 12 mg, 18 mg. Given via large peripheral or central vein followed by saline flush. Some practitioners use 6mg as the first dose.

Please be aware that administration of adenosine may cause the patient to feel very unwell (“hot, flushed, nauseous”) and you should warn the person beforehand if possible.

REFERENCE:

AMIODARONE

This is currently the drug of choice for AF with rapid response in the ICU, given as a 5-10 mg/kg loading dose over 20 – 60 minutes (occasionally bolus) and followed by 1200 mg / day infusion. Its advantage over digoxin is the rapid (within one hour) control of the ventricular rate; unlike digoxin, it may aid return of sinus rhythm. It is also quite successful for cardioversion of SVT although it is rarely used for this purpose.

Acutely, amiodarone blocks the AV node (prolonging the PR interval in sinus rhythm); there is no immediate effect on the sinus rate, QRS duration or QT interval. It prolongs action potential and lengthens the effective refractory period throughout the heart; hence slowing of the sinus rate and prolongation of the QT interval follow.

Amiodarone is best given via a central vein as it causes severe thrombophlebitis. Other side effects are flushing, nausea and transient hypotension. In patients with LV dysfunction, overt failure and shock may occur. In these patients it is wise to omit the bolus and start with an infusion. Long-term side effects are serious and well known; they are rarely of significance in the ICU.

VENTRICULAR ARRHYTHMIAS

The pharmacological therapy is mostly concerned with treatment of VT and prevention of VF. Isolated VEBs, accelerated idiofocal rhythms, escape beats or parasystole are usually treated by mistake.

LIGNOCaine

Lignocaine had for a long time been the drug of choice for the emergency treatment of ventricular arrhythmias. Its use has now largely been replaced by amiodarone.

Its great advantage is its relative lack of toxicity; its equally great disadvantage is the frequent (80-90%) ineffectiveness in VT.

The toxicity is mostly on the CNS, with slurred speech, twitching and seizures; a rare change in intraventricular conduction is usually trivial, but interesting.

The standard dose is 75 mg, followed by 2-4 mg / min infusion in 5% Dextrose.

AMIODARONE

Is effective for sustained monomorphic VT and has some activity in VF. It is the drug of choice for VT in ICU.

SOTALOL
Sotalol, in addition to its amiodarone or bretylium-like class activity, is also a non-selective β blocker (in its l-isomer). It prolongs QT and PR intervals and appears to produce more episodes of torsade de pointes than amiodarone (but probably less than flecainide). It is excreted unchanged in the urine.

A loading dose is 0.5-1.5 mg/kg over 10 minutes, followed by infusion of 0.2-0.4 mg/kg/hour in 5% Dextrose or by oral tablets. It is a significant negative inotrope; some VT patients in the emergency department setting had to be shocked “at the end of the needle”. A lower initial dose is prudent. Other side effects, like asthma, are shared with β-blockers. On the other hand, the β blockade is a reason for its being the drug of choice for VT in patients with IHD.

Its use in the treatment of supraventricular tachycardias is not recommended.

**MAGNESIUM**

A major indication is true torsade de pointes VT, where 2-4 g bolus is followed by 3-20 mg / min infusion. Magnesium is packaged in 5ml ampules containing a 50% solution of magnesium sulphate. Each 5 ml ampule 50% MgSO4 contains 2.5 grams Mg = 20 mEq Mg = 10mmol Mg. This should be diluted to at most a 20% solution for intravenous administration.

Polymorphic VT with normal preceding QT is usually seen in the setting of acute ischaemia sustained monomorphic VT very rarely respond to magnesium. Amiodarone, β-blockers and urgent revascularisation are the best strategy here.

**PHENYTOIN**

Beside digoxin-FAB, diphenylhydantoin is the drug of choice for VT caused by digoxin toxicity; it is also effective for digitalis-induced paroxysmal atrial tachycardia with block, but less so for non-paroxysmal junctional tachycardia. It is best given, as 100 mg boluses every 5 min; the usual antiarrhythmic dose is approximately 700 mg (beyond 1000 mg it is unlikely to succeed).

**COMMONLY ENCOUNTERED ANTIARRHYTHMIC AGENTS**

<table>
<thead>
<tr>
<th>Agent</th>
<th>Administration</th>
<th>Uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>Acute:</td>
<td>Drug of choice for AF with rapid ventricular response</td>
</tr>
<tr>
<td></td>
<td>5 mg / kg (commonly 300 mg) IVI over 30-60 minutes</td>
<td>Drug of choice in ICU for ventricular tachycardia</td>
</tr>
<tr>
<td></td>
<td>Followed by 1200 mg over the remainder of the next 24 hours</td>
<td>Drug of choice in treatment of ventricular fibrillation</td>
</tr>
<tr>
<td></td>
<td>Chronic: decremental dosing</td>
<td>Highly irritant given IVI into peripheral veins</td>
</tr>
<tr>
<td></td>
<td>200 mg 8hrly po for 1 week</td>
<td>Diverse and relatively common side effects with prolonged use</td>
</tr>
<tr>
<td></td>
<td>200 mg 12 hrly po for 1 week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>200 mg daily po as long as</td>
<td></td>
</tr>
<tr>
<td>Drug</td>
<td>Dose and Administration</td>
<td>Best Proven Indication</td>
</tr>
<tr>
<td>----------</td>
<td>----------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Magnesium</td>
<td>5-10 mmol IVI slow bolus (1 ampoule = 10 mmol = 2.5 g)</td>
<td>Best proven indication in torsade de pointes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>May be helpful in treatment of some supraventricular tachycardias, such as multifocal atrial tachycardia and atrial fibrillation</td>
</tr>
<tr>
<td>Adenosine</td>
<td>Incremental dose IVI until desired effect:</td>
<td>Drug of choice in version of AVNRT and AVRT</td>
</tr>
<tr>
<td></td>
<td>3 mg → 6 mg → 12 mg → 18 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rapid push with flush</td>
<td></td>
</tr>
<tr>
<td>Verapamil</td>
<td>Bolus:</td>
<td>Prior to adenosine was drug of choice for AVNRT or AVRT</td>
</tr>
<tr>
<td></td>
<td>5 mg IVI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>if ineffective follow with 10 mg 10 minutes later</td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>Loading dose:</td>
<td>May assist in rate control for stable patients with CCF</td>
</tr>
<tr>
<td></td>
<td>0.25 to 0.5 mg × 3 doses, 4 hours apart</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total dose 1.0-1.5 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IVI or PO</td>
<td></td>
</tr>
</tbody>
</table>
The Registrar on duty is expected to accept the patient on return from theatre. The Duty Cardiac Intensivist should also be notified of new admissions arriving.

The patient’s pre-operative condition taken together with intra-operative events will usually define the parameters required for a given patient.

It is important to document and communicate clearly any expected deviation from the normal pathway of post-operative care.

**ADMISSION OF A CARDIOTHORACIC PATIENT**

On accepting a patient into the unit the following must be clarified in addition to a general admission:

Pre-operative morbidity including:

- Actual renal function (elderly people with normal creatinine may have less than 50% residual renal function as calculated by Cockroft and Gault)
- Left ventricular function and effort tolerance
- Drug history, particularly the use of anti-platelet therapy leading up to surgery
- Co-existent vascular disease, particularly neurovascular disease (e.g. carotid artery, previous TIA / CVA’s)

Intra-operative management issues:

- Number of grafts, vessel harvesting, valve replacement (prosthetics or tissue)
- LV appearance and behaviour
- Fluid loading behaviour on table (i.e. some patients respond to higher “filling pressures”)
- Surgical haemostasis: The surgeon may predict a patient will ooze, and therefore be willing to accept different blood loss parameters prior to actioning re-opening of the chest
- Review postoperative ECG and CXR

**RESPIRATORY CARE**

**DEFAULT VENTILATOR SETTINGS ON RETURN TO ICU**

The nursing staff will prepare a ventilator (in consultation with the Cardiac Anaesthetic Staff delivering the patient) generally consistent with the following:

- FiO2 as decided by Cardiac Anaesthetic Staff
- Mode: SIMV/IPS, at rate 10 breaths / min, 8ml/kg tidal volume, PEEP 5
Nursing Staff change to IPS 10/5 after initial observation period and ascertain whether respiration adequate.

## EXTUBATION

Once the patient is awake and comfortable they may be converted to spontaneous ventilatory modes.

Extubation may be considered once the following criteria have been met:

- Awake, comfortable patient
- Normothermic (Temp > 36.0 °C)
- Cardiovascular stability: Allowing only minimal inotropes, but accepting continued requirement for cardiac pacing
- Adequate gas exchange: PaO2 > 70 mmHg on FiO2 > 0.4, with normal PaCO2
- Normal acid-base status: pH 7.35-7.45, HCO3- > 20 mmol/L, BE of -5 mmol/L to +5 mmol/L
- Minimal Bleeding: < 100 ml/hr ideally. Do not extubate patient if blood loss > 200 ml/hr

## MANAGEMENT OF BLEEDING

### INTRODUCTION

A set of baseline bloods including clotting profile are performed on accepting the patient from theatre. A TEG (thromboelastogram) may have been performed which may assist component transfusion therapy. Standard coagulation tests remain the main indicators for component therapy in the bleeding post cardiac surgical patient.

### LIMITS FOR INITIATING REVIEW BY CARDIAC SURGEONS

Alert cardiac surgeons if the following is exceeded (note the threshold for this is less than that practiced in some other centres).

- 200 ml of blood in intercostal drains in the first hour in ICU
- 400 ml of blood in any given hour thereafter would be indicative of surgical cause of bleeding and the surgeon should be notified immediately
- 200 ml of blood per hour for any 2 consecutive hours
- A significant drop in Hb without excessive drain output indicates occult bleeding (eg into chest cavity, abdomen)

### THERAPEUTIC INTERVENTIONS

In the presence of bleeding, the patient should be warmed, acidosis addressed and treatment should be aimed at correcting the deficit ie:
- TCT high, consider heparin reversal with protamine (TCT may be elevated in other conditions). Heparin rebound may occur suggestive by initially normal APTT/TCT which on repeat testing is subsequently abnormal.

- INR > 1.5 or APTT > 1.5 times normal: Consider FFP as first line treatment.

- Fibrinogen < 1.0 consider cryoprecipitate.

- Platelets should be transfused at a low threshold following surgery with CPB even if quantitatively normal, especially with preoperative aspirin or clopidogrel use.

The role of DDAVP and other anti-fibrinolytics is not yet clear. Ask the Duty Intensivist prior to administering these products.

Special care is needed in those with inherited bleeding disorders and a discussion with the haematologist may be necessary.

Avoid hypertension, as it may exacerbate bleeding.

Factor VIIa should only be considered in refractory bleeding once coagulopathy has been treated and a surgical cause for bleeding addressed. This must be discussed with the oncall intensivist. There is concern FVIIa is associated with graft occlusion and thromboembolism but there is no definitive evidence to support this.

If no evidence of bleeding exists, treatment of coagulation abnormalities is not recommended.

**TRANSFUSION**

Transfusion threshold in an uncomplicated cardiac patient with complete revascularisation is 70g/L. A lower threshold to transfuse should be considered eg 80g/L in those with on going bleeding, evidence of incomplete revascularisation or ischaemia.

**REFERENCE:**


THROMBOELASTOGRAPHY (TEG)

The TEG® device monitors the interaction of platelets within the fibrin mesh of the clot during clot formation and lysis and so monitors haemostasis as a dynamic process. It is frequently monitored during cardiothoracic surgery and the immediate post operative period.

TYPICAL TEG TRACING

TEG® PARAMETERS:

R – measures the time until the onset of clotting; this is the point at which all other coagulation tests stop measuring. An elongated R represents a factor deficiency and can be corrected by administering FFP.

K - a measure of the rapidity to reach a certain level of clot strength

? - measures the rapidity (kinetics) of fibrin build-up and cross-linking, that is, the speed of clot strengthening. K and ? both measure similar information and both are affected by the availability of fibrinogen. An elongated K and reduced ? therefore represents a low level of fibrinogen and can be corrected by administering CRYO

MA - measures the maximum amplitude. A direct function of the maximum dynamic properties of fibrin and platelet bonding via GPIIb/IIa and represents the ultimate strength of the fibrin clot. A small MA represents thrombocytopenia or platelet dysfunction and can be corrected by administering platelets.

CI - Coagulation Index is the linear combination of the above parameters.

LY30 and LY60 measure the rate of amplitude reduction 30 minutes and sixty minutes after MA. This measurement gives an indication of the stability of the clot. LY30 greater than 7.5% represents hyperfibrinolysis and may be corrected by administering antifibrinolytic drugs such as Amicar®, tranexamic acid.
Aspirin should be given for patients as soon as oral intake is possible and chest drainage is <100ml/hr – often Q6H post return from theatre. Initial dosage given should be 300mg then 100mg daily thereafter.
Clopidogrel should be given in the second post operative day for those patients with diffuse coronary artery disease and acute coronary syndrome due to benefits in terms of improved early graft patency and reducing stroke - 75mg/day.

**HYPOTENSION**

If you are concerned that the patient is about to have a cardiac arrest (or if they have had one), a “Cardiac Reopening” Call should be put out via the operator. Only brief CPR should be given while preparation for re-sternotomy is made. Re-sternotomy should be performed within five minutes of arrest, by staff attending the patient at the time, usually ICU Staff.

Patients should have their Blood Pressure maintained at a mean arterial pressure (MAP) of 70-90 mmHg, unless otherwise specified by the surgeon and agreed to by the ICU specialist. Patients with longstanding mild hypotension (often with long-standing valvular disease) may be tolerant of MAP’s as low as 60mmHg.

Only in exceptional circumstances and with agreement of the Duty Intensivist should a MAP of < 60mmHg be allowed to persist or be aimed for.

Where a patient is returned to the ICU on an infusion of inotropes or vasopressor, clear direction should be sought as to their indication and limits.

Where hypotension is not seen to respond to simple measures and checks given below consult the Duty Intensivist without delay. Where cardiac index remains low despite adequate volume resuscitation a cause for low output should be actively sort and then inodilating agents (milrinone, levosimendan) considered.

A request for transoesophageal echo (by the cardiac anaesthetist) should be directed by either the senior registrar or duty intensivist. The on duty intensivist may also be able to perform TOE depending on skill mix on the day.

**APPROACH TO HYPOTENSION (SYSTOLIC BP < 90 MMHG / MAP<60) IN THE CARDIOTHORACIC PATIENT**

**EXCLUDE HYPOVOLAEMIA**

Maintain CVP at least 8-10 mmHg

If a pulmonary artery catheter is in situ then maintain left atrial pressure as estimated by pulmonary capillary wedge pressure > 10-12 mmHg

Or maintain “filling” pressures at levels suggested as optimal during surgery (defined on accepting patient into ICU).

TOE is also a useful tool to visualise filling.
Patients may receive up to 2000 ml of fluid IVI according to the parameters defined above. No further fluid should be given beyond that limit without informing the Duty Intensivist unless the patient is bleeding. In the case of excessive bleeding early Cardiac Surgical consultation is advised.

**MYOCARDIAL (PUMP) FAILURE**

Assess acute ischaemic changes on ECG

Consider tamponade

Cardiac tamponade is a surgical emergency classically indicated by:

- Refractory hypotension, with evidence of hypo-perfusion (cold extremities, diminishing urine output, increasing acidosis, poor response to increasing inotropes), and occasionally pulsus paradoxus
- Diminishing drainage from chest drains
- Increasing right heart pressures (CVP or PA pressure)
- Globular heart on chest X-ray
- Echocardiographic evidence of tamponade

If patient is electrically paced, ensure adequate capture, and rate

If no acutely reversible cause for pump failure is identified:

- Cardiac output should be assessed by PAC, Vigileo, PICCO or Echo
- Inodilators such as milrinone and levosimendan are utilised. A loading dose is often omitted as hypotension is aggravated. Commonly commenced when CI is <2.2

**MYOCARDIAL ISCHAEMIA**

Causes include:

- Incomplete revascularisation
- Closure of graft from spasm, kinking, or thrombus
- The primary indication to consider coronary angiogram in the acute setting is:
- New ST elevation
- Hypotension associated with ST depression or echo evidence of new regional wall abnormality.

Discussion should be initiated with the cardiac surgeon in the first instance, and then to involve the cardiologist if indicated. Unfortunately aggressive medical therapy is limited due to associated hypotension and bleeding risk. Aspirin and a statin should be continued. With significant instability, beyond coronary angiography, IABP therapy can be considered.
CORONARY SPASM

Little evidence exists for the use of prophylactic arteriodilators to prevent coronary artery spasm in arterial conduits. IV GTN and IV milrinone have been used at the surgeons request if arterial conduits are utilised (eg LIMA, RIMA, radial). This can be supported in the absence of hypotension.

Conduit vasospasm is rare. Diagnosis is made only in the cath lab. Patients are often hypotensive, and therapy utilized to dilate vessels exacerbates hypotension.

INTRA AORTIC BALLOON PUMP

This mechanical form of cardiovascular support provides afterload reduction without causing hypotension and enhances coronary perfusion; Indications include myocardial pump failure, predominantly associated with coronary ischaemia. Severe MR and VSD are also indications. Severe AR and lack of adequate vascular access would be a contraindications.

IABP’s post cardiac surgery can be utilised prophylactically in patients with poor LV function. It may be a necessary therapeutic tool in patients who have difficulty separating from bypass and/or who have severe hypotension associated with pump failure.

Positioning of the IABP is distal to the left subclavian artery. This can be confirmed at insertion in the cath lab, or by TOE guidance. On CXR, optimal positioning is approximately 2cm above the level of the carina.

Anticoagulation is not routinely used when pump is at 1:1, but if the ratio is less than this for longer than 6 hours then anticoagulation is recommended (unusual).

Most trouble shooting is performed by the perfusionist on call, or the intensivist.

Monitor pulses hourly and concerns over limb perfusion need to be promptly addressed.

Weaning occurs once cardiac index is deemed to be satisfactory and stable and the majority of inotrope has been weaned. This is at the intensivists discretion.

Once removed, balloon pump exit site requires monitoring for bleeding and aneurysm formation.

In general, removal is performed by the service who has inserted the balloon pump. However, if placed by cardiology to facilitate cardiac surgery, the cardiac surgical team will take responsibility for its removal.

Reference:


VASODILATATION

Patients may have a systemic inflammatory response to the preceding surgery and bypass procedure.

A vasopressor (usually noradrenaline) is required to defend perfusion pressure. Other drugs (eg vasopressin) will be used at the discretion of the Intensivist.
HYPERTENSION

INTRODUCTION
A systolic blood pressure > 140 mmHg MAY be detrimental to the patient ie:

- Increased afterload and myocardial oxygen consumption
- Increased incidence of stroke
- Increased risk of bleeding and vascular graft suture line dehiscence

APPROACH TO HYPERTENSION IN A CARDIOTHORACIC PATIENT
Confirm veracity of reading, exclude pain, agitation, or other reversible cause not requiring antihypertensive.

Consider short acting agent as first line treatment:

- GTN infusion (50 mg in 50 ml dextrose water) 1 mg/hr to 20 mg/hr max
- (Sodium nitroprusside may be considered if GTN unsuitable or ineffective)

Only if high blood pressure sustained or refractory consider:

- Captopril 6.25 mg NG, repeat in 1-2 hours if necessary
- Metoprolol 1 mg IVI, (1mg dose increments to 10 mg max.) prn or 25-50 mg 8 hrly po / NG (or other beta-blocker with which you are familiar)
- Clonidine 15-45 microg prn IVI

ANTIBIOTIC PROPHYLAXIS ADMINISTRATION
Standard prophylaxis is Cephazolin Q8H with weight based dosing:

- <60kg, 1G Dose
- >60kg <120kg, 2G Dose
- >120kg 3G Dose

Dose Reduction in Renal Impairment:

- Serum Creatinine >200, not on dialysis - Halve daily weight based dose
- Serum Creatinine >300, or on Dialysis – Give one third weight based daily dose

Duration of dosing is 48hrs post induction of anaesthesia. Presence or absence of chest drains or central lines is no longer considered to influence the duration of prophylaxis.
Severe Penicillin Allergy (Anaphylaxis):

The incidence of penicillin cross reactivity with cephalosporins is very low. Unless there is a history of severe penicillin allergy, cefazolin is usually used despite a history of allergy.

If severely allergic the patient should have received Vancomycin 25mg/kg to Max 1.5G dose at time of commencement of surgery. One further dose of the same size should be given 12 hours later. In the case of significant renal failure consider delaying or omitting this dose depending on a serum vancomycin level.

PULMONARY HYPERTENSION

Pulmonary hypertension is an important prognostic factor in cardiac surgery associated with increased morbidity and mortality. Normal systolic pulmonary artery pressure is 15-30mmHg with MPAP 9-16mmHg. The mechanism of PHT in cardiac surgery is complex and can result from several mechanisms acting alone or in combination.

The presence of PHT before the operation or appearing during or after it will have an impact on survival mostly through its effect on right ventricular function. The treatment of pulmonary hypertension with right ventricular dysfunction can be summarised in the following way.
Agents used to reduce right heart afterload for the treatment of significant right heart failure with or without significant pulmonary hypertension include the following. If the right heart is failing, it may not generate high pulmonary pressures.

**NITRIC OXIDE**

Technicians must be called in to set up the initial treatment.

When inhaled, nitric oxide dilates the pulmonary vasculature and because of efficient scavenging by haemoglobin has minimal effect on the vasculature of the rest of the body. It appears to increase the partial pressure of oxygen by dilating pulmonary vessels in better ventilated areas of lung moving pulmonary blood flow away from lung segments with low ventilation / perfusion (V/Q) ratios towards segments with normal or better ratios. Nitric oxide therefore reduces pulmonary arterial pressure, improves oxygenation and increases cardiac output.

**INDICATIONS**

- Reduce RV afterload through reduction of pulmonary hypertension
- Salvage therapy in patients with acute RV failure secondary to pulmonary embolus
- Treatment of pulmonary hypertension associated with ARDS

**DOSE**

Commonly commenced at 10-20 ppm – range 5 – 40 ppm. Dose should be tapered slowly over hours to prevent rebound pulmonary hypertension.
Nitric dioxide and met-haemoglobin levels are monitored hourly to assess for toxicity though this is unlikely at clinically used doses.

**ILOPROST**

Prostacyclin analogue with vasodilatory effect. Similar indications to Nitric oxide.

Dilute 1 ampule (50mcg in 0.5mls) to a volume of 10mls in N/Saline (5mcg/ml) Nebulise 3-5mls (15-25mcg) repeated every 2-4 hours depending on effect on pulmonary pressures, RV function and cardiac output. Haemodynamic effects typically last for approximately 1 hour.

Also available in 20mcg in 2mls ampules for nebulising.

**SILDEDANFIL AND INODILATING AGENTS**

Sildenafil may be requested by the cardiothoracic surgeons for certain patients with pulmonary hypertension. This can be prescribed if the patient is not on significant doses of vasopressor.

Levosimendan and milrinone also have vasodilatory effects on the pulmonary vasculature as well as providing positive inotropic effects on the Right Ventricle. Use of these agents is only at the Intensivists discretion.

**PACING**

**INDICATIONS**

Temporary epicardial pacing wires are placed at the time of surgery and provide both diagnostic and therapeutic roles post operatively. Atrial and or ventricular pacing wires are used. Function is variable and trouble-shooting is frequently required. Less commonly failure of pacing necessitates other forms of pacing to be utilized in pacing dependant patients.

Indications include:

- In circumstances where “medical” pacing with a chrono / (ino) trope has failed or is inappropriate
- Symptomatic bradycardias, including β-blocker intoxication
- Complete heart block
- Bifascicular block in association with evolving infarct (particularly anterior)
- Elective: following cardiac surgery in “at risk” patients
- Valve replacement or repair, VSD repair or repair papillary muscle rupture
- Acute myocardial ischaemia
- Persistent A-V block: A temporary pacing wire may be required as a bridge to sequential pacing
- Tachyarrhythmia’s may respond to overdrive suppression pacing
TYPES

- Balloon flotation lead
- Modified PA catheters
- Epicardial leads. Usually placed electively during cardiac surgery. Depending on surgeons preference and patient selection these may be uni or bi-polar and either ventricular alone or atrial and ventricular. Where leads have been placed at the time of surgery, their nature and use should be clearly documented in the patient case notes
- Bipolar pacing lead placed under image-intensifier guidance, usually by cardiology team

PULSE GENERATOR

All staff should familiarise themselves with the code of pacing and the pacing box.

PACING PROBLEMS TROUBLESHOOTING GUIDE

FAILURE TO PACE

No pacing spike left and no paced QRS complex seen:

Oversensing:

- Pacemaker is sensing noise as native QRS complexes and therefore does not pace
- Treatment: reduce sensitivity (can turn down completely (ie increase the mV setting) to asynchronous VOO pacing mode if patient has underlying asystole)

Battery or connection problem:

- Treatment: replace battery or reattach connections
- Checking all connections; changing the connecting cord
- Increasing the output of the pulse generator to maximal current (20 mA)

Using a different wire electrode as the negative (conducting) electrode (reversing polarity).

Unipolarizing the pacemaker by attaching the positive lead to a surface ECG electrode or skin pacing wire.

FAILURE TO CAPTURE

Pacing spike but no paced QRS complex seen.

Lead migration: always watch for epicardial lead migration which may lead to ventricular perforation.

Treatment: can verify lead location with the following:

- CXR
• ECG: Paced QRS should have LBBB morphology with a superior axis

• Unipolar electrogram: this may be obtained by connecting V1 electrode to distal most pacing electrode; normal endocardial contact produces an electrogram with a predominantly negative QRS inflection with ST elevation; positive or a biphasic QRS electrogram may reflect coronary sinus or extracardiac location of lead

• Physiological alterations: myocardial ischemia, hypoxia, acidosis, alkalosis, hyperglycemia, hypercapnia, drug administration

  Treatment: correct underlying abnormality and increase pacing output.

  Battery or connection problem (as above).

**FAILURE TO SENSE**

Pacing spikes seen on TOP of native QRS complex (risk of R on T)

  • Lead migration
  
  • Sensitivity set too low

  Treatment: increase sensitivity (decrease mV setting)

  Do NOT stop the pacing to determine the underlying rhythm. Instead bring down the rate slowly whilst watching for the underlying rhythm to appear

**REFERENCES:**


**POSTOPERATIVE ATRIAL FIBRILLATION (NEW ONSET)**

Post operatively AF is a common problem with an incidence of 20-40% during the patients stay in hospital. Most patients tolerate this well, however, where possible, it is ideal to maintain sinus rhythm.

**NEW / PAROXYSMAL AF**

Consider cardioversion.

  • If haemodynamically compromised, (electrical) DCCV
  
  • If CVS stable, Chemical cardioversion with amiodarone. 300mg load and +/- 900mg infusion over 24 hours

Additional rate control with digoxin or beta-blocker can be utilised but beware of the risk of bradycardia and conduction block in conjunction with amiodarone.
# KNOWN CHRONIC AF

Accept and rate control.

Options include B blockers, digoxin, Ca channel antagonists and amiodarone.

# POST OPERATIVE ATRIAL FIBRILLATION PROPHYLAXIS

This is not routinely used in all patients.

B blocker therapy and amiodarone has been shown to reduce the incidence of post-operative atrial fibrillation, but its use is often limited by hypotension.

Atrial pacing is often requested by specific surgeons. This is also shown to reduce post op AF, most effectively in conjunction with beta blockade. The use of statins and corticosteroids has also shown some benefit.

# INTRA-AORTIC BALLOON COUNTERPULSATION (IABP)

IABP’s are used at times in the unit, usually in patients returning from cardiac surgery, but occasionally as an optimising procedure prior to surgery or in severe potentially reversible cardiogenic shock.

IABP study days and education sessions occur from time to time and are co-ordinated by the Unit Nurse Educator. If you are unable to attend one of these sessions you should familiarise yourself with the equipment by asking senior staff (including nursing staff) and consulting the unit IABP introduction folder kept in unit 1 area.

Usually IABP’s are sited by the Cardiothoracic Team or a Cardiologist.

# INDICATIONS

- Ischaemic Heart Disease
- Low cardiac output states following cardiac surgery
- Cardiogenic shock associated with angiography, stenting or PTCA
- Acute mitral incompetence (papillary rupture) or VSD associated with septal infarct
- Myocardial disease
- Severe contusion
- Myocarditis with cardiac failure
- Cardiomyopathy
- Severe beta blocker overdose
CONTRA-INDICATIONS

ABSOLUTE

- Aortic regurgitation
- Aortic dissection or unstable aneurysm

RELATIVE

- Severe peripheral vascular disease
- Tachyarrhythmias
- Coagulopathic states

TRIGGER

The balloon inflation may be triggered in a number of ways:

- ECG: Inflation at peak of T-wave and deflation before next QRS
- Arterial waveform
- External pacemaker

TIMING

Check balloon inflation against pressure wave-set to peak of dicrotic notch.
Check balloon deflation against ECG-prior to QRS complex.
Check diastolic augmentation on pressure wave.
Select augmentation ratio 1:1 or 1:2.

MAINTENANCE

Heparin is not needed when balloon is on 1:1. Therefore the ratio should stay on 1:1 except when actively weaning.

Control CXR: the tip of the IABP is radiologically opaque and should be sited at 2cm above level of carina.
The limb distal to the insertion site should be monitored neurologically and for adequate distal perfusion.

IABP FUNCTION DURING ARRHYTHMIAS

Arrhythmias markedly affect IABP function and they should be actively treated.

- Ectopics: IABP should remain on ECG trigger, causing automatic deflation on an ectopic
- Atrial Fibrillation: move deflate slide to far right, which cancels automatic R-wave inflation
• VF / VT: proceed as per ACLS guidelines, IABP mechanism is not affected by cardioversion

Cardiac arrest in a patient with an IABP in-situ: proceed to external cardiac massage

• Where CPR or arrhythmia is associated with effective cardiac output: Change IABP to pressure trigger
• No cardiac output with CPR or arrhythmia: Set internal IABP mode to fixed rate of 40 inflations per minute and 20 ml augmentation

WEANING

The IABP should only be removed once the patient has stabilised and the Duty Intensivist and Cardiothoracic Surgeon / Cardiologist agree.

Generally the augmentation rate is sequentially decreased to 1:3 and then removed.

If the patient has been anti-coagulated then the heparin should be discontinued for 3 hours prior to removal of the IABP.

CATHETER REMOVAL

Do not turn off the IABP and leave in situ.

Majority are removed by the inserting team ie. cardiothoracic department.

If open Arteriotomy has been used, surgical closure will be necessary.

Disconnect IABP tubing but do not manually aspirate the balloon before withdrawal.

Withdraw balloon until resistance if felt (balloon tip is at the distal end of the sheath), then remove the sheath and balloon in one movement.

Apply manual pressure until haemostasis achieved. Do not apply occlusive dressing.

If pressure bag or “fem-stop” pressure device applied then this must not be done in a way that might obscure ongoing haemorrhage.

COMPLICATIONS

• Limb ischaemia
• Haemorrhage
• Infection
• Aortic or femoral dissection
• Aortic arch vessel or splanchnic arterial occlusion if balloon improperly sited
• Thrombocytopenia
• Balloon rupture with gas embolism
EXTRA-CORPOREAL LIFE SUPPORT (ECLS) IN ADULTS

INTRODUCTION

ECLS is mechanical support of circulation and/or gas exchange. While not especially difficult to implement, it is highly resource intensive (staff, disposable costs, blood product usage). Its implementation may impact on our ability to care for other patients, and therefore deserves careful consideration before implementation.

Indications are either refractory cardiac failure or life-threatening failure of gas-exchange refractory to non-mechanical intensive care measures. These conditions clearly may co-exist. It is the adequacy of cardiac function that dictates the method employed.

ECLS should not be planned electively if patients are to be managed in Waikato ICU. Patient outcome-volume relationships are assumed to exist that mean the minimum volume of patients in a centre offering ECLS should be greater than 6-12 per year.

It is difficult to anticipate all patients who may require ECLS post-cardiotomy. It is also unlikely that CVICU would accept a post-cardiotomy patient on ECLS. Finally, it is unlikely that a Cardiac Surgeon here would want to transfer a patient to another centre post-operatively. It is from this “triangle” that a practical patient-centred outcome must be found.

However, ECLS may have to be initiated on an emergency basis, even prior to transfer to CVICU at ACH or in postcardiotomy patients in the Operating Room who will need to be managed in this ICU. In both these cases, the assistance of Cardiac Surgery and Perfusion is essential.

All potential ECLS cases should be discussed with CVICU at ACH as soon as possible to allow transport of patient prior to ECLS for logistic reasons.

INDICATIONS FOR ECLS

Hypoxia. Fulfilling both criteria below:

- PaO2 : FiO2 ratio < 60 measured on at least 2 occasions 2 hours apart, despite appropriate ventilator management (including recruitment manoeuvres, patient positioning, NO etc).
- Supercarbia: When hypercarbia considered unacceptable in acute situation (asthma)

Reversible circulatory failure: Veno-arterial ECMO may rarely be implemented when reversible circulatory failure is present (cardio-depressant drug overdose or post cardiotomy).

EXCLUSIONS TO ECLS

Treatment failure is associated with:

- Age > 50 years (not absolute)
- Presence and severity of other organ dysfunction
- Prolonged, non-lung protective ventilation (>7 days)
- Certain lung or cardiac pathologies
**ACTIONING ECLS**

The patient must be discussed with the Intensivist on duty at CVICU (also contactable through 0800 ADULT ECMO). The referral should be made by the Intensivist on duty (or if applicable, the Senior Registrar) at Waikato. CVICU will want details including:

- Patient details: name, age, height, weight, blood transfusion restrictions
- Clinical Details: clinical summary, detailed knowledge of current ventilation status and good understanding of ventilation history, cardiovascular assessment in detail, neurological status, infection status, blood results

**IF ACCEPTED FOR ECLS THE MOBILE TEAM WILL REQUIRE:**

- Four units crossmatched RBC’s in chilly bin to accompany patient to CVICU
- Platelets available if count < 100
- FFP available if INR>1.5
- RIJ access to be preserved-if cannulated, leave in situ but establish infusions elsewhere
- Establish large bore iv access
- Ask for a scrub nurse from Operating Room
- Get large set-up trolley (e.g. from Operating Room)
- Sedate/paralyse patient
- De-prone patient if advised
- Photocopy all notes
- Arrange for Radiology to send images to Radiology at ADHB
- Make relatives to be available to ECMO team
- Advise relatives of how to get to ACH

**STANDARD OPERATING GUIDELINES FOR ECLS**

The most current ELSO guidelines will be the cornerstone of ICU involvement in managing ECLS patients.


For detailed guidelines consult the Cardiac Technicians ECLS manual.
STANDARD TRANSFUSION PRACTICES

• Red Blood Cells:
  o Haemoglobin concentration is maintained above 120 g/l to minimise pump speed required

• Platelets:
  o Platelet count maintained above 80 x10⁹/l.
  o Platelet count should be checked q6h, or more frequently if platelet count drops severely between counts. In this case a cause must also be sought (HITS, alloimmunisation)

• Coagulation:
  o Heparin should be prescribed to maintain ACT at 180-240s. ACT should be the target rather than APTT
  o Coagulation screens should be performed b.d. and fibrinogen levels maintained above 2.5 g/L

VENTILATOR MANAGEMENT

Less intensive ventilation is possible if ECMO is used as some CO₂ will be removed. Adjust minute ventilation down as possible.

ASSESSMENT OF OXYGENATION

Pulse oximetry should be measured from the right hand preferably or either ear, but not the feet or left hand as blood mixing in the aorta may overstate oxygenation to the upper body.

A right radial arterial catheter is preferred.

ASSESSMENT OF CANNULATED LIMB PERFUSION

A limb that has an arterial ECMO cannula inserted should have palpation of the pulses and if required doppler assessment hourly with clear documentation of same.

If a previously felt or detected pulse disappears urgent assessment is required, particularly if there is new pulse asymmetry.

At the time of writing this document, it is unclear which surgical service should be notified of this development first. Since ECMO is nearly always initiated by the cardiac surgical service who are also responsible for cannulation, it is suggested that they be urgently notified first of suspected limb ischaemia, with an expectation that corrective action is determined within an hour of detection.

REFERENCES:

Alpard K A, ZwischenbergerJB. Perfusion 1998; 13: 3-15

INTERCOSTAL CATHETER / UNDERWATER SEALED DRAIN

INSERTION

Local Anaesthesia is mandatory in awake patients, and should be used in sedated patients.

Strict aseptic technique.

28F catheter inserted into 3-4th intercostal space, mid-axillary line (within “triangle of safety”), using blunt dissection as described and recommended in the ATLS guidelines.

The catheter must be guided through the ribs without use of sharp instruments (preferably finger). Trochar aided insertion techniques are not acceptable.

MAINTENANCE

Drains placed in un-sterile environs should be removed as soon as possible.

Drains should remain in-situ until radiological resolution has occurred and there is no further bubbling or drainage of significance (< 150 ml / 24hrs).

Drains placed electively in theatre are the responsibility of the surgeon.

COMPLICATIONS

• Incorrect placement
• Pulmonary laceration
• Pneumothorax
• Bleeding as a result traumatic drain insertion (intercostal or, lateral thoracic artery, lung etc)
• Empyema
NEUROSURGICAL GUIDELINES

NEUROTRAUMA

The effective management of neurotrauma relies upon early notification of the neurosurgical team, and close liaison at all times.

For obvious reasons we have no control over the magnitude and mechanism of the primary injury, however we can influence patient outcome by preventing a secondary insult through hypoxia, hypotension, or electrolyte / metabolic derangement.

ACUTE TRAUMA RESUSCITATION

- Safe retrieval and transport around the hospital, and during emergency surgery.
- Cardio-pulmonary / renal / metabolic homeostasis.
- Maintenance of cerebral perfusion.

MONITORING THE HEAD INJURED PATIENT

- Real time (invasive) arterial blood pressure monitoring
- CVC accessed and pressure transduced
- ICP monitoring

There is general consensus regarding the benefit of generic ICU monitoring (arterial oxygenation, blood pressure etc) to prevent secondary brain insult. The role of intracranial pressure monitoring appears accepted, what is less clear is the threshold for therapeutic intervention and the benefits thereof. Where possible, an ICP maintained less than 20 mmHg in adults and lower in children seems beneficial. An increase in ICP despite active measures would normally lead to an attempt to manipulate cerebral perfusion pressure (generally maintained > 60 mmHg). More advanced (and experimental) monitoring devices are sometimes used, including brain tissue oxygen tension (Licox) and jugular venous oxygen saturations, but they are not routine.

INDICATIONS FOR ICP (INTRA-CRANIAL PRESSURE) MONITORING

- Severe closed head injury GCS < 8 / 15 after adequate resuscitation
- Abnormal CT scan head (haematoma, contusion, oedema, effaced basal cisterns) and GCS<8
- Consider in patients where cerebral status cannot be determined for other reasons (ie sedation for ventilation in polytrauma) and CT brain shows features of structural damage. ICP monitoring is not required if CT brain is normal and there are no radiological features of raised ICP
- Consider in patients with a normal CT scan in the presence of a GCS < 8 if the patient is older than 40 years, has motor posturing, or is prone to marginal hypotension (SBP < 90 mmHg)
- Monitoring hydrocephalus/long standing raised ICP (shunt malfunction, BIH). Shunt malfunction and BIH are usually monitored on the ward and usually do not require ventilation or ICU level care
Warning:

It is inevitable the EVD catheter can migrate once the CSF is drained. It may be in an unacceptable position (happens often), and will cause problems when the EVD catheter is used to measure ICP.

If the catheter does not drain CSF freely (it may pulsate like p wave) and a CT scan shows malposition of the catheter a separate ICP bolt would be justified to measure the pressure. Measuring CSF pressure from a blocked catheter will not provide useful information!!

**VENTILATION OF THE HEAD INJURED PATIENT**

Maintain $P_{a}O_{2} > 10.0$ kPa

Maintain normocapnia: $P_{a}CO_{2}$ between 4.5-5.5 kPa. Hyperventilation to $P_{a}CO_{2}$ as low as 3.3 kPa may decrease ICP temporarily, however there is a short term trade off in cerebral blood flow, and after 6 hours tachyphylaxis occurs with a potential hyperaemia on correction to a normal $P_{a}CO_{2}$. Hyperventilation should not be used therefore unless it forms a short term bridge to definitive treatment (i.e. impending surgery).

Low level PEEP (<15cm H2O) does not affect outcome of head injury adversely, and may be beneficial in the prevention of secondary pulmonary pathology.

With lowered GCS ICP monitoring would be standard.

**HAEMODYNAMIC PRIORITIES**

Maintain perfusion pressure:

- Mean arterial pressure (MAP) > 80 mmHg in the absence of an ICP monitor

- Cerebral perfusion pressure (see algorhythm below) > 60 mmHg where ICP is being monitored (CPP = MAP-ICP). Note may be >70mmHg for first 24 hours

Avoid inotrope or vasopressor use until patient adequately fluid resuscitated.

**FLUID MAINTENANCE**

Aim for euvoalaemia – most importantly avoid hypovolaemia.

Use crystalloid (usually normal saline) unless a specific contra-indication exists.
OSMOTHERAPY

Indications:

- Sustained rise in ICP (>10 minutes) despite adequate sedation and normal carbon dioxide level
- Signs of trans-tentorial herniation
- Progressive neurological deterioration not attributable to systemic pathology

The patient should be euvolaemic prior to initiating osmotherapy.

Hypertonic saline: 10-20 ml of 23.4% saline to a serum Sodium concentration of 155-160mmol/L - first line therapy at Waikato ICU – ease monitoring, less diuresis and possibly less rebound. Mannitol 0.25 g / kg or (100 ml of 20%) up to 1g/kg – often used in ED. Measured osmolality should generally not exceed 320 mmol/l with mannitol.

Prolonged serum hyperosmolality will promote intracellular generation of “idiogenic osmoles” leading to a rebound in cellular fluid uptake (and ICP) if osmolality is allowed to correct rapidly beyond day 3 of therapy.

HEAD AND NECK POSITIONING

The neck should not be flexed excessively with care taken to ensure tapes securing the ETT tube are not too tight around the neck. The cervical collar should also be loosened and the head of the bed elevated to 45 degrees.

SEDATION

First 24-48hrs: It may be appropriate to use a short acting agent such as propofol and remifentanil to facilitate repeated neurological assessment, particularly where no ICP measurement exists. In those patients managed with ICP monitoring it may be prudent to maintain sedation / ventilation for the first 48hrs or at least until a follow-up CT brain is performed.

- Propofol infusions >4mg/kg/hour can precipitate propofol infusion syndrome. Rate should not exceed 20mls/hr in long-term use
- Labile neurogenic hypertension, sympathetic storming or emergence agitation: Consider β-blockade or clonidine, or opioids to treat possible withdrawal
- Benzodiazepine infusion – longer-term sedation to avoid propofol infusion syndrome or in addition to propofol / remifentanil
- Barbiturate use: Phenobarbitone may be used at intensivist discretion

OTHER MANAGEMENT ISSUES RELATING TO NEUROTRAUMA

STEROIDS

Has not been proven useful and likely harmful in the trauma setting.
ANTIBIOTICS

- A single dose of antibiotic is sufficient to cover insertion of monitoring catheters
- A fractured base of skull is not an indication for antibiotic prophylaxis in the absence of a CSF leak
- Routine CSF sampling should be avoided unless the patient is being treated for CSF infection or if infection is suspected
- Intrathecal antibiotics should be given by neurosurgical staff

SEIZURE PROPHYLAXIS

Waikato Neurosurgeons have asked for the following patients to have prophylactic phenytoin:

- Moderate head injuries with haematomas, and contusion
- Depressed skull fracture with dural tear, CSF leak and/or cortical injury

THROMBOPROPHYLAXIS

All patients should be fitted with thigh length ECS, see section on anticoagulants for details. Patients unsuitable for drug prophylaxis should have SCDs on continuously. Consider IVC filter early if likely to have LMWH contraindications.

STRESS ULCER PROPHLAXIS

Consider if patient likely to be ventilated for > 48hrs, and not tolerating enteral feeding.

TEMPERATURE CONTROL

Hyperthermia should be avoided and treated. Hypothermia may be induced to lower intra-cranial pressure. This is a consultant led decision.

TIGHT GLYCAEMIC CONTROL

This is currently not practised in neurosurgical patients requiring ventriculostomy or ICP monitoring (i.e. as a marker of significant brain injury) because of microdialysis studies showing evidence of cellular distress at low normal glucose levels.

REFERENCE:


INTRODUCTION

Insertion of an ICP monitor is performed by neurosurgical staff.

MONITOR SET-UP AND CALIBRATING THE SENSOR

- Connect the microsensor to the ICP express cable without touching the transducer tip
- Place the tip of the microsensor just under the surface of sterile water placed in the microsensor pack tray
- Now switch on the unit
- Press the P0 button on the panel. A reference number will appear. Record this in the patient records and on the white microsensor
- Press the menu button- an ICP reading should appear with an appropriate wave form

Calibration with the main monitor can be performed at a suitable stage using the 20’ or 100’ button as appropriate

COMPLICATIONS

Apart from the procedure itself, infection remains the main risk. The manufacturers of the Codman system claim an infection rate of 1.5 episodes per 100 days use.
CEREBRAL PERFUSION PRESSURE ALGORITHM

Optimise Cerebral Perfusion Pressure - Goals
1. Maintain euvoilemic using IV crystalloid or colloid
2. Ensure normocarbia: PaCO₂ 35-40mmHg
3. Maintain measured serum osmolality between 310-320 mmolL⁻¹
4. Commence inotrope infusion to a Cerebral perfusion pressure (Mean arterial pressure - intracranial pressure) > 70mmHg.

ICP

If ICP > 20mmHg > 10 minutes
1. Consider CSF drainage if intraventricular catheter in situ.
2. Ensure adequate sedation with morphine and midazolam (or propofol and narcotic)
3. Exclude contributing factors:
   - Non-neutral head position
   - Check pt 30 deg. head up
   - Keep PaCO₂ approx 35mmHg
   - Maintain normothermia
   - Occult seizure activity
   - Obstructed venous drainage (cervical collars or ETT ties)

ICP not responsive
Urgent CT Scan and alert neurosurgical team
Temporary measures:
Hyperventilation (short term): P₅₅CO₂ 30mmHg
Bolus mannitol (100ml of 20%) or hypertonic saline 10-20ml of 20% per hour
Surgical Intervention

No surgically remedial lesion
Maintain CPP > 70mmHg

If ICP < 20mmHg
Nil further required

If ICP < 25-30mmHg and static, but requiring large doses of catecholamines
Consider resetting CPP at > 60mmHg with guidance of a jugular venous oxygen saturation device

ICP > 25 -30mmHg
Consider:
Barbiturate coma
Induced Hypothermia
Cranectomy or insertion EVD
STATUS EPILEPTICUS

DEFINITION

- Prolonged or repetitive seizures that occur without a period of recovery between attacks greater than 5 minutes
- Refractory status epilepticus refers to ongoing seizures for more than 30 minutes

Serial seizures may occur within a brief period, but as long as the patient regains consciousness in between this is not an indication for ICU admission.

PRINCIPLES OF ICU MANAGEMENT

- Basic resuscitation protocol: Secure Airway, Breathing, Circulation
- Control Seizures using drugs described in table below
- Consider precipitating causes and treat as appropriate:
  - Glucose: administer an empiric dose if in doubt, or estimation delayed
  - Electrolytes: Na, Ca\(^{2+}\), Mg\(^{2+}\), K\(^{+}\), PO\(_4\)^{3-}\n  - Pregnancy – exclude in all potential patients
  - Metabolic derangement: hypoglycaemia, thiamine deficiency, intoxication or withdrawal
  - Known epileptic: review medication compliance and recent changes
  - Intracranial pathology: CVA, tumour, infection
- Prevent secondary insult: Hypoxia, hyperpyrexia, prolonged seizures- rhabdomyolysis.
- CT scan head: where cause of seizure unknown, and of new onset.
- EEG. May be useful where:
  - Pseudoseizures are suspected
  - The patient has complex partial seizures with intermittent generalisation
  - Where muscle relaxants have been administered to the patient
  - Continuous EEG is not currently available.
- Lumbar Puncture: LP’s are generally not indicated.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Comment</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam</td>
<td>First line acute treatment.</td>
<td>5-10 mg prn IVI.</td>
</tr>
<tr>
<td>Midazolam</td>
<td>Consider only in patients with protected airway in ICU.</td>
<td>1-10 mg/hr via infusion.</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>Alternative to Diazepam in status epilepticus</td>
<td>1-2 mg prn IVI as bolus followed by 0.5-1.0 mg/hr IVI infusion.</td>
</tr>
<tr>
<td></td>
<td>Medium to long acting benzodiazepine, beware accumulation.</td>
<td></td>
</tr>
<tr>
<td>Phenytoin</td>
<td>First line treatment along with Diazepam (or other benzodiazepine).</td>
<td>Loading dose 15-20 mg/kg IVI over 30 minutes with telemetry.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Maintenance 300 mg/24hrs titrated to therapeutic range.</td>
</tr>
<tr>
<td>Valproate</td>
<td>Consider if phenytoin not suitable or not effective.</td>
<td>15-20mg/kg IV load Q30mins to max 60mg/day then IV infusion 1mg/hr.</td>
</tr>
<tr>
<td>Phenobarbitone</td>
<td>Consider if seizure uncontrolled within 20 minutes. Consider only if benzodiazepine and phenytoin therapy failed.</td>
<td>Loading dose 20 mg / kg over 30 min 1.5 mg/kg/min as infusion.</td>
</tr>
<tr>
<td>Levetiracetam</td>
<td>Particularly for myoclonic status (ie post anoxic).</td>
<td>500-1500mg bd IV. Adjust in renal failure.</td>
</tr>
<tr>
<td>Ketamine</td>
<td>NMDA receptor antagonist, may be helpful when GABA receptor response to other drugs less effective.</td>
<td>Loading dose: 1 – 5 mg / kg Infusion: 1 – 5 mg / kg / hr.</td>
</tr>
<tr>
<td>Propofol</td>
<td>Anaesthetic agent used to control refractory status in the intubated patient.</td>
<td>1-2 mg/kg followed by 2-10 mg/kg/hour.</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>Volatile anaesthetic agent via ANACONDA.</td>
<td>1-2 MAC</td>
</tr>
</tbody>
</table>
Thiopentone: Reserved for failed “standard treatment”, where endotracheal intubation is required.

Loading dose: 5 mg/kg

Infusion: 1-3 mg/kg/hour (approx 150 mg/hr) titrated to EEG activity at the bedside.

Once emergency treatment has been implemented it is expected that the assistance of the neurology team will be sought in adjusting treatment in known epileptics, or those with focal or complex partial seizures.

Levetiracetam is a newer anticonvulsant for Status Epilepticus. The Neurologists have a preference for levetiracetam instead of phenytoin. The Established SE Treatment Trial (ESETT) a randomised controlled trial comparing Valproate and/or Levetiracetem to Phenytoin as second-line treatment is in progress.

REFERENCE:

Agarwal, P, Kumar, N, Chandra, R, et al. Randomized study of intravenous valproate and phenytoin in status epilepticus. Seizure 2007; 16:527


SUBARACHNOID HAEMORRHAGE

INTRODUCTION

Patients will be admitted either electively following planned surgery or as a result of acute aneurysmal rupture, generally with impaired level of consciousness. For patients with a sufficiently good prognosis, aneurysm isolation will be planned to occur within 48 hours of initial rupture.

PLANNING OF SURGICAL INTERVENTION IN PATIENTS WITH ACUTE RUPTURE

- Early (< 3 days):
  - Advantages: Prevents re-bleeding, may assist with reduction in associated vasospasm (blood products removed) and subsequent cerebral ischaemia
  - Disadvantages: More technically difficult, higher risk of intra-operative rupture

- Late (> 11 days):
  - Advantages: Easier procedure. Allows a period of observation, avoiding surgery in potentially non-salvageable patients
  - Disadvantages: re-bleed or rupture. Increased risk of vasospastic complications
CLASSIFICATION OF SUBARACHNOID HAEMORRHAGE

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>GCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Non-ruptured</td>
<td>15</td>
</tr>
<tr>
<td>1</td>
<td>Asymptomatic, minimal headache, slight neck stiffness</td>
<td>13-15</td>
</tr>
<tr>
<td>2</td>
<td>Moderate headache, neck stiffness, neurology limited to cranial nerve pathology.</td>
<td>13-14</td>
</tr>
<tr>
<td>3</td>
<td>Drowsy, confused, mild focal defect</td>
<td>13-14</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate to severe hemiparesis, early decerebrate rigidity</td>
<td>7-12</td>
</tr>
<tr>
<td>5</td>
<td>Deep coma, decerebrate rigidity, moribund patient</td>
<td>3-6</td>
</tr>
</tbody>
</table>

PRINCIPLES OF ICU MANAGEMENT

MONITORING

- Pulse oximetry
- Invasive arterial monitoring
- Central venous access (particularly during administration of nimodipine)
- ICP monitoring in patients returning with a ventricular drain:
  - Instructions for drain height and CSF drainage should be obtained from the surgical team involved
- Neurological observations half hourly:
  - A deterioration in GCS that cannot be easily explained or corrected (eg. sedation) may be due to a re-bleed, vasospasm or hydrocephalus in which case the neurosurgical team should be notified

THERAPEUTIC INTERVENTIONS

Prior to definitive aneurysm management, MAP is kept in the range of 80-100. Following aneurysm isolation, the MAP may be allowed to rise higher and on occasion deliberate hypertension will be used in the management of vasospasm.
Aim to minimise secondary damage due to cerebral vasospasm, using Nimodipine, preferably given orally or gastrically (60mg q4H) or if necessary IV 1 mg/hr IVI (preferably via CVL). Nimodipine administration may precipitate hypotension, in general noradrenaline is used to maintain the desired mean arterial pressure (MAP). Where possible stopping nimodipine should be avoided with dosage reduction preferred if necessary.

Fluid administration:

- Maintain at least normovolaemia using 0.9% saline IVI (generally minimum 2.5l/day)
- Avoid hypovolaemia. Hypervolaemia has been advocated as a means of maintaining cerebral blood flow, however this must be balanced with the respiratory side effects of over hydration.

**GRADE 1 & 2**

EVD intraoperatively and remaining in place for 24 hrs. Majority do not need ventilator support and are managed in HDU. There is no need to place an ICP probe in this situation.

**GRADE 3-5 HIGH GRADE**

Surgery frequently delayed, majority will need ICU/ventilator care. Hydrocephalus if often of concern and hence an EVD is required. Major issues will be vasospasm, on going infarction and oedema. Codman intraventricular monitor (Catheter and ICP probe) or EVD + ICP probe useful to drain CSF and monitor ICP due to spasm, infarction and oedema. If ICP increases and the CT shows more pre operative or post operative oedema, decompressive craniectomy will be considered.

**ADJUNCTS TO TREATMENT**

Direct and chemical (papaverine) angioplasty may play a role in refractory cerebral vasospasm, and should be discussed with Neurosurgeon and Neuroradiologist.

The use of tranexamic acid has been investigated in the prevention of rebleeding after aneurysmal SAH. A meta-analysis of nine trials concluded that antifibrinolytic treatment reduces the risk of rebleeding but does not show any evidence of reducing poor outcome defined as death, vegetative state, or severe disability. Currently administration of 72hrs of tranexamic acid 1G IV Q8H is practiced at Waikato Hospital. This is stopped as soon as definitive treatment (clipping / coiling) is completed.

**REFERENCE:**

APPROACH TO DIAGNOSIS AND MANAGEMENT OF CEREBRAL VASOSPASM

All patients receive nimodipine, continuation of statin therapy\(^1\)
Daily TCD (MCA/CCA insonation)
Adequate isotonic fluid intake\(^2\)

Suspicion of vasospasm on TCD and/or clinical grounds\(^3\)
Other causes considered\(^4\)
Discuss with Neurosurgeon, Radiologist
CT head/CTAngio/CT perfusion based on discussion
Possible Angiogram to follow

Aneurysm isolated
MAP 90-110, fluid challenge unless contraindicated
Consider neuroradiological intervention, especially if no improvement

Aneurysm not isolated (generally poorer grade)
Consider prognosis and treatment options\(^5\)
If no improvement consider IH, or IABP in selected patients\(^6\)

\(^1\) As evidence accumulates, commencement of statins may prove beneficial
\(^2\) Generally 2L normal saline per day
\(^3\) LR×2, MCA flow<200cm/s: increase 5L per 24 hours
\(^4\) Consider systemic disturbance, hydrocephalus, rebleed, generalized cerebral oedema
\(^5\) These are currently unproven treatment options
\(^6\) Most patients with good prognosis would receive definitive treatment prior to vasospasm risk period

REFERENCE:

GUIDELINES FOR PERFORMANCE OF SENSORY EVOKED POTENTIALS

INTRODUCTION
Short and long latency sensory evoked potentials (SEPs) have shown utility for prognostication in severe diseases of the central nervous system. In particular, SEP’s are a very useful adjunct to clinical assessment in patients with hypoxic-ischaemic encephalopathy and traumatic brain injury.

PATIENT PREPARATION
Testing is preferably performed with a core temperature of > 36 degrees centigrade.
Patients not ventilated with controlled modes will require a controlled mode, minimal sedation as below and paralysis (generally 0.1 mg/kg vecuronium will produce up to 30 minutes paralysis, with additional smaller doses sometimes required to complete recording) to reduce artefact during recording.

Heavy sedation should not be used. Long latency potentials are affected by sedatives at high dose. However, a dose of 10ml/hour maximum of propofol would not be expected to significantly delay or abolish even long-latency potentials. Remember that this is a test used almost exclusively when the patient is in coma and one that is performed in outpatients with minimal discomfort.

**RECORDING**

Request is made to the Technicians specifying whether N20’s alone or N20’s and N70’s are to be performed. This is at the discretion of the Intensivist.

- N20’s are recorded using an 8 mV stimulus of the Median nerve delivered at 5Hz for 100 seconds resulting in waveforms averaged from 500 sweeps of 50 ms duration
- N70’s require a 250 ms sweep duration and a 3 Hz stimulus rate to accommodate the recording of the longer latency potentials. 300 sweeps are averaged to derive the waveforms

**PRECAUTIONS**

- An assurance of spinal column stability prior to the procedure is preferred, but not essential
- In post craniectomy patients, the technique may still be performed in the usual way. Ensuring the electrodes are sub dermal only is more important here. Keeping as far away from an actual wound as possible is prudent (personal Communication, James Judson, DCC, Auckland City Hospital, Linda Hill, Charge Neurophysiology Technician, Auckland City Hospital)

**INTERPRETATION**

Clinical correlation is vital. Individual intensivists may appropriately interpret recordings they have requested. There is an expectation within this particular ICU for Intensivists to acquire or maintain some expertise in clinical correlation and interpretation of SEPs.

The threshold for reporting absence of a potential is difficult. Some authorities quote minimal amplitude that needs to be achieved before regarding a potential as present. Other authorities simply report a potential as present if they believe it to be discernible. It is important to note that some potentials occur earlier than 13ms after stimulation, and their presence should not be confused with presence of an N20.

If the Intensivist is not comfortable with interpretation, they may ask for a Neurologist interpretation, either directly by ringing the person concerned then faxing the result, or asking the technician to do so. Either way, this person must be contacted directly to be able to report the test

**REFERENCE:**

SPINAL INJURIES

ICU ADMISSION POLICY

ASSESSMENT AND ADMISSION TO ICU

There are no hard and fast rules about which patients should be admitted, or when, when the issue is purely related to a spinal injury.

Generally speaking patients with mid or high cervical injuries with evidence of spinal cord injury may progressively deteriorate because of swelling in adjacent cord segments over time, or because their respiratory independence is impaired.

It would be reasonable, where there is some expectation of either of these scenario’s occurring, to admit patients to ICU following their initial observation period in the emergency department.

RADIOLOGICAL ASSESSMENT OF THE SPINE

This vexed area remains incompletely resolved.

What is clear is that complete radiological clearance of the cervical spine is required in those patients where clinical examination cannot be relied upon to do so. The question that remains unanswered is what constitutes a reasonable interpretation of complete clearance?

The issue of when to attempt screening clearance of the thoracic and lumbar spine is becoming clearer from the literature, but no guidelines are in current use at Waikato Hospital. An individual decision is thus required for each situation.

The below algorithm gives some guidance as to the process employed at Waikato Hospital.

CRITICAL CARE MANAGEMENT OF SPINAL CORD INJURY

As in other injuries and critical illnesses, priorities revolve around airway, breathing and circulation as the starting point of treatment.

In an emergency or with an unco-operative patient a rapid sequence induction with MILS and avoiding suxamethonium beyond the first 48 hours is the norm.

In the relatively uncommon critical care scenario of elective intubation, awake fibreoptic intubation may be considered. The evidence base for preferring this over careful conventional laryngoscopic intubation seems weak.

The concept of preventing secondary neurological injury in the spinal cord is also accepted despite an incomplete evidence base. This is usually achieved with appropriate fluid loading, but may require vasopressor support.
NB. If the patient has any persistent neurological symptoms or signs they must be seen by ortho in ED.
BURNS

Burn injury is characterised by a hypermetabolic response with physiologic, catabolic and immune effects. Burn mortality is most strongly predicted by: increasing %TBSA burn, increasing age, presence of inhalational injury and delay greater than 2hrs to initial resuscitation.

Severe burn management has become increasingly specialised over time with established referral criteria to tertiary burns centres. Waikato Hospital is designated as one of the four Regional Burns Units in New Zealand by ANZBA (Australian and New Zealand Burns Association). Criteria for consideration of transfer to the National Burns Unit at Middlemore hospital are as follows:

- Burns > 30%
- Patients requiring prolonged ventilation
- Full thickness burns greater than 15% TBSA in the very young or very old
- Electrical burns – high voltage with underlying tissue damage
- Significant chemical burns

BURN ASSESSMENT

Depth can be divided into Epidermal (erythematosus, no blistering, painful):

- Superficial dermal
- Mid dermal
- Deep dermal
- Full thickness

Epidermal burns should not be included in TBSA calculations.

%TBSA can be calculates by;

- Adult Rule of Nines Chart – 11 areas of 9% plus 1% for perineum (usually overestimates)
- Palmer surface- palm and fingers = 1%

Lund-Browder Chart

- Most accurate
- Children or Adults
- Seldom used as difficult and time consuming if not familiar
Paediatric burns TBSA assessment – children have larger head and smaller leg contributions to TBSA. Use palmer surface, paediatric rule of nines chart or Lund Browder Chart. From the age of ten, proportions become the same as the adult.

**EARLY MANAGEMENT**

Cooling in running water for 20mins within 3hrs of injury reduces progression of wound area. Be mindful of hypothermia. Stop cooling if patients body temperature falls below 35 degrees. Chemical burns require copious irrigation for a minimum of 30 mins. Electrical burns require consideration of potential compartment syndrome, cardiac dysrhythmias, muscle necrosis and multiorgan involvement. Early management decisions regarding escharotomies, fasciotomies and debridement should be made in consultation with the Burns/Plastics team on call. Tetanus prophylaxis, appropriate analgesia, venous access and urinary catheter should also be established at this point. Wounds should be covered with clean, dry material or non adherent gauze. Active temperature management with fluid warming and increased ambient temperature may be needed.

**FLUID MANAGEMENT**

Due to increased vascular permeability and oedema formation patients with severe burns are prone to hypovolaemia and subsequent shock. Fluid Resuscitation should be considered in adults with >15-20%TBSA burns and children with > 10% TBSA burns. An estimation of the first 24hr fluid requirement (mls) post burn can be made using the Modified Parkland formula:

- 3-4ml x %TBSA burn x wgt (kg)
- Half given in the first 8hrs. The other half given over the remaining 16hrs

This formula should be used as a guide with further titration of fluid therapy directed by clinical volume assessment, hemodynamics, serum lactate and urine output (0.5ml/kg/hr in adults and 1ml/kg/hr in paediatrics) Hartmans is used as first line fluid therapy.

- Be mindful of “fluid creep” (excessive volume loading)
- Watch for signs of rhabdomyolysis. (myoglobinuria)

**INHALATIONAL INJURY**

Diagnosing inhalational injury is somewhat subjective, based on physical findings such as: facial burns, singed nasal hairs, soot in proximal airways, carbonaceous sputum, wheeze, stridor and voice changes. The decision to intubate should be discussed with the on call consultant if patient acuity permits. Specific considerations include:

- Maximum wound oedema takes place at 12-36hrs after injury
- Prepare for difficult intubation
- Suxamethonium can be used in the first 24-48hrs
• ETT may need dental wiring to secure
• Post intubation bronchoscopic assessment and suctioning
• Consider escharotomies for chest wall burns with poor ventilatory compliance
• Carbon monoxide and cyanide toxicity may also complicate inhalational burns
• NAC and heparin nebulisation for mucolysis and prevention of fibrin plug, accumulation respectively, has been used in some centres

NUTRITIONAL SUPPORT

Severe burn patients are characterized by a prolonged and persistent hypermetabolic response that can lead to muscle wasting, hyperglycemia, and severe cachexia. Resting energy expenditure can increase by more than 100% of usual resting basal metabolic rate following a large burn injury. The commencement of early enteral nutrition (either gastric or or post pyloric) has been seen to have metabolic and clinical benefits for the burn injured patient as well as assist in preventing gastroparesis and/or ileus.

REFERENCES


Australia and New Zealand Burns Association Manual 2012

Standardizing the diagnosis of inhalation injury using a descriptive score based on mucosal injury criteria, C Ikonomidis et al, Burns 2011, Vol 38, Issue 4 513-519


DRUG / TOXIN OVERDOSE

The majority of overdoses are poly-pharmacological and respond to general supportive measures. Overall mortality is low and usually relates to cardio-respiratory arrest and / or uncontrolled seizures prior to admission.

Despite an unreliable correlation between depth of coma and preservation of glottic reflexes, over the last decade emergency departments have become more aggressive at intubating patients.

While specific reversal agents such as Naloxone (opioids) or flumazenil (benzodiazepines) have some short term use, their relatively short half lives restrict their efficacy in definitive treatment.

ADMISSION TO ICU

- Intubated patients
- Uncontrolled seizures
- Coma
- Persistent hypotension
- ECG abnormalities consistent with significant ingestion (may be suitable for HDU monitoring in the absence of other features listed above):
  - Ventricular or supraventricular tachyarrhythmias
  - Sinus tachycardia > 140 / min
  - 2nd or 3rd degree heart block
  - QT-prolongation (preferably index QTc)
  - QRS duration > 0.12ms

GASTRIC LAVAGE

The place of gastric lavage in acute poisoning is debatable, and is only of benefit in the hyper-acute phase of poisoning (< 1 hour).

Patients must be awake with a preserved gag reflex, or already be intubated, failing which the risks and benefits of intubating specifically to perform gastric lavage need to be evaluated.

PROCEDURE

- Insert 16G nasogastric tube (not a large bore sump)
- Instil 1 ml/kg warm water only, and then attempt recovery of the lavage
- Do not continue to instil water until the previous volume has been removed
- Continue until lavage is clear
Charcoal aspiration has a high morbidity and mortality. As for gastric lavage above, this should not be attempted in patients without a safe or protected airway.

Instil 50g as soon as possible and 50g 4 hrly thereafter while indication persists. Co-administration with sorbitol has not been shown to increase efficacy.

In general charcoal should be given in a ratio of 10:1, charcoal dose to drug ingested dose.

**INDICATIONS FOR ADMINISTERING ACTIVATED CHARCOAL**

Virtually all patients presenting with a drug overdose.

**CONTRA-INDICATIONS**

- Elemental metals (lithium, iron)
- Pesticides
- Strong acids or alkalis
- Cyanide
- Late presentations > 4-6 hrs post ingestion

**SPECIFIC OVERDOSES**

The Hospital intra-net site contains a link to “Medline and other Biomedical Databases”, in which directory you will find “Micromedex” which contains both “Poisindex” and “Drugdex” two accessible and readable databases relating to drug and toxin ingestion.

Consult the Duty Intensivist prior to commencing therapy not considered part of basic resuscitation measures.

The lab can measure both methanol and ethanol, but ethylene glycol is sent away and takes several days. Ethylene glycol poisoning is suspected if high osmolar gap (adjusted for ethanol and negative for methanol), calcium oxalate crystals in urine, or a lactate gap.
MANAGEMENT OF THE UNCONSCIOUS, UNDETERMINED OVERDOSE

Acute resuscitation:
- Airway
- Breathing
- Circulation

Immediate adjuncts to consider:
- Hypoglycaemia Rx 50ml 50% dextrose
- Opiates Rx Naloxone ivi 200 mcg increments
- Alcoholics Thiamine 100mg
- Significant Paracetamol ingestion (before 4 hours) Rx N-acetyl cysteine

Patient
- Relatives
- Ambulance staff
- Circumstantial

Exam exclude
- Trauma
- Neurologic disease
- Metabolic Encephalopathy
- Endocrinopathy

ABG

Biochem: ICU profile
- Drugs: Paracetamol (salicylates/theophylline)
- ? Hepatitis Serology
- ? HIV (if strong suspicion)

Routine screen:
- Barbiturates, opiates, benzodiazepines, tricyclics
- Specific if indicated:
  - Cannabis, cocaine

Urine

No Metabolic Acidosis
- Sedatives
- Hypnotics
- Paracetamol
- Theophylline
- Anticholinergics
- Carbon Monoxide
- Organophosphates
- Phenothiazines
- Lithium

Metabolic Acidosis
- Normal anion gap

Increased anion gap
- Osmolar gap
  - <10
  - >10

Lactate Acidosis (check ABG)
- Diabetic Coma
- Salicylates
- Cyanide

Methanol
- Ethanol
- Ethylene glycol

Osmolar gap

Increased anion gap
RENAL REPLACEMENT THERAPY

INDICATIONS FOR DIALYSIS

The threshold for dialysis in a critically ill patient is different from that of an ambulatory ward patient. Mortality in critically ill patients is related to time averaged urea during their stay, so that dialysis should be started earlier with the aim of maintaining an optimal state, rather than cyclical clearance of urea and metabolites.

The presence of two of the following would suggest dialysis should be considered:

- Oliguria < 200 ml / 24hrs
- Oliguria < 50 ml / 12hrs
- Severe acidaemia
- Hyperkalaemia
- Plasma Urea > 30 mmol/L or uraemic syndrome (pericarditis, pneumonitis, bone marrow suppression)
- Plasma Creatinine > 300umol / L
- Pulmonary oedema
- Diuretic resistant cardiac failure
- Anasarca (generalised oedema)
- Selected overdose (salicylates, methanol, theophylline)
- Imminent or ongoing massive blood product use

The attempted removal of cytokines and inflammatory mediators is not yet proven to reduce mortality in humans.

MODES OF DIALYTIC THERAPY IN THE ICU

Standard intermittent dialytic therapy: Although still used in this ICU, it is limited by resource availability and is probably not suitable for use in unstable patients due to hypotension issues.

Sustained low efficiency dialysis: essentially slow intermittent dialysis (SLEDD). Typically completed over 4-6 hours at higher filtration rates. Frees up nursing time and facilitates patients requiring other interventions outside of the ICU. Essentially a hybrid therapy between intermittent “standard” dialysis and continuous modes.

Continuous veno-venous renal replacement therapy (CVVHD): A growing field of therapy in the ICU, this modality has become the mainstay of renal replacement in the critically ill at The Waikato Hospital.

CONTINUOUS RENAL REPLACEMENT: -DEFAULT PRISMA SETTINGS

- Mode CVVHDF
• Blood flow rate 150 ml/min
• Dialysate flow rate Filtration rate 1000ml/hr 50/50 pre and post filter 1000 ml/hr

ANTICOAGULATION

The filter is primed with 5000 units of unfractionated heparin as part of the start up procedure.

If considered safe, a bolus of 2000-5000 units of heparin is administered to the patient IVI at the commencement of dialysis.

There is no evidence that anticoagulation prolongs filter life and prevents clotting in the filter. Anticoagulation is however widely practiced, the ideal aim being to anti-coagulate the filter but not the patient. Therefore:

Heparin 10 000 IU is made up to 50ml with Normal Saline, and starting at 5 ml / hr is infused via stand alone syringe pump “pre-filter”

Regional anticoagulation with Protamine reversal has now been largely abandoned due to problems with rebound bleeding two to four hours after the end of dialysis as the reticuloendothelial system releases free heparin from the protamine-heparin complex back into the general circulation

Bicarbonate containing dialysate is now used at Waikato Hospital. Citric acid-based dialysate has the advantage of reduced clotting in no-heparin dialysis (by lowering serum calcium enough to interfere with the clotting cascade but not enough to cause symptomatic hypocalcemia) but is not yet widely used

Patients with deranged coagulation due to sepsis or low / abnormal platelet function may not require heparin administration at all.

POTASSIUM REPLACEMENT

The haemodialysis counter current should mean that with an effective filter in situ, the plasma exiting the filter has the same potassium concentration as the dialysate entering the filter. Potassium supplementation should therefore only occur in the dialysate fluid.

Standard haemofiltration fluid is bicarbonate buffered and contains \([K^+] = 1 \text{ mmol/L}\). Add \(K^+=\)according to the table below.

POTASSIUM SUPPLEMENTATION IN RENAL REPLACEMENT (CVVHD)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 6.0 mmol/L</td>
<td>Nil to first bag and repeat serum K+ at 4 hours</td>
<td>1 mmol/L</td>
</tr>
<tr>
<td>3.0-6.0</td>
<td>15 mmol</td>
<td>4 mmol/L</td>
</tr>
<tr>
<td>&lt; 3.0</td>
<td>20 mmol to first bag and repeat serum K+ at 4 hours</td>
<td>5 mmol/L</td>
</tr>
</tbody>
</table>
If potassium supplementation exceeds that described above, it should be given parenterally in the normal way until a desired serum potassium concentration is achieved.

**FLUID REMOVAL**

The machine will allow you to set a net fluid removal volume per hour. This is calculated by the machine based on the weight of the ultrafiltrate bag (i.e. gravimetric method), and the set flow rates of replacement and dialysate fluids. The volume to be removed from the patient must be discussed with the Duty Intensivist, and form part of the daily management plan.

**COMPLICATIONS OF CONTINUOUS RENAL REPLACEMENT THERAPY**

- Haemorrhage (and other consequences of exposure to heparin (H.I.T.S.)
- Hypothermia, or masking of hyperthermia (prevention of hyperthermia may be clinically useful)
- Complications of (prolonged) venous access
- Exposure to extracorporeal circuits and filter (activation of complement, sequestration of platelets)
- Air embolism
- Increased requirement for experienced staff, and increased nursing workload

**REFERENCE:**

RENAL DRUGS

GENERAL PRINCIPLES

Acutely ill patients are at risk for developing, or exacerbating, renal dysfunction. Good intensive care practice, and renal care, encompasses:

• Avoiding renal hypoperfusion: ICU patients generally do not have the ability to autoregulate renal blood flow and GFR, as these become increasingly dependent on systemic perfusion pressures. For this reason urinary output is a sensitive marker of total body perfusion, and resuscitation status

• Ensure adequate volume resuscitation

• Avoid renal toxins if possible: aminoglycoside antibiotics, contrast mediums etc

• Consider local complicating conditions: eg. abdominal compartment syndromes

Administration of agents such as dopamine in low dose, or frusemide, may help maintain urine output with some inherent advantages in fluid management. They are not however reno-protective, and their use should be carefully weighed up in each clinical scenario.

DIURETICS

INDICATIONS

• Symptomatic fluid overload without intravascular depletion

• Pulmonary oedema

• Congestive Cardiac Failure / Cor Pulmonale

• Ascitic states where abdominal volume is thought to be a compromising factor

• Hypertension

• Conjunctive therapy in Cardiac failure (not primarily diuretic): ACE-I and thiazide, Low dose (25 mg / day) spironolactone

• Metabolic alkalosis: eg recovering ventilated patients allowed permissive hypercapnoea, prolonged renal replacement therapy with bicarbonate overshoot (ie. acetazolamide)

CONTRAINDICATIONS

• Hypovolaemia

• Anuria: Frusemide in particular acts on the luminal side of the renal tubule. States where there is no, or low, GFR will not respond to drug administration, and may complicate hypotension by direct afterload reduction

• Failure to respond to trial dose
- Drug hypersensitivity: NB Sulphonamides

### COMPLICATIONS

- Hypovolaemia (often hyperosmolar)
- Hyponatraemia or hypernatraemia
- Electrolyte disturbance of K+, Mg2+ and PO43-

### COMMONLY ENCOUNTERED RENAL DRUGS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Administration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frusemide</td>
<td><strong>Bolus:</strong> 20-100 mg PO / IVI prn.</td>
<td>Potent loop diuretic</td>
</tr>
<tr>
<td></td>
<td><strong>Infusion:</strong> 2.5-10 mg/hr (larger doses have also been in used, however this practice is not well documented in standard literature)</td>
<td>More effective administered as infusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Toxicity (deafness and interstitial nephritis) increased with co-administration of aminoglycosides</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓K+, Mg2+ P043-common</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>25-100 mg bd</td>
<td>Potassium sparing diuretic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>May prevent pathological cardiac modelling in low dose</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2nd line agent, diuretic resistant heart failure</td>
</tr>
<tr>
<td>Mannitol</td>
<td><strong>20% solution (200 mg/ml)</strong></td>
<td>Potent osmotic diuretic</td>
</tr>
<tr>
<td></td>
<td><strong>Bolus:</strong> 100 ml prn IVI</td>
<td>May cause hyper-osmolar state, with increased osmolar gap (measured-calculated osm)</td>
</tr>
<tr>
<td></td>
<td><strong>Traditional doses of 0.5-1.0 g/kg body weight are probably excessive</strong></td>
<td>Dose limited by ceiling of 300 mosm/L</td>
</tr>
<tr>
<td></td>
<td><strong>Max dose 3 g/kg/day or see opposite</strong></td>
<td>Limited role: acute head injury with raised ICP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Has been used but has no proven role in Tx of myoglobinaemic /-uric states</td>
</tr>
</tbody>
</table>
Anticoagulation in critically ill patients is a challenging issue, with patients at risk of bleeding diatheses as well as hypercoagulable states. Often a single patient will move through a state with a high risk of bleeding (including surgical sites) to one of high risk of developing venous stasis and thrombosis. The decision to administer anticoagulation is often based on a relative risk-benefit assessment.

Where anticoagulants are contra-indicated, alternative methods should be employed to prevent venous stasis in the lower limbs (graded compression stockings and sequential calf compressors), although it is unclear as to whether these confer adequate protection against thrombosis and embolisation.

As a general rule heparin infusions should be used to effect anticoagulation, titrated intravenously to a therapeutic APTT where this is required, or administered subcutaneously for DVT prophylaxis. Low molecular weight heparins may require measurement of anti-factor Xa to quantify effect, and are more difficult to reverse than unfractionated heparin.

Where any doubt exists with regard the use of an anticoagulant in a given surgical or trauma patient, this should be confirmed with the Surgeon involved.

**INDICATIONS FOR THE USE OF WARFARIN**

- Post operative prosthetic valve (According to cardiothoracic guidelines)
- Previous thrombo-embolism: Selected cases only
- Maintenance of thromboprophylaxis in selected high risk patients only

**INDICATIONS FOR THE USE OF HEPARIN**

- DVT prophylaxis (LMWH)
- Proven venous or arterial thrombo-embolism
- Myocardial ischaemia syndromes
- Prosthetic heart valves
- Prior to commencing oral anticoagulants
- During an acute illness where oral anticoagulation is unsuitable
- Atrial Fibrillation -sustained
- Intra-Aortic Balloon Counterpulsion: (See guidelines on IABP. Heparin use not routine)
- Continuous Renal replacement therapy (See below)
DVT prophylaxis should be commenced within 24-36 hrs of admission to the ICU. Low molecular weight heparin is generally considered as safe, and in some instances marginally superior (eg. orthopaedic patient populations) to unfractionated heparin. Enoxaparin (Clexane) is the chosen LMWH in the Waikato Hospital ICU (40mg daily).

**DVT prophylaxis using enoxaparin 40mg subcut once daily should be universal in ICU patients with exceptions listed below:**

Non-pharmacological methods of DVT prophylaxis: elasticated compression stockings (ECS) or sequential compression devices (SCD) may confer some protection against DVT formation.

**Exclusions to heparin DVT prophylaxis:**

- Active Bleeding, coagulopathy or thrombocytopenia
- Therapeutic anticoagulation (eg Warfarin, heparin)
- Significant intra-cerebral haemorrhage
- Heparin Induced Thrombocytopenia

**DVT prophylaxis by category**

- Medical ICU patients: Enoxaparin when bleeding risk felt to be minimal. When bleeding risk high (e.g. first three days after intracerebral haemorrhage), use ECS and SCD
- Non-neurosurgical, non cardiac Surgical patients: ECS plus enoxaparin when possible. Use SCD if enoxaparin contraindicated
- Head injury with CT evidence of frank haemorrhage or haemorrhagic stroke: ECS and SCD for 72 hrs. Substitute enoxaparin for SCD at 72 hours if appropriate. Check if EVD in place; consider not using enoxaparin if EVD placement seems likely
- Neurosurgical patients - tumours, SAH-ECS alone; meningiomas- ECS, enoxaparin delayed at least 7 days; aneurysm surgery –ECS; enoxaparin delayed at least 10-15 days or longer if intracerebral haematoma; chronic SDH-ECS, anticoagulants restarted 15-21 days, possibly after repeat CT
- Spinal Cord injury with intra-spinal haemorrhage on MRI: As for intra-cerebral haemorrhage above
- Pelvic fractures and patients with significant trauma: Thrombotic and initial bleeding risk high. If enoxaparin felt inappropriate at 24-36hrs then consider placement of temporary caval filter

**REFERENCE:**

THERAPEUTIC ANTICOAGULANT THERAPY

Which heparin?

- Enoxaparin is currently our first line heparin in most DVT, PE and in acute coronary syndromes
- IV unfractionated heparin may be used in patients undergoing procedures or at risk of bleeding

Dalteparin was being used in cancer associated VTE but only enoxaparin is funded in NZ.

ENOXAPARIN

- The usual dose of enoxaparin is 1mg/kg subcutaneously twice daily:
  - This dose needs to be changed in extremes of body weight and with renal impairment
- Dosing for body weight:
  - The treatment dose of enoxaparin or LMWH is based on actual weight

Studies have included obese patients up to 150kg with standard doses. There may be a reduced response in obesity and 1mg/kg BD may be preferable to 1.5mg/kg once daily. In morbidly obese patients (>150kg) there is limited data for dosing. We suggest the dose is capped at this level and monitor using anti-Xa (see below).

Enoxaparin may be more effective / accumulate in the underweight (BMI <20). We suggest using usual doses but consider anti-Xa monitoring.

DOsing in renal failure:

The dose should be reduced in renal failure. Recent data suggests that dose reduction should begin if the creatinine clearance is < 60 mls/min and that twice daily dosing with a reduced dose is safer. See the table for the recommended doses.

A loading dose of 1mg/kg should still be given initially to help get therapeutic levels.

In severe or worsening renal failure IV unfractionated heparin may be preferable. Note that the eGFR is not a good measure of renal function in acute kidney injury. Seek advice.

If enoxaparin is used in renal failure monitor using the anti-Xa assay (see below).

Note that it was PREVIOUSLY RECOMMENDED that if the creatinine clearance was < 30mls/min the enoxaparin dose should be reduced to 1 mg/kg given once daily and not BD.
CLEXANE DOSE IN RENAL FAILURE

<table>
<thead>
<tr>
<th>CrCl</th>
<th>Enoxaparin dose (12 hourly)</th>
<th>Enoxaparin dose (24 hourly)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;60ml/min</td>
<td>1mg/kg</td>
<td>1.5mg/kg</td>
</tr>
<tr>
<td>30-59ml/min</td>
<td>0.8mg/kg</td>
<td>N/A</td>
</tr>
<tr>
<td>&lt;30ml/min</td>
<td>0.66mg/kg</td>
<td>N/A</td>
</tr>
</tbody>
</table>

PRIOR TO COMMENCING TREATMENT:

- CBC, U&E, LFTs, urinalysis
- Short coagulation screen (indicate it is a pre-treatment sample and part will be saved)
- Identify any bleeding risk (see below)
- Consider stopping aspirin, although it is usually continued in acute coronary syndromes

COMMENCE SUBCUTANEOUS ENOXAPARIN:

- Chart subcutaneous dose at 1mg/kg twice daily
- 1.5mg/kg once daily may be just as effective but should be avoided in extremes of body weight and in renal impairment

Syringes come in 20mg, 40mg, 60mg, 80mg, 100mg, 120mg and 150mg strengths so consider rounding dose to 10 units nearest to patient’s weight for easier administration

USE WITH WARFARIN:

- In VTE start warfarin at the same time. Use the age-adjusted warfarin loading regime
- Enoxaparin is continued until the INR has been within the therapeutic range on 2 consecutive days AND until a minimum of 5 days treatment has been completed
- For patients who are simultaneously on warfarin, a daily INR is necessary initially

MONITORING OF ENOXAPARIN WITH ANTI-XA ASSAY:

The APTT is not useful; monitor by anti-Xa assay if needed.
• Test if:
  - Prolonged course (>14 days) of enoxaparin
  - Renal failure
  - If appropriateness of dose is in question (e.g., obesity or low body weight)
  - Suspected bleeding

• Best guide is peak levels (4 hours post-dose)

• Target is 0.6-1.0 IU/ml for bd dosing and 1-2 IU/mL for daily dosing

• Test after minimum 48 hours of therapy (5 half-lives)

• Re-testing is not necessary if therapeutic levels

In the majority of patients with AF, enoxaparin cover is not required when initiating warfarin (exceptions may be: AF less than 48 hours, multiple TIA’s or cardioversion patients).

**HEPARIN INFUSION**

Give a loading dose of 5,000 units IV stat bolus; this may be omitted in certain circumstances, for example after stroke or if previously on SC heparin with demonstrated prolongation of APTT:

• Use a 50ml syringe driver add 10,000 units to 0.9% saline to make up 50 mL; 1 mL = 200 units

• On wards a standard infusion pump is often used - add 10,000 units to 0.9% saline to make 100 mL; 1 mL = 100 units

• The initial rate of the heparin infusion is determined by the risk of haemorrhage

• Patients at an increased risk of haemorrhage include:
  - The elderly
  - Major surgery within 14 days
  - Known peptic ulcer disease
  - Visceral (especially cerebral) tumours
  - Severe hypertension
  - Thrombotic stroke within 14 days
  - Thrombocytopenia
  - Bleeding disorders
HIGH RISK = ONE OR MORE RISK FACTORS ARE PRESENT:

Start infusion at 5 mL/hr with syringe driver or 1000 units/hr = 10 mL/hr with infusion pump.

AVERAGE RISK IF ALL THESE RISK FACTORS ARE ABSENT:

Start infusion at 7 mL/hr with syringe driver or 1400 units/hour = 14 mL/hr with pump:

- APTT measurements must be taken:
- 6 hours after starting heparin infusion
- 6 hours after changing a dose
- 6 hourly if control is poor
- Otherwise once daily though this is unusual in ICU patients
- APTT measurements may be misleading in patients with lupus anticoagulant, factor deficiencies
- Therapeutic range for most indications is 60-100 seconds
- The target APTT should be 50 to 70 seconds after thrombolysis to reduce the risk of bleeding
- Alter the infusion rate according to APTT:

<table>
<thead>
<tr>
<th>APTT (secs)</th>
<th>CHANGE IN HEPARIN INFUSION RATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Target 50-70s)</td>
<td></td>
</tr>
<tr>
<td>&gt; 50 seconds above</td>
<td>Reduce by 500 units/hour</td>
</tr>
<tr>
<td>21-50 seconds above</td>
<td>Reduce by 300 units/hour</td>
</tr>
<tr>
<td>Up to 20 seconds above</td>
<td>Reduce by 100 units/hour</td>
</tr>
<tr>
<td>Therapeutic range</td>
<td>No change - therapeutic</td>
</tr>
<tr>
<td>Up to 15 seconds below</td>
<td>Increase by 100 units/hour</td>
</tr>
<tr>
<td>16-24 seconds below</td>
<td>Increase by 200 units/hour</td>
</tr>
<tr>
<td>&gt;24 seconds below</td>
<td>Increase by 400 units/hour</td>
</tr>
</tbody>
</table>

- Heparin therapy may cause thrombocytopenia in a small number of patients
- The risk may be less on low molecular weight heparin
- Platelet count should be monitored every second day if on unfractionated heparin
An age-adjusted warfarin loading protocol is available for the initiation of warfarin in hospital which reduces the risk of overtreatment and achieves a target INR more safely than fixed doses:

- The age-adjusted loading protocol is used when starting warfarin with heparin or warfarin alone
- The dosing is based on an Australasian trial published in the Aust NZ J Med 1999;29:731
- Alternative regimes may be used in the community in atrial fibrillation where there is less urgency in achieving a target INR. See the New Zealand Guidelines Group AF Guidelines

The dose of warfarin should be reduced for significant co-morbidities:

- Decrease the dose by one third (33%) if the patient has one or more of the following:
  - Severe congestive cardiac failure (EF <30% and/or biventricular failure)
  - Severe COPD (oxygen or steroid dependent, or dyspnoea at rest)
  - Concurrent amiodarone use
- Also consider liver disease and other drugs that interfere with warfarin pharmacokinetics

Prior to commencing warfarin check INR and CBC, U&E, LFTs, urinalysis. Consider stopping aspirin or other antiplatelet agents. Patients undergoing cardiac surgery may be sensitive to warfarin – confirm dose with surgeons.

### AGE-ADJUSTED WARFARIN DOSE PREDICTION CHART

<table>
<thead>
<tr>
<th>Day</th>
<th>INR</th>
<th>Up to 50yrs</th>
<th>51-65 yrs</th>
<th>66-80 yrs</th>
<th>&gt;80 yrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;1.4</td>
<td>10</td>
<td>9</td>
<td>7.5</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>&lt;1.6</td>
<td>10</td>
<td>9</td>
<td>7.5</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>&gt;1.6</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>3</td>
<td>&lt;1.8</td>
<td>10</td>
<td>9</td>
<td>7.5</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>1.8 - 2.5</td>
<td>4.0 - 5.0</td>
<td>3.5 - 4.5</td>
<td>3.0 - 4.0</td>
<td>2.5 - 3.0</td>
</tr>
<tr>
<td></td>
<td>2.6 - 3.0</td>
<td>2.5 - 3.5</td>
<td>2.5 - 3.5</td>
<td>2.0 - 2.5</td>
<td>1.5 - 2.0</td>
</tr>
<tr>
<td>INR</td>
<td>1.0 - 2.0</td>
<td>1.0 - 2.0</td>
<td>0.5 - 1.5</td>
<td>0.5 - 1.5</td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
<td></td>
</tr>
<tr>
<td>3.1 - 3.5</td>
<td>1.0 - 2.0</td>
<td>1.0 - 2.0</td>
<td>0.5 - 1.5</td>
<td>0.5 - 1.5</td>
<td></td>
</tr>
<tr>
<td>3.6 - 4.0</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>&gt;4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>&lt;1.6</td>
<td>10.0 - 15.0</td>
<td>9.0 - 13.0</td>
<td>7.5 - 11.0</td>
<td>6.0 - 9.0</td>
<td></td>
</tr>
<tr>
<td>1.6 - 1.9</td>
<td>6.0 - 8.0</td>
<td>5.5 - 7.0</td>
<td>4.5 - 6.0</td>
<td>3.5 - 5.0</td>
<td></td>
</tr>
<tr>
<td>2.0 - 2.6</td>
<td>4.5 - 5.5</td>
<td>4.0 - 5.0</td>
<td>3.5 - 4.5</td>
<td>2.5 - 3.5</td>
<td></td>
</tr>
<tr>
<td>2.7 - 3.5</td>
<td>3.5 - 4.0</td>
<td>3.0 - 3.5</td>
<td>2.5 - 3.0</td>
<td>2.0 - 2.5</td>
<td></td>
</tr>
<tr>
<td>3.6 - 4.0</td>
<td>3.0</td>
<td>2.5</td>
<td>2</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>4.1 - 4.5</td>
<td>Omit next day's dose, then</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;4.5</td>
<td>1.0 - 2.0</td>
<td>0.5 - 1.5</td>
<td>0.5 - 1.5</td>
<td>0.5 - 1.0</td>
<td></td>
</tr>
</tbody>
</table>

Nil. Hold dose

**REFERENCE:**


**TARGET INR**

**CURRENT RECOMMENDATIONS FOR TARGET INR VALUES ARE:**

INR 2.0-3.0 In AF and treatment of first DVT and PE or recurrent VTE in patients not on warfarin.

INR 2.5-3.5 For most mechanical valves but recommendations may vary.

INR 3-3.5 For recurrent DVT and PE in patients already receiving warfarin.
Deviations require dosage adjustment; see the table for dose adjustment.

It is recommended that warfarin doses be taken in the late afternoon so that morning INR testing can be completed and results indicating a dosage change may be actioned during the day.

### WARFARIN DOSE ADJUSTMENT

The following table gives guidelines for warfarin dose adjustment for patients on long-term therapy with a target INR of 2.5:

<table>
<thead>
<tr>
<th>INR</th>
<th>Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2</td>
<td>Increase weekly dose by 5 to 20 percent</td>
</tr>
<tr>
<td>3-3.5</td>
<td>Decrease weekly dose by 5 to 15 percent</td>
</tr>
<tr>
<td>3.6-4</td>
<td>Withhold no dose to one dose AND Decrease weekly dose by 10 to 15 percent</td>
</tr>
<tr>
<td>&gt;4</td>
<td>Withhold no dose to one dose AND Decrease weekly dose by 10 to 20 percent</td>
</tr>
</tbody>
</table>

### BLEEDING ON ANTICOAGULANTS

- Remember anticoagulation is not usually the only cause of bleeding
- The cause will need to be looked for and treated
- Anticoagulation alone is never the cause for haematuria
- Spontaneous bleeding is unusual if the INR / APTT are not excessive

### IV HEPARIN REVERSAL

- Common bleeding sites are gastrointestinal, surgical wounds or retroperitoneal
- If the APTT is prolonged excessively but no active bleeding or only minor bleeding, stop heparin
- Consider administering protamine sulphate IV 25 to 50mg at a rate not exceeding 5mg/minute
- Blood transfusion may be required
ENOXAPARIN REVERSAL

- Enoxaparin has a longer half-life than unfractionated heparin
- If there is only minor bleeding stopping the enoxaparin is all that may be needed
- If there is major bleeding, use protamine sulphate at a dose of 1mg per 1mg of enoxaparin
- A second infusion of 0.5mg protamine per 1mg enoxaparin may be given if bleeding continues
- Protamine reversal may only be around 60% as it never completely neutralises anti-Xa activity
- The time elapsed since the enoxaparin dose is important in the calculation of the protamine dose

Based on pharmacokinetic data, the following table should serve as a guide

<table>
<thead>
<tr>
<th>Time Elapsed Since Last Enoxaparin Dose</th>
<th>Protamine Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 8 hours</td>
<td>1mg protamine per 1mg enoxaparin</td>
</tr>
<tr>
<td>&gt;8 and &lt;12 hours</td>
<td>0.5mg protamine per 1mg enoxaparin</td>
</tr>
<tr>
<td>&gt;12 hours</td>
<td>May not be required (due to half-life)</td>
</tr>
</tbody>
</table>

Particular care should be taken to avoid over dosage with protamine.

Protamine should not be administered at a rate exceeding 5mg per minute.

WARFARIN REVERSAL

- The approach to management depends on a number of factors:
  - Severity of bleeding
  - Level of INR
  - Risk of haemorrhage vs risk of clotting
  - Indications for anticoagulation (AF vs mechanical valve replacement)
  - The bleeding risk increases as INR increases but 50% bleeding episodes occur while INR is < 4
  - The risk of bleeding rises sharply with INR values > 5
  - The risk of bleeding is greatest in the first 3 months after initiation therapy

- For most patients 1 to 2 mg IV Vitamin K is sufficient
• IV therapy takes 4 - 8 hours for effect but carries a small risk of anaphylaxis

• For complete urgent reversal a combination of FFP and Prothrombinex is effective

• Vitamin K should also be given to sustain reversal

• If anticoagulation is to be restarted avoid large doses of Vitamin K because high doses can cause warfarin resistance and prolong the time taken to achieve therapeutic INRs on restarting warfarin

• Prothrombinex may work more rapidly than FFP and achieve more complete normalisation

• FFP takes longer to administer

• The combination of Prothrombinex and FFP is recommended as there is a relatively low amount of factor VII in Prothrombinex but this may not be clinically important

• Remember that giving FFP means a fluid load

• Once bleeding is controlled, decide whether and when to restart anticoagulation

• If the reason for anticoagulation is AF, anticoagulants might be discontinued

See the following table from the Australasian Warfarin Reversal Consensus Group, Australasian Society of Thrombosis and Haemostasis, Position Statement MJA 2004;181:492
<table>
<thead>
<tr>
<th>Clinical Setting</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>INR higher than the therapeutic range but &lt; 5.0; bleeding absent</td>
<td>Lower the dose or omit the next dose of warfarin. Resume therapy at a lower dose when the INR approaches the therapeutic range. If the INR is only minimally above therapeutic range (up to 10%), dose reduction may not be necessary.</td>
</tr>
<tr>
<td>INR 5.0–9.0; bleeding absent</td>
<td>Cease warfarin therapy; consider reasons for elevated INR and patient-specific factors. If bleeding risk is high, give vitamin K1 (1–2 mg orally or 0.5–1 mg intravenously). Measure INR within 24 hours; resume warfarin at a reduced dose once INR is in therapeutic range.</td>
</tr>
<tr>
<td>INR &gt; 9.0; bleeding absent</td>
<td>Where there is a low risk of bleeding, cease warfarin therapy, give 2.5–5 mg vitamin K1 orally or 1 mg intravenously. Measure INR in 6–12 hours, resume warfarin therapy at a reduced dose once INR &lt; 5. Where there is high risk of bleeding, cease warfarin therapy, give 1 mg vitamin K1 intravenously. Consider Prothrombinex-VF (25–50 IU/kg) and fresh frozen plasma (150–300 mL), measure INR in 6–12 hours, resume warfarin therapy at a reduced dose once INR &lt; 5.</td>
</tr>
<tr>
<td>Any clinically significant bleeding where warfarin-induced coagulopathy is considered a contributing factor</td>
<td>Cease warfarin therapy, give 5–10 mg vitamin K1 intravenously, as well as Prothrombinex-VF (25–50 IU/kg) and fresh frozen plasma (150–300 mL), assess patient continuously until INR &lt; 5, and bleeding stops. OR If fresh frozen plasma is unavailable, cease warfarin therapy, give 5–10 mg vitamin K1 intravenously, and Prothrombinex-VF (25–50 IU/kg), assess patient continuously until INR &lt; 5, and bleeding stops. OR If Prothrombinex-VF is unavailable, cease warfarin therapy, give 5–10 mg vitamin K1 intravenously, and 10–15 mL/kg of fresh frozen plasma, assess patient continuously until INR &lt; 5, and bleeding stops.</td>
</tr>
</tbody>
</table>
Dabigatran (Pradaxa) is a direct thrombin inhibitor with a half-life of 12-14 hours. Dabigatran is primarily renally excreted and the half-life is prolonged in renal impairment.

The major adverse effect of all anticoagulant medications is bleeding. Two issues should be considered in managing bleeding events with dabigatran:

- Control bleeding and provide general support for haemodynamic state; and
- Attempt to reverse the anticoagulant effect where life-threatening bleeding is present

Initiate standard resuscitation measures.

Check coagulation screen including activated partial thromboplastin time (APTT), thrombin time (TT) and fibrinogen assay. Indicate time of last dabigatran dose when requesting test.

Check full blood count, renal function and electrolytes (including calcium)

There is no specific reversal agent for dabigatran currently and its anticoagulant effect will not be reversed by administration of vitamin K or FFP infusion.

**STOP Dabigatran Therapy**

**Mild bleeding**

- Local haemostatic measures
- Mechanical compression
- Tranexamic acid orally/topically, 15mg/kg four times a day
- Delay next dose of dabigatran or discontinue treatment as appropriate

**Moderate to Severe Bleeding** (reduction in Hb ≥ 20g/L, transfusion of ≥ 2 units of red cells or symptomatic bleeding in critical area or organ (for example, intraocular, intracranial, intraspinal, intramuscular with compartment syndrome, retroperitoneal, intra-articular or pericardial bleeding).

- Consult Haematology service
- Local measures / Mechanical compression
- Consider surgical intervention or wound packing
- Fluid replacement
- Maintain good urine output as dabigatran excreted renally
- Blood product transfusion
- Consider platelets if levels less than 70-80 X 10^9 /L or patient on anti-platelet agent
- Administration of anti-fibrinolytic agent
- Tranexamic acid IV (15-30mg/kg) +/- continuous infusion (1mg/kg/hr)
- Oral charcoal application if dabigatran ingestion <2 hours ago
- Consider Prothrombinex-VF 25-50 iu/kg. Repeat if necessary with Haematology guidance (note based on preclinical data only)

Life Threatening Bleeding – symptomatic intracranial bleed, reduction in Hb ≥ 50g/L, transfusion of ≥ 4 units of red cells, hypotension requiring inotropic agents or bleeding requiring surgical intervention.

- Implement measures for Moderate to Severe bleeding and consider:
  - Recombinant factor VIIa (Novoseven) (100mcg/kg by iv bolus) Repeat if necessary with Haematology guidance
  - Haemodialysis especially if renal failure present
  - Charcoal haemofiltration

REFERENCES:


D Garcia, M Crowther, W Ageno, Practical management of coagulopathy associated with warfarin BMJ 2010;340:c1813

Guidelines for management of bleeding with dabigatran.

MONITORING OF DABIGATRAN

Routine testing is not required during treatment with dabigatran. However, testing may be required in:

- Patients with moderate or severe reduction of renal function
- The perioperative setting
- In the event of bleeding

Tests that can measure the anticoagulant effect of dabigatran exist but are not yet well understood. Note that the INR is relatively insensitive to dabigatran with only supra-therapeutic concentrations of dabigatran resulting in an INR of approximately 2.0

The recommended tests for assessing the effect of dabigatran are:

- Activated partial thromboplastin time (APTT)
Moderately sensitive but has reduced responsiveness at higher doses. Result approximately twice baseline value at dabigatran treatment doses of 150 mg bid but varies for different test brands. Result of >80 seconds at trough (when the next dose is due) is associated with a higher bleeding risk.

- Thrombin time (TT):
  - Very sensitive with linear dose-response relationship
  - Significantly raised at therapeutic doses

Always indicate time of last dabigatran dose when requesting tests.

**INTERPRETATION OF DABIGATRAN COAGULATION RESULTS**

- APTT and TT normal:
  - No drug effect present Safe to proceed with surgery

- APTT normal or slightly prolonged and TT abnormal:
  - Drug effect present but likely low level

- APTT prolonged and TT abnormal:
  - Drug effect present and/or other haemostatic defect

Other tests which can be done to guide the treatment of a patient on dabigatran include:

- **Fibrinogen assay:**
  - May be useful to monitor for (DIC) and determining whether replacement treatment is required
  - Note that reagents vary in responsiveness to dabigatran for fibrinogen assays and some brands may give misleading results
  - If fibrinogen concentration is below ~1.5 g/L (note this is dependent on assay reagents), a dose of 1 bag of Cryoprecipitate per 30 kg body weight will increase fibrinogen by approximately 1 g/L.

- **Platelet count:**
  - Useful to determine whether replacement is required
  - Transfusion of Platelet Concentrate is indicated where the platelet count is below 70-80 X 10^9 /L
  - If the patient has been treated with an anti-platelet agent, a dose of 1 to 2 bags of Platelet Concentrate is appropriate for adults

- **Ecarin clotting time (ECT) (if available):**
  - Sensitive with a linear dose-response relationship
- Result increased 2-4 times at dabigatran doses of 150 mg bid

- **Haemoclot® thrombin inhibitor assay (if available):**
  - Sensitive with a linear dose-response relationship
  - Clotting time from 30 to 75 seconds at dabigatran dose of 220 mg/day

### PERIOPERATIVE MANAGEMENT OF DABIGATRAN

Semi-acute or elective surgery. Assess the risk of bleeding against the risk of thrombosis when considering discontinuing anticoagulation. For minor procedures, dabigatran may not need to be discontinued.

If dabigatran does need to be stopped, it is important to plan ahead as there is no treatment available to immediately reverse dabigatran.

Dabigatran is primarily renally excreted; therefore, the timing of discontinuation is dependent on the patient’s renal function. Renal function should be checked at the pre-admission clinic and the patient should be given clear instructions about when to stop dabigatran treatment.

<table>
<thead>
<tr>
<th>Renal Function (CrCl mL/min)</th>
<th>Half-life of Dabigatran (hours)</th>
<th>Timing of Discontinuation After Last Dose of Dabigatran Before Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Standard Risk of Bleeding</td>
</tr>
<tr>
<td>&gt; 80</td>
<td>13 (11-22)</td>
<td>24 hours</td>
</tr>
<tr>
<td>&gt; 50 to ≤ 80</td>
<td>15 (12-34)</td>
<td>24 hours</td>
</tr>
<tr>
<td>&gt; 30 to ≤ 50</td>
<td>18 (13-23)</td>
<td>At least 2 days (48 hours)</td>
</tr>
<tr>
<td>≤ 30</td>
<td>27 (22-35)</td>
<td>2-5 days</td>
</tr>
</tbody>
</table>

If there is a risk of thrombosis, consider bridging anticoagulant therapy:

- **Urgent surgery:**
  - Stop dabigatran
Check full blood count, electrolytes (including calcium), renal function and coagulation screen including APTT, TT and fibrinogen assay. Indicate time of last dabigatran dose when requesting test

Consider delaying surgery if appropriate until coagulation screen (APTT, TT and fibrinogen assay) is normal or until sufficient time has elapsed for drug clearance

Where urgent life-saving surgery cannot be delayed, consult with Haematology Service over measures (for e.g. recombinant factor VIIa) to control bleeding prior to and during the surgery

- Re-starting dabigatran after surgery:
  - The appropriate time to re-start dabigatran after surgery will be determined by the nature of the surgery, the urgency for restarting thromboprophylaxis and the haemostatic state of the patient. Discussion with a Haematologist is appropriate to determine individual case management

In elective situations where the wound is stable and haemostasis is satisfactory, it is suggested that dabigatran is re-started with a single capsule (75 mg, 110 mg or 150 mg depending on the indication) 1–4 hours after surgery with the usual daily dose commenced the following day.

A delay in restarting dabigatran will be appropriate if the wound is not stable and clinically significant wound losses are still present. Short term use of an alternative reversible anticoagulant (bridging anticoagulation) may be appropriate where thromboprophylaxis is required but the risks from wound bleeding are increased. The risk for thrombosis should be assessed.

## HEPARIN INDUCED THROMBOCYTOPAenia

HITS occurs in two forms:

- **Dose related:** Platelet clumping as an effect of the larger glycosaminoglycans containing the active pentasaccharide of heparin. Immediately obvious, dose related and usually mild

- **Auto-immune:** IgG antibody mediated. Therefore usually occurs 7-10 days after exposure in non-sensitised patients. Idiosyncratic, often severe. Can occur earlier if recent exposure

HITS may appear more commonly in the setting of continuous renal replacement therapy. This might reflect patient condition and platelet adsorption to dialysis filter.

### DIAGNOSIS

- **50% Decrease in platelet count:** Usually < 50 000×10^9 /L. Rarely < 20 000×10^9 /L

- **Skin lesions at heparin injection sites**

- **Dominant finding of thrombosis** (not bleeding)

- **Formation of Heparin antibodies** (heparin – PF4 ELISA is Sensitive but not specific)
TREATMENT MEASURES

- Stop all Heparin immediately and reconsider indication for anti-coagulation. Warfarin, if commenced, should not be used alone as it exacerbates thrombotic risk
- Use of Low Molecular Weight Heparin in these patients is not considered safe (cross-reactivity rates in excess of 90% reported)
- Institute alternative anticoagulant (eg: Thrombin inhibitors or Danaparoid Sodium). Bivalirudin considered first line for HITS

BIVALIRUDIN – DIRECT THROMBIN INHIBITOR

- Dose 0.15mg/kg/hr to achieve APTT 1.5-2 times baseline. Adjust in severe renal or hepatic failure (0.05-1mg/kg/hr). Coags return to normal approx one hour after stopping with normal renal function
- Danaparoid is no longer available

REFERENCE:

ENDOCRINE DRUGS

INSULIN

Glycaemic control in the critically ill has become one of the most debated aspects of care. This area of ICU practice is evolving and requires regular review. Considering the results of the NICE-SUGAR study we have relaxed our target glycaemic control.

**Target blood sugar level = 8.0 11.0 mmol/L in the Waikato Hospital ICU**

**INDICATIONS FOR INSULIN IN THE ICU**

- Diabetic emergencies: NB Rapid glycaemic control is not a priority in patients with either hyperosmolar or ketotic diabetic states. In fact rapid correction of severe hyperglycaemic states may aggravate cerebral oedema

- Hyperglycaemia in diabetics and non-diabetics, particularly with AMI or neurological conditions

- Treatment of hyperkalaemia: ie 50 ml 50% dextrose administered with 10 units actrapid insulin

**ADMINISTRATION OF INSULIN**

- Mix regular short acting insulin (Actrapid) with normal saline to a concentration 1 IU/ml. 30IU in 30mls N/Saline 0-10ml/hr

- Administer in a 50 ml syringe via syringe driver

- Discard at 24 hrs of use

**MONITORING OF BLOOD GLUCOSE**

Blood sugar levels should be monitored hourly until stable within desirable range. Once stable, monitor at least 2 hrly in the first 48 hrs of ICU admission, and 4 hrly thereafter.
**SCALE ONE: STARTING OR RECOMMENCING ACTRAPID INSULIN INFUSION**

<table>
<thead>
<tr>
<th>Measured blood sugar</th>
<th>Actrapid insulin infusion</th>
</tr>
</thead>
</table>
| **Less than 3.9 mmol/L** | • Check glucose on blood gas machine and use this value  
• Give 20mL of 50% glucose.  
• Ensure adequate glucose intake  
• Repeat blood sugar 15 mins after glucose load |
| **4 – 5.9 mmol/L** | • Ensure adequate glucose intake  
• Repeat blood sugar in 30mins if BGL falling rapidly  
• Otherwise repeat BGL 1 – 2 hourly if stable |
| **6 – 11 mmol/L** | Repeat blood sugar in 1 hour |
| **11.1 – 12 mmol/L** | • Start Actrapid infusion at 1 unit/hr  
• Repeat blood sugar in 1 hour |
| **12.1 – 15 mmol/L** | • Start actrapid infusion at 2 units/hr  
• Repeat blood sugar in 1 hour |
| **Greater than 15.1 mmol/L** | • Start actrapid infusion at 3 units/hr  
• Repeat blood sugar in 1 hour |
## SCALE TWO: ONGOING ACTRAPID INSULIN INFUSION

<table>
<thead>
<tr>
<th>Measured blood sugar</th>
<th>Actrapid insulin infusion</th>
</tr>
</thead>
</table>
| **3.9 mmol/L or less** | • Stop actrapid infusion and check blood glucose on blood gas machine  
• Give 20mL of 50% glucose  
• Ensure adequate glucose intake  
• Repeat blood sugar in 15 minutes after glucose load |
| **4 – 5.9 mmol/L** | • Stop actrapid infusion and ensure adequate glucose intake  
• Repeat blood glucose in 30 minutes if falling rapidly, otherwise repeat in 1 hour  
• Move back to ‘following Scale One’ |
| **6 – 7.9 mmol/L** | • Decrease actrapid infusion rate by half  
• Ensure adequate glucose intake  
• Repeat blood sugar in 1 hour |
| **8 – 11 mmol/L** | • Leave actrapid infusion unchanged  
• Repeat blood sugar in 1 hour  
• Once stable in 2 readings, repeat 2 – 4 hourly |
| **11.1 – 12 mmol/L** | • Increase actrapid infusion rate by 1 unit/hr  
• Repeat blood sugar in 1 hour |
| **Greater than 12.1 mmol/L** | • Increase actrapid infusion rate by 2 units/hr  
• Repeat blood sugar in 1 hour |

### INSULIN-DEXTROSE IN EXCEPTIONAL CIRCUMSTANCES

The above guideline only applies to patients receiving IVI dextrose or significant enteral caloric intake. Should this be interrupted, insulin is to be stopped and a BSL checked after 1 hr.

Where the desired patient maintenance fluid does not contain dextrose (i.e Normal Saline), 10 ml/hr of 50% dextrose should be run concurrently, and insulin administration continued as above.
ONGOING REQUIREMENT FOR INSULIN BEYOND ACUTE PHASE:

Patients requiring insulin for established or known diabetes should be converted to subcutaneous insulin as a medium or long acting form with / without short acting insulin constructed according to subcutaneous sliding scale. As these patients may need long term follow-up, they should be referred to the endocrine service for assistance.

REFERENCE:

The NICE SUGAR study investigators.

Intensive vs. Conventional glucose control in critically ill patients. NEJM 2009; 360(3)

DDAVP

For diabetes insipidus

GENERAL

DI may occur in the following settings:

- Evolving brain death or severe brain injury
- Post ablative pituitary surgery, or injury to pituitary stalk (anterior cranial fractures)
- Nephrogenic causes are typically mild and do not require treatment

Fluid mobilisation during convalescent phase of injury should not be mistaken for DI

INDICATIONS FOR DDAVP IN DIABETES INSIPIDUS

- Persistent polyuria > 300 ml/hr for more than 3-4 hours with incremental hypernatraemia
- Low urine osmolality in the presence of high plasma osmolality (or hypernatraemia)
- Pre-existing hyperosmolar state or intravascularly deplete patient

DOSE OF DDAVP IN DIABETES INSIPIDUS

1-2 microg IVI bd as required.

FLUID ORDERS:

Isotonic fluid replacement in under-resuscitated patients.

5% Dextrose or 0.45% Saline in patients where hypernatraemia exists (maximum decrease in serum Sodium should not exceed 2 mmol/L/hour).
**DDAVP FOR PLATELET DYSFUNCTION**

**INDICATIONS**

Adjunctive treatment in bleeding patients with platelet dysfunction as a result of:

- Uraemia
- Cirrhosis
- Von-Willebrand’s Disease
- Drug (NSAID-s or aspirin) or cardiac surgery related platelet dysfunction

**CONTRAINDICATIONS**

Use in patients with severe coronary or cerebrovascular atherosclerosis may cause arterial thrombosis.

**DOSE**

0.3 microg / kg IVI over 30 minutes or 300 microg intra-nasally.

In some instances a second dose may be administered, although a rapid “fall-off” in effect per dose (tachyphylaxis) is the norm.

**STEROIDS**

**GENERAL**

The use of steroids in the critically ill has been the subject of much debate and some research.

**PROVEN INDICATIONS**

- Hypoadrenalism (Addison’s disease or crisis)
- Acute severe asthma
- Panhypopituitarism
- Haemophilus meningitis in children (discuss with paediatric team first)
- Adult pneumococcal meningitis – area of debate but often used for pneumococcal meningitis
- Collagen Vascular diseases
- Active Immunosuppression (GVHD, solid organ transplant)
- Myasthenia Gravis
- Treatment peritumoral oedema in the central nervous system
**UNPROVEN ICU INDICATIONS**

- Non-infected (fibroproliferative) ARDS: Meduri protocol = Methylprednisolone 2 mg/kg for 14 days, tapered 1.0-0.5 mg/kg for next 14 days
- Shock associated with vasodilated states which are refractory to high dose, or prolonged administration of inotropes
- Myocarditis
- Exacerbation of chronic airway obstruction
- Bronchiolitis obliterans
- Anaphylaxis

**CONDITIONS WHERE STEROIDS ARE NOT INDICATED OR ACTIVELY CONTRA-INDICATED**

- Active infection
- Head injury
- Guillain-Barre Syndrome
- Fat embolism syndromes

**RELATIVE STEROID POTENCIES**

<table>
<thead>
<tr>
<th>Steroid</th>
<th>Equivalence (mg)</th>
<th>Glucocorticoid Activity</th>
<th>Mineralocorticoid Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydrocortisone</td>
<td>100 mg</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Prednisone</td>
<td>25</td>
<td>4</td>
<td>0.3</td>
</tr>
<tr>
<td>Methylpred.</td>
<td>20</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>4</td>
<td></td>
<td>250</td>
</tr>
<tr>
<td>Fludrocortisone</td>
<td>1</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>
MICROBIOLOGY GUIDELINES

INTRODUCTION

Sepsis is a common cause of death in critically ill patients. The detection of active infection, as opposed to colonisation, is difficult but important. Regular routine microbiological examination is not cost effective in the ICU. Infective screens should only be ordered for specific indications, using the guidelines listed below.

Simple preventative measures are important in the containment of infection and the prevention of bacterial resistance:

- Strict aseptic technique for all invasive procedures
- Rational prescription of antibiotics
- Hand washing (5 moments of hand hygiene)

INFECTION CONTROL MANAGEMENT

Infection Control is the responsibility of every staff member in the CCD. There are a number of initiatives in place supporting infection control management such as ‘The 5 Moments of Hand Hygiene’ and the ‘CLAB project’.

The Ministry of Health sets standards around infection control for example the 5 Moments of Hand Hygiene in which the ‘moments’ are continuously audited by our Department Gold Auditors (Christine Carter – ICU ACNM, Emma Johnson – ICU RN and Gwyn Bassett – HDU RN), then these results are collated and reviewed quarterly. Hand Hygiene New Zealand (2012) has set a target of 70% compliance to the 5 Moments of Hand Hygiene for each DHB in New Zealand.

CONSIDERATIONS

To ensure your hand hygiene is effective it is important to:

- Remove bracelets, wrist watches and rings with stones or ridges when providing clinical care. Only flat rings may be worn during patient care activities. If worn, the surfaces under rings must be washed and dried frequently to remove bacteria
- Make sure your sleeves are above the elbow and do not interfere with effective hand hygiene practice
- Keep nails short and clean and do not wear nail polish. Artificial nails (gel or acrylic) are not permitted
- Cover any breached skin (cuts, dermatitis or abrasion) with a waterproof film dressing
- Avoid long ties and lanyards. If wearing a tie ensure it is tucked in or secured

If you experience any skin problems you must inform your manager and complete an Incident Form immediately.
FIVE MOMENTS OF HAND HYGIENE

• Moment 1: Before patient contact:
  o Why: To protect the patient against pathogens carried on your hands

• Moment 2: Before a procedure:
  o Why: To protect the patient against pathogens, including the patient’s own, from entering his/her body

• Moment 3: After a procedure or body fluid exposure risk:
  o Why: To protect yourself and the healthcare environment from patient pathogens

• Moment 4: After patient contact:
  o Why: To protect yourself and the healthcare environment from patient pathogens

• Moment 5: After contact with patient surroundings:
  o Why: To protect yourself and the healthcare environment from patient pathogens
REFERENCES


DEFINITIONS

INFECTION

The invasion of normally sterile tissues by micro-organisms.

COLONISATION

The detection of micro-organisms on or in a patient that are neither pathogenic nor elicit an inflammatory response.

NOSOCOMIAL INFECTION

Infection which was not present or incubating at the time of admission to hospital (generally developing >48 hours after admission).
**SEPSIS**

The presence of infection (documented or probable) together with systemic manifestations of infection, including some of the following:

<table>
<thead>
<tr>
<th>General Variables</th>
<th>Inflammatory Variables</th>
<th>Hemodynamic Variables</th>
<th>Organ Dysfunction Variables</th>
<th>Tissue Perfusion Variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature &gt;38°C or &lt;36°C</td>
<td>Leukocytosis (WBC &gt;12 x10⁹/L)</td>
<td>SBP &lt;90mmHg or SBP decrease &gt;40mmHg in adult</td>
<td>Hypoxemia (PaO2/FiO2 &lt;300)</td>
<td>Hyperlactatemia (&gt;1 mmol/L)</td>
</tr>
<tr>
<td>Heart Rate &gt;90 beats per minute or &gt;2 standard deviations above normal for age</td>
<td>Leukopenia (WBC &lt;4 x10⁹/L)</td>
<td>MAP &lt;70mmHg</td>
<td>Oliguria (UO &lt;0.5ml/kg/hr for at least 2 hours despite adequate fluid resuscitation)</td>
<td>Decreased capillary refill or mottling</td>
</tr>
<tr>
<td>Respiratory Rate &gt;20 breaths per minute</td>
<td>&gt;10% immature forms</td>
<td>Creatinine increase &gt;44 micromol/L</td>
<td>Coagulation abnormalities (INR &gt;1.5, aPTT &gt;60s)</td>
<td></td>
</tr>
<tr>
<td>Altered mental status</td>
<td>CRP &gt;2 standard deviations above normal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Significant oedema or positive fluid balance (&gt;20ml/kg over 24 hours)</td>
<td>PCT &gt;2 standard deviations above normal</td>
<td>Ileus (absent bowel sounds)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperglycemia (glucose &gt;7.7 mmol/L in absence of diabetes)</td>
<td></td>
<td></td>
<td>Thrombocytopenia (Plts &lt;100 x10⁹/L)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hyperbilirubinemia (Bilirubin &gt;70 micromol/L)</td>
<td></td>
</tr>
</tbody>
</table>
SEVERE SEPSIS

Sepsis induced tissue hypo-perfusion or organ dysfunction, with any of the following thought to be due to infection:

- Sepsis induced hypotension
- Hyperlactatemia
- Oliguria
- Acute lung injury with PaO2/FiO2 <250 in the absence of pneumonia or PaO2/FiO2 <200 in the presence of pneumonia
- Creatinine >176.9 micromol/L
- Bilirubin >34.2 micromol/L
- Platelets <100 x10⁹/L
- Coagulopathy (INR>1.5)

SEPTIC SHOCK

Sepsis induced hypotension persisting despite adequate fluid resuscitation (30ml/kg for crystalloids some of which may be albumin). It is characterized by markedly decreased systemic vascular resistance often with an elevated cardiac output.
## SEPTIC SCREEN IN ICU

### EMPIRIC SEPTIC SCREEN

<table>
<thead>
<tr>
<th>Specimen</th>
<th>Comment</th>
</tr>
</thead>
</table>
| **Blood cultures**               | Minimum of 2 sets of blood cultures should be taken prior to administration of antibiotics (provided it doesn’t delay the administration of antibiotics by >45 minutes)  
One culture should be taken from fresh peripheral stab and one from existing CVL or arterial line if >48 hours old and correctly labelled as such  
Cultures taken off a new CVL or arterial line at the time of insertion should be labelled **peripheral** |
| **Urine microscopy and culture** | Symptomatic bacteruria = $>10^3 \text{ cfu/ml}$ of pathogenic bacteria in the presence of signs or symptoms compatible with UTI in a patient with an indwelling urinary catheter or intermittent catheterization or removal of catheter in the last 48 hours  
Asymptomatic bacteruria = $>10^5 \text{ cfu/ml}$ of pathogenic bacteria in the absence of symptoms and signs compatible with infection (common and does not require treatment)  
Pyuria (elevated WCC $>10 \text{ cells/microL}$) is common in the ICU and may not be indicative of active infection  
Sequential courses of antibiotics will not eradicate infection while the catheter remains in situ |
| **Tracheal aspirate** (Generally not recommended) | Performed by advancing a catheter through the ETT until resistance is felt and then applying suction  
Qualitative culture of $>1 \text{ million cfu/ml}$ of a respiratory pathogen is sensitive (but not specific) for VAP in the setting of clinical signs and symptoms consistent with VAP  
ETT becomes quickly colonized resulting in false positives (30%) therefore only performed immediately after intubation |
**DIRECTED SEPTIC SCREEN**

**Community Acquired Pneumonia (Send on admission)**
- Blood cultures x2 sets.
- Urine for pneumococcal antigen
- Serum (red tube) for Mycoplasma IgG and IgM, Legionella IgG (requires second sample in 2 weeks), Legionella PCR
- Nasopharyngeal swab (blue swab) for respiratory virus PCR (Influenza A and B, RSV, hMPV)
- Consider tracheal aspirate from new ETT (as above) or BAL for community acquired pneumonia. Indicate if patient immunocompromised for special panel of tests (pneumocystis jirovecii, CMV, Aspergillus, coronavirus, rhinovirus and TB)
- Consider pleural fluid sample (TB, pneumococcal antigen, pH, protein, LDH, culture and gram stain)
- Consider early morning sputum for TB (or tracheal aspirate or BAL)
- Consider nasopharyngeal swab for pertussis
- Consider SARS and MERS if travel to endemic areas
- Consider chlamydia psittacii if bird exposure

**Broncho-alveolar lavage (BAL) for Ventilator Associated Pneumonia**
- Performed by infusion and aspiration of sterile saline through a flexible bronchoscope wedged into a bronchial segmental orifice.
- BAL may be undertaken to confirm suspicion of ventilator associated pneumonia (VAP) in patients with clinical signs and symptoms consistent with VAP and CXR changes (allows rationalisation of antibiotic therapy without reducing mortality, duration of ICU stay, duration of mechanical ventilation).
- Quantitative culture of >10⁴ CFU/ml of a respiratory pathogen is used as a threshold for diagnosis of VAP in clinically appropriate circumstances.
- Alternatively >5% intracellular organisms is diagnostic.
- Adequacy of sample is indicated by <1% epithelial cells.

**Lumbar Puncture**
- **Bacterial Meningitis:**
  - WBC >1000/microL
  - Predominance of neutrophils
- **Viral Meningitis:**
  - WCC <250/microL (usually)
  - Predominance of lymphocytes
Lumbar puncture cont.
- Elevated protein
- Low glucose (levels <1 mmol/L strongly predictive of bacterial meningitis)
- Positive gram stain

Low protein

Normal glucose (can be low with HSV, mumps, enterovirus, HZV)

Positive HSV PCR

Ascitic Tap

SBP:
- PMN count >250/mm³
- Total protein <10g/L
- Glucose >2.8 mmol/L
- Positive gram stain

Secondary Bacterial Peritonitis:
- PMN count >250/mm³
- Total protein >10g/L
- Glucose <2.8 mmol/L
- LDH >serum upper limit
- Elevated amylase (pancreatitis or gut perforation)
- Elevated bilirubin (biliary perforation)
- Polymicrobial gram stain

* TOE

* Sinus CT

* Biopsy of skin lesions

CATHETER RELATED BLOODSTREAM INFECTIONS

INTRODUCTION

Catheter Related Bloodstream Infections (CRBSI) = blood stream infection in the setting of a central venous line (CVL) with no other apparent source.

Rate = 0.5-4.8% per 1000 catheter days.

Mortality = 12-25% for each infection.

It is no longer common practice to remove or replace central access routinely, but only when infected or no longer required.

Implementation of CVL bundles and the used of antibiotic impregnated central venous lines has significantly reduced the rate of CRBSI.
CVL INSERTION BUNDLE

- Optimal catheter site selection (subclavian > internal jugular > femoral depending on indication and experience)
- Hand hygiene
- Chlorhexidine skin antisepsis (2% chlorhexidine gluconate in 70% isopropyl alcohol)
- Maximal barrier precautions using cap, mask, sterile gown, gloves, large sterile drape with small opening for site of insertion

Sterile guide-wire exchanges may only be performed where mechanical problems complicate new catheter site within a few hours of sterile insertion

CVL MAINTENANCE BUNDLE

- Daily review of line necessity with prompt removal of unnecessary lines
- Dedicated access for TPN
- Asepsis when accessing CVL lumens
- Daily review of entry site for inflammation

CLINICAL PRESENTATION OF CRBSI

CRBSI should be suspected in any patient with a CVL present for >48 hours who develops:

- Fever (High sensitivity, low specificity)
- Inflammation or purulence at the insertion site (High specificity, low sensitivity)
- Increasing WCC or inflammatory markers
- Hemodynamic instability
- Altered mental status
- Positive blood culture with likely organism and no other likely source of infection

DIAGNOSIS

Requires at least two sets of blood cultures (each set should include a peripheral sample taken from a fresh stab and one from the central line).

Catheter tips are not sent for culture.

Criteria used to identify catheter related blood stream infection:
1. Patient has a recognised pathogen cultured from both a peripheral and central sample (not related to an infection from another site)

2. There is clinical suspicion of CRBSI and positive culture of the same organism from at least two blood samples (peripheral and central venous access) which meet quantitative criteria or differential time to positivity:
   - Quantitative criteria = colony count from central line sample >3x higher than the colony count from the peripheral sample
   - Differential time to positivity = growth detected from the central line sample >2 hours before growth detected from the peripheral sample (sensitivity 85%, specificity 91%)

Clinical improvement within 24 hours following catheter removal is suggestive of CRBSI but not diagnostic.

A single positive central line culture in the setting of a negative concurrent peripheral culture suggests colonization. Further cultures should be taken. Monitor the patient for signs of infection. Consider removing the catheter.

**MANAGEMENT**

Remove line on suspicion of infection (line removal will usually result in resolution of clinical sepsis) or if no longer needed.

Antibiotics are indicated only if sepsis is severe, progressive following removal of the line, or if the patient is high risk (e.g. prosthetic implants).

Refer to antibiotic guidelines for selection of antibiotics.

**SPECIAL POINTS ON LONG TERM VASCULAR ACCESS**

Long stay, surgically implanted catheters may be precious, and should not be removed without consulting the Duty Intensivist.

Perform peripheral blood cultures, and remove the offending line if:

- Patient unstable
- Blood culture grows a fungus
- There is obvious catheter tunnel infection is present

Stable patients with a positive blood culture should receive 3 days of appropriate antibiotic therapy.

If they are judged to have responded adequately to therapy, a trial of line locking may be used to save the intravascular catheter.

A 70% ethanol solution is often used first line (twice daily and after each use of the line).

Antibiotic line locking may also be trialled (Gentamicin 5 mg/ml or Vancomycin 1mg/ml is used depending on the microbe grown). Infectious disease guidance should be sort.
Procedure:
- Dilute the appropriate antibiotic in normal saline
- Inject 2ml of locking antibiotic (most lines have a capacitance of < 1ml – check if you are unsure)
- Leave antibiotic lock in-situ for >12 hr per day

VENTILATOR ASSOCIATED PNEUMONIA

INTRODUCTION
Ventilator Associated Pneumonia develops after more than 48 hours mechanical ventilation.
Incidence = 10-25% of ICU patients.

CLINICAL PRESENTATION
New or progressive pulmonary infiltrate on CXR and one or more of the following:
- Fever
- Purulent tracheobronchial secretions
- Increased WCC
- Increased respiratory rate
- Decreased tidal volume
- Increased minute ventilation
- Decreased oxygenation

DIAGNOSIS
CXR showing new or progressive pulmonary infiltrate in a patient who is clinically suspected to have VAP.
Bronchoscope guided BAL (see above) should be conducted whenever possible prior to antibiotic therapy to confirm the diagnosis and identify the likely pathogen.

TREATMENT
Empiric treatment should be guided by the initial gram stain.
See ICU Antibiotic guidelines.
FUNGAL INFECTIONS

INTRODUCTION

The incidence of systemic fungal infections in Intensive Care has increased in recent years as a result of:

- Increased use of broad spectrum antibiotics
- Increasing numbers of immunosuppressed patients being referred to ICU (solid organ transplant, bone marrow transplant, HIV, malignancy)
- Use of invasive devices (surgical drains, intravascular catheters)
- Co-existent use of immunosuppressive therapy

ORGANISMS

Candida albicans is the most common cause of candidemia and invasive candidiasis (meningitis, endocarditis, intra-abdominal infections).

Non-albicans candida include:

- Candida glabrata
- Candida parapsilosis
- Candida tropicalis
- Candida krusei

Invasive mould infections (lung, sinus, CNS) are mainly due to Aspergillus.

DIAGNOSIS

Diagnosis by:

- Culture from blood or tissue (delays in culture can prolong time to appropriate anti-fungal treatment)
- Serum Aspergillus precipitans IgG (Aspergillus only)

INDICATIONS FOR ANTIFUNGAL PROPHYLAXIS

Routine antifungal prophylaxis in ICU patients is not recommended.

Prophylaxis should be used in special patient groups:

- Bone marrow transplant
- Neutropenic patients
Indications for antifungal therapy:

- Single positive blood culture in a high risk patient (diabetes mellitus, renal failure, severe pancreatitis, invasive devices, broad spectrum antibiotics, parental nutrition, prolonged mechanical ventilation/ICU stay, malignancy, solid organ transplant)

- Isolation of candida from any sterile body site (except urine) or isolation of fungi in two anatomically discrete sites in selected patients

- Histological evidence of yeast or mycelial forms in tissue from high risk patients

**TREATMENT**

See ICU Antibiotic Guidelines.

Consult ID if there is any doubt with regards to the initiation of anti-fungal therapy.
ICU ANTIBIOTIC GUIDELINES

PROLOGUE

Emerging bacterial resistance is one of the major challenges facing modern intensive care.

It is the duty of all members of staff to actively participate in the appropriate use of anti-microbials, while adopting proven infection control behaviour.

INTRODUCTION

The Waikato Hospital Intensive Care Unit supports in general the Waikato Hospital Antimicrobial Guide.

This section cannot be a comprehensive guide, but should aid staff with the Waikato Hospital ICU preferences in antibiotic prescribing practice.

SPECIAL POINTS

All drug doses and dosing intervals provided in this section are intended for the general population. When prescribing drugs for patients who are elderly, critically ill or who have significant renal or hepatic insufficiency, doses may require adjustment to allow for modified pharmacokinetics.

Antibiotics should not be charted or changed without prior discussion with the Duty Intensivist.

All antibiotic charting must be reviewed daily and the indication recorded on the drug chart.

SURGICAL PROPHYLAXIS

GENERAL

Prophylactic antibiotic therapy decreases the incidence of surgical site infections following certain surgical procedures.

Use should be restricted to surgical procedures in which:

- The risk of postoperative infection is high (ie. Clean-contaminated surgical procedures)
- The consequences of postoperative infection would be potentially disastrous (eg. prosthetic implants, spinal surgery)

Other risk factors for surgical site infection include operative technique, length of surgery and over-all patient health.

TIMING

Prophylactic antimicrobial therapy requires an adequate concentration of antibiotic in the tissues at the time of incision and throughout the operation until wound closure is complete.

Antibiotics for the purpose of surgical prophylaxis should be administered at the time of anaesthetic induction (administration should be completed within 30-60 minutes of surgical incision).
A second dose of antibiotic may be required in the following circumstances:

- Delay in starting surgery
- Prolonged surgery >4 hours duration (repeat dose of Cephazolin after 4 hours, Clindamycin after 6 hours)
- In specific circumstances (amputation of ischemic limb, significant blood loss >1500ml)

There is little evidence to support extending prophylactic antibiotic therapy beyond the operative period with few exceptions.

**PREFERRED ANTIBIOTIC**

The specific antimicrobial agent used for surgical prophylaxis should be directed against the most likely causative organisms taking into account local hospital susceptibility patterns. It is not rational to attempt to cover all possible microbes (some will not contribute to the development of postoperative infections).

Cephazolin is the drug of choice for most procedures due to its spectrum of activity, duration of action, safety and low cost (dose adjust in renal failure).

Vancomycin is warranted for patients known to be colonized with MRSA or with penicillin anaphylaxis.

Clindamycin can be used as an alternative for patients with penicillin anaphylaxis.

Additional gram negative cover (e.g., Metronidazole) is required for certain procedures (head and neck or general surgical procedures).

The antibiotic choices below constitute a rational approach to surgical prophylaxis, however it is not the role of the ICU staff to direct surgical choice of agent or duration of surgical prophylaxis.

Please confirm antibiotic choice and duration with each individual surgeon at the time of admission of the patient to the ICU.

<table>
<thead>
<tr>
<th>Surgical Specialty</th>
<th>Likely Pathogens</th>
<th>Antibiotic Dose and Duration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiothoracic Surgery</strong></td>
<td>Staphylococcus epidermidis</td>
<td>Cephazolin 2g IV Q8H for 48 hours post-incision</td>
<td>Antibiotic prophylaxis for 48 hours post cardiac surgery remains commonplace in the absence of evidence to establish the optimal approach. No evidence that presence of drains and lines should require extended duration antibiotic prophylaxis beyond</td>
</tr>
</tbody>
</table>
48 hours post incision.

<table>
<thead>
<tr>
<th>Section</th>
<th>Common Pathogens</th>
<th>Antibiotics</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Neurosurgery</strong></td>
<td>Staphylococcus epidermidis</td>
<td>Cephazolin 2g IV</td>
<td>Single pre-incision dose recommended for elective craniotomy, CSF shunt and intrathecal pump operations.</td>
</tr>
<tr>
<td></td>
<td>Staphylococcus aureus</td>
<td></td>
<td>No consensus on the role of prophylactic antibiotics in EVD and ICP placement although this is widely requested.</td>
</tr>
<tr>
<td></td>
<td>Propionibacterium acnes</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Orthopaedics</strong></td>
<td>Staphylococcus aureus</td>
<td>Cephazolin 2g IV</td>
<td>Antibiotic prophylaxis indicated for implantation of prosthetic material and spinal surgery.</td>
</tr>
<tr>
<td></td>
<td>Staphylococcus epidermidis</td>
<td></td>
<td>Little evidence to support use beyond a single pre-incision dose but this may be requested.</td>
</tr>
<tr>
<td></td>
<td>Beta-hemolytic strep</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gram negative bacilli</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Head and Neck Surgery</strong></td>
<td>Streptococci</td>
<td>Cephazolin 2g IV</td>
<td>Single pre-incision dose for clean-contaminated procedures with incision through the oropharyngeal mucosa</td>
</tr>
<tr>
<td></td>
<td>Staphylococci</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anaerobes</td>
<td>Metronidazole 500mg IV</td>
<td>Or Amoxicillin/Clavulanate 1.2g IV</td>
</tr>
<tr>
<td></td>
<td>Enterobacteriaceae</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>General Surgery</strong></td>
<td>Anaerobes</td>
<td>Metronidazole 500mg IV</td>
<td>Single pre-incision dose. If peritoneal contamination complete full treatment course for 5 days.</td>
</tr>
<tr>
<td></td>
<td>Aerobic gram negative bacilli</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Streptococci</td>
<td>Cephazolin 2g IV</td>
<td>And either Gentamicin 3-5mg/kg IV</td>
</tr>
<tr>
<td><strong>Vascular Arterial Surgery</strong></td>
<td>Staphylococcus aureus</td>
<td>Cephazolin 2g IV</td>
<td>Single pre-incision dose generally recommended.</td>
</tr>
<tr>
<td>involving prosthesis, abdominal</td>
<td>Staphylococcus epidermidis</td>
<td></td>
<td>No good evidence that prophylactic antibiotic use beyond 24 hours beneficial (one RRT showed reduced SSI)</td>
</tr>
<tr>
<td>aorta or groin</td>
<td>Enteric gram negative</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
incision bacilli Gentamicin 3-5mg/kg IV with antibiotic prophylaxis up to 5 days postop while venous access insitu however antibiotic used not recommended for prophylaxis).

Lower Limb Amputation

Staphylococcus aureus

Staphylococcus epidermidis

Enteric gram negative bacilli

Clostridia

Cephazolin 2g IV Q8h for 24 hours post-incision and

Metronidazole 500mg IV Q12H for 24 hours

Some evidence that antibiotic prophylaxis for 24 hours post incision decreases SSI.

Active infection and staged amputations require antibiotic treatment.

ANTIBIOTIC THERAPY

GENERAL

The institution of empiric antibiotic therapy prior to definitive bacteriological diagnosis should be based on localizing signs and symptoms, potential pathogens and local antimicrobial sensitivity patterns.

Whenever possible appropriate cultures (blood culture x2, urine, sputum, infected tissue) should be obtained prior to antibiotic therapy, provided it does not delay antibiotic administration by >45 minutes. The exception is meningitis where potentially life-saving antibiotic therapy should not be delayed while awaiting lumbar puncture.

Appropriate empiric antimicrobials should be continued for at least 48 hours before another empiric antibiotic is added or substituted.

Once gram stain or culture results become known, the choice of agent should be rationalised.
### SCENARIO’S REQUIRING EMPIRICAL USE OF ANTIBIOTICS IN ICU

<table>
<thead>
<tr>
<th>Infection</th>
<th>Likely Pathogens</th>
<th>Antibiotic Dose And Duration</th>
<th>Comments</th>
</tr>
</thead>
</table>
| **Severe Community Onset Infection of Unknown Source** | Gram negative organisms  
Staphylococcus aureus  
Streptococcus pneumoniae  
Neisseria meningitidis | Ceftriaxone 2g IV Q12H  
and  
Gentamicin 5mg/kg IV Q24H | Review empiric antibiotic choice at 48 hours (or as culture results become available). |
| **Central Line Sepsis**                | Coagulase negative Staph  
Staphylococcus aureus  
Gram negative bacilli  
Canidida | Flucloxacillin 2g IV Q6H  
and  
Gentamicin 5mg/kg IV Q24H  
Consider giving Vancomycin 1g IV Q12H | Same organism grown from central and peripheral blood cultures.  
Removal of infected catheter is often sufficient to “treat” localised access infection.  
If patient is clinically septic from invasive line or at risk of devastating infection (ie. prosthetic valve or graft) remove catheter and commence treatment. |
| **Severe Community Acquired Pneumonia** | Streptococcus pneumoniae  
Hemophilus influenze  
Mycoplasma pneumoniae  
Legionella pneumophilia  
Staphylococcus aureus  
Moraxella catarrhalis  
Klebsiella pneumoniae  
Chlamydia pneumonia  
Influenza | Erythromycin 1g IV Q6H  
and  
Augmentin 1.2g IV Q8H  
Erythromycin can be stopped when Legionella excluded | Severe CAP indicated by 2 or more of:  
- RR >30/min.  
- PaO₂ <60mmHg.  
- PaCO₂ >50mmHg.  
- Diffuse or multi-lobar infiltrate on CXR.  
- Hemodynamic compromise.  
- Mechanical ventilation  
Influenza is typically characterized by sudden}
<table>
<thead>
<tr>
<th>Ventilator Acquired Pneumonia or Severe Hospital Acquired Pneumonia</th>
<th>Multi-drug resistant organisms</th>
<th>Tazocin 4.5g IV Q8H</th>
<th>Pneumonia developing &gt;48 hours after initiation of mechanical ventilation or admission to hospital.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Staphylococcus pneumonia</td>
<td>or</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hemophilus influenzae E.coli Klebsiella Enterobacter Proteus Serratia Pseudomonas</td>
<td>Meropenem 1-2g IV Q8H</td>
<td></td>
</tr>
<tr>
<td></td>
<td>If Legionella suspected: add Erythromycin 1g IV Q6H or Ciprofloxacin 400mg IV Q12H until excluded.</td>
<td>Treatment duration 10 days</td>
<td></td>
</tr>
<tr>
<td>Treatment duration 7-10 days</td>
<td>Superinfection with Hemophilus and Staph aureus can cause severe pneumonia and should be covered empirically.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>Likely Pathogens</td>
<td>Antibiotic Dose And Duration</td>
<td>Comments</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------------------------------------------------</td>
<td>------------------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Aspiration Pneumonia</td>
<td>Aerobic gram negative bacilli</td>
<td>Augmentin 1.2g IV Q8H</td>
<td>Antibiotics are not indicated for cases of mild to moderate aspiration.</td>
</tr>
<tr>
<td></td>
<td>Staph. Aureus</td>
<td>or</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pseudomonas</td>
<td>Clindamycin 600mg IV Q8H</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anaerobes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Treatment duration 10 days</td>
<td></td>
</tr>
<tr>
<td>Infective Exacerbation COPD</td>
<td>Streptococcus pneumoniae Hemophilus influenzae</td>
<td>Augmentin 1.2g IV Q8H</td>
<td>Antibiotics are only effective when all 3 signs of bacterial infection are present:</td>
</tr>
<tr>
<td></td>
<td>Moraxella catarrhalis</td>
<td></td>
<td>• Increased dyspnoea.</td>
</tr>
<tr>
<td></td>
<td>Viruses</td>
<td></td>
<td>• Increased sputum production.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Treatment duration 5-10 days</td>
<td>• Purulent sputum.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>depending on clinical response</td>
<td>Positive sputum culture may indicate colonization only.</td>
</tr>
<tr>
<td>Intra-abdominal Sepsis</td>
<td>Enterobacteriaceae</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Enterococcus</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anaerobes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Streptococcus milleri (abcesses)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe Pancreatitis</td>
<td>Enterobacteriaceae</td>
<td>Meropenem 1g IV Q8H</td>
<td>Antibiotics are not indicated for pancreatitis unless evidence of infection.</td>
</tr>
<tr>
<td></td>
<td>Enterococcus</td>
<td></td>
<td>Surgical drainage is indicated when enhanced CT suggests infected necrosis.</td>
</tr>
<tr>
<td></td>
<td>Anaerobes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>Likely Pathogens</td>
<td>Antibiotic Dose And Duration</td>
<td>Comments</td>
</tr>
<tr>
<td>------------------------------------</td>
<td>------------------</td>
<td>------------------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Biliary Sepsis</strong></td>
<td>Enterobacteriaceae</td>
<td>Augmentin 1.2 g IV Q6H 6 and</td>
<td>Antibiotic therapy should be seen as an adjunct to drainage.</td>
</tr>
<tr>
<td></td>
<td>Enterococcus</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anaerobes</td>
<td>Gentamicin 5 mg/kg IV Q24H</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>or Metronidazole 500mg IV Q8H</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>or Cefriaxone 1g IV Q24H</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>or Tazocin 4.5g IV Q8H</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Treatment duration 5-10 days</td>
<td></td>
</tr>
<tr>
<td><strong>Infection Likely Pathogens</strong></td>
<td><strong>Antibiotic Dose and Duration</strong></td>
<td><strong>Comments</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Bacteruria</strong></td>
<td>Enterobacteriaceae</td>
<td>Meropenem 1g IV Q8H</td>
<td>Remove urinary catheter if possible.</td>
</tr>
<tr>
<td></td>
<td>Pseudomonas</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Treatment should only be considered if patient shows signs of systemic illness / sepsis, supported by significant pyuria, is pregnant or is undergoing genitourinary instrumentation.</td>
</tr>
<tr>
<td><strong>UTI Precipitating ICU Admission</strong></td>
<td>Enterobacteriaceae</td>
<td>Meropenem 1g IV Q8H</td>
<td>Treat as for severe pyelonephritis.</td>
</tr>
<tr>
<td></td>
<td>Pseudomonas</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>or</td>
<td></td>
</tr>
</tbody>
</table>
### Hospital acquired or complicated UTI

<table>
<thead>
<tr>
<th>Organism</th>
<th>Antimicrobial Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.coli.</td>
<td>Meropenem 1g IV Q8H or Cefepime 1-2g IV Q12H</td>
</tr>
<tr>
<td>Enterococcus</td>
<td></td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>Particularly associated with instrumentation, indwelling catheter or nephrostomy, immunosuppression, renal transplant, male, renal tract obstruction or structural abnormality, renal failure, pregnancy, diabetes.</td>
</tr>
</tbody>
</table>

| Treatment duration | 7 days |

### Cellulitis or Soft Tissue Infection

<table>
<thead>
<tr>
<th>Organism</th>
<th>Antimicrobial Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptococcus pyogenes</td>
<td>Flucloxacillin 1g IV Q6H</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>Change to Benzylpenicillin 1.2g IV Q4H if Strep</td>
</tr>
<tr>
<td>Enterobacteriaceae</td>
<td></td>
</tr>
<tr>
<td>Anaerobes</td>
<td></td>
</tr>
</tbody>
</table>

| Treatment duration | 5 days |

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Change to Benzylpenicillin 1.2g IV Q4H if Strep</td>
<td></td>
</tr>
</tbody>
</table>

### Complicated cellulitis involving ulcer

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Flucloxacillin 1g IV Q6H and Augmentin 1.2g IV Q8H</td>
<td></td>
</tr>
</tbody>
</table>

| Treatment duration | 10 days |

| Benzylpenicillin 2.4g IV Q4H and Ceftazidime 2g IV Q8H and Clindamycin 600mg IV Q8H | Suspect Necrotizing Fasciitis if: |

| or Meropenem 1g IV Q8H or Ciprofloxacin 400mg IV Q12H and Clindamycin 600mg IV Q6H |
|---|---|

| Rapidly progressive inflammation and necrosis of the skin and underlying tissues. |
|---|---|
| Oedema beyond the area of erythema. |
| Tissue crepitus. |
| Systemic toxicity. |
| Pain out of keeping with the clinical findings. |

Antibiotic therapy is not a substitute for emergent surgical debridement.
### Suspected Bacterial Endocarditis

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Antibiotic &amp; Duration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viridans streptococcus</td>
<td>Benzylpenicillin 1.8g IV Q4H and Flucloxacillin 2g IV Q4H and Gentamicin 3mg/kg IV Q24H</td>
<td>All patients with suspected endocarditis should have at least 3 sets of blood cultures taken from 3 different sites prior to starting antibiotic therapy.</td>
</tr>
<tr>
<td>Streptococcus bovis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HACEK organisms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enterococci</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Native valve</em></td>
<td></td>
<td>Discuss with ID and Cardiology to direct treatment and investigations.</td>
</tr>
<tr>
<td><em>Prosthetic valve</em></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Infection Likely Pathogens Antibiotic Dose And Duration Comments

<table>
<thead>
<tr>
<th>Infection</th>
<th>Likely Pathogens</th>
<th>Antibiotic Dose And Duration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suspected Systemic Fungal Disease</td>
<td>Candida</td>
<td>Fluconazole 200-400 mg IV Q24H (adjusted to renal function) or Caspofungin 70mg IV then 50mg IV Q24H or Amphotericin B 0.5-1.5 mg/kg IV Q24H</td>
<td>Candida albicans and Candida parapsilosis are sensitive to Fluconazole. Candida glabrata and Candida krusei are often not susceptible to Fluconazole.</td>
</tr>
<tr>
<td>Viral (HSV) Encephalitis or Meningo-</td>
<td>Herpes Simplex Virus</td>
<td>Acyclovir 10 mg/kg IV Q8H</td>
<td>Consider HSV if focal neurology, seizures or altered LOC.</td>
</tr>
</tbody>
</table>
**Encephalitis**

Fever and personality changes uniformly present in HSV encephalitis.

LP abnormal with mononuclear cells.

PCR on CSF very sensitive and specific.

<table>
<thead>
<tr>
<th>Bacterial Meningitis</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptococcus pneumonia</td>
<td>Ceftriaxone 2g IV Q24H</td>
<td>Meningitis is the one exception to getting cultures prior to administering antibiotics – if LP cannot be done promptly give empiric antibiotics first.</td>
</tr>
<tr>
<td>Neisseria meningitides</td>
<td>Gram positive cocci on CSF gram stain:</td>
<td></td>
</tr>
<tr>
<td>Hemophilus influenza</td>
<td>add</td>
<td></td>
</tr>
<tr>
<td>Listeria monocytogenes</td>
<td>Dexamethasone 10mg IV and Vancomycin 30mg/kg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>If Listeria possible:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>add</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Amoxicillin 2g IV Q4H</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Treatment duration:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Meningococcal 3 days</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pneumococcal 10 days</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Listeria 14 days</td>
<td></td>
</tr>
</tbody>
</table>

**Burns**

No antibiotics indicated unless infection proven.
<table>
<thead>
<tr>
<th>Microbe/Infection</th>
<th>1st Choice</th>
<th>2nd Choice</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gram Positive Organisms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Staphylococcus aureus</strong></td>
<td>Flucloxacillin 2g IV Q6H</td>
<td>Cephazolin</td>
</tr>
<tr>
<td>MRSA (10%)</td>
<td>Vancomycin 1g IV Q12H</td>
<td>Refer to ID</td>
</tr>
<tr>
<td>Coagulase Negative Staph (frequently contaminants)</td>
<td>Vancomycin 1g IV Q12H</td>
<td>Daptomycin</td>
</tr>
<tr>
<td>Coagulase Negative Staph</td>
<td></td>
<td>Linezolid</td>
</tr>
<tr>
<td>Coagulase Negative Staph</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coagulase Negative Staph (frequently contaminants)</td>
<td>Vancomycin 1g IV Q12H</td>
<td>Daptomycin</td>
</tr>
<tr>
<td>Streptococcus pneumoniae</td>
<td>Benzylpenicillin 1.2g IV Q4-6H</td>
<td>Ceftriaxone 1g IV Q24H</td>
</tr>
<tr>
<td>Beta Hemolytic Strep</td>
<td>Benzylpenicillin 1.8g IV Q4H</td>
<td></td>
</tr>
<tr>
<td>Viridans Strep</td>
<td>Benzylpenicillin 1.8g IV Q4H</td>
<td></td>
</tr>
<tr>
<td>Enterococci</td>
<td>Amoxycillin 1-2g IV Q6H</td>
<td>Vancomycin 1g IV Q24H</td>
</tr>
<tr>
<td>Clostridium Skin Infection</td>
<td>Surgical debridement,</td>
<td>Surgical debridement,</td>
</tr>
<tr>
<td></td>
<td>Benzylenicillin 2.4g IV Q4H and</td>
<td>Meropenem 1-2g IV Q8H</td>
</tr>
<tr>
<td></td>
<td>Gentamicin 5mg/kg IV Q24H and</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Metronidazole 500mg IV Q12H</td>
<td></td>
</tr>
<tr>
<td>Clostridium Difficile Diarrhoea</td>
<td>Vancomycin 125mg po Q6H</td>
<td></td>
</tr>
<tr>
<td>Anaerobic cocci (Peptostreptococcus)</td>
<td>Augmentin 1.2gIV Q8H</td>
<td>Metronidazole 500mg IV Q8H</td>
</tr>
<tr>
<td>Listeria</td>
<td>Amoxycillin 2g IV Q4H and</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gentamicin 3-5mg/kg IV Q24H</td>
<td>Cotrimoxazole</td>
</tr>
<tr>
<td>Gram Negative Organisms</td>
<td>1st Choice</td>
<td>2nd Choice</td>
</tr>
<tr>
<td>------------------------</td>
<td>------------</td>
<td>------------</td>
</tr>
<tr>
<td>E.coli</td>
<td>Gentamicin 5mg/kg IV Q24H (switch to beta lactam as soon as sensitivities available).</td>
<td>Ceftriaxone 1g IV Q24H</td>
</tr>
<tr>
<td>Klebsiella</td>
<td>Gentamicin 5mg/kg IV Q24H (switch to beta lactam as soon as sensitivities available).</td>
<td>Ceftriaxone 1g IV Q24H</td>
</tr>
<tr>
<td>ESBL producing E.coli and Klebsiella</td>
<td>Meropenem 1g IV Q8H</td>
<td></td>
</tr>
<tr>
<td>Enterobacter</td>
<td>Gentamicin 5mg/kg IV Q24H</td>
<td>Ciprofloxacin 200-400mg IV Q12H</td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>Dual antibiotic therapy: Tazocin 4.5g IV Q6H and Gentamicin 5mg/kg IV Q24H</td>
<td>Dual antibiotic therapy: Cefepime 1-2g IV Q12H and Ciprofloxacin 400mg IV Q12H</td>
</tr>
<tr>
<td>Stenotrophomonas</td>
<td>Cotrimoxazole</td>
<td></td>
</tr>
<tr>
<td>Legionella species</td>
<td>Erythromycin 500mg-1g IV Q6H</td>
<td>Ciprofloxacin 400mg IV Q12H</td>
</tr>
<tr>
<td>Neisseria meningitidis</td>
<td>Benzylpenicillin 1.2g IV Q4-6H</td>
<td>Ceftriaxone 2g IV Q24H</td>
</tr>
<tr>
<td>Hemophilus influenzae</td>
<td>Ceftriaxone 1g IV Q24H</td>
<td>Augmentin 1.2g IV Q8H</td>
</tr>
<tr>
<td>Anaerobic bacilli</td>
<td>Augmentin 1.2g IV Q8H</td>
<td>Metronidazole 500mg IV Q8H</td>
</tr>
<tr>
<td>Yeast</td>
<td>1st Choice</td>
<td>2nd Choice</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-----------------------------------</td>
<td>-----------------------------------</td>
</tr>
<tr>
<td><strong>Candida albicans</strong></td>
<td>Fluconazole 400mg IV</td>
<td>Amphotericin B</td>
</tr>
<tr>
<td><strong>Candida parapsilosis</strong></td>
<td>Fluconazole 400mg IV</td>
<td>Amphotericin B</td>
</tr>
<tr>
<td><strong>Candida tropicalis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Candida glabrata</strong></td>
<td>Caspofungin (check susceptibility)</td>
<td>Amphotericin B (check susceptibility)</td>
</tr>
<tr>
<td><strong>Candida krusei</strong></td>
<td>Caspofungin</td>
<td>Voriconazole</td>
</tr>
<tr>
<td><strong>Candida lusitaniae</strong></td>
<td>Fluconazole</td>
<td>Caspofungin</td>
</tr>
<tr>
<td><strong>Cryptococcus</strong></td>
<td>Fluconazole</td>
<td>Amphotericin B</td>
</tr>
<tr>
<td><strong>Pneumocystis</strong></td>
<td>Co-trimoxazole 320/1600mg IV Q6H +</td>
<td>Pentamidine 4 mg/kg/day IV +</td>
</tr>
<tr>
<td></td>
<td>methylpred 40mg BD x 5 days, then</td>
<td>Steroid therapy</td>
</tr>
<tr>
<td></td>
<td>methylpred 40mg daily x 5 days, then</td>
<td></td>
</tr>
<tr>
<td></td>
<td>methylpred 20mg daily x 11 days</td>
<td></td>
</tr>
<tr>
<td><strong>Mold</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aspergillus</strong></td>
<td>Caspofungin</td>
<td>Amphotericin B</td>
</tr>
</tbody>
</table>

**REFERENCES**


UpToDate.
PAEDIATRICS

A number of paediatric patients (usually around 200) are admitted to the Waikato Hospital ICU each year. Registrar staff need to maintain full involvement with these patients irrespective of their previous experience with children. Liberal involvement of the Paediatric Specialist responsible for the child and their resident staff may be required to assist in management.

However, as with the adult patients in the ICU and unless the question is clearly outside the provenance of intensive care (e.g. napkin rash, questions of dietary intolerance, immunisation etc.), the intensivist should be involved first. Many patients (particularly postoperative patients) are here for nursing care, but the ICU registrar will meet many of their basic medical needs.

PAEDIATRIC ADMISSION POLICY

A patient under the age of 15, or if under the care of a paediatrician, is considered paediatric. While patients under the age of 15 years requiring HDU care are generally managed in the ICU, common sense needs to prevail. Conditions commonly managed in the HDU and never in the ICU (e.g. venous thrombosis) in a child close to the cut-off age should logically be managed in the HDU. Older diabetic children are frequently under the care of an adult Diabetologist, and care in HDU may be appropriate.

At the other end of the spectrum, it may be appropriate for carefully selected neonates who have already left hospital or who were “outborn” to be managed in the Newborn unit. The responsible Paediatrician, Neonatologist and Intensivist should liaise to determine appropriate disposition. Clinical assessment is based significantly on the derangement of physiological parameters from normal. In health these values are determined by the child's age, settling at what can be considered adult values in the early teenage years. Infants less than 5kg will generally be managed in the NICU but this is often on a case by case basis.

Patients younger than their eighth birthday, anticipated to need >24 hours invasive ventilation should be discussed with Starship with a view to transfer when possible. Patients older than their eighth birthday (but of course prior to their 15th birthday) who in addition to a projected requirement for invasive ventilation > 24h require inotrope/vasopressor infusion or renal replacement therapy should be discussed with Starship with a view to transfer when possible. Clearly there are patients of 13 to 14 years age who may be considered adult and some flexibility is important, without constantly “pushing” the lower limit of this age criterion. Such 13 and 14 year olds may be considered “adult” if: they present with adult diseases (e.g. overdose, alcohol intoxication, driver involved in road accident), and to some extent if they will be likely be managed by adult specialists at the hospital during their entire stay.

The reader is referred to the very detailed document in the Paediatric Guideline folder for guidance on the specifics of management of paediatric patients within ICU, including so called HDU admissions to ICU and the details of drug prescription, responsibility for discharge etc.

HDU have a complementary policy which describes management of that age group in that unit.

In general, the care of paediatric patients follows that of adult patients, with some differences that it is important to be aware of.

Weight: It is important to know the weight of the child being admitted, as this determines rate of fluid administration and drug dosing. All medications should be clearly prescribed as amount/weight/time. E.g. mcg/kg/min, mg/kg/dose. This will be checked by the bedside nurse prior to any dose being administered.
The recommended source for medication dosing in children is the book “drug doses” by Frank Shann, available on the paediatric trolley. There is also a BNF for children available.

In general, children > 40kg receive standard adult doses, and no child should receive a dose higher than that recommended for adults.

Weight can be approximated by:

<table>
<thead>
<tr>
<th>Age</th>
<th>Wt (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn/6months</td>
<td>~3.5kg/~7kg</td>
</tr>
<tr>
<td>1-9 years</td>
<td>(2 x age) + 4</td>
</tr>
<tr>
<td>&gt;9 years</td>
<td>3 x age</td>
</tr>
</tbody>
</table>

What follows is an ABCD approach to the physiological and anatomical factors that affect assessment and management of the paediatric patient.

**A Weight Based Paediatric Resuscitation Sheet Printout for Vitals / Drugs is Available on the ICU Computer Desktop to Assist in Management**

**GENERAL PAEDIATRIC CARE IN ICU**

**AIRWAY**

Many patients will be intubated prior to ICU arrival. For children who need intubating in the unit, a senior clinician with experience with paediatric airway should be present. Tracheal tube size selection can be estimated from the formula: Age/4 + 4.

The expected orotracheal tube length can be estimated by: Age/2 + 12 and nasally by Age/2 + 15.

Traditionally cuffed tubes have been avoided in very young children over concerns of sub-glottic trauma, although this is contentious. Cuffed tubes can allow greater protection against pulmonary aspiration and minimise ventilator leak by creating an airtight seal with the tracheal wall.

At Waikato Hospital the smallest cuffed tracheal tubes we have are size 3.0. From a practical point of view any child under 4 years should probably have an uncuffed tracheal tube.

**Any child that will require a size 5.0 ETT or larger should have a cuffed tube**

Please remember this, especially in the ED.

If choosing a cuffed tube consider using a “half-size” smaller ETT.
NB The narrowest part of a child’s airway is at the level of the cricoid cartilage. If resistance is encountered having passed the tracheal tube through the glottis, select a smaller tube size.

In children less than 5 years old a nasal ETT is usually inserted after orotracheal intubation to assist with tube stability unless a contraindication exists. If such a child is being transferred to ICU from theatre or from another hospital, please ask for this.

There are several differences to be aware of:

**Anatomy.**

Children have relatively large heads, and if on a pillow their necks will be over flexed. It is usually necessary to remove the pillow, and sometimes place a folded blanket under the shoulders to achieve neutral alignment. Over extending the head and neck can cause kinking and obstruction to soft airways and can make bag mask ventilation impossible. Children have small mouths, large tongues and high, anterior larynxes which can all make intubation challenging. The epiglottis is long and floppy in babies, they are usually easier to intubate with a straight (miller) blade to lift the epiglottis. A stylet in the ETT to mould a ‘hockey stick’ shape can make intubation easier. Cricoid pressure is best avoided in babies and young children (no evidence it helps, and can be physically difficult to apply as well as obscuring view).

The initial intubation is oral, however converting to a nasal tube is preferred, as this is better tolerated and easier to secure. The method of securement is outlined in the information folder on the paediatric trolley. The trachea is short, so even if the ETT is correctly positioned on initial CXR, it is important to secure the tube firmly, and prevent excessive head movement (head extended = ETT high, head flexed = ETT low).

**Physiology.**

Young children readily become bradycardic in response to suxamethonium given for intubation; consideration should be given to pre-treatment with atropine (20mcg/kg, minimum 100mcg). An alternative to suxamethonium is 1mg/kg rocuronium for intubation. Children have higher metabolic rates than adults and lower FRC, and therefore desaturate rapidly. Pre oxygenation and rapid securement of the airway is therefore crucial - ensure you can bag-mask ventilate the child prior to giving a long acting relaxant for intubation. LMAs in appropriate sizes are also available in an emergency.
<table>
<thead>
<tr>
<th>Age</th>
<th>ETT size*</th>
<th>ETT length at lip</th>
<th>ETT length at nostril</th>
<th>LMA size</th>
<th>Suction catheter size</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 months</td>
<td>3.5mm</td>
<td>10</td>
<td>12</td>
<td>1 (&lt;5kg)</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.5 (5-10kg)</td>
<td></td>
</tr>
<tr>
<td>1 year</td>
<td>4.0mm</td>
<td>11</td>
<td>14</td>
<td>1.5 (5-10kg)</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 (10-20kg)</td>
<td></td>
</tr>
<tr>
<td>&gt; 1 year</td>
<td>4 +Age/4</td>
<td>12+ Age/2</td>
<td>15+ Age/2</td>
<td>2.5 (20-30kg)</td>
<td>2X size ETT</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3 (30-50kg)</td>
<td></td>
</tr>
</tbody>
</table>

*ETT. A leak should be present at a pressure of 20cm of water PPV. ETT can be uncuffed (usual) or cuffed (cuff usually left deflated) to ensure the right fit.

**BREATHING**

Due to their higher metabolic rate, children have a higher respiratory rate than adults, and respiratory distress is a common finding in children who are ill for any reason. Signs of respiratory distress include grunting, nasal flaring, subcostal recession, paradoxical bradypnoea and head bobbing, in addition to the ‘adult’ signs of tachypnoea, cyanosis, indrawing and tracheal tug.

Young children are diaphragmatic breathers, and tolerate gastric distension poorly. An NGT may be needed, and is mandatory on intubated children.

**NIV.**

Widely used, particularly in children with bronchiolitis or asthma. If tolerance is an issue, it may be appropriate to give a dose of sedative such as chloral hydrate, 15-30mg/kg NG, as long as the child is appropriately monitored.

**POSITIVE PRESSURE VENTILATION.**

Preferred mode is SIMV pressure control (rather than volume), as it is better able to compensate for an ETT leak. A good starting point is PEEP 5, PIP 15-20, aiming for TV 6-8ml/kg, RR 18-25 (titrate to CO2). Chest should be seen to rise and fall. Manipulation of ventilator parameters is similar to that in adults.

Respiratory rate should be assessed at rest in isolation of medical or nursing cares.

Also note the extent of chest excursion, use of accessory muscles, tracheal tug, nasal flaring, sternal or subcostal recession, the presence of inspiratory or expiratory noises and the presence of cyanosis.

Normal age related respiratory rate is tabulated below:
NORMAL RESPIRATORY RATES

<table>
<thead>
<tr>
<th>Age</th>
<th>Respiratory rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>30-40</td>
</tr>
<tr>
<td>1-2</td>
<td>25-35</td>
</tr>
<tr>
<td>2-5</td>
<td>25-30</td>
</tr>
<tr>
<td>5-12</td>
<td>20-25</td>
</tr>
<tr>
<td>&gt;12</td>
<td>15-20</td>
</tr>
</tbody>
</table>

Having given these values, infants are not usually considered significantly tachypnoeic until RR exceeds 60.

DON'T GET CAUGHT OUT:

Normal oxygen saturation on oxygen therapy does not preclude significant respiratory embarrassment.

A slow respiratory rate in a sick child should be considered pre-terminal.

It is common for a referral to include the statement “(s)he is getting tired‖. If clinical exhaustion is occurring it is highly significant and should be acted on. Don’t forget though that a child whose general condition is improving (e.g. response to bronchodilators) or even one who is not so well may be catching up on lost sleep. The more reliable signs of clinical exhaustion include obtundation and confusion. Conscious level impairment should be formally and carefully evaluated as in any other patient. Hunger, handling, parental absence and performance of monitoring / procedures is usually distressing to children. Agitation then is neither sensitive nor specific as a measure of serious disease in children without other objective measures.

Tachypnoea with a clear chest x-ray often signifies acidosis or shock.

A study of grunting respiration in paediatric patients revealed a high level of serious disease—not all of it was pulmonary in origin.

ISSUES SURROUNDING ARTIFICIAL VENTILATION

Under normal conditions aim for tidal volumes of 10mls/kg and rate as suggested above.

PEEP “dose” is the same at any age.
Positive pressure ventilation with an uncuffed tube often results in a significant air leak. Monitor expiratory gas volumes to assess adequacy of ventilation. Pressure controlled ventilation may provide greater constancy of tidal volume under these circumstances. Setting a larger tidal volume, in volume control modes, to accommodate the leak could be dangerous should the leak disappear eg with re-positioning of the child.

It may, of course, be appropriate to change the tracheal tube to a cuffed version or larger size.

Blood gas analysis, capnography or transcutaneous carbon dioxide measurement can assess adequacy of ventilation. Pulse-oximetry does not allow assessment of ventilation.

**It is mandatory to use capnography in every intubated paediatric patient in Waikato Hospital ICU unless overriding reasons exist**

Sedation involves opiates, benzodiazepines and sometimes other agents such as chloral hydrate. Propofol may be used but never in high dose or prolonged periods due to the risk of propofol infusion syndrome.

**CIRCULATION**

Children have comparatively higher water content than adults. They dehydrate easily. Stroke volume is relatively fixed, so tachycardia is the main response to poor cardiac output. Hypotension is a late sign of circulatory insufficiency, it is important therefore to note earlier signs such as prolonged capillary return (>2-3secs). Fluid resuscitation in the ICU is generally given IV as bolus warmed 0.9% saline, 10-20ml/kg in the absence of significant cardiac disease. As circulating blood volume is 80ml/kg, by the time the child has received 2 boluses (total 40ml/kg, ½ circulating volume), consideration should be given to administering blood products. 4ml/kg packed cells can be expected to raise Hb by 10g/l. If in a hurry or using an IO access use a 3-way tap and syringe. Calling for someone more experienced to put a drip in during a life-threatening emergency is an indication for you to place an IO access before they arrive.

Full maintenance IV fluid is usually 5% dextrose with 0.45% saline or 0.9% saline. (0.9% saline with dextrose preferred in head injury / neurosurgical patients) 'Rule of thumb' maintenance fluid for a WELL child is:

**4/2/1 RULE OF FLUIDS. GIVE.....**

- 4ml/hr/kg for first 10kg of weight +
- 2ml/kg/hr for next 10 kg weight +
- 1ml/kg/hr thereafter

Note that this is recommended for a well child, not a sick child who is ventilated in ICU. Give ~ 2/3 of recommended amount to ventilated children. (sick, ventilated children have increased ADH production, leaving them vulnerable to water retention / fluid overload and hyponatraemia.) Consider giving maintenance fluid as NG breast milk / formula / standard enteral nutrition if appropriate. What ever amount of volume is given, accurate 24 hours fluid balance is essential.

Urine output is comparatively higher in small children. A baby should have a urine output 2ml/kg/hr minimum, an older child 1ml/kg/hr minimum. An IDC is not mandatory, but moderate opiate doses (>20mcg/kg/hour) will
lead to sphincter dysfunction and urinary retention, so in a child with low urine output, estimation of bladder volume is paramount.

Some children (eg: those with gastro) will need slow rehydration, depending on estimated degree of dehydration / fluid deficit.

DEFG.

Don’t ever forget glucose. Small children have low glycogen stores and utilize available glucose rapidly, making them prone to hypoglycaemia. BSL should be monitored regularly, and fluids should contain dextrose. Any child with a low GCS or seizure should have a BSL measured, and glucose administered (5ml/kg 10% dextrose) if found to be low.

Assess for signs of end-organ hypoperfusion such as cool peripheries, delayed capillary refill, oliguria (<1ml/kg/hour), agitation, drowsiness and coma. Tachypnoea is an early sign of shock. Tachycardia can be difficult to interpret in the distressed child, but should be taken seriously, especially if there is other evidence of shock.

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>110-160</td>
</tr>
<tr>
<td>1-2</td>
<td>100-150</td>
</tr>
<tr>
<td>2-5</td>
<td>95-140</td>
</tr>
<tr>
<td>5-12</td>
<td>80-120</td>
</tr>
<tr>
<td>&gt;12</td>
<td>60-100</td>
</tr>
</tbody>
</table>

Expected systolic blood pressure can be calculated by: 80 + (age x 2). Don’t get caught out:

**Hypotension is a late sign and thus not required for a diagnosis of shock**
Defining level of consciousness can be difficult in young children. A modified version of the GCS can be used, but remember this is not what it was developed for. Valuable information can be gained from the parents.

### AGE APPROPRIATE COMA SCORING

<table>
<thead>
<tr>
<th>Eye Score</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>3</td>
<td>Speech</td>
</tr>
<tr>
<td>2</td>
<td>Pain</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Motor Score</th>
<th>&lt; 12 months</th>
<th>&gt; 12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>Normal movement</td>
<td>Obeys commands</td>
</tr>
<tr>
<td>5</td>
<td>Localises to supraocular pain</td>
<td>Localises to supraocular pain</td>
</tr>
<tr>
<td>4</td>
<td>Flexion withdrawal from nailbed pressure</td>
<td>Flexion withdrawal from nailbed pressure</td>
</tr>
<tr>
<td>3</td>
<td>Abnormal flexion to supraocular pain</td>
<td>Abnormal flexion to supraocular pain</td>
</tr>
<tr>
<td>2</td>
<td>Extension to supraocular pain</td>
<td>Extension</td>
</tr>
<tr>
<td>1</td>
<td>No response to supraocular pain</td>
<td>No response to supraocular pain</td>
</tr>
<tr>
<td>Verb al Score</td>
<td>&lt; 2 years</td>
<td>2-5 years</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
<td>-----------</td>
</tr>
<tr>
<td>5</td>
<td>Babbles, words or sentences as normal</td>
<td>Appropriate</td>
</tr>
<tr>
<td>4</td>
<td>Less than usual ability</td>
<td>Inappropriate</td>
</tr>
<tr>
<td>3</td>
<td>Cries to pain</td>
<td>Cries/ screams</td>
</tr>
<tr>
<td>2</td>
<td>Moans to pain</td>
<td>Moans to pain</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

A child with a GCS of 8 (or unresponsive to pain in the AVPU system) or under should be considered unconscious and unable to protect their own airway. This may necessitate tracheal intubation. Don't forget to exclude hypoglycaemia as a cause of decreased consciousness.

Hypoglycaemia is an ever present risk in critically ill children especially if not fed enterally.

Regular glucose monitoring is necessary. See nursing policies.

**DRUGS**

Use Drug Doses book or other recognized sources for drug dosing.

Try and give or supplement potassium intake enterally whenever possible. It is rarely practical to put a central line in conscious children. Concentrated potassium infusions are never given via peripheral veins in any patient irrespective of age.

**INVESTIGATIONS**

It may well be reasonable for paediatric patients to receive few blood tests or chest X-rays on admission to hospital or during their stay. When however they are referred for consideration of intensive care admission a more liberal, yet still considered approach is necessary. Basic haematology, biochemistry and blood culture tests are often necessary. A chest X-ray is commonly required, but always for respiratory conditions.
“Microcollects” can be arranged for the morning bloods and will often be performed by lab staff if the Night ICU registrar speaks kindly to the Nursing staff to arrange.

SOURCES OF HELP

Lines of communication are established above and in the Paediatric Admission policy.

The same offer made to contact the Intensivist on duty for matters of concern exists as for any patient in or referred to ICU.

Informal contact with paediatric resident staff may be helpful over some matters, but needs to be treated with the same caution as when you are informally approached by other resident staff for advice.

These guidelines.

Two specific references are chained in the workroom for your use

Shann’s “Drug Doses” is very useful to carry with you.

At the bedside of each paediatric patient should be an individualised weight based print out of the online Starship Drug calculator which now includes a “Waikato ICU” drop down menu including resuscitation drugs and IV infusions.

REFERENCE:


ACUTE ASTHMA MANAGEMENT

DIAGNOSING ASTHMA

Diagnosis of asthma is likely if the following are present:

- A history of fluctuating wheeze
- This may fluctuate spontaneously or in response to bronchodilators or steroids
- Three or more episodes of wheeze

It may be hard to tell with the chronically wheezy infant or child. A fixed obstruction must be ruled out if the wheeze is continuous. Do not diagnose on the first or second episode.

PHYSICAL SIGNS OF AIRWAYS OBSTRUCTION

- These are the signs elicited on the Asthma Severity Score (see over page) plus over-inflation and prolonged expiration
- No alternative diagnosis
- Such as foreign body, cystic fibrosis. If in doubt ask your consultant
Family History

There is often a family history of atopy

Age

Asthma is rarely diagnosed in a child who is less than 1 year old (check with your consultant). For those 1 to 2 years old, a higher degree of certainty is required than for older children. In particular, consider whether the child may have acute bronchiolitis or bronchopneumonia.

If in doubt discuss with a senior

ASSESSMENT OF ASTHMA SEVERITY

SIGNS OF LIFE-THREATENING ASTHMA

- Respiratory – hypoxia/exhaustion
- Neurological – agitation, confusion, drowsiness
- Cardiovascular – pulsus paradoxus, worsening tachycardia

Consider diagnoses other than asthma, especially in infants with poorly responsive respiratory distress.

In very severe cases, because of extremely poor air entry, you may not hear wheeze.

Deterioration despite maximal therapy on severe asthma pathway.

Oxygen saturations can remain normal in life-threatening asthma

SEVERE ASTHMA

- Too breathless to talk or feed
- HR > 120/min (over 5 years) or >130/min (2-5 years old)
- RR > 30/min (over 5 years) or > 50/min (2-5 years old)
- Marked accessory muscle use
### Asthma Severity Score (ASS)

Add wheeze and muscle subtotals to give score

#### Wheeze (beware of silent chest*) Score

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<td>Heard without stethoscope</td>
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**Sub Total**

#### Accessory Muscle Use / Indrawing

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**Sub Total**

**TOTAL**
ACUTE ASTHMA MANAGEMENT ALGORITHM

MILD ASTHMA A.S.S = 0-2
- Salbutamol 6 puffs of 100mcg MDI via spacer. Repeat salbutamol only if indicated.
- Consider prednisolone or prednisone 1mg/kg

MODERATE ASTHMA A.S.S = 3-5
- Oxygen if SpO2 in air < 92%
- Salbutamol 6 puffs of 100mcg MDI via spacer every 20 minutes for up to 1 hour.
- Prednisolone or prednisone 1mg/kg (max 40mg)

SEVERE ASTHMA A.S.S = 6
- Oxygen
- Salbutamol 6 puffs of 100mcg MDI via spacer or 5mg nebul. Every 20 minutes for up to 1 hour.
- Ipratropium bromide 4 puffs of 20mcg MDI via spacer or 0.25mg nebul. Every 20 minutes (max 3 doses in 1 hour)
- Prednisolone or prednisone 1mg/kg (max 40mg)

If not improving:
- Review by senior
- Continuous salbutamol
- Consider further management according to life-threatening asthma guideline

Rеassess Sеverity аt 60 minutes

Discharge
see Discharge Guidelines

Continue management in CED with frequent reassessment

Admit
see Guidelines for Admission

Re-assess severity every 1-2 hours in CED / short stay

If patient’s condition deteriorates, reassess, and manage as LIFE-THREATENING ASTHMA

In CED discuss with CED senior
On wards inform senior & arrange PICU referral
STEROIDS

Prednisolone syrup (Redipred®) is the preferred medication for young children because it is better tolerated and easy to use. Prednisone tablets can be used for older children who are able to swallow them.

- Mild asthma Consider on basis of history
- Moderate or severe asthma Dosage is 1mg/kg/day once daily (max 40mg) for 3-5 days
- Severe May require IV hydrocortisone if not tolerating oral medication or if slow to respond

REFERENCES:


LIFE THREATENING ASTHMA

RECOGNITION OF LIFE-THREATENING ASTHMA

- Deterioration despite maximal therapy (see severe asthma treatment section)
- Respiratory – cyanosis/exhaustion
- Neurological – confusion/drowsiness
- Cardiovascular – pulsus paradoxus

Consider diagnoses other than asthma, especially in infants with poorly responsive respiratory distress.

No infant (< 1 year) should be started on intravenous bronchodilators without discussion with a consultant

MANAGEMENT

If the patient's condition is improving therapy can be de-escalated at any stage – see “Severe” section of Asthma Guideline:

- Call for assistance
- OXYGEN – use high flow oxygen via mask (e.g. 15L/min)
- IV access
- Give Hydrocortisone 4 mg/kg IV as soon as possible
• Nebulised bronchodilators - Continuous nebulised salbutamol 5 mg/dose for all ages. Add ipratropium bromide 0.25 mg to the second nebuliser, if there is inadequate response to the first salbutamol nebul. Repeat ipratropium every 20 minutes for 3 doses, then every 4 hours

• IV salbutamol bolus Give 10 micrograms/kg (single dose maximum 500 micrograms). Over 2 minutes. Give in a minimum volume of 5ml (can be diluted with 0.9% Saline). Repeat dose at 10 minutes if still not improving

• IV magnesium sulphate bolus. Use magnesium sulphate 49.3% (493mg/ml). Give 0.1 ml/kg (approx 50mg/kg) over 20 minutes (dilute to 20mls with normal saline and infuse via syringe driver). Maximum dose 5 mls (2.5 g) (1 ampoule = 10 mmol = 2.5 g)

• IV aminophylline bolus. Give 10 mg/kg IV (maximum dose 500 mg) over 1 hour (dilute to 1mg/ml – the total volume will be 10ml/kg, compatible with fluid containing Sodium chloride and/or Dextrose and/or Potassium). If the child is already on oral theophylline, do not give IV aminophylline unless you have obtained a baseline serum level and can calculate a reduced loading dose. If patient is on any other medications you must check for potential interactions and adjust dose accordingly (see below)
If inadequate response to bolus therapy then start further IV therapy in form of salbutamol +/- aminophylline infusion(s). These children require admission to PICU

Remember if child is improving therapy can be de-escalated at any stage.

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SALBUTAMOL INFUSION

Dose: 5 - 10 microgram/kg/min for 1 hour then reduce to 1 - 2 microgram/kg/min.

- If Patient Weight < 16kg: Add 3 mg/kg of IV salbutamol solution (1 mg/ml) to a 50 ml syringe and make up to 50 ml with 5% dextrose, then 1 ml/hr = 1 microgram/kg/min
- If Patient Weight > 16kg: Draw up neat IV salbutamol solution (1 mg/ml) into a 50ml syringe (i.e. not diluted), then rate (ml/hr) = 0.06 x weight (kg) x dose (microgram/kg/min)

For example if you have a 20 kg child and want to infuse salbutamol at 5 microgram/kg/min then set rate at 0.06 x 20 x 5 = 6 ml/hr.

AMINOPHYLLINE INFUSION

**DOSE IF PATIENT AGED 1 – 9 YEARS: 1.1 MG/KG/HOUR.**

- Add 55 mg/kg of IV aminophylline solution (25 mg/ml) to a 50 ml syringe and make up to 50 ml with 5% dextrose, then infuse at 1 ml/hr
- If weight between 23-30kg (50th centile for 9 year olds) then use neat IV aminophylline solution (25mg/ml) in a 50ml syringe and run at 1ml/hr

**DOSE IF PATIENT AGED 10 – 15 YEARS AND WEIGHT < 35 KG**

- 0.7 mg/kg/hour: Add 35 mg/kg of IV aminophylline solution (25 mg/ml) to a 50 ml syringe and make up to 50 ml with 5% dextrose, then infuse at 1 ml/hr

**DOSE IF PATIENT AGED 10 – 15 YEARS AND WEIGHT > 35 KG: 0.7 MG/KG/HOUR**

- Draw up neat IV Aminophylline solution (25 mg/ml) into a 50 ml syringe, then infuse at 0.028 ml/kg/hr

For example if you have a 40 kg child then infusion rate will be 40 x 0.028 = 1.12 ml/hr

**DOSE ADJUSTMENT FOR OBESITY**

Use 50th percentile of expected weight for age.

**FACTORS INCREASING AMINOPHYLLINE CLEARANCE**

- Tobacco
- Phenytoin
- Carbamazepine
- Phenobarbitone
FACTORS DECREASING AMINOPHYLLINE CLEARANCE

- Influenza vaccination
- Pulmonary oedema
- Hepatic or renal dysfunction
- Cimetidine
- Erythromycin
- Ciprofloxacin

REFERENCES:

British Guideline on the Management of Asthma
(http://www.sign.ac.uk/guidelines/published/support/guideline63/download.html)


Cheuk DKL, Chau TCH, Lee SL. A meta-analysis on intravenous magnesium sulphate for treating acute asthma. Arch Dis Child 2005; 90: 74-77

BRONCHIOLITIS

Children may be admitted to the ICU / HDU for ventilation, CPAP or observation if there is sufficient concern that they are likely to require emergency respiratory support. As with all ICU admissions, the Intensivist must be involved. All admissions must be notified to the Paediatrician on call by on-call paediatric staff, who have the ability to attend as they see fit or as they are requested to.

The most likely infecting organism is respiratory syncytial virus (RSV) which is isolated in 75% of children less than two years of age hospitalized for bronchiolitis.

CLINICAL FEATURES

Profuse coryza, congestion and a low-grade fever.

Signs of lower respiratory tract involvement include:

- Cough
- Dyspnoea
- Wheezing
- Feeding difficulties
Severe cases

Respiratory distress with:

- Tachypnoea
- Nasal flaring
- Retractions
- Head bobbing
- Obtundation and *possibly* cyanosis

Infants under six months of age are the most severely affected due to smaller, more easily obstructed airways and decreased ability to clear secretions. Apnoeas are common in infants – central non-obstructive - may require intubation in 10%. Otitis media, retractions, fine rales and diffuse, fine wheezing occur. Rarely - myocarditis, supra-ventricular and ventricular dysrhythmias and inappropriate secretion of anti-diuretic hormone may occur.

Chest radiographs - usually reveal hyperinflation and 20-30% will show lobar infiltrates and/or atelectasis. May be normal.

**LABORATORY**

Blood - CBC and electrolytes just prior to or at admission and electrolytes daily.

Microbiology - Blood cultures are advisable (incidence of bacteraemia 9% ICU admissions with bronchiolitis in one study).

Nasopharyngeal aspirates for RSV antigen – more sensitive than nasal swabs (with green viral swab).

**TREATMENT**

If severe enough for ICU admission, the child will require:

- Oxygen to maintain saturation 92-94%
- Barrier nursing
- Disturbance of the child is minimized
- Secure IVI access and appropriate dextrose containing fluid infusion – paediatric / other staff may need to assist with this before or after ICU admission

Keep NPO until confident intubation will not be required. IV maintenance at 75% of their daily volume as calculated by the 4:2:1 rule. In the absence of vomiting and anticipated intubation, nasogastric feeding appears safe and can be commenced as continuous and then bolus feeds according to the severity of illness. Eventual oral feeding may be re instituted as work of breathing improves, either in ICU or in the paediatric ward.
• Bronchodilators – see below

• Caffeine or aminophylline are prescribed if requested by Intensivist or Paediatrician

### CPAP

CPAP is used as a trial of treatment for children with moderate to severe respiratory distress or frequent relatively brief apnoeas. Prolonged apnoeas do not usually respond satisfactorily to CPAP. 5cm H2O usually prescribed and adjusted upwards if felt necessary. CPAP has not been proven to have a role in bronchiolitis in any clinical trial, but at the least seems to provide symptomatic benefit.

- Appropriate monitoring: If on CPAP or unstable, monitor PaCO2 transcutaneously continuously if available

### INVASIVE VENTILATORY SUPPORT

These patients should be nasally intubated with the appropriate size tube.

The ventilator should initially be set on a pressure regulated mode with a pressure of 20cm H2O (volumes may be misleading in children with large leaks).

Look for chest movement and EtCO2 trace – adjust the ventilator as appropriate to ensure PaO2 > 60mmHg and the desired PaCO2. Higher PaCO2 may be tolerated. End-tidal CO2 monitoring must be used (see separate guideline).

If ventilated, an arterial line should be inserted if possible. Papaverine added to prevent arterial line spasm as per guideline.

Insert a nasogastric tube and feed enterally.

Treatment is largely symptomatic.

### SEDATION

Sedation would involve the use of morphine 0.5mg/kg diluted in 50ml 5% dextrose plus / minus midazolam 3mg/kg (1ml/hr = 1ug/kg/min) diluted in 50 ml dextrose at 0 – 4ml per hour. Phenobarbitone and chloral hydrate may also be considered. Muscle relaxants are often used for the first several hours.

### EMERGENCIES IN VENTILATED CHILDREN WITH BRONCHIOILTIS

Prolonged, disturbing episodes of arterial desaturation often accompanied by frank cyanosis, bradycardia and even cardiac arrest are relatively frequent in children ventilated for respiratory distress. A simple plan is followed to try and safely manage this disturbing problem:

“Bag” the child with 100% oxygen using a Laerdal bag - sometimes this action, plus suction relieves the situation. Note whether a suction catheter passes fairly easily or not at all
Rapidly confirm ETT position using capnography and/or direct laryngoscopy and visual inspection of depth of insertion compared to known previous position.

Remove ETT rapidly if it appears oesophageally misplaced or blocked based on above tests.

- Reoxygenate and reintubate, calling for help as necessary and confirming ETT position with capnography and if necessary direct laryngoscopy
- Commence cardiac compressions and use appropriate drug treatment if indicated
- Consider a chest X-ray even if you do not reintubate- most frequently this does not lead to a change in treatment or even show a change however
- Reconsider the ventilation strategy, sedation and paralysis regimen in consultation with the Intensivist. Ensure gastric decompression with NG tube
- Frequently, the problem is recurrent and despite its disturbing nature, has to be accepted as part and parcel of what has become a life-threatening condition. Maintaining ETT security and patency, not unduly prolonging the period of sedation/paralysis and appropriate ventilatory treatment minimizes problems

## DRUGS

- Bronchodilators - despite negative results in well designed trials there remains enthusiasm in some clinical quarters for use of bronchodilators. These are administered if requested by Intensivist or Paediatrician
- Antibiotics - should be strongly considered in a sick young infant, especially presenting with apnoeas. Use in bronchiolitis is controversial, but the incidence of bacteraemia in this group is said to be approx 9% which may justify relatively liberal initial empirical use until blood cultures are proven negative. Antibiotics are given if requested by Intensivist or Paediatrician
- Ribavirin is not used
- The role of Methylxanthines is unclear. A single non-randomised, retrospective trial of intravenous caffeine in a group of 7 infants between 32-38 weeks post gestation suggested benefit

## REFERENCE:


Ralston S et al APP guidelines e1474-1502 Pediatrics October 2014
CROUP

Croup refers to the clinical syndrome of harsh “barking” cough and inspiratory stridor.

The commonest cause is a viral laryngotracheobronchitis (LTB).

Other causes of stridor must be considered:

- Foreign body
- Typically sudden onset
- Epiglottitis (very rare in usually well, immunized children - no childhood cases in the Waikato since 1996):
  - Short history (over a few hours), high fever, sitting and drooling saliva. Child “refuses” to cough or swallow (too painful). External laryngeal tenderness often present. Not so much inspiratory stridor as muffled expiratory noise. Difficult to confuse with croup
- Retropharyngeal abscess
- Acute tonsillar hypertrophy
- Bacterial tracheitis:
  - Starts with typical croup followed not by improvement over 1-2 days but by deterioration, with sick, toxic child (rare)
- Laryngomalacia:
  - Subacute/chronic history
- Tumour/subglottic haemangioma
- Trauma

LTB typically affects children aged between 6-36 months with a peak incidence at 12-24 months. It is usually a mild self-limiting illness but may occasionally cause severe airway obstruction. Typically it develops over several days along with a concurrent coryzal illness.

Spasmodic croup, thought related to “allergy” to viral antigens, often arises suddenly in the middle of the night, rarely requires hospital admission or indeed any specific treatment and often resolves within a few hours.
CLINICAL FEATURES OF SEVERE CROUP

- Inspiratory AND expiratory stridor at rest
- Tachypnoea
- Marked tracheal tug and chest wall retraction
- Agitation or exhaustion

SIGNS OF IMMINENT AIRWAY OBSTRUCTION / CARDIORESPIRATORY ARREST INCLUDE:

- Decreasing level of consciousness
- Cyanosis, especially if on oxygen
- Bradycardia
- Ineffectual respiration
- Slowing respiratory rate (without other signs of improvement)

EXAMINING THE CHILD WITH SEVERE STRIDOR

- Do not lie the child flat
- Do not instrument the airway (eg with wooden spatula)
- Do not distress the child unnecessarily (eg separating from parents)
- Do not send or allow to be sent unaccompanied to Xray department (e.g. for lateral neck Xray)

MANAGEMENT OF SEVERE CROUP

- Exclude alternative diagnoses eg foreign body, epiglottitis etc
- Oxygen:
  - Often poorly tolerated by young children, but deliver by method best tolerated
- Humidification has not been shown to be of any benefit
- IV access
- If ICU admission is seriously being sought because the risk of requirement for intubation is thought high, serious consideration must be given to IV access placement in the ED. If the child is being transported to theatre for laryngoscopy under GA, the timing of IV access can be discussed with the responsible Anaesthetist
- Steroids:
  - Dexamethasone 0.3mg/kg PO/IV/IM, then 0.15mg/kg 6hly. Prednisone is often given, as it is more readily available
• Nebulised adrenaline:
  o 0.5ml/kg 1:1000 adrenaline nebuliser (max 5mls = 5mg) will give temporary improvement only and may “buy time” for steroids to become effective

**INTUBATION**

Indications include:

• increasing respiratory distress

• cyanosis unresponsive to oxygen

• exhaustion or confusion

• high oxygen requirement (>60-70%)

• cardiorespiratory arrest

• poor response or transient (<1hour) response to adrenaline

Intubation will be performed in theatre by an Anaesthetist. Contact Intensivist first if you think intubation may be required.

**Issues surrounding drug prescription:**

Systemic corticosteroids are the mainstay of drug treatment.

Nebulised budesonide is used in mild to moderate croup of all types - i.e. unsuitable for children thought to require ICU admission.

Nebulised adrenaline is an emergency rescue treatment. It buys you time (up to 2 hours) to allow the corticosteroid to become effective. Any child receiving nebulised adrenaline will have already received or will immediately receive systemic corticosteroids.

Antibiotics are not indicated in uncomplicated croup.

**No patient may leave the emergency department bound for ICU without having received systemic corticosteroids, be they orally, intravenously or intramuscularly administered**

**DRUG DOSES**

• Dexamethasone 0.3 mg/kg PO/IV then 0.15mg/kg/12hourly for maintenance

• Prednisone 2mg/kg PO then 1mg/kg/12hourly for maintenance

• Budesonide 2-4mg Neb then 2mg/12 hourly for maintenance

• Adrenaline (1:1000 ie 1mg/ml)-1ml 1:1000 starting minimum dose up to 0.5ml/kg (max 5mls) Neb
SEIZURES IN CHILDREN

DEFINITION OF STATUS EPILEPTICUS

Generalised convulsion lasting >30 minutes, or repeated convulsions occurring over a 30 minute period without recovery of consciousness between each convulsion.

(N.B. cf. definition of typical febrile convulsion up to 20 mins).

Many children arrive having received rectal diazepam from parents or paramedics. The general consensus is this should NOT be considered and that treatment should commence at the start of the algorithm.

STEP 1

- Initial assessment should follow ABCDEFG (Don't Ever Forget Glucose) principle
- Oral or nasal (teeth clenching or “too awake” to tolerate oral airway) airway if necessary
- Many seizure patients you are called to will not require consideration of intubation
- High flow oxygen
- Brief bag mask ventilation is sometimes required after benzodiazepine administration
- Take brief history & exam (considering differentials such as tonic spasm secondary to RICP, drug-induced dystonia)

Most seizures will stop within 5 minutes, but treatment should begin within 10 minutes:

- If intravenous access is immediately established give 0.1–0.25 mg/kg diazepam over 60 seconds. Midazolam 0.1mg/kg IV is an alternative if the attending doctor is more familiar with this
- If no IV access then give rectal diazepam 0.5mg/kg or midazolam intramuscularly as above. Consider intraosseous access

If after 10 minutes the initial seizure has not stopped or another has begun then progress to step 2.

STEP 2

Establish IV or IO access and give phenytoin 20mg/kg unless child already on it. Max rate 1mg/kg/min-usually given over 30-45 minutes however. Further doses of benzodiazepine may be necessary during this period.
STEP 3

Discuss case with Intensivist if ICU admission is sought by Paediatric staff, it is clear seizures are not controlled or if a severe acute underlying condition is suspected based on history.

- IV phenobarbitone 20mg/kg over 10 minutes or sodium valproate 15mg/kg over 45 mins are alternatives if phenytoin cannot be given for some reason
- Optimise phenytoin dosage as necessary when levels available

STEP 4

- Intubation may prove necessary if seizures continue, respiration becomes inadequate or if further barbiturate dosing is envisaged
- Further investigation as appropriate and directed by Paediatrician/Intensivist

Several approaches are possible:

- Midazolam infusion (0.15mg/kg over 30 mins, followed by 1mcg/kg/min infusion, increasing by 1mcg/kg/min every 15 mins if seizures do not abate)
- Further phenobarbitone, valproate etc.

**No child may leave the ED bound for ICU with a diagnosis of status epilepticus without a firm reason for not receiving an adequate loading dose of long acting anticonvulsant. This applies to children where the status abates prior to ED arrival or with the use of benzodiazepines.**

In these patients, seizures frequently recur and often “needlessly” so. Rare patients are on multiple anticonvulsants or cannot receive standard long acting anticonvulsants for some reason. A different approach is needed for these children.

REFERENCE:


GUIDELINES FOR THE MANAGEMENT OF A CHILD WITH DKA PART 1

**CLINICAL FINDINGS**

- Dehydration
- Hyperglycaemia
- Ketoacidosis
• Electrolyte derangement
• Hyperosmolar state

**TREATMENT RATIONALE**

The greatest mortality and morbidity in the setting of paediatric DKA is as a consequence of cerebral oedema. It is thought that rapid correction of the hyperosmolar state produces an osmotic dysequilibrium and can result in cerebral oedema. This occurs in approximately 7 per 1000 paediatric DKA episodes. It is more common in newly diagnosed diabetics.

In view of this, there has been a recent trend to replacing volume deficit slowly, as well as slowly correcting hyperosmolality.

**INITIAL MANAGEMENT**

• Ensure adequacy of airway and breathing
• If shocked give 20mls/kg bolus 0.9% saline and reassess
• If not shocked give 10mls/kg bolus 0.9% saline over an hour
• Assess conscious level

**INVESTIGATIONS**

Electrolytes, urea, creatinine, serum osmolality, glucose, CBC, blood gas (venous if not arterial), blood cultures, plasma betahydroxybutyrate, urinalysis and CXR.

**LINES & TUBES**

X2 IV lines and consider a urinary catheter

Consider central venous access (rarely practical) and/or arterial line as regular sampling will be necessary

**VOLUME RESUSCITATION**

Prescriptive volume resuscitation can not accommodate the wide variation of clinical presentation, but may be used to guide treatment.

Remember the principle of low volume resuscitation and slow (36-48hours) correction of deficit.

**ESTIMATE VOLUME DEFICIT**

Based on clinical assessment of the child.

Calculate deficit as 10mls/kg for every % point dehydrated:

- Total Deficit = Estimated % dehydration x 10 x weight (kg)
• Subtract volume of any fluid boluses given and replace remaining deficit over 36-48 hours (depending on osmolality at presentation) in addition to maintenance fluids

Remember this is purely an estimate, but will help guide initiation of therapy.

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<tr>
<td>Use 0.45%Saline/ 5%Dextrose (equal volumes 0.9%Saline &amp; 10%Dextrose) when glucose &lt;15mmol.</td>
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<td>Should be done slowly. Aim for 2-4mmol/l/hour.</td>
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<td>In practice, glucose tends to drop much quicker than this on initiation of volume resuscitation. On the basis of this, commence insulin infusion 30-60 minutes after fluid initiation:</td>
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<td>• Start insulin (Actrapid) infusion at 0.05units/kg/hour (titrate +/- 0.025units/kg/hour)</td>
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<tr>
<td>• No bolus of insulin, unless life threatening hyperkalaemia</td>
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<tr>
<td>• Aim to keep glucose 10-20mmol/l until acidosis resolving (pH&gt;7.3, HCO₃⁻ &gt; 15)</td>
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<tbody>
<tr>
<td>Should be done slowly. Aim for drop of less than 5mosmol/l/hour. For the same reasons as mentioned above, this is difficult to achieve in the first couple of hours:</td>
</tr>
<tr>
<td>o Calculated Osmolality = ([Na⁺] x2) + [urea] + [glucose]</td>
</tr>
<tr>
<td>o From a practical view, ensure Na⁺ is not falling</td>
</tr>
<tr>
<td>N.B. a blood sample should be sent to the lab to measure osmolality, in case unmeasured osmoles (e.g. ketones or lactate) present in significant amounts.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CORRECTING ACIDOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bicarbonate is rarely indicated. Consult Intensivist.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>POTASSIUM REPLACEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium deficit is often very large. On presentation, however, it is common for plasma potassium to be normal or even high, but as acidosis resolves it drops rapidly</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PHOSPHATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypophosphataemia is common and can be corrected if desired with potassium dihydrogen.</td>
</tr>
</tbody>
</table>
OBSERVE FOR SIGNS OF RAISED ICP

Hourly neuro observations should be undertaken for the first 24 hours, and half hourly if GCS <15.

Persistently low or deteriorating scores are highly suggestive of cerebral oedema.

Give mannitol 0.25g/kg = 1.25ml/kg of 20% solution and repeat as necessary (guided by senior staff):

- If hyponatraemic consider 4M Saline 0.25mls/kg (up to 20ml)
- Liaise with senior staff

Once the child can drink, remember to cut down or stop IV fluid infusion.

Don’t routinely try and replace polyuria.

Remember that so called maintenance infusion rates in children probably overestimate the fluid intake necessary in sick children, so don’t be afraid to round down fluid intake calculations at every opportunity.
Use in conjunction with Part 1 which will give background to the condition. This is a simplified, therefore more usable therapeutic guide.

This guideline is compatible with all major recent guidelines published on paediatric DKA.

<table>
<thead>
<tr>
<th>Timeline</th>
<th>Fluid Volumes/Composition</th>
<th>Insulin</th>
<th>Electrolytes</th>
<th>Monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnosis at 0 minutes</strong></td>
<td>0.9% saline : 20 ml/kg bolus if in shock</td>
<td>-</td>
<td>No potassium added to 20 ml/kg fast bolus for shock.</td>
<td>Place venous or arterial sampling line if feasible.</td>
</tr>
<tr>
<td></td>
<td>: 0.9% saline 10 ml/kg over 1 hour if not shocked, while fluid calculations made and insulin infusion started</td>
<td></td>
<td>Include 20 mmol/l potassium per litre of fluid for the 10 ml/kg slow bolus, unless hyperkalaemic.</td>
<td></td>
</tr>
<tr>
<td><strong>30-60 minutes post fluid commencement</strong></td>
<td>Use a <strong>conservative maintenance infusion rate</strong> of 0.9% saline (see below (1)), aiming to rehydrate over 48 hours.</td>
<td>0.05 U/kg/hour suitable for all ages, preferred if &lt;3 years age. Dose of 0.1 U/kg/hour also used.</td>
<td>Add potassium to maintenance/rehydration fluid unless hyperkalaemic.</td>
<td>Commence hourly BM stix. Minimum two more measurements of potassium and acid-base over next 12 hours, but 2-4 hourly samples drawn from sampling line preferred.</td>
</tr>
</tbody>
</table>

*Take into account any fluid boluses/other infusions.*

- Use 8% dehydration as your maximum estimate-dehydration is easily overestimated.

- Check infusion rate will not be excessive by using stopping rules below (2).

- Using Hartmann’s or PL148 is rational but there is little in the literature about this.

Understanding the principles and the tools available is the key.
| Blood sugar < 15 mmol/l | 0.45% saline/5% dextrose  
At same rate as before. | Do not turn off insulin unless hypoglycaemic. Reduce insulin infusion to 0.025 U/kg/h if BM dropping too fast - if this is insufficient, increase dextrose intake (e.g. saline/dextrose 10%) | BM stix measurements may be done 2 hourly if stable. |
|------------------------|------------------------|-------------------------------------------------|--------------------------------------------------|
| Child begins to drink   | Reduce fluid infusion  
appropriately. |MITTED MAINTENANCE FLUIDS | |
| Child getting ready to eat, anion gap acidosis resolved | Stop fluid infusion once transition to subcutaneous insulin is instituted. | Consult Paediatrics for subcutaneous insulin dosage/timing and insulin infusion cessation timing. | Potassium replacement orally if required. | BM stix 2-4 hourly. |
| CONSERVATIVE MAINTENANCE FLUIDS | | | |
| Weight (kg) | 0-12.9 | 80 ml/kg/day | |
| | 13-19.9 | 65 ml/kg/day | |
| | 20-34.9 | 55 ml/kg/day | |
| | 35-59.9 | 45 ml/kg/day | |
| | > 60 | 35 ml/kg/day | |

From Edge JA, BSEPD Recommended DKA Guidelines Nov 2013
STOPPING RULES FOR HOURLY FLUID INFUSION.

Use these to check for the possibility that you are going to give too much fluid according to the degree of dehydration you have assessed to be present.

The simple calculation used for estimating fluid deficit is: Total Deficit = Estimated % dehydration x 10 x weight (kg)

Caution must be exercised to avoid excessive fluid infusion in paediatric DKA. Using the table below, taken from the NZ National clinical network for child and youth diabetes (2014 (in turn modified from the RCH, Melbourne clinical practice guidelines), and the ISPAD 2014 guidelines) allows for recognition of simple mathematical calculation errors in calculation of total fluid infusion.

*Remember: always account for other fluids given as boluses, diluents and infusions and don’t be afraid to round down calculations, then don’t give more fluid per hour than:*
### POTASSIUM REPLACEMENT

Restrictions on potassium solution administration depend mainly on venotoxic concentrations. Usually a maximum of 30 mmol/l of diluent will hold a reasonable balance between infusion pain, venotoxicity and serious hypokalaemia. One of the decisions that must be taken by individual practitioners is where the balance of risks lies. Central access may be preferred, but in general is not feasible without sedation.

Simpler stopping rules are: don’t exceed 3l/metre squared BSA per day

- don’t exceed twice maintenance rates, with maintenance rates defined in (1) above.
Arrhythmias in children due to hypokalaemia are fortunately very rare. When possible, oral potassium intake may be useful (as chlorvescent). 

REFERENCES


MENINGITIS

This condition is particularly challenging in the paediatric population as symptoms and signs are often non-specific, especially in infancy. A high index of suspicion should be maintained, and empirical treatment commenced until the diagnosis can be excluded.

Meningitis is classically subdivided into bacterial and aseptic forms depending on the appearance of CSF at microscopy.

BACTERIAL MENINGITIS

Likely causative organisms for bacterial meningitis depend on age.

Likely organism involvement according to age

<table>
<thead>
<tr>
<th>Age</th>
<th>Common Organisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 month</td>
<td>Grp B Streptococci</td>
</tr>
<tr>
<td></td>
<td>Enterococci</td>
</tr>
<tr>
<td></td>
<td>Gram -ve bacilli</td>
</tr>
<tr>
<td></td>
<td>Listeria monocytogenes</td>
</tr>
<tr>
<td>1 to 3 months</td>
<td>As above &amp; below</td>
</tr>
<tr>
<td>&gt; 3 months</td>
<td>S. pneumoniae</td>
</tr>
<tr>
<td></td>
<td>N. meningitidis</td>
</tr>
<tr>
<td></td>
<td>H. influenzae</td>
</tr>
</tbody>
</table>

ANTIBIOTIC RESISTANCE

- Penicillin and cephalosporin resistant strains of S. pneumoniae are becoming increasingly prominent throughout the world although uncommon in the UK and Australasia

- Add vancomycin if Gram +ve diplococci are seen on Gram stain

THE STEROID CONTROVERSY

The contention is that corticosteroids may attenuate the inflammatory meningitic process thus reducing the incidence of long-term neurological sequelae.
Steroids are given if requested by Paediatrician or Intensivist

**MENINGOCOCCAL DISEASE**

A high index of suspicion allows early recognition of the disease and institution of aggressive treatment.

Meningococcal disease presents as meningitis in ~50%, septicaemia in ~10% and mixed picture in the remaining ~40% of cases.

20% of cases will not have a petechial rash. It is also important to note that other bacteria and viruses can present with petechiae, albeit less commonly.

From a practical point of view any child with a fever and petechial rash should be assumed to have meningococcal disease until proven otherwise. Give cefotaxime or ceftriaxone immediately, DO NOT wait for laboratory results.

**ASEPTIC MENINGITIS**

Causes can be subdivided into infectious and non-infectious causes.

**CLINICAL FEATURES**

The classic triad of headache, neck stiffness and photophobia may be present in older children but often absent in infants. A history of poor feeding, high temperature, vomiting, apnoeas, convulsions, irritability and lethargy should raise suspicion. Don't forget to ask about immunisations.

Classical signs (Kernig's, Brudzinski and nuchal stiffness) are often absent. If these signs are present they are neither sensitive nor specific for differentiating between viral and bacterial meningitis.

Petechial rash is often said to be pathognomonic of meningococcus but does occur with viral and other bacterial causes. Don't forget to examine the fontanelle (if appropriate) and ears, nose and throat for possible septic focus.

**DIFFERENTIAL DIAGNOSES**

- **Viral encephalitis:**
  
  - Classically presents with fever, headache and decreasing GCS. Focal neurology and seizures are common findings. With a compatible history, acyclovir is commonly given

- **Viral meningitis:**
  
  - Usually mild and self limiting, but severe forms can be easy to confuse with bacterial meningitis. CSF analysis will help

- **Other causes of non-traumatic coma (see separate section)**

A GCS < 13, complex seizures or focal neurology should raise the suspicion of a diagnosis other than bacterial meningitis the most important of which include cerebral oedema or space occupying lesion. CT scan, and not LP, is indicated under these circumstances.
This is a guide to the initial investigations that you should consider. Obviously CSF will not be available in all cases.

### INVESTIGATIONS IN CNS INFECTIONS

<table>
<thead>
<tr>
<th>Sample</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>Routine haematology, coagulation, biochemistry, renal &amp; hepatic function</td>
</tr>
<tr>
<td></td>
<td>Culture &amp; sensitivity</td>
</tr>
<tr>
<td>Urine</td>
<td>Pneumococcal antigen</td>
</tr>
<tr>
<td>Blood &amp; urine</td>
<td>Influenza A, CMV, mycoplasma, chlamydia serology</td>
</tr>
<tr>
<td>Blood +/- CSF</td>
<td>PCR meningococcus, enterovirus &amp; HSV</td>
</tr>
<tr>
<td>CSF</td>
<td>Microscopy &amp; Gram stain</td>
</tr>
<tr>
<td></td>
<td>Biochemistry (glucose &amp; protein) NB don't forget serum glucose at same time as LP</td>
</tr>
<tr>
<td></td>
<td>Culture &amp; sensitivity</td>
</tr>
<tr>
<td></td>
<td>Enterovirus PCR</td>
</tr>
</tbody>
</table>
### Other investigations

<table>
<thead>
<tr>
<th>Other investigations</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT scan</td>
<td>GCS &lt;13 with sudden, subacute (&gt;24 hours) or chronic symptoms (these suggest other aetiologies)</td>
</tr>
<tr>
<td></td>
<td>complex seizures</td>
</tr>
<tr>
<td></td>
<td>focal neurology</td>
</tr>
<tr>
<td>MRI &amp; EEG</td>
<td>Viral encephalitis a possibility</td>
</tr>
<tr>
<td>CSF</td>
<td>TB &amp; cryptococcus if history suggestive</td>
</tr>
</tbody>
</table>

### OTHER DISEASE MIMICKING MENINGITIS

A proportion of children with symptoms or signs suggestive of meningitis will have another diagnosis eg tumour, encephalitis, abscess or haemorrhage. The onset and duration of symptoms, presence of focal signs, complex seizures or depressed level of consciousness indicate the possibility of these diagnoses.

Under these circumstances CT scan is the first investigation of choice.

### LUMBAR PUNCTURE

Lumbar puncture is the definitive investigation for diagnosing meningitis and should be performed in all cases of suspected meningitis unless there are specific contraindications before antibiotics are given.

**Contraindications to LP**

- Signs of raised intracranial pressure
- Complex convulsive seizures
- Focal neurological signs
- GCS <13
- Coagulopathy
- Obvious shock or respiratory failure
- Local superficial infection

**Benefits of lumbar puncture:**

- A definitive diagnosis of meningitis is made, organisms being seen in approximately ¾ of cases
• Allows identification of unusual organisms (especially at the extremes of age and immunocompromised)

• Allows identification of resistant organisms (eg resistant pneumonocci)

• Once sensitivities are known, broad spectrum antibiotics can be replaced with narrow spectrum monotherapy

• Allows discontinuation of antibiotics if the CSF is clear

• Removes differential diagnoses eg encephalitis